

Xmas 1902

To my dear Father : —

Wishing you a very Merry
Xmas & a Happy New Year with many
more to follow.

This book was purchased with part of the
money of the "Sutton Scholarship". A
prize which I won solely by your early
advice & teaching, & by your never failing
kindnesses.

From your affectionate, &
grateful son

Austin C. LeRoussignol

THE
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QUAIN'S DICTIONARY OF MEDICINE

BY VARIOUS WRITERS

THIRD EDITION

LARGELY REWRITTEN AND REVISED THROUGHOUT

*WITH FOURTEEN COLOURED PLATES AND NUMEROUS OTHER
ILLUSTRATIONS*

EDITED BY

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PREFACE

IN preparing the new edition of this Dictionary my object has been to produce a book which shall serve as a reliable and readily available work of reference for the Practitioner and Student of Medicine.

The general scheme of the book—so skilfully planned by the late Sir Richard Quain and his assistant editors—has been preserved, and the special emphasis laid on the diagnosis and treatment of disease has been maintained, although the pathology and ætiology have also been very carefully considered and revised. Some departure from the original plan has been deemed advisable. Many articles—excellent in themselves but not in accord with the special object in view—have been omitted, while repetitions have been as far as possible excluded. It has thus been found practicable to include a large number of new articles, to rewrite many others, and yet to publish the Dictionary in a single volume. The recent and ever-increasing incursions of Surgery into the realm of Medicine have received adequate recognition, and more space has been allotted to what are generally known as ‘special branches.’ A large number of cross references have been inserted in order to bring under the notice of the reader articles giving further information on the various subjects.

My sincere thanks are in the first place due to the Contributors, without whose labour and hearty co-operation it would have been impossible to have produced this work in the short period of eighteen months. For the readiness with which they received suggestions, for the self-suppression with which they permitted deletions, and for the rapidity with which they completed their articles I can hardly be sufficiently grateful. For the pathological and more strictly scientific articles I have secured the co-operation of those in touch with experimental work, and have throughout freely availed myself of the services of the younger members of the profession.

To Dr. Harold and Dr. Bosanquet, the Assistant Editors, my obligation is greater than I can easily express. Without them the task could never have been performed; and my principal regret is that the readers of this work can have no conception of the untiring labour which they have bestowed upon it.

The enterprise and courtesy of the Publishers deserve hearty editorial recognition. They have enabled us to insert twenty-one full-page plates (fourteen of them in colour), and have transformed my own task into a delight.

For the information of readers it should be stated that in articles for which two contributors are jointly responsible the names are, where possible, placed side by side, but that when the article written by one contributor has been revised by another the name of the reviser is placed under that of the original contributor.

H. MONTAGUE MURRAY.

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DICTIONARY OF M E D I C I N E

A

ABDOMEN, Diseases of the.—Excluding a few peculiar affections, the diseases of the abdomen may be arranged under the following groups:—

I. Diseases of the anterior abdominal walls.

II. Diseases of the peritoneum and its folds.

III. Diseases of the organs and systems contained within the abdominal cavity, namely:—1. Stomach and Intestines; 2. Hepatic structures, including the liver, gall-bladder, gall-ducts, and vessels; 3. Spleen; 4. Pancreas; 5. Suprarenal capsules; 6. Urinary apparatus, viz. the kidneys and ureters, and the bladder; 7. Female generative organs, including the uterus and its broad ligaments, the Fallopian tubes and the ovaries; 8. Absorbent glands.

IV. Diseases of the abdominal vessels, especially the aorta and the iliac arteries.

V. Diseases of the sympathetic or other nerves contained within the abdomen.

VI. Diseases originating in connection with the cellular tissue, such as inflammation or abscess.

VII. Diseases springing from the posterior boundary of the abdomen; from the pelvis or the structures lining it; or from the diaphragm, and thence invading the abdominal cavity.

VIII. Diseases encroaching upon the abdomen from other parts, especially from the thorax.

It must be borne in mind that the groups of diseases above mentioned may be presented in various combinations, two or more structures being not uncommonly implicated at the same time.

Several of the abdominal organs are very liable to so-called *functional disorders*, and these disorders often give rise to prominent and troublesome symptoms, which are urgently complained of by the patient. Definite *organic diseases* are also of common occurrence, many of them being of a very serious character. Some of the organs contained within the abdomen are liable to *malposition* or *displacement*, as well as to *malformations*, these being either congenital or acquired; while the hollow viscera may be the seat of *obstruction* or *accumulations* of different kinds. Each of these conditions may become clinically important.

Abdominal lesions are frequently purely *local* in their origin, but several of them are but local manifestations of some more *general* condition, being associated either with certain acute febrile diseases, e.g. typhoid fever; or with such affections as cancer or tuberculosis. Again, symptoms connected with

the abdomen may depend upon disease in some remote part of the body; or some of its organs may become the seat of morbid changes as a consequence of disease in other structures. For instance, vomiting is frequently associated with cerebral disorders; while affections of the heart are liable to lead to troublesome symptoms, as well as to serious lesions, in connection with many of the abdominal viscera. Lastly, a morbid condition of one organ within the abdomen may be the direct means of originating *secondary* mischief in other structures.

FREDERICK T. ROBERTS.

ABDOMEN, Examination of.—The neglect of submitting patients to a satisfactory physical examination is a frequent source of error in diagnosis in cases of abdominal disease, and there ought to be no hesitation or delay in resorting to this method of clinical investigation whenever it seems called for. The precise course to be pursued must vary according to circumstances, but the following outline will serve to indicate the plan of procedure ordinarily required.

First, there are certain modes of examination which are applied to the abdomen externally, including *Inspection*; *Palpation* or *Manipulation*; *Mensuration*; *Percussion*; and *Auscultation* (see **PHYSICAL EXAMINATION**). Of these, inspection, palpation, and percussion are by far the most important, and have, in the large majority of cases, to be relied upon for the information required. In exceptional instances *Succession*, or shaking the patient, proves serviceable, by bringing out certain sensations or sounds. In order to carry out these methods properly, it is necessary to expose the abdomen sufficiently, due regard being paid to decency in the examination of females; to place the patient in a suitable position; and to see that the muscles of the abdominal walls are duly relaxed. The best position is for the patient to lie on the back, in a half-reclining attitude, with the head and shoulders well raised, and the thighs and knees more or less flexed. This posture serves to relax the abdominal muscles, which may be further aided by taking off the patient's attention by conversation or in other ways, as well as by directing him to breathe deeply. The position, however, has often to be varied in the investigation of particular cases, and much information is frequently gained by noticing the effects of a change in posture.

The objective conditions which may be revealed by the modes of examination thus far considered are as follows:—1. The state of the superficial structures. 2. The size and shape of the abdomen, generally and locally, as indicating any alteration in the volume of the ordinary contents of the abdomen, or the presence of some new or fresh element, such as dropsical fluid or a tumour. 3. The characters of the abdominal respiratory movements; and the presence of any unusual sensations during the act of breathing, such as friction-fremitus. 4. The sensations experienced on palpation and percussion over the abdomen, either as a whole, or in any particular part of it, such as its mobility, degree of resistance, regularity, consistence; as well as the presence of certain peculiar sensations, e.g. fluctuation, or hydatid-fremitus. 5. The presence and characters of any pulsation. 6. The occurrence of abnormal movements within the abdomen, as those of a fetus. 7. The sounds elicited, generally and locally, on percussion. 8. The presence of certain sounds within the abdomen, heard on auscultation, such as friction-sounds; murmurs connected with aneurysm or due to pressure on an artery; or murmurs and sounds associated with the pregnant uterus.

Here it may be remarked that it is highly important in all cases to see that no accumulation of feces exists within the bowel, and that the bladder is properly emptied, otherwise very serious mistakes are liable to be made. Purgatives and enemata are needed in order to remove any fecal collection. The urine should also be properly tested in every instance; and much information may often be gained in the investigation of affections of the alimentary canal, from a personal inspection or more complete examination of feces or vomited matters.

Secondly, it not uncommonly happens that special modes of examination have to be applied to particular organs within the abdomen, in order to arrive at a diagnosis with any certainty. Thus it is sometimes requisite to have recourse to the use of the exploring trocar or aspirator; or to the administration of chloroform. The latter may afford direct information in certain abdominal conditions, and it may also materially assist in carrying out other methods of exploration. The use of Röntgen rays may assist in the diagnosis of renal calculus, though this method of examination is of less use in abdominal disease than in disorders of the chest or of the limbs (*see* RÖNTGEN RAYS). In certain cases more serious operative procedures have to be adopted for diagnostic purposes. *See* PARACENTESIS.

The abdomen has been, for purposes of description, artificially divided by anatomists into regions, and the seat of any local morbid condition can thus be defined and described. These regions are thus named: epigastric, hypochondriac, hypogastric, iliac, lumbar, and umbilical. *See* plate in PHYSICAL EXAMINATION.

The following is a summary of the usual procedure:—

INSPECTION.—Herniæ, inflammations, new growths &c. in wall. Pigmentation, lineæ albicantes. Thin, fat; shape, sunken, distended uniformly; more in the central regions (pointed abdomen); or more in the flanks (broad abdomen); local swelling; distended stomach (bagpipe tumour); coils of intestine; tumours; visible peristalsis; point of ending of movements; condition of umbilicus,

stretched transversely, depression obliterated, protruding, translucent; movements of wall, impaired locally or generally, falling in; condition of skin and superficial veins.

PALPATION.—Flaccidity; tension of wall from distension; general rigidity; local resistance; tenderness, local, general, superficial, deep; gurgling (e.g. right iliac fossa); succussion-splash; friction; fulness; swelling; tumour.

In the case of a tumour, note its position, size, shape, surface, edges, mobility, consistence, pulsation, occasional hardening or contraction, ballottement, fetal parts and movements.

PERCUSSION.—Locality of any change in resonance; effect of change in posture.

AUSCULTATION.—Friction; vascular bruit; foetal heart.

Liver and gall bladder . . .	} The position and condition of the abdominal viscera, and of tumours, to be ascertained by inspection, palpation, percussion, the exact limits being noted according to the regions of the abdomen, and by measurements from fixed points, or recorded on diagrams.
Spleen . . .	
Stomach . . .	
Colon . . .	
Kidneys . . .	
Bladder . . .	
Uterus or ovaries . . .	

RECTUM AND ANUS.—Physical examination, if necessary. Place patient on left side, thighs well drawn up. Inspect anus, natural, contracted, patulous; note any abnormal condition. Palpate anus and surrounding parts; examine any swelling; probe fistulæ; extent; internal opening. Pass finger well oiled; note excessive pain and any abnormality within bowel. Try gently to pass finger into or through any stricture or new growth while patient 'bears down,' and to move it among surrounding parts; bleeding excited; pass duckbill or other speculum (if necessary), and examine whole circumference of bowel. Bougies may be passed through a stricture with care. *See also* BLADDER, EXAMINATION OF; STOMACH, EXAMINATION OF; UTERUS, EXAMINATION OF.

FREDERICK T. ROBERTS.

ABDOMEN, Exploration of.—*See* PARACENTESIS.

ABDOMEN, Tumours of.—*See* ABDOMINAL CYSTS; and PHYSICAL EXAMINATION.

ABDOMINAL ANEURYSM.—Abdominal aneurysm includes aneurysm of the aorta, and of any of its branches within the abdomen.

Aneurysm of the Abdominal Aorta is essentially a disease of middle age. Of 59 cases collected by Crisp, 33 were under the age of forty. It is more common in the male than in the female sex, in the proportion of about 8 to 1; and is usually traceable to strain or injury; not infrequently it is embolic in origin (*see* ANEURYSM). The aneurysm is most frequently located in that portion of the vessel included between the aortic opening in the diaphragm and the origin of the superior mesenteric artery, being seldom met with below this point. In this situation the tumour is deeply seated; liable to tension from the crura of the diaphragm; and likely to involve the great splanchnic nerves, the semilunar ganglia, and the solar plexus. Aneurysm of the abdominal aorta is usually of the *false* variety; and, as contrasted with thoracic aneurysm, it is less often associated with extensive atheroma of the aorta, and

with fatty or other structural disease of the heart. The symptoms referable to excentric pressure are also fewer, and, with the exception of pain, are less urgent than in the thoracic variety.

SYMPTOMS AND SIGNS.—Of the symptoms, *pain* is the most characteristic and the most urgent; it is of two kinds, which are not, however, necessarily associated. The first is due to the irritation and stretching of the nerves above mentioned. It is essentially neuralgic; it is intermittent, lancinating, and paroxysmal,—encircling the body like a girdle, or radiating through the abdomen, back, pelvis, and base of the thorax, and not infrequently into either groin or testicle. The accession is sudden, and usually attributable to some definite cause of vascular excitement. The duration extends over a period varying from one to three hours, rarely longer; and the cessation is equally abrupt, leaving the patient in a state of exhaustion, but quite free from actual suffering. The second kind of pain referred to is continuous and boring; fixed at a particular point of the vertebral column; aggravated by pressure at this point, by active movement or stamping, and by gently turning the patient half round upon his axis in the standing posture; but relieved by assuming the prone position or by leaning forward. Pain so characterised is pathognomonic of erosion of the vertebræ. Pressure of an aneurysm may affect the functions of several organs within the abdomen. Thus *jaundice* may result from pressure upon the hepatic, or common biliary duct: it is, however, more frequently due to an aneurysm of the hepatic, or of the superior mesenteric artery. *Interference with the urinary secretion*, and the consequences thereof, from pressure upon the renal vessels; *dysphagia*, from pressure upon the œsophagus; *vomiting*, from obstruction of the pylorus; *displacement* of the liver forwards, or of the heart upwards—though rare symptoms—may be likewise due to the same cause. The radial pulse is not often affected. Symptoms of *constitutional irritation* and impaired nutrition are rarely exhibited, and appear only at the termination of protracted and painful cases, associated with great suffering and want of sleep.

Physical signs.—A tumour can often be felt in the epigastric and left hypochondriac regions. It is smooth, rounded, and elastic; and tends to descend. It communicates to the hand alternate movements of lifting and expansion with increasing tension, and of subsidence with relaxation. The pulsation is all but invariably single, and synchronous with the radial pulse; it is limited to the tumour, and occasionally accompanied by thrill. Pressure upon the aorta below the tumour will increase the force of impulse, diminish or abolish the thrill, and arrest the collapse. In a few recorded examples the tumour was hard and uneven on the surface, and non-expansile; and in a still smaller number no pulsation was perceptible, the aperture of communication with the artery having been blocked, or the vessel compressed on the proximal side by the growth of the aneurysm itself. Owing to the position of the hollow viscera in front, and the mass of lumbar muscles behind, the evidence from percussion is less conclusive in regard to abdominal than thoracic aneurysm. If, however, the abdominal muscles be relaxed, and the stomach and bowels free from flatus, absolute dulness to the extent of the tumour may be detected. A sound, single or double, as distinguished from a murmur, is rarely heard in

front in connection with abdominal aneurysm; whereas the existence of a sound, usually double, without a murmur, at a point on the posterior wall of the abdomen corresponding to the tumour, is the rule, and when detected, is of the utmost diagnostic value. A murmur in the recumbent posture is rarely absent in front; it is single, blowing, prolonged, post-systolic, and not transmitted into the vessel beyond. It may, however, be musical, or it may present both these characters, but at different points of the tumour; in one instance it was of a buzzing quality. Should the aneurysm have taken an exclusively backward course, which is the exception, a single murmur, not audible in front, may be heard in the back. In a few recorded cases a double murmur has been heard over the aneurysm in front. In the erect posture the murmur is usually suspended; but in a few published cases it was audible in both the erect and the recumbent posture, and in one at least in the erect posture only. These peculiarities depend upon the various conditions of the sac, its orifice, and its contents. A small aneurysm involving the posterior wall of the vessel only, and eroding the vertebræ, may be latent as to physical signs, though attended with severe fixed pain in the back, and weakness of the lower extremities. In some cases a slight retardation of the pulse in the femoral arteries has been noted.

DIAGNOSIS.—The diagnosis of abdominal aneurysm has reference mainly to its physical signs. The higher the aneurysm the more obscure will be the physical signs and the more marked the evidences of pressure. Strong pulsation of the aorta, simulating that of aneurysm, may exist in connection with hysteria, uterine or intestinal irritation, dyspepsia, or marked æmia. But in all these cases, irrespective of the positive and specific evidence presented by each, throbbing exists throughout the aorta, and is propagated into the main arteries of the lower limbs, whereas it is localised and expansile in aneurysm; and a careful exploration of the aorta—if necessary, under the influence of chloroform—will show that, in the case of a pulsating aorta, the dimensions of the artery are at all points normal. In these cases, too, although a murmur may be produced by strong pressure with the stethoscope, it does not exist when pressure is withdrawn. A cancerous or other tumour pressing upon the aorta may likewise produce a murmur, and may exhibit pulsation communicated from the aorta; but in most cases both these phenomena are promptly arrested by placing the patient in the ‘knee and elbow’ position. A tumour, in that position, will fall away from the vessel; whereas the pulsation of an aneurysm will often be more easily felt. The fixed local pain in the back, aggravated by pressure and motion, may be simulated by ordinary lumbago; and the paroxysmal visceral pain, by biliary colic. The differential diagnosis must rest upon the specific evidence in each case.

Aneurysm of the Branches of the Abdominal Aorta.—The branches most liable to aneurysm are the common iliacs and their divisions; the coeliac axis and its branches; the renal, and the superior mesenteric. *Aneurysm of the Iliac Arteries* belongs to the domain of surgery, and will not be further referred to here. *Aneurysm of the Celiac Axis* and of its branches of division, and of the *Superior Mesenteric Artery*, are, in addition to the ordinary signs, characterised by mobility of the tumour; and the first two varieties by the occurrence

of jaundice, hæmatemesis, and mælena, from pressure. *Renal* aneurysm may cause infarction in the kidney, or venous congestion by pressure on the renal vein.

DURATION AND TERMINATIONS.—The duration of life in abdominal aneurysm in a number of cases observed varied from fifteen days to eleven years. Death occurs usually: (1) by rupture of the sac into (a) the retro-peritoneal tissues, unless a secondary sac be formed; (b) the cavity of the peritoneum; (c) the left pleural cavity or corresponding lung; (d) the intestinal canal; (e) the inferior vena cava; (f) the psoas muscle; (g) the pelvis of the kidney; (h) the spinal canal; or (i) the ureter, biliary passages, or œsophagus—the order given representing the relative frequency of the fatal result; or (2) by exhaustion or syncope. The duration of life after the rupture of the aneurysm has ranged from a few minutes to several weeks. A consecutive false aneurysm, retroperitoneal in site, is specially characterised by feeble pulsation of the tumour, and diminished or arrested circulation in the femoral artery of one or both sides.

TREATMENT.—The *curative* treatment of abdominal aneurysm may be considered under three heads—*operative*, *postural* and *dietetic*, and *medicinal*. *Operative* treatment consists in pressure applied to the aorta on the proximal side of the sac, by means of tourniquets, so as completely to stop the circulation. The bowels should be first well moved and freed from flatus; and during the continuance of pressure the patient should be kept under the influence of chloroform or ether. As a rule, compression should not be employed above the level of the duodenum. Five cases, if not more, in which a cure was effected by these means have been reported. The object sought to be attained being that of effecting rapid coagulation in the sac, the period during which pressure needs to be continued in these cases varies from three quarters of an hour to ten hours and a half. Pressure on the distal side is dangerous from its liability to cause rupture. Under all circumstances, pressure must be used with care, as inflammation of the peritoneum or of the bowels has been known to result from it. Galvano-puncture, and the introduction of foreign bodies, such as iron wire and horsehair, into the aneurysmal sac have also been employed. In a small proportion of cases the latter method has been attended with a favourable result, but less often in abdominal than in thoracic aneurysms.

Bellingham introduced the plan of treatment by *posture* and *restricted diet*. Under this plan perfect repose of mind and body is, as far as practicable, to be maintained; the bowels being kept moderately free, and the dietary restricted to 40 oz. including solids and liquids. According to the method of the late Mr. Tufnell, which is based upon the same principle, but is more rigid, the patient is strictly confined to the horizontal posture for a period varying from eight to thirteen weeks, as determined by the effect upon the aneurysm, movement in bed being effected with caution; whilst, by a special arrangement, the bowels and the bladder may be evacuated without disturbance of the body. For breakfast, 2 oz. of white bread and butter, with 2 oz. of cocoa or milk, are allowed; for dinner, 3 oz. of meat, with 3 oz. of potatoes or bread, and 4 oz. of water or claret; and for supper, 2 oz. of bread and butter, and 2 oz. of milk or tea. The total amount in the twenty-four hours would be: solids 10 oz.,

liquids 8 oz. This system is not often rigidly enforced, and is not so frequently employed as formerly.

Of the various *medicinal* agents used with a view to favouring or effecting a deposit of laminated fibrin in the sac, chloride of calcium (10 to 15 grs. thrice daily) is the best, though acetate of lead and iodide of potassium are often used. Iodide of potassium may be given in doses of 10 to 20 grs. thrice daily, with a view to reducing vascular tension, and thereby relieving pain and promoting deposition in the sac, whilst perfect rest in the recumbent posture and a restricted dietary are observed. The intramuscular injection of 20 to 80 c.c. of a sterilised 1 to 3 per cent. solution of gelatine in 1 per cent. solution of sodium chloride on alternate days has been recently employed, but with very varied success. In all cases the allowance of liquids must be reduced to the lowest possible standard, whilst excretion is promoted. Alcoholic stimulants may be given in small quantity and at long intervals, if the pulse exhibit debility and the patient complain of a sensation of sinking; otherwise they should be prohibited.

The application of a few leeches, followed by a warm poultice, is very efficacious in relieving pain. The hypodermic use of morphine is still more rapidly effective. See THORACIC ANEURYSM.

C. J. NIXON.

ABDOMINAL CYSTS.—This article includes a short description of cystic tumours of the abdomen, not described under other headings.

1. **Allantoic.**—See 11. URACHAL CYSTS.

2. **Dermoid Cysts.**—Dermoid cysts are occasionally found in the peritoneum without the existence of an ovarian dermoid; it is not improbable that in reality the cyst originated in the ovary, but became detached and secondarily adherent elsewhere. When an ovarian dermoid ruptures it may give rise to *implantation cysts* in various parts of the peritoneal cavity.

3. **Hæmorrhagic or Sanguineous Cysts.**—Hæmorrhagic cysts are occasionally met with in the abdomen. In rare instances they are due to softening and extravasation of blood into sarcomata.

Simple Sanguineous Cysts may be due to traumatism and are usually found around the pancreas, in the transverse mesocolon, or mesentery. They then closely resemble peripancreatic cysts; but they may arise elsewhere; in one case, examined after death, a large hæmorrhagic cyst arose in the pelvis.

The symptoms are much the same as those of pancreatic cysts. The proper treatment is incision and drainage.

4. **Hydatid Cysts.**—See ENTOZOA.

5. **Hydronephrosis.**—See HYDRONEPHROSIS.

6. **Mesenteric Cysts.**—These cysts are rare. They may be divided into (1) *Lymphatic*, the most frequent form; either serous or chylous, due to dilatation of lymphatic vessels or to dilatation and degenerative changes in lymphatic glands; (2) *Hæmorrhagic*, usually due to traumatism, but also possibly the result of hæmorrhage into lymphatic cysts or into growths; (3) *Hydatids*; (4) cases thought to be congenital, such as dermoids, and cysts derived from the Wolffian body; (5) possibly some mesenteric cysts are due to developmental errors in the intestine.

Mesenteric cysts are more often seen in females. The characteristic signs are a fluctuating, very

movable tumour near the umbilicus with a band of resonance in front of it, due to the intestine. Though there may be no symptoms, colic, diarrhoea, or constipation is generally present, while sometimes acute obstruction occurs.

The *diagnosis* is difficult: ovarian, hydatid, and pancreatic cysts must be thought of, and solid tumours or a floating kidney borne in mind.

The *treatment* is surgical, viz. removal or incision and drainage.

7. **Omental Cysts.**—Hæmorrhagic cysts of the omentum have been met with. Dermoid cysts may be implanted on the mesentery and are derived from ovarian cysts of the same nature. It is possible that an ovarian cystadenoma might be torn away from its original site and become adherent to the omentum. In very rare instances the omentum contains a multitude of small cysts, possibly lymphatic.

8. **Ovarian Cysts.**—See OVARIES, Diseases of.

9. **Pancreatic Cysts.**—Under this heading are included cysts which clinically appear to arise in or from the pancreas. Cystic dilatation of the pancreatic duct may be due to obstruction and constitute a true pancreatic cyst. Cysts may possibly be due to hæmorrhage and inflammatory processes arising inside the pancreas, or in rare cases to cystic change in a new growth. These true pancreatic cysts may be single and large, multilocular and of considerable size, or multiple and small.

Probably a considerable proportion of what appear to be pancreatic cysts clinically are in reality peripancræatic cysts and due to localised peritonitis in the lesser sac of the peritoneum. The inflammatory process closes the foramen of Winslow and so leads to an encysted peritoneal effusion in the immediate neighbourhood of the pancreas. What the numerical relation of true pancreatic to peripancræatic cysts is it is difficult to say at the present time, but the belief that a large proportion of those requiring treatment are peripancræatic is gaining ground.

ÆTIOLOGY.—Injury, straining, and inflammation of the pancreas are the usual antecedent conditions.

SITUATION.—CONTENTS.—The cysts most often arise from the tail of the pancreas and may project below the stomach, above it, or push that viscus forwards. The cyst may reach a large size and contain many quarts of fluid. The fluid may be clear, but is more often dark brown from altered blood; it is alkaline, of a sp. gr. 1010–1020, and contains the fat-splitting, amylolytic, and proteolytic ferments of the pancreas; the presence of the last named is of most importance from a diagnostic point of view, inasmuch as the other two may be met with in effusions elsewhere.

CLINICAL FEATURES.—There is a tense tumour in the upper part of the abdomen, usually in the epigastrium or left hypochondrium. It is slightly movable, and, if the stomach be distended, can be shown to arise behind that organ. There may be no symptoms, but, as a rule, the patient suffers from attacks of pain with vomiting and collapse. Pressure-symptoms, when the cyst is large, may occur; jaundice is rare. Sometimes the progress is rapid, but more often the condition is chronic; considerable alteration in the size of the cyst, both enlargement and diminution, may be noted.

DIAGNOSIS between true and peripancræatic cysts is impossible until the anatomical conditions are examined, and even then may not be conclusive during life. Pancreatic cysts must be distinguished

from hydronephrosis on the left side, a dilated gall-bladder, hydatid cysts, and sometimes from large ovarian cysts.

PROGNOSIS.—If left alone the larger cysts may rupture into the general peritoneal cavity with fatal results.

TREATMENT.—Tapping the cyst with a trocar through the abdominal walls is a very dangerous proceeding and should never be countenanced. Laparotomy with incision and drainage of the cyst is the proper course unless it is found possible to remove it entirely.

10. **Retro-peritoneal Cysts.**—Other than pancreatic retro-peritoneal cysts are rare; they may be hydatid, derived from the remains of the Wolffian body, or pseudo-cysts due to softening of retro-peritoneal sarcoma or suprarenal growths. Retro-peritoneal collections of blood, chyle, or urine have been occasionally seen, usually after traumatism. The clinical features of retro-peritoneal cysts are those of a deep-seated tumour; they may easily be mistaken for a pancreatic cyst or a hydronephrosis.

Their *treatment* is removal by Laparotomy.

11. **Urachal Cysts.**—The urachus, that part of the allantois which runs in front of the peritoneum as a fibrous cord between the urinary bladder and the umbilicus, not uncommonly shows minute cystic dilatations. In rare instances large cysts develop; they occur in either sex. The cyst is situated in the middle line between the umbilicus and the pubes and in front of the peritoneum. A communication with the urinary bladder may exist. The walls of the cyst contain smooth muscular fibres—the expanded muscular walls of the urachus. The treatment of these cysts is complete removal, or, if this is impossible, incision and drainage.

Under the name Allantoic cysts, Lawson Tait and Robinson described a different condition occurring in women, which they ascribed to an abnormal process of development on the part of the allantois. Alban Doran, however, makes it clear that these so-called allantoic cysts are really encysted tubercular peritonitis, the infection spreading from the Fallopian tubes.

H. D. ROLLESTON.

ABDOMINAL WALLS, Diseases of.—Most of these are but local forms of diseases which are fully described in other parts of this work. The parietal peritoneum will be excluded from consideration, as its morbid conditions are treated of separately.

1. **SUPERFICIAL AFFECTIONS.**—*a.* The skin covering the abdomen may be the seat of various eruptions. The rash of typhoid fever is chiefly observed over this region. *b.* When the abdomen is greatly enlarged, its cutaneous covering becomes stretched and thinned, often presenting a shining appearance; or it may even give way, so that it exhibits superficial cracks or fissures. If it has been distended for a considerable time or on several occasions, as after repeated pregnancies, the skin becomes impaired in its nutrition, and is often the seat of permanent white lines or furrows—*lineæ albicantes*. In this connection allusion may be made to the umbilicus, which, in certain forms of distension of the abdomen, may become pouched out, everted, or actually obliterated. *c.* The superficial veins frequently become enlarged and tortuous,

when the return of the blood which is normally conveyed through them is in any way impeded. *d.* The cutaneous sensibility over the abdomen is sometimes materially altered. In certain nervous diseases it may become more or less impaired or lost; but the most important deviation is a marked increase of sensibility — *hyperæsthesia* — which is occasionally observed in hysterical females, and which may simulate more serious affections, particularly peritonitis, especially if it is accompanied with symptoms of much depression. This condition is characterised by extreme *superficial* sensibility or tenderness of the abdomen, the slightest touch being resented; but if the patient's attention can be taken off, and deep pressure be then made, this is borne with little or no indication of distress. The exact position of the tenderness, the aspect of the patient, the presence of other symptoms indicative of hysteria, and the absence of pyrexia usually serve to distinguish this affection from others of a graver nature. The surface of the abdomen may also be affected with neuralgia, which is sometimes very severe.

2. **SUBCUTANEOUS ACCUMULATIONS.**—*a.* The chief morbid condition coming under this head is *œdema* or *dropsy* of the subcutaneous tissue. This generally follows anasarca of the legs, and may be associated with ascites. The fluid tends to collect especially in the lower part of the abdominal walls and towards the flanks. The skin often presents a white pasty aspect; the abdomen may be more or less enlarged; the umbilicus appears depressed and sunken, if the *œdema* extends up to this level; the superficial structures pit on pressure, and yield the peculiar sensation of dropsical tissues; and the percussion-note is frequently muffled. *b.* The abdominal subcutaneous tissue is, in many persons, the seat of an abundant collection of fat, which may be important from causing apparent enlargement, and simulating or obscuring other more serious morbid conditions which enlarge the abdomen. *c.* In rare instances gas accumulates under the skin in this region, constituting *subcutaneous emphysema*. The abdomen then appears to be enlarged, but the condition is readily recognised by the peculiar crackling or crepitant sensation felt on digital pressure; the resonant or tympanitic sound elicited by percussion; and the dry crepitant sound heard when pressure is made with the stethoscope.

3. **AFFECTIONS OF THE MUSCLES AND APONEUROSSES.**—*a.* The abdominal walls may be the seat of muscular rheumatism, which is particularly likely to follow undue straining, such as that caused by violent coughing or vomiting. It is characterised by pain, sometimes severe, evidently located in the muscular and tendinous structures, accompanied with much soreness and tenderness. The affected parts are kept as much at rest as possible, and any action which disturbs them materially aggravates the pain. *b.* As the result of violent strain, the muscular or aponeurotic tissues may be more or less torn or ruptured, or the normal openings enlarged (separation of the recti). As a consequence a protrusion of some internal structure is likely to take place, forming a hernia. *c.* The abdominal muscles are liable to be the seat of spasmodic contraction, cramp, or rigidity. These disorders are not uncommonly excited in sympathy with grave disturbance of the alimentary canal, as in cholera. In certain painful internal affections, also, some of the abdominal muscles are occasionally kept in a

state of more or less rigid tension, as if they were involuntarily contracted in order to protect the diseased parts underneath from injury. The spasmodic contractions in tetanus not infrequently cause great suffering over the abdomen. *d.* On the other hand, the abdominal muscles are occasionally paralysed, as the result of central nervous disease. The movements of respiration are then altered in character; while the expulsive acts in which the abdominal muscles naturally take part are much interfered with.

4. **RELAXED ABDOMINAL WALLS.**—All the structures forming the walls of the abdomen are often in a relaxed and flabby state, yielding to any pressure from within, so that the abdomen becomes enlarged and prominent, especially if, as is frequently the case, this condition is associated with much flatulence. It materially weakens the act of defecation, and promotes constipation.

5. **INFLAMMATION AND ABSCESS.**—Local inflammation may be set up in any of the abdominal structures, and this may terminate in suppuration and the formation of an abscess. Purulent accumulations from within, as in cases of perityphlitis and suppuration in the gall-bladder, as well as certain abscesses originating in disease of bones or joints, may likewise extend among the tissues of the abdominal walls, causing thickening and induration, or may make their way outwards, directly or through a sinus. Subsequently permanent sinuses or fistulæ may be left.

6. The abdominal wall may be the seat of *extravasation of blood*; and various kinds of *tumour* or *new growth* may form in its structures.

FREDERICK T. ROBERTS.

ABORTION.—The act of abortion signifies the expulsion of the contents of the pregnant uterus before the seventh month of gestation. An abortion is a fœtus prematurely expelled. See MISCARRIAGE.

ABSCESS (*abscedo*, I depart).—SYNON.: Fr. *Abcès*; Ger. *Abscess*, *Eitergeschwulst*.

DEFINITION.—A circumscribed collection of pus. Circumscription is the characteristic feature which distinguishes 'abscess' from 'diffuse suppuration' (purulent infiltration), which will also be considered in this article.

VARIETIES.—Two varieties of abscess are described—the *acute* and the *chronic*, but there is little besides the name common to the two conditions.

A. Acute Abscess.—**ÆTIOLOGY** of Acute Suppuration.—Suppuration is the result of the action upon the tissues of certain irritant substances. The irritant must be of considerable intensity, must act continuously for some time, and must have the power of inhibiting the formation of fibrin or of dissolving any that is formed, together with small portions of tissue.

In the vast majority of cases acute suppuration is excited by the growth of micro-organisms in the tissues. Some microbes are only rarely associated with the formation of pus; others are constantly met with in this connection. The latter, along with such of the former as are not known to occur in other morbid conditions, are grouped together under the title of *pyogenic* organisms, since their usual, though not invariable, characteristic is the production of pus. Of these different species the *Staphylococcus pyogenes aureus* is that most often found; the *Staphylococcus pyogenes albus* is next in fre-

quency, followed by the *Streptococcus pyogenes* (figs. 1 and 2). The staphylococci of one or other variety seem to occur about four times as often as the streptococcus, while the two species are found mixed in 5 per cent. of all cases. Suppuration due to mixed infections is often very severe. Cases due to staphylococci are usually circumscribed and end in abscess, while the streptococcus is specially associated with diffuse processes, and is said to be the organism most commonly found in secondary pyæmic abscesses.

The *Staphylococcus aureus* forms in its growth a continuous supply of irritant substances, among

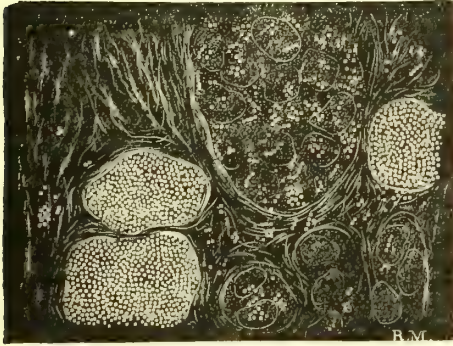


FIG. 1.—Micrococci in capillaries of kidney in Multiple Septic Abscess. Showing masses of cocci like *Staphylococcus pyogenes aureus*. $\times 800$ diam.



FIG. 2.—Streptococci of Suppuration.—Dried pus from an empyema of some duration. Showing chains of streptococci, some of which are swollen, probably from degenerative changes. $\times 800$ diam.

which ammonia and trimethylamine have been recognised; it also gives rise to a toxin (*phlogosin*), which can by itself excite suppuration. Moreover, it has the power of peptonising substances with which it is in contact, probably by means of an enzyme, and it can thus prevent the appearance of fibrin and of small sloughs in the fluid exudate. But this cannot be the sole means by which such a result is brought about, for the *Streptococcus pyogenes* is not known to peptonise; nor do many of the other germs less frequently associated with suppuration. Possibly leucocytes have peptic power.

The proof that these micro-organisms cause suppuration is absolute. With the exception of the rare

instances of aseptic suppuration, of which mention will subsequently be made, one or other is constantly present in pus; they have been cultivated through several generations, the resulting cultures have been inoculated upon animals and have caused suppuration, and from these lesions they have been recovered in pure culture. Finally they have been experimentally inoculated upon man, with similar results.

On the other hand, the mere presence of pus-organisms in the blood or tissues is not sufficient to excite suppuration, as is evident from cases of puerperal septicæmia and other forms of this disease, in which pyococci are eliminated with the milk, urine, &c., but no abscess is anywhere formed. Unless certain circumstances favourable to the cocci exist in an infected person, they die out or are eliminated. Until the pyococci were discovered, these predisposing circumstances were regarded as the causes of suppuration.

In the first place, to excite suppuration the cocci must come to rest in some spot. Then ensues a struggle between them and the tissues. Man is only moderately predisposed to suppuration: the predisposition doubtless varies with the individual and may be increased by depression of health from any cause, especially by certain diseases, e.g. diabetes, chronic renal disease, and acute fevers; and by chronic intoxication, such as alcoholism. The point at which the cocci come to rest appears to be an important factor, for all tissues do not seem equally susceptible. Local tissue-susceptibility may be increased by injury, by exposure to cold or wet, or by a previous attack of inflammation. The pyococci, on their side, must possess certain qualities, to enable them to prevail. They must be sufficiently virulent to overcome the resistance of the tissues in which they settle—and there are no organisms which seem more liable to attenuation by unfavourable environment—and they must be in sufficient numbers, for ‘resistance’ means that the tissues can dispose of a certain number of bacteria of a given virulence. They are also greatly favoured by circumstances which afford them protection from the healthy tissues until they can form, *in loco*, sufficient toxin to protect themselves. Such protection may, for example, be afforded by envelopment in a bit of blood-clot, which is often already saturated with their products.

The possibility of the occurrence of ‘aseptic’ suppuration, i.e. of the formation of pus from causes other than micro-organisms, has been much debated, but it is probable that it can be induced experimentally, and it may rarely occur clinically. Competent observers have failed to find bacteria in a few cases of suppurating hydatids and in cases of chancrous buboes which, when opened, have assumed the characters of soft chancres. Most observers regard the exudation induced by the injection of turpentine, croton oil, ammonia, and other substances as ‘pus;’ others decline to give it this name. Lastly the injection of some toxins (freed from germs)—such as phlogosin and cadaverin—or of the dead bodies of certain bacteria, e.g. boiled tubercle-bacilli (Koch), will induce suppuration. It is characteristic of such ‘suppuration’ that it soon ceases to extend.

HABITAT OF PYOGENIC GERMS.—These microbes are very widely diffused, being often found in dirty water and in dust. Staphylococci are common in the skin and frequently occur along with streptococci, in the mouth, pharynx, and vagina. Pyococci

are specially frequent upon the hands of those who have to deal with wounds.

MODE OF ENTRY INTO THE BODY.—Usually they enter through a lesion, possibly quite minute, of skin or mucosa, but they may enter through unbroken skin, being pressed into and growing up the orifices of glands (sebaceous, mammary). It appears probable that, like other particles, they may pass through uninjured mucous membranes. In a wound of any depth, the infection may affect primarily any layer of the tissues affected, and this may suppurate while the rest heals. From the point of entry the organisms may be carried by the lymph- or blood-stream, and thus deposited in distant parts. The disturbance at the point of entry may be very slight or nil.

RESULTS OF LOCAL INFECTION BY STAPHYLOCOCCUS AUREUS: FORMATION AND ANATOMY OF ACUTE ABSCESS.—When the cocci settle at a spot under conditions permitting their development, they begin to multiply and to excrete their irritants. The result is an inflammation which passes more or less rapidly through all the usual stages, dilatation of vessels with retardation of the blood-stream and margination and free migration of leucocytes being found in the immediate neighbourhood of the cocci, while the earlier stages are seen in the outer zone of the inflamed area. In highly cellular tissues (kidney, liver) a clear area of coagulation-necrosis often forms round the cocci; but in connective tissue this is not evident. In either case the cocci pass outwards from the point of inoculation and the leucocytes press inwards towards this point. It appears that the substances formed by the bacteria exert an attractive influence (*positive chemotaxis*) upon the leucocytes, which are thus drawn towards the seat of infection (*see* CHEMOTAXIS). Soon cocci and leucocytes become mixed up; many of the organisms are ingested by leucocytes and by swollen fixed cells, others remain free. The capillaries at the centre become thrombosed and together with the tissue-elements are peptonised and removed; their place is then occupied by fluid containing numerous leucocytes and cocci. So long as free cocci pass outwards—which they do chiefly along the lines of vessels and their accompanying lymphatics—spread of the inflammation and formation of fresh foci in the neighbourhood of the primary lesion continue. These secondary foci, as they enlarge, usually coalesce with the primary cavity. After a time the cocci are hemmed in by leucocytes, and granulation-tissue forms in which no micro-organisms are found. Enlargement of the abscess may, however, still go on under the influence of tension due to the increasing quantity of fluid poured into it, to interference with the circulation in the surrounding tissues, and to softening of these by the inflammatory exudation. Extension takes place in the direction of least resistance—towards a free surface when possible—but when pus forms beneath a dense fascia, which is traversed by few blood-vessels or lymphatics and is difficult to soften, it often burrows for a considerable distance before reaching the surface. The term ‘least resistance’ implies not only slighter mechanical obstacles but also facility of passage for cocci. Finally the fluid exudation lifts up the epidermis, which softens and gives way, and the abscess empties itself more or less completely. The elasticity of the tissues around at once causes great diminution of the size of the cavity, which rapidly becomes filled up with

granulation-tissue. This is gradually converted into fibrous tissue; and finally the epidermis grows over the surface and covers it.

CHARACTERS OF PUS.—The pus yielded by a healthy but not aseptic granulation-tissue was formerly spoken of as ‘healthy’ pus—*pus bonum et laudabile*. Its appearance indicated a local or general change for the better. Such pus is yellowish-white in colour, opaque, creamy in consistence, and neutral or alkaline in reaction, with sp. gr. 1030–1040. It has a faint sickly odour and does not putrefy readily. With an equal quantity of liquor potassæ it forms a gelatinous mass. On standing it separates into a deep sediment and a clear supernatant fluid (*liquor puris*). The latter is highly albuminous, does not coagulate spontaneously, and contains salts in about the same proportion as plasma, along with a certain quantity of peptones. The deposit consists of leucocytes, for the most part dead and fattily degenerated, with the addition of a varying number of red corpuscles which may render the pus *sanious* or give it merely a saffron or orange tinge from the presence of hæmoglobin. It also contains fine shreds of tissue, fatty and albuminous particles, and cocci, which are distinguished by their grouping and by appropriate methods of staining. The commonest departure from this type is that the pus becomes thin and acid, irritating to the tissues, and liable to rapid putrefaction (*ichor*). It sometimes has a very offensive odour, especially when the abscess has formed near the alimentary tract or vagina, owing to the presence of the *Bacillus coli*; but offensive pus in other situations, e.g. in the thigh, may be met with, and is then due to the action of the *Bacillus pyogenes fetidus*. Bluish or greenish pus is very rare and is due to the *Bacillus pyocyaneus*.

SYMPTOMS.—A fairly circumscribed area of the skin is found to be swollen, more or less red, hot, painful and tender; and these signs are all most marked towards the centre. Pressure with the finger tip or nail produces pitting owing to the presence of œdema. Fluctuation can be obtained by the method given below. As the abscess advances the centre of the affected area becomes more prominent, and fluctuation becomes more distinct: the red colour deepens into purple or blue, and the skin becomes progressively thinner. This sequence of events is known as the ‘pointing’ of an abscess. Finally the epidermis gives way and the contents of the abscess are discharged.

The method of obtaining fluctuation is as follows: the pulps of three fingers, if possible of each hand, are placed upon the swelling and kept as far from each other as the size of it permits. The fingers of one hand remain resting lightly on the surface, while those of the other hand gently compress the swelling. The fluid is thus driven beneath the resting fingers and raises them up. Experience is necessary to avoid mistakes: if the fingers are too close together, fat or œdema may give a sensation of fluctuation. It is never advisable to try to obtain fluctuation across a muscular belly, but always longitudinally in the direction of the fibres of any underlying muscle.

Constitutional symptoms vary greatly in nature and severity. Pyrexia varying from 100° to 104° F. is usually present, being generally proportionate to the acuteness of the inflammation, the amount of tension existing, the size of the abscess and its situation, absorption being more free from some parts, e.g.

knee, than from others. Not uncommonly the onset of suppuration is marked by a rigor, but 'chills' are more frequent: the rigor may be repeated more than once without any sign of pyæmia appearing. As the pus becomes surrounded by a wall of granulation-tissue the temperature falls; and it may even reach normal if the abscess remains long unopened.

The lymphatic glands above an abscess are often swollen and tender; lymphangitis occurs much less commonly.

Special symptoms arise in connection with pressure upon, or extension of œdema to, important parts.

In sub-fascial abscesses, e.g. in connection with acute necrosis of bone, the part swells diffusely and does not at first pit. If there be difficulty in localising the pus, it is advisable to look for the point of maximum swelling, and of maximum tenderness, and for any appearance of redness or œdema of the skin.

The discussion of abscesses occurring in special organs will be found under the heading of the respective parts involved.

DIAGNOSIS.—Diagnosis is usually easy. If an acute localised inflammation persists four or five days with the signs above mentioned well marked, pus is almost certainly present, even though fluctuation cannot be obtained. Some rapidly growing sarcomata have given rise to all the physical signs and symptoms of inflammation and have seemed to fluctuate: such cases are, however, very exceptional. Exploration may be necessary to determine the presence or absence of pus. The sacs of aneurysms sometimes suppurate, usually owing to some injury—either accidental or in the course of treatment. A history pointing to the existence of aneurysm is all-important in such cases. See ANEURYSM.

TERMINATION.—An acute abscess usually bursts through the surface of the skin and then heals. If it discharge on to a mucous surface, draining is often imperfect and serious septic absorption may occur. If it rupture on to a serous or synovial surface, diffuse inflammation of the cavity may be the result. Far more commonly, however, this last occurrence is prevented by the formation of adhesions round about the abscess before it bursts, so that in many cases, e.g. in the peritoneum, the pus crosses the cavity in safety and makes its way to the surface of the skin or into some hollow viscus. Absorption of an acute abscess is very rare: rarely, too, it becomes chronic. Delayed healing with the formation of an ulcer, sinus, or fistula is a common termination.

TREATMENT.—At first the ordinary treatment of inflammation (see INFLAMMATION) should be employed. As soon as pus is found or its existence strongly suspected, an incision should be made with a straight knife—not with a curved Syme's knife which inflicts the maximum of pain and makes relatively the smallest opening into the cavity. Except where it is important not to leave a scar, the opening should be free enough to drain, unaided, a small abscess, and to admit a finger to explore a large one. The finger should carefully seek out all 'pockets' and small-mouthed recesses, which should be well opened up into the main cavity. Formerly the formation of a dependent opening was considered necessary, but now it is more important to keep the opening away from any point from which infection is likely to occur. If this is avoided the discharge soon becomes thin and drains 'up hill' readily. Still, drainage should be thorough, and

in septic cases it may be necessary to make many incisions at various points. A wide incision or an 'anchored' tube, with a moist compressive dressing or fomentation, is the best means of draining away pus. The outer end of the tube should be kept flush with the surface of the skin, the deep end being shortened as necessary. When the discharge has fallen to a small amount, the tube should be removed experimentally, careful watch being kept for local discomfort or rise of temperature. Next day, or the day after, a probe should be passed along the track of the tube to see if pus has collected, and the tube replaced if necessary.

When it is wished to reach pus at some depth through a small wound, the skin and fascia may be cut through and a pair of closed sinus-forceps pushed in the direction of the cavity until resistance ceases; the forceps are then withdrawn with the blades open, a probe having been previously introduced along them as a guide (Hilton). Small vessels thus escape injury. But in cases of real danger to vessels or other important structures the wound should be large enough to allow the operator to work with a knife under guidance of sight.

B. Chronic Abscess.—**DEFINITION.**—A circumscribed collection of puriform fluid developing slowly with little or no sign of inflammation and with slight, if any, fever.

ÆTIOLOGY.—Although a very few acute abscesses quiet down and run a chronic course, and a few abscesses, due to staphylococci and other germs, run a chronic course throughout, the term '*chronic abscess*' practically means a softened 'tubercular focus.' The exciting cause is therefore the *Bacillus tuberculosis*: the proofs of this are absolute. Pre-disposing causes are an inherited tendency to tuberculosis; certain general depressing influences—such as faulty hygienic conditions, measles, and diabetes mellitus—which favour infection by this organism; and slight injuries which act similarly but locally.

MODE OF ENTRY AND OF SPREAD.—The tubercle-bacillus may gain access to the tissues (1) by inoculation upon a scratch or larger lesion of skin or mucosa (e. g. tonsil); (2) perhaps by passage through the uninjured respiratory or intestinal mucous membrane in company with other particles.

The bacilli may give rise to a tubercular ulcer at the point of inoculation; but it is common to find that before they have been able to cause superficial mischief they have been taken up by the lymph-stream and carried to the nearest glands, where they have been deposited. In such a case no history or sign of the lesion by which they entered can be discovered, as is frequently noted when the cervical or the mediastinal glands are the seat of infection. From the gland first reached they may be carried by lymphatics to many others. Less commonly the bacilli are arrested in the lymphatics leading from the point of entry to the glands. From any tubercular focus extension by the blood-path may occur: usually a tubercular phlebitis is excited, and the inflammatory deposit in the vessel-wall softens and breaks down, thus discharging bacilli into the vein. More rarely an artery is affected. It is conceivable that leucocytes may carry bacilli in small numbers into the circulation. On first entering the venous circulation the bacilli are carried to the lungs, and may even pass through the pulmonary capillaries and reach other parts of the body, where they are deposited. If they here encounter conditions un-

favourable to their growth, they will be destroyed or eliminated: but if they find a favourable soil they multiply and give rise to tubercular foci. Tuberculosis of bones, joints, testicles, kidneys, and other parts, removed from direct infection or infection through lymphatics, is brought about in this manner.

RESULT OF LOCAL INFECTION.—A tubercular infiltration slowly develops in which 'giant-cell systems' are usually discoverable. Fatty degeneration and softening of the inflammatory tissue soon take place (see CASEATION). Upon a free surface the result of this process is ulceration; but in the subcutaneous tissue or any deeper part, such softening gives rise to a 'chronic abscess.' Why softening occurs in some cases of tubercular infiltration and not in others is unknown.

ANATOMY OF CHRONIC ABSCESS.—If a small subcutaneous abscess be cut out along with its surrounding fat, it will present on section a central cavity containing puriform material limited by walls which are ragged and irregular. To the naked eye the wall seems to consist of granulation-tissue which becomes opaque and yellowish towards the cavity, and more or less fibrous towards the surrounding fat. Here and there between the fat-bubbles small foci of granulation-tissue may be seen. Starting from the periphery the microscope shows round-celled infiltration among the fat with more or less tendency to the formation of a fibrous capsule. Passing inwards the round-celled infiltration becomes denser, and soon 'giant-cell systems' appear embedded in it in greater or less numbers. Still nearer to the centre patches of fatty degeneration are noticed, and this soon becomes uniform and universal as the cavity is approached. No structure is seen—only fatty granules which seem to be dropping into the cavity when the lining layer is reached. Outlying nodules of granulation-tissue show similar tubercular structure. They are due to the passage of bacilli along a lymph-path from the primary focus, and exhibit the same series of changes. They gradually enlarge and ultimately coalesce with the primary focus. Spreading in this way and exercising harmful pressure on the vessels supplying the surface, a chronic abscess renders the skin over it thin and purple. Finally this gives way and the contents are more or less completely discharged. When a tubercular focus in bone softens—e.g. in a lumbar vertebral body—the 'pus' slowly makes its way to the surface of the bone. Here it may form a collection under the periosteum and extend along the line of least resistance. Thus it may pass back into the loin along the lumbar vessels (lumbar abscess), or escape from the bone into a process of origin of the psoas muscle on one or both sides. In the latter case, the pressure of the increasing exudation causes atrophy of the muscle-fibres, while the fibrous tissue is thickened and forms a dense sheath for the pus. The cavity thus formed becomes lined by a layer of tubercular granulation-tissue, poorly supplied with vessels and very loosely attached. Vessels of considerable size and nerves may persist, stretching across the cavity as isolated strands. Such an abscess not uncommonly raises the iliac fascia and fills the iliac fossa (*iliac abscess*) before it passes beneath Poupart's ligament (*psoas abscess*) to pass down the thigh along the profunda artery or back along the internal circumflex vessels. These are types of chronic abscesses. In all, the walls are tuberculous, and until this lining is removed or cast off, healing will not occur.

CHARACTERS OF THE 'PUS.'—The fluid in these abscesses is thin and watery. It often contains small yellowish-white masses like curds, and sometimes small calcareous or bony particles. Under the microscope fatty granules and degenerated crumbling leucocytes are seen. Hardly any normal white corpuscles are to be found.

SEATS.—The commonest seats of chronic abscess are the subcutaneous tissue, especially in children; the lymphatic glands; cancellous bones such as the vertebrae, the epiphyses, and the ribs; the synovial membranes of joints; and the epididymis.

SYMPTOMS.—Symptoms are often absent until a swelling is accidentally noticed. The swelling is smooth and rounded; it is painless, and fluctuation is readily obtained. The skin over it is not tender, red, hot, or cedematous until the abscess is on the point of bursting. There is often no thickening at its margin, but sometimes a ring of infiltration is felt. When the lesion originates in a bone, the base of the abscess is naturally fixed. When a collection of tuberculous matter reaches the surface from the thoracic or abdominal cavity, the swelling is not uncommonly stated by patients to have appeared suddenly. Coughing causes such an abscess to swell up and to impart an impulse to the hand laid upon it; but this phenomenon does not occur invariably. The erectors spinæ, being expiratory muscles, seem to yield an impulse on coughing.

More than one chronic abscess may be met with in the same patient: sometimes a large number may co-exist. Little or no fever accompanies the process. Sometimes an abscess may press upon a nerve and cause pain—which may even be severe; but pain is usually a symptom of the disease in the bone or joint whence the abscess springs. The symptoms of such disease are superadded to those of the abscess, and must be carefully investigated in order to determine the source of the 'pus.'

The patients are usually children or young adults, though no age is exempt. A history or physical evidence of tuberculosis is often obtainable. The general condition may leave nothing to be desired, but often it is poor. The presence of considerable fever with an unopened or aseptically opened chronic abscess indicates the existence of some complication, perhaps general tuberculosis.

COURSE OF A CHRONIC ABSCESS.—When not interfered with a chronic abscess may occasionally become absorbed: usually it enlarges more or less rapidly, and may reach enormous dimensions. Ultimately the skin over it at some spot becomes thin, red, or bluish in colour, and eventually gives way. The contents escape imperfectly; infection of the cavity by pyogenic and septic germs occurs; and the patient may die of acute septicæmia. In other cases hectic fever ensues, and in spite of the establishment of counter-openings, the abscess burrows and appears at fresh spots. Amyloid disease of viscera follows, and, if recovery occurs, it is with a ruined constitution. Such is the course of a large spinal abscess untreated or treated unsuccessfully as regards asepsis. Even when such an abscess heals, tuberculous tissue locked up in the deep scar often infects the surrounding parts afresh, and recurrence (*residual abscess*) sooner or later occurs. Smaller abscesses, and those not connected with bone, heal more readily even under such conditions.

DIAGNOSIS.—Effusions of blood, serous cysts, herniæ, fatty tumours and soft sarcomata, and some-

times firmer, deeply seated, malignant growths have all to be carefully differentiated from Chronic Abscess. Exploratory puncture or operation must be undertaken as a last resort. The diagnosis of 'tumour' should not be made until inflammation has been eliminated.

Sometimes in cases of spinal disease with psoas abscess the hip-joint is persistently flexed, and pain is caused by any attempt to extend it; probably, the psoas-sheath is filled unusually tightly and nerves are pressed upon. Disease of the hip-joint, which may cause an abscess above Poupart's ligament, is thus simulated, but may be eliminated by noting that the outline of the trochanter is in no way blurred, and that movements of the hip other than extension are free and painless. Signs of spinal disease are usually, but not always, discoverable. The impulse on coughing which is communicated to a collection of fluid in communication with the thoracic or abdominal cavity has been already alluded to.

PROGNOSIS.—The prognosis will vary with the number and size of the abscesses, their source, the age, health, and means of the patient, and the condition of sepsis or asepsis existing in the cavity. An origin from bone or joint is more serious than from glands and soft parts. The older the patient, after childhood, the worse is the prognosis.

TREATMENT.—The following are the chief therapeutical indications. Disease of spine or joint must be carefully attended to. Everything possible should be done to secure fresh air and sunlight, and good nourishing food for the patient. Cod-liver oil is generally valuable in these cases, and the use of an antiseptic internally, such as creasote increased drop by drop to large doses, is recommended by some authorities. With regard to the abscess, whatever operative treatment is adopted should be conducted with scrupulous attention to asepsis.

1. Aspiration, generally repeated more than once, has occasionally been followed by drying up of the abscess. Usually, however, it fails, and may result in the formation of a fistula or give origin to a septic infection of the cavity. Further, even when it succeeds, it leaves all the tuberculous tissue behind. Sometimes aspiration has been combined with irrigation with some antiseptic solution, or with the introduction of iodoform. The needle should always be introduced through a considerable thickness of tissue—not through the thinnest point: in the latter case infection of the track and the formation of a sinus will inevitably result.

2. Excision of the abscess with some healthy tissue around it is the ideal treatment. It can be practised in subcutaneous and glandular foci, and in abscesses connected with some bones and joints—the chisel or gouge being used to remove infected portions of the bone. The wound is to be completely closed and allowed to heal like any other aseptic wound.

3. When excision is impossible, the cavity should be laid open fully; as much as possible of the wall should be excised, and the rest should be carefully scraped with a spoon and rubbed with a dab, after which pure carbolic acid may be lightly applied, followed by a little iodoform. The wound should be closed without drainage; but if this cannot be done it should be plugged with iodoform-gauze.

4. In psoas and similar abscesses, practically no excision can be done. Incisions must then be

made in the loin, groin, and thigh, as may be convenient, in order to allow the lining membrane to be scraped away from the whole cavity, which is kept flushed meanwhile with saline solution. As a rule, even in the lumbar operation for lumbar disease, it is impossible to find the track by which the cavity communicates with the diseased vertebra, but if this can be found, there need be no hesitation in scraping it also. Iodoform-emulsion should be introduced into the cavity and the wounds closed. If after three or four days the cavity is distended with fluid, it should be aspirated. The fluid should somewhat resemble honey and smell strongly of iodoform. If left to accumulate it sometimes bursts through the stitches.

5. When the cavity is septic, too free drainage cannot be provided. Plugging from the very bottom with iodoform-gauze is best. Failing this, large tubes may be introduced through suitable openings, and irrigation practised with either sterile saline solution or iodine-lotion (one drachm of the tincture to one pint of water).

The tubes should not be abandoned nor smaller sizes substituted until it is certain that they have no more work to do. The openings must be kept dilated.

Bad cases of this kind require the exercise of the greatest patience. Good hygiene and careful feeding are very important; also fixation of the spine or any joint which may be affected. The discharge should be regular in amount, not varying in quantity from day to day. Rise of temperature may be due to an unusual amount of septic absorption, especially after rough treatment of the granulations; to retention of discharge or formation of some fresh pocket; to general tuberculosis; or to some accidental cause. It is an indication for a careful examination of the patient with a view to removal of the faulty condition. No avoidable operation should be done until the cause of the fever is determined.

C. Acute Diffuse Suppuration.—The subcutaneous tissue, especially that of the upper limb, is the most common seat of acute diffuse suppuration.

ÆTIOLOGY.—The principal exciting causes of this condition are: (1) *Extravasated urine*, especially if it be septic; (2) the *Streptococcus pyogenes*; (3) the virus introduced in *dissection-wounds*. This last is probably also due to the streptococcus, which is the common cause of ordinary diffuse cellulitis. The lesion of the surface, through which infection occurs, is generally easy to discover, but a few cases appear to arise spontaneously; these may possibly be due to organisms which have entered at distant points of injury or have passed through uninjured mucous membranes, and have been deposited in the infected area by the blood-stream. Slight wounds of the hands, especially those occurring in dissectors, surgeons, butchers, fishermen, &c., are specially liable to be followed by this complication; so also are lesions of the tonsils and of the cervix uteri, and all infected wounds which pass through the deep fasciæ, e.g. that of the scalp.

MORBID ANATOMY.—The affected part is diffusely swollen; the epidermis is often raised into bullæ here and there by effusion of fluid beneath it, and may separate, leaving grey or yellow patches of denuded corium exposed. Irregular openings in the skin may exist, through which pus escapes and sloughs can be seen. The fat is opaque and of a pale-yellow colour. The structure of the tissues is

rendered cloudy and indistinct owing to infiltration with a turbid fluid, which here and there forms collections of thin pus. Pieces of fascia and connective tissue, which have undergone necrosis and separation, are visible as dirty yellowish masses, resembling sodden wash-leather. The process may be confined to a comparatively small part, or it may involve a whole limb or even more. It may remain superficial to the deep fascia, or extend deeply between the muscles, leading to sloughing of tendons or even of the muscular substance. In rare cases it may lead to suppuration of joints, or necrosis of bones. The veins contain softening thrombi, and portions of such clots may be carried away by the blood-stream and give rise to secondary pyæmic foci. Streptococci are plentiful in the pus, the lymph-paths and the softening blood-clot. The disease may be set up at a distance from the point of infection: thus the axilla may be the seat of diffuse suppuration as the result of a pricked finger.

SYMPTOMS.—The onset is marked by all the usual signs of inflammation, which extend over a wider area than in cases of simple abscess and show no tendency to circumscription. The fever is more severe, and the occurrence of one or more rigors is not uncommon. As the disease progresses the red colour of the skin becomes deeper at certain spots, and a finger passed over the surface tends to sink in a little at these points. This 'bogginess' or localised softening is the best sign of the presence of collections of pus. Fluctuation may be nowhere distinct until great damage has been done, or it may seem present everywhere on account of the great oedema. As the process extends, fresh spots of softening appear; then the skin gives way and pus escapes. Sloughs protrude from the openings, which rapidly enlarge; and extensive tracts of skin are undermined and may undergo necrosis. Fever, suffering, and repeated incisions tell severely on the patient; and in bad cases septicæmia or pyæmia may ensue and prove fatal, or a condition of hectic fever may be established and threaten a fatal termination. The sloughs are slow in separating, and if important tendons are involved, the question will arise whether the part, even if it can be saved, will be subsequently of any value, since the process of healing necessarily results in a matting together of the parts by a mass of dense fibrous tissue.

DIAGNOSIS.—In cases of acute diffuse inflammation of a part it is often impossible to recognise at once the existence of suppuration (*see* CELLULITIS). In such instances exploratory incisions may be necessary. Thus in axillary and cervical cellulitis (*Angina Ludovici*), and in other cases of deeply seated inflammation, the local swelling and the fever not only justify but necessitate incision, since the patient may die before fluctuation is obtained. No distinction can be maintained between acute diffuse suppuration and the condition known as cellulotaneous erysipelas.

TREATMENT.—It is advisable to immediately disinfect the point of inoculation, when this is distinguishable, with pure carbolic acid, and to adopt the ordinary methods of treating inflammation. Elevation of the affected part is especially necessary. If these measures prove ineffectual, the part must be freely incised, but the deep fascia should not be opened up, unless the disease has already invaded parts lying beneath it. All bleeding should be care-

fully arrested. Sloughs must be removed and collections of pus drained. Pure carbolic acid is to be applied to the dried exposed surfaces. The wounds are then plugged lightly with iodoform-gauze, and fomentations are applied when all danger of bleeding has ceased. It is useful in freely suppurating cases to keep the limb in a warm bath of boric-acid lotion for some hours every day. When granulation occurs, movements of all damaged parts should be regularly practised. Careful feeding is most important, especially as the pyrexia abates, and a moderate amount of stimulants may be beneficial. When a part is hopelessly injured or life is threatened by prolonged suppuration, resort must be had to amputation or excision of the seat of disease.

STANLEY BOYD.

ABSCESS, Subdiaphragmatic.—**SYNON.** : *Subphrenic Abscess.* *See* SUBDIAPHRAGMATIC ABSCESS.

ABSINTHISM.—**DEFINITION.**—The condition induced by an excessive consumption of absinthe.

From the mode in which absinthe is taken, we should expect that the symptoms induced by its excessive consumption would be generally obscured by, and intermixed with, those of alcohol (*see* ALCOHOLISM). That it has a special effect on the organism, and that this may be diagnosed from alcoholism, is, however, generally recognised. In persistent absinthe-drinkers, vertigo and epileptiform convulsions are marked symptoms, and come on much earlier than when alcohol, in other forms, is habitually drunk. Hallucinations occur also without any other symptom of delirium tremens; and, when tremors coexist, these are limited more particularly to the muscles of the arms, hands, and shoulders, since absinthe acts chiefly on the cervical portion of the spinal cord.

JOHN CURNOW.

ABSORBENT AGENTS.—In *surgery*, absorbents are substances used to absorb fluids, as cotton-wool, lint, or compressed moss; in *medicine*, the term is made use of to designate remedies, such as the preparations of mercury and iodine, which are believed to possess the property of promoting the absorption of morbid products.

ABSORBENT VESSELS and GLANDS, Diseases of.—*See* LYMPHATIC SYSTEM, Diseases of; BRONCHIAL GLANDS, Diseases of; and MES-ENTERIC GLANDS, Diseases of.

ACANTHOSIS.—This term, originally used by Auspitz to signify diseases of the skin produced by anomalies in the spinous or prickle-cell layer of the epidermis, has been adopted more recently by Unna to specify proliferation of the prickle-cell layer as the result of mitotic division of its cells.

JAMES GALLOWAY.

ACANTHOSIS NIGRICANS.—This rare affection of the skin was first definitely recognised in 1890.

Patients affected by this disease present an exceedingly characteristic appearance. When the disease is fully developed certain regions of the body show an increase of pigmentation, giving rise to a brownish or even black colour. At the same time, the surface of the skin becomes rough, and this roughness may be so exaggerated as to give rise to papillary excrescences as large as, or even larger than, ordinary warts. The axillæ, the genito-crural region, the neck, the flexor surfaces of the joints, the face and mammary regions, are usually affected symmetrically.

In addition to these areas, any part of the surface of the body may show increase of pigmentation, and frequently, instead of actual warty or papillary excrescences on the surface, an exaggeration of the normal lines of the skin is noted. The mucous membranes may also be affected. See ADDISON'S DISEASE.

Histological examination of the skin shows that the main lesions occur in the epidermis. There is increase in the thickness of the prickly-cell layers of the epidermis—the condition which gives the name to this disease—the stratum granulosum is retained intact, and the horny layer is hypertrophied. In consequence of this over-growth of the epidermis, the interpapillary processes are elongated and hypertrophied, and coincidentally the papillæ themselves are elongated or irregularly enlarged. The pigmentation which gives such a striking colour to the patient consists of an exaggeration of the normal pigment, and its granules are contained in the basal and one or two of the overlying layers of the stratum mucosum.

A large number of the described cases of the disease have been associated with various forms of abdominal tumour, the majority of which have been of malignant character. Beyond this very striking coincidence, no suggestion has yet been obtained as to the relationships of the disease. It may commence at any period of life, but more usually develops in adults. Cases are, however, recorded commencing at two, three, and eleven years of age. The duration of the malady is indefinite, and the course is that of a gradually increasing dystrophy of the skin. If a malignant tumour is associated with the disease the prognosis depends upon the character of the growth.

TREATMENT appears to have no effect whatever in arresting the disease. Care must be taken to observe *special cleanliness* of the skin, especially in the groins, axillæ, and similar regions. Occasionally anti-pruritic lotions may be required. It is sometimes necessary to remove the papillomata if they become troublesome on account of their situation or their size, and this may be done by excision or by the cautery.

JAMES GALLOWAY.

ACARUS.—*Acar*i or *mites* constitute the tracheal order of the class *Arachnida*, several species of which are parasitic. The *Acarus scabiei* or *Sarcoptes hominis*, and the *Acarus folliculorum*, or more properly the *Demodex folliculorum* or *Steatozon folliculorum*, are the only human parasites belonging to this family.

DESCRIPTION.—1. The *Acarus Scabiei* is a small roundish animal, just visible to the naked eye. Examined under the microscope it is seen to be flattened, and to resemble a tortoise in shape; when fully developed it has eight legs. Over its body are scattered a few setæ, and on the dorsal surface are several rows of triangular spines (see fig. 1). The female is larger than the male, and is provided with terminal suckers on the four anterior legs, while setæ occupy a similar position on the posterior ones: in the male, however, the two posterior median legs have suckers like those on its fore limbs. The *young acarus* has only six legs, the two hindmost ones, which are distinctive of the sex, being wanting; it acquires these after shedding its skin two or three times. The male *acarus* lives near the surface of the skin, while the female burrows within the middle horny layer, and deposits

from ten to fifteen eggs in the cuniculus or burrow; these eggs hatch in about a fortnight. The young *acar*i escape from the burrow, but the parent does not leave it and dies when she has finished laying eggs. The *Acarus scabiei* is the cause of the skin-affection termed *Scabies* or *Itch*. See SCABIES.

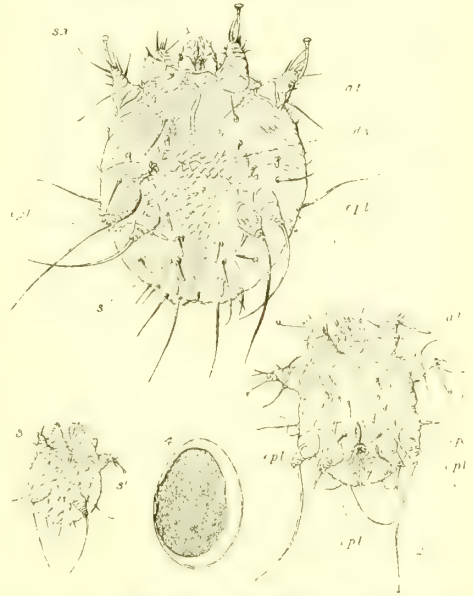


FIG. 1.—*Acarus scabiei*. 1, female; 2, male; 3, ovum; 4, anterior legs terminating in stalked suckers (s.s.); a.l., posterior legs terminating in baristles; i.p.l., posterior median legs of male terminating in stalked suckers; d.s., dorsal spines; s., setæ; s.p., support for penis. $\times 90$. (From 'Green's Pathology'.)

2. The *Acarus folliculorum* is a very minute parasite found in the sebaceous glands of the face, ear, and trunk. In this animal the head is continuous with the thorax, and to the latter are attached eight very short legs, each armed with three strong claws. On each side of the head are short jointed pedopalmi. The abdomen varies in length from twice to three or four times that of the thorax; it is pointed at its distal extremity. The presence of this parasite in the skin is unimportant. It is occasionally associated with a peculiar discoloration.

ROBERT LIVEING.

ACCOMMODATION, Disorders of.—See VISION, Disorders of.

ACEPHALOCYST (ἀ, priv.; κεφαλή, a head; and κύστις, a bag).—A hydatid cyst without any scolices in its interior. See ENTOZOA—Hydatids.

ACHOLIA (ἀ, priv.; and χολή, bile).—Absence or deficiency of bile. See BILE, Disorders of.

ACHONDROPLASIA (ἀ, priv.; χόνδρος, cartilage; πλάσσω, I form).—SYNON.: *Fætal Rickets*; *Fætal Crétinism*; *Chondrodystrophia fætal*.

DEFINITION.—A disease of fœtal life, causing stunted growth of the limbs and consequent dwarfing.

PATHOLOGY.—Longitudinal growth ceases in the diaphyses of certain bones, which develop from cartilage and ossify early: those principally affected

are the long bones of the limbs, the ribs, the innominate bones, and those forming the base of the skull. The cause of the condition is unknown. It is probably unconnected with any failure of thyroid-secretion or with ordinary rickets. The morbid process is confined to the early months (3rd to 6th) of foetal life; it appears to consist in a defective formation of the cartilaginous precursor of the bone, and is no longer active at birth. Abortion frequently occurs when the foetus suffers from this disease, but in those which survive the general health and intelligence are good.

CLINICAL CHARACTERS.—The arms and legs are abnormally short: the fingers may not reach below the crest of the ilium, and the legs may be so stunted that the patient sits down on the ground and rises again without apparent effort. The epiphyses are enlarged, the long bones curved and the fingers divergent, the angle between the middle and ring fingers being specially marked. The bridge of the nose is depressed and the base of the skull generally contracted, so that the cranium above appears unduly large. The body is of normal length, but the total height of an achondroplastic adult may be little over four feet. The gait is waddling, with lumbar lordosis; and the patients are helpless owing to the shortness of the arms, which causes inability to wash, dress, and attend to themselves. The disease is occasionally confounded with cretinism, but the deformity is really quite different, and the intelligence unaffected.

W. CECIL BOSANQUET.

ACHOR } ($\acute{\alpha}\chi\acute{\omega\rho}$, scurf or dandruff,
ACHORION } $\acute{\alpha}\chi\upsilon\rho\acute{\omega}\nu$, chaff).

The word Achor is now almost obsolete; it was formerly applied to a small follicular pustule of the scalp.

Achorion is the term assigned to one of the three principal dermatophytes or epiphytes of the skin. It constitutes the crust of Favus and belongs to the group of fungoid plants denominated *Oidium*.

As *Achorion* was the first discovered of the cutaneous epiphytes, in compliment to one of its early observers, Schönlein, it has been named *Achorion Schönleinitii*. It is the agent of contagion in favus (see FAVUS), but it is also found in *Onychomycosis*, a condition in which the matrix of the nail is invaded.

JOHN HAROLD.

ACHROMA ($\acute{\alpha}$, priv.; and $\chi\rho\acute{\omega}\mu\alpha$, colour).—Absence of colour; an achromatous or colourless state of a usually coloured tissue, due to absence of pigment. In reference to the skin achroma is synonymous with leucoderma, albinism, and aliphsis. See PIGMENTARY DISEASES OF THE SKIN.

ACHROMATOPSIA ($\acute{\alpha}$, priv.; $\chi\rho\acute{\omega}\mu\alpha$, colour; and $\acute{\omega}\psi$, sight).—More or less complete inability to distinguish colours from each other. See VISION, Disorders of.

ACIDITY.—The term acidity is used in medicine (1) in the ordinary sense to denote that a fluid possesses an acid reaction; (2) to indicate hyper-acidity, as in the expression 'acidity of the stomach;' or (3) to mean diminished alkalinity although the reaction may not become acid, as in the phrase 'acidity of the blood.'

Most of the fluids of the body give a variable result with different indicators, because their

reaction is due to weak acids such as carbonic acid, or to acid salts. Thus, saliva is alkaline to litmus but usually acid to phenol-phthalein. This varying reaction is capable of yielding a certain amount of information as to the character of the dissolved salts causing the reaction. By the use of very stable indicators it may also be determined whether an acid reaction is due to a free acid or to an acid salt; and also within certain limits whether the reaction is due to a strong (mineral) acid or to a weak (organic) acid. It is on this principle that all the many 'colour-reactions' for determining the presence or absence of free hydrochloric acid in the stomach are based.

Even those fluids in the body which are commonly regarded as alkaline, because of their giving an alkaline reaction with organic indicators such as litmus, contain acid salts such as sodium bicarbonate and di-sodic phosphate in addition to excess of carbonic acid, and hence in this sense may be spoken of as possessing acidity. In such fluids the acidity, as well as the alkalinity, can accordingly be determined and its variation under morbid conditions studied; this has been done particularly in the case of the blood.

1. ACIDITY OF THE BLOOD.—The acidity of the blood becomes increased in the sense defined above when there is either an increased amount of incompletely oxidised organic substances of an acid character formed by the tissues, or a less perfect elimination of such substances by the kidneys. Accordingly, a marked increase of acidity is found in fever and in diabetic coma; for example, Kraus states that the acidity of normal blood is equivalent to (i.e. capable of neutralising) 0.162 to 0.232 grm. of sodium hydrate per 100 grm. of blood, while under febrile conditions it may be increased to 0.272 grm. and in diabetic coma to 0.347 grm. Other observers have even detected an acid reaction of the blood to litmus in diabetic coma and in the cold stage of cholera. Increased acidity of the blood is also found in gouty and rheumatoid conditions, and occasionally in eczema and other skin-affections.

2. ACIDITY OF THE URINE.—The urine of persons fed upon a mixed diet, when collected over a period of twenty-four hours, has normally an acid reaction to litmus, but when tested with those more stable indicators which are only changed by free acid (such as methyl-orange or tropaeolin), no acid reaction is shown. This demonstrates that the acidity of urine is due to acid salts, and quantitative estimation further proves that the acidity and degree of acidity to such indicators as litmus is due to a variable mixture of mono-sodium and di-sodium phosphates. The former of these salts has an acid reaction to litmus, and the latter an alkaline one, and the reaction of any variable mixture of the two, such as is excreted by the kidneys, depends in kind and amount upon the total quantity and the ratio of the two salts in the urine.

Since one of the most important functions of the kidneys is to maintain a constant degree of alkalinity of the blood, it follows that the degree of acidity or alkalinity of the urine must vary directly with that of the blood, and indeed can be taken as an index of the condition of that fluid as to reaction. An increased alkalinity of the blood from any cause leads to the excretion of a less acid, or it may be an alkaline, urine, in order that the alkalinity of the blood may be reduced to normal. As an example

of this may be taken the so-called 'alkaline tide' met with in the urine during the first period of digestion after a meal. Here the secretion of acid gastric juice raises the alkalinity of the blood, and accordingly the kidneys excrete more alkali from it, so giving rise to a more feebly acid or it may be alkaline urine. At a later period, the alkaline juices poured into the intestine are separated from the circulating blood while the separation of hydrochloric acid ceases. Accordingly the alkalinity of the blood falls below normal and the kidneys secrete a hyper-acid urine in order to compensate. The character of the food also tends to alter the degree of alkalinity of the blood, and this tendency is compensated by corresponding variations in the urinary excretion. It is for this reason that the urine of herbivorous animals is normally alkaline, for during inanition their urine becomes acid. So also in man the reaction of the urine can be made alkaline, or its acidity reduced, by a diet of green vegetables. The alkali in such green food is present in the form of alkaline salts of organic acids, and when these acids undergo oxidation in the body, the bases become combined as carbonates which raise, or tend to raise, the alkalinity of the blood. This may be shown by administration of such salts as sodium or potassium citrate, tartrate, or acetate, or of the carbonates or bicarbonates of the alkalis. By such means the alkalinity of the blood is increased, and the acidity of the urine diminished or even replaced by an alkaline reaction.

An excessive meat-diet, on the other hand, increases the acidity of both blood and urine by giving rise to incompletely oxidised organic acids, such as uric acid, which cannot be excreted as such, and which therefore combine with a certain amount of the alkali of the blood, so reducing the degree of alkalinity. The sulphur and phosphorus of proteid are also oxidised in the body to sulphates and phosphates respectively, and this removal of alkalis in combination also tends to diminish the alkalinity of the blood. This process may be carried so far that the alkali is reduced to the level at which it no longer reacts readily with weak organic acids to form salts, and in the case of uric acid, which is almost insoluble in the free form, this may lead to its deposition in the tissues or in the urine. In this way either a uric acid diathesis, gout, or urinary calculi may originate. Since the primary cause is incomplete proteid-oxidation, the first thing indicated is a diminution of the patient's 'luxus-supply' of proteid, along with exercise, and a liberal diet of green vegetable food. These measures should be accompanied by the moderate use of alkalis until the crisis has been relieved. Since the lithium-salts of uric acid are the most soluble, lithium carbonate or lithium citrate is the form of alkali usually administered.

It is remarkable that in man and in the carnivora the acidity of the urine is not increased by the administration of even the mineral acids, except when the dose is so large as to lead to violent intoxication. Before acid intoxication takes place considerable amounts of the stronger mineral acids can be neutralised by the formation of ammonium salts, a corresponding decrease in the amount of urea excreted being observed. In herbivora, which are not subjected from the nature of their food to any danger of acid intoxication, it is interesting to observe that no such protective compensation takes place, and such animals quickly

succumb to comparatively small doses of mineral acids.

It is easy to deduce from the above statements that the *character of the food* is the primary factor in influencing the degree of alkalinity of the blood and the acidity of the urine. A diet too rich in proteid food, and especially animal proteid, leads to a diminished alkalinity of the blood, and the secretion of a hyperacid urine; while a diet rich in green vegetable matter has an opposite effect.

The reaction of the urine may change under pathological conditions from acid to alkaline, after it has been excreted by the kidney and before it is voided, because of bacterial fermentation set up in the urinary bladder or passages. Occasionally the alkalinity may be due to the secretion of alkaline mucus as a result of irritation of the mucous membrane. Fermentation is also often set up in cases of retention or incontinence of the urine, and the infection by the *Micrococcus ureæ* may occur either naturally by the urethra or as a result of careless catheterisation. The alkalinity is accompanied by cystitis which still further favours continued infection.

3. ACIDITY OF GASTRIC JUICE.—The gastric juice is the only fluid in the body which owes its acidity to free acid. It has been clearly shown that practically all the acidity of pure gastric juice unmixed with food is due to hydrochloric acid. During digestion of carbohydrates, lactic and other organic acids are formed by bacterial action, and under certain conditions favouring fermentation, such, for example, as chronic dyspepsia accompanied by distension of the stomach, the amount of organic acid formed may be very considerable and lead to gastric catarrh.

The percentage of hydrochloric acid varies considerably under morbid conditions. There is usually hyperacidity accompanying gastric ulcer, and this condition is also common in nervous dyspepsia. In the latter condition the acidity may be doubled, but the amount is variable, and there may often be either a normal amount or even sub-acidity. In both acute and chronic gastritis the amount of hydrochloric acid is subnormal and the organic acids are increased. In chronic catarrh, hydrochloric acid may be completely absent, and this is the usual condition in cancer of the stomach, where absence of the acid is an important, though by no means infallible, diagnostic sign (*see* STOMACH, New Growths of). Hydrochloric acid is stated also to be occasionally absent in hysteria and tabes dorsalis.

4. ACIDITY OF THE INTESTINE.—There is at the present time considerable difference of opinion as to the reaction of the intestine. The older view that the acidity of the chyme escaping from the stomach is neutralised, and the reaction changed to alkaline in the duodenum by the alkali of the digestive juices with which it is there mixed, has been controverted by recent observers. It is maintained by some of these authorities that the hydrochloric acid is never completely neutralised in the intestine, while others hold that the reaction of the intestine is normally acid owing to the presence of organic acids formed by bacterial action. According to the latter view bacterial action in the small intestine takes, under quite normal conditions, a considerable share in the digestion of carbohydrates.

The writer's own experiments have led him to regard the older view as to the reaction of the contents of the *small intestine* as more correct than

these modern statements. These experiments have shown that the degree of alkalinity of the intestine varies with the nature of the food, being decreased by a meat-diet, and increased by a carbohydrate-diet; further, that the alkalinity increases with the distance along the intestine, and that there is practically never an acid reaction to any indicator except phenol-phthalein in the lower portion. This last fact indicates that the alkalinity is due to acid salts of the alkalis, such as bicarbonates and bibasic phosphates. The reaction of the *large intestine* is variable, but most commonly acid, owing to the presence of organic acids formed as a result of bacterial action.

5. ACIDITY OF THE SWEAT.—The reaction of the sweat in man is normally slightly acid, even when it is collected from parts free of hair so as to be uncontaminated by sebaceous secretion. The acidity is due to volatile organic acids. In long-continued profuse sweating it is stated that the reaction may change to alkaline. *See* ALKALINITY.

BENJAMIN MOORE.

ACIDS.—**DEFINITION.**—Substances which combine with alkalis, and destroy their power of turning red litmus-paper blue. Most of the acids also redden blue litmus, and have a sour taste.

ENUMERATION.—Acids may be divided into *inorganic* or *mineral*, and *organic*. The mineral acids used in medicine are Boric, Carbonic, Chromic, Hydrochloric, Hydrobromic, Nitric, Nitro-hydrochloric, Phosphoric, Sulphuric, and Sulphurous acids. The organic acids thus employed include Acetic, Benzoic, Carbonic, Chrysophanic, Citric, Gallic, Hydrocyanic, Lactic, Oleic, Salicylic, Tannic, Tartaric, and Valerianic.

ACTION.—The stronger acids—sulphuric, nitric, hydrochloric, chromic, glacial acetic, and lactic acids—destroy animal tissues, and act as caustics when applied to the surface. When swallowed they produce the symptoms of irritant poisoning (*see* POISONS). An antidote for these poisons which is always at hand is carbonate of lime, in the form either of whiting, or of plaster chipped from the nearest wall. Other antidotes are alkaline carbonates and bicarbonates, milk, oil, and soap. Diluted acids, taken into the mouth, increase the secretion of saliva; and hydrochloric acid forms an important constituent of the gastric juice, without which digestion does not go on. When absorbed into the blood, dilute acids act on the heart generally, slowing its pulsations and reducing the temperature. They are excreted in the urine and milk.

USES.—Nitric acid is employed as a caustic application to piles, to poisoned wounds, and to spreading or unhealthy sores. Glacial acetic acid is used to destroy corns or warts. Diluted acetic acid or vinegar is applied as a lotion to relieve headache; to allay the itching of prurigo, lichen, and psoriasis; to check perspiration; and sometimes to hasten the appearance of exanthematous eruptions. Diluted acids, especially citric, tartaric, and hydrochloric, as well as acid tartrate of potassium, are administered in fevers as refrigerants, because they relieve the dryness of the mouth, and diminish the thirst by increasing the secretion of saliva, as well as lower the temperature and pulse-rate. Under the like circumstances, the organic acids, acetic, citric, and tartaric, when combined with alkaline carbonates in a state of effervescence or otherwise, form compounds which act on the skin and kidneys.

In febrile conditions, anæmia, and some forms of dyspepsia, the proportion of acid in the gastric juice is insufficient for the proper digestion of food, and the administration of dilute hydrochloric acid immediately before or after meals is useful both by aiding digestion and by preventing the formation of butyric and other acids, which give rise to sour eructations. Nitro-hydrochloric acid, before meals, is likewise beneficial in preventing acidity. It appears to have some action on the liver; and is used, both internally, and externally as a lotion or footbath, in jaundice and biliousness (*see* BALNEOLOGY). It generally relieves the frontal headache common in young women, which is felt just above the eyebrows, and not accompanied by constipation. Dilute acids, especially aromatic sulphuric acid, are useful in checking diarrhoea, colliquative sweats, hæmorrhages, and mucous discharges. By lessening the alkalinity of the urine, they tend to prevent the formation of phosphatic calculi, phosphoric, nitric, and lactic acids being most frequently employed for this purpose. Care must be exercised in their administration to nursing mothers, as they are excreted in the milk, and sometimes cause griping and diarrhoea in infants at the breast. Several acids have a special action of their own, and are considered under their respective groups, such as hydrocyanic and hydrobromic acids, which are sedative; boric and carbolic, antiseptic; chrysophanic, parasiticide; salicylic, antipruritic; gallic and tannic, astringent.

T. LAUDER BRUNTON.

ACIREALE, in Sicily.—Cold sulphur waters, and winter climatic health-resort. *See* MINERAL WATERS.

ACNE.—**SYNON.**: *Acne vulgaris*; *Acne adolescentium*; *Acne disseminata*; Fr. *Acmé*; *acné*; Ger. *Acne*.

DEFINITION.—A chronic disease of the skin, confined to the face, back, shoulders, and chest. The eruption is met with in young adults, and consists of pimples which are caused by an inflammation of the sebaceous glands and hair-follicles. The disease leaves small depressed scars.

ÆTIOLOGY.—The ætiology of acne is obscure. It is seldom met with before puberty, and is usually fully developed at about the age of eighteen or twenty, and then gradually disappears before the age of thirty. There are, however, many exceptions to this rule, especially in men, who are liable to a severe form of acne of the shoulders and back, which may continue until middle life. Acne appears to be equally common in males and females, and is especially met with in those who have what is called a slow circulation in the extremities—that is, who suffer from cold hands and feet, and perhaps chilblains. In accordance with this, the disease often improves, or even disappears, during warm summer weather, to return again in the following winter. The suppuration of the *comedones* is naturally attributed to pyogenic cocci (*see* ABSCESS), but these are often few in number and occasionally absent. Payne contrasts the pustules of acne with those of impetigo, in which cocci are plentiful. Unna attributes the disease to his *acne-bacillus*.

SYMPTOMS.—Common acne is a pimply eruption, met with chiefly on the face and shoulders, less often on the back and chest. Many of the pimples are of a pale colour with a small dark centre, and consist simply of follicles over-distended with sebum, the black centres marking the orifices of the follicles.

These pale little papules, called *comedones*, are apt to become red and inflamed, thus forming the ordinary acne-pimple, which ultimately suppurates, and discharges a minute quantity of pus; the spot then heals, leaving a small scar. In all severe cases of acne we find, in addition to the ordinary acne-spots, large pimples, resembling 'blind boils,' which suppurate slowly, and often leave very ugly scars. Acne develops in successive crops, so that as one set of pimples dies out, others appear, and thus the disease becomes chronic. Acne is almost always distributed symmetrically, so that both sides of the face and back are pretty equally affected. The exceptions to this rule are very rare.

DIAGNOSIS.—The differential diagnosis of acne is usually easy when the symmetrical distribution of the eruption and its period of development are taken into consideration. The two eruptions with which it is most likely to be confounded are: (1) certain forms of acne rosacea (*gutta rosea*), and (2) acne-like dermatosyphilis. Acne may best be distinguished from acne rosacea by the fact that this latter disease is chiefly met with in middle life, and is always attended with more or less general congestion of the face, and the subjective sensations of burning or tingling—characters which are absent in common acne. Acne rosacea is exclusively confined to the face, while common acne attacks also the shoulders and back.

Acne-like syphilitic eruptions are best distinguished by the absence of comedones; and by the tendency the pimples have to form into groups, which are not always symmetrical, and not strictly confined to the acne-regions. Some other syphilitic symptoms would probably be also present.

VARIETIES.—There is an interesting and somewhat rare eruption, commonly called *acne varioliformis*, which requires a very brief notice. In general appearance the pimples resemble those of acne, but are not especially associated with comedones. The eruption is usually confined to the scalp and upper part of the forehead, but is occasionally seen on the neck and chest. The scars formed by this disease are much deeper than those of common acne, and very closely resemble the pits left by small-pox. The region affected and the character of the scars are a sufficient guide to diagnosis. Recurrence is the rule.

TREATMENT.—As acne arises, for the most part, from the formation of comedones and plugging of the follicles, the treatment should be directed to stimulating the sebaceous glands of the skin to action, and hindering the growth of bacteria. For this purpose the application of sulphur is most useful. The sulphur may be applied in the form of an ointment or lotion. The objection to a lotion is, that during the night the powdered sulphur is apt to get into and inflame the eyes a little. There is not, however, the same objection to the use of a sulphur lotion during the daytime. A good plan is to apply a mild sulphur ointment or Wilson's hypochlorite of sulphur ointment every night, and wash it off in the morning with hot water, soap, and a soft flesh-brush. The daily use of a soft flesh-brush with hot water and soap is especially beneficial, as it prevents the formation of comedones.

When sulphur does not produce the desired effect, the linimentum saponis should be applied every night and allowed to dry on, and be washed off in the morning with warm water. In very severe

cases of acne, there are always a certain number of boil-like spots, which suppurate very slowly, and sometimes last for months without coming to a head. These boils should be carefully touched with a small piece of wood dipped in pure carbolic acid, or a saturated solution of potassa fusa in water; or the acid nitrate of mercury solution may be used, great care being taken that only the top of the boil is touched with the caustic. Cod-liver oil is the most generally useful tonic in cases of acne, but other tonics, as iron, are occasionally beneficial. For the treatment of acne varioliformis cod-liver oil, iodide of potassium, and arsenic are the best measures internally, while a dilute nitrate of mercury ointment may be used locally.

ROBERT LIVEING.

ACNE ROSACEA.—**SYNON.**: *Rosacea*; *Gutta rosea*; *Fr. Couperose*; *Ger. Das kupfrige Gesicht*.

DEFINITION.—A chronic inflammation of the face, attended with sensations of burning or stinging, and leading to a permanent enlargement of vessels and the formation of pimples.

SYMPTOMS.—There are two chief varieties of acne rosacea. The more severe form is met with chiefly in men, and is often the result of over-indulgence in alcohol. It especially attacks the nose, and leads to considerable enlargement of the vessels, and also, if of long standing, to hypertrophy of other parts of the skin, especially the sebaceous glands. The milder form of *gutta rosea* is very common in women after the age of thirty. In the first instance it may consist of a simple flushing of the face—generally of reflex origin, coming on at certain periods of the day, as, for example, after meals or in the evening. This congestion has, however, a tendency to become more persistent; and then, the vessels being permanently dilated, the redness does not entirely disappear, although it varies in degree from time to time. *Gutta rosea* in women is not confined to the neighbourhood of the nose, but usually extends in a butterfly-shaped patch to both cheeks, and sometimes to the forehead and chin. It is always attended with subjective sensations, which are generally those of burning, and less frequently of stinging or itching. Acne-like pimples are only occasionally met with, and when present are due to follicular inflammation. There is often seborrhœa nasi.

ÆTIOLOGY.—*Gutta rosea* in women is for the most part associated with cold hands and feet, and not uncommonly with dyspepsia and constipation, or irregularity in menstruation. In men, as before stated, the hypertrophic variety is sometimes, but by no means always, caused by over-indulgence in alcohol.

TREATMENT.—The severer form of *gutta rosea*, in men, is best dealt with by: (1) abstinence from alcohol; (2) the longitudinal division of the enlarged veins with a lancet, or the employment of electrolysis; and (3) the subsequent daily use of sulphur ointment, which should be well rubbed on every night. Great hypertrophy of the nose (*rhinophyma*) can only be removed by surgical methods. In treating *gutta rosea*, as it is commonly met with in women, the following points require special attention: (1) The diet should be most carefully regulated, and medicines ordered to remove, as far as possible, any dyspepsia or gouty tendency or any menstrual irregularity that may exist, and also to regulate the bowels. (2) A very mild sulphur (or hypochlorite

of sulphur) ointment should be applied in a small quantity every night, and washed off in the morning with warm soft oatmeal-water. In the daytime, a calamine and oxide of zinc lotion should be applied, and a little allowed to dry on. Gutta rosea is difficult to cure, but a steady perseverance in a rational plan of treatment will always produce a marked improvement in the disease, and sometimes quite remove it.

ROBERT LIVEING.

ACONITE, Poisoning by.—SYNON. : Fr. *Empoisonnement par l'Aconite*; Ger. *Eisenhutvergiftung*.—The common garden-plant, *Aconitum napellus*, known also as 'wolfsbane' or 'blue-rocket,' as well as other species of aconitum, are poisonous, and owe their poisonous properties to the presence of an alkaloid, *aconitine* or *aconitia*, or rather to a mixture of alkaloids passing under this name. The same or similar alkaloids have been obtained from the Indian aconite, *A. ferox*, and from Japanese aconite roots. All parts of *A. napellus* are poisonous. Aconitine is perhaps the most poisonous alkaloid known. All parts of the aconite plant when chewed, and aconitine when placed upon the lips or tongue, produce, after a few minutes, a disagreeable acrid burning sensation, followed by numbness, loss of sensibility of the part, salivation, and an after-sensation of searedness. These sensations may last for several hours.

The fresh root of aconite has frequently been eaten in mistake for horse-radish, to which it bears a remote resemblance. The root of horse-radish is whitish on the exterior, is long and of fairly uniform diameter, has a pungent odour when scraped, and the scraped surface retains its white appearance; whereas aconite-root is brown and conical, is destitute of pungent odour, and speedily acquires a pink colour when scraped and exposed to the air. Mistakes more frequently occur from liniments containing aconite being swallowed in error. In two cases the root has been administered with homicidal intent; and in one case a young man was killed by the administration of, as it is supposed, two grains of English aconitine. Accidents have also arisen from the administration of the potent English aconitine in mistake for the impure inert exotic or German alkaloid, or mixture of alkaloids passing under that name.

ANATOMICAL CHARACTERS.—After poisoning by aconite there may be gastric congestion or inflammation; but these may be absent.

SYMPTOMS.—When aconite, or any of its preparations, is taken by the mouth, the first sensation, transitory and mainly due to the action of the solvent, is followed in about three minutes by an intolerable burning and numbing pain, extending from the place of application to all the surrounding parts of the mucous membrane. There is salivation; and the burning sensation extends down the gullet to the stomach. Occasionally, when the poison has been rapidly swallowed, no marked symptoms may supervene for half an hour. The general symptoms are very varied, but may all be referred to weakening of the heart's action, disturbances of respiration, and paralysis of sensation on the surface of the body. This last may be described as 'numbness' or 'drawing of the skin,' or by some equivalent term. There is pain in the epigastrium, violent vomiting, and often purging; the pulse, at first rapid, quickly diminishes in frequency and force till it is imperceptible; the skin is cold,

clammy, and livid; respiration is laboured. The pupils, at first contracted, afterwards dilate; and this dilatation sometimes occurs suddenly and transitorily, and is accompanied by blindness. Convulsions are not common; but vomiting is often due to spasmodic contraction of the diaphragm, causing frothing at the mouth. Consciousness is retained till near the end of life.

DIAGNOSIS.—The peculiar sensation in the mouth—burning, feeling of searedness, numbness, &c., the great cardiac depression, and the difficulty of respiration, will generally serve to determine the nature of the case.

PROGNOSIS.—Death usually occurs within four or five hours. If the patient survive twelve hours, recovery is usually rapid and complete.

Fatal dose.—Of the root, sixty grains—probably much less might suffice. Of the pharmacopœial *tinctura aconiti* (1 in 20) half a fluid ounce to one fluid ounce. *Fleming's tincture* is about fifteen times as strong as the official tincture, and twenty-five minims have proved fatal. Four grains of the formerly official *alcoholic extract* have proved a fatal dose. English *aconitine* or *aconitia* (the alkaloid) is terribly potent: 1-2000th grain will produce a very decided sensation on the tongue, and it is probably as poisonous as the crystallised aconitine-nitrate, one-sixteenth of a grain of which has killed an adult within five hours.

TREATMENT.—In proceeding to treat a case of poisoning by aconite we must, first, wash out the stomach by means of the stomach-tube, and promote vomiting by warm emetics, of which carbonate of ammonium is the best. Stimulants must be freely administered by mouth or rectum; also strong black coffee or tea. Brandy and ether may be injected subcutaneously. Digitalis is a counter-poison, and may be administered with effect subcutaneously, in doses of twenty minims of the tincture, repeated in an hour or so if necessary. Inhalations of nitrite of amyl may afford some relief. The patient must be kept strictly in the recumbent position, warmth being applied to the surface; and, as a last resort, artificial respiration must be used. See POISONS.

THOMAS STEVENSON.

ACQUI, in the Province of Alessandria, Italy.—Thermal sulphur waters. See MINERAL WATERS.

ACQUIRED DISEASES.—Diseases which originate independently of hereditary transmission.

ACROCHORDON (ἄκρον, a point or end; and χορδή, a string).—An outgrowth from the skin, having the form of a slender cylinder. Such outgrowths are occasionally met with in elderly persons, especially on the neck or trunk. Microscopically, an acrochordon is composed of loose areolar tissue.

TREATMENT.—This consists in snipping off the outgrowths with scissors, in touching them with a strong solution of potassa fusa, or in applying the liquor plumbi fortis, or a 4 per 1000 solution of mercuric chloride.

JOHN HAROLD.

ACRODYNIA (ἄκρος, extreme; and δόλην, pain).—Acrodynia is the name applied to a disease epidemic in Paris in 1828. It was described by Alibert as a dermatitis, affecting particularly the palms and soles, accompanied by formication, anæsthesia or hyperæsthesia with stinging and smarting

pains. The pain may extend over the whole body, and be accompanied by gastro-intestinal disorder. The skin is at first bright red, then deeper tinted and brown, with subsequent pigmentation and desquamation, the cuticle sometimes being shed in a single piece. Occasionally there are pimples, pustules, and blisters. The disease runs a course of several weeks.

TREATMENT.—This consists in the treatment of the local inflammation by appropriate measures. See PELLAGRA; ERGOTISM.

JOHN HAROLD.

ACROMEGALY (ἄκρον, an extremity; and μέγας, large).—SYNON.: Fr. *Acromégalie*.

DEFINITION.—A remarkable dyscrasia, the striking characteristic of which is an enlargement of the bones, and most obviously of the bones of the hands and feet. Hence the name applied to it by Marie, who first described the disease in 1886, though other cases had been noted previously under different titles.

PATHOLOGY.—Acromegaly occurs most frequently, but not exclusively, in females between the ages of thirty and forty, but has been observed as early as fifteen and as late as forty-eight. It is very chronic; and though probably often fatal, it sometimes appears to become arrested after advancing for some years. In females it is usually preceded by early stoppage of the menses. Its relation to rheumatism is very doubtful.

Acromegaly is generally associated with enlargement or atrophy of the thyroid body; and enlargement of the thymus gland has been observed. In many post-mortem examinations tumours of the pituitary body have been met with.

DESCRIPTION.—In this disease the bones of the hands and feet are very greatly enlarged, but the long bones do not escape, the natural prominences and elevations being exaggerated sometimes to the dimensions of exostoses. The scapulæ, clavicles, and the sternal ends of the ribs become quite massive; the lower jaw also assumes vast proportions, the lower teeth spreading out and projecting far in front of those of the upper jaw. This gives a characteristic shape to the face, which is that of an egg with the large end downwards, as opposed to the oval of myxœdema, and the inverted egg-shape of osteitis deformans, with both of which diseases acromegaly may be confounded. The upper jaw and the cranial bones occasionally participate in the change. The bones, though enlarged, do not become curved; but the spine is often bent, so that the patient loses considerably in stature.

The cartilages of the nose, ears, eyelids, and larynx may be thickened; the last change probably accounting for an occasional great alteration in the voice.

The subcutaneous tissues are not obviously altered. The skin may be unchanged, but on the face it is apt to become coarse, and to show clearly the orifices of the sweat-glands. The hair does not become thin, as in myxœdema. Profuse perspiration and thirst are generally present.

The tongue is enlarged, and taste may be much impaired. Blindness is not uncommon, from atrophy of the optic nerve, possibly connected with a pituitary tumour. The sense of smell is often impaired; that of hearing does not appear to be modified, nor does that of touch.

There may be shortness of breathing, depending

upon asthmatic attacks, and the heart may be hypertrophied. The urine is not usually abnormal. The temperature is not raised. The intellect remains perfect. See MYXŒDEMA.

DIAGNOSIS.—It is not necessary to indicate the difference between this disease and osteitis deformans or myxœdema, the two diseases with which it is most likely to be confused, because, though there is certainly a superficial resemblance to each, the points of divergence are wide.

A somewhat similar disease, hypertrophic pulmonary osteo-arthritis, has lately claimed much attention. It was also first described by Marie. It occurs in persons suffering from chronic disease of the lungs. The face is unaltered, the deformity in the hands and feet is almost limited to the phalanges, and the spinal curvature is more marked than in acromegaly and confined to the lower dorsal and lumbar regions.

TREATMENT.—None of the numerous methods of treatment hitherto applied in acromegaly have proved of the slightest use. See BONE, Diseases of; OSTEITIS DEFORMANS.

RICKMAN J. GODLEE.

ACTINOMYCOSIS (ἀκτίς, a ray; and μύκης, a fungus).—DEFINITION.—A chronic disease, due to infection by a fungus known as *Actinomyces bovis*, attended by the formation of abscesses, chronic interstitial inflammation, or tumour-like growths of granulation-tissue, affecting various organs and structures of the body.

HISTORY.—This disease, which especially affects horned cattle and swine, and other animals which feed on grain, has long been known in them under various names, such as 'woody-tongue' in cattle. Its parasitic nature was demonstrated by Bollinger in 1876, and the name 'actinomycoses' or 'ray-fungus,' given by him, has since been employed to name the disease. Our present knowledge of the disease in man is due mainly to the researches of J. Israël (1877), Ponfick (1879), and A. Poncet and L. Bérard (1898). The work of the two latter writers is of especial value, and may be referred to for most of the literature.

ETIOLOGY.—The fungus which causes the disease may first be briefly described. The *Actinomyces* or *Oospora bovis* belongs to the Streptothricæ, an order of fungi intermediate between the higher bacteria and the mould-fungi. The spores (or conidia) grow into cylindrical threads which branch dichotomously, and form a radiating mycelium in which the threads are often densely felted together. The hyphæ are often much twisted, almost resembling spiral bacteria. As growth advances they frequently break into short segments, or rows of coccus-like bodies. These are not, however, regarded as true spores. The spores are formed in rows on special fruit-bearing hyphæ, which on a free surface grow up into the air. The spores are rounded, of somewhat larger diameter than the hyphæ.

The various shapes of the different elements of the growth, the presence in a colony of densely felted threads, irregularly twisted, of shorter rod-like bodies, and of ovoid and spherical spore-like bodies, has led to needless emphasis as to the 'polymorphism' of the fungus. All are simply stages of normal growth.

The tendency to radiating growth, whether in the tissues or in cultures, leads to the formation of spherical masses. These are well seen in a 'shake

culture' in gelatine medium, or in liquids. The central part usually undergoes degeneration, and has a hyaline appearance under the microscope, while in a liquefied culture it looks hollow. The dense felting of the filaments binds the mass together, and makes separation difficult, while it accounts for the presence of the little balls of the fungus in the puriform fluid from actinomycotic abscesses.

The character which especially attracts attention is the peculiar pear- or club-shaped swelling of the ends of some filaments. This is especially seen in the growth in the tissues, most markedly in bovine animals, the swollen ends often becoming calcified. But this character is not always well developed, especially in human actinomycosis, and the diagnosis must sometimes be made in its absence or with very partial development. In artificial growth it occurs only in old cultures, and is undoubtedly due to degeneration, with swelling of the sheath of the mycelium.

Cultivation of the fungus when once made can be easily continued, growth readily occurring in a great variety of media. But culture cannot usually be employed for diagnosis, as it is difficult to separate the other associated bacteria. Success is attained only by anaerobic culture at first, though aerobic growth is afterwards easy.

On agar-medium the colonies are usually hemispherical, discrete, of yellow beeswax colour, becoming very hard and cracked in the centre when about the size of a mustard seed. Sometimes they are whiter and small, the varieties depending largely on the moisture of the medium. On potato-paste the growth is especially characteristic, with the spore-bearing hyphae, and the surface powdered by the spores, and showing oily droplets. In gelatine-medium slow liquefaction takes place, and the colonies float as fluffy spheroidal balls with long filamentous offshoots in the liquid.

The spores have great vitality: when dry, they may be kept alive for three or four years. They are destroyed by exposure to moist heat of 75° C. for five minutes, while the mycelium is killed by shorter exposure to 60° C.

MODE OF INFECTION.—All the evidence at present attainable points to the extreme rarity of inoculation from diseased animals or from their flesh, although experimental inoculation has been successful. Some cases of alleged infection from cattle must be regarded as doubtful, for the disease is of very slow course, and other sources can hardly be excluded.

It is certain that the fungus is common on various cereals, although it is not certainly known what kind of grain most frequently harbours it. Some kinds of barley, including wild barley, appear especially liable. In a large number of recorded cases, a fragment of some cereal has been found embedded in the centre of the original focus of the disease, and in others the disease was known to have followed the lodgment of ears of barley or other corn. Indeed, although the fungus is probably common, and the spores have so great vitality and can be easily inhaled, some additional factor appears necessary in most cases to enable them to gain a hold on the tissues, and this may be afforded by such means as the presence of a large number of spores on the sharp awns of barley, which can penetrate and irritate the tissues. Chewing raw grain or straw, and inhaling dust in threshing, win-

nowing, or chaff-cutting are known modes of infection.

LESIONS OF ACTINOMYCOSIS.—The lesions of actinomycosis, like those of tuberculosis, present very great varieties in form, according to the organ affected, the intensity, duration, and character of the change produced, and the superadded action of other bacteria.

Most of the changes fall into one of two groups:—(1) a slowly progressive infiltration of 'granulomatous' type, resembling the chronic infiltrations of syphilis or tuberculosis; (2) a diffuse necrotic and suppurative change, leading to diffuse puriform infiltration or abscess-formation. The necrotic and suppurative changes seem especially apt to occur when the disease involves the viscera, and many bacteriologists believe them to be due to the added effects of septic bacteria.

In the suppurative forms the process may be somewhat acute, but more commonly it is at first slow, and has become advanced before any rapid necrosis or suppuration occurs. Around the colonies of fungus there is developed granulation-tissue modified in its arrangement by the nature of the virus. Each minute colony becomes surrounded by cells, some of which assume the epithelioid type; around these are zones of smaller proliferating cells and sometimes leucocytes, as with other parasitic irritants. Giant-cells are sometimes seen, but have not the peculiar characters or distribution found in tuberculosis. Indeed the only special character of the masses is the presence of the colonies of fungus, which are treated by the animal economy as all other irritating and noxious substances are, viz., by attempts to remove or destroy them, or to enclose them in a dense fibrous capsule. Hence from these younger cellular growths are formed masses of dense fibrous tissue, and the colonies of fungus may be arrested in growth and degenerate. It is especially in these denser fibrous masses that we find the older colonies, composed largely of radiating clubs, partly calcified, which were formerly regarded as the usual and typical form. Such are well seen in the 'woody-tongue' of cattle (see fig. 1). But if the growth is more rapid and cicatrization less complete, the colonies may be much larger and show little sign of clubs, except in some of the radiating offshoots. The various forms of mycelium, &c., already described, may then be seen.

Staining is easy and various methods may be used, such as carbol-thionin blue, methylene blue, or gentian violet. The colour is not discharged by iodine with Gram's method. The degenerated ends or clubs lose their affinity for aniline dyes, but are well stained with picricarmin or eosin.

In some of the cells which surround the colony, portions of the fungus are often seen. These cells, though probably phagocytic, may also serve to transport the fungus to other parts, and thus spread the infection. Although the extension is usually by slow continuous infiltration, widespread diffusion frequently occurs; this takes place especially, it is believed, by way of the blood-vessels. Indeed, one marked difference from tuberculosis is the comparatively slight involvement of the lymphatics and lymph-glands.

By this continuous infiltration, especially along the connective-tissue planes, large masses of granulation and fibrous tissue may be formed, resembling tumour-growths. But at some period local softening may occur, either purely necrotic or with actual

suppuration; and sinuses are formed which open on some free surface. As already stated, in some organs, a diffuse infiltration with softening and pus-formation is common. This is no doubt often aided by bacteria, especially by the common agents of suppuration; but a purely necrotic softening may also occur.

The 'pus' from the 'abscesses' is often thick, sometimes of peculiar odour, and contains minute particles, some of which may be visible to the naked eye, or with a lens, of sulphur-yellow colour, or sometimes greenish or black, especially where decomposition has occurred, as in the abdominal viscera. The detection and examination of these little masses, the



FIG. 1.—The woodcut shows the characters of a minute centre of growth in an actinomycosis-nodule from the tongue of a cow. The upper portion is sufficiently described in the text. The mass of fungus is represented as much less dense than in reality, otherwise no detail could be shown. (Magnified about 800 diameters.)

The lower four small figures show the characters seen in the same growth under a somewhat higher power. (1) A group of cells containing a cluster of the clubbed filaments. (2) A single cell with actinomyces growing in it. (3) A group of clubbed hyphae much larger than the others. (4) Filaments from centre of mass, showing bacillus-like characters, and branching of mycelium.

colonies of the fungus, are of especial diagnostic importance. They are somewhat hard and difficult to tease out, owing to the dense interlacing of the mycelium. In any suspected case, if they are not readily found, it is well to treat some pus with caustic alkali, mix with a quantity of distilled water, allow to settle in a conical glass, or separate with a centrifuge and then search carefully. If crushed, and stained with aniline dyes, they show the characteristic appearances, contrast-staining with picrocarmine or picric acid bringing out the 'clubs' well.

The general effects produced by these slowly spreading proliferative and necrotic changes vary much with the organ or tissue involved.

When upon a mucous membrane, slowly spreading ulceration and induration may occur, and the surface be covered by masses of epithelium containing the fungus. But after infection of subjacent parts has occurred, the ulcer may heal and the site of inoculation be thus obscured.

The infiltration may simulate a tumour, especially sarcoma, and cases of 'osteosarcoma' of the jaws &c. in cattle have proved to be actinomycotic. It may also resemble any of the so-called chronic inflammatory infiltrations, such as those of tuberculosis or syphilis. Finally it may cause chronic abscesses, usually following slow induration, but, in some organs, e.g. the liver, widespread necrotic and suppurative changes may occur.

SITES AND ORGANS INVOLVED.—Actinomycosis is one of the diseases which may affect almost any organ in the body or any group of organs, and which when once established may spread to distant parts. Cases are recorded of abscesses in the brain, kidney, ovary, thyroid gland, &c. But it especially occurs in regions adjoining the sites of easy lodgment of grain or like substances, which can convey the fungus. More than one half of the cases are in the cervical and maxillary regions, infection being by the gums, crypts of tonsils, pharynx, or more commonly by the buccal mucous membrane (Poncet). Next in frequency come the alimentary canal and peritoneum, the œsophagus and the cæcum being especially common starting-points. Then the lungs and pleuræ, and the mediastinum and the vertebæ. In the case of the lungs infection may enter through the bronchi, but in some cases has spread from the pharynx or œsophagus. Of other sites may be mentioned the skin, where the inoculation may be local, and the bones and periosteum, which are usually infected secondarily to some other tissue.

A brief account of some of the chief clinical forms must suffice.

The *tongue* is probably a not uncommon site in man as in cattle. Small nodules, resembling and probably often mistaken for syphilitic gummata, may be formed, and may cause ulceration or become indurated as in cattle. Probably many heal without secondary infection. Any part of the mouth, the gums, or buccal mucous membrane may also be primarily affected.

The *palpebral* and *conjunctival mucous membrane* may be locally affected; and the skin also of any part, especially that of the face and hands. But in most cases the affection of the skin and subcutaneous tissue is secondary to the formation of sinuses. It is probable that some intractable sores, analogous to Madura Foot, may be due to Actinomyces, although Madura Foot itself is now thought to be due to an allied species of fungus.

Where deeper infection and spread occurs, the locality will depend on the point of lodgment. If the infection is from the *buccal mucous membrane*, gums, or fauces, the 'cervico-maxillary' region is involved; if from the *pharynx* or *œsophagus*, that tube itself may be affected, with extension to the mediastinum, the vertebæ, and to the pleuræ and lungs, which may also be infected through the bronchi; if from the *stomach* or *intestines*, abdominal lesions, especially of the peritoneum, the liver, and more rarely of the kidneys and ovaries, are likely to occur.

But when once any extensive hold has been gained, infection may be carried to distant organs, such as the brain, by the blood-stream.

Cervico-maxillary forms.—In nearly two-thirds of the cases recorded the site of the principal lesions has been in the face and neck. According to Poncet the parasite usually enters by way of the buccal mucous membrane, and spreads to the tissues surrounding the temporo-maxillary articulation, or to the submaxillary region. The disease may extend up or down in various directions. Its course is usually subacute or chronic: sinuses are generally formed, sooner or later. If the periosteum is extensively involved, necrosis of bone may follow.

In this region actinomycotic lesions may simulate a variety of diseases, such as tumours of the jaw, chronic periostitis, tubercular enlargements of glands, and parotid tumours. They may, moreover, be complicated by secondary lesions in the brain, thorax, and elsewhere. Among the features of especial importance in the diagnosis from these various diseases are, according to Poncet, (1) the early and marked pain, (2) early trismus—due to the irritation of the masseter and temporal muscles—and especially common when the disease is in the temporo-maxillary region; (3) the peculiar swelling, which has an unequal and peculiar consistence; and (4) the relatively slight glandular enlargement. But in many cases the diagnosis is uncertain until sinuses appear, or until careful exploration has shown the presence of the fungus.

Abdominal forms.—In the abdominal forms infection appears to start most commonly from the large intestine, especially the cæcum, including the ileo-cæcal valve and vermiform appendix. In some cases the source of infection has been traced to foreign bodies embedded in the mucous membrane. Various forms of intestinal ulceration are occasionally found. There is an especial tendency to adhesions and thickening of the peritoneum, with the formation of chronic abscesses, which burrow in various directions, and sometimes involve adjacent organs. These chronic peritoneal lesions give rise to most of the symptoms and physical signs met with in this form of the disease.

Liver.—Actinomycotic abscess of the liver has been frequently observed. It is practically always secondary to infection from the intestine or some other part. It may, however, be the most obvious lesion during life. The abscess may be solitary, with adhesions and induration around; or it may be multiple, especially where infection is through the portal vein, and other organisms accompany the actinomycetes; or there may be a diffuse infiltration, with widespread necrosis. Enlargement of the organ may usually be detected, and may be great, but if the abscess is deeply seated in the upper or posterior part, it may, as in tropical abscess of the liver, be difficult to diagnose. It may simulate cancer, solitary or multiple, or other hepatic enlargement. Except in the more rapid forms, where the fever may be like that either of chronic or of acute tropical abscess of the liver, the disease gives rise to no special hepatic symptoms.

Thoracic and pulmonary forms.—Ulceration or stricture of the œsophagus may occur. The disease may spread in the mediastinum and may involve the vertebræ and ribs. It may extend to the pleuræ and thence to the lungs, or inversely from the bronchi to the lungs and pleuræ. In the *lungs and pleuræ* the symptoms are those of very chronic phthisis or of interstitial pneumonia, with pleural thickening, followed by some gradual excavation. The formation of sinuses through the

chest-wall is not uncommon, the periosteum and bone of the ribs being often infiltrated and destroyed in the course of the disease.

In the majority of cases of pulmonary lesions the bases are involved while the apices remain free. Pyrexia may be slight or absent; the temperature is much less irregular than in tuberculosis, and does not show marked evening rise. The sputa are often fetid at an early period, before marked signs of cavity-formation appear; while hæmoptysis and nummular sputa are rare. There is less disturbance of respiration, and far less impairment of general health. The lymphatic glands in the neck and axillæ are not enlarged (Poncet and Bérard). There may be serous pleuritic effusion. Upon the whole the pulmonary forms more resemble obscure interstitial pneumonias or new-growths in the lung than phthisis—at least during a considerable part of their course. The detection of the fungus in the sputa may be the only means of positive diagnosis, and in some cases it has not been found, though carefully and repeatedly sought for.

TREATMENT.—Treatment is mainly of two kinds, medicinal and operative. But it must be especially insisted on, that actinomycosis is a disease against which there are strong natural powers of resistance, and that the parasite gains hold with difficulty in most healthy subjects, and may be overcome even when it has gained a lodgment. Hence the great importance of all means of increasing the general health, and of avoiding any source of irritation, or of septic infection.

Medicinal Treatment.—Potassium iodide in large and increasing doses has proved successful in many cases in aiding the arrest of the disease, although it is not in any sense a specific. In early cases it should always be employed in addition to surgical means, or where these are not available. When the disease is only diagnosed late, or if bacterial infection has occurred, it may entirely fail, though its use even here has not proved injurious. It does not act directly on the fungus, for one per cent. of potassium iodide in culture-media does not prevent the growth of the parasite. The doses may begin with thirty grains daily, and rise to ninety grains or more. Other iodides have not yet been sufficiently tried.

Operative Treatment.—In cases in which the disease is accessible, operation is usually beneficial, even if the entire extent of the infiltration cannot be reached.

Free excision, with curetting or scraping, should be employed, followed by irrigation with antiseptics; the exact steps varying with the site and extent of the disease. Of the antiseptics corrosive sublimate has been most used, but it is almost certain that a solution (in excess of potassium iodide) of biniodide of mercury (1 in 4,000), with addition of 10 per cent. of glycerin, would be far more effective, from its greater antiseptic and penetrating power. Iodide of starch paste, to which the solution of biniodide of mercury in the proportion of 1 in 8,000 has been added, is valuable as a continuous application. Where possible, the eradication of all infected tissues is desirable, but where less complete removal is possible, much benefit may follow partial removal, if free access is left for antiseptic applications and for spontaneous evacuation. This applies even to lesions of internal organs.

The injection of antiseptic solutions into the nodules, which has been advocated, is not without

risk, and should never be employed where excision is possible, or where any serious risk would be incurred by acute inflammatory reaction and swelling, which usually follow. In any case, the closure of the wound should not be premature, as it is rarely possible to be sure that all the foci of the disease have been reached.

Of other suggested lines of treatment, such as by antagonistic sera, or by mixed culture-fluids, by tuberculin, and so on, it may be stated that no protective or antiserum has yet been produced, nor has any benefit resulted from the substances tried hitherto. Such treatment is of course possible in the future, but in advanced cases the use of the knife will probably still be essential.

PROPHYLAXIS.—What has been stated on the mode of infection sufficiently indicates the measures needed. Ordinary cooking would destroy the infection of meat, which can hardly ever be the actual source. Except for those who have to attend diseased cattle, the recognition of the possible modes of infection by grain, and means to avoid risk, are chiefly necessary.

Further knowledge of the exact kind of grain most affected, and of any ready means of the detection and prevention of its invasion by the parasite, would be of great value. W. S. GREENFIELD.

ACUPUNCTURE (*acus*, a needle ; and *punctum*, a prick).—SYNON. : Fr. *Acupuncture* ; Ger. *Nadelstich*.

DESCRIPTION.—Acupuncture is an ancient mode of treatment for the relief of painful affections, now but little used, consisting in the introduction of fine round needles through the skin, to a varying depth. The needles used are about two inches in length, and are set in round handles, so that they can be introduced with a gentle rotatory movement. Acupuncture is now employed chiefly in lumbago and sciatica, in which affections it undoubtedly may give relief. The operation is thus performed:—the patient being laid upon his face, tender spots are sought for—in lumbago over the erector spinæ, in sciatica along the course of the sciatic nerve. The needles are then pushed in vertically for a depth of from one and a half to two inches, and allowed to remain for from half an hour to two hours. The number of needles employed may vary from one to six. In sciatica it is recommended, if possible, to make the needle actually penetrate the nerve. This result is known by the patient complaining of sudden pain shooting down the back of the leg. The mode of action is uncertain, but in sciatica it has been supposed that the puncture of the nerve-sheath allows the escape of fluid.

ACUTE.—This word, when associated with a disease, signifies that such disease runs a rapid course, and is attended with urgent symptoms. It is also employed to express intensity of a particular symptom, e.g. an acute pain.

ADDISON'S DISEASE.—SYNON. : Bronzed Skin Disease ; *Melasma Addisonii* ; Fr. *Mélano-dermie Asthénique* ; *Maladie d'Addison* ; Ger. *Addison'sche Krankheit*.

DEFINITION.—A constitutional disease characterised by extreme muscular weakness, asthenia, a tendency to syncope, nausea, vomiting, and an exaggeration of the normal pigmentation of the skin. After death alteration of the suprarenal bodies is the chief lesion found.

Although pigmentation is one of the most striking signs of the fully developed disease, there are cases without any pigmentation where the symptoms come on acutely, rapidly lead to death, and are found to be associated with extensive disease of the suprarenal capsules. This condition is spoken of as '*Addison's disease without pigmentation*,' and is possibly due to acute insufficiency of the suprarenal glands.

HISTORICAL.—This disease was first described in 1854 by Thomas Addison, physician to Guy's Hospital. The discoverer's name was subsequently attached to the disease by Trousseau.

FREQUENCY.—This is a rare disease and is probably diagnosed erroneously more often than it is overlooked. It appears to be less rare in this country than in America. Its detection in dark-skinned races is a matter of great difficulty.

It is commoner in males (65 per cent.) than in females (35 per cent.) and occurs most often about 30 years of age, though it has been met with at both extremes of life (one week and eighty-one years). There is no evidence that it is proportionately rarer in the well-to-do than in the labouring classes.

ÆTIOLOGY.—As 80 per cent. of the fatal cases are due to tubercular disease of the suprarenal capsules, it has been thought that the disease is more likely to occur in families where tuberculosis is rife. This is, however, rather an assumption than a proved fact. Exceptionally it has been known to kill two members of the same family ; probably in such instances the resistance of the suprarenal glands is particularly low. Blows on the back or strains from laborious occupations have been regarded as disposing to tubercular infection of the capsules. It is possible that traumatism by giving rise to hæmorrhage into the suprarenal bodies, such as is not uncommonly seen in still-born children after difficult labours, and occasionally in later life after severe injuries, may so reduce the resistance of the organs that tuberculosis develops there more readily.

In some instances the suprarenal bodies are the only parts of the body showing tuberculosis ; but usually other organs are affected, and in a certain proportion of fatal cases the disease is found to have spread by continuity from tubercular osteitis of the vertebrae.

MORBID ANATOMY.—In about 80 per cent. of the fatal cases the suprarenal capsules show tuberculous infiltration : this is undoubtedly their most frequent condition, but other lesions may give rise to Addison's disease.

The tubercular change begins in the medulla as granulation-tissue, which becomes in due course caseous, and may either dry up, become calcified, or soften down into a puriform fluid. The tubercular inflammation spreads to the cortex and from thence to the tissues around. In this way the important sympathetic plexuses and ganglia may become involved. The suprarenal bodies become enlarged, deformed, and nodular. Bacteriological examination leaves no doubt as to the tubercular nature of the change.

In some exceptional, but typical, cases of Addison's disease the suprarenal capsules show atrophy without any inflammatory change ; the atrophy may be so extreme that only a yellow streaking of the fat in the normal situation of the glands is left ; it is not surprising therefore that their absence is sometimes recorded. Chronic inflammation with atrophy, there being no evidence of tuberculosis, is the lesion

found in a small percentage of cases. Very occasionally some of the symptoms of Addison's disease are manifested when secondary malignant growths are present in the suprarenals; these cases are naturally not typical, since the patient is suffering from two morbid conditions—malignant disease of some other part of the body and suprarenal inadequacy. Primary malignant growth of the suprarenal bodies does not present the clinical features of typical Addison's disease. In a few fatal cases of the disease the suprarenal bodies themselves are healthy, while there is some lymphadenomatous or other growth in the immediate neighbourhood which may involve the adjacent sympathetic or compress the efferent lymphatics and veins of the gland.

The *sympathetic nerves and plexuses* in the immediate neighbourhood of the affected suprarenal bodies are frequently invaded by inflammatory tissue and microscopically degenerated; in other instances, as in simple atrophy of the suprarenal glands, they are healthy. The inconstancy in their condition is strong evidence against the view that the symptoms of Addison's disease depend solely and entirely on irritation and neuritis of the great sympathetic plexuses in the neighbourhood of the suprarenal glands.

Other morbid lesions.—The heart is small and atrophied. The lymphoid tissue in the alimentary canal is often increased in amount. The thymus has sometimes been found to be persistent and the spleen softened: these last lesions are by no means constant.

PATHOGENY.—The relation of the morbid changes in the suprarenal glands to the symptoms of Addison's disease has given rise to a great deal of discussion. Three theories may be mentioned. (1) The *nervous* theory—that the symptoms depend on the irritation of the sympathetic nerve-fibres either in the solar and other large plexuses, or of those actually embedded in the suprarenal glands, and are really independent of the functional destruction of the glands themselves. For the reason given above this view is no longer accepted as a complete and exclusive explanation. (2) *The theory of suprarenal inadequacy*—that the symptoms are due to an absence of the normal internal secretion of the glands: Addison's disease would thus be analogous to myxedema. This theory is supported by experiments on animals and by the fact that two prominent symptoms of the disease—low blood-pressure and loss of muscular tone—are the exact converse of the physiological effects of suprarenal extract. The absence of the normal internal secretion might theoretically lead solely to a condition of apathy and want of tone, or in addition, by upsetting the metabolism of the organism, to a toxæmic state. The theory of suprarenal inadequacy is widely accepted at the present time, but there are difficulties in accepting it, such as the facts that in some cases the glands are extensively destroyed without symptoms, that in other instances symptoms are present in association with healthy glands, and that the administration of suprarenal extract so frequently fails to relieve the symptoms. (3) *The theory of nervous irritation and glandular inadequacy*—that while some cases are due to glandular inadequacy alone, others are due to this factor plus irritation of the sympathetic plexuses in the immediate vicinity. Under treatment by suprarenal extract the cases of the first category improve while those in the second category receive little or no benefit.

CLINICAL COURSE AND SYMPTOMS.—The onset is usually very insidious and slow, but sometimes the patient dies of acute symptoms that have rapidly developed without any suspicion having arisen as to the existence of disease of the suprarenal bodies: the nature of the disease is then only revealed at the autopsy. In an ordinary case there is gradually increasing weakness and loss of muscular power accompanied by dyspepsia, loss of appetite, and gastric disturbance. The heart's action becomes depressed, the pulse is of low tension, remarkably soft and weak, and there is shortness of breath on exertion and a tendency to syncope. The loss of strength and vigour is out of all proportion to the general nutrition; anaemia is rarely a prominent feature. The temperature is, like other functions, depressed, fever and leucocytosis being exceptional, and when present probably due to some complication. Mental apathy and depression are marked features in most cases. Headache, giddiness, sighing, yawning, or hiccough may develop as the disease progresses. Pain in the back, thought to be due to traction on the nerve-fibres around the suprarenal bodies, may be troublesome, while pains in the joints and limbs are occasionally observed. Nausea, retching, vomiting, and flatulence may be very prominent and recur over long periods; sometimes vomiting may so exhaust the patient as to bring him to death's door. Epigastric pain and 'sinking' feelings are not uncommon. The urine does not present any constant or characteristic features.

After a varying interval pigmentation appears; in rare instances it is the first thing noticed, but usually it is preceded by constitutional symptoms and in acute cases may be absent. It is an exaggeration of that seen in health and is increased by any irritation, so that the exposed parts of the body and those liable to pressure by dress, &c., become pigmented; the face, neck, backs of hands, arms, anterior folds of axillæ, the shoulders, nipples, spine, waist, linea alba, genitals, and the knees are likely to be darkened; while the nails, palms, soles, and scalp where it is covered by hair, are not affected. The writer has, however, seen marked pigmentation of the palms. Around scars or blisters the pigmentation is exaggerated. The hair becomes darker. Small black spots like moles may also develop on the skin. Occasionally the disease is associated with leucoderma. The pigmentation may vary considerably in degree even within quite short periods of time. The patient often feels worse when the skin is darker, and *vice versa*. In some cases the pigmentation may be like that of the darker races, but it is noticeable that the nails are quite free from darkening. Often the bronzing is like that due to sunburn, while sometimes the skin merely looks dirty.

The pigment, melanin, is that normally present in the stratum Malpighii of the epidermis, only it is manufactured in larger quantities; the material from which it is formed is apparently conveyed to the stratum Malpighii from the blood-vessels of the corium by 'carrier cells.'

Pigmentation of the mucous membrane of the lips, the tongue, and inside of the mouth occurs much less frequently and appears to be due to some manifest irritation, such as carious teeth. The conjunctivæ are usually pale and contrast with the bronzed skin of the face, but in rare instances pigmentation has been noticed in them.

From time to time the patient has attacks in which all the symptoms are exaggerated; sometimes the pulse becomes extremely feeble and with difficulty felt at the wrist; vomiting or, rarely, diarrhoea may become uncontrollable and kill the patient. If the patient survive an attack he rallies, but is left permanently weaker. As time goes on the general condition deteriorates until a final exacerbation carries him off; sometimes death occurs suddenly, with little warning, from syncope.

Death may be preceded by convulsions, by delirium, by coma, or by a period of such extreme weakness that although quite conscious the patient can hardly make any sign of life.

DURATION AND PROGNOSIS.—The *duration* of the disease varies very widely: the extremes are ten years in the most prolonged cases and a few days in fulminating or acute cases. In chronic cases the disease may become latent, and it is possible that it may sometimes remain so: at any rate cases so diagnosed by most competent authorities have been known to live for very many years and die from other causes. In hospital cases the average duration is said to be about two years, while in persons better provided with the comforts and luxuries of life five or six years may elapse between the onset of symptoms and a fatal termination.

The *prognosis* is very bad; when the disease is sufficiently marked to be definitely diagnosed, a fatal termination must sooner or later be expected. The course of the disease is, however, very irregular, and intervals of fair health may sometimes occur even when the patient has been extremely weak. On the other hand, death may occur suddenly from syncope early in the course of the disease. Progressive loss of strength and shortness of breath with increasing apathy are bad symptoms. There is some reason to think that the outlook is better in those cases that improve under suprarenal extract.

DIAGNOSIS.—In well-marked cases there is little difficulty. The association of extreme debility with the characteristic pigmentation in the absence of any discoverable cause should always suggest the disease. In cases without pigmentation the diagnosis is difficult and is made by a process of exclusion; pernicious and other anæmias should be eliminated by examination of the blood; latent malignant disease of the stomach might be suggested by the ascertained absence of hydrochloric acid from the gastric contents. Pigmentation of the skin occurs in a number of conditions, but is not necessarily associated with debility and gastro-intestinal symptoms. In a woman pigmentation and vomiting may occur in pregnancy. In hæmochromatosis the pigmentation may imitate that of Addison's disease, but there is usually diabetes mellitus (*diabète bronzé*), and frequently enlargement and cirrhosis of the liver at the same time. In malarial cachexia melanæmia and melanoderma may occur, but the history, the enlarged spleen, and the examination of the blood should prevent mistake. In phthisis, chronic peritonitis, and granular kidney, the face may be considerably pigmented. In exophthalmic goitre, rheumatoid arthritis, syphilis, pellagra, and extensive melanotic sarcoma of the liver, the skin is occasionally markedly pigmented, but the other features of the primary disease can hardly be overlooked. In *acanthosis nigricans* the skin is warty and rough as well as pigmented and is thus distinguished from Addison's disease. See **ACANTHOSIS NIGRICANS**.

The dirty colour of the skin due to past jaundice

is hardly likely to be mistaken for the bronzing of Addison's disease. Long-continued irritation of the skin by pediculi, combined with abstinence from washing (vagabond's disease), imitates the pigmentation of Addison's disease, but yields to soap and water.

Men employed in gas- and tar-works, or those much exposed to heat and weather, naturally become bronzed, while a long course of silver (argyria) or arsenic may produce very noticeable pigmentation.

TREATMENT.—When the disease is diagnosed the patient should adopt a quiet life and retire from work that makes any demands on his physical or mental powers. He should live in the country in a sunny bracing place protected from cold and east wind and not subject to rapid alterations of temperature. When the general condition is good, gentle exercise may be allowed, but fatigue, worry, and strain must be carefully avoided. Any faintness should be the signal for complete rest in the horizontal position for some days.

The *Diet* should be simple and nutritious: meat should be given, and stimulants when required. When vomiting occurs the diet must be modified so as to minimise the irritation of the stomach and to keep up the patient's failing strength. Ice may be sucked before food; liquid and predigested food should be given cold in small quantities and at more frequent intervals; and iced champagne may be employed as a stimulant.

Medicinal treatment may be divided into (1) the administration of suprarenal gland-substance and (2) symptomatic treatment.

Suprarenal gland-substance is usually given in the form of an extract; but the glands of sheep have been given in a sandwich. Oliver and Schäfer have shown that the physiologically active extract, which is only obtained from the medulla of the suprarenal bodies, is not destroyed by contact with gastric juice *in vitro*, and hence the extract has been largely given by the mouth. Nevertheless it is not by any means certain that suprarenal extract taken by the mouth is so effective as hypodermic injection. When given by the mouth a quantity of the extract equal to 15 grains of the gland should be given twice a day at first, and gradually increased until an amount equal to two drachms of the gland substance is taken daily. If any stiffness of the muscles, sickness, or depression arise, the administration of the extract should be temporarily stopped, though ill effects can but very rarely be traced to the extract. Hypodermic injections should be tried, if administration by the mouth fails: the initial dose should be about one-third of that given by the mouth and should be increased cautiously. The effects of this form of treatment are disappointing as compared with those of thyroid-extract in myxœdema; temporary improvement, sometimes of a very striking nature, may occur, but relapses are not prevented by it.

Tonics such as iron, strychnine, and arsenic may be given, and, if it can be borne, an emulsion of cod-liver oil. Vomiting should be combated by salts of bismuth, oxalate of cerium, and small hypodermic injections [$\frac{1}{40}$ – $\frac{1}{20}$ gr.] of morphine. Diarrhoea should be controlled by opium and bismuth. If constipation occur, mild purgatives only must be employed, since vigorous treatment in this direction may be followed by severe symptoms. It does not appear that surgical treatment can be invoked with much likelihood of success. H. D. ROLLESTON.

ADENALGIA (ἀδὴν, a gland; and ἄλγος, pain). Pain in a gland.

ADENITIS.—Inflammation of a gland. See the several glands.

ADENOCELE (ἀδὴν, a gland; and κήλη, a tumour).—A tumour connected with a gland.

ADENODYNIA (ἀδὴν, a gland; and ὀδύνη, pain).—Pain in a gland.

ADENOID (ἀδὴν, a gland; and εἶδος, form).—Glandular: resembling the structure of a gland, whether secreting or lymphatic. The term 'adenoids' is often used to denote certain post-nasal growths. See NOSE, Diseases of.

ADENOMA (ἀδὴν, a gland, and the termination *oma*, adopted to indicate a tumour).—A morbid growth, the structure of which is of glandular nature. See TUMOURS.

ADENOMA SEBACEUM.—SYNON.: *Nævus sebaceus* (Bandler); *Excrescences vasculaires* (Kayer); *Nævus pilosus cornutus* (Darier).

DEFINITION.—Multiple tumours consisting of hypertrophied sebaceous glands, situated on the face, and of congenital origin, although not usually present or noticed at birth.

ÆTIOLOGY.—The disease is congenital, but is seldom noticed before the sixth or seventh year, and often not till puberty, when it usually becomes much aggravated. The growths, which are purely hypertrophic in character, arise from isolated portions of the germinal epithelium, thus justifying the contention of Bandler that they ought to be classified as forms of *nævus*, using the word in its broadest sense. In a very large proportion of cases the patients are mentally defective, many being epileptics, hystero-epileptics, or idiots. Such patients are usually relegated to asylums, and the existence of the disease, which is by no means extremely rare, often passes unrecognised. Other nævoid conditions of skin are usually co-existent, e.g. warts, moles, vascular, hairy, or pigmentary nævi, and fibromata, often in great abundance. In exceptional cases analogous hypertrophic changes, both as regards number and size, occur in the sweat-glands, constituting, according to Radcliffe Crocker, a 'pilo-sebaceous hidradenoma.'

SYMPTOMS.—The disease manifests itself as little nodules or tumours situated on the face and in those regions of it where the sebaceous glands exist in greatest abundance. Thus they are most numerous and most closely crowded together on the nose and naso-labial folds; less so on the cheeks and chin; and least of all on the forehead. The distribution is, therefore, much the same as that of rosacea, which sometimes complicates adenoma sebaceum, increasing the disfigurement materially. The essential nodules vary in size from a pin's point to a split pea; they are firm and painless, standing out boldly from the skin-surface. Although closely grouped together they never coalesce to form plaques. The epidermis over them is generally smooth, but exceptionally harsh and even warty. When the nodules are pricked and squeezed, inspissated sebum emerges. In uncomplicated cases the little tumours are waxy, yellowish or brownish in tint; but in almost all cases there is much vascular telangiectasis both over and around the sebaceous tumours, so that the affected area assumes

a pink or even vivid red colour. When these blood-vascular changes are abundant—and they may even mask the essential lesions—the disease is said to be of the 'telangiectatic type.'

PATHOLOGICAL ANATOMY.—The epidermis is usually normal, but its interpapillary prolongations may be hypertrophied. The increased thickness of the cutis is due to a great increase in size and complexity of the sebaceous glands, the cells of which are, however, of normal size, and functionally active. Around the growths there is no small-celled infiltration or other inflammatory or hyperplastic change.

PROGNOSIS.—Adenoma sebaceum usually attains its maximum of intensity at or about puberty, after which it remains unaltered as a rule. Occasionally spontaneous absorption or cystic degeneration in the centre of the nodules may occur, leaving depressed scars; but such occurrences are rare. The subjects of the disease are, naturally, almost always short-lived.

DIAGNOSIS.—The disease may be simulated by xanthoma, adenoma of sweat-glands, colloid milium, lymphangioma or even nodular lupus vulgaris. Excision and microscopical examination of a nodule will readily clear up all doubt.

Unilateral or asymmetrical patches of hypertrophied sebaceous glands may arise in adult life and in persons of normal intelligence, but the writer is of opinion that they ought to be carefully differentiated from adenoma sebaceum.

TREATMENT.—Discrete growths may sometimes be satisfactorily destroyed by electrolysis, the negative pole and a current of about five milliamperes being employed. More closely grouped lesions may be occasionally successfully treated by repeated scraping, curetting, and scarification; or more localised patches may be excised, Thiersch-grafts being afterwards applied. See SKIN-GRAFTING.

J. J. PRINGLE.

ADHESIONS.—Structures are said to be *adherent* when they become abnormally united together, the morbid formations by which this union is effected being termed *adhesions*. These are most frequently met with in connection with serous surfaces, being usually the result of an inflammatory process, but they may be observed in other structures. Adhesions vary considerably in extent, number, mode of arrangement, firmness, and other characters: they may merely consist of a few loose, slender, and delicate bands; or these bands may be thick and strong; or the contiguous surfaces may be blended and matted together to a greater or less extent, so that they cannot be separated without tearing or cutting them asunder. In structure adhesions consist of fibrous tissue.

EFFECTS.—Adhesions are often found at post-mortem examinations, which have been of little or no consequence during life, as, for instance, many of those which form in connection with the pleural surfaces. If, however, they are extensive and firm, or if they occupy certain regions of the body, they may prove of serious moment. The principal evils which are liable to result from adhesions may be thus indicated:—1. They often bind parts together, and interfere with the movements of important organs, such as the lungs, heart, stomach, or intestines; in this way preventing the due performance of their functions. 2. When an organ is displaced in any way, as, for example, the heart by

pleuritic effusion, it may become fixed in its new position by the formation of adhesions, its functions being thus disturbed. 3. It is highly probable that extensive adhesions may lead to hypertrophy of an organ, e.g. the heart, by embarrassing its movements, and hence affecting its action. 4. On the other hand, atrophy or degeneration of structure may ensue, in consequence of the adhesions interfering with the due supply of blood by pressing upon the vessels, so that the nutrition of the tissues becomes impaired. In the young, also, their development may be checked. 5. Adhesions may involve important structures, such as nerves or vessels, pressing upon or destroying them, thus giving rise to symptoms of a serious nature. 6. Tubes or canals for the passage of secretions or other materials are sometimes narrowed or obliterated by adhesions. 7. When formed within the abdominal cavity, especially when they take the form of bands, adhesions may prove highly dangerous by compressing, constricting, exerting traction upon, or strangulating some portion of the intestine, in either of these ways leading to intestinal obstruction.

It is frequently difficult or impossible to determine the existence of adhesions by clinical investigation during life; but the history of some past illness during which they were likely to be formed, the results of physical examination, especially in connection with the heart and lungs, and the symptoms present, not uncommonly enable them to be discovered. FREDERICK T. ROBERTS.

ADIPOCERE (*adeps*, fat; and *cera*, wax).—

SYNON.: Fr. *Adipocire*; Ger. *Fettwachs*.

DEFINITION.—A fatty substance formed in the process of decomposition of animal bodies in the presence of water.

DESCRIPTION.—In appearance adipocere is a waxy substance, varying in colour from white to darkish brown. When moist it is soft and greasy; when dry it 'somewhat resembles spermaceti in appearance, but is less crystalline in fracture' (Quain). Traces of the structure of the parts from which it has been formed may be present in the form of blood-vessels, muscular fibres, or connective tissue: to such is due the reticulum of insoluble matter left when adipocere is dissolved in ether. It has a peculiar unpleasant odour, which becomes stronger on heating. Its density is lower than that of water.

CHEMICAL COMPOSITION.—Adipocere is probably not a definite chemical compound, but a mixture of different substances, the proportions of which may vary according to the conditions of its formation. In this way the differences observed in its colour and consistency may be explained. Analysis by Chevreul showed it to consist of an ammoniacal soap, containing traces of colouring matter, of a bitter principle, and of a volatile odorous substance. The fatty acids present are palmitic, stearic, and oleic, the first of these being in great excess, while the oleic acid which forms so large a constituent of human fat is found in very small quantity (Wetherill). In many specimens which have been formed in water containing a large amount of calcium-salts, a quantity of lime-soap is found along with the ammoniacal compound. In such cases the lime is probably a substitution-product, replacing the ammonia in the previously formed adipocere (Orfila).

METHOD OF FORMATION.—In order that formation of adipocere (saponification) may take place, it is necessary that there should be present not only fatty acids, derived from the breaking up of animal fat, but also a supply of ammonia to act as a base. The latter is derived from the decomposition of the proteid portions of the body. The fatty acids are mainly derived from the normally existing adipose tissue, but parts which contain little fat may also be converted into adipocere. In this case the proteid-molecule probably breaks down and gives rise to a fatty substance, which is subsequently saponified. The presence of water in considerable quantity is necessary for the formation of adipocere, which therefore occurs in bodies which have been buried in damp soil or left lying in ponds or rivers. Warmth is favourable to the process, which takes place with great rapidity in tropical countries. The outer parts of the body suffer first, and the process spreads irregularly inwards. Portions containing much fat, such as the buttocks, breasts, and hollows of the cheeks, are rapidly saponified: the abdominal viscera are next affected, and the muscles still later. Fat persons and children are favourable subjects for this form of change, which is also facilitated by the existence of other decomposing matter in the neighbourhood, whereby a free supply of ammonia is provided. Thus bodies which have lain in a cesspool or in a crowded damp graveyard will be readily saponified. Since bacterial action is necessary for the decomposition of animal bodies, it is evident that it is a necessary factor in the production of adipocere.

MEDICO-LEGAL IMPORTANCE.—When once formed adipocere remains unaltered for an indefinite period of time. It thus tends to preserve the features of dead bodies from the effects of decomposition, and to render identification possible. It also perpetuates the exact nature of any wounds or injuries received, by causing the parts to maintain their shape and mutual relations. Its formation is further of some importance as an aid to estimating the length of time which has elapsed since death. Unfortunately no very definite limits of time can be laid down for the commencement and progress of saponification. In India instances have been observed in which large portions of dead bodies were converted into adipocere in two or three days (Mackenzie). In temperate climates it is probable that little saponification will be found in less than five or six weeks. An instance is, however, quoted by Taylor in which a body which had lain in running water was converted into adipocere in five weeks, so that the change in this case must have begun much earlier. It is necessary to take into consideration the conditions to which a body has been subjected in order to estimate the rapidity with which adipocere may be formed. In hot weather, in shallow water and in presence of decomposing animal matter, superficial parts might be saponified in temperate climates in two or three weeks, but total conversion of soft parts into adipocere would seldom occur under ordinary conditions in less than three or four months, probably because the conditions of moisture necessary for its formation are usually here associated with a degree of cold by which decomposition is materially retarded. In the majority of cases the process is considerably slower even than this, and years may elapse before saponification is complete.

WM. CECIL BOSANQUET.

ADIPOSIS.—A term which properly signifies either general corpulency, or accumulation of adipose tissue in or upon an organ. See **FATTY GROWTH**; and **OBESITY**.

ADIPOSIS DOLOROSA.—A disease of middle life, occurring principally in women, characterised by the formation of irregular masses of subcutaneous fat, and occasionally by degenerative changes in the nerves of the affected areas and in the thyroid gland. The symptoms are pain and tenderness, and in some cases impaired sensibility. The administration of thyroid extract is occasionally beneficial.

ADIRONDACK MOUNTAINS, In New York State.—A series of ranges rising from a plateau 2,000 feet above the sea, with picturesque lakes between them. Highest altitude, Marcy, 5,337 feet. Commodious hotels on the shores of the lakes. Much frequented as a summer climatic resort. Recommended in phthisis. See **CLIMATE**, Treatment of Disease by.

ADRENALS. See **SUPRARENAL GLANDS**.

ADYNAMIC (ἀ, priv.; and δύναμις, power).—A term indicating serious depression of the vital powers. The adjective is applied to diseases in which the so-called 'typhoid condition' is especially liable to occur. See **TYPHOID STATE**.

ÆGOPHONY (αἴξ, a goat; and φωνή, voice).—A peculiar alteration of the resonance of the voice, as heard on auscultation of the chest, compared to the bleating of a goat. See **PHYSICAL EXAMINATION**.

ÆTIOLOGY (αἰτία, cause; and λόγος, word).—That branch of pathological science which deals with the causation of disease.

AFFUSION.—A method of treatment which consists in pouring a fluid, usually water, either cold or warm, upon some part of the patient. See **BATHS**; and **WATER**, Therapeutics of.

AFRICA, SOUTH.—See **SOUTH AFRICA**.

AGEUSIA (ἀ, priv.; and γεῦσις, taste).—Loss of taste. See **TASTE**, Disorders of.

AGGLUTININS.—Substances causing the agglutination of bacteria. See **IMMUNITY**.

AGONY (ἀγών, strife or struggle).—Agony implies bodily pain or mental suffering so intense that it cannot be endured, but excites a struggle against it. It is also applied to the final struggle that may precede death.

AGORAPHOBIA (ἀγορά, a market-place, hence any open space; φόβος, fear).—**SYNON.** : **Fr.** *La peur des espaces*; **Ger.** *Platzangst*.—A morbid emotional state characterised by a feeling of fear at any open space. A person thus affected may be unable to cross a street or a square alone, or even in some cases to leave his room. It is accompanied by palpitation, tremblings, cold perspiration, a feeling of weakness in the legs, and sometimes by fainting. See **IMPERATIVE IDEAS**.

AGRAPHIA (ἀ, priv.; and γράφω, I write).—This term is applied to defects of intellectual expression by writing. These defects may occur alone, or in association with defects of speech, according to the extent and situation of the lesion. See **APHASIA**.

AGUE.—A popular synonym for malarial fever. See **MALARIA**.

AGUE-CAKE.—A form of enlargement of the spleen, resulting from the action of *Hamatozoon malariae*. See **SPLEEN**, Diseases of; and **MALARIA**.

AIKIN, In south-west division of South Carolina.—A mild, bracing, dry climate, said to resemble that of Mentone in warmth and dryness, but with larger monthly temperature-range. Soil, sandy. Recommended for pulmonary affections. See **CLIMATE**, Treatment of Disease by.

AINHUM (Nat., to saw).—This disease is peculiar to the dark-skinned races, being found not only among the inhabitants of tropical Africa, where it is very common, but also among the Hindoos, as well as among the negro population of America, especially of South America. At its commencement, a groove or furrow is seen at the base of the little toe (the part almost invariably attacked), situated on its inner and inferior aspect, and corresponding to the digito-plantar fold. The furrow soon extends to the entire circumference of the toe; and, as it becomes gradually deeper, the latter is left hanging by a slender pedicle, which can only be brought into view by separating the walls of the furrow. The distal portion swells into an ovoid mass, about twice its natural size; finally some accident snaps the pedicle, and the toe drops off in from four to ten years from the commencement of the disease.

The furrow is caused by a constricting band of hardened and contracted skin—a local scleroderma—which leads to faulty nutrition and degenerative changes in the parts beyond.

Ainhum is not a painful affection in itself, but the extreme mobility of the little toe causes trouble and inconvenience, for which patients often seek relief in amputation.

Occasionally the sides and bottom of the furrow ulcerate. Not infrequently both little toes are attacked by the disease; occasionally the other toes become similarly affected. Males are more subject to it than females. The microscope reveals only hypertrophy of the fibrous tissues at the seat of constriction, and atrophic and degenerative changes in the distal portion of the toe. The cause of ainhum is entirely obscure. Treatment by early division of the constricting band has been suggested.

PATRICK MANSON.

AIR, Therapeutic use of.—Atmospheric air is employed in the treatment of disease in many ways and for many purposes. The application to the body generally of air that has been warmed, or warmed and loaded with moisture, will be found described in the article on **BATHS**; while its administration to the respiratory organs, either in this form or as a vehicle for such substances as creasote, carbolic acid, alkaloids, and sulphurous acid, will be discussed under **INHALATIONS**.

GENERAL AËROTHERAPEUTICS.—The effects of compressed air on the body as a whole have been studied in the *air-bath*. This is a mechanical arrangement in the form of an iron chamber, which can be filled with air at any pressure, whether above or below the normal, by means of steam-power.

The principal physiological effects of air condensed by three-sevenths of an atmosphere were ascertained by von Vivenot to be:—Pallor of the skin and mucous membranes; a sensation of pressure in the ears;

diminished frequency of respiration, the act becoming easier; enlargement of the lungs and increase of the vital capacity; depression of the cardiac force, and diminution of the size and strength of the pulse; rise of temperature; increased vigour of muscular action, secretion, and nutrition generally; compression of the gaseous contents of the intestines; and, perhaps, increased absorption of oxygen and excretion of carbonic acid. When the pressure is excessive, dangerous or even fatal symptoms may supervene (see CAISSON DISEASE). Frequent exposure to condensed air will induce considerable increase of the vital capacity; and most of the other effects, both physical and chemical, will tend to persist. In a word, it may probably be said that the air-bath acts on the system, first, by increasing the general mechanical pressure; and, secondly, by admitting an increased amount of oxygen. In employing the air-bath, the patient is kept in it for a period of two hours, at first daily, but after some weeks less frequently. The pressure, which is employed in different cases at one-fifth to two-thirds of an atmosphere above the normal, must be slowly raised on admission, and reduced on removal of the patient.

Uses.—The number of diseases in which the air-bath may be employed is limited: (1) In certain forms of dyspnoea. It gives great relief in *spasmodic asthma*, and may also afford temporary relief in *emphysema*; but its prolonged use appears to be positively injurious, as it increases the pulmonary distension. (2) In hyperemia and *catarrh of the air-passages*, including pertussis. (3) In imperfect expansion or threatened retraction of the chest, as in the subjects of phthisis and chronic pleurisy. In these cases it is of doubtful service. Compressed air has also been extolled in some forms of cardiac disease, and in general malnutrition.

Artificially rarefied air is never employed in the form of the bath; but the natural supply in elevated regions has found favour as a means of treatment. See CLIMATE and PHTHISIS.

LOCAL AÉROTHERAPEUTICS.—When it is desired to bring compressed or rarefied air into contact with the *respiratory* surface only, other apparatus must be employed. Different forms have been in use for some years, respecting which it will be sufficient to state that the air contained in a portable gas-holder is compressed or rarefied by simple mechanical means, and thereafter brought into relation with the air-passages by an arrangement of tubes and valves. There are four possible methods of application: (1) inspiration of condensed air; (2) expiration into condensed air; (3) inspiration of rarefied air; and (4) expiration into rarefied air.

Another apparatus, called the *pneumatic cabinet*, has been introduced more recently. It consists of an air-bath, built of steel and glass, to accommodate both the patient and the administrator. The disturbance of pressure within the chest is effected by rarefying the air in the cabinet by means of a bellows, and then allowing the patient to inspire from the external atmosphere through a tube from without. This procedure is in effect mainly inspiration of a relatively condensed air.

The *physiological effects* of the several methods of application may now be briefly stated.

Inspiration of condensed air.—Inspiration of air that has been condensed by one sixtieth to one fortieth of an atmosphere produces a sensation of extreme distension of the chest, accompanied by an actual expansion of the thorax and lungs, and an

increased admission of air, so that inspiratory dyspnoea, if present, is relieved. At the same time the other thoracic contents are compressed, the systemic vessels fill, the arterial pressure rises, and the jugular veins become distended. The lungs and heart will be comparatively anæmic. If the application of condensed air be frequently repeated, the vital capacity, the size of the chest, and the respiratory force may all be increased, and partial relief may be permanently afforded to dyspnoea.

Expiration into condensed air is most difficult of accomplishment, and the effect on the circulation does not differ essentially from that just described.

Inspiration of rarefied air.—Inspiration of air that has been rarefied by one two-hundred-and-fortieth to one one-hundred-and-twentieth—or even, after a time, by one sixtieth—of an atmosphere, immediately causes the phenomena of inspiratory dyspnoea; the thoracic viscera are congested, and hæmoptysis may result, for the effect may be regarded as that of dry-cupping the pulmonary alveoli. The heart at the same time becomes full, and the jugular veins collapse.

Expiration into air that has been rarefied by one sixtieth of an atmosphere is attended with a sense of extreme compression of the thorax; at the same time there is actually a partial retraction of the lungs, an increase in the volume of expired air, and a corresponding diminution in the amount of residual air in the chest. Expiratory dyspnoea, if present, is relieved. While the lungs thus diminish in size, the other thoracic viscera are dilated—the heart and the pulmonary and other vessels within the chest being filled at the expense of those external to it, both arteries and veins. If the expiration into rarefied air be frequently repeated, the circumference of the chest will be diminished, while the vital capacity will be actually increased, along with increase of the inspiratory and expiratory force.

Uses.—The method of *inspiring condensed air* is obviously indicated in diseases where inspiratory dyspnoea is an urgent symptom. Spasmodic asthma, stenosis of the air-passages from anatomical causes, acute and chronic bronchitis, and atelectasis, have all been successfully treated by this method. In croup, where urgently indicated, it is most difficult or even impossible to employ it. In threatened phthisis it is used prophylactically, and in chronic phthisis it may usefully develop the healthy portions of lung; but it is contra-indicated in pyrexial cases, and may prove dangerous by inducing hæmoptysis. In chronic pleurisy it may prevent or remove the effects of collapse and retraction of the chest-wall. The inhalation of condensed air should also be of use in certain forms of cardiac dilatation, especially that due to mitral incompetence. Improving, as it does, the general nutrition, it may be combined with other remedies for anæmia. In the administration of condensed air, a 'sitting' should last from ten to thirty minutes, once a day—seldom twice.

Expiration into condensed air is not used therapeutically.

Inspiration of rarefied air may be regarded as a means of exercising the inspiratory muscles. Like the atmosphere of great altitudes, it may therefore be employed in persons with badly developed chests; and even in phthisis it may assist, by increasing the amount of blood in the lungs. In disease of the right side of the heart, it would assist the flow of blood from the veins into the lungs, but it is not likely to be employed for this purpose.

Expiration into rarefied air promises to be the most successful and most extensively employed of all the methods. In it, according to Waldenburg, we have the physical antidote for emphysema, and in his hands the majority of such patients are said to have been either cured or radically benefited. It has also afforded great relief in some cases of bronchitis, where it increases expectoration.

Other local applications of the physical properties of the air, as seen in aspiration, cupping, Junod's boot, and inflation, are described elsewhere in this work. See OXYGEN, Treatment by.

J. MITCHELL BRUCE.

AIR-PASSAGES, Diseases of.—See RESPIRATORY ORGANS, Diseases of; also LARYNX, TRACHEA, and BRONCHI, Diseases of.

AIX-LA-CHAPELLE, Waters of.—Thermal sulphur waters. See MINERAL WATERS.

AIX-LES-BAINS, Waters of.—Thermal sulphur waters. See MINERAL WATERS.

AKINESIA (*ἀ*, priv.; and *κίνησις*, motion).—A synonym for paralysis of motion, whether partial or general. See PARALYSIS, MOTOR.

ALASSIO.—In Italy on the Mediterranean coast, between San Remo and Genoa. A mild bracing winter resort, sheltered by hills except on the south and east. Comparatively free from wind and dust. See CLIMATE, Treatment of Disease by.

ALBINISM (*albus*, white).—**SYNON.**: Congenital Leucoderma; Congenital Achroma; Albinismus.—**DEFINITION.**—A state of whiteness or absence of colour of the integument and certain other tissues, consequent on congenital absence of pigment and unaccompanied by structural cutaneous change. The want of colour may be *complete* or *incomplete*; *partial* or *universal*; *congenital* or *accidental*. Partial albinism may be limited to a spot of small dimensions; or there may be many such spots of variable extent, dispersed over the surface of the body, and giving rise to the appearance which is denominated *pie'd* or *piebald*; whereas in universal albinism the defect of pigment is not restricted to the integument, but is especially remarkable in the iris and choroid and the hair.

GENERAL CHARACTERS.—Persons affected with universal albinism are called *albinos*. It would seem more correct to limit the term 'albino' to those in whom the absence or defect of pigment is universal, and demonstrable not only in the integument but likewise in the eyeball. In the true albino, therefore, the skin is white or pinkish (when thin enough for its vessels to show through) and more or less transparent, and this both in the fairer and in the darker races of mankind; but in certain of the latter, where the pigmentary function is simply defective and not totally wanting, the colour of the skin may be grey, and more or less variegated and freckled. The long, soft, fine hair is of a pure silvery or white colour, may be diversely tinged, or may be red (Folker); and in some instances the whole body is covered with a white down. The iris is pinkish, in accordance with the density of its fibrous structure, and the consequent facility of penetration of the colour of its vascular layer; or, as generally happens in the negro, it is blue. The pupil is contracted and brightly red, from the

absence of the screen of protection usually afforded to the choroid membrane by its pigmentary layer; and for the same reason the rays of light penetrating the sclerotic and iris give a brilliancy of appearance to the fundus oculi. The absence of pigment in the eyeball is productive of several peculiarities of character in the albino. In the first place the excess of luminous rays penetrating the coats of the eyeball interferes with the correctness of vision; he stoops his head, or droops his eyelids, to shelter his eyes; he sees with more comfort in the dimness of evening than in sunlight; his vision is more or less imperfect; and nystagmus, nictitation, and photophobia are present in various degrees.

ÆTIOLOGY.—Certain animals, as rats, mice, ferrets, and some birds, are subject to albinism, and the affection may be met with in several members of the same family. Albinism is met with among all races of mankind and in every country, but is most common among those who are subjected to insalubrious conditions of climate and hygiene. For these reasons it is not uncommon among the natives of the marshy coast of Africa; among negroes who are transferred to unhealthy districts in South America and the West Indies; among the inhabitants of the western coast of South America and Mexico; in certain of the islands of the Indian Ocean; and even in the northern regions of Europe. When albinism is congenital, it has been assumed to be due to an arrest of development: but when accidental, its existence must be referred to exhaustion of chromatogenous or pigment-producing function. Albinos are usually mentally and bodily weak, but it is well known that albinism is often associated with perfect physical strength and intellectual vigour. Albinism exceptionally is hereditary. The usual sites of partial albinism are the mammæ, face, scalp, and genitals. The patches vary in size; their margins are well or ill defined; and they are asymmetrical, and on the non-pigmented areas the hairs are white. See PIGMENTARY DISEASES OF THE SKIN: (1) *Nævus Pigmentosus*.

TREATMENT.—Albinism baffles treatment.

JOHN HAROLD.

ALBUMENS.—**DEFINITION.**—Albumens are substances closely resembling egg-albumen, the chief constituent of white of egg or albumen. To distinguish between the white of egg and its chief constituent, the latter is sometimes spelt *albumin*. Albumens constitute a subdivision of the class of albuminous bodies, which includes all substances having a general resemblance to albumen. See ALBUMOSES.

ENUMERATION.—The sub-class properly contains only two members, *egg-albumen* and *serum-albumen*; but the name *Bence-Jones's albumen* has been given to an albuminous body differing very considerably in its properties from the other two.

CHARACTERS.—Egg-albumen and serum-albumen are semi-transparent, yellowish, and structureless when dried. They are soluble in water; and this solution is coagulated by boiling. From the same solution they are precipitated by: (a) nitric acid; (b) salts of the heavy metals, for example, copper-sulphate; (c) acetic acid with potassium-ferrocyanide; (d) boiling with acetic acid and a neutral salt, for example, potassium-sulphate; (e) alcohol. Egg-albumen is distinguished from serum-albumen by the coagulum which it forms with nitric acid being insoluble in excess, while that of serum-

albumen is soluble. Bence-Jones's albumen gives no precipitate with excess of nitric acid unless left to stand, or unless heated and left to cool, when it forms a solid coagulum. This coagulum redissolves on heating, and again forms on cooling. It is therefore an albumose (see ALBUMOSES). It may be separated from ordinary albumen by adding nitric acid, boiling, and filtering when hot. The ordinary albumen will remain on the filter while Bence-Jones's albumen will pass through, and will coagulate when the filtrate cools.

MODIFICATIONS.—By the action of acids and alkalis albumen may be converted into *acid-albumen* and *alkali-albumen* respectively, neither of which is coagulated by boiling.

Acid-albumen may be formed in two ways: First, by dissolving solid albumen in concentrated nitric or other mineral acid with the aid of heat. Secondly, by heating an aqueous solution of albumen with one of these acids very much diluted (1 in 500). Although soluble in very concentrated or very dilute acids, acid-albumen is insoluble in moderately dilute acids. Therefore when the solution in concentrated nitric acid is diluted with water, a precipitate is formed, which redissolves when much water is added. And, conversely, when acid-albumen is made by boiling a solution of albumen in water with very dilute nitric acid, the addition of more acid will throw down a precipitate, which redissolves if a very large excess of the concentrated acid be added, and especially if it be heated at the same time. On neutralising a solution of acid-albumen, a precipitate is thrown down, which dissolves very readily in excess of alkali.

Alkali-albumen, or *alkali-albuminate* as it is also called, is formed by dissolving albumen in caustic potash or soda; or by adding either of these to its aqueous solution and allowing this to stand, or heating it. This modification is not precipitated by heat, but is precipitated by neutralisation; the precipitate dissolving very readily in slight excess of acid. If alkaline phosphates are present in the solution, as they are in urine, alkali-albumen requires a slight excess of acid to throw it down, and is not precipitated by exact neutralisation, as acid-albumen would be under similar circumstances.

T. LAUDER BRUNTON.

ALBUMINOID DISEASE.—See AMYLOID DISEASE.

ALBUMINURIA.—**DEFINITION.**—A condition characterised by the presence of albumen in the urine. Other albuminous bodies, not albumens, may be present in albumosuria, hæmoglobinuria, hæmaturia, peptonuria, pyuria, and spermatorrhœa.

SYMPTOMS.—Albumen may occur in the urine without any symptoms whatever, but its continuous loss leads to anæmia and changes in the circulation, which usually originate a series of symptoms. These are: a pallid pasty complexion, dry skin, and tendency to œdema of the cellular tissue, noticeable on the eyelids and ankles; derangement of digestion, flatulence, occasional nausea, and irregularity of the bowels; nervous disorder, shown by muscular weakness, languor, lassitude, vague pains about the loins, and headache; calls to make water during the night; attacks of difficult breathing; palpitation, and frequently accentuation of the second sound of the heart over the aortic cartilage, and reduplication of the first sound over the septum ventriculorum.

TESTS FOR ALBUMEN.—The two tests usually employed to detect albumen in the urine are—first, boiling; and, secondly, the addition of nitric acid; both of which produce a cloud or precipitate. If the urine is turbid, the albuminous cloud may not be noticed; and therefore such urine should be filtered before the application of either test, unless the turbidity, being dependent on the presence of urates, is removed by heat cautiously applied.

Method of employing the test by boiling.—With the object of saving time the urine is often boiled at once, but the results thus obtained are liable to several fallacies, which will be subsequently described. In order to avoid such fallacies the following method should be pursued:—Ascertain the reaction of the urine; and if it be alkaline or very strongly acid, add acetic acid in the one case, or liquor potassæ in the other, until its reaction is only slightly acid. Fill a test-tube to about one-third of its capacity with the urine, and hold it obliquely in the flame of a spirit-lamp, in such a manner as to heat the upper part of the fluid only, until it boils. If it be turbid from urates, it should be first warmed throughout until it becomes clear, and then the upper part only should be boiled. Finally, add a drop or two of acetic or nitric acid.

If albumen be present, it will form a cloud or a coagulum, more or less dense according to its amount. When there is much albumen, its quantity may be roughly estimated by allowing the boiled urine to stand for a definite number of hours, so that the coagulum may subside, and then observing whether it forms a fourth, a third, or a half of the whole length of urine in the test-tube. A small quantity causes a cloud, but no distinct coagulum; and, if merely a trace be present, a faint haze only will be observed, which is best seen by looking through the test-tube at a dark object. The advantage of heating the upper part only of the urine is, that the lower portion, which remains clear, affords a standard by comparison with which a faint cloud in the heated part may be more readily detected.

Fallacies of the test by boiling.—The first fallacy is that albumen may be present, and yet no cloud or coagulum be produced on boiling. This may occur if the urine be alkaline or very strongly acid, because alkali-albumen or acid-albumen, which are soluble in water, may be formed. It is to prevent the formation of *alkali-albumen* that acetic or nitric acid should be added to alkaline urine before boiling. This addition of acid also causes the coagulum to separate more readily; and it should therefore be made when the urine is neutral. On the other hand, urine rarely or never contains sufficient acid to form *acid-albumen*, unless the patient has been taking mineral acids; and therefore the addition of liquor potassæ is not necessary except under these circumstances. The second fallacy of the test by boiling is, that a cloud resembling that of albumen may be produced, although the urine is free from this substance. This occurs when the acidity of the urine is too slight to hold the earthy phosphates in solution, the heat probably affecting the relation between the basic and acid phosphates which are normally in solution, whereby deposition of insoluble phosphate results, forming a cloud like that of albumen. The two clouds are readily distinguished by the addition of a drop or two of acetic or of nitric acid, when if due to phosphates the cloud will disappear by solution, but if caused by albumen it will remain. If an excessive quantity of nitric acid

be added, an albuminous cloud may also clear up; for albumen coagulated by heat is soluble in strong acid, though only to a slight extent.

Application of the nitric acid test.—Pour some urine into a test-tube, and then allow about one-fourth of its bulk of strong colourless nitric acid to trickle slowly down the side of the tube, so as to form a layer below the urine without mixing. Or the acid may be put in the test-tube first, and the urine poured on it. Both processes give the same result. If albumen be present, a haze or cloud will form close to the line where the liquids meet.

Fallacies of the nitric acid test.—1. Albumen may be present and yet escape detection, if the nitric acid is simply poured into the urine and mixed with it, as is sometimes done. For if there be too much or too little acid, acid-albumen is formed and dissolved; whereas, if the liquids form two distinct layers, as in the process already described, the acid gradually mixes with and shades off into the urine, so that, at a greater or less distance from the line where they join, it is certain to be of the proper strength to precipitate the albumen. 2. Albumen may be supposed to be present when it is not, from the formation of a cloud by the precipitation of acid urates. This cloud disappears on the application of heat; and another specimen of the urine tested by boiling gives no cloud. To avoid this fallacy, it is common to employ the test by boiling, in addition to that by nitric acid. 3. The third fallacy is not of common occurrence. It is due to the presence of fat or saponified fats in the urine. Urine containing these when simply boiled gives no cloud; but if nitric acid is added to it in the cold, or acetic acid when it is hot, the fatty acids are precipitated and form a cloud resembling albumen. This is distinguished by not being formed if along with dilute acetic acid some ether is added to the urine before boiling; the ether retaining the fatty acids in solution. If the precipitate produced by nitric acid be collected on a filter, and treated with ether, it will be dissolved, while an albuminous precipitate will not. Copaliba, which can be recognised by its odour, sometimes causes an opalescence in the urine, which is increased by nitric acid, but is removed by heat.

Additional tests for albumen.—When urine contains *mucus*, which would render the presence of an albuminous cloud obscure, a solution of ferrocyanide of potassium followed by acetic acid should be added: this will produce a cloud if albumen be present, whilst it rather clears up a turbidity due to mucus. It has a similar action on nucleins. The terms nucleins and nucleo-albumens have been given of late to the substances formerly known as mucins. In the opinion of the writer this is unadvisable because it tends to direct attention away from the fact that these substances bear the same relation to the mucous membranes of the urinary passages that mucous excretion does to the mucous membrane of the respiratory passages. A solution of pyrophosphate of sodium also precipitates albumen. A strong solution of trichloroacetic acid also gives a precipitate with albumen. If a few drops of albuminous urine be poured into a test-tube containing one or two drachms of a saturated solution of picric acid, a precipitate is formed which does not dissolve on boiling, and thus differs from that due to albumoses. This test can therefore be used like the succeeding one for albumoses as well as albumens. Alkaloids in the urine also give a precipitate with picric acid which clears up with heat and reappears on cooling, so that

in the case of patients taking quinine, caffeine, &c., this source of fallacy requires to be kept in mind. A concentrated solution of salicyl-sulphonic acid has been used both as a test for albumen and as a method of distinguishing serum-albumen from albumoses and peptone. With all these substances this reagent added to an acid urine gives a turbidity or precipitate. If the urine is not acid it must be previously acidulated with acetic acid. Should albumoses or peptone only be present, the precipitate disappears on boiling and recurs on cooling. If albumen also be present, the precipitate simply becomes less on heating and again becomes denser on cooling. These tests are sometimes useful in determining the presence of albumen in the urine in doubtful cases.

QUANTITATIVE ESTIMATION OF ALBUMEN.—There are three methods in common use for this purpose. The first is easy but inexact. It consists in boiling the urine with dilute acetic acid in a test-tube, allowing the coagulum to subside for a definite number of hours, and then estimating the proportion it bears to the quantity of urine boiled—for example, a fourth, a third, &c. The second is the most exact, but is troublesome. It is like the first; but the urine is carefully measured before boiling, and the amount of coagulum is ascertained by collecting it on a weighed filter, washing, drying, and again weighing it. The third method is easy and tolerably exact. A tube of known length is filled with urine and placed in a polarising apparatus. From the amount of rotation which the polarised ray undergoes in passing through the urine, the amount of albumen it contains may be calculated. A fourth method was recommended by Roberts. It consists in diluting the urine with water until it gives a haze on the addition of nitric acid, which does not become visible until between one half and three quarters of a minute after the acid has been added. This dilute urine contains 0.0034 per cent., or 0.0148 grain of albumen per fluid ounce; and from the degree of dilution required the amount contained in the urine may be calculated. A fifth method is that of Esbach. It is less accurate than Roberts's, but is convenient. It consists in precipitating the albumen from urine in a graduated tube by means of picric acid, and reading off the quantity of albumen precipitated after the tube has stood for twenty-four hours. The precipitant consists of 1 part of pure picric acid and 2 parts of citric acid in 100 parts of water. The tube is filled up to a mark with the urine, and about two thirds of its bulk of the precipitant is then added; the exact amount necessary being indicated by another mark on the tube. The lower part of the tube is graduated with lines numbered $\frac{1}{2}$ up to 7, and these indicate the parts by weight of albumen in 1,000 parts of urine by measure. Thus if the upper level of the precipitate after twenty-four hours stood at 2, the proportion of albumen would be 2 in 1,000, or $\frac{1}{500}$ of a grain of dry albumen to the ounce of urine. The results given are rather too low, and the method gives the best results when the proportion of albumen is small, so that urines containing much ought to be diluted before the test is applied. It is unsuitable if quinine, antipyrin, or thallin is likely to be present in the urine.

PATHOLOGY.—Albuminuria has been divided by some authors according to the sources of the albumen. When the albumen has come from the kidneys, it has been called true albuminuria, and false when it is derived from some other part of the

urinary passages. As diagnosis has improved, this classification is less employed and the terms true albuminuria and false albuminuria are now used to denote different forms of albumen, the albuminous body, in each case, passing through the kidney. Albuminuria has been said to occur in consequence of various conditions: e.g. changes in the blood, changes in the circulation, changes in the kidney. Thus abstinence from salt, or a diet of eggs alone, is said to produce albuminuria by altering the constitution of the blood; and an alteration in this fluid is supposed to be partly the cause of the albuminuria observed in high fevers, scarlatina, diphtheria, and osteo-malacia. The albuminuria of heart-disease depends on changes in the circulation; and that of nephritis on alterations in the kidney. In order to distinguish more clearly between the different kinds of albuminuria we may divide them into—(1) *false albuminuria*, in which some other albuminous body than serum-albumen is present; (2) *true albuminuria*, in which serum-albumen, frequently accompanied by globulin, appears in the urine. In *true albuminuria* there is always some change either in the circulation through the kidney, or in the structure of the kidney itself. In *false albuminuria* the albuminous body passes out through the kidney, without there being any alteration either in its circulation or structure.

(1) *False albuminuria*.—The chief albuminous bodies occurring in false albuminuria are hæmoglobin, egg-albumen, globulin, and Bence-Jones's albumen (albumose). Hæmoglobin occurs in the urine whenever blood is present in it (see HÆMATURIA), in which case it is contained in the corpuscles; or it may occur free (see HÆMOGLOBINURIA), the blood-corpuscles, while still circulating in the vessels, having undergone solution. This may result from the inhalation of arseniuretted hydrogen, or from the introduction of bile-acids or of a large quantity of water into the veins. Hæmoglobin is also found in the urine in paroxysmal hæmoglobinuria, but the cause of the solution of blood-corpuscles in this disease is unknown. Egg-albumen is excreted by the kidneys, and appears in the urine, whenever it is injected directly into the circulation or under the skin, or when it is absorbed unchanged from the stomach or rectum. When taken into the stomach it is usually completely digested before it undergoes absorption; but when taken in such large quantities that the whole of it cannot be digested, part of it is absorbed unchanged and is excreted in the urine. Thus a diet consisting exclusively of eggs, especially when continued for several days, produces false albuminuria, and large enemata of eggs have a similar effect in animals, and probably also in man. Bence-Jones's albumen is of very rare occurrence. It is found in osteo-malacia. Like egg-albumen, it is excreted by the kidneys when it is injected into the circulation, or in large quantities into the intestine. It is almost, if not quite, identical with the hetero-albumose which Kühne finds to be one of the products of imperfect digestion. It seems probable that those cases of albuminuria which appear to depend on imperfect digestion are due to the passage into the systemic circulation of albuminous bodies, which have not undergone the proper transformation in the alimentary canal or liver. See ALBUMOSES; ALBUMOSURIA; GLOBULINURIA.

(2) *True albuminuria*.—In true albuminuria there must be some change either in the circulation

or structure of the kidney, for serum-albumen differs from the other albuminous bodies just mentioned in not being excreted by the healthy kidney. Some regard the alterations in the circulation which produce albuminuria as of two kinds:—(a) increased pressure of blood in the renal arteries; (b) increased pressure in the renal veins. Increased pressure in the arteries may depend either on general high arterial tension, or on an increased local supply of blood to the kidney, owing to dilatation of the renal arteries, such as follows division of their vaso-motor nerves. Experiments seem to show, however, that increased tension in the renal arteries does not produce albuminuria, and that the only change in circulation which will cause it is increased pressure in the renal veins. Congestion of the renal veins may be produced by ligature of the renal arteries; and when the flow of blood through the kidney is temporarily arrested by ligature of the artery, the urine secreted after the removal of the ligature is albuminous. Venous congestion of the kidney also occurs whenever the onward flow of venous blood is obstructed, either by a ligature on the renal veins; by thrombosis of the renal veins or of the inferior vena cava above them; by the pressure of a tumour or of the pregnant uterus upon them or the vena cava; by disease of the liver obstructing the vena cava; or by disease of the heart or lungs, such as tricuspid or mitral regurgitation, or chronic bronchitis and emphysema. The temporary albuminuria sometimes observed after cold bathing may also be due to venous congestion; and it is probable that albuminuria consequent upon lesions of the nervous system is due rather to the changes which these produce in the circulation than to any direct action of the nerves upon the tissues of the kidney itself. The albuminuria observed after varnishing the skin is due either to the chill produced by the cutaneous vascular dilatation caused by the varnishing, or possibly in part to the production or the retention of some substance which acts as a poison. The structural changes in the kidney which cause albuminuria are acute and chronic inflammation, amyloid degeneration, and cirrhosis. See KIDNEYS, Diseases of.

FORMS OF ALBUMINURIA. A good many cases of albuminuria are more or less intermittent. They may be intermittent in the sense that they last only for a certain part of the day and then disappear, or that they may last for several days, weeks, or even months together, and then disappear for a prolonged period. Among the intermittent albuminurias which last for a short time are those which come on after exertion. It has been noted, especially in Germany, that after soldiers have marched for some distance a number of them exhibit albumen in the urine, although in every other respect the men appear to be perfectly healthy. The same thing has been noted as consequent upon violent exertion among athletes after playing heavy games, or after long walks or severe climbs. It is noted also after violent exertion due to pathological causes, e.g. after an epileptic fit, where all the muscles have been in violent contraction. Exertion of any kind tends to raise the blood-pressure, and probably the appearance of albumen in the urine in these cases has been due to some disturbance of the circulation. Blood-pressure is also raised by exposure to heat. Heat as a rule dilates the peripheral vessels and allows the blood to flow more rapidly through, so that the blood-pressure should, accordingly, fall;

but the dilatation of the peripheral vessels seems to be more than compensated by the increased rapidity and increased force of the cardiac beats ; so that the blood-pressure rises instead of falling. After exposure to great heat, whether it be external heat, as in various trades, or to the sun in hot climates, or heat generated in the body, as in infective fevers, a certain amount of albumen is apt to appear in the urine.

How far the albuminuria in such cases is due to general venous congestion during the exertion it is difficult to say. In the case of infective fevers local changes produced in the renal capillaries by the infecting microbes probably play a great part in the production of albuminuria. In cases of renal calculus, albuminuria also follows exertion and may be accompanied by hæmaturia. It comes on after passive movement, such as jolting in a carriage or railway train, as well as after active exercise, such as walking or riding. It is generally, but not invariably, accompanied by pain in the lumbar region.

Infarct in the kidney, in cases of cardiac disease or atheroma, may give rise to temporary hæmaturia and albuminuria. In poisoning by mercury, the tubules of the kidney have been found stopped up by plugs of carbonate of lime. It would almost seem as if the mercury had displaced carbonate of lime from the bones and caused it to form plugs in the tubules. Possibly this may be the cause of a form of temporary albuminuria, which has been noticed by the writer in men who had gone through a mercurial cure for syphilis and in whose urine albumen was found when they subsequently tried to insure their lives. Such cases sometimes recover under a course of iodide of potassium. Albumen occurs very frequently in the urine of young men and is known as the albuminuria of adolescence. The pathology of this form of albuminuria is not well understood, but it frequently occurs after a seminal emission, and is probably due to the close connection between the spinal centres of the kidneys and of the genital organs. If the urine, in such cases, be examined a day or two after any genital excitement has passed off, it may be found free from albumen. The albuminuria of adolescence is probably closely associated with the albuminuria of posture, which rarely or never occurs in adult men, but only in adolescents or children. It disappears completely when the individual is kept on his back, but reappears when he is upright. In this it resembles the œdema of cardiac disease, severe æmæmia, and chlorosis, and it is probably due to laxity of the renal vessels, or to some alteration in their permeability, such as occurs in chlorosis.

Another form of intermittent albuminuria is that due to digestion. It comes on after a meal, while the products of digestion are being absorbed. It is probably due to some imperfection in the digestive process.

DIAGNOSIS OF ALBUMINURIA.—Admixture with blood, or pus, or semen, or prostatic mucus, may give rise to the presence of albumen, but if no more is present than can be accounted for by their admixture, it is probable that the kidneys themselves are healthy. The persistent presence of albuminuria with casts points to the existence of some form of renal disease, for the diagnosis of which see **KIDNEYS, Diseases of**. At the same time the writer has seen cases in which albumen, sometimes in considerable quantities, was constantly present for years, the patient being, to all appearance, in perfect health.

Albuminuria in Life Assurance.—When albuminuria is definitely ascertained to be dependent upon some organic form of kidney disease, the life is always uncertain, and if taken at all can only be taken under very heavy extra payment. One of the chief difficulties in taking such lives even if the premiums are to be paid within a limited period is their uncertainty. Persons suffering from chronic nephritis, either tubular or interstitial, may continue under favourable conditions to live for many years, but at the same time a sudden chill, or intemperance in eating or drinking, or an intercurrent attack of some infective disease may carry them off in a few days. In waxy kidney the probability is that the life will not be prolonged more than ten years. The subject of albuminuria occurring in persons apparently healthy was investigated for the United States Life Assurance Company in 1877, 1878, and 1879 for the purpose of ascertaining the subsequent health and longevity of those who had exhibited this condition when examined for life assurance. Albuminuria was found in from 10 per cent. to 12 per cent. of those who presented themselves for examination presumably in perfect health. According to the writer's experience this is a very much larger proportion than is found in this country, for out of 150 consecutive persons who presented themselves for examination only four had albuminuria, or a little under 3 per cent. The total percentage of deaths in the Mutual Life Insurance Company of New York in 1875 was also a little under 3 per cent. The health of all those who had albuminuria, although apparently sound when examined, tended to deteriorate, and their lives were shorter than the usual expectation of healthy persons. The experience of men who have extensive experience in examining candidates for posts abroad is that however healthy the young men may seem, the presence of even transient albuminuria renders them less fitted for foreign service, less energetic, and less likely to retain their health than those who are free from such an affection.

TREATMENT.—(1) In *false* albuminuria, where hæmoglobin appears in the urine, the treatment indicated is to counteract the solution of blood-corpuscles ; and for this purpose quinine is very often useful. When other kinds of albumen appear in the urine, and are probably due to imperfect digestion, the treatment is to give some artificial digestive fluid. Arsenic is also useful. Regarding those cases of osteo-malacia in which Bence-Jones's albumen occurs, we unfortunately know very little.

(2) In *true* albuminuria, depending on venous congestion, the obstacle to free circulation should be removed if possible, and the congestion lessened, both by drawing the blood from the interior to the surface of the body, and by causing contraction of the renal vessels. The blood may be drawn from the interior to the surface by means of warm baths ; but in some cases these prove injurious rather than useful, and the employment of a wet pack, which has a similar effect on the distribution of blood without exciting the heart, is to be preferred. Cupping over the loins is serviceable : it probably acts by causing reflex contraction of the renal vessels rather than by actually draining blood away from them. The tone of the renal vessels may be increased by the employment of digitalis (see **DIURETICS**) ; and this drug is useful even when no cardiac disease is present, although its good effects are still more marked when the congestion is dependent on disease of the heart. The constant drain

of albumen from the body occasions anæmia, which not only produces many unpleasant symptoms, but tends to cause fatty degeneration of various organs, from which there is no reason to believe that the kidneys are exempt. The administration of iron, therefore, is the chief medicinal remedy in structural disease of the kidneys. It is useful by diminishing or removing the symptoms of anæmia, and the tendency to fatty degeneration consequent thereon; and also by increasing the tone of the vessels, thus diminishing the loss of albumen.

T. LAUDER BRUNTON.

ALBUMOSES.—**DEFINITION.**—Albumoses are proteid bodies derived by the action of digestive ferments (pepsin chiefly) upon albumens and globulins, and in their properties may be classed as intermediate between these proteids and the final product of digestion (peptone). They are formed during natural digestion in the stomach and also in artificial digestion.

ENUMERATION.—Many varieties of albumoses have been described, collectively termed *protooses*. The individual bodies differ somewhat, according as they are formed from albumens (albumoses) or globulins (globuloses): for all practical purposes the term albumose is the best to use. The forms of albumose important in medicine are *hetero-albumose*, *proto-albumose*, and *deutero-albumose*, the last being closely allied to peptone.

CHARACTERS.—**I.** *Hetero-albumose* ('Bence-Jones's albumen') is insoluble in water, but soluble in dilute saline solutions, from which it is precipitated by heat if no free acid is present. In some cases it is precipitated at a temperature of 43° to 50° C., and re-dissolves on heating the liquid to the boiling point. Artificially prepared hetero-albumose is rendered partly insoluble by heating; but the coagulum is soluble in dilute acids and alkalis, thus distinguishing it from the coagulum formed on heating a solution of serum-albumen or serum-globulin. Hetero-albumose is also precipitated from solution by saturation with magnesium sulphate or sodium chloride.

2. *Proto-albumose* and *deutero-albumose* are soluble in water, are not precipitated by heat, and are thus sharply distinguished from hetero-albumose.

These three albumoses have one reaction in common (besides those given by all proteids), viz. that with a trace of copper sulphate and an excess of liquor potassæ they give a pink-red colour (biuret reaction), the colour given by hetero-albumose being the least marked. They all behave in a peculiar way to nitric acid: if added drop by drop to a solution of hetero- or proto-albumose, nitric acid causes a precipitate, which is soluble in excess of the acid, but is also soluble on heating, re-appearing on cooling, and so on. With deutero-albumose, however, nitric acid causes no precipitate, unless common salt be added until the liquid is nearly saturated with it: the precipitate which is then produced by the acid behaves like the nitric-acid precipitates of proto- and hetero-albumose. (For other distinctive reactions see ALBUMOSURIA.) Albumoses are distinguished from peptones by the nitric-acid reaction; as well as by their precipitation with acetic acid and potassium ferro-cyanide, and with neutral ammonium sulphate added to saturation. The characters of true peptones are that they are not precipitated by heat, by nitric acid under any condition, by acetic acid and potassium ferro-cyanide, or by saturation

with neutral ammonium sulphate. This last test is the most distinctive. In the presence of ammonium sulphate to saturation they give the biuret reaction, and are thrown down by tannin.

SIDNEY MARTIN.

ALBUMOSURIA.—**SYNON.** : Propeptonuria.

DEFINITION.—A condition in which albumoses are present in the urine. The condition where peptones are present (peptonuria) is not at present distinguishable from albumosuria. Peptonuria will therefore be considered under this heading.

TESTS FOR ALBUMOSE OR PEPTONE IN URINE.—Urine containing albumoses or peptones shows ordinarily no special characteristics indicating their presence; except, in some cases, frothing when shaken. It may be dark- or light-coloured, of high or low specific gravity, and with or without deposit. In one case Kühne found a whitish deposit, consisting partly of albumose; and in a similar case Bence-Jones found casts, ordinary albumen being absent. As a rule, however, albumose and peptone are present in solution in the urine. In searching for these, coagulable albumen must be absent, or if present removed by filtration after coagulation. The following methods are, therefore, to be used: Heat the top of the column of urine in a test-tube in the usual way, but without the addition of acid. If a cloudiness appears, it is due to the precipitation of coagulable albumen, of phosphates, or of hetero-albumose (Bence-Jones's albumen). Continue the heating to the boiling-point; the cloudiness, if due to hetero-albumose, will clear up, that due to phosphates and albumen will remain. A precipitate in urine, then, caused by a moderate heat and redissolving on boiling, is hetero-albumose. This may be verified by determining the exact temperature at which the precipitation occurs: hetero-albumose is precipitated between 43° and 50° C. (109.4° and 122° F.), serum-albumen or globulin at 73° – 75° C. (163.4° and 167° F.), and phosphates only when near the boiling-point; this last precipitate also redissolves on cooling. If the urine contains albumen or globulin, it must be boiled after the addition of a drop of acetic acid, and filtered. The filtered urine must then be tested for albumose in the way to be described. The presence of hetero-albumose is indicated by the test last mentioned; in addition, it gives a characteristic nitric-acid test. If to the cold urine nitric acid be added drop by drop, a precipitate forms which dissolves on heating, re-appears on cooling, redissolves on heating, and so on: this is perfectly characteristic of the body, since the similar precipitate of albumen or globulin does not redissolve on heating. By neither of these reactions is deutero-albumose or peptone indicated, and urines containing these two bodies (one or both of them) are those usually classed as examples of 'peptonuria.' Their presence is shown by the following tests, which must be applied after hetero-albumose has been shown to be absent, and after coagulable albumen has been removed:—(a) A drop of dilute solution of copper sulphate added to the urine, followed by an excess of liquor potassæ, causes a pinkish-red coloration (biuret reaction); or a drop of diluted Fehling's solution may be added to the urine, and then an excess of liquor potassæ if necessary. If the deutero-albumose and peptone are present in small quantity, this reaction is not obtained. (b) The urine is added drop by drop to

a saturated solution of picric acid; both deuto-albumose and peptone are precipitated, and are redissolved on heating, thus distinguishing them from mucin and from albumen.

The only accurate way of distinguishing deuto-albumose from peptone is to shake the clear urine in a test-tube with solid neutral ammonium sulphate. If the urine gives the two tests just described, and gives a precipitate with ammonium sulphate, it contains deuto-albumose. If it gives the tests and no precipitate with the salt, it contains peptone, and the case is one of true peptonuria. It is best, in applying these tests, to evaporate the urine to a small bulk.

Proto-albumose has not as yet been found in urine.

Quantitative Estimation. This is of but little clinical value. It may be done by precipitating a measured quantity of urine with a large excess (ten times its bulk) of alcohol, collecting, drying, and weighing the precipitate.

PATHOLOGY.—The presence of albumoses and peptones in the urine is a pathological, not a physiological, phenomenon. These bodies are formed during normal digestion by pepsin and hydrochloric acid in the stomach, and peptones are also formed in pancreatic digestion. But although they are thus formed in the gastro-intestinal canal, they are not found in the absorbent vessels of the stomach and intestine in any appreciable quantity; neither in the veins nor in the chyle-vessels. In the general systemic veins and lymphatics they are not present; and their occurrence in arterial blood in small quantities is extremely doubtful. Lastly, they are not present in the living tissues, nor in the fluids which bathe them. If they are found in the urine, they are therefore either derived from the gastro-intestinal canal, or are formed from some pathological condition in the tissues, or perhaps in the blood. With regard to their first source, it is quite reasonable to suppose that the metamorphosis (probably into serum-albumen) which albumoses and peptones normally undergo when passing through the intestinal wall, may not take place in certain diseases where absorption is deficient, and that they may thus pass into the circulation, and be excreted by the kidneys in the urine. This, however, is a mere conjecture to explain the occurrence of 'peptonuria' (albumosuria) in some cases of chronic dyspepsia with dilated stomach. The second source of the origin of albumoses and peptones in the body is a complicated one. If these bodies are formed in any organ, tissue, or fluid of the body, they are absorbed into the blood, and then find their way into the urine. It is known that if albumoses or peptones are injected into the blood of an animal they are excreted in the urine; that hetero- and proto-albumose pass out in the urine mainly as deuto-albumose; that deuto-albumose passes out chiefly as peptone; and that peptone is unchanged as it passes from the blood into the urine. The same facts probably hold good for the human organism.

Bence-Jones's albumen (hetero-albumose) was first found in the urine of a case of osteo-malacia; and Virchow found a similar body in the diseased bones. This, then, is a clear case of excretion of the albumose from the diseased part, the excretion of a proteid which is outside the normal proteid-metabolism of the body. A similar albumose has also been found in a case, not of osteo-malacia, but of glycosuria (Gowers). In the case of all abscesses, whether

acute or chronic, in empyema and purulent peritonitis, albumoses (or peptones) are found in the urine. Here the explanation is simple, since the pus contains albumoses, and these are simply excreted by the urine after absorption into the blood. In the case of abscesses and of osteo-malacia there are collections of cells which undergo disintegration, the proteids of the cells (consisting almost solely of coagulable albumen and globulin) becoming partly transformed into albumoses. This transformation takes place by the agency of bacteria present in the pus, but it may also possibly be due to a digestive ferment set free by the dying cell. This action of bacteria with the disintegration and gradual death of exuded cells, and the formation of albumoses, probably explains the occurrence of albumosuria and peptonuria in phthisis, in pneumonia, in epidemic cerebro-spinal meningitis, in cases of cancer and other malignant growths, in the puerperal state, in typhoid fever, and in organic liver-disease. In phosphorus-poisoning there is also great disintegration of many tissues and organs, and albumosuria (peptonuria) occurs; in scurvy also and acute infectious diseases (especially measles) it is sometimes present.

One form of peptonuria has been ascribed to an origin in the kidney-cells in cases of chronic nephritis, where some of the coagulable albumen which is being continually excreted is changed into albumose or peptone. Peptonuria may indeed alternate with albuminuria in these cases, and peptone may be present as well as albumen. Peptonuria with albuminuria is not uncommon in advanced chronic phthisis. Albumose and peptone are also found in the urine in many other conditions, in which no explanation of their presence is evident; such as many inflammatory eruptions of the skin, pemphigus, urticaria, and nervous diseases—cerebral hemiplegia or psychoses.

SYMPTOMS.—From the account given of the pathology of albumosuria and peptonuria, it will be seen that they are only to be regarded as symptomatic of other graver pathological conditions; and no symptoms can be very directly ascribed to the presence of albumoses and peptones in the body, as associated with the diseases which have been already enumerated. But there are certain physiological effects of these proteids which are important to recollect. When injected into the circulation of a dog, albumoses (and peptones to a less extent) markedly reduce the blood-pressure, and produce coma and death, while the blood remains uncoagulated for a long time after death. These effects are not necessarily associated with, or threatened by, the presence of albumosuria in man; possibly because the amount of albumose present in the blood at one time is never sufficient to produce them. When such appearances as coma and uncoagulated blood after death are observed in the diseases already enumerated, they are ascribed either to the result of the grave lesion on the body generally, or the brain in particular, or to the retained chemical products of tissue-disintegration. Albumoses and peptones form one group of these toxic products, aiding the production of coma and of *post-mortem* fluidity of the blood.

Another physiological effect of albumoses is that of producing fever when injected into the circulatory system. The fever is to some extent proportional to the dose, and is produced even when the dose is insufficient to cause coma and fall of blood-

pressure, and in animals (such as rabbits) whose circulation and cerebrum are not affected by the poison. The fever of infective disease is partly due to the absorption of these albumoses.

According to our present knowledge, it may be said that the chief clinical significance of a large amount of peptone or albumose in the urine is an extensive cell- (and proteid-) disintegration in the body or some infective process. SIDNEY MARTIN.

ALCOHOL.—SYNON. : Ethyl-Alcohol ; Vinic Alcohol ; Spirit of Wine. (C_2H_5O .)

Alcohol is the product of a process of fermentation induced by the action of a microscopic fungus, *yeast*, upon certain kinds of sugar, especially grape-sugar, but also upon milk-sugar and that derived from the different varieties of starch. In this process a peculiar metamorphosis takes place, by which alcohol and carbon dioxide are produced in considerable amount, together with very minute quantities of succinic acid, glycerine, and other bodies.

The discovery that the ferment by which the sugar is decomposed into alcohol and carbon dioxide is formed by a living cell was made simultaneously in 1836 by Th. Schwann in Germany, and by Cagniard-Latour in France.

Alcohol may also be produced synthetically from its elements—carbon, hydrogen, and oxygen.

As alcohol is very volatile, boiling at 172° F. (78° C.), it may be separated by distillation from the water with which it is at first combined. Other means must be resorted to, however, in order to separate the ultimate parts of this water, as a strong attraction exists between the two liquids.

Alcohol, diluted with about 95 per cent. of water, and subjected to the action of another microscopic fungus, is oxidised into aldehyde and further into acetic acid.

PHYSIOLOGICAL EFFECTS.—Applied to the skin, alcohol produces a sensation of coolness, due to its rapid evaporation, but, if the application be continued sufficiently long, *irritation* is excited. The latter effect ensues immediately if alcohol is brought into contact with a mucous membrane. Its strong attraction for water seems to be the chief cause of this action.

Alcohol is a powerful *antiseptic*, probably from the fact that it is capable, even when diluted, of preventing the development of septic germs, such as vibrios and bacteria, as well as of paralysing the activity of those already formed.

There is scarcely any other therapeutic agent the *internal action* of which varies so much according to the dose given. In *small quantity*, and slightly diluted with water, alcohol promotes the functional activity of the stomach, the heart, and the brain, while a like quantity, largely diluted, exerts but a limited influence upon these organs : if, however, the dose of alcohol be often repeated, it is readily assimilated, and, becoming diffused throughout the system, undergoes combustion within the tissues of the body, imparts warmth to them, and yields vital force for the performance of their various functions. Simultaneously with this consumption of alcohol the body of the consumer is often observed to gain in fat, a circumstance due to simple accumulation, the fat furnished by the food remaining unburned in the tissues, because the more combustible alcohol furnishes the warmth required, leaving no necessity for the fat to be used for that

purpose. A quantity of 100 cubic centimetres of alcohol *per diem* (about three and a half fluid ounces) —equivalent to about one litre of Rhine wine of medium strength—is sufficient to supply between one third and one quarter of the whole amount of warmth requisite for the human body during the twenty-four hours. The warmth so supplied cannot be measured by a thermometer, however, any more than can that furnished by the internal combustion of other hydrocarbons, such as the oils and sugars. The subjective impression of increased warmth usually experienced after taking a dose of any alcoholic liquid is deceptive, and is only due to an irritation of the nerves of the stomach, and to the increased circulation of blood through the cutaneous vessels, particularly those of the head.

The increase of the action of the heart after taking alcohol is well known, and has often been expressed in figures. Former experiments had also established this fact with regard to the respiration. The quantity of the inhaled and exhaled air in healthy men was increased by 7 to 9 per cent. ; in one instance, after taking champagne, by 15 per cent. The more recent experiments of Binz and Wendelstadt have furnished the following results :

1. Moderate quantities of alcohol produced in healthy persons a rise of 60 to 100 per cent. in the amount of air respired when the subjects were tired by hard bodily work.

2. A rise of a lower percentage was present when the persons were asleep under the combined influence of work and of alcohol. This proves that the vital force of the respiratory nerves and muscles may be increased by the alcohol even when the psychical centres are transiently paralysed by it.

3. An unfavourable reaction, such as the occurrence of a period of unusual diminution of respiration following the period of increased activity, has never been observed.

4. The stimulating function of alcohol on the tired respiration showed itself best when alcohol was given in the form of a thoroughly natural fine-flavoured wine.

Doses somewhat larger, but still sufficiently moderate not to cause intoxication, act, for the most part, in the same way ; but, as an additional effect, they produce a distinct decrease in the temperature of the body, lasting half an hour or more. As far as the matter has hitherto been explained, this latter effect depends upon a directly depressing influence exerted by alcohol upon the working cells of the body, and upon a temporary paralysis of the vaso-motor nerves. The latter is followed by dilatation of the superficial vessels, particularly those of the head, in consequence of which a larger surface of blood is exposed, and the loss of heat by radiation into the air is increased, the temperature of the circulating fluid being thus lowered ; while, the combustion carried on by the cells being retarded, the generation of heat from this source is diminished. The quantity of carbon dioxide eliminated is thus diminished, as is also the amount of urea excreted. After the organism has become inured to the action of alcohol, these effects upon the temperature of the body are less distinctly marked, and may be entirely absent.

The agreeable excitement at first caused by such doses of alcohol is succeeded by a reaction, characterised by lassitude and drowsiness, the latter condition usually lasting longer than the previous one of exhilaration.

The symptoms of intoxication produced by *large doses* of alcohol are sufficiently well known. When the abnormal condition of excitement in the brain induced by this stimulant has been kept up, almost without intermission, for a length of time, or when it is suddenly withdrawn after the organ has been long subjected to it, the disturbance brought about is so great and persistent as to result in a complete overthrow of the reasoning faculties, and the condition known as *delirium tremens* ensues. At the same time that this pernicious influence is being exerted upon the cells of the brain, fatty accumulation may take place in other organs, particularly in the liver, heart, and connective tissues; the blood-vessels become diseased, and, in many instances, cirrhosis of the liver, kidneys, and meninges makes its appearance, as part of the general disorder of nutrition. The shrinking of connective tissue, characteristic of this last-mentioned complication, seems to depend upon the direct irritation caused by the presence of unoxidised alcohol.

Under ordinary circumstances, and after the consumption of moderate quantities of alcohol, only slight traces of it are to be detected in the urine, in the breath, and in the perspiration; none is present in the milk. A long series of experiments made by the writer's assistants, Dr. Bodlaender and Dr. Klingemann, on healthy men and animals proved that altogether, at the most, 5 per cent. reappears. The feces do not contain any alcohol at all. These experiments have been repeated by Dr. Strassmann (Berlin), and confirmed in every essential point. As regards the breath, pure alcohol imparts no odour to the exhalations of the body; the ethers and fusel oil, on the other hand, do so by reason of their being less readily combustible. It is very probable that alcohol is completely oxidised into carbon dioxide and water during the process of assimilation; at least, no other secondary products resulting from its disintegration have as yet been detected.

THERAPEUTICAL APPLICATIONS.—There can be no doubt that a healthy organism, supplied with sufficient food, is capable of performing all its regular functions without requiring any specially combustible material for the generation of heat and the development of vital force. But the case assumes a different aspect in sickness: here, while the metamorphosis of tissue goes on with its usual activity, or with increased energy, as happens in many diseases, the stomach, refusing to accept or to digest ordinary food, fails to supply material to compensate for this waste. Under such conditions, a material which can be most readily assimilated by the system, and which, by its superior combustibility, spares the sacrifice of animal tissue, is especially called for; and such a material is found in alcohol. *Small but often repeated doses* of alcohol, largely diluted with water, are generally well tolerated by the weakest stomach; and, thus given, the absorption and oxidation of the spirit goes on without difficulty or effort on the part of the patient's system. The less patients have been previously accustomed to the use of alcohol, the more marked are the good effects derived from the drug in the event of illness.

According to the experiments of Dr. Frankland and others, the burning of 1.0 gramme of alcohol yields sufficient heat to raise the temperature of seven litres of water 1° C.; and the burning of 1.9 gramme of oil suffices for nine litres. Now,

in taking three tablespoonfuls of oil daily, we yield about the same amount of warmth to the body as is given by four tablespoonfuls of absolute alcohol—the quantity contained in a bottle of light claret or hock. The oil, however is digested and oxidised by the organs of the body with difficulty, while, for the assimilation of the alcohol, scarcely any exertion of the working cells is required. Thus, it follows that heat-producing material, sufficient to supply nearly one-third of the whole amount of warmth required by the body within twenty-four hours, is offered in a quantity of 100 grammes (about three and a half fluid ounces) of alcohol. In this sense alcohol is a *food*; for we must regard as food not only the building material, but all substances which, by their combustion in its tissues, afford warmth to the animal organism, and, by so doing, contribute towards the production of vital force, and keep up the powers of endurance. Alcohol, therefore, diluted with at least 90 per cent. of water (in any convenient form of beverage), may be given with advantage, in small but oft-repeated doses, in most of the acute and chronic diseases where the digestive organs, from any cause, refuse to tolerate a more substantial form of nourishment, at least in quantities that would answer the necessities of the case. In such cases it is certainly not sufficient to call alcohol merely a *stimulant*. If alcohol served here only in the quality of a stimulant, its effect would soon pass away, leaving the patient more exhausted than ever; for the human organism is so constituted that it cannot be driven to perform its functions by the application of measures that simply stimulate, without supplying some new force to take the place of that put forth by the organs of the body under the impulse of excitement. To take a familiar illustration, alcohol thus given stimulates no more than does the readily combustible coal which we put in small quantities upon a languid fire, to prevent its going entirely out.

Experiments in the writer's laboratory (Geppert) have contributed the following facts to this part of the question. 20 to 75 cubic centimetres of absolute alcohol, taken in water or in the form of port, brandy, or sparkling hock by healthy adults, who were accustomed to take no alcohol or only a moderate quantity, hardly altered the quantity of the consumed oxygen, and likewise left unaltered or very slightly decreased the excretion of the carbonic acid.

The principal point in this result is the unchanged standard of the consumption of oxygen. We know that the consumed alcohol is burnt in the organism, a minute quantity excepted. The alcohol was thus unable to increase oxidation, as its adversaries have still continued to assert; it had not contributed to a more rapid wasting of the organism; nor had it made the regular equal march of this wasting irregular and slow. On the contrary, it had substituted itself simply as fuel in order to sustain the normal temperature of life, the existence of which is necessary to keep the whole machine in motion. Alcohol acts here, generally speaking, as if we had given the person experimented upon oil or sugar. A part of the disposable oxygen which would serve for oxidising other substances serves for the combustion of the alcohol, and keeps them intact for the organism.

Medium doses of alcohol act powerfully upon the brain and heart and are therefore serviceable as

real stimulants in cases where it is desirable to excite the cerebral and circulatory systems to greater activity. We must not forget, however, that while exciting this increased activity, such doses do not elevate the temperature of the body; on the contrary, where the effect can be measured, it is found that they depress it a little. By continuing to exhibit such doses, we can sometimes (in erysipelas, puerperal peritonitis, and similar diseases) lower febrile heat by alcohol where even quinine proves ineffectual. The consequences of this decline of fever-heat are an immediate restoration to consciousness, if delirium or stupor has been present; and, in any case, a general improvement in the feelings of the patient. Todd and his school, before the application of the thermometer, called this *the effect of stimulus*, while in reality the improvement is, to a great extent, due to the diminution of febrile disturbance. As fever-patients can tolerate large quantities of alcohol without showing any sign of intoxication, it is allowable, and sometimes even necessary, to increase the scale of doses beyond the limits ordinarily prescribed.

Alcohol has been given during the night to hectic phthisical patients as a preventive against copious and exhausting attacks of sweating, and with a gratifying amount of success. Such patients certainly tolerate the remedy much better than has hitherto been generally supposed. It need hardly be said that, in cases of cardiac excitement, not resulting from fever, alcohol is to be used with great caution.

MODE OF ADMINISTRATION.—One of the most important, but at the same time most difficult, points for decision is the exact nature and quality of the alcoholic drink to be prescribed or allowed to a patient who may require alcohol in some form. For *general use*, a pure claret, hock, or Moselle wine is the form of alcohol most to be recommended. Cognac, champagne, old gin or whisky, and the heavier Southern wines, may also be used according to circumstances. But whatever drink may be selected, it must at least be free from fusel oil to such an extent that a healthy man, even after imbibing a considerable quantity, will not feel any other effects than those of a pure stimulus; that is to say, an agreeable exhilaration of spirits, neither accompanied by a sense of weight in the head, nor followed by that persistent overfilling of the cerebral vessels and dulness of ideas characteristic of the physiological effects of fusel oil.

The *fusel oils* (so called from their oily qualities) consist chiefly of propyl, butyl, and amyl alcohols, of which the last-named forms the largest proportion. In order to examine any specimen of alcohol with reference to its purity from these objectionable constituents, it is only necessary to rub a few drops between the palms of the hands for half a minute, by which rapid evaporation is caused, and then to smell the moist spot left on either palm. If the alcohol be pure, no odour whatever should remain, as ethyl alcohol evaporates very quickly; amyl alcohol, on the contrary, is much less volatile, and, if present in the liquid, will not have evaporated, so that its peculiar and unmistakable odour will remain to attest its presence as an impurity in the specimen examined.

This test is not applicable to the more complicated liqueurs and wines, as these all contain certain odoriferous organic principles of their own, which might disguise the smell of the fusel oil. The

inoffensive quality of any given preparation, as a wine or spirit, can only be relied upon when one knows by experience that it *is* pure; and then it should always be obtained, if possible, from the same source, so as to ensure uniform purity.

To facilitate the process of estimating the quantity of any particular beverage necessary to be administered in order to produce a given effect, a table is subjoined showing the percentage of absolute alcohol contained in average specimens of the different kinds of wine, beer, &c., in common use.

Absolute Alcohol contained in—

Kumis (a fermented liquor made from milk or whey), from 1 to 3 vol. per cent. Kefir is the same, but derived from the milk or whey of the cow, while kumis comes from the milk or whey of the mare. Both contain much carbonic acid, which renders them easily digestible.

German Beer: from 3 to 5 vol. per cent.

English Beer: from 4 to 7 vol. per cent.

Hock or claret: from 8 to 11 vol. per cent.

Champagne: from 10 to 13 vol. per cent.

Southern wines (port, sherry, madeira, &c.): from 14 to 17 vol. per cent.

Brandy and strong liqueurs: from 30 to 50 vol. per cent.

For *antipyretic purposes* one will need to give an adult daily not less than the equivalent of fifty cubic centimetres (about two fluid ounces) of absolute alcohol, in divided doses within an hour or two. Taking this as a starting-point, the dose suitable for each individual case can be estimated accordingly.

The great quantity of carbon dioxide contained in certain 'sparkling' wines acts upon the temperature of a fever patient much in the same favourable manner as the alcohol itself; and when alcohol is to be taken as a food, it would seem that the impregnation with carbon dioxide facilitates its absorption.

All that has been stated thus far with regard to the use of alcohol in sickness applies to children as well as to adults. Of course no reasonable person would accustom healthy children to the use of alcoholic beverages; but, in cases of disease, really good and pure wine or brandy can be advantageously employed, even for infants, either as a *stimulant*, an *antipyretic*, or an *article of food*, according to circumstances.

In conclusion the conviction must be expressed that a healthy and well-nourished individual has not the slightest necessity for alcohol at any time or in any form, and that the practice of drinking strong alcoholic liquors cannot be too strongly condemned.

Physicians prescribing alcohol should take every care as to the conditions under which it is administered, lest the patient, while recovering from one disease, should contract another, scarcely less disastrous—a continuous craving for stimulants. The good effects of alcohol are most prominent in patients who have not been accustomed to it while in health. See ALCOHOLISM.

For *external use* as an antiseptic and disinfectant, alcohol has been superseded by various more modern agents, of which carbolic acid, iodoform, and perchloride of mercury may be mentioned as the most important. Recently, however, its external use has been revived to a large extent, for cleaning instruments and fingers before performing surgical operations and for the application of lasting bandages (*Dauerverband*) in cases of acute infectious skin-diseases, such as erysipelas and phlegmon. The results are highly praised. At all events, alcohol is the least obnoxious of all chemical disinfectants.

C. BINZ (Bonn).

ALCOHOLIC INSANITY.—See ALCOHOLISM.

ALCOHOLISM. — **DEFINITION.** — **GENERAL FACTS AND PRINCIPLES.** — Alcoholism is a term now generally used to denote all the morbid effects of the excessive use of alcohol, not only on the brain and nervous centres, but also on the other bodily organs and functions and on the mental faculties. In this article the nervous and mental effects will be chiefly referred to, as the pathological changes in the other organs will be found described among the diseases to which each organ is subject.

The physician cannot rightly approach the subject of alcoholism except by keeping in mind that the strongest affinities and the first effects of alcohol are always manifested in relation to the nervous system; that the most marked of these effects are observed on the brain-cortex in its highest functions and most delicate co-ordinations; that its injurious results on the peripheral organs—looking on the brain-cortex as the centre of the organism—are largely due to its stimulating and irritating effects on the cortical centres, where the various organs and functions are represented and from which impulses are transmitted peripherally. Almost equally important is the fact that every function of brain and nerve becomes affected in every case of alcoholism, though in different cases mental, motor, sensory, vaso-motor, trophic, and sympathetic neuroses may differ enormously, the disturbances in one function being so great, and in the others so slight, as to make it appear as if one function only were affected. The explanation of the fact that in different individuals alcoholism varies so greatly in its effects seems to be found in (1) the hereditary and innate nervous constitution and qualities of the person affected; (2) the general habits and occupations of the patients, their ages, social surroundings, and general environment; and (3) the kind of drink taken and the way it is imbibed—e.g. whether taken with food or not, whether taken in bouts or by steady soaking. The nervous, unstable youth, whose mother was insane, may have a sharp attack of mania; the professional head-worker may have an ordinary attack of delirium tremens; the town artisan may have convulsions, and the farmer and outdoor sportsman may show no appreciable effects whatever, although each of the four has drunk the same amount of alcoholic liquor. Steady over-drinking may in one man cause tremors of the limbs, in another peripheral neuritis, in another sensory disturbances, in another spinal paralysis, in another hallucinations, in another delusions and insane impulses; or some of these symptoms may exist, and commonly indeed do exist, in combination. In one man there may be elevated temperature, in another profuse perspiration, in another weakened action of the heart and lowered vascular tone, in another arterial and capillary degeneration, in another hepatic disturbance, in another renal disease, and in another an abnormal deposit of fat—all as the results of alcoholic excess.

A partial 'tolerance' of alcohol may be established in certain individuals, and there are strong grounds for believing in Archdall Reid's theory that an alcoholic 'immunity' is established in a race or community through many successive generations having drunk hard, the weakest and the most susceptible to the influence of the drug being gradually eliminated and the fittest only surviving. There are unquestionably racial differences of a marked kind in regard to the effects of alcohol. Most savage and primitive peoples, however strong

and well-developed physically, succumb to its morbid influences very soon and very completely, many of them suffering speedy extinction whenever they take to it. Yet, on the other hand, some of the advancing and dominating modern races—notably the Scandinavians, English, and Germans—have been great consumers of alcohol, excess being frequent in them all. Whether or not it is owing to the fact that alcohol is more consumed by urban than by rural populations, it is at all events certain that alcoholism is proportionally far more common in our modern cities than in the rural districts.

Another most important general principle to be kept in mind is that alcohol brings out, complicates, and aggravates the effects of every other disease. Every physician in extensive practice knows this well, and always takes it into account in his prognosis and treatment. No fact is better recognised by the medical student of vital statistics than that a high disease-rate and a high mortality-rate invariably accompany an excess in the use of alcohol in every form in any population. The universal human craving for, and use of, some nerve-stimulant or sedative, apart from food, is also an ultimate physiological and psychological fact that has to be boldly faced by the philosophic student of the effects of alcohol. Haschisch, tea, tobacco, coca, opium, and alcohol, have a common fascination for the human brain. The same brain which craves for alcohol and tends to exceed in its use is also that which is liable to a craving for morphine, chloral hydrate, cocaine, and all such stimulant-sedatives. The effects on the nervous system of excess in the use of all these have certain common characters with those produced by alcoholism. Certain of the toxins of the infective diseases such as syphilis and diphtheria, and some of the mineral poisons, such as mercury, lead, and arsenic, have also some effects resembling those of alcohol, both on the peripheral nerves and on the nerve-centres.

Another general principle to be kept in mind is that commonly alcohol attacks the *weakest point* of the organism, and attacks it at its *weakest time*—whether the weakness be due to heredity, critical periods, over-exertion, injury, or disease. A man may have stood a moderate amount of alcohol well and had no uncontrollable craving for it till he had a severe attack of influenza; yet after that he may become a drunkard and rapidly develop alcoholism. The same thing may happen to another man after his energies and tissues have been exhausted or stimulated by stress of business, or have begun to weaken through senile changes. In the case of women it is common enough to have morbid alcoholic cravings with consequent alcoholism developed at menstruation, during pregnancy or nursing, or at the climacteric period. The same thing may occur after injury to the head through railway accidents and other traumatism. It is a consideration well worth more careful study than it has yet received, whether some of the symptoms of alcoholism may result not only from the direct toxic effects of the stimulant on the nerve-elements and other tissues, but also through secondary toxins produced or let loose by the interference with normal metabolism which the drug doubtless causes. The writer thinks it certain that the syphilitic poison may be dormant till an excess of alcohol wakes it into destructive activity. Some of the alcoholic scleroses and cirrhotic conditions, both in the nervous centres and in other organs, may perhaps be accounted for in this way.

All these considerations enable us to understand in some degree the enormous range and variety of the pathological phenomena met with in alcoholism, and to realise its contributory effects in producing a part of the human degeneracy which we see in our modern large cities and manufacturing districts.

FORMS OF ALCOHOLISM :—

1. *Mania a Potu* or *Delirium Ebriosum* is that transitory insanity or delirium which is seen in the case of persons—usually young—with unstable brains from a bad heredity. To produce this in such persons very little drink is needed. A few glasses of spirits may in such cases set up a wild unmanageable fury and leave as little sane consciousness as we see in epileptic acute mania, which in some ways it resembles. Sleeplessness, mental and muscular excitement, violence, sometimes homicidal and suicidal tendencies, often a slightly raised temperature and a wild distracted facial expression are the chief symptoms. The onset is very sudden. The liquor admittedly acts on such a brain as a violent toxin to the cortex, disturbing the output of energy, destroying for the time inhibition, and causing mental explosiveness. The drunkenness often runs into the mania, and the one therefore is sometimes confused with the other. There are none of the hallucinations, tremors, high fever, or exhaustion characteristic of the true *delirium tremens*. The excitement only lasts for a few hours or a day or two at the most, and then passes off completely, leaving a little mental confusion. The proper treatment is a large dose of chloral hydrate and bromide of potassium, or of sulphonal, with as much liquid food as possible. The patient must be carefully watched.

2. *Delirium Tremens* or *Acute Alcoholism* is the next form of alcoholism in the ascending scale of seriousness and duration. From the psychiatric point of view this is a typical 'excited melancholia' with intense depression, hallucinations of sight, fleeting delusions, often suicidal impulses, incoherence of speech, failure of attention and memory, and a tendency to mistake the identities of persons about the patient. In addition, there are motor restlessness, muscular inco-ordination and tremulousness, constant purposeless movements, complete loss of the appetite for food, often sickness, foul loaded tremulous tongue, a rapid loss of body weight, a haggard expression of face, a very weak pulse, and a temperature of from 99° F. to 104° F. The patients are often suicidal or homicidal. The writer believes that a vast number of the sensational suicides reported in the newspapers, which are preceded by the murder of wife and children, take place when men are passing into acute alcoholism. If there is a taint of insanity in the blood the mental symptoms are apt to be more accentuated, and persons of a specially neurotic constitution are more liable to the disease than persons of more stable cerebral constitution. The whole condition is one of great weakness and real brain-exhaustion; in some cases there is even danger to life. It is a pronounced cerebral toxæmia. Sometimes the ordinary motor reflexes are exaggerated; at others, especially if many previous attacks have occurred, they are diminished. The proper treatment consists in careful nursing and watching night and day by a sufficient number of skilful trained nurses or attendants; and in frequent feeding with beef-tea, milk and potash-water, and soups. Rest in bed is advisable at first, but very soon the

patient should be taken into the fresh air and allowed moderate exercise. Hypnotics and sedatives may be given judiciously and with liquid foods. Paraldehyde is a very safe drug, but needs to be given in large doses (two to five drachms), and each dose may be aided by half a drachm of bromide of potassium. Chloral hydrate with bromide of potassium and sulphonal are also useful. If the patient cannot be induced to swallow these, paraldehyde may be given by the rectum, or hyoscine injected hypodermically ($\frac{1}{160}$ grain, or at most $\frac{1}{72}$ grain); morphine is not to be recommended for general use, but it is valuable in a few special cases. The patients usually recover in from three days to three weeks. Some of them die suddenly of exhaustion, especially if there has been pre-existing renal, cardiac, or cerebral disease. If many attacks have preceded the one under treatment, hallucinations of hearing are apt to follow, complicating the visual hallucinations and persisting long after they have disappeared. Such patients may 'hear voices' for months after an attack, and this condition may develop into delusional insanity. In the case of the well-to-do, trained attendants are needed; in that of the poor, a hospital or an asylum is the only means through which supervision, safety, and proper treatment can be obtained. Mechanical restraint of the patient is to be avoided if possible.

3. *Chronic Alcoholism* is a term covering a large extent of pathological territory, and its multiform symptoms in different cases are not easy to enumerate and describe in a small space. Every mental faculty may be affected, so that the patient is technically insane; or so little diminution of mental energy or such limited affection of mind may be present that the patient may not legally be of 'unsound mind.' Every other brain-function as well as mind—voluntary motion, special and general sensation, nutrition, digestion, excretion, and the metabolism of every tissue and organ may all suffer. The cause of the disease is always a steady persistent drinking for months and years, so that the toxæmia is general and the tissue-changes widely diffused. Such changes are apt to be of a structural kind, especially after repeated attacks and steady drinking. The sufferer from Chronic Alcoholism is in fact always an 'habitual' drunkard and not one who merely indulges in bouts and sprees. The first symptoms of the condition are always mental and moral. The patients become mentally obtuse, inaccurate, lazy, changed in their sense of duty and in their affection towards relations and dependents. They do not feel 'up to the mark,' but conceal this fact or assign every cause for it but the true one, which they hide and vigorously deny. They are irritable, forgetful, and frequently suspicious to a morbid degree. They become generally untrustworthy in conduct, in business, and in friendship. The face and eye—those 'mirrors of the soul'—undergo a deterioration and lowering in expression, the one being red and sodden and the other bleary. The sleep is bad and unrefreshing, and the bottle is perpetually resorted to more and more in the vain hope of securing sound sleep, with the result that all the symptoms are increased next morning. The muscles begin to show a tremor, so that the patient cannot write well, cannot hold things steadily or perform satisfactorily delicate muscular co-ordinations. The speech is often tremulous and suggestive of early General Paralysis of the Insane. The ordinary

motor reflexes, particularly the knee-jerks, are commonly found to be dulled or even lost; but there are exceptions to this rule. There may be signs of incipient Spinal Alcoholic Paralysis, to be fully developed later on. The tongue is commonly furred, flabby, and tremulous; there is no appetite; and there may be sickness from chronic gastric catarrh. The early symptoms of *peripheral neuritis*, especially in the female cases, may then be appearing. These neuritic symptoms commonly consist of tenderness of the limbs on pressure or in walking, with wasting and weakness of the extensor muscles leading to foot-drop and wrist-drop. There may be discomfort, pains, or tingling in almost any part of the body, through the toxic influence of the spirit on the sensory centres or afferent nerves in different cases. The muscles are at first flabby to the touch: as the disease becomes fully developed there is rapid loss of flesh. There may be alcoholic convulsions in the early stage, just as in acute alcoholism. Insomnia is a marked symptom and tends to increase still further the nervous exhaustion and mental disturbances. The patient's mental state gets worse and resembles that of an insane person. The temper becomes so bad and so uncertain that 'nobody can live with' the patient; or he gets really dangerous and commits assaults on members of his family or on his nurses. Hallucinations of hearing come on, and the voices or sounds or noises believed to be heard are always disagreeable or irritating. The writer has known several cases of homicide, and many of suicide, attempted or committed at this stage; indeed it is well for this danger to be constantly kept in mind in treatment. Suspicions and suspicious delusions about those near them, especially about those who are trying to stop the supplies of liquor, are very common. The patients say they are being poisoned or starved or cheated or cruelly treated. They think men are in the house to rob and murder them, and they sometimes buy and keep in their rooms weapons with which to defend themselves against their imaginary enemies and persecutors. Some of the cases are impulsive and mentally epileptiform in character. Such patients may suddenly and without warning break windows and furniture, upset tables covered with dishes, jump into water, and assault without any provocation those near them. The power of attention and concentration of mind is markedly paralysed. The affective nature is commonly much changed. The natural and previously existing love and affection for relatives and friends changes to indifference or to actual dislike. The memory, especially for recent events, becomes very bad. The ethical condition of the patient steadily deteriorates, so that there are seen senseless lying, cruelty, degeneration of personal habits, and loss of the social instincts. In fact, the higher intellectual, emotional, and moral faculties undergo a steady degradation tending towards their suspension. Some authorities divide the mental change into two periods, the one here described being the first, and the second being one of dementia, in which the memory especially almost disappears. The motor condition of this second stage is that of paresis or paralysis; this in time extends even to the sphincters. The first stage is typically one of tremor, hallucination, irritability, and moral perversion, the second that of paresis and mental extinction.

PATHOLOGY.—The pathological changes accompanying the first stage are those of cortical conges-

tion, thickening of the pia-arachnoid, and enormous proliferation of the neuroglia-elements of the cortex. The coats of the cerebral arterioles and capillaries are thickened and fatty, and atheromatous changes occur; heaped masses of proliferated nuclei appear on the external coat which almost block up the lymphatic perivascular space. The neuroglia forms a dense 'felted' layer extending from the pia mater through one fourth of the thickness of the cortex. In many cases the pia mater adheres to the outer layer of the cortex in places, and the lining membranes of the ventricles become granular. The cells undergo all kinds and degrees of pigmentary and granular degeneration. The second stage corresponds pathologically to the general atrophy of the brain with especial wasting of the convolutions, and the deposit of masses of amyloid bodies between the pia mater and cortex as well as throughout the brain substance. The brain-cells have meanwhile largely undergone the process of atrophy. Bevan Lewis directs special attention to the fact that the motor-cells of the fifth layer exhibit fatty change, and together with the layer of spindle-cells below them undergo extensive degeneration and absorption. Fatty embolisms and minute aneurysmal dilatations are also commonly found in the small vessels of the white substance of the brain. Thus it is seen that modern methods of microscopical examination of the brain exhibit sufficient pathological changes in the membranes, neuroglia, vascular and lymphatic structures, and cells to account fully for the mental and motor defects of the Chronic Alcoholic. His brain under the microscope after death is as pathologically distinctive and as far from its average normal state as is the man himself in mind and body while alive. See *INSANITY*, Pathology of.

DIAGNOSIS.—The chief conditions that are liable to be mistaken for Chronic Alcoholism are General Paralysis of the Insane in its early stages; certain syphilitic vascular and gummatous affections of the brain-cortex; certain kinds of cerebral softening and apoplectic states, especially those attacking cortical areas; the effects of sun-stroke in certain cases; and post-epileptic states. The clinical resemblance between certain early General Paralytics and certain Chronic Alcoholics is so close that it may be necessary to wait for a time before a definite diagnosis is made. The history of continuous drinking is of course the most relevant fact in determining the disease as distinguished from the other states just mentioned. But General Paralytics and people with sunstroke and brain-softening often enough drink hard, and there may thus be produced a confusing combination of Alcoholic and other symptoms.

PROGNOSIS.—The prospects of the Chronic Alcoholic are on the whole bad; but a certain number of cases do recover and are able to resume their business and places in society. Recovery is never a speedy process. A certain number are half cured, and live for long periods, leading a sort of sub-life with damaged minds and brains. But the majority go on from bad to worse, and die in a few years from the advancing destruction of nerve-elements, or from disease of the heart, liver, or kidney.

TREATMENT.—The treatment of such cases begins by an insistence on a *total* abstinence from alcoholic drinks as the most essential condition of cure. The use of tonics, nerve-stimulants such as the mineral acids and strychnine, and liberal doses of

iodide of potassium are at first indicated. Sometimes the continuous galvanic current may be employed. Simple diet combined with a regulated physiological life spent largely in the fresh air, with work and with a good moral atmosphere, comprises the chief means of treatment. Control from without in some shape is essential in most cases. If the patient is technically insane and can be certified and sent to an asylum, this course affords in most cases the best chance of cure, at least until a proper law for the treatment of inebriates is passed by the Legislature.

Alcoholic Dementia and Degeneration.—There can be no doubt that a man may induce some of the most marked and far-reaching pathological effects of alcoholic excess without having once been 'drunk'—without ever having had acute or chronic alcoholism or mental disturbance in any active form. He may ultimately reach almost complete intellectual dissolution, utter moral and affective perversity, and total loss of memory through the steady brain-poisoning of a respectable and almost unobserved excess. He takes nips of whisky morning, noon, and night, so that his brain-cells are never free from its influence, but he never reaches intoxication. The result is that at forty he becomes inaccurate and makes mistakes in business; at forty-five he is unstable and stupid; at fifty or sixty his memory is gone, he is unfit to do any business whatever, his muscles are flabby, his brain is undergoing atrophy, and he is an 'old man,' well on in his alcoholic dotage. This is a real dementia of a special kind where the memory is specially affected, there often being a stage of irritability and confusion before it disappears. Dr. George R. Wilson points out that retention of the impression, reproduction of the image, and recognition are all affected alike in intoxication and in alcoholism. Often, too, a stage of silly, easily imposed on optimism precedes the more complete dissolution of dementia. Alcoholic dementia in some degree may and does usually follow uncured Chronic Alcoholism if the patient has a strong enough constitution not to have been killed off by that disease.

5. *Dipsomania* is a condition which is connected with and may lead to Alcoholism, but is essentially different in itself, being in reality a psychological defect or disease with no necessary bodily symptoms. See DIPSOMANIA.

6. *Alcoholic Neuritis.*—See NEURITIS, MULTIPLE.

7. *Alcoholic Spinal Paralysis.*—In addition to the paresis and paralysis from neuritis, certain cases suffer from complete or partial paralysis from chronic degeneration of the spinal cord. Such cases are incurable.

RELATION OF ALCOHOLISM TO SUICIDE.—There is a close relationship between alcoholism and suicide. A very considerable proportion of the suicides of the country take place in the early stages of, and as the result of, attacks of this disease. Those tragedies especially where a father first kills his wife and children and then himself are due to this disease. Dr. W. C. Sullivan has lately shown by exhaustive statistics and observations that alcoholism and suicide have a close and special relationship, and that alcoholic suicide and suicidal attempts are apt to be of a special type, being 'more impulsive and more directly and immediately related to organic conditions in the individual.' The great practical

consideration to the medical practitioner is that the possibility of suicide should be thought of in every case of alcoholism which he has to treat, and should be specially guarded against where it exists. Homicidal attempts, alone or combined with suicide, are more commonly met with in chronic than in acute alcoholism.

HEREDITY AND PREDISPOSITION.—No exhaustive statistics exist which show the relationship of alcoholic excess to previously existing excess in ancestors or collaterals, or the precise danger of its transmission to descendants; nor has it been ascertained precisely what period of life is most liable to be affected by it, or what are the causes which most predispose to it, other than a general nervous instability and the regular and excessive use of intoxicants. There is an almost universal consensus of opinion on the part of medical men who see most of it that there is a very close connection between alcoholic excess and a certain quality of brain which is characterised by instability or explosiveness, or 'nervousness,' or depression, or a general weakness of inhibition, or by a tendency to periodic mental disturbance. Whether any acquired peculiarity can be transmitted or not, all medical experience goes to show that in many families successive generations of men and women are drunkards; that in most of the members of such families a tendency to excessive drinking will 'break out' in sudden extraordinary ways, sometimes at very early periods of life; and that in other members of such families insanity, epilepsy, developmental defects, and the neuroses generally are encountered with undue frequency. In some of the most marked of such 'hereditary' cases the craving for alcohol is manifestly a diseased craving, being uncontrollable by any effort of will on the part of the patient. In such families, too, there are certain crises and periods of life that are dangerous in this connection to many of its members, notably adolescence, pregnancy, childbearing, menstruation, and the climacteric. Some of the members of such families exhibit signs of intoxication after taking very small quantities of alcohol which would not so affect average people. In such families, too, the lowering effects of diseases, such as fevers and influenza, are especially liable to take the form of a craving for alcohol and a special intolerance to its effects. The members of such families, also, show special tendencies not only to alcohol, but to morphine, chloral hydrate, cocaine, and other nerve stimulants or sedatives. All the cases of alcoholism, however, do not come from such a neuropathic stock. A vast mass of the alcoholism of the country simply arises from excess which was led up to by moderate drinking; excessive use finally becomes habitual, gradually inducing a craving, and causing paralysed inhibition simply through its damaging effects on the brain-structure. Nearly all primitive and savage races become veritable dipsomaniacs when they have access to strong alcoholic drinks; and this is one of the chief causes of their disappearance in the face of advancing civilisation. They had no hereditary tendency to alcoholism, or to any of the neuroses of civilisation. This fact is really the strongest argument used by Dr. Archdall Reid as to acquired alcoholic insanity occurring in the nations that have had for thousands of years access to alcoholic beverages, so that now all such old nations—the Jews, Greeks, Italians, and French—are the most sober peoples. The great drinkers among them have gradually effected their

own destruction and elimination, and through the survival of the fittest the non-alcoholic cravers now survive.

T. S. CLOUSTON.

ALEPPO EVIL.—See ORIENTAL SORE.

ALEXANDERSBAD, in Bavaria.—Chalybeate waters and hydropathic establishment. See MINERAL WATERS.

ALEXIA (*a*, priv.; *λέξις*, word).—A term sometimes used in the nomenclature of aphasia to denote an inability to recognise written and printed characters.

ALGID (*algidus*, cold).—A word implying extreme coldness of the body, used only when it arises in connection with an internal morbid state, such as cholera, or a special form of malignant remittent fever.

ALGIERS.—Warm winter climate. Mean winter temperature 61° F., liable to rapid changes. Heavy rains not infrequent. See CLIMATE, Treatment of Disease by.

ALIMENT.—Food or aliment furnishes the elements required for the growth and maintenance of the organism; and, through its action with the other life-factor, air, forms the source of the power manifested.

The *alimentary products* as supplied by nature are resolvable by analysis into a variety of definite chemical compounds. These constitute the *alimentary principles*. Some are common to both animal and vegetable food, as, for instance, albumen, casein, fats, &c.; others are peculiar to either the animal or vegetable kingdom. Starch, for example, is met with only in vegetable, and gelatine only in animal products.

With reference to the alimentary principles, it must be understood that in no case do they exist in natural products in an isolated form, and no single alimentary principle is capable of supporting life. Although, however, it is with the alimentary products as a whole that we are practically concerned, yet, regarded from a scientific point of view, a knowledge of their constituent principles is required, to enable us to assign to them their proper value as alimentary articles; and for the purpose of systematic consideration some kind of classification is needed.

CLASSIFICATION.—The following grouping of the alimentary principles, based on chemistry, furnishes a classification which involves no theoretical proposition, and is practically convenient:—

Food is primarily divisible into *inorganic* and *organic* principles.

The *inorganic* principles consist of water, and the various saline matters required by the system. They are as much needed for the support of life as the organic portion of food.

The *organic* principles are subdivisible into *nitrogenous* and *non-nitrogenous*; and the *non-nitrogenous* are again further subdivisible into *fats* and *carbohydrates*.

The *nitrogenous* principles contribute to the growth and nutrition of the various bodily tissues, and furnish the active constituents of the secretions. They also undergo resolution in the system into urea, which is excreted, and a complementary portion, which is susceptible of application to force-production. They are thus capable of administering

to all the purposes fulfilled by the organic portion of an aliment.

The *fats* are applied to the production of heat and other forms of force. They seem also to be essential to tissue-development generally, besides yielding the basis of the adipose tissue.

The *carbohydrates* (starch, sugar, gum, &c.) contribute to the formation of fat, and are also applied indirectly, if not directly, to force-production.

There are a few principles, such as alcohol, the vegetable acids, and pectin or vegetable jelly, which do not strictly fall within either of the preceding groups. Alcohol occupies a chemical position intermediate between the fats and carbohydrates (see ALCOHOL); while the others are more highly oxidised compounds than the carbohydrates.

All *alimentary products* in the form supplied by nature contain organic and inorganic principles, and the organic principles comprise more or less of the nitrogenous and non-nitrogenous divisions. The non-nitrogenous division, however, is not always represented, looked at broadly, by both fat and carbohydrate. Milk, which may be regarded, from the position it holds in nature, as furnishing a typical representative of an alimentary article, contains principles from each of the groups specified in the preceding classification. See DIET.

F. W. PAVY.

ALI WAL NORTH.—See SOUTH AFRICA.

ALKALINE TIDE.—See ACIDITY.

ALKALINITY.—The blood and the majority of the fluids derived from it possess an alkaline reaction to most organic indicators. This reaction is in all cases due, not to free alkali, but to bicarbonates and bi-basic phosphates of the alkalis, of which the sodium-salts are most abundant. As a result, the tissues which are fed by these fluids also have an alkaline reaction, but here there is a selective preference for potassium-salts, and as a result there is found in most tissues a preponderance of potassium bicarbonate and di-potassic phosphate instead of the corresponding sodium-salts. It may be here incidentally mentioned that the alkalinity of the tissues is decreased after death by the formation of organic acids, and, as a result, *post-mortem* testing often gives an acid reaction. The blood-corpuscles also contain organic substances of a basic nature which are set free on 'laking' and as a result the alkalinity of laked blood is almost double that of whipped blood.

The normal alkalinity of the blood, as estimated by titration against standard sodium hydrate after addition of excess of standard tartaric acid and using litmus as an indicator, is given as equivalent to 0.2 grm. of sodium hydrate per 100 grams of blood.

It is a common error to suppose that the alkalinity of the blood is permanently affected by the secretion of gastric juice on the one hand, or by the alkaline secretions of the mouth and intestine on the other, for the acid and alkali of these secretions never leave the body, but are either neutralised or reabsorbed. The formation of such fluids, therefore, merely causes a fluctuation in the alkalinity of the blood. This can only be permanently affected by those fluids which are removed from the body, viz., the urine and sweat. See ACIDITY.

Of these two the urine is the more important regulator of the reaction of the blood, the sweat playing only a subordinate part, except under pathological conditions.

The degree of alkalinity of the chief alkaline secretions, in terms of equivalent amounts of sodium carbonate (Na_2CO_3), are as follows: Saliva, 0.08 per cent.; pancreatic juice (temporary fistula), 0.3 to 0.5 per cent.; succus entericus, 0.4 to 0.5 per cent.; bile, 0.3 to 0.5 per cent.

The causation and variation of the reaction of the blood are discussed under ACIDITY.

BENJAMIN MOORE.

ALKALIS.—**DEFINITION.**—Inorganic substances, which turn syrup of violets green, and turmeric brown; and restore the blue colour to litmus which has been reddened by acids: they combine with acids to form salts; and their carbonates are soluble in water.

ENUMERATION.—The only substances which correspond with the above definition are—Potash, Soda, Lithia, and Ammonia. The alkaline earths—Lime, Magnesia, Baryta, and Strontia—and the alkaloïds have a similar action on vegetable blues and yellows; but the carbonates of the former group are almost insoluble in water; while the latter contain carbon, and are therefore classed with organic substances.

PROPERTIES.—Ammonia is distinguished from the other alkalis by its volatility. The non-volatile alkalis are readily recognised by their spectra, and by the colour they impart to the blowpipe-flame, potassium giving it a violet, sodium a yellow, and lithium a carmine colour. Potassium and sodium are present as constituents of the body in considerable quantities; ammonium exists to a smaller amount; and lithium probably in traces. Sodium is found chiefly in the blood, potassium in the muscles.

ACTION.—When applied to the skin, dilute alkalis and their carbonates act as rubefacients. Pure ammonia is a vesicant, and potash and soda have a caustic action. Both caustic potash and caustic soda absorb water from the tissues, and form a corrosive fluid, which destroys the parts around, as well as that to which the caustic has actually been applied. To prevent this effect they are sometimes mixed with lime, which absorbs the water. A mixture of potash and lime forms the 'Vienna paste.' When inhaled, ammonia causes irritation of the respiratory passages, and increased secretion of mucus. This irritation excites reflex contraction of the blood-vessels and consequent rise of blood-pressure. When swallowed in quantity, the caustic alkalis and their carbonates produce symptoms of irritant poisoning. In the case of ammonia these symptoms may be accompanied by those of inflammation of the air-passages, caused by the irritant vapour. The best antidote is dilute acid, such as vinegar. In small quantities and diluted, alkalis increase the secretion of gastric juice before meals. After absorption into the blood they render this fluid more alkaline; while potash appears especially to accelerate tissue-change, and is accordingly classed among the alteratives. When injected directly into the blood, potash acts specially on the muscles, which it paralyzes. Ammonia stimulates the motor centres in the brain and spinal cord, the respiratory centre in the medulla oblongata, and the accelerating nerves of

the heart. When injected into the veins it therefore causes convulsions like those of strychnine, and quickening of the respiration and pulse. Alkalis are chiefly excreted by the urine; and potash, soda, and lithia lessen its acidity or render it alkaline. Ammonia is partly excreted unchanged, but a portion passes out in the form of urea and uric acid; and it does not render the urine alkaline like the others. Potash and lithia act as diuretics; soda to a less extent; and ammonia least of all. The diuretic action does not depend on any change in the blood-pressure. Potash and ammonia are diaphoretic. Potash lessens the tenacity of mucus. *See* ALKALINITY.

USES.—Dilute solutions of potash and soda relieve itching in skin-diseases. Caustic potash or soda is used to destroy warts; to cauterise poisoned wounds; and to establish issues. Ammonia neutralises the formic acid which renders venomous the stings of bees, ants, and mosquitoes, and is therefore applied to relieve the pain which they cause. The intravenous injection of ammonia has been recommended as an antidote in snake-poisoning; but the value of the remedy is not established. Mixed with oil, so as to form a liniment, ammonia is used as a rubefacient in sore throats, bronchitis, rheumatic pains, and neuralgia. It is inhaled to relieve headache; as a restorative in syncope and shock, when it raises the blood-pressure; and to facilitate expectoration in chronic bronchitis. Alkalis administered after meals act as antacids, and relieve heartburn. When given before meals they increase the secretion of gastric juice, quicken digestion, and relieve weight at the epigastrium, pain between the shoulders, and flatulence. Bicarbonate of sodium is usually given for this purpose, but when the stomach is very irritable, liquor potassæ is preferred, as it is considered to have a sedative action on the mucous membrane. Alkalis appear to lessen the transformation of glycogen into sugar, and they are used on this account in diabetes mellitus. Liquor potassæ sometimes helps to reduce obesity. Alkalis are used in the treatment of scrofula, rheumatism, and gout; but in the last-mentioned disease lithia is considered the most valuable, while potash is preferred to soda, as the urate of lithium is most soluble, and the urate of sodium least so. The salts of certain organic acids, such as the acetates or citrates, may be employed as remote antacids to render the urine alkaline, as they undergo combustion, and are converted into carbonates in the blood. Alkalis are given to lessen the acidity of the urine in inflammation of the bladder or urethra, and potash is employed as a diuretic in dropsies. On account of its stimulating action on the heart and respiration, ammonia is administered in adynamic conditions and in chronic bronchitis.

T. LAUDER BRUNTON.

ALKALOIDS and other ACTIVE PRINCIPLES.—**DEFINITION.**—An alkaloid is a substance having a definite composition as regards the proportions of the chemical elements of which it is composed, and capable of combining, like an alkali, with acids to form salts. All alkaloids contain nitrogen, carbon, and hydrogen; and all, except conine, nicotine, sparteine, and some others, contain oxygen in addition.

Besides alkaloids there are other active principles found in plants, which have also a powerful influence on the animal economy, but do not possess the

chemical properties just stated. They are mostly neutral in reaction; they do not form salts with acids; and only a very few of them contain nitrogen.

CHEMICAL COMPOSITION AND RELATIONS.—These are briefly expressed in the above definition. Thus morphine, for example, one of the alkaloids of opium, has always the chemical composition represented by the formula $C_{17}H_{19}NO_3 \cdot H_2O$, and it may unite with acetic acid to form morphine acetate, just as potassium may unite with the same acid to produce potassium acetate. But the empirical formula $C_{17}H_{19}NO_3$ represents only the percentage composition of the substance in the simplest numbers, and does not express how the atoms of the different elements are related to each other. For, just as the ethylic alcohol, with the composition C_2H_5O , is believed by the chemist, from its behaviour towards other bodies, to contain a 'radicle,' or group of atoms, C_2H_5 , having certain chemical properties resembling those of a base, such as potassium, K; and just as this radicle, C_2H_5 , may replace one of the hydrogens of water, so as to form alcohol ($C_2H_5 + H_2O = \overset{H}{\underset{|}{C}}H_3O + H$); so chemists have good

reason for believing that alkaloids belong to the group known as *amines* or *amides*, which are really ammonia, NH_3 , in which one or more of the atoms of hydrogen are replaced by a radicle; in other words, they are ammonia bases, combining with HCl without elimination of H_2O . Most alkaloids are derivatives of pyridine.

It is obvious that two or more alkaloids may resemble each other in percentage composition and still be very different, both in their chemical *structure* and in their physiological action. Thus strychnine ($C_{21}H_{22}N_2O_2$), quinine ($C_{20}H_{21}N_3O_2$), and cinchonine ($C_{19}H_{21}N_2O$), differ only in a few atoms of carbon or of oxygen, more or less; but they have different physiological actions, showing that their chemical structure, which is not indicated in these formulæ, must also be different. The physiological action of an alkaloid may also be modified by combining it with another substance. Thus, as was pointed out by Crum-Brown and Fraser, compounds of strychnine with methyl, ethyl, and amyl, do not present the well-known physiological action of that substance, but one analogous to that of curarine.

ENUMERATION.—The alkaloids and other active principles most familiar to the physician are:—Morphine, Apomorphine, Codeine, Thebaine, Narcotine, Papaverine; Atropine, Hyoscyamine, Hyoscyne, Daturine; Nicotine; Conine; Physostigmine or Eserine; Strychnine, Brucine; Quinine, Cinchonine, Cinchonidine; Caffeine; Cocaine; Theobromine; Acetanilide; Phenazone (antipyrine); Aconitine; Veratrine; Digitalin; Curarine; Muscarine; Santonin; Emetine; Pilocarpine; Salicin; and Strophanthin. For the alkaloidal substances formed in dead bodies and in animal tissues, see PTOMA.

SOURCES.—The majority of alkaloids are formed by plants. The function which they subserve in the economy of the plant is not known. Some plants produce only one alkaloid, while in others two or more are formed. A few of the alkaloids have been produced synthetically by the chemist.

PHYSIOLOGICAL ACTION.—Alkaloids have various degrees of physiological activity when introduced into the animal body. Many are slow in their action, and a large dose is required to produce any observable effect; while others act more rapidly,

and are so potent that even a minute dose may destroy life. Compare, for example, narcotine, one of the alkaloids of opium, with nicotine, the alkaloid of tobacco. Twenty to thirty grains of the former have been taken by the human subject without producing any marked symptoms, while the twentieth part of a grain of the latter may induce symptoms so severe as to threaten life. It is also well known that alkaloids may have a different kind of action on different animals. Thus one-fourth of a grain of atropine will produce serious symptoms of a complex character in a dog, while three or even four grains may be given to a rabbit without causing any more marked effect than dilatation of the pupil. In considering the physiological actions of these substances, it may be borne in mind that some of them do not readily decompose and are excreted in whole or in part unchanged. Others split up quickly into simpler bodies and produce rapid but transient physiological effects; while others again which resist decomposition in the blood or tissues may produce no appreciable results for a time, but when they do begin to break up, the effects may be sudden and violent.

Alkaloids have frequently a double action on different parts of a great physiological system; and their action on a particular group of animals will depend on the relative degree of development of the parts of the system in that group. Thus most of the alkaloids of opium have such a double action—a convulsant action resembling that of strychnine, due to their influence on the spinal cord, and a narcotic or soporific action, due to their influence on sensory centres in the brain. Hence, in animals where the spinal system predominates, as in frogs, these alkaloids act most markedly as convulsants; while in the higher mammals their principal action is apparently on the encephalic centres, which have now become largely developed.

Passing to the consideration of the action of the individual substances, we cannot do more than give, by way of example, a brief *résumé* of our knowledge regarding a few of them.

1. **Morphine** ($C_{17}H_{19}NO_3$)—an alkaloid of Opium. In the frog this substance has a narcotic followed by a tetanic action. At first there is a state of depression of the brain and spinal cord, followed sooner or later by tetanic spasms or increased reflexes. Death occurs from exhaustion. Pigeons have been found to possess a remarkable power of withstanding the influence of this drug—an ordinary-sized bird requiring about two grains to kill it. Rabbits become partially somnolent, show a tendency to reflex spasms, and tolerate a large dose—say about one half to one grain per pound weight of the animal. In the dog the intravenous injection of even one tenth of a grain (for a small animal) causes agitation followed by sleep; the pulse and respiratory movements are slowed; the smaller arteries become dilated; the pupil is contracted; and, if the dose be large, death may be preceded by convulsions. In the higher mammals morphine acts chiefly on the sensory apparatus, both peripheral and central.

2. Other alkaloids of opium have also been investigated:—(a) **Narceine** ($C_{23}H_{29}NO_3$) has practically no action. Even in large doses it does not produce convulsions or sleep. (b) **Codeine** ($C_{18}H_{21}NO_3$) has an action mainly like that of morphine but much feebler. (c) **Thebaine** ($C_{19}H_{21}NO_3$) causes tetanic convulsions, thus

resembling strychnine. (d) **Narcotine** ($C_{17}H_{17}NO_2$) is very slightly narcotic. (e) **Papaverine** ($C_{20}H_{21}NO_4$) is also not very active. It is evident, therefore, that opium, which may contain more or less of all of these substances, must have an action on the body of a very complicated character.

Apomorphine ($C_{17}H_{17}NO_2$), a derivative of morphine, has none of the characteristic actions of that substance, but acts chiefly as a muscle- and nerve-poison, and as an emetic.

3. **Strychnine** ($C_{21}H_{22}N_2O_2$)—the alkaloid of *Strychnos nux-vomica*. In the frog very minute doses cause general convulsions, excited by peripheral irritation. These convulsions are due to the action of the poison on the spinal cord, as they persist after decapitation. In warm-blooded animals the reflex character of the convulsions is less evident; they have more of a tonic character, and chiefly affect the extensors. The poison heightens the reflex sensibility of the cord, acting especially on the anterior cornual cells. Death is usually the result of asphyxia from arrest in spasm of the respiratory mechanism, but it may result from exhaustion. **Brucine** ($C_{23}H_{26}N_2O_4$), another substance found in *nux-vomica*, has an action like that of strychnine, but more feeble.

4. **Atropine** ($C_{17}H_{23}NO_3$)—the chief alkaloid of *Atropa belladonna*. In the frog it causes tetanic reflex spasms after an interval of paralysis. Herbivorous animals, as a rule, have a tolerance of this poison, so that its effects are best studied in carnivora. Even in these the action is somewhat uncertain. Respiration may be paralysed without general convulsions; the pulse is quickened, from paralysis of the inhibitory action of the pneumogastric nerve on the heart; and the arterial pressure is increased. After very large doses the arterial pressure may be diminished, with paralysis of all parts containing involuntary muscular fibre. Secretions are greatly diminished. The pupil is dilated—apparently by a direct influence of the poison on the centres or nervous arrangements in the iris itself, as the effect may be observed even in an eye removed from the head. **Hyoscyamine** ($C_{17}H_{23}NO_3$) and **Hyosine**, the alkaloids of *Hyoscyamus niger*, and **Daturine**, the alkaloid of *Datura stramonium*, have actions allied to that of atropine, as well as the same formula.

5. **Digitalis** leaf contains at least three active principles—digitalin, digitalein, digitoxin—having the typical action of the drug on the circulation. The formulæ for these are at present unknown. A small dose of any one of these causes slowing of the heart and contraction of the arterioles with great rise of blood-pressure. If the dose be increased, the heart is arrested in diastole, and will not respond to direct excitation. These effects are brought about by an action on the muscle of the heart and small vessels, combined with a stimulant action on the vagus and possibly also on the intracardiac ganglia. It exerts a potent action on voluntary muscle, which, after small doses, becomes feebler in contractile power, while large doses may abolish contractility altogether.

6. **Physostigmine** or **Eserine** ($C_{15}H_{21}N_3O_2$)—the active substance of *Physostigma venenosum*, or Calabar bean. As has been pointed out by Professor Fraser, this alkaloid has an action antagonistic to that of atropine. Sensibility and consciousness remain until death; the voluntary muscles are paralysed, but previously they and the

involuntary muscles show tetanic contractions; respiration is at first accelerated, and afterwards slowed; the vessels become alternately dilated and contracted; secretion, especially that from the lachrymal and salivary glands, is increased; and the pupil is contracted. Physostigmine appears to paralyse the anterior cornua of the spinal cord.

7. **Curare**—a watery vegetable extract containing an alkaloid, **Curarine**, of the composition ($C_{18}H_{35}N$), obtained from certain parts of South America, and used by the natives of these regions as an arrow-poison. It is obtained from one or more plants belonging to the genus *Strychnos*. Its distinctive physiological action is abolition of the power of all voluntary movement, in consequence of its action, as was proved by Claude Bernard, upon the peripheral terminations of motor nerves—the 'terminal plates' of muscle. Respiratory movements are arrested as the result of paralysis of the muscles of respiration, but the heart may continue to beat for a considerable time. If artificial respiration be established, the circulation may be maintained for several hours, while the animal is completely under the influence of the substance. All the secretions are increased, and the mean temperature falls.

8. **Muscarine** ($C_8H_{15}NO_2$)—the alkaloid of *Agaricus muscarius*. It causes arrest of the heart's action in diastole, an effect which may be removed by the influence of atropine, thus affording an instance of physiological antagonism. In warm-blooded animals muscarine slows the heart's action; the blood-pressure falls; respiration is first embarrassed, and may be completely arrested; parts containing involuntary muscle are in a state of tetanic spasm; the pupil is contracted; and secretion is increased.

9. **Santonin** ($C_{15}H_{18}O_3$)—the neutral crystalline principle of *Artemisia maritima*. This drug may cause nausea, vomiting, hallucinations, vertigo, and a peculiar state of visual sensation—the field of vision usually appearing yellow, but sometimes violet. It is said that the stage of violet rapidly passes into that of yellow, and therefore it is probable that santonin may first excite the retinal fibres sensitive to violet (according to Thomas Young's theory of colour-perception), and afterwards paralyse them. In large doses, santonin causes loss of consciousness, tetanic convulsions, and death.

10. **Ergot**.—The active principles of Ergot have only been obtained in an impure state. The one which causes contraction of the small arteries and probably also of the uterus has been called *Sphacelinic Acid*; another which causes spasm has received the name of Ergotin (C₁₇H₄₀N₂O₆). Many others have been described. Ergot causes contraction of the smaller blood-vessels, contraction of the uterus, and slowing of the pulse; the animal may die in consequence of arrest of the action of the heart.

11. **Quinine** ($C_{20}H_{24}N_2O_5$)—one of the alkaloids of Cinchona. In small doses quinine accelerates the heart in the warm-blooded animal; in moderate doses it slows it; and in large doses it may arrest it, and cause convulsions and death. Research shows that its action is essentially upon the central nervous system. It destroys all microscopic organisms, killing vibrios, bacteria, and amæbæ. It arrests the movements of all kinds of protoplasm, including those of the colourless corpuscles of the blood. It arrests fermentative processes which depend on the presence of animal or vegetable organisms, but it

does not interfere with the action of digestive fluids.

12. **Nicotine** ($C_{10}H_{14}N_2$)—the alkaloid of Tobacco. It stimulates and then paralyzes the secretory nerves of glands, and also the nerves of involuntary muscles. It causes cold sweats, a feeble circulation, and fainting; a large dose causes death by failure of respiration, attended by severe convulsions. The blood-pressure falls at first, but it may then rise, and the pulse-rate is rapid. Nicotine first stimulates and then paralyzes the ends of the vagi in the heart.

13. **Cocaine** ($C_{17}H_{21}NO_4$)—from *Erythroxylon coca*. This is a local anæsthetic, acting on the terminations of the nerves of the skin and of mucous membranes. Its general action resembles that of caffeine, in small doses lessening fatigue, in larger doses causing wakefulness, or excitement and inability to think clearly. Its action is first on the cerebrum, then on the medulla, and lastly on the spinal cord. Very large doses may cause convulsions, of cerebral origin, or due to paralysis of the respiratory centre; the sensory columns of the cord are paralysed, while the motor are unaffected. Small doses raise the blood-pressure and quicken the pulse; large doses have the reverse effect. The secretions are generally diminished.

14. **Pilocarpine** ($C_{11}H_{16}N_2O_2$)—from *Pilocarpus pennatifolius* (Jaborandi). Pilocarpine stimulates the secretory nerves of glands, causing especially copious secretion from the salivary and sweat-glands. It also increases the secretion from the bronchial mucous membrane, from the glands of the stomach and intestines, and from the kidneys. It does not increase the secretion of bile, and its action on the mammary gland is uncertain. In large doses it may cause unsteadiness of movement, with a tendency to rotate, twitches of muscles, shivering, and dyspnoea, showing an action on nerve-centres. In these circumstances the pupils contracted, and there is indistinct vision, from spasm of the ciliary muscle. Large doses paralyse the vagus-endings in the heart, and there may also be spasmodic contractions of the muscular walls of the stomach, intestines, and bladder. During profuse sweating the temperature rises. There may be death from sudden collapse.

15. **Strophanthin** ($C_{21}H_{48}O_{12}$)—the active principle of *Strophanthus hispidus*. It resembles digitalin in its general action: it stimulates all striated muscles; in large doses, it may arrest the heart in systole; and it also acts as a diuretic.

16. **Acetanilide** (C_8H_9NO), **Phenazone** ($C_{11}H_{12}N_2O$), and **Phenacetin** ($C_{10}H_{13}NO_2$) depress the grey matter of the cord and brain, and thus diminish painful sensations. They also lessen a febrile temperature. In large doses they may cause collapse from extreme depression of the nerve-centres and an action on the hæmoglobin of the red blood corpuscles. JOHN G. MCKENDRICK.

ALKAPTONURIA (alkaptone; *οὐρον*, urine).—This very rare abnormality is characterised by the passage of urine which becomes brown and ultimately black on exposure to air, and much more rapidly on the addition of an alkali. The urine reduces Fehling's solution, and deeply stains any linen or woollen fabric with which it comes in contact. The condition, of which less than forty cases have, up to now, been recorded, is usually congenital and persists throughout life. It is apt to

occur in several members of a family. It apparently in no way detrimental to health, and is chiefly inconvenient on account of the staining of clothes, or as an obstacle to life insurance, since it is readily mistaken for glycosuria. The peculiarities of the urine depend upon the presence in it of homogentisin (hydroquinone-acetic) acid, $C_6H_5(OH)_2CH_2COOH$, but a second aromatic acid, uroleucic, $C_9H_{10}O_5$, has also been occasionally present. The essential fault appears to be a perversion of tyrosin-metabolism, for the administration of tyrosin by the mouth, although it does not cause alkaptonuria in normal individuals, greatly increases the output of homogentisinic acid in these cases. See LIFE ASSURANCE.

A. E. GARROD.

ALLANTIASIS (ἀλλὰς, forced meat).—Sausage-poisoning. See POISONOUS FOOD.

ALLEGHANY SPRINGS, in Montgomery county, Virginia, U.S.A.—Calcic or earthy waters. See MINERAL WATERS.

ALLEVAUD, in France (Isère).—Sulphur waters. See MINERAL WATERS.

ALLOCHIRIA (ἄλλος, another; and *χείρ*, the hand).—A peculiar disturbance of cutaneous sensibility in which a tactile sensation is referred to another part of the body than the seat of actual impression; or to a corresponding part of the opposite side of the body. See SENSATION, Disorders of.

ALOPECIA.—See BALDNESS.

ALOPECIA AREATA (ἀλώπηξ, a fox).—SYNON.: *Area*; *Alopecia circumscripta*; *Porrigo decalvans* Fr. *Alopécie*; Ger. *Fuchsräude*.

DEFINITION.—An atrophic disease of the hair, distinguished by the rapid development of more or less circular bald patches; in rare cases the baldness is general.

SYMPTOMS.—Alopecia areata is most common on the scalp, and is generally limited to that region, the occiput being the part most frequently affected. In some instances the beard and eyebrows suffer, and in very rare cases the whole of the hair is lost. Alopecia areata is usually confined either to a single round perfectly bald spot, or to several spots irregularly scattered about the scalp; when, however, the disease attacks the occiput, it is often roughly symmetrical. The same remark applies when the eyebrows and eyelashes are affected, and in all severe cases, which have a tendency to become symmetrical. A characteristic feature of the disease is the rapidity with which the hair falls off over a limited area, leaving very few, if any, stumps on the bald patch, which is bounded by hair of apparently natural growth. When, however, the hair round the area comes out very easily, it may safely be predicted that the spot will increase in size. The part affected is smooth and sometimes pinker than the surrounding skin, but much more commonly it is of the same colour, or paler. There is sometimes a slight atrophy of the skin, as well as of the hair, so that the area has a shallow, cupped character; this is comparatively rare, and therefore cannot be due simply to the loss of hair.

ÆTIOLOGY AND PATHOLOGY.—The causes of alopecia areata are obscure. The immediate origin of the disease is probably some nerve-disturbance, leading to atrophy of the roots of the hair and rarely also of the skin. This view is confirmed by the fact

that there is occasionally, though rarely, a temporary loss of sensibility over the area, and even when this is not the case, the skin is less sensitive to irritants than is normal. Sometimes the loss of hair is preceded by neuralgic pains, or tenderness on pressure, but more commonly there are no subjective sensations. The disease is rather more common in childhood and youth than in middle life, while it is seldom seen in old age; the writer has, however, met with one case in a man over seventy years of age who completely recovered. Alopecia is probably equally common in males and females, though on this point there is some difference of opinion. The old idea that the disease was due to a parasitic fungus is still common; but many of the clinical features of the disease, especially its great liability to recur after complete absence for perhaps years, is not at all in accordance with a parasitic origin. There is certainly sometimes a family tendency to alopecia. In extreme cases of alopecia areata, in which all the hair is lost, the nervous origin of the disease is generally admitted. Sabourard has found no less than fifteen species of micro-organisms in cases of alopecia areata, all of which he regards as casual denizens having no ætiological relation to the disease.

Radcliffe Crocker remarks, 'No one hypothesis will explain all cases, and the clinical facts can only be accounted for by explaining some cases on a trophoneurotic supposition and others on a parasitic.'

With regard to its morbid anatomy, it may be said that it is often difficult to find stumps, but when present they are very characteristic. They are also sometimes valuable for the purpose of differential diagnosis, since they are unlike the stumps of ringworm, being straight and thicker at the free end than at the point of insertion, which is contracted into a sort of neck, ending in a small rounded atrophied root looking like a note of admiration sign, !. A very slight amount of traction removes them entire; in this respect they differ much from ordinary ringworm-stumps.

DIAGNOSIS.—Ringworm is the only disease which is liable to be mistaken for alopecia areata. The difficulty arises thus: in common ringworm, either from treatment or from some unknown cause, the hair sometimes falls rapidly off, instead of breaking as it usually does. When the hair comes out in this way, with root attached, a smooth bald spot is left, exactly like alopecia areata; under these circumstances, unless a stump can be obtained for examination, the diagnosis is very difficult. The presence of other cases of ringworm in the same house, and the history of the patient, may be a guide to diagnosis.

Dr. Crocker mentions a very interesting and instructive series of cases of 'eight children in one family, who, while at the seaside, had each a few small perfectly bald spots on their heads; they were quite bare from the first, and never larger than half an inch in diameter.' After a time the governess contracted the disease, which was believed by her doctor to be alopecia areata, and not contagious; she, however, slept with her sister, who afterwards showed similar spots. Dr. Crocker says: 'The hairs round were loose; there were no short hairs, but one pulled out of the border showed distinct fungus-elements, indistinguishable from tinea tonsurans. In no case were there more than three spots, and they were all small. In one child there was a history of a red ring on the side of the cheek.' The

writer has himself met with three similar examples. The first was in a school of about forty-five girls, where there were upwards of twenty girls affected with small bald spots on the head, mostly free from stumps; there were, however, in a few cases a sufficient number of stumps full of ringworm-fungus to make the diagnosis certain. The medical officer to the school correctly diagnosed ringworm, although the appearance of most of the children would have deceived any one had the cases occurred singly. The second instance was in a boys' school, in which sixteen or seventeen cases of bald spots on the head occurred. The writer saw in the first place only two boys, who were sent to him before it was known that any others in the school were affected. Both boys had small bald spots like area, but on one of the spots were ringworm-stumps. The writer recommended that all the boys in the school should have their heads examined; this was done, and then many others were found to have bald spots. He subsequently saw one or two other boys from the same school with bald spots, which he had no doubt, under the circumstances, were ringworm. In this instance the disease had been called alopecia areata, and pronounced not contagious. The third case was in a family of children.

In all these cases there was a very close resemblance to 'area,' so that it would be almost impossible to diagnose an isolated case as tinea tonsurans, unless some of the affected hairs remained. In ringworm, when the hairs have all been shed, the risk of further contagion is much diminished; on the other hand, the disease spreads very rapidly at first, for every hair that is shed is a centre of infection. Syphilitic alopecia may resemble alopecia areata very closely. The hair on the rest of the scalp is, however, generally scanty, and some evidence of syphilis usually coexists elsewhere.

TREATMENT.—The prognosis in most cases of alopecia areata is favourable, depending on the duration of the trouble, but the disease is very tedious. In the neurotic form the new hair is frequently white, and remains so for a long time. The treatment is partly local and partly general; the local treatment consists in the application of stimulating remedies to the bald patch, of which the best is cantharides. The acetum cantharidis, diluted with one or two parts of spirit, and painted on daily with a camel's-hair brush, answers very well. The lotion used should not be strong enough to blister, because its daily use cannot then be continued. Capsicum, oleum pini, perchloride of mercury, and faradism find favour with some dermatologists. A stimulating parasiticide, as chrysarobin ointment (5j to ʒj lanolin), is most useful, but its employment has drawbacks. The general treatment should consist of suitable tonics, and wine or beer, with plenty of rest and bracing air. Continued physical fatigue appears in many cases to favour the development of alopecia areata.

ROBERT LIVEING.

ALPHOS and ALPHOIDES (ἀλφός, white).—Terms signifying white and white-looking, associated with the whiteness of the scales of common psoriasis: hence *lepra alphos* and *lepra alphoides*. See PSORIASIS.

ALPHOSIS (ἀλφός, white).—Whiteness, or the process of turning white. See ACHROMA.

ALTERATIVES.—**DEFINITION.**—Medicines which gradually restore the nutrition of the body to

a healthy condition, without producing evacuations, or immediately exerting any very evident action upon the nervous system.

ENUMERATION.—The principal alteratives are—Nitric and Nitro-hydrochloric acids; Chlorine and Chlorides; Iodine and Iodides; Sulphur and Sulphides; Potassium and its salts; Mercury and its salts; Phosphorus and Hypophosphites; Antimony; Arsenic; Taraxacum; Sarsaparilla; Hemidesmus; Guaiacum; Colchicum; and Mezereon.

ACTION.—Healthy nutrition depends on the digestion of the food, its assimilation by the tissues, the decomposition of the tissues during the exercise of their functions, and the removal of their waste products being performed in a proper manner—in due proportion one to another. If the food is not properly digested, as in dyspepsia; or is not properly assimilated, as in diabetes; or if the tissues break up too rapidly, as in fever; or if the waste products are not properly removed, as in some cases of kidney-disease, nutrition suffers. Digestion and excretion may be improved by tonics, purgatives, and diuretics; but alteratives seem to exert their action upon assimilation and tissue-change. The digestion of food is effected by means of ferments, such as those of the salivary glands, stomach, pancreas, &c. Some also of the changes, such as the conversion of glycogen into sugar, which the food undergoes after absorption in the liver, and even certain so-called vital actions—such as the coagulation of the blood—are produced by a similar agency. It is not improbable that the histolytic changes in the tissues are also effected by ferments. They do not depend upon oxidation, for although during health the products of tissue-decomposition are oxidised as fast as they are formed, yet under certain circumstances the tissues are split up so rapidly that the products which they yield are only partially oxidised. This is seen in poisoning by antimony, arsenic, and still more markedly by phosphorus, where such tissues as the muscles become decomposed, yielding nitrogenous substances, such as leucin, tyrosin, or urea, and fat. The former are excreted in the urine; while the last, instead of undergoing combustion, accumulates in the place formerly occupied by the muscular tissue, which is accordingly said to be in a state of fatty degeneration. It is possible then, although by no means certain, that alteratives influence nutrition, either by modifying the activity of ferments, or by altering the susceptibility of the tissues to their action.

Mercurials in purgative doses, taraxacum, nitric and nitro-hydrochloric acids, probably act by modifying the digestion of the food in the upper part of the small intestine, or by affecting the changes which it undergoes in the liver after absorption. Potash has probably an action on the muscles. Antimony, arsenic, and phosphorus especially affect the nervous and cutaneous systems. Mercury has a peculiar power of breaking up newly formed fibrinous deposits, and particularly syphilitic growths. Iodine, iodides, and probably chlorides, act upon the lymphatic system and promote absorption.

USES.—Purgative doses of mercurials, taraxacum, nitric and nitro-hydrochloric acids are useful in cases of frontal headache, general malaise, and depression of spirits, associated with symptoms of so-called biliousness, or with the appearance of urates or of oxalates in the urine. Potash and colchicum are employed in the treatment of gout. Phosphorus

and arsenic are used in cases of nervous debility, as well as in nervous diseases, such as neuralgia and chorea, in which antimony is also serviceable. Arsenic is also given in diseases of the skin; and antimony in inflammation of the mucous membrane of the bronchi. Mercury in alterative (that is, small) doses, which are absorbed into the circulation without purging, is used to break up newly deposited fibrinous masses, as in iritis, pericarditis, &c., and to counteract the effect of syphilitic virus upon the soft tissues in the secondary stage of this disease. Iodine and iodides act on the lymphatic system, and are useful in removing glandular swellings. By stimulating the absorbent system they may also assist in the removal of the fibrinous deposits and syphilitic growths disintegrated by mercury. The iodides are sometimes given in the secondary, but are still more valuable in the tertiary, stage of syphilis.

T. LAUDER BRUNTON.

ALVENEU, in Switzerland.—Sulphur waters, and chalybeate water, with iodine and common salt. See MINERAL WATERS.

ALVEOLAR.—A word used in pathology as descriptive of any morbid growth which consists of small cavities or spaces (*alveoli*), usually occupied by cells, and bounded by walls formed of cells or fibres. See CANCER.

AMAUROSIS (ἀμαύρωσις, a darkening or dulling of the sight).—The term ‘amaurosis’ has now almost fallen into disuse. It was originally employed, before the use of the ophthalmoscope became general, to denote partial or total blindness unaccompanied by any obvious change in the eyeball. The large majority of cases formerly called amaurotic are now more accurately classified. See EYE and its APPENDAGES, Diseases of; vii. Diseases of the OPTIC NERVE and RETINA.

AMBLYOPIA (ἀμβλωπία, dim-sightedness).—Obscurity of vision.

AMBULANCE (*ambulo*, I move about).—**SYNON.** : Fr. *Ambulance*; Ger. *Feldlazareth*.

DEFINITION.—A term adopted from the French word, which signifies the movable hospital of an army.

The use of an ambulance is to convey from place to place those who are sick, wounded, or otherwise incapable of moving.

DESCRIPTION.—An ambulance consists of a *vehicle* or caravan, upon or within which one or more *stretchers* can be laid or fixed.

Stretchers.—It is important that stretchers should be of uniform pattern, so as to admit of easy transference from one carriage to another. Each stretcher consists of two poles kept apart by metal traverses, supporting a canvas stretched between. The length of a stretcher (7ft. 9in.) is often inconvenient when conveying a patient through passages or up staircases. To obviate this difficulty, the poles of stretchers used for civil ambulances should be made of metal tube capable of being telescoped.

The *vehicles* or carriage-frames are of two kinds : (1) those for *hand-ambulances*; and (2) those for *horse-ambulances*.

1. *Hand-ambulances*, or litters, of which there are several varieties, consist of a skeleton carriage, firmly supported on easy springs, and provided with two, three, or four wheels.

Upon a framework thus formed, the stretcher fits, and is immovably fixed by straps or bolts. The carriage is made with a view to convenience of size and lightness of structure, consistently with strength. Facility in turning should be secured. A complete covering or hood protects the patient from exposure. In all ambulances special care should be taken, by springs and other contrivances, to prevent the suffering caused by jolting.

2. *Horse-ambulances* are used for (a) civil, and (b) military purposes.

(a) The first of these, as used in towns, are drawn by one horse. They usually consist of coaches which are unlined, so as to allow of washing and complete disinfection. One or two doors are placed behind, through which a stretcher or bed may slide on to a shelf, occupying about one lateral half of the interior of the coach. A seat for an attendant completes the fittings.

Ambulances for civil purposes should be easily obtainable at hospitals, police-stations, and other recognised centres.

(b) *Military ambulances*, more commonly termed ambulance-wagons, are of many different patterns. They consist of a wood-framed body provided with a cover, curtains, and hood of canvas extended on a skeleton-framed roof. They carry likewise a reservoir for water. These ambulances are drawn by two or more horses. In the English army they are generally arranged to carry two patients on stretchers, and five others less seriously injured, two in front and three behind. In the German army they accommodate four stretchers—two on the floor of the wagon, and two on a plane above—besides five other persons slightly wounded.

In war the ambulance-wagons are never sufficiently numerous to meet the demands arising after a heavy battle; in such emergencies, farm wagons and carts of the country are therefore requisitioned and the wounded are laid upon a bed of straw.

Ambulance Field Hospital.—Captain Tomkins, of the Victoria Rifles, and the writer have designed an ambulance-wagon which supports on its exterior a folding double tent 35 x 22 feet. Twenty folded stretchers, each convertible into a bedstead, one foot high by two feet two inches wide, line the side of the wagon. There are also provided the requisites for twenty beds, surgical instruments and appliances, a furnace for cooking, and for warming the tent in winter, together with rations for three days. This arrangement obviates the necessity for assistance from the main army.

A. T. NORTON.

AMBULANT (*ambulo*, I move about).—Moving, unfixed, shifting. Applied to diseases that shift from one part to another, such as erysipelas; in connection with blisters applied in succession to different parts of the body, also called 'flying blisters'; and to moving or 'field' hospitals, as distinguished from fixed or 'base' hospitals.

AMBULATORY (*ambulo*, I move about).—A term used in connection with mild or latent cases of acute diseases, in which the patient continues to walk about, or work, up to a late period of the attack, as in typhoid fever—*typhus ambulans s. ambulatorius*. See TYPHOID FEVER.

AMÉLIE-LES-BAINS, in France (Pyrénées). Thermal sulphur waters and climatic health-

resort. See MINERAL WATERS, and CLIMATE, Treatment of Disease by.

AMENORRHOEA (ἀ, priv.; μήν, a month; and ῥέω, I flow).—Absence of the menstrual flow during any portion of the period of life when it ought to be present. See MENSTRUATION, Disorders of.

AMNESIA (ἀμνησία, forgetfulness).—SYNON.: Fr. *Amnésie*; Ger. *Gedächtnisschwäche*.—A general term signifying loss or defect of memory; commonly, however, used in reference to one particular class of defect of memory, viz. that for words (*amnesia verbalis*). Sometimes there is a mere forgetfulness of proper names; sometimes a substitution of wrong words; at other times the pronunciation of a meaningless jargon, not at all representing words. The production of these defects is due to disease in different parts of the brain, owing to the memories of words being threefold—auditory, visual, and kinæsthetic. See APHASIA.

AMCEBA COLI.—A species of amoeba, found in connection with ulceration of the colon and with tropical abscess of the liver. See DYSENTERY; and LIVER, Abscess of.

AMPHORIC.—A peculiar hollow, metallic quality of sound, elicited occasionally by percussion, but more commonly heard in auscultation. Amphoric breath-sound resembles that produced by blowing into an empty glass or metallic vessel with a narrow neck (*amphora*). See PHYSICAL EXAMINATION.

AMYLOID DISEASE (ἄμυλον, starch).—SYNON.: *Lardaceous Disease*; *Albuminoid Disease*; *Waxy Degeneration*. Fr. *Dégénération amyloïde*; Ger. *Amyloide Entartung*; *Speckartige Degeneration*.

DEFINITION.—A condition characterised by a peculiar degeneration of the cells of certain organs, whereby a substance known as amyloid or lardacein is produced.

ÆTIOLOGY.—The most frequent cause of this condition is protracted suppuration from a deeply seated focus, such as diseased bone, or from a cavity not admitting of effectual drainage. Hip-disease, empyemata, and chronic pulmonary tuberculosis are thus frequent antecedents of amyloid degeneration. It also occurs in tertiary syphilis and in the hereditary form of this disease, apart from any suppuration, and occasionally in cases of dysentery, malaria, leuchæmia, and malignant tumours. Very rarely it appears to be a primary disease, especially in children. Amyloid degeneration is met with as a merely local change in the neighbourhood of syphilitic gummata.

The formation of amyloid substance is probably due in all cases to the action of toxins produced by micro-organisms. This is suggested by its association with ill-drained cavities rather than with profuse but unimpeded suppuration, and is confirmed by the experimental induction of the change by inoculating animals with cultures of the *Staphylococcus pyogenes aureus*. In cases of suppuration experimentally produced the appearance of the degeneration may be exceedingly rapid. Amyloid substance is very resistant to putrefaction and to the action of digestive ferments. Chemically it is stated to be a compound of an organic acid (chondroitin-sulphuric acid) with some form of albumen (Krawkow). It is probable that this latter component may vary

somewhat in different specimens, and that thus slight differences of behaviour to reagents may be accounted for.

ANATOMICAL CHARACTERS.—The organs principally affected by amyloid degeneration are the liver, spleen, kidneys, intestine, and lymphatic glands; more rarely the other digestive organs, the generative organs, muscles, serous membranes, and cerebro-spinal meninges may be involved. The change is primarily confined to the connective tissue; it is doubtful whether epithelial cells ever undergo this transformation. Affected organs are enlarged, pale and bloodless, firm in consistency, with smooth surfaces and somewhat thickened rounded edges. On section they present in advanced cases a peculiar glistening appearance, as if dotted with small masses of transparent material. Early stages of the change can only be recognised by the *characteristic colour-reactions*; if the cut surface of an amyloid organ be washed and treated with a weak solution of iodine, the degenerated parts assume a mahogany-brown hue, while the healthy tissue is stained a pale yellow. For microscopic purposes sections are stained with methyl- or gentian-violet, excess of the dye being washed out with dilute (10 per cent.) acetic acid: thus treated the amyloid masses are stained a magenta colour, the normal tissue appearing blue. If sections be stained with iodine and mounted in glycerine, the addition of a drop of strong sulphuric acid placed at the edge of the cover-glass will produce a blue or greenish-blue coloration in the amyloid substance.

In the *liver* the degeneration affects principally the capillaries of the hepatic artery, and therefore appears first in the middle zones of the lobules. The liver-cells are pressed upon by the new material, and become fatty and finally disappear. In very rare cases the capillaries of the portal vein may be affected, and ascites may ensue. In the *spleen* the disease may affect either the Malpighian bodies alone, which then resemble grains of boiled sago set in the substance of the organ (sago-spleen), or the connective tissue forming the trabecule (diffuse form): rarely both forms occur together (mixed form). In the *kidneys* the glomeruli generally suffer early, and along with them the middle coat of the smaller arteries is invaded; later the larger arteries, the intertubular tissue, and the tunicae propriae of the tubules also suffer. A certain amount of tubal nephritis generally accompanies the degeneration, being due either to pressure and irritation caused by the masses of amyloid material or to the action of the same toxins which cause the degeneration. Casts are formed in the tubules, and it is said that they may give the characteristic amyloid reaction with iodine; more frequently they are either hyaline cylinders or covered with fatty or granular epithelium. In the *intestine* the vessels running in the centres of the villi are chiefly affected, so that treatment with iodine causes the interior of the gut to appear stippled with closely set brown dots.

EFFECTS.—The effects of amyloid degeneration of the liver and spleen cannot be distinguished from those of the primary condition from which the patient is suffering, though there can be little doubt that the general constitutional disturbance is aggravated by the degeneration of these organs. If the intestine is affected, severe and uncontrollable diarrhoea results, probably due to increased transudation of serous fluid through the affected vessels. Ulcers may also be formed in the

intestine, apparently by rubbing off of villi rendered brittle by the change. In the kidney increased transudation leads to polyuria and to the appearances of albumen in this secretion. It is here often difficult to distinguish the respective parts played by amyloid change and by the accompanying tubal nephritis.

CLINICAL CHARACTERS.—Patients suffering from secondary amyloid disease are weak, anæmic and emaciated. The abdomen is often enlarged owing to the increase in size of the liver and spleen, which may together occupy a large portion of this cavity. Ascites is exceptional, and there is not often pain in the affected organs: when pain occurs it is probably due to the weight thrown upon their attachments or to slight accompanying peritonitis. The enlarged organs are smooth and firm on palpation and not generally tender. In primary cases and in amyloid disease affecting the kidneys alone (frequently syphilitic) there is less emaciation and debility, but anæmia is constant. The urine is pale and abundant, and contains an amount of albumen which increases as the disease advances. The occurrence of subacute attacks of tubal nephritis may cause the urine to be temporarily scanty, high-coloured, and even bloody: in such cases uræmia may result. The intestine is generally affected late in the disease.

DIAGNOSIS.—Enlargement of liver and spleen occurring in leucæmia, in cirrhosis of the liver, and in children the subjects of rickets or congenital syphilis, have to be distinguished from amyloid disease. In most cases of this degeneration the existence of suppuration in some part gives a clue to the nature of the condition. If any question of leucæmia arise, it should be borne in mind that suppuration may be associated with some increase in the number of leucocytes present in the blood, but that in such a case the additional white corpuscles will be of the multinucleated variety. The existence of amyloid disease of the kidney alone must practically be inferred by a process of exclusion. A history of syphilis or signs of this disease may be of assistance as pointing to a cause of amyloid change. The form of renal disease for which the amyloid kidney is most likely to be mistaken is the contracting large white kidney: the presence of œdema is in favour of the inflammatory condition, but the supervention of tubal nephritis upon the degenerative process may make the existence of the latter impossible to recognise.

PROGNOSIS.—If the cause of the amyloid degeneration can be removed, it is rendered fairly certain by the results of experiment that the local change may disappear. Unfortunately the removal of the cause may often be impossible, and in such cases it is unlikely that the patient will survive the appearance of amyloid disease for more than two years. Many cases terminate fatally at a much earlier period. The occurrence of diarrhoea, showing implication of the intestine, indicates that the fatal issue will not be long delayed. In syphilitic cases the outlook is less unfavourable, and recovery may take place under appropriate treatment.

TREATMENT.—The treatment of amyloid disease must be directed mainly to the primary cause. Towards prevention of its occurrence much has been done by antiseptic methods of surgery, both in lessening the number of cases which suppurate and in checking suppuration when it has occurred. Even after amyloid change has set in every attempt

must still be made to treat the pyogenic focus by the most radical methods available. The patient must in all cases be placed in the most favourable conditions. Carefully regulated nutritious diet is indispensable: sea-air and sunshine are valuable auxiliaries. Cod-liver oil, malt-preparations, hypophosphites, and iron are useful remedial agents. In syphilitic cases iodide of potassium must be given in full doses. Care must be exercised in prescribing any preparations of mercury to patients who are the subjects of renal lesions. For the diarrhoea due to the intestinal affection, opium, bismuth, and other astringents may be tried, but the condition is seldom amenable to any form of treatment.

WM. CECIL BOSANQUET.

AMYOTROPHIC (ἀ, priv.; μῦς, a muscle; and τροφή, nutrition).—Associated with muscular wasting. See SPINAL CORD, Diseases of.

ANÆMIA (ἀ, priv.; and αἷμα, blood).—SYNON.: *Spanæmia*; *Hydræmia*; *Oligæmia*; Aglobulism. Fr. *Anémie*; Ger. *Anämie*; *Blutarmuth*.

DEFINITION.—Deficiency, absolute or relative, of the most important constituents of the blood, particularly albuminous substances and red corpuscles.

This definition is purely pathological, and the condition thus expressed presents many varieties, anæmia in the widest sense of the term including oligæmia, oligocythæmia, hydræmia, and spanæmia, as well as chlorosis (see CHLOROSIS; and BLOOD, Morbid Conditions of). From the clinical point of view, anæmia is a condition of system in which impoverishment of the blood, whether from want or from waste, is associated with symptoms of imperfect discharge of the vital functions.

ÆTIOLOGY.—The causes of anæmia are generally multiple and complex. First, the supply of blood to the body may be insufficient, and that from a variety of causes, of which the chief are:—derangements of alimentation, including insufficient food, constipation, and morbid states of the lymphatic and blood-glands; such defective hygienic conditions affecting the formation and nutrition of the blood as want of light, air, and muscular exercise; prolonged exposure to the influence of certain poisons, as lead, mercury, and malaria; and, lastly, interference with the free circulation of the blood by cardiac or vascular disease, such as valvular disease or dilatation of the heart and aneurysm of the aorta. Secondly, the consumption of blood is increased by hæmorrhage; by profuse discharges, such as suppuration and albuminuria; by hæmolysis; by rapid growth and development; by frequent pregnancy and superlactation; by excessive muscular exertion; and by the presence of pyrexia, or of new growths, which rob the system of nutritive material. In a third group of cases of anæmia both the supply and the consumption are at fault. Thus derangement of the organs and of the whole process of sanguification is frequently associated with profuse discharges from various parts; and in acute febrile diseases, malignant diseases, and the 'chronic constitutional diseases,' such as syphilis, tuberculosis, Bright's disease, albuminoid disease, Addison's disease, and others, the cause of the anæmia is extremely complex. But the majority of the cases of anæmia that are regarded and treated as such fall into the class to which the name of *idiopathic* has been applied. In such cases the anæmic condition

is due, not to any disease so called, but to disturbance of nutrition generally—that is, of the healthy relation between the demands of the system and the supply of nutrient material. This condition occurs chiefly in children and young women, at the period of bodily growth and of the development and early activity of the sexual functions; and when, as so frequently and unfortunately happens, the air, food, occupation, and nervous relations of the individual are all more or less unwholesome.

ANATOMICAL CHARACTERS.—The blood suffers three principal changes in declared anæmia, namely: (1) increase or deficiency (oligæmia) in amount; (2) deficiency in red corpuscles or hæmoglobin (oligocythæmia, aglobulism); and (3) deficiency in albuminous constituents (hypalbuminosis). Of these oligæmia is the rarest, the total amount of blood being often increased (see CHLOROSIS). Aglobulism is a very early and common, as well as the most obstinate, change in the blood. Hypalbuminosis is the most advanced and perhaps the most serious alteration of the three (see BLOOD, Morbid Conditions of). The blood is watery and pale; has a diminished specific gravity; and coagulates slowly and loosely, or in aggravated cases not at all, settling into three layers—consisting respectively of red corpuscles, white corpuscles, and plasma. The body presents certain changes directly due to the state of the blood. Whether the anæmia be local or general, the corresponding parts are blanched and 'bloodless.' The cells of the tissues become atrophied and degenerate, in consequence of, and in proportion to, the interference with their plastic and functional activity respectively; and the so-called 'anæmic' form of fatty heart, liver, kidneys, and other organs is the result. If death occur suddenly from acute anæmia, the heart is found empty and contracted.

PATHOLOGY.—When the volume of blood in the body has been reduced by repeated small hæmorrhages, the phenomena that supervene, while they express the want of blood as a whole, and of its several constituents, are chiefly referable to the loss of two of these constituents—the albuminous substances and the red corpuscles or hæmoglobin, that is, of the oxidisable and the oxidising materials. The same effects will be produced by a drain of the liquid part only of the blood, or by poverty of the blood from any of the causes enumerated above, whether of the nature of waste or of want; inasmuch as loss of plasma speedily affects the nutrition of the red corpuscles. These phenomena constitute the symptoms of the anæmic condition whatever may be its cause; their relative prominence naturally varying according to an immense number of circumstances.

SYMPTOMS.—The subjects of anæmia are usually girls and young women. Their general appearance, which is striking, is one of pallor, debility, and variable loss of feminine fulness. The visible parts of the surface are pallid, often with a tinge of dusky brown on the eyelids and the backs of the hands; the clearness of the complexion varies with the normal pigmentation of the body; the skin is soft, satiny, and rather loose. The mucous surfaces also are blanched; the sclerotic is pearly blue. The loss of flesh may be moderate, or it may be considerable. The extremities are cold, and the legs and lower eyelids are often œdematous. Pyrexia is occasionally present, the temperature rising as high as 102° F. or even more. Bodily strength is

reduced; muscular force is diminished, while myalgia is common; an air of languor and want of vigour pervades the whole demeanour; and the patient is sleepy, dull, and depressed. The subject of anæmia generally complains of weakness, various pains about the body and head, and marked shortness of breath on the least exertion. The last symptom is unaccompanied by other evidence of respiratory derangement; in character the breathing is regular, and short or even panting. The symptoms referable to the circulation consist chiefly of palpitation on exertion; a tendency to faint; and pain or distress over the cardiac region. The physical signs indicate cardiac enlargement of variable degree. A rough systolic murmur is usually heard over the pulmonary artery; a soft systolic murmur at the aortic base, in the mitral area, possibly over the whole præcordia, and even at the scapular apex; the second sound is often universally accentuated. Over the cervical vessels a systolic murmur is commonly audible, followed by a sharp sound; in the neck a loud venous hum. The cervical vessels may throb; the radial pulse is small, weak, and of variable but usually increased frequency and suddenness. There is a tendency to hæmorrhages, especially epistaxis; and petechiæ are occasionally observed. The digestive system is markedly affected, as shown by loss or perversion of appetite; an anæmic, often clean, but variable tongue; dyspepsia, nausea, and sickness after meals or on rising; and constipation, which is present in the majority of cases, and is frequently prolonged and severe. The menstrual functions are almost always deranged: amenorrhœa is common, in some form; menorrhagia is rare (except as a cause of anæmia); dysmenorrhœa is frequently associated; and leucorrhœa is the rule. The urine varies greatly. Headache, tinnitus aurium, and other cerebral symptoms are common. There may be swelling of the optic disc. Blood drawn from the finger shows aglobulism. See BLOOD, Examination of.

COURSE AND TERMINATIONS.—The course of anæmia in this form is essentially slow and progressive, unless it is checked; the *duration* is perfectly indefinite. The course of the symptomatic form will naturally vary with its cause. Idiopathic anæmia rarely terminates fatally; and, when it does so, the event may be referred, with few exceptions, to some complication. Occasionally, however, it progresses steadily to death. See ANÆMIA, PERNICIOUS.

Intercurrent diseases may be expected to be severe in an anæmic condition, in proportion to its degree. See COMBINED DEGENERATION OF THE SPINAL CORD.

DIAGNOSIS.—Anæmia is generally recognised with the greatest ease, and the chief question of diagnosis relates to its *cause*. The first point to be determined, therefore, is whether it is not *symptomatic* of some more grave state, such as tuberculosis, syphilis, albuminoid disease, or some other of the many possible causes of poverty of blood. Having settled that the anæmia is *idiopathic*, we must next exclude two diseases with which it may be confounded, namely chlorosis and leuchæmia. Chlorosis, in which the plasma is not considered to be altered, and which possesses otherwise a special pathology, is expressed by the yellow tint of skin, by the absence of wasting and of dropsy, as well as by other features (see CHLOROSIS). The diagnosis of

pernicious anæmia is fully given in the article on that subject. Leuchæmia is recognised by examination of the blood and spleen. The starting-point of the blood-change in idiopathic anæmia can only be discovered by investigation of all the facts of the case.

PROGNOSIS.—The prognosis of anæmia is favourable as regards life. In simple anæmia from loss of blood, the patient may be assured of speedy and complete recovery. In idiopathic anæmia, however, this promise can be given only when the cause can be removed or avoided. Under favourable circumstances and judicious treatment, improvement will begin almost immediately; and health should be restored after a few weeks or months.

TREATMENT.—The treatment of anæmia, when it is symptomatic of some more grave condition, such as Bright's disease or phthisis, does not require notice here. When blood has been lost in serious quantity, without other injury of consequence, it will be naturally restored if but sufficient time be given, and interference otherwise avoided. Attention to the ordinary rules of health, abundance of food and air, and moderate exercise, will surely if slowly restore the patient, without the administration of a single drug. Even in this case, however, treatment may be of great service, by arresting, if necessary, the cause of the anæmia, such as menorrhagia or epistaxis; and by assisting nature, if the condition should threaten at any time to become intensified by its own effects.

But before the blood can be restored in the large and ill-defined group of cases known as idiopathic anæmia, the unhealthy influences under which the patient is placed, and the functional and other derangements which are usually accountable for the imperfect sanguification, must be discovered and corrected. Each case must be treated on its own merits, routine being avoided. Where the ætiology is complex, treatment must be equally general, and the whole system of life will have to be reformed. On the other hand, in the rapidly growing child and youth, and still more in girls at puberty, the great demand for nutritive material must be duly considered, and every obstacle to its supply removed. When other than direct discharges are draining the blood, they must be checked. Lactation may have to be forbidden; and leucorrhœa and spermatorrhœa will sometimes demand local treatment.

The removal of the cause being thus made the first element in treatment, means must next be adopted for the restoration of the blood. To accomplish this, it will be necessary to bring the alimentary tract and the organs of sanguification into a healthy state. Dyspepsia and constipation require immediate treatment. The best plan is to begin with a brisk purge, and to follow this up with a course of iron and aloes in pills every night, so as to secure one, and only one, daily evacuation of the bowels. Therewith a bitter and alkaline stomachic mixture may be given before meals for a few weeks. The food must be carefully ordered, so that it shall not only supply the albuminous elements that are specially deficient in the blood, but be retained, digested, and absorbed.

The process of sanguification may be successfully assisted by means of drugs. Iron is the sovereign remedy for aglobulism; and, practically speaking, it speedily becomes a question in the medicinal treatment of a case of anæmia in what form iron is to be given. The compound iron mixture of the pharma-

copeia, perhaps, answers more frequently than any other; but, on the one hand, when there is constipation, as is so often the case, combinations of aloes and iron in pill, or a mixture containing the protosulphate with purgative saline sulphates, will be more suitable for a time; on the other hand, when there is a tendency to discharges, the persalts with bitters, combined, if necessary, with sulphate of magnesium, will answer the purpose better. Compounds of iron with quinine or strychnine should be given in cases where less marked anæmia occurs in older subjects, with nervous depression and general want of vigour. In special cases the ferrum redactum, saccharated carbonate, or the vinum ferri (with compound decoction of aloes in constipated subjects), ammonio-citrate of iron, or Blaud's pill may be ordered. Some patients cannot take iron except in the effervescing form; others only if it is presented to them highly diluted—for instance, as a few drops of the tincture of the perchloride in a glass of water. To meet this difficulty, as well as to secure the benefit of many other recuperative influences, a course of chalybeate waters may be the best means that can be devised (see MINERAL WATERS). The addition of arsenic to the iron is invaluable in many cases; in other instances arsenic alone, given as Fowler's solution, proves eminently successful after iron has failed. According to some authorities, manganese assists the action of iron. Cod-liver oil is sometimes prescribed with success. Other symptoms must be treated on ordinary principles. Uterine complaints demand special attention; and bromides, ergot, opium, and other sedatives and astringents are indicated where excitement and excessive discharge are present.

While these dietetic and medicinal measures are being carried out, it is impossible to insist too strongly upon attention to bodily and mental hygiene: the use of the tepid morning bath; the proper disposal of time in relation to exercise, education, and amusement; healthy clothing, or rather 'dress'; the avoidance of unwholesome excitement; early hours; and sufficient sleep. In a large number of cases change of air fulfils many of these conditions. On the other hand, in acute anæmia, as well as in severe cases of every kind, rest in bed is imperative for a few days. Time is an essential element in the cure. A frequent change in the form of the medicinal remedies is also advisable.

J. MITCHELL BRUCE.

ANÆMIA, LYMPHATIC.—A form of anæmia which is associated with a peculiar affection of the lymphatic system. See LYMPHADENOMA.

ANÆMIA, PERNICIOUS.—**SYNON.** : Fr. *Anémie pernicieuse progressive*; Ger. *Progressive perniciose Anämie*.

DEFINITION.—A progressive hæmolytic process affecting both sexes, especially in middle life, characterised by fatty degeneration of the heart and retinal hæmorrhages, and in nearly all cases terminating fatally.

Pernicious anæmia includes two varieties: the primary, essential, or 'idiopathic anæmia' of Addison; and the deuteropathic, symptomatic, or secondary anæmia of German authors. The clinical symptoms in the two varieties are, on the whole, alike. Whilst it is possible that pernicious anæmia is really not a disease *per se*, but a group of symptoms, yet for clinical purposes these symptoms are constant enough to constitute a distinct disease.

ÆTIOLOGY.—Pernicious anæmia occurs about equally in men and women, unless we include all cases of fatal or severe anæmia secondary to uterine hæmorrhage. In women the disease is most frequently met with between the ages of 20 and 40 (the period of child-bearing), whereas in men it occurs most frequently between 40 and 60. The youngest age on record is 7, the oldest is 68. Although the disease is more common among country people than town-dwellers, this cannot be ascribed to their respective occupations.

Among the better classes the disease is uncommon; when it does occur, no cause can be ascertained, excepting, in a few instances, mental worry, grief, or fright. It occurs with especial frequency in countries the inhabitants of which are poor and insufficiently supplied with food, particularly nitrogenous food, and who live in small, over-crowded, badly lighted, and ill-ventilated houses. Repeated pregnancies, especially in women who are badly nourished or suffer from ordinary bloodlessness, are also a cause of pernicious anæmia. It seldom affects primiparæ. Excessive lactation, severe puerperal or other hæmorrhages, dyspepsia, and the uncontrollable vomiting frequently met with during pregnancy, are considered by many authors to rank as causes of the secondary form of this disease: some German authorities include parasites, such as the *Ankylostoma duodenale* and the *Bothriocephalus latus*. Malaria and yellow fever have also been antecedent to this form of anæmia; and Müller has called attention to the connection of constitutional syphilis with this disease. Septic infection has been urged as a cause of the disease, and William Hunter has recently called attention to the frequency with which patients suffering from this form of anæmia are the subjects of septic infection arising from carious teeth and stumps. He considers the disease to be a toxæmia, the result of a specific infection of the digestive tract, and the chief source of infection to be through the mouth in connection with long-continued cario-necrotic conditions of the teeth; sometimes possibly from stomatitis arising from other causes, e.g. drain-poisons.

ANATOMICAL CHARACTERS.—As a rule the body is not emaciated; indeed a thick layer of subcutaneous fat, of canary-yellow colour, is usually found. The muscles are of good colour. The heart and the great vessels contain but little blood; this is of a pale colour and often uncoagulated. The heart itself presents well-marked fatty degeneration, having the characteristic 'tabby cat' appearance. The fatty degeneration affects the ventricles more than the auricles, the left ventricle more than the right, and the muscoli papillares more than any other part. This is the most constant of the morbid anatomical conditions. The cavities are sometimes dilated; the valves are normal. Flecks of fatty degeneration are commonly seen affecting the intima of the large arteries, and the same change probably occurs in the capillary vessels, explaining perhaps the occurrence of petechiæ and retinal hæmorrhages. Small ecchymoses occur in the brain, serous membranes, and elsewhere. Passive effusions into the serous cavities are not uncommon.

All the organs of the body, with the exception of the spleen, look very pale, as if their vessels had been washed out with water. The liver is generally fatty, but is not much enlarged. The spleen may be normal, small, or somewhat enlarged, but never attains great dimensions, as in leucæmia. The

kidneys are pale, often showing slight fatty change. The mucous membrane of the stomach and intestines often exhibits hæmorrhagic erosions; and atrophy of the tubular glands of the former has been described.

The marrow of the bones may be normal, or the red marrow increased; in some cases the yellow marrow of the long bones has been replaced by red—probably a result of the disease rather than the cause of it.

SYMPTOMS.—The patient suffers from weariness, lassitude, headache, vertigo, and dizziness, with faintness, palpitation, and breathlessness on exertion. These symptoms make their approach in so slow and insidious a manner that the patient is unable to fix the date of their onset, and probably does not seek medical advice until he is incapacitated for mental or bodily work.

The aspect of the patient is characteristic: the mucous membranes are pale; the skin is usually of a light lemon-yellow colour, 'like white wax changed by age.' The conjunctivæ are white, though the presence of yellow fat beneath might lead a careless observer to believe there was slight jaundice. The patient is seldom emaciated. There is often slight oedema of the lower eyelids and of the feet, but these symptoms are generally late in appearing. The intellect is usually unimpaired, but answers to questions are slow, apathetic, and delivered in a low tone of voice, sometimes interrupted by long-drawn sighs. Sleep is occasionally, but not usually, heavy, insomnia being a more constant symptom. The tongue is pale, smooth, and dry; the breath offensive, with a cadaveric odour in severe cases. Stomatitis and bleeding gums are sometimes met with. In many cases dyspepsia, flatulent eructations, vomiting, and a painful sinking feeling at the epigastrium are prominent symptoms. These gastric disturbances are attributed by certain authorities to irritation of the cerebral centres by anæmia, by others to atrophy of the gastric glands. The gastric juice in a number of cases has been found to be deficient in hydrochloric acid, and in some few cases this acid has been absent, thus accounting for slow and imperfect digestion, anorexia, and nausea. Some patients, however, eat well, even to excess. Constipation or diarrhoea may occur, but usually the stools are of normal colour. Dyspnoea is sometimes the first symptom complained of. The breathing is generally accelerated, and, in advanced cases, is deep and noisy. The laboured breathing increases with the diminution of hæmoglobin, but not so rapidly as other symptoms, and is often absent as long as the patient is at rest. The action of the heart is regular and frequent. Sometimes there is visible evidence of dilatation, the impulse being diffused, and the area of præcordial dullness increased laterally. Systolic murmurs are heard over the cardiac region, sometimes loudest at the base, sometimes at the apex. A venous *bruit* in the neck is generally present. The pulse ranges between 100 and 120, and is regular, compressible, not small, often jerky, and possibly dicrotic.

One of the most striking clinical features of pernicious anæmia is the supervention of *hæmorrhages*. Epistaxis, menorrhagia, sometimes cerebral hæmorrhage, and towards the close of life petechial hæmorrhages and purpuric eruptions, may occur; but by far the most important are retinal hæmorrhages, because they serve as a diagnostic symptom of great value. The condition of the optic disc is

shown in the accompanying figure (fig. 1). For purposes of comparison reference should also be made to the coloured plates in the article on Ophthalmoscopy in Medicine. The fundus is pale, the arteries are narrow, the veins broad; the hæmorrhages are generally multiple, and flame-shaped, from their situation in the layer of nerve-fibres. They appear as linear striæ, rounded spots or



FIG. 1.—Retinal hæmorrhage. After QUINCKE.

patches, clustered around the optic disc, and may have whitish or yellowish centres, due in part to leucocytes, in part to degeneration. These hæmorrhages may be quickly absorbed, often lasting only a few weeks. There may be no disturbance of vision, unless the hæmorrhages are situated in the maculæ luteæ. In all the six cases observed by the writer retinal hæmorrhages were present.

Pyrexia is the rule in pernicious anæmia. It may be continuous for months, the evening temperature reaching 100° – 101° F.; or it may be intermittent with sudden exacerbations. The pyrexia as a rule is not severe, but occasionally the temperature may rise to 105° or 106° F. On the other hand, it may fall even as much as 10° below normal previous to death. Possibly the intermittent pyrexia may have some relationship to the toxæmia which causes the hæmolytic process.

The urine may be normal in quantity, except at the close of life, when it is often considerably diminished. It seldom contains albumen and never sugar. It is often of a deep colour, containing excess of urobilin, and there may be a direct association between the depth of colour and the hæmolytic process. In other instances, however, it is quite pale. The amount of nitrogen excreted *per diem* may be less than normal; but, as a rule, it is more than can be accounted for by the nitrogenous ingesta. Consequently it must be derived from the excessive waste of blood and tissue; but an incomplete oxidation of the nitrogenous substances leads to an excretion of urea and a deposition of fat. Uric acid often occurs in considerable quantities; indican, pathological urobilin, and pentamethylene-diamine have also been found in the urine, suggesting a morbid process in the gastro-intestinal tract. Free iron has been discovered in the urine by William Hunter.

Blood.—The blood in pernicious anæmia presents no changes absolutely characteristic of the disease. When drawn, it appears pale and watery, and the specific gravity is often greatly reduced. Examined microscopically, it invariably shows a considerable diminution in the number of red corpuscles, which may be reduced to 500,000 per c.mm.— $\frac{1}{10}$ of the normal—or even less in the last stages of the disease, the lowest estimation on record being 143,000 (Quincke). Generally there is a striking variation in the form and size of many of the red corpuscles (*see* BLOOD, Morbid Conditions of—coloured plate). It will be seen that some are much larger than normal (macrocytes); many, again, are pear-shaped or have an irregular contour (poikilocytes); and, finally, there are to be seen numbers of small, imperfectly developed corpuscles (microcytes). In a stained film-preparation numbers of nucleated red corpuscles are usually found, and sometimes eosinophilous cells. The corpuscles are usually of good colour. There is no leucæmia, but granular masses are common. Hayem holds that the presence of nucleated red corpuscles is a grave omen. There is a great diminution of hæmoglobin in the blood—sometimes in proportion to the falling off in numbers of the corpuscles; but, owing to many of the corpuscles being more highly charged with colouring matter than normal, the hæmoglobin-value of the blood may be excessive in relation to the number of the corpuscles. *See* BLOOD, Morbid Conditions of.

The spleen and liver are in some cases enlarged, especially the former. There is also evidence of affection of another important structure connected with the elaboration of the blood—namely, the bones. These may be tender, and the sternum is especially liable to be so affected.

NERVOUS SYMPTOMS.—Lichtheim was the first to call attention to a combined degeneration of the posterior and lateral columns of the spinal cord, in cases of profound anæmia. Some of the cases in which this degeneration has been found are rather cases of associated grave anæmia than of true pernicious anæmia. It is possible that a toxæmia which causes the hæmolytic process may also cause the degeneration of the tracts of nerve-fibres of the spinal cord. Such degeneration cannot be produced by experimental anæmia in animals; and although the anæmia would undoubtedly tend to accelerate a degenerative process, the evidence rather points to a toxin as the essential cause. The symptoms are mostly related to disorders of motility, viz. ataxia and paraplegia (*see* COMBINED DEGENERATION OF THE SPINAL CORD). The anæmia may however be assigned as the cause of various functional nervous disturbances, viz. irritability, loss of memory, insomnia, and in severe cases delirium and other mental disturbances, and convulsions and coma which sometimes precede the fatal termination. Monoplegia, hemiplegia, and other symptoms indicating destructive lesions of the brain may occasionally arise from cerebral hæmorrhage.

COURSE, DURATION, TERMINATIONS, AND PROGNOSIS.—Of 110 cases collected by Sidney Coupland, the total duration of the symptoms in 52 was from 1 to 6 months; in 24 from 6 to 12 months; in 25 from 1 to 2 years; in the remaining 12 cases they lasted for periods of over 2 years. Of 130 cases of primary idiopathic anæmia collected by Pye-Smith, 30 are said to have recovered. Death is usually the result simply of anæmia, and

special complications are exceptional. The prostration and weakness increase; the headache, vertigo, and vomiting become more distressing; the breathing becomes hurried, laboured, and deep ('air-hunger'); the cardiac palpitation is more marked; and the patient usually falls into a drowsy, lethargic state, either lying impassive, or tossing about in restless delirium. Occasionally he remains conscious to the last, unable to sleep, and feeling utterly miserable and dejected in consequence of the distressing headache and vomiting. Death may occasionally occur suddenly from syncope.

Some authors dispute the possibility of true pernicious anæmia being cured, and urge that those cases which recover cannot be considered 'pernicious.' The prognosis is always grave, if not absolutely bad; and even when a patient improves under treatment, and the condition of the blood would give hopes of a cure, a guarded opinion should always be given; for a fresh exacerbation of pyrexia, with hæmolysis, and a return of the symptoms in an aggravated form, generally occur without any apparent cause.

PATHOLOGY.—The examination of the blood and urine during life, taken in conjunction with the changes found in certain organs after death, has thrown some light upon the disease. In health a constant equilibrium is maintained between hæmogenesis and hæmolysis. The pigment of the urine (urobilin) and the pigment of the fæces (stercobilin) are both iron-free derivatives of hæmoglobin. The daily loss of these iron-free derivatives of hæmoglobin affords evidence of a constant and fairly uniform destruction of red blood-corpuscles which is continually being balanced by a corresponding hæmogenesis. The new corpuscles are formed probably in the marrow of bone, although some authorities consider that the blood-platelets may be their antecedents. Experiments on animals in which anæmia has been produced by successive blood-lettings have shown that the marrow of bone here takes on an abnormal functional activity, and that nucleated corpuscles, which are a characteristic of pernicious anæmia, may appear in the circulating blood. There are many reasons for believing that pernicious anæmia is due not to a failure in hæmogenesis but to excessive hæmolysis caused by the absorption from the alimentary canal of a toxine which induces a destruction of the blood in the portal circulation. Quincke first showed that in the spleen and kidneys and especially in the liver an iron-derivative of hæmoglobin (hæmosiderin) existed in so great abundance as to constitute a post-mortem characteristic of the disease. It can be readily demonstrated by placing slices of the organ in a solution of ammonium sulphide which turns it coal black, or better still by placing them in a solution of ferrocyanide of potassium acidulated with hydrochloric acid. If thin sections be prepared in this way, it will be found that the blue particles are most numerous in the portal zone of the lobule. In one acute case, observed by the writer, the blood-corpuscles contained in the branches of the portal vein were in all stages of disintegration, and in the clot numbers of blue-stained granules could be seen, thus affording presumptive evidence that the hæmolytic process was due to the absorption from the alimentary canal of some agent which induced this chemical change in the hæmoglobin. The amount of free iron which can be obtained from a normal liver is comparatively small, but in

pernicious anæmia it is of considerable quantity. Thus from a liver weighing 1,240 grammes Vasey obtained for the writer 27 grammes of ferric oxide. Not only does the liver contain this free iron, but also the spleen and kidneys, though to a much less extent. There is thus evidence of the existence in these organs of iron which is not again utilised for the formation of hæmoglobin. By the side of this fact we must place another, namely the presence of excess of urobilin in the urine, as further evidence of increased destruction of the red corpuscles. Along with this hæmolytic there may be found in a great number of the cases evidence of an increased hæmogenesis in the fact that there is an increase of red marrow and often a conversion of the yellow marrow into red. This myelogenic change in some cases is so marked that some authorities consider it to be a cause of the disease and not a result.

Now red corpuscles containing a due amount of hæmoglobin cannot be manufactured without iron; and although in pernicious anæmia the colour of the corpuscles may be even darker than normal, this does not show that they contain a sufficiency of iron. Since there is a large amount of iron lying useless in the liver, it might be suggested that this organ has a ferrogenic function (Delépine) which is disarranged, so that the iron cannot be re-utilised and that consequently although the disease may be primarily hæmolytic yet there is a disturbance of the hæmogenic function, progressive in character. Not only, however, do the corpuscles diminish in number, but the specific gravity of the blood is often much lower than normal. The evidence seems to support the view that there is absorbed from the alimentary canal some poison which causes chemical change in the blood of the portal system. The fact that in many cases the hydrochloric acid is diminished or even absent from the gastric juice would support the view of a change in the mucous membrane, possibly due, as Hunter suggests, to an infective catarrh, especially when gastric disturbances form so prominent a feature of the disease. The absence of the hydrochloric acid, however, alone could not cause pernicious anæmia, for in cancer of the stomach, in which this acid is usually absent, we do not have the clinical and

possible that the normal vital resistance to the absorption of poisonous substances engendered by fermentative processes occurring therein might be in abeyance, and poisons could be absorbed which would lead to an altered condition of the portal blood, disintegration of the corpuscles and deposition of iron in the liver, with excess of the iron-free derivative of the hæmoglobin (urobilin) in the urine. The fever which often shows exacerbations coincident with evidence of increased hæmolytic suggests a toxæmia; the fact that a few successful cures have been reported (Gibson) by antiseptics such as β -naphthol suggests the plausibility of this hypothesis. The writer's experience does not support the view that the anæmia is due to hæmorrhages. Some authors include in pernicious anæmia cases of grave and fatal anæmia due to definite causes, such as *Ankylostoma duodenale*; it may however be remarked that in connection with the anæmia caused by this parasite there is no great excess of free iron in the liver. The evidence in favour of a specific bacterial cause is still very incomplete.

Although no *specific* organisms have been found in the blood or organs in this disease, yet many of the symptoms—viz. the gastro-intestinal disturbances, the peculiar cadaveric odour of the breath, the irregular attacks of pyrexia, and the presence of indican and other bodies in the urine—suggest the absorption by the portal circulation of some poison formed by the growth of micro-organisms. It has been shown that the blood in pernicious anæmia, as in many septic diseases, after being drawn allows hæmoglobin-crystals to form spontaneously. When arsenic is administered, the blood is said to lose this property, suggesting an explanation of the value of the drug in the treatment of the disease. Again, if putrid serum be added to healthy blood, crystals of hæmoglobin form. It is possible therefore that some intestinal poison sets up a hæmolytic process in the portal circulation, and that the hæmoglobin is broken up in the liver—into urobilin, which escapes by the urine, and free iron, which remains in the cells and capillaries of the portal zone. The urobilin, which passes from the hepatic veins into the general circulation, gives rise before its excretion to the lemon-yellow colour of the skin and fat (urobilin-jaundice).

DIAGNOSIS.—The principal diagnostic features of pernicious anæmia are the following: (1) the patient is usually *middle-aged*; and, except in the case of women, there is nothing to account for the onset of the disease; (2) the absence of any organic disease; (3) the severe and progressive anæmia; (4) the great diminution of the corpuscles and colouring matter of the blood; (5) alteration in the shape and size of the corpuscles and the presence of nucleated corpuscles; (6) signs of fatty degeneration of the heart, without arterial degeneration or valvular disease; (7) retinal hæmorrhages; (8) irregular and occasional pyrexia; (9) no emaciation, but frequently rather an excess of subcutaneous fat; (10) lemon-yellow coloured skin and high-coloured urine; (11) gastric disturbances; (12) carious teeth and stomatitis.

The diseases for which pernicious anæmia has been mistaken are Bright's disease, ulcerative endocarditis, tuberculosis, Addison's disease, cirrhosis of the liver, cancer of the stomach and malignant disease of other internal organs, chronic ulcer of the stomach, parasitic affections, such as ankylostomiasis and pseudo-leucæmia myelo-

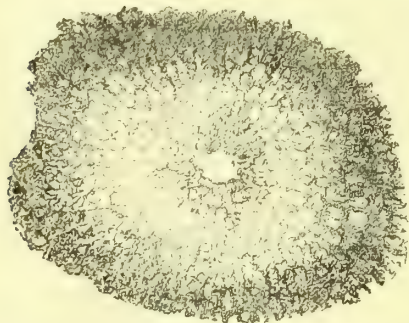


FIG. 2.—Section of the liver showing a single lobule after treatment with ferrocyanide of potassium solution and hydrochloric acid. The dark shading indicates the deposit of iron in the portal zone. $\times 75$.

pathological phenomena of the disease, notwithstanding the associated anæmia (see BLOOD, Morbid Conditions of). But if the *infective* catarrh spread to the intestine, as suggested by Hunter, it is

genica. It occurs at an age when idiopathic anæmia or chlorosis is unlikely to be met with; moreover an examination of the blood should serve to differentiate it. See BLOOD, Examination of.

TREATMENT.—Preparations of iron are useless in pernicious anæmia, and occasionally even injurious. Most of the recorded cases of recovery have been treated with arsenic. Although it is not a specific, it generally prolongs life. Liquor arsenicalis should be given in 3-minim doses, rapidly increased to 10 minims, three times a day; and if it cannot be tolerated, on account of gastric disturbances, it may be administered subcutaneously. Hayem recommends in addition the inhalation of 15 litres of oxygen a quarter of an hour before each of the two principal meals; and after meals a one per cent. solution of hydrochloric acid. Ferratin and bone-marrow are also remedies which have been tried with varied success. A few most successful results have been obtained by the use of antiseptic drugs such as salol, β -naphthol, and bismuth salicylate. If, as seems probable, the toxine is generated in the alimentary canal by a fermentative process of microbial origin, and absorbed by the portal system, it is easy to understand that drugs which would arrest such a process might yield beneficial results. Irrigation of the intestine and *lavage* of the stomach have also been tried in some cases with apparent success. It is necessary to give a nutritious diet, very easy of digestion, such as milk, beef-tea, eggs, scraped raw beef, and meat-juice. It is sometimes necessary to limit the diet to peptonised or humanised milk, whey, and white of egg. If vomiting be very severe, nutrient enemata may be used. Transfusion has been tried in some cases, but it is doubtful whether it has ever done more than prolong life for a short time. Defibrinated human blood should be used, and not more than half a pint should be injected at once.

FREDERICK W. MOTT.

ANÆSTHESIA: Local.—**DESCRIPTION.**—To produce local anæsthesia or, more correctly, local analgesia, one of the salts of cocaine or its analogues is injected into the tissue about to be operated on. The detailed method now employed is based upon the observation of Corning that cocaine applied to a sensory nerve in its course abolishes sensation to pain throughout the tissues to which it is distributed. A sterilised watery solution of hydrochlorate of cocaine (1 to 2 per cent.) is injected by means of a fine hollow needle and syringe all over the area to be operated on. In the first place it is distributed along the line of first incision directly under the epidermis, i.e. in the papillary layer of the skin, in such a way as to produce a white wheal. Then by successive injections it is distributed layer by layer through the subjacent tissues, following as far as possible the distribution of the nerves. If done properly the tissues are rendered analgesic in about five minutes and can be incised and dissected without causing pain for about half an hour. This analgesia may be rendered more complete and rapid in the case of a hand, foot, finger, or toe, by previously checking the return of blood from the part by encircling the part on the central side with a piece of rubber-tubing.

There is, however, always some risk of the toxic effects of cocaine being developed. This has led to a search for some equally effective but less dan-

gerous anæsthetic, and such appears to have been found in an allied chemical substance, β -Eucaine hydrochloride, which seems to be less toxic than cocaine in the proportion of one to five. This is now used in the following solution: β -eucaine 1 part, sodium chloride 8 parts, water 1000 parts. The addition of eight parts of common salt per thousand of water neutralises the osmotic effects of the latter, and thus eliminates one element in the production of pain. Such a solution may be used in almost any amount required in the manner above described, as much as 10½ oz. having been employed in a single operation without any ill effect. To avoid causing pain the solution must be used at body-heat and injected slowly. A syringe, sterilised by boiling, and having a capacity of 10 c.c., is the best instrument to employ. The most convenient method for making the solution is to have powders made up in papers containing enough β -eucaine and pure sodium chloride to make 7 oz. of solution. This amount of boiling water is placed together with one of the powders in a beaker and boiled for a few minutes. The vessel is then covered to keep out impurities and placed in a basin of cold water until cooled to blood-heat, after which it is kept in a warm water-bath to maintain the desired temperature.

No toxic effects result from the use of β -eucaine employed in this way. But there are certain disadvantages from the operator's point of view. It is difficult for him to divest his mind of the impression that the patient, who is perfectly conscious, is also sensible of pain, especially as the sense of touch is not abolished. He is therefore inclined to operate more slowly and deliberately. Indeed this is necessary: for it must be remembered that any dragging on tissues may affect structures outside the zone of analgesia and so produce pain. For instance, in the case of a radical operation for the cure of inguinal hernia, though the first incision and dissection may be quite painless, any dragging on the cord will be felt acutely, as the proximal part of this structure cannot be reached by the injection. Bearing this fact in mind, however, we may avoid the evil by careful manipulation, though with some expenditure of time. The artificial oedema produced by the injection is of little moment in most operations, but would be dangerous about the mouth or fauces, and the method should never be used there.

The chief advantages of the method are the avoidance of the risks, immediate and remote, of general anæsthesia. Moreover, the patient is not denied food before or after the operation. There is usually no vomiting, and this in itself eliminates much distress and some risk, especially in abdominal operations. The method is seen at its best in such cases as strangulated hernia in old people who are much run down, or where colotomy has to be performed. These patients are proverbially bad subjects for chloroform or ether, and suffer much in many cases from subsequent vomiting. Again in the excision of thyroid-tumours the method answers admirably. The writer during the last few years has employed the method in numerous other operations, such as those for the radical cure of hernia, for empyemata, for intestinal anastomosis, and for the removal of small tumours.

The method has recently been extended in a very remarkable way. Professor Bier caused some cocaine solution to be injected into his own lumbar spinal cord and into that of a friend. The result was com-

plete analgesia of both lower extremities. As a result of these experiments amputations of the legs have been performed under similar conditions, and, according to the published accounts, absolutely without pain. The cases, however, are not numerous, and are insufficient to enable surgeons to decide as to the practical utility of the method.

ARTHUR E. BARKER.

ANÆSTHETICS, GENERAL.—DEFINITION. General anæsthetics may be defined as drugs capable of temporarily inducing *general surgical anæsthesia*, i.e. a loss of consciousness and sensibility throughout the entire organism.

METHODS OF PRODUCING ANÆSTHESIA.—The anæsthetics commonly employed are nitrous oxide, ether, chloroform, and the A.C.E. Mixture.

Nitrous oxide is usually supplied, in liquefied form, in suitable cylinders. Fifteen ounces of the liquefied anæsthetic will furnish about fifty gallons of gas. Nitrous oxide may be inhaled (1) in its pure state; (2) it may be given mixed with air, either by letting the patient take an occasional breath of air, or by allowing the admixture of a definite percentage of air (10 to 20 per cent. according to the type of patient) with the anæsthetic gas before it is inhaled; or (3) it may be administered mixed with oxygen (5 to 9 per cent.), a regulating stopcock being employed. Two cylinders should be coupled together in case one is emptied during the administration. The average quantity of nitrous oxide required to anæsthetise patients for dental operations is five or six gallons, but if breathing 'to and fro' into the bag be permitted towards the end of the administration, a smaller quantity will suffice.

Ether prepared from rectified spirit is preferable to that obtained from methylated spirit, although the latter variety may pass the Pharmacopœial tests. Great care is necessary in using ether in the proximity of a naked flame, as the vapour is highly inflammable. There are two systems of etherisation, which may be termed the *semi-open* and the *close* respectively. In the first or *semi-open* system a felt cone or Rendle's mask is used; ether is poured upon the sponge within the cone or mask, and all expirations escape in the air. The second or *close* system involves the employment of a bag-inhaler, and there are two methods for applying this system, viz. Clover's and Ormsby's.

Chloroform should be obtained in small bottles, which should be kept in a cool dark place. The author has been unable to detect any clinical differences between Duncan and Flockhart's chloroform prepared from rectified spirit and the chloroform of the same firm prepared from methylated spirit. There is only one system at present known of employing chloroform, viz. the *open* system, without re-breathing; but there are several methods of applying this system. Of these two may be mentioned—Skinner's and Jünker's. Skinner's mask, upon which chloroform is sprinkled from a drop-bottle, has advantages over lint, handkerchief, or towel, in that it is more manageable, and the chloroform is less likely to injure the skin. Jünker's inhaler is principally of use when only analgesia is required, as in normal labour, and for maintaining anæsthesia during operations about the upper air-passages. It is a mistake to suppose that Jünker's inhaler necessarily robs chloroform of its dangers. For general use a Skinner's frame is preferable to any other appliance.

The A.C.E. mixture, which consists by volume of alcohol 1 part, chloroform 2 parts, and ether 3 parts—the last two constituents being made from rectified spirit—is useful in many cases. Equally good results are obtainable without alcohol. The mixture is best administered by means of a Skinner's mask, followed by a Rendle's mask, but in the case of infants the former will suffice.

It is advisable in many cases to use first one anæsthetic and afterwards to change to another. The following '*successions of anæsthetics*' are valuable: (1) the nitrous oxide—ether sequence; (2) the A.C.E.—ether sequence; and (3) the ether—chloroform sequence. The best method of administering 'Gas and Ether' is to adapt a partly full gas-bag to a Clover's portable ether-inhaler. Clover's combined gas and ether apparatus is too cumbersome and complicated to be recommended. The great advantage of this sequence is that unconsciousness is rapidly induced without discomfort, and without struggling or excitement. The second of the sequences mentioned is valuable when nitrous oxide cannot be obtained. Anæsthesia is induced by the A.C.E. mixture, and at the commencement of rigidity the Rendle's inhaler is changed for an Ormsby's apparatus charged with ether. The third of the sequences mentioned is one which is often called for in consequence of difficulties experienced in etherisation. While it is an exceedingly valuable one, considerable care and experience are needed in changing from ether to chloroform. As a general rule, patients should evince distinct signs of approaching recovery from ether-anæsthesia before chloroform is applied, and under no circumstances should a change be effected during violent struggling.

PREPARATIONS AND PRECAUTIONS.—However simple the case may appear to be, it must never be forgotten that the administration of an anæsthetic is a responsible task. Indeed, as a general rule, difficulties are more likely to arise in perfectly healthy patients, about to undergo or undergoing trivial operations, than in cases which are apparently more serious. Anæsthetics should never be administered by unqualified persons. It is necessary to have had considerable experience before even moderate success can be achieved, and lives are undoubtedly often sacrificed owing to the administration of anæsthetics being placed in incompetent hands. There are certain preparations and precautions to be adopted on all occasions. (1) The anæsthetist should have at hand a Mason's gag, tongue-forceps, an efficient form of mouth-opener, instruments for tracheotomy, two or three pieces of sponge, a small mouth-prop, a towel and a small basin. (2) All constricting clothing should be unfastened, artificial teeth removed, and loose teeth carefully noted. (3) With regard to diet, an interval of five hours between the last meal and the administration is generally advisable. For operations before 10 A.M. nothing whatever should be given after the evening meal of the preceding day. For midday operations a light breakfast of tea and toast five hours before may be permitted. In the case of very feeble subjects an enema of beef-tea and brandy an hour before the operation is advantageous. (4) An aperient should be given the night next but one before the operation, and an enema on the morning of the operation. (5) The best posture for the administration is the supine, the head, but not the shoulders, being slightly raised and turned to one side. In

cases of intestinal obstruction, or in others in which vomiting has been taking place, the patient should be anæsthetised with one shoulder raised by a pillow, and the head turned well to the opposite side. Whatever posture be adopted, the head should be neither flexed nor extended upon the trunk. The semi-recumbent posture should be avoided, and the same may be said of a special posture, which is erroneously regarded as suitable for operations upon the throat, viz. that in which the head is extended over the end of the table. Patients should be instructed to breathe through the mouth, rather than through the nose. (6) The chest should always be examined before selecting the anæsthetic.

CHOICE OF ANÆSTHETICS.—Whenever practicable, nitrous oxide should be employed, either with oxygen, with air, or alone, since it is the safest known anæsthetic. If inadmissible or unobtainable, ether should be chosen. In other words, nitrous oxide and ether should be the two routine anæsthetics for general surgical work, the A.C.E. mixture and chloroform being reserved for special cases and for special operations.

THE PATIENT.—As a rule women are more easily anæsthetised than men. Infants and senile subjects usually take anæsthetics well, the A.C.E. mixture producing good results. Men of powerful build with thick necks must be anæsthetised with care. Feebly developed subjects usually pass into anæsthesia without trouble. Vigorous, full-blooded, alcoholic patients, and very nervous subjects usually require considerable quantities of the anæsthetic, and the excessive use of tobacco may give rise to difficulties. Patients suffering from any respiratory difficulty, as well as those in whom respiration is particularly prone to become embarrassed under the anæsthetic, must be treated with caution. The worst subjects for anæsthetics are (1) obese patients suffering from acute intestinal obstruction, with vomiting and abdominal distension; (2) cases of emphysema with much dyspnoea; (3) patients with dyspnoea from tracheal pressure, especially if the obstructed breathing be associated with bronchial secretion or pulmonary oedema; and (4) fat, flabby, alcoholic men suffering from some exhausting disease. Special care is needed in anæsthetising patients with nasal obstruction or enlarged tonsils. In many of such cases, a small mouth-prop should be inserted between the teeth. Those who are subject to dyspnoea should be permitted to lie in the position most comfortable to them: when anæsthetised they can be cautiously moved. Generally speaking, the presence of even moderate dyspnoea calls for chloroform; but patients with chronic bronchitis, emphysema, and chronic lung-affections may usually be successfully anæsthetised by the A.C.E. mixture. The subjects of *morbus cordis* usually take anæsthetics well, provided that no asphyxial strain be thrown upon respiration or circulation. As a general rule, the A.C.E. mixture gives the best results in cases of advanced heart disease. 'Close' etherisation should be avoided in this class of patients. Should there be a history of antecedent cerebral hæmorrhage, ether is contra-indicated.

THE OPERATION OR CONDITION REQUIRING ANÆSTHESIA.—Short operations within or about the *mouth, nose, and nasopharynx* may be performed under nitrous oxide or ether. The available period of anæsthesia after a dose of pure nitrous oxide is thirty seconds; after a full administration of nitrous

oxide and oxygen, forty-four seconds; after three or four minutes' etherisation, about ninety to one hundred and twenty seconds. By administering ether for ten or twelve minutes an anæsthesia of about four or five minutes may be secured. Tooth-extraction, the removal of nasal polypi, tonsils, and adenoid growths, and numerous other short operations may thus be performed. When a longer anæsthesia is desired, it is usually best to maintain it by means of chloroform, employing a Jünker's inhaler for the purpose, the tube of which is passed into the nose or mouth. A large experience is needed to decide what particular degree of anæsthesia should be maintained. The best posture for cases of this group is, undoubtedly, the lateral. In this posture the tongue may be removed, the antrum opened up, and numerous other operations performed without the slightest embarrassment to breathing. When the actual cautery is to be used, the patient must be under chloroform, as ether-vapour is inflammable. Should the purely dorsal or the sitting posture be employed, careful and repeated swabbing with coarse sponges, unattached to holders, will be needed. Speaking generally it is advisable to retain the swallowing and coughing reflexes in these cases. In intra-laryngeal operations (for which chloroform is needed) these reflexes must be abolished. For operations about the *neck*, and particularly in thyrotomy, tracheotomy, and thyroidectomy, chloroform should be used.

In *abdominal surgery* very deep anæsthesia is needed, and, unless the administrator has had a large experience with chloroform, he should employ ether, with which anæsthetic the margin of safety is sufficiently large to allow of its being freely pushed. It is not always safe to abolish all traces of reflex abdominal rigidity, although this can be done in most cases. In very prolonged operations, chloroform, or some mixture containing it, should be used in preference to ether. There is often considerable shock in these cases, especially during manipulations in the region of the solar plexus.

In operations upon the *pleura or lung* it is generally taught that chloroform or the A.C.E. mixture should be given; and this course may be followed in most cases. But, in the worst subjects, ether (administered by the semi-open system) is to be recommended, since, if respiration become suspended even for a short time while chloroform is being used, fatal syncope may rapidly follow.

In operations upon the *genito-urinary organs and rectum* very deep anæsthesia is needed: and ether is preferable to other anæsthetics. Operations upon the *kidney* are often attended by considerable shock. The same remark applies to operations upon the *breast*. In *cerebral surgery* chloroform is generally indicated.

When anæsthetics are required for their analgesic effects, as in *normal labour* and *biliary colic*, chloroform may be advantageously chosen. When profound muscular relaxation is needed, as in reducing dislocations, very deep ether-anæsthesia is safer than a corresponding state of chloroform-narcosis. Muscular relaxation cannot be depended upon under nitrous oxide, even when administered with oxygen.

PHENOMENA OF ANÆSTHESIA.—(a) *Nitrous oxide*. The phenomena will depend primarily upon the system of administration adopted. When the pure gas is administered through accurately working valves, and when each expiration escapes, the

following phenomena appear:—respiration becomes deeper and quicker; the pulse smaller and more frequent, and the colour of the face increasingly dusky. The pupils dilate; deep guttural stertor becomes audible; and clonic muscular twitchings of an epileptiform character make their appearance. The average inhalation-period is about one minute, and it is mechanically impossible to administer the gas after deep stertor has set in. The average available period of anæsthesia after removing the face-piece is thirty seconds. The lividity, stertor, and jactitation may all be lessened or completely removed by administering oxygen with nitrous oxide. For this purpose a regulating stopcock is necessary, and, after starting with 2 or 3 per cent. of oxygen, the percentage may be increased to 8, 9, 10, or even 20 per cent., according to the type of subject. In the best forms of anæsthesia thus induced, the breathing is sleep-like or softly snoring, and the pulse slightly accelerated; there is little or no change of colour; and clonic movements are completely absent. The effects produced by nitrous oxide and air are more satisfactory than those obtainable by the pure gas, but less satisfactory than those produced by nitrous oxide and oxygen. Careful preparation as to diet is necessary in using the last-named mixture, as there is undoubtedly a greater tendency to vomiting during and after its use than in the case of nitrous oxide given alone or administered with occasional breaths of air. Stertor and 'jactitation'—one or both—are the signs that pure nitrous oxide has been administered to its full extent; and when either or both occur, fresh air must be admitted. Caution is needed in pushing the administration to its full extent in short-necked subjects, in patients with affections of the throat or nose, in very young children, in persons with any marked dyspnoea, and in senile subjects. The signs of non-asphyxial anæsthesia are softly snoring breathing, and an insensitive conjunctiva.

(b) *Ether*.—This anæsthetic is usually given by Clover's Portable Regulating Inhaler, which, in cold weather, and when about to be used for anæsthetising powerfully built subjects, should be very slightly warmed. The figures on the inhaler indicate the extent to which the to-and-fro air-current is made to pass over ether, and not the relative proportions of ether and air. When properly used, anæsthesia can be secured in about three and a half minutes without excitement or struggling.

The following method of administration should be adopted:—1. The patient's head should be turned well to one side. 2. He should be instructed to breathe freely through the *mouth*. 3. The inhaler, charged with ether, and with the indicator at 'o,' should be applied so as to catch sufficient expired air to fill the bag. 4. To-and-fro breathing, with the indicator at 'o,' should be permitted for half a minute. 5. The ether reservoir should then be rotated continuously but almost imperceptibly, '1' being reached at the end of the first minute, '2' at the end of the second minute, and '3' at the end of the third minute. 6. One breath of fresh air should be admitted when stertor begins. 7. After this breath of air has been given, the patient's colour, rate and depth of breathing, and general condition, will indicate the extent to which further supplies of fresh air may be allowed. When once the cornea has become in-

sensitive to touch, the inhaler should be removed and fresh air admitted at frequent intervals. No definite rule applying to every case can be formulated; but the weaker the patient and the longer the administration, the greater should be the quantity of air allowed. Should excitement and struggling occur, the administration must be continued. In very exhausted subjects but little ether will be needed, and the air-supply must be ample. The signs of anæsthesia are:—Regular snoring breathing; very slight duskiness, disappearing when plenty of air is allowed; moderate dilatation of the pupil; general muscular flaccidity; a quick, full pulse; and an increased secretion of mucus and saliva. When ether is given by the semi-open system, excitement and struggling generally occur, large quantities of the anæsthetic are needed, and several minutes elapse before surgical anæsthesia takes place.

c. *Chloroform*.—It is a mistake to administer this anæsthetic either too sparingly or too recklessly. In the former case, prolonged emotional disturbances, lividity, pallor, vomiting, and feebleness of pulse will be produced, while anæsthesia will be delayed; and syncope may even be initiated. In the latter case, 'holding the breath,' coughing, struggling, and asphyxial complications may ensue. It is also important to remember that with concentrated vapours a dangerous or fatal dose of chloroform may easily be inhaled. A very weak vapour should be used at the outset, and a continuous atmosphere of a *moderately strong* vapour kept up till anæsthesia begins to appear. Should excitement and struggling arise, frequent breaths of fresh air must be permitted. By degrees the hampered breathing of the excitement-stage will subside, and regular softly snoring respiration will become audible; the muscles will relax, the pulse will become somewhat slower than the normal; the face less florid; the corneæ insensitive to touch; and the pupils moderately contracted. An attempt should be made to maintain this condition, taking as guides (1) the corneal reflex, (2) the respiration, (3) swallowing movements, (4) the pupil, (5) the state of the muscular system, (6) the colour of the face, and, when working without a trace of corneal reflex, (7) the pulse. In many patients it is possible to retain a very slight corneal reflex; in such cases, provided respiration be free and there be no indications of shock, the pulse may be disregarded. The pulse is, however, of great value as a guide in profound anæsthesia, for it will become slower and smaller with more chloroform, and quicker and fuller with less. Provided the patient be properly anæsthetised, the breathing will become quieter with less chloroform, and deeper with more; but in too profound an anæsthesia more of the anæsthetic will induce a quiet form of breathing from incipient bulbar paralysis.

As compared with ether anæsthesia, that of chloroform is less satisfactory so far as respiration and circulation are concerned. But by carefully watching the above signs it is possible to administer chloroform safely. The utmost vigilance, however, is necessary in conducting the administration in cases requiring the profound effect of the drug.

d. *A.C.E. Mixture*.—The effects of this are almost identical with those of chloroform, except that the pulse and breathing are of a more satisfactory type.

THE DIFFICULTIES, ACCIDENTS, AND DANGERS CONNECTED WITH ANÆSTHESIA.—Much miscon-

ception prevails with regard to the nature and mode of origin of the various difficulties which may arise in connection with surgical anæsthesia. It is probably not an exaggeration to say that in five out of six cases in which dangerous symptoms arise, such symptoms are not directly dependent upon an overdose of the anæsthetic. There is a remarkable tendency under all anæsthetics for respiration to become embarrassed, and this tendency is, if anything, more marked during light anæsthesia, when the muscular system is not relaxed, than during surgical narcosis. Again, it by no means follows, because a patient is exceedingly weak or is the subject of cardiac disease, that there will be an unusually great liability to dangerous symptoms. The strongest and most vigorous patients often give the greatest trouble to the anæsthetist.

(a) *Primary respiratory failure.*—Respiration may be interfered with in one of three ways:—(1) from occlusion of the upper air-passages, (2) from conditions affecting the bronchi, pleuræ, or thoracic parietes, or (3) from paralysis of the nervous mechanism of respiration. The symptoms presented by the patient will depend upon the anæsthetic employed, the degree of anæsthesia at the time of the arrested breathing, and the cause of the arrest.

(1) Occlusion may take place at various points. The teeth may become clenched; the tongue engorged and swollen; and the nasal air-way shut off by the soft palate. This state is not uncommon in certain subjects during incipient anæsthesia. Or the tongue may be spasmodically drawn backward in the course of stertor, till the glottis is completely covered. 'Falling back of the tongue,' in its literal sense, is rare, only occurring in certain positions of the head, and in complete muscular relaxation. Partially performed deglutition will necessarily arrest breathing, and is, perhaps, the most common cause of temporary suspension of the respiratory functions. Laryngeal spasm is also a common cause of obstruction. It may be of reflex origin, i.e. due to the operation, or it may depend upon local irritation caused by the anæsthetic vapour, or by mucus or blood. There is also a paralytic state of the superior aperture of the larynx which may induce obstruction. Again, foreign bodies, including blood, mucus, pus, and vomited matters, may obstruct breathing. Lastly, there are various pathological conditions of the naso-pharynx which may cause obstruction during anæsthesia, either from alteration in position of parts, or from increase in size owing to venous engorgement. Whatever the nature of the occlusion may be, the effects produced upon the patient will be the same, viz. the appearance of asphyxial symptoms. If the anæsthesia at the time be light and the circulation good, increasing duskiness will occur, abortive attempts at breathing as evidenced by thoracic and abdominal movements will continue for a time, the pulse will gradually fail, and pallor may replace cyanosis. A vigorous man who becomes asphyxiated owing to obstructive stertor under nitrous oxide may retain a pulse at the wrist for several minutes; whereas a flabby and obese subject with a dilated heart, who is being anæsthetised by chloroform, may lose his pulse almost immediately if the slightest embarrassment to breathing take place. Such a case may easily be mistaken for one of primary cardiac failure. The treatment in all cases should be to remove the slightest embarrassment to breathing immediately it is detected. The lower jaw should

be pressed forwards; the head slightly extended; the mouth opened by a Mason's gag; the tongue-forceps applied; any foreign body removed; and forcible compression of the chest employed. Blood, mucus, &c., must be removed by sponging, attention being paid to posture. Should the larynx have been invaded by these fluids, forcible compression of the chest, with sponging, will usually suffice to restore breathing. Forcible traction of the tongue will usually relieve laryngeal spasm. If these measures fail, artificial respiration should be attempted with the hope of overcoming the obstruction, and if this be fruitless tracheotomy must be performed. (2) Little need be said with regard to cessation of breathing dependent upon conditions affecting the bronchi, lungs, pleuræ, and thoracic parietes, for in such cases some pathological condition is generally present. Spasm of the respiratory muscles, which is occasionally met with during the stage of struggling and excitement, is, however, a serious complication. It usually passes off spontaneously, but occasionally resists all treatment. Artificial respiration, forcible inflation of the lungs, and venesection constitute the treatment mostly likely to succeed. (3) With regard to paralytic cessation of breathing, this takes place as the immediate result of a toxic dose of the anæsthetic. There is no obstruction; there is no spasm; but breathing fails, sometimes gradually, sometimes abruptly. The treatment is immediate artificial respiration. The mouth should be opened, the tongue pulled forwards, and Sylvester's method of artificial respiration applied. See ARTIFICIAL RESPIRATION and RESUSCITATION.

(b) *Primary circulatory failure.*—It occasionally happens that the pulse ceases at the wrist while respiration is proceeding regularly. This condition may arise under all anæsthetics, and, while it undoubtedly calls for careful attention, and in many cases treatment, it by no means implies imminent danger. By far the most common cause of the condition is the surgical procedure itself. Surgical shock may occur in all stages of anæsthesia. It may be characterised by slight evidences of cardiac inhibition—the pulse becoming slow, feeble, or irregular—or it may be so marked as apparently to threaten the life of the patient. It is questionable, however, whether surgical shock, occurring independently of hæmorrhage, is ever actually fatal. It is most common in renal, abdominal, mammary, and cervical operations. It may take the form either of cardiac inhibition or of vaso-motor paralysis, leading to an accumulation of blood in the venous system. In minor cases of surgical shock the lips and cheeks should be briskly rubbed with a cloth, and somewhat less anæsthetic given. In more grave cases the anæsthetic should be withheld, the head lowered, the legs raised, strychnine injected, and an enema of hot brandy and water administered. In the worst cases intra-venous saline injections should be given, and posture and freedom of respiration carefully studied. When chloroform is administered in toxic doses the pulse may cease before respiration, even in the healthiest subjects, but with ether this never occurs, save in patients in a very feeble condition. Primary circulatory failure has also been met with by the writer under nitrous oxide in a patient with aortic disease. Impending vomiting is sometimes associated with feeble or absent pulse. The treatment for primary circulatory failure produced by the anæsthetic and not by the operation is artificial respiration. The

very grave error has often been committed of treating this condition by drugs and stimulants. Experience shows these latter remedies to be absolutely useless. Indeed they are actually dangerous because they may stand in the way of prompt and efficient blood-aëration. Attention to posture is important, and when any symptoms of syncope arise, partial or complete inversion may be advantageously adopted in combination with artificial respiration. By way of summary it may be said that, putting aside surgical and pathological causes of difficulties, reliance must be placed upon artificial respiration and posture, and that any other measures should be regarded as of secondary importance.

AFTER-EFFECTS OF ANÆSTHETICS.—The after-effects of anæsthetics depend upon the presence or absence of food within the stomach at the time of the operation, and upon the duration of the administration. There are of course other factors, such as the type of subject, the quantity of anæsthetic used, and the kind of anæsthetic. The after-effects of nitrous oxide are very slight, provided care be taken in regulating the diet. Vomiting after ether is more common than after chloroform, although protracted vomiting is more often met with under the latter anæsthetic. In the experience of the writer the best results, so far as the absence of after-effects is concerned, are to be obtained by the gas-ether-chloroform sequence, the first two anæsthetics being used for inducing anæsthesia, and chloroform for maintaining it. By keeping the head turned upon one side, so as to allow the escape of mucus, and by maintaining an equable anæsthesia, after-effects are less likely to occur than under other circumstances. As a general rule no nourishment should be given till the patient feels inclined to take it. After short administrations a little tea or coffee is often much appreciated. After long administrations clear soup, beef-tea, or solid meat-essence may be given. Milk and eggs must be avoided. In cases of much nausea or vomiting two-ounce doses of very hot water may be taken. Inhalations of vinegar are also recommended. In very severe cases a blister may be applied to the epigastrium. It is important that directly the administration is suspended the patient should be placed upon his side, in order that mucus or vomited matters may pass freely from the pharynx and mouth; and he should be left undisturbed in a quiet and darkened room. Jolting and other movement of the bed should be, if possible, avoided. The room should be well ventilated, but care should be taken not to subject the patient to draughts.

F. W. HEWITT.

ANAKHRE.—**SYNON.** : *Goundou*; *Henpuye*. *Fr. Gros nez.*—This singular deformity, the result apparently of a chronic osteoplastic process affecting the adjacent parts of the superior maxillæ and nasal bones, is not uncommon in the natives of certain parts of the West Coast of Africa, particularly on the Ivory and Gold Coasts and their respective *hinterlands*. It occurs, though rarely, among West Indian negroes. In well-marked examples oval, symmetrical, smooth, bony outgrowths form prominent swellings at the sides of the nose. In some instances the growths are so large that they interfere with the line of vision, the patient being obliged to lower the head in order to see over them. The integuments are not involved, nor is there any obstruction of the nasal ducts or of the nostrils.

The swellings consist of a thin cortex of dense bone enclosing a core of cancellous tissue. They commence—according to Chalmers—during or shortly after an attack of yaws—with nasal discharge and much headache. After a time the discharge and pain cease but the swellings continue to increase. Growth may be spontaneously arrested at any stage, or it may steadily progress till each mass attains the size of a goose's egg, producing a hideous deformity and perhaps completely occluding vision, although not destroying the eyes. Occasionally, though rarely, the disease is confined to one side of the face. It never ulcerates, breaks down, or spontaneously subsides.

Chalmers considers that anakhre is a sequel of yaws, the result of the interference with the nutrient vessels of the bones—a consequence of intra-nasal ulcers. Further evidence is however required before this view of its origin can be considered as established. It is difficult on such an hypothesis to account for the symmetry of the swellings and for the singularly limited geographical range of the disease. Macleod regards anakhre as parasitic; Strachan suggests that it may be a racial peculiarity: neither of these views can explain all the facts.

TREATMENT.—The swellings are easily removed by surgical means and do not recur.

PATRICK MANSON.

ANALGESIA (ἀ, priv.; and ἄλγος, pain).—Absence of sensibility to painful impressions. *See* SENSATION, Disorders of.

ANAPHRODISIA (ἀ, priv.; and Ἀφροδίτη, Venus).—Absence of sexual appetite. Sometimes used to express impotence. *See* IMPOTENCE.

ANAPHRODISIACS.—**DEFINITION.**—Medicines which diminish the sexual passion.

ENUMERATION.—The agents employed as anaphrodisiacs are:—Ice; Cold Baths—local and general; Bromide of Potassium; Bromide of Ammonium; Iodide of Potassium; Conium; Camphor; Digitalis; Purgatives; Nauseants; Circulatory Depressants; and Bleeding.

ACTION.—The erection which occurs in the genital organs during functional activity is due to dilatation of the arteries in the erectile tissues, and is regulated by a nervous centre situated in the lumbar portion of the spinal cord. From this centre vaso-inhibitory nerves pass to these arteries, and cause them to dilate whenever it is called into action. It may be excited reflexly by stimulation of the sensory nerves of the genital organs and adjoining parts, or by stimuli from the alimentary canal. It may also be excited by psychical stimuli passing to it from the brain. Some anaphrodisiacs may act by lessening the excitability of the nerves of the genital organs, as the continuous application of cold, and probably, also, bromide of potassium; some by diminishing the excitability of the genital centres in the spinal cord and brain, as bromide and iodide of potassium, and conium; others by influencing the circulation, as digitalis. There are also adjuvant measures, of a hygienic and moral character, which greatly assist and may even replace anaphrodisiac medicines, such as a meagre diet, especially of a vegetable nature, the avoidance of stimulants, and the pursuit of active mental and bodily exercise. Everything tending to stimulate the genital organs, or to increase the flow of blood to them or to the lumbar portion of the spinal cord, should be avoided, such

as warm and heavy clothing, or pads about the hips or loins; and a hard mattress should be used in place of a feather-bed. Everything likely to arouse the passions, such as certain novels, pictures, theatrical representations, &c., should also be shunned.

USES.—Anaphrodisiacs are employed to lessen the sexual passions when these are abnormally excited in satyriasis, nymphomania, and allied conditions. As such excitement may sometimes depend on local irritation of the genitals, in consequence of pruritus of the external organs, excoriations of the os uteri, or balanitis; or on the presence of worms in the rectum or vagina; these sources of excitement should be looked for, and, if present, should be subjected to appropriate treatment.

T. LAUDER BRUNTON.

ANARTHRIA (ἀν, neg.; and ἄρθρον, a joint).—**SYNON.**: Fr. *Anarthrie*; Ger. *Gliedlösigkeit*.—A term applied to those defects of speech consisting of disordered articulation.

ANASARCA (ἀνά, up; and σάρξ, the flesh).—An effusion of serous fluid into the subcutaneous connective tissues, not limited to a particular locality, but becoming more or less diffused. See DROPSY.

ANEURYSM.—An aneurysm may be defined either as a localised dilatation of an artery; or as a sac, containing fluid or partially coagulated blood, in communication with an artery. It may result from the weakening of the wall of the vessel (1) by disease; (2) from some congenital defect of development; or (3) from direct injury. The disease which most commonly leads to the formation of aneurysm is arterio-sclerosis, especially where syphilis is one of its causal factors. The combination of syphilis, alcoholism, and over-exertion, which frequently occurs in soldiers, renders them specially liable to aneurysm; but it is also common in labourers and in those whose occupation involves the lifting of heavy weights. Its relation to over-strain seems to be indicated by its vastly greater frequency in Great Britain, where the labouring classes work much harder than on the continent of Europe. Acute arteritis, especially the septic form found in malignant endocarditis, where the intima of the aorta is first damaged by the impact of the fragments of the ulcerated valve and then necrosed by the pyogenic organisms which attack it, is an occasional cause of aneurysm of the first part of the aorta. What is termed *embolic aneurysm* may be produced in one of the smaller arteries, such as those of the brain, by the impaction in it either of a calcareous plate derived from an atheromatous aorta, or of a portion of a diseased valve containing micro-organisms. The inner and middle coats in the former case are eroded by the mechanical action of the sharp edge of the calcareous plate, and, in the second, are necrosed by the organisms contained in the embolus. As will be easily understood, such aneurysms are very prone to rupture. The outer coat of an artery as it passes across, or along the wall of, a phthisical cavity in the lung, may be attacked and necrosed by the tubercular process, so that the two inner coats bulge through the weakened spot and produce the so-called *hernial aneurysm*, the rupture of which produces the hæmoptysis that so often proves fatal in advanced pulmonary tuberculosis. Men are more subject to aneurysm than are women, in the proportion of 8 to 1, and they tend to be specially affected between the ages of

40 and 50. Where aneurysm occurs at an early age a congenital defect, a syphilitic lesion, or an embolic origin, may be suspected.

According to their method of formation aneurysms may be fusiform, saccular, or dissecting. The *fusiform* variety is simply a dilatation of a portion of an artery in which the inner and middle coats are more or less degenerated, but in the walls of which all the coats are present. The *saccular* aneurysm is generally produced by the rupture of the inner and middle coats of the artery, frequently as the result of some definite overstrain. A sac is thus produced which communicates with the artery by a small opening, and is bounded externally by the outer coat, with here and there remnants of the inner and middle coats in various stages of fatty and calcareous degeneration. In the *dissecting* variety one or both of the inner coats becomes ruptured, the blood forces its way between the coats for a varying distance, and then either escapes back again into the vessel or, as often happens in aneurysm of the aorta, into the pericardial sac. Such aneurysms have been found extending from the arch of the aorta as far as the division of the abdominal aorta into its two branches. In such cases the aneurysm forms a second tube between the walls, through which the blood passes before it re-enters the channel of the vessel.

The fusiform dilatation of the artery when once established does not necessarily continue to increase, but a saccular aneurysm tends to progressive enlargement, although the rate of this enlargement varies very greatly in different cases. In its growth it causes an irritative proliferation of the surrounding connective tissue, which for a time gives some support to the thinning outer coat of the artery, but in its turn also becomes attenuated by the continuous stretching. If the sac approaches bone or cartilage, it causes absorption of both, but the erosion is much more rapid in the case of bone, as its vascularity favours the occurrence of a rarefying osteitis, whereas the cartilage is merely absorbed from its surface. If the aneurysm approaches a serous membrane, it may cause an obliterative inflammation in the cavity, as is commonly the case in the pleura, or it may rupture into the serous sac before adhesion takes place, as is more frequent in the case of the pericardium. Pressure upon a mucous membrane may lead to its perforation, as frequently occurs in the trachea, and more rarely in the œsophagus and stomach. Pressure upon a blood-vessel may lead to the narrowing and occlusion of the vessel, or to rupture into it, with the establishment of an aneurysmal varix, as may happen, though somewhat rarely, in the superior vena cava. When an aneurysm approaches the skin it seldom ruptures externally, although such a result often appears imminent. When it does, however, death is not necessarily immediate. Saccular aneurysms may contain fluid blood or, more frequently, laminated thrombi. In aneurysm of the larger arteries, it is rare to find any considerable degree of organisation in such thrombi, doubtless owing to the imperfect adhesion of the thrombus to the wall, to the firmness of the clot, to its constant disturbance by the blood-current, and to the degenerate state of the vessel-walls forming the sac. Notwithstanding this, the thrombus breaks to a certain extent the force of the blood-current on the wall of the sac, and has thus a certain remedial power by limiting the constant tendency to expansion, and, when a mucous surface is approached, by

delaying the moment of rupture. See ABDOMINAL ANEURYSM; BRAIN, Aneurysm of; HEART, Aneurysm of; THORACIC ANEURYSM.

ALEXANDER BRUCE.

ANGIECTASIA (ἀγγεῖον, a vessel; and ἔκτασις, extension).—Extension or hypertrophy of the capillaries and minute vessels of the surfaces of the body, especially the skin; hence *angiectasia capillaris*, a term applicable to several forms of vascular nævus. See TUMOURS: *Angioma*.

ANGEIOLEUCITIS (ἀγγεῖον, a vessel; and λευκός, white).—Inflammation of lymphatic vessels. See LYMPHATIC SYSTEM, Diseases of.

ANGINA (ἄγω, I seize by the throat, strangle, or choke).—SYNON.: Fr. *Angine*; Ger. *die Bräune*.

The term *angina* was originally applied to conditions in which difficulty of breathing and of swallowing exist either together or separately, caused by disease situated between the mouth and the lungs, or between the mouth and the stomach. By a special affix to the original term, significative of the seat or the nature of the disease, several morbid processes are known and described, for example:—*angina parotidea*, or mumps; *angina tonsillaris*, or quinsy; *angina laryngea*, or laryngitis; *angina Ludovici*, diffuse suppurative cellulitis in the submaxillary region; *angina pectoris*, or breast-pang; *angina maligna*, or malignant sore-throat; *angina membranosa*, or croup.

ANGINA PECTORIS.—SYNON.: *Syncope Anginosa*; *Angor Pectoris*; Suffocative Breast-pang; Stenocardia. Fr. *Angine de poitrine*; Ger. *Brustbräune*.

DEFINITION.—A paroxysmal affection of the chest, characterised by severe pain, faintness, and anxiety; coming on more or less suddenly; essentially connected with disorders of the pneumogastric, the sympathetic, and spinal nerves, and their branches; and frequently associated with structural disease of the heart and arteries.

ÆTIOLOGY.—In searching for the *causes* of *angina pectoris*, we frequently notice: (1) The well-known conditions which lead to arterio-sclerosis and myocarditis. (2) Morbid changes in the nerve-tissues. (3) The existence of a peculiar condition of the nervous system, which may be described as an undue susceptibility to impressions. It would seem to be often hereditary, and to be found in those temperaments in which there is a high development of the nervous element. (4) The influence of *age* in relation to *angina* is conspicuous; the disease is rare before puberty; and quite eighty per cent. of the cases occur after forty years of age. (5) *Sex* also displays a marked influence among the predisposing causes of this disease. True *angina pectoris* is comparatively rare among women. Out of 49 fatal cases, collected by Forbes, only 2 occurred in females, and out of 15 non-fatal cases only 2—facts which entirely correspond with our experience. In the hysterical temperament a form of spurious or false *angina* is not uncommon. (6) It is associated in many instances with certain habits of life, such as sedentary employments and indulgences at the table, and occasionally with those states of the system termed hysterical. (7) Certain toxic agents, either metabolic as in gout and diabetes, or extraneous, as tobacco and lead.

ANATOMICAL CHARACTERS.—In a few cases no lesions have been found *post mortem*. The following have been the most important changes.

(a) Involvement of the nerves, either of the branches of the cardiac plexus in mediastinal tumours, in aneurysms, and in adherent pericardium, or positive changes in the intrinsic nerves and ganglia, of which there have been a number of observations recorded.

(b) Lesions of the arch of the aorta and of the coronary arteries, which are the most constant anatomical changes. * In the aorta there may be chronic arterio-sclerosis, with much atheroma and calcification, or there may be acute aortitis, as seen in syphilis. The changes in the coronary arteries may be either fibroid or calcareous.

(c) The myocardium may be normal, but in many cases it has been fibroid; in others there has been extensive fatty degeneration. Huchard gives 145 autopsies gathered from the literature. In 17 cases there was mention only of a lesion of the coronary arteries without further specification; of 128 there were 68 with lesions of both coronary arteries, 37 of the left vessel, 15 of the right, and in 12 the seat of the lesion was not stated. In the 128 cases obliteration or stenosis of the vessels had occurred, and of these in 121 there was atheromatous narrowing or thrombosis, in 5 embolism, and in 2 compression.

It may be said that, while there is often in *angina pectoris* an absence of any evident structural disease, the morbid state producing the anginous symptoms may be situated in the medulla oblongata; in the course of the nerves, or in their branches; or in the cardiac ganglia themselves. It may be the result of congestion or inflammation of the nerves, such as occurs in the gouty diathesis; or of other textural changes, such as connective-tissue growth or other growths involving the nerve-fibres and ganglia. An attack may be produced by emotions acting centrifugally; or by irritation acting centripetally, reflected, as just said, through the vaso-motor centre, from impressions made on the peripheral extremities of nerves. Thus acidity of the stomach, the result of indigestion, often gives rise to symptoms which very closely resemble, if they do not constitute, an attack of *angina*. It is by a similar mechanism that anginal symptoms have been produced by irritation reflected from the fifth nerve, as in pivot-teeth; by such irritation of the surface of the skin as results from severe herpes; by cold, or by exposure to wind. But the most frequent source of the symptoms of true *angina* is to be found in those structural affections of the heart already described.

The attack has been ascribed to cramp in the heart-muscles, distension of the chambers, and to an intense neuralgia of the nerves of the heart. Of late years the theory of *intermittent claudication* has been brought forward to explain the phenomena of the attack. Allan Burns, of Glasgow, first formulated the theory in the following terms: 'In health, when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action the heart and every other part has its power augmented. If, however, we call into vigorous action a limb round which we have with a moderate degree of tightness applied a ligature, we find that then the member can only support its action for a very short time, for now its supply of energy

and its expenditure do not balance each other; consequently, it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence. A heart, the coronary vessels of which are cartilaginous or ossified, is in nearly a similar condition; it can, like the limb begirt with a moderately tight ligature, discharge its functions so long as its action is moderate and equal. Increase, however, the action of the whole body, and along with the rest that of the heart, and you will soon see exemplified the truth of what has been said, with this difference, that as there is no interruption to the action of the cardiac nerves, the heart will be able to hold out a little longer than the limb.' This theory has been accepted by Potain, and gives perhaps the most satisfactory explanation of the attack. Irregular action, with weakness of the myocardium, results from the blocking of a main branch or from spasm of the arteries. Not that this or any other theory accounts in full for the agonising character of the pain in the attack; and there are plenty of cases in hospital practice in which we find all the arterial conditions (general and cardiac) which favour intermittent claudication without the history of attacks of pain.

SYMPTOMS.—An attack of angina pectoris commences suddenly with pain in the region of the heart, generally on a level with the lower end of the sternum; occasionally it may be traced from a remote point, following the course of the nerves, even from the ends of the fingers. The pain is severe, and of an oppressive, grasping, crushing, or stabbing character. It extends sometimes across the chest, but more frequently backwards to the scapula, and upwards to the left shoulder and arm. The pain is accompanied by a distressing sense of sinking, of faintness, which causes the patient to seek support, or of impending death. The action of the heart is generally irregular. The pulse at the wrist corresponds; but in many well-marked cases of angina it is tense and resisting, yielding a sphygmographic tracing indicative of extremely high tension, more especially in the early stage of the attack. A fear of aggravating the pain prevents the patient from breathing deeply, though the respiratory function may not be really interfered with. The expression is anxious, the face is pallid, the lips are more or less livid. The whole surface of the body is pale, cold, and covered with a clammy sweat. Flatulence is often present; urine in some cases is passed at short intervals, and generally in abundance.

The attack, more or less severe, having lasted a variable time—from a few minutes to one or two hours—comes to an end, either suddenly or by degrees. The pallor and coldness of the surface are replaced by a uniform glow—the face may even flush; the pulse becomes soft and full; and there is a general feeling of relief. A sense of numbness or tingling occasionally remains along the course of the nerves derived from the brachial and cervical plexuses of the affected side.

All the phenomena of an anginal seizure as above described may be more or less modified. The attack may come on when the patient is at rest, and occasionally it sets in during sleep; but it is more commonly induced by emotion or by physical exertion, especially by walking up an ascent or by exposure to cold air or wind. The pain may be comparatively slight, and as such may recur with interruptions, it may be, during months or even

years, representing anginoid symptoms rather than true angina pectoris. On the other hand it may be so severe as to mark a first, a single, and a fatal attack.

The pain may be almost limited to the region of the heart, or the lower part of the sternum; it may extend all over the chest to both arms, or spread to the sides of the head and neck and down one or both legs; and it may, in some cases, involve the diaphragm. The action of the heart may be slow, weak, and fluttering; or excited and bounding—causing a distressing sense of palpitation; it may be regular or irregular. Obstruction to the circulation from spasm of the arterioles is among the most constant phenomena of the seizure. The breathing sometimes assumes an asthmatic character, with comparatively little pain. There may be much wheezing, and a condition resembling acute emphysema. There may be laryngeal spasm. The mental functions are generally undisturbed; but there is sometimes slight wandering as the attack passes off, and unconsciousness is said to be occasionally observed. The sense of danger of impending death is a characteristic symptom of angina, and one not often absent; while a sensation of gasping or choking, with difficulty in swallowing, is occasionally present. The position of the patient varies. Sometimes he stands, resting his arms on any convenient object; sometimes he stoops or leans forward, unwilling to be disturbed, even to speak. As a rule the attack passes off abruptly, as it commenced, leaving the sufferer free from discomfort; in other cases its disappearance is slow. The varieties in the symptoms of angina pectoris, thus seen to be remarkably numerous, characterise a form of disease which may be comparatively mild and of long duration, or one of intense suffering, hastening to a fatal termination.

COMPLICATIONS.—Among the diseased conditions with which angina pectoris may be said to be associated, rather than complicated, are disorders of the liver and of the digestive organs, gout, renal disease, diabetes, and certain affections of the nervous system. Indeed, so marked is the latter connection, that Trousseau dwelt on the relation which he believed to exist between epilepsy and angina—a relation which seems to depend on that susceptibility to nervous maladies already noticed as presented by some individuals, rather than on any special pathological connection between these two diseases. The subjects of angina may present the features of the so-called Stokes-Adams disease—permanent slow pulse with vertigo, syncope, or epileptic convulsions.

PROGRESS, DURATION, AND TERMINATIONS.—The progress and duration of angina pectoris will depend wholly upon the nature of its cause. Many cases in which a first attack proved fatal have been recorded. The symptoms in these cases were those of the most severe form of angina pectoris. On the other hand, cases present themselves in which symptoms may occur at intervals for years. Thus in many instances individuals present all the symptoms of marked angina, accompanied by most of its distressing phenomena, and by the anxieties and fears that they beget; yet these cases, having a neurotic or gouty origin, are controlled by treatment, the sufferers being restored to health, or continuing for years to enjoy comparative comfort. On the other hand, in the cases in which angina is connected with structural disease of the heart or of

the nerves controlling cardiac action, the attacks progress in frequency and severity, and tend, with more or less certainty, to a fatal termination—it may be within a few days or weeks.

OTHER FORMS OF ANGINA.—Under the terms pseudo-angina, neurotic angina, and angina pectoris vaso-motoria are described cases of cardiac pain which, while it may be of terrible severity, yet experience has taught us is, as a rule, without the gravity of the true angina. Nothnagel brought forward the view that this form is associated with spasm of the arteries, and introduced the term angina pectoris vaso-motoria, under which heading Douglas Powell has grouped the hysterical, toxic, and reflex varieties.

It is by no means easy to make a good classification. The hysterical and neurosthenic varieties are very common, occurring chiefly in women, often persisting for many years, and, while causing great alarm and much distress to the poor victim, they are so rarely fatal that a hopeful prognosis can always be given. Occasionally a fatal termination may happen, as in one case lasting many years, in a young man of twenty-six, a neurosthenic, and much addicted to tobacco, in whose heart and arteries *post mortem* no change whatever could be determined.

DIAGNOSIS.—A typical case of angina pectoris, such as has been already described, can hardly be mistaken. But when the several symptoms constituting an attack are variously modified, some being lessened in severity and others exaggerated; or when these symptoms depend on, so to speak, remote and removable causes; it is often difficult to say how far the disease is what may be regarded as a remediable neuralgia, or an attack of what is commonly recognised as true angina pectoris. So also it may be difficult to say, in cases of true angina, whether the seizure is dependent on structural lesions which admit of no improvement, or on some condition that may be amenable to treatment. But it is with this affection, as with so many others, more difficult to determine the nature of the cause on which the symptoms depend, than merely to recognise the presence of the disease itself. With reference to the diagnosis of the structural diseases of the heart above referred to, it is unnecessary to describe here that which will be found fully treated of under special heads. It remains but to say that in every case the closest scrutiny must be made into the condition of the heart and great blood-vessels, with a view to determine the presence or absence of disease. The investigation must further extend to other viscera, such as the liver and stomach, and to the digestive organs generally, as well as to the several other sources from which symptoms of angina may be excited by the reflected irritation already described. Certain symptoms resulting from the presence of other diseases should not be confounded with angina—such, for example, as the pain and dyspnoea caused by pressure of aneurysms or of tumours within the chest; by rheumatic or gouty neuralgia of the chest-walls; by pleurodynia, or acute pleurisy; or by indigestion, acidity, and flatulence. Each and all of these several conditions must be considered by way of exclusion in determining the nature and origin of the disease.

PROGNOSIS.—In anticipating the future of an attack of angina pectoris, we must be guided chiefly by a knowledge of its cause; in some respects also by its severity; and by the previous history of the case. Thus, if we can ascertain that the attack has

been brought on by some clearly established and removable cause, such as fatigue, a chill, or indigestion, a favourable prognosis may be fairly entertained. On the other hand, if the history of the case tells that there have been several previous attacks, each increasing in severity and connected with heart-disease, we can scarcely avoid being led to the conclusion that the complaint will tend, with more or less rapidity, to a fatal termination. Between these two classes of cases there exist a large majority of instances in which the symptoms of angina, of greater or less severity, depend on neurosis, on gouty diathesis, or on other sources of nerve-disorder, amenable to treatment; and in which, therefore, a favourable prognosis may to some extent be given. But in all cases great caution should be exercised; for many instances occur in which, from slight and obscure beginnings, severe and even fatal examples of the disease have been developed.

TREATMENT.—The treatment of angina pectoris must, first, have reference to relief of the attack itself; and, secondly, during the interval, to the removal, if practicable, of the causes on which the attacks may depend.

During the attack, it is necessary first, if possible, to lessen the patient's anxiety and fear. He should be allowed to retain the position in which he feels most comfort, and an attempt must immediately be made to relieve the suffering. If the exciting cause be one that can be removed, this should be accomplished. For example, if the stomach be full of undigested food, an emetic of mustard might be given with advantage; or if flatulence be present, peppermint, ether, and other antispasmodics will be useful. If cold have produced the seizure, the feet and hands should be immersed in hot water, hot bottles applied to the surface of the body, and poultices of linseed or mustard, or embrocations of chloroform or laudanum, should be applied to the chest. For the more immediate relief of pain some of the agents now known to be effectual for the purpose should be administered. The nitrite of amyl, originally introduced by Lauder Brunton, has been found a more efficient remedy than any other hitherto recommended for the direct relief of the distressing symptoms of the disease. Five or six minims of this drug, which is usually dispensed in capsules, should be carefully inhaled from a handkerchief or piece of lint, and, if necessary, the inhalation may be repeated.

Nitro-glycerin, suggested by Murrell, seems also to act efficiently in doses of $\frac{1}{100}$ grain, in the form of the official tabellæ or 1 per cent. alcoholic solution. The dose may be repeated three or four times at short intervals; but these drugs must be used with caution, as in many cases they produce headache and superficial congestion.

The action of these agents depends on the power they possess of relaxing the arterioles, thus diminishing the peripheral resistance, freeing the heart's action, and readjusting the circulation. As a rule, in the severer paroxysms morphine must be used, and should be given freely. It is to be remembered that, as Burney Yeo pointed out, these patients show a remarkable tolerance of this drug. The inhalation of chloroform may be necessary and is often useful while waiting for the morphine to act.

In those cases in which debility and exhaustion exist, the ordinary stimulants will be required, and various antispasmodics, such as ether, ammonia, &c., may be used. A hypodermic injection of

ether with nitro-glycerin may be given at once. In cases of sudden cardiac failure MacWilliam recommends faradic excitation of the heart, in the form of a series of periodic single induction-shocks, at the normal rate, approximately, of cardiac action. These should be sent through the heart so as to affect the auricles as well as the ventricles, one electrode being applied over the seat of the cardiac impulse, the other over the fourth dorsal vertebra. Large sponge-electrodes should be used, they and the skin being well moistened with salt and water. The shocks employed should be strong—sufficient to excite powerful contraction in the voluntary muscles. In a few cases cardiocentesis has been performed. Inhalations of oxygen are strongly recommended by Douglas Powell.

During the intervals.—It is desirable to avoid all causes likely to bring on an attack of angina, such as mental and sexual excitement, bodily exertion, exposure to cold, and indulgence in indigestible food or heavy meals. The leading principle in treatment, however, should be to endeavour to determine and to remove the cause of the attacks. Whether they depend on organic disease of the heart, on simple neuralgia, on gout or dyspepsia, on debility, or on fulness of habit—to each of such conditions must appropriate treatment, as described in other parts of this work, be directed. The use of tobacco should be prohibited. A variety of specific remedies has been recommended, such as iodide of potassium or sodium in diseased states of the arteries, arsenic, phosphorus, iron, zinc, and the different sedatives and antispasmodics. In cases with a history of syphilis, and in middle-aged men with marked arterio-sclerosis, the use of the iodide may be followed by permanent cure. Bradbury advocates the erythrol tetra-nitrate, which has a very persistent influence upon the blood-pressure. Galvanism, in the form of the continuous current from thirty cells, has proved of use in some uncomplicated cases, the positive pole being placed on the sternum, and the negative on the lower cervical vertebræ. Huchard, in his exhaustive work on angina pectoris, discusses the action of antipyrin and phenacetin, which he regards as useful in false angina. He describes as useless, or even dangerous, the employment of chloral hydrate, atropine, and certain other drugs. Excellent, however, as many of the remedies above named may be under special and suitable circumstances, the result of treatment must entirely depend on the cause of the disease, and how far it is within reach of well-directed agencies. Some cases of apparently severe angina will be found to yield to treatment; while, as might be expected from the nature of the complaint, others unhappily proceed to a fatal termination in spite of every effort directed to their relief.

RICHARD QUAIN.

WILLIAM OSLER.

ANGIOKERATOMA.—This name has been applied to a peculiar superficial form of multiple hæmangioma. The large amount of epithelium covering the lesions causes them to assume a wart-like appearance.

Angiokeratoma makes its appearance in children or in young people and lasts throughout life, but after the patient reaches adult years the disease shows little tendency to increase. The condition affects especially persons who suffer from difficulty or feebleness of the circulation of the blood in the extremities. Thus it is common in those who

suffer from chilblains, and has been seen not infrequently on the extremities of those suffering from lupus erythematosus. The disease does not necessarily involve the actual area affected by chilblains. As the disease progresses, the hands become thickly studded over with small tumours. A congenital tendency to the formation of angiomas also plays a part in the causation of the disease. It has, moreover, been observed that angiokeratoma may appear in several members of the same family.

The chief anatomical lesion consists of well-marked permanent dilatations of the superficial capillaries of the affected part. On section these are obvious to the naked eye, and on microscopic examination are found full of red and white blood-cells and fibrin, which may be seen in various stages of degeneration, where thrombosis has occurred. The dilatations are separated from each other by more or less substantial partitions of connective tissue, in which a certain amount of cellular infiltration may be present. Some observers state that the lymphatic vessels of the part have also undergone dilatation. The cavernous spaces produced in this way ultimately fill the papilla and come in contact with the overlying epithelium.

It is difficult to determine if the hæmangioma thus produced is caused by the permanent dilatation of the pre-existing blood-vessels, or by the formation of new vascular spaces, or by the bursting of the blood-vessels into the lymphatic channels. The amount of epithelial change which takes place is very variable. The epithelium over the affected papilla is usually thin, but between the papillæ it is increased in amount and penetrates deeply into the substance of the tumour. On the whole, a considerable amount of thickening of epithelium occurs, its keratinisation appearing to proceed normally. The stratum granulosum appears always to be present. As the epithelial processes penetrate into the tumour, the cavernous spaces are surrounded, thrombosis occurs, and, the epithelium becoming gradually rubbed off, these spaces show as small dark points on the surface of the tumour.

The disease shows itself in the first instance as small dark red congested spots. In a short time this little area becomes more sharply limited and shows itself as a deep purple spot or papule from two to five millimetres in diameter. By coalescence of neighbouring lesions even larger tumours may make their appearance. On careful examination of the surface minute dark red points may be observed corresponding to the vascular spaces of the angioma. Later, as the epithelium increases in amount and keratosis occurs, the purple spots become raised, hard, and more or less warty. As might be expected, pricking of the surface of the little tumours produces a considerable loss of blood.

When the angiokeratoma has passed into the warty stage, it remains permanent. It is probable, however, that many of the tumours gradually atrophy, leaving small pigmented spots. As a rule, very little inconvenience is caused by the actual presence of the lesions, though occasionally a certain amount of itching is complained of. The lesions are often scattered in large numbers over the affected area, and have no tendency to any special arrangement. They are most usually found on the dorsal and lateral surfaces of the fingers, more rarely on the palmar surfaces. They occupy similar positions on the feet, and they have been observed on the ears and other parts.

The foregoing description of the lesions and their ætiological relationships will enable angiokeratoma to be distinguished from the two conditions most resembling it, verruca vulgaris, and the superficial hæmangioma or lymphangioma.

TREATMENT.—The small tumours may be readily destroyed if necessary by a fine point of the thermo-cautery or galvano-cautery, producing a very minute scar, or they may be very successfully destroyed by careful electrolysis.

JAMES GALLOWAY.

ANGIOMA (ἀγγείον, a vessel).—A tumour consisting mainly of vessels. See TUMOURS.

ANGIONEUROTIC ŒDEMA.—SYNON.: *Acute circumscribed Œdema*; *Quincke's Disease*.—This term is applied to a characteristic form of localised evanescent swelling of the skin.

After slight prodromal symptoms connected with the alimentary tract, swellings appear on the skin with remarkable suddenness, affecting as a rule the face, extremities, and genitals. These swellings take the form of sharply circumscribed tumours varying in size up to that of the palm of the hand. The smaller ones are hemispherical, the larger ones flattened. By coalescence large areas of the skin become affected. The swellings are tense, and present the peculiar waxy or almost translucent appearance suggestive of œdema, or are in some cases reddish in tint. The surrounding skin may be of normal appearance, but is usually erythematous. The tumours persist for a few hours, or as long as two days, and then vanish rapidly. During the attack feelings of tension or warmth may be remarked, but there is a remarkable absence of pruritus or other uncomfortable sensations. Fresh lesions may appear on other parts of the body, and although the duration of each lesion is short, the duration of the attack may extend for an indefinite period. Usually the attack passes off in a few days, but a marked tendency to recurrence is the rule, and after a variable interval a new outbreak appears.

The mucous membranes may become involved, and when the buccal and pharyngeal regions are affected tracheotomy may become necessary. With the exception of such complications, and the discomfort produced by the tumours of the skin, the general health is as a rule maintained. It is reported, however, that albuminuria and hæmoglobinuria have occurred during attacks of the disease.

The disease seems to be the result of sudden exudation of blood-serum into the skin and connective tissues, apparently under the direct control of the vaso-motor nerve-apparatus. It is difficult to associate local irritants with the appearance of the lesions; and any relationship between this disease and disturbance of the functions of the alimentary tract is not nearly so readily established as in the case of the polymorphous erythema, and urticaria, with which angioneurotic œdema has many points in common. A family tendency to this disease has been observed.

TREATMENT.—Local sedative remedies, such as dusting-powders or weak solutions of the liquor plumbi acetatis, may occasionally be used with advantage; but as pruritus gives little trouble, it is usually sufficient to protect the oedematous surfaces from injury. Internal medication has been used freely with very little good result; the administration of ergot and salicin has been strongly

recommended. It is advisable, with our present knowledge, to regulate the diet so as to obtain efficient digestion and assimilation of food, and to avoid articles of food or drugs likely to cause erythematous and urticarial lesions.

Salt-water douches to the spinal region, with subsequent frictions of the skin, have been highly recommended in chronic cases of the disease.

JAMES GALLOWAY.

ANIDROSIS (ἀ, priv.; and ἰδρώς, sweat).—Absence or want of perspiration. See SUDORIPAROUS GLANDS, Diseases of.

ANIDROTICS (ἀ, priv.; and ἰδρώς, sweat).—Agents which check perspiration. See SUDORIPAROUS GLANDS, Diseases of.

ANILINE POISONING.—The aniline dyes, used for dyeing stockings, gloves, &c., are apt to produce an intense form of inflammation and vesication of the skin, which is rebellious against treatment, and liable to relapse many months after the original attack has subsided. See DERMATITIS.

ANILINE, Workers in.—See OCCUPATION DISEASES.

ANKYLOSIS (ἀγκύλος, crooked).—Partial or complete immobility of a joint due to fibrous bands (*fibrous a.*) or to bony union (*bony a.*) between the ends of the bones. When the immobility is due to extra-articular changes, the term *false ankylosis* is employed. See JOINTS, Diseases of.

ANKYLOSTOMA (ἀγκύλος, crooked; and στόμα, a mouth).—A genus of nematode worms. See ENTOZOA.

ANODYNES (ἀ, priv.; and ὁδόνη, pain).—DEFINITION.—Medicines which relieve pain by lessening the excitability of nerves or of nerve centres.

ENUMERATION.—Anodyne medicines include Opium and its alkaloids—Morphine and Codeine; Phenazone (Antipyrine), Acetanilide (Antifebrin), Phenacetin, and Exalgin; Bromide of Potassium; Cannabis indica; Belladonna and its alkaloid—Atropine; Hyoscyamus and Hyoscyamine; Stramonium; Cocaine; β-Eucaine; Aconite and Aconitine; Veratrum and Veratrine; Conium and Conine; Lupulus and Lupulin; Gelsemium; Chloroform, Ether, and their allies; Chloral Hydrate; Butyl-chloral Hydrate; Menthol and Camphor.

ACTION.—Pain is due to a violent stimulation of a sensory nerve being conveyed to some of the encephalic nerve-centres (probably the cerebral hemispheres), and perceived there. The impression produced on all sensory nerves, except the cephalic nerves, is conveyed, for a part of its course, to the head along the spinal cord. The primary impression which is felt as pain is usually made upon the peripheral ends of the sensory nerves; but it may also be made upon their trunks, upon the spinal cord, or possibly upon the encephalic centres directly, without any affection of the nerves themselves, as, for example, hysteria. Pain may therefore be relieved, while the source of irritation still remains, by lessening the excitability of the ends of the sensory nerves which receive the painful impression; of their trunks; of the spinal cord along which the impression travels; or of the encephalic centre in which it is perceived. Opium

act by lessening the excitability of the sensory nerves, the spinal cord, and the encephalic ganglia. Bromide of potassium is also believed to act on all three, although to a much less degree than opium. Phenazone and its allies probably affect the conduction of painful stimuli through the spinal cord or sensory tracts in the brain. Belladonna and atropine affect the sensory nerves, as probably does hyoscyamus. Stramonium, aconite and aconitine, veratrine, chloral hydrate and butyl-chloral hydrate, lupulus and lupulin, and gelsemium probably act on the encephalic centres.

USES.—As opium and morphine act upon all the nervous structures concerned in the production of pain, they may be used to relieve pain whatever its cause. Cannabis indica and bromide of potassium may be employed under the same circumstances as opium, but they have very much less power. Phenazone, acetanilide, phenacetin, and exalgin are powerful analgesics, and relieve pain without disturbing the brain. They are very useful in neuralgic pains, in headaches, and in pain depending on some affections of the spinal cord—for instance, to relieve the lightning-pains in locomotor ataxy. Chloral hydrate seems to relieve pain only by inducing sleep, and does not produce an anæsthetic effect unless it is given in dangerous doses. Butyl-chloral hydrate also induces sleep, but seems to have a special sedative action on the fifth nerve; as likewise has gelsemium—and hence both these agents are used in the treatment of facial neuralgia. As the action of belladonna is exerted chiefly on the peripheral ends of the sensory nerves, this remedy is usually applied directly to the painful part in the form of plaster, liniment, or ointment. Aconite, veratrine, cocaine, and opium are also used as local applications in several forms, for the relief of pain. The various anodynes may be administered not only by the mouth but by other channels, such as by inhalation, by enema or suppository, by hypodermic injection, or by endermic application.

Several therapeutic measures are employed as local anodynes, such as the application of dry or moist heat; cold; electricity; various forms of counter-irritation; acupuncture; and the abstraction of blood.

T. LAUDER BRUNTON.

ANOREXIA (ἀ, priv.; and ὄρεξις, appetite).—Want or deficiency of appetite. See **APETITE**.

ANOSMIA (ἀ, priv.; and ὀσμή, smell).—Loss of the sense of smell. See **NOSE**; and **OLFACTORY NERVE**.

ANTACIDS.—**DEFINITION.**—Medicines used to counteract acidity of the secretions.

ENUMERATION.—The antacids include Potash, Soda, Lithia, Ammonia, Lime, Magnesia and their Carbonates; as well as the salts which the alkalis form with vegetable acids, such as Acetates, Citrates, and Tartrates.

ACTION.—Antacids are divided into: (1) those which act *directly*, lessening acidity in the stomach; and (2) those which act *remotely*, diminishing acidity of the urine. The alkalis and alkaline earths and their carbonates, with the exception of ammonia, have both a direct and remote influence; for when swallowed they act on the stomach, and being absorbed from the intestinal canal, they are excreted by the kidneys, thus lessening the acidity of the urine. Ammonia and its carbonate are direct but not remote antacids; for, although they

neutralise acidity in the stomach, they are partly excreted in the form of urea, and do not diminish the acidity of the urine. The acetates, citrates, and tartrates of the alkalis and alkaline earths, on the other hand, are remote but not direct antacids. They have no antacid effect in the stomach, but undergo combustion in the blood, being converted into carbonates, in which form they are excreted in the urine, and diminish its acidity.

USES.—Excessive acidity of the contents of the stomach gives rise to acid eructations and heartburn. It may sometimes depend on the secretion of a too acid juice by the stomach, but is generally caused by the formation of acid from the decomposition of food when the process of digestion is slow and imperfect. Antacids are given after meals to lessen acidity in the stomach, and afford immediate relief to its attendant symptoms. They may prove even more efficacious by preventing acidity when given before meals (see **ALKALIS**). If the action of the bowels be regular, soda is preferable; but lime should be used if they are relaxed, and magnesia if there is a tendency to constipation. Remote antacids are given to lessen the acidity and irritating qualities of the urine in cystitis and gonorrhœa; and to prevent the deposition of uric acid gravel or calculus in gouty persons. For this purpose potash and lithia are preferable, as their urates are more soluble than those of the other bases. See **ALKALINITY**.

T. LAUDER BRUNTON.

ANTAGONISM.—This term is employed to express the fact that the physiological action of certain substances may be affected, even to the extent of neutralisation, by the presence in the body, at the same time, of other substances having an action of an opposite character. It is important to distinguish between *antidotal action* and *physiological antagonism*. By an *antidote* is meant a substance which so affects the chemical or physical characters of a poison as to prevent its having any injurious action on living animal tissues. Thus acids and alkalis neutralise each other, so as to form innocuous salts; tannin may render tartar emetic and many vegetable alkaloids insoluble; and the hydrated sesquioxide of iron may be used to precipitate arsenious acid. In these cases, the action is limited chiefly to the alimentary canal; and the object of administering the antidote is to form insoluble salts, or compounds which will be physiologically inert. But the *physiological antagonism* of certain substances is presumed to take place in the blood or in the tissues. When such a substance as strychnine, for example, is introduced into the alimentary canal, it is quickly absorbed, and carried by the blood throughout the body. It does not, so far as observation has discovered, influence all the tissues; but it so affects the spinal cord, and possibly the brain, as to give rise to severe tetanic convulsions, chiefly of a reflex character. This effect is, no doubt, due either to some interference in the nutritional changes between the blood and the tissues composing the nerve-centres, or to some specific action of the poison on the nerve-centres themselves. These changes, which are termed physiological, and on which the normal action of the nerve-centres depends, are probably of a molecular or chemical nature; and it is possible to conceive that they may be modified in different ways by different substances. Thus has arisen the

idea of physiological antagonism ; and experiment has shown that, within certain limits, which will no doubt vary in each case, such an antagonism is possible. Antagonism may be either local, affecting one organ, as is seen in the opposite effects upon the pupil of opium or morphine upon the one hand, and stramonium, hyoscyamus, or belladonna upon the other ; or it may extend apparently to more important organs or groups of organs, as in the case of the antagonism between strychnine and the hydrate of chloral. The most important investigations upon the subject of physiological antagonism are the following :—

(1) *Physostigmine and Atropine*, by T. R. Fraser—an inquiry which showed that the fatal effect of three and a half times the minimum fatal dose of physostigmine may be prevented by atropine. (2) *Atropine and Prussic Acid*, a research by Preyer—of a more doubtful character as regards the point to be proved, but still sufficient to show that, within certain limits not yet indicated, it is possible to prevent the fatal action of prussic acid by atropine. (3) *Atropine and Muscarine* (the active principle of *Agaricus muscarius*)—which were found by Schmiedeberg and Koppe to have entirely antagonistic actions on the ganglia of the heart—muscarine exciting the intracardiac inhibitory centres, and stopping the heart in diastole, while atropine has the contrary effect. (4) *Chloral and Strychnine*—an antagonism first pointed out by Oscar Liebreich, who showed that minute doses of strychnine might so rouse an animal from the effects of an overdose of chloral as even to save its life. (5) *Strychnine and Chloral*—with respect to which Hughes Bennett demonstrated the converse of the last-mentioned observation, namely, that in the rabbit a fatal dose of strychnine might be so antagonised by a dose of chloral as to save life. Other examples of antagonism are : chloroform and amyl-nitrite, cocaine and morphine, atropine and jaborandi, strychnine and hydrocyanic acid, thebaine and chloral, and digitalin and aconitine.

CONCLUSIONS.—It has unfortunately to be admitted that the practical results of the preceding researches have not been very encouraging. In all of these investigations it was quite apparent that the limits of physiological antagonism were very narrow. Three elements affect the chances of success in the way of saving life :—(1) the age and strength of the animal ; (2) the amount of the doses of the two active substances—so that if either the one or the other active substance be given slightly in excess, death will probably take place ; and (3) the time between the administration of the two active substances. If the stronger be introduced first, and be allowed to manifest distinctly its physiological action, it is almost impossible to counteract this by that of another substance ; but if the two substances be introduced simultaneously, or if the supposed antagonist to the more active substance be introduced first, the chances of success are much greater. It is apparent, therefore, that the facts relating to physiological antagonism at present known in science do not hold out much hope of good results from their application in practice ; but still the physiological facts are so definite as to indicate a precise mode of treatment. For example, no one acquainted with the investigations mentioned above would hesitate in attempting to relieve the tetanic spasms of a case of poisoning by strychnine by repeated doses of chloral hydrate, or by the

administration of chloroform. A practical result of such researches is that the principle of physiological antagonism may serve as a guide to the application of remedies in disease. Thus excessive secretion, say from mucous membranes or from salivary glands, may be modified or arrested by the use of sulphate of atropine, a striking experimental demonstration of which may be seen in the antagonism between bromal hydrate and sulphate of atropine in the rabbit.

JOHN G. MCKENDRICK.

ANTEFLEXION.—A bending forwards of any organ. The term is specially used in relation to the uterus, when this organ is bent forwards at the line of junction of its body and cervix. *See* UTERUS, Diseases of.

ANTEVERSION.—A displacement forwards of any organ. The term is particularly applied to a change of position of the uterus, in which this organ is bodily displaced in the pelvic cavity, so that the fundus is directed against the bladder, and the cervix towards the sacrum. *See* UTERUS, Diseases of.

ANTHELMINTICS (ἀντί, against ; and ἔλμινς, a worm).—**DEFINITION.**—Medicines which kill or expel intestinal worms.

ENUMERATION.—The principal anthelmintics are :—Oil of Male Fern ; Kamala ; Koussou ; Oil of Turpentine ; Pomegranate Root ; Santonica (Wormseed, so called), and its active principle, Santonin ; Areca ; Mucuna ; Rue ; and drastic purgatives. As purgatives only expel the worms, they are termed *vermifuges* ; while the other anthelmintics which kill the worms are called *vermicides*.

ACTION.—The oil of male fern, kamala, koussou, oil of turpentine, and bark of pomegranate root, act as poisons to tape-worms. Santonica and santonin remove round-worms, and also thread-worms. They were formerly supposed to kill the worms, but it would appear that they only weaken or intoxicate them, so that they are more readily expelled. Castor oil, jalap, scammony, and other purgatives do not kill the worms, but dislodge and expel them, by the increased peristaltic action which they occasion.

USES.—Drastic purgatives may be used for worms of any sort ; areca for both tape- and round-worms ; and the other agents for the worms on which they severally act as poisons. Vermicides are generally given after the patient has fasted for several hours, in order that, the intestines being empty, the drugs may act more readily on the worms. A purgative is usually given some hours afterwards, in order to expel the dead worms. As thread-worms chiefly inhabit the rectum, they are most effectually killed by enemata, which may consist of a strong infusion of quassia ; salt and water ; vinegar and water ; solution of the sulphate or perchloride of iron ; oil of turpentine ; castor oil ; decoction of aloes ; or infusion of senna. As abundance of mucus in the intestines forms a convenient nidus for the growth of worms, anything that diminishes this tends to prevent their occurrence, and for this purpose preparations of iron and bitter tonics are useful.

T. LAUDER BRUNTON.

ANTHRACOSIS (ἀνθραξ, a coal).—A deposit of black material in the tissues or organs. *See* PNEUMOCONIOSIS.

ANTHRAX (ἄνθραξ, a coal).—A synonym for carbuncle, and for malignant pustule. See CARBUNCLE, and PUSTULE, Malignant.

ANTIDOTE (ἀντί, against; and δίδωμι, I give).
DEFINITION.—An antidote is any remedy which, by its physical or its chemical effect upon a poison, or in both ways, is capable of preventing or counteracting the physiological effects of that substance (see ANTAGONISM). Sometimes, however, the term is used in a more comprehensive sense, so as to include the general treatment of a person affected by a particular poison. Thus, in poisoning by opium, the use of the stomach-pump, enforced exertion, chafing the limbs, and artificial respiration may be included in the general antidotal treatment.

MODES OF ACTION, AND APPLICATION.—Most antidotal substances form with the poison insoluble or innocuous compounds. Without attempting to give a complete list, the following are examples of the more common poisons and their respective antidotes:—(1) *arsenious acid*: solution of dialysed iron administered freely, or light magnesia; (2) *prussic acid*: newly precipitated oxide of iron with an alkaline carbonate; hypodermic injection of 2 to 4 m of liquor atropinæ sulphatis every half-hour; (3) *oxalic acid*: chalk, common whiting, or magnesia suspended in water; (4) *tartar emetic*: tannin, catechu, or other vegetable astringents; (5) *acetate of lead*: sulphate of magnesium, or the phosphates of sodium and magnesium; (6) *caustic potash*: dilute acetic acid, fixed oils, lemon-juice; (7) *corrosive sublimate*: albumen, white of egg, flour, or milk; (8) *mineral acids*: chalk, common whiting, plaster from the walls or ceiling, or carbonate of magnesium; (9) *chloride of zinc*: albumen, milk, or carbonate of sodium.

Vegetable poisons cannot thus be counteracted. If they have been taken in the form of seeds, leaves, or roots, the proper course is to remove them from the stomach or bowels as soon as possible by emetics and purgatives, and at the same time to sustain the flagging strength of the patient by the administration of stimulants. On the other hand, if the alkaloid has been taken, it is so soon absorbed that emetics and purgatives are of little avail, or may even be injurious. In these circumstances we must rely on the administration of the physiological antagonist of the poison (such as chloral hydrate in the case of strychnine-poisoning), and on supporting the strength of the patient. The following are the best antidotes to the vegetable poisons most frequently met with:—(1) *aconite root*: emetic of sulphate of zinc and stimulants; (2) *belladonna leaves, berries, or root*: emetic of sulphate of zinc, ammonia, stimulants, and after some time an active purgative; (3) *digitalis*: emetics, stimulants, and the maintenance of the recumbent position; (4) *hyoscyamus leaves*: emetics and stimulants; (5) *hydrochlorate or meconate of morphine, or any of the preparations of opium*: hypodermic injection of apomorphine; external stimulation by warmth, turpentine or camphor liniments; enforced exertion; artificial respiration, and small repeated doses of sulphate of atropine; (6) *chloral hydrate*: the same as for opium; (7) *strychnine or nux vomica*: animal charcoal suspended in water, repeated large doses of chloral hydrate, or chloroform; (8) *atropine*: stimulants, subcutaneous injection of caffeine, morphine, or pilocarpine, or of very minute doses of physostigmine; (9) *calabar bean*: atropine, or

stimulants, artificial respiration; (10) *mushrooms*: subcutaneous injection of 2 to 4 m of liquor atropinæ sulphatis, stimulants, castor oil; (11) *tobacco or nicotine*: stimulants, subcutaneous injection of minute doses of strychnine. See POISONS.

JOHN G. MCKENDRICK.

ANTIMONY, Poisoning by.—See TARTAR EMETIC, Poisoning by.

ANTIPERIODICS.—This term was applied to a group of medicines which were found to prevent or relieve the paroxysms of certain periodically recurring disorders, the nature of which was unknown.

The chief antiperiodics are:—Cinchona-bark and its alkaloids—Quinine, Cinchonine, Quinidine, and Cinchonidine; Salicin, Salicylic Acid and its salts; Eucalyptus globulus; and Arsenic. See MALARIA.

ANTI-PHLOGISTIC (ἀντί, against; and φλέγω, I burn).—A term for any method of treatment that is intended to counteract inflammation and its accompanying constitutional disturbance.

ANTIPYRETICS (ἀντί, against; and πυρετός, a fever).—**DEFINITION.**—Therapeutic agents which reduce the temperature in fever.

ENUMERATION.—The principal agents used as antipyretics are—Cold Baths, Cold Applications, Ice; Diaphoretics; Alcohol; Chloral Hydrate; Quinine; the Salicyl compounds; Phenazone (Antipyrine); Acetanilide (Antifebrin), Phenacetin, and allied bodies; Eucalyptol; Essential Oils; Aconite; Digitalis; Green Hellebore; Purgatives; and Venesection.

ACTION.—The temperature of the body may be reduced, either by increasing the abstraction of heat, or by lessening its production. The direct application of cold, by means of baths, affusion, or sponging, or by enveloping the body in sheets wrung out of cold water, is the most powerful and rapid means of abstracting heat. But the loss of heat which constantly occurs, even in health, by evaporation of the sweat, and the radiation and conduction of heat from the skin, may be increased by the use of diaphoretics, such as salts of potassium, preparations of antimony, or acetate of ammonium, or by such medicines as dilate the cutaneous vessels, so as to allow the heated blood to circulate freely through them, and to become cooled by the external media surrounding the skin. Alcohol, in the form either of wine or spirits, and chloral hydrate have an action of this sort, though alcohol also influences the production of heat. Alcohol, quinine, the salicyl-compounds, antipyrine (phenazone), acetanilide, phenacetin, pyrocin, and their allies, eucalyptol, and essential oils lessen the production of heat within the body, probably by diminishing oxidation of the tissues (see ALCOHOL). Aconite, digitalis, and green hellebore reduce the temperature, but their mode of action is not precisely ascertained.

USES.—Antipyretics act much more powerfully in reducing the temperature of the body in fever than they do in health. They may be used when the temperature has risen either from exposure to a high external temperature, as in thermic fever; in consequence of inflammation, as in pneumonia, pericarditis, or phthisis; or in specific fevers, as acute rheumatism, typhus, and scarlatina. The most rapid and powerful antipyretic remedies are cold baths; next probably come acetanilide, phenazone,

zone, phenacetin, large doses of salicylic acid or salicylate of sodium, and quinine. In acute rheumatism the salicylates are the most useful antipyretics. Quinine seems to act very efficiently in thermic fever when injected subcutaneously. See HYPODERMIC MEDICATION.

T. LAUDER BRUNTON.

ANTIPYRIN-HABIT.—See DRUG-HABITS.

ANTISEPTICS (*ἀντί*, against; and *σηπτικός*, from *σήπω*, I make putrid).—SYNON. : *Antiputrescents*.

DEFINITION.—An antiseptic is a chemical substance that retards or prevents the change in organic materials which is called putrefaction. Putrefaction is the chemical breakdown that occurs in non-living animal or vegetable matter which is brought about by the activity of bacteria and similar micro-organisms. It is usually characterised by the production of malodorous gases. The change is one of the numerous class of phenomena which are called by the general name 'fermentation,' a term originally employed in connection with the production of alcohol and carbon dioxide from sugar through the agency of the yeast-plant. The actual agents that cause this and similar changes are called ferments, and these are generally divided into two groups, the organised and the unorganised. The unorganised ferments or enzymes are chemical substances probably proteid-like in nature; they are shed out by animal or vegetable cells, and the chemical phenomena they produce are usually of the nature of hydrolysis; in some cases they appear to produce oxidation. Diastase, a product of germinating plant-cells, and pepsin, a product of the gastric epithelial cells, are familiar instances of these; the former produces the hydrolytic cleavage of starch into smaller molecules first of dextrin and then of sugar; the latter brings about the similar cleavage of proteid into smaller molecules first of proteose and then of peptone. The enzyme itself does not participate in the composition of the final products. How the organised ferments, like yeast-cells and bacteria, produce their respective decompositions has long been a matter of controversy; here again the actual chemical change is usually hydrolysis. Liebig in 1848 put forward the theory that the organisms that lie at the root of the change shed out what we now call enzymes, and that these chemical substances are the immediate causes of the chemical decomposition. In the case of the *Micrococcus ureæ*, and of the inverting action of yeast, this was proved to be the correct view by the actual separation of active enzymes from the organisms. It is now generally accepted as the correct explanation in all cases; and the experiments recently made by Buchner on yeast are quite conclusive; by crushing the yeast-cells he has succeeded in obtaining from them an enzyme which produces the alcoholic fermentation. The distinction between enzymes and organised ferments, though useful as indicating a difference of degree, has no real foundation as a difference in kind. In all cases the active agent is an enzyme or enzymes, and in all cases these are produced by the activity of living cells.

MODE OF ACTION.—It will be gathered from the preceding paragraph that there are obviously two ways in which an agency can counteract the activity of a micro-organism: (1) it may remove,

destroy, or inhibit the activity of the living cells or organisms; (2) it may remove, or neutralise the enzyme shed out by the organisms. Thus a removal of the organisms by efficient filtration will stop the production of the enzyme and so bring decomposition to a close. All ferments act best in the presence of moisture, and at a temperature somewhat above that of the body. But cold will stop fermentation of any kind, first because it prevents the activity of living cells, and secondly because it inhibits the activity of all chemical agents, including enzymes. It has, however, been shown that even the extremest cold does not kill the organisms in question. On the other hand, a high temperature stops all kinds of fermentation and does so permanently because it destroys the agents that produce the fermentation. Heat acts in one or both of two ways: (1) it kills the micro-organisms; (2) it destroys the enzymes which, as we have seen, are proteid-like in nature, and like most proteids they are coagulated by heat. In the case of the chemical antiseptics, our knowledge is as yet hardly exact enough to state definitely in which of the two ways they may act. Some doubtless act by destroying the protoplasm of the living agents, others by chemically neutralising or precipitating the enzymes, and others again doubtless act in both ways. As a general rule it may be stated that the living protoplasm is more readily affected than the more stable enzymes which they shed out; thus weak solutions of salicylic acid, and chloroform-water are sufficiently strong antiseptics to prevent putrefaction by putting a stop to the growth of micro-organisms, but they will nevertheless allow the action of enzymes to go on without serious impediment. This fact enables an investigator to determine in a mixed case what changes are produced by the one class, and which by the other class of ferments. It is nevertheless important to bear in mind that the difference is only one of degree, in view of the extensive use of antiseptics as preservatives of food. An antiseptic may be beneficial by hindering putrefaction, but it may also be harmful in retarding or even preventing those actions of enzymes which constitute the principal processes that occur during digestion.

ENUMERATION.—The principal antiseptics are the following:—Chlorine, Sulphur Dioxide, Nitric Oxide, Nitrogen Peroxide, Carbolic Acid, Creasote, Benzol, Sulphites, Hyposulphites, and Hypochlorites, Mercuric Chloride, Mercurio-Zinc Cyanide, and several other Mercurial compounds, Zinc Chloride, Arsenious, Chromic, Boric, Tannic, Benzoic, and Salicylic Acids, Borax, Potassium Permanganate, Naphthol, Resorcin, Formaldehyde, Alcohol, Iodoform, Quinine, Charcoal, Common Salt, and Nitre.

USES.—The uses to which antiseptics are applied are numerous; the following are the principal:—

1. As external applications to the body, as in the treatment of skin-diseases due to micro-organisms and similar fungoid growths, e.g. ringworm.

2. As applications to the mucous membranes of the orifices of the body and other readily exposed parts; for instance, in the cleansing of the mouth, teeth, throat, conjunctiva, nasal cavities, vagina, &c. The cleansing of the bladder, where inflammation has been set up by bacterial infection, and of the stomach in similar conditions, may also be included under the same head. The stomach normally

possesses a natural antiseptic in the free hydrochloric acid of the gastric juice.

3. In the treatment of surgical operations and open wounds, carbolic acid and mercurial compounds are the most frequently employed. It is this important branch of treatment which is especially associated with the name of Lister. *See* ANTISEPTIC TREATMENT.

4. In the internal treatment of diseases due to micro-organisms.—This branch of therapeutics is limited by the circumstance that substances which would kill micro-organisms are also inimical to the life of the tissues in which they grow. For instance, the treatment of phthisis by inhalations of such a substance as formalin-vapour has been all but relinquished because of its irritating action on the lungs.

5. As disinfectants. *See* DISINFECTION.

6. As preservatives of anatomical and similar museum-specimens. The preservation of dead bodies as in the process of embalming may also be here mentioned.

7. As preservatives of foods. The difficulty of transport and storage of food for large communities has led to a wholesale use of such substances as borax, boric acid, and formalin for commercial purposes. The practice is to be deprecated because these foreign admixtures are in a greater or less degree poisons to the human body as well as to micro-organisms. This is particularly the case in certain individuals who are abnormally susceptible to such substances. Thus boric acid and borax are often responsible for dyspeptic troubles, and will sometimes cause a skin-eruption like psoriasis. Of the two, borax is more soluble and is the more powerful antiseptic; it also hinders the action of enzymes in the alimentary canal; for instance a very small admixture with borax will completely prevent the curdling action of the rennin-ferment on milk. The use of formalin is still more to be deprecated. It has, even in dilute solutions, a hardening effect on proteid substances, rendering them less digestible, and also has a pronounced inhibitory effect on the digestive enzymes. It appears to be desirable that the practice of adding these and similar foreign substances to food-stuffs should be prohibited, and replaced by a general use of the methods of cold storage and transport.

W. D. HALLIBURTON.

ANTISEPTIC TREATMENT.—PRINCIPLES. The 'Antiseptic System' of treatment, introduced by Lister about 1867, has in great measure revolutionised modern surgery. It depends upon certain postulates, the appreciation and acceptance of which are essential to its successful application. They are:—

I. Septic processes in a wound, and the constitutional changes resulting from them, are caused by the development in it of minute living organisms.

II. These living organisms are (with some few exceptions) always introduced from outside the body, either by the hands or instruments of the operator, by floating particles of dust, by lotions or dressings, or in some similar way.

III. There are means at our disposal, such as the employment of certain germicidal agents, superheating, &c., by which the vitality of these organisms may be destroyed, and their entrance into a wound in an active state effectually prevented, or their influence combated if they be present.

APPLICATIONS.—In order to carry out an antiseptic operation it is necessary:—

1. To purify the skin of the region to be operated upon, for a considerable distance beyond the part which will be occupied by the wound.

2. To purify the hands of the operators, the instruments, sponges, and everything coming in contact with the wound; and to ensure against their defilement until the operation is completed.

3. To prevent any living septic organisms falling upon the wound during an operation, or to destroy their vitality before the dressing is put on.

4. To apply a dressing containing a store of some antiseptic material; and to change it before this material has been so much washed away by the discharges that a track is left along which septic organisms may be able to spread from the edge or surface of the dressing to the wound.

1. **Carbolic Acid** (*Phenol*).—This is occasionally used pure (liquefied by adding 6 to 10 per cent. of water to the crystals) to purify some already septic wounds; or a 1 in 20 watery solution is used for purifying the skin of the part to be operated upon, the hands and instruments of the operator, or in the spray, if that be ever employed. A 1 in 40 watery solution may be used for the sponges, or for bathing the wound during the operation and at the changing of the dressing. Carbolic-acid gauze is not so much used as formerly, but in some cases is still the best dressing at our disposal.

Carbolic acid has the advantages of being very diffusible, and of having an attraction for oily and greasy substances, and hence a superiority over mercurial salts for application to the skin; it does not blacken steel or silver instruments. It has the disadvantage of being irritating, and hence causing more effusion of serum than do the mercurial salts. Dressings containing carbolic acid must be kept in air-tight boxes.

2. **Mercurial Salts.**—(a) *Perchloride of mercury* is employed in solutions of 1 in 500, 1 in 2000, or still weaker. The 1 in 500 solution is used after the carbolic acid lotion, for purifying the skin, or for purifying an already septic wound; the 1 in 2000 solution may be used for the sponges, and for application to a wound during an operation and at the changing of the dressings. Mercuric chloride forms an insoluble albuminate when mixed with blood; hence the solutions are practically much weakened immediately they come into contact with a bleeding surface. Its advantage is its high germicidal quality; its disadvantages are that a 1 in 500 solution applied for some time to the skin causes vesication, and that if mixed with tap-water a large proportion of the salt is thrown down as insoluble chlorides. The latter difficulty is overcome by adding a small quantity of chloride of sodium; or by substituting for the simple corrosive sublimate the very soluble but decidedly irritating *sal alembroth*, a double chloride of ammonium and mercury, which for a while was very largely employed, but is now almost superseded. It is thus obvious that mercuric chloride alone is not a very suitable substance for the active principle of a permanent dressing.

(b) *Biniiodide of mercury* has many of the characteristics of the perchloride; but it does not form so copious a deposit of insoluble albuminates when added to blood. It is more irritating, but is largely used by some surgeons.

(c) *Cyanide of mercury*, in the form of a kind of double salt with *cyanide of zinc*, is the essential constituent of the latest antiseptic dressing (gauze and wool) introduced by Lister. This is very insoluble and very unirritating, but at the same time powerfully antiseptic, and at present promises to take the place of all other materials for a permanent antiseptic dressing.

3. **Boric Acid** is a mild and not very trustworthy antiseptic. It is, however, useful either in the form of boric lint, boric ointment, or boric lotion (a saturated watery solution) for application to superficial granulating surfaces. It is very unirritating.

4. **Salicylic Acid** is also a mild antiseptic. It is used in the form of salicylic wool, as an external dressing outside one containing some more potent material. In powder it is highly irritating to the nasal mucous membrane.

5. **Iodoform** is a powerful deodorant, with a most objectionable smell. Its antiseptic qualities are not great. It is used with great benefit in the form of crystals or powder for application to septic and tuberculous wounds. In the form of gauze it is used for plugging cavities and draining peritoneal incisions.

6. **Chloride of Zinc** in solution—gr. xl. to ʒj.—is invaluable as an application to surfaces which it is necessary to leave exposed to the air, such as those of the mouth or rectum, or for purifying septic cavities. It usually prevents septic changes for two or three days.

7. **Lysol** is very largely used in Germany, and has recently met with some favour in this country. It forms a turbid mixture with water and is very slightly irritating.

8. **Alcohol**, either undiluted or mixed with 15 per cent. of water, has been much extolled. Some surgeons now use no other antiseptic. See ALCOHOL.

This list might be multiplied almost indefinitely, and would have to include various coal-tar products, such as creoline, which is good for application to ulcers, naphthaline, &c., oil of eucalyptus, thymol, iodol, iodine, and countless others, but it is impossible to mention in detail more than those most commonly in use.

USE.—(1) The steps of a simple operation, *where the skin is unbroken*, such as the removal of a tumour or the opening of an abscess, will now be described as an illustration.

The skin of the part to be operated upon, and the hands of the operator, are, with the aid of a nail-brush, washed first with soap and water and usually, though this is of doubtful utility, with turpentine and ether; then with 1 in 20 carbolic acid solution, and then with 1 in 500 sublimate solution. The instruments should have been previously boiled or placed in a flat tray filled with carbolic-acid solution 1 in 20. Knives, scissors, and other cutting instruments may conveniently be sterilised by immersing them in alcohol. A large supply of sublimate-solution, 1 in 2000, or carbolic acid 1 to 40, should be prepared, in which the sponges are to be washed out, and the hands of the surgeon and his assistants dipped from time to time. It is convenient to surround the part to be operated upon with towels wrung out of one of the antiseptic solutions, so that instruments may not be thoughtlessly laid down upon an unpurified surface.

It was formerly the custom to irrigate the wound from time to time during the course of an operation with the weaker mercurial or carbolic-acid lotion.

This is not so frequently done now as formerly, and by many the plan has been quite abandoned. Others thoroughly wash the wound at the conclusion of the operation with boiled water or weak antiseptic solutions before applying the sutures, and during their insertion cover the greater part of the wound with swabs soaked in the same antiseptic solution.

Before changing an antiseptic dressing, scissors and forceps, and a piece of gauze or wool should be placed in a bowl of the antiseptic solution preferred, and the fingers of the surgeon should be similarly purified. As soon as the deep part of the dressing is removed, some of the solution should be dropped upon the wound by means of the gauze or wool, and the process should be frequently repeated until the deep part of the new dressing is in position. The spray has now been almost completely abandoned.

(2) *If the skin have been previously injured*, as in a compound fracture, or in abscesses which have burst, the strongest antiseptic solutions must be applied with great vigour to the exposed surface, their influence being often advantageously supplemented by the use of the sharp spoon or the nail-brush.

Besides these matters the acceptance of the antiseptic principle has effected marvels in the improvement of the hygiene of hospital wards and sick-rooms, by the dressing of septic wounds at frequent intervals with antiseptic and deodorising agents, by proper ventilation, by the methodical purification of bed-pans, catheters, urinals, and other utensils, and in a host of other ways too numerous to mention.

RICKMAN J. GODLEE.

ANTISPASMODICS (ἀντί, against; and σπᾶσμα, a spasm).—DEFINITION.—Medicines which prevent or allay spasm.

ENUMERATION.—Antispasmodics may be arranged in groups as follows:—Valerian, Valerianic Acid and its salts; Musk, Castor, Asafetida, Sumbul and Galbanum; Camphor, Brominated Camphor, Oil of Amber; Ammonia and its Carbonate; Alcohol, Ether, Acetic Ether, Chloroform, Nitrite of Amyl; Bromide of Potassium, Bromide of Ammonium; Conium, Lobelia, Opium, Gelsemium, Indian Hemp, Belladonna, Stramonium; and the Essential Oils. As adjuvants may be mentioned:—Cold Baths, moderate Exercise, Friction, Heat, and Moisture; and also Quinine, Arsenic, Zinc, and Silver.

ACTION.—Certain nerves and nerve-centres, when excited, produce contraction of voluntary or involuntary muscular fibres; other nerves and centres arrest movements; and by the combined action of these two systems the motions of the various contractile structures in the body are regulated, and subordinated to the requirements of the organism as a whole. Excessive contraction or spasm of one part of the body may therefore arise either from excessive action of the motor, or deficient action of the inhibitory centres. Spasm may affect the involuntary muscular fibres of the intestines—as in colic; of the vessels—as in some forms of headache, and in vaso-motor neuroses of the uterus and bladder; of the bronchi—as in spasmodic asthma; of single voluntary muscles, or groups of muscles—as in various forms of cramp; or it may involve the muscular system generally—as in tetanus, epilepsy, and hysteria. Antispasmodics may act by lessening the irritability of motor centres, as, for example, bromide of potassium and conium; or by stimulating those portions of the nervous system

which restrain and co-ordinate movements, as alcohol probably does. There are no direct experiments to show the action of antispasmodics on the inhibitory centres; but it seems probable that they have such an action, although it may not be confined to these parts alone. Thus small doses of alcohol and ether, which stimulate the nervous system generally, and usually increase motor activity, will restrain and co-ordinate excessive muscular action, as in colic, nervous agitation, trembling, and hysteria. It is at present impossible to localise the part of the nervous system affected by valerian, asafetida, and other drugs of this class. As spasms occur when the nervous system is deficient in power, nervine and general tonics, such as quinine, zinc, and iron, are often found to be useful adjuvants.

USES.—In such convulsive diseases as epilepsy, laryngismus stridulus, and infantile convulsions, bromide of potassium is the most powerful antispasmodic; in hysteria—valerian, asafetida, and the bromides; in chorea—arsenic, conium, copper, and zinc; in spasmodic asthma—lobelia and stramonium; in spasm of the blood-vessels—nitrite of amyl. In all spasmodic affections, cold baths or sponging, exposure to sunlight, moderate exercise, and a plain but nutritious diet should be employed; and late hours, a close atmosphere, exhausting emotions, or excessive bodily or mental work should be avoided.

T. LAUDER BRUNTON.

ANTITOXINS: Antitoxic and Antibacterial Serums.—**DEFINITION.**—Antitoxins are substances which are engendered under certain conditions in the bodies of living animals, and which are able to render inactive certain corresponding noxious substances.

Antitoxic Serums.—By treating animals with certain kinds of poisons (toxins), bodies which have the power of rendering these toxins inactive are produced: these are most abundant in the serum of the blood, and are called antitoxins. The scope of action of an antitoxin is limited and specific, in that it neutralises only the particular kind of toxin by means of which it was itself prepared. The kinds of poison which are capable of giving rise to antitoxins are:—(1) *certain bacterial toxins* not yet isolated chemically, such as those made by the *Bacillus pyocyaneus*, and the organisms of diphtheria and tetanus; (2) *certain vegetable poisons* of apparently proteid nature, such as abrin (from jequirity seeds), ricin (from castor oil seeds), and crotin (from croton oil seeds); (3) *snake-venom*, not yet separable from proteids by chemical means; (4) various kinds of *poisonous blood-serum*, one of the most highly toxic of which is that of the eel; (5) *rennet-ferment* and other bodies against which an 'anti'-body has also been prepared. From the above list it will be seen that the 'toxins' for which antitoxins can be made are closely associated with proteid molecules, if indeed they are not essentially proteid in constitution. Attempts to prepare antitoxins to alkaloids, such as morphine and strychnine, have all failed.

Antibacterial Serums.—Besides the bacterial toxins mentioned, many kinds of bacteria or their products may be used for the immunisation of animals, in which process 'anti-bodies' are formed. These, however, have no effect upon the essential toxin of the bacteria, although they exert a definite influence upon the bacteria themselves

(destructive or bacteriolytic, and inhibitory to growth), and are therefore called *antibacterial*. In the case of the organisms of cholera, typhoid fever, &c., only this form of serum has yet been obtained, while in that of the *Bacillus pyocyaneus* both antitoxic and antibacterial serums have been made by appropriate methods. Since antibacterial serums have no power of neutralising toxins, they are not of much use for therapeutic purposes.

Care must be taken not to confuse the liquids containing bacilli and their products, such as are used for giving protection against typhoid fever, plague, &c., with antitoxins. Even in medical literature the former are sometimes referred to erroneously as serums.

MODE OF PREPARATION.—The horse has been chosen as the most useful large animal for the production of antitoxin, one point of value being the low degree of toxicity of its serum for man. The general principle is to give the animal gradually increasing quantities of the desired toxin until it is able to endure a very considerable multiple of what would at first have been a certainly fatal dose. It is generally advisable to give doses sufficiently large to cause a considerable febrile disturbance. After a given interval has elapsed, several litres of blood are removed, with strict precautions against the occurrence of accidental bacterial contamination. The blood withdrawn is allowed to clot, and the serum is removed by sterilised apparatus as soon as it has separated; a small proportion of an antiseptic (such as phenol 0.5 per cent., or trikresol 0.4 per cent.) is then added, and the serum thus prepared is allowed to stand to facilitate the settlement and subsequent removal of any sediment which may tend to form. The resulting fluid is usually passed through germ-proof filters by means of pressure, and is then measured off into quantities suitable for single doses, which vary according to its potency; these are either placed in phials or sealed in tubes. All the manipulations are carried out with sterilised apparatus and strict aseptic precautions. To prevent the risk of distributing glanders or tuberculosis, the animal is given test-injections of mallein and tuberculin before the immunising treatment is carried out.

At the commencement it is advisable to give a foundation-immunity by injecting toxins which have been modified in certain ways (toxoids)—thus in the case of diphtheria the filtered culture may be heated to 60°–70° C., and in the case of tetanus the filtered culture is modified by the addition of carbon disulphide. Some authors recommend a course of injections of antitoxin before or during the toxin-treatment. The actual details of the treatment cannot be given here, since the production is largely for commercial purposes, and some of the methods employed are of the nature of trade-secrets. The mere feeding of animals on some poisons such as ricin and abrin may cause the appearance of antitoxins in the blood; but this does not occur with the toxin of tetanus or of diphtheria. The serum may be dried, generally upon flat plates, when it assumes the form of brittle scales; for use these are dissolved in recently boiled (sterile) water which has been allowed to cool.

Concentrated serums are also made by freezing and thawing repeatedly; by these procedures much water can be removed. Diphtheria-antitoxin, as at present used, seldom has less than 250 antitoxic units (*v. infra*) in each cubic centimetre; but it has been obtained with as many as 1,250 units; possibly

in the future still more potent preparations will be available.

The *antibacterial* class of serum is produced by injecting into an animal gradually increasing doses of whole cultures (i.e. bacteria with or without their products); the organisms may be previously killed by means of heat (60° C.) or antiseptics (e.g. chloroform); or they may be given in the living condition, but the latter method is not so safe for the worker.

Poisons giving rise to the formation of antitoxins.—The toxins present in the filtrates of cultures of the bacilli of diphtheria, tetanus, and botulism are the types of bodies which will lead to the production of antitoxins.

There are many pathogenic bacteria to the toxins of which living animals have no power of forming antitoxins. Thus highly toxic products are formed in cultures of the gonococcus and of the influenza-bacillus, but hitherto endeavours to get even a semblance of *antitoxin* for these have failed. In the case of the bacteria of plague, enteric fever, pneumonia, and erysipelas, no satisfactory toxin (that is, one which is at all comparable to those of diphtheria and tetanus) has yet been made; the same is true in the case of cholera. Attempts to produce *antibacterial serums* for the influenza-bacillus and the gonococcus have also failed, nor has any success been attained with the bacterium of fowl-cholera—a matter which is of interest in that this was one of the first microbes against which an active immunity was produced by Pasteur. For plague-bacilli, typhoid-bacilli, streptococci, and cholera-vibrios antibacterial serums can be prepared, which possess in some cases a high degree of potency (e.g. 1 cubic millimetre of good typhoid-serum will quite prevent the action of 10–12 fatal doses of highly virulent bacilli).

Since we do not know whether the proper toxins of these bacilli can be produced artificially, we cannot tell whether they possess the chemical constitution necessary for the production of antitoxins. Thus certain poisonous bodies such as alkaloids (morphine, strychnine, &c.) appear to be incapable of causing the formation of antitoxin, although a certain amount of individual tolerance may be attainable in some cases. The chemical nature of the poisons of the antitoxin-producing type (diphtheria, tetanus, and botulism) is not yet completely known, but the observations of Brieger and Boer tend to the conclusion that they are not proteids. It is, however, difficult, if not impossible, to isolate them entirely from what is possibly adherent proteid. The poisons of jequirity and ricinus &c. seeds are also either proteid or linked on to proteid molecules; the same is true of snake-venom.

NATURE OF ANTITOXIN.—Complete knowledge of the chemical nature of antitoxin is not yet forthcoming. Most observers are agreed that antitoxin has some connection with globulins, for precipitants of these bodies (saturation with neutral salts, &c.) carry down the antitoxin. Antitoxin probably has a large molecule, since it will not dialyse nor will it pass through a gelatine-choked filter (C. J. Martin); in these points it resembles globulin. It is closely allied in these and other characters to the active matter of antibacterial, antihæmal, and agglutinating serums.

It appears that when carbon dioxide is used to precipitate globulin the antitoxin is destroyed.

Heating up to temperatures below 60° C. for half

an hour has little if any effect upon the activity of most antitoxins, though prolonged periods of exposure cause some alteration; higher temperatures, as from 60° to 70° C., cause weakening or destruction according to the length and degree of the heating.

Strong *Light* has considerable destructive effect; but the diffused daylight in a room may cause no appreciable loss of activity after several months' exposure. On analysis it is found that actinic rays (blue and green) cause marked loss in a few months, while the more non-actinic rays (yellow and red) have very little effect in the same time.

Exposure to oxygen and air has a deleterious effect.

In well-filled bottles shielded from light but kept at ordinary room-temperature, antidiphtheric serum has been kept for several years without impairment; at body-temperature there appears to be some deterioration in much shorter time. Anti-venomous serum has been re-tested after a journey to and from the tropics and found unimpaired. Where the maintenance of exact degrees of potency is important, as in the case of standard serum for testing purposes, the serum is dried completely (eventually by the aid of anhydrous phosphoric acid), and kept *in vacuo*.

MODE OF ACTION OF ANTITOXIN.—An antitoxin only possesses the power of neutralising the corresponding toxin—thus diphtheria-antitoxin neutralises diphtheria-toxin but not tetanus-toxin, antiabrin does not affect ricin, and so on; in the case of antivenene there is perhaps some slight capacity for acting upon the venoms of different kinds of snakes, but this is limited in extent. Similar limitations are found in the case of antibacterial serums; thus there is no reason to think that an infection with staphylococci is benefited more by a dose of antistreptococcic serum than by the serum of an untreated animal.

Formerly it was thought that the action of antitoxin upon toxin only took place by aid of living tissue-elements; thus Metchnikoff introduced the term 'stimuline,' for he thought that the antitoxin stimulated the cells (especially leucocytes) to resist the toxin. Experimental evidence however is in favour of a *direct action* of a chemical nature between the two bodies, analogous to that of an acid and a base or to the formation of a double salt (Ehrlich). Toxin is neutralised by antitoxin only when the two substances come into contact with each other. Thus a quantity of antitoxin which will exactly neutralise a given amount of toxin when the two are mixed together and injected subcutaneously into an animal is not sufficient to nullify the action of the toxin when the two are given separately in different parts of the animal; in the latter case some 10 to 20 times the quantity of serum may be required to prevent the death of the animal, even though the one to which the mixed dose (containing only $\frac{1}{20}$ as much antitoxin) has been given may show no trace of illness or lesion. A given quantity of antitoxin will neutralise a given quantity of free toxin; from this it follows that the 'law of multiples' should hold good—that is, ten times the quantity of serum should neutralise ten times the quantity of toxin. This has been found to be the case with snake-venom; and even with the extremely complex filtrates of diphtheria-cultures the results obtained by giving thousandfold quantities (Cobbett) are surprisingly close to the theoretical effect which should be obtained. Divergences which occur

are thought to be due to the presence of other poisons which are not capable of union with antitoxin.

Earlier experimenters thought that in a neutral mixture the antitoxin and toxin remained side by side without mutual action, until they were introduced into a living animal; for if means were used to destroy the one but not the other, undestroyed antitoxin or toxin (according to circumstances) was found in the free condition. It has since been shown that this is only the case when sufficient time has not been allowed for the mutual interaction to take place. This action takes place more rapidly in concentrated than in dilute solutions, and in the warm than in the cold. Martin and Cherry made mixtures consisting of 1 c.c. of antivenomous serum with three times the fatal dose of the heat-resisting constituent of snake-venom; after leaving them in contact for various periods of time the mixtures were exposed to a temperature of 68° C. for ten minutes, whereby the antitoxin is entirely destroyed. An animal which was injected with the unheated mixture remained entirely without symptoms of poisoning; so also did one which received a dose kept thirty minutes before heating. Those which were given doses kept ten and fifteen minutes were severely ill but recovered; while those which received doses which had only been mixed two and five minutes before heating died with symptoms of poisoning. These experiments show that the serum was capable of completely neutralising the venom if sufficient time was allowed for their interaction. Similar effects have been obtained with other toxins and their respective serums.

It has been found (Ehrlich) that toxins may become modified and lose their toxicity, but that notwithstanding this they are still capable of action upon antitoxin. When different preparations of filtered diphtheria-cultures are tested against *exactly the same amount of antitoxin*, it is found that in a completely neutral physiological mixture in one case there may be only 20 toxic units, or there may be as many as 120. Again, in one and the same filtrate the toxicity diminishes on keeping, but the amount of antitoxin necessary to produce a physiologically neutral mixture remains constant, although the toxicity may have sunk to one-third of that originally present. Such facts are clearly explained by Ehrlich as showing that the filtrate contains the same number of equivalents which are capable of combining with the unit-quantity of antitoxin; and those molecules of toxin which have altered so as to lose their toxicity he calls *toxoids*.

ESTIMATION OF ANTITOXIC POWER OF SERUMS.—The method used for the measurement of diphtheria-antitoxin will form an example. In earlier days measurement was attempted by ascertaining what quantity of serum was necessary to prevent the action of a tenfold minimal fatal dose, but from what has just been said it will be clear that this method soon had to be given up. In dealing with questions involving the use of minimal fatal doses, it should be remembered that an animal is able to overcome a certain amount of poison up to the *maximal non-fatal dose*; by adding a small excess, merely a fraction of this maximal non-fatal dose, it becomes a fatal dose. A so-called tenfold fatal dose is the sum of ten components, each consisting of one maximal non-fatal dose with the addition of a fractional part of this sufficient to ensure death. It is only the nine and the fractional parts which have to be

neutralised to ensure the survival of the animal. The test is now made by comparing the action of the unknown serum with that of *standard serum*, which is kept dry *in vacuo* (see above), upon a toxic culture-filtrate which has been kept under toluol for some months; filtrates are not suitable for use until the loss of toxicity which occurs at first has ceased, and they have become stable. In order to avoid errors arising from the varying susceptibility of animals, young guinea-pigs of 250 to 280 grammes weight are used, and the criterion is death on the fourth day. The injection of mixed toxin and serum is made accurately in the subcutaneous tissue. The *antitoxin unit* was founded originally on the amount of antitoxin requisite to neutralise a tenfold fatal dose of the toxin; this quantity was taken as one-tenth of a unit; a unit therefore completely neutralised one hundred fatal doses. The *unit (antitoxin or immunisation unit)* is now measured by means of the standard dried serum, which is kept for comparison, and, roughly speaking, it is effective in nullifying about 100 fatal doses. Ehrlich's improved method is to measure out equal quantities of a solution of known strength made from standard dry serum, each of which shall contain *one unit of antitoxin*. Each dose is made up to 4 c.c. with saline solution, and after being mixed with measured quantities of stable toxin-solution is injected subcutaneously. By varying the quantities of toxin the amount is found which, when mixed with one unit of antitoxin, will just kill the animal upon the fourth day; this quantity of the toxin forms the test-dose. In order to ensure that a serum of unknown strength possesses not less than a given number of units, a corresponding quantity is mixed with the test-dose of toxin; should the animal die in the four days the serum does not possess the given strength, while if it die on the fifth or sixth day an allowance of five to ten per cent. is recommended, in order that the dose-strength claimed should not be too close to the border line.

MODE OF ACTION OF ANTI-BACTERIAL SERUM. It is not settled whether the essential action of anti-bacterial serum is direct (as the writer believes) or indirect, taking place through the agency of the living tissues. The frequent presence of the qualities of inhibiting the growth of, or of agglutinating, the bacillus in question cannot be discussed here. As might be expected when a living virus is given, there is no *law of multiples* (such as may be found with non-living toxin) when appropriate quantities of serum are added to the doses. Thus, with certain bacteria, such as those of typhoid fever or cholera, a minute quantity of active serum added to ten times the smallest certainly fatal dose may safely ward off all signs of illness, but, on the other hand, 200 times as much serum may not prevent death from thirty fatal doses. With certain other bacteria of which the virulence can be raised to so high a pitch that nine or ten germs may be enough to cause death (e.g. pneumococcus), 1 or 2 c.c. of efficient serum may be enough to secure the safety of the animal, although as many as 20,000 fatal doses are given with it (Pane). Anti-bacterial serums do not possess antitoxic power, although antitoxic serums always possess some, and it may be considerable, power of inhibiting the action of living bacteria. Some bacteria, such as the *Bacillus pyocyaneus*, will yield a toxin, whereby an antitoxic serum can be produced, while by treating an animal with the living culture an anti-bacterial serum can be pre-

pared. Wassermann has prepared these two forms of serum by treating a goat first for a period with toxin and later with living culture. At the end of the first period the antitoxic power was such that 0.5 c.c. would protect against four fatal doses of toxin, and 0.2 c.c. was effective against a fivefold fatal dose of living bacilli; at the end of the second period 0.005 c.c. protected against the fivefold living dose, but 1 c.c. had no retarding action on the fourfold fatal dose of toxin. From numerous experiments by different authors, it appears that anti-bacterial serums are of no value for therapeutical purposes in cases where the neutralisation of toxins is essential.

Antibacterial serums are of great value for the precise recognition of certain bacteria, such as those of cholera and typhoid fever. This is effected (Pfeiffer) by mixing about a tenfold fatal dose of a young culture (the fatal dose should not exceed about $\frac{1}{10}$ to $\frac{1}{2}$ milligramme of bacteria) with a few cubic millimetres of efficient serum. The mixture is injected into the peritoneal cavity of a guinea-pig, and samples of the peritoneal fluid are examined after the lapse of about $\frac{1}{2}$ to 1 hour; at the end of this time, if the bacterium is of the same sort as that with which the serum was prepared, all the bacteria will be found to have degenerated into coccus-like bodies; in a 'control' animal, unaltered bacteria will be seen if the culture has the requisite virulence for the test.

As in the case of antitoxins the action is specific in so far as it is not manifested when quite unrelated forms of bacteria are tested; when closely allied forms are tried, it is subject to slight limitations, but probably to a less extent than is the case with the clumping action which most of these serums also possess. For these tests the serums must be free from added antiseptics.

DISTRIBUTION AND SITE OF ORIGIN OF ANTITOXIC AND ANTIBACTERIAL BODIES.—It has been found that the specific substances appear earlier and in greater quantity in the blood-forming organs (lymphatic glands, spleen, bone-marrow) than in the blood after inoculation with cultures. As they disappear from these organs the quantity in the blood increases, and it is inferred from this that these organs are the sites of production of the specific substances. In the case of antitoxins the matter has not yet been followed out with the same thoroughness. In both cases it is fairly certain that the production does not occur directly from the constituents of the circulating blood. The tendency is for the substances to accumulate in the blood, and they may remain in recognisable quantity for long periods after cessation of the active or direct immunisation; on the other hand, when passive immunisation is carried out (i.e. by injecting antitoxin), they gradually disappear, especially when the serum injected is obtained from a different species of animal. The duration of the immunity is dependent partly also on the quantity of antitoxin given, the ordinary dose of 200 to 300 immunisation-units of diphtheria-antitoxin for prophylactic use remains effective for about three weeks. It should be remembered that there may be sufficient 'anti-substance' in the whole blood to protect against further attack although the amount in the small quantities which can be used for testing may not be determinable. The 'anti-substances' (antitoxic, antibacterial, and clumping) also pass out in the milk and some other secretions. They do not pass into the urine unless the kidney is damaged.

Bile of normal animals (probably certain of its protoid constituents) has considerable power of destroying many of the toxins, but we do not know that the action is that of true antitoxin. In certain cases, however, distinct indication of specific substances can be found in treated animals. Thus, the bile of an animal dead of rabies has preventive action when mixed with virulent rabies-material, while that of a normal animal has none. The substance of the central nervous system has a power of neutralising certain toxins (those of tetanus and botulism, but not that of diphtheria) when mixed with them. It is not at all certain that this action is really due to the presence of an antitoxin, for it appears that the effective agent is not soluble, while some other substances, like carmine, have a similar power.

THEORIES OF ORIGIN OF ANTITOXINS.—Various theories have been propounded to account for the nature of antitoxin; it has been suggested (1) that antitoxin is modified toxin (there is no evidence in support of this); (2) that it is secreted by the tissues solely in consequence of the presence of a given toxin; and (3) that it is present in normal animals, but that its production can be increased by the exhibition of the given toxin. The last theory is at present most in vogue and finds its explanation in the 'Side-chain' theory of Ehrlich. Ehrlich pictures the cell-protoplasm as possessing a number of different chemical molecular groups (side-chains) in its chemical constitution; each of these has a peculiar affinity for particular substances whereby the ordinary nutrition of the cell with various substances is effected. The protoplasm of any cell contains a number of different 'side-chain' molecules, each of which only picks up the kind of substance for which it has an affinity. The toxin-molecule is pictured as consisting of two parts, one of which (toxophore) is the essentially poisonous agent, while the other (haptophore) is the means whereby the toxin-molecule becomes fixed to an appropriate side-chain. If the toxophoric group is destroyed the haptophore (toxoid) may remain and no noxious action is produced; if the haptophore is attached to a liberated side-chain the combined group has no power of again fixing itself, and becomes harmless. The side-chain thus has the power of neutralising the given toxin-molecule. Antitoxin (for instance, in serum) simply consists, according to this theory, of side-chains which have become detached from cell-protoplasm. By means of treatment of an animal with a toxin the cells are stimulated to produce an unnecessarily large quantity of appropriate side-chains (overproduction): these are liberated into the blood-stream and the antitoxic power of the serum rises. On this theory it should be possible to produce antitoxin by the aid of the haptophoric group only (toxoids), and at any rate a foundation-immunity can be thus obtained. It is suggested that the injection of toxoids may be useful for therapeutical purposes by inducing the production of antitoxic side-chain molecules before the production of toxin by the infecting agent has become too great. According to this theory antitoxin is produced only in tissue-cells in which appropriate side-chains exist, and consequently different antitoxins may be formed in different tissues or organs. There is much yet to be done before this theory can pass beyond the realm of a 'working hypothesis,' but as such it has already led to advance in our knowledge.

THERAPEUTICAL ACTION OF ANTITOXINS.—It has already been stated that the maximum effect of antitoxin is obtained when the toxin and antitoxin are brought into contact before they are introduced into an animal. It is found that as the interval between the introduction of the toxin and the subsequent injection of the antitoxin is increased, so the amount of the latter which is necessary to banish symptoms also increases, but in a disproportionately rapid ratio, so that at last it is no longer possible to prevent death even when enormous multiples of the dose requisite to make a physiologically neutral mixture are given. The following instance (Dönitz) will serve as an illustration: a series of rabbits received twelve fatal doses of tetanus-toxin each at various intervals after 600 neutralising doses of antitoxin were given (in each case directly into the veins); when the interval was two and three-quarter hours no tetanic symptoms occurred; when four hours, only a few animals recovered, and all these had more or less severe tetanus; while, if an interval of five hours had elapsed, not a single animal survived. In another series two fatal doses were given, and it was found that a like amount of serum (as above) did not prevent death if there was an interval of twenty-four or more hours. It is therefore inferred that a combination of toxin with the tissues occurs; at first this is loose and can be upset by the antitoxin; later it becomes so firmly bound that antitoxin is unable to withdraw the toxin and neutralise it. In severe poisoning with tetanus at least one fatal dose is thus firmly bound out of reach of antitoxin in the short period of four to eight minutes in rabbits. Similar effects are produced with diphtheria-toxin, the fixation by the tissues not being quite so rapid. *It is of paramount importance to commence antitoxin treatment at the earliest possible moment in order to forestall the formation of an indissoluble union of the toxin with the tissue-elements. As time advances the value of antitoxin becomes rapidly less until it is no longer of avail.* It will be clear that for studying the statistics of serum-treatment a knowledge of the period of the disease at which it was administered is of great importance, as is shown by the following figures for diphtheria, taken from the Transactions of the Clinical Society of London (1898 Antitoxin Committee):—

	Treatment commenced on				
	1st day	2nd day	3rd day	4th day	5th day and after
Mortality, p.c.:					
No antitoxin	22·5	27·0	29·4	31·6	36·8
Antitoxin	10·0	10·8	15·0	20·0	25·5

The effect of antitoxin-treatment is very strikingly shown by the mortality in tracheotomy-cases; figures taken from the same source show a mortality of 16 per cent. when antitoxin was given within the first three days; 40·7 per cent. in cases treated within the next three days; and 64·7 per cent. in those treated on and after the seventh day; the general mortality without antitoxin varies between 60 and 75 per cent. It will be perceived that the mere presentation of the mortality of cases is not sufficiently exact to show the real effect of serum-treatment. In tetanus the cases which have been collected are less numerous; before antitoxin was introduced the death-rate was 96·7 per cent. (Poland) when the incubation-period was less than ten days, while in 48 cases treated with

antitoxin (Köhler) with the same limit of incubation the death-rate was 43·8 per cent.; and in twenty such cases where the treatment was begun in the first two days after the onset of symptoms it fell to 64·5 per cent. But since much improvement has recently been made in the potency of antitetanic serum there is ground for hope that future results may be still more successful.

MODES OF ADMINISTRATION.—Scrupulous attention to asepsis is absolutely necessary. The skin is cleansed with soap or solution of caustic alkali followed by some antiseptic, and the syringe is made absolutely sterile by boiling. The site of injection is usually under the loose skin in the flank. The dose required is regulated by units of potency, and not by the quantity injected. It is advisable to keep careful record of the time of administration, the exact dose used, and the effects produced in the patient.

Direct injection into the veins is not to be recommended on account of the enhanced effect of the toxic action of the serum of one species of animal upon another when introduced by this method. This toxic action has no relation to the antitoxin or to the nature of the toxin-treatment of the animal supplying the serum, for it is found to exist in the serum of normal animals of the same kind. Individuals among mankind as well as among the lower animals have a varying susceptibility to these toxic substances. A few cases have been recorded where human beings have succumbed or shown great symptoms of distress within a few minutes of an injection. An instance is given by Rauschenbusch in which five children and two servants received prophylactic injections from a single bottle of serum, the quantity amounting in each case to 0·8 c.c.; one of them, a child of ten, became suddenly ill—the symptoms being giddiness and faintness, vomiting and cutaneous irritation with urticarial wheals—within a few minutes of the injection. Eight hours later she still felt ill, but was nearly well on the following day. The symptoms and rapid onset noted in this case are exactly similar to those seen in susceptible guinea-pigs. The other four persons showed not the slightest ill effect. A few isolated cases are recorded where death has occurred apparently from a similar high susceptibility, but these are infinitesimal in proportion to the many thousands of persons to whom injections, often in large quantity, have been administered without any harm ensuing. Besides these immediate toxic effects the injection of the serum of horses may cause remote disturbances, which again do not appear to bear any very definite relation to the amount of serum injected. The most frequent of these effects are *rashes*—erythematous, scarlatiniform, urticarial, or more rarely morbilliform. They commonly appear in the second week after injection, and do not as a rule last more than about four days. Out of 9581 cases examined by the German Collective Investigation rashes occurred in 678 instances: the statistics collected by the Clinical Society of London give as many as 33 per cent., but only 633 cases of injections were investigated. Joint-pains with or without swelling may also occur. The temperature may be raised, but there is no evidence of a deleterious action on the kidneys. Similar effects may be produced by any of the serums (antitetanic, antistreptococcic, &c.), for they are dependent upon the fact that it is horse-serum, which is about the least toxic of animal serums for man. The entire elimination of the

noxious bodies and the preparation of a pure antitoxin are problems for future investigation.

Absorption of Antitoxin from Mucous Surfaces.—Experimentally antitoxin is not absorbed when given by mouth or rectum; 1000 times the dose which will neutralise and stay an intoxication when given under the skin has not the slightest effect upon the course of the poisoning when given by mouth or rectum. This mode of administration in acute conditions is merely to be regarded as a waste of valuable serum. It appears however that some of the immunity which is transmitted to suckling animals from the mother is acquired through the milk. Thus Ehrlich showed that normal sucklings when nurtured by an immunised foster-mother acquired some degree of immunity, while the milk of the foster-mother was proved to contain antitoxin. It is possible that long-continued administration by the mouth may lead in adults to a sufficient absorption of immunising substances for protective purposes, but this is of no value for the treatment of disease where time is important.

Finally a few notes of the serums which are in use may be appended:—

Diphtheria. Nature—antitoxic. Prepared by treatment with filtered germ-free cultures. For therapeutic use 1000 to 6000 units or more are given and repeated if necessary. Some recent observations give promise of reduction in frequency of the occurrence of paralysis by continuing the injections after immediate danger has disappeared. For prophylaxis 200 to 300 units are given; the protection lasts about three weeks.

Tetanus. Nature—antitoxic. Prepared by treatment with modified (toxoid) and active germ-free toxins. Immediate effects of the serum are not probable, since symptoms are probably due to toxin which has been fixed many hours before. Serum should be persevered with, if used, on account of the capacity of the tissues for fixing the toxin. Intracerebral injections may be given, following Roux and Borrel's experiments. (Rambaud collected sixteen cases with seven recoveries.) Prophylactic injections have been given in infected puerperal wards and to horses with satisfactory (negative) results.

Streptococcus. Nature—antibacterial. Prepared by treatment with cultures. Much difference of opinion exists as to its value both in the laboratory and at the bedside. The probable explanation is found in the observation that there are several varieties of streptococci, each of which is unaffected by serum prepared by means of any of the other varieties. Many clinical reports of apparent good effects are vitiated by want of demonstration that the condition of the patient was due to streptococci and by the impossibility of eliminating the possible effects of other methods of treatment (uterine douches, &c.).

Pneumonia. Probably more antibacterial than antitoxic. Prepared by treatment with cultures. In the laboratory the serum is highly effective; moreover its action on varieties is not so limited as is the case with the streptococci. Not yet sufficiently tried on man.

Plague. Nature—doubtful; has some slight antitoxic action. Prepared by treatment with living, dead, or filtered cultures. Good results have been obtained in laboratory experiments; it probably has some value for treating man. It has been used for prophylaxis in man, but is not to be confused with

Haffkine's preventive material, which consists of killed bacilli.

Typhoid and Cholera. Nature—antibacterial. Prepared as last. In the laboratory they are highly effective for preventing infection, but of no curative value for highly acute infections. The effects produced on enteric fever and cholera respectively in man are not very well marked, but they are probably not entirely without some, if light, efficacy. They have been used for prophylaxis, but are not to be confused with Wright's and Haffkine's preventive treatment with killed bacilli.

Snake-venom. Nature—antitoxic. Prepared by treatment with a mixture of venoms (chiefly that of the cobra) heated to 80° C. It has not the effect upon all snake-venoms which was claimed by Calmette. Trials on man are full of promise, but ordinary precautions should not be discarded.

Jequirity. Nature—antitoxic. Prepared by treatment with the poison of abrus seeds. It is very effective in laboratory experiments. Calmette suggests its use for controlling the course of jequirity-ophthalmia in ophthalmic practice.

Tubercle. Nature—doubtful. Prepared by treatment with tuberculin or other preparations obtained from tubercle-bacilli. Its practical value is not yet established.

Further a number of other materials—bacterial (staphylococci) and chemical (ricin)—have been made to yield 'anti-serums,' but these are at present only of theoretical and experimental importance. Other preparations, 'antisyphilitic,' 'anticancerous' serums, &c., are at present little better than chimerical.

HERBERT E. DURHAM.

ANTRUM, Diseases of.—See NOSE, Diseases of.

ANURIA.—Absence of urination, whether from suppression or retention of urine. See MICTURITION, Disorders of; and URINE, Suppression of.

ANUS, Diseases of.—The principal affections of this part are:—1. Congenital Abnormalities; 2. Epithelioma; 3. Irritable Sphincter Ani; 4. Irritable Ulcer; 5. Prolapsus; 6. Pruritus; and 7. Tumours and Excrescences. For Fistula in Ano see RECTUM, Diseases of.

1. Congenital Abnormalities (atresia) may be classed as follows:—1. Imperforate anus, without deficiency of the rectum. 2. Imperforate anus, the rectum being partially or wholly deficient. 3. Anus opening into a *cul-de-sac*, the rectum being partially deficient. 4. Imperforate anus in the male, the rectum being partially deficient, and communicating with the urethra or neck of the bladder. 5. Imperforate anus in the female, the rectum being partially deficient, and communicating with the vagina or uterus. 6. Imperforate anus, the rectum being partially deficient and opening externally in an abnormal situation by a narrow outlet. 7. Narrowness of the anus. These imperfections can be remedied, if at all, only by operation.

2. Epithelioma.—The anus, like other parts where a junction takes place between the skin and mucous membrane, is liable to epithelioma. It is easily recognised by the ordinary characters of the sore. Warty growths and flaps of skin at this part are subject to this form of degeneration. The treatment applicable to this disease is to remove the growth by excision.

3. Irritable Sphincter Ani.—In this complaint the anus is strongly contracted and drawn in by the action of the sphincter. Any attempt to examine the part produces spasm, and the finger passed through it is tightly grasped as if girt by a cord. In old-standing cases the muscle becomes hypertrophied, and forms a mass encircling the finger like a thick unyielding ring. This state is the source of serious trouble in defecation, owing to the expulsive power of the bowel being insufficient to overcome the impediment caused by the muscle to the passage of the feces. Irritability of the sphincter, independent of fissure or ulcer, occurs generally in hysterical females, and is relieved by mild laxatives, the local application of cocaine or an opiate ointment, and the occasional passage of a bougie coated with a sedative ointment.

4. Irritable Ulcer.—This is a small superficial sore, situated just within the circle of the sphincter, usually at the back part, commonly known as *fissure*, from its appearance in the contracted state of the part. The feces passing over the sore excite spasm of the muscle, and cause a sharp burning pain which lasts for two or three hours. The distress often does not come on till an interval of ten minutes or more has elapsed after defecation. The pain is sometimes so acute that patients resist an action of the bowels, and allow them to become costive. The irritable ulcer occurs usually in middle life, and is more frequent in women than in men. It seldom gets well under the influence of local applications, but an incision through the centre of the sore sets the muscle at rest, and allows the part to heal. Forcible dilatation of the sphincter with the fingers under an anæsthetic is a very effective mode of treatment. When the suffering is moderate, a cure may be attempted by giving a laxative to ensure soft evacuations, by enjoining rest in the recumbent position, and by the application of an ointment containing morphine, belladonna, or cocaine.

5. Prolapsus.—See RECTUM, Diseases of.

6. Pruritus.—Itching, though a common symptom in disorders of the lower bowel, may occur as a distinct affection, a neurosis liable to paroxysms. It is caused by worms in the rectum, and by congestion of the hæmorrhoidal veins. Patients suffer more after taking stimulating drinks and when heated in bed. The itching is extremely teasing and annoying, especially at night, keeping the sufferer awake for hours. Friction aggravates the mischief, excoriates the skin at the margin of the anus, and causes it to become dry, harsh, and leathery. As regards treatment, stimulants and condiments are to be avoided. The bowels should be regulated, and the part should be washed with soap and water after each evacuation. Every effort should be made to avoid friction. Cotton-wool should be used after defecation in the place of paper. A piece of cotton-wool soaked in oxide of zinc lotion may be kept applied to the anus, or the part may be smeared with some mercurial ointment, such as one of calomel (5j) and vaseline (3j). Lotions of cocaine and glycerine, of borax and morphine, or of carbolic acid are often efficacious. In weak persons quinine and arsenic administered internally help the cure.

7. Tumours and Excrescences.—Besides the flaps and folds of integument consequent on external piles, tumours of a *fibrous* texture sometimes form in the subcutaneous areolar tissue, which as they increase become pedunculated. They are usually small in size, lobulated, and have a firm feel.

These growths may be easily and safely removed by excision. *Papillomata* are liable to be developed around the anus, and sometimes grow so abundantly as to constitute a large cauliflower-like excrescence. They then form projecting processes of various sizes, densely grouped together, with their summits isolated, expanded, and elevated on narrow peduncles. They give rise to a thin, offensive discharge. They probably originate in want of cleanliness. In some persons there is so strong a disposition to the formation of warts, that it is difficult to prevent their growth. They require to be removed by excision, the quickest and most effectual mode of treatment. Flattened growths from the skin, commonly called *mucous tubercles*, a secondary result of syphilis, are liable to occur around the anus. They yield readily to the local application of mercury and general specific treatment. See MUCOUS TUBERCLES.

FREDERICK TREVES.

AORTA, Aneurysm of.—See ABDOMINAL ANEURYSM; and THORACIC ANEURYSM.

AORTA, Congenital Stenosis of.—The commonest seat of this condition is the point of entrance of the ductus arteriosus. There may be complete occlusion, or varying degrees of narrowing of the orifice. The proximal part of the aorta becomes greatly dilated, and the left ventricle hypertrophied. The circulation is carried on by the development of the collateral circulation through the transverse cervical, internal mammary, epigastric, and intercostal arteries, which may become so dilated as to simulate aneurysm, even to the extent of eroding a rib. The most important symptoms are dyspnoea on exertion, cough, hæmoptysis, and cardiac murmurs of various kinds. The condition usually shortens life greatly, but some cases have reached the age of fifty. Death is caused by rupture either of the aorta or of some of the collateral vessels, acute endocarditis, endarteritis, failure of the dilated heart, acute pulmonary complication, or exhaustion.

The aorta may be stenosed throughout and its walls thin, in the condition termed Virchow's Chlorosis (see CHLOROSIS). The aortic orifice may be so small as to scarcely admit the little finger. This hypoplasia, as it really is, is almost invariably associated with a hypoplasia of the generative organs and not seldom with interstitial nephritis. See HEART, Malformation of.

ALEXANDER BRUCE.

AORTA, Dilatation of.—In cases of dilatation of the aorta, there is a more or less uniform enlargement of the vessel affecting most frequently the ascending arch. When it extends into the transverse arch it usually stops short at the origin of the subclavian artery. There is no sharp line of delimitation between simple dilatation and aneurysm. The condition in question is caused by exhaustion of the resisting powers of the wall, and is most frequently associated with sclerosis of the aorta, or with the general rise of blood-pressure which exists in chronic Bright's disease, or in the diffuse sclerosis of smaller arteries. It may also be due to hypertrophy of the heart, whether secondary to any of the above conditions, to aortic valvular disease, or to persistent acceleration of its own action.

Dilatation of the aorta may occur without pre-existing disease of the artery, and, in this case, it

may simply be due to exhaustion, either temporary or permanent, of the elastic fibres of the middle coat. When the dilatation is sufficiently great to produce any physical signs, there is dullness on percussion over the upper part of the sternum, and perhaps also for some small distance to the right of it. There may be palpable pulsation in the episternal notch. In the aortic area a systolic murmur may be heard, while the second sound is generally accentuated or has a booming character. There may be breathlessness, and pain behind the sternum, aggravated on exertion. The dilatation may become lessened or even disappear under rest and other appropriate treatment, or it may remain stationary, or if untreated may even increase so as to deserve the name of aneurysm.

The treatment varies according to the cause of the condition. Rest is required in all cases, and the treatment by diet and drugs will be that of the existing disease, whether Bright's disease, anæmia, vascular excitement, or arterio-sclerosis.

ALEXANDER BRUCE.

AORTIC VALVES, Diseases of.—See HEART, Valves and Orifices, Diseases of.

APERIENTS (*aperio*, I open).—Medicines which produce a gentle action of the bowels. See PURGATIVES.

APHAGIA (*ἀ*, priv. ; and *φάγω*, I eat).—Inability to swallow. See DEGLUTITION, Disorders of.

APHASIA (*ἀ*, priv. ; and *φῆμι* or *φάω*, I speak). SYNON. : Aphemia ; Alalia ; Fr. *Aphasie* ; Ger. *Sprachlosigkeit*.

DESCRIPTION.—Aphasia is the name given to a defect of speech from cerebral disease. The patient is found to be unable to utter any proposition, though his occasional distinct pronunciation of some one or two words shows that his speechless condition is not due to a mere difficulty in the more mechanical act of articulation. Moreover, the patient's intelligent manner and gestures may plainly show that he understands what is said, and is capable of thinking, even though he is quite unable to give expression to his thoughts. This kind of powerlessness as regards speech is most frequently encountered in persons suffering from right hemiplegia, though it is occasionally met with in those who are paralysed on the left side, and at other times in persons who are not hemiplegic at all.

The aphasic condition is not always, as it ought to be, clearly distinguished from that of another group of cases which may be encountered in association with some amount of hemiplegia or independently, and to which the name *amnesia* is given. The essence of this latter defect lies in the fact that the patient's speech is defective because of his inability to recall the proper words for the expression of his thoughts or wishes ; and he very frequently substitutes wrong words or names in the place of those he wishes to employ, as when speaking of his 'hat,' he calls it a 'brush,' or when seeking a 'pen,' he asks for a 'knife.' In a bad case of this kind the patient may be quite unable to arrange words into a sentence capable of conveying a definite meaning, so that his speech is rendered unintelligible. Where this species of defect exists there seems to be either a defective or an inco-ordinate action of one or other of those higher cerebral centres whose function it is to translate

thought into the corresponding motor acts of speech, so that we get hesitation in the utterance of right words, or the substitution occasionally of entirely wrong words or even of a meaningless set of sounds. These amnesic or inco-ordinate defects were not at first recognised as being distinct in nature from those of an aphasic type, in which there is rather a loss than a misdirection of power in one or other of the higher centres whence the incitation to the motor acts of speech proceeds. The two kinds of defects, indeed, not infrequently co-exist to some extent in the same individual.

When occurring in association with hemiplegia, aphasia varies much in intensity according to the degree of general mental impairment with which it may be combined. During the first week or ten days after the onset of such an attack the special defect may be scarcely recognisable, owing to the masking influence of the general mental impairment. But after a time such a patient may regain a considerable amount of general mental power. He may be left more or less hemiplegic, and may also present the aphasic defect to a marked degree. He readily comprehends everything that is said to him, and can often understand what he reads.

But at other times, though able fully to understand when read to, he does not seem to understand when he himself attempts to read—a disability now often spoken of as 'word-blindness.' He can, perhaps, play draughts or chess well, and by means of gestures and pantomime can make his wants and most of his wishes fairly well understood by those accustomed to interpret them. Yet he may only be able to articulate some one or two words, or else combinations of mere unmeaning sounds, such as 'poi, boi, bah,' 'sapon.' On rare occasions, under the influence of strong emotion, the patient may blurt out some simple expletive or short phrase, such as 'oh dear !' He cannot be made to repeat a word, or even the simplest vowel-sound, which he has just heard uttered. In a few cases the patient has seemed unable to understand what is said, as though from some difficulty in realising the meaning of words. Words may have to be uttered very slowly and repeated several times to such a patient, and even then they may fail to convey their meaning—a defect known as 'word-deafness.' Yet the language of gesture, appealing as it does to the sense of sight, may be at once understood.

The patient's power of writing is necessarily interfered with when aphasia, as is so often the case, co-exists with right hemiplegia. Many such patients, however, learn to write with the left hand to a variable extent. The variations as regards the power of writing are, in fact, almost as marked as the variations in power of speaking, though these two classes of defects by no means run parallel with one another in the same individual. The writer has known patients who were quite unable to express themselves in spoken words, or even utter a single articulate sound, write a good letter with no, or very few, mistakes (*aphemia*) ; on the other hand, the performance of an aphasic patient, without a copy before him, may be, and most commonly is, limited to writing his own name. At other times the patient shows an amnesic defect in writing, and writes much as an amnesic patient speaks. Still more rarely it is found that an aphasic patient is, though not from want of manual power, unable to write even a single letter (*agraphia*)—in attempting to do so he makes mere unmeaning strokes.

The disability spoken of above as 'aphemia' may be complete, and then the patient is absolutely speechless. But incomplete aphemia or aphemic defects exist in many degrees of completeness. These terms are now often applied to defects in the power of articulation—the mere mechanical part of speech—in which difficulties exist such as go with diseases in the bulb or the pons, or the slighter defects in utterance, constituting mere 'thickness of speech.' This kind of defect differs from (and may be easily distinguished from) aphasia, seeing that the subjects of it will always attempt at once to utter any word or simple sound, when they are bidden to do so. The term 'anarthria' has been used by some writers for what would be called bad aphemic defects—such, for instance, as are met with in bulbar diseases, where the articulatory motor centres are themselves the seat of lesion.

Aphasia occasionally supervenes, independently of paralysis or convulsions, in individuals who have been subjected to great excitement or prolonged overwork, when it may be due, perhaps, to mere functional derangements. In other cases it presents itself as a temporary condition, lasting only for a few hours or a few days, in a patient who has just had an attack of right-sided unilateral convulsions; or, lastly, as has already been indicated, it occurs in conjunction with a right-sided hemiplegia produced either by brain-softening or by cerebral hæmorrhage. Cases belonging to the latter category vary very much amongst themselves as regards the degree of co-existing hemiplegia. If the third left convolution alone is damaged by softening, the hemiplegic condition may be absent, or transient and incomplete—never, perhaps, affecting the leg appreciably. This condition is often induced by a small hæmorrhage, or by a patch of softening produced by an embolism of that branch of the middle cerebral artery which supplies the third frontal convolution; but where the hæmorrhage is larger, or where the main trunk of the middle cerebral artery is obliterated, either by an embolus or a thrombus, the aphasia, or aphasia with amnesia, is combined with much graver and more persistent hemiplegic symptoms.

PATHOLOGY.—The modern concentration of attention upon these defects of speech was started by the enunciation of Broca's views as to the dependence of the aphasic defects upon lesions in or about the third left frontal convolution. All pathologists are now agreed as to the fact that such lesions are much more prone to give rise to aphasic symptoms than are corresponding lesions on the right side of the brain. It is commonly believed, moreover, that *amnesic* defects of speech are most commonly produced by superficial lesions of the left hemisphere, though by lesions situated farther back—that is, in the neighbourhood of the posterior extremity of the Sylvian fissure.

The third left frontal convolution is not now supposed, as Broca put it, to be the seat of any 'faculty of language,' though the anatomical investigations of Meynert and of Broadbent have shown that its relations with other convolutions are exceptionally complex. Whether certain higher 'motor' centres for speech are situated in this part of the brain, as is commonly believed, or whether such centres are rather, as the writer believes, kinæsthetic centres, it must at least be conceded that this convolution is intimately concerned with the physical expression given to thought in articulate speech.

We know that the left hemisphere is the one from which the volitional incitations proceed in the case of written language, and it is presumed that the same half of the brain also takes the lead in the production of articulate speech. It is, therefore, a point of much interest when we find that, in some of the exceptional cases in which aphasia has occurred in association with lesions on the right side of the brain and left hemiplegia, the individuals had been left-handed during life. Some of the other exceptional cases, however, have not admitted of this interpretation.

Different kinds of Word-memories.—For the proper understanding of these different defects of speech and writing many details are essential. There are four different kinds of word-memory—three essentially different types, but one of them existing in two forms, so as to make four varieties in all. These varieties of verbal memory are as follows: (1) Auditory memory—the memory of the *sounds* of words, that is, of the auditory impressions representative of different words; (2) Visual memory—the memory of the *visual appearances* (printed or written) of words, that is, of the visual impressions corresponding with different words; (3) Kinæsthetic memory—(a) the memory of the different groups of sensory impressions resulting from the movements of the vocal organs during the utterance of words (impressions from muscles, mucous membranes, and skin)—these the writer has proposed to speak of as '*glossokinæsthetic*' impressions; (b) the memory of the different groups of sensory impressions emanating from the muscles, joints, and skin during the act of writing individual letters and words—these the writer has proposed to speak of as '*cheirokinæsthetic*' impressions.

The organic seat of each of these different kinds of word-memory is in relation with its own set of afferent fibres; and the several centres are also connected with one another by commissural fibres, so that the recollection of a word in one or other of these modes doubtless involves some amount of simultaneously revived activity in one or two of the other word-centres. The relative intensity (in the process of recollection of words for ordinary speech) of the memorial revival in each of these centres is probably subject to more or less marked variation in different individuals. In the majority of persons the revival of words in the auditory centre is the most potential process, and that of which such persons are most conscious. In a smaller percentage of persons the revival of words in the visual centre seems to be the most essential process. While in a very much smaller percentage it seems possible that revival in the kinæsthetic speech-centre may be the most conscious process occurring during the recollection of words.

Owing to the fact of the existence of this multiple memory of words, it happens that loss of speech by no means always, or often, implies the loss of the memory of words. Many a patient who cannot speak can yet revive his auditory or his visual memory of words, so as more or less perfectly to understand what he hears, or what he sees (in print or writing), or perhaps what he both hears and sees.

It is also important to bear in mind that for ordinary persons (that is, those who are neither congenitally blind nor congenitally deaf) the four memories of words are mainly called into play in definite couples, namely, the auditory and the glossokinæsthetic revivals taking place in ordinary speech;

and the visual and the cheiro-kinæsthetic revivals taking place during ordinary writing. So that in expressing oneself in spoken words the memories of such words are first principally revived in the auditory centre, and then the nerve-units thus called into activity rouse in immediate succession the corresponding glosso-kinæsthetic elements before the pronunciation of the word can be effected through the aid of the motor centres in the medulla. Similarly, when expressing our thoughts by writing, though the memories of words are probably first revived in the auditory word-centre, like memories are almost simultaneously revived (through the intervention of the audito-visual commissure) in related parts of the visual word-centre; and from this region stimuli must pass through corresponding cheiro-kinæsthetic elements, before the actual writing of the word can be effected through the instrumentality of motor centres in the cervical region of the cord.

There can be no doubt that the functional association existing between the auditory and the glosso-kinæsthetic centres is of the closest kind. But the bond of association between the auditory and the visual centres is no less intimate. These latter centres are often necessarily called into activity in immediately successive units of time. This happens, for instance, in two such common processes as reading aloud and writing from dictation.

In reading aloud the primarily excited visual word-centre must arouse (through the visuo-auditory word-commissure) related parts of the auditory word-centre, since this is the part which ordinarily calls the glosso-kinæsthetic centre into activity, and from it properly co-ordinated incitations issue to call into play the motor-centres in the medulla.

Again, in writing from dictation, the sounds of words reach the auditory word-centre, and the activity thus aroused becomes transmitted (through the audito-visual commissure) to related parts of the visual word-centre, this being the part which usually arouses the cheiro-kinæsthetic centre into activity for the production of writing movements.

In deaf-mutes, in the congenitally blind, and also in certain cases of disease, as it would seem, the relations between the centres are rather those indicated by the dotted lines (*f*, *e*) in fig. 1.

Concerning the Localisation of the different Word-centres.—Looking to the extremely important part that 'words,' either spoken, written, or printed, play in our intellectual life, and to the manner in which they are interwoven with all our thought-processes, it becomes highly probable that most important sections of the auditory and visual perceptive centres are devoted to the reception (and consequently to the revival in thought) of impressions of words; so that, for convenience of reference, we may speak of these sections as auditory and visual 'word-centres' respectively. Similarly, there must be kinæsthetic word-centres of two kinds—the one in relation with speech movements, and the other in relation with writing movements. It is possible that the parts of the general auditory and visual centres which are in relation with word-impressions may be as distinctly defined as are the analogous parts of the general kinæsthetic centres that are in relation with speech movements. Certain it is that there are some varieties of amnesia in which the part of the visual centre in relation with words seems to be specially at fault (causing 'word-blindness'); just as there are other cases in which the

part of the auditory centre in relation with words is either wholly or partially inactive (causing 'word-deafness'), in each case without evident defect in other parts of the general auditory or visual word-centres.

In regard to the visual centre as a whole, it seems to be now established that it is more or less diffused through the occipital lobe. As to the localisation of the general auditory centre, there is some clinico-pathological evidence to show that the auditory word-centre may be in, or in the immediate neighbourhood of, the upper temporal convolution.

The situation of the two kinæsthetic word-centres can, however, be rather more precisely localised. The writer holds the opinion that the so-called cortical 'motor-centres' of Ferrier and others are really sensory centres of kinæsthetic type, by means of which movements are guided (*see* KINÆSTHESIS). That being so, Broca's region, or Ferrier's centre for the movements of the mouth and tongue (*viz.* the posterior part of the third frontal and the inferior part of the anterior ascending frontal convolution), is, in reality, the part of the brain which we have been alluding to as the glosso-kinæsthetic word-centre. The situation of the cheiro-kinæsthetic word-centre cannot, however, be localised with as much confidence, though the strong tendency is to follow Exner, who believes it to be situated in the posterior part of the second frontal convolution.

It must be supposed, therefore, that the auditory and the visual word-centres are situated, the one not far away from, and the other actually within, some part of the cortex of the occipital lobe, and that they are connected together by a double set of commissural fibres (*fig. 1, a, b*). We must also

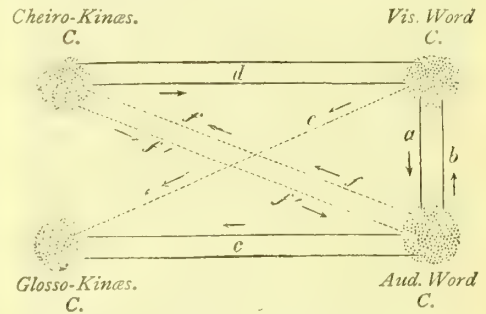


FIG. 1.—Diagram showing the commissural connections between the different word-centres.

- a.* The visuo-auditory commissure.
- b.* The audito-visual commissure.
- c.* The audito-kinæsthetic commissure.
- d.* The visuo-kinæsthetic commissure.

suppose that two other sets of commissural fibres exist which are of much importance in regard to speech and writing, *viz.* one set (*c*) through which the auditory word-centre acts upon the glosso-kinæsthetic centre, for the production of articulate speech; and the other (*d*) by means of which the visual word-centre acts upon the cheiro-kinæsthetic centre, for the production of writing.

Thus, for speech we have the combined auditory and glosso-kinæsthetic word-centres, acting from the cortex through internuncial fibres, upon motor centres in the medulla; and for writing we have combined visual and cheiro-kinæsthetic word-centres, acting from the cortex through internuncial fibres, upon motor centres in the cervical

region of the cord. In the study of speech-defects generally it is, therefore, needful to consider the effects of lesions: (1) in the different kinds of word-centres; (2) in the different commissures by means of which the centres are connected; (3) in certain internuncial fibres; and (4) in certain motor centres.

Defects of Speech from Lesions in Word-Centres.—According to the writer's views it is not at all needful, as many authorities have thought, to postulate the existence of a separate 'centre for conceptions or ideas.' The supposed theoretical necessity for assuming the existence of some such centre may in most cases be obviated by a fuller recognition of the different degrees of functional excitability that may exist in the auditory and the visual word-centres respectively. It should, in fact, be constantly borne in mind by those who study these defects of speech that each perceptive centre is capable of being called into activity in three modes: (a) by 'voluntary' recall of past impressions, as in an act of recollection; (b) by 'association,' that is by impulses communicated from another centre during some act of perception or during some thought process; (c) by means of external impressions. Now the excitability of these centres is liable to vary much—as a result of advanced age, or of different general or local morbid states. The molecular mobility may be so much lowered that they are only capable of responding to powerful stimuli; in such cases either 'volitional' recall alone, or 'associational' recall as well, may be impossible or more or less difficult, responses occurring only to external impressions—that is, the patients are only able to repeat words pronounced, or to copy words written before them.

The effects of lesions of the different word-centres are as follows: (1) A lesion of the glosso-kinæsthetic centre produces simple aphasia; whilst (2) one of the cheiro-kinæsthetic centre produces simple agraphia. In neither condition is the power of thought very appreciably interfered with. These are commonly supposed to be motor defects, but this the writer believes to be an erroneous interpretation—since they are defects due to the loss of certain sensorial aptitudes (of the kinæsthetic order) just as much as are the other forms of aphasia now about to be mentioned, the existence of which was first pointed out and explained by the writer in 1866, though they were subsequently more prominently explained in 1874 by Wernicke under the name of 'sensory aphasias.' One of these has been known as 'word-deafness' and the other as 'word-blindness.' But defects in the activity of the auditory and of the visual word-centres respectively give rise to varied and complicated results, which can only be very inadequately summarised by these phrases. Thus under the head of (3) lesions of the auditory word-centre, we must refer to totally different defects according to the degree of functional degradation of the centre: (a) slight auditory amnesia from functional defect of the centre, leading to impaired spontaneous speech, owing to forgetfulness of names or nouns; (b) profound auditory amnesia, in which, from similar causes, both voluntary and associational speech are greatly impaired, though imitative speech is retained, and the almost speechless patient may be able to read aloud correctly and fluently; (c) word-deafness and aphasia owing to a destructive lesion of the auditory word-centre, in which the patient is not only unable to comprehend speech, but is himself

speechless, and may also be unable to write spontaneously. Lesions of (4) the visual word-centre, whether functional or structural, reveal themselves in less varied modes; when severe they would result in the production of word-blindness together with agraphia.

Defects of Speech from Lesions of the Commissures between Word-centres.—It seems to the writer better to reserve the word 'commissure' as an appellation for the fibres that connect centres of the like kind, that is, either sensory centres or motor centres; and to name 'internuncial' the fibres which connect sensory with motor centres.

As already stated, double commissures exist between the auditory and the visual word-centres (fig. 1, a, b) which are habitually called into play in certain mental operations, so that their destruction leads to very definite defects. Thus, in naming objects at sight, or in reading aloud, stimuli have to pass from the visual to the auditory word-centre (by the visuo-auditory commissure) before the naming or the reading aloud can occur. Again, in writing from dictation, and probably also in writing any spontaneous effusion, stimuli require to pass between these two centres in an opposite direction, namely, from the auditory to the visual centre, and through a different set of fibres (the audio-visual commissure). In 1880 the writer published (*The Brain as an Organ of Mind*, p. 640) some details concerning a man (who has ever since been under observation from time to time) who suffers from a lesion destroying the commissures in question. This man understands perfectly all that is said to him and all that he reads, yet he cannot read aloud a single word or even name a single letter, though immediately that he hears the word or letter pronounced he can repeat it at once. Again, he cannot write a single word, or even letter, from dictation, though he can at once, with his left hand, proceed to copy any such word or letter that may have been written for him on a sheet of paper. Cases have been recorded also in which the separate halves of the commissure between the auditory and the visual word-centres have been damaged; so that reading from dictation or naming at sight has been interfered with and not writing from dictation, or *vice versa*.

The other commissures whose lesions have to be considered are those (fig. 1, c, d) connecting the auditory with the glosso-kinæsthetic, and the visual with the cheiro-kinæsthetic word-centres. Defects in the course of these commissures are of interest, more especially from the point of view of a regional diagnosis. The writer some years ago pointed out, for instance, that a lesion of any part of the audito-kinæsthetic commissure (fig. 1, c) should produce an aphasia indistinguishable from that which would be produced by damage to Broca's convolution, that is, to the glosso-kinæsthetic centre itself. This is, of course, a very important point, since it shows that it is an error to suppose that typical aphasia can only be caused by a lesion in Broca's region. A precisely similar result should also be produced by complete destruction of the audito-kinæsthetic commissure, in any part of its course between that region and the neighbourhood of the occipital lobe where the auditory word-centre is situated. If the latter centre itself be damaged, as before stated, we then get the aphasia complicated with word-deafness, and thus have the means of arriving at an accurate regional diagnosis. Once clear of the centre, however, a lesion involving any part of the audito-

kinæsthetic commissure should produce a typical aphasia absolutely indistinguishable clinically from that resulting from a lesion in Broca's region itself. This explains the occurrence of aphasia in association with lesions in the island of Reil, and other sites nearer the posterior extremity of the Sylvian fissure (fig. 2, *b*).



FIG. 2.—Diagram showing the possible location of an aphasia-producing lesion, either in the glosso-kinæsthetic centre (*a*), in the audito-kinæsthetic commissure (*b*), or, in association with word-deafness, in the auditory word-centre (*c*).

This point, like the question of the existence and interpretation of aphemia, will be found to be a touchstone for testing the truth of the doctrines here set forth concerning the pathogenesis of speech-defects, as compared with those of Stricker and of Hughlings Jackson. According to their views, the commissural fibres, now referred to, pass between the auditory and a true 'motor' centre for articulation. According to Stricker (*Le Langage et la Musique*, 1885, p. 73), a damage occurring in the course of these fibres would cause not aphasia but word-deafness. His view being that words are realised mainly by memorial revival in motor cortical centres, in the case assumed there would be word-deafness because stimuli could not pass from the auditory to the assumed motor centres in which the essential part of the apperceptive process is carried on. It seems clear also that these doctrines necessitate the view that an isolated lesion in Broca's region only should also cause word-deafness as well as aphasia. This, however, is certainly not the case—a fact which is itself sufficient to negative the truth of the views of Hughlings Jackson and of Stricker.

Damage to any part of the visuo-kinæsthetic commissure (fig. 1, *a*) would also be capable of producing agraphia of just as complete and typical a kind as if the lesion had been in the cheiro-kinæsthetic centre itself.

Defects of Speech from Lesions of Internuncial Fibres.—We have here to do with a kind of defect known as aphemia. This is a speech-defect, and there is no analogous defect of writing, except that which is occasioned by actual paralysis of the right hand. In complete aphemia there is absolute loss of articulate speech without any mental impairment, and with the power of communicating the thoughts by writing preserved absolutely intact. An excellent example has been recorded by the writer in the *Brit. Med. Jour.*, Nov. 5, 1887.

Such a condition is produced when the glosso-kinæsthetic centre is itself uninjured, but where the whole of the internuncial fibres emanating from it and proceeding to the true motor articulatory centres in the bulb are damaged in some part of their

course—whether just beneath the cortical centre, in the internal capsule, in the peduncle, or in the pons. Sometimes the defect may be of a functional rather than of a structural type, constituting Charcot's 'hysterical mutism in the male.'

Incomplete aphemias, however, exist of all degrees of severity, and they are about the commonest of all speech-defects occurring in association with hemiplegia. They are due to partial damage to the group of internuncial fibres above referred to, and are represented by defects of articulation, varying from almost complete unintelligibility to the mere blurred utterance or thickness of speech which is so common.

Aphemia is clearly not a sensory defect—it is not a form of amnesia—because the subjects of it can revive words in all possible modes, and are, therefore, able to think and express their thoughts with an unimpaired freedom by writing. If the aphemia be in any way incomplete, moreover, such a case can be easily discriminated from a case of aphasia by the fact that the aphemic patient will always at once make an attempt, when bidden, to pronounce some simple word or syllable (however poor the attempt may be), while the typical aphasic patient is unable to make any such attempt—he will not try even to repeat the simplest vowel-sound.

Defects of Speech due to Lesions in the Bulbar Articulatory Centres.—In these cases, as where the lesions occur in the internuncial fibres, all the centres in which the memory of words can be revived are intact. There is consequently nothing whatever to interfere with the flow of thought, and in incomplete cases nothing to prevent attempts at articulation being made. These two classes of cases are indeed less separable clinically than they are from a theoretical or scientific point of view.

It would be well to reserve the term 'anarthria,' as introduced by Kussmaul, for the defects of speech caused by morbid states of the bulbar articulatory nuclei—with the full understanding, however, that aphemia and anarthria will often be indistinguishable one from another by their own intrinsic characters, though they will generally be quite capable of being diagnosed one from another by taking into consideration the mode of onset and the particular grouping of other attendant signs.

DIAGNOSIS.—The means of distinguishing the different aphasic and amnesic speech-defects one from another has already been fully enough set forth in the preceding sections. The diagnosis of the exact nature of the defect is often exceedingly difficult, unless the patient be thoroughly examined in a systematic manner, and in accordance with some definite plan. Elsewhere (*Paralyses; Cerebral, Bulbar, and Spinal*, 1886, p. 125) the writer has given such a 'Schema for the Examination of Aphasic and Amnesic Persons,' which has been used with success for many years. The other side of the question, viz. the pathological diagnosis, must be made in accordance with the rules generally applicable to this part of the diagnostic problem in cases of brain-disease.

TREATMENT.—Where aphasia occurs after excitement or overwork, without paralysis, it is a warning of much importance, since it may be the precursor of much graver symptoms. Under such circumstances the patient requires an absolute cessation from work for a time, and most careful watching. Stimulants may need to be diminished;

and bromide of potassium, with sumbul and other sedative remedies, should be administered. Where aphasia is a temporary condition in association with right-sided convulsions, or where it is lasting and co-exists with right-sided paralysis, the treatment of the aphasic condition becomes merged in that of the associated convulsive tendency or paralytic condition, since, as a rule, an amelioration takes place in the patient's power of speaking coincidentally with his improvement in other respects. This, however, is not always the case where aphasia has co-existed with a partial hemiplegic condition: the paralysis may be recovered from, while the aphasic defect may remain more or less as it was. Where this is the case, an attempt should be made to teach the patient to speak again, especially if the defect be of the aphemic type. Such efforts have occasionally been crowned with success, but much judgment and untiring patience have to be called into play in order to obtain satisfactory results.

H. CHARLTON BASTIAN.

APHEMIA (ἀ, neg.; and φημί, I speak).—A term differently applied by different writers, but commonly understood to be a designation for certain articulatory defects (*see* ANARTHRIA) dependent upon injury, direct or indirect, to the internuncial fibres connecting Broca's convolution with the motor centres of the bulb. Where the aphemia is incomplete the nature of the defect is generally obvious; where it is complete the patient is absolutely speechless, and is apt to be loosely described as aphasic, though the fact that he can express himself freely by writing shows that this is not a correct designation of his condition. *See* APHASIA.

APHONIA (ἀ, priv.; and φωνή, the voice). Absence of voice, that is, of intonated utterance. *See* VOICE, Disorders of.

APHRODISIACS (Ἀφροδίτη, Venus).—DEFINITION.—Medicines which increase the sexual appetite and power.

ENUMERATION.—The direct aphrodisiacs include: Nux Vomica and Strychnine, Phosphorus, Cantharides; Quinine; Urtication and Flagellation; Cannabis Indica, Damiana, Opium, and Alcohol in small doses. Iron and bitter tonics; meat-diet; warm clothing, especially around the hips and loins; and abstinence from severe mental and bodily work, act as indirect aphrodisiacs.

ACTION.—Aphrodisiacs may act by increasing the excitability of the nerves passing to or from the genital organs, or of the genital centre in the spinal cord (*see* ANAPHRODISIACS), as, for example, strychnine, nux vomica, and probably phosphorus; by causing irritation of the nerves of the genital or urinary organs or of adjoining parts, as cantharides and urtication; or by stimulating the brain, as Indian hemp or small doses of opium. Alcohol in large doses has a double action, increasing the sexual desire by stimulating the brain, while lessening the power of erection, probably by weakening the nervous mechanism through which erection is produced. As the sexual passion becomes diminished when the nervous system is weakened with the rest of the body, and increases with returning strength, iron with bitter tonics, and generous diet act indirectly as aphrodisiacs.

USES.—When the sexual functions are abnormally depressed, strychnine and phosphorus are the most

generally useful of the direct aphrodisiacs. Cantharides, although sometimes valuable, must be employed with caution.

T. LAUDER BRUNTON.

APHTHÆ (ἀπρω, I inflame).—SYNON.: Aphthous Stomatitis; Fr. *Muguet*; Ger. *Fasch*.

DEFINITION.—A form of catarrh of the buccal mucosa, characterised by the appearance of one or more patches (*aphtæ*) possessing a peculiar structure.

ANATOMICAL CHARACTERS.—Aphthæ have at first the appearance of vesicles; but microscopical sections show that they are due to elevation of the epithelium by a solid fibrinous exudation, over which the epithelium usually dies, soon separating with the fibrinous flake. By the time this is effected the mucosa is again, as a rule, thinly covered by epithelium, so that a true ulcer is never present; but slight suppuration may be established, with the destruction of the superficial layer of the mucosa.

SYMPTOMS, COURSE, AND DURATION.—Aphthæ occur chiefly in children during the first dentition, upon any part of the buccal mucosa—most frequently inside the lower lip and on the tip of the tongue. At first they look like small vesicles, but after a few hours they resemble ulcers with adherent grey or yellowish sloughs, each being surrounded by a ring of injection. The patches vary in size from that of a lentil downwards; they are single or multiple; grouped or scattered; two or three may coalesce, but the patches never reach a large size. Aphthæ cause slight pain, and are especially sensitive to substances like salt or sugar. Dyspeptic troubles are usually present, and the breath is often foul.

Each aphtha runs its course in three or four days, and when multiple the patches come out in a crop. This may be succeeded by other crops, so that all stages of the lesion may be present at one time. The trouble may thus continue for weeks. In other cases patients are liable to crops of aphthæ at varying and sometimes long intervals.

DIAGNOSIS.—Aphthæ have chiefly been mistaken for patches of 'thrush.' Absence of the red areola, and presence of the characteristic fungus, will distinguish thrush even when no history or statement of symptoms can be obtained.

TREATMENT.—The occasional application of a little powdered borax to the aphthæ causes their speedy healing. In prolonged cases, the health in general, and that of the stomach and intestines in particular, must be attended to.

STANLEY BOYD.

APHTHOUS.—A term applied to diseases in which aphthæ are present.

APNEUMATOSIS (ἀ, priv.; and πνεῦμα, respiration).—A synonym for atelectasis. *See* ATELECTASIS.

APNŒA (ἀ, priv.; and πνέω, I breathe).—Apnœa, literally signifying breathlessness, is used by some medical writers as synonymous with asphyxia, the condition which supervenes on suspension or obstruction of the respiratory function. *See* ASPHYXIA.

By physiologists, and with more correctness, the term is employed to signify the cessation of respiratory movements which is brought about by hyper-

oxygenation of the blood; as when an animal is made to inhale oxygen, or to breathe more rapidly than the needs of its economy require.

APOLLINARIS, Waters of.—Acidulous alkaline table-waters. See MINERAL WATERS.

APOPLEXY (ἀπο-πλήσσω, to strike to earth, or disable in mind or body).—The term was at first, and is still, chiefly used to signify sudden abolition of consciousness and power of motion, which, in common English, is also called a *stroke*.

Cerebral hæmorrhage is the most frequent cause of apoplexy; thence 'hæmorrhage into the brain' and 'apoplexy' came to be used as synonymous expressions. Subsequently the effusion of blood itself was spoken of as *the apoplexy*, the word being used to designate the pathological condition causing the symptoms which it at first epitomised. Ultimately it was applied to such spontaneous hæmorrhage elsewhere; and thus extravasations into the substance of the lung, the spleen, or the retina were, and by some writers still are, termed pulmonary, splenic, or retinal 'apoplexies.'

W. R. GOWERS.

APOPLEXY, CEREBRAL.—SYNON. : a 'stroke;' Fr. *Apoplexie*; Ger. *Schlag*. See also APOPLEXY.

DEFINITION.—Loss of consciousness, of voluntary motion, and usually also of evidence of sensation, except that which is afforded by reflex action, coming on more or less suddenly, lasting more than a few minutes, and due to a morbid state of the brain.

The condition of *coma* is termed 'apoplectic' when of sudden or rapid onset. Loss of consciousness may be due to other causes acting directly on the brain, such as defective or excessive supply, or altered condition, of blood; but it is customary to include among the forms of *apoplexy* that only which is due to cerebral hæmorrhage or cerebral congestion, and those which result from distinct toxæmia are sometimes distinguished as apoplectic states. But the word is now seldom used in connection with blood-states, 'coma' being preferred. Unconsciousness resulting from acute anæmia of the brain, or such as attends an epileptic fit, is not termed apoplectic. The term has, of necessity, become restricted, as increasing knowledge has explained the nature of many conditions previously confounded with apoplexy. It should be further observed that the term has always been restricted to such conditions as occur without conspicuous external cause. The same state, when due to an injury, is not called by the name which is employed if there is no injury. The explanation of the inconsistency is to be found in the idea that is to be traced alike in the native and the foreign word. The word 'stroke' has reference to an unseen striker.

The essential cause of apoplexy is thus a sudden cerebral lesion—usually hæmorrhage—occurring without external injury and abolishing for a time the functions of the brain. The same functional state may be caused by obstruction of a vessel by thrombosis or embolism. Profound coma is rarely due to any other spontaneous cerebral lesion. A very small hæmorrhage may cause apoplexy.

Apoplectic symptoms may, however, occur without obvious lesion of the brain. The coma, which is associated with congestion of the head, is sometimes termed 'congestive apoplexy,' and that which occurs in the absence of any recognisable cause has

been called 'simple apoplexy.' This will be referred to presently.

PATHOLOGY.—In all these cases the apoplexy is in relation chiefly to the extent and suddenness of the lesion, and the intensity of the damage. Roughly speaking, its occurrence may be said to depend on the suddenness of the cerebral mischief; its degree on the extent of the same. But the occurrence of apoplexy depends sometimes on the size of the lesion; and the degree varies not only directly with the extent of the mischief, but with the extent of brain-tissue which is exposed indirectly to the irritative influence of the primary lesion. Hence position of lesion has an influence in determining the apoplectic symptoms. For these several reasons apoplexy is especially profound when the hæmorrhage affects both hemispheres, either by simultaneous independent extravasation on each side, or as the result of hæmorrhage into the lateral ventricles, or of hæmorrhage into the pons whence fibres pass to both sides.

The precise condition on which the essential element in apoplexy—the arrest of all manifestation of brain-function—is immediately dependent has been a matter of much dispute. It was formerly ascribed to the pressure exerted by the extravasated blood on the rest of the brain, either influencing directly the cerebral tissue, or pressing on and emptying its capillaries. That such pressure is exerted by a large hæmorrhage is unquestionable. The convolutions on the side of the extravasation are flattened, and the falx is bulged to the opposite side. It cannot be doubted that the intensity of the apoplexy in these cases is due in part to this cause. But this will not explain the occurrence of the symptom in small hæmorrhages, by which no general pressure is exerted, or not more than is at once relieved by the displacement of the mobile fluid which surrounds the vessels. It will not explain symptoms in laceration of the brain, nor the instant loss of consciousness at the very onset of a severe hæmorrhage, in which, as Jaccoud insists, it should, if merely due to pressure, be a late rather than an early symptom. There can be little doubt from these considerations, and from the cases in which there is no recognisable brain-lesion, that what is called shock is an important element in the causation of apoplexy. Thus, in cerebral hæmorrhage the apoplectic symptoms are due in part to the influence on the rest of the brain of the irritation of the nerve-elements by laceration. A hæmorrhage, for instance, stops for a few hours all spinal reflex action by the irritative inhibition downwards, and it stops also the cortical functions by the same process acting upwards. We can thus understand why vascular occlusion causes a slighter degree of apoplexy, since the immediate irritation of the local anæmia is less than that of laceration of the nerve-elements; and also why lesions of the pons produce, as they do, such deep and long-continued coma, since the irritated fibres are connected with a large part of the cortex of both cerebral hemispheres.

Simple apoplexy was a term given by Abercrombie to the cases, once thought to be frequent, in which apoplexy occurs without recognisable brain-mischief or blood-poisoning. Some of these cases were probably instances of uræmic poisoning, and others may have been due to undiscovered and extensive embolism, causing death before there was time for softening to occur. But cases are not infrequent to which neither of these explanations

applies: in which death occurs in an apoplectic form attack, all the organs are found healthy, and the brain only exhibits, in common with the other organs, that passive congestion which results from an asphyxial mode of death. The nature of these cases is still mysterious, but they may be grouped with those in which fatal coma follows an epileptic attack, and is apparently due to the brain-shock produced.

Serous apoplexy is a term formerly applied to cases of fatal apoplexy in which no lesion was discovered except an excess of serum on the surface of the brain. It is now understood that such serous effusion is associated merely with senile atrophy of the brain. There is no reason for associating its presence with the apoplectic symptoms. The cases described under this term were probably instances of uræmia, or of 'simple apoplexy' in old persons with atrophied brains.

SYMPTOMS.—Apoplexy is itself a symptom, and can only claim to be treated as a disease on account of the frequency with which it presents itself as the prominent element in a patient's state. The striking feature of apoplexy is loss of consciousness without failure of the heart's action. The onset is often instantaneous, so that the sufferer falls to the ground. The face may be flushed or pale—it is rarely very pale. The heart and arteries vary in their state; at first the pulse may be feeble from shock, even in cases in which it soon becomes full and tense. Respiration continues, but is laboured and stertorous. The limbs are motionless. In severe cases no reflex action can at first be excited. The pupils may be dilated, contracted, or unchanged: in profound coma they are usually dilated; and they often vary in size spontaneously, and are sluggish in their action to light. The patient can usually swallow, although often with difficulty. The sphincters may permit the escape of urine and feces, or the urine may be retained. In a case of moderate severity the reflex action soon returns, the conjunctivæ become sensitive, the patient can be roused to exhibit response to excitation, and he soon shows returning power of motion. At first there is merely withdrawal of a limb when it is pinched or pricked, but afterwards distinct purposive movements, e.g. opposing disturbance. Afterwards more prolonged and voluntary movements are produced. The patient opens his eyes when spoken to, and tries, when told to do so, to protrude his tongue. On the other hand, the apoplexy may continue or even may deepen in intensity; the respiration assumes the Cheyne-Stokes rhythm, the heart becomes irregular, and the patient dies at the end of a few hours or a few days. Death rarely occurs in a shorter time than two or three hours. In very rare instances an extensive hæmorrhage into the pons or medulla or an effusion of blood into the fourth ventricle may stop the respiration or heart, and kill the patient, in a few minutes.

It is not often, however, that there is this simple loss of cerebral function, uniformly distributed, and gradually deepening or passing away. Much more commonly the symptoms of a local cerebral lesion are added to those of apoplexy. Frequently such symptoms precede the loss of consciousness—unilateral weakness. A unilateral difference may often be recognised during the apoplectic state. The limbs on the one side exhibit more complete muscular relaxation than those on the other; they

fall more helplessly when raised; or there is unilateral rigidity or clonic spasm. Inequality of pupils may be observed, or rotation of the head and conjugate deviation of the eyes. As a patient recovers, these local symptoms become more and more distinct, movement of the face is unequal; one arm is motionless, and the tongue deviates on protrusion.

In *ingravescent* apoplexy the commencement of the cerebral mischief is marked by symptoms of general shock, without any, or with merely transient, loss of consciousness. There is commonly pain in the head, and there may be other localising symptoms. After some hours, during which the patient may continue his occupation, coma gradually comes on and deepens into death. This form of apoplexy, first described by Abercrombie, is usually due to a slowly increasing cerebral hæmorrhage.

The temperature in cerebral apoplexy is at first lowered in most cases, but usually the fall is small, and is succeeded, after twelve to twenty-four hours, by a rise. In some cases there is a considerable rise within the first six hours, and the temperature then commonly continues to rise, reaching 104° or 106° F., and even higher. Such a symptom is a precursor of death.

DIAGNOSIS.—The first diagnostic problem is whether the patient's state comes under the general name of apoplexy; or is one of the morbid conditions in which there is a loss of consciousness, similar in general characters to apoplexy, but due to one of the causes excluded in the definition given above. From the unconsciousness due to cardiac *syncope*, apoplexy is easily distinguished. In the former the heart's action fails, the pulse is weak and imperceptible, the face is very pale, the respiration is sighing and irregular, reflex action is rarely abolished, the sphincters are seldom relaxed, and the patient soon revives.

From the several forms of *toxæmia* the diagnosis is often easy, sometimes extremely difficult. It is easy when, on the one hand, the symptoms of apoplexy are preceded or accompanied by those of a local cerebral lesion; or when, on the other hand, the direct or circumstantial evidence of poisoning is clear, or the symptoms of toxæmia unmistakable. Where there are no local symptoms, and where no guiding history is to be obtained, the diagnosis is difficult, but a correct opinion may commonly be formed by an attentive comparison of the symptoms present.

There may be, as just observed, indirect evidence of toxæmia: the breath may smell of opium or alcohol; the urine may contain albumen. But albuminuria or a smell of spirit may mislead. Cerebral hæmorrhage often occurs after drinking; spirit is usually given to a person who loses consciousness. A smell of spirit must therefore only be allowed weight in the absence of any evidence of cerebral mischief. So, too, albumen is always present in the urine in uræmia, but it is also very frequently present in cases of cerebral hæmorrhage. Alone, this evidence of Bright's disease is of little value, except there be general œdema and the patient be young; then uræmia is more probable than cerebral hæmorrhage. The urine should always be examined microscopically for evidence of structural changes in the kidneys. But with other symptoms which suggest uræmic poisoning, albuminuria is conclusive.

The age of the patient should be considered. Late life is in favour of brain-disease. The history of a fall or a blow on the head adds weight to other symptoms of cerebral mischief.

The character of the coma will sometimes guide us. In uræmia, and commonly in alcoholism, it is less profound than in cerebral mischief, so that the patient can readily be roused. In apoplexy, in opium-poisoning, and in the most intense form of alcoholic poisoning, the coma may be profound. On the other hand, in cerebral hæmorrhage the patient may sometimes be roused to answer questions. Violent struggling is strongly in favour of drink.

The mode of onset of the coma is important. In apoplexy it is sudden; in uræmia it is slow. The uræmic patient becomes first drowsy, then comatose. But with convulsions uræmic coma may come on suddenly. The onset of the coma of opium- and alcohol-poisoning is also slow. In-gravescent apoplexy is of deliberate onset, but a profound degree of coma is quickly reached.

General convulsions at the onset exclude drunkenness, and usually opium-poisoning, while they favour the probability of uræmia, especially if they are followed by rigidity that changes its seat. When uræmic convulsions begin locally, the place at which they begin often varies. Cerebral mischief sometimes commences with a convulsion, but the convulsion is then commonly unilateral, at least in onset, and the preponderant affection of one side at a time is indicated by the deviation of the head. It is seldom that well-marked deviation of the head and eyes occurs, first to one side and then to the other, as the fit progresses, unless there is an organic lesion of the brain, and one-sided symptoms are almost always to be recognised afterwards. Rigidity of limbs or local muscular twitching during the coma is, if constant in seat, in favour of cerebral mischief; if variable in position, it is in favour of uræmia (Reynolds). In diabetic coma, the odour of the breath, and the result of an examination of the urine (never to be omitted) will prevent mistake, even in the absence of a history of diabetes. But the absence of local cerebral symptoms must be carefully noted, since the presence of diabetes does not exclude a cerebral lesion. Post-epileptic coma is of course preceded by a convulsion, and should be borne in mind. In all cases the state of the limbs on the two sides should be carefully observed. When there is brain-disease it is common to have some movement on one side, and great flaccidity on the other. There is often also a difference in reflex action and in the effect of sensory stimulation of the skin.

The state of the pupils alone is of little importance. Great contraction occurs in and suggests opium-poisoning, but it is present in hæmorrhage into the pons Varolii. The pupils may be either normal or dilated in uræmia, in alcoholic or in belladonna-poisoning, and in apoplexy. Inequality of pupils, a unilateral symptom, points to brain-mischief. The retina should be examined, since the presence of albuminuric retinitis points, in the absence of the signs of a localised cerebral lesion, strongly to uræmia.

Lastly, the temperature should be noted. In uræmia there is persistent uniform depression; in cerebral lesions the initial depression is succeeded by a rise to a point above the normal.

The second problem in diagnosis is the precise

cause of the apoplexy, the existence of which has been ascertained by the considerations just reviewed. This element, however, will be described more fully under the heads of the chief lesions that produce the state—cerebral congestion, hæmorrhage, and softening. It may be here pointed out that slight and transient apoplexy, without local symptoms, with flushed face, and coming on during effort, points to cerebral congestion; slight and transient apoplexy with marked local symptoms points to softening; early and profound loss of consciousness to cerebral hæmorrhage. Post-epileptic coma may be distinguished by the history of epileptic attacks; or, if this be not forthcoming, it may be suspected if symptoms of a local cerebral lesion or indications of toxæmia are absent, if the patient be under forty, and exhibits indications of speedy recovery, and also if he has bitten his tongue and passed urine. 'Simple apoplexy' cannot be diagnosed during life, since freedom from the symptoms of a local lesion does not afford ground for inferring that there is no such lesion.

PROGNOSIS.—The prognosis in cerebral apoplexy depends in part upon the intensity of the attack. As long as unconsciousness is complete, and reflex action abolished, the patient is in danger of speedy death. The longer the apoplectic condition lasts without improvement, the less prospect is there of recovery. Persistent depression of temperature, or a rise of several degrees above the normal either with or without an initial fall, is of grave significance; such cases rarely recover (Charcot, Bourneville).

The nature, extent, and position of the cerebral lesion, when they can be inferred, furnish other prognostic indications. In hæmorrhage the prognosis is more serious than in softening. A sudden occurrence or increase of apoplectic symptoms, a few hours or days after a slighter attack, is always grave, indicating a fresh extravasation. If such apoplectic symptoms become profound and uniform, the prognosis is fatal, rupture into the ventricles or on the surface of the brain having probably occurred. If the localising symptoms point to a lesion of the medulla or pons, the prognosis is almost as unfavourable. Early return of consciousness and slightness of alteration in temperature are favourable signs. Previous cerebral disease renders the prognosis worse. Lastly, the prognosis must be influenced unfavourably by any impairment of the organic functions of circulation and respiration, whether independent of or due to the cerebral lesion. Previous alcoholism, from its effect on the nutrition of all tissues, is especially serious.

TREATMENT.—The treatment of cerebral apoplexy must be guided by the indications of its cause. Where none can be obtained, it should be treated by a system that is a compromise. The chief causes are hæmorrhage and thrombotic softening of the brain. Embolism seldom causes pronounced apoplexy. The measures therefore must be such as are suitable to both states, those being excluded which, while they would do good in one, would do harm in the other. But such a compromise is seldom needed. Diagnostic indications can usually be traced with clearness, and as a rule also several coincide distinctly in this direction, while those that do not point in this direction are neutral. Hence, as a matter of fact, there should seldom be the hesitation that interferes with the proper energy of treatment. Stillness is the most important con-

dition. The patient should be moved as little as possible, but placed in the recumbent posture with the head slightly raised. The neck should be freed from constriction. If the extremities are cold, warmth may be applied to them; and cold to the head if there is local heat or flushing. Sinapisms to the neck and extremities sometimes seem to hasten the return of consciousness. The administration of stimulants should be regulated by the state of the heart. In thrombosis or embolism, with signs of cardiac weakness, the heart should be kept up to the normal by very careful administration of alcohol, ether, or ammonia. In hæmorrhage it may be allowed to fall a little below the normal, but indications of failing power should be watched for and counteracted. Where no causal indications exist, the latter is the wiser plan. Venesection and purgation are remedies of similar effect, but different in degree, and are indicated by high arterial tension and cephalic congestion, shown by incompressibility of the pulse and flushing of the face. Venesection is useful where the heart acts strongly, and the pulse is full as well as incompressible. Its effect is proportioned to the rapidity with which the blood is taken, rather than to the quantity removed. Purgatives remove serum from the blood, and lessen the amount of blood within the skull by causing an afflux to the capacious intestinal vessels. The best purgative is croton oil or calomel. With a failing heart and pale surface they should be avoided. When in doubt we should give a mild aperient that acts quickly, such as senna or a little solid extract of cascara rubbed up in water, and the action should be ensured by a glycerine enema. Diuretics may always be used to relieve the vascular tension. As the apoplexy clears, the nature of the case becomes evident, and the treatment of the several conditions is described elsewhere (*see* BRAIN, Hæmorrhage into, and BRAIN, Softening of). For treatment of the other causes of the apoplectic state, *see* ALCOHOLISM, POISONS, and URÆMIA. W. R. GOWERS.

APPENDIX VERMIFORMIS, Inflammation of.—SYNON. : Appendicitis; Epityphlitis.

DEFINITION.—A large proportion of cases of this disease tend to involve the peritoneum locally (*perityphlitis*) or generally, so that the name *appendicitis* has come clinically to have a wider range of application than mere inflammation of the appendix vermiformis, and to include peritonitis starting from the appendix. Indeed, appendicitis apart from peritonitis can scarcely be said to occur clinically.

ÆTIOLOGY AND PATHOLOGY.—Inflammation of the appendix conforms in its anatomical characters to the general features exhibited by this morbid process in mucous membranes elsewhere. Various forms such as 'catarrhal' and 'suppurative' are described, but the interest lies rather with the causes, both predisposing and exciting, which lead to these states, and with the results which follow on their becoming established.

There is little doubt but that the essential cause is *microbic infection*, and it is most probable that the *Bacillus coli communis* is the organism that, in the majority of cases, brings about the disease (*see* B. COLI COMMUNIS). Other microbes, such as *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*, and *Diplococcus pneumoniae*, have been met with in association with the B. coli, and, in very rare cases, have been the only organisms found. It would

seem that the B. coli, which is a constant inhabitant of the intestines in health, may take on virulent characters; and it is possible that the presence of the cocci contributes to this end, as also may other conditions existing in the canal which are to be regarded as predisposing causes. Among these latter is the presence in the appendix of foreign bodies which are only very occasionally derived from the ingesta—such as stones or seeds of fruit—but which far more commonly consist of fecal concretions having for a nucleus masses of bacteria, and often simulating vegetable seeds in their appearance. The irritation of these substances impacted more or less firmly in the appendix may be the starting point of the inflammation.

Most cases of appendicitis are to be attributed to some morbid condition of the alimentary canal. One of the commonest precursors of an attack is a large and indigestible meal, taken perhaps when in a state of fatigue after a long abstinence, or when in a condition of mental depression or excitement. The result is that a considerable portion of the food escapes gastric and intestinal digestion, and may be found scarcely altered in the cæcum. Constipation is also a very frequent cause, and it is supposed that this state assists in increasing the virulence of the B. coli. An inadequate emptying of the cæcum and appendix is immediately responsible for a large proportion of the cases. Increased and irregular peristalsis extending into the appendix may easily render any existing concretion more dangerous; and the appendix may also be involved in the extension of any neighbouring inflammation, ulceration, or new-growth, as for example actinomycosis or tubercular ulceration. Similar conditions may of course originate in the appendix itself. That appendicitis is often due to disease of the right ovary or Fallopian tube is undoubted, and the communication known to exist between the lymphatics of these several structures probably explains the fact. Whether the ovary may be secondarily affected from the appendix is not so certain.

Direct injury to the appendix is sometimes, though very rarely, the sole cause, but it may light up a morbid state already in existence. Kinks or twists of the organ are occasionally the origin, and these owe their occurrence to differences in the length and position of the appendix and to the extent of the mesentery which it possesses. If the lumen be thus occluded, the secretions are retained and swelling of the distal part takes place, thereby favouring the growth and diffusion of any organisms present, as well as impeding the blood-supply.

Certain other anatomical features are regarded as predisposing causes. Thus the fact that the appendix is an obsolete structure is supposed to favour the incidence of inflammation; while the large amount of lymphoid tissue contained in its walls has suggested a comparison with the tonsils, and to some has justified the existence of a 'rheumatic appendicitis'—an assumption that will much depend upon the view taken as to the nature of 'rheumatism.' The occasional association of arthritis with appendicitis has also been regarded as supporting a 'rheumatic' origin for some cases. However this may be, it is certain that those cases in which 'chill' appears to be the provoking factor are often very completely relieved by salicylate of sodium. Again, a difference in the arterial supply of the two sexes has been noted, and may seem, in part, to explain the greater proportion

of cases among males, in whom there is but one arterial branch, while in females an additional twig is derived from the ovarian artery, an arrangement which diminishes the chance of gangrene. Of still greater importance is the discovery that the sub-mucous tissue is continuous with the subserous tissue through intervals in the muscular layers, a disposition which obviously facilitates the extension of an inflammation of the mucosa to the peritoneal coat.

It is significant that the *results of experiments* designed to set up appendicitis in animals (rabbits) tend to confirm the relative insignificance of foreign bodies as a cause of the disease, and the preponderant importance of blocking of the lumen of the organ in increasing the virulence of the contained organisms—a result which, together with the impaired resistance of the tissues, is aggravated by the impeded blood-supply consequent on twisting of the organ with compression of the vessels.

Nearly three-quarters of the cases occur between the ages of ten and thirty, and more than half before the age of twenty, but the disease has been met with in children three years old, and as late in life as eighty. Statistics show the disease to be more prevalent in summer and in hot climates.

MORBID ANATOMY.—As regards the morbid changes met with in the appendix great variety is to be seen. In those cases which run an acute course, the earliest conditions may require a microscopic examination for their detection, and an appendix that exhibits no naked-eye appearances of disease is not therefore on that account to be considered as healthy. In such cases a simple catarrh of the mucosa is found with proliferation of the epithelium, an excessive mucous secretion, and innumerable micro-organisms of the varieties mentioned—all filling up the lumen of the appendix. Minute ulcerations of the mucosa may be detected, and an invasion of the submucosa by the organisms. In a number of cases the change may be limited to this, and many of those which recover may be of this character, but the possibility of extension to the peritoneum is apparent. In other cases suppuration develops in the wall of the organ, being determined by the invasion of micro-organisms through the damaged mucous membrane. Extension to the peritoneum takes place and a local peritonitis is set up, but the liability for this to become general is obvious. In other cases, ulceration of the mucosa, extending through the thickness of the wall, leads to perforation and escape of the appendicular contents, with subsequent general septic peritonitis, unless this be limited by adhesions. In the worst cases the appendix may rapidly become wholly or in part gangrenous, and in that way lead to peritonitis. It is the occurrence of general peritonitis that confers the grave character on this disease, and this complication is clearly liable to ensue in any case, however mild the initial process may be.

An *appendicular abscess* is primarily intraperitoneal, and is an encysted form of suppurative peritonitis. In the majority of instances the centre of the abscess is behind the cæcum. The contained pus is very rarely of wholesome appearance and free from offensive odour. In the large majority of the cases, however, it has a distinctly fecal smell, a circumstance which attends most abscesses in connection with the lower bowel, and which does not of necessity imply that the contents of the intestine have escaped into the abscess-cavity. In the

minority of the examples the abscess does contain fecal matter, and the lumen of the intestinal canal is opened up. The fecal material may have escaped through a rent in the appendix, or may have followed gangrene of that process, or, less often, may have made its way through an aperture in the cæcum. This perforation may have proceeded from within outwards, but more usually it is due to the bursting into the cæcum of an abscess which had commenced outside the caput coli. The direction in which the abscess tends to burst is illustrated by sixty-seven cases collected by Bull. In twenty-eight instances it burst through the anterior abdominal parietes; in fifteen it entered the cæcum; in eight the general peritoneal cavity; and in two instances each it made its way respectively into the thorax, the rectum, and the bladder. The abscess often shows a tendency to extend upwards behind or along the ascending colon, and it is by this route that the thorax has been reached and an empyema produced. The abscess, when it extends to the pelvis, may pursue the course of any other pelvic abscess, and may thus make its way out into the buttock and perinæum, and may even extend down the thigh. The comparative frequency with which a suppurative collection, started by disease in the appendix, may make its way into the cæcum has given rise to an exaggerated and erroneous idea of the frequency with which the cæcum is primarily involved in these cases. Suppurative pylephlebitis may be met with in association with perityphlitis.

As a sequence of repeated attacks of inflammation, the lumen of the appendix may become obliterated and its walls converted into tissue of tedious hardness. In these chronic cases, the organ may be embedded in dense fibrous tissue, the result of a progressive adhesive peritonitis.

SYMPTOMS AND COURSE.—The signs and symptoms which are associated with the structural changes just described, and determined by the causes just indicated, exhibit a marked variety in mode of onset, course, severity, and duration; so much so that descriptions of extreme cases are scarcely recognisable at first as pertaining to what we regard as the same disease. The exact relation of the clinical phenomena observed to the existing anatomical changes is sometimes uncertain, more especially in the milder cases which end in complete recovery, and cannot be investigated *post mortem*. Cases of severe pain over the right iliac fossa, the point of greatest intensity and tenderness being midway between the umbilicus and the right anterior superior iliac spine but without any recognisable physical signs, have been termed 'appendicular colic'—an unfortunate phrase, since there is no reason to suppose that their cause is a painful muscular contraction, though there are good grounds on the other hand for believing that such symptoms are associated with the early structural changes above described.

Three other well-marked groups may be recognised, to one or other of which most cases may be referred, though occasionally the disease runs a course which cannot be exactly included within any one of these types.

(a) In the first group will be found those cases which, occurring in apparently healthy persons, or with but very little and unimportant previous symptoms, run an exceedingly rapid course of a few hours, death resulting at the outside within two days. Here the symptoms are mainly those of

perforation of the intestine, with very little indication of the seat of the lesion. The patient is suddenly attacked with severe abdominal pain, by no means necessarily felt in the neighbourhood of the cæcum, but often about the umbilicus, followed by all the symptoms of collapse, which may be very soon fatal (*see* INTESTINES, Perforation and Rupture of). The necropsy reveals perforation of the vermiform appendix or gangrene of that organ and more or less diffused peritoneal inflammation. Intestinal toxæmia with rapid loss of strength and often marked vomiting are features of this variety.

(b) The onset of the symptoms in the next group also takes place as a rule without any history of previous discomfort, constipation, or illness; and while less rapid than in the preceding, they are usually well marked. Often commencing with a chill or rigor, these cases are also characterised by acute pain, indifferently referred to the belly generally, or perhaps specially to the umbilical or cæcal region. The pain may cause a moderate degree of collapse, but not nearly so severe as that associated with acute perforation, which we may surmise in the cases under consideration has not yet actually taken place; or if it have occurred its effects are strictly confined to the immediately adjacent region. With the pain, which becomes paroxysmal and often radiating to the testis, pudendum, thigh, or bladder, there is considerable tenderness, which may be distinctly limited to the right iliac region and most marked at the point previously mentioned, and thereby becomes an important factor in the diagnosis. Other signs of localised peritonitis quickly supervene: the abdomen becomes distended and tympanitic; and a slightly increased resistance is perceptible over the cæcum, due in part to the rigidity of the abdominal muscles, and partly perhaps to retained feces in the cæcum, or inflammatory effusion around that structure. The temperature rises two or three degrees, or even to 104° or 105° F.; the pulse is frequent and thready; respirations are quickened; and the aspect of the patient is that of severe illness. There is constipation; often severe tenesmus; and vomiting, which may be mild and only excited by food, or of a bilious character, but its occurrence is not followed by any sensation of relief. The tongue is thickly coated, or dry brown and cracked, with sordes on the teeth. Some observers have noted a very abundant flow of urine, containing an excess of indican; and frequency of micturition. The patient may remain in much the same state for two or three days, when the pain and tenderness spread over the abdomen. The legs are drawn up and the arms raised above the head so as to relax the parietes and increase the capacity of the abdominal cavity; and with an intensification of the above symptoms there exist all the evidences of general peritonitis, from which the patient may succumb within the next few days, though recovery even at this stage is not quite unknown. Or the inflammation may assume a distinctly suppurative character, leading to the formation of a large quantity of pus encysted within the peritoneal cavity in the cæcal region, giving rise to an increasing fulness and resistance, which may be detected through the abdominal wall, or sometimes by rectal or vaginal examination. The abscess thus formed may extend, and burst in one or other of the directions already indicated. On the abdomen being opened, either by the surgeon or *post mortem*, the mischief is seen to have started from ulceration and perforation of the appen-

dix; and these cases only differ from those in the former group in degree of severity and rapidity, their longer duration affording the opportunity for the development of additional symptoms. It may be observed that cases of this severe type do occasionally begin with mild symptoms such as characterise the following group, or the graver manifestations may be delayed in their occurrence; but it is estimated that 68 per cent. of the fatal cases die within eight days.

(c) The third class of cases—certainly the most numerous—include those which are much less severe in their character, more prolonged in duration, and generally recover. Contrary to what obtains in the previous groups, the patients are more frequently women, and a previous history of constipation or of ovarian trouble is the general rule. After perhaps a more than usually obstinate confinement of the bowels, or some gross error in diet, such as eating a very large quantity of nuts, the patient complains of very definite pain over the cæcum, which is found to be tender, swollen, resistant, and dull on percussion, due to fecal accumulation. It is the prominence of the local symptoms which specially characterises this class of cases, the seat of the trouble being obvious from the commencement. There is also a moderate and very irregular rise of temperature, rarely exceeding 103° F., with soft quick pulse, and general febrile symptoms. The constipation persists, owing to the arrested peristalsis from inflammation of the bowels; and nausea or vomiting is of frequent occurrence, though not often very severe. It is in these patients that is commonly noticed the drawing-up of the right leg, which is flexed across the other thigh, so as to relieve as far as possible the pressure of the abdominal wall on the swollen region. The pain continues of a dull aching character, now and then with sharper twinges, and often spreads across the lower part of the abdomen, and suggests an extension of the peritonitis, though it is doubtful whether this is really the case; it also shows a great tendency to pass down the inner and anterior surface of the right thigh, due to pressure on the genitocrural nerve. Sometimes there is oedema of the right leg, from thrombosis of the iliac vein induced by pressure. The duration of these cases is very uncertain, and much depends on their treatment; some last but a few days, others may continue for two or even three weeks, varying much in the severity of the symptoms, for some days the patient being gravely ill and then appearing considerably better, fluctuating from day to day. The extent of the swelling and dullness is frequently changing, as also to a less degree the pain and general symptoms. A small proportion of the cases which run the course described end in suppuration, and the formation of a circumscribed abscess, such as occurs in the former group; but the greater majority end in resolution. How far the appendix is concerned in these cases is doubtful, and certainly it is unlikely that gross lesions of that structure occur in any but the few which suppurate. It is probable also that in certain of the milder cases the cæcum is more at fault, being in a state of inflammation and probable ulceration, determined by the irritation of the hardened feces, and that it is a true typhlitis—the *stercoral typhlitis* of writers—the well-defined swelling being partly formed by the thickened wall of the inflamed bowel, with perhaps some peritonitis (perityphlitis), and still more by the retained intestinal contents.

Relapsing appendicitis.—A marked characteristic of the morbid state under consideration is its liability to relapse—40 per cent. of the cases are estimated to do so. A slight indiscretion in diet may be sufficient to re-induce it, and it may recur from this cause again and again; or it may be that the patient has got about before the attack has completely subsided; or in many cases relapse may occur when every care has been taken, and successive recurrences are likely to be of increasing severity. In women attacks are often coincident with the 'period.' When suppuration has taken place as a result of perforation of the appendix and escape of the original irritant, concretion, or foreign body in the discharge from the abscess, recurrence is not likely to happen; but so long as it remains it is an ever-present source of danger: there will be no relapses if the appendix have become gangrenous and separate. The exciting causes of these relapses may be found in the condition of the appendix itself, which by becoming twisted or bent on itself, fixed by adhesion, or stenosed by a cicatrix of an old ulcer, permits an accumulation of mucus within it, and consequent distension of its distal end; this condition is sufficient to set up a localised peritonitis, which recurs until the pseudo-cyst is emptied, and the lumen of the process finally and completely occluded. The attacks which form the third group of cases above described, which are closely associated with fecal retention, are also likely to be repeated unless the bowels are kept regularly and thoroughly opened. The liability to some accumulation taking place, even when there is a daily evacuation, is not to be forgotten. It is very common to find that the subjects of relapsing appendicitis have imperfect teeth or bolt their food.

DIAGNOSIS.—The condition known as appendicitis requires to be distinguished from several other intestinal affections. In ileo-cæcal invagination, which may give rise to many of the symptoms above described, the evidences of obstruction rather than of peritonitis predominate, the pain is more acute, and vomiting is usually more severe and persistent, while the stools contain bloody mucus; tenesmus also in these cases is much more marked. Other forms of acute intestinal obstruction may be mistaken for it, and often cannot be distinguished; the presence of distinct swelling, dulness, tenderness, and pain restricted to an area in the iliac fossa are of primary importance in determining the case to be appendicular. In appendicitis the temperature is raised, in acute obstruction it is usually lowered. Occasional cases of enteric fever, where there are constipation and absence of rash, may be confused with the malady under consideration, especially as the neighbourhood of the cæcum is the situation where the most distinct signs occur in both diseases; but the history, the course of the fever, and the characteristic tongue and general appearance of the patient in typhoid fever, together with the sequence of headache and delirium and splenic enlargement, should afford grounds for discrimination. Less often may tubercular peritonitis need to be separated, but here again the history, temperature, and coincidence of other evidences of tuberculosis elsewhere must be considered. Suppuration in connection with the ovary, or behind the peritoneum from caries of bone or perinephritic abscess, may simulate the fecal abscess following appendicitis.

PROGNOSIS.—Owing to the liability of even the mildest case to become severe, appendicitis should

always be regarded as serious and the prognosis as uncertain. The general mortality of all types taken collectively is probably 5 per cent., but while the simplest (group 3) are very rarely fatal, of the really fulminant cases at least 80 per cent. die; while among those cases in which suppuration occurs and an abscess is formed, a mortality of about 30 per cent. occurs.

The forecast in any single case is concerned with the immediate recovery, and the liability to relapse.

As regards recovery, this will obviously depend on the stage at which the case is seen and the previous duration of it, as well as the particular form which the malady has assumed. The general basis of prognosis in this respect may be inferred from the figures just given.

Much difference of opinion, due to absence of sufficient information, is held as to the likelihood of relapse in a case in which there has been but one attack. It is probable that a relapse occurs in 40 per cent. of the cases, the recurring symptoms exhibiting all degrees of severity from a more or less constant slight pain over the cæcal region with local tenderness, discomfort on much movement, perhaps occasional vomiting and constipation, to a definitely acute attack. Such a recurrence may be repeated again and again. It cannot be said that recurrent attacks are less dangerous than the first; patients have survived ten attacks and have died of the eleventh.

As to the necessity for operation very opposite views are held. While few would dispute the propriety of laparotomy where there is reason to suppose perforation has occurred or gangrene has set in, i.e. in the severest cases, unless the patient be actually moribund; or the need for opening the abdomen if suppuration has developed and an abscess has formed; much difference of opinion and of practice exists as to the proper treatment of the mild cases. At one extreme are those who would remove the appendix at the first evidence of trouble, at the other are those, relying on the great probability of recovery and taking the chances as to relapse, who would counsel reliance on the expectant treatment. Between such views it is difficult to take a middle course, and the practical question resolves itself into what are the indications of pus-formation which may occur in any case and should necessitate operation. Some help on this point may be gained from the general condition of the patient, which may be gradually getting worse, with possibly, but by no means necessarily, a further rise of temperature, perhaps a rigor; in some cases an increase in pain and tenderness, in others not so; a hurried respiration and a brown tongue with a tendency to restlessness and progressive sense of illness; an increase in the local swelling and area of dulness with increased tenderness. However suggestive such indications may be collectively, individually they have but little value, since pus may develop in the most opposite conditions of their occurrence. A more reliable sign is the rate and quality of the pulse. It may be simply affirmed that an increase in the frequency of the pulse beyond 120, or much above what it had hitherto been, points to suppuration, provided the increase be not due to any temporary excitement or disturbance.

Complications such as pyelephlebitis and pyæmia, or the supervention of pneumonia or other lung-complication; or the exceptional occurrence of uncontrollable diarrhoea and vomiting will necessarily add to the gravity of the prognosis.

The common causes of death are collapse, septic intoxication, the effects of suppuration, pneumonia, pyelophlebitis, pyæmia, and such accidents as hæmorrhage.

TREATMENT.—This must obviously differ very considerably according to the severity of the case, and may conveniently be considered according as operative interference is or is not called for.

When the disease is distinctly associated with fecal impaction, and when the symptoms are milder and somewhat more prolonged in duration, recovery is to be expected without operation, although, as stated, occasionally an abscess forms, which may require to be opened. For such patients as would be included within the third of the above-described groups, rest is essential, and the treatment consists in a judicious application of means to remove the contents of the cæcum, together with such an amount of sedative as will serve to keep the parts concerned as quiet as is consistent with the necessary evacuation. To meet the first indication enemata of plain water or soap and water are the most satisfactory. They should not be too large—fifteen to twenty ounces for an adult—and should be repeated, but not too frequently; it is rarely desirable for the enema to be given more than once in the day, and every other day is usually often enough. Much harm may be done by inducing a violent evacuation, though there may be a large quantity to be removed, and it is far better for this to be effected gradually. Some advise, in addition, frequent small doses—twenty grains—of sulphate of magnesium or sodium, not with a view of direct purgation, but to induce a moderate intestinal secretion, whereby the hardened feces may be somewhat softened. Anything in the nature of a violent aperient is strongly to be deprecated, and it is better to give occasional doses of opium, gr. $\frac{1}{4}$ to $\frac{1}{2}$, by the mouth, whereby pain is relieved and the risk of peritonitis appears to be diminished. For the same object, three or four leeches applied over the cæcum are most effective; or hot fomentations, either made with poppy-heads or applied after the surface of the abdomen has been painted with extract of belladonna and glycerine. The diet should be restricted in amount as much as possible; there is usually anorexia, and abstinence from food is beneficial; occasional teaspoonfuls of hot water or hot weak tea, and a few ounces of peptonised food are amply sufficient as a rule, and stimulants are very rarely needed. It cannot be too strongly insisted upon that the patient should be kept in bed, even for some days after complete cessation of all symptoms, as a very little exertion or slight excess of food may be sufficient to induce a relapse.

If the symptoms persist, if the temperature keep high and the pulse increase in frequency, and if the local phenomena become more prominent, then an incision over the swelling will be called for. It is seldom until after the fifth day that surgical interference is demanded. In the cases dealt with by the surgeon the dulness will have increased, the local tenderness will be more marked, and possibly redness of the skin and oedema may indicate the importance of immediate treatment.

The treatment of cases of the first group is identical with that followed in instances of acute perforative peritonitis. A prompt abdominal section carried out without a moment's delay offers practically the only prospect of saving life.

In dealing with cases of the second group, the general measures already described are employed. If the local and general symptoms persist, an incision will be called for. This, as already stated, is seldom indicated before the fifth day.

In cases of relapsing appendicitis the operation for the removal of the appendix is called for when the attacks are severe or are increasing in severity, when the attacks are becoming numerous, when the last attack has been of an alarming character, or when the repeated illnesses to which the patient is rendered liable are undermining his health, or interfering with the pursuit of his calling or his enjoyment of life.

In any case in which this operation is undertaken it may be assumed that the enlarged appendix can be made out after the acute symptoms have subsided. The removal of the offending appendix should be carried out during the period of quiescence, and after all the usual symptoms of appendicitis have disappeared.

It has been shown that the danger of the operation is less than that attending another attack.

W. H. ALLCHIN. FREDERICK TREVES.

APPETITE. Disorders of.—In disease the desire for food may be either *lessened* or *increased*; or the appetite may be *perverted*, and a longing for various substances unfitted for or incapable of digestion may be displayed.

Loss of appetite—*Anorexia* accompanies fevers, almost all forms of congestion of the mucous membrane, and acute or chronic gastritis; and as these affections constantly co-exist with other diseases, great variety as regards the desire for food is manifested in various complaints. In acute gastritis there is often not merely a loss of desire for, but a positive aversion to food, and the patient resolutely resists any attempt at obliging him to take either solid or liquid nourishment. In the more chronic forms of gastritis the distaste for food may be only slight; in some cases the appetite is increased, but is quickly satisfied as soon as a small quantity of food is taken. In chronic ulcer of the stomach the appetite, as a rule, remains good, and the patient is only prevented from indulging it by the fear of the pain that will result from so doing. Whenever the secreting structure of the organ is extensively diseased the appetite fails. Thus, in atrophy of the stomach the desire for food generally lessens along with the diminishing strength of the invalid. In cancer of the stomach there is always an extensive destruction of the glandular structure, and loss of appetite is a constant and prominent symptom. It must be remembered that a loss of appetite may be more apparent than real. The physician is constantly consulted on account of this symptom, when a little inquiry will show that the patient is really digesting as much as his system requires, but that by a habit of eating without allowing a proper interval between his meals, or by indulging in food of too nutritious a nature, or in an undue amount of alcoholic stimulants, hunger is prevented. For *Anorexia nervosa* see NEURASTHENIA.

Increase of appetite—*Bulimia* usually occurs where there is a necessity for an increased supply of food. Thus it is common after all febrile diseases, where the stomach has been long inactive. Again, in diabetes, where a large portion of the food is passed off in the form of sugar instead of being converted into the material required to keep

up the nutrition of the body, there is an unusually large appetite. A craving sensation is a common symptom in chronic catarrhal gastritis. It probably arises from the irritation set up by the mucus and fermenting substances long retained in the stomach, and is temporarily relieved by eating. The best treatment for such cases is to give alkalis about half an hour before the craving usually occurs, at the same time that the affection of the mucous membrane is combated by appropriate diet and remedies. In some persons the sensation of extreme hunger appears to arise from an irritable condition of the stomach, by which the food is passed into the duodenum before digestion is completed. The sensation is mostly complained of at night, and the writer has found it a good plan to let the patient have some beef-tea or meat-lozenges, for example, either just before retiring to rest or during the night. In children a craving for food is a frequent symptom, and arises either from the irritation of worms, or from chronic catarrh of the mucous membrane of the small intestines.

Perversion of appetite (Pica).—Is most common in pregnant or hysterical females. Articles, such as chalk, cinders, and slate-pencil, are sometimes swallowed. In the insane and in idiots articles of an indigestible nature, such as string, paper, cocoanut fibre, are not infrequently introduced into the stomach.

It is a matter of great importance that all persons, but especially dyspeptics, should accustom themselves to control their appetites. Whenever a larger amount of food is taken than the stomach is capable of digesting, the residue is apt to ferment and thereby to produce gastric catarrh. This is more especially the case where the digestive powers have been enfeebled by previous attacks of gastric inflammation. S. FENWICK.

APYREXIA (ἀ, priv.; and *πυρεσσω*, I am feverish).—Absence of fever. It is often used to denote the interval between two paroxysms of intermittent fever. See FEVER.

ARACHNITIS.—Inflammation of the arachnoid membrane. See MENINGES.

ARCAÇON, West Coast of France. Summer and winter resort. Part near sea frequented in summer. Winter resort on higher ground among pine-woods. Equable temperature. See CLIMATE, Treatment of Disease by.

ARCUS SENILIS.—SYNON.: Gerontoxon.

DESCRIPTION.—Arcus senilis is a crescentic or annular opacity just within the margin of the cornea, often seen in old persons. It usually begins as a greyish or whitish crescent at the upper part of the cornea, and subsequently a similar crescent makes its appearance in the lower part. These crescents gradually increase in opacity, in width, and in length, until their horns coalesce and form an *annulus* or ring. A true arcus is not quite continuous with the sclerotic, but is separated from it by a narrow rim of transparent cornea. The breadth of the crescent or ring generally ranges from one-twentieth to one-tenth of an inch. The opacity is more pronounced at its outer border, but fades insensibly towards the central portion of the cornea, which is of normal transparency.

ÆTIOLOGY AND PATHOLOGY.—The proximate causes of arcus are not known. Some families are

especially prone to it, and it most frequently occurs in persons of fifty years of age and upwards. The opacity is commonly believed to be due to fatty degeneration of the corpuscles and fibrillæ of the cornea, and to be indicative of degenerative changes within the heart and blood-vessels and other textures. There is, however, a growing consensus of opinion that its pathological as well as clinical significance has been overrated. Certainly it is, in itself, no criterion of age, either as regards years or constitution. On the one hand it may occur in young persons, and on the other hand it may be absent in extreme old age. Again, it may exist in persons free from any appreciable signs of arterial or cardiac disease, and it may be absent from those who have pronounced degeneration of the heart and blood-vessels. It is, nevertheless, true that arcus senilis is due to degenerative changes within the cornea, and that it does sometimes co-exist with atheroma or with fatty heart. According to some investigations made by the writer, in conjunction with Mr. E. T. Collins, in the laboratory of Moorfields Eye Hospital, the change in the cornea consists of the presence of fine highly refractive molecules, distributed along the course of the lymphatic spaces and channels of the superficial layers of the periphery of the cornea near the loop-endings of the capillaries of the conjunctival and episcleral blood-vessels. The greater portion of these molecules are not fatty, as is generally supposed; for, unlike fat, they are neither blackened by osmic acid nor dissolved by ether. They probably arise from mucoid degeneration of the protoplasm within the lymphatic channels and spaces of the cornea, and to some extent of the fibrillæ themselves. A few doubtful, blackened, fatty molecules may be seen here and there in sections stained by osmic acid. The fibrillæ are slightly wrinkled, and are more loosely held together than natural, and the spaces between the laminae are wider. In support of the non-fatty nature of arcus senilis it may be stated that wounds of the cornea, whether through the opacity, or to its inner side, or to its outer, and whether surgical or traumatic, heal in a natural way.

TREATMENT.—The condition is incurable, and is unaffected by any kind of treatment.

JOHN TWEEDY.

ARDOR (*ardor*, heat).—A sensation of heat, burning, or scalding. It may be felt either along the urethra during the passage of urine (*ardor urinae*), or in connection with the stomach (*ardor ventriculi*).

ARGYRIA (ἀργυρος, silver).—The slate-coloured stain of the skin produced by the internal use of the salts of silver. See PIGMENTARY DISEASES OF THE SKIN.

ARKANSAS SPRINGS, in Arkansas, United States.—Thermal waters. See MINERAL WATERS.

ARSENIC-HABIT.—See DRUG-HABITS.

ARSENIC, Poisoning by.—Arsenic is classed as a metallic irritant poison, though its action is by no means limited to that of an irritant. It acts specifically on the gastro-intestinal mucous membrane, whatever be the channel by which the poison gains access to the system. The most usual source of acute arsenical poisoning is the administration of

white arsenic or arsenious acid; but the sulphides, various arsenites, and impure commercial articles, such as dyes, wall-papers, articles of food, beer, and pigments, may be fertile sources of arsenical poisoning. Poisoning by arsenic may be either *acute* or *chronic*.

ANATOMICAL CHARACTERS.—These are the same by whatever channel the poison has gained access to the system. There is marked petechial inflammation of the stomach and duodenum, usually of the small and large intestines also; but not uncommonly the inflammation is limited to the stomach, duodenum, and rectum, the intervening alimentary tract having escaped. If the poison has been administered in a solid form, white patches of the arsenical compound may be found embedded in yellow or bloody mucus and inflammatory exudation. Portions of the white arsenic are also sometimes converted by the sulphuretted hydrogen evolved during decomposition into the yellow sulphide. Ulceration of the stomach is rare. Neuritis may be observed. An echymosed condition of the heart is often noticed; and fatty degeneration of the liver, as in poisoning by phosphorus, has been described.

A. Acute Arsenical Poisoning.—This is the usual form of poisoning ensuing on the nefarious administration of any preparation of arsenic, but usually the oxide (*arsenious acid*) is employed.

SYMPTOMS.—The symptoms do not, as in the case of corrosive poisoning, come on immediately after the administration of the poison. There is most commonly an interval of half an hour or an hour between the swallowing of the agent and the onset of prominent symptoms. The quantity of the noxious agent, and its state as regards solubility, have also an obvious relation to the commencement of symptoms. Most commonly, after a sense of faintness and depression, intense burning pain is felt in the epigastric region, accompanied by tenderness on pressure. Nausea and vomiting quickly supervene, increased by every act of swallowing. Unlike what occurs in an ordinary bilious attack, with which arsenical poisoning may be at first confounded, the pain and sickness are not relieved by the act of vomiting. The vomited matters are extremely varied, and present no characteristic appearances. At first they usually consist of the ordinary contents of the stomach, but at a later stage are largely charged with bile which has regurgitated into the stomach in consequence of the violence of prolonged emesis; and they may be tinged with blood. In most cases vomiting is speedily followed by violent purging, and great straining at stool, the motions being often streaked with blood. Purging may, however, be entirely absent. Other prominent symptoms are great thirst, a feeble irregular pulse, and cold clammy skin. The patient, as a rule, dies within eighteen to seventy-two hours, in a state of collapse; but tetanic convulsions are not uncommon, and even coma and paralysis may close the scene.

DIAGNOSIS.—From an ordinary bilious attack, induced by improper diet or by decomposing food, arsenical poisoning is diagnosed by the persistence of the symptoms after the removal of the apparent cause; and not infrequently by the symptoms remitting and again supervening on the administration of food or drink of a particular kind, or given by a particular person. From choleraic diarrhoea it is distinguished by the *sudden* onset of symptoms, thirty to sixty minutes after food or drink has been

taken; by the absence of rice-water stools, or of lividity of the skin; and by the symptoms not yielding to treatment. Moreover, in poisoning by arsenic, there is usually greater tenderness over the epigastrium; the diarrhoea is less passive, and accompanied with more tenesmus than in choleraic diarrhoea; the stools are more often bloody; and nervous symptoms may be more pronounced. The diagnosis is, however, often very difficult, except when aided by a chemical analysis of the matters ejected from the stomach or of the excreta, which should always be made in doubtful cases.

PROGNOSIS.—This must always be uncertain, since it is rarely possible to ascertain the quantity of poison taken, or to ensure its entire evacuation from the stomach.

TREATMENT.—Emetics, diluents, and demulcents are the appropriate remedies. The stomach-pump may also be usefully employed. In administering emetics, tartar emetic should be avoided, as it increases the depression, and its presence complicates a chemical analysis. Moreover, tartar emetic sometimes contains traces of arsenic, and, in the event of an analysis being made, an unfounded suspicion may be raised. No confidence can be placed in the so-called antidote, *ferric hydrate*, but dialysed iron may be freely given.

B. Chronic Arsenical Poisoning.—This form of poisoning is not uncommon, and is, unlike the acute form, generally accidental. The inhalation of arsenical vapours in factories, or of arsenical dust, as from green and other wall-papers, and in the process of manufacturing artificial flowers, is a common source of chronic arsenical poisoning.

Those who are chiefly exposed to this form of poisoning are persons employed in the manufacture of pigments, especially green pigments; paper-hangers and decorators; manufacturers of artificial flowers; milliners; persons exposed to the fumes of heated metals, particularly zinc and brass; manufacturers of dyes; and leather-dressers (*see OCCUPATION-DISEASES*). In the process of depilating sheep-skins, previous to the tanning or the tawing process, a mixture of lime and orpiment (*sulphide of arsenic*) is used; and serious ulceration of the hands, scrotum, nose, and cheeks not infrequently results. Persons living in rooms the walls of which are covered with arsenical paper, especially bright green papers containing arsenite of copper, are liable to suffer from chronic arsenical poisoning. It is uncertain whether this is entirely caused by the mechanical transfer of pigmentary dust to the air-passages, or is partly due to volatilisation of the arsenic, probably in the form of arseniuretted hydrogen. Many brown wall-papers also contain arsenic, and arsenious acid is sometimes added to the size; such papers have been known to produce the specific symptoms of arsenical poisoning.

That some persons can take arsenious acid internally with impunity in relatively large doses (arsenic-eating) is now a well-established fact.

SYMPTOMS.—The first symptoms of chronic arsenical poisoning are usually loss of appetite, precordial pains, irritability of the bowels, and occasionally headache. Suffusion of the eyes, a peculiar and characteristic appearance of the conjunctiva, often amounting to actual conjunctivitis, puffiness beneath the eyelids, and intolerance of light are early manifested. The muscular power of the limbs is impaired pretty constantly, and actual paralysis, extending upwards from the lower extremities, is

occasionally observed (*see* PARALYSIS, TOXIC). A brownish or sepia pigmentation of the skin of the face, neck, axillæ and abdomen, and thickening of the horny layers in the palmar and plantar regions, may be noticed. Males who handle arsenical preparations are liable to ulceration of the scrotum and penis, obviously due to a mechanical transference of the poison to the genitals when these are touched. Herpes and other affections of the skin are observed. Multiple neuritis is common. If the source of the disease be not removed, progressive emaciation, exfoliation of the cuticle, and nervous prostration supervene; and convulsions may precede the fatal termination. The effects of green arsenical pigments are sometimes manifested by bleeding from the nose.

DIAGNOSIS.—When a patient suffers more or less from the symptoms above described, and is also known to be exposed to any of the sources of danger from arsenical poisoning enumerated above, the diagnosis is not difficult.

TREATMENT.—The source of poisoning should invariably be removed. It is found that those who suffer from working in arsenic make no progress towards recovery until they are removed from contact with the poison. Wall-papers which contain arsenic, and are suspected to be the cause of symptoms, should be taken away. Quinine, or other tonics, iron, and attention to the digestive organs will be needed. Removal to fresh country air is often productive of marked benefit. Soothing lotions to the skin, and careful attention to eroding ulcers, especially of the cheek, may be necessary. Shampooing and warm baths form the best treatment for paralytic lesions.

T. STEVENSON.

ARTERIES. Diseases of.—I. **Arterio-Sclerosis.**—This disease will be described as consisting of two varieties, (1) *General or diffuse arterio-sclerosis*, and (2) *Local or nodose arterio-sclerosis*.

1. **General or Diffuse Arterio-sclerosis.** In this condition there is an increased rigidity of the arteries, due to a thickening of their walls involving mainly the inner coat. The process may be either generalised or limited to certain arteries, may affect the large vessels only, or leave them intact and select the smaller arteries. In its more advanced stage it is often termed *atheroma*, or *endarteritis deformans*.

ÆTIOLOGY.—Arterio-sclerosis is a physiological process in old age, but it may also occur in young adults, without any obvious direct cause, when there is an inherited tendency to disease of the arteries. Of the direct causes the most important is probably long-continued muscular strain, especially when associated with unfavourable conditions, such as exposure or insufficient food. Gout and articular rheumatism are very common forerunners of the disease, as are also certain infective and toxic processes, especially syphilis (congenital or acquired), scarlet fever, influenza, endocarditis, typhoid fever, and malaria. It may be produced by the action of alcohol, lead, or phosphorus, and in many cases is the result of auto-intoxication from habitual over-eating. It also occurs as a constant feature in chronic interstitial nephritis. As regards *sex*, men are as a rule more affected by arterio-sclerosis than women owing to their greater exposure to its causes; but since women become more subject to the disease after the climacteric, the liability of the two sexes tends to become equalised after fifty.

PATHOLOGY.—Sclerosis of the smaller arteries is

most generally diffuse in its distribution, but tends to involve specially the arteries of the brain, kidney, and heart, and of the distal parts of the upper and lower extremities. The distribution of the lesion is seldom uniform, but affects the various areas in different degree according to peculiarities of individual constitution or of the ætiological factors.

The sclerosis of the artery affects mainly the inner coat, which becomes thickened by the formation of dense layers of fibrous tissue owing to the proliferation of the sub-endothelial connective-tissue cells. This thickening is never uniform, and is generally greater at one side of the vessel—that which is exposed to the greatest strain. The elastic lamina becomes granular and its continuity may be interrupted. The condition of the media varies considerably. Where there has been high arterial tension, as in chronic interstitial nephritis, the muscular fibres may be hypertrophied; indeed, this may be the main source of the thickening of the artery. In other cases it may be the seat of hyaline, fatty, or even calcareous degeneration, and may show patches of nuclear proliferation, especially around capillaries which have entered the coat. The tunica adventitia may be normal or show some thickening, usually slight in degree. The affected vessels increase in length as well as in thickness, so that they become tortuous, as can be well seen in the temporal and radial arteries. When palpated the artery feels resistant and cord-like, rolling under the finger in a characteristic manner. The amplitude of the pulse-wave is diminished.

The mode of production of this process is still *sub judice*. In the opinion of Thoma, which is entitled to great weight, the first step in the process is a diminution in the contractility of the muscular wall of the arteries, as a result of long-continued overstrain or of the direct action on it of some of the toxic substances already mentioned. The affected arteries become dilated, and to compensate for this the inner coat becomes thickened. In consequence of the new-formation of fibrous tissue, the artery becomes more rigid and thus offers greater resistance to the blood-stream. Two sets of effects arise directly from this. The heart hypertrophies to overcome the increased resistance, and this hypertrophy causes an increased strain on the arterial walls leading to their further sclerosis, which in its turn is productive of further hypertrophy of the heart. So long as the strain is not excessive and nutrition is well maintained, the hypertrophy and sclerosis may balance each other, but a time generally arrives when degenerative changes appear in the arteries, in the heart, or in both together. In the arteries, the changes appear as a partial hyaline degeneration, with the subsequent formation of fatty and calcareous deposits (*atheroma*), so that eventually yellowish patches are visible on the vessel walls. The calibre of the artery loses its uniformity, and may vary very greatly, while the rigidity of the artery causes it to gape when transversely divided. Subsequently the endothelium may become eroded, and the contents of the atheromatous patch may be discharged into the circulation, leaving a rough surface on which thrombi may be deposited, thus leading possibly to the occlusion of the vessel (for other changes which may occur *see* ANEURYSM, and BRAIN, HÆMORRHAGE INTO). The degenerative changes which appear in the heart may be due to exhaustion from the continued over-strain produced by the increased peripheral resistance, from

participation in the general toxic process, or from sclerosis of the coronary arteries. The compensatory hypertrophy fails, dilatation increases, and mitral incompetence ensues, with the usual signs of failure of compensation. Anginous and syncopal attacks may occur, especially when the coronary arteries are affected, and there is increasing breathlessness on exertion. Pathologically the muscle of the heart may show brown atrophy or fibroid or fatty changes, and when the branches of the coronary arteries become narrowed or impervious, localised areas of softening appear. Rupture of the heart may occur spontaneously or after sudden strain. These morbid conditions of the heart and vessels lead to evident general changes. There is anemia and lowering of the nutrition generally, shown either by emaciation and atrophy (as in the senile form), or by flabbiness in more obese patients. Digestion is impaired, and the appetite becomes capricious. The loss of vaso-motor control is the cause of attacks of vertigo, or even syncope, especially on changing position suddenly, and epileptiform seizures may occur.

As the disease progresses mental phenomena may appear, such as irritability, a feeling of torpor, and incapacity for clear, connected thought; and at intervals there may be temporary attacks of absence of mind without loss of consciousness, in which external circumstances pass unnoticed. This has been named lately the 'temporary aberration' of elderly people. Its practical significance as regards certain occupations demanding continuous attention is very obvious. As a rule there is marked insomnia. Headache, migrainous in some cases, is common, and tingling or numbness may be felt in the extremities. Cramp is often troublesome, especially at night, occurring either spontaneously, or after some unusual muscular fatigue. When the arteries are gravely affected and the heart weak, these sensory symptoms may be the precursors of gangrene, due to the occlusion of the vessels by thrombi and the failure of the collateral circulation. This occurs most readily in the senile form of arterio-sclerosis. Hemorrhages may also occur readily from rupture of the arterial walls, more especially of the cerebral vessels. Pain, most marked in the extremities, is often present, and is generally regarded as rheumatic in origin. Thoma terms it 'angio-neurotic' pain and points out that it is probably due to affection of the nerve-endings in the arterial walls.

The pathological permeability of the capillaries leads to localised serous accumulations from hypostasis. The oedema varies according to position, being present in the feet at night and in the eyelids in the morning. Serous effusions into the closed cavities also occur readily, more especially in the pleural sac, and there is also a marked tendency to cerebral oedema. Polyuria is usually present, similar to that which occurs in chronic interstitial nephritis, and sometimes due to pathological changes in the kidney, in addition to the general vascular changes. Frequency of micturition at night is one of the characteristic features of sclerosis of the renal arteries. The urine has a low specific gravity and there may be albuminuria, often transient. Pulmonary changes are shown by the ready occurrence of bronchitis and asthma.

The general impairment of nutrition and the changes in the vessels lessen the normal reaction to inflammatory lesions, and for this reason in cases of

arterio-sclerosis the danger of acute inflammations, such as pneumonia, and of surgical operations is greatly increased.

The physical signs in the circulatory system are characteristic. The left heart may be hypertrophied or dilated, its apex being displaced downwards and outwards. The sounds over the mitral area are normal; or, in later stages, there may be a systolic murmur. In the aortic area the second sound is accentuated. The radial artery is thickened and may be incompressible. The pulse-wave is smaller than in health. The sphygmographic tracing shows a slow rise, a rounded top, and a delayed and small dirotic wave.

TREATMENT.—When fully established the disease is incurable and tends to be progressive, but much may be done to arrest or delay its course and to ameliorate the symptoms, especially if treatment is begun early, and if the cause is removable, as is the case with alcoholism and plumbism. Preventive treatment should be adopted in members of families in which there is a tendency to arterial disease, or to gout or rheumatism. Such individuals should be warned as to their special liability to vascular weakness, and their lives should be regulated as far as possible in accordance with the principles of the general treatment advised below.

The general treatment is of the first importance in dealing with arterio-sclerosis, and consists chiefly in a very careful regulation of the daily life in relation to exercise, occupation, and diet. The exercise should be moderate in amount, in due proportion to the powers of the individual, always stopping short of fatigue, and should be taken regularly, since moderate exercise tends to lower the vascular tension. Emphasis should be laid on the necessity for avoiding any sudden violent muscular exertion, or any prolonged strain even when moderate. Especially must the subjects of arterial disease be warned not to run to catch a train or omnibus, or to make any rapid ascent. Sudden mental or emotional shock, or long-continued worry should be also guarded against. When feasible a quiet existence involving a fair amount of light outdoor employment should be advised. The dietary should be simple and should consist of three light meals a day with an interval of four or five hours between them. Sweet wines and malt-liquors must be forbidden, but where some stimulant is necessary, well-diluted whisky may be taken with the solid food. As a general rule it is advisable to take the requisite amount of fluid between rather than during meals. The skin should be kept acting freely by the use of warm clothing and of frequent baths. A daily evacuation of the bowels is necessary. The place of residence should be in an equable and moderately warm climate and at a low altitude. The subjects of arterial disease should never ascend to any great height.

When the vascular changes are fully established two main indications as regards treatment arise:—viz. the moderation of the peripheral tension and the nutrition of the hypertrophied heart. To obtain the first the iodides and the nitrites may be used, and such cardiac tonics as arsenic and strychnine in small doses may be employed to act directly on the cardiac muscle, and also on the general nutrition. Any tendency to dyspepsia must be controlled by the usual means, and occasional free purgation obtained by the use of a mercurial followed by a saline draught. In late stages of the disease, when

the heart-muscle weakens and compensation fails, cardiac tonics such as digitalis and strophanthus must be resorted to, but these should always be used in combination with the iodides or the nitrites in order that the peripheral tension may be lessened at the same time.

These different details of treatment are often carried out most easily during residence at a watering place, the choice of which must depend upon the special features of each case.

The various complications, such as angina pectoris, asthma, gangrene, insomnia, must be treated as they occur.

2. Localised or Nodose Arterio-sclerosis may be found in the larger or the smaller arteries. It may exist alone or may be combined with the diffuse form. It is specially met with in the arteries which are subjected to the highest blood-pressure, such as the aorta, the arteries of the lower extremities, of the brain and, in obstructive mitral disease, of the lung. In the aorta, in the early stages of the disease, the patches appear as irregular, slightly raised areas, of the consistence and colour of hyaline cartilage. Later, when degeneration takes place within them, the patches become yellow and opaque in the centre. In what may be termed their final stages, they show calcareous plates of various sizes. There is always a certain amount of irregular dilatation of the affected vessel, especially in the case of the aorta, hence the name often given to it of *arteritis deformans*. The process seems, according to the researches of Thoma and his pupils, to be secondary to localised weakening of the middle coat of the artery. This weakening leads to a local yielding of the coat, which bulges outwards. The proliferation of the intima and of cells which penetrate into it along with the vasa vasorum which grow in the adventitia, produces the dense laminated fibrous tissue found in the first stage. This must be looked upon as a process compensating for the weakening of the media. In course of time the continued stretching of this new tissue affects its nutrition, so that there follows hyaline, then fatty, and finally calcareous degeneration, especially in the deeper parts of the thickened intima. The change, however rare, remains limited to the intima. The weakened media may also show a proliferation round the vessels which penetrate into it from the outer coat, and it may in its turn become the seat of fatty and calcareous degeneration.

The consequence of sclerosis of the larger vessels, such as the aorta, will be referred to under the sections on AORTA, Dilatation of the, and ANEURYSM.

II. Syphilitic Arteritis. 1.—**SYPHILITIC ENDARTERITIS.**—This is in most cases a chronic affection of the intima, differing from arterio-sclerosis in two important respects. It does not undergo fatty or calcareous degeneration, although it may occasionally show a tendency to caseation; and it tends to narrow or to obliterate the lumen—hence the name *obliterative endarteritis* often given to it. Such obliteration does not occur in ordinary sclerosis unless a thrombus forms within the vessel and undergoes organisation. Syphilitic endarteritis affects mainly the arteries of the brain, the spinal cord, and the heart, but it does not altogether spare the arteries of the extremities. It is as yet undetermined whether it may occur in the aorta, although the relation of aneurysm to sclerosis of the aorta and to syphilis suggests this possibility very strongly.

In the brain the anterior and middle cerebral arteries in their cortical and internal branches appear to be most frequently affected; but no artery within the cranium is altogether immune. The affected portions of the arteries appear greyish-white, and finally resemble firm greyish threads or solid cords (*see* BRAIN, Syphilis of; Tumours of). Microscopically, there is an irregular, often nodular thickening of the intima, which at first is loose in texture and composed of spindle or stellate cells, and later becomes more condensed and fibrous. The elastic lamina is often granular and may appear double. The middle coat may appear normal or may be more or less infiltrated with round cells. The outer coat also may show similar infiltration. The vasa vasorum may penetrate through the media into the thickened inner coat and even into any thrombi forming in the lumen. The symptoms of syphilitic endarteritis are mostly those of ischaemia of the organ affected. If the coronary arteries are involved there may be the sign of heart-weakness and even rupture. Endarteritis of the cerebral arteries may cause transient or permanent disorders of speech, paresis or paralysis, confusion of thought, impairment of memory, and insomnia. If the spinal arteries are attacked the symptoms of myelitis will be produced. If the arteries of the extremities are involved they may be felt as solid cords; the distal parts may be cold and livid; and there may be even limited gangrene of parts of the fingers or toes. In estimating the possibility of recovery of function when the brain or spinal cord is involved, one must remember that a portion of the nerve-tissue may have undergone permanent softening before treatment is begun, but that, on the other hand, the symptoms may be due merely to a temporary disturbance of the circulation. The tendency to relapse in all cases must also be borne in mind. Antisyphilitic treatment must be vigorously pushed and continued till long after the symptoms have disappeared or become stationary.

2. **SYPHILITIC PERIARTERITIS** may occur independently of any endarteritis or may be associated with it. The process affects mainly the arteries of the brain, viz. those in the cerebellar cortex, those of the medulla and pons, as well as the arteries of the interior of the cerebral hemispheres and of the nuclei of the cranial nerves. It occurs in three forms, a diffuse, a nodular, and a gummatous caseating form. Of these the first-named tends to appear the soonest after the original infection, sometimes as early as six or nine months. It leads to a diffuse infiltration of the adventitia and of the perivascular lymph-space with round cells, a similar but slighter infiltration of the middle and inner coats, and a tendency to early thrombosis with consequent softening of the part supplied. The nodular form produces minute excrescences on the wall of the artery, about the size of a pin's head and resembling in miniature the gnarling seen on old oak trees. These are also composed of round and plasma-cells, with a slight tendency to caseation. This form may be combined with the diffuse variety. In the caseating form, which represents the most chronic variety, the nodules are larger, reaching the size of split peas and showing distinct caseation. They appear later in the history of the disease than either of the previous varieties. Periarthritis may be suspected if cerebral symptoms appear within a short period of syphilitic infection, and calls for very vigorous and persistent anti-syphilitic treatment.

III. Nodose Periarteritis.—There is a form of nodose periarteritis, due to some as yet undiscovered probably infective cause, which runs a rapid course and is accompanied by wasting and enlargement of the spleen. This form presents both macroscopic and microscopic appearances very similar to those found in syphilitic periarteritis. It tends to produce thrombosis, but it differs from the syphilitic form in the frequent occurrence of aneurysmal dilatations of the weakened vessel-walls. The affection seems to spare the cerebral arteries and to involve those of the muscles and of the various thoracic and abdominal viscera, which are studded with small whitish nodules.

IV. Acute Arteritis.—This may arise from infection of an artery by pyogenic organisms which gain access to its walls either from the blood-stream or from the surrounding tissues. The aorta immediately above the semilunar valves is not infrequently, during the course of an ulcerative endocarditis, injured by the impact of a portion of vegetation on one of its cusps, which may be driven against the intima by the force of the blood-current. Micro-organisms derived from the vegetation attack the intima and give rise to a verrucose endarteritis similar to that already present on the valves. Then may follow necrosis and softening of the wall with the formation of an acute aneurysm which may rupture into the pericardial sac or between the coats of the artery. Again, if a portion of vegetation containing septic organisms be transported to and impacted in a distant artery, it will cause suppurative necrosis of the wall of the artery with the formation either of an abscess or of an embolic aneurysm which readily ruptures. The arterial coats may also be attacked from without by an inflammatory process in the tissues. In such cases a thrombus is always formed; this will be simple or puriform according to the nature of the causal condition. It is well to note that a bright or brownish-red staining of the intima so frequently seen *post mortem* is not an indication of arteritis, but is due to staining by hæmatin liberated from the blood-corpuscles after death. The symptoms of an arteritis are always obscured by those of the general condition which produce it. It is doubtful if the symptoms assigned to acute arteritis, viz. acute dilatation of the aorta, a systolic aortic murmur, dyspnoea, and cough, are really due to the process in question.

V. Fatty degeneration of the arteries may be present apart from atheroma, and may occur as a senile process, in anemia or in marasmus, or may be found in certain individuals without any obvious cause. The process appears in the intima, forming opaque, velvety, yellowish areas, which tend to become eroded, so that a portion of the intima is destroyed. The muscular coat, which is usually fatty also, may yield to the localised strain and rupture, the result being either the formation of a dissecting aneurysm, or, if the outer coat yields, rupture of the artery. These grave conditions are, however, seldom met with as a result of uncomplicated fatty degeneration.

VI. Calcareous degeneration of the arteries, unassociated with atheroma, is rare. It is almost exclusively a senile change, but has been known to occur in young subjects. It usually attacks the middle coat of the arteries, and affects mainly those of medium size, especially in the brain and the extremities. The arteries may become converted into rigid tubes from the deposit of calcareous salts

between the muscular fibres. The effect of the rigidity of the vessels on the nutrition of the heart and of the tissues is practically identical with that produced by advanced arterio-sclerosis.

VII. Amyloid degeneration of arteries.—*See* AMYLOID DEGENERATION.

VIII. Hyaline degeneration of arteries.—*See* DEGENERATION.

ALEXANDER BRUCE.

ARTERIES, Examination of.—*See* PHYSICAL EXAMINATION; and PULSE.

ARTHRALGIA (ἄρθρον, a joint; and ἄλγος, pain). Pain in a joint. The term is more particularly applied to articular pain in the absence of objective disease. *See* JOINTS, Diseases of.

ARTHRITIS (ἄρθρον, a joint).—A term generally used to signify any disease whatever involving a joint, but more correctly confined to articular inflammation. It is also employed to designate inflammation of *all* the structures forming a joint, as distinguished from mere synovitis. *See* JOINTS, Diseases of.

ARTHRODYNIA (ἄρθρον, a joint; and ὀδύνη, pain).—Pain in a joint. *See* ARTHRALGIA.

ARTICULAR RHEUMATISM.—Rheumatism affecting joints. *See* RHEUMATISM, ACUTE.

ARTIFICIAL RESPIRATION.—The method of exciting and keeping up the movements of the chest, so as to supply air to the lungs, is a subject of the highest importance, since the hopes of recovery depend on its due performance in many cases of narcotic poisoning, in the apparently drowned or asphyxiated, and in the collapse of the advanced stage of the condition induced by anesthetics.

PRECAUTIONS.—For its effective employment it is essential to see that no foreign body obstructs the air-passages. Children and old people are liable to swallow large pieces of meat or crust, which become impacted in the pharynx or œsophagus. In operations upon the nasopharynx portions of new-growth or clots of blood may occlude the rima glottidis; even in Dental Surgery under Nitrous Oxide the possibility of broken instruments, broken props, and teeth, more especially wisdom-teeth and bicusps, becoming impacted in the larynx should never be overlooked.

Artificial dentures may become dislodged and wedged in the pharynx, and vomited material may gain entrance to the larynx. In all cases therefore of suspended respiration the mouth should be opened and the finger swept round the oral cavity and pharynx, and any foreign body dislodged before commencing artificial respiration; should, however, a foreign body be detected which cannot be at once removed, then immediate laryngotomy followed by artificial respiration is the only treatment likely to be followed by success. Tracheotomy is rarely necessary in an uncomplicated case. A knife-handle held between the molar teeth is a ready and useful gag to keep the mouth open. A button-hook, in the absence of pharyngeal forceps, is sometimes very serviceable. Vomited matter should be quickly removed with a sponge or cloth twisted round a

piece of wood or more readily with the hooked index finger. For the special treatment of persons drowned see DROWNING, Death by.

METHODS.—The simplest method of commencing artificial respiration is to compress the chest and abdomen simultaneously, then remove pressure so as to allow air to enter the chest, and again repeat the pressure every two or three seconds. If the sound indicates that air is passing into and out of the lungs, this method may be continued until spontaneous breathing returns; but if we are not sure that the air is exchanged, and in all cases if the patient's condition is not decidedly improved in half a minute, we should resort to one or other of the following methods:—

1. *Howard's method.*—This method is so important that it is considered separately under RESUSCITATION.

2. *Sylvester's method.*—Place the patient on his back on the floor, with a block or pillow under his shoulders in order that the head may become partially extended; then kneel at the patient's head and grasp the arms above the elbows; press them first well into the sides in order to expel air, then drag them outwards and upwards over the head until they are in a line with the trunk, and keep them in this position so long as air enters the chest. Some arrangement is needed to prevent the body from being dragged towards the operator. For this purpose the plan of raising the chest on a high cushion or box has been adopted, but as a condition of cardiac anemia is often present, this is objectionable. It is better to effect the object by placing a book in front of the thighs while kneeling at the head of the patient. It may be needful to draw forward the tongue; but generally if the head falls back over a cushion placed behind the neck, this is not required. An artery-forceps, or a noose of string, or a handkerchief will enable an assistant to keep the tongue well forward.

As soon as the sound produced by the entrance of air into the chest ceases, the arms should be brought down to the side and a little towards the front of the chest, and pressed firmly and steadily against it for about one second after air is heard escaping. In cases of drowning it is enough to repeat this operation every four seconds, but in the collapse resulting from chloroform or other anæsthetic, the necessity for getting the vapour quickly out of the chest justifies a more rapid performance of the movements during the first five minutes. After this time the movements should be carried on more slowly, but they should be continued for half an hour at least, and even longer if the warmth of the surface and diminution of lividity give any reason to hope that the heart has not entirely ceased to act.

3. *Marshall Hall's ready method.*—This is performed by placing the body on its face, the thorax being supported upon a roll of clothing, cushion, or pillow; firm pressure is made on the back to produce expiration, and then the body is rolled on to its side to produce inspiration; this alternate rolling from the face to the side should be repeated about fifteen times a minute. It is recommended in cases of drowning, as fluids can drain away from the mouth and so clear the upper lung. The plan is not nearly so effective as Sylvester's, but if no assistant is at hand it is the best mode of artificial breathing that can be adopted.

4. *Mouth-to-mouth insufflation.*—This method is not to be depended upon, because of the difficulty

both of keeping the larynx open, and also of preventing the air going down the gullet.

The administration of *oxygen* is indicated in most cases of artificial respiration where the cyanosis is deep, but unfortunately it is not readily attainable. Now that the gas can be had in a compressed state, and can be given by means of a tube or an inhaler, it will probably be more extensively utilised; but it is certain that in all cases of impending asphyxia, time is of so much importance that anything which would delay the supply of oxygen would not be compensated for by giving it pure, instead of in the form of common air. Tracheotomy is not to be thought of in the first instance in any case in which air can be made to pass, even in very small quantity, through the trachea.

For supplemental and after-treatment, see RESUSCITATION.

C. CARTER BRAINE.

ASBESTOS-WORKERS.—See OCCUPATIONAL DISEASES.

ASCARIDES.—See ENTOZOA.

ASCITES (ἄσκός, a leathern sac: a large belly).
SYNON.: Dropsy of the peritoneum; *Hydrops peritonei vel abdominis*; *Hydroperitoneum*. Fr. *Ascite*; Ger. *Die Bauchwassersucht*.

DEFINITION.—An accumulation of fluid within the cavity of the peritoneum more or less serous in character.

ÆTIOLOGY AND PATHOLOGY.—Ascites occurs in the course of many different diseases. It may be due to any of the following causes:—

I. Direct mechanical obstruction affecting the portal circulation.

1. Obstruction of the trunk of the portal vein before it enters the liver, either from external pressure or an internal block.

2. Pressure upon or obliteration of the branches of the vein within the liver.

3. Pressure upon the hepatic veins, or upon the inferior vena cava after it receives these veins.

II. Cardiac or pulmonary diseases obstructing the general venous circulation.

III. Disease of the kidneys.

IV. Morbid conditions of the peritoneum.

V. Miscellaneous.

I. Any direct obstruction interfering with the portal circulation must necessarily lead to congestion and over-distension of its tributaries, one of the consequences of which is excessive transudation of fluid into the peritoneal cavity, while absorption is checked. See DROPSY.

1. The portal trunk may be pressed upon, as it lies in the fissure, by prominences from the liver itself, enlarged glands in its vicinity, a neighbouring tumour (as cancer of the pancreas, or a growth in the small omentum), a hepatic aneurysm, or inflammatory thickening resulting from peri-hepatitis. The pressure may absolutely close up the vessel, but it more commonly causes a local clot to form, and thus its channel is blocked. A thrombus is also, in exceptional instances, produced in connection with a diseased condition of the portal vein, such as inflammation or calcification; obstruction to the circulation within the liver; or feebleness of the circulation, with an abnormal tendency to coagulation of the blood.

2. Pressure upon, or obliteration of, the branches

of the portal vein within the liver can only arise as a consequence of some morbid condition involving the actual substance of this organ. The hepatic disease which most commonly leads to this result, and which is one of the most frequent causes of ascites, is cirrhosis of the liver. Occasionally it accompanies syphilitic and other forms of contracted and indurated liver, or it may be associated with infiltrating cancer. Occasionally a mass within the liver obstructs a considerable branch of the portal vein.

3. Obstruction of the hepatic veins, or the upper end of the inferior vena cava, is a rare event, but may arise from the pressure of a growth connected with the liver itself, or of some neighbouring tumour.

II. Diseases of the lungs or heart, or mediastino-pericarditis, which impede the general venous circulation, may thus lead to ascites. Usually, however, in cases of this kind, the legs are the seat of considerable anasarca before peritoneal dropsy is observed. In course of time the passive congestion causes structural changes in the liver, so that ultimately the ascites may become the most prominent manifestation of the general dropsy.

III. Ascites may constitute a part of the dropsy which so often accompanies renal disease. The amount of intra-peritoneal fluid is, in such cases, generally small.

IV. Effusion into the peritoneal cavity is a usual result of peritonitis, and sometimes occurs so gradually that its inflammatory origin may be at first unsuspected. Moreover, true ascites is observed occasionally as a sequel of peritonitis in consequence of the adhesions or other causes of passive congestion which may occur. Both tuberculosis and malignant disease of the peritoneum may cause ascites in this way.

V. Among the chief *miscellaneous* causes to which ascites has, without much evidence, been attributed are: exposure to cold or wet; the sudden suppression of habitual discharges, or the rapid cure of chronic cutaneous affections; and extreme anæmia and debility. Fluid may collect within the peritoneum as the result of the rupture of a cyst within the abdomen, especially an ovarian cyst.

It must be remembered that ascites may be due to a combination of two or more of the foregoing causes. For instance, portal cirrhosis of the liver may co-exist with mitral incompetence and cardiac failure.

ANATOMICAL CHARACTERS.—The amount of fluid within the peritoneal sac may range from a few ounces to some gallons. Ordinary serous ascitic fluid is generally thin, limpid, and watery in consistence; colourless or slightly yellow; clear and transparent; and of alkaline reaction. In exceptional instances, however, it may be coloured by blood or bile; or more or less turbid and dirty-looking or milky (chylous); or of unusually thick and somewhat gelatinous consistence. Soft fibrinous masses occasionally float in the fluid, or these may form spontaneously when it is allowed to stand. Very rarely the reaction is neutral or acid. The specific gravity varies considerably. Chemically, the fluid contains the same proportion of saline constituents, but a larger proportion of albumen than dropsical fluid from the legs. Occasionally it contains fibrin, cholesterol, bile-elements, or, in cases of renal dropsy, urea.

The fluid will distend the abdominal wall, push up the diaphragm, and compress the rest of the

abdominal contents. There will also be the signs of the morbid condition upon which the ascites depends; and there may also be indications of changes resulting from the long-continued pressure of the fluid.

SYMPTOMS AND SIGNS.—Ascites usually sets in very gradually and advances steadily. Occasionally, however, the fluid collects with considerable rapidity. The clinical phenomena associated with this pathological condition may conveniently be considered under the following heads, namely: 1. *Physical signs.* 2. *Mechanical effects of the dropsical accumulation.* 3. *General symptoms.*

1. *Physical signs.*—(a) If fluid collects in the peritoneum in any quantity, the abdomen presents more or less general enlargement. This is often the first change which attracts the patient's attention, and it may also have been noticed that the increase in size commenced below. The enlargement may be so extreme that the skin is tightly stretched and thinned, presenting a smooth and shining appearance, or sometimes white lines—*lineæ albicantes*—due to laceration of its deeper layers. The umbilicus may be more or less stretched and everted, and finally obliterated, or in some cases pouched out, when it may form a considerable prominence. Should there happen to be a weak portion of the abdominal walls, such as a hernia, this will be unduly protruded. The enlargement is of a rounded form, though tending to be more prominent or to bulge towards the hypogastric region or in the flanks, according to the posture of the patient; it is quite symmetrical in shape, when the patient stands or lies on his back, but alters considerably with a change of position, the abdomen becoming then more prominent in the dependent region, in consequence of the gravitation of the fluid, and this may actually be seen to move as the posture is changed. In contrast with the enlarged abdomen, the chest often looks small and depressed, and the fluid may cause the margin of the ribs to become everted, or it may push forwards the xiphoid cartilage. Mensuration is of service for giving more accurate information as to the size of the abdomen in cases of ascites, and for determining their progress.

(b) The abdomen is perfectly smooth over its entire surface. It usually gives a sensation of tension of the walls, without any hardness underneath. In some instances an obscure feeling of fluctuation is experienced on palpation with the fingers.

(c) The tendency of ascites is to interfere with the abdominal respiratory movements, by preventing the diaphragm from acting properly. At the same time the writer has not uncommonly observed that, even in cases where the accumulation of fluid has been very considerable, abdominal respiration did not seem to be obviously impeded.

(d) *Percussion* affords some of the most important signs of peritoneal dropsy; and when the fluid is present in small quantity this is the only mode of examination that can be relied upon for its detection. In the first place, marked dullness is elicited over the seat of the fluid; while a clear tympanitic sound is heard over the intestines. When there is but little fluid, it may be impossible to detect any abnormal dullness as the patient lies in the recumbent posture; but on placing him on his hands and knees, the fluid gravitates towards the front of the

abdomen, and dulness may then be noticed in the umbilical region. In most cases, however, there is no difficulty in making out the dulness, and when the patient lies on his back, the lower part and sides of the abdomen are dull, while its upper and front part is tympanitic. As more and more fluid collects, so the dulness increases in extent, gathering in, as it were, from below and from the sides, until finally the entire abdomen may be dull, except the umbilical region, which usually remains tympanitic to the last. The boundary line between the dulness and tympanitic sound is usually well defined. As the posture is changed, so will the site of the dulness vary, the part which is undermost presenting this sign, while that which becomes highest is tympanitic, and thus the relative situation of these two sounds, as well as the shape of the dulness, can be altered in a variety of ways. When the patient sits up, the prominence between the recti muscles gives a tympanitic sound on percussion. In exceptional instances a distended colon gives rise to a tympanitic sound along each side of the abdomen, even when there is abundant fluid present.

Another important sign brought out by a form of percussion is the sensation specially termed *fluctuation*, which is the peculiar wave-like movement realised on placing the fingers of one hand over one side of the abdomen, and flipping or tapping the opposite side with the fingers of the other hand. This sensation is very easily brought out if there is much fluid present, provided it is free to move, and sometimes the motion is actually visible. Change of posture will modify the situation over which fluctuation can be produced.

(e) In exceptional instances, when the diagnosis is obscure, it is requisite to resort to a *digital examination* through the *rectum*, and in females through the *vagina*. The fluid collects in the recto-vesical pouch, and on examination *per rectum* the finger detects the sensation yielded by this fluid through its anterior wall. The vagina is usually felt to be shortened, while the uterus is pushed down and flexed. In extreme cases of ascites the posterior wall of the vagina, or even the uterus itself, may protrude through the vulva.

(f) Now and then it is requisite to remove some of the fluid by means of an aspirator or a small trocar, in order to determine its nature. This procedure is also useful when ascites is associated with some other morbid condition within the abdomen, which frequently cannot be made out so long as the fluid remains in the peritoneum.

The ordinary physical signs of ascites will be materially modified or obscured under certain circumstances. For example, the fluid may be so abundant that dulness is observed over the entire abdomen, and fluctuation may be very indistinct. The existence of peritoneal adhesions—for instance, those which may be formed as the result of repeated paracentesis—also renders some of the most characteristic signs of ascites very ill-defined. Again, the association of peritoneal dropsy with some other abdominal morbid condition, such as a new growth, an enlarged liver or spleen, or an ovarian tumour, will also modify the signs elicited. The mesentery may be abnormally short, or the intestines may be adherent, and thus prevented from floating forwards, so that the usual relative positions of dulness and tympanitic sound are not observed.

2. *Mechanical effects of the dropsical accumulation.*—The patient often experiences a feeling of

uneasiness and discomfort in the abdomen, as well as more or less tension and fulness, if there is much fluid present; while there may be a sense of fatigue and aching about the loins or abdominal walls. As a rule no particular pain is felt, but colicky pains are liable to occur from time to time, and extreme distension of the structures constituting the abdominal wall may also cause painful sensations. When the fluid is abundant, the patient is conscious of its weight when he walks; and during progression he throws the head and shoulders back, at the same time keeping the legs apart. Symptoms connected with the alimentary canal are of common occurrence, but these are often to a great extent due to the same cause which originates the ascites, though the fluid must necessarily tend to interfere with the functions of the stomach and intestines. The bowels are usually constipated, but in some instances diarrhoea or dysenteric symptoms may arise. Flatulence is very commonly complained of, even a small amount of gaseous accumulation in the intestines being felt unduly, producing much discomfort, and increasing the enlargement of the abdomen temporarily. Occasionally vomiting occurs, in consequence of interference with the stomach. When a large amount of fluid in the peritoneum presses upon the inferior vena cava, it may lead to anasarca of both lower extremities, with enlargement of the superficial abdominal veins. Exceptionally the anasarca attracts attention at an early period. The flow of blood through the renal veins may also be obstructed, inducing mechanical congestion of the kidneys, with consequent diminution in the quantity of urine and sometimes albuminuria. In rare instances the fluid has been known to accumulate to such an extent as to rupture some part of the abdominal wall.

Ascites also frequently interferes with the thoracic organs. The lungs are more or less compressed and congested, and the breathing becomes chiefly upper-costal, accompanied with various degrees of dyspnoea, and, it may be, cough with frothy expectoration. The heart is likewise liable to be disturbed in its action, as evidenced by palpitation, irregularity, or a tendency to faintness. This organ may also be displaced, so that its apex-beat is too high and too far to the left: in rare instances a basic systolic murmur has been produced by this displacement.

DIAGNOSIS.—The abdominal enlargements from which ascites has to be more commonly distinguished are those due to flabby relaxation of the walls of the abdomen, combined with flatulence; accumulation of fat in the subcutaneous tissue and in the omentum; abundant subcutaneous oedema, which may be associated with and obscure ascites; an ovarian cyst; or a pregnant uterus. Among the more rare conditions with which ascites is liable to be confounded may be mentioned a greatly dilated stomach; colloid disease of the omentum; distension of the uterus with fluid; great accumulation of urine in the bladder; a very large hydatid tumour, usually connected with the liver; extreme cystic enlargement of the kidney; and the so-called 'phantom tumour.' Most of these conditions are described in other parts of this work, and their several diagnostic characters need not be discussed here; but a consideration of the history and existing symptoms of each particular case, and the results of adequate physical and other methods of examination, constitute the data upon which the diagnosis is founded. It must be remembered that ascites

may co-exist with other morbid conditions in the abdomen, their physical signs being then combined. Should there be an enlarged organ or other solid mass, it may often be recognised by making sudden firm pressure with the fingers or *dipping* over the corresponding part of the abdomen, when the fluid is pushed aside, and the underlying resistance can be felt; or paracentesis may be performed, and further examination carried out after the evacuation of the fluid.

Another most important point in the diagnosis of ascites is to make out its cause. If it results from cardiac or renal disease, ascites always follows dropsy in other parts of the body, to which it is also generally subordinate; when it is due to hepatic disease or some neighbouring morbid condition, the peritoneal dropsy appears first, and is, as a rule, throughout most prominent. Should the inferior vena cava be obstructed at its upper part, anasarca of the legs will be observed simultaneously with, or even before, the ascites.

PROGNOSIS.—The prognosis of ascites will mainly depend upon its cause; on the amount of fluid present; on the general nutrition of the patient; on the condition of the principal organs; and on the results of treatment. In some cases ascites is in itself a source of danger on account of the pressure it exerts, especially upon the thoracic organs, and still more if these organs are in a diseased condition. When ascites is due to local interference with the portal circulation, great relief can be afforded and life prolonged by appropriate treatment; while, if the local cause can be removed, the ascites may not infrequently be permanently cured.

TREATMENT.—The principles or treatment applicable to cases of ascites are: (a) to treat the conditions upon which the dropsy depends; (b) to promote absorption of the fluid; (c) to improve the general nutrition; (d) to remove the fluid by operation, if absorption cannot be effected; and (e) to treat any symptoms needing special attention.

(a) The treatment of the causes of ascites is discussed in the articles on the diseases concerned.

(b) Absorption of ascitic fluid is often promoted by acting upon the bowels, skin, or kidneys. In ascites due to local causes, hydragogue and saline purgatives must, however, be administered with caution, and the strength of the patient maintained. In certain instances balsam or resin of copaiba has proved useful as a diuretic in the treatment of peritoneal dropsy. Assistance may be derived from the action of various diaphoretic baths upon the skin. Digitalis and squill may be of service as diuretics; the application of poultices of digitalis leaves over the abdomen is occasionally attended with benefit. The administration of iodide of potassium also seems to aid absorption in some cases, more especially if there be any tendency to syphilitic disease of the liver. It has been recommended to treat ascites merely by a diet of skimmed milk, without any medicine. The application of galvanism to the abdominal walls has been found efficacious in producing absorption of peritoneal dropsy in a few instances.

(c) Treatment directed to the general condition of the patient, and to the state of the blood, is essential in many cases of ascites. Tonics are often of decided service, and preparations of iron are specially indicated for improving the quality of the blood, if there is a tendency to anæmia. Not only

do these remedies sustain the patient, but they may also have an influence in promoting absorption. The diet should be nutritious, and contain but little fluid.

(d) In a considerable proportion of cases, no effect upon the dropsical accumulation is produced by any of the measures thus far considered. It then becomes necessary to determine whether it is desirable to remove the fluid by operation. The fluid may be taken away either by means of the aspirator, by the ordinary trocar and cannula, by Southey's trocars, or by a special apparatus. When ascites is of cardiac or renal origin, the operation can, in the majority of cases, only afford temporary relief, so that there is no object in resorting to it unless the mechanical effects of the accumulation are such as to cause troublesome or dangerous symptoms. When ascites is a local dropsy, the fluid is often so considerable in amount as to necessitate its removal for the mere purpose of giving relief for the time. In cases of ascites associated with malignant disease, for instance, this is all that can be hoped for, as the fluid will certainly collect again. When, however, the condition is due to some local disease which is not in itself fatal, and especially to cirrhosis of the liver, the writer has found signal benefit result from the repeated performance of paracentesis, and has for a long period advocated this plan of treatment as a curative measure, so far as the ascites is concerned. Performed with antiseptic precautions the operation rarely gives rise to any immediate ill-effects, and it is frequently found that remedies will act much more efficiently after the removal of the pressure caused by the fluid than they did previously. In the writer's experience paracentesis, even after one operation, or repeated as often as the fluid re-accumulated, has ultimately led to a complete cure in several instances; in others the cure was partial, a certain quantity of fluid remaining in the peritoneum, limited by adhesions; while in others life has been greatly prolonged, and much comfort afforded. For a few days after the removal of the fluid, the application of a bandage firmly round the abdomen, so as to exert even pressure, may prove of service in aiding the absorption of what remains, and preventing the recurrence of the ascites; and this measure may also be useful when a certain amount of fluid continues after the repeated performance of paracentesis. When the ascites is not cured by repeated tapping, advantage has been found in some instances from allowing the fluid to drain away continuously, or making a free incision.

Operative treatment was introduced in 1896 by Dr. Drummond and Mr. Rutherford Morison, of Newcastle, in cases of ascites from cirrhosis of the liver, having for its object the re-establishment of the portal circulation, by obtaining adhesions between the liver, spleen and omentum, and the parietes. It has since been practised in a few instances, but can only be exceptionally applicable, and is attended with serious dangers.

(e) The symptoms resulting from ascites which are likely to require attention are those connected with the alimentary canal; dyspnœa; and cardiac disturbance, or a syncopal tendency. These should be treated on ordinary principles; but it must be observed that marked dyspnœa or cardiac disorder, if evidently due to the fluid, is an indication for the immediate performance of paracentesis.

FREDERICK T. ROBERTS.

ASEPSIS and ASEPTIC SURGERY.—During the last few years surgeons have been endeavouring to diminish, as far as is compatible with safety, the strength of the antiseptic agents employed during operations and in the dressing of wounds, because it is now recognised that these materials not only increase the amount of exudation, but also diminish the resistance of the tissues to any microbes that may fall into the wounds; in other words, they tend to inhibit the potent antiseptic process of phagocytosis (*see* PHAGOCYTOSIS). There is no doubt that the deleterious effects of these substances upon the epidermis of the surgeon's hands have given a further impetus in this direction; for rough hands are uncomfortable to use and difficult to purify.

Many surgeons, therefore, now repudiate the appellation *antiseptic* and in contradistinction call their method of treatment *aseptic*. This is, however, somewhat of a misnomer, for few, if any of them, dispense with the sterilising of their instruments and dressings by means of the most potent of all antiseptic agencies, namely heat; nor do they neglect the preliminary purification of the skin of the patient, as well as that of the surgeon and of his assistants. They use, however, no germicidal lotions, limiting themselves to boiled water or boiled saline solution, and they employ for dressings only super-heated gauze or cotton-wool. There is more to be said both theoretically and practically for the abandonment of germicidal lotions than for that of antiseptic dressings. The latter have little or no irritating effect upon the wound, the edges of which are usually in contact, and are protected from the dressings by a layer of dried blood or serum; while, on the other hand, should the discharge be sufficiently copious to soak through a simple *aseptic* dressing, there is an obvious danger of the wound becoming infected. It is largely, therefore, a question of convenience. An *aseptic* dressing must be of large size in order to be perfectly safe, and as it is inconvenient in proportion to its bulk, the inconvenience generally outweighs the advantages. Lister himself always taught that *aseptic* surgery was the ideal surgery. During the last few years of his practice he had his sponges wrung out of 1 to 40 carbolic acid solution until they were as dry as possible, and gave up irrigating the wound with this lotion. His results were as good as could be desired.

The description given of an *antiseptic* operation (*see* ANTISEPTIC TREATMENT) applies, with very slight modification, to an *aseptic* one. The skin of the patient is purified with whatever antiseptic agent is in vogue, perhaps with alcohol mixed with 15 per cent. of water, some time before the operation, and is then covered with sterilised gauze until the operation begins. The instruments and swabs are sterilised by boiling. The surgeon's hands are very thoroughly washed with soap and water and subsequently with alcohol (75 per cent.). They are then placed in boiled water or saline solution, and nothing except sterilised water or saline solution comes into contact with the wound during the operation. The dressing is of sterilised gauze or cotton-wool. This method of treatment, in the hands of well-trained and careful surgeons, gives excellent results; but it requires constant attention to the minutest details and is hardly fitted for the exigencies of general practice. The path of wisdom appears to be to use sterilised water or saline solu-

tion during the operation, and afterwards to employ an efficient *antiseptic* dressing.

In abdominal surgery the abandonment of strong lotions has proved of great benefit. The peritoneum is seriously damaged by such solutions, and is thus rendered less able to exert its remarkable power of destroying or subduing the micro-organisms that come in contact with it. There can be no doubt that the abandonment of strong antiseptic solutions has had much to do with the rapid strides that have been made recently in this department of surgery. RICKMAN J. GODLEE.

ASHEVILLE, in North Carolina.—Situated between the Blue Ridge and Alleghany Mountains, in 35° 36' N. lat., 2,250 feet above the sea. A winter and summer resort; of repute in the treatment of phthisis. Temperatures: summer, 70°-7°; winter, minimum has never fallen below 8° F.; range 80°. Rainfall, 40 inches. Possesses a sanatorium. *See* CLIMATE, Treatment of Disease by.

ASIATIC CHOLERA.—*See* CHOLERA, ASIATIC.

ASPHYXIA (ἀ, priv.; and σφύξις, pulse).—*SYNON.*: Apnoea; Fr. *Asphyxie*; Ger. *Erstickung*.

DEFINITION.—The term *asphyxia*, though literally signifying *pulselessness*, is generally understood to mean the condition that supervenes on interruption of the function of respiration. The term *apnoea*, formerly suggested as a more exact one, is now definitely employed by physiologists in a totally different sense, viz. the cessation of the respiratory movements consequent on artificial hyperoxygenation of the blood.

ÆTIOLOGY.—Asphyxia may result from many causes which obstruct or interrupt the respiration. They may be divided into two categories, internal and external.

Internal.—These include paralysis of the respiratory nerve-centres by disease or injury of the medulla oblongata; paralysis of the nerves or muscles of respiration; a rigid fixation of the respiratory muscles; collapse or disease of the lungs; occlusion of the air-passages by organic disease or spasm of the glottis, pressure of tumours, and the like.

External.—To this group belong occlusion of the air-passages by foreign bodies; pressure on the chest not capable of being overcome by the muscles of respiration; closure of, or external pressure on, the air-passages, as in suffocation, strangulation, or hanging. To these external causes are to be added those conditions in which, though the respiratory movements are free, the surrounding medium is incapable of oxygenating the blood, viz. submersion in a liquid medium (drowning); or being surrounded by a medium devoid of oxygen, such as nitrogen or hydrogen. These gases have a purely negative effect; but many other gases which are classed as asphyxiants, such as carbonic oxide, sulphuretted hydrogen, chlorine, chloroform-vapour, &c., have positive poisonous effects, and should therefore be called by some special name, such as *toxic asphyxiants*, to distinguish them from those which have no such properties.

PHENOMENA.—The phenomena of asphyxia have been studied (1) by placing animals in an atmosphere devoid of oxygen, and (2) by suddenly obstructing the trachea (slow and rapid asphyxia respectively). The results, substantially the same, may be divided into three stages, the first being marked by increased respiratory movements, both of inspiration and

expiration. The second is characterised by increased expiratory efforts and general convulsions. In these two stages the blood-pressure rises. In the third stage the blood-pressure falls, the reflexes are abolished, insensibility sets in, and the inspiratory efforts are long drawn out and finally cease. The failure of respiration is followed after a time by that of the circulation, the heart stopping in diastole. Though the time required for producing complete asphyxia varies to some extent in different animals and in the same animal under different conditions, death from rapid asphyxia takes place usually in four or five minutes after complete obstruction of the trachea.

PATHOLOGY.—The presence of non-oxygenated blood in the vessels by stimulating the respiratory centre causes increased respiratory efforts, and by irritating the vaso-motor centre induces contraction of the arteries. In consequence, the blood-pressure rises and the cardiac cavities become more and more distended. The distension finally becomes so great that the heart is unable to cope with the accumulation of poisoned blood, the cardiac movements become slow and feeble, the blood-pressure falls, and finally the heart ceases to beat.

In the production of asphyxia two chief factors must be considered, namely the deprivation of oxygen and the accumulation of carbonic acid; but it is to the deficiency of oxygen rather than to the excess of carbonic acid that the phenomena of asphyxia are to be ascribed, for it has been shown that if an animal be placed in an inert atmosphere (nitrogen) all the phenomena of asphyxia ensue, though there is no impediment to the exit of carbonic acid. On the other hand, when an animal is made to inhale an atmosphere containing both an excess of carbonic acid and plenty of oxygen, the effects resemble those of a narcotic poison; there is laboured and increased frequency of breathing but no convulsions. Practically, therefore, under experimental conditions asphyxia is tantamount to deprivation of oxygen, but in clinical and medico-legal practice it is rare to meet with instances of such pure asphyxia. The cases designated as asphyxia are really combinations in varying degree of the two conditions (asphyxia and carbonic acid poisoning), and very often the toxic effects of carbonic acid cannot be considered to hold a subordinate place.

ANATOMICAL CHARACTERS.—The blood is of a dark colour, owing to complete reduction of the hæmoglobin. The proportion of carbonic acid is increased, and owing to its excess the blood coagulates slowly or imperfectly; hence it remains fluid or forms few and soft coagula. The large veins, the pulmonary artery, and the cavities of the right side of the heart may be distended with dark fluid blood, but this is by no means necessarily the case. The left side is usually empty or nearly so. The lungs may be congested, but are often pallid and anæmic, though the dependent parts usually exhibit appearances of hypostatic engorgement. The abdominal viscera, especially the kidneys, are often congested. The appearance of the brain varies; it may be anæmic or more or less congested. Special signs characterise special modes of causation of asphyxia.

TREATMENT.—Resuscitation from pure asphyxia is possible so long as the heart continues to beat. After cessation of the heart's action treatment is unavailing, except in cases of cessation from mere

over-distension, in which bleeding from the external jugular vein may be resorted to with success. The chief indication in the treatment of asphyxia is to effect oxygenation of the blood by the introduction of air into the lungs. See **ARTIFICIAL RESPIRATION**; and **RESUSCITATION**.

D. FERRIER.

R. G. HEBB.

ASPHYXIA, Local.—A synonym for Raynaud's Disease. See **RAYNAUD'S DISEASE**.

ASPIRATOR.—See **PARACENTESIS**.

ASPIRATOR.—**SYNON.**: Fr. *Aspirateur*.

DESCRIPTION AND MODE OF EMPLOYMENT.—There are two forms of 'aspirator' in common use. (1) That designed by Dieulafoy in 1869 consists of a glass syringe, with a capacity of one to four fluid ounces, having at its lower end two openings provided with stopcocks. If the piston is raised after the cocks are closed, a vacuum is created in the syringe, which can be maintained by fixing the piston in the withdrawn position. An india-rubber tube is fitted into each of the two openings, and these tubes are provided with coils of wire inside to prevent them from collapsing. At the end of one tube is fixed a fine hollow needle. The needle should have only one opening, at the point. The instrument is thus used:—A vacuum having been created in the syringe, the needle is introduced into the part to be operated upon. As soon as the opening of the needle is beneath the skin the stopcock leading to it must be opened. The vacuum will then extend to the point of the needle; and consequently, if it be gently pushed onwards, the moment it encounters fluid this will jet up into the glass syringe, when its nature may be ascertained. In aspiration thus performed it is impossible to pass the needle through a collection of fluid without discovering it. If the fluid is sufficient in amount to fill the syringe, the stopcock leading to the needle is to be closed and the other opened: by this the fluid may be discharged. The vacuum may then be re-established, and the operation repeated as often as is necessary. The aspirator may be converted into a siphon by opening both stopcocks when the syringe is full, and allowing the discharge-tube to hang down. The action of the instrument may also be reversed, and it may be thus employed for injecting fluids. (2) In Potain's aspirator the receiver is a glass bottle, from which the air is exhausted by means of an attached air-pump. An objection to this is that, if the needle becomes choked, it cannot be cleared by pushing down the piston and driving some of the fluid back through it; but it has the advantage of being less liable to get out of order, as the fluid does not touch the exhausting syringe. The disadvantage can be avoided by the use of an attached trocar and cannula, but this modification is suitable only when the exact locality of the fluid is known, for the instrument is thereby converted into a mere 'suction-trocar.' The needles employed vary in size. Dieulafoy recommends that they should be about $\frac{1}{10}$, $\frac{1}{20}$, $\frac{3}{20}$, and $\frac{1}{12}$ of an inch in diameter, and calls them Nos. 1, 2, 3, and 4 respectively.

The following rules must always be observed in using the aspirator:—1st. See that the skin is thoroughly cleansed (see **ASEPSIS**), the needle pervious and clean, and the syringe in order before using it. It is advisable to boil the needle before use. Any parts made of platinum can be

sterilised by heating over a spirit lamp. 2nd. The needle must be pushed straight on in one direction only. Its course should not be altered while the point is under the skin. If no fluid is found, it may be withdrawn and reinserted. It must be held as steady as possible during the aspiration. 3rd. If the fluid will not flow with the pressure of the atmosphere, it is of no use squeezing and pressing the part. This can only do harm. 4th. Aspiration must cease at once when blood comes in any quantity, especially in abscesses. 5th. Keep up the vacuum during the withdrawal of the needle, lest some of the morbid fluid be left in its track. 6th. If the needle becomes choked, force a little of the fluid back through it in order to clear it. In Potain's instrument the trocar can be re-inserted. 7th. After use the whole apparatus must be thoroughly cleaned with plain water for completely removing pus, blood, and other albuminous fluids, which would be coagulated by carbolic acid. After this is thoroughly done, the needle, tubes, and syringe should be disinfected by boiling. *See ASEPSIS.*

USES.—Aspiration is used for purposes of diagnosis and treatment. Dieulafoy asserts that with the No. 1 needle it is possible to search for fluid without danger, whatever may be its seat or its nature; and experience has proved this to be practically true. In treatment it has been employed in the following affections, but is now much less used than formerly, owing to the marked diminution in the risk of septic contamination of open incisions, and the proved necessity either for continuing the drainage of a cavity from which fluid has been withdrawn, or for combining other surgical procedures with the removal of the morbid fluid.

Abscesses.—In acute abscesses simple aspiration is usually of little value, as the pus soon re-accumulates. In chronic abscesses connected with diseased bone it usually fails, and is often impracticable, from the presence of cheesy masses in the pus. If performed with proper antiseptic precautions, however, it can do no harm, and may, if repeated, diminish the size of the sac before other means are adopted, or even effect a cure. Instead of the small needle, a large trocar, from $\frac{1}{8}$ to $\frac{1}{4}$ inch in diameter, is fitted to the aspirator. If necessary a small incision is made in the skin to facilitate the introduction of the trocar. The abscess having been emptied of its contents, the cavity is distended with a solution of perchloride (1 in 4,000), or of biniodide (1 in 5,000) of mercury, which is also withdrawn by the aspirator. This must be repeated again and again, till the fluid returns perfectly clear. Finally, an emulsion of iodoform and glycerine (1 in 10) is injected, the trocar is withdrawn, and the opening sealed with collodion. Most commonly the abscess fills again in a few days, after which the fluid may be gradually absorbed, but it is frequently necessary to repeat the operation more than once. The method has often to be supplemented by that of free drainage.

Diseases of the Liver.—Hydatid cysts were formerly treated by aspiration. *See PARACENTESIS.*

Retention of Urine may be safely relieved by using No. 2 needle above the pubes; but great care must be taken that the needle is sharp as well as clean.

Diseases of Joints.—Aspiration is occasionally useful in acute synovitis, especially of the knee-joint (*see JOINTS, Diseases of*). No. 1 or 2 needle should be used. *Spina bifida* is occasionally

treated by aspiration, with or without the subsequent injection of Morton's fluid. In *Pleurisy* accompanied by serous exudation, aspiration is frequently employed (*see PLEURA, Diseases of; and PARACENTESIS*). In *Ascites*, except for purposes of diagnosis, the aspirator presents no advantages over a trocar properly constructed so as to exclude air (*see PARACENTESIS*). *Pericarditis.*—The operation of aspiration has frequently been successfully performed for grave pericardial effusion. It is thus carried out:—A spot is chosen 2 to 2½ in. beyond the left edge of the sternum, in the 4th or 5th interspace. No. 2 needle is then passed obliquely upwards and inwards, care being taken to turn on the vacuum as soon as the eye is covered. The moment the fluid jets into the syringe the needle must be held steadily till the flow ceases. If this be done there is no danger of wounding the heart. If there is any doubt as to the existence of fluid, No. 1 needle must be employed, with which the heart may be punctured without great danger.

FRED. C. WALLIS.

ASTHENIA } (ἀ, priv. ; and σθένος, strength).—
ASTHENIC }

Terms signifying want of strength. As applied to the entire system, they indicate considerable general debility; in connection with particular diseases, they imply that these are attended with marked weakness. *See DEBILITY.*

ASTHENOPIA (ἀ, priv. ; σθένος, strength ; and ὤψ, the eye).—Weakness of sight. *See VISION, Disorders of.*

ASTHMA, SPASMODIC (ἄσθμα, panting ; from ἄω, I blow).—**SYNON.** : Bronchial asthma ; Fr. *Asthme* ; Ger. *Bronchial-Asthma*.

DEFINITION.—An affection characterised by severe paroxysmal dyspnoea recurring at more or less definite periods, generally in the night, the dyspnoea being due to spasmodic contraction of the bronchi, produced by a variety of causes.

ÆTIOLOGY.—The causes that induce an attack of asthma are very various, and may be roughly classed according to their action, *direct* or *indirect*, on the respiratory organs. In the former class the exciting cause immediately affects the mucous membrane ; in the latter it does so in a more circuitous manner, generally through the blood or the nervous system :—

Direct	{	Dust.
		Vegetable irritants.
		Chemical vapours.
		Animal emanations.
Indirect	{	Climatic influences.
		Bronchial inflammation.
		Through the nervous system.
		Through the blood
	{	Centric.
		Excito-motor.
		Gout.
		Syphilis.
Heredity.	{	Skin-diseases.
		Renal diseases.

Direct causes.—Common roadside dust ; fluff from woollen clothing ; the dust of mills, threshing-floors, or bakehouses ; and any mechanical particles when inspired, will produce in some persons an asthmatic seizure ; dust of low specific gravity being more apt to have this effect than heavy particles like coal, steel-filings, &c., from which arise lesions in the lung of a more permanent and serious character.

The odour evolved by certain vegetables, such as

ipecacuanha; the pollen of many grasses and plants (see HAY-FEVER); certain chemical vapours, as that of pitch, sulphurous acid, and phosphorus-fumes; the peculiar smell of some animals, as dogs, cats, horses, and hares, may each provoke a spasm in individual cases.

A still more powerful cause is climatic influence, the action of which on different patients cannot, unfortunately, be reduced within the limits of a law, but depends mainly on the idiosyncrasy of the individual. Extremes of temperature, or excessive dryness or dampness, may produce an asthmatic seizure, but in the largest number of cases one of two elements appears as the chief factor. One large class of sufferers trace the attack to *dampness*, whether of soil or of atmosphere, in combination with either heat or cold; another to *closeness* of atmosphere and a want of proper circulation of air, such as is found in deep valleys and thick forests, and during thundery weather—this last class experiencing great relief when a breeze springs up. Malaria plays an important part occasionally in the causation of asthma.

Far more general and intelligible in its action is *bronchial inflammation*, which is the cause in 80 per cent. of asthmatic cases. It frequently happens that after whooping-cough, measles, or infantile bronchitis the tendency towards asthma begins to appear. These diseases, implicating as they do both bronchial muscle and nerve supplying it, leave their mark behind, either in irritability of the mucous membrane; in induration of some portion of the lung, generally at the root; or in enlargement of the bronchial glands, causing pressure on the pneumogastrics, or on some of the branches of the pulmonary plexuses (see BRONCHIAL GLANDS, Diseases of); and thus lay the foundation of asthma in after-life. Asthma following on arrested phthisis is not rare in adults, probably from enlargement of the bronchial glands. In other instances the indirect cause may be found in morbid states of the nose or throat.

Indirect causes.—This class of causes includes those acting through the general nervous system; those acting through the blood; as well as the hereditary influences.

The *centric* subdivision embraces attacks arising from emotion, anger, or fright; as well as the curious alternations of asthma, neuralgia, angina, and gastralgia due to some centric irritation in the medulla, involving the origins of the fifth and eighth pairs of nerves, and manifesting itself by affecting first one branch and then another of these nerves.

Excito-motor causes may be illustrated by indigestion or costive bowels giving rise to a paroxysm of asthma. In the first case, irritation of the medulla is induced through the gastric branches of the pneumogastric, and a motor effect is reflected through the pulmonary branches. These *peptic* attacks, as they are called, occur more frequently after suppers than dinners, probably because reflex irritability is always exalted by sleep, as we know to be the case in epilepsy and the convulsions of childhood.

Gout, towards old age, often takes the form of asthmatic seizures, which alternate with the articular affections. In like manner attacks of the disease have been attributed to syphilis, but here the poison generally acts by enlarging the bronchial glands.

The connection between asthma and various kinds of skin-disease is intimate: the subsidence of eczema, of urticaria, or of psoriasis, has often been accompanied by fits of spasmodic breathing, which have

ceased on the reappearance of the eruptions. Here, again, the state of the blood is presumed to be the origin of both maladies, as in disease of the kidneys, which will be presently referred to.

Heredity can be traced in about 40 per cent. of asthmatics, though the tendency often does not show itself till late in life. The characteristic form of chest is frequently transmitted from parents to children; and even when this is not so, a disposition towards spasmodic symptoms in catarrhal attacks is often seen in the children of some asthmatics.

SYMPTOMS.—The patient may retire to bed with few or no premonitory symptoms, and sleep for some hours, but is disturbed in the late night or early morning—2 A.M. is a common time—by a feeling of oppression approaching to suffocation, referred either to the throat, sternum, or epigastrium, which obliges him to sit up in order to breathe. Sometimes the onset is more gradual: the patient, having fallen asleep in spite of uneasy sensations, begins to wheeze during sleep, and is only aroused when the dyspnoea becomes severe. The breathing is accompanied by a humming sound, which gradually develops into a great variety of discordant noises.

In order to increase the capacity of his chest to the utmost, the asthmatic patient sits up and fixes his shoulders, either by placing his hands on either side of him, or by supporting his elbows on his knees; or sometimes he stands leaning over the back of a chair or other support. In one or other of these positions he remains immovable, with chest, back, shoulders, and head fixed; unable to speak or even to move his head; the lips being parted; the face pale, anxious, and, if the dyspnoea continue, livid; and the eyes prominent and watery. Every muscle of respiration, ordinary and extraordinary, is brought into requisition; those passing from the head to the shoulders, to the clavicles, and to the ribs become rigid, and, in place of moving the head and neck, act the reverse way, being used from fixed points to raise and dilate the thorax. The trapezii and levatores anguli scapulae by their contraction elevate the shoulders, in order that the muscles connecting these with the ribs may act as elevators to the latter. Even the muscles of the back are pressed into the service, and they almost cease to support the back; consequently the patient stoops. At each inspiration the sterno-cleido-mastoids stand out like cords, leaving a deep hollow between their sternal attachments; the diaphragm is contracted, and hence the stomach, liver, and heart are somewhat displaced. With all this display of muscular force, the chest remains almost motionless, being expanded to a variable extent.

In spite of the great dyspnoea, respirations are not proportionately frequent, seldom exceeding thirty, and sometimes falling to nine a minute. The expiration is prolonged, being generally two or three times as long as the inspiration. The pulse is usually slow and feeble; the temperature rarely exceeds 99° F., and is often below 98° F. Analysis of the expired air shows the oxygen to be almost entirely replaced by carbonic acid, which may increase from the normal to as much as 11 per cent., the nitrogen varying from 89 to 93 per cent. The arrest of expiration is probably the cause of the accumulated carbonic acid, but the total disappearance of the oxygen is hardly to be explained, for that oxidation of the tissues does not proceed is shown by the pale urine passed after the fit.

PHYSICAL SIGNS.—These reveal less than might be expected. The percussion-note is somewhat raised over the whole chest, most so in the posterior regions, where a drum-like sound occasionally prevails; this hyper-resonance is probably due to accumulation of air induced by obstructed expiration, and in incipient cases passes off with the attack. The normal areas of cardiac and hepatic dullness often disappear during the attack, owing to the emphysema temporarily induced. When the attack has passed off, careful examination will sometimes detect dullness in the supra-scapular or inter-scapular regions, on one or both sides of the chest, indicating probable enlargement of the bronchial glands.

Auscultation shows an entire abolition of the normal breath-sounds, and the existence of dry sibilant or sonorous rhonchi everywhere, varying in tone according to the calibre of the bronchial tubes; the smaller tubes giving the high notes, and the larger ones the deep notes. These sounds continually change their position, springing up under the listening ear, and as quickly vanishing again, to give place to profound silence.

PROGRESS AND DURATION.—A paroxysm of asthma, when once established, lasts from half an hour to several days, and generally terminates with expectation—thin and transparent if the seizure be short; but abundant and more or less opaque if the fit be prolonged, or if the case be chronic. The sputum has also been found to contain, in greatest abundance at the close of the attack, (1) the so-called Charcot-Leyden crystals, which are minute octahedra of unknown composition, but soluble in warm water; (2) Curschmann's spirals, which consist of closely packed epithelial cells, arranged in a spiral form surrounded by mucoid material; and (3) a large number of eosinophile cells. None of these are, however, pathognomonic of asthma. The urine is light-coloured and plentiful; the skin is bathed with perspiration; and flatus is often expelled from the bowels. Little or no food is taken during the attack, at the close of which the patient falls asleep.

The recurrence of the attacks when once they have been excited is generally periodic; but much depends on the presence or absence of the exciting cause. In many cases the patient is quite free from wheezing and dyspnoea in the intervals, and feels and acts like other people; but when the attacks follow each other closely, a more or less wheezy condition remains behind, and a few signs of obstructed breathing are generally to be detected in the inter-scapular regions.

COMPLICATIONS AND SEQUELÆ.—If the asthmatic attacks become habitual, their effects are seen on the patient's frame and on the organs implicated. The shoulders become raised, the head being buried between them; the muscles of the back, owing to their being called on to act as extraordinary muscles of respiration, are diverted from their use as erectors of the spine, which, accordingly, yields in the anterior direction, and the patient stoops. The frequent occurrence of spasmodic contraction of the bronchi causes hypertrophy of their muscular coat; and this, with or without the congestion of the mucous membrane accompanying it, leads in time to thickening of the tubes and permanent narrowing of their calibre. The more common result of asthma is emphysema, arising from the difficulty of expiration. The emphysema, at first temporary, becomes in chronic cases permanent, and gives rise

to displacement of the adjoining organs (*see EMPHYSEMA*). Contraction of the bronchi largely influences the pulmonary vessels, and considerable obstruction of the pulmonary circulation is the result. The vessels become gorged, and the lungs sometimes cedematous. If the emphysema be extensive, we may in time expect dilatation of the right side of the heart and marked prominence of the veins of the breast and neck, and the effects may be carried so far as to cause oedema of the lower extremities and albuminous urine, as the writer witnessed in a case where these symptoms disappeared on the subsidence of the asthma.

PATHOLOGY.—Patients rarely, if ever, die of spasmodic asthma, though death may ensue from some of its complications and sequelæ; and the disease, being a functional one, cannot be said to have any morbid anatomy. The onset and departure of the attack, and the ever-changing physical signs, led Laennec to think that whatever obstruction in the bronchial tubes caused the phenomena must be of a spasmodic and transitory nature. He therefore concluded that asthma was due to a spasm of the bronchial muscles which had been described by Reisseissen. Laennec also showed that an asthmatic sufferer could sometimes, after holding his breath, actually breathe naturally for one or two respirations, thus clearly demonstrating that the spasm was capable of momentary relaxation. Other theories were put forward, and doubt was thrown on the existence of muscular fibres in the bronchi, until Dr. C. J. B. Williams proved their existence by his experiments on the lungs of oxen, dogs, rabbits, and other animals, when he caused contraction of the trachea and bronchial tubes by the application of electrical, chemical, and mechanical stimuli. The muscular coat was shown to be more abundant in the smaller tubes than in the larger, the former contracting sufficiently to obliterate their passages entirely. In asthma, excitation of the muscles probably takes place through the anterior and posterior pulmonary plexuses, which are made up of branches from the pneumogastric, recurrent laryngeal, and spinal nerves, and from the ganglia of the sympathetic, thus giving the bronchial tubes a very wide area of connections. The branches of the pulmonary plexus form a network round the bronchial tubes, and contain some minute ganglia. When the cause is direct, as dust of any kind or climatic influence, the spasm may be induced by reflex action through these small ganglia, or through the pulmonary plexus, though it soon extends, involving the pneumogastrics, and affecting the thoracic muscles through the upper cervical, phrenic, and dorsal nerves. Where emotion, fright, or laughter starts the fit, the irritation is centric, and causes a motor effect on the pulmonary plexus through the pneumogastrics. Where, again, indigestion excites it, the sensation passes through the gastric branches of the pneumogastric, and is reflected by the motor filaments of the pulmonary plexus. Lastly, where gout, syphilis, albuminuria, skin-disease, and heredity are the excitants, we may regard the blood itself as causing the local irritation. Spasmodic asthma may, therefore, be considered as a neurosis of the pulmonary branches of the plexus of that name similar to other neuroses, as hemicrania and sciatica, and giving rise, through the motor nerves of the plexus, to spasm of the bronchial muscle. *See also BRONCHIAL GLANDS*; and *BLOOD, Morbid Conditions of*.

DIAGNOSIS.—Asthma is distinguished from *bronchitis* by the fugitive physical signs; by the spasmodic character of the dyspnoea; and by the scant expectoration. The breathing in bronchitis, when at all difficult, is hurried; in asthma it is slow, wheezy, and prolonged; this feature also contrasting strongly with the gasping, panting dyspnoea generally accompanying pneumonia, pleurisy, and some forms of heart-disease. From *croup* it is recognised partially, but not entirely, by the age of the patient; and partly by the character of the dyspnoea, which in croup is inspiratory, whereas in asthma it is mainly expiratory. This characteristic also distinguishes asthma from spasm of the glottis and the various forms of laryngeal dyspnoea. The diagnosis from *emphysema*, which is so often mixed up with spasmodic asthma, is founded chiefly on the paroxysmal character and violence of the asthmatic dyspnoea, and on the complete freedom of the intervals, the dyspnoea of emphysema being more or less permanent.

Aneurysm of the aorta and other *mediastinal tumours* often give rise to symptoms so exactly simulating spasmodic asthma as to make the diagnosis difficult, and this is to be accounted for by these tumours pressing on the pneumogastric nerve and its branches, and thus inducing an asthmatic spasm. These cases are, as a rule, however, accompanied by a certain amount of stridor arising from laryngeal spasm, not present in asthma, and this symptom is often of great diagnostic value in obscure cases. As the tumour enlarges, it causes greater pressure on the lungs, trachea, œsophagus, sympathetic ganglia, or other structures, and produces shrill cough, dysphagia, difficulty of inspiration, pain in the chest, impulse in the thoracic wall, and other well-known aneurysmal symptoms. Moreover, certain physical signs become evident, e.g. dulness over the first portion of the sternum or to the right of it, or between the scapulae; tubular sounds and bronchophony close to the sternum, or above one or both scapulae; or some form of bruit or murmur in the course of the aorta. These and other symptoms and signs contrast sufficiently with those of spasmodic asthma to make the diagnosis from developed aneurysm comparatively easy.

In some cases of *renal disease* in which albumen appears in the urine, a form of dyspnoea appears, which is occasionally marked by paroxysmal features, and has been mistaken for spasmodic asthma. Renal dyspnoea differs, however, as a rule, in being more continuous, and in having for its origin œdema of the lung rather than bronchial spasm. See **RESPIRATION, Disorders of.**

PROGNOSIS.—The question of recovery in cases of asthma depends, to a certain extent: first, on the possibility of the removal of the exciting cause; secondly, on the age of the patient; thirdly, on whether the attacks increase or not in frequency; fourthly, on the condition of the lungs and the breathing in the intervals.

If the patient be young (say, under fifteen), the chest well formed, the attacks tending to diminish in frequency and intensity, and the lungs free in the interval, a most hopeful prognosis can be given. If, on the other hand, the patient be middle-aged, the attacks increasing in number and severity, and the breathing more or less short in the intervals, we may conclude that there exists a considerable amount of permanent emphysema, which renders the prognosis of an unfavourable character. In every

instance the detection and removal of the exciting cause or causes, as the case may be, exercise a chief influence over the prognosis.

TREATMENT.—The principal difficulty in the treatment of asthma lies in clearly ascertaining the nature and origin of the exciting cause. When this is discovered two great principles should guide us, namely: first, to avoid or remove the exciting cause; secondly, to allay and prevent the spasm.

Many of the cases arising from direct causes, as from dust and chemical vapours, are cured by simple avoidance of the exciting cause. Where bronchial inflammation induces the spasm, the inflammation must be subdued by salines and expectorants, combined with some anti-spasmodic, as belladonna, henbane, or stramonium. In more chronic instances, where some thickening of the walls of the larger bronchi and enlargement of the bronchial glands exist, iodide of potassium in doses of grs. v. to xv., or iodide of sodium in doses of grs. v., or a combination of both iodides, has been found very beneficial when persisted in for long periods. Symptoms of iodism may be generally averted by temporarily diminishing the dose and diluting more largely with water. The waters of Woodhall and Purton in England, of Kreuznach in Germany, of Saxon in Switzerland, and Salsomaggiore in Italy, contain iodides largely diluted, and may be beneficially administered in asthma. Affections of the nose or throat will require attention (see **NOSE, Diseases of**). Where the attacks depend on gout, syphilis, or renal disease, or are connected with skin-disease, treatment must be directed to the condition of the blood. Arsenic proves of signal service in asthma co-existing with eczema, psoriasis, or other skin-affections.

Where heredity is the predisposing cause, the origin of the disease lies generally in defective development of the frame or of the lung-structure of the patient. For such persons gymnastic exercises, carefully conducted swinging on the trapeze, and other means of expanding the upper part of the chest and correcting the asthmatic stoop are to be employed, combined with tepid or cold sponging and as much outdoor life as possible, with walking and riding without fatigue. The tendency to catarrh is thus lessened, and the frame of the patient developed and fortified. In a large number of cases—as, for instance, those arising from climatic influences—we have to treat a simple neurosis, and to allay the spasm either by climatic or by medicinal means, of which the former is often the more important, and, owing to the leading part played by the idiosyncrasy of the patient, generally the most difficult. In obstinate cases the doctrine of contrasts appears the only safe one. Where the disease has been contracted in a moist climate, a dry one must be tried; if in an inland district, the seaside must be resorted to; but for by far the majority of asthmatics the atmosphere of large towns is suitable and the smokier the air and the closer the streets the more good do the sufferers appear to receive. London, Glasgow, Birmingham, and Bristol are all favourable resorts for cases of neurotic asthma; and the points of difference between their atmospheres and that of the country consist (1) in greater dryness; in deficiency of oxygen; and (3) in excess of carbonic acid and carbon: all of which peculiarities appear to exercise a sedative effect on the neurosis. Damp, whether of soil or

atmosphere, is usually found to be hurtful, and is one of the chief exciting causes of asthma. Nothing exemplifies this more completely than the excellent results in cases of asthma produced by climates of which the atmosphere is dry.

The medicines most useful in asthmatic attacks are antispasmodics, either stimulant or sedative. The former, including alcohol, strong coffee, nitroglycerine, ether, and nitrite of amyl, will prove more efficacious where emphysema is present; the sedative comprise stramonium, belladonna, Indian hemp, lobelia, *Datura tatula*, tobacco, opium with its alkaloids, and drugs of a similar action. These may be taken internally in the form of extracts or tinctures; or smoked in pipes or as cigarettes; or inhaled as vapour diffused through the room by burning powders or pastilles containing them. A popular and often reliable remedy is the vapour arising from the combustion of nitre-paper, and other useful forms of powder may be devised; but these should be recommended with caution, lest an unfortunate habit of indulgence in fuming preparations be acquired. In the severest attacks the patient can neither smoke nor swallow, and in this difficulty of introducing medicines into his system we find the hypodermic injection of atropine (gr. $\frac{1}{100}$), morphine (gr. $\frac{1}{4}$ – $\frac{3}{4}$), and chloral hydrate, or suppositories of morphine and belladonna, prove effectual; but if albuminuria or emphysema be present, or if the pulse be weak, they must not be used. Chloroform often acts like a charm in the worst cases, and after inhaling 20 to 60 minims the patient will gain the sleep which has been denied to him for hours or even days; but as the effect is generally transitory, and the use of the remedy not free from risk, it should be given, if at all, in capsules containing a measured dose. Iodide of ethyl (m. v.-x.) can be inhaled with advantage. Ether is safer, but not so effective. Chloral hydrate in doses of 15 to 20 grs., repeated every four hours until the spasm subsides, has produced not only temporary but even permanent good in a large number of asthmatic cases, and, if watched, may be persisted in for some time. In the writer's hands it has proved a most successful remedy. In the use of antispasmodics we must avoid judging the effects of one from the failure of another of the same class; but in difficult cases we must try each in succession—for it often happens that the successful remedy is only arrived at after repeated trials.

The mineral waters of Mont Dore and La Bourboule contain arsenic, and appear to act favourably in the more purely neurotic cases of asthma, while those of the Pyrenean sulphur springs of Eaux Bonnes, Eaux Chaudes, and Cauterets, are reputed to exercise a beneficial influence over the malady, but they have not been successful in the writer's experience, and it is more probable that they relieve by reducing the catarrhal symptoms than that they either allay the spasm or prevent its recurrence.

Compressed-air baths at pressures varying from one and a half to one and two thirds of an atmosphere, and lasting two hours, are of the greatest benefit to asthmatics, and appear to reduce the sensitiveness of the pulmonary plexus and to diminish cough and spasm, but they must be taken in numbers varying from 24 to 50 to be of permanent value. See AIR, Therapeutic Use of.

The dietetic treatment varies in individual cases, but as a rule asthmatics should dine early, and for the rest of the day limit themselves to liquid food,

such as beef-tea, soups, and milk, combined with such an amount of stimulant, in the form of a pure spirit, as may be necessary, thus avoiding any distension of the stomach and intestines before retiring to rest. The diet should consist of brown bread, dry toast, and biscuits—excess of starch in any form being studiously avoided—a fair supply of plainly cooked meat, fish, or poultry, and a limited amount of vegetables and fruit, care being taken to select only the most digestible of each class. Stimulants may sometimes be taken, but with discernment.

C. THEODORE WILLIAMS.

ASTIGMATISM (ἀ, priv.; and *στίγμα*, a spot or point).—SYNON.: Astigmism.—Want of symmetry in the anterior refracting surfaces of the eyeball, in consequence of which rays of light proceeding from a point are not brought to a focus upon the retina as a point, but only as a diffused spot. See VISION, Disorders of.

ASTRINGENTS.—DEFINITION.—Medicines which cause contraction of tissues.

ENUMERATION.—The chief astringents are: Nitrate of Silver; Sulphate of Copper; Sulphate of Zinc; Acetate of Lead; Perchloride of Iron; Alum; Tannic and Gallic Acids, and vegetable substances containing them, such as Oak Bark, Galls, Kino, and Catechu; and Dilute Mineral Acids. Some authors also include in this class of remedial agents such articles as Ergot of Rye, which contracts the blood-vessels and lessens hæmorrhage after it has been absorbed into the blood, although it has no local astringent action.

ACTION.—With the exception of gallic acid, the substances already mentioned coagulate or precipitate albumen. Dilute mineral acids do not coagulate albumen, but precipitate many albuminous bodies from the alkaline fluids by which they are held in solution. When applied to a surface from which the epidermis has been removed, the other astringents combine with the albuminous juices which moisten this surface, as well as with the tissues themselves, and form a pellicle more or less thick and dense, which in some measure protects the structures beneath it from external irritation, at the same time that they cause the structures themselves to become smaller and more dense. On a mucous membrane they have a similar action, and they lessen its secretion. It was formerly supposed that their action was partly due to their causing the blood-vessels going to a part of the body to contract, thus lessening the supply of fluid to it; as well as to their effect on the tissues themselves. But experiment has shown that, while nitrate of silver and acetate of lead possess this power, perchloride of iron and alum do not, and that tannic and gallic acids actually dilate the vessels. The astringent action of these latter drugs must therefore be exerted upon the tissues.

USES.—Astringents may be employed locally in various forms. In the solid form, as a powder, or in various preparations such as lotions, ointments, plasters, and glycerines, they are applied, especially the metallic astringents, to wounds and ulcers for the purpose of reducing the size and increasing the firmness of exuberant granulations, as well as of protecting the surface by forming a pellicle over it. They are used to lessen congestion and diminish the secretion of the various mucous membranes: as a lotion to the eye and mouth; as a

gargle or a spray to the throat ; in the form of an injection to the nose, urethra, and vagina ; and as a suppository to the rectum. Administered internally, several astringents have a powerful effect in checking diarrhoea, and certain of them may have a local action upon the stomach and intestines.

The *remote* action of such astringents as acetate of lead and gallic acid, when absorbed into the blood, in lessening hemorrhage, is made available in the treatment of hæmoptysis, hæmatemesis, hæmaturia, and loss of blood from other parts of the body.

T. LAUDER BRUNTON.

ASTURIAN ROSE.—*See* PELLAGRA.

ASYSTOLE (ἀ, neg. ; and *συστολή*, a contraction). *SYNON.* : Fr. *Asystolie* ; Ger. *Mangelnde Zusammenziehung*.—The most advanced stage of progressive failure of the heart, in which the ventricular walls have become so feeble that the systole is incomplete and highly irregular, and the cavities are dilated from over-distension with undischarged blood. *See* HEART, Dilatation of.

ATAVISM (*atavus*, a grandfather).—The inheritance of a disease or constitutional peculiarity from a generation antecedent to that immediately preceding. *See* GOUT.

ATAXIA } (ἀ, priv. ; and *τάξις*, order).—Terms
ATAXIC } which originally meant any irregularity or disorder, but are now specially applied to irregularity of associated or co-ordinated muscular movements. Ataxia is frequently used as synonymous with the disease known as *locomotor ataxy*. *See* also CO-ORDINATION ; EQUILIBRIUM ; and FRIEDREICH'S DISEASE.

ATAXIC PARAPLEGIA.—*See* COMBINED DEGENERATION OF THE SPINAL CORD.

ATELECTASIS (ἀτελής, imperfect ; and *ἐκτασις*, expansion).—Absence or imperfection of the expansion of the pulmonary alveoli which normally takes place at birth, the lungs thus remaining more or less in their fetal condition. *See* LUNGS, Collapse of.

ATHEROMA.—*See* ARTERIES, Diseases of.

ATHETOSIS (ἄθετος, without fixed position).—*DEFINITION.*—A name given to a condition in which the hand and foot are in continual slow, irregular movement, and cannot be retained by the will in any given position.

DESCRIPTION.—The special character of the movements in athetosis is that they are slow and deliberate. They usually affect the arm and leg on one side only. Voluntary power is considerable, but is interfered with by the slow spasm. The fingers are irregularly flexed and extended : at one moment they spread wide apart, the thumb being outstretched ; thereafter, first one, then another, is bent into the palm, and again extended, the movements being extremely varied. They can be arrested for a moment in certain positions by the will, but are then renewed with increased force. The foot is usually inverted, the toes being flexed or extended, but in less constant movement. The spasm may cause pain. The muscles sometimes become hypertrophied. The movements in most cases cease during sleep, in some they do not. Sensation is often but not always impaired.

The onset of this condition is generally after an attack of hemiplegia, usually slight, and when it has been thought to be primary it has probably been the sequel to an attack of paralysis so trifling as to escape definite notice. Sometimes it has been preceded by sudden slight hemianæsthesia. Most patients who have presented the typical form have been in middle life.

Athetosis differs little from the spastic movement so common after hemiplegia in children. The slowness of the movements is its chief distinction, with the slighter degree of weakness and the fact that the movements are less related to voluntary movement. These distinctions are not absolute. The disorder of movement after hemiplegia may be identical in character with athetosis, and both are essentially the same in general relations. In what is termed 'athetosis' the cerebral lesion is so placed as to cause in most cases no distinct hemiplegia ; but its occurrence is indicated by the sudden symptoms which precede the slow movement. Typical athetosis may succeed hemiplegia.

PATHOLOGY.—It is probable that the seat of the lesion in athetosis is generally the optic thalamus or its neighbourhood. The sudden onset of the disease and the slight affection of sensation are in favour of this explanation of cases in which there is no distinct hemiplegia. Charcot believed that all post-hemiplegic chorea-like movements depend on the implication of fibres outside the optic thalamus. In a case of simple ataxy after hemiplegia—an analogous condition—the writer has found a cicatricial sclerosis extending across the optic thalamus, and probably left by a patch of softening. The symptoms, perhaps, may be produced in various ways, since the regulation of the motor processes in the cortex is complex. One cause may be a state of disordered action of the motor cells of the cortex, due to a prevention of the upward influence from the cerebellum. The great frequency of such 'mobile spasm' (as it may conveniently be termed) after infantile hemiplegia, when the lesion involves the white substance as well as the central ganglia, and even the motor cortex, suggests that this form may be due to disordered action of imperfectly controlled structures capable of greater recovery of function than in adult life.

PROGNOSIS.—This is unfavourable ; but slight cases may improve and more use of the hand be regained by practice.

TREATMENT.—Nervine tonics may do some good. The continuous current has been said to lessen the spasm, but, as a rule, it fails. The positive pole may be placed on the spine or brachial plexus, the negative on the muscles involved. The sedulous cultivation of the power of ordered movement by regulated gymnastics for the hand probably does more than any other measure. Upward massage sometimes seems to be of slight service. But the condition is one very little amenable to treatment.

W. R. GOWERS.

ATONY } (ἀ, priv. ; and *τόνος*, tone). Terms
ATONIC } implying want of tone, power, or vigour of particular organs, especially of those which are contractile. *See* TONE, Want of.

ATRESIA (ἀ, priv. ; and *τίρημι*, I pierce).—Absence of a natural opening or passage, whether congenital or caused by disease.

ATROPHY, GENERAL.—SYNON.: Marasmus.

DEFINITION.—Atrophy means, etymologically, simply want of nourishment (*ἀ, priv.*; and *τροφή, nourishment*), but the term is commonly applied to the condition resulting from this—namely, wasting. *General atrophy* is used to denote wasting in which the whole body participates. All acute diseases are accompanied by emaciation. The use of the word 'atrophy' is, however, confined, as a rule, to cases where the interference with nutrition has been gradual, and the loss of flesh consequently slow.

ÆTIOLOGY.—Atrophy is common enough at all periods of life. In infants and children it is due, in the majority of cases, to chronic functional derangements which interfere with the digestion and elaboration of food. Less frequently it is a consequence of organic disease. In adults general atrophy seldom results from any other cause than organic disease. In old age atrophy is a common consequence of the degenerations of tissue which accompany the decline of life. The interference with nutrition may, however, be aggravated by the presence of disease.

In *infants under twelve months old* there are four principal causes to which persistent wasting can usually be referred—namely, unsuitable food; chronic vomiting (gastric catarrh); chronic diarrhoea (intestinal catarrh); and constitutional diseases, such as inherited syphilis or tuberculosis. Bad feeding, by setting up a chronic catarrhal condition of the stomach and bowels, is a frequent cause of both vomiting and diarrhoea, but it may produce atrophy without either of these symptoms. When an infant is fed, for instance, with large quantities of farinaceous matter—a form of food which is alike indigestible and innutritious—a very small part only can enter as nutriment into the system. The remainder passes down the alimentary canal, and is ejected at rare intervals in an offensive putty-like mass, or in hard roundish lumps. The child, therefore, although overloaded with food, is really under-nourished, and loses flesh as long as such a diet is persisted in. If, as often happens, diarrhoea or vomiting be set up by the irritation to which the digestive organs are subjected, wasting is more rapid and the danger of the case is increased. Wasting, indeed, will be found in every case where the food selected is unfitted for the child, and thus it is not infrequently seen in infants who are fed upon milk and water alone. The casein of cow's milk is difficult of digestion by many infants on account of its tendency to coagulate into a large firm clot like a lump of cheese. In this respect it differs from the curd of human milk, which forms light small flocculent coagula, and is digested without difficulty. Special preparation is therefore generally required to render cow's milk a suitable diet for a young child.

It is not only, however, unsuitable food which is a cause of atrophy in infants. Catarrh of the stomach and bowels may be present, although the feeding is in all respects satisfactory. Infants are excessively sensitive to chills, and catarrh of their delicate digestive organs is easily excited. Now, catarrh of a mucous membrane is always accompanied by an increased flow of mucus, and this alkaline secretion soon begins to swarm with bacteria which set up decomposition in the food taken. Gastric disturbance from this cause may be seen in new-born infants, who thus are rendered for the time incapable of digesting even their mother's milk. In such cases the fault is usually attributed to the milk, which is said to be unsuited to the

child; and the mother is compelled, much against her will, to wean her baby and feed it in a different way. So long as the derangement continues, however, no food appears to agree, and the child often after a time dies exhausted.

Between the ages of *one and three years* atrophy is commonly associated with rickets. In these cases the wasting is noted chiefly about the chest and limbs, for the belly is large and swollen from flatulent accumulation. At this age children are still liable to waste from catarrh of the stomach and bowels; indeed, rickets is itself often complicated by such derangements. Sarcoma of the internal organs is also sometimes found at this time of life, and is attended with extreme emaciation.

After the fifth or sixth year chronic pulmonary tuberculosis begins to appear. Cases of heart-disease as the result of acute rheumatism are also more frequently seen. Diabetes mellitus, too, is sometimes met with. All these diseases may produce much interference with nutrition.

From the time that the child begins to take other food than that furnished by his mother's breast, he is liable to worms in the alimentary canal. The presence of worms is frequently accompanied by loss of flesh, not, perhaps, so much on account of the parasites themselves, as on account of the derangement of the digestive organs which is associated with them. Emaciation due to this cause may sometimes be extreme.

In the *adult* marked atrophy is almost invariably a sign of serious organic disease. All chronic ailments are not accompanied by marked wasting. Purely local diseases lead to little loss of flesh unless they affect some part of the digestive apparatus, or of the glandular system which is concerned in the elaboration of nutritive material; or otherwise directly influence the processes of nutrition. Thus, emaciation quickly results from gastric ulcer or chronic dysentery, but chronic tuberculosis may produce little diminution in weight so long as the disease remains a limited local tuberculosis, and so long as there is no pyrexia, diarrhoea, or profuse expectoration. The most marked atrophy is produced by general tuberculosis, cancer, and the infective diseases such as syphilis; by those which set up a persistent drain upon the system, such as severe albuminuria, chronic hemorrhages, and long-continued suppurations; or by those which directly impede the passage of nutritive material into the blood; and in the latter class of diseases, influences which act directly upon the thoracic duct, such as obstruction to its passage from pressure by aneurysm or other tumour, must not be overlooked. There is a form of atrophy sometimes seen in hysterical females, depending upon disordered innervation, in which the most extreme emaciation may be reached. Such cases are marked by a dislike to food which may amount to absolute loathing. See NEURASTHENIA.

ANATOMICAL CHARACTERS.—The most marked post-mortem appearance in this condition is diminution or loss of fat, especially of the subcutaneous adipose tissue; and this is accompanied by wasting of the tissues and organs generally. The histological elements are reduced in size without undergoing, as a rule, actual numerical diminution. With the atrophy is often associated a certain amount of fatty degeneration.

SYMPTOMS.—The symptoms of general atrophy are loss of flesh, loss of colour, and loss of strength,

combined with other special phenomena arising from the particular disorder to which the impairment of nutrition is due.

TREATMENT.—The treatment of general atrophy consists in removing, if possible, the impediment to efficient nutrition. In the case of a child the diet must be selected with care. Excess of farinaceous food is to be avoided, and cow's milk must be sterilised and if necessary diluted with barley water. Warm woollen clothing should be insisted upon, and special attention should be paid to the warmth of the feet. Any gastric or intestinal derangement must be at once remedied, plenty of fresh air should be obtained, and perfect cleanliness strictly enjoined. In an adult the disease which is the cause of the malnutrition must be sought for and submitted to treatment. Efforts should be made, on the one hand, to arrest any drain upon the system; and, on the other hand, by a judicious arrangement of the dietary and by attention to the eliminatory organs, to remove all obstacles to nourishment. Even in cases of organic and incurable disease much benefit may often be derived from due observance of physiological laws.

EUSTACE SMITH.

ATROPHY, LOCAL.—This condition signifies atrophy of a *part* of the body which may be produced by various causes.

Physiological Atrophies.—These form a distinct class, where atrophy of a part of the body takes place in the ordinary course of development. Such are the wasting of the thymus gland in early life, and of the mamme and sexual organs after a certain age. Most commonly the atrophy is here closely connected either with the involution or perhaps the development of some correlated organ; but it is not possible to say what the nature of this connection is, whether one of nutrition or innervation.

Acquired Atrophies.—The conditions thus distinguished possess most interest for the practical physician. Wasting of any part of the body during life, when not physiological, usually depends either upon some interference with the blood-supply, or some disturbance of innervation; but to these must be added, in the case of organs which have an active and continuous function, disuse or overstimulation. Deficient blood-supply, which causes atrophy, may be produced by the obstruction of a nutrient artery, especially if it be gradual, since sudden blocking will produce more complicated phenomena. Constant pressure is a cause of atrophy, because it interferes both with the blood-supply and with the vital actions of the tissue-elements. Intermittent pressure, on the other hand, by causing hyperæmia, is more likely to lead to hypertrophy. Moreover, inadequate renewal of blood—that is, filling of the vessels, even to excess, with venous blood—or venous engorgement, though at first it may cause enlargement, mostly leads to atrophy in the end; as is seen in the intralobular zones of the liver in cases of disease of the heart obstructing the circulation. Many forms of atrophy in old age are clearly dependent upon senile obstruction of the arteries, for example that of the skin, spleen, and kidneys. The instances of atrophy from disturbed innervation are less easy to discriminate, except where there is actual paralysis. In diseases where the nutritive centre is destroyed—progressive muscular atrophy and infantile spinal paralysis—loss of power in the muscles is accompanied by wasting, far more rapid than that which results from disuse

alone. Division of the nerve of a limb produces rapid wasting of the muscles no longer used, and this is accompanied in the end by some diminution in the size of the bones and accessory parts. Local atrophy of the skin is sometimes seen in regions limited by the distribution of a nerve, especially some branch of the fifth; and more extensive atrophy of one side of the face or head, equally marked out by nervous distribution, and resembling some cases of congenital atrophy, has also, though rarely, been observed. In such cases the writer has found anaesthesia of parts corresponding to branches of the fifth nerve, but absence of muscular paralysis, such as would be due to affection of the seventh nerve.

Disuse produces atrophy only in organs whose functions are active and constant, such as nerves and muscle. Nervous tissue wastes constantly, and sometimes rapidly, when impulses cease to traverse it. This is seen not only in the nerves of paralysed limbs, but even in the nerve-centres where any interruption of the nervous channels, either above in the cerebrum, or below in the nerve trunks, is followed by degeneration, ending in atrophy, of the whole nervous tract leading from the cerebral cortex to the peripheral termination—so-called *secondary degeneration* of the cord. In muscular tissue the wasting is almost as constant, but hysterical paralyses make an exception, the helpless limbs preserving their nutrition in a surprising manner. In organs whose functions are intermittent or periodic, disuse does not appear necessarily to produce atrophy, as is seen in the testicles, ovaries, and mammae.

That excessive stimulation or overwork may produce atrophy is seen in degenerative diseases of the nerve-centres arising from undue mental activity; and of the sexual organs from excessive indulgence. Overwork of muscles very rarely produces atrophy; but the writer has seen instances where special muscles were exposed to strain while the general nutrition was low. Atrophy of muscles is said to occur in soldiers when badly fed, after long exhausting marches.

Unexplained Atrophies.—Cases of local atrophy occur of which it is impossible to give any satisfactory explanation. Such are the conditions known as linear atrophy of the skin; some remarkable cases of atrophy of bone, especially of the skull (*fragilitas ossium*), and of some parts of the cerebrum. Atrophy of the thyroid gland appears to be certainly the cause of the disease called myxoedema, but the cause of the original atrophy is unknown.

It is possible that deficiency of special kinds of food may lead to atrophy of special organs—thus deficiency of lime may make the bones soft, and deficiency of iron arrest the development of blood-corpuscles; but even these familiar instances must be accepted with a little reserve. In the same way it is still doubtful whether any special drugs, such as iodine, can produce atrophy of special glands.

PATHOLOGY.—Wasting may occur simply, or as a consequence of change of substance, or from the intrusion of some new material; in other words, there may be *simple* atrophy, atrophy from *degeneration*, or atrophy by *substitution*. The first is probably rare; generally some change of substance occurs. The most frequent degenerative process is fatty degeneration; the albuminous substance being replaced by fatty matter, which, if afterwards absorbed, leaves a void. Organs thus

affected may be apparently enlarged, though the original substance is wasted. Atrophy from substitution is seen when the connective tissue of an organ, for instance, increases, compressing and destroying the other tissue-elements; and these not being renewed when the newly formed connective tissue contracts, the whole organ is diminished in bulk. This is seen in all the changes called cirrhosis or fibroid degeneration, as in cirrhosis of the liver and kidneys. *See FIBROSIS.*

TREATMENT.—No general rules can be laid down for treating all cases of local atrophy. Where the blood-supply is deficient, we have rarely any means of supplementing it; where innervation is at fault, it is seldom under our control. In general, harm rather than good results from any attempt to attract blood by artificial irritation. In the case, however, of atrophy from disuse of the nervo-muscular system, a line of treatment, and more especially of prophylaxis, is very early indicated; this is, to keep the muscles in exercise by artificial means, particularly by electricity, or by the processes of friction and kneading combined with passive movement (*see* MASSAGE). In this way so much of the atrophy as is due simply to disuse may be checked for the future, and even the former loss repaired. We shall, moreover, never do harm by attempting to supply some special elements of food which appear to be deficient, as iron for the blood and phosphorus for the bones or nervous system.

J. F. PAYNE.

AUDITORY MEMORY.—*See* APHASIA.

AUDITORY NERVE, Diseases of.—*See* EAR, Diseases of; and HEARING, Disorders of.

AURA (*αἶρα*, a breeze).—A peculiar sensation, subjective in origin, immediately preceding an epileptic or hysterical convulsion, and named respectively *aura epileptica* and *aura hysterica*. The word was adopted at a time when the arteries were believed to contain air because the sensation described was that of the passage of cold air from the trunk or extremities to the head; but it has been extended so as to include any phenomenon, whether sensory or motor, that ushers in a fit of epilepsy or of hysteria.

AURAL DISEASES.—*See* EAR, Diseases of.

AUSCULTATION (*ausculto*, I listen).—A method of physical examination, which consists in listening over various parts of the body, either by the direct application of the ear (*immediate auscultation*), or by the aid of special instruments (*mediate auscultation*), for the purpose of studying certain sounds produced in health and disease. *See* PHYSICAL EXAMINATION.

AUSCULTATORY PERCUSSION.—A method of physical examination, in which the sounds elicited by percussion are studied by means of auscultation. *See* PHYSICAL EXAMINATION.

AUSTRALASIA.—The portion of Polynesia lying between 10° and 50° S. latitude, and 110° E. and 170° W. longitude, which may be said to include Australia, Tasmania, New Zealand, the Fiji Islands, the New Hebrides, and some less important islands.

Australia.—The climate of the vast continent of Australia, which is partly temperate and partly

tropical, depends, first, on its latitude, and, secondly, on its conformation, the mountain ranges being distributed along the coast-lines, especially on the eastern shores. In the interior, which is comparatively flat, and believed to be for the most part a sandy desert, there is great heat and little rain. The hot winds from the interior are often sufficient, in the summer, to raise the thermometer to 127° F., and on the amount of protection from these enjoyed by the various towns depends their climate. There are also sea-winds from the N. and N.E. The southerly winds, prevailing chiefly from November to February, blow from the Antarctic Circle, and are cold winds of great velocity, ending in heavy thunderstorms. In the tropical region the rainfall is from November to April; and in the temperate, which lies to the east and south, it prevails only in the winter season.

The following are among the principal towns or centres to which invalids proceed:—

Adelaide, the capital of South Australia, lat. 35° S., long. 135½° E. This place suffers from great heat and drought. The mean temperature is 65°, the maximum 115°, and the minimum 34°; the range being 81°, and the mean daily range 20°. The humidity is 60 per cent., and the rainfall 21 inches, but varies greatly. The soil is sandy.

Brisbane, the chief town of Queensland, lat. 27½° S., long. 153° E. The climate is almost tropical. The mean temperature is 70°, the maximum is 108°, the minimum 34°; the range 74°, with a mean daily range of 21°. The rainfall is 51 inches, and the mean humidity 76 per cent. Queensland is for the most part elevated; and the climate of the Darling Downs, on an average 2,000 feet above sea level, is considered very fine. The townships at Toowoomba and Warwick in this region may be recommended.

Melbourne, the capital of Victoria, lat. 38° S., long. 145° E. It has the reputation of being a healthy and agreeable residence; the climate being dry and temperate, and far cooler in summer than that of Sydney. Mean temperature 57°, maximum 111°, minimum 27°, showing a range of 84°; daily range 18°. Mean humidity 72 per cent. Rainfall, 26 inches.

Perth, in Western Australia, very healthy, but as yet little suited to the requirements of invalids. The temperature is 63° (mean), and the rainfall 30 inches in 110 days.

Sydney, the capital of New South Wales, lat. 34° S., and long. 151° E. The climate of New South Wales is clear and dry, the temperature depending more on the altitude than on the latitude. The plains in the interior, swept by hot winds, are very dry, while the coast-districts have abundant rain. Mean temperature 62·5°, maximum 107°, minimum 36°; range 71°; mean daily range 14°. Humidity 72 per cent., and rainfall 50 inches. Paramatta, on Port Jackson, is drier and cooler than Sydney (Lindsay); and the Illawara district, including the stations of Eden and Twofold Bay, is suitable for invalids; but the finest climate in Australia for pulmonary cases is to be found in the Riverina, a district lying between Queensland, the Blue Mountains, and the central desert, consisting of rolling prairie and undulating downs, with a rainfall ranging from 5 to 24 inches, the average being 14 inches, and a clear bright atmosphere, most exhilarating in its effect on invalids, who at the

settlements of Deniliquin, Bathurst, Menindee, and the various sheep-farms, pursue an open-air life by day, and often by night, with great advantage. The district of Bourke, 500 miles from Sydney, is also well spoken of.

Tasmania.—Tasmania lies 150 miles south of Australia, between lat. $40^{\circ} 40'$ and $43^{\circ} 38'$, and is mountainous, with a deeply indented coast-line. The climate is more temperate and equable than that of the south coast of Australia. In winter the cold is sufficient to produce thin ice in the low lands, and snow-showers in the higher ranges. The mean temperature of *Hobart Town* on the S.E. coast is 54° , the summer mean being 62° and the winter 47° . The rainfall varies greatly, from 100 inches at Macquarie Harbour on the W. coast to 24 inches at Hobart Town, distributed over 145 days. The prevalent winds are from the N.E. and S.W. The climate is favourable to infant life, and the country is regarded as a sanatorium for invalids.

New Zealand.—New Zealand lies between $34^{\circ} 50'$ and $47^{\circ} 50'$ S. lat., and consists of a North and a South besides smaller Islands. The North Island is for the most part volcanic, and abounds in hot springs, which are extensively used, and active craters, which impart an important influence to its climate. The South Island contains a lofty range of snow-clad mountains, whose lower slopes form on the eastern shore a series of terraces known as the Canterbury Plains, and other fertile regions.

The climate is mild and bracing, but decidedly of a windy character, and not suited for all invalids: at Auckland in 1876 no calm day was recorded, the prevalent winds being W.S.W. The mean temperature of the North Island is 58° , of the South 54° . The maximum varies from 87° , at Christ Church, to 75° , at Hokitika, and the minimum from 25° to 34° in the South Island. Cold is as a rule unknown in the North Island, while in the South there are a few snowy days each year on the coast. The rainfall varies from 32 inches in 135 days at Christ Church, to 131 inches in 186 days at Hokitika.

Fiji Islands.—The Fiji Islands, partly of volcanic and partly of coralline origin, have a tropical climate, moderated by the trade-winds, so that the mean temperature does not exceed 80° , the minimum being given as 65° . The rainfall is chiefly from October to April—the hot season—and varies from 124 to 215 inches in 170 days. See CLIMATE, Treatment of Disease by.

C. THEODORE WILLIAMS.

AUTO-INTOXICATION (αὐτός, self; τοξικόν, poison).—DEFINITION.—By auto-intoxication is meant a process of poisoning brought about by some substance or substances produced by the body of the poisoned animal.

DESCRIPTION.—Certain diseases which are not strictly included in this definition are sometimes quoted as examples of auto-intoxication.

(1) In the first place, the specific infectious fevers must be mentioned. The symptoms of these diseases are produced by products of the specific microbe, and not by substances formed in the course of the patient's metabolism. (2) Secondly, the so-called gastric and intestinal auto-intoxications are, for an analogous reason, not to be regarded as instances of this kind of poisoning, for the condition of the urine and fæces in these cases shows that there

is an increase of bacterial putrefaction in the alimentary canal. When this putrefaction is lessened, these symptoms disappear; and we are therefore justified in concluding that these are cases of intoxication by substances manufactured in the alimentary canal by micro-organisms. The fact that these substances are produced to a lesser extent in health shows that these morbid conditions may be merely exaggerations of the ordinary events of healthy life, but is not a valid reason for including these cases among the auto-intoxications.

Instances of auto-intoxication will be here considered (1) experimentally, and (2) clinically.

1. Experimental.—The most striking instance of auto-intoxication among higher animals is the series of events resulting when the animal breathes in a closed space, although in the final asphyxia other factors probably play a more important part.

Among bacterial forms of life auto-intoxication is a common occurrence under artificial conditions of growth. It is produced, not by deficient excretion of a poison from the cell, but by the accumulation of a poisonous substance in the culture-medium. The causes of this auto-intoxication are known in some cases. For instance, *Bacillus pyocyaneus* produces an enzyme-like body, which can be precipitated and purified by alcohol, and which destroys fresh cultures of *Bacillus pyocyaneus*, as well as those of other micro-organisms.

One argument for the occurrence of auto-intoxication in man and higher mammals is based on experimental evidence. Various excretions, secretions, fluids, and solids of one healthy animal are toxic for another animal of a different species. It is possible, therefore, that these products may be toxic also for the animal by which they are formed. Failure of excretion, or of destruction, of these poisons might lead to auto-intoxication. Generally speaking, substances poisonous for one species of mammal are also poisonous for another species: various species, however, differ largely in susceptibility; moreover, in the case of certain toxins which destroy the red corpuscles, there are many exceptions to this generalisation. Only in a few cases has an animal product been shown to be toxic for the organism which forms it. These cases are mentioned in the following account of the toxicity of various organic fluids and solids.

Urine.—The urine of many animals (especially of herbivora) is toxic for other animals. The symptoms produced by intravenous injection of human urine into a rabbit are: myosis, acceleration of respiration, frequency of micturition, lowering of temperature, and finally coma, with or without convulsions. French observers give the average minimal lethal dose as 40 c.c. per kilogramme of animal. The toxicity of human urine varies under different circumstances in health. It is diminished in old age and by fasting, and increased by muscular exercise. The toxicity is said to be least when sleep begins, and much greater during waking hours. Qualitative differences between the urine of the night and day have been recorded. The urine contains a large number of substances which are able to produce morbid symptoms and ultimately death. In view of recent research, it is probable that much of the toxicity of the urine is due to the salts of potassium which it contains. After dialysis, however, the urine is stated to be still toxic. Further, a certain connection has been traced in some cases between the toxicity of the urine and the

amount of conjugated sulpho-acids present. The latter are evidence of increased bacterial putrefaction in the alimentary canal.

On comparing the figures given by various authors as representing the average toxicity of normal urine, they are found to differ very widely one from another. Bearing this in mind, the statements that have been made as to the toxicity of the urine in various pathological conditions must be received with great caution. These statements are for the most part unconfirmed.

Blood.—The serum of one species of animal is, generally speaking, toxic for another species—apart from its hemolytic, coagulative, or agglutinating powers. Albuminuria has been recorded as the result of the injection of serum from another animal of the same species but of different sex. Doubtless some of the effects of the serum are due to changes in the plasma brought about by clotting; among these is the rise of temperature. Recently it has been found that the blood of the cat after clotting becomes toxic for the same animal; and Richet has recorded a choreiform condition in dogs brought about by repeated injection of dog's blood.

Among other toxic substances should be mentioned *bile*, *saliva*, *gastric juice*, and extracts of *supra-renal capsules*, *thyroid gland*, and *muscles*. Sterile sweat is said to be non-toxic (Brieger).

The kidneys excrete many poisonous substances from the body, but there is another organ—the *liver*—whose function as a destroyer of poisons can be regarded as well established. The liver has the power of destroying certain poisons (e.g. some alkaloids) when introduced into the blood-stream. Doubtless the liver acts similarly in health; and we might therefore expect that in diseases of the liver auto-intoxication would occur from impairment of this function. Other organs have been supposed to act as destroyers of poisons, notably the thyroid gland; they will be mentioned later.

2. Clinical.—The occurrence of auto-intoxication has been thought probable in the following conditions:

1. Breathing in a closed space.—The mere excess of carbon dioxide in the air does not account for the resulting symptoms; for air containing a large quantity of this gas may not give rise to them, although air that has been 'vitiated' by breathing does, and yet contains less carbon dioxide than in the first case. It is not quite clear, moreover, to what extent the symptoms are due to a substance excreted by the lungs, or by the skin. It is of course possible that the bacteria of the skin may produce a toxic substance.

2. Diseases of the kidneys.—In *uræmia* and *puerperal eclampsia* some of the symptoms at least are probably due to auto-intoxication. With regard to uræmia, it may be said that no substance known to occur in the urine is capable of producing all the symptoms of this condition, though many (urea,

salts of potash and ammonia, carbamic acid, &c.) have been suggested as causes.

3. Diseases of the Liver.—Among these may be mentioned *acute yellow atrophy*, and cases of severe *jaundice* from any cause. It is stated that bile-salts cause diminished frequency of the heart-beats—a common symptom in jaundice.

4. Diseases of the thyroid gland.—*Myxædema*, *cachexia strumipriva*, *exophthalmic goitre*.

5. Diabetes.—The causes of *diabetic coma* are unknown, though acetone and β -amido-butyric acid have been suggested.

6. Addison's disease.—Many physiological experiments can be explained by the hypothesis that the supra-renal capsules render certain poisonous substances innocuous. It is therefore conceivable that the abolition of the function of these glands would lead to an auto-intoxication.

7. Burns.—Some of the symptoms of severe burns are due to the circulation in the blood of some poisonous substance, produced at the seat of the injury.

8. Gout.—The statement that gout is an auto-intoxication does not alter the degree of obscurity in which the pathology of this disease is shrouded. The presence of sodium urate in the various joint structures is doubtless a cause of the local symptoms.

In the present state of knowledge, auto-intoxication should be regarded as a suggestive conception founded upon analogies. Although its occurrence is probable in certain diseases, the experimental evidence is insufficient to allow of very definite statements as to the conditions in which auto-intoxication plays a part, and still less as to the substances producing it.

WALTER MYERS.

AUTOPHONIA (*αὐτός*, himself; and *φωνή*, the voice).—A physical sign obtained by studying the modifications of the resonance of the observer's own voice during auscultation. See PHYSICAL EXAMINATION.

AUTOPSY.—See NECROPSY.

AX-LES-THERMES, in the South of France. — Thermal waters. See MINERAL WATERS.

AXON (*ἄξων*, an axle).—The largest and longest of all the processes proceeding from a nerve-cell is called the axon. The axon is the channel by which impulses are conveyed from the cell.

AZORES; St. Michael.—Warm, very moist, equable climate. Mean winter temperature 58° F. Prevailing winds N. and E. See CLIMATE, Treatment of Disease by.

AZOTURIA.—A condition of the urine in which there exists an absolute and relative excess of urea, without accompanying pyrexia. See URINE, Morbid Conditions of.

B

BABINSKI'S SIGN.—This term is applied to a modification of the superficial plantar reflex, obtained by stroking or tickling the sole of the foot. In normal persons the reflex starts with flexion of the toes, followed by dorsiflexion of the ankle and flexion of the knee and hip (*flexor response*). In patients who are the subject of degenerative changes in the lateral columns of the spinal cord (pyramidal tracts) the reflex occurs more slowly and starts with extension of the great toe, followed by the other toes, with increase of the curvature of the plantar arch (*extensor response*). This phenomenon was first described by Babinski: it is of value in differentiating cases of organic disease of the lateral columns from so-called 'functional' conditions.—See SPINAL CORD, Diseases of.

W. CECIL BOSANQUET.

BACILLI (*bacillum*, a little rod or staff).—SYNON.: Fr. *Bacilles*; Ger. *Bacillen*. See BACTERIA.

BACILLI, various.—See under Diseases produced by them.

BACILLUS COLI COMMUNIS.—SYNON.: *Bacterium coli commune*; Colon-bacillus.

The *B. coli communis*, a constant inhabitant of the intestinal canal of man and several other mammals, is a short rod with rounded ends, sometimes ovoid in shape, 2 or 3 μ in length by 0.4 to 0.6 μ in breadth. 'Thread' forms, 6 to 10 μ or more in length, are of common occurrence in artificial cultures. The bacillus is feebly motile—its activity varying greatly in different specimens: it possesses from 2 to 10 lateral flagella. The organism stains well with all the aniline dyes, but is decolorised by Gram's method.

It is a facultative anaërobie, growing readily upon artificial nutrient media (even those containing as much as 0.15 per cent. of phenol) at temperatures ranging from 20° to 42° C. Spore-formation does not occur. Its thermal death-point is 60° C., the time of exposure being ten minutes.

CULTURAL CHARACTERS.—*Gelatine Plate-cultures.*—The superficial colonies, at first round and almost transparent, later become opalescent, spread rapidly and show irregular crenated margins, with granular surface marked by ridges and furrows, and heaped-up centres. The deep colonies are spherical, of a pale buff colour, and granular in appearance. The gelatine is not liquefied.

Gelatine Streak-culture shows a white shining flat growth, with irregular margins, spreading out laterally from the needle-track. In old cultures the growth becomes iridescent, and a hazy 'ground glass' appearance of the adjacent gelatine is noted.

Gelatine Slab-culture.—White spherical colonies develop in the entire extent of the needle-puncture, afterwards becoming confluent. Growth also spreads over the surface of the gelatine from the point of entrance. One or more gas-bubbles may appear in the depths of the medium.

Sugar-gelatine Shake-culture.—Abundant formation of gas (CO₂ and H) is evidenced by the appearance of numerous bubbles of gas in the substance of the medium.

Bouillon.—A uniform turbidity is noted in this

medium after from 10 to 12 hours, a fair amount of deposit being apparent at the bottom of the tube at the end of two days. A peculiar faint odour is given off from bouillon-cultures, and the presence of indol can be demonstrated in from 24 to 72 hours.

Agar Streak-culture shows a luxuriant growth in the form of a raised thick moist shining greyish-white sticky layer.

Streak-culture on Inspissated Blood-serum.—Growth as on agar. The medium is not liquefied.

Potato-culture.—A thick slimy layer of a buff or brownish-yellow colour develops upon the surface of the potato. The substance of the potato may or may not be discoloured.

Milk culture.—The milk is curdled in from 24 to 48 hours as a result of the action of the lactic acid produced by the growth of the bacillus.

PATHOGENESIS.—The *B. coli communis* is pathogenic for the ordinary laboratory animals, i.e. rabbit, guinea-pig, mouse, although variations in virulence among different specimens are quite common. Those strains which are but slightly pathogenic can usually be exalted in virulence by means of intraperitoneal or intrapleural passages. Virulent cultures, when introduced into serous cavities, frequently cause a true septicæmia, and the colon-bacillus can then be recovered *post mortem* from the heart-blood. When less virulent cultivations are injected, or the subcutaneous method of inoculation is employed, death ensues either from toxæmia or from pyæmia, small masses of the bacilli being found in the spleen and other organs, giving rise to localised collections of pus; in other cases again, a local abscess only is produced—the animal ultimately recovering.

In man the colon-bacillus has its normal habitat in the intestinal canal; in any other situation its presence must be regarded as abnormal, and pus-formation be expected. It has been shown by Malvoz that, given an injury to the bowel-wall, the colon-bacillus is capable of penetrating it at that spot and so making its way into the peritoneal cavity, even when such injury is insufficient to cause perforation, thus explaining the common occurrence of peritonitis as a sequel to strangulated hernia or appendicitis. Sanarelli showed that the colon-bacillus isolated from a patient suffering from typhoid fever possesses a higher degree of virulence than one obtained from a normal individual; and it has been further shown that a greater proportion of the pyogenic processes complicating the later stages of enteric fever are due to the colon-bacillus than to the typhoid-bacillus.

Many urinary infections such as pyelitis, ureteritis, and cystitis, notably that following operations upon the rectum, are also due to the presence of the colon-bacillus in an abnormal situation. See APPENDIX VERMIFORMIS, Inflammation of.

JOHN EYRE.

BACTERIA (*βακτήριον*, a rod).—SYNON.: Fr. *Bactéries*; Ger. *Bakterien*.—INTRODUCTORY.—Bacteriology is concerned with one of the lowest orders of living things, for which the name *bacteria* has been adopted. In its original application the term bacteria was restricted to such of these organisms as were of an elongated rod-like

shape, but its connotation has been extended to include all members of the same group irrespective of form, and it has thus come to have a generic significance. In fact it is practically synonymous with 'micro-organism,' which in its strict sense might be applied to any living body of microscopic dimensions. In medical parlance, however, both terms are limited to those minute organised structures which may be associated causally or otherwise with disease, and indeed are generally understood to refer only to such as are presumably of a vegetable nature. The terms 'microbe,' 'microzyme,' 'germ,' &c., may be regarded as equivalent.

It is well to indicate at the outset that the scope of the present article is limited. Any systematic description of individual bacteria is excluded, each being dealt with under the heading of the morbid condition with which it is specially associated. The important subjects of toxins, antitoxins, and immunity are also separately considered. The aim has been to present in a succinct form some of the more important facts as to the morphology and life-history of bacteria and the methods of investigation employed.

Moreover, reference will especially be made to those bacteria which are concerned in the production of disease in man. It is, however, noteworthy that myriads of bacteria exist in the air, in water, and in the soil, and that only a small proportion have any pathogenic effect. The vast majority display other activities, many of which have a far-reaching beneficial result. For example, bacteria are the chief agents in the decomposition of effete animal matter, in the purification of sewage, in many processes of fermentation of great industrial importance, and in the fixation of free nitrogen and oxidation of ammonia and nitrites to form compounds adapted for the nutrition of higher organisms.

NATURE OF SPECIFIC CONTAGIA: CONDITIONS OF CAUSAL RELATION. (For general considerations see INFECTION.)—The grounds on which any micro-organism can justly be regarded as the contagium or actual cause of disease may be briefly indicated. 1. It must be constantly associated with the disease, being present in the fluids or tissues of the diseased animal, and in the virus by which the disease is communicated. It may indeed be in a different form in the virus, e.g. in the form of spores, while in a more developed form when in the full activity of disease-production. We should expect that if there is a characteristic lesion, such as an eruption, or some special affection of one or more organs, the organisms should be concentrated there. 2. The organism should be capable of absolute isolation and separate cultivation outside the body; and then, being introduced in the form of a pure culture, it should produce the same disease and be capable of re-transmission to other individuals.

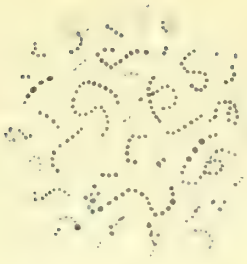
It is obvious that this rigorous scientific proof can be attained almost solely in the lower animals, seeing that we cannot, in the more serious diseases, thus experiment on man. And even in the lower animals we meet with numerous inherent difficulties. 1. Some micro-organisms can be cultivated outside the body only with great difficulty, others at present not at all. 2. The specific disease-producing properties may be greatly diminished, or even lost, in artificial conditions of growth. 3. A further difficulty, which forms an especial barrier to

the study of human diseases, is found in the fact of the variations of disease when communicated from one class of animal to another, and in the relative degree of susceptibility to disease of different classes, or even of species or individuals of a given class. Thus we find (a) that a virus which causes a general blood-poisoning in one class of animals will produce only a local lesion in another, (b) that none of the conditions produced in lower animals may correspond with the disease as we are familiar with it in the human subject, or again (c) that some human diseases are transmissible to animals only with difficulty, or not at all.

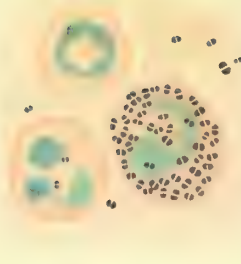
If, by analogy in all essential points, a disease comes into the group of contagious specific diseases, and is strictly comparable with one in which a bacterium has been proved to be the actual virus; if, further, there is constantly present in the body, and especially if in the specific lesion and in the virus, an organism having definite characters, we may provisionally regard it as the essential contagium. And still more may we do so if we find that the disease can be transmitted with the same characters to another individual by employment of fluid or tissue which contains this organism, and that it is not transmitted if the fluid or tissue employed for inoculation does not contain the organism, although derived from an animal suffering from the disease and containing the organism in other parts.

Of the diseases affecting man, anthrax and tuberculosis may be mentioned as examples of conditions, the bacterial origin of which is absolutely proved. Leprosy is one in which the final proof of inoculation of isolated cultures is yet wanting, but is the only missing link. Relapsing fever is an instance of inference from transmission of the organism and the fever to monkeys. Syphilis is an example of a disease in which there exists evidence from analogy only, although some of the organisms discovered may prove to be the virus.

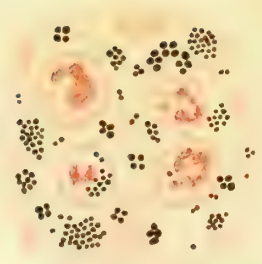
GENERAL MORPHOLOGY AND BIOLOGY.—*Higher and Lower Forms.*—In the consideration of bacteria it is well to regard as a separate order, somewhat higher in the grade of vegetable life, those forms which show a higher degree of complexity and greater variation in their forms or stages of development under different conditions of life. Hence it is convenient to speak of the *higher* and the *lower* forms of bacteria, and to describe them more or less separately. Apart from minor details the higher bacteria differ in two important characters from the lower forms—in structure and in function. 1. In structure the elements which constitute the higher forms are more intimately related to each other. They form filaments which may be septate or non-septate, and which may or may not show a true branching. In every case the component parts of each filament are closely bound up with one another in a common sheath; and in the more highly developed members of the group the filaments present a uniform appearance with no trace of their individual constituents. In structure, therefore, they represent a transitional stage between the more complex mycelium of a fungus and a thread of bacilli in which each component is a separate individual. 2. While morphologically the constituents of the higher forms have become mutually more dependent, there has also been developed a greater functional differentiation or specialisation. Thus one extremity of the filament



1.

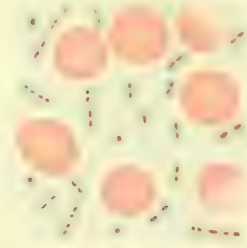


2.



3.

1. *Streptococci* from culture—some swollen and degenerating (arthrospores?)—methylene blue.
2. *Gonococci* in pus—flattened diplococci mostly within leucocytes—methylene blue, eosin.
3. *Staphylococci* and tetrad cocci in pus, Gram's method, saffranin.



4.

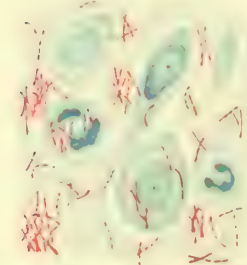


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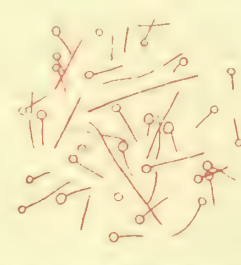


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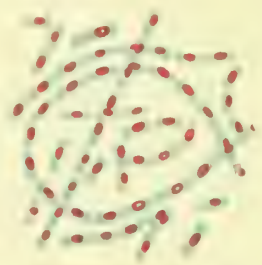
4. *Pneumococci* in blood of rabbit—red diplococci of lanceolate form within blue capsules—p.132.
5. *B. pestis* from culture—chains of short bacilli with polar staining—methylene blue.
6. *B. diphtheriae* from culture—involution-forms in lower field—methylene blue.



7.



8.



9.

7. *B. tuberculosis* in sputum—slender bacilli irregularly stained—Ziehl-Neelsen method.
8. *B. tetani* from culture—uniform bacilli with terminal spores unstained—dilute carbol-fuchsin.
9. *B. anthracis* from culture—large blue bacilli containing red spores—special method, p.132.



10.



11.



12.

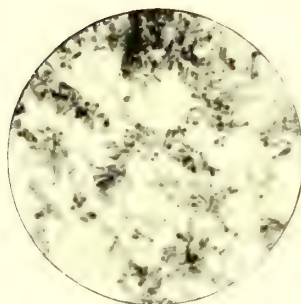
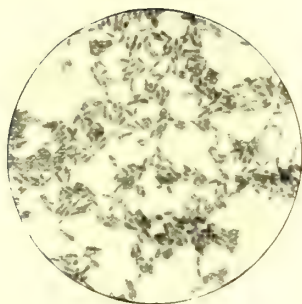
10. *B. typhosus* from culture—numerous lateral and terminal flagella—Muir's method, p.132.
11. *Sp. cholera* from culture—scanty terminal flagella, coccoid bodies in lower field—same method.
12. *Sporidium* of relapsing fever in blood—long, delicate, wavy spirilla—methylene blue, eosin.

Magnified 1000 diameters.

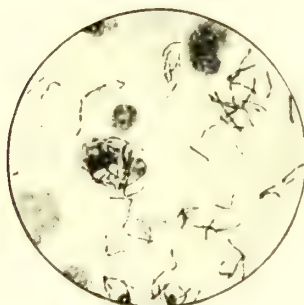
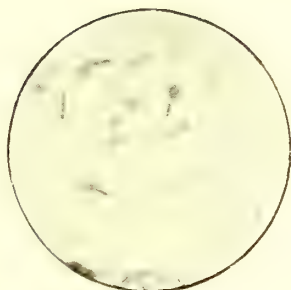
R. M. Muir

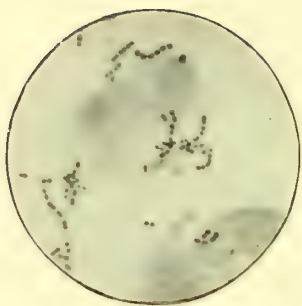
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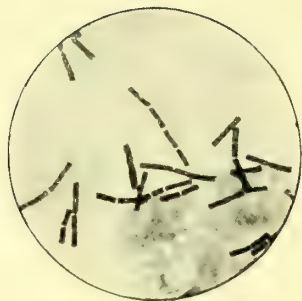


B. typhosus

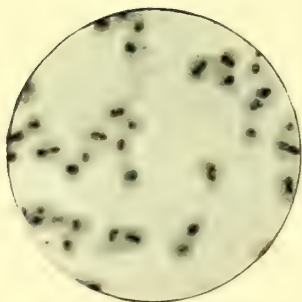




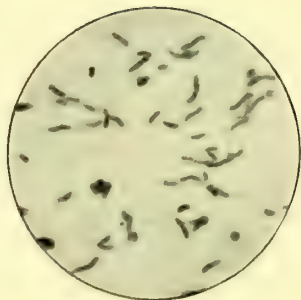
Aspergillus



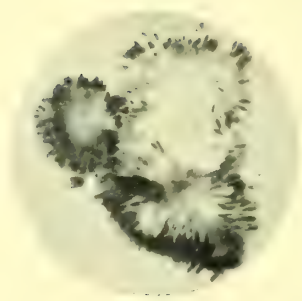
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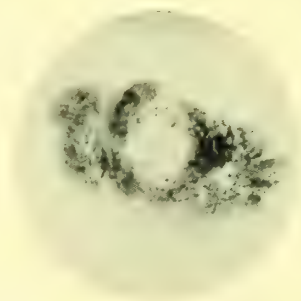
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may be concerned merely in fixation, while certain of the free extremities or hyphæ are set apart for the reproduction of the species. In the lower forms any individual may take upon itself the part of multiplying to form other individuals, but in the higher forms this power is confined to a limited number of elements.

The higher bacteria include such forms as *Beggiatoa*, *Leptothrix*, *Cladothrix*, and *Streptothrix*, but as a group they are less concerned in human disease than are the lower forms. Of this group most of those known to be pathogenic belong to the *Streptothriciæ*, and among them the member of greatest pathogenic importance is *Streptothrix actinomycetes*. See *ACTINOMYCOSIS*.

The lower bacteria are minute unicellular organisms belonging to the lowest class of chlorophyll-less algæ, and constituting the most elementary form of vegetable life. It may be noted that in some non-pathogenic bacteria chlorophyll granules have been described, but the general fact is as stated. They multiply by fission; hence the name *Schizomycetes*, but for many of them there is also a mode of propagation or a resting-stage constituted by the formation of spores. In structure they consist of a protoplasmic body having peculiar chemical and physical characters, enclosed within a cell-envelope. Many are spontaneously motile.

Form, Size, and Grouping of the Cells.—The bacterial cell may present great variety in *form*, but is for the most part symmetrical, though slight degrees of asymmetry are very common. If there is no great disparity in length between any one diameter and the others, the cell has a more or less spherical or ovoid form, and the organism is spoken of as a *coccus*. If, however, one diameter definitely exceeds in length the other two, the cell has a cylindrical or rod-like form to which the terms *bacillus* and *spirillum* have been applied—the former when the rod is straight or only slightly curved, the latter when the curvature is more marked. Even in the case of the cocci considerable diversity of form is met with. Some are approximately spherical, except when undergoing processes of division or degeneration. Others have one diameter rather longer than the other two—e.g. the *Pneumococcus*, in which also one extremity is rounded and the other so pointed that the term ‘*lanceolatus*’ has been applied to it. In others one diameter is rather shorter than the other two, so that the organism has a flattened shape and discoid outline, as in the *Gonococcus*, *Diplococcus intracellularis meningitidis*, &c. Among the bacilli there is still greater variation in form. Some are so short as to resemble oval cocci, as in Friedländer’s pneumobacilli, while others attain a relatively great length, as in anthrax. On cross-section some are exceedingly slender, while others are relatively thick. In some the extremities are rounded or even pointed, as in clostridium-forms, whereas in others they are abruptly truncated. Some present the appearance of a uniform cylinder with parallel sides; others show bulbous swellings throughout their length or at one end. The spirilla may occur, as in cholera, in the form of minute rods, each with a single curve and tapering towards the ends, or, as in the spirillum of relapsing fever, in the form of delicate spiral threads, each showing several twists or curves.

In *size* they are extremely minute. Most of the rounded forms do not exceed 1μ or $\frac{1}{25000}$ inch in diameter. The largest of the pathogenic rod-like

forms, the *Bacillus anthracis*, rarely exceeds in length the diameter of a red blood-corpuscle, and most bacilli are less than half that length. In thickness they vary from a maximum of about 2μ to a minimum of less than 0.5μ . Most of the pathogenic spirilla are very short, rarely exceeding 2μ in length, but the slender spirillum of relapsing fever may reach a length of several times the diameter of a red blood-corpuscle.

The arrangement and *grouping* of the individual cells depend partly upon the viscosity of the cell-envelope and pericellular material, partly upon the mode of fission, and partly upon the consistence of the medium in which the organism is growing. Hence, after fission, some cocci tend to remain in pairs or groups of four and eight, some form chains, others irregular bunches. Some bacilli occur chiefly in pairs, others in long filaments, and others singly. Growth in a fluid medium favours the development of characteristic grouping of bacterial cells.

The Cell-Protoplasm.—In the unstained condition bacteria appear as sharply outlined, highly refractile, almost colourless bodies. The cell-protoplasm has a marked affinity for the basic aniline dyes, and when stained it may present a uniform homogeneous appearance, but is frequently granular, showing differences in the staining-reaction in different parts. More profound changes affecting the entire cell may be observed during the processes of spore-formation and of involution or degeneration. In some bacteria, and very typically in the bacillus of bubonic plague, the protoplasm appears to be condensed at each extremity or pole, and there takes on a deeper stain than in the centre. Such organisms are said to show *polar staining*, and the deeply stained parts are sometimes spoken of as *polar granules*. In other bacteria, e.g. *B. diphtheriæ*, the cell-protoplasm shows a series of condensations throughout its length, so that each bacillus appears to be made up of a row of deeply stained granules, and many resemble a short chain of streptococci. When suitably stained these granules may take on a different colour from that of the rest of the cell-protoplasm and are known as the *metachromatic granules*. Vacuolation may be observed in the cell-protoplasm of many bacteria. The presence of a *nucleus* is disputed. The more deeply staining granules in the cell-protoplasm have been regarded as the first indication of nucleus-formation (Migula). From observations on some of the larger non-pathogenic forms, it has been inferred that the greater central part of the bacterial cell corresponds to the nucleus, and that the cell-protoplasm is represented by a peripheral layer within the envelope, so scanty as to elude observation in the majority of instances (Bütschli). It is an open question how far these appearances correspond to normal structural features, and how far they are due to degenerative changes in the bacteria or are produced by artificial means. In the fixation of films for examination an interchange may take place between the cell-protoplasm and the surrounding fluid, so that phenomena of *plasmolysis* may occur, giving rise to granulation, vacuolation, and apparent nucleation (Fischer). The presence of a protoplasmic meshwork (Bütschli) and of lymph-spaces (Migula) in the bacterial cell has also been described.

Chemistry of the Bacterial Cell.—The chemical composition of the protoplasm appears to vary in different species, and in the same species under different conditions. Nencki extracted from the

bodies of certain bacteria a peculiar albuminous substance which he termed *mycoprotein*, and from the spores of anthrax a somewhat different substance, *anthraco-protein*. Buchner also isolated certain nitrogenous bodies or proteins, which differed from those of Nencki in containing phosphorus, and other observers have described various albuminous constituents. Granules of definite chemical composition have been found in the cell-protoplasm—e.g. sulphur in *beggiatoa* and *thiothrix*, starch-granules in *B. amylobacter*, droplets of oil or fat and iron-compounds in other organisms.

Bacterial Pigments.—Not a few organisms, chiefly non-pathogenic forms, when grown in mass show distinctive colours, pink, red, yellow, orange, green, violet, black, &c. Whether the pigment belongs to the protoplasmic body or to its envelope cannot in most cases be determined, as the small size of the cells renders the colouring matter invisible when they are examined singly. In many relatively large forms the pigment appears in the cell-contents; in others it is deposited in little granules between the cells, and in others it is held in solution by the medium. Certain conditions of growth, e.g. relation to oxygen, temperature, &c., may modify or arrest the production of pigment. The chemical nature of bacterial pigments is not always the same. Some belong to the group of lipochromes—pigments analogous to those derived from animal fat and widely distributed throughout the animal and vegetable kingdom (Zopf)—and include bacteriopurpurin found in the large sulphur-bacteria. They are insoluble in water and frequently colour the growths of bacteria in nutrient media. Others are soluble in water, and are therefore not confined to the colonies, but are diffused throughout the media. They have been isolated from the fluorescent bacteria and in a dry state consist of an amorphous albuminoid substance. Other bacteria furnish colouring matter of which the chemical nature is still less known. The occurrence of chlorophyll in some forms has been noted.

The Cell-Envelope.—The protoplasm of all bacteria is enclosed in, and usually sharply defined by, a delicate membrane. In some of the higher forms a distinct sheath can be seen, but in many of the lower forms the envelope is revealed only in certain conditions or by special reagents such as iodine, tannic acid, &c. As regards its physical characters the envelope appears to consist of two layers. The inner layer is a thin, highly flexible, and elastic membrane which readily adapts itself to any movement of the bacteria. The outer layer is less defined and has in many species a peculiar glutinous or viscid consistence, so that after fission the individual elements may form characteristic groups. To the partial solution of this material is due the intercellular substance of zoöglea-masses.

In some bacteria the envelope has a tendency to become greatly swollen, forming the so-called 'capsule.' This occurs most typically during the parasitic stage of the organism, and is usually absent or only barely evident in cultures. It forms a characteristic structural feature of Fraenckel's pneumococcus, Friedländer's pneumobacillus, and other forms. It remains unstained by ordinary aniline dyes, but may be stained by special methods (*vide infra*). An important point is that capsules appear to have greater powers of resisting necrosis and degeneration than the bacteria themselves, so that in an old empyema, for example, it is often

possible to detect the persistent capsules of pneumococci, although their protoplasmic bodies have entirely disappeared.

In its chemical constitution the envelope appears in most instances to be a carbohydrate related to cellulose, or to contain also chitinous substances related to the cellulose-group in their reaction. The envelope of some (e.g. *B. xerosis*) is supposed to be largely fatty (Neisser).

Motility and Flagella.—When examined in suspension in a fluid, many bacteria are found to be actively *motile*, showing spontaneous movements of a darting, rotatory, or undulatory character. In some of the higher forms (e.g. *Beggiatoa*) independent movements are due apparently to the contractility of the cell-protoplasm: but in practically all the lower motile forms special structures termed *flagella* are known to be present; and most of the spirilla, many bacilli, and a few cocci are furnished with them. They consist of relatively long and exceedingly delicate wavy filaments, which proceed from the sides or ends of the organism. When terminal they may be single or double or manifold, and placed at one end only or at both ends of the cell; when lateral they are often very numerous. Whether they are direct prolongations of the cell-protoplasm or merely modified appendages of the envelope is an open question. In a few very large organisms the unstained flagella may be seen, but in the majority they are not revealed except by special methods of staining (*vide infra*).

Motility is not usually characteristic of an organism throughout the whole cycle of its life-history. It becomes modified by age and by sporulation, and this modification is associated with loss of the flagella. As a rule young cultures show most active motility, but in suitable conditions movement may persist and flagella be demonstrated after many days. In older cultures flagella tend to disappear and all movement to cease. The onset of sporulation in a motile organism usually coincides with shedding of most of the flagella and a loss of motility, e.g. *B. tetani*. But without loss of flagella motility may be affected by such conditions as the nature of the medium, the temperature, and the presence of agglutinins in antisera. On this depends the characteristic reaction of the bacteria to the serum in such diseases as typhoid fever, cholera, &c. The movements of bacteria in the phenomena of positive and negative chemiotaxis are not necessarily associated with the presence of flagella.

Involution-Forms.—As age advances and in certain conditions of environment all bacteria tend to undergo degenerative changes, and the intracellular vacuoles and granules above noted probably indicate the onset of such changes. But many organisms in process of degeneration undergo very remarkable transformations which are known as 'involution-forms.' Thus pneumococci and gonococci in cultures rapidly lose their definite shape and size, become swollen and globular, and show irregular staining. Many bacilli also undergo similar changes, notably those of plague and diphtheria. In the former extreme variations are possible, the small oval bacilli becoming swollen to rounded, ovoid, or pyriform bodies, and even adopting the appearance of a branching mycelium (Muir). In the latter, granularity and segmentation of the protoplasm, and a tendency to form large club-shaped bodies are prominent features. The cholera-spirillum too may undergo irregular swelling and clubbing, and

many individuals become transformed into faintly staining coccoid bodies.

Reproduction.—(a) In the higher forms certain elements are set apart for reproductive purposes, and multiplication occurs from conidia borne on special hyphæ. (b) In the lower forms reproduction takes place by direct division or fission of the cells, the segments being often of nearly equal size. Transverse fission is the rule in the simpler rod-shaped and spiral forms, and the daughter-cells elongate, so that, if the segments do not separate, they come to form long filaments. Long or short chains of streptococci are similarly produced. But in some of the rounded forms division may occur in two planes at right angles to one another, thus forming groups of four (*tetrads*), or in three dimensions forming cubical groups of eight (*sarcinae*). During the process of fission a constriction appears in the plane of approaching segmentation, and a delicate line may be seen across the cell-protoplasm cutting it in two. The constriction increases and the delicate transverse line undergoes a gelatinous swelling until two individuals appear, separated by an interval of varying width. The ultimate arrangement of the individual elements depends partly on the planes of fission, partly on the viscosity of the cell-envelope, and partly on the consistence of the medium, and can therefore be regarded only as a subordinate character, although it affords a basis for an elementary classification. The rate of multiplication varies greatly in different species and in different conditions of growth. The bacillus of tuberculosis grows relatively slowly, but most organisms proliferate with great rapidity in a favourable environment. It has been stated that a bacillus may attain its full growth and divide in less than half an hour, and at this rate one individual would reproduce itself nearly seventeen million times within twelve hours, and 281,475,000,000,000 times in twenty-four hours.

Spores.—The production of spores and the properties with which they are endowed are among the most important facts regarding bacteria. Discovered at first in some of larger size, the process is now known to be common to many bacilli and to some spirilla. It is doubtful if any of the cocci ever form true spores. But from known facts in the life-history of many species in which the existence of spores is not proved, there is a possibility that in them also there exists a like capacity, given favourable conditions of life. Spore-formation is to be regarded as a resting stage in the life-history of a bacterium, which, therefore, may pass through two stages, (1) a vegetative, spore-free stage in which rapid multiplication may occur, producing other vegetative forms, (2) a spore or resting stage in which no reproduction takes place, for each organism produces only one spore, and each spore, when it germinates, gives rise to a single vegetative form.

The conditions of spore-formation are somewhat peculiar, and appear to depend upon a combination of circumstances, some of which are adverse and others conducive to vegetative growth and development. For example, sporulation does not in most instances occur until vegetative growth has been arrested by some change in the medium, probably due rather to the formation of substances inimical to growth than to the exhaustion of the nutriment. But this alone is not sufficient, for spores need not form unless the temperature is

maintained within favourable limits, and the limits for spore-formation are even more restricted than those for vegetative growth, though the optimum temperature in each case practically coincides. Free oxygen also appears to be essential to some forms, and even in certain anaërobic organisms sporulation is assisted by its presence. It cannot therefore be held that spore-formation occurs only in the absence of the optimum pabulum; it is more properly regarded as a normal phase in the life-history, occurring especially in certain favouring conditions of growth.

The mode of spore-formation can best be studied in some of the larger bacilli, such as *B. subtilis* or *B. anthracis*. Within the protoplasm of the vegetative cell, and usually towards one end, there appears a minute rounded granule of higher refractive index which gradually enlarges at the expense of the parent cell until it becomes a mature spore. The spore is always smaller than the original cell, and is apparently derived from condensation and chemical alteration of its protoplasm. This method of spore-formation is known as 'endogenous,' and the bacteria in which it occurs are spoken of as *endosporous*. In some cases (e.g. *B. anthracis*) the parent cell soon becomes granular and shrunken, stains more faintly, and finally disappears, so that the spore is left enclosed in the shrivelled remains of the original cell-envelope. In other cases (e.g. *B. tetani*) the protoplasm of the bacterial cell may show little alteration long after the spore has been formed. Swelling may and frequently does take place at the site of the spore, so that the organism may appear beaded, and the swelling may be so considerable that the diameter of the spore may be twice as great as that of the cell, or even more (e.g. *B. tetani*). This appearance is apt to be exaggerated owing to the different refraction of the spore and the cell-protoplasm.

In form spores are usually ovoid, more or less elongated, sometimes nearly spherical. In structure they consist of a highly refracting protoplasmic mass with great endowments of vitality and peculiar staining-reactions, surrounded by a capsule or cell-wall resembling that of the parent cell but of greater resisting capacity. Their staining-reactions differ from those of the cell-protoplasm, and may do so to a remarkable degree. Solutions which stain ordinary bacteria do not stain the spores; but by selective staining spores may take on a different colour from the rest of the cell (*vide infra*). In one of the common bacteria of water (*B. erythrosporus*) the unstained fresh spores normally show a definite colouration of a reddish tinge.

When placed in conditions favourable to vegetative life, spores begin to *germinate* and reproduce cells like those from which they originated. The process begins by the spore losing its high refraction, and continues by its gradual expansion to the size and form of the vegetative cell. The spore-capsule may be ruptured in various ways, sometimes being torn right across so that at each end of the growing cell part of the membrane appears as a cap, sometimes splitting longitudinally or at one end only. More rarely the capsule is simply stretched and absorbed by the expanding cell within. As growth advances movement appears in motile forms, and then follow multiplication and grouping of the vegeta-

tive forms, culminating later in spore-formation. Often only a few hours elapse between the first onset of germination and active vegetative growth.

Another form of spore-formation has been described in certain species which do not produce endospores, e.g. *Spirillum cholerae*, *streptococcus*, &c. According to this view certain of the vegetative cells pass into a resting stage and adopt the rôle of spores. They are then known as *arthrospores* (Hueppe). Structurally they may be of much the same form as the ordinary vegetative cell, or some modification of it. Functionally they are supposed to represent a more permanent condition by means of which the species is propagated, and from which subsequent germination may occur. They are also supposed to have greater powers of resisting adverse influences than the ordinary vegetative cell. It is doubtful if such a degree of specialisation of individuals ever occurs among the lower bacteria; and, indeed, all the known facts regarding the so-called arthrospores are consistent with known variations in morphology and biology among different individuals in the same culture.

Classification of Bacteria.—Many attempts have been made to arrive at some satisfactory division of bacteria, but hitherto with very limited success. The difficulty arises in great measure from defective knowledge of their life-history and of the variations which they may show in different conditions of growth. Most of the attempted classifications are valueless and are also unscientific. The only true basis is, of course, a botanical one, and such properties as the production of colour, of fermentation, or of disease are useless except as indicating provisional distinctions. The nomenclature usually adopted is founded on certain morphological data—chiefly the form of the cells and the arrangement commonly seen in their growth—but it must be understood that the names given are merely convenient appellations and not scientific distinctions. Reference has already been made to the division of bacteria into higher and lower forms.

1. The higher forms are characterised by greater structural independence of the elements and greater functional specialisation. They include such forms as *Beggiatoa*, *Thiothrix*, *Crenothrix*, *Cladothrix*, and *Streptothrix*. For more detailed information the article on ACTINOMYCOSIS may be consulted. See also STREPTOTHRIX.

2. The lower forms are divided into three main groups according to the form of the cells. (1) *Cocci* or *micrococci* consist of rounded or ovoid cells, and are subdivided according to their most usual grouping. Thus *diplococci* are formed when after fission the cells tend to remain in pairs; *tetrads* when after fission in two planes at right angles they are arranged in groups of four; and *sarcinae* when after fission in three dimensions cubical masses of eight are formed. *Streptococci* consist of cells forming single rows or chains of varying length, and *staphylococci* of cells forming irregular groups like bunches of grapes, all depending on the mode of fission and degree of subsequent cohesion among the individuals. (2) *Bacilli* include those cells of an elongated rod-like form, whether straight or slightly curved, and whether single or in filaments. Sometimes the term *bacillus* is restricted to the motile forms, and bacterium

applied to the non-motile (*Migula*), or, again, bacilli are regarded as characterised by endogenous sporulation and bacteria by its absence (Hueppe). (3) *Spirilla* also consist of cylindrical cells in which the curvature is more pronounced, either in the form of short rods with a single curve, as in the spirillum of cholera, or in the form of long filaments with several curves, as in the spirillum of relapsing fever. The terms *vibrio* and *spirochete* have also been introduced to distinguish degrees of curvature (Flügge), presence and characters of flagella (*Migula*), and relation to spore-formation (Hueppe). Such terms are of no practical value, and the possibilities of confusion which they introduce are sufficiently obvious.

Constancy of Species and Variation among Bacteria.—Modern bacteriology postulates the constancy of species among bacteria, and all known facts are in harmony with this conception. Moreover, direct attempts to transform one species into another have met with no success; even such closely allied forms as the typhoid- and colon-bacilli are not interchangeable, nor can the cholera-spirillum be transmuted into other species of spirilla.

Within certain limits, however, morphological, biological, and pathogenic variations are possible and indeed common among different members of the same species in different conditions. Morphological variations are not infrequent, particularly in regard to size; difference in shape may become extremely marked in those bacteria which show involution-forms, and most of the higher forms exhibit cyclical changes in the course of normal development and growth. Flagella are usually absent at some stage of the life-history of motile organisms, and many other characters may undergo change. Biological variations are still more common, and depend chiefly on varying conditions of growth. Thus differences are seen in rate of development, in appearance of cultures, in production of colour, in fermentative effects, and in power of resistance. The capacity for spore-formation may be temporarily or even permanently lost by exposure to certain influences. Most common of all is variation in the pathogenic properties of bacteria according to the attenuation or intensification of their virulence.

On the other hand it must be observed that among members of what was originally regarded as one species, differences so profound may be met as to suggest a group rather than a single species. Thus *Streptococcus pyogenes* has been regarded as a species distinct from *Streptococcus erysipelatis*, and the term *B. coli communis* has been held to include several closely allied species. Without discussing such questions fully it may be permissible to say that all recent evidence points rather to considerable potential variation within definite species than to a multiplicity of slightly differing and ill-defined species.

Conditions of Growth and Vitality.—Certain conditions are essential to bacterial life, while others have a deleterious effect. It is important to note that within certain limits growth may be inhibited while vitality remains unimpaired or is only modified, so that conditions which arrest growth and development do not necessarily destroy bacteria. Different species present great variety in their power of resisting adverse conditions, and even in the same species different organisms may show marked variation in resisting capacity. When spores are present, the capacity for resistance is enormously increased,

as compared with that of the corresponding vegetative forms.

Nutritment.—Most forms of plant-life obtain the essential elements of their food-supply from very simple chemical combinations, e.g. carbon from carbon dioxide, nitrogen from nitrates, &c. But most bacteria feed and grow on the more complex organic substances that constitute the tissues of plants and animals. All the pathogenic bacteria are not only capable of deriving their nutrition from the highly complex proteid diet that animal tissues and fluids afford, but they grow best within the body of the living animal. They are therefore essentially parasitic in character. But nearly all the pathogenic forms are also capable of a saprophytic existence outside the living body, and for growth and development in these circumstances they require the presence of some proteid or carbohydrate substance. Hence in the preparation of artificial culture-media (*vide infra*) such material must be supplied. A great host of microbes that are rarely or never parasitic lead a saprophytic existence in effete animal and vegetable products which they resolve into more primitive chemical compounds. The great majority of bacteria, therefore, require at least a trace of some organic substance on which to live, but many saprophytic organisms can and do thrive on purely inorganic materials. Reference has already been made to the remarkable powers of certain bacteria in the soil, not only of elaborating ammonia and nitrites into assimilable nitrates for the maintenance of plant-life, but even of fixing free nitrogen and restoring it to the general nitrogenous circulation.

When the food-supply is deficient or unsuitable, growth is arrested, and if these conditions persist, either the bacteria die or sporulation occurs, and in this form their life is prolonged. An important means by which the pabulum is rendered unsuitable is by the diffusion into it of the products of bacterial growth. On artificial media an organism may at first grow luxuriantly, then cease growing, and finally die or spore, not because the pabulum has been exhausted, but because deleterious substances have been formed by its own activity.

But the medium in order to be adapted for bacterial life must also contain a sufficiency of moisture, and the effect of drying is to deprive the organism of an element essential to growth and to vitality. Desiccation not only arrests development, but quickly kills most vegetative forms, though spores may be kept in a dry condition for months or years. Among the vegetative forms there are very pronounced differences in their capacity for resistance; some, as the spirillum of cholera, being destroyed by a few hours' drying, whereas others, and notably the bacillus of diphtheria, may resist drying for weeks or months.

Again, most organisms grow best on a medium that is slightly alkaline to litmus, and the slightest trace of free acid is sufficient in many cases to arrest development (e.g. of the cholera-spirillum). Hence the reaction of the medium is also an important factor in determining the conditions of growth.

Heat.—A suitable temperature is one of the most important conditions for the growth and development of bacteria, and heat is also one of the most potent agencies in modifying or destroying their vitality. The same agency which is essential to growth is also one of the most effective germicides. For any organism there is an optimum temperature at which growth takes place most abundantly, and

there are also upper and lower limits of temperature beyond which growth ceases. The optimum temperature for vegetative growth varies considerably in different organisms, and three main groups may be recognised. (1) The pathogenic bacteria which are definitely parasitic grow best about 37° C.—the normal blood-temperature—or in other words the 'body temperature.' For many such organisms the limits of temperature consistent with growth are very restricted, so that development is arrested at a point a few degrees above or below the optimum. Thus the temperature limits of growth for the pneumococcus and diphtheria-bacillus are about 20° C. or 22° C. to 42° C., for the gonococcus and influenza-bacillus 25° C. to 42° C., and for the tubercle-bacillus 28° C. to 42° C. For others the range is wider, and growth may readily take place at lower temperatures. Thus the limits for the anthrax-bacillus are about 12° C. to 45° C., for the typhoid-bacillus 9° C. to 42° C., and so on. It has been already noted that the limits within which sporulation may occur are even more restricted. Anthrax-bacilli, for example, do not spore below 18° C. or above 42° C. (2) Those bacteria which are normally saprophytic, being concerned in processes of fermentation and decomposition, grow most actively at a lower range of temperature, usually from 18° C. to 22° C. as the optimum—a warm summer temperature, or, as it is sometimes called, the 'room-temperature.' (3) A remarkable group of organisms, known as the thermophilic bacteria, thrive best at such unusually high temperatures as 60° C. to 70° C.

Beyond the points at which growth ceases, there is a variable range of temperature within which the vitality is impaired but not destroyed. In this intermediate condition many vital processes and activities become modified and deranged. Thus the production of colour, acids, toxins, &c., may be altered or arrested, the capacity for spore-formation may be lost, and the virulence of the organism may be markedly attenuated. Temperatures above the limits of growth are probably more effective in producing these changes than those below; in other words excess of heat appears to exercise a more prejudicial effect than mere deficiency.

At certain temperatures the effect of heat is to destroy bacteria. In estimating the lethal effects of any temperature the duration of exposure must also be considered, as a brief exposure to a high temperature may be more destructive than a longer exposure to a lower temperature. Obviously too a lethal temperature is such as to destroy all the individuals exposed to it, so that, when they are subsequently transferred to a suitable medium, no growth whatever takes place. A temperature which is lethal to vegetative forms may be resisted by the same organism in a spored condition. Hence there may be stated for any organism the lethal effects of low and of high temperature for exposures of fixed duration both in the vegetative and in the spore-form. In regard to low temperature, however, it is difficult to determine the freezing death-point, owing to the occurrence of physical changes in the medium and mechanical pressure-effects, but many organisms, even in the vegetative form, may be kept for some time below the freezing-point of water without being killed, e.g. those of cholera, anthrax, &c. The minimum lethal temperature for definite exposures at high temperatures varies greatly for different organisms, and for the same

organism in different conditions. Most spore-free bacteria are destroyed at 60° C. in less than half an hour, and at 100° C. in a few minutes, e.g. the organisms of cholera, typhoid fever, diphtheria, plague, &c. A few organisms that are not known to spore may survive even higher temperatures: thus the *Staphylococcus pyogenes aureus* withstands 80° C. for half an hour before death occurs, and the *Bacillus tuberculosis* has much the same powers of resistance. When spores are present, even 100° C. may be withstood by some for a few minutes, by others for more than an hour.

Alternations of temperature are very prejudicial to bacterial life, although the upper limit may not be high. Repeated exposure to a temperature of less than 60° C. for an hour daily will destroy all organisms, whether spored or not, within a week, and alternate freezing and thawing have a like effect.

These data have reference to the effects of heat on bacteria in a normal moist environment. In a dry condition they can resist much higher degrees of temperature. Even vegetative forms may survive 100° C. for an hour, and spores may withstand 140° C. for several hours.

Oxygen.—Most known species grow best in the presence of free oxygen and are therefore termed *aërobic* bacteria or *aërobes*. Of these some are strictly aërobic, do not grow in the absence of oxygen, and are known as obligatory aërobes; but the majority, while growing best in the presence of oxygen, may also grow in its absence, and are known as facultative anaërobes or as being capably anaërobic. Most of the pathogenic aërobes belong to the latter group. Many organisms, however, thrive best in the absence of free oxygen and are termed *anaërobic* bacteria or *anaërobes*. Here also some are strictly anaërobic while others are capably aërobic, and the more important pathogenic anaërobes, as the bacilli of tetanus and of malignant œdema, belong to the former group. But even obligatory anaërobes may grow in the presence of air when associated with aërobic organisms, and it is supposed that the latter produce substances which render this possible. Moreover some bacteria may be aërobic in certain conditions and anaërobic in others, for thermophilic bacteria thrive aërobically at high temperatures, but only anaërobically at lower temperatures (Rabinowitsch).

The chief means taken to exclude oxygen from cultures of anaërobes are (a) replacing oxygen by an indifferent gas, such as hydrogen or nitrogen, (b) adding some reducing agent to the medium, such as glucose or formate of sodium, (c) using deep tubes of agar or gelatine. It is often advantageous to combine these methods. Further the gaseous products of metabolism which frequently appear in artificial cultures of anaërobes may afford them protection against injurious oxygen-tensions in the media.

Light.—Direct sunlight has a most prejudicial effect on the vitality of bacteria, even when spores are present, and, if the exposure be sufficiently prolonged, its action is germicidal. The violet and ultra-violet rays are the most potent, and their effect is independent of the action of heat, but is conditioned by the presence of oxygen and of moisture. Diffuse daylight and the electric light have similar but less pronounced effects, while the Röntgen rays have not been shown to produce much change.

Chemical agents.—Many chemical substances may

inhibit the growth and development of bacteria, and are known as *antiseptics*; many others have a destructive action and are known as *disinfectants* or *germicides*, but most bodies that are classed as antiseptics are also bactericidal. Among inorganic substances the salts of the metals of high atomic weight (Ritchie) have the most powerful germicidal action, especially the perchloride and periodide of mercury. Among organic bodies the members of the aromatic series, of which carboic acid is a typical example, take highest rank as disinfectants.

MODES OF STUDY.—*Introductory.*—The complete study of any microbe demands a long and technical investigation in the pursuit of which many different methods and processes must be adopted. In all essential particulars the main steps of such an inquiry are the same for all bacteria, but the details vary widely with different organisms. When a tissue or fluid or other material suspected to contain bacteria is submitted for examination, one of the most important points is the *preparation and staining of specimens for microscopic work*. Not only should cover-glass films be made and suitably stained, but unstained moist films must also be examined to determine motility and other characters associated with bacteria in a fresh condition. Sections of tissues also show not only certain characters of the bacteria, but especially their site and relation to the tissue-elements and the processes of tissue-reaction to microbic invasion. The significance of the microscopical examination, however, is less than might be supposed, for histological characters, even when aided by special staining-reactions, do not afford a criterion for discriminating between allied forms. Nevertheless, when these characters are very definite and when the organism is found in constant association with a peculiar change, as in the case of the tubercle-bacillus, its identification in the tissues is comparatively easy.

Fortunately in the case of a large number of micro-organisms there is available a far more perfect mode of study, that of *cultivation or pure culture*, in other words the study of an organism growing outside the body on some artificially prepared medium and isolated from all other species. An incalculable advance on previous bacteriological research was made when this method was introduced; for it was found that by taking advantage of certain conditions of growth not only could a microbe be separated from all others, but that in suitable media successive growths or subcultures could be perpetuated through a long series of generations and maintained in a pure state. It is impossible to exaggerate the importance of this discovery in the advance of bacteriology. By the study of pure cultures there were revealed many morphological characters previously obscure or entirely unsuspected, the physiology and biology of bacteria became for the first time amenable to study, the chemical products of bacterial growth and activity were disclosed, and the way was cleared for the whole vast subject of the experimental inoculation of animals with pure cultures or their products and the acquisition of immunity. By culture, especially when aided by microscopic study, the recognition of various forms becomes more definite and certain; for each organism possesses its own peculiar characters, especially when grown upon or in semi-solid media. These characters comprise the form of the mass, the colour, rate of growth, results and effects upon the nutrient media, the relation to the presence or

absence of oxygen, the production of acids, indol, and other substances, and the capacity of growth on different media and at various temperatures. They may vary for the same organism in different media, and when taken together constitute almost absolute distinctions of most of the various known forms. By the aid of a lens the earliest groups and colonies may be studied, and will often be found to branch and grow in a characteristic way.

In certain groups of bacteria, however, several forms are so closely allied both in morphological and in biological characters that microscopical and cultural distinctions alone fail to afford more than a reasonable presumption of identity. In the case of the *Bacillus diphtheriae*, for example, it is known that other allied and relatively innocuous forms exist, which under the microscope and in cultures are practically indistinguishable, and the same is true regarding the *Spirillum cholerae* and others. Hence any inference based on such data must in certain cases be made with caution until another mode of investigation has been undertaken—the *inoculation of animals* (p. 132).

I. Cultivation of Bacteria.—It is obvious from what has been said that the most essential part of bacteriology is concerned with the study of pure cultures. Hence the means of obtaining them is of the first importance. But micro-organisms are practically ubiquitous, and are present in the air, in water, and in the dust covering all apparatus and utensils, so that every object and material that is to come into contact with bacteria must first be freed from all such adventitious germs by processes of *sterilisation*. And, further, all media must be protected from subsequent contamination by methods adequate to the *exclusion* of extraneous organisms. Moreover in natural processes of infection it is only in certain tissues and in a few conditions that the specific bacteria are found entirely free from admixture with other species, and there is additional risk of contamination when the tissues are obtained after death. Hence methods of *isolation* are required in order that any one organism may be separated from all other varieties that may be accidentally or otherwise associated with it. But an essential preliminary to all methods of isolation and cultivation is the preparation of *nutrient media* in which the growth of bacteria may take place, and this nutriment must be available in such conditions of moisture, temperature, &c., as conform to the special biological requirements of the organism. The study of pure cultures accordingly necessitates: (1) methods of sterilisation and exclusion; (2) preparation of nutrient media; (3) isolation of pure cultures; and (4) maintenance of cultures in conditions adapted for vitality and growth.

Methods of Sterilisation.—Any agency which can destroy bacteria and their spores will render a material sterile, and the most potent of such agencies are either chemical or thermal. Mechanical processes also may be employed to remove bacteria from fluids, as in the separation of organisms from fluid media by special methods of filtration. Obviously all these agencies are not equally suitable in any given case. It is often necessary not merely that adventitious bacteria should be destroyed, but that the apparatus or medium should be left in such a condition as not to interfere with the vitality and growth of the organism which it is desired to cultivate. Chemical disinfectants, therefore, are only of limited utility in the preparation of material for

bacteriological work. See ANTISEPTICS; and DISINFECTION.

The agency which is most effective as a germicide and which leaves behind no deleterious influence is heat. Hence, where possible, sterilisation is best effected by heat or by alternation of heat and cold. Heat may be conveyed either by hot water or by steam at various temperatures, and in this form is far more powerful than when conveyed by dry air at the same temperature.

Methods of Exclusion.—The exclusion of all external organisms is effected mainly in two ways: (a) by sterilised cotton-wool plugs; (b) by the action of gravity.

Preparation and Uses of Culture-Media.—The considerations which regulate the preparation of media suitable for the growth of bacteria outside the living body include an adequate supply of pabulum, a sufficiency of moisture, a suitable reaction, the presence of elements essential to growth, and the absence of inhibitory influences. Such media vary greatly with the varying needs of different organisms, but it is possible roughly to classify those in common use.

(1) *Media containing body-fluids in a natural or only slightly altered condition* are prepared from blood-serum, exudations into serous cavities, aqueous humour, milk, urine, &c. They approximate most closely to the conditions of nutrition during the parasitic stage of bacterial life, and all pathogenic organisms grow best on them. Delicate organisms, such as the pneumococcus and influenza-bacillus, with little saprophytic vitality, may not grow at all on more artificial media. For such organisms 'blood-agar' is also invaluable, and is prepared by smearing the sloped surface of ordinary agar (*vide infra*) with a drop of sterile blood (Pfeiffer).

(2) *Artificial proteid media* are nearly all derived from a watery extract of the flesh of the ox, horse, sheep, calf, or fowl. Not only do most pathogenic organisms grow well on them, but the growths display a greater number of specific cultural distinctions than on other media. With this meat-extract as a basis, fluid media are prepared by the addition of peptone and sodium chloride, and the result is known as *peptone-broth* or *bouillon*. Its most important use is in the growth of organisms for separation of toxins and other soluble products by subsequent filtration. Semi-solid or gelatinised media have the same composition as bouillon with the addition of gelatine or agar-agar: the former are known as *peptone-gelatine* or *nutrient gelatine*, the latter as 'ordinary' agar or agar-agar. The disadvantages of gelatine media depend upon the relatively low melting-point of gelatine, which restricts its use to the cultivation of the more vigorous organisms which can flourish at the ordinary room-temperature from 18°C. to 22°C. But for such organisms there are certain outstanding advantages from the characteristic appearances which most cultures present, among which may be noted the presence or absence of liquefaction, the form of the surface-growth and that along the needle-track in stab-cultures, and the characters of the superficial and deep colonies in gelatine plates. Agar-media possess one great advantage in the fact that they can be incubated at the body-temperature without melting; hence, a greater number of bacteria can be successfully cultivated on them. But there is a certain sameness in the appearance of the growths on agar as compared with the varied cultural features of the corresponding growths on gelatine.

For special purposes various substances may be added to bouillon, gelatine, and agar media—e.g. glycerine for the growth of the tubercle-bacillus, glucose and formate of sodium for anaerobes, lactose for testing fermentative effects, and litmus for indicating the production of acids.

Neutralisation of media derived from meat-extract. The reaction of the crude meat-extract and its derivatives is invariably acid, owing to the presence of organic acids, amphoteric organic bodies, and acid phosphates. The usual mode of neutralisation is to add a few drops of a solution of sodium hydrate or carbonate until the mixture becomes just alkaline to litmus. Such a medium, however, is still acid to a more delicate indicator, such as phenolphthalein, owing to the presence of dibasic sodium phosphate (Na_2HPO_4), which, being alkaline to litmus, has not been neutralised. As the amount of this salt varies, the degree of acidity is unknown and inconstant. To obviate this objection endeavours have been made to obtain media of constant or 'standard' reaction. By the addition of decinormal and normal solutions of sodium hydrate the medium is first rendered neutral to phenolphthalein, and then 'standardised' by the addition of one and a half per cent. of a normal solution of hydrochloric acid—the reaction being still slightly alkaline to litmus. It is found that the optimum reaction for the growth of most organisms is afforded by a medium that is slightly alkaline to litmus and slightly acid to phenolphthalein (Fuller, Eyre, and others).

(3) *Artificial carbohydrate media* comprise chiefly boiled potato and bread-paste. The appearances of growths on potato often reveal characteristic differences between allied species, but the reaction of the potato must be noted, and in special cases modified. Bread-paste is most useful for the cultivation of moulds, torulae, and the chromogenic bacteria.

(4) *Other artificial media* include vegetable infusions and various chemical combinations, such as Cohn's fluid, Pasteur's original solution, Winogradsky's media for the culture of nitrifying organisms, &c. Many are of merely historical interest.

Methods of Separation and Isolation.—(1) *Repeated dilution of the bacteria and mechanical separation of the colonies.*—This is one of the most useful and most commonly employed of all methods, and depends upon a mode of 'plate-cultivation' introduced by Koch. The principle is, that single organisms in suitable conditions give rise to single colonies of the same organism, and that accordingly, if the colonies are sufficiently separated, pure cultures may be obtained from each.

(a) *In ordinary aerobic conditions* the more important methods include Koch's gelatine plates or Petri capsules, Esmarch's roll-tubes, agar plates, successive strokes on agar-surfaces, &c. *Gelatine plates* are prepared by taking three or four tubes of melted gelatine, inoculating the first from the suspected material, then the second from the first, the third from the second, and so on. The fluid contents of each tube are poured out into a separate plate and allowed to solidify. Colonies may appear very abundantly in the first plate, less in the second, and still less in the third; and from the colonies thus separated, pure cultures may be made. *Esmarch's roll-tubes* are prepared in the same way, but instead of being emptied into plates, the tubes are rolled under a stream of cold water, so that the gelatine solidifies on the wall of each tube. *Agar plates* are used in the manner above described, and

can of course be incubated at the body-temperature. *Successive strokes on agar-surfaces* are made by charging a platinum needle with the suspected material and gently stroking the surface of three or four agar tubes or plates, without recharging the needle.

(b) *For anaerobic organisms* the method is essentially the same so far as the processes of dilution and mechanical separation are concerned. Hence gelatine and agar plates and gelatine roll-tubes are all useful so long as means are taken to exclude oxygen. Vignal's method is frequently of value. Three or four tubes of melted agar are successively inoculated and their contents sucked up into separate lengths of quill glass tubing, the ends of which are sealed in the flame of a Bunsen-burner. Separate colonies appear at various intervals, and may be obtained by breaking the glass over them.

(2) *Inhibition of immaterial growths by various agencies.*—Advantage is sometimes taken of the fact that conditions which arrest the growth of some bacteria may only slightly retard that of others. Thus phenolated media have been employed in the isolation of typhoid-bacilli from the stools.

(3) *Separation of spores by the destruction of spore-free bacteria.*—If spores are present in the causal organism, they may be freed from any vegetative forms associated with them owing to their greater powers of resistance. Thus a short exposure to a temperature of 80°C . to 100°C . will allow only spores to survive. This method is often combined with others, and is specially useful for isolation of anaerobes.

(4) *Separation by inoculation of susceptible animals.*—In cases where there is some special difficulty in the way of obtaining pure cultures it may be possible to isolate the organism in the blood or tissues of certain animals. Thus the pneumococcus, an organism of extremely delicate growth, may be isolated in the blood of a rabbit, and the tubercle-bacillus, which is characterised by its extremely slow growth, may be cultivated from certain viscera. The glanders-bacillus too may be readily isolated by similar means.

II. Microscopic Examination of Bacteria.

In the fresh unstained condition bacteria may be examined in a variety of ways. The appearances of colonies in gelatine or agar plates, test-tubes, &c., are best seen under comparatively low magnifying powers. The actual development and mode of growth of any organism should be studied in hanging-drop cultures. The characters of living bacteria and many vital phenomena in which they are concerned may be investigated by means of fresh films examined in fluids under a cover-glass, a warm stage being employed when necessary. By this means the grouping of the cells, the presence or absence of motility, and the processes of chemotaxis, phagocytosis, and agglutination may be conveniently studied.

Permanent preparations are obtained after some preliminary process of fixation with subsequent staining of the bacteria; and these processes are applied chiefly to two forms of preparations, (1) films, and (2) sections of tissue.

Methods of fixation: (1) *Film-preparations.*—Of all modes of microscopic study this is of widest application and of greatest value. Films may be made from almost any material containing bacteria,

and fixation is effected in many different ways according to the nature of the material in which the organisms are present and the special structural features to be examined.

Films from cultures.—Films are prepared from growths in solid media either by first making a dilute emulsion of the growth in a few drops of sterile distilled water, transferring a loopful to the cover-glass and spreading it out in a thin layer, or by first placing a loopful of water on the cover-glass and mixing with it a very minute quantity of the growth. The organisms should be so diluted as to render the film almost invisible. When growths occur in fluid media or in the water of condensation from solid media, films may be made by spreading a loopful of the fluid directly over the cover-glass. In any case the film is dried by heating over the flame of a Bunsen-burner at the highest temperature the hand can comfortably bear. The amount of heat required to thoroughly dry the film is usually also sufficient to fix it, but, if not, it may be passed two or three times quickly through the flame.

Films from animal fluids and tissues.—From pus, sputum, and other highly cellular fluids, films are best obtained by smearing a loopful over the cover-glass with as little manipulation as possible. Some parts will be found too thick for examination, but the thin parts between should suffice. In the case of watery exudations, e.g. ascitic and pleuritic effusions, and in the case of watery secretions, e.g. urine, any bacteria present are usually so scanty as to elude direct observation. The fluid should therefore be allowed to stand for several hours in conical glasses, or better, centrifuged and a loopful of the deposit then spread out over the cover-glass. From the solid organs and tissues films are prepared by gently rubbing the portion to be examined over the cover-glass. The fixation of the films thus prepared may be effected according to one of two methods named, (a) the dry, and (b) the wet, according to the initial step in the process. (a) In the dry method the films are fixed by gentle heat in the manner above described. In this way the bacteria are well preserved, but more highly organised structures, e.g. leucocytes and tissue-cells, are not so well seen. (b) In the wet method the film is never allowed to dry, but is at once placed in some fixing solution, the best being a saturated solution of mercuric chloride prepared by boiling this salt in a solution of sodium chloride (0.75 per cent.). In this the films remain from two to four minutes, and they are then washed in salt-solution and in spirit to remove all crystals of mercuric chloride. They are now ready for staining.

Films from blood.—Special methods of preparation and fixation are required. The skin having been thoroughly cleansed and rendered aseptic, a small puncture is made, and as much blood as will lie in the loop of a platinum needle is transferred to the centre of one cover-glass. Another cover-glass is placed above the first, and, if both are clean, the blood should spread in a thin film between them. They are then rapidly separated, and in the dry method they are dried by waving them in the air. In order to preserve the cellular constituents the film must be fixed either in a saturated solution of mercuric chloride for several minutes, or in a mixture of alcohol and ether in equal proportions for half an hour, or in the hot-air steriliser at 120° C. for the same time. The wet method of fixation is applicable to blood-films also.

(2) *Fixation of tissues.*—For the examination of bacteria this is best accomplished by placing thin slices in a saturated solution of mercuric chloride for twelve to twenty-four hours. The pieces of tissue are then transferred to methylated spirit, and finally embedded and cut in paraffin so that the section may be as thin as possible. A good result may also be obtained by direct fixation in methylated spirit or absolute alcohol. The full details of these various processes would occupy too much space to be given here.

Methods of Staining.—Owing to differences in their chemical constitution the various structural elements of the bacterial cell differ in their reaction to colouring agents, so that it is necessary to consider methods of staining as applied to (1) the cell-protoplasm, (2) the cell-envelope or capsule, (3) flagella, (4) spores.

(1) *The protoplasm of the bacterial cell* resembles the nuclear chromatin of the animal cell in showing a marked affinity for the basic aniline dyes, e.g. thionin and methylene blue, gentian violet, fuchsin, &c. The simplest form of staining reagent is a watery or alcoholic solution of one of these dyes, and many bacteria take up such a stain with great readiness and intensity, provided that a sufficient interval of time is allowed to elapse and a sufficient concentration of the stain is employed. But a more complex process is usually indicated. In the first place all bacteria do not stain with equal readiness, and some resist the action of staining substances to a marked degree. Hence it becomes necessary to make use of mordants which aid the penetrating power of the stain and tend to fix it in the bacteria. The mordants in common use include carbolic acid, aniline oil, tannic acid, and various metallic salts and alkalis. The effect of heat should be noted as accelerating and intensifying all staining processes, and the result of lengthening the duration of the exposure to any stain, or of increasing the strength of the solution, is much the same as would be produced by the addition of a mordant. In the second place, when bacteria are stained in sections of tissue or in films along with other cellular structures, the tissue-elements are apt to take up the stain so deeply as to obscure the organisms, and accordingly various methods have been devised to remove the stain wholly or partially from the tissues and leave the bacteria coloured. This may be effected by the use of suitable decolourising agents, among the more important of which are sulphuric and other mineral acids, acetic acid, clove-oil, alcohol in the form of methylated spirit or absolute alcohol, and warm water. Very frequently use is made of the combined action of more than one decolourising agent, just as more than one mordant may be employed. After decolourising it is usually advisable to counterstain the tissue-elements in such a way that their structure is shown in some colour different from that in the bacteria themselves. The combinations of stain, mordant, decolouriser, and counterstain are very numerous, and only a few of the more typical examples can be given here. Among the more important simple stains may be noted *carbol-thionin blue*, which consists of thionin blue (1 gramme) dissolved in 100 c.c. of a watery solution of carbolic acid (1 in 40). One volume should be diluted with three volumes of water before use. Stain for about five minutes, wash thoroughly in water, and, if necessary, decolourise in water slightly acidulated with

acetic acid. *Dilute carbol-fuchsin* is prepared by diluting Ziehl-Neelsen's carbol-fuchsin (*v. infra*) in ten volumes of water. Stain for about five minutes, wash, and, if necessary, decolourise in warm water. In *Löffler's methylene blue* 30 parts of a saturated alcoholic solution of methylene blue are added to 100 parts of a watery solution of potassium hydrate (1 in 10,000). Films may be stained for five minutes or more; sections for several hours, as they are not readily overstained, and, if required, can be decolourised in a weak solution of acetic acid.

Some bacteria lose the stain so readily that they may become decolourised in the process of dehydration with alcohol. To obviate this Weigert suggested that sections, after being stained in an alkaline solution of gentian violet, should be dehydrated with pure aniline oil, cleared in xylol, and mounted in balsam. Another method, applicable to sections cut in paraffin, was recommended by Greenfield and Muir in the previous edition of this article. The sections, which ought to be as thin as possible, are floated on the surface of a weak solution of the aniline stain, and the liquid is very gently heated. The sections become quite flat, and the stain is seen to penetrate into the tissue. After a few minutes they are washed well on the surface of water, placed on a slide, and allowed to dry. The paraffin is then removed by xylol and the section is mounted in balsam.

On the other hand many bacteria do not readily take up the stain but retain it with greater tenacity. For such organisms *Gram's method* may be used. The staining solution consists of 1 part of a saturated alcoholic solution of gentian violet in 10 parts of a saturated watery solution of aniline oil (or preferably 10 parts of a five per cent. watery solution of carbolic acid), and should be prepared just before use. Stain for five or ten minutes; wash in water; apply for about one minute Gram's iodine solution (iodine 1 part, potassium iodide 2 parts, water 300 parts), and again wash. Rapidly remove water with absolute alcohol (or methylated spirit); apply clove-oil for 10 to 30 seconds; rapidly remove clove-oil with alcohol, and at once wash in water. The sections or films should then have only a faint violet tinge. The best contrast stain is saffranin. Bacteria stained by Gram's method appear of a dark violet colour, sometimes almost black, while most tissue-elements and bacteria that lose their stain in Gram's method are stained red by the saffranin.

Tubercle-bacilli are particularly resistant to staining agents and correspondingly tenacious when stained. *Ziehl-Neelsen's carbol-fuchsin stain* contains basic fuchsin 1 part, alcohol 10 parts, a five per cent. watery solution of carbolic acid 100 parts. The stain is applied to sections or films, heated gently till steam begins to rise, and allowed to steam for three to five minutes. Decolourise in 20 per cent. sulphuric acid and wash thoroughly in water. The tissue should then have a faint pink tinge. Counterstain for two minutes in a saturated watery solution of methylene blue. The tubercle-bacilli appear bright red, the tissues and other bacteria blue. Leprosy-bacilli may be stained in a similar way, but, as they are more easily decolourised, only a five per cent. solution of sulphuric acid should be used.

(2) *Staining of capsules*.—In the pneumococcus, pneumobacillus, &c., capsules may be stained by the use of a special mordant containing tannic acid

(Richard Muir). The mordant consists of 2 parts of a twenty per cent. watery solution of tannic acid, 2 parts of a saturated watery solution of mercuric chloride, and 5 parts of a saturated watery solution of potash-alum. Films, which should be as thin as possible, are first dried gently over a flame; the mordant is applied for two minutes, and the film washed in methylated spirit and water. Then apply Ziehl-Neelsen's carbol-fuchsin and heat very gently for two to four minutes. Wash in water, re-apply mordant for half a minute, again wash in water, and finally stain in a saturated watery solution of methylene blue for about five minutes. Differentiate in methylated spirit until the thinnest parts of the film appear blue, dehydrate in alcohol, clear in xylol, and mount in balsam. In this way the organisms are stained red, the capsules blue.

(3) *Staining of spores*.—By ordinary methods of staining spores remain uncoloured, but they may be stained by a method similar to that described for the bacilli of tuberculosis and leprosy. The stain is carbol-fuchsin heated as before, but the sulphuric acid solution should not exceed 2 per cent. in some cases, though it may be 5 per cent. in others. Before counterstaining in methylene blue, it is usually advisable to apply either a saturated watery solution of alum or the mordant recommended in staining capsules. Spores should then appear red, while the rest of the organism is blue.

(4) *Staining of flagella*.—The methods commonly employed are those devised by Löffler, Van Ermengem, Pitfield, and Richard Muir, but unfortunately all give somewhat uncertain results. Richard Muir's modification of Pitfield's method has the advantage of relative simplicity, and is at least not more uncertain than the others. Two solutions are required, (1) a mordant composed of ten parts of a 10 per cent. watery solution of tannic acid filtered, five parts of a saturated watery solution of mercuric chloride, five parts of a saturated watery solution of alum, and five parts of Ziehl-Neelsen's carbol-fuchsin (*vide supra*); (2) a stain composed of ten parts of a saturated watery solution of alum, and two parts of a saturated alcoholic solution of gentian violet. The mordant keeps well for several weeks, but the stain should be freshly prepared. Both solutions are used unfiltered. A thin film of the growth is carefully prepared and gently dried. The mordant is applied and the film heated for about two minutes, washed thoroughly in water and gently dried. The stain is then applied and the same processes repeated of heating, washing, and drying. Before the final drying Gram's iodine-solution (*vide supra*) may be applied for two minutes in order to darken the stained flagella.

RELATIONS OF MICRO-ORGANISMS TO THE LIVING BODY.—It may now be accepted as an established fact that the healthy tissues and fluids within the substance of the body are free from microbes; but the surfaces, both external and internal, constantly teem with them in the most varied forms.

The mechanism, so to speak, by which this bacterium-free condition is maintained during healthy life has been the subject of much experiment and controversy. Since foreign particles of various kinds, much larger than microbes, can and do enter the lymphatics and blood-vessels of the air-passages, alimentary canal, and even the skin,

it is certain that microbes can also enter. Experiment has shown that even if large quantities of certain cultivations are injected subcutaneously or into the blood-stream, they rapidly disappear, and in some cases after a few hours they can hardly be detected in the blood, or even in the lungs, spleen, and kidneys, where they appear to be largely arrested. Those discovered are often in various stages of degeneration or decay, and sometimes many are contained in leucocytes and connective-tissue corpuscles.

According to some, it is principally by the activity of leucocytes and connective-tissue corpuscles that micro-organisms are destroyed. This view has been especially developed by Metschnikoff and his followers, who have alleged for some leucocytes the special rôle of 'phagocytes,' i.e. of ingesting, amœba-like, noxious foreign particles, and having as one of their highest functions the elimination of microbes.

That the freedom from organisms is not solely due to the leucocytes is shown by the fact that blood-serum and other materials, which are free from leucocytes, also exert a destructive influence upon microbes. The freedom from bacteria may therefore be regarded as due in part to the fact that the healthy fluids and tissues of the living body exert an antagonistic action to, or do not serve as a suitable soil for, the growth of bacteria. The chemical or physical nature of the antagonistic condition is a highly important question. That the antagonistic condition may be a very minute chemical change is suggested by the facts of immunity, natural or acquired, and modified susceptibility under variations of nutriment. See IMMUNITY.

During the last stages of life, and apparently also in some conditions of disease, the power of resistance may be so lowered that common bacteria are found in the blood, and penetrate more deeply into the tissues with which they are normally in contact, e.g. in the alimentary system.

III. Inoculation of Animals.—Inoculation of animals may be undertaken for many different purposes. Certain organisms may be isolated from contaminated material in the blood or tissues of appropriate animals; but the special object of this mode of study is to investigate the pathogenic effects of pure cultures and of the toxic substances produced by bacteria. These effects are extremely varied and depend upon several factors: as regards the *virus*, upon (a) its quantity, (b) its form, and (c) its virulence, and as regards the *animal*, upon (a) its susceptibility, and (b) the site of inoculation. Some of the difficulties attendant on this mode of study have already been alluded to in an earlier part of this article.

1. The *virus*.—(a) The *dose* or amount of the injection materially modifies the result. A small number of bacteria or a small dose of toxin may produce little or no appreciable effect, for within certain limits the tissues and fluids of the living body possess remarkable powers of overcoming and destroying microbes. In fact, bacteria may with impunity be introduced beneath the skin, into the peritoneal sac, or even into the blood-stream, in quantities that depend upon their virulence and the potential resistance of the animal. Introduced in greater numbers, bacteria begin to multiply within the animal body and so infect it. Moreover, the form of the resultant lesion may vary

with the number of organisms injected. A small dose of pyogenic cocci in the subcutaneous tissue may give rise only to a localised inflammation or suppuration, whereas a larger dose at the same site may allow some of the cocci to enter the blood-stream and so cause a fatal septicæmia or pyæmia.

(b) The virus may be introduced in a variety of *forms*, as living bacteria, as dead bacteria, or as toxic products alone. *Living bacteria* produce their effects by multiplying within the body, and there elaborating toxic substances which may have both a local and a general action. *Dead bacteria* of course cannot multiply, but may be carried to various sites and there give rise to localised lesions due to the action of intracellular ferments or toxins, e.g. dead tubercle-bacilli may give rise to nodules of granulation-tissue, which often show typical giant-cell systems. The *toxic products* of bacterial growth may be isolated, and when injected reproduce many of the pathogenic effects. In some diseases (e.g. diphtheria, tetanus, &c.) the most important lesions are due to the action of separable toxins, and are induced when toxins alone are injected. When living organisms are introduced, the amount of toxic substances generated by them within the body is indefinite and uncertain, but by the use of toxins freed from bacteria, more exact data are obtainable, since a definite dose of toxin will produce definite effects per unit of body-weight of animal.

(c) The *virulence* of the material injected has an even greater bearing on the ultimate result. Thus the *Streptococcus pyogenes* may have its virulence so intensified that a few introduced beneath the skin will cause a rapidly fatal septicæmia, while the same organism in an attenuated form may be injected in large numbers with only a slight local reaction. Modifications in virulence may be artificially accomplished. The most important means of exalting the virulence of an organism is that known as the 'method of passage.' It depends on the fact that an organism which has produced disease in one animal is thereby rendered more capable of reproducing the disease in another similar animal. Hence, by inoculating a number of such animals, one from another—in other words, by passing the organism through the series—its virulence may be intensified to a high degree. In certain cases, and more especially when its pathogenic powers are impaired, the virulence of an organism may be restored or exalted by injecting it along with cultures or products of other organisms. Thus, the typhoid-bacillus, which is usually non-pathogenic to the lower animals, may have its virulence so intensified by repeated injections along with sterilised cultures of *Streptococcus pyogenes* or of *B. coli*, that ultimately the typhoid-bacilli alone come to have a lethal effect. In processes of natural infection in man it is not uncommon for the action of one organism to be aided by that of another. Probably the virulence of one or of both is thereby increased, and the site of the local lesion rendered more favourable for the growth of the predominant microbe. As examples may be noted the frequent association of the *Streptococcus pyogenes* with the bacillus of diphtheria, and the presence of putrefactive organisms in many of the local wounds in tetanus. On the other hand virulence may be weakened or attenuated in many ways. After successive subcultures outside the body most organisms are found to have lost their original patho-

BARBADOES LEG—A form of Elephantiasis. *See* ELEPHANTIASIS.

BARBIERS.—A synonym for Beriberi. *See* BERIBERI.

BARÈGES, in France.—Thermal sulphur waters. *See* MINERAL WATERS.

BARTFELD, in Hungary.—Alkaline saline chalybeate springs, with iodine. *See* MINERAL WATERS.

BASEDOW'S DISEASE.—A synonym for exophthalmic goitre. *See* EXOPHTHALMIC GOITRE.

BATH, in Somersetshire.—Simple thermal and earthy waters. *See* MINERAL WATERS.

BATHS.—Baths may be regarded as *simple* and *composite* (*medicated*, *artificial*), and as liquid, vapour-, and air-baths. We shall consider them under these heads in the following description.

A. SIMPLE BATHS.—**I. Simple Liquid Baths.** The clothes by which the body is usually covered form a kind of thermal zone about the skin, the temperature of which remains ordinarily within a few degrees of 89° F. The skin therefore is thoroughly accustomed to this temperature, and an ordinary bath must be given at a decidedly higher or lower temperature, if much reaction be desired. Even however at neutral temperatures—say between 85° and 95° F.—a reaction may be obtained by mechanical stimulation of the skin, as in wave-baths and douches, or by drying the skin with a rough towel after the bath; in the case of mineral waters and medicated baths stimulating effects may likewise be obtained by the salts, carbonic acid gas, &c., contained in the water.

1. The Cold Bath.—By a cold bath is meant the immersion of the body in water below the temperature of 70° F. Anything below 50° is considered a very cold bath. The first effect of the bath is a sensation of cold, with slight gasping for breath. If the cold is intense and prolonged, there is a certain degree of numbness of the skin; while the pulse becomes small, and may become less frequent by ten to twenty beats in the minute, and there may be shivering. Owing to the constriction of the cutaneous blood-vessels the skin is pale, and the contraction of the unstripped muscle-fibres gives rise to the phenomenon of 'goose-skin.' These first effects of a cold bath may give place to the 'reaction' while the bather still remains in the bath, or only when he has left it. The pallor of the skin changes to slight redness, accompanied by an agreeable subjective sensation of warmth, easy breathing, and a feeling of comfort and capability for exertion.

The reaction is delayed in the weak and feeble, but takes place rapidly in the robust and strong, especially when accustomed to cold baths. In strong persons, who are in the habit of taking cold baths, the preliminary vaso-constriction and sensation of coldness are less marked than in others, whereas the reaction, together with the agreeable subjective phenomena which accompany it, occurs more readily. In irritable nervous subjects signs of reaction may sometimes be rapidly and readily obtained, though the reaction may be imperfect and too transient. In a given case the rapidity and kind of reaction depend on the temperature of the water, the length of the application, and the

accompanying amount of mechanical stimulation; it is greatly assisted by voluntary muscular action (such as swimming), and by friction to the skin before, during, or after the application. The reaction may be hindered, if the cold bath is of too long duration, or, if it has already occurred, it may pass off and give place to depression. The best reaction with the least loss of heat is generally obtained by cold applications of short duration, administered when the patient is hot.

The vascular and respiratory phenomena produced by a cold bath are generally considered to be mainly, if not entirely, due to reflex action. In persons who feel the loss of heat excessively, especially if preliminary warming by exercise be difficult, a cold bath or douche (which should of course be of very short duration) may be immediately preceded by a short warming application, such as a hot bath, a hot douche, or a hot vapour-bath. A preliminary hot bath with soaping renders the skin more sensitive, and facilitates the reaction. In suitable cases, when the organism can stand the abstraction of heat, and when the circulatory, respiratory, and digestive systems react sufficiently, treatment by cold baths should strengthen the heart's action, exercise the respiratory organs, increase the appetite, and aid digestion; while metabolism should be rendered more complete, and the elimination of waste products facilitated. The quality of the blood and the nutrition of the whole body should be improved, and the capacity for muscular and mental work should be increased.

2. The Warm Bath.—A warm bath of 96° to 104° F. produces no shock; it causes a moderately increased flow of the circulating fluids to the surface, augmenting the frequency of the pulse; and scarcely affects the respiration. There is not the depression or the excitement of a cold bath. It rather retards tissue-change. With a hot or very hot bath—from 104° to 114°, the central nervous and circulatory systems are more affected. The frequency of the pulse increases greatly. The respiration becomes anxious and quickened. The skin is in a hyperemic condition, and in many persons a free perspiration breaks out.

3. The Tepid Bath.—Tepid baths of the temperature of 85° to 95° F. are intermediate between cold and warm. Their effects seem to be confined to the peripheral extremities of the nerves, and they do not excite the nervous centres or the circulatory system. Neither the pulse nor the excretions and secretions are affected. As no heat is confined in the system or taken from it, there is no reaction, and the body-temperature is unaltered. A macerating action on the superficial epidermis can be obtained by prolonged tepid baths, similar to that effected by the prolonged tepid mineral-water baths of *Loèche-les-Bains* in Switzerland.

It need scarcely be added that drying and rubbing after a bath materially assist its action on the skin; or that, according to circumstances, it may be convenient to order a whole bath, a hip-bath, or a slipper-bath. The foot-bath is a very useful and convenient one, especially when some stimulant substance is added to the simple water. Wet packing and hot and cold affusion, whether as shower-baths or as douches, are referred to in the articles on DOUCHE, and HYDROTHERAPEUTICS.

The duration of a bath must depend on a variety of circumstances—for instance, on the age and constitution of the patient, on the nature of his malady,

and on the temperature of the bath. It may vary from a few minutes to many hours. A very hot or a very cold bath can be supported for a much shorter time than a tepid one.

ACTION AND USES.—Cold baths are indicated for the strong, for youth, and for manhood; warm baths for the delicate, for women, for early childhood, and for old age. Tepid baths are suitable for almost all constitutions, sexes, and ages. Cold baths may, in a general way, be considered tonic and bracing; they are useful when judiciously employed in many nervous affections, as in chorea and hysteria, and they are the best of all for general hygienic purposes. Of late years they have been specially employed in the treatment of pyrexia, and of the infectious diseases (especially typhoid fever) of which fever is a symptom (*see TEMPERATURE; FEVER; and HYDROTHERAPEUTICS*). The great value of warm baths, besides their hygienic value, as better detergents than cold ones, is in soothing and reducing excitement; in relieving spasms, such as colic and retention of urine; in the convulsions of children, combined with the affusion of cold water on the head; in cases of gout and rheumatism, and generally when action on the skin is desired. Where prolonged immersion is wanted, tepid baths are indicated, as in calming many chronic nervous disturbances, and in many cutaneous affections.

As to contra-indications, all baths, and especially prolonged baths, even when tepid, are not suited for the asthenic. Both hot and cold baths are to be avoided where there is a weak, fatty heart, or any tendency to apoplexy. No one should ever enter a cold bath when exhausted, and such baths are also contra-indicated when there is a tendency to congestion of internal organs. Under such circumstances a warm bath is usually both safer and more refreshing. The too long and too frequent use of hot baths is debilitating.

II. The Simple Vapour-Bath.—A vapour-bath is one in which the skin is exposed to the action of hot water presented in the form of vapour. The vapour-bath may be taken in a box with the head included or not; or in the more common form of the Turkish or Russian baths, where a large room is filled with vapour, and where therefore the vapour is inhaled; or by vapour obtained from a small and suitably constructed apparatus, which vapour may be diffused over the whole body or directed to a particular part. A very simple apparatus for the vapour-bath may be prepared by placing under a chair a shallow earthenware or metallic pan, containing boiling water to the depth of three or four inches, and from which abundant vapour can be obtained by placing in it one or two red-hot bricks. The patient sitting on the chair, surrounded by blankets and other suitable covering, will receive the full benefit of a vapour-bath. Vapour-baths produce profuse perspiration, and act in cleansing the skin much as hot-water baths do, only more powerfully. Vapour being a slow conductor does not act so fast on the body as water. Vapour-baths can be borne hotter than warm-water baths, but their use cannot be continued so long, as vapour interferes with radiation of heat from the body. In such baths a heat of more than 122° F. is not borne comfortably. The vapour-bath, though falling considerably short in temperature of the air-bath, raises the heat of the blood somewhat more. The great virtue of these baths lies in their sweat-producing properties. The

average loss of perspiration by the use of a Russian bath has been set down at from $\frac{2}{3}$ lb. to 3 lbs. In the Russian bath a slight degree of stimulation of the skin is caused by switching it with twigs of birch, and the alternation of depression and excitement of the cold bath is obtained by placing the patient, when in a state of profuse perspiration, under a douche of cold water. In Russia a roll in the snow sometimes takes the place of the final cold douche or plunge.

III. The Simple Hot-Air Bath.—There are two forms in which the hot-air bath is administered: according as the patient does or does not breathe the heated air. The action of the former closely resembles that of a vapour-bath, but differs from it in not impeding the respiration, as the latter does, by accumulation of moisture in the bronchial tubes. The lungs, instead of having to heat up the inspired air, are subjected to a temperature above their own. Hot-air baths favour the highest degree of perspiration; the moisture of vapour-baths somewhat opposes it, though in certain persons sweating commences more readily with moist than with dry heat. Very hot-air baths raise the temperature of the body by several degrees.

As the arrangements for vapour- and hot-air baths are practically the same (except that in the latter it is attempted to exclude all vapour from the *calidarium* or *sudatorium*, the hottest room), the following description of an ordinary hot-air bath, the arrangements of which are closely copied from the Romans, will answer for both.

The patient, after unclothing, first goes into the *tepidarium*, which has a temperature of 113° to 117° F., in which he remains until the perspiration bursts forth, which happens in from twenty-five to forty minutes. He next proceeds to the hottest room or *calidarium* (in which the air is heated by hot-air pipes which are inserted in the walls, to a temperature of 133° to 140° F.), and remains there until the perspiration runs down his skin, in twelve to eighteen minutes. An attendant then rubs off the perspiration with a woollen glove, and kneads all the muscles for four or five minutes. The patient next betakes himself to the *lavacrum*, where he has water poured over him of the temperature of 81° to 86° F.; then the whole body is soaped over, the suds are removed, and the patient goes to the *frigidarium*, where he lays himself on a couch and waits till his skin is completely dry. This may occupy twenty-five to thirty minutes, when the patient dresses and leaves the bath greatly refreshed.

Such is a brief account of these baths, the revived use of which is at present so general. The arrangements vary in detail. In all large baths of this kind, used by many persons, special care has to be taken that the arrangements for ventilation are sufficient. For ordinary purposes it is easy to furnish either vapour or hot-air baths. Different kinds of apparatus have been invented for this purpose, but they all have this in common, that the patient, placed in bed or on a seat, has the bedclothes or other covering secured from contact with him by the employment of a framework or cradle. Beneath this hot air or vapour is introduced, either directly or indirectly, from a suitable apparatus.

USES.—Both hot-air and vapour-baths are indicated when increased action of the skin is desired. They are used most for the cure of catarrhs, of neuralgic and rheumatic pains, and sciatica. They

are sometimes employed in the treatment of obesity, and are useful for general hygienic purposes, but are apt to be given too indiscriminately. Hot-air and vapour-baths are often locally applied with great advantage to a hand, or leg, or arm, in rheumatism or thickened joints.

Electric light has of late been much employed for administering both local and general hot-air baths. It has been maintained that owing to the action of the light as well as the heat, these electric light or 'radiant heat' baths have some peculiar effect of their own, and that they can produce sweating without accelerating the heart's action to the same extent as ordinary hot-air and vapour-baths. These questions have, however, not yet been settled.

The Sand-Bath.—We may here mention baths of sand. At the present time establishments for supplying them exist in various towns. They are a convenient way of applying dry heat either locally or generally, and are principally employed in chronic rheumatoid arthritis. Bags filled with heated sand (or salt) are useful in hospital and in domestic practice.

Carbonic-Acid Gas-Baths.—These should be likewise mentioned, though they are not much employed. The carbonic acid gas arising from mineral waters (as at Franzensbad and Marienbad) has usually been employed for the purpose. The patient sits thinly clad in an atmosphere of the gas, care being taken that the gas does not rise higher than the shoulders and that none of it is inhaled. A subjective sensation of warmth in the lower limbs, and the part of the body bathed by the gas, is produced; but the therapeutic value of this method of treatment is doubtful. Local baths and douches of carbonic acid have been employed for neuralgic and superficial inflammatory conditions.

B. COMPOSITE, MEDICATED, OR ARTIFICIAL BATHS.—A great variety of substances have been used in baths at different periods. We must confine ourselves to such as are at present in use and appear to be of real value, omitting even some that are employed, such as baths of iodine, of iodide of potassium, of iron, of fermented grapes, and of whey.

I. Composite Liquid Baths.

1. *The Sea-Water Bath.*—The average amount of salts in sea-water may be set down at 3 per cent.; this may therefore be considered a suitable strength for ordinary salt baths. The quantity commonly used in London hospitals is about 9 lbs. of salt to 30 gallons of water. Owing to the high price of sea-salt in inland continental places, various natural salts, some of them containing a comparatively small amount of chloride of sodium, have been suggested as substitutes; and also, for economy's sake, 22 to 25 gallons have been set down as a minimum amount of water for the bath of an adult. The value of these substitutes can only be determined by observing the degree in which they stimulate the skin. Apparently it does not matter much what particular salt is employed to produce the stimulation. A salt bath can of course be increased to any strength by the addition of what is termed 'mother-lye' (German 'Mutterlauge').

The chief uses of salt-water baths are as tonic remedies, especially for the young, when there is any tendency to scrofula or debility in convalescence from many diseases.

2. *Effervescent, Gaseous, and Artificial Nauheim*

Baths.—These baths have been considerably employed of late years, since the natural tepid effervescent salt baths of Nauheim have been much advocated in the treatment of certain chronic cardiac disorders. The bath-water may be charged with carbonic acid from cylinders of the compressed gas. The stimulating effect of gaseous baths is due chiefly to the mechanical action of the small bubbles of the gas on the skin. Two or three per cent. of common salt and two or three per thousand of calcium chloride should be dissolved in the bath-water, when it is desired to imitate the natural baths of Nauheim.

3. *Alkaline Baths.*—Alkaline baths may be made by adding 6 ounces of crystallised carbonate of sodium, or 3 ounces of carbonate of potassium, to 25 or 30 gallons of water. Alkaline baths are of use in a great variety of cutaneous affections.

4. *The Corrosive-Sublimite Bath.*—This is commonly made by adding 3 drachms of corrosive sublimate and 1 drachm of hydrochloric acid to 30 gallons of water. It has been occasionally employed in some skin-affections, and in secondary syphilis.

5. *Sulphuret of Potassium Bath.*—Baths of sulphuret of potassium are made by dissolving from 4 to 8 ounces of that salt in 25 to 30 gallons of water. A little diluted sulphuric acid is sometimes added. These baths have long been extensively employed in the treatment of cases of skin-disease in which the sulphur they contain is indicated.

6. *The Nitro-Muriatic Acid-Bath.* The nitric or rather nitro-muriatic acid bath—used by some in India and in England for liver-affections—is made by adding nitro-muriatic acid to water. The ordinary proportion is one ounce of acid to one gallon of water. The discolouring action on clothing makes a full bath of this kind inconvenient for domestic use, and it is best to take it in a bathing establishment. For the ordinary purposes of a foot-bath at home, the following directions are sufficient. The vessel must of course be of wood or earthenware. Four to six ounces of the acid are added to three gallons of water. This makes a strong foot-bath. The patient keeps his feet immersed for thirty minutes; and the bath is to be repeated every other day for two or three weeks. The axillæ, the groin, and the region of the liver are to be sponged with the acid solution. The bath causes slight tingling of the skin and a taste in the mouth, and is believed occasionally to produce salivation.

7. *The Bran-Bath.*—The bran-bath is made by boiling four pounds of bran in one gallon of water, straining, and adding the liquor to a quantity of water sufficient for a bath. Such a bath is useful in allaying irritability of the skin, and also in diminishing the stimulating effect of other baths.

8. *The Fucus-Bath.*—This is made by adding a decoction of seaweed, or the seaweed chopped up, to an ordinary bath; it will become more or less gelatinous if enough be added. Such baths go popularly by the name of *Ozone Baths*. They are useful in the same cases as sea-baths.

9. *The Mustard-Bath.*—An extremely useful stimulating bath is the well-known mustard bath, which is made by adding a handful or two of mustard to the ordinary hot bath. The foot-bath is its most useful form.

10. *Pine-Baths.*—Baths of the balsam of pine-leaves may be prepared extempore by making decoctions of the fresh leaflets at certain seasons; but the usual way is to add about one pound of the extract, which is prepared from the leaves, and is

everywhere for sale—at least in Germany. The extract dissolves in the bath, which is then ready for use; but of late it has been usual to add a small amount of an essence which is also prepared from the leaflets. It floats to the surface of the water, and attaches itself to the person on leaving the bath, and its aroma is grateful. These favourite baths are largely employed at bathing establishments in every part of Europe. They are slightly stimulant, and are much used in hysterical, rheumatic, and gouty affections, and also as an adjunct to the internal use of mineral waters.

It is scarcely necessary to add that, as a rule, all composite liquid baths should be of a temperature a little above the tepid; and that their strength, and the time that the patient is to remain in them, must be determined by the special circumstances of the case.

II. Composite Vapour-Baths.—Vapour-baths impregnated with *fir-balsam* are popular, and are considered to be more powerful in their operation than pine baths. The vapour which rises in making the decoction of pine leaves is conveyed to a box in which the patient is enclosed.

Aromatic vapour-baths may be given by making the steam of hot water pass through bunches of fresh aromatics (*conium*, *lavender*, &c.) before reaching the box in which the patient is placed. Such baths may be useful in hysteria.

III. Composite Air-Baths.

1. *Sulphurous Acid Bath.*—A valuable mode of applying sulphur in the form of a bath is by using its fumes—in other words, sulphurous anhydride. The patient is seated on a cane-bottomed chair, and his body is encircled with a cradle, over which oil-cloth is thrown, the head remaining uncovered. Sulphur is placed on a metallic plate, to the lower surface of which the flame of a lamp is applied, when sulphurous acid is disengaged.

2. *The Mercury- and Calomel-Baths.*—Very similar is the mode of applying the fumes of mercury. Under the chair are placed a copper bath containing water, and a metallic plate on which are put from 60 to 180 grains of the bisulphuret or of the yellow or red oxide of mercury. Spirit-lamps are lighted under the bath and under the plate. The patient thus experiences the effects both of aqueous and of mercurial vapour. At the end of five or ten minutes perspiration commences, which becomes excessive in ten minutes or a quarter of an hour. The lamps are then to be extinguished, and when the patient becomes moderately cool he is to be rubbed dry. He should then drink some warm liquid and remain quiet for a time (see *SYPHILIS*). Calomel, in quantities of from 20 to 30 grains, is administered in a similar manner, under the name of the *calomel-bath*. It may be given locally by a suitable apparatus.

IV. Electrical Baths.—Electricity is sometimes applied in association with baths or douches of water. See *ELECTRICITY IN MEDICINE*.

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BATHS, Natural.—See *MINERAL WATERS*.

BATTAGLIA, in the province of Venice in Italy.—Thermal muriated saline waters, with sulphate of lime. See *MINERAL WATERS*.

BAZIN'S DISEASE.—See *ERYTHEMA INDURATUM*.

BED-SORE.—See *ULCER* and *ULCERATION*.

BELL-SOUND.—A peculiar physical sign associated with pneumothorax. See *PHYSICAL EXAMINATION*.

BELL'S PARALYSIS (named after Sir Charles Bell).—A synonym for paralysis of the facial nerve. See *FACIAL PARALYSIS*.

BERIBERI.—SYNON.: *Kakke* (Japanese).

DEFINITION.—A disease characterised by multiple peripheral neuritis, and presenting symptoms of paralysis, oedema, or both, varying with the kind and position of the nerves affected; tending generally to recovery, but frequently ending in death from implication of the nerves controlling or regulating the chief organs of circulation and respiration.

ETYMOLOGY.—Beriberi is a Cingalese word, and means simply 'a bad sickness,' or 'a sickness from which the patient is in danger of death,' the duplication of the syllables intensifying the meaning.

GEOGRAPHICAL DISTRIBUTION.—Beriberi is endemic in Japan, the Malay Peninsula, Sumatra, and many other places in the Eastern Archipelago; it is met with in epidemic form in China, Manila, many parts of India, Africa, and probably other tropical and subtropical countries; it breaks out as an epidemic occasionally on board ships; and of late years outbreaks have been reported from the West Indies, Fiji, Western Australia, the Richmond Asylum in Dublin, and other places where the disease was previously unknown, from which it would appear that a tropical climate is not absolutely necessary for the propagation of the poison.

ETIOLOGY.—The immediate cause of beriberi is not yet determined. It has been generally described as a disease of low-lying, damp localities, with associated conditions of overcrowding and defective sanitation, and there can be no doubt that its prevalence in epidemic form is favoured by these conditions, and that most or all of them are found in places in which the disease is endemic. Still the fact remains that no one of these conditions and no combination of any of them is necessary, and that beriberi will occur sporadically and as an epidemic in places in which not one of these conditions is present. This has been verified over and over again in the Straits Settlements, where beriberi has broken out and prevailed for a time in institutions which are neither damp nor low-lying, in which all matters relating to food have been carefully attended to, and in which no sanitary defect can be found. As regards the immediate cause of the disease, deficiency in nitrogenous food, some disease affecting rice, malaria, deficiency in fatty constituents of food, specific bacterial organisms, have all been from time to time put forward, and the writer, in over twenty years' experience of the disease, has been a convert to several of them in turn, but none of them has been found equal to accounting for all outbreaks of the disease. There can be no doubt that beriberi is caused by a specific poison, and the latest ideas seem to indicate that this poison is of the nature of a toxin, and that it is formed outside the body. Manson inclines to the opinion that the toxin or poison is manufactured in the soil, or in the houses and surroundings of beriberi spots; and it is certain that places, especially places which fulfil the conditions above mentioned as favourable to the spread of the disease, can become infected.

Pekelharing and Winkler first called attention to the presence of diplococci in the blood, to which they attributed the disease, but their culture-experiments must be looked upon as far from conclusive. Many other observers have experimented with the same bacteria, but cultivation and inoculation, though followed sometimes by a certain amount of success, in that symptoms of neuritis (as in several of the experiments of Hunter with rabbits) followed the inoculation, have not by any means proved the causation of the disease. Two points may be mentioned in this connection: the first that Ellis, in working at beriberi at Singapore, found an apparently identical diplococcus frequently present in the blood of persons to all intents healthy; and the second that von Tuzelmann produced, by cultivation on agar-agar from a kind of fungous growth found on the woodwork of a hospital-ward in which cases of beriberi had occurred, growths of organisms also apparently identical with the same diplococcus. These points may perhaps tend to indicate that it may yet be found that the bacterium in question may exist innocuous in the blood, though capable of manufacturing the poison in suitable environment outside the body.

PATHOLOGY AND MORBID ANATOMY.—The pathology of beriberi is that of a multiple peripheral neuritis, the paralytic symptoms and aberrations of sensation being caused by affection of the motor and sensory nerves, the oedema and effusions of fluid by affection of nerves of vaso-motor function. If the disease does not last long (and the nerve-degeneration seems capable of rapid repair); the only pathological condition is the neuritis, but if the paralysis lasts any considerable time, as occasionally happens, the muscles affected become shrunken and atrophied. The præcordial oppression and vomiting that usher in alarming symptoms, the oedema of the lungs, the paralysis of the diaphragm, and the paralysis and dilatation of the heart, are all caused by neuritis of supplying nerves. The cause of death is not always the same; paralysis of the diaphragm has been put forward as a frequent cause of death, but a patient will occasionally lie for days with purely thoracic breathing, and ultimately recover; pericardial effusion no doubt sometimes causes death, but only rarely in the experience of the writer. Death most frequently results from paralysis and dilatation of the heart, helped often by pulmonary oedema; it may also occur from pulmonary oedema alone, the heart-muscle remaining unaffected. The post-mortem appearances vary with the cause of death. Oedema and effusion into serous cavities may be present in varying degree; three ounces or more of fluid is nearly always found in the pericardium. In death from uncomplicated pulmonary oedema the lungs are found gorged with blood and frothy fluid, which pours out when the organ is incised; in such a case the heart may be found with ventricles in systole, but containing fluid blood, the large veins and auricles being distended; ecchymotic spots are sometimes found in these cases under the visceral pericardium. In death from paralysis of the heart that organ is found dilated, often enormously, with ventricles in diastole, and all cavities and great vessels full of blood and clot; the lungs are congested and may be somewhat oedematous; there is generally pericardial effusion to the amount of three or four ounces. Neuritic degeneration can be demonstrated in the affected nerves,

most readily in the peroneal and tibial, but it has also been found among others in the vagi, phrenic, and renal nerves, and those of the cardiac plexus. If the muscles of the legs or arms are much atrophied, the fibres will be found to have lost their striation, and the teased specimen will look like a collection of fine fibrils with homogeneous spaces between them.

SYMPTOMS AND DIAGNOSIS.—The disease may present symptoms of oedema, or muscular paralysis, or both. These different groups of symptoms are best described separately, it being borne in mind that varying degrees of both may, and usually do, occur simultaneously, though one kind generally predominates, and that there is no true division into *wet* and *dry* varieties of beriberi. The oedema commences with slight swelling over the lower part of the shin, increasing and spreading upwards over legs, thighs, trunk, upper extremities, and face, so that, when the disease is, so to speak, fully developed, the patient lies in a bloated, helpless condition. There may also be ascites, hydrothorax, or hydropericardium. The urine in these cases is scanty, high-coloured, and of high specific gravity; it is never albuminous. These symptoms are generally accompanied by more or less difficulty in standing or walking, loss of patellar reflex, and tenderness on deep pressure over the gastrocnemii and adductors of thigh. When the *paralysis* predominates, there is first some difficulty in walking, and afterwards absolute inability to walk or stand; if the patient be supported, and told to try and walk, the leg and foot are lifted by the thigh-muscles, and hang loosely from the knee, the foot being dropped from the ankle; there is loss of patellar reflex from the outset, and tenderness on squeezing the gastrocnemii and inner thigh-muscles; there is often a feeling of numbness and anaesthesia of the skin over the front and outer part of the leg and dorsum of the foot. Hyperaesthesia of the skin is occasionally met with. If the arms are affected there is wrist-drop, and often anaesthesia and numbness of the skin over the back of the forearms; sometimes anaesthesia is found around the mouth, and there may be paralysis of the lip-muscles, or occasionally of the muscles of the larynx, leading to loss of voice. There is never any paralysis of bladder or rectum, and the cremasteric and abdominal reflexes are, so far as the writer knows, never lost. These symptoms, as above mentioned, may be accompanied by oedema in varying degree. Functional cardiac murmurs are said to be frequently heard, and Manson calls attention to an alteration in the heart-rhythm. If the disease last long, the paralysed muscles become wasted, and recovery is slow; but often under appropriate treatment both kinds of symptoms subside rapidly. At any time in the course of the disease alarming symptoms may set in, and a patient who does not seem seriously ill may die in a few hours. Dangerous symptoms are generally ushered in by a feeling of præcordial oppression, and the vomiting of frothy fluid; then restlessness and dyspnoea set in, with rapid action of the heart, distended jugular veins, and terrible distress generally. In death from asphyxia due to pulmonary oedema, the distress is worse even than in death from cardiac paralysis. The dyspnoea is worse, the jugular veins are more distended, the pulsation in the neck is more marked, the face more livid, and the pulse hard; in death from cardiac paralysis the restlessness is

the same, but there is breathlessness rather than dyspnoea, the pulse is rapid but soft, the temperature falls, and the heart, becoming more and more dilated, as evidenced by percussion, gradually fails. Death from pulmonary oedema alone is infrequent, cardiac paralysis and dilatation being usually present to a greater or less degree, and adding to the danger. Cardiac murmurs are frequently heard, generally systolic; and præcordial pulsation is marked. The presence of pulmonary oedema is shown by impaired resonance, and crepitation, especially over back of chest. The diagnosis of beriberi, when it occurs in epidemic form, is not difficult, but it must be remembered that the symptoms are indistinguishable from those of peripheral neuritis produced by some metallic poisons, and poisoning by some of these may occur in epidemic fashion; epidemics of beriberi are, however, generally met with in tropical or sub-tropical countries. Sporadic cases are probably never met with except in the last-named localities, and here the history will help in distinguishing them from cases of neuritis caused by alcohol. *See* NEURITIS, MULTIPLE.

PROGNOSIS.—The prognosis in beriberi, in consequence of the liability to dangerous symptoms, must always be grave. Unfavourable signs are præcordial pain and pulsation, vomiting, restlessness, evidence of cardiac dilatation (as shown by percussion, and, as pointed out by Manson, by alteration of the cardiac rhythm), increasing rapidity of pulse, and a fall of temperature.

TREATMENT.—The medicinal treatment of the disease consists in the treatment of symptoms. The oedema is best dealt with by free purgation with elaterin and the administration of diuretics, of which scoparium is probably the best. Pineapple-juice has been found of service in increasing the flow of urine, and this fruit may with advantage be given as an adjunct to treatment by drugs. For the paralytic symptoms arsenic and strychnine are probably the best remedies; later, if the muscles are wasted, galvanism may be used. As regards the dangerous symptoms, if the pulse is hard and the distress great, with physical signs indicative of lung-oedema, great relief, and in uncomplicated cases often cure, is given by nitroglycerin in doses of two to four drops of the one per cent. solution dropped upon the patient's tongue, every hour at first, and afterwards at longer intervals as relief is given. The drug may be assisted by blisters to the back of the chest. A case is hopeless in proportion to the degree of paralysis and dilatation of the heart present; in these cases nitroglycerin gives too often only temporary relief. Probably the best remedies when the heart is dilated, the pulse rapid but soft, and the temperature falling, are digitalis, or injections of strychnine, with the use of artificial warmth externally. Pericardial effusion, if endangering life, should be dealt with by aspiration. The most important measure in treatment is the removal of patients from the place where they have contracted the disease, and this removal alone is often conducive to speedy recovery, even sometimes when the advent of alarming symptoms may seem imminent. The place to which removal is made should of course be one of which the position and hygienic conditions are the reverse of those mentioned as favourable to the spread of the disease; a sea-voyage often acts beneficially. As regards diet, it should be good; a great deal has

been said as to the necessity for a rather large proportion of nitrogenous food, and in the case of natives this is best effected by the substitution of beans or wheaten flour for a portion of the rice which is usually the chief article of diet. In the case of epidemics on ships, in schools, gaols, and similar institutions, patients should be removed if possible; but in any case cleaning and disinfection should be practised as though a contagious disease were being dealt with.

MAXIMILIAN F. SIMON.

BETHESDA SPRINGS, in Waukesha County, Wisconsin, U.S.A.—Calcareous waters. *See* MINERAL WATERS.

BEX, in Switzerland.—Strong muriated saline waters, with some iodide and bromide of magnesium. *See* MINERAL WATERS.

BIARRITZ, in France, on the Bay of Biscay.—A fashionable seaside resort. The climate is bracing. *See* CLIMATE, Treatment of Disease by.

BILE, Disorders of.—The bile is an excretory fluid through which are removed not only products of blood-destruction such as the bile-pigments, but also other effete products filtered off by the liver from the blood arriving from the digestive tract—products the presence of which in the general system would be attended with ill effects. Furthermore, in relation to the process of digestion it materially aids the assimilation of fats from the intestine and has valuable laxative properties; but an even more important function is the one recently discovered (1897) by T. R. FRASER, namely that it has a remarkable power of antagonising and rendering inert powerful organic poisons, such as that of snake-bite. There are many poisonous products generated in the intestine, capable, if absorbed, of producing ill effects. The symptoms so commonly ascribed to 'sluggish liver' or 'want of bile' may be due, in part at least, to the absence of a sufficient amount of healthy bile and to the consequent absorption of products that would otherwise have been rendered inert by it. The undoubted value of cholagogue substances—'stimulants of bile-secretion'—so long employed on empirical grounds alone, may thus obtain a satisfactory explanation.

The amount of bile secreted daily varies greatly under the influence of many different factors. The chief of these factors in health is the taking of food. The flow of bile increases when food is taken, and falls when food is withheld. Throughout the 24 hours its flow is irregular: being highest in the middle of the day (12 to 4), and lowest in the early hours of the morning. The flow of bile is greatly influenced by the amount of fluid taken; it is the flushing of the biliary system with water which serves to explain the remarkably beneficial action of so many Mineral Waters. The influence of drugs in promoting the flow of bile is probably overrated. No drug is so powerful in this respect as the bile or the bile-salts. Among the various drugs accredited as cholagogues are:—Alkalies, chloride of sodium, sulphate of sodium, sulphate of potassium, phosphate of sodium, mercuric chloride, ipecacuanha, colchicum, jalap, aloes, colocynth, rhubarb, benzoate of sodium, and salicylate of sodium. The only one whose power in promoting an increased flow of bile has been fully ascertained is salicylate of sodium.

No factor is more important in producing so-called 'bilious' disorders than *diminished secretion of bile*. The trouble it occasions arises not so much from any diminished output of the bile-pigments and bile-salts as from (1) the retarded flow of bile, giving opportunity for the absorption of its constituents and for disturbance of the liver itself; and (2) the lack of bile in the intestine, permitting the absorption of injurious products which might otherwise have been rendered innocuous. This diminished secretion may be the outcome of defective action on the part of the liver itself and a consequently defective *vis a tergo*; such is probably its character in all febrile conditions. But another and, in the opinion of the writer, more potent factor is any increase of resistance to its flow (at all times under very low pressure) along the bile-passages. This increase of resistance may arise from two causes: from increased secretion of mucus from the bile-passages and corresponding increased viscosity of bile, or from sluggish muscular action of the walls of the bile-ducts. The latter is of doubtful importance; the former is the condition that is generally present in cases of 'biliousness.'

This increased viscosity is largely due to the action on the lining mucous membrane of a morbid or altered bile. One of the chief functions of the liver is not merely to excrete, but to destroy various products carried to it from the digestive tract. These products are formed in the stomach and intestine and carried to the liver in the portal blood. The liver partly destroys and partly excretes them. If the character of those excreted is such as to irritate the bile-passages along which they pass, the result is an increase of mucus, an increased viscosity of bile, and, as a secondary consequence, a retardation of its flow and a diminution in its quantity.

A large number of substances introduced into the blood may also in this way be *excreted* by the liver, e.g. iodide of potassium, cane- and grape-sugar, sulphate of copper, oil of turpentine, iron, lead, arsenic, silver, chlorate of potassium, salicylate of sodium, toluylenediamin—in some cases very quickly. Thus in the case of the last-named drug the writer detected it in the bile within half an hour, and in 3 to 4 hours it was present in quite appreciable quantity.

On the other hand other drugs are not excreted but *destroyed* by the liver, e.g. atropine, muscarine, strychnine, antipyrine, and quinine. Interference with the destroying function of the liver causes disorder in two ways. (1) By permitting products to pass through the liver into the general circulation, it is probably accountable for certain of the more common symptoms usually ascribed to 'disordered liver'—giddiness, cloudiness of intellect, drowsiness, irritability, and depression (*see* LIVER, Functional Disorders of). (2) By permitting irritant products to pass into the bile-channels, it is a common cause of biliousness. No harm would happen did the liver pour its secretion into the intestine through a wide channel. Unfortunately it happens that the bile with any irritant products it contains has to pass through a whole system of narrow bile-channels lined with secreting cells. The effect of the irritation is to excite an increase of mucus. If it be very intense the mucus secreted may be so great in quantity and so viscid in consistency that the flow of bile is arrested altogether and jaundice results, just as intense as if the flow of bile had been stopped by a ligature applied around the main duct.

Fortunately such poisons are rare and they are only met with as the cause of the more severe forms of jaundice (*see* JAUNDICE). But there can be no doubt that many products of abnormal digestion do possess certain irritant qualities, the excretion of which may occasion a certain retardation in the flow of bile. This is one of the conditions underlying the ailments known as 'biliousness,' or 'torpor of the liver.'

As above shown, in most cases the primary fault leading to disorder of the bile, whether in quantity or quality, arises, not within the liver, but outside it, in the stomach or intestine. The only fault the liver is responsible for is that it has excreted products which have proved more or less injurious to its bile-ducts. *See* GALL-BLADDER and BILE-DUCTS.

WILLIAM HUNTER.

BILHARZIA.—The name given to a genus of flukes, discovered by Dr. Bilharz, of Cairo. *See* ENTOZOA.

BILIARY CALCULUS.—*See* CONCRETIONS; and GALL-STONES.

BILIARY FISTULA.—There are two kinds of biliary fistula: one, in which a communication exists between the gall-bladder and the surface of the body; the other, in which there is a communication between the gall-bladder and other internal organs. Both are rare as the result of disease; but they are not infrequently produced by operation in cases of obstructed bile-duct undertaken in order to divert the flow of bile either outwards or into the intestine.

There are a few cases on record in which a tumour has formed in the neighbourhood of the gall-bladder, and finally has opened spontaneously and discharged a quantity of pus, bile, and gall-stones. Suppuration of the gall-bladder, associated with the presence of gall-stones, is the common cause of these fistulae.

In the second kind of biliary fistula the gall-bladder may communicate with the duodenum or colon; or even with an abscess of the liver; with the portal vein, or with some part of the urinary tract. Gall-stones are in nearly every case the cause of the fistulous opening. *See* GALL-STONES.

WILLIAM HUNTER.

BILIOUS.—This term is used popularly to designate a particular temperament and to describe a peculiar sallow appearance of the skin, suggestive of slight jaundice. When vomit is bile-stained, it is also called 'bilious.' One of the most frequent applications of the term is to certain so-called *bilious attacks* or *biliousness*, which, however, are commonly attacks of migraine (*see* MEGRIM), or are due to digestive disturbances (*see* BILE, Disorders of). In the latter case, the most prominent symptoms are anorexia, furred tongue, a bitter taste, sickness, constipation, and headache, with a feeling of marked depression and general malaise. Such attacks are most effectually prevented and treated by careful regulation of diet. Salicylate of bismuth, cholagogue aperients, and saline effluents may be given, but alcoholic stimulants should be avoided.

BIRTH-PALSIES.—*See* CEREBRAL DIPLEGIA.

BITTER ALMONDS, Poisoning by.—*See* PRUSSIC ACID, Poisoning by.

BLACK VOMIT.—Vomited matters may be more or less black in different diseases, but the term *black vomit* is generally restricted to the vomit occurring in the severest forms of yellow fever (see **YELLOW FEVER**). The rejected matters are acid in reaction, and a sediment is deposited consisting of disintegrated blood-corpuscles and glandular epithelium, granules of pigment, and bacteria. The black colour is due to the altered blood. See **HEMATEMESIS**.

BLACKWATER FEVER.—**SYN.** : Hæmoglobinuric Fever; Bilious Hæmoglobinuric Fever; Fr. *Fèvre Bileuse Hémoglobinurique*; Ger. *Schwarzwasser-Fieber*.

DEFINITION.—An acute infective disease running an indefinite course, characterised by rapid hæmolytic, and giving rise clinically to pyrexia, hæmoglobinuria, and icterus.

ETIOLOGY.—The disease was first described by French surgeons in Madagascar in 1850. Since then it has been observed to occur throughout tropical Africa, in some of the southern states of America, in the West Indies, in Assam and other parts of India, and in Sicily and Sardinia.

Blackwater Fever, at the present time, vies with malignant malarial fever in the amount of invaliding and mortality it causes among Europeans in Tropical Africa. The specific agent is, as yet, undetermined; by some it is believed to be a purely malarial affection, because it occurs only in malarious regions, because the malarial parasite is sometimes found, and because it is prone to attack those who have previously suffered from malarial fever. On the other hand, the seasonal prevalence of the two diseases frequently differs, the malarial parasite is not usually found in the blood, and new arrivals occasionally contract the disease.

Sambon has suggested that the disease is closely related to Texas fever of cattle, and Koch has advanced the view that it is nothing more than quinine-poisoning, although it has been shown that individuals who have never taken quinine suffer from the disease. It is probable that it is caused either by an organism closely allied to, yet distinct from, any of the known species of malarial parasite, by some unknown conditions which modify the ordinary malarial parasite. Chills predispose to attacks. It is very prone to relapse, and the disease may occur for the first time some months after the patient has left the endemic region and returned to Europe.

SYMPTOMS.—The attack may be preceded by fever and malaise, but it is usually abrupt and ushered in by a rigor, or a succession of rigors, the temperature reaching 103° to 105° F. There is pain in the head, back, loins, and abdomen. Bilious vomiting sets in early and persists. Micturition is frequently painful. Within a few hours the skin and sclerotics assume a saffron-yellow tint. In four to six hours sweating occurs and the temperature falls. The rigor may return the next day or within a few hours and be repeated for five or six days. In mild cases there may be only a single rigor. In severe cases, with each succeeding rigor, the symptoms become aggravated. The patient becomes restless, breathless, and complains of thirst, and the vomiting is incessant. In such cases death may supervene as early as the second day, or as late as the second or third week. When death occurs early it appears to be due to the rapid hæmolytic, which may give rise to symptoms like those of hæmorrhage. When it occurs

late in the course of the illness, it is usually due to syncope, suppression of urine, or intercurrent disease. Death from syncope may occur at any time during the attack.

On examination of the blood the plasma is coloured yellow from the dissolved hæmoglobin; the red corpuscles are misshapen and vacuolated; and shadow-corpuscles are present. There is a marked and rapid diminution in the number of the red corpuscles, which may fall to one and a half or one million per cub. mm. in two or three days. The hæmoglobin may also fall to 20 per cent. of the normal amount. There is a mononuclear leucocytosis, and pigmented leucocytes are generally present. The malarial parasite is often absent.

The urine varies in colour from cherry-red to black. On shaking, the froth is pinkish. On standing a copious deposit forms, of a greyish-brown colour, consisting of corpuscular debris, hyaline and blood-casts, and a few red corpuscles. On heating a dense coagulum of globulin forms. Spectroscopic examination reveals the absorption-bands characteristic of hæmoglobin or occasionally of methæmoglobin (see **SPECTROSCOPE IN MEDICINE**). As the patient improves the urine clears rapidly and the globulin disappears. The urine should be examined daily during convalescence, as nephritis occasionally supervenes. The liver and spleen are usually enlarged and tender.

MORBID ANATOMY.—The fluid in the serous cavities is of a bright yellow colour. The liver is enlarged and yellow in colour, and the gall-bladder is usually distended. The spleen is enlarged and its parenchyma soft. The kidneys are congested, and on microscopical examination the convoluted tubules are seen to be filled with hæmoglobin-casts. If pyrexia has been a marked feature of the case, cloudy swelling of the various organs will be present.

DIAGNOSIS.—Blackwater fever has to be distinguished from Bilious Remittent Malarial Fever and from Yellow Fever. In the former there is no hæmoglobinuria, but the urine contains bile and little, if any, albumen. Rigors are not a marked feature, and the temperature is remittent in type. The malarial parasite is invariably present in the blood. The urine on shaking presents a yellow, not a pink, froth. In yellow fever icterus and hæmaturia are late symptoms, and true hæmoglobinuria does not occur. The fever is sthenic in character at the onset, the pulse is slow, and a period of apyrexia precedes the asthenic stage of the disease.

PROGNOSIS.—The mortality varies in different years and in different places. In recent years it has been higher in Central Africa than in West Africa. It fluctuates between 12 and 60 per cent. Mild cases usually recover rapidly, and sometimes apparently hopeless cases pull through. A relapse is often less severe than the primary attack.

TREATMENT.—No specific treatment for blackwater fever has yet been discovered. If the malarial parasite be found, quinine should be administered cautiously, bearing in mind that the drug occasionally aggravates the hæmoglobinuria. A favourite remedy in West Africa, at the present time, is salicylate of sodium. A preliminary purgative of five grains of calomel should be given. Copious draughts of barley-water or some other demulcent drink, containing some mild diuretic, such as citrate of potassium, should be administered. Symptoms of collapse must be promptly treated with hypodermic injections of strychnine or ether. The

usual remedies for vomiting may be tried ; as a rule, however, they do little good. Nourishment must be given if necessary *per rectum*, but it is advisable to persevere with fluids by the mouth, even if they be vomited. In extreme cases, normal saline solution may be injected subcutaneously or intravenously. During convalescence arsenic and other blood-restoratives are indicated. The patient should be strongly advised to leave the endemic area on account of the continued liability to relapses.

DAVID C. REES.

BLADDER, Diseases of.—SYNON.: Fr. *Maladies de la Vessie* ; Ger. *Blasenkrankheiten*.

The bladder may be the seat of the following morbid conditions:—Inflammation, infective (including abscess and ulceration) or non-infective ; Neuralgia ; Atrophy or Hypertrophy ; Mechanical Distension, with chronic engorgement and retention of urine, commonly, but erroneously, termed 'Paralysis' ; Sacculations ; Displacements, such as hernia in the male, or, very rarely, inversion and protrusion in the female ; Tumours or Growths of various kinds, from simple Mucous Polypi to Sarcoma, and Carcinoma ; Cysts ; and Vesico-vaginal or Vesico-intestinal Fistula. The bladder may also be the subject of true paralysis, partial or complete, as the result of injury to, or disease of, the brain or spinal cord.

1. Inflammation of the Bladder or Cystitis may be divided into two classes: (1) *Infective Cystitis*. (2) *Non-infective Cystitis*.

(1) **Infective Cystitis.**—This class includes by far the larger proportion of cases. The inflammation is caused by the presence of some pathogenic micro-organism which infects the urine or the tissues of the bladder, separately or both at the same time. The organism may be introduced into the bladder from *without*, by the passage of a septic instrument, by an injury, or by the extension of gonorrhœa. On the other hand the infection may arise from *within*, being part of a general infection, as in tuberculosis, typhoid fever, or diphtheria ; or as a local infection from a neighbouring organ, as in disease of the rectum, uterus, and alimentary canal. Infective cystitis may be conveniently subdivided into *Acute* and *Chronic* varieties.

A. Acute Infective Cystitis is due to infection from without. The disease is usually ushered in, a few hours after infection, by discomfort in the region of the perinæum and bladder, chilly feelings in the back—in severe cases even rigors—and general malaise. These symptoms quickly give place to frequent and painful micturition, small quantities of urine being voided with urgency and often with straining. There is constant aching and feeling of weight in the perinæum and rectum, with sometimes slight suprapubic pain and tenderness. The temperature is slightly raised, the rise corresponding to the degree of local inflammation. The urine is slightly acid, or alkaline in reaction, cloudy from the presence of mucus, pus, epithelial cells, micro-organisms, and occasionally, in severe cases, blood-corpuscles. Among the organisms commonly met with are the *Bacillus coli communis*, the *Gonococcus*, and various pyogenic cocci. The infective process is confined to the mucous membrane, involving either part or the whole of the surface, which is swollen, congested, and, in severe cases, dotted with small hæmorrhages.

The disease generally subsides in the course of a week or so, though its duration depends to a great extent on the amount of septic infection to which the bladder has been subjected.

TREATMENT.—The *general* treatment consists of rest in the recumbent position, of very hot bidets or hip baths, and hot applications in the form of poultices, fomentations or hot-water-bottles to the lower part of the abdomen and perinæum. To relieve distressing symptoms opium or morphine administered by the mouth, or better by suppositories or hypodermic injections, may be required, but as a rule the preparations of belladonna and hyoscyamus are alone necessary. The diet should be simple ; all alcoholic stimulants should be forbidden and the patient should drink freely of diluents. The *special* treatment is directed to the elimination of the micro-organisms, while at the same time great care should be exercised to prevent reinfection. For this purpose some antiseptic preparation is injected into the bladder. Among those employed may be mentioned solutions of boric acid (2 to 4 per cent.), carbolic acid (.5 to 1.0 per cent.), resorcin 1 per cent., nitrate of silver (1 in 1,500 to 1 in 2,000), perchloride of mercury (1 in 10,000 or weaker), normal salt-solution, and iodoform-emulsion. The internal administration of salicylate of sodium, boric acid, benzoic acid, salol, or urotropine is generally recommended, though perhaps not so immediately effective.

B. Chronic Infective Cystitis arises (1) from the introduction of micro-organisms from without into a bladder already subject to abnormal conditions, such as retention of urine, stone, stricture, tumours, and the presence of morbid urine ; and (2) from infection of the bladder by micro-organisms conveyed from some other part of the body. It not uncommonly commences with all the symptoms of an acute attack, and when these have subsided the patient may have little or no inconvenience for long periods, the only objective symptom being the cloudy condition of the urine. In other cases the disease comes on quite insidiously, the severity of the symptoms gradually increasing. In a few instances the symptoms are pronounced from the beginning and continue throughout the course of the disease. In all forms the patient is liable to acute exacerbations. The symptoms are then very severe, consisting of great pain and frequency of micturition, much straining, and pain in the abdominal and lumbar regions. Rigors are frequent, the temperature remains high throughout the attack, and is associated with grave constitutional disturbance. The urine is thick, offensive, and alkaline. It contains much ropy muco-pus, vesical epithelium, often blood, organisms of many kinds, and various crystals, due to the decomposition of the urine. The structural alteration of the bladder is much more marked and involves all the coats. The mucous membrane is of a greyish appearance, is covered with muco-pus, is thickened and congested, and contains hæmorrhagic extravasations. Exfoliation of the epithelium in large masses may occur, thus giving rise to ulceration, while infection of the deeper layers without erosion may result in *abscess*. The muscular coat is hypertrophied, especially the superficial interlacing fibres, causing the roughened and rugose condition so often noted in bladders long affected with this disease. The blood-vessels of the mucous membrane are dilated, especially towards the neck of the bladder ; these may rupture and give rise to severe hæmorrhage.

Cases of chronic infective cystitis do not often recover completely, unless it is possible to cure the co-existing abnormalities of the bladder.

TREATMENT.—The same general treatment should be adopted for chronic as for acute cystitis, especially when exacerbations occur, but in addition the abnormal condition of the bladder and the sources of infection from neighbouring parts should be removed if possible. Thus in cases of retention the urine should be drawn off by catheter at regular intervals. Stones must be removed, strictures dilated, and specific treatment adopted in tuberculosis. The septic condition of the bladder should be dealt with by injections as already indicated. To the list of internal remedies previously mentioned should be added the infusions and decoctions of *Triticum repens*, *Uva ursi*, *Alchemilla arvensis*, *Buchu*, and *Pereira*, together with the salts of sodium and potassium, as well as the natural mineral waters of Evian, Vichy, and Contrexéville.

(2) **Non-infective Cystitis.**—This form of inflammation is caused (1) by the toxic effect of drugs, as cantharides or turpentine; (2) by the introduction of strong chemical irritants into the bladder, as occurs when instruments that have been disinfected with strong solutions of carbolic acid are passed; (3) by an aseptic injury, as may be caused by some sterile instrument, or by some crystalline deposits from the urine; and (4), although this is questioned, by the presence of residual urine.

The symptoms are similar to those of acute infective cystitis. The urine is generally acid, and cloudy with mucus and epithelium; but it contains no pus or micro-organism. Blood is generally present in considerable quantities when the cystitis is induced by a toxic agent. The attack is transient, subsiding in the course of a few hours after the removal of the exciting cause.

2. **Neuralgia.**—It is impossible to deny that the bladder may be subject, like other parts of the body, to symptoms which are described as neuralgia, although the occurrence is an extremely rare one. All the writers can say is, that they have occasionally met with cases in which they were unable to account, by the existence of any lesion, for pain and frequency in micturition, or for difficulty in performing that act, and where these symptoms have been more or less periodic in their appearance. In such instances quinine or arsenic was given, and occasionally great relief followed a few doses; more frequently this has not been the case. It is invariably necessary to investigate the general health, as well as the habits and diet of the patient. This, perhaps, may be the place for stating that in all chronic and slight deviations from natural and healthy function in the urinary organs, it is necessary to inquire into the state of the digestive organs, and, if possible, to correct by diet, and by medicine when necessary, any imperfect action on their part. Constipation alone, when habitual, may produce considerable irritability of the bladder, a slight affection of the organ already existing; so also may the unnecessary use of purgatives. A daily action of the bowels, and a healthy condition of the digestion, should be ensured as far as possible in all patients complaining of frequent, difficult, or uneasy micturition; and many such may be completely cured of so-called urinary affections by strict attention to these matters. For such patients, the natural aperient waters of Germany and Hungary are of inestimable benefit.

3. **Hypertrophy and Atrophy.**—Where obstruction to the outflow of urine exists, as from stricture of the urethra, enlarged prostate, or tumours, the muscular walls of the bladder become the seat of compensating hypertrophy. Thickening of the muscular walls may also result from long-standing inflammation. Such changes in the interlacing muscular fibres are readily followed by **Sacculation** due to protrusion of the lining membrane between the bands so produced; or the trabeculae may obstruct the orifice of the ureters, causing dilatation of those passages. In some cases the muscular coat undergoes colloid or fatty degeneration. In the former condition the bladder loses its flexibility and elasticity, and in the latter is so weakened that perforation may easily occur. On the other hand, when no increased muscular force avails to empty the bladder, as may occur when the prostate is much enlarged, the bladder becomes gradually distended, its coats become expanded, thinned, and weakened, and a certain degree of **Atrophy** takes place. The power of the organ to expel urine is lost or diminished; and a catheter is the only means of relief. But atrophy and loss of power, needing the habitual use of the catheter, may also occur from complete or partial loss of nervous influence over the bladder, as in those who are the subjects of paralytic states commencing in the spinal cord or brain. Indeed the difficulty in micturition may be the first recognised sign of organic disease of the nervous system.

4. **Tumours.**—Tumours of the bladder are more frequently met with in men than in women. In order of frequency papilloma ranks first, carcinoma second. The following kinds may be enumerated.

(1) **Mucous Polypus.**—Mucous polypus is not to be confounded with prostatic outgrowths of the same form, these being inadmissible in any scheme comprising vesical tumours. It resembles polypus of the nasal cavity, but is more compact and solid in structure, and is chiefly composed of myxomatous tissue. Hitherto this product has been found in young children only.

(2) **Papillomatous Tumours.**—These growths consist of masses of soft papillae (*see* TUMOURS), and were formerly termed 'villous,' to which it was not unusual to add the word 'cancer,' in many cases erroneously, yet in some not an altogether inappropriate term, as carcinoma is liable to develop in the base of a papilloma. These growths, which may be pedunculated or sessile, single or multiple, frequently spring from the fundus of the bladder. When fully developed they give rise to repeated attacks of hæmorrhage, which in time become continuous and copious, thus terminating life. Painless hæmaturia is the characteristic symptom, though in certain cases pain in the lumbar and renal regions is noted from the first, due no doubt to obstruction of the ureters by the growth. In addition there is sometimes increased frequency of micturition, a 'tickling' sensation along the urethra, and occasionally sudden stoppage of the flow of urine. Moreover, on careful enquiry it may be ascertained that, while at the commencement of the act of micturition the urine is clear or almost bloodless, at the end of the act bright florid blood is mixed with the stream, or appears alone at the close. The hæmorrhage arising from papillomata is not infrequently attributed to a renal origin, but the above mentioned symptoms coupled with the fact that microscopical examination of the urine will sooner or later (especially if the

bladder is washed out with sterile water and the débris collected) detect characteristic specimens of growth, and establish the diagnosis beyond a doubt.

TREATMENT.—Papilloma, especially the sessile variety, even when freely removed by operation, frequently recurs. This fact led the writers to adopt the practice of injecting into the bladder a solution of nitrate of silver, with the idea of checking the hæmorrhage and repressing the growth. During the last ten years fourteen undoubted cases have been subjected to this treatment with a result that all have been relieved of symptoms for long periods. The treatment is commenced by injecting daily into the bladder a solution of half a grain of nitrate of silver in four ounces of sterile water (99° F.) acidulated with a small quantity of free nitric acid. The solution is kept in the bladder half a minute and is then allowed to run out. The injections are continued without intermission for four, five, or six months, the strength being gradually increased until a solution of two grains of nitrate of silver to four ounces of water is used. The treatment is then relaxed and the patient not uncommonly remains free from symptoms for a considerable time; though it should be again resorted to whenever blood reappears at short intervals or in large quantities.

(3) *Myomata*.—These are not common, and are unaccompanied by any characteristic signs of their presence.

(4) *Fibromata*.—Both these and the preceding kind of tumour involve the deep structures of the organ, and cannot generally be removed entirely by operative procedures unless they are detected in an early state.

(5) *Carcinomata*.—Primary carcinoma of the bladder is rare, if those forms developing in the base of papillomata be excluded. It usually appears after middle life. Epithelioma is the commonest form, though cases of scirrhus have been recorded. In the early stages these growths may give rise to no marked symptoms, and the disease has probably invaded the organ to a considerable extent and even ulcerated, before the recognised symptoms of pain, frequency of micturition, and hæmorrhage occur.

(6) *Sarcomata*.—These are most commonly met with in children.

The diagnosis of malignant disease of the bladder in its early stages is often attended with much difficulty. At a later period examination of the rectum furnishes valuable information. Scirrhus cancer is very hard, irregular in form, and thus unlike any simple prostatic enlargement occurring in elderly men. Sarcomata are rounded in form, more elastic to the touch than 'prostatic enlargement,' and far more rapid in their rate of increase. Epitheliomata, slow and restricted in growth, may exist some years before presenting a tumour sufficiently dense to be recognised by palpation or sounding. In all forms of malignant disease pain is the most constant symptom, and some hæmorrhage is liable to appear, increasing at a later date, thus contrasting with papilloma, which latter is rarely recognisable through the rectum, commences with hæmorrhage, and is not painful. The progress of epithelioma is slow, and without any very distinctive characters. Microscopical examination of the urine for shreds of tissue and cells furnishes valuable information, but the principal aid in forming an early diagnosis is the judicious use of the cystoscope, which not only demonstrates the

kind of tumour present, but also indicates its size and position. *See* BLADDER, Examination of.

If a diagnosis is made in the earliest stages it may be possible to extirpate the disease. But if the growth largely involves the organ, operation to remove it entirely is almost impossible. Rapid recurrence follows although large masses have been removed and an apparently empty cavity has been left as the result of the procedure. The palliative treatment for all patients thus affected consists in the avoidance of habits, occupations, and forms of exercise found, by the experience of each individual case, to produce serious increase of hæmorrhage. Instrumentation, whenever necessary, should be undertaken with the strictest antiseptic precautions, for the occurrence of infective cystitis adds very greatly to the suffering and distress of the patient. Lastly, all means should be adopted to alleviate suffering by the free use of morphine.

5. Cysts.—They are exceedingly rare, and when they occur usually occupy the posterior wall of the bladder. They may be derived either from remnants of embryonic structures, or produced by the dilatation of some existing canal, or may be of parasitic origin (hydatid). *See* ENTOZOA.

HENRY THOMPSON.

H. T. HERRING.

BLADDER, Examination of.—The following methods are now available for the systematic examination of the bladder:—

1. *Examination by metal instruments.*—The sound is used almost entirely for the detection of stone. It may, however, in rare cases fail even when the stone is of considerable size. In such cases failure is usually the result of the stone being more or less hidden behind a greatly enlarged prostate. The sound, even when its beak is turned downwards, may not hit the stone. Calculi also may be concealed in hernial pouches of the mucous membrane, communicating with the general cavity of the bladder by a small aperture. A mistake frequently made in sounding for stone is to depress the handle of the instrument between the thighs of the patient, and so to keep it during the examination. When once the beak has passed over the prostate and is safely in the bladder, the handle should be somewhat raised, so that the beak is depressed behind the prostate, the most usual position for vesical stone. The sound also detects any roughness or trabeculation of the walls of the bladder. In conjunction with a finger in the rectum the limits of an enlarged prostate may be determined, and the condition of the lower portion and trigonal area of the bladder ascertained.

By the use of a metal evacuating cannula and wash-bottle, such as are used in the operation of litholapaxy, the presence of small stones may often be determined. The calculus is sucked against the eye of the catheter, the resulting click being distinctly audible at some distance from the patient.

2. *Bimanual Examination.*—Rectal or vaginal examination, combined with pressure upon the lower part of the abdomen, is often of great service. In a spare individual the examining hands can almost be made to meet, and the condition of the bladder-walls, the prostate, and the seminal vesicles can be to a certain extent ascertained. With a full-sized bougie or sound in the urethra the condition can be determined still better.

3. *Exploratory operations* are seldom necessary,

or their place is taken by the cystoscope. Still, when the cystoscope fails, an operation may be necessary to determine the condition present. In such cases the bladder may be opened for digital examination either (1) by a perineal section, or (2) by the suprapubic method. For exploratory purposes the latter operation is to be preferred. Great assistance is afforded by the use of caissons or porcelain specula passed through the suprapubic wound and moved over the internal surface of the bladder. A brilliant light can be thrown upon the mucous membrane from an electric lamp worn upon the brow of the operator. The surface can be dried by means of small sponges or cotton swabs in sponge-holders, and the condition of the interior accurately discovered. It is generally advisable to

The catheter-cystoscope is simply an ordinary cystoscope enclosed in an outside metal tube. This tube or metal catheter is first introduced with an obturator in position. The obturator is withdrawn, the bladder washed out, and the cystoscope proper introduced in the place of the obturator. This device obviates the necessity of introducing an instrument more than once in the performance of cystoscopy.

The irrigation-cystoscope can be understood from a glance at the accompanying figure (fig. 2). By its aid a constant stream of liquid can be passed through the bladder, the viscus being under observation the while.

Lastly must be mentioned the highest development of the instrument at present attained—namely, the

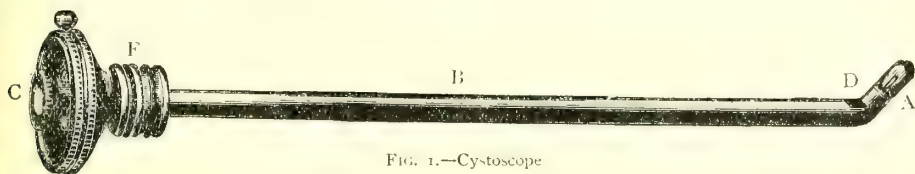


FIG. 1.—Cystoscope

consist upon general anæsthesia for careful bladder-examinations. Local anæsthesia is sufficient to abolish pain, but great inconvenience may be and often is experienced from the spasmodic contraction of the abdominal and perineal muscles, often rendering it difficult to use sound or catheter.

4. *The Cystoscope*.—Many forms of this instrument are now in use. In the instrument preferred by the writer (fig. 1) the illumination is supplied by a small incandescent lamp held in a metal cap A, in which is a small glass window, screwed to the end of the metal tube, B, which contains the telescope, C. At D is another small window, beneath which is the termination of the telescope, and an arrangement of prisms by which the image is projected to

cystoscope or catheterisation of the ureters (fig. 3). The instrument shown in the illustration is that of Max Nitze. A ureter-cystoscope bears upon its upper surface a covered slot or canal, in which fits a mandrel, terminating at the ocular end in a stopcock, while at the other extremity it protrudes through a small opening just behind the lens-window. A screw placed close to the eye-piece communicates to the end of the ureteral catheter a change in direction, illustrated in the diagram by the dotted lines. Through this mandrel is passed the elastic catheter when the instrument is in position in the bladder. When the ureteral catheter is *in situ* in the ureter, the instrument can be withdrawn leaving the catheter in place.

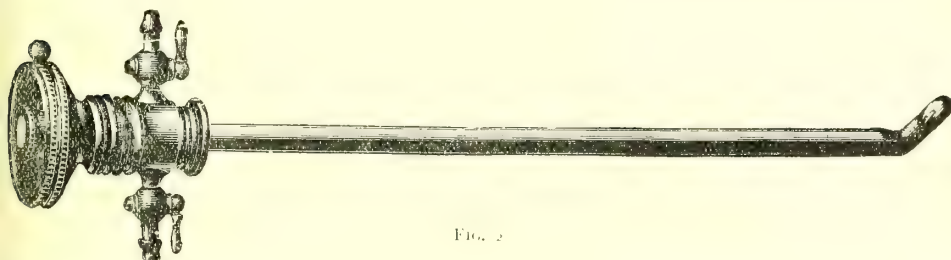


FIG. 2

the eye at C. The size of the instrument equals that of a No. 22 catheter (French gauge). The telescope magnifies the image several diameters, and the observer must consequently allow for this in his observations. The appearances, as in the laryngoscope, are reversed. The wires are connected at F to a rotatory plate, thus enabling complete rotation of the instrument, without the wires being carried with it. By this means one instrument only is necessary for a complete examination of the whole of the bladder, whereas formerly a cystoscope with a window on the under surface of the shaft was used for the base and trigonal area. The ocular rim should be made of ebonite, to avoid shocks should the eyebrows of the observer touch the instrument.

Cystoscopy in the female subject can be performed with the instruments already described. A shorter, wider cystoscope, however, can be employed, the visual field being thereby much enlarged. Howard Kelly, of Baltimore, uses plain metal tubes or vesical specula, through which light is thrown from a brow-mirror. This is practically an adaptation of the laryngoscope and otoscope to the examination of the bladder.

USE OF THE CYSTOSCOPE.—General anæsthesia in male subjects is desirable, but not essential. In the case of females local anæsthesia is sufficient. The patient should be placed on his back with the pelvis raised by means of a hard cushion. If general anæsthesia is declined or undesirable, the urethra and bladder can be rendered sufficiently anæsthetic

by cocaine (*see* ANÆSTHESIA, LOCAL). A drachm of a 2 per cent. solution of cocaine or of β -eucaine, if preferred, is injected into the urethra and retained there for five minutes. A soft rubber catheter is then passed and the bladder emptied. About two ounces of the cocaine-solution is injected into the empty bladder, and the catheter being slowly withdrawn, a drachm or two of the solution is left in the membranous-prostatic urethra. After five minutes' delay the solution is drawn off, and the bladder and urethra will be found in the majority of cases sufficiently anæsthetic for the examination to be pro-

to practise on a 'phantom-bladder.' By this means a certain amount of dexterity in the manipulation of the instrument is obtained.

In certain cases cystoscopy is not practicable. This is the case where the prostate is very much enlarged on its bladder-aspect. The window of the instrument in such a case may not pass from between the immensely enlarged lateral lobes. Hæmorrhage from an enlarged prostate disturbed by instrumentation is sometimes so great that no amount of washing will suffice to clear the medium and enable observations to be made. In other cases a large

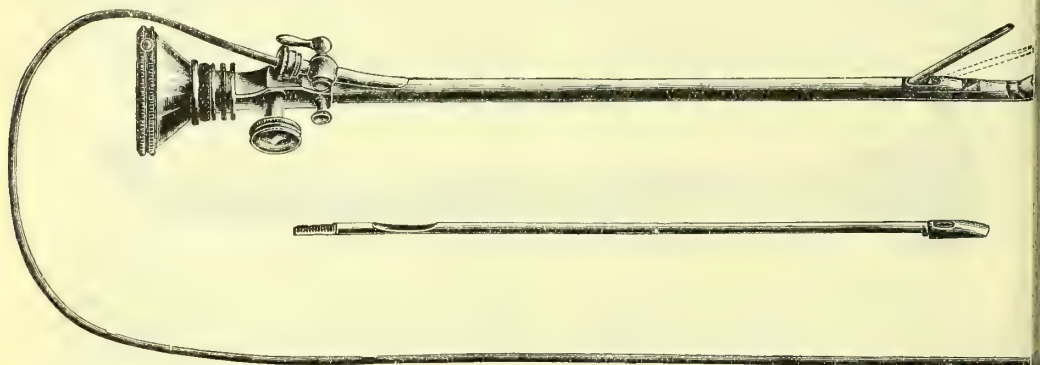


FIG. 3

ceeded with. The bladder is filled with five or six ounces of a boric-acid solution, or warm boiled water, the cystoscope, which has been immersed in a weak carbolic-solution, is gently introduced, and the light turned on. The surgeon should first examine the neighbourhood of the ureteral openings and then the remainder of the bladder. The lamp must not be allowed to come in contact with the bladder-wall for any length of time, or scorching will result. If immersed in fluid but little heating occurs. The light should be turned off, however, for a few moments before the cystoscope is withdrawn. Experience is needed, as in the case of the ophthalmoscope and laryngoscope, to ensure the correct interpretation of the appearances observed. The writer has known an air-bubble mistaken for an ulcer, and a somewhat enlarged prostate for a malignant growth.

In the use of Kelly's tubes the patient is placed in the knee-elbow position, or the exaggerated lithotomy position. The urine is drawn off and the urethra dilated. A tube is passed and the obturator closing it removed. Air rushes in and balloons the bladder. The light is then thrown down the tube and the mucous membrane examined. With a finger in the vagina the posterior wall of the bladder can be pressed towards the end of the tube. Urethral catheters can be passed by the aid of this contrivance. As urine collects in the bladder it is removed by a long-necked sucker or exhaust-syringe. It will be seen that by this method the bladder is dilated with air instead of water, and that the bladder-walls are examined directly. In some cases the bladder is fixed by pelvic inflammation and air does not suffice to dilate it. To these cases the method is of course inapplicable.

It would be of little benefit to enumerate and describe the appearances seen with the cystoscope. These can only be learned satisfactorily by actual practice with the instrument. It is certainly of use

villous growth may wrap itself around the instrument and entirely obscure the view. In stricture of the urethra, it may be found impossible to introduce a cystoscope until full dilatation or internal urethrotomy enables the examination to be carried out.

F. SWINFORD EDWARDS.

BLASTOMYCETIC DERMATITIS.—*See* DERMATITIS.

BLEB.—A large vesicle or bulla, containing serous fluid, as in pemphigus, erysipelas, or burns and scalds. *See* BLISTER.

BLÉNORRHOËA, BLÉNORRHAGIA (βλέννα phlegm; and *ῥέω*, I flow βλέννα, phlegm; and *ῥήγνυμι*, I burst out).—These terms are most correctly used to express excessive flow of mucus from any mucous surface. By means of an affix, the locality or nature of the discharge is expressed e.g. *blenorrhoëa oculi, nasalis, urethralis*.

BLEPHARITIS (βλέφαρον, an eyelid).—Inflammation of the eyelids. *See* EYE AND ITS APPENDAGES, Diseases of.

BLEPHAROSPASM (βλέφαρον, an eyelid; and *σπᾶσμα*, a spasm).—Spasmodic movement or contraction of the eyelids. *See* EYE AND ITS APPENDAGES, Diseases of.

BLINDNESS.—Loss of sight. *See* AMAUROSIS and VISION, Disorders of.

BLISTER.—*SYNON.*: Bleb; Bulla; Fr. *Bulle* Ger. *Blase*.

DEFINITION.—A vesicle of the skin caused by the separation of the horny cuticle from the rete mucosum by the transudation of serous lymph beneath the former.

ÆTIOLOGY.—Blisters may occur in certain skin-affections such as pemphigus. They are also seen in the cutaneous manifestations of some of the acute infectious diseases, such as small-pox and chicken-pox. In addition they are met with under the influence of any cause which depresses the vitality of the integument, such as burns and scalds, or as an effect of powerful irritants, such as cantharides or the aniline-salts. Micro-organisms have been obtained from the contents of some vesicles, especially in certain forms of pemphigus.

DESCRIPTION.—A blister ranges in size from that of a pea to a turkey's egg. It is more or less convex according to the amount of exudation contained in it. It conforms in colour with that of its contents, being sometimes yellow or amber-coloured and transparent like serum, sometimes opalescent from the presence of pus, and sometimes red or purple from admixture with blood. The fluid of a blister is generally limpid and free, is sometimes held in the meshes of a delicate network resulting from the stretching of the cells lying between the rete mucosum and the horny epidermis. This is peculiarly the case in blisters developed under the influence of acute inflammation and especially in the dermatitis affecting workers in aniline. Blisters may be scattered over the body, or aggregated, or even single as in pemphigus.

TREATMENT.—Blisters are essentially asthenic in their nature and call for general tonic treatment. Locally they should be punctured so as to admit of the gradual escape of their contents and then dusted over with some antiseptic or absorbent powder such as oxide of zinc or boric acid.

ERASMUS WILSON.
E. F. TREVELYAN.

BLISTERING.—A therapeutic measure, which consists in the artificial production of blisters on the skin. See COUNTER-IRRITATION.

BLOOD, Transfusion of.—See TRANSFUSION OF BLOOD.

BLOOD, Abstraction of.—SYNON.: Bleeding; Blood-letting; Fr. *La Saignée*; Ger. *Der Aderlass*.

The withdrawal of blood from the body for therapeutic purposes may be effected (1) by the opening of a vein (venesection); (2) by leeches, and, more rarely (3), by scarification, and (4) by wet-cupping.

1. Venesection (see VENESECTION).—This procedure is employed in those cases in which extreme venous engorgement has produced so much dilatation of the right side of the heart that the cardiac muscle is unable to contract efficiently upon its contents and there is a danger of a cessation of its action during diastole. Such a condition may arise in valvular diseases of the heart, in acute and chronic diseases of the lungs, and in other conditions giving rise to the phenomena of asphyxia. In such cases the withdrawal of from four to eight ounces of blood may sufficiently relieve the intra-ventricular pressure on the right side of the heart, and enable it to contract more vigorously upon its contents. Venesection is not likely to be of much use if the walls of the heart are extensively degenerated; and it should always be avoided in the aged and the feeble. It is very rarely required in childhood. The cessation of the progressive improve-

ment in the character of the heart's impulse and of the radial pulse furnishes the best test of the moment when the flow of blood should be arrested.

Venesection is sometimes advocated in diabetic coma, uræmic convulsions, and other conditions believed to be due to poisons circulating in the blood. In these cases it is of doubtful service, and if employed should be supplemented by the infusion of normal saline solution.

2. Leeches.—From two to twelve leeches may be applied to any part of the skin, although care should be taken to avoid parts over which compression cannot easily be made should the subsequent bleeding be otherwise uncontrollable. The track of a superficial vein should also be avoided.

In applying a leech the skin should first be cleansed and the part wetted with a little boiled milk. A leech-glass facilitates the operation and ensures the application of the leech to the exact place selected. An average leech will at most abstract half an ounce of blood; as a rule the amount is distinctly less than this. If it is desired to continue the bleeding a fomentation may be applied after the leeches have fallen off; but should the bleeding continue too freely, dry aseptic dressings, styptics, or pressure may be applied.

Leeches are now principally employed for the relief of severe pain due to an acute local inflammation, and are thus used in iritis, pericarditis, appendicitis, and many other conditions.

3. Scarifying.—In this method a number of small parallel cuts, of a depth not exceeding an eighth of an inch, are made in the tissue from which it is desired to withdraw the blood. Scarifications are thus occasionally made in the cervix uteri and in the tongue, but the method is not so frequently resorted to as formerly.

4. Wet-cupping.—The method adopted in this almost obsolete procedure is at first the same as in dry-cupping (see CUPPING). The glasses are then removed, the raised portion of skin is scarified, the glass re-applied and finally removed when the desired amount of blood has escaped.

Wet-cupping is very rarely employed on the loins in nephritis, and on the chest when venesection or the application of leeches is impracticable.

JOHN HAROLD.

BLOOD, Clinical Examination of.—The systematic examination of the blood involves the estimation of (1) the number and condition of the red corpuscles including their hemoglobin-content; (2) the number of the white corpuscles and the relative proportion of the different kinds; (3) the number of blood-plates; (4) the rapidity of coagulation; (5) the specific gravity; (6) the alkalinity; (7) spectroscopic changes; (8) the serum-changes; and (9) the bacterial contamination.

Fortunately, the first two and the last two of these procedures generally give all the information required. In this article it is proposed to give only such a short and simplified account of them as will be found suitable to the requirements of the practitioner.

1. The obtaining of the blood.—The lobe of the ear is rubbed first with a wet and then with a dry towel; antiseptics are unnecessary. The ear is steadied and a moderately deep prick is sharply made with an ordinary three-sided surgical needle in the convex border of the lowest part of the lobe. The blood should flow freely and no pressure should

pipette is shaken, a drop deposited on the ruled disc of the counting chamber, and the cover-glass adjusted all exactly as before, but instead of counting the leucocytes in certain squares only, they are counted over the whole ruled space, i.e. 400 squares. The process is repeated with a second drop, and, if

vaseline or oil round the rim of the cover-glass, the leucocytes may be studied for a long time and a differential count be made even when the cells are still alive.

4. ESTIMATION OF THE HÆMOGLOBIN.—The total amount of hæmoglobin is diminished in all

anæmias, and its percentage is often lowered in proportion to the loss of red cells and often also independently thereof. Taking the percentage of hæmoglobin as the numerator, and the percentage of red cells as the denominator, we get a fraction $\frac{\text{Hb. percentage}}{\text{R.C. percentage}}$ which represents the relative richness of the individual corpuscles, and is conveniently spoken of as their colour-index. It is = 1 when normal. It is lowest in chlorosis, where the loss in corpuscles is often comparatively slight, and highest in pernicious anæmia, where alone of all anæmias the hæmoglobin is usually relatively increased, although the diminution in red cells is so great. In such cases the colour-index often exceeds 1.

The percentage of hæmoglobin is estimated by means of a hæmoglobinometer. Oliver's (fig. 2) is preferable to that of Gowers or of von Fleischl. It consists of 12 tinted glass discs arranged in two rows and corresponding to definite percentages of hæmoglobin, ranging from 10 to 120 (a). The intervening percentages are ascertained by the help of small squares of tinted glass known as 'riders,' and having values of 5 per cent., 2·5 &c., which being placed upon any of the primary discs deepen its tint. The blood is collected in a short pipette (c) which is first cleaned out and dried by passing through it a needle threaded with darning cotton. The blood should fill the pipette in an unbroken column. The rubber nozzle of the mixing pipette (d) previously charged with water is fitted over its blunt end, and the blood is gently washed out with the

first few drops of water into the mixing chamber (e). The water is gently added drop by drop until the chamber is full to the brim, the handle of the pipette (c) being used as a stirrer. An oblong piece of colourless glass is provided, and this is now adjusted as a cover-glass, and the small air-bubble which forms beneath it shows that the cell has been properly filled. The blood-chamber is now brought close to the tinted discs for comparison. The reading is done by candle-light (small Christmas candles are best) placed about four inches away,

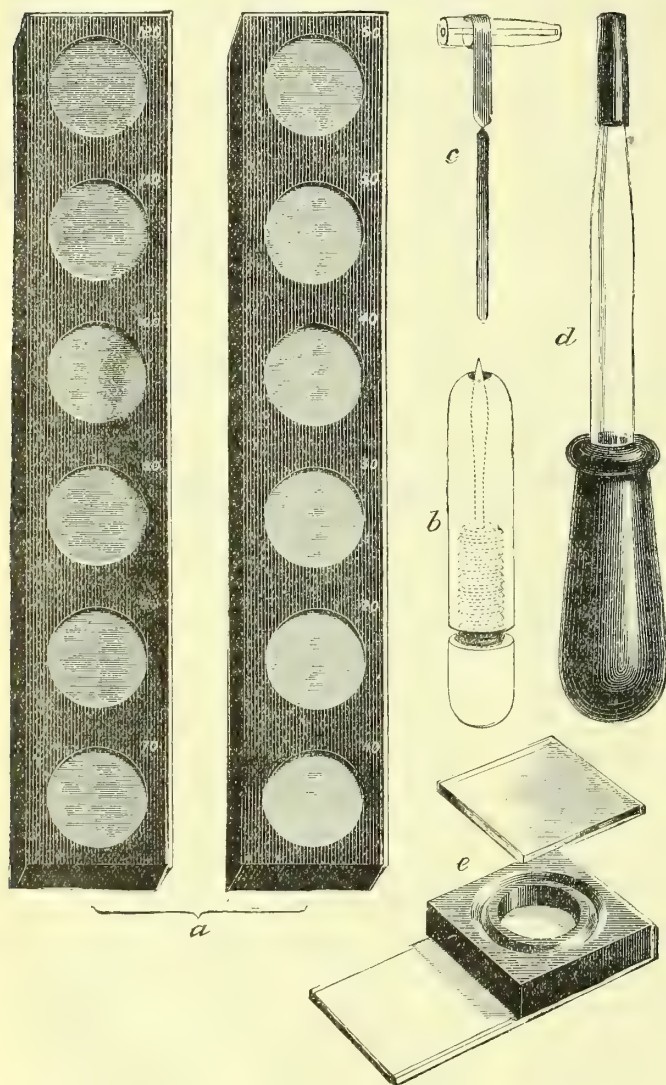


FIG. 2

necessary, with a third, an average being taken as before. In this case,

$$\frac{\text{No. of leucocytes in 400 squares} \times 10 \text{ (the dilution)} \times 4,000}{400}$$

= the no. of leucocytes in 1 c.mm. of blood. 7,000 to 8,000 may be taken as the average in health. By using Toisson's diluting fluid, or Sherrington's, viz. Ehrlich's methylene blue, 0·1 grm.; sodium chloride, 1·2 grm.; neutral potassium oxalate, 1·2 grm.; distilled water, 300 c.c.; and painting

and the same above the level of the discs and blood-chamber, and placed so that the light falls upon and is reflected from both equally. In daylight this must be done in a dark place. A set of discs can be procured for use by daylight, but they are less accurate. All side-light is excluded by using the flexible camera-tube. Short observations of about ten seconds each should be made, as the eye is thereby rested and can better appreciate delicate differences. If the colour of the blood-chamber exactly corresponds to any one of the standard series, the percentage of hæmoglobin can at once be given, but if it lies intermediate between any two, it is placed opposite the lower, upon which riders are placed until this approaches the tint of the blood-chamber.

5. The specific gravity of the blood gives us the same information as the hæmoglobin-value, and the method of ascertaining it need not be described.

6. THE EXAMINATION OF THE SERUM.—The serum-diagnosis of typhoid fever, cholera, plague, and other diseases will be found referred to in the articles on those diseases, in which the utility of the method has been established.

7. THE DETECTION OF BACTERIA IN THE BLOOD.—Bacteria are rarely present in such numbers as to be capable of detection in films, and usually can only be obtained by cultures: the *Bacillus anthracis* and the *Spirillum* of Relapsing fever are, however, notable exceptions to this rule. The skin over the flexor aspect of the elbow is sterilised and then washed with boiled water to remove all traces of the sterilising solutions. Pressure is exerted on the arm above the elbow to bring out the veins. The needle of a hypodermic syringe (previously sterilised by boiling) is inserted into the most prominent one, and about 2 c.c. of blood are drawn into the syringe by gently raising the piston. The needle is then withdrawn and the blood at once expelled into a sloped agar or blood-serum tube so that it runs over the surface of the medium and collects a little at the bottom. The tubes are incubated, and any culture obtained is examined in the usual ways. A pad of sterilised gauze is placed over the puncture. The discovery, in this way, of pneumococci, staphylococci, gonococci, or other bacteria, not only assists in the diagnosis, but warrants a grave prognosis.

III. The Examination of Fixed Blood.—This is the most generally useful and valuable of all the methods, and should be practised in all cases whether any other method is used or not. It enables us after some practice not only to estimate roughly the number and condition of the red cells, their hæmoglobin-content, and the number of the white cells; but it also brings out the finer morphological structures of the latter and tells us the relative numbers of the different varieties. It is carried out as follows:

I. THE PREPARATION OF THE FILMS.—A small drop of blood is received upon the centre of one clean cover-glass (*v. supra*), which is then gently dropped, blood downwards, upon another, when the blood will spread itself out in an even layer between the two. A corner of each is now grasped in a pair of forceps and they are gently slid apart and allowed to dry, face upwards, in the air, care being taken that they are never pressed together either before or during separation, and that they are afterwards protected from dust. Beginners almost always take too large a drop, and hence they

may use the ungummed edge of a cigarette-paper or bit of gutta-percha tissue, which is moistened in the drop of blood and then rapidly drawn along the surface of a slide; or they may transfer a correct drop to the cover-glass by a measured platinum loop. Whichever plan is adopted, the procedure must be quickly carried through.

2. THE FIXATION OF THE FILMS.—(a) *By formalin-vapour*.—The films are exposed to the vapour of 40 per cent. formalin for five minutes. The formalin may be put in any convenient dish, e.g. the half of a Petri capsule, a watch-glass, or even a wine-glass. In any case, the top of the dish or glass is closed by a cover (e.g. of cardboard), with a central aperture large enough to enable the cover-glass to be placed film downwards and exposed to the vapour of the formalin. The films are next exposed to the air but protected from dust for some time, as any traces of formalin in them interfere with staining. (b) *By heat*.—This may be applied in various ways, e.g. by passing the cover-glass, film upwards, about twenty times through the flame of a Bunsen lamp, but is best done by placing the films in a dry steriliser, or some simple substitute, and gradually raising the temperature to 120° C., which is kept up for ten minutes to two hours before removing the films. Even better results may be obtained by gradually raising the temperature to 150° C., allowing to cool, and then removing the films. This takes generally about ten minutes. Insufficient heating is a commoner error than overheating. (c) *By absolute alcohol and other (equal parts)*.—The films are simply placed therein for at least half an hour. They are then taken out and dried in the air. This method is most successful with the red cells, but also gives good results with the white.

3. THE STAINING OF THE FILMS.—For good results, Gruber's stains, which are now readily obtainable, should be used. A combination of a basic and acid aniline stain, such as methylene blue and eosin, or Ehrlich's triacid stain must be used. Both give beautiful and reliable results. The first is applied as follows: (1) Place the film in a two per cent. watery solution of eosin for two to four minutes, (2) wash and dry, (3) dip in water, (4) place in saturated watery solution of methylene blue for half to two or three minutes according to result observed (test by placing under microscope at intervals), and (5) wash in water, dry, and mount in Canada balsam. The red cells are stained pink, the nuclei of the white cells are coloured blue, and their coarse granules deep pink. These are therefore called *eosinophile*; while the fine granules, staining pale pink or remaining colourless, are called *neutrophile*. Ehrlich's triacid stain is a mixture of orange G., acid fuchsin, and methyl green combined in varying proportions by different workers. The stain is applied for several minutes, and the film is then washed in water, dried and mounted. The red cells are stained orange, the nuclei of the white various shades of blue, the eosinophile granules copper-colour, and the neutrophile granules a violet or pink tint.

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BLOOD, Morbid Conditions of.—In considering this subject the following arrangement will be adopted.

A. CHANGES IN THE CORPUSCULAR ELEMENTS.—I. The red corpuscles. II. The leucocytes. III. The blood-plates.

B. CHANGES IN THE FLUID PART OF THE BLOOD.—I. Those affecting the blood as a whole, e.g. alterations in volume, specific gravity, and chemical reaction. II. Those due to variations in normal chemical constituents or to the presence of abnormal substances.

A. CHANGES IN THE CORPUSCULAR ELEMENTS.

1. **The Red Corpuscles.**—Variations in the red corpuscles are of common occurrence in disease, and these may be considered under the following heads: (a) Number per c.mm.; (b) Size and shape; (c) Richness in hæmoglobin; (d) Staining-reaction and other physical characters; (e) The appearance of abnormal elements in the blood, especially nucleated red blood-corpuscles.

(a) *Variation in number.* Although the total number cannot be readily ascertained, much is known regarding the variations in a given quantity of blood, 1 c.mm. being usually taken as the unit. *Polycythæmia*, or an increase above the normal number of 5,000,000 per c.mm., is met with in a few conditions. 1. What may be called a physiological increase takes place when the person ascends to higher altitudes, an increase of about 2,000,000 being observed at a height of 6000 feet. This increase begins within a few hours, and is said to be due to an increased number of small red blood-corpuscles and poikilocytes, which are probably formed by splitting or budding of the original corpuscles; there is no increase of the hæmoglobin. Some days afterwards there is an increased number of red blood-corpuscles of normal appearance, and the percentage of hæmoglobin also rises. These changes are supposed by some observers to indicate an increased formation of red corpuscles—a compensatory change brought about by the diminished supply of oxygen. Others, however, maintain that concentration of the blood by loss of water is sufficient to account for the phenomenon. On return to a lower level, the number of red corpuscles shows a comparatively rapid fall. 2. In general chronic venous congestion, e.g. in mitral and congenital heart-disease, there is often an increase in the red blood-corpuscles, and sometimes this is very marked, a count of 7,000,000 per c.mm. being by no means exceptional. This is probably brought about by altered conditions of the circulation, and is hardly to be regarded as of compensatory nature. 3. In poisoning with phosphorus and with carbonic oxide there is usually polycythæmia. 4. In conditions in which there is a large withdrawal of fluid from the blood, e.g. in cholera, a varying degree of polycythæmia results.

Oligocythæmia, or a diminished number of blood-corpuscles, is of common occurrence. When a single large hæmorrhage occurs, usually about a day elapses before the number of red blood-corpuscles per c.mm. reaches its lowest level. This of course corresponds with the period when the restoration of the fluid has been completed by absorption, partly from the tissues, partly from the alimentary canal. Then follows the period of regeneration, in which the number of red blood-corpuscles rises—slowly at first, more rapidly later—until the normal number is regained. The percentage of hæmoglobin is, however, not restored until some time later, the corpuscles in the meantime showing a deficiency in hæmoglobin. In healthy animals more than a million corpuscles per c.mm. may be restored in the course of a week, and sometimes the process is

equally rapid in the healthy human subject. More frequently, however, the restoration is much more gradual. After a single large hæmorrhage there may be practically no alteration in the appearance of the red blood-corpuscles with the exception that some may show the polychromatophile reaction (*vide infra*). If, however, hæmorrhages are repeated, and a chronic oligocythæmia is produced, variations in the size and shape of the corpuscles appear, many corpuscles being under-sized and containing a relatively smaller amount of hæmoglobin. Nucleated red blood-corpuscles of normoblastic type may be present in the general circulation. Leucocytosis and increase of the blood-plates usually accompany these changes. When such a condition has been established, restoration occurs much more slowly than in the case of a single large hæmorrhage. The source of red blood-corpuscles in the adult is from the nucleated red blood-corpuscles in the bone-marrow, and after hæmorrhages the number and proliferation of the latter are much increased.

Of the various diseases attended with oligocythæmia pernicious anæmia is that in which the most extreme degree is met with. The number frequently falls below a million (*see ANÆMIA, PERNICIOUS*). In chlorosis the number is in many cases only slightly reduced (*see CHLOROSIS*). Oligocythæmia is common in many chronic diseases associated with a greater or less degree of cachexia, e.g. malignant disease, phthisis, prolonged suppurations, lead-poisoning, chronic gastric catarrh, &c. Such cases of oligocythæmia are included under the term 'secondary anæmia.' The greatest fall is probably met with in cancer of the alimentary canal, the number sometimes falling to 1,000,000 per c.mm. In chronic malaria the anæmia resulting from the direct destruction of red corpuscles may be very extreme, and may in fact reach a similar or even lower level. In the production of oligocythæmia in these various diseases, excessive or abnormal destruction and deficient formation of red corpuscles are variously concerned (*see ANÆMIA, PERNICIOUS; CHLOROSIS*). After acute infective fevers and various toxic conditions, a distinct fall in the number of corpuscles may be observed. This is in chief part due to destruction of the red corpuscles, distinct evidence of which may be found in the spleen; the presence of pathological urobilin in the urine is usually regarded as another indication of the same process.

(b) *Variations in size and shape* are often associated. Corpuscles larger than normal are usually called *megalocytes*; those smaller than normal, *microcytes*. Megalocytes are common in pernicious anæmia, the round corpuscles reaching 10 or 12 μ in diameter, the oval corpuscles 14 μ or even more in their long axis. In fact, although microcytes are also common, the average size of corpuscles in this disease is usually above the normal, and there is an increase in the amount of hæmoglobin per corpuscle. In other anæmias, variations in size also occur, but megalocytes are relatively few in number, microcytes being much more numerous; as a result the average size of corpuscles is diminished. Microcytes may be 4 μ in diameter, or even less. Abnormally shaped corpuscles or *poikilocytes* (*see fig. 5*) may be met with in practically any variety of oligocythæmia when it is severe. Their presence in itself has therefore no diagnostic significance. Poikilocytosis, however, is most striking in cases of pernicious anæmia, where the presence of a great variety of forms is a striking feature. In chlorosis

it is by no means uncommon, and in severe post-hæmorrhagic and in cachectic anæmia the presence of numerous small poikilocytes is often noteworthy. The significance of such changes in shape is doubtful. According to Ehrlich's view it is to be regarded as a salutary process in which splitting or budding of the corpuscles takes place in order to provide an increased respiratory surface; hence he called these irregular forms *schistocytes*. According to another view the condition is simply to be interpreted as the effect of impoverished nutrition on the stroma of the red blood-corpuscles. Poikilocytes can be produced artificially by carefully heating the blood outside the body. The smaller microcytes are probably formed by the same agencies as the poikilocytes, there being no evidence that they represent younger corpuscles which afterwards undergo enlargement.

(c) *Variations in hæmoglobin*.—The amount of hæmoglobin in a given volume of blood (see BLOOD, Examination of) is expressed as a percentage of the normal. The amount of hæmoglobin per corpuscle, or 'colour-index,' is obtained by dividing the percentage of hæmoglobin by the percentage of corpuscles, taking the normal as $\frac{100}{100} = 1$. In the various anæmias the percentage of hæmoglobin is diminished, but to a different extent. In chlorosis we find a much greater diminution of the hæmoglobin than of the number of red corpuscles (see CHLOROSIS). In pernicious anæmia, on the other hand, the hæmoglobin is less diminished than the number of red blood-corpuscles, and the colour-index is raised, the average being 1.05 to 1.1. In the secondary anæmias and in the so-called simple anæmias the colour-index is slightly less than normal, though not so low as in chlorosis; in leucocythæmia also it is generally a little under the normal. As already stated, the colour-index is also lowered after hæmorrhages, and the normal condition is not regained until some time after the number of red corpuscles has reached the normal level.

(d) *Alteration in staining-reaction, &c.*—Of these the most important is what is known as 'polychromatosis,' 'polychromatophile regeneration,' or 'anæmic degeneration,' a condition in which the normal staining-reaction of hæmoglobin is diminished or lost. Thus in staining with Ehrlich's eosin and hæmatoxylin, the affected corpuscles are coloured violet or dull blue instead of a pure pink colour (fig. 5A). At the same time they have diminished consistence, their form being less easily preserved than that of the normal corpuscles; thus in film-preparations they often show an irregular margin. According to one view this is a degenerative change due to impaired nutrition. Another view is that these corpuscles are young forms somewhat labile and deficient in hæmoglobin, and in support of this view it has been pointed out that various nucleated red blood-corpuscles show in their protoplasm the same staining-reaction and other alterations. The matter cannot be definitely settled, but it is probable that some of the corpuscles showing the change mentioned are really in a degenerated state. The conditions in which this change is met with are chiefly the various anæmias and cachectic conditions.

A diminished consistence with increased adhesiveness, whereby the corpuscles tend to clump into irregular masses, may be observed in some severe toxic conditions, malignant jaundice, typhus fever, &c. It may be added that absence of rouleaux-

formation when the blood is shed is common where there is marked oligocythæmia.

One other subject may be referred to here, viz. alterations in the resistance of the red corpuscles to various physical and chemical agencies, e.g. heat, electricity, dilution of the plasma. In the case of the last-mentioned, which has been most studied, the greatest dilution which may be made without producing diffusion of the hæmoglobin is obtained, this being called the 'isotonic point,' or 'isotonic concentration.' The isotonic point, which in the case of normal blood is about .45 per cent. as regards sodium chloride, will be of course higher when the resistance of the corpuscles is diminished. This has been found to be the case in various toxic and infective conditions, in anæmia, &c. Accordingly these results obtained are not of much diagnostic value. In jaundice on the other hand the isotonic point is lowered.

(e) *Presence of nucleated red corpuscles, &c.*—These cells, which are present only in the bone-marrow in the normal state, appear in the circulating blood in a number of conditions of disease. In the medullary form of leucocythæmia they are practically always present and are met with in greater number than in any other disease. In some cases three or four may be found in every microscopic field. In lymphatic leucocythæmia, on the other hand, they are very scanty, and in fact often entirely absent till a late period of the disease. In pernicious anæmia the presence of nucleated red blood-corpuscles is of almost invariable occurrence, though they may be few in number and difficult to detect. In severe post-hæmorrhagic anæmia, they may be fairly numerous, and in cachectic anæmia they may appear in the blood, though usually in very small numbers. They may also be present in severe malarial anæmia. In chlorosis, on the other hand, their presence is exceptional. Lastly, in severe toxic and infective conditions, nucleated red blood-corpuscles may occasionally be detected, usually in association with myelocytes; the presence of these two classes of cells indicates a grave condition.

Nucleated red blood-corpuscles are usually divided into two main classes, *normoblasts* and *megaloblasts* (fig. 6). Two other varieties, *microblasts* and *poikiloblasts*, are also distinguished. A normoblast measures as a rule 8–9 μ in diameter, and usually contains a single spherical nucleus, about 3–5 μ in diameter, often more or less eccentric in position. The larger nuclei show a coarse reticulum of chromatin; those of smaller size appear more or less homogeneous. Occasionally there are two or even more nuclei of small size. In all cases the nuclei stain very deeply with nuclear stains. The megaloblast is of large size, sometimes reaching 12 μ or even more in diameter. Its nucleus is fairly large and stains faintly, sometimes being diffusely stained, sometimes showing a fine reticular or granular appearance. It must be noted, however, that some megaloblasts have a fragmented nucleus, with deeply staining chromatin. The protoplasm of the megaloblasts in film-preparations often shows an irregular margin and gives the polychromatophile reaction. Of the various conditions noted, pernicious anæmia is the only disease in which megaloblasts are met with as a rule. This circumstance agrees with the fact that the average size of red corpuscles in this disease is raised. Ehrlich believes that in pernicious anæmia the formation of red blood-corpuscles is of a different type (viz. the megaloblastic

type) from that in other diseases. Cases of this disease vary very much as regards the number of megaloblasts present. In some instances they are comparatively numerous, in others it may be extremely difficult to find any. The prognosis is considered by some observers to be more serious in the former condition. It may also be mentioned that in the anæmia produced by the *Bothriocephalus latus* megaloblasts may be present in the blood, the various changes closely resembling those in pernicious anæmia. Here, however, the prognosis is much less grave, as cure often results on the removal of the parasite. In the other diseases mentioned the nucleated red blood-corpuscles are of the normoblastic type. They are usually comparatively scanty, but in certain states, believed to be associated with increased regeneration and called 'blood-crises,' a large number of nucleated red blood-corpuscles may appear in the circulation. As a general rule the presence or absence of normoblasts in the conditions specified cannot be said to have much prognostic significance.

Microblasts are small nucleated red blood-corpuscles 5-6 μ in diameter. They are sometimes found along with megaloblasts in pernicious anæmia. A *poikiloblast* is merely a nucleated red blood-corpuscle of irregular form and may be of the normoblastic or megaloblastic type. It may further be noticed that the appearance of nucleated red blood-corpuscles in the circulation occurs more readily in earlier years than in adult life.

As other abnormal structures we may mention irregular fragments of red corpuscles, which have been observed in the blood after severe burns and after certain poisonings, e.g. with pyrogallol acid. In hæmoglobinæmia (*vide infra*) decolourised red corpuscles may sometimes be detected, their stroma presenting a pale shadow-like appearance (*shadow-corpuscles*).

II. The Leucocytes.—In connection with changes in the leucocytes in disease, we have to consider (1) variations in the total number; (2) variations in the relative proportions of the different varieties; and (3) the presence of abnormal forms. The measurements that will be given are those obtained from the fresh blood; in a dried film the leucocytes may appear larger, owing to flattening.

In normal conditions the number per c.mm. may be said to be 6,000 to 10,000, though it may sometimes pass beyond these limits. The chief varieties (*see fig. 1*) are:—(a) Lymphocytes, about 7-8 μ in diameter, with spherical and deeply staining nucleus and scanty protoplasm; non-amœboid and non-phagocytic; numbering about 20-22 per cent.; (b) Large mononuclear or hyaline leucocytes, 9-12 μ in diameter, with spherical, oval, or slightly indented nucleus; in some the nucleus is more lobulated, the so-called 'transitional forms.' The protoplasm is more abundant and the nucleus stains less deeply than in the case of the lymphocytes. They are more or less amœboid and phagocytic, numbering 4-5 per cent. (c) Finely granular neutrophile or polymorphonuclear leucocytes, 9-10 μ in diameter. The nucleus is single, but much lobulated so as to present a great variety of forms; hence these leucocytes are often spoken of as 'multinucleated.' The nucleus is rich in chromatin and stains deeply. The protoplasm contains granules which give a 'neutrophile' reaction, though they can also be stained with acid dyes, that is, are feebly 'oxyphile.' These leucocytes are actively amœboid and phago-

cytic; they number about 70 per cent. (d) Coarsely granular or eosinophile leucocytes. The nucleus is polymorphous and the granules large, with strong affinity for acid dyes. They are actively amœboid, but not as a rule phagocytic. They number 3-5 per cent. (e) Leucocytes with mast-cell granules. These are coarse granules which stain deeply with basic dyes and give a metachromatic reaction, staining, for example, a violet tint with methylene blue. They are rarely present in normal blood.

LEUCOCYTOSIS.—Under normal conditions slight increase in the number of leucocytes occurs in physiological states, e.g. after a meal, especially when rich in proteids, during pregnancy, and after parturition. Such increase—physiological leucocytosis—rarely reaches more than 25 per cent. of the normal. In numerous pathological conditions the number undergoes marked variations. When it rises above normal the term *leucocytosis* is applied (leucocythæmia, which will be described separately, being excepted); the converse condition, a degree below the normal, is called *leucopenia* or *leucocytopenia*. As a distinct leucocytosis is nearly always due to an increase of the neutrophile leucocytes, the term 'leucocytosis' or 'ordinary leucocytosis' is employed as equivalent to a neutrophile leucocytosis. Lymphocytosis signifies an actual increase of the lymphocytes, though it is often employed when there is a percentage increase of these cells. According to the latter significance it will be present when there is a marked fall in the number of neutrophile leucocytes. Eosinophile leucocytosis or eosinophilia signifies an increase of the eosinophiles; this is in most cases approximately indicated by the percentage of these cells. We shall now consider the conditions in which these variations occur.

1. Ordinary or neutrophile Leucocytosis.—As already stated, the essential factor is the increase of the neutrophile leucocytes (*fig. 2*). The degree varies in different cases. 15,000 and upwards per c.mm. may be considered a well-marked leucocytosis, 30,000 and upwards an extreme leucocytosis, though 50,000 or even more may sometimes be reached. The proportion of neutrophiles may rise as high as 90 per cent. or even higher. The conditions of occurrence, which are both numerous and various, may be considered under the following heads:—(a) *Inflammatory and infective leucocytosis*. Here may be included a large number of bacterial infections, and also some of the specific fevers in which the virus has not been discovered. Leucocytosis is usually well marked in pneumonia, erysipelas, diphtheria, scarlet fever, plague, small-pox in the suppurative stage, in many acute inflammatory conditions, especially when suppuration is present, e.g. in pyæmia, peritonitis, arthritis, &c., and also in many spreading gangrenous conditions. The degree varies in different cases of the same disease and shows irregular fluctuations during its course. In contrast to the neutrophiles in these conditions the eosinophiles are diminished in number or may be absent; in pneumonia, for example, it is often impossible to find any of these cells in the ordinary course of the disease. On the subsidence of the disease the leucocytosis disappears: when defervescence occurs by crisis the fall is rapid, when by lysis it occurs more slowly. The evacuation of a large quantity of pus is usually accompanied by a rapid fall. A fall in the number of leucocytes without improve-

SPECIMEN PLATE.

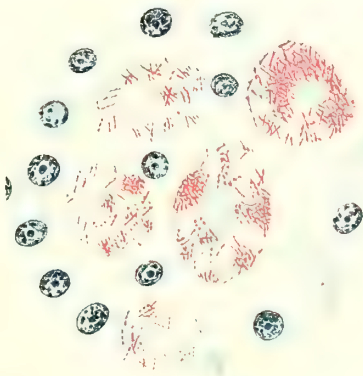


FIG. 1.—*B. lepre* in large phagocytic cells in sinuses of spleen ($\times 1000$ diam.). Carbol fuchsin and meth. blue.

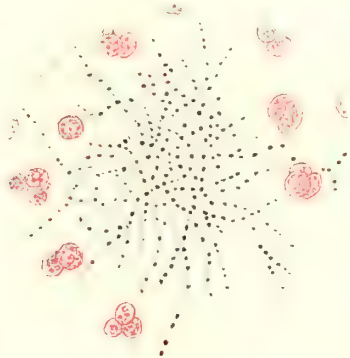


FIG. 3.—Pus from case of *Actinomycosis*, showing filaments of *Actinomyces* ($\times 1000$ diam.). Gram's method and safran.

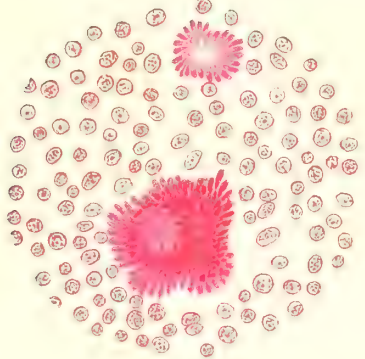


FIG. 4.—*Gonorrheal* pus, gonococci in leucocytes ($\times 1000$ diam.). Formalin, eosin, and meth. blue.

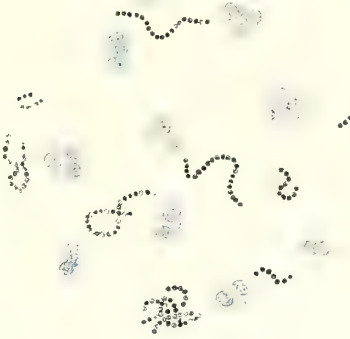


FIG. 5.—*Tubercle bacilli* in phthisical sputum ($\times 1000$ diam.). Ziehl-Neelsen contrast stain.



FIG. 6.—*Actinomyces*. Tongue of cow ($\times 500$ diam.). Acid fuchsin and methylene blue.



FIG. 8.—*Spirilla of Relapsing Fever*, blood film ($\times 1000$ diam.). Formalin, eosin, and meth. blue.

FIG. 7.—*Bryskipelas*, *Streptococcus Pyogenes* in pus, *Bingyema* ($\times 1000$ diam.). Eosin and meth. blue.

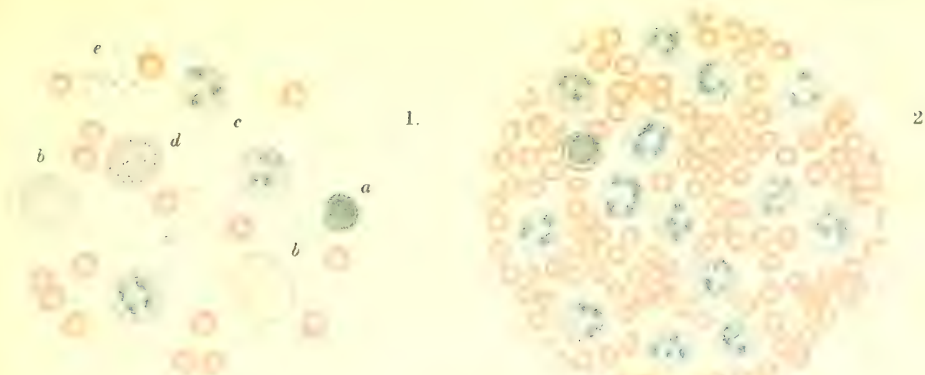


Fig. 1. Histological elements of normal blood:—*a*, lymphocyte; *b*, hyaline leucocytes; *c*, polymorpho-nuclear neutrophile leucocytes; *d*, eosinophile leucocyte; *e*, blood-plates.

Fig. 2. Blood from a case of extreme leucocytosis, showing great excess of neutrophile leucocytes.

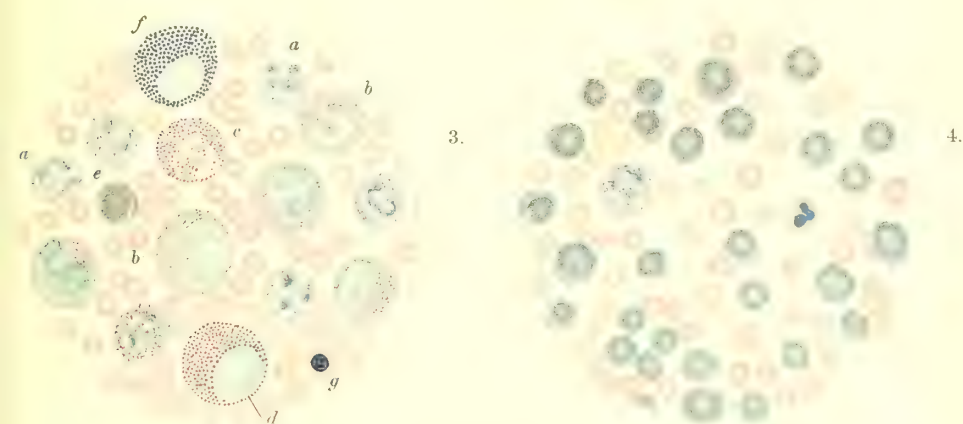


Fig. 3. Blood in medullary leucocythæmia:—*a*, neutrophile leucocytes; *b*, neutrophile myelocytes; *c*, eosinophile leucocyte; *d*, eosinophile myelocyte; *e*, lymphocyte; *f*, leucocyte with "mast-cell" granules; *g*, nucleated red corpuscle.

Fig. 4. Blood in lymphatic leucocythæmia, showing great excess of lymphocytes, most of which are of small size.

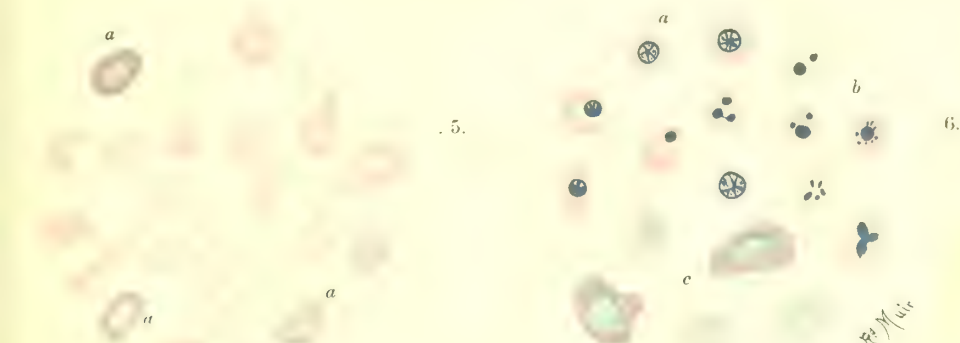


Fig. 5. Poikilocytes, megulocytes, and microcytes, from a case of pernicious anæmia:—*a*, red corpuscles showing polychromatophile degeneration.

Fig. 6. Varieties of nucleated red corpuscles:—*a*, normoblasts; *b*, normoblasts with fragmented nuclei; *c*, megablasts, two of which show polychromatophile degeneration.

film-preparations stained with eosin and methylene blue. Figs. 1—4 $\times 500$. Figs. 5 and 6 $\times 750$.

ment of the general condition has however quite a different significance, and it may be stated that absence of leucocytosis or disappearance of the leucocytosis without general improvement is an unfavourable sign in the conditions mentioned. This fact is of prognostic value in pneumonia, diphtheria, and in some cases of septicæmia. Towards the end of the leucocytosis, the hyaline leucocytes may show increase; and after its subsidence the eosinophile leucocytes reappear in the blood, and their percentage may even rise above normal. (b) Under the heading *toxic leucocytosis* may be included a number of conditions in which there is no evidence of infection, e.g. malignant jaundice, lead-poisoning, chronic Bright's disease. Here the leucocytosis has the same characters, but is usually less marked than in the previous group. (c) *Post-hæmorrhagic leucocytosis*. After a large hæmorrhage leucocytosis is usually well marked within three or four hours, that is, during the period when the fluid part of the blood is being restored. If the hæmorrhage is not repeated, the leucocytosis diminishes, and may be absent during the time of blood-regeneration. When marked anæmia with cachexia results from repeated hæmorrhages, there is usually a well-marked leucocytosis of chronic nature. (d) *Cachectic leucocytosis*. This is probably an instance of chronic toxic leucocytosis. It is well seen in the cachectic stage of malignant disease.

2. *Eosinophile leucocytosis or eosinophilia*.—The diseases in which this condition is present form quite a distinct class from those in which ordinary leucocytosis occurs. The following are the most important of them: (a) In certain acute and chronic diseases of the skin, pemphigus, psoriasis, urticaria, &c. It is also of interest to note that eosinophiles are also numerous in the local tissue lesions in these diseases. (b) In *asthma*, more especially during the paroxysms, the proportion of eosinophiles may reach 10–20 per cent.; eosinophiles are also abundant in the bronchial secretion. (c) In infections with *parasitic worms*, especially round worms, e.g. *Trichina spiralis*, *Ankylostoma*, and others, the number of eosinophiles may run as high as 50 per cent. of the total leucocytes. (d) In some cases of *malignant growths in the bone-marrow*. The most important condition under which the proportion of eosinophiles is less than normal is an acute neutrophile leucocytosis, when, as already mentioned, they may practically disappear from the circulating blood.

3. *Lymphocytosis*.—An actual increase of the lymphocytes (apart from lymphocythæmia) is comparatively rare. It has been found in certain malignant growths in the lymphoid tissues, in some cases of chronic gastric and intestinal catarrh; and it is sometimes present in whooping cough. As a rule it occurs more readily in children than in adults. A relative lymphocytosis will be present in those conditions in which the number of neutrophile leucocytes falls, as will be explained below.

4. *Increase of hyaline leucocytes*.—These cells have been less completely studied as regards their variations than the other classes. An increase is, however, common towards the end of an ordinary leucocytosis, e.g. in acute pneumonia about the time of the crisis. Their number is often increased in malarial fever, especially when of some duration, and an increase has also been noted in cases of gout.

LEUCOPENIA.—(a) In a certain number of *acute infective conditions*, the proportion of leucocytes remains constant or is diminished. Leucocytosis is usually absent in typhoid fever, in measles, in acute tuberculosis without cavity-formation, and in most cases of influenza. In typhoid fever there is usually marked leucopenia, especially during the late stages, when a number of 3,000 is not uncommon. Again, in various affections, where leucocytosis is the rule, leucopenia may supervene, the condition then becoming one of great gravity, e.g. in pneumonia and septicæmia. Conversely, in the diseases just mentioned, leucocytosis may appear if some suppurative or inflammatory conditions become superadded as a complication. (b) *Leucopenia occurs in a number of chronic diseases*. In pernicious anæmia, in chlorosis, in purpura, in hæmophilia, and in some cases of splenic anæmia, the number is almost invariably below the normal. In pernicious anæmia, for example, the number is often about 1,000 per c.mm., though some cases run their course with a considerably higher number. Here again the diminution is chiefly on the part of the neutrophile leucocytes, although in extreme cases all varieties may be affected. The condition of the leucocytes in these anæmic conditions presents a contrast to that in the secondary anæmias, where leucocytosis is the rule.

PRESENCE OF ABNORMAL ELEMENTS. (a) *Myelocytes*.—These are large cells measuring up to 15μ in diameter, and normally present only in the bone-marrow. They have a large oval, or slightly indented, nucleus, which stains comparatively faintly. The protoplasm contains fine granules, giving a neutrophile reaction, like those in the finely granular leucocytes. As will be shown below, they occur in large numbers in the medullary forms of leucocythæmia. Small numbers may, however, appear in the blood in certain other diseases. They have been observed, for example, in pernicious anæmia and in some cases of secondary anæmia due to malignant disease, and they also appear towards the termination of severe infective conditions, usually when the number of leucocytes is small. In the last group of diseases their appearance has on the whole a grave significance. Very often a few nucleated red blood-corpuscles can be detected along with them.

(b) *Cells with mast-cell granules*.—Although these have been noticed in normal blood, their presence must be considered as exceptional. In leucocythæmia these cells may be numerous, and a considerable proportion may be present in certain wasting diseases.

Significance and nature of leucocytic variations.—Only the most important facts can be enumerated. The non-granular and granular cells are affected by different conditions and must be considered as physiologically distinct. The granular cells multiply by mitotic division only in the bone-marrow, the non-granular cells in the lymphoid tissue throughout the body. The granular cells (neutrophile and eosinophile) are guided in their variations by chemotaxis, the two classes being differently affected by different substances, i.e. the neutrophiles are attracted by substances which may have no effect on the eosinophiles, and *vice versa*. The local migration into the tissue is brought about by chemiotactic substances, and when such substances are absorbed in large quantities into the blood, they exert a corresponding action on the

increased, but when there have been many hæmorrhages, with marked anæmia as a result, coagulation occurs more slowly than normal. In scurvy and many purpuric conditions, the coagulation-time is lengthened, though the amount of fibrin is usually increased (Hayem). In hæmophilia coagulation is considerably delayed, the clot is imperfectly formed, and fibrin is said to be decreased, though on the last point more definite information is desirable. In urticaria and in many other conditions with serous exudation, coagulation takes place more slowly than normal (Wright).

(b) *Presence of free hæmoglobin, &c.*—The term hæmoglobinæmia is applied when there is free hæmoglobin in the plasma. Its presence may be shown by centrifugalising the blood or by allowing it to stand and obtaining the serum in the usual way, the serum being of course tinted. This condition reaches its highest degree in paroxysmal hæmoglobinuria, the free hæmoglobin being rapidly excreted by the kidneys. Hæmoglobin has also been found free in the serum in a number of severe infective or toxic conditions, e.g. icterus gravis, malignant scarlet fever, typhus fever, snake-poisoning. This may be looked upon as an example, in more severe form, of the tendency which the hæmoglobin has to diffuse out of the red blood-corpuscles after death in many infective diseases. In poisoning with a number of substances, not only is the hæmoglobin set free in the circulation, but is also converted into methæmoglobin, the condition being called *methæmoglobinæmia*. As examples of such poisons may be mentioned chlorate of potassium, many nitrites (nitro-benzol, nitrite of amyl), pyrogallol acid, ferrocyanides, &c. In such conditions fragments of red blood-corpuscles or their decolourised stroma may also sometimes be detected microscopically in the blood. In poisoning by carbonic oxide the blood has a light red colour owing to the combination of this gas with the hæmoglobin. In sulphonal-poisoning, hæmatoporphyrin is present in the urine and has also been detected in the blood in some cases. See SPECTROSCOPE IN MEDICINE.

(c) *Increase of Urea &c.*—In fevers and other conditions in which there is an increased production of urea and other nitrogenous extractives, no marked increase of these substances occurs in the blood owing to their rapid excretion. Such increase may however be met with in Bright's disease, especially in the catarrhal form with marked albuminuria and deficiency of urine, and to a still greater degree when there is uræmia. In health the amount of urea in the blood is about '015 per cent., while in uræmia it may rise to more than twenty times this proportion. Urea in itself is relatively non-toxic, but the fact just stated is of importance as indicating the probable retention in the blood of some other toxic substance. Other nitrogenous extractives are increased along with the urea, both in the blood and in the dropsical exudations, but it has so far been found impossible to reproduce the characteristic symptoms of uræmia by the injection of any of these substances. The hypothesis that uræmia is due to the presence of ammonium carbonate in the blood has not been substantiated by direct observations. In puerperal eclampsia, it may be mentioned, there has been found to be only a slight increase of urea. See URÆMIA.

Uric acid (urates) in the blood.—In normal conditions the amount of uric acid (in combination) is so

small that it cannot be detected by chemical means. In gout there is relatively a considerable quantity; Garrod found '00175–'0025, per cent., and though estimations by improved methods have given rather lower figures, the general result has been confirmed. The presence of uric acid can usually be demonstrated by the 'thread-method' (see GOUT). The presence of uric acid is, however, not peculiar to gout, as it has been observed in other diseases, e.g. in cases of severe anæmia, in pneumonia, in cardiac disease with dyspnoea, and in nephritis. In leucocythæmia it has been found by some observers, while others have obtained negative results; the presence of hypoxanthin and other members of the xanthin series in the blood, and increase of uric acid in the urine, are, however, well-established facts, and usually ascribed to the increased disintegration of nuclein contained in the leucocytes. In the other conditions mentioned, diminished oxidation and interference with excretion may also be variously concerned in the presence of uric acid in the blood.

(a) *Increase of Fat—Liphæmia.*—The amount of fat in normal conditions becomes more abundant after meals; it is also increased in a variety of diseased conditions. In diabetes it was found many years ago that on post-mortem examination in some cases the fat was so abundant that it formed a distinct milky scum on the surface, and the name 'liphæmia' was applied to the condition. Later observations have shown that in a certain proportion of cases of this disease liphæmia can be detected during life, though often in very slight degree; in some cases of diabetic coma it has been found well marked. Its presence can be shown by filling a fine glass tube with blood and examining microscopically the superficial layer after the blood has been allowed to stand for some time. An oil-immersion lens should be used, as the droplets are sometimes very minute. It can also be demonstrated by staining film-preparations with osmic acid. The exact circumstances determining the presence of liphæmia and regulating its degree in diabetes are unknown. Liphæmia has also been found to occur in cases of obesity, phthisis, alcoholism, some fevers, e.g. typhus, and in conditions of dyspnoea. Fatty acids, in combination as salts, have also been demonstrated in the blood in various fevers, in some cases of diabetes, in leucocythæmia, and in acute yellow atrophy of the liver, their presence being supposed to be due to defective oxidation. Fat may also enter the blood in comparatively large droplets in fractures of bones and give rise to capillary embolism. See EMBOLISM.

(e) *Increase of Sugar—Melithæmia.*—In normal conditions the amount of dextrose in the blood is about '015–'05 per cent., showing variations according to the food &c. In diabetes the amount is greatly increased, rising in severe cases to '5 per cent. Along with this the specific gravity of the serum is high (unless there is marked cachexia), and the alkalinity tends to be diminished, being markedly so in the condition of diabetic coma, owing to the formation of diacetic, oxybutyric, and other fatty acids. The view that acetone plays an important part in the stage of coma is now generally discarded. In some cases there is a marked increase of fat (*vide supra*). In poisoning with chloroform, morphine, curare, nitrites, carbonic oxide, &c. the amount of sugar in the blood is distinctly increased. On the

other hand, in 'phloridzin' diabetes, though a large quantity of sugar is excreted by the kidneys, the amount in the blood does not exceed the normal. The percentage of sugar has been found to be raised in some cases of cancer, sometimes to three times the normal, and to be diminished in nephritis (Freund and Trinkler).

(f) *Cholamia*.—This condition may be said to result from the absorption of constituents of the bile by means of the lymphatics, and is manifested by the presence of jaundice. Formerly a 'hæmatogenous' form of jaundice was believed to occur in certain diseases, but it now appears that in such cases there is increased destruction of red corpuscles, increased formation of bile-pigments in the liver, and thickening and stagnation of bile, with consequent absorption. In jaundice the serum has a bright yellow colour, as can be readily demonstrated, and even the tint of the blood may be appreciably altered. The bile-pigments are, however, relatively non-toxic, as considerable quantities may be injected into the blood of animals without producing serious effects. On the other hand, the salts of the bile-acids are much more important, as they have a markedly toxic action, and to their presence most of the symptoms occurring in jaundice, e.g. slowing of the pulse-rate, lassitude, somnolence, are in all probability due. In severe 'toxæmic jaundice,' however, many of the symptoms are due to the toxins which produce the jaundice. One point of interest is that in jaundice the red corpuscles have a greater resistance to dissolving agencies than in health, or in other words their isotonic point is lowered (*vide supra*)—a condition which von Limbeck explains as being due to the circumstance that the less resistant red corpuscles have been destroyed by the bile-acids. The presence of cholesterin has also been demonstrated in the blood in cases of jaundice, but it is doubtful what part it plays in the production of symptoms.

(g) *Melanæmia*.—This term, which is now less used than it was formerly, signifies the condition of the blood in malaria when it contains minute granules of black pigment which may be either free or contained within leucocytes. Similar particles may be seen in the interior of the *Hæmatozoon malariae*, and there is no doubt that the pigment is elaborated from hæmoglobin by the activity of this organism. See MALARIA.

Presence of bacteria, toxins, &c.—This subject will be treated of in connection with the individual bacterial infections, but we may here summarise a few of the chief facts. (1) Bacterial infections in which the organisms are so numerous in the circulating blood as to admit of their easy detection by means of the microscope are rare in the human subject, though they are not uncommon in certain of the lower animals. Relapsing fever is almost the only example in which the blood may be said to abound in organisms. (2) In a considerable number of diseases bacteria are present in the circulating blood, but only in comparatively small numbers. Sometimes they can be found on careful microscopical examination, e.g. in plague, influenza, anthrax, but usually only in very severe cases and often only shortly before death. Sometimes methods of cultivation are necessary in order to demonstrate their presence, and for this purpose a considerable quantity of blood removed from a vein and distributed on the surface of the culture-

medium gives the best results. By this method cultures can usually be obtained in severe pyogenic infections, ulcerative endocarditis, puerperal septicæmia, infections by the pneumococcus, &c. (see BLOOD, Examination of). (3) In a number of diseases special organisms pass by the blood-stream to various organs where they settle and multiply, but they do so in such small numbers that it is usually quite impossible to obtain them from the blood even by means of cultivation: this is the case in tuberculosis, glanders, actinomycosis, typhoid fever, and often in some of the diseases referred to in the preceding section. (4) In yet another class the organisms are practically confined to the local lesions in the tissues; if a few enter the blood-stream they are destroyed, and accordingly secondary or metastatic lesions do not occur. Cholera, tetanus, and diphtheria (at least as a rule) may be mentioned as examples of this group. In connection with the examination of the blood for bacteria and the interpretation of results obtained, it should always be borne in mind that towards the end of life, especially when death occurs slowly, the normal bacterium-free condition of the blood gradually becomes lost. In this way various bacilli and micrococci may be present in the various organs owing to an *ante-mortem* dissemination by the blood-stream.

With regard to the presence of toxins in the blood, it need only be remarked here that we have no physical or chemical means of detecting them (see TOXINS). Reference may, however, be made to various antagonistic substances (*Antikörper*) which may appear in the blood during an attack of certain diseases, as well as be developed experimentally by the injection of the corresponding bacterium or toxin. This fact is of prime importance in connection with the defence of the organism and the cure of disease. Such substances may be possessed of various properties. (a) They may be *antitoxic*, as in the case of diphtheria, in which disease it has been shown that the serum of a patient who has recovered has the property of neutralising a certain amount of diphtheria-toxin. (b) They may have a *protective* property (without being antitoxic), as shown by injecting some of the serum with the corresponding organism into an animal. In some instances, e.g. in the case of typhoid fever and cholera, this is associated with a *lysogenic* or *bacteriolytic* action, i.e. one which aids in the solution and destruction of the bacteria. (c) They may be *agglutinative*, i.e. produce clumping of the corresponding bacterium. This property—observed in the case of typhoid, cholera, Malta fever, plague, and many other infections—often appears early in the disease and constitutes the method of serum-diagnosis (see SERUM-DIAGNOSIS); it is probably closely related to the protective property, though it is not always manifested by a serum which has protective powers (see ANTITOXINS). All these substances are within certain limits specific, and are probably highly complicated in constitution, but possessing a specific chemical affinity for the substance introduced, e.g. bacterial protoplasm or toxin, as the case may be. It has been recently shown that other antagonistic sera can be produced by the injection of various organic substances; for example, by the injection of red corpuscles, leucocytes, or spermatozoa an anti-serum may be obtained which has the power of dissolving these respective bodies.

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cases, attention being drawn to the distinguishing features in any one of them. The following may be the starting points of the disease. (1) The periosteum and superficial bony layers (acute infective periostitis or osteoperiostitis). (2) The medullary cavity and inner layers (acute infective osteomyelitis or endostitis). (3) The epiphyseal lines beginning on the diaphyseal side (acute epiphysitis).

From mere continuity of tissue other parts of the bone than the point of origin may be involved. This is particularly seen in an osteomyelitis where from the medulla both epiphyseal lines may be invaded, and the whole length of the periosteum separated, causing total necrosis of the diaphysis. Again the continuity of the periosteum with the epiphyseal lines and the joint-capsule explains the frequent extension from such a starting-point to these structures. The actual point of origin may be difficult to determine in the early stage, but later may be inferred from the extent of the subsequent necrosis.

PATHOLOGY.—Rapid exudation, soon becoming purulent, takes place into the deeper layers of the periosteum, separating it from the bone, or into the medullary cavity. In either case the blood-supply to the bone is interfered with, causing necrosis, which is likely to be more extensive where the origin is osteomyelitic. The separated periosteum forms a new shell of bone, but in it certain apertures, 'cloacæ,' are usually left due to the escaping pus; through these, later on, the sequestrum may extrude. When the disease is central, septic thrombosis is very liable to occur in the veins of the bone, and emboli, being discharged into the circulation, set up pyæmia. Usually the disease is arrested at the epiphyseal junction, but in other cases extension may take place into the joint and a septic arthritis be established: this occurs particularly in the hip-joint and is due to the mutual relations of epiphysis and joint. Within the past few years it has been established by Becker, Krause, Rosenbach, and others that this disease is due to a micrococcus identical with the *Staphylococcus pyogenes aureus*. The micrococci from osteomyelitis have been injected subcutaneously and have produced local abscesses; and, on the other hand, staphylococci from abscesses &c., when injected into the circulation, produce the same results as osteomyelitis. As in other abscesses, more than one kind of organism may be present, and Fraenkel has recorded cases due to the pneumococcus.

SYMPTOMS.—One of the earliest symptoms of acute periostitis is sudden and severe pain in the affected bone, which is often attended by shivering or a rigor and intense fever. The temperature mounts quickly to 104° or 105° F. There is sweating, rapid feeble pulse, and delirium, aggravated at night. On the second or third day deep-seated swelling may be felt, somewhat indefinite at first; and the limb is swollen, hot, tense, and tender. An obscure sense of fluctuation may be distinguished at an early period; but when fluctuation becomes distinct very extensive damage will usually have taken place. After an interval varying from three to five days, the inflammation approaches the surface, the skin becomes oedematous, pits on pressure, and finally reddens and inflames. The length of interval depends on the thickness of muscles and soft parts covering the affected bone. Other things being alike in respect of pain and

amount of fever, the longer the delay in the appearance of external swelling, the greater the probability that the medulla with the adjacent bone is the first and chief tissue engaged, the inflammation having reached the periosteum secondarily; while the early appearance of swelling and fluctuation externally suggests that the inflammation is chiefly periosteal. Septicæmia or pyæmia may supervene.

DIAGNOSIS.—This disease may be obscure at the commencement, and its nature overlooked. It has often been mistaken for acute rheumatism, on account of the swollen joints; for phlegmonous erysipelas; for acute cellulitis, or for typhoid fever. The only malady which acute periostitis really resembles is an idiopathic inflammation of the deep-seated cellular tissue in a limb. This disease is rare. When we observe the chain of symptoms above described in a young person, we may safely assume the presence of an acute osteoperiostitis.

COURSE AND TERMINATIONS.—The disease almost invariably terminates in suppuration and necrosis; resolution happens rarely, but necrosis is not inevitable, even after suppuration. In a few cases, especially in young children, if the matter be speedily evacuated, the abscess collapses, the periosteum reunites with the bone, and no necrosis takes place. This result is unfortunately quite exceptional. Resolution without suppuration may be the case where more than one bone has been involved, the pain and tenderness in those later attacked being succeeded only by thickening.

PROGNOSIS.—This must be founded on the extent of the necrosis; whether blood-poisoning has taken place; and whether the adjacent joints are implicated in the disease. Cure cannot take place until the dead bone is cast off or removed, and this is often long delayed. The usefulness of a limb may be permanently impaired by the disease, or it may require amputation; or the patient may lose his life from the exhaustion of the discharge or some intercurrent malady. On the other hand, the use of the limb, and the health of the patient, may become completely re-established.

TREATMENT.—Early and energetic treatment is of the greatest importance, as it affords the best prospect of averting the disastrous consequences of acute periostitis, but in hospital practice the cases are rarely seen sufficiently early. In the first stage the limb should be elevated, and cold compresses applied. As soon as the nature of the affection is suspected free incisions down to the bone, so as to divide the periosteum, are indicated, even before there is clear evidence that pus is formed. They relieve pain and tension; and, by permitting the timely escape of pus as soon as it is formed, the amount of periosteal separation, and consequently of necrosis, is limited. It is the more important to make an early incision, because evidence of fluctuation is at first by no means easy to make out; and an incision down to the bone should be made in the centre of the inflamed area in all cases of doubt. Antiseptic precautions should always be taken. Sometimes the abscess-cavity does not readily collapse owing to its walls being stiff and infiltrated, and its contents may become septic, thus greatly increasing the patient's risks. If there be synovial effusion into a neighbouring joint, it should be kept at rest by means of a splint or a fixed bandage, but on the slightest suspicion of the nature of the effusion it may be aspirated for diagnostic purposes. As the acute symptoms subside, the

abscess-cavity contracts, one or more sinuses remain, and the dead bone begins to separate. See NECROSIS, page 167.

Where an epiphysis is engaged in the disease the case is more urgent; the fever runs higher, the suppuration is greater, and the degree of joint-implication more intense, proceeding in extreme cases to suppurative inflammation and destruction of the articulation. In such involvement of the joint free incision and drainage will be needed. The affected limb should always be supported on a splint. (Edema of the limb often indicates a deep-seated phlebitis, the precursor of septic poisoning. Under these circumstances, amputation is often the only resource. It is imperative to amputate where there is extensive bone-destruction, and the symptoms indicate commencing pyæmia; or where, with the death of a large portion of the shaft, one or both of the neighbouring joints may have become gravely implicated, and great suffering and loss of strength forbid us to temporise. It is precisely in these cases, however, where the diaphysis has become necrosed up to the epiphyseal junction, that good results are attainable by the immediate extraction of the dead bone. The part of the shaft which joins the epiphysis becomes rapidly detached and loose, and may be readily separated and removed, while the portion beyond the limit of the necrosis in the other direction can be divided with a chain-saw. It is difficult, however, in the early stage to diagnose the extent of the necrosis. Where the joints both above and below are involved, amputation is usually necessary.)

(b) *Chronic periostitis*.—This disease is usually due to some diathetic cause, but may result from injury, or from some continuous pressure or source of irritation. It is most frequent on the superficial parts of the skeleton, as the tibia, clavicle, skull, and ribs, but may affect any bone; and it is often observed at the origin or insertion of muscles. When the disease arises from a general cause, such as syphilis, many parts of the skeleton are affected; when from a local one, usually only one.

SYMPTOMS.—Chronic periostitis often takes the form of what is called a *node*—a tender, more or less painful, rounded or oval swelling; at first tense and hard, afterwards softer or even fluctuating. The pain is more severe at the outset, from the tension of the parts involved, and is generally worse at night. Subsequently the swelling becomes indolent, and painless, unless it is pressed upon; and the effused matter may become organised first into fibrous tissue and then into bone. Nodes are due to a localised inflammation, forming a well-marked projection on the bone, which may undergo resolution, suppurate, or ossify, according to circumstances.

PROGNOSIS.—In chronic periostitis this is usually favourable. Under the influence of early and suitable treatment, the inflammatory products are completely absorbed, and the bone resumes its natural shape. If the chronic inflammation of the periosteum be permitted to proceed unchecked, a deposit of new osseous lamellæ usually takes place on the surface of the affected bone, giving rise to permanent thickenings, or even to osteophytic growths. These are composed of light porous bone, with a rough surface. The skeleton of a syphilitic subject will often present numerous thickenings of this nature. On making a section of the bone, it is easy to see that the new bone is superimposed upon the old, and is formed by the periosteum.

TREATMENT.—When due to a local cause, the swelling will often spontaneously subside with removal of the cause and rest to the part; but in obstinate cases iodide of potassium internally, and iodine-ointment or blistering externally, may be required. If the subject be unhealthy, or if the original injury be considerable, suppuration may take place, when the treatment will be that of an inflammatory abscess. *Syphilitic nodes*, which are a very common expression of chronic periostitis, yield rapidly to the influence of iodide of potassium; and this in some cases may usefully be combined with a mercurial course. Blistering or friction externally is hurtful in such cases. Syphilitic nodes are not at first prone to suppuration, and even when they become soft and fluctuating, and the skin reddens over them, they should not be mistaken for abscesses, as they do not require incision, and will readily be absorbed under suitable treatment.

Periostitis after fevers.—A peculiar form of periostitis is occasionally observed as a sequel of specific fevers, especially typhoid fever. It is due to a mild infection from some ulceration, such as Peyer's patches in typhoid fever or the throat in scarlet fever. In typhoid fever it occurs during convalescence, and without general symptoms. It takes the form of hot, painful, and tender nodes, frequently symmetrical, and often placed on the tibia; the disease is also found on the ribs and other bones. It may be associated with necrosis, but if so the extent of the dead bone is small in proportion to the inflamed area of periosteum. The general health is not seriously affected by the periostitis, and the disease is amenable to treatment by iodide of potassium, combined with iodide of iron.

(B) **Osteitis.**—Osteitis is an inflammation chiefly affecting the bone-substance. It may be either *acute* or *chronic*.

(a) *Acute osteitis* is neither clinically nor pathologically to be distinguished from acute osteomyelitis or endosteitis. See OSTEOMYELITIS, p. 166.

(b) *Chronic osteitis* is a disease beginning in the bone, in which from first to last the chief changes occur, the periosteum being secondarily engaged. This affection may result from injury, or be excited by exposure to cold; but it is often associated with syphilis, tuberculosis, gout, or rheumatism, the first being the most frequent cause. It may occur in any part of the skeleton; the chief changes, when produced by syphilis, occur in the shafts of the long bones. They consist mainly of hypertrophy, and the bone is ultimately increased in thickness, in length, and generally in density: its interior is often transformed into sclerosed bone-tissue, and the medullary cavity is obliterated. Another form, associated with the strumous diathesis, is generally seated in the joint-ends of the long bones, and in the spongy bones. It is prone to end in suppuration, accompanied by either caries or necrosis, or may terminate in the condition known as *osteoporosis* or *rarefying osteitis*. The gouty and rheumatic forms are associated with evidence of the presence of either of these diatheses.

PATHOLOGY.—Increased vascularity first takes place, the Haversian canals enlarge, the canaliculi disappear, the cancelli, containing the inflammatory products, enlarge, and the earthy matter diminishes. Hence the inflamed bone softens, and, if macerated at this stage, will be found comparatively light and porous. When the inflammation affects the superficial laminae of the bone, the peri-

osteum becomes thick and vascular; if the deeper parts are involved, similar changes will occur in the endosteum. The porous condition of the bone may become permanent, when the condition is called *osteoporosis*, the result of so-called rarefying osteitis; or the granulations become transformed into new bone, and the cancellous structure is filled with osseous deposit, so that the whole of the inflamed area becomes very dense, and is then said to be sclerosed; or the inflammatory process may terminate in suppuration, followed by caries or necrosis; or an abscess may form in the interior of the bone, and be either diffused or circumscribed.

SYMPTOMS.—These are insidious, very obscure at the outset, and may be mistaken for those of chronic rheumatism, or simple periostitis. They consist chiefly in aching, gnawing pain in the affected bone, with characteristic remissions and nocturnal exacerbations. The bone is tender on pressure, and feels increased in bulk at first, from the infiltration of the immediately surrounding soft tissues; subsequently the bone itself enlarges. There is often increase of heat in the limb. The progress is chronic. If unchecked by treatment, chronic osteitis may give rise to considerable deformity.

TREATMENT.—Treatment should be directed to the cause of the disease. If this be syphilis, antisyphilitic treatment will be followed by good results; even in chronic bone-inflammation not dependent on syphilis, iodide of potassium is often of great service. Local counter-irritation may also be employed. Often the cause cannot be made out, and if iodide of potassium fail in producing an effect we must fall back on general treatment. In the early subacute stage, rest, with elevation of the affected part, is very desirable. Warm fomentations, followed by iced compresses, relieve the suffering. If there be much pain and tension, leeches should be applied. Puncturing the tissues down to the inflamed bone, with a tenotomy-knife or fine bistoury, relieves the tense periosteum, and allows extravasation beneath it to escape, so that the pain promptly abates.

(C) **Osteomyelitis.**—This is an inflammation of the medullary cavity and the endosteum. Like osteitis it may be either acute or chronic.

(a) *Acute simple osteomyelitis* will occur in a varying degree in any injury to the medulla which remains aseptic, as in simple or compound fractures or after amputations.

Acute infective osteomyelitis has been referred to before under acute infective periostitis, and here the infection had been derived from the blood. Any open wound, whether due to a compound fracture or amputation, where the medullary cavity has been opened, may become contaminated with septic products, and an *acute septic osteomyelitis* develop. This condition is very liable to be associated with septic thrombosis and pyæmia. The appearance of the wound will change and there will be some swelling of soft parts and tenderness about the bone. The discharge alters and may be offensive. With this there will be some fever and probably a rigor. On opening up an amputation stump which has become septic the periosteum will be found separated from the bone, which is discoloured, and the medulla will have lost its normal vascularity. The prognosis in these cases is generally bad.

TREATMENT.—Directly such a complication is suspected the wound must be most thoroughly ex-

amined. If, after cleansing the wound well and getting rid of all tension, the medulla near the surface seems vascular, we may temporise, provided that the constitutional symptoms allow of it. But as this is rarely the case amputation is usually required. To get above the diseased medulla it is generally held that no operation is of any avail unless the whole of the diseased bone is removed. In dealing with the upper end of the femur the medulla has been scraped out and packed, and by some this method has been extended to other bones instead of resorting to a high amputation.

(b) *Chronic osteomyelitis* is an obscure affection not to be distinguished either clinically or pathologically from chronic osteitis. It may terminate in sclerosis, or in the formation of an abscess. See **CHRONIC OSTEITIS**, p. 165.

2. **Abscess.**—This is a term usually applied to a limited suppuration in the bone, unattended by any marked necrosis. Young adults are most prone to the disease, or boys about the age of puberty; it is very rare in women. It is the result of a chronic inflammation of bone, which may be associated with some injury or due to tuberculosis. This affection is most frequently met with in the upper and lower extremities of the tibia, just external to the epiphyseal cartilage, less frequently in the ends of the femur, only occasionally in other bones, and very seldom in the compact tissue anywhere.

SYMPTOMS.—A circumscribed, slightly elevated, very tender and painful swelling may be discovered on the surface of the bone. This is due to a local periostitis with new bone-deposit. In old-standing cases the bone is often half an inch or an inch longer than its fellow, by reason of the chronic hyperæmia and consequent increase of activity of growth at the epiphysis. The skin and superficial parts are unchanged at first, or there may be but trifling subcutaneous œdema. There is often slight local increase of temperature. The pain, aggravated on deep pressure at the central point, is often intolerable. It is intermittent at first, but generally worse at night. After a time it becomes continuous, and deprives the patient of all rest, owing to its severity. The abscess may persist with little change for months or years. The symptoms generally resemble those of osteitis, from which at the outset it is difficult to distinguish this affection. When the abscess approaches the periosteal surface, the soft parts become involved, and there will be slight redness and œdema of the skin. More rarely the pus makes its way into the adjacent articulation, in which it sets up destructive inflammation; but usually the joints are free from implication. The subjects of the disorder have often suffered from antecedent bone-disease. Evidence of this should be looked for, as giving a clue to the diagnosis.

TREATMENT.—Spontaneous cure cannot occur: even if the abscess discharge itself, a permanent fistula will usually remain. It is necessary to lay the abscess-cavity freely open. A crucial incision must be made through the soft parts, down to the bone, at the most tender and prominent point, and a disc of bone removed by the bone-trephine—an instrument without a shoulder, about half an inch in diameter. The sudden loss of resistance indicates the piercing of the abscess-cavity. The layer of granulation-tissue lining its interior should not be interfered with, but the cavity simply washed.

out. The pus is often foul, and greenish in colour. The wound should be dressed antiseptically; granulations presently fill it, which are subsequently transformed into a fibrous cicatrix. Immediate and permanent relief follows the operation. If the abscess is missed, the trephine may be re-applied, or drill-punctures made in the most likely directions in the adjacent bone, in order to discover the pus. Sometimes an error of diagnosis is committed, and the symptoms are found to arise from chronic osteitis, without suppuration. The operation, nevertheless, affords relief in these cases also. When there is doubt, a preliminary course of iodide of potassium will often remove it.

3. **Caries.**—The term caries does not express any definite disease, although so frequently used as a synonym of tuberculosis of bone, but only a pathological state which may be produced by several causes. It has been spoken of as ‘ulceration of bone’ or ‘molecular death.’ Its essential feature is a rarefying osteitis which involves usually the cancellous tissue, the bony framework gradually disappearing in front of the granulation-tissue.

Many forms of caries have been described. ‘Simple caries’ may be met with in its most typical form (1) in an acute septic arthritis, where, after the destruction of the cartilage, the ends of the bone become involved; or (2) on the surface of a bone where the soft parts are destroyed. ‘Caries fungosa,’ ‘caries necrotica,’ and ‘caries sicca’ are variations in the tubercular process, and will be referred to under that heading (*vide infra*).

4. **Necrosis.**—The complete arrest of nutrition in a portion of bone from any cause is followed by the death or necrosis of the bone, and by a series of inflammatory changes in the adjacent parts, which result in the complete separation of the dead from the living tissue. It is a common result of the acute inflammation of bone which is invariably connected with some septic or infective cause.

ÆTIOLOGY.—Necrosis is most frequently the result of acute bone-inflammation or severe injury, as after amputation, compound fracture, or contusion. It is especially prone to happen in the compact tissue, but it also occurs in the spongy structure, as the ends of the long bones, or the tarsus and carpus, where it is usually associated with more chronic forms of inflammation, and is more limited, as in necrotic caries. The peculiar nature of the blood-supply to bone, and the facility with which it may be interfered with or arrested under the pressure of inflammatory exudation, go far to explain the frequency of necrosis as a result of bone-inflammation. Acute suppurative osteoperiostitis or osteomyelitis rarely terminates without necrosis. Whether the dead bone will be in the superficial or the deep lamellæ, or include the whole thickness, depends on the seat of the inflammation, and on the extent to which the periosteum and endosteum are respectively implicated. The long-continued action of crude phosphorus, as observed in match-makers, and also of mercury, may induce necrosis. Syphilis is a frequent cause of necrosis, through its tendency to produce chronic osteoperiostitis, the sclerosed bone thus originated being afterwards prone to necrose. It is not an uncommon sequel during convalescence from some eruptive and continued fevers. After scarlatina, osteoperiostitis, followed by necrosis, is by no means rare, although affections of the joints are

more common. It is probable that many cases of necrosis occurring in childhood are connected with an antecedent attack of scarlet fever, e.g. necrosis of the petrous portion of the temporal bone as a consequence of otitis. Arterial thrombosis and embolism are occasional sequelæ of typhus, and may produce a local gangrene, not only of the soft parts, but of bone. This is, however, more frequent in connection with typhoid fever. In endocarditis the nutrient artery of a bone has been found obliterated by an embolus, thus producing necrosis.

PATHOLOGY.—After the death of a portion of bone, the living tissue, in immediate contact with the dead, becomes inflamed. The Haversian canals and canaliculi become distended with migratory cells; the leucocytes multiply, and by degrees consume the hard substance of the bone; the trabeculæ are absorbed or eaten away; loops of capillaries form from the pre-existing vessels: a granulating surface, in fact, is formed in the layer of living bone, surrounding or in contact with the dead, in a manner precisely similar to what takes place in the soft parts when a slough is being detached. The periosteum separates from the bone, and becomes thick and vascular, while the osseous surface beneath is smooth and white, like macerated bone. In cases of syphilitic necrosis, as well as in that resulting from phosphorus, the surface is rough from antecedent periosteal deposit, and in the former the interior may also be sclerosed. How the osseous trabeculæ are dissolved or disintegrated at the surface of separation, so as to loosen the dead bone, is not certain. Probably the granulation-tissue that forms from the living bone possesses phagocytic properties, and thus disposes of the calcareous particles. The pus that is formed has a mechanical influence, while according to one theory lactic acid is produced, which transforms the insoluble into soluble salts of lime. While this loosening process is going on, new bone, formed chiefly from the periosteum, which becomes thick and vascular, is being deposited, constantly becoming thicker, and with one or more openings in it for the escape of pus, called *cloaca*, so that eventually the dead portion becomes completely invaginated, and is named, from its position, a *sequestrum*. This sequestration of the dead bone is not invariable. For instance, in the spongy bones, the bones of the skull, and the upper jaw, or where from any cause the periosteum has been destroyed, no sheath of new bone will be formed. Necrosis very rarely takes place without suppuration; when this does happen the nature of the case is very obscure. It has been aptly called ‘quiet’ necrosis. Occasionally nearly the whole shaft of a long bone has been found necrosed, and after an interval of months, or even years, no suppuration may have taken place. Such forms of necrosis are usually central and limited in extent. They may very closely simulate malignant disease, and often cannot be relieved or even recognised save after amputation. A chronic osteitis, followed by hypertrophy and sclerosis of the bone, is the most common antecedent condition of this form of necrosis.

TREATMENT.—The changes already described, which separate the dead bone from the living, do not cause its expulsion from the body. On the contrary, they shut it up, like a kernel within its shell, and nothing so imperatively demands surgical interference as the presence of necrosed bone.

acts as a foreign body, is a constant source of risk to the patient, and should be removed as soon as practicable. Its continued presence excites the periosteum to further formation of bone, so that the invaginating sheath becomes of great thickness in old-standing cases. The period at which an operation is usually undertaken is when the sequestrum has become loose, and the time required for this purpose varies with the extent of the necrosis. In the actively growing bones of the young, especially when the sequestrum involves the epiphyseal junction, the process of separation is accomplished more quickly than in the adult. Roughly estimated, a period of from three to six months might be named as that within which loosening of the sequestrum usually occurs. Beyond the latter term an effort to extract the dead bone should not be delayed, even if it cannot be felt to be loose. Among other risks involved by delay may be that of amyloid degeneration of the viscera, principally the liver, kidneys, and spleen, which are subject to this change as the consequence of long-continued discharge from bone-disease. In order to remove a sequestrum, a director should first be introduced through a cloaca as a guide, and the soft parts sufficiently divided. An adequately large opening must now be made in the encasing sheath of new bone with the chisel, trephine, small saw, or cutting-forceps, and the dead bone extracted, either in one or several pieces, as may be the more convenient. The operation may prove difficult on account of great thickness of the soft parts, or of the sequestral envelope, or because the sequestrum itself is extensive. After the removal of the dead bone the cavity fills with granulations, which subsequently ossify, and the soft parts cicatrise. When the loss of bone-substance is very large the reparative process has been much facilitated by the implantation of small pieces of bone from a freshly killed rabbit. Finally the sequestral envelope of new bone is partly absorbed, partly consolidated, just as the redundant callus is after fracture, and the bone tends more or less to resume its normal size and shape.

5. Diseases of Infective Origin (*granulomatosa*).—(a) Tuberculosis.—A very large majority of the cases of chronic bone-disease are the result of tubercular infection. This form of local tuberculosis is most frequent in young subjects, but it may arise after middle life and form one of the lesions designated as 'senile tuberculosis.' The parts usually involved are (1) the cancellous tissue of the small bones of the carpus and tarsus, (2) that in close relation with the epiphyseal cartilages at the end of the long bones, or (3) that of the vertebrae. The small long bones of the hand and foot are frequent sites, but here the lesion is mostly an osteomyelitis. Involvement of the shaft of the long bones, although it occurs, is rare.

PATHOLOGY.—The disease of the bone is a chronic inflammation causing a rarefying osteitis or 'caries,' but occurring secondarily to the formation and degeneration of 'tubercles.' This invasion of the healthy bone by the granulation-tissue is progressive. True pus is not present unless there is an additional infection from pyogenic bacteria. Certain clinical terms are associated with the starting-point of the disease and with the pathological changes. The frequent origin beneath the articular cartilage of a joint accounts for the term 'subarticular caries.' The usual sequel here is absorption and perforation of this cartilage with involvement of the joint-cavity,

the granulations frequently being exuberant and sprouting (*caries fungosa*). During the invasion of the bone by the granulation-tissue certain areas of rarefied bone may be isolated before they have become completely absorbed. Should degenerative changes now occur in these granulation-barriers, these portions of bone are cut off from their blood-supply and appear as small sequestra (*caries necrotica*). If the products of degeneration are occluded in the surrounding bone a *chronic abscess* of bone is formed. A more favourable course may be run when the bone is progressively absorbed without any such degenerative changes in the granulation-tissue, the latter becoming organised and the neighbouring bone often sclerosed (*caries sicca*), a process most frequently met with at the upper end of the humerus. Miliary tuberculosis may be met with, particularly in the medulla, but it only occurs as a part of a general tuberculosis, has no clinical significance, and is very rare.

SYMPTOMS.—Of the general process there are no special symptoms beyond those related to the part involved. The condition of health varies considerably, but a history of any hereditary tendency to tuberculosis aids in making a diagnosis. Pain and tenderness with restriction of normal range of movement may first attract attention; later on this is followed by swelling. Often a chronic abscess or some other complication may be the first sign. As the softened products make their way to the surface the covering tissues become gradually thinned, till at last the skin gives way. In this way a *sinus* is formed leading down to the diseased area, and lined by weak granulations discharging a thin watery fluid; the skin forming the margin of the opening is often irregular and undermined.

TREATMENT.—In cases of this kind general tonic treatment becomes of the greatest importance. Rest must be given to the affected part, and exercise to the body generally, combined with fresh air both day and night, and simple nourishing food. An operation becomes necessary when softening has occurred, the degenerated products being removed by incision and scraping. When the bone is extensively diseased, it must either be excised, or the part amputated. The presence of the tubercular diathesis does not forbid an operation, the local source of irritation and drain upon the system being thus removed, and a healthy traumatic surface substituted for one infiltrated with inflammatory products. The removal of the local disorder often proves a comfort to the patient; increases his chance of regaining health and strength; and diminishes the liability to dissemination.

(b) **Syphilis.**—In both the acquired and inherited disease lesions are met with which are practically of the same nature. In *acquired syphilis*, during the secondary period, a local periostitis, forming a node, is most frequent. There may be symmetry in the lesions, or more rarely many nodes may be studded over several bones. The local changes are, as a rule, very limited and, under the use of appropriate treatment, speedily disappear, leaving little or no trace behind. It is a different matter, however, with the lesions in the late stage. Here the changes are much more marked and the inflammatory process may involve periosteum, bone, or medullary cavity, and be limited or diffuse. The production of new bone is a feature which causes great thickening and irregularity of the surface, and in the bone itself reduction in size of the Haversian canals, as

well as increased density. The osteo-sclerosis may be so intense as to cause necrosis, and the sequestrum so formed, as a rule, separates very tardily. Where the changes are limited a gumma is formed, which may soften or resolve.

In *Inherited Syphilis* the commonest early change is inflammation about the epiphysis associated with fluid in the neighbouring joint, and a varying amount of periostitis up the diaphysis. Later on much the same changes are found as in the acquired disease, but the lesions are frequently symmetrical. The treatment indicated is that for syphilis in general, but whether the bone-lesions occur in the early or later stages, the nocturnal pain, which is distressing and constant, may be relieved by iodide of potassium.

(c) **Actinomycosis.**—This disease arises from the transmission to man of a vegetable parasite found usually in the bovine race. Its development in bone mainly occurs in the jaws (generally the lower), in which it causes great swelling. The jaw is distended by a mass of material chiefly inflammatory, consisting of a fibrous basis which is riddled with tracts occupied by the fungus. When suppuration occurs, the abscess-cavity presents the appearance of a mesh-work. See ACTINOMYCOSIS.

6. **Hypertrophy.**—True hypertrophy means an increased size of the bone, which still preserves its normal shape and structure. Apart from inflammation hypertrophy is rare. The conditions may be met with in association with increased muscular development or in a congenitally hypertrophied limb. Hypertrophy of the fibula has been observed, owing to increased weight thrown upon it, in cases of ununited fracture of the tibia.

7. **Atrophy.**—This is met with as a constant condition in old age, leading to enlargement of the medullary cavity at the expense of the cortex. Disuse and abnormal pressure are also frequent causes. The thinning of the bone gives rise to lessened resistance, so that fractures may occur spontaneously, or from very slight causes.

8. **Disorders of normal ossification.**—In the first place the arrangement of the skeleton may be followed, but yet be of altered proportions, as in cases of true dwarfism or gigantism; the former condition must be differentiated from that due to rickets or achondroplasia, the latter from acromegaly. Departures from the normal type are met with in suppression or addition of parts, generally of congenital origin. Various deformities occur from fractures, both intra-uterine and subsequent to birth.

The following two diseases exhibit metaplastic changes in the skeleton of either generalised or limited distribution; they are not dependent on any infective process and are generally associated with deformity.

(a) *Rickets.*—See RICKETS.

(b) *Achondroplasia or Chondrodystrophia fetalis.*—See ACHONDROPLASIA.

9. **Diseases associated with degenerative changes in normally formed bone.**—The pathological factor here is metaplasia of the normal bone-structure into another form of connective tissue, and is well illustrated in osteomalacia and osteitis deformans. Both of these diseases are non-inflammatory, and in them the architecture of the bone is greatly altered; the bones are softened and bend, causing various deformities. In osteomalacia there is a disappearance of the normal structure, and its place is taken by similar material to that occupying

the central canal, the change being usually eccentric, whereas in osteitis deformans the medullary tissue and normal bone are replaced by a spongy form of bone. Cases showing transitions between these extremes have been described, and in young subjects the victims of fragilitas ossium, apart from rickets, there is some such bony change. Another point to note is that in both osteomalacia and osteitis deformans the metaplasia may be accompanied by tumour-formation.

(a) *Osteomalacia or Mollities ossium.*—See MOLLITIES OSSIIUM.

(b) *Osteitis Deformans.*—This affection is one chiefly of elderly men. In a few cases only a single bone may become thickened and bent, but when many bones are involved a very characteristic clinical picture is produced. The head is enlarged and, owing to the general curvature of the spine, projects forward almost on to the sternum. The legs are bowed, giving the skeleton a simian type. The involvement of individual bones of the limbs varies, but there is a rough symmetry to be noticed. The progress of the disease is very slow, death generally resulting from some chest-affection, which is very prone to occur from the fixation of the ribs.

The bones on examination show great thickening with replacement of the compact tissue by porous bone, which also fills up the medullary cavity. There is no special treatment to be advocated for this slowly progressive disease.

(c) *Acromegaly.*—See ACROMEGALY.

(d) *Leontiasis ossea.*—The bones of the face and cranial vault are the sites of bony masses which get progressively larger, lead to marked deformity, and involve nerves, particularly the optic nerves, causing blindness. This hyperostosis implicates the entire bone, and more or less symmetry will be noted.

(e) *Anakhre.*—See ANAKHRE.

10. **Tumours of Bone.**—These are mainly of the connective-tissue type, either embryonic or of a more highly developed character. Primary epithelial growth of bone has been described, but it is a pathological curiosity and may be looked on with some suspicion; secondary epithelial tumours are not infrequent, associated especially with carcinoma of the mamma. Osteomata, chondromata, fibromata, and sarcomata, may occur primarily in bone. See TUMOURS.

Sarcomata.—These may be divided into those of central and periosteal origin.

(i) *Central.*—It is a debatable point whether the so-called *myeloid sarcomata* or *myelomata* should find a place under Sarcomata. These giant-celled tumours occur in the jaws and at the ends of the long bones, especially the tibia, lower end of femur, and radius. They are soft and absorb the bone, so that after a while there is only a bony shell produced by the expanded periosteum, which gives the egg-shell crackling. Cystic changes frequently occur, and sometimes they pulsate. They are considered to be non-malignant, and on these grounds local removal is recommended as far as it can be done to leave a useful limb; if recurrence occur, it is from admixture of other sarcomatous elements.

The central sarcomata may be spindle-celled or round-celled, especially the latter. Clinically, they are rounded and may give 'egg-shell' crackling; spontaneous fracture frequently occurs. Pain may precede any swelling. These soft tumours are very vascular and often pulsate: the ill-supported

vessel-walls frequently give way, causing hæmorrhage into the tumour.

(ii) The *periosteal* sarcomata may be spindle-celled or round-celled, more frequently the former. The growth on the shaft of a long bone shelves down to the shaft, giving it a fusiform shape; it is often irregular on its surface, and hard from the amount of ossification which occurs. Pain is not so marked, and spontaneous fracture is not so likely to occur, as in central growths: unfortunately, however, their standard of malignancy seems higher and they tend to recur locally. The treatment of these true sarcomata is to amputate well above the disease as soon as possible.

Osteo-aneurysm is a term that has been applied to certain pulsating bone-tumours which are usually central sarcomata. Berger has recently suggested that many of these cases are endotheliomata of bone which pulsate, are very malignant, and are very prone to be rapidly generalised. The secondary growths all show the same characteristic pulsation.

Cysts.—Hydatids are rarely met with. The commonest cystic bone-tumours are on the jaws either from retained teeth, *dentigerous cysts* or the *dental cyst* met with in association with decayed stumps.

WILLIAM MACCORMAC.

HENRY BETHAM ROBINSON.

BORBORYGMI (βορβορύγμ, I rumble).—Rumbling sounds produced in the abdomen by the movements of gas within the bowels or stomach. See FLATULENCE.

BORDIGHERA, on the Italian Riviera.—A suitable winter residence for patients suffering from some forms of renal and of chest-disease. The climate is warm and dry. See CLIMATE, Treatment of Disease by.

BORMIO, in Italy.—Thermal waters. See MINERAL WATERS.

BOTHRIOCEPHALUS (βόθριον, a pit; and κεφαλή, the head).—A genus of cestode entozoa, characterised by the possession of two pits, or depressions, one on each side of the head, in place of the four sucking-discs usually present in tapeworms. See ENTOZOA.

BOTULISM (*L. botulus*, sausage).—A term at first applied generally to sausage-poisoning, but now generally limited to a set of symptoms, including dryness of the mouth, difficulty in swallowing, dilatation of the pupils, and ptosis, due to the action of the toxins produced by the growth of the *Bacillus botulinus* in meat.

BOURBONNE-LES-BAINS, in France.—Common salt waters. See MINERAL WATERS.

BOURBOULE, LA, in France.—Thermal alkaline and arsenical waters. See MINERAL WATERS.

BOURNEMOUTH, in Hampshire.—A suitable winter residence for patients suffering from certain forms of chest-disease. The climate is mild and slightly humid. See CLIMATE, Treatment of Disease by.

BOWELS, Diseases of.—See INTESTINES.

BRACHIAL PLEXUS, Lesions of.—Paralysis due to lesions of the brachial plexus differs according

to whether the whole plexus is damaged or only some of the roots of which it is composed. When the whole plexus is damaged, as from violent traction on the arm or after reduction of a dislocated shoulder, there is paralysis of all the muscles of the upper limb with complete anaesthesia up to the shoulder.

When only certain of the spinal roots which form the plexus are injured, the resulting paralysis is that of the muscles which receive their nerve-supply from the particular root or roots affected. The most common association of muscles involved is that of the deltoid, biceps, brachialis anticus, and supinator longus. Erb showed that this combination of muscles was supplied by the 6th cervical spinal root, and discovered a point in the posterior triangle of the neck situated behind the posterior border of the sterno-mastoid and 2·3 centimetres above the middle of the clavicle, stimulation of which by the faradic current caused contraction of these muscles only. This combination of paralysed muscles has been called *Erb's paralysis*. Since then, anatomical, clinical, and experimental observations have shown that this root is more probably the 5th and not the 6th cervical. In addition to the deltoid, biceps, brachialis anticus, and supinator longus, the supra- and infra-spinati and sometimes the supinator brevis are also paralysed.

This combination may be due to a rheumatic neuritis, or to pressure from a tumour, but the most common cause is a fall from a height on to the side of the head and the shoulder of the same side, forcing them apart so that the brachial plexus is violently stretched, and the upper roots, the 4th and 5th cervical, being attached furthest away from the angle formed by the upper limb and the spine, are more violently stretched than the lower roots which are below the level of the clavicle. The paralysis is occasionally caused by direct pressure on the 5th cervical root, as in a case published by the writer, of a woman who went to sleep with her neck resting on the sharp edge of a table, and on waking up had a tender spot over the posterior border of the sterno-mastoid four centimetres above the middle of the clavicle corresponding to the part pressed upon. In addition to paralysis of the deltoid, supra- and infra-spinati, biceps, brachialis anticus, supinators longus and brevis, and teres minor, she had a feeling of numbness with anaesthesia of the thumb and the radial border of the forearm and arm up to the shoulder. Another form in new-born children is known as *Obstetrical paralysis*. It occurs after pressure of the forceps on the brachial plexus, or in breech presentations from severe traction on the shoulder. At birth one arm is noticed to be in the typical position due to paralysis of the same muscles as in Erb's paralysis.

In Erb's paralysis as in the obstetrical paralysis the loss of power occurs immediately after the injury, and the position of the upper limb is very characteristic. The humerus hangs straight down, and when lifted up and let go it drops like a flail; it is adducted to the trunk, and rotated inwards; the elbow is kept extended and the forearm is pronated. The patient is unable to advance the humerus or to abduct it away from the chest wall, and in attempting to do this the shoulder is elevated and the scapula is rotated with the inferior angle forwards, but no abduction occurs. The patient cannot flex the elbow, and supination of the forearm is lost or very weak. Flexion and extension of the wrist and fingers, and all the movements of the thumb and separation

of the fingers by the interossei are well performed. In Erb's paralysis sharp pains are felt along the shoulder and the outer border of the arm and the radial border of the forearm down to the thumb, and anæsthesia is found on the outer half of the arm and the radial half of the forearm involving sometimes the thumb.

If the lesion of the brachial plexus is more extensive, other muscles in addition to those given above may be involved, as in two cases published by the writer: in one, besides the supra- and infraspinati, deltoid, biceps, and supinator longus, the triceps, pectoralis major, latissimus dorsi, pronator teres, and flexor carpi radialis were almost completely paralysed; while in the other, the extensors of the wrist and digits were also affected. These cases were due probably to the 6th and 7th roots being also involved. Cases in which the triceps, latissimus dorsi, and lower half of the pectoralis major are involved without the deltoid-biceps group are not usually met with, though they might occur.

In Erb's paralysis the muscles of the wrist and digits escape entirely, but a group has been observed in which the converse occurs, and these muscles are involved while the deltoid-biceps group escape. These cases are not produced by falls on to the head and shoulder, but by direct injury, as gunshot-wounds below the clavicle involving the 1st dorsal root and perhaps the 8th cervical root of the plexus. The thenar and hypothenar muscles and the interossei are paralysed, so that it is not possible to abduct and adduct the thumb properly or separate the fingers or to abduct the little finger; the little and ring-fingers suffer more than the other fingers and thumb. Owing to paralysis of the interossei the fingers become 'clawed.' Anæsthesia and sharp pains occur in the 3rd and 4th fingers, along the ulnar border of the forearm, and sometimes along the inner border of the humerus up to the axilla. Ocular troubles are noticed in these cases, and consist in inequality of the pupils, especially marked when the eyes are shaded. Moreover it is noted that the pupil on the same side as the paralysis, though it contracts to light, does not dilate fully when shaded; also the upper eyelid falls slightly and the palpebral fissure is smaller on the paralysed side. These changes are due to paralysis of the dilator fibres of the iris and of the fibres of Müller's muscle, which come from the spinal cord through the sympathetic and have been found in the dog to leave the cord by the 1st dorsal root.

In all these cases of paralysis the affected muscles waste, and give the typical reaction of degeneration (see ELECTRICITY IN MEDICINE). If the roots are ruptured and do not join again, the muscles in time lose all their excitability and do not then recover. The *diagnosis* has to be made from other forms of neuritis, by the characteristic combination of the paralysed muscles; and from progressive muscular atrophy, by the sudden onset and the presence of anæsthesia. The *prognosis* differs according to the cause and the degree of the injury. In cases of falls on to the head and shoulder recovery would depend on whether the roots of the plexus were only stretched or actually ruptured, but even after slight pressure, as in the case of the woman mentioned above, it was two or three months before the muscles recovered, and in severe cases it may be a year or more; but as long as the muscles react to the constant current there is a chance that they may recover. The obstetrical cases as a rule recover,

but not invariably. The *treatment* consists in keeping up the nutrition of the muscles by massage and by a gentle constant current, using only just enough to cause a contraction of the muscles. In cases of paralysis due to a tumour growing on, or pressing on, the roots of the brachial plexus, removal of the tumour and suturing the ends of the nerves might be required.

C. E. BEEVOR.

BRADYCARDIA.—An infrequent pulse (below 60), corresponding to infrequent heart-beat. The term is not properly applied to cases in which a frequently or normally beating heart only transmits few pulsations to the wrist. The condition is not a disease in itself; it is merely a sign which may point either to grave conditions, or to what is practically physiological. The only point of importance is the means of diagnosis between the two groups.

A. PHYSIOLOGICAL.—(1) This variety may be congenital, present in several members of a family, and consistent with vigorous life; (2) it may develop in many old people; (3) it may be present in all states of hunger; (4) it may follow vomiting, an important sequence, as it may give rise to a groundless suspicion of cerebral disease; (5) it is often present during the puerperium; and (6) it may occur as a result of certain drugs, especially digitalis and the salicylic compounds.

B. PATHOLOGICAL.—(a) *Temporary.*—This form is very commonly met with in convalescence from acute diseases, especially pneumonia and influenza, and less often after diphtheria, enteric fever, erysipelas, and rheumatism. It is usually a good sign; it lasts a few days and then gradually passes off. If it does not pass off, and the pulse remains below 40 for some months, the prognosis is bad. This is especially noticeable after influenza. It is of very serious import if it occurs at the height of a fever, e.g. diphtheria, a pulse below 40 pointing to a fatal termination. It may also occur in jaundice, uræmia, and lead-poisoning.

Paroxysmal bradycardia is comparable to paroxysmal tachycardia, but much less common. Very few well-described cases are on record. A patient apparently in good health gets sudden attacks of slow-beating heart, with palpitation, dyspnoea, nausea and vomiting, even convulsions, and returns to apparently normal health after the attack is over. The cases are rare, and their pathology obscure. The condition does not occupy the well-defined position of paroxysmal tachycardia.

(b) *Permanent.*—A continuous pulse-rate below 40 lasting some months after an acute fever, e.g. influenza, is ominous. This form may be associated with nervous diseases, especially cerebral abscess, epilepsy, and affections of cervical vertebrae involving the spinal cord; or may accompany certain cardiac conditions, such as aortic stenosis, occlusion of the coronary arteries, and mitral stenosis. Bradycardia and organic heart-disease may be associated with recurring attacks of convulsions. The prognosis in these cases is generally bad, but a small number do well for even many years.

Pathology of the conditions leading to bradycardia.—Either (1) there is a temporary or a permanent lowering of activity in the cardiac centre of the medulla oblongata, due to various causes, among which are direct poisoning, exhaustion, lessened blood-supply; or (2) the heart itself is affected in its action by changes in its muscle, or blocking of its coronary vessels.

The treatment is that of the underlying conditions.
GUSTAVE SCHORSTEIN.

BRADYLALIA (*βραδύς*, slow; *λαλιά*, speech).—An abnormal slowness of utterance.

BRAIDISM.—SYNON.: Hypnotism.—Braidism is the name which, after its inventor, James Braid, has been applied to a therapeutic method destined to utilise the undoubted powers of mind over body for the cure of various diseases. In essence it consists of a species of Mesmerism, the patient being reduced to a partial or complete trance-like condition, by being made to look fixedly for a few seconds at a bright object held by the operator at 'about eight to fifteen inches above the eyes, at such a distance above the forehead as may be necessary to produce the greatest possible strain upon the eyes and eyelids, and enable the patient to maintain a steady fixed stare at the object.' The patient must be made to understand that he is to keep his eyes steadily fixed on this object, and his mind riveted upon the image of it. After so short a time as ten or fifteen seconds some patients may be intensely affected; and if so, it will be found, on gently elevating the arms and legs, that the patient has a disposition to retain them in the situation in which they have been placed. 'If this is not the case,' Braid writes, 'in a soft tone of voice desire him to retain the limbs in the extended position, and thus the pulse will speedily become greatly accelerated, and his limbs in process of time will become quite rigid and involuntarily fixed.' By slightly prolonging this process a condition of profound 'nervous sleep' may be induced, in which operations may be performed as easily and in as painless a manner as if the patient had been under the influence of chloroform. All this has been abundantly proved by Esdaile and others, who performed numerous operations upon Hindoos, with absence of all pain, while they were in the hypnotic state. In his attempts to cure morbid conditions, however, Braid only rarely proceeded so far as to induce actual unconsciousness. While in a semicataleptic condition the patient's attention is strongly directed to the morbid part, and some very marvellous instances of relief are recorded by him, said to have been effected under the influence of this faculty only, without the aid of imagination, since some of the patients operated upon were quite incredulous as to any good being likely to result. The method has now fallen into disrepute. This has been due to two causes. First, because of the facility with which it may be practised by non-medical persons, and the difficulty often experienced, even by medical men, in rousing persons from the state thus induced. And, secondly, because, even when practised by medical men, disagreeable consequences are apt to follow with some patients: fits may be induced, or the patient's moral and emotional control may be for a long time greatly impaired. The labours of many French physicians have however done much of late years to render definite and precise our knowledge of the various phases of hypnotism, of the modes in which they are inter-related, as well as concerning the best means of inducing or terminating this or that particular phase. See MESMERISM.

H. CHARLTON BASTIAN.

BRAIN, Abscess of.—See BRAIN and MENINGES, Septic Diseases of.

BRAIN, Anæmia of.—DEFINITION.—A condition in which the blood contained within the brain, and especially within the capillaries of the brain, is deficient in quantity, or defective in nutritive quality, or both.

ÆTIOLOGY.—*General cerebral anæmia* may be due to the following causes:—(1) It may be a part of systemic anæmia—defect in quantity or quality of the whole blood, due to causes which are considered elsewhere. This is often seen in cases of hæmorrhage, of exhausting discharges, or of defective blood-formation, as in chlorosis and pernicious anæmia. (2) The supply of blood to the brain may be deficient, the quantity of blood in the body being normal. The cerebral anæmia is then part of a cephalic anæmia. In consequence of posture in relation to gravitation, the supply of blood to the head is that which is especially interfered with by any interference with the movement of the blood. At the same time the fact that the brain is contained in a closed cavity, and the unreadiness of the cerebrospinal fluid to undergo rapid change of distribution, preserve the blood in the head from the extremely rapid diminution to which it would probably otherwise be liable—possibly, indeed, to a degree incompatible with life. Cephalic anæmia may be due to cardiac weakness, or to causes acting through the nervous system on the heart, as in swooning. In general anæmia, the lessened cardiac power increases the cerebral deficiency. Whatever lessens the amount of blood discharged from the heart at each systole, such as aortic or mitral disease, may be a cause of cerebral anæmia. Pressure on the vessels conveying the blood to the head, as by an aortic aneurysm, has a similar effect. Unequal distribution of the systemic blood is another cause. The intestinal vessels, if dilated, are capable of containing a large part of the blood of the body, and the effect of their engorgement is often seen after paracentesis abdominis. One theory of shock ascribes its mechanism to vaso-motor dilatation of these vessels, and consequent anæmia of the rest of the system. Severe diarrhœa or dysentery is an occasional cause of symptoms of a very pronounced and prolonged character. (3) The capacity of the cerebral vessels may be diminished by pressure on the brain, exerted by effusions of fluid (hydrocephalus), of blood (in cerebral and meningeal hæmorrhage), or by growths within the skull. (4) General contraction of the arteries of the brain is a supposed cause, probably confined to some toxic states, such as that of Bright's disease in which such contraction can be seen in the retina.

Partial cerebral anæmia is due to local obstruction to the passage of the blood through an artery. To be permanently efficient such obstruction must be situated beyond the circle of Willis. Ligature of one carotid causes immediate symptoms of cerebral anæmia, loss of function in one hemisphere, but permanent symptoms are not frequent. Pressure on, or disease of, one carotid, for the same reason, rarely gives rise to symptoms. Obstruction in certain arteries of the brain may cause local anæmia, sudden or gradual, temporary or permanent, according to its cause. Such obstruction may be due to narrowing of the calibre of the vessel by atheromatous changes in its wall, or may be due to actual occlusion by embolism or thrombosis. The pressure-effects of an intruding substance within the skull (tumour or clot) act most intensely in one region of the brain, and may influence it only. Arterial

spasm has been thought to be an occasional cause of local anæmia, but on doubtful grounds. Extensively as it has been invoked as a mechanism to explain symptoms, its occurrence has not yet received any definite proof.

It is obvious that of these causes some act suddenly, others gradually, and the symptoms produced will differ accordingly.

ANATOMICAL CHARACTERS.—The principal anatomical effect of cerebral anæmia is pallor of the brain, observable chiefly in the paler tint of the cortical substance, and the diminished number of red spots in the white centre. The pallor may be partial or general. The membranes are usually pale, but in some cases of partial anæmia they are hyperæmic from dilatation of the vessels and stasis in them. Effusion of serum in the meshes of the pia mater and between the convolutions may be found in general anæmia.

SYMPTOMS.—The symptoms of this condition vary according as the anæmia is suddenly or slowly produced, and as it is general or partial.

(1) In *sudden general anæmia* of the brain the sufferer feels drowsy; the special senses are dulled; noises in the ears and vertigo are complained of; the pupils are at first contracted; sight may fail; muscular power is weakened; respiration is sighing; the skin is pale, cold, and moist; nausea is common; and headache is rare. If the anæmia is more intense, consciousness is lost; there is universal paralysis; and general convulsions may occur, epileptic in character, these being especially frequent in sudden extensive loss of blood. The pupils dilate, and the coma may deepen to death. The loss of sight may persist in cases which recover, but secondary retinal changes are then found.

(2) When general anæmia of the brain is *slowly* produced, the state of the cerebral functions is usually that of 'irritable weakness.' Their action is imperfect in degree, and excited with undue facility. There is mental dulness and drowsiness; sometimes, however, insomnia is troublesome. Delirium is common in severe cases, and is conspicuous in some forms of imperfect blood-nutrition, as in the so-called 'inanition delirium.' Headache, usually general, is a common symptom. Sensory hyperæsthesiæ, tinnitus, *muscæ volitantes*, and vertigo are frequent. Convulsions are rare, but muscular power is generally deficient. All these phenomena are more marked in the erect than in the recumbent posture, especially when the erect posture is suddenly assumed. It has been remarked that some anæmic persons can think well only when lying down. In young children, after exhausting discharges, as diarrhoea, symptoms referable to cerebral anæmia are common, namely, somnolence and pallor, with depressed fontanelle and contracted pupils. The somnolence may deepen to coma with insensitive conjunctiva, and the coma increase to death. Such symptoms have been called *hydrocephaloid*, from some resemblance to those of 'acute hydrocephalus' (acute meningitis).

(3) *Partial cerebral anæmia* causes, if complete, loss of function in the affected area; and if it be permanent, as in obstruction of a vessel beyond the circle of Willis, necrosis of the cerebral tissue results (*see* BRAIN, Softening of). If incomplete and sudden, there is a temporary arrest of function. Ligature of one carotid artery, for instance, causes transient weakness and numbness in the opposite half of the body. There may be at first an over-

action of grey matter, causing, in certain regions, unilateral convulsions. The return of the blood-supply, as when a plug shifts its position or is broken up, may not immediately be followed by restoration of function, which may be put in abeyance for some time by the effect of the sudden influence. The loss of memory is occasionally of a remarkable character, involving only the power of retaining new impressions. If slowly developed, as in atheroma of arteries, pain and vertigo are common, with recurring local symptoms, such as numbness, tingling, and weakness.

In all cases of long-continued cerebral anæmia, prolonged damage to the nutrition of the brain may result. In the child the development of the brain may be arrested; in the adult, loss of memory and of general mental power indicates the deterioration of structure. These may last for many months, but commonly pass away at last.

PATHOLOGY.—The symptoms are, as already stated, dependent mainly on the defective quantity and quality of the blood circulating in the brain. Some influence may probably be ascribed to the diminution in the blood-pressure to which the nerve-elements are ordinarily exposed (Burrows). Nothnagel has pointed out that the symptoms indicate an early affection of the respiratory centre in the medulla, and of the cortical grey matter. The convulsions in acute anæmia have been ascribed to irritation of the medulla or of the pons Varolii, but our present knowledge suggests that they are due to the altered function of the motor region of the cortex. This has been confirmed by the discovery of changes in the motor cells in such cases; the staining particles found within the cells blend into a uniform mass, and the nucleus loses its central position, and may be sometimes extended. These changes are widely spread and explain the interference with function. When they have occurred to only a moderate degree the normal condition may be apparently restored.

DIAGNOSIS.—The diagnosis is not difficult. It rests on the recognition, in a given case, of the cause of cerebral anæmia; and on the exclusion of graver maladies, as organic cerebral disease. With the latter, it should be remembered, anæmia of the brain, local or general, often co-exists. Some symptoms of hyperæmia of the brain closely resemble those of anæmia. A common pathological state of imperfect blood-renewal exists in both conditions.

PROGNOSIS.—The extent to which the cause of the anæmia is amenable to treatment, and is of transient character, must influence the prognosis. As a rule this is favourable when there is no organic disease of heart, vessels, or brain. In pernicious anæmia, the prognosis is, of course, unfavourable. Hydrocephaloid symptoms in infants, if met by prompt and suitable treatment, are often recovered from.

TREATMENT.—The treatment necessarily varies in the several forms of the affection, but it is in the main causal. The beneficial effect of the recumbent posture in affording immediate relief to the symptoms, and obviating permanent damage to the cerebral nutrition, must be always remembered. In acute anæmia from loss of blood, the head must be kept continuously low, stimulants freely administered, and as a penultimate resort bandages applied to the limbs from below upwards may increase the proportionate supply of blood to the brain. If

this fails transfusion must be had recourse to. In chronic anæmia sudden change of posture should be carefully avoided, and ferruginous tonics are needed. More important than any other element is physical rest, permitting more of the scanty amount of oxygen that can be conveyed by the blood to be used for the nutrition of the protoplasm of the tissues on which the vital functions depend. All use of the oxygen by muscles should be avoided. In spasm of the cerebral vessels, nitrite of amyl or nitroglycerin may be employed; and a course of the latter, in a small dose two or three times a day, lessens any liability to irregular contraction that may exist. In the cerebral anæmia of syncope, the recumbent posture, stimulants to the skin, cold water, faradisation, sinapisms, and ammonia to the nasal mucous membrane, assist the recovery of cardiac action and the return of consciousness. In all cases, carefully regulated food and stimulants are needed; beef-tea should be given in small quantities at frequent intervals. The group of symptoms called hydrocephaloid requires similar treatment.

W. R. GOWERS.

BRAIN, Aneurysm of.—See **BRAIN, Vessels of, Diseases of.**

BRAIN, Carcinoma of.—See **BRAIN, Tumours of.**

BRAIN, Compression of.—See **BRAIN, Injuries of.**

BRAIN, Concussion of.—See **BRAIN, Injuries of.**

BRAIN, Congestion of.—See **BRAIN, Hyperæmia of.**

BRAIN, Hæmorrhage into.—**SYNON.** : Cerebral Apoplexy; *Fr. Hémorrhagie cérébrale*; *Ger. Hirnblutung*; *Hirnschlag*.

DEFINITION.—Escape of blood, by rupture of a vessel, into the substance or cavities of the brain. Hæmorrhage into the meninges is separately described.

Cerebral hæmorrhage is commonly due to the rupture of an artery; very rarely hæmorrhage into the ventricles is due to the rupture of a vein. Occasionally, minute extravasations are caused by rupture of capillaries, and may cause, by their number, an infiltration of the cerebral tissue with blood. This is seen in cases of venous thrombosis. Hæmorrhage from arteries or veins may also be due to their laceration by injury.

ÆTIOLOGY.—Arterial hæmorrhage is usually due to the coincidence of weakening of the vascular wall and increased pressure within the vessel. The changes in the walls permit the formation of military aneurysms which give way. Larger aneurysms may also cause cerebral hæmorrhage (see **BRAIN, Vessels of**). The causes of these conditions may be regarded as the conditions *predisposing* to cerebral hæmorrhage. Hereditary influence is sometimes distinctly seen, as a tendency to vascular degeneration, or to conditions which, as renal disease, produce such degeneration. Similarity of vascular distribution may also be inherited, and may determine the locality of strain, and, therefore, first of degeneration and expansion and ultimately of rupture. Cerebral hæmorrhage is most frequent after fifty years of age, but occurs at any age, though rare during the first half of life. It becomes

less common in extreme old age. It is nearly twice as common in men as in women. It is said to be more frequent in temperate than in tropical climates, in winter than in summer, and at high than at low elevations. Certain acquired conditions act as predisposing causes. Chronic Bright's disease leads to early and extreme degeneration of vessels, as well as directly to hypertrophy of the heart and increased blood-pressure; hence it predisposes powerfully to cerebral hæmorrhage. In purpura and scurvy, cerebral hæmorrhage occasionally occurs, and also in pernicious anæmia and leuchæmia. In the latter it is a not uncommon cause of death. In these blood-states the mechanism is doubtless impaired nutrition of the walls of the smaller vessels, which permits them to be bulged and thinned by a blood-pressure that may even be below the normal. The state of vascular repletion known as 'plethora' was formerly thought to be a frequent cause of cerebral hæmorrhage. It probably does aid other causes, but rarely co-exists with the more efficient, and so takes a subordinate position. Chronic alcoholism and opium-eating are said to promote vascular degeneration.

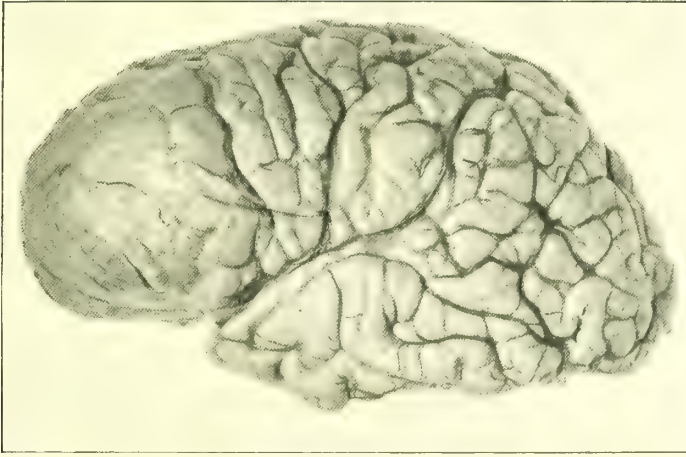
Among other remote causes must be reckoned heart-disease and syphilis, both of which commonly act by causing an aneurysm larger than those that are the special mechanism of hæmorrhage. Nevertheless, such aneurysms often escape even the most careful search.

The *proximate* causes of cerebral hæmorrhage are the weakened state of the wall of the vessel, and commonly some increase of blood-pressure. The vessel-wall is weakened by degeneration, and is often imperfectly supported in an atrophied brain. The increase of pressure within the vessels may be permanent, as in peripheral obstruction, with or without hypertrophy of the heart; or temporary, as in excited action of the heart, or impeded circulation through the lungs during effort. These causes are considered more fully in the article on **BRAIN, Vessels of, Diseases of**.

Hæmorrhage from a vein is rare, except as the result of laceration by direct injury, or of ulceration invading the vein from without. Varicose veins in the pia mater or ventricles may sometimes rupture.

Considerable capillary hæmorrhage is usually due to the venous obstruction already mentioned. It may also result from cephalic congestion due to any cause, from capillary embolism, and from the blood-states which cause larger vessels to give way. In all these blood-states similar extravasations are often seen in the retina from the same immediate cause.

ANATOMICAL CHARACTERS.—In intra-cerebral hæmorrhage, the blood is extravasated into the substance or into the ventricles of the brain—into the ventricles usually by rupture of a previous extravasation within the cerebral substance. In the latter situation the blood occupies a cavity formed by laceration of the brain-tissue; rarely, when very minute and 'capillary,' by merely separating the fibres. In size an extravasation varies from that of a pea or even smaller, up to that of the fist. The blood is found clotted, and reddish-black in colour; fragments of brain-tissue are mingled with it. The cavity containing it is often very irregular in shape; its walls are uneven, present projecting shreds of lacerated brain-substance, and are blood-stained and softened—at first by imbibition of serum, and later by inflammation.



CEREBRAL ABSCESS.

Female, aged 20 years. Four days after confinement developed symptoms, diagnosed as 'puerperal melancholia.' Some weeks later was admitted to an asylum in a stuporose condition, and died in three days. Cause of abscess was a left pyo-nephrosis following blocking of upper end of ureter by a calculus. Reproductive organs normal. Figure shows the abscess-wall formed by the markedly stretched frontal convolutions. The cavity was ragged, and contained irregular trabeculae and thick, greenish pus. The rest of the brain and membranes were very oedematous, but otherwise normal.



CEREBRAL HÆMORRHAGE.

Figure shows a hæmorrhage into the right external capsule which resulted in left hemiplegia. Death occurred 30 hours after the onset of symptoms. The site of the rupture into the right lateral ventricle, on the outer side of the anterior horn, is visible, as is also the blood-clot in the anterior and posterior horns of both the lateral ventricles.

J. S. BOLTON.

Many small extravasations are often seen in the neighbourhood of a larger clot. Usually there is only one large extravasation; sometimes, however, there are two or three. The extravasated blood exerts pressure; the convolutions are flattened; the falx is bulged to the opposite side; and the rest of the hemisphere is anæmic. The effused blood may tear its way into the lateral ventricle; it then speedily distends both lateral ventricles and the third and fourth ventricles, and may escape by the openings at the lower extremity of the fourth ventricle, central and lateral, into the subarachnoid space. Or the blood may escape to the surface, infiltrate the pia mater, and tear its way into the subarachnoid cavity, often by a very small opening. It is rarely that the artery from which the blood has escaped can be detected. Occasionally the extravasation can be traced to the rupture of an aneurysm of some size. In other cases miliary aneurysms may be found on many vessels. The larger arteries commonly present atheromatous changes, but these are only associated as the result of some of the causes, and especially of age.

After a time the extravasated blood undergoes changes. The clot shrinks and gradually becomes, first chocolate-coloured, then brown, and ultimately a reddish-yellow; and it then contains chiefly fat-globules, pigment and other granules, and hæmatoidin-crystals. The rapidity with which it undergoes this change varies. It is said that the distinctive blood-colour has disappeared as early as the twentieth day. Meanwhile the walls of the cavity undergo changes. The inflammation, in rare cases excessive and purulent, is usually conservative, and leads to the formation of connective tissue. A firm wall is thus developed, the inner surface of which ultimately becomes smooth by the softening and removal of the loose fragments of brain-substance; by this means a cyst is formed. It is said that connective tissue may extend across its cavity, and that in rare cases, the fluid being absorbed, the cyst-walls may unite, and a cicatrix result. Such cicatrices are, however, much more frequently due to softening than to hæmorrhage.

Hæmorrhage may occur in any part of the brain, but is more frequent in some situations than in others. The most frequent seat is the corpus striatum and the region just outside it: nearly half the intra-cerebral hæmorrhages are in this situation. Other primary seats, in the order of frequency, are the centrum ovale, the cortex, the pons and peduncles, the cerebellum, the optic thalamus (often affected by an extension of the hæmorrhage from the corpus striatum), the posterior portion and the anterior portion of the hemisphere. The frequency of hæmorrhage in the cerebrum is twenty times greater than into the cerebellum. The frequent extravasation into and outside the corpus striatum is explained by the vascular supply. As the rupture is often near the edge of the lenticular nucleus, the blood tears up the white substance; and this makes the frequency of primary hæmorrhage into the centrum ovale difficult to ascertain.

Traumatic hæmorrhage occurs into and from a lacerated portion of brain, and is most frequently found on the surface, occupying mainly the middle of the convex portion of each convolution, and some other regions much exposed to injury, as the surface of the temporo-sphenoidal lobe, and the under surface of the frontal lobe. Ventricular hæmorrhage sometimes results from traumatic rupture of a small

vein on the surface of the corpus striatum (Prescott Hewett).

Soft tumours (especially gliomata) are sometimes the seat of hæmorrhage. The distinction from simple hæmorrhage (sometimes difficult) rests on their position being commonly one in which cerebral hæmorrhage is rare; and on some gelatinous-looking tumour-substance being found, into which hæmorrhage has not occurred, and which has characteristic microscopic features.

Other organs may be healthy, or present the changes which have been mentioned as predisposing causes; the lungs are usually secondarily congested, often intensely.

SYMPTOMS.—The occurrence of cerebral hæmorrhage is indicated by cerebral symptoms of two classes, the one general and more or less transient, the other local and more or less permanent. In addition to these there are sometimes premonitory symptoms; and commonly general symptoms, manifested by pulse, temperature, &c., which are secondary to the brain-lesion.

Premonitory symptoms, somewhat rare, are those of altered cerebral function due to associated atheroma, headache, vertiginous feelings, local weakness or numbness, slight mental changes, and slight affection of speech (*see* BRAIN, Vessels of, Diseases of; and BRAIN, Anæmia of). But the disease causing them is commonly distinct from that causing the hæmorrhage. The miliary aneurysms cause no symptoms, and hence it is only in the case of aneurysm of larger vessels that there are true premonitory symptoms. Hence they are less frequent than in cases of softening.

The *onset* of hæmorrhage is usually accompanied by apoplexy, i.e. loss of consciousness and of power of motion and of sensation, often with relaxation of the sphincters and loss of reflex action (*see* APOPLEXY, CEREBRAL). These symptoms are profound and lasting, according to the size of the hæmorrhage and its position; being especially marked in large and double effusions, in intra-ventricular hæmorrhage, and in hæmorrhage into the pons. In a case of moderate severity they last only a few hours, and gradually pass away. In severer cases they may deepen until death occurs from failure of respiratory power. Death is rarely very speedy, life being usually prolonged for some hours even in the most rapidly fatal cases. In rare instances of hæmorrhage into the medulla, and also in meningeal hæmorrhage, death has occurred in five minutes, probably in each case from the rapid interference with the respiratory centre.

The temperature is at first lowered one or two degrees; the pulse becomes less frequent; and the respiration slow. The Cheyne-Stokes respiratory rhythm often precedes death. After a few hours the temperature rises to the normal and in mild cases stops there, but in graver cases it rises above the normal two or three degrees. In some very grave cases the initial fall or subsequent rise may be extreme and go on until death occurs, sometimes reaching 90° in the one case, and 107° or 108° in the other (Bourneville). The urine is at first abundant, of low specific gravity, and acid in reaction.

In slight cases of cerebral hæmorrhage there may be no loss of consciousness. Vomiting in such cases is not infrequent. In other rare cases of large hæmorrhage, especially between the external capsule of the corpus striatum and the island of Reil (Broadbent), the loss of consciousness comes on

gradually, after other symptoms, as of shock, for example, have lasted for an hour or two. These cases have been termed *in Gravescent*.

Local symptoms, chiefly unilateral, often permanent, and always of longer duration than the loss of consciousness, are present in all cases of circumscribed cerebral hæmorrhage, except in the instances in which, by its central position in the pons and medulla, it causes directly bilateral effects only, which are added to and intensify the general symptoms caused indirectly. In a unilateral cerebral lesion, the direct symptoms are unilateral loss of power of voluntary movement and often of sensation, accompanied sometimes by convulsion or rigidity. These local symptoms may commence a few minutes or longer before the loss of consciousness. They co-exist with the apoplectic condition, and may often be recognised, even during coma, by the flaccidity of the paralysed limbs, which fall more helplessly than those of the opposite side; by inequality of the mouth and of the pupils; by conjugate deviation of the head and eyes towards the side of the brain injured; by convulsive movements; and, as the apoplexy clears, by the detection of unilateral defect of sensibility. The coma passing away, these signs become more distinct, and all the symptoms of hemiplegia remain, varying in intensity and extent according to the position of the lesion. The apoplectic state may recur after its disappearance—a grave symptom, usually indicating that a fresh hæmorrhage has occurred in the same or the other side of the brain, or more frequently that the blood has escaped into the ventricles. In the former case the unilateral symptoms, conjugate deviation of the head and eyes, &c., are increased on the same, or transferred to the opposite side; in the latter the unilateral symptoms disappear, and general powerlessness and deep coma supervene, with stertor, relaxation of the sphincters, lowered temperature, and impeded respiration. Death always ensues.

Convulsion may be a conspicuous symptom at the onset or subsequently. It is usually unilateral in its course or commencement, beginning on the side paralysed, rarely affecting only the non-paralysed side. In cases in which the blood escapes slowly, convulsions may occur on one side, and in the course of an hour or two may cease, the side at the same time becoming powerless. This is due to the motor structures, commonly those of the cortex, being at first irritated by the hæmorrhage, and afterwards interrupted or compressed so as to be incapable of function. Where convulsion is not met with, muscular twitching or rigidity may occur. General or widely spread rigidity or twitching points to a bilateral lesion; if with coma, to ventricular hæmorrhage.

After a day or two symptoms of irritation about the cerebral lesion come on, such as headache, delirium, and rigidity in the paralysed limbs. During this period the temperature rises above the normal, and the pulse becomes quick. On their subsidence these symptoms, if the lesion is slight, may be scarcely recognisable, and a stationary period ensues, at the end of which recovery of power over the paralysed limbs begins. In slight cases power may be recovered very speedily. Its return depends upon the structural recovery of slightly damaged tissue, and on other parts taking on an increased function in compensation for that which is destroyed. The electric irritability of the muscles exhibits little change. Sometimes, how-

ever, when there is great irritation at the lesion propagated downwards to the cord, a marked initial increase in the irritability may precede a considerable depression, coincident with rapid wasting. Recovery of power is rarely complete except in those cases in which the area of damage is very small; and when the damage is large and affects an important motor region, there may be no recovery, loss of power persisting, commonly with more or less 'late rigidity' in the paralysed limbs. Slight permanent mental change often remains, and as the motor power is recovered, ataxic and other disorders of movement may supervene in the limbs which were paralysed, although much less commonly than after softening.

DIAGNOSIS.—The diagnosis of cerebral hæmorrhage rests on the symptoms of a localised cerebral lesion occurring suddenly. The conditions from which it has most commonly to be distinguished are—congestion of the brain; softening of the brain, embolic and thrombotic; and sometimes tumour. For the distinction from it of other causes of apoplectic loss of consciousness, uræmia, post-epileptic coma, &c., see APOPLEXY, CEREBRAL.

From *congestion* the chief distinction of the cerebral hæmorrhage lies in the transient nature of the loss of consciousness and in the slightness and general character of the symptoms which characterise the former. Congestion generally, hæmorrhage only sometimes, comes on during effort; and the absence of history of effort is in favour of the latter. Similarly, the premonitory symptoms which are usually present in congestion are generally absent in hæmorrhage. The loss of motor power, and symptoms of cerebral shock, are much greater in hæmorrhage than in congestion. It must be remembered that the two states may co-exist.

From *softening* consequent on *embolism* cerebral hæmorrhage has also to be distinguished. The subjects of the latter are usually of an earlier age than those of hæmorrhage; their vessels are healthy, but they have organic heart-disease, which is often grave. There may be evidence of embolism elsewhere, in spleen, kidney, or retina. Loss of consciousness is often absent in embolism, and the paralysis may come on suddenly or deliberately, and is sometimes brief in duration but sometimes enduring. Deep apoplectic coma should always lead to a suspicion that hæmorrhage is the lesion, and this especially in cases of young persons with heart-disease. It is important not to let the presence of heart-disease exclude the idea of hæmorrhage, because it is in such cases that the most severe forms of hæmorrhage are met with, in consequence of aneurysm, due to embolism, which has ruptured.

From *softening* due to *thrombosis* the distinction is often difficult. Age is no longer significant, since both occur in later life, but thrombosis is more common in extreme age. The presence of chronic Bright's disease is slightly in favour of hæmorrhage; but atheroma, the cause of softening in later life, is also induced by it. In middle life advanced kidney-disease makes hæmorrhage far more probable. The most important diagnostic indications, however, are those drawn from the state of the heart and circulation, whether these are such as to conduce to the rupture of an artery or to allow clot to form in it. On the one hand we have a full or tense pulse and hypertrophied strongly acting heart; on the other a feeble pulse and weak often irregular heart. These indications

are of great weight. Excitement at the time of the onset suggests hæmorrhage; depression, physical or mental, suggests softening. The occurrence of previous slight hemiplegic or other cerebral attacks points to softening. In the attack, loss of consciousness is much more considerable, in proportion to the subsequent paralysis, in hæmorrhage than in softening. But the distinction on this ground is often very difficult, since loss of consciousness may be absent in slight hæmorrhage, and considerable in an extensive softening. A deliberate onset is in favour of softening; and so are much mental change, and early rigidity. A definite fall of temperature is generally caused by rupture of a vessel, and so also is a rise within twelve hours. Paralysis of sudden onset, in cases of *tumour*, may be ascribed to hæmorrhage into the growth, to which it is generally, but not always, due. Usually, enquiry elicits a history of gradual, long-continued symptoms; headache and intense optic neuritis are strongly suggestive of a tumour into which a hæmorrhage may have occurred, but moderate optic neuritis may be the effect of Bright's disease and is often associated with headache.

Hæmorrhage into the substance of the brain is distinguished from *meningeal hæmorrhage* by the pain and mental excitement being less conspicuous, convulsion rarer, and by the presence of symptoms of a local lesion. *Hæmorrhage into the ventricles* resembles meningeal hæmorrhage in the generality of its symptoms, but these usually succeed the symptoms of a circumscribed lesion.

PROGNOSIS.—During the early stages of the attack the prognosis in hæmorrhage into the brain must be guided by the intensity of the symptoms, and by the place of the lesion, as far as that can be ascertained. Death is probable if the coma is profound, or has not begun to lessen at the end of twenty-four hours, and if the early depression, or the subsequent rise, of temperature and pulse-rate is great. Most cases are hopeless in which there is marked interference with respiration—even hicough is a cause of anxiety. When the symptoms indicate ventricular hæmorrhage, or hæmorrhage into the pons, there is little chance of survival. In meningeal hæmorrhage with coma the prognosis is exceedingly grave. After some consciousness has been recovered, most of the danger of immediate death is over, the freedom from much secondary pyrexia, from lung-congestion, and from early bed-sores, are favourable indications. The chance of recovery from the paralysis is estimated by evidence of position of the lesion, and especially by any indications of improvement. In most cases the power of standing and walking, at least a little, returns, because each leg can be innervated to some extent by the hemisphere of the same side. Recovery of the hand can only be foretold by the observed degree of movement regained, and its rapidity. Early rigidity is unfavourable. The danger of recurrence is in proportion to the extent of vascular disease, and the existence of irremovable causes of increased arterial tension. Hence the prognosis is rendered unfavourable by advanced age, or chronic renal disease, and by the evidence of general premature decay.

TREATMENT.—*During the attack.*—Rest is the most important. The patient should remain, as far as possible, where he is seized; stillness must be secured, and all effort avoided. The posture

should be recumbent, with the head raised, but without flexion of the neck. Any cause of passive cerebral congestion, such as a tight collar, must be removed. Venesection was formerly almost always employed in such cases—certainly too universally, but is now discarded—perhaps too absolutely. Loss of blood lessens the force of the heart and vascular tension; it thus hastens the cessation of hæmorrhage. It may be used with probable advantage if the pulse is full, bounding, and incompressible, the heart acting strongly, and there is reason to believe that the intracranial hæmorrhage is increasing. A small quantity of blood (e.g. eight ounces) should be withdrawn rapidly. In ventricular hæmorrhage, venesection is probably powerless for good. When the indications are not perfectly clear, it is not wise to employ a measure now out of fashion.

The object to be aimed at by other measures must be to lower the arterial pressure by diverting the blood as far as possible from the brain. If the limbs are cold, warmth may be applied to them, aided by sinapisms. Purgatives, as croton oil or calomel, will divert the blood to the capacious intestinal vessels. Drugs which would cause contraction of the vessels are to be avoided, since, their influence being on the smallest vessels and universal, their tendency is to increase arterial tension and hæmorrhage. To this, however, digitalis is an exception, since its action seems to be chiefly on arteries not smaller than those that burst. Diuretics are useful for the same object; nitrous ether and juniper may be combined with digitalis. An attempt may be made to promote contraction of encephalic vessels by cold to the head or sinapisms to the neck, according as the head is hotter or colder than normal. The heart should be allowed to fall a little below the normal in force, but conspicuous failure of power should be avoided. Convulsion is effectually checked by ice to the head; bromide may be given if there is a tendency to the recurrence of convulsions.

After the attack.—During the stage of irritation, rest must be maintained, and the patient preserved from all sources of excitement. The bowels should be kept gently open by laxatives or by injections. Stimulants must be avoided, and the diet should be light. Pain or delirium may be relieved by cold to the head, a blister to the neck, or by Indian hemp, bromide of potassium, or a small dose of hydrobromate of hyosine.

During reparation the diet must be nutritious, but carefully regulated; and constipation of the bowels avoided. Upward massage of the limbs, and their gentle passive exercise, are wise; and after all symptoms of irritation have passed, say in two months, gentle faradisation may improve muscular nutrition, and accelerate any distinct return of power. Nervine tonics probably aid a little the recovery of slightly damaged structures: quinine and strychnine or very small doses of phosphorus if there is no kidney-disease. But their influence is small. As a rule the spontaneous tendency to such recovery as the destruction of brain-tissue permits, obscures the influence of therapeutical measures. But these may wisely be adopted in the hope that they have some influence in augmenting the gradual improvement. Change of scene, and cheerful mental surroundings are useful adjuncts, especially in the later stages of recovery.

W. R. GOWERS.

BRAIN, Hyperæmia of.—**SYNON.** : Congestion of the Brain.

DEFINITION.—Increase in the quantity of blood within the capillaries of the brain, other than that which is merely the early stage of inflammation.

Since neither the arteries nor the veins of the brain-substance can be over-distended with blood without capillary hyperemia, and since it is to capillary hyperæmia that the functional disturbance of the brain is related, this may be justly taken as the essential pathological element in cerebral congestion. The congestion may be *active*, when the capillaries contain, in consequence of arterial distension or dilatation, an increased amount of oxygenated blood passing rapidly through them; or it may be *passive*, when, from venous obstruction, the increased blood in the capillaries is slowly moving, and is becoming, or is in great part already, venous.

'Congestion of the brain' is often alleged because no other explanation of symptoms occurs to the mind of the practitioner, although there may be no real evidence of such a state. Like 'congestion of the liver,' it is often adopted merely because it is a convenient refuge for a destitute diagnosis, and pleases the patient as an explanation which both satisfies and gratifies him. Indeed, it is a malady of which both symptomatology and pathology are open to much question. Its existence should never be assumed on purely negative evidence. Its assertion is often so groundless as to be most discreditably to the profession. Some descriptions of it have been written entirely from cases of the cephalic sensations that attend hypochondriasis.

ÆTIOLOGY.—(A) *Active* congestion of the brain may be general or partial. Of the *general* form the *remote* causes are as follows:—Men are said to be more liable to it than women. Age increases its frequency, chiefly from the greater frequency of its causes; yet children, from the sensitiveness of their vaso-motor system, occasionally seem to suffer from an active form. The 'plethoric' condition, with its tendency to general overfilling of the vessels, is a predisponent. The *immediate* causes of excessive flow through the arteries of the brain may be thus stated: (1) Increase in the blood-pressure—either general, from excessive action of the heart (as in extreme hypertrophy or functional overaction); or partial, from an obstruction elsewhere, throwing an undue proportion of the pressure upon the cerebral vessels. This is seen in contraction of the aorta beyond the origin of the vessels to the head, and in sudden contraction of a large number of the systemic arterioles, as those of the surface, in exposure to cold and in ague. It may result from cardiac excitement, whether due to mental or physical causes. (2) Active arterial dilatation of vaso-motor origin may be excited by prolonged mental work, severe emotion, insolation, digestive disturbances, or from the presence in the blood of various poisons, such as alcohol, and amyl-nitrite or nitroglycerin. In these cases the vaso-motor disturbance may precede and cause, or may succeed and result from, the overaction of the brain-tissue, which is intensified by it. It results from the cardiac excitement of exophthalmic goitre. It seems to occur in the second stage of many attacks of megrim. In alcoholic poisoning the cerebral congestion may be in part, as Niemeyer suggested, secondary to the disturbance of brain-tissue, although the simultaneous flushing of the face makes this doubtful. In pyrexia the headache and delirium have been thought to be due to conges-

tion, but they more likely result from the blood-state. (3) Lessened atmospheric pressure may cause congestion of the brain, as in the 'Zenith' balloon-catastrophe; and it probably occurs when divers pass from the increased to normal pressure. (4) Gravitation in the recumbent posture may alone cause cerebral hyperæmia, or may powerfully aid other causes in producing it.

Partial active congestion of brain-tissue occurs chiefly from disease of the arteries, which perverts the local distribution of the blood-pressure; in organic brain-diseases, as tumour, hæmorrhage, &c.; after blows on the head; and probably also from local overaction of special centres. In many of these cases it is really a slight and transient condition which is indistinguishable from that of local inflammation.

(B) *Passive* congestion of the brain, when *general*, is the result of impeded return of blood from the head. It is intense in death from all forms of suffocation or by hanging. It may be due to pressure on the veins in the neck, as by tumours; pressure on the innominate veins by tumours or aneurysm; or obstruction to the circulation from violent respiratory efforts, as exertion, cough, or blowing wind-instruments. It may be caused by impediments within the circulation, such as tricuspid insufficiency and its causes in the lungs, or even by disease on the left side of the heart. The recumbent posture assists all these influences.

Partial passive congestion may occur from pressure on a cerebral vein, from local venous thrombosis, or from pressure by a growth on one of the cerebral sinuses.

ANATOMICAL APPEARANCES.—The capillaries are not visible to the naked eye even when over-distended, but with the microscope they are seen to be dilated, often to twice their normal calibre. Their distension is indicated by a deeper and reddish tint of the grey substance; and the fulness of the small arteries and veins shows itself in an increase, often very great, in the number and size of the red points visible on section of the white matter. In active congestion the arteries may be distinctly larger than normal; the minute vessels of the meninges are also distended. Indeed in all forms of general congestion, the state of the vessels is generally more distinct in the pia mater than in the substance of the brain. In all, however, it is chiefly the active congestion that would pass on to inflammation, or is really its first stage, that leaves distinct traces. In passive congestion the veins and sinuses are gorged with blood. It must be remembered, however, that such engorgement of the veins occurs in all cases of death from interference with the passage of the blood through the thorax. The state of the cerebral veins must therefore be carefully compared with that of the veins of other organs. Active congestion may sometimes leave no visible traces. After a time blood-pigment collects outside the vessels (Bastian), and serous effusion into the membranes and ventricles may be found. Later on the vessels may be permanently distended; degenerative changes, consequent perhaps on the effusion of serum, may lead to the production of wide spaces around them; and the whole brain may have undergone distinct wasting. It must, however, be pointed out that the absence or paucity of *post-mortem* evidence of active congestion is equally true of the skin, and does not establish its absence during life.

SYMPTOMS.—It is probable that many symptoms

have been erroneously ascribed to cerebral congestion; some because hyperæmia, due to the mode of death, was found *post mortem*; others because an assumed congestion was the readiest explanation.

The symptoms commonly referred to cerebral congestion may be grouped in two classes—those of excitement, and those of depression. Either of these may exist alone; those of excitement may precede those of depression; or they may partially co-exist. They may be slight or severe; acute or chronic. In all cases they are increased by the recumbent posture or depressing the head, by expiration, cough, and effort; and they are usually aggravated by constipation, and by indulgence in alcohol.

1. *General*.—Among the symptoms of excitement may be mentioned mental irritability; headache, slight or violent, with feelings of fullness or throbbing in the head, and vertiginous or other sensations; derangement of the special senses, such as flashes of light and noises in the ears; contraction of the pupils; sleeplessness; restlessness, startings, twitchings or slight actual convulsions; and mental excitement. The pulse is quick. There may be vomiting. The face may participate in congestions of circulatory origin. In other forms of supposed congestion, as in those which result from excessive brain-work, the face may be pale, but the nature of these cases is doubtful.

Among the symptoms of depressed brain-function are dulness of the special senses; motor weakness; mental indifference and slowness; somnolence, especially after meals; dilatation of the pupils; and infrequency of the heart's action. Consciousness may be lost suddenly, and the loss, it is commonly believed, may deepen into coma, but this is seldom absolute. As a rule there is no fever, but in children the temperature may be raised.

In the *chronic* forms of cerebral congestion, these symptoms, variously grouped and moderate in degree, continue for days, weeks, or months. Their course is marked by great variability.

In the more *acute* forms of cerebral congestion, the symptoms of muscular spasm, of mental disturbance, or of loss of consciousness, may be so predominant as to give a special character to the attack:—

a. In the *convulsive* form pain or uneasiness in the head commonly precedes the muscular spasm. The latter is usually deliberate in onset. It is said sometimes to be unilateral at first, and afterwards general, but more frequently it is general throughout. Tonic may precede clonic spasm, and the attack resemble an epileptic fit, or clonic spasm only may be present, or slight general tonic spasm. The writer has witnessed in an adult a general attack of clonic convulsions as a result of a violent attack of cough, causing extreme cyanosis.

b. The *delirious* form is seen under two aspects—(1) in old age, after emotional excitement; in this the wandering is slight, and often related distinctly to the recumbent posture, or to a low position of the head; (2) a much more violent delirium, which may be due to cerebral congestion (but is less distinct in nature), is seen sometimes after mental work or emotional excitement, or after alcoholic poisoning. It often resembles and may end in mania, but there is no pyrexia. Occasionally death results.

c. The *apoplectic* form is marked by sudden loss of consciousness, occurring commonly during effort in

plethoric persons. The unconsciousness usually lasts only a few minutes, and slight general weakness may remain for a day or two. In rare cases the loss of consciousness deepens into coma, with stertorous breathing and relaxed sphincters, and death may occur from the extension of the cerebral depression to the respiratory centres without any distinct lesion of the brain. It is doubtful if such cases should be regarded as examples of mere congestion.

d. *Vertiginous* sensations sometimes give a character to an attack. In children congestion of the brain is said to be a rare but occasional cause of convulsion or delirium. Headache and contracted pupils make up a group of symptoms resembling meningitis, but fever is rarely present, and if it exists it is slight, and the symptoms usually come on suddenly and pass away in a day or two. Death, however, sometimes occurs soon after the onset, especially in the convulsive form. The nature of such attacks is open to doubt. But children sometimes pass rapidly into a state of headache, intolerance of light, and considerable fever, often with vomiting, and recover completely after twelve or twenty-four hours. Such attacks may occur periodically and are ascribed to 'congestion of the brain.' The subjects, however, usually become liable, subsequently, to characteristic *mègrim*.

2. *Partial*.—Partial hyperæmia leads to localised symptoms of excitement or depression of function. Limited convulsion or paralysis may result. Headache may be intense and local. If nothing more than congestion is present the symptoms usually soon pass off, but such cases are extremely rare, to say the least.

PATHOLOGY.—The possibility of the variations in the quantity of blood within the brain has been denied on the ground that the cerebro-spinal circulation is carried on within a closed cavity. But the statement is untrue in the sense that is relevant to the problem at issue, since the communication of the venous sinuses of the skull, and especially of the venous plexuses within the spinal canal, with external vessels, is free, and affords a ready mechanism by which the amount of blood, even in the distant cerebral vessels, may be varied by the displacement of the cerebro-spinal fluid. This, it will also be remembered, can pass into the perivascular spaces, and can readily yield to increased intravascular pressure, and thus make room for an increase in the quantity of blood within the arteries or veins.

An excessive supply of arterial blood is, in all organs, attended with functional activity, and it is easy to understand that active congestion should result in symptoms of excitement (mental activity is often conspicuous in the subjects of aortic regurgitation, in which arterial pulsation is so vigorous). Subsequent depression of function has been accounted for by inferring compression of brain-tissue by effused serum. The reaction of exhaustion may contribute. In passive congestion the nerve-tissue is imperfectly supplied with oxygenated blood, and its functions are impaired by the presence of effete product. Hence the predominance of symptoms of depression over those of irritation.

DIAGNOSIS.—The diagnosis rests on the discovery of circulatory and other causes of cerebral congestion; on the circumstances of posture, effort, &c., under which the symptoms came on; on the

existence of concomitant congestion in other adjacent parts ; on the general character of the cerebral symptoms ; on their speedy disappearance ; and on the absence, in the adult at any rate, of material elevation of temperature. The diagnosis of the special forms of cerebral congestion from the diseases which they most resemble is considered under the head of those diseases. It may, however, be mentioned that persistent focal symptoms exclude mere congestion. On the other hand, the condition is most improbable in cases in which the various cephalic sensations of pressure, fulness, heat, &c., seem to the hypochondriacal sufferer to be certainly due to this condition. It is in these cases that it is within the power of the doctor to do great harm by encouraging the erroneous opinion.

PROGNOSIS.—The prognosis is generally immediately favourable ; but it must always be determined by the gravity of the symptoms. In a severe attack of any form of supposed congestion death may occur. The apoplectic form is most dangerous, the convulsive least so. Degenerated vessels increase the immediate risk to life. After many attacks, permanent nutritional changes in the brain often supervene.

TREATMENT.—The most important elements in the treatment of definite cerebral congestion are posture, removal of blood, purgation, cold to the head, and warm and stimulating applications to the surface. Whatever be the cause of active congestion, it is important to raise the patient's head, so that gravitation may impede the flow and aid the return of the blood. By this means alone, insomnia from this condition may often be prevented. The removal of blood is useful in extreme forms of congestion, either active or passive, especially in those forms of active congestion in which the face participates. Venesection or leeching behind the ears may be employed, according to the severity of the attack. The relief which, in such cases, follows epistaxis illustrates the benefit thus afforded. We cannot indeed induce such direct relief, but a leech may be applied behind the ear, where there are communications between the circulation inside and outside the cranium. This is not advisable in those cases in which, from over-action of brain tissue, we may assume encephalic congestion while the surface remains pale. In most forms of congestion, especially in plethoric states, purgation is useful. It removes from the blood some of its serum, and it affords immediate relief to the cerebral circulation, by causing an afflux of blood to the capacious intestinal vessels. Diuresis is also useful. Cold to the head is of most value in reflex or secondary dilatation of the cerebral vessels, as after mental work, insolation, fatigue, and in some toxæmic states. In the same class of cases, stimulation of the peripheral nerves by sinapisms, blisters, &c., applied to the neck, and even to the soles of the feet and calves, will not only draw blood to the surface, but also, by reflex influence, assist in obtaining arterial contraction. Hot applications to the limbs act in a similar manner, causing local afflux of blood and thus lessening the tendency to encephalic engorgement. Recourse may also be had to mechanical appliances for increasing the quantity of blood in the limbs. This element in treatment is most useful in active congestion. Alcohol and opium should be avoided in all forms of active congestion, but in passive congestion they may be of service. Bromide of

potassium is useful in those cases in which the congestion is produced by vaso-motor disturbance. In passive congestion from heart-disease the treatment is that of the cardiac condition, digitalis, strophanthus, and diuretics being of especial service. All persons liable to congestion of the brain should live regularly, avoid hot rooms, and attend carefully to the stomach and bowels, keeping the latter regular by an adequate daily aperient.

W. R. GOWERS.

BRAIN, Hypertrophy of.—This term is applied to an overgrowth of the interstitial tissue of the brain, the nerve-cells and fibres being often actually diminished both in size and number. The condition must be distinguished from some forms of œdema, in which the whole brain may be considerably larger than normal, and also from a normal brain of unusual size. In this so-called hypertrophy, the brain may or may not be larger than usual, but its density is considerably increased ; beyond this the naked-eye examination often shows nothing unusual. In other cases many of the convolutions are slightly raised, are quite smooth and extremely hard ; while the pia-arachnoid is detached from them with great ease. On cutting through one of these, considerable resistance is felt, the section is pale, smooth, and somewhat mottled, and the cortical striæ are obscured. In some instances small dense globular excrescences may project from the walls of the lateral ventricles. The disease is most common on the superficial parts of the brain, but any such part may be affected.

On microscopical examination of one of these portions there is seen to be a great increase in the number of neuroglia-cells, their processes are very numerous, much thickened, and form a dense network ; the nerve-cells may be fewer in number and slightly atrophied, their arrangement being most irregular, so that it is almost impossible to make out the ordinary cortical layers. The perivascular connective tissue may be also slightly increased, but as a rule the vessels themselves are unchanged. In addition to these somewhat localised aggregations of neuroglia, there is a general increase of glia throughout the organ, and this with the absence of vascular change or of secondary degeneration suffices to distinguish the condition from disseminated sclerosis and secondary change following vascular lesions.

ÆTIOLOGY AND SYMPTOMS.—The cause of this condition lies in a developmental error ; there is a tendency to undue growth of neuroglia similar to that occurring in some forms of syringomyelia, and this may be associated with a defective development of the nervous elements. The subjects of the disease frequently come of a neurotic or insane stock ; they are usually either imbeciles or epileptics, often both. Beyond this the condition does not appear to give rise to any sensory or motor disturbances, and there are therefore no certain means of recognising it during life. A. F. TREGOLD.

BRAIN, Injuries to the.—These will be considered under the headings of *concussion* and *compression*.

I. Concussion.—As the result of a violent blow or fall upon the head the brain may be severely shaken within the cranium, the elasticity of the latter also allowing of considerable squeezing or temporary compression being applied at the moment of the injury. The mere shaking of the

cerebral mass accounts for the symptoms of 'concussion' in slight cases, but it is certain that in the more severe examples of concussion there are definite organic lesions present. Of these the chief are hæmorrhages from the thin-walled blood-vessels on the surface of the brain or into the cortex. Should it happen that veins of fair size in the pia or dura mater have been torn, the resulting hæmorrhages may be so extensive as to cause actual symptoms of compression, but more commonly the extravasations are scattered widely, each being attended with localised tearing or bruising of the delicate tissues of the brain at that spot.

In the case of injuries to the head from falls or blows without perforation, certain definite parts are prone to suffer from contusion and laceration. Usually some slight evidence of injury is found immediately beneath the part of the skull upon which the blow was received, but by far the chief bruising will be at the opposite point; thus if the occiput be struck, the anterior lobes will be contused, and if one parietal eminence, the opposite temporo-sphenoidal lobe. This law, however, is greatly modified by the differing conditions under which different parts of the brain-mass are placed. In the posterior half of the skull the brain-mass is bulky, and between its hinder lobes and the cerebellum is a strong flexible membrane, well calculated to break vibrations gradually, and thus to prevent contusion. Nor are there in these regions any strongly marked bony ridges against which the brain might be forced. These conditions are reversed as regards the frontal and temporo-sphenoidal lobes, and the consequence is that while severe contusions are often seen in the latter, they are much more rare in the cerebellum and posterior two-thirds of the hemispheres. In cases of compound fracture, with tearing of the dura mater and deep depression of bone, the brain-substance may, of course, be injured at any part, but even in respect to this kind of violence the hinder regions of the skull are specially protected. Occasionally the contusion of part of the brain with its attendant hæmorrhage gives rise to localising symptoms. Thus injury to the left inferior frontal convolution may produce aphasia, to one or other part of the ascending frontal and parietal convolutions weakness of the opposite arm or leg or of some group of muscles in either limb (see Fig. 1, in BRAIN, Tumours of). Such paralytic symptoms following concussion are as a rule wholly recovered from after the lapse of a few days or weeks. This is, unfortunately, not always the case if one or other of the cranial nerves have been torn across or otherwise damaged at the time of the injury to the head. The olfactory lobes, the optic nerves, and occasionally the third, fourth, or sixth nerves have been known to be completely and permanently paralysed as the result of a severe fall on the head, and this is most likely to occur if a fracture of the cranial base has passed through the foramina at which the nerves involved emerge. With a fracture of the middle fossa either the facial nerve in the aquæductus Fallopii or the auditory nerve is often damaged. These injuries to cranial nerves are so important from the point of view of prognosis (since they are likely to prove permanent in their results, while the recovery from the accompanying concussion of the brain is in most cases complete) that they should be carefully sought for in every case. Their occurrence is to be suspected whenever

the parts supplied by a cranial nerve are completely paralysed without accompanying symptoms indicative of severe lesion of the brain-mass. Here it should be noted that even small traumatic hæmorrhages into certain parts of the brain have an importance quite out of proportion to their size, owing to their special site. Thus in some cases of concussion which have proved fatal, punctiform hæmorrhages have been discovered in the pons Varolii and floor of the fourth ventricle, where the cardiac and respiratory centres are placed. In such examples of fatal concussion the whole surface of the brain is found to be intensely congested at the time of the *post-mortem* examination. We may safely assume that in all severe cases of concussion which do not prove fatal, i.e. the great majority, there is immediately after the injury a period of shock and depression of the heart's action in which the brain is comparatively bloodless, and its functions therefore suspended, but that this stage is succeeded by a reaction in which all the cerebral arteries dilate and the whole brain becomes engorged with blood, especially the parts situated near the surface (see BRAIN, Congestion of). This high degree of congestion no doubt explains the severe headache so constantly met with, and may also account for the occasional occurrence of general epileptiform convulsions. The latter are more frequently met with in children than in adults, and may be so severe and so frequent as to threaten a fatal issue. It is important to remember that the administration of a general anæsthetic (ether or chloroform) will probably put an end to such dangerous convulsions.

SYMPTOMS.—In slight cases there is no real unconsciousness, merely a temporary giddiness and loss of brain-power, succeeded by headache with perhaps some strange or inconsequent action. In the gravest cases there may be found prolonged insensibility, and then the periods of reaction and of gradual recovery are of equally long duration. The symptoms may be conveniently grouped into four stages. The first stage is that of *collapse*, in which there is more or less complete loss of consciousness, pallor of the skin, and coldness of the extremities, due to feebleness of the heart's action, the respiration showing a similar depression and being shallow and feeble. In fact, the condition and appearance of a man badly concussed may closely simulate death, and the mistake has frequently been made on the field of battle and elsewhere. The state of the pupils is neither constant nor of any practical importance, so long as fixed mydriasis be not present, in which case compression of, or very severe damage to, the brain is to be suspected. It is of great importance, in this stage, to establish the absence of all forms of paralysis. If any asymmetrical symptoms are present, the case is more than concussion. The second stage is that of *Reaction*. After the collapse has lasted some little time (half an hour to two, three, or more hours), it begins to pass off. The patient moans, manifests discomfort, turns on his side, and draws his knees up. Very often at this period sickness occurs, and it is almost invariable if the patient's stomach was full at the time of the accident. Consciousness is now usually restored, and, by rousing, the patient may be induced to speak and to tell his name. Gradually during a period of some hours the case passes into the third stage of *drowsiness*. The pulse is now relaxed and full, the skin warm or even hot, the

face somewhat flushed, and the pupils contracted. The patient can only be awakened with difficulty. It is, however, always possible to awaken him, and he usually rouses himself to the calls of nature. His answers to questions are often confused or irrelevant, and there may be extreme irritability on being roused, sometimes even delirium. Very commonly the pulse is irregular, especially if the patient be young. At this stage again care must be taken to ascertain whether there are any asymmetrical symptoms—any weakness of a limb, of one side of the face, or of any single eye-muscle. If the patient passes his urine or fæces into the bed, or if there is any long-continued retention of urine, the case is not one of ordinary concussion. The sleepy stage may last for a day or for a week, and it is in severe cases so well-marked that the patient's eyelids may be held open and the pupils examined without awaking him. During this stage the measures of treatment called for are spare diet, purgation, cold to the head, and quiet. It may be necessary to use the nasal tube for feeding. When the sleepy stage passes off, the patient is left weary, torpid, unfit for mental effort, and often with distressing headache. This is the final stage of gradual *convalescence*, and its duration, before complete return to health ensues, varies greatly. Loss of memory, irritability of temper, and unusual susceptibility to alcoholic stimulation, are three results of severe concussion, which are the most constant and the most persistent. Loss of sexual power, homicidal or suicidal impulses, epileptiform fits, and true insanity are some of the graver and less common results of injuries to the head. But however long the patient may be incapacitated from work and however hopeless the condition may appear to be, a favourable prognosis may safely be given in the great majority of cases. Recovery from a condition of complete mental torpor, delirium, or even mania lasting several weeks has often been observed. On the other hand death may occur during the first stage, but in these fatal cases there is as a rule extensive damage to the brain associated with fracture of the skull. It is most important to remember that compression of the brain from meningeal hæmorrhage may supervene on concussion, and its symptoms be grafted on to those of the latter. The following symptoms should arouse suspicion:—(1) relapse into *complete* unconsciousness, from which the strongest stimuli will not rouse the patient; (2) stertor of breathing; (3) flushing of the surface, with a steadily rising temperature; (4) convulsive movements of the limbs followed by their paralysis; (5) fixed wide dilatation of the pupils (mydriasis).

TREATMENT.—During the stage of collapse there is but little required beyond assisting the circulation by the application of hot blankets to the skin. If the pulse be extremely feeble a subcutaneous injection of strychnine (gr. $\frac{3}{16}$) is better than the administration of alcohol.

After reaction has set in, a free action of the bowels should be obtained by giving 5–8 grains of calomel, followed by a saline draught. The room or bed should be darkened, and during the sleepy stage the patient should only be roused for purposes of feeding. The diet for some days should consist principally of fluids. Care should be taken that the bladder does not become over-distended, a point somewhat apt to be overlooked. To relieve

subsequent headache, and to diminish the irritability, often present in the third stage, cold to the head is most useful, and is best applied by means of iced-water running through Leiter's tubes, or pounded ice in an india-rubber cap, secured beneath the chin. Antipyrine, bromide of potassium, and sulphonal are sedatives worthy of trial in cases of very marked reaction or delirium. Constant watching may be necessary for many days or weeks after severe concussion, and it must be remembered that such cases are liable to violent outbursts if allowed to indulge in alcohol. The only safe course is total abstinence for long afterwards.

II. Compression.—The brain is compressed in the pathological sense whenever its structure is so squeezed that its functions are in any degree interfered with. This squeezing may be effected either by the effusion of blood within the skull, the growth of a tumour, the accumulation of pus or serum, or lastly by the depression of some large portion of the bony parietes. As already noted, a temporary compression occurs in many cases of ordinary concussion with or without fracture, but the general belief that depressed fractures are frequently the cause of compression is probably a mistake. In such cases the fragment displaced is rarely of sufficient size to cause serious compression of the contents of the skull, and the symptoms usually supposed to indicate that state are really due in most cases to laceration and contusion, or to subsequent inflammation.

By far the best examples of uncomplicated compression of brain are supplied to us by the not very infrequent cases in which one middle meningeal artery is injured, and a large blood-clot is effused between the dura mater and bone. It is from observation of cases of this kind that the assertion is justified, that a very considerable intrusion into the skull may be permitted without the production of any symptoms. No doubt the suddenness or otherwise has much to do with the results, but there seems good reason to believe that, as a rule, the brain will easily accommodate itself to quantities not exceeding an ounce or two, and that usually as large a quantity as four or five ounces is required to cause death. It is very rarely indeed that a depression of bone in the least approaches such an extent of intrusion as this, and the majority of such cases are, as regards the amount of possible squeezing, quite trivial. The manner in which the accommodation is effected is by the removal of the fluid contents of the skull—first the subarachnoid fluid, and secondly the blood. Of the blood-vessels the veins and venous sinuses are probably emptied first, and lastly the arteries and capillaries. A brain in a state of strong compression is an exsanguine brain. In this respect, the brain in the last stage of compression differs very much from that in cases of insensibility from concussion or contusion. It by no means follows that because the brain is pale the face should be pale also; but it is perhaps usually the case that extreme compression so much enfeebles the heart's action that the pulse is weak or flickering, the respiration shallow and irregular, and the skin pale and cold. That stertorous breathing, a laboured pulse, and a suffused and dusky countenance are (as according to the classical description) symptoms of compression, is probably for the majority of cases a mistake. Such a group much more frequently denotes laceration, contusion, or central extravasation. Nor is it true that hemiplegia, excepting of the most transitory kind, is often due to compression.

A clot of blood poured out over one hemisphere may, if rapidly effused, produce for a while weakness of the opposite limbs, but the brain-mass is soft enough to allow of considerable yielding, and in the course of a few hours the effects of the displacement will have become general, and not local. In a case recorded by the writers in which a *post-mortem* examination, some weeks later, proved the presence of a large blood-clot, there had been partial hemiplegia without unconsciousness at first, but on the next day all trace of it had disappeared, and it never returned. In many of the cases of bleeding between the dura mater and the bone, from the meningeal artery, the hæmorrhage takes place on several different occasions, with, it may be, intervals of a day or two, much as is often observed in wounds of arteries, such as the palmar arch. Thus, the observer is able to appreciate the symptoms caused by different degrees of compression, and further proof is afforded that, if the intruded quantity be but moderate, the brain bears it without obvious inconvenience. Often at the autopsy it is quite easy to distinguish clots of very different dates, and to feel sure that the original one was of considerable size. It is clear then that in speaking of the symptoms of compression we must allow for differences in amount of the compressing substance, and also for differences in the rapidity or suddenness of its application.

When compression is produced suddenly, as by a large fragment of bone driven down, the case is almost invariably complicated by contusion. If paralysis or even insensibility be present, it is usually impossible to say to which lesion they are really due. We may, however, hold it almost certain, from what we know of other cases, that the effects in such would be a temporary hemiplegia, with symptoms of shock, if the depression were but moderate; and insensibility, probably soon followed by death, if the depression were very great. The cases in which depression of bone has alone been sufficient to produce long-continued compression with insensibility are possibly somewhat apocryphal. It is possible that compression under such circumstances might be attended by stertor and laboured pulse, but it is possible also that the pulse might be extremely feeble, the countenance pale, and inspiratory efforts weak and irregular.

The depression of bone is perhaps the only condition which can be supposed capable of producing compression suddenly. When blood is poured out from a ruptured artery, the symptoms come on rather gradually. The patient complains perhaps of headache, and then becomes more or less confused in manner, his gait is unsteady, and the limbs on the side opposite to the injury show special weakness. Vomiting may occur and the weakened limbs may twitch, and unless, as is often the case, the intracranial bleeding stops, these symptoms are soon lost in a state of complete insensibility, with pale face, feeble pulse, and symptoms of shock. Convulsions may now occur, and death often supervenes very quickly. In such a case the whole course of the symptoms may occupy less than an hour. The surgeon ought, of course, to trephine and let out the blood, and he must be prompt, or his patient may die during his preparations. More commonly this rapid termination occurs unexpectedly after one or more previous attacks of temporary head-symptoms, and the patient may have appeared quite well in the intervals. In cases in which the symptoms progress without interruption, their rapi-

dity, no doubt, depends upon the size of the vessel ruptured. Certain special symptoms will also depend upon the precise position taken up by the clot, which may chance to press upon special nerve-trunks as well as upon the brain-mass. In ordinary cases the clot is beneath the squamous bone and the lower part of the parietal, and passes downwards into the middle fossa. In the latter region it may press upon the nerves going to the sphenoidal fissure; and it is of importance for the surgeon to know that dilatation of the pupil on the affected side is often produced. This important symptom is probably due to pressure upon the third nerve.

There is yet another class of cases of compression in which that condition is produced by the slow accumulation of the products of inflammation within the skull. Much will depend, as regards special symptoms, upon the position of the abscess, either within or without the brain. If in the substance of the brain, it must more or less disorganise its structure, and thus cause symptoms due to laceration as well as compression. Under such conditions some degree of hemiplegia, with, probably, preceding spasms of limbs, can scarcely fail to be present. Now and then cases occur in which an irregular sinus leads into an abscess-cavity in the brain, and this sinus being sometimes free and sometimes blocked, the surgeon has repeated opportunity of estimating the effects of filling of the cavity. In such cases, headache, stupor, unilateral twitching of limbs, partial hemiplegia, with perhaps vomiting and, it may be, general convulsions, are the symptoms to be expected. The position of the abscess as regards different regions of the brain is also of much importance, but its discussion cannot be entered upon in any detail here, and it obviously concerns rather disorganisation of structure than simple compression. In one case, seen by the writers, epileptic convulsions, repeated so frequently as to be almost continuous and accompanied by complete unconsciousness, were cured by trephining and letting out a quantity of clear fluid which had existed under high tension in the substance of the brain. The condition had apparently resulted from old injury to the head.

When a large accumulation of pus takes place between the bone and dura mater, the symptoms produced are much the same as those caused by blood-clot. We must make, however, much allowance for the fact that these cases are almost always attended by meningitis, and thus the symptoms of compression are masked by those due to inflammation. Chronic abscess under the bone without arachnitis may occur now and then in syphilitic and other disease of the cranial bones, but such cases are very rare. They will differ from those of hæmorrhage in that the symptoms are always produced very slowly. The writers once had the opportunity of watching such a case, in which the patient died of compression, very gradually produced by an increasing collection of pus between the bone and dura mater, and without any complication. The chief symptom was constant wearing headache, which prevented sleep. The man was pale and feeble, but not paralysed in any part, excepting that both eyelids drooped. He was rational, but spoke slowly, as if in a state of partial stupor. During the last two or three days of life he had convulsions; and finally, for twenty-four hours, he was in a state of increasing insensibility.

DIAGNOSIS.—It will be seen from what has been said above that the diagnosis of compression by symptoms is exceedingly difficult, and that the utmost use must be made of the history in each individual case. In those of compression by blood after injury to a meningeal artery, there is almost always the fact that the patient, between the date of the injury and the supervention of symptoms, had an interval during which there appeared to be little the matter, or in which the ordinary symptoms of concussion were present. This history is, if the symptoms have developed rapidly and without the signs of inflammation, by itself conclusive for diagnosis. The pupils become widely dilated, and do not respond to light. The pupil on the side of the hæmorrhage is first and most affected. The limbs become first convulsed and then paralysed, those on the opposite side to the hæmorrhage are first and most affected. Under such conditions trephining ought to be at once resorted to. A trephine of at least one inch diameter should be applied over the anterior branch of the middle meningeal artery (on the side indicated by the above rules). If no extravasation is found between the dura mater and bone, the former should be incised and any clot on the surface of the brain gently removed. In rare cases the posterior branch of the meningeal is torn, necessitating the opening being made through the posterior part of the parietal bone.

The diagnosis of abscess in the brain-substance is discussed elsewhere. See BRAIN AND MENINGES, Septic Diseases of.

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BRAIN, Laceration of.—See BRAIN, Injuries of.

BRAIN, Malformations of.—See MONSTROSITIES and MALFORMATIONS.

BRAIN, Malignant Diseases of.—See BRAIN, Tumours and New Growths of.

BRAIN, Membranes or Meninges of, Diseases of.—See MENINGES, Diseases of.

BRAIN, Morbid Growths of.—See BRAIN, Tumours and New Growths of.

BRAIN, Œdema of.—**DEFINITION.**—Infiltration of the brain and pia mater with serum.

ÆTIOLOGY AND PATHOLOGY.—In chronic maladies attended with general œdema, especially Bright's disease, there is an increased amount of fluid around the brain—in the meshes of the pia mater and between the convolutions—and also in the ventricles. Occasionally the cerebral substance is infiltrated; but this is uncommon, and it probably occurs only when there is some change in the cerebral structure. The perivascular canals normally afford a ready means of escape for effused serum, and in Bright's disease, at least, the substance of the brain is often remarkably firm, so as to present a contrast to the condition of other organs. But apart from any general disease, all spaces that result from a diminution in the volume of the structures within the skull are filled by serum. Thus in senile atrophy of the brain, the space between the sunken convolutions is occupied by serum, and the ventricles contain an excess of fluid. The brain-tissue may also appear to contain more fluid than usual, in consequence of the presence of serum in the enlarged

perivascular canals. Such effusion is also met with in those forms of insanity in which there are degenerative changes in the brain with a diminution in its volume. It is seen also in cases of rapid course, in which functional excitement is attended with some vascular disturbance. Conditions of hyperæmia and inflammation are attended with effusion of serum in the brain, as in other organs, and it is in cases of inflammation that the greatest amount of œdema is met with, but this condition is not included in the common use of the name. In passive congestion, however, such as results from heart-disease, enough serum may escape from the distended vessels to give rise to a condition of œdema; but it is probable that when the excess is more than trifling, room is made for it by wasting of the nerve-elements, the result of the continued pressure to which they are exposed.

When the degree of œdema is considerable, the cerebral substance may be enlarged, the convolutions being flattened; and the tissue is lessened in consistence owing to the infiltration of the cerebral tissue by the liquid, and the separation by it of the nerve-elements. A microscopical section shows empty round and oval spaces between the cells and fibres, limited by delicate tracts of the separated neuroglial tissue. Similar softening is seen in the neighbourhood of effusions of fluid into the ventricles; the brain-tissue, for a depth of some lines from the ependyma, being softened to a pulpy consistence. *Post-mortem* imbibition always increases the apparent amount of the œdema and the degree of the softening, because the nerve-elements quickly begin to break up after death. In simple œdema the tissue remains pale, and the grey substance may be paler than usual.

SYMPTOMS.—Little is known of the symptoms of œdema of the brain. The pathological state is usually secondary to some other condition, the symptoms of which mask any that the œdema itself may possibly cause. General œdema seems attended by slow diminution of mental power and motor force, which may be in part due to it, but has other possible causes in most cases. The effusion of serum in congestion, and consequent pressure on the nerve-elements, has been considered as the cause of the symptoms of depression common in that condition. Cases occasionally occur in which effusion of serum into the ventricles and the pia mater is the only *post-mortem* condition to be found after an apoplectic seizure, and such cases are often spoken of as instances of *serous apoplexy*. See APOPLEXY, CEREBRAL.

TREATMENT.—The treatment of cerebral œdema is usually secondary to the condition, commonly conspicuous enough, which is its cause—Bright's disease, passive cerebral congestion, &c. If œdema be suspected where no causal indication for treatment exists, purgatives and diuretics, with iron if there be debility, are the remedies most likely to be of service. But where a diagnosis is speculative, treatment necessarily lacks a confident basis.

The effusion of fluid into the ventricles is described under HYDROCEPHALUS.

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BRAIN, Sclerosis of.—See SPINAL CORD, Special Diseases of: Multiple Sclerosis.

BRAIN AND MENINGES, Septic Diseases of.—It is impossible to draw any line of demarcation between a septic inflammation, whether

general or localised, originating on the surface of the brain internal to the dura mater, and a similar inflammation originating in the substance of the brain. In all cases these inflammatory affections are caused by the introduction of septic micro-organisms. The same micro-organism may cause an abscess to originate on the surface, or may commence its action deep in the cerebral mass. The same predisposing cause, e.g. chronic purulent otitis media, may cause an inflammation on the surface, diffused or localised, with or without the formation of pus. It may also be the predisposing cause of a deep cerebral abscess—indeed, the two conditions may be and often are present at the same time.

Pathologically, no line of demarcation can be drawn, since the ordinary form of diffused septic inflammation affecting the arachnoid and pia mater or *septic lepto-meningitis* is always associated with more or less inflammation of the tissue of the brain, commencing on the surface where the vessels enter from the dura mater, and gradually spreading inwards so that lepto-meningitis and encephalitis are always present together. In a similar way, if the inflammation is localised an abscess may form on the surface, invading to a greater or less extent the brain-substance.

For practical clinical work it is most important that these points should be thoroughly appreciated. It is disappointing at first to think that the differential diagnosis between an abscess on the surface and an abscess deep in the substance of the brain is very uncertain, yet experience shows that even moderate certitude is impossible.

ÆTIOLOGY AND PATHOLOGY.—It is obvious that, in certain injuries of the head and brain, septic micro-organisms enter the meninges, or substance of the brain, or both, at the moment the injury is inflicted. These may give rise to primary encephalitis or meningitis. In certain cases of injury and in some cases of disease the organisms reach the area of their operations in an indirect, and sometimes by an unknown route. Thus are produced secondary encephalitis and meningitis.

The organisms may reach the brain and meninges by several routes. The direction of their line of march is sometimes clearly evident; in other cases the route is rather hypothetical. Where the organisms are introduced as the result of injury or of disease, they generally take one of the following routes: (1) By direct extension from the region of the scalp or skull, or both. The dura mater is a good barrier against septic infection of the meninges, but in some cases it is unable to resist the growth of germs, and so we may get either local lepto-meningo-encephalitis, or an abscess upon or in the brain, or a general lepto-meningo-encephalitis. (2) By the venous system: infection may take place by progressive thrombosis, or by a process known as 'reflex propagation', owing to a thrombus causing a reversal of the blood-stream, which may sweep organisms towards a certain region. (3) By the perivascular lymphatic sheaths of the arteries. (4) By an anastomosing lymphatic space. Thus germs may pass from the galea to the subdural space, as in erysipelas of the scalp. (5) By perineural lymphatics, such as those communicating with the sub-arachnoid space. (6) By metastatic infection, the primary septic focus being in the head or some other part of the body, i.e. ordinary pyæmic infection.

I. Diffused Septic Inflammation (*septic meningo-encephalitis*).

PATHOLOGY.—The earliest change is intense hyperæmia with punctiform hæmorrhages in the pia mater, the vascularity of which becomes very evident. Very soon the inflammatory exudation is poured into the subarachnoid space and increases the amount of subarachnoid fluid and consequently alters the character of the cerebro-spinal fluid. The exudation, clear at first, soon becomes red, and finally turbid owing to the presence of leucocytes. This condition may increase so that there is definite pus and flakes of lymph are deposited upon the pia mater and arachnoid. The inflammatory process extends into the cortex of the brain, which becomes softer than normal and oedematous. Under the microscope the inflammatory process can be seen extending from the pia mater into the cortex along the periarterial lymphatic sheaths. In injuries of the skull the inflammation usually commences in the region of the fracture, either on the vault of the skull or on the base; so clinically two forms of lepto-meningitis are found, viz. of the convexity and of the base. In disease the basal form is by far the more common, owing to the great frequency of complications from chronic suppurative otitis media.

SYMPTOMS.—The onset is usually indistinguishable from that of any acute septic disease—headache, fever, shivering, or even rigors. This stage lasts from a few hours to 24 hours.

Stage of irritation.—The patient becomes very restless and often delirious. The pain in the head increases and may become distressing. There is marked photophobia and intolerance of noise. The pupils are equal and contracted, reacting to light. Optic neuritis usually commences in this stage. Convulsions are not constant in their appearance, and when present vary both in degree and distribution. As the convulsions recur the muscles concerned become progressively weaker; the patient becomes more and more drowsy and finally insensible. During this period the pupils begin to dilate, as a rule unequally, and their reaction to light is slight or absent.

Stage of paralysis.—Convulsions, if present, cease and the muscles become paralysed. The paralysis sets in even if no convulsions have occurred, and may vary in extent from a small group of muscles to hemiplegia, or complete muscular paralysis. The patient lies insensible and motionless and the respirations are accompanied with marked stertor. This comatose condition varies in duration from one to two or three days, and the patient usually dies within a week of the onset of the disease. The pupils become widely dilated on both sides and are insensible to light. The pulse is quick and bounding at first, and as the compression increases it becomes slower, only to increase again and gradually weaken during the final stage. The respirations are regular and deep at first, become rapid and superficial in the final stage, and cease before the heart stops. The temperature as a rule is raised, varying from 101° to 104° and rising to 106° F. towards death. Sometimes it is scarcely raised at all, and may even be subnormal.

DIAGNOSIS.—At the onset this is impossible, unless there are reasons for suspecting its occurrence. For the differential diagnosis from the effects of contusions or lacerations of the brain, see BRAIN, Injuries of. Differences in the symptoms may help

us in distinguishing meningitis of the convexity from meningitis of the base. In *basal meningitis* (a) retraction of the head and rigidity of its extensor muscles come on early, and are fairly characteristic; (b) optic neuritis is more constant in its presence, is more intense, and comes on earlier; (c) squint from spasm, or paralysis of one or more of the ocular muscles, is usually an early and characteristic symptom. In *convexity-meningitis* (a) convulsions come on earlier, and are more frequent; (b) the convulsions are more likely to be localised at first to definite groups of muscles.

PROGNOSIS AND TREATMENT.—It is probable that no case of diffused septic meningo-encephalitis has recovered. No treatment, medical or surgical, appears to have any definite influence upon the course of the disease. Draining the subarachnoid space has been tried several times without success.

II. Cerebral Abscess.—**ÆTIOLOGY.**—All abscesses of the brain are caused by micro-organisms, but in many cases it is impossible to state the exact path by which the germs reach the particular region of brain-substance affected. This question has already been discussed at the commencement of the article. The causes of cerebral abscess are: (1) *Chronic purulent otitis media* is by far the most common cause, and the resulting abscess is situated generally either in the temporo-sphenoidal lobe or in the cerebellum. An abscess is rather more frequent on the right side than on the left. Abscess in the cerebrum from this cause is about four times as frequent as in the cerebellum. (2) *Injuries of the head* may be followed by abscess of the brain, which is most common in the frontal and parietal regions. (3) *Disease of the cranial bones*, usually syphilitic or tubercular, is a rather uncommon cause. Necrosis of the upper jaw from phosphorus-poisoning has been known to cause cerebral abscess. (4) *Pyæmic* abscesses may occur in the brain and are usually multiple. Empyema and bronchiectasis form the most common primary foci.

PATHOLOGY.—The abscess may be a very acute one, with no distinguishing wall demarcating it from the surrounding cerebral substance. These are generally caused by septic infection from a punctured wound. As a rule the ordinary cerebral abscess, e.g. the one secondary to chronic purulent otitis media, forms gradually and has a very definite wall or capsule which definitely separates it from the surrounding brain-tissue. In such cases the pus often smells offensively. The abscess may remain in a quiescent state for quite a long time and may then suddenly increase in size, causing rapidly progressing symptoms. In some cases the abscess bursts into the ventricles of the brain, or on to the surface, causing a suppurative lepto-meningitis. In rare cases the pus may be discharged to the exterior, either into the ear by perforating the tegmen tympani or through an erosion in the temporal bone.

SYMPTOMS.—(1) *Acute cerebral abscess.*—The symptoms of this are sometimes very similar to those of meningitis; in fact lepto-meningitis is usually present at the same time. Intense headache with high temperature associated with rigors is rapidly followed by symptoms of cerebral compression, and the patient dies in a comatose condition within from one to three weeks of the onset.

(2) *Chronic cerebral abscess.*—In this case there are well-recognised general symptoms, caused chiefly by the increased cerebral pressure: they are often present in conditions causing such pressure apart from the abscess. These symptoms are:—

Persistent headache, often most distressing and limited to one side of the head, soon accompanied by drowsiness. The onset of optic neuritis is usually early and should be carefully looked for. It may be absent altogether or develop late in the progress of the disease. The temperature is characteristically subnormal, with an occasional sudden rise to 104°, often accompanied by a rigor. Vomiting is generally present.

In addition to these general symptoms there may be others which may aid us in localising the abscess. Such localising symptoms are not always present, and in these cases the patient may die with the general symptoms only. The local symptoms depend upon the situation of the abscess.

(a) *Abscess in the temporo-sphenoidal lobe*, by exerting pressure upon the internal capsule, may cause paresis or paralysis of the whole or a part of the opposite side of the body. If on the left side it may lead to disturbance of speech.

(b) *Abscess in the cerebellum* is usually accompanied by very intense optic neuritis, and the vomiting is usually persistent and distressing. In the early stage inco-ordination of the patient's gait is characteristic. Later on retraction of the head and symptoms indicating unusually severe pressure upon the medulla oblongata, such as slow pulse and respiration, are usually present. Cases have been recorded in which there was paralysis of the muscles of the same side of the body as the abscess in the cerebellum.

(c) *Abscess in the sensori-motor area* is shown by the presence of convulsions, or of localised paralysis according to the exact situation of the lesion.

DIAGNOSIS.—The diagnosis of abscess of the brain is often a matter of extreme difficulty. On the one hand we have to distinguish it from the other septic diseases of the brain and its meninges, and, on the other, from tumours of the brain, especially in the case of the chronic abscesses which may closely resemble the former in their clinical features. In addition to this we have to bear in mind the possibility of phlebitis of the lateral sinus being confused with cerebral abscess: indeed, it is occasionally present at the same time. *Headache* and *vomiting* vary so much in character and in degree that they often afford but little help in the diagnosis, e.g. a very localised headache may be present both in the case of abscess of the brain and in thrombosis of the lateral sinus; on the other hand, in both of these conditions the headache may be quite diffused. *Vomiting*, too, aids us but little in the diagnosis excepting when its unusual violence leads us to suspect an abscess in the cerebellum. *Optic Neuritis* is never present in uncomplicated phlebitis of the lateral sinus. It is usually present in abscess of the brain and can be looked upon as a most important diagnostic symptom. This symptom is usually present in meningo-encephalitis, but the patient usually dies before the condition becomes at all marked. The pulse when very slow is in favour of a cerebral abscess; if very rapid and very weak, phlebitis of the sinus is more probable. A subnormal tempera-

ture is in favour of abscess of the brain, whereas a markedly remittent one rather indicates sinus-thrombosis. As far as these general symptoms are concerned, they aid us but little, if at all, in diagnosing between a marked cerebral abscess and a tumour of the brain. To diagnose between these two conditions it is most important to look for any predisposing cause, such as a previous injury or the existence of chronic suppuration in the neighbourhood of the cranium, whether in the ear, nose, or other region.

PROGNOSIS.—If the pus be not evacuated the prognosis is most serious, nearly every case ending fatally. With prompt surgical interference the results are extraordinarily good, e.g. MacEwen reports 18 recoveries out of 19 cases.

TREATMENT.—This is of course purely surgical. As soon as the presence of pus is suspected an attempt should be made to evacuate it. When the situation of the pus is indicated with reasonable certainty, the opening in the skull should be made as nearly as possible directly over the abscess. When in the temporo-sphenoidal lobe, the pus is usually found in that part of the lobe adjacent to the tegmen tympani. The best spot for the centre of the opening in the bone is a point a quarter of an inch behind, and three quarters of an inch above, the centre of the external auditory meatus. When in the cerebellum, the centre of the opening should be one and a half inches behind, and a quarter of an inch below, the centre of the external auditory meatus. When the abscess is thought to be in other parts of the brain an opening should be made over the most likely spot. In cutting a flap of scalp one must bear in mind that it may be necessary to enlarge considerably the opening in the bone some distance from the original one; hence the scalp-flap should be of sufficient size to allow for such a contingency. When the presence of pus in the brain is fairly certain, but when there are no indications concerning its position, the operator must be prepared to search in every likely spot. Fortunately in the great majority of cases the pus can be reached by one scalp flap and one opening in the bone. The operation performed by the writer is as follows: A suitable semicircular scalp-flap having been made, the bone is perforated by a trephine-drill or other suitable instrument at a spot one and a quarter inches behind and a quarter of an inch above the centre of the external auditory meatus. By this means the lateral sinus, just as it begins its downward turn, is exposed together with the dura mater above and below it. By enlarging the bone opening upwards, so as to obtain sufficient room above the lateral sinus, and by incising the dura mater the temporo-sphenoidal lobe can be thoroughly explored with a trocar. In a similar way, by enlarging the opening in the bone downwards, the cerebellum can be equally well explored. If no pus is found in these situations, it is quite likely that the inflammation is a diffused one, i.e. a meningitis. In these cases the pressure can be easily reduced temporarily by inserting a trocar inwards and slightly upwards immediately above the lateral sinus so as to enter the descending cornu of the dilated ventricles. The writer has left a drainage tube in the ventricles in such cases, but with no permanent benefit. The condition of the lateral sinus can readily be ascertained during the removal of the bone. The mastoid emissary vein may be seen. If this is thrombosed it will

generally be found that the sinus is plugged also. After exposing the sinus any doubt on this score can be dispelled by inserting a small trocar into it. The operation can be completed in thirty minutes or less, and the operator will seldom fail in finding pus, if any be present in that region.

HENRY PERCY DEAN.

BRAIN, Softening of.—**DEFINITION.**—A pathological state of brain-tissue, attended by diminished consistence; usually local; and indicated, during life, by mental, motor, and sensory symptoms, which vary according to the seat of the lesion. It is produced rapidly in the vast majority of cases, and is then almost always dependent on vascular obstruction. Very rarely it is chronic in development, and the nature of the actual process is not known. The following description applies to the acute form.

ÆTIOLOGY.—Acute softening of the brain, occurring during life, and not due to a traumatic cause, is referable to one of two conditions, inflammation or vascular obstruction. Most cases were formerly thought to be due to inflammation; but it is now known that very few are. Inflammatory softening is described in another article (*see* BRAIN AND MENINGES, Septic Diseases of). The vascular obstruction, which is the usual cause of softening, may be arterial or, rarely, capillary. Venous obstruction also causes softening, but the loss of consistence does not involve the whole of the tissue of the part, and enough remains to permit an ultimate restoration of firm tissue and even the production of induration. The arterial obstruction may be due to thrombosis, or to embolism (*see* BRAIN, Vessels of, Diseases of; EMBOLISM; THROMBOSIS). The common concomitant conditions are—in thrombosis, arterial disease; in embolism, valvular disease of the heart; and as predisposing conditions we usually find—in cases of thrombosis, advanced age, Bright's disease, chronic alcoholism, gout, or especially in earlier life, syphilis;—in cases of embolism, acute rheumatism, chorea, or scarlet fever. Senile vascular degeneration is the most common cause of thrombotic softening, and hence the disease is met with most frequently in the old. In rare cases, thrombosis occurs without disease of the vessels, from a state of the blood alone. It is probable that this is sometimes a cause of hemiplegia coming on a few days after child-birth, when there seems to be a physiological increase in the coagulability of the blood. Embolism, due to valvular disease of the heart, and thrombosis due to syphilitic disease, are the most frequent causes of acute local softening in the young and middle-aged.

ANATOMICAL CHARACTERS.—The characteristic feature of cerebral softening is diminished consistence. This may, however, arise from either *ante-mortem* or *post-mortem* changes. In each case the diminished consistence depends on the breaking-up of the material of which the nerve-elements are composed into globules and granules, and the separation of these by an increased quantity of fluid. Thus the continuous structures of which the brain consists are broken up into disconnected fragments, and the consistence of the tissue is accordingly diminished. In *post-mortem* softening there is nothing more. The globules of myelin are often large, and the separating fluid abundant. The softened tissue has the tint of the normal

cerebral substance. The process is the result of the imbibition of fluid from some collection of serum, in the ventricles or elsewhere, and occurs in the greatest degree in the immediate vicinity of this. In *ante-mortem* softening there are, in addition, certain changes in the tissue-elements. The process of segmentation of myelin results in the formation of finer granules. These are in part aggregated into round or oval masses of globules and granules, sometimes contained within a distinct cell-wall. Some of these bodies may arise by simple aggregation, others by the aggregation of products of nerve-degeneration within cellular elements of various kinds. The walls of vessels in the softened area also present fatty degeneration. No further change may exist, and the area affected may present simply a diminution of consistence, its colour remaining unchanged. It is then called *white* or *grey softening*. Very frequently, however, in the part thus diseased, distension of capillaries with blood occurs, most considerably in the periphery, in consequence of the arrest of circulation and strain on the vessels, and blood is actually effused, chiefly by rupture of capillaries, in trifling degree perhaps by diapedesis of corpuscles. In proportion to the amount of blood mingled with the softened brain-tissue, the colour of the affected area is changed, and thus *red softening* is produced when the amount of blood is considerable. In the change which results from closure of a vein, the tissue is crammed with punctiform extravasations, and a purplish colour results; but, as already stated, there is not the general diminution of consistence that brings the alteration into the category of 'softenings.' After a time, the blood effused in red softening degenerates, its tint becomes altered to yellow or orange, and *yellow softening* is found. Ultimately, it is said, the colour, if at first moderate, may be removed and white softening result, but the pigment that gives rise to the yellow tint may remain for many years.

Red softening is found chiefly in the grey substance, where the vessels are numerous, especially in the cortex and central ganglia. The tint varies; the red colour is usually punctiform, or mingled with yellow and white. According to the amount of effusion of serum and blood there is swelling, and the diseased area may project above the cut surface. Inflammatory changes result from the vascular distension, and in proportion to these, increase in the nuclei of the neuroglia is found, especially at the circumferential portions. From this cause, and from the migration of white-corpuscles, pus-like cells appear among the products of degeneration in varying quantity. The vessels are dilated, and may present a moniliform appearance. Their perivascular sheaths are often distended with blood.

Yellow softening results from red softening, by degenerative changes in the blood effused attended by a change in its pigment. It has a similar seat, being frequently met with in the convolutions, where it constitutes the *plaques jaunes* of the French. Its consistence is usually slight, its aspect granular. The colour depends on the presence of minute pigment-granules, diffused colouring matter, and hæmatoidin-crystals.

White softening has the tint of the normal cerebral substance. In consistence it varies: it may be only a little below that of the cerebral substance, or it may be diffuent. Its aspect is

uniform, or white flakes are scattered through it. The limits are usually gradual. Under the microscope it presents the detritus of nerve-elements and connective tissue, and, ultimately, corpora amylacea. White softening is chiefly found in the white substance of the hemispheres. It occasionally has a gangrenous odour, and then may be found in the white or in the grey substance; probably this form results from the obstruction of capillaries by septic material. It is theoretically probable that when softening is white from the first, the capillaries are the seat of the primary obstruction. It occurs also in the zone of cerebral tissue in which meet the regions supplied by the arteries of the cortex and of the central ganglia.

Ultimate changes.—White and yellow softening may remain for years unchanged. Usually the products of disintegration of the nerve-elements are gradually removed, so that a cavity remains, across which bands and trabeculae pass—the remains of vessels and other structures that have escaped total necrosis. Sometimes the changes in the elements of the neuroglia and the extravasated white corpuscles result in the formation of a considerable quantity of connective tissue, consisting of fine fibre-cells and fibres, most abundant in the margins of the softened areas, which become firm and dense, while the trabeculae of the connective tissue, crossing the cavity, are thickened in like manner. After a time when the amount of this tissue is considerable, the fluid may be absorbed, and a sort of cicatrix result from the union, partial or complete, of the walls. In other cases the walls alone are thus altered, the solid particles are removed from the softened tissue, and a cyst is formed. The outer portion of the cyst or cicatrix may be limited by a zone of dilated blood-vessels, presenting, under the microscope, a peculiar and quite characteristic feature.

Seats of softening.—There is no part of the brain in which softening has not been found, but its most frequent seats are the cortex, the corpus striatum, and the optic thalamus. In the pons Varolii and medulla it is also frequently found, but it is rare in the cerebellum. Its frequency in the medulla is underrated because only minute areas of softening are compatible with a duration of life sufficient to permit them to assume a characteristic aspect. Its occurrence, position, and characters depend on the distribution of the vessels. The small arteries of the corpus striatum and optic thalamus are 'terminal arteries,' having only capillary communication with other vessels, insufficient for a collateral circulation adequate to maintain the vitality of the tissue. The arteries to the cortex of the brain are sometimes terminal, but sometimes possess anastomosis by arterioles with other branches. Hence obstruction in the central arteries leads invariably to softening, while obstruction in the superficial arteries may also cause softening (which involves the grey substance of the convolutions and some of the subjacent white centre to which the vessels penetrate), but often the anastomoses of the superficial vessels are so free that softening does not result. An obstruction of a main trunk (as the middle cerebral) may lead to softening of the central region (corpus striatum), while the convolutions escape; but usually both suffer. For the same reason, the softening of the cortex is apt to be irregular in distribution, and partial even within the region of the obstructed artery, a feature considered

further in the article on Diseases of the Vessels of the BRAIN.

SYMPTOMS.—The *premonitory* symptoms of softening of the brain depend upon its mechanism, and are considered in greater detail in the articles describing its causes. In embolism other symptoms than those of cardiac disease are usually absent. Occasionally a slight attack of loss of cerebral function, due to a slight embolism, may precede a graver attack. In softening due to arterial disease, premonitory symptoms of local cerebral anæmia are frequently present; these may exist for months before the onset, or only for a few days, or even for a few hours. There is also, in many cases, sufficient disease to impair the nutrition of many parts of the brain, revealed by symptoms of wide range—mental deterioration, numbness, and pains in the limbs, pain in the head, or slight local weakness. These symptoms are of especial significance when associated with evidence of degeneration elsewhere in the arteries; with the conditions—as chronic Bright's disease, alcoholism, and senility—in which atheroma of the cerebral arteries is common; or with constitutional syphilis. Such slight cerebral symptoms may also precede the softening due to syphilitic arterial disease, but the most common antecedent of this is headache for a week or two, often considerable.

The symptoms of *actual* softening are those of loss of function in the damaged portion of the brain. Strictly, indeed, these symptoms are those of the anæmia that causes the softening, and which, depriving the brain-tissue of the material for its functional action, arrests the latter with rapidity. The onset of the symptoms may be actually sudden, as in embolism, and sometimes in thrombosis; or it may be gradual, as occasionally in thrombosis, and less commonly in embolism. When sudden, the symptoms of initial shock are added to those due to the structures involved (*see* BRAIN, Tumours of). Hemiplegic symptoms and mental disorder are the most common effects. Hemiplegia especially occurs in embolism, on account of the frequency with which the middle cerebral artery is obstructed, and of the important motor regions (corpus striatum and motor parts of the convolutions) to which that artery is distributed. From the distribution of the artery to the lower frontal convolutions and adjacent region, aphasia is frequently present when the obstruction is on the left side. Hemianæsthesia is also common, and also hemianopia, which often escapes detection.

When the onset is sudden it is often after some fatiguing exertion, or during exhaustion from any cause, or in embolism after some cause of undue excitement of the heart's action. If the area damaged be extensive, there is loss of consciousness, and there may be all the symptoms of an apoplectic seizure. The loss of consciousness is rarely profound, and the symptoms of apoplexy soon pass off. In the most severe cases, however, they may deepen to fatal coma, especially when an important artery is occluded in each hemisphere. Thus the closure of both middle cerebral arteries causes symptoms indistinguishable from those of ventricular hæmorrhage and equally fatal in ultimate result. Symptoms of irritation commonly succeed the onset as the collateral hyperæmia sets in, or they may be marked from the first. Convulsions, usually unilateral, may occur and be repeated for days, chiefly when the softening involves the motor region of the cortex, and spares the path from it through the central ganglia. Convul-

sions may also occur in a few weeks or during the stage of secondary inflammation which occurs from the third to the seventh day after the onset, and is usually attended by slight pyrexia, some pain in the head, and occasionally some delirium. The patient may even pass from the apoplectic condition into one of delirium. In the aged, delirium may be the chief symptom of the onset in cases in which the softened areas are small, multiple, and cortical in situation. According as these symptoms are chiefly marked at the onset three varieties have been described, the *apoplectic*, *convulsive*, and *delirious* forms.

Recovery from the special symptoms of the attack is often incomplete; permanent weakness may remain, as hemiplegia, and mental power is lessened in the old, the patient passing into the chronic state about to be described. The persistent hemiplegia is often accompanied by rigidity, or, when the loss of power is incomplete, by mobile spasm, such as, in its most marked form, has been described as *athetosis*. When the cause is arterial degeneration, return or relapse is common, and it is almost invariable where senile arterial disease is widely spread.

Chronic softening of the brain is a term applied to a group of symptoms, of wide range, indicative of failure of cerebral power. It is a term that has become firmly rooted in popular nosology, where it denotes chiefly general paralysis of the insane, but includes also all maladies attended by a similar mental failure, profound in degree, permanent in duration, and progressive in course. The symptoms indicated by the term may supervene on more acute symptoms of softening, or may be gradual in their onset. There is mental dulness, defective perception, drowsiness, loss of memory (especially for recent events), often slight wandering; emotional manifestations are easily excited. Physical power is defective—as a rule generally, sometimes locally. The more delicate motor actions are imperfectly adjusted: articulation becomes indistinct, and the handwriting imperfect. These symptoms may progress into actual imbecility, or may be cut short by some more profound cerebral seizure, or by some intercurrent pulmonary affection, rendered grave by the deficient muscular respiratory power. They are met with chiefly in the aged, and are probably due to concomitant degeneration of cerebral tissue and of the arteries supplying it with blood—the two pathological elements varying in relative degree and in relation. Spots of softening, often widely spread, may be found, and are, indeed, the cause of many of the symptoms. But the state may also come on without any local softening, and without the degeneration of vessels to which such softening is usually due. A similar state often follows any grave local lesion of the brain in advanced life.

Progressive symptoms, local or general, or both, sometimes of the character just described, and sometimes such as are caused by a cerebral tumour, have been met with in a few cases in which extensive local softening of the cerebral tissue has been found without vascular disease to explain it, and without correspondence with arterial territories. Most subjects have been old, and the disease has not commonly been diagnosed during life. Some patients with well-marked symptoms of this character have been the subjects of strong constitutional gout, and it has been conjectured that a local gouty inflammation of the cortex has been the cause.

DIAGNOSIS.—The *acute* form of softening has to be distinguished from acute congestive apoplexy and

from cerebral hæmorrhage. It is distinguished from the former by the occurrence of the symptoms indicative of local mischief, and by the absence of evidence of cephalic hyperæmia. From hæmorrhage the diagnosis is often difficult, except during the first half of life, when hæmorrhage is so rare that it should only be thought of when loss of consciousness is profound and prolonged. In softening from thrombosis, the initial apoplectic symptoms may be absent, or, if present, they are slight and brief. They are more often preceded by slight local cerebral symptoms, due to the vascular disease, than is the onset of cerebral hæmorrhage. In the latter such symptoms are merely associated, and are not due to the cause of the hæmorrhage, except in the rare cases in which there is an aneurysm of one of the larger intracranial arteries. Most important is the indication afforded by the state of the heart; evidence of its hypertrophy is seldom absent in cerebral hæmorrhage, while it is feeble and often irregular in the softening from atheroma, and presents evidence of valvular disease in embolism. The pulse is tense and often full in hæmorrhage, and in thrombosis from atheroma it is small, feeble, and often irregular, like the action of the heart. In either state evidence of degenerative valvular disease may co-exist. Improvement occurs earlier than in cerebral hæmorrhage. The temperature rises soon after the attack, but falls in a day or two, to rise again slightly during the stage of secondary inflammation. There is more marked mental change than in hæmorrhage, shown at first in excitement, subsequently by depression and deterioration of power. In the cases in which the onset is sudden and the apoplexy profound, a diagnosis from hæmorrhage can only be made by the state of the heart and circulation. In embolism the onset of the attack is commonly sudden, without preceding symptoms, since the state of the brain is normal until the artery is suddenly obstructed. The loss of consciousness is less profound than in hæmorrhage, and is often absent. This is true also of softening from syphilitic disease, in which there are often premonitory symptoms, especially headache. This once proved an accurate indication in a case in which coincident valvular disease suggested embolism. The diagnosis is often also aided by a history of the causal malady. Too much weight must not be laid on the absence of such a history or on the absence of a history of constitutional symptoms, while neither the thoroughness of former treatment, nor a considerable period of freedom from symptoms, has any value whatever as a contra-indication. But with the lapse of time, it does become unlikely that softening is due to this cause; still, it is only after twenty years, and in the absence of any suspicion of a second infection, that the improbability has considerable weight. Optic neuritis, developing after the lesion, occurs in rare cases of softening from embolism, and is practically absent in hæmorrhage (except as a result of Bright's disease). In capillary embolism, if extensive, a distinction from hæmorrhage often cannot be made: the loss of consciousness is profound and lasting. Bilateral softening, such as sometimes results from disease of both middle cerebral arteries, may also (as already mentioned) cause symptoms indistinguishable from those of ventricular hæmorrhage because the second side is usually affected after the first.

The distinction of softening from other cerebral diseases is described in the articles on these.

PROGNOSIS.—The immediate and ultimate pro-

gnosis in an attack of softening of the brain depends on the degree and extent of the symptoms as indicating the extent of the lesion; and on their character, as indicating the region of the brain damaged. Both the near and the remote prognosis is much graver in damage to the medulla and pons Varolii than when the corpus striatum or cerebral hemispheres are affected. Locality is of special importance in regard to the prognosis in softening, because it is by far the most frequent lesion of the medulla or pons that is survived. The intensity of the coma in softening indicates a greater degree of gravity than does the duration of slighter impairment of consciousness. When actual softening has occurred, the damaged tissue never regains its functional power. The congested periphery may recover in proportion to the integrity of the tissues, and to the freedom of the vessels from disease. The prognosis of the residual symptoms corresponds to that of those due to hæmorrhage described in the article on that disease. The chances of a recurrence of softening in another situation depend on the extent to which its causal condition is widely spread or can be removed. In thrombosis from atheroma recurrence is almost certain, but is likely to be deferred in proportion as the attack was due to an adequate exciting cause. In embolism recurrence is rare. The prognosis in syphilitic disease of the vessels depends upon the recognition and treatment of the syphilitic influence so far as recurrence is concerned; but the prognosis of developed symptoms is independent of the fact of their syphilitic cause, or of any treatment to which the patient may be subjected. Treatment can only influence the disease of the wall of the vessel, and this can have no effect in restoring the circulation, since the vessel beyond is closed by clot, or in restoring the structure of the destroyed tissue of the brain. As a matter of fact the course of hemiplegia due to this cause is precisely the same as that of hemiplegia due to softening produced by any other vascular mechanism such as embolism; improvement or recovery is determined by the position of the lesion, and the extent to which the symptoms are not due to the actual destructive softening, and to which they can be compensated. Improvement or persistence of symptoms may coincide with treatment, but the one is not due to it, nor the other the result of its absence.

TREATMENT.—The treatment at the onset of cerebral softening is of great importance, because, although nothing can probably be done to lessen the mischief that has occurred, much may be done to prevent its extension or increase. The closure of a vessel by clot is the great fact of what may be termed the therapeutic pathology of these cases, and the measures to obviate the increase in this process have much in common in the various forms of softening. The variations rendered necessary by the differences in mechanism are considered in the articles on the several vascular diseases. During the acute stage the patient must be kept at perfect rest, with the head moderately raised, flexion of the neck being avoided. During the initial stage of shock, warmth by hot-water bottles, &c., should be applied to the extremities, to equalise the circulation. The bowels, if confined, should be made to act gently; but, unless the evidence of encephalic congestion be early and conspicuous, strong purgation should be avoided. The determination of blood from the brain to the

intestines, involved in purgation, and desirable in cerebral hæmorrhage, is to be carefully avoided in cerebral softening. It is important also to maintain the circulation steady and uniform, avoiding alike undue quickening or slowing of the blood-current. Hence digitalis or strophanthus is useful in moderate doses when there is cardiac weakness or irregularity. In all senile cases, or if there is a gouty diathesis, nitrous ether or other diuretic drug is useful, and, in the latter case, some lithia may be added. Thus the tendency of the blood to clot is lessened, and the risk of the extension of the lesion—always a danger in the early stage of the affection—is lessened or obviated. Stimulants must be given or withheld according to the state of the heart and circulation; when there is doubt whether they should be given or not, it is better to give a small quantity in softening, and to withhold them in hæmorrhage or if the diagnosis from hæmorrhage is doubtful. After the stage of depression has passed, the irritation due to secondary inflammation, indicated by headache and elevation of temperature, needs quietude, laxatives, and sometimes cold to the head. When convulsions are an early and recurrent symptom, mustard plasters to the neck and extremities, and bromide of potassium internally, are sometimes effective; in the early period, ice to the head may arrest them. The fits that attend the actual onset are seldom influenced by treatment; they cease because the tissue is destroyed that is essential for their occurrence. When these fail, hyoscine hydrobromate may be given cautiously, and in small doses ($\frac{1}{300}$ gr.). In embolism the treatment of the cardiac condition is of chief importance. In syphilitic disease iodide of potassium should be given promptly but in doses not exceeding fifteen grains, since a larger amount, from its tendency to cause clotting, may lead to an extension of the thrombus. Although it cannot influence developed symptoms, it is necessary because we do not know what other arteries may be diseased and how near the patient may be to a serious increase in the symptoms.

After the attack has passed, recovery must be aided by maintaining the general health in the best possible condition. The secretions should be kept free, the digestive organs in good order, the habits strictly regulated, and nerve tonics—cod-liver oil, hypophosphite of sodium, strychnine, quinine, and iron—may be given with advantage. The symptoms of chronic softening, whether occurring after an acute attack or coming on gradually, should be treated in a similar manner.

W. R. GOWERS.

BRAIN, Syphilis of the.—SYNON.: Ger. *Gehirn-syphilis*; Fr. *Syphilis cérébrale*.—Syphilis is one of the most important factors in the production of organic brain-disease. The virus appears to act in two ways: (1) Directly upon the blood-vessels and connective tissues generally, with secondary destructive changes in the nervous tissue—a true specific inflammation; (2) by direct influence upon the vitality of the neurones themselves, producing systemic degenerative changes, of which tabes dorsalis and general paralysis are by far the most common and important; these diseases are frequently spoken of as 'parasyphilitic' or 'metasyphilitic' affections.

ÆTIOLOGY.—It was generally accepted, and is still held by many authorities, that syphilitic brain-

disease occurs especially in the later (tertiary) periods. One of the first observers to doubt this view was Rumpf, and he was followed by Bruns, who collected 100 cases, in nearly half of which brain-symptoms occurred in the first year after infection. Naunym, from an analysis of 45 of his own cases and 290 collected cases, came to the conclusion that syphilitic disease of the nervous system appears most frequently during the first year after infection, and that its frequency diminishes from year to year. In the experience of the present writer, out of 40 cases in which the date of infection and the time which had elapsed before the onset of the cerebral symptoms could be determined, four occurred in the first year, seven in the second year, six in the third year, three in the fourth year, and the remainder at subsequent periods. Lang has pointed out that often during the outbreak of the syphilitic exanthem, headache, giddiness, pyrexia, increased frequency or slowness of the pulse-rate, and inequality of the pupils may occur; and he considers these symptoms are probably due to meningeal irritation. The question whether mild or severe forms of syphilitic infection are more liable to be followed by disease of the nervous system is one of importance. Broadbent was one of the first to point out that severe nervous symptoms often follow mild attacks. The writer's experience seems to show that all forms of syphilis may be followed by severe nervous symptoms, and that the mild forms are prone to be followed by serious disease because they are often inadequately treated. It seems, however, a fact that mild forms of syphilis are especially liable to be followed by the parasyphilitic affections; and this fact finds an analogy in the occurrence of diphtheritic paralysis. Most of the leading authorities are of opinion that brain-syphilis is much less likely to follow when the disease has been efficiently treated. Hjellmann has made accurate observations upon this subject, and he has found that 82-85 per cent. of the cases of brain-syphilis were those in which only an imperfect treatment had been carried out.

Traumatism.—Injuries to the head have long been recognised as a cause of syphilitic brain-disease; and frequently a history of a blow or an injury to the head precedes the symptoms of a localised gummatous meningitis of the convexity. Of all the causes which predispose to syphilitic brain-disease, chronic alcoholism is the most important, but all conditions which tend to produce cerebral congestion, excitation, or stress may be contributory factors in the production of the disease, e.g. sun-stroke, excesses '*in Baccho et Venere*,' lead-poisoning, &c.

Sex and age.—The disease affects particularly males between the years of twenty and forty-five, because syphilis is more common in men, and because during this period of life infection is more likely to take place.

Heredity.—Families in whom there has been no trace of syphilis are probably more prone to severe sequelæ, owing to lack of immunity. Hereditary predisposition seems to play a part. As an instance of this, the writer had under his care two brothers who at nearly the same period after infection developed almost identical symptoms of a cerebral gumma. Both were treated with anti-syphilitic remedies and made good recoveries.

Seeing that syphilis is more common in cities

and large towns, this form of brain-disease is more frequently met with in urban than rural populations, but there is not the same disparity in numbers as in general paralysis of the insane.

It is usual to consider syphilitic brain-disease under the following headings:—basic meningitis, meningitis of the convexity, cerebro-spinal meningitis, arteritis, encephalitis, and neoplastic formations or gummata. All these conditions are frequently combined in the severe and early forms of the disease.

1. Basic Syphilitic Meningitis.—SYMPTOMS. *Headache* is one of the commonest, earliest, and most constant symptoms; occasionally it precedes all other phenomena by months and even years. It is not infrequently associated with *vomiting* of a cerebral character; this last symptom may, however, be absent through the whole course of the disease. The pain is paroxysmal, and when it reaches its greatest intensity, it may be boring and excruciating, but this may gradually die down, leaving a dull aching. It is curious but very characteristic that the pain should be most severe at night or in the early morning; it may even awake the patient at a regular time. In the basic form of the disease, the pain is referred to the frontal, temporal, and parietal regions, is deep-seated, and cannot be definitely localised; it may later on spread to the occipital region, the neck, and the spine. Sometimes pain is referred to the back of the eye; in this form of the disease the pain is not usually influenced by pressure, and there is no definite local tenderness as when the convexity is affected.

Stiffness of the neck may be complained of, and there is sometimes retraction of the head and neck. The patient is subject to attacks of vertigo, reeling, staggering, and loss of balance.

Temperature.—It is generally recognised that syphilitic meningitis differs from other forms of meningitis, e.g. tubercular, by the absence of pyrexia, the temperature being usually normal or sub-normal.

Ocular affections.—Of all the symptoms of syphilitic basic meningitis, affections of the optic chiasma and of the nerves supplying the muscles of the eyeball are most frequent and important. Uthoff concludes from his observations that in only about 15 per cent. of all cases of cerebral syphilis are ocular disturbances of some form or other absent. Unilateral paralysis of the oculo-motor nerve, which may be partial or complete, is so common that both Ricord and Fournier designate this ophthalmoplegia '*la signature de la vérole*.' The sixth and fourth nerves are very much less frequently affected than the third. Brain-syphilis may be accompanied by the following ophthalmoscopic changes: (1) choked disc, due to intracranial pressure; (2) optic neuritis; (3) neuritic atrophy; (4) simple atrophic degeneration. Generally speaking, there is a considerable difference in the intensity of the affection of the two eyes. Optic neuritis, as a sole and uncomplicated manifestation of basal syphilis, is extremely rare. The optic neuritis, according to Gowers, is not due to syphilitic disease of the retinal arteries; and in three cases, examined by the writer, in which there was universal endarteritis cerebri, there were no changes in the retinal arteries.

The pupil is more or less dilated, the reflex movement of the iris sluggish or absent both to light and accommodation, and the 'syndrome of Weber' is not infrequently met with, viz. paralysis of the

motor oculi on one side with hemiplegia or hemiparesis of the opposite side. Oppenheim speaks of hemianopsia as being frequent, due either to affection of the optic tract of one side, or to a destructive lesion of one occipital lobe. As a result of the meningitis, other cranial nerves may be affected, but the lesion is usually unilateral. When the fifth nerve is affected, the patient may complain of severe pains over the distribution of the nerve, with hyperæsthesia if an irritative lesion exist; sometimes hypæsthesia or even complete anæsthesia may be found in cases presenting a paralytic lesion. The corneal reflex may be absent; conjunctivitis or even suppurative keratitis may occur; and loss of taste and loss of smell may be found on the same side, due to affection of the mucous membrane of the nose. A lesion of the facial nerve is almost as frequent as of the fifth, and is often associated with deafness, due to the adjacent auditory nerve being involved. Symptoms of affection of the vagus nerve relate mainly to the circulation and respiration; and especially characteristic is a rapid change of the pulse, which may be quick, slow, or irregular; various respiratory disturbances towards the end of life are referred to affections of this nerve. The writer has seen several cases of affection of the spinal accessory nerve causing paralysis of the soft palate and larynx. Unilateral atrophy of the tongue may arise from a destructive lesion of the hypoglossal nerve. It is, however, an astonishing fact that with extensive gummatous meningitis affecting the whole base of the brain, only one cranial nerve, usually the fifth, may be partially paralysed, and yet microscopical examination may reveal extensive infiltration of other nerves by inflammatory material.

Basic meningitis frequently gives rise to polydipsia, polyuria, and sometimes glycosuria. There is usually constipation. There may be paresis in the limbs, but as a rule, unless there be blocking of an artery, there is not paralysis. The spinal membranes are usually affected, but not enough to give rise to symptoms; in five cases, however, the writer found extensive spinal as well as cerebral meningitis, and in several of these, spinal irritative phenomena presented themselves during life in the form of opisthotonus and tetaniform spasms.

Psychical symptoms arise in brain-syphilis when there is general affection of the arteries associated with a gummatous meningitis. In the cases examined by the writer the mental symptoms shown were very probably due to circulatory disturbances caused by an obliterative arteritis, evidence of which was invariably present *post mortem*, as well as to the meningitic process. Heubner showed that when two large branches entering into the formation of the circle of Willis are profoundly affected by obliterative arteritis, mental symptoms arise.

Disturbances of consciousness are usually present and indicate severe and multiple lesions. One of the commonest symptoms is somnolence and a kind of drunken stupor from which the patient is with difficulty aroused. Associated sometimes with this condition is a purposeless motile activity and restlessness. Such patients may or may not respond to questions; they usually have loss of control over the sphincters. The stupor may deepen, or the patient may recover consciousness and hope of recovery be aroused, when quite unexpectedly the drowsy stupor will recur, perhaps deepening into coma, and the patient dies without regaining con-

sciousness. Recurrent attacks of drowsiness, stupor, and coma always suggest the existence of basic syphilitic brain-disease. In the drowsy condition, the tonus of the muscles remains and the reflexes are present, but as the coma deepens both become lost. In all cases of severe brain-syphilis dementia of varying degree is a constant symptom, and all stages between slight impairment of intelligence and gross dementia may be met with: this variability of the dementia is especially characteristic of the disease. Delirium with acute maniacal excitement may be the first indication of mental symptoms; or loss of temper, moroseness, and excitement may usher in the condition. It is not uncommon for a person suffering from syphilitic dementia to be found wandering at large, and be taken up by the police. A frequent condition is mental depression with delusions of persecution and attempt at suicide, and such patients may be dangerous to themselves and others. Mental excitement may alternate with mental depression, and such disturbances may further alternate with comatose attacks and with periods in which consciousness is only partially disturbed and the intelligence is fairly good. Epileptiform fits are not uncommon, and partial or unilateral clonic spasm may occur with consciousness slightly or not at all disturbed. As a rule the fits are transitory and not persistent; they are due to affections of vessels or to gummata or softening in or near the motor area. Tetaniform spasms with opisthotonus occasionally occur in the severe forms of cerebro-spinal meningitis, and, according to Wunderlich, a cataleptic state may be seen.

Should patients recover—and many do partially recover under suitable treatment—they are left mentally enfeebled. There is lack of expression; the speech is slow, often thick and slurred—bradyaphasia, or dysarthria, but seldom complete motor aphasia. There is weakness of memory, slowness of thought and action, loss of will power, often a stolid indifference with a tendency to causeless outbursts of passion or laughter. The patient is emotional, easily moved to tears or mirth, showing a loss of the higher controlling functions of the mind. Sometimes there is a permanent paralysis of the ocular or some other cranial nerve, blindness or deafness, monoplegia, hemiparesis, hemiplegia, dysarthria, or general paresis.

2. Syphilitic disease of the Convexity of the Brain.—A hard and fast line cannot be drawn between disease of the base and of the convexity. Of 24 *post-mortem* examinations of syphilitic brain-disease made by the writer, the majority showed either multiple lesions or diffuse gummatous meningitis.

The process may start in the base or meninges, and remain *circumscribed*, forming a gummatous tumour, or become *diffused* as in meningo-encephalitis. Specific inflammation of the membranes causes extremely severe pains, paroxysmal in character, and besides general headache, there is usually a well-defined localised tenderness on pressure or percussion, which may be independent of spontaneous headache. The general symptoms of meningitis of the convexity may be vomiting, stupor, giddiness, and psychological disturbance. If, however, the lesion be situated over 'a silent part' of the brain, headache may for a long time be the only symptom. Optic neuritis is uncommon; it may even be absent when nearly a whole hemisphere is affected. Gummatous growths like tumours

produce localising symptoms when situated over portions of the brain having specific functions, e.g. the motor area (*vide* fig. 1. BRAIN, Tumours of).

3. Primary Syphilitic Arteritis.—Even without any meningeal affection, one of the earliest and most important symptoms present may be headache (usually more severe than that of arteriosclerosis), giddiness and drowsiness, and various psychical disturbances not unlike some of those of general paralysis. Heubner asserts that headache, giddiness, sleeplessness, irritability, and weakness of memory often precede an apoplectic seizure. These symptoms, together with a transitory monoplegia, hemiplegia, hemiparesis, hemianopsia, hemianæsthesia, word-blindness, and deafness, but especially aphasia, lasting a few minutes, hours, or days, and indicating a temporary circulatory disturbance, are among the most important early indications of endarteritis syphilitica. As long as the lumen of the affected arteries is not obliterated by a spreading thrombosis, a collateral circulation can re-establish the blood-supply to the parts before actual death of tissue occurs. As the endarteritis advances this restoration of blood-supply becomes more and more difficult, and the tendency to stasis and the formation of a thrombus increases, until sooner or later (if active treatment be not adopted) a permanent loss of function occurs. Paralysis may gradually affect the leg, arms, and face; or a hemiplegia may come on quickly, or even suddenly, following an apoplectic fit. Sometimes the loss of consciousness deepens to coma, and the patient dies: usually he regains consciousness, and a partial recovery from the ensuing paralysis occurs; but in the course of weeks, months, or years the paralysis may return or affect the other side of the body, and a pseudo-bulbar or cerebro-bulbar paralysis results. In severe cases of multiple syphilitic arteritis with thrombosis, death usually follows; or should the patient recover, he is generally demented and paralysed.

4. Multiple Gummata.—A patient suffering with this severe form of brain-syphilis is demented, frequently maniacal or stuporose; there may be slurred speech with tremors of the face-muscles; the pupils are unequal and sluggish in reaction to light and accommodation, or do not react at all. He does not respond to the calls of nature. Such cases closely resemble general paralysis, inasmuch as there is always progressive dementia and paresis; there is usually, however, some evidence of a coarse lesion such as a monoplegia or hemiplegia, or strabismus with diplopia, ptosis, and optic neuritis. It is, however, remarkable that in multiple gummata optic neuritis may for a long time be absent. It has been the writer's experience to find multiple gummata always associated with general endarteritis, periarteritis, and basic meningitis. This severe form of brain-syphilis occurs most often in the first few years after the primary infection, and therefore is met with usually in young adults; as a rule it is intractable to remedies, either ending fatally or leaving the patient mentally deficient.

5. Cerebro-spinal Meningitis.—In nearly all the cases of basic meningitis which the writer has examined, he has found evidence, both gross and microscopic, of some involvement of the spinal membranes. As a rule, the brain-symptoms are so predominant that the spinal symptoms are overlooked: three out of seven cases, however, exhibited symptoms during life—namely stiffness in

the neck, pains in the back, radiating pains and girdle-sensation, with hyperæsthesia or anæsthesia over the buttocks, trunk, or extremities, indicative of irritation or destruction of the posterior roots. There may in severe cases be tetaniform spasm and opisthotonus, followed by atrophic paralysis indicating affection of the anterior roots. These symptoms, pointing to affection of the spinal membranes, naturally add to the gravity of cases of basic meningitis. Finally it must be remembered that syphilitic brain-disease in all its forms may now and then be accompanied, preceded, or followed by tabes dorsalis or general paralysis.

PATHOLOGY.—*Naked-eye appearances of Brain-Syphilis.*—Essentially the changes are the same in all cases; the process is one of inflammation which has nothing absolutely 'specific' about it, but which exhibits certain features characteristic of syphilis. There is considerable resemblance between tubercular and syphilitic brain-disease, and it is often extremely difficult to distinguish one from the other by the naked-eye appearances.

Syphilitic new-growths belong to the 'granulation tumours' of Virchow, or the 'infective granulomata' of Klebs; they are termed gummata. The gummatous process, which nearly always starts in the membranes, may extend over the surface of the brain in the form of an exudative meningitis or along the vessels into the substance of the brain, causing a definite localised tumour. If the process starts anywhere about the base, as in the interpeduncular space and optic chiasma, it usually spreads into the Sylvian fissures, and over the whole base of the brain, covering the vessels and cranial nerves with a gummy exudation which has a semi-solid consistency not unlike agar or gelatine culture-media in appearance. The membranous deposit may be removed with the finger when of comparatively recent origin; when of some standing it is drier and firmer; it may be grey or grey and red in colour, or semi-translucent, yellowish or speckled. Under treatment, this inflammatory exudation can, in great measure, be absorbed. The vessels appear thickened and of a dirty white colour like wet wash-leather; laid on the finger they can easily be cut transversely, and the lumen may be seen partially or completely occluded; the endarterium is markedly thickened, usually more on one side than on the other, giving the section a 'half-moon' appearance. The vessels may feel stiff like cartilage, but this degree of resistance is not met with in acute cases. Besides the nodular and general endarteritis, there is always a similar periarteritis. Small nodules, visible to the naked eye, lie scattered about the base of the brain. In some cases these miliary gummata upon microscopical examination seem to consist of small vessels affected with endarteritis and nodular periarteritis; they somewhat resemble miliary tubercles.

Localised new-growths (gummata).—These may be single or multiple, and vary in size from a pea to a pigeon's egg. When single and circumscribed they usually start from the meninges of the base of the brain or hemispheres, causing adhesive inflammation; but when multiple, they may be scattered about in various positions, such as the hemispheres, pons, crura cerebri, the base of the brain, the basal ganglia, and even the lateral ventricles; they do not occur in the cerebellum. Gummata vary much both in consistency and

colour, as well as in general appearance; their consistency is usually unequal in different parts, the younger peripheral growing tissue being soft, succulent, and vascular, the older dry, firm, and non-vascular. Hence the edges appear grey, greyish-red, or red, while the central portions are yellowish, owing to the occurrence of necrotic changes akin to caseation. The caseous mass does not usually form a well-defined nodule, but is more often broken up into irregular areas which coalesce here and there by grey fibrous strands, or soft greyish-red granulation-tissue. There is little tendency to calcification or to the formation of an abscess.

Microscopical appearances.—The neoplastic formation, whether circumscribed or diffuse, is essentially the same. It consists of an inflammatory exudative product containing enormous numbers of round cells, indistinguishable from migrated leucocytes, in a coagulated serous fluid. This forms the main constituent of the membranous deposit formed at the base of the brain; it invades all the adjacent structures, the membranes, the walls of the vessels, the pial septa, and the roots of the cranial and spinal nerves. The acute inflammatory process causes congestive stasis in the vessels, so that, wherever this inflammatory process has extended, venous and capillary stasis occurs, independently of and apart from the organic changes in the walls of the vessels. Besides this, there is active proliferation of the mesoblastic connective tissue-elements in the form of abundant spindle-shaped and stellate cells. In the older parts of the gummatous process these formed elements may alone remain, the inflammatory products having been absorbed. The changes around the vessels are essentially of the same nature, namely, infiltration of the walls of the vessels with inflammatory products, causing a periarteritis, which may assume the form of a nodule (*periarteritis nodosa*). In the older parts of a gumma, the recognition of the existence of a vessel is only possible by the crinkled line of the still undestroyed elastic lamina.

Syphilitic endarteritis is characterised microscopically by a cellular proliferation of the endarterium; it frequently gives rise to the appearance of little nodules on the arteries, owing to the process affecting one side of the vessel especially, but it may affect the whole of the circumference. It is one of the most frequent results of syphilis, and may be associated with meningitis. It affects especially the vessels of the circle of Willis, the arteries of the Sylvian fossa, and the lenticulo-striate vessels. It may or may not be associated with periarteritis, which is usually present where there is meningitis. The endarteritis is the result of irritation and thickening of the endarterium, and is brought about by proliferation of the subendothelial connective-tissue cells; the inflammatory exudation and round-celled infiltration distends the spaces between these proliferated cells. According to Heubner, the characteristic feature of endarteritis syphilitica is that it never undergoes caseation, and therefore never becomes atheroma. This dictum is, generally speaking, true, but there are many reasons for believing that it is too exclusive.

Congenital syphilitic brain-disease.—According to Barlow and Bury, the most common brain-lesion met with in hereditary syphilis is a cortical sclerosis, which microscopically examined shows atrophy of cells and overgrowth of neuroglia-tissue.

The condition may arise (a) as a result of specific endarteritis; (b) secondary to a chronic meningitis; or (c) it may be primary and apart from meningitis or arteritis. The symptoms, as in the acquired form, may be mental or physical. The mental condition may indicate arrested development (idiocy and imbecility) or premature decay (dementia). These authors state that the clinical type of intracranial hereditary syphilis is a spastic paresis of the limbs associated with dementia or amentia, the patient being very frequently the subject of epileptiform convulsions.

DIAGNOSIS.—The early diagnosis of syphilitic brain-disease is of great importance on account of its curability, provided the disease is treated before destructive tissue-changes have occurred. Such changes, however, in the 'silent parts' of the brain may proceed to an advanced degree without producing well-defined symptoms. Thus the diagnosis of syphilitic brain-disease which is producing only mental symptoms is attended with far greater difficulty than when there co-exists some definite objective symptom, such as paralysis of a cranial nerve. It has already been pointed out how frequently ptosis and other evidences of paralysis of the third nerve occur in brain-syphilis, and it is noteworthy that cerebral syphilis seldom strikes without warning symptoms. Even in the absence of any paralysis, or evidence of syphilis, and in spite of persistent denial of infection, if a young or middle-aged person suffers with mental symptoms accompanied by headache, especially nocturnal headache, or if there is a history of a fit followed by transitory aphasia, paralysis, or paresis, it is justifiable to suspect brain-syphilis, although the symptoms might point equally to general paralysis. Frequently the correctness of such a diagnosis is confirmed by rapid improvement under energetic anti-syphilitic treatment.

In women far greater difficulties arise in diagnosis: the primary sore and secondary symptoms are often overlooked, and there are frequently no residua on the body; but 'parchment' scars, enlarged cervical glands, evidence of old iritis, and especially a collar of pigmentation around the neck should be looked for, in the absence of more obvious syphilitic manifestations. A very important matter is the history of miscarriages, abortions, dead fetuses, and premature births. According to Birsch-Hirschfeld, syphilis is responsible for two-thirds of such cases. It is, however, equally important to remember that syphilitic parents may have perfectly healthy children.

Seeing that syphilis may attack any part of the cerebro-spinal nervous system, causing general or local irritation or destruction, the complex of symptoms arising therefrom may simulate almost any disease of the nervous system. Such diseases are—new growths, including tuberculosis; tubercular meningitis; disseminated sclerosis; arterio-sclerosis; epilepsy; hysteria; neurasthenia; alcoholism; uræmia; lead-poisoning; tabes dorsalis; general paralysis of the insane; mania; melancholia; epilepsy with dementia; dementia; and local or diffuse softening, the result of embolism or thrombosis.

It is of such importance to the practitioner to be able to differentiate these diseases from brain-syphilis that some of the more important diagnostic points may here be indicated; but for further information reference may be made to the special articles upon each of these subjects.

New growths (including tubercle) seldom show the same spontaneous remissions and exacerbations as gummatous syphilitic meningitis. Symptoms of intracranial pressure, e.g. optic neuritis and vomiting, are usually much more severe in cases of new growths. The headache is continuous, and as a rule does not tend to be nocturnal to the same extent as in syphilis.

Tubercular meningitis, although common in young children, is relatively rare in adults; it is acute and progressive, and there are usually signs of tubercular disease in the lungs; there are, as a rule, more stiffness and retraction of the neck, and more muscular rigidity than in brain-syphilis. The irritative phenomena precede the paralytic, and the pyrexia is more definite and continuous than in syphilitic meningitis. Syphilis and tuberculosis may be combined, and then syphilitic remedies prove useless.

Multiple disseminated sclerosis.—Seeing that syphilitic disease of the arteries may produce multiple foci of softening, a differential diagnosis is extremely difficult and sometimes impossible. The true insular sclerosis usually affects young women rather than men. It may commence with an ocular paralysis, but it is much more often the sixth nerve than the third which is affected; and whereas nystagmus is rare in syphilis, it occurs in 60 per cent. of the cases of disseminated sclerosis.

Pupil-symptoms are common in syphilis; likewise aphasia and alexia and cortical epilepsy; the scanning speech characteristic of disseminated sclerosis should be differentiated from the dysarthria and bradyphasia of syphilis. Visual affections are common in both diseases, but complete optic atrophy and hemianopsia are not met with in disseminated sclerosis. Finally, it should be borne in mind that whereas syphilis attacks the membranes first and the cortical and spinal substance later, insular sclerosis commences in the white matter of the cerebro-spinal axis, and therefore meningeal and nerve-root symptoms point to syphilis.

Arterio-sclerosis.—Many cases of this are of syphilitic origin, but the arterio-sclerosis of people past middle life is a degenerative process, and when it affects the cerebral arteries, it is attended by various psychical disturbances, arising from disorder of the cerebral circulation, such as fainting fits, vertigo, mental depression, and headache. As a rule, however, the headache is much less severe than in syphilitic endarteritis.

Partial, unilateral, or universal convulsions may occur in *epilepsy*, *lead-poisoning*, *uræmia*, *general paralysis*, and *alcoholism*, as well as in cerebral syphilis. In such cases very difficult problems often present themselves. In cases complicated by alcoholic excess, it is well to wait until the effects of the alcohol have passed off. Epileptiform convulsions due to brain-syphilis are frequently Jacksonian in type and caused by syphilitic meningitis, and are therefore accompanied by severe headache and tenderness on percussion and pressure in an area of the skull corresponding to a portion of the motor area which presides over the group of muscles in which the fit starts.

Early cases of *Tabes dorsalis* with ocular paralyses may be mistaken for syphilitic meningitis. Ophthalmoplegia interna points rather to tabes or dementia paralytica than to brain-syphilis. The parasymphilitic affections are as a rule comparatively

painless. It must, however, be borne in mind that some cases of true brain-syphilis may be accompanied or followed by the progressive primary degenerative process. It is well to remember that just as brain-syphilis may simulate almost any form of organic brain-disease, so it may occasion or simulate almost any form of mental disease.

PROGNOSIS.—Naunyn states that out of 88 cases of cerebral syphilis 24 recovered, and 49 improved under treatment; 5 died and 10 showed no improvement. The writer's experience agrees closely with this; foreign observers give a less favourable percentage. All the writers on this subject are agreed as to the beneficial influence of early treatment. It appears to be the case that nervous affections are more likely to occur when mercurial treatment has either been insufficient or not practised. The most favourable cases are those which point to a localised gummatous process; the most unfavourable are those which simulate general paralysis, thus showing signs of universal affection of the brain: cases which indicate multiple softening from arterial occlusion are also very grave. Probably endarteritis is the most dangerous because it is the most insidious of all forms of cerebral syphilis; the warning symptoms are apt to be neglected, and if the transitory paralyses which have been alluded to are not followed by treatment, permanent loss of function ensues, for anti-syphilitic treatment, however vigorous, cannot replace nerve-tissue once destroyed. Pure basic meningitis does not indicate a bad prognosis, neither does involvement of a cranial nerve, e.g. the motor oculi—rather the reverse, for it brings the patient early under treatment. Marked optic neuritis may disappear, and restoration of sight may be complete under treatment, whereas progressive neuritic atrophy generally ends in blindness. Basic meningitis is much more favourable when limited to one side and when there is reason to suppose that it is not associated with extensive arteritis; basic endarteritis is liable to cause softening in the pons or medulla, leading to serious paralyses and fatal complications. The prognosis of brain-syphilis largely depends upon the age, habits, life, and occupation of the individual. All causes which tend to lower the general vitality of the tissues of the body or throw stress upon the nervous system influence unfavourably the prognosis, especially sexual and alcoholic excess. Age is a relative thing; the more worn out a man's tissues are, the more active usually is the syphilitic poison, and it attacks the *locus minoris resistentiæ*.

TREATMENT.—*Prophylactic.*—All causes which tend to lower the general vitality of the body or which produce nervous excitement or depression should be avoided by a patient suffering with symptoms of secondary syphilis.

Therapeutic.—As a rule the writer has found the most benefit to result from mercury combined with iodide of potassium; some authorities prefer only the latter in doses of 10 to 30 grains three times a day; but it is better to combine with it mercurial inunction, or even mercurial injection, when it is necessary to get patients rapidly under the influence of anti-syphilitic remedies. If the patient can afford it, a systematic inunction-course at Aix is advisable in many cases. If there is local pain, as in meningitis of the convexity, the head may be shaved and mercurial ointment, combined with lanoline, rubbed in over the painful part as well as into the body. Should the patient, owing to vomiting, be unable to

retain medicine and food, iodide may be given in the nutrient enemata. For out-patient practice, or for people who are not under constant supervision, or for whom it is impossible to adopt treatment by inunction, a combination of the liquor hydrargyri perchloridi (1 to 2 drms.), with 20 to 30 grains of iodide, and tincture of cinchona is of great use. Care should be taken to tell the patient to clean the gums and teeth after each meal, and a mouth-wash of chlorate of potassium may be ordered. Many patients' gums are 'touched' long before they have had efficient mercurial treatment, owing to neglect of these precautions. As a rule good effects will begin to show themselves in the first week or two, but the treatment should be continued for months; for if at first it does not lead to visible improvement, it may prevent the extension of the disease. It is well to keep patients for months and even years on moderate doses of iodide occasionally combined with mercury, according to whether it is an early or late case after infection. It is a most important element in the treatment to point out to the patient the desirability of leading a sober and steady life, and also it is the duty of the medical man to cheer up the patient, thereby promoting digestion, nutrition, and sleep, and counteracting the tendency to syphilophobia and hypochondriasis.

FREDERICK W. MOTT.

BRAIN, Tuberculosis of.—See BRAIN, Tumours of; and MENINGITIS, Tubercular.

BRAIN, Tumours of.—**SYNON.** : Fr. *Tumeurs cérébrales*; Ger. *Gehirngeschwülste*.

DEFINITION.—Under the term 'cerebral tumours' it is convenient to include not only tumours in the strict sense but also all forms of cysts, parasitic or otherwise, and aneurysms, as their clinical phenomena correspond closely with those of tumours. Syphilitic tumours (gummata) are, however, dealt with in a separate article. See BRAIN, Syphilis of the.

PATHOLOGICAL ANATOMY AND HISTOLOGY.—The following varieties of tumour may occur within the cranium: (1) *Gliomata*.—These are usually solitary, and are of a greyish-white, greyish-red, or dark-red colour. Sometimes this form of tumour may have the general appearance and consistence of a local hypertrophy of the normal grey or white matter; it can usually, however, be recognised by difference in colour and consistence from the surrounding normal substance. It may appear shining grey and transparent, sometimes greyish-white or red; or it may have a mottled appearance. The red colour depends upon congested vessels and hæmorrhages; and the vascularity may be so great in some cases as to give rise to the name *angio-glioma*. As the tumour is due to an exuberant proliferation of the normal embryonic connective tissue, it has no defined limits, but passes gradually and insensibly into the surrounding normal structures. Cavities and cysts may form in the tumour, owing to degenerative changes, and these cysts contain sometimes a clear, sometimes a turbid, sanguinolent brownish-black fluid. From the rupture of dilated vessels, hæmorrhages may occur of varying size, causing sudden symptoms during life, resembling an apoplectic seizure; and at the autopsy, the extravasated blood tearing up the brain tissue may be so obtrusive, that the true nature of the disease may be overlooked. Again, it may simulate a focus of softening, owing

to the fatty degenerative changes which have occurred in the growth. The tumour may be of almost any size, from that of a pea to that of the fist, or even larger. All authorities agree that this form of tumour does not spring primarily from the meninges, and it is probable that, as in the spinal cord, it arises in the grey matter. It is relatively frequent, for of 600 collected cases of cerebral tumours 91 were said to be gliomata. This form of tumour is met with most frequently—in order—in the following situations: centrum ovale, cortex cerebri, cerebellum, basal ganglia, pons, and medulla. When occurring in the two last-named situations, it is frequently combined with a similar growth in the spinal cord. Histologically the structure corresponds to the neuroglia, hence its name; the growth consists of delicate cells, with round or oval nuclei surrounded by a smaller amount of protoplasm; and projecting from the cells are numerous branched processes. A felt-work of delicate fibrils, consisting probably for the most part of the cell-processes, is seen in a microscopical section lying between the cells. The fibrillary structure may be so abundant in some tumours as to give them a tough fibrous character, or mucoid changes may lead to a soft tumour, termed a *gliomyxoma*. The tumour usually contains abundance of nerve-elements—fibres and ganglion-cells—in those parts where hæmorrhages or degenerative changes have not taken place, for the growth tends to infiltrate and push aside the nervous elements, without destroying them. Such growths may be looked upon as local malformations of the brain, arising as a result of abnormal embryonic development, although frequently a history of a blow on the head has preceded the manifestation of symptoms.

2. *Sarcomata*.—Sarcomata occur more frequently in the adult than gliomata. According to Allen Starr, of 600 recorded cases of cerebral tumour, 120 belonged to the sarcoma-group, 86 occurring in adults and only 34 in children or young people under twenty. Sarcomata seldom arise in the brain-substance, and very rarely in the cranial nerves. Much more frequently they commence in the fibrous structures of surrounding tissues, e.g. the membranes of the brain, the periosteum of the cranial bones, and the bones themselves, especially those of the base of the skull. All known types of sarcoma may occur (*see* TUMOURS: Sarcoma). Many of these arise as a result of metastatic deposits. Sarcoma arising in the membranes is usually either a round nodular tumour, or a diffuse superficial thickening, with a well-defined border, often pressing the brain-substance before it, and forming as it were a nest. Primary sarcomata are usually solitary and may vary greatly in size. The consistence varies according to the nature and vascularity of the growth. In the hard variety, the cut surface appears fibrous, and of a greyish-white colour; the soft variety may be yellowish, yellowish-red, or greyish-red, according to the amount of degenerative change in the growth, its vascularity, and the presence of hæmorrhages of recent or remote date. Cystic degeneration sometimes occurs, especially in the cerebellum, and rarely calcification or even ossification of a sarcoma may take place. Although primary sarcomata are usually solitary, they may be multiple, and occur simultaneously in the brain, spinal cord, and ganglia.

3. *Endotheliomata*.—According to Ziegler, a

new growth may arise from the endothelial cells of the pia-arachnoid membrane. Examined microscopically such a growth shows a stroma of fibrous tissue in which there are nests of endothelial cells.

4. *Rarer forms of Tumour* are pure fibroma, cystic fibroma, lymphangioma, osteoma, osteofibroma, osteo-sarcoma, enchondroma, exostosis, and psammoma. This last form of tumour only occurs in the meninges, in the pineal gland, and rarely in the choroid plexus. It usually causes no symptoms. When the tumour is cut into, the knife grates, owing to the existence of calcareous concretions. Examined microscopically, these growths are seen to contain round concretions of various sizes consisting of superimposed flattened cells, with a homogeneous appearance, arranged concentrically like an onion. Cholesteatomata, angio-sarcomata, angiomas, dermoid cysts, lipomata, and other growths, e.g. tumours of the hypophysis cerebri, are in rare instances met with.

5. *Carcinomata*.—Carcinoma is nearly always secondary, but sometimes primary. It has been described as primary in the cranial bones, the meninges, and the choroid plexus. It is probable that in some instances a primary growth has been overlooked in some other part of the body, and it is likely that a number of those described as carcinomata were really sarcomata.

6. *Tubercular masses*.—Tubercular 'tumours' occur most frequently in young people. Of 300 tumours which occurred in young people, 152 were tubercular: of 300 occurring in adults only 41 were of this character. The earliest age at which a tubercular tumour has been found *post mortem* was 23 days; the mother of the child was also tuberculous. The tumour was of the size of a hazel-nut (Demme). Statistics disagree as to whether tubercular tumours are more frequently solitary or multiple. According to Starr, only about one-fifth were multiple; according to Gowers, less than one half were solitary. The size may vary from a pea to a chestnut. They are met with most frequently, according to Gowers, in (1) the cerebellum, (2) the cortex, (3) the pons. The naked-eye appearance is usually characteristic. On transverse section they have a more or less sharply defined peripheral zone of a grey or greyish-red colour, with a yellowish cheesy centre, which sometimes, owing to its having undergone softening, contains a turbid, yellowish-white fluid. Microscopically, the peripheral zone appears to consist of a granulation-tissue, consisting of round cells and giant-cells: in the giant-cells tubercle-bacilli can usually be found, whereas they may not be discovered in the central part, which has undergone caseation. Very rarely a tubercular abscess may occur; and it is not very rare on the *post-mortem* table in asylums to find old calcified tubercular tumours in the brains of patients who have suffered with epilepsy and dementia. It is seldom that 'solitary tubercle' of the brain forms the primary seat of infection, tuberculosis being usually met with in other parts of the body. Tubercular tumour is not uncommonly combined with tubercular meningitis, and is not infrequently the cause of an accompanying hydrocephalus.

7. *Syphilomata* (gummata).—*See* BRAIN, Syphilis of the.

8. *Cysts*.—As already mentioned, cysts may be formed in the brain as a result of degenerative processes occurring in tumours, such as glioma,

sarcoma, and carcinoma; but occasionally in England, and not at all uncommonly abroad, parasitic cysts occur in the brain. Of these there are two forms, viz. (1) *Cysticercus cellulosæ*, and (2) *Tænia echinococcus* (see ENTOZOA). The former are not at all uncommon in Germany, for in 5,300 autopsies made at the Berlin Pathological Institute there occurred 87 cases of this parasite, 72 of these being in the brain. Hydatid cysts are not infrequently met with in the brain in those countries where infection by this parasite is common, e.g. Australia.

9. *Aneurysms*.—Apart from the miliary variety intra-cranial aneurysms occur usually on the arteries of the base or their branches before their entrance into the brain-substance. They occur frequently at the point of bifurcation of an artery; they are usually single, but sometimes multiple. Of 156 cases collected by Gowers, the aneurysm was seated in 44 on the middle cerebral artery, in 41 on the basilar, in 23 on the carotid, in 14 on the anterior cerebral, in 8 on the posterior communicating, in 7 on the vertebral, and in 6 on the posterior cerebellar. It will thus be seen that more than half occurred on the two first-named arteries, a fact of some importance in connection with the relation of symptoms to diagnosis, since the neighbouring parts of the brain may be compressed or indented, atrophied or softened. If adjacent to brain-substance, the aneurysm may by pressure form a recess, in which it lies. Sometimes the bones of the skull may be eroded. Frequently one or more cranial nerves may be compressed by the aneurysm, the pressure resulting in their atrophy; and sometimes a nerve may be found adherent to the wall of the aneurysm. It is not uncommon for a cerebral aneurysm to become filled with a laminated clot, which may result in obliteration of the sac and spontaneous cure; but more often the aneurysm suddenly ruptures, causing death from hæmorrhage.

ÆTIOLOGY.—A number of the new growths are due to congenital developmental anomalies. These include the rare dermoid cysts, teratomata, angiomas, and probably gliomata. Very little is known of the causes underlying cerebral tumours, except those of infective or parasitic origin, nor can we explain why in some people these tumours should occur in the brain and not elsewhere. A neuropathic inheritance plays no essential rôle in the development of cerebral tumours, nor does mental stress; but experience seems to point to the fact that blows and injuries of the head are followed by symptoms indicating the existence of an intra-cranial tumour, although it must be remembered that a certain number of cases, in which apparently a tumour followed a blow or injury of the head, may be explained by the effects of a hæmorrhage into a tumour in which the symptoms were previously absent or ill-defined. This is especially liable to occur in the case of gliomatous tumours. Again, sudden onset of fatal symptoms may follow a blow or injury of the head in aneurysm owing to rupture of the sac. Aneurysm usually is associated with degenerated arteries elsewhere. It occurs especially in syphilitic persons; it may, however, result from infective arteritis, the result of plugging of a cerebral artery by an infective embolus in ulcerative endocarditis.

The *pathological effects* of tumours are related to (1) direct irritation or destruction of adjacent

nervous substance, inflammation, and cedema; (2) destruction of the brain-substance or the cranial nerves by pressure and obliteration of the vascular supply. The disturbance and loss of function occasioned by these processes form the principal guides in inferring the situation and to some extent the size and nature of the growth. But a tumour seated within the cranial cavity by the production of increased intra-cranial pressure causes a number of general symptoms, common to all cerebral tumours, consisting of disturbances in the circulation and the production of internal hydrocephalus.

SYMPTOMS.—As a rule general symptoms precede local, and the onset of both may be slow and gradual; sometimes the symptoms have been so ill-defined as to have been overlooked during life, and the condition has only been discovered *post mortem*. Not infrequently localising symptoms may be entirely absent during the whole course of the disease.

A. *General*.—1. *Headache* is one of the earliest, most constant, and most distressing of symptoms, and is seldom absent during the course of the disease. It may be a dull and continuous pain affecting the whole head, and increased by every forced act of expiration, such as coughing or sneezing. It is the most severe and persistent of all forms of cephalalgia, and even when the patient is in a stuporose or semi-conscious condition, he still manifests by his moaning and the holding of his hands to his head the intense pain which the disease is causing. The pain is subject to exacerbations, which usually wake the patient in the early morning, a symptom which is very frequent in syphilitic tumours; it is often diffuse, sometimes deep-seated and not localised to any particular part of the head. It frequently affects the frontal region, but this does not necessarily show that the tumour is in the frontal lobe; on the other hand, a continuous pain over the back of the head and neck rather suggests the posterior fossæ of the skull as the seat of the tumour, especially if the pain radiates down the spine and upper limbs. This *general* pain, which is due to increased intra-cranial pressure, may be associated with very acute, excruciating, burning *local* pains, limited to a precise circumscribed spot, which frequently indicates the involvement of the meninges by the growth, and therefore its exact situation; over this seat there is often marked tenderness on deep pressure or percussion. Pain confined to one side of the head is usually but not invariably indicative of tumour of that side. All conditions which tend to cause hyperæmia of the brain increase the pain, e.g. alcohol, mental excitement, muscular exercise, forced acts of expiration, and, in some cases, even change of position. The pain persists, however, when all exciting influences are removed, and it cannot be completely and continuously averted by narcotics.

2. *Optic neuritis* is the most important symptom of cerebral tumour, as it is only in isolated cases that this symptom has been proved to have been absent during the whole course of the disease. It occurs at some period before death in three-fourths of the cases, and is generally bilateral, but usually one eye is affected more than the other. It may be present without the patient complaining of affection of sight, and the visual acuity may be quite normal; in advanced cases vision is generally affected and there is an irregular diminution of the

visual field and a lowering of the acuity of central vision, which may go on to complete blindness. Transitory amaurosis may occur, due, not to the direct result of the papillitis, but to internal hydrocephalus, which causes distension of the third ventricle and depression of its floor, thus compressing the optic tract. Post-neuritic atrophy may result; as a rule, however, the production of this takes a considerable time; even when there is atrophy, there are usually signs of papillitis present (see OPTIC NERVE, Diseases of). The causes of changes in the fundus, which English authorities speak of as optic neuritis, and German as Stauungspapilla or choked-disc, is a matter of controversy. There are three views: (1) That it is due to the obstruction to the return of venous blood by the ophthalmic vein, the increased intra-cranial pressure causing compression of the cavernous sinus; but this theory of von Graefe has been disproved by the fact that free anastomosis occurs between the ophthalmic and facial veins. (2) That the intra-cranial pressure interferes with the return of lymph from the sheath of the optic nerve into the sub-arachnoid space, causing hydrops of the sheath, which in turn compresses the veins and obstructs the outflow of blood from the papilla. (3) That the condition is due to irritation and inflammation of the sheath of the optic nerve, produced either by the presence of toxic substances in the cerebro-spinal fluid produced by the tumour, or by direct downward extension of a meningitis. According to Gowers a combination of causes may be in operation. No doubt mechanical stasis in the veins would soon be followed by exudation, and then all the appearances of inflammation would be produced. One argument in favour of the mechanical theory (2) for the explanation of this condition is the fact that choked disc or optic neuritis may be greatly benefited or even disappear after trephining has been performed and the cerebro-spinal fluid allowed to drain off. Some of the cases have been noted as improved in the first day after the operation, and in the course of some weeks the neuritis has completely disappeared. The only conclusion at present possible is that intra-cranial pressure is an essential and fundamental, if not the sole, cause of this important symptom. In many cases of tumour an ampullary swelling has been observed where the sheath is weak at the entrance to the eyeball, but it is often absent, even in acute cases, and the writer has seen it well marked in a case of meningeal hæmorrhage, causing great compression of the cerebral substance. Degeneration of the posterior roots and posterior columns of the spinal cord is sometimes observed and is probably also due to the increased pressure of and chemical changes in the cerebro-spinal fluid.

3. *Vomiting*.—Vomiting is a frequent but not constant symptom. It occurs in rather more than one-third of the cases (Jacoby). When present it is an early and most persistent symptom, and then forms a most troublesome feature of the disease. It may occur without the presence of food in the stomach, e.g. on waking in the morning. It is especially liable to come on when the headache is severe; the tongue, however, usually remains clean, and the appetite may not be much affected. It often ushers in or follows an epileptiform attack. Its cause is probably irritation of the vomiting centre in the medulla, brought about by the increased intra-cranial

pressure or by reflex irritation through the meningeal nerves.

4. *Vertigo*.—Vertigo is usually associated with vomiting, but if severe, and especially if it occurs early in the disease, it must be considered as a special rather than a general symptom, pointing to the existence of a tumour in the cerebellum or its peduncles.

5. *Pulse*.—Permanent or temporary slowing of the pulse occurs pretty frequently. It is usually independent of headache, and, when it occurs as an early symptom, points to a tumour of the medulla; but it may arise as a result of increased intra-cranial pressure, for Horsley has found that after relief of the pressure by trephining, the pulse has quickened. Slowing of the pulse may be accompanied by respiratory disturbances.

6. *Fainting Fits and Apoplectic Seizures* may occur, produced by hæmorrhage into the tumour, causing sudden increase of intra-cranial pressure.

7. *Epileptiform Convulsions*.—Epileptic attacks, with loss of consciousness, may arise in every stage of the disease; although usually a late symptom, epilepsy may belong to the earliest phenomena, in fact may precede other symptoms by many years. A certain number of cases of tumour occur, in which fits form a prominent symptom, although the tumour is not seated in the Rolandic area. The writer has seen three cases of internal hydrocephalus, caused by tumours seated in the third ventricle, in which general convulsions formed a prominent symptom, associated with paroxysmal attacks of drowsy stupor and lethargy. Usually, however, epileptiform convulsions indicate affection of the central convolutions, especially when they are Jacksonian in type; but in slowly growing tumours, symptoms like idiopathic epilepsy may ultimately develop. Instead of typical epilepsy, attacks of simple loss of consciousness may occur, with or without automatic and forced movements. Again, the spasms may resemble an hysterical attack, or simple convulsions may occur with retention or only impairment of consciousness. In tumours of the posterior fossæ tetanic contractions of the muscles of the trunk, with retraction of the head, have been observed.

8. *Psychical Symptoms*.—The expression of the patient is often characteristic of a profound mental change. There is a dull, heavy, drowsy or stuporose condition. The patient in conversation does not reply readily, is listless and easily fatigued, lacks attention, and is unable to collect his thoughts. The speech is slow, sometimes slurred and confluent, and the reaction-time in answering questions is much prolonged. The intelligence often suffers, the memory is enfeebled, and sometimes there is great mental depression, and in rare cases mania. Attacks of mad excitation with delusions and hallucinations may occur, and cause the patient to be taken to an asylum; and it is important to remark in this connection that psychical symptoms may form the first evidence of the brain-disease. Indeed tumours of the brain are not uncommon on the *post-mortem* table at asylums (at least 2 per cent.). Later, as the intra-cranial pressure increases, the symptoms become more marked and the existence of disturbances of consciousness is manifested by the drowsy stuporose expression, by the loss of interest to surroundings, and by the passage of urine and fæces in the bed. In the last stages the loss of consciousness becomes more profound and tends to coma.

9. *General Wasting and Bodily Weakness* occurs, due to the vomiting, headache, and insomnia, and to the knowledge too often of an incurable disease. These together produce a marked change in the metabolism of the whole body, which in a measure is increased by auto-intoxication brought about by obstinate constipation.

10. *Polyuria with or without Polydipsia* may occur, especially in syphilitic tumours of the brain. Glycosuria is met with sometimes and indicates, when other symptoms agree, tumour of the medulla.

B. LOCAL SYMPTOMS AND LOCAL DIAGNOSIS.—Although tenderness on pressure and a high-pitched resonant note over a particular portion of the skull may in some cases afford indication of the existence of a tumour beneath, yet the local diagnosis of the lesion depends mainly upon the regional or focal symptoms caused by direct or indirect involvement of structures possessing particular functions (see fig. 1)

Small tumours, and even tumours of considerable size, may exist in certain regions of the cerebral

patient was trephined over this region, but the *post-mortem* examination showed that this was only one of several tumours.

Tumours of the Hemispheres.—*The motor area.*—A tumour situated in the central convolutions of the Rolandic region of the cortex may produce two classes of localising symptoms: (a) irritative; (b) destructive; the former are characterised by cortical epilepsy, the latter by cortical paralysis, generally a monoplegia. Cortical epilepsy is manifested by tonic followed by clonic spasm affecting one limb, or a portion of the limb, or the face, commencing in a group of correlated muscles, which preside over a representative movement, and spreading in a definite way, according to the situation of the tumour. In a great number of cases the muscular spasm is preceded by sensory phenomena, such as a feeling of cramp or numbness, and tingling of the skin of the parts. The march of the fit is of great importance to observe, for it will commence in the same region and spread in the same way, e.g.

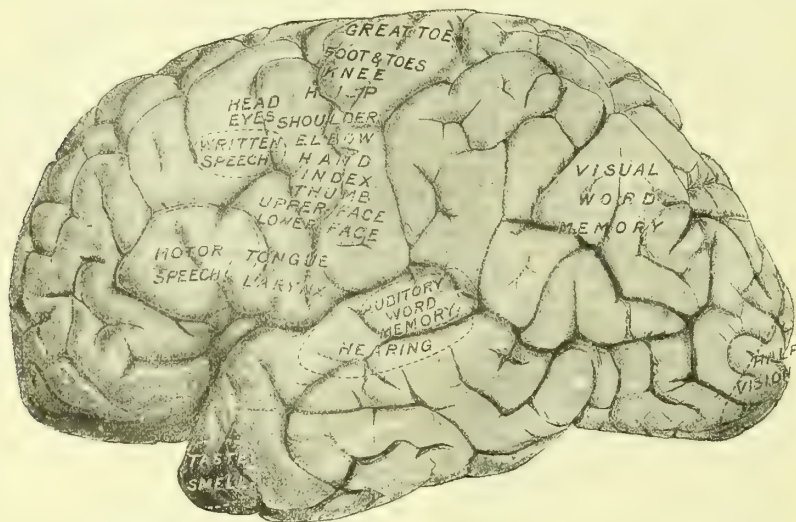


FIG. 1.—Left hemisphere, showing the situation of the cortical projection-centres

hemispheres, especially the right, without giving rise to any symptoms other than those due to increased intra-cranial pressure. Again, tumours such as the infiltrating gliomata may be situated in the white substance of the hemisphere, and either give rise to *no* localising symptoms, or by pressure, extension, or hæmorrhage, affect structures with particular localising functions situated in the neighbourhood, or even at some considerable distance. A false notion of the position and size of the tumour may thus arise. For example, a large tumour in the right frontal lobe might extend to the motor area, and the first localising symptom might be Jacksonian epilepsy. Likewise, multiple tumours may exist, but only one of these be situated in a region which would occasion localising symptoms; consequently an erroneous prognosis as to the beneficial result of operative procedure might be arrived at. The writer recently saw a case of multiple tubercular tumours in which the most prominent localising symptoms were cortical epilepsy, starting in the right facial muscles, and partial aphasia. The

it may begin in the face, indicating involvement of the lower portions of the Rolandic region; in the fingers, wrist, thumb, or shoulder, indicating the middle portion; or in the toes, ankles, or leg, indicating the upper portion of the central convolutions (see fig. 1).

The muscular spasm usually spreads rapidly over the whole half of the body and may extend to the other side and become general. Consciousness may remain throughout the fit, especially if there is not much spread. When both sides of the body are involved in the muscular spasm, consciousness is lost. It is important to determine the state of the parts affected after the convulsion, and to note whether there is paresis or anaesthesia. Destructive lesions produce usually a monoplegia; thus monoplegia cruralis, brachialis, and facialis are especially symptomatic of tumour. As the tumour grows the paralysis may increase, and the monoplegia become a hemiplegia. Sometimes with the hemiplegia there is a blunting of sensibility, especially of the sense of touch and the sense of position.

Pre-frontal Region.—A tumour situated in the frontal lobe of the left hemisphere may cause aphasia (partial or complete) when Broca's convolution is involved. Again, from proximity to the motor area, the tumour may give rise to cortical epilepsy. Tumours which grow from the orbital surface of the left frontal lobe may attain a considerable size without giving rise to any affection of speech. It is to be remarked that sometimes the speech-affection arising from a tumour in the left frontal region may take the form of bradylalia. Anosmia from compression or destruction of the olfactory nerve or tract sometimes occurs in tumours of the frontal lobe, and when it can be determined, is of value as a localising symptom. Bruns has pointed out that tumours of the frontal lobe often cause ataxy of the cerebellar type, and thus tend to simulate cerebellar tumours, but this form of ataxy is frequently associated with monoparesis or hemiparesis. Optic neuritis occurs late in the course of the disease, whereas mental dulness, stupor, and a condition resembling imbecility are early symptoms (fig. 2).

Parieto-occipital region.—Tumours often attain a considerable size in this region without producing definite localising symptoms. When the tumour is situated in the left hemisphere, involving the angular gyrus, word-blindness and mind-blindness may occur. If it be situated in the occipital lobe, causing destruction of the optic radiations or the centre for half-vision, hemianopsia would be the result; or if it were an irritative lesion, starting in the meninges, it might give rise to unilateral visual hallucinations.

Temporal lobe.—Important localising symptoms only arise when the tumour is situated on the left side; if it affects the posterior two thirds of the first temporal convolution, word-deafness, amnesia, or paraphasia may result. Occasionally auditory hallucinations occur in irritative lesions of this region.

Parietal lobule.—Tumours in this portion of the brain may cause compression of the Rolandic area, with resulting motor irritation or paralytic phenomena, accompanied by disturbances of sensibility and of the sense of position.

Corpus callosum.—The symptoms of tumours in this situation are not very definite, but, according to Bristowe, the following symptoms indicate a tumour in this region: (1) disturbance of intelligence or stupor, combined with a non-aphasic disturbance of speech; (2) absence of cranial nerve-affections; (3) hemi-parietic phenomena combined with a less-marked paresis of the other side of the body; (4) gradual progressive increase of these symptoms; (5) absence, as a rule, of the general symptoms of tumour.

Basal ganglia.—Tumours in the *corpus striatum* may occur without producing any definite symptoms,

but hemiplegic symptoms may be produced owing to direct or indirect involvement of the internal capsule. Tumours of the *optic thalamus*, for the same reason, usually give rise to hemiplegia frequently associated with hemi-anæsthesia, hemiathetosis, and sometimes hemianopsia, from involvement of the optic radiations.

Tumours of the *third ventricle* may present no definite localising symptoms, but the general symptoms may be very manifest, such as amaurosis, headache, dulness, stupor, and vomiting, due as a rule to internal hydrocephalus. In four cases of tumour of the third ventricle recently seen by the writer, the symptoms were recurrent attacks of drowsy stupor, which sometimes deepened to coma, preceded by restlessness and sometimes by convulsions, general muscular weakness, progressive mental enfeeblement, and optic neuritis.

Cerebellum.—The symptoms of tumours in this situation are ataxy, characterised by a peculiar oscil-

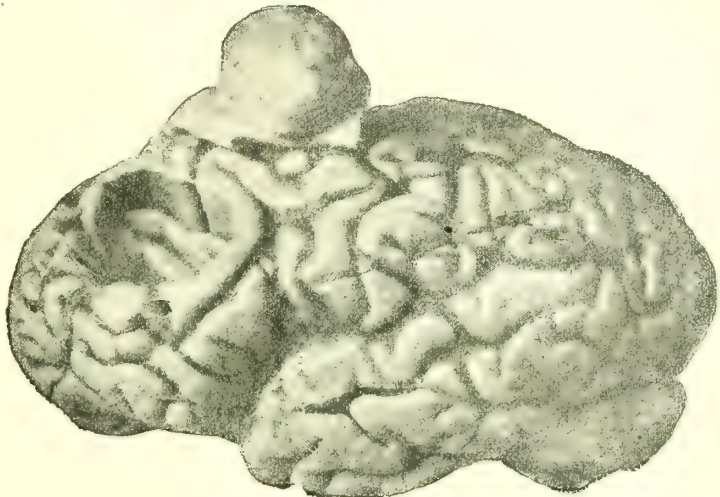


FIG. 2.—Fibro-endotheliomatous tumour growing from the Dura Mater, and pressing on the Frontal Convolution. There was no paralysis during life, nor were signs of intra-cranial pressure observed, probably owing to the very slow growth. Patient was subject to fits and was demented.

lating gait, like that of a drunken man, nystagmus, vertigo and tendency to fall in one direction; muscular weakness on the same side as the lobe of the cerebellum affected; headache, especially over the back of the head, combined with stiffness and rigidity of the neck. Vomiting and slow pulse usually occur. Whereas optic neuritis usually occurs early and is very marked, mental symptoms in the form of apathy, dulness, and stupor develop late, affording thus a contradistinction to frontal tumours.

Tumours of the *base of the brain* are frequently met with. They are usually syphilitic, sarcomatous, or tubercular, but a number of rare tumours may also arise, especially in the neighbourhood of the pituitary fossa. It has already been stated that aneurysms commonly affect vessels of the base of the brain, and may consequently cause symptoms resembling those of a tumour situated in this region.

According to the seat of the tumour and its size, so various structures about the base of the brain are affected, and it is not surprising to find that among the commonest symptoms of tumours in this situation are paralyses of the cranial nerves. The nerves

supplying the eye-muscles are especially liable to be affected, causing various forms of squint and double vision, and ptosis associated with paralysis of the sphincter of the iris, if the third nerve is involved. The optic tract may be compressed and cause hemianopia; or there may be pressure on one optic nerve, when there will exist unilateral symptoms and changes in the fundus oculi; or the optic chiasma may be compressed, as frequently occurs in tumours of the hypophysis cerebri, resulting in bi-temporal hemianopia. If the fifth nerve is involved, there is unilateral anaesthesia of the face, loss of taste and smell on one side, and sometimes sloughing of the cornea. The facial nerve may be affected, and usually the auditory is associated in the destructive process, deafness and peripheral facial paralysis resulting. In such case the muscles of the upper part of the face will be involved as well as the lower; the eye will remain open in sleep, and the muscles will show qualitative and quantitative alterations in the electrical reactions. Some degree of pressure upon the crus, pons, or medulla may be associated with these cranial nerve-paralyses, causing weakness or paralysis of one or both sides of the body, from pressure upon the pyramidal systems of fibres.

Crura cerebri or corpora quadrigemina.—A tumour in this situation is very liable to involve one or more of the nuclei of origin of the third nerve, or the nerve itself. The so-called 'syndrome of Weber' may occur, namely, paralysis of the motor oculi of the side on which the growth is situated, and complete hemiplegia of face, tongue, and limbs of the opposite side. If the corpora quadrigemina are involved, there is usually loss of pupil-reaction, nystagmus, and ataxy.

Pons and Medulla.—Tumours of the pons may be unaccompanied by optic neuritis. Paralyses of the cranial nerves of one or both sides having their nuclei of origin in this situation, associated with paralysis or paresis of the face and limbs according to the size and seat of the tumour, indicate affection of this region. Especially characteristic is the alternate hemiplegia, in which the facial, abducens, and trigeminal nerves, or one of these nerves on one side and the limbs on the opposite side, are paralysed. When there is double hemiplegia, there is usually dysarthria and dysphagia.

Medulla.—Tumours of the medulla may produce symptoms somewhat similar to those of a tumour affecting the lower part of the pons, except that the eighth to the twelfth cranial nuclei may be damaged and cause deafness, dysarthria, dysphagia, aphonia, disturbances of the heart's action and of the respiration, with hicough, and (in many cases) glycosuria. The limbs may be paralysed on one or both sides, and sometimes there is cerebellar ataxy.

DIAGNOSIS.—Three points must be considered: (1) The existence of a cerebral tumour; (2) its situation; and (3) its probable nature. The existence of a tumour is probable when there are severe headache, characteristic vomiting, optic neuritis, and slow pulse. The situation must be inferred from the consideration of the localising symptoms already discussed, and the nature of the growth may be sometimes correctly diagnosed or guessed at by considering the history of the onset and progress of the disease, together with the previous health, sex, and age of the patient. A number of cerebral tumours, e.g. psammoma, lipoma, cholesteatoma, and cysts may remain latent,

and give rise to hardly any symptoms. When a patient comes complaining of severe and continuous headache, the possibility of a tumour must be considered, and an examination of the fundus oculi should always be made. Should neuritis be found to be present, it is necessary to exclude other possible causes, e.g. meningitis, cerebral abscess, hydrocephalus, and nephritis, for some forms of Bright's disease are associated with severe headache and retinitis. It is necessary to remember that in marked chlorotic anaemia, a patient may suffer with severe headache, and an examination of the fundus may even reveal in rare cases neuritis. In chronic lead-poisoning, many symptoms occur which might lead to a mistaken diagnosis of tumour: thus, there may be headache, optic neuritis, and cortical epilepsy. Migraine, hysteria, or neurasthenia may be the cause of severe headache, but the headache in these affections is profoundly influenced by psychical conditions, and may even disappear when the attention is diverted. Stupor, drowsiness, progressive dementia, and attacks of maniacal excitement or melancholia may occur in patients suffering with tumour, and lead to their being certified as insane.

A tumour with focal symptoms, causing Jacksonian epilepsy, must be differentiated from uraemia, alcoholism, lead-poisoning, syphilitic and tubercular meningitis-encephalitis, and in rare cases chronic brain-softening, all of which affections may be attended by convulsive epileptiform seizures. Absence of the general symptoms of tumour, and the history of the case and its progress, will help in coming to a decision; and it is especially in such cases that tenderness and alteration of the note on percussion will be valuable as an aid to diagnosis. In dementia paralytica the patient often complains of headache, and he may be the subject of unilateral fits, but it is very seldom that an examination of the fundus oculi reveals any optic neuritis.

In general paralysis of the insane a sluggish reaction of the pupils to light or the Argyll-Robertson pupil is seldom absent.

COURSE AND PROGNOSIS.—The course of the disease is usually chronic and may extend over years. Some forms of tumour are slower in growth than others, e.g. glioma, osteo-sarcoma, and 'solitary tubercle'; others, e.g. the soft sarcomata and carcinomata, are very rapid. If syphilis can be excluded, the prognosis is most unfavourable. As a general rule, death takes place in one or two years, but occasionally tubercular tumours become stationary, and even undergo regressive metamorphosis. When once the symptoms have become pronounced, death usually takes place in a comparatively short time from exhaustion, coma, or some intercurrent disease, but it may be sometimes quite sudden and unexpected. Some forms of tumour, e.g. angioma, cholesteatoma, and glioma, may remain latent for many years, and then an acute onset of symptoms which rapidly terminate fatally may arise.

TREATMENT.—*General Management.*—It is well in all cases to try mercurial inunction and large doses of iodide of potassium upon the chance that the tumour may be syphilitic. In some cases mercurial inunction over the painful spot on the head may give relief. Allen Starr recommends large doses of iodide of potassium in non-syphilitic tumours, and he has recorded cases which have apparently derived much benefit from heroic doses of this drug. He has given as much as 250 grains

a day. If the tumour is suspected to be tubercular, treatment usually adopted for this disease may be tried, such as cod-liver oil and tonics. Again, liquor arsenicalis as a nerve-tonic may be prescribed. All these remedies however are as a rule of little use, and, unless operative procedure be adopted, the most that can be done is to treat the symptoms and relieve the sufferings of the patient. As already pointed out, most of the drugs which are used for the relief of headache (phenacetin, antipyrine, exalgin, &c.) are of little use, and the only remedy which is of service is morphine or some preparation of opium. The patient may have either hypodermic injections of morphine or some preparation of opium administered internally. Gowers recommends cannabis indica as a narcotic. Leeches to the painful part, also ice-bags accompanied with hot bottles to the feet, and purgatives may help to relieve pain by drawing the blood from the head and relieving intra-cranial pressure. The convulsions as a rule are not relieved by bromides; chloral hydrate appears to be more effective, and inhalations of chloroform in some cases have to be resorted to. The vomiting may be most troublesome, and may necessitate feeding by nutrient enemata. Ice can be ordered to be sucked, opium given, and rest in bed upon the back should be enjoined.

Operative Measures.—The results are not so satisfactory as at first this branch of surgery promised. Many patients die of shock. In only a limited number of cases can the tumour be removed without risk of return, and, not infrequently, the patient is left paralysed, speechless, or mentally afflicted. If the wound becomes septic, hernia cerebri or meningitis is very likely to occur. In recommending operation all these facts should be taken into account, and the surgeon should be guided by the following considerations: (1) that, according to the statistics of Allen Starr, only 7 per cent. of cerebral tumours are suitable for operation, (2) that it is difficult to be sure of correct localisation, (3) that if the patient is suffering from severe symptoms arising from intra-cranial pressure, although it may be impossible to remove the growth, many of the distressing symptoms of the disease may be relieved by operation. If operation is decided upon, its early performance is advisable, and it is essential to success that it should be done in a hospital, and only by those who have had considerable practice in the performance of such operations. In this way delay is avoided, shock, which is the most dreaded feature, is obviated, and complications and hæmorrhage are more skilfully met.

FREDERICK W. MOTT.

BRAIN, Ventricles of, Diseases of.—See VENTRICLES OF BRAIN, Diseases of.

BRAIN, Vessels of, Diseases of.

1. **Aneurysm.**—The larger arteries of the brain, and their minute branches in the cerebral substance, are both liable to aneurysmal dilatation. The 'miliary' aneurysms on the smaller branches within the brain, which are the cause of cerebral hæmorrhage, are described later.

(a) *Aneurysm of the larger cerebral arteries* is more common than that of those of a similar size elsewhere. The large vessels of the base, or their primary branches, may be affected. The basilar and middle cerebral arteries are those most frequently diseased, aneurysms of these two vessels

constituting more than half the cases. Next in frequency is the internal carotid. The vertebral, anterior and posterior cerebrals, anterior and posterior communicating, and anterior cerebellar arteries are occasionally, but less frequently, affected. In one or two recorded cases the aneurysm has been situated in the interior of the pons Varolii or cerebellum. The disease is rather more frequent on the left side, but this is chiefly due to the greater proclivity of the left middle cerebral compared with the right, a fact which is explained by the ætiology. There is occasionally more than one aneurysm, situated on different arteries, or on different branches of the same artery. The aneurysm is usually sacculated, rarely dissecting. Its size usually varies from that of a pea to that of a hazel nut, but aneurysms have been met with as large as a hen's egg. When the size is considerable the brain-tissue is pressed upon and softened, as by any other kind of tumour.

ÆTIOLOGY AND PATHOLOGY.—These aneurysms are rather more common in men than in women, after actual childhood, and are far more frequent before the ordinary degenerative period than aneurysms elsewhere. Nearly half the recorded instances have occurred between ten and forty years of age, and about one-sixth between ten and twenty. The change in the arterial wall resembles that giving rise to aneurysm elsewhere—a fibroid degeneration, with loss of muscular and elastic tissue. Distensible tissue, which yields permanently before the blood-pressure, is substituted for the resilient elements of the wall, which, normally, bring back the vessel to its previous size after each expansion. The alteration is occasionally part of a widely spread arterial change, but more frequently is local. It is then due to some local morbid process, usually one of three kinds: (1) syphilitic disease, which has altered the wall, but has not obliterated or greatly reduced the lumen of the vessel; (2) injury—a blow or a fall on the head, which probably acts by causing a local periarteritis, since extensive traces of old inflammation are sometimes found in the neighbourhood of the aneurysm in such cases; (3) incomplete embolism, imperfect occlusion of the vessel by a plug, from a valve affected with active endocarditis. This cause explains the greater frequency of aneurysm in the left than in the right middle cerebral artery (since this is more prone to embolism), and also the fact that those who suffer from cerebral hæmorrhage at an earlier age than degeneration occurs usually have valvular disease of the heart: in such cases an aneurysm is often found. Whatever be the cause of the alteration in the arterial wall, the efficient agent in the production of the aneurysm is the high blood-pressure in the cerebral arteries, coupled perhaps with a slighter external support than is possessed by the vessels in other parts. See ANEURYSM.

Rupture has occurred in more than half the recorded cases of cerebral aneurysm, and probably occurs in a much larger proportion, since a small aneurysm is easily overlooked in a large extravasation. The blood may escape rapidly or slowly, and the hæmorrhage may take place into the subarachnoid space, or into the adjacent cerebral substance. In the former case, meningeal hæmorrhage is the result. Rupture into the cerebral substance is not uncommon. An aneurysm in the fissure of Sylvius may cause a hæmorrhage into the substance of the brain, ultimately bursting into the lateral

ventricle; and an aneurysm on the posterior cerebral artery may burst into the substance of the pons. Two causes may determine this rupture into the substance of the brain—first, thickening of the subarachnoid tissue adjacent to the aneurysm, and especially over it, hindering its rupture outwards; secondly, the small size of the opening and consequent gradual escape of the blood, producing a slow disintegration of the brain-tissue, in the direction of least resistance. The amount of blood which is effused is often very large, and may make it uncertain from what artery it has come, and the difficulty of discovering an aneurysm is thereby increased. In rare cases a communication forms with a venous sinus, and constitutes an arteriovenous aneurysm. This has occurred between an aneurysm of the internal carotid and the cavernous sinus.

SYMPTOMS.—Symptoms of the existence of an aneurysm may be entirely absent. When present, they depend on the pressure which the tumour exerts on neighbouring parts. They vary widely according to its seat, and they are rarely by themselves distinctive. Mental disturbance is uncommon. Headache is a very frequent symptom. It is often intense, sometimes throbbing, and may be localised, as to the occiput in basilar aneurysm. Giddiness is not uncommon, and is complained of in aneurysms in all situations. Convulsions are not frequent except in cases in which the aneurysm is in the neighbourhood of the motor region of the cortex, i.e. is on the middle cerebral artery or its branches. Palsy in some situation often occurs, and depends on the pressure of the tumour; the most common is that of the cranial nerves which lie adjacent to an aneurysm at the base, as of the nerves of the orbit in aneurysm of the internal carotid. Optic neuritis is only occasionally present. The symptoms are thus those of an intracranial tumour, and they suggest an aneurysm when they are such as to show that the tumour is in the position of one of the arteries liable to be affected; and the probability is increased if one of the known causes of aneurysm can be traced. If there is a history of syphilis the diagnosis lies between an aneurysm and a gumma, and the former is only probable if the symptoms are not lessened by appropriate treatment. The presence of heart-disease has less often definite diagnostic importance than its causal relation might suggest, because embolic aneurysms usually rupture before causing pressure. Injury, it must be remembered, may also cause a neoplastic tumour. Aneurysms elsewhere would, of course, give great additional significance to the symptoms, but the coincidence is too rare to be of practical importance. In some cases a murmur can be heard by the patient, and in still rarer cases (of aneurysm of the internal carotid) it has been audible on auscultation. But even an objective murmur, alone, cannot afford actual proof of the existence of an aneurysm, since pulsation, the chief sign of an accessible aneurysm, can never be perceived.

Rupture of an intracranial aneurysm gives rise to symptoms which vary according as the blood escapes quickly or slowly. If quickly, the blood usually passes into the meninges and causes sudden apoplexy with general paralysis, rapidly deepening to a fatal issue. If slowly, the symptoms are less sudden, and unilateral paralysis or convulsions may occur. This is especially the case when the blood escapes

slowly into the cerebral substance; the unilateral symptoms commence suddenly, and gradually increase during a few hours or days, with or without initial loss of consciousness, but ending in fatal coma.

TREATMENT.—Little can be done in cases where intracranial aneurysm is suspected. Even when it is of syphilitic origin, drugs are powerless to alter the damaged and dilated vessel, since the change which has given rise to it is of the nature of a cicatricial transformation of the syphilitic tissue, on which drugs can no longer exert the influence they possess over the earlier stages of the growth in the wall. Hypodermic injection of ergotin ($\frac{1}{8}$ -grain) has been recommended by Langenbeck and others. Iodide of potassium may also be given in the hope of promoting coagulation within the sac, from the undoubted evidence of the power it has to effect this in aneurysms elsewhere. This is also true of the hypodermic injection of a solution of gelatine, if its alleged influence is confirmed (*see* HYPODERMIC MEDICATION). Rest is important. All causes of increased intravascular pressure, such as effort and low positions of the head, are to be avoided. The bowels should be kept regular. In rare cases where progressive paralysis of orbital nerves suggests the probability of aneurysm of the internal carotid, and a murmur renders the diagnosis certain, ligature of the common carotid may be, and has been, resorted to with success. It is, moreover, probable that intracranial surgery may be able to cope with aneurysms on some other vessels, but on this point experience is still to be gained.

(b) *Minute 'miliary' aneurysms* occur in the small arteries of the pia mater and substance of the brain, and are the chief cause of cerebral hæmorrhage. They are met with almost exclusively in the second half of life, and increase in frequency with age up to about seventy-five, the less frequency of cerebral hæmorrhage in extreme old age making it probable that they are less common in very late life. They may involve vessels not more than the $\frac{1}{100}$ inch in diameter, but are most common on vessels a little larger than this. The walls suffer fibroid degeneration of the outer and middle coat, commencing, it is said, as nuclear proliferation. The muscular tissue of the middle coat disappears, and distensible tissue replaces it. Hence under the influence of the blood-pressure, which is high in the arterioles prone to be thus changed, the artery at the spot becomes dilated into a sacculated aneurysm, varying in size from the $\frac{1}{25}$ to the $\frac{1}{35}$ of an inch. These dilations have been found in all parts, but most frequently in the central ganglia, and next most frequently in the pons Varoli, the convolutions, the cerebellum, the medulla oblongata, the cerebellar peduncles, and the centrum ovale. They are to be found in most cases of cerebral hæmorrhage; and it is probable that such hæmorrhage is due to their rupture. Similar minute aneurysms of the retinal arteries have been observed to co-exist in rare cases.

The rupture of a minute artery into its perivascular sheath distends this with blood, causing what has been termed a minute dissecting aneurysm. Such are frequently met with in cases in which the vessels are exposed to extreme pressure, as in death from asphyxial conditions; or in the increased tension in collateral vessels when vascular obstruction has occurred.

SYMPTOMS.—No symptoms are known to be

associated with the existence of these minute aneurysms, and this fact explains the absence of premonitory indications of a liability to cerebral hæmorrhage. The symptoms of rupture are described under BRAIN, Hæmorrhage into.

2. **Degeneration.**—(a) *Of Arteries.*—The larger cerebral arteries are very common seats of degenerative changes which are fully described in the article on ARTERIO-SCLEROSIS. The cerebral arteries, however, are peculiarly liable to these changes. The cause of this liability is probably the strain to which the badly supported cerebral vessels are exposed in consequence of their direct origin from comparatively large vessels, rendered more effective by the deficient support from adjacent structures. This deficiency increases as life goes on, because the atrophy of the brain-substance leads to an increase in the size of the peri-arterial spaces. It is not easy to explain the occasional freedom of these vessels from atheroma when this is abundant elsewhere.

Degeneration of the cerebral vessels may lead to anæmia of the brain, rupture of the vessels, or thrombosis in their lumina. Anæmia has already been considered (*see* BRAIN, Anæmia of); rupture and thrombosis will be dealt with in the present article.

SYMPTOMS.—Atheroma of the cerebral vessels leads to the symptoms of local anæmia of the brain, and is a common cause of the transient cerebral symptoms so frequent in the old, before a definite seizure occurs. Tingling in the limbs of one side, and slight loss of power, as well as more general symptoms, as headache, may be thus produced, and are frequently the precursors of an attack of hemiplegia from the closure of the vessel. It is possible also that the loss of memory and mental failure common in the old may be the result of more general interference with the supply of blood to the brain in consequence of this arterial disease, but, without symptoms of sudden onset, it is more often due to senile failure of cerebral nutrition.

TREATMENT.—Tonics, especially cardiac, and substances which, as cod-liver oil, promote the nutrition of the tissues generally, may be given with possible advantage. It is not likely, however, that any treatment can modify the state of the arterial wall, or that its tissue ever resumes, in any degree, its normal characters. The maintenance of the general nutrition in a good state may lessen the tendency to fatty degeneration and subsequent calcification of the tissue, and thus postpone or prevent the disastrous consequences of occlusion of the vessels. In this, however, we can only adjust our treatment on rational grounds, since the nature of the case precludes any possible evidence of beneficial result. Of especial importance is it, in any case in which there is reason to believe or to suspect the presence or future development of atheroma, to do all in our power to keep the arterial tension within normal limits, and to preserve the blood from the presence of the products of imperfect digestion and the accumulation of metabolic products, which favour thrombosis. It is important to adopt in the old a regimen and treatment likely to lessen the gouty tendency which so often attends atheroma and often increases the tendency to thrombosis.

(b) *Of Veins.*—Degeneration of the walls of the veins is much less frequent than degeneration of the arteries, perhaps on account of the less degree of pressure to which they are exposed. Disease of

their walls is generally the result of inflammation spreading from without. Thrombosis in them may thus result from adjacent inflammation, tubercular or other. Occasionally the veins of the pia mater may be found varicose in advanced life, and in one case rupture of such a dilated vein was the cause of meningeal hæmorrhage.

3. **Embolism.**—**DEFINITION.**—The obstruction of arteries or capillaries of the brain by solid particles carried by the blood-current from some other part of the vascular system.

ÆTIOLOGY.—The source of the embolic particles is almost invariably situated between the pulmonary capillaries and the obstructed vessels, i.e. in the pulmonary veins, the left side of the heart, or the arteries. In arterial embolism it is necessarily so, since no particles large enough to obstruct even a small artery could pass through the capillaries of the lungs. In almost all cases the heart is the source of the plug, a particle of fibrin being washed by the blood from a deposit on a diseased valve or in some recess (as the auricular appendix). Endocarditis, or chronic valvular disease, therefore, usually co-exists with the embolism. Mitral stenosis is an especially frequent source of emboli, probably because the surface is commonly much altered, and the blood-current is in part slow (in diastole, allowing deposit), and in part very rapid (in auricular systole, detaching loose particles). Moreover the dilatation of the left auricle, and the imperfect degree in which the blood is expelled from it, frequently lead to a coagulum in the appendix. Disease of the aorta—atheroma or aneurysm—is the next most frequent source, and, less frequently, disease of the carotid or vertebral arteries, and coagulation in the pulmonary veins, large or small—the latter in some rare cases of inflammation and growths.

Particles obstructing capillaries may come from some softened atheromatous patch or fibrinous deposit, from pigmentary formations, or from deposits in ulcerative endocarditis. In the last case the obstructing material has a septic character, probably due to bacterial organisms, and the inflammation it causes may be suppurative.

Embolism occurs with equal frequency in both sexes, and at all periods of life, but most frequently between later childhood and middle life. This is explained by the fact that it occurs more readily from valvular disease due to endocarditis than from that which results from the degeneration of age, but most frequently when the lapse of time has led to secondary changes in the valves damaged, or during second attacks of endocarditis—inflammation of valves previously diseased being especially prone to give rise to embolism. Hence most subjects of embolism have suffered or are suffering from one of the diseases known to cause endocarditis, especially from rheumatic fever, occasionally from chorea or scarlet fever.

ANATOMICAL CHARACTERS.—Almost any of the cerebral arteries may be obstructed; but the vessels most prone to suffer are the middle cerebral, in consequence of the more direct course of the circulation to them. Next in frequency are the internal carotid and the anterior cerebral. Embolism is less frequent in the arteries to which the vertebrals convey blood, but has been met with in most of them; even the basilar may be obstructed by a clot small enough to pass through the vertebral, but too large to enter the posterior cerebral.

The cerebellar arteries are those less frequently affected. Obstruction of several vessels is sometimes found, having occurred at the same or different times. Embolism is more common on the left side, but the difference is not so great as is often stated, being not greater than as four to three. It is due to the more direct origin of the left carotid, into which a plug can therefore be more easily carried than into the right; hence it depends on the greater frequency of the occlusion of the branches of the internal carotid, and especially (because it is the vessel most often affected) of the middle cerebral. The left vertebral is also rather more often plugged than is the right, but embolism of this artery is too rare materially to influence the numerical relation between the vessels of the two sides in their liability.

The plug is usually arrested at some spot at which the vessel is narrowed by a branch being given off. Here the fragment may be found, usually decolourised, and commonly closing altogether the lumen of the vessel. On each side of this is a secondary clot, distinguished by its darker tint: the distal extends far into the contracted branches of the vessel, the proximal as far as the next large branch. The obstruction may lead to inflammation of the wall of the vessel at the spot, especially when the plug has been carried from a place at which inflammation is going on. The inflammation leads to change of texture and degeneration, fibroid or fatty; the former, when the obstruction is incomplete, may permit an aneurysm to be formed, the latter may cause a thickened patch, in which calcification may occur. The inflammation may spread to the adjacent tissue, leading to induration around the spot. It is most intense when the plug comes from a valve the seat of active endocarditis, and may then determine a peculiar irritative character in the secondary inflammation, as is shown by the fact that optic neuritis sometimes develops in such cases.

The effect of embolism is to arrest the blood-supply to the part of the brain to which the artery is distributed, unless the blood can reach it by some other channel; the cerebral tissue, deprived of its nutrition, undergoes necrosis and softening (*see* BRAIN, Softening of). This result, however, is often prevented by the anastomoses that exist in many parts, and as these are very irregular in the cortex of the brain, both in different persons and in different parts of the same arterial area, considerable variations exist in the extent of softening that results from occlusion of the same artery. But in the central ganglia anastomoses are absent, and softening uniformly follows. Thus embolism of the trunk of the middle cerebral may cause softening of the corpus striatum, while the cortex supplied may be intact, or softened in irregular areas, or it may be totally destroyed. Capillary embolism also causes softening, and when the obstruction is from a septic source, the inflammation adjacent may be suppurative, and 'metastatic abscess' may result. Thus cerebral abscess results from suppurative disease in the thorax and other parts. One of the rare curiosities of pathology is the production of such abscess by the embolic obstruction of the vessels with fragments of *Oidium albicans* gaining access to the circulation from the throat. A plug of fibrin lodging in an artery sometimes quickly breaks up, and is carried into the

capillaries, so that the artery is pervious, although the brain-tissue it supplies is necrosed. Necessarily, when the capillaries are obstructed, a collateral blood-supply is of no avail to prevent softening.

For the SYMPTOMS, DIAGNOSIS, and TREATMENT of cerebral embolism, *see* BRAIN, Softening of.

4. **Rupture.**—Rupture of cerebral arteries is common and is the cause of cerebral hæmorrhage; rupture of capillaries is not infrequent. Rupture of veins is extremely rare, except as the result of injury.

(a) *Of Arteries.*—The proximate causes of rupture are weakening of the arterial wall, and increased pressure within the vessel. The conditions which give rise to these two factors are the remote causes of rupture. The actual rupture is often due to a temporary sudden excess of intravascular pressure, but this often does not immediately precede the symptoms, which are only manifest when the initial opening in the wall has increased.

ÆTIOLOGY.—The wall of the vessel is weakened, especially by degenerative disease. Aneurysmal dilatation and thinning have usually resulted from the chronic change. In some diseases attended with a tendency to extravasation (purpura, hæmophilia, &c.) it is probable that the vascular walls undergo rapid degeneration in the acquired, and are unusually thin in the congenital maladies. Defective external support, from atrophy of the brain, causing increased size of the perivascular canals, was formerly thought to be a potent cause, and is now perhaps underrated. The mobile perivascular fluid which surrounds the vessels must afford a less efficient support than cerebral tissue, and sudden variations in the size of these canals will expose the vessels to sudden differences in the amount of tension to which their walls are exposed.

When vessels are much weakened, they may rupture when the intravascular pressure is at, or even below, the normal; very commonly, however, there co-exists increased pressure. Loss of arterial elasticity leads to less equable pressure. Arterial degeneration, and still more constantly the contraction of the minute arterioles, in Bright's disease, cause increased tension by obstruction; and the hypertrophy of the heart, which develops to overcome the obstruction, adds materially to the pressure within the arteries. Hypertrophy to overcome an obstacle near the heart has probably no influence in causing rupture of cerebral vessels.

The immediate cause of rupture is generally some temporary increase of the blood-pressure due to effort—as in cough, straining at stool, or vomiting; excited action of the heart due to exertion or emotion; suddenly developed heart- or lung-disease obstructing the circulation; local obstruction to return of blood; or the action of gravitation in the recumbent posture. The last cause probably determines the frequent occurrence of rupture during sleep in which contraction of the arterioles is probably added.

The conditions which produce these proximate causes are the remote causes of rupture. The most efficient are those which lead to arterial degeneration (*see* ARTERIO-SCLEROSIS). In addition to these certain local considerations must be borne in mind. The position of degeneration is probably largely influenced by the distribution of the vessels; and the latter may be hereditary, as the retina

sometimes strikingly shows us: the same arrangement of the branches of the retinal artery may be seen in parent and child. So, too, correspondence in the way in which the cerebral arteries of the two sides divide and are distributed may be observed to coincide with a close correspondence in the position of spots of visible degeneration, and doubtless also determines a like symmetry in the invisible degeneration of the minute aneurysms; hence rupture sometimes occurs at the same place in each hemisphere.

Some of the cerebral arteries give way more frequently than others, especially the arteries of the corpora striata and pons Varolii. This seems due (1) to their direct origin from vessels of very considerable size (basilar and middle cerebral), and their consequent exposure to the full pressure within the parent-trunk; and (2) to their 'terminal' character, which precludes collateral relief when a general increase of intra-arterial pressure augments disproportionately that in an artery in which it is normally above the average for the size of the vessel. One artery, which very frequently gives way, passes from the middle cerebral through the anterior perforated spot, onwards between the island of Reil and the lenticular nucleus, the outer part of which it perforates, and then passes through the white fibres of the 'internal capsule,' between the lenticular and caudate nuclei, to ramify in the anterior part of the latter. The arterial branches supplying the convolutions on the surface of the brain are not often ruptured, except from injury. They are exposed much less directly to the blood-pressure, and sometimes possess considerable anastomoses.

SYMPTOMS.—The consequence of rupture of an artery is cerebral hæmorrhage, the symptoms and treatment of which are described elsewhere (see BRAIN, Hæmorrhage into; and BRAIN, Injuries of).

(b) *Of Capillaries.*—The minutest vessels and the capillaries rarely rupture, except when exposed to sudden pressure by venous thrombosis; the obstruction in the minute vessels from which the vein carries away the blood may determine numerous capillary hæmorrhages into the cerebral substance. In general intense venous congestion, as in asphyxial states, such hæmorrhage may occur; and a more frequent result is extravasation into the perivascular sheath, which thus becomes distended with blood.

(c) *Of Veins.*—Rupture of veins is only the result of intense mechanical congestion, such as violent paroxysms of whooping-cough or intense asphyxia, as from a violent epileptic fit. The effects are the symptoms of a local cerebral lesion especially of the cortex, sometimes perhaps of the cerebellum.

For SYMPTOMS see BRAIN, Hæmorrhage into.

5. **Syphilitic Disease.**—See BRAIN, Syphilis of.

6. **Thrombosis.**—Thrombosis is the coagulation of the blood *in situ*. It may occur in the cerebral arteries, or in the cerebral veins and sinuses.

(a) *In the Arteries.*

ÆTIOLOGY.—The causes of arterial thrombosis are considered in the article on THROMBOSIS.

ANATOMICAL CHARACTERS.—The arteries occluded may be one or several, large or small. Thrombosis occurs most frequently in the internal carotid, middle cerebral, vertebral, and basilar arteries, or their branches. It is not uncommon for a clot to form in a branch that comes off at a diseased spot, while the main artery continues pervious. The orifice of the branch is narrowed so that enough blood does not enter to maintain the circulation in

the part beyond. The wall of the artery at the seat of the thrombosis may present any of the local causal conditions mentioned elsewhere, or, rarely, it may be healthy. After a time thickening from secondary arteritis occurs. Within the vessel is a coagulum which usually fills its interior, and is adherent to the wall. It sometimes does not fill the vessel, either because originally incomplete, or because the clot has shrunk. A recent quickly formed coagulum is red, but after a time it becomes pale and yellow. A slowly formed coagulum is pale, and may be laminated. A secondary clot usually forms far into the contracted distal branches, and on the proximal side as far as the nearest large branch. The clot after a time may undergo calcification, or, with the artery, contracts and shrinks to a cord. The brain-tissue, to which the artery was distributed, is at first anæmic, but quickly becomes the seat of congestive stasis and undergoes softening—red, yellow, or white, according to the amount of vascular distension and blood-pigment remaining in it. If the collateral circulation is free, it may remain unsoftened, and scattered areas may escape from this cause, in the region supplied.

SYMPTOMS.—Where chronic arterial disease is the cause of thrombosis, it may be preceded by the symptoms of local cerebral anæmia (see BRAIN, Anæmia of). The thrombosis itself leads to the symptoms of loss of function in the part to which the artery passed. The onset of these symptoms is slow or sudden, according to the rapidity with which the coagulum forms; and their degree depends on the size of the vessel occluded, its position, and its relation to other vessels which may supply blood to the area involved. Thrombosis of a small vessel in the cerebral substance usually leads to transient brain-disturbance, headache, vertigo, tingling, and temporary weakness in the limbs, soon passing away if a collateral circulation is established; much more slowly, if softening ensues, by compensatory action elsewhere. The occlusion of a large vessel causes commonly very marked symptoms. Complete hemiplegia is frequent, and its onset may be attended by loss of consciousness. See BRAIN, Softening of.

DIAGNOSIS.—The diagnosis rests on a combination of the symptoms of local cerebral disease with the causal condition—vascular mischief (indicated by probable age, degeneration elsewhere, or syphilis), and with conditions leading to feebleness of the blood-current, and increased coagulability of the blood. The diagnosis is rendered more probable by the symptoms, if slight, being transient, if severe being of gradual onset, and, whether slight or severe, being preceded by the premonitory indications of local cerebral anæmia (headache, and local symptoms in the limbs, &c.).

The **PROGNOSIS** and **TREATMENT** of thrombosis in arteries are considered under the resulting condition. See BRAIN, Softening of.

(b) *In Cerebral Veins and Sinuses.*

ÆTIOLOGY.—Thrombosis in sinuses may be primary, and due to changes in the constitution or the circulation of the blood; or secondary, and due to local causes inducing coagulation directly at the spot affected. Secondary thrombosis is the more common. The same conditions of retarded circulation and altered blood-state which permit coagulation in arteries favour it also in veins, and it is often seen in such conditions as phthisis and cancer, and

especially in marasmic states in children. Hence the primary form is most common in children, but the secondary form occurs with nearly equal frequency through life. Local retardation of the circulation from narrowing of the sinus, or compression of the jugular vein, occasionally assists. The blood-current in the front part of the longitudinal sinus is one of extreme sluggishness, on account of the physical conditions of the circulation. Local change causing coagulation is usually the extension to the sinus of adjacent inflammation, or of a clot produced in a tributary vein by such inflammation. Caries of the bones of the skull, especially of the temporal bone, and meningitis are common causes of thrombosis in adjacent sinuses. Lastly, injuries of the skull involving the sinuses sometimes cause coagulation in them.

ANATOMICAL CHARACTERS.—Of primary thrombosis the superior longitudinal sinus is the most common seat, and thence the clot spreads into the veins on either side, and occasionally also into the lateral sinuses. When secondary, the thrombosis occurs in the sinus nearest to the local disease; in that of the petrous bone, the lateral sinus is usually involved. The sinus is distended by firm clot, commonly (not invariably) adherent, sometimes in concentric layers. The walls of the vessel are healthy when the thrombosis is primary or is secondary by extension of clot, but thickened and brittle when invaded directly by adjacent inflammation. After a time the clot may soften and break down—more quickly in cases in which the adjacent inflammation is of a septic character.

The consequence of venous thrombosis is local arrest of the blood-current, the tributary veins and capillaries becoming enormously distended with blood. Sometimes the smaller vessels may give way, causing numerous minute extravasations; the cerebral substance is then crammed with small hæmorrhagic foci, which often coalesce. The condition is especially seen in the convolutions. Blood is also effused into the meshes of the pia mater, and into the subarachnoid space. Into the looser tissues and into the ventricles serum may escape. Thrombosis of the veins of Galen is one cause of ventricular effusion. Ultimately the brain-tissue undergoes softening, first red, and then yellow or white, as the blood-pigment is slowly removed. Occasionally, in septic cases, the softening of the clot leads to pyæmia.

SYMPTOMS.—The cerebral symptoms produced vary in their character. Sometimes they are at first those of excitement and irritation, namely, intense headache and muscular spasm, manifested by contractions in the limbs, or by convulsion, often beginning locally, according to the position of the secondary congestion. These symptoms, after one or several days, are succeeded by those of depression, with coma, and dilatation of pupils. In other cases the coma may come on suddenly, and the stage of excitement, above described, may be absent or little marked. Now and then hemiplegia comes on slowly, without initial loss of consciousness, and develops to a complete degree in the course of a few hours or a few days. Convulsions, when they occur, often continue till death. When the superior longitudinal sinus is plugged, epistaxis, œdema of the forehead, and exophthalmos have sometimes been observed. When the lateral sinus is obstructed, there may be painful œdema behind the

ear, and the jugular vein on that side has been noticed to be less full than on the other.

DIAGNOSIS.—The diagnosis rests on the occurrence of severe cerebral symptoms in association with a causal condition, constitutional or local. Of the latter, ear-disease and injury are the most important.

PROGNOSIS.—This is always serious, death being, in most cases, speedy.

TREATMENT.—The indications for treatment are for the most part causal. In primary thrombosis stimulants and nutritious diet are necessary, and tonics if they can be taken. The patient should lie with the head and shoulders a little elevated, and care be taken that the neck is not bent, and that there is no constriction of it by tight clothes. In secondary thrombosis, occurring in robust individuals, leeching or cupping may be employed; purgatives should be given; and in the less severe cases a blister may be applied near the probable seat of thrombosis, especially behind the ear, and the utmost care taken to afford free exit for pent-up inflammatory products. Pain and convulsion are relieved most effectually by ice to the head.

W. R. GOWERS.

BRAIN-FEVER.—A name popularly applied to any kind of febrile state in which symptoms of cerebral excitement are prominent; as well as to cases of inflammatory disease of the brain or its membranes.

BRASS-POISONING.—**DESCRIPTION.**—Brass is an alloy of copper and zinc in somewhat variable proportions, generally containing about 70 per cent. of the former metal. Its preparation may be roughly divided into two processes, casting and manufacturing; and the employment of the workman on the first or on the second of these mainly determines the acuteness or chronicity respectively of the ill effects from which he is liable to suffer. In each case, as a rule, the poison is acquired by inhalation—in the acute variety, commonly known as *brass-founder's ague*, by inhaling the fumes given off in the act of casting; and in the chronic form, by the inhalation of the minute particles of triturated brass that permeate the atmosphere of the shops wherein the manipulative procedures are carried out.

Which constituent of the fumes is responsible for the production of brass-founder's ague is a question on which opinions differ, some authorities blaming the copper, and others the zinc; but there is little doubt that in the chronic affection, at least, the principal offender is the copper.

I. Brassfounder's Ague.—This is an acute paroxysmal complaint which attacks the casters or founders, but is not altogether unknown in other departments of the trade. These attacks, though of frequent occurrence, show no tendency to regular periodicity. They generally develop on the first day after a holiday, but they may also be influenced by the direction of the wind or the foggy weather, either of which conditions may retard the escape of the poisonous fumes from the shop.

SYMPTOMS.—Several hours after commencing work, usually in the afternoon of a casting day, the first symptoms begin to make their appearance. These consist of malaise, a sense of fatigue and oppression, and frequently constriction of the chest, accompanied perhaps by nausea and muscular pains. This forms the prelude to a definite cold stage in

which the patient sits shivering and trembling, his teeth chattering, with pains in his head, back, and limbs. With great difficulty he makes his way home to bed, where for hours these symptoms persist, and he is unable to obtain warmth or sleep. Vomiting may or may not occur; but eventually the shivering ceases, the skin becomes warm, and a stage of profuse sweating ensues, in the course of which the patient falls into a slumber from which he awakes in the morning with a feeling of sickness and prostration not severe enough, however, to prevent his returning to work.

II. Chronic Brass-poisoning.—Some persons may be employed for months or years, filing, polishing, or otherwise manipulating brass, even under adverse conditions, without suffering any ill effects; others become affected almost from the commencement of their occupation; while others, again, exhibit symptoms only after many years.

SYMPTOMS.—In most instances, especially in young persons, the complaint is ushered in by *anæmia*. The usual accompaniments of *anæmia*—palpitation and dyspnoea on exertion, dyspeptic symptoms, and *anorexia*—are frequently present in some degree, although not generally very acute. Tachycardia is of fairly common occurrence, while nausea, vomiting, thirst, colic, and constipation are not infrequent (*see COLIC*). Slight indefinite pains of a neuralgic character may be complained of, with headache, accompanied by a sense of weakness, malaise, and nervousness, greater than is generally to be found in ordinary cases of *anæmia*.

A green line upon the teeth may usually be observed before the first symptoms of poisoning appear. This is not diagnostic of brass- or copper-poisoning. It is merely an indication that the individual is in the habit of coming into intimate contact with the metal, and that the process of absorption has commenced. The green line is very distinct. Instead of being situated on the gums like the line in plumbism, it forms a band of varying depth in front of the bases of the teeth just where they emerge from the gums, and is most marked on the teeth of the upper jaw. The colour is usually a brilliant green, but may vary in intensity from a light green to a dark olive.

As the disease advances, the subcutaneous fat gradually diminishes, the muscles atrophy, and a condition of progressive emaciation supervenes, which, in course of time, becomes one of the most marked features of the complaint. There is great loss of strength; and muscular tremors, mainly confined to the hands and the tongue, are of very frequent occurrence. The knee-jerks are normal or slightly exaggerated, except when an actual peripheral neuritis exists. Headache is almost always complained of, as well as pains, very variable in character and situation, affecting the abdomen, legs, back, chest, or other parts. The direct contact of brass-dust with the mucous surfaces produces symptoms of laryngeal or pharyngeal catarrh, with aphonia, sensations of dryness, discomfort, or constriction in the throat, and a metallic taste in the mouth. There may be a dry tickling cough, sometimes accompanied by the expectoration of thick tenacious sputum, and occasionally by hæmoptysis. This chronic catarrh of the bronchial tubes may ultimately end in fibrosis of the surrounding lung-tissue. As a rule the dyspepsia with loss of appetite and gastralgia persists, while sickness and even vomiting are occasionally met with. The tongue is furred,

moist, and tremulous; the bowels are irregular; and the breath emits a disagreeable metallic odour. A feeling of oppression or nervousness, sometimes of a very intense character, is common among such patients, who complain also of repeated attacks of faintness or 'sinking' sensations. Profuse sweatings and sensations of localised coldness, probably of vaso-motor origin, are a prevalent source of annoyance. The sweat is observed to be of a greenish hue by its staining the undershirt green, and the same colour, undoubtedly due to the presence of salts of copper, may be noticed in the grey hair of old workmen, and in the palms of the hands, as well as in the complexion, which is of a peculiarly sallow type, quite different from the wax-like pallor that characterises the premonitory *anæmia*. On various parts of the body may be found itching skin-eruptions of an eczematous or acneiform character, due either to the contact of the skin with brass-dust and sand, or to the salt or salts of copper dissolved and excreted in the sweat. Itching, however, may be complained of without any visible eruption. It is generally believed that brass-workers are peculiarly liable to contract phthisis, and this is probably true; but there is no doubt as to the prevalence of pulmonary fibrosis among this class of operatives. An ataxic form of multiple neuritis due to the action of brass has also been described.

TREATMENT.—**I. Preventive.**—The first consideration, both in the acute and in the chronic variety, is to deal with the cause, and to this end it is necessary that the ventilation of the workshops should be thoroughly efficient. Special Rules, issued by the Chief Inspector of Factories, insist on the provision of suitable exits for the fumes and dust in the mixing and casting shops, on the promotion of cleanliness among the workers, and on the prohibition of eating during the process of casting. Similar regulations are equally necessary in the other departments of brass-working where the atmosphere is laden with finely divided particles of metallic dust. Food should on no account be taken in the workshops, neither should it be eaten with unwashed hands.

2. Remedial.—Milk as a beverage has long been recognised as a corrective. It should be drunk freely both in the chronic form of poisoning and in the 'Ague.' Lobelia and tobacco are reputed to be of value in the treatment of the paroxysms of brass-founder's ague. Abstinence from smoking is recommended until the attack comes on, 'then let him smoke *ad nauseam* the strongest tobacco he can procure.' Other emetics are likewise useful.

For the treatment of chronic brass-poisoning iodide of potassium has been much employed; but phosphorus in doses of $\frac{1}{30}$ grain, or diluted phosphoric acid in fifteen-minim doses, given three times a day, seems to act almost as a specific.

Considering the difference in susceptibility of different individuals to brass-poisoning, it is well to advise any one who shows symptoms either to change his occupation entirely, or at all events to endeavour to get transferred to some other department of the works.

WILLIAM MURRAY.

BREAK-BONE FEVER.—A synonym for dengue. *See DENGUE*.

BREAST, Diseases of.—**SYNON.** : Fr. *Maladies de la Mamelle*; Ger. *Brustdrüsenkrankheiten*.

This subject will be treated under the following divisions:—

I. *Abnormalities and disorders of early life.* II. *Disorders of adult life.* III. *Diseases affecting the rudimentary organ in the male.* IV. *Diseases of the nipple.*

I. **Abnormalities and disorders of early life.**—Although in the human female the breasts are two in number, instances are not wanting of multiplicity. The additional breasts are usually found in the axilla or on either side of the median line. In exceptional and rare instances breasts have been found on the arm, buttock, or outer side of the thigh. Care must be taken not to confound them with lipomata. While supernumerary breasts may swell and contain milk at times of lactation, far more often they are functionless and little more than nipples, or pigmented wart-like spots, with no true gland-substance beneath them. The latter are common, especially in the male, and often pass for moles. Supernumerary breasts in the axilla are generally connected by a string of gland-tissue with the main breasts. When they enlarge at lactation they give rise to alarm: they are sometimes the seat of cancerous and other growths. Occasionally the breasts are absent (*amazia*), and this abnormality may be associated with absence of the pectoral muscle, or part of the chest-wall. On the other hand, the breasts may pass the boundary of physiological increase, and even attain many pounds in weight, so as to form a distressing malady. Enormous increase in the size of the mammae has also been observed in elephantiasis. Hypertrophy of the breasts has been noted in the male (*gynecomazia*). Some of the cases have been associated with malformation of the testes. Enlargement has also been noted in association with pulmonary tuberculosis, and cases are recorded of men who have undertaken the duties of the opposite sex and helped to suckle their offspring.

In the *rudimentary period* of the gland the tissues composing it are rarely liable to morbid derangement. Soon after birth, especially in male infants, the rudimentary nipple and the skin of the region within the zone of the areola become slightly elevated and of a pink hue. In some infants a secretion, slightly milk-like, oozes from the ducts. In this state, rubbing may lead to inflammation and suppuration. When this happens the abscess must be incised and treated on ordinary principles.

II. **Disorders of adult life.**—*About adult age, in both sexes*, when the generative organs approach maturity the breast-gland enlarges, and may be painful, thus causing anxiety to the individual. In the female, the breasts generally develop simultaneously, but exception to this rule need cause no alarm.

As the development of the breast reaches maturity, the gland becomes closely associated with the pelvic generative organs, being tender and enlarged just before each menstrual period.

Neuralgia and disorders of pregnancy.—After conception the breasts begin to enlarge, and minute pisiform indurations may be felt at their borders and on their surface. Very rarely this increase in bulk is attended with considerable reflex pain referred to the back, neck, inside of arms, shoulders, and side of thorax—over, in fact, the area of distribution of the branches of those intercostal nerves which send filaments to the breasts. This state is most likely to occur in a first concep-

tion, and in women of excitable, nervous temperament. Attention to ordinary hygienic measures affords relief to the pain.

Genuine neuralgia of the mammae may be experienced, a severe and distressing complaint, and one difficult to cure. It is often found in women who undergo excessive sexual stimulation without the natural sequence of pregnancy. Such cases are sometimes dependent upon small fibrous tumours, inflammatory areas of thickened tissue, or little cysts. The removal of these cures the pain. In other cases, there is some definite mischief in the pelvic organs of generation to be detected and remedied. In other cases the patient is anæmic or depressed. In the treatment of such cases, iron and blisters, with nourishing diet, are indicated. Galvanism, judiciously employed, is a most valuable remedy in mammary neuralgia.

In large, lax, pendulous breasts, the separate lobes of which each is composed may excite apprehension of the existence of a tumour. But new-growths are so rarely developed during pregnancy, that the greatest caution must be exercised in the diagnosis of their nature.

Very rarely, no change whatever in the breasts accompanies pregnancy (*agalactia*), under which circumstances there is an absence of the secretion of milk after parturition.

Inflammation of the Breast.—Abscess.—**SYNON.:** Mastitis.

ÆTIOLOGY.—Before lactation acute inflammation of the breasts is uncommon: after lactation, it is frequent. The slightest unusual fullness or ‘knottiness’ discovered after the infant has been sucking, and when the ducts and their terminal secreting vesicles should be empty, requires immediate attention. Congestion of a lobule or lobe with milk produces the nodule, and the cause of the impediment to its escape should be sought for. The state of the nipple is generally the origin of the difficulty. In many women the nipple is very small and undeveloped, perhaps only on one side, so that the infant, especially if not very strong, has great difficulty in obtaining sufficient milk, and its efforts cause pain in the part. This circumstance induces the mother to prefer suckling with that breast the nipple of which is perfect, and the infant soon appreciates the advantage of that side. Consequently the gland-tissue of the breast with the imperfect nipple becomes congested. Every time the infant sucks it becomes worse, more and more pain and irritation are excited, the orifices of one or more of the ducts in the nipple become blocked, and perhaps the infant refuses to suck the breast. In the meantime the gland becomes more and more distended, and the nipple deeply buried, until at last suckling is impracticable. Probably none of these increasing troubles have been stated to the attendant surgeon; and, when he is consulted, he finds the breast to be in the state presently to be described. Either the orifice of a duct is obstructed by epithelium, or a superficial ulceration exists around it. The entrance of pyogenic bacteria through such a lesion near or upon the nipple is the immediate cause of the mammary abscess.

Inflammation of the breast is most frequent in primiparæ, and within the first month after parturition.

Inflammation does not often occur at the time for weaning the infant, nor in the passive state of the breast. Small chronic abscesses with thickened walls

are, however, occasionally found in middle life, and the accompanying enlargement of the axillary glands and slight retraction of the nipple may lead to such cases being mistaken for cancer.

SYMPTOMS.—First, hardness is felt, ‘a knot,’ in some part of the breast; this enlarges, and may attain to considerable dimensions before causing pain or even uneasiness. Next, pain is felt during suckling; this increases each time the infant sucks. The integuments then become pink, and afterwards red, tense, and shining; more or less of the breast feeling very inelastic, firm, prominent, and heavy. Pain is now often very severe, and great constitutional disturbance is excited. In the centre of the redness the skin becomes of a purplish tint; around this it is oedematous; and with the finger, at the centre of the purple zone, a slight depression and softening spot can be detected. An abscess now exists, and fluctuation is more or less marked in proportion to the quantity of pus. At the purple centre the cuticle has probably by this time separated from the cutis; and a vesicle containing serum, either yellow, or slightly tinged with blood, indicates that ulceration of the cutis is proceeding, and that the pus will soon escape. The above is a brief description of the objective signs noticeable in untreated cases of local inflammation advancing to and terminating in supuration and abscess. It is not possible to state the exact period of time required for the accomplishment of these definite changes, as this varies with many local and constitutional circumstances.

VARIETIES.—When supuration has taken place, the exact site of the abscess should be carefully ascertained. It may be situated over the body of the gland, within it, or beneath it.

When *overlying* or superficial to the body of the breast, the course of the disease is rapid. In these cases the constitutional disturbance is usually trifling.

Both the local and constitutional symptoms are much more severe when inflammation affects the *body* of the gland, and pus collects between its lobes. The progress of the disease is tedious; the pointing of the pus is slow, and the exact spot at which it may reach the surface is for a long time doubtful. In the majority of cases it makes its way between the ducts, and reaches the surface near the areola or within its area; usually to the sternal side of the nipple, where the gland-tissue is thinnest.

When the abscess forms *beneath* the breast the local appearances are quite characteristic. The gland itself seems little involved, but it is pushed prominently forwards and seems to rest upon a cushion of fluid. To the touch the elasticity of the swelling is very striking, and, without producing additional pain, a slight bulging of the walls of the abscess may sometimes be produced at the periphery of the gland when, with the palm of the hand and outspread fingers, compression is made from the front backwards against the thorax. The patient should be recumbent. The pus in these cases often points somewhere around the borders of the body of the gland.

TREATMENT.—Great attention should be given to the nipples of primipare. Prophylactic measures should be instituted when there is reason to fear that a defective development of the nipple will interfere with the free flow of the milk, and it may occasionally be necessary to forbid suckling on account of the mechanical impediments and the pain caused by the attempt. If the nipple be imperfect, precautions should be taken to prevent the gland itself from

becoming congested, and if the infant cannot draw the milk sufficiently, some mechanical means should be employed to effect this object. The nipple itself should be examined. If its end be more than usually coated with secretion, or the openings of the ducts seem to be obstructed with an excess of epithelium, attempts may be made to remove it. If abrasions, ulcers, ‘cracks,’ or ‘chaps’ are visible between the rugæ, a soothing application, or a weak solution of carbolic acid, should be used. Ablution with warm boiled water, or the contact of a little moist boric lint, covered with gutta-percha tissue, is often sufficient; or, if there be much secretion from the glands on the nipple, after cleansing its surface, some dry powder is beneficial, such as carbonate of magnesium, oxide of zinc, or starch powder.

When actual congestion of the gland-tissue exists, mechanical means should be used to reduce it. Supporting the gland with strips of plaster and a bandage is sometimes very useful. When inflammation is excited the infant must be weaned, and the milk drawn off by the breast-pump from the opposite mamma. The patient must be purged and placed on a dry diet, while iodides and belladonna must be administered internally. The application of glycerin of belladonna and a suitable support of the breast are also of great service, and gentle massage by the hand of the nurse, directed from the nipple towards the periphery of the gland, is often beneficial.

As soon as the presence of pus is ascertained, it should be evacuated by means of a short incision radiating from the nipple. This must be kept open by as large a drainage-tube as the incision will admit, and the strictest antiseptic treatment should be employed. The omission of the latter is very likely to lead to burrowing of pus and the development of troublesome sinuses.

The constitutional powers of the patient must be well supported, and the general health maintained by every possible means.

The *sequelæ* of supuration in an organ composed of so much connective tissue, and endowed with its peculiar function, sometimes cause great trouble; they are, however, much less frequently met with now than before the introduction of the antiseptic treatment. They are protracted induration, sinuses, and fistulæ, through which last the milk persistently escapes. Induration of the whole or part of the breast subsides when lactation ceases, and the organ in due time resumes its healthy state. Sinuses and fistulæ may require incisions, but the ordinary methods for their cure should be adopted before having recourse to a treatment often involving much subsequent deformity.

Any considerable fullness of the breast occurring when the process of weaning the infant is begun may be relieved by mechanically drawing the milk in just sufficient quantity to diminish the fullness.

In the chronic abscess of middle life an exploratory incision, which often furnishes the only certain guide in diagnosis, constitutes the most satisfactory treatment.

Tubercular Abscess of Breast.—This affection is rare, but very important. The abscesses are generally chronic or ‘cold’ abscess, the skin over them being blue, and the pus they contain thin and watery. The *Bacillus tuberculosis* may be found in the discharge. The abscesses are frequently multiple, and the breast-tissue may be extensively destroyed. The axillary glands are enlarged and caseous. The surgical treatment lies in either

opening and erosion of the cavities and glands, or removal of the entire disease. The climatic and other general treatment for tubercular maladies should be thoroughly carried out.

Galactocoele.—An accumulation of milk, to which this term is given, may result from the bursting or the obstruction of a lactiferous tube, and form a tumour in the connective tissue of the breast. The swelling always appears first during lactation. It may vary in size from time to time; sometimes enlarging rapidly as suckling goes on. Two varieties are met with. In the one form there is a single swelling near the nipple, quite superficial, and quickly recognisable by its objective signs. In the other there may be several swellings distinguishable in the substance of the gland, as well as on its surface, all of comparatively small size, very firm and globular. In the same gland they vary very much in size and in the degree of resistance they offer to manipulation. The discrimination between these tumours and others in the breast is easy, if it can be ascertained with exactness that the swelling appeared somewhat suddenly during suckling, and that its size varied with that function.

In cases of long standing, the contents of the cyst become solid in proportion to the quantity of the fluid constituents of the milk absorbed, and the cyst-wall itself is very often rigid and even may become calcareous. Such cases have been confounded with cancer.

TREATMENT.—The treatment of the chronic cases consists in dissecting out the cyst.

Chronic lobular mastitis.—When the tissues of a mature but perfectly passive gland are examined with the microscope, the caecal terminations of the ducts are found to be small, and little else than fibrous tissue is seen. Here and there, at their blind ends, spaces may be detected containing minute aggregations of epithelium. But when, under some sympathetic excitement, with derangement of the functions of the pelvic generative organs, the secreting cells of the gland become active and are distended with epithelium, they induce more or less enlargement of the breast. This condition of things is called *chronic lobular mastitis*, and should perhaps more properly be included among the inflammatory diseases of the organ. But of such affections there is this important fact to be noticed. The whole breast need not be necessarily involved. On the contrary, one lobe only may be excited, and when this occurs the existence of a tumour is declared. When, after excision, such enlarged lobes are carefully examined with the microscope, normal gland-tissue is seen, the caecal ends of the ducts are readily recognised, and their immediate association with the excretory channels may be observed. The former are gorged with epithelium; and true gland-tissue, less its peculiar secretion, has been developed.

SYMPTOMS.—Associated with this state of the issues of the breast, the patient complains of pain, both local and spreading over a very wide area. To express as briefly as possible the superficial regions affected and over which pain is felt, the reader must be reminded of the distribution of the descending cutaneous branches of the cervical plexus, and of the lateral and anterior cutaneous branches of the second, third, fourth, and fifth intercostal nerves. From these, special filaments are distributed to the breast; and to the site of exit of one or more of them at the intercostal

foramina, the source of the pain is referred by the patient. The skin of the neck, shoulder, side of thorax, and inside of arm receives filaments from the same source. Hence an explanation of the widely diffused pain.

It is of the first importance to discriminate between this state of the gland-tissue and substantial new-growths, especially because the latter cannot be removed by natural processes, while the former most probably will be. The objective signs are the following: to the touch the implicated gland-tissue is nodular, and mingled with the body of the organ. If the whole breast be large and relaxed, the tips of the fingers may be insinuated between the borders of the indurated lobe and the lobes not affected. If the entire body of the gland be morbidly firm, it feels like a disc-shaped mass lying on the thorax, under the borders of which the fingers can be pressed. Occasionally, at one or more spots along the periphery of the gland, irregular nodules are perceptible, projecting into the connective tissue around them. When one lobe is affected, the shape of the induration may correspond with that of a lobe—i.e. broad at the periphery and gradually narrowing towards the areola. Pain is of assistance in forming a diagnosis in these cases; but the source and course of the pain must be carefully traced. Generally manipulation of the induration produces increased pain; occasionally a mere touch is intolerable. Light pressure should be made over the points of exit of both the anterior and lateral branches of the intercostal nerves, when the pain excited thereby will correspond with the nerve-filaments of the affected lobe. Pressure along the upper dorsal spinous processes usually excites pain also. The morbid affections above described occur in single women, married but sterile females, and young widows, at any age between twenty and forty years. More or less disturbance of the catamenia co-exists, either in relation to the frequency or quantity of the discharge. The patient complains of languor and inability for bodily or mental exertion; and she is desponding and often alarmed at the consequences suggested by sympathising friends. She is irritable, emotional, restless at night, and loses appetite for food, as well as all desire for social enjoyments.

DIAGNOSIS.—An exact diagnosis may be made if the manipulator examines the organ methodically. He should, first, gently grasp the induration between the thumb and fingers, when it will be distinctly appreciable. Afterwards, placing the palmar surface of the fingers over the surface of the breast and gently pressing backwards against the thorax, the induration will not be detected. Should there still remain any doubt on the subject, the patient should be placed on her opposite side on a sofa, and in this posture, if there exists any substantial mass, the integument is usually elevated by it.

As a general rule it may be stated that if a distinct lump is felt by the fingers of the surgeon when laid flat upon the mamma this is more than mere chronic mastitis. Such a lump or prominence will be probably a cyst or an early cancerous nodule.

TREATMENT.—The treatment of these cases consists in careful attention to the general health. Local soothing applications are usually futile, and, except in cases of extreme pain, are unadvisable; but strapping with belladonna plaster may give

their remarkable mobility, slight lobulation of surface, and firmness, coupled with the youth of the patient, are sufficient indications of the harmlessness of their nature. Usually they occur singly and in one breast: they may be multiple and in both breasts. The only means by which they can be removed is excision. In young persons, however large the tumour, its removal should be always attempted without cutting away any portion of the normal breast. In most cases this can be done, especially if the patient be youthful and the growth of medium size, even should it be developed in the body of the gland and extend through it to the pectoral muscle. After thirty-five years of age it is expedient to remove the breast as well; for these tumours are then sometimes associated with cancerous growth. A section shows a solid, uniform surface, divided into lobes and lobules by fibrous septa, sometimes slightly broken up by fissures or clefts in which there appears a little clear tenacious fluid. The section is often very succulent, at other times only moist; its hue may be greyish, yellow, or almost white. Its vascularity is scarcely perceptible. The prognosis in these cases is invariably favourable.

Carcinoma and Sarcoma.—The usual period for the appearance of cancer in the female breast is between the ages of 35 and 55, but instances occur in younger and also in older women. Blows, inflammation, old abscesses, and retracted nipples, are potent predisposing causes. Various kinds of carcinomata are described, but the spheroidal-celled growth is the common one. This may undergo colloid or fatty change, and may also be mixed with blood-cysts. Thus the terms 'colloid cancer' and 'hæmorrhagic cancer' have originated. A peculiar kind of cancer has also been described which appears to originate in papillary growths in the ducts. The epithelium is columnar in shape, and the tumour is soft and honeycombed with cysts of various sizes, containing coloured fluid (*duct-cancer*). The commencement of cancer of the breast is a peculiar affection of the skin round the nipple known as 'Paget's disease' or 'malignant dermatitis of the nipple' must not be overlooked. See NIPPLE, Diseases of.

SYMPTOMS.—The clinical history of cancer of the breast is of surpassing importance; in its early beginning this serious malady is too often ignored or concealed, and the extreme necessity of detecting and treating it in its earliest stage is still often overlooked. The affection may be noted accidentally as a small lump or tumour, or a mere hardening not larger than a pea or bean in the breast-substance. Pain is usually absent.

Rarely, the whole organ is simultaneously infiltrated, but most frequently one lobe only is affected. The infiltration is often central in the body of the gland, especially when the organ is atrophied, and the reverse occurs when the extreme edge of a lobe is affected at its periphery. The infiltration may steadily increase until the whole breast forms a rigid, solid mass; but most frequently the larger part of the organ remains unaffected. The disease gives rise by its contraction to much deformity of the region, to dimpling, corrugation, and irregularities of the otherwise rotund integumental surface. The nipple, just in proportion to the effect of the growth upon the ducts, becomes retracted or drawn towards the tumour. Sooner or later the integument over the growth becomes adherent to it,

infiltrated, and red, and advances towards ulceration. An ulcer now forms, the edges of which are everted, ragged, and attached to the growth beneath. A hole extending into the tumour becomes deeper and deeper; ichorous discharges, more or less profuse, continue without much pain; and the patient becomes at last worn out by fever and septic infection, or succumbs to the ravages of a cancerous growth in a vital organ.

The rate of progress of this disease varies remarkably. In some individuals the disease does not destroy life. In general terms its progress may be said to vary directly with the age and vigour of the individual. In young women, especially when pregnant or during lactation, cancer advances with such rapidity that no operative measures seem of much avail. In old and withered women, on the other hand, the disease may remain quiescent for years, or may actually retrogress (*atrophic cancer*). It is upon these natural vagaries of the disease that quacks and impostors flourish; for any remedy taken in a case of atrophic or withering cancer is sure to receive the credit for what is really the natural course of the malady. The diagnosis of cancer of the breast is usually obvious; on the other hand it may be most difficult even for experts.

TREATMENT.—Nothing but operation is of any value. All vaunted remedies, such as electricity and patent preparations, do not bear the investigation of scientific and unprejudiced observers. Removal of the ovaries and the administration of thyroid extract have seemed to check the growth, and especially the pain of inoperable recurrent cancer. For other cases this treatment is more severe and not so certainly beneficial as the ordinary operation. The results of the modern operation for cancer enable the surgeon to give a far more hopeful view of the future of the patients than was formerly possible. Cases of prolongation of life for periods of from three to six years are fairly common, and even in cases where the disease returns the avoidance of a huge fungating mass upon the chest-wall is a boon which can hardly be over-estimated. The two essentials to success are early and free removal. In cases which have been neglected and concealed, and in which glands are extensively involved, respite is generally all that can be hoped for.

The modern operation consists in not only removing the breast and all its outlying lobules, but also the fascia over the pectoral muscle, and the muscle itself, should the growth be at all extensive. The glands and connective tissue in the axilla are in *all* cases to be carefully dissected out. Should the pectoral muscle not be cut away, it may be divided, and this will give very free access to the axilla and permit the removal of all axillary tissue. The cut muscle can be reunited with catgut or kangaroo-tendon. Great care must also be exercised in the removal of all integument involved, and, should the parts not come together, the method of Thiersch, or some other form of skin-grafting, must be employed (see SKIN-GRAFTING). If strict asepsis be ensured these extreme operations are not dangerous. They should, however, only be performed by experienced operators who will not hesitate to dissect out the fat and axillary glands from the nerve-trunks and great vessels. Imperfect removal of the disease is followed by rapid recurrence, and the operation falls into

undeserved disrepute owing to the imperfect and faulty action of its performer.

Sarcoma.—Under this heading, as well as that formerly employed of 'fibroplastic tumour,' we include a group of new-growths composed of sarcoma-tissue, with round, spindle, or mixed cells, in which are interspersed somewhat modified acini and ducts of the gland.

The microscopical characters of the tumours are very varied and peculiar, and in some instances closely resemble the remarkable tumours (myeloma) present in connection with the medullary tissue of bone.

Sarcomatous tumours of the breast are found in young or middle-aged women. They have been seen in girls of sixteen and twenty. The physical signs are those of a round or oval tumour, which increases progressively or grows by fits and starts. The veins over the tumour are enlarged, and the integument is dusky and sometimes the seat of ecchymoses. The rate of growth and the associated symptoms correspond with the degree of malignancy of the tumour. Some of the worst kinds are soft and jelly-like, and are often confounded with cysts or abscess. Very free removal of the whole breast and of the axillary lymphatics should be adopted in these cases. They are apt to recur *in loco* after removal, and when of rapid growth in pregnant and lactating women the prognosis is usually of a gloomy nature. In the more slowly growing and encapsuled forms, the prospect for the patient is good.

III. Diseases of the Male Breast.—The male has sometimes a well-developed mammary gland, and the part is subject to the same diseases as in the female. Thus cancerous tumours, fibroadenomata, and cysts may all occur in the male breast. Their symptoms and clinical history are identical with what is found in the female. All these affections are exceedingly rare. The simple enlargement of the organ is harmless, and should not be interfered with. At the age of puberty the mammary region often becomes painful, owing in part to the pressure of the dress upon the mamilla and the rudimentary organ. Inflammation followed by suppuration has been observed at this time.

IV. Diseases of the Nipple.—A defective formation of the nipple is of grave importance, and when it exists measures should be adopted to assist its elongation. This is to be done by using an exhausting-glass, such as those employed to empty the gland of milk.

Inflammation and its effects produce much suffering, and at the period of suckling frequently excite deep-seated mischief. The small ulcers, called 'cracks' or 'chaps,' which form between the rugæ on the apex and sides of the nipple, may be cured by a dressing of boric lint, or by powdering the part with carbonate of magnesium or oxide of zinc or by bathing with a weak solution of carbolic acid.

Eczema round the nipple is common, and must not be confounded with malignant dermatitis. See NIPPLE, Diseases of.

Pendulous cutaneous growths occur on the nipple and should be excised.

Cystic follicular tumours are sometimes seen within the zone of the areola.

JOHN BIRKETT.

A. MARMADUKE SHEILD.

BREATH, The.—The expired air, or what is familiarly termed *the breath*, is important both from an ætiological and a clinical point of view, and the object of the present article is to present a brief summary of the main facts relating to this subject, from a clinical point of view.

a. The breath has been made use of to distinguish between *real* and *apparent death*. See DEATH, Signs of.

b. The *temperature* of the expired air may be altered. In some conditions, as in the collapse-stage of cholera, it becomes cold, and this may be perceptible to the hand, or it may be visible in consequence of the moisture in the expired air being condensed, even when the surrounding atmosphere is warm. On the other hand, the temperature of the breath may be raised, as in febrile diseases.

c. *Chemical examination* of the breath may prove of service. In certain affections, as during an attack of asthma, or in cases of extensive bronchitis, the amount of carbonic acid in the expired air is more or less increased; in others, such as in the collapse-stage of cholera, this constituent may be very deficient. Again, chemical examination of the breath may reveal the presence of a poison in the system, introduced from without, for example, hydrocyanic acid. It has also been employed to show the existence of deleterious products generated within the body, especially in cases of renal disease.

d. *Microscopical examination* of the expired air has been attempted, but at present no results of practical value have been obtained.

e. The *odour* of the breath is the most important character demanding attention from a practical point of view. At the outset it must be observed that in some individuals the breath seems to have naturally a disagreeable odour, which cannot be referred to any particular cause. This is sometimes only temporary, and in females may only be noticed at the menstrual periods. It must be borne in mind that the breath is frequently unpleasant, either temporarily or constantly, owing to indulgence in certain articles of food, or in certain habits, such as smoking or chewing tobacco.

(i.) The odour of the expired air may aid in the recognition of a poison inhaled or swallowed. The smell of prussic acid or laudanum, for instance, may be revealed when either of these is present in the stomach. In cases of acute alcoholic poisoning, the odour of the alcohol or of its products is at once apparent; and in persons who are found in a state of unconsciousness, the smell of the breath is made use of as one of the diagnostic signs of drunkenness, if it be previously ascertained that no alcohol has been administered as a restorative. It is in the chronic forms of alcoholism that the breath gives the most valuable information. In marked cases of chronic alcoholism it has a quite characteristic odour; moreover, this peculiar odour often enables us to detect dram-drinkers, and to explain the symptoms of which they so frequently complain; as well as to detect intemperance in candidates for life-insurance. Again, the breath may reveal the presence of certain metallic poisons in the system, of which mercury is the most important example, but lead may also affect its odour. In cases of diabetes mellitus, and especially diabetic coma, the breath may have a sweetish, fragrant, or ethereal odour, attributed to acetone.

(ii.) The breath has peculiarities of odour in many different diseases. That which is associated

with the febrile condition is well known. In various disorders of the digestive organs the breath is often very offensive, but it is not practicable to refer any particular odour to any particular disease of any of these organs; it may, however, be affirmed that an unpleasant smell is frequently associated with habitual constipation. In cases of stercoraceous vomiting the breath may have a distinctly faecal odour. In this relation it may be mentioned that in some cases of phthisis the writer has noticed a sickly smell of the breath which is quite characteristic, and which seems to depend upon the state of the stomach. In cases of cerebral disease, also, the breath often becomes exceedingly offensive, on account of the condition of the alimentary canal. Local morbid conditions about the mouth, throat, or nasal cavities constitute a most important class of affections which influence the odour of the breath; in many cases it becomes extremely foul, and may be quite peculiar in its characters. Among these conditions should be specially mentioned want of cleanliness of the mouth and teeth; decayed teeth; diseased bone in the mouth or nose; ulceration or gangrene about the mouth, especially cancrum oris, and gangrenous ulceration along the gums; suppuration, ulceration, or gangrene in the throat, either of local origin, or associated with syphilis, scarlatina, diphtheria or other general diseases; ulceration of the nasal mucous membrane, and chronic *oæna*; and malignant disease. The smell of the breath is of value in drawing attention to some of these conditions, for, in the occasional absence of other symptoms, the patient may be quite unconscious that there is anything wrong. Again, certain conditions of the respiratory organs are liable to affect the odour of the expired air, and may render it unbearably foetid. Among these may be mentioned sloughing ulceration about the larynx, pulmonary gangrene in any form, and the decomposition of retained morbid products in dilated bronchial tubes or in certain cavities. Here, again, the smell of the expired air may reveal what otherwise is liable to be overlooked, and especially when the patient coughs, and so expels some of the reserved air. Lastly, the breath may have a peculiar odour in some special diseases, such as pyæmia and its allies; and after the administration of certain drugs, such as paraldehyde.

TREATMENT.—It is only intended here to offer a few remarks as to the treatment of *foulness of breath*. The first great indication is, of course, to seek out the cause of this symptom, and to endeavour to remove it. The habits should be duly regulated; the mouth and teeth properly cleansed; the alimentary canal maintained in good order; and any special affection requiring treatment attended to. When unpleasant breath depends on the stomach, it may often be improved by taking charcoal, at the same time remedies being employed suitable for the particular affection present, and calculated to promote the functions of the alimentary canal, the bowels being also kept freely open. When the bad smell depends on local causes, it may be diminished by the use of antiseptic mouth-washes, gargles, sprays, or nasal douches of Condy's fluid, peroxide of hydrogen, carbolic acid, or creasote. Antiseptic inhalations or injections are indicated in fœtor from the air-tubes. See BRONCHI, Diseases of.

FREDERICK T. ROBERTS.

BRICKLAYERS' CRAMP.—See OCCUPATION-DISEASES.

BRIDES-LES-BAINS, Savoy.—Thermal alkaline saline waters. See MINERAL WATERS.

BRIGHT'S DISEASE.—SYNON.: Fr. *Maladie de Bright*; Ger. *Die Bright'sche Krankheit*.

Bright's Disease may be defined as an acute or chronic inflammatory affection of the cortex of the kidneys, often accompanied by secondary lesions in other organs of the body, especially the heart and blood-vessels.

A great number of morbid processes have been included under the name of Bright's disease by different writers, but at the present day the term is becoming limited to certain acute and chronic inflammatory affections. All forms of nephritis are, however, not to be included under the name of Bright's disease, although in many respects the changes produced in the kidney during acute nephritis are essentially similar to those seen in the early stages of acute Bright's disease. The distinction between nephritis and acute Bright's disease depends essentially on the fact that the lesion in the latter tends to be progressive, and, further, that general effects, such as dropsy, occur which are unknown in cases of simple nephritis. The distinction between a typical case of acute nephritis and a typical case of acute Bright's disease is thus easy, but many anomalous forms are seen where this distinction breaks down. Acute Bright's disease is sometimes seen unaccompanied by dropsy during the acute stage, but where the subsequent history of the case, or maybe the death of the patient, shows clearly that the disease was progressive and accompanied by the other phenomena such as uræmia, secondary inflammations, and cardio-vascular changes characteristic of Bright's disease. The difficulty of differentiating between nephritis on the one hand and Bright's disease on the other is so great that some writers consider Bright's disease and nephritis synonymous terms. One objection to this view is that some forms of Bright's disease are of such a slow and insidious onset that they do not present an inflammatory character at any period of their course. A further difficulty is introduced by the fact that the ætiology of many forms of nephritis and that of Bright's disease are essentially similar. The poison of scarlet fever during the height of the malady may produce a condition of slight nephritis characterised clinically merely by the presence of albuminuria. On the other hand during the subsidence of scarlet fever typical Bright's disease may be produced characterised by copious albuminuria, onset of dropsy, and all the toxic symptoms associated with the disease. Either two forms of nephritis must be recognised, one transitory and partial in its distribution, the other more general and tending to be progressive in its course; or else the term *nephritis* must be limited to the former, and acute Bright's disease to the latter. Some writers have ventured to describe acute Bright's disease under the form of acute nephritis, restricting the term of true Bright's disease to the chronic forms of the disease. Bright's disease is separated with difficulty not only from nephritis but also from certain chronic degenerative processes of the kidney. Many writers have included the waxy kidney under the term of Bright's disease, and Bright did this himself. This confusion arose partly from the fact that in many forms of true Bright's disease amyloid degeneration of portions of the kidney is not uncommon. Further, the amyloid kidney presents great resemblances clinically to

some forms of chronic Bright's disease, and there is a superficial resemblance in their morbid anatomy. Lastly, both the amyloid kidney and Bright's disease may arise as complications of one and the same malady, e.g. phthisis, syphilis. The amyloid kidney, however, has only a superficial resemblance to Bright's disease and should be sharply separated from it, if for no other reason, because frequently its treatment is quite different from that of Bright's disease. In many forms of Bright's disease there is a considerable overgrowth of the fibrous tissue of the kidney, and there are also marked changes in the heart and blood-vessels, and in this way there is some resemblance brought about between some forms of Bright's disease and what is ordinarily spoken of as the granular kidney. The true granular kidney, however, has no real relationship with Bright's disease and is an independent affection.

The term 'Bright's disease' in this article will therefore not include certain forms of nephritis nor the amyloid kidney nor the granular kidney.

Varieties.—Bright's disease, even thus limited, presents many varieties which have been classified differently by different writers. All writers are agreed that an acute form and a chronic form may be recognised. In the acute form all the constituent elements of the kidney suffer, but in different cases to different extents. In some forms of acute Bright's disease the main effects of the morbid process are seen in the glomeruli, in others the most marked results are the degenerative changes occurring in the epithelium of the tubules. In others perhaps the most marked effects of the inflammatory changes are seen in the interstitial tissue. In all forms, however, these three elements of the kidney are always affected. Not only are the different elements of the kidney affected to different extents in different cases of acute Bright's disease, but even in one and the same case the lesion is developed to varying extents in different portions of the organ. It is one of the most characteristic features of Bright's disease that although the morbid change affects both kidneys the severity of the effects produced varies considerably in different parts of the organs.

Chronic Bright's disease presents many varieties both from the point of view of the clinician and that of the morbid anatomist, and it is customary to recognise two fundamental varieties: (1) the so-called large white kidney, and (2) the contracted or atrophic white kidney sometimes called granular or cirrhotic, but the latter terms are better restricted to the description of the true granular kidney, which is not here included under Bright's disease. Not only do these two forms of chronic Bright's disease present marked anatomical differences, but there are also differences in their clinical course, and they may probably be recognised as real varieties. For the purposes of this article, therefore, *three* forms of Bright's disease will be described: acute Bright's disease and two varieties of chronic Bright's disease.

ÆTIOLOGY.—Bright's disease both in its acute and chronic forms is probably dependent in the great majority of instances on the action of some toxic agent present in the blood-stream, on the renal structures, and more especially on the glomeruli and the epithelium of the cortex. The toxic agent producing these ill effects may be one produced in the body by the action of microbic processes, as is the case in all probability in the acute infective diseases. The toxic agent, however, in a very large number of instances is one that is ingested, as in the case of

alcohol or of mineral poisons, such as lead and mercury. It is also possible that toxic substances having a deleterious action on the kidney may be formed in the body as a result of deranged processes of metabolism. Although toxic agents will account for the great majority of cases of acute and chronic Bright's disease, it is held by many clinicians that these maladies may arise in other ways, as for instance as a result of exposure to cold. The rôle played by cold in the causation of morbid processes is not now held to be so direct as it formerly was. In many instances where cold is reputed to act as the direct exciting cause it really only produces its effects indirectly. Thus many diseases imputed to the action of cold are really dependent on microbic infection, which is more able to produce its effects owing to the lowered resistance produced by the cold. In the case of renal diseases instances are undoubtedly seen where the malady has apparently arisen as the result of exposure to cold, and especially to damp cold. Thus the disease is not uncommonly seen in workmen who work in hot shops and pass out into the open air scantily clothed; also in boiler-cleaners, engineers, and others who experience great vicissitudes of temperature, but in many of these cases it is difficult to exclude the action of other and toxic agents, as, for instance, alcohol, lead, &c. It has also been thought that mechanical causes, such as the pressure of the pregnant uterus on the renal veins, might lead to Bright's disease, but here also it is probable that the association, which is a real one, is dependent rather on the action of toxic substances than on the mere mechanical effects of pressure. The action of cold in the production of renal disease has been thought to be probably owing to the intimate relationship existing between the action of the skin and that of the kidneys, and it has frequently been taught that exposure of the skin to cold, causing contraction of the cutaneous vessels, produced reflexly an engorgement of the kidney. There is, however, no experimental evidence justifying this hypothesis, and it has been suggested that if cold is really an important factor in the production of renal disease, the effects produced are not dependent on any congestion of the renal vessels brought about by the nervous system, but rather that the arrest of the cutaneous secretion leads to the retention of toxic material in the blood-stream, causing an injurious action on the renal structures. Although perhaps it cannot be denied that congestion of the kidney may be brought about through the nervous system, yet at the present day the only definite manner in which nephritis can be produced is by the action of toxic agents.

Renal poisons may be divided into three groups. The first consists of bodies like cantharides or like the virus of scarlet fever, which produce almost similar results of which the most marked is glomerulo-nephritis. A second group of renal poisons consists of the metallic salts, the oxides of the heavy metals, and such bodies as chromic acid, all of which produce a coagulation-necrosis of the cells of the convoluted tubules. A third group of renal poisons consists of certain animal and vegetable toxic substances, such as abrine, ricine, serpent-venom, and the blood of certain fish, e.g. the eel. These complex bodies produce a degeneration of the epithelial cells of the kidney, which, however, is not so complete as the coagulation-process produced by the action of the heavy metals. Recently it has been asserted that renal lesions are produced by a number of

bodies belonging to the group of substituted ammonias, and more especially by such bodies as vinylamine, cadaverine, and putrescine. There is thus a wide range of toxic substances capable of producing a great variety of renal lesions.

Bright's disease, especially in its acute form, is most frequently the sequela of an acute specific disease, and more especially of scarlet fever. It is, however, also seen after other acute diseases, such as diphtheria, erysipelas, pneumonia, measles, rheumatism, pyæmia, and typhoid fever. It is interesting, as remarked above, that most of these diseases, cause during their height a febrile albuminuria dependent on the existence of transitory nephritis. Acute Bright's disease, however, arises at a later period of the progress of the malady when convalescence is approaching. Pregnancy may also be recognised as a cause; and Bright's disease sometimes arises in the course of certain chronic diseases, such as heart-disease, gout, and diabetes. Acute Bright's disease is also seen as a complication of burns, and in certain skin-diseases, especially pityriasis and eczema, and it is possible that in this latter instance the lesion is dependent on the formation of toxic bodies in the skin and their subsequent absorption and excretion by the kidneys. Alcohol, cantharides, turpentine, mineral acids, and carbolic acid may all cause acute Bright's disease. Cold is also held by many observers to be a direct and exciting cause of some of the most acute forms of Bright's disease. Toxic agents are therefore most frequently the direct and exciting cause of acute Bright's disease, but there are some predisposing causes, such as occupation and indulgence in alcohol. Engineers, boiler-cleaners, glaziers, potters, painters, and barmen are all especially liable to suffer from this disease. Bright's disease is more common in men than in women in the proportion of three to two, and is most common between the ages of three and forty. In some instances hereditary influences seem to play a part; this is more especially the case in the chronic forms of the disease. Passive congestion of the kidney from heart-disease is also held to be a powerful predisposing cause.

The ætiology of chronic Bright's disease is similar to that of the acute disease. The disease is frequently the result of gout and of the action of such poisons as alcohol and lead; it also supervenes in certain occupations necessitating exposure to cold. Chronic Bright's disease is not produced, however, by the action of the poisons of the specific fevers directly; whenever it results from scarlet fever or other acute specific, it is really the sequel of an attack of acute Bright's disease produced in these diseases. Chronic Bright's disease may therefore arise as a sequel of the acute form, or it may be chronic from the commencement; large numbers of cases of chronic Bright's disease have never been preceded by any attack of the acute disorder. Sometimes chronic Bright's disease supervenes directly as a sequel of acute Bright's disease, in other cases the acute stage of the malady subsides and health is regained, and subsequently, as a result perhaps of exposure to cold or from indiscretions in diet, a further attack of acute or subacute nephritis is induced, and the chronic disease dates from this second or maybe from a third attack.

The relationship of the two forms of chronic Bright's disease, the large white kidney and the contracted white kidney, is somewhat doubtful. It is possible that in some cases the contracted form of

the disease is a terminal stage of the other, the diminution in the size of the organ being dependent largely on atrophy of the renal elements, together with great overgrowth of connective tissue. Frequently, however, the contracted white kidney is seen in the *post-mortem* room without there being any previous history suggesting the occurrence of the large white kidney. Many patients dying with a contracted kidney have never suffered from dropsy, which is such a characteristic feature of the large white kidney. In some instances where the contracted white kidney is well marked, a history of an acute specific fever followed by acute nephritis may be obtained, and it would seem as if in these cases this form of Bright's disease was the terminal lesion and in some way related to the occurrence of acute nephritis, and more especially of scarlatinal and glomerulo-nephritis. It is not uncommon, however, for the contracted white kidney to occur not only without the history of any previous renal illness, but also without there being any history of any acute disease; therefore it would seem that the contracted white kidney may exist as an independent disease, and that all cases of it are not necessarily preceded either by acute nephritis or by the large white kidney.

MORBID ANATOMY.—*Acute Bright's disease.* In the acute stage the kidney is considerably enlarged, weighing seven or more ounces, the capsule strips off readily, and the organ is gorged with blood, dark-red in colour, the congestion being most marked in the stellate veins and in the pyramids. The colour of the section and the colour of the surface depend largely on the duration of the disease; in the early stages they are of a deep-red colour, but after the lapse of a week to ten days the surface is mottled or marbled, owing to the presence of yellowish or greyish-yellow areas, caused by degeneration of the epithelium; if the duration of the disease has been as long as six weeks, both the external surface and the section show the cortex of a more or less uniform, peculiar yellowish-grey colour dotted with spots and streaks of congestion. The pyramids are usually intensely congested, and the glomeruli can sometimes be seen as bright-red points. Microscopically the glomerular chamber often contains blood-corpuscles and exudation, and the vessels of the tuft have frequently undergone hyaline degeneration. The vessels generally are gorged, and this is especially marked in the straight vessels passing up in the medulla. There may be a considerable amount of leucocytic infiltration of the interstitial tissue, and this is often especially well marked in the connective tissue round the glomerulus. The epithelium of the glomerular chamber may undergo considerable proliferation, forming a crescentic mass between the basement membrane and the capillary tuft. The cells of the convoluted tubules are frequently shed, occupying the lumen of the tubules, which are dilated to varying extents. Where the epithelium remains the cells have undergone degeneration, cloudy swelling being marked, and in other instances the cells have lost their striated border and have assumed a glass-like or hyaline appearance. The lumen of the tubules is occupied by blood-corpuscles, disintegrated renal cells and casts, the casts consist in some cases of coagulated blood-plasma, forming hyaline and granular casts, or of aggregations of renal cells, which have undergone degeneration. The changes in the medulla are similar, but not so well marked. Although in

acute Bright's disease the changes in the cortex of the kidney are widespread and cause considerable apparent increase in the amount of cortex, yet these changes are rarely evenly or uniformly distributed, and this statement really applies to all forms of Bright's disease. Areas are seen where the glomerular and tubular changes are far more marked than in adjacent areas, and in some parts the glomeruli and renal tubules may present appearances but little different from those of health.

Chronic Bright's disease.—In the so-called large white kidney, the organ, as the name implies, is enlarged and perhaps double its usual size, but the anatomical changes present are essentially similar to those seen where the organ is but little increased in size. The capsule strips off readily, leaving a smooth, opaque, yellowish-white surface, on which the stellate veins are conspicuous. The colour of the surface may be somewhat mottled; the colour depends largely on the degree of congestion. On section, the cortex is relatively increased and presents the same appearances as those seen on the surface. The medulla is usually deeply congested and stands in marked contrast to the cortex. The pyramids seem unduly separated owing to the increase in the amount of cortical substance. The glomeruli, the tubules, and the interstitial tissue all show changes. In the large white kidney the glomeruli are usually larger than normal and the capsule is thickened to a moderate extent, there is some cellular infiltration of the glomerular tuft, the epithelial lining of the chamber has undergone marked proliferation, and the walls of the capillaries frequently show signs of hyaline degeneration. The tubules of the cortex are in places dilated, in others collapsed, in others presenting over small areas perhaps fairly normal characters. In the dilated tubules the epithelium may be shed, but where it remains it has commonly undergone hyaline, and in some places fatty, degeneration. The tubules generally contain casts. The interstitial tissue shows usually considerable overgrowth of fibrous tissue, and there is more or less abundant small-celled infiltration. The vessels are not commonly unduly prominent on section, but microscopically they frequently show thickening of the sub-endothelial layer of the intima to a moderate extent.

In the other form of chronic Bright's disease, where the kidneys are white and contracted, the capsule is usually thickened and on stripping leaves a granular surface, the granulations of which are frequently large and exceedingly well marked. The cortex is notably diminished in thickness, measuring frequently not more than one-eighth or one-sixteenth of an inch. The capsule although thickened is not invariably unduly adherent. Microscopically in this form of the disease there is a great increase of fibrous tissue; the glomerular capsules are frequently thickened, and it is not uncommon for many of the glomeruli to be reduced to small rounded masses of fibrous tissue in the centre of which are the remains of the glomerular tuft in a condition of hyaline degeneration. The epithelium lining the glomerular chamber frequently presents no very obvious changes. The tubules are in places dilated, but over large areas of the kidney they are contracted and the diminution in the size of the whole organ is very largely dependent on this contraction. Where the tubules are dilated they are frequently lined by cubical glass-like epithelium. Here and there tubules may be found lined with

more or less normal epithelium. In some parts the dilatation of the tubules is sufficiently marked to produce cysts, and these are commonly lined by a low, flattened epithelium. In many parts the epithelium is shed and occupies the lumen of the tubules. The connective tissue is considerably increased in amount, and septa may be seen passing in from the capsule towards the centre of the organ. The vessels are frequently unduly prominent and show very marked thickening of their coats, the thickening being mainly dependent on an overgrowth of fibrous tissue in the sub-endothelial layer, but the muscular coat may have undergone some increase in thickness. Although the renal vessels are commonly affected to a marked extent in this form of Bright's disease, the degree of arterial degeneration does not vary *pari passu* with the shrinkage of the kidney. Cases of very small contracted kidney may be seen where the arterial changes are less well marked than in larger specimens of the same malady.

Both in acute and in all forms of chronic Bright's disease lesions are frequently found in other organs of the body. Thus secondary inflammations, pleurisy, pericarditis, peritonitis, and pneumonia are common, pericarditis especially so, both in the dry form and with effusion. Where effusion is present it may be serous, sanguinolent, hæmorrhagic, or purulent. Both in acute and chronic Bright's disease the heart and blood-vessels often show signs of disease, and the heart may show definite signs of hypertrophy in as short a time as six weeks from the onset of acute Bright's disease. Although, speaking broadly, the cardiac hypertrophy is most marked in cases of contracted white kidney, this is not invariably the case, and in some instances of large white kidney very considerable cardiac hypertrophy is found. The degree of the cardiac hypertrophy would seem to be correlated rather with the degree to which changes in the sub-endothelial coat of the arteries are widespread. It certainly is not directly correlated with the diminution in the size of the kidney, as cases of extremely contracted white kidney may be seen with little or no cardiac hypertrophy, and, on the other hand, this may be marked and even cause death from cerebral hæmorrhage in cases of so-called large white kidney.

SYMPTOMS.—The onset of acute Bright's disease is usually sudden, and the initial symptom may be the occurrence of dropsy. In other cases general symptoms, such as fever, headache, vomiting, occur at the onset, and pain or diffuse aching in the back may also be present; pain, however, is not usually a prominent symptom. The dropsy is most marked in the subcutaneous tissues, more especially over the sacrum, and in vulva and scrotum; puffiness of the eyes is also well marked. In some cases the anasarca affects almost the whole of the subcutaneous tissues from the commencement. In all cases of acute Bright's disease the urine undergoes profound changes, and in some cases the urinary changes are the first to attract attention, the general symptoms being ill marked. The urine is diminished in quantity, and in some cases suppression very soon occurs; it is more usual, however, for the suppression to be partial. Where complete suppression occurs severe uræmic symptoms, such as coma and convulsions, are very rapidly developed and death soon takes place. The urine is generally smoky in acute Bright's disease, and it frequently contains large quantities of

blood, which gives it a uniform red colour; the blood-pigment may however have undergone changes so that the urine is dark or even porter-coloured. The urine of acute Bright's disease deposits casts and blood-corpuscles in large quantities. Albumen is always present, and commonly the urine becomes solid on boiling during the acute stage of the disease. Occasionally the quantity of albumen is less than this, but it is always large. The excretion of urea is diminished, but this is in part dependent on the diminished appetite, nausea, and vomiting which are commonly present in the disease. In some cases of acute Bright's disease the urinary changes are exceedingly well marked, the urine being highly albuminous, scanty, and containing large quantities of blood, and yet dropsy and the general symptoms of the disease may be comparatively slight. In others dropsy and the general symptoms are more prominent characteristics, but the urine, though highly albuminous and scanty, does not contain large quantities of blood. Uræmic symptoms of different degrees of severity occur in all forms of acute Bright's disease, and gastric symptoms such as anorexia, nausea, and vomiting are almost always present; and in the severe forms of the malady accompanied by suppression of urine the more serious uræmic manifestations such as delirium, dyspnoea, coma, mania, epileptiform seizures, are commonly present, and death in acute Bright's disease is not infrequently dependent on these uræmic complications.

Anæmia is always a prominent symptom and is developed rapidly, and the skin of the body is dry and harsh.

Epistaxis, hæmorrhage into the gums, and a general sponginess of the gums are frequently present.

Secondary inflammations such as pleurisy, pneumonia, pericarditis are common. Cardiac dilatation apart from the occurrence of pericarditis may also occur even early in the course of the disease.

The pulse in most cases of acute Bright's disease soon shows signs of high tension, and in the course of a few weeks, if the disease does not subside, the heart may show signs of evident enlargement dependent on hypertrophy.

Retinal changes are not so common in acute as they are in chronic Bright's disease, but papillitis, hæmorrhages, and retinitis may occur.

It is probable that the acute Bright's disease following scarlet fever and characterised by marked changes in the glomeruli, is more frequently accompanied by hæmaturia than the Bright's disease said to be due to exposure to cold.

Chronic Bright's disease.—As mentioned above a very large proportion of cases of Bright's disease are chronic from the beginning, but in a certain proportion the malady directly follows on the acute disorder or is a sequel to it; in other words some cases of acute Bright's disease pass on to the chronic stage without the urine regaining its normal characters, in others the chronic disease begins insidiously after an attack of the acute disease and with an intervening period of good health.

The initial symptoms of chronic Bright's disease present great variety. In a large group of cases the initial symptoms are a progressive failure of strength together with considerable wasting. In others the occurrence of dropsy more or less suddenly is the first sign of ill health. In others uræmic symptoms such as nausea, vomiting, and cramps, and in others,

severe headache, palpitation, and symptoms dependent on the disturbance of the circulatory system are those which first attract attention.

In the form of the disease associated with the so-called large white kidney dropsy is not only a prominent but often a very persistent feature of the illness and produces the well-known bloated appearance associated with a certain group of cases of chronic Bright's disease. Dropsy is not only well marked in the subcutaneous tissues but often also in the serous cavities, more especially the pleura. The dropsical fluid is of low specific gravity and contains relatively a small amount of albumen, much less than that seen in cardiac dropsy. The subcutaneous dropsical fluid contains a smaller percentage of proteins than that seen in the fluid in the serous cavities.

Anæmia is a marked feature of the malady, the skin and mucous membranes being pale and the complexion pasty.

Purpuric hæmorrhages in the skin are not uncommon, and in severe cases similar hæmorrhages in the mucous membrane of the mouth are also present. Epistaxis may also occur.

Patients with chronic Bright's disease are always weak and frequently considerably wasted, but the emaciation is often masked by the presence of dropsy. Uræmic symptoms are usually present in all cases that are severe, and are especially apt to assume a gastro-intestinal form, so that nausea, vomiting, hiccough are commonly present in all but the slightest forms of the disease. In the more serious forms dyspnoea, drowsiness, delirium, and epileptiform seizures may occur, and death in chronic Bright's disease is not uncommonly due to such complications, although fatal uræmic seizures are more frequent in association with contracted white kidney than with the so-called large white kidney. Cardiac symptoms such as palpitation, præcordial anxiety and distress, and shortness of breath are also usually present, and patients presenting these symptoms usually show signs of cardiac hypertrophy and dilatation and not uncommonly of mitral regurgitation dependent on degenerative processes in the ventricle leading to extreme dilatation.

The pulse usually shows signs of high tension, but not to the degree that is seen associated with the contracted white kidney. In cases where high tension is marked, a throbbing headache, greatly aggravated by changes of posture, is often present. Degeneration of the vessels can often be detected by ophthalmoscopic examination, which reveals not only the wiry and thickened arteries, but also the presence of albuminuri retinitis, hæmorrhages, and papillitis. See OPHTHALMOSCOPE IN MEDICINE, Coloured Plate.

In the large white kidney the urine is diminished in amount when dropsy is present, twenty ounces or less being secreted in the twenty-four hours. The urine is usually pale in colour and contains a large but variable amount of albumen, so that it may deposit after boiling as much as a half, three-quarters, or even become solid. Casts are usually present in abundance, especially hyaline and granular, but in addition casts containing renal epithelial elements and showing signs of degeneration are frequently found. Fatty casts are especially of grave import. The urine is always greatly diminished in quantity when dropsy is present, and fluctuations in the quantity of urine are often associated with fluctuation in the amount of the dropsy.

In the form of chronic Bright's disease where the kidneys are contracted, described above as contracted white kidneys, the symptoms are somewhat different; dropsy in these cases is not only not a marked symptom, but it may be completely absent, and when present it is generally slight in amount. On the other hand, anæmia and nutritional symptoms such as wasting and loss of strength are prominent, and such patients usually come under observation on account of a progressive failure in health, or else owing to the sudden onset of uræmic symptoms. Many such patients seek advice on account of persistent headache, and on examination of the fundus oculi albuminuric retinitis is found.

Uræmia is in the opinion of the writer extraordinarily prone to occur as a complication or even as an initial symptom in these cases. In other words the malady is apt to run a latent course until the onset of uræmic symptoms denotes the gravity of the underlying disease. Many such patients present no symptoms of ill-health until a series of epileptiform seizures or the occurrence of delirium, drowsiness, and coma attract attention to the renal lesion. Cardio-vascular degeneration is usually well marked in such cases; the heart is obviously hypertrophied and a high-tension pulse is present. Such patients not uncommonly suffer from cerebral hæmorrhage.

Albuminuric retinitis and papillitis are marked features in these cases and are more frequently present than in the other form of chronic Bright's disease. The urine is abundant, pale, of low specific gravity, but contains usually a considerable quantity of albumen, e.g. a quarter, a third, or even a half.

It is possible that some of the cases presenting these symptoms are to be regarded as the later stages of the so-called large white kidney, but, as insisted on above, many of these cases give no history of dropsy, and the whole illness is latent, or gradual and insidious.

COMPLICATIONS.—Many of the complications that occur in Bright's disease have been enumerated among the symptoms, as, for instance, the secondary inflammations and the occurrence of uræmia. Some, however, are of sufficient importance to merit separate mention.

Nausea and vomiting frequently occur and are dependent usually on uræmia. Sometimes, however, actual *gastritis* is present, leading to severe and incessant vomiting, and is thought to be dependent on the vicarious excretion by the stomach of retained urinary constituents such as urea.

Profuse *diarrhœa* dependent on enteritis is also not uncommon, and in a small proportion of cases ulcerative colitis is present in the large intestine and in the vicinity of the cæcum. *Peritonitis* is also occasionally seen as a complication of Bright's disease, and it may occur with or without effusion.

Pericarditis is a very serious and frequent complication of all forms of Bright's disease, and is more especially important from the fact that it is apt to be latent and so not produce marked symptoms. In some cases the effusion in pericarditis has been found to be sterile, but it is most frequently dependent on secondary microbic infection.

Phthisis is occasionally seen in cases of Bright's disease, or more usually Bright's disease is seen as a complication of phthisis; and although waxy disease of the kidney is especially liable to be present under these circumstances ordinary chronic Bright's disease is sometimes associated with phthisis.

Inflammatory complications such as pneumonia

and pericarditis and such diseases as phthisis may run an afebrile course when associated with chronic renal disease.

The *cardiac hypertrophy* usually present may result in the development of such cardiac lesions as dilatation, mitral regurgitation; and the arterial thickening and high tension may lead to the development of *cerebral hæmorrhage*. This is more especially apt to occur in the contracted white kidney, but is also seen in the other form of chronic Bright's disease.

Not only are *secondary inflammations* apt to occur in patients suffering from Bright's disease, but erysipelas and cellulitis may develop in the subcutaneous tissues, more especially after incision.

Cutaneous rashes, eczema, papular eruptions, erythematata, and bullæ are not uncommonly associated with chronic Bright's disease and more especially when uræmia is present, and occasionally these rashes are seen as prodromal or initial manifestations of uræmia.

DIAGNOSIS.—The diagnosis of acute Bright's disease is readily made in the majority of instances owing to the presence of dropsy together with the changes in the urine characteristic of acute inflammation, such as the presence of blood, albumen, and casts. Difficulties occasionally present themselves owing to the absence of dropsy in some cases and owing to the fact that blood may be present in the urine in other conditions besides acute Bright's disease. See KIDNEY, Diseases of.

Acute Bright's disease may have to be distinguished from (1) transitory nephritis seen in acute febrile illnesses, (2) hæmorrhagic infarction, and (3) passive congestion of the kidney secondary to heart-disease.

In the nephritis of acute febrile diseases dropsy is usually absent, and if hæmaturia be present the amount of blood is usually small; in the vast majority of instances hæmaturia is not present.

In hæmorrhagic infarction the urine not uncommonly contains a considerable quantity of blood and albumen, and the onset of the condition may be sudden and accompanied by pain and fever, so as to produce a superficial resemblance to the onset of acute nephritis. The effects however are usually transitory, and there is no dropsy. It is important to differentiate between hæmorrhagic infarction and acute Bright's disease, more especially in cardiac cases, as the prognosis of the two conditions is very different.

In passive congestion of the kidney blood and albumen may be present in the urine, and inasmuch as the condition arises usually in cases of heart-disease dropsy is frequently present, and it may be difficult in such cases to determine accurately whether acute inflammation is or is not present. In passive congestion the hæmaturia is usually though not always scanty, in acute inflammation it is more often abundant. In passive congestion hyaline and blood-casts are commonly present, in acute Bright's disease casts containing definite renal elements occur, and in this way a differential diagnosis may be made. Further, the dropsy of cardiac disease is usually different in its distribution from that of renal disease.

Chronic Bright's disease and granular kidney may sometimes be confounded with acute Bright's disease, inasmuch as in both the former conditions hæmaturia profuse in amount may occur, and in some cases this hæmaturia is dependent on hæmorrhage from the mucous membrane of the pelvis of the kidney. This is more especially the case in the granular kidney, and

the contracted white kidney, and such cases present considerable difficulties in diagnosis. Usually the presence of cardio-vascular changes and of albuminuric retinitis will afford evidence that the patient is not suffering from a primary attack of acute Bright's disease.

It is said that some cases of acute Bright's disease occur where, notwithstanding the presence of dropsy, there is not only no blood in the urine but no albumen. In these cases however the absence of albuminuria is usually temporary.

The main difficulty in the diagnosis of chronic Bright's disease is to determine accurately what form of chronic Bright's disease is present. In addition to this it is sometimes not easy to differentiate between some forms of chronic Bright's disease and the so-called physiological or *cyclical* albuminuria.

Chronic Bright's disease when accompanied by dropsy is usually readily diagnosed. Mistakes are most apt to occur in the form of the malady described above as the contracted white kidney where dropsy is absent. If the urine is not carefully examined in such cases, the presence of headache and vomiting may lead to the error of some intra-cranial disease being suspected, and this is especially liable to occur if some cerebral complication such as hemiplegia be actually present. Uremic manifestations such as epileptiform fits are not uncommon as initial manifestations of the contracted form of white kidney, and in such cases the error of diagnosing epilepsy, where the much more serious disease is really the cause of the symptoms, is not unusually made.

Examination of the fundus oculi and the detection of cardio-vascular changes, together with the examination of the urine, will generally enable a differential diagnosis to be made, but it must be remembered that albuminuria may occur in epilepsy. See EPILEPSY.

The presence of considerable wasting, together with the occurrence of hæmaturia in some cases of contracted white kidney, has led to this variety of chronic Bright's disease being confounded with malignant disease of the kidney.

Physiological albuminuria can usually be diagnosed owing partly to the absence of signs implicating the cardio-vascular system and the fundus oculi, and also owing to the variability of the albuminuria and the influence of diet, exercise, and posture upon it.

Cases of chronic renal disease are occasionally seen with marked symptoms of uræmia and disturbance of nutrition, such as great wasting, without the presence of albumen in the urine, although it is probable that in such cases the absence of albumen is temporary. These cases present great difficulties in diagnosis, and mistakes are occasionally unavoidable.

In a few cases pigmentation in chronic renal disease occurs to such a marked degree as to produce a superficial resemblance to that seen in Addison's disease. See ADDISON'S DISEASE.

PROGNOSIS.—Acute Bright's disease complicating scarlet fever has often a more serious prognosis than that depending on other causes, although the ultimate outlook in these latter cases may also be unfavourable. In other words, acute Bright's disease following scarlet fever is more immediately dangerous, but that following exposure to cold or dependent on alcoholism may lay the foundation for subsequent chronic Bright's disease.

The unfavourable symptoms in acute Bright's disease are the persistence and increase of the dropsy

and anæmia, together with the development of uræmic symptoms associated with the suppression of urine.

Acute uræmia occurring in acute Bright's disease is, however, by no means invariably fatal.

In a considerable proportion of cases of acute Bright's disease the dropsy subsides, the urine ceases to be albuminous, and the patient regains health. In a certain proportion of instances, however, the condition persists and becomes chronic. Even in cases where the urine regains its normal composition, the patient runs considerable risks of a recurrence of the trouble, as the result of indiscretions in diet or exposure to damp and cold.

The prognosis of chronic Bright's disease, speaking generally, is unfavourable, although patients may live for many years. The prognosis is very uncertain owing to the fact that patients suffering from chronic renal disease even when apparently in good health may develop complications rapidly fatal, such as cerebral hæmorrhage or acute uræmia. Cases are seen, however, where patients may for ten or even twenty years pass a urine scanty in amount and containing large quantities of albumen. It is usually stated that where the condition has persisted for as long as a year recovery does not occur. This statement, although doubtless generally true, is not of universal application.

The presence of signs of marked cardio-vascular changes and of albuminuric retinitis is unfavourable, and it is usually said that when albuminuric retinitis is present death occurs generally within a year. This, however, is not true of all cases, as exceptionally life may be prolonged for as long as five, six, or even seven years after the presence of albuminuric retinitis has been detected.

TREATMENT.—In acute Bright's disease the patient must be kept warm and confined to bed until all the acute symptoms have subsided. The diet must be restricted to one as simple in character as possible, such as milk, gruel, and barley-water in small quantities. During the height of the disease from one to three pints of milk in the twenty-four hours is sufficient. Milk should be given freely diluted with soda-water or barley-water. Stimulants should not be given. The activity of the skin must be promoted by hot drinks, the wet pack, and hot-air baths (see BATHS). The temperature of the bath should be from 120° F. to 170° F., and they should not be persisted in if they cause cardiac weakness. Sweating may be also promoted by the administration of jaborandi or pilocarpine, but it is not advisable to push these drugs to the extent of producing their well-known toxic symptoms, and it is often difficult to procure efficient sweating unless this be done. The combination of hot-air bath with the administration of jaborandi is often useful.

The partial suppression of urine that is present in all severe cases may be treated by the administration of diluents, but it is doubtful whether the stimulant diuretics are of much use or even suitable for these cases. Citrate of potassium and also nitrate of potassium may be used. Caffeine, however, is probably not suitable.

The cardiac weakness which is sometimes present during the early stages of the disease may be treated by the administration of digitalis and strychnine.

The dropsy may be treated by measures directed to promote the activity of the skin and of the bowels, and such simple drugs as the saline purges, sulphate of sodium, phosphate of sodium,

and sulphate of magnesium, may be used for this purpose. Jalap is also useful, but violent purgation is not advisable in acute Bright's disease, inasmuch as enteritis is one of the complications of the malady, and a profuse diarrhoea started by the administration of drugs may become uncontrollable and greatly increase the weakness.

Uræmic symptoms must be treated on the same lines as the dropsy, by promoting the activity of the skin and the bowels, but if they are severe these methods may fail, and then venesection and saline transfusion are useful. Chloroform may be used to diminish the severity of the uræmic convulsions, and morphine also may be used with discretion for this purpose, but is held by some authorities to be dangerous, and many forbid its use altogether.

The severe anæmia that accompanies acute Bright's disease and persists during the convalescence must be treated by the administration of iron and arsenic.

In convalescent cases the diet should be gradually increased. Fish and white meats should be given first, and all highly seasoned and elaborately cooked articles of food avoided.

The treatment of chronic Bright's disease is in some respects similar to that of acute, in that if dropsy be present, the activity of the skin must be promoted by the same methods. If the disease is of recent origin or severe, the patient should be kept in bed and the diet restricted to milk. In chronic Bright's disease a larger quantity of milk will be required than in the acute malady, since four pints *per diem* is a minimum quantity for an adult. A milk-diet is also indicated in all cases of chronic Bright's disease presenting uræmic symptoms. The milk-diet should be persisted in for some weeks, but if under this treatment the dropsy and the other symptoms of the disease do not improve, it is probably advisable to increase the diet to a moderate extent, allowing fish and white meats, and if improvement occurs the ordinary meats may be allowed. Many cases of chronic Bright's disease, where anæmia is a marked symptom and where the disease is of some duration, progress more favourably if given a moderate diet, and it is probably unadvisable to treat all cases of chronic Bright's disease with a rigid milk-diet. In all cases of chronic Bright's disease highly seasoned articles of diet and rich concentrated foods should be avoided, and at all times such patients should take only small quantities of meat. Alcohol is probably harmful in all forms of chronic Bright's disease, and should be only given, if at all, in small quantities and freely diluted.

The dropsy of chronic Bright's disease is often very persistent, and in some cases it is of mixed origin, being partly true renal dropsy, and partly cardiac dropsy dependent on cardiac failure secondary to the renal disease. In some cases where the dropsy is not relieved by hot-air baths and other measures directed to increase the activity of the skin, incisions over the malleoli may become necessary, but in cases of renal disease extra precautions require to be taken to prevent septic complications.

The anæmia of this disease is often very marked and should be treated by the administration of iron and arsenic.

JOHN² ROSE BRADFORD.

BROMIDROSIS (*βρώμος*, a stench; and *ἰδρῶς*, sweat).—A term for fetid perspiration. *See* PERSPIRATION, Disorders of.

BROMISM.—*See* DRUG-ERUPTIONS.

BRONCHI, Diseases of.—SYNON.: *Fr. Maladies des Bronches*; *Ger. Bronchien-Krankheiten*.—The diseases of the bronchi will be discussed in the following order:—1. Acute inflammation; 2. Chronic inflammation; 3. Plastic inflammation; 4. Dilatation; 5. Narrowing or obstruction; 6. Cancer.

1. Acute Inflammation—Acute Bronchitis—Acute Bronchial Catarrh.

DEFINITION.—An acute inflammation of the bronchial tubes.

ÆTIOLOGY.—The causes of acute bronchitis may be classed as (*a*) *predisposing*, and (*b*) *exciting*.

(*a*) *Predisposing causes.*—Of these age is one of the most important. The disease is most frequently met with in the young and the old; and in these subjects it assumes its most serious characters. The imperfectly developed thorax of the infant, and the diminished vitality of the aged, seem to render them especially liable to attacks of bronchitis, and to make the disease exceptionally fatal in them. Sex appears to have no influence as a predisposing cause. The habits of life have an important influence in the causation of bronchitis. The practice of living in heated rooms, especially where gas is largely consumed, and of breathing the vitiated atmosphere produced by the assemblage of large numbers of persons in a single room, is undoubtedly a fertile predisposing, as well as exciting, cause of the complaint; so also is the practice of keeping children too much within doors on the one hand, or, on the other, of exposing them to inclement weather when improperly clad. The state of the general health exercises a powerful influence. A weakly constitution, or one weakened by overwork, improper food, &c., predisposes to bronchitis; while such affections as Bright's disease, gout, and diseases of the heart, alike favour its occurrence. The climate most favourable to the production of bronchitis is probably that which is at the same time both cold and damp, and where sudden variations of temperature occur. The seasons of the year in which it prevails most are the late autumn, the winter, and the early spring.

(*b*) *Exciting causes.*—Although undoubtedly cold directly applied to the surface of the body is in a large number of cases the exciting cause of bronchial inflammation, still the transition from cold to heat—passing from a cold atmosphere to a heated one—is an important factor of the disease. There can be little doubt that bronchitis is often produced directly by the effects of heated and vitiated air on the bronchial mucous membrane. Bronchitis may also be caused by the direct action of irritants contained in the air—as irritant vapours, minute particles of steel, cotton, or ipecacuanha, and the pollen from flowering plants. The toxins which are present in the system in the course of specific febrile affections often act as exciting causes of the disease; as do also the poison of syphilis, and the altered condition of the blood produced by gout. Bronchitis, moreover, must be regarded as a constant accompaniment of influenza. According to Ritchie acute bronchitis is an infective disease. The bronchitic secretion in the smaller tubes contains in every case many varieties of bacteria. In some the number of these is so great as to suggest a causal relationship, especially in the case of the *Diplococcus pneumoniae* and the *Streptococcus*.

ANATOMICAL CHARACTERS.—The mucous membrane is mainly affected in acute bronchitis, but morbid changes may be produced in the deeper structures. The mucous membrane is red—the redness being arborescent, streaked, or mottled, but not usually spread uniformly over a large surface. The injected condition of the membrane does not, as a rule, extend into the finer bronchial tubes, but in some cases where there have been frequent attacks of inflammation, these tubes have a red appearance. The membrane is sometimes thickened and soft, but ulceration is very rare. The tubes are generally found more or less filled with secretion, either frothy mucus, muco-pus, or even actual pus. Sometimes the secretion is very abundant, filling all the tubes. Fibrinous masses are occasionally met with, which may form casts of the tubes. Collapse of portions of lung-substance is not infrequently found, as are also patches of lobular pneumonia. The venous system and the right side of the heart are overloaded, and the blood is dark. In many cases fibrinous deposits are found in the cavities and great vessels of the heart.

In discussing the pathology of bronchitis, it is necessary to refer to the distribution of the bronchial blood-vessels. The bronchial arteries when they have fairly entered the lungs have no accompanying veins. The so-called bronchial veins are some small vessels which return the blood supplied to the structures about the roots of the lungs. The blood which is supplied to the bronchial tubes, when they have commenced their divisions, passes into radicles of pulmonary veins, and is returned directly to the left side of the heart. It is uncertain whether there are any anastomoses between the bronchial arteries and the pulmonary artery. If such communication exist, it is only slight. The blood of the bronchial arteries, after supplying the mucous membrane and other structures of the tubes, passes, either wholly or in a very large part, to the left side of the heart, not having circulated through the aerating portion of the lungs. The circumstances of this anatomical arrangement are most important from a practical point of view. Anything which embarrasses the circulation on the left side of the heart—such as mitral regurgitation—must necessarily cause a very loaded condition of the bronchial vessels; and all physicians are familiar with the form of bronchitis which is so common in these cardiac affections. The congested mucous membrane, and the profuse bronchial secretion, are the result of the direct impediment to its circulation which the blood meets with, from passing at once into vessels which go straight to the left side of the heart. The relief often afforded in this form of bronchitis by the administration of digitalis is explained by the circumstance above referred to.

SYMPTOMS.—The symptoms of acute bronchitis differ according as the inflammation is located in (a) the large and medium-sized tubes, or (b) the small ones. To the latter form of the affection the name of *capillary bronchitis* has been given.

1. Acute bronchitis of the *larger tubes*.—The attack is usually ushered in by symptoms of catarrh—sneezing, lachrymation, a sense of fullness about the nose and eyes, with frontal headache; the throat becomes dry and sore, and then increased secretion sets in; the follicles at the back of the pharynx become enlarged; the upper part of the larynx is often involved, there being slight hoarseness; and the affection gradually creeps down into the

bronchial tubes. The disease is not ushered in by decided rigors, but chills and sometimes shiverings are experienced; the pulse is not much affected, but its frequency is increased in some cases; there is a general sense of malaise, as well as a want of energy. When the disease has set in fully certain local symptoms are found. More or less pain is felt behind and above the sternum; the sensation is increased by a deep inspiration; the pain shoots at times over the chest in the direction of the larger bronchial tubes; and there is a tickling or unpleasant irritation felt behind the sternum, which gives rise to a cough. Dyspnoea is not a marked feature of this form of bronchitis; it exists, however, sometimes; and in the most severe cases a sense of oppression, weight, and tightness about the chest is experienced. Cough is one of the earliest and most prominent symptoms; it is at first dry, and there is usually at this period some hoarseness. The cough is paroxysmal, and often very violent; it becomes attended with expectoration as the disease progresses. This varies at different stages of the affection; at first watery and frothy, and almost transparent, it becomes, as the disease progresses, more consistent, viscid, and opaque, consisting successively of mucus, muco-pus, and pus; it is sometimes distinctly nummulated. Small streaks of blood are occasionally seen mixed with the sputa. Examined under the microscope the sputa are found in the early stages of the disease to contain epithelial cells from the mucous membrane; and, later, many of the so-called exudation-corpuscles, molecular and granular matter, pus-cells, and occasionally blood-discs.

In the milder cases of this form of bronchitis there is but little general disturbance; and even in the more severe cases the febrile reaction is not usually very great. The pulse rises a little, but does not become very frequent; the temperature rarely becomes high; there is in many cases but little interference with the appetite. A general feeling of depression, which in some cases is very marked, is usually experienced.

2. Acute bronchitis of the *smaller tubes*.—*Capillary bronchitis*.—This is a very formidable disease. It attacks the finer bronchial tubes, and probably extends to their smallest ramifications. Its symptoms are very grave. Some of the worst cases of capillary bronchitis are met with in connection with emphysema of the lungs. It may be an extension of inflammation from the larger tubes; or the capillary tubes may be attacked simultaneously with the larger ones, or alone. The early symptoms are more severe than those of ordinary bronchitis, and rigors are more common. Dyspnoea is marked; it may vary from mere rapid respiration to constant or paroxysmal orthopnoea. The respirations may rise to fifty in a minute. Cough is almost continuous, at times becoming very violent and most distressing. Expectoration is attended with difficulty. The sputa soon become very abundant, and rapidly assume a purulent character; or they are very viscid and ropy. *See* SPUTUM, Examination of.

The general symptoms are very severe. The temperature may reach 103° F. or even higher, though it is rarely as high as in acute tuberculosis or pneumonia; and the pulse may be 120 or 140. There are often profuse perspirations, and in some cases excessive debility is felt. If the disease progresses unfavourably, symptoms of very imperfect

aëration of the blood come on. The face becomes turgid and bloated, the lips and ears get livid, the veins are distended, the temperature falls, cold clammy perspirations break out, the pulse becomes very small and rapid, delirium supervenes, the respiration is shallow and catching, and the patient dies of asphyxia, or from the presence of fibrinous clots in the heart and great blood-vessels.

PHYSICAL SIGNS.—The physical signs of both forms of acute bronchitis may be referred to together. Inspection reveals little of practical value in simple bronchitis. The form of the chest is not altered. In severe cases the abdominal movements are in excess. The costal movements are frequently those of elevation rather than expansion. In extreme cases the lower end of the sternum and the connected cartilages sink with inspiration; while the expiratory movements are slow, laboured, and inefficient. If the hand is applied to the chest, rhonchal fremitus may be often felt, sometimes over a large area. The percussion-sound may be somewhat exaggerated from over-distension of the lungs, especially in children; not appreciably altered; or deficient in resonance, owing to the accumulation of secretion at the bases of the lungs, to œdema or congestion (as in typhoid fever), or to pulmonary collapse. In young children a sound resembling the cracked-pot sound may be occasionally produced, variable in site. The sounds heard on auscultation vary according to the stage of the disease. The breath-sounds are loud when the tubes are free; when the latter are plugged by secretion, they often become feeble or even totally suppressed, from closure of a tube leading to a portion of the lung. The adventitious sounds of bronchitis include the various rhonchi and râles; the dry rhonchi are heard in the early stages of the disease for the most part, but when once secretion has set in, the moist râles are more or less extensively heard, depending for their character on the size of the tubes which are the seat of inflammation. Thus they are called *mucous* when produced in the large tubes, *sub-mucous* and *sub-crepitant* when produced in the finer ones; the latter term being used to characterise the râles of capillary bronchitis. When the large bronchial tubes are filled with a secretion which is not viscid, the sounds may have a rattling character. The various rhonchi may be heard over different parts of the lungs at the same time, according to the seat and stage of the bronchitis. In capillary bronchitis sub-crepitant râles, accompanying inspiration and expiration, are abundantly heard, especially towards the bases of both lungs. As a rule there is no displacement of organs in bronchitis, but the diaphragm is sometimes depressed from great distension of the lungs, and the heart is occasionally displaced towards the right.

DIAGNOSIS.—The diagnosis of acute bronchitis, except in a few instances, presents no great difficulty. In the early stages of whooping-cough it is impossible to decide whether the case is one of simple bronchitis or not, but subsequently the paroxysmal character of the cough settles the point. In some cases of bronchitis occurring in children the breathing may resemble that of croup; but here the presence of catarrh, the wheezing nature of the respiration, the absence of much fever, the characters of the sputa—obtained by wiping the back of the tongue—and their freedom from membranous shreds, and the physical examination of the chest indicating the presence of rhonchi, will be

sufficient to establish a diagnosis. From laryngitis the discrimination is not difficult.

Pneumonia may generally be easily diagnosed from capillary bronchitis, with which form it can perhaps be alone confounded. Capillary bronchitis is not ushered in, as pneumonia usually is, by a well-marked and prolonged rigor; the general febrile disturbance is less, and the temperature not so high; moreover the absence of dulness on percussion, and of increased vocal resonance and fremitus, will aid in the differentiation. From lobular pneumonia in children the diagnosis is not always easy. In this disease there is often no dulness to be perceived on percussion; while, on the other hand, dulness may exist in bronchitis from pulmonary collapse.

The diagnosis of capillary bronchitis from acute phthisis often presents difficulties. The main points to be relied on, independently of the family history, which may aid, are that in capillary bronchitis the fever is less and the temperature lower; signs of asphyxia soon come on; and there is free expectoration of muco-purulent matter, from which the tubercle-bacilli are absent. In one form of acute phthisis there is evidence of pneumonic consolidation, followed by signs of the formation of cavities. In the miliary tubercular form there are in many cases scarcely any physical signs except râles, most marked at the apices of the lungs. Here, however, the diagnosis may be aided by the examination of the sputa under the microscope, which may reveal the presence of tubercle-bacilli.

PROGNOSIS, DURATION, TERMINATION, AND MORTALITY.—The prognosis in an ordinary case of bronchitis is favourable, but should always be guarded when the disease occurs in the very young or the aged. In the milder forms the affection may last only a few days, or two or three weeks. Severe cases are more protracted. The disease may terminate in perfect recovery, in death, or by passing into the chronic form. It may be the starting-point of emphysema of the lungs, or of certain forms of phthisis. The mortality is much influenced: (1) by age, being greatest in the very young and the very old; (2) by the previous state of health, which, if lowered by any circumstances, will render recovery more doubtful; (3) by the extent of the inflammation, especially when the disease is of the capillary form; (4) by the existence or non-existence of any organic disease of the heart, lungs, or kidneys; (5) by the disease being epidemic in connection with influenza or otherwise; and, lastly, by the time the case has come under treatment, whether early or late.

TREATMENT.—In the treatment of bronchitis care must be taken to ascertain whether the disease is secondary to some organic affection, or the result of mechanical irritation, or of the presence of gout or rheumatism in the system, or of influenza; or whether it arises as a primary disease. The treatment of the disease as a primary affection will be considered first.

In an ordinary case of acute bronchitis the patient should be confined to his room, and, if the case is at all severe, to his bed. The temperature of the apartment should be maintained at from 60° to 65° F. In the early stages of the attack it is well to allow the air of the room to be more or less saturated with steam. A free action of the skin should be promoted; and for this purpose warm drinks, with or without some form of alcohol or

some diaphoretic medicine, may be given; or a hot-air bath may be used in bed. Great relief is often experienced from the application of a large mustard or mustard and linseed-meal poultice to the chest, but severe counter-irritation is to be avoided; and it is well, if mustard is applied first, to apply immediately afterwards a large hot linseed-meal poultice, to be renewed every few hours. This constant application of warmth and moisture to the chest is often productive of very great relief to the symptoms.

It is generally desirable to act on the bowels, and a mercurial, followed by a saline purgative, will often be of great service. In the old and debilitated, as also in the young, all lowering treatment must, however, be avoided. In the early stages of the affection, before secretion has commenced, and when the mucous membrane is dry and the cough hard, diaphoretics with *ipeacuanha* may often be given with advantage; but as soon as secretion is fairly established, carbonate of ammonium, spirit of chloroform, ether, *cascarilla*, *senega*, or similar drugs should be administered. Indeed in almost every stage of bronchitis carbonate of ammonium is one of the most valuable remedies we possess. Care should be exercised, especially with the aged, that nothing should be given which will so nauseate as to prevent food being taken. In the exhibition of medicines to alleviate the cough, regard must be had to the condition of the patient and the stage of the disease. Opium in all its forms should be given with caution, and is generally inadmissible, especially in the young and old. It no doubt often succeeds in checking cough, but in doing so it also checks expectoration, and causes an accumulation in the bronchial tubes, which sometimes becomes very dangerous to life. Chloral hydrate in small doses is often of great use for relieving cough, and it may be combined with oxymel of squill. It has also a good effect in allaying spasm of the tubes, if this exist. In some cases of bronchitis the question of procuring sleep becomes an important one. Chloral hydrate may be given with safety, and the recovery of a patient may sometimes be dated from the sleep which this agent procures.

Alcoholic stimulants, except in the early stages, and in certain cases dependent on a gouty or rheumatic condition, should usually be given in smaller or larger quantities. They increase expectorating power, and ward off the tendency to asphyxia. In the old they are especially called for, and, together with carbonate of ammonium, should form the main therapeutic agents to be relied on. In the treatment of capillary bronchitis, ammonia and alcoholic stimulants should be exhibited from the commencement, and the quantity must depend on the symptoms of each case. There is one source of danger in capillary bronchitis which should always be borne in mind, viz. the formation of fibrinous clots in the heart and great blood-vessels. These deposits become the proximate cause of death in many cases, and they are especially liable to form when there is emphysema of the lungs. Their presence may often be diagnosed during life from the respiration becoming very rapid, shallow, and laboured; from the pulse being quick, weak, and small, although the heart may at the same time be felt beating vigorously; from the voice becoming feeble, and the mental faculties seriously impaired. After death a large portion of the cavities of the heart may be found occupied by these deposits, the calibre of the pul-

monary artery and the aorta being also materially diminished by them.

In many cases of bronchitis when the acute symptoms have passed off, but the secretion continues profuse, as well as in those cases called bronchorrhoea, the exhibition of iron is often of great service. It seems to give tone to the relaxed capillaries of the mucous membrane, and to diminish the secretion. It may be given in combination with carbonate of ammonium, in the form of the ammonio-citrate; or the tincture of the perchloride with ether or spirit of chloroform may be employed, or the ethereal tincture of the acetate (*Ph. Ger.*), which is a very valuable preparation in some cases.

Inhalations are useful for allaying cough, in the earlier stages of the affection, or for the relief of spasm. In some cases of severe bronchitis where asphyxia has been threatened, recovery has followed the exhibition of large doses—half an ounce—of oil of turpentine. In this dose, however, it sometimes produces alarming symptoms, and it is perhaps better to exhibit it in smaller quantities tentatively. An emetic may be serviceable, especially in children, if the tubes are much loaded. Children suffering from severe attacks of bronchitis should not be allowed to sleep long, for fear of dangerous accumulation in the tubes, and care should be taken that the secretions do not collect about the back of the mouth.

Patients should not be kept on a low diet even at the beginning of an attack, and as the disease progresses the quantity of food allowed may be increased according to the appetite. In the treatment of gouty bronchitis, colchicum and the alkalis must be given, and the general measures used which are applicable to the constitutional condition. If bronchitis depend on a gouty state, it will not yield to the ordinary treatment, but when its cause is recognised and the appropriate remedies are administered, the symptoms usually soon begin to improve.

In the cases of bronchitis which are connected with heart-disease, and especially with mitral regurgitation, digitalis is often of great value. By steadying the action of the heart it relieves the overloaded pulmonary veins, and thus directly diminishes the congestion of the mucous membrane, as mentioned in the paragraph relating to the pathology of the disease.

It is impossible in the scope of this article to refer specially to the treatment of bronchitic attacks arising from the various kinds of mechanical irritation. There is, however, one form of bronchitis which may be mentioned, viz. that connected with hay-fever, arising either from the inhalation of pollen, or caused by some peculiar atmospheric influence acting on a peculiar nervous system. It is very difficult of cure; no remedies seem to have any particular influence over it, and it is usually only to be relieved by removing the patient from the exciting cause of the affection. In the treatment of bronchitis depending on constitutional syphilis, the appropriate measures for that affection must be resorted to.

2. Chronic Inflammation—Chronic Bronchitis—Chronic Bronchial Catarrh.

DEFINITION.—A chronic inflammation of the bronchial tubes.

ÆTIOLOGY.—Chronic bronchitis very frequently results from repeated attacks of the acute disease, but it may be chronic from the beginning. Emphysema of the lungs, dilated bronchi, and tuber-

cular infection are causes of the complaint; as are also various forms of heart-disease, and some other diseases, such as gout. The prolonged inhalation of slightly irritating particles gives rise to chronic bronchitis; and it is also met with in connection with chronic alcoholism. It is most common among the old.

ANATOMICAL CHARACTERS.—The bronchial mucous membrane is discoloured, being of a dull-red tint, greyish, or brownish. The discolouration is usually partial, but sometimes general. There is swelling and increased firmness of the mucous membrane, and the sub-mucous tissue in old-standing cases becomes infiltrated and indurated. The fibrous tissue is increased; the cartilages in the larger tubes are sometimes calcareous; and there is generally more or less emphysema of the lungs.

SYMPTOMS.—The symptoms of chronic bronchitis vary greatly in different cases. They resemble in kind those of the acute affection. There is cough, expectoration, pain, soreness or uneasiness behind the sternum, with more or less dyspnoea. The constitutional symptoms may be very slight, scarcely any effect on the general health being apparent; or they may be very severe. Three forms of chronic bronchitis are recognised clinically:—(1) that which includes the ordinary cases of the disease, varying much in severity; (2) that characterised by excessive secretion—*bronchorrhœa*; and (3) that form which is called *dry catarrh*.

1. In the first form of chronic bronchitis the cough is at first slight, perhaps only occurring during the winter, being altogether absent in the summer. After a time the attacks become more frequent, and at last the patient is never free from the affection, which is aggravated at times. The cough in such cases is more or less severe, but usually most so in the morning. It is often paroxysmal, and sometimes very violent. The expectoration in some cases is scanty, viscid, and difficult to discharge, in others, especially old-standing cases, it is copious and easy. The sputa vary much both in appearance and quantity. They may be yellowish-white, muco-purulent, or more decidedly purulent, of a greenish-yellow or bright or dark green colour; they are but little aerated, sometimes not at all, so that they sink in water; at times they are nummulated and quite opaque. In some cases the expectoration is fetid, constituting the form of the disease denominated 'fetid bronchitis,' the odour resulting either from sloughing of minute portions of the mucous membrane, or from chemical changes taking place in the sputa. Occasionally streaks of blood are met with. Microscopically the sputa are found to consist of epithelium, pus-cells, and granular matter, with at times blood-corpuscles.

The constitution does not suffer much in mild attacks, but when chronic bronchitis is permanent and general, the system at large sympathises more or less severely: the appetite fails, sleep is disturbed by the cough, emaciation sets in and sometimes becomes marked, but it does not proceed beyond a certain point, unlike that of phthisis, which is usually progressive. In all cases of chronic bronchitis there is great risk of an acute attack coming on, especially among the aged. These attacks are very dangerous, in consequence of the rapid extension of the disease throughout the lungs, and its asphyxiating character.

2. The second class of cases is characterised by

excessive secretion from the bronchial tubes—*bronchorrhœa*. This form is often met with in the old and feeble, and especially in cases of valvular disease of the heart. The cough is paroxysmal, and attended with the expectoration of a large quantity of thin watery glairy fluid, or of thick ropy gluey matter, like white of egg. The quantity expectorated is sometimes very large. This form of bronchitis may cause death somewhat suddenly by asphyxia. During the paroxysms of cough there is dyspnoea, but at other times it is absent, except when heart-disease exists. The constitution suffers little, and the flux seems sometimes to be beneficial in cases of obstructive cardiac disease.

3. The third variety, or '*catarrhe sec.*' is characterised by very troublesome cough, oppression of breathing, tightness of the chest, and sometimes severe dyspnoea. Expectoration is either absent or very scanty, the sputa consisting of small masses of tough, viscid, semi-transparent mucus. There is usually no febrile disturbance. The disease is met with in gouty people, and is often associated with emphysema of the lungs.

PHYSICAL SIGNS.—Inspection reveals nothing abnormal in the form or size of the chest, unless emphysema of the lungs is present. The expansion in long-standing cases is usually deficient; the chest being raised more than in health. Expiration is often prolonged. Rhonchal fremitus may be felt more or less, depending on the state of the bronchial tubes. There is often increased resonance, from the presence of emphysema. The breath-sounds are more or less changed: they may be harsh and loud, and the expiration is prolonged in cases that have existed for a considerable period. The adventitious sounds vary: they are dry, coarse, moist, or bubbling according to the condition and contents of the tubes. Vocal resonance varies: it may be bronchophonic, normal, or deficient.

DIAGNOSIS.—There is usually but little difficulty in the diagnosis of chronic bronchitis. The affection is most likely to be confounded with phthisis, but the character and degree of the wasting, and the absence of increased temperature, of hæmoptysis, of tubercle-bacilli from the sputa, and of the physical signs of consolidation, will generally enable the practitioner to decide in favour of the less serious disease. The main difficulty lies in the diagnosis of cases where the bronchi are dilated; this will be referred to later on.

PROGNOSIS.—Although in itself not a dangerous malady, chronic bronchitis becomes so in consequence of the liability which exists to the occurrence of acute symptoms; when once established in middle or advanced age it is almost incurable. The complaint is further serious from its tendency to produce emphysema and dilatation of the bronchi. *Per se* it can scarcely ever be said to kill.

TREATMENT.—No case of chronic bronchitis can be successfully treated without due regard to the constitutional condition of the patient. In some cases it is impossible to cure the disease, and all efforts should be directed towards preventing its extension; alleviating the symptoms to which it gives rise; and warding off acute attacks. Attention must be paid to the state of the heart and kidneys; the duration of the affection; the age of the patient; the characters of the expectoration; the state of the lungs, as to the existence of emphysema or other morbid conditions; and the presence of gout or rheumatism. The functions of the liver must be

looked to: the administration of a few doses of blue pill with a saline aperient often gives great relief, and alters the character of the bronchial secretion. If the gouty diathesis is present, colchicum with alkalis and other remedies for gout, such as a course of Friedrichshall, Hunyadi, or Carlsbad waters, will prove of great service. If cardiac disease exist, whether in the form of valvular incompetence, or of weak, flabby, or dilated heart, digitalis combined with iron frequently produces marked benefit.

Chronic bronchitis, not dependent on any organic disease or constitutional condition, has a tendency to lower the health and to diminish strength, and therefore the various tonics may often be given with great benefit. Of these the most useful are quinine, the preparations of iron, and those of zinc. Cod-liver oil is also very valuable in some cases where there is much wasting. The cases of bronchitis marked by excessive secretion are generally best treated by tonics; while those in which the secretion is slight—cases of ‘dry catarrh’—being often associated with a gouty condition of the system, are more amenable to the use of colchicum, the alkalis, iodide of potassium, and mineral waters.

In what may be called the symptomatic treatment of the affection, the various expectorants are useful—carbonate of ammonium, ipecacuanha, squill, cascarrilla, senega, chloroform, &c., and these may often be beneficially combined with some form of tonic. In many cases of chronic bronchitis the expectorating power is diminished, and stimulating expectorants are of great service. Much caution must be exercised in the administration of opiates and other narcotics or sedatives. When, however, the mucous membrane is very irritable, and when there is but little secretion, with a troublesome cough, these remedies are indicated. Opium is of great value, and chloral hydrate is also very useful, as well as, in some cases, henbane and hydrocyanic acid, or, whenever spasm is present, stramonium, lobelia, the ethers, and cannabis indica. Inhalations are sometimes very beneficial, as of the vapour of warm water, iodine, creasote, and other substances. The inhalation of creasote is especially valuable if the expectoration is fetid.

Counter-irritation is one of the most important means we possess of relieving chronic bronchitis. The irritation should not be excessive, but should be long-continued. The application of iodine over a large surface of the chest, so as to keep up a constant slight inflammation of the skin, is perhaps the best that can be used; but other irritants may be tried, such as sinapisms, or the various stimulating liniments.

The general management of the patient is most important. A mild climate should, if possible, be chosen in the winter. The patient should live as far as possible in an atmosphere which is mild and dry. Although some cases are benefited by a moist and warm atmosphere, the majority of cases of chronic bronchitis do better in a drier one. The skin must be carefully looked to, its action should be well maintained, and warm clothing always worn. A moderate amount of some alcoholic stimulant is generally desirable; and the food should be nutritious and of easy digestion. Relief will often be found from wearing a respirator.

3. Plastic Bronchitis.—This is a rare form of disease, and of its particular causes nothing is known. It is perhaps connected with some peculiar diathesis. It may occur in either sex, and at any period of life,

but is most frequent in those who are of a tubercular constitution. It has been known, however, to attack persons of apparently healthy frame and in the enjoyment of robust health.

ANATOMICAL CHARACTERS.—Plastic bronchitis is anatomically characterised by the formation of concretions in the bronchial tubes. These concretions consist of fibrinous exudation from the mucous membrane; they form casts of the tubes, and are expectorated. These casts are either solid or hollow, and on examination are always found to consist of concentric laminae. They are, for the most part, poured out into the finer bronchial tubes, sometimes, however, into the larger ones, but never into the trachea. The casts are of a whitish colour, but they are often stained with blood. Microscopically they consist of an amorphous or fibrillated material, with leucocytes, granular matter, and oil-globules.

SYMPTOMS.—This disease is essentially chronic, but it has been met with as an acute affection in children. At the times when the casts of the tubes are expelled, exacerbations occur, the patient being attacked with pain and a sense of constriction across the chest, dyspnoea, and an irritating cough. After a time, varying from some hours to a few days, the dyspnoea becomes very urgent, and the cough very severe; then, after a paroxysm of coughing, it is found that the patient has expectorated some solid material, either with or without blood, usually intermixed with ordinary bronchitic sputa. The dyspnoea and cough now subside, to recur after an interval of a few hours or longer. This disease may last for weeks, months, or even years, marked from time to time by severe accessions, and relieved by the expulsion of further concretions. The matter expelled is often in small masses, but at times casts of bronchial tubes with several ramifications are expectorated. The disease may recur at intervals for many years; the general health in such cases does not seem to suffer, the breathing during the intervals being unaffected. There is, in some instances, an absence of febrile symptoms during the attack, while in others the fever is more marked. With the general symptoms are combined the *physical signs*. These are somewhat peculiar. The bronchial tubes being obstructed, portions of lung are deprived of air; the breath-sounds are therefore faint or absent. There may be dulness more or less complete on percussion, from collapse of the lung-substance, or, as occurs in some cases, from localised pneumonia. Asphyxia may be threatened if a large tube is blocked up.

DIAGNOSIS.—The diagnosis of this affection turns on the peculiarity of the expectorated matters. Doubtless the disease may be mistaken for ordinary bronchitis or pneumonia; but when once the fibrinous casts of the tubes are observed in the sputa, the nature of the case becomes clear.

PROGNOSIS, DURATION, TERMINATION, AND MORTALITY.—The prognosis, if the disease is uncomplicated, is favourable; but there is a great liability to recurrence. The complaint may last for many years, and may terminate in complete recovery. A fatal result generally depends on the presence of some other organic disease, such as phthisis or pneumonia.

TREATMENT.—But little can be advanced as to the value of any special treatment for this affection. Iodide of potassium is said to have been employed with success. The chief object should be to maintain the general health by hygienic measures, and the

exhibition of tonics, such as iron, quinine, and cod-liver oil, especially if there be any tubercular taint. During the exacerbations the administration of ammonia and the use of inhalations should be resorted to, and the general principles on which ordinary bronchitis is treated should be carried out.

4. Dilatation—Bronchiectasis.—This is a rare disease, which arises as a secondary affection. It is often associated with serious pulmonary mischief, and is at times difficult of diagnosis. There are two forms of bronchiectasis, namely, *general or fusiform dilatation*; and *saccular or ampullary dilatation*.

(1) The *general* or uniform bronchiectasis consists in a cylindrical dilatation of one or more of the tubes throughout a considerable portion of their extent. The tubes are evenly widened for the most part, and the dilatations end abruptly.

(2) The *saccular* form of bronchiectasis consists of a globular dilatation of a tube at one point, or at several points. The dilatations vary in size, being from half an inch to an inch or more in diameter. On the tracheal side they usually communicate with a slightly enlarged bronchial tube, while on the peripheral side the continuity of the tube is almost or entirely lost from narrowing or actual obliteration. Sometimes the cavities communicate with one another. The two forms of bronchiectasis often co-exist.

The walls of the dilatations undergo changes in the course of the disease. The mucous membrane becomes granular, swollen, and congested; while at a later stage it presents a velvety or villous appearance, and in some cases there is even ulceration with superficial necrosis. The muscular and elastic coats become atrophied, and, coincidently with this, dilatation increases. At times the wasting of these coats is partial; some portions of the walls retaining their natural volume, and forming bands or ridges elevated above the surrounding membrane. The dilated tubes occasionally present an appearance of hypertrophy; the walls are thickened, but the thickening depends on changes which have taken place in the mucous membrane. The cartilages resist the destructive metamorphoses longer than the other structures, but they sometimes partake of them. The contents of the tubes may be either muco-pus, or pus; and casts of the minute bronchi are met with. At times the contents are very foetid, and sometimes contain fragments of pulmonary tissue. It is said that the contents may become calcareous.

Dilatation of the bronchi may be unattended by any change in the surrounding lung-tissue, but, generally speaking, condensation of the latter takes place, as the result either of pressure or of chronic pneumonia. In some instances the tissue forms an abscess, in the centre of which the walls of the bronchus are found, while in others the walls of the bronchi and the surrounding tissue are destroyed by gangrenous inflammation. It is generally not difficult to distinguish between a phthisical cavity and a dilated bronchus. The latter is not characterised by the broken irregular surface which usually exists in the former; its shape is generally more regular; and it is usually continuous with bronchial tubes. The surrounding lung-tissue has no tubercular infiltration.

The *mechanism* of bronchiectasis has occupied much attention. It is probable that the elastic and muscular fibres lose their elasticity and contractility as the result of chronic inflammation, and thus yield

to the distending influence of coughing. When once a dilatation is produced, accumulation of the secretions takes place, which tends further to increase the dilatation.

SYMPTOMS.—The symptoms of bronchiectasis are those of chronic bronchitis aggravated in some important respects. The cough is frequent and paroxysmal. The expectoration is very abundant, very purulent, and, when the disease has lasted some time, very foetid. The expectoration of large quantities of purulent secretion at longer or shorter intervals is highly characteristic of this disease. The breath also becomes foetid. Hæmoptysis is occasionally met with, even to a considerable extent. There is more wasting than in ordinary bronchitis, and the blood is more imperfectly aerated. Night-sweats are not uncommon. In fact, the general symptoms approach those of phthisis. The digestive functions are usually not much impaired.

PHYSICAL SIGNS.—The movement of expansion is diminished in bronchiectasis, while that of expiration is prolonged. Over the affected portions of the lung there may be slight retraction. Vocal fremitus is increased, and rhonchal fremitus is sometimes well marked. The percussion note is altered. If a dilated tube is surrounded by condensed lung-tissue, or is full of secretion, there is dulness on percussion; but if it is situated near the surface and empty, some degree of tubular resonance may exist. Cracked-pot sound may be, at times, elicited. The respiratory sounds are harsh, or loudly bronchial with a more or less blowing character, and they may be distinctly cavernous. Vocal resonance is often greatly increased. The pulse becomes rapid in the later stages. The temperature rarely if ever reaches the height that it does in phthisis with cavities, and the daily oscillations are not so marked.

DIAGNOSIS.—The main difficulty as regards diagnosis is in the differentiation of certain cases of phthisis with cavities from bronchiectasis with large globular dilatations. The points to be relied on are that in the latter disease the morbid physical signs are usually met with at the middle and lower parts of the lungs, while in ordinary phthisis they are found at the apex; that the temperature differs in the two affections as mentioned above; that emaciation and night-sweats are not so marked in bronchiectasis; and that, if cases are watched, there is usually observed a progressive advance of symptoms in phthisis, while in bronchiectasis the symptoms may remain stationary. In phthisis signs of consolidation precede those of cavities, while they follow them in bronchiectasis, and in the latter disease tubercle-bacilli are absent from the sputa. Bronchial dilatations and tubercular cavities have been found in the same lung. The foetor of the breath and sputa in bronchiectasis may cause a suspicion that gangrene of the lung is present; but the general symptoms will usually enable the practitioner to differentiate between the two affections.

PROGNOSIS.—Bronchial dilatation is probably never cured. It may last for years.

TREATMENT.—The treatment of bronchiectasis must be that of chronic bronchitis with the use of such measures as are applicable to wasting diseases in general. The foetor of the breath is best relieved by the inhalation of creasote, or some similar antiseptic. The disease has, in some cases, been treated successfully by incision and drainage.

5. Narrowing or Obstruction.—Narrowing or obstruction of the bronchial tubes is by no

means uncommon, and may depend on intrinsic or extrinsic causes. Complete obliteration of a tube is sometimes found in connection with bronchiectasis, immediately beyond a globular dilatation.

ÆTIOLOGY.—The *intrinsic* causes of obstruction are a thickening of the mucous membrane resulting from bronchitis; the cicatrization of tertiary syphilitic ulcers; the retention of viscid secretions; the exudation of plastic material into the interior of the tubes; and the deposition therein of tubercle or cancer.

Among the principal *extrinsic* causes are the pressure of adjacent tuberculous or cancerous deposits; the contraction of plastic matter exuded into the tissues surrounding the tubes; solid formations in the pleura; enlarged bronchial glands; and aneurysmal and other thoracic tumours.

Obstruction is most frequently met with in the smaller tubes, but the pressure of thoracic tumours not infrequently causes obstruction, or even obliteration of a main bronchus, which occasionally—as in the case of aneurysms—becomes perforated.

SYMPTOMS.—If a large bronchial tube become suddenly and greatly obstructed, dyspnoea of an urgent character sets in, and death from asphyxia may speedily result, unless the obstruction be removed. When the obstruction is on a smaller scale, being confined to the smaller tubes, or when a large tube suffers only from slow, gradually increasing obstruction, the symptoms are by no means urgent for a time, and slight dyspnoea, sometimes accompanied by stridor, is the most marked feature.

PHYSICAL SIGNS.—Complete obstruction of a bronchial tube invariably leads to collapse of the portions of the lung to which the tube is distributed, and thus an entire lung may collapse if its main bronchus be obliterated. Where partial collapse is produced, emphysema of the neighbouring lung-tissue commonly follows, and if one lung become collapsed, the opposite lung becomes enlarged and emphysematous. The existence of collapsed lung gives rise to dullness on percussion over the affected part, unless this be situated away from the chest-walls, or masked by the presence of emphysema. Further, obstruction of the bronchi causes a weakness or deficiency of the respiratory sounds, with a prolonged expiratory murmur, attended at times by sonorous and sibilant rhonchi. Over the collapsed portion of the lung, or over portions of the lung supplied by a tube which has become completely obstructed, the breath-sounds are absent. Deficiency or absence of vocal vibration is another physical sign of obstructed bronchial tubes.

TREATMENT.—The treatment of obstruction of the bronchi must depend on the nature of its cause. The chief interest of the affection arises from the means of diagnosis of thoracic tumours which it may afford.

6. Cancer.—Cancer of the bronchial tubes occurring independently of cancer in the lungs, or mediastina, is probably never seen; but cancerous matter has been found in the tubes: (1) in cases where the lungs have been infiltrated with a similar deposit; (2) where a cancerous tumour connected with the root of a lung has perforated a tube; and (3) in some cases of cancerous disease of the lung, a tumour of a similar nature being found connected with the mucous membrane of a tube.

(4) Cancerous matter has also been found *in transitu* in a tube, having been detached from a cancerous mass.

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BRONCHIAL GLANDS, Diseases of.—

SYNON.: Fr. *Adénopathie Trachéobronchique*; Ger. *Krankheiten der Bronchialdrüsen*.

GENERAL ANATOMY.—The bronchial glands, about twenty-five in number, receive the lymphatic vessels from the lungs, bronchial tubes, and visceral pleura. Some, often called the *tracheal glands*, are situated around the lower end of the trachea; others, including the largest of all, lie immediately below the tracheal bifurcation, while smaller ones are placed along the two main bronchi. In addition, a number of still smaller glands, about forty on the right side and thirty on the left, lie actually embedded in the lung substance near its 'root,' between the divisions of each main bronchus to the third or fourth ramification; these are sometimes spoken of separately as the *pulmonary glands*.

While anatomically the term 'bronchial' is restricted to these groups, it frequently happens that when they are diseased others in the thorax are involved also, especially the *superior mediastinal or cardiac glands*, an important group situated in the upper part of the interpleural space, in the neighbourhood of the arch of the aorta and continuous below with the bronchial glands proper. Disease of this group may cause some of the symptoms and physical signs commonly attributed to enlargement of the bronchial glands only.

It is also of importance to recollect that some lymphatic vessels from the posterior mediastinal glands pass into the bronchial group. As the former receive lymphatics from the diaphragm, a channel thus exists whereby infection, as in tuberculosis, may be conveyed from the abdomen to the bronchial glands. These glands, especially if those of the superior mediastinum be included, are in close relation to a large number of important structures: the trachea and main bronchi; the pulmonary vessels; the innominate veins and superior vena cava; the arch of the aorta; the vagus nerves and their recurrent branches—especially on the left side; the pericardium; the œsophagus; and the adjacent nerve-plexuses. The bronchial glands are relatively larger in children than in adults, the largest being about the size of a small almond. In infancy they are of a pale red or pinkish colour, but in a few years become studded with dark spots, and in later life, especially in town-dwellers, are often almost black from inhaled particles of carbon. As the largest glands, and those most liable to become considerably hypertrophied, are grouped round the lower end of the trachea, it is important to bear in mind that the tracheal bifurcation corresponds on the surface with the junction of the manubrium and body of the sternum in front and with the fourth dorsal spine behind.

MORBID ANATOMY AND PATHOLOGY.—The lymphatic glands throughout the body are most active in childhood; greater functional activity involves greater proneness to disease, at least to inflammatory processes of all kinds; and in accordance with this general rule we find that diseases of the bronchial glands are met with especially in children.

The chief morbid processes affecting the glands are the following :

1. *Lymphadenoma*.—In this disease the bronchial glands, along with the other groups in the thorax, are often involved, though usually after those situated externally. The enlargement may be very great. In the spleno-lymphatic or rarer form of lymphadenoma the bronchial glands frequently present a moderate degree of hypertrophy. See LYMPHADENOMA.

2. *Mediastinal growths*.—Lympho-sarcoma when commencing in the thorax usually starts in the bronchial glands and may give rise to an extreme degree of enlargement. Carcinomatous deposits, nearly always secondary, also occur.

3. *Syphilis*.—In both the secondary and tertiary stages, this often causes swelling of the lymphatic glands generally, in which the bronchial glands participate. The latter are most hypertrophied in cases of tertiary syphilis affecting the lungs, in which enlargement of the glands up to the size of a pigeon's egg has been recorded.

4. *Congestion and Inflammation*.—In practically all persons, but most of all in children, dying from acute bronchitis, pneumonia, or broncho-pneumonia, the bronchial glands are found swollen, hyperæmic, and somewhat softer than normal; in extreme cases they may be pale from excessive cell-proliferation. This, of course, is merely an example of the general law that acute inflammation in any part of the body tends to cause swelling of the associated lymphatic glands. The enlargement is most marked in cases of measles and whooping cough, especially in the large gland placed exactly in the tracheal bifurcation. The peculiar cough of pertussis has been attributed to the pressure of this and of other glands, but, inasmuch as an equal or greater degree of swelling may be met with in cases in which there has been no whoop, this explanation can hardly be regarded as adequate. The mistake has probably arisen from the fact that enlarged bronchial glands may give rise to a spasmodic cough which more or less resembles that of pertussis. See WHOOPING COUGH.

5. *Chronic Hypertrophy*.—Chronic hypertrophy of the bronchial glands is apt to be a sequel of the inflammatory condition just described, if this is prolonged by repeated catarrhs or by chronic pulmonary disease. Its chief importance lies in the fact that it undoubtedly predisposes, at least in early life, to tubercular lesions in the glands. The course of events is probably precisely similar to that occurring in the cervical glands, in which a chronic adenitis is maintained by some simple irritant, such as chronically enlarged tonsils, or a carious tooth, until the chance arrest of tubercle-bacilli in these unhealthy glands results in definite tuberculosis.

6. *Suppuration*.—Suppuration of the bronchial glands is a rare event, and the conditions under which it occurs are not very clear. Probably it is most frequently associated with the presence of tuberculosis; very rarely it may be secondary to acute pneumonia.

7. *Caseation*.—Caseation of the bronchial glands is found in a large proportion of autopsies upon cases of ordinary pulmonary tuberculosis. There is no need to describe the process in detail; small scattered milium granulations appear, enlarge, become caseous, and gradually coalesce, until

entire glands are converted into caseous masses, generally—though not necessarily—with considerable enlargement. In adults this is a purely secondary process and rarely a matter of clinical importance, any symptoms to which it may give rise being overshadowed by those due to the primary lung-disease.

Caseation of the bronchial glands in children is, however, of far greater importance, and three special features call for notice.

(a) *Its great frequency*.—In children who have died with tubercular disease in any part of the body, the bronchial glands are usually affected, just as in adults the lungs rarely escape; and as statistics from the *post-mortem* rooms of the Children's Hospitals in London prove that fully one-third of the cases show evidence of tuberculosis, it is easy to see how extremely common caseation of these glands must be. Out of 120 autopsies on children presenting tubercular lesions, the writer found caseation of the bronchial glands in 96 (80 per cent.), and a more careful examination of some of the remaining cases would probably have still further increased this number. Some observers state that caseation of the mesenteric is more frequent than of the bronchial glands, but this certainly does not conform with the experience of the London Hospitals.

(b) *Its occurrence independently of lung disease*.—Caseation of the glands is frequently present when no pulmonary lesion whatever is to be found: a complete contrast to what usually obtains in adults. This primary involvement of the glands may conceivably occur in four different ways, of which the first is probably the most common.

(i) It is now definitely recognised that tubercle-bacilli may pass through the pulmonary alveoli without producing any lesion, and subsequently lodge in the glands, where they give rise to characteristic changes. Unquestionably a similar primary infection of the mesenteric glands may be set up by bacilli which have passed through the intestinal mucous membrane. (ii) The bacilli may enter by the intestine and infect the bronchial glands indirectly *via* the posterior mediastinal glands, as previously described. (iii) The bacilli may enter by the tonsils and pass *via* the cervical and tracheal to the bronchial group of glands. (iv) Possibly a few bacilli having gained access to the blood, no matter how, may lodge and develop in the bronchial glands, merely because these happen to present a suitable soil, just as an injury to a joint under similar conditions may be followed by tubercular arthritis. In this case the lodgment of the bacilli is facilitated by the glands being in a condition of chronic engorgement and hypertrophy, with stagnation in the lymph-channels passing through them. Hence it is that measles, pertussis, and any chronic catarrhal condition of the bronchial tubes or lungs in children so greatly predispose to the development of tubercular changes in the bronchial glands.

(c) *Its varied and serious secondary consequences*.—The most important are the following:—

(i) *Ulceration into adjacent parts*.—Around the bronchial glands, as around others, when caseous, some periadenitis is often set up, leading to adhesions between them and neighbouring structures. At the same time the caseous material may gradually soften and eventually ulcerate through into adjacent parts. Naturally, from the position of

the glands, perforation occurs most frequently into the trachea, near its bifurcation, or into one of the main bronchi, commonly the right. The caseous matter is then either gradually expectorated or else sets up a diffuse broncho-pneumonia or tuberculosis. Occasionally the gland enters the trachea suddenly *en bloc* and causes rapid asphyxia and sudden death—a somewhat rare event in childhood. The glands may also ulcerate into the oesophagus, and sometimes a communication is established between the latter and the air-passages, diffuse gangrene of the lungs resulting. In fact gangrenous abscess of the lung in children is more frequently produced by caseous bronchial glands than by any other cause except a foreign body. Very rarely the glands may ulcerate into the pericardium, the pulmonary artery, the aorta, or the mediastinum; in the latter case giving rise to a mediastinal abscess, which may point externally. It has already been suggested that some of the recorded cases of suppuration of the bronchial glands may have been primarily instances of caseation.

(ii) *Secondary extension to the lungs*.—In young children the most usual origin of pulmonary tuberculosis seems to be by extension from caseous bronchial glands. In these cases there is advanced caseation of the bronchial glands, especially of the so-called pulmonary group which lie embedded in the lung-substance. They soften and discharge their contents into the bronchi, leaving small cavities; the process being just the same as the ulceration into the trachea already described, and, as in the latter, the inhalation of the infective products into the bronchioles may lead to an acute diffuse tuberculosis of the lung. More commonly however the disease advances slowly into the adjacent pulmonary tissue, until at last the lung, or it may be one lobe, for some distance round its root, is converted into a caseous mass riddled by small cavities; further out isolated caseous nodules are met with, evidently of more recent formation, while the apex and peripheral parts of the lung may be healthy or contain only scattered tubercles. In a series of autopsies every intermediate step can be traced, from the commencement in the glands onwards to complete destruction of the lung.

The difficulties in diagnosis which such cases may present, especially in the earlier stages, are obvious, and are increased by the fact that occasionally narrowing of a main bronchus by enlarged glands may lead to bronchiectatic changes in the lung behind the obstruction.

(iii) *General blood-infection*.—A softening mass of caseated tubercles is always liable, by means of the blood-stream, especially in children, to give rise to acute general miliary tuberculosis. While in adults the infective tubercular focus is generally found in the lung, in children it is usually situated in the bronchial glands, one or more of which are caseous and softened. So far as the writer's personal observations go, in 44 autopsies upon cases of tubercular meningitis in children, specially examined with the object of determining the primary focus of infection, the bronchial glands appeared to be the primarily affected part in more than half the number.

(iv) *Obsolescence*.—In a large number of cases, fortunately, caseous bronchial glands dry up, and become surrounded by a firm fibrous capsule, while the caseous contents often undergo calcification—similar changes to those which may occur in

other tuberculous organs. Such glands may remain quiescent for an indefinite number of years, though probably still potentially capable of fresh activity.

Of the above-mentioned causes of enlargement of the bronchial glands the first two are described elsewhere (*see* LYMPHADENOMA; AND MEDIASTINUM, Diseases of); the subsequent parts of this article will therefore deal mainly with caseation of the glands and non-infective enlargement.

SYMPTOMS.—(a) *General*.—Active tuberculosis in the bronchial glands, as in any other part, causes fever of a hectic type, with wasting and general malaise. When the disease is no longer active symptoms will of course subside.

(b) *Local*.—Very considerable enlargement of the glands may exist without giving rise to any clinical phenomena, and it is only in extreme cases that any of the symptoms to be presently enumerated are met with. Symptoms are most typical when the enlargement is due to lymphadenoma or lympho-sarcoma. *See* LYMPHADENOMA.

1. *Cough*.—This is the most frequent symptom. It varies in character. Its occasional resemblance to that characteristic of pertussis has already been referred to. When due to disease of the glands, the crowing inspirations are less marked, the paroxysms shorter, the invasion and period of accession of the cough less regular in their sequence, while the fever and emaciation are more distinct.

2. *Expectoration*.—Some expectoration of mucus from slight bronchial catarrh is not uncommon. It is likely to be abundant should the enlarged glands compress the pulmonary veins and so lead to passive congestion and oedema of the lungs. When softening glands ulcerate through into the trachea, thick pus-like expectoration may be coughed up, and the case may even be mistaken for an empyema which has ruptured into the lung.

3. *Hæmoptysis*.—There may be slight, severe, or even fatal hæmoptysis. This may be due to ulceration into the air-passages, to erosion of vessels in the lung, or to congestion from glands compressing the pulmonary veins. In a few cases a large vessel has been perforated as well as the trachea or main bronchus, so that profuse hæmorrhage has taken place directly into the latter.

4. *Pain*.—In a considerable number of cases pain is complained of and is usually referred to the back, at the level of the fourth and fifth dorsal vertebrae, occasionally to the front, behind the upper end of the sternum. Sometimes there is associated tenderness.

5. *Dyspnœa and stridor* may be caused by direct pressure of the enlarged glands on the trachea or one of the main bronchi. Dyspnœa may also be brought about indirectly by pressure on the vagus or recurrent laryngeal nerves, causing laryngeal spasm or palsy, perhaps with hoarseness or aphonia.

The question naturally arises here what relationship, if any, exists between enlargement of the bronchial glands and asthma. Some writers go so far as to state that most cases of 'asthma' in early life are due to direct pressure of enlarged glands upon the air-passages. They may indeed, as just explained, cause dyspnœa in this way, and such dyspnœa may be more or less paroxysmal in character; or they may indirectly, by pressure on the vagus nerve, give rise to attacks simulating asthma; but dyspnœa so produced has no more claim to the name of asthma than has that due to the pressure of an aneurysm. True asthma is essentially a neurosis: the actual attack

may be excited by a great number of causes, and it is possible, even probable, that one of these causes in a predisposed subject may be enlarged bronchial glands, adherent to or pressing upon and irritating (without necessarily narrowing) the walls of the trachea or bronchi. Therefore when in any particular case of asthma, especially a child, we are trying to discover the actual exciting cause of the attacks, the possibility of enlargement of the bronchial glands should not be overlooked. They may not be so common a cause as gastric irritation or bronchial catarrh, but at any rate they should not be ignored, when such remote factors as nasal polypi, uterine disorders, or even emotions are looked upon as conceivable excitants of the attacks.

6. *Dysphagia*.—Occasionally dysphagia is produced by compression of the œsophagus.

7. *Vomiting*.—Vomiting has been attributed to pressure on the vagus nerves, but is perhaps more often due to the severity of the cough.

PHYSICAL SIGNS.—Considerable enlargement of the bronchial glands may exist without giving rise to any definite physical signs. Those which may be met with are the following.—1. *Inspection and Palpation*.—Signs of venous congestion are not uncommon from pressure of the glands upon the innominate veins or superior vena cava. There is often marked visibility of the cutaneous veins over the upper part of the chest, especially about the manubrium sterni, and sometimes actual puffiness or œdema of the face and neck. Obstruction of one bronchus may lead to deficient expansion or to actual flattening of the affected side; general recession of the soft parts of the chest-wall may be caused by tracheal obstruction.

2. *Percussion*.—The enlargement of the glands may be sufficient to give rise to deficient resonance or actual dulness; this is most frequently present over the manubrium and at the inner end of the first and second intercostal spaces, also occasionally in the interscapular region, at the level of the fourth or fifth dorsal vertebrae, at any rate in cases of enlargement from lymphadenoma or new-growth, but very rarely when the glands are simply caseous.

3. *Auscultation*.—Breathing may be bronchial over the dull area. If a main bronchus be compressed the breath-sounds will be weak over the corresponding lung and perhaps proportionately loud over the other lung. Eustace Smith lays much stress on the significance of a venous hum heard over the manubrium when the head is thrown back as far as possible; he attributes it to the enlarged glands compressing the left innominate vein, the retraction of the head tilting forward the lower end of the trachea and with it the glands. Undoubtedly this sign is frequently present in children, but whether it always indicates glandular enlargement is as yet unsettled.

DIAGNOSIS.—Various diseases with which enlargement of the bronchial glands may be confused have already been referred to. When the enlargement is extreme the diagnosis is an easy matter, for the symptoms and the physical signs may alike be characteristic. The really important question is what are the points to which attention should be directed in endeavouring to arrive at a diagnosis in a doubtful case, when, as in tubercular disease, the enlargement is not great and yet may be important to recognise. Not only may physical signs and local symptoms alike be absent, even when the glands are extensively diseased, but nearly all the

symptoms and some of the physical signs before enumerated may be due to lung-disease alone. Now we have seen that such disease is very apt to follow caseation of the glands, and although when this has taken place the condition of the latter becomes of secondary importance, it is important to be able to recognise that they are enlarged before the serious consequences have supervened.

No one sign or symptom is ever pathognomonic, but a very suggestive combination of conditions is (a) enlargement of cutaneous veins over the top of the chest, (b) some deficiency of resonance over or on either side of the manubrium, and (c) a venous hum in the same position when the head is retracted; especially if these occur in a thin delicate-looking child, suffering from a cough, for which no cause can be detected in the throat or lungs, and from slight intermittent evening pyrexia without assignable cause. Enlargement of glands elsewhere, especially in the neck, may afford further help in diagnosis. Skiagraphy is unlikely to be of much service in cases where other indications are absent, but where they establish a probability, it may afford confirmatory evidence.

In children it is very common to meet with slight deficiency of resonance just to the left of the manubrium, without any other indication of disease, and the presence of the thymus as a cause of impairment must not be forgotten. When at its maximum development, at two years of age, this organ occupies the superior mediastinum behind the sternum as far as the fourth rib-cartilage; its two lobes are generally of unequal size, sometimes the right and sometimes the left being the larger.

Bearing in mind the large number of children who, as shown by subsequent *post-mortem* examination, suffer from tuberculosis of the bronchial glands, and the great probability that this has caused, at some time or other, fever and malaise, we should make it a rule in children with definite emaciation or persistent unexplained evening pyrexia, not to be content with a diagnosis of gastro-intestinal catarrh, but to recollect that the symptoms may be due to caseation of the bronchial glands, especially if the child has recently suffered from measles, pertussis, or bronchitis.

PROGNOSIS.—Calcified glands may remain perfectly quiescent for the remainder of the patient's life, especially if he is placed under favourable hygienic conditions. It is moreover probable that complete arrest may occur even when glands have ulcerated into the trachea or bronchi; but if tuberculosis once extend to the lungs a fatal result will probably ensue. At the best, however, a caseous mass in any part of the body is always to be regarded, like a chronic otorrhœa, as a potential source of danger. Tubercle-bacilli may retain their vitality in it for an indefinite period, and should anything occur to impair the general health, the enveloping fibrous capsule may break down, and either local or general tuberculosis follow.

TREATMENT.—This must be conducted on exactly the same general lines—climatic, hygienic, dietetic, and medicinal—as in dealing with tuberculosis in other parts of the body. It is especially for tuberculosis of the lymphatic glands in children that the watering-places on the north-east coast of Kent seem peculiarly suitable. Every effort must be made to improve the child's general health and nutrition and to check any tendency to catarrh of the respiratory passages, in order to allow of the

subsidence of the congestion and hyperplasia of the glands resulting from it; or, if caseation have occurred, to place the patient in the best possible position for the encapsulation and calcification which offer the only prospect of an even imperfect cure. If this result follow, it still remains equally important to maintain the health at as high a level as possible, remembering that any failure in this may result in recrudescence and extension of the local disease. Cod-liver oil and syrup of the iodide of iron or tincture of iodine internally, with local counter-irritation by iodine externally, are to be especially recommended. Should there be the least suspicion that the glandular enlargement is of syphilitic origin, a suitable line of treatment must of course be adopted. See CLIMATE, Treatment of Disease by.

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BRONCHIECTASIS.—See BRONCHI, Diseases of.

BRONCHOCELE (βρόγχος, the throat; and κήλη, a tumour).—A synonym for goitre. See GOÏTRE.

BRONCHOPHONY (βρόγχος, the throat; and φωνή, the voice).—The resonance of the voice, as normally heard on auscultation over those parts of the chest which correspond with the main bronchi, and, in certain morbid conditions, beyond these situations. See PHYSICAL EXAMINATION.

BRONCHO-PNEUMONIA.—A synonym for catarrhal pneumonia. See LUNGS, Inflammation of.

BRONZED SKIN.—A term generally used to denote the peculiar discolouration of the skin associated with Addison's disease. See ADDISON'S Disease.

BROW-AGUE.—A synonym for frontal neuralgia, or tic-douloureux. See MALARIA; NEURALGIA; and TIC-DOULOUREUX.

BRUIT (Fr. *bruit*, a noise).—A word used to designate various abnormal sounds heard on auscultation, in connection with the heart or vascular system. See PHYSICAL EXAMINATION.

BUBO (Βουβών, the groin).—SYNON. : *Apostema inguinis*; *Dragoncelus*; Fr. *Bubon*; Ger. *Leistenbeule*.—Primarily, as synonyms show, a swelling of an *inguinal* gland, usually of venereal origin. The term is now practically limited (1) to glandular swellings secondary to chancres and chancroids (genital or extragenital), or to gonorrhoea; and (2) to glandular swellings, often inguinal, in 'bubonic' plague. See PLAGUE.

BUBO, VENEREAL.—DEFINITION.—An inflammatory swelling of one or more glands, usually inguinal, due either to chemical or to infective irritants absorbed from a primary venereal lesion, usually genital.

VARIETIES.—Buboes are divided into :—1. Simple bubo; 2. Chancroid or virulent bubo; and 3. Syphilitic bubo. The gland first affected is always that in most direct communication with the sore. When an acute inflammation is excited the irritant does not, as a rule, pass from the gland first affected to another more remote in the series. On the other hand, the more slowly acting irritant of

syphilis affects gland after gland of a group till all are involved.

Buboes occur most frequently on the side of the body occupied by the source of irritation; they may occur simultaneously on both sides, and may be even limited to the side opposite to the position of the exciting sore. When buboes form in both groins, there is usually a sore on each side or one at the mesial line, which infects lymphatics passing to each groin. When the gland affected is on the opposite side to the sore, there is an unusual distribution of the lymphatics. Bubo, including simple and virulent, occurs in about thirty per cent. of chancres; how often with urethritis and other lesser venereal affections is not known.

1. **Simple Bubo.**—This variety may be either non-suppurating or suppurating. In either case it may be caused by the introduction of pyogenic organisms into the lymphatics, whence they are conveyed to the glands; or it may be due to chemical irritants produced by organisms at the primary lesion. Simple venereal buboes are thus pathologically identical with cases of non-venereal lymphadenitis due to absorption of similar irritants from infected wounds, scratches, fissures, boils, herpetic and other erosions; for in this instance the venereal lesions—whether balanoposthitis, anterior urethritis (gonorrhoeal), chancroid or ulcerating chancre—merely serve as foci whence the above-named common irritants are absorbed. Whether the primary focus be non-venereal or venereal in origin, absorption from it and consequent suppuration of glands are much favoured by dirtiness of, and retention of discharge in contact with, the primary lesion.

It is important to remember that the inguinal glands receive their lymph-supply from the penis (a few deep vessels pass behind the pubic arch), scrotum, perineum, anus, skin of the buttock and of the lower abdomen, and in the female from the vulva. If the starting-point of the infection cannot be found upon the genitals, the other regions from which the glands are supplied must be carefully examined. It is possible that the infecting virus may be absorbed by the lymphatics without producing a sore on the surface (*bubon d'emblée* or primary bubo).

SYMPTOMS.—There are two forms of simple bubo :—1. Slight swelling and tenderness of one or more glands, ending in a few days by resolution. This occurs most frequently in urethritis, balanoposthitis, or simple chafings. In genuine chancroid the next and more serious form is most frequent. 2. Swelling, commonly of one, seldom of several glands; brawny thickening of the surrounding tissues (periadenitis); redness of the integuments; great tenderness and pain, especially when walking. In a few days this condition terminates either by abscess, the most frequent culmination, by gradual subsidence to the normal state, or by chronic induration of one or more glands. When the bubo suppurates, the matter, instead of pointing at once, may burrow in various directions, and burst through the skin at various spots, leaving very chronic sinuses. Occasionally, in a mass formed of many glands, abscess follows abscess and fresh sinuses undermine the skin. The patients are often in feeble health, and it is said that the glands have become tuberculous: bacteriological or even histological proof of this does not seem to have been given.

PROGNOSIS.—This form of bubo is seldom dangerous, but when chronic suppuration of several glands and burrowing occurs, it is very tedious and exhausting to the patient.

2. Black Chancroidal Bubo.—**Virulent Bubo.**—**ÆTIOLOGY.**—The exciting cause of lymphatic abscess secondary to chancroids is unknown and is likely to remain so until the cause of the chancroid is discovered. In some cases ordinary pyogenic organisms have been found; such have been included under the previous heading. In the majority of such abscesses which have been examined no specific organisms have been found, but it seems premature to conclude that the suppurative is due to a chemical irritant absorbed from the ulcer. The development of a bubo is favoured by exercise, unclean linen, and retention of discharge by scabbing or phimosis. Size and multiplicity of sores seem to have little effect. It is most common in the second to the fourth week, but may be almost simultaneous with the chancroid, or occur even after this has healed. It is much less common in women than in men. Perhaps 30 per cent. of hospital cases develop an abscess. The great majority of these run the course of a simple bubo; exceptionally, two or three days after incision, one proves itself chancroidal—in appearance and in inoculability. This form is never a consequence of gonorrhœa or of a true syphilitic chancre, but only of a chancroid. It may be generated in two ways: 1. by the infecting of a simple open bubo with chancroidal pus; and 2. by the entrance of the chancroidal virus into the lymphatics leading from the chancroid to the gland most directly connected with the sore. The first view is probably correct.

SYMPTOMS.—Chancroids being rarely extra-genital, buboes secondary to them, whether simple or virulent, are usually inguinal. Both varieties may occur elsewhere. At first the symptoms of specific bubo are those of acute abscess forming rapidly in and round a single gland. Thus far they differ nowise from those of simple bubo. Two or three days after bursting the simple suppurating cavity becomes a spreading ulcer, which rapidly destroys the skin and exposes the base of the cavity. A large amount of matter escapes; it is thin, yellowish-grey, or yellowish-red, with shreds of a chocolate colour floating in it.

The further progress varies. In the least severe variety the edges of the skin ulcerate irregularly for a short distance, then thicken and begin to granulate; the floor of the ulcer loses its unevenness and rises up to the level of the skin; and cicatrization follows. But commonly a much greater destruction of tissue is effected. The skin is eaten away into wide gaps; long channels form under it or dive deeply among the great vessels. In other cases the ulceration produces a large shallow sore. The contagious condition of these buboes often continues for weeks or months, so that consecutive inoculation of the skin is not infrequent. The characters of the fully formed virulent bubo are those of the chancre. The surface is greyish-brown, dug-out, 'worm-eaten'; the borders at some parts are thickened, everted, and pared away, at others sharply eroded or undermined and curled in. The matter is thin, shreddy, plentiful, reddish in colour, and inoculable. Like the chancre, the virulent bubo passes through periods of *extension*, *arrest*, and *repair*. The virulent, as well as the simple bubo, is liable to erysipelas and gangrenous sloughing.

The *duration* of virulent bubo for the reason stated is wholly indefinite—in a few cases ending in a week, in many cases lasting for months.

DIAGNOSIS.—The virulent bubo has in its early stage no distinguishing mark from the simple acute bubo. After it has become inoculated with the contagious matter it is distinguished from every other affection by the characters already described. Nevertheless, in some cases the signs of virulence are so feebly marked throughout that the diagnosis remains uncertain. This difficulty may be increased by the primary sore having healed before the suppurative of the bubo, or by its being hidden in some unusual position (urethra, anus).

PROGNOSIS.—This is not always grave. Virulent buboes often reach cicatrization in a few weeks, and meanwhile cause no serious inconvenience. On the other hand, if the patient have suffered from syphilis some years previously, or be in depressed health from alcoholism, diabetes, albuminuria, and the like, phagedæna may attack the ulcers, and thereby permanently cripple the patient, or even destroy life by hæmorrhage or sepsis.

3. Syphilitic bubo.—*Indolent multiple bubo; pléiade ganglionnaire.*—This enlargement of the group of lymphatic glands in nearest connection with the initial sore is an almost constant symptom of early syphilitic infection. In this it differs much from the bubo of chancre or gonorrhœa, in which affections the bubo is more often wanting than present. Three conditions have been noted to exist in patients in whom the enlargement of the glands could not be detected—namely: 1. Phagedæna of the sore. 2. Obesity; in such persons the lymphatic system as a rule is small. 3. Scantiness of the induration of the primary sore, the sore itself being ill-developed. But these exceptions are most rare. Fournier gives five instances only of absence of indolent multiple bubo in 265 cases of hard sore, accompanied by well-marked general syphilis (2 per cent.). In 176 similar cases noted by Berkeley Hill three had no apparent inguinal enlargement. One of the patients was a very fat man, and in another the initial sore was only slightly hardened. The enlargement of the glands is first perceived about eleven days after the induration of the sore, though probably the affection commences at the end of the incubation of the poison. In extremely rare instances enlargement is delayed until the third or fourth week after the induration of the sore. When patients acquire both a genital and extra-genital initial lesion simultaneously or nearly so, not only the glands of the groin, but also those of the extra-genital region are enlarged.

The seat of these buboes is, in all genital and perigenital cases, the inguinal glands. The glands of other regions—epitrochlear, axillary, sub-maxillary (sub-mental), cervical and nuchal (pre-auricular)—are all occasionally found primarily enlarged in extra-genital cases.

SYMPTOMS.—The glands are enlarged in both groins, those on the side of the sore being most affected. In a few cases the enlargement is limited to the glands situated on the same side of the body as that of the sore, and in rare instances to those of the side opposite to that of the sore. The enlargement affects first the gland nearest to the lesion, and then rapidly involves the other members of the group. The patient usually makes no complaint of the buboes. Each of the affected glands is found to be swollen, but is rarely larger than an almond or hazel-

nut ; it is of gristly hardness, insensitive to pressure, isolated from, and mobile among, its fellows, there being no periadenitis ; the skin over the glands is quite normal. Such glands are often spoken of as 'amygdaloid' or 'bullety.' The gland in most direct communication with the sore is most affected ; in rare exceptions only a single gland is enlarged. Great enlargement of the gland does not always attend marked induration of the sore, nor is slight induration of the sore always accompanied by small swelling of the glands, though commonly this is the case. So also, though absence of tenderness is the rule, the glands may be slightly painful if pressed. Again, though the glands remain distinct in most cases, they have been known to coalesce into a single mass, which becomes fixed to the fascia. In many patients the dorsal lymphatic vessel of the penis becomes indurated sufficiently to be easily distinguished under the skin.

These buboes reach full development in one or two weeks, and remain without apparent change for several weeks, or even for two or three months. Then they begin to diminish slowly, but are generally still evident in the fifth or sixth month after infection, and now and then even for years afterwards. In rare cases the enlargement vanishes in two or three weeks. The long duration of enlargement renders these buboes a valuable sign of constitutional syphilis when the primary sore has disappeared. Again, the conspicuous enlargement of a group of glands may indicate the place of entry of the syphilitic poison when that is hidden.

TERMINATIONS.—In most cases the glands revert to their natural state. Sometimes they degenerate into masses of fibrous tissue. Now and then suppuration takes place, not from the syphilitic virus, but from pyogenic irritation. In tuberculous persons it is said that tubercular inflammation may supervene upon the syphilitic. The glands enlarge still more, coalesce, and slowly soften.

DIAGNOSIS.—This is generally easily drawn from the character of the swelling, aided by the presence of other syphilitic signs (hard sore, rash on the skin, &c.). These buboes may be confounded with chronic inflammatory enlargement, but in such cases the history and attendant symptoms remove doubt.

PROGNOSIS.—Apart from its connection with syphilis, the prognosis is good. The only untoward termination is tuberculosis.

TREATMENT.—Prophylactic treatment demands thorough disinfection of the sore in all cases. When possible it is desirable to apply pure carbolic acid or the caustic, cocaine or β -eucaine being used.

1. *Simple acute bubo*, from whatever cause it may arise, is treated like an acute abscess. In the early stages, purgatives, rest in bed, and constant application of a cream made of equal parts of extract of belladonna and glycerine, hot poultices, fomentations, or rags soaked in lead-lotion with an india-rubber hot-bottle over them are requisite. All operations should be done with full attention to asepsis, and infection from the primary sore must be carefully guarded against. The dressings should be dry when speedy union is expected, but when there is much suppuration and periadenitis, boric-acid or corrosive-sublimate (1:2000) fomentations are best. When a sinus is present without much glandular swelling, rest, with fixation of the hip in a Thomas's hip-splint, and constant elastic compression by cotton-wool and a spica bandage over a moist and antiseptic dressing, should be fairly tried. If heal-

ing does not occur, or if when first seen there are multiple sinuses or enlarged glands, the sinuses should be opened up, the cavity curetted, and the affected glands dissected out, or scraped out and carbolicised. These operations necessitate large wounds, and bleeding from the first branches of the femoral artery may be troublesome : the femoral vein has more than once been wounded.

If glands remain inconveniently swollen in spite of rest, constant pressure, and counter-irritation, they must be dissected out.

Should pus not form, subsidence of the glands may be aided by pressure with a pad and spica bandage. When pus has formed, or as soon as it becomes evident that the above treatment is not succeeding, and before much periadenitis has arisen, the gland or glands should be carefully dissected out without infecting the tissues. If this is done the wound will rapidly heal. If the gland is adherent to its surroundings, or lies in a periglandular abscess, a vertical incision, opening the cavity fully, should be made and the pus evacuated. The cavity should then be lightly curetted ; the walls dried and painted with phenol ; and the wound plugged with iodoform gauze. Early evacuation checks the undermining of the skin and prevents burrowing.

2. The *chancreoidal bubo*, not being distinguishable from an ordinary simple bubo during the inflammatory swelling and consequent suppuration, requires the same treatment as that given above. When its chancreoid nature has become clear, it should be treated as a chancreoid with iodoform and corrosive-sublimate fomentations, frequently changed, all discharge being wiped away from recesses at each change. If necessary 10 per cent. cocaine may be applied to the surface, followed by pure carbolic acid which is allowed to soak in. Should this fail to check the ulceration, the patient must be anaesthetised, the whole surface curetted and carefully cauterised, the surrounding area thoroughly disinfected, and the parts dressed as above directed.

Phagedena.—When the ulceration by its rapid extension and obstinacy deserves this title, if there be any suspicion of syphilis, the patient should be brought under the influence of mercury as quickly as possible, by giving two or even three grains of blue pill thrice daily, or by the injection into the thick muscles of the trunk of some suitable mercurial solution, such as that of Raggazzoni (perchloride of mercury, 1 part ; iodide of sodium, 1 part ; distilled water, 64 parts : 8 minims being a dose). When the patient's general health is good, the ulceration seldom fails to yield to iodoform and fomentations, applied thoroughly in the manner directed. While the nocturnal gnawing pain continues, morphine should be given. The cessation of pain is a signal that the eroding action has stopped. When iodoform alone is insufficient, the continuous warm hip-bath must be employed. By it pain is at once arrested and healing soon set in motion. As continual immersion in a bath becomes extremely irksome, the ulceration may in many cases be arrested by keeping the patient in water for sixteen hours every day, and allowing the night to be passed in bed—in this interval the wound should be carefully packed with iodoform and fomented. When the ulceration is stopped, the bath may be discontinued, and iodoform alone used. If the water-bath fail, curetting and phenol or the caustic must be used.

The strength of the patient, generally much exhausted, should be restored by tonics, good diet, stimulants, and other general means.

3. The *syphilitic bubo* usually causes no trouble, and gets well during the course of treatment which is carried out for the cure of the general disease. If tenderness or aching occur, rest and a few warm baths are sufficient. If suppuration takes place the gland must be either enucleated, or incised and fomented. Should syphilitic glands become tuberculous and give rise to chronic abscesses, they should be dissected out and every attention should be paid to general hygiene. The treatment of syphilis must be continued.

BERKELEY HILL.

H. LITTLEWOOD.

BUHL'S DISEASE.—A synonym for Icterus Neonatorum.

BULB.—A synonym for the globe of the eye, and also for the medulla oblongata. Its adjective, 'bulbar,' is commonly restricted in application to the latter, and has for a considerable time been commonly thus employed, as in the terms 'bulbar symptoms,' 'bulbar nerves,' and the like. This use of the adjective has led to a more extensive employment of 'bulb' for the medulla oblongata, because the term 'medulla' is also applied to the spinal cord, the 'medulla spinalis,' as well as in its general sense of 'marrow.'

BULBAR PARALYSIS.—A synonym for labio-glosso-laryngeal paralysis; derived from the pathological relation of the disease with the bulb or medulla oblongata. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

BULIMIA (βοῦς, an ox; and λιμός, hunger).—Excessive or voracious appetite. See APPETITE, Disorders of.

BULLA (bulla, a bubble).—See BLISTER.

BURNS.—The morbid local effects produced by the direct application of excessive dry heat. See HEAT, Effects of Severe or Extreme.

BURSÆ MUCOSÆ, Diseases of.—Bursæ mucosæ are spaces in the connective tissue lubricated with a small amount of serous fluid, and situated at points exposed to repeated pressure or friction. Structurally they are composed of a layer of condensed connective tissue, fusing externally with the areolar tissue of the part, and lined internally with an imperfect layer of flattened endothelial cells, similar to those found in the pleura or peritoneum. Bursæ are normally present in many parts of the body; but new bursæ, equally perfect in their structure, may form at any part exposed to abnormal pressure and friction, as over the outer malleolus of a tailor, under an old corn, or over the head of the metacarpal bone of the great toe (*bunion*). Like the great serous cavities, bursæ are in direct communication with the lymphatics, and inflammatory products are consequently absorbed from them with great readiness, often giving rise to a diffuse inflammation of the surrounding areolar tissue, closely resembling phlegmonous erysipelas in appearance, and always accompanied by high fever.

Bursæ are liable to the following diseases:—

1. **Acute inflammation and suppuration**—**Acute bursitis.**—This is usually the result of

some more or less violent mechanical injury, some abrasion of the cuticle over the site of the bursa providing a means for the infection of its cavity with pyogenic organisms. This disease may occur in any bursa, but is most common in the bursa patellæ, in that over the olecranon, and in the small false bursa formed beneath an old corn, or in a bunion. The symptoms are those of acute inflammation generally, but the redness and swelling often extend a considerable distance up and down the limb. Thus, a drop of pus beneath a corn may cause œdema and redness to the knee. The febrile disturbance is usually proportionately severe.

TREATMENT.—Hot fomentations, and the application of glycerine and extract of belladonna (equal parts), may be of use. An early incision into the inflamed bursa is very desirable, and pus, if present, should be let out at once, for it may burrow extensively, especially about the knee. The treatment of the wound should be carried out on strictly antiseptic lines. The incision not uncommonly reveals the fact that there has been hæmorrhage into the cavity of the bursa.

2. **Chronic Bursitis—Dropsy of the bursa.**—This consists of an accumulation of serous fluid distending a bursa more or less tensely. The wall becomes somewhat thickened and opaque, but is otherwise unchanged. The contents are either fluid or jelly-like in consistency, clear, straw-coloured, and albuminous in character. The cause of the disease is usually repeated slight mechanical injury. The symptoms are merely those of a collection of fluid in the situation of the bursa, perhaps accompanied by a feeling of weakness in the neighbouring joint. There is no pain or tenderness. The commonest form of this disease is the so-called 'housemaid's knee,' but it is not infrequent in the bursa which lies in the popliteal space between the semi-membranosus muscle and the inner head of the gastrocnemius.

TREATMENT.—Avoidance of the mechanical injury, whatever it may be, which has caused the disease, is most important. A series of small blisters may be applied over the swelling; or, provided the bursa does not communicate with the neighbouring joint, it may be tapped and pressure then applied to it for a few days. If these fail, an incision may be made into the swelling with antiseptic precautions, and the bursa dissected out, or the closure of the cavity may be insured by the application of liquefied carbolic acid, or of a strong solution of zinc chloride, so as to destroy the lining. In the case of the popliteal bursa, which normally communicates with the knee-joint, if active measures are necessary, aspiration followed by pressure may be tried, or the bursa may be dissected out.

3. **Chronic enlargement of the bursa, with fibroid thickening of its walls.**—This affection is most common in the bursa patellæ, but may occur in that situated over the olecranon or in that over the tuberosity of the ischium. The bursa becomes converted into a dense fibroid mass of almost cartilaginous hardness. On section it is found to be composed of concentric layers of dense fibrous tissue. There is usually a small central cavity containing a little fluid. The cause of this change, as of simple dropsy, is repeated mechanical injury of a slight nature. In some cases it seems to be connected with syphilis. The only treatment is removal by the knife.

4. **Chronic enlargement of the bursa, with the presence in it of the so-called melon-seed bodies.**—In this form of disease, in addition to some thickening of the wall and accumulation of fluid in the bursa, small oval, flattish, smooth bodies of a white colour are found floating freely in its interior. These are similar in nature to those found in some forms of ganglion (*see* **GANGLION**). This condition is recognised by the peculiar soft crackling feeling perceived on palpation, combined with the ordinary symptoms of an enlarged bursa.

TREATMENT.—The affected bursa may be dissected out; or the alternative treatment given under 'chronic bursitis' may be applied.

5. **Tubercular disease.**—The bursa most often affected is that over the great trochanter of the femur beneath the gluteus maximus muscle. The cavity may be filled with tuberculous granulation-tissue, or a layer of this tissue may line the cavity, and the latter may then become distended with semi-fluid products resulting from progressive necrosis of the surface of the lining membrane and from serous exudation.

TREATMENT.—If not too large, the bursa may be dissected out. Or it may be laid freely open and its cavity thoroughly scraped out so as to

remove the whole of the lining membrane. The fibrous sac remaining should be treated with liquefied carbolic acid or with strong zinc-chloride lotion and the wound subsequently sutured with or without drainage, according to circumstances.

6. **Syphilitic disease.**—Effusions into bursæ in secondary syphilis are rare. Gummata of bursæ (most frequently of the bursa patellæ) are much more common. The gumma usually supervenes upon the condition described under section 3, and both knees are often affected. The cavity of the bursa becomes filled with the inflammatory products, and these subsequently undergo necrosis. The skin over the summit of the bursa becomes adherent and may give way later. Thus is produced a characteristic punched-out cavity with undermined edges and with a yellow adherent slough on its floor.

TREATMENT.—The bursa should be excised, and appropriate constitutional treatment adopted.

G. B. MOWER WHITE.

BUXTON, in Derbyshire.—Simple thermal waters. *See* MINERAL WATERS.

BUZIAS, in Hungary.—Strong muriated chalybeate springs. *See* MINERAL WATERS.

C

CACHEXIA (*κακός*, bad; and *ἔξις*, condition).

The term Cachexia is applied to the state of general constitutional disturbance produced by some grave disease. It is most frequently used of the condition presented by patients suffering from malignant tumours, but it is also sometimes applied to the state of ill-health caused by syphilis, dysentery, malaria, and other chronic maladies. The subjects of cachexia are generally wasted, anæmic, and debilitated; the skin is wrinkled and inelastic, and often presents a yellowish tinge. The tongue is generally furred, the breath foul, and the appetite absent or disordered.

To explain the cachexia associated with malignant growths it has been suggested that the cells of tumours may give rise to some harmful product which acts as a general poison. There is little evidence in favour of this view. Tumours may grow to a large size without giving rise to cachexia, the occurrence of which is generally explicable on the ground of interference with the digestive processes or as a consequence of the drain on the system caused by the continued discharge from an ulcerated surface.

W. CECIL BOSANQUET.

CADAVERIC (*cadaver*, a dead body).—This word signifies 'belonging to the dead body;' and it is applied to the aspect, colour, odour, and other phenomena resembling those of death, which are sometimes observed in the living subject.

CADAVERIC RIGIDITY.—*See* **DEATH**, Signs of.

CADAVERIC SPASM.—*See* **DEATH**, Signs of.

CÆCUM, Diseases of.—*See* **APPENDIX VERMIFORMIS**; and **INTESTINES**.

CAISSON-DISEASE.—**SYNON.** : Compressed-air illness.—Caisson-Disease is the name given to the illness which is produced by immersion in an atmosphere of compressed air. Immersion in such an atmosphere is involved by the now extending practice among engineers of carrying out subaqueous undertakings, such as the sinking of the foundations of bridges and the boring of tunnels through a water-bearing soil, by the assistance of an artificially compressed atmosphere. With this assistance larger volumes of water can be kept out of the working space than could possibly be dealt with by mere pumping. The illness to which the workers are liable resembles in many respects that which is met with among divers.

The workmen enter the atmosphere of compressed air by means of an 'air-lock' provided at each end with a door opening towards the compressed air; in this lock or chamber the pressure is raised gradually; about 2 or 3 minutes are usually allowed for raising the pressure to 30 lbs. per square inch above the normal.

SYMPTOMS.—The raising of the pressure may be accompanied by pain in the ear, due to closure of the Eustachian tube which prevents the increased pressure reaching the middle ear. If this warning pain be disregarded, rupture of the tympanic membrane may take place, and suppuration of the middle ear may follow. These disorders are often absent, depending only on the accidental lack of patency of the Eustachian tube.

The characteristic symptoms of the illness follow the *exit* from the 'air-lock', which, like the entrance, has to be accomplished slowly. These symptoms may commence immediately the pressure is lowered or may be postponed until some hours after coming

out: the usual interval is from a few minutes to one hour. By far the most common symptom is pain in the extremities, known by the workmen as 'bends.' The pain mostly affects the legs, and principally the parts about the knees. It may be present in one or all of the limbs. It has a sudden onset and rapidly increases for a short time afterwards. The pain may be only slight or it may be of a very severe character. Usually there is no local physical sign. It may pass off in a few hours, or may last for one or two weeks. In a small number of cases severe epigastric pain occurs as well.

In severer cases paraplegia results with or without loss of control over the bladder and rectum. Occasionally the less common symptom of auditory vertigo is met with, generally accompanied by partial deafness and sometimes tinnitus. Attacks of unconsciousness have been described; formication sometimes occurs; epistaxis and hæmoptysis are rarer events. Entry into a compressed-air chamber is in most cases accompanied by a slowing of the pulse and a rise of blood-pressure.

ÆTIOLOGY.—The name of the illness is derived from the fact that while the boring operations are proceeding downwards the compressed atmosphere is contained in the vertical cylinder or *caisson* which is being sunk; when, as in tunnelling operations, the boring is horizontal, the caisson may or may not contain compressed air; the latter may be contained in the tunnel itself; the name therefore indicates neither the causation nor the nature of the illness.

Among the factors which influence the incidence of the disease are the following:—

1. The degree of pressure. The higher the pressure the more numerous and severe are the cases of illness.

2. The duration of the exposure. The longer the stay in the compressed atmosphere the greater is the liability to illness.

3. The purity of the compressed air. The better ventilated the compressed atmosphere the less frequent are the illnesses. The importance of this factor was clearly demonstrated during the construction of the Blackwall Tunnel, where the pressure went up to a maximum of 37 lbs. to the square inch above the atmospheric pressure: it was there shown that the number of cases of illness varied inversely with the amount of fresh air pumped in. At high pressures, however, a very large supply of air, even as much as 12,000 cubic feet per head per hour, may be necessary to minimise the danger.

4. By most observers it is stated that the arrangements for exit form a considerable factor in the causation of the illness; and it is probable that the liability to illness is increased whenever this process is accomplished too rapidly.

5. Certain personal conditions appear to have some influence: (a) Plethoric persons are especially liable to be affected; (b) older men are more apt to suffer than younger men, and (c) those newly employed than the older hands; (d) excessive drinking and great fatigue are predisposing causes.

PATHOLOGY.—Very numerous theories have been advanced to account for the illness; that most worthy of attention was formulated by Paul Bert, who suggested that the symptoms depended on the increased solution of gases by the blood during the sojourn in compressed air, and on their escape during 'decompression.'

PROGNOSIS.—The most common symptom, pain in the limbs, is of the least serious import; the pain often disappears by itself in from one or two hours to one or two weeks. The epigastric pain may assume an alarming character, but usually does not last long. Paralysis usually passes off in a few weeks unless accompanied by bladder-troubles; the fatal cases have generally died from cystitis and bedsores. In the cases of auditory vertigo associated with affection of the internal ear the prognosis may be said to be generally good, though partial or complete deafness may be permanent; in exceptional cases the attacks of vertigo may continue for years.

TREATMENT.—1. *Preventive.*—The indications for prevention have been sufficiently considered in the section on ætiology.

2. *Curative.*—If the patient is seen soon after the commencement of the illness, 'recompression' generally allays the symptoms in a marvellous way, and this improvement may be maintained if the recompression be followed by a very slow exit of, say, half or three-quarters of an hour. A medical air-lock is therefore an important adjunct to any large engineering works where compressed air is used. Rubefacient liniments are useful for persisting 'bends'; anodynes may be of service in some cases. Paraplegia, occurring as a complication, must be treated on general medical principles. Prolonged cases of vertigo are but little amenable to treatment.

E. HUGH SNELL.

CALCAREOUS DEGENERATION.—A form of degeneration characterised by the deposit in the tissues of earthy salts, especially those of calcium. See CONCRETIONS.

CALCULI (*calx*, chalk).—The term 'calculus' is now applied to any kind of concretion formed in the ducts or passages of glandular organs. See CONCRETIONS.

CALIPERS.—An instrument employed for measuring diameters, used more especially in clinical medicine for measuring the head and chest. See PHYSICAL EXAMINATION.

CALLOSITY (*callositas*, *callum*, hard skin).—A local thickening of the horny layer of the epidermis due to intermittent pressure, slight friction, or chemical irritation, continued over a considerable period. See CORN.

CALLUS.—The reparative material formed by proliferation of the periosteum and other tissues around the ends of a broken bone. The exact structure of callus varies with the time which has elapsed since the fracture. At first it consists of tissue closely resembling granulation-tissue (*provisional callus*); later on it contains cartilage, osteoid tissue, and fibrous tissue. The tissue actually uniting the broken ends finally undergoes true ossification (*permanent or definitive callus*).

CALVITIES (*calvus*, bald).—SYNON.: *Alopecia calva*.—A synonym for baldness. See BALDNESS.

CANARIES, The, in North-east Atlantic Ocean; Teneriffe.—Mean temperature in winter, 64° to 85°. Warmer, drier, but more variable, than Madeira. East winds from Africa. See CLIMATE, Treatment of Disease by.

CANCER (*cancer*, a crab).—SYNON.: *Carcinoma*; Fr. *Cancer*; Ger. *Krebs*.

DEFINITION.—The word 'cancer' is without histological meaning. We find it and its synonym *carcinoma* used as long ago as the time of Hippocrates, and the latter term was then applied to any new-growth of a malignant character. The name originated in the large ramifying veins and puckered furrows which spread from a cicatrising cancer that is involving the skin. When the broad distinction between the epithelial and connective-tissue types of tumours was established by Virchow and others, it was decided to retain the word *cancer* as the name for the more malignant or epithelial growths; while the equally meaningless but less formidable word *sarcoma* has been, from this time, confined to those tumours which have connective tissue for their type. See TUMOURS.

In this sense of the word the cancers form a class which is, on the whole, easily distinguished by definite microscopical and clinical characters; but at two points, at least, the difference from simpler growths is almost imperceptible. First, as a matter of accident, one sarcoma (the alveolar) resembles a cancer so closely in microscopical structure that it is impossible to distinguish between them without reference to clinical facts; and, in the second place, as cancers are essentially depraved modifications of epithelial, epidemic, or glandular structures, they may be found to differ so slightly in histological characters from simple hypertrophies that the fact of ultimate malignancy is often all that can decide between, say, a papilloma and an epithelioma, a glandular cancer of the rectum and a simple polypus, or a scirrhous of the breast and an adenoma.

ÆTIOLOGY.—Before speaking of the minute structure of cancer it will be well to consider what is known of the nature of this morbid process. In what follows, sarcoma is regarded equally with carcinoma, since their general laws are approximately the same.

The term 'malignant' applied to both these classes of tumours was originally used merely in the sense of destructive, incurable, and fatal; recently it has received a more exact meaning, founded on the mode of growth and characters of these neoplasms as distinguished from simple tumours. Malignant tumours show the following characters: (*a*) local extension from the point of origin, causing infiltration or destruction first of the part originally invaded, and later of surrounding parts; (*b*) wider extension, by mechanical means, chiefly through the lymphatic or blood-circulation, so as to produce secondary foci of disease—similar to the primary focus; (*c*) a general affection or intoxication of the whole body, leading to malnutrition or cachexia; (*d*) the malignant growth is persistent, and does not subside of itself, spontaneous arrest of growth, if it ever occurs, being exceedingly rare.

These characters sufficiently distinguish malignant growths from simple tumours, and suggest that though these two classes of growths may be sometimes much alike in minute structure, they may be due to entirely different causes.

On the other hand the 'malignant' characters do not distinguish cancer in the wide sense from specific infections and inflammations, especially from the more chronic kinds. Local extension or infiltration is shown by the inflammations due to pyogenic cocci; still more by syphilitic, tubercular, and leprosy lesions. The difference in 'malignancy'

between lupus, destructive syphilitic lesion, and rodent ulcer or epithelioma is mainly one of degree. The specific infections may be distributed through the body in the same way as malignant growths, and produce marked intoxications or cachexia.

On these grounds carcinoma at all events (even if the conclusion is less clear as regards sarcoma) must be regarded not as a growth only but as a specific morbid process comparable to the specific diseases produced by microbes. However, there are certain differences between malignant diseases and the specific microbial diseases which cannot be ignored.

1. In cancer the elements first affected are not destroyed, as in cases of infection, but are stimulated to enormous overgrowth.

2. In malignant disease there is little local defensive reaction (inflammation) or perhaps none at all, apart from contamination with pathogenic organisms.

3. In malignant disease, though a rise of temperature is sometimes observed, fever is not commonly produced.

4. The secondary growths of malignant disease are produced, so far as is known, by transplantation of cells of the original growth to other parts of the body. But the secondary foci of specific inflammations may be set up by the conveyance of the microbes which caused the original focus of disease, the cells of the tissue first affected not taking any part in the process.

These differences appear to some pathologists fundamental; but nevertheless the opinion is gaining ground that malignant disease may, like the specific infections, be due to some specific living organism—in other words, that *cancer may be a parasitic disease*.

Very numerous researches, of which we can only give the barest outline, have been devoted to discovering some pathogenic parasitic organism in cancer. In the first place, as was natural, search was made for bacteria, but the result of these researches has been entirely negative.

Another class of organisms suspected of causing cancer is the protozoa. Certain appearances in the cells of cancer were thought to indicate the presence within them of animals of the order Sporozoa allied to the coccidia found parasitic in the liver. But these supposed organisms have never been isolated or cultivated, so that they are not proved to be living organisms, and they are now generally regarded as due to degenerative changes merely. A third class of organisms which has been supposed to be the parasitic cause of cancer-growth is that of yeast-fungi (Blastomycetes). Various appearances in cancers have been thought to indicate the presence of such organisms, the appearances being partly the same as those formerly interpreted as sporozoa. These bodies have been observed especially by Italian pathologists, Sanfelice and Roncali; and also by Plimmer and others in this country. Further it has been shown (first by Sanfelice) that yeast-organisms obtained from fruits, if injected into animals, are capable of producing tumour-like masses which have been regarded as true new-growth. Finally, yeast-organisms have been cultivated from cancer-tissues (by Plimmer in a long series of cases), which introduced into animals have produced tumour-like masses resembling those produced by yeasts from vegetable sources.

Against this it is objected that the masses produced in experiments on animals by blastomycetes are only inflammatory or granulation-tumours, not true tumours: further that the mere presence of blastomycetes in cancerous growths, even if proved, does not show that they have any causal relation to them.

From the facts of which a broad outline has been given above, we should conclude that the parasitic theory of cancer is not yet established; it has not been proved that cancer is caused by bacteria or sporozoa or blastomycetes, or any other organisms.

In support of the theory of a specific origin for cancer some other considerations have been adduced.

1. Contagion.—Though cancer is certainly not in the ordinary sense contagious, there are facts showing that it may be transplanted from one part to another of the same individual. Also in very rare cases, it has been thought to pass from one person to another having very intimate relations, such as those of husband and wife, by local contagion. Cancerous tumours of certain animals have also been transplanted to other animals of the same species; but the process resembles grafting more than inoculation.

2. Local Distribution.—Careful observation has shown that cancer is more prevalent in some countries or districts than in others. Moreover certain dwellings appear to confer on their successive inhabitants a special liability to cancer: so that the existence of 'cancer houses,' in rural districts, where there is little movement of population, seems to be well established. The favouring physical conditions, if any, are imperfectly known, but certain river-valleys and low-lying basins have been thought to show special liability. These considerations would seem to point to some specific external or exogenous cause.

Assuming from the above facts that there is ground for regarding cancer, at least provisionally, as a specific disease, we will point out the best known favouring conditions and laws of the disease.

1. Age.—No disease is more distinctly a disease of later life than cancer. It is very rare under twenty-five; and probably most of the deaths recorded in early life as due to cancer are really cases of sarcoma. The deaths below thirty-five are comparatively few, and the largest number of deaths occurs between fifty-five and sixty-five. But if regard be had to the actual number of persons living at successive age periods, the liability to cancer is seen to go on increasing up to advanced age.

Sex.—It is generally recognised that cancer is commoner in the female sex than in the male; the difference being chiefly due to the predominance of cancer of the generative organs in the former.

General Health.—There is no reason for thinking that any kind of cachexia or ill-health predisposes to cancer. On the other hand, the subjects of this disease are generally free from other disease, and on the whole robust. It is thought to be more prevalent in long-lived families, and probably with justice.

Habits of Life.—Poverty, want of cleanliness, and what is generally called bad hygiene, do not at all favour the production of cancer. It is supposed, though not easily proved, that it is commoner in the upper classes than in the lower. Abundant nutrition and it would seem especially abundance

of animal food are favouring causes. It has been supposed that dwellers in the country are more liable than town-people, but at the present time the 'cancer death-rate' both for England and Scotland is distinctly higher in towns than in rural districts.

Heredity.—It seems clear on the whole that the children and descendants of those who have died of cancer show more than the average liability to the disease. This inherited liability is distinctly more marked in women than in men; and in the former is especially seen in cancer of the reproductive organs. It is not supposed that a germ or seed of cancer is inherited, but only a greater predisposition to become affected by the disease. Since cancer rarely causes death before the reproductive age, this inherited predisposition seems *a priori* likely to become more widely spread in the population.

Nervous influence.—It has been thought that depressing causes acting on the nervous system, such as shock, grief, &c., predispose to cancer, but the number of cases of this kind is too small to establish an induction.

Chronic Inflammation or Trauma.—It has often been observed that cancer occurs in tissues which have been exposed to chronic traumatic irritation leading to inflammation. The effect of smoking, especially pipes of a certain kind, in predisposing to epithelioma of the lip or tongue; and of alcoholism, in predisposing to cancer of the stomach; the occasional growth of epithelioma on the basis of lupus, and of carcinoma on chronic gastric ulcer, and other instances might be quoted. The liability of chimney-sweeps to cancer is a fact of the same kind. Occasionally an acute trauma has been apparently the starting-point of cancer. These facts seem to show that damaged tissues are more liable to the invasion of cancer, but do not explain that invasion.

Increase of Cancer.—The increase is generally more rapid in males than in females, especially in England; but this difference is much less marked in Scotland. Since the predominant cancers of the male sex are those affecting the digestive organs, while in the female sex cancers of the breast and the uterus predominate, the difference points to the conclusion that the increase of cancer is largely due to increased frequency of cancer of the digestive organs. Where statistics of cancer show the part of the body affected (as in Ireland), it is found that cancer of the digestive organs, including cancer of the tongue, increases more rapidly than the average increase; but that there is an increase in cancer of the reproductive organs in women, including the mamma. In St. Thomas's Hospital 'digestive cancers' have come to predominate over those of reproductive organs, even in the female wards.

It has been objected that cancer is now more certainly diagnosed and more accurately registered than in former days, and that therefore the increase shown by statistics is only apparent. But though this objection might have weight if the comparison were with the statistics of thirty or forty years ago, it would not explain the increase during the last ten years in which the conditions of diagnosis and registration have remained virtually constant. The objection is supported by the consideration that the increase being chiefly in the male sex refers to internal cancers which are more difficult of diagnosis. But the more rapid increase of cancer in males is not so marked in other countries as in England. Moreover, this would not account for the

increasing cancer in the breast and the tongue, about which, especially when fatal, there is no difficulty in diagnosis. Moreover, the increasing success of operation in curing cancer of accessible parts is a factor which ought to tell in the contrary direction. So that there seems no reasonable ground for disbelieving in an actual increase of cancer: and this is now admitted by the heads of the statistical departments in England, Scotland, and Ireland.

The explanation of this increase of cancer is very difficult. It has been supposed to be due to an increased average longevity of the population, by which a larger proportion of the population survive to the age at which they are liable to cancer. But the census of 1891 *did not* show a larger proportion of persons in the United Kingdom living at the higher ages, as compared with 1881. On the contrary the proportion living at certain age periods above thirty-five was even less than it had been. Moreover, Dr. Newsholme has shown that if the cancer-mortality is calculated only for the population at higher ages (above thirty-five), there is still an increase in the registered mortality from cancer. Another explanation is that the improved diet of the working classes who form the mass of the population, and especially the enormous increase in the consumption of meat, have brought them into the state of nutrition most favourable to cancer. Considering the predominance of cancer of the digestive organs, this explanation has much plausibility. But observations in countries where the customs of diet are different are needed to confirm it. The partial explanation suggested by the facts of heredity has been already referred to.

On the whole, we must conclude that the cause of the increase of cancer, like the cause of cancer itself, is yet undiscovered.

HISTOLOGICAL STRUCTURE.—Histologically, cancers are distinguished by consisting partly of cells of an obviously epithelial origin and partly of connective tissue. The connective tissue forms alveolar spaces, and may vary in structure from a loose fibro-cellular material to strong and old fibrous tissue. The alveolar spaces communicate with each other and contain the epithelial cells. These vary much in shape, size, and arrangement, but are always easily separable from the surrounding connective tissue, while they are never separated from one another by a stroma of any sort.

CLINICAL CHARACTERS.—Clinically, cancers are distinguished by the structures in which they originate; by the method of their recurrence and their mode of growth; as well as by a few characteristics apparent to the eye and touch.

Seat.—As their nature would *a priori* have rendered almost certain, cancers probably never originate except in connection with epithelial or epidermic structures—i.e. in skin, mucous membrane, or secreting glands; but as the epidermis and epithelium, the original upper and lower layers of the embryo, are widely diffused throughout the body, and often intimately associated with the descendants of the cells of the middle layer, it is not surprising that primary cancers have been described as occurring in organs which have their origin from connective tissue only. Such are the instances of primary cancer of bone and lymphatic glands, the possibility of the occurrence of which may be at present considered undecided.

Recurrence.—The first recurrence is almost with-

out exception in the lymphatic glands, which collect their supply of lymph from the seat of the original tumour; when this has occurred the process may be repeated in the next proximal lymphatic glands, or numerous distinct tumours may appear in different parts of the body; but if a single growth occur in another locality without previous glandular enlargement, the case may probably be looked upon as one of double primary development. A soft cancer may break into the abdominal cavity, where its small particles may stick to various parts of the peritoneum and form the starting-points of new growths (disseminated cancer of the peritoneum); it is probable that a similar seeding may take place into the lungs when an ulcerated epithelioma projects into the trachea. The recurrence is usually of precisely the same structure as the original growth.

Mode of growth.—Cancers increase in size by infiltration of the surrounding tissues, and this gives rise to the very important clinical facts that they are not enclosed by a capsule like many simpler growths, and that they have a great tendency to implicate the skin and cause ulceration.

Naked-eye appearances.—The contraction of the connective tissue forming the alveoli in its advance towards fibrous tissue gives rise to puckering of the surrounding skin; and the looseness of the connection between the epithelial and connective-tissue elements causes a milky juice, consisting of the former, to escape on scraping a recent section. This characteristic was made much of by our predecessors before the word *cancer* had lost its inclusive meaning; we know now that many rapidly growing *sarcomata* yield a similar juice, but in less abundance than cancers; and thus it has come to pass that a milky juice is now more diagnostic of the malignancy than of the genetic origin of the growth.

This completes the list of the signs by which cancers may be distinguished from other tumours. Tables have been published to show the relative frequency with which cancer attacks different organs; they are not, upon the whole, trustworthy, and this question will be best considered in discussing the subdivisions of the genus.

CLASSIFICATION.—The subdivisions of cancers are as follows:—

<i>Hard cancer</i> or <i>Scirrhus</i> .	}	GLANDULAR TYPE.
<i>Soft cancer</i> or <i>Encephaloid</i> .		
<i>Lobular</i> or <i>Flat-celled Epithelioma</i> .	}	EPITHELIAL and EPIDERMIC TYPE.
<i>Cylindrical Epithelioma</i> .		
<i>Colloid cancer</i> .		

Typical hard and soft cancers stand obviously at opposite ends of one series which is built upon the type of the secreting gland: between the two are an infinitude of intermediate stages. The two forms of epithelioma are, quite as evidently, monstrous growths of skin or mucous membrane. Colloid is probably the result of degeneration of any one of the other forms. Besides these, other varieties are often mentioned which do not justify a more complicated classification; among these are tumours, which though of nearly normal glandular structure are nevertheless malignant, and those which have received the names *melanotic*, *telangiectatic*, *osteocancer* &c.

DIAGNOSIS.—The diagnosis depends upon the clinical characters of the several groups. That of

an advanced case of cancer is generally easy; in the early stages it is mostly impossible.

PROGNOSIS.—The prognosis is always bad, especially in encephaloid cancer, but least so in epithelioma; this suggests the much-debated question of the constitutional nature of the disease. If in its origin a cancer be purely local, early removal ought to effect a permanent cure; but if there be at the bottom a constitutional taint, a reprieve should merely be granted until a suitable fresh irritation arise.

COURSE.—The course of a cancer depends upon its seat, and the symptoms must accordingly be sought among the articles on diseases of special regions. If, however, life be not shortened as a result of interference with the functions of the organ attacked, death is caused either by marasmus—the result of prolonged suppuration and pain—or by extensive or repeated hæmorrhages. The rate of progress is more slow as age advances.

TREATMENT.—The treatment of cancer in the early stages belongs to the surgeon, the practitioner giving such counsel as will improve the general health and support the strength. In the later stages the physician may be called upon to treat symptoms; but up to the present time all the specifics introduced either by regular practitioners or by charlatans have proved quite inefficient, if not actually harmful.

We shall now discuss the varieties of cancer.

I. Scirrhus.—Scirrhus, as its name implies, is among the hardest of tumours, if bony growths be excepted. Its hardness, as compared with soft cancer, depends upon the large proportion which the alveolar stroma bears to the contained cells; and this is probably the consequence of the soil in which the tumours originate, and the rapidity of their growth, rather than of any specific difference between them.

Seat.—The female breast is the most common seat for scirrhus, but it also occurs in the stomach, uterus, tongue, œsophagus, and the liver and other glands, and it has been described as primary in the prostate, testicle, skin, and other structures.

Naked-eye appearances.—A section through the centre of a matured hard cancer of the breast presents to the naked eye well-marked and constant appearances, which, with the exception of such peculiarities as are due to the situation, will serve as a description of such a tumour occurring elsewhere. These are clearly explained by the microscopical arrangement, and, when looked at by the light which it affords, fully account for all the clinical characters. The knife passes through it with a creaking noise, and the cut surfaces are at once hollowed in the centre. There is not a sharp edge to the growth, and the circumference is of a greyish or pinkish-white tint, projecting a little above the surrounding tissues, into which it sends small lobular prolongations; the hollow centre is very hard and of a glistening white colour. Scirrhus is evidently fibrous in structure, and receives from all quarters fibrous bands, which often extend far into the fat of the breast or the skin, and some of which can nearly always be traced to the principal milk-ducts. Between the centre and the edge the greater part of the tumour is on the whole of a pinkish-yellow colour, but notably pink and soft externally, and yellow and hard internally. The surface yields a milky juice on scraping, and may in certain cases show some of

the following appearances, which are, however, accidental: round the circumference little masses of healthy fat may be included, though this but rarely happens; cysts containing grumous grey or red fluid may have formed by the breaking down of the new-growth or by hæmorrhage; or such a hæmorrhage may have resulted in patches of yellow or even black pigmentation.

Microscopical appearances.—Without discussing the merits of the opposing theories as to the origin of cancer-cells, the following may be taken as the undoubted microscopical appearances of scirrhus. The grey outer layer is made up of indefinite smallish round cells, resembling white blood-corpuscles, infiltrated through the tissue into which the growth is spreading, among which are scattered a few which have the appearance of epithelial cells. The next or pink layer represents full development, and shows fibro-cellular stroma, enclosing large epithelial cells, and containing a copious supply of vessels. In the third or yellow layer the stroma has become fibrous, and the cells are undergoing fatty degeneration; and in the inner white centre the cells are replaced by indefinite masses of granular debris, and the stroma consists of firm and old fibrous tissue. See Plate, figs. 8, 10, 11.

Clinical features.—The relation of these appearances to the clinical peculiarities of scirrhus is as follows: the excessive hardness is explained by the great development of fibrous tissue; the peculiar indefiniteness of the edge, and the tendency to involve the skin and ulcerate, by the manner of growth; while the puckering, retraction of the nipple, and indirectly (from the manner in which cutaneous nerves are involved) the pricking and shooting pains, are due to the contraction or cicatrization of the stroma. To the latter is also due a very important but not generally recognised diagnostic character of an early scirrhus—namely that long before the skin is involved it is seen to be dimpled when gently moved to and fro over the growth.

A scirrhus which has involved the skin forms a purplish-red, flattened, and shining tumour, covered with small veins and tender to the touch. The ulcer which results from its breaking down is ragged, with a hard base and hard irregular undermined edges, and a dirty surface covered by knobby masses resembling granulations, which have a great tendency to bleed, and often slough. After removal it often returns in the scar. When occurring in the liver it is softer than elsewhere, and the name of scirrhus-encephaloid is often given to it. See fig. 7.

II. Encephaloid.—Encephaloid, medullary, or soft cancer, so named from its usually brain-like appearance and consistence, is softer and grows more rapidly, and is more frequently observed in internal organs than scirrhus, often indeed forming enormous intra-abdominal tumours.

Seat.—It has hitherto been observed as primary in the salivary and mammary glands, testicle, ovary, and prostate, the thyroid body, and in the mucous membrane of the nose, the liver, and the stomach. It has with some degree of looseness been called the cancer of childhood by those who consider scirrhus as almost peculiar to mature age.

Naked-eye appearances.—To the naked eye a fresh section usually presents a convex surface; it is whitish, but generally mottled by coloured patches the result of old or recent hæmorrhages; and yields very copiously a milky juice on scraping.

Microscopical appearances.—Encephaloid cancer

differs from scirrhus only in the relative proportion of the two chief factors. The cells are more numerous and are contained in larger spaces; they are sometimes small, but generally much larger than in scirrhus; and the stroma is delicate and fibro-cellular and very small in amount. See fig. 9.

Its method of extension is the same as that of other members of the class. It is by far the most malignant form of cancer, because of its rate of growth and recurrence, and the rapidity with which it causes general cachexia.

III. Lobular Epithelioma.—Lobular or squamous epithelioma, epithelial cancer, or cancrroid, develops in connection with skin and mucous membrane, and, though consisting essentially of squamous epithelium, may start from a part which is covered by the cylindrical variety. It occurs near the natural orifices of the mucous tracts—as, for example, on the mouth and tongue, anus, penis, or vulva; but also at other parts of the skin—as on the scrotum (chimney-sweep's cancer) and at the upper end of the œsophagus. The history of a local irritation as its origin is often obtainable, but more frequently nothing of the kind can be discovered.

Naked-eye appearances.—The first appearance is that of a pimple, which soon breaks down in the centre, forming a small sore. When fully developed there is an irregular ulcer, with an extensive, hard and nodular, generally inflamed base and circumference; the edges are abrupt or undermined, and the floor grey or reddish, very uneven, discharging a foul pus, and with a great tendency to bleed. As a rule there is considerable pain, and the proximal lymphatic glands are very generally enlarged. A section to the naked eye shows a number of minute cylinders of yellowish-white colour, cut sometimes longitudinally, sometimes transversely, fusing together into an indefinite mass superficially, but more or less discrete below, and infiltrating the subjacent tissues. On squeezing the section little nodules like sebum appear on the surface.

Microscopical appearances.—The cylinders or lobes of epithelioma are found to be made up of squamous epithelium, the cells of which generally exhibit in parts a crenated margin (Max Schultze's 'prickle-cells'). As in the skin, the deeper—that is the circumferential—layer of cells in each lobe, which are the youngest, are roundish or oblong, with large nuclei which stain readily; further in, the cells are larger and flatter, and in the centre are found the well-known globes or nests. These were at one time considered peculiar to epithelioma, but are now known to occur in warts and corns; they consist of onion-like arrangements of epithelial cells, varying much in size and the number of concentric layers, and containing in the centre sometimes an amorphous mass, sometimes large and irregular cells. The tissues beneath and between the lobules are infiltrated with small cells, and often contain in sections what appear to be isolated masses of epithelium; these are, however, the ends of divided divergent lobules. Opinions differ as to the exact starting-point of an epithelioma, the share which the sweat and other glands take in it, and also as to the rationale of the formation of the globes. See fig. 2.

Epithelioma seems to be more local in its nature than other cancers—that is, a complete and early removal has not infrequently given the patient a long lease of life. It recurs, as a rule, in the lymphatic glands (which inflame and break down or suppurate) or in the scar; and generally proves

fatal from the constitutional disturbance which these local manifestations give rise to. Later, but more rarely, it may appear in the internal viscera, bones, &c.

IV. Cylindrical Epithelioma.—The cylindrical epithelioma—badly named adenoid or glandular cancer—is specially the cancer of the alimentary mucous membrane, but may occur in the bladder and elsewhere.

Naked-eye appearances.—To the naked eye it forms at first a prominent tumour in the interior of a viscus, which has a tendency, like other cancers, to ulcerate and involve surrounding tissues, so that the mass may reach an enormous size, and may even make its appearance through the skin. To the naked eye a section is generally whitish and has a granular appearance, which is given to it by the tubules of which it is made. It frequently causes death by obstruction of the bowel; but if it last sufficiently long, it recurs in the lymphatic glands, and then in the viscera and other parts of the body. It is not unusual to find recurrences in the liver with little if any implication of lymphatic glands. The recurrence reproduces precisely the structure of the original tumour.

Microscopical appearances.—Cylindrical epithelioma consists essentially of irregular tubules lined with columnar epithelium in one or more layers, which are the much overgrown crypts of Lieberkühn: it differs in microscopical structure from simple papilloma of the digestive tract only in the greater irregularity of the cells and in the larger proportion of connective-tissue stroma between the tubes. Compare figs. 4 and 5.

V. Colloid Cancer.—Colloid cancer, named from its jelly-like appearance, has given rise to much discussion in reference to the question whether it is developed originally in its mature form, or whether it results from the degeneration of one of the varieties of cancer already described. The latter view is that most widely held, though it must be allowed that epithelioma seldom degenerates in this way, and also that the colloid change usually takes place *pari passu* with the growth of the tumour.

Seat.—Colloid cancer is found most frequently in the abdominal viscera and peritoneum, but may occur elsewhere, as in the breast. Its malignancy is great, but is shown chiefly by the rapidity with which it involves surrounding tissues; it thus forms primary tumours of enormous size, but as a secondary growth is less common; it does, however, occur in lymphatic glands and other parts. It causes death in most cases by interference with the functions of the organs attacked.

Naked-eye appearances.—Colloid cancer consists to the naked eye of a mass of semi-transparent jelly, varying slightly in colour, but mostly pale yellow: this is intersected by delicate white fibrous bands, forming alveolar spaces of different sizes, visible to the naked eye. The consistence of the growth depends upon the relative proportions of these two constituents.

Microscopical appearances.—The bands are found to be actually fibrous; the contained jelly is arranged in concentric laminae between which are minute granules, and in the centre of which is a granular mass, sometimes quite indefinite, but often showing clearly that it consists of the remains of altered cells. These cells are seen in the more recent parts of the growth to be the subjects of

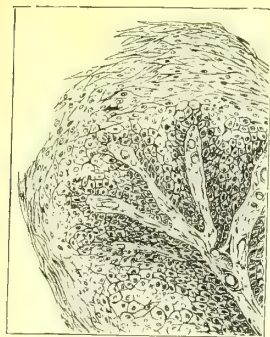


FIG. 1.—Papilloma of Soft Palate.

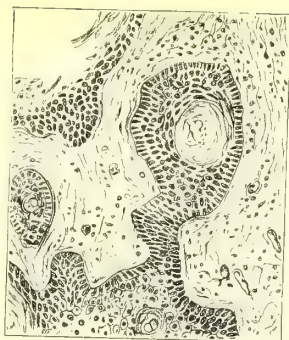


FIG. 2.—Epithelioma of Lip.

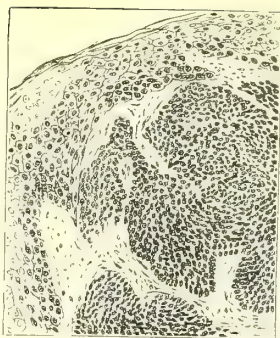


FIG. 3.—Edge of Rodent Ulcer.

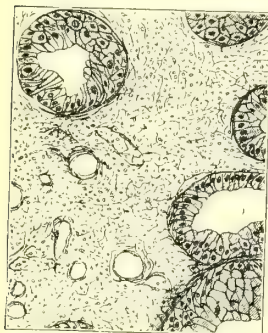


FIG. 4.—Simple Polypus of Rectum.

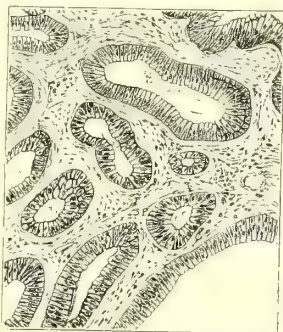


FIG. 5.—Columnar Epithelioma of Intestine.



FIG. 6.—Colloid Cancer of Breast.

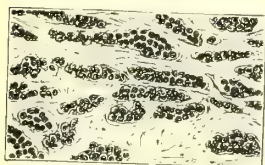


FIG. 7.—Cancer of Liver (Scirrhus-encephaloid).

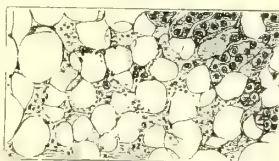


FIG. 8.—Scirrhus infiltrating Fat.

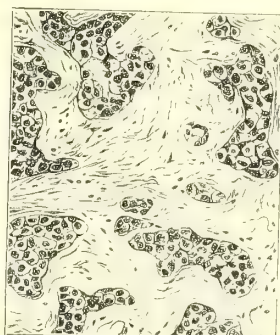


FIG. 11.—Scirrhus of Mamma.



FIG. 9.—Encephaloid Cancer.



FIG. 10.—Cicatrising Cancer.

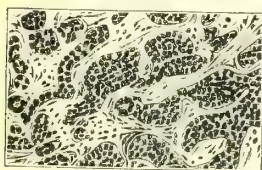


FIG. 12.—Adenoma of Upper Jaw (Benign).

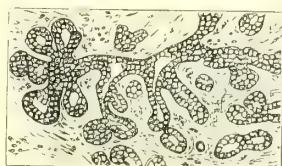


FIG. 13.—Adenoma of Breast (common type).

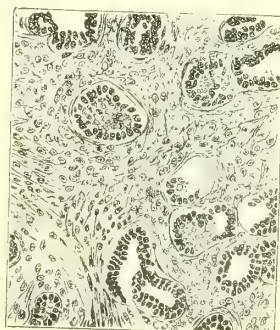


FIG. 16.—Adeno-sarcoma of Breast.



FIG. 14.—Ulcerated Adenoma of Paucid (Malignant).



FIG. 15.—Adenoma of Breast (epithelial element in excess).

colloid degeneration. The source of the colloid material must be considered still undecided; that some of it is formed by the cells is certain, but it is not equally clear whether the stroma takes any share in its deposition. See fig. 6.

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CANCERUM ORIS (*cancrum*, a sore; and *oris*, of the mouth).—SYNON.: Gangrenous Stomatitis; Noma; Water-canker; Fr. *Nome*; Ger. *Wasserkrebs*.

DEFINITION.—A rapidly progressive gangrenous ulceration of the cheek, gum, and lip.

ÆTIOLOGY.—Cancrum oris is usually seen in ill-fed, ill-tended children, living under the most faulty hygienic circumstances. It frequently ensues upon some acute degenerative illness, especially measles (110 times in 226 cases—Sannée). Occasionally two or three cases occur together in a school, suggesting the possibility of contagion. Lingard has demonstrated long threads of small bacilli in the spreading edge of noma in man, and in a similar disease of calves; and inoculation on calves of a pure culture from man induced the disease.

SYMPTOMS.—Cancrum oris commences on the inner surface of the cheek, often near the angle of the mouth, or upon the gum; and at this stage some superficial ulceration will usually be found here—that is, ulcerative stomatitis. The disease rapidly advances. The cheek becomes much swollen, raw, shining, and red; a livid spot appears in the centre, and here a spreading black slough forms, while the surrounding parts become purplish or mottled. If the patient can open his mouth sufficiently, the gums will be seen to be red and spongy, ulcerating, or sloughing; even the tongue may show some destruction. The cheek is lined with a tough, adherent, greyish-yellow or soft purplish slough; the jaw may be laid bare, the teeth loosened, and portions of the alveolus necrosed. The breath is very foul, and the flow of saliva is excessive and fetid. Fever may be moderate or high. Pain is slight or absent, and the child, though lethargic, is conscious almost to the end. Not more than one in twenty cases recovers, death being generally due to septic broncho-pneumonia.

DIAGNOSIS.—Formerly mercurial stomatitis sometimes ran on to gangrene in debilitated patients; this inflammation was general, and affected the tongue markedly. No hard line can be drawn between ulcerative stomatitis and noma.

TREATMENT.—This consists in complete and careful excision by knife and sharp spoon of the diseased area, regardless of the deformity which must result. To parts which cannot be excised, nitric acid or pure phenol should be freely applied. Iodoform-gauze is the best dressing. The mouth must be frequently and carefully syringed and sponged out with an antiseptic lotion, especially before food is taken. As far as possible, the child should lie so that foul saliva &c. may dribble from the mouth and not down the throat. Goodhart thinks that even tracheotomy might be done to prevent inhalation of septic material into the lungs. Fluid food, especially pancreatised beef-tea or milk-gruel, should be given regularly and frequently, by mouth, nasal tube, or enemata. Ammonia and bark or other tonics should be exhibited. In cases in which recover some plastic operation will be required.

STANLEY BOYD.

CANITIES (*canus*, hoary or grey-haired).—Whiteness or greyness of the hair. See HAIR, Diseases of.

CANNABIS INDICA Habit.—See DRUG-HABITS.

CANNES, in France, on the Mediterranean coast. A dry, bracing, fairly mild winter climate. Exposed to N.W. Abundant accommodation, both near and at some distance from the sea. See CLIMATE, Treatment of Disease by.

CANTHARIDES, Poisoning by.—SYNON.: Fr. *Empoisonnement par la Cantharide*; Ger. *Cantharidenvergiftung*.

Cantharides, the so-called Spanish Fly, owes its poisonous properties to the presence of *cantharidin*, a non-alkaloidal body. All the preparations of the drug are highly poisonous.

SYMPTOMS.—Soon after taking a poisonous dose of cantharides, the patient is seized with burning pain in the pharynx, and a sense of constriction in the œsophagus. The pain soon extends to the abdomen, and vomiting ensues; the abdominal pain becomes aggravated; and usually purging sets in. The stools are numerous, often scanty, passed with great pain and straining; they are at first mucous, and finally bloody. If carefully examined, little iridescent specks—portions of the elytra of the beetle—may be observed in the fæces and vomited matters. These are of course only observable when the powdered insect has been taken; and they frequently escape observation. Up to this period of the case, should portions of the beetle not have been detected, there is nothing to distinguish the case from one of ordinary irritant poisoning; except, perhaps, that salivation and swelling of the salivary glands are usually prominent symptoms. The gastrointestinal inflammation may be so intensely and rapidly developed, that death may occur from collapse before strangury, the diagnostic symptom, is set up. Generally, however, the course is somewhat different, genito-urinary irritation and inflammation setting in; the symptoms of which are aching pains in the lumbar region, frequent desire to micturate, with violent tenesmus of the bladder, till eventually a few drops of albuminous or bloody urine only can be passed, or none at all. Priapism, erotic excitement, and swelling of the genitals are of frequent, though not of constant, occurrence. Delirium, tetanic convulsions, or paraplegia, may be noted in some cases. Eventually the intolerable agony gives way to collapse, stupor, coma, and death. Abortion not infrequently occurs in pregnant women, the drug being formerly much used as an abortifacient.

DIAGNOSIS.—The intense strangury, the swelling of the genitals, and the bloody stools, will leave little or no doubt as to the nature of the case; and the presence of particles of the drug in the ejecta will be conclusive.

Fatal dose.—Less than half a drachm of the powder, and an ounce of the tincture, have alike proved fatal.

TREATMENT.—Evacuation of the stomach by the use of the stomach-pump, syphon-tube, or an emetic is the first indication in poisoning by cantharides. It is best to thoroughly wash out the stomach. Mucilaginous drinks, white of egg (not the yolk), and demulcents, may be freely given; but oil in any form is to be avoided, as tending to dissolve cantharidin. Opium by the mouth or rectum, or the hypodermic injection

of one-third of a grain of morphine is advisable. Leeches to the region of the bladder, warm fomentations, and warm sitz-baths may afford relief. Chloral hydrate should also be given or the patient kept cautiously under the influence of chloroform. Collapse may be met by ammonia and other stimulants. The hypodermic injection of ether is useful. There is no known antidote for cantharidin.

THOMAS STEVENSON.

CAPE OF GOOD HOPE.—A warm, generally dry climate, but very variable, and liable to sudden storms. *See* SOUTH AFRICA; and CLIMATE, Treatment of Disease by.

CAPILLARY BRONCHITIS.—Inflammation involving the minute bronchial tubes. *See* BRONCHI, Diseases of.

CARBOLIC ACID, Poisoning by.—*See* POISONS.

CARBONIC ACID, Poisoning by.—The inhalation of carbonic acid causes injurious or fatal results, according to the length of time and degree of concentration. Carbonic acid accumulates in large quantities, almost undiluted, in pits, cellars, wells, mines (especially after explosions, constituting what is called choke-damp), volcanic grottoes, fermenting-vats, lime-kilns, &c. A continuous contamination of the atmospheric air with carbonic acid goes on from the respiration of animals and the combustion of fuel. The generation of CO_2 by a human being averages about 0.6 cubic foot per hour; that of an ordinary gas-burner 2 cubic feet of CO_2 for every one of gas burnt; that of an oil lamp relatively more. The gradual exhaustion of oxygen and proportionate accumulation of carbonic acid in ill-ventilated apartments is one of the factors of the evil results of bad ventilation, but not the only one, as other animal exhalations contribute largely to the result.

As a rule excess of carbonic acid means corresponding deficiency of oxygen; an atmosphere containing 3 to 4 per cent. produces hyperpnœa, and 10 per cent. cannot be inhaled without causing great distress. Haldane breathed air containing 18.6 per cent. for a minute and a half to two minutes, which caused hyperpnœa, cyanosis, and confusion of mind; such a percentage of carbonic acid, and probably one much lower, could not be long inhaled without causing death. Much less than this causes injurious consequences if long inhaled; and less than 2 per cent. cannot be breathed for any length of time with impunity.

If the amount of oxygen be not correspondingly diminished, carbonic acid, if present in sufficient quantity in the atmosphere respired, will still act fatally. Thus Bernard found that a bird died instantaneously in an atmosphere of equal parts of oxygen and carbonic acid; and Snow found that 20 per cent. of carbonic acid in an atmosphere containing the normal proportion of oxygen soon proved fatal to small animals, and that even 12 per cent. might cause death after a longer interval.

SYMPTOMS.—Undiluted carbonic acid is not readily inhaled, as it tends to induce spasm of the glottis, but immersion in such an atmosphere is rapidly fatal. It seems to act as a narcotic. The patient falls down prostrate and insensible, and death occurs almost immediately. This effect is seen occasionally when labourers incautiously de-

scend an old well, or when miners enter a region filled with choke-damp. Not infrequently several fall victims, as one goes to see what has happened to the other and meets the same fate.

When the carbonic acid is more diluted, the symptoms are headache, giddiness, and sense of oppression, followed by drowsiness, and singing in the ears. Ultimately a condition of insensibility ensues, with stertorous breathing and muscular prostration; death usually occurring quietly and without convulsions. If the excess of carbonic acid coincides with deficiency of oxygen, we have, in addition to the essentially narcotic effects of carbonic acid, the dyspnœa and other symptoms of asphyxia. *See* ASPHYXIA.

POST-MORTEM APPEARANCES.—These are largely those of asphyxia, namely, a general engorgement of the venous system. This is generally seen in the brain, more frequently than in asphyxia pure and simple. The blood is dark and fluid. The hæmoglobin is completely reduced. Animal heat is said to be retained long after death, and rigidity is well marked and enduring.

PATHOLOGY.—As has already been said, carbonic acid does not merely act as a negative asphyxiant by taking the place of oxygen, but has a distinctly toxic narcotic effect. Very frequently in cases of poisoning by carbonic acid there is a combination of asphyxia, essentially due to deficiency of oxygen, with the narcotic symptoms due to carbonic acid.

TREATMENT.—1. *Prophylactic.*—Caution should be exercised in exploring wells, mines, &c., where there is any likelihood of the accumulation of carbonic acid. The introduction of a lighted candle is a rough and ready test of considerable value. The mere fact of a candle continuing to burn in an atmosphere is no test of its being respirable with impunity, for a candle will burn in an atmosphere containing 10 per cent. of carbonic acid if the oxygen is present in the normal amount. If carbonic acid reaches the proportion of 16 per cent. the candle will be extinguished. If a candle is extinguished the atmosphere should be regarded as unsafe. If carbonic acid is present, it should be expelled by creating a draught of some kind. Thus wells may be swept by some such contrivance as an inverted umbrella, and a stream of air or steam can be directed into enclosed spaces.

2. *Restorative.*—Artificial respiration and its various accessories are needed to restore a person actually in a state of coma from carbonic acid. This treatment, of course, is subsequent to instant removal from the impure atmosphere. Pure oxygen should also be administered if at hand. *See* ARTIFICIAL RESPIRATION; and RESUSCITATION.

D. FERRIER.

J. DIXON MANN.

CARBONIC OXIDE, Acute Poisoning by.—On account of its cumulative action, carbonic oxide is a much more dangerous toxic agent than carbonic acid. Pure carbonic oxide is rarely generated out of the chemical laboratory, but it is not infrequently met with in admixture with other gases. The most common sources of carbonic oxide as a poison are:—(1) illuminating gas, whether ordinary coal-gas, which contains from 5 to 8 per cent. of carbonic oxide; or carburetted water-gas, which contains 20 per cent. or more; (2) the fumes given off from burning charcoal, from imperfectly fitted slow-combustion-stoves, from brick-kilns, from certain ex-

ploives used in mines, and from the combustion of inflammable materials in the interior of buildings which are on fire.

The toxic effects produced by carbonic oxide are determined by two conditions: the percentage of the gas present in the atmosphere which is inhaled, and the duration of the inhalation. From experiments made on himself, Haldane came to the conclusion that 0.05 per cent. of carbonic oxide in otherwise normal air produces distinct toxic symptoms in man, and that 0.2 per cent. causes urgent symptoms. One per cent. is usually accepted as a fatal admixture. A concentrated atmosphere of carbonic oxide rapidly causes death; a more dilute atmosphere of not less than 1 per cent. is equally fatal if the period of inhalation is prolonged.

PATHOLOGY.—The powerful toxic action of carbonic oxide is due to its affinity for hæmoglobin, which is 140 times greater than the affinity of oxygen for hæmoglobin; the consequence is that when carbonic oxide is inhaled it displaces the oxygen of the hæmoglobin and forms carboxyhæmoglobin, which is a much more stable compound than oxyhæmoglobin. Blood thus changed is unable to take up oxygen; consequently internal respiration is arrested and death takes place from asphyxia.

Carboxyhæmoglobin is bright red in colour and yields a two-banded spectrum like that of oxyhæmoglobin, except that the bands are slightly nearer the violet end of the spectrum (*see SPECTROSCOPE IN MEDICINE, Plate*). A reducing agent such as ammonium sulphide, which changes the spectrum of oxyhæmoglobin to that of reduced hæmoglobin, produces no alteration in the spectrum of carboxyhæmoglobin; in other words, carboxyhæmoglobin is irreducible, which accounts for the blood retaining its bright colour, although death is due to asphyxia. Death usually occurs before the whole of the hæmoglobin is converted into carboxyhæmoglobin, and recovery is possible if there is enough free hæmoglobin left to carry on internal respiration until the carbonic oxide is dissociated from the rest of the hæmoglobin; for, although a stable compound, carboxyhæmoglobin passing through lungs respiring pure air tends slowly to part with its carbonic oxide, the hæmoglobin thus liberated regaining its oxygen-carrying function.

SYMPTOMS.—A sensation of heaviness in the head, dizziness, noises in the ears, oppression in the chest, vomiting, muscular feebleness, followed by complete relaxation of all the muscles, including the sphincters, drowsiness merging into coma, hyperæmic conjunctivæ, dilated insensitive pupils, cold surface, small thready pulse, and frequently froth on the lips, are the chief results of a fatal dose of carbonic oxide. Convulsions and vomiting not infrequently precede death.

POST-MORTEM APPEARANCES.—*Externally.*—The appearance is very characteristic: the whole body is more or less light red in colour, almost the colour of a child with scarlet fever; the *post-mortem* stains, instead of being dull red or slate-coloured, are a bright pink. Cadaveric rigidity is well marked and passes off slowly.

Internally.—The blood is cherry-red in colour and is fluid; the viscera generally are much lighter in colour than is usual, especially the intestines, which are characteristically pink. The lungs may be œdematous, and the trachea and bronchi may contain froth. The blood and the tissues resist putrefaction for a very considerable time.

Carboxyhæmoglobin may be recognised by its spectroscopic appearances. Any hæmoglobin, however, which remains free undergoes reduction as usual, the result being that the persistent bands of carboxyhæmoglobin are superimposed on the broad band of reduced hæmoglobin. If some of the blood in a test tube be diluted with 20 times its volume of water, and an equal volume of a solution of sodium hydrate (sp. gr. 1.34) be added, a bright red colour is produced; normal blood similarly treated yields a dirty brown colour.

TREATMENT.—The elimination of the poison is effected through the lungs; but, the process being slow, prolonged artificial respiration is required, along with the inhalation of oxygen. External warmth should be maintained and stimulants administered, either brandy by rectal injection or other subcutaneously. Bleeding to a limited amount, followed by transfusion of defibrinated human blood, has occasionally proved successful.

J. DIXON MANN.

☒ CARBONIC OXIDE, Chronic Poisoning by.

Cases of chronic poisoning by carbonic oxide may occur among individuals who work for long hours in imperfectly ventilated rooms heated by slow-combustion stoves; among furnacemen and stokers; among workmen engaged in the production of water-gas; and, since the introduction of water-gas as an illuminant has become so common, in ordinary dwelling-houses where there is very slight leakage from the gas-fittings.

SYMPTOMS.—The early symptoms are headache, neuralgic pains, anæmia, breathlessness, along with general indications of defective nutrition. Later on peripheral neuritis may supervene.

TREATMENT.—Removal from the influence of the poison with general symptomatic treatment will fulfil all the indications.

J. DIXON MANN.

CARBUNCLE (*carbunculus*, a small coal—*carbo*).—**SYNON.** : Fr. *Anthrax*; Ger. *Karbunkel*.

DEFINITION.—A carbuncle is an inflammatory swelling of variable size commencing in the pilo-sebaceous follicles, spreading into the adjacent skin and subjacent subcutaneous tissue, and causing gangrene of a part of the above structures. It is accompanied by general symptoms which are often serious. A carbuncle is essentially an accumulation of boils aggregated together and complicated with local inflammation of the subcutaneous tissue.

ÆTIOLOGY.—Carbuncles are more commonly met with in the male sex and in old age; they are rare in infancy and childhood. Local irritation plays an important rôle. A carbuncle may originate in a furuncle, especially in cachectic states. Cachexia predisposes to carbuncle by lowering the resistance of the tissues to invasion by the *Staphylococcus pyogenes aureus*. Fatigue, over-indulgence in alcohol, and Bright's disease are predisposing causes. The chief predisposing cause, however, is diabetes mellitus. Indeed the diabetic condition is often first discovered on testing the urine for sugar in a patient suffering from carbuncle. It must be remembered, however, that temporary glycosuria may be associated with a carbuncle.

SITE.—Carbuncles may be found on any part of the body, particularly where the skin is thick, as on the nape of the neck and the back. Carbuncles are generally single, but they are frequently sur-

rounded by a circle of daughter-furuncles due to infection with staphylococci contained in the purulent discharge.

SYMPTOMS.—Malaise, anorexia, headache, and slight febrile disturbances may precede the discovery of any local lesion. In a short time, however, a swelling, acutely painful on pressure, dusky-red in colour, and of brawny hardness, is seen in the skin. At first this swelling presents the appearance of a large boil, but it increases more rapidly in size and prominence while a purplish hue develops in its centre. A little later, vesicles appear on the surface; these contain blood-stained serum, but this is quickly replaced by pus. The pustules rapidly burst, and the true skin is thus exposed, dotted with yellowish purulent spots which soften and leave orifices honeycombing the skin and discharging pus. At the bottom of these openings a greenish-white slough is generally visible. Pain is a nearly constant symptom. It is usually lancinating and frequently so severe as to prevent sleep, thus markedly aggravating the patient's condition.

VARIETIES.—Two distinct types of carbuncle may be described:—(1) Small or medium-sized circumscribed carbuncles.—These vary in size from that of a grape to that of a small orange; they have a well-defined margin and the sloughs usually separate in small shreds. With the separation of the sloughs the gangrenous process ends and healing commences, the malaise and fever rapidly disappearing.

(2) Large diffuse carbuncles.—These indicate a condition of extreme gravity and are accompanied by severe local symptoms and marked constitutional disturbance. From the first there is no tendency for the carbuncle to become defined, as in the more benign form. It seems to be constantly extending its periphery, continuously invading the neighbouring structures. Such carbuncles may cover a superficial area of 100 to 150 square inches, and may thus involve the greater part of the back, reaching, in extreme cases, from the occiput to the lumbar region, and laterally from one acromion process to the other. Even after the greater part of the slough has been discharged spontaneously, or has been removed surgically, extension of the infective process may still occur at the periphery of the carbuncle. In such cases, huge sloughs, consisting of subcutaneous tissue and even in some cases pieces of deep fascia, may be extruded through the crater-like opening. The deep fascia, however, generally forms an efficient barrier restraining the further spread of the disease. In these diffuse carbuncles the temperature may range from 104° to 105° F. Drowsiness and coma or delirium are not infrequent. In the worst cases pyæmia or septicæmia may supervene.

Not infrequently, even after the separation of the slough, death results from the prolonged suppuration and exhaustion. Large sloughs require many days to separate spontaneously, and the healing process is at best slow and tedious. Six weeks to four months, according to the size of the carbuncle, will be required before the healing process is complete. When, however, healing has taken place, the resulting scar is generally much smaller than would have naturally been anticipated.

The carbuncle commonly met with in diabetic subjects is of the gravest danger. It is generally of the large diffuse variety; its onset is sometimes gradual and insidious; and, even when the swell-

ing is very large, the fever and pain are generally only moderate in degree. The gangrenous and ulcerative processes are always extensive, and continuous infection of the margins of the carbuncle is frequently seen. Healing either does not take place or is very much retarded.

Among the most frequent complications may be mentioned erysipelas, infective spreading cellulitis, septic phlebitis (especially serious in carbuncle of the face), and suppuration either around or underneath the carbuncle, or in other parts of the body (pyæmic abscess).

PROGNOSIS.—This depends upon the nature of the lesion, whether small and circumscribed, or large and diffuse; upon the presence or absence of complications; and very largely upon the general condition of the patient and the existence of any of the predisposing causes.

The mortality in small circumscribed carbuncles, in the absence of any dyscrasia and of any complications, is low; but it is quite otherwise in large diffuse carbuncles. In these the mortality is at least 10 per cent. in non-diabetic cases, and 35 per cent. in those suffering from diabetes. In the markedly gangrenous type occasionally met with in diabetics, death results almost invariably.

DIAGNOSIS.—In its early stages a carbuncle may be mistaken for a boil, and indeed often commences as such. The essential difference between the two conditions is that the inflammatory induration is subcutaneous in a carbuncle, not purely cutaneous as in a furuncle.

TREATMENT.—*General.*—To combat the pain and to enable the sufferer to obtain sleep, opiates are generally necessary.

In diabetics codeine in doses of half a grain every six hours is preferable to opium. During the febrile stage ample fluid nourishment, e.g. milk, chicken-broth, meat-essences, and gruel, must be given, and these should be peptonised if digestion is much impaired. During the separation of the sloughs and the healing of the resulting surface tonics and generous diet are required. When the patient is passing into a poisoned adynamic condition, liberal stimulation with alcohol is urgently called for. Alcohol, however, is best avoided in the early febrile painful stage, as it generally increases the sufferings of the patient.

Local.—In the early stages abortive treatment may be tried, but owing to the inflammatory process having its seat largely in the subcutaneous tissue, abortive treatment is not nearly so effectual as in furunculosis, in which the condition is purely cutaneous. Good results are, however, generally obtained by smearing the surface of the inflammatory swelling with the following ointment: *R*, acidi carbonici cryst. gr. xx, extract. ergotæ liquid. ʒj ss, zinci oxidi ʒj ss, lanolin ad ʒj. Even if this fail, as it generally does, to arrest the carbuncle, it lessens the pain and inflammatory congestion (see BOILS). Warm antiseptic fomentations may also be employed. The application of caustics is always to be condemned.

The local surgical treatment of the majority of carbuncles, in which it is clear that suppuration has occurred or is about to occur, consists in either *excision* or *incision*.

Excision may be employed for circumscribed carbuncles not exceeding an orange in size. An oval or circular incision is made outside the diseased margin, and the mass is dissected out down to the deep fascia. The risk in this opera-

tion is that of infection of the wound ; hence it is essential that the mass be not burst or cut into during the excision, and that the raw surface be painted with a strong antiseptic, preferably with liquefied pure carbolic acid. An antiseptic dressing is then applied, and later on skin-grafting after Thiersch's method may be necessary. Little loss of tissue is occasioned by excision, as the greater part of the skin and subcutaneous tissue removed would in all probability have sloughed. The advantage of the method is that in successful cases, i.e. those in which infection of the raw surface is avoided, the pain disappears at once and the patient is saved from the suppuration and risk of septic complications which a carbuncle frequently entails.

Incision, preferably combined with the cutting away and curetting of all the infected and sloughy tissues, is the method of treatment commonly adopted in all large carbuncles, and in those of medium size which are not well circumscribed, i.e. have no definite indurated margin.

The incisions may be crucial or multiple according to the dimensions of the carbuncle. They must in all cases extend to the extreme periphery of the carbuncle and traverse the entire depth of the infected area. Incision alone relieves tension and facilitates the discharge of pus and sloughs, but it should be in all cases combined with the removal of the sloughs and infected honeycombed skin. This removal is often a tedious matter, but by the use of scissors and curette the entire necrotic tissue can generally be removed. The bleeding may be free, but is always arrested by pressure. The large raw wound must now be freely painted with pure carbolic acid and stuffed with antiseptic gauze wrung out of 1 to 30 carbolic solution or 1 to 2,000 biniodide of mercury lotion. Until the wound has cast off all its slough and has assumed a clean granulating appearance, it is necessary to dress it daily with gauze and to wash the surface with an antiseptic solution. To hasten the process of cicatrization, skin-grafting by the method of Thiersch will frequently be found necessary.

In certain cases of carbuncle, in which the general symptoms are very slight and the pain is very moderate, no operative treatment is called for ; the carbolic acid and ergot ointment, or hot antiseptic fomentations, may be applied, and the carbuncle allowed to extrude its sloughs spontaneously. Carbuncles on the face, owing to the gravity of the complications (septic phlebitis, meningitis, orbital cellulitis, &c.) which frequently attend them, call for immediate incision and disinfection of the purulent focus of infection (*see BOILS*).

HERBERT F. WATERHOUSE.

CARCINOMA.—*See* CANCER.

CARDIAC DISEASES.—*See* HEART.

CARDIALGIA (*καρδία*, the heart ; and *ἄλγος*, pain).—A synonym for heartburn, originating in a popular impression that this painful sensation, which starts from the epigastrium, is connected with the heart. *See* HEARTBURN.

CARDIOGRAPH (*καρδία*, heart ; and *γράφω*, I write).—This is an instrument for recording the impulse of the heart against the chest-wall. The graphic tracing so obtained is known as a *cardiogram*. Chauveau and Marey introduced this method

of observation in 1861. The principle is as follows : a small button applied over the so-called apex-beat transmits the variations of pressure which it receives to the rubber membrane of a small air-tight capsule. This receiving tambour, as the capsule is called, is connected by rubber-tubing to a recording tambour, upon the rubber-membrane of which is attached a lever. Every movement of the button is thus transmitted by the air enclosed in the apparatus to the lever, which records the magnified movement upon the smoked paper of a revolving cylinder.

In France the term 'cardiograph' is applied also to a form of the instrument so modified that the small recording tambour can be introduced through the carotid artery or the jugular vein of a horse or dog, into the aorta and left ventricle, or into the right auricle and right ventricle, as the case may be. The graphic records of the pressure in these different chambers of the heart are of value in the interpretation of the tracing obtained with an ordinary cardiograph applied to the chest-wall.

The curve obtained with the cardiograph has been the subject of much discussion, but most authorities give a curve similar to fig. 1. It consists of (1) a very steep ascending limb, (2) a plateau with three small waves, and (3) a very steep descending limb. The slight undulation *a-b* is due to the contraction of the auricles forcing blood into the ventricles. The steep ascent *b-c* is caused at its beginning by the filling, and then by the contraction of the ventricles, which is maintained until *e*, when the aortic and pulmonary valves are closed. The steep descent *e-f* is due to the relaxation of the ventricles. The opening of the sigmoid valves occurs according to some authorities at the point marked *, according to others at *c*. The first sound of the heart begins soon after the rise at *b* ; the relation of the second sound to the cardiographic curve is doubtful : some state that it begins at *e*, others at *f*. The cause of the undulations upon the systolic plateau *c-e* is uncertain, but they are not due to imperfections in the recording instrument. Roy and Adami think that they are caused by a want of synchronism in the contraction of the different parts of the ventricle. Marey holds that they are due to retrograde waves from the aorta towards the left ventricle.

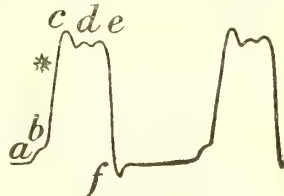


FIG. 1.—Curve of the impulse of the heart of man (after Marey)

The cardiogram is a complex curve ; it represents not only changes due to the contraction of the cardiac muscle, but also changes in the ventricular blood-pressure and in the volume of the heart. Further, it is necessary to remember that the position of the heart in relation to the chest-wall varies during the different phases of the cardiac cycle, and that the resistance and thickness of the intercostal spaces differ even in healthy subjects.

According to Marey and his followers the cardiogram is a curve of the intra-ventricular pressure, but this view has been contested by many physiologists and physicians in this country and in Germany.

The practical value of the cardiograph in clinical medicine is very limited, and it must be confessed

that the tracings obtained by its use are liable, owing to the difficulties of interpretation, to lead to erroneous conclusions. Further, there are great difficulties in obtaining typical tracings even in perfectly healthy men; in many cases, owing to the conformation of the chest, the development of muscle or adipose tissue, or extensive overlapping of the lung, the apex-beat is obscured and does not give sufficient impact to yield a typical curve. Another source of difficulty often experienced is the movement of the chest-wall in respiration. Roy and Adami state as the result of their numerous experiments that 'the cardiographic curves from man have not as yet been found fitted to give much information regarding the action of the heart either in health or in disease.'

The chief value of the cardiograph is in the determination of the beginning and end of the ventricular systole. It can also be used for recording the pulsation of aneurysms, and for this purpose it is sometimes advisable to replace the receiving tambour by a glass funnel, which can be pressed down upon, and made to enclose the area of skin under which the pulsation is observed.

M. S. PEMBREY.

CARDIOLITH.—See CONCRETIONS.

CARIES (*caries*, rottenness).—A destructive inflammatory disease of bone, analogous to the ulceration of soft tissues. See BONE, Diseases of.

CARLSBAD, in Bohemia.—Thermal alkaline sulphated waters. See MINERAL WATERS.

CARMINATIVES (*carmino*, I card, or cleanse). DEFINITION.—Substances that aid the expulsion of flatus from the stomach and intestines, and relieve griping.

ENUMERATION.—The principal carminative remedies are—the Essential Aromatic Oils, for example, Peppermint and Cloves; Chloroform; Charcoal; Ethers; and Camphors, and substances containing them.

USES.—The uses of carminatives are sufficiently indicated in the preceding definition. They are extensively administered in cases of flatulent dyspepsia, especially when it is associated either with disease or disorder of the heart, or with a nervous or hysterical state of the system. A combination of several different carminatives is usually more successful than the exhibition of a single drug, one of the best being a mixture containing Spirit of Ether, Aromatic Spirit of Ammonia, and Tincture of Orange. Given along with antacids they are useful in correcting acidity, as in the favourite Soda-mints; and they are frequently prescribed with purgatives to prevent pain—an application illustrated by the official Compound Rhubarb Pill.

T. LAUDER BRUNTON.

CARNIFICATION (*caro*, flesh; and *fio*, I become).—A condition of the lung in which it resembles flesh. The term was formerly applied to the transformation of any tissue into a flesh-like substance. See LUNGS, Collapse of.

CARPHOLOGY (*κάρφος*, chaff; and *λέγω*, I collect).—The movements of the hands and fingers observed in certain delirious patients, as if they were searching for or gathering imaginary objects. A familiar illustration of the act is 'picking of the bedclothes.' See TYPHOID FEVER.

CARPO-PEDAL CONTRACTIONS.—See TETANY.

CARRION'S DISEASE (*Verruga Peruana*).—This name is applied to a remarkably infectious disease, usually characterised by the formation of multiple granulomatous tumours on the skin. Its course in such cases is chronic or subacute, but it may also run an acute course and frequently terminate fatally.

At present it has been mentioned as occurring only in the moist valleys of the Andes of Peru, at an altitude of from one thousand to three thousand metres. On the Oroya Valley Railway Extension, 50 per cent. of the engineers and 75 per cent. of the European navvies died of the disease. The disease appears to be very strictly limited to these localities.

Three clinical types are described: the simple eruptive form, the rheumatic form, and an acute form which may kill the patient in a few days, before the eruption has had time to appear. The last type has also been described as Oroya Fever. The identity of the chronic verrucous form and Oroya Fever was established when Daniel Carrion, a Peruvian student, inoculated himself with *verruca*, the lesion of the chronic type, and died in nineteen days from the acute form of the disease.

Careful investigations have been carried out both in the benign and in the malignant types of the disease. Several organisms have been described in the wartlike granulomata, but no specific organism has yet been isolated.

This malady affects persons of all ages; and animals, such as mules, suffer from a slightly specialised form of the disease.

JAMES GALLOWAY.

CARTILAGE, Diseases of.—SYNON.: Fr. *Maladies du Cartilage*; Ger. *Knorpelkrankheiten*.

Cartilage being generally non-vascular, its inflammation is of a modified type, and may present degenerative changes as the result of impaired nutrition. In uncomplicated disease of cartilage there is no inflammatory exudation, and when lymph or pus is found in a joint, it is obvious that other structures have become inflamed. In those cartilages in which vessels are found, e.g. rib-cartilages, true central inflammation may occur. When cartilage has been destroyed it may be replaced by fibrous tissue or bone, but it is never reproduced.

1. The ensiform and costal cartilages, with those of the trachea and larynx, show a great tendency to *ossification*, as the result of senile changes; they are also liable to *necrosis*. The articular cartilages never ossify, but large or small portions of them may perish and be detached, in consequence of some interference with their supply of nutriment from the subjacent bone. In pyæmic joint-effusions the cartilage softens, and is rubbed off the most prominent portions of the articular surfaces in a manner quite characteristic. In osteo-arthritis, one of the earliest of the pathological changes occurs in the articular cartilage. The cells proliferate, the primary capsules enlarge and fill with secondary capsules, and the matrix fibrillates and softens. By degrees the cartilage is worn off the central part of the articulation, exposing the bone underneath; laterally, where the pressure and friction are less, the cartilage-cells proliferate and form a tuberculated margin, which subsequently ossifies.

2. The cartilages of the epiglottis, ears, nose, eyelids, and Eustachian tube are liable to *ulceration*, especially of the syphilitic variety; in these cases the disease commences in the skin or mucous membrane, and spreads to the cartilage by contiguity.

3. The cartilage of the external ear is often the seat of 'chalk-stones' in gouty persons, and similar *deposits of urate of sodium* are found in the articular cartilages.

4. The epiphysal cartilage may be the seat of *inflammatory changes*, generally very acute, which lead to separation of the shaft from the epiphysis, a condition which, whether the result of disease or accident, is of great moment, inasmuch as the destruction of this layer of cartilage may check further growth at this end of the bone.

Epiphysitis may occur as a primary disease: in other cases inflammation may spread from the extremity of the diaphysis. Syphilis, especially of the inherited form, is the most common cause of this disease. Injury, particularly in strumous children, is also a factor in its production. The acute form is often associated with general septicæmia, and is followed by destructive changes in the adjacent joint. In children who suffer from the syphilitic form of the disease, the joint is generally preserved and the subsequent growth of the bone little impaired.

5. Cartilage is not primarily attacked by *cancer*, but it may become involved by the spread of a malignant tumour. The *epithelial* form of cancer not infrequently extends from the mucous or cutaneous surface, in which it originated, to the subjacent cartilage.

6. The articular cartilages are liable to certain structural changes as the result of disturbed nutrition; and the fibro-cartilages are also subject to the same abnormal conditions.

Ulceration, absorption, degeneration of cartilage, are terms used to denote a series of destructive changes which take place in the substance of articular cartilage, and lead to its partial or complete removal. These changes may originate in the cartilage itself, or they may be secondary to disease of the bone or synovial membrane; however this may be, the morbid action is the same, and consists in increased cell-development, with disintegration of the hyaline substance. The cartilage-cells become enlarged, filled with nucleated corpuscles, and arranged irregularly. The matrix softens; and in acute cases rapidly disintegrates and is discharged: when the disease is more chronic, it splits up into fibres which remain attached by one end to the cartilage, and at the other project loosely into the interior of the joint, giving a villous appearance to the affected spot. The remains of the cartilage are ultimately converted into fibrous tissue, and constitute the sole medium of repair when a cure is effected. The different appearances which the affected cartilage presents under different circumstances have led to the several terms *fibrous*, *fatty*, and *granular degeneration* being applied to this disease, in the belief that they were really distinct pathological conditions. Changes of this kind may be primary, as in osteo-arthritis, but they are commonly secondary to disease in the articular ends of the bones, the result of syphilis, tuberculosis, or injury. See JOINTS, Diseases of.

W. MAC CORMAC.

W. CECIL BOSANQUET.

CASEATION (*caseus*, cheese).—This name is applied to a peculiar process by which masses of tissue are converted into a yellowish-white, pulpy material, somewhat resembling cream-cheese. It is seen chiefly in tubercular foci, but a similar degeneration is met with in the central portions of gummata, and in nodules of carcinoma and sarcoma: it may also be found in connection with some of the rarer irritant organisms, such as the actinomyces, the bacillus of pseudo-tuberculosis, and certain moulds (aspergillus). The tubercle-bacillus appears to secrete a peculiar acid substance which has the power of causing necrosis of tissues: the process is assisted by the tendency to fatty degeneration that exists in the centre of a tubercular mass, owing to the blood-supply being cut off by contraction of the fibrous tissue formed around it. The occurrence of caseation in individual tubercles before fibrosis has occurred, shows that the action of this latter cause is not indispensable. In gummata the nutrition of the central portions is defective owing to narrowing of the blood-vessels by syphilitic endarteritis. In tumours the growth of cells is so rapid that the mass outgrows its blood-supply, while it is possible that the cells thus rapidly and irregularly formed are themselves of deficient vitality. The affected cells become first granular, then fatty, and finally all structure in the part is lost and the tissue is converted into a mass of soft amorphous debris. At the periphery of the caseous portion some free nuclei may be seen lying amidst the degenerative products, since these bodies resist the process of disintegration longer than the protoplasm of the cells. The caseous material consists of fatty granular particles, leucocytes, cholesterin-crystals, and a certain amount of fluid exuded from the lymphatics. The fate of caseous foci varies: in some instances they dry up and become infiltrated with calcium-salts, giving rise to masses of stony hardness; in others softening occurs and the caseous mass is converted into a milky fluid somewhat resembling pus. Cavities filled with this material are known as *chronic* or *cold* abscesses. Caseous masses are frequently met with in the bronchial, mesenteric, and cervical lymphatic glands, and in the lungs, brain, and bones. In the great majority of cases they are due to tuberculosis, but the existence of caseation is not by any means pathognomonic of lesions due to this cause.

W. CECIL BOSANQUET.

CASTS (derived from the Middle English word *casten*, which is adapted from the Old Norse *kasta*, to cast or throw).—SYNON.: Fr. *Cylindres*; Ger. *Cylindern*.

DEFINITION.—A term applied to moulds of gland-tubules and hollow viscera thrown off in certain states of disease.

CLASSIFICATION.—The varieties of casts met with may be thus grouped:—

A.—CASTS OF GLAND-TUBULES.

- | | |
|-------------------------------|-------------------|
| I. Of the Uriniferous Tubules | Blood-casts. |
| | Leucocyte-casts. |
| | Epithelial casts. |
| | Granular casts. |
| | Fatty casts. |
| | Waxy casts. |
| | Hyaline casts. |
| | Cylindroids. |
| | Bacterial casts. |
| | Pseudo-casts. |

- II. Of the Seminal Tubules.
- III. Of the Gastric Tubules.
- IV. Of the Bile-ducts.
- V. Of the Cutaneous Glands.

B.—CASTS OF HOLLOW VISCERA AND PASSAGES.

- I. Of the Alimentary Canal.
- II. Of the Urinary Bladder.
- III. Of the Female Genital Passages.
- IV. Of the Respiratory Passages.

A.—CASTS OF GLAND-TUBULES.

I. Of the Uriniferous Tubules.—Though it is known that renal casts may be formed in any part of the kidneys, their exact mode of origin is still unsettled. According to one view they are formed by exudation of the blood, and due to the rupture of engorged vessels; according to another by transudation of blood-plasma, which in its passage through the renal epithelial layer undergoes some modification of composition; a third speculation is that they are due to a secretion from the cells of the tubules which have undergone some pathological change; while a fourth ascribes them to a fusing together of degenerated epithelial cells. Thus, in the last two instances the cells are directly, in the second indirectly or less directly, and in the first not at all concerned in the production of casts. It is quite possible, however, that these bodies may be derived in all the four ways; but though it is customary to distinguish several modes of origin it will be found that more than one kind of cast is nearly always present in the same urine, and that different sorts are connected by transitional gradations.

None of these views, however, explains very satisfactorily why casts which give the 'amyloid' reaction may exist alike with and without lardaceous change in the viscera; nor do they account for the presence of hyaline casts in non-albuminous urine and under anephritic conditions. Moreover, there is not complete agreement as to nomenclature; for while some writers distinguish between hyaline, colloid, waxy, fibrinous, and lardaceous casts, holding them to be of different composition and origin, others seem inclined to place all those cylinders having a homogeneous structure and a more or less transparent appearance in one category. The term 'granular cast' is somewhat unfortunate, for it includes casts which are composed of granules (intrinsic granulation) and casts covered with granules (extrinsic granulation). Very similar remarks apply to fatty casts: every kind of cast is prone to exhibit fat-globules, but it is only when they are a very prominent feature that the term 'fatty' becomes an appropriate designation. Practically, however, there is unanimous agreement that casts are indicative of abnormal states of the kidneys, and are therefore of great clinical importance.

In shape renal casts are mostly cylindrical, but not infrequently are bent and occasionally forked. In length they vary considerably: often quite short and stumpy, they not infrequently stretch right across the field of the microscope. In diameter they mostly range between $\frac{1}{1000}$ and $\frac{1}{500}$ inch, the majority being of medium size, i.e. about $\frac{1}{700}$ inch.

Castes are composed of an albuminous or of an albuminoid matrix, within which may be embedded epithelial cells and blood corpuscles, crystals (e.g. oxalates, uric acid), oil-globules, and other particles, and

upon the surface of which may be deposited minute crystals, granular debris, oil-globules, pigment, and cells.

When microscopic sections of diseased kidneys are examined, tubules may be seen to contain red corpuscles, whole or broken up; leucocytes; free epithelial cells, globose and otherwise altered; granular debris and crystals; but more frequently the contents of the tubules are cylinders composed of a homogeneous substance, which in appearance is more or less transparent or faintly granular. These moulds often exhibit colour-reactions similar to those of 'amyloid.' In connection with epithelial casts it may be mentioned that sections of the kidney in acute nephritis do not exhibit the loss of epithelium that might be expected from the ordinary inspection of the urinary sediment, and this leads to the suspicion that many of these cell-casts are really leucocytic. In some cases of nephritis, however, the appearance of some of the tubules, owing to degenerative changes in the epithelium, presents a striking resemblance to granular casts. It is difficult to estimate the value of this observation, and too much importance should not be attached to it. Sometimes one cylinder inside another may be observed *in situ*.

Blood-casts consist mainly of red corpuscles included in a fibrinous basis. They are indicative of acute nephritis (primary or secondary) and hæmorrhagic conditions of the kidney. In hæmoglobinuria casts of disintegrated corpuscles may be present. Blood-casts are of a yellowish colour and often have a coarsely granular appearance. See fig. 1.

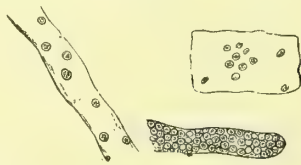


FIG. 1.—Blood-casts.

Leucocyte-casts are composed of agglomerations of white blood-corpuscles, and when in the condition known as pus their presence is indicative of a suppurative process, but casts from acute and subacute nephritis frequently contain large numbers of leucocytes.

Epithelial casts are such as contain epithelium from the tubules, and their presence is indicative of a desquamative nephritis. The condition of the epithelial cells is very variable; often of fairly normal appearance, they are sometimes so de-



FIG. 2.—Hyaline casts.

generated that they are scarcely recognisable as such, and rather resemble a cylindrical aggregate of granules of variable size. Many cell-casts which if inspected in the unstained condition present appearances resembling epithelium are really leucocytic. See fig. 3.

Granular casts.—Under this term are included casts which exhibit a finely or coarsely granular

appearance. It includes casts composed of broken-up blood-corpuscles, degenerated epithelial cells, and casts containing fatty globules. A granular appearance is also exhibited when a hyaline matrix is covered with granular debris, minute crystals, fat-globules or crystals. Their diagnostic value is therefore variable and depends on their origin, but in most cases their presence is of unfavourable import and is indicative of nephritis. See fig. 5.



FIG. 3.—Epithelial casts.

Fatty casts are such as contain fatty globules or upon which oily spherules and fat-crystals are deposited. The globules may be few or many and are of variable size. If numerous, the casts have a granular appearance. They are found in subacute and chronic nephritis, and are of unfavourable import when they can be assigned to a condition of progressive deterioration, but on the other hand fatty degeneration in casts may be indicative of resolution. See fig. 4.

Waxy casts, sometimes also called *colloid*, are homogeneous, refracting cylinders frequently of large



FIG. 4.—Fatty casts.

size and sometimes giving the 'amyloid' reaction with iodine and methyl violet. They are most frequent in chronic nephritis where there is much albumen in the urine. The 'amyloid' reaction of these casts, however, is not a sure indication of the lardaceous change in the viscera, for it may be present when there is no lardaceous degeneration, and *vice versa*.

Hyaline casts are highly transparent and but little refractive cylinders of small to medium size. They may be found in most cases of Bright's disease, most

health and of disease, and most frequently in that of children. Whether they are of renal origin is uncertain, and albuminuria does not always accompany them.

Bacterial casts.—Casts consisting of micro-organisms have been met with in septic affections of the kidneys. They are distinguishable from other granular casts by their staining-reactions with aniline dyes, and by the regular shape and size of their constituent elements.

Pseudo-casts.—This term has been applied to certain artificial or accidental aggregates of minute crystals (e.g. urates) or granular debris which from their size and cylindrical shape simulate the appearance of renal casts.

Casts containing hæmatoidin, bile-granules, and indigo are occasionally met with.

CLINICAL SIGNIFICANCE.—In regard to diagnosis and prognosis there are but few considerations which discount the importance of casts in the urine as a criterion of disease of the kidneys. It is true, however, that hyaline casts have been observed in non-albuminous urine after severe physical exertion, in cases of protracted jaundice, in lithuria, oxaluria, and diabetes mellitus. They may occur too in conditions of transitory congestion of the kidneys, and one observer has recorded their appearance after epileptic fits. In instances of this description there may be no other symptoms or evidence to point to organic disease of the kidneys. Such examples are, however, fairly uncommon. As a rule casts are accompanied by more or less albumen, and their presence affords valuable assistance both as regards diagnosis and prognosis. It would not, however, be a warrantable deduction that the gravity of any given case was commensurate with the number of these cylindrical excreta. For in some cases casts are few and the organic change in the kidney is great; and on the other hand they may be many, though the prognosis will be favourable. For example, in advanced contracted granular kidney the casts are often few; in acute nephritis they are often numerous and may even exhibit the appearances of fatty degeneration. It is a question less of number than of kind, though of course numbers are unfavourable where the kinds are indicative of severe and permanent damage. Moreover, the number of casts may have little relation to the quantity of albumen, for the amount of albumen may be large and the number of casts few—a point which has some bearing on the mode of origin of these bodies. It must also be borne in mind that one disease or one kind of morbid change in the kidneys does not produce merely one kind of cast, for in the same urine, whether from acute or chronic nephritis, two or more kinds of casts may be found. So the diagnosis of the present and the prognosis of the future condition of the organs must be made from a sort of rough average struck between the various kinds of casts, their relative importance, and their increase or decrease as time goes on.

It is always necessary to examine the urinary sediment for casts on several, indeed frequent, occasions, not only to be satisfied as to the particular class of cast, but also to ascertain whether they present indications of improvement or the contrary. And in connection with these examinations it may be laid down that for practical purposes casts may be placed in three categories according as they indicate *Acute* or *Chronic* disease or an acute attack



FIG. 5.—Granular casts.

often in contracted granular kidney, but have been observed in non-albuminous urine and under conditions when nephritis could be excluded. See fig. 2.

Cylindroids.—The name 'cylindroids' has been given to certain long, colourless, ribbon-like bodies which may have some resemblance to hyaline casts. They are somewhat irregular in outline, are longitudinally striated, and give the mucin-reaction with acetic acid. They have been found in the urine of

supervening on a chronic condition. The first is characterised by the presence of blood-, epithelial, and granular casts; the second by more or less transparent (waxy, colloid, hyaline), and also by fatty and granular casts; while in the third are found casts typical of the acute and chronic conditions, though those characteristic of the latter usually predominate.

For the method of examination for casts of the uriniferous tubules see URINARY DEPOSITS.

II. Of the Seminal Tubules.—Spermatic casts are largish hyaline cylinders composed of a mucoid matrix and containing spermatozoa. They are supposed to arise from the seminal tubules, and their presence is not necessarily associated with albumen in the urine. They are of rare occurrence, and their pathological significance is doubtful.

III. Of the Gastric Tubules.—In inflammation of the gastric mucosa, especially in scarlet fever, a desquamation of the epithelial coat involving the glands has been observed. The casts of the follicles have been found in the vomit, and more abundantly in the contents of the stomach *post mortem*. Their length is variable, and in width they range from $\frac{1}{1000}$ to $\frac{1}{1000}$ of an inch. Their substance is described as being fibrinous and is covered more or less completely with altered epithelial cells and granular debris.

IV. Of the Biliary Ducts.—In the centre of gall-stones have been found long hair-like threads, often branched and generally under $\frac{1}{200}$ inch in diameter, consisting of mucus, inspissated bile, and epithelium, which have been regarded as casts of the smaller biliary tubes.

V. Of the Cutaneous Glands.—In the various skin-affections which are associated with desquamation of the cuticle, casts of varying length, coming from the sweat and sebaceous glands, are thrown off as part of the general shredding of the epidermis. Such bodies are hollow tubes and bear no resemblance to the renal casts in nephritis.

B. CASTS OF HOLLOW VISCERA AND PASSAGES.

I. Of the Alimentary Canal.—Solid casts of the tonsillar crypts and branches, consisting of inflammatory products, are occasionally expectorated in follicular tonsillitis and diphtheria.

More or less perfect muco-epithelial casts of several inches in length coming from the œsophagus have been met with, and more rarely pieces from the stomach have been detected in the vomit. In cases of corrosive poisoning, should the patient tide over the first effects, more or less complete casts of the œsophagus and stomach may be ejected. Casts of portions of the gullet, formed of dense masses of aphthæ, have also been described.

Intestinal casts and small flocculent pieces from the surface of the colon are far from uncommon, especially in association with chronic constipation. The casts consist of viscid mucus and epithelial cells, matted together into tough coherent pseudo-membranes; they often form complete hollow moulds of the tube, large or small, and vary in length from a few inches to several feet. Occasionally they retain on their surface the impression of the follicles, sacculations, and other inequalities of the intestinal surface.

II. Of the Urinary Bladder.—A complete exfoliation of the mucous membrane of the bladder

has been occasionally observed in puerperal women. It does not appear to be always the result of inflammation, and though detachment may be complete, perfect recovery may follow. In structure such bodies consist of epithelial cells in varying stages of degeneration, felted together by mucus and fine granular material. The surface is frequently thickly coated with urinary salts. The conditions giving rise to their formation are quite unknown, though retention of urine is associated with their occurrence. A few instances of moulds of the ureter and of the urethra have been recorded.

III. Of the Female Genital Passages.—Casts of the uterus and vagina have been frequently noticed. The latter occur as more or less complete hollow moulds of the canal or as membranous plaques of variable size (membranous vaginitis). They are greyish-white in colour, often translucent, are fairly smooth on both sides, and are composed of layers of squamous epithelium. They have been known to follow the use of astringent injections, and cases are recorded of their occurrence at the menstrual periods, and without obvious exciting cause.

Partial casts of the vagina formed of diphtheritic membrane have also been seen.

Uterine casts are of commoner occurrence, and are of various kinds. Exclusive of solid moulds of the cavity, formed of coagulated blood and of fibrin, the states of pregnancy and menstruation are each liable to give rise to the formation of membranous casts of the surface, complete or partial, usually accompanied by much pain in their extrusion (membranous dysmenorrhœa). The membranous cast may be entire, in which case it resembles a triangular bag, measuring from $1\frac{1}{2}$ to $1\frac{3}{4}$ inches in length, by about 1 inch in breadth, and from $\frac{1}{16}$ to $\frac{1}{8}$ inch in thickness. More frequently, however, the membrane comes away in pieces of varying size. The external surface is smooth and the internal somewhat shaggy, and on microscopical examination shows the ordinary structure of the uterine mucosa, in which are plainly visible the characteristic gland-follicles and some blood-vessels. Scattered throughout its texture are often seen leucocytes and small round cells.

Sometimes a uterine cast is the result of an early abortion and may present appearances closely resembling those of a dysmenorrhœal membrane. But in addition to being thicker, microscopical examination may reveal the presence of large decidual cells and chorionic villi. If no villi be found it is not advisable to give a definite opinion as to impregnation, for the large decidual cells are not conclusive evidence.

In extra-uterine pregnancy the membrane is distinguishable from that of membranous dysmenorrhœa by being much thicker and by the presence of large decidual cells; and from that of uterine pregnancy in the absence of reflexa and of amnion.

IV. Of the Respiratory Passages.—In diphtheria a false membrane may be formed on the respiratory mucosa, from the larynx downwards. In extent and thickness it may vary from thin soft scattered patches to thick tough complete casts of the tubes, reaching to the finer bronchi.

Such membranes are composed of a network of coagulated fibrin containing bacteria and entangling leucocytes and red corpuscles. According to their thickness, so is the subjacent mucosa more or less involved in a necrotic process. Such false-membranes are frequently expectorated or expelled through the tracheotomy wound, usually as shreds

or ragged masses, though occasionally as more or less perfect tubes.

Fibrinous moulds of the bronchi may be found in the sputum of lobar pneumonia and of plastic bronchitis. Of the latter disease, these 'bronchial polypi' are pathognomonic, and though it is rare for more than very limited areas of the air-passages to be affected, yet perfect casts of the entire extent of these areas from the larger bronchi to the alveoli may be ejected. Expectoated as irregular, rolled-up and twisted masses, they may be shaken out in water into ramifying whitish or pinky-white moulds of the tubes, varying in length from one and a half to six or seven inches, and of a maximum diameter of half an inch, often with swollen ends corresponding to the infundibula. They are either hollow and membraniform or solid, and nearly always present indications of being made up of concentric layers. Sometimes only one of these casts may be expectoated, especially if a large one; at other times they may be re-formed again and again, and large quantities may be coughed up daily. Microscopically they consist of a fibrinous coagulum entangling epithelial cells, leucocytes, red corpuscles, fat-globules, and occasionally octahedral crystals. See BRONCHI, Diseases of.

In hæmoptysis small casts of bronchioles, consisting chiefly of coagulated blood, are sometimes found in the sputum.

Curschmann's Spirals.—In spasmodic asthma and also in pneumonia and bronchitis, spirally twisted bodies of a whitish colour may be seen in the sputum. They are said to be composed of a central slender core of a fibrinoid substance, enveloped in a layer of mucin. They are of variable length and breadth, are frequently covered with epithelial cells, and with colourless elongated octahedral crystals. See CHARCOT-LEYDEN CRYSTALS.

W. H. ALLCHIN.

R. G. HEBB.

CATALEPSY (κατάληψις, a seizure).—SYNON. : Fr. *Catalepsie*; Ger. *Starrsucht*.

DEFINITION.—A disease of the nervous system, characterised by attacks of powerlessness, commonly with loss of consciousness, and accompanied by a peculiar form of muscular rigidity, in which the limbs remain for a time in the position in which they are placed by passive movement. On account of the trance-like state which exists in the attack, the name is sometimes applied to simple trance, but should be restricted to the condition in which there is the peculiar state of the muscular system. The origin of the name is connected with the old spiritualistic pathology, which referred such sudden states to the action of an immaterial agency, and recognised as such only some immaterial agent of personal nature.

ÆTIOLOGY.—Catalepsy may occur at all ages between six and sixty years, and in both sexes, but it is incomparably more frequent in the female sex and in early adult life, at or soon after puberty. It is, in the majority of cases, associated with distinct evidence of hysteria. In other cases, in which no hysterical symptoms have preceded it, the affection may be traced to such exciting causes as give rise to the hysterical paroxysm. Nervous exhaustion is the common predisponent; and emotional disturbance, especially religious excitement, or sudden alarm, or blows on the head and back, are frequently immediate causes. It occasionally appears in the course

of mental affections, especially melancholia, and is said to occur as an early symptom of epilepsy, but it is much more likely that the fits thought to be epileptic were really hysteroid. In an imperfect form it has appeared to be due, in some cases, to paludal poisoning or to other toxæmic states, as chloroform-narcosis. In a few instances meningitis, and other organic cerebral or spinal diseases, have caused a cataleptoid condition; but these cases are too rare and diverse to allow of any inference from them.

SYMPTOMS.—Catalepsy is usually paroxysmal, attacks lasting hours or days being separated by periods of freedom; less commonly a single prolonged attack constitutes the whole affection. In some cases, headache, giddiness, or hiccough has preceded the attack. The onset of the special symptoms is usually sudden, commonly with loss of consciousness. The whole or part of the muscular system passes into a state of rigidity. The limbs remain in the position they occupied at the onset, as if petrified. The muscular rigidity is at first considerable, and movement is resisted; but after a short time the limbs can be moved, and then remain in the position in which they may be placed. The resistance to passive movement is peculiar; it is as if the limbs were made of wax, and hence the condition has been termed *flexibilitas cerea*. The rigidity commonly yields slowly to gravitation. The countenance is usually expressionless. The respiratory movements and heart's action are weakened. Substances placed in the back of the mouth are swallowed, but slowly. The state of sensibility varies; in profound conditions of catalepsy it is lost to touch, pain, and electricity, and no reflex movements can be induced even by touching the conjunctiva, a state of mental trance being associated. In other cases partial sensibility remains, and reflex phenomena may be excited. In rare instances hyperæsthesia is present. In induced catalepsy there is often a peculiar local exaltation of reflex action, so that stroking the skin over a superficial muscle causes an increased contraction of it. This involves a change of posture, in consequence of a definite sensory impression, and an analogous effect may be produced through the special senses, especially hearing; if a ticking watch be brought near the subject's ear, the posture is changed with a start, the head turned towards the object, and the eyes, if previously closed, may be opened, although the cataleptic condition continues as before. Consciousness, although usually lost, may remain, but it is seldom intact, being generally in an obscured condition. The temperature is commonly lowered in a long seizure. The attack may last a few minutes or several hours. Recovery is gradual or sudden; sometimes the patient at first is unable to speak. A sudden respiratory stimulus—such as blowing on the face—will often terminate an attack of induced catalepsy. Sometimes a strange periodicity may be observed in the occurrence of the paroxysms. In the intervals between the attacks, headache, giddiness, or hysterical manifestations may be present, or the patient may feel and seem perfectly well.

PATHOLOGY.—Concerning the nature of the disease there has been much speculation, but little definite knowledge. It may probably be placed between epilepsy and hysteria in the scale of maladies, but nearer the latter than the former, and as regards the nature of its chief feature, it may be regarded as essentially one of the motor manifesta-

tions. But there is also distinct interference with the intellectual processes, and interruption of the connection between the will and the motor centres.

To say this is only to express obvious facts in general terms, and affords no explanation of them. A careful consideration of their nature may give us at least the material for thought on the subject. The wax-like rigidity is an excess of the action by which muscles are constantly adapted to posture, i.e. to the distance between their attachments, and yet preserve the same degree of physiological tone. This is effected by a reflex process, the afferent impulse for which probably comes from the afferent muscle-nerves, and is generated by the sum of the related tension and compression of the interstitial tissue in which these nerves begin. The motor cells (and therefore the muscular fibres) are active in inverse proportion to these impulses, and as the opponents of a passive movement relax, the muscles shorten that correspond to it in their action; they are thus adapted to posture, and, if this process is in excess, the cataleptic rigidity must of necessity ensue. But it is probable, on many grounds, that the afferent impulses from the muscles have a double action—a lower on the motor centres of the spinal cord, and a higher through the cerebellum, on the motor centres of the cortex cerebri. The phenomena suggest that the action is similar on each; since the cortical cells govern the spinal cells, their functional states must be in related correspondence. Thus the afferent impulses that regulate adaptation to posture must do this through both cortical and spinal centres simultaneously. The excessive action of catalepsy may be in the cortical and not in the spinal mechanism. This would enable us to understand the otherwise strange fact, that the cataleptic state may be induced by influences acting on the brain, as in the induced catalepsy of hysteria. The motor cortical centres, whence the pyramidal fibres proceed, are probably the lowest of the centres of the cortex; the induction of catalepsy may consist simply in the inhibition of higher centres that act on and through them and normally restrain them. The over-action of these entails the over-action of the spinal centres, and the wax-like rigidity results. Thus, too, we can understand that consciousness should be lost or strangely changed during the attack, and its relation to emotion as a cause. So, too, it is clear that every forced change of posture will leave the condition of the centres essentially unchanged, although there will be a slightly different distribution of activity. Arrest the inhibition which prevents the higher cortical centre from acting on the lower, and the resumed control involves restraint of the over-action of the latter and of the spinal centre, and the cataleptic state is at an end.

DIAGNOSIS.—The peculiar rigidity of catalepsy is characteristic, invariable, and renders the diagnosis a simple matter. Hysteria with tonic spasm has been erroneously termed catalepsy, but is distinguished by the local position of the spasm, and the fact that it does not yield to passive force. The malady most likely to be confounded with catalepsy is tetany, since in it there are attacks of tonic spasm involving all the limbs. But in this the rigidity is fixed; it cannot be overcome, and the posture of the limbs changed; while the hands are in a characteristic posture, with the fingers straightened at the middle joints, and their points and that of the thumb brought together. The condition is some-

times simulated: in true catalepsy the rigid limb slowly yields to the influence of gravitation, and more rapidly if a weight be attached to it; in the feigned form the limb and weight are held firm.

PROGNOSIS.—The prognosis is favourable in simple catalepsy in proportion to the freedom of the intervals from affections of sensibility or motion. In pronounced hysteria and psychical affections the condition is often obstinate; and, by interfering with the due nourishment of the system, may cause grave inanition.

TREATMENT.—During the attack itself little can be done save an attempt, which may be repeated at intervals, to rouse consciousness by external stimulation. The ordinary applications—ammonia to the nostrils, cold douches, &c.—often fail in the spontaneous form, but this does not hold true of most cases of induced catalepsy, in which any stimulus that excites a respiratory effort commonly arouses the patient and cuts short the attack. A pinch of snuff will, however, often succeed in both varieties. Another effectual stimulant is faradisation. It may be applied to a limb or to the cervical spine. The current should be gentle at first, and gradually increased. Emetics are also useful in arresting an attack.

The writer has found subcutaneous injection of apomorphine, $\frac{1}{10}$ to $\frac{1}{2}$ of a grain, an efficient remedy for similar paroxysmal conditions; with the onset of nausea, about five minutes after the injection, consciousness is regained, and all spasm ceases. In the intervals between the attacks the treatment is that of hysteria. Iron, antispasmodics, especially valerian, aloetic aperients, and cold baths, are the most effectual measures. Firm moral treatment is also indispensable. Removal from home-influences is often necessary to effect a cure.

W. R. GOWERS.

CATAMENIA, Disorders of.—See MENSTRUATION, Disorders of.

CATAPLASM (κατά, down; and πλάσσω, I mould or smear).—A synonym for a poultice. See POULTICE.

CATARACT (*cataracta*, an obstacle).—SYNON.: Fr. *Cataracte*; Ger. *Staar*.

DEFINITION.—Cataract is an opacity, partial or complete, of the substance of the crystalline lens or of its capsule. The former is called *lenticular*, the latter *capsular*. Either may exist separately, or the two may occur together (*capsulo-lenticular*). From a surgical point of view it is sometimes necessary to embrace within the definition of cataract some extra-capsular opacities, such as inflammatory deposits or the remains of embryonic structures within the pupillary area of the anterior or of the posterior layer of the capsule. Such opacities are called *false* cataracts.

ANATOMY AND PHYSIOLOGY.—The lens consists of three parts: first, the substance of the lens, which is made up of fine, flattened fibres; second, a layer of endothelium lining the anterior part of the capsule; and, third, the capsule, which surrounds and encloses the lens-substance and the endothelium. The fibrous portion of the lens consists of a harder central part, or *nucleus*, and a softer external portion, or *cortex*. The cortex is disposed around the nucleus in thin concentric layers, each of which is subdivided into sectors, separated by fine radiating

fissures, which make up the rays of the stella existing at the anterior as well as at the posterior surface of the lens. The lens-fibres and the endothelium are descended from the external layer of the blastoderm—the epiblast. The capsule of the lens seems to have a double origin, the inner layer being derived from the lens-substance, while the outer is the offspring of mesoblastic elements. The growth of the lens continues throughout life. It is effected by the apposition upon the nucleus of lens-fibres derived from the endothelial cells lining the anterior capsule.

The crystalline lens, being a non-vascular body, is nourished by imbibition from surrounding fluids. It is probable that these fluids are modified by their passage through the vitreous humour and through the capsule; for if the anterior or the posterior capsule be ruptured, the lens becomes opaque. In health, the nutritive fluids pass from the periphery to the centre through the stellate clefts and other channels which exist throughout the lens. Waste-products are apparently driven inwards to the centre, to undergo elimination in a manner not yet fully understood.

After middle life the fibres of the lens gradually assume a yellowish or brownish tint, and the rate of formation of new fibres diminishes. As age advances they become drier, harder, and less elastic, and the capsule thickens. If a senile lens be ophthalmoscopically examined with a strong magnifying-glass, semi-translucent lines, flaws, spaces, and vacuoles, and fine dusty films, may often be discovered, even when there are no coarse physical opacities. These phenomena are due to shrinking of the lens-fibres and loosening of the lamellæ from one another, with accumulation of fluid and lens-debris in the spaces thus formed. Still later, separation of the cortex may take place, both from the nucleus and from the capsule.

PATHOLOGY.—Any fault in the original constitution or development of the lens, or any alteration in its nutrition at any subsequent period, will, sooner or later, induce opacity of some portion of the lens or of its capsule.

The chief *embryonic* abnormalities are (1) inherent faults of the epiblastic cells which constitute the rudimentary lens. The fibro-cellular portions of the lens either do not become transparent, or, if they do clear, their vitality is low, and they soon degenerate. The whole lens is either opaque from the beginning (*congenital cataract*), or becomes opaque within a few months or years (*infantile* or *juvenile cataract*). (2) A temporary or permanent disturbance may occur during intra-uterine life to thwart the development of a lens. If the interference be violent its effects may be permanent, so that the whole of the lens becomes opaque; but if the check be of a temporary character, only a thin layer of opacity may be produced. The nucleus of the lens, though not unaltered, is usually translucent and the cortex transparent; but the intermediate layers—that is, the sub-cortical or perinuclear—are more or less opaque. This form of opacity is called *lamellar cataract*, because in sagittal section it is seen to be disposed in layers around the nucleus; or *zonular cataract*, because the opaque portions, when looked at *en face*, are bordered by a transparent zone of clear cortex. It would seem that the layers which are opaque are either those which were immediately sub-capsular at the time when the disturbance took place, or those which were produced by the endothelial cells during the period

of disturbance. In some rare cases there are two or more concentric layers of opacity, with intervening layers of clear substance, as if there had been several disturbances at different epochs, followed by intervals of normal growth. Lamellar cataracts may be found in eyes otherwise healthy, but they are sometimes associated with developmental defects of the eye and of the general organism, an almost invariable concomitant being deficiency of enamel of the permanent teeth, especially the first molars, the incisors, and the canines (Hutchinson), and not infrequently there is a clinical history of 'fits,' or other evidences of a neurosis. (3) As the result of inflammation of the cornea, uveal tract, or vitreous humour during intra-uterine life, single or multiple opacities may form in the capsule and sub-capsular layer. These are called *congenital capsular cataracts*, and are most common at the anterior pole. They may, however, be scattered about the anterior surface, or lie about the posterior pole of the lens. These opacities consist of proliferation of the endothelium and granular disintegration of the superficial cortical layers, similar to those changes which produce capsular and sub-capsular opacities after birth. It is, however, not improbable that congenital anterior polar cataracts may sometimes be due, not to inflammation, but to retarded separation of the involuted lens-vesicle from the cornea, and delayed closure of the vesicle. This retardation may permit of the intrusion of mesoblastic elements within the vesicle, and these would still further increase the opacity about the capsule. This may conceivably cause an axial opacity, the so-called *spindle-shaped cataract*. (4) *Congenital false cataracts* may be due to the presence of extra-capsular opacities upon the anterior or the posterior pupillary areas. These opacities may be the results of inflammatory exudations, but more commonly they are due to an arrest of development in its final stage, and more particularly to the defective removal of the embryonic vascular capsule of the lens. This state of incompleteness may be designated by the Aristotelian term *Ateleia*.

The alterations in nutrition which, *after birth*, lead to the formation of cataract may be classified as follows:—

(1) *Diminution in the quantity* of the nutrient elements, without material alteration in their quality. This diminution may be due to many causes: (a) mechanical obstacles impeding due access of the fluids; (b) damage to the secretory apparatus consequent upon chronic atrophy of the ciliary body and choroid; (c) general malnutrition, anæmia, cardiac, renal, or other visceral disease; or (d) simple old age.

Mere diminution of the quantity of nutritive elements operates slowly on the nutrition of the lens, and the opacity which results first manifests itself in or about the nucleus—that is to say, the parts most remote from the sources of the nutrient fluids.

(2) *Alterations in the quality* of the nutrient elements. These may be due to: (a) congestion, inflammation, or any other morbid state of the secretory organs of the nutritive fluids—namely, the ciliary body, iris, choroid, and retina; (b) changes in the vitreous or in the aqueous humour; (c) morbid states of the blood and tissues, as in gout, glycosuria, albuminuria, &c.; (d) changes in the physical, chemical, or vital properties of the capsule or of its endothelium, in consequence of which the

fluids that pass through the capsule are not properly prepared for the due nutrition of the lens; or (*e*) impeded elimination of waste-products, whether from the lens itself or from the whole eyeball.

Alterations in the quality of the nutrient elements act more or less speedily, and if the alteration be very great, as in rupture of the capsule, they are very prompt in producing opacities. These affect primarily the sub-capsular and the cortical layers, with which the altered fluids first come into contact. If the alteration be intense, but of brief duration, as in the case of perforating ulcer of the cornea in purulent ophthalmia of children, the change may be limited to a circumscribed area of the capsule and sub-capsular layers, giving rise to an *anterior polar* cataract, or, if this opacity assume a conical shape, *anterior pyramidal* cataract. In alterations of less intensity, but of continuous duration, due to chronic morbid states in the posterior segment of the globe, such as retinitis pigmentosa, slowly forming opacities may occur in the posterior sub-capsular layers of the lens, beginning at the pole of the lens, and extending thence in a radial direction along the rays of the posterior stella, giving rise to *posterior polar* or *stellar* cataracts.

(3) *Alterations of quantity and quality combined.* This co-operation obtains in most of the cases of cataract; and the effects of the two conditions are variously combined. The great majority of cataracts are cortical, because there are comparatively few cases, even in apparently healthy eyes, where there does not exist some difference in the quality of the nutrient elements, as well as falling off in quantity.

MORBID ANATOMY.—In senile cataracts, and in most slowly forming cataracts, the opacity of the lens-fibres is merely an extension of physiological senile changes, and is usually preceded by the formation of spaces and vacuoles between the fibres and the lamellæ due to shrinkage and sclerosis, as already described. Priestley Smith's investigations have shown that a diminished rate of growth of the lens precedes cataract. According to Otto Becker the initial cause of senile cataract is drying of the nucleus and its separation from the cortex, the gap between them being filled by a fluid which is probably derived from the fibres. In more rapidly forming cortical cataracts it may often be observed that the opacity begins in the sub-capsular layers and about the borders of the rays of the anterior stella. Instead of shrinkage and sclerosis, the fibres may swell and become softer. In some cases the rays of the stella are broadened, but whether they are or not, a hazy line may often be traced along one or both of the edges of the ray, in the ends of the cortical fibres. From the ends of the fibres the opacity extends along their lengths towards the equator, until the whole of the cortex is implicated. The endothelium also undergoes changes, giving rise to triangular patches of glistening opacities which may present a mother-of-pearl appearance. When once the opacifying process begins in any part of the lens, it tends to progress till the whole is involved. The cataract is then said to be *ripe* or *mature*.

If a ripe cataract be allowed to remain within the eye indefinitely, it may undergo further degenerative changes and become what is called *over-ripe*. In some instances the lens-substance shrinks and becomes hard and calcareous, and the capsule thickens and grows more opaque. In other cases

the cortex liquefies, allowing the harder nucleus to move within it (*Morgagnian* cataract). Over-ripe cataracts may also become loosened, in consequence of softening or rupture of the suspensory ligament. Fluidity of the surrounding vitreous humour may likewise occur, and then on slight provocation the loosened lens may fall into the vitreous.

A peculiar form of degeneration sometimes occurs in the lens, producing what is called *black* cataract. This is commonly thought to be due to the staining of the lens by blood-pigments, and in some cases this may be so; but, according to a careful spectroscopic examination by MacMunn, no blood-pigments were found, but the lens was infiltrated with a pigment analogous to melanin.

ÆTIOLOGY.—Besides the causes and conditions already described, there are other circumstances which may induce cataract, or at least predispose to it. The most important of these are as follows:—

Age.—Cataracts occur more frequently in infancy and in old age than in early adult and middle life, except when due to disease of the eye or to injuries.

Sex.—The two sexes are about equally affected, except in respect of traumatic cataract, in which males greatly preponderate. *Heredity.*—Hereditary influences may determine the occurrence of cataract in several ways. There may be the inheritance of actual (congenital) cataract or of a disposition to cataract. In either case the descent seems to pass more through the male than the female line. *Occupation.*—Certain occupations predispose to cataract, especially such as necessitate close application of the eyes to near work, or involve stooping positions or exposure to bright light, more particularly if light be combined with heat (as in the occupation of smiths, cooks, stokers, &c.), and occupations in which the eyes are exposed to irritating fumes and vapours. Uncorrected *errors of refraction* and accommodation may favour the production of cataract by overstraining the accommodation, and by the frequent and successive changes which the capsule has to undergo. These strains not only increase the wear and tear of the lens, but facilitate the loosening of the cortex of the lens from the capsule. Hypermetropia and astigmatism may predispose to cataract in another way. The incessant strain upon the ciliary muscle required to correct hypermetropia involves constant congestion of the ciliary body and the base of the iris. The nutritive fluids secreted during this state are not perfectly normal; they are in some measure altered in quality and will therefore sooner or later make their influence felt on the transparency of the lens.

Injuries.—Wounds of the capsule are generally soon followed by opacity, which occurs more quickly in proportion to the size of the rent and the youth of the patient. Perforating wounds of the cornea, especially when followed by suppuration, may induce cataract even though the capsule have not been perforated. Blows on the head, face, or brow, shocks and jars of the body, may also be followed by cataract, either from causing flaws or cracks or rents in the capsule or by rupture of the suspensory ligament. Other conditions which accompany and apparently predispose to cataract are degenerative changes in the blood-vessels, especially of those of the head and neck and of the eye; diseases of the orbit, or changes in the orbital vessels; cardiac disease, renal disease, gout, and diabetes. It is probable, however, that the influence of diabetes in the production of cataract has been overrated. The

number of diabetic persons who get cataract is comparatively small, and when cataract co-exists with glycosuria there is no necessary causal relationship between the two. It has been stated that ergotism often leads to cataract (J. Meyer).

CLASSIFICATION.—Cataracts have been classified in many ways, as (1) according to *age*—congenital, juvenile, senile; (2) to *stage*—incipient, progressive, stationary, immature, mature; (3) *consistency*—fluid, soft, hard, and mixed.

Following the indications afforded by the consideration of the pathology and causation of lenticular opacities, cataracts may be arranged into five main groups according (1) as they are due to developmental abnormalities—*embryonic*; (2) as they occur independently of obvious disease of the eye—*idiopathic*; (3) as they are associated with obvious disease of the eye—*sympathetic*; (4) as they follow wounds or injuries of the eye—*traumatic*; and lastly (5) as they are due to the opacification of lens-matter remaining within or on the capsule at the time of operation for the removal of cataract, or of lens-matter which may have grown subsequently and then become opaque—*deutero-pathic* or subsequent (*δευτεροπαθές*, I suffer later). Within one or other of these groups all the varieties of cataract may be arranged according as they implicate (1) the nucleus; (2) the perinuclear region (lamellar); (3) the axis (spindle-shaped); (4) the cortex, anterior or posterior; (5) the equator; (6) the capsule, anterior or posterior; (7) the external surface of the capsule—extra-capsular—which may be congenital or acquired.

SYMPTOMS.—The chief subjective symptom of senile and other acquired cataracts is impairment and 'fogginess' of sight, especially for distant objects. As the cataract advances, this dimness increases, until the appreciation of form and colour may be lost; but, in eyes otherwise healthy, the power of discerning light always remains, even though the lens be quite opaque. In ordinary daylight the eye may still be able to 'count fingers' at a distance of six to twelve inches, or see the movement of a hand at a distance of twelve to eighteen inches (hand-movement); and in a darkened room the eye should be able not only to see a lighted candle at a distance of ten to fifteen feet, but should be able also quickly to indicate its position in various parts of the field of vision without movement of the eyes or head. This would indicate that there is a good *perception* of light, good *projection*, and a good *field of vision*. In the early stages of cataract, especially when the opacity occupies the pupillary area, patients may see better when the eyes are shaded, the pupil being thereby made to dilate; a bright light embarrasses and distresses them. *Muscae volitantes* are also often seen; these may be due to congestion of the ciliary blood and choroid which may have preceded cataract, or they may be the results of the straining efforts to see through a hazy lens. Another common symptom is the multiplying or splitting of objects, especially of lights (*monophthalmic polyopia*). The amount of impairment of sight is not always in proportion to the amount of appreciable opacity; diffused haziness about the nucleus or about the pupillary area is more hampering than well-defined striæ or sectors with clear interspaces. There may be dense opacities at the equator with perfect acuteness of vision. An occasional early symptom of cataract is a change in the refraction of the eye,

leading to what some persons describe as getting 'the second sight.' Eyes that have required strong convex glasses for near work prefer weaker ones, or none at all; and those who have needed convex glasses for distance may find that they see better without them, or even with concave glasses. Many children with zonular cataract are thought to be merely short-sighted, and often they are short-sighted, but the true cause of the defect is frequently not discovered till the 'optician' has failed to get good vision by means of spectacles.

DIAGNOSIS.—In all cases of failing sight, whether opacities are to be seen or not by means of direct light or by so-called focal or oblique illumination, the media of the eye should be examined with the ophthalmoscope. Owing to thickening of the lens-capsule, the pupils of old persons are sometimes grey instead of black, and the lenses may seem to be opaque when they are not really so; and in glaucomatous eyes the change of colour which takes place in the cornea and in the lens not infrequently suggests cataract even when the media are translucent. Diagnosis of advanced cataract is easy, but in slighter cases careful examination of all parts of the lens may be needed. The aims of diagnosis are to recognise the presence of the cataract, to ascertain its seat, extent, consistence, and its relations or complications, and to discriminate it from other morbid states which may be mistaken for it. Examination of the anterior part of the lens, the capsule, anterior cortex, and the nucleus may be made with 'focal illumination,' i.e. by concentrating light upon the eye by means of a magnifying-glass. The equator of the lens and the posterior cortex require the ophthalmoscope for their examination.

Anterior extra-capsular opacities, whether inflammatory deposits or remains of the capsulo-pupillary membrane, project beyond the level of the capsule. The latter have usually attachments to the anterior surface of the iris (*circulus minor*), whereby they are distinguished from inflammatory adhesions (posterior synechiæ) which are connected with the edge and posterior surface of the iris. Capsular cataracts may be single or multiple. Anterior polar cataract occurs as a small circumscribed dot in and behind the centre of the capsule; when viewed in profile the dot can be seen to project slightly beyond the general level of the capsule; occasionally it projects considerably and is conical (pyramidal cataract). Both these cataracts are generally associated with nebula or leucoma of the cornea. Multiple capsular opacities may be scattered irregularly or arrayed in a circle. Cortical opacities assume many shapes—striæ, wedges, or irregular patches. When the opacities are in the anterior cortex, they are convex in their arrangement; when in the posterior they are concave. Equatorial opacities may be partial or complete, continuous or interrupted, limited as a corona to the periphery, or send tapering offshoots towards the anterior and posterior cortices. Perinuclear opacities may be circumscribed or diffused. The circumscribed (lamellar or zonular cataracts) are best seen with the ophthalmoscope when the pupil has been dilated by a weak solution of atropine or other mydriatic. Lamellar cataract appears as a greyish disk surrounded by a clear zone. If the nucleus be translucent the disk is most opaque at its edge; but if the nucleus be itself opaque, then the opacity is densest in the centre. The edge of the disk may be sharply

defined, or indented or broken by centrifugal striæ. Nuclear opacities may be dense and diffuse, as in some congenital cataracts, or they may appear as a brownish or yellowish undefined blur or cloud, as in early senile cataract. Posterior polar cataract can be properly seen only with the ophthalmoscope. It may assume the form of a smaller or larger dot, or of several dots, or of radiating streaks. The position of the opacity is determined partly by its remoteness from the pupil, and partly by the small excursions it makes in the various movements of the eye, the posterior pole of the lens being immediately in front of the centre of rotation of the globe.

Whenever practicable, it is desirable in all forms of cataract to examine the state of the optic nerve and fundus of the eye. The data thereby obtained may influence the prognosis and the later treatment. When the cataract is mature the pupil is uniformly white or greyish, and the opacity comes up to and seems to touch the pupillary edge of the iris, and no reflex can be obtained by means of the ophthalmoscope. If the cortical layers are not opaque, a shadow of the iris can be thrown into the lens by means of oblique illumination, giving the appearance of a gap between the opacity and the edge of the iris. For surgical purposes it is not necessary that the whole of the lens-substance should be opaque; it is enough that the cortical layers be opaque, for then the connection between the cortex and the capsule is broken, and the endothelial cells have lost their power of growing fresh lens-fibres. It is not always possible to estimate the consistency of a cataract; but, as a rule, cataracts which begin in the cortex, or occur in persons under the age of thirty years, or are rapidly formed, are usually 'soft'; such cataracts are generally whiter and more opaque than harder ones, and the opacity is often patchy or diffused.

The failing sight of chronic glaucoma is sometimes ascribed to cataract. By the ophthalmoscope and by palpation, the error may easily be avoided. When the lens is not opaque in glaucoma the diagnosis is simple, mere absence of opacity excluding cataract; but when, in glaucoma, the lens is also opaque, the diagnosis may present some difficulty. In chronic glaucoma the characteristic symptom is increased hardness of the eyeball. Usually a history of haloes seen round artificial light may be elicited, with perhaps attacks of neuralgia and redness of the eye. The pupils are semi-dilated and very sluggish, if not immobile, and the anterior chamber is shallow, and the sight is more impaired than the cataract accounts for. In advanced (absolute) glaucoma there is an entire abolition of the perception of light.

PROGNOSIS.—Cataract may be considered, as regards its prognosis, from two points of view: first, when it is immature, as to its probable course; and, secondly, when it is mature, as to the prospects of recovery of sight after operation. Some cataracts may remain stationary throughout a long life: such are anterior polar, anterior pyramidal, and zonular cataracts. Others progress slowly, such as posterior polar cataracts. Cortical cataracts, as a rule, advance more rapidly than nuclear ones, and soft cataracts more rapidly than hard. Generally the progress is quicker the earlier the cataract occurs, and the closer its association with morbid states of the eye or with injury. When cataracts are sharply defined, confined to the

equator of the lens, or present only a haze or blur in or about the nucleus, or when the cortical striæ are few and fine and well-defined, with clear interspaces, and the eyes are otherwise healthy, and the sight good, the progress will usually be slow; but when the opacities are dense, irregular, ill-defined, diffuse, especially where they extend far along the anterior or posterior cortical layers, with rapidly failing sight, or with sight bad out of all proportion to the amount of actual opacity, or when there is obvious irritative or inflammatory disease of the eye, or when there is grave visceral or other constitutional disease, such as gout, diabetes, nephritis, then the progress will probably be rapid. But it should be remembered that cataracts which may have been very slowly progressing may at any time take a sudden and rapid start and progress quickly. The usual course, in senile cataract, is from one to four years or more.

As regards *mature* cataract, the prognosis in reference to operation is good in proportion as the eye is otherwise sound, and the general health good. Where there is a history of attacks of pain or of redness in the eye, or where there are signs of past disease, whether iritis or choroiditis or retinitis, or where there is a high degree of myopia, or where the iris is discoloured and lustreless, and sluggish or immobile, or where it is tremulous, or where the anterior chamber is much deeper than natural or much shallower, or where there is a chronic conjunctivitis, ciliary blepharitis, or catarrh of the lacrimal sac, or where the tension of the eye is too high or too low, the prognosis is more or less unfavourable. Nystagmus, squint—especially divergent squint—are unfavourable concomitants; and the prognosis is, of course, less favourable when it is known that there was antecedent disease of the fundus, or when, failing this knowledge, the perception of light is poor, and the projection bad, and the field of vision contracted or irregular. Where there is no perception of light, the prognosis is hopeless.

TREATMENT.—From a medical point of view the treatment of cataract is rather preventive and palliative than operative.

Preventive.—Little can be done, directly, to prevent many of the embryonic forms of cataract, and those other cataracts which are due to simple diminution of the *quantity* of nutrition, of which pure senile cataract is the type. On the other hand, it is at least ideally possible to prevent the occurrence of many of those cataracts which are due to alterations of *quality* of nutrition. By the prevention or the avoidance of those general and local morbid conditions which induce these qualitative alterations, or by their early and effectual treatment, *secundum artem*, by obedience to the laws of personal and public hygiene, by conforming to the requirements of healthy sight, by the scientific correction of errors of refraction and accommodation, by adequate protection of the eyes during those occupations which entail risks of physical, thermal, or chemical injuries, cataract may often be prevented, or its progress arrested, if perchance initial changes have already begun.

Palliative.—If opacity of the lens exists, but not to an amount sufficient to materially impair sight, it is sometimes possible, by general and local measures, to improve the special nutrition of the lens, and thereby to retard the progress of the cataract. In some cases, where slight endothelial and cortical

opacities occur during transitory inflammation of the cornea, iris, or choroid, or during exacerbations of diabetes and other diathetic diseases, the opacities may gradually disappear on the resumption of normal nutrition after the subsidence of the inflammation or the amelioration of the general health. But, except within these narrow limits, genuine cataractous opacities are incurable and irremovable by any means known to positive science other than operation.

Nevertheless the inconveniences which attend progressing cataract may be palliated by alleviating local irritation, by the use of tinted glasses, by shading the eyes from bright light, by correction of optical errors, or by the use of stenopæic glasses. When the opacity, whether congenital or acquired, is limited to the pupillary area of the lens, vision may be improved by moderate dilatation of the pupil, by means of weak solutions of atropine (gr. $\frac{50}{1000}$ to gr. $\frac{1}{10}$ in an ounce of water), or other mydriatic. In any case, if the eyes be otherwise healthy, moderate use of the eyes may be indulged in, short of discomfort and fatigue.

Operative.—The operative procedures for the removal of cataract are: First, *discission*, or *keratonyxis* (κέρας, horn [cornea]; ρύσσω, I prick), in which the anterior capsule is torn by a fine needle passed through the cornea. The lens-substance, being thereby exposed to the disintegrating action of the aqueous humour, gradually liquefies and is ultimately eliminated by the absorbent vessels. Secondly, *discission with evacuation*. The capsule is torn as in discission, but the lens is more freely broken up. The fragments are then, or within a few days, removed by a grooved curette through a narrow incision immediately within the corneal margin (linear extraction), or withdrawn by aspiration through a fine tube (suction). The third operation is *extraction*, in which an incision is made at or near the corneal margin, the capsule divided and the lens extruded by gentle pressure. Until quite recently a portion of the iris was in the modern operation removed (iridectomy) at the time of the extraction-operation, or a few weeks or months previously (preliminary iridectomy). But some surgeons now try to dispense with the iridectomy, or only do it when the cataract cannot otherwise be safely and efficiently removed. Discission with or without evacuation is applicable to 'soft' cataracts, while extraction is usually reserved for 'hard' cataracts. Soft cataracts may be operated on almost at any time and in any stage; but hard cataracts are, as a rule, not extracted until they are 'mature,' though there are many circumstances which may suggest and justify departure from this rule. An immature cataract may be operated on whenever both eyes are so affected that the patient is unable to read ordinary print or to follow his occupation or profession.

It may here be remarked that when zonular cataract is slight, and does not cause appreciable impairment of sight, operation may be indefinitely deferred. If, on the other hand, the opacity is more pronounced, and the degree of impairment of vision is such that education or occupation cannot be comfortably pursued, operative interference is called for. The precise procedure will depend upon circumstances.

When the eye has recovered from the effects of the operation, spectacle-lenses are needed to correct the error of refraction due to the absence of the

crystalline lens (*aphakia chirurgica*). Except in very short-sighted eyes convex lenses are required. The strength of the lens will depend upon the refraction of the eye, but ordinarily for distance a lens of about 10 dioptries (4-inch) is needed, and for near work about 14D. ($2\frac{3}{4}$ -inch). If astigmatism (see VISION, Defects of) be present, then cylindrical lenses must be added to the spherical. In any case, it is desirable that an eye which has been operated on should not be used too soon, nor, at first, for too long a time.

Sometimes it happens after the removal of a cataract that, owing to the presence of hazy or opaque lens-capsule within the pupil, it is not possible to obtain useful vision by the most accurate optical correction. It will then be necessary to tear through the membrane with one or two fine needles (*needling*), and thereby effect a clear opening opposite the pupil.

JOHN TWEEDY.

CATARRH (κατά, down; and ῥέω, I flow). SYNON.: *Coryza*; Fr. *Catarrhe*; *Coryza*; Ger. *Katarrh*; *Schnupfen*.

The term catarrh is applied to inflammation or congestion of any of the mucous membranes, attended with increased secretion. The term is, however, often limited to an inflammatory affection of the upper part of the air-passages. See NOSE, Diseases of.

CATARRHAL (κατά, down; and ῥέω, I flow).—Pertaining to catarrh, both in its pathological and clinical significations—for example, catarrhal products, catarrhal pneumonia, catarrhal attack.

CATHARTICS (καθαίρω, I cleanse).—This word is sometimes used as a synonym for purgatives; but in a more limited signification it means purgatives of moderate activity. See PURGATIVES.

CAUDATE NUCLEUS.—See CORPUS STRIATUM.

CAUSTICS (καίω, I burn).—DEFINITION. Substances or measures which destroy organic tissues with which they may be brought in contact.

ENUMERATION.—The caustic substances in most common use are Potash, Soda, and Lime; Nitric, Hydrochloric, Sulphuric, Chromic, Lactic, and Glacial Acetic Acids; Red Oxide, Acid Nitrate, and Perchloride of Mercury; Carbolic Acid; Chloride of Zinc; Chloride of Antimony; and Arsenic. The ordinary caustic measures are the galvano-cautery; Paquelin's cautery; the red-hot iron; and moxæ. See also POISONS.

USES.—Caustics are chiefly employed to destroy unhealthy, exuberant, or malignant growths; to establish issues for the purpose of counter-irritation (see COUNTER-IRRITATION); and to destroy poisons when introduced into the body by breach of the external surface.

T. LAUDER BRUNTON.

CAUTERETS, in the French Pyrenees.—Sulphur-waters. See MINERAL WATERS.

CAVERNOUS.—A peculiar quality of sounds heard on auscultation of the lungs, indicative of the presence of a cavity. See PHYSICAL EXAMINATION.

The term is also used to denote a special kind of tissue consisting of fibrous trabeculae arranged to form intercommunicating spaces, found in the penis

and in some varieties of new-growth. See TUMOURS; ANGIOMA.

CAVITY, Pulmonary.—SYNON.: *Vomica*; Fr. *Vomique*; Ger. *Lungengeschwür*.

DEFINITION.—A cavity is a hollow space in the substance of the lung, formed by destruction of pulmonary tissue. Spaces due to dilatation of normally existing channels, such as the bronchi, are not properly called cavities unless the surrounding substance of the lung is invaded and forms part of the wall. The term is sometimes unnecessarily restricted to such cavities only as communicate with a bronchus.

ÆTIOLOGY.—(1) The great majority of pulmonary cavities are due to the softening of tubercular foci; it is therefore to tubercular cavities that reference will here be principally made; (2) the next most frequent cause of excavation of the lung is ulceration extending through the wall of a dilated bronchial tube (bronchiectasis); other causes are (3) the action of rarer forms of micro-organisms, such as actinomyces or aspergillus; (4) abscess of the lung, due either to acute lobar or lobular pneumonia, to extension from the pleural or abdominal cavities, to pyæmia, or to inhaled foreign bodies (coins, tracheotomy tubes); (5) gangrene of the lung; (6) ulceration of new-growths; (7) hydatid cysts, the contents of which have been evacuated, may form spaces closely resembling true cavities.

MODE OF FORMATION.—(1) *Tubercular cavities.* The deposit of tubercle-bacilli in the lung is followed by the formation of the small inflammatory nodules known as tubercles or grey granulations. Around these is a zone of inflammatory consolidation of the lung. As the process spreads, masses of these tubercles coalesce and considerable areas of consolidation may result. At the periphery of these areas much inflammatory fibrous tissue is frequently formed. Partly owing to the cutting-off of the blood-supply to the central portions of the consolidated mass by the contraction of this fibrous tissue, and partly by the direct action of the toxins of the tubercle-bacillus, necrosis of the central portions of the solid area is brought about. The process is then known as caseation (see CASEATION). In course of time the destructive process invades the wall of a bronchial tube, which gives way and permits the entry of the caseous material into the air-passages. This is then coughed up, and the space in which it originally lay remains empty, constituting a cavity.

(2) In the condition known as bronchiectasis dilatation of the bronchi in some portion of the lung occurs, and stagnation of the contents of these tubes takes place (see BRONCHI, Diseases of: 4. BRONCHIECTASIS). Decomposition of the retained secretion ensues; the walls of the bronchus are irritated by the products of putrefaction, and ulceration is the result. The process extends into the surrounding lung-tissue, which is generally already the seat of chronic interstitial fibrosis, and thus irregular excavations are formed: The relation of such cavities to the original bronchus is often difficult to trace: the fact that an inflamed and expanded bronchial tube enters and leaves the cavity, and that traces of bronchial tissue may be found in its wall, together with the existence of dilatation of bronchi in surrounding parts, may throw light on the nature of the process.

VARIETIES OF CAVITY.—According to the acuteness or chronicity of the disease from which the

excavation results, and the subsequent changes which may take place, cavities present very different characters. In the most acute cases of tuberculosis, as in instances of abscess or gangrene of the lung, the cavity consists of an irregular space, with softened ragged walls, lying in the midst of consolidated or partly consolidated lung-tissue. If gangrene has occurred the walls may be black, sloughy, and very fetid: in any case they will be soft and friable, with shreds of necrotic tissue adherent to their surfaces. In less acute cases, constituting the majority of tubercular cavities, the walls are less soft and ragged, consisting to a great extent of fibrous tissue, the result of chronic inflammation of interlobular septa; to their surfaces masses of caseous material may be seen here and there adherent. In still more chronic cases the walls may wholly consist of fibrous tissue, lined by a thin layer of granulations. The walls are then smooth and firm, and the cavity will tend to have a more distinctly rounded shape, owing to obliteration of irregular outgrowths by contraction of the fibrous capsule. This appearance is presented by old cavities in which the actual process of destruction has ceased and the disease become quiescent. The process may, however, at any time again become active and ulceration take place in the walls, so that mixed forms of cavity result, in which the walls are smooth and resistant in some places, and ragged and ulcerated in others.

CONTENTS.—Since an active cavity is practically an abscess, the contents of such consist largely of pus—that is, of leucocytes and albuminous fluid. In tubercular cases this will be mixed with caseous material which may contain elastic fibres from the broken-down lung-tissue. The walls, even of quiescent cavities, often continue to secrete purulent material, so that expectoration may continue after active disease has ceased. In bronchiectatic cavities and in some tubercular vomicæ the contents are very offensive owing to putrefaction, and cause the breath of the patient to be intolerably fetid. Rarely in old cavities organisms from the air may effect a lodgment, and moulds, such as aspergillus, may grow on the walls and appear in the expectoration. In cases due to actinomycosis small yellowish masses of the ray-fungus may be found in the pus.

BLOOD-VESSELS IN CAVITIES.—In the process of excavation blood-vessels, being more resistant than the other tissues, may remain for long periods intact. Thus bands consisting of blood-vessels, with a certain amount of fibrous tissue, may frequently be seen crossing tubercular cavities. In course of time the walls of such vessels are invaded by the inflammation, and thrombosis with obliteration of the lumen generally results. In some cases, however, the wall of the vessel is weakened without the occurrence of thrombosis; dilatation results and an aneurysm is formed. This will tend to fill the cavity while the latter is small, and to increase gradually along with it. Sooner or later a point is reached when no further expansion of the vessel wall can take place, and rupture of the aneurysm occurs, leading to profuse and even fatal hæmorrhage. In rarer cases direct ulceration of a vessel may take place before thrombosis has been effected within it, and hæmorrhage may again be the result. Vessels running in the walls of cavities are specially liable to be thus eroded; and this cause of bleeding

is not uncommon in connection with bronchiectatic cavities, and with those resulting from the ulceration of malignant growths.

SEAT AND DISTRIBUTION.—Tubercular cavities occur most frequently in the upper lobes of the lungs. The two sides of the body are affected with practically equal frequency, and both lungs are often the seats of excavation at the same time. In some instances only a single cavity may be found, and may vary in size from that of a pea to a chasm occupying a whole lobe of the lung. In other cases many cavities occur together, a whole lobe, or even more than one, being honey-combed with excavations. These may communicate one with another, and may ultimately coalesce to form a single cavity. Partial septa are frequently found in vomicae, constituting the remains of partitions which originally separated distinct spaces. Tubercular cavities in the lower lobes are not, however, very uncommon, and retention of the secretions with consequent decomposition may occur in these as in bronchiectatic cavities, which usually occupy this position.

EFFECTS.—The existence of cavities in the lung, surrounded by tissue in a condition of chronic inflammatory fibrosis, causes alteration in the shape of the chest-walls and in the relative positions of the thoracic viscera. The tendency of fibrous tissue to contract being aided in this case by the existence of hollow spaces which offer no obstacle to the process, there takes place a considerable shrinkage of the affected part. By this means the size of the cavity is reduced (although it is probable that a vomica when once formed never completely disappears), and owing to the existence of pleural adhesions the thoracic wall is drawn in and the diaphragm elevated into the chest. If only one lung is affected the sound lung of the opposite side expands so as to occupy more space and do more work (vicarious emphysema). At the same time, owing to diminished support from the shrinking lung and occasionally to the actual traction of adhesive bands, the heart is moved from its normal position and may be found beating in the left axilla if the left lung be shrunken, or to the right of the sternum in cases of right-sided cavities. In patients with old and very extensive cavities, the alteration in the shape of the thorax may be so great that actual curvature of the spine is produced, the concavity of the curve being towards the affected side.

In the majority of cases adhesion takes place between the two surfaces of the pleura over an excavated spot. Rarely in the absence of this protective process the vomica may extend and burst into the general pleural cavity, causing pneumothorax. The constant drain of pus from a chronic cavity, and the absorption of bacterial toxins from it, may finally result in the onset of amyloid (lardaceous) degeneration of other organs.

DIAGNOSIS.—The phenomena from which it is possible to infer the existence of a cavity in the lung are dealt with at length in the article on **PHYSICAL EXAMINATION**. Little can be learned from inspection of the chest, since fibrosis of the lung without excavation is capable of giving rise to great retraction of the thoracic wall. Percussion is of value in some cases. It is necessary to bear in mind that, although percussion over a cavity should, and does, give rise to a tympanitic note under favourable circumstances, in the great

majority of instances this resonance is modified by the existence of a layer of solidified lung between the chest-wall and the cavity, and also frequently by thickening of the pleura overlying the affected area. It is thus only in cases of very superficial cavities that tympanitic resonance is obtainable. Usually the normal percussion-note is impaired over a vomica, the sound obtained being often of a high-pitched 'boxy' character, which is very suggestive but not distinctive of excavation. By very vigorous percussion, the patient's mouth being open, it is possible to elicit the 'cracked-pot' sound; but it is inadvisable to use the amount of force needful to elicit this phenomenon, owing to the danger always existing of setting up hæmorrhage from some weakened or aneurysmal vessel. In the case of very large cavities with fluid contents, alteration of the posture of the patient from the vertical to the horizontal position, or *vice versa*, may cause changes in the percussion-signs, comprising alterations both in the respective areas of resonance and dullness, and in the quality of the sound obtained. The most characteristic sign audible on listening over a cavity is the so-called cavernous or amphoric breath-sound. This differs from bronchial or tubular breathing in the deeper pitch of the note and in possessing a certain musical quality which has been compared to the sound obtained by blowing across the neck of a bottle. Vocal sounds are increased over a cavity, and occasionally a peculiar resonating effect is produced within it. Whispering pectoriloquy is also very marked, but is not distinctive of excavation, as it may be heard equally clearly over consolidated lung. Air passing through liquid contained in a cavity gives rise to bubbling sounds (râles) of peculiar hollow metallic quality, and the dropping of fluid from the roof of a large vomica may occasionally produce 'metallic tinkling.' The bell-sound may also be heard over large empty cavities on tapping one coin upon another placed against the chest-wall. When the patient coughs splashing of the fluid in the cavity may result and be audible to the observer, and the sound of the cough may like the voice be resonated in the cavity. At the end of the effort of coughing a peculiar suction-sound ('post-tussic suction') may be heard over a vomica, comparable to that produced by the sudden expansion of a wet india-rubber ball.

It must be distinctly understood that the phenomena just enumerated do not occur in the case of all cavities, some or all of them being absent in the great majority of instances. It is probably impossible to ascertain the existence of a cavity less than one inch in diameter, and spaces even of this size may be entirely latent if deeply situated. The two most valuable signs are probably the 'boxy' percussion-note and cavernous breathing. If the bronchus leading to a cavity become blocked, all characteristic signs on auscultation will necessarily be absent.

PROGNOSIS.—No patient in whom a pulmonary cavity exists can be said to be free from danger. The principal risk is always that of the occurrence of hæmorrhage, since it is impossible to make sure of the absence of aneurysmal dilatation of vessels crossing the cavity, while further ulceration of the walls leading to vascular erosion may at any time occur. Apart from this danger, the outlook is not necessarily bad. It is now well established that in

tuberculosis: arrest of the disease may take place at any stage, and that even after extensive excavation has occurred the malady may become quiescent and the patient recover a fair measure of health and activity. In individual cases judgment as to the probable future course must be based upon a review of all the circumstances. Gain of weight, absence of fever and sweating, good digestion, and regular sleep point to quiescence of the disease. Long previous duration apart from signs of marked deterioration of health is in favour of continued chronicity. The presence of some cough and expectoration in such cases is not necessarily of evil import, since a cavity may continue to secrete for an indefinite period. Even the presence of tubercle-bacilli in the sputum is not of necessity evidence of active tuberculosis, since it seems that these organisms may continue to exist as saprophytes after they have ceased to do active mischief.

TREATMENT.—In cases with tubercular excavation of the lung, the treatment is that of the general condition (*see* PHTHISIS), and no special modification need be introduced owing to the existence of a cavity. When the disease has reached this stage, however, and is still active, it is not advisable to send the patient away from home either for a sea-voyage or for residence at a distance, on account of the danger of hæmorrhage. When the disease has become quiescent, cases with cavities may be much benefited by residence in such climates as permit of continuous life in the open air without danger of chill. Wintering abroad is generally advisable, and it is important that those affected should not remain contented with the good results of one such winter sojourn, but should be encouraged to spend two or three winters away from home (Mitchell Bruce).

Treatment directed to the cavity itself may be necessary in cases in which the contents stagnate and putrefy. Here much may be done by means of medicated inhalations. Eucalyptus oil or terebene may be used as a moist inhalation, one drachm of either fluid being put into a pint of hot water and the patient directed to inhale the steam thrice or more daily. A kettle of water containing a small quantity of one of these substances kept simmering in the sick-room is of the greatest service in rendering the atmosphere around the patient tolerable to others. Dry inhalations may be given on an oronasal respirator, and may consist of a few drops of a mixture of equal parts of spirit of menthol, oil of eucalyptus, and spirit of chloroform, used for half an hour three times a day or oftener. In basal cavities, the factor of which does not yield to these remedies, intralaryngeal injections may be practised with apparent benefit. The injection is made through the glottis with a special curved syringe, guided by the laryngoscopic mirror, the patient being directed to lean over towards the affected side, to ensure gravitation of the fluid towards the seat of the cavity. A solution of menthol in olive oil forms a very suitable fluid for such injection. Injections by means of a hypodermic needle passed directly into pulmonary cavities at the apex and elsewhere have been tried, but are not to be recommended for general use. Surgical interference is necessary in some cases of gangrene of the lung or abscesses that drain badly. It is impossible to lay down general rules as to the indications for such treatment. If very fetid expectoration continue in spite of medical remedies, and if the patient's

general condition be deteriorating owing to absorption of septic products, the question of incision and drainage of the cavity must be carefully considered. Resection of portions of one or more ribs will probably be necessary, if operation be decided upon in order to secure free drainage. In cases of quiescent cavities with resulting deformity of the chest, carefully planned and graduated gymnastic exercises are of use to exercise the muscles and lessen deformity. Residence at high altitudes is sometimes beneficial in such cases, owing to the tendency which it exerts to cause expansion of existing healthy lung-tissue. *See* CLIMATE, Treatment of Disease by.

W. CECIL BOSANQUET.

CELL (*cella*, a cell in a honeycomb; a closed chamber).—The term 'cell' was for a long time applied in anatomy to various spaces in the body large enough to be recognisable by the naked eye; hence 'cellular' tissue, a name which has been used to designate areolar or connective tissue. But the word came afterwards to be applied by botanists in an entirely different sense, namely, to the hollow, bladder-like particles of which many of the parts of plants were shown by the microscope to be composed; and the name was extended so as to apply to similar particles in the animal body. It has since been retained and used in histology in that sense, although it has long been recognised that these particles, whether in plants or animals, but especially in the latter, do not necessarily conform to the original definition of a cell, the principal factor in which was the presence of a definite cell-wall.

Cells may be either *free* or *fixed*. Instances of free cells are to be found in the white corpuscles or leucocytes which occur in blood, in lymph, and in pus, and which constitute the bulk of lymphatic or lymphoid (adenoid) tissue. As instances of fixed cells may be mentioned the various kinds of epithelial cells; the cells of the connective tissues, including cartilage and bone; the cells of nervous tissue; and those which constitute the several kinds of muscular fibres. To these may be added the cells of most tumours.

All the cells in the body can be shown to have been ultimately derived from the ovum, or egg-cell, which, after impregnation by and union with a spermatozoon or sperm-cell, divides at first into two, these again into two, and so on by a binary process of subdivision until a small mass of cells is ultimately produced. As the cells continue to multiply, this mass presently becomes hollowed out by the accumulation of fluid in its interior, and is converted into a vesicle, the wall of which is eventually found to show an arrangement of its component cells in three layers, which collectively form the *blastoderm*. The three layers are termed respectively, from without in, the *epiblast*, *mesoblast*, and *hypoblast*, or *ectoderm*, *mesoderm*, and *entoderm*, and give origin in course of development to the different tissues and organs of the body. The epiblast produces the cuticle and nervous tissues, and the essential parts of the sense-organs; the mesoblast the muscular and connective tissues, including the blood and blood-vessels; the hypoblast forms the epithelium of the alimentary canal, and of the glands which open into it, including the epithelium of the pulmonary air-passages.

Cells vary very much in size, but few are large enough to be visible to the naked eye. Every cell

in the body possesses one or more nuclei, although in some cells, in consequence of chemical alterations, the nucleus may have disappeared completely. Such cells are, however, no longer living and active, and undergo only passive changes; of the living cell the nucleus appears, at least in all the higher animals and plants, to be an essential part.

The main part of the cell, in which the nucleus is embedded, is known as the *cell-body* or *cell-substance*. In most cells it is chiefly formed by a soft albuminous material, named *protoplasm*. It is upon the presence of this material that the so-called 'vital' phenomena which are manifested by the cell obviously depend, such as the amoeba-like movements or changes of shape which are exhibited by free cells such as the white blood-corpuscles; the production, storage, and eventual expulsion from the cell of materials which are to take part in the formation of the secretions, as in the fixed cells of glands; and the internal changes, molecular or molar, which result in the wave-like transmission of impulses or movements, as is seen in the manifestations of activity which occur in muscle and nerve, and in ciliated cells. Most of these changes are or may be produced or modified by the incidence of external stimuli; and the property by virtue of which the protoplasm responds to these or other (unknown) stimuli is termed its 'irritability' or 'excitability.'

When protoplasm is examined chemically in the dead state it is found to be mainly composed, besides water, of certain forms of globulin and albumen, which do not materially differ from those which occur in fluids, such as egg-albumen and blood-serum, which exhibit none of the phenomena of life. But there is reason to believe that the molecular constitution of the protoplasm is very different during life from that which is found after death, being in the former condition of especially unstable character and undergoing changes with great readiness. It is indeed in the highest degree probable that chemical and physical changes are *continually* proceeding during life, and that the chemical changes are of two antagonistic kinds, the one kind tending to produce a building up of fresh protoplasmic substance from the proteid and other material supplied by the blood and lymph, and the other kind tending to produce a breaking down of such substance, and a formation of simpler products. To those two hypothetical antagonistic processes which proceed during life, the terms *anabolic* and *katabolic* have been applied, while to the whole of the chemical changes which go on, both in the individual cells and in the collective organism, the name *metabolism* is given.

The forms assumed by the cells are very various: thus they may be spherical, ovoidal, spindle-shaped, flattened or scale-like, columnar, stellate—in short, of any conceivable shape. Fixed cells retain the same shape for an indefinite time, or undergo only passive alterations; free cells are frequently observed to alter their shape continually by the spontaneous protrusion and retraction of processes of their protoplasm (*pseudopodia*). These spontaneous changes or amoeboid movements may, under certain circumstances, produce an actual locomotion of the cell, as is noticed to occur in the process of *diapedesis*, or passage of the white blood-corpuscles through the walls of the capillaries and venules.

Cells may lie scattered and isolated in a tissue, or they may be united by their processes into a network, as in the connective tissues, or closely packed

and joined edge to edge to form either a membranous stratum or a solid mass of cells, which may constitute the bulk of the tissue to which the cells belong, as in the epithelial tissues. Or the cells of a tissue may undergo special development and structural modification, associated with the assumption of special properties of conduction and contractility, as in the nervous and muscular tissues. In ciliated epithelium the protoplasm of the cell is prolonged at the free border into minute hair-like projections, which during life execute a spontaneous lashing movement (*vibratile cilia*).

In spite of the numerous researches which have been made of late into the structure of cells, very little is known positively regarding the structure of the protoplasm. Until a comparatively recent date it was described as entirely without structure; and in many cells, especially those of the free type, it is difficult to make out any definite appearance of structure, especially in the living condition. It is true that under the influence of certain hardening re-agents, such as alcohol and chromic acid, a reticular appearance is produced in protoplasm, but the same appearance can equally well be produced in solutions of albumen and mucin under similar circumstances, so that the reaction in question by no means proves that the apparent structure is pre-formed; and it is still the opinion of some histologists that protoplasm is essentially structureless. It is certain, however, that in many *fixed* cells a network may be observed even in the living condition, the nodes of the network causing a characteristic finely granular appearance in the protoplasm; and most cytologists accordingly describe the living substance of a cell as being composed of a spongework (*spongioplasm* or *reticulum*), and interstitial matter or matrix occupying the meshes of the spongework (*hyaloplasm* or *enchylema*). Whether the vital phenomena exhibited by protoplasm depend upon the spongioplasm or the hyaloplasm, or belong to both these parts of the living substance, is entirely unknown.

The protoplasm of a cell may contain other materials embedded in its substance, which may either have been formed by the protoplasm from materials obtained from the blood, or in the case of 'free cells' may have been taken bodily into the cell-protoplasm by aid of the amoeboid movements of which these cells are capable (inception of particles). These materials may be in the form of granules of albuminous, fatty, or carbohydrate nature, or of globules of watery fluid containing substances in solution (vacuoles). Such non-protoplasmic ingredients of the cell-substance, when occurring within the cell as ordinary products of its nutrition, are collectively termed *deuteroplasm*. This term also includes such portions of protoplasm as may have become converted within, or at the surface of, the cell into non-living material, as when the external layer of protoplasm becomes transformed by physical and chemical alterations into a firmer and more resisting covering to the cell, which thus becomes invested with a *cell-wall*. Pathological changes in cells are usually due to abnormal chemical transformations of the protoplasm, which in this case are usually termed *degenerations*, such as the *fatty*, *mucous*, and *colloid*.

The protoplasm of many cells, especially of free cells, such as the white blood-corpuscles and the marrow-cells, contains *granules*, embedded in the protoplasm, which are of a proteid or nucleo-proteid

nature and appear to be special products of the cell-metabolism. They are not, however, all chemically identical, for while some are stained more readily by basic dyes (*basophile granules*), others take up acid dyes (*oxyphile granules*), while others react to both basic and acid dyes (*amphophile*), and others again most readily to neutral stains (*neutrophile granules*).

Every cell in the body possesses at least one nucleus, but some cells may have two or three nuclei; and others, such as the giant-cells which are met with in normal red marrow and in certain pathological formations, may contain a large number. The nucleus of a cell is usually spherical; it is situated near the centre of the cell, and appears to possess the function of presiding over the nutritive changes of the protoplasm, as well as that of initiating the division of the cell.

Most nuclei have a well-marked reticular structure, which is visible even in the living cell unaltered by reagents. The filaments of the network have the property of becoming darkly stained by hæmatoxylin and many other staining reagents; hence the substance of which they are composed has been termed *chromatin*, to distinguish it from the part of the nucleus which remains unstained, and which is termed in contradistinction *achromatin*. The filaments are aggregated at the surface of the nucleus into a closely reticulated membrane which serves to limit the nucleus externally. The nodes of the network, as seen in optical sections of the nucleus, give the latter a coarsely granular appearance, especially because the substance of the filaments tends to become aggregated at the nodes. Besides these aggregations there are usually present in the nucleus one or two larger masses of chromatin, staining somewhat differently from the rest, and not always situated at a node of the nucleus-network. To these larger masses the term *nucleoli* is applied. In some nuclei the usual network is replaced by a sort of convoluted skein of nuclear filaments, the latter being somewhat thicker than usual, and exhibiting, when sufficiently magnified, a peculiar transversely striated appearance. This condition is, however, usually only met with in nuclei which are about to undergo division.

In many cells there has been detected in the protoplasm, usually near the nucleus, a minute particle (*attraction-particle* or *centrosome*), which stains in a special manner with certain reagents, and appears to exert an attractive influence upon the neighbouring protoplasm, which shows lines converging towards the centrosome. Prior to the division of the nucleus, the centrosome is always found to have divided, and its two parts are united by a system of fine lines or fibrils. This ultimately becomes developed into a spindle-shaped structure of considerable size, which envelops the dividing nucleus and appears to offer directing lines, along which the parts of the nuclear chromatin, after division, pass towards opposite poles of the cell. It is then known as the *directing spindle*, and each half of the original centrosome is termed a *polar particle*. This, after the formation around it of the daughter nucleus, eventually takes up a position in the protoplasm as the centrosome of the daughter-cell.

The division of the nucleus was formerly believed to occur by a simple process of fission, preceded by the division of the nucleolus. It is doubtful, however, if this simple process of so-called *direct* division occurs regularly in many cells. In nearly every case

of cell-division, both in normal tissues and in pathological formations, the nucleus undergoes a complete series of changes, which are collectively known under the name *karyokinesis* or *karyomitosis*, and which result in the division first of the nucleus and ultimately of the cell (so-called *indirect* division). The changes which are thus undergone are as follows:—The whole of the chromatin, including the substance of the nucleoli and the membrane of the nucleus, first becomes collected into one or more filaments, which have a convoluted skein-like arrangement, the skein being at first closely wound, and its filament or filaments fine, but becoming gradually more open, and its filaments coarser. Next, it is found that the skein has become subdivided into a definite number of short filaments, which often assume a bent V-shaped form, with the apices of the V's directed towards the centre of the nucleus (*aster-phase*). The V's then undergo an alteration in relative position, becoming interlocked at the equator of the now elongated nucleus, and having their apices directed towards the two poles. At or before this *equatorial phase* of karyokinesis the filaments undergo a process of longitudinal cleavage, so that there are now twice as many as at first. The resulting finer V-shaped filaments then begin to pass into two groups, which gradually tend towards either pole of the nucleus, assuming as they pass thither a radiate arrangement, with the apices of the V's towards the centre of each group. This is the *diaster-phase*. Finally, in each of the two groups, the V-shaped filaments unite end to end to form a skein, which subsequently becomes converted into a network, and the formation of the daughter-nuclei may now be regarded as complete. Around each one the corresponding half of the protoplasm of the cell becomes aggregated, so that two daughter-cells have become formed by division of the parent-cell.

While these changes are going on in the chromatin of the nucleus, others are proceeding in the achromatin substance. Within this the spindle-shaped system of fibrils connected with the divided centrosome makes its appearance, both the poles and the equator of the spindle corresponding with those of the nucleus. At each pole of the spindle is the spherical particle termed the *polar particle*, and from this particle other fine fibrils radiate into the adjacent protoplasm, so that a system of achromatin-fibres penetrates not only through every part of the nucleus, but throughout the greater part of the protoplasm of the cell as well. The fibres of the nuclear spindle appear, as already mentioned, to serve as directing lines along which the above-described movements of the chromatin-filaments towards the poles of the nucleus occur, and the equator of the spindle determines the plane of separation both of the nucleus and of the cell.

E. A. SCHÄFER.

CELLULITIS.—Cellulitis is a term used to denote inflammation of the great planes of cellular or connective tissue. The subcutaneous tissue is most often affected: occasionally the intermuscular, mediastinal, pelvic, and other deep layers.

Cellulitis may be *acute* or *chronic*, *circumscribed* or *diffuse*, *non-suppurative* or *suppurative* (see **ABSCESS**: Acute Diffuse Suppuration).

ÆTIOLOGY.—Acute non-suppurative cellulitis is the result either of infection by organisms (usually the pyococci) which fail for one reason or another to

induce suppuration (*see* ABSCESS), or of the entry of some irritant, like urine, into the tissues. The organisms may be (1) inoculated directly through a wound—even the smallest puncture or abrasion; or (2) conveyed to the part by the lymphatics or the blood-vessels from some distant source of absorption, their deposit being often localised by some simple injury; or (3) they may spread to the connective tissue from some adjacent or embedded structure, e.g. from the skin, lymphatic gland, kidney. Alcoholism, albuminuria, and general depression of the resisting power favour all inflammations.

SYMPTOMS.—These are the redness, swelling, heat, and pain of inflammation: more or less widespread, and more or less marked according to the vigour of the inflammatory reaction and superficiality of the process. When the subcutaneous tissue is affected, the part usually ‘pits;’ not so when the inflammation is entirely subfascial. Impaired function and fever are present, but vary much in degree.

Perfect resolution and subsidence of symptoms is the usual ending; but in some cases as the more acute process subsides, and in others, often of unknown ætiology, which have throughout run a chronic course, a less favourable result is obtained; and thickening of tissues, matting together of parts such as tendons and their sheaths, contraction and shrinking of the connective tissue causing marked and persistent displacement of organs (e.g. uterus) may occur (*see* PELVIC CELLULITIS). Repeated slight mechanical irritation will result in the formation of long-lasting thickening. Rarely, as the result of infection by certain very intense irritants, or of greatly impaired tissue-resistance (e.g. in an anasarctous limb), gangrene may occur without any suppuration.

DIAGNOSIS.—The one difficulty is to determine whether or not suppuration is occurring. It is indicated by increasing intensity of the physical signs at one or more spots. Absolute certainty is not of great importance, because if there is good ground for suspecting pus, incision (aseptic) will be beneficial.

TREATMENT.—In the first place disinfect with phenol any source of absorption. Elevate the inflamed part as much as possible and give local rest, or general, if the case is severe. Compression by bandage over cotton wool in large quantities is often valuable; in other cases warm baths, fomentations, or poultices (the skin being unbroken) are more beneficial. Should gangrene threaten, incisions should be made at once and freely. For the treatment of suppuration *see* ABSCESS. In dealing with the results of chronic cellulitis, warm bathing, douching, movements, and massage are useful.

STANLEY BOYD.

CEPHALALGIA (κεφαλή, the head; and ἄλγος, pain).—Pain in the head. *See* HEADACHE.

CEPHALHÆMATOMA (κεφαλή, the head; αἷμα, blood; and ὄμα, a formative suffix indicating a tumour).

DEFINITION.—An effusion of blood occurring in newly born infants, forming a tumour upon the head; situated beneath the pericranium, upon the surface of the skull; or more rarely beneath the skull, between it and the dura mater.

DESCRIPTION.—This disease is of very rare occurrence, and must not be confounded with the *caput succedaneum*, which is an effusion of serum

external to the pericranium, and of common occurrence. The blood is generally extravasated immediately beneath the pericranium, over one of the parietal bones, most frequently the right, but it may occur over the frontal or occipital. Combined with this, or arising independently, but of extreme rarity, may be an effusion beneath the cranium. The origin of cephalhæmatoma has been attributed to a variety of causes, but is most probably due to the constriction of the margin of the os uteri during labour. It is generally observed some hours or a day after birth, as a circumscribed swelling, slightly tense and fluctuating; and its peculiarity consists in a bony circle surrounding and limiting it.

DIAGNOSIS.—These tumours have been mistaken for hernia cerebri; but their situation over the bone away from the fontanelles, the absence of pulsation, and the existence of fluctuation in cephalhæmatoma should prevent this mistake.

PROGNOSIS.—Generally the blood becomes absorbed, but occasionally suppuration occurs, or the bone may become necrosed; if beneath the skull, serious consequences, including idiocy, may ensue.

TREATMENT.—As a rule, cephalhæmatoma is not to be interfered with. If suppuration take place the pus must be evacuated.

CLEMENT GODSON.

CEREBELLUM, Lesions of.—The cerebellum is liable to the same diseases as the brain and nerve-centres generally, such as hæmorrhage, abscess, softening, atrophy, and tumours. The nature of the pathological condition is to be determined by the symptoms peculiar to each, so far as this is possible. Its locality in the cerebellum is to be diagnosed, first, by certain symptoms which are due to the cerebellar lesion as such, which may be termed the *direct* symptoms; and, secondly, by those symptoms which depend more on the influence exerted by the lesion on neighbouring or subjacent centres and structures. These latter may be termed the *indirect* symptoms.

It is by no means easy to separate these symptoms from each other, and to say how much is due to interference with the functions of the cerebellum, and how much to interference with the functions of other parts. There are few diseases which have a purely local organic or functional limitation. Hence, in order to arrive at the symptoms peculiar to cerebellar lesions, it is necessary to exclude all pathological affections which in their very nature affect the whole of the intracranial centres, such as tumours or meningitis. The most satisfactory conclusions, from a pathological point of view, are to be drawn from cases of atrophy or degeneration of the cerebellum, and, from a physiological point of view, from the results of experimental lesions of this organ in the lower animals.

The evidence from these two sources is mutually supporting.

DIRECT SYMPTOMS.—The characteristic symptoms of cerebellar disease are disorders of equilibrium, shown, on attempts at locomotion, in a reeling or staggering gait (titubation), and a continual tendency to stumble or fall over the most trifling obstacle, or on hurried movements.

These symptoms may be confounded with those of locomotor ataxy, but careful observation will show that in cerebellar disease there is no true ataxy. The movements are quite co-ordinated with each other, and are such as would instinctively be made

to prevent falling, or to preserve the equilibrium; and have none of the precipitate, irregular, and sprawling character seen in ataxy. They are not specially intensified on closure of the eyes, which is such a marked feature in ataxy. Nor are they accompanied by any of the sensory affections of ataxy, whether in the form of pains or anæsthesia.

There is no true motor paralysis in cerebellar disease as such, and the various volitional movements of the limbs can be carried out perfectly well in the recumbent posture. Sensation, general and special, is not directly affected. Nystagmus may occur as a direct symptom of cerebellar disease. It would appear as if the more pronounced nystagmoid jerks were on the side of the lesion.

Lesions of the cerebellum may be entirely latent, or not cause any obvious symptoms during life. This has been observed in cases of congenital atrophy, or when the disease has been of a slowly progressive character, or limited to one lobe. Although affections of the middle lobe are specially calculated to induce the characteristic symptoms of cerebellar disease, it by no means follows that lesions of the lateral lobes are without their localising phenomena. Of these may be mentioned: rotatory movements towards the side of lesion, more especially if the middle cerebellar peduncle is also involved, and tremors of the limbs on volitional effort upon the side of the lesion. The knee-jerks are directly affected by experimental ablation, that on the side of extirpation being at first exaggerated. In disease the state of the knee-jerks is not constant, so that this symptom is of little localising value.

INDIRECT SYMPTOMS.—*Pain in the head*, more particularly at the back, though not constantly situated there, is frequently associated with tumour of the cerebellum.

Vomiting is also very frequently observed, perhaps more commonly than in connection with disease of other parts of the brain. There is, however, no reason to regard this as due to cerebellar disease as such. As a general rule, lesions tending to encroach on the space of the posterior fossa, or to increase the pressure in this region, have a similar effect.

Hemiplegia is not uncommon in connection with cerebellar disease, particularly in cases of tumour or hæmorrhage in the lateral lobe of the cerebellum. The hemiplegia is often on the side opposite the lesion. This does not prove that the hemiplegia is due to the destruction of the cerebellar lobe, or that the cerebellar lobes have cross relations with the limbs. Experimental physiology and anatomical investigations tend to show that the cerebellar lobes are functionally related to the motor tracts on the same side. There is however no direct strand of fibres issuing from the cerebellum towards the spinal cord; the cerebello-spinal system being an indirect and uncrossed one through the nucleus of Deiters and the descending antero-lateral tract. The cerebro-cerebellar relation is on the other hand crossed, as old-standing disease or atrophy of one cerebral hemisphere is occasionally associated with atrophy of the opposite cerebellar hemisphere. The hemiplegia from cerebellar disease, whether direct or crossed, is, therefore, in all probability, due to compression or some affection of the subjacent motor tracts, which decussate at the pyramids. The fact that it occurs chiefly when the disease is limited to the lateral lobe is what might be expected on anatomical grounds.

Affections of Sensation, common and special, have been observed in cases of cerebellar disease. Diminution of tactile sensation on the opposite side of the body, when the disease is situated in the lateral lobe, may be explained by the pressure of the new-growth upon the sensory tracts in the tegmentum pontis. As regards the special senses, affections of sight have been most frequently noted. Sight is certainly not abolished by destruction of the cerebellum in the lower animals, and when blindness occurs in man in connection with diseases of this organ, it is due to secondary implication of the optic tracts as the result of neuritis.

A special feature of *tumours* of the cerebellum, more especially of the middle lobe, is a tonic rigidity of the muscles of the back of the neck, with retraction of the head, associated frequently with flexion of the forearms, and extension of the lower extremities with pointing of the toes. In these cases also, psychical affections, more particularly mental hebetude or stupor, occur as the result of secondary dropsy of the ventricles, caused by pressure on the veins of Galen and obstruction of the iter Sylvii. The symptoms then become those of hydrocephalus.

The most important indirect symptoms of localising value are paralysees of the cranial nerves. Paralysis of the fifth cranial nerve is not uncommon with symptoms of cerebellar disease, and points to a lesion of, or involving, the middle peduncle. The facial nerve also is paralysed in some cases of tumour of a lateral lobe extending towards the base of the brain. Of most frequent occurrence is paralysis of the eighth nerve, shown by deafness. Less common is paralysis of the hypoglossal nerve. As a rule paralysis of the cranial nerves shows that the lesion is upon the paralysed side. *Hæmorrhage* into the middle lobe of the cerebellum, in addition to the ordinary symptoms of apoplexy, has been frequently found to cause vascular excitement of the genital organs—in the male marked *priapism*. This symptom, of which several cases were first reported by Serres, led this observer to modify the view of Gall that the cerebellum, as a whole, was related to the sexual instinct, and to regard the middle lobe only as having any such function. The facts, however, are susceptible of a totally different interpretation, and one more in harmony with other data of physiology and pathology. There is absolutely no evidence of the slightest value in support of Gall's hypothesis.

Cerebellar Peduncles.—Cases are on record in which lesions have been found involving principally either the superior or middle cerebellar peduncles. The symptoms, in the main, agree with those observed by Magendie on section of the middle peduncle in the lower animals. The chief effect of this lesion was to cause an irresistible tendency to roll over towards the side of lesion, and this has been amply confirmed by recent experimental work upon monkeys. Together with this rotatory disturbance of the equilibrium, a peculiar distortion of the optic axes was observed, the eye on the side of lesion being directed downwards and inwards, the other looking upwards and outwards. Other symptoms of importance following this lesion were tremors or voluntary movement, such as are seen in cases of disseminated sclerosis.

In a case described by Curschmann, along with appearances of basilar meningitis, which somewhat complicated the case, there was found a focus of softening, surrounded by capillary hæmorrhage,

in the right *superior cerebellar peduncle*. The symptoms observed in this case were a rotatory distortion to the right side, to which position the patient invariably returned when resistance to this movement was withdrawn. There was no motor paralysis, nor was there any distortion of the optic axes. In Bannister's case the patient lay with the head and body in a forced position towards the right side, the lesion being a small hæmorrhage in the right superior peduncle. A case has been put on record by Nonat of apoplectic extravasation into the right *middle peduncle* of the cerebellum and right cerebellar hemisphere. In this case the head and trunk were twisted towards the right side, and the ocular symptoms were also present, the eyes being immovable in a position of skew deviation. Other cases are on record in which the cerebellar peduncles have been involved in more extensive lesions; but the symptoms, though not opposed to those above related, are incapable of differential analysis. The special diagnostic symptoms, therefore, of lesion of the cerebellar peduncles are what are frequently termed 'forced movements' (*Zwangsbewegungen*), or distortions of the normal axis of the trunk. The exact direction of the distortion, in consequence of lesions specially limited to one or other peduncle on the right or left side respectively, is somewhat doubtful, though as a rule it has been found towards the side of lesion. In lesion of the middle peduncle the fifth nerve is most likely affected.

The effects of lesion of the *inferior cerebellar peduncle* are limited to the results of experimental lesion. These are chiefly 'forced movements' and a tendency to rotation to the side of lesion, with adduction of the limbs on the same side, and abduction on the opposite side. In a case of Brissaud's, the cerebellum was also slightly implicated, and the patient showed, in addition to a cerebellar gait, increasing deafness of the ear on the side of the lesion.

D. FERRIER.

W. A. TURNER.

CEREBRAL ABSCESS.—See BRAIN AND MENINGES, Septic Diseases of.

CEREBRAL APOPLEXY.—See APOPLEXY, Cerebral.

CEREBRAL ARTERIES, Diseases of.—See BRAIN, Vessels of; and ARTERIES, Diseases of.

CEREBRAL DIPLEGIA.—SYNON.: Birth-palsy; Little's disease.

DEFINITION.—An affection that is usually congenitally installed, but which may also be due to post-natal causes; whose manifestations commonly date from or soon after birth; whose chief characteristics are bilateral rigidity, motor weakness, and, it may be, perverse movements; and in whose symptomatology are included all degrees of mental deficiency down to complete idiocy. The name 'Little's disease' is commonly limited to the paraplegic form of the affection, which is also known as 'Congenital Spastic Paraplegia' and 'Cerebral Spastic Paraplegia.'

ÆTIOLOGY.—In the majority of cases the disease is congenitally determined; and although no cause can be assigned in many instances, it is possible that such cases are due to some morbid condition of the mother during her pregnancy. The maternal states that are regarded as of consequence are general ill-health, the results of over-work and

of defective feeding, neurasthenia, syphilis, specific fevers, eclampsia, blows to the abdomen, and abnormal psychic states. Failure of the physiological process of generation plays an important rôle in the ætiology of some cases, and it is noteworthy that the youngest members of large families are especially liable to be affected, while pregnancies subsequent to the birth of a diplegic child may result in still-births. Consanguinity of the parents has been noted in some cases, and hereditary transmission of the disease from a parent to her offspring has occurred. There is one form which is met with almost exclusively in the Hebrew race.

Disorders of parturition account for some cases, though the frequency of this cause appears to have been over-estimated. The causes included in this group are: premature birth, prolonged labour, precipitate labour, instrumental or other injuries at birth, and asphyxia neonatorum; although the evidence that premature birth is a cause is not convincing.

Post-natal affections of the child claim only a subsidiary place in the ætiology of cerebral diplegia; nevertheless, acute specific fevers, encephalitis, vascular lesions, and epilepsy appear at times to be in direct causal relationship. It is, however, possible that, in at least some of the cases, these post-natal factors may only serve to bring out a congenitally installed disease.

SYMPTOMS.—Several clinical forms of the affection are recognised according as rigidity and motor weakness or spontaneous movements dominate the picture; according to the distribution of the rigidity and motor weakness; and according to the degree of mental impairment that is present. The forms are: (1) Generalised rigidity, (2) Paraplegic rigidity, (3) Bilateral athetosis, (4) Choreiform diplegia, and (5) Profound mental defect associated with slight generalised rigidity. Various combinations of the different varieties are met with; but the cases of generalised rigidity and those of paraplegic rigidity far outnumber all other forms of the affection.

The symptoms usually date from birth; stiffness of the limbs may first attract attention, or, when present, athetoid or choreiform movements may first be noticed. When the defect is slight, nothing abnormal may be observed until the child commences to crawl or walk. In other cases mental deficiency, including backwardness in talking, is the first prominent symptom. Concomitant congenital defects, such as strabismus, nystagmus, or pes cavus, may supply a clue to the true nature of the malady.

When the disease is post-natal in origin it is commonly ushered in by febrile manifestations, and, it may be, vomiting and convulsions. The rigidity in the limbs develops in a few days, or, when there have been convulsions, paresis may precede the rigidity. In other cases there are no premonitions; the rigidity and motor weakness gradually appear without obvious cause, or they may follow some injury to the head.

Although motor weakness and rigidity are both present, the latter is usually greater than the loss of motor power. All parts of the body may be affected, but not in equal degree; the legs suffer most and are alone affected in the paraplegic form; the arms suffer less, and the face least of all. The arms may, however, be more affected than the legs in some cases, and still more rarely the face may be more affected than either the arms or legs. All

movements are executed slowly and in a clumsy manner, and they may be further hampered by contractures. Patients capable of walking may do so on their toes, as contracture of the calf-muscles may prevent the heels from reaching the ground; the knees may be partly flexed owing to similar contracture of the hamstrings, and spasm of the adductors of the thighs may occasion cross-legged progression. In pronounced cases of generalised rigidity the sitting posture may be impossible, and in the paraplegic form the legs may remain extended when the patient sits. Various attitudes are assumed in cases of generalised rigidity. The patient may be curled up into the smallest possible space, with the head and trunk bent forward, the elbows pressed to the sides, the forearms crossed over the chest, and the legs drawn up in extreme flexion at the hip and knee. Or an 'attitude of adoration' may be assumed, with the head drawn back, the eyes staring, lordosis of the spine, the elbows pressed to the sides, and the hands supinated below the face, while the legs are rigidly extended. Instead of either of these postures, all the limbs may be rigidly extended, and the forearms supinated: more rarely they are markedly pronated. Perverse movements of different kinds may be seen, notably facial over-action, grimaces, athetoid or choreiform movements, and intention-tremor; and it is especially the upper limbs that are thus affected. One or other form of spontaneous movement may be present alone, or they may be combined, while rigidity and paresis are usually only slight in such cases. In athetosis the movements are slow and are most marked in the distal portions of the limbs; the face is often similarly affected, and even the tongue may participate. In choreic diplegia, on the other hand, the movements are quick and shock-like, resembling those seen in myoclonus multiplex; they are most marked in the proximal segments of the limbs, and they are only rarely seen in the face. The muscles are unduly firm in some cases, and if athetosis is a feature they become hypertrophied; but in the late stages of severe generalised rigidity the muscles waste, a feature that is especially characteristic of the form of diplegia that is met with in Jews. Fibrillary tremors of the muscles are common, especially in adult cases.

The tendon-jerks are increased, though they are often difficult to elicit owing to the great rigidity of the limbs, and it is not usual to obtain ankle-clonus. Babinski's sign is almost invariably present. Cutaneous sensibility and the muscular sense are not impaired. The sphincters are not affected. The pupils may be unequal, and may react sluggishly to light. Coarse spontaneous nystagmus is common, and convergent strabismus is present in a good many cases. Primary optic atrophy is also frequent, and is constant in the form of diplegia that occurs in Jews; another characteristic of this form of the disease is a whitish-grey patch in the macular region about twice the size of the optic disc, with the fovea centralis appearing as a cherry-red spot in the centre of the patch. When affected, the face may have a 'starched' appearance owing to lack of change of expression; the palpebral fissures are wide, the lids retracted, and the mouth usually widely open. The tongue is often unduly large, and can only be protruded with difficulty. The arch of the hard palate is commonly high and narrow. Difficulty in swallowing is common, and dysphonia or stridor may occur.

There may be no mental impairment, or all degrees of defect may be met with down to complete amentia; but there is no relationship between the degree of mental impairment and the amount of physical disability: the former may be great, while the latter is slight, and *vice versa*. The patients are as a rule placid; but they may be mischievous, irritable, and passionate. The child may never learn to speak; it may lose this faculty if it has already been acquired; or it may be backward and may ultimately only speak very indistinctly. Convulsions may occur, and are more common in cases of generalised rigidity than in cases of spastic paraplegia; they are also frequent in cases with choreiform movements and in bilateral spastic hemiplegia. Permanent epilepsy may be established in some of the patients. Deformities of the skull are common, notably microcephaly in congenital cases; while in some there is hydrocephalus and in others the skull is asymmetrical. Deformities of the spine are also frequent, especially kyphosis; but there is often scoliosis and sometimes lordosis. Pes cavus is often present; sometimes there is talipes equino-varus, and sometimes genu recurvatum. The development of the affected parts is retarded, though this is less easy to determine than in infantile hemiplegia where there is the normal side for comparison.

MORBID ANATOMY AND PATHOLOGY.—The morbid changes found are not always the same, and they moreover differ according as an examination is made while the initial lesions are still in existence, or when only the secondary consequences of these remain. The brain may show no abnormality to the naked eye, although degenerative changes are evident on microscopical examination. There may, on the other hand, be pronounced atrophy of the convolutions, with a worm-eaten appearance of their surface, and increase in the width of the sulci. Instead of being uniform, however, the atrophy may be limited to some parts, so that a knobbed appearance results from alternate shrinking and projection of the convolutions. Or the changes may be even more gross, and porencephalic cavities may be present.

Cases that have come to necropsy at an early stage have shown marked degeneration of the neurons of the cerebral cortex, without evidence of interstitial or inflammatory changes; whereas those examined years after the onset of the disease have shown sclerosis of the brain, with increase of the glial tissue, thickening round the blood-vessels, disappearance of the nerve-elements, and, in some cases, porencephalus. The lesions are superficial, whereas in bilateral spastic hemiplegia they are deep-seated.

There are various views as to the probable nature of the initial lesion in cases of diplegia; but it appears probable that primary degeneration of the neurons of the cerebral cortex accounts for most cases, and that the degeneration is due to the action of some toxic agent. Other lesions to which the condition has been attributed are encephalitis, meningeal hæmorrhage, hæmorrhage into the substance of the brain as a result of trauma or asphyxia neonatorum, embolic occlusion of branches of the middle cerebral arteries, and thrombosis of cortical veins, and of the longitudinal sinus. Finally, there are the views that some cases of cerebral diplegia are due to arrest of development, and that premature birth before the pyramidal tracts are fully developed accounts for others.

The pyramidal tracts in the spinal cord show a variable amount of descending degeneration in most cases; but in a few instances they are said to have been normal. These tracts have also been absent, which suggests that the disease of the brain occurred before the pyramidal fibres had developed; but in other cases they have been merely reduced in size, possibly as a result of a lesion limited to part of the motor areas occurring at an equally early period of development.

DIAGNOSIS.—Several affections may be confounded with cerebral diplegia. In *double hemiplegia* paralysis is in excess of rigidity, the arms suffer more than the legs, and defective development of the affected parts is more pronounced. Competent observers state that *disseminated sclerosis* occurs in children; but a large proportion of the supposed cases have probably been cases of cerebral diplegia. *Friedreich's disease* usually develops later in childhood; there is no spasticity, and the knee-jerks are nearly always absent. *Hereditary cerebellar ataxy* does not give rise to symptoms until still later in life, and, except that the knee-jerks are increased, there are no spastic phenomena; optic atrophy is a constant feature. *Family spastic paralysis* appears later than cerebral diplegia, and does not give rise to any symptoms that indicate affection of the brain. In *compression-paraplegia from spinal caries* the distinction is easy; cerebral symptoms are here absent, there is usually anaesthesia and loss of control over the sphincters, and tenderness and angular deformity of the spine are common.

PROGNOSIS.—The course of the disease varies in different cases; in some it is progressive and terminates in death, a result which is almost constant in the form that occurs in Jews, and which is common in cases of severe generalised rigidity. Instead of this the disease may reach a certain stage and then remain stationary; and in a third group there may even be some amelioration after a time, a result that is especially likely to occur in the paraplegic form and in cases of slight generalised rigidity.

TREATMENT.—Careful nursing, warm clothing, and proper feeding are essential. Cod-liver-oil, extract of malt, and cream are all useful as foods, and some preparation of iron is the most suitable tonic. No effort should be spared in training the child so as to improve its mental and physical powers. Where there is much cerebral defect special training is required by a person accustomed to teach children who are mentally deficient. Massage, passive movements, and appropriate gymnastic exercises are of great value in improving the physical development, and in preventing deformities, which must further be guarded against in every possible way. When deformities have already occurred much good may still be done by regulated exercises, passive movements, and slow extension or division of the contracted tendons.

J. S. RISIEN RUSSELL.

CEREBRAL HÆMORRHAGE.—See BRAIN, Hæmorrhage into.

CEREBRAL LOCALISATION.—See BRAIN, Tumours of.

CEREBRAL SPASTIC PARAPLEGIA.—See CEREBRAL DIPLEGIA.

CEREBRO-SPINAL MENINGITIS.—See MENINGITIS, Epidemic.

CEREBRUM, Diseases of.—See BRAIN, Diseases of; also CONVOLUTIONS OF THE BRAIN, Diseases of; CORPUS STRIATUM, Diseases of; &c.

CERTIFICATES.—See LUNACY LAWS.

CESTODA.—See ENTOZOA.

CHALICOSIS (χάλις, gravel).—SYNON.: Sili-cosis, Stone-mason's phthisis. See PNEUMOCONIOSIS.

CHALK-STONES.—This name is applied to the deposits which are formed in connection with the gouty diathesis, especially in the joints. They are thus denominated from their appearance and physical characters, in which they more or less resemble chalk; but in their chemical composition they are entirely different, consisting mainly of urate of sodium. See GOUT; and CONCRETIONS.

CHALYBEATE WATERS (*chalybs*, steel).—Mineral waters which contain iron. See MINERAL WATERS.

CHANCRE (Fr. *chancre*).—Hard chancre is the initial manifestation of syphilis. See SYPHILIS. For Soft chancre, see VENEREAL DISEASE.

CHANGE OF LIFE.—SYNON.: Climacteric epoch; Sexual involution; Fr. *Ménopause*; Ger. *Menstruationsende*.

DEFINITION.—The time of life in a woman when the functions of the uterus and ovaries cease, menstruation terminating—a period when disease of these organs is especially prone to occur, and when various constitutional disturbances are almost certain to arise.

ANATOMICAL CHARACTERS.—Great changes occur in the sexual organs—the ovaries lose their smooth outline, and after a while become shrivelled up, occasionally only a trace of them remaining; the Fallopian tubes diminish in size, and sometimes become obliterated; the walls of the uterus atrophy, its cavity becomes much smaller, and the cervix disappears altogether.

SYMPTOMS.—The term 'change of life' is used among women very widely to signify everything which affects them at this critical time. It is so rare for the transition from activity to inactivity to take place without some disturbance locally, or constitutionally, that women are apt to neglect seeking advice for symptoms which should demand careful treatment, believing as they do that it is natural to suffer in such ways at 'the change.' There is no fixed period for the climacteric epoch, though roughly it may be said to occur between the ages of forty-five and fifty. Certain causes are apt to determine the time—among these are parturition and lactation, febrile attacks, such as typhus or acute rheumatism, profuse hæmorrhages, fright, &c.

The symptoms vary much. In some women the change is abrupt, menstruation ceasing all at once after perfect regularity; in others and more frequently, the change is prolonged, the catamenia being irregular for many months, and varying as to periodicity and quantity. Frequently, after a long interval, a profuse flow with clots occurs, and this is very often attributed to a miscarriage. This loss is frequently beneficial, and if it do not take place, and if relief be not derived from vicarious discharges, such as bleeding from piles, the excess of blood

gives rise to headaches, flushes, vertigo, and a host of other unpleasant symptoms. The balance between the nervous and circulatory system is upset; irritability of temper, hyper-sensitiveness, and all sorts of fancies arise, or depression sometimes amounting to melancholia ensues. If germs of disease exist, the uterus is especially prone at such times to develop them, so that carcinoma, fibroid disease, and polypus frequently present their first symptoms at this epoch. The importance, therefore, of an early examination cannot be too forcibly dwelt on, or the mischief of delay from considering the abnormal condition as typical of 'the change of life,' and as a natural consequence, which will right itself. At these times pruritus of the vulva, vascular growths at the orifice of the urethra, and cutaneous eruptions are especially likely to occur. There is a tendency to grow fat, and to become coarse; hairs frequently appear on the face. The breasts often become very large and pendulous, and this, with the increase in the size of the abdomen from flatus, and the deposition of fat in its walls, together with the cessation of menstruation, not infrequently gives rise to the supposition of pregnancy. To this imaginary state the term *pseudocyesis* has been applied, and it is often almost impossible to set aside the opinion of the woman regarding her supposed condition. The headaches, neuralgia, loss of memory, and nervous symptoms appear to be due to disturbance in the ganglionic system of nerves, with which the uterus and ovaries are largely supplied. If insanity arise, the most common form it assumes is hypochondriasis or melancholia.

TREATMENT.—This must be directed to regulating the secretions. Generally constipation, previously troublesome, becomes aggravated; and portal congestion frequently occurs. Saline purgatives are especially beneficial, and these may be administered in the form of mineral waters, such as Rubinat, Hunyadi János, Condal, or Friedrichshall. Blue pill with aloes is often very useful. The headaches and reflex nervous symptoms may be best combated by the administration of bromide of potassium, and this drug appears to act as a direct sedative to the sexual organs, besides diminishing the amount of blood determined to them. Occasionally, bleeding from the arm or cupping gives great relief. Attention must above all be paid to the diet. It should be plain and unstimulating: beer and spirits should be prohibited, and only light wines, if any, allowed. Tepid baths are useful. Late hours, heated rooms, and excitement of all kinds should be avoided. If local troubles arise, they must be treated according to their indications. It is clearly impossible to map out any empirical line of treatment for a condition in which the symptoms are so variable.

CLEMENT GODSON.

CHAPPED NIPPLES.—See BREAST, Diseases of; and NIPPLE, Diseases of.

CHAPS.—SYNON.: *Rhagades*; *Rimæ*.—Cracks or fissures of the skin occur where the integument has lost its elasticity and become hardened by infiltration. *Rimæ* not infrequently extend deeply and may bleed, and are then very painful. Their favourite sites are the hands and wrists in cold weather, the lips, and the skin around joints. The treatment for chaps consists in protection from the atmosphere; careful drying after previous thorough inunction with glycerine. The skin will thereby

be kept elastic and moist. Glycerine of starch, glycerine and rose-water, 'cold cream,' and hazeline-cream are also efficacious remedies.

CHARCOT'S DISEASE.—See TABES DORSALIS.

CHARCOT-LEYDEN CRYSTALS.—Colourless octahedral crystals found principally in cases of leucocythæmia (blood and sputum), and of bronchial asthma (sputum). They contain phosphoric acid combined with a base, generally called 'spermine.' The crystals are believed to be identical with those obtained by the evaporation of normal semen.

CHEIROPOMPHOLYX.—See DYSIDROSIS.

CHELOID (χηλή, a claw).—SYNON.: Keloid; Alibert's Keloid; Cheloma; Fr. *Chéloïde*; *Cancroïde*; Ger. *Keloid*; *Knollenkrebs*. The phonetic spelling 'keloid' has given rise to false derivations from κήλη, a tumour, and from κηλís, a stain or mark.

DEFINITION.—A disease allied to tumour-formation, causing overgrowth of scar-tissue.

DESCRIPTION.—Cheloid starts as a small rounded excrescence of a white or pink colour, abruptly elevated above the surrounding skin. This increases slowly in area and tends to throw out irregular lateral projections, which have been compared to claws. As it grows the centre of the cheloid-nodule may become slightly cupped, and its surface puckered owing to contraction of the fibrous tissue of which it is composed. The edges are often hyperæmic, and enlarged vessels may be visible coursing over the tumour. The growth arises in the scar caused by some injury, which may be so slight as to have escaped previous notice: thus not only the scars of wounds or burns may be affected, but also those left by leech-bites, acne, small-pox, or the operation of piercing the ears for adornment with ear-rings. The commonest seat of cheloid is over the sternum, but any part may be affected. The tumour when it has once appeared tends to increase slowly for an indefinite time, but spontaneous disappearance may occur. The growths are often tender on pressure and may be the seat of more or less severe pricking or stabbing pain. They may be multiple and are occasionally distributed symmetrically, suggesting a nervous origin.

Cheloid was formerly divided into two varieties, the 'true' and the 'false,' the former appearing spontaneously, the latter only in pre-existing scars. It is, however, probable that in the apparently spontaneous cases the original lesion was so minute as to be overlooked. The term 'cheloid' was applied by Addison to the condition now known as morphea or localised scleroderma, but this confusion of terms has been abandoned.

PATHOLOGY.—The process of formation of a cheloid-growth consists in a multiplication of connective tissue-cells along the course of the blood-vessels. Appearing first as round cells, the new elements develop into bands of fibrous tissue. Microscopically vessels may be found affected at considerable distances from the actual growth. This is situated in the corium, the epidermis and papillæ being at first intact; later on the skin becomes stretched over the tumour and the papillæ may disappear, but the epidermis remains uninvolved.

TREATMENT.—This is unsatisfactory, as the growth generally returns after removal: occasionally however this may be successful if the knife be carried very wide of the tumour. Deep transverse incisions into the growth have been recommended. Continuous pressure may be tried in parts which admit of this treatment, but care is necessary to prevent the occurrence of ulceration. Electrolysis has proved effectual in some cases in causing disappearance of cheloid-nodules and appears worthy of more extended trial. Good results have seemed to be due to inunction of mercurial ointment, but in estimating the value of treatment it is necessary to bear in mind the fact that these growths may in some cases disappear spontaneously. If the pain in the cheloid is severe, injections of cocaine into the neighbouring tissues may afford relief.

W. CECIL BOSANQUET.

CHELTENHAM, in Gloucestershire.—Common salt waters. See MINERAL WATERS.

CHEMOSIS (χῆμη, a hole).—A swollen condition of the conjunctiva, caused by effusion into its tissue around the cornea, which thus appears as if placed in a hole or hollow. See EYE, AND ITS APPENDAGES, Diseases of.

CHEMOTAXIS (χημεία, chemistry; and τάξις, arrangement).—The property possessed by certain cells and fluids of attracting or repelling other cells. See IMMUNITY; and PHAGOCYTOSIS.

CHEST, Deformities of.—Under this head are included all deviations in shape from the normal chest.

Deviations from the shape of the typical thorax are appreciable by careful physical examination. Of the various methods employed for this purpose, by far the most valuable are inspection and palpation. Although in some few cases it may be important to determine the exact amount and nature of the deformity by a measuring tape, calipers, or cyrtometer, there are very few deviations in shape or size of the thorax, the degree of which cannot be sufficiently estimated for clinical purposes by the eye and hand.

Deformities of the chest may be due either to abnormality of the parietes, or to disease of internal structures.

DESCRIPTION.—Deviations from the form or size of the typical thorax may be either *general* or *local*—that is, the abnormality may involve the whole thorax, or a part only.

1. General Deformities.—**1. General Diminution.**—The chest may be too small—that is, diminished in all its diameters without being in other respects deformed. This is due to an increase in the obliquity of the ribs. The angle formed between each of the true ribs (excepting the first) and its cartilage is more acute than normal. The intercostal spaces of the true ribs are widened about the junction of the ribs with their cartilages, and at the same time the ribs posteriorly are approximated more closely to each other, the closeness of the approximation being in proportion to the diminution in the size of the thorax. The vertical diameter of the thorax is lessened by an increase in the height of the arch of the diaphragm. The very oblique position of the false ribs, and the height to which the diaphragm rises into the chest, cause several of the false ribs to lie in contact with the diaphragm,

and thus no portion of lung is under these ribs: they are, practically speaking, no longer part of the chest-walls. The sub-costal angle is diminished in proportion to the diminution of the size of the thorax—that is, to the obliquity of the ribs. The obliquity of the ribs also causes the shoulders and the sternal ends of the clavicles to droop, and at the same time to incline forwards; the upper part of the scapula is carried by the shoulder forward, the inferior tilted backward.

General and symmetrical diminution in the size of the thorax has only one cause, namely, small size of the lungs. Small lungs may be congenital, due to developmental peculiarity; or the consequence of atrophic degenerative changes incident to age. In both these cases the lungs are, in relation to the length of the ribs, disproportionately small, and, as a necessary consequence, the relatively too long ribs are arranged more obliquely than they are in a well-formed chest, and the diaphragm is pushed by the abdominal organs higher into the thorax. When the small size of the lungs is due to atrophy, the supra-clavicular fossæ are deepened, and the vertical diameter of the chest proportionately diminished. In advanced life the congenitally small lungs are frequently reduced still further in size by the super-vention of atrophic emphysema. It is the congenitally small lungs of childhood which are prone to become the seat of tuberculosis in youth, and the subjects of atrophic emphysema in old age.

2. General Enlargement.—The thorax may be too large, increased in all its diameters, without being otherwise deformed. It is simply bigger than it should be, having regard to the height of the subject.

When the thorax is abnormally large, the ribs are placed more horizontally than they are in the normal thorax. The angle formed between each rib and its cartilage is greater than in health; while the intercostal spaces, especially the lower, are widened, and the ribs less closely approximated, the arch of the diaphragm is lessened in depth, and a considerable mass of lung lies under the lower false ribs, between them and the diaphragm. The chest is increased in all its diameters. The shoulders are raised. The sub-costal angle is greater than natural.

Increase in the size of the whole thorax has but one cause, namely, increase in the size of the lungs. General and fairly uniform increase in the size of the lungs is the consequence of disease, and of one disease only, namely, hypertrophic emphysema. When the increase in size of the thorax is moderate in degree, this is effected by the altered position of the ribs; but when the lung-disease is extreme, then a certain amount of the enlargement is caused by pressure on the inside of the chest during the violent expiratory efforts of severe cough.

3. Irregular General Deformities.—In the deformities above described the antero-posterior and the lateral diameters retain more or less perfectly their normal proportion—both are increased or both are diminished; in the former case the chest is on the whole more barrel-shaped than natural, but the deviation from the normal form is not considerable. If, however, the chest-walls are from any cause unduly soft or unduly rigid, then the actually or relatively soft portions will fail to expand or may actually recede during each inspiratory act, and local deformity of the chest follows. The diameter of the chest at the part where the absolutely

or relatively soft portion of the parietes is placed will be diminished. The special deformities of the chest which result are due, therefore, primarily to the state of the parietes, and are not, as those previously described, secondary to conditions of the lungs themselves.

a. Diminution in the antero-posterior diameter of the thorax.—The antero-posterior diameter of the thorax is frequently less than that of the normal thorax, the lateral diameter being proportionately increased. The chest has an oval form—it is flattened from before backwards.

The thorax flattened from before backwards is usually associated with small lungs, but the mechanical cause of the flattened form is the want of full resisting power in the ribs without loss of rigidity in the cartilages. These conditions of thorax are common in the subjects of tuberculosis.

The flattening of the thorax is increased by all impediments to the free passage of air through the air-tubes. In some children suffering from even slight bronchial catarrh, the flattening of the chest is seen to be increased at each inspiration; and if the impediment to the entrance of the air to the pulmonary tissue be constant or extreme, not only is the flattening increased at each inspiration, but the sternum is also depressed, especially at its lower half, below the level of the costal cartilages, and thus the antero-posterior diameter of the thorax is still further diminished in the median line.

b. Increase in the antero-posterior diameter of the thorax.—In rickets the cartilages of the ribs are very firm, while the adjoining growing ends of the ribs are softer than natural. The consequence of the extreme softness of the ribs at this part is that at each inspiration the weight of the atmosphere presses inwards the softest part of the ribs, while the sternum is borne forwards by the firm cartilages. The result is great increase in the antero-posterior diameter of the thorax, and diminution of the lateral diameter at the parts corresponding to the softest part of the ribs. The depression of the softest part of each rib is increased by the want of resilience of the softened structures.

A groove is thus formed in the thoracic walls just outside the ends of the costal cartilages; and this groove being deepened at each inspiration, the part of the lung adjacent is compressed in place of being expanded during the inspiratory act. At the same time, in consequence of the cartilages and sternum being thrust forward at each inspiration, air enters with undue force into the lung-tissue subjacent to these parts. The consequence of the excessive expansion of the anterior part of the lung is vesicular emphysema, and the recession during inspiration of the softened and imperfectly resilient and therefore deeply grooved part of the chest-wall leads to collapse of the subjacent pulmonary tissue; and, as the effect of these two conditions, the lungs, when the chest is opened, present a vertical groove corresponding to the groove in the chest-walls. The antero-posterior diameter of the thorax in rickets is still further increased by the curvature of the spine. The muscles are weak, the child is unable to sit upright, that is to say, it is unable, in consequence of the weakness of its muscles, to support the weight of the upper part of its body; the bones of the spine are, in common with the other bones of the body, softened; and the result of the weakness of the muscles and the softness of the vertebræ is the dorsal bow.

When deformity of the chest is the result of undue softness of the chest-walls, the position of the solid organs subjacent to the parietes is frequently perceptible to the eye. The liver supports the lower ribs on the right side, the heart supports the ribs and cartilages over it on the left side, and thus these organs cause local prominence of the chest-walls, without being themselves in any way abnormal.

In the so-called *pigeon-breast*, the antero-posterior diameter of the thorax is increased in the middle line, the lungs are small, the ribs and cartilages are firm, the ribs are placed obliquely and the chest-walls are flattened laterally, and the sternum as a consequence is thrust forwards; thus a horizontal section through a pigeon-breasted chest has a triangular form, the apex of the triangle being the sternum. Impediment to the free entrance of air into the lower lobes of the lungs will favour the production of and increase the deformity. The chests of children who suffer from repeated attacks of bronchitis may exhibit this deformity, while there is increased expansion and subsequent enlargement of the upper part of the chest, the lungs being more or less collapsed below and emphysematous above.

c. Transverse anterior constriction of the lower part of the thorax is the consequence of small size of the lungs, or of imperfect inspiratory expansion, permanent or frequently recurring in youth. In these cases the lower ribs are little used in respiration, while below they are borne outwards or supported by the liver, stomach, and spleen, and thus an imperfectly formed transverse depression is produced in the front of the chest on a level with the base of the ensiform cartilage (*Harrison's sulcus*).

II. Local, Asymmetrical, and Unilateral Deformities. — 1. *Fulness of the supraclavicular region.*—The supraclavicular region, corresponding to the portion of the thoracic cavity above the clavicle, may be fuller than natural. The causes of this local bulging are—*a.* Development of adipose and cellular tissue as in myxœdema. *b.* Distension of the deep-seated veins. *c.* Enlargement of lymphatic glands in a stout person. *d.* Large-lunged emphysema, in which disease there is occasionally distension of that part of the cavity of the thorax which lies above the level of the clavicle due to pressure within the lungs during the powerful expiratory effort of cough.

2. Depression of one supraclavicular fossa is caused by any pathological condition of the apex of the lung which produces diminution of its bulk, for example, atrophic emphysema, or chronic consolidation of the apex.

3. Elevation of one shoulder.—Occupation is a common cause of elevation of one shoulder; thus in clerks, who sit much at the desk, the left shoulder is permanently a little higher than the right, and the upper portion of the spine is slightly curved, the convexity being to the left; so in those who carry heavy weights on one arm, the opposite shoulder is elevated and the spine curved. Whatever necessitates an increase in the capacity of one side of the thorax causes elevation of the shoulder on the same side; thus, considerable dilatation of the heart, fluid in the pericardium, fluid in the pleura, aneurysm of the arch of the aorta or of the innominate artery, all lead to elevation of the shoulder. The shoulder is depressed and carried forward when, from any cause, the whole or upper part of one side of the chest is diminished in size, as when the apex of the lung is the seat of fibrosis in chronic tuberculosis.

4. *Uniform dilatation of one side* of the thorax is due, with one exception, to fluid or air in the pleura. The exception is those rare cases of malignant tumour of the lung, in which the new-growth is uniformly diffused through the lung-tissue, and in amount so great that the affected lung decidedly exceeds in bulk the healthy lung inflated with air by inspiration.

In uniform dilatation of one side of the thorax, the shoulder is raised, the ribs are placed more horizontally than on the healthy side, the intercostal spaces are widened, and the spine is slightly curved. When the enlargement is moderate in amount, the increase in capacity is effected by the altered position of the ribs; but when the increase in size is very considerable, then it is due in part to the pressure exercised by the air, fluid, or cancer-loaded lung on the inner side of the chest-wall.

5. *Uniform contraction of one side* of the thorax is the consequence of any condition which leads to general and uniform reduction in the size of the lung, such as cancer of the lung, chronic tuberculosis of the lung, chronic pneumonia, or the change in the texture of the lung which follows long-continued compression by fluid in the pleura. When the whole of one side of the thorax is reduced in size, the shoulder on that side is depressed, the ribs are placed more obliquely and are more closely approximated than on the opposite side, the intercostal spaces are narrowed, and the spine is curved, often considerably, the concavity of the curve being towards the contracted side.

6. *Lateral curvature of the spine*, instead of being the consequence, may be the cause of deformity of the thorax: the ribs are then approximated on the side and at the part where the concavity of the curvature is placed, while they are separated and the shoulder is raised on the side of the convexity.

7. In *angular curvature of the spine* the deformity of the thorax varies with the seat and the extent of the vertebral disease; but, speaking generally, it may be said that in angular curvature of the spine, while the capacity of the chest is much diminished, the antero-posterior diameter of the thorax is increased in proportion to the amount of destruction of the bodies of the vertebrae, and that the ribs are in a corresponding degree approximated.

8. *Extreme depression of the lower part of the sternum* is the consequence of softness of the cartilages of the ribs, and impediment to the free passage of the air to the pulmonary tissue. This deformity is never congenital, although the subjects of it often affirm it to be so; it may, however, commence to be formed directly after birth if there be a congenital impediment to the entrance of air into the lungs—for example, atelectasis. Later on, it may result from other impediments, especially if two or more co-exist—for example, post-nasal adenoids, enlarged tonsils, and chronic bronchitis.

This deformity may be the result of direct pressure. In certain occupations pressure has to be exerted on the lower part of the sternum—thus, some shoemakers use a wooden instrument which has to be kept in its place by pressure against the lower part of the sternum. For direct pressure to produce this deformity it must have been applied in early youth, while the parts are still flexible, and have been exerted frequently over a long period of time.

9. *Congenital deformities* of the thorax are few in number and are due to arrest of development—for

example, cleft sternum, and defective formation of one or more ribs or cartilages. See MONSTROSITIES and MALFORMATIONS.

10. *Asymmetrical diminution in size of a part* of the thorax is produced by any pathological change which reduces the size of the subjacent part of the lung. Chronic tuberculosis in the apex of the lung is attended by diminution of the bulk of the part of the lung which is the seat of the lesion. Considerable loss of pulmonary tissue is usually accompanied by falling inwards of the chest-wall over the cavity. The formation of a cavity is often attended by some fibrosis, and this increases the local depression of the chest-wall. In chronic thickening of the pleura, the chest-wall at the part is, by the contraction of the new fibrous tissue, drawn inwards, and the lung subjacent to the thickened pleura being often involved in the same process, the chest-wall is also forced in during inspiration by atmospheric pressure. Hence, after pleurisy, limited in extent, it is common to find permanent flattening of the thoracic parietes at the base of the chest on the side affected.

In cancerous infiltration of the lung, limited in extent, the lung-tissue is sometimes so much condensed that the bulk of the cancer and lung is less than that of the healthy lung, and the chest-walls as a consequence are flattened over the seat of disease.

11. *Asymmetrical localised bulging*.—If the ribs are, in relation to the size of the lungs, disproportionately long, and their cartilages soft, then one or more of the cartilages may be knuckled forwards; the cartilage, being compressed between the end of the rib and the sternum, bends in an angle outwards. Although the prominence is trifling, it often causes anxiety to parents and its subject. Local deformity of this kind is occasionally the result of repeated lateral compression of the chest-wall in the athletic sports of young boys, such as cricket.

All the diseases of the chest which are accompanied by general enlargement of both or one side of the chest, when localised, are attended by local bulging; thus, a common cause of abnormal fullness of the lower part of the left side of the thorax, posteriorly, is emphysema of the corresponding part of the lung; and a moderate amount of fluid in the pleura is attended by fullness of the lower part of the chest on the same side. In both these cases the ribs are raised into an abnormally horizontal position; the chest-walls are not pushed outwards, but the ribs are raised, and the intercostal spaces are to that extent widened. The ribs are put into the position which gives the greatest capacity to the thoracic cavities containing the fluid or the enlarged lung. Local bulging may be produced by aneurysm of the arch of the aorta or of the innominate artery; by growths, malignant or other, within the chest; by chronic pleurisy with effusion circumscribed by dense false membrane; by hydatids; or by abscess; and in all these cases the prominence is due to direct pressure on the inner side of the chest-wall, and to changes in the chest-wall itself.

Hypertrophy and dilatation of the heart and fluid in the pericardium are attended by fullness of the præcordial region. The bulging from these diseases is much greater in the child than in the adult. In these cases a little of the fullness is produced by a more horizontal arrangement of the ribs; but when the prominence of the præcordial region is at all considerable, it is the result of the pressure exercised by the fluid or by the large and powerfully acting

heart on the inner surface of the corresponding part of the chest-wall.

At the part corresponding to the junction of the first and second bones of the sternum, opposite the cartilage of the second rib, the sternum projects forwards. This prominence is called the angle of Ludovicus. Any impediment to the free entrance of air into the lungs may cause depression of the lower part of the sternum; if the ossification of the sternum is not complete at the junction of the first and second bones, undue prominence of this part is the result. Subsequently a formation of bone takes place at this spot, and increases the prominence.

WILLIAM JENNER.

H. MONTAGUE MURRAY.

CHEST, Examination of.—See PHYSICAL EXAMINATION.

CHEST-WALLS, Deformities of.—See CHEST, DEFORMITIES OF.

CHEST-WALLS, Morbid Conditions of.—

The walls of the chest may be the seat of various morbid conditions, and the affections of this portion of the framework of the body demand more attention than they are accustomed to receive. All that can be done within the limits of this article is to indicate their nature; to notice briefly such of them as are not described in other parts of this volume; and to point out the principles of treatment. They may be practically considered according to the following arrangement:—

1. **Superficial Affections.**—Under this group are included morbid conditions of the skin and subcutaneous structures. (a) Cutaneous eruptions are of common occurrence over the chest. Among these may be specially mentioned the eruptions of some of the exanthemata, herpes zoster, and chloasma. (b) The superficial vessels are liable to become enlarged under certain circumstances. This enlargement is usually seen in the veins over the front of the thorax, which may be distended on both sides, or only on one side, or in some particular region. The larger divisions may alone be involved; or a more or less extensive network of smaller veins is visible, and occasionally even the capillaries seem to be implicated. This condition generally arises from some obstruction interfering with the circulation through one or other of the principal veins which, either directly or indirectly, receive the blood from the veins of the thoracic wall. Thus the superior vena cava, or either innominate, subclavian, or axillary veins may be implicated, being, for example, pressed upon by new-growths or enlarged glands, the distribution and extent of the venous distension varying accordingly. Occasionally one of the smaller veins is thus interfered with. The writer has met with cases in which considerable enlargement of the veins was visible over portions of the thorax, where the cause was by no means evident, the patients asserting that this condition had existed ever since they could remember, and being regarded by them as perfectly normal. Probably it has resulted from some local obstruction occurring during early life. It must be remembered that women who are suckling frequently present considerable enlargement of the superficial veins over the front of the chest, which usually subsides when the period of lactation is at an end, but may become permanent after several children

have been nursed. Again, more or less venous and capillary engorgement in this region may occasionally be observed in cases of cardiac or pulmonary disease. Sometimes a ring of enlarged veins and capillaries is seen round the lower part of the chest. When the venous distension is due to obstruction of the superior vena cava, the skin presents a more or less marked cyanotic tint; and in cases of general cyanosis, the chest, in common with other parts, may have a cyanotic appearance. Occasionally one or other of the small arteries which supply the thoracic walls is enlarged, and it may attain a considerable size. (c) Subcutaneous œdema is sometimes observed over the chest. In most instances this is a local condition, being the result of venous obstruction; but it may be a part of general dropsy, particularly in connection with renal disease. This morbid state is evident on inspection and palpation, and the affected part pits on pressure. (d) The thorax is one of the most common seats of subcutaneous emphysema, in which gas, consisting usually of air which has escaped from the respiratory apparatus, collects in the cellular tissue, and travels, to a greater or less extent, over the body. The causes of this condition are described elsewhere (see EMPHYSEMA, SUBCUTANEOUS). Subcutaneous emphysema is attended with evident swelling of the part, which usually develops rapidly, and may be very great, all the normal anatomical outlines being obliterated. It has an elastic feel, and pits readily on pressure with the finger, but speedily rises again; the peculiar minutely crepitant sensation accompanying this condition is elicited on palpation and percussion; percussion yields a curious, superficial, muffled, tympanitic sound; and on auscultation a superficial small, dry, crepitant sound is heard. (e) As belonging to the superficial affections of the chest may be just mentioned diseases of the mammary gland or nipple, which of course constitute a most important class of diseases in females. See BREAST; and NIPPLE.

2. **Muscular and Tendinous Affections.**—

(a) The muscles of the chest or their tendinous attachments may be the seat of certain painful affections. These are of the nature of so-called muscular rheumatism or myalgia, of inflammation, or of more or less injury or strain. They are induced by cold, constitutional conditions such as gout, overwork, straining, violent coughing, fatigue from prolonged sitting, and other causes. The painful condition is usually localised, but different muscles are involved in different cases, sometimes those which are superficial being affected, in others the deeper muscles, including the intercostal; or the complaint may be confined to a single muscle. Pleurodynia, dorsodynina, and scapulodynina are the terms applied to muscular rheumatism affecting the side of the chest, the upper part of the back, and the scapular regions respectively. As the result of severe coughing, muscular pains are very common around the lower part of the chest. The pain is usually more or less aching in character, and not severe, but it may be very intense, especially in acute cases. Whatever brings the affected muscles into play aggravates the suffering, such as moving the arms or shoulders when the superficial muscles are affected, coughing, sneezing, and similar actions. In some instances the pain is not felt when the affected structures are kept quite at rest. There may be local tenderness on pressure, or

diffused pressure may give relief; while posture often influences the sensations experienced, such as whether the patient assumes the recumbent or sitting posture, or lies on one or other side. Fatigue generally increases the pain. Muscular affections connected with the chest are not necessarily accompanied with any other local symptoms; and physical examination reveals nothing, except that perhaps the act of breathing is voluntarily restrained, on account of the pain thus induced. (b) The muscles of the chest may be affected, on the one hand, with spasm or cramp; on the other, with paralysis. The former is attended with more or less pain, usually localised, which may be very severe; the latter is indicated by loss of power in the muscles involved. These disorders generally depend on some lesion of the central nervous system, but may result from local nerve-disease. Muscular cramp may arise from a sudden twist of the body, and it is usually supposed to be the cause of the 'stitch' in the side brought on by running, but this is doubtful. In cases of hemiplegia from cerebral mischief, the muscles of the thorax on the affected side are often temporarily weakened, but they usually subsequently soon regain their power. When the upper part of the spinal cord is injured or diseased, all the muscles of the chest become paralysed. (c) Atrophy or degeneration may involve more or less of the thoracic muscles. In cases of pulmonary phthisis either the whole of these muscles or certain of them are not uncommonly wasted out of proportion to the general emaciation. They may also be implicated in progressive muscular atrophy; and occasionally a single muscle undergoes marked wasting. The writer has seen a striking example of this local atrophy in connection with the pectoralis major, but the serratus magnus or other muscles may be implicated. The wasting is probably in most cases due to disease of the nerves supplying the affected muscles. It is quite evident on examination, and the movements which are usually performed by the involved structures cannot be executed properly. (d) On the other hand, the chest-muscles may become hypertrophied. This may be a natural result of athletic exercises and training; or it may occur in consequence of their being called upon, either habitually or at intervals, to act excessively, as in cases of emphysema or asthma. In exceptional instances the condition known as pseudo-hypertrophic muscular paralysis has extended up to the chest. (e) The thoracic muscles not uncommonly present marked irritability under percussion or friction. This has been regarded as an important sign of phthisis; but the writer has often found it equally if not more evident in cases where the lungs were perfectly healthy. (f) As the result of injury and other causes, some portion of the muscular structures of the chest may be lacerated, ruptured, or perforated, either alone or along with other structures. This condition will be further alluded to presently.

3. Nervous Affections.—(a) Neuralgia is very common in different parts of the chest, especially in the side, and particularly the left side—*intercostal neuralgia*. The pain is localised, being usually referred to a point where a branch of nerve becomes superficial. It is more or less constant, in some cases being at times very severe. It often precedes or follows an eruption of herpes. It may be increased by deep breathing or coughing, but is not as a rule so much affected by these and similar

actions as are other painful chest-affections. Shooting and darting sensations often radiate from the principal point, and certain spots of tenderness—*points douloureux*—may be recognised (see *INTERCOSTAL NEURALGIA*). (b) Intercostal neuritis is occasionally met with, and this affection is attended with great pain, localised and radiating, with much tenderness, the suffering being aggravated by whatever causes any local disturbance. It is probable that the severe pains experienced in some instances where the complaint is supposed to be muscular, are due to branches of nerves being inflamed. Severe pain often precedes or follows herpes zoster, and this probably depends upon actual nerve-changes. It may also be mentioned here that pains around the sides may be associated with disease of the spine or of the spinal cord. (c) Some individuals, especially females, exhibit a remarkable superficial tenderness or hyperæsthesia over the thorax or in parts of this region, especially the anterior and upper portions. The slightest touch is resented, and the most delicate percussion cannot be borne. This condition may exist without any actual disease, or it is sometimes observed in phthisical cases. (d) Sensation may be more or less impaired over the chest, or in limited portions, either in connection with central or local nervous disease, or in hysterical and neurotic persons. Various paræsthesiæ are also frequently referred to this region by the class of individuals just mentioned.

4. Diseases of the Bones or Cartilages.—

The morbid conditions which may be referred to the bony and cartilaginous framework of the thorax are as follows: (a) The chest-walls are often unduly rigid and firm, owing to an excessive deposit of calcareous matter in the sternum and ribs, with ossification or petrification of the cartilages. This is a normal condition in old people, being one of the degenerative changes to which they are liable, but it also occurs not uncommonly in younger individuals, as the result of hard work, or excessive athletic exercises, or in connection with certain pulmonary diseases. This state of rigidity interferes more or less with the respiratory movements, and not infrequently causes serious embarrassment. (b) On the other hand, the ribs and cartilages may be deficient in firmness, and consequently too yielding and elastic (see *CHEST, Deformities of*). (c) Acute or chronic periostitis or perichondritis is sometimes observed in connection, respectively, with the sternum or ribs, or with the cartilages. The acute affection gives rise to limited pain and tenderness, which may be accompanied with superficial redness and swelling, and may simulate some more serious disease. The chronic complaint usually assumes the form of a node, being the result of syphilis. The writer has occasionally observed a small swelling at the junction of one of the ribs with its cartilage, painless, unaccompanied with redness, but with fluctuation. (d) The bony and cartilaginous structures themselves may be the seat of disease in some part of the chest, and here must be included the portion of the spinal column which limits this region posteriorly. Thus there may be acute inflammation, caries, or necrosis. Among the more important causes which may originate these conditions are injury, syphilis, tuberculosis, empyema opening externally, and thoracic tumours or aneurysms growing outwards. They may lead to serious consequences, both local and general, and frequently cause

more or less deformity of the chest. (e) Permanent thickening and distortion of portions of the ribs occur after fractures which have united improperly.

5. Inflammation and Abscess.—It is expedient to make a separate group of those cases in which inflammation, resulting in the formation of one or more abscesses, occurs in some portion of the soft structures entering into the construction of the chest-walls. This may be of local origin, arising from injury, bone-disease, or other causes; or it may be due to the opening of an empyema into the tissues; to suppuration extending and burrowing from the axilla or other parts; or to pyæmia. If deep-seated, an abscess may be difficult to detect with certainty, but usually the signs of this condition become sufficiently obvious. Sinuses or fistule may be left as a consequence of suppuration in the chest-wall, especially when pus makes its way outwards from within.

6. Tumours and New-growths.—These morbid conditions also demand brief notice. They may be connected with any of the structures of the chest-walls, and are of various kinds; among those which have come under the writer's notice may be mentioned fibromata, fatty growths, cystic tumours, enlarged sebaceous glands, and infiltrated carcinoma. Tumours may make their way inwards from the chest-walls, encroaching upon the cavity of the thorax; or, on the other hand, the walls may be involved by growths extending from within. It need scarcely be mentioned that mammary tumours constitute a distinct and by far the most important group associated with the structures covering the chest. See BREAST, Diseases of.

7. Perforations and Ruptures.—The muscular structures of the thoracic wall may be more or less destroyed in some part, either by sudden rupture or gradually, allowing a hernial protrusion of the lung to take place between the ribs. As already noticed, empyema may make its way externally through the chest-wall. Aneurysms and tumours extending outwards from within the thorax frequently cause serious destruction of the tissues, including the bony and cartilaginous as well as the soft structures. This destructive process is often attended with severe pain and suffering, and leads to grave mischief. Congenital clefts or more extensive deficiencies are sometimes met with in the sternum or other parts of the thoracic walls.

8. Variations in Form and Size.—The chest often presents deviations from the normal shape and size, and these are so important that they demand special consideration. See CHEST, Deformities of.

TREATMENT.—In many cases where the chest-walls are in a morbid state, either they do not need any special treatment, or no treatment can be of any avail. The chief circumstances under which the practitioner may be called upon to interfere, and the measures to be adopted, may be briefly indicated as follows:—

(a) Painful affections of the chest-walls, depending upon conditions of the muscles or nerves, frequently demand local applications for their relief. Thus in different cases it may be requisite to employ hot fomentations, dry heat, or cold applications, in the form of wet rags frequently changed, ice, or evaporating lotions; or to use anodyne applications of various kinds, such as belladonna plaster or liniment, opium plaster or liniment, oleate of morphine, tincture or liniment of aconite,

or ointment of aconitine or of veratrine. Anodynes may also sometimes be added to fomentations with advantage. Friction is often of much service, and at the same time stimulating liniments may be employed, such as one containing camphor, chloroform, or turpentine. For ill-defined muscular pains about the chest, which are frequently complained of, free douching with cold water every morning, followed by friction with a rough towel, is often highly efficacious. When pain is localised and obstinate, much benefit may in many cases be derived from the application of a sinapism, mustard-leaf, or even a small blister. In other instances the use of the ether-spray is serviceable, repeated more or less frequently; or sometimes much relief may be obtained from applying over a painful spot a mixture of equal parts of chloroform and belladonna liniment. Galvanism is another agent which may be of the greatest service in relieving painful sensations about the chest, whether connected with the muscles or nerves. Subcutaneous injections of hot water, morphine, or atropine may be demanded in some cases, and constitute a most valuable mode of treatment if pain cannot otherwise be assuaged.

In the treatment of many painful affections of the chest-walls much assistance may be derived from attention to posture, especially in connection with certain occupations; from the avoidance of undue fatigue, or of any violent actions which are known to influence this part, such as cough; and from the adoption of measures tending to support the structures, or to keep them in a state of rest. The writer has found great benefit in a large number of instances from strapping the side more or less extensively, in the manner advocated by him for the treatment of pleurisy; and if there is any localised pain, some limited anodyne application, such as a piece of belladonna plaster, may be placed over this spot under the strapping.

(b) In many affections of the chest-walls, treatment directed to the general system, or to some special constitutional condition, is often of the greatest service. Thus, in the painful complaints already noticed, there are frequently marked general debility and anæmia; and essential benefit is derived from the administration of quinine, preparations of iron, strychnine, cod-liver oil, preparations of phosphorus, and other tonics. Some of these are also useful when there is disease of bone and its consequences. Again, certain affections of the thoracic wall may be associated with rheumatism, gout, or syphilis, and then the particular treatment indicated for these several diseases is called for.

(c) When disorders of the muscles of the chest-walls occur, such as paralysis or spasm, associated with some disease of the central nervous system, the treatment must usually be directed to this disease, and but little can be done for the local disturbance. In some instances, however, electrical or other modes of treatment may be of some service, by influencing the action of the muscles, but no definite rules can be laid down.

(d) Local inflammation and its results in connection with the thoracic walls must be treated as in other parts of the body, but it is unnecessary to discuss this subject in the present article.

(e) Surgical treatment may be called for under certain circumstances. Of course this will be the case if the chest-walls are injured in any way. Among other conditions likely to demand surgical interference may be specially mentioned sub-

cutaneous emphysema, abscesses, disease of the bones, and tumours.

FREDERICK T. ROBERTS.

CHEYNE-STOKES RESPIRATION.—A peculiar disturbance of breathing, first described by the late Drs. Cheyne and Stokes, of Dublin. See *RESPIRATION, Disorders of*.

CHICKEN-POX.—**SYNON.** : *Varicella* ; Fr. *La Varicelle* ; Ger. *Wasserpocken*.

DEFINITION.—An acute specific infectious disease, characterised by the appearance, in successive crops, of red spots, which in the course of about a week pass through the stages of pimple, vesicle, and scab.

ÆTIOLOGY.—It is certain that this disorder arises from contagion, but the specific organism has not yet been isolated. It may be met with at almost any age, but is most common in young children.

SYMPTOMS.—The illness commences without any, or with but slightly marked, premonitory symptoms. There is usually some headache or feeling of lassitude. Within a few hours an eruption appears, generally on some part of the back or chest, but there are many exceptions to this rule. It may commence on the face, neck, chest, abdomen, or extremities, or upon several of these parts at the same time ; the scalp is almost invariably invaded. The eruption consists of small, faintly papular rose-spots, varying in number from a few to a hundred or more. These, in the course of eight, twelve, or, at the most, twenty-four hours from their appearance, change into vesicles, which, at first small in size and clear as to their contents, become quickly large ; globular, or semi-ovoid, in form ; translucent, glistening, and opalescent in appearance ; and surrounded with a faint areola. Towards the end of the second day the vesicles attain complete development, and about this time a few may be seen on the sides of the tongue, on the mucous membrane of the lips, cheek, or palate, and sometimes upon that of the genitals. About the third day a few of the vesicles may have a pustular appearance, and sometimes a few pustules are seen ; but regarding the eruption as a whole, pustulation forms an incident rather than an essential feature in its progress. On the fourth day the vesicles begin to dry up, and by the sixth complete scabs are formed. These fall off in a few days, leaving in their place faintly red spots, and sometimes a few pits. A single crop of eruption may be said to complete itself in five or six days ; and, as two or three crops appear on as many successive days, the illness will last rather more than a week. In the event, however, of there being a succession of crops, it may be prolonged for another week, but this is unusual. With the appearance of the eruption, the temperature may rise two, three, or even more degrees, and this may recur with each successive crop of spots, but in many cases there is no rise of temperature at all. The pulse is sometimes slightly increased in frequency ; the tongue is moist, and sometimes covered with a slight fur. As a rule, however, there is but little constitutional disturbance, although it is occasionally severe.

PATHOLOGY.—Chicken-pox is due to the reception of a specific poison, which, after an incubation of not less than thirteen or more than twenty days, shows itself by an eruption upon the skin. It affects the same individual once only. The infection

may be conveyed as soon as the eruption has appeared, if not before, and a patient may be regarded as free from infection, and therefore fit to go home or rejoin school, when every scab has fallen off, particular attention being paid to the scalp. Chicken-pox is not one of the diseases to which the *Infectious Diseases Act* (1889) applies.

COURSE AND TERMINATION.—Chicken-pox almost invariably runs a favourable course ; in tubercular children occasionally the vesicles instead of drying up in the usual way go on to the formation of ulcers and the child dies of exhaustion ; this form has been called *varicella gangranosa*. In rare instances grave lesions of the central nervous system have followed an attack of chicken-pox.

DIAGNOSIS.—In the earliest stage there may be a difficulty in distinguishing between chicken-pox and a mild attack of modified small-pox, but in the latter there should be the premonitory symptoms of illness, and especially the pain in the back. Moreover, the rash is at first ‘shotty,’ which it never is in chicken-pox. The vesicles in small-pox should always have a central depression ; in chicken-pox this is absent, the vesicles rapidly becoming scabbed. The fever subsides when the eruption appears in small-pox, the reverse being the case in chicken-pox.

TREATMENT.—The patient should be confined to his room, perhaps in the more marked cases to his bed, and isolated from those who have not had the disorder. No special treatment is called for, and complete recovery is the almost invariable result.

JOHN ABERCROMBIE.

CHIGOE.—The chigoe or jigger is a minute parasitic insect belonging to the genus *Pulex* and known as the *Pulex penetrans* or sandflea. It is common in the West Indies, tropical America, and tropical Africa.

The chigoe resembles the ordinary flea, but the head is proportionately larger and the abdomen deeper. It lives in dry and sandy soil, especially when this is mixed with animal refuse. It attacks any warm-blooded animal. After impregnation the female buries herself beneath the skin, the two posterior segments of her abdomen plugging the aperture through which she had entered. The feet, especially between the toes and at the root of the nails, are favourite sites ; but any part of the body may be attacked, and any number of jiggers, from one to one hundred or more, may be present in a single patient. The abdomen of the parasite swells during gestation to the size of a pea, the enlargement being accompanied by suppuration and necrosis of the skin round the aperture, leading to the formation of a small ulcer from which the parasite is finally expelled. The ulcers may become infected with the organisms of tetanus, phagedæna, or other diseases, and serious and even fatal results ensue. In the meantime the ova, being expelled, fall on the ground and pass, in the usual way, through the larval and cocoon-stages until the fully formed parasite is finally developed in less than a fortnight after the expulsion of the ova.

TREATMENT.—This should be in the first place prophylactic. Wherever the parasite is known to exist, the ground-floors of all houses and out-houses should be carefully cleansed. Shoes should be worn invariably. The frequent application of carbolic acid, and other antiseptic lotions, to the feet is said to offer some protection against the parasite. Any irritation in the foot should be at once attended

to, and any chigoe that can be found on, or in, the skin should be at once removed, in the latter case by enlarging the opening and extracting the parasite with a needle.

PATRICK MANSON.

CHILBLAIN. — SYNON. : *Erythema Pernio* ; *Pernio* ; Fr. *Engelure* ; Ger. *Frostbeule*.

DEFINITION.—A form of dermatitis induced by cold.

ÆTIOLOGY.—Chilblains are commonest in children and young persons. They are most prevalent in winter, and are more frequent in girls than boys, doubtless owing to the greater susceptibility of the vaso-motor system, but they indicate more than a defective circulation. They may be considered as an indication of debility and deficient vital power. Their occurrence is influenced more by the strength of the individual than by the degree of cold, and they continue in some persons throughout the entire year. Their tendency is to cease with the full development of the system : they occasionally reappear in advanced life.

DESCRIPTION.—The regions of the body usually affected with chilblains are the toes, outer margin of the little toe, edge and dorsum of fingers, the chin, nose, ears, and heel (hence the term ‘pernio,’ from *πέρνα*, the heel). A chilblain presents three stages or degrees of severity, namely, *erythematous*, *vesicular*, and *ulcerated* ; and it may be arrested at the first or second stage by the withdrawal of the cause. The *erythematous* stage is restricted to dusky redness, swelling, and burning and intolerable itching, the itching being increased by heat, as by sitting in front of the fire, or by active exercise. The congested spot is circular in figure, somewhat tumid, brightly red at first, but later on roseate crimson, purple, or livid in colour. The ‘local asphyxial stage,’ which constitutes the mildest form of Raynaud’s disease, is a somewhat similar pathological condition (see RAYNAUD’S DISEASE). The second stage exhibits the blister resulting from effusion of serum beneath the cuticle ; the permanent colour of the swelling is now purple or livid ; and the contents of the blister a limpid serum. In the third stage the blister is broken, and a small ulcer results, which is often difficult to heal—this constitutes the ‘broken-chilblain.’

Cold first causes vaso-constriction of the arterioles followed by dilatation from vaso-motor paralysis ; and then follow the other inflammatory phases. See CIRCULATION, Disorders of.

Erythema pernio has to be diagnosed from an early stage of lupus erythematosus ; occasionally the two affections are co-existent. See LUPUS ERYTHEMATOSUS.

TREATMENT.—The principles of treatment of chilblains require to be modified to suit the different degrees. Prophylactic treatment is the best. This implies keeping the extremities warm, wearing thick solid loose-fitting boots, wash-leather socks, double-lined gloves, velvet bracelets ; daily systematic out-of-door active exercise (running, walking, cycling, skipping). In the first stage, the indication is to restore normal circulation by gentle friction : then some soothing liniment may be employed ; and, finally, a stimulating liniment, covering the part afterwards with ointment of menthol and lanolin and cotton-wool, or shielding it with lead or opium plaster spread on wash-leather. The applications most in favour for this purpose are the

soap liniment with chloroform and laudanum, the compound camphor liniment, the turpentine liniment, the tincture of iodine, and Goulard’s Extract. In the vesicular stage a similar treatment may be used to the erythematous portions, while the blister should be snipped and the broken surface pencilled with the compound tincture of benzoin, and afterwards dressed with unguentum resinae or an ointment of Peruvian balsam. In the third stage the erythematous phenomena still require attention, and the ulcer should be dressed with unguentum resinae, either alone, or in combination with antiseptics.

To obviate constitutional debility, the diet should be nutritious and generous, and recourse may be had to nerve tonic remedies, such as liquor arsenicalis and the best hygienic surroundings. See FROSTBITES ; COLD, Effects of Severe and Extreme ; RUBEFACIENTS.

JOHN HAROLD.

CHILDREN, Backward. — See CHILDREN, Training of.

CHILDREN, Clinical Examination of. — See INFANTS, Diseases of.

CHILDREN, Training of. — Training may be begun before the use of words, and a proper understanding of them, are acquired.

The general characters of brain-action observed in an infant or young child—regard being had to its age—indicate its potential mental capacities. These signs vary at different stages of evolution. The final result much depends upon continuous growth and healthiness ; hence these characters should be watched and duly cultivated. Spontaneity of action in the nerve-centres is indicated in childhood by general mobility ; this forms the basis of future mental power. While the infant is awake spontaneous movements are seen in the limbs, especially in the small parts ; the fingers and toes move individually or collectively, but without uniformity and in no apparent order. These movements are slower than those of adults ; they are almost constant except in sleep, and are but little under the control of sensory impressions. This spontaneous movement of infancy the writer has described under the term *Microkinesis*. Gradually in the advancing months of early life the centres begin to act in combinations, co-ordinated to some extent by sight and sounds. At about the fourth or fifth month the sight of an object may temporarily inhibit the movements, and this may be followed by turning of the head, eyes, and hands towards the object seen, i.e. the co-ordinated action is consequent on a period of inhibition following stimulation. From such observations we infer that at birth the nerve-centres act slowly and independently of one another ; that, as evolution advances, their action may be temporarily suspended by sensory stimulation ; and that, during the time when no efficient nerve-currents are passing to produce visible movements, the centres undergo a physiological change—possibly due to action in their dendrons—subsequently indicated by new and co-ordinated movements. Such observations show the existence of an initial faculty of ‘mental attention.’ The imbecile infant does not exhibit this microkinesis in a normal degree ; its nerve-centres are wanting in spontaneity, and, later on, in the capacity for co-ordination.

The object of early training is to bring the spontaneous activity of the nerve-centres under control, so that they may subsequently act in harmony with the child's environment, and that the evolution of the brain may be guided in such a way as to fit it for the work of mental life. In the Infant-class, bright faces, ever moving hands and fingers, with readiness for chatter or response, show spontaneity of the brain-centres, free action, ready for the methodical training which may co-ordinate the brain but not suppress its activity.

The principal means of controlling the brain and producing impressions upon it are through the eye by sight and the ear by hearing; but besides these, stimuli may reach the brain through the other senses and from the muscles and other organs of the body.

Muscular sense as a means of training the brain has been too much neglected in educational methods. It is easily employed before the use of words is possible; while, further, the response to impressions thus produced can be *seen* by the teacher, and then the improvement in accuracy produced by repetition is observable. Muscular sense may produce effects in the brain in two ways, which, as they differ in methods of employment and their uses in training, may be classed separately, as—(1) Impressions by muscle-sense during contraction; (2) impressions produced by the muscles in tension or by the strain of weights or pressure. As examples of the former variety we know that impressions are produced by movements of the eyes or the hand, as in looking at or pointing to objects in succession, and in counting them, or in following the movements of a pendulum; also in measuring the length and size of objects with the fingers. Illustrative of the latter group we have the impressions produced by muscle-tension, as when feeling a weight in the hand, the strain felt being proportional to the weight used. These two kinds of impressions by muscular sense are here separated because in training they are employed differently, and lead to different modes of mental comparison and description; the size of objects may be compared, but must not be confused with their number and their weight any more than the characters of hardness and of colour. Each kind of impression should be produced separately in elementary training. The size of a coin can be seen by eye-movements or be felt in the hand with the fingers closed around it while the limb is supported, so that the weight shall not be felt; thus weights and sizes can be compared respectively.

Early training should be designed to develop control rather than to impart information. Training must proceed stage by stage; each method used needs to be often repeated to produce an effect, and to ensure accuracy and retentiveness. Each point in training should be planned to bear on future work, and no more difficult brain-process attempted till the elements of the action which it involves have been acquired. One of the earliest requirements is to control the child's eyes: this applies equally to the very young child, to the neglected and untrained pupil, and to those adults who are childish and dull in make of brain.

Let the teacher try to get the child to look at her face, or at a coin or bright object held in the hand and moved in different directions, following it as it moves. At first the child will do this by moving his head only without moving the eyes in his head. *Eye-drill* is very useful. Let the child look at

objects in the room, at a ball as it is thrown, or at a light flashed from a candle. At first, sight stimulates eye-movements; later he moves his eyes, directed by sound as he is told what to look at. Sight impresses the brain, so do eye-movements, and these produce impressions by muscular sense which are subsequently useful when the child is learning the meaning of numbers, as in counting, when the eyes are turned to one object after another.

In speaking of *hand-movements* reference will be made to training intended to educate the brain, and thus to lead to mental faculty, rather than to manipulative processes, games, or gymnastic exercises, all of which are none the less useful. Let the child imitate the action of his teacher, holding out one hand straight and free, on a level with the shoulder, the elbow straight and the fingers all in the same plane. Instinctively the child's left hand imitates the teacher's right hand. When slow movements are made in bending the fingers one or two at a time, these are imitated at first very inaccurately, but by frequent practice imitation becomes more exact according to the degree and the time of each finger-movement. The good training which is thus afforded exercises control; as each centre in the teacher's brain moves a finger, the corresponding centre in the child is brought into activity in similar degree and relations of time of action; further, the effect produced can be observed. Such exercises soon fatigue a young brain, and should not be continued for more than a minute at a time. It will be found that in succeeding lessons such series of movements become more regular, more exactly controlled by sight, and that a slighter stimulus produces the desired effect till the exercise is retained or remembered and can easily be reproduced without further guidance. We infer that during practice the brain-centres are being gradually prepared for action in harmony with one another, so that they become more capable of regulated or co-ordinated action. At first the nerve-centres are merely brought into action in certain groups; after repetition they seem to grow together, so as to tend to act in one union of cells. It may be that they become functionally connected through their dendrons. These impressions made on the cells are strengthened by repeated training, as an organisation is built up. Such previous training proves useful later on in the process of teaching the significance of numbers; the child feels that four movements of his arm are greater than two. Imitation is important in speech-teaching, which is needed by all children. *See DUMBNESS.*

Muscular sense in tension is also to be exercised. Let the child hold out his hands in front, supinated, with the fingers open; then place an ounce weight on the fingers so that the strain is felt as apart from dimensions or bulk. When subsequently it is required to distinguish weight from size, or to make comparisons as to size and weight respectively, the previous separate impressions received will be useful in giving an understanding of the subject taught. Let the child feel the strain of the weight on his hand, then feel the size with his fingers closed, while his arm is supported or rests on the table so that he does not feel the weight. For instance, in teaching the coins it is best to produce each class of impression separately: the feeling of weight and size respectively by muscular sense, then the colour by sight; let the child learn to pronounce the name of the coin, then look at it, and

finally name it at sight ; the previous training is used in describing what is seen. Accurate impressions received one at a time enable comparisons to be made, but the comparison must be of impressions of like kind : weight may be compared with weight, size or dimensions with those of like character, and colour may be matched at sight. In comparison and description impressions have to be classed ; therefore each kind should be implanted separately in early training. Another point is that one class of impression at a time is more easily received and retained in the brain and is less confusing and fatiguing. Speak to the child ; show him a colour, an object, or make him feel either its size or weight, but do not produce each kind of impression at once in the earliest methods of training. Proceeding thus stage by stage, the child is prepared for more advanced educational methods. Eye-movements well under control lead to accurate seeing and reading ; they are employed in counting objects and in adding figures on paper : the student who does not fix his eyes well becomes an inaccurate observer. Trained muscular sense leads to a ready appreciation of weight and dimensions, their comparison, proportions, and description ; impressions made separately upon the brain are retained, and become associated with one another and connected with ideas and the use of words in education.

Standards of weight, measurement, and time, as well as the use of numbers, must be taught. For this purpose impressions are produced one at a time by carefully placing in the open hand in turn weights of one, two, and three ounces—first produce the impression, then name the weight. A knowledge of length may be taught by making the child move his eyes in looking at horizontal and vertical lines on the blackboard, which should be vertical against the wall, the lines being divided into inches, but care must be taken that the eyes are moved in looking. Movements made with the finger one inch at a time also produce impressions of length. Impressions of the duration of time may be implanted by counting the movements of a seconds-pendulum, or by noting how long it takes to walk a hundred yards, or in other ways, giving ideas that can afterwards be used.

Discrimination or the appreciation of agreement and difference in objects may be cultivated by training, planned to produce at first a few impressions of each kind. A very young child can distinguish between the objects and colours presented, and will respond readily to certain sounds ; sorting out objects of like size, weight, or colour is an elementary preparation for making comparison, and precedes the faculty of judgment. Retention and discrimination of sensory impressions under guidance lead to experience, which forms the basis of intelligent choice. In training stage by stage, the pupil can be made to *feel* the greater strain of four ounces in his hand after the two-ounce weight ; he may then be taught that this is the 'greater weight,' 'twice the weight.' He *feels* the difference and then learns how to express this ; similarly he can be made to feel and then express which is the longer stick. The child must learn how to look at and examine objects, and be taught the means of comparing and describing what he sees and feels. All this requires much time and practice in observing things which are alike and unlike, noting in what characters the agreement or difference consists.

The teacher must carefully consider some one point for comparison—it may be weight, length, size, or colour ; each of these characters should have been taught previously by many examples.

In seeking a scientific basis for the art of training we must not be satisfied with a connected train of ideas in our own minds which may be implanted in the child's brain by the use of words only ; we should endeavour to trace out the impressions produced in good teaching which become associated in later teaching and experience. With pupils a little advanced in their mental training it may be useful to let them examine an object for themselves, say a bean or a flower ; they thus acquire many different kinds of impressions at the same time as they see, feel, and handle the object. They should gain impressions as to the number of parts, their form, size, colour, weight, &c. When however the child proceeds to make comparisons and give a description, he must separate and classify the different impressions received ; in this process guidance will be necessary till trained mental habits have been acquired. When sufficiently advanced in education, the pupil can be trained to observe and note the time of events, with the order in succession of their occurrence, as preparatory to studying Natural History and the Physical Sciences. Impressions produced by observation of facts in the order in which they were seen must be retained in the brain. Further training and practice will be needed to educate the child to see and understand that when peas are soaked in water they become increased in size and in weight, while a proportionate weight of water disappears from the vessel containing them. If the peas are placed on damp moss for a few days they will be seen to sprout, the root growing downwards, while the stem grows more slowly upwards. When light reaches the plant the young leaves become green, but not till *after* the light comes to them ; if grown in darkness they are not green. If the plant is kept in a room, as the stem grows longer it is bent towards the light. To follow the teaching given, the pupils must be previously practised in estimating length by sight, also size and volume, and must also be conversant with the comparison of lengths, sizes, weights, and the colours respectively, so as not to become confused in mental action and try to compare size and weight which may not be proportional. It requires much previous training to enable the child to see that the concave side of the stem of the plant is shorter than that which receives less light. Familiarity with the methods of noting and remembering the order of events will show that the presence of water precedes the swelling of the peas ; that the unequal illumination of the two sides of the stem precedes unequal bilateral growth, and that bending in the direction of the light results in consequence. In such methods of educational training we teach observation of antecedent and sequent events in place of speaking of cause and effect, or allowing verbal explanations expressed in terms *because, therefore*. The order of associated impressions and ideas received is important, and tends to be retained in the brain, while it is strengthened by repetition ; co-ordinate physical exercises help to cultivate this mental faculty. Associated impressions and their attendant thoughts when firmly retained revive in the same connection. The cultivation of character and voluntary power may be partly effected by the training here briefly

sketched, as well as by implanting thoughts and principles through the use of words.

Retention of the order in which the pupil has made his observations has much to do with his logical thinking and understanding antecedents and sequents or cause and effect; the order of the numerals together with the degree of the weights and lengths compared respectively, and expressed by use of the numerals, is necessary in mental estimation of the weight, bulk, mass of objects, and also to appreciation of the probable value of the quantities of material seen, as in the practice of 'school shop.'

Dull and backward children and those organically 'feeble-minded,' as well as all young children, need carefully planned training to help in evolving their mental faculties. Besides cultivation of the natural characters of brain-action, any phenomena indicating faults and defective conditions of the nerve-centres should be observed, and when possible replaced by better forms of activity. These faults are expressed in movement and postures seen in the body; the observation of these detailed neuromuscular signs and balance of the limbs and face will afford definite guidance as to the method and degree of training adapted to the individual child. Observation of the pupil should always accompany and direct training.

The *face* may be taken as an index of the brain. The expression of a child's face should be lively, changeful, mobile, showing spontaneity and response: not fixed, defective, or absent. Stimulus and proper occupation will often improve expression and also remove for the time some of the uniformly repeated nerve-signs to be described. The *frontal muscles* which produce horizontal creases on the forehead may be seen to move spasmodically and irregularly; these muscles are generally quiet when the child is interested, and may thus afford a guide to this condition of the mind. *Corrugation* or 'knitting' of the eyebrows is often associated with some form of mental stress, such as fright or hard mental effort. Observation of the conditions under which it subsides or increases may supply useful indications for management. *Frowning* is frequently associated with the presence of headache, and should suggest the possible existence of ocular defects (myopia, &c.). Spasmodic movements of the facial muscles may develop into habits which are often difficult to eradicate. *Fulness under the eyes*, due to relaxation in tone of the orbiculares oculorum, is an early sign of fatigue and common accompaniment of headache; if the child smiles or laughs it disappears for the moment. The fatigue may be temporary, as from mental work, insufficient feeding, a close atmosphere, or late hours. *Grimacing*, or over-smiling, with deepening of the nasolabial lines, is seen with low-class brains as a uniformly repeated action, spontaneous or occurring on every stimulation. This may improve and subside under general training and occupation; it is worst in deficient children who are allowed to be idle and loafing. The habitually *open mouth* suggests obstruction in the nose and throat: adenoids and hypertrophic rhinitis are very common in cases of mental deficiency.

It has been shown that the brain may be trained by the use of exercises for the hand, and receives impressions from its movements: the hand may be observed as an index of brain-status, the faults of which may thus be seen and corrected. A healthy well-trained child when told to hold out his hands

in front responds by raising each arm to a level with the shoulder, horizontal and straight at the elbow, the arms being parallel to one another. *The hands* should balance straight at the wrist and knuckles, with the fingers and thumbs in the same plane; this is the normal attitude with good brain-activity in preparation for action under guidance. Assuming this position momentarily helps to put the brain under control, just as removing all slouching habits improves the child. With inertness or fatigue the wrist droops, the metacarpal bones become adducted or folded, while the thumb falls and the fingers are partially flexed; the elbows are apt to be somewhat bent, and the left hand held at a lower level than the right. In the nervous *neurotic child* the hand tends to balance in a different position: the wrist droops, the palm is contracted and looks narrow, the thumb is bent back, and each finger extended beyond the straight line at the knuckle-joint, thus producing a claw-like appearance which is frequently seen in children convalescent from chorea, in bad sleepers, and in those who suffer much from headaches. These children may also present twitching of the fingers, either vertically or laterally. Bad postures are frequently imitated in school from other pupils; asymmetrical balance of the head and spine, as well as lordosis, should therefore be avoided, while good postures both in sitting and standing are taught.

Response of the brain is seen in action following some stimulus or direction given to the child; when this action is in harmony with the direction the response is intelligent. In response dependent upon mental action in the brain, a period of inhibition of spontaneity—partial or complete—occurs, corresponding to interaction among the centres in attention or thinking. A mere act of imitation helps in training, but the repetition of a mode of expression associated with the direction received is not a very intelligent action, though it is as much as may be expected from a very young pupil. It is little more than a reflex brain-action without much interaction among the nerve-centres, calling into play the results of experience and education: such is mechanical or parrot-like memory. For this reason a prompt response is sometimes of less value than a little delay in reply, if this indicates thoughtfulness. Accuracy in action, readily corresponding to the direction given, is of greater value than correct verbal expression or promptness and quickness: response in correct language is not to be looked for till a full vocabulary and the accurate use of terms have been gained by teaching. In mental arithmetic, when the pupil is told to add the numbers mentioned, and gives the correct answer, it does not much matter if his hands twitch and his eyes move about, while the feet shuffle, owing to some spreading or 'overflow' brain-action. When a sum in addition is worked out on paper, sight and eye-movements direct the brain-processes, while the fingers express the result; here irregular eye-movements and twitching fingers may lead to incorrect addition and mistakes in writing.

Besides receiving aid in the evolution of the general characters of brain-action and in the correction of faults, the child must also be trained in good mental habits. Efficient and well-arranged teaching can do much to render the brain healthy and strong for work during adolescence, and may make it less liable to neurotic disturbances and to the spreading or extra action exhibited in excessive

emotion or in mental confusion, which are as wasteful of energy as attacks of passion, epilepsy, or hysteria.

Higher Education.—In training the power of observation and in teaching methods of procedure, it is all-important that the pupil should know what *to look at* and what *to look for*; but beyond this, the processes of observation must be implanted in methodical order. This is analogous to making the pupil learn the order of the cases and genders according to which a Latin adjective is declined, before he learns the terminations and forms of each respectively; such fixed methods of thinking save the pupil's time and much unnecessary mental labour. Each mode of brain-action, necessary to the kind of work undertaken, should have been previously trained by practice: thus, before teaching the study of comparison and description of form, size, and area (geometry), the pupil requires training in the use of sight, eye-movements, and muscular sense, as well as in mental procedure according to the directions given and retained from practice. The boy may look at the face of his master who is teaching history or language, but he must learn to turn his eyes to the blackboard or map during a lesson in Euclid or geography. In Latin the prefix and termination of the words must be separately seen; in doing sums on paper the lines of figures must be followed in their order.

Mental fatigue should be avoided till the brain has grown, and its natural spontaneity has been in part co-ordinated by training; but at about seven or eight years old the brain of a healthy well-grown child has acquired an average weight of about 46 drs. (Dr. Thurman), and can begin to bear some temporary fatigue from mental effort without harm resulting. The fatigue of the day should be completely removed by sleep at night, and periods of mental effort must be followed by ample intercurrent activity in other directions, and by times for rest. Dr. Clement Duker recommends the following hours for work per week and sleep per night:—

	Ages	Work	Sleep
Children . . .	7-8	12	12½
„	10-11	25	11
„	15-17	45	9
„	17-19	50	8½

Weariness may be due to many causes: diet and ventilation may be defective, leading to the circulation of impoverished or impure blood through the brain; excessive muscular exercise may exhaust nerve-energy; late hours may lead to morning inertness; spontaneous self-contained thinking and imagining, with habits of mental introspection, sometimes lead to exhaustion in unoccupied and lonesome children.

The signs of fatigue should be known and appreciated by the teacher. In addition to those previously mentioned affecting the face, the fingers twitch, the shoulders are shrugged, the feet shuffle, and chattering or whispering begins; the head is held less erect, and the hands in their balance may show lessened neuro-muscular strength, as explained earlier. Action and response become slow and inaccurate, the mental status being reduced to a more childish condition. The question put may be repeated in place of a reply; the pupil is less controllable, while attention is not easily co-ordinated. Fatigue is not necessarily

proportional to the amount of school-work accomplished; it is important to observe the general expression of brain-action, not only its special results in the class-occupations. In the child the impressions retained are less exact than in the adult, while spontaneity is more abundant and may interfere with any established order of thinking; but spontaneous thoughts arising in the brain add to fatigue. At an early stage in education there exist but few established modes of thinking ready to come into play in forming a mental conclusion; effective training by implanting series of impressions, ready for methodical employment, does much to lessen the chances of mental fatigue.

Mental hygiene and careful general training promote good education, guided by observation; they aid brain-growth and the evolution of mental faculty; but to these must be added some experience of the conditions of life and social environment. This is specially necessary for children in boarding schools and institutions, who may otherwise experience less of the varied circumstances of life than those living at home and attending day schools. Besides abstract knowledge the child must also learn his position among the circumstances of life, and his social relations to other persons. Experience will teach the need of self-protection, of preparation for labour in earning a living, of honesty and good manners, as well as the value of learning. Hence children need comradeship as well as the influence of adults; school-habits and discipline lead to some understanding of cause and effect in social and in intellectual living. Teaching, if explanatory by words only, gives but little experience that can be of use to the child till actual impressions have been felt. If the boy has felt the pain of scalding water he may readily understand that hot things hurt, whether it be the coals and lamp which are alight, or the teapot which does not look hot; and he may understand further that it is dangerous to play with matches. Experience with animals is useful: children find out that dogs can bite, but that they are friendly if kindly treated. It is experience that couples the thought in an act of judgment leading to acceptance or refusal; a choice must be made between two offers, or a decision as to some line of action, and this necessity helps to form character.

The principles of hygiene should have some place among other subjects of instruction, whether this be formally taught or not; experience in feeling pleasure and activity amid plenty of light and air is educational. The effects of light and feeding or of starvation and partial darkness on plants may easily be shown in the school-room and in the practice of daily attending to them.

In dealing with an individual child, or when advising as to methods of class-training, the physician will consider the constitution of each child in relation to organised education; there are great differences, as well as inequalities, in the physical and mental abilities of children. Space will not allow a detailed account to be given of the examination of children for the purposes of educational classification, but the following propositions summarise the results of the writer's inquiries among 100,000 school-children:

I. The main classes of defect among children include a larger proportion of boys than girls.

II. The main classes of defect are much associated in the groups of cases.

III. Children with developmental defects often present also abnormal nerve-signs, and are delicate and dull.

IV. Children with indications of brain-disorderliness—that is, abnormal nerve-signs—are often dull pupils.

V. Dull pupils are often delicate, with indications of brain-disorderliness.

VI. Girls with developmental defect, or with brain-disorderliness, are more apt to receive harm and less good from their environment than boys.

VII. The effects of good physical training in school are to diminish the number of cases with signs of brain-disorderliness, and the number of dull children.

Weak or nervous children need careful training and management, which must be adapted to the child's needs, but not necessarily in all particulars according to his inclinations, for physical weakness predisposes to habits of indolence and loafing, which are best removed by the spirit and method of school-life, both in play and work, under the stimulus of emulation. This process of trying to level the boy up to the average of his companions calls for careful consideration and consultation in the school, when it is determined 'to make the boy work and play.'

Medical officers have in recent years been appointed by most of the School Boards in large cities to look after questions concerning illness among the children, and to take part in the selection of pupils who come under the provisions of the Education Act (Defective and Epileptic Children) 1899, and also to advise as to the methods of their training.

Feeble-minded children may be recognised and certified by observation of the *abnormal nerve-signs* already referred to, and by mental tests, as well as by *defects in developmental growth*, which will now be briefly described. The cranium may be bossed on either side of the frontal bone and at the parietal centres; the forehead may be narrow and shallow or present a vertical ridge. The volume of the cranial interior and its ossification are of great significance; the normal circumference at seven years is 20–21 inches. The palate is defective if narrow laterally, too much arched, or angular in front—i.e. V-shaped, with the upper incisors prominent; a small mouth-opening often co-exists with adenoids and nasal obstructions. The nasal bones may remain ill-developed, broad, and flat; this may improve with growth. The external ears are frequently defective in boys—large, outstanding, coarse in texture, the antihelix absent and the helix ill-formed, presenting a cave-like ear. The features are often defectively developed; the face as a whole may be small and infantile, or large, round, flat, with small features rising from its plane; the palpebral fissures small, with the transverse axis sloping upwards and downwards or upwards; a small mouth is more frequent than a large one. The lower jaw may be prognathous, large and heavy, or too small in proportion to the cranium. Defectively developed children, especially girls, are often stunted in growth, delicate, and ill-nourished. The presence of several developmental defects, with want of the general characters of good brain-action, combined with abnormal nerve-signs and absence of mental ability, indicates the probability of the child being feeble-minded.

The conditions of *sight* and *hearing* and of the mouth and throat, as well as the general health, need to be investigated; a careful physical examina-

tion of the child said to be dull is necessary before giving a diagnosis and prognosis; the facts thus observed will suggest treatment and the best methods of commencing brain-training.

FRANCIS WARNER.

CHILL.—A sensation of coldness accompanied by shivering, experienced during the rise of temperature associated with the onset of acute specific fevers or other inflammatory diseases; also occurring in nervous individuals, and in attacks of *megrim* without any rise of temperature.

In popular language a 'chill' is employed to mean exposure to cold or wet, followed by some acute disorder. Thus a cold draught playing upon the face may be followed by facial paralysis, sore throat, or bronchitis; that is to say, cold applied locally may excite disease in the neighbourhood of its application or in distant organs. It is probable that a 'chill' may act in several ways: (1) it may cool the blood circulating in the skin, especially when the vessels fail to contract rapidly under its influence; and the cooled blood may be supplied to internal organs; (2) it may affect the nerve-endings in the skin and by reflex action interfere with the circulation in the associated viscera (*see PAIN IN VISCERAL DISEASE*); in both these ways it may diminish the resisting power of the tissues to injury, and enable micro-organisms to gain a footing; (3) exposure to cold may check the secretions of the skin, the mucous membrane, or other parts.

Disease of the nose, throat, larynx, lungs, and kidneys is frequently traced to a 'chill.' Moreover diarrhoea, dyspepsia, acute and chronic rheumatism, and many other disorders are often traceable to no other cause. In some instances, as in acute pneumonia, it seems certain that the effect of chill is limited to the lessening of the resisting power, the immediate cause of the disease being the multiplication of bacteria in the body, and the dissemination of their toxins in the tissues.

The 'chill' so often 'taken' by those returning from densely crowded resorts late at night is probably the complex resultant of many forces, including the inspiration of organisms from the vitiated air; the lowered resisting-power depending on the effect of fatigue, and of over-heated air surcharged with carbonic acid and organic matter; and the effect of the sudden change of temperature on the nerves and dilated vessels of the skin, before referred to. Individuals differ in their susceptibility to 'chill'; and in any given individual the susceptibility of each organ is by no means identical. Moreover this susceptibility of special organs may vary with the age of the individual, the season of the year, and many other conditions.

TREATMENT.—The susceptibility to 'chill' may be diminished by the wearing of woollen under-clothing, and the careful regulation of individual habits, such as the temperature of the morning bath, the ventilation of sitting-rooms, and other details. The curative treatment of the maladies ascribed to chill is described under their respective headings.

H. MONTAGUE MURRAY.

CHIN-COUGH.—A synonym for whooping cough. *See WHOOPING COUGH.*

CHLOASMA (χλόα, a green herb).—SYNON. : Liverspot; Fr. *Ephélide*; Ger. *Leberfleck*.

An indefinite term often applied to any pigmentary

discolouration of the skin of a yellowish-brown or liver-colour tint, occurring in blotches. Its synonym, *ephelis gravidarum*, indicates its occasional association with pregnancy. It is sometimes used as a synonym of *linea versicolor* to denote the pigmentation caused by the *microsporon furfur*. See PIGMENTARY DISEASES OF THE SKIN.

CHLORAL HYDRATE, Poisoning by.—

Poisoning by chloral hydrate is a very common occurrence, this medicament being frequently taken in fatal quantity by misadventure. There is reason to think that it is also largely used for suicidal purposes.

ANATOMICAL CHARACTERS.—There may be an entire absence of any characteristic appearances after death by chloral hydrate; and at most these consist in more or less modified signs of asphyxia—especially a dark colour of the blood, and pulmonary and cerebral hyperæmia.

SYMPTOMS.—The most striking symptom of poisoning by chloral hydrate is the rapid supervention of quiet sleep, at first simulating natural sleep. In this stage the patient can be easily roused, but he speedily drops off again. The pupils are contracted; the respirations are full, deep, and regular; the pulse is not much affected. This condition rapidly deepens into full coma. The respirations slacken; and the pulse is either weak and slow, or, more commonly, rapid and irregular. The temperature of the body is reduced; the muscular system is totally relaxed. The pupils now dilate; and with feeble, thready pulse, the anæsthesia and paralysis gradually end in death, preceded by lividity and collapse. Exceptionally, in fatal cases, burning pain in the mouth, fauces, and throat, and symptoms of gastritis have been observed. In one case of recovery the patient became idiotic. The urine contains an abundance of glycuronic acid.

DIAGNOSIS.—The history of the case, or the finding of a vessel containing the medicine, coupled with the symptoms, will usually set all doubts at rest. Otherwise the case may be mistaken for poisoning by opium or other narcotic, for carbolic-acid poisoning, or for cerebral congestion. The pupil is not so contracted as in opium-poisoning; and, as the coma deepens, the pupils dilate instead of undergoing further contraction. There is an absence of the olive-green or black urine so commonly noticed in carbolic-acid poisoning, and of the peculiar odour of the breath, and of stains about the mouth and lips.

PROGNOSIS.—This will depend upon the state in which the patient is found, and upon the length of time which has elapsed since the ingestion of the poison.

TREATMENT.—Evacuation of the stomach by the aid of the stomach-tube is the first step in the treatment of a case of poisoning by chloral hydrate. Emetics, unless given early, usually fail to excite vomiting. The patient must be persistently roused, if possible, as in opium-poisoning (see OPIUM, Poisoning by). It is of primary importance to see that the temperature of the body be kept up by warm applications. Stimulants may be freely given, and hot coffee injected into the rectum. Strychnine ($\frac{1}{16}$ gr.) should be injected subcutaneously. Inhalations of amyl nitrite, and artificial respiration are advisable.

THOMAS STEVENSON.

CHLOROFORM-HABIT.—See HABITS.

CHLOROFORM, Use of.—See ANÆSTHETICS.

CHLOROSIS (χλωρός, green or sallow).—**SYNON.**: Green-sickness; Fr. *La chlorose*; Ger. *Chlorose*; *Bleichsucht*.

DEFINITION.—A variety of anæmia occurring in a peculiar diathesis or habit of body, characterised by defective formation of the blood and vascular system.

ÆTIOLOGY.—Chlorosis occurs most commonly in young women from the age of puberty to twenty-one, but it may appear or reappear up to the thirtieth year; it is found occasionally in children and older women; it is very rare in men. It is believed to be more common in the higher ranks of life. Beyond these predisposing causes, the origin of the disease lies in peculiar characters of the blood and blood-vessels, to be presently described, which are believed to be congenital, and perhaps hereditary. In such subjects, and under the preceding circumstances, any of the numerous causes of anæmia may be sufficient to excite the appearance of chlorotic symptoms; but those which do so most commonly are bodily growth and development, the establishment of menstruation, disturbances of alimentation—particularly constipation, and an insufficient enjoyment of air, light, and wholesome muscular exercise.

ANATOMICAL AND CHEMICAL CHARACTERS.—The blood in chlorosis presents one strictly essential imperfection. The individual red corpuscle contains less than the normal amount of hæmoglobin, although the total amount of hæmoglobin in the blood in all probability does not fall below normal. The total quantity of blood is much increased (Lorrain Smith), and its specific gravity is much reduced. Both red and white corpuscles are sometimes deficient in numbers, and that proportionately. The liquor sanguinis is unchanged.

With this condition of blood certain associated abnormalities of the aorta and arterial system generally have been described by Virchow. The most striking of these is a hypoplasia or dwarfed condition of the aorta, represented by small calibre, increased elasticity, anomalous origin of the branches, and unequal thickness and fatty degeneration of the intima. The heart is, as a rule, small in early life, but full-sized or even hypertrophied at a later period, with traces of endocarditis. The condition of the ovaries and uterus varies extremely. In some cases the generative organs are described as 'infantile,' while in others they are either immoderately developed, or perfectly normal in every respect. The subcutaneous fat is often abundant; and the viscera present various degrees of fatty change. When the cardio-vascular changes are marked and advanced, there may be extensive secondary disease throughout the body.

SYMPTOMS.—The symptoms of simple chlorosis are those of mild anæmia, with certain important differences which become fewer and less marked, and finally disappear as chlorosis advances to the more serious disease. The present article will be devoted to a description and discussion of them; an account of the symptoms of anæmia will be found under its proper heading.

The appearance of the chlorotic girl is peculiar, inasmuch as the pallor of her complexion is accompanied by natural or even increased fulness, from the excess of subcutaneous fat. In blondes the

CHLORAL-HYDRATE HABIT.—See HABITS.

transparency of the skin is increased; in brunettes it is diminished, and a dull yellowish-grey colour of skin is the result, which, in contrast with the greyish-blue of the eyelids, may appear of a sickly green.

The patient's usual complaint is of this alteration of colour, menstrual disorder, debility, great breathlessness, cardiac symptoms, and various pains. She probably believes that she has heart-disease; her mother fears that she is consumptive. The symptoms and the cardiac and vascular signs closely resemble those of anæmia. But there is this important difference in the phenomena connected with the heart: that in many cases of chlorosis they indicate enlargement. Venous or arterial thrombosis may occur in the limbs and brain. The alimentary system is usually deranged, constipation often being an urgent symptom. The urine is abundant, watery, and pale. There is no dropsy in simple uncomplicated chlorosis. Rheumatic symptoms are occasionally associated. Pyrexia (99° – 103° F.) has been observed in some instances.

The *chlorotic constitution or diathesis* may be recognised by the following characters, which are variously associated in different cases:—Diminutive stature; imperfect sexual development; a history of peculiar anæmia in childhood, of anæmia with menstrual irregularity at puberty, and of previous attacks of symptoms of chlorosis; evidence of cardiac enlargement or mitral disease in the absence of all the ordinary causes of these; the occurrence of endocarditis during pregnancy or *post partum*; and the presence of any of the diseases which will be referred to under the heading of Complications.

COURSE, DURATION, AND TERMINATIONS.—The commencement of chlorosis is generally gradual. If neglected it tends to develop into confirmed anæmia. It is for this reason that pure chlorosis is a rare disease, while anæmia associated with the chlorotic diathesis is comparatively common. The duration of chlorosis is variable. It may reappear, and that more than once; but it is unlikely to return after the age of thirty, especially in the married woman. Death from chlorosis directly is excessively rare, but it may occur rapidly from cerebral thrombosis.

COMPLICATIONS AND SEQUELÆ.—According to Virchow, serious valvular disease and cardiac enlargement may be traced in some of the worst cases of chlorosis to the associated vascular condition; and the mitral valve is peculiarly liable to be attacked by endocarditis in rheumatic, puerperal, or septic fever. Hemorrhages, gastric ulcer, and exophthalmic goitre are believed to occur with comparative frequency in persons of the chlorotic diathesis.

PATHOLOGY.—The deficiency of the individual red corpuscle in hæmoglobin, and the deficiency of the blood in red and white corpuscles, indicate an imperfect growth of the corpuscular elements of the blood. With this blood-state there is associated a hypoplastic or dwarfish condition of the blood-vessels. In the embryo the blood and blood-vessels are developed from the same elements, the former making its appearance within the cells which produce the latter. The view of the pathology of this disease, therefore, adopted by the writer is, that the anomaly of blood and the anomaly of vessels are to be considered as together an expression of some congenital defect of the blood-vascular system, leading to imperfect growth

both of blood and of vessels. Any individual possessing such a blood-vascular system labours under a peculiar diathesis, and is said to be a *chlorotic subject* or to possess the *chlorotic constitution or diathesis*. If the other systems of the body are full-sized (which is not always the case), the dwarfish condition of the arteries of the chlorotic subject and the scanty supply of hæmoglobin may have some difficulty in satisfying the ordinary demands for blood, and especially for oxygen. This difficulty of the blood-vascular system will become a breakdown when the processes of alimentation are deranged, or when the blood is wasted by the excessive demands of growth, development, or hæmorrhage. Such a result is most likely to happen at periods of extraordinary demand within the economy, of which the establishment of menstruation is the chief. The symptoms of uncomplicated chlorosis are due to deficiency of oxygen in the system, while the cardiac enlargement and valvular disease are directly referable to the vascular hypoplasia, that is, to the obstruction caused by the narrow calibre of the aorta. The connection between the blood-vascular condition and that of the generative organs is more complex. On the one hand, the chlorotic diathesis or actual chlorosis interferes with the development and activity of the ovaries and uterus; on the other hand, disorders of the sexual functions are occasionally exciting causes of aglobulism. Many other hypotheses have been advanced to account for chlorosis. Andrew Clark maintained that when the bowels are inadequately relieved ptomaines and leucomaines are produced within the intestine and absorbed into the blood, 'where they originate in girls of a nervous type of organisation those alterations of the constitution of the blood which constitute the true pathology of this anæmia.' Another suggestion is, that sulphuretted hydrogen generated in the bowels as a product of indigestion destroys the organic compounds of iron which go to form hæmoglobin. According to a third suggestion, substances containing animal gum, which are developed in excess within the alimentary canal of the girl or woman, being required for the after nourishment of the embryo, act injuriously on the hæmoglobin-molecule. Still another theory is, that, from a deficiency of hydrochloric acid in the system, iron enters the blood in forms which cannot be assimilated by the corpuscles. Lloyd Jones suggests that chlorosis is the exaggeration of the blood-change that has for its end the storage of nutrient materials for the fetus *in utero*.

DIAGNOSIS.—Chlorosis has chiefly to be distinguished from pernicious anæmia and leucocythæmia. *See* ANÆMIA, PERNICIOUS; LEUCOCYTHÆMIA.

PROGNOSIS.—A speedy cure may be assured in uncomplicated cases subjected to careful treatment, though the liability to recurrence and the possibility of fatal thrombosis must not be forgotten.

TREATMENT.—The administration of iron is invariably successful, provided that, at the same time, a free supply of sunlight be insisted on, the diet and digestion rationally controlled, and physiological rest of the blood and of the organs of circulation be assured. *See* ANÆMIA.

J. MITCHELL BRUCE.

CHOLÆMIA (χολή, bile; and αἷμα, blood). **DEFINITION.**—This term denotes that condition in which the blood contains some or all of the bile-

constituents which have been secreted by the hepatic cells, and subsequently reabsorbed, either directly by the capillaries or *via* the lymphatics. See JAUNDICE; and BLOOD, Morbid Conditions of.

CHOLAGOGUES (χολή, bile; and ἄγω, I move).—**DEFINITION.**—Substances which lessen the amount of bile in the blood.

ENUMERATION.—The principal cholagogues are Mercury and its preparations—especially Calomel and Blue Pill; Podophyllum and its resin; Dry Extract of Euonymus ('Euonymin'), Iridin; Ipecacuanha; Aloes; Rhubarb; Sodium Salicylate, Sodium Phosphate, Sodium Sulphate, and Ammonium Chloride.

ACTION AND USES.—The liver has a twofold action—it *forms* bile, which is poured into the duodenum; and it also *excretes* the bile which has been re-absorbed from the duodenum and carried back to the liver by the portal circulation. Much bile thus circulates continually between the liver and duodenum, while part is carried down the intestine with the feces, and its place supplied by newly formed bile. When the quantity circulating in this way is too great to be completely excreted by the liver, it enters the general circulation and produces symptoms of *biliousness*. These are removed by the so-called cholagogues, which probably act by stimulating the duodenum, and thus carrying the bile so far down the intestine as to interfere with re-absorption. Among the best cholagogues are the preparations of mercury, which, with the exception of the perchloride, do not increase the secreting power of the liver, nor augment the quantity of bile formed by it. Their utility is greatly increased by combination with a saline purgative, which still further clears out the intestine, and completely prevents any re-absorption of bile. Other cholagogues, such as podophyllum resin, rhubarb, and aloes, actually increase the secretion of bile by the liver. At the same time, they probably prevent its re-absorption, in a similar way to mercurials and salines.

T. LAUDER BRUNTON.

CHOLANGITIS.—See GALL-BLADDER AND BILE-DUCTS.

CHOLECYSTITIS.—See GALL-BLADDER.

CHOLELITHIASIS (χολή, bile; and λίθος, a stone).—The conditions associated with gall-stones. See GALL-STONES; and CONCRETIONS.

CHOLERA, ASIATIC.—**SYNON.**: Epidemic, Spasmodic, or Malignant Cholera; Fr. *Choléra Asiatique*; Ger. *Asiatische Cholera*; Ital. *Colera Asiatico*; Hind. *Haijah*; Chin. *Ho-louan*.

DEFINITION.—Asiatic cholera is a specific disease, characterised by violent vomiting and purging, with rice-water evacuations, cramps, suppression of urine, prostration and collapse, tending to run a rapidly fatal course; followed, in favourable cases, by febrile reaction, which may be mild or excessive, simple or complicated. These phenomena resemble those caused by the products of the early decomposition of meat, fish, shell-fish, and certain mushrooms (see PTOMAINES).

ÆTIOLOGY.—The disease is caused by a toxin produced by a spirillum which, gaining access to the intestinal canal, undergoes rapid and enormous multiplication on and in the mucosa and is

discharged with the evacuations. These constitute the medium and means of infection, which takes place most commonly through soil, water, and food; or may be conveyed by clothing or dust; or carried by flies; or in ill-ventilated rooms by exhalations from the bodies of the sick and dead. The disease is spread by human agency, mostly along trade-routes by land, sometimes in ships. Alluvial soil, low level, heat, moisture, organic pollution of soil and water, crowding, and neglect of domestic and personal hygiene are the conditions under which the disease is most apt to prevail.

The more we study the early history of Asiatic cholera, the better shall we understand that every outburst of the disease which has occurred beyond the confines of India can be traced back through a series of cases to that country. The disease prefers, in its extension, land-routes to sea-routes, and localities are invaded by cholera in consequence of communication with places previously attacked, commencing with Lower India.

The evidence in favour of the communicability of cholera by means of water or food contaminated with cholera-dejecta is overwhelming.

The bacteriology of the disease is discussed in a special article. See CHOLERA, Bacteriology of.

ANATOMICAL CHARACTERS.—The external appearances of the bodies of those who have died of cholera include the mottled skin, shrunken and livid appearance of the limbs, and other features hereafter described as characteristic of the disease during the stage of collapse. The temperature of the body rises after death, and it remains warm for some time. Rigor mortis sets in speedily, and is sometimes accompanied with muscular contractions, which displace the limbs of the corpse.

After death in the stage of collapse the only alteration to be discovered in the tissues and the blood is to be accounted for by the rapid drain of water into the intestinal canal which has occurred during life. The mucous surface of the stomach and small intestines is injected and swollen, and sometimes covered in places with an aphthous pellicle. The epithelium is shed during life, and drops off from the mucous membrane in large patches within an hour and a half after death. Dr. Koch states that the cholera-bacillus in many cases is found between the epithelium and the basement-membrane, and also within the tubular glands; 'it also settles in large numbers on the surface of the villi of the intestines, and often had penetrated into their tissue, and in some instances had passed as far as the muscular layers of the intestine.' The epithelial cells lining the kidney-tubules, bladder, and other organs are found detached from the basement-membrane, but they, like the blood, do not contain the cholera-bacillus. In both cases the epithelial cells show signs of degeneration.

Abnormalities of a specific nature, especially with reference to the amount of blood contained in the right side of the heart and lungs, have been described by pathologists as being characteristic of Asiatic cholera; and in many instances after death from this disease, if the *post-mortem* examination is delayed for a few hours, the right side of the heart will be found full of blood, together with the pulmonary artery and its divisions, while the lungs are collapsed and bloodless. But there are numerous exceptions to this state of the heart and lungs, and the condition above described is not infrequently

due to *post-mortem* changes; for if the bodies of those who have died of cholera be examined immediately after death, the left side of the heart will often be found as full of viscid blood as the right side. A peculiar shrunken condition of the lungs exists, depending on the dry and empty state of the bronchi, which allows the elasticity of the organs to drive the air out of them more completely than usual after the chest is opened. On the other hand, when death has occurred during reaction, the smaller tubes are often found full of pus, and parts of the lungs may be oedematous, or even in a state of broncho-pneumonia. The tissues in the acute stages are dry, and the blood is inspissated. Dark or light clots are prone to form in the heart and large vessels. In the stage of reaction inflammatory and even necrotic lesions of the viscera are often seen.

SYMPTOMS.—Asiatic cholera is most virulent and deadly at the commencement of an epidemic, and then usually begins without premonitory symptoms. The patient feels well up to within a few hours of the attack, or, it may be, goes to bed and sleeps soundly through the night, and immediately on rising in the morning is seized with violent purging and vomiting. After the first outburst of the epidemic, as a rule, cholera commences with diarrhoea, the stools being copious and watery, followed by great prostration of strength, with a peculiar feeling of exhaustion at the pit of the stomach; the sick person suffers from nausea, but seldom from actual vomiting or pain at the outset of the attack. If judiciously treated, many patients recover from this, the *first*, or *invasion-stage* of cholera; but if neglected the tendency of the disease is to grow rapidly worse. The stools become very frequent, and resemble in appearance and consistency the water in which rice has been boiled. These liquid evacuations flow away from the sick person with a sense of relief rather than otherwise. But the patient now commences to vomit, first throwing up the contents of his stomach, and subsequently all the water he drinks, mixed with mucus and disintegrated epithelium. The fluid is ejected from his mouth with considerable force, and this adds to the increasing prostration which is one of the most urgent and marked features of the disease. The patient complains of intense thirst, and a burning heat at the pit of his stomach; he suffers also excruciating pain from cramps in the muscles of the extremities, occasionally also of the trunk; he is terribly restless; and his urgent cry is for water to quench his thirst, and that some one may rub his limbs, and thus relieve the muscular spasm. Although the temperature of the sick person's body falls below the normal standard, he complains of feeling hot, and throws off the bed-clothes in order that he may keep himself cool. The pulse is rapid and very weak; the respirations are hurried; and the patient's voice becomes husky. His countenance is pinched, and the integument of his body feels inelastic and doughy, while the skin of his hands and feet becomes wrinkled and purplish in colour. The duration of this, the *second*, or *evacuation-stage* of cholera, is uncertain. It may last for two or three hours only, or may continue for twelve or fifteen hours; but so long as the pulse can be felt at the wrist, there are still good hopes of the sick person's recovery. The weaker the pulse becomes, the nearer the patient is to the *third*, or *collapse-stage* of cholera, from which probably not more than thirty-five per cent. recover. The result, however, depends much on the condition of the

patient's heart. It is quite possible, although the cases must be rare, that a sudden outpouring of fluid into the intestinal canal has been sufficient to cause syncope and death in persons suffering from weak heart, before the liquid contents of the bowels have had time to be ejected through the mouth or anus (*cholera sicca*).

In the third stage of the disease the vomiting and purging continue, although in a mitigated form; and the skin is covered with clammy perspiration, especially if the cramps are still severe. We are now unable to feel the pulse at the wrist, the lividity of the extremities and surface of the body increases, the patient cannot speak above a low whisper, his breathing is very rapid, his eyeballs are deeply sunk in their sockets, and his features are marvellously changed within a few hours. The urine is suppressed. The surface-temperature of his body may fall as low as 94° F. The patient remains terribly restless, longing only for sleep, and that he may be supplied with water. His intellect is clear, but he seldom expresses any anxiety regarding worldly affairs, although fully conscious of the dangerous condition he is in; sleep, and a plentiful supply of drinking-water, are the sole desires of a person passing through the collapse-stage of cholera. This condition seldom lasts for more than twenty-four hours, and reaction either commences within that period, or the patient dies in collapse, or he passes on into the *tepid stage*, which in ninety-nine cases out of a hundred ends speedily in death. In the tepid stage of the disease the patient's body feels cold to the touch, but the temperature, as shown by the thermometer, begins to rise rapidly, sometimes marking 99° or 100° F., or even higher in the rectum. The purging and vomiting cease; and the patient lies in a semi-comatose state, his eyes half-open, the ocular conjunctiva being deeply congested, the cornea hazy, and the pupils fixed. The pulse can be felt at the wrist, but the respirations are very hurried, suppression of urine continues, the patient's body is bathed in a cold clammy perspiration, the skin becomes of a dusky red hue, and death too frequently closes the scene within a few hours. The condition in some cases resembles the typhoid state.

On the other hand, the sick person having been in the collapse stage of cholera some twenty-four hours (it may be a longer or shorter period), the temperature of his body may begin to rise, gradually creeping up to the normal standard; the respirations diminish in frequency; the pulse returns; the patient can sleep, and after some thirty-six hours may pass a little urine: in fact, the functions of animal life are slowly restored, and the sick person recovers his health. This desirable result, however, is not infrequently thwarted by various complications which arise during the stage of reaction. Of these events the following are the most important:—suppression of urine; gastritis and enteritis; pulmonary congestion; meningitis; sloughing of the cornea; abscesses over the body; the formation of coagula in the right side of the heart or pulmonary arteries; hæmorrhage from the bowels; and roseola choleraica. Variations in the intensity, symptoms, and complications of cholera have suggested a definition of different forms of the disease, but such divisions are artificial, and the multiplication of names serves no useful purpose.

DIAGNOSIS.—The question of the diagnosis of Asiatic cholera is discussed in the articles on

CHOLERAIC DIARRHŒA, and CHOLERA, Bacteriology of.

PROGNOSIS.—The means of forming a prognosis in cholera may best be gathered from the preceding account of the disease. Speaking generally, the prognosis depends largely upon the previous health and condition of the patient, the rapidity of development of the disease, the severity of the symptoms, and upon the period in the epidemic at which the case occurs—that is, according as the patient has been seized at the outbreak, at the height, or towards the end of an epidemic.

TREATMENT.—In the first stage of Asiatic cholera we should endeavour to stop the purging, and without doubt opium is the drug upon which we may with the greatest confidence rely for effecting this purpose. When practising in the endemic area of cholera, one of the writers was in the habit of carrying about pills containing one grain of opium and four of acetate of lead, so that, if called to see a patient suffering from the disease in its early stage, no time was lost in administering one of these pills suspended in water. Similar combinations of opium with astringents and anti-spasmodics enter into the composition of a great variety of cholera-pills and tinctures employed in India. The next thing done was to make a large mustard poultice, and apply it over the whole surface of the abdomen. The patient was ordered to remain in bed, the evacuations being received in a bed-pan, and he was allowed nothing in the shape of food or water; but he might suck as much ice as he felt inclined for.

If the patient was purged after the first pill, a second was given, followed by a third if the second was ineffectual. It often happened that the first or second pill, together with the mustard poultice, ice, and rest, sufficed to check the progress of the disease, and the patient recovered. Supposing this treatment did not succeed, or that on first seeing the patient he was found to have passed into the second stage of the disease, we should still prescribe the pill, as above directed, suspending it in water, because in the solid form it might be rejected entire, and under any circumstances it would take time to be dissolved by the fluid contained in the stomach; the mustard poultice also should be applied, and the sick person kept warm and in bed. Ice is invaluable in this stage of the disease, and unless a person has passed through an attack of cholera, it is impossible to realise the immense relief it affords. It should be given in small lumps, the sick person eating and swallowing as much as he chooses. He will frequently devour a pound or two of ice in the course of an hour, and he cannot take too much of it. In the treatment of cholera there can be no question as to the value of ice. The patient should be prohibited from drinking water or any other fluid beyond that which he gets from the ice. The practitioner must be firm on this point, turning a deaf ear to the entreaties of the sick man or his friends that he may be permitted to swallow even a small quantity of water; for if they once break through this rule, it will be impossible to limit the amount of liquid the patient may consume. If this treatment does not check the progress of the malady, we may prescribe three grains of acetate of lead and fifteen drops of diluted acetic acid in water every second hour, and fifteen drops of diluted sulphuric acid in water every alternate hour, so that the patient should take a draught, first of one mixture then of the other,

every hour. Five drops of spirit of camphor may be added to each dose of the medicine, but this drug requires care in its administration, and should seldom be continued beyond five or six doses of from five to ten drops each. Should the vomiting be very severe, in spite of the free administration of ice, a second mustard poultice should be applied over the abdomen; all medicine must then be omitted for an hour and a half, after which time twenty grains of calomel may be sprinkled on the patient's tongue, and he should be made to wash it down with a little iced water. The cramps are best relieved by hand-friction, and if very severe, ease may from time to time be given by allowing the patient to inhale some ether. Powdered ginger is used in India to aid friction of the clammy surface, which may in cases of profuse cold sweating be rubbed with a soft towel. Hot-water bottles should be applied to the soles of the patient's feet, and also to his legs and abdomen.

Should the disease have reached the collapse-stage, there is but little we can do for the patient. Ice must still be given, and, if the purging is frequent, the sulphuric-acid draught (but no opium) may be administered every hour; heat and friction may with advantage be applied to the surface of the body; and the patient may now be permitted to drink iced water in moderation, provided it does not increase the vomiting. Wine and stimulants, if given by the mouth, do harm in this stage of cholera; but, if the purging has abated, enemata of warm beef-tea and brandy may be administered by the rectum every third hour. The restoration from collapse which follows intravenous saline injections is marvellous; but in most cases the fluid escapes rapidly through the intestines and the patient relapses. For this reason Cox, of Shanghai, has proposed continuous injection until reaction has been fully established. This measure is still on trial. Injections of water have been made into the cellular tissue, bladder, and peritoneum, but without much, if any, advantage. Injection of sulphuric ether has also been tried with evident, but mostly temporary, success. When reaction comes on, we must guard against doing too much—it is very rare indeed to see a patient in this condition sink from exhaustion, but probably many lives are lost by endeavours erroneously made, under the idea of keeping up the patient's strength. Iced milk or arrowroot is all that should be allowed to be given by the mouth for some time after reaction has set in; but enemata such as those above mentioned, administered *per rectum* every five or six hours, or nutrient suppositories, are often beneficial, especially if the stomach remains irritable. Under these circumstances we not infrequently find that a small quantity of solid food is digested, when soup and liquids are rejected. In each case, however, the dictates of common sense and experience must guide the medical practitioner in his treatment of the sick person through the convalescent stage of the disease.

With reference to the treatment of suppression of urine after cholera, we should get the patient to drink about half a pint of water every second hour, so as to add fluid to his blood. Dry-cupping over the loins should be employed; and ten drops of the tincture of cantharides in water administered every hour, until a drachm of the drug has been given. It need hardly be remarked that suppression of urine after cholera is a most dangerous

complication, and there is very little that can be done to restore the suspended functions of the kidneys.

PREVENTIVE TREATMENT.—The prophylaxis of cholera includes precautions against attack of individuals, protection of communities, and avoidance of invasion. The best safeguards against the disease are the maintenance of a high standard of health and scrupulous personal and domestic cleanliness. Alcoholism, bodily fatigue, and mental worry are special predisponents, and anything which tends to upset digestion or derange the bowels must be rigorously shunned. The use of saline purgatives when cholera is about is dangerous. Haffkine's method of anti-choleraic inoculation confers protection for a time, and diminishes the risk of contracting the disease (*see* CHOLERA, Bacteriology of). As a person affected by the disease is undoubtedly capable of communicating it to others and serving as a nidus and centre of diffusion, he is obviously an object of concern; but seeing that the communication from the sick to the healthy is effected solely by means of the intestinal evacuations, the safety of the community is sufficiently ensured by dealing with these effectively, with a view to the destruction of the contagium which they contain. If this is done the patient need not be considered a subject of danger or dread, and need not be isolated or avoided.

Among persons predisposed to its influence, the infecting organisms of Asiatic cholera will manifest their effects on the system within five days of having been swallowed, but the germs of the disease do not always engender symptoms of virulent cholera. Nevertheless, in milder cases of the disease the evacuations passed by the patient may contain the microbes of cholera, and may therefore, under favourable conditions for its development, produce a deadly type of the malady. Consequently, the following remarks are applicable to instances of so-called *cholérine*, as well as to the severer forms of cholera. If the disease has appeared within a neighbourhood, a searching examination must be made into the condition and source of the local water-supply, not overlooking that of the milk, which is too often diluted with water before being sold. All surface- and doubtful wells or reservoirs (especially those in the proximity of drains and cesspools) should be immediately closed; and it is desirable that the drinking water, before being consumed, should be carefully filtered and boiled. All accumulations of house refuse and filth must be removed; and dirty places, both within and without, must be freely disinfected and cleansed. There is no necessity when the disease is prevalent for making any alteration in the usual diet; but in times of cholera we cannot too strongly insist on at once checking any tendency to diarrhoea, especially if it be of a watery nature.

If called to treat a case of Asiatic cholera, care should be taken that the rice-water stools, and the matters vomited, are disinfected by means of a solution of mercuric chloride or some other germicide, which should be poured over the bottom of the vessel into which the evacuations are received from the patient; and directly the dejecta are passed, an equal quantity of a solution of one part of carbolic acid to twenty of water should be added to them, and they must be immediately taken from the patient's room, and disposed of as

follows:—If the sewage of the locality is conveyed away by means of a constant water-supply, the disinfected cholera evacuation should be thrown at once into the sewer. Drains used for a purpose of this kind must, however, be constantly flushed with a mixture containing about an ounce of ferrous sulphate to a pint of water. But if the drainage of the place passes into a cesspool, the disinfected cholera-stools should be buried in a deep hole in the ground, removed from wells, and, if possible, from human habitations. It is a most dangerous practice, however carefully cholera-stools have been disinfected, to allow them access to a cesspool. The room in which the patient has been treated must be freely disinfected, and his bedding subsequently burnt. If the sick person should die, the corpse is at once to be placed in a coffin containing a mixture of lime, charcoal, and carbolic acid: in fact, the body should immediately be buried in a mixture of this kind, and the coffin with its contents committed to the grave, or, better, consumed by fire within twenty-four hours of the patient's death.

By far the most important preventive measures to be adopted against cholera are to provide a pure supply of drinking-water, good drainage, ventilation, and cleanliness; for these means, if rightly enforced, must prevent the cholera-germ from spreading among human beings. In India when cholera breaks out among troops or prisoners, the practice is to move them into cholera-camps, situated at a distance from the infected locality, and located where the disease is absent and rare. This measure has been attended with good results, and is applicable to individuals as well as communities.

The Vienna Cholera Conference has decided that quarantine is applicable to the circumstances of cholera; but this subject, together with the duties incumbent on sanitary and port authorities with reference to the preventive treatment of the disease, hardly falls within the scope of this article. *See* QUARANTINE.

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KENNETH MACLEOD.

CHOLERA, ASIATIC, Bacteriology of.—

The *Comma-Bacillus* of Koch, the *Spirillum* or *Vibrio Cholera Asiatica*, now regarded as the specific cause of Asiatic cholera, is a minute rod-shaped organism, each cell measuring about 1.2μ in length and 0.5 to 0.6μ in thickness. These rods are curved to a varying degree, some forming a small segment of a circle, others being semicircular, and others again united in pairs so as to form an S-shaped curve. The name 'comma-bacillus,' by no means a good one, as the organism bears little resemblance to a printer's comma, has been universally adopted. Under cultivation, longer filaments, more or less spirillar or cork-screw in form, develop. The cells are actively mobile and should possess a single terminal flagellum, though there is some variation in this respect: thus the Massowah cholera-spirillum is described as having two flagella at each end, and an Indian variety has been stated to be non-flagellated. The cholera-spirillum does not form spores, though in old cultures certain vacuoles or granules of formed material have been described as such, and the rods lose their regular shape, and swollen and distorted involution-forms occur. The organism can be readily stained with the aniline dyes, a weak solution of fuchsin being perhaps the best;

but it is decolourised by Gram's method. See BACTERIA: coloured plate.

It is aerobic and facultative anaerobic, and may be readily cultivated upon the ordinary laboratory media at a temperature of from 20° to 37° C. According to Percy Frankland, although it grows readily in an atmosphere of hydrogen it does not develop in one of carbonic acid gas. In gelatine plates at 22° C. the cholera-spirillum forms roundish colonies which have slightly irregular margins and an uneven surface. At an early stage (20 hours) the colonies on gelatine plates appear whitish and somewhat less transparent at the centre than at the margins; later (40 hours) they become finely granular with uneven margins and with a marginal band of fine radial markings. Slow liquefaction of the gelatine occurs, and in consequence after 24 hours the plate is pitted and appears to be covered with minute air-bubbles. In a gelatine-stab-culture a cream-coloured growth develops along the puncture with slow liquefaction commencing at the surface and spreading downwards; in 48 hours there is a goblet-shaped area of liquefaction the size of a pea just at the surface, and having the appearance of a large air-bubble. The rate of liquefaction of gelatine varies considerably with different strains; in old laboratory-stocks it may be extremely slow. As liquefaction proceeds the liquefied area first assumes a hemispherical and later an elongated finger-like shape, to the bottom of which the whitish flocculent masses of growth sink.

On agar-agar, the surface-growth is thick, moist, smooth, and cream-coloured, generally with crenated margins. In stab-sugar-agar the growth is mostly confined to the surface and upper portion of the puncture, and there is no production of gas. In broth there is a free growth, the broth becomes turbid with a slight deposit, and a delicate film on the surface. In peptone-water the organism develops rapidly, especially at the upper layers of the fluid, with the formation of a distinct surface-pellicle; growth being best when the medium contains 1 per cent. of salt (Durham's modification). On potato a thin brownish layer develops at 37° C. In milk the organism multiplies rapidly with curdling and the production of acid, as is also the case on glucose-agar. Two important chemical changes are brought about by the growth of the organism in suitable media, viz. the formation of indol, and the reduction of nitrates into nitrites. Peptone-water is by far the most suitable medium for cultivating the organism in order to demonstrate these changes. A 24-hour culture treated with a little pure hydrochloric or sulphuric acid gives a bright rose-red colour, the formation of which is explained as follows:—The organism during its growth has formed indol and has also reduced the traces of nitrate present in the culture-medium to the condition of nitrite, the addition of the acid liberates nitrous acid which combines with the indol to form a red-coloured compound, nitroso-indol. Many organisms during their growth produce indol, the commonest example being the colon-bacillus, but it is the exception for nitrates to be reduced; hence in employing this reaction as a test it is necessary to use a pure acid, i.e. one free from nitrous compounds; should these be present, common organisms such as the colon-bacillus might give the reaction.

The cholera-spirillum retains its vitality in cultures for many weeks; it is readily killed by heat, its thermal death-point according to Kitasato being

55° C. with an exposure of 10 minutes; weak solutions of the common antiseptics also destroy it. In ordinary moist soil it remains alive for from 4 to 8 weeks, but in dry soil and in peat it dries out in a few days. In sterilised distilled water it usually dies within about 24 hours, but the addition of a small amount of sodium chloride greatly increases its longevity, and in sterilised potable water it may survive for many months; in unsterilised potable water its survival is greatly influenced by the presence of salt and of other bacteria. In unsterilised sewage it may live for 2–4 weeks (Houston).

The nature of the chemical products elaborated by the cholera-spirillum is but little known. It produces a peptonising and also a diastatic ferment, and Brieger has isolated cadaverin, putrescin, and two other basic bodies; but we now know that, owing to the methods of isolation employed, these are not improbably artifacts. Pfeiffer believes that the poison is intracellular. Brieger and Fränkel isolated a toxalbumen, Gamaleia a ferment-like body, and Westbrook various proteoses and proteid-like substances from cultures of the cholera-spirillum. Lastly, Metschnikoff has obtained a toxin by cultivating in a salt peptone-gelatine medium.

Relation of the Cholera-Spirillum to the Disease.—The cholera-spirillum may be said to be constantly found in the intestinal contents and discharges in cases of true Asiatic cholera, reaching the maximum degree of abundance at the height of the disease, and then almost entirely replacing the common bacteria of the intestinal canal, so that it is present almost as a pure cultivation; it has not been found in the excreta of healthy individuals. The organism is not met with in the internal organs or blood, but is confined to the intestinal canal, where it may penetrate beneath the intestinal epithelium.

The lower animals do not suffer from any condition at all resembling cholera (chicken-cholera is an entirely different disease), and it has therefore been difficult to reproduce the disease experimentally by inoculating or by feeding with pure cultivations of the spirillum. Koch, however, by washing out the stomach of guinea-pigs with alkaline solutions and paralysing peristalsis by means of opium or morphine, previous to injecting the cultivations into the stomach, induced a condition closely resembling cholera. Metschnikoff also has found that young suckling rabbits suffer from a choleraic diarrhoea when fed with cultures. Cases of accidental infection of man through pure cultivations are also on record. Thus in 1884, a student who was attending Koch's course on cholera in Berlin became ill with a severe attack of cholera, although there was at that time no cholera in Germany. A similar case is reported by Metschnikoff; and Dr. Oergel, of Hamburg, while injecting a guinea-pig, accidentally infected himself and died from the effects. On the other hand, Klein and others have swallowed cultures with negative results. Metschnikoff ascribes the immunity of animals to intestinal cholera to the inhibitory action of the other intestinal bacteria upon the cholera-spirillum, and he believes that the immunity or susceptibility of man and animals to intestinal cholera is largely influenced by the microbial flora of the digestive tract. Undoubtedly, in man, digestive disturbances are an important predisposing cause to an attack. The acid gastric juice probably plays an important rôle. Thus Macfadyen found that when the spirilla were administered in water to a fasting animal, they

could be detected in the intestine, but when the vehicle was milk none could be found, the inference being that the acid gastric juice, secreted on the introduction of the milk, prevented their passage.

The Bacteriological Diagnosis of Cholera.—The bacteriological diagnosis of cholera is carried out as follows. Cover-glass specimens of the rice-like flakes present in the evacuations or intestinal contents are prepared, stained with Löffler's alkaline methylene-blue, washed, dried, and mounted. If, upon a microscopical examination, large numbers of curved rods lying parallel to each other in groups are detected, the diagnosis of Asiatic cholera may be made with certainty; this, however, only occurs in perhaps half the acute cases. If this fails cultivations must be prepared. Sets of gelatine plate-cultures should be made, using a loopful of the dejecta, or, better still, an emulsion of a rice-like flake; a set of agar-plates may also be prepared by smearing a rice-like flake over agar-films in Petri-dishes. The plates being incubated at 22° and 37° C. respectively, the colonies of the cholera-spirillum will be recognisable in the gelatine-plates in from 24 to 30 hours, and in the agar-plates in from 12 to 16 hours. Peptone-water cultures should also be prepared at the same time from the rice-like flakes, the peptone-water being preferably contained in small Erlenmeyer flasks and forming a shallow layer at the bottom. In from 8 to 12 hours the upper layers of the culture-fluid may be examined for the presence of spirilla and gelatine-plate cultures, and fresh peptone-water ones also made for subsequent examination. After this the peptone-water cultures may be tested for the cholera-red reaction by the addition of a little *pure* hydrochloric or sulphuric acid.

The blood-serum of cholera-patients produces an agglutination of the cholera-spirillum similar to that produced by typhoid-serum on the typhoid-bacillus, and this test may be employed for diagnosis. It is stated that this reaction may be obtained as early as the first day.

The bacteriological diagnosis of cholera has of recent years become somewhat complicated by the detection and isolation from the waters of the Seine, Marne, Elbe, Rhine, Danube, and Spree, of spirilla bearing a close resemblance to the cholera-spirillum, but differing from it in certain minor particulars. Considerable light has been thrown upon the relation of these spirilla to the cholera-spirillum by the Bordet-Durham (agglutination) test, and by Pfeiffer's specific reaction. In the latter a mixture of an emulsion of the organism with a small quantity of the serum of a highly immunised animal is injected into the peritoneal cavity of a healthy guinea-pig. Half an hour to one hour afterwards the peritoneal fluid is examined microscopically, and if the reaction be positive, the organisms will be found in all stages of degeneration, in which case, according to Pfeiffer, the organism is to be regarded as belonging to the same species as that employed for immunising the animal from which the 'test' blood-serum was obtained. These cholera-like spirilla are now generally regarded as being harmless varieties of the cholera-spirillum—descendants probably of a cholera-spirillum of some cholera-epidemic of bygone years. The variability of the cholera-spirillum is demonstrated by Klein's experiments.

Other spirilla.—Mention must be made of two other well-known spirilla, which bear a superficial resemblance to the cholera-spirillum, but which can

be easily distinguished from it. They are the spirillum of Finkler and Prior, and the *Spirillum Metschnikovi*. The former, obtained from the dejecta in certain cases of so-called cholera nostras, liquefies gelatine much more rapidly than the cholera-spirillum and only gives a feeble cholera-red reaction after 2-3 days' growth. The *Spirillum Metschnikovi*, obtained from the intestinal contents of poultry, also liquefies gelatine more rapidly than the cholera-spirillum, but, like the latter, gives the cholera-red reaction; it is very pathogenic to pigeons. A spirillum obtained from cheese (Deneke's spirillum) likewise liquefies gelatine rapidly and does not give the cholera-red reaction. Litmus-milk is decolourised by the cholera-spirillum owing to reduction, but not by the Finkler-Prior or Deneke spirillum (Cahen's test).

Preventive Inoculation against Cholera.—Various methods of producing immunity to cholera have been suggested, but that of Haffkine alone needs mention. The vaccines employed are two in number: the first or weak, and the second or strong. The first vaccine is prepared from ordinary laboratory-cultures of the cholera-spirillum—attenuated by growing on surface-agar at 38° C. in tubes through which a current of moist air is being continuously passed. The second or strong vaccine is prepared from cholera-cultures, the virulence of which has been artificially increased by growing in the peritoneal cavity of a series—generally about twenty—of guinea-pigs. The vaccine itself is made by emulsifying agar-cultures with some sterile broth and adding a small quantity of carbolic acid or lysol. The dose of vaccine is injected hypodermically, the second or strong vaccine being administered from 3 to 5 days after the first or weak one. The injections cause some malaise, headache, and slight fever, which pass off in two or three days. The results of the anti-cholera inoculations seem to be decidedly favourable. For example, in Calcutta, taking the same households during the same period of time, there were among 654 uninoculated, 71 deaths, a mortality of 10·86 per cent.; among 402 inoculated, 12 deaths, a mortality of 2·99 per cent., a reduction of mortality by 72·47 per cent.

Cholera Anti-Toxin.—Attempts have been made to prepare an anti-toxic serum for cholera, but without much success. Metschnikoff by means of his toxin (*vide ante*) has immunised goats, so that 1 c.c. of their serum would neutralise four times the lethal dose of the toxin. Using young rabbits and producing an intestinal cholera by feeding, experiments showed that of the rabbits treated with this anti-toxic serum 51 per cent. survived, while of the untreated only 19 per cent. recovered.

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CHOLERA INFANTUM.—See DIARRHŒA.

CHOLERA NOSTRAS.—See CHOLERAIC DIARRHŒA.

CHOLERAIC DIARRHŒA.—SYNON.: Cholérine; Sporadic Cholera; Cholera Nostras; Infantile Cholera; Fr. *Choléra sporadique*; Ger. *Sporadische Cholera*.

DEFINITION.—An acute catarrhal affection of the mucous membrane of the stomach and small intestines, attended with vomiting and diarrhœa and considerable prostration.

ÆTIOLOGY.—The disease is caused by an organic poison, probably of microbic origin. It prevails

sporadically in single or small groups of cases and does not exhibit any tendency to travel or spread. This form of diarrhœa is especially liable to attack those living in unhealthy surroundings.

In a hot and moist climate like that of Lower Bengal, choleraic diarrhœa is an affection which we meet with at all seasons of the year, and it is especially prevalent among infants who are being reared on cow's milk, or on other kinds of food prone to undergo putrefaction. Among the fish-eating Hindoos we frequently see several members of the same family who have been seized with symptoms of choleraic diarrhœa, attributable to the patients having partaken of fish which was slightly tainted. In fact, there are few more certain sources of this form of diarrhœa than stale fish; and it is evident that, whatever the deleterious material may be which food of this description contains, the mere fact of keeping it in boiling water for some time does not destroy its poisonous properties.

It occasionally happens that cases of choleraic diarrhœa occur among people residing in malarious districts, the diarrhœa taking the place of the cold stage of a fit of ague; patients in these circumstances may be seized with all the symptoms of severe cholera, but they almost invariably recover from the attack.

SYMPTOMS.—Choleraic diarrhœa begins suddenly: that is, the patient, whether an infant or an adult, has probably up to the commencement of the attack been in good health; there are, in fact, seldom any premonitory symptoms. A *child* may perhaps look somewhat paler than usual, and has a dark ring under his eyes, but beyond this appears to be perfectly well. Shortly after taking food, the infant vomits up a quantity of uncoagulated milk, the evacuation not being curdled like that from an overloaded stomach, the gastric secretion no longer having the power of coagulating the casein of milk. Soon after vomiting, or it may be before, the child commences to pass from the bowels an acid greenish-yellow fluid, containing flakes and often lumps of undigested food. The patient becomes very thirsty, is restless, and evidently in considerable pain, crying and drawing his legs up towards the abdomen. If these symptoms continue, the evacuations become colourless, resembling in appearance the rice-water stools of Asiatic cholera. The temperature of the body falls, the face becomes of a dusky hue, the features are pinched and the eyeballs deeply sunk in their sockets. The fontanelles are depressed; the child is evidently terribly prostrated, his pulse can no longer be felt at the wrist, and his crying passes into a weak whimpering; he eagerly drinks water when offered him; and, as the exhaustion increases, convulsions supervene, and the child dies within a few days or even hours. On the other hand, the symptoms may abate at any stage of the disease, and the little patient gradually recover his health.

In the *adult* the symptoms of choleraic diarrhœa are much the same as those above detailed. There are seldom any premonitory symptoms, and the attack begins with nausea and vomiting, together with a sensation of exhaustion referable to the pit of the stomach; the vomiting is speedily followed, or it may be preceded, by purging; copious watery discharges are thus passed out of the body, and the larger and more rapid the evacuations the more they come to resemble the serum of the blood, which, in fact, drains into the intestinal canal and

passes away from the stomach and bowels. The patient naturally complains under the circumstances of intense thirst. He is very restless; and at the commencement of the attack suffers from colicky pains in the abdomen, and subsequently from spasms and cramps, which often seize the muscles of the extremities. The pulse becomes small and weak, the respirations are hurried, the voice feeble, and the countenance pale and shrunken. The urine is scanty or suppressed; and the temperature of the body falls one or two degrees below the normal standard. These symptoms, as a rule, gradually subside, the purging and vomiting cease, and the patient falls off to sleep, waking more or less exhausted in proportion to the severity of the attack, but he usually recovers his health rapidly.

DIAGNOSIS.—The discrimination of the various forms of cholera rests upon data which may be classed as clinical, epidemiological, and bacteriological. The clinical features of all forms present a very close resemblance; so that given a mild case of cholera Asiatica and a severe one of cholera nostras, it would be difficult if not impossible to establish a diagnosis by comparison of the phenomena presented by each. To the practitioner who has had experience of both diseases the faces of cholera Asiatica reveals special malignity which, when the induction is large, can hardly be mistaken. Inquiry regarding the circumstances of cases and outbreaks is a valuable aid to diagnosis. A history of having partaken of tainted food or mushrooms, and the limitation of seizures to those who have so partaken, are sure guides to the formation of an opinion. In mushroom-poisoning fragments of the material may be discovered in the vomit or stool. A knowledge that epidemic cholera is in the locality or neighbourhood causes suspicion to attach to even mild cases of diarrhœa. The case-mortality of the disease when it attacks numbers is a strong indication of its nature. For the bacteriological diagnosis see CHOLERA, Bacteriology of.

PROGNOSIS.—Although choleraic diarrhœa in its more severe forms resembles mild cases of Asiatic cholera, it is a comparatively harmless disease. Unless among young infants, or old and sickly people with weak hearts, no matter how threatening the symptoms may be, however great the collapse and depression of the patient may seem, a previously healthy adult seldom dies of choleraic diarrhœa.

TREATMENT.—In cases of simple cholera occurring in the child, the important point we must enforce in our treatment is that the affected part shall have rest. In practice, however, it is often difficult to persuade parents and nurses that an infant can exist uninjured for ten or fourteen hours on iced water; nevertheless, we must insist on a plan of this kind being carried out. The little patient will eagerly swallow cold water, either from a bottle or spoon, and the child may be allowed to take as much cold water as he requires, and to suck ice, which may be wrapped up in the corner of a handkerchief and put into his mouth. A poultice made of equal parts of mustard and flour, applied over the abdomen, is often very useful in this form of disease. With reference to drugs, should the treatment above indicated not relieve the symptoms, or should the vomiting be very constant, from two to four grains of calomel may be given, and repeated if necessary in an hour's time; however, if the diarrhœa is the more prominent symptom, calomel is not required, but a teaspoonful of castor oil should

be administered. After the bowels have been cleared out, if the serous discharge continues, we should order astringents, in the form of $\frac{1}{2}$ of a grain of acetate of lead every hour, or $\frac{1}{16}$ of a grain of nitrate of silver, until the purging subsides. Tannic acid in combination with diluted sulphuric acid and sugar is frequently a useful combination to administer to children in cases of this description. With reference to opium, much as we dislike prescribing it for infants, it may be necessary in cases of simple cholera; but it should hardly be given in a mixture to be administered from time to time by a nurse. Opium under these circumstances can only be admissible when given by the medical attendant himself, in doses of one, two, or three minims of laudanum in a little weak brandy and water, carefully watching its effects. If the drug causes the child to sleep for a few hours, it may act almost like a charm: the infant awakes comparatively well. But if the opium has no such effect, we may be tempted to repeat the dose, but can scarcely give it a third time, at any rate until some hours have elapsed since the administration of the second dose. The symptoms having subsided, the child's diet must be strictly attended to, a good healthy wet-nurse as a rule being an urgent necessity in the case of infants. Lime-water may with advantage be mixed with the child's food. The diet must be most carefully ordered. See DIARRHŒA.

With reference to the treatment of adults suffering from choleraic diarrhœa, we must bear in mind that, unless among old and debilitated persons, the patient will, as a rule, recover without medicine. If therefore called to prescribe for a case of this complaint, we may order fifteen minims of laudanum, or a drachm of the compound tincture of camphor in water, to be taken (supposing the patient is very sick) immediately after vomiting; half the above dose may be given at the expiration of one hour; and again after another hour, unless the symptoms have in the meantime subsided. A large mustard poultice should be applied over the abdomen, and the patient must be confined to bed, and kept on ice and iced water; he should not, however, be permitted to swallow too much liquid. If the vomiting be severe, a scruple of calomel may be given to an adult, or in the first instance an effervescent mixture with hydrocyanic acid may be employed to allay the sickness. On the other hand, should the diarrhœa be excessive, we may with advantage prescribe four grains of acetate of lead and ten drops of diluted acetic acid every second hour; or pills containing a drop of creasote, a quarter of a grain of nitrate of silver, a grain of camphor, and two grains of Dover's powder, to be repeated after each loose motion.

Among old and weakly persons, and also in the case of infants, it is often necessary to administer brandy and water from time to time, according to the state of the patient's pulse.

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KENNETH MACLEOD.

CHOLERINE.—A term applied to a class of cases which occur during the prevalence of cholera, in which the milder symptoms of the disease are present. It has also been used to designate the poison on which cholera is supposed to depend. See CHOLERA, ASIATIC; and CHOLERAIC DIARRHŒA.

CHOLESTEATOMA (χολή, bile; and στεάτωμα, [Galen], a fatty tumour).—An encysted tumour, consisting chiefly of cholesterin. See CYSTS.

CHOLESTERIN (χολή, bile; and στερεός, solid). The term *cholesterin* occasionally used is derived from χολή, bile; and στέαρ, suet).—SYNON.: Fr. *Cholestérine*; Ger. *Cholesterin*; Gallenfett.

CHEMICAL AND PHYSICAL CHARACTERS.—Cholesterin is a mon-atomic alcohol represented by the formula $C_{27}H_{45}OH$ ($C_{26}H_{43}OH$ is also used). It exists under more than one isomeric condition. When pure it occurs as white, tasteless, inodorous, glittering scales which form needles when crystallised out from solution in chloroform, and as rhombic plates when derived from alcoholic and ethereal solutions. These plates are often deficient at one corner, are not infrequently imbricated, and their angles measure $79^{\circ} 30'$ and $100^{\circ} 70'$. Cholesterin is insoluble in water, alkalis, and dilute acids, but readily dissolves in ether, chloroform, boiling alcohol, benzol, turpentine, and solutions of the bile-acids, and its solutions are lævo-rotatory. It melts at $145^{\circ} F$. The crystals heated with strong sulphuric acid give a carmine-red colour, or with strong nitric acid a yellow colour. When treated with iodine and sulphuric acid a play of colours, red, blue, green, is produced. When dissolved in chloroform and an equal bulk of sulphuric acid added, it gives a red colour changing to purple with green fluorescence of the subjacent acid. A more delicate test consists in dissolving in 2 c.c. of chloroform, adding ten drops of acetic anhydride, and then H_2SO_4 drop by drop, the resulting colours being red, blue, green.

Cholesterin is present in most protoplasmic structures, and is apparently an essential constituent of all animal and vegetable cells. By some it is regarded as 'a cleavage product constantly formed in the metabolic changes of the living cell.' But the fact that it is found under conditions so widely different as normal nerve-tissue, blood-corpuscles, egg-yolk, bile, sebum, and numerous morbid products, is of itself sufficient to indicate the difficulty of stating precisely the why and wherefore of its existence. It must play some important part in tissue-formation and maintenance; its presence in the bile must be regarded as excretory and possibly due to the elimination by the liver of the products of the metabolism of nervous tissue; under pathological conditions it arises from a degeneration of the cells of inflammatory and other morbid products.

PATHOLOGICAL RELATIONS.—The pathological occurrence of cholesterin is varied and of unknown import. It is liable to be much increased in quantity in the normal places of its occurrence, e.g. in some instances of jaundice it is present in the blood in marked excess. In the bile it may be so abundant as to form crystals or be deposited as gall-stones, of which it forms the greater portion and occasionally the whole; under these circumstances the bile-salts and acids must be diminished in quantity (or the amount of cholesterin much in excess), so that these fluids can no longer hold it in solution, and hence it is deposited on foreign particles or on pre-existing concretions.

In the fæces, when they are free from bile, a considerable increase of cholesterin and fats may be met with. In exceptional cases of renal disease (as in lipuria, chronic nephritis; hydatids and

stone in the kidney) crystals have been deposited in the urine. The fluid of cysts, especially hydatid and ovarian, seems to be more liable to contain cholesterol than effusions into serous cavities. But sometimes it occurs in considerable quantities in hydrocele fluids, and it has been met with in old pleural and peritoneal effusions. Its presence in atheroma is well known. Occasionally it forms definite tumours, sometimes of no inconsiderable size, e.g. in kidney, broad ligament, brain, and elsewhere. Crystals of cholesterol have been detected in the sputum of tuberculosis and other pulmonary excreta and exudations of inflammatory origin.

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R. G. HEBB.

CHOLINE.—See LECITHIN.

CHOLURIA.—The presence of bile in the urine. See URINE, Morbid Conditions of; and JAUNDICE.

CHONDRODYSTROPHIA FÆTALIS.—See ACHONDROPLASIA.

CHORDEE (χορδή, a harpstring).—DEFINITION.—Painful imperfect erection of the penis during gonorrhœa. It occurs in slighter intensity in gouty and septic urethritis.

ÆTIOLOGY AND PATHOLOGY.—Chordee is most common in the second and third weeks of gonorrhœa. It rarely attacks the patient after the third week unless the inflammation has been re-kindled by alcohol or venery. In rare cases, chordee, absent in the acute stage, is violently developed after the primary inflammation has subsided. Erotic indiscretion is usually the cause. The bulbous portion of the urethra is generally intensely inflamed when chordee happens. It is uncommon when urethritis is strictly limited to the anterior or posterior portions of the canal. It invariably occurs when marked penile œdema and lymphangitis are present. The mechanism of chordee is probably as follows:—Inflammation from the surface of the urethral mucous membrane spreads deeply owing to invasion of its multitudinous ducts by micro-organisms. Inflammatory effusion around the adjacent erectile tissue of the corpus spongiosum ensues. An impeded vascular path results. With erection, the corpora cavernosa, being unaffected, distend and elongate normally, whereas the impaired corpus spongiosum does not. This causes the penis to assume an arched shape, the corpus spongiosum being tense and bowed. The sensory nerve-endings supplying the inflamed mucous membrane are thus pressed upon. Another explanation is that the inflammatory condition of the mucous membrane and sub-mucous tissue at the bulbous part excites reflex spasm of the muscles surrounding that part, thus cutting off the vascular supply to the corpus spongiosum by compression.

ÆTIOLOGY.—The causes of chordee are *indirect* or *direct*. The most common indirect cause is urethritis, urethral congestion, and irritating injections. Direct causes are the reflex irritants which usually produce erection during sleep, such as stimulating food and drink, strongly acid urine, excessive bed-warmth, a distended bladder or rectal scybala.

SYMPTOMS.—The organ grows suddenly turgid and assumes a bowed or crooked form, causing acute pain, which is felt at the part and towards the perinæum. In severe cases the strain causes

rupture of the mucous membrane and spongy tissue, with hæmorrhage. The loss of blood is usually limited to a few drops and gives relief to the pain. Rarely the hæmorrhage is free and prolonged.

TREATMENT.—Abstinence from stimulants of all kinds and late suppers, light clothing, and a hard mattress at night, are the best means of preventing chordee. Micturition whenever the patient wakes during the night must be enjoined. The urine should be rendered bland and alkaline and the erectile tendency combated. A mixture containing full doses of citrate of potassium, tincture of hyoscyamus, liquid extract of *Salix nigra* in decoction of *Triticum repens* should be given every sixth hour. At bedtime a pill containing two grains of camphor and one-third of a grain of extract of belladonna should be administered.

A suppository containing one quarter of a grain of morphine is useful, or the hypodermic injection into the perinæum of one sixth of a grain of acetate of morphine may be given. Both these applications should be followed by an aperient saline draught next morning. A nocturnal dose of twenty to twenty-five grains of chloral hydrate in syrup and water, repeated in four or six hours if needed, is trustworthy. Bathing the genitals and perinæum with very hot water for ten minutes before going to bed sometimes proves successful. The application of a spiral coil of narrow india-rubber tubing round the penis and scrotum, through which a continuous current of ice-cold water flows, is an excellent preventive. If rectal scybala be suspected as the exciting cause, the bowel should be cleared out at bedtime by means of a simple cold-water enema.

To subdue an attack of chordee the best measures are voiding urine; the application of cold to the perinæum, by sitting on a cold seat, or applying an evaporating lotion, or ice, and the upright posture.

CAMPBELL WILLIAMS.

CHOREA (χορεία, a dance).—SYNON.: Chorea Minor; St. Vitus's Dance; Fr. *Danse de St.-Guy*; *Chorle*; Ger. *Veitstanz*.

DEFINITION.—A disease of the nervous system, characterised by a succession of irregular, clonic, involuntary movements of limited range, occurring in almost all parts of the body.

The distinctive features of the movements are the entire absence of either rhythm or method in their recurrence; that not individual muscles but co-ordinate groups are affected; and not one or more groups only, but almost all the muscles in turn. There is not actual loss of command over the muscles, but voluntary movements are interfered with by superaddition of involuntary movements. As a rule the movements cease during sleep. There is frequently also muscular paresis varying in degree.

ÆTIOLOGY.—Chorea is a disease of childhood: it is most common between the ages of eight and twelve, very rare before six, and rare after sixteen. It is more than twice as frequent in girls as in boys, especially after the age of nine. It occurs more frequently in families in which nervous diseases are hereditary than in others. It is more common in large towns than in the country; and far more frequent among the poor than among those in comfortable circumstances. Want of proper food, neglect, ill-usage, constipation, with the anæmia induced by these means, are very common antecedents, or chorea may follow measles or other febrile disease of childhood. Children well nourished and

with a good colour, exposed to none of these causes, may however suffer. An intimate association between chorea and rheumatism has long been recognised. A large proportion of the children suffering from chorea have had acute or subacute rheumatism, and many will be found subsequently to develop symptoms of rheumatism if their history be followed up. Some of the most terrible cases met with, especially after the age of puberty, are those in which the chorea comes on during or just after acute rheumatism and is complicated by pericarditis. There is very frequently found in chorea a cardiac murmur, usually due to mitral incompetence. This may be the result of cardiac dilatation, in which case it will disappear on recovery. More often it is due to a valvular lesion caused by rheumatic endocarditis, either active or of older date. In most fatal cases of chorea which have been examined after death, the results of endocarditis or of pericarditis have been present. In adults, pregnancy divides with rheumatism the causation of this affection; recovery generally speedily follows delivery, and can rarely be brought about till this has taken place. Chorea may develop at any period of pregnancy, but most frequently commences in the third month. Bad habits, disorders of menstruation, intestinal worms, or other reflex sources of irritation, have appeared to set up the disease, but most commonly fright or some powerful emotion is assigned as the cause; and it is seldom that parents are not prepared with the instance required. Though, on cross-examination, frequently the history of the source of the fright is indefinite, or its date too distant to admit its causal relation, yet so many well-authenticated cases are on record of an attack of chorea following on a severe fright within a few hours to a few days, that we must regard fright or emotion as an important ætiological factor. Imitation has been assigned as a cause, but it is doubtful if true chorea can ever be attributed to this.

MORBID ANATOMY AND PATHOLOGY.—The study of chorea, as of epilepsy and many other affections of the nervous system, has been hampered by its being regarded as a morbid unity. The view here maintained is that it is a symptom rather than a disease, and that the characteristic movements are in relation not with the *nature* of the morbid change, but with its *seat*. Chorea has been called 'insanity of the muscles'; a better phrase (as one of the writers has said elsewhere) is 'delirium of the sensori-motor ganglia.' In delirium there is loss of control over the mental processes, with rapid succession of incoherent and imperfect ideas; in chorea loss of control over the motor apparatus, with movements excessive in point of number and extent, but wanting in vigour and precision.

In some cases of chorea nothing abnormal has been detected after death, but usually the minute methods of investigation now pursued yield positive results. Dr. Dickinson, in a paper read before the Royal Medico-Chirurgical Society of London in 1875, described dilatations of the minute arteries as existing throughout the brain and cord, more especially, however, in the corpus striatum and optic thalamus, with small hæmorrhages; and considered the disease to be due to a widely spread hyperæmia of the nerve-centres. Capillary embolisms with hyperæmia have been found by Tuckwell, Dana, and other observers, predominantly in the central ganglia, but also in the central con-

vulsions and spinal cord, accompanied by patches of softening and minute hæmorrhages. Swelling and hyaline degeneration of the pyramidal cells of the cortex and of the cells of the central ganglia have been described by different writers. Elischer found peculiar concentric refractile bodies, which have been termed 'chorea corpuscles,' in the perivascular sheaths of the vessels of the corpus striatum and internal capsule, but they have also been found in persons who have never had chorea. Conflicting results have been obtained in bacteriological investigations.

The *post-mortem* appearances consequently are indefinite and do not indicate any localisation of the morbid change. But it is to be remembered that the fatal cases are those in which there is not only extreme violence in the choreic movements, but usually also delirium and other symptoms. There are in fact multiple symptoms, just as there are multiple lesions, and we are called upon to distribute the symptoms, and assign them to their respective sources, by such knowledge of the functions of the different nerve-centres as physiology affords us.

The seat of the disease.—There is conclusive evidence that the seat of the disease is in the brain and not in the spinal cord. The arguments in favour of this are: (1) that *tonic* and not *clonic* spasm is characteristic of persistent spinal irritation; (2) the degree of control over the movements retained by the will; (3) their increase under emotion; (4) their cessation during sleep; (5) diminished reflex action; (6) the phenomena of hemichorea and its relations with hemiplegia. The evidence afforded by hemichorea is so conclusive that other considerations are of minor importance. The parallelism between hemichorea and hemiplegia is so perfect as to suggest at once that the two affections represent different conditions of the same nerve-centres, and is made more complete by the very discrepancies, as they may at first sight appear, which have been considered to be objections. In hemiplegia there are certain muscles which more or less completely escape paralysis: the *motores oculorum*, *orbiculares palpebrarum*, and other facial muscles, the muscles of the neck, chest, back, and abdomen. In hemichorea the irregular movements cross the median line and invade the opposite side in these same muscles. This has been explained by the hypothesis that since all these muscles act in compulsory concert with the corresponding muscles of the opposite side, the nerve-nuclei of the bilaterally associated muscles will be commissurally associated in the cord, so as to become in effect a single nucleus. This single nucleus for muscles on each side of the body, being connected with both hemispheres, is thrown into action by the sound hemisphere when its fellow of the opposite side is damaged, as in hemiplegia, thus preventing paralysis; and, on the other hand, is reached by the irregular impulses from the side of the brain affected in hemichorea, thus causing bilateral chorea in the parts enumerated.

In addition to the correspondence between hemichorea and hemiplegia just described, there are transitions from one to the other, and combinations of the two to be mentioned below. Hemiplegia may be succeeded by hemichorea (the *post-hemiplegic chorea* of various observers), although the movements are not strictly analogous; or chorea may be followed by paresis; or, as in a case reported by one of the writers, there may be with chorea of the

limbs on one side, first chorea, then paralysis (hemiplegiform), and then again chorea of the same side of the face.

The explanation of the phenomenon of post-hemiplegic chorea generally given is, that it is due to irritation of the motor fibres in the internal capsule by the lesion which originally caused the hemiplegia. But the most common site of the lesion in such cases is in or adjoining the optic thalamus, implicating, it may be, the sensory fibres in the posterior third of the internal capsule or the caudate and lenticular nuclei as well. In tumours also of the optic thalamus there are frequently choreic or ataxic movements of the limbs on the opposite side of the body. It is conceivable therefore that in both instances these irregular movements may be a reflex response to irritation of the cells in the optic thalamus, and that chorea may have an analogous origin. The character of the movements in the two diseases is, however, very dissimilar, and though it is a tempting theory to regard the optic thalamus and corpus striatum as a compound sensori-motor nucleus for complex co-ordinated movements and as the seat of the lesion in chorea, this hypothesis cannot be maintained in the present state of our knowledge of the functions of the basal ganglia.

Much is to be said in favour of the view of Sir William Gowers that the seat of the disease is in the motor cells of the cortex. Impairment of nutrition and functional disorder of these cells might produce the irregular movements, and a further degree of impairment of their functional activity, the paresis of chorea. The unilateral distribution of chorea which is common, and its subsequent extension to the other side, has its counterpart in epilepsy which is attributed to the spread of discharge from the motor cortex on one side to that on the other. The mental changes also that may occur in chorea point to impairment of function of the cortex.

The balance of evidence therefore is in favour of the seat of the disease being in the cerebral cortex, though in the absence of any definite characteristic lesion all views as to the pathogeny of chorea must be purely speculative.

The question which now remains to be considered is the cause of the derangement of function of the nerve-cells, affected in chorea. The frequent association of endocarditis with chorea in fatal cases gave rise to the well-known embolic theory advocated by Kirkes and ably maintained by Hughlings Jackson, that chorea is due to shreds of fibrin dislodged from the affected valves and obstructing capillaries in the brain. But the fact that in a large proportion of cases of chorea which recover there is no evidence at any time of endocarditis, and that in some fatal cases none is found *post mortem*, prove that this, if it be a cause of chorea, cannot be the only one.

So intimate is the connection between chorea and rheumatism that for the most part we must look on chorea as a manifestation of the rheumatic diathesis. That cases occur in which no history or evidence of rheumatism can be obtained is indisputable, but not infrequently articular or cardiac manifestations of rheumatism declare themselves at a subsequent period, and careful observation during an attack of chorea will often detect some dilatation of the heart, and it may be a soft mitral murmur which suggest inflammation of the myocardium of rheumatic origin. That chorea is a cerebral manifestation of rheu-

matism analogous to the articular and cardiac varieties is a plausible theory, and as the microbic origin of rheumatism is now conclusively established, it is possible that chorea is due to irritation of the motor cells in the cortex of the brain, by the organisms or their toxins.

Dr. Sturges ably maintained the view that chorea is a purely functional affection, and compared it with epilepsy. In favour of this view are the facts that an attack of chorea may be induced by fright or some emotion, or pregnancy, and that the disease is one usually limited to the period of childhood or early adolescence, a time when the nerve-cells are in an unstable and susceptible condition.

SYMPTOMS.—The disease usually begins with slight twitching of the face or spasmodic jerky movements of the hand. In a slight case the patient, usually a child, may be perfectly quiet when lying down, and for a short time even when sitting or standing, if not conscious of being under observation; but when walking, or while under examination, there will be various fidgety actions—abrupt flexion of the fingers, a sudden pronation of the forearm, or hitching up of one shoulder, or twist of the body, or there is shuffling of a foot on the floor, or, again, a jerk of the head or twitch of the mouth or eyes. If the patient be told to do anything, the movements will be multiplied and exaggerated in the muscles employed, and there is marked inco-ordination or voluntary movement. A small object will be picked up with difficulty, the hand being brought down upon it hastily and after various irregular excursions, and it may be dropped involuntarily. Sometimes, in the early stages, a child is punished for thus dropping an object and breaking it, or for fidgeting, before the nature of the disease is recognised. In a more severe case the grimaces, contortions, and jerks succeed each other without intermission. The gait is now very peculiar, being slow, shuffling, and uneven; the steps of irregular length and unequal time; and the line of progress deviating. In the worst forms of this disease every muscle appears to be thrown in turn into violent contraction, the face is distorted this way and that, the eyes roll to and fro, the teeth are snapped or ground together, the whole body writhes, and the limbs are in unceasing motion. It is to be remarked that, even in extreme cases, the movements, violent as they may be, are in some degree circumscribed; the arms, for example, are not thrown up over the head, nor do the legs go to the full extent of their range of motion; the tongue is rarely bitten, though the lips may be. Deglutition is greatly interfered with in a severe attack, and the evacuations may be discharged involuntarily. In the mildest forms the diaphragm and muscles of the chest and abdomen are affected, causing irregularity in respiration. The action of the heart may also be irregular, but this is probably secondary to the respiratory variations in frequency and depth, and is not attributable to chorea of the heart. Speech is very commonly more or less affected and occasionally completely lost for a time. The difficulty is usually articulatory, chorea of the muscles of respiration, phonation, and articulation interfering with utterance of words; but there is in some cases true aphasia, and when this is so, there is the same tendency to the association of aphasia with right hemichorea as with right hemiplegia. The intellect may suffer; the face has often an idiotic expression, usually

from the muscular contortion or atony, but sometimes indicative of temporary imbecility. In the violent and fatal forms there is almost always delirium. There is generally impairment of motor power: at times this amounts to complete paralysis, and the relations and combinations of chorea and paralysis, and especially of hemichorea and hemiplegia, throw much light on the disease. Paralytic chorea has been described as a special variety of the affection, but no line of demarcation exists between this and the common form. There may undoubtedly be impairment of sensation, though this is disputed by some observers. It is difficult to ascertain in all cases, but is most readily detected in hemichorea when the sound side can be employed for comparison; but in the violent forms of the disease, when the skin is gradually worn through by incessant friction, there is often so little complaint of pain that sensibility must, it would seem, be blunted. Reflex sensibility is also commonly dull.

It has already been stated that the movements cease during sleep; this is a rule to which exceptions are rare though not unknown, even in mild cases, and especially in hemichorea.

Chorea is usually gradual in access, even in the cases which ultimately become severe; it is very commonly one-sided for a time, and occasionally throughout, when the name hemichorea is given. It is not, however, strictly unilateral in these cases, as the movements transgress the median line and affect the corresponding muscles of both sides of the body at those parts where these are bilaterally associated, and where in hemiplegia there is immunity from the paralysis, as, for instance, the oculo-motor muscles, and the muscles of the neck, chest, and abdomen.

DURATION, TERMINATIONS, AND PROGNOSIS.—The average duration of chorea is about two months; if prolonged beyond three months it may be exceedingly chronic and go on, better and worse, for one or two years. There is a tendency to spontaneous recovery; but on the other hand relapses are common. Chorea is rarely fatal in children; when it is so, the case is usually acute and violent from a very early period of the attack, and is complicated by pericarditis or endocarditis: it is rare for a case to run the usual course for a time and then take on a very severe character. After puberty, and especially when it supervenes on acute rheumatism, it is often dangerous, and is usually serious when associated with pregnancy. See *INSANITY IN SPECIAL DISEASES*.

DIAGNOSIS.—It is scarcely necessary under this head to warn against the mistake of confounding with the movements of chorea the tremor or jactitation of disseminated sclerosis, which, though most common in adults, is not unknown in childhood. From 'spasmodic tic' chorea may be diagnosed by the limitation of the spasm to certain groups of muscles and by the sudden spasmodic character of the movements in the former disease. The rigidity and partial wasting of the limbs affected, with the rhythmical character of the movements and the history of the case in athetosis or post-hemiplegic chorea, will readily distinguish this affection from true chorea.

The diagnosis of paralytic chorea may be sometimes a matter of considerable difficulty, especially where the paresis is hemiplegic in distribution and no choreic movements are present. The paralysis is, however, never so complete as in hemiplegia due to organic disease, and frequently twitching

movements of the face are present when absent in the limbs. The onset of the paresis is usually gradual, and careful observation will after a time almost invariably detect twitching movements of the limbs or face when these are apparently absent at first.

TREATMENT.—In a large proportion of cases of chorea, especially such as come into the hospitals of London, rest and food, with perhaps aperients, are all that are required for recovery. But it can scarcely be denied that medicinal treatment often renders important services, especially in cases of a lingering character. The causation and pathological condition being various, it is to be expected that the remedies required will be different, and the attempt should be made to adapt the treatment to the special features of the case, the basis of all being the endeavour to improve the nutrition of the body generally, and of the nervous system, by good food, rest, and warmth. The food may be supplemented by cod-liver oil, and stimulants may be necessary in severe cases. Any recognised cause of reflex irritation should be removed, such as constipation or worms; irregularities or suspension of the catamenia should receive attention; when there is pregnancy it may perhaps be necessary to induce premature labour. Iron in some form is very generally useful, especially when the patient is anæmic. The drug that has been found most useful is arsenic, given in doses of three minims of the liquor arsenicalis three times a day, and gradually increased up to ten or even fifteen minim doses. Care must be taken not to continue it for too long, as neuritis or other symptoms of chronic arsenical poisoning may be induced. Trousseau sometimes gave strychnine in gradually increasing doses till its physiological effects manifested themselves. On the other hand, conium, recommended by Dr. John Harley, has been extensively employed; the only trustworthy preparation is the juice, which should be given in gradually increasing doses, beginning with a drachm and going up to one or two ounces if necessary, till its depressing effect on the muscles becomes evident. It has not, in the writers' hands, given satisfactory results. It is doubtful if any drugs are of great service, and the same may be said of the application of ether-spray along the spine. Baths, warm and cold, especially shower-baths, spinal douches, spinal ice-bags, and especially wet packs, may be useful in inducing sleep in severe cases.

In the terrible cases of acute chorea the great indications are to procure rest for the sufferer and keep up the strength. Milk, eggs, beef-tea, and other forms of concentrated fluid nourishment should be given freely, together with wine or brandy. Conium, hyoscyamus, cannabis indica, bromide of potassium or ammonium, and chloral hydrate, have been tried separately or in combination as sedatives, with more or less appearance of success, and chloroform has been administered. Chloral hydrate by the mouth or rectum, and hypodermic injection of morphine, with free administration of brandy, have, in the writer's judgment, appeared to be of most service as sedatives where the movements are very violent. It is in these cases that tartar emetic in full doses has been recommended; it is certainly tolerated in an astonishing degree. Restraint of the violent movements is often a great comfort to the patient: the limbs should be carefully bandaged with flannel and bound—the legs together, the arms to the sides, a folded blanket being placed across

the abdomen and hips, to keep down the body. If half-done it only adds to the suffering, but when properly carried out it gives a feeling of relief and favours sleep.

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CHOREA, Electric.—**SYNON.** : Dubini's Disease.

The term 'Electric chorea' has been applied to a somewhat rare disease, met with in Northern Italy, which was first described by Dubini, an Italian physician. It is not related to true chorea, to which it appears to bear but a superficial resemblance. It is characterised by sudden spasmodic jerky movements of the extremities, the muscular contractions resembling those produced by electrical stimulation. Wasting of the muscles with paralysis of the limbs ensues and the disease usually proves fatal in a few months or, it may be, weeks.

It may occur at any age, and the onset is attended with pyrexia of varying degree and with pains in the back. The characteristic spasmodic movements are at first usually limited to one upper extremity, but gradually increase in intensity and spread to the leg on the same side, and then over the rest of the body. The limbs first affected then become feeble, the muscles waste and do not respond to faradism, and absolute paralysis succeeds, which may become general. Epileptiform seizures may occur, which sometimes prove fatal at an early period, or death may be postponed for several months.

The aetiology and pathology of the disease are at present unknown, but the clinical features suggest some acute infection or toxæmia which gives rise to organic change in the nerve-centres, apparently in the motor cells of the brain and spinal cord.

All treatment has hitherto proved unsuccessful.

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CHOREA, Hereditary Progressive.—

SYNON. : Huntington's chorea.

This disease was first described by Dr. Waters, an American physician of Long Island, in 1842. In 1863 Dr. Lyon read a paper on the subject, and in 1872 Huntington gave a comprehensive description of the disease, which is therefore generally named after him.

The distinguishing features of the malady are (1) that it is hereditary; (2) that it is a disease of adult life, usually developing between the ages of thirty and forty, rarely before the age of twenty-five; (3) that it is progressive and incurable; (4) that it is accompanied by mental deterioration usually terminating in a condition of dementia.

ÆTIOLOGY.—The only known cause of this affection is its hereditary transmission from one generation to another. It is always hereditary, and frequently several members of the same family are affected. It is not associated with rheumatism, and no special exciting cause is known. Males and females are equally liable to the disease, and it does not occur till after puberty.

MORBID ANATOMY.—Thickening of the dura and pia mater, degenerative changes in the pyramidal cells of the cortex, atrophy of the cerebral convolutions, chronic diffuse encephalitis have been found by different observers, but there appears to be no characteristic or constant lesion.

SYMPTOMS.—The symptoms are both motor and mental. The former are the first to appear, and

consist of twitching or jerking movements of the face or limbs, resembling those of ordinary chorea, or rather, according to Dana, those of spasmodic tic. The movements are slight at the outset and can be controlled during voluntary action, but later on they become more violent and extensive, and quite beyond control. Every part of the body may be affected, so that speech and deglutition become difficult. The movements cease during sleep. Locomotion is peculiar when the disease is advanced: the patient sways from side to side, and walks with a high-stepping gait and jerky action of the feet.

The mental symptoms develop with the motor, usually commencing with gradual loss of memory and depression. There may be attacks of excitement or irritability, but the patient is usually apathetic and depressed, gradually passing into a state of dementia.

The average duration of the disease is ten to fifteen years, but some patients live for twenty or even thirty years from the onset of the disease.

No treatment is known which is able to arrest the course of the malady.

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CHOROIDITIS.—Inflammation of the choroid. *See* EYE, AND ITS APPENDAGES, Diseases of.

CHROMIDROSIS (χρῶμα, colour; and ἰδρῶς, sweat).—Coloured perspiration. *See* SUDORIPAROUS GLANDS, Diseases of.

CHRONIC (χρόνος, time).—This word is applied to a disease when its progress is slow, and its duration prolonged.

CHVOSTEK'S SYMPTOM.—The term is applied to the increased mechanical excitability of the motor nerves and muscles of the affected parts, occurring in tetany. *See* TETANY.

CHYLOCELE.—The presence of milky fluid in the tunica vaginalis. *See* FILARIASIS.

CHYLURIA (χυλός, chyle; and ὄρον, urine).—**SYNON.** : Galacturia; Chylous urine; Fr. *Urine laiteuse*; Ger. *Chylurie*; *Milchsaftiger Harnabgang*. This term is applied to a state of the urine in which it presents the appearance of milk. The condition is caused by the admixture of chyle with the urine, and is generally due to the presence of the adult *Filaria sanguinis hominis* in the body. *See* FILARIASIS.

CICATRISATION.—*See* WOUNDS, Healing of.

CINCHONISM.—A condition induced by the administration of quinine, the chief active principle of cinchona. *See* QUINISM.

CIRCULAR INSANITY.—*See* FOLIE CIRCULAIRE.

CIRCULATION, Disorders of.—Abnormal excess and deficiency of blood are known as *hyperæmia* and *anæmia* respectively. Each of these may be *general* or *local*. We also recognise as a disorder of circulation stagnation or *local cyanosis*.

I. Hyperæmia.—*General* hyperæmia signifies excess of blood in the body, and is also called *plethora*. *Local* hyperæmia means excess of blood in a part. Such excess may be caused either by

superabundant supply, or by deficient removal of blood through the agency of the blood-vessels. Dilatation of the arteries, however produced, causes more copious afflux of blood, which fills the capillaries and veins in a corresponding degree, so that there is excess of blood in all the vessels of the part. This condition is called *arterial* or *active hyperæmia*, *active congestion*, or *determination of blood*. If, on the other hand, blood is imperfectly removed by the veins, these vessels, as well as the capillaries, become engorged, and the condition called *venous* or *passive hyperæmia*, or *venous congestion*, results. It is doubtful whether there can be *capillary hyperæmia*, except as the result of one of these conditions.

A. *Arterial or Active Hyperæmia*.—An excessive amount of blood can be conveyed by the arteries only under two conditions:—(1) Enlargement of these vessels by relaxation of their muscular walls; (2) Increased pressure within them, from obstruction of collateral channels with which they communicate, i.e. collateral hyperæmia.

(1) Relaxation of the muscular walls may be caused *directly* by violence or by warmth, as is illustrated in the redness of the skin produced by a blow, by friction, by heat, or by the reaction after intense cold. Sudden withdrawal of pressure has the same effect, as is sometimes seen on evacuating a hydrocele or fluid-collection in a serous cavity. Dry-cupping produces similar but more complex results, the veins being acted upon as much as the arteries. Relaxation of the muscle-fibres is produced also *indirectly* through the vaso-motor nerves. If these are paralysed, relaxation of the fibres occurs, and the arteries dilate. Experimental section of the cervical sympathetic in animals shows this most clearly; but the same result follows less constantly if other nerves containing vaso-motor fibres are divided or injured, such as the mixed nerves of the limbs, or branches of the trigeminus. Wounds of the brachial plexus have been found to cause hyperæmia of the fingers (*glossy fingers* of Paget). When the section is complete, hyperæmia is only transitory, and is soon followed by a return to the normal condition, or even by undue anæmia, which is permanent. In irritative lesions, on the other hand, such as gunshot wounds, hyperæmia continues as long as the irritation. In such cases it is possible that the lesion is not paralytic, but depends upon stimulation of the actively dilating vaso-motor fibres which physiologists have now shown to exist in many parts of the body, since stimulation of these produces the same result as paralysis of the inhibitory fibres. The hyperæmia of surfaces produced by friction or slight irritation has also been explained as a reflex action dependent on a stimulus conveyed by the sensory nerves to the medulla oblongata, and a resulting efferent current passing through the vaso-motor nerves to the peripheral vessels. But the same result may be produced when communication with the nerve-centres is entirely cut off. For instance, in a lesion of the fifth nerve causing complete anæsthesia in its cutaneous distribution, hyperæmia of the conjunctiva or skin may be produced by gentle friction. The action is therefore either a direct one on the vessels, or it is a reflex from sympathetic ganglia in the walls of the arteries. Hyperæmia often accompanies neuralgia, both depending upon some morbid condition of the nerve. The starting-point of the neurosis in all these cases may be, and often is, in

the central nervous system, and hence chronic diseases of the spinal cord or brain are often accompanied by general paralytic hyperæmia—that is, flushing, or by congestion of special parts. The same result may come from reflex nervous action, set up by disturbances of the digestive organs, the organs of generation, or of other parts.

(2) Collateral hyperæmia is a consequence of the rise of pressure produced by the blocking-up of arterial channels in the adjoining parts. It is usually effected by the enlargement of existing vessels. It occurs not only in the familiar instances of surgical ligature, and the sudden blocking of an artery by a plug (*see EMBOLISM*), but in the gradual obstruction which accompanies atrophic and sclerotic processes. When the chief efferent blood-channels to an organ become obstructed, its peripheral parts are very liable to become hyperæmic, as is seen in such cases as cirrhosis of the liver, granular kidney, and sclerosis of the brain.

SIGNS AND RESULTS.—The colour of parts in a state of active hyperæmia is, during life, bright red, the arteries, large and small, being visibly injected, while the capillaries, filled with arterial blood, produce a diffuse red colour. In experimental hyperæmia the blood may remain bright red even in the veins. The temperature of external parts becomes elevated, though not above that of internal parts. Sometimes there is obvious pulsation or throbbing. There may be swelling, which is due to simple enlargement of the vessels, not to exudation of fluid, since this does not occur from arterial hyperæmia alone. The nerves, both those of common sensation and those of special sense, are more excitable than they are normally. There is usually a subjective sensation of warmth, and there may be pain or itching.

Arterial hyperæmia may last for a long time without producing any change whatever in the part affected, but may, under conditions little understood, give rise to hypertrophy, which sometimes, though rarely, results from section of the cervical sympathetic. Transitory but repeated hyperæmic conditions more regularly produce this result, as in thickening of the skull from excessive exposure of the head to the sun; in hypertrophy of the skin of the face and its glands from frequently recurring hyperæmia (*acne rosacea*). This kind of hyperæmia constantly precedes inflammation, but can hardly be said to produce it. It does, however, render the tissues more vulnerable, bringing them into a condition in which a slight cause will set up inflammation. Unless the vessels are unsound, simple arterial hyperæmia does not lead to hemorrhage.

B. *Passive Hyperæmia or Venous Congestion*.—This may be due to (1) Feeble circulation; or (2) Obstruction in the veins.

(1) Blood may be imperfectly removed from a part, owing to the imperfect action of the forces which normally maintain the flow of blood in the veins. These are, besides the action of the heart, the pressure of muscles (combined with the arrangement of the valves in the veins), and the movement of the thorax in inspiration. If these are deficient, the venous current will be everywhere delayed, but notably in those parts where it has to overcome the action of gravity. In the erect posture this will be the case in the lower limbs; and hence venous congestion is common in the legs, ankles, and feet. In decumbent patients, for analogous reasons, the nates, sacrum, shoulder-blades, and the bases of the lungs

behind become the seat of what is called *hypostatic congestion*. Very general obstruction, such as results from imperfection of the heart itself, may lead to the condition called *cyanosis*, which is essentially venous congestion, and to similar congestion of the lungs, liver, kidneys, and other internal organs, with very serious results. See DROPSY.

(2) Obstruction in the veins may result from coagulation of blood within them (see THROMBOSIS). Another cause is external pressure, such as that of tumours, of the gravid uterus, or of the intestinal contents, as in the case of the hæmorrhoidal veins. Finally, indurative changes in the solid viscera lead to venous obstruction, as is seen in cirrhosis of the liver, which produces congestion of the whole portal system.

SIGNS AND RESULTS.—The colour of parts in a state of passive hyperæmia is bluish, the veins, large and small, being injected with venous blood, and the capillaries, in which the blood is also venous, producing a uniform purple colour. If the congestion is extreme, collateral venous channels are likely to be established, which are sometimes the only evident sign of internal venous obstruction. The surface is cooler than normal. Swelling very frequently occurs, and depends on actual serous effusion from the vessels. See DROPSY.

Venous congestion produces more important and permanent results than arterial. In experimental venous obstruction, besides engorgement of the vessels, two nearly constant phenomena are seen—copious transudation of serum, and passage of a number of red blood-discs through the walls of the capillaries and smaller veins. Few or no white corpuscles emigrate, and the arterial circulation is unaltered. In ordinary pathological venous congestion all these changes are seen to some extent; extravasation of red blood-discs being shown by the pigmentation of parts in chronic congestion, though this is not evident in the acute condition. Chronic venous congestion increases the hardness and density of organs (see FIBROSIS). Such organs are at first enlarged, but ultimately diminish in size, and undergo fatty degeneration and atrophy, because venous blood is inadequate to the proper nutrition of tissues. These changes are seen in the liver and kidneys in cases of obstructive heart-disease. External parts, as the skin of the lower part of the leg, show by a tendency to ulceration that they are imperfectly nourished, and are also liable to become inflamed from slight causes (varicose eczema).

POST-MORTEM CHARACTERS.—The colour of the blood is of little value, since all blood becomes florid when exposed to the air, unless it have previously undergone some *post-mortem* change, or some morbid alteration during life. The only important point is the fulness of the three kinds of vessels. Arteries are usually empty, unless diseased: the larger veins almost always full. If the smaller veins and arteries are conspicuously and brightly injected, the part may be described simply as congested; a uniform colour indicates fulness of the capillaries, which may be confirmed by the microscope. Uncomplicated arterial hyperæmia leaves no trace after death; the appearance of it is produced by inflammation. Simple venous hyperæmia can only be recognised as such after death by comparison with the same part under normal conditions. Chronic venous congestion is indicated by many of the same characters as during life.

Care should be taken not to mistake for arterial hyperæmia mere staining with blood-pigment of the walls of the vessels; nor for venous congestion mere *post-mortem* hypostasis, or the settling down of the blood, if fluid, after death.

II. Anæmia.—*General* anæmia is a morbid condition in which there is a deficiency of blood, or, more correctly, of pigment and red corpuscles in the blood, throughout the whole body (see ANÆMIA). *Local* anæmia, with which we are here concerned, signifies deficiency of blood in a part. It may be complete or partial. Complete local anæmia can only occur when the blood-supply of a part is totally cut off by obstruction of its arteries. The conditions and consequences of such obstruction are discussed elsewhere (see EMBOLISM). Partial anæmia or *ischæmia* may be produced by direct pressure, or else by arterial obstruction, permanent or transitory. Permanent anæmia of many parts results from gradual obstruction of arteries by atheromatous change; or, still more strikingly, by a form of endarteritis (*endarteritis obliterans*) generally due to syphilis; or, again, from deposition of fibrin on the diseased vascular wall. Temporary anæmia results from spasmodic contraction of the annular fibre-cells in the muscular coat of the artery. Such a contraction may be produced experimentally by direct electrical stimulation, or by stimulation of the sympathetic branches distributed to the vessel; and in pathological conditions we find such contraction occurring in consequence of some derangement of the nerve-centres, or from reflex irritation, or from unexplained causes. Neuralgia and migram are often accompanied or caused by spasm of the arteries, and epilepsy has, with less probability, been attributed to the same cause. In these cases it is possible, as is held by some, that anæmia of the nerve-tissue is the cause of the disturbed innervation. Hysterical blindness, and possibly other hysterical affections, may be explained in the same way. Partial anæmia may, as shown elsewhere, lead to local cyanosis, with the appearance of venous hyperæmia. Total anæmia necessarily causes necrosis of the part; and even partial anæmia produces characteristic changes.

SIGNS AND RESULTS.—An anæmic part is pale, its temperature in the case of external parts is diminished, and there is weakened or arrested arterial pulsation. A permanent condition of partial anæmia produces degeneration, ending in atrophy of the affected part. The wasting of the skin, and possibly that of the kidneys in old age, is due to this cause. Transitory anæmia causes necessarily a cessation of functional activity in the part, as is obvious in the nerve-centres and the muscles; but does not, so far as we know, produce any permanent change. Compression or obstruction of the abdominal aorta produces symptoms of temporary paraplegia, from anæmia of the lower part of the spinal cord.

III. Local Cyanosis.—It must not be supposed that a venous condition of the blood in a part, with consequent purple colour, low temperature, and deficient vitality, is necessarily due to venous engorgement. It may be due, as is obvious in external parts, to mere stagnation in the capillaries. When blood is stagnant, or nearly so, it becomes venous, having given up its oxygen to the tissues; and the part shows the characters of venous congestion. This is seen in the familiar instance of the effects of external cold on the extremities. A

similar condition, which may be called *local cyanosis*, sometimes occurs independently of, or only assisted by, the action of cold, in certain parts, such as the extremities of the fingers and toes, the tips of the ears and the nose. It is possible that similar conditions of internal parts may occur. A part affected with local cyanosis may undergo partial necrosis or inflammation, as in the case of frost-bite or chilblain.

Some, mostly young, persons have a constitutional proclivity to this condition, especially in cold weather, and have been described as having a 'chilblain-circulation;' but it is not necessarily associated with chilblains, properly so called. In other cases the cyanosis is preceded by, or alternates with, a condition of anæmia in the part, and is evidently due to a functional disturbance of circulation, more or less transient. In its extreme form, when occurring suddenly, and affecting deeper parts as well as the skin, and leading to necrosis, this constitutes Raynaud's disease. The condition is nearly always symmetrical, affecting corresponding parts on both sides of the body.

ÆTIOLOGY.—The constitutional or habitual form of local cyanosis is generally associated with malnutrition and sluggish circulation; but it does not appear that mere weakness of the heart is sufficient to produce it, or is more than a contributory factor. The fault of circulation appears to lie in the arteries, and to consist either in habitual spasm, or want of tone. Imperfect nutrition of the walls of the capillaries may also, by retarding the flow, assist in producing stagnation. In the more transient forms there would seem to be spasm of the smaller arteries, causing anæmia, on which capillary stagnation supervenes. The slighter forms occur chiefly in women, accompanying hysteria or menstrual disturbances, or at the climacteric period. Such persons present an alternation of 'dead fingers' with cyanotic circulation. The severe form known as Raynaud's disease is described elsewhere. In both these forms there must be some antecedent derangement of the vaso-motor nerves, dependent probably upon deeper-lying disturbances of the central nervous system, which cannot here be discussed.

CONSEQUENCES.—The slighter form may cause no material change, or may lead to the same series of changes as seen in chilblains—namely, necrosis of epidermis, with formation of imperfect bullæ, and inflammation, ending possibly in ulceration. It is difficult to draw the line between these changes and those produced in healthy persons by frost-bite or by Raynaud's disease.

TREATMENT.—Young persons, with a tendency to local cyanosis, should be well fed, warmly clothed, and encouraged to take vigorous exercise for short periods. Cod-liver oil is the most useful drug; but iron or arsenic may also have their place. When the condition is paroxysmal, rather than constant, large doses of sulphate of quinine (5 gr. twice or thrice daily) are sometimes extremely useful. In the spasmodic anæmia, followed by cyanosis, of hysterical women, or at the climacteric period, arsenic has appeared to the writer to be by far the most useful medicine. See RAYNAUD'S DISEASE.

J. F. PAYNE.

CIRRHOSIS (κίρρῶς, yellow).—SYNON.: Fr. *Cirrhose*; Ger. *Cirrhose*.

The term 'cirrhosis,' originally invented to describe a particular state of the liver, gradually acquired a

more extensive meaning, and was applied to processes resulting in an increase in the fibrous connective-tissue of other organs, though the name itself, derived from the yellow colour of the liver in this disease, was not properly applicable. In later years the term *fibrosis* has been introduced to designate the increase in the fibrous connective-tissue of any organ, while that of *cirrhosis* is restricted to its original use. See FIBROSIS.

CIRRHOSIS OF LIVER, LUNG, &c.—See LIVER, LUNG, &c., Diseases of.

CIVIL INCAPACITY.—A chief cause of civil incapacity is mental weakness or disease, and it is one of the duties of the physician to aid in determining the existence and nature of such conditions. There is a kind of incapacity which is implied in the restriction of a person's liberty when he is placed under care in an asylum or other special place for treatment. The necessary information regarding this will be found in the article, LUNACY, Laws of. But the question of incapacity is more directly raised when it is proposed that a person should be declared unfit to exercise his civil rights, to require the shield of the law to prevent his being imposed on, and to obtain special protection for his property. Medical evidence must be taken if it becomes necessary for a Commission of Lunacy to be issued by order of the Lord Chancellor. This is a proceeding which ought not to be adopted if it can properly be avoided. But until a person has been found lunatic by inquisition he is, though placed in an asylum under regular certificates, not debarred from exercising his rights in the disposal of his property. The acts of any person either in or out of an asylum may, however, be declared invalid if it can be shown that at the time they were performed the person laboured under such form of insanity as rendered him incapable of performing them rationally and without harmful consequences. On this principle any person may be found to have been incapable of contracting marriage, of executing a deed, contracting a debt, making a will, or giving credible evidence. The principle, it must be carefully noted, is not that the mere existence of insanity in the person performing them invalidates such actions, but that if the insanity has materially affected the character and quality of the actions they may be thereby invalidated. This is one of the most important principles that a medical jurist has to keep in mind, as it is not an infrequent mistake to suppose that a person is necessarily incapacitated from the performance of every civil act the moment he can be proved to labour under any condition to which the term insanity may be applied. Perhaps the case in which the validity of a civil act is most easily endangered by the existence of any form of insanity is the contract of marriage. This proceeding is supposed to so affect the whole relations of life, that almost any form of unsoundness of mind may be sufficient to interfere with that intelligent and deliberate consideration which is essential to the giving of rational consent.

The different kinds of mental disease will be found described elsewhere (see INSANITY), and it is necessary that the practitioner, when dealing with medico-legal questions, should be fully acquainted with them. But it is chiefly important that he should distinguish the two following classes: (1) diseased perversion of the mental faculties; and (2) weakness or enfeeblement of these faculties, re-

sulting either from defective development, disease, or decay. The first class includes all kinds of insanity which are the result of active disease. These would include the simple forms of delirium, mania, melancholia, and monomania; as well as the similar primary conditions which are found in general paralysis, and other diseases which present maniacal, melancholic, or monomaniacal symptoms. It is in this class that the special knowledge of the physician can be most successfully applied in aiding the administration of justice. In order to establish the incapacity of a person said to labour under any of these forms of disease, it is necessary that an experienced physician should not only be able to detect their characteristic symptoms, but also to show that the performance of the duties or the exercise of the rights under consideration would be modified or obstructed by the existence of such disease. The second class includes congenital imbecility, and all the forms of what is called chronic dementia—all those enfeeblements of mind which are sometimes the remaining effects of acute disease, sometimes the concomitants of chronic disease, and sometimes only the mental phase of senile decay. Here, again, the information which may be communicated by the physician must be of great importance. But in estimating the extent to which a condition of mere mental weakness will disable a person from the performance of a certain class of actions, so much special medical knowledge is not required as is necessary in the consideration of more active disease.

Marriage.—As has been already stated, the mere existence of any form of insanity in one of the parties may render a contract of marriage void. In one case which terminated in this manner, a man who had been insane, and when in that state had voluntarily contracted marriage, instituted the suit himself.

Civil Contracts.—These may be held binding although made by lunatics. If the person with whom a contract is made had no knowledge that the person contracting was insane, and if no attempt was made to take undue advantage of the latter, the contract would be held good.

Wills.—A person is considered to be of a disposing mind—that is, capable of making a valid will—if he knows the nature of the act which he is performing, and is fully aware of its consequences. It is in regard to the making of wills that the law has carried out most thoroughly the principle, that the validity of an act ought to be maintained in cases of insanity, unless at the time the act is performed the state of mind of the agent can be shown to render him unfit to perform that particular act in a rational manner. Persons have made valid wills while inmates of lunatic asylums. One will was held to be good though the testator had committed suicide within three days after its execution. The existence of delusion which has been regarded by lawyers as of such importance in cases of alleged insanity does not invalidate a will; for it has been declared to be ‘compatible with the retention of the general powers of the faculties of the mind,’ and to be ‘insufficient to overthrow the will, unless it was calculated to influence the testator in making it.’ On the other hand, a will may be invalidated on account of the existence of mental states which would not be regarded as insanity from either a legal or medical point of view. Drowsiness and stupor resulting from erysipelas or fever, extreme weakness from cholera or other disease, and failure of memory in old age, have been sufficient to render wills void. It fre-

quently happens that a medical man is called on to be witness to a will. On such an occasion it is his duty to satisfy himself as to the testamentary capacity of the testator. His subsequent evidence in regard to this will, in case of dispute, be of almost decisive influence, if he has taken proper means of forming an opinion. In all cases, therefore, where there may be a possibility of doubt, it is well to require the testator to show that, without extraneous aid, and without referring to the document itself, he remembers and understands all the provisions of the deed.

Evidence of the Insane.—Lunacy was, till a recent date, regarded by the law as incapacitating a patient from giving evidence in court. But according to the much more extended signification which the term ‘lunacy’ has received, it now includes states of mind which are looked on as compatible with testimonial capacity. Where the judge is satisfied that the lunatic understands the obligation of an oath, and can give a rational account of such things as happen before his eyes, the evidence may be admitted. But the weight to be attached to such evidence will still depend on the extent to which it fulfils the conditions commonly required to constitute credibility. It has been held, however, that when a person has suffered from an attack of insanity between the occurrence of the transaction and the time he tenders his testimony, his evidence cannot be admitted.

Management of Property.—Where persons are supposed to be unable, from unsoundness of mind, to undertake the management of their own property, it may be necessary that they should be placed under the protection of the Court of Chancery; but this proceeding is not usually had recourse to unless there is urgent necessity, or a strong probability that the person’s incapacity will be permanent. It is consequently resorted to chiefly in chronic or congenital cases where there is no room for doubt as to the mental condition of the individual; and in cases of recent insanity, where it is necessary to have recourse to an asylum for the protection of the individual, it may also be necessary to obtain protection for his property by the aid of the Court of Chancery. In giving evidence or framing a statement in such a case, it is important, if incapacity is to be proved, to show that within *two years* before the time of the inquisition the person was found, when placed in circumstances requiring such capacity, unable to perform the acts which the management of property necessitates. In cases of active insanity it is specially required to show, not merely that there is delusion or other symptoms of insanity, but that the insanity is of such a nature as specially to disable the person from duly performing the duties which would be required of him. Difficulties most frequently occur in cases of imbecility and dementia; but the verdicts in such cases, when disputed, will generally be found to rest rather upon the impression produced by evidence of the actual behaviour of the individual, than upon the mere medical view of his mental condition. The most effectual aid that the medical witness can render in such cases is to show whether there are or are not such peculiarities in the conduct of the person under inquisition as are known to be characteristic of imbeciles or demented persons. In undisputed cases, where the duty of the medical man consists merely in making an affidavit, there is no special difficulty to be encountered. Brevity, scrupulous accuracy, and attention to the fact that such unsoundness of mind as involves incompetency

to manage property must be established, are the most important requirements. A person found by the Court to be incapable is placed under the control of a 'committee of the person,' and the property under a 'committee of the estate.' In Scotland, proceeding by petition to the Court of Session, or, in the case of small estates, to a sheriff, for the appointment of a *curator bonis*, takes the place of the English inquisition. The chief peculiarities of the Scotch process are that it is cheaper, more easily effected, and more easily annulled, and that it does not affect the person of the lunatic. The functions of the curator correspond to those of the committee of the estate in the English court. The Scotch procedure for the appointment of a guardian of the person was virtually in desuetude until the passing of a recent statute (31 & 32 Vict., c. 100). Under this Act a brief for the cognition of an alleged lunatic is issued from Chancery, and tried before a judge of the Court of Session and a special jury. The procedure is similar to that of juries in other civil causes in Scotland, and both medical and other evidence must be produced. If the person so cognosed be found 'furious, fatuous, or labouring under such unsoundness of mind as to render him incapable of managing his affairs,' his person is placed under the guardianship of the nearest male relative found competent.

Drunkenness.—This condition is not held to deprive a man of civil capacity unless it has at the time rendered the individual unconscious of what he was doing.

JOHN SIBBALD.

CLAVUS HYSTERICUS (*clavus*, a nail).—An acute pain, often associated with hysteria, but occurring also in other conditions, which is felt in a localised point in the head, and is compared by the sufferer to the sensation that might be produced by a nail being driven into the part. See HYSTERIA.

CLIMACTERIC (*κλιμακτήρ*, a step of a ladder). This word, which properly signifies 'by degrees,' was originally employed to indicate certain epochs or periods in the life of an individual, which were looked upon as critical, and at which the body was supposed to have undergone a complete change, so that it had become entirely renewed in its structural elements. The years in which these epochs terminated were called climacteric years—*anni climacterici*—and their number was variously estimated. Thus, some recognised only three climacterics; the Greek physiologists held that there were five, ending at the seventh year, the twenty-first (7×3), the forty-ninth (7×7), the sixty-third (7×9), and the eighty-first (9×9); others made them multiples of seven or nine, or multiples of seven by an odd number. Most regarded the sixty-third year as the *grand climacteric*, but the Greeks recognised two grand climacterics, terminating respectively at the sixty-third and eighty-first years, and this special denomination was given because there was little, if any, prospect of life being extended beyond these periods. At the present day the word 'climacteric' has lost much of its original meaning, and is generally applied to certain times of life, without any reference to numbers of years, at which marked physiological or developmental changes occur, such as the period of puberty, or that of the cessation of menstruation.

A particular *climacteric disease* has been described,

which is said to occur either about or subsequent to the sixty-third year or grand climacteric, and supposed to be distinct from the natural decay and degeneration which takes place in advanced life, inasmuch as recovery often ensues. It is stated that the complaint comes on suddenly, but advances insidiously, the symptoms being at first loss of flesh and weakness, followed by loss of appetite and dyspeptic symptoms with a white tongue, which are regarded as sympathetic, sleeplessness or disturbed and unrefreshing sleep, constipation, pains in the head and chest, a frequent pulse, swelling of the legs, and an emaciated or bloated appearance of the face. The urine does not present any abnormal characters, and most of the viscera seem to perform their functions properly. Whether there is any independent disease deserving this special denomination is, in the writer's opinion, extremely doubtful.

FREDERICK T. ROBERTS.

CLIMACTERIC INSANITY (*κλιμακτήρ*, the step of a ladder; *in*, negative; *sanus*, sound).

Disorder of the sexual functions in the female is a common symptom in all forms of insanity, but in climacteric insanity the sexual changes are not merely a symptom, but probably form the basis of the mental disorder. At the menopause the individual undergoes a profound change mentally and physically, and in a person predisposed to insanity the result may be disastrous.

ÆTIOLOGY.—As in other mental disorders unstable inheritance plays an important part in the causation of the disease. During the last fifteen years in Bethlem Hospital heredity of insanity was found in 54 per cent. of the cases suffering from climacteric insanity, and the heredity from the mother's side was about 15 per cent. higher than on the father's side. 56·3 per cent. of patients were married; 43·7 per cent. were single. Governesses and other persons who have worked hard for their living often get depressed at this period probably owing to increasing anxiety as to their future. The usual stresses, such as privations, ill-health, physical disease, and the like, may be an exciting or predisposing cause as in other forms of insanity.

FORMS OF INSANITY.—There is no special form of insanity which can be classed as climacteric insanity. All writers agree that melancholia is more common, at this period, than any other variety of insanity. It is usually of a sub-acute type, or the case may be agitated or resistive in character. Sub-acute mania and delusional insanity, though less common, are by no means rare.

PRODROMATA.—The mental alterations and the somatic disturbances frequently exhibited by the healthy woman at the menopause may be the prodromata of actual insanity, especially in the presence of insane heredity.

Mental disturbances, such as the following, may be observed: insomnia, failure of attention, difficulty in performing household duties, alteration of temper, impairment of memory, altered affection towards her husband, suspicions, jealousies, and even false accusations. Sexual perversions are not uncommon: Some complain of noises in the ears and temporary deafness.

Somatic disorders are common and may be thus classified: (1) those referable to the vascular system, such as general flushings, congestion of the head, and giddiness; (2) gastro-intestinal disturbances; (3) vagaries of the menstrual function, such as

gradual cessation, sudden cessation, or irregularities in quantity and periodicity; (4) growth of hair about the face.

Drunkenness in women in this country is more common at this period than at any other. Also during the climacteric years individuals are very prone to contract drug-habits such as those of morphine and cocaine. For this reason the use of these drugs for medical purposes should be avoided if possible. *See HABITS.*

SYMPTOMS.—These are mainly an elaboration of the prodromata. The gastro-intestinal symptoms often give rise to delusions of poisoning and consequent refusal of food. The altered sensation about the limbs may be construed into delusions of electricity and magnetism. Hallucinations of the various sense-organs are usually present, those of hearing being the most common, the next in frequency being those of vision. Some observers have endeavoured to show a connection between disorders of the sexual function and *hallucination of smell*. Married women not infrequently develop delusions of suspicion regarding their husband. They accuse him of loss of affection towards them, and later of intimacy with other women. These cases are of great importance, as serious medico-legal questions have arisen from such delusions. The accuracy of the woman's statements should be carefully tested, and the physician should thoroughly examine the patient's mental condition. Full notes must be taken, and a consultation with a second medical man is advised. On the other hand many women at this time make accusations against themselves and frequently give themselves up to the police for some great crime. Confessions of this kind must be carefully investigated, notes being recorded of the patient's mental condition at the time. In the melancholic type, the feeling of depression that the patient experiences is out of all proportion to that which is justified by the circumstances in which she finds herself. The depression usually shows itself by the position of the body, which is one of general flexion, by the expression of the face, and by gestures, such as constantly swaying to and fro. Her conduct is in keeping with the mental state. She is unoccupied, and every little thing is a burden to her. Some patients are restless and agitated, whereas others remain passive. The delusions that she expresses are commonly explanations either of her altered sensations or altered feelings.

The abdominal sensations may be construed into ideas of pregnancy, or the anomalous cutaneous sensations may form the basis of delusions of electricity and the like. The feelings of unworthiness may be explained from a religious standpoint, and the patient will say that she is 'lost' or has committed the 'unpardonable sin.' The danger of suicide must always be remembered: attempts of all kinds are frequently made, and these patients should never be left alone. Suicide in women in this country is most common between the ages of forty and fifty. Memory is often deficient for recent events, but this chiefly owing to disorders of attention. With all these mental changes the physical condition of the patient suffers. The tongue is often furred, there is anorexia, and almost constantly obstinate constipation. The pulse is infrequent and very high in tension except in the agitated cases, when there is a frequent small low-tensioned pulse. The skin is dry and harsh, and perspiration deficient. The

patient commonly complains of pain or a feeling of weight upon the top of the head.

The number of cases that suffer from acute mania is small compared with the type of disorder above described. The symptoms are those commonly found with excitement. The chief danger to be encountered is the risk of exhaustion. Food of a nourishing kind must be pushed, and a furred tongue with sordes about the lips and mouth in no way contra-indicates forced feeding.

Delusional insanity is usually overlooked at first, as the symptoms are so slight and indefinite that they frequently pass unnoticed by the friends of the patient. From time to time the individual may do a strange act or make a peculiar complaint regarding the relationship of others towards her. As time goes on her actions become so unaccountable that they attract the notice of others, but in conversation she may be, apparently, as usual. Moving her abode from place to place with no definite object is not uncommonly the result of an insane suspicion that she is followed or annoyed by others. Usually for some time such persons are more insane in their conduct than in their conversation. Another class of patient may dog the steps of the clergyman or medical attendant under the impression that these latter have exhibited signs of matrimonial intentions towards them. Delusions may be of almost any type, and medical men should be on the alert, as it is most important that the condition should be diagnosed in its initial stages with a view to early treatment.

TERMINATIONS.—Most observers are agreed that about 40 per cent. of these climacteric cases recover, 10 per cent. are relieved, 6 per cent. die, and 44 per cent. remain uncured. The maniacal patients are more liable to pass on to dementia than the melancholiac. The delusional cases not infrequently progress into an organised insanity, but seldom become demented.

Influence of the Climacterium on existing Psychoses.—Experience does not justify the hope that improvement will occur in an existing psychosis at the menopause. In the majority of cases the conditions remain unaltered or undergo definite deterioration. Matusch gives the following figures: out of sixty cases, thirteen showed improvement in the existing psychosis, fourteen a deterioration, while thirty-three remained unaltered.

The *Climacterium in Men* is much later than with women, as it frequently does not occur until about sixty-five or even later. Therefore climacteric insanity in men is closely connected with senile insanity. *See DEMENTIA.*

PROGNOSIS.—In giving a prognosis the following factors must be taken into consideration: heredity; previous attacks; early treatment; form of insanity; and the general physical condition of the patient. The greater the number of previous attacks, the more guarded must be the prognosis. The outlook is better with the affective insanities (mania and melancholia) than the delusional type of insanity. The prognosis is generally fairly good, but as the disorder is usually sub-acute in character, the average duration of insanity is generally about a year, and some persons do not recover for eighteen months.

PATHOLOGY AND MORBID ANATOMY.—As in most forms of insanity the pathology is obscure. Krafft-Ebing and others are of opinion that senile brain-changes take place in climacteric insanity, and regard the disorder as a premature senility. This

view is incompatible with the recovery-rate. Degenerative changes are found in the neuron as in most mental disorders, but there is no change which can be looked upon as characteristic of climacteric insanity. See INSANITY, Morbid Anatomy and Pathology of.

TREATMENT.—When symptoms which may be prodromata of insanity appear at the climacteric period in a person with an unstable inheritance, or in a patient who has had a previous attack of insanity, prophylaxis of a general kind should be adopted, such as good feeding, rest, and change of environment. At this time women not uncommonly consult their medical attendant regarding obscure symptoms referred to the uterus and appendages, and are apt to receive local treatment for conditions more or less vague. Such measures are undesirable and often lead to self-concentration and may convert the patient into a hypochondriacal invalid. When actual insanity is present the patient ought to be placed under care with as little delay as possible, for the tendency to suicide must not be forgotten. Where the patient is to be treated of necessity depends upon the financial position of the individual. Obviously a patient whose insanity is characterised by suspicions and jealousies is best treated away from her home surroundings. The probability of the attack lasting for twelve to eighteen months must be borne in mind in deciding the course to pursue in these cases. If the patient is depressed the treatment is that of ordinary melancholia. A similar rule holds good in the case of mania. Patients must be carefully dieted with a good supply of milk and eggs. Insomnia must be treated by such drugs as sulphonal (30 grains), or paraldehyde (120 minims), or trional (20 grains). The bowels should be regulated by the use of salines or natural mineral waters. Care must be exercised in ordering stimulants, as the patient easily contracts a habit of relying on alcohol. A syrup composed of the various hypophosphites is a useful therapeutical combination in these cases. MAURICE CRAIG.

CLIMATE.—Formerly the word 'climate' (from the Greek word *κλίω*, I incline) was a term of astronomical or mathematical geography, which implied a portion or zone of the earth's surface comprised between two lines parallel to the equator, and measured by the length of time during which the sun there appears during the summer solstice, that is, by the sun's inclination. The space between the equator and the pole was divided into half-hour climates, in which the length of each day increased by half an hour; and also into monthly climates. This unequal division of each hemisphere is now replaced by a division of the interval between the equator and the poles into ninety degrees, which constitute what are called degrees of latitude, and the word 'climate' has received a more extended application.

By climate are now understood those conditions of heat, moisture, atmosphere, wind, soil, and electricity, which impress certain conditions, uniform even when apparently irregular, on given portions of the earth's surface, and which modify, also in a uniform manner, vegetable and animal life.

CLASSIFICATION.—Climate, when thus interpreted, is still principally dependent on astronomical facts, on the sun's position or inclination with regard to the earth, and on the amount of heat which it supplies to different portions of the surface of the

latter. Climate may be studied generally and locally. The division of the earth's climates is necessarily arbitrary, and many different classifications have been proposed. The most simple is that which recognises three principal kinds of climate, each susceptible of subdivision, viz.: *warm* climates from the equator to 35° lat.; *temperate* climates from 35° to 50° or 55° lat.; *cold* climates from 50° or 55° to the pole. As subdivisions we may recognise equatorial, tropical, sub-tropical, sub-polar, and polar climates; and also *insular* and *maritime* or moist climates, and *continental* and *mountain* or dry climates.

1. **Warm Climates.**—Warm climates, extending from the equator to 35° lat., that is 12½ beyond the tropics, comprise nearly all Africa and its islands, South Asia, most of the islands of Polynesia, and the portions of North and South America comprised between California and the north of the La Plata territory. In the equatorial regions the medium temperature for the year is from 80° to 84° F., the min. being 54°, the max. 118°. Near the equator the annual mean temperature decreases slowly as we recede from it, the decrease not amounting to more than 2° F. for the first 10° lat. The difference of temperature during the day is slight, but much greater during the night, owing to radiation. The general variations of the barometer are slight, but the periodical or diurnal variations are very marked. It ascends and descends regularly twice in the twenty-four hours. It ascends from 4.13 a.m. to 9.23 a.m., and descends until 4.8 p.m., ascending again until 10.23 p.m. Electrical phenomena are very decided. The rainfall is variable, but 40 inches may be given as a mean. It is generally supposed that heat is greater at the equator, and diminishes as we recede from it, but both observation and astronomical induction lead to the conclusion that not only the maximum of temperature in warm climates is attained at or near the tropics, but also the highest annual mean. The countries in which the highest degree of heat is known to be attained are near the tropic of Cancer, as, for instance, the banks of the Senegal, the Tehama of Arabia, and Mehran in Baluchistan. Moreover, the snow-line, or the line of perpetual snow, is higher at the tropics than at the equator. In the Bolivian Andes, near the tropic, it is 17,000 feet, whereas, in the Ecuador Andes, on the equator, it is only 16,000 feet. These facts are partly explained by the unequal progress of the sun after the equinox in its course towards the tropic. In the first month it passes through 12° of latitude, in the second month through 8°. At the end of the second month, therefore, it is 20° from the equator, and there remain only 3½° to be traversed in the third month. The sun receding from the tropic at the same rate at all places between 20° and 23½° of latitude, the solar rays during two months fall at noon either perpendicularly or at an angle which deviates from a right only by 3½° at most.

Another cause which tends to diminish heat in the regions near the equator is the prevalence of rain. For about five degrees north and south of the equator, in the region of the equatorial calms, there are few consecutive days in the year without rain. The principal cause both of the calms and of the rains has been attributed to the meeting in the upper atmospheric regions of the trade-winds, north and south. They neutralise each other and precipitate the vapour they hold in solution.

Regions that lie between 5° and 10° of latitude have usually two rainy and two dry seasons. The greater rainy season occurs when the sun in its passage to the nearest tropic passes over the zenith, lasting from three to four months. The lesser rainy season occurs when the sun on its return from the nearest tropic approaches the parallel of the place. The rains then last only from six weeks to two months, and are much less abundant and continual. Countries more than 10° or 12° from the equator have only one rainy and one dry season; the first begins when the sun approaches the nearest tropic, and ends some time after, when in its course from the tropic it has passed the parallel of the place. It lasts from four to six months. Local conditions may modify the course of the dry and wet seasons, as is the case in India, where the dry and rainy seasons depend principally on the monsoons. The amount of rain that falls in a short time within the tropics is very great, much more so than in more northern regions, but these heavy rains do not last continuously, as is supposed. Days of continued rain, even in the rainy season, are rarer than in the north. Still, heavy rains are apt to cause great inundations, and to cover large extents of low or level country with water, producing swamps and marshes, very injurious to health.

In the vicinity of the tropics there is a belt, extending over several degrees of latitude, where it seldom rains. This rainless tract is precisely the region which has been already mentioned as that of greatest heat. These belts of rainless regions, extending around the globe on each side of the equator, may be said to separate the countries which lie on each side of the equator from the temperate zones. Thus in Africa the rains cease on the southern border of the desert of Sahara at about 16° N., and begin again at 28° N. On the banks of the Nile the rain ceases about 18° or 19° , to begin again between 28° and 29° . The Tehama, or low coast of Arabia, is all but rainless. This rainless tract crosses Asia as far as China, where there is no rainless region, owing, probably, to the fact that all parts of China between 22° and 30° N. lat. are traversed by high mountain chains.

The influence of warm climates impresses certain peculiarities on the people who inhabit them. They are the abode of the Ethiopian and Mongolian races of mankind, and appear to have impressed the same characteristics, in a minor degree, on the Caucasian races that inhabit them: a dark complexion and black hair. The inhabitants of these countries are indolent and apathetic. The functions of the skin and liver are peculiarly active, a circumstance which exposes them to severe disease of these organs. The digestive functions are sluggish, and the nervous system is alternately excited and depressed. Remittent and intermittent fevers, dysentery and yellow fever, are common. Pulmonary consumption is frequently met with in the towns, in contradiction to received opinions.

2. Temperate Climates.—Temperate climates may be said to occupy the zones of the earth's surface comprised between 35° and 50° or 55° lat. They comprise Southern and Central Europe, with its islands; the parts of Asia which extend between the Black Sea and the Mediterranean on the west, and Japan on the east; the greater part of North America; a part of Chili and La Plata and Patagonia in South America. The mean temperature may be stated at from 60° to 50° F.

The climates in which the mean temperature is from 60° to 68° are often spoken of as temperate, but in reality they approximate closely to warm climates. The four seasons, winter, spring, summer, and autumn, are well marked, but are very variable both as to barometrical and thermometrical conditions. The mean temperature in the central regions is, for winter 38° , for spring 51° , for summer 68° , and for autumn 53° . The regions which are near the south and north limits of the northern temperate zone approximate to the meteorological characters of the warm and cold climates respectively. The periods of the year when storms, rain, and general versatility of meteorological phenomena are principally observed correspond with the vernal and autumnal equinoxes.

The influence of a temperate climate on the human organisation is salutary, extremes of heat and cold being both trying. Thus the healthiest climates of the world's surface are found in this zone. Intense heat, or even moderate heat if persistent, throws a physiological strain on the liver, skin, and digestive system, and renders mankind prone to severe and fatal diseases of these organs. Intense cold throws a physiological strain on the lungs and kidneys, and exposes them also to severe and fatal disease. The healthiest temperate climates are those in which the winter is not very cold and the summer is not very warm, and in which, consequently, there is no great or continued strain on any one class of organs. The diseases of temperate regions are those that are the best known, as their study and description constitute the foundation of pathological science, ancient and modern.

The climate which, perhaps, the best deserves the appellation of temperate is that of the Mediterranean basin. The winters are not severe on any part of its north shores, and the summers are not intensely hot on its south shores; at least the heat falls short of that of the tropics. There are many conditions of physical geography which conduce to this result. The north shores are protected from north winds by the ranges of high mountains of Southern Europe which skirt them, and the south shores are in close proximity to the hot rainless tract of Northern Africa—the desert of Sahara, which favourably modifies winter temperature. Moreover, the Mediterranean is a warm sea, but few cold rivers of considerable size flowing into it from the north, a fact which increases the temperature on its shores and islands.

3. Cold Climates.—Cold climates comprise the regions which extend from 50° to 55° F. lat. to the poles. They may be subdivided into *cold*, with a mean of from 50° to 40° F.; *very cold*, with a mean of from 40° to 32° ; *glacial*, with a mean below the freezing-point. In the austral hemisphere the zone contains but little known land, although the existence of an antarctic continent is suspected; in the northern hemisphere it comprises, in Europe, the north of Scotland, Denmark, Sweden, Norway, Iceland, Finland, Lapland, Northern Russia, Spitzbergen, Nova Zembla; Northern Asia, and some of its large plains below 50° lat., Siberia, and Kamchatka; in America, Canada, including some regions below 50° lat., the northern lands and islands of Hudson's and Baffin's Bays, and Greenland. In this zone the decrease of the mean temperature is much more rapid as we recede from the equator than it is in the tropical regions. Thus from the equator to 20° lat. the variation of the

mean temperature is not more than 7° or 8° F., whereas the variation between 55° and 75° lat. amounts to from 22° to 27° F. The coldest region of the globe is not, it would appear, at or near the pole, but at about 80° lat., or 10° from the pole, north of Behring's Straits: the cold of the glacial climates has been exaggerated. At the latitudes of from 70° to 78° , the extreme limit of human habitation, the mean annual temperature is between 10° and 17° F., i.e. 13° to 15° below the freezing-point. The extreme of cold registered, however, reaches a hundred degrees or more below the freezing-point. Owing to astronomical conditions there is great disproportion between the length of the nights and of the days at different seasons of the year. In the more northern regions, for several months in the winter the sun never appears above the horizon, and in the summer for several months the sun never disappears below it. Spring, during which the extreme cold is mitigated, lasts but a very short time, and is succeeded by summer, which is in its full strength in June and July. Temperature rises rapidly from 35° to 55° and 60° . In some northern localities it rises to 86° or 90° . Under the influence of the prolonged or persistent days, and of the increased temperature, the vegetation peculiar to each locality passes through all its phases with extreme rapidity. Towards the end of July rain and fog reappear, and are followed by snow and intense cold, the highest expression of which is in January and February. The barometrical changes are the reverse of what obtains in the tropics. Above 60° lat. the diurnal or periodical changes are scarcely perceptible, whereas general or occasional variations become more marked as we approach the pole. Electrical phenomena become less marked, and above 68° lat. they are scarcely perceptible, with the exception of the aurora borealis. The winds which predominate are the N.E. and S.W., and they change rapidly from one point of the horizon to the other, and thus frequently occasion tempests which extend over considerable areas. The quantity of rain that falls in cold climates is much less than in the tropical and temperate, with some exceptions. Between 60° and 90° lat. it amounts to only a few inches, and falls principally in the form of snow.

The influence of cold climates is shown on the inhabitants of these countries, who vary much in stature, and possess a vigorous constitution, a sanguineous temperament, great muscular development, active digestive functions, and sluggish nervous powers. Notwithstanding the severity of the climate, they generally succeed in preserving health, and live to old age, presenting few diseases referable to climatic influences. They are, however, subject to ophthalmia and amaurosis, owing to the reflection of light from the snow in the polar regions, and to scrofula and scurvy, the result of a poor and incomplete dietary. Agues and intermittent fevers from marsh-influences are rare, and not severe, and disappear entirely as we approach the pole. Continued fevers are met with, but seldom if ever epidemically.

4. Insular Climates.—Insular climates present important peculiarities. The temperature of the sea is more equable than that of the land. Owing to the action of currents, and to the circulation of its waters under the influence of heat, its superficial temperature is warmer in winter and cooler in summer—more equable—than that of the land. It

has thus a tendency to warm in winter and to cool in summer the island which it surrounds. Moreover, there is constantly watery vapour arising from the sea, which extends to island atmospheres, veils the sky more or less, shields the surface from the ardour of the sun in summer, and prevents great radiation both in summer and in winter. Thus it is that the climate of all islands is more equable than that of continents. This fact is more especially recognisable in the climate of the British Isles, which is also modified—rendered warmer and moister—by the waters of the Gulf Stream impinging on their shores. The warm Gulf Stream, commencing in the tropics, in the Gulf of Mexico, passes northwards along the shores of North America, crosses the Atlantic to the south of Newfoundland, and strikes the shores of the British Islands, of Norway up to Cape North, of Holland, and of France; everywhere raising temperature and the annual mean.

5. Maritime Climates.—Maritime climates participate in these influences: temperature is more equable, warmer in winter and cooler in summer, on the shores of seas and oceans than it is inland. Owing to this cause most of the winter sanatoria have been chosen in islands or on the coasts of oceans and seas: such as Hastings, Ventnor, Bournemouth, Torquay, Funchal, Malaga, Cannes, Nice, Mentone, Naples, Salerno, and Algiers.

6. Continental Climates.—Continental climates exhibit conditions the reverse of those which obtain in maritime climates. The tendency is to cold winters and warm summers, owing principally to the absence of the equalising influence on temperature of large masses of water. A very short distance from the sea is sufficient to establish this difference. Thus the central parts of France are very much warmer in summer and colder in winter than the coasts of Normandy and Brittany. But it is when we reach the centre of continents—Russia, Central Asia, Central America—that the difference is the most marked.

7. Mountain Climates.—As we rise above the level of the sea, we meet with two important meteorological conditions. The air becomes more and more rarefied, and the heat diminishes, independently of the more or less obliquity of the sun's rays. The higher we rise above the sea-level the more the air is rarefied, and the more the degree of heat due to the solar rays diminishes. We arrive at last, even at the tropics or the equator, at a height, variable according to latitude, where the sun's heat is insufficient to melt the snow. This is termed the snow-line. Mountains attract clouds and watery vapour, and the coolness of their atmosphere causes the precipitation of the vapour in the form of rain or snow. Thus, mountains, mountain ranges, and the glaciers they contain, are the principal cause and origin of rivers.

The influence of mountain climates, notwithstanding the clouds, fogs, and rain which characterise them, is proverbially a healthy one, owing to the purity of the air, to the shelters found in the valleys, and, perhaps, to the sparseness of population. They have of late been much recommended for phthisis. The conditions above mentioned, together with sunshine to a limited extent, are quite sufficient to account for the rarity of phthisis among their inhabitants, and for the improvement of the phthisical who resort to them.

Soil modifies climate considerably. Wet and marshy soils are cold, engendering fog and mist.

Sandy, dry, well-drained soils are comparatively warm. All sandy plains are warm in summer and cold in winter.

Such are the features which characterise the climates of the terrestrial globe, generally. Each locality, however, each mountain, plain, and valley, each city, village, and house has a climate of its own, modified by all the meteorological elements which we have successively considered. To discover what each climate is, we must study carefully the meteorological conditions and influences which we have rapidly surveyed in their application to it.

THERAPEUTICAL APPLICATIONS.—See CLIMATE, Treatment of Disease by.

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CLIMATE, The Treatment of Disease by. Though it can scarcely be said with accuracy that change of climate is a specific for disease, yet much can be effected by it in relieving symptoms, and in assisting the recuperative powers of the organism by thus improving the general health.

Change of climate, we must premise, is only a relative term. It does not necessarily involve the idea of removal to a great distance from the patient's home. A few miles' journey from the town to the country, from inland to the seashore, from the plain to the mountain, often suffices to produce marked results. One use of climate being to expose the organism to the effects of *contrast*, the element of distance comes in most when we wish to make the contrast greater; for instance, in ordering change from a cold to a warm climate or *vice versa*.

The therapeutic elements of most importance in any climate are: (1) pure air, free from dust and organic particles, or excessive damp; (2) abundance of sunshine, without excessive heat, so that much time can be spent in the open air; (3) a temperature adapted to individual requirements, some cases benefiting by warm climates, some by cold, and others by equable ones; (4) absence or presence of wind.

These four elements should be present in each of the subdivisions of climates which a therapeutic classification renders necessary, namely, climates of: (1) *the seashore*; (2) *mountains*; (3) *the desert*; and (4) *the open sea*.

The epithets 'moist' and 'dry,' which are applied to climates, are merely relative, and depend on local peculiarities of rainfall, soil, &c., as well as to some extent on season; and the essential differences between the climate of the seashore, the woodland, and the mountain remain everywhere the same. We shall say a few words about each of these, with the indications for their use. The climate of the open sea will be referred to in speaking of sea-voyages.

1. Climate of the Seashore.—The special peculiarities of this variety of climate are that the air is saturated with moisture, except when dry land winds prevail; it is dense, and, as a rule, therefore, bulk for bulk, contains more oxygen than air of any higher level; its density is liable to great and frequent but regular variations, which increase the activity of the circulatory and respiratory organs, and thus favour their functional activity; it is more equable; and, lastly, it contains saline particles in suspension.

According to Beneke, sea-air cools the body relatively more quickly than mountain air, and thus

hastens more the processes of tissue-change. Hence, the seaside should be ordered where we wish for a highly stimulating effect, as in persons of scrofulous tendency, in chronic succeeding acute diseases, or in the later stages of convalescence from the latter, in convalescence from surgical operations, or in some surgical diseases where we wish to accelerate tissue-change, without exertion on the patient's part. On account of the equability of the climate, some patients who cannot bear great changes of temperature do well at the seaside. Persons suffering from overstrain, mental or bodily, with a fair digestive power, and not liable to nervous irritability, may also be sent there. Patients liable to attacks of biliousness and liver-congestion had better avoid the seashore.

2. Mountain Climates.—Mountain climates are distinguished from seaside climates by the lessened density of their atmosphere; their lower and less equable temperature; by less humidity, though, owing to local winds, mist and cloud often form; and by relatively reduced night-temperatures in clear weather, owing to the dryness of the air, and consequent great radiation. They are characterised by diathermancy or the increased facility by which the sun's rays are transmitted through the attenuated air. This causes a difference between sun and shade temperatures of 1° for every rise of 235 feet, and produces a great diminution in the quantity of the sun's rays absorbed by the atmosphere. Mountain climates are also aseptical, being far freer from germs of all kinds than seaside ones.

The general action of mountain air is to produce a freer circulation of the blood and greater vascularity of the lungs, owing to deeper and more frequent inspirations and greater ease of bodily movement. Owing to the cooling of the body by the lowered temperature more food is required, the appetite improves, and the body becomes better nourished and gains weight. Another effect is the greater development of the heart and lungs and the enlargement of the thorax.

The intensity of the effect is, broadly speaking, directly as the height. The term 'mountain climate' is applied in medical parlance to elevations in Europe of from 3,000 to 6,000 feet, though in South America patients have been sent as high as 10,000 feet, or higher.

Mountain climates are indicated: (1) in cases of hereditary tendency to phthisis in young persons with narrow shallow chests, and who are growing too fast. (2) In chronic phthisis and pneumonia; remembering, however, that phthisis occurs at all elevations. The coolness of the mountain air in the height of summer is an important element in phthisical cases, which always suffer from great heat. (3) As a tonic and restorative in persons suffering from overwork in business or literary pursuits, and who have no real organic disease. (4) Generally to complete the convalescence from acute diseases of individuals not past middle life, with a fair amount of muscular power and bodily activity. Mountain climates are not advisable in cases of chronic bronchitis, heart-disease, emphysema, Bright's disease, chronic rheumatism, hæmoptysis, nor for aged persons.

3. Climate of the Desert.—This is the combination of warmth and dryness, which is to be found in the great deserts of Australia, of the United States and of Africa, such as the Egyptian and the Great Sahara. The characteristics of desert

climate are: (1) warmth; (2) large radiation as shown in the difference between night and day temperatures; (3) dryness of atmosphere as shown in either the absence of rainfall or its small amount, and by the low percentage of atmospheric moisture, and the wonderful preservation of human bodies as mummies; (4) great atmospheric purity, shown by the total absence of carbonic acid, and the fact that meat, instead of putrefying, dries up without decomposition.

Desert climates are specially suited to chronic rheumatism, granular disease of the kidney, bronchitis, bronchial asthma, and emphysema, and some forms of pulmonary tuberculosis.

4. Ocean Climates: Sea-Voyages.—Voyages have been much recommended in the treatment of phthisis in its early stages, with a view to enable the invalid to spend much time in an exceedingly pure and fairly equable atmosphere, and to secure a sufficient amount of bodily movement without great fatigue.

The drawbacks to the sea ought to be provided against as far as possible, although certain of them from their very nature must be endured. The principal of them are the impossibility of escape from bad weather, and the confinement, perhaps, to ill-ventilated cabins, when such occurs; the absence of sufficient light and air below decks, the latter being felt very much at night; want of variety in the diet after a certain time, and in many instances of fresh food, milk, &c.; monotony in society and occupations; and, lastly, the inconveniences arising from crowding of the maindeck with hen-coops, sheep-pens, &c., and in steamers from the smoke of the engine, and the smell and vibration of the machinery, though it must be admitted that in the most modern vessels these are generally got rid of.

The routes generally recommended to invalids are chiefly: (1) to Australia or New Zealand, 90 days in a sailing vessel, 30 to 40 in a steamer; (2) to the Cape of Good Hope, 16 to 20 days; (3) to the West Indies and back, 6 to 8 weeks; and (4) the Brazilian voyage occupying 2 to 3 months, where the steamer touches at Lisbon, Teneriffe, Bahia, Rio de Janeiro, Monte Video, and Buenos Ayres, and experience of tropical climates is mingled with that of temperate ones. Short cruises in the Mediterranean, or to the latitudes of the Canaries and Azores, are suitable for certain cases where expense is no object. Hence where a long sea-voyage is indicated, route (1) is decidedly the best. England is quitted in the beginning of October, Australia (Sydney or Melbourne), or New Zealand (Wellington), is reached early in January, and the return voyage is begun not later than the end of February.

Patients should not remain in the coast-towns of Australia in summer on account of the heat and the dust. They should go to the Australian Alps, or to the Darling Downs in Queensland, or else they should cruise from port to port, or cross to New Zealand or Tasmania.

In returning, the route round Cape Horn should be avoided, on account of the great risk of the climate of the South Pacific Ocean, and the chance of encountering icebergs, fogs, and unfavourable winds. Either the patient should come back round the Cape of Good Hope, which is the best course, or, if his strength permit, he may cross to California, travel overland to New York and thence by steamer; or, lastly, he may come by the

Red Sea, Suez Canal, and Mediterranean, which from the many climatic changes is not to be recommended.

The invalid must expect about twenty wet days on the long voyage out. The temperatures met with range from 40° to 80° F., the coldest and most uncertain weather occurring in the North Atlantic and South Pacific Oceans.

Choice of Vessel.—The following considerations may be useful in deciding between steam and sailing vessels:—

For a steamer there is the greater certainty in predicting the length of the voyage, and the calms of the tropics are sooner passed.

Against steamers there is the nuisance of steam and smoke on deck; much space is taken up by the engines, stokers' rooms, &c., and hence other parts of the ship are more crowded; the bilge water is tainted with the engine-grease; there is the wearying grind of the screw by day and by night; while more seas are shipped, because a steamer can run against a head-wind.

For sailing vessels there are the advantages of more room, light, and air in the cabins, and the absence of the above-mentioned disagreeable conditions.

Against them there is the longer and more uncertain duration of the voyage, the necessity of shaping their course by the prevailing winds, the possibility by route No. 1 of being becalmed at the equator for days or weeks, in a humid atmosphere of 80° to 85° F., and the greater risk of running short of stores.

The vessel selected should not be under 1,000 tons, and her age, class of passengers, the character, temper, and standing of her captain, as well as the reputation of the owners, and the size of her cabins, should be carefully ascertained. Of course it is a *sine qua non* that she should carry a duly qualified medical man.

The cabin for the outward voyage to Australia should be on the port side, so as to get the breeze in hot weather, and *vice versa* in returning.

The cabin outfit should include a spring-mattress, with hair (not wool) mattress over, a folding easy chair, chest of drawers, carpet, curtains, and sponge-bath (Faber). Clothes of various degrees of thickness are essential, both for body and head, and a waterproof suit is necessary for bad weather.

Plenty of linen must be taken, as washing is difficult on board, and there should be a supply of preserved milk, meat or essence of meat, fruit, and light wines.

The *indications* for a sea-voyage are hereditary tendency to phthisis, hæmorrhagic phthisis, apyrexial cases of limited excavation, especially in middle-aged people, chronic pleurisy and chronic empyema, various neuroses, the result of overwork and especially insomnia. Of course a tendency to protracted sea-sickness is a distinct contra-indication.

The invalid should be careful not to overtax his digestion too much; he should take regular daily exercise on deck to the extent of his strength, have some definite occupation to beguile the time, and, if possible, be accompanied by a personal friend (Faber).

Choice of a Climate—General Hints.—It is a good plan, if possible, to order a patient a climate with that mean temperature and relative humidity which he is known to tolerate well (Sigmund). The patient's disposition must be considered, and

a lively or a quiet place chosen according to his temperament. We should not send a poor man to a place beyond his means, otherwise he has to grudge himself many comforts, and loses much, if not all, the benefit of the change. In sending patients to the South of Europe this rule is too often neglected.

The special indications for the climates of particular places can only be understood by studying their local aspect. Generally speaking, as far as Great Britain is concerned, the climate of the east coast is colder and drier than that of the west and south coasts. In Europe the north and west coasts are moister and cooler than the shores of the Mediterranean. As to season, mountain climates include some very important winter stations. Certain parts of the sea-coast are adapted for invalids at all seasons of the year; but as a rule the northern coasts of Europe and the eastern or south-eastern coasts of Great Britain are best suited for summer, and the south, west, and south-western for winter residence. The Mediterranean coast is only to be recommended from mid-October to the middle or end of May, and Egypt should be quitted not later than April. A word may be added as to the advantages of wintering in the South of Europe. It is incontestable that the invalid gets a milder winter, a longer autumn, and an earlier spring. Although there is no place where some days of bad weather do not occur, or where uninterrupted calms are met with, yet the number of rainy days is fewer, there is more sun, little or no fog, and, except in the neighbourhood of the Pyrenees, little or no snow or ice. The scenery is picturesque and attractive, and the invalid is able to spend much time in the open air, and to sit out of doors on many days, even in mid-winter, though under the open-air system this is done in England. The drawbacks to the South are the risk of chills, owing to the difference between sun- and shade-temperatures, especially at first, when persons are unaccustomed to the climate, and fail to take sufficient care; the occasional occurrence of high winds, especially in spring; the more limited accommodation, owing to the expense of rooms and living; and the absence of many so-called 'home comforts.'

Those who visit the South must remember that the curative value of the climate consists in its *allowing much time to be spent in the open air*, and in its milder temperature and drier air, which protect the respiratory organs from fresh inflammatory attacks. A south room and warm clothing of the texture usually worn in England in autumn are essential, and a coat or wrap should always be carried out of doors in mid-winter to put on in passing from sun to shade.

Patients with acute diseases of the respiratory organs should not be sent to the South; and high fever, excessive weakness, or the necessity of remaining in bed, are also contra-indications, owing to the fatigue and risks of the journey and the need of home comforts. Cases of mental disease with excitement, where rest and protection of the mind and body are of primary importance, should likewise not be sent.

In the convalescence from acute diseases occurring in autumn, where a cold northern winter would prevent open-air exercise, and probably set up fresh exacerbations, southern winter climates are of great value.

In ordering change of climate the accommodation,

food and water-supply, soil and drainage of the locality chosen, should be carefully considered, especially if the distance be a long one. The best climate may be unavailable for the invalid, owing to defects in one or more of these particulars.

Lastly, the patient's own feelings should be carefully consulted before he is sent far away from home. In some cases all the benefits of climate are counteracted by 'home-sickness.' *Celum non animum mutant qui trans mare currunt.*

We shall now discuss the use of change of climate in reference to various groups of disease.

1. Of the Nervous System.—For most diseases of the nervous system no special climate is indicated; as a rule the bracing ones do good, and hot climates are to be avoided. The calm still atmosphere of pine-woods has had a beneficial effect in inducing slumber. For all neuroses, which result from overwork, if change of climate is advisable at all, sea-voyages in temperate climes are indicated, and chiefly do great good by promoting sleep.

In hysteria and hypochondriasis, trips by sea or judiciously conducted land-tours often do good; the effect produced being chiefly due to diversion of the attention by the change of scene, although the bracing influence exerted on the system at large must be taken into account.

Temperate and bracing climates are, as a rule, to be recommended in nervous diseases, to restore the general tone of the system.

The immediate neighbourhood of the sea not infrequently causes nervous excitement, neuralgia, and sleeplessness.

2. Of the Respiratory and Circulatory Systems.—In chronic bronchitis, emphysema, bronchial and spasmodic asthma, as well as in chronic pharyngeal and laryngeal catarrh and laryngeal ulceration, the following climates may be recommended:—Africa (South), Algiers, Australia, the Riviera (East and West), Upper Egypt, Southern California, and the English South Coast from Dover to Penzance.

Change of climate is of great value in convalescence from the acute, and as a prophylactic and curative measure in the chronic forms of bronchitis; but we must remember that where there is copious expectoration a dry climate such as that of Egypt is indicated, while in the irritative forms with scanty sputa (*bronchitis sicca*) a moderately moist warm climate like that of Madeira is generally suitable. In emphysema we should choose a mild and not too dry climate, if possible in the neighbourhood of pine-woods, such as Arcachon on the west coast of France, or Bournemouth. In spasmodic asthma the choice of climate must be partly a matter of personal experience, but, as a rule, dryness is required. Bournemouth in England, and Hyères in the South of France, are probably most worthy of trial.

In the early, active, and quiescent forms of phthisis, the climates of the high altitudes have been proved to be more effective than any other to arrest the disease. Such are:—Davos, St. Moritz, Arosa and Leysin of the Swiss mountain stations; the South African Highlands of the Cape Colony, Orange River and Transvaal; the States of Colorado and New Mexico in the United States, and the various sanatoria in the Himalaya, and in the Andes. For more chronic and more advanced cases the warmer climes without altitude are

indicated, such as the Riviera, the Mediterranean Isles, Algeria, and Malaga; while for those who cannot travel far the English South Coast stations are suitable. But whether at home or abroad, the 'open-air system' with all hygienic and dietetic details should be carried out carefully in all weathers, and the southern resorts have the great advantage of promoting, by their fine climate, the practice of open-air life.

Chronic pleurisy, chronic pneumonia, bronchiectasis, and bronchial asthma are all largely benefited by the climate of the desert.

In chronic endocarditis, pericarditis, and in heart-disease generally, a rather bracing climate without extremes is usually indicated. Here both the tonic effects of climatic change, and the prevention of pulmonary complications and fresh rheumatic attacks, must be taken into account. Mountain resorts of moderate height, well protected from sudden changes of temperature, may be prescribed in summer.

3. Of the Renal Organs.—In convalescence from acute nephritis, and in all forms of chronic Bright's disease, but especially *catarrhal* nephritis, warm dry climates are indicated. Among the best are Upper Egypt, the Riviera, the Cape of Good Hope (inland), Bombay; and, in England, Brighton, Folkestone, Hastings, and Ventnor.

In renal calculus, removal from particular districts in which stone is known to be prevalent may possibly be of use in some cases.

4. Of the System at large.—Change of climate is here nearly always indicated:—

(1) In convalescence from typhus and typhoid fevers, scarlet fever (at the end of the desquamative stage), measles, diphtheria, and acute rheumatism; also in the third stage of protracted whooping-cough.

(2) As a prophylactic against hay fever, influenza, and malaria; also against rheumatism and phthisis by withdrawal from damp districts, and against goitre and cretinism by removal from the *ensemble* of conditions to which the latter are due.

(3) In rickets, scrofula, chlorosis, general anæmia, and functional debility. Here, where a pure air and a sunny atmosphere are the chief indications, the combination of warmth and a stimulating atmosphere is desirable. In anæmia bright sunshine and warmth have an extraordinarily beneficial effect and rapidly promote the increase of red corpuscles. Seaside climates, like the East Coast ones of Margate, Ramsgate, Cromer, and Aldeburgh, are most useful.

EDWARD SPARKS.

C. THEODORE WILLIAMS.

CLINICAL (κλίνη, a bed).—This word literally signifies 'of or belonging to a bed;' but it has been especially applied to the practical study and teaching of disease at the bedside, and has more recently been extended to all that relates to the practical study of disease in the living subject generally.

CLONIC (κλόνος, tumultuous movement).—This word is applied to spasmodic movements which are of short duration, and alternate with periods of relaxation. See CONVULSIONS; and SPASM.

CLONUS (κλόνος, tumultuous movement).—The term now applied to the movement of a portion of a limb by striking or forcibly stretching one of its tendons. See SPINAL CORD, Diseases of; § 5. SPINAL REFLEXES.

CLOT.—A clot, or coagulum, is the product of the formation of fibrin (*see* BLOOD, Morbid Conditions of). Coagulation of the blood within the blood-vessels is described under THROMBOSIS; the coagulation of extravasated blood under EXTRAVASATION, and BRAIN, Hæmorrhage into; LUNGS, Hæmorrhage into, &c.

CLOTHING.—See HEALTH, Personal.

CLOUDY SWELLING.—See DEGENERATION.

CLUBBING OF FINGERS.—See FINGERS, Clubbing of.

COAGULATION-NECROSIS is a peculiar change which tissues may undergo in the process of dying. The cells affected present a hyaline or very slightly granular appearance, and subsequently suffer fatty degeneration. The condition is probably due to the toxins of bacteria and is found only in parts which are freely supplied with lymph. Coagulation appears to take place both in the protoplasm of the cells and in the lymph with which they are surrounded.

COAGULUM (*coagulo*, I curdle).—See CLOT.

COAL-GAS, Poisoning by.—Coal-gas, so largely employed for illuminating purposes, is a compound containing—in addition to olefiant gas and analogous hydrocarbons, on which the luminosity principally depends—certain so-called diluents, which burn with a non-luminous flame, namely, hydrogen, marsh-gas, and carbonic oxide, along with what are termed impurities, of which the chief are carbonic acid, sulphuretted hydrogen, and bisulphide of carbon. On these impurities the characteristic odour mainly depends. This odour, which is perceptible even to the extent of 1 in 10,000, is a valuable safeguard against accidents from escape of gas. The recently introduced water-gas, made by decomposing aqueous vapour over burning coke, while it is more dangerous—only 1 per cent. being necessary to produce a fatal result—has not even this safeguard, for it is quite odourless.

A mixture of coal-gas with the air inhaled exerts a deleterious effect on the system, and proves fatal when it reaches a certain percentage. In addition to the danger from inhalation, fatal accidents frequently occur from the explosive nature of the compound which is formed when the gas reaches the proportion of 1 to 10 of the atmosphere. Much less than this, however (a non-explosive mixture, therefore), proves fatal if long inhaled.

MODE OF ACTION.—The toxic agent in coal-gas is carbonic oxide. Ordinary coal-gas contains from 4 to 8 per cent. of carbonic oxide; coal-gas which has been adulterated with water-gas contains from 14 to 16 per cent. or more; carburetted water-gas—that is, a mixture of water-gas and oil-gas—contains 18 to 20 per cent. The toxic effects produced are those of carbonic oxide. See CARBONIC OXIDE, Acute Poisoning by.

SOURCES OF POISONING.—Poisoning by coal-gas is only known of as an accident. Occasionally sudden fatal consequences ensue among workmen from exposure to a sudden rush of undiluted gas from gasometers and mains. More commonly slowly fatal cases result from the gas-tap in a bedroom being left open carelessly, from accidental

extinction of the light, or from leakage of gas-pipes in a house or at a distance. In the latter case the gas gains access to the house through cellars, walls, and more especially by means of drains and sewer-pipes, most commonly during winter and severe frosts, presumably from the ground-surface being frozen and preventing escape into the streets. After passing underground, coal-gas loses its odour, which is such a safeguard; most of the carburetted hydrogen and marsh-gas is absorbed, and consequently the proportion of carbonic oxide is increased.

SYMPTOMS.—Gas, even when in comparatively small proportion and just sufficient to cause an unpleasant odour, acts deleteriously if long breathed, and gives rise to headache and general depression of health. A very slight escape of coal-gas—just sufficient to produce a close smell, not like that of gas, and therefore not recognisable as such—will cause relaxed or ulcerated throat, probably from the irritation of the sulphur-compounds.

In severe and fatal cases the symptoms which have been noted are headache; nausea or vomiting; vertigo; and loss of consciousness, passing into deep coma and muscular prostration, which resembles the apoplectic state, the individual lying insensible and incapable of being roused, with livid features, stertorous breathing, and froth at the mouth. Death usually occurs quietly, in this state of coma, but occasionally with convulsions.

The state of the pupils does not seem to be constant, though they are generally dilated before death.

FATAL PERIOD.—The fatal period of poisoning by coal-gas is extremely variable, and a remittent character of the symptoms sometimes gives rise to fallacious hopes of recovery in cases which ultimately prove fatal.

DIAGNOSIS.—The smell of gas in the clothes, breath, and perspiration, which continues for a considerable time after removal from the polluted atmosphere, is the best indication of the cause of the coma.

POST-MORTEM APPEARANCES.—The appearances are usually those of poisoning by carbonic oxide, with, in addition, the odour of coal-gas, which is invariably perceptible on section of the body, especially when the ventricles of the brain are newly laid open. In some cases the blood may be dark in colour; usually it is cherry-red, producing the characteristic appearances seen in carbonic oxide poisoning. A positive reaction with the spectroscope and with the chemical tests for carboxy-hæmoglobin determines the cause of death. See SPECTROSCOPE IN MEDICINE, *Coloured Plate*.

TREATMENT.—Instant removal from the polluted atmosphere is the first thing to be attended to. Attempts must then be made to cause oxygenation of the blood, by artificial respiration and excitation of the respiratory centres, by reflex stimulation of the face, chest, &c. Pure oxygen may be administered. As, however, the compound which carbonic oxide makes with the colouring-matter of the blood is a very stable one, and not easily broken up by the introduction of atmospheric air or oxygen, it not infrequently happens that these measures prove of no avail. In such cases it would be highly advisable to perform venesection, and then transfuse fresh blood, a plan of treatment which has been found successful in poisoning by carbonic oxide.

D. FERRIER.

J. DIXON MANN.

COARCTATION (*coarcto*, I straiten).—A pressing together, narrowing, or stricture of any hollow tube, such as the aorta, intestine, or urethra.

COCAINE-HABIT.—See HABITS.

COCCYGODYNIA (κόκκυξ, the cuckoo; and δόνη, pain).—SYNON.: Fr. *Coccygodynie*; Ger. *Steissbeinschmerz*.

DESCRIPTION.—This name has been given to a peculiarly severe and obstinate neuralgic pain in the neighbourhood of the coccyx, incited by such actions as defecation, walking, sitting down, or rising from a recumbent position. It may arise from injury, such as fracture, or luxation, or horse-exercise; or it may be the result of inflammatory affections of the sacro-coccygeal joint or of the pericoccygeal fibrous structures. Coccygodynia is also a concomitant symptom of many rectal, vulval, vaginal, and uterine disorders, being excited whenever any of the muscles which have their insertion in or near to the coccyx are brought into play. It not infrequently is a sequela to a difficult instrumental parturition, or may be a neurotic condition associated with gout or rheumatism. See NEURALGIA.

TREATMENT.—In cases where the affection is primary, recourse must be had to rest in the lateral posture, anodyne suppositories of extract of belladonna and opium, and to the careful regulation of the bowels. If the pain be very severe, a local hypodermic injection of morphine may have to be administered. To be successful in our treatment we must, however, always endeavour to differentiate by means of a carefully conducted systematic examination the cause or causes which may be at work, and to attend to the proper indications. Tenotomy and excision of the coccyx have not proved to be uniformly successful, and should therefore only be practised in specially selected cases. Attention must be paid particularly to the nervous system, and the general nutritive processes improved by the administration of arsenic, quinine, and iron.

JOHN HAROLD.

COLD, A.—A popular name for Nasal Catarrh.—See NOSE, Diseases of.

COLD, Effects of Severe or Extreme.—**INTRODUCTORY.**—The general effect of exposure to severe or extreme cold is to lower, even to extinction, all vital activity. The blood-vessels, especially the smaller arteries and capillaries, after a brief period of congestion, become contracted, the latter to such an extent as no longer to permit the passage of the red corpuscles; the normal condition, composition, and structural integrity of the various tissues are more or less impaired, or altogether destroyed; the red corpuscles become first crenated and later disorganised and broken up, the watery elements of the blood thicken, and when frozen become more or less separated from the saline and other constituents; and those processes of chemical and physiological change which are essential to every manifestation of life, being only possible within certain very narrow limits of temperature, are hindered or absolutely prevented.

The more special effects vary in degree and kind:—1st, with the degree of cold, the duration of the exposure, and the medium or manner of application; 2nd, with the part, and extent of surface exposed; and 3rd, with the general constitution and physiological condition of the sufferer.

Moderate cold, acting during a short time, or even severe cold during a still shorter time, followed by the glow of speedy reaction, exercises a tonic and stimulating influence. If, however, the cold is very severe, or the exposure too long, the glow of reaction does not occur, but a sense of depression is experienced, from which, at best, recovery takes place but slowly. Continued exposure to such a degree of cold as is yet not incompatible with the maintenance of life nevertheless keeps at a low ebb activity of nutrition and function alike.

In the case of non-hibernating mammals cooling of the body to 64°F . is in a few hours followed by death, unless artificial respiration and heat be applied. Reincke and Nicolaysen have recorded cases of drunkards whose rectal temperature was as low as 76°F . owing to exposure to cold; the patients were unconscious, but under treatment they completely recovered in two or three days.

Dry cold is much less readily injurious in its influence than cold associated with wet. The better conductor of heat the medium is, the more speedily and completely does it reduce the temperature of the part with which it is in contact. Immersion in water cools more rapidly than exposure to air of the same temperature; and contact with wool, wood, or metal, of the same degree of coldness, excites in each case a different sensation, and leads to a different result, or to similar result, but with very different rapidity. Constant renewal of the medium in contact hastens the cooling effect; and a continuous draught of only moderately cold air may do more to chill than temporary exposure to an intensely cold but still atmosphere. If some external part, and a comparatively small extent of surface only, be acted upon, the effect may be simply local, and the general disturbance of the system scarcely appreciable. But if the whole body, or a considerable extent of surface, or any important internal organs be acted upon, a proportionately serious general effect is produced.

The young (infants especially) and the aged alike ill sustain exposure to cold, and are most liable to suffer, not only from its direct effects, but also from the various maladies to which it gives rise. The feeble, ill-nourished, and broken in health, especially the subjects of organic disease, or of degeneration due to habitual intemperance, readily succumb, or only slowly and imperfectly recover. Among the healthy and otherwise vigorous, hunger, fatigue, sleep, anxiety of mind, fear, and mental depression of whatever kind, lower—too often even to fatal issue—the power of resistance to the deadly influence of cold.

LOCAL EFFECTS.—For local effects of exposure to cold, see CHILBLAIN; FROST-BITE; and GANGRENE.

GENERAL EFFECTS.—The general effects and symptoms produced by exposure to severe or extreme cold vary in different cases. Temporary exposure produces, first, a sense of coldness or chilliness, associated with paleness and corrugation of the skin (the so-called *cutis anserina*), then shivering and tingling sensations, followed by numbness and diminution of muscular activity and power. Healthy reaction restores more or less quickly the normal condition. Prolonged exposure to extreme cold gives rise to a series of symptoms, graphically described by Beaupré somewhat as follows:—Reaction has a limit, and a moment arrives when the powers are exhausted. Shiverings, puckering, paleness

and coldness of the skin, livid spots, muscular flutterings, are symptoms of the shock given to the vital forces; syncope approaches; the stiff muscles contract irregularly; the body bends and shrinks; the limbs are half-bent; lassitude and languor invite to repose; a feeling of weight and numbness retards the steps; the knees bend; the sufferer sinks down or falls; the propensity to sleep becomes irresistible; everything grows strange; the senses are confused; the mind grows dull, the ideas incoherent, and the speech stammering or raving; respiration, at first interrupted, becomes slow; the heart's action is feeble, quick, hard, irregular, and sometimes painful, and the pulse progressively smaller; the pupils dilate; the brain becomes stupefied; and finally deep coma indicates the approach of death.

Other and somewhat different effects and symptoms, attributable to differences of circumstances and condition, have been from time to time observed. Distressing and almost intolerable thirst, with loss of appetite for food, is often experienced; and the attempt to obtain relief by sucking snow or ice only adds to the suffering. Somnolence is by no means so constant an effect as is commonly supposed—at any rate in the earlier stages and less extreme cases. On the other hand, inability to sleep has proved a common cause of suffering and consequent loss of strength. The manifestations of brain-disturbance due to exposure to cold, varying as they do from dulness, incoherence, wandering, and thickness of speech to raving delirium, are especially worthy of note, inasmuch as they resemble, and are liable to be mistaken for, the effects of alcoholic intoxication.

Death from the direct and immediate effects of cold is rare in the British Islands; but it is estimated that in the Russian Empire, on an average, 694 deaths occur annually from this cause. The length of time during which exposure can be sustained varies greatly with the condition of the individual and with surrounding circumstances, as well as with the degree of cold. Under ordinary circumstances, an hour's exposure to intense cold, without due protection against the loss of natural heat, often suffices to determine a fatal result. At the same time, well-authenticated cases are on record, in which persons buried for days in snow have nevertheless survived and ultimately recovered with little permanent damage.

MODE OF DEATH.—Asphyxia and syncope have been given as the immediate cause of death from exposure to severe cold; the evidence, however, is insufficient. The true explanation appears to be this—the chemical processes of the living tissues of a warm-blooded animal can only take place within a certain range of temperature; before the minimal point (about 68°F .) is reached, the activity of the nervous system is profoundly depressed, the interaction between the component parts of the organism ceases, physical and chemical changes occur in the cells and render life impossible.

ANATOMICAL CHARACTERS.—The appearances presented on *post-mortem* examination are somewhat differently described and estimated by different observers; but none of them are absolutely pathognomonic, and some are as likely to be produced by exposure of the body after death as during the process of extinction of life. Among the more noteworthy are the following:—strong cadaveric rigidity; paleness or waxy whiteness of skin, with patches of more or less bright redness about the face,

neck, and limbs, especially on exposed or prominent parts; a contracted and shrunken condition of the male genital organs; comparative bloodlessness of superficial and external parts; accumulation of blood in and about the thoracic and abdominal viscera; great distension of all the cavities of the heart, with more or less clotted and often bright-coloured blood; the blood in other parts also sometimes of brighter colour than usually seen on *post-mortem* inspection; hyperemia and congestion of the lungs; hyperemia of the brain, overfulness of the sinuses, and excess of serous fluid in the ventricles, and at the base, in some cases; in others, comparative bloodlessness of the surface of the brain, and no distension of the sinuses; excessive fulness of the urinary bladder; and, lastly, separation of the cranial bones along the coronal and sagittal sutures. The lines of reddish or brownish staining along the course of the superficial blood-vessels, relied on by some as pathognomonic, are certainly not so, inasmuch as they depend upon exosmosis of the colouring-matter of the blood set free by disruption of the corpuscles, which may be effected by freezing after death, as well as before.

TREATMENT.—The treatment of sufferers from the effects of cold consists in the restoration of warmth, and the rekindling of those processes by which the natural heat of the body is maintained. But this must be done gradually, and with great care. As in the treatment of a frost-bitten part, so in the treatment of the body generally, all sudden or rapid elevation of temperature must be avoided. The sufferer, divested of the clothing previously worn, and wrapped in blankets, should be placed in the recumbent position in a room the air of which is dry, still, and cold, but capable of being gradually warmed. Gentle but continuous friction should be made over the trunk and limbs, care being taken that rigid or frozen parts be not damaged by rough manipulation. At first, ice or ice-cold water may be used; afterwards dry rubbing with flannel or with the hands is better; later still some stimulating liniment may be employed. Some recommend immersion of the body in a bath of cold—at first ice-cold—water, the temperature of which can be gradually raised. This method would seem easy and advantageous, if means are at command. When the sufferer can swallow, warm gently stimulating drinks—as tea, coffee, aromatic infusions, beef-tea, or soup—may be given, at first without, but later with some wine or spirit. Alcohol, though useless or injurious if taken to fortify against cold, is useful and beneficial when judiciously administered as a restorative after exposure. In all cases of insensibility and even apparent death from cold, every effort must be made to restore animation; artificial respiration should be performed, and the attempt must be persevered in for a considerable time before given up as hopeless. It is often difficult, sometimes impossible, to judge whether life is absolutely extinct or not. And while, on the one hand, it is important that the temperature be not raised too quickly, lest reaction should be too strong or dangerously irregular, on the other hand it is equally if not more important that the needful measures be adopted without delay, and carried out not too slowly, lest the chance of revival should be lost. In the less severe cases, restoration of warmth may be comparatively quickly accomplished. The state of the bladder should always be examined, and relief afforded, if needful,

by aid of the catheter. Attention to the general health is often requisite for long after recovery from the more immediate effects of exposure has taken place. Rest, good nourishment, and tonics are indicated.

Cold as a Cause of Disease.—As a predisposing and exciting cause of disease, cold proves, in the British Islands, year by year, more fatal in its effects probably than any other single condition or influence. Any considerable fall in the thermometer below the average standard during the colder months of the year is constantly followed by a corresponding rise in the death-rate, and an increase in still greater proportion in the amount and extent of sickness and suffering. The Reports of the Registrar-General clearly prove this, so far as the death-rate is concerned. A striking instance may be quoted. In the week ending December 19, 1863, in the London district, 1,291 deaths were registered. Severe frost set in, and in the week ending January 9, 1864, the number rose to 1,798. The week following, ending January 16, no fewer than 2,427 deaths were registered. This enormous increase could be attributed to no other cause than the effects of the severe cold which prevailed. The Registrar-General also shows that after the age of from twenty to forty the mortality from cold increases in something like a definite ratio with increasing years.

General depression of the vital powers, congestion and functional derangement of various internal organs—the lungs, liver, and kidneys—catarrhal and other forms of inflammation of the mucous membranes, especially of the respiratory tract, but also of the intestinal canal and bladder, paralysis from central or peripheral lesion, together with rheumatism, chilblain, frost-bite, and gangrene, constitute the list of maladies most commonly caused and fostered by exposure to the influence of cold.

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COLD, Therapeutic Use of.—**GENERAL PRINCIPLES.**—The general effects of cold, however applied, are to lower temperature, to diminish sensibility, and to contract the tissues and vessels. The cold bath and cold sponging alike have the effect of lowering the temperature of the body. The fall of temperature sometimes is but transient, reaction setting in and heat of surface returning when the body is withdrawn from the cooling medium. At other times the temperature does not rise to the same extent after the individual is removed from the bath. Cold baths and their uses are treated of in another article (*see BATHS*), but attention may here be drawn to the practice of cold sponging over the surface as an efficacious means of lowering preternatural heat and relieving dryness of the skin during fever. The addition of enough solution of potassium permanganate to give a slight purple tinge to the water makes it especially refreshing to the patient. Reduction of hyperpyrexia by means of cold is now a well-established therapeutic method. *See FEVER; TEMPERATURE; and FEBRIFUGES.*

METHODS OF APPLICATION AND USES.—There are several methods of applying cold therapeutically:—

1. **Cold Affusion and Wet Packing.**—In many nervous affections, such as maniacal delirium, chorea, and hysteria, cold douches, shower-baths,

and affusions are valuable as restorative and curative agents.

To resuscitate those who are in danger of death from a narcotic such as chloroform or opium, wrapping the patient severely with a cold wet towel is an efficient method. In sunstroke, cold affusion over the head and neck may be resorted to, provided the skin be not cold and clammy, and the patient in a syncopal state. In conditions of nervous spasm—of the larynx, for example—cold touches over the neck may prove useful. In cases of extreme debility, with damp cool skin, low muttering delirium, and very feeble pulse, cold affusions are dangerous.

In some cases of fever, where for any cause a cold bath is objectionable, the patient may be wrapped in a wet sheet and then covered with a few blankets. The sheet as it becomes heated may be changed for one fresh and cold, or very cold water may be squeezed from a sponge over the sheet as the patient lies rolled up in it on a waterproof cloth. Allowing fragments of ice slowly to melt on the sheet which covers the patient is, in the writer's experience, a successful way of treating the hyperpyrexia of acute rheumatism. The cases best suited for treatment by the wet pack are those where the skin is very dry and hot, and the patient exceedingly restless and delirious.

2. Cold Compresses, Ice-Bags, Irrigations, Lotions, and Injections.—Cold may be continuously applied with a view to abating undue heat of a part of the body. Iced-water rags or compresses may be placed over an inflamed throat, or on the head in inflammation of the brain. In acute pneumonia Niemeyer recommended the use of cloths wrung out in cold water, and applied so as to cover the chest, and especially the affected side. These compresses are repeated every five minutes. Pain and dyspnoea are much relieved; sometimes the temperature falls a degree. The necessity of having to change the compress so often, and thus disturb the patient, is a great objection to this mode of applying cold. To avoid this disadvantage a bag of ice is sometimes applied over the inflamed lung. A frozen compress over the cæcum is useful to stay peristalsis and check hæmorrhage in typhoid fever.

A powerful sedative and antiphlogistic effect of cold can be obtained by *irrigation*; i.e. allowing cold water to fall drop by drop on a cloth, so as to keep it continually wet with fresh supplies of water. This may be done by suspending over the part to be irrigated a bottle of water, in which a few pieces of ice may be put; one end of a skein of cotton, well wetted, is then allowed to hang in the water, while the other end is brought over the side of the bottle. This, acting as a siphon, causes a continual dropping upon the part to be irrigated. In injuries of joints this process, which abstracts heat gradually and without disturbance of the part, is most valuable. Irrigation of the shaven scalp in cases of meningitis is carried out by applying a cap of india-rubber or coil of tubing over the head and back of neck, so arranged that a current of cooled water may flow continuously through it. Where pounded ice is applied to the head in a bladder, this should be suspended by a string from the bedstead, so that the head of the patient may not have to sustain the weight of the bag and its contents. Five ounces of sal ammoniac and five ounces of nitre in a pint of water will form an

efficient cooling mixture, which can be applied in a bladder when ice is not at hand. The ice-bag, and cold-water compresses renewed every three minutes, have been used as an appliance to strangulated hernia, and to prolapsed rectum, to reduce the volume of the part and so facilitate reduction. Care must be taken that the cold application be not continued so long as to cause gangrene. Cold wet compresses should not be applied over dry bandages with which wounded or broken parts are secured. Several cases are recorded where a hand or arm has become gangrenous in consequence of having been bound up with dry bandages, and then treated with cold-water compresses. The dry bandages, as they become wet, contract tightly on the limb and stop the circulation. Ice-bags must be carefully watched to see that no injurious effect occurs.

A mixture made of spirit of wine and water, or of eau de Cologne and water, is a simple form of *cooling lotion*. The spirit evaporates and so carries off heat from the surface. One fl. oz. of rectified spirit to 15 of water makes a good spirit-lotion, and the addition of 4 drachms of nitrate of potassium, or chloride of ammonium, will add to its cooling and sedative effect. 4 drachms of the chloride of ammonium with half a fluid ounce of diluted acetic acid, and the same quantity of rectified spirit, in 15 fl. oz. of camphor water, is another form for a very serviceable lotion. These lotions, applied by means of a piece of soft rag or lint over the skin, act as refrigerants, cooling the head when it is hot or painful. Cold water, and cold lotions of vinegar and water, are familiar means for stopping hæmorrhage.

In cases of severe uterine hæmorrhage, *injections* of ice-cold water into the vagina, or into the rectum, frequently succeed in checking the bleeding. If the cold injection fail, hot water (110°–120° F.) may be tried in its place. In cases of bleeding internal piles, an injection of cold water, after the action of the bowels, braces the parts and constricts the bleeding vessels. Iced water has been used in the form of antipyretic enemata.

3. Dry Cold. Uses of Ice.—Professor Esmarch, in cases of fracture, and in various forms of traumatic inflammation, has applied ice for periods of twenty or thirty days with the best results. Minor cases of bruise with inflammation may be treated by cold employed in the form of a common bottle filled with cold water and kept pressed against the part. After operations upon the eye, the extraction of cataract, for example, a small ice-bag is very useful in relieving pain and keeping down inflammation.

Ice-bags placed along the course of the spine have been found effectual remedies in many forms of nervous disorder. In cases of epilepsy, where the circulation is sluggish, the hands and feet being always clammy and cold, an india-rubber bag of ice applied along the spine has been found to restore warmth, at the same time relieving headache and symptoms of incipient paralysis. Cold to the spine is asserted by Dr. Chapman to lessen the excitomotor power of the cord. In the severe pain of an inflamed ovary or testicle ice in a bag may often be employed beneficially as an anodyne.

Lumps of ice swallowed are invaluable in arresting hæmorrhage from the throat and stomach. In tonsillitis and diphtheria a small piece of ice in the mouth tends to reduce inflammation and cool the

throat of the patient. Obstinate vomiting can often be checked by swallowing fragments of ice.

Cold as an Anæsthetic.—This is now almost superseded by the use of cocaine (*see* ANÆSTHESIA, Local), but is occasionally employed in the form of a spray of ether. When the jet is turned on to the skin, a marked degree of local anæsthesia is produced, but not enough for surgical purposes. By driving over the ether under atmospheric pressure, however, the surgeon can produce cold even 6° below zero; and by directing the spray upon a half-inch test-tube containing water, he can produce a column of ice in two minutes. Such operations as the removal of small tumours, opening abscesses, and inserting sutures may be thus painlessly performed.

JOHN C. THORWOOD.

COLEY'S FLUID.—*See* ERYSIPELAS, Curative.

COLIC.—SYNON.: Fr. *Colique*; Ger. *Die Kolik*.

DEFINITION.—Originally having reference only to pain in the large intestine (*τὸ κόλον*, debased derivative *καλίκος*), the term colic has been extended in a vague but useful way to include all pains in the abdomen which have the following characters:—The pain is usually severe, and is described by the epithets sharp, griping, cutting, tearing, stretching, or twisting. It is paroxysmal, either completely ceasing at intervals, or rising and falling in severity. With a few exceptions it is not attended with pyrexia. In most instances, if not in all, it is due to violent and irregular contraction of unstriated muscular tissue.

ÆTIOLOGY.—Pain as thus defined may have its origin in stomach, intestine, gall-bladder and bile-ducts, kidney and ureter, and pancreas.

1. Pain arising from disease of the stomach seldom has the characters of colic. But colic may occur in cases of pyloric obstruction for any cause, when the stomach-wall is hypertrophied, and also, though rarely, in association with hyperchlorhydria, in which condition it is possible that the pain is due to pyloric spasm. The gastric crises of *tabes dorsalis* may be included under this head.

2. In the intestine colic occurs under many conditions. A common cause is lead-poisoning, which affords the best example of the characteristic pain due to spasm of portions of the bowel. Brass-workers also are liable to colic (*see* BRASS-POISONING). Similar pain is produced by the violent peristalsis which is excited by the presence of hard fecal matter, undigested food, foreign bodies, or flatus in large quantity. In all forms of intestinal obstruction the pain, in an early stage at all events, is of the nature of colic. It is produced by many purgatives. It occurs also in inflammatory conditions of the bowel, in the enteritis due either to the ingestion of irritating or indigestible material, unripe or over-ripe fruit, or decomposing food (*see* POISONOUS FOOD), or to exposure to chill; in the enteritis of children often produced by improper feeding; in dysentery and ulcerative colitis, in cholera (Asiatic and English), and in mucous colitis. Attacks of colic may occur in the neighbourhood of the right iliac fossa without pyrexia, and these have been attributed to spasm of the wall of the vermiform appendix excited by a concretion or an accumulation of mucus. Typical colic also occurs in Henoch's form of purpura, possibly produced by hæmorrhage into the bowel-wall (*see* PURPURA). The nervous system also may be a factor. Thus intestinal pain is sometimes a manifestation of the hysterical condition, and severe attacks may occur

in association with angio-neurotic cedema (*see* ANGIO-NEUROTIC EDEMA). It is possible also that gout and rheumatism may favour the occurrence of colic, though there is no justification for considering either of these blood-states as an immediate cause.

3. Biliary colic is a common and severe form. It is due to the passage or impaction of a gall-stone in the cystic duct or common bile-duct.

4. Renal colic is equally common and severe. In its typical form it is produced by the passage of a calculus down the ureter, but in many cases of renal colic the calculus is too large for descent, and it remains in the pelvis of the kidney or perhaps becomes engaged in the commencement of the ureter. It must be remembered that a pain which closely simulates that of a true renal colic may be of the nature of neuralgia, and that similar attacks—'Dietl's crises'—are sometimes associated with a movable or floating kidney.

5. Pancreatic colic, a rare occurrence, is produced by the passage or impaction of a calculus in the pancreatic duct.

SYMPTOMS.—Pain having the general characters of colic thus occurs as a symptom of a large number of disorders, but the special characters belonging to the different groups must be recognised. (1) Gastric colic is localised in the epigastrium. In cases of pyloric obstruction it may be clearly paroxysmal, and peristaltic waves passing from left to right along the stomach may be visible, or gently rounded eminences may appear and disappear. In hyperchlorhydria any pain of the nature of colic is uncommon, but a severe spasmodic pain is occasionally met with, which is apparently independent of organic disease of the stomach. Such attacks are usually felt to the left of the middle line, and they may be located under the left costal margin near the apex of the heart. (2) Intestinal colic is most commonly felt in the umbilical region, but it may shift from place to place, as the bowel moves and as different parts become affected. The following two classes must be recognised:—(a) *Lead-colic*.—The paroxysms of pain are severe and the patient may writhe and cry aloud. Between the paroxysms and after the subsidence of all acute pain, there remains a feeling of soreness or dull aching which is frequently limited to one side of the abdomen. Sometimes relief is obtained by pressure, but in severe cases the abdomen often becomes too tender to be handled. As a rule the abdominal wall is rigid and retracted, and peristalsis is not visible. Obstinate constipation is the rule, and an action of the bowels, if obtained, is not necessarily a signal of relief. Flatus, however, may be passed either naturally or after an enema. Vomiting is usually associated with the height of the attack. The amount of urine is diminished. The face is commonly pale and expressive of suffering, and perspiration often breaks out after a paroxysm of pain. Pyrexia is exceedingly rare. The pulse is seldom quickened, and it is often diminished in frequency: it may be enfeebled, but sometimes its tension is raised. The radial pulses and the pupils are said to be often unequal. There may be a history of previous colic, other evidence of lead-poisoning may be present, and the characteristic blue line on the gums will certainly be observed. A lead-colic with hourly or even more frequent paroxysms of pain, obstinate constipation, and vomiting, may persist for some days or a week or more, but it yields to treatment and need not give rise to anxiety. *See* LEAD-POISONING.

(2) When due to the presence of hard fecal matter, flatus, or indigestible material, the colic has the same characters as when due to lead, but it is far less severe and relief is quickly obtained by an action of the bowels or even by the passage of flatus. Vomiting is uncommon. The pulse is little, if at all, affected. The abdomen tends to become distended rather than rigid and retracted, and the distension of some portion of the bowel, especially the cæcum, may often be seen or recognised by its tympanitic note on percussion. There is sometimes constipation which requires energetic treatment, but there is often diarrhoea, and such cases pass under the heading of enteritis. In the case of infants, colic is manifested by restlessness, screaming, drawing up of the legs, rigidity or distension of the abdomen, and vomiting, and there is usually constipation with an occasional attack of diarrhoea, or an excessive discharge of uric acid in the urine.

Appendicular colic is usually limited to the right iliac fossa, but it is sometimes felt above this region and in the right flank. It may be attended with vomiting, but there is no pyrexia. Tenderness at 'McBurney's point' is commonly present.

(3) In biliary colic the pain is often accurately localised below the right costal margin in the region of the gall-bladder, but it may be more widely diffused over the right hypochondrium and lower thoracic region and about the angle of the right scapula. Sometimes it extends over the epigastrium or up to the right shoulder and in rare cases even to the left shoulder. The pain is usually more intense than in intestinal colic, and may be agonising. It is attended with vomiting, sweating, and collapse. Often there is shivering, and this is sometimes associated with a rise of temperature to 103° F. or more, so that a suspicion of malaria may arise. There is often tenderness on deep pressure under the right costal margin, and a distended gall-bladder may be felt. Jaundice occurs if the common bile-duct is blocked, and search should be made for gall-stones by careful sifting of the stools. Such an attack of pain may last for an hour or so, and may not be repeated; or a series of attacks may occur with but slight intermissions for a week or more, and the association of such continued pain with severe vomiting may in itself be a danger in weakly individuals.

(4) A renal colic commences in the flank, and, in typical cases associated with the actual descent of a calculus into the bladder, it may be observed to pass down the course of the ureter and is felt in the testicle and inner side of the thigh. In other cases when the calculus does not leave the pelvis of the kidney, the pain remains more nearly limited to the flank occupying both anterior and posterior aspects, so that if further evidence is wanting it may be difficult on the right side to distinguish it from a biliary colic. In rare cases the pain has been referred to the opposite kidney. The pain is as intense as in biliary colic. It is similarly accompanied with vomiting, sweating, shivering, and collapse, but pyrexia is less common. Micturition is usually frequent and may be painful, and the urine often contains blood. The affected flank is tender. An attack may last for an hour or it may continue for a day or two. Sudden relief is sometimes experienced as the stone passes into the bladder. The influence of bodily movement in precipitating an attack of renal colic may sometimes be noted. If a calculus remains in the kidney, the

presence of albumen, pus, blood, and crystals of uric acid or calcium oxalate in the urine may be an important aid in diagnosis. *See* RENAL CALCULUS.

(5) The pain produced by a pancreatic calculus is deeply situated in the epigastrium, with a tendency to radiate towards the spine and the left scapula. It may be indistinguishable from biliary colic, but in comparison with this it is exceedingly rare. As a rule no other symptoms are present, but if secondary change is set up in the pancreas there may be wasting with loose stools containing fatty acids, or glycosuria. By blocking of the duct a cyst may be subsequently developed. *See* PAIN IN VISCERAL DISEASE.

DIAGNOSIS.—Colic is thus a symptom common to many morbid conditions. Of many of these disorders which have been already enumerated, the nature is obvious. But in others the pain may be for a time the only symptom, and it may be difficult to ascertain its origin. The distinction between these five forms of colic is made (1) from the previous history of the patient, (2) from the position of the pain, (3) from the presence or absence of other signs or symptoms pointing to gastric, intestinal, biliary, renal, or pancreatic disease. The occasional difficulty in distinguishing between a biliary and a renal colic in the right side has been mentioned. Difficulty may also be experienced in the case of appendicular colic, when the pain is not accurately localised in the right iliac fossa (*see* APPENDICITIS). In gastric pain the existence of *tabes dorsalis* in an early stage must never be overlooked. The association of colic with constipation and the passage of mucus forms a definite type of disease (*mucous colitis*) (*see* COLITIS, 6. *Mucous Colitis*). The most important point, however, in view of the necessity of early surgical interference is the distinction between the various forms of colic, especially simple intestinal colic, and the early pains of intestinal obstruction. In many cases neither the history of the patient nor the position of the pain serves as a reliable guide, and a diagnosis of intestinal obstruction must rest on greater constitutional disturbance, rapid pulse, dryness of the tongue, facial expression, absolute constipation, more urgent vomiting, greater tendency to distension of the abdomen, and visible peristalsis. On the other hand the passage of flatus in non-obstructive forms of colic must be remembered. In infants showing colic, the possibility of intussusception must be borne in mind, and the occurrence of frequent straining, the passage of blood, and the presence of an abdominal tumour should be recognisable.

TREATMENT.—In severe colic from any cause, opium or morphine (hypodermically) must be used, and in most cases this may be supplemented with advantage by the local application of warmth. This treatment, however, is directed merely to the immediate relief of pain, and it has limitations. The origin of the pain must be ascertained, and so far as possible the primary disease, whether gastric, intestinal, biliary, pancreatic, or renal, must receive appropriate treatment. More particularly in an intestinal colic whether due to lead-poisoning or to irritating or indigestible material, though morphine is often necessary and beneficial, attempts should be made from the first to obtain a free action of the bowels. For this purpose an ounce of castor oil guarded by the addition of 15 minims of tincture of opium should be given and repeated if necessary, or,

if vomiting interferes with this, enemata should be used. But cautious observation is required if there is any ground for suspecting the existence of intestinal obstruction.

HERBERT P. HAWKINS.

COLITIS.—SYNON.: Fr. *Colite*; Ger. *Entzündung der Schleimhaut des Kolons*.

DEFINITION.—Under the heading colitis are collected a large number of diverse conditions, which are loosely linked together by the presence of some degree of inflammation of the colon. Six groups may be recognised.

1. Catarrhal Colitis without ulceration.

(a) The early stage, or it may be one distinct form, of tropical dysentery falls under this head. (b) Apart from this specific affection a simple colitis is uncommon, but in cases which are described under the term 'enteritis,' and which have the same causation as that condition, the colon is often affected as well as the small intestine. In some such cases it may be inferred from the situation of the pain and tenderness that the colon is indeed the chief seat of the trouble. This form is not uncommon in measles; and persons who have suffered from true dysentery are particularly liable to attacks of simple colitis. The main symptom is diarrhoea, which may be accompanied by the passage of blood and mucus. There is gripping pain, tenderness in some part of the course of the colon, a furred tongue, and sometimes pyrexia. The condition is seldom severe, and a quick recovery in all but the dysenteric cases is the usual termination. (c) A similar colitis may be set up by the medicinal use of mercury and arsenic in large doses. (d) The diarrhoea which is apt to occur in association with septicæmia, pyæmia, chronic nephritis, and more rarely pneumonia, may arise from colitis, and after death the mucous membrane of the colon may show hyperæmia, swelling, and hæmorrhages.

2. Ulceration of the Colon.—(a) Inflammation and ulceration of the colon occur in typhoid fever and dysentery, and as a result of tuberculosis and carcinoma. (b) The colitis of septicæmia, pyæmia, chronic nephritis, and pneumonia may pass on to the formation of ulcers. (c) In chronic nephritis, more particularly in the interstitial form of the disease, ulcers occasionally occur both in colon and in small intestine, when there is but little appearance of a general colitis. Such ulcers may possibly arise from hæmorrhage into the submucous tissue and subsequent necrosis of the mucosa. The importance of this condition lies in the fact that it may be the cause of death by perforation and peritonitis. (d) Ulceration, especially in the cæcum, is a common occurrence when the bowel is much distended as a result of long-continued obstruction. In such cases perforation readily occurs, and the symptoms of obstruction are then replaced by those of a general peritonitis. (e) Ulceration arising in the solitary follicles is occasionally found in the *post-mortem* room in cases of severe intestinal disease of any kind. Such a follicular ulceration is probably a late phenomenon, and it has no clinical importance. (f) Rare cases have been recorded of ulceration of the colon in connection with chronic cerebral or spinal disease, and it is possible that it is dystrophic in origin. (g) Ulcers arise also by vascular disturbance. Thus the venous hyperæmia which occurs in cirrhosis of the liver may lead to ulceration both of large and small intestine. Em-

bolism, thrombosis, and atheromatous narrowing of mesenteric arteries are also rare causes, but they are apt to produce gangrene of the bowel-wall rather than localised ulceration.

3. Ulcerative Colitis.—This affection requires separate description as being a well-defined morbid entity, for which this general name must be retained until its specific cause has been discovered.

ÆTIOLOGY.—No specific cause of ulcerative colitis is known, but its close resemblance to amoebic dysentery is suggestive. It occurs in temperate climates, in town and country; and it has no apparent relation with water-supply, locality, or season. Both sexes are liable, females perhaps more so than males, and though cases have been known under the age of 20 and over 50, it generally occurs during middle life.

SYMPTOMS.—The first and most marked symptom is diarrhoea. The bowels are open three or four times in the day, sometimes much more frequently. There may be short periods of constipation in an early stage, during which the stools are partly formed, and this effect may sometimes be produced by treatment, but as a rule the motions consist of brown offensive fluid with little or no solid fecal matter. Blood is commonly present, either just reddening the fluid, or in small clots, or in large amount, and hæmorrhage has been known to be the immediate cause of death. Mucus is not commonly seen except in an early stage. There is seldom the rectal pain or tenesmus which is met with in dysentery, but abdominal pain of a gripping character is a common feature and it may be very severe. It is generally connected with an action of the bowels, but is sometimes more constant. The tongue is covered with dirty white fur, and in a late stage becomes dry. The temperature may be normal throughout, but occasionally there is a moderate pyrexia. The abdomen is often slightly distended and sometimes tender; and owing to the thinness of its wall peristalsis is often visible. An ulcer may sometimes be felt *per rectum*. There is progressive emaciation, anaemia, and cardiac failure, and death occurs from exhaustion, or more rarely from hæmorrhage or perforation and peritonitis. Hepatic abscess is rare. In many cases a fatal event is reached in two months. Sometimes, however, the disease undergoes temporary abatements, so that the history may extend over a year or more, but it is doubtful if a permanent recovery ever occurs.

MOREID ANATOMY.—The ulceration is usually found to be extensive, so that the appearance to the naked eye does not differ from that of dysentery. Ulcers having muscular tissue for their floor may be found in the whole length of the colon (rarely in the small intestine), the remaining mucous membrane being represented by islands or polypoidal tufts of oedematous tissue, of which the edges are often somewhat undermined. Less commonly the ulceration may be limited to a part of the colon, or a single large ulcerated patch may be found. If opportunity arises for examination in an early stage, general hyperæmia and swelling of the mucous membrane are found, and the appearance of the ulcers at this time suggests that they arise by a necrosis of this layer secondary to the accumulation of inflammatory products in the submucosa. The liver is generally fatty, the cardiac muscle is often pale and soft, and in many cases some degree of chronic interstitial nephritis is present.

4. **Ulcerative Colitis of Asylums.**—It is not certainly known that this disease is distinct from the sporadic ulcerative colitis already described, but it deserves separate notice from its importance in relation to the treatment of the insane. Until the specific causes of these affections and of tropical dysentery are known, it is impossible to adopt a scientific classification.

ÆTIOLOGY.—The disease occurs in epidemics in large asylums, and in some of these institutions it is endemic, a few cases occurring every year. As a rule the patient attacked has been an inmate of the asylum for some years, but he may fall a victim within a few days of admission if an outbreak of the disease is in progress at the time. Both sexes are affected, and though it occurs at any age the average age of patients attacked is about 50. The disease shows no relation to any special form of mental derangement, but the feeble are more liable to it than the strong, and the general health of the patient is an important factor. Some observers have noted a special prevalence during late autumn and early winter or in cold weather. Medical officers and attendants are very rarely affected, but nurses whose duty it is to administer rectal injections are occasionally developed colitis.

Bearing on the question of infectiousness, there is strong evidence to show that diminution of the disease has followed the adoption of measures ensuring the isolation of patients and the complete disinfection or destruction of excreta and soiled linen. No specific cause, protozoic or bacterial, is known, but suspicion has been thrown on *Bacillus enteritidis* sporogenes and on a virulent form of *Bacillus coli* communis. The origin of the disease is obscure. Some epidemics have been clearly connected with defective sanitation. A localised outbreak has been known to occur in one block of a large asylum and to cease abruptly upon the discovery and repair of a leaking sewer placed in a subway which was in communication with the ventilating chamber of the wards affected. On the other hand, in some instances no sanitary defect has been discovered, and the disease has made its appearance in the newest and most perfect asylum, and has there accounted for 6 per cent. of the deaths. There is no evidence to connect it with impure water, milk, or food.

SYMPTOMS.—In its clinical characters, at any rate during an epidemic, this disease bears a closer resemblance to tropical dysentery than does the sporadic form of ulcerative colitis. In isolated cases an initial diarrhoea which may attract little attention is a common feature, and in this stage the stools contain fecal matter. But during an epidemic the onset is more sudden and severe, and the stools from the first consist of a thin offensive fluid with blood and mucus and occasionally sloughs. Vomiting often occurs at the commencement. There is considerable abdominal pain and sometimes tenesmus. A rigor may mark the onset of the disease, and there is commonly a moderate pyrexia throughout. Hepatic abscess is of occasional occurrence, but has not the frequency met with in tropical dysentery. Pneumonia, lobar or lobular, is a common and grave complication, and the kidneys are often found to be cirrhotic, as is the case in sporadic ulcerative colitis. Perforation is rare. The mortality varies between 15 and 75 per cent., and it is thus less fatal than the sporadic disease. On the other hand, it is usually much more acute: an attack may prove fatal in a few

days, and is often fatal in two or three weeks. Some cases, however, run on for many months before death occurs.

MORBID ANATOMY.—In cases which die in an early stage the mucous membrane of the colon is found to be uniformly reddened, swollen, and covered here and there with a layer of blood-stained mucus. Membranous patches may be present which consist of inflammatory exudation and necrosed superficial tissue. In later stages ulcers of varying size and depth are found in all parts of the colon, especially at the flexures, and the undermining of their edges is very marked. The small intestine is simultaneously affected with much greater frequency than in the sporadic disease.

5. **Croupous or Diphtheritic Colitis.**—By this term is meant an inflammation of the colon which leads to the formation of a membrane upon the mucous surface, consisting of a fibrinous exudation and of necrosed mucosa. Such a membrane may be present here and there in the form of scattered flakes seated upon a reddened base, or it may be a thick continuous grey sheet covering many square inches of the mucous surface, and in such cases the whole thickness of the bowel-wall may be swollen, reddened, of doubtful vitality, or actually gangrenous.

Such a condition occurs, though rarely, as a primary disease. Its cause is unknown, though it is reasonably attributed to bacterial agency. The symptoms have a close resemblance to those of intestinal obstruction or peritonitis. The onset is sudden, being marked by abdominal pain and distension with vomiting. There is generally absolute constipation, though flatus may be passed. Collapse sets in and rapidly ends in death.

Much more commonly the condition is found unexpectedly after death from such diseases as septicæmia, pyæmia, pneumonia, and chronic nephritis, in connection with which simple colitis and ulceration have been already mentioned. It may be caused also by severe abdominal injury and by the swallowing of corrosive fluids, and it has been met with in diabetes mellitus, and in association with carcinoma of the colon. It is marked by diarrhoea and occasionally by the passage of blood, but arising as it usually does in a late stage of other diseases it often escapes attention.

6. **Mucous Colitis.**—The name 'mucous colitis' is given to an affection which is characterised mainly by the passage of mucus in large quantity.

ÆTIOLOGY.—It occurs most commonly in adults between the ages of twenty and forty, and it is far more common in women than in men. It is by no means rare, but it is not often seen in hospital practice. The patients affected are usually nervous and excitable, with a tendency to dyspepsia and neurasthenia, and the disease is apt to lead them to depression and hypochondriasis. It is frequently associated with catarrhal appendicitis. Its nature is unknown, but from its long course, its constant association with constipation, and the negative character of the few observations that have been made, it is perhaps more reasonably considered as a nervous disturbance than as an inflammatory affection of the bowel.

SYMPTOMS.—In the best marked examples the patient who has commonly suffered from constipation for many years experiences definite attacks of abdominal pain and passage of mucus, which may last a week or more, and which may appear two or

three times a year or may recur at such short intervals that life becomes a burden. The pain is of a gripping nature and may be very severe. It is usually worse before and during the act of defæcation, the completion of which often seems to afford relief. It may be referred to any part of the abdomen, and it is often felt in one or other of the iliac fossæ. If the pain and tenderness are limited to the right iliac fossa, a close resemblance to appendicitis is established, and in many cases both conditions are present. The abdomen is often somewhat distended, and the site of the pain may be tender on pressure. Sometimes there is nausea and even vomiting. Pyrexia is rarely noted. The mucus may be passed in pellets or masses, but sometimes it is discharged in the form of tough translucent tubular casts of the bowel from an inch to a foot in length, which may be unfolded under water and consist of mucus embedding degenerate epithelial cells, fæcal particles, and phosphatic crystals. Occasionally a little blood is passed. Such an attack slowly subsides, and the patient returns to the previous state of comparative health marred by constipation, anæmia, and listlessness or depression.

More commonly, however, the attacks are not so sharply defined, but even in these slighter cases the association of constipation with pain and the passage of mucus are to be recognised by careful observation. On the other hand, rare cases have been recorded, in which the attacks have been so frequent and severe that the resulting exhaustion has led to death.

Mucous colitis is always intractable. It certainly may disappear, but it often continues for many years unchecked by any form of treatment.

TREATMENT.—A simple colitis should be treated on the lines laid down as regards enteritis. But in the cases which occur in those who have had dysentery it is advisable also to wash out the large bowel daily with two or three pints of saturated boric-acid solution at a temperature of 98° F.

The treatment of ulcerative colitis is unsatisfactory. Absolute rest in bed, with a diet mainly consisting of milk, is essential. No specific cure is known, and bismuth subnitrate in twenty- to thirty-grain doses, with enough opium or compound ipecacuanha powder to control the pain and diarrhoea, seems to give the best results. If a patient is seen who presents undoubted symptoms of the disease in an early stage, it is well to consider the propriety of making an artificial anus above the cæcum, a step which gives rest to the diseased portion of the bowel and allows of thorough flushing with boric-acid solution or creolin.

In 'asylum-colitis' the same medicinal treatment may be employed, or a mixture containing salol, salicylate of bismuth, and compound ipecacuanha powder may be used, in conjunction with large rectal injections of a solution of boric acid. In view of the rapid exhaustion, every effort must be made at the outset to maintain the patient's strength by suitable food and alcohol.

In mucous colitis the main efforts must be directed to the improvement of the general health. A simple and regular diet must be arranged, from which there should be no departure. The plan of life must be as healthy as possible, and companions and surroundings must be bright and cheerful. A change of scene and absence from home is often beneficial. In some cases a course of massage, with tonic treat-

ment, will be of use, and in all cases every effort must be made to lessen the constipation without the use of strong purgatives. There is evidence to show that bismuth salicylate and salol are of some service in diminishing the frequency of attacks. During a severe attack the pain must be combated by hot fomentations, and belladonna may be administered, but the use of opium in such patients is to be avoided, as its discontinuance will be difficult. Small doses of castor oil are of use in aiding the expulsion of mucus, and large injections of boric-acid solution at 98° F. administered by gravity may prove beneficial.

HERBERT P. HAWKINS.

COLLAPSE.—**DEFINITION.**—Collapse is a condition of extreme depression of the nervous system, especially of the cardiac and respiratory centres. It is usually due to powerful afferent stimuli, but may be produced by slighter impulses, if preceded by conditions tending to exhaustion. Its chief manifestations are those of mental and physical prostration.

RELATION TO SHOCK.—The terms 'collapse' and 'shock' are often used indiscriminately in reference to conditions of sudden prostration due to injury of any kind. They are not, however, synonymous terms. *Shock* is essentially a reflex vaso-motor paralysis with cardiac inhibition, the effect being produced through the vagi, depressor, and other nerves. The venous system thus becomes intensely congested, the condition being virtually equivalent to the occurrence of an internal hæmorrhage. The output of blood from the heart is diminished, and the resulting diminution of arterial pressure causes acute arterial anæmia, in which the brain, muscles, skin, &c., partake. Combined with this anæmia is inhibition (i.e. slowing) of the heart's action, and lessened frequency of the pulse. But the flow of blood from the veins into the heart being deficient, the pulse, instead of being infrequent and full, becomes infrequent and weak. Thus the heart has less work to perform as measured by output, and its pause or recuperative period is longer; hence it does not become exhausted. In *collapse*, failure of the heart and respiration of central origin is the essential feature, as shown by the frequent weak pulse and shallow breathing. Shock may precede, merge into, and terminate in collapse, or the latter may result directly from the cause of the shock if it is prolonged; but shock is neither a necessary antecedent nor an invariable accompaniment of collapse.

PATHOLOGY.—When an intense stimulus of a certain nature reaches the cardiac and respiratory centres in the medulla, it sets up such a profound disturbance as to cause either immediate death or intense depression of the centres; consequently, a sudden breakdown occurs in the functions of the circulatory and respiratory organs. Such impulses, however, are not confined to these centres, but are diffused, involving all the central nervous system in varying degrees; hence, all the bodily functions are demoralised, or may be completely destroyed.

If the centres have been previously subjected to overwork, or have been insufficiently nourished, the disturbance which results from a given afferent impulse will be more profound, and a slighter impulse will be necessary to produce a serious disturbance. Cerebral anæmia, resulting from hæmorrhage or any other cause, will therefore tend to increase the effect of a given impulse and will retard recovery of the centres from their exhausted condition.

The cause of the collapse may at the same time cause cardiac inhibition or vaso-motor breakdown, but neither of these conditions is essential to the production of collapse; they tend to prolong the condition or to intensify it by retarding the recovery of the nervous centres. Owing to the exhaustion of the various centres in the brain, the heart's action is weakened; respiration is feeble and shallow, sensation is blunted, voluntary motion is in abeyance, and a fall in animal heat takes place as a consequence of the diminished metabolism. The rapid action of the heart proves that its action is not usually inhibited; it is rather the cardio-inhibitory centre which is paralysed.

ÆTIOLOGY.—The chief cause of collapse is *injury* in some form or other, and the intensity of the condition varies with: (1) The nature and severity of the injury; (2) the importance of the part injured; (3) the nervous susceptibility of the person injured; and (4) the anticipation of, or unexpectedness of, the injury.

1. The *severity of the injury* is dependent upon both its superficial extent and its depth. Thus, if the lesion involve an extensive area it affects numerous nerve-terminals, which are especially sensitive parts of the nervous mechanism; while, on the other hand, the greater the depth the greater must have been the violence applied.

The nature of the instrument used and the character of the injury are important factors: thus, a large superficial burn or scald, especially of the trunk, or head and neck, results in severer symptoms than complete incineration of a limb. Severe lacerations, crushes, and gunshot wounds cause more disturbance of function than incised wounds. In cases of amputation collapse is greater the closer the site of severance to the trunk, as thereby a greater number of nerve-fibres are injured.

2. *The importance of the part injured.*—Injuries to the brain and spinal cord result in the most profound collapse, but the symptoms due to the local lesion are of such importance that the condition is not so described. In other cases, the more essential to life the organ injured, the nearer it is to the cerebro-spinal axis, and the closer its connection with the sympathetic system, the greater is the collapse produced. Injuries to the heart, vagi, and abdominal viscera, as well as crushing of joints and of the testicle, result in intense collapse.

3. *The Nervous Susceptibility of the Individual.* Age, race, sex, occupation, temperament, moral tone, previous ill-health, the presence of pain, and idiosyncrasy, are all factors which modify the effect of injury. The greater the culture and the more 'highly strung' the individual, the greater will be the liability to collapse. Outdoor occupations and constant exercise tend to nullify the effect of injury; indoor occupations and sedentary living to increase it. Experience shows that criminals of a low type bear severe operations and injury with equanimity and fortitude; and that members of white races suffer severe collapse from injuries which would affect savages, negroes, and certain Eastern tribes but slightly. The very young and the aged are especially liable to suffer from collapse. Idiosyncrasy is shown in the collapse which in some persons follows the passage of a urethral bougie or small operations upon the urinary tract.

Previous good health, and exultation and success at the moment of infliction of the injury, as opposed to dejection and defeat, tend to prevent the occur-

rence of collapse. Any cause, in brief, which tends to good health and against exhaustion will decrease the injurious effect upon the vital centres.

4. The *anticipation* of an impending injury really increases the nervous susceptibility of the person, and tends to exhaustion.

Besides traumatism arising from without, certain conditions, such as the passage of gall-stones and renal calculi, torsion of the pedicle of an ovarian cyst, rupture of the sac of an ectopic pregnancy, and lesions involving the abdominal organs, such as strangulated hernia, intussusception, and acute peritonitis, cause severe collapse. Certain poisons, such as tobacco, ptomaines, and other gastro-intestinal irritants, may also produce this condition: it is further met with in many acute diseases, such as cholera. Intense and prolonged pain will of itself produce collapse.

The main causes of collapse may be classified as follows:—

(a) *Cardiac Causes.*—These include (1) injury to the heart by gunshot, stabs, or fractured ribs; (2) injury to the vagi or their main branches by blows, wounds, and operations on the neck or larynx; (3) lesions of the cardiac centre in the medulla, produced by gunshot, fractures of the skull, &c.; and (4) direct paralysis of the cardiac muscle by inhalation of excessive doses of chloroform.

(b) *Respiratory Causes*, such as (1) injury to the thorax, producing collapse of the lung; (2) lesions involving the respiratory mechanism, such as injury to the phrenic nerves; (3) injuries affecting the respiratory centre in the medulla; (4) mechanical interference with the exchange of gases; and (5) excessive doses of anæsthetic drugs arresting respiratory action.

(c) *Excessive hæmorrhage*, especially venous. Hæmorrhage may also be a cause of collapse, independently of the amount of blood lost, and therefore apart from the production of cerebral anæmia, as is seen in cases of slight intraperitoneal hæmorrhage.

(d) *Vaso-motor Causes*, such as transverse section of cord, and division of the splanchnic nerves.

SYMPTOMS.—These vary with the severity of the collapse. All degrees are seen, from the trifling amount met with in acute febrile diseases, manifested by peripheral coldness, to the most intense form where the patient scarcely differs in outward semblance from a dead body, and vitality is reduced to a minimum. In a severe case of collapse the expression is anxious and dull; the face is deathly pale and drawn, with well-marked furrows; the eyes may be sunken and the tissues relaxed. Consciousness is often unimpaired: it may, however, be hazy or seemingly benumbed, but if pressed the person will usually respond to questions in a coherent manner. The voice is weak and low, and may be slightly hoarse. The skin is cold and clammy: the forehead especially may be moist; the amount of sweating is said to be increased if there be hæmorrhage.

The eyes are dull and glassy, staring or languidly rolling about, giving a superficial impression that the person is looking about the room intelligently. The conjunctivæ may be insensitive. The pupils are usually dilated and react sluggishly to light; in very severe cases they may not react at all. The *pulse* may be imperceptible and the heart's action inaudible. When present the pulse is *frequent* and weak, often irregular and thready. Respiration

is shallow, feeble, slow, and irregular, with occasional sighing or gasping inspirations. The nostrils are dilated and quivering. The temperature is usually slightly subnormal (98° to 96° F. or lower); the fall is greater if there be much hæmorrhage, and in elderly persons.

The patient lies on his back making no voluntary effort, and if a limb be raised from the bed it falls back again as if dead. The sphincters may be relaxed and the urine and feces voided involuntarily; or there may be retention of urine.

The temperature of the internal organs varies; collapse may occur along with high internal temperature, as in Asiatic cholera, or the converse may obtain, as is frequently seen in fatal cases.

In these the pulse and respiration get gradually worse, and death ensues from pure asthenia. If recovery occurs a modified stage of reaction appears, when the general condition improves, and there may be vomiting and slight rise of temperature: this stage is best marked in cases associated with hæmorrhage or vaso-motor disturbances. For treatment and differential diagnosis see SHOCK.

P. L. DANIEL.

COLLAPSE, Pulmonary.—A condition in which the lung is more or less devoid of air. See LUNG, Collapse of.

COLLIQUATIVE NECROSIS (*colliqueo*, I melt) is the name given to the process by which certain cells in dying break down into a fluid material; the most typical instance is seen in cerebral softening due to vascular occlusion. It is here due to a simple retrogressive change after death of the cells, but it may also be produced by the action of bacteria, as in some forms of gangrene.

COLLOID (κόλλα, glue; and εἶδος, like).—A peculiar product resembling in some of its characters thin glue or jelly, and found associated with cancer and other forms of new-growth. See CANCER; and DEGENERATION.

COLLOID MILIUM.—SYNON.: *Milium Colloid*. Colloid disease, or degeneration of the skin.

DEFINITION.—A very rare disease of the skin characterised by the development of small tumours chiefly round the orbits.

ÆTIOLOGY.—Colloid milium is of equal frequency in the two sexes. It never appears before puberty, and is not associated with any affection of the general health. It is not a disease of the sebaceous glands and has no relationship to common milium, although its original describer, E. Wagner, considered it as a colloid degeneration of Milium-bodies.

SYMPTOMS AND COURSE.—It consists of small cyst-like formations embedded in the skin and varying in size from a hemp-seed to a split-pea; they are usually distinctly yellow in colour, glistening and semi-translucent, while the epidermis over them is unbroken and shows no excretory opening. Upon incision gelatinous material is extruded. The little growths give rise to no subjective symptoms. Their distribution is characteristic:—they are always present in greatest abundance, and sometimes in groups, around the orbits and on the contiguous parts of the cheeks, nose, forehead, and zygomatic regions. More rarely they have been seen on and behind the ears, and even on the neck and arms

(Living). Perrin has observed them on the backs of the hands and on the ocular conjunctivæ.

As a rule they are indolent and persist indefinitely, but sometimes they undergo spontaneous absorption or, on the other hand, suppurate and dry up, leaving very faint pits.

PATHOLOGY.—Payne considers that the colloid change begins in isolated foci deep in the corium, which are nothing else than the degenerated sweat-glands themselves; thence the colloid degeneration extends through the connective tissue to the epidermis. Philippson thinks the degeneration is one of heterotopic epithelial cells, cut off during the development of the corium, and has attempted to demonstrate that pathologically the disease is identical with adenoma of the sudoriparous glands; his views have, however, received no support.

DIFFERENTIAL DIAGNOSIS.—The glistening and translucent characters of the small growths, the gelatinous nature of their contents, as well as their more general distribution, serve to distinguish them from Xanthoma. See XANTHOMA.

TREATMENT.—In most cases no treatment is called for or advisable. Destruction by caustics and electrolysis or removal by erosion or excision may, in exceptional cases, be resorted to, but necessarily leave visible scarring.

J. J. PRINGLE.

COLON, Diseases of.—See COLITIS; and INTESTINES, Diseases of.

COLORADO SPRINGS in Colorado, U.S.A.—A clear, cold, and very bracing climate, with great dryness and diathermancy of the atmosphere; 6,028 feet above the sea. No snow in any quantity, and consequently no snow-melting season. Dry and dusty winds in spring. Camping out at nearly all seasons tolerably safe. A high-altitude station, useful in asthenia and in chronic phthisis, especially with limited lesions and no pyrexia. The name of 'springs' is added from the proximity of Colorado City to Manitou Springs, six miles distant. See CLIMATE, Treatment of Disease by.

COLOUR-BLINDNESS.—A defect of vision, the subject of which is unable to distinguish certain colours. See VISION, Disorders of.

COMA (κῶμα, deep sleep).—A condition of profound insensibility. See CONSCIOUSNESS, Disorders of.

COMA VIGIL (κῶμα, deep sleep; and *vigil*, wakeful).

DEFINITION.—A symptom, or set of symptoms, where continuous sleeplessness is associated with partial unconsciousness.

Coma vigil occurs towards the end of diseases in which the nervous system is involved either directly or indirectly, especially where sleeplessness has been a symptom in the earlier part of the disease. Thus it frequently appears towards the end of an attack of typhus or of delirium tremens, when these are about to terminate fatally.

SYMPTOMS.—The patient lies quiet with his eyes half-closed, inattentive to everything around, but not absolutely unconscious. If the eyelids are touched, they are closed, and perhaps the head is slowly turned away. The eyes have a dull, half-glazed look, and slowly follow any moving object near

them. The pupils are neither much dilated nor contracted, and they move under the influence of light, but very sluggishly. The mouth is generally somewhat open and dry, as are also the lips. The power of swallowing is much impaired: if a small quantity of fluid be put into the mouth, an effort is made after a short time to swallow it, and this effort is for a time successful; but after the symptoms have been present for some time, the effort is so feeble that no result follows. The patient lies mostly on the back; if turned on the side, he either remains as placed, or often slowly turns to the former position. The limbs are occasionally moved a little; and if the hand or arm be raised, a slight resistance is offered. If the bladder or rectum be emptied, there is slight consciousness of the act, as if a feeling of discomfort preceded it. The pulse is quick and weak. The respirations are weak, but otherwise normal. The symptoms continue unbroken throughout, nothing like natural sleep occurring.

PATHOLOGY.—As being little more than a symptom, coma vigil has, strictly speaking, no pathology. It seems to coincide with the gradual suspension through exhaustion of the functions of the nervous centres; the cerebral hemispheres being nearly if not quite inactive, while the action of the rest of the centres is kept up weakly but continuously, till the little remaining nervous power is exhausted, when death ensues. It differs from coma, inasmuch as in the latter the medulla oblongata is the only centre left active, the functions of the rest being entirely suspended. It differs from concussion, inasmuch as the symptoms of the latter attending the temporary unconsciousness are more those of irritation than of pure suspension of function.

DIAGNOSIS.—Coma vigil is distinguished from coma by the presence of a certain amount of consciousness, by the quick pulse, and by the absence of stertorous breathing. It is distinguished from concussion of the brain by the pupil not being contracted, by the history of the case, and by the absence of coldness of the skin and of any sign of shock.

PROGNOSIS.—The prognosis is unfavourable: coma vigil is almost invariably a fatal symptom. It may last from a few hours to three or four days; from twenty-four to forty-eight hours being the most common duration. It may deepen into actual coma; but more usually the symptoms change but little, save that the pulse becomes quicker and weaker, and the respiration more feeble, and death by asthenia then results.

TREATMENT.—Coma vigil does not call for treatment directly itself, but is an urgent indication for nutritive and stimulant measures calculated to relieve the primary disease.

JOHN BANKS.

COMBINED DEGENERATION OF THE SPINAL CORD.—**SYNON.** : Changes in the spinal cord in cases of profound anæmia. Ger. *Combinirten Strängerkrankung des Rückenmarkes*.

DEFINITION.—Under this title a sub-acute disease of the spinal cord has been described in which tracts of different function are concomitantly affected.

ÆTIOLOGY.—The disease most commonly occurs between the ages of 30 and 50, although many cases have been recorded both above and below

these limits. Females appear to be rather more liable to the affection than males, although the difference of incidence on the two sexes is not marked. In some cases the disease has followed acute affections such as influenza: others have been preceded by prolonged diarrhoea or by long-continued suppuration. Syphilis and alcoholism can almost be excluded as ætiological factors. By some authorities anæmia has been supposed to cause the disease. There is no doubt that anæmia often occurs in advanced stages of the disease, but it is by no means always present during the earlier stages of the affection, although in some cases it has been known to precede the onset of the spinal symptoms by several years. The ascertained facts make it probable that the changes in the spinal cord and the anæmia are due to a common cause rather than that the spinal changes are due to the anæmia. In the majority of cases the blood does not show the changes occurring in pernicious anæmia, although in a certain proportion of cases (given by Dana as 10 per cent.) such changes are undoubtedly present.

MORBID ANATOMY AND PATHOLOGY.—No naked-eye changes are visible in the brain or external surface of the spinal cord, or in the membranes, but on section of the spinal cord grey areas of degeneration in the posterior and antero-lateral tracts are easily seen. The morbid changes in the spinal cord are among the most constant features of the disease, varying only in degree with its duration. The stress of the disease falls upon the *mid-dorsal region* of the cord, and on examining sections from this level marked destruction of the white matter is seen all round the periphery of the cord, while the grey matter, and a small area of white matter, immediately surrounding it, remain practically normal. Sections of the cord in the *cervical region* reveal marked sclerosis of the posterior columns, the columns of Goll being more affected than those of Burdach, and there is also sclerosis in the dorsal and ventral cerebellar tracts and to a less degree in the crossed pyramidal tracts; but although the sclerosis is most marked in these tracts it is by no means limited to them, for scattered areas of degeneration are present in other regions of the cord. The degeneration in the posterior columns can be traced up to the nuclei of the posterior column, and in the dorsal and ventral cerebellar tracts into the inferior cerebellar peduncle and velum respectively, although with regard to the latter tract the degeneration can only be traced by the Marchi method which reveals nerve-fibres that are in a recent state of degeneration. The degeneration in the pyramidal tracts can only, as a rule, be traced as high as the middle of the pons, although in exceptional cases degenerate fibres can be found in the internal capsule and corona radiata. Sections from the *lumbar region* of the cord show sclerosis of the posterior columns and of the crossed pyramidal tracts, while in the sacral region the degeneration is almost limited to the pyramidal tracts; but although this is in the main true, even in this region scattered areas of degeneration are present outside the limits of these tracts. The walls of the blood-vessels in the sclerotic areas are thickened, but in the unaffected portions of the cord they show but little change. The grey matter, the cells of the anterior horns, of Clarke's column, and of the cerebral cortex, as well as the anterior and posterior roots, all appear normal, as do also the ganglia of the posterior roots, with the exception of rare instances when changes have

been found in them in connection with the occurrence of herpes or subcutaneous hæmorrhage. In cases that have reached an advanced stage considerable degeneration can be found in the nerves and marked atrophy of the muscle-fibres, although in cases that come to autopsy earlier neither of these changes is present.

Two distinct processes account for the changes found in the spinal cord, (1) a focal destructive lesion, (2) a systemic lesion. That the changes in the cord are in some way dependent on its vascular supply is probable, but there is no evidence to show that they are due either to thrombosis of the vessels or to hæmorrhage. Most of the ascertained facts suggest that the changes in the spinal cord are due to the action of a toxic agent and that the anæmia is due to the same poison, but the existence of such a cause has yet to be proved.

COURSE.—For convenience of description, the course of the disease may be divided into the three following stages, although it is not to be expected that all cases will conform in every detail to the following description: (1) a stage of ataxia of the lower limbs associated with slight spastic paraplegia, together with subjective sensations in the extremities of the limbs; (2) a stage of severe spastic paraplegia with considerable loss of sensation over the extremities; (3) a stage of flaccid paralysis of the lower limbs with loss of all forms of sensation up to a definite level on the trunk.

The duration of each of these stages is subject to considerable variation. The first is usually long, persisting some fifteen months or more, but in one case it lasted only two months. The second varies greatly in duration, in some cases lasting only a month and in others being prolonged over many months. The third stage is as a rule short, lasting only a few weeks, although in one case it continued for nine months. Thus no exact time-limit can be fixed for the disease, although it usually runs its course in about two years.

SYMPTOMS.—In most cases the onset of the affection is gradual, and the earliest symptoms are numbness and tingling in the extremities. It, however, occasionally happens that the onset is rapid: it may then be attended with headache and vomiting.

1. During the *first stage* of the disease the patient complains of numbness and tingling in the toes and the extremities of the fingers, which feel to him thick and clumsy. A girdle-sensation and lightning-pains may be complained of, and there may be considerable difficulty in walking in the dark and in standing with the eyes closed. There may be a little pain on passing water, but as a rule there is at this stage no incontinence of urine. On examination of the patient no anæsthesia, analgesia, or hyperæsthesia can be made out, but some loss of the sense of position may be present. The gait is slightly spastic, and there is a tendency to stand with the legs wide apart so as to secure a wide base: when the feet are placed close together and the eyes closed marked unsteadiness results. The act of attempting to pick up a pin is performed in a clumsy manner, and on attempting to touch the tip of the nose with the first finger, the eyes being closed, lack of co-ordination and slight intention-tremor become evident. There is slight rigidity of the lower extremities, the knee-jerks are increased, ankle-clonus may be present, and the plantar reflex gives the 'extensor response' (see BABINSKI'S SIGN).

The superficial reflexes are present, and the myotatic irritability of the muscles is increased. The pupils react well to light and on accommodation.

2. The onset of the *second stage* of the disease is often rapid. In the course of a few hours or days the patient may lose the power of standing, the inability being due rather to the want of co-ordination in the muscles of the legs than to actual loss of power, for when lying in bed the patient is often able to move the legs with considerable force. Definite anæsthesia now becomes marked, commencing at first over the feet and lower portion of the legs, and gradually extending up the limbs, while the sensation in the tips of the fingers also becomes affected. At this stage the impairment of sensation does not follow a segmental distribution, but is rather of peripheral type, the longest neurons being affected both in the legs and arms. Sensation to all forms of impressions may now be gradually lost to a variable height on the trunk, where the distribution of the anæsthesia is segmental in character. A girdle-sensation in the mid-dorsal region is frequently experienced, as well as severe intercostal pain, which may in rare cases be accompanied by herpes zoster and even by subcutaneous hæmorrhages in the distribution of nerve-roots. During this stage the nutrition of the patient remains fairly good, and the muscles do not waste to a marked degree, retaining their myotatic irritability and reacting normally to faradism and galvanism. Irregular rises of temperature frequently occur, and the patients are liable to attacks of diarrhoea. The knee-jerks are still exaggerated, ankle-clonus is present, the plantar reflex gives the 'extensor response,' and all the superficial reflexes remain active in spite of the anæsthesia and analgesia. Exceptional symptoms occurring at this stage are weakness of one or other of the eye-muscles, slight nystagmus, and retinal hæmorrhages. Unilateral paresis of one vocal cord has once been observed.

3. The mode of onset of the *third stage* is one of the most striking features of this disease. The spastic condition formerly present is rapidly replaced by flaccidity, a change that occurs in a few hours or a few days. The temperature always rises at the commencement of this stage, but it subsides when the flaccidity becomes fully established. The inability to perceive all forms of sensory impressions over the lower limbs and trunk now becomes absolute, and there is complete incontinence of urine and feces. The knee-jerks are rapidly abolished, ankle-clonus and patellar clonus disappear. The plantar reflex however still maintains its extensor character, and this, in association with the absent knee-jerk and flaccid paralysis, constitutes one of the most diagnostic features of the disease. The other superficial reflexes persist and are active. Œdema of the legs and trunk often occurs at this period. The muscles now waste rapidly, and, if this stage is prolonged, the wasting in the lower extremities becomes extreme. The upper extremities become similarly affected at a later period. The muscles rapidly lose their faradic excitability, and the galvanic excitability is reduced, but there is no polar change such as is seen in complete reaction of degeneration.

Delirium at night is common during the third stage, and the patient often has varied delusions. Convulsive attacks which give rise to fine clonic movements without loss of consciousness have been observed in some cases. The patient's condition is now one of extreme asthenia. Cystitis frequently occurs, and bed-sores appear in nearly every case.

Paralysis of the lower abdominal and lower intercostal muscles occurs during this stage, and the diaphragm becomes weak and finally ceases to act. Death may thus result from failure of respiration, or from sudden syncope; it may however happen that the patient lingers on in a condition of profound anæmia and sepsis and dies of asthenia. A few cases have been recorded in which the position of the legs in the last stage is that of extreme flexion.

DIAGNOSIS.—The diseases with which combined degeneration of the cord is most liable to be confused are those of tabes, disseminated sclerosis, peripheral neuritis, acute myelitis, and a tumour of the spinal cord. The symptoms which in the earlier stage of the disease suggest the diagnosis of *tabes* are the ataxia and girdle-pains, while in the late stage the loss of knee-jerk and flaccid paralysis of the limbs are those which bear the most striking resemblance to that disease. The diagnosis during the first stage depends upon the spastic condition of the legs, the increase of knee-jerks, the presence of ankle-clonus, and the absence of the Argyll-Robertson pupil. In the later stage when the knee-jerks are absent, the complete loss of power of movement in the legs, the absence of the pupil-phenomenon, and the presence of an 'extensor response' on testing the plantar reflex, together with the history of a previous spastic stage, make the diagnosis clear.

The symptoms which most closely resemble those of *disseminated sclerosis* are the slight nystagmus, ataxia, and spasticity, and the differential diagnosis has to be based rather on an aggregate of small points than on any one symptom. The subjects of disseminated sclerosis are as a rule younger—between twenty and thirty rather than between thirty and fifty—remission of symptoms frequently occurs, nystagmus is more pronounced, and the sphincters are usually affected early. On the other hand, the slight affection of the arms while the legs are markedly affected, the symmetrical affection of all four limbs, and the presence of girdle- and lightning-pains are points in favour of the diagnosis of sub-acute combined degeneration.

It is only during the third stage of the disease that there is any liability to mistake the disease for *peripheral neuritis*. Even without a definite history of a spastic stage, the incontinence of urine, the anæsthesia of the trunk, the girdle-pain, and the 'extensor response' of the plantar reflex are sufficient to exclude the peripheral affection.

From *acute myelitis* the diagnosis is by no means easy in the absence of a distinct history of a gradual onset. A rapid onset of symptoms with early bladder trouble, marked pain and tenderness in the back, absence of any affection of the arms, greater disturbance of temperature, more rapid wasting of the muscles, and complete loss of reflexes would all be in favour of myelitis; nevertheless certain cases of acute myelitis clinically very closely resemble those of combined degeneration of the cord.

A tumour involving the spinal cord and giving rise at first to a spastic paraplegia followed by flaccid paralysis might suggest the possibility of combined degeneration of the cord, but the presence of severe radiating root-pain, complete absence of any affection of the arms, and the early onset of bladder-symptoms would all be in favour of the diagnosis of tumour.

PROGNOSIS.—The disease in most cases runs a steadily progressive course. In certain cases, how-

ever, temporary improvement has taken place; while in others the disease has run through the various stages in less than six months.

Although the prognosis is most unfavourable, almost complete recovery has taken place in cases in which the diagnosis seemed clinically certain.

TREATMENT.—Treatment has almost no influence on the course of the disease, though the anæmia frequently improves on the administration of arsenic and iron. Even the most careful nursing generally fails to ward off bedsores in the third stage of the disease, and cystitis nearly always occurs, though it usually yields to ordinary treatment.

FREDERICK E. BATTEN.

COMEDONES (*comedo*, a glutton).—**SYNON.** : Fr. *Acné Ponticue*; Ger. *Mittesser*.—The name 'comedo' is applied to the little cylinders of sebaceous and epithelial substance which are apt to accumulate in the follicles of the skin, and to appear on the surface as small round black spots; they generally constitute the first stage of acne. They may occur in all parts of the body where sebaceous follicles exist, but are most common on the face, the nose, the front and back of the neck, the shoulders, the penis, the breast, and within the concha of the ears, in the latter situation often attaining a considerable size. The accumulation of this substance is due to want of expulsive power of the skin, and to the slight impediment which is afforded by the aperture of the follicle to its exit. When squeezed out, a comedo is found to vary in colour, in figure, and in density, according to the period of its detention. When recent, the comedones are soft and white, and modelled into an exact cylinder by compression at the mouth of the follicle. If impacted for a considerable time, they acquire the yellow tint, the transparency and hardness of horn; they assume a bulbous figure from the dilatation of the follicle below the constricted orifice of the epidermis; and by their bulk they sometimes stretch the hair-follicle so far as to obliterate it completely. Besides their usual composition of sebaceous substance and epithelial cells, they frequently contain lanuginous hairs, and not rarely the Entozoon folliculorum in its different phases of development (see ACARUS). Comedones run a chronic course, they may occur at any period of life, and frequently co-exist with acne (see ACNE). In many cases symmetry is evident. A bacterial origin has been suggested.

Of special interest are the 'grouped comedones' in children and adults, described by Radcliffe Crocker, Colcott Fox, and Thin. Certain tracts of skin, as the temples and cheeks, scalp, the upper part of occiput and forehead are the seats of election. They do not inflame, and remain as passive papules. They are peculiar in their development, site, distribution, evolution, and 'close setting.' These 'grouped comedones' are in many instances most unsightly, and the source of much annoyance and disfigurement to those afflicted.

TREATMENT.—Comedones are most frequently met with in young persons in whom the powers of the constitution are not yet established, and who are benefited by generous diet and tonic treatment. Locally, we must stimulate to more active circulation the affected areas by thorough and systematic steaming, shampooing with a spirit of green soap, and water. This will be found of great service, and is best applied at bedtime. The comedones

may be *gently* squeezed out by the finger-nail, or by means of a comedo-extractor.

JOHN HAROLD.

COMMUNICATED INSANITY.—See 'FOLIE À DEUX.'

COMPLICATION (*con*, with; and *plico*, I fold).—It is difficult to give a strict definition of what ought to be included under the term *complication*; but the word signifies the occurrence during the course of a disease of some other affection, or of some symptom or group of symptoms not usually observed, by which its progress is therefore 'complicated,' and not uncommonly more or less seriously modified. The difficulty lies in determining what should be looked upon as essentially part of the original disease, and what as a mere accidental occurrence. For instance, many regard the cardiac affections which so often arise during the progress of acute rheumatism as a part of the complaint, others as complications. The same remark applies to the relationship of renal disease to scarlet fever, as well as to numerous other cases.

Complications arise in different ways. They may, as just indicated, be considered as developments of the original morbid condition, resulting from the same cause and being more or less allied; or they are independent and accidental, of which an illustration is to be found in the association of ague with scurvy or dysentery, or in the co-existence of two or more of the exanthemata. The most important class of complications, however, are those which follow the primary disease as more or less direct consequences. These may further be induced in various ways. Thus, for example, in febrile diseases secondary lesions are liable to arise as a result of changes in the blood; a mechanical act, such as cough, may lead to complications in the course of phthisis and other pulmonary affections; cardiac diseases frequently bring about consecutive changes in other organs, by inducing obstruction of the venous circulation; or emboli may originate under certain conditions and produce their usual consequences. It is of great practical importance to be acquainted with the complications which are liable to be met with in various diseases, and especially in those which are of an acute nature, in order that measures may be taken for their prevention, and that they may be recognised and treated at the earliest possible period, if they should occur.

FREDERICK T. ROBERTS.

COMPOSITORS' DISEASE.—See OCCUPATION-DISEASES.

COMPRESS.—A compress is a pad made of folds of lint or other material, and is used for the purpose of producing pressure, or for the application of hot or cold water or medicinal agents to the surface of the body. In the latter case the compress may be rendered waterproof by being covered with a piece of gutta-percha tissue or mackintosh cloth. See HYDROPATHY.

COMPRESSED-AIR DISEASE.—See CAISSON-DISEASE.

COMPRESSIBLE.—A term implying comparatively slight resistance, and applied specially to the pulse when it yields readily under the finger. See PULSE, The.

COMPRESSION OF THE BRAIN.—See BRAIN, Compression of.

COMPRESSION OF THE LUNG.—See LUNG, Compression of.

CONCRETIONS (*con*, together; and *cresco*, I grow).—SYNON.: Calculus; Fr. *Concrétion*; *Calcul*; Ger. *Concrement*.

DEFINITION.—A concretion is a deposit of solid products derived from the fluids and tissues of the body; such a deposit may be more or less diffuse (*Interstitial Concretion*), or form clearly defined bodies, usually, but not necessarily, hard (*Calculus Concretion*). The former are produced in the substance of tissues and are generally known under one or several of the following names: *Infiltration*, *Impregnation*, *Incrustation*, *Petrification*. The latter occur in pre-existing cavities, more especially glandular acini, ducts, canals, or diverticula, and are usually termed *Calculi* or *Stones*.

Nature of Concretions.—Concretions are due to the precipitation or accumulation of products which are more or less completely insoluble in the fluids of the part in which they are deposited. These insoluble products may be roughly classified as follows:—

1. *Colloidal, proteid, or fatty substances*, sometimes retaining traces of organisation, e.g. coagulated blood, fibrinous exudation, mucus, colloid matter, lardaceous matter (*corpora amylacea*), partly saponified fat and feces.

2. *Crystalline or granular products derived from proteid and fatty substances*, e.g. uric acid, xanthin, cystin, cholesterin, fatty acids, indigo, pigments, &c., in various states of combination.

3. *Crystalline or granular substances of simpler chemical structure* (generally called *inorganic compounds*), e.g. phosphates, carbonates, oxalates, of calcium, magnesium, and ammonium, &c.

4. *Foreign bodies* frequently form the nucleus of concretions belonging to the previous groups, e.g. animal and vegetable parasites, débris of food, &c. Some concretions are almost entirely composed of such foreign bodies, e.g. '*sable intestinal*,' *hair-balls*, &c.

PATHOGENESIS.—A. The concretions made of *proteid and fatty substances* differ from those of groups 2 and 3 in their mode of production. Thus *blood-concretions* are blood-clots which have undergone various degrees of condensation, granular or hyaline degeneration, superficial erosion, and imperfect calcification. They form in cavities where hæmorrhages or thrombosis are liable to occur, e.g. blood-concretions in urinary passages, globular thrombi in cardiac cavities.

Fibrinous, colloid, and mucous concretions are produced in the same way: they generally take the form of casts or of rounded concretions, e.g. muco-fibrinous casts of the nasal cavities, bronchi, and intestine. By further changes these masses may assume characters more generally associated with concretions. Thus they may become calcified, or undergo certain chemical changes, e.g. production of cholesterin, as in one recorded case of nasal cholesterin-concretion.

Amlyoid concretions or corpora amylacea are found either in the midst of old hæmorrhages or inflammatory exudations, e.g. pneumonic; in degenerated tissues (lymphatic glands, cartilage, nerve-tissue), syphilitic scars, and tumours. They

are evidently produced at the expense of the proteid constituents of tissues which have undergone morbid changes. They are also produced in the lumen of prostatic acini, where they are due to the accumulation of cells which gradually undergo chemical changes giving rise to the formation of a proteid substance having the reactions of lardacein. They are generally small brown grains, microscopical or just visible to the naked eye. They have a stratified structure.

Fatty concretions.—These may form in the intestine as the result of the ingestion of large quantities of fatty substances (as in the treatment of gall-stones by olive oil). The incompletely saponified fat may form well-defined rounded concretions. Some solid undigested particles of food generally act as nuclei. Fatty concretions (*urostealiths*) may also be produced in the urinary passages.

Closely allied to this last kind of concretions are those composed chiefly of undigested food-residue, or foreign bodies, accumulating in the intestine from one cause or another.

Seeds, the sclerenchymatous tissue of certain fruits, *vegetable fibres, hair, husks* of certain cereals, e.g. oats, may lead to the formation of concretions composed in great part of cellulose, known as 'sable intestinal' and *soft enteroliths*.

The continuous administration of large doses of insoluble drugs, such as the carbonates and other insoluble salts of calcium, magnesium, and bismuth, may lead to the formation of intestinal concretions composed of these substances.

Finally, in the lower animals *hair-balls* (*bezoars* or *agagropili*) may be formed by the accumulation in the intestine of hairs swallowed by them while licking their skin.

B. The mode of formation of *crystalline and granular concretions* composed principally of the substances belonging to groups 2 and 3 is more complex, and is of such importance that the presence of several of these concretions has been taken to indicate certain constitutional states; among which may be mentioned the uratic lithiasis or uric-acid diathesis; oxalic and phosphatic lithiasis, and cholelithiasis. None of the general theories which have been offered is by itself sufficient to explain the formation of all kinds of concretions. The views which are best supported by facts may be summarised as follows:

1. *An excess of the compounds capable of forming insoluble precipitates is thrown into the blood.* Deposits occur wherever the circulation becomes imperfect, the amount of carbonic acid or other acids, necessary to keep the salts in solution, diminishes, the quantity of water (as in the urinary secretion) is insufficient, foreign bodies are present, which act as nuclei, &c.

The excess in the blood would be due to one of the following causes: (a) *Excessive formation of certain waste products*, as uric acid, owing to disordered metabolism, imperfect activity of the tissues, or other causes. (b) *Resorption of bone* causing the presence of an undue amount of calcareous salts in the blood (calcareous metastasis). (c) *Excessive absorption of salts* (e.g. oxalates, phosphates, carbonates) *from the intestine*, as in vegetable diet. (d) *Absorption of products of fermentation* taking place in the intestine or other parts of the body—as of cystein or indol.

2. The deposition of insoluble compounds is due to *chemical changes which have previously taken*

place in the parts which are the seat of the precipitation. These chemical changes are in most cases attributable to lowered vitality or necrosis of cells and intercellular substance. Dead tissues are not the seat of those chemical exchanges which take place in living cells; they undergo certain fermentations, become alkaline, and do not generate carbonic acid; and under these conditions earthy phosphates and carbonates which constitute the greater part of most interstitial concretions may readily be precipitated, whether they are or are not in excess in the fluid bathing the abnormal tissues. The *incrustation of foreign bodies* may be explained in the same way, for when such bodies are situated in the midst of organs they are rapidly encapsulated by cicatricial tissue, badly supplied with blood, and liable to undergo degeneration. When these bodies are situated in a duct or cavity, they soon become covered with a layer of cells, fibrin, or mucus which rapidly undergo various degenerative changes. The presence of an organic substratum, reproducing the structure of the concretion, has been demonstrated in various forms of concretions. The precipitation of urates and of calcareous salts in connective, muscular, epithelial, and nervous tissues has been found to begin in badly nourished or necrosed cells. Calcification usually begins by the formation of globular bodies (*calcospherites*) in the midst of necrosed or degenerated tissues or products, and these spheroidal masses are similar in appearance to those produced artificially by the slow precipitation of salts of lime in presence of albumen.

3. The production of certain concretions is undoubtedly the result of fermentations, due to extraneous organisms, which alter the composition of the medium in which the concretion is formed. Thus when, owing to the penetration of bacteria into the urinary passages, urine undergoes alkaline fermentation, phosphates, carbonates, and urates of calcium, magnesium, and ammonium are precipitated and rapidly form calculi or incrustations of the diseased mucous membrane of the bladder or ureter.

Fermentation taking place within the mouth may cause the precipitation of tartar on teeth, or of calculi within the ducts of salivary glands. There are greater analogies between this kind of precipitation and that taking place in necrosed tissues than are apparent on first consideration.

CHARACTER OF CONCRETIONS.—A. *Interstitial concretions.*—These occur chiefly in connective tissues. Tissues infiltrated with earthy phosphates, carbonates, oxalates, and urates are generally white and opaque; not much altered in consistency in the early stages, they frequently become in the later stages hard and even brittle. Focal accumulations of carbonates, phosphates, and urates may give rise to the formation of chalky-looking masses which may be hard or pultaceous (e.g. tophi, calcified tubercles, calcified clots, calcified tumours). Plates of calcareous matter may become semi-crystalline, transparent, and brittle (e.g. calcareous plates in atheromatous arteries).

B. *Calculus concretions.*—These are generally produced in glandular ducts, in canals or in diverticula connected with them. Urinary and biliary concretions are by far the most common.

Calculi vary considerably in colour, consistency, and density. When they occur singly they are generally spheroidal, oval, or ovoid; occasionally, however, *solitary calculi* take more or less closely the form of the cavity in which they are produced

and may be very irregular in shape. Their surface is either smooth, rough, nodular, or even spinous. When several calculi are formed within the same cavity, they are usually more or less clearly *faceted* and may assume the shape of irregular cubes, pyramids, &c.

The characters of the chief concretions may be summarised as follows :—

1. *Ammonio-magnesian calculi*.—These are rare: they have a radiating crystalline structure, and are heavy but very friable.

2. *Calcium-phosphate calculi* are very rare: they are white, heavy, denser, more opaque than the last variety.

3. *Mixed phosphatic calculi* composed of *calcium*, *magnesium*, and *ammonio-magnesian phosphates*, with varying proportions of carbonate and oxalate of calcium, and of urates, are the most common phosphatic concretions. They may reach a very large size; their surface is often rough. According to the density of their structure they are either heavy or comparatively light; they are soft and easily crushed. Their colour varies, but is usually white or greyish-white; sometimes they take up certain pigments such as indigo, which gives them a pinkish or purplish colour. In the urinary passages they have often a nucleus of urates (this nucleus may be of very large size). Their structure is stratified, and some of the layers may be crystalline owing to the presence of crystals of triple phosphate.

4. *Carbonate of calcium calculi*.—These may form in the ducts of any of the glands; but although carbonate of calcium is present in a large number of concretions, calculi entirely composed of carbonate of calcium are not common. They are usually small, opaque, white, and easily crushed.

5. *Oxalate of calcium calculi*.—These are found in the urinary passages. They vary considerably in size, are usually irregular in shape, and their surface is generally nodulated (*mulberry calculi*). They are heavy and hard, difficult to crush, and usually dark brown in colour owing to pigmentation of their surface with blood-pigment, their presence frequently causing hæmorrhages.

6. *Calculi* almost entirely composed of *uric acid* are uncommon; they are hard and dense, yellowish or reddish-brown in colour.

7. *Calculi* composed of *mixed urates* (urates of sodium, ammonium, calcium, and magnesium), among which *acid urate of sodium* predominates, are the most common of all urinary calculi. Oxalate of lime and phosphates are frequently deposited on their surface, and in many cases these calcareous salts form a large proportion of the calculus. Uratic concretions vary much in size; when very small they constitute the so-called *urinary sand or gravel*. When larger they are usually rounded, smooth, yellowish- or reddish-brown, sometimes greyish-brown, occasionally almost colourless. They are hard and heavy, and have a stratified, very seldom a distinctly crystalline structure.

8. Small concretions composed almost entirely of *acid urate of ammonium* may be found in the excretory tubules at the apex of the Malpighian pyramids, where they take the form of dark brown spheroids which often unite to form irregular casts. Calculi almost entirely composed of urate of ammonium occur chiefly in children.

9. *Xanthin-calculi* are very rare and occur in the urinary passages. Their size varies from that of a pea to that of a pigeon's egg. They are soft,

light, brown or cinnabar-red in colour, and composed of amorphous easily separated layers.

10. *Cystin-deposits* have been described as occurring in the liver and kidney. Calculi are formed less infrequently than is generally believed in the urinary passages. These concretions are rounded or oval, light, soft, yellowish, translucent, waxy-looking, acquiring a greenish colour after exposure to air. They have a crystalline structure.

11. *Cholesterin*.—Cholesterin may occur as deposits in tissues which have undergone degeneration, and in unabsorbed pathological exudations. It is the most important constituent of *biliary calculi*. Cholesterin-concretions have also been described in connection with the urinary passages and the nasal cavity. *Calculi almost entirely composed of cholesterin* are not common. They seldom exceed the size of a walnut, and are frequently very much smaller; they are rounded, generally smooth, light, soft, pale yellow, and translucent. They have a radiating crystalline structure. Gall-stones are generally more or less deeply stained with bile-pigment, and contain various proportions of calcium-salts and organic matter (*mixed biliary calculi*).

12. *Bile-pigment calculi*, chiefly composed of *Bilirubin-calcium*, are uncommon. They are small, hard, heavier than cholesterin gall-stones, and reddish-brown or almost black in colour.

The characters of fibrinous, amyloid, and blood-concretions have previously been described.

Some of the chemical properties, recognition of which may be of use in the rapid diagnosis of the most important concretions, are given in the following table. It must be remembered, however, that few concretions are composed of only *one* substance. The nature of a calculus is therefore usually determined by finding out its most important constituent.

MODE OF OCCURRENCE OF THE CHIEF FORMS OF CONCRETIONS.

A. **Concretions occurring in various tissues**.—*Calcification* or *petrification* (calcareous impregnation, infiltration, or degeneration) is of common occurrence. It is apparently due in most cases to some combination taking place between the altered proteid constituents of dead tissues or exudations and the lime-salts of the blood or lymph. The deposition of insoluble lime-salts may begin in cells (nerve-cells, epithelial cells, muscular fibres), or in the intercellular substance, e.g. cartilage-matrix. It ultimately becomes diffuse (see description of concretions). Calcification may occur in the following products or organs :—

1. *Thrombi*.—Cardioliths, arterioliths, phleboliths, endocardial vegetations.

2. Unabsorbed fibrinous, hæmorrhagic, or purulent *exudations*, occurring in the various serous cavities and organs of the body. Also exudations or secretions covering foreign bodies.

3. Lesions characterised by necrosis of tissues, as *atheromatous* patches in arteries, unabsorbed *infarcts*, caseous *tubercles*, *gummata*, *actinomycomata*.

4. Tissues separated from their normal anatomical connections, e.g. dead extra-uterine foetus (*Lithopedion*), loose bodies in joints and other serous cavities, free bodies in peritoneum, various parasites.

5. Dense, hyperplastic, lamellated, and more or less degenerated cicatricial connective tissue formed in connection with inflamed serous membranes,

SOME OF THE REACTIONS AND SOLUBILITIES OF THE MOST IMPORTANT CONSTITUENTS OF CONCRETIONS, AVAILABLE FOR PURPOSES OF RAPID DIAGNOSIS.

SMALL PORTIONS ARE TAKEN FROM VARIOUS PARTS OF THE CONCRETIONS, FINELY POWDERED IF POSSIBLE, AND SUBMITTED TO THE FOLLOWING TESTS:—

A. Strongly heated on platinum foil	B. Treated by dilute hydrochloric acid	C. Treated by acetic acid	D. Treated by strong solution of caustic potash (hot)	E. Special tests (chiefly confirmatory)	Substance
I. Slightly charred, but in great part incombustible, an abundant residue being left.					
A. Fusible (blow-pipe)	Soluble without effervescence	Soluble without effervescence	Ammonia evolved	Reactions of phosphoric acid, calcium, and magnesium	Ammonio-magnesian phosphate and phosphate of calcium
B. Not fusible	Soluble without effervescence	Soluble without effervescence	No ammonia evolved	Reactions of phosphoric acid and calcium	Phosphate of calcium
	Soluble with effervescence	Soluble with effervescence	Insoluble . .	Reactions of carbonic acid and calcium	Carbonate of calcium
C. Abundant charring, but residue considerable	Soluble without effervescence	Insoluble . .	Insoluble . .	Reactions of oxalic acid and calcium	Oxalate of calcium
	Partly soluble (uric acid separates)	Partly soluble (uric acid separates)	Soluble . .	Murexide reaction	Urates of sodium and potassium
	Slowly soluble (uric acid separates)	Very slowly soluble (uric acid separates)	Very slowly attacked	Murexide reaction and calcium reaction	Urate of calcium
II. Combustible, leaving very little residue.					
A. Burns without giving a strong characteristic smell	Partly soluble (uric acid separates)	Partly soluble (uric acid separates)	Soluble (ammonia evolved)	Murexide reaction	Urate of ammonium
	Insoluble . .	Insoluble . .	Soluble . .	Murexide reaction	Uric acid
	Insoluble ? . .	Insoluble . .	Soluble (solution reddish-yellow)	Does not give the usual murexide reaction	Xanthin
	Insoluble . .	Insoluble . .	Soluble . .	Soluble in ammonia. Hexagonal plates on evaporation. Burns with bluish flame, & gives off aromatic smell	Cystin
	Insoluble . .	Insoluble . .	Insoluble . .	Soluble in hot alcohol. Notched rhomboid plates separate on evaporation	Cholesterin
B. Produces a purplish vapour, giving crystalline sublimate	Insoluble . .	May be partly soluble	Soluble . .	Gmelin's reaction .	Bile-pigments. Hæmatoidin
	Insoluble . .	Insoluble . .	Insoluble . .	Blue solution in fuming sulphuric acid	Indigo
C. Gives off a smell of burnt horn	Partly and slowly soluble, coloured solution	Partly and slowly soluble, coloured solution	Partly soluble, coloured solution	Reaction of iron after complete incineration. Various absorption-bands in spectrum	Blood-concretions
	Swells up slowly and softens	Swells up slowly and softens	Soluble . .	Proteid reactions .	Fibrin-concretions
	Insoluble . .	Insoluble . .	Soluble (i.e. gradually decomposed)	Reactions of lardacoin	Amyloid concretions
D. Burns with smoky flame, acrid smell of burnt fat	Insoluble . .	Insoluble . .	Soluble (saponified)	Structure of hair	Hair-balls
E. Burns with flame and smell of burnt wood	Insoluble . .	Insoluble . .	Insoluble . .	Soluble in ether .	Urostealith
				Brown with iodine. Structure of vegetable cells	Fatty concretions

Few of these concretions occur in a pure state. The last seven are usually encrusted or impregnated with variable amounts of inorganic salts, especially carbonates, phosphates, and oxalates.

around foreign bodies, &c., e.g. calcareous plates in pleura, peritoneum, pericardium, tunica vaginalis, meninges, &c. (generally associated with calcification of old exudation). Fasciae, tendons, articular capsules, fibrous capsule of various parasites, as echinococcus-cysts, trichinae.

6. The muscular coat of arteries in the neighbourhood of atheromatous patches, and all the coats of arteries, and sometimes of veins in senile degeneration of the vessels.

7. The stroma of tumours in various parts of the body, especially of uterine myomata.

8. The diseased membrana tympani.

9. The crystalline lens in certain forms of cataract.

10. Nerve-cells in the neighbourhood of traumatic inflammatory lesions; the epithelial cells of the kidney and cardiac muscular fibres in certain intoxications.

11. To these various lesions, which are distinctly connected with previous local degenerative changes, it is necessary to add the calcifications due to so-called *calcareous metastasis*. In people between fifty and seventy extensive calcification of the costal, laryngeal, and bronchial cartilages, of arteries and sometimes of veins, which may become rigid tubes, of the substance of the kidney and even of that of the lung, may occur. This calcification has been attributed to the circulation of a great excess of lime-salts in the blood owing to senile involution of bone leading to considerable reabsorption of calcareous matter.

Calcareous metastasis occurs also in younger individuals who are affected with diseases of the bones associated with absorption of lime-salts from them. In none of these cases can the effects of degeneration of tissues in the parts which become the seat of calcification be entirely excluded.

12. Concretions in the Pineal Body, and Choroid Plexus ('Brain-sand').—Small sand-like granules composed of layers of proteid matter (degenerated cellular products), concentrically arranged and impregnated with calcareous salts (phosphate and carbonate of calcium with some phosphate of magnesium and ammonium) are normally present in the pineal gland and the choroid plexus. Under certain pathological conditions they may become excessive in number and size. Concretions of the same kind are not uncommon in connection with sarcomatous tumours of the meninges, in which the blood-vessels and part of the connective-tissue are also frequently calcified. When such calcareous concretions are present in these tumours, they receive the name of *Psammoda*.

13. Uratic concretions. (Uratic impregnation or infiltration; tophi; chalk-stones; gouty concretions or nodes.)

The deposition of urates in the tissues is certainly due to important disorders of metabolism. According to some these disorders bring about an accumulation of uric acid in the blood causing the deposition of insoluble urates in certain parts of the body, where they cause inflammatory and necrotic changes. According to others the deposition of urates in the parts affected is preceded by inflammatory or necrotic changes, so that the formation of uratic concretions would be brought about much in the same way as the deposition of earthy salts.

The relations of uric acid, xanthin, hypoxanthin, guanin, and adenin to nucleins are not sufficiently well understood to make clear the pathogenesis of the *uric-acid diathesis*.

Deposits of urates have been found in some cases to start in the cells of cartilage and other connective tissue; at a later stage, however, the infiltration is intercellular or diffuse.

The most common seats of uratic deposits are certain articular cartilages, also the cartilages of the ear, eyelids, nose, and larynx, the fibrous tissue of articular capsules, tendons, fasciae, bursae, and skin. Deposits have also been found in bone. In the kidney, the interstitial tissue, as well as the epithelium of the tubules, may be infiltrated with urates.

These deposits are composed of acicular crystals or of granular material.

Tophi or *chalk-stones* are localised accumulations of the same products; they vary much in size and are usually formed under the skin in the neighbourhood of affected joints. They may be comparatively soft and pulsatious, or may have the consistency and appearance of chalk. They are composed of urates of sodium, calcium, magnesium, and sometimes ammonium, with various proportions of phosphate and carbonate of calcium and of organic matter. They often cause inflammation, suppuration, and ulceration of the thin skin covering them, and may in this way be discharged.

B. Urinary concretions.—Minute concretions are not infrequent in the substance of the kidney; they are generally uratic. Thus in infants dying between the second and fourteenth day after birth spherules and casts of ammonium urate may be found in the straight tubules, giving rise to the *uratic infarcts of infants*. In gouty subjects deposits of *urate of sodium*, and other urates, may be found in the connective tissue, and occasionally accumulate to such an extent in the tubules as to block them.

Small masses of spheroidal, 'dumb-bell' and octahedral crystals of *oxalate of lime* are also occasionally present in cirrhotic kidneys. *Calcareous deposits* may form near the apex of the Malpighian pyramids in calcareous metastasis.

The other concretions found in the urinary organs are *calculi* or '*stones*', which are usually situated in the pelvis of the kidney (nephrolithiasis) or in the bladder. Their mode of formation, composition, and characters have already been considered.

The existence of some chronic irritation of the kidney and urinary passages predisposes to the formation of calculi, desquamated cells, small masses of mucus, blood-clot, or fibrin frequently acting as nuclei upon which the precipitation of salts begins. Débris of ulcerating tumours, foreign bodies such as portions of catheter or hair-pins, may act in the same way. An abnormal composition of the urine due to disorder of metabolism is, however, an important factor in the formation of certain calculi, such as those composed of uric acid, urates, oxalate of calcium, cystin, and xanthin.

The presence of a calculus in the urinary passages by causing inflammation of the mucous membrane and retention of urine, and by predisposing to secondary bacterial infection, favours the further precipitation of salts, especially when bacteria have caused the urine to undergo ammoniacal fermentation. When the urine has become alkaline a rapid precipitation of phosphates or carbonates takes place.

While inflammation of the urinary passages favours the formation of calculi, these concretions when formed often become a source of further morbid changes, such as cystitis, ureteritis, pyelitis, pyelonephritis, hydronephrosis, pyonephrosis, renal

abscess, perinephric abscess; lacerations and ulcerations of the pelvis, ureter, bladder, and urethra; fistulous openings between the ureter or bladder and the intestine, or other cavities.

The presence of calculi in the substance or pelvis of the kidney is a frequent source of irritation and hæmorrhage; their passage down the ureter gives rise to great pain (renal colic). The presence of stones in the bladder is also a source of irritation and pain.

Uric-Acid and Uratic concretions.—According to various statistics these concretions constitute from 60 to 80 per cent. of all the urinary calculi, the differences in the estimates being probably due to the difficulty of determining in some mixed calculi whether phosphates or urates are the most important constituents. Uratic calculi are formed in acid urine, but when the reaction of the urine becomes alkaline, earthy phosphates, carbonates, and oxalates are deposited more or less abundantly on the surface of the concretion previously formed, which is thus frequently rendered rough instead of remaining smooth, as is usual in the case of these stones. Uratic calculi vary considerably in size, and may be single or multiple.

Phosphatic Urinary concretions come next to the uratic calculi in order of frequency. Mixed calculi in which phosphates predominate form from 30 to 40 per cent. of all urinary calculi. Stones almost entirely composed of phosphates are much less frequent—probably less than 8 per cent. Phosphatic calculi are frequently multiple. When solitary they may reach a large size.

Oxalate of Lime calculi are less common than the phosphatic stones (from 2 to 5 per cent. of all urinary calculi). Concretions entirely composed of oxalate of lime are rare; this salt may form a thick crust on the surface of uratic stones acting as nuclei. *Mulberry calculi* vary considerably in size.

Calculi composed of almost pure carbonate of calcium are very rare in the urinary passages, but this salt enters frequently into the composition of mixed phosphatic calculi.

Cystin-calculi are rare, but in some published statistics they reach the high proportion of 1·4 and 2·8 per cent.

Concretions formed of Xanthin, Cholesterin, and Indigo are very rare. Concretions consisting of *blood and fibrin* and *urosteoliths* are also uncommon.

C. Biliary concretions (*Biliary calculi* or *gall-stones*).—These occur in the gall-bladder, hepatic, cystic, and common bile-ducts. The state associated with their presence is known by the name of *Cholelithiasis*. Stagnation of bile in the bile-passages is a frequent source of gall-stones; these concretions are not uncommon in elderly people. Inflammation and neoplasms of the gall-bladder and ducts favour the formation of gall-stones, which in their turn are often a source of retention of bile and inflammation of the biliary passages. The irritation produced by calculi has also been thought capable of causing primary tumours of the gall-bladder.

Biliary calculi may escape from the bile-passages into the intestine, and during their migration frequently cause more or less complete obstruction of the common bile-duct, giving rise to pain (*hepatic colic*) and to jaundice. When a stone becomes impacted in the common bile-duct, it may give rise to dilatation of the biliary passages, biliary pigmentation of the liver, degeneration of the liver-cells, cirrhosis &c. Under these conditions secondary bac-

terial infection of bile-ducts is liable to occur, and this gives rise to various forms of cholangitis, as well as acute and chronic biliary hepatitis (including hepatic abscesses). Ulceration of the gall-bladder or bile-ducts leads to perforation and purulent peritonitis or retroperitonitis, or to localised peritoneal adhesions through which ulceration may extend in various directions. In this way fistulous channels may be produced between the gall-bladder and the duodenum, colon, or surface of the body, or between the common bile-duct and the duodenum (biliary fistula). These passages may allow the direct escape of gall-stones into the duodenum, the colon, &c.

Biliary concretions may be so small as to be microscopical, or may in some exceptional cases reach the size of a hen's egg. The former, which are usually composed of bile-pigments, are found in the hepatic ducts, and even in the bile-canalliculi, and the intracellular rootlets of these canaliculi. The larger (of which cholesterin is usually the chief constituent) occur in the gall-bladder and main bile-ducts. The largest stones are found in the dilated gall-bladder, which may be entirely filled by one or several of them. Solitary gall-stones are usually rounded, ovoid, or pyriform. They may be smooth or rough. Multiple gall-stones are faceted, seldom exceeding the size of a hazel nut.

There are three main types of gall-stones.

1. Gall-stones of which *cholesterin* is the chief constituent. These calculi contain in addition to cholesterin various proportions of organic matter, bile-pigments, and earthy salts. They have usually a nucleus composed of pigmented cellular débris and mucus derived from the mucous membrane. Around this cholesterin-crystals form more or less pigmented layers, which show a radiating crystalline arrangement. (a) *Cholesterin* may form up to 90 per cent. of the constituents of these calculi, in which case they are pale yellow, translucent, and very light (pale cholesterin-calculi). (b) The commonest cholesterin-stones contain more or less bile-pigment, which gives them a yellow, reddish-brown, greenish-brown, or almost black colour (dark cholesterin-calculi). (c) *Mixed biliary calculi*, containing a larger proportion of biliary pigment, carbonate of calcium, and other salts, are also frequent. They are heavier, less crystalline, more friable, than the preceding varieties. They vary in colour from greyish-white to almost black.

2. *Bile-pigment calculi*.—These are small, reddish-brown, or almost black calculi, and are uncommon. They are chiefly composed of *bilirubin-calcium* which may form from 28 to 61 per cent. of their constituents. Biliverdin, bilifuscin, biliprasin, and bilihæmin are also found in these stones.

3. *Calcium-carbonate calculi*.—These are rare. In addition to the various elements already mentioned as entering the composition of gall-stones, the following may occur in very small quantities: zinc, iron, copper, manganese, silicon, uric acid, and fats.

D. Intestinal concretions (*Enteroliths* or *Intestinal calculi*).—These are much less frequent in man and carnivora than in herbivora (especially ruminants).

In man they are generally formed round a nucleus composed of some indigestible content of the intestine, some hard mass of feces—scybala or fecal concretions—a gall-stone or some foreign body introduced into the alimentary canal.

Sluggishness of the intestine, habitual constipation,

any condition causing stagnation of intestinal contents, or the ingestion of large quantities of indigestible material, all favour the formation of intestinal concretions.

Enteroliths are usually situated in the large intestine, and more especially in the cæcum, the appendix vermiformis, or other intestinal diverticula. They may be a source of obstruction, of inflammation, ulceration, or perforation, and their consequences. Appendicitis and perityphlitis are among the special lesions which may be produced by intestinal concretions.

According to the nature and size of the bodies which act as nuclei, and the amount of the calcareous deposits forming on their surface enteroliths present various characters. The following are the most important varieties of intestinal concretions:—

1. *Intestinal sand or gravel* ('*Sable intestinal*').—Small concretions, generally numerous, composed chiefly of undigested seeds (e.g. groups of fig-seeds) or of groups of vegetable cells with thick cellulose walls (Sclerenchyma) such as are found in gritty pears. These masses are in great part composed of cellulose, are encrusted with a variable amount of calcareous salts of the same kind as those found in the larger concretions.

2. *Calculi* (*Compact or Hard Enteroliths*).—These are heavy and dense concretions seldom exceeding a walnut in size. They are single or multiple; in the latter case they may be faceted. They have a stratified structure, the various layers being frequently differently coloured, white, yellow, brown, or almost black.

They consist chiefly of magnesium, calcium, and ammonio-magnesian phosphates, carbonate and oxalate of calcium, traces of silica and bile-pigments or some of their derivatives. The nature of the nucleus is variable. Such stones are uncommon in man; in horses and cattle they are less rare and may reach a considerable size.

3. *Spongy or soft enteroliths*.—These are light, elastic, porous masses, often irregular in shape and having a felt-like structure. Those occurring in man are mostly composed of vegetable fibres or husks of seeds with their hairy appendages (such as may be formed in coarse oatmeal), inspissated fæces and scanty deposits of earthy salts (compact enteroliths). In the ruminants and other animals which are in the habit of licking their fur and swallowing much hair, balls almost entirely composed of hair may be formed; these are known as *hair-balls* (ægagropili or bezoars).

4. Calculi composed of carbonate and other insoluble salts of magnesium, calcium, or bismuth have resulted from the continued administration of large doses of carbonate of magnesium, chalk, or bismuth salts.

5. *Fatty concretions*, composed of partly saponified fat, have been observed after the continuous administration of olive oil—as in a certain form of treatment for gall-stones. These concretions present the characters of partly decomposed and saponified olive oil; they may be very numerous and be as large as a large bean; they may be well defined, rounded or ovoid, but are usually irregular in shape. They have a yellowish-green colour and a rancid smell. They melt by heat and burn with a smoky flame. They are very light. They have been mistaken for gall-stones, but the slightest care would prevent such an error.

E. **Pancreatic concretions**.—These concretions are comparatively rare. They may take the form of small sand-like grains disseminated through the substance of the gland.

Calculi, solitary or multiple (sometimes very numerous), are occasionally formed in the pancreatic ducts and their branches. These stones may reach a large size (up to one inch in diameter). They are rounded, ovoid or irregular, and branched; their surface is sometimes nodulated or uneven, having a 'worm-eaten' appearance. They are usually soft, whitish in colour, and composed of carbonate of calcium with a small amount of phosphate of calcium and a comparatively large quantity of organic matter.

The formation of these calculi is usually associated with inflammation of the ducts and degenerative changes in the parenchyma of the gland. The ducts may be dilated and the seat of suppuration: the rest of the gland is usually in a state of cirrhosis.

F. **Salivary calculi**.—Concretions are occasionally found near the orifice of Stenson's and Wharton's ducts, which they obstruct more or less completely. They are usually rough and irregular in shape, composed of earthy phosphates, carbonate of calcium, and organic material, and often contain a nucleus of some foreign matter (vegetable debris, splinter of bone, bacteria, &c.), which has passed from the mouth into the duct.

G. **Tartar and Tooth-stones** (buccal or dental concretions).—These deposits are closely allied to salivary calculi. The mineral salts of saliva are frequently deposited upon the teeth, especially about their neck; the precipitate may be so abundant as to cover entirely the crown. The molars are specially liable to be affected in this way.

Tartar contains a large proportion of inspissated altered mucus and cells, and many bacteria, especially the *Leptothrix buccalis* and *Spirillum buccale*.

H. **Tonsillar concretions** (*calculi*).—Plugs of desquamated epithelium, leucocytes, mucus, fat, debris of food and bacteria, are frequently formed in the tonsillar crypts. They are soft, greasy-looking, and have usually a very fætid odour. When calcified they form calculi varying from a millet seed to a bean in size; they are usually soft and contain a large proportion of carbonate of calcium. They may be a source of considerable irritation.

I. **Mammary concretions**.—*Calcification of the stroma of carcinomatous tumours* may occur in the mamma as in other parts. *Small colloid and fatty concretions* are frequently found in the dilated acini and ducts of the mamma when this organ is affected with lesions, such as tumours, causing obstruction of the ducts.

Mammary calculi have been observed in the teat-ducts of cows; they consist of calcium carbonates, earthy phosphates, fat, and other organic substances.

K. **Prostatic concretions**.—*Corpora amy-lacea* (or amyloid concretions) are very common and may be considered almost as physiological products; they occur at all ages. Amyloid concretions are usually small microscopic bodies, but may become large enough to be visible with the naked eye as brown grains distributed all through the substance of the prostate.

Prostatic calculi.—They are formed in the same way as the corpora amy-lacea, by the accumulation of cells and secretion in the prostatic acini, but in addition they are calcified. They vary in size,

being often microscopic, but they are not infrequently as large as millet seeds, and may reach the size of a small pea. They are usually numerous. These concretions are common in old people, especially when the prostate is the seat of disease.

L. Spermatic or Seminal concretions.—These occur in the seminal vesicles. They are caused by the accumulation of spermatozoa and other secretions, which undergo degenerative changes and become calcified.

M. Preputial calculi.—These are observed in cases of phimosis, owing to the retention, degeneration, and calcification of masses of smegma. Calculi escaping from the urethra may also be retained in the preputial sac and continue to enlarge by further deposition of organic matter and lime-salts on their surface.

N. Vaginal concretions.—Pessaries and other foreign bodies retained in the vagina for a long time may become encrusted with calcareous salts; the presence of these bodies also causes inflammation, ulceration, and the formation of much granulation tissue, in which vaginal concretions are frequently embedded.

O. Nasal concretions.—Mucus, sometimes mixed with blood, or inflammatory exudation retained in the nasal cavities occasionally becomes inspissated and much altered, forming crusts or almost complete casts of part of the passages. One case has been recorded in which the nasal fossæ were filled with masses of cholesterin, which must have been formed out of such retained products.

Nasal calculi (Rhinoliths) may be produced by the calcification of retained secretions, but are more usually due to calcareous incrustation of foreign bodies, such as seeds, beads, boot-buttons, &c., which may find their way into the nasal cavities (usually of children). These stones sometimes reach a large size, and, besides causing obstruction and unhealthy discharges, may lead to ulceration and nasal deformities.

P. Bronchial concretions, similar in nature to the nasal concretions, occur occasionally.

Q. Otoliths, Ceruminous plugs.—Accumulation of secretions in the external auditory meatus often tends to the formation of plugs of wax (cerumen) which may dry, become hard and almost black, and give rise to irritation and deafness. These masses may contain, in addition to the cerumen, epithelial cells and hairs, and they offer a suitable nidus for the growth of several moulds and bacteria. *See* EAR, Diseases of; OTOMYCOSIS.

By a deposition of carbonate and phosphate of calcium, *calcareous concretions (otoliths)* are formed, but this is of rare occurrence in man. These pathological *otoliths* have no relation to the *otoliths* which are found normally in the fluids of the internal ear. These physiological concretions are composed of crystals of carbonate of lime embedded in mucus.

SHERIDAN DELÉPINE.

CONCUSSION (*concutio*, I shake together).—This term is used to indicate a condition induced by a more or less violent shaking or physical commotion of the general system, or of some particular organ, whereby serious symptoms may be induced, but no definite lesion can be detected to account for them. The nerve-centres are the parts most liable to be thus affected, concussion of the brain or spinal cord being of considerable moment, giving rise to more or less complete abolition of their functions,

though this effect is usually only temporary (*see* BRAIN, Concussion of; and SPINAL CORD, Diseases of). General concussion of the body is highly important at the present day, in connection with railway accidents, after which persons seem to be uninjured, or only to be slightly shaken, but subsequently more or less grave symptoms, associated with the nervous system, set in. *See* RAILWAY ACCIDENTS, Results of.

FREDERICK T. ROBERTS.

CONCUSSION OF BRAIN, SPINE, &c.—*See* BRAIN, Concussion of; and SPINAL CORD, Diseases of.

CONDYLOMA (*κονδύλωμα*, a knob).—*SYNON.*: Fr. *Condylome*; Ger. *Feigwarze*.

This vague term has been used to describe at least two different things—namely, (*a*) papillomata, or warty growths in the neighbourhood of the anus and genital organs; and (*b*) the syphilitic lesions called mucous patches or mucous tubercles. Of these the former only will be dealt with here. The latter are described under MUCOUS PATCH.

DESCRIPTION.—Condylomata are commonly found in connection with the constant moisture and irritation due to acrid secretions, whether natural or morbid, especially the discharges of venereal affections. The growths occur most frequently and attain the greatest size in dirty persons, in whom also they are most liable to become inflamed, ulcerated, and fissured. The enormous masses sometimes seen in pregnant women are often called ‘cauliflower’ growths.

TREATMENT.—Frequent washing, followed by the free application of some absorbent powder, such as oxide of zinc, is essential. When the growths are very large, or fail to disappear under caustics, removal by scissors, knife, or cautery will be necessary.

ARTHUR COOPER.

CONFLUENT (*confluo*, I run together).—Applied chiefly to a variety of small-pox and other exanthemata, in which the eruption runs together or coalesces.

CONGENITAL (*con*, together; and *genitus*, begotten).—Existing at birth. *See* MONSTROSITIES; and FÆTUS, Diseases of.

CONGENITAL SPASTIC PARAPLEGIA.—*See* CEREBRAL DIPLEGIA.

CONGESTION (*congero*, I accumulate).—Overfulness of vessels, caused by accumulation of their contents: generally applied to blood-vessels. *See* CIRCULATION, Disorders of.

CONGO SICKNESS.—*See* SLEEPING SICKNESS.

CONIUM, Poisoning by.—*SYNON.*: Fr. *Empoisonnement par la Ciguë*; Ger. *Schierlingsvergiftung*.—All parts of the hemlock plant (*Conium maculatum*; Greek, *κάνειον*) are poisonous. Both the leaves and fruit are used in medicine. Its toxic properties were known in ancient times; the plant was used for the destruction of criminals by the ancient Greeks, and there is no doubt that Socrates was poisoned by it. Death from conium in this country has perhaps always been the result of misadventure or suicide; but on the Continent the active principle of the plant, *conine*, an alkaloid,

has been administered for the purpose of wilful homicide, death resulting from a dose of 10 to 15 drops.

ANATOMICAL CHARACTERS.—The signs of asphyxia, engorgement of lungs and of the right heart, and a general venous condition of the blood, appear to be constant after death from conium. There is nothing else specially noticeable.

SYMPTOMS.—Preparations of conium, as well as the alkaloid, or mixture of alkaloids, known as *conine*, when taken in toxic doses, produce excessive muscular weakness beginning in the lower limbs, and extending gradually upwards, with giddiness and disordered vision. These symptoms are in some cases preceded by nausea and vomiting, with dryness or burning pain in the mouth and fauces. There is a desire to remain quiet, and a peculiar heaviness or drooping of the eyelids, the patient lying with his eyes shut. This, and the impairment of vision, appear to be due to paralysis of the ocular muscles. The pupils may be natural, but later they become dilated. The pulse is slow till death is actually impending. The paralysis progresses gradually upwards, till eventually heart and respiration are affected, more especially the former. Convulsions and impairment of the mental faculties—hitherto intact—now set in; finally sensation is impaired, and death ensues from asphyxia.

DIAGNOSIS.—The paralysis of motion, progressing gradually upwards, with sensation long unimpaired, and the peculiar drooping of the eyelids, are perhaps diagnostic of the nature of the poison.

PROGNOSIS.—As no antidote is known which counteracts the effects of conine, the prognosis must always be a guarded one, and will depend entirely upon the general condition of the patient.

TREATMENT.—The stomach must be emptied by the stomach-pump or siphon-tube, and well washed out. Emetics may also be used to evacuate the stomach. Tannin and other astringents must be freely administered, to precipitate the active alkaloid, and prevent its absorption. Castor oil, administered by the mouth or rectum, may aid the removal of the alkaloid when it has been rendered insoluble by tannin. Strong coffee, brandy, ammonia, and stimulants generally are serviceable, as may also be the hypodermic injection of ether. Hypodermic injections of 1-40th grain of sulphate of atropine are very promising, especially in the later stages; atropine acting as a respiratory and cardiac stimulant. Persistent artificial respiration, and stimulation of the respiratory and cardiac functions by the use of electricity, ought not to be neglected when these are affected.

THOMAS STEVENSON.

CONJUNCTIVITIS.—Inflammation of the conjunctiva. See EYE, AND ITS APPENDAGES, Diseases of.

CONSCIOUSNESS, Disorders of.—The disorders of consciousness are so numerous as to make it desirable briefly to consider them in one article, with a view to their classification and the better comprehension of their mutual relations. We shall, therefore, here group and arrange the varied morbid conscious states, not aiming to produce a strictly scientific classification so much as one which will be practically useful.

1. Exaltation.—Under this head may be ranged certain states of consciousness more or less distinctly

bordering upon the unnatural, to be met with in persons under the influence of 'mental excitement' from various causes, as from sudden good news, or generally pleasant surroundings; also from a slight degree of poisoning by alcohol, opium, hashisch, or other drugs; or from an early stage of some forms of insanity, or of delirium. In this state of mental exaltation the individual's powers of perception, apprehension, recollection, thought, emotion, and volition, would seem to be all more or less intensified, just as in that of hebetude or dementia they are diminished, and consciousness is proportionately dwarfed.

2. Perversions.—Many of the various defects here to be referred to are very partial in the extent to which they implicate consciousness, though others are general. In what is known as an *illusion* some object of sense is not correctly perceived; or in other words, some sensorial impression is quite wrongly interpreted—as when a feverish or a maniacal patient, looking at some inanimate object, declares that it is a cat or a dog about to fly at him, or, hearing even the slightest noise in any part of his room, interprets it to be the voice of some friend or imagined enemy. In the case of an *hallucination*, however, forms are declared to be seen, or voices heard (by a patient suffering from delirium tremens, for instance), where no appreciable external realities could have started the notion. And in these cases, it is not that the patient sees or hears without believing; he implicitly believes that the visions or voices, which have been conjured up subjectively by the mere disordered working of his own brain, have a real existence in the outside world. It is necessary to make this distinction, because it is by no means uncommon, in regard to the olfactory sense (especially in some epileptics), for odours or smells to be perceived which the patient soon comes to know are purely subjective or devoid of any external correlative.

Hallucinations and illusions, moreover, though occasionally existing alone, are quite commonly associated with a very important and more general derangement of consciousness, namely, *delirium*. This is a symptom very common in many fevers, in certain low states of the system, after severe frights, in inflammatory or other lesions of the brain and its membranes, as a result of some narcotico-irritant poison, or occasionally in a person who is recovering from an epileptic attack or from the stupor sequential to a series of convulsive attacks. The state itself varies much in intensity. Three fairly distinct types exist. In (*a*) *low* or *muttering* delirium the patient lies still and more or less heedless of what is occurring around; or, if heeding at all, the impressions which he receives give rise to erroneous perceptions (illusions), which are woven into the incoherent fabric of his rambling thought. In (*b*) *delirium tremens* the patient is more restless; tremors of the limbs and of the muscles of the face are often easily induced; hallucinations of sight and hearing are common; and the character of the delirium reveals that the patient is, to an unusual extent, possessed by fears, terrors, and other emotions of a depressing type. In (*c*) *wild* or *raving* delirium we have to do with a much more active state. The patient raves loudly and incoherently, more in regard to his fleeting dream-like thoughts than in connection with external impressions, of which he is more or less heedless. He is often violent in demeanour, and difficult to be restrained,

persons in this state being capable of great and prolonged muscular exertion. The bodily activity accompanying this form of delirium is, in fact, just as characteristic as the great intensity of the mental processes. It is met with occasionally in some fevers, but more commonly in meningitis and in acute mania.

In its early stages delirium is principally noticed during the transition-period between waking and sleeping—at times, that is, when the nervous system most needs the re-invigorating influence of sleep. It is in these cases, too, that beef-tea or stimulants may for a time dispel all traces of the wandering thought. While illusions and hallucinations enter largely into the mental activity of a delirious patient, *delusions* also are generally well-marked components. That is to say, the person becomes for a time possessed by an idea, notion, or fancy, for which there is no real warranty, though he believes and wishes to act as though it were true.

Somewhat allied to delirium in nature, though much lower in intensity as a mental process, is that *incoherence* of thought which is met with in many chronic maniacs, or in non-febrile patients suffering from various organic brain-diseases. In its slightest degrees this incoherence displays itself as mere 'rambling' talk; the patient has not sufficient brain-power to follow up the main subject of thought, and is frequently diverted into collateral channels. This, which is a natural state with some persons, may be distinctly indicative of disease in others whose mental power has previously been of a more vigorous type. At times the incoherence is seen to be governed principally by mere verbal suggestion, the patient being led away from point to point in new directions, owing to the associations of some word which has been used becoming for the time dominant. This state is often well seen in the sub-acute exacerbations of chronic mania, though it may occur also where multiple softening or indurations of the brain exist. At other times the incoherence is more absolute—wayward transitions from subject to subject, connected by no discoverable bond, rapidly following one another. The result in such a case is a mere unmeaning jumble of words, interspersed here and there with brief propositions having a limited significance of their own, though often wholly unrelated to that which precedes or follows.

Hypochondriasis is a perverted state of consciousness, having some resemblance to that of illusion, but in which some internal or visceral state becomes the starting-point of impressions (possibly not actually painful) which, when magnified and perverted as they are in the mind of the patient, fill him with false and gloomy apprehensions of various kinds. This perversion of consciousness is more generalised than that which exists in the case of illusion; and also, instead of being a more or less temporary defect, it is one that may last for weeks, months, or even years. The state of mind of an hysterical patient is often not altogether different from that of the hypochondriac.

3. Partial Loss.—Defects of this order are numerous and may exist in great variety. They may implicate almost equally nearly all the varieties of conscious mental activity, or some more than others. They may be either congenital, or acquired during the life of the individual.

In *idiocy* we may have from birth defect in the power of concentrating the attention, a defective

power of apprehension and of thinking, and a defective volition, shown alike by an inability to guide or control thought, and by a deficient vigour of bodily movement. Again, as a result of epilepsy, of organic brain-disease, or of injuries to the head, the patient may gradually lapse into such a condition from one of health, so as to become, as it is termed, 'demented.' While this state of *dementia* may supervene at any age, it is much more common as a consequence of the brain-diseases frequent in advanced life. There is, moreover, a form known as *senile dementia*, in which without any typical disease, but as a consequence of impaired tissue-vitality and diffused degenerative changes throughout the nervous system, the mental faculties undergo a more marked degradation than is usually met with in old age. This condition in its minor degrees goes by the name of *hebetude*. In all such states or grades of idiocy and dementia, we meet with an undue tendency to sleep in the daytime as a result of the listless and languid mental condition. This is but another sign of the general lowering of conscious vigour.

Here we must include, also, a peculiar group of conditions, having some alliance with one another, which are all characterised by loss of consciousness to some extent, either partial in range or general. They are—*reverie*, *somnambulism*, *ecstasy*, *coma vigil*, *cataplexy*, *hypnotism*, and *trance*. They are merely enumerated here, but are defined or described in their several places. In the last of these conditions the loss of consciousness, in the ordinary acceptance of the term (namely, loss of perceptive power), is so absolute, that some may think it ought rather to be included in the next section. Loss of perceptive power, however, would not seem to be absolutely synonymous with loss of consciousness. There is good reason to believe, for instance, that where the influence of chloroform and other anaesthetics is not pushed to the fullest extent, a condition of *anesthesia* intermediate between slight and profound is produced, in which, while there is absolutely no consciousness for external impressions, so that pain is altogether unfelt, there is still a certain amount of cerebral activity—as evidenced by rambling and indistinct speech on subjects altogether apart from what the surgeon may be doing. There is mental activity clearly, though the nature of this, as revealed by the patient's speech, may preclude the notion that pain is at the time being felt. Sensorial consciousness is blotted out, while a kind of ideational consciousness remains. We have an approximation to such a condition, also, in the case of *sleep* when dreams are rife. But here sensorial consciousness is not completely in abeyance. Again, in certain rare and anomalous epileptiform attacks we may find the patients, after the first paroxysms, bereft of some senses, though not of others. They may hear what is said by those around them, though they continue for a time quite unable to see or speak.

4. Complete Loss.—In very profound sleep (*sopor*), in that prolonged form of it in which the person, if he can be momentarily roused, drops off again immediately (*lethargy*), and also in profound *anesthesia*, there is complete loss of consciousness. The terms 'sopor' and 'lethargy' are now rarely used, and authors are not even agreed as to the precise state which would be designated by the latter word. It is sometimes regarded as a synonym for 'trance.'

In *syncope* we have insensibility resulting from a cutting off of the proper supply of blood to the brain; while in *asphyxia* we have a like result following upon an interference with respiration, and a consequent engorgement with impure blood.

A condition of *narcosis* or profound insensibility may result from opium or other drugs and poisons, among which alcohol is to be included as one of the most common producers of such a state. It may also be due to the deficient elimination of waste products by the kidneys, when 'uræmic coma' is produced; or to the abnormal production of chemical compounds in the system, which lead on to blood-poisoning, as in 'diabetic coma,' and in the final stages of septic poisoning.

Complete loss of consciousness exists for some time during the ordinary form of *epileptic fit*, or during an attack of convulsions; though in other epileptiform fits, not infrequently met with—having some of the characters of hysterical convulsions—there seems to be a loss of sensorial consciousness only (loss of perception), while a certain amount of ideational consciousness remains. In *apoplexy* also there may be for hours or days a more or less profound loss of consciousness. In the less profound attacks, as well as after an epileptic fit or an attack of convulsions, the loss of consciousness is not complete, and we have a condition now commonly known as *stupor*. This state is also frequent as a result of concussion or other injuries of the brain, and it occasionally follows a severe fit of hysterical convulsions. It may last for hours, days, or even weeks in some cases. In it the patient lies with his eyes closed, taking no heed of what is passing around, though he may show obvious signs of feeling when touched or pinched, and may be capable of being momentarily roused, so as to give a short monosyllabic answer if slightly shaken or spoken to in a loud voice. On these occasions, signs of impatience are often shown. Though such a patient will not ask for food, he will often drink freely when it is offered. He will of his own accord, when his bladder is full, sometimes get out of bed; find the chamber-pot, use it, and return to bed without saying a word—and then speedily relapse into his previous state of stupor. When the insensibility is more profound, both urine and feces are passed incontinently.

The state just spoken of is referred to in this section because it is so intimately allied to and connected by all sorts of transition conditions with another, known as *coma*, in which the loss of consciousness is more complete and absolute. There are different degrees of stupor and there are different degrees of coma; the former is commonly spoken of as slight or deep, while a comatose condition, coma, and profound coma (the latter being what the older writers termed *carus*) are the phrases ordinarily used to denote the increasing insensibility of the graver state, which is more especially characteristic of the apoplectic condition. Coma may result from long-continued exposure to cold, from sunstroke, from poisons of various kinds, from erysipelas of the head and face, from inflammations of the meninges, multiple embolisms, the effects of hyperpyrexia, or from cerebral hæmorrhage. The most common cause of very profound coma is cerebral hæmorrhage (apoplexy). In this condition the breathing is often loud and stertorous, and consciousness is entirely obliterated, so that there is an utter absence of reflex movements when

a limb is pinched or when the conjunctiva is touched. The patient in the deeper forms of coma often cannot be roused at all, even for a moment; and if this state does not terminate in one way or another before the expiration of twenty-four hours, or if it does not gradually pass into one of mere stupor, a fatal result may be considered imminent.

H. CHARLTON BASTIAN.

CONSTIPATION (*con*, together; and *stipo*, I cram).—**DEFINITION.**—Slow passage of the feces from the cæcum to the anus, leading to infrequent or incomplete alvine evacuation, and to retention of feces.

ÆTIOLOGY.—The causes of constipation may be *local*—an impediment to the onward movement of the feces in the large intestine or from the rectum; or *general*—pertaining to habits, diet, and other conditions.

Local.—The local causes of constipation include: (a) Lesions inducing narrowing of some part of the large intestine, as from cicatrising of an ulcer, new-growths, or displacement of the bowel. (b) Collections of scybala, intestinal concretions, &c., in the cæcum, sigmoid flexure, or rectum. Fæces are more especially apt to collect when too solid or when deficient in bile. (c) Pressure on the rectum, by uterine fibroids or ovarian tumours, uterine displacements, the gravid uterus, or an enlarged prostate. (d) Defæcation thwarted, as when the expiratory abdominal muscles are enfeebled, as in pregnancy—especially when repeated or after twins, obesity, old age, or in some painful affection of the abdomen, such as rheumatism of the abdominal walls and diaphragm, chronic dysentery, piles, fissure, or the evacuation of a hard mass of feces. (e) Feeble contraction of the intestinal muscular fibres, as in distension of the large intestine or a portion of it by gas, feces, or lumbrici, inflammatory affections (chronic intestinal catarrh, chronic peritonitis), lead-poisoning, senile atrophy, or in delicate females with lax muscular fibre. (f) Pain in the pelvic viscera, and probably elsewhere, may induce paralysis of the sympathetic nerves supplied to the intestinal walls; thus may be explained obstinate constipation in painful uterine and ovarian diseases, which cannot be accounted for by pressure on the bowels or otherwise.

General.—The general causes of constipation are: (a) Sluggishness of function—lymphatic temperament, hereditary influence, anæmia, especially with amenorrhœa; or, on the other hand, disposition to great activity of the muscular and nervous system. (b) Certain habits, such as sedentariness; railway travelling—long journeys; too great muscular activity; mental application, especially when excessive or prolonged; the continued and apparently necessary use of aperients or enemata after the relief of temporary constipation—falling under the tyranny of aperients; habitual disregard of defæcation, or hurry in the act; prolonged hours of sleep; the excessive or even moderate use of alcohol, tea, tobacco, or opiates. (c) Dietetic errors: the diet too nutritive—leaving little intestinal residue, or poor and insufficient; or containing too little fluid; improper feeding, especially in infants and children; and the use of indigestible substances, such as cheese, nuts, or uncooked vegetables. The presence of lime-salts in drinking water is a fertile cause of constipation in many districts.

Constipation is frequently a prominent symptom

n diseases of the stomach; of the liver; of the heart, inducing congestion of the portal system; and of the nervous system; in acute febrile states; as well as in connection with diabetes, excessive perspiration, prolonged lactation, and morbid discharges.

The causes of constipation are such as evidently induce one or all of the following conditions, namely, (1) increased resistance to the onward passage of feces along the intestine either from dryness or hardness of its contents resulting from deficient secretion or too active absorption of fluid; or to actual narrowing of the lumen of the bowel; and (2) impaired contraction of the muscular fibres of the large intestine.

DESCRIPTION.—In constipation the evacuations are infrequent, solid, deficient in quantity, and sometimes unusually offensive; they often consist of dry, hard, dark or clay-coloured masses or scybala. Defecation is generally difficult or even painful. As a rule the depth of colour, and the scybalous character of the motions, are in proportion to the duration of the lodgment of feces in the large intestine. Infrequency of defecation regarded alone is an untrustworthy sign of constipation, or of constipation demanding medicinal or other treatment, inasmuch as it often depends on individual peculiarity. Good health may be consistent with departures from the ordinary rule—a daily evacuation; not infrequently there is no relief from the bowels for several days or even for a week, and yet without inconvenience, so long as the infrequent defecation is habitual or can be ascribed to idiosyncrasy.

The disturbances of function usually associated with constipation may be local or may extend to distant parts.

The *immediate* or *local* effects are such as may arise from retention of feces:—signs of fecal collections in the cæcum, colon, sigmoid flexure, or rectum; irritation of portions of the intestine, indicated by colic, inflammation, dilatation, ulceration, and perforation of the intestines, followed by peritonitis; intestinal obstruction—a portion of the bowel loaded and distended with feces being no longer capable of peristaltic movement; pressure of fecal accumulations on the intra-pelvic vessels and nerves, inducing menorrhagia, uterine catarrh, seminal emissions, varicocele, hæmorrhoids, cold feet, neuralgia, numbness, and even œdema of the legs (see FÆCES, Retention of). Constipation frequently exerts a pernicious influence on primary digestion, indicated by foul tongue, foetid breath, anorexia, acidity, flatulence, biliary disturbance—even jaundice—and urine loaded with lithates.

The *remote* or *general* effects of constipation are lassitude of body and mind; headache, flushing, heat of head, and vertigo; palpitation; anæmia, and wasting. Headache and nausea depending on retention of feces may be either pretty frequent without loaded tongue; or may recur with vomiting and much coating of tongue every three or more weeks—these periodical attacks resembling migrain, but always cleared up by an aperient.

TREATMENT.—Constipation depending on individual peculiarity is rarely relieved permanently by treatment. The bowels, having acquired from early life the habit of infrequent evacuation, may be stimulated for a time, and are then apt to become more sluggish than before. In all cases the habit of the patient in this respect from childhood should first be determined, either as a caution against

active or prolonged treatment, which may prove injurious, or as a guide to the adjustment of directions and remedies—affording as it does a limit which should not be overstepped. A healthy daily discharge of feces should not fall far short of five ounces, and should form a coherent cylindrical-shaped mass of five or six inches in length, which should float; and it is an essential condition of healthy and efficient defecation that the colon should always be moderately full of retained feces. The practitioner should bear in mind that many persons acquire the erroneous belief that they are not discharging a sufficiency of feces, and in consequence betake themselves to aperients, which daily remove the normal residuum of feces that should exist in the colon—healthy peristalsis and defecation being thus out of question. This apparent constipation can only be met by completely suspending the use of aperients.

1. *Ætiological, hygienic, and dietetic treatment.*—In treating constipation the causes should be met. Local causes, such as those inducing contraction of or pressure on some part of the large intestine, or feeble or ineffectual contraction of the intestinal muscular fibres or of the expiratory muscles, should first be eliminated. When defecation is obstructed by the sphincter ani either remaining firmly contracted, or failing to relax sufficiently, it may be effectually relieved by forcibly dilating the anus under ether. Habits disposing to constipation should also be corrected. Persons who are much preoccupied or careless are apt either to disregard the call to stool, or to perform the act of defecation hurriedly, incompletely, and at irregular intervals. Such persons should be strictly enjoined to direct their attention, expectancy, and will to the attainment of complete and regular evacuation. The sensibility of the nerves of the rectum becomes blunted by the constant contact of feces, and the promptings of nature at last cease. Hence the periodical removal of collections in the lower part of the large intestine is an essential element of the treatment. This is best secured if possible by well-timed and well-directed and sustained natural efforts. The patient should be told to attempt defecation every day after breakfast or other suitable time, and to persevere in this habit even though the result be occasionally or frequently unsuccessful, and in order to develop his solicitation into the daily habit of evacuation he should continue to practise it always at the same hour, and should disregard any prompting that may arise at other times. The position in which the act is performed is important and should be made to resemble the natural attitude as far as possible by the use of a low seat or a foot-stool; for thereby less impediment is offered to the passage of the feces, and the abdominal walls are well supported during straining. While straining to relieve the bowels, he may facilitate evacuation by pressing firmly the fingers in front and on each side of the coccyx, thus supporting the levator ani during contraction, reducing the concavity of the rectal pouch, and causing the mass of feces to glide forwards through the sphincter, which should be smeared with vaseline; or he may attain the same end by alternately contracting or relaxing the anal sphincter. Failing to obtain relief on the second day, a small cold-water enema should be used to prevent further accumulation of feces in the rectum, and to restore tone in this region. The enema should never be larger than is required to dislodge

the motion from the pouch of the rectum, nor should it be warm; at first it may, however, be tepid, but should afterwards be cold. Occasionally large enemata may have to be employed to dislodge fecal accumulations high up in the colon. Such enemata should be given by a continuous pressure under the force of gravity from a suitable douche-can. When evacuation is obstructed by the lower part of the fecal mass becoming dry, relief may be obtained from a small emollient enema administered on rising (such as infusion of linseed, glycerin in small quantities, solution of white of eggs, olive oil alone or in oatmeal gruel or combined with ox-gall) and allowed to remain until after breakfast; or by the use of suppositories at bed-time consisting of cacao-butter, or honey hardened by heat, either alone or combined with extract of belladonna. The clothing should not constrict the abdomen or waist, and should be warm—especially about the feet and legs. Sedentary habits should be abandoned. Exercise on foot or on horseback, or on a cycle, is especially to be commended, and carriage-exercise to be avoided. While studying or reading, the patient should walk about, and stand rather than sit at the desk. Gymnastics and out-of-door games (such as lawn-tennis, cricket, football, golf) are useful when a limited time only can be devoted to exercise. Excessive and exhausting exertion should be avoided. It is generally advisable to recommend early rising and cold bathing, followed by brisk general towelling in the morning. In different cases one or other of the following may be found serviceable: a shower- or sponge-bath containing vinegar or bay-salt, or consisting of sea-water, or a cold sitz-bath; douches directed to the abdomen; a cold-water compress applied to the abdomen during the day or night, or from three to four hours in the morning; friction or kneading, or slapping with the half-closed fist in the course of the colon every morning, and when at stool; an abdominal belt (flannel or elastic)—especially if the abdomen be pendulous.

Diet.—The diet should be varied, should not consist too exclusively of animal food, but should contain a good proportion of fresh vegetables, and especially green vegetables (cabbage, lettuce, spinach). Fresh and ripe fruit (such as grapes, apples, oranges, figs, and bananas) should also find a prominent place, and are most effective when taken in the early morning, or at breakfast. When fruit induces acidity a tumblerful of alkaline (Vichy or Vals) water should be taken at the same time. Oils and fats are generally serviceable when they do not disturb the digestion, such as the addition of a dessert-spoonful of olive oil to green vegetables or to potatoes. A tea-spoonful of glycerin with each meal is often helpful. The diet should not be too dry. As to beverages, much must be left to individual experience: sometimes coffee, or beer, or cider answers best. A tumblerful of cold water should be taken at night, and in the morning on awaking; sometimes carbonic-acid water in the early morning is found preferable to plain water. Coarse articles of food—such as bran- or wheatmeal bread, oatmeal porridge, &c.—are often commended, and now and then they do favour the continuance of more efficient evacuation; more frequently, however, they fail—especially after a time—and they then aggravate the evil by further adding to the undue collection of residue in the large bowel, and are apt to set up catarrhal irritation. Acidity and other

symptoms of dyspepsia contra-indicate the use of these indigestible foods. Honey or marmalade with bread, gingerbread, and Spanish or Portugal onions, plainly boiled, may be found useful in certain cases. Dujardin-Beaumez recommends infusion of linseed immediately before a meal. As a rule, eggs, milk, cheese, farinacea, astringent red wines, and tea increase constipation; and besides these articles it is best to avoid pickles, salted meats, nuts, and pastry in any form.

2. **Treatment by massage and electricity and gymnastics.**—Many observers in recent years have reported the complete cure of constipation by a course of massage alone. The manipulations consist of kneading (*pétrissage*), stroking (*effleurage*), and slapping (*tapotement*), each having its special effect: the first loosening impacted fecal masses, the second favouring their passage along the bowel, and the third improving peristaltic action. In some instances the forms of massage best adapted to the case should be applied to the whole of the large bowel from the cæcum onwards; in others it should be addressed specially to certain portions, as the cæcum, transverse colon, or sigmoid flexure. Sometimes all the manipulations, in varying proportions and degrees, are required in succession, or only one or two kinds—for instance, in atony of the bowel depending on sedentary habits, gentle and then deep stroking should give place to strong slapping with the half-closed hand; and in cases marked by flabbiness of the abdominal muscles, the recti should be forcibly separated by the thumbs and fingers, and then strong transverse strokings from the median line should be made, and the patient should take deep inspirations to strengthen the diaphragm. Each case should therefore be treated with discrimination, and a routine method should be avoided. The massage should be applied for from twenty to thirty minutes every or every other morning, and should be continued without interruption for from five to eight weeks. **Electricity**, either alone or with massage, has also proved successful in certain cases; a mild galvanic (continuous) current being applied by inserting the negative pole well within the sphincter ani, and drawing the positive pole along the colon, and the faradic (interrupted) current being passed through the abdominal muscles.

Properly selected and regulated gymnastic exercises may in some cases be indicated, and by restoring the tone of the abdominal muscles prove a useful addendum to other remedial measures.

3. **Medicinal treatment.**—Should the foregoing directions fail to establish the habit of a regular action of the bowels, either daily or on alternate days, they may be supplemented by the prescription of medicinal agents, to compass the twofold aim (1) of relieving the large bowel of the excess of feces and gases which, by over-distending it, opposes peristalsis, and (2) of giving tone to its walls. To this end care should be taken to so adjust the dose (for it varies considerably in different cases) as to secure if possible efficient assistance to the discharge of feces, without going so far as to clear away or to considerably reduce the normal residuum which should always exist in the colon. If, therefore, any prescribed dose has exceeded this limit, it should be reduced, and should not be given next day, but after the lapse of a day or two. Repeated purging exhausts torpid bowels, and perpetuates constipation. Nor should the bowels be pushed to more frequent relief than has been habitual with the

patient from early life. If the evacuations habitually contain scybala, and if feces are generally found, on examination, in the rectum, the injection of a teaspoonful of glycerin, or the insertion of a glycerin suppository, twenty minutes or so before the time of the desired evacuation, may alone suffice, or may enable a smaller dose of medicine to deal effectually with the difficulty. If gentle assistance only is required, and if griping and other signs of irritation are readily set up by the ordinary aperients, one or other of the following may be tried:—a teaspoonful of castor oil alone, or with an equal part of glycerin, beaten together and then added to a tablespoonful or two of milk, or with olive oil or almond oil, at bedtime or in the early morning; the official sulphur lozenges at bedtime; rhubarb and dried carbonate of sodium (gr. ij. or iij. of each), or a small piece of rhubarb-root before the midday meal; pills of dried ox-gall and soap. Or, if a more decided laxative is necessary, a preference may be given to the compound liquorice powder, or confection of senna (with or without confection of sulphur or the sulphur lozenge), or the infusion of a few senna-pods in half a tumbler of water let stand over night, or extract of cascara sagrada. During late years cascara sagrada has acquired a high position as a tonic aperient, and is prescribed either in the form of solid extract, in pills, alone or variously combined, or in that of the liquid extract—the dose of the latter varying from *m̄v.* to *m̄xxx.* alone or associated with tincture of *nux vomica*, tincture of belladonna, or tincture of podophyllum, in a teaspoonful of glycerin, or along with dried extract of euonymin in pill. Combinations on the lines of the favourite formula of Sir Andrew Clark also meet the indications well in the majority of cases (aloin, extract of *nux vomica*, extract of belladonna, sulphate of iron, powdered myrrh, powdered soap, of each half a grain; and, if the feces are dry and hard, add powdered ipecacuanha half a grain). Some prefer a pill of Socotrine aloes with henbane and sulphate of iron and quinine. In a well-organised course of medicinal treatment the form of the aperient should be varied every now and then, and the dose very gradually reduced until at last it is almost withdrawn, while the intestinal tonics (belladonna, *nux vomica*, and iron) should be continued. The time when the dose should be taken is important, for it will vary—be it before lunch, or dinner, or at bedtime—with the slowness or quickness of the response of the bowels to the attainment of relief after breakfast.

In anemic patients a prolonged course of hæmatics should be aided by aloes, *nux vomica*, and arsenic. The official *Pilula Aloes et Myrrhæ*, *Mistura Ferri Composita*, and *Decoctum Aloes Compositum* are all excellent preparations for such patients. In hæmorrhoidal complications aloes should, as a rule, be avoided, and laxative electuaries should be prescribed. In obstinate cases colocynt—either alone or variously combined—is most useful; such as the tincture of the Prussian Pharmacopœia, *m̄v.* or more on sugar or in liquid extract of liquorice (to disguise the bitterness) three or four times a day, or 10 to 20 minims an hour before breakfast; or the official extract or compound pill with small doses of calomel. A full dose of opium may liberate the bowels after the failure of the strongest purgatives, and constipation depending on inhibition of the sympathetic nerve from pain will be relieved by opium with belladonna. When

constipation is associated with a deficient flow of bile, cholagogues (such as calomel, euonymus, or podophyllum resin) should be prescribed with the other remedies; and dried ox-bile in pills, and nitric acid or nitro-hydrochloric acid often prove useful. Sometimes enemata (simple or medicated) answer better than purgatives; then, when frequently required, they should be small in quantity, and at first tepid, afterwards cold; for occasional use, for the purpose of clearing away feces loading the large bowel, they should be large (from two to six pints) and warm (*see FÆCES, Retention of*). The frequent use of large warm injections perpetuates constipation. Purgative waters, such as the Rubinat, Condal, Friedrichshall, Pullna, Apenta, Hunyadi János, or Carlsbad, given well-diluted in warm water in the early morning, are often valuable adjuncts to other treatment. A combination of the sulphates of iron, magnesium, and sodium with strychnine and belladonna, after food twice a day, is often efficacious; or a course of sodium phosphate and ammonium chloride; and in young children carbonate of magnesium (gr. v. to *xx.*) in milk, compound decoction of aloes, or syrup of senna are favourite aperients.

GEORGE OLIVER.

WILFRED EDGEcombe.

CONSTITUTION.—*SYNON.*: Diathesis; Habit; Conformation of Body; *Fr. Constitution*; *Ger. Leibesbeschaffenheit*.

The constitution may be *sound* or *unsound*. A sound constitution may be defined as the harmonious development and maintenance of the tissues and organs of which the body is made up. It originates with the union of healthy sperm- and germ-cells, continues with the growth of the product under the most favourable conditions to adult life, and becomes gradually enfeebled with advancing age by the process of natural decay.

The constitution may be unsound in consequence of *deficient vitality*. This deficient vitality may be general, as is sometimes observed in the children of parents one or both of whom are in advanced life, or whose vitality on one side or the other has been reduced by excesses, such as alcoholic or venereal. Exhausted vitality from prolonged disease, e.g. phthisis or tertiary syphilis, affecting either parent, may determine the death of the offspring at an early period, from mere failure of nutrition, or may cause it to succumb to acute disease not necessarily associated with any inherited tendency of a special kind. The deficient vitality may be restricted to certain tissues or organs—namely, those concerned in the nervous, vascular, respiratory, or digestive systems. Thus among the most strikingly hereditary of diseases are those of degeneracy, such as emphysema, structural heart-diseases, atheroma of vessels, and certain kidney-diseases. Rightly interpreted, these diseases are of the nature of premature senility, attacking certain tissues or organs—as it may be seen to attack the hair or the cornea—from some inherent defect in their vitality.

The constitution may, in the second place, be unsound from some definite *inherited* form of disease. Although the constitution of an individual begins with his life, it is nevertheless the resultant of the constitutional peculiarities of many antecedents. This being so, tendencies to disease may date far back in the pedigree, to be called forth from time to time by favouring circumstances. We need,

however, practically only go back a few generations in inquiring for those diseases which are well recognised as being hereditary. These form one section of the group of *constitutional diseases*.—Congenital syphilis, gout, scrofula, tuberculosis, cancer, asthma, and certain neuroses, are all diseases which are apt to appear at certain periods of the life of the offspring, in consequence of some specific inherent defect of blood or tissue derived from his progenitors.

The constitution may, thirdly, become unsound at any period *subsequent to birth*. (a) This may be due to the surrounding conditions of life being evil. Deficient or impure air, insufficient or improper food, defective sunlight, overwork, intemperance, &c., may injure the constitution, and give rise to diseases whose constitutional nature is sometimes strikingly shown in the tendency of some of them to become hereditary. Rickets, phthisis, and scrofula are examples. (b) The introduction of certain poisons into the system affects the constitution profoundly, and in some cases permanently, after the more obvious effects of the poisons have passed away. All the acute specific zymotic diseases, including vaccinia, would come under this category. They render the organism, for a long period or for life, proof against subsequent attacks of the same disease. Only in certain cases, however, can the soundness of the constitution be said to be *impaired* by such diseases, and then it is usually through the occurrence of sequelæ. R. DOUGLAS POWELL.

CONSTITUTIONAL DISEASES.—These may be regarded as diseases generated from within, in the course of the wear and tear, nutrition and waste, of the body, in consequence of inherent or acquired weakness in its construction.

The applicability of the term 'constitutional' to disease is sufficiently explained in the preceding article on **CONSTITUTION**. The term may, however, be associated with a group of so-called 'general' diseases, in opposition to that which includes 'zymotic' or 'specific' diseases, which are generated by the introduction of some definite poison from without.

In our present state of knowledge, however, no very rigid lines can be drawn to separate local, general, constitutional, and specific diseases from one another. R. DOUGLAS POWELL.

CONSTRICION (*constringo*, I bind together). A narrowing, to a limited extent, of a canal or hollow organ, due either to a textural change in its walls, or to the pressure of a band surrounding it.

CONSUMPTION (*consumo*, I waste).—A term for any wasting disease, but generally applied to pulmonary tuberculosis. See **LUNGS**, Tuberculosis of.

CONTAGION.—See **INFECTION**.

CONTINUED FEVERS.—An almost obsolete expression employed to designate a group of fevers, comprising typhus fever, typhoid fever, and relapsing fever.

CONTRACTURE, Muscular (*contraho*, I draw together).—A term applied to the permanent shortening of a muscle from any cause. See **SPASM**.

CONVULSIONS IN CHILDREN

CONTRA-INDICATION.—Any circumstance which forbids the employment of therapeutic measures otherwise indicated.

CONTRE-COUP (Fr.), Counter-stroke.—An injury of a part, opposite to, but distant from, that to which force is applied, as by a fall or direct blow. Contre-coup is chiefly observed in injuries of the skull.

CONTUSION (*contundo*, I bruise).—A bruise or injury of the soft parts without breach of surface.

CONVALESCENCE (*con*, with; and *valesco*, I grow well).—The *period of convalescence* signifies that period during which a patient is progressing towards recovery, and is returning to a state of health after having suffered from an illness. The word is used most commonly in association with fevers, inflammatory diseases, and other acute affections.

CONVOLUTIONS OF THE BRAIN, Lesions of.—See **BRAIN**, Tumours of; and **INSANITY**, Pathology of.

CONVULSIONS.—SYNON.: *Eclampsia*; Fr. *Convulsions*; Ger. *Krampf*.

DEFINITION.—This term is commonly given to more or less general, purposeless, muscular contractions, occurring simultaneously and successively for a variable time—constituting a 'fit.' It is occasionally applied to certain localised purposeless contractions, though these are more appropriately termed *spasms*. See **EPILEPSY**; and **SPASM**.

CONVULSIONS IN CHILDREN.—**ÆTIOLOGY.** In the earlier months and years of life convulsions are frequently met with. Of these no classification, which is at the same time scientific and useful, exists. The principal point of practical importance in dealing with any given case is to discover the share taken by the different causal agents in the production of the attack. For convenience of description these will be placed in three groups:—(1) predisposing causes responsible for a certain instability of the nerve-centres; (2) direct and often trivial and removable exciting causes; and (3) serious and often unsuspected organic disease.

1. Predisposing Causes.—(1) *Age.*—During the first two years of life convulsions may be induced by any of the exciting causes which will presently be enumerated, although it is usual to find more than one predisposing cause present. The influence of age gradually declines and, with the exception of hysterical convulsions, disappears at puberty.

2. Heredity.—The natural instability of the nervous system in infants is much greater in some cases than in others. Children prone to convulsions show muscular twitching and hypersensitiveness to light and sound at the slightest provocation. In many instances this temperament seems due to abnormal conditions in the parents; and a family history of epilepsy, alcoholism, any of the so-called functional nervous diseases, or some marked disproportion between the ages of the parents is not infrequently present.

3. Rickets.—Convulsions occur so frequently in the subjects of this disease that it is generally admitted that one of the changes in rickets is an increased instability of nerve-centres.

4. Intra-cranial Disease.—The relation of brain-

disease to convulsions is usually that of an exciting cause; but occasionally, when a convulsion is apparently due to some trivial exciting cause, the absence of other predisposing causes and the subsequent development of meningitis or new-growth renders it almost certain that the irritant acted on nerve-centres, the stability of which was lessened by organic disease.

II. Directly exciting causes.—These are usually connected with the ear, the mouth, or the alimentary tract. Irritation of the external ear has been credited with the production of convulsions, but inflammation of the middle ear is unquestionably a commoner cause. Teething is occasionally the responsible agent, but only when some clearly recognisable lesion of the gums is present. Defective dentition may be associated with convulsions, but has no causal relationship except in so far as it is a manifestation of rickets. Irritation of the mucous membrane of the stomach and intestine is the most frequent of these causes. Stones and other foreign bodies, indigestible articles of food, and worms, especially ascarides, are the commonest sources of the irritation. It must, however, never be forgotten that any irritation in ear, mouth, stomach, or intestines is unlikely to produce a convulsion without the co-existence of one or more of the predisposing causes already mentioned. Narcotic poisons, poisonous fungi, and burns (*see* AUTO-INTOXICATION) may cause convulsions without the existence of any predisposing influence.

III. Organic disease.—(1) Convulsions frequently mark the onset of acute febrile diseases including not only the various acute specific disorders of childhood, and lobar pneumonia, but infantile paralysis either of the cerebral or the spinal variety. (2) A convulsive attack often preceded by muscular twitching may be the first indication of uræmia, especially in cases of unrecognised scarlet fever. (3) The circulatory disturbance caused by sudden collapse of a considerable portion of the lung, such as occasionally happens in whooping cough, may result in a convulsive attack. Somewhat allied to this are the convulsions that may occur in post-diphtheritic paralysis. These are due to syncope, depending on heart-failure, and are almost invariably fatal. In whooping cough a convulsion may also occur as the result of intracranial hæmorrhage. (4) Organic brain-disease, including meningitis, new-growths, and cortical vascular lesions, may lead directly to convulsions. (5) Epilepsy may for the sake of completeness be included, though no definite change in the brain has been associated with this disorder.

DIAGNOSIS OF CAUSE OF CONVULSIONS.—It is frequently impossible to make an exact diagnosis on the day a fit occurs. If an opportunity is afforded of observing the convulsion, the following points should be first noted: whether the attack involves all the muscles generally; whether the convulsed parts are affected with uniform intensity; whether consciousness is absolutely lost or to what extent the child can be roused; whether there is any change in colour—pallor, or cyanosis; and whether the rectal temperature is much raised.

The previous history should next be ascertained, including the age of the patient, the condition of its previous health, and the exact character of the onset of the present attack. Inquiries concerning previous health should include the possible occurrence of previous fits, and the existence of sore

throat, rash, whooping cough, improper ingesta, vomiting, constipation, headache and giddiness, and, in infants, rolling or retraction of the head. If the onset of the attack was observed, inquiries should be made concerning the part first affected.

When the child is undressed, its hands and feet should be examined for evidence of œdema and desquamation, its chest for signs of rickets, collapse of the lung, or pneumonia. As soon as the convulsion has subsided, the limbs and face of the patient should be carefully examined for paralysis. If the mouth and urine are normal, and no satisfactory cause for the convulsion has been ascertained, the ears must be carefully examined. After twenty-four hours the temperature should be again taken, an ophthalmoscopic examination made, and any deviation from complete recovery noted.

The diagnosis in a case of convulsions must depend upon the discovery in this way of evidence of one or more of the exciting or predisposing causes already mentioned. It is practically useless to attempt to refer the case to any one of the special lists sometimes suggested. Even when due to local disease in the brain, convulsions in a child are usually general. If, however, the onset should be local, the distribution partial, the consciousness even for a time retained, and any weakness of the convulsed parts result, local disease of the brain or spinal cord is undoubtedly present, the nature of which must be determined in the usual way according to the evidence subsequently obtainable (*see* BRAIN, Tumours of). The diagnosis of epilepsy should not be made unless all other causes can be excluded, or unless the fits recur without any assignable cause.

PROGNOSIS.—As a rule convulsions are not immediately fatal. If, however, a series occur without any discoverable exciting cause and without the presence of any known organic disease, the prognosis both as regards life and subsequent mental development is grave. When convulsions are incidents occurring in the course of organic diseases, they often mark the occurrence of some complication, and are accordingly of unfavourable omen; when dependent on organic brain-disease complete recovery is rare.

TREATMENT.—If any organic disease has been detected the treatment of the convulsion must be subordinated to that of the general condition. In the absence of such indications it is customary to place the child in a warm bath (95° F.), the temperature of which must be carefully adjusted, as in the hurry of the moment mistakes are liable to be made. The utility of the bath is questionable, but it is devoid of danger except when the convulsion is due to syncope or collapse of the lung. If the rectal temperature be high, a somewhat cooler bath may be employed with advantage. Should a bath be contra-indicated, the child should be put to bed and care taken that no accidental injury occur.

In the majority of cases the convulsion ceases in the course of half an hour; so that, by the time assistance is procured, drugs can generally be administered by the mouth. But when one convulsion rapidly succeeds another, or when unconsciousness persists, inhalations of chloroform or rectal injections of chloral hydrate and bromides may be administered, except in those cases in which heart or lungs are affected. Indeed, the routine administration of drugs is only applicable when no organic disease and no removable cause can be discovered. In such cases bromides in large doses and chloral

hydrate, with or without digitalis, may be given by the mouth, and when these fail the administration of phenazone or phenacetin is occasionally attended with good results. Emetics and purgatives may be needed to remove the contents of stomach or intestines. A grain of calomel placed on the tongue is often administered as routine treatment in all cases—in many of them with excellent results. If the fit does not recur, it is still advisable to continue the administration of bromides for some days, combined with a light diet and as complete bodily and mental rest as may be possible. For the treatment of convulsions depending upon organic disease, the reader is referred to the special articles on epilepsy and the various organic diseases in which they occur.

H. MONTAGUE MURRAY.

CO-ORDINATION.—This term is used in reference to muscular movements principally. Certain parts of the nervous system have more especially to do with the calling into activity, and therefore with combining, the contractions of different muscles, both simultaneously and in succession, in the precise order in which they occur in the several motor acts of which they are capable. The nervous arrangements upon which these actions depend have come into being, both in the race and in the individual, by processes of organic growth and development *pari passu* with the possibility of executing these several movements. It would be wrong to expect, therefore, that an isolated organ should exist solely for co-ordinating muscular movements. The execution of the most habitual of these must depend, to a large extent, upon the activity of the ordinary motor (and related sensory) tracts of the spinal cord and brain. The extent or precise mode in which the cerebellum intervenes in certain higher forms of co-ordination is still involved in much obscurity. That it has some share in such functions may be regarded as certain, though it probably intervenes far less than some would have us believe, who regard the cerebellum as the organ for the co-ordination of muscular movements.

Many nervous affections exist in which the co-ordination of muscular movements is more or less impaired. One of the most familiar of these is locomotor ataxy, a disease dependent upon a morbid process in the posterior columns of the cord. Sclerosis of the antero-lateral columns of the cord also not infrequently disturbs the execution of muscular movements, especially those of the upper extremities. Chorea gives rise to very similar uncertainties in the execution of muscular acts. Spasms of all kinds, in short, tend to interfere with the harmony of the muscular movements in the course of which they intervene. Stammering is an affection of this kind, implicating some of the muscles of articulation; and certain disturbed cardiac actions characterised by disordered rhythm can only be regarded as belonging to the same category.

The above-mentioned are common instances of impaired co-ordination of muscular movements dependent upon structural or functional changes in parts of the nervous system other than the cerebellum. Certain diseases of this organ, however, are known to give rise to a distinct form of inco-ordination. It is characterised by a reeling, unsteady gait in walking, with legs straggling, and mostly wide apart, to which the term 'titubation' is commonly applied. Other kinds of inco-ordination

may hereafter be proved to depend upon diseases of the cerebellum. Its morbid conditions are still very imperfectly recognised, and this is especially true in regard to its merely functional perturbations.

Certain inco-ordinations in speech and writing are common. Instances are to be found in that use of wrong words or misapplication of terms which we meet with in aphasic and amnesic persons; also in the substitution of wrong words in the act of writing, or of wrong letters in the writing of words when such substitution is mechanical and unintentional—and when it is wholly distinct, therefore, from mere inability to spell. These defects are inco-ordinations of a complex kind, dependent upon the perverted action of higher cerebral centres, in the same way that incoherent speech generally is dependent upon incoherent thought. There is reason to believe, indeed, that the same kind of ultimate defective nervous action which leads to inco-ordinations of movements when certain motor regions of the nervous system are affected, may, on the other hand, give rise to perverted perceptions (*illusions*) or to perverted thought (*incoherence*) when the disturbed nervous action occurs in other and higher parts of the central nervous system.

H. CHARLTON BASTIAN.

COPPER, Poisoning by.—SYNON.: Fr. *Empoisonnement par le Cuivre*; Ger. *Kupfervergiftung*.—*Metallic copper* may be regarded as innocuous when swallowed, and the recent researches of Hirt show that those who are engaged in its metallurgy and in the manufacture of copper utensils are not specially liable to any diseases which can be attributed to copper as such. It is, indeed, stated that workers in copper enjoy an immunity from cholera, a conclusion which is based on very insufficient premises. It is contradicted by the occurrence of cholera among coppersmiths in Breslau in 1866, and by certain other cases of a like nature reported by Hirt. That the disease is seldom found among workers in copper is true, but that the copper has anything to do with this result is not proved.

Though pure copper may be regarded as innocuous, it is otherwise with *alloys of copper*, more particularly those with zinc and tin, known under the names of brass and bronze respectively, and with compounds of copper with lead or arsenic. In these the injurious agent would appear to be the alloy, and not the copper itself. An affection of a febrile character, and known as 'brass-founder's ague,' occasionally occurs on fusing days, and it is attributable to the zinc fumes which are generated by the melting process. See BRASS-POISONING.

The *salts of copper*, on the other hand, are capable of causing injurious and fatal results. The more important salts, from a medico-legal point of view, are the sulphate, blue vitriol, or bluestone; the acetates (basic and neutral) constituting artificial verdigris; and the carbonate or natural verdigris. The manufacture of verdigris is carried on to a large extent in the South of France. Plates of copper are acted on by the skins of grapes, which are allowed to undergo the acetous fermentation. Those engaged in this industry on the whole enjoy good health, and it is only rarely that symptoms can be directly traced to the work; and then only when through sheer carelessness and uncleanness quantities of the salt have been ingested. It is even said that dogs eat the refuse grape-skins

without appearing to suffer from poisonous symptoms. On the other hand, symptoms of poisoning of a family have been recorded where they have eaten haricot beans, grown, as is the custom, between the vines, which latter had been watered with a solution of sulphate of copper.

1. Acute poisoning by copper.—**SYMPTOMS.** The salts of copper, when taken in sufficient quantity, cause symptoms of acute poisoning, frequently terminating fatally. Twelve to fifteen grains of the acetate have been sufficient to kill a dog within an hour. The fatal dose in man is not quite determined; but doses above the usual emetic dose of the sulphate (ten to fifteen grains) have caused serious symptoms, and death has resulted within four hours after swallowing some pieces of the sulphate. Starr (*Med. Record*, May 1882) records a case of death in four days from one ounce of the sulphate. Half an ounce would probably cause a fatal result. The symptoms are essentially those of irritant poisoning, namely, styptic or coppery taste, constriction of the fauces, epigastric pain, violent vomiting and purging, followed by collapse and death, usually with tetanic or convulsive symptoms. That which characterises copper-poisoning more especially, as compared with other irritants, is the frequent occurrence of jaundice. Hæmoglobinuria with albuminuria has also been observed (Starr's case). In dogs copper usually causes death with symptoms of paralysis of the hinder extremities, proceeding like a form of ascending paralysis, in addition to the usual irritant symptoms. It is said also to have a paralysing action on the heart. Such effects have only followed, at least constantly, introduction into the veins; when taken by the stomach most of it seems to be stopped by the liver, or in the case of some of the salts (i.e. acetate) may be reduced by the sugar of the food.

2. Chronic poisoning by copper.—It is generally stated that the long-continued introduction of copper into the system in small doses gives rise to a form of chronic poisoning known under the name of 'copper colic.'

SYMPTOMS.—The symptoms are essentially those of gastro-intestinal irritation, with nausea or sickness and diarrhoea. They have none of the characters of colic in the sense in which the term is usually employed. The hair and the cutaneous secretions of workers in copper and brass are sometimes found of a green colour; and a line is sometimes found at the margin of the gums and teeth, variously described by authors—Corrigan calling it purple, while Clapton calls it green.

Though symptoms of gastro-intestinal irritation, as above described, have been found among workers in copper, the question is whether they are in reality due to the copper, or merely symptoms of a not uncommon affection showing themselves among copper-workers. That copper does gain access into the system, and may be detected in the urine and more especially in the faeces during life, and found in the bones after death, without the individual showing any manifest symptoms during life, seems pretty well established. But though we may regard it as certain that symptoms of copper-poisoning are more rarely found than those of lead-poisoning among those who have to deal with these metals, yet it would be a very unwarrantable conclusion, and contrary to all that we know of the action of poisons, to assert that a substance which is undoubtedly poisonous can be taken freely into

the system with impunity. This is a point of considerable interest in reference to the accidental or wilful adulteration of articles of food with copper salts. Many cases are on record of severe symptoms resulting from the use of copper utensils in cooking, or more frequently from the storage of water or articles of food in copper vessels, especially if the food contains oil—which, on turning rancid, dissolves the copper—or vegetable acids, or even large quantities of ordinary salt.

Copper-salts are also employed intentionally to impart a green fresh colour to pickles and preserved vegetables, such as peas. It is asserted that the quantity of copper necessary to produce this effect is infinitesimal, and that no poisonous effects can be proved to have resulted even from long-continued employment of these vegetables as articles of food. This is strongly maintained by M. Galippe, who has tried them on himself and family. Assuming the impossibility of proving the injurious effects of copper-tinted vegetables, the question comes to be principally a social and economic one, as to the propriety or legality of adulteration of food at all, and especially with a substance undoubtedly poisonous. Copper is said by Odling and Dupré to be a natural constituent of the human body. They have found it in the blood, tissues, viscera, and in many animal products, such as cheese, eggs, and also in many vegetables. It takes the place of iron in the colouring-matter of the blood of some invertebrates (cray-fish), and is found as a constituent of the colouring-matter of the feathers of the turaco (plantain-eater).

DIAGNOSIS.—The greenish or bluish colour of the vomited matters, which turn bright blue on the addition of ammonia, renders the diagnosis comparatively easy.

TREATMENT.—In *acute* cases the stomach should be evacuated by encouraging vomiting, or by the stomach-pump. Albumen in some form, as milk or white-of-egg, should be given, in order to precipitate the copper. Iron filings may be given for a similar purpose.

In *chronic* poisoning the cause should be discovered and removed, or the individual removed from the cause.

D. FERRIER.

J. DIXON MANN.

CORN.—**SYNON.**: *Clavus*; *Fr. Clou*; *Cor*; *Ger. Leichdorn*; *die Hühnerauge*.

DEFINITION.—A corn is a thickening of the epidermis, caused by undue pressure and friction, as by boots, shoes, or implements of occupation. It is usually situated on a prominence, such as that of a joint, where the skin is subjected to double pressure and is therefore unable to yield, or between the toes. Corns are most common on the feet.

DESCRIPTION.—A corn usually begins as a general and uniform thickening of the epidermis, which is termed a *callosity* (*tyloma*, *tylosis*, *Schwiele*). Callosities may occur on any part of the integument. Thus they may occupy the prominence of a joint, or spread over the heel or the metatarsal cushion of the foot from pressure in walking, or occupy the metacarpal prominences of the hand, as in boatmen.

The callosity is composed of laminated epidermis; is thickest in the centre, becoming thin towards the circumference; and is more or less hard and condensed, smooth and hornlike in appearance, and yellowish in colour. When the irritation

which gives rise to a callosity is prolonged, effusion is apt to take place beneath it, and it is raised like a blister, the effused fluid being sometimes serous and sometimes sero-purulent. Whenever this happens, the subsequent separation of the horny layer results in spontaneous cure.

When the pressure giving rise to a callosity, instead of being diffused, is concentrated on a central point, the epidermis corresponding with that point increases in thickness, by its under surface, and forms a conical prominence; further pressure increases the length and breadth of the cone, and in this way a corn is established. Continued irritation enlarges the corn by hyperplasia of epidermic cells, and produces absorption of the derma by pressure, sometimes extending to the bone itself. Not infrequently effusions of serum or blood take place beneath the conical prominence; and, in rare instances, a bursa is found between the corium and the joint.

At a late stage of its growth the corn has the appearance of a central core—technically, *the eye of the corn*—surrounded by a collar of smooth epidermis in the state of callosity. The core is a lamellated ovoid mass, corresponding in external figure with the cup by which it is produced; and consisting in substance of vertical cup-shaped lamellæ closely packed one within the other. In an old corn the shape of the entire core is conical, the point resting on the sensitive skin, and the signification of the term *clou* or nail thus applied by the French is made manifest. The substance of the corn resembles horn both in colour and density, but between the toes, where moisture is generally present, it remains white and soft, like sodden cuticle, and is thence named a *soft corn*. Effusion at its base is more common in the soft than in the hard corn.

TREATMENT.—The treatment of a corn is to remove its causes, namely, pressure and friction; but when this is impracticable, to equalise pressure, by which the corn will revert to the state of callosity. The second indication is best effected by some simple unirritating application, such as the soap- or lead-plaster spread on washleather. As a preliminary to this application, as much of the hard epidermis as possible should be removed by soaking and scraping, and the core turned out with a blunt-pointed instrument. The soft corn may be removed as the hard one, or by snipping with scissors. When there is inflammation about the corn, it should be treated by an antiseptic fomentation. Chronic corns are much benefited by an application composed of Salicylic Acid, 6 parts; Extract of Indian Hemp, 1 part; Flexile Collodion, 48 parts, or by Salicylic acid plaster.

CORNEA, Diseases of.—See EYE, AND ITS APPENDAGES.

CORNUAL (*cornu*, a horn).—Relating to the cornua or horns of grey matter of the spinal cord, e.g. *cornual* myelitis. See SPINAL CORD, Diseases of.

CORONARY ARTERIES, Diseases of.—**SYNON.**: Fr. *Maladies des Artères Coronaires*; Ger. *Krankheiten der Kranzarterien*.—Diseases of the coronary arteries are specially important owing to their effect upon the nutrition of the walls of the heart. See ARTERIES, Diseases of; HEART, Disease of walls of; and ANGINA PECTORIS.

CORPORA AMYLACEA (*corpus*, body; and *ἄμυλον*, starch). See CONCRETIONS.

CORPORA QUADRIGEMINA, Lesions of.

The facts of comparative anatomy and experimental physiology tend to show that the corpora quadrigemina (*corpora bigemina* or *optic lobes* of the lower vertebrates), though related to the optic tracts, do not correspond in their development to the eyes or oculo-motor apparatus, but appear to be largely concerned in those functions, such as equilibration and locomotor co-ordination, which are independent of the cerebral hemispheres. The connections of the two pairs of bodies are entirely different. The anterior stand in relation to the optic tracts on the one hand, and on the other to the occipital lobes. The posterior pair are more especially related to the lateral fillet, the trapezoid body, and the peripheral auditory apparatus.

The facts of human pathology, though not opposed to these data, cannot be made the basis of very precise conclusions as to the diagnostic indications of disease of these ganglia, as it is exceedingly rare to find disease, such as local softening, limited to this region, and hæmorrhage is unknown.

The corpora quadrigemina are, however, not infrequently involved in lesions which invade neighbouring parts, such as meningitis—simple and tubercular, and tumours, especially of the pineal gland.

It is stated by experimenters on the lower animals (pigeons &c.) that when the anterior ‘tubercles,’ which are more especially connected with the optic tracts, are destroyed, vision is abolished, and that if the lesion is unilateral, the blindness occurs on the side opposite the lesion. There is no clear evidence that these results occur after experimental ablation in the higher mammals, or as a result of disease in man apart from indirect implication of the optic nerves and tracts (neuritis).

It has been found experimentally in animals, and also in man, that atrophy of the opposite ‘tubercle’ ensues when the eye has been destroyed. Irido-motor action is also paralysed by destruction of the corpora quadrigemina, a result which, however, is stated not to occur unless the injury is more than superficial and implicates the oculo-motor nuclei.

In the latest experiments performed by the authors upon ablation of the quadrigeminal bodies, no disturbances of equilibration were observed provided the lesion was limited to the ganglionic masses. If, however, the cerebellum or the subadjacent cerebellar peduncles were implicated, symptoms characteristic of their destruction were apparent.

Irritation of the corpora quadrigemina on one side causes dilatation of the pupil and a hemipisthotonos of the opposite side, which becomes general if the irritation is prolonged or bilateral, the head being retracted and the legs extended, trismus also being very marked.

Tumours implicating the corpora quadrigemina are generally associated with a reeling gait, which is an early symptom, and also frequently with symptoms of ophthalmoplegia from implication of the oculo-motor nuclei. The affection of the ocular muscles is unequal, according to the degree in which the respective nuclei may be affected by the lesion, the most common being ptosis on one or both sides and impairment in the upward move-

ment of the globes. The statement has been definitely made that lesion of these bodies in no way impairs vision, except as the result of an associated inflammatory or destructive condition of the optic nerves or tracts, or from internal hydrocephalus. Owing to the mental dulness of patients with quadrigeminal tumour, no clear facts as to the state of hearing are usually obtained, but it has been stated that impairment of hearing is commonly present, and usually upon the side opposite the lesion. Interference with motion and sensation is entirely referable to extension of the growth forwards into the internal capsule, or downwards into the tegmentum cruris and pyramids. Should the superior cerebellar peduncles become involved, tremors of an 'intentional' character are observed, such as are seen in cerebellar disease. There is nothing characteristic about the state of the knee-jerks, these being in some cases absent, in others exaggerated. Their presence or absence probably depends upon the state of general intracranial pressure.

The two chief phenomena which characterise a lesion of the quadrigeminal region are a combination of partial ophthalmoplegia with a reeling and unsteady gait.

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W. A. TURNER.

CORPULENCE (*corpus*, a body).—An undue accumulation of fat in the body. See OBESITY.

CORPUS STRIATUM, Lesions of.—The corpus striatum of English anatomy and pathology comprises various structures which have received special names—namely, the *nucleus caudatus*, or intra-ventricular nucleus, which is exposed to view by laying open the lateral ventricle; the *nucleus lenticularis*, or extra-ventricular nucleus, consisting of three divisions, and subjacent to the convolutions of the island of Reil; together with the anterior division of the *internal capsule*, or peduncular expansion, which connects the cortex and crus cerebri.

This differentiation is necessary, as the effects of lesion of the corpus striatum will differ according to whether the grey matter alone, or the internal capsule, is involved.

Physiological experiment has not succeeded in defining the respective functions of the ganglionic masses of the corpus striatum. The experiments of Aronsohn and Sachs, Ott, Hale White, and others seem to show that lesions of these ganglia in animals cause a rise of several degrees in body temperature from increased heat-production, but both the experimental and the clinical evidence on this point is conflicting.

The symptoms of lesion of the corpus striatum are usually those of the immediately adjacent motor tracts, viz. hemiplegia, and with more extensive lesions, hemianesthesia and hemianopsia. Owing to the artery of this ganglion—the lenticulo-striate—being frequently the seat of miliary aneurysms, its rupture is one of the most common causes of hemiplegia. Should rupture occur upon both sides at different times, a condition of double hemiplegia is seen in which the bulbar symptoms are most pronounced. This forms one of the varieties of the so-called 'pseudobulbar' paralysis, and is regarded as indicating bilateral lesions of the corpus striatum.

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W. A. TURNER.

CORPUSCLE (*corpusculum*, a little body).—In physiology and pathology this word is generally used as synonymous with cell. See CELL.

CORROSIVE SUBLIMATE, Poisoning by.—See MERCURY, Diseases arising from.

CORYZA (*κόρυζα*, a running from the head).—A synonym for nasal catarrh. See NOSE, Diseases of.

COUGH.—SYNON.: *Tussis*; Fr. *Toux*; Ger. *Husten*.

ÆTIOLOGY.—The immediate cause of cough is the presence of an irritant, mechanical or sympathetic, affecting the surface of the air-tubes or the nerves that supply them; and it is the object of the cough to remove this source of irritation.

The sensibility of the respiratory surfaces is greatest at its commencement, the glottis being an ever-watchful janitor. It may be increased by congestion or inflammation, or by the continued act of coughing. Even the mere inhalation of cool or dry air may, in asthma or bronchial congestion, be sufficient to excite cough. The result of the irritation is to increase the natural secretion, and to alter its characters. See SPUTUM.

Cough may be due to numerous reflex causes, such as gastric irritation, ear-disorder, aneurysmal, glandular, or other pressure on the vagus, recurrent, or sympathetic nerves. The act may also be caused by a long uvula or enlarged tonsil; a granular state of the pharyngeal or laryngeal mucous membrane; polypi or other foreign bodies in the larynx, trachea, or even in the external auditory meatus; various affections of the bronchial tubes—for example, undue dryness, hyperæmia, alteration in the quality or quantity of the bronchial secretion, or inflammatory affections; inflammation of the lung or pleura; or tubercular, malignant, or other growths in or near the lung.

DIAGNOSIS.—Cough is not a disease to be treated, but a symptom to be traced to its source. An inspection of the pharynx and larynx, and a physical examination of the chest, will generally suffice to detect the cause.

The character of the cough is often quite pathognomonic—e.g. the 'whoop' of whooping cough; the 'bark' of hysteria; the catching, painful cough of pleurisy; the slight 'hack' of early phthisis, and the equally distinctive cough of advanced phthisis with laryngeal ulceration; the tearing cough of empyema threatening bronchial fistula; the loud clanging cough due to pressure on the trachea or laryngeal nerves; the spasmodic, suffocative cough of asthma.

The 'tightness' or 'looseness' of cough, indicating the absence or presence of secretion, is a valuable guide in diagnosis and treatment.

The absence of cough is no proof of the absence of serious lesion; thus, while the presence of a few tubercles in the lung is often productive of incessant and uncontrollable cough, long-continued destructive disease may exist without it.

TREATMENT.—Before prescribing for a cough it is of course essential to ascertain its cause; and the simplest and most innocuous remedies should be first used. The routine treatment of cough by sedatives is highly injurious. The secretions which ought to be removed are thus locked up, and the irritation, which would have been transient, becomes established.

If the tonsils are found much enlarged, or the uvula pendulous and irritating the epiglottis, caustics or surgical measures will remove the evil. If a granular state of the pharyngeal membrane exists, dependent on torpid or engorged abdominal viscera, gout, or hepatic obstruction, it may be treated by local astringents and general deobstruents.

A lax or congested state of the laryngeal membrane, due to overwork of voice, or the undue direction of attention to the vocal apparatus (clergyman's sore throat), is best treated, according to the writer's experience, by the local application of a solution of zinc sulphate—ten grains to an ounce of water—or of iodine dissolved in spirit and olive oil. Undue dryness, simple hyperemia, or hyperæsthesia of the respiratory mucous tract, may often be relieved by the act of sipping and slowly swallowing cold water, or a decoction of Iceland moss, fruit-lozenges, gum arabic, liquorice, or linseed-tea. Sucking ice or inhaling steam is very often all that is needed. In the early stage of catarrhal sore-throat, chlorate of potassium in crystals, or in the form of lozenges, used in moderation, should not be neglected. The use of glycerin of tannin, or nitrate of silver dissolved in glycerin (half a drachm to one ounce), is of more service in relaxed throat than alum or tannin gargle; indeed, the free use of well-selected lozenges has rendered the employment of gargles well-nigh obsolete. The former can be constantly, the latter but seldom applied.

Medicinal treatment.—If it is desired to increase the fluidity of the secretion, squill or ipecacuanha may be used, or, better still, tartar emetic in small doses, which is best given in effervescence with carbonate of ammonium and citric acid. It must not be forgotten that syrups and nauseating expectorants are apt to do harm by enfeebling or disturbing digestion. Tincture of aconite, in three-drop doses, is often of value in allaying irritable cough, especially when fever is present. Gelsemium is a more recent remedy for the same purpose. Of the direct sedatives, morphine is the most valuable; it proves of service in very small doses, $\frac{1}{12}$ gr. in a lozenge being often adequate. Conium, with or without morphine, suits some persons; hydrocyanic acid still more; and Indian hemp is also of value. The bromides, in combination with chloral hydrate, have gained great repute; the latter should be given with caution. The power of the bromide of ammonium in allaying spasmodic cough is remarkable. An emetic of ipecacuanha, sulphate of zinc, or mustard may be useful in relieving cough, by expelling secretion when this has accumulated in large quantity. If cough causes vomiting, food should be taken in small quantities, fluids should be limited, and a little capsicum or spiced brandy 'stays the stomach.'

External applications.—The use of counter-irritants must not be neglected. In the inflammatory stage of bronchitis, for instance, linseed and mustard poultices, and, in the later stages, iodine, are of great use; and in some cases the application of a small blister or vesicating fluid is a remedy not to be forgotten.

Inhalations.—Infusion of hop as an inhalation is a useful calmate; iodine is indicated in relaxed conditions in strumous subjects. Chloroform (10–15 minims) mixed with eau de Cologne, and inhaled from a handkerchief, is useful in other cases. By means of an atomiser spray-inhaler, many non-volatile preparations may be applied to the respiratory

passages. A solution of carbonate of sodium is very useful in liquefying tenacious secretion. Tannic acid, alum, perchloride of iron, and nitrate of silver, are all valuable. Of sedatives, henbane, conium, menthol, camphor; and of antiseptics, sulphurous and carbolic acids, are serviceable as inhalations. The vapour of tincture of iodine, 15 drops to 2 ounces of water, in the steam-draught inhaler is often useful. In chronic granular disease of the pharyngeal and laryngeal mucous membranes, the sulphurous waters of Aix-la-Chapelle, Aix-les-Bains, and St. Sauveur in the Pyrenees, when inhaled in an atomised state, are of distinct service.

Patients may be taught *how to cough* as follows: Try to suppress the inclination, until the secretion that causes the cough is within reach, then take a deep and deliberate inspiration, and the accumulated phlegm is removed at a single effort. By inhaling steam from a hot sponge or a basin of boiling water on first waking from sleep, the inspissated secretion, which is apt to be difficult to move, may be easily loosened and expelled. An ipecacuanha lozenge may serve a similar purpose.

E. SYMES THOMPSON.

COUNTER-IRRITANTS.—SYNON: Fr. *Contre-stimulants*; Ger. *Gegenreizmittel*.

DEFINITION.—The term 'counter-irritation' implies any irritation artificially established with a view to diminish, counteract, or remove certain morbid processes which may be going on in a more or less remote part of the system. The substances employed in establishing this state are called counter-irritants, and may be classified as follows, according to the degree of their action:—1. *Rubefacients*. 2. *Epispastics*, *vesicants*, or *blistering agents*. 3. *Pustulants*.

That irritation of the surface can influence deeper parts has been demonstrated. Brown-Séquard found that irritation of the skin of the back, over the kidneys, caused a contraction of the arteries supplying those organs. Zulzer found that when cantharides-collodion was painted over the back of a rabbit for fourteen days the vessels underneath the skin were congested, while the deeper parts, including the lungs, were pale and anæmic. From these experiments we can understand how a blister may affect the circulation in an internal viscus. See PAIN IN VISCERAL DISEASE.

1. **Rubefacients.**—**ACTION.**—These remedies, applied to the skin, produce local warmth and redness from increased flow of blood in the cutaneous vessels. The local hyperæmia thus induced subsides gradually on ceasing to employ the rubefacient; but sometimes, when the action of this has been prolonged, the epidermis may peel off, and more or less local soreness remain. Rubefacients are usually quick in action; their local after-effects are trifling; and they may, therefore, be applied without injury over a large extent of surface.

ENUMERATION AND APPLICATION.—Examples of rubefacients are found in Ammoniacal liniments or embrocations; Mustard plasters and the official Liniment; Volatile Oil of Mustard; Oils of Turpentine and Cajuput; Capsicum, in the form of plasters or the official ointment; and Iodine. Hot water is at times applied on a sponge or flannel to produce a speedy counter-irritant and derivative effect in relieving sudden internal congestion and muscular spasm, as in the early stage of croup, laryngitis, and laryngismus stridulus. The mustard poultice

is a useful and rapidly acting rubefacient in inflammation, spasm, and neuralgic pain. Sir Alfred Garrod recommends a very useful sinapism, made by mixing 10 minims of volatile oil of mustard with 1 oz. of spirit of camphor, and sprinkling this on impermeable piline. Mustard leaves or the Charta Sinapis (B.P.), applied to the skin, produce a speedy rubefaction of the surface. Linimentum Sinapis is also a very active rubefacient. Vinegar should not be added to mustard poultices; but if some oil of turpentine or a little powdered capsicum be mixed with the mustard, the poultice will act with much greater rapidity. Where, on the other hand, a gentle stimulation with warmth and moisture on the surface is desired, as in some cases of pneumonia, a linseed-meal poultice may be used with its surface sprinkled lightly over with mustard meal. Generally twenty minutes is as long as an ordinary mustard poultice can be safely borne on the skin. In persons who have a very delicate skin, a layer or two of muslin should be placed between the mustard application and the surface of the body. In applying mustard poultices to those who are unconscious of pain, caution is necessary, for it has happened that the poultice being left on for a long time has produced dangerous ulceration and sloughing of the surface. A mustard foot-bath is at times employed with a view to a revulsive and counter-irritant effect. To prepare a mustard-bath, two tablespoonfuls or more of mustard should be tied in a cloth and agitated well with cold water; then hot water may be added to make the bath.

USES.—Rubefacients are used in chronic inflammation and irritation of the mucous surfaces, as in bronchitis, and irritation about the air-passages. Troublesome cough, in cases of phthisis, is often relieved by applying tincture of iodine, or acetic acid and turpentine liniment, to the chest. Rubefacients are of service in removing lingering irritation about a joint, their use also tending to promote the absorption of chronic thickening or effusion in the joint; but friction with a rubefacient liniment over a joint must not be employed till all active inflammatory action has entirely ceased. Various degrees of persistent counter-irritation may be maintained by applying, after the skin has been well cleansed with soap and water, the official Emplastrum Picis or Emplastrum Calefaciens. A mustard plaster applied to the nape of the neck has proved useful in cases of irritable brain with sleeplessness. The same application made to the foot or great toe is a valuable revulsive where gout attacks more important organs. A mustard plaster has the advantage over a blister in rapidity of rubefacient action; and, from the sharp pain caused, the mustard plaster is preferable when it is a matter of moment to rouse one who is in a state of lethargy or torpor from narcotic poisoning by opium, or alcohol, or from coma in the course of a fever. Where we wish to exercise a prolonged action over chronic inflammation in an organ, we should use a blister rather than a sinapism.

2. Vesicants, Epispastics, or Blistering Agents.—**ACTION.**—A blister acts primarily as a rubefacient and powerful stimulant to the cutaneous vessels. The papillæ of the skin become reddened and raised; minute vesicles soon appear on the elevations; and these, gradually coalescing, form a bleb, or large vesicle, between the true skin and epidermis, containing an albumino-fibrinous fluid.

ENUMERATION AND APPLICATION.—The agent

most commonly employed for blistering purposes is the Cantharis or Spanish beetle; but there are others that have been used for a similar object. Glacial Acetic Acid applied to the skin produces intense redness and pain, with rapid vesication, but its action may extend too deeply as a caustic, and cause a troublesome sore. Liquor Ammoniac dropped on a piece of lint, applied to the skin, and covered with a watch-glass, very soon causes redness and rapid vesication in most persons. This is a good way of raising a blister when it is desired to apply powdered morphine endermically to relieve severe pain. The official Liquor Epispasticus applied with a brush soon raises a blister on the surface. Blistering Collodion is used for a similar purpose, but is slower in action.

The application of blisters should not be made directly over an inflamed part, lest the injured area be increased. Blisters should not be applied where the skin is loose, nor over any prominence of bone, nor to the breast during pregnancy. It should moreover be borne in mind that the cantharidine of a blister may be absorbed by the skin, and act on the kidneys, producing strangury and bloody urine. This accident may be obviated by sprinkling powdered camphor over the blister before placing it on the skin, or a thin piece of silver paper may be interposed. In persons of feeble vitality, a blister left on for too long a time has been known to induce dangerous sloughing.

When vesication is specially desired, there is no need to leave the blister on for twelve hours or more, for it may be removed at the end of six or eight hours, and a warm linseed poultice applied. If the blister be opened, which is best done by pricking the most dependent part with a disinfected needle, olive oil and cotton-wool is the best dressing. The practice of maintaining a blister as a running sore or *exutoire*, by applying irritating ointment, is not often resorted to now. The process causes great pain and exhaustion of the system, and is one rather of depletion than of counter-irritation. In the case of children, blisters should be used with caution, being kept on for about one hour or till the skin is well reddened, when they should be replaced by a poultice. It is well not to open the blister, as the effused serum forms the best dressing covered with cotton-wool. By following this plan the child is saved much worry and pain.

USES.—In its primary effect a blister acts as a local stimulant, but when it remains on long enough to produce extensive vesication and discharge of serum, it acts as a depletive and depressing agent. This primary and secondary action of blisters was much insisted on by the late Dr. Graves of Dublin, who found great benefit in cases of fever with apathy and prostration from the application of *flying blisters* to various parts of the surface. Thus a blister over the precordial region, kept on for about one hour, and then removed, was observed to rouse and stimulate a flagging heart. In other cases the flying blister might be placed at the chest or back, or else behind the head on the neck. Care should be taken not to leave the blister on long enough to cause actual vesication, and the size of the blister should be fairly large.

In hysterical paralysis narrow blisters completely encircling the affected limb have proved curative. A similar blister round the throat may

cure nervous aphonia. A blister one inch wide may possibly sometimes stay the spread of erysipelas along a surface. In cases of pleuritic or pericarditic effusion the repeated application of blisters to the chest-wall is of manifest advantage. In effusions into joints (hyarthrosis) blisters aid absorption; and it has seemed to the writer that absorbent remedies, such as iodide of potassium, often begin to do good as soon as a blister appears to have once set the absorptive process in action.

In the obstinate acid vomiting of gouty patients, a blister over the epigastrium often gives relief. Some forms of neuralgia, as for example pleurodynia, may yield to a blister over the seat of the pain. At times obstinate pleurodynia, or mastodynia, can be relieved by flying blisters applied in the vertebral groove on the affected side, where a tender spot can often be detected on pressure. Blisters should be avoided in cases of renal and vesical inflammation, as the absorption of the cantharidine may increase the mischief.

3. Pustulants.—ACTION.—The agents belonging to this class of counter-irritants produce a pustular eruption on the part to which they are applied, and their use is not recommended.

ENUMERATION AND APPLICATION.—Among pustulants may be placed Croton Oil, Tartarated Antimony, and strong solution of Nitrate of Silver. When croton oil is applied to the skin, it acts as an intense irritant, producing an eruption which is at first papular, but very soon becomes pustular. The oil is best employed in the form of the official Linimentum Crotonis. Tartarated Antimony is a powerful counter-irritant, producing pustules which resemble those of variola. It should not be applied to parts usually uncovered, as the pustules leave marks behind them; and under all circumstances the remedy, being a painful one, must be used with caution.

JOHN C. THOROWGOOD.

COURT-SAINT-ETIENNE in Belgium.—See MINERAL WATERS.

COW-POX.—See VACCINIA.

COXALGIA (*coxa*, the hip; and *αλγος*, pain).—Pain in the hip-joint. See JOINTS, Diseases of.

CRACKED-METAL or **CRACKED-POT SOUND.**—SYNON.: Fr. *Bruit de pot fêlé*.—A peculiar sound elicited by percussion, resembling that emitted on striking a broken jar or a metallic vessel. See PHYSICAL EXAMINATION.

CRAFT-PALSIES.—See OCCUPATION-DISEASES.

CRAMP.—SYNON.: Fr. *Crampe*; Ger. *Krampf*. This name is applied to certain painful varieties of tonic spasm. In its most familiar form it affects the calves of the legs, coming on principally at night, on the occasion of some slight movement of these parts. The affected muscles, mostly on one side, contract with such energy as to give rise to a board-like rigidity, together with sensations of an agonising character. The attack rarely lasts more than a minute or two, though it may more or less speedily recur. It is perhaps best cut short by a vigorous but steady voluntary contraction of the opposing extensor muscles of the foot. Where it is more obstinate than usual, firm pressure around the thigh or upon the great sciatic nerve sometimes

gives relief. Cramp is often associated with some irritation of the stomach or of the intestines, especially in children or delicate nervous persons. In this way it is produced not infrequently when arsenic in medicinal doses has been continued for some time, and is beginning to exert some slightly poisonous effect upon the system. In a more general form it often occurs, to a marked extent, in cholera. Localised, painful, cramp-like contractions of muscles may also be due to irritation of anterior root-fibres, either in their intra-medullary or extra-medullary course. Other forms of painful spasm are by no means common, if we except colic. See COLIC; SPASM; and THOMSEN'S DISEASE.

H. CHARLTON BASTIAN.

CRANIOTABES (*cranium*, the skull; and *tabes*, thinning).—A morbid condition of the cranium in children, consisting in spots of local thinning of the occipital, parietal, and (rarely) the frontal bones, as well as in general abnormal flexibility of the osseous tissue. See SKULL, Diseases of.

CRAW-CRAW.—A native term adopted by Europeans on the West Coast of Africa and used vaguely to include various vesicular and pustular skin-eruptions—most of them of parasitic origin.

CREPITANT (*crepito*, I make a noise).—When applied to a body, this word signifies that it is capable of yielding the sensation or sound of crepitation. It is also used to indicate a peculiar character which râles sometimes possess. See PHYSICAL EXAMINATION.

CREPITATION (*crepito*, I make a noise).—A sensation or sound of crackling. It may be observed in morbid states of the bones, joints, or subcutaneous tissue; but the term is more frequently applied to a physical sign connected with the lungs. See PHYSICAL EXAMINATION.

CRETINISM (*crétin*, Swiss *patois* for *chrétien*, a Christian).—SYNON.: Fr. *Crétinisme*; Ger. *Cretinismus*.

DEFINITION.—A condition of idiocy arising from endemic causes, associated with imperfect development and deformity of the whole body, varying however in degree.

This condition of physical and mental degeneracy is not limited to any nationality. It obtains in the great mountain-chains of Europe, Asia, and America. In Europe it is met with in the valleys of Switzerland, Savoy, and Piedmont; and it abounds in the neighbourhood of Salzburg, Styria, and the Tyrol. It is less frequently met with in the Pyrenees and in the valleys of the Auvergne in France. Even in England it has been met with in various parts, among others in the dales between Lancashire and Yorkshire. Although more frequently met with in valleys, it is not unknown on plains which are subject to inundations.

ÆTIOLOGY.—The conditions for the development of cretinism are hereditary predisposition; the action of deteriorating influences on the parents, such as unwholesome dwellings and non-nutritious diet; and accidental causes operating on the infant during the period when its physical and intellectual life is developing. The last-named causes are atmospheric and possibly geological conditions, peculiar to special localities. Humidity of the soil

and air in valleys where there is little interchange of the atmosphere, and the existence of magnesian limestone in the soil, are probably the most potent factors. Cretinism is not met with as an endemic disease on elevated plateaux, nor in cold countries where sudden changes of temperature are uncommon. Goitre is a frequent accompaniment of cretinism, and would appear to be developed under the same conditions. The cases of cretinism met with in England present features which are indicative of a scrofulous origin. There is a condition of idiocy associated with arrest of growth and development at the period of first dentition or about the fifth year, though congenital cases are not unknown, frequently met with in England, which has been termed *sporadic cretinism*.

In an analysis made by the writer some years ago of 116 cases of this affection, of whom 16 had been under his care, it was found that several predisposing causes were generally present, and that the chief of these were intemperance and insanity in the parents. These were each present in fourteen cases, or 12·07 per cent. Inherited mental disease did not play such an important part, only nine being attributed to it, or 7·75 per cent. Fright of the mother during pregnancy was traceable in no less than thirteen cases, or 11·20 per cent., while protracted labour only accounted for seven, or 6·03 per cent. Consanguinity was assigned as a cause of the disease in six cases, but in each of these other important factors were present. Two children were born in a valley which was foggy and marshy. These cases are associated with absence, atrophy, or hypertrophy of the thyroid gland, and with the development of fatty swellings in the supra-clavicular spaces, and occasionally in the armpits. They resemble children permanently, and have a remarkable resemblance one to another as regards their bodily conformation, facial physiognomy, mental characteristics, and the symptoms generally. See MYXŒDEMA.

Cretinism has been thought by some authors to be due to premature ossification of the cranial sutures, especially of the speno-basilar suture; and this change, in its turn, has been attributed to drinking water largely charged with lime. It is impossible, however, to regard this premature ossification, when it does occur, as other than one of the results of the malady, not its cause. Moreover there are numerous examples where the synostosis is deferred instead of being premature. The real *proximate cause* of the affection is interference with the function of the thyroid gland.

ANATOMICAL CHARACTERS.—Pathological anatomy shows that the bones of the cranium are thickened and without diploe, and the sutures well united. In some cases the foramen magnum is smaller than normal, the basilar process horizontal and narrowed laterally, and the clivus, or inclined plane formed by the union of the basilar process of the occipital with the sphenoid bone, is very steep. The cerebellar fossæ are flattened. The skull in endemic cretinism is brachycephalic, but in sporadic cretinism it approaches more the dolichocephalic in shape. The brain in the latter affection is of normal size, but the arrangement of the convolutions is simple; there is none of the complexity which is seen in the brains of ordinary children. On microscopical examination the cells are seen to be pear-shaped or rounded, and are deficient in processes. There is excess of mucin in the skin, and excess of

fibrous tissue in the lungs, liver, spleen, and kidneys. The bones of the extremities are short and curved, in accordance with the statement of Dr. Bury, that there is arrest of bones that develop in cartilage, while bones formed in membrane, as are those of the skull, are normal or show excessive growth.

DESCRIPTION.—The degrees of cretinism are numerous. In any of the valleys where this affection exists the various steps of degeneracy can be traced, commencing with those who are taking part in the industrial life of the valley, down to the helpless individuals who are leading only a vegetative existence.

The typical cretin presents a very marked physical conformation. He is stunted in growth, rarely reaching five feet in height. His skin is of a tawny yellowish hue, thickened and wrinkled, and looks as if too large for the body. There is also a great increase of subcutaneous areolar tissue. His tongue, large and thick, with hypertrophied papillæ, always displays lessened power of co-ordination; and often hangs from the mouth. The mouth is partly open, margined by thick fissured lips, and with the saliva running over the chin. The face is large; the lower jaw is drooping, and its angle obtuse. The eyes are often affected by strabismus, obliquely placed, and small; and the lids are commonly puffy. The belly is pendulous from the laxness of the skin. The lower limbs are generally short and deformed, and the gait is waddling. The head is deformed, the forehead retreating, the top flat, and the occipital region ill-developed. The cranium is brachycephalic. The nose is broad and flattened. Puberty is often delayed to the twentieth year. The mammae in the female are large and pendulous; the same remark applies to the genitals in the male. The intellectual faculties are imperfectly developed. The cretin is often unable to speak, and his hearing is frequently defective. The affection is usually associated with more or less enlargement of the thyroid gland. His viability is low, few living beyond thirty years of age. The sexual functions are abnormal; masturbation is frequent; and the subjects of cretinism are often impotent.

The description of the sporadic cretin is somewhat different. He is much diminished in stature, and his head, which is large in proportion to the size of the body, is flat at the top, spread out at the sides, often narrow in front and broad behind. The hair is sparse, coarse, and dry, like horsehair. The eyelids, of a pale, bluish hue, are swollen and infiltrated with a solid œdema which does not pit on pressure; and the subcutaneous tissue of the hands and feet is similarly affected. The nose is broad, the cheeks full and flabby, the mouth large, the lips thick and often slightly apart; through these an enlarged tongue slightly protrudes. The teeth are frequently carious and irregularly implanted; the second dentition is either deficient or completed at an advanced age. The chin is small, but there is often a layer of fat beneath it, forming a double chin. The ears are large, but not badly formed; and the neck is short and thick, and usually no thyroid gland can be felt. Well-developed fatty swellings, as before mentioned, are seen in most cases on each side of the neck, and sometimes in the armpits or other regions. The abdomen is large, distended, and contains a quantity of subcutaneous fat; frequently there are umbilical and occasionally inguinal herniæ. There is arrested development of the genital organs; the testicles are small and

descend late, and the uterus and ovaries are often badly developed. The menstrual function is either not established or is irregular: there is no sexual appetite. The arms and legs are short and curved, and the hands and feet, though often small, are broad, thick, blue, and frequently dry and scaly. The skin of the body, as well as of the upper and lower limbs, is thick, easily separable from the subjacent muscles, whitish in colour, dry and rough, owing to the almost entire absence of perspiration; on the face it is yellow or waxy; and gives rise to a sallow colour. The pulse is small and feeble, the temperature subnormal, and hence there is great susceptibility to cold. The voice is hoarse, harsh, or squeaky, the gait clumsy and waddling, and there is great repugnance to making any movement. General sensation is below normal, but the special senses are well developed, with the exception of smell and taste. The knee-jerks are usually present, and the plantar reflexes normal. Speech is generally limited to a few words, and is sometimes only monosyllabic, but the patient can usually give his name. He is good-natured, placid in temperament, and fond of those who have to attend upon him.

In the cretin who has undergone treatment, certain differences will be noted. The fatty swellings in the supra-clavicular spaces disappear, the prominent abdomen subsides, and the umbilical hernia becomes lessened or disappears. The skin of the body desquamates, and is no longer dry or scaly, and the temperature rises to normal. In young children the hair falls out, but is replaced by a new crop, which is softer in character. The development of the teeth is accelerated. The patient begins to grow, the weight is lessened, and cutaneous sensibility becomes normal. The voice is less guttural, the expression more intelligent, and there is more independence and inquisitiveness.

DIAGNOSIS.—The diagnosis of cretinism may be made in childhood, from the slowness of the development of the body, the stupid expression, the postponement in the evolution of the teeth, and of the ossification of the fontanelles and sutures, the tawny yellow colour of the skin, the thick and goitrous neck, the slaving, and the delay of speech and of walking.

Sporadic cretinism is sometimes mistaken for Mongolian imbecility, but in the latter condition the obliquely placed eyes, flattened bridge of the nose, rounded pinna of the ears, and the hypertrophied papillæ of the tongue, which presents transverse fissures, sufficiently distinguish it from sporadic cretinism.

TREATMENT.—This consists in removing the child as early as possible from the circumstances which have produced the disease. He should be taken to a locality where the soil is dry and porous, and should have frequent baths with friction to the surface of the body. The diet should be of the most nutritious kind—a diet into which animal food largely enters. The administration of cod-liver oil and of the lacto-phosphates of lime and iron is indicated. Early education should be commenced as to habits of cleanliness, followed by systematic physical exercise of the various muscles. All intellectual advancement must be sought for through the improvement in every way of his physical condition. The lower animal life may thus be supplemented, if earnest efforts are used, by increased capacity for rational enjoyment, and a more or less useful existence.

As regards sporadic cretinism very young children should be given one grain of the dried thyroid gland, two minims of the official *Liquor Thyroidei*, or one-fifth of a grain of the dried colloid matter daily. For other children, tabloids containing five grains of thyroid extract will be found useful, and should be administered once a day. The patient must be carefully watched, and if he is found to lose weight too rapidly, and the pulse and temperature rise much above normal, the treatment must be stopped for a time or the dose be reduced. The writer has noticed that the younger the child the greater is the improvement; adolescents react much more slowly to treatment and never improve so much. A complication which is sometimes noticed is bending of the legs; this must be stopped as much as possible by keeping the patient from walking too much, and by giving full diet and tonics.

The treatment must be continued during the whole of the patient's life, or he will relapse into his former condition. FLETCHER BEACH.

CRETINISM, *Fœtal*.—See ACHONDROPLASIA.

CRIME, IRRESPONSIBILITY FOR.—**HISTORICAL SUMMARY.**—A medical opinion as to the condition of an accused person is often necessary in order to determine whether he or she can be held accountable for criminal acts. Such an opinion generally depends on the presence or absence of insanity, or on the connection which may be traced between this mental condition and the act in question. In the article on the Lunacy Laws, it is explained that it is only within a comparatively recent period that insanity has been admitted as an excuse for crime, except in those comparatively rare cases in which, as Justice Tracey expressed it in 1723, a person does not know what he is doing, 'no more than an infant, a brute, or a wild beast' (see LUNACY LAWS). This view fairly represents the state of public and of legal opinion until the later years of the eighteenth century. The subsequent enlightenment of the public mind did not receive juristic expression until the trial of Hadfield in 1800, when Erskine first enunciated the doctrine, that 'delusion where there is no frenzy or raving madness is the true character' of such insanity as implies irresponsibility. The most important case in the history of this question was that of Bellingham, who was executed in 1812 for shooting Mr. Spencer Perceval. In this case Sir James Mansfield, C.J., said, that if a person labouring under mental derangement were capable in other respects of distinguishing right from wrong, 'he could not be excused for any act of atrocity which he might commit.' 'It must be proved beyond all doubt,' he added, 'that at the time he committed the atrocious act he did not consider that murder was a crime against the laws of God and nature.' The trial of MacNaughton in 1843 for the murder of Mr. Drummond led to the most authoritative statement of the law which has ever been obtained in this country. MacNaughton was acquitted on Chief Justice Tindal's direction that the point for the jury to consider was whether 'at the time the act was committed' the accused 'had that competent use of his understanding as that he knew that he was doing by the very act itself a wicked and wrong thing.' The general application of this doctrine would have greatly enlarged the area of irresponsibility, and its enunciation at that time produced

considerable surprise and even consternation. The matter was indeed regarded as so urgent that the House of Lords immediately ordered a series of questions to be laid before the fifteen judges with the view of settling the state of the law. In the answers to these questions it was in substance laid down that, to entitle an accused party to acquittal on the ground of insanity, it is necessary either that he be prevented by unsoundness of mind from discriminating between right and wrong in respect to the act with which he is charged; or, that he be under some delusion which made him regard the act as right. But this statement has been far from effecting a final settlement of the question.

Most writers on medical jurisprudence have insisted that the real criterion of responsibility is the freedom of the will, or the power of the individual to control his actions. This has been more or less advocated by Esquirol, Marc, Ray, Pagan, Jamieson, Mittermaier, and von Krafft-Ebing. Esquirol dwells strongly on the importance of the freedom of the will. Ray includes it in the comprehensive statement which has received the approval of so many medical jurists. 'Liberty of will and action,' he says, 'is absolutely essential to criminal responsibility, unless the constraint upon either is the natural and well-known result of immoral or illegal conduct. Culpability supposes not only a clear perception of the consequences of criminal acts, but the liberty, unembarrassed by disease of the active powers which nature has given us, of pursuing that course which is the result of the free choice of the intellectual faculties.' Pagan observes that the 'loss of control over our actions, which insanity implies, is that which renders the acts which are committed during its continuance undeserving of punishment.' Jamieson puts the question: 'Had the lunatic at the time of committing the deed a knowledge that it was criminal, and such a control over his actions as ought, if it existed, to have hindered him from committing it?' Dr. Taylor says ('Taylor and Stevenson's Medical Jurisprudence,' 1894): 'The true test for irresponsibility in all doubtful cases appears to be, whether the person at the time of the commission of the crime had or had not a *sufficient power of control to govern his actions*.' Sir John Bucknill's view is substantially the same. 'Responsibility,' he says, 'depends upon power, not upon knowledge, still less upon feeling. A man is responsible to do that which he can do, not that which he feels or knows it right to do. If a man is reduced under thralldom to passion by disease of the brain, he loses moral freedom and responsibility, although his knowledge of right and wrong may remain intact.' The latest German code puts responsibility upon the same basis. 'An act is not punishable,' according to it, 'when the person at the time of doing it was in a state of unconsciousness; or of disease of the mind, whereby free volition was prevented.' Mittermaier and von Krafft-Ebing sanction the attempt to render the meaning of 'free volition' more definite by describing it as made up of *libertas judicii* and *libertas consilii*—freedom of judgment and freedom of choice. Casper somewhat obscurely defines criminal responsibility as 'the psychological possibility of the efficacy of the penal code.' Mr. Balfour Browne gives as the best definition 'a knowledge that certain acts are permitted by law, and that certain acts are contrary to law, and, combined with this knowledge, the power to appreciate and be moved

by the ordinary motives which influence the actions of mankind.' Dr. Guy held that every person who is insane must be regarded as wholly irresponsible, and that the law of England ought to be assimilated to that of France in the declaration that 'il n'y a ni crime ni délit lorsque le prévenu était en état de démente au temps de l'action.' Dr. Maudsley and others hold that the determination of responsibility in cases where insanity is alleged depends on whether a connection can or cannot be traced between existing disease and the act.

Insanity has been pleaded as an excuse for acts of theft; but such cases are rare, and never occur except where the social position of the accused adds importance to the decision. Indeed it may almost be said that the plea is never raised except in order to avoid capital punishment. Hence it is, that in the discussions which have arisen the question has been intimately associated with the law of murder and homicide. A special inquiry into the state of this law by a Committee of the House of Commons (1874) consequently gave occasion to the enunciation of important views as to the legal relations of insanity and responsibility. Evidence was furnished to the Committee by Lord Chief Justice Cockburn, Baron Bramwell, Mr. Justice Blackburn, and Sir James Fitzjames Stephen. The immediate object of the Committee was to examine a bill drawn by Sir James Stephen for the codification of the law of homicide. In the clause of the bill which deals with the relations of disease and responsibility, homicide is stated to be 'not criminal if the person by whom it is committed is at the time when he commits it prevented by any disease affecting his mind—(a) from knowing the nature of the act done by him, (b) from knowing that it is forbidden by law, (c) from knowing that it is morally wrong, or (d) from controlling his own conduct.' But it is stated to be 'criminal, although the mind of the person committing it is affected by disease, if such disease does not in fact produce one of the effects aforesaid in reference to the act by which death is caused, or if the inability to control his conduct is not produced exclusively by such disease.' It was, however, proposed in the bill that, 'if a person is proved to have been labouring under any insane delusion at the time when he committed the homicide, it shall be presumed, unless the contrary appears or is proved, that he did not possess the degree of knowledge or self-control hereinbefore specified.' That is to say, where delusion exists, the burden of proving moral capacity would be shifted, the prosecutor having to prove its existence, instead of the accused having to prove its absence. The opinions elicited during the inquiry showed that the law is regarded by legal authorities as being at present too uncertain in its operation, and as failing to recognise some of the most important elements in the question. The divergent character of the recommendations which were made showed, however, that legal opinion is much divided, not only as to the proper relations of insanity and crime, but also as to the essential elements of responsibility. In the meantime, therefore, the statements of the fifteen judges after the MacNaughton case remain the chief exposition of the English law where insanity is pleaded in excuse for crime.

PRESENT STATE OF THE QUESTION.—It is necessary, in order to justly appreciate the present aspect of the subject, thus to trace its more recent history, and it may be useful to present an estimate

of the comparative value of the several tests or criteria which have been proposed for the determination of cases in which insanity has been alleged. These criteria may be broadly summarised in the following six propositions. According to one view a person should be held irresponsible for an act if at the time of committing it (1) he laboured under insanity of any kind or degree; according to another if (2) he laboured under delusion; or (3) if he was ignorant of right and wrong; or (4) had not power to appreciate and be moved by ordinary motives; or (5) had lost the controlling power of the will; or (6) if the act is traceable to, or its nature has been determined by, mental disease affecting the agent. The last of these views is the only one to which fatal objection may not be raised, both on the theoretical and practical sides. The others are all too vague to be of much advantage; and they rather tend to introduce new difficulties than to remove those already existing. The proposition therefore which seems to approach nearest to a solution of the difficulty is that irresponsibility must be admitted whenever the act is traceable to, or its nature is determined by, mental disease affecting the agent. It will of course be understood that under such a rule the term 'mental disease' must be held to include both congenital and acquired disorders; arrest of development being as much a morbid condition as functional or structural change. This view of the subject may not be ultimately accepted in the precise terms of the proposition here given; but the principle on which it rests seems to afford the only safe basis upon which we can go. As has already been shown, it has not hitherto been regarded in this light by the majority of the judges; but there have been indications of late years that judicial views are tending in that direction. The late Lord Wensleydale and others have given sanction to the principle in their judicial statements. And a late Lord Justice-General of Scotland (Ingis) gave definite expression to it in one case (*Brown*, Sept. 1866). He told the jury that the main question was 'whether the prisoner was in such a state of insanity at the time as not to be responsible for the act which he had committed:' and in order to constitute such insanity he said that 'it must be clearly made out that at the time of committing the act the prisoner was labouring under mental disease in the proper sense of the term, and that that mental disease was the cause of the act.' In America the doctrine has been frequently acknowledged, but never more fully and tersely than by Judge Doe, of New Hampshire (*State v. Pike*), who comprehensively defined the medical relations of both criminal irresponsibility and civil incapacity, when he stated that a 'product of mental disease is neither a contract, a will, nor a crime.' One important point is to prevent persons from being punished for actions which are the direct outcome of pathological processes. But it is of equal importance to avoid the adoption of a principle which would make the existence of slight mental irregularities incompatible with responsibility. There does not appear to be any danger of this in acting on the principle which is here enunciated. For it lies in the very nature of the cases in which the doctrine could be applied, that the condition to which it is proposed that irresponsibility should be attached must be one which is known to exhibit itself in acts of serious and even criminal character. If the trained observer of disease is able to recognise in an act—which is ordinarily followed by severe

punishment—a direct result or a characteristic feature of a morbid process, of the existence of which there may otherwise be sufficient proof, the question of responsibility cannot present any serious difficulty. It may admit of doubt whether a person is responsible for not controlling his actions, or for not knowing right from wrong, or even in some cases for the harbouring of a delusion. But once let it be proved that an act is the natural result of a disease under which a person is known to labour, and there can be little doubt that he will be held irresponsible. Before, however, the principle can be accepted as fully satisfactory, it is necessary to inquire whether its application would permit insane persons to be held responsible who ought not to be so considered. In other words, are there states of insanity in which a person is irresponsible for acts to which he has not been predisposed or impelled by the insanity? It is perhaps impossible to give such an answer to this question as would be both definite and complete; but for practical purposes we think it may be answered in the negative. Where the insanity is of such a nature that it does not modify the whole conduct, we believe it will be found in practice necessary to admit the existence of responsibility for acts where there is no demonstrable connection between them and the mental disease. The insane persons who on this principle might be held responsible would be found solely among those whose irresponsibility could only be admitted after very searching inquiry, and whose insanity was of that kind and degree which has often been declared by medical writers to be consistent with responsibility. It is not to be supposed that under this rule difficulties would cease. It will always be possible that an act may have been really the result of mental disease, and that we may fail to obtain satisfactory evidence of the existence of such disease. This, however, is due not to any defect in the principle, but to that imperfection of our knowledge which renders the perfect application of any principle impossible. Let the task of the medical witness be limited to the demonstration of facts indicative of disease and its consequences, and he will at least be acting quite within his special province, and may expect that reasonable weight will be attached to his opinion. And if juries were instructed that the law does not hold a person responsible for acts committed under the influence of disease, it is scarcely conceivable that any one would be found guilty where good cause had been shown even for the reasonable supposition of such an influence. Whatever may be the view ultimately adopted, it would seem to be in every way desirable that the attention of the medical expert should be confined to the elucidation of the medical facts, and that he should not be asked to deal with questions which are legal and abstract, and in no way specially medical. It is not the business of a medical witness to say whether a person is or is not responsible. That is a purely legal question. He has only to deal with the existence of disease—using this word in its widest sense—and its relation to the conduct of the person, and in giving evidence as to this, a witness cannot be too careful in making certain that his opinion is based on facts which can be proved. The condition known as *diminished responsibility* has not been alluded to in this article. It is only indirectly recognised by British law, and therefore, though much is to be said in favour of its recognition, it is unnecessary to deal with the subject here.

The mental disturbance produced by drink is not held to confer irresponsibility unless the mind has been so substantially or permanently impaired as to amount to insanity (see *Reg. v. Burns*, Liverpool Assizes 1865, Bramwell, B.; *Reg. v. McGowan*, Manchester Assizes 1878, Manisty, J.; and *Reg. v. Baines*, Lancaster Assizes 1886, Day, J.).

JOHN SIBBALD.

CRISIS (*κρίσις*, a decision, a turn).—SYNON.: Fr. *Crise*; Ger. *Krise*.—Crisis is a term used to denote two entirely different phenomena.

1. The term 'crisis' as applied to fevers signifies the rapid deferescence of an acute febrile disease. It has wandered somewhat from its original meaning, which was 'judgment'—primarily an operation in the mind of the observer, but reflected upon the phenomena observed. The converse term, employed to designate a gradual subsidence of fever, is *lysis*.

In order to constitute a true crisis the deferescence should occupy less than forty-eight hours, and it often takes place in a much shorter time. The fall of temperature should be accompanied by a corresponding reduction in the frequency of the pulse, and should coincide with a feeling of relief and a return of strength; the skin will be warm and soft, the tongue moist, and there will be indications of reviving appetite; there may or may not be a critical evacuation, but the secretions will become more natural in amount and character.

This favourable mode of termination of an acute febrile disease is more common than is usually supposed. The circumstances under which it is most likely to occur are when the attack begins abruptly and the temperature rises rapidly, the natural course of the disease being short and not attended with organic lesions, such as will of themselves keep up fever. After twenty-one days, termination by crisis is not to be expected. The more marked the onset—as, for example, by a definite rigor—the more rapid the rise of temperature, and the greater the height to which it reaches, the greater the probability of an early critical termination.

OCCURRENCE.—The diseases in which the conditions favouring a crisis are realised, and in which this mode of termination is observed, belong to various classes.

Among the *specific fevers*, eruptive and continued, it occurs frequently in variola, but in severe cases it is interfered with by the febrile disturbance excited by the eruption. In measles it is very common. In scarlet-fever a true crisis is seen only in mild cases, though the onset of this disease is peculiarly abrupt. The mode of termination of typhus is essentially critical, but as a rule the crisis is not sharp. Relapsing fever affords the best examples of crisis, which is moreover attended by a critical evacuation in the form of profuse perspiration, the temperature sometimes falling 10° F. in as many hours, and the patient passing from a state of extreme suffering and oppression to almost perfect ease and comfort. In enteric fever *lysis* is the mode of termination.

Remittent fevers often present crises, which may be true and curative, or false and illusive; and the sun-fever and common continued fever of hot climates, and tropical diseases generally, have a tendency to fever running high very early and breaking abruptly at a critical period.

In this country *feverish colds*, attacking the throat or taking the form of influenza or catarrh, often terminate critically in three or four days. Erysipelas may so end, but at a later and less definite period. The sharp *febrile attacks* which sometimes occur *after childbirth* often exhibit a very decided crisis. In *pneumonia* the natural termination is by a well-marked crisis, which may take place as early as the fifth day, or be deferred to the ninth, after which a critical termination is not to be expected, and the suspicion may be entertained that the case is not one of frank pneumonia, the prognosis becoming grave. As has been already stated, the general improvement precedes the indication by physical signs of resolution in the inflamed lung. Pleurisy is said also to terminate critically, but it is not in the same definite way as pneumonia.

The *critical evacuations* which entered into the original notion of a crisis are really a common attendant. The most common is a profuse warm perspiration, which may occur whatever the disease may be. Occasionally the evacuation is a copious flow of urine, or it may take the form of diarrhoea. Epistaxis or hemorrhoidal flux is a more rare and doubtful critical evacuation. A common critical phenomenon is a prolonged, sound, and refreshing sleep.

Indications of an approaching crisis are often given two or three days beforehand in slight remissions of fever. By the presence or absence of such remissions at a certain period of the attack, or by a continuous rise of temperature where a remission might be expected, important prognostic information may be afforded and indications for treatment obtained.

THERAPEUTIC INDICATIONS.—The main therapeutic deduction from a study of crisis as a termination of acute disease is, that we should not hastily interfere with the reactions by which the system adjusts itself to altered conditions or meets the incidence of the causes of such disease, but contribute to their completion. We do not assume the existence of a *vis medicatrix* tending invariably to the restoration of health; but we must recognise the power inherent in a living organism to respond by internal changes to external influences, and to regain the balance when this has been disturbed. In this process a certain cycle of changes must be gone through, and the great opportunity for treatment of an active kind, should any be required, arises when the course, direction, and probable duration of these changes are known, and when agencies can be brought to bear at a given moment which will contribute to bring about the appropriate critical evacuation or a critical sleep, through which a return to a normal condition would naturally be effected.

2. The term 'crisis' is also applied to painful paroxysmal attacks occurring in cases of tabes dorsalis. Various hollow viscera (especially the stomach) and other parts may be involved, and the function of the affected organ is for the time seriously impaired. See *TABES DORSALIS*.

Recently the term has been employed to designate similar attacks occurring in cases of movable kidney and accompanied by nausea (*Dietl's Crises*).

WILLIAM H. BROADBENT.

CRITICAL.—Having relation to a crisis. See *CRISIS*.

CROUP (Scotch, *croup* or *roup*, hoarseness).—
 SYNON.: *Cynanche trachealis*; Fr. *Croup*; Ger. *Croup*.

DEFINITION.—An acute febrile affection, occurring in childhood, accompanied by inflammatory swelling of the mucous membrane of the larynx, and in some rare cases by the formation of a fibrinous exudation on the surface.

ÆTIOLOGY AND PATHOLOGY.—The symptoms of the most usual form of croup are due to an acute catarrh of the laryngeal mucous membrane, without any formation of false membrane. The mucous membrane swells by reason of the congestion of its blood-vessels, and the pouring out of inflammatory exudation into its tissue. Mucus, too, is prone to accumulate on its surface. These conditions are precisely the same as occur in the ordinary catarrhal laryngitis of adults. But in the child the glottis is much narrower than in adults, while the swelling of the mucous membrane from inflammation is greater, and reaches its height more rapidly. Moreover, the larynx is in children more irritable than in adults, and therefore its muscles are more easily excited to spasm. Thus when a simple catarrhal laryngitis makes its appearance in a child, it is very soon followed by laryngeal stenosis, partly from the swelling of the mucous membrane, partly from accumulation of mucus, and partly too from spasm of the laryngeal intrinsic muscles. The condition is generally attributed to exposure to cold and damp, especially at a time when the body is heated. It is caused also by scalds of the larynx, when unduly hot fluids are drunk by accident, by wounds, by the irritation of noxious gases, and by the presence of foreign bodies in the larynx. It is more common in male than in female children, and is usually met with between the ages of two and nine years.

There also exists, however, an inflammation of the larynx which is accompanied by the formation of a false membrane, and it is in respect to this condition that so much discussion has arisen. It is, of course, well known that in diphtheria the characteristic membrane may spread from the pharynx, its usual site, into the larynx, and in doing so may produce all the symptoms of so-called croup, in addition to those due to the diphtheria proper. But much of the confusion which existed as to the pathological entity of croup, resulted from the non-recognition of those cases of diphtheria in which the local lesion had its primary seat in the larynx. Pathological observations, however, have now placed it beyond doubt, that such cases really exist and form the majority of what were formerly diagnosed as 'membranous croup.' Death may occur from the laryngeal stenosis so produced, as in all other obstructions of the larynx. The actual diagnosis can now be rendered certain, if any portions of the present membrane be examined bacteriologically (see DIPHTHERIA). The diphtheritic nature of the affection may, however, be obvious, from the supervention of other symptoms, such as great asthenia, heart-failure, hemorrhages from various parts, albuminuria, and the formation of diphtheritic membrane on sores far removed from the larynx. Moreover, when, in fortunate cases, the acute symptoms have passed away, the characteristic paralyzes of diphtheria may show themselves. The facts of contagion are also of importance, for from supposed cases of croup other cases may arise, in which the diphtheritic membrane appears first in the pharynx,

and the disease then runs its ordinary and generally unmistakable course.

There remains still a certain proportion of cases of membranous laryngitis, which are not due to the action of the diphtheritic poison. Some of these are produced as secondary complications by the poisons of other infective fevers, and among these are to be mentioned scarlet-fever, small-pox, measles, enteric fever, and whooping-cough.

It is known that the tendency of an inflammatory exudation to coagulate on a mucous surface depends on the power of the causative injury to produce extensive coagulation-necrosis of the epithelium. The large majority of such inflammations are due to the action of bacteria. There are, however, many cases recorded in which a membranous laryngitis has been caused by the contact of boiling water with the back of the throat, by burns, and even by foreign bodies.

Further, membranous inflammation has been produced experimentally by the injection of acrid irritants into the larynx of animals. Practically, however, it must be concluded that such cases are exceedingly rare, and that false membranes do not form in the laryngitis, which forms the basis of what is met with as the simple non-diphtheritic croup of children. No reliance can be placed on structural differences in the membrane itself. This varies according to its position, more than according to its cause. The existence of any specific organisms in any marked quantity is the only point needing investigation.

SYMPTOMS.—In accordance with the view expressed above, little need be said about cases other than those of non-membranous croup. The child affected by this disorder may suffer from general catarrhal symptoms for a time, varying from a few hours to two or three days. More commonly he goes to bed apparently well, though probably after some exposure to cold or damp. In a few hours, however, he wakes suddenly, as if frightened, breathing hurriedly, with loud, hoarse, inspiratory, laryngeal stertor. He coughs frequently, making a harsh metallic noise, but at first removing no mucus from the larynx. He cries, and perhaps speaks, in a shrill cracked tone, which soon gives place to a mere whistling sound. The character of the breathing, voice, and cough gave rise to the Scotch vernacular name for the disease, 'croup,' now generally employed. The child is very restless, tosses his body and limbs about incessantly, clutches at his throat as if to remove the obstruction there, and seizes any surrounding objects with his hands. The face is at first flushed, the eyes bright, and the expression exceedingly alarmed and anxious. Soon, however, the flush becomes dusky, gradually pallor supervenes, and the whole body may be covered with a cold sweat. The feet, and sometimes the hands, not infrequently assume the position found in tetany, and in extreme cases general convulsions may occur. The pulse is frequent and feeble, and the temperature is slightly raised. The difficulty with which air enters the chest is obvious from an observation of the patient's position and respiratory movements. The head is thrown backwards and the chin held out prominently, this position being accentuated at each inspiration by a contraction of the sterno-mastoid and other muscles of inspiration. All the extraordinary muscles concerned in chest-expansion are brought into play, and the nostrils dilate at each breath. Yet, in spite

of the extra force employed, the thorax is not completely filled with air. During inspiration the supraclavicular fosse, the lower ribs, the epigastrium, and even the lower portion of the sternum move inwards instead of outwards. The yielding of the chest-wall is, as might be expected, more pronounced in rickety children, and in such subjects the whole of the ribs may be seen to give way at the sides as each breath is drawn. If the chest and larynx be auscultated, at first nothing is heard but laryngeal sibilus; afterwards rattling noises, due to the presence of mucus, are perceived in the larynx, while in the chest rhonchi may be found. The cough, which was at first dry, as the attack continues, displaces small pellets of thick tough mucus; sometimes threads of this substance are expectorated, and have been mistaken for false membrane.

Usually, after a time varying from half an hour to two or three hours, the difficulty in respiration subsides, and the child falls asleep again, but not quite free from symptoms indicative of his ailment. His face is somewhat flushed, and there is some degree of pyrexia. The restlessness does not quite disappear even in sleep. The hands and feet frequently retain their tetanoid position. The breathing is still husky and somewhat hurried, and the occasional slight cough has the clanging character noticed during the paroxysm; during the next day, or for the next two or three days, the breathing is hurried and somewhat stertorous, and the attack may return during the next and even succeeding nights. For some time after recovery is apparently complete, any slight cough is prone to assume a 'croupy' character, just as when, after an attack of whooping-cough, the characteristic whoop may at times recur. After one attack, too, or series of attacks, the child may suffer from repetitions of the disorder, even for several years.

Other signs of laryngeal stenosis will be described in the article on DIPHTHERIA.

PROGNOSIS.—The prognosis of non-membranous croup is almost invariably favourable in so far as the actual attack is concerned. In spite of the apparent severity of the symptoms, they nearly always subside naturally. The friends of the patient should, however, be warned of the probability that the attack will be repeated at times, and even for some years, after its first appearance. Membranous croup, on the other hand, is a much more serious matter. At any moment the false membranes, whatever may be their cause, may accumulate or curl up in the larynx, and entirely close the glottis. Nor is the danger merely that of the laryngeal affection. The laryngitis occurs, usually, as we have said, only in severe cases of the specific fevers, and the blood-poisoning of the original disorder, just as in diphtheria, is usually a most fatal condition.

TREATMENT.—As soon as the attack of croup has commenced, the child should be placed in a warm bath, to which preferably a little mustard has been added. Sponges, wrung out of water as hot as can be borne, should be applied over the larynx, and renewed as often as they become cold; or a mustard leaf may be applied there. An emetic, too, is useful. This may be administered in the form of ipecacuanha wine, in doses of one teaspoonful every ten minutes until vomiting comes on, and may be followed by a dose of calomel and jalap. It is well to place the child in a tent-bed,

under the cover of which the nozzle of a steam-kettle is introduced.

Prophylactic treatment is of importance, when the disease has once shown itself. In cold weather the child should be clothed in woollen under-garments, from neck to foot, and protected from chills and draughts. At the same time, the larynx may be accustomed to cold, by being sponged once or twice a day with cold water. General tonics, too, are of use in warding off future attacks. When obstruction of the larynx has been produced by false membrane, its treatment must be that recommended for the same condition occurring in diphtheria. See DIPHTHERIA. ROBERT MAGUIRE.

CROUP, FALSE.—A term commonly applied to laryngismus stridulus. See LARYNX, Diseases of.

CROUPOUS, CROUPY (Scotch *croup*, hoarseness).—These terms were originally employed with reference to the peculiar crowing or stridulous character of the respiration, cough, and voice in certain affections of the larynx, and signified 'belonging to croup' in its clinical relations; for example, 'croupy cough,' 'croupy symptoms.' When morbid anatomy demonstrated the occurrence of a fibrinous exudation or false membrane upon the affected surface in a special form of croup, the word 'croupous' was used also to designate this false membrane; thus 'croupous exudation' and 'croupous membranes.' The application of the term was afterwards further extended; and though now falling into disuse it is still employed to indicate the process that leads to a fibrinous exudation in any situation whatever; such as 'croupous inflammation' and 'croupous pneumonia.' See CROUP; DIPHTHERIA; and INFLAMMATION.

CROWING CONVULSION.—A popular synonym for laryngismus stridulus. See LARYNX, Diseases of.

CRURA CEREBRI, Lesions of.—In the crura cerebri lie the paths of communication, both afferent and efferent, between the cerebral hemispheres and the bulbo-spinal centres. The motor or pyramidal tracts lie in the *foot* or ventral portion of the crus, which is separated from the dorsal portion, or *tegmentum*, by the *locus niger*. The pyramidal tracts of the internal capsule constitute only the middle three fifths of the foot of the crus; the mesial fifth connects the frontal lobe with the grey matter of the pons, and, indirectly perhaps, with the opposite cerebellar hemisphere (Flechsig); the outer fifth connects the temporal region of the hemisphere with the grey matter of the pons, and probably indirectly with the cerebellum. The sensory tracts run in the tegmentum or dorsal division of the crus cerebri, from which they pass into the optic thalamus. The fibres of the third nerve pass through the internal, or mesial, aspect of the crus on their way to the oculo-motor nuclei in the floor of the aqueduct of Sylvius.

From the anatomical and functional relations of the crus cerebri, lesions of this region, such as may arise from hæmorrhage, tumour, &c., are apt to cause paralysis of motion and sensation on the opposite side of the body, as well as oculo-motor paralysis on the same side as the lesion. The degree of implication of the sensory and motor tracts and fibres of the third nerve varies in individual instances; but an *alternate paralysis*, in which

there is some degree of oculo-motor paralysis on the one side, and paralysis of the limbs and of the lower part of the face on the other, is diagnostic of lesion of the crus. Owing to this association having been first pointed out by Weber, it has received the name of 'Weber's symptom.' Of the oculo-motor phenomena, ptosis is the most frequent and usually the earliest, the whole of the third nerve becoming eventually involved. Should the lesion be within the crus, more or less affection of sensation will co-exist with the motor weakness, and by this means an intracranial lesion may be diagnosed from one of meningeal nature. There have also been described tremors of an 'intentional' nature, probably from implication of the superior cerebellar peduncle and red nucleus. In paralysis from lesion of the crus cerebri, there has been described also vaso-motor paralysis, and the temperature of the paralysed side may be two or three degrees above that of the other.

D. FERRIER.

W. A. TURNER.

CRUSTA LACTEA (*crusta*, a crust; and *lactea*, milk-like).—Milk-crust: a synonym for *eczema pustulosum* of the face and head, met with in infants at the breast. See ECZEMA.

CRUVEILHIER'S PARALYSIS.—A synonym for progressive muscular atrophy. See PROGRESSIVE MUSCULAR ATROPHY.

CUPPING.—SYNON. : Fr. *Action de ventouser*; Ger. *Schröpfen*.

Cupping is a mode of treatment formerly employed to relieve congestion or inflammation of internal parts by drawing blood to the surface of the body. When the blood thus attracted to the superficial parts is actually abstracted from the body by means of incisions, the operation is called *wet-cupping*; and this has been described in the article BLOOD, Abstraction of. In *dry-cupping* no scarifications are made, the blood being simply drawn towards the surface by atmospheric exhaustion.

Dry-cupping was formerly employed as a counter-irritant. See COUNTER-IRRITATION.

MODES OF APPLICATION.—Dry-cupping is performed as follows: The flame of a spirit-lamp, being allowed to burn for an instant in the dome of a cupping-glass, is quickly withdrawn, and the cup (the rim of which has been slightly moistened) is then rapidly and evenly applied to the skin over the affected part previously sponged with hot water to render it more supple and vascular. The heat expands the air contained in the glass cupola, and, owing to the contraction which ensues on cooling, the skin is forcibly sucked up into the cup.

PRECAUTIONS.—Cupping-glasses should be applied where the skin is thick and cushiony, as over the loins, nape of the neck, pectoral region of the chest, and not where bony or other irregularities are likely to interfere with the accurate apposition of the cupping-glass. The edge of the glass must not be so hot as to burn the skin.

USES.—Cupping was often employed in cases of supposed cerebral congestion, the cup being applied to the nape of the neck; and in inflammation or congestion of the lungs, kidneys, or other viscera.

JOHN HAROLD.

CUTIS ANSERINA (*cutis*, the skin; and *anser*, a goose).—A state of roughness and pallor of the

skin, resembling that of a goose when plucked, produced by prominence of the hair-follicles and compression of the vessels. It is due to contraction of the arrectores pilorum, and is commonly occasioned by cold or by fright.

CYANIDES, Poisoning by.—See ANTIDOTE; and PRUSSIC ACID, Poisoning by.

CYANOSIS (*κυανός*, blue).—Cyanosis is the peculiar blue or livid colour of the surface of the body, especially in certain parts, which is observed in several affections that interfere with the circulation and oxygenation of the blood. The condition is most commonly associated with certain forms of congenital malformation of the heart, and reaches its highest development in them. It thus happens that cyanosis is not uncommonly used as a synonym for congenital heart-disease. Lesser degrees of similar discolouration are, however, not infrequently noticed in cases of ordinary cardiac failure due to disease developed after birth, and they may also accompany pulmonary affections which materially obstruct the circulation. A cyanotic appearance is also one of the obvious effects resulting from all modes of suffocation, and it is observed in the collapse-stage of cholera. The upper half of the body may become extremely cyanotic as the result of obstruction of the superior vena cava. For the pathology of cyanosis, see HEART, Malformations of.

CYCLIC ALBUMINURIA.—See ALBUMINURIA.

CYCLOPLEGIA (*κύκλος*, a circle; and *πληγή*, a stroke).—Paralysis of the ciliary muscle, causing loss of accommodation.

CYNANCHE (*κύνων*, a dog; and *ἄγχω*, I strangle).—SYNON. : Fr. *Angine*; Ger. *Bräune*.

This word is used to express an inflammatory condition of the throat, or contiguous parts, in which difficulty of breathing or of swallowing exists, accompanied by a sense or feeling of choking. The term is often used as synonymous with *angina*; an affix, indicative of the seat or nature of the affection, being employed as a designation for each of the several forms or varieties of disease affecting the throat or adjacent parts. Such, for example, are the terms *cynanche laryngea*, or croup; *cynanche maligna*, or malignant sore-throat; *cynanche parotidea*, or mumps; *cynanche pharyngea*, or inflammation of the pharynx; and *cynanche tonsillar*, or quinsy. See these several diseases.

CYRTOMETER (*κυρτός*, curved; and *μέτρον*, a measure).—An instrument for measuring the absolute and relative dimensions and movements of the chest-wall. See PHYSICAL EXAMINATION.

CYSTIC LYMPHANGIOMA.—Cystic Lymphangioma, Cystic Hygroma, and Congenital Multilocular Cystic Tumour, are the names given to a fluid tumour of the neck, which is of congenital origin and consists of a number of cystic spaces each lined with a single layer of flattened epithelial cells and filled with a clear or yellowish-coloured serous fluid.

SEAT.—These tumours are met with at birth and then appear as irregular or bosselated swellings of variable size seated in the deeper tissues of the

neck. In some cases, however, they do not become evident until some time after birth.

The tumour in every instance lies underneath the deep fascia and is commonly located in one or other antero-lateral region of the neck, although in some cases both sides may be simultaneously involved. It occasionally happens that the swelling is situated in the middle line of the front of the neck. In rarer instances, the tumours are found posteriorly in the region of the nape of the neck, a short distance below the external occipital protuberance. The tissues of the anterior triangle and underneath the antero-lateral portion of the lower two thirds of the sterno-mastoid muscle are the most frequent seats of these tumours, but they may be limited to the submaxillary regions, at least in the earlier stages of their development. When the tumours have existed for a long time they may extend downwards into the superior mediastinum or along the cords of the brachial plexus into the corresponding axilla.

A 'cystic lymphangioma,' on palpation, presents the physical signs of a soft lobulated swelling, not attached to the overlying skin, with a fairly definite margin superficially, but with indistinct or indefinite deep connections. This latter characteristic is especially marked when the tumour extends in a backward direction underneath the anterior margin of the sterno-mastoid muscle, and has close connections with the sheath of the carotid vessels. The bossed outline is due to the presence of a number of separate cysts.

HISTOLOGY.—Each tumour consists of a number of separate cysts of very variable size, some as small as a grain of wheat, and others one inch or more in diameter, united together by a quantity of fibrous and connective tissue. Each cyst has a wall of fibrous tissue lined internally with a layer of thin flattened endothelial cells. These cells in many respects resemble those which line lymphatic spaces, but differ from these latter in the fact that they have not dentate outlines. The stroma or connective tissue may be of the nature either of fully developed fibrous tissue or of embryonic tissue which has many points of resemblance to sarcoma. Small polypoid intracystic growths may often be observed on the internal aspect of some of the larger cysts. The fluid contents of the cyst are usually of a clear light yellow or greenish colour, but occasionally of a darker hue, owing to the presence of blood-corpuscles or pigments. It contains a quantity of albumen (4 to 10 per cent.) and a variable amount of sodium-salts, chiefly chloride, carbonate, and phosphate. Crystals of cholesterol and derivatives of blood-pigments are not infrequently met with.

PATHOLOGY.—In all probability these tumours arise in connection with the lymphatic tissues in the affected portions of the neck. An error in development has been assumed to be the cause, but the exact nature of this has never been demonstrated. Some investigators have thought, without much evidence, that they arise in connection with the submaxillary salivary glands, or as a result of changes in the inter-carotid body.

DIAGNOSIS.—The congenital cystic lymphangioma must be distinguished from dermoids and from congenital lipomata. Dermoid tumours have a more rounded outline, owing to the fact that they consist of a single cyst; their walls are thicker, and they usually appear at a later period of life than the

cystic lymphangiomata. Congenital lipomata are often with difficulty distinguished from cysts; in the majority of instances they are, however, more localised, do not give a definite sense of fluctuation on palpation, and are of moderately slow growth.

PROGNOSIS.—These tumours are benign in nature. They may remain stationary for a long time and then undergo a spontaneous cure as the result either of an inflammatory process, or of an accidental opening through the skin. In other cases they rapidly increase in size, when the prognosis becomes much worse on account either of pressure by the tumour on important adjacent structures, or of the state of debility and of marasmus which is induced.

TREATMENT.—The treatment depends upon the size and connections of the tumour, its rate of growth, the symptoms to which it gives rise, and the condition of the general health and the age of the patient. When the tumour is small and localised, a condition which is rarely met with, removal of the tumour by dissection ought to be practised at once. When the tumour is of moderate or large size and has important or deep connections, a surgical operation for its removal ought not to be undertaken unless the patient is in a good state of general health and the surgeon is prepared to carry out an elaborate and complicated dissection of the neck, and to run the risk of being unable to get away the entire tumour. Puncture and evacuation of the contents of the tumour, with or without the subsequent injection of iodine and carbolic acid, incision and drainage, and the introduction of setons have all been practised, but with only a small amount of success. Extirpation is the operation to be recommended, the entire tumour being taken away; or if this is impossible, removal of as much of the tumour as is feasible. It sometimes happens, after the latter proceeding has been carried out, that the portions of tumour left behind shrivel up and finally disappear.

H. J. WARING.

CYSTICERCUS.—See ENTOMOA.

CYSTINURIA.—Cystinuria is a very rare condition which is characterised by the presence in the urine of a crystalline sulphur-compound known

as cystin $\left(\begin{array}{c} \text{CH}_3 \\ | \\ -\text{S}-\text{C}-\text{NH}_2 \\ | \\ \text{CO}\cdot\text{OH} \end{array} \right)_2$ —which is apt to be

deposited therefrom as a greenish-white sediment, consisting of colourless hexagonal plates. Cystitis is a frequent complication, but cystinuria derives its chief clinical importance from the great liability which exists to the formation of calculi composed of cystin, which may attain to a considerable size. See URINARY DEPOSITS, *Plat.*

The condition has been met with both in children and in adults, in males more often than in females, and in not a few instances in several members of a family. It may be that, like Alkaptonuria, it is a congenital and lifelong abnormality (see ALKAPTONURIA). It has been shown that in these cases cadaverin or putrescin, or both these diamines together, are apt to be present in the urine and faeces. They have sometimes been present continuously for years, sometimes only intermittently, and in some instances they have been altogether absent on the few occasions on which the excreta were examined.

The association of such rare urinary constituents as cystin and the diamines can hardly be accidental, and indeed the latter have been very seldom met with in any other morbid states. Cadaverin and putrescin are recognised products of bacterial activity, and it has been suggested that cystinuria itself may be indirectly due to bacterial processes in the intestinal tract. There is some evidence that cystin, which is not found in the fæces, is an intermediate product of normal metabolism, and it may be that it owes its preservation from further destructive changes to the presence of the diamines or to the process which leads to their production. However the excretion of cystin continues unchecked even when the diamines disappear from the urine and fæces, and it remains to be shown whether the diamines here met with may not themselves be products of a perverted tissue-metabolism. See CONCRETIONS. A. E. GARROD.

CYSTITIS (κύστις, the bladder).—Inflammation of the bladder. See BLADDER, Diseases of.

CYSTOSCOPE (κύστις, the bladder; σκοπέω, I inspect).—An instrument designed for inspecting the interior surface of the urinary bladder. See BLADDER, Examination of.

CYSTS.—DEFINITION.—The word 'cyst' (κύστις, the urinary bladder) is used in pathology without strict scientific accuracy for a closed cavity containing fluid or soft matter; which cavity will have, from the nature of its contents, a spherical or spheroidal shape. The objects thus defined differ much among themselves, and are associated together rather from convenience than on account of any real pathological similarity.

CLASSIFICATION.—Cysts may be classified according to their structure, as simple or compound; according to their contents, as serous, mucous, fatty, &c.; or according to their mode of origin. The latter, though not free from objection, is the basis of description which will be here adopted. Cysts may originate: (1) from *dilatation* of previously existing closed cavities; (2) from *retention* of the products of secretion of a secreting gland; (3) as a part of *new-growth*; (4) by a vice of *development*. These kinds are distinct structures with a wall lined by epithelium. But the name is often given, less accurately, to spaces containing fluid which result from softening of solid tissue, whether in a new-growth or in an inflammatory product. Cystic parasites have also been confounded with cysts properly so called. See ABDOMINAL CYSTS; and ENTOZOA.

1. Cysts from Dilatation.—Spaces, normal or newly formed, in connective tissue may, by irritation and consequent excessive exudation, be converted into cysts; or the same result may happen from the confluence of several such spaces. In proportion as their walls become smooth, and their shape uniform, they may be called cysts. Bursæ, whether normal or pathological, are cysts. Ganglion in the sheath of a tendon is clearly a pathological cyst. To these and like structures the name *hygroma cysticum* has been given. They all contain clear serous fluid, and are lined by an endothelium. Encysted hydrocele may also be called a cyst. One class of ovarian cysts comes under this head, those, namely, which are due to simple dropsy of the Graafian vesicle. Tubo-ovarian dropsy has the same

explanation; and cysts of the broad ligament are enlargements of normal structures which are left as relics of the development of the ovary. The thyroid gland seems, from its structure, containing as it does so many closed follicles, particularly disposed to this kind of cyst-formation, and this is doubtless the explanation of bronchocele. The cause of such an enlargement of closed follicles is very obscure, but appears to be immediately due to increased production of the material normally filling the space.

2. Cysts from Retention.—Cystic formations may result from the obstruction of the natural outlet of a secreting organ, and the consequent retention of secretion. It is necessary that the walls of the secreting cavity should admit of enlargement, and that the tension should not become so great as to check secretion.

All secreting glands present instances of such cysts. The sebaceous glands of the skin are particularly liable to obstruction of their ducts, and in this way are formed sebaceous cysts, the contents of which are sometimes epithelium and the products of normal secretion, sometimes abnormal products. The same term has, with less propriety, been applied to the sebaceous productions called milium and comedo.

The glands or mucous surfaces are liable to similar obstructions, and *mucous cysts* result, such as are sometimes seen in the mouth. Larger cysts in the mouth (*ranula*) result from the obstruction of the ducts of the salivary glands, or are perhaps connected with an abnormal production of gland-substance. The stomach very frequently, other parts of the intestinal canal more rarely, show similar cysts, which, when they project and become complicated in structure are called polypi. They are occasionally seen in the larynx and trachea. In no part are mucous cysts more frequent than in the uterus, where, indeed, similar formations, the *ovula Nabothi*, must be regarded as normal. The varieties here met with have, as Virchow has pointed out, a close analogy with the various forms of retention-cysts in the skin.

In the mamma, cysts may result from the cutting off of portions of the gland-follicles, but the cysts contained in mammary tumours are not always formed in this way, some being part of new-growths. In the testicle obstruction and cutting off of seminiferous tubes may lead to small cysts, but these are more often connected with new-growths. The curious cysts known as *spermatoceles*, containing spermatozoa, appear to arise from a similar distension of detached portions of testicle-substance, which, by an error of development, have failed to become connected with the excretory ducts. The testicle is also liable to a general cystic degeneration, usually called cysto-sarcoma. Cysts of the kidney are of various kinds, but many, no doubt, both large and small, result from the dilatation of uriniferous tubules and capsules of glomeruli when their outlet is obstructed, as occurs in the cirrhotic form of Bright's disease. The origin of the very numerous microscopic cysts has been much disputed. The writer inclines to the belief that they arise from moniliform contraction of the uriniferous tubes, especially such as contain the hyaline cylinders, known as fibrinous casts. Another form of cystic disease of the kidney is developmental. In this the whole of the organ is converted into a mass of cysts, and is usually much enlarged. This condition may be congenital, and the organ may be so large as to obstruct parturition.

It is attributed by Virchow to inflammation of the calyces during intra-uterine life.

To guard against a common error of language, it should be pointed out that the condition of the kidney which results from the obstruction of the ureter, or of the urinary passages lower down, though sometimes called cystic dilatation of the kidney, is not properly a case of cyst-formation, and is better called *hydronephrosis*.

3. Cysts from New-Growth.—In many forms of new-growth cysts are produced, but not always in the same way. Occasionally, as in myxoma and enchondroma, softening of portions of new-growth already formed produces spaces containing fluid, which are sometimes, though hardly correctly, called cysts. In many sarcomata, the production of new tissue goes hand in hand with that of cysts, and is sometimes effected, as in glandular organs, by the formation of new follicular structures without an outlet, sometimes by new-growth into the dilated cavities. Polypoid or pedunculated growths on a free surface may sometimes, by the fusion of their extremities, enclose spaces which become converted into cysts. We do not often find cysts forming by themselves a new-growth of so definite a character as to deserve a separate name. When they do so, they may be called *cytoma*.

4. Cystoma or Cystic Tumour.—Cysts are met with in the ovary which come under the definition just given, namely, so-called *compound multilocular cysts*, which constitute the well-known formidable cystic disease of the ovary, and sometimes produce tumours of immense size. In these the originally simple primary cyst appears to become complicated by the formation in its walls of secondary cysts, which may encroach upon or project into the primary. Again there may be papillary growths starting from the inner wall of the primary cyst, which either fill it up, or by fusion enclose spaces, which become secondary cysts. Very complicated structures thus result. The contents may vary in consistence and colour, from clear, pale, albuminous liquid to gelatinous matter, and may be stained through hemorrhage, or purulent through inflammation. The origin of these structures, which have no precise parallel in other parts of the body, is extremely obscure. It is not even certain whether the primary cysts commence, as might seem *prima facie* highly probable, in the Graafian follicles; but they are plainly due to an error of development, possibly beginning in early intra-uterine life, and are not set up by any external causes. The presence of a tubular gland-tissue, such as is found in the rudimentary but not in the perfect ovary, confirms this view, by throwing cyst-formation back into an early stage in the development of the organ.

In another, but rarer, form of cystic disease of the ovary, equally due to an error of development, and sometimes congenital, the whole organ is found converted into a mass of small cysts, with no striking inequality of size. This variety resembles one form of cystic disease in the testicle and kidney.

Dermoid cysts are those of which the wall is composed of skin or mucous membrane and generally lined by a layer of flat cells resembling epidermis. The wall may be complicated with connective tissue, forming papillæ resembling those of true skin, and may contain hairs, sebaceous glands—either in connection with them or un-

attached—and sudoriparous glands. The accumulation of fatty matter within the cysts is doubtless the result of the continuous activity of the sebaceous glands, the products of which cannot escape. Large masses of hair may also be found, from continuous growth, and there are often numerous detached epidermic scales. Distinctly formed mammary glands, with nipples, are sometimes found. Such a cyst has the characters of a portion of skin, which might be imagined inverted and included by the growth of the surrounding parts in an early stage of development—an explanation which applies only to those situated in certain parts of the integument where a junction has normally taken place—e.g. along the lines of the branchial fissures in the neck, in the anterior middle line of the body, &c.

These simple dermoid cysts are sometimes complicated by containing teeth, it may be in very large numbers (*dentigerous cysts*); but since teeth may also be regarded as cutaneous products, the cyst may still have originated in the skin. This explanation no longer holds, however, when masses of bone are found, sometimes serving for the attachment of teeth, sometimes separate; as well as other tissues, e.g. nervous tissue and striated muscle. Cysts with this variety of contents have been called *proliferative*. Dermoid, dentigerous, and proliferative cysts appear to be always congenital structures, but may show further growth and development in after-life. At least two-thirds of the known cases have occurred in the ovaries. Next to these organs, the testicles are the most frequent seat, but these cysts have been also found in other parts of the body-cavity, in the mediastinum, lung, and even within the skull. The origin of these growths is extremely obscure; but it is desirable to reject entirely the hypothesis that a mixed tumour of this kind can be the remains of an undeveloped foetus included in the perfect individual: an hypothesis rendered improbable by the extreme irregularity of the tissues produced, the teeth, for instance, sometimes numbering one hundred or more. It would rather appear as if, in some instances, a portion of embryonic epithelial tissue had become misplaced at an early period of development. In other cases, these cysts originate in obsolete canals, representing rudimentary structures: such as the central canal of the nervous system, the post-anal gut, the branchial clefts, the vitello-intestinal duct, and others.

Several parasitic animals infesting the human body may appear in an encysted form, and may resemble in appearance true pathological cysts. The commonest—the larval form of *Tenia echinococcus*, or *hydatid cyst*—is known by its laminated wall, and by containing a fluid which is not albuminous, but holds in solution sodium chloride. *Cysticercus cellulosa* has a transparent wall and clear contents. The other encysted parasites are either very small, as *Trichina spiralis*, or unimportant.

CONTENTS OF CYSTS.—The serous cysts and hygromata contain an albuminous fluid like that of serous cavities, which may hold enough fibrinogenous material to coagulate spontaneously. Leucocytes may also be present. If inflammation be set up, the proportion of albumen and of leucocytes becomes greatly increased. In the fluid of mucous cysts mucin is contained; in that of colloid cysts, little-known substances which are allied to gelatine.

Sebaceous cysts contain neutral fats—sometimes hard, sometimes fluid, and cholesterin. Both mucous and sebaceous products may harden into concretions, and even become calcareous. In renal cysts urea has been found; in biliary cysts, bile-pigment; and in general the products of special secretion may be found in cases of retention, at least in early stages; but if retention last too long,

special secretion may cease. Various exceptional contents have been already enumerated.

TREATMENT.—The treatment of cysts is referred to under the head of TUMOURS.

J. F. PAYNE.

CYSTS, Abdominal. — See ABDOMINAL CYSTS.

D

DACTYLITIS (δάκτυλος, a finger).—A term meaning inflammation of the finger. It is especially applied to syphilitic and tubercular inflammations of the part.

DANCER'S CRAMP.—A spasmodic contraction of the muscles of the calves of the leg, liable to occur in those who dance on the tips of their toes.

DANDRIF, or Dandruff (from Celtic *ton*, skin; and *drwg*, foul).—Scurf of the head (see HAIR, Diseases of). Dandruff is the result of one form of Seborrhoea Sicca, and is met with in Pityriasis, Seborrhoeic Eczema, and Psoriasis.

DANDY FEVER.—A synonym for Dengue. See DENGUE.

DARIER'S DISEASE.—This name is applied to a chronic disease of the skin of unexplained etiology. The disease is named after Darier of Paris, who published important observations on its pathology.

DESCRIPTION.—The disease may make its appearance on any part of the skin, but is most common in the regions where the sweat and sebaceous glands are well developed, as on the axillæ, the inguinal regions, the central parts of the thorax, and the neck. The early lesions of the disease appear to be reddish-brown, flattened or conical, papules, capping the orifices of the sweat and sebaceous follicles. Some inflammatory redness is apparent round these papules, and the coalescence of neighbouring lesions causes larger areas of the skin to become affected. These areas become covered with greyish-brown or black crusts, composed of epithelial scales, sebaceous material, and foreign matter. On removing the crusts, the underlying follicles are seen to be dilated, and the glands themselves full of a greyish cheesy material composed of degenerated epithelial cells and retained secretions. The areas of disease are readily infected by pus-forming organisms; as the result inflammatory nodules are produced, which may coalesce and produce small tumours, from which a purulent foul-smelling discharge may exude, and which proceed to the formation of ulcers.

No deterioration of health results, except as a result of pain produced by the raw surfaces and the absorption of septic material from the areas of suppuration.

The disease may commence in childhood, and may occur in more than one member of a family. It pursues a steadily progressive course.

ANATOMY.—The main anatomical changes are seen in the neighbourhood, and especially in the

upper part, of the pilo-sebaceous follicle. The mouth of the follicle is dilated and filled with a plug of horny epithelium, continuous with the crust or spine which projects from the follicle. The epithelium of the upper part of the follicle is much thickened, the horny layer especially being increased. Prolongations of the rete mucosum pass downwards into the neighbouring tissues in the well-known manner characteristic of papillomata. Various forms of degeneration make their appearance in this rapidly growing epithelium. In certain portions of the epithelium, especially in the upper part of the rete mucosum, and in the stratum granulosum, may be observed rounded or oval cells with double contour. They have usually a well-marked nucleus and possess clear or granular protoplasmic contents. These bodies were described by Darier as coccidia, but the general opinion of pathologists is that these structures are epithelial cells, which have undergone a peculiar hyaline degeneration.

DIAGNOSIS.—The disease is a rare one; but, in a well-marked case, the peculiar brownish-grey crust with the projecting cones and spines from the follicles, along with the tendency to form inflammatory nodules and superficial ulcers, render the recognition of the disease easy. In the early stages, especially before inflammatory reaction has taken place, the disease has been mistaken for lichen planus.

TREATMENT.—The main indications of treatment are thorough cleansing of the surface, and the subsequent use of antiseptic remedies to prevent the occurrence of local pyoderma. In one case under the writer's observation, great advantage was obtained by the use of baths of warm water and thorough application of soap to the parts; subsequently the use of salicylic acid the strength of 5 to 10 per cent. as plasters, unguents, or powders, proves satisfactory. The judicious application of the actual cautery or the use of the curette would no doubt be of much advantage in certain cases of the disease. No case of the disease has been cured.

JAMES GALLOWAY.

DAVOS-PLATZ, in North Engadine, Switzerland, an Alpine valley at an altitude of 5,177 feet. Well sheltered; atmosphere still and cold. A winter health resort, particularly for cases of phthisis. See CLIMATE, Treatment of disease by; and PHTHISIS.

DAY-BLINDNESS.—A disorder of vision, characterised by the patient being unable to see in the daylight; called also hemeralopia. See HEMERALOPIA; and VISION, Disorders of.

DEAFNESS.—Loss of the sense of hearing. *See* EAR, Diseases of; and HEARING, Disorders of.

DEATH, Signs of.—It is not always easy to determine when life has become extinguished. The two most obvious indications of animal life—the movements which accompany the circulation and the respiration—may be so feeble as to be indistinguishable to the unaided senses, or they may have ceased for a time and yet death may not have taken place. The tissues of which the body is composed do not lose their ‘vitality’ at the moment that the heart and lungs cease to act, and so long as this ‘vitality’ is retained resuscitation after temporary cessation of the circulation and the respiration is possible; hence the occasional difficulty of determining, at the moment, whether death has, or has not, taken place.

Cessation of the heart's action affords the most reliable proof that death has taken place. This, however, is not to be inferred from mere pulselessness, inasmuch as the heart may still be beating although no pulsation is perceptible in the arteries; under such conditions, the skilled use of the stethoscope over the cardiac area is needed in order to determine whether, or not, the heart continues to beat. In doubtful cases it is necessary to prolong the auscultation for a considerable time before forming an opinion, as the heart, if acting at all, may beat so feebly as to be barely audible. A number of devices have been suggested to enable unskilled persons to determine whether the circulation continues, or has ceased. The application of a tight ligature round a finger or a toe produces no change if the circulation has ceased; if it continues, the ligatured member, in time, assumes a livid tint from strangulation of the venous flow; and on removal of the ligature, a ring of arterial anæmia is observable round the part to which it was applied. If the hand be examined by transmitted light, the tips and the web of the fingers appear red and translucent during life; after death they are opaque up to their margins.

Cessation of the respiration is another sign that death has taken place. In doubtful cases, careful auscultation is needed to determine whether the lungs have or have not ceased to act; prolonged investigation being possibly required to justify the expression of an absolute opinion. When auscultation of either the heart or the lungs is performed with the object of determining whether the individual is living or dead, it should be conducted amid absolute silence. Among the popular methods of ascertaining the continuance or the cessation of respiration are:—holding a cold mirror before the mouth and nostrils, and then examining it for indications of moisture; placing a flock of cotton-wool on the lips to ascertain whether air enters or is expelled; placing a cup of water on the bare chest and observing whether the reflection from its surface moves or remains still.

With the cessation of the circulation, the skin becomes ashy-pale, and the tissues lose their elasticity. The eyeball becomes less tense, and the cornea becomes opaque. The pupils cease to react to light; and there is no vital reaction on the application of irritants to the skin. Though the body is dead as a whole, certain parts may continue to retain their independent vitality after somatic death. This is seen in the muscles, which may retain their electrical contractility from two to three hours after death.

Some of the subsequent changes which occur in

the dead body not only indicate the fact of death, but aid in fixing the probable period at which death occurred.

1. *The cooling of the body.*—The body after death, except under certain special circumstances, as in fatal cases of cholera and yellow fever, ceases to be a source of heat-production, and therefore is to be looked upon as an inert mass, possessed of a higher temperature than the surrounding medium, which parts with its heat according to certain physical laws. The superficial coldness of collapse, which is due to cessation of the peripheral circulation, must not be mistaken for the cadaveric coldness, for there is still an amount of internal heat which has to be parted with; and the body, cold to the touch before death, may after death rise in temperature, as the internal heat radiates. It is impossible to describe here in detail all the circumstances which modify the rate of cooling of the body; but it may be said in general that all circumstances which favour radiation, convection, and conduction of heat in inorganic bodies are equally applicable here, while the opposite conditions retard. Therefore a thick coating of adipose tissue, clothing, &c., retards cooling. The rate of cooling is not uniform, but is proportional to the difference between the temperature of the body and that of its surroundings; so that more heat is given off immediately after death than later on, when the temperature of the body more nearly approximates to that of its surroundings. During the first few hours the loss of heat may average 2° to 3° F. per hour; subsequently it is reduced to 1° , and less, per hour, the last few degrees of body-heat being dissipated very slowly. Under ordinary conditions the body takes from 15 to 20 hours to cool. It is to be borne in mind that the loss of body-heat must be progressively continuous before it can be accepted as a sign of death; the temperature at any given moment is valueless, as it may be considerably subnormal during life.

2. *Hypostasis or post-mortem staining.*—After death the blood gravitates into the capillaries and venous radicles of the most dependent parts of the body, both externally and internally, giving rise to livid discolourations termed hypostases, or *post-mortem* stains. These stains begin to appear in from four to twelve hours after death; occasionally they may be seen earlier. They first appear as detached patches, which subsequently coalesce and form large areas of discolouration. They are characterised by having well-defined margins; by not being elevated above the level of the surrounding skin; by occupying the most dependent parts of the body, except those parts which are in contact with the substance on which the body lies. So long as the blood remains fluid, the stains continue to form, and if the position of the body be reversed after they have begun to form, the original stains disappear and others make their appearance on the parts which are now most dependent. When the blood is coagulated, the stains cease forming, and those which are formed are permanent, notwithstanding any change in the position of the body. *Post-mortem* stains differ from bruises, inasmuch as the blood is still retained within the vessels—it has not extravasated into the tissues. In a bruise, the blood has escaped into the tissues from rupture of one or more vessels by violence. The distinction can be made by cutting through the skin: in a *post-mortem* stain, no blood is seen except that which escapes from the small

vessels which have been divided by the knife; in a bruise, blood which had been previously effused is seen.

3. *Rigor mortis*.—Immediately after death the muscles lose their tonus and become flaccid; after an interval of from four to ten hours, or earlier, they become stiff, the muscles of the lower jaw being first affected, and in the course of two or three hours the rigidity spreads over the whole body. The average duration of this rigor mortis, or cadaveric rigidity, is from twenty-four to forty-eight hours; but it may pass off in an hour or less, or it may last for a week or more. Cadaveric rigidity is due to coagulation of the muscle-plasma and formation of myosin within the sarcolemma of the muscle-fibres. Previous to its occurrence the muscles undergo slow *post-mortem* contraction, and, on account of the greater power of the flexors as compared with their antagonists, the tendency is for the limbs to become flexed and to stiffen in the bent position. If, after cadaveric rigidity is fully developed, a joint be forcibly moved to and fro, the stiffness does not return: in cataleptic rigidity in the living, forcible straightening of a limb is followed by renewed flexion and rigidity. During the state of absolute cadaveric rigidity, some indications of the presence of joints may be evoked by attempts to bend the limbs; on the other hand a frozen body is as rigid as a marble statue.

The period after death at which cadaveric rigidity occurs is to a great extent determined by the vitality of the muscles at the moment of dissolution: if their vitality is high, the onset of cadaveric rigidity is delayed; in enfeebled or exhausted subjects it comes on quickly. Generally speaking, the earlier rigidity appears, the sooner does it pass off.

As already stated, the ordinary type of cadaveric rigidity is preceded by a period of muscular relaxation which, however fugitive, is sufficient to allow the limbs and the body to obey the laws of gravity before becoming stiff. An exceptional form of rigor mortis occasionally occurs which is known as cadaveric spasm, in which the final vital contraction is immediately prolonged into the rigidity of death. Illustrative of this instantaneous rigor are the following cases: a man was shot dead, the bullet passing through the base of the skull; he fell on his back with the arms stretched forwards and the legs extended in the air, the whole body being at once so stiff that it could be moved about, by the arms, like a piece of wood. The dead body of a man poisoned with water-gas was found standing upright with one hand grasping the rung of a ladder. A revolver is not infrequently found tightly grasped in the hand of a suicide who has shot himself through the head. Merely a momentary period of muscular relaxation after death would have sufficed to prevent the occurrence of these and like instances. Experimental evidence leaves little doubt that the nervous system is concerned in the production of all forms of *post-mortem* muscular contraction; this is especially exemplified in instantaneous rigor.

4. *Putrefaction*.—Free exposure to air accelerates putrefaction, especially if the air is moist and warm; if the body be protected from air by clothing, or by being enclosed in an air-tight coffin, putrefaction occurs more slowly. In a body exposed to air at a moderate temperature, the signs of putrefaction, which afford the sole absolute proof of death, are first seen, under ordinary circumstances, about the second or third day after death, as a greenish dis-

colouration on the abdomen. This is quickly followed by foci of discolouration on other parts of the body, which gradually spread all over it; after a time the soft structures are entirely disintegrated. Internally, the mucous membrane of the larynx and trachea is the first tissue to succumb; it begins to undergo changes in colour and consistence shortly after the early external signs appear on the abdomen. The less compact tissues putrefy first; those which are fibrous, such as the bladder and the blood-vessels, resist longer, the uterus resisting the longest of all, being usually recognisable after the rest of the soft structures are reduced to a pulp. In bodies submerged in water the external putrefactive changes commence on the face, instead of on the abdomen, as in air, and then spread downwards; the progress of putrefaction is slower in water than in air. These statements apply equally to the bodies of those who have been drowned and to bodies submerged immediately after death from other causes than drowning.

The rate of putrefaction is extremely variable: so much so as to render it very unsafe to make any dogmatic assertions, based on the stage of putrefaction reached by the body, as to the length of time that has elapsed since death took place. Cases have been recorded in which ten or twelve hours after death putrefaction was as far advanced as is usually met with in as many days; and on the other hand putrefaction has been so strangely retarded that three or four weeks after death scarcely any evidence of its presence could be detected.

J. DIXON MANN.

DEBILITY (*debilis*, feeble).—SYNON.: Feebleness; Weakness; Asthenia; Fr. *Faiblesse*; Ger. *Schwäche*.

DEFINITION.—The body or any of its organs is said to be in a state of debility when its vital functions are discharged with less than the normal vigour, and when the amount of activity which it displays and of work which it can accomplish is diminished.

ÆTIOLOGY.—Debility is frequently constitutional and inherited; but it is more often developed after birth. It is most commonly due to impaired nutrition, whether this be prolonged and moderate, as in defective hygiene or chronic illness, or, on the other hand, rapid and extreme, as in acute disease. Such failure of nutrition, when local, may arise from the blood being deficient in quality or quantity, or possibly from some interference with the nervous supply. Another frequent cause of debility is the abuse of the affected organ. Over-use of a part leads to fatigue, and if frequently repeated to exhaustion, the chief feature of which is extreme debility, as in cases of sustained and mental exertion, or of repeated strain of the heart. On the contrary, an organ may become feeble from want of exercise. Paralysed muscles furnish the best examples of this condition, but the same may be seen in all organs after unnatural rest. Debility is more liable to occur at certain ages, such as the periods of active growth and development, and in connection with the menopause; and it is one of the manifestations of advanced life.

SYMPTOMS.—The natural ability of the organs to perform their functions varies extremely with sex, age, previous exercise, and many other circumstances. Debility, or the loss of this functional power, is therefore frequently ill-defined; and, when un-

questionably present, may vary greatly in different cases, from a condition in which fatigue comes on only somewhat earlier than usual, as in muscular debility, to a state in which the slightest exertion may exhaust the whole of the vital energy, and the functional life of the part may cease—as is seen in the cardiac asthenia of acute fevers, and scarcely less markedly in certain chronic diseases, such as idiopathic anæmia and Addison's disease.

Debility may be *general*, affecting the whole body; or *local*, individual organs only being involved. Speaking broadly, the symptoms of debility of an organ may be said to be chiefly two. These are, first, increased irritability, or an unnatural readiness of the part to respond to stimulation; and, secondly, a tendency to untimely exhaustion. The phenomena of irritability and exhaustion naturally vary with the organ involved. The symptoms of muscular asthenia are few and simple; those of digestive feebleness are more complex; and in debility of the nervous system the whole of the mental processes, as well as the functions of organic life, may be involved. For a specific account of the phenomena of each of these cases, and of debility of other parts, the reader is referred to the articles upon diseases of the several organs.

DIAGNOSIS.—Debility pure and simple is, as a rule, easily distinguished from *disease* by the absence of all evidence of organic alteration, and especially of physical signs of anatomical change. It is more difficult to separate debility from *disorder* or *derangement*, but careful observation will generally determine in the case of pure debility that the functions are normally discharged as long as the demands made upon them are not excessive.

PROGNOSIS.—Debility due to acute disease may, in the absence of complications, be expected to disappear during convalescence. If the cause have been more chronic, and be less easily removed, recovery will certainly be more slow and less satisfactory. The prognosis of inherited constitutional debility, as regards its disappearance, is nearly always unfavourable.

TREATMENT.—Debility must be treated according to its cause. If nutrition have failed, it must be restored as far as possible; and until this can be done, stimulants and suitable tonics are indicated—especially in the case of acute disease. Rest is of the first importance in most instances; and frequently it is alone sufficient to restore the vital force. In a few cases, however, the opposite line of treatment must be followed, as in muscular debility from want of exercise or in some forms of paralysis. Where the vital activity is low from constitutional defect, age, or sex, the condition may not be remediable; and the treatment of such cases is chiefly prophylactic. The principal indication then is to secure the subject of debility against exposure to damaging influences.

J. MITCHELL BRUCE.

DEFÆCATION, Disorders of.—Disorders of the act of defæcation are characterised either (1) by an undue relaxation of sphincters and other perineal muscles by which the rectal contents are normally retained, leading for the most part to involuntary action of the bowels; or (2) by insufficient relaxation, due to spasm of these structures, tending to favour fecal accumulation and retention. Associated respectively with these opposite conditions may

be an exaggerated or diminished peristalsis, and an increased or decreased power of the expiratory muscles.

1. Involuntary defæcation.—Since the nervous governance is only in part under the control of the will, it would follow that the chief causes of the involuntary discharge of the fæces are to be found in those conditions which diminish the normal tonicity of the sphincter. At the same time, any circumstances which may increase the pressure of the abdominal muscles, or the activity of the bowels, much beyond what ordinarily occurs, may lead to an involuntary discharge.

ÆTIOLOGY.—The causes of involuntary discharge of fæces may be thus grouped:—

1. Violent contraction of the expiratory muscles. This cause of involuntary defæcation is such as may be induced by strychnine-poisoning. It is of rare occurrence in tetanus.

2. Increased peristaltic action of the intestines. Increased peristalsis is chiefly dependent on causes of irritation situated in the bowels themselves, such as inflammation or ulceration of the walls; irritating contents; or worms. Extreme fluidity of the fæces is frequently sufficient to induce their partially involuntary discharge, and is noticeable in the looseness sometimes associated with accumulation of fæces in the lower bowel, and the diarrhoea of infants and children.

3. Abnormal relaxation of the sphincter ani. As already said, the previously mentioned causes are powerless to produce the discharge of the fæces until the sphincter yields; and how far the sphincter may relax as a result of their efforts, or independent of them, is not easy to determine. The nervous influence emanating from the lumbar centre which normally determines the tonic state of the sphincter may be inhibited by various causes:—

a. Reflexly.—Reflex inhibition of the sphincter ani may arise from such causes as worms, fissure of the anus, or calculus vesicæ. It is quite true that the immediate result of irritating the sphincter is to determine in a reflex manner an increase of its contraction, as may be experienced in introducing instruments into the rectum; but it would also seem that at a certain point the irritation may lead to an arrest of the tonic influence, and so allow the sphincter to yield, and this is particularly the case with affections of the bladder.

β. From cerebral disease.—That certain emotional conditions may lead to involuntary defæcation is well known, and it is not an uncommon event for a similar result to follow some diseases of the cerebral centres. The paralysis of the sphincter which occurs under these circumstances is brought about by inhibition of the normal tonic stimulus discharged from the lumbar centres. Involuntary evacuations frequently occur in epileptic fits; in states of profound coma induced by apoplexy, or by opium- and other forms of poisoning; and in death by hanging or suffocation. Its occurrence can scarcely be regarded as indicating lesion of any one part of the cerebral centres, but rather as a result of general brain-states. It constitutes a troublesome complication in such chronic forms of brain-disease as softening, general paralysis of the insane, &c. In these cases the act of defæcation takes place whenever the need of expulsion arises, without any power of the patient to induce or restrain it.

γ. From disease of the spinal cord.—The relaxa-

tion of the sphincter may be produced by disease or injury of the lumbar enlargement of the spinal cord. Inasmuch as it is from the cells of this region that the normal tonic influence is understood to issue, destruction of the nervous tissues can readily be understood to prevent the origination of such stimulus to contraction. Certain lesions also above the lumbar enlargement may lead to involuntary passing of feces by interfering with the cerebral control of the sphincter. It is noticeable that diffused changes in the substance of the cord, such as acute or chronic myelitis, intra-spinal hæmorrhage, and compression, are far more frequently accompanied by paralysis of the sphincter than are the inflammatory affections of the spinal meninges or the diseases limited to special tracts—e.g. anterior polio-myelitis, lateral sclerosis, progressive muscular atrophy.

8. *From local lesion.*—The control over the sphincter may be lost from injury to the muscle itself. This occurs in laceration of the perinæum, involving the lower end of the bowel; sometimes it is the result of surgical operation, such as deep division of the sphincter in connection with fistula.

TREATMENT.—No direct treatment of incontinence of feces is of avail, beyond removal, when possible, of the cause. This may be done with success when the involuntary discharge depends on the presence of some local irritant, such as worms or fecal impaction. As a distressing complication of certain diseases of the nerve-centres, which are too frequently incurable, little remains to be done for it beyond adopting such measures as will permit of the escape of the discharge with every regard to cleanliness.

II. *Inadequate defæcation.*—Serious troubles in defecation may also arise from excessive as well as from weakened action of the *muscles* concerned in this function. The sphincter may be irritable or subject to spasm, and resist too forcibly the expulsive actions of defecation (see ANUS, Diseases of). Or the muscular fibres of the rectum may lose their tone, and be defective in power and incapable of properly extruding the feces. Patients thus situated are often obliged, when at stool, to use the finger to dislodge masses retained in the weakened bowel. An atonic condition of the rectum usually arises from over-distension. It may be produced as the result of accumulations, or by too free and frequent use of enemata, the quantity injected being so large as to dilate the bowel and impair the power of its muscular coat. This atonic state of the bowel is itself apt to give rise to fecal accumulation, and thus the evil is materially intensified.

The rectum may become gradually dilated and blocked up by a collection of hard dry feces, which the patient has not the power to expel, being unable from loss of tone in the distended bowel to overcome the resistance of the sphincter to the passage of so great a body. Some indurated lumps from the sacculæ of the colon, on reaching the rectum, perhaps coalesce so as to form a large mass; or a quantity accumulated in the sigmoid flexure, on descending into the rectum, becomes impacted there. In several instances a foreign body has been found in the centre of the mass. The persons most subject to these troubles are those enfeebled by age or disease, especially women. They may also occur in infants who have been operated on for imperforate anus, when the artificial aperture contracts, or is left too small for the free passage of the feces.

In these cases the distension of the bowel is sometimes excessive, and its expulsive functions are seriously impaired and weakened. See CONSTIPATION; INTESTINAL OBSTRUCTION.

SYMPTOMS.—Such a collection gives rise to considerable distress, a sensation of weight and fullness in the rectum, tenesmus, and forcing pains which women liken to those of labour. In cases of some duration, where the hardened feces do not quite obstruct the passage, they excite irritation and a mucous discharge, which, mixing with recent feculent matter passing over the lump, causes the case to be mistaken for diarrhoea.

TREATMENT.—Injections have no effect in softening the indurated mass: they act only on the surface, and return immediately, there being no room for their lodgment in the bowel. The practitioner on passing his finger finds the rectum blocked up with a large lump, which feels almost as hard as a stone. In such cases the only mode of giving relief is by mechanical interference. The mass requires to be broken up and scooped out—an operation which may require the use of a cocaine suppository, or the administration of an anæsthetic. After the breaking up and extraction of the larger portions, injections of soap and water will be sufficient for the removal of the remainder.

W. H. ALLCHIN. FREDERICK TREVES.

DEFERVESCENCE (*de*, down; and *fervesco*, I grow hot).—The decline of fever, characterised by a fall of temperature and of pulse, and by other phenomena. See FEVER.

DEFORMITIES.—See MONSTROSITIES.

DEFORMITIES OF THE CHEST.—See CHEST, Deformities of.

DEGENERATION.—SYNON.: Fr. *Dégénérescence*; Ger. *Entartung*.

DEFINITION.—Degeneration is the name applied to pathological processes, by which abnormal substances are formed within living cells, or the entire cells are transformed into abnormal material. The products of degeneration are for the most part inert bodies, the existence of which indicates or causes a defective vitality in the cells. They may theoretically be formed in any one of the following ways:—(1) by breaking down of the cell-protoplasm into simpler chemical substances; (2) by building up of unusual compounds, owing to disorder of the 'digestive' function of the cells, by which they assimilate the nutritive materials brought to those by the lymph; and (3) by failure of the cells to eliminate products which are normally excreted or used up as soon as they are formed. The majority of degenerative processes—granular, fatty, colloid, and mucoid—are generally assigned to the first of these classes. Amyloid degeneration may perhaps be placed in the second category; and it is possible that some instances of fatty accumulation may be due to the third method of causation, a deficient supply of oxygen causing a diminished destruction of fat.

The name 'degeneration' is also sometimes applied to the process by which dead or dying tissues become the seat of a deposit of calcareous material which soaks into them from the lymph (calcareous degeneration). To this the name 'infiltration' may more properly be applied. Certain conditions in which an abnormal amount of fat is stored up

in tissues which physiologically contain this substance (e.g. liver, connective tissue) have also been termed infiltration, but it seems better to keep this term for the purely passive process of calcareous infiltration, and to call the former 'fatty accumulation.'

ÆTIOLOGY.—The causes leading to the defective nutrition of cells, from which the different forms of degeneration result, are not certainly known. A defect in the quantity of nutritive material supplied to a part may apparently lead to fatty degeneration of cells; defects in the quality of the lymph are probably responsible for other forms of degeneration. There is ground for believing that the poisonous products of bacteria are the cause in many cases, of which caseation, due to the toxins of the tubercle-bacillus, and amyloid degeneration afford the most clearly established examples. Other poisons, such as phosphorus, arsenic, mercury, and probably alcohol, may give rise to fatty change.

VARIETIES.—The following forms of degeneration may be distinguished: (1) Granular degeneration (cloudy swelling); (2) Fatty degeneration; (3) Hyaline degeneration; (4) Amyloid degeneration; (5) Colloid degeneration; (6) Mucoid degeneration. Reference must also be made under the present heading to so-called 'Fibroid degeneration,' although this is not an instance of a degeneration in the exact sense of the word.

The substances the formation of which characterises the different forms of degeneration are not always sharply differentiated one from another; indeed, it is probable that variations in chemical composition may be met with in different specimens of the same degeneration. There is probably a physiological prototype for all the degenerative materials, colloid being found physiologically in the thyroid gland, mucin in goblet-cells of mucous membranes, and amyloid possibly in the coats of normal arteries (Krawkow).

1. Granular Degeneration (SYNON.: cloudy swelling; parenchymatous degeneration; albuminous infiltration).—This change in the protoplasm of cells is found chiefly in cases associated with a high degree of pyrexia. It is uncertain whether the alteration in the substance of the cells is directly due to the heat to which they have been exposed, or is caused by toxins circulating in the blood and lymph. It is conceivable that certain albuminous constituents of the cell may coagulate at a temperature only slightly higher than that of the body in health, just as it has been shown that muscle-proteid contains several forms of myosin, with different coagulation-points. The other explanation seems, however, more probable: since a change resembling cloudy swelling may be found in poisoning by phosphorus and other substances, without the occurrence of pyrexia.

The liver, kidneys, heart, and voluntary muscles are the seats of cloudy swelling; it has also been stated to occur in the epithelium of the lung, and it is probable that the change is general throughout the body. To the naked eye affected organs are usually somewhat pale, but may be hyperæmic; they are slightly swollen and softer than normal, and any distinctive markings on cut surfaces tend to appear blurred and indistinct. Under the microscope the cells look granular, presenting an appearance somewhat like ground glass. The granules are unstained by osmic acid and insoluble in ether,

but soluble in acetic acid: they are therefore formed of albuminous material. In advanced cases fatty globules appear in the granular cells; and it is probable that cloudy swelling constitutes the first stage of fatty degeneration. Cells which have undergone granular degeneration may recover when the cause of the condition is removed. The weakness of the heart in fevers, shown by the indistinctness of the first cardiac sound, is due to granular degeneration of the muscle-fibres; the lack of secretion from organs such as the kidneys, liver, and salivary glands seen in febrile conditions may be associated with this change in the glandular cells.

2. Fatty Degeneration.—See FATTY DEGENERATION.

3. Hyaline Degeneration (ὑαλος, crystal or glass).—This variety of degeneration is met with chiefly in the coats of arteries and in connective tissue. It is also found in old thrombi and in some inflammatory products (false membranes, scar-tissue). Little is known of the nature of this change. The affected tissue is homogeneous, translucent and somewhat swollen. Coagulated fibrin may assume a hyaline appearance; and some authorities have described a 'fibrinous degeneration' by which cells of connective-tissue are converted into this substance; but the existence of this metamorphosis is doubtful. In some cases hyaline degeneration appears to be a preliminary stage in the formation of amyloid material.

The peculiar change in muscle-fibres known as *Zenker's degeneration* may be considered a form of hyaline change. The condition is most commonly met with in enteric fever, but may occur in any acute infective disease as well as in the neighbourhood of burns and of inflammatory foci. In enteric fever the muscles principally affected are the adductors of the thighs, the recti abdominis, and the diaphragm. The fibres which have undergone this change are homogeneous and translucent; the normal striation is lost; and the fibres tend to break up transversely into irregular masses. Rupture of the muscle-fibres may occur and may be associated with effusion of blood into the sheath of the muscle. Probably the process is one of coagulation-necrosis, the cells being poisoned by the products of micro-organisms, and the hyaline change indicating their death. Some authorities, however, consider that rupture of the fibres first takes place, and that the degeneration is secondary to this occurrence.

4. Amyloid degeneration (SYNON.: lardaceous degeneration; albuminoid degeneration; waxy degeneration).—See AMYLOID DISEASE.

5. Colloid degeneration (κόλλα, glue).—This change is met with physiologically in the thyroid gland, the acini of which always contain colloid material. Pathologically it occurs principally in some forms of carcinoma arising in the abdominal viscera (stomach, intestine, omentum) and in ovarian cysts; in the thyroid gland the change may occur to an excessive extent and constitute one variety of goitre. Colloid degeneration was formerly confused with mucoid change; the material however differs from mucin in being somewhat less fluid, in forming a precipitate with acetic acid, and in containing a certain amount of sulphur. It first appears within the cells as transparent drops; gradually the cell becomes distended with the new material, the nucleus and protoplasm disappear, and the colloid substance coales-

ces into large shining masses that may present under the microscope a concentrically laminated appearance. To the naked eye the affected tissue appears studded with minute shining points. Colloid offers no characteristic staining reaction, and is gelatinous in consistency when fresh; when treated with alcohol it becomes tough or brittle. Tumours which are the seat of colloid change tend to grow slowly and may attain a large size. Softening of the colloid masses may give rise to the formation of cysts. The cause of the change is unknown.

6. **Mucoid degeneration** is found physiologically in the cells of the umbilical cord (Wharton's jelly) and in certain cells of mucous membranes (goblet-cells) which break down into mucous material, and thus keep the surface moist. In catarrhal states of mucous membranes the change occurs to a greatly increased extent, giving rise to the characteristic secretion seen in coryza, bronchitis, or cystitis. This form of degeneration is also met with in tumours (myxoma, myxosarcoma), and in ovarian cysts; and as a senile change in cartilage and bone. The affected cells first contain within them drops of clear mucus; these drops increase in size, and the whole cell is ultimately converted into the new material. In other cases, the intercellular substance is the seat of the change. Tissues thus degenerated are transformed into a soft jelly-like substance, which is colourless and transparent, or may have a pink colour due to the presence of blood. Cysts may be formed by softening of the mucoid material. In the disease known as myxœdema, it was originally stated that the subcutaneous tissues contained an excess of mucin, but this view is not now generally accepted.

7. **Pigmentary degeneration** occurs principally in the cardiac muscle, constituting the condition known as 'Brown Atrophy of the Heart.' The muscle-fibres waste, the protoplasm breaking down into masses of brownish pigment, which are seen scattered throughout the fibres or grouped round the nucleus. The condition is met with in old persons or in the course of wasting diseases, and may be associated with some fatty degeneration. *See PIGMENTATION.*

8. **Calcareous degeneration.**—*See CONCRETIONS.*

9. **Fibroid degeneration.**—This term is sometimes applied to any condition in which the essential cells of an organ atrophy, and are replaced by fibrous tissue. The best example of this occurs in the heart (*see HEART, Fibroid Disease of*), when the coronary arteries are narrowed by atheroma, and the blood-supply to the myocardium is thus rendered insufficient. Under these circumstances the muscle-fibres become fatty, die, and are absorbed, their place being taken by an increased growth of the connective tissue of the part, the cells of which are less highly organised and are therefore able to live on a smaller supply of nutriment. The process is in reality one of repair (cicatrisation), secondary to degeneration of the muscular elements. A similar process is seen in the central nervous system following upon degeneration of special tracts of fibres. *W. CECIL BOSANQUET.*

DEGLUTITION, Disorders of.—**SYNON.:** Dysphagia; Disturbances of Swallowing; Fr. *Troubles de la Déglutition*; Ger. *Störungen des Schluckens*.

All affections of the throat modify in some way

the power of swallowing, and may thus render the act of deglutition painful and difficult. Thus:—1. We meet with it as one of the symptoms in acute catarrh of the pharynx, in tonsillitis, and in ulceration of the throat. 2. Similarly, diseases of the larynx may give rise to disorders of deglutition; such as laryngitis, inflammation of the perichondrium, and laryngeal polypi. 3. Specific diseases—for example, phthisis, syphilis, cancer, scarlet fever, measles, and croup—are another fertile cause of difficulty of swallowing, owing to their affecting the throat in various ways. 4. So also are nervous affections—for instance, post-diphtheritic paralysis, hysterical disorders, general paralysis of the insane, progressive muscular atrophy, and glosso-pharyngeal paralysis. 5. Affections of the salivary glands, such as parotitis, may interfere with deglutition. 6. Œsophageal disorders, whether functional, or causing organic obstruction, are important causes of dysphagia. 7. Difficulty of deglutition may result from pressure upon some part of the œsophagus, as by an aneurysm of the thoracic aorta, a solid tumour, whether malignant or benign, or a retro-pharyngeal abscess. 8. It may be set up by the character of substances swallowed—e.g. when these are irritant, corrosive, or very hot. All these causes, though in different degree, offer some impediment to the act of deglutition.

SYMPTOMS.—Difficulty in the act of swallowing is the essential symptom: this is often accompanied by pain; indeed not infrequently, in cases of inflammation of neighbouring structures, swallowing is difficult only because it is painful. When the calibre of this portion of the alimentary canal is reduced by disease affecting its own structure, or when it is subjected to pressure by an aneurysm or by morbid growth, a mechanical impediment is set up, which necessitates a certain amount of voluntary effort to accomplish the act. This supplementary aid is usually sufficient to propel the bolus onwards; and deglutition, though slower than in health, and usually attended with pain, may be successfully performed. It occasionally happens, however, that the obstruction is so great as to prevent the passage of at least the larger portion of the food downwards, and regurgitation takes place through the mouth or nostrils.

A similar result is brought about when paralysis affects any portion of the muscular structures concerned in the act of swallowing, but obviously in a different manner. For example, in post-diphtheritic paralysis, in consequence of the implication of the soft palate and neighbouring structures in this loss of power, the food, instead of passing into the gullet, returns through the posterior nares.

The dysphagia due to spasm of the pharynx or œsophagus in neurotic persons differs from those forms already described in the inconstancy of its occurrence and the variability of its position.

For the examination of the œsophagus *see* (ŒSOPHAGUS, Diseases of).

TREATMENT.—This will manifestly depend upon the recognition of the cause which gives rise to the impediment in swallowing. Cases of simple catarrh of the mucous membrane of the throat, and those produced by the action of irritant substances generally, yield, after a short interval, to the use of bland articles of diet and demulcents, such as olive oil, milk, linseed tea, &c. It must, however, be borne in mind that permanent stricture of the

œsophagus may be the result of causes such as those last mentioned. Of course, when post-pharyngeal or other form of abscess is the cause of the dysphagia, the evacuation of its contents will give immediate relief. In dysphagia dependent upon diminution of the calibre of the œsophagus, the question of its treatment by the use of bougies, stomach-tubes, and surgical interference should be considered. See **ŒSOPHAGUS**, Diseases of.

The dysphagia dependent upon specific disorders of the larynx, such as that occasioned by phthisis, syphilis, or cancer, may often be greatly mitigated by the use of warm medicated sprays containing sedative drugs, such as bromide of ammonium, opium, or chlorine-water; a 15 or 20 per cent. solution of menthol in liquid vaseline or olive oil, injected into the larynx and pharynx; also a 4 per cent. solution of cocaine. In cases of a nervous origin, the treatment must bear reference to the general nervous disorder of which the dysphagia is but a symptom. Hysterical dysphagia may occasionally be removed by the application of galvanism in the neighbourhood of the œsophagus. Post-diphtheritic dysphagia usually disappears as the health of the patient improves, and is to be treated by the administration of nerve tonics, such as strychnine, iron, and quinine.

The food should be of semi-solid consistence, concentrated, and small in bulk—e.g. iced milk, cream-ice, jellies, switched egg or the unbroken raw egg, custard, and oysters.

C. MUIRHEAD.

DELHI SORE, or BOIL.—See **ORIENTAL SORE**.

DELIRIUM (*deliro*, I rave).—A derangement of consciousness, characterised by incoherence of thought, and evidenced by various expressions and actions. See **CONSCIOUSNESS**, Disorders of.

DELIRIUM TREMENS (*delirium tremens*, trembling delirium).—A form of acute alcoholism, chiefly characterised by delirium and tremors. See **ALCOHOLISM**.

DELUSION (*deludo*, I deceive).—An unfounded belief in regard to some fact or occurrence which almost invariably concerns the patient, and of the falsity of which he cannot be persuaded, either by the evidence of his senses, by his own knowledge and experience, or by the declarations of others. Such delusions, when distinguished from merely erroneous judgments upon complicated or abstract questions, generally indicate insanity. See **CONSCIOUSNESS**, Disorders of; and **INSANITY**, **DELUSIONAL**.

DEMENTIA (*de*, without; and *mens*, a mind). **SYNON.**: Fr. *Démence*; Ger. *Wahnsinn*.

DEFINITION.—A mental weakness, a deficiency or suspension of mental power rather than an aberration. It may be *primary* and curable, or *secondary* and for the most part incurable, depending on brain-disease, chronic alcoholism, or chronic insanity; it may be the result of disease in infancy or of senile decay, or may be congenital and is then termed Idiocy.

1. **Acute Primary Dementia.**—This has also been called *Stupor with Dementia*, and has been confounded with *Stupor with Melancholia* (*melancholia cum stupore*), a different disorder, occurring at a different age. It comes on rapidly, is marked

by profound vacuity and loss of mental power, yet passes away leaving the patient sane as before.

ÆTIOLOGY.—The patients are young persons, boys and girls,—more frequently girls. The complaint is seldom seen in any case beyond the age of thirty, and chiefly in those under twenty. It seems to be a collapse of all mental power, due to great physical weakness and deficient nerve-force. Owing to imperfect development, to bad food and living, or to the patients having outgrown their strength, the mental condition becomes so weakened that, with or without some moral cause, as a fright or a scolding or something apparently more trivial, or after some illness slight or severe, they suddenly or gradually present that condition which is now to be described. If the immediate exciting cause is some mental shock or fright, the symptoms may come on rapidly. If they are due to ill-health or some protracted exhausting occupation, the access may be gradual; and if they are set down to sulkiness, temper, or idleness, the measures adopted for the correction of the latter may quickly indicate the real state of things.

SYMPTOMS.—Nothing can appear more hopeless than the appearance many of these patients present. The face is vacant, with a fatuous grin, and often the saliva dribbles continuously. The sufferer sits motionless and lost, or automatically wags the head, snaps the jaws, or moves the limbs for hours together, unconscious of fatigue. Or if a limb is placed in any position, it is retained there for a time in a way that no effort of will could accomplish. There may be a repetition of some word or sentence, but all conversation is abolished, and the patient has to be fed, washed, and tended like a baby.

The physical condition of these patients is peculiar, and corresponds closely to the mental. The heart's action and the circulation are so reduced in strength that the blood in the extremities is stagnant. Hands and feet are blue with cold even in the heat of summer. In cold weather they are covered with chilblains, and great care must be taken, otherwise these will give rise to obstinate sores. The tongue is pale and flabby. The pupils are dilated. There is no rapid emaciation, for the waste here is not great; neither is sleep absent, as in mania, but it is irregular and uncertain.

PATHOLOGY.—The external physical manifestations sufficiently indicate the condition of the brain in these patients. It is the very opposite of that in acute sthenic delirious mania. In the latter there is an excessive discharge of nervous force, a hyperæmic state of brain, and rapid brain-circulation; the whole leading in a short time to death by exhaustion, if relief does not come. In acute dementia we see the very opposite. The brain-action is reduced to the lowest point, and the circulation is stagnant, as in the extremities, giving rise to passive congestion and œdema.

COURSE, TERMINATIONS, AND PROGNOSIS.—In acute dementia there is no sudden exhaustion; but death, if it occur, is caused, not by the brain-disease, but by a general failure of the bodily strength or by some low form of lung-disease—phthisis, pneumonia, or gangrene. Death, however, in this disorder, is the exception. When taken in time and properly treated, the majority of these seemingly hopeless cases recover, and recover perfectly.

TREATMENT.—The treatment of acute dementia may be carried out in a family, or even at home, if means are ample, and if the necessary measures are

strictly enforced. But it may be necessary to feed the patient by force, and that for a considerable time, and relations do not always care to enforce this to the extent required. Abundant nutrition is imperatively demanded in order to restore the force that is so greatly in default; and unless abundant nutrition is administered, there will be no recovery, but the patient will die, or sink into permanent dementia. There is not, as a rule, violent resistance to food, but it may be kept in the mouth without being swallowed, and care must be taken in feeding, even if a stomach-tube be not necessary. Food should be given frequently, and so a habit of taking it engendered. Stimulants—wine and brandy—will be necessary, especially in the early stages. Equally necessary is warmth; an amount of heat is required which to those in health would be oppressive, for the greatest heat of summer fails to warm the hands and feet. Warm clothing must be provided, and the circulation aided by a short sharp shower-bath, cold or tepid, and plenty of friction afterwards. Exercise is useful for the same purpose, but this is to be taken under proper supervision, for it must not be fatiguing, and due regard ought to be had to the debilitated state of the individual. In addition to the stimulus of the shower-bath, that of electricity is of great use in acute dementia. Here, and in certain cases of melancholia, marked benefit follows the application of the continuous current. Of drugs the most useful appear to be iron and quinine. The former, in this as in almost every form of insanity, is a most valuable tonic: the choice of the particular preparation should depend upon the state of the patient at the time.

2. **Secondary Dementia.**—This is a sequel of some prior disorder, and with rare exceptions incurable, though there may be some amelioration.

a. It may be due to fits, brain-disorder, or injury in infancy or childhood, whereby mental development is arrested and the patient never attains the standard of his race.

b. It is often the termination of other forms of insanity, as mania, melancholia, or general paralysis. It frequently follows an attack of insanity in young people who are strongly predisposed and the victims of masturbation. It may be the final stage of those who have experienced repeated attacks of mania and recovered from them. At length they fail to rally, and sink into dementia.

c. It may be the outcome of chronic alcoholism, loss of memory being the first symptom of a progressive dementia. From this surprising recoveries sometimes occur if the drinking is not of long standing and is entirely stopped.

d. It may be caused by epileptic attacks if they are numerous, and here also loss of memory is often the first symptom. If the epilepsy ceases, recovery or great improvement may take place. The dementia will vary according to the number and severity of the fits.

e. It may follow apoplectic seizures where there has been hæmorrhage or softening. The prognosis here is very unfavourable.

f. It is frequently the result of senile decay. The age at which it commences will vary considerably, some becoming senile prematurely, worn out by a hard life at a comparatively early age.

PATHOLOGY.—The morbid appearances in those who die in a state of dementia differ according to the origin of the disorder. The alcoholic will differ from the epileptic (*see* ALCOHOLISM; EPI-

LEPSY). The lesions in dementia following years of insanity will not be the same as those of old age, but, generally, signs will be found of old-standing disease of brain and vessels, atrophy of convolutions, degeneration of cells, thickening of membranes, and increase of neuroglia. *See* INSANITY, Pathology and Morbid Anatomy of.

TREATMENT.—Though most patients suffering from chronic secondary dementia are incurable, much may often be done to improve their condition, if through carelessness or ignorance this has been greatly neglected. Their friends think that nothing can be done or need be done, and they are allowed to lie in bed, often in a filthy condition, or roam about and get into mischief for want of care and skilled attendance. Many demented patients have far more mind than is generally imagined by the uninitiated, and can be taught to be cleanly, to take their meals in an orderly fashion, and to keep themselves tolerably neat. They are susceptible of amusement, and open to reward for good behaviour. The vital powers of demented patients are low, and they suffer much from cold. In winter their minds, like their bodies, are enfeebled, and with warm weather they recover somewhat of their energy. There is a tendency in many of these patients, especially women, to become very fat, and in this condition they are subject to bronchitis, and may succumb to acute attacks of this disorder. They require warmth and good diet, for it is difficult to make them take sufficient exercise.

G. F. BLANDFORD.

DEMODEX (*δημός*, fat; and *δῆξ*, a worm).—*See* ACARUS.

DEMULCENTS (*demulceo*, I stroke softly).

DEFINITION.—Substances which soften, protect, and soothe mucous membranes. They are generally of a mucilaginous character. When applied to the skin they are termed *emollients*.

ENUMERATION.—The demulcents in ordinary use are: Linseed-tea, Gum, Starch, Bread, Honey, Figs; Linseed, Almond, and Olive Oil; Glycerine, Barley Water, White-of-Egg, Gelatine, and Isinglass.

ACTION.—The chief action of demulcents is a mechanical one, in forming a smooth soft coating for an inflamed mucous membrane, and thus protecting it from external irritation.

T. LAUDER BRUNTON.

DENDRONS.—The processes of a nerve-cell, other than the axon, are called *dendrons*. The dendrons are the channels by which impulses are conveyed to the cell. The branches of the dendrons are called *dendrites*.

DENGUE.—**SYNON.**: Dandy Fever (West Indies); Three-day Fever; Break-bone Fever; Fr. and Ger. *Dengue*.

DEFINITION.—An infectious eruptive fever; commencing suddenly; and characterised by severe pain in the head and eyeballs, swelling and pain in the muscles and joints—prone to shift suddenly from joint to joint; a primary diffuse erythematous and secondary rubeoloid eruption; catarrhal symptoms, sore-throat, and congested conjunctivæ.

NATURAL HISTORY AND GEOGRAPHICAL DISTRIBUTION.—Dengue occurs epidemically and sporadically in India, Burmah, Persia, Asia Minor, Syria, Egypt and other parts of Africa, North and

South America, the West Indies, and Australia. Epidemic visitations of dengue, extending over wide tracts of country, occur at considerable intervals. In suddenness of appearance and rapidity of diffusion these epidemics resemble influenza.

SYMPTOMS.—Dengue attacks persons of all ages, from infancy to extreme old age. The characteristic symptoms are the presence of severe continuous arthritic and muscular pains; great debility and prostration; the occurrence of an initial scarlet and a terminal rubeoloid rash; fever, which is subject to remission and recurrence—the primary and secondary fevers; the possibility that convalescence may be tedious and painful, and complicated by the continuance of general cachexia, pain and swelling of joints, enlargement of glands, orchitis, weakness of eyes, deafness, visceral disease (such as diarrhoea or dysentery of a chronic and intractable character, and hepatic derangement), boils, carbuncles, and perhaps insanity. In the female, uterine hæmorrhage and miscarriage may occur.

The *invasion* of dengue is usually sudden, the patient feeling well up to the period of attack. The earliest symptom is severe pain in some joint, probably of a finger, which rapidly extends to all the other joints and bones: this pain during the progress of the disease often passes from one joint to the other by an unexplained process. Sometimes there is a period of preliminary malaise, of one or more days' duration, marked more especially by pains in the body, limbs, and joints—notably of the fingers and toes. The attack is often, however, strikingly sudden. In some epidemics certain phenomena are more prominently marked than others.

The fever is accompanied by redness of the face, which is puffy and swollen; sore-throat; congested conjunctivæ; and a general redness, extending over the whole body. The tongue is red at the tip and edges, and loaded with white fur, through which the red papillæ protrude. The pulse is rapid, ranging from 100 to 120, or even 140; respiration is hurried; and the temperature rises to 103° or even 105° F. These symptoms mark the occurrence of the initial fever and rash, and endure for a period varying from twenty-four to forty-eight hours. After this the rash disappears; the fever subsides; and the remission lasts for a period of two, three, or four days. A slight and brief recurrence of febrile symptoms then takes place, accompanied by the second or terminal rash. This differs in character from the first, resembling a rubeola, often showing itself first on the palms of the hands, and in some cases resulting in profuse furfuraceous desquamation of the cuticle, which, however, may sometimes be so slight as to be barely perceptible. These symptoms gradually subside, leaving the patient weak, exhausted, and often still tortured by swelling and pain of the joints, especially the smaller ones, which may continue in this state for weeks, making convalescence tedious and painful. In rare instances there may be repeated relapses, prolonging the suffering and protracting recovery.

VARIETIES.—The symptoms vary in different cases, as to the character of the rash, the temperature, and the muscular or osseous pains.

The *rash* not only varies considerably in colour, character, and duration, but it is sometimes almost absent; in other cases it is attended with so much

hyperæmia and action of the skin that excessive desquamation results. This hyperæmia also sometimes expresses itself by hæmorrhage from the mouth, nose, bowels, and uterus.

The *fever* is sometimes accompanied by delirium, or in children by convulsions; in the latter, indeed, these occasionally initiate the disease.

COURSE, DURATION, AND COMPLICATIONS.—The period of *incubation* of dengue is probably from one to six days.

In simple and uncomplicated cases the average period for the *duration* of the disease may be taken as about eight days; but it is frequently prolonged over weeks, and recovery is slow and painful; the constitution often being so much shattered that complete restoration to strength and vigour does not occur for months. It is rarely fatal. Recurrences of the disease during the same or subsequent epidemics are not infrequent.

Some of the *sequelæ* already mentioned may remain to torture the patient and retard his recovery. Albumen is occasionally present in the urine; but it is not, as in scarlatina, especially in the cases of children, a frequent or dangerous result of the disease.

DIAGNOSIS.—The distinction betwixt scarlatina and dengue is well marked; though during the outset there is considerable resemblance between the two diseases. There is a high temperature at first in both, but it is more quickly attained, and is transient in dengue; in scarlatina it endures for several days, while in dengue the fastigium gives a temperature of 103° F., or even up to 105° or 107°, and, this being attained, it rapidly declines. It is exceptional to find a temperature above 102° maintained in dengue. In scarlatina the period of decline extends over several days, and is marked by slight exacerbations in the evening. In dengue it occupies a few hours, and the temperature may even fall below the normal. The severe muscular and arthritic pains of dengue do not occur in scarlatina; and the pulse in the latter is much more rapid during the early stages than in the former. There is no secondary fever and rubeoloid eruption in scarlet fever; in dengue the throat symptoms are insignificant, and desquamation, when present, does not occur in large flakes as in scarlet fever. Dengue is strictly a disease of warm climates; scarlet fever of cold or temperate climates, rapidly dying out in the tropics.

PROGNOSIS.—An attack of dengue does not confer absolute protection from a recurrence of the disease, though it does so to a great extent.

TREATMENT.—The treatment is simple, and if judiciously directed mitigates the sufferings, and materially aids recovery.

Purgatives aggravate the suffering by the muscular movements necessarily induced, and should be avoided. Salines, such as the acetate of ammonium, or citrate of potassium, with spirit of nitrous ether, combined with aconite, are good during the pyrexia. In cases of very high temperature (105° to 107°) Dr. Charles suggests cold sponging as beneficial; he recommends it when 105° is reached. The danger to life of such a high temperature during the intense heat of the hot months in India is great; and it is then that cold sponging or the cold bath is indicated. Antipyrine, or phenacetin, relieves headache and lowers temperature, and may be used with discretion.

During convalescence tonics and a carefully

regulated nutritious diet are indicated. Depletive measures must be avoided. The tonics should be of the bitter vegetable kind, such as gentian and calumba; with these may be combined a small quantity of quinine, with a mineral acid; or, in some cases, the diluted phosphoric acid, combined with nux vomica or small doses of strychnine.

Alkalies, colchicum, and salicylates have little, if any, effect in relieving the pains of dengue.

As to wines, claret is probably the best, but others may be given.

For the irritation of the skin, which is sometimes very troublesome, the application of camphorated oil and the use of warm baths have been suggested.

As in so many other diseases, especially those that affect Europeans in tropical climates, complete restoration to health is promoted by change of air; and in severe cases return to the patient's native climate for a season is desirable.

JOSEPH FAYRER.

PATRICK MANSON.

DENTITION, Disorders of.—SYNON.: Teething; Fr. *Troubles de la Dentition*; Ger. *Zahnung*.

GENERAL REMARKS.—The period of cutting the teeth has been always recognised as a critical time, during which the health of young children is especially liable to become disturbed; and it has been a common practice among mothers to attribute every illness occurring in early life, from the irritation of scabies to the distortions of rickets, to the same baneful influence. The evolution of the milk-teeth is no doubt attended with some irritation, especially as at this period the follicular apparatus of the intestines is undergoing considerable development; and we know that, on account of the impressibility of the nervous system in young children, any irritation is apt to be followed by general disturbance. It is no doubt also the case that local functional derangements are frequent at this period, but it is often unfair to attribute these *directly* to the irritation of an advancing tooth. One of the most common direct results of teething is pyrexia, which may be intense; and a feverish child is particularly susceptible to impressions of cold, and to the irritation of unsuitable food. Catarrhal attacks coming on at this time need not be therefore the immediate result of the condition of the gum. It is at least admissible to attribute them to the ordinary causes of such derangements, acting upon a body rendered for the time particularly susceptible to injurious influences. This view is supported by the fact that diarrhoea, which is a very common complication of dentition, is especially frequent during the warmer months, when the temperature is apt to undergo rapid variations, while the dress of the child remains unchanged; and is far less common during the winter, when the temperature is more uniform, and the child is more carefully guarded against the cold.

As a rule, the first milk-tooth appears in the seventh month after birth; but dentition may begin at an earlier period. It is not rare for an infant to cut a tooth at the age of four months; and occasionally at the time of birth one tooth is found to be already through the gum. In cases where the ordinary time of teething is anticipated, a pause generally ensues after the appearance of one or two teeth, and further dentition is delayed until the usual age. Thus syphilitic children and children prone to tuberculosis cut their teeth early, while in rickety children the teeth are very slow to appear.

In the majority of cases the teeth pierce the gum in the following order: lower central incisors, upper central incisors, upper lateral incisors, lower lateral incisors, first molars, canines, back molars. A child of twelve months old should have eight teeth and be cutting his first molars; and the whole number (twenty) should be through the gums soon after the end of the second year. The order given above is frequently departed from. The incisors, in particular, are often cut irregularly, and it is not rare for the molars also to appear out of their proper order. A double tooth will often be seen to precede the lateral incisors, and may indeed in exceptional cases be the first tooth to appear through the gum. The canines seldom or never precede the first molars.

Variations may also be met with in the number of teeth cut in the first dentition, for the full number of twenty may not be reached. Thus, the lower lateral incisors may be absent, or the whole of the canines be missing in cases where the other teeth have been cut with regularity and dentition has apparently come to an end. On the other hand, instead of too few teeth there may be too many: five perfect incisors in the lower jaw is a not uncommon phenomenon.

Some infants suffer more than others from the cutting of a tooth, and it is not always in cases where the eruption of the teeth has been delayed that dentition, when it occurs, is attended with special discomfort. On the contrary, in severe rickets, where the delay is great, the teeth are often cut with singular ease.

The second dentition begins at about the sixth year. The permanent teeth are cut with more regularity than is the case with the earlier crop. The first to be seen are usually four molars, which appear behind the last of the temporary teeth. Next come the central incisors at about the eighth year; the lateral incisors at about the ninth; the first and second bicusps, in the place of the temporary molars, at the tenth and eleventh; the canines between the twelfth and thirteenth; and the second molars at about the time of puberty. The last four permanent molars are cut later.

SYMPTOMS.—The phenomena which may be looked upon as natural to the first dentition are salivation; swelling of the gum, which becomes more and more tense, hot, and painful; slight general pyrexia, with flushing of one or both cheeks; irritability of temper; and some degree of restlessness at night. These all subside when the point of the tooth appears through the gum. The complications not necessarily attendant upon the process are high fever; inflammation of the mouth and aphthæ; vomiting (gastric catarrh); diarrhoea (intestinal catarrh); cough (pulmonary catarrh); otitis, with purulent discharge from the auditory meatus; various eruptions of the skin, with, sometimes, swelling, and even suppuration of lymphatic glands; and certain troubles of the nervous system, such as convulsions, squinting, &c.

The peculiarity of the pyrexia of dentition is its irregular character. It is often higher in the morning than at night, and varies in intensity in a remarkable manner from day to day. A temperature of 104° F. at 8 A.M. is not at all uncommon in a teething infant; indeed such an amount of fever in the morning should lead us at once to inspect the mouth, as few diseases are marked by so much pyrexia at that hour of the day. We must not, however, in every case where the gums are swollen

and tense, rush to the conclusion that they are the sole cause of the symptoms, for the most serious cerebral disease may co-exist with the eruption of a tooth. Thus, headache, delirium, vertigo, startings, twitchings, and convulsive attacks, may merely indicate functional disturbance of the brain or cord, such as is common to many disorders, and these phenomena are not necessarily symptomatic of cerebral disease; but if the bowels become obstinately confined, the pulse slow and irregular, and the respiration unequal and sighing; and if in addition there be photophobia, with sullenness and tendency to drowsiness, we may conclude that something more than mere functional derangement is present, and that there is every reason to suspect the onset of tubercular meningitis.

In the case of the second dentition there may also be digestive derangement. During this period the child is subject to gastro-intestinal catarrh, and is often irritable in the day and restless at night. Night terrors are common, and he may wake up from his sleep screaming with fright at some horrible dream. Moreover, in addition to the discomfort arising from fermentation of food and acidity, considerable loss of flesh may be the consequence of digestive derangement if long continued.

TREATMENT.—The treatment of the complications which occur during dentition must be conducted upon ordinary principles. Aphthæ of the mouth are readily cured by the administration of rhubarb and soda, and the application to the mouth of a solution of chlorate of potassium or borax (ten grains to the ounce) in water, sweetened with glycerin. Perfect cleanliness is necessary, and the child's mouth should be washed out each time after taking food with a piece of soft rag dipped in warm water.

Vomiting is best checked by clearing out the stomach with an emetic of ipecacuanha wine, giving a teaspoonful every ten minutes until sickness is produced. Afterwards, a few grains of bicarbonate of sodium may be given with one drop of liquor arsenicalis in a teaspoonful of water three times a day. Diarrhoea should be treated on the same principle: first a dose of castor oil to remove irritating products from the bowels; then a mixture containing chalk and catechu, or oxide of zinc (one grain to the dose). If afterwards the motions continue large, pasty-looking, and offensive, and are passed too frequently in the day, one drop of tincture of opium may be added to the mixture, as there is usually in such cases too rapid peristaltic action of the intestines. In the case of either of these derangements (vomiting or diarrhoea), it is of great importance to cover the abdomen with a broad flannel bandage and to attend to the warmth of the feet and legs. The diet also should be temporarily modified, reducing the quantity of farinaceous matter that is being taken, on account of the tendency to acid fermentation of food which is set up by such a condition of the alimentary canal.

A catarrhal condition of the bowels should be cured as quickly as possible, especially during dentition, for at this time the susceptibility to chills is heightened, and the danger of severe diarrhoea being set up correspondingly great. In some cases of teething, where the lungs as well as the bowels are the seat of catarrh, and there is a risk of bronchitis, Troussseau recommends that the intestinal derangement should not be suddenly put an end to; but even in these cases a dose of castor oil may be safely

given, to remove irritating matters from the canal, for any irritation of the bowels is apt rather to increase than to diminish pulmonary mischief.

Lancing the gums is a practice which should not be pursued indiscriminately. If, however, there be much inflammation and swelling, and the child seem to be in pain, relief may be obtained by lancing, but in this case the object is merely to relieve tension. In the case of convulsions more benefit is to be gained by the use of bromide of potassium, warm baths, cold applications to the head, and the gentle action of a mild alterative aperient.

In the second dentition, care should be taken to keep the child's feet warm and his body properly protected from cold. Attacks of indigestion and diarrhoea must be treated by appropriate remedies, and attention should be paid to the diet, limiting the quantity of starches and sweets allowed, on account of the tendency to fermentation and acidity. Night-terrors, often merely attacks of nightmare, can frequently be cured by a mild aperient to clear away undigested food from the bowels, and by a careful diet. Sweets, starches, and fruit should be forbidden; and the child must be fed on meat, fish, poultry, milk, butter, cooked green vegetables, eggs, stale bread, and any kind of non-fermentable food suited to his age.

EUSTACE SMITH.

DENVER, in Colorado.—Situated 5,200 feet above the sea, on a plain backed by the Rocky Mountains. A dry, cold, clear, and bracing climate. Diathermancy considerable. Temperature: mean daily range, 24°, showing great variation; mean temperature, 49°. Rainfall 15 inches. A high-altitude station for phthisical patients. Railway communication and accommodation excellent. For invalids the suburbs of Denver are preferable to the city itself.

DEODORANTS, (*de*, from; and *odoro*, I cause to smell).—**DEFINITION.**—Deodorants are bodies capable of destroying or removing noxious or offensive odours.

DESCRIPTION.—Odorous bodies may be either offensive or agreeable, noxious or the reverse, and although deodorants are only employed for removing offensive odours, yet a true deodorant will attack either class effectually, and the value of any one depends upon its power of absorbing or changing the nature of the odour, and not simply upon its ability to cover another odour by its own. This should be borne in mind when choosing one of the volatile deodorants. And it should also be remembered that of those which are desirable from a purely chemical point of view, some, such as chlorinated lime, have sickly odours which are objectionable to many people and especially to invalids, while others equally or perhaps more efficacious, such as terebene, do not exhibit the foregoing objectionable character. 'Natural selection' therefore tends towards the adoption of the latter class.

True deodorants may be divided into two classes: (1) gaseous or volatile; and (2) solid (or liquid). The former class aims at destroying or absorbing the offensive odour, the latter at destroying the substance which generates it.

1. *Gaseous or Volatile* deodorants include chlorine, bromine, sulphur dioxide, ozone, peroxide of hydrogen, formaldehyde, essential oils, terebene, camphor, turpentine, naphthalene, and the vapours of phenol and allied products.

The chemical action by which noxious odours are destroyed is principally one of oxidation, and this class of deodorants are generally oxidising agents. The characteristic power of deodorisation possessed by essential oils, emanating either from the flowers themselves, or through the use of the oil extracted from the plant, is its capability of combining with atmospheric oxygen and moisture to produce peroxide of hydrogen, which, while used almost exclusively for its deodorant properties, possesses equal, if not higher, germicidal powers.

The rate of deodorisation depends upon the chemical activity of the deodorant, and the more rapidly oxidation can be made to proceed the greater is the efficiency of the substance employed. The deodorising powers of the various hydrocarbons obtained from the turpentine and other sources (such as the terebene and allied preparations) depend to a very great extent upon the formation and liberation of peroxide of hydrogen. The enormous power of the active oxygen contained in the atmosphere as ozone is one of the great factors in the natural destruction of noxious odours.

2. *Solid or Liquid* deodorants include charcoal, dry earth, lime, chlorinated lime, dry sawdust, and solutions of sulphate of iron, chloride of zinc, nitrate of lead, alum, and permanganate of potassium.

Solid (or liquid) Non-volatile Deodorants.—The chief application of these is in their uses for the deodorisation of sewage, especially as regards their effects upon the noxious gases given off during the decomposition of both animal and vegetable matter, the liberation of the ammoniacal elements in their free state from their sulphurated compounds, or the retention of them as innocuous chemical compounds. The unique power possessed by charcoal is exhibited in its capabilities for the oxidation and destruction of noxious gases such as sulphuretted hydrogen. Peat-charcoal and seaweed-charcoal have both been recommended for deodorising manure on a large scale. Permanganates prevent putrefaction for a short time and remove the odour of putrefying sewage, but require to be used in large quantities. Dry earth, lime-salts, oxide of iron, and solutions of various chemicals all act as precipitants, the noxious elements being destroyed by the liberation of the nascent oxidising agents and the subsequent formation of innocuous products.

FRANK A. ROGERS.

DEPILATORIES (*depilis*, without hair).

DEFINITION.—Depilatories are measures used for the destruction of superfluous hairs.

Hypertrichosis.—The patients who present themselves for treatment by depilation are, of course, almost always women. In the immense majority of cases the disfiguring hairs are on the upper lip, chin, neck, nose, or in front of the ears, the annoyance and mental depression caused by them being often so extreme as to justify any procedure likely to give permanent relief, provided it is rationally employed and its limitations are properly understood. It must be carefully borne in mind, however, that in a certain number of cases hypertrichosis is only temporary, dependent perhaps on pregnancy or menstrual disorders, or associated with acne and other sebaceous affections, or the result of their treatment by sulphur and other irritants; and that in these circumstances it may disappear spontaneously, or as the result of treatment directed to

these associated conditions. As a rule, the disfigurement is first noticed at about the age of eighteen, and is progressive till twenty-five or thirty, after which it remains permanent till the menopause, when it frequently becomes aggravated.

ENUMERATION.—Various caustic applications are occasionally used as depilatories. Erasmus Wilson recommended the following:—Quicklime (three parts), sulphide of sodium or of barium (one part), diluted with starch (four parts). The powder, mixed with water to the consistence of a thin paste, is laid on the affected part by means of an ivory paper knife, and allowed to remain for from five to fifteen minutes. It must then be carefully scraped off, when the hair, shrivelled and burnt, comes away with it. The skin must then be washed with water, dried, and anointed with cold-cream.

Although the immediate result of this method is sometimes satisfactory, a troublesome degree of dermatitis is generally occasioned by it. The hairs, too, always return, their roots not being destroyed, but only stimulated, by the treatment; and the subsequent growth is invariably stronger, darker, and more disfiguring than the original, while considerable scarring too frequently results.

Many women keep the disfigurement due to hypertrichosis in abeyance by habitually shaving or extracting the hairs with forceps. There are many obvious objections to these procedures, but in some cases, where no other treatment is applicable, they answer fairly well; as their result, however, the growth becomes darker, stronger, and more bristly. In a certain number of cases good cosmetic results are obtained by bleaching the hair with a saturated solution of peroxide of hydrogen.

Recently, 'cures' have been reported as due to exposure to Röntgen rays; but doubts must be cast upon their permanence, while it is certain that a current sufficiently strong to destroy or even damage the hair-bulbs must be fraught with danger of producing the peculiarly intractable form of ulcerative dermatitis, so often resulting from exposure to the rays.

Depilation by Electrolysis.—The only efficacious and permanent method of destroying superfluous hair is by electrolysis.

INDICATIONS.—Generally speaking, the cases in which electrolysis is most applicable are those where—(1) the growth is limited in extent, and stationary, not progressive; (2) the hairs are strong and dark; (3) the amount of downy undergrowth is slight; (4) the patient stands pain well, is sufficiently intelligent to understand the bearings and restrictions of the process, and is in a position to submit to its necessarily protracted duration. There is little doubt that downy hairs in the regions operated upon sometimes become dark and strong with undue rapidity as the result of electrolysis; but it is also more than probable that, independently of it, these hairs would ultimately, although more slowly, become sources of disfigurement. Cases in which the growth is most abundant upon, or confined to, the upper lip are, as a rule, unsatisfactory, owing to the large number of hair-follicles there present, the extreme sensitiveness of the part, the small number of hairs which can be extracted at a sitting (seldom exceeding twelve to fifteen), and the amount of inflammatory oedema set up in the lax tissue.

APPARATUS.—The necessary apparatus for depilation by electrolysis comprises—(1) a reliable

constant-current battery of from ten to twenty cells, the nature of the elements being a matter of indifference; (2) an absolute dead-beat galvanometer, graduated in milliamperes, and preferably incorporated with the battery; (3) long, light, pliable rheophores; (4) a suitable needle-holder, connected with the negative pole of a battery, many forms of which are now made (those which have the current-interrupting apparatus in the holder are handy and convenient); (5) a fine steel sewing-needle—Abel Morrall's 12's, the only disadvantage of which is their brittleness; (6) a carbon or brass cylinder covered with chamois-leather, constituting the positive electrode, and connected with the positive pole of the battery. A steady hand and good vision are indispensable on the part of the operator, all suggested arrangements of magnifying-glasses being unsatisfactory. The best of such is a watchmaker's glass mounted on a spectacle-frame.

METHOD.—The patient being placed in a good light, and preferably in the recumbent posture, seizes the positive electrode—previously well soaked in hot water—firmly in one hand; the needle is introduced into the hair-follicle for a distance of $\frac{1}{16}$ to $\frac{1}{8}$ of an inch or more, the follicle, being, as it were, catheterised. The experienced touch is even able to distinguish when the bottom of the follicle is reached, by a slight increase in the resistance encountered. The current is closed by the operator pressing upon the interruptor, and a sharp pain is experienced at the moment of closure, which persists, but in a modified form, throughout the destruction of the hair. The hair may then be seized with forceps, held in the left hand; and, when destroyed, it comes away without appreciable traction. The current is then broken, and the needle is withdrawn. While the current passes, a little froth, evidence of the electrolytic action, wells up by the side of the hair, and an urticarial wheal forms. This soon subsides, leaving a pink hardish papule, which lasts a few days; while occasionally a minute scab forms, which may take about a week to separate. In either case a tiny white depressed scar is left, but it is usually practically imperceptible after a short time. Occasionally, however—especially in persons of dark complexion—the scars are rather deeply pigmented, and the resulting disfigurement disappears but slowly. A current of five milliamperes employed for half a minute is sufficient to destroy the strongest hairs, if the follicle is accurately struck; and from six to ten cells are usually sufficient to generate this current strength. Weaker currents are effectual in destroying finer hairs, but it is strongly inadvisable to attempt to destroy lanugo. The amount of pain experienced varies within very wide limits; it is seldom so intolerable as to contra-indicate the operation. In some cases ointments containing 10–20 per cent. of cocaine, rubbed in for ten minutes beforehand, diminish it sensibly. The number of hairs extracted at a sitting ought never to exceed fifty, and an interval of several days ought always to be observed between the destruction of hairs close together. A few recurrences necessarily take place, their number being in inverse ratio to the expertness with which the operation has been performed. They are easily recognisable, and are generally most abundant beneath the chin and on the neck, where it is difficult to determine the precise direction of the hair-follicles.

It is impossible here to dwell upon all the details

of the procedure, the success of which mainly depends upon skill, patience, and experience.

J. J. PRINGLE.

DEPLETION (*depleo*, I empty out).—SYNON.: Fr. *Dépétion*; Ger. *Entleerung*.

DEFINITION.—By the term 'depletion' is understood: (a) the unloading, or rendering less full, of that which is over-burdened or over-full, for example, portions of the turgid vascular system—as the portal vessels; or (b) excessive evacuation, causing exhaustion—as in choleraic or other severe diarrhoea.

USES.—Depletion, local or general, as a therapeutic agent, may be practised in a variety of affections, such as cerebral congestion, venous turgescence, engorgement of the portal system, pulmonary congestion, renal ischæmia, aneurysm, or general plethora.

The agencies whereby depletion may be produced are blood-letting, general or local; purging; vomiting; sweating; and abstinence from food and drink.

Nothing depletes so readily, efficiently, and safely as hydragogue purgatives which deplete by removing serum from the blood-vessels without the loss of red corpuscles. Depletion by such measures is often of service. See BLOOD, Abstraction of; DIAPHORETICS; BATHS; and VENESECTION.

JOHN HAROLD.

DEPOSITS.—See CONCRETIONS.

DERBYSHIRE NECK.—A synonym for goitre, which is thus called from the prevalence of the disease in that county. See GOITRE.

DERIVATIVES (*derivo*, I drain).

DEFINITION.—Medical appliances or remedies believed to control inflammation, in one part of the body, by producing a flow of blood or lymph to another part.

ENUMERATION.—Derivatives include Local Bleeding, Cupping, Leeches, Blisters, Sinapisms, and Setons.

ACTION.—The name 'derivative' was applied in ancient times under the belief that diseases were caused by morbid humours, which might be drawn away from the part which they were affecting. It is now used chiefly to signify the diminution of blood in an inflamed part, by increased circulation in some other vascular district, either adjoining or remote from it.

T. LAUDER BRUNTON.

DERMATITIS (*δέμα*, the skin).—Inflammation of the skin: a term applicable to every variety of inflammation of the integument, but especially to an acute inflammation attended with exfoliation of the cuticle and copious desquamation—for example, *dermatitis exfoliativa*, the *pitiriasis rubra* of Devergie.

DERMATITIS HERPETIFORMIS.—See HYDROA.

DERMATITIS REPENS.—This name has been used to describe a spreading dermatitis affecting the extremities and usually following injury. Radcliffe Crocker is inclined to regard the condition as a trophoneurosis.

The dermatitis is characterised by the exudation of serum which undermines the epidermis and may

cause bullæ in the first instance, but finally brings about complete exfoliation of the upper layers of the epidermis. The diseased surface so produced has a defined margin with undermined epithelium, and shows a marked tendency to spread during the acute stage of the disease. The denuded surface continues to ooze serum profusely. As the affected area heals, it presents a thin shining appearance very different from that of a healed eczema rubrum. The disease may last for many months or even years.

TREATMENT.—In spite of the evidence of nerve-injuries or inflammation, as the cause of the disease, the lesions become readily infected by pyogenic organisms, and the treatment consists in using antiseptics in the first instance, followed by protective dressings such as glycerine-gelatine pastes, oxide of zinc ointment, &c. JAMES GALLOWAY.

DERMATITIS VENENATA.—This term embraces all inflammations of the skin set up by external irritants.

Strong irritants, such as cantharides, tartar emetic, or croton oil, will excite acute severe inflammation in any part of the skin to which they may be applied. *Mild irritants* vary in their effects according to the susceptibility of the individual, the virulence or concentration of the poison, and the length of exposure to its influence. Thus in some persons their effects are severe, in others slight, in others unnoticed. Arnica and Rhus toxicodendron are examples of this diversity of effect.

The inflammatory lesions thus produced are wheals, erythema, papules, vesicles, bullæ, pustules, or gangrene.

Eczematous subjects are peculiarly sensitive to all irritants, and in them mild irritants may not only excite a severe local dermatitis, but even a widespread or general eczema, the inflammation starting in foci far away from the original irritant. Some persons who are not unduly susceptible to irritants generally are especially affected by certain kinds, and if circumstances expose them to these frequently their special susceptibility increases progressively until an intense inflammation may be excited by an incredibly weak and transitory application. This increased susceptibility is sometimes seen in surgeons to whose skin iodoform acts as an irritant. On the other hand, the skin of some persons may become habituated to what at first acted as an irritant. Irritants may be applied to the skin intentionally by malingerers (*see* FEIGNED DISEASES), criminals, or hysterical persons; or they may be used medicinally for the purpose of counter-irritation (*see* COUNTER-IRRITANTS), or in ignorance of the qualities of the drug (as in the popular use of arnica) or of the peculiar idiosyncrasy of the patient. Similar irritation may result from the contact of under-clothing coloured with aniline dyes contaminated with arsenic; or may be the consequence of the occupation, as in the use of bichromate of potassium by French-polishers, or the handling of Rhus toxicodendron by gardeners. Chrysarobin, so often prescribed for psoriasis and ringworm, produces a peculiar and unsightly erythema usually extending beyond the site of application. It should, therefore, never be prescribed for the face or any neighbouring part.

Exposure to Röntgen rays, if too prolonged, too frequently repeated, or carried out with the patient too near the glass tube, may result in a

severe dermatitis with complete destruction of the whole skin, sometimes with an obstinately adherent gangrenous slough; at others with a molecular ulceration; in either case leaving an ulcer which may take many months to heal, and give rise to an extensive scar, which, as in two cases seen by the writer, may contain a dense network of dilated vessels.

The following list of irritants and their usual effects may be useful:—

<i>Drugs</i>	
Croton oil and tartar emetic	} Pustules
Cantharides, mustard, and turpentine	
Chrysarobin	} Aspecial erythema, sometimes vesication
Mercurial inunction	
Iodoform	} Pityriasis rubra or eczema Acute vesicular eruption like a severe eczema

<i>Plants</i>	
Rhus venenata and toxicodendron	} All grades of inflammation from slight to very severe vesicular or pustular eruptions, erythema, or wheals.
Primula obconica	
Urtica urens	

<i>Animal</i>	
Various insect-bites	} Special lesions not merely due to scratching
Acarus scabiei	
Contact with the 'woolly bear' caterpillar or the jelly-fish	} Generally an erythema

<i>Physical agencies</i>	
Direct solar rays	} Vesication or ulceration
Röntgen rays	

DIAGNOSIS.—The diagnosis of dermatitis venenata is important in the case of malingerers, or where the sufferer is frequently exposed to the irritant without knowing that it is the cause of his trouble, as in the case of animal parasites, irritant plants, or chemicals. Speaking generally, the more intense and the more localised the inflammation, the more probable that it is of local origin. The lesions, moreover, are irregularly distributed over the affected area; the position of the rash also assists in the diagnosis. In occupation-eruptions the hands are the chief parts affected, and the palms more than the backs in most cases. In self-inflicted eruptions the lesions are always in accessible positions on the front of the body or limbs, and usually on the left side in right-handed people. Not infrequently the lesions are angular in outline or show the shape of the instrument, e.g. a brush, used to apply the irritant; or there may be particles, or discolouration, or a streak where the fluid has run down, to indicate the nature of the irritant. Further, such a rash seldom conforms exactly to what may be called a natural eruption. In other cases, the sudden onset while in good health and the repetition of the attacks under similar circumstances should lead eventually, if not at once, to a suspicion of the origin, which can then be soon verified (*see* FEIGNED DISEASES). When the inflammation is of slight degree, or when an actual eczema is produced, the diagnosis may be impossible, judging by the eruption alone, but careful inquiry into its history and mode of development will sometimes put the inquirer on the right track. The progress of the eruption while under observation is often a

valuable aid, for a dermatitis of direct irritant origin usually gets well as soon as the cause is removed, unless an actual eczema has developed.

TREATMENT.—Local treatment is alone required, and the actual applications should be similar to those used in acute erythema or eczema.

H. RADCLIFFE CROCKER.

DESQUAMATION (*de*, signifying separation; and *squama*, a scale).—The process of separation or shedding of the epithelium of any surface. It is of most importance in connection with the skin in scarlatina, where the epidermis usually desquamates extensively.

DESQUAMATIVE NEPHRITIS.—A synonym for certain forms of renal disease, applied on account of their being characterised by shedding of the epithelium lining the tubules. See KIDNEY, Diseases of.

DETERGENTS (*detergo*, I cleanse).

DEFINITION.—Substances which cleanse the skin.

ENUMERATION.—The principal detergents are—Water, Soap, Alkalis, Ox-gall, Milk, Vinegar, Alcohol, Charcoal, Sand, Oatmeal, Sawdust, Pumice-stone, Oil, and Borax.

USES.—Detergents are used to remove either extraneous dirt adherent to the skin, or epidermal scales which have accumulated upon it and interfere with its function. The chief detergent is warm water, but its action is greatly aided by such substances as soap, alkalis, borax, or vinegar, which act chemically in the removal of dirt or epidermis; or by such substances as oatmeal, sawdust, charcoal, pumice-stone, and sand, which act mechanically. Oil, or alcohol in the form of *eau de Cologne*, removes the resinous deposit left on the skin by plasters. Where the skin is tender, as in the case of the scalp, and where at the same time the detergent employed cannot very readily be removed, borax with elderflower water may be found preferable to the more irritating soaps as a means of removing scurf.

T. LAUDER BRUNTON.

DETERMINATION OF BLOOD.—Increased flow of blood to a part or organ: synonym with *active hyperæmia* or *active congestion*. See CIRCULATION, Disorders of.

DEVELOPMENT, Arrest of.—See MONSTROSITIES.

DEVELOPMENTAL INSANITY.—See INSANITY.

DEVONSHIRE COLIC.—A synonym for lead-colic, which has arisen from the frequency of lead-poisoning in that county, supposed to be due to the contamination of cider with lead. See LEAD, Poisoning by.

DIABETES INSIPIDUS (*διαβαίνω*, I walk with the legs apart; *διαβήτης*, a thing with outstretched legs, a pair of compasses, a siphon—whence the idea of free flow is derived; *insipidus*, tasteless).—**SYNON.**: Polyuria; Fr. *Diabète insipide*; *Diabète non sucré*; Ger. *Polyuria*. See POLYURIA.

DIABETES MELLITUS (*διαβαίνω*, I go through; and *μέλι*, honey).—Fr. *Diabète*; Ger. *Zuckarnruhr*.

DEFINITION.—Diabetes mellitus is a disease

characterised by the presence of grape-sugar in the urine in quantities readily detectible by clinical tests, and in most cases by polyuria and wasting.

The normal urine contains, according to many authors, a very minute quantity of grape-sugar (glucose or dextrose); according to Breul, between 0·04–0·2 per cent. If it contain more than this the condition is termed glycosuria. The terms ‘glycosuria’ and ‘diabetes mellitus,’ as used clinically, are not synonymous. The term ‘glycosuria’ is generally restricted to the temporary appearance of sugar in the urine, such as may occur after the ingestion of a large quantity of sugar (*alimentary glycosuria*), after fever, after injuries, or as a result of the action of toxic bodies (e.g. carbon monoxide), though many also apply the term to milder forms of diabetes mellitus.

PATHOLOGY.—The normal blood contains a fairly constant quantity of grape-sugar, 0·09–0·1 per cent. This proportion is not decreased by starvation, nor increased by the ingestion of carbohydrates; but should the quantity of sugar in the blood increase, an appreciable amount of grape-sugar will appear in the urine.

The sugar is derived to a great extent from carbohydrates in the food, to a smaller extent from the splitting up of albuminous bodies and possibly also from fat. The glucose and other sugars, thus formed from food in the digestive tract, pass through the portal vein into the liver; hence the portal vein during digestion may contain as much as 0·4 per cent. of sugar; while a small quantity of sugar may pass into the chylous vessels and thus reach the blood through the thoracic duct.

It is generally believed that in the liver the sugar is converted into glycogen, and by most observers that the glycogen in the liver is changed again into sugar and passed gradually into the blood; but Pavy and others maintain that the liver absorbs the sugar and prevents it from passing into the circulation. It must further be remembered that the muscles also contain glycogen, and that they contain more when large quantities of carbohydrates are taken as food and when the muscle rests, but less during starvation and during increased muscular exercise. It is generally held that the liver and muscles by converting the sugar into glycogen act as storehouses, and that the glycogen remains stored up to be again oxidised; fat may also be formed from the sugar; this also becomes oxidised later on.

EXPERIMENTAL PATHOLOGY.—If sugar appears in the urine there must be either an over-production or a diminished consumption of sugar. 1. Glycosuria may occur when excessive quantities of saccharine food are taken, although in healthy individuals very large quantities of grape-sugar (about 200 grammes) may be taken before sugar appears in the urine. If, however, the stomach be empty, a much smaller quantity suffices, and the same may be said if cane-sugar or milk-sugar be substituted for the glucose, while starch by itself, even in very large quantities, will not give rise to glycosuria. This form of glycosuria is named according to Naunyn *alimentary glycosuria e saccharo*. Much smaller quantities of grape-sugar suffice to give rise to glycosuria if certain pathological conditions be present, e.g. cirrhosis of the liver, various neuroses, injuries to the nervous system, mental diseases.

2. *Phloridzin-Diabetes.*—Phloridzin is obtained from the bark of the root of apple and cherry trees.

If this substance be given to dogs, either by the mouth or subcutaneously, grape-sugar appears in the urine, even when the animal has been kept on a nitrogenous diet or has been starved. The glycosuria may persist for some days, though the blood does not show an increased percentage of sugar. Similar results have been obtained, though not invariably, in man. This form of glycosuria is looked upon by some as dependent on the kidneys, phloridzin being probably split up there. Klemperer and others have described a form of diabetes in man, in which they believe the glycosuria is due to some changes in the kidney: in these cases there was no increase of sugar in the blood.

3. *Glycosuria of nervous origin.*—According to the well-known experiment of Claude Bernard, glycosuria is produced by injury to the fourth ventricle, the blood containing an increased amount of sugar. Experimental injuries to other parts of the nervous system, such as section of the spinal cord, extirpation of the first cervical ganglion, section of the sciatic nerve, may also produce temporary glycosuria. The glycosuria in these cases is most likely due to vaso-motor disturbance in the liver, thereby affecting the glycogen-formation in the liver. In man diabetes may occur in various affections of the nervous system (cerebral hæmorrhage, tumours of the brain and other parts of the nervous system, multiple sclerosis, mental diseases, shock, traumatic neuroses, epilepsy).

4. *Pancreatic Diabetes.*—Complete removal of the pancreas in dogs produces all the symptoms of severe diabetes, with increased thirst, rapid emaciation, and death. The sugar in the blood is very much increased, the glycogen in the liver rapidly diminishes, and the glycosuria remains even if only nitrogenous food be given. If the pancreas is only partially extirpated, or if a portion of the extirpated pancreas is transplanted under the skin, no diabetes occurs.

Various explanations of this form of diabetes have been given. The most plausible theory perhaps is the one which assumes that the pancreas has an internal secretion which exerts an inhibitory effect on sugar-formation, either acting directly on the liver-cells or indirectly through the nervous system. The pancreas has been found affected in various ways in persons who have died of diabetes, so that the experimental investigations on pancreatic diabetes are in some degree supported by the observations on diabetic patients. In only a few cases of acute pancreatitis or necrosis of the pancreas has sugar been found in the urine. In some of the cases of diabetes, however, a complete atrophy of the pancreas has occasionally been found; on the other hand, in many cases of cancer and other affections of the pancreas diabetes does not occur. See PANCREAS, Diseases of.

ÆTIOLOGY.—Diabetes may occur at any age from six months upwards; its frequency increases with increasing age. Men appear more frequently affected than women; it is decidedly more common among Jews; and appears to have become more prevalent generally within the last twenty years. The question of heredity has been frequently discussed, and there can be no doubt that it not infrequently happens that several members of the same family are affected with diabetes, and occasionally one parent and one or more children; more frequently, however, there is a family history of gout or renal disease, or a neuropathic predisposition.

Schmitz and others record several cases of diabetes occurring in both husband and wife, but there is so far no evidence of the infectious nature of the disease. Exposure to cold and wet is also an assigned cause; occasionally diabetes has been known to follow mental emotions, sexual excesses, external injury, and acute infectious diseases, especially epidemic influenza and malaria. Diabetes occurring in persons who have had syphilis does not yield to antisiphilitic treatment. Of greater importance is the relation of diabetes to:

(1) *Gout.*—Persons of advanced age suffering from gout are prone to have glycosuria; often both affections occur together.

(2) *Obesity.*—In very stout people, especially women at the climacteric period, diabetes of the milder form is very common (*Diabète gras, lipogenic diabetes*). The diet and mode of life are said to be the cause of the association of these two affections, yet it is not known that a diet rich in carbohydrates suffices to produce permanent glycosuria.

(3) *Arterio-Sclerosis.*—Atheroma of the arteries is often found in mild cases of diabetes. The cause of this connection is not known: atheroma of the pancreatic arteries may possibly account for it; often, however, in addition to the arterial disease these patients suffer from cirrhosis of the liver, gout, and nervous affections.

(4) *Diabetes and the puerperal state.*—It is a well-established fact that during the puerperium milk-sugar may appear in the urine, due to an excess of milk in the breast which causes the absorption of milk-sugar into the blood; this is noticed when women suddenly stop suckling. Apart from this, true diabetes has occasionally been noticed soon after confinement.

SYMPTOMS.—The symptoms vary according to the severity of the affection, its causation, the age of the patient, and many other factors. The most prominent sign is of course the presence of sugar in the urine, and dependent on this, and varying with the amount of sugar, are increased thirst, increased appetite, increased diuresis, dryness of the skin, emaciation, and loss of muscular strength. Many other symptoms are noticed, some of which must be looked upon as complications.

The *onset* is in most cases insidious; in some, however, especially the so-called pancreatic form, it may be acute: increased thirst, increased diuresis, and rapid emaciation being the principal features.

The *general aspect* of the patient varies according as we have a mild or a severe form. In the milder form the patient is often stout and of middle age; in the severe form he is young, has lost flesh, and has a shrunken face and a nervous anxious expression; his skin is dry, pale, and in some rare cases may have a bronzed appearance (*diabète bronzé, hæmochromatosis of Osler*).

The *Urine.*—The *quantity* of urine passed is in most cases increased, and may be the first sign of the disease. In slight cases the quantity passed varies from 100 to 200 ounces in twenty-four hours, in severe cases it may reach 400 ounces or more; in stout people there may be no increase whatever, and sometimes in very severe cases, as the end approaches, the amount of urine may also be small. It is reduced by rigid diet, by certain inter-current affections, such as chorea, phthisis, gout, disease of the heart or of the kidneys, and acute febrile diseases; and generally diminishes with the onset of diabetic coma.

The colour of the urine varies with the quantity passed. In the severe cases it is pale and clear, and becomes opalescent on standing, owing to the growth of the yeast-fungus.

The odour has, especially in the severe forms and just before and during the stage coma, a peculiar aromatic character, somewhat like the odour of chloroform: this is due to acetone, and is a sign of grave prognosis.

The specific gravity is almost always increased: the higher the specific gravity the more severe the case. It ranges between 1030 and 1040, but may reach or even exceed 1060. In some cases, especially when complicated with Bright's disease, the specific gravity may be low; cases have been recorded in which the specific gravity was below 1008. The reaction is acid.

The characteristic feature of the urine is the presence of grape-sugar. The best clinical tests for the detection of this are:

1. *Fehling's Test*.—In this the reducing action of the grape-sugar on metallic salts in alkaline solutions is made use of.

The official formula of the British Pharmacopœia may be used for all qualitative analysis (Solution 1, copper sulphate (crystals), 34.64 grammes; sulphuric acid, 0.5 cc.; distilled water to make 500 cc. Solution 2, sodium potassium tartrate, 176 grammes; sodium hydroxide, 77 grammes; distilled water to make 500 cc. For use mix equal volumes of each solution). The solutions should be mixed when required. The fluid thus formed is of an intense blue colour, clear and bright. When the test-fluid is to be used, a small quantity of it should first be raised to the boiling-point to test its purity, as, after a time, the tartaric acid tends to become converted into racemic acid, a substance capable of reducing copper salts. If on boiling the test-fluid no copper is reduced, the suspected urine should be added drop by drop, and the mixed fluid again raised to the boiling-point. The boiling should never be prolonged, lest the oxide be formed without the presence of sugar. If the sugar be present in quantity, it will throw down the copper in the form of a red or orange precipitate of the suboxide of copper. The quantity of urine added should never exceed half the bulk of the test-fluid. Should the suboxide be thrown down before the urine is added, fresh test-fluid must be prepared. If the urine contain less than half per cent., the precipitation does not take place immediately, but occurs as the liquid cools—in five, ten, or twenty minutes—and the manner of the change is peculiar. First the mixture loses its transparency and passes from a clear olive-green to a milky opacity. An increased delicacy can be imparted to this method by first filtering the urine through animal charcoal until it is completely colourless. A little pure water is then passed through the filter, and to this water the test is applied in the usual way. An exceedingly minute trace of sugar (0.01 per cent.) can be detected by this procedure.

It must be borne in mind that urine rich in uric acid may give, if boiled for some time with Fehling's solution, a dirty greenish-brown colour which might be mistaken for a minute quantity of sugar, and that other bodies (lævulose, maltose, pentose, glycuronic acid, creatin and creatinin, pyrocatechin and hippuric acid) behave similarly; it is therefore advisable in a doubtful case to apply another test, such as the fermentation-test.

2. *Fermentation-Test*.—The fermentation-test is

useful, as it is a positive test for glucose and therefore assists in the differentiation of it from the other bodies which give a reaction with Fehling's solution.

The test depends on the decomposition of glucose in the presence of yeast, with the formation of carbon dioxide and alcohol.

The test is best carried out by taking two test-tubes and placing in each a piece of yeast equal in size to about one-fiftieth of the capacity of the tube; one tube is filled to the brim with the urine suspected of containing sugar, and the other with normal urine to serve as a control experiment. The mouth of each tube is closed by an indiarubber stopper, through which passes a small glass tube, bent twice at right angles. The two test-tubes are inverted and put in a warm place (75° to 80° F.) and kept there for 18 to 24 hours. If sugar is present in the urine, carbon dioxide gas collects at the top of the test-tube, while in the normal urine only a few bubbles are given off from the yeast, which may contain a little gas, and the two test-tubes can then be readily compared.

The test is not so sensitive as other tests, but it is a positive test, for if gas is given off from the suspected urine in excess of that yielded by the normal urine, we may conclude that sugar is present. In rare cases the urine may contain a substance preventing fermentation, and to avoid such a fallacy two similar test-tubes are sometimes used, each containing in addition to the yeast a small equal amount of grape-sugar: one is then filled with normal urine and the other with the suspected urine.

3. *Phenylhydrazine-Test*.—Phenylhydrazine acts on many sugars forming crystalline compounds—osazones—and according to the form of sugar used these compounds show variations in crystalline appearance, melting-point, and solubility. Phenylhydrazine acting on grape-sugar forms phenylglucosazone, a crystalline body, the crystals consisting of bright yellow needles. For clinical purposes R. T. Williamson has devised an excellent method of applying this test. A test-tube of ordinary size is filled for about half an inch with hydrochlorate of phenylhydrazine (in powder), then acetate of sodium is added for another half-inch. The test-tube is then half filled with urine and boiled over a spirit lamp by applying the flame to the bottom of the tube; the powders soon pass into solution, the boiling is continued for two to three minutes, and the tube is then allowed to stand for a short time. If sugar be present a yellow deposit forms at the bottom of the test-tube, which on microscopical examination is found to consist of bright yellow needle-shaped crystals arranged in bundles or sheaves. If no sugar is present only brownish amorphous globules or yellowish scales are found in the deposit. The phenylhydrazine-test has the advantage over Fehling's test that it gives no reaction with those bodies which give the copper-test with the exception of glycuronic acid, which if present in the urine may give rise to a deposit of crystals similar to those of phenylglucosazone. As glycuronic acid does not often occur in the urine, its presence generally depending on the administration of certain drugs, the objection to this test is not of any great moment. Another objection, however, is that the test is too sensitive, for even in normal urine a deposit of yellow needles may be obtained, although they are smaller than the crystals

from diabetic urine. Williamson states that, with his modification of the test, he never found these yellow crystals in normal urine. It is therefore valuable not only as a positive but also as a negative test, and urine which gives no reaction by this method may be declared free from sugar. See URINARY DEPOSITS, *Coloured Plate*.

4. *Examination by the polariscope* is useful in differentiating dextrose from those bodies in the urine which turn the polarised light to the left (lævulose, glycuronic acid, β -oxybutyric acid). The apparatus used is Veutcke-Soleil's or a simple one by Zeiss.

In examining urine in which the presence of sugar is doubtful and in which Fehling's solution may show only very slight reduction (being unchanged at first, but becoming turbid and greenish-yellow when the tube is allowed to cool), we should apply the phenylhydrazine-test, as a positive reaction with this means the presence of sugar. But as possibly glycuronic acid may be the cause, it is well to apply the fermentation-test. If the suspected urine causes reduction of the copper with Fehling's solution, while the filtrate after the fermentation-test has been applied fails to reduce the copper, we may conclude that the urine contained sugar. See also URINE, *Morbid Conditions*.

Quantitative Analysis.—1. *Fehling's volumetric method.*—This method, now in common use, is founded on the fact that the proportion in which sugar reduces copper is constant. One equivalent of grape-sugar decomposes exactly ten of sulphate of copper, or 180 parts by weight of grape-sugar decompose 1246·8 parts by weight of sulphate of copper. If a standard solution of copper be used, it is therefore easy to determine the quantity of sugar in any given specimen of urine:—The following is the formula for the standard solution: Sulphate of Copper (crystals) 40 grammes: Tartrate of Potassium (neutral) 160 grammes: Liquor Sodæ (sp. gr. 1·12) 750 grammes: Water to 1154·5 cubic centimetres. These should be carefully mixed; or, what is better, the copper and alkaline solutions are made separately, so that five cubic centimetres of each, or ten of the mixed fluids, will be decomposed by ·05 grammes of sugar.

In mixing, the copper should be added to the alkaline solution to prevent the formation of any precipitate. Most specimens of diabetic urine, containing too much sugar for accurate testing, first require dilution with water; and the most convenient degree of dilution is when one-tenth of the fluid is urine. Next put ten cubic centimetres of the mixed Fehling's solution, with an equal quantity of water, into a small porcelain capsule. The porcelain capsule with its contents is to be placed on an iron retort-stand, at such a level that the flame of a spirit-lamp will easily play on the capsule. Meanwhile a burette, graduated from above downwards, in cubic centimetres, is filled up exactly to the zero in the graduated scale with the diluted urine. When the solution of copper is boiling, the urine is added to it from the burette, drop by drop, stirring carefully the while, until signs are shown of a decolouration of the cupric solution. The moment all the copper has been thrown down as suboxide, and all shade of blue or green has disappeared, the addition of the diluted urine is stopped, and the quantity already used read off on the graduated burette. A minute must be allowed for the suboxide to settle, but this interval

must not be prolonged, or a faint blue colour will return. To ascertain the quantity of sugar in the urine is now a simple calculation:—We know that 10 c.c. of Fehling's solution is reduced by exactly ·05 gramme of sugar, and we can, therefore, conclude that this is precisely the amount of sugar contained in the diluted urine which has been required to reduce the Fehling's solution. If therefore we divide the total amount of urine passed in the twenty-four hours by the actual amount of undiluted urine used, and multiply the result by the ·05 gramme, we shall ascertain the number of grammes of sugar passed in the twenty-four hours. In all such analyses the sample examined should be taken from the mixed urine passed during the whole twenty-four hours. That passed after prolonged fasting is the richest in sugar, that passed three or four hours after a meal the poorest.

2. *Roberts's fermentation-method.*—The fermentation-test for sugar in urine has already been described. It remains to point out Roberts's method for utilising the process as a quantitative test. It is as follows:—

Take two twelve-ounce glass bottles. Into one put about four ounces of the suspected urine and add a piece of dry yeast, about the size of a cobnut, after dividing it into small pieces. Cork the bottle with a grooved cork to allow the escape of gas. Fill the companion bottle quite full with the same urine. Cork quite tightly, and set the two side by side for 20 to 24 hours in a warm place. By the end of that time fermentation will have ceased, and the yeast have fallen to the bottom. The specific gravity of the two specimens must now be carefully compared. The fermented urine will have lost density from two causes: firstly, the sugar which gave the increased specific gravity has been destroyed; and, secondly, in its place have been formed alcohol, which is lighter than water, and carbon dioxide, which has escaped. Every degree of specific gravity thus lost represents approximately a grain of sugar in the ounce of urine. Thus, if there is a loss of twenty-five degrees of specific gravity, the urine would contain twenty-five grains of sugar in each ounce. Multiply this by the total number of ounces passed, to get the amount of sugar discharged *per diem*. This plan is convenient for noting the relative quantity of sugar passed day by day, as it can be easily undertaken by the nurse or friends of the patient.

The quantity of sugar varies considerably. In mild cases it amounts to 1 or 2 per cent., in severe cases 4 to 8 per cent.; it may reach 10 and 12 per cent. The daily quantity varies correspondingly from 300 grains to 1 pound or more.

The quantity of sugar secreted varies during the 24 hours. It is generally less at night. It diminishes two or three hours after food and during fasting; it is increased by starchy or saccharine food, and diminished by nitrogenous food. Of the various carbohydrates, dextrose, starches, dextrin, and maltose appear to increase the extent of sugar more than lævulose, inulin, and milk-sugar (lactose), but even in the severest cases a portion of the carbohydrate is oxidised and not excreted as dextrose. The variation in the urine with the quality of the diet is of great importance from a prognostic point: in the lighter cases the sugar will disappear if the food contain but a very small quantity of carbohydrates; in less mild cases it will not disappear unless the diet be limited to nitrogenous

foods, while if the urine continues to contain sugar in spite of a pure nitrogenous diet, the case must be looked upon as severe, for in these cases sugar is formed from the disintegration of the albuminous products in the body; in some cases an increase of the purely nitrogenous diet has actually caused an increase in the amount of sugar passed, while in others starvation for 24 hours, or a diet limited to fat, was followed by disappearance of the sugar.

Fat and alcohol have no effect on the excretion of sugar. Muscular work short of fatigue diminishes the excretion of sugar in the earlier stages of the disease; mental shock and worry increase the excretion of sugar and often the other symptoms of the disease as well.

Of other changes in the urine in diabetes we must notice (1) that occasionally the urine contains levulose, pentose, and glycogen; (2) that, owing to the increase of nitrogenous food taken, urea, uric acid, and creatinin are generally increased, as also are phosphoric and sulphuric acids; (3) that oxalate of lime is often found deposited in the urine; (4) that ammonia is increased in many cases from three to four times its normal quantity, that this has been especially noticed in diabetic coma, and that there is a close relationship between its appearance and that of oxybutyric acid; (5) that albuminuria occurs not infrequently and has not always the same significance. We may distinguish three different types. In the first type, occurring especially in mild cases among the old, and in more severe cases among the young, traces of albumen appear from time to time, and if a deposit is found this shows, besides mycelium, spores, epithelial cells, and a few hyaline casts. The albumen in such cases is of no great significance. In a second series of cases we have to do with diabetes complicated with nephritis. This is often of the chronic interstitial variety and occurs especially in gouty middle-aged persons. In these cases we often find granular casts, and the urine has a low specific gravity: moreover the sugar gradually becomes less and may eventually quite disappear with the progress of the case, and all the symptoms of a granular kidney develop. In a third series of cases the albumen appears with the presence of acetonuria either before or with the onset of diabetic coma, and is often of grave prognostic omen. In these cases it is probably due to an irritation of epithelial cells of the convoluted tubes by a toxic agent. Naunyn on the other hand states that he has observed many cases of diabetes with acetonuria going on for a long time without showing any albumen.

Diabetic acidosis (acid intoxication): β -oxybutyric acid, aceto-acetic acid, acetone (acetonuria). It is now admitted by most clinicians that in severe cases of diabetes, and especially in diabetic coma, abnormal acids are formed, the best known of which is the β -oxybutyric acid, which easily splits up into aceto-acetic acid, and this again into acetone and carbonic acid. See URINE, Morbid Conditions of.

The presence of β -oxybutyric acid may be detected as already indicated when the diabetic urine after complete fermentation rotates the plane of polarised light to the left, or when the amount of sugar determined by Fehling's method is considerably higher than when estimated by the polariscope. For clinical purposes it is sufficient to test for aceto-acetic acid by Gerhard's test, which consists in the addition of a few drops of *Liquor ferri*

perchloridi to the urine, which causes at first a precipitation of the phosphates, which on further addition of the perchloride dissolve again, the urine assuming a claret-red colour. This reaction is often found in the severe cases of diabetes and almost always in diabetic coma, and should be looked upon as a grave sign. Both the breath of the patient and the urine have a peculiar sweet smell like chloroform, and this is due to acetone which may easily be detected in the urine and in the breath, and is often so strong that it pervades the whole room and can be perceived at some distance from the patient.

It must be noted that if a patient be taking a compound of salicylic acid the urine gives with perchloride of iron a purplish-brown colouration, which is especially noticed when the urine is diluted. Moreover such urine does not possess the characteristic odour. It is further to be borne in mind that the aceto-acetic acid reaction may be noted in other affections besides diabetes, such as affections of the digestive tract, chronic alcoholism, Graves's disease, and hysterical vomiting.

The skin.—The skin in many severe cases is dry and harsh, contrasting with the peculiar soft and velvety appearance sometimes seen in diabetes insipidus; in some cases however the skin feels moist, and perspiration may be excessive. Sugar in the sweat has been noticed in a few cases. In one case it has been found in the tears. Pruritus is common and ceases with the disappearance of sugar from the urine; it is not often general but is usually confined to the genital organs (vulva, glans penis, prepuce, scrotum); pruritus of the vulva with its attendant excoriation is often one of the first signs of diabetes in stout women. Bronzing of the skin in rare cases has been noticed.

The skin and subcutaneous tissue are often the seat of various other changes which are either due to the presence of sugar, to the disturbance in the circulation, or to other as yet unascertained causes. The most common of these changes are: erythema and eczema, especially of vulva, glans penis, urticaria (rare), psoriasis, boils, carbuncles, and gangrene; the latter may occur spontaneously, after slight injury, or as a sequence of slight inflammation of the skin or cellular tissue: in most cases of gangrene lumen of the arteries is diminished by atheroma, and the blood-supply consequently deficient; hence the intermittent limping (*claudication intermittente*) noticed by Charcot. This consists in the sudden onset of pain in the legs when walking quickly, sufficient to produce marked limping and temporarily to prevent the patient from walking. Rarer symptoms are perforating ulcers, more or less localised oedema, and xanthelasma. See XANTHOMA.

Digestive system.—Thirst and dryness of the mouth, especially in severe cases, are early and prominent symptoms; they are unrelieved by the quantity of fluid taken, however large. The appetite is often but by no means always increased, and the patient is often troubled with gastric catarrh and other digestive troubles; the bowels are usually constipated; occasionally there is diarrhoea, often due to the non-assimilation of the nitrogenous and fatty food, but sometimes to tubercular enteritis. Severe gastric pain and vomiting are often the first symptoms of approaching diabetic coma.

There is very little saliva secreted, and its reaction is usually acid; the gums are often red and spongy; the teeth often become carious without giving rise

to much pain, and drop out as the disease progresses; the tongue appears large, red, clean, and fissured; the tonsils may become the seat of abscess or gangrene; white deposits due to the growth of fungus are seen on the mucous surface of the mouth and pharynx; while the breath has in severe cases a peculiar sweet odour due to acetone.

The stomach, in rare instances, may be dilated; the gastric juice is free from sugar, unless carbohydrates are taken; and the quantity of free hydrochloric acid varies, being occasionally absent for some time. The liver may be found enlarged: in many cases this is due to fatty or cirrhotic changes unconnected, or only remotely connected, with the disease; in others (according to Frerichs) the enlargement is due to active hyperæmia of the liver. Gall-stones have not infrequently been noticed in diabetic patients; in a few cases passage of a gall-stone has been followed by disappearance of the diabetic symptoms.

Respiratory system.—Pulmonary tuberculosis is one of the most common complications of diabetes, and it may be said that more than one-third of the cases of diabetes die from phthisis. It is often latent and may run a rapid course with early and extensive excavation. Other pulmonary complications are gangrene, which also may run a rapidly fatal course; broncho-pneumonia, and acute croupous pneumonia which occasionally gives rise to few subjective signs, but is almost always fatal.

Circulatory system.—The heart is usually unaffected, though hypertrophy and dilatation are occasionally met with. Towards the termination of the case the heart's action is often very weak and feeble, and the heart is then found small and atrophied.

Atheroma of the arteries is very common among the older patients, but is not dependent on the glycosuria. It is a cause of the anginal attacks and cardiac asthma so frequently noticed in diabetic patients.

Blood.—Opinions are much divided on the composition of the blood in diabetes. Without going into minute details, it may be affirmed that the blood, though alkaline, is less alkaline than normal, that during the diabetic coma and in very debilitated subjects the percentage of water is generally slightly diminished and the specific gravity of the blood somewhat increased; that the red blood-corpuscles are increased as well as the glycogen; that fat, in the form of fine fat-drops, may be present in the severe cases, and sometimes to such an extent that the blood may have a pink colour, and on standing a creamy sediment; and that the sugar in the blood is increased, reaching, according to Pavy, '27 to '57 per cent. Williamson describes a simple method of distinguishing diabetic from non-diabetic blood: a drop of diabetic blood is added in definite proportion to an alkaline solution of methylene blue; on heating this mixture the blue colour will be discharged.

Nervous system.—Diabetic patients are often subject to nervous affections of the most varied kind. Commencing with the *peripheral nerves* we notice frequently cramps in the calf-muscles, myalgia, neuralgic pains in the distribution of one or more spinal nerves, especially the sciatic nerve; while typical multiple peripheral neuritis has been observed repeatedly: this often affects the lower extremities, the patient complaining of weakness in walking and of sensory disturbances (pain, paræsthesia of peri-

pheral nerves). The knee-jerks are absent, the affected muscles are wasted, and vaso-motor and trophic disturbances (perforating ulcer) are occasionally superadded. Regarding the knee-jerk, it must be noted that it is generally absent in the more severe cases, apart from any other signs of peripheral neuritis.

Affections of the *spinal cord*, such as tabes dorsalis and disseminated sclerosis, may occasionally precede the appearance of diabetes, but only rarely occur as complications of it. Changes, however, have been found in the posterior columns, when during life there were few if any spinal symptoms, and these changes are comparable with similar changes noticed in severe exhaustive and constitutional diseases, such as pernicious anæmia and cancer. See COMBINED DEGENERATIONS OF THE SPINAL CORD; and INSANITY IN SPECIAL DISEASES.

Cerebral complications, which may cause hemiplegia or monoplegia, are often due to hæmorrhage, or to softening resulting from the atheromatous condition of the arteries or due to some toxic agent which causes the symptoms without producing any recognisable lesion; on the other hand, when a cerebral tumour is present, it is generally the cause of the diabetes. When diabetes and epilepsy occur together, the latter may be either the cause or the result, or bear no relation to the former.

Organs of Special Sense.—1. *Eye.*—Soft cataract is a common lesion in severe cases and runs a fairly rapid course. Albuminuric retinitis occurs in cases complicated with albuminuria, but in non-albuminuric cases retinitis is occasionally observed, characterised either by punctiform hæmorrhages scattered over the retina, by small bright patches near the centre of the retina or near the optic disc, or by a combination of these two forms. Defective accommodation is often noticed, and, according to Hirschberg, is an early symptom of diabetes. Hæmorrhagic glaucoma, iritis, purulent keratitis, and atrophy of the optic nerve are met with rarely.

2. *Ear.*—Kuhn draws attention to the frequent occurrence of furunculosis of the external ear and rapidly developing otitis media. See BOILS.

Diabetic Coma.—By the expression 'diabetic coma' is meant a group of symptoms in which coma, followed by death, is the prominent feature. It forms one of the most serious and most frequent complications of diabetes, about half the number of cases of diabetes dying from diabetic coma. It may come on early or late in the disease, and occurs more frequently in persons under forty years of age. Of exciting causes one may note muscular fatigue, shock, anxiety, obstinate constipation, sudden change from the non-diabetic to the diabetic diet, or some intercurrent disease. In many cases the symptoms come on suddenly; in others they are preceded by a rapid loss of flesh, a tendency to drowsiness, some gastric derangement, the characteristic odour of acetone in the breath, or the presence of aceto-acetic acid in the urine. In many cases the appearance of a large number of finely granular or hyaline casts in the urine immediately precedes the onset of the coma.

In by far the largest number of cases the onset of the symptoms is more or less sudden. The patient complains of pain in the abdomen, with vomiting, restlessness, and great languor. These symptoms are soon followed by dyspnoea ('air-hunger' of Kussmaul), the breathing becoming deeper, but not more frequent. Drowsiness follows, and this

gradually develops into coma. The characteristic odour of the breath often pervades the whole room, the pulse becomes quick and small and the temperature sub-normal, the patient's appearance is pale and occasionally cyanotic, the tongue is dry, there is often constipation, the pupils generally react till deep coma has set in, twitching or convulsions occasionally occur, while the urine shows the characters already mentioned. The coma deepens, and death takes place from 12 to 48 hours after the onset. When the coma is fully developed, death almost invariably follows. In some cases, especially if large doses of alkalies are given, the symptoms stop short of the coma, and improvement—often, however, of only temporary character—may follow.

Besides this form of diabetic coma, which has been called the dyspnœal form, there is a second type in which there is sudden collapse, probably due to failure of the heart, and a third in which the symptoms resemble acute alcoholic intoxication.

The pathology of diabetic coma has been studied by many observers, and it is only within the last few years that a more satisfactory explanation of the symptoms and their causation has been found.

The symptoms suggest some form of intoxication, and this view is supported by the absence of any recognisable lesions found after death, except perhaps hyaline and necrotic changes in the renal epithelium. The constant presence of acetone and aceto-acetic acid in the breath and in the urine drew the attention of observers to these substances. Large doses of both of them may, however, be taken with impunity, and they may occur in other affections without producing symptoms of coma. As both acetone and acetic acid are derived from β -oxybutyric acid, and as this body has been found in very large quantities in the urine of persons suffering from diabetic coma, Stadelmann fixed upon this acid as the causal agent of the coma. According to him, and he is supported by many other observers, there is in these cases an increased production of acid (*acidosis*). The acid is believed to combine with alkali and is in this form secreted by the urine. The quantity of alkali in the body necessary to neutralise the acid is thereby diminished, and hence the quantity of ammonia in the urine is considerably increased. The presence of the increased amount of ammonia and of large quantities of β -oxybutyric acid in all cases of diabetic coma, and the experiment of Walther, who by giving animals large doses of hydrochloric acid produced coma with dyspnœa, support this view.

Among other explanations of diabetic coma may be mentioned that of Schmitz, who regards the coma as the result of intoxication due to putrefactive decomposition of the albumen in the intestine, and that of von Noorden, who believes that the active agent consists of toxic substances which cause destruction of the protoplasm of the organism with the formation of oxybutyric acid.

COURSE, TERMINATION, AND PROGNOSIS.—The severe form of diabetes, occurring mostly in the young, and accompanied by profuse glycosuria and marked emaciation, against which no dietetic treatment avails, generally runs a course of some months or even a year or two, and then terminates fatally. Some of the acute pancreatic cases may terminate within a few weeks after the onset of the symptoms.

The prognosis is better in the milder forms

occurring in older people, in persons presenting a gouty history, or suffering from chronic renal disease, or who have partaken of large quantities of carbohydrates, and in cases in which a small amount of sugar is excreted. When there is no loss of weight and the patient observes a careful diet, the sugar often completely disappears, and even if it does not, the patient may live for many years, not much inconvenienced by the glycosuria. Cases are not rare in which sugar has been present in the urine for 15 to 20 years, and in which death took place from some intercurrent affection. Milder forms of diabetes may undergo complete cure. In other cases the glycosuria becomes intermittent. In a few apparently cured cases the symptoms, especially if dieting be neglected, reappear; and in others the mild form of diabetes may, under similar conditions, change into the severe type.

PATHOLOGICAL ANATOMY.—Numerous lesions have been found in persons who have died of diabetes, but they are not constant, and have generally been due to complications or intercurrent affections. The nervous system and the pancreas are chief seats of the causal lesions. According to Hansemann the pancreas is found affected in about 50 per cent. of the fatal cases, the lesions being atrophy and cirrhosis, calculi, cystic degeneration, and cancer of the pancreas, and hæmorrhagic pancreatitis. In the nervous system, tumours, cysts, hæmorrhage, œdema of the brain and of the meninges, minute changes in the medulla, cord, and sympathetic nerves have been found. The changes in the lungs, liver, heart and kidneys have already been mentioned.

TREATMENT.—*Prophylactic treatment.*—Persons with a family predisposition to diabetes, or who are very stout or gouty, should restrict the carbohydrates in the diet, especially if by taking about 100 grammes of grape-sugar for a few days temporary glycosuria is thereby produced (von Noorden).

Curative.—The principal treatment of diabetes is the *dietetic* treatment; but while our object must be to render the urine free from sugar, we must never forget the importance of maintaining the nutrition and general condition of the patient.

In mild cases this object is often easily attained by excluding carbohydrates from the diet, and if after a few weeks the sugar has completely disappeared, fruit, with the exception of grapes, and milk in not too large quantities may be permitted, since the patient after the sugar has disappeared can better assimilate a certain amount of carbohydrates. Such cases should remain under treatment for some time; the urine should be examined daily, that any reappearance of the sugar may be checked. The weight of the patient should be carefully watched so as to detect any decrease of the body-weight. In dieting the patient it is important to take into account not only the quantity of food but also the quality according to its heat-giving value. In many of the mild cases, in persons above forty, the sugar will disappear at once, even though carbohydrates are not entirely excluded. In the more severe cases a strict diet is necessary for a considerable time before the sugar disappears, and even then, with the allowance of a small quantity of carbohydrates, the sugar reappears, though in small quantity. But as a strict diet for a long period is often not well borne, we may, after a time, allow

some carbohydrates, provided the weight does not diminish and the patient does not begin to feel weak. Phthisical patients do badly on a strict diet. In the severe cases, even with a strict diet, the sugar does not completely disappear.

When such a patient comes under treatment a strict diet should be instituted *gradually*; but if after this has been continued for some days the patient's weight steadily diminishes, or if the urine shows the characteristic reaction with perchloride of iron or any other symptom of acid intoxication, the patient should be allowed carbohydrates, including milk, fruit, and vegetables, and, at the same time, large doses of alkalis should be given. In order to raise the heat-giving power of the food and to produce the necessary calorific effect, fat in various forms should be given in considerable quantities.

Articles of diet. Animal and nitrogenous food.—Butcher's meat, flesh-meat of various kinds, poultry, game, and fish may be taken in any form; also tongue, bacon, potted beef, preserved meats, sardines, beef-tea, broth, and soup (liver, oysters, cockles, and mussels, owing to the large amount of glycogen they contain, should not be taken; also the interior of crabs and lobsters). Eggs form a useful form of food on account of the nitrogenous and fatty nature of their contents.

Milk is also useful, but as it contains 5 per cent. of lactose caution is necessary. In mild cases fairly large quantities may be given; in the severe cases the milk should be limited to about one pint. The urine should be carefully watched, for while in many cases the milk has no effect on the excretion of sugar, in others the glycosuria may increase with the administration of milk: in these cases the milk should be stopped.

Cream, which contains much less lactose and a good deal more fat than milk, should be freely taken with or without milk.

Fatty food is, as already stated, most essential to maintain the body-weight of the patient, and should be given in various forms—butter, cream, cheese, suet, and eggs. The patient should also take oil either in the form of salad-oil with his meals, or cod-liver oil after food.

Vegetable Foods.—*Cane- and grape-sugars* should be avoided; milk-sugar is less injurious, while small quantities of *lævulose*, according to Grube and Hale White, may be given in the milder cases. As the sugar in many fruits consists chiefly of *lævulose*, these may be allowed with cream, especially cooking-apples, raspberries, stewed gooseberries, peaches, apricots, and melons. When not sweet enough, *saxin* should be added. Nuts, cocoa-nuts, and almonds may be freely given, but chestnuts, dates, figs, currants, dried raisins, prunes, and plums should be prohibited.

The substitutes for sugar are Saccharine, Saxin, and Dulcin.

Starch is less injurious than sugar, but in all severe cases it should be avoided. Of vegetables, potatoes, rice, sago, tapioca, groats, arrowroot, macaroni, peas, haricot beans, turnips, carrots, beetroot, artichokes, and vegetable marrow are prohibited.

The following vegetables may be allowed freely: salad (mustard and cress), Brussels sprouts, spinach, sorrel, cucumber, mushrooms; other vegetables like celery, onions, and French beans may be allowed in many cases.

Bread.—The ordinary (wheaten) bread containing about 50 per cent. of carbohydrates is a most unsuitable article, and as the patient cannot take his food for any length of time without bread, substitutes for ordinary bread have been devised; but it must be remembered that there is no diabetic bread which does not contain carbohydrates: in many of them the quantity is only less, in some it is even more, than in the ordinary bread. If the patient is therefore to be strictly dieted, bread should be entirely excluded. After a time small quantities of toasted bread may be allowed. Of the substitutes for bread, gluten-bread prepared from gluten-flour is best known, although many of the specimens of gluten-flour are very rich in carbohydrates; almond-bread and almond-cakes are preferable. Saundby gives the following directions: one pound of ground almonds, four eggs, two tablespoonfuls of milk, a pinch of salt. Beat up the eggs, and stir the almond-flour, divide into twelve flat tins, and bake for about fifteen minutes on a moderate oven. Cocoa-nut may be made into biscuits and cakes. Aleuronat—a powder containing 80–90 per cent. of vegetable albumen, and only 7 per cent. of carbohydrates, is a useful article of diet, but it can only be made into bread by the addition of an equal quantity of wheaten flour. Soja-bread made from the flour of a bean used in Japan is recommended by some, but according to Attfield the flour contains 30 per cent. of carbohydrates.

Beverages.—Tea, coffee, aerated waters, light wine containing very little sugar, such as *bordeaux*, *hock*, *moselle*, and the Hungarian wines, dry sherry, whisky and brandy may be allowed, and are often useful, partly because they enable the patient to take fatty food, and partly on account of their calorific value. Cocoa, chocolate, and the sweet wines, such as port, madeira, champagne, and lemonade containing sugar, should be forbidden.

General management and hygienic treatment.—The patient should avoid all mental worry and excitement, take a fair amount of exercise without over-fatiguing himself, clothe himself in wool to avoid getting cold, and have warm baths.

A mild sunny climate is often beneficial. Slight cases and those in which there is a gouty diathesis may be sent to Carlsbad, Vichy, or Neuenahr.

Medicinal treatment.—Drugs are of no great use, but many have been recommended. The most useful are opium and its alkaloids, codeine and morphine. Pavy strongly recommends codeine, given at first in half-grain doses several times daily, and gradually increased till 10–15 grains are taken daily. Other drugs to be mentioned are: the alkalis, arsenic, quinine, sodium salicylate (in gradually increasing doses), salicylate of bismuth, jambal, and phenazone (antipyrine)—strongly recommended by French observers. Pancreatic preparations have not given any encouraging results.

If the onset of diabetic coma be suspected, large quantities of bicarbonate of sodium (50–150 grains three times daily) should be given, an ordinary diet ordered, and alcoholic stimulants administered. If there is any constipation, this should be relieved by enemata or castor oil. Should the symptoms of diabetic coma set in, other measures besides these have been recommended, such as the inhalation of oxygen, the intravenous injection of normal saline solution or of solution of sodium carbonate to the extent of 3–4 pints. *See SALINE SOLUTIONS, INFUSION OF.*

JULIUS DRESCHFELD.

DIAPHORESIS (*διά*, through; and *φορέω*, I convey).—The act of perspiring. The term is generally applied to perspiration artificially induced.

DIAPHORETICS (*διά*, through; and *φορέω*, I convey).

DEFINITION.—Remedies which increase the secretion of sweat. When the increase is so great as to cause the perspiration to stand in beads upon the surface, they are usually termed *sudorifics*.

ENUMERATION.—The principal diaphoretic measures are—The Vapour-Bath, Turkish Bath, and Wet Pack; Warm drinks, Warm Clothing; Jaborandi, Pilocarpine; preparations of Antimony; Ipecacuanha; Opium and Morphine with their preparations; Resorcin, Aconite, Guaiacum, Salicin and Salicylates, Senega, Camphor; Sulphur; Ammonia and its Carbonate, Acetate, and Citrate; Alcohol; Ethers (especially Nitrous Ether); and Chloroform.

ACTION.—The secretion of sweat usually consists of two stages, namely, a free supply of blood to the sweat-glands, and the abstraction from it of the materials for sweat by the cells of the gland. These two processes sometimes occur independently of each other. In fevers the supply of blood to the glands is abundant, but they do not always secrete, although experimentally a rise in the temperature of the blood induces sweating; and a similar condition is observed in belladonna-poisoning. Belladonna and atropine possess the power of paralysing the secretory nerves of the sweat-glands, just as they do those of the salivary glands, and thus the skin remains dry, although the cutaneous vessels are much dilated. In collapse the cutaneous glands secrete a profuse cold sweat, due to the stimulating effect of venous blood on the central mechanism, though the supply of blood to them is deficient.

The secreting cells appear to be under the influence of nerves, by exciting which secretion occurs. The centres for the secretory nerves of the sweat-glands appear to be situated in the spinal cord, and in the medulla oblongata. The fibres seem to run in the same path as the vaso-motor nerves. The secretory nerves of the sweat-glands may be excited directly by stimulation of the nervous trunks in which they run; and the sweat-centres may also be reflexly excited by irritation of various sensory nerves. Certain substances, such as picrotoxin, nicotine, and carbonic acid, seem to stimulate the sweat-centres; while other drugs, such as pilocarpine, either act upon the terminations of the secretory nerves in the sweat-glands, or possibly on the secreting structures themselves. Several remedies, at the same time that they excite secretion, likewise increase the flow of blood through the skin, rendering it redder, warmer, and more vascular. Others, again, excite the secretion at the same time that they diminish the cutaneous circulation. Diaphoretics have therefore been divided into two classes, the former kind being termed *stimulant*, and the latter *sedative* diaphoretics. The exact mode in which each drug already enumerated produces diaphoresis has not yet been ascertained; but antimony, ipecacuanha, and jaborandi are classed as sedative diaphoretics, and all the others as stimulating ones. The supply of blood and the secretion of sweat are both increased by the application of warmth, by the ingestion of warm fluids, and by the action of jaborandi.

USES.—Diaphoretics are employed to increase the flow of blood to the surface, and possibly to aid

the elimination of excrementitious products in internal congestion, such as catarrh of the respiratory passages or digestive tract, and in febrile conditions generally. In fevers, the cutaneous circulation is generally active, and the so-called sedative diaphoretics are then useful, although acetate of ammonia and nitrous ether are largely employed. Diaphoretics are also used to increase the elimination of water by the skin, and thus lessen the accumulation of fluid in dropsy, or to relieve other excreting organs, such as the kidneys in albuminuria and diabetes insipidus, or the intestines in diarrhoea. In these cases stimulant diaphoretics are indicated.

T. LAUDER BRUNTON.

DIAPHRAGM, Diseases of the.—For practical purposes these may be conveniently discussed according to the following arrangement:—

1. PHYSICAL INTERFERENCE WITH THE DIAPHRAGM, AND DISPLACEMENT.

2. FUNCTIONAL DISORDERS. (a) *Paralysis*. (b) *Spasm*.

3. ORGANIC LESIONS. (a) *Injury, Perforation, and Rupture—Diaphragmatic Hernia*. (b) *Inflammation, acute or chronic*. (c) *Muscular Rheumatism*. (d) *Atrophy and Degeneration*. (e) *Morbid Formations*.

1. PHYSICAL INTERFERENCE WITH THE DIAPHRAGM, AND DISPLACEMENT.—The diaphragm is frequently interfered with by morbid conditions within the chest or abdomen, which impede its movements, displace it more or less, either upwards or downwards, or render it tense and stretched. The entire structure may be thus affected, or only a portion of it, such as one lateral half or its central part. The chief thoracic conditions by which the diaphragm may be thus affected are pleuritic effusion, empyema, pneumothorax, emphysema of the lungs, abundant pericardial effusion, enlargements of the heart, and tumours within the chest. Chronic contracting affections of the lung and pleura tend to draw it upwards. Pleuritic adhesions, acting alone, tend to depress it. The principal abdominal conditions which may raise the diaphragm are a distended stomach, tympanites, ascites, peritonitis, pregnancy, large fecal accumulations, and tumours or enlarged organs, which attain considerable dimensions, especially ovarian, hepatic, splenic, or renal tumours. It sometimes happens that the diaphragm is interfered with both from its thoracic and its abdominal aspects. Tight lacing may materially embarrass and alter the position of this structure.

SYMPTOMS.—A sense of uneasiness and discomfort is often experienced around the lower part of the chest, amounting sometimes to considerable tension or tightness. There is not any actual pain, but in some instances, where the diaphragm is much pushed down, the patient complains of a painful sensation referred to the ensiform cartilage, as if the attachment of the diaphragm at this point were being severely dragged upon. The act of respiration is impeded, and this seems to be the cause of the discomfort experienced. A sensation frequently complained of by patients is that they cannot take a full breath. Respiration may be much hurried, or oppressed and laboured; and not uncommonly the normal relation between the thoracic and abdominal movements is markedly altered, and the diaphragm may so act as to draw in the lower part of the chest-walls in inspiration. In acute peritonitis the movements almost cease and breathing becomes

wholly thoracic. Occasionally a kind of spasmodic cough or hiccough seems to be excited by the tension of the diaphragm produced by certain conditions. The act of coughing is also rendered more difficult and less effectual.

The actual position of the different parts of the diaphragm in a particular case can only be determined by carefully noting the physical signs afforded by the structures above and below it.

2. **FUNCTIONAL DISORDERS.**—The affections of the diaphragm included within this group are (*a*) **Paralysis**; and (*b*) **Spasm**. It will be understood that in the cases now under consideration there is no structural change in the diaphragm itself.

a. Paralysis.—The diaphragm is completely paralysed when the spinal cord is destroyed at the origin of the phrenic nerve, whether as the result of injury or disease. If one or both phrenic nerves be injured or compressed, one lateral half or the whole of the diaphragm will be paralysed, according as one or both nerves are involved. This structure may also be implicated in diphtheritic paralysis; lead-palsy; and hysteria. See PHRENIC NERVE, Diseases of.

SYMPTOMS.—Where paralysis of the whole diaphragm is suddenly produced, death speedily ensues from the impediment to the respiratory function resulting therefrom. If the paralysis is brought about gradually, or if only part of the structure is involved, there may be a subjective sensation of a want of power to breathe; while the respiratory movements may be hurried and shallow. When the patient is at rest, breathing is often quite easy, but any exertion causes it at once to become frequent, and brings the extraordinary muscles of respiration into play. Physical examination shows that, if a deep inspiration be taken, the epigastrium and hypochondria sink in instead of protruding, while during expiration these regions bulge out again. If only one half of the diaphragm is affected, this sneezing movement is unilateral. Coughing and sneezing cannot be performed efficiently, and sputa cannot be easily expelled; while the abdominal acts for which a tense diaphragm is required, such as defecation or vomiting, are also ineffectual or impracticable. A peculiar dysphonia has been described, in which the voice loses its power as the day advances, becoming at last a mere cracked whisper. The lower parts of the lungs tend to be more or less collapsed and congested; and if bronchitis sets in, the patient is in grave danger.

b. Spasm.—The diaphragm may be the seat either of clonic or tonic spasm or cramp. The disorder may depend upon disease of the nerve-centre at the origin of the phrenic nerves; irritation of these nerves in their course; direct excitation of the diaphragm; or reflex causes. Tonic spasm is most strikingly observed in cases of tetanus; of poisoning by strychnine; or of hydrophobia. A form of asthmatic attack has also been attributed to this condition of the diaphragm; and it may result from immoderate laughter.

SYMPTOMS.—Tonic contraction of the diaphragm gives rise to severe pain, and a sense of constriction in the corresponding region, which may come on in paroxysms; clonic spasms also originate painful sensations after a time, which may become very considerable. Hiccough is probably due mainly to a clonic spasm of the diaphragm (see HICCOUGH). If this structure should become rigidly fixed, respiration is gravely interfered with, and the patient soon

presents the phenomena of asphyxia, which will end fatally if the spasm is not relieved. In the form of asthma supposed to be due to diaphragmatic spasm, expiration is very difficult and greatly prolonged, inspiration being short and abrupt; the lungs are distended; great distress is felt; and there may be signs of impending death from suffocation. A spasmodic cough may be due to clonic spasm of the diaphragm.

3. **ORGANIC LESIONS.**—These may be briefly considered in the order in which they were enumerated at the commencement of this article.

a. Injury, Perforation, and Rupture.—**Diaphragmatic Hernia.**—The diaphragm may be perforated, lacerated, or ruptured in connection with various forms of injury, such as crushing accidents, fractured ribs, penetrating wounds, or gunshot injuries. Should the patient recover, a permanent opening may be left. Rupture of the diaphragm may also occur from violent strain, as of persistent retching, or during parturition; and from the effort to conceal or suppress the cries of pain in parturition. In medical practice perforation of this structure may be met with as a congenital condition; as the result of the bursting of some fluid-collection through it, such as an empyema, pulmonary abscess, a sub-diaphragmatic, hepatic, renal, or other abscess, or a hydatid cyst; or from its destruction in the progress of some organic lesion, such as malignant disease, an aneurysm, or a chronic gastric ulcer. It may occasionally occur independently of these causes, owing to the yielding of a weak portion of the diaphragm, especially between the attachment to the ensiform cartilage and the seventh rib. Congenital perforation is rare, but a considerable portion of the diaphragm may be thus deficient. The size and other characters of the perforation differ much in different cases. If it is produced by the opening through the diaphragm of a fluid-accumulation, this fluid escapes from the abdominal into the thoracic cavity, or *vice versa*, the latter being exceptional; in other instances portions of the thoracic or abdominal organs pass through the perforation, constituting forms of *diaphragmatic hernia*. The stomach and colon most frequently pass through the diaphragm. In rare instances the small intestines, spleen, or other structure form part of the hernia.

SYMPTOMS.—To recognise clinically a perforation or rupture of the diaphragm is generally no easy matter. In cases of sudden and extensive rupture, there will probably be grave collapse and speedy death, and the patient may present marked *risus sardonius*. Often there are no symptoms referable to the diaphragm, though there may be signs indicating that its functions are more or less impeded. The occurrence of sudden perforation may be known from the previous existence of some condition likely to cause this event, such as empyema, or an abdominal abscess or hydatid tumour; the supervention of acute pain, accompanied by indications of shock or collapse, and the disappearance or modification of the signs of the original morbid condition; followed by the development of phenomena revealing that fluid has passed through the diaphragm, and accumulated in the thoracic or abdominal cavity, as the case may be, or that some secondary affection has been set up as the result of the perforation, such as peritonitis or pleurisy. A fluid-collection may, however, penetrate the diaphragm, without giving rise to any very evident disturbance. When an organ passes through this structure, the symptoms

present, if any, are more likely to be associated with this organ than with the diaphragm, and physical examination may possibly detect the displacement. In the case of hernia of the stomach already referred to, the chief symptom was urgent vomiting, which occurred immediately after taking any food or drink.

b. Inflammation.—The serous coverings of the diaphragm are not uncommonly involved in cases of acute pleurisy, pericarditis, or peritonitis respectively, and the inflammatory process may penetrate its structure through the lymphatics. Acute inflammation of the substance of the diaphragm may further arise from injury, direct irritation, septicæmia, or without any evident cause. The anatomical conditions observed are increased vascularity and sometimes ecchymosis; the formation of lymph upon its surfaces, or exudation into its substance; softening and degeneration of its muscular tissue or central tendon; or, in rare instances, suppuration, an abscess forming in the substance of the diaphragm, or pus collecting under one or other of its serous coverings. Ulceration and gangrene have been noticed in exceptional instances. *Chronic* inflammation of the diaphragm may occur, leading to a fibroid change in its muscular portions, either by extension from neighbouring structures, or as the result of chronic local irritation.

SYMPTOMS.—The symptoms of acute inflammation of the diaphragm are generally very obscure. The condition may be indicated by severe constrictive pain in the region of this structure, obviously increased by breathing, so that the respiration becomes instinctively thoracic, as well as hurried and shallow; and also much aggravated by coughing, defæcation, or any other act which disturbs the diaphragm, as well as by movements of the trunk, and pressure over the epigastrium. Sighing, hiccup, painful dysphagia, risus sardonius, and violent delirium are other symptoms described. The patient is obviously distressed, and has more or less fever. If an abscess form, this may burst either into the chest or abdomen, and thus lead to secondary pleurisy, peritonitis, or pericarditis. Chronic inflammation and its consequences may possibly be suspected from a want of free movement in the diaphragm, associated with conditions likely to originate this change; but it could scarcely be recognised clinically with any certainty.

c. Muscular Rheumatism.—This affection is characterised by pain referred to the diaphragm. So long as this is kept at rest, there may be no discomfort, but deep breathing causes considerable pain, so that the respiration is carried on in a shallow manner, and may be entirely thoracic. Such acts as coughing or defæcation cause much pain and a sense of aching.

d. Atrophy and Degeneration.—The diaphragm may be involved in the course of progressive muscular atrophy; it may also be atrophied from causes which produce general wasting; or undergo senile atrophy and degeneration; or be similarly affected from local causes, such as interference with its blood-supply from vascular degeneration, want of action, or after chronic inflammation. Fatty and fibroid degeneration are the chief forms met with. Brawny induration has been noticed in scurvy. These conditions tend to give rise to more or less evident interference with the functions of the diaphragm, which in extreme cases amount to their total cessation, diaphragmatic

breathing being rendered impossible, the symptoms being then the same as when the diaphragm is paralysed. In cases of progressive muscular atrophy the fatal termination may arise from this cause. There is no pain, but uncomfortable sensations may arise from the impeded respiration.

e. Morbid Formations.—The diaphragm is occasionally the seat of malignant disease, being involved by extension from some neighbouring structure. Syphilitic and other non-malignant solid growths have in rare instances been found in it. Parasitic formations may also occur in it—such as hydatids, cysticercus, and *Trichina spiralis*. Tuberculosis may involve the diaphragm. Possibly malignant disease might be indicated by signs of impeded diaphragmatic movements, with localised pain, accompanying indications of cancer in other parts. The implication of the diaphragm in trichinosis may also be recognised in some instances by severe pains, spasmodic contractions, and serious interference with diaphragmatic respiration. In most cases, however, the presence of any morbid growth in connection with the diaphragm cannot be diagnosed during life.

DIAGNOSIS.—In addition to the indications given in each section, it may be noted generally that Röntgen rays are of considerable service in the diagnosis of disorders of the diaphragm. Its position and movements can be readily observed on the fluorescent screen.

TREATMENT.—But little can be done in most cases in the way of direct treatment. One of the most obvious indications is to get rid, if possible, of any condition which is mechanically displacing it, or impeding its movements, and preventing it from performing its functions. In the next place, any disease of which the condition of the diaphragm is but a part must receive due attention, such as progressive muscular atrophy, central nervous disease, diphtheritic paralysis, lead-poisoning, hysteria, or trichinosis. Collapse or shock due to a sudden diaphragmatic lesion must be treated on the usual principles. Painful affections might be relieved by local applications of dry heat, fomentations, support by a bandage, or anodynes; and if acute inflammation is suspected, a few leeches might be applied. Paralysis or spasm of the diaphragm may call for the employment of electrical treatment, applied through the phrenic nerve; and various remedies are found useful in hiccup. See PHRENIC NERVE, Diseases of; and HICCUPH.

FREDERICK T. ROBERTS.

DIAPHRAGMATIC HERNIA.—See DIAPHRAGM, Diseases of the.

DIARRHŒA (διάρρῶν, I flow away).—SYNON.: *Defluxio*; *Alvi Fluxus*; *Purgus*; Fr. *Cours de Ventre*; *Dévoisement*; Ger. *Der Durchfall*; *Bauchflus*; *Durchlauf*.

DEFINITION.—A frequent and profuse discharge of loose or of fluid alvine evacuations, without tenesmus, due to increased secretion of fluid into the intestine, or diminished absorption of fluid from it, or to increased peristalsis of the large intestine.

ÆTIOLOGY.—The causes *predisposing* to diarrhœa are individual peculiarity; childhood—especially the period of first dentition; the climacteric period; and hereditary or acquired weakness of the digestive organs.—The *exciting* causes may be thus classified:

1. *Direct irritation of the intestines* by: (a) *Food* in

excess, or of improper quality—for example, salted meat, shell-fish, sour unripe fruit, and vegetables—diseased, decomposed, or imperfectly masticated; the products of faulty digestion prematurely passing the pylorus; imperfectly elaborated and fermenting chyme; impure water; or imperfectly fermented malt-liquors. Acute diarrhœa, especially in hand-fed infants, is very frequently caused by milk—the irritant effect being due either to undigested casein, to the products of acid or alkaline fermentation, to the formation of poisonous ptomaines, or to the presence of bacteria. (b) *Purgative medicines and irritant poisons.* (c) *Microbic poisons.* (d) *Bile,* excessive or acrid. (e) *Fæces,* retained. (f) *Entozoa*—lumbriçi, tæniæ, trichinæ, and *entophyta*—mycosis enteritis. (g) The contents of a *ruptured abscess* or *hydatid cyst.* (h) *Intestinal lesion*—such as tubercular or other ulceration. 2. *Defective hygiene.*—Diarrhœa may arise from the dwelling being damp, cold, dark, and unventilated; or from foul emanations from decaying organic substances, especially animal matter, sewage, or fæcal collections. 3. *Chills, climatic variations, &c.* Diarrhœa has been attributed to insufficient clothing; sudden exposure to cold and damp; chills, as from wet feet, and damp bed or clothing; and rapid variations of temperature, such as hot days and cold nights. 4. *Nervous disturbances,* for example, depressing emotions—fright, grief; neuralgia, dentition, and other causes of reflex disorder. 5. *Defective absorption with augmented peristalsis* of the small intestines, so that the food is passed unaltered—*lenteric diarrhœa.* 6. *Symptomatic* in various morbid states, for instance, in passive congestion of the portal vein from disease of the liver, heart, or lungs; peritonitis, especially puerperal; organic disease of the intestines—ulceration (simple, stercoral, typhoid, tubercular, cancerous), lardaceous degeneration, enteritis, acute or chronic; cholera; typhoid fever; dysentery; occasionally in pyæmia, septicæmia, measles, scarlatina, confluent small-pox, malaria, gout, Bright's disease (its later stages), and in anæmia and exhaustion, as from over-lactation, phthisis, cancer, Addison's disease, Hodgkin's disease, lymphosarcoma of the bowel, exophthalmic goitre, leuchæmia, and other affections.

Frequently diarrhœa arises from the combined action of several exciting causes, as when the disease is epidemic during summer and autumn. Pollution of air, water, and food by foul emanations from organic matter decomposing in very hot weather, overcrowding, food (and especially fruit) in a state of incipient decay, excessive heat, and chills, may then collectively determine the result. In children the exalted irritability of the nervous system during dentition predisposes to diarrhœa from slight determining causes. In several forms of diarrhœa fermentation of the intestinal contents plays an important part—e.g. in infantile and summer diarrhœa.

DESCRIPTION AND VARIETIES.—Diarrhœa may be broadly divided into the *acute* or occasional, and the *chronic* forms; and the numerous clinical and pathological peculiarities of different cases are conveniently grouped into typical varieties. The general effects, varying according to the intensity and duration of the flux, are mainly these:—Emaciation, and, in children, also arrest of growth—the weight either diminishing or ceasing to be progressive; anæmia; desiccation of the tissues from the rapid draining of serum from the blood—hence the thirst, and the very concentrated, acid, and even albuminous urine

observed when there is a copious watery outflow from the bowels, as in choleraic and similar forms of diarrhœa.

It will be expedient to describe briefly the principal forms of diarrhœa.

1. **Irritative Diarrhœa.**—**SYNON.** : *Diarrhœa crapulosa* (Cullen).—Simple flux from direct irritation of the intestines is the most common variety of diarrhœa. The evacuations, usually preceded by severe griping pains, are at first fæculent and usually foetid and sour, then watery. In children (especially hand-fed) they are often like pale clay or putty, or they contain dense masses of undigested casein before being loose; after evacuation they frequently become greenish, like chopped spinach, from contact with very concentrated acid urine converting the brown colouring matter of the bile into green biliverdin; or they are dark green when passed, and may be so acrid as to excoriate the anus, the genitals, the inner parts of the thighs, and even the heels. Fever is usually absent.

Diarrhœa from irritation is frequently a preliminary stage of the inflammatory, dysenteric, and choleraic varieties.

2. **Inflammatory Diarrhœa.**—**SYNON.** : *Diarrhœa serosa.*—When the causes of simple irritation excite inflammation of the mucous membrane of the bowels, fever sets in, and the diarrhœa increases. Usually the evacuations become more serous, and contain shreds of fibrin or mucus or pus. Before the attack passes off the large bowel is apt to be the main seat of inflammation; then the motions are scanty, frequent, more mucous or glairy, contain streaks of blood, and are passed with severe straining. At the same time the skin is hot and dry.

3. **Choleraic Diarrhœa.**—**SYNON.** : *Thermic diarrhœa.*—This form prevails in hot weather. The onset, indicated by vomiting and purging, is usually sudden. At first the vomited matters are mucous and bile-tinted, and the dejections are fæculent—both quickly, however, becoming more and more abundant, watery, and colourless. The copious and incessant outflow of serum may in a short time, and especially in children, induce a striking resemblance to the symptoms of Asiatic cholera—a drawn, sunken, and cyanotic appearance, fall of temperature, scanty secretion of urine, insatiable thirst, and cramps. Even in extreme cases, however, the fluids from the stomach and bowels are rarely free from bile, and are not so like rice-water as in true cholera. The collapsed algid condition, as a rule, rapidly gives place to recovery in previously healthy adults, while it is apt to become fatal in delicate children, children prematurely weaned, the debilitated, and the aged. Rarely, the cold stage being outlived, the patient becomes hot, and passes into a state of stupor, with either bilious vomiting or purging and tympanites—the typhoid stage. In children death is almost inevitable if the cold stage exceeds twenty-four hours. See CHOLERAIC DIARRHŒA.

4. **Nervous Diarrhœa.**—The peristaltic movements and the activity of the glands of the alimentary canal are often increased by causes operating through the nervous system. Diarrhœa from mental, and especially emotional, disturbance is the most common example. Even a chronic looseness may be maintained by debility of the nervous system, induced by worry and anxiety. Exalted innervation of the bowels may be natural, a prone-

ness to diarrhœa from slight exciting causes having always existed; or acquired, when, for instance, a flux once established is apt to be maintained. The instability of the nervous system during the periods of rapid development and of the climacteric change predisposes to it. It is often an important factor in chronic diarrhœa. The intestinal nerve-centres may become so sensitive (as in delicate children) that every meal, however small, may induce an immediate call to stool, the motions being liquid or pultaceous, and pale, but otherwise healthy. The peristaltic movements may be even so increased as to hurry the food through the stomach and bowels, so that it appears unchanged in the stools. Time is not allowed for digestion or absorption to be even begun. This form has been termed *Diarrhœa lenterica*, and is most frequent in children before the period of the second dentition. Indigestion is the usual cause in adults. The appetite is, as a rule, voracious; and debility may become extreme.

5. Morning Diarrhœa.—This form is not uncommonly met with, and is characterised by frequent action of the bowels in the early morning or forenoon, the disturbance usually ceasing during the latter half of the day. It occurs in association with alcoholism, dilatation of the stomach, and irritability of the sigmoid flexure of the colon. Usually it is not severe, but is persistent and does not yield readily to treatment.

6. Vicarious Diarrhœa.—Embarrassment or suppression of the functions of the skin, kidneys, or lungs may lead to the bowels performing additional excretory work. The flux thus set up is salutary, because compensatory. Diarrhœa from chills (suppressed perspiration) is a common instance, while that from renal and pulmonary causes is less frequently observed, and may be misconstrued by the practitioner; for inasmuch as diarrhœa diminishes the quantity of urine, the causal influence of renal disease accompanied by diminution in the amount of water passed in producing diarrhœa may easily be overlooked. Even when forewarned, the observer may at times—especially when the urine is free from albumen—find it difficult to determine whether the diarrhœa is a cause or an effect of imperfect renal elimination: a distinction having all-important bearings on the treatment. The uræmic and eliminatory character of it may be easily decided when the kidneys are known to be diseased; not so, however, when the only thing ascertainable is scanty urine, or total suppression of urine in an elderly patient. Diarrhœa from pulmonary embarrassment generally affords relief to breathing and cough. The chronic looseness of some gouty patients is also eliminatory: when it is checked, gout is apt to advance, and the health to suffer.

7. Diarrhœa from Mechanical Congestion.—Draining of serum into the bowels is a common result of overloading of the portal vein from an impediment to the flow of blood, either in the vein itself, the vena cava, or the right side of the heart.

8. Chronic Diarrhœa.—**SYNON.** : *Cachectic diarrhœa*.—Chronic diarrhœa is frequently, if not generally, unconnected with intestinal lesions. It may be maintained by chronic catarrh of the intestines, or by an exhausted and impoverished state of the system, as in inanition, either from insufficiency of food or from enfeebled digestion, or in chronic wasting diseases, such as syphilis, malaria, or scurvy.

The flux increasing, the debility on which it depends thereby perpetuates itself, and this vicious circle tends more and more to destroy life by anæmia and exhaustion, and even after apparent recovery the diarrhœa has a strong disposition to relapse. These clinical features of chronic diarrhœa are well illustrated by the malady which, from the paleness of the stools, is commonly known in India as 'White Flux' (see *PSILOSI*). When accompanied by fever and night-sweats, chronic diarrhœa is nearly always due to intestinal tuberculosis.

DIAGNOSIS.—The different forms of diarrhœa may be readily distinguished from each other by a careful consideration of the causes and symptoms. The diseases most apt to be mistaken for diarrhœa are epidemic cholera, dysentery, and mucous irritation of the bowels from retention of fæces.

(a) *Cholera*, in its less definite forms, may resemble bilious diarrhœa and choleraic diarrhœa. The probability in favour of it may be determined by the absence of ordinary causes of diarrhœa, the paleness and watery character of the stools, tormina being slight or absent, the suppression of urine, and the early exhaustion. The presence of bile in the stools is always in favour of diarrhœa. Vomiting is more frequent in cholera; when it occurs in diarrhœa the vomited matter usually contains bile and undigested food, while in cholera it is a colourless fluid.

(b) *Dysentery* is usually characterised by fever, tormina, and tenesmus, and frequent scanty mucosanguinolent evacuations. Sometimes, however, in the early stage, the motions are copious, watery, and feculent, as in ordinary diarrhœa; but the presence of tormina and tenesmus, and tenderness in the regions of the cæcum and sigmoid flexure, indicate the dysenteric nature of the disease. Chronic diarrhœa may be distinguished from chronic dysentery by the absence of a history of acute dysentery, and of mucus and tenesmus, and by the less frequent discharge of blood in the evacuations.

(c) *Mucous irritation of the bowels* from retention of fæces may induce a condition resembling diarrhœa—frequent, thin, muco-fæculent evacuations, which are, however, shown, on inquiry, to be somewhat scanty, and voided with straining. See **CONSTIPATION; DEFÆCATION, Disorders of; and** **INTESTINAL OBSTRUCTION.**

TREATMENT.—(a) *Diet and Hygiene.*—In *acute* or *occasional* attacks of diarrhœa, everything should be taken in small quantity, and tepid or cold, never hot. Farinacea—arrowroot, sago, rice, tapioca, flour, and the like—are useful, and may be taken in milk, or in chicken- or mutton-broth, or weak beef-tea. Animal broths—and especially beef-tea—when concentrated, or in large quantity, are apt to aggravate diarrhœa. Demulcent drinks—white of egg in water or milk, rice-, barley-, or arrowroot-water; and astringent liquids—infusion of dried whortleberries or roasted acorns, red light wines—may be given. Brandy is often of service, and may be given in an aromatic water or with the farinacea. Lime-water with milk is in many cases of much value.

Rest in bed secures a uniform warmth of skin, and favours the cessation of diarrhœa.

In children, errors of feeding should be corrected. Lumps of casein in the motions and infantile diarrhœa should be treated by sterilising, or partly peptonising, the milk, and by adding thin arrowroot, barley-water, or lime-water; and by regu-

lating the time between meals, providing a wet-nurse, or substituting the milk of a goat or ass for that of the cow. The feeding-bottles, when not in use, should be kept in a solution of boric acid. Sometimes, however, milk must be given up, and barley-water or thin rice-water, sweetened by saccharin or milk-sugar, or albumen-water substituted. The abdomen should be protected by a flannel bandage, and the feet and legs by warm clothing. See INFANTS, Diseases of; and DENTITION, Disorders of.

Inasmuch as in *chronic diarrhœa* the flux is perpetuated by the debility and anæmia which it induces, and by the activity of intestinal digestion, it has become a leading principle of treatment to prescribe food rich in materials for the construction of the blood and the tissues, and almost wholly disposed of by the stomach. Hence the happy results frequently observed from a diet exclusively animal, either raw or lightly cooked, the digestion of which may be aided by hydrochloric acid, alone or with pepsin. Individual peculiarity may be gratified, and variety obtained, by the use of mutton, veal, chicken, pigeon, and game. Beef, the tough parts of veal, and pork are, as a rule, to be avoided. Milk and farinacea are gradually permitted during the progress towards recovery, but the period during which they should be interdicted may require to be very prolonged—even months. The treatment with *raw meat*, strongly advocated by Trousseau and Niemeyer, has been successfully applied to nearly every variety of chronic diarrhœa, but especially to that obstinate form occurring from the time of weaning to the close of the first dentition. The meat may be pounded into a pulp or finely minced; then mixed with salt, sugar, fruit-jelly, or conserve of roses; or diffused through clear gravy soup, or chocolate made with water or wine; or the juice may be extracted from it by pressure. Notwithstanding the prohibition of other food, it is best to begin with a small quantity, and to increase it gradually. The only drink allowable is water containing white of egg. Trousseau found opium in small doses, chalk, and bismuth, at and between meals, to assist this regimen. When a restricted animal diet causes loathing, or aggravates the flux, other varieties of food may be added, and the feeding should be as generous and varied as possible, and adapted to the digestion of the individual. Articles of diet appearing undigested in the motions should be avoided. Low and damp situations should be exchanged for dry and open ones. Warm clothing, flannel next the skin, and flannel waist-belts should be worn. Chronic diarrhœa (lienteric and chronic tropical diarrhœa) has also been successfully treated by a strict milk-diet and rest.

(b) *Medicinal Treatment.*—A routine prescription of astringents is much to be deprecated. When the flux is moderate and salutary—for example, removing undigested or indigestible materials or irritating secretions, relieving an engorged portal vein, or supplementing a suppressed secretion—it may be left uncontrolled by medicine, or may be encouraged by laxatives, such as castor oil, rhubarb, or a saline aperient, combined with a mild sedative, such as henbane or opium. As a rule, the treatment of diarrhœa should begin by removing irritating substances from the alimentary canal by laxatives, guarded by small doses of opium; and astringents, such as chalk-mixture with kino, catechu, hæmatoxylum, and opium, should be held in reserve and

not resorted to until intestinal disinfectants (see below) have been tried. Castor oil in small doses is by far the most useful remedy for children, as well as for adults. For the former it should be emulsified in gum and syrup, for the latter in yolk of egg; and as occasion requires it may be combined with a small opiate, for example, compound tincture of camphor, or tincture of opium, in proper doses.

In *choleric form* or *summer diarrhœa*, the best results are obtained from castor oil guarded by a small dose of laudanum at the commencement, and repeated if the disease is severe; while astringents and opiates alone are withheld until the bowels are relieved of offensive materials, as in the later stages, the stools being copious and watery, griping and distension of the abdomen absent, and the tongue clean. Vomiting should be encouraged by copious draughts of warm water, and, if need be, by emetics of mustard or ipecacuanha. In children, when the motions are colourless, profuse, and incessant, it is best to give Hydrargyrum cum Cretâ in small doses every hour or two, and a very small enema of starch, containing acetate of lead or sulphate of copper with laudanum, which may be repeated if necessary. Remedies which arrest abnormal intestinal fermentation (such as salicylate of bismuth, naphthaline, calomel in small repeated doses, perchloride of mercury, potassio-mercuric iodide, carbolic acid, resorcin, salol, salicylate of sodium) are of great service in the treatment of infantile and summer diarrhœa. Dr. Luff prefers the following formula: \mathcal{R} Liquoris hydrargyri perchloridi $\text{m} \times \text{ij}$.; potassii iodidi gr. $\frac{3}{4}$; chloral hydratis gr. j.; aquæ ad \mathfrak{z} j.; to be given every four hours to infants up to six months, and the dose to be doubled for children of more than one year old. In the cold stage, there have been recommended mustard-baths (for twelve or fifteen minutes, several times a day); emetics (ipecacuanha 2 to 3 grains twice or three times in twenty-four hours); diffusible stimulants (ether in syrup every hour or half-hour). In the stage of reaction, saline aperients or calomel in small doses may be given; white of egg in water as a drink throughout; and, vomiting having ceased and diarrhœa being established, bismuth, chalk, or lime-water.

In *nervous diarrhœa* the first indication is to allay reflex excitability by the bromides; or, these failing, by opium. When diarrhœa is excited by food, the dose should be given shortly before meals. In *lienteric diarrhœa* arsenic is invaluable. Mal-digestion should be treated according to the indications (see DIGESTION, Disorders of). Occasional doses of castor oil—alone, or with bismuth or small doses of opium or henbane—are useful in clearing away fermentable matters. Astringents should only be prescribed after the failure of these or similar measures.

Morning diarrhœa is best treated by avoiding the ingestion of liquids during the late afternoon and evening, all fluids being taken during the early part of the day. The consumption of alcohol must be avoided or strictly limited.

In *vicarious diarrhœa* the skin should be made to act freely by warm baths, or hot-air or vapour-baths. In renal disease counter-irritation across the loins, digitalis, and nitrate of potassium may be likewise indicated. The diarrhœa should not be arrested or even checked unless it be profuse and exhausting, especially after restoring or augmenting the action of the skin and the kidneys; it is sometimes advisable to encourage it.

Diarrhœa from passive congestion of the portal vein is to be met by treating the cause, for example, disease of the heart, by digitalis, iron, and other remedies.

The flux of *chronic diarrhœa* (lienteric and the chronic tropical diarrhœa known as sprue or psilosis) cannot, as a rule, be stopped altogether by astringents only—the evacuations while thus retained may decompose, and induce flatulence and colic, or fever. The general health should be restored and anæmia removed; the secretions will then generally improve and the diarrhœa subside. Tonics—iron, arsenic, quinine, strychnine—may be aided by astringents—mineral acids, opium, bismuth, chalk, or hæmatoxylum. The best preparations of iron are iron-alum—3 to 5 grains, and liquor ferri pernitrat—10 to 40 minims. Ipecacuanha and taraxacum are useful when the skin and liver are inactive: from 1 to 3 grains of powdered ipecacuanha may be given night and morning. 2 or 3 minims of the official tincture of podophyllum three or four times a day is indicated when the motions are watery, pale, or high-coloured, and passed with severe cutting pains. Saline purgatives in the early morning have been recommended—2 drachms of sulphate of sodium, sulphate of magnesium, or soda tartarata on the first day; then 1 drachm for fourteen days, dissolved in a small bulk of water, with avoidance of fluids after the dose. The profuse sweating and colligative diarrhœa of hectic fever is best met by hæmatoxylum and diluted sulphuric acid, or opium with astringent mineral salts—nitrate of silver, sulphate of copper, or acetate of lead—given by the mouth or rectum.

Suppressed secretions, particular cachexiæ, disturbed innervation, congestion of the portal vein, and organic diseases of the intestines, form special indications for treatment when diarrhœa is present.

GEORGE OLIVER.

WILFRID EDGECOMBE.

DIATHESIS (*διαθήκη*, I dispose).—A morbid constitution, predisposing to the development of a particular disease. See CONSTITUTION, with which, in a somewhat more limited sense, this term is synonymous.

DIATHETIC DISEASES.—Constitutional diseases. See CONSTITUTIONAL DISEASES.

DICROTISM (*δῖς*, double; and *κρότος*, a stroke) is a term applied to the second great wave of the pulse. This dicrotic wave, or dicrotism, is due to a second expansion of the artery which occurs during the diastole of the ventricle. The *pulsus bisferiens* of the old authors was a pulse in which a second beat became perceptible to the finger: an occurrence observed occasionally as an antecedent of hæmorrhage, and also in the course of fevers. The second beat perceived by the finger is not always the true dicrotic wave, but may in some cases be an exaggerated tidal wave. This is the wave perceived in the high arterial tension sometimes antecedent to hæmorrhage. Dicrotism is favoured by a low state of arterial tension, by elasticity of the arterial coats, and by quick and strong ventricular contractions. It is central in its origin, and is a secondary wave of pressure produced in the blood-column by the elastic recoil of the aorta after its distension by the blood injected at each ventricular systole. The recoil of the aorta causes the

wave to spring towards the periphery from the closed aortic valves as a *point d'appui*. See PULSE.
B. WALTER FOSTER.

DIET.—**DEFINITION.**—The term 'diet' as here employed may be understood to express the regulation of food to the requirements of health and the treatment of disease.

GENERAL PRINCIPLES.—In order to properly sustain life, a diet must consist of a right apportionment of the following alimentary principles:—

1. Nitrogenous principles (proteids, &c.).
2. Non-nitrogenous principles (fats, carbohydrates, &c.).

3. Inorganic materials (saline matters and water).

Milk may be regarded as furnishing us with a typical dietetic representative of all these principles. The egg also holds a like position, and, as all the parts of the young animal are evolved from it, must needs comprise all the materials for the development and growth of the body.

The required principles are contained in food derived from both the animal and vegetable kingdoms, and the diet may be drawn from either; but, looking to man's general inclination and the conformation of his digestive apparatus, it may be assumed that a mixed diet is that which is best fitted for his subsistence.

The standard diet framed by Moleschott has been accepted as furnishing a model of what may be considered the requisite proportion of aliment for a person of average weight under exposure to a temperate climate, and during the performance of a moderate amount of muscular work. He gives the following weights of dried food-stuffs:—proteid, 4·5 oz.; fats 2·9 oz.; carbohydrates 14·2 oz.; salts 1 oz.

This, it will be seen, furnishes a supply of about 23 ounces of dry solid matter, of which one fifth is nitrogenous. If we reckon that ordinary food composed of bread and meat contains about 50 per cent. of water, then 23 ounces will correspond to 46 ounces of solid food in the condition in which it is consumed. To complete the alimentary ingesta, a further quantity of from 50 to 80 ounces of water may be assumed to be required to be taken daily under some form or other.

Looked at from the following point of view, it will be seen that an admixture of animal and vegetable food more economically supplies what is wanted than either kind taken alone, unless the adjustment should be made with the proper apportionment of fat as a representative of a non-nitrogenous article. It is estimated that for a man of medium weight, performing a moderate amount of work, about 300 grains of nitrogen and 4,800 grains of carbon are required to be introduced daily into the system with the food, to compensate for the outgoing of these elements that occurs. Now these amounts of the two elements are yielded approximately by 2 lbs. of bread and $\frac{3}{4}$ lb. of meat—that is, 44 ounces of solid food, of which about one fourth consists of animal matter. If the lean of meat only were consumed, rather over 6 lbs. would be needed to furnish the requisite amount of carbon, and there would be a very large surplus of unutilisable nitrogen; while if bread only were taken, the amount necessary to supply the requisite quantity of nitrogen would be rather more than 4 lbs., and this contains nearly double the amount of carbon wanted.

In order to preserve health it is necessary that a portion of the food consumed should be in the fresh state, and this applies to both animal and vegetable food. There may be no lack of quantity, and yet disease and death may be induced by inattention to this fact. See SCURVY.

Climate influences the demand for food, and instinct leads to the adaptation of diet to the requirements that exist. Not only is there a correspondence between the amount of food required and the inclination for taking it, but the nature of the food selected is in harmony with the requirements. The dwellers in the arctic regions, besides eating a large amount, consume much fatty food, which yields a large proportionate supply of heat on oxidation in the body. In the tropics, on the other hand, it is upon vegetable products, largely composed of carbohydrates, that the native inhabitants mainly subsist.

Labour necessitates a supply of food in proportion to the amount of work done. The employer finds that the appetite of a workman may be taken as a measure of capacity for work—in other words, that a falling off of the appetite means a diminished capacity for the performance of labour.

It was formerly thought that the energy required for muscular work could only be obtained from the ingestion of proteid food; but it is now known that carbohydrates and fatty food can be utilised for supplying the energy for muscular work, and that under ordinary circumstances the greater part of the energy required for muscular contraction is so supplied from the non-nitrogenous portion of the diet.

Persons who lead a sedentary and indoor life naturally require less food than those engaged in active work, and less should be consumed by them to prevent the system becoming clogged with effete products, which act perniciously in various ways upon the body. The food should also be largely constituted of non-nitrogenous principles, as these tax the excretory organs less than the nitrogenous.

The diet of *infants* is a branch of dietetics the importance of which can scarcely be overrated. The proper food during the first period of infancy is that which has been provided by Nature for the young of mammals—namely, milk. Up to about the eighth month the infant is designed to be sustained solely by its parent's milk. The teeth, which ordinarily begin to show themselves about this time, indicate that some solid food should now be given, and one of the farinaceous products is the most suitable with which to commence. Bread, baked flour, plain biscuit, or one of the numerous kinds of nursery-biscuits that are made, may with sterilised cow's milk be employed for a time as a substitute for the former food. By the ninth month the child should be entirely weaned. Custard-pudding and gravy or beef-juice are next added to the dietary. As the child advances through its second year, and the teeth become more developed, meat may be given. If the mother cannot suckle the child and a wet nurse cannot be obtained, cow's milk is the readiest and usual substitute. It should be diluted to twice its bulk with water, and to each pint of the milk and water about three-quarters of an ounce of lactose and the same quantity of lime-water should be added, and the mixture then boiled or efficiently sterilised by heat. After the first few weeks the infant should be fed only every three hours. Ass's

milk and diluted goat's milk not infrequently prove digestible when cow's milk does not; there are however many efficient methods of preparing the latter, thus in great measure obviating the necessity of obtaining milk from other sources. As the child grows the proportion of milk is gradually increased. Scrupulous cleanliness and regularity must be observed. Neglect of these precautions is the cause of much disease. See ATROPHY, GENERAL.

THERAPEUTICAL APPLICATIONS.—The application of the principles of dietetics may be successfully brought into use in the treatment of corpulency and thinness. A diet rich in nitrogenous matter, conjoined with exercise, promotes the growth of muscle, but the fat undergoes no increase. The conditions most conducive to an increased accumulation of fat are a diet rich in either fat or carbohydrates (provided the requisite amount of nitrogenous matter be present for affording what is wanted for the reparative operations of life), exposure to a warm atmosphere, and inactive habits. A supply of fat in a direct manner leads to an increased deposition of fat in the system, but the carbohydrates require in the first place to undergo assimilative change before they can be applied in the same direction. Notwithstanding this, it has been shown that carbohydrates are much more prone to lead to fatty accumulation than the fats themselves.

The details of the dietary to be prescribed where the aim is to produce increased stoutness and an improved condition of the body, should comprise such articles as fat meats, butter, cream, milk, cocoa, chocolate, bread, potatoes, farinaceous and sweet-puddings, oatmeal porridge, sugar and sweets, sweet wines, porter, stout, and ales.

The converse mode of dieting is necessary for reducing stoutness. See OBESITY.

The art of dietetics not only bears on the maintenance of health, but is capable of being turned to advantageous account as a therapeutic agency; and it is not too much to say that success in the treatment of disease is oftentimes dependent upon a display of judicious management in regard to food.

In the therapeutic application of dietetics the maxim should be held in view that, while the particular requirements are secured, there should otherwise be no greater deviation from what is natural than the special circumstances of the case demand.

The quantity of food consumed may require to be regulated as well as its nature. The quantity administered at a time should stand in relation to the power of digesting it; and to properly compensate for a diminished capacity for taking quantity there should be a corresponding increase in the frequency of administration.

In febrile, acute inflammatory, and other conditions where there is a failure of digestive power, the food administered should be such as not to tax the stomach, and should therefore consist of liquid materials. The available articles under such circumstances are beef-tea, mutton-, veal-, or chicken-broth, solution of milk-proteid, whey, calf's-foot and other kinds of jelly, arrowroot and similar farinaceous articles, barley-water, rice-mucilage, gum-water, fruit-jelly, and the juice of fruits, as of lemons, oranges, &c., made into drinks. Where a little latitude is allowable, the employment of milk and of eggs in a fluid form may be sanctioned. As circumstances permit, an advance may be made to

solid substances which do not throw much work on the stomach, as rice, sago, tapioca, bread- and custard-puddings, and stale bread or toast sopped. Next may be allowed fish, beginning with whiting. As power becomes restored, calves' feet, chicken, game, and butcher's meat—mutton to begin with—may be permitted to follow.

Directions concerning the diet in dyspepsia and other disorders of the alimentary tract will be found in the articles on DIGESTION, Disorders of; and STOMACH, Diseases of. The diet suitable in other diseases will be found in articles dealing with those subjects. F. W. PAVY.

DIETL'S CRISES.—See CRISIS.

DIGESTION, Disorders of.—The function of digestion is of a physico-chemical nature, being compounded of certain muscular acts, and of certain processes exercised by the digestive fluids on the ingesta, which are thereby converted into a fluid and diffusible state.

Any interference with the due performance of the several components of the function will lead to indigestion; and though it may for convenience be desirable to consider these disturbances separately, it must be remembered that the occurrence of one condition is apt to be quickly associated with another, and hence the forms of dyspepsia as they usually present themselves are of a complex nature, however simple the primary fault may have been. It appears to the writer to be very undesirable to restrict the application of the term *indigestion* or its synonym *dyspepsia* to the perverted actions of any one part of the alimentary system, such as the stomach, or to any one special morbid state, such as gastric or intestinal catarrh; but rather that it should include all departures from normal function which any part of the digestive tract may present. Nor is it possible to consider irregularities of digestion only from the point of view of the organs immediately concerned. Complicated as our organism is, disturbances of other functions will speedily make themselves felt in the one under consideration; and failures in the absorption of the digested food, or in its subsequent metabolic changes and elimination, will tell back sooner or later on that process which is, strictly speaking, limited to its preparation.

CAUSES OF DYSPEPSIA.—Dyspepsia may be immediately traced to (1) *the food*; (2) *disturbances of the so-called mechanical processes*—namely, the muscular acts, &c.; (3) *deficiencies in the chemical changes exercised by the digestive secretions*; (4) *abnormal microbic action in the intestinal contents*, or (5) *imperfect absorption of digested products*.

Certain of these causes included under groups 2, 3, and 5 are due to structural changes in the digestive organs, which may have been primary (e.g. ulcer, carcinoma), the dyspepsia then being symptomatic; or the changes in the organs may have been actually induced by the irritating effects of the chronically ill-performed digestive process; while in some cases the indigestion may be regarded as purely functional, the organs concerned being unaffected, at least at first.

1. **Imperfections of Food.**—Imperfections of food, whether in quality or quantity, are among the most frequent causes of digestive disorders.

(a) *Deficiency of food.*—Except under rare conditions, such as famine, this is not a common cause

of disease (see ALIMENT; DIET; and FASTING). But there are frequent occasions when, with no deficiency in the total bulk of food taken, there is yet a serious want in one or more of the needful constituents, and this is especially liable to occur in the case of children, often leading to disastrous results. See ATROPHY, GENERAL; and RICKETS.

The deficiency in food taken may result not so much from a defective supply of nutriment, as from a disinclination to eat, a common symptom of many diseases, especially febrile states; self-imposed fasting too frequent or prolonged; the anorexia of the hysterical temperament; or from obstruction to the entrance of food into the stomach from stricture of the œsophagus. Or the appetite may be impaired by over-indulgence in alcohol or tobacco. See APPETITE.

(b) *Excess of Food.*—There is very little doubt but that most individuals take more food than is actually required to restore the tissue-waste, many active lives being led on an amount far below what is ordinarily regarded as being necessary; and there is equally little doubt that much of what is taken is not in the most digestible form.

An habitual excess of food, at least in this country, usually errs in the disproportionate amount of nitrogenous matter it contains. Many of the substances resulting from the metabolism of nitrogenous matter are liable to become positive poisons in the economy, and the proper elimination of such materials is specially provided for by such organs as the kidneys and skin. The frequency with which these organs become the seat of disease suggests the probability of errors of diet being an important factor in determining the morbid changes, especially as considerable relief is often the result of a restriction of nitrogenous food.

The results of an excessive ingestion of food are as numerous as they are diverse. In many cases there does not seem to be either impairment of health or shortening of life. In some obesity and in others leanness ensues. In a large majority of individuals whose food is much in excess of their wants, particularly if the exercise taken be but little, there are variable symptoms of indigestion, such as a general feeling of lassitude and want of energy, a liability to headaches chiefly frontal, constipation, or more rarely diarrhoea, high-coloured urine depositing abundance of urates, a general disposition to sleep, various skin-eruptions, particularly acne, and not infrequently a feeble heart's action from the deposition of fat among its fibres. Any or all of these symptoms may exist, and may be relieved by a restricted diet. It is impossible to lay down any exact rules for the quantity of food that should be daily consumed: since, while the tendency is to take too much, age, season of year, and occupation are all circumstances determining variations both in quantity and kind.

(γ) *Improper Food.*—Setting aside those extreme cases of perverted appetite occasionally seen in the hysterical condition, there yet remains a very constant violation of the dietetic proprieties. These errors may be classed under the following heads:—
1. Substances which are indigestible, either from their natural composition or from their imperfect preparation. 2. Substances which, though digestible, are innutritious or even poisonous. In the first group are included such bodies as the pips and seeds as well as the skins and rinds of fruits,

the husks of corn and bran, the stalks and fibres of leaves, and gristle, elastic tissue, and hairs in animal food. For the reduction of these to a fluid and diffusible condition no chemical arrangement exists in the human organism, and they are voided very much in the same state as they are swallowed. Many articles of diet depend in great part for their digestibility on their proper preparation by such division and cooking. Thus most vegetables when taken in the raw state are but imperfectly digested, and such nutritious food as potatoes is when uncooked positively harmful. The apparent value of raw green vegetables, as lettuce, endive, cress, &c., would seem to depend on the peculiar condition of their mineral constituents, rather than on the vegetable tissues. Such substances as the above-mentioned are apt to produce perversions of digestion in virtue of the mechanical irritation to which they give rise, indicated by more or less pain of a griping character (colic), and frequently accompanied by diarrhoea. The constant ingestion of the more irritating of them may even set up a gastro-enteritis. Occasionally articles of food, such as brown bread and oatmeal porridge, are taken for the necessary aperient action they induce, owing to the irritating nature of the indigestible husks they contain. Symptoms of acute dyspepsia frequently follow the taking of meat enveloped in greasy sauces, since the fat, being undigested in the stomach, prevents the action of the gastric juice on the proteid matter, which then passes on into the intestines, setting up irritation like any indigestible substance. Very interesting are those articles of diet which, though easily digested and generally taken with impunity, may be poisonous to certain individuals. Such, for example, are mushrooms, eggs, shell-fish, or indeed any fish. Remarkable cases are authentically recorded of serious and even fatal results following their ingestion, although other persons have partaken of the same food with no ill results. The symptoms may be those of an acute gastro-enteritis; or, as is very frequently the case, an urticaria is the result, with or without swelling of the eyes and throat. Severe nervous prostration has been met with. The writer is acquainted with a gentleman who for many years was unable to remain in the room when fish of any kind was on the table, its presence inducing severe vomiting, abdominal pain, and general illness; and although the effects of merely smelling such food diminished after some years, very marked symptoms followed on partaking of any.

The most digestible and nutritious articles of food may determine indigestion when taken too hot or too cold, or if passed into the stomach without sufficient subdivision.

It is very desirable that the food consumed daily should be distributed over at least two or three meals, taken regularly at fairly equal intervals during the waking hours. Habit, occupation, and mode of life may compel various modifications, but frequent violation of an established routine is a common provocative of dyspepsia.

2. Irregularities of the Mechanism of Digestion.—The several stages of the entire process are mastication, deglutition, the churning movements of the stomach, the peristaltic action of the intestines, and defæcation. Each of these is liable to impairment, in the direction of increased activity or of deficiency (paralysis), due either to lesions, or to reflex stimulation, of the nerve-centres

whence the motor stimuli emanate, of the nerve-fibres by which these stimuli are conveyed, or of the muscular tissue by which the movements are performed. Not infrequently more than one of these tissues may be at fault. Interference with the movements may be caused by tumours, cicatrices, adhesions to adjacent structures, &c.

Irregularities of mastication, deglutition, and defæcation are fully considered under their respective headings.

(1) *Paralysis.*—Apart from structural affections of the intrinsic nervous mechanism of the gastrointestinal tract, of which, indeed, little is known, arrest of the peristaltic action of the gullet, stomach, or intestines is undoubtedly often associated with diseased conditions of the central nervous organs. Those lesions which interfere with the action of the vagus nerve, and remove its accelerating influence over the peristaltic movements of the stomach and less certainly of the intestines, have been regarded as most likely to bring about this condition; but stimulation of the splanchnic nerves, by which inhibitory impulses reach these viscera, will produce the same result. Arrest of the movements also follows stimulation of the central end of the divided vagus, or of any sensory nerve. The nervous exhaustion induced by long fasting, continued vomiting, previous violent peristalsis from purgatives, hysteria, and such diseases as chronic anæmia, prolonged fevers, also excessive brain-work, certain psychical states, and extremes of temperature, have been found to be accompanied by symptoms indicating loss of power of the muscular coat of the bowel. Possibly in these cases the result is due to arrest of the 'vagus influence.'

Paralysis of the stomach and intestines is a frequent result of affection of these organs themselves. Inflammation of the peritoneal or mucous coat often involves the muscular layer, and thus materially diminishes the power of the contractile tissue. Degenerations, especially amyloid degeneration, may invade the muscular coat, and interfere with the movements of the part. Over-distension from whatever cause will also enfeeble the contractile power of the muscular fibres of the stomach or intestine. The movements of the alimentary canal may be considerably lessened by the administration of certain drugs, such as opium, which diminish the motor excitability of the augmentor centres, or stimulate the inhibitory control.

The results of these various paralytic affections are in most cases sufficiently apparent. The palsied lips and cheeks and tongue tell their own tale by the half-opened mouth, the dribbling saliva, and the cheeks distended with food which cannot be kept between the teeth. When the fauces and pharynx are affected, the painful efforts at swallowing, the rejection of food through the nose, and the passage of food into the larynx, are signs not to be mistaken. Paralysis of the stomach and intestines is mainly recognised by the constipation from inability of the canal to propel its contents, and by symptoms of dyspepsia, such as flatulence, due to deficient gastric secretion from want of the requisite stimulation afforded by the churning of the stomach-contents, and to consequent delayed digestion, leading to abnormal fermentative changes. A paralytic condition of the sphincter ani will be indicated by an inability to retain the feces. It should not be forgotten that, normally, the gastric movements cease during sleep, or when the stomach is empty.

(2) *Excessive activity*.—Excessive activity of the muscular structures of the alimentary canal will be manifested by an increased peristalsis of the stomach and intestine, or by tonic spasms of limited regions. The former condition, by hurrying along the contents at the expense of their proper digestion and absorption, is an effective cause of diarrhoea. The latter, which is altogether abnormal and not a mere exaggeration of a healthy action, will, in proportion to its severity and extent, determine obstruction to the passage of the food. The spasmodic affections are almost always associated with pain—intestinal cramp, colic, tormina, &c.; while varying degrees of discomfort usually accompany exalted peristalsis.

The causes leading to these states may, as in paralysis, be referred either to the cerebro-spinal centres, or to the local neuro-muscular structures. (a) Excluding spasms of the muscles of mastication, deglutition, and defecation, which are elsewhere treated of, increased gastro-intestinal movements may follow on (i.) emotional states; (ii.) some structural lesions of brain and spinal cord, such as basilar meningitis and locomotor ataxy; (iii.) the increased excitability of the neuro-muscular apparatus caused by drugs—as strychnine and some toxins; (iv.) cutaneous irritation, as from cold, reflexly stimulating the accelerator nerves; affections of other viscera, especially uterine and ovarian, may act in a similar manner. While the first and last-mentioned causes favour increased peristalsis of the canal, the other conditions mentioned rather lead to tonic spasm. (b) Causes which may be regarded as exciting gastro-intestinal movements by their local effect may be grouped thus: (i.) irritation due to the character of the contents, such as their indigestible nature, acidity, extreme coldness or even excessive bulk, foreign bodies in the food, as pins, buttons, &c., irritant drugs, or worms; (ii.) drugs which appear to act directly on the gastro-intestinal plexuses and muscle-fibres, as lead, nicotine, &c.; (iii.) increased irritability of the mucous surface, as from inflammation or ulceration, favouring the action of the ordinary stimulants to contraction. Certain blood-states, such as that occurring in the gouty state, anæmia, or any other condition in which there is a deficiency of circulating oxygen and overcharge of carbonic acid, are liable to excite spasm or increased peristalsis, probably by acting on the nerve-centres concerned, as well as directly on the tissues of the canal itself. This result, however, is far from being constant, as very distinct inhibition of the intestinal movements is known to follow a condition of anæmia. Spasm of the pylorus may be induced by the attempted passage of solid lumps of food which are often forced back towards the cardia for solution or by an excessive acidity of the chyme. The normal contraction of the sphincter ani may give place to painful spasm, a condition which is very apt to complicate fissure and ulcer of the anus.

(3) *Mechanical difficulties*.—The due performance of the mechanism of digestion may be interfered with by alterations in the condition of the alimentary canal caused by various kinds of obstruction or dilatation. Thus deglutition may be rendered difficult or even impossible by a swollen tongue or tonsils, post-pharyngeal abscess, tumours of the œsophagus or larynx, or new-growths situated at the cardiac aperture of the stomach. The various obstructive diseases of the pylorus and intestines will obviously interfere with the proper passage of the contents, and

in those dilatations of the canal which are liable to develop above a stricture, the food accumulates and is delayed in its passage. The adhesion of coils of the bowels to each other or to adjacent structures is a further source of imperfect movement.

(4) *Diseases of the teeth*.—See MASTICATION; and TEETH.

3. Imperfections in the Chemical Changes.

Our knowledge of the normal chemistry of digestion, much as it has advanced of late, is still very far from complete.

The *saliva* may be increased in amount (see SALIVATION), this fluid being poor in solids and ferment. It may be difficult to trace any definite digestive troubles to this condition, since there is no evidence that the carbohydrates are insufficiently converted; but it is probable that the quantity of slightly alkaline fluid swallowed may interfere with the changes in the stomach. The general wasting which frequently follows on this state may be in part attributable to the anorexia which is so commonly associated with it. Nor can a diminution in the saliva, such as occurs in fever, be held responsible for any special dyspeptic symptom. Should the fluids of the mouth become acid, as from lactic or other fermentation of food therein, a stomatitis of varying severity may be induced, as is commonly seen in infants. Although an increased salivary flow is excited by many sapid articles of diet, the efficacy of the juice is much diminished or even destroyed by acid fluids, such as vinegar; and it is this circumstance that determines the cessation of salivary digestion after the food is swallowed.

Within the past few years considerable attention has been paid to the behaviour of the *gastric juice* in disease, and much valuable information has been obtained, mainly in reference to its acid constituents. See STOMACH, Examination of.

Notwithstanding that the normal *pancreatic juice* effects active digestive changes in the proteid, fatty, and carbohydrate constituents of the food, the results which follow its deficiency in quantity or quality cannot be indicated with precision: its effective working largely depends on the previous gastric digestion having been properly carried out. There is no doubt but that in all cases, when the pancreatic fluid is completely cut off from the duodenum, the digestion of fats is very considerably interfered with, as shown by the character of the stools, and the rapid emaciation of the patient; but this result is most marked when the bile is also wanting. In fever the secretion is said to be diminished.

An excessive secretion of *bile* is not clinically recognised except as the result of the administration of certain drugs, but a deficiency or complete absence is of common occurrence in those morbid states associated with obstruction of the bile-ducts, and, to a less degree, in fever. The dyspeptic symptoms directly referable to this state are specially due to the impaired digestion of fat, as evidenced by the stools. Abnormal fermentation, manifested by flatulence, may also take place. See FLATULENCE.

So little is known of the action of the *intestinal juice* in health, that nothing can with certainty be affirmed of the part it may play in disease. An excess of the secretion appears to occur, and to form the bulk of the fluid in the diarrhoea of cholera.

4. *Abnormal Microbic Action*.—In addition to the changes in the ingesta effected by the above-mentioned secretions, there is reason to believe that changes of a solvent character may also be brought

about by the action of *micro-organisms* in the stomach and intestines. Recent observations have demonstrated that numbers of fungi are normal inhabitants of different parts of the alimentary canal, and that certain species are peculiar to special regions. Introduced soon after birth, they propagate enormously, especially when digestion is impaired, being subject to great variation in number, and to some extent in kind, with the food taken. These microbes are said to play a minor part in ordinary digestion. How far such changes are normal is uncertain; but there is no doubt they may be very readily induced and very considerably extended—such as the butyric- and acetic-acid fermentations. In this way also may be formed the products of putrefactive decomposition—ptomaines and toxalbumens—often of a poisonous character, and mainly responsible for many remote dyspeptic symptoms; as well as accumulations of such gases as carbonic acid, hydrogen, sulphuretted hydrogen, and marsh-gas, productive of all degrees of flatulence. Delay in the propulsion of the gastro-intestinal contents is probably an important factor in the production of these gases; and still more so are the defective composition and quantity of the different digestive secretions, by which many toxic organisms are undoubtedly held in check.

There appears to be some reason to attribute some dyspeptic conditions to septic infection of the stomach and intestines by organisms developed in the mouth as the result of decayed teeth, dirty tooth-plates, &c.; and this would be more likely to occur, should the resisting power of these organs be diminished by a general state of malnutrition, which the indigestion might have determined or intensified.

5. Deficient Absorption of the Digested Products.—Conditions upon which an impaired absorption of the digesta may be assumed to depend are (i.) failure in converting the food into a diffusible state from inefficiency of the secretions; (ii.) impaired peristalsis, whereby the materials are not brought into sufficiently close contact with the mucous membrane; (iii.) deficient circulation of the blood and lymph in the chylipoietic area; (iv.) disease of the vessel-walls, impairing their permeability; and (v.) degeneration of the epithelium, with the same result.

SYMPTOMS OF DYSPEPSIA.—The almost numberless symptoms which indicate the perverted functions above described, whether primarily dependent on morbid structural changes in the organs concerned, or essentially due to the resulting mal-digestion, may be conveniently grouped into *local* and *remote*. Many so-called dyspeptic symptoms, however, are more properly referable to subsequent perversions of metabolism than to actual digestive imperfections.

The *local* symptoms—those, that is, which are connected directly with the affected structures—are, perversions of sensation; constipation or diarrhoea; vomiting; pyrosis and acidity; hæmatemesis; flatulence; eructation and foul breath; salivation or dry mouth; morbid states of the tongue; abnormal character of the stools. Excepting the first-mentioned, these receive detailed description in the present work under their respective headings.

Ordinarily we are unconscious of the process of digestion, but in disease it may be accompanied by alterations of sensation varying from a mere sense of weight and discomfort to severe spasmodic pain. Such sensations, however, are not constant, for definite

indigestion may exist without the patient complaining of any abdominal symptoms. Pain and probably other paræsthesiæ are immediately due either to a hypersensitiveness or actual injury of the mucous membrane or to muscular spasm; and it is desirable to differentiate as far as possible the cause in any given case. The ingestion of food may be followed by a feeling of abnormal repletion, or of emptiness with craving for food; or there may be heartburn, an ill-defined sense of burning felt in the epigastrium or over the chest or extending to the throat; or positive pain, or tenderness over some tolerably definite area. Sensations as of excessive movements of the bowels, of sinking, or of tightness across the abdomen, are of frequent occurrence.

Among the numerous *remote* symptoms are pain in shoulder, back, or limbs; headache—frontal, occipital, and vertical; vertigo and giddiness; muscæ volitantes, tinnitus aurium; cramps in the limbs; muscular weakness; palpitation, cardiac irregularity, flushings, and anginal attacks; cough and singultus; impaired appetite; all degrees of mental perversion, from irritability to apathy and hypochondriasis; drowsiness or insomnia; a peculiar sallown, muddy-looking skin; various cutaneous eruptions, chiefly papular; jaundice; and abnormalities of urine. The absorption of the products of abnormal digestion may even determine a condition of fever, so severe as to reach the typhoid state and end in coma. Such general perversions of nutrition as wasting and obesity may result from indigestion. Many of these symptoms may be regarded as reflex in causation, such as distal pains and cough, the afferent nerve-path for which is probably the vagus; others are doubtless to be attributed to actual want of proper materials for the nourishment of the tissues, owing to their improper preparation in the alimentary canal; and others again are possibly toxic in character, due to poisonous substances produced in the course of the perverted digestion.

The extreme diversity of these symptoms is remarkable: there is scarcely a function of the body that may not be implicated in the disturbance of digestion, and contribute its share to the total symptoms of any given case. The degree to which these manifestations may be present is, moreover, most varied: in one patient the malady may be mainly represented by a headache; in another the evidences of illness are most numerous. Again, the different ways in which these symptoms are associated in different cases are almost as many as the cases themselves. It is impossible, therefore, to attempt any description of a case of indigestion which shall have other than the most limited application; and since the symptoms set forth find their fuller explanation elsewhere in this work, the bare enumeration of them must suffice here. It is important to remember that many of the symptoms mentioned as indicative of disordered digestion are of not infrequent occurrence in the course of maladies quite distinct, or at least primarily so, from digestive diseases: headache, vomiting, cough, palpitation, &c., are illustrations of this. Therefore, although the recognition of the symptoms is not difficult, care and experience are needful to ascribe them to dyspepsia. But supposing that causes other than indigestion have been excluded, there still remains the task of referring the manifestations of disease which any case may present to their causal perversions in the digestive process: of determining, that

is to say, what may be the morbid change which has taken place in the secretions and consequent chemical processes, or the disturbances of motility, or maybe the error of diet, and, if possible, of inferring beyond these conditions the underlying structural lesions. The intimate interdependence of the various factors of digestion renders the discrimination of the primary fault from the symptoms present a matter of exceeding difficulty; and it is this which makes the accurate diagnosis of a case of dyspepsia so uncertain, and its treatment often so empirical. Many of the signs and symptoms, without doubt, indicate with tolerable certainty the region of the alimentary system which is at fault, and may even suggest the nature of the disease; but how far, and in what manner, morbid change in one part of the canal, or at one stage of the digestive process, may determine subsequent changes, and what the exact nature of these may be, and their relation to the symptoms manifested, are data at present almost unknown. The progressive character of the function of digestion—that is to say, the continuous series of stages, whereby the later ones are dependent on those preceding—renders affections of this system very different in their detection from those of other organs. Alterations in the gross anatomical characters of the digestive organs, such as a dilated stomach or an enlarged liver, may usually be ascertained with accuracy, and some symptoms may be reasonably asserted to follow from these conditions, while their relation to other morbid manifestations is often of doubtful inference. Modifications in the quantity or quality of the digestive secretions may be ascertained, but their connection with many of the symptoms which may be present is often most obscure. And again, there may be most persistent and serious disturbances of digestion, with severe discomfort to the patient, and it may be quite uncertain which organ is primarily at fault, and still more doubtful what the real morbid change is. So far as individual symptoms may be referred to definite diseases of the various digestive organs, this is attempted in the articles treating of the diseases of the stomach, intestines, liver, pancreas, &c., which are complementary to this section.

TREATMENT.—The essential basis for the successful treatment of indigestion is the recognition of the cause. Oftener perhaps than may be supposed this is a removable one; in every case it must be well sought for, and corrected if possible. A carefully regulated diet, both as regards ordinary food and special idiosyncrasies, is all-important, and the means from which much good is to be expected, both by way of prevention and cure. To lay down a requisite diet entails a general knowledge of the average composition of food-stuffs and of the changes which they normally undergo in the process of digestion, together with the nature of the influence exerted by various ingredients of the food upon the digestion of the others—e.g. ‘the retarding effect of tea and coffee on peptic digestion’ (Roberts). The relative digestibility of different articles of food, as estimated by the differences in time occupied by digestion in the stomach, is often an important point for consideration in the framing of a dietary for an impaired digestion. Whether or not any given article of diet should or should not be allowed must depend upon its digestibility in the given case, apart from what may be its value under healthy circumstances, and also upon what harmful by-products of digestion it may give rise to under the

diseased conditions which may be present. Exact information on several of these points is not yet available, and at the same time it is to be remembered that articles of food which normally are regarded as of the most digestible and innocent character may at times seriously disagree, while most indigestible articles may be taken with impunity. The writer is convinced that, while fully recognising the great importance of diet, it is nevertheless the case that not a few dyspeptics may trace their ailment to a long-continued adhesion to a strict diet which might theoretically fulfil all physiological requirements, and that many cases of indigestion may be considerably benefited by a laxity which might not always be justified on physiological grounds. As useful adjuncts to dietetic treatment are the various artificial partially or wholly digested food-stuffs, such as maltines, peptones, and emulsions of different kinds. Such details of general hygiene as exercise, bathing, occupation—mental and physical—change of scene and air, require attention scarcely, if at all, secondary to diet. Undoubtedly, much may be done with drugs, both in relieving the symptoms and in treating the conditions on which the symptoms may depend. The latter are met by such means as abstinence from food for a certain period, with consequent rest to the organs concerned, the requisite nutriment being supplied *per rectum*; by artificially digested aliment, thus helping the digestive juices; and by supplying the elements of the secretions—acids, alkalis, and ferments, when there is reason to suppose they may be deficient. Irregularities of movements of the canal may be remedied by such drugs as strychnine, nuxvomica, belladonna, and opium, or more active aperients or astringents. Pain and other sensory disturbances of the stomach or intestines may be relieved by alkalis, salts of bismuth, hydrocyanic acid, morphine, or opium. Among the long list of drugs of which the value is assured in different cases of dyspepsia are arsenic, iron, the vegetable bitters, silver, creosote, charcoal, valerian, the thiosulphates, and the various carminatives. *See* CARMINATIVES.

W. H. ALLCHIN.

DILUENTS (*diluo*, I wash or dilute).

DEFINITION.—Remedies which increase the proportion of fluid in the blood.

ENUMERATION.—Water is the only real diluent. It is given for this purpose in various forms—soups, *ptisanes*, barley-water, toast and water, milk, lemonade, aerated waters, &c.—to quench thirst, and increase secretion.

USES.—Dilutents are employed to lessen thirst, as in fever and diabetes, and to remove the products of tissue-waste. As the thirst may depend upon local dryness of the throat, as well as upon general want of fluid in the system, the power of water to quench thirst may be greatly increased by adding to it a little vegetable or mineral acid, or some aromatic, such as lemon or orange peel, which will stimulate the flow of saliva, and thus tend to keep the mouth moist after the liquid itself has been swallowed. The thirst-quenching power of water is also aided by the addition of mucilaginous substances, such as oatmeal, or linseed tea, which leaving a mucilaginous coat on the inside of the mouth and pharynx, retard evaporation, and thus lessen the dryness of the mucous membrane. The free use of water, and especially drinking hot water on rising in the morning, or on going to bed at night, and between

meals, is useful in gout, chronic rheumatism, and biliary lithiasis, lessening or preventing the occurrence of acute attacks.

T. LAUDER BRUNTON.

DIPHTHERIA (*διφθερία*, a skin).—SYNON. : Fr. *Diphthérie*; *Diphthérie*; Ger. *Diphtheritis*; *Diphtherie*.

DEFINITION.—An acute, specific, infective, and often epidemic, general disease; accompanied by pyrexia and great weakness; having as its local manifestation inflammation of various mucous membranes, particularly those of the throat, nose, and larynx, and the formation upon them, or upon external wounds, of a fibrinous exudation; and often followed by paralysis of varied distribution. The symptoms are due to the vital activity of a specific micro-organism, usually known as the Klebs-Löffler bacillus.

The period of incubation, though of variable length, is commonly from two to four days. Undoubted cases are on record, however, in which less than twenty-four hours elapsed between exposure to infection and the commencement of symptoms. The incubation is usually short when an open wound becomes infected, and when a large amount of infective material is received directly on to a mucous surface, as when a patient coughs into the open mouth of his attendant, or in those lamentable instances in which a person has been rash enough to suck a tracheotomy wound, in the hopes of clearing the obstruction.

A difficulty in determining the period of incubation is caused by the invasion of the disease being not infrequently insidious. During the incubation, while there may be no symptoms at all, there may be indefinite feelings of malaise, not to be distinguished from the early symptoms of invasion in a mild attack.

There is reason to believe that the more intense the poison, or the more susceptible the patient, the shorter is the period of incubation likely to be.

SYMPTOMS.—*Invasion.*—According to the severity of the disease in its clinical aspects, three separate types of diphtheria may be distinguished—the benign, the ordinary, and the malignant. These are not natural orders, but relative terms. Numerous cases occur which throughout their course may be classified as belonging to one or other of these groups. Yet, on the other hand, instances are also numerous which show a transition from one to the other group, which, beginning, for example, after the benign type, afterwards show the most serious symptoms or sequelæ of the disease; or which, presenting only ordinary symptoms at the onset, rapidly assume the features of the malignant type.

1. It will be convenient to describe in the first place the symptoms of the disease as it is *ordinarily* met with.

In such cases, after a period of incubation, the patient, as a rule gradually, but often almost suddenly, feels ill. He has wandering aches and pains, disinclination for exertion, slight headache, chilliness, loss of appetite, nausea—and rarely, even vomiting, thirst, and the general discomfort of a febrile condition. The temperature of the body is raised, but not to a great degree—in most cases, at the onset, to not more than 100° or 101° F. At this time, however, the local symptoms of the disease appear in the form of soreness and tender-

ness of the throat, and slight enlargement of the glands at the angles of the jaws. The fauces, tonsils, soft palate, and pharynx are swollen, and show a diffuse redness. The pulse and the respiration are hurried, in accordance with the general febrile state. The urine presents the usual febrile condition.

The symptoms rapidly increase in severity. The bodily weakness becomes extreme, and anæmia soon presents itself. The glands, not only those at the angles of the jaws, but their lymphatic connections also, become greatly swollen, painful, and tender; and their covering of skin may be reddened. The pulse is frequent, feeble, and of low arterial tension; the respirations are hurried. The local disorder now becomes characteristic. The tonsils themselves assume a paler aspect, but are surrounded by the reddened mucous membrane. Soon a white haziness appears in patches on the tonsils, the soft palate, or the pharynx. While the swelling of the parts already mentioned greatly increases, the haziness becomes more pronounced, until in disseminated patches a distinct false membrane is seen, slightly raised from the surrounding parts, yellowish-white in colour, ragged in outline, and surrounded by a zone of congestion. At first the membrane can be detached from the underlying parts; and when removed it leaves an intensely red surface behind. But very soon it becomes more adherent, and its removal is only attained by laceration of the mucous membrane and the exposure of a bleeding ulcerated surface, on which a new layer of false membrane is formed in a short time. The membrane spreads, and may cover the whole of the fauces and pharynx. When this occurs there is usually so much swelling of the parts that scarcely any opening of the fauces can be seen. Further, the aspect of the membrane changes. From being yellowish-white, thin, and delicate, it becomes thicker, of a brownish tint, firm and leathery in texture. The difference in colour is due to admixture with decomposed blood, the entanglement of atmospheric particles, and drying of the exudation. Signs of coryza are present, and from the nostrils a thin acrid discharge issues.

The tongue is dry, coated with a white pasty fur, and red at the tip. Appetite is entirely lost, and as a rare symptom there may be vomiting. Swallowing is difficult and painful. The bowels are, as a rule, constipated. The temperature becomes higher, but does not attain the extreme degree seen in many other fevers. Its ordinary height is between 102° and 103° F., but it usually declines rapidly on the adoption of efficient local treatment. The examination of the chest at this time, provided the case is uncomplicated, reveals nothing abnormal. The urine is still febrile, and in a large number of cases contains albumen. Slight delirium may occur, but is not a frequent symptom in the ordinary type.

When such cases tend to a favourable issue, the duration of the disease is variable. Even after such a moderately severe form as that just described, the symptoms may begin to decline at about the fourth day; the patches of false membrane, after separating, cease to re-form; and the patient rapidly resumes his ordinary health. Usually, however, both the general and the local symptoms are prolonged for a much longer period, say up to the twelfth or fourteenth day, when slow convalescence begins. Should, however, the case have been treated with adequate doses of antitoxic serum, an arrest

of the disease, if not a decided improvement, is usually to be seen within twenty-four to thirty-six hours of the commencement of the treatment.

Such is a description of the form in which diphtheria ordinarily presents itself, without any of the numerous complications which may occur. Many of the symptoms, however, require a more detailed description, and this will be given below.

2. The *benign* form is so mild that a considerable number of patients pass through it with so little feeling of illness that they consider it unnecessary to consult a medical man at all. There can be no doubt that such ambulant cases are important factors in spreading the disease. The patient suffers from but slight malaise and weakness, with loss of appetite and slight fever. The urine is usually not albuminous. No complaint whatever may be made of soreness of throat; and for this reason the nature of the complaint is frequently overlooked, even by medical men. Generally, however, there is some soreness in swallowing, and a little enlargement of the glands at the angles of the jaws. On examination, a few white patches of false membrane, easily detached, will be seen on the tonsils, rarely on the soft palate or pharynx. Very rarely, with only mild general symptoms, extensive deposit may be seen in the throat. In a few days the patches disappear, either by being coughed out or by gradual dissolution; no more form; the symptoms, slight as they were, decline; and the patient rapidly resumes his usual health. During the whole period of the disease it is possible for the patient to walk about, and even attend to his usual duties. But this is very undesirable, because such cases are capable of spreading infection.

3. The *malignant* form of diphtheria is characterised by severity both of the general symptoms and of the local lesion. Commonly, from the first the symptoms are severe, though the early features of the case may be those of the ordinary or even of the benign form. There is extreme prostration; the pulse is rapid, feeble, and of low tension; and the respirations are hurried and shallow. The skin is dry, pungent to the touch, and of a generally dusky hue. The tongue is dry and brown. Very soon the 'typhoid' state develops, and there may be sometimes a muttering delirium, though in most cases this is absent. A tendency to hæmorrhage is a frequent phenomenon in this condition, and is shown by the occurrence of hæmorrhages from various mucous membranes, or beneath the conjunctiva, and by the appearance of a purpuric rash on the skin. The temperature may be highly febrile, or not unduly raised. In some of the worst cases, except at the outset, it is subnormal. The urine is albuminous. The false membrane at first is tough and fibrous, sometimes gelatinous, but later it is soft, putrescent, and very dark—even black, in colour. It is often extensive; and the tissues around are sloughing or gangrenous. A foul smell comes from the putrefying mass at the back of the throat. In most cases, too, the false membrane spreads to the nose; and a sanious discharge, also of foul smell, issues from the nostrils, and by its irritation excoriates the skin around, with which it comes into contact. The glands of the neck are always much enlarged and inflamed. Such a condition in most cases ends fatally in a short time.

This general description of the three types of diphtheria requires to be supplemented by a detailed account of some of the symptoms.

Formation and Spread of the False Membrane.—The presence of the false membrane is the characteristic feature of diphtheria, and that from which it derives its name. It is the special local manifestation of the disease. In the benign form we very occasionally find the general symptoms very slight, or practically absent, while a quantity of membrane may be seen in the throat. The question then arises whether we may occasionally meet with general symptoms of diphtheritic fever, not only without signs of any local lesion, but without the appearance of any false membrane, and with merely congestion and swelling of the fauces, having no specialised character. It is in the experience of clinicians that this occurs, but certainly very rarely. It is possible that, in the course of an epidemic of diphtheria, instances of the benign form may be met with, without more local lesion than the early congestion described; yet in such cases a slight formation of false membrane may easily be overlooked. Because of the existence of such a condition, Virchow originally described a catarrhal form of diphtheria in which no membrane at all was formed. But it has been stated that the poison of diphtheria may, in extremely rare cases, act so powerfully upon the system as to kill the patient before the local lesion has time to develop. It is only when such cases as these are really authenticated that they can without fear of error be used for argument; and, since they have been recorded by competent observers, it must be admitted that diphtheria may at times occur without any specific local lesion being detected.

In by far the majority of cases, however, false membrane does appear. Generally it occurs first on some part of the throat—either on the tonsils, soft palate, or the back of the pharynx. Usually, too, it is seen in one or more spots simultaneously on both sides, and most commonly on the tonsils. Yet, occasionally, at an early period only one side may be attacked, but soon, however, an affection of the opposite side follows. The membrane can, at quite its early appearance, be detached with only slight difficulty, leaving a very red or hæmorrhagic surface behind. But very soon its connections with the subjacent parts become much firmer; it can be removed only by some force, and leaves behind a rough, bleeding, and ulcerated surface. In all only extremely slight cases, or in those treated with antitoxin, a new formation of membrane then occurs; and this may be repeated many times if the disease continues. The detachment of membrane also takes place naturally, not only at the termination of the case, but during its height, new membranes being again formed. In this way, the patient may expel from the mouth, usually by coughing, large quantities of the false membrane, in small or large shreds. Where the larynx and trachea are attacked, complete casts of these cavities may be thus expectorated; and in still more serious cases even moulds of the smaller bronchi are occasionally coughed up.

In the early stages, the membrane appears as a thin whitish deposit, which rapidly thickens. The colour, too, soon changes, passing through stages of grey, greyish-yellow, yellow, brown, and even black. Its consistence increases, becoming in most cases very firm, and resembling shreds of kid-leather. In the malignant form, the membrane eventually becomes softened, and even putrescent. Its colour then is still darker, and the membrane becomes mixed with blood or the products of its decomposition.

The isolated patches in which the membrane first appears soon spread and amalgamate, until the whole of the throat, as seen on opening the mouth, is entirely covered. But the affection spreads farther. One of the most dangerous complications of diphtheria, and also one of the most frequent, is the *spread of the membrane to the larynx*. This is more frequent in children; and, while dangerous in adults, is still more so in children, because of the greater ease with which it obstructs the lumen of the glottis. The signs by which the complication may be diagnosed are the following. The patient feels soreness in the larynx, and is greatly troubled by cough. The breathing becomes first wheezy, then stridulous, and always more hurried than before. The cough is harsh; the voice is hoarse and husky, and finally lost. The patient, if a child, clutches at the throat as if to remove an obstruction. Soon the narrowing of the larynx interferes greatly with the entrance of air into the lungs. All the extraordinary muscles of inspiration are brought into action. The lower ribs cease to move outwards, and later on are depressed at each inspiration. The supra-clavicular spaces are similarly depressed. These signs are more obvious in children: in very young subjects, and especially in those affected by rickets, the whole chest-wall may be depressed as each breath is drawn. Signs of cyanosis may now be observed in blueness of the lips and cheeks; but more commonly the face is pallid. The pulse is very rapid. The temperature is no more raised than it was before, and may indeed be only slightly above the normal. The forehead, and even the whole body, becomes covered with a cold clammy sweat. If unrelieved by operation, this condition soon ends in death, occasionally preceded, in young subjects, by convulsions.

The opinions of observers of great experience vary as to the period of the disease at which extension of membrane from the pharynx to the trachea is most likely to occur. Sir William Jenner stated that he had never known this complication commence after the expiration of the first week of the disease, and this is correct for the majority of cases; and we may place the usual time of appearance of secondary laryngeal symptoms at three to six days after the onset of the diphtheria. It is rather in the mild and in the ordinary forms than in the malignant that this complication may be looked for. Yet its appearance is a sign of extreme danger; since a large proportion of the deaths from diphtheria are due to this cause. Death usually occurs from one to five days after the onset of laryngeal symptoms unless the obstruction be relieved.

The membrane frequently spreads to the *nares* also. This is almost invariably the case in the malignant, but occurs, too, in the ordinary form. The symptoms of the complication are snoring or snuffling respiration, and the discharge of an acrid fluid from the nostrils, which excoriates the surrounding skin. The excoriations may in their turn become covered by diphtheritic membrane when the disease is severe. Shreds of membrane, too, may be discharged from the nostrils. Epistaxis is of frequent occurrence. Sometimes, as stated in the account of malignant diphtheria, this is due to the general tendency to hæmorrhage found in septic diseases, but it is also a sign of invasion of the nasal mucous membrane. From the nose the lachrymal ducts, and even the conjunctivæ, may be infected, leading occasionally to destruction of the eyeball; but this is excessively rare.

Spreading from the throat, the membranous

formation easily reaches the upper pharynx, and by way of this proceeds to the middle ear. Great pain and sense of distension are thereby produced, and severe *otitis media* with destruction of the parts may result. Membranous extension along the œsophagus, even to the cardiac end of the stomach, has been recorded.

Sometimes false membrane may appear in various parts of the mouth, probably by the infection of abrasions of surface.

In most cases, as above described, the membrane makes its first appearance in the throat. Yet numerous cases are now on record in which it has first been seen elsewhere, as on the surface of wounds and abrasions of the skin.

The *primary laryngeal form* of the disease is well recognised, and has been known by the name of Diphtheritic Croup. The general symptoms of the disease are then the same as in the ordinary form, but signs of the trouble in the larynx occur early and before any affection of the pharynx can be detected. With the laryngoscope, if the patient will allow of it being used, false membrane, having the ordinary appearance, can be seen covering the posterior surface of the epiglottis, the whole of the interior of the larynx, and even of the trachea. The affection may remain localised in the larynx until death, or may extend thence to the trachea and bronchi, or to the pharynx and other parts.

A *primary nasal form* is rare, but sometimes met with. Here the coryzal symptoms and the mucopurulent or sanious discharge from the nostrils occur early in the disease; and, when the affection is advanced, false membrane may be seen near the anterior nares. Purely nasal attacks are usually very mild. From the posterior nares the lesion may spread to the pharynx, as in the laryngeal form. Such cases are often overlooked in their early stages, because of the obscure position of the characteristic membrane. They are generally severe in character.

External wounds or abrasions may be secondarily infected and covered by false membrane in the course of the ordinary disease. But, on the other hand, there happen cases in which not only has the first local appearance of the disease been seen in an external wound, but the inoculation of such a wound has been the means of infecting the general system. Thus diphtheria has originated from the use of an infected instrument in the operation of circumcision, diphtheritic membrane first developing on the wound. Diphtheria of the vulva is sometimes seen.

Affection of the Glands.—The glands beneath the jaw are swollen and tender in all but very exceptional cases of diphtheria. In most they are so large as to be easily seen; in nearly all they can be felt. But in by far the larger majority of cases the swelling of the glands is a very marked feature. In severe cases, too, it is not only the glands beneath the jaw, but also all those of the neck, on both sides, though the affection of one side may predominate. The skin covering them is occasionally swollen, reddened, and œdematous. In which case, too, the tissue lying between the glands is greatly inflamed; and in a few cases a brawny hardness, extending from the jaw to the clavicle and involving apparently all the tissues of the neck, an *angina Ludovici*, has been observed. Rarely, the glandular inflammation may proceed to suppuration, causing a large and formidable abscess. The affection of the glands and surrounding tissue is more marked in the severe, and especially in the malignant forms.

Condition of the Urine.—The urine in diphtheria resembles in its general characters that of other febrile diseases, though the features are less marked than in other affections. Albuminuria is very frequent, and is found, according to statistics, in one-half of well-marked attacks. The amount of albumen may be only slight, or, on the other hand, very great; and it may disappear from day to day, to again return. With it may be found in the urine hyaline or epithelial casts, but only very rarely blood; and the occurrence of albuminuria is in only extremely exceptional cases accompanied by anasarca or followed by any uremic symptoms. Whether albuminuria is present or not, the daily excretion of urea is normal, or increased. It differs markedly from the albuminuria of scarlet fever in making its appearance nearly always during the acute course of the disease: very seldom at the time of convalescence. In most cases it appears before the end of the first week. As a general rule, the greater the quantity of albumen in the urine, and the earlier its appearance, the more severe is the case; but the mere presence of albuminuria does not affect the prognosis of the diphtheria. In most cases the albumen disappears during convalescence; though, as the writers can confirm, it may continue in the urine for months in moderate amount, and unaccompanied by other symptoms, beyond slight weakness and anæmia. On the other hand, the writers have reason to believe that in exceptional cases chronic nephritis may have its origin in an attack of diphtheria.

Temperature.—The temperature in diphtheria has no definite course. It rises suddenly early in the disease, and as a rule continues, with morning remissions, until the beginning of convalescence, when it declines rather rapidly. The average temperature at the height of the attack is from 102° to 103° , and very rarely does it exceed 104° F. It may become raised above the average limit by the advent of complications, such as broncho-pneumonia or suppuration in the neck; but, with these exceptions, the height of the temperature is no certain guide to the severity of the general disease or to the extent of the local lesion.

The Pulse and Heart.—The pulse in severe diphtheria is throughout of an asthenic character, short, ill-sustained, and of low tension. In the majority of cases it is frequent, above the degree to be expected from the fever. Sidney Phillips analysed 100 cases of diphtheria with reference to the states of the circulation which may occur. In three per cent. of his cases the pulse was unduly slow, and these instances were characterised by vomiting and by a fatal issue. He concludes that a very rapid pulse, also a very dangerous symptom, may occur without any other phenomenon, but may be accompanied by vomiting and extreme drowsiness. Irregularity both in the rhythm and the force of the pulse is not uncommon and not necessarily unfavourable. The heart-sounds are feeble, and the first sound short. Disturbances of the intervals between the sounds are, in the opinion of the writers, of great importance. A rapid sequence of the second sound upon the first, but especially 'spacing' of the sounds, as heard at the apex of the heart—that is, an equalling of the intervals between the first and second, and the second and succeeding first sound respectively—is of most serious import, and indicates a liability to sudden cardiac failure, the most dangerous complication of diphtheria.

These alterations in the sounds of the heart may be detected both at the height of the fever and during convalescence, and are equally important at either time. Acute dilatation of the heart, usually of the right ventricle, sometimes of the left, is not infrequent in diphtheria. It may be accompanied by a systolic murmur, heard at the apex or over the area of the pulmonary artery. But such a murmur is not due to endocarditis. The cardiac dilatation does not occasion dropsy, as it does in most other conditions, but the weakness of the heart's impulse is the cause of the subnormal temperature and coldness of the extremities and of the thrombosis of the large veins which sometimes, though rarely, occurs.

Rash.—A certain degree of efflorescence on the skin is not uncommon in diphtheria, and especially in the severe variety. It is usually seen about the neck and the upper part of the chest. It is a diffuse redness, not punctate, and not papular in the slightest degree. This is not a specific rash, and must be carefully distinguished from that of scarlet fever. In the malignant forms purpuric spots of different sizes may be observed, scattered over the whole surface of the body in great numbers.

COMPLICATIONS AND SEQUELÆ.—Many of the complications have already been described in the foregoing account of the disorder, and such will be here only enumerated.

1. Extension of the false membrane to the larynx and lower air-passages, to the nose, ear, and eye.

2. Formation of false membrane on external wounds and abrasions.

3. Extensive ulceration, and even gangrene, of the tissues of the throat.

4. Great inflammation in the neck, and rarely suppuration.

5. Albuminuria.

6. Dilatation of the heart.

7. Cardiac failure, syncope, and sudden death. This complication is the most dangerous which can occur in diphtheria, and may come on at any period of the disease, and even during convalescence. It is preceded usually by a very frequent, sometimes by a slow, pulse, but its special warning is an alteration in the sounds of the heart, as described above. The cardiac failure usually shows itself by a gradual failure of the circulation, very rarely by sudden syncope, and is nearly always fatal. The most distressing cases in which this occurs are those in which the patient, some time after the decline of pyrexia, is in full convalescence, or even in apparently good health, yet suddenly faints and almost instantly dies. In the majority of cases, however, the heart fails gradually and death occurs in two or three days, during which vomiting, pallor, subnormal temperature, sighing respiration, and anæmia gradually supervene. The mental faculties are retained to the end, which is usually quite sudden. Cardiac failure rarely occurs except in severe attacks, and usually comes on between the tenth and fourteenth day, but no patient is absolutely safe from this fatal mishap until five or six weeks after the apparent end of the disease.

8. Hæmorrhages. These may occur, not only from the ulcerated mucous membranes, but also from any mucous surface. They may be so copious as seriously to weaken the patient, and are evidence of a malignant attack.

9. Thrombosis of veins.

10. Affections of the bronchi and lungs.

General bronchitis may be met with in diphtheria before the larynx is attacked, and may be followed by extensive broncho-pneumonia. Both these complications are, however, more frequent when the air-passages become involved in the special diphtheritic inflammation. Collapse of lung, emphysema, and hæmorrhage into the tissues of the lung are also met with at times. The special symptoms characteristic of these conditions are added to those of the original complaint, and need not be here detailed.

11. Paralysis. Statistical evidence shows that this most characteristic complication occurs in about 20 per cent. of cases. The paralysis as a rule appears about two or three weeks after the decline of the pyrexia, but is often earlier, and it may be delayed until the fourth or fifth week of the attack, and, in rare cases, even a week or two later. As Landouzy has shown, the older the patient the more liable he is to this sequela. It is far more common after severe cases of diphtheria, and its onset in such is usually earlier.

The first part to be affected by the paralysis is commonly the soft palate. The patient then speaks with a nasal voice, and is unable to swallow fluids, since the immobile palate no longer closes the entrance to the posterior nares, and so fluids regurgitate through the nose. The palate hangs motionless during respiration and phonation, and is insensitive to touch. Occasionally, and for a time only, one side of the palate remains unaffected, and then the uvula is drawn to one side and the curves of the palatal edges are unequal. In rare cases the inability to swallow extends to solids also, from paralysis of the muscles of the pharynx.

The next most common paralysis is that of the eye. The most obvious effect of this paralysis, and that which most readily attracts the attention of the patient's friends, is strabismus from paralysis of one of the rectus muscles. But this is not the most common result. More frequently we meet with a paralysis of the ciliary muscle, usually in both eyes, which has as its effect a loss of the power of accommodation. Unless the patient has previously been myopic, he is now unable to adjust his eye for near objects, as, for instance, in the act of reading, while vision for distant objects remains unaltered. The pupil, too, is occasionally sluggish in contracting to light. Other muscles of the eyeball may suffer, so that various forms of squint are met with, and sometimes partial ptosis; while, in exceptional cases, one or even both eyes have become absolutely motionless from paralysis of all their muscles. Concentric contraction of the fields of vision has been described.

Other organs of sense suffer but rarely, but Gowers states that he has met with loss of taste, and loss of smell without loss of taste.

The limbs next suffer from the paralysis, and usually the lower before the upper limbs. The paralysis is generally preceded by tingling feelings, sense of 'pins and needles,' or actual pain, in the parts about to be attacked. Then gradually and slowly the paralysis sets in. It is but rarely complete, but always sufficient to render the muscles practically useless. The muscles are flaccid and soon begin to atrophy. The knee-jerks are lost; but this phenomenon may be met with early in the primary disease; and there may have been a previous exaggeration of the knee-jerks. All reflexes are abolished. The affected muscles after a

time cannot be stimulated to contraction by the faradic current, though they still retain their reaction to galvanism. Together with the paralysis, and sometimes without it, a certain amount of ataxy is not infrequently seen, most obvious in the legs, though also sometimes found in the arms.

Sensation, in one or all of its varieties, is usually affected to a greater or less extent, but not always in proportion to the loss of muscular power. The distribution of the anæsthesia varies. It may be spread over the whole limb, or appear only in patches. Paræsthesia is usually most marked in the fingers and toes, in which it often takes the form of 'pins and needles.'

These affections of the limbs are usually bilateral, though not necessarily equal on the two sides.

There is scarcely a part of the body which may not be affected by the paralysis, but other parts than those mentioned are comparatively rarely attacked, and only in severe cases. The intercostal muscles may be paralysed, and less frequently the diaphragm, so that respiration becomes difficult or even impossible. The head falls forward, if the patient is in the upright position, from weakness of the neck-muscles. The muscles of the larynx are sometimes attacked. The muscles attached to the epiglottis cease to close the orifice of the larynx, and food may then reach the air-passages. More rarely, the muscles of the vocal cords themselves are weakened or paralysed, and the voice is altered in quality or entirely lost. The bladder and rectum may be affected in severe cases, but generally they are unaffected. Among the muscles most rarely attacked are those of the face and tongue.

In the most severe cases it is possible for nearly all the muscles above mentioned to be paralysed at the same time, so that the patient lies in bed utterly helpless. This condition, however, is extremely rare. More usually the paralysis recovers in one part while it progresses in another, and as a rule does not recur in a part it has already left. A patient the subject of diphtheritic paralysis is in some danger of heart-failure, for this complication, though usually arising earlier, occasionally occurs during paralysis. The nutrition of the body may fail from the inability to swallow food; and from food entering the larynx pneumonia may arise. In very rare cases death ensues from paralysis of the respiratory muscles. Yet all these dangers are comparatively rare; and usually the paralysis, however severe, after a time gradually and entirely disappears. According to Gowers, the average duration of the paralysis is from six to eight weeks in cases that recover, but it remains longest in the limbs, and may there last for as long as four or six months, in exceptional instances.

TERMINATIONS.—(1) *Resolution.*—When the attack ends favourably the fever declines by lysis; the membranes soften, separate, and do not reappear; the patient gradually recovers strength, and resumes his ordinary health. The duration of ordinary cases is about ten to fourteen days, but the attack may be prolonged by recrudescences to a much longer period.

(2) *Death.*—Death occurs in diphtheria (a) by rapid anæsthesia from excessive effect of the poison; (b) by cardiac failure; (c) by asphyxia from spread of the membrane to the larynx, or paralysis of the respiratory muscles; or (d) by any of the complications enumerated above.

ANATOMICAL CHARACTERS.—*False Membrane.* The naked-eye appearances of the false membrane have already been sufficiently described. When a section of the mucous membrane covered by the exudation is examined under the microscope, the false membrane is seen to consist of either threads or shapeless masses of fibrin, in which are embedded leucocytes, more or less degenerated, red blood-corpuscles, and shrivelled cell-nuclei. In the deeper layers of the false membrane the outlines of epithelial cells can be seen, showing their bodies swollen into a hyaline or granular mass; the nuclei are not visible; and these cells no longer easily take the ordinary stains. No definite line of demarcation between the false membrane and the true mucous membrane can be seen; the one joins the other gradually and irregularly. The mucous membrane shows swelling and proliferation of its epithelial cells, and is infiltrated with leucocytes. Its blood-vessels are congested, sometimes thrombosed, and hemorrhages are seen in the tissue. Numerous micro-organisms are found, not only in the false membrane, but in the tissue of the mucous membrane, its veins and lymphatics. In the later stages further degenerative changes are found in the false membrane. It is then much more granular, as seen under the microscope; contains numbers of fatty globules and crystals of fatty acids and cholesterin; and swarms with the micro-organisms of putrescence. The false membrane consists in part of a fibrinous exudation, but mainly of the products of coagulative necrosis of the epithelial cells, and contains large numbers of diphtheria-bacilli.

Glands.—The swollen glands are, on section, soft, red, and pulpy, resembling splenic tissue. They show the changes of acute lymphadenitis, and contain micrococci. The lymphatics leading to them are also enlarged and inflamed, and contain various micro-organisms, among which, in cases which have been characterised by great severity, the specific bacillus can usually be demonstrated. Spots of suppuration are occasionally, but rarely, found in the glands.

Respiratory organs.—The larynx, trachea, and bronchi may be covered by false membrane. Its characters here are slightly different from those found in the pharynx. It is thinner, somewhat softer, and more easily separated from its attachments. These characters are due to no peculiarity in the exudation itself, but probably to the anatomical peculiarities of the mucous membrane. The false membrane may be prolonged into the smallest bronchi which can be followed out. More commonly, however, after proceeding down the trachea a little way, it becomes soft, loose, and at last continuous with a thick purulent, or muco-purulent exudation, which covers the mucous membrane of the bronchi, but is not attached to it. The lungs may be congested, or show the changes of broncho-pneumonia, or of pulmonary apoplexy. Very frequently, too, emphysema or insufflation of the lungs is seen in patches. In laryngeal cases the broncho-pneumonia is frequently of a specific nature, the air-cells being found crowded with characteristic bacilli.

The mediastinal glands may be swollen, and resemble in appearance those of the neck.

Circulatory organs.—The cavities of the heart, especially those of the right side, contain *ante-mortem* clots firmly adherent to the endocardium. Endocarditis is not found. The muscular tissue of the heart is nearly always softened, and may sometimes show

carditis or fatty degeneration. In a few cases, as one of the writers has found, colonies of micro-organisms may be seen embedded in the muscular tissue. Frequently, and especially if there has been difficulty of respiration, small punctiform hemorrhages may be observed beneath the visceral layers of the pericardium and pleuræ. In the malignant form, the blood after death is found to be of a dark chocolate colour, and very fluid.

Digestive organs.—The false membrane has been found extending down the whole of the œsophagus, and affecting the mucous membrane of the stomach, and, it is said, even that of the intestines. The liver is congested. The spleen is enlarged, but not excessively so, and on section is found to be softened and pulpy.

Urinary organs.—The kidneys, in all cases, show the change of their epithelial cells called 'cloudy degeneration.' When albuminuria has been present during life, the kidneys may present the usual appearances of acute parenchymatous nephritis. The organs are swollen, pale in colour, and soft on section. The cortical portion is unduly enlarged, and of a yellowish-white colour. On microscopical examination, the cells of the convoluted tubes, and also those of the glomerular and capsular epithelium, are seen to be swollen, and occasionally proliferated.

Muscles.—The external muscles may show the waxy degeneration of Zenker. Evidence of fatty degeneration affecting some of the fibres of the cardiac muscle is present in most severe cases, occasionally before the end of the first week.

Nervous system.—Associated with the paralysis, changes have been found in the peripheral nerves. The change consists in an acute parenchymatous degeneration of the medullated fibres. The white substance of Schwann first breaks up and then disappears, followed by subsequent disintegration of the axis-cylinder. The primitive sheath remains intact. Certain of the fibres in a nerve only are affected, a fact which accounts for paresis of the muscle rather than paralysis. Degenerative changes have also been found in the sympathetic nerve (Sidney Martin). The degeneration of the nerves is seldom found in the whole of their extent, and may attack the portions near the spinal cord or those more remote. The change corresponds in its distribution and degree with the region and severity of the paralysis. The affected muscles show granular and fatty degeneration.

PATHOLOGY.—It may now be asserted without fear of contradiction that diphtheria is primarily a local affection. The general symptoms of the disorder result from the absorption into the circulation of certain toxic products elaborated by the bacillus which multiplies at the seat of inoculation. This, by reason of its reactive influence upon the tissues in which it is growing, is productive of the membranous inflammation which constitutes the lesion so characteristic of the disease. That diphtheria, like tetanus, is essentially an intoxication and not a strictly infective process, as is the case with anthrax or relapsing fever, in which the blood becomes infested with the actual micro-organisms, is now firmly established.

But though, as a general rule, the bacillus in diphtheria remains localised at the seat of inoculation, its toxic products alone entering the circulation, in certain lethal attacks the bacillus may be found after death not only in the blood, but even in remote organs, viz. the lungs, spleen, and more

rarely, the liver and kidneys. This widespread dissemination of the bacilli probably occurs shortly before death, and would seem to represent an overflow, as it were, into the blood-stream, as the result of a breakdown of the normal resistance of the tissues.

The specific bacillus was first demonstrated in the false membrane by Klebs in 1883, and in the following year its causal relation to the disease was practically confirmed by Löffler by cultivation and inoculation of the lower animals.

The specific bacillus is invariably present in the false membrane, usually in irregular or roundish clumps which are more numerous in the superficial layers of the exudation. Smaller clusters and isolated bacilli are found scattered throughout its deeper portion, though more sparsely. Bacilli, too, occasionally invade the lymphatic spaces in the underlying mucous membrane, and in exceptional instances may even be detected in the contiguous lymphatic glands.

Although the bacillus may frequently be obtained in pure culture from the deeper part of the exudation, in the superficial layers, where they are more abundant, the bacilli are associated with numerous other organisms, chiefly pyogenic and putrefactive. A few streptococci, however, are usually to be found in the deeper layers. The pyogenic organisms are believed to play an important part in the symptomatology of the disease. Cases of so-called 'mixed infection,' in which streptococci are abundantly present in the blood and tissues, are almost invariably severe. The toxin of diphtheria appears to be of the nature of a ferment, and, according to Sidney Martin, the toxic effect is dependent upon various chemical derivatives, notably certain albumoses, and an organic acid, which are produced by its catalytic action upon the tissues and juices of the host. The filtered toxin, obtained by growing the diphtheria-bacillus for a week or two in glucose-free broth contained in shallow flasks, if injected into guinea-pigs, will not only result in paralysis and death of the animal, but will set up morbid changes in the viscera and peripheral nerves similar to those found in human diphtheria. Valuable light is thus shed upon some of the most characteristic secondary symptoms of the disease. Gradual cardiac failure is apparently determined by actual disease of the heart-muscle; hyaline followed by fatty degeneration of its individual fibres, resulting from circulation of the toxin. More or less ventricular dilatation is often present, sometimes attended in fatal cases with thrombosis in the cardiac cavities.

It is possible that the sudden cardiac failure which occasionally occurs in severe attacks may be dependent upon direct paralysis of the heart-muscle as the result of the circulation of toxin of an extremely virulent character. The peculiar degenerative changes in certain of the peripheral nerves in the human subject, associated with the muscular paresis and sensory disturbance described in the previous section, have their counterpart in the crural paralysis dependent upon similar histological changes, which soon appears in the guinea-pig after injection of the toxin. The albuminuria, so frequent in diphtheria, is probably caused in most cases by the irritant action of the toxin upon the renal epithelium, for similar degenerative changes result from the subcutaneous injection of the virus into guinea-pigs. A slight degree of albuminuria, however, appearing early in the disease in cases attended with a high temperature, may possibly be of pyrexial origin, and

it is quite possible that the copious albuminuria which sometimes comes on during convalescence, in conjunction with widespread paralysis, may be due to circulatory or vaso-motorial disturbance in the kidney.

In the spread of diphtheria personal intercourse is the most important agent. The disease may be directly conveyed by kissing, or by means of a cup, spoon, or handkerchief previously used by a person suffering from diphtheria. Surgeons who have inadvisedly sucked the wound to clear it of membrane after tracheotomy have contracted the disorder from their patient, and cases have occurred where the disease has been inoculated by means of an infected surgical instrument. The contagium may be given off in a particle of mucus, or a minute fleck of membrane ejected from the mouth or nose by the simple act of coughing, sneezing, or laughing in an explosive manner. This may be received directly into the mouth, nose, or eye of an attendant; or it may lodge on some article of furniture, the floor, the wall, or upon the clothing of a by-stander; whence after desiccation it may be conveyed in an almost imponderable form by a current of air on to the mucous membrane of a susceptible person. In confined air, in the absence of bright light, the bacillus in a dry condition may retain its infective properties for several months. Milk is an occasional channel of infection. The bacillus grows well in milk, and it is therefore quite capable of conveying the disease as the result of accidental contamination. Dr. Klein has shown good reason for believing that in some cases the milk may derive its infectivity from the cow itself as the result of the animal suffering an eruptive disorder which he believes to be the counterpart of human diphtheria. Various other animals react to inoculation, but, with the exception of cats, have no practical bearing upon the propagation of the disease. Cats, however, contract diphtheria very readily, its manifestations in their case being mainly pulmonary; and they have been shown to be concerned in giving the disease to healthy children. There is no evidence that diphtheria is ever conveyed by means of the water-supply. Virulent diphtheria-bacilli have not infrequently been found in the throats, even during the acute stage, of scarlet-fever patients. Though in most cases no clinical evidence of the disease is apparent, the bearing of this observation upon the frequent supervision of *post-scarlatinal diphtheria* cannot be ignored.

ÆTIOLOGY.—The predisposing causes of the disease may shortly be discussed as follows:

1. *Age.*—The statistics of the incidence of diphtheria at various ages show that approximately three-fourths of attacks occur in children under ten years of age, and that of these more than half occur in the second to the fifth year of life. The excess is probably due to the greater exposure of children to infection during this period owing to their aggregation in schools.

2. *Sex.*—The disease is more frequent in females than in males, in the proportion of about six to five.

3. *Heredity.*—There undoubtedly are some families in whom a more than usual number of cases of diphtheria occur, and this probably is due to an inherited weakness, rendering the members unduly susceptible to the poison.

4. *Season.*—A considerable increase in the number of cases of diphtheria begins in the third

quarter of the year ; the number attains its maximum in the fourth quarter, and, while declining in the first quarter, nevertheless is still in excess of the number occurring in the second quarter.

5. *Rank*.—The disease is more prevalent among the poor, partly, no doubt, by reason of a lower physique, and also because overcrowding increases facilities for infection.

6. *Hygienic conditions*.—There is no doubt that exposure to emanations from sewage is a fruitful source of diphtheria. How this comes about is not precisely certain. Thorne-Thorne, however, was of opinion that sewage-gas produces a morbid condition of the fauces which renders the patient more liable to the reception of the diphtheria-poison.

In former years diphtheria was more common in country districts than in towns, or, to speak more accurately, in the sparsely populated than in the densely populated parts. In recent times this relation has been reversed, and now diphtheria is more common in thickly populated districts. Statistics also show that geological conditions have but little effect on the incidence of the disease. According to Thorne-Thorne this is only true as regards the structure of the soil itself, in relation to drainage. A clayey soil may be rendered immune from diphtheria by the possibility of free drainage. But where moisture is accumulated, and also allowed to remain stagnant, there diphtheria is rife.

7. *Influence of other diseases*.—The way for the diphtheria-poison may be prepared by other diseases, such as tonsillitis, scarlet fever, measles, or whooping cough, rendering the fauces or respiratory mucous membrane a vulnerable point ; or by diseases which weaken the system and so diminish its power of resistance. Probably the puerperal state should be associated with the latter group of conditions.

By 'secondary diphtheria' is understood the occurrence of diphtheritic lesions and symptoms after various other diseases, among which the principal are scarlet fever and measles. It must be remembered in connection with this subject that the mere appearance of a false membrane is not a proof of the existence of diphtheria. Thus, in scarlet fever, the occurrence of a false membrane on the fauces may be due to the activity of certain pyogenic organisms. Nevertheless, cases not infrequently occur—and one of the writers has had many opportunities of examining such—where, concurrently with positive symptoms of scarlatina, large quantities of diphtheritic membrane have formed in the throat, and have even extended to the larynx, and where also after a time the characteristic paralyses of diphtheria have shown themselves. Such cases are specially prone to occur when epidemics of scarlet fever and diphtheria co-exist, and there is reasonable ground for believing that they are due to a dual infection by the poison of both diseases.

8. *Previous attacks*.—One attack of diphtheria confers no prolonged immunity upon its subject. Even during convalescence the patient has been known to develop the disease afresh, and this may be repeated more than once.

DIAGNOSIS.—In a well-developed attack of diphtheria the diagnosis should present no difficulty, but where the characteristic lesion is not visible to the eye, and in very mild attacks, a bacteriological examination of the suspected exudation, or of the secretion in the fauces, nares, or larynx, may be expected to yield evidence of the greatest value.

A brief description of the main bacteriological indications is therefore necessary.

The Klebs-Löffler bacillus is a polymorphic organism, in length varying from 2 to 6 μ , and in breadth from .5 to 1 μ . It is either straight or slightly curved, and often shows a slight swelling of one or both ends. In some instances the swelling of one end may be inordinately great, while the other perhaps tapers off to a fine point, with the result that the bacillus takes the form of a club ; or should the bacillus be a short one, it presents a pyriform appearance. This tendency to 'club formation' is best marked in older cultures, and is evidence in most cases of involution. It is customary to speak of two forms of diphtheria-bacilli, the long and the short, each of which usually retains its characteristics under cultivation. In cover-glass preparations taken direct from the throat, variations both in the shape and size of the individual bacilli are usually to be noted : but when taken from a culture, the tendency is, for the majority of them, to conform to a common type, but few, relatively, being of different proportions. The bacillus is non-motile and does not form spores. It stains readily with the usual basic aniline dyes, but takes the stain irregularly, a peculiarity which imparts to it a beaded or even segmented appearance. This irregularity of staining is most distinctive, and is best brought out by using Löffler's alkaline methylene-blue, when dark-blue dots, usually one at each end, are seen in the protoplasm of the bacillus. These are usually spoken of as 'polar granules.' If stained with gentian-violet after Gram's method, the characteristic irregularity of the staining is very apparent. Neisser has devised a double stain, by means of which he claims the diphtheria-bacillus can always be differentiated from allied forms. His procedure is as follows : A cover-glass preparation, made from a one night's serum-culture incubated at 35° C., is immersed for 1-3 seconds in a solution made as follows :—Dissolve 1 gramme methylene blue (Grübler's) in 20 cc. of a 96 per cent. alcohol. Mix this with 950 cc. distilled water, containing 50 cc. glacial acetic acid ; then immerse cover-glass for 3-5 seconds in a solution made by dissolving 2 grammes of vesuvin in 1 litre of boiling distilled water, subsequently filtered. The film is then washed in distilled water, dried, and mounted. By this method the body of the bacillus is stained a faint brown colour, while several (usually two to four) blue dots can be seen in its substance, which, according to Neisser, are distinctive of the true bacillus. According, however, to most observers in this country, the test in their hands has not proved equally reliable, but it may be regarded as still on its trial. At the temperature of the body, 37° C., the diphtheria-bacillus grows readily on the ordinary culture media. Below 20° C. no growth takes place. The most suitable medium is blood-serum, especially when prepared by the methods of Löffler or Lorrain Smith. On blood-serum at 37° C. the colonies, in 12-16 hours, may be recognised as minute dead-white circular dots, and by the end of twenty-four hours are as large as a fair-sized pin's head, the central portion of each being thicker and less translucent than the periphery. On glycerine-agar the growth is very similar, but somewhat less rapid. In stroke-cultures numerous out-lying colonies may be discerned along the margin of the streak, some of which are very minute. On nutrient gelatine at room-temperatures, the

growth, though slow, is very characteristic. The colonies gradually develop a faintly yellow tint, and their centres become more raised and often surrounded with concentric markings, which give them a ripple-like appearance. This, however, is hardly apparent before the colonies are ten days old. No liquefaction of any of these solid media takes place. When the bacillus is grown in broth the medium becomes turbid in a few hours, and a chalky precipitate is deposited on the bottom and sides of the flask. The broth, moreover, in from twenty-four to forty-eight hours becomes acid in reaction, but after the lapse of a few days again becomes alkaline, in fact more so than before. The acidity, however, will not develop should the broth have previously been rendered free from glucose.

Occasionally in diphtheritic lesions, but far more frequently in non-diphtheritic or healthy throats, bacilli are found which closely resemble the Klebs-Löffler bacillus in most, if not all, of their morphological and cultural characters, but differ from it in that they are not virulent when inoculated into the lower animals. Such organisms are usually spoken of as 'pseudo-diphtheritic bacilli' on the supposition that, though resembling the true bacillus, they are of a distinct species. The term, however, should not be applied to an organism which differs from the true bacillus *solely* in respect to its virulence, since this property is capable of enormous modification even by artificial means. It is safer, therefore, at any rate until the limits of normal variation can be more clearly defined, to regard such an organism as a true bacillus which has undergone attenuation. Among the various organisms to which the term pseudo-bacillus may be rightly applied, the bacillus originally described by Löffler and by Hofmann, in the years 1887 and 1888 respectively, is the most widely recognised. The Hofmann bacillus is frequently present both on healthy fauces and in non-diphtheritic sore-throats. In appearance it is very similar to the short diphtheria-bacillus, but usually more pyramidal in shape. The bacilli often occur in pairs with their bases in apposition, and two or three pairs may frequently be observed lying side by side in the field. On ordinary media its cultures are practically indistinguishable in appearance from those of the true bacillus. Further points which are of assistance in the differentiation of pseudo-diphtheritic bacilli from the true species are—(1) no blue granules are developed when Neisser's stain is employed (according to Pakes and other observers this is also occasionally true of the diphtheria-bacillus); (2) no acid is developed when the organism is grown in broth containing a trace of glucose (there is, however, at least one exception to this rule, the *Bacillus acidum-faciens* of Kurth); (3) they are not virulent to guinea-pigs.

In order to obtain a bacteriological examination of the throat the material is usually obtained in the following way:—a swab of sterilised cotton-wool, mounted on a stiff piece of wire, is firmly pressed against the affected surface and rotated so as to thoroughly impregnate the cotton-wool with secretion. The swab should then be gently rubbed over the surface of the blood-serum contained in a culture-tube so as to ensure insemination. The tube should then be incubated at 37° C. and the culture examined after the lapse of 16 to 24 hours. A cover-glass preparation may with advantage be made from the material on the swab at the time it is taken, and

examined forthwith. In this way valuable time may often be saved, should the examination be conducted by a skilled observer. The result, however, particularly if negative, should always be confirmed by subsequent examination of the culture, since bacilli are frequently revealed by cultivation, though they may not have been detected microscopically in the secretion taken direct from the throat.

At the present day the bacteriological test enjoys a wide reputation, and it is undoubtedly one of considerable value. There are, however, certain disabilities attaching to the test, as usually carried out, which seriously impair its value as an altogether reliable indication of diphtheria.

The uncertainty of our knowledge as to the essential criteria of the diphtheria-bacillus not infrequently leads to considerable difference of opinion between bacteriologists as to the specificity of some particular specimen. Its relative virulence, in so far as it concerns the particular patient, may be determined, it is true, by observing its effects when inoculated into one of the lower animals; but this implies the loss of valuable time, several days at least; and even then, the question of specificity may not be satisfactorily settled. Apart from the question of the identity of bacilli, it must be remembered that a negative examination simply implies a failure to detect the bacillus—not necessarily that it is not there. One of the writers has met with several cases in which, though the patient died from typical diphtheria, repeated examination of the material taken by the usual method failed in the hands of an expert of wide experience to reveal a single diphtheria-bacillus. These, it is true, are exceptional instances; nevertheless, a negative result in practice is often contradicted by a subsequent examination.

In view of these inherent drawbacks, it cannot be too strongly urged that the diagnosis of a case of diphtheria should not be founded upon bacteriological evidence alone, but rather upon its clinical appearances, supplemented by bacteriological examination. If the clinical aspect of the case be negative, though apparently genuine diphtheria-bacilli have been detected in the culture, the bacteriological enquiry should include the results of injection into animals. Moreover, in cases where the culture taken from the throat reveals no diphtheria-bacilli, a further examination should be made; indeed it is safer to require three examinations, made if possible on consecutive days, in each instance with a negative result, before deciding that the case is not one of diphtheria.

Almost any affection of the throat *may* be mistaken for diphtheria, but the mistake is only likely to happen in the case of acute follicular tonsillitis, scarlet fever, and syphilitic affections. Their points of clinical difference may be summarised thus:—

1. *Acute follicular tonsillitis*.—Here the amount of fever may be equal to or even greater than that found in diphtheria. Usually the fever begins more suddenly, and is accompanied by more pain in the throat and more disagreeable general symptoms than occur in the early stage of diphtheria. In fact, the less serious disease has in most cases the more severe symptoms. In acute follicular tonsillitis, even from the first, exudation can be seen issuing from the tonsillar crypts, forming many small white points upon the surface of the swollen tonsils, and nowhere else. In diphtheria some hours, or even days,

usually elapse before more than one patch of membrane is seen, and this may possibly not be upon the tonsil; or, if there be more than one, a patch situated elsewhere than on the tonsil will decide the diagnosis. The diphtheritic membrane, too, often becomes thick and leathery, and is usually not easily removed, while the exudation of tonsillitis remains soft and creamy and is easily wiped away with a brush. In acute tonsillitis nasal catarrh is not often met with, while in diphtheria of moderate severity it is frequently an early symptom.

2. *Scarlet fever*.—Diphtheria rarely begins with the same suddenness as scarlet fever; and vomiting, too, is not usually an early symptom. The temperature in scarlet fever rapidly rises to a height which is more rarely found in diphtheria. After twenty-four hours, too, the characteristic rash of scarlet fever appears. This may possibly be mistaken for the erythema rarely seen in diphtheria. But the diphtheria-rash, when present, does not spread over the whole body, being limited to the upper parts of the chest and the neck; it is not punctate; and it is never an early symptom. The special albuminuria of scarlet fever first makes its appearance late in the disease or at the beginning of convalescence; and if albuminuria should occur earlier, it is only slight, and such as may accompany any febrile state. In diphtheria, on the other hand, albuminuria is an early symptom; and in a doubtful case, if the urine contains much albumen after only two or three days of the fever, a diagnosis of diphtheria is the more probable. The urine in diphtheria rarely contains blood. The throat-affections of the two diseases may resemble each other very closely, for in scarlet fever not only may the slough of the mucous membrane simulate the false membrane of malignant diphtheria, but, as already stated, there may occur in scarlet fever a false membrane having much the same appearance as that of diphtheria. Yet in scarlet fever ulceration is more prominent than in diphtheria; shreds of membrane are not expectorated, and there is no tendency of the throat-affection to spread to the larynx. One of the writers has already mentioned, as his opinion, that in the cases where laryngeal affection has ensued upon scarlatinal symptoms, there has been a double infection by the two poisons.

3. *Syphilitic affections*.—Syphilis, in the secondary stage, may produce white patches over various parts of the throat, due to coagulative necrosis of the epithelial cells. These may be mistaken for diphtheritic membrane. But they are never accompanied by so much fever as occurs in diphtheria; they are not so raised as diphtheritic patches; and the parts around are not so swollen as in diphtheria. Moreover, the rash of secondary syphilis present on the body-surface at the same time is diagnostic. The ulceration of the throat which may appear in the tertiary stage of syphilis might be mistaken for a diphtheritic condition during an epidemic of diphtheria; but the remembrance of the possibility of its occurrence should be sufficient to remove the chance of error.

The special characters of the paralysis distinguishing it as of diphtheritic origin are—the history of the throat-trouble, if this be obtainable; the peculiar progress of the paralysis; and the early affection of the palate and eyes, which are rarely touched by other forms of peripheral neuritis. *See NEURITIS, MULTIPLE.*

PROGNOSIS.—The prognosis in diphtheria must

always be guarded, even in the mild cases, for serious symptoms may at any time break out. The prognosis is the more serious the greater the quantity of membrane to be seen in the throat, the higher the temperature, and the greater the weakness and the severity of the general symptoms. The early signs of heart-failure—namely, an unduly slow, very rapid, or intermittent pulse, and the alterations in the cardiac sounds already detailed—are warnings of impending great peril. The malignant form is nearly always fatal; and consequently the occurrence of much hæmorrhage from the throat or nose, purpura, a gangrenous condition of the false membrane and surrounding parts of the throat, or signs of much nasal affection, are of grave significance. The occurrence of albuminuria adds nothing at all to the severity of the original disease, and therefore is *per se* of no consequence in judging the prognosis. But, with this exception, any one of the complications mentioned above adds greatly to the gravity of the attack. This is especially the case with extension of the disease to the larynx, which if unrelieved by operation, in nearly all cases not treated with antitoxin will kill the patient. After operation, too, a large number of patients die either from the severity of the original attack, or from extension of the membrane down the bronchi and consequent occurrence of broncho-pneumonia. In the experience of one of the writers, those cases of laryngeal diphtheria which have followed on scarlatinal symptoms are specially fatal. Generally speaking, the prognosis is the less favourable the younger the subject.

The prognosis of diphtheritic paralysis is on the whole favourable. Nearly always the paralysis recovers. The unfavourable cases are those in which the laryngeal, respiratory, and pharyngeal muscles are paralysed.

TREATMENT.—(i.) *General arrangements.*—The patient should be isolated in a bright, well-ventilated room. This should be cleared as far as possible of superfluous furniture, and the carpet preferably removed. Its temperature should be maintained by means of an open fire at about 60° F., but in laryngeal attacks it may be 5 or 6 degrees warmer, and the air moistened by means of a steam kettle. Any mucous discharge from the throat or nose should be received upon pieces of rag or cotton-wool, and immediately burned; as should all pieces of membrane coughed up by the patient. Such material, if required for the doctor's inspection, should be kept in some antiseptic solution. No cat should be allowed in the room under any circumstances. The patient should be isolated for at least three weeks after the throat has become clear, and with children it is well to insist on a week or even a fortnight longer. Quarantine, moreover, should be maintained until the complete cessation of any mucous discharge from the mouth, throat, or ears which may have been present. The practice of regulating a patient's discharge according to the observed presence or absence of the diphtheria-bacillus, though excellent in theory, is not altogether satisfactory. The foregoing indications, however, may be followed with confidence.

(ii.) *Antitoxin-treatment.*—The introduction of antitoxin towards the close of the year 1894 has revolutionised the treatment of diphtheria. A previous mortality in the public fever-hospitals of the metropolis of over 30 per cent. has been reduced to less than 12 as the result of its employment. The improvement is even more striking in laryngeal attacks,

the mortality after tracheotomy having fallen from over 70 per cent. to less than 40. Were it possible to treat the cases at the beginning of the disease, the results would be even better. In the administration of antitoxic serum two things are essential to complete success. Firstly—the *treatment should be commenced early*. During the years 1895-7 the mortality in the Asylums Board hospitals among patients brought under treatment with antitoxin on the first, second, and third day of illness was 3·8, 12·1, and 20·9 respectively as against 22·5, 27·, and 29·4 in the previous year, when antitoxin was not available. If, however, the remedy be withheld until late in the disease, after the tissues have been irretrievably damaged by the toxin, no form of treatment, antitoxic or otherwise, can avail. Secondly—*The dosage should be adequate, and the serum reliable*. A dose varying from 2,000 to 8,000 units should be injected in proportion as (1) the case is brought late under treatment, (2) the attack is severe, and (3) laryngeal extension threatens. In urgent cases a second dose of 2,000 to 4,000 units may be injected at the end of 12 hours, and the injection repeated at the expiration of another 24, unless the membrane have definitely commenced to separate. In regulating the dosage the age of the patient need not be considered. The antitoxic value of the different sera in the market varies considerably, but a reliable one of such strength that a thousand antitoxic units are contained in from three to five cubic centimetres can usually be obtained from any of the leading London chemists. The more concentrated sera are the most expensive. The injection is preferably given into the loose subcutaneous tissue of the flank or abdomen. The best syringe for the purpose is Roux's, as it can be readily taken to pieces and boiled. If the injection is conducted with strict aseptic precautions, as it always should be, a large amount of serum may be injected (20 to 40 cc.) without any bad result. The distension of the part need cause no alarm, as it subsides within half an hour, usually less. The degree of pain attending the injection varies greatly in different cases. Apparently, no benefit is gained by giving more than 16,000 units within the first 24 hours. In the case of a young child the administration of antitoxin should never be omitted, however mild the attack. Frequently, as an effect of the horse-serum injected, a rash, sometimes attended with considerable itching, of an erythematous or urticarial character appears during the second week, and lasts a few days. It is usually accompanied by pyrexia, and occasionally (5 to 6 per cent.) pain and tenderness, with slight swelling of one or more of the joints. Occasionally an abscess will develop at the seat of injection, the result, probably, of defective aseptic precautions. These after-effects of the serum are sometimes productive of considerable discomfort while they last, but are trivial in comparison with the enormous benefit which the treatment usually confers.

(iii.) *Local treatment*.—With the object of hindering the multiplication of the diphtheria-bacillus and other micro-organisms associated with it, it is always advisable to periodically syringe out the fauces, and if necessary the nasal fossæ, with some non-irritating and astringent antiseptic solution. By this means offensive secretions are cleared away, and a more healthy action of the mucous membrane encouraged. For this purpose nothing is better than a weak acid solution of chlorate of potassium containing free chlorine, prepared by pouring strong hydro-

chloric acid upon the powdered salt; and then shaking up with water in the proportions of 4 minims of the acid to 9 grains of chlorate of potassium in each ounce of water; or a solution of formalin (1 in 200) or of chinolol (1 in 600) may be substituted. A warm solution of boric acid (5 grs. to the ounce) is also to be recommended. The treatment is best effected by means of a four-ounce india-rubber ball-syringe, which can be used with one hand, the other being free to restrain the movements of the patient's head, if a child; or to assist, if need be, in holding the basin into which the solution is received as it runs out of the mouth. The nozzle of the syringe should be introduced between the teeth, and passed over the back of the tongue, while the patient's head is held forward over the basin. The throat should be somewhat forcibly syringed in this manner every two, three, or four hours according to the severity of the attack. In comparison with this method, that of gargling, swabbing, or spraying is relatively useless. Should the patient be a young child, and actively resist the treatment, it is better to desist in view of the exhaustion which it might entail; for in cases treated with antitoxin the insistence on local treatment is less important. The forcible removal of any membrane which is still adherent, or the application of any strong caustic after its removal, is to be strongly condemned. In cases attended with profuse rhinorrhœa the nasal fossæ also should be irrigated at intervals with a weak solution of boric acid.

In laryngeal attacks should the stridor become pronounced, the question of its relief by operation will arise. One of the most satisfactory features of the serum-treatment is, that in addition to the improved results after tracheotomy, it also brings about an early separation of the membrane, and consequently the necessity for operative interference less frequently arises. The necessity for operation will depend upon the urgency of the dyspnoea, the degree of cyanosis, the amount of muscular effort required to maintain the act of respiration, and whether antitoxin has been given sufficiently long to afford a reasonable expectation that the membrane may be expelled before the child's strength is exhausted. Tracheotomy is in most cases preferable to intubation, especially in private practice where the doctor is not always at hand. When the operator is skilled in tracheotomy the opening should be made as low down the trachea as possible, as the after-results are more satisfactory. Intubation is contra-indicated when there is much faucial membrane, when the nares are blocked, and when the patient is *in extremis*. In the early stage of laryngeal stenosis the patient may be placed in a steam-tent, and sponges wrung out of hot water frequently applied over the larynx. After tracheotomy the kettle may be discontinued.

(iv.) *Symptomatic treatment*.—In faucial attacks, hot fomentations applied to the neck will afford some ease to the inflamed and tender glands, and, possibly, tend to promote resolution. Except in very mild attacks in adults, absolute recumbency in bed should be insisted on, and no exertion of any kind permitted. Care is particularly needed during the second and third weeks of severe diphtheria. Both the pulse and the action of the heart should be carefully watched from day to day, and the position of the apex-beat followed. An abnormally quick pulse, associated with a normal temperature; or an unduly slow pulse, are signs of the greatest danger, as is also a sharp short systolic sound, or other sign of weakness or commencing cardiac dilatation.

Under these circumstances, liquor strychninæ should be given in repeated small doses, combined, if thought desirable, with a few grains of citrate of caffeine; and a half to one drachm of brandy may be administered every two hours to a young child. Should persistent vomiting arise, the strychnine must be given hypodermically, and the brandy by rectum.

The albuminuria of diphtheria requires no special treatment; and that of anuria, if present, is comprised in the treatment of the cardiac failure with which it is associated. During the acute stage the employment of drugs is worse than useless; as is that of digitalis in heart-failure. They tend to induce vomiting, which should be prevented if possible. During convalescence a mixture containing bark and ammonia is often useful; as also is iron in the form either of tinctura ferri perchloridi, or Easton's Syrup, which may be continued for several weeks. The period during which the patient should be kept in bed should be mainly regulated by the previous severity of the attack, in the absence of any sign of cardiac weakness or paralysis. Should, however, the former appear, the greatest care should be enjoined, and the patient kept strictly recumbent until the heart's action is again deemed to be satisfactory. Diphtheritic paralysis calls for no special treatment in the majority of cases. Rest in bed should be maintained until definite improvement has commenced, when the patient may be allowed to sit in a chair; and if it continue, to gradually extend his operations. Galvanism early in the affection is liable to do harm, but when the paralysis is stationary, or but slowly improving, it may be cautiously employed, combined with gentle massage, and the daily administration of Easton's Syrup. Should the patient be unable to expel the mucous secretion from his larynx, owing to paralysis of the diaphragm, especially if combined with paralysis of the larynx and pharyngeal constrictors, small doses of liquor atropinæ tend to lessen distress by checking any excess of secretion.

(v.) *Dietetic treatment.*—During the acute stage the diet should consist of milk, beef-tea, soup, and broths; with the addition of beaten-up eggs, jelly, and ripe fruit if desired, and as soon as the patient can swallow with comfort, a light solid diet may be permitted. Stimulants are only required when the patient is taking his food badly; when gradual cardiac failure threatens; and in elderly persons. The vomiting associated with cardiac failure entirely precludes feeding by the mouth, and for a time food in peptonised form must be administered by rectum. With children who will not swallow in consequence of the distress it entails, and in pharyngeal paralysis, nasal feeding must be employed, a method which is also to be recommended when diaphragmatic paralysis is combined with laryngeal anæsthesia. ROBERT MAGUIRE.

F. FOORD CAIGER.

DIPHTHERITIC.—Relating to diphtheria. The term is applied to the membrane formed in diphtheria; and it is also associated with certain symptoms occurring in the course of the disease, such as diphtheritic paralysis. See CROUPOUS; and DIPHTHERIA.

DIPLOE, Diseases of.—See SKULL, Diseases of.

DIPLOPIA (διπλός, double; and ὄπτομαι, I see). Double vision. See STRABISMUS.

DIPSOMANIA (δίψα, thirst; and μανία, madness). SYNON.: Narcomania, Inebriety, Habitual Drunkenness, Oinomania; Fr. *Manie ébrieuse ou crapuleuse*; Ger. *Trunksucht*.

DEFINITION.—A moral insanity, characterised by an irresistible desire to experience the sensations produced by the physiological action of alcohol upon the nervous system.

ÆTIOLOGY.—Drunkenness as met with in everyday life may be classified as vicious and diseased. The care of the vicious drinker is the work of the social reformer rather than of the physician, except for the important influence that habitual wilful excess has in the production of the graver condition. Many cases of drunkenness are, through ignorance of the subject, treated as vicious, though really diseased, and consequently as objects for abuse instead of pity. Any individual who exhibits a morbid desire for intoxication, an incapability of resisting that impulse, and an impossibility of drinking alcoholic liquors without proceeding to excess, should be classed as diseased. The presence or absence of the *power to resist the craving* is the only logical division between the vicious and insane drinker. Dipsomania, as a term, has been too exclusively limited to one variety of the condition—the 'periodic' inebriate—in neglect of the facts that many 'constant' drinkers are no less insane in their habits and no more capable of resisting the impulse to drink. The failure to recognise the diseased aspect of the 'constant' dipsomaniac has no doubt been due to the ill-defined line of demarcation between that class and the vicious drunkard.

The causes of dipsomania are:—(1) *A transmitted functional or structural defect*, evidenced by a weakened power of resisting morbid impulses.

There is reasonable doubt concerning the actual transmission of drunkenness, *per se*, from parent to child. Clinically, however, there exists ample evidence of the greater liability of the children of drunken parents to become drunken. This is probably due to nervous degeneracy which evidences itself in actual drunkenness owing to early example, influences, and the facilities for drinking afforded through life. Insanity, epilepsy, and neuroses generally will predispose to inebriety in offspring. On the other hand, insanity and epilepsy may appear concurrently with, or in the place of, drunkenness among the children of inebriate parents. The three conditions act, *inter se*, as cause and effect with triangular reciprocity.

2. *Long-continued vicious drinking.*—In a large percentage of dipsomaniacs careful search will reveal an inebriate or neurotic history; but in other cases this is not obtainable. Observation makes it certain that the condition may be acquired as a result of perpetual careless drinking. The successful struggle, however, that a man of high moral sense makes against a growing habit, as compared with that made by the man of low moral capacity, lends colour to the belief that even in these acquired cases some physical or functional nervous degeneracy is the real cause of the ultimate result. It is probably true that the appetite is to a great extent acquired in most cases, and that if no one ever tasted alcoholic liquors there would be no longing for them; but the point to bear in mind is that there are many who, from transmitted tendency, or defective physical, mental, or moral stamina, fall an easy prey to inebriety.

3. *Traumatism.*—Some cases are undoubtedly

of traumatic origin. Loss of self-control may follow falls, blows on the head, sunstroke, or other bodily injury. Instances have also occurred after exhausting diseases, or have been associated with painful disorders, and especially with uterine and ovarian troubles.

4. Drunkenness may be the first evidence of insidious *mental disease*, as in general paralysis of the insane.

5. *Early habitual use*, as in the case of children to whom small quantities are administered regularly.

6. Mental degeneration resulting from the *high pressure of modern life*; continual excitement and irregular living among the well-to-do; business anxiety and domestic worry in the man struggling for existence; imperfect hygiene, insufficient food, hardship, and excessive temptation associated with certain trades and professions.

7. The attractive *physiological effect* of alcohol in producing temporary relief, and the facility with which it can be obtained by all classes.

VARIETIES.—Three varieties of dipsomania are met with:—(a) periodic; (b) constant; (c) constant with periodic exacerbation.

(a) *Periodic* inebriety presents a marked analogy to recurrent mania and to the nervous discharge of epilepsy. Attacks often recur with marked regularity, having intervals of sobriety varying from one or two weeks to three months or even longer. The majority of these cases cannot touch liquor without inducing a paroxysm. The attacks may therefore appear at irregular intervals from occasional accidental lapses, and an accidental lapse occurring during an interval may postpone the regular outbreak and so still further mask the periodicity. Seizures are usually preceded by a longer or shorter period of restlessness, intense nervous irritability, and depression of spirits. In some cases there is headache, neuralgia, insomnia, delusions of suspicion, or dread of impending calamity. This premonitory stage may last for days before the actual outbreak, the patient struggling meanwhile until the craving becomes irresistible and the torture no longer bearable. On the other hand it is occasionally so short as to be hardly recognisable. Instances are known where a victim will, after weeks of abstinence, start up in the midst of some quiet occupation and rush to the nearest public-house to drown acute desire in the oblivion of excess, or be changed during a walk of a hundred yards from a quiet reasonable being into an excited irresponsible maniac capable of committing any action, however gross, to satisfy his morbid impulse. The attack usually lasts from three to seven days, during which time liquor is literally poured down without intermission, and after which the craving suddenly disappears. The patient then ceases drinking without difficulty and apparently recovers normal health after a day or two of physical discomfort and mental distress. He may then remain a total abstainer until the time arrives for a recurrence of the cycle. By the gradual lengthening of attacks and abbreviation of intervals, some periodic cases develop gradually into constant inebriates. Repeated attacks produce moral and intellectual degradation, and cases may terminate in dementia or melancholia. This periodic type most commonly occurs in young men between twenty and thirty, and is often accompanied by maniacal frenzy and violence during attacks; as a rule, the younger the case the more may violence be anticipated. Among females,

periodic inebriety is often associated with menstruation owing to the functional disturbance occurring in many women at the approach of each period. Furthermore, in the latter case some pathological condition often exists which causes a desire for the sedative influence of an anæsthetic agent.

(b) *Constant or habitual* inebriety may be traceable to hereditary neurotic influences as definitely as is the periodic variety. From knowledge and analysis of some 2,000 cases the writer has found a family history of drunkenness, insanity, or allied neurosis in 42 per cent. of periodic, and in 39 per cent. of constant inebriates. These figures do not include 'free living,' and make no allowance for the difficulty in obtaining true records from the patient's friends who may be ignorant of or reluctant to confess the presence of inebriety or insanity in the family history. Constant inebriety is the type most commonly assumed when the disease appears later in life and is excited by long-continued vicious habits. The constant drunkard, when he becomes a confirmed inebriate, is every whit as insane a drinker as is the periodic; and many of this type (although true dipsomaniacs) have been treated as vicious drunkards simply because clinically they were regular and not periodic habitués. When the condition has become confirmed the individual is literally the slave of his impulse. He is impelled to drink, against his reason and against his wish, just as in orthodox lunacy the maniac is impelled to suicide or homicide. He will drink, knowing that so doing will certainly bring poverty and ruin upon himself or on those dependent upon his efforts; this notwithstanding a previous life of careful or even parsimonious living. He drinks, aware that that way lies a shortened life, mental injury, or even crime. Inhibit his liquor, point out to him when sober that the first glass taken will inevitably have this result; but if restraint be withdrawn too soon, that first glass will certainly follow.

Some accompanying characteristics of this condition are: moral depravity, disregard of personal appearance, disposal of property to obtain drink, or even appropriation of that belonging to others, inconsistent lying, and sexual perversion. The termination of these cases is that of chronic alcoholism (see ALCOHOLISM). Whereas in periodical inebriety the results are mainly temporary, in the constant variety they are mainly permanent and due to tissue-changes.

(c) *The mixed variety*—constant with periodic exacerbation—is less common. It may be considered the connecting link between the foregoing classes, and is often the transition stage between them, exhibiting the combined characteristics of both.

PATHOLOGY.—The pathology of dipsomania is unknown. It is easy to define the *post-mortem* changes due to actual alcoholism, but it is impossible to indicate any lesion which gives rise to the uncontrollable desire to resort to excess. The condition is probably due to minute changes resulting in an unstable condition of nerve-tissue and consequent loss of volitional balance.

PROGNOSIS.—Probably a third of all cases that submit to proper treatment recover. Higher percentages are claimed by some, but the figures available are scanty and unreliable. Undoubtedly there would be a much larger percentage of recoveries if control and treatment were resorted to at an earlier stage of the disease instead of being used as a *dernier*

ressort; and if longer periods of detention were accepted as necessary. The records of our reliable institutions show many instances of three months' residence being considered sufficient for patients whose drinking-habits extended over 10 to 15 years. The chance of recovery is better for the constant than for the periodic inebriate, and in either case the earlier in life the habit becomes confirmed the worse the prognosis. Drinking-habits commencing between the age of 30 and 40 are most amenable to treatment. The impression that less hope of recovery exists for women than for men is probably erroneous; the experience of some well-conducted philanthropic institutions suggests the reverse.

TREATMENT.—Few other known conditions have given rise to as much constant effort to discover an antidote, or some charm that would act as specific and prophylactic, relieving the drunkard from his insatiable craving, and curing him without effort of will and without the distress attendant upon removal of liquor. Wonderful discoveries have been recorded, but none have stood the light of scientific investigation, and all have proved utterly valueless in averting or curing habitual drunkenness. When a man is drinking hard it is possible, by substitution of other drugs, to assist in the removal of the acute condition, but when that substituted remedy is discontinued, the original condition—the insane desire for intoxication—still remains unabated. Many so-called ‘cures’ afford the victim assistance in breaking off the acute condition—the actual drunkenness—but are devoid of any restorative power in the direction of mental or moral renovation. Cases here and there exhibit sufficient will-power when once made sober to avoid return to former habits, and, few as these are, judicious advertisement of them has lent a vogue of value to some of the nostrums and methods in common use. Again, some highly neurotic inebriates are subject to periods during which any influence, acting strongly on the imagination, will tend to temporary improvement. Such cases swell the lists of cures resulting from hypnotism, faith-healing, and temperance or religious revivals. The susceptibility of such cases to hyper-enthusiasm is a symptom in itself of an ill-balanced mental condition; and for this reason the prospect of permanent cure, when the emotion has subsided, is small indeed. The dipsomaniac must be considered as mentally diseased, and must be treated on the same lines that prove of value in more definite forms of lunacy. In this relation there are three primary indications—none of which must be neglected: (1) removal of a drunkard from his surroundings; (2) abstinence from liquor for long-continued periods; (3) treatment with such medicinal remedies and general hygienic measures as are necessary for recovery of the impaired functions and tissues.

In a confirmed inebriate, moral suasion, argument, and religious influences, although of value in subsequent treatment, are initially useless. Every medical man in general practice is called upon from time to time to deal with dipsomaniacs, and every one who has done so has encountered innumerable difficulties. To attempt to treat such a case in the patient's own home is almost hopeless. With the aid of an attendant or by constant watching, it is possible to arrest acute drinking. But the patient is sick during the process and possibly amenable. After the acute symptoms have passed, and just at the time when continued restraint is invaluable, the patient reasserts himself and declines to be further

controlled—with the usual result. It is almost impossible to remove a drunkard from his surroundings, keep him (often forcibly) from liquor for long-continued periods, and maintain continuous medical treatment and supervision, except in institutions specially designed for the purpose, and endowed with legal power to restrain. Unfortunately the power at present available to cope with such cases is strictly limited and ineffective. Recognition of the necessity led to the introduction and passing of the *Habitual Drunkards Act* of 1879. During its progress through Parliament, however, it was shorn of its most important feature, viz. the power to commit (against his will) an inebriate to proper care and control. When it actually became law, this measure, while arranging for and regulating the establishment of ‘Retreats,’ made it essential that any patients entering such institution shall do so of their own free will and accord. Later legislation—the Act of 1898—insinuates the principle of compulsory detention, but limits its application to cases convicted of crime caused by or attributable to drink, and to such cases as are repeatedly convicted of drunkenness in police courts. Two classes of inebriates only can therefore be legally dealt with under existing powers: the one that consents to restraint, and the one that brings itself within the clutches of the law. The great majority of inebriates are not included in either class, and consequently cannot at present be subjected to adequate restraint. Notwithstanding this, many cases can be induced to consent to restraint, especially when convalescent after an illness brought on by their habits. Then follows the selection of a Retreat and the signature of documents.

A Retreat should have pleasant surroundings and be conducted on firm but kind principles. Given good personal supervision, only such physical measures for restraint should exist as are absolutely necessary for safe custody. There should be regular medical attention and ample provision for the employment and interest of patients.

To obtain the full advantage of legal power two documents require execution: (1) a ‘*Request for Reception*’ signed by the applicant in the presence of a Justice of the Peace. This may be done privately at the house of the magistrate or patient. (2) A ‘*Statutory Declaration*’ signed by any two friends before a Justice of the Peace or a Commissioner to administer Oaths to the effect that the applicant is an inebriate within the meaning of the Act. Both forms are prescribed by the Act. When these papers are signed the person can be received into a Retreat by consent of its managers, and there detained until the expiration of the period for which he signed.¹ As a general rule very little drug-treatment is required after the acute distress accompanying discontinuance of liquor has disappeared. Ordinary tonic remedies are indicated, and attention should be paid to any pathological condition that may be present, remembering that any such condition may have largely contributed to the origin or continuance of drunkenness, and consequently will have an important influence upon the patient's future condition.

Too much importance cannot be laid upon the fact that many cases, if permitted, would soon become as dependent upon drugs (narcotics in particular) as they were previously upon alcohol,

¹ A List of Retreats is published annually in the Home Office Report by H.M. Inspector under the Inebriates Act.

and continued administration of any drug is therefore inadvisable. The habit of drinking fluids of any sort, more than is necessary, should be discountenanced, and patients taught to resist the abnormal demand of a stomach accustomed, by long use, to over-distension. Regular habits of life and plain but good nutritious diet are both essential, and the moral influence of regular mental exercise and physical employment is an important factor in successful treatment, since improvement in physical health is generally followed by mental improvement and increased receptivity to moral influences.

With regard to the period of detention, it is impossible to lay down any hard and fast lines. Each case must be considered on its merits, and be judged, as far as possible, by the mental and moral improvement resulting from treatment. Perhaps the best general rule is signature for twelve months on an understanding (with the Licensee of the Retreat) that, should everything be satisfactory during the first nine months of residence, the last three should be spent by the patient on leave of absence as allowed by the Act.

In dealing with periodic inebriates this course may require modification. Sufficient time should elapse to allow at least six or eight of the periods—that would otherwise be drunken outbreaks—to pass without resort to liquor. It is interesting, in the majority of cases of this type, to note the gradual diminution of restlessness in succeeding periods as they become modified by continual absence of alcohol.

It is absolutely necessary that all cases should be taught the vital importance of abstinence for the rest of their lives. There is little probability of any case ever being able to drink in moderation, even after years of abstinence, without running the risk of immediately or eventually reverting to excess.

R. WELSH BRANTHWAITE.

DISCRETE (*discerno*, I separate).—This adjective is used in reference to certain cutaneous eruptions, in which the spots or pustules are separate from each other; for example, discrete small-pox as distinguished from confluent.

DISCUTIENTS (*discutio*, I drive away). A term formerly used to designate local applications supposed to remove or disperse the congestion and effusion of inflamed parts, and the swelling of the skin over them. They included:—Friction; Pressure; Warm moist applications; Counter-irritation by blisters or liniments; Mercury and its preparations; and Iodine and its preparations.

DISINFECTION.—SYNON.: Fr. *Désinfection*; Ger. *Desinfection*.

DEFINITION.—Disinfection, in the proper sense of the term, means any process by which the contagium of a given disease is destroyed or rendered inert.

Disinfectants should not be confounded with *deodorants*, which merely cover or destroy offensive odours without necessarily affecting the contagia; and with *antiseptics*, which prevent the growth and multiplication of micro-organisms. See ANTI-SEPTICS; DEODORANTS; INFECTION; BACTERIA.

The known contagia consist of minute living bodies, and we infer that all contagia are of the same nature; consequently dilution lessens the chance of infection, but does not prevent a specific effect if the disease be taken. See INFECTION.

Contagia are apt to exist as clouds in air, water, or milk, instead of being equally distributed. When floating in the air (unlike gases) they are not absorbed by solid or liquid disinfectants which do not come into direct contact with them. Infected air should be removed by ventilation; it is futile to sterilise it by gaseous disinfectants, since to be effective these must be present in quantities incompatible with the existence of human beings. From this it follows that saucers of disinfecting fluids, or irritating vapours and gases in the sick-room, are a useless annoyance to the patient, except in so far as they may be desired as deodorants. The proper use of volatile disinfectants is the purification of walls, ceilings, and inaccessible places; and for this purpose, if possible, enough should be used to saturate the atmosphere, remembering that the virulent particles are most likely protected by being buried in a bit of epithelium or surrounded by an albuminous envelope. It is by no means easy to render an ordinary room gas-tight, and gaseous disinfectants therefore rapidly disappear. Even when aided by the presence of abundant moisture, free gases are much less effective for the disinfection of surfaces than solutions of the same gases applied as washes or in the form of spray.

The nature of the medium in which contagious particles are suspended has the most important bearing upon the selection of a disinfectant. This possibly explains in part the contradictory results obtained from experiments with disinfectants. Some media exhaust the active oxygen yielded by practicable quantities of certain disinfectants before the microbes are affected. The presence of albumen is found to protect septic germs to a considerable extent against the action of perchloride of mercury, permanganate of potassium, and chlorine, but has little or no influence upon the action of sulphurous acid and carbolic acid.

Schill and Fischer found that fresh tuberculous sputum was not disinfected in twenty-four hours by an equal volume of solution of perchloride of mercury, 1 in 500.

Increased experience will doubtless show which is the most appropriate disinfectant for each contagium in particular circumstances, but we can even now say that typhoid-bacilli and anthrax-spores are highly resistant to carbolic acid, and that perchloride of mercury is not the best disinfectant for tubercle-bacilli.

Disinfection should be carried out as near the source of the contagion as possible. Regular washing and change of clothing will do much to protect the purity of the air against contagium proceeding from the skin in such diseases as scarlet fever and small-pox. Discharges from the mouth, nose, and bowels, as well as the urine, should be received in vessels containing disinfecting solutions to cover them and prevent contamination of the air; and then larger quantities, or more concentrated solutions, as the case may be, should be thoroughly incorporated with the discharges before they are removed from the original vessels. For similar reasons dust should be damped before it is removed.

Of all agencies for preventing the spread of communicable disease, cleanliness is one of the most important. Sunlight and fresh air destroy the vitality of many bacteria. Moist anthrax-spores and tubercle-bacilli are soon killed by sunlight, but the action is confined to the surface of the medium

in which they exist. Pending the removal of accumulations of dirt a covering of coal-tar powder or chlorinated lime should be used to keep away flies which may convey contagium to fresh fields of action.

The ultimate fate of contagium is to be destroyed by putrefaction, and this appears invariably to destroy its specific infective power. In certain cases where real disinfection is impracticable, as in dealing with the accumulation of manure and litter from a number of animals suffering from cattle-plague, the natural processes may be hastened by stacking the material so that it shall 'heat,' or may even be destroyed by spontaneous combustion. Certainly putrefaction should not be delayed by small additions of disinfectants, which cannot accomplish the destruction of all noxious matter present. A coating of dry earth would prevent the access of flies and help to deodorise escaping gases.

SPECIAL DISINFECTANTS.—1. Heat.—Heat is the best disinfectant we possess. High temperature and length of exposure are to a certain extent mutually compensatory, but it appears that a temperature below 140°F . (60°C .) will not disinfect vaccine even with long exposure. Dry heat penetrates so slowly into fabrics that they are injured before a sufficiently high temperature is attained throughout, and has therefore now fallen into disuse.

Moist heat is a far more satisfactory disinfectant. Delépine found that anthrax-spores were killed by a temperature of 212°F . with the following exposures: in Manchester air, five hours; in the same air saturated with water-vapour, half an hour; in saturated running steam free from air, thirty-five seconds. Saturated steam is rapidly fatal to all pathogenic organisms, and penetrates clothing and bedding quickly, but may take some time to raise the temperature of sheltered articles, especially if wet. Modern steam disinfectors are made with a jacket-space, to which 'live' steam may be admitted, so that the internal chamber may be used as a simple hot-air chamber to dry the contents after steaming. With a proper apparatus the articles come out dry. Absence of air is a great help to the action of steam, and a vacuum-attachment is a valuable adjunct to a steam apparatus. Failing this attachment much air may be expelled by manipulating the escape and supply valves. The usual pressure is from 10 pounds (237°F .) to 20 pounds (259°F .), and the time 15 minutes. Rapid currents of low-pressure steam (at or slightly above 212°F .), used in a machine with a large outlet guarded by a light valve, sweep out the air and kill all organisms. The machines are cheap, but the boiler should be capable of filling the steam-chamber once in half a minute.

Steam instantly ruins leather, and fixes permanently all stains of blood or discharge. Stained clothing should be previously soaked in cold water, and the water afterwards disinfected by perchloride of mercury.

It has been abundantly proved that ordinary boiling and washing will completely disinfect the linen of a fever-hospital. The water should attain the full temperature of 212°F .

Nearly all the chemical agents used in disinfection are mutually incompatible, and therefore only one should be used at a time.

2. Carbolic Acid or Phenol.—This substance is poisonous and, though a strong antiseptic, is an untrustworthy disinfectant, probably inferior to

Cresol. The solution should not be of less strength than 1 in 20 of water and should also contain 15 per cent. of common salt. Large quantities are required. Anthrax-spores required to soak over two days in 5 per cent. carbolic solution to be destroyed, though blood containing anthrax-bacilli was almost instantly sterilised by a 1 per cent. solution. Combinations of carbolic acid are greatly inferior to the pure acid, and its solutions in oil and alcohol are inert. Carbolic-acid vapour is quite useless. Very small quantities of the liquid acid mixed with organic fluids enable them to remain fresh and resist decay for a long time. So little as one-fifth per cent. preserves milk. It is obvious then that small quantities of this disinfectant, instead of destroying contagium, may actually preserve its activity, when otherwise it would have succumbed to the action of natural agencies. This danger may accompany the limited use of any disinfectant that has a 'pickling' or preservative action when used in small quantity. Owing partly to the volatility of carbolic acid, which removes it in time, and partly to this peculiarity of its action, another danger attends its use. The acid may temporarily deprive a contagium of its infective power without permanently abolishing it, and the virulent properties may be regained whenever the acid has evaporated. This has been proved experimentally by Dougall, who found that vaccine mixed with pure phenol (1 in 50) regained its infective power after 10 days' exposure to the air.

For removing odour from the hands after working in the dissecting-room, a 1 per cent. solution of carbolic acid is superior in efficacy to permanganate of potassium, even when strong enough to stain the skin, and is also preferable to chlorinated lime.

3. Sulphur Dioxide.—The aqueous solution of this substance contains sulphurous acid. It destroys sulphuretted hydrogen thus, $2\text{SO}_2 + 4\text{H}_2\text{S} = 4\text{H}_2\text{O} + 3\text{S}_2$, and combines with ammonia. For aerial disinfection it is usual to burn sulphur in very large quantities or to use the gas liquefied by pressure. Even in presence of abundant moisture, the gas is unsatisfactory as a disinfectant. By volume 10·5 per cent. of the gas in a room failed to affect spores of anthrax, in twenty-four hours, whether wet or dry. Sulphur dioxide destroys the activity of dry vaccine on points very rapidly, and even when much diluted stops the amoeboid movements of living cells, kills vibrios, and acts deleteriously on vegetation. It bleaches vegetable colours, attacks iron, and injures cloth and leather—facts to be remembered in practical disinfection. 1 lb. of sulphur, when burned, produces 11·7 cubic feet of sulphur dioxide gas.

4. Chlorine.—Chlorine is most easily obtained from 'chloride of lime' or bleaching powder, by adding hydrochloric or sulphuric acid. Two parts of bleaching powder and three of strong hydrochloric acid may be taken. The acid should be diluted before use. Chlorine bleaches colours, attacks metals unless coated with vaseline, and injures cloth stuffs if long exposed. The most marked character of chlorine is its strong affinity for hydrogen, which enables it to break up compounds containing that body, and to set free in a nascent or active state the oxygen combined with hydrogen in water. It is, therefore, one of the most universally applicable and powerful deodorants in existence. When merely used as a deodorant, enough euchlorine (chlorine and chlorine dioxide) may be expelled from moist 'chloride of lime' by

the carbonic acid of the air for most purposes. In air saturated with moisture 0·3 per cent. chlorine kills all minor organisms that are freely exposed; but any cover, such as a piece of blotting-paper, prevents the result. For disinfection 0·5 per cent. should be used, which would imply about 3 lbs. of bleaching powder per 1,000 cubic feet.

5. Permanganate of Potassium.—This substance is non-poisonous, is free from odour, and its aqueous solution shows, by loss of colour, when it is exhausted. Permanganate of potassium is a true disinfectant, oxidising and destroying contagia as well as putrid matters; but the quantity required and the price render its use almost impossible, since it is necessary to employ sufficient permanganate to destroy both the medium or vehicle bearing contagium and the contagium itself. The solution should contain 5 per cent. permanganate after all chemical action has subsided. Condy's fluid is a solution of this substance in water. When the virulent liquid or matters are small in quantity, permanganate solution forms a capital recipient, and may stand by the bedside as a deodorant till required as a disinfectant. Permanganate has no effect in restraining the appearance of bacteria, or preventing the onset of putrefaction.

6. Acids.—The mineral acids and glacial acetic acid have all disinfecting power when used in sufficient quantity for a long time, but there are serious difficulties in the way of their use. Hydrochloric acid, 2 per cent. in solution, required ten days to kill anthrax-spores.

7. Formaldehyde.—This soluble gas is a valuable disinfectant for objects which are damaged by damp, chlorine, and heat, although it does not always succeed with anthrax-spores. It may be directly generated from methyl alcohol, but is more conveniently obtained by vaporising paraform, or by rapidly distilling formalin over a Bunsen burner—five ounces with 10 grains of borax per 1,000 cubic feet. The gas is aided by moisture and high temperature, and should be allowed to act for some hours. *Formalin* is a 40 per cent. solution of formaldehyde. Mackenzie adds five ounces of glycerin to each gallon of formalin to prevent polymerisation of formaldehyde before diluting to make a solution for the spray. Formalin does not injure wall-paper, colours, or fabrics, when applied as a spray in solutions of 2 to 10 per cent. strength, and has been much praised. It is said that acrolein, the liquid aldehyde of allyl alcohol, is a more powerful disinfectant than formaldehyde, but more experience is needed before its merits can be accurately determined.

8. Chlorinated Lime.—Bleaching-powder, commonly known as 'chloride of lime,' gives off chlorine easily, and this probably explains its disinfecting power. It is very cheap and manageable, and when fresh contains 34 per cent. of available chlorine. Mixed with water it corrodes cloth and sponge if they soak in it for any time. A 10 per cent. mixture with water is very potent, and even 1 per cent. is sufficient in many cases. Acids aid the action.

9. Perchloride of Mercury.—*Mercuric Chloride, Corrosive Sublimate*, the most powerful chemical disinfectant known, dissolves in 16 parts of cold water, is extremely poisonous, and corrodes metals. With albuminous matters it forms insoluble precipitates, which are prevented by addition of common salt, hydrochloric acid, or tartaric acid.

Precipitates must be prevented where disinfection has to be repeated, as they might be dangerous. The solution with tartaric acid is not stable, and should be freshly made. Solutions should be coloured to prevent accidents, and kept in glass or wood. A spray of 1 in 1,000, or washing with 1 in 5,000, killed resistant organisms in ten minutes. Enough should be used to leave 1 in 5,000 after any of the perchloride of mercury is precipitated by sulphur or albumen. After disinfection is finished, copious washing with water removes the remaining mercury salt. For utensils, clothing, &c., 1 in 2,000, and for excreta an equal bulk of 1 in 1,000 solution, are recommended.

10. Metallic Salts.—Metallic salts include:—*(a) Bichromate of potassium.* *(b) Sulphate of copper.* *(c) Chloride of zinc.* A 5 per cent. solution had no effect on anthrax-spores in a month, and 1 per cent. failed to kill *Bacillus prodigiosus* in forty-eight hours. *(d) Chloride of aluminium.* *(e) Ferric chloride*, which, if strong, liberates offensive fumes from animal matters, but is a fair antiseptic and preservative. *(f) Ferrous sulphate.* *(g) The waste chlorides*, from the manufacture of chlorine, contain $MnCl_2$, Fe_2Cl_6 , and free HCl, which cost next to nothing, and might be used for larger masses of filth or drains.

11. Ozone.—This body oxidises organic matter, and so destroys odours. Terebene and preparations containing terebene are good deodorants, and give rise to ozone.

12. Charcoal.—Charcoal condenses gases within its pores, where combustible gases are destroyed by the condensed oxygen. Contagium, unless in water, does not enter the pores, for, being particulate, it is not absorbed from the air as gases are.

PRACTICAL DISINFECTION.—In conclusion, a few remarks may be offered as to the modes of carrying out disinfection under circumstances in which it is commonly required.

1. Clothing and Bedding.—In dealing with the ragged and worthless articles of the poor, local authorities will generally find it most satisfactory to both parties to burn them and replace with new. If not burned, clothing may be steamed as described in this article under *Heat*, or well boiled with soda. Before coming to the washhouse they may be steeped in 5 per cent. carbolic solution, or perchloride of mercury (1 in 2,000).

2. Rooms.—The essential process for disinfecting rooms is thorough removal of dust and cleansing with soft soap and hot water. The walls and ceiling should be brushed, and wall-paper removed if possible. Furniture, if iron—a bedstead for example—is to be taken down and washed with carbolic solution, and removed from the room. Textile fabrics should be steamed or boiled, or spread out in the room, which should then be well sprayed with a solution of formalin, and closed until next day. Brushing with a 1 per cent. solution of bleaching powder several times repeated is used by Delépine for disinfecting rooms which have been occupied by tuberculous patients. In whitewashed rooms the walls should be scraped, and then washed with hot lime in addition to fumigation with chlorine.

3. Drains, Water-closets, &c.—Proper drains remove sewage so swiftly and completely that little or no sewage-gas is formed if ventilation is given. For bad drains carbolic acid, chloride of zinc, or

waste chlorides from the manufacture of chlorine, are fair palliatives. Excreta from cases of infectious diseases should be reduced to a liquid form, mixed with a large quantity of concentrated disinfectant, and set aside for at least one hour before being thrown into the water-closet. When a reliable amount of disinfectant is in these cases sent down the pipes, it is apt to corrode them unless it has been allowed to expend its energy on the excreta alone in the first place. If small quantities of disinfectants are poured down water-closets, it is better to mix them with the after-flush water which fills traps and basins, so that the little energy available may be devoted to the destruction of any slime adhering, or portions of organic matter retained. Permanganate of potassium is the most pleasant agent for this purpose, though expensive. When there are no water-closets, the excreta, in cases of cholera and typhoid fever, should be received in a vessel containing half a pint or more of a 1-in-20 solution of commercial hydrochloric or sulphuric acid, and then put, along with two ounces of chlorinated lime, into a covered stoneware vessel in the back yard. After a few hours the contents of this vessel may be thrown into the cesspool or upon the midden.

4. Dead Bodies.—Dead bodies, if putrid or bearing contagium, should be wrapped in sheets wet with 1-in-20 carbolic solution, or 1-in-1,000 perchloride of mercury; or, if confined, sawdust saturated with one of these solutions should be packed around them.

It is necessary clearly to keep in view the object desired when selecting disinfectants, deodorants, or antiseptics: whether it be destruction of contagium, merely 'pickling' and preserving, arresting putrefaction and fermentation, or deodorisation. From all that has been said it is evident that the different 'disinfecting' nostrums, applied as their inventors direct, can have little effect upon contagium, but may have more or less power in the other directions indicated.

JAMES A. RUSSELL.

DISSECTION-WOUNDS.—See POST-MORTEM WOUNDS.

DISSEMINATED SCLEROSIS.—See SPINAL CORD, Diseases of.

DISSOCIATED ANÆSTHESIA.—This term is applied to a condition in which the sense of touch is preserved, while sensation to pain and to temperature is impaired or lost. See SYRINGOMYELIA.

DISTOMA.—See ENTOZOA.

DISTOMA RINGERI vel **PULMONALE.**—Under this name the writer originally described in 1882 a species of fluke, the mature form of which inhabits the human lung, where it was first found in Formosa by B. S. Ringer in 1879. The ova of the parasite are associated with a peculiar form of recurrent hæmoptysis, to which the term *endemic hæmoptysis* has been applied.

SYMPTOMS AND PATHOLOGY.—The symptoms of disease associated with the presence of the *Distoma Ringeri* are a slight cough; the expectoration of a characteristic rusty-brown, viscid mucus; and at times hæmoptysis, slight or copious. The hæmorrhage occurs at irregular intervals during many years. The expectoration of rusty bronchial mucus is persistent, and in this the ova are readily discovered

with the microscope. These are pale brown bodies ($\frac{1}{300}$ in. \times $\frac{1}{500}$ in.), ovoid, double-outlined, operculated at the broad end, and containing protoplasmic globules having very active molecular movements. If the sputum is occasionally shaken up in fresh water there is developed in most of the ova, in the course of six weeks to two months, an active ciliated embryo which in time escapes by forcing back the operculum. It may be concluded from this that drinking-water, or a fresh-water animal acting as intermediary host, is the medium by which the disease passes from one human subject to another. See ENTOZOA.

Recent observations have shown that *D. Ringeri* may invade the brain and other organs besides the lung. When it affects the brain it gives rise to a form of Jacksonian epilepsy. The concurrence therefore of endemic hæmoptysis with cerebral symptoms should suggest the diagnosis of invasion of the brain by this parasite.

This disease has hitherto been found only in Japan, Corea, and Formosa, but its distribution is probably more extended.

The mature parasite measures from 8 to 10 mm. by 5 to 6 mm. It resides in cavities and burrows connected with the smaller bronchi, into which it discharges its ova. As many as twenty of these parasites have been found in one case.

TREATMENT.—Inhalations of sulphurous acid, sprays of turpentine, as well as koussou, quassia, and santonin, have been administered without much apparent benefit. If the brain be involved and if there be definite localising symptoms, surgical interference is advisable.

PATRICK MANSON.

DITTRICH'S PLUGS.—Brownish-coloured masses formed in the bronchial tubes and found in the expectoration in cases of gangrene of the lung, and of bronchiectasis. See BRONCHI, Diseases of; and LUNGS, Gangrene of.

DIURESIS (*δαί*, through; and *οὐρέω*, I pass water).—A free excretion of urine, whether induced naturally or artificially. See DIURETICS.

DIURETICS (*δαί*, through; and *οὐρέω*, I pass water).

DEFINITION.—Remedies which increase the secretion of urine.

ENUMERATION.—The following comprise the most important diuretics: Water; Milk; salts of Potassium, Sodium, and Lithium; Calomel; Alcohol, Nitrous Ether, solution of Ethyl Nitrite; Turpentine, Juniper, Copaiba; Cantharides; Digitalis, Strophanthus, Squill; Caffeine, Theobromine, and its compound with Salicylic Acid and Sodium termed 'Diuretin'; Tobacco; Scoparium, and Sparteine. The action of diuretics is often aided by brisk purgation, depletion, counter-irritation over the loins, and sometimes by the use of mercury.

ACTION.—The secretion of urine may be increased by anything which raises the blood-pressure throughout the system generally, or in the renal arteries locally. The systemic blood-pressure may be raised by cold to the surface, digitalis, squill, and tobacco. Digitalis and possibly other drugs have also a local action on the renal arteries, which are more readily affected by some drugs than other arteries in the body. Nitrous ether and solution of ethyl nitrite probably act as diuretics by dilating the renal arteries. The exact mode of action of the other diuretics is not determined; but common salt, nitrate of potassium, urates, and urea increase the flow of

urine even although the pressure in the vessels of the kidney is very low. It is therefore probable that they, as well as caffeine, theobromine, and very probably all bodies belonging to the xanthin group, stimulate secretion by acting on the nerves or secreting cells in the kidney itself.

USES.—Diuretics are employed to increase the flow of urine, and thus remove water or excrementitious products from the body. They are used in cases of general dropsy, or of accumulation of fluid in the peritoneum or pleura. In febrile conditions they are given to aid in the elimination of waste matter. They are also employed in order to render the urine more watery, and thus prevent the deposition of solids from it, and the formation of calculi in the kidney or bladder, or to redissolve such concretions when they are already formed. Digitalis and squill are most useful in dropsy dependent on heart-disease; the other remedies are more effective in dropsy dependent on disease of the kidneys or liver. The action of digitalis and squill is greatly assisted by the addition of a little Blue Pill; and when the kidneys are much congested or pressed upon from without by accumulation of fluid in the abdominal cavity, diuretics sometimes fail to act until the congestion has been relieved by depletion from the loins or the use of a brisk purgative, and the pressure removed by paracentesis. *See DROPSY.*

T. LAUDER BRUNTON.

DIZZINESS.—*See VERTIGO.*

DOCHMIUS DUODENALIS.—A synonym for the *Ankylostoma duodenale*. *See ENTOZOA.*

DOUCHE (Fr.).—**DEFINITION.**—A jet of water propelled against some part of the body through a *doccia* or pipe. The size of the jet of water, the degree of its impetus, and its temperature can all be regulated. A douche differs from simple affusion in its impact being more local, and in the force with which it is applied being often greater.

APPLICATION AND ACTION.—Douches of cold and of hot water, of vapour, and occasionally of gas, are employed; but those by far the most commonly used, except where there are hot natural waters, are of cold water.

The immediate effect produced by a cold douche on those who are unaccustomed to it is a feeling of shock, spasmodic shortness of breathing, increased action of the heart, and sometimes pain in the back of the head. Locally the first effect of a douche is to deaden the sensibility of the part to which it is applied; but if the douche be powerful enough, reaction comes on in about forty seconds. The reaction lasts for a time; but if the douche be kept up for three or four minutes, the pulse falls seven or eight beats, the deadening of sensibility returns, and the temperature of the part is greatly lowered; when the douche is withdrawn, reaction again takes place. This alternating effect increases the flow of blood in the part, and thus favours tissue-changes. The mechanical effect of the force with which the douche is applied must not be overlooked. In a given individual at a given moment the amount and kind of the reaction will depend on the temperature of the water, the length of the application, the force and size of the douche, and the part of the body to which it is applied.

Different portions of the body have different degrees of tolerance of the douche. Thus the extremities and the head bear it better than the chest,

and the chest somewhat better than the abdomen; and the posterior aspect of the body bears it much better than the anterior. Patients soon get accustomed to the cold. Warm douches produce less shock, and are more easily borne, but they are, comparatively speaking, little employed in private houses. An alternation of hot and cold douches, known by the name of *Scotch*, is a valuable means of obtaining easily a great amount of reaction; the term *Scotch douche* seems to have originated on the Continent, where it was first employed by a Savoy physician of the eighteenth century as synonymous with the English shower-bath, with which he became acquainted at Edinburgh.

The *Aix-douche* should be mentioned here, as its employment is not limited to Aix-les-Bains and any kind of hot water may be used for it. It is more correctly termed *douche-massage*, and is a combination of douche and massage, consisting in the methodical application by specially trained attendants of a hot douche, together with manual massage of the part against which the douche is directed. There are various slightly different modes of administering the *douche-massage* besides the original method employed at Aix-les-Bains; thus the patient may either be seated on a stool during the application, or be lying on an inclined table; treatment may be applied, as usually at Aix, by two attendants working at the same time on different parts of the body, or the administration may be confided to a single attendant. The local employment of the *douche-massage* is of great service in many chronic affections of the joints, but the application should be general when it is desired to obtain an effect on some gouty or other chronic disordered state of the metabolism.

Under particular circumstances it may be expedient to use a jet of steam, but this, of course, must be done with caution; and a jet of carbonic acid has sometimes been made use of for the neck of the uterus and the eye. Douches are sometimes applied locally to the perinæum, to the vagina, to the rectum, and to other parts; the rectal douche is called by the French *douche ascendante*, but in England the term *ascending douche* is sometimes used more vaguely to include perineal and other douches. In a certain sense what the English call *pumping* is a variety of the douche, and the *shower-bath* is in reality merely a multiplication of fine douches. The action of douches is more or less general according to the portion of the body to which they are applied. Thus the application of a douche to the head has the most general action, and that to the spine the next. In either case it is impossible to limit it very strictly, and there is a certain amount of affusion besides the direct douche. A douche, again, applied to one of the extremities may easily be localised; and a douche may be applied only to one part or to several parts of the body in succession.

Douches merely require a pipe with nozzles of various sizes in connection with a cistern at a certain elevation, or with a pumping machine; they can easily be improvised. Shower-baths can be procured with equal facility. A vapour-douche can be got by attaching a pipe to a vessel of boiling water. In the case of fine douches used for the eye, the water is propelled with sufficient force by the action of a caoutchouc bag worked by the hand. Carbonic acid is little used, and only where there is an abundant natural supply of the gas.

Perhaps 50° F. may be considered the average temperature of a cold douche, and its duration varies from a few seconds to a good many minutes. The course of douching will probably extend at least over a fortnight. As to the actual temperature of the water, the sensation it produces in the patient depends mostly on his condition. Thus water of 45° F. may feel ice-cold to one who has just quitted a hot bath. In like manner a douche of slightly heated water may appear quite warm when applied to a part cooled by a cold douche. The temperature of a douche should vary according to the condition of the patient.

USES.—As a general rule we may say that douches are only applicable in cases of *chronic* disease; that cold douches are most useful in constitutional diseases; and that warm douches, and the alternation of hot and cold douches, and the douche-massage are most suitable in local affections.

The cold douche, when it is employed with judgment, is found serviceable in chlorotic and hysterical conditions, in hysterical paralysis, and in oversensibility of the skin, with tendency to catch cold; and it forms a part of the treatment of phthisis at many of the sanatoria for this disease. As cold affusion on the head is very serviceable in infantile convulsions, so the application of a douche of cold water to the head is a calmative and hypnotic in some maniacal cases. It is, perhaps, not so much used in this way as formerly, because it has, like the shower-bath, come to be considered a sort of punishment to troublesome lunatics. Douches have likewise been employed as revulsives in chronic congestion of the liver &c.

Locally, douches are principally used in cases of old sprains, in chronic rheumatism or gouty thickenings of joints, in lumbago and in some neuralgias. The *Scotch* and the *Aix-douche* are far the most effective for the chronic articular troubles just alluded to. Douches might be used more extensively in private houses; still, as assistance is always required by the patient, public baths have advantages for their application. See BATHS.

JOHN MACPHERSON.
F. PARKES WEBER.

DRACUNCULUS (*dracunculus*, a little dragon).
See ENTOZOA.

DRAINAGE.—See PUBLIC HEALTH.

DRASTICS (δρᾶω, I act).—Violent purgatives. The principal drastics are:—Hellebore, Podophyllin, Gamboge, Elaterin, Scammony, Jalap, and Croton Oil. See PURGATIVES.

DREAMS.—See SLEEP.

DRIBURG, in Westphalia.—Strong Chalybeate Waters. See MINERAL WATERS.

DROITWICH, in Worcestershire.—Common Salt Waters. See MINERAL WATERS.

DROPSY (ὑδρᾶψ: from ὑδωρ, water; and ᾤψ, aspect, appearance).—SYNON.: Fr. *Hydropisie*; Ger. *Wassersucht*.

DEFINITION.—Accumulation of serous fluid in the subcutaneous cellular tissue, or in a serous cavity.

Dropsy is known by various other names, according to the portion of the body affected. When

confined to the subcutaneous cellular tissue it is termed *œdema* or *anasarca*; to the peritoneal cavity, *ascites*. The term is often limited to these two forms of the disease; and exudations similar to that of ascites in other cavities are termed *hydro-pericardium*, *hydrocephalus*, *hydrocele*, *hydrops oculi*, *hydrops articuli*, and *hydrothorax* or *pleural effusion*, according as they are contained in the pericardium, arachnoid, tunica vaginalis, eye, joint, and pleural cavity respectively.

PATHOLOGY.—The sole source of the fluid in the connective-tissue spaces is the blood which circulates through the capillaries. The removal of the fluid can be effected in two ways: firstly, by the lymphatics, and secondly, under special conditions, by the blood-vessels themselves. Owing to the extremely free anastomosis between the lymphatics, an alteration of absorption by these channels can rarely be made responsible for an excessive accumulation of fluid. We are therefore compelled in almost all cases to ascribe the production of dropsy to an increase in the transudation of fluid from the blood-vessels. What are the factors which influence this transudation? The capillary-wall consists of a single layer of endothelial cells in contact, but not in continuity, at their margins. Fluid may pass from one side of this membrane to the other in consequence either of differences of pressure on the two sides or of differences of concentration. In the first case the movement of fluid will take place by a process of filtration, in the second case by one of osmosis. It is also possible that there may be a direct transmission of material across the membrane in consequence of a secretory activity of the endothelial cells themselves; but of the existence of such a process we have no evidence.

We may deal first with the increased production due to the differences of concentration. Wherever activity or disintegration of tissue is going on, there must be a setting-free of chemical substances in the tissues, which will increase the extra-vascular concentration and so tend to produce a movement of fluid from blood-vessel into connective-tissue space. Since, however, the endothelial wall is, so far as we know, permeable to all kinds of diffusible substances, any such difference of concentration, due to local activity or injury, will be a temporary phenomenon, and will tend to disappear in consequence of diffusion between blood and lymph. Hence, although this factor may play some part in the causation of the local œdema resulting from injury, it cannot be of any great importance in the production of the more enduring dropsies.

On the other hand, the existence of a circulation through the blood-vessels involves a constant difference of pressure between the blood in the vessel and the fluids in the tissue outside. There will, therefore, always be a tendency to the passage of fluid outwards into the connective-tissue spaces, the amount of transuded fluid depending on the capillary-pressure, and on the permeability of the capillary-wall. The normal balance between the production and absorption of tissue-fluid may be upset by an increase in the capillary-pressure, or by an increase in the permeability of the vessel. The relative importance of these two factors varies according to the permeability of the vessel under normal conditions. In the capillaries of the liver, for example, a rise of pressure from 20 to 40 mm. Hg. increases the transudation of lymph tenfold. On the other hand capillaries of the connective

tissue of the skin and of the limbs are very much less permeable. The normal pressure in their capillaries gives only a very small transudation of lymph, and the pressure in them may be raised to 100 mm. Hg. without producing any marked augmentation in the tissue-fluid. Thus ligature of the femoral vein in a normal animal, or paralysis and consequent dilatation of the arteries of the limb, causes only a minimal increase in the production of tissue-fluid. Were it otherwise, the mere assumption of the vertical position in man would cause oedema of the legs, in consequence of the hydrostatic pressure of the column of blood between the legs and the heart.

The permeability of a vessel is, however, so intimately dependent on the vitality of the cells composing its wall, that any alteration or diminution in the vital condition of these cells increases the permeability of the vessel-wall. Thus they may be injured by thermal means as by scalding, by mechanical means as in the production of a crush or bruise, or by chemical means as by the injection or introduction of poisons into the connective tissues, or into the circulating blood. Examples of this last class of agent are furnished by the poison of the bee-sting, by the poison contained in shell-fish, which on ingestion give rise to nettle-rash, or by the injection of turpentine or croton oil, as in the experimental production of inflammation. When thus modified the vessels of the limbs will react to changes of pressure within them in the same way as the liver-capillaries. A slight increase of pressure, whether produced by changes of position or by obstruction to the venous outflow, increases at once the transudation of fluid from the vessels to such an extent that the absorbents are inadequate for its removal, and the excess of fluid remains in and distends the tissue-spaces, thus giving rise to oedema or dropsy.

We do not, however, need the presence of actual poisons for the production of this alteration in permeability. The life of the endothelial cell, as of all other cells of the body, is intimately dependent on the adequate and constant supply of oxygen and food. Thus stagnation of blood in the vessels, whether produced by obstruction of artery or vein, damages the vessel-wall and increases its permeability. The same effect is produced by the circulation of blood impoverished in its chief constituents.

It has already been stated that ligature of the femoral vein in a healthy animal is not followed by the production of oedema. If, however, the blood has been impoverished by frequent and excessive loss of blood before the experiment, ligature of the femoral vein causes oedematous swelling of the hind limb. In the same way the blood may be rendered more watery, and the capillary-pressure raised in all parts of the body by the intra-vascular injection of large quantities of normal salt-solution, without, in the normal animal, giving rise to any oedema of the subcutaneous connective tissues. If, however, the vessels be damaged by the previous injection of a solution of arsenic, widespread oedema of the skin is the result.

We thus see that for the production of the ordinary dropsy of the subcutaneous tissues there must always be some alteration of the vessel-wall. This alteration may be primary, and the sole cause of the increased transudation of fluid under the normal capillary-pressure, or it may be secondary to some factor, which is at the same time responsible for an

increased pressure within the blood-vessels. In many cases these changes are assisted by an alteration in the composition of the blood itself, rendering the transudation of its fluid parts more easy.

We are now in a condition to see how far these three factors are involved in the production of the various forms of dropsy known clinically.

Local Venous Obstruction.—A frequent cause of a localised dropsy is the existence of some obstruction to venous outflow from the part. Thus oedema of one leg may be produced by a growth pressing on the femoral vein, or by the occurrence of thrombosis in the vein itself. Simple aseptic ligature of the femoral vein in man may not cause dropsy. In most cases of venous obstruction, however, the capillary vessels are altered in consequence of the long stagnation of blood within them, or in consequence of the general malnutrition of the individual. The very production of thrombosis in a vessel postulates the existence of some deviation from the normal of the endothelial lining of the vessel, or the presence of poisonous substances in the blood.

General Venous Obstruction.—The commonest form of so-called mechanical dropsy is that associated with uncompensated heart-disease. In this case, owing to the incomplete action of the cardiac pump, the blood tends to accumulate on the venous side of the heart. The pressure in the arteries is apparently low, while that in the large veins is increased. It has been very generally assumed that this high venous pressure is responsible for the production of the dropsy in heart-disease. This assumption is certainly true for the organs, such as the liver, which drain directly into the big venous trunks near the heart, but it is very doubtful whether the pressure in the capillaries of the limbs is any higher than under normal circumstances. What is altered in such cases is the rate of circulation of the blood through the vessels, and it is the stagnation of blood rather than its increased pressure which must be made primarily responsible for the oedema that occurs in such cases. The diminution in vitality and consequent increased permeability of the capillaries render them unable to withstand the variations in pressure that occur in the normal life of the individual, and the mere increase in hydrostatic pressure, such as is caused by a dependent position of a limb, suffices to cause dropsy of that limb. The treatment of such a condition is obviously the treatment of its exciting cause, namely, the stagnation of the circulation. Relieve the heart by bleeding, paracentesis, or administration of digitalis, or strengthen the heart-muscle by the use of the latter drug or by graduated exercise, and the venous congestion will disappear and the capillary-wall be quickly restored to a normal condition.

Toxic Oedema.—Under this heading we may, with some justification, group cases of universal dropsy which are associated in many cases with renal disease, especially with acute or subacute parenchymatous nephritis. The type of Bright's disease attended with renal cirrhosis, or small contracted kidney, does not primarily give rise to oedema, any dropsy which may occur in the course of this disorder being dependent on changes in the heart, secondary to the contracted kidney. In those cases of Bright's disease which are associated with general dropsy, we find both blood and dropsical fluid poor in solids. The impoverishment of the blood has been ascribed by some authors to the loss of albumen by the urine,

but it is evident that such a loss could be easily made good in the food, and the hydræmia cannot be due to the abnormal output of a few grammes of proteid *per diem*. We must assume either that there is a general toxæmia, due to the deficient excretion of toxic bodies in the urine, causing an undue permeability of the vessel-wall, or, more probably, that both the renal and vascular changes are the simultaneous results of some morbid metabolism and consequent auto-intoxication.

Under the same heading of toxic œdemas we may place the severe cases of urticaria attended by general œdema which sometimes result from the ingestion of poisonous shell-fish. In milder cases of urticaria the increased susceptibility of the vessel-wall is shown by the wheals which may be produced by slight local irritation.

Inflammatory œdema.—In this form the increased transudation from the vessels is due in great part to the alteration in the vessel-wall excited by mechanical violence, by the presence of poisons, bacterial or otherwise, or by the action of the products of tissue-disintegration consequent on local necrosis.

œdema due to Lymphatic Obstruction. The only form of dropsy which we know to be associated with lymphatic obstruction is that occurring in elephantiasis. This type is of very slow production, and is associated with increased growth of connective-tissue, so that the hypertrophy of the connective-tissue is the most prominent factor in the enlargement of the limb found in such cases.

Dropsy in Serous Cavities.—Although developed apart and anatomically separate from the lymphatic system, the serous cavities may be regarded from the physiological standpoint as great lymph-spaces, since both dissolved and suspended material can be absorbed from these cavities into the lymphatics with extreme ease and rapidity. Here, as in the general connective-tissue of the body, the production of effusion depends on an upset of the normal balance between production and absorption of the serous fluid. Here, also, the chief responsible factor is in almost all cases an increased transudation. Owing to the great permeability of the vessels of the intestines and liver, simple mechanical obstruction of veins probably plays a greater part in the causation of ascites than it does in other regions of the body. Moreover, owing to the same factor, the effused fluid will be usually richer in proteids than that found in other parts of the body. A common cause of ascites is obstruction of the portal vein, due either, as in cirrhosis of the liver, to constriction of its hepatic branches by the contracting connective tissue, or to the pressure of growths or inflammatory exudation on the trunk of the vein itself. Even in cirrhosis, however, we generally find some sign of inflammatory changes in the peritoneum, and therefore probably also of the blood-vessels, in addition to the portal obstruction. In uncompensated heart-disease two factors will concur in increasing the peritoneal fluid, i.e. the greatly increased lymph-production in the liver, resulting from the high pressure in the inferior vena cava, and the obstruction to the flow from the thoracic duct into the great veins. The forms of ascites which are associated with changes in the peritoneum, such as tuberculosis or cancer of the peritoneum, must be placed in the class of inflammatory œdemas, although the changes in the sub-peritoneal tissue probably hinder at the same time the normal ab-

sorption of the effused fluid by way of the lymphatics.

Effusion into the pleural cavities is found in heart-disease as well as in inflammatory conditions of the pleuræ. In the normal animal it is impossible, experimentally, either by means of hydremic plethora or by venous obstruction to produce effusion into these cavities, although these same measures may cause at once a rapid and even fatal effusion, if the pleura and underlying vessels have been injured in any way. It is probable, therefore, that in the pleurisy of heart-disease the first essential is some injury of the vascular wall, in consequence of the deficient renewal of blood, the high pressure in the veins near the heart playing only a secondary part. The fluid which exudes into the serous cavities from the blood-vessels is, in the peritoneum and pleura, removed, at least in part, by a pumping action in the movements of respiration. The central tendon of the diaphragm contains spaces, the walls of which are alternately drawn apart and pushed together during its ascent and descent. Their separation draws up lymph from the abdominal cavity, and their compression forces it onwards through the lymphatic vessels. The same thing occurs in the costal pleura, during the respiratory expansion and contraction of the chest.

Hence the mere existence of a large pleural or peritoneal effusion tends, by obliterating these spaces, to prevent its own absorption, and it is often found that the removal by paracentesis of part of the fluid enables the rest of the effusion to be absorbed by the natural ways.

Effusion of fluid into the pleural and peritoneal cavities is of frequent occurrence in Bright's disease, associated with albuminuria. Its causation is probably the same as that of the general subcutaneous œdema which is present at the same time.

E. H. STARLING.

DROPSY, Treatment of.—The first thing to be considered in the treatment of dropsy is the removal of its cause, if this be at all possible. Where it is due to obstruction of a vein we must hinder, as much as possible, the accumulation of fluid in the vein, by preventing the part from remaining in a dependent position, while at the same time we try to aid the absorption of fluid by the lymphatics by gentle upward friction. Where it is due to obstruction of the circulation in the lungs, we must diminish, as far as possible, all obstruction to the pulmonary circulation by inhalations, emetics, and expectorants, pushed if necessary so far as to cause nausea or even vomiting. Where the obstruction is due to dilatation or valvular disease of the heart, we must lessen the work the heart has to do, and insist in bad cases on absolute rest in bed or on a couch, in which the patient's position can be altered, but which he should not leave. An absolute milk-diet, like that used in typhoid fever, is often of the greatest possible service, if it can be borne. At the same time we should aid the heart to contract more powerfully by the use of cardiac stimulants, such as alcohol and digitalis. Digitalis probably has a threefold action in cardiac dropsy, by strengthening the heart, by contracting the vessels, and by stimulating the kidneys. It strengthens at the same time that it slows the cardiac pulsations, and by making the heart contract more powerfully it keeps up the onward current of the blood more efficiently, and at the same time lessens the dilatation which tends to render the valves

incompetent. Besides its effect on the heart, digitalis has also an action on the vessels, causing the arterioles to contract, and probably reducing the dropsy in this way. For the contraction of the arterioles produced by digitalis is exactly the converse of the condition which occurs after division of the vaso-motor nerves, and which, as we have seen, produces dropsy whenever any obstruction of the circulation exists. It is not known at present whether digitalis also causes increased absorption, but it seems highly probable that it does so, because we know that it stimulates the vaso-motor centre, and stimulation of this part of the nervous system has been shown by Goltz to increase greatly the rapidity of absorption from the lymph-sac of the frog. In addition to this action of the heart and vessels generally, digitalis possesses a specific action upon the vessels of the kidney. It is a powerful diuretic, and by thus lessening the amount of water in the blood it will tend to increase the absorption of serous fluid either from the cellular tissue or serous cavities. When digitalis alone does not succeed, the addition of squill and of a small quantity of Blue Pill frequently increases its efficacy. Digitalis succeeds best in dropsy caused by valvular disease or dilatation of the heart. Sometimes *strophanthus*, *convallaria*, or *Adonis vernalis* may succeed when digitalis fails; but in general digitalis is best. It is not so useful in dropsy arising from renal disease, and here other diuretics are preferable. One of the best is spirit of juniper, given either as a mixture or in the form of "Hollands" gin. Spirit of nitrous ether, nitre, bitartrate of potassium, and scopolium are useful in all forms of dropsy. Caffeine is sometimes very useful. Theobromine, the active principle of cocoa, seems also to be useful (*see* DIURETICS). *Copaiba* occasionally succeeds where other diuretics fail. It seems to be most successful in dropsy due to cirrhosis of the liver. Hydragogue cathartics, such as compound jalap powder, elaterin, &c., which cause copious watery secretion from the intestines, employed judiciously, supplement the action of diuretics, and relieve or remove dropsy by removing water from the body, as well as altering its nutrition. Calomel seems to have an almost specific diuretic action, and is useful either alone or in combination with other diuretics or purgatives. In some cases of Bright's disease considerable relief has been obtained by the profuse sweating induced by vapour-baths, hot-air baths, *jaborandi*, or *pilocarpine*. When the dropsy does not yield to other remedies, the fluid must be removed by paracentesis in the case of serous cavities, and by small superficial incisions, with strict antiseptic precautions, in the case of the limbs.

T. LAUDER BRUNTON.

DROWNING, Death by.—Drowning is a mode of death from asphyxia, caused by submersion of the mouth and nostrils in a liquid medium, so that access of air to the lungs is cut off until death takes place. The effects upon the lungs of asphyxia thus produced differ from those which occur in other forms of asphyxial death, such as hanging and strangulation. In the one case the access of air is simply cut off; in the other, the inspiration of an irrespirable medium is superadded; the ensuing changes in the air-passages and lungs constitute most valuable *post-mortem* indications of death from drowning. Death may take place in water and yet not by drowning. Syncope, or shock from the head or abdomen striking against some hard solid body at the

moment of submersion, may suddenly cause death. In such cases no attempts at respiration are made while under water, and therefore none of it is drawn into the lungs.

The specific gravity of the human body is slightly greater than that of water, so that an individual who falls into deep water tends to sink. The involuntary movements of the limbs, made in attempts at self-rescue, are sufficient, however, to bring the body, at least momentarily, to the surface, when air along with water is forcibly drawn into the lungs. Sooner or later, according to the strength and the natatory power of the victim, the body is again submerged. In fatal cases this alternation may be repeated several times before the body finally sinks. The submergence is then permanent until the gases of putrefaction accumulate in sufficient amount to make the specific gravity of the body less than that of the medium in which it lies. This usually occurs within a week, when the body again comes to the surface. The bodies of men, unless there is excess of abdominal fat, float in the prone posture with the head and limbs below the water level. The bodies of women, on account of the adipose tissue of the breasts and abdomen, float in the recumbent posture.

Post-mortem appearances.—If the body be examined a few hours after death, there is, as a rule, little difficulty in determining whether death was due to drowning or not. On the other hand, when the body has remained in the water until putrefactive processes are in progress, the difficulty may be insuperable.

Externally.—The surface is pallid, the face appears tranquil, the eyes and mouth being partly open. The skin often has the appearance known as goose-skin, and, if the body has lain upwards of twelve hours in the water, that of the hands and feet is sodden by imbibition. The most important external sign is the presence on the mouth and nostrils of a fine froth (possibly blood-stained), composed of air, mucus, and the medium in which drowning took place; if the body remains three or four days in water, the froth disappears. Exceptionally, fragments of weeds, or other small objects, may be found tightly grasped in the hands.

Internally.—On opening the thorax the lungs, grey in colour, with reddish staining, are seen to be very voluminous, a condition known as ballooning, partly due to infiltration of the lung-tissue with some of the medium in which drowning took place, and partly to true oedema. The lung-substance is inelastic and pits on pressure. On section, fluid and froth, resembling that found on the lips, exude from the divided air-passages. Minute sub-pleural hæmorrhages are not infrequently present, and the pleural cavities may contain fluid. The stomach often contains water or other liquid which was involuntarily swallowed during attempts at respiration. Occasionally, some of this fluid is forced by vital action into the intestines. With one exception, the remaining appearances are those met with after death from asphyxia. The exception is constituted by the blood, which is not only dark-coloured and fluid, as is usual after death from asphyxia, but is also diluted by the imbibition of water from the stomach and lungs. The duration of submersion which is compatible with resuscitation is variable and is to some extent dependent upon subsequent events. In recent cases, if efficient treatment, with all accessories such as hot baths and

blankets, is commenced immediately after the body is withdrawn from the water, recovery is possible after five minutes' submersion; with less favourable conditions, two minutes may be regarded as the limit. The exceptional occurrence of total insensibility from syncope, or shock at the moment of submersion, tends to prolong the recovery-limit because, no attempts at breathing being made, water is not drawn into the lungs, which therefore do not become sodden and inelastic; consequently they more readily respond to the stimulus of artificial respiration.

In England and Wales during the decennium 1889 to 1898 the total number of deaths from drowning was 32,151. Of these 26,203 were accidental; 5,740 were suicidal; and 168 were homicidal.

TREATMENT.—The treatment of the drowned consists firstly in removing as much as possible of the frothy fluid from the air-passages, and secondly in supplying air by artificial respiration, together with the application of means to counteract the abstraction of body-heat.

To remove the frothy fluid it is not sufficient to turn the body on the face for a few seconds and then to press on the back as is sometimes advised, as the frothy fluid can only be squeezed out in small quantities, and until this is efficiently removed only a small amount of air can be introduced. The patient should be placed on his face, a procedure generally followed by the escape of a large quantity of fluid from the stomach and a small amount from the lungs. On replacing the patient on his side, preferably the right side, a small amount of air takes the place of the fluid which has been expelled. The body should be then again rolled over on to the face and the back pressed upon, when more fluid comes away to be replaced by more air as the lateral position is again resumed. This procedure must be repeated many times. Bowles considers it dangerous to change the side acted upon, as the fluid in the still loaded lower lung will gravitate into the opposite side if the lung containing it be placed uppermost, and entering the then lower and clearer lung will be churned afresh into foam by the ingoing air. Long and steady persistence is essential. During this process a partial use of Silvester's method is of service, that is, the extension of the upper arm while the body is in the lateral position, although, as above stated, the side should never be changed.

When the smallest sign of spontaneous respiratory movement is observed, the artificial respiration may be discontinued, though the patient must be carefully watched to see whether the automatic movements increase in force.

If assistance be available, other measures should be simultaneously adopted. The wet clothes should be removed and the body wrapped in warm dry clothes obtained from bystanders, pending the arrival of warm blankets, hot bottles or bricks from the nearest house. Assiduous friction of the extremities from below upwards, and the administration of stimulants *per rectum*, and of strychnine hypodermically, should not be neglected. See **ARTIFICIAL RESPIRATION**; and **RESUSCITATION**.

J. DIXON MANN.

DROWSINESS.—Inclination to sleep. See **SLEEP**, Disorders of.

DRUG-ERUPTIONS.—SYNON. : *Dermatitis medicamentosa*.

Many medicines produce various eruptions or

discolourations of the skin, owing to idiosyncrasy, irritability of the gastro-intestinal mucous membrane, or defective power of elimination on the part of the patient; or from the largeness of the dose or long-continued administration of the drug.

These toxic lesions may be divided into three classes :—(1) Those inflammatory eruptions which are *special to the drug*, such as the agminated pustular eruptions produced by compounds of bromine or iodine (see **IODISM**). (2) Various inflammatory eruptions *not distinctive* in themselves, their nature being only recognisable from the surrounding circumstances. (3) *Discolourations* of the skin, such as are produced by arsenic and nitrate of silver. See **PIGMENTARY DISEASES OF THE SKIN**.

The great bulk of medicinal eruptions are either erythematous or urticarial, but the kind of eruption produced by the same drug often varies according to the individual who takes it. With the exception of the special eruptions due to iodides and bromides, the lesions are probably due to irritation of the gastro-intestinal mucous membrane acting reflexly on the vascular nerves of the skin; for in many instances the dose has been too small, and the eruption has followed too quickly on the ingestion of the drug, for the rash to be due to the absorption and circulation of the substance itself. In some instances a direct toxic action on the vaso-motor nerves, central or peripheral, is probable. With the exception of the special bromide- and iodide-eruptions, the diagnosis cannot be made from the aspect of the rash. The following lists do not pretend to be quite complete, but represent all the chief eruptions and the drugs which produce them after absorption :—

Local effects from the application of drugs to the sound skin are described under **DERMATITIS VENENATA**.

List of Drugs and of the Rashes which they produce.

Antifebrin (Acetanilide).—A slate-coloured anemia, simulating venous cyanosis, but probably due to a dyeing effect as in aniline-poisoning. Exalgin and monobrom-acetanilide have produced a similar effect—usually from large doses.

Antipyrine (Phenazone).—Erythema, morbilliform, is the most common, but it may be scarlatiniform, diffuse, patchy or acuminate papules. Urticarial, purpuric, and less often vesicular and bullous eruptions. It is sometimes localised, e.g. on the back of the hands.

Arsenic.—Erythema—diffuse and erysipelas-like, papular, acuminate or morbilliform, less often scarlatiniform; and urticaria, vesicles, pustules, herpes zoster, furuncles, and gangrene, keratosis palmæ et plantæ, general sepia pigmentation.

Belladonna.—Erythema—diffuse, scarlatiniform or patchy.

Benzoate of sodium.—Erythema—patchy and papular—rare.

Borax.—A scaly eruption like psoriasis—Eczema? A peculiar dryness of the skin and mucous membrane, and sometimes shedding of the hair.

Boric acid.—Erythema, followed by vesicles.

Bromine-compounds.—Pustular, erythematous, urticarial, bullous and squamous, in the same order of frequency.

The majority are pustular. These may be discrete and are then acneiform or furuncular, or they may be confluent and then anthracoid, but

are soft instead of brawny, and the process is superficial and largely epithelial, and seldom scars. These agminated pustules on a soft raised red base are very characteristic and recognisable even without a history of administration of bromide. Although usually due to large doses or to long-continued administration, very small doses may produce this form in infants and in patients with renal or heart-disease interfering with elimination, and it continues to come out for days or even weeks after the drug has been stopped. It has a predilection for scar-tissue, and may be produced in suckling infants whose mothers are taking the drug. Arsenic is to some extent prophylactic and hastens involution. Intestinal disinfectants, such as salol and β -naphthol, are also said to have a prophylactic effect.

Cannabis Indica.—Vesicles (one case).

Cantharides.—Erythema.

Capsicum.—Erythema—general papulo-vesicular eruption (one case).

Chinolin.—Erythema, when given in typhoid fever (six out of twenty cases).

Chloral hydrate.—Erythema—dusky-red, diffuse, patchy, papular, and scarlatiniform, involving oral and pharyngeal mucous membrane; urticaria, vesicles, bullæ, purpura, shedding of nails, axillary abscesses, and persistent high temperature. In rare cases vesiculation and deep ulceration over points of pressure.

Chloralamide.—Scarlatiniform erythema (one case).

Chlorate of potassium.—Erythema—papular; discolouration, bluish or icteric.

Chloroform-inhalation.—Purpura.

Cod-liver oil.—Vesicular, pustular (one case of each).

Cocaine.—Erythema—general.

Copaiba.—Erythema—diffuse, patchy, or scarlatiniform; miliary papules, petechiæ, vesicles, bullæ, urticaria.

Cubebs.—Erythema—papular, miliary, or morbilliform.

Digitalis.—Erythema—scarlatiniform, papular; universal erythema, followed by urticaria in huge plaques, complete shedding of skin, hair, nails.

Dulcamara.—Erythema, urticaria.

Ergot.—Erythema, petechiæ, vesicles, bullæ, pustules, boils; loss of scalp-hair and nails.

Guarana.—Urticaria (one case).

Guaiaicum.—Erythema—miliary.

Iodine-compounds.—Pustular, vesicular or bullous, purpuric, erythematous and urticarial, anthracoid, gangrenous, sarcoma-like, vegetating, and subcutaneous infiltrations. The pustular are the most common, and may be discrete or confluent, like bromide-eruptions, but tend more to be bullous than their bromide-analogues. Many are only apparently bullous, being solid when pricked. Tumours like sarcoma are on record; in one case ending fatally, generalised sarcomata were simulated. The nodules are apt to break down into ulcers. Like those due to bromide the lesions continue to appear for some time after the drug has been stopped, and the eruptions have appeared in suckling infants. Renal and cardiac disease are also predisposing factors.

Iodoform.—Erythema, urticaria, and purpura occur after absorption, and vesicular eruptions like severe eczema often ensue when iodoform is applied to the sound skin of predisposed persons.

Lactophenine.—Large erythematous plaques.

Mercury.—Erythema sometimes simulating erysipelas after ingestion; scarlatiniform after injection of calomel; bullous after an intra-uterine injection; pityriasis rubra after inunctions, are examples of eruptions from different modes of administration; vesico-pustules, urticaria, purpura, and ulceration are other forms which have been observed.

Morphine.—Erythema—usually morbilliform with severe itching and pricking, also scarlatiniform and conically papular or papulo-vesicular (only one case, after hypodermic injection, and that scarlatiniform).

Phenacetin.—Erythema—acuminate and flat papules (rare).

Phosphoric acid.—Pemphigoid bullæ (rare).

Phosphorus.—Bullæ, purpura, icterus, but only in poisonous dose.

Quinine.—Erythema—diffuse, patchy, or scarlatiniform, morbilliform or acuminate papules, urticaria, vesicles, bullæ. Fractions of a grain will produce an eruption in predisposed persons, whether given by the mouth, rectum, or skin, but as a rule the severer lesions follow large doses. Persistent exfoliation has been observed after the eruptions, which may be general or confined to one region.

Rhubarb.—Hæmorrhagic, bullous, pustulo-bullous 'recurrent scarlatiniform eruption.'

Salicylic acid and Salicylates.—Erythema—diffuse or patchy, with oedema, scarlatiniform, morbilliform and urticarial, less frequently vesicular, bullous or purpuric; salipyrin produced an infiltrated red oedema in one case, and salol an urticaria, but the latter is quite exceptional.

Santonin.—Urticaria.

Stramonium.—Erythema.

Strychnine.—Scarlatiniform erythema (rare).

Sulphonal.—Erythema—diffuse and macular, scarlatiniform, vesicular; purpura with hæmaturia in urine.

Tannin.—General urticaria after application to pharynx.

Tar.—Erythema—locally, an acneiform eruption.

Terebene.—Erythema—papular.

Toxins.—Such as Tuberculin and Diphtheritic and anti-streptococcic serums often produce erythema—scarlatiniform, morbilliform, patchy or diffuse; urticaria is less common.

Turpentine.—Erythema—diffuse morbilliform and minute papules and vesicles with intense itching.

List of Eruptions and of the Drugs which produce them.

Diffuse or patchy erythema.—Antipyrine, arsenic, belladonna, benzoate of sodium, boric acid, bromides, chloralamide, chloral hydrate, cocaine, chrysarobin, copaiba, iodides, lactophenine, mercury, quinine, salicylic acid, stramonium, tar.

Scarlatiniform erythema.—Antipyrine, arsenic, belladonna, chloral hydrate, copaiba, digitalis, iodoform, mercury, quinine, rhubarb, salicylates, strychnine, benzoate of sodium, bromide of nickel, sulphonal.

Papular or morbilliform erythema.—Antipyrine, arsenic, bromides, chloral hydrate, cubebs, guaiaicum, morphine, phenacetin, quinine, salicylates, terebene, turpentine, toxins.

Nodose erythema.—Bromides and iodides.

Urticaria.—Antipyrine, arsenic, bromides, copaiba, digitalis, iodides, iodoform, morphine, quinine, resins, salicylic acid, salol, santonin, toxins.

Vesicles.—Antipyrine, arsenic, cannabis indica,

capsicum, chloral hydrate, cod-liver oil, copaiba, ergot, iodides, morphine, quinine, salicylic acid, sulphonal, and turpentine.

Bulle.—Antipyrine, bromides, cannabis indica, chloral hydrate, copaiba, ergot, iodides, mercury, morphine, phosphoric acid, quinine, rhubarb, salicylates.

Pustules.—Arsenic, bromides (confluent), chloral hydrate, cod-liver oil, iodides (isolated), mercury, salicylic acid.

Purpura.—Antipyrine, chloral hydrate, chloroform inhalation, iodides, mercury, quinine, salicylic acid, sulphonal.

Pityriasis rubra.—Bichromate of potassium, mercuric inunction.

Psoriasis (?).—Borax, bichromate of potassium.

Eczema.—Bicarbonate of potassium, bromides, chrysarobin, iodoform.

Gangrene.—Arsenic, ergot, iodides, quinine.

Persistent desquamation.—Quinine.

Cyanosis.—Antifebrin, exalgin, monobrom-acet-anilide.

Abscess.—Quinine.

Furuncles.—Arsenic, bromides, ergot, quinine.

Keratosis palmae et plantae.—Arsenic.

Pigmentation.—Arsenic, nitrate of silver, picric acid.

Herpes Zoster.—Arsenic.

H. RADCLIFFE CROCKER.

DRUG-HABITS.—See HABITS.

DUBINI'S DISEASE.—See CHOREA, Electric.

DUCHENNE'S PARALYSIS.—See PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

DUCTUS ARTERIOSUS, Patency of.—See HEART, Malformations of.

DUMBNESS.—Persons incapable of producing the elementary vocal sounds and pronouncing words are said to be dumb. This condition may be due to partial or complete deafness, congenital or acquired in early life; the vocal organs are usually normal, but dumbness arises from their non-employment. The absence of speech may also be caused by structural cerebral defect or disease, or by such nervous changes as often accompany epilepsy or follow severe illness.

The Census Report 1891 shows, per million living of corresponding sex:—

	Males.	Females.
Deaf and dumb . . .	548	434
Deaf only . . .	444	592

Among deaf-mutes, as is always the case with the various forms of congenital defect, males far outnumber the females, and the male rate of deaf-mutes per million living is higher than the female at all ages. With the advance of age the deaf-mutes die off more rapidly than their fellows of the same age.

Congenital deaf-mutes are generally the offspring (1) of congenital deaf-mutes; and (2) of consanguineous parents.

When one parent is a congenital deaf-mute, one-tenth of the children are deaf; and when both parents are thus affected, one-third are born deaf. Consanguineous marriage in a family in which there is already congenital deafness increases the probability of deafness in the offspring; the family peculiarities, whatever they are, become increased. Such marriages may also give rise to dumbness as a result

of the cerebral deficiency so common among the offspring.

Cases with speech-deficiency may be classed as: (1) deaf-mutes; (2) speaking deaf, or persons with semi-speech; including children who have learned to speak and then lost the sense of hearing at five to seven years of age; and others with some power of utterance, but whose use of words and language is disconnected; (3) persons who hear but are dumb or semi-mute; these are generally also feeble-minded from congenital cerebral deficiency.

When a child in his third or fourth year is found to be without speech, questions arise as to the probability of permanent dumbness. In such cases, it is necessary to investigate the hearing and mental ability. The child's physical growth and general health must be accurately ascertained; defects in the development of the head and features are especially noteworthy, as indications of some coincident defect of the brain. See CHILDREN, Training of.

Mental ability is assessed by observation of the child's co-ordinated movements and response under impressions received by sight, muscular sense, or otherwise. It often happens that a child who has not been previously trained, and usually makes no response in words, will respond to distinct impressions made one at a time, and even use a word or two as well as action in expressing what he has felt, seen, or heard. Thus, if one-ounce weights are placed in his hand, added one after another, he at last drops them; if the experiment is repeated he may say 'heavy.' If a test-tube is placed in his hand, then one containing warm water substituted, he may say 'hot.' It is not unusual to find that, when speech is almost absent, hearing is thought to be defective, whereas the brain-faculties necessary to 'attention' are often imperfect or uncultivated; in feeble-minded children response is usually delayed, slow, and inaccurate. Spreading action on stimulating the child, expression of emotion, as well as rapid crawling at sight of a toy on the floor, all indicate cerebral spontaneity capable of control. Sometimes such spreading movements are mostly in the large muscles and joints, while the digits move but little; thus, the child may sweep objects off the table by flinging his arms, and fail to handle his spoon. Co-ordinated movements of the eyes under control, and of the hands and fingers in exact imitation of those shown, indicate some faculty for attention and imitation of the facial movements used in speech. Sight and hearing should be carefully examined.

Hearing-power is perfect when the child replies to a whispered question at a distance of twenty-five feet, either ear being stopped alternately, while the face of the speaker is not seen. Unless he can hear an ordinary voice at a distance of two or three feet from the ear, the child is unable to acquire language by the ordinary methods of instruction. Any degree of deafness is important, therefore all causes of it should be looked for and treated; attention to the healthiness of the teeth and mouth helps to promote a healthy throat. Myopia with deafness seriously interferes with any child's school-education. In cases of feeble brain-faculty the hearing power cannot be easily diagnosed; it may be impossible to determine whether sounds reach the brain and fail to produce response, or whether the organ of hearing be defective. Careful examination must be made by the aural speculum and rhinoscopy; all causes of mouth-breathing, and

obstructions in the nose or from post-nasal adenoids, should be removed. This is equally important with those partially dumb, the feeble-minded, and imbeciles. Any degree of hearing, however slight, is valuable in enabling the child under training to acquire greater accuracy and precision in articulation and intonation.

The *palate* is often narrow or highly arched in deaf-mutes. In any child this indicates defective development secondary in importance only to that of the cranium; but a badly formed palate, except when cleft, is perfectly consistent with good speech; the narrow palate often co-exists with nasal and faucial obstructions. Among thirty-one boys and twenty girls deaf and dumb, in five cases the palate was vaulted or arched, and in two others narrow, and of these five were congenitally deaf.

Some children with little or no speech, but not deaf, suffer from myxœdema, syphilitic disease of the brain, or epilepsy—conditions calling for special treatment as well as training. It may be added that excessive administration of bromide of potassium greatly lessens mental activity and readiness of speech.

Dumb children are apt to be solitary in their habits and mentally introspective, thinking too much of themselves and any slight ailment or discomfort. If cut off from companionship they loaf, and degenerate mentally from inertness and want of opportunities for expressing their thoughts or receiving impressions from other persons. Sometimes they are noisy, and at the same time unaware of the discomfort they cause to other persons. They need elementary brain-training at an early age, like all children. Whenever possible some elementary exercises in proper breathing should be commenced in play at the age of four or five years, as preparatory to speech-training (see CHILDREN, Training of). Breathing exercises improve both the respiratory and the vocal powers, which are apt to be deficient in the dumb; they develop the lungs and improve health.

Special training in speech should be used at six or seven years old. Exercises may be employed in imitating the expression of sounds and words from the teacher, as the child looks at her face in a good light; this gives practice to the muscles of expression and articulation, the lips and the tongue, and to some extent strengthens them, while their nervous apparatus is co-ordinated and evolved. The child must learn to bring the lips together firmly, to close the teeth, and to direct the tongue as seen in the formation of articulate sounds; this also leads to a better use of the muscles of the tongue and cheek in mastication and deglutition. This is called 'lip-reading,' but is in fact a '*pars pro toto*' name for the reading of the various visible movements of all the organs of speech. The power to articulate, added to the faculty of vocalising and regulating the voice, may enable the deaf pupil to speak to his fellows and understand what they say.

Sign-language by the hand and gesture is not found to be compatible with the oral method; two different vehicles for thoughts being thereby established which, combined, retard mental development.

The primary consequence of want of hearing-power is dumbness; secondary ones are want of the natural and most important channels and means of intellectual development, with mental isolation. The absence of hearing-power does not necessarily correspond with the absence of intellectual capacity; but this capacity must not be allowed to lie idle, and requires *only* culture by the accessible channels—

viz. the senses which are intact—as otherwise mental degeneration and deficiency must result from this neglect.

General activity in occupation and, as far as possible, organised games with other children are advisable.

Deaf adults would do well to learn 'lip-reading'; their speech is often to a great extent influenced by their want of hearing, inasmuch as it becomes less distinct, and is either too loud or too soft, because the deaf person does not hear his *own* voice and is thus deprived of the guiding control of his speech. Special voice-training and vocal instruction will enable him to regulate his voice so as to be understood, just as lip-reading will help him to understand, by sight, the speech of others.

FRANCIS WARNER.

DUODENUM, Diseases of.—See **INTESTINES, Diseases of.**

DUPUYTREN'S CONTRACTION.—SYNON. Contraction of the Palmar Fascia; Fr. *Rétraction de l'aponévrose palmaire*; Ger. *Retraction der Palmaraponeurose*.

DEFINITION.—This affection is characterised by a slowly progressive and painless thickening and retraction of the cutaneous and fascial structures of the palm, resulting in permanent flexion of the digits.

ETIOLOGY.—The deformity is rare before the age of forty, and occurs much more frequently and with greater severity in men than in women. It usually begins very insidiously and without apparent cause, but appears in some instances to be occasioned by often-repeated pressure on the palm, as in the use of a spade or other implement. The evidence that it is of gouty or rheumatic origin does not appear conclusive, but undoubtedly the tendency to the affection may be inherited.

MORBID ANATOMY.—The fibrous nodules and bands which develop in the palm and on the fingers do not exactly correspond with the palmar fascia and its radiations to the fingers. They result, according to Anderson, from 'an inflammatory hyperplasia commencing in the skin and subcutaneous tissue of the palm, involving the fascia secondarily, and replacing the adipose connective tissue which serves as an elastic cushion for the palmar surface of the hand and fingers.' It is only in aggravated cases of very long standing that a secondary shortening of the tendons and alterations in the articulations of the fingers occur.

SYMPTOMS.—In a typical case of Dupuytren's contraction the first symptom is the presence of a hard nodule or band in the palm over the position of the metacarpo-phalangeal joint, usually of the ring-finger. The overlying skin is slightly thickened and adherent to the band beneath. Very slowly the hardness increases and extends towards the finger, puckering the skin and preventing full extension of the metacarpo-phalangeal joint. The contraction increases and involves the joint between the first and second phalanges, so that eventually the finger is drawn immovably into the palm, while the last joint usually, but not always, escapes. Sometimes the little finger is the first to be affected, and nearly always it is involved soon after, and nearly as markedly as, the ring-finger. Of the remaining digits the middle finger rarely escapes, but is usually not so contracted as the two inner ones. The

thumb and index, however, are often not affected; but in extreme cases they also may be involved, the thumb being drawn into the palm by fibrous bands extending over the thenar eminence. The deformity is usually bilateral, but not symmetrical except in severe cases of long standing, so that, while in one hand certain fingers may be markedly contracted, the only evidence of disease in the other hand may be the presence of a hard nodule or band in the palm.

TREATMENT.—In cases in which there is at the most only a slight degree of contraction, massage and passive movements should be practised, the hardened skin of the palm softened by inunction with lanolin, and a simple digitated splint, maintaining the fingers in the extended position, worn at night. In the extreme degrees of the deformity sometimes met with in old persons no treatment is likely to be of any service. In the majority of cases presenting degrees of deformity between these two extremes, operation is attended with a fair measure of success if the rather tedious after-treatment is carefully carried out. The simplest operation consists in the subcutaneous division of the fibrous bands in the palm and on the fingers with a sharp-pointed tenotome. Multiple punctures are usually necessary, and care must be taken not to cut outwards through the skin, or so deeply as to injure the tendons. In many cases, however, better results are obtained by excising the fibrous bands through suitable longitudinal incisions. The skin is preserved as far as possible; but if any part is so adherent to the fascia as to require removal, the gap should be filled by skin-grafts. After the operation a digitated splint must be worn for four or five weeks, the position of the fingers being usually capable of considerable further improvement by frequent readjustment. For a further period of several months the splint should be worn at night, and massage and passive movements perseveringly practised.

RAYMOND JOHNSON.

DURA MATER, Diseases of.—See MENINGES, Diseases of.

DUROZIEZ'S SIGN.—This term is applied to a double murmur which can be produced in cases of aortic incompetence by pressure on the carotid or any large artery. See HEART, Diseases of the VALVES.

DYNAMITE-POISONING.—Poisoning from dynamite may be due to the gases evolved when it explodes, to the fumes when it is burnt, and to swallowing the substance itself.

1. The explosion of dynamite is accompanied by the production chiefly of carbon dioxide and nitrogen, but also of carbon monoxide. In confined spaces, such as mines, this mixture of gases often gives rise to disagreeable symptoms, such as dyspnoea, prostration, and giddiness, which pass off on removal to a purer atmosphere. The effects are sometimes more severe, and are characterised by asphyxia. Recovery may be retarded by bronchitis and pharyngitis.

2. When dynamite burns, its fumes are pungent, suffocating, and sulphurous, and exposure thereto causes vomiting, headache, dyspnoea, cyanosis, and unconsciousness. The symptoms may not come on directly, but may be delayed for some hours. The chief dangers are from pulmonary oedema, engorgement of the lungs, and bronchitis.

3. The swallowing of dynamite is followed by

violent pains in the head and abdomen, by vomiting and purging with bloody stools, by excessive arterial pulsation, profuse perspiration, cyanosis, dyspnoea, great muscular weakness or palsy, collapse, with coldness of the extremities and unconsciousness.

Poisoning by dynamite is usually accidental, but cases where it has been used with homicidal intent are recorded.

The chief morbid appearances are marked redness of the gastro-intestinal mucosa, the presence of sanio-serous fluid in the cerebral ventricles, and oedema of the lungs.

As dynamite is a mixture of nitroglycerin and an infusorial earth, the presence of diatoms and other minute organisms would be strong evidence of its use or administration.

Persons poisoned by dynamite-fumes and gases have recovered after treatment with cold affusion, artificial respiration, and venesection, followed by transfusion. Purging and sweating have also been successfully employed. For cases where dynamite has been swallowed the stomach-tube, warmth, and stimulation are indicated. R. G. HEBB.

DYNAMOMETER (*δύναμις*, power; and *μέτρον*, a measure).

DESCRIPTION.—The dynamometer is an instrument designed for measuring and accurately recording the strength of the hand-grasp. It consists of an elliptical ring of steel, to the inner and anterior face of which is attached a brass semicircular dial graduated with two rows of figures representing pounds or kilogrammes. The compression of the steel ring, by lessening its shorter diameter, moves a metal bar projecting from and sliding in a groove behind the dial, and this by rack-work communicates its movement to the index.

USES.—The dynamometer enables us to estimate the relative compressing powers of the two hands in cases of incipient or actually developed hemiplegia, and also to learn in a positive and definite manner, from time to time, the amount of improvement or the reverse which may have taken place. Since the power of the muscles of the forearm and hand, like that of other groups of muscles, varies a good deal with the general state of health of the patient, the dynamometer is also capable of yielding information concerning the strength of the patient, even where we have to do with general debility rather than with paralysis.

H. CHARLTON BASTIAN.

DYSÆSTHESIA (*δυσ-*, with difficulty; and *αἰσθάνομαι*, I feel).—A term applied to impairment of any of the senses, but especially to that of touch. See SENSATION, Disorders of.

DYSCRASIA (*δυσ-*, with difficulty, or badly; and *κράσις*, a mixture).—A morbid constitution due to the presence of some general disease.

DYSENTERY (*δυσ-*, with difficulty; *έντερον*, an intestine).—SYNON.: Fr. *Dysenterie*; Ger. *Kuhr*.

DEFINITION.—Dysentery was formerly held to be an acute specific disease; but recent researches have shown that it is not one specific malady, but that under this term are included several affections, the existence of which is manifested by the passage of frequent stools containing blood and mucus, accompanied by tormina (gripping abdominal pains) and tenesmus (painful straining and desire to go to stool).

ÆTIOLOGY.—(1) A connection between *malaria* and dysentery was formerly supposed to exist. In almost all, if not in all, situations where malarial fevers abound, dysentery prevails in proportion to the intensity and frequency of these fevers; where the latter have been extinguished by drainage, &c., dysentery becomes unknown. Further, some cases of dysentery are cured by quinine. Whether or not *malaria per se* can cause dysentery cannot be certainly laid down; but there can be no doubt that it is associated with it either as a cause, a complication, or a sequela, and that dysentery is especially liable to attack those whose constitution is undermined by *malaria*.

(2) *Conditions of the ingesta.*—*Unwholesome drinking-water* is a fertile exciting cause of the disease. This is especially the case where the water has become contaminated with the excreta of patients suffering from dysentery. In our tropical campaigns in Egypt, Ashanti, Abyssinia, Malay, and China the influence of drinking-water has been abundantly proved. *Alcoholic intemperance* is a fertile predisposing cause. *Salted or tinned provisions*, if in excess, will bring about the disease; thus, in the first Burmese War in 1824–26, the troops for six and a half months were kept on salt rations, and 48 per cent. perished within ten months, principally of dysentery. If the flesh be decomposed before salting, dysentery will be still more likely to occur. *An excessive animal diet of fresh meat* also will bring about the disease. After raids in Algeria, French soldiers have been seized with diarrhoea and dysentery as a result of an excessive diet of fresh mutton from the large quantity of captured sheep. *Insufficiently cooked grain* will cause the disease, as was exemplified in the large number of native followers falling ill with dysentery in Lord Roberts's great march from Cabul to Candahar: owing to their late arrival in camp they were too tired to cook their food properly. *Unsound fruit* will also excite it. Many hold also that *unripe fruit* has the same influence. In the writer's experience this was not found to be the case. Lastly, a *long-continued monotonous diet* will start the disease.

(3) *Habitual constipation* was pointed out by Virchow as a factor; and he showed in this connection how the cæcum and flexures of the colon, which are especially affected in dysentery, are also the seats liable to be loaded with feces.

(4) *Chill.*—During a campaign, if the troops become chilled at night, and especially where cold nights have followed hot scorching days, the men soon become affected with dysentery. This was well shown in the Ashanti, Looshaï, and Soudan campaigns.

(5) *Conditions of soil.*—Camping on irrigated lands, in the vicinity of large rivers, on decayed vegetation, in dense forests, on old camping-grounds, or on a site that is not changed at regular intervals, will predispose to the disease.

(6) *Season.*—The hot and rainy seasons in the tropics are those in which dysentery is apt to occur. Of 546 epidemics collected by Hirsch, fourteen fifteenths began in the summer months. Annesley points out how the disease rages in Bengal in the rainy season.

(7) *Effluvia from sewage.*—See COLITIS.

(8) *Infection.*—According to Sir Joseph Fayrer, the effluvia of the discharges of a patient can transmit the disease to other patients in the same ward. Heubner states that 'there is a limited contagion by

means of the excreta of dysentery, which, however, only becomes active when a large mass of excreta is collected together.' Roth, citing Mongeot, gives the following striking instance: 'A patient suffering from dysentery came to the Hôtel Dieu from Madagascar. At the time of his admission there were no cases of dysentery in hospital. The man went out uncured: after eight days from his admission cases broke out in the Hôtel Dieu. The man from the hospital went to an inn: the waiter thereof was shortly after seized with the disease. After leaving the inn the man went to a village in Aube: in the village, again, cases broke out after his arrival. Finally, he went to a family at Brienne: and of that family several members were then seized with dysentery.'

(9) *The influence of micro-organisms.*—During recent years numerous organisms have been mentioned in causal relation to the disease. Kruse and Pasquale divide cases of dysentery, with reference to micro-organisms, into three classes: (i.) Those in which *amœbæ* are found. (ii.) The cases of Japanese dysentery showing the special organism of Shiga and Ogata. (iii.) The class containing the various catarrhal and diphtheritic forms in which organisms of different kinds exist without the occurrence of the two former. To these a fourth may perhaps be added—viz. (iv.) The cases occurring in asylums. See COLITIS.

(i.) *The amœba of dysentery.*—Under the microscope this parasite presents a roundish or irregular shape, is of a pale greenish hue, and strongly refracts light. In diameter it measures usually 25 to 35 μ . At rest, the shape is more or less rounded, but in movement (which is sluggish unless on a warm stage) changes of shape occur, and two parts can be distinguished—viz. an *ectoplasma* and an *endoplasma*. The latter is more or less granular and darker; it constitutes the greater part of the organism. It often contains a varying number of vacuoles. In most cases a nucleus, with a nucleolus, is visible, especially when the organism is at rest. This is placed eccentrically at the edge of the endoplasma, is about 6 μ in diameter, and is more highly refractile than the vacuoles. Red blood-cells are frequently seen in the amœbæ; leucocytes are more rarely found, and never fat-globules. The outer portion, the *ectoplasma*, is homogeneous, of a lighter colour, forming a zone of variable thickness round the endoplasm, and having the appearance of finely ground greenish glass. Some observers have described an encysted form of smaller size, 10 to 15 μ in diameter. The capsule presents occasionally a double contour; the central protoplasm may or may not show a nucleus. The most striking characteristic of the amœba is its motility, shown either by an alteration in its shape or by a change of place, as in the case of other amœbæ. At a temperature lower than 75° F. the amœbæ become motionless, and at the end of 24 hours, when the stools are acid, every trace of them has generally disappeared. Therefore, in examining for these organisms no delay should take place. They are most easily detected in the little masses of mucus in the stool by smearing a small film on the cover-glass, and using a warm stage (100° F.). According to Lafleur, they may be identified by hardening the substance in Müller's fluid, and subsequently cutting and staining in the usual way. Unna's method is the following:—Harden in alcohol; stain in Unna's polychrome methylene blue from a

quarter of an hour to ten hours; decolourise in a small dish of water to which are added a few drops of Gruber's glycerine-ether mixture; wash well in water, and finally treat with alcohol and oil of bergamot, and mount in xylol-balsam. The best method, however, is to search for them in the fresh stool.

(ii.) *The Japanese organism of dysentery.*—In Japan Shiga and Ogata found no amœbe, but large numbers of a bacillus of the same thickness as, but shorter than, the tubercle-bacillus. It was motile; stained with Gram's method; grew on, and at ordinary temperatures liquefied gelatine. It was pathogenic to cats and guinea-pigs, the large intestine being chiefly affected and showing inflammation, hæmorrhages, and ulceration.

(iii.) *The micro-organisms of the various catarrhal and diphtheritic forms.*—(a) Ziegler describes a short bacillus within the lumen and under the epithelium of Lieberkühn's follicles, associated with the increase of which resulted inflammation and necrosis of tissue. (b) Maggiora noted the presence of large numbers of the *Bacillus coli*, *Proteus vulgaris*, and other organisms. (c) Bertrand and Baucher found the following in the stools: *septic anaërobic vibrios*, the *Bacillus coli communis*, three varieties of *Staphylococcus pyogenes* (*aureus*, *albus*, and *citreus*), two varieties of *streptococcus*, and the *Bacillus pyocyaneus*. This latter organism was also found in the stools in large numbers, as well as in the drinking-water in almost pure culture, in connection with a recent epidemic in the United States mentioned by Osler. The *Cercomonas intestinalis* has occasionally been found in large numbers. Lastly, in numerous cases occurring in Cochin China Treilk has observed the *Paramacium coli*. (d) The latest micro-organism is one discovered by Flexner of Philadelphia. It was not found in normal fæces, and in the intestinal contents of persons suffering from diseases other than dysentery in the same locality. It had close affinities with the *Bacillus typhosus*, but was distinguished from it by its action on litmus-milk, its slight motility, and tendency to become immobile in artificial cultures, and the serum-reaction, the organism agglutinating with the blood-serum of dysentery.

Relationship of the above organisms to the disease.—Some of the above are present under normal conditions, notably the *Bacillus coli communis*. Under the influence of catarrh of the mucosa they are held to become virulent, and able to penetrate into the subjacent tissues owing to the loosening of the epithelium. Celli and Fiocca also state that, in consequence of the presence of other bacteria, the *Bacillus coli communis* is able to secrete a special toxin which can excite dysentery.

(iv.) *The organism of Asylum-dysentery.*—See COLITIS.

(10) *Acclimatisation.*—No immunity but, on the contrary, an increased susceptibility, is effected by continuous residence in countries in which dysentery is rife.

VARIETIES.—Clinically, the various forms of the disease may be considered under the following heads:—

- A. Acute Catarrhal Dysentery.
- B. Diphtheritic Dysentery.
- C. Tropical or Amœbic Dysentery.
- D. The Dysentery of War.
- E. Chronic Dysentery.
- F. Modified Dysentery.

A. Acute Catarrhal Dysentery.—This form may be preceded for a few days by diarrhœa, or it may come on at once; outside the tropics the former mode of onset is perhaps the commoner, and the diarrhœa may last for 3–14 days. In India the writer has not noticed this preliminary diarrhœa. Where, however, it does occur, the griping pains become more severe and paroxysmal, and the patient is then seized with an uncontrollable desire to go to stool, a large mass of feces covered with mucus being perhaps evacuated. No sense of relief, however, is felt, and increasingly frequent efforts to empty the bowel follow, which only result in the evacuation of small quantities of foul-smelling mucus and blood. There are constant tormina and tenesmus. The stools are generally alkaline in reaction, and number from 10 to 20 in 24 hours. Heubner, from a series of investigations on the actual weight of feces passed in the 24 hours, found this to vary from 28 to 42 oz. The character of the evacuations will be described more in detail later on; but it is to be noted that, although the patient is constantly going to stool, very little fecal matter is passed. The actual condition has been rightly described as really one of constipation. The abdomen may be flat and hard. In this stage recovery may ensue in a day or two up to ten days.

Should the attack be more severe, febrile symptoms become pronounced, with much headache or occasional rigors. The calls to stool soon become incessant; tenesmus is almost constant; while the matters evacuated contain more blood than those of the milder form. They have been likened to meat-washings; their fœtor is most pronounced. The bowels may act from 50 to 60 times even to 200 times in 24 hours. Burning pain is felt at the anus, and there is a sense of something constantly present in the rectum that needs to be voided. The bladder sympathetically responds, the patient constantly desiring to make water. The abdomen, not at first distended, becomes so later on; tenderness is generally present along the course of the inflamed intestine.

The most aggravated form of this variety of the disease is known as the *Gangrenous*. According to Scheube, it may also arise after being preceded for a few days only by a simple diarrhœa, with but little fever and tenesmus. The latter symptoms then rapidly increase, while the discharges take on a brownish-red or black colour and smell horribly. Contained in them are cylindrical tubular masses, sometimes a foot long, held by some to consist of exfoliated parts of the intestinal walls, but by Heubner and others only of mucus. The number of stools in 24 hours is extraordinarily great—150 to 200—so that the unfortunate patient seems to pass the greater part of his time on the night-stool. In addition, he may pass a large quantity of blood, and death may ensue from this cause. With the above symptoms the general condition of the patient becomes rapidly and progressively worse; the pulse quickens and weakens, the skin becomes cold and clammy, the temperature subnormal, and often the evacuations are passed involuntarily. Death closes the scene in about the second week from exhaustion; or at the last cerebral symptoms, commencing with delirium and ending in stupor, may supervene; or finally, the end may be brought about by pyæmia or perforating peritonitis.

B. Diphtheritic Dysentery.—Here from the beginning the patient is very ill, has high fever, much abdominal pain, and frequent discharges.

Delirium sets in early. The abdomen is distended. The discharges, according to Osler, are diarrhoeal in character, blood and mucus not being so constant as in the catarrhal form.

C. Tropical or Amœbic Dysentery.—Dysentery occurring in the tropics has of late years been differentiated as *Amœbic Dysentery*, and is held by many to be due to the *Amœba coli*. But in the opinion of many, and in that of the writer, it is doubtful if the amœba be not rather an epiphenomenon than the actual exciting cause. The arguments *against* its being an actual exciting cause are as follows: It is found in healthy fæces and in other diseases affecting the alimentary canal, while many observers in Egypt have altogether failed to find it in cases of dysentery. There is no relation between the severity of the illness and the actual number of the amœbæ; thus, in two fatal cases recorded by Gasser in Algiers they were very scanty, while they were found in large numbers in the cases that did not succumb. Indeed, some observers, noticing the fact that in the parts of the intestine in which amœbæ were found most frequently other microbes are scanty, assert that the organism is actually beneficial, inasmuch as it destroys the other microbes. Where the disease has been produced experimentally by the injection of the amœbæ, this may have resulted from other micro-organisms being injected simultaneously. Finally, the abundance of amœbæ found in the affected part of the intestine may be due only to the fact that this affords a good nutrient site, the growth of the amœbæ being only the *result* of the disease.

On the other hand, the following are the arguments *in its favour*: It is said that the amœbæ found in health are not of the same nature: they have no pathogenic action on cats, whereas the amœba of dysentery, when injected into the rectum of this animal, will cause an affection resembling dysentery. The amœba of health is also said to be smaller in size, with a more finely granular protoplasm, and it does not take up the red blood-corpuscles. Again, the *Amœba coli* was found by Kartulis in the stools of all the cases (150) which he investigated, also in the walls of the intestines and in liver-abscesses, while it is not found in any other disease. The occurrence of the organism in liver-abscesses is of great weight. Staff-surgeon Bassett Smith, R.N., has reported a case of liver-abscess in which only the amœba was found, the pus being sterile as regards other organisms.

Symptoms of Amœbic Dysentery.—The following symptoms are said to characterise this form of the disease: Its chronicity and irregular course; its relapses alternating with periods of comparative quiescence; the great liability to abscess of the liver; the less frequent occurrence of pain and tenesmus; the fluidity of the stools, and the presence in them of amœbæ.

Now in India dysentery is by no means marked by an irregular and chronic course; everywhere one attack predisposes to a second; pain and tenesmus the writer has not observed to be less frequent. There is, in fact, good reason for the opinion that there is no special form of amœbic dysentery, but that the amœbæ are merely concomitant, and that the only relation they have to the disease is that they find in the diseased intestine a suitable nutrient medium for their development.

D. The Dysentery of War.—In the operations of war, as a rule bowel-complaints begin with diarrhoea; then a few cases of dysenteric diarrhoea

set in, culminating in true dysentery. At other times they may begin as true dysentery. It has often attacked our camps with terrible severity in an epidemic form, where the sites were not properly kept in a sanitary condition. It was very fatal in the Crimea in 1854; in the armies of the United States in 1862-65; and again in the Franco-German War of 1870. In New Zealand in the wars of 1862-65 it severely attacked our troops. In this form the disease does not particularly affect any given season; it is extremely fatal, and frequently takes on a contagious character.

E. Chronic Dysentery.—This occurs as a sequel of the acute form, and is specially frequent in cases of tropical dysentery. It has also been observed to follow cases of diarrhoea, blood appearing in the stools, with colic and tenesmus. The stools are in number from 2 to 6 in the twenty-four hours. Pain and tenesmus are not prominent symptoms, and may be altogether absent. Diarrhoea and constipation may appear alternately. The patient continually loses flesh; the appetite is capricious and bad; the tongue red, smooth, and fissured; the hair becomes thin, and the countenance careworn. Death ensues from exhaustion, liver-abscess, or other intercurrent disease.

F. Modified Dysentery.—The disease may be associated with *scurvy*—a fatal form unless the complication be recognised; with *malaria*, and with *typhus* and *typhoid fevers*. A form of the disease called *Arthritic Dysentery* has been observed in Caen, Norway, and Ohio. In the last variety the large joints, especially the knee, are affected. Heart-complications are rare, and there is no greater liability found to it among rheumatic subjects. It may be either polyarticular or monarticular; the pain is dull in character, and there are no exacerbations. The local temperature is not modified and the integuments are normal, while the peri-articular tissues are swollen. Only rarely does effusion take place into the synovial membrane. The prognosis of this form of arthritis is good, neither suppuration nor ankylosis occurring as a rule.

MORBID ANATOMY.—*In the acute catarrhal forms* the large intestine is the seat of the disease, especially the descending colon, sigmoid flexure, and rectum; rarely, the small intestine is also affected. The mucous membrane is injected, swollen, and covered with a tenacious and blood-stained mucus; it is infiltrated, together with the submucous tissue, with serous fluid and leucocytes. The most prominent appearances, however, are due to the enlarged solitary glands. In very acute cases the picture is that of an acute follicular colitis; in the less acute, the follicles suppurate and ulcerate, while in the intervening parts occur areas of necrotic tissue. Thus ulcers result either from the follicles, or from necrosis and sloughing of the intervening tissue. The ulcers vary in size, have ragged and undermined edges, and a grey and sloughing floor. The edges are much thickened, owing to infiltration of the submucosa. The muscular coat and peritoneum may be implicated. In many cases a large portion of the mucous membrane may die *en masse*, and be thrown off as a tube. Manson has called attention to the occurrence of enormous growths of polypoid tumours occurring in the mucous membrane. The mesenteric glands are enlarged and congested.

In Diphtheritic Dysentery an exudate is seen surmounting the folds of the colon. The latter is very much enlarged, the walls stiffened and thick,

and the mucosa from the ileo-cæcal valve to the rectum 'represented by a tough yellowish material, in which on section no trace of the glandular element can be seen' (Osler). The condition in advanced cases is described by Rokitsky as 'a black, rotten, friable, charred mass.'

In the so-called *Amœbic Dysentery* the cæcum and ascending colon are generally affected, though the changes may extend to the rectum. The process consists in a thickening of the bowel, followed by necrosis and more or less deep ulceration. Thickening is very constant here, and is especially marked in the submucosa; it consists of a general œdema, and in localised areas of infiltration with leucocytes. These latter areas stand up above the surface as sharply circumscribed projections over which the mucous membrane is discoloured. Later on, the mucous membrane sloughs and is separated, leaving a small opening through which is seen the necrotic material in the submucosa. The ulcer thus formed spreads. The viscid material contains amœbæ, more or less abundant. The most characteristic ulcers have soft, undermined, ragged borders, raised somewhat above the level of the mucous membrane. Where the ulceration is deeper it advances along the intermuscular septa to the subperitoneal connective tissue, which then becomes œdematous and necrotic, and thus areas of the muscular coat are dissected off. This progressive implication of the connective tissue is one of the most characteristic features of the disease. The amœbæ are constantly present in the ulcers, and most abundant in the submucosa; in the mucous membrane itself they are seldom found, and then chiefly in its lower portion. In the submucosa they lie especially in the zone immediately surrounding the central necrotic part of the ulcer, being scarce in the necrotic area itself. In *chronic dysentery* the ulcers remain with little tendency to heal, sometimes penetrating more deeply and leading to suppuration in the neighbourhood especially of the rectum. The remaining mucous membrane is swollen. The intestinal wall may be adherent to the surrounding parts. If the ulcers heal the symptoms may still continue; there is then much atrophy of the intestinal coats, the glandular structures disappear, and the wall becomes thin and translucent.

INCUBATION.—According to Hammerle, this may be five days; according to Czernicki seven; and according to Liebermeister, three to eight days. Some authors, indeed, state that the period may be as long as four to six weeks or even months, but this is probably erroneous.

THE STOOLS.—1. *In the milder forms.*—Here, generally, first a solid motion is passed, covered more or less with greyish or colourless mucus. Next, the motion consists of small quantities of offensive mucus, with minute fecal lumps. Then there are passed many small quantities of mucus stained with fecal matter and often mixed with blood. Scybala are passed at intervals, though only occasionally. As the disease subsides, fecal matter reappears in the stools until they become normal.

2. *In severer forms.*—After the bowel has discharged its contents (often solid) there is next passed whitish or coloured jelly-like mucus, which quickly becomes bloody, and may contain rather large quantities of partly clotted blood. The motions are frequent, about two to three drachms in quantity. This constitutes the so-called *mucous* or *mucosanguineous stool*: it forms a slightly yellowish glairy

quivering mass, lying in balls or clumps without any fecal material, or surrounding a formed mass of feces. If the discharge be very fluid, the masses of mucus unfold into hyaline shreds—the so-called *shreddy stool*. This class of stool is passed in the first stage of the disease.

The sanguineo-purulent stool.—This marks a still more advanced stage. Here in a small amount of yellow fluid, generally without feces, float a number of reddish lumps as large as a pea or bean, like raw meat. This is known as the *lotura carnea*.

The purely bloody stool.—This results either from superficial ulceration, or from ulceration into a large vessel.

The simple purulent stool occurs only in the later stages, and is due to the formation of submucous abscesses, or destruction of the mucous membrane. The pus is generally discharged pure and odourless, but is often mixed with fecal matter and blood.

The gangrenous stool.—Here there is a blackish fluid with a putrid odour, containing bits of gangrenous tissue. Tubular structures may occur in the discharge, which at one time were supposed to be separated portions of the intestinal wall, but according to Heubner they consist only of mucus.

The 'frog's spawn' or 'boiled sago' stool.—This consists of clumps of hyaline mucus. They are supposed to be formed as follows: mucus is pressed into the cavities out of which the intestinal follicles have fallen, and after being moulded in these cavities again falls out into the bowel. It occurs mainly in the stools of chronic dysentery.

The porridge-like stool described by Fayrer is very offensive, without blood or mucus, and occurs in the gangrenous form of the disease.

The stools of chronic dysentery.—These vary much in character: are thin, watery, of varying colour, and very offensive. Mucus, blood, and pus are mixed in varying proportions. Sometimes the blood is so intimately mixed that the whole stool is of a brownish or dark-red colour.

Microscopically the glairy bloody stools show red blood-corpuscles, leucocytes, and large swollen epithelioid cells, containing fat-drops or vacuoles which may be mistaken for amœbæ. Crystalline bodies of a finely pointed octahedral form, like Leyden's crystals in asthma, have also been noted. They are not found in normal feces, and their significance is unknown.

COMPLICATIONS AND SEQUELÆ.—The following are the chief complications and sequelæ: *Malaria*; *Scurvy*; *Abscess of the Liver*; *Perforative Peritonitis*; *Stricture of the Intestine* (though not frequently occurring this can result from cicatrization of the ulcer); *General Dropsy* during convalescence; and *Nervous affections*, such as *Cerebral embolism*; *Paraplegia*; and *Neuritis*.

DIAGNOSIS.—A. *Of the disease itself.*—*Malignant disease of the lower bowel* in patients of an advanced age may simulate dysentery. Digital examination should clear up the case.

Ulcerative colitis in temperate climates does not present the tenesmus, burning pain at the anus, and frequent desire to go to stool of dysentery (see COLITIS). The possibility of the existence of *siphilitic ulceration* of the rectum should be borne in mind.

In young children *intussusception* has been mistaken for the disease; and cases of *enteric fever* have also been attributed to dysentery where the usual characteristic features of the former have been absent.

Epidemic gangrenous proctitis, a very fatal disease occurring in the north of South America and in Fiji, has many of the symptoms of dysentery, such as passage of bloody stools, or of a slimy, foetid, semi-liquid substance streaked with blood. It may be difficult to distinguish, and by some is supposed to be allied to dysentery.

B. *Of the part of the intestine involved.*—The nearer to the rectum the disease is situated the greater is the tenesmus; the nearer to the cæcum, the greater the gringing. If the cæcum and ascending colon be chiefly affected, the stools are more abundant and feculent, but less frequent and passed with little straining: they are pale, thin, watery, parti-coloured and mixed with blood and mucus. The points of localised tenderness and thickening of the gut should be ascertained. In mild cases, probably the lower part of the large intestine only is involved. In severe cases the whole of the large bowel, and even part of the ileum, may be affected.

PROGNOSIS.—This will depend upon the general condition of the patient as well as upon the characters of the stools. A. *From the stools.*—A good result can be foreshadowed in the cases in which the blood and mucus disappear, and the ordinary fecal character soon manifests itself.

A bad result may be prognosticated, according to Fayer, in those cases which are accompanied by porridge-like stools without blood or mucus. Where fluid fecal matter is from time to time passed throughout the illness, the import is rather unfavourable, as showing the disease to be extensive, and affecting especially the upper part of the large intestine, as well as of the small. Where the stools consist of blackish-red or blackish fluid with a horribly putrid odour, containing bits of gangrenous tissue, the prognosis is of the worst character. The writer has never known a patient passing these stools to recover.

B. *From the General Symptoms.*—The gravity of the case is to be judged from the aspect of the patient. Where the features become sunken, while a cold sweat supervenes, and a fetid odour is given off from the body, the worst is to be feared. Other unfavourable symptoms are suppression of urine, sordes on the lips and tongue, involuntary passage of the contents of the bowel, and cessation of all pain and tormina with increasing prostration and hiccough.

In chronic dysentery the prognosis is recorded by most authors as unfavourable, but Stephen Ward, from his long experience at the Seamen's Hospital, Greenwich, held that recovery took place in the majority of these cases when they are properly treated. The writer can confirm this statement from his own experience. In scorbutic dysentery the prognosis is bad, unless the recognition of the scorbutic element is promptly recognised; even then the cases will cause much anxiety.

Lastly, where abscess of the liver has developed the prognosis is extremely unfavourable.

TREATMENT.—A. *Prophylactic.*—In hot countries the greatest caution must be exercised with respect to the ingesta. The water-supply must be kept in a state of purity. If there be any cause for suspecting it, it should be filtered through a Pasteur-Chamberland or Berkefeld filter, and boiled, not boiled first and then filtered. The various points before mentioned regarding the causation of the disease by the solid constituents of the food should be borne in mind. Wet clothes must never be allowed to dry on the wearer. A cummerbund should

always be worn. The stools of patients should be disinfected, or better burnt, being previously mixed with sawdust. The bedding, &c., must also be disinfected.

In war-time the chief points to bear in mind are first as regards the selection of men. All men with a previous history of bowel-complaints should be rejected for service. During the China war of 1860 it was found that the disease chiefly affected the men who had previously suffered from dysentery in India and Hong Kong. Men who have had a previous unhealthy medical history should not be selected, nor should those with a strong malarial history be taken. Again men from up-country districts in India should not be chosen for campaigning in marshy countries or those abounding in jungles. The hot and rainy seasons should be avoided for the operations if possible. Cholera-belts must always be worn. Marches should not be too long or fatiguing. The camp should not be located in the vicinity of large rivers, on irrigated ground, on ground with decayed vegetation, or on old camping grounds, nor if possible on the field of battle. The site of the camp should be changed at frequent intervals, be well drained, and kept strictly clean; the tents must not be crowded together. Fatigue duties are especially dangerous when the men are working in water. The precautions as regards food above set forth must be rigidly observed. The men should not be fed on salt rations for a long-continued time. At night they should sleep if possible raised off the ground, and should be cautioned that cummerbunds are always to be worn, and the abdomen not exposed to the air. Parades should be held to see that the men are not suffering from digestive derangements, and that cholera-belts are worn. Parkes recommended that daily bathing parades be held, as invigorating the body against vicissitudes of temperature; the issue of prophylactic rations has been recommended. In China in 1860 quinine was given, in Tonquin the French received bismuth. Sulphuric acid has also been given.

B. *Curative treatment.*—At the commencement of this article, it was stated that there is probably no such specific disease as dysentery; this opinion gains weight from a study of the different results of treatment in different regions of the world.

I. *In all the acute forms of the disease the two chief essentials are (1) rest; and (2) regulation of diet.*

1. *Rest.*—The dysenteric patient should at once take to his bed. Rest must be complete: the use of the bed-pan is essential. By this rest the movements of the bowel are controlled. Mr. Cantlie in addition urges the application of a large thick pad broad enough to cover the whole front of the abdomen. It may be either wet or dry; Maclean also recommended the use of the water-belt.

2. *Food.*—This should be reduced as much as possible, and consist of milk, with whey and ordinary weak tea. If the thirst is excessive, the patient can suck small pieces of ice. Where milk cannot be taken by the sick man, strong beef-essence is indicated. If much weakness is present, some form of alcoholic stimulant should be administered.

3. *Drugs.*—The choice of the drug would appear to vary with the country. In India the drug *par excellence* is *ipecacuanha*. Docker who revived this method of treatment found that by it his mortality was reduced from 10–18 per cent. to something less than 2 per cent. In giving it, the patient should first take 30 m of Tinctura Opii, a mustard leaf or

poultice being applied to the abdomen. Half an hour after, he is given 30 grains of ipecacuanha. After taking the latter the patient should lie perfectly still; as a rule no vomiting will then occur. Should the drug not be retained, it is advisable to wait two hours and then repeat the dose. Should it again be rejected, some good will have been effected notwithstanding, and an interval of 8 hours should elapse before repetition. The administration of the drug should be twice a day in 30 or 20 grain doses. Improvement is shown by the appearance of feculent motions, while the tormina and tenesmus cease. This treatment must be continued for 2-3 days, and when by this time the blood and mucus have ceased, subnitrate of bismuth in 20-grain doses, or salicylate of bismuth or salol, may be given.

The French authorities recommend that ipecacuanha should be given in doses of 10-12 grains every ten minutes until copious vomiting results, as they consider the essence of the treatment to lie in a powerful evacuating effect on the stomach. The ipecacuanha in a de-emetised condition was advocated in India a few years ago. Experience militates against this, so that the French view would seem to be correct.

In cases where ipecacuanha cannot be retained, *cinnamon* in doses of a drachm night and morning, in a bolus, has been found very efficacious; but in the writer's experience, it is useful chiefly in the milder attacks.

The saturated solution of *sulphate of magnesium* has of late also been much used. Patients who have taken it have found it extremely nauseating and unpleasant. It is best not to give it in saturated solution.

In South Africa, on the contrary, ipecacuanha does not seem to exert the same good effect. In the present war (1901) the treatment by salines has been chiefly followed. Dr. McKenzie recommends a mixture containing sulphate of magnesium with sulphate of quinine and aromatic sulphuric acid. To this may be added a minute quantity of perchloride of mercury. The first 4-6 doses may be given every 15-20 minutes, then every hour. In 24-48 hours improvement sets in, and bile appears in the motions: it is then given less frequently.

Another method of treatment is by the castor-oil emulsion, preceded by 3-4 grains of calomel.

In Africa again, tincture of *Monsonia ovata* has been strongly advocated (strength, 1 in 8 of alcohol, 90 per cent. Dose, 1-4 drachms every 4 hours). Its utility, however, seems to be limited to that region.

In China the treatment by *salines* is by many preferred to that by ipecacuanha.

In North America, *simaruba bark* has been found very efficacious; 1 ounce of the bark is boiled in 3 pints of water, till the latter is reduced to 1½ pints. The dose is half this quantity night and morning.

In Malaya *mangostine bark* has its advocates. The rinds of three dried mangostines are boiled in 30 ounces of water till the latter is reduced to 20 ounces. The dose is ½ part three times daily.

Bael fruit has been recommended, but it is in the scorbutic form of the disease that its use is especially indicated.

Opium should not be given. It only masks the disease, and shuts up the germ in the alimentary canal.

In all cases at the onset, where the discharges are scanty and a source of irritation, it is a good

plan to give a *tepid-water enema* of 2-4 pints. If malaria complicate the case, *quinine* should be given. If scurvy, the usual *antiscorbutics* must also be administered.

II. Treatment of Chronic Dysentery.—Dr. Ralfe strongly advocated the following plan of treatment: Every night an *opium suppository* is to be given, and every other night 3 ij of *castor oil*. His theory was to keep the ulcerated mucous membrane clean by the oil, and to restrain the tenesmus and tormina by the opium. In other cases 20 grains of subnitrate of bismuth with one quarter of a grain of Dover's powder were given. How this minute dose acted was doubtful, but no doubt could be entertained of the fact that these small doses acted better than larger ones, and also that the combination of drugs acted better than the bismuth-salt alone. Ralfe also obtained good results by giving 20 m of oil of turpentine, three times a day. Manson always begins the treatment with *ipecacuanha*. Jameson advocates large enemata of *warm milk*. Hillier, in South Africa, used 3 j—5 j of castor oil, with 4-10 m of Tinctura Opil. *Simaruba* is also useful in chronic dysentery.

Treatment by large enemata containing astringents, though practised by some, is not advisable in acute dysentery, as the irritability of the rectum will be an almost insuperable obstacle, although it is said to have been overcome by a 4 per cent. cocaine-solution. The bowel is here washed out after every evacuation with 3-6 pints of a warm water enema, containing quinine 1-1,000 to 1-5,000. In chronic dysentery it is successful. Here astringents are used. *Nitrate of silver* of the strength of 20-30 grains to the pint of water is injected in the proportion of 3-6 pints, at a temperature of 100° F. In obstinate cases an artificial anus may be made above the cæcum to secure rest and disinfection of the affected intestine. See COLITIS.

In *amabic dysentery*, medicated enemata are strongly recommended by those who consider this to be a special form of the disease. These may consist of: *quinine* (1-5,000 to 1-1,000), *perchloride of mercury* (1-10,000 to 1-5,000), *hydrogen peroxide* (1-20 to 1-5), *silver nitrate* (1-3,000 to 1-1,000), *zinc chloride* (1-2,000 to 1-1,000), or *methylene blue* (1-1,000 to 1-100). The enemata should be given once or twice a day. By some they are used alternately: thus quinine may be used with hydrogen peroxide or with methylene blue on alternate days.

In the case of children, ipecacuanha in India is as efficacious as in adults. For a child six months old it is advisable to give ½ grain of ipecacuanha with one grain bicarbonate of sodium, and a little sugar of milk: for succeeding ages, a grain with twice the amount of soda for every year of age. Huntley, of Han-Yang, recommends also a combination of salol and calomel: or one drachm every hour of a solution of perchloride of mercury, 1-10,000.

For the *tenesmus* and *distressing tormina*, a hypodermic injection of morphine should be given to adults.

Diet in chronic dysentery should consist of milk. Should curds appear in the stools the quantity must be reduced and egg-albumen given in addition.

ANDREW DUNCAN.

DYSIDROSIS (δυσ-, with difficulty; ἰδρώς, sweat).—SYNON.: Pompholyx; Cheiro-pompholyx.

This name is applied to a well-defined disease affecting chiefly the hands and feet, characterised by the appearance of small deep-seated vesicles. The supposition, now disproved, that the vesicles were due to the retention of sweat, or were actually retention-cysts in the course of the sweat-ducts, accounts for the name of the disease.

The early lesion of the disease consists of a minute vesicle set deep in the epidermis. Its form is distinctly rounded and it may project clearly above the surrounding epidermis. In other cases, the vesicle continues to occupy a deeper position and causes no elevation of the superficial epithelium. The skin surrounding the vesicle may show no alteration, but is usually slightly reddened even in the early stage of the disease. The vesicles contain a perfectly clear fluid, free from the gummy feeling of the exudation of eczema and usually of alkaline reaction. After lasting a few hours, the vesicles may rupture and their contents be discharged on the surface. Healing may take place rapidly under a slightly desquamating surface. In other cases, and perhaps more commonly, neighbouring vesicles coalesce and larger lesions result till at length the upper layers of the epidermis may be raised over areas corresponding to nearly the whole palm or the whole extent of a finger. It may be readily understood that the vesicles, either in the small or in the large form, are very liable to infection by various pus-forming organisms, and the disease may have as a secondary development purulent dermatitis of almost any degree of severity.

As a result, eczema of various types may follow dysidrosis and affect wide areas of the body. It is also not uncommon to find that absorption from the purulent surface of the extremities produces severe adenitis of the lymphatic glands of the axilla, groin, and other regions, giving rise to the formation of abscesses. The duration of any individual attack varies indefinitely from a few days in the mild and uncomplicated form of the disease, to a much longer period in the severer and complicated forms. In certain cases the condition remains in a subacute and chronic state, the patient constantly showing a few of the characteristic vesicles. Healing takes place finally, but there is always a very marked tendency for recurrence of this malady.

The anatomical lesion of the disease consists in the formation of a cavity, which becomes filled with the characteristic limpid fluid and which shows practically no leucocytic exudation. The epithelial cells surrounding the cavity are altered by maceration and tumefaction, and the degenerated cells float free in the contents of the vesicle. In later stages a variable number of leucocytes make their appearance in and surrounding the vesicles, and as the result of inflammatory reactions fibrin may be seen present. The actual place of origin of the vesicle has given rise to much discussion among histologists, and probably the position varies. As a general rule the early lesion is situated in the upper layers of the rete mucosum or occasionally in the stratum granulosum. It appears also to be produced both in the inter-papillary portions of the epidermis, and in the parts covering the papillæ. So far as can be ascertained, the vesicles do not arise by dilatation of the sweat-ducts, but it can be readily understood that many of the vesicles in the process of their formation must open up communication with these ducts. The later

anatomical changes of the disease are those of inflammatory reaction due to the presence of pus-forming micro-organisms.

The symptoms of the disease are chiefly those of mechanical discomfort produced by the swelling of the tissues in the early stages of the disease; later the effects of exfoliation of the epidermis, or of septic absorption from infected surfaces, may appear. Patients rarely complain of pruritus, but suffer much from feelings of tension, heat, and throbbing of the parts, or actual acute pain. The amount of suffering varies much in different individuals. The disease is frequently seen in those of neurotic disposition, and in persons suffering from loss of health. In such persons these painful symptoms are severely felt, and are sufficient to cause much loss of rest, and further nervous exhaustion. During an attack of even moderate severity it is quite impossible to use the hands or to walk on account of the pain experienced.

The disease may appear at any age, and in young subjects its effects may be as well marked and as severe as in adults. In the opinion of most observers, there appears to be some relationship between the disease and conditions of hyperidrosis. In those who habitually sweat profusely on the hands and feet, the disease is more frequent than in others, and during hot weather when perspiration is profuse, attacks of the malady are more common. It must be stated that this association is not constant. The disease is present in those who do not sweat unduly, or who have not sweated profusely, and it appears that contact with certain irritant substances induces an attack of the disease in individuals subject to the malady. Thus one patient is known to develop dysidrosis on handling the materials necessary for painting in oils, and a chemist may suffer from recurrences of the disease when by inadvertence or carelessness he handles certain chemical reagents. In addition to the relationship to hyperidrosis and to contact with certain irritants, there seems also to be a congenital disposition to the production of the vesicles which are the primary lesion of the disease. The results which have been obtained by histological examination do not lend support to the supposition that the disease is one of disorder of the sweat-apparatus. Certain bacteria, which have been discovered in the vesicles and which have been claimed as specific, have probably no causal relation to the malady, and at present the exact pathogenic relationships of the disease are obscure.

DIAGNOSIS.—It is important to distinguish the disease from certain varieties of eczema, especially those of the vesicular type. The deep-seated translucent ('sago-grain') vesicles of dysidrosis; their repeated occurrence on the usual situations—on the adjacent surfaces of the digits and the inter-digital clefts, as well as on the other parts of the hand and feet; the tendency to form bullæ, and the widespread exfoliation of the epidermis which ensues; as well as the peculiar limpid nature of the fluid contents of the vesicles, will all serve to distinguish the disease from the types of eczema which resemble it. The course of dysidrosis with its tendency to recurrence and to the rapid cure of the slighter attacks will confirm the diagnosis.

Hydrocystoma, a condition which has been comparatively recently recognised, bears certain resemblance to dysidrosis. This disease appears to have close relationship to the sweat-apparatus,

and the small clear cysts which are characteristic of the eruption are supposed by certain observers to be actually retention-cysts. This point, however, is not absolutely confirmed. The eruption is situated usually in the central regions of the face, and the individual lesions are of much longer duration; they do not readily become inflamed or give rise to exfoliation, as in the case of dysidrosis. The lesions of hidrocystoma tend to disappear in the winter and become more pronounced in the summer or during periods of exertion and profuse perspiration.

Scabies may be mistaken for this disease, but the presence of the burrows of the acarus in addition to the vesicles, as well as the actual discovery of the acarus itself, will serve to distinguish scabies from dysidrosis. The distribution of scabies, the pruritus, and many other characteristics are wanting in the case of dysidrosis.

TREATMENT.—There seems to be no drug at our disposal to control the disposition of the patient to dysidrosis. In those who are proved to be liable to the disease, the avoidance of such irritants as are known to produce it, or of severe exertion, while an attack is imminent, must be enjoined. When the attack makes its appearance, the utmost care must be taken to prevent infection of the vesicles by pyogenic organisms. It is consequently necessary to observe strict cleanliness, and it is well to have the hands and feet frequently bathed with a mild antiseptic. For this purpose solutions of boric acid appear to be of the most service. Weak solutions of carbolic acid, however, are often used with good effect. During the time of the rupture of the vesicles or the exfoliation of the skin, still greater care must be taken to keep the parts affected free of irritants or of any chance of septic infection. These precautions must be specially noted in the case of working people. Most serious results due to suppuration in the groin and axilla are known to occur in labourers and others, who are liable to the disease. If the attack becomes severe and bullæ appear with much exfoliation of the epidermis, the parts must be carefully dressed two or three times daily with emollient preparations such as Carron oil, simple soft paraffin, oxide-of-zinc

ointment, or boric-acid ointment. In such cases absolute rest to the hands or to the feet must be ensured. If the parts become purulent more strenuous measures must be taken to prevent spread of the septic infection, and absolute rest becomes a necessity; the neighbouring lymphatic glands must also be watched to observe signs of abscess-formation. At the same time the antiseptics used for dressing the raw surfaces must be sufficiently mild not to cause a spread of dermatitis as the result of treatment. During the period of healing the hands and feet should be protected as far as is possible, and the use of an antiseptic dusting-powder containing from 5 per cent. of boric acid, with oxide of zinc, siliceous earth, and starch, is often satisfactory.

After complete recovery from an attack, patients should be warned as to its liability to recurrence, and be directed to take special precautions to prevent irritation or contamination of the parts liable to be affected.

JAMES GALLOWAY.

DYSMENORRHEA (δυσ-, with difficulty; μήν, a month; and βέω, I flow).—Difficult and painful menstruation. See MENSTRUATION, Disorders of.

DYSPEPSIA (δυσ-, with difficulty; and πέπω, I digest).—A synonym for indigestion. See DIGESTION, Disorders of.

DYSPHAGIA (δυσ-, with difficulty; and φάγω, I eat).—Difficulty in swallowing. See DEGLUTITION, Disorders of.

DYSPHONIA (δυσ-, with difficulty; and φωνή, the voice).—Difficulty in producing vocal sounds, so that the voice is more or less enfeebled. See VOICE, Disorders of.

DYSPNŒA (δυσ-, with difficulty; and πνέω, I breathe).—Difficulty of breathing. See RESPIRATION, Disorders of.

DYSURIA (δυσ-, with difficulty; and οὐρέω, I pass water).—Difficult or painful micturition. See MICTURITION, Disorders of.

E

EARACHE.—See PAIN in VISCERAL DISEASE.

EAR, Diseases of.—SYNON. :—Fr. *Maladies de l'oreille*; Ger. *Ohrenkrankheiten*.

The natural division of the ear into external, middle, and internal suggests a rational as well as a convenient classification of the disorders to which the auditory apparatus is liable.

I. EXTERNAL EAR.—In examination of the external meatus and tympanic membrane, bright diffused daylight, or, when this is not obtainable, light from a bull's-eye lamp lit with gas or electric light, is the best for illumination, and the light should be reflected from a concave perforated mirror of eight-inch focus down a tubular speculum. In any operative proceedings the mirror should be worn on the forehead, as in examining the throat, but otherwise should be held in the hand. As great variations in the calibre of the auditory meatus are met with, it

is necessary to be provided with specula of several sizes, the most convenient form being that known as Gruber's.

Of the affections of the external ear the most important are the following :—

1. Eczema.—Although the acute form of eczema occasionally affects the auricle and external auditory meatus, it is far more common to meet with the chronic variety. Elderly females are especially subject to eczema of the ear, and it is to its long continuance that the remarkable narrowing of the external meatus throughout its whole extent, met with occasionally in the subjects of this complaint, is generally attributable. Such narrowing will often amount to almost complete closure, and it is in these instances that eczema becomes the cause of greatly impaired hearing; for when this condition is arrived at, the passage down to the tympanic membrane is at times so small as only to admit a very small

probe. It is for this reason that, although no special methods of treatment are called for, beyond what is necessary when parts other than the ear are affected with eczema, it is of the greatest importance to keep the meatus sedulously free from secretion; and this occasionally is not a very easy matter.

2. **Changes in cartilage.**—Another condition in which the external passage becomes subject to partial closure is shrinking of the cartilaginous part of the meatus. This, again, is a complaint of old age, and is attributable to no known cause. It is readily relieved by the patient wearing a piece of silver tube, to keep the passage patent.

3. **Bony Growths.**—Bony growths in the external auditory canal are of two kinds, true exostoses and hyperostoses.

(1) *True exostoses* arise from the junction of the osseous and cartilaginous portions of the canal, owe their origin to acute inflammation, and are somewhat rapid in their growth. They present very little difficulty in their removal, inasmuch as they are attached by a pedicle, and are not composed of very dense tissue.

(2) *Hyperostoses* arise from the osseous part of the canal. These growths are of ivory hardness, and have a large base. They are occasionally single and confined to one ear, but generally are multiple, and more usually than not affect the meatus of both ears simultaneously. The form which they habitually assume is that of three growths, the apices of which meet, or nearly so, in the axis of the canal. Thus, seeing that a very small opening is quite enough to permit of the passage of sonorous vibrations to the tympanum, their presence remains undiscovered until a minute piece of cerumen completely closes an already nearly closed canal, and by the deafness thus produced leads to their detection. Their growth is very slow, as they are observed to remain without increase for many years, and although without doubt they are at times congenital, it would seem that their existence is often called into being by a local irritation, first, because in a large number of cases the individuals so suffering have been for long periods addicted to diving; secondly, they are frequently preceded by a discharge passing for years through a perforated membrane and through the canal.

TREATMENT.—The occasions when these growths should be removed ought to be strictly limited to two conditions: 1st, when they interfere with the escape of purulent discharge, and so place the patient in danger of cerebral inflammation; 2nd, when by absolutely closing the canal they induce great deafness. In the large proportion of cases the occasional removal of cerumen which blocks up the small opening that remains is all that is necessary, and this should be done by the use of a small hook rather than by syringing, as the passage of water behind the growths is to be avoided. When, however, it is imperative to remove bony growths for either of the above-named reasons, the best method consists in drilling them away with a dental engine, to which may be attached drills of various shapes and sizes. This operation should be performed under reflected light, and ether must be given. Owing to the extreme hardness of the growths, this process occupies considerable time, but with due care it is not a dangerous proceeding. When the object in view is simply the relief of deafness, it is sufficient to drill away the apices of the growths, but when it is done for the purpose of providing a complete escape to

purulent matter, it is desirable to remove the chief part of the enlargement.

It must not be thought that the drilling process can always be conducted through the external meatus. The extensive nature of the growth or growths, the small size of the meatus, or the deep-seated position of the growths makes it frequently necessary to detach the auricle before drilling. When a curved incision has been made behind the auricle, this is then pushed forward and held in this position. The growths are then exposed and can be drilled away without using reflected light. Afterwards the auricle is replaced in its normal position and sutured. Detachment of the auricle is sometimes necessary in the removal of exostoses.

4. **Inflammation.**—The external auditory meatus is subject to inflammation, diffuse or circumscribed, the latter occurring in the form of small abscesses or boils. Both affections are attended by acute pain, and in each the general health of the patient has been out of order for some time previous to the local trouble.

TREATMENT.—Treatment in the direction of improving the general health, and local bleeding by means of leeches applied in front of the tragus, will often rapidly relieve the diffused form of inflammation; but when it has continued for a long period (as it not infrequently does), in addition to the soft tissues the periosteum becomes affected. The passage then throughout its whole extent becomes so swollen as to nearly close the external opening, and pain is constant. The only treatment which gives complete and permanent relief under these circumstances is to make two or three free incisions down to the bone, along the whole extent of the osseous part of the canal. A convenient instrument for this purpose is a small sharp-pointed curved bistoury. As to the propriety of opening abscesses in this situation there can be no question, for, owing to the extreme denseness of the tissues and their approximation to bone in the external auditory canal, abscess in this part is slow in its progress and attended with very great suffering. These abscesses being especially liable to recur, a proper regimen and medicines appropriate to the failure in general health are required.

5. **Otomycosis.**—The external auditory meatus is occasionally the seat of varieties of a vegetable fungus, namely, *Aspergillus niger*, *fumigatus*, and *flavescens*. To the inflammation thereby induced the term 'otomycosis' is applied. The symptoms to which they have given rise have been great irritation and a slight serous discharge.

TREATMENT.—Frequent syringing with weak solution of perchloride of mercury or other suitable antiseptic, and instilling diluted alcohol after all flakes have been removed, will suffice to effect a cure.

6. **Polypus.**—Polypus of the ear is usually preceded by inflammation in the tympanic cavity and perforation of the membrane; it is considered along with diseases of the middle ear.

7. **Hæmatoma Auris.**—See HÆMATOMA AURIS.

II. MIDDLE EAR.—All affections of the middle ear originate in some part of that tract of mucous membrane which, commencing where the Eustachian tube opens into the pharynx, forms the lining of this tube and of the cavity of the tympanum, finally becoming the innermost layer of the tympanic membrane. To the character of this tissue is due the term 'catarrh,' which, in its two forms of

purulent and non-purulent, is used in describing any deviation from health which, directly or indirectly, is the cause of pathological change in the Eustachian tube or tympanum.

1. Obstruction of the Eustachian Tube.—

One of the most frequent conditions under which the Eustachian tubes become the seat of obstruction is that met with in children or young persons. The subjects of this affection present a very characteristic aspect. They breathe almost entirely through the mouth, which, sleeping or waking, is kept partially open; their tonsils are often enlarged, and they snore loudly during sleep. The mucous membrane of the nares and pharynx is swollen, and secretes in excess. Owing to this tumid state of the fauces the passages to the Eustachian tubes in this situation do not admit of the constant necessary supply of air to the tympana. The air in these cavities undergoes partial absorption, and thus becomes more rare than that external to the tympanic membrane; the density of the outer air remaining the same, the equilibrium of pressure is destroyed; the membrane, consequently, is retracted, the chain of ossicles is pressed inwards, and thus the conduction of sound becomes interfered with—in short, the patient is more or less deaf. In these cases inspection of the tympanic membrane at once reveals the state of affairs. As the cavity of the tympanum is not involved in the catarrhal change, its translucency and lustre are not impaired; the handle of the malleus is tilted inwards, the head of this bone is unusually prominent, and there is a distinct fold crossing the upper part of the posterior section of the membrane. Where the obstruction has lasted for a long period, the membrane will appear to have almost fallen upon the walls of the tympanum, and the promontory and incus may be distinguished. If under these conditions the tympanum be inflated on Politzer's plan, an instant return to good hearing follows. This method consists in passing a stream of air from an india-rubber bag through one nostril while the patient swallows some water. The operator at the same time closes one nostril with the forefinger of the left hand, and completes the closure of the other with the thumb. The mouth must be kept firmly shut. In the course of a few days the improved hearing partially dies away, leaving the patient, however, in some degree better than before the operation.

In all these cases a thorough examination of the pharynx should be made, inasmuch as it will be frequently found that the symptoms above mentioned are due to masses of adenoid growths in the vault of the pharynx. See NOSE, Diseases of.

TREATMENT.—When adenoid growths are present they should be removed, and if this is done thoroughly the patients recover their hearing completely in the course of a few weeks, and, what is almost of equal importance (this applies to the general health), nasal respiration is quite restored. When it is found upon examination that adenoid growths are not present, the treatment of obstruction of the Eustachian tubes will be of a more simple character. It should include the gentle syringing of the inferior nares with warm alkaline solutions. The most useful of these contain bicarbonate of sodium and borax. In using them the head should be bent forward, and a small nasal syringe employed; this is preferable to the nasal douche, since the stream from the douche is so powerful as sometimes to rush into the Eustachian

tubes, and so excite inflammation of the tympanum. Politzer's inflation should be employed, but not more frequently than two or three times in a week.

If the tonsils are much enlarged it will be necessary to remove them; but the reason for this proceeding is not that they press upon the openings of the Eustachian tubes, but that their presence keeps up the unhealthy condition of the pharynx.

Under this routine of treatment the patients completely recover their hearing; the space of time during which it is necessary to continue treatment varying according to the obstinacy which each case manifests.

Obstruction of the Eustachian tubes in *adults* presents certain well-marked differences from the affection as it prevails in *children*. An ordinary cold is the beginning of the trouble. It is more usual to find one instead of both tubes obstructed, and more often than not the tympanic cavity is involved in the catarrh. Where this is not the case—and it will be evident from the retained lustre and transparency of the membrane—the same principles of treatment as are pursued in the case of children will hold good, except in so far that the affection in grown-up persons is less persistent after the tube has been once artificially opened; and that, to effect this, Politzer's method is sometimes not sufficient, or, even if so, not as efficacious as the Eustachian catheter. It must also be borne in mind that in the treatment of cases, in which one ear is healthy, by means of the catheter, the affected ear exclusively may be subjected to the air-douche, while with Politzer's method it is impossible to avoid forcing a stream of air into the healthy tympanum, and this is not always an advisable proceeding.

The Eustachian Catheter.—The following is the mode of using the Eustachian catheter: 'Place the patient in a chair, and let him lean back, and steady his head with the left hand firmly fixed on the top of it; hold the catheter lightly in the right hand, with the curve downwards, and pass it quickly in this position through the inferior meatus of the nose to the posterior wall of the pharynx. When this is felt, withdraw the catheter about half an inch, and tilt the point of the curved end rather upwards, and to the left or right, according to the side which is being operated upon. Now hold the catheter and end of the patient's nose steadily between the thumb and the first two fingers of the left hand. All this time the ear of the patient and that of the surgeon are connected with the otoscope. The point of the catheter is now supposed to be in the pharyngeal orifice of the Eustachian tube; but the only certain sign of this being the case is that when air is forced into the catheter it will be heard through the otoscope to impinge upon the tympanic membrane.'

The catheter may be made of silver or of vulcanite, but, of whatever material, it must be inflexible while being used. Beyond this, suffice it to say here that in practised hands its employment is invaluable, and indispensable in the treatment of most affections of the middle ear, not only in overcoming obstruction of the Eustachian tube, but also as a means by which fluids may be introduced into the cavity of the tympanum. In making use of the air-douche an india-rubber bag fitted to the catheter should be employed, and in using injections to the tympanum a similar arrangement is necessary.

2. Catarrhal Inflammation of the Tympanum.

—When the tympanic cavity has become involved in the catarrhal state, or when the affection,

instead of proceeding up the Eustachian tubes, begins in the tympanum, as it frequently does, those changes have commenced which, of all others, form the most frequent impediments to the conduction of sound—in other words, which make the subjects in whom they are found more or less deaf; and it may be broadly stated that the extent to which this affection is remediable depends directly upon the time at which the patients suffering from it apply for treatment. In the early stages, the obstruction to the passage of sound through the tympanum is solely due to the effusion of mucus in this situation, and this is easily demonstrated by the moist gurgling sound which inflation of the tympanum produces, as may be heard upon connecting the ears of the patient and surgeon by means of a piece of india-rubber tubing. Afterwards comes what may be termed the dry stage, i.e. when the fluid portion of the mucus has suffered absorption, and when any of the products of inflammation may have become more or less organised, or at least in a condition which, if not interfered with, suffers no further change. The morbid conditions which result from non-purulent catarrh of the tympanum are twofold: firstly, those which affect the tympanic membrane, and are, therefore, demonstrable during life; secondly, those which are met with after death in the tympanic cavity. The first of these include changes in curvature, in colour, and in consistence.

The slighter changes in curvature have been noticed in speaking of obstruction of the Eustachian tubes, which condition is necessarily more or less present in all cases where the tympanum has been the seat of catarrh; and these changes are indefinitely increased until the state of complete collapse is reached. In this condition the membrane has the appearance of being in close apposition to the walls of the tympanum, and lapped round the ossicles, so that the forms of the malleus, incus, and sometimes the stapes are distinctly traceable.

In so extreme an example, the membrane is generally bound down to the tympanic wall by adhesions. The first change which the membrane exhibits is a loss of its lustre and transparency: it becomes opaque. Further alterations in colour, in cases of long standing, consist in the formation of patches of brown, yellow (colour of parchment), and white. Variations in consistency will include thickening throughout the membrane, or in parts of it, especially in the cases of dense chalky deposits (phosphate of lime); and thinning in places, so observable sometimes that inflation will induce bladder-like protrusions, which, as inflation is suspended, fall back again: changes in all these respects completely metamorphosing the appearance of the membrane. After death, within the tympanum may be found collections of dried mucus around the ossicles; thickening of the lining membrane; bands of adhesion in all directions; and ankylosis of the ossicles to each other as well as between the stapes and fenestra ovalis.

As additional evidence during life of obstruction in the tympanum, it may be mentioned that sounds from a vibrating tuning-fork placed on the vertex are intensified when such obstruction exists, and the nerve remains unimpaired. This test is especially valuable when one ear is healthy, inasmuch as the sound will be heard exclusively on the deaf side, this being due to the fact that vibrations of sound thus conveyed to the auditory nerve, on their passage outwards through the tympanum, meet with the

obstruction in this position, and are reflected on to the labyrinth. The appearances above described, together with the history of the case, serve sufficiently to distinguish affections of the conducting from those of the nervous apparatus; and the sounds which are produced upon inflation of the tympanum, whether of a moist or dry character, give evidence as to whether the mucus in the cavity of the tympanum is in a more or less fluid state, or has reached the dry stage when the fluid part of the secretion has become absorbed, the more solid portion remaining.

TREATMENT.—In the first of these conditions, the inflation at once increases the hearing-power; in the second, it produces no change in the hearing. An indication in this direction is a most useful guide in respect of treatment, which may be said to include injections of fluid or vapours into the cavities of the tympana. In the matter of the selection of these remedies, the greatest diversity of practice exists in the hands of capable authorities. Astringent solutions of sulphate of zinc, alkaline injections of bicarbonate of sodium, iodide of potassium, chloride of ammonium, and the vapours of iodine or chloride of ammonium, each and all find favour. If fluids are injected, a few drops only are to be introduced through the Eustachian catheter, a small bulb of india-rubber being used to force the fluid through the catheter. Vapours are employed by means of the many apparatus in general use. The diversity of opinion on this subject shows how difficult a problem is the treatment of the ulterior effects of catarrh of the tympanum.

The degree of improvement varies within wide limits, but the greater benefits may always unhesitatingly be predicted during the moist stage of the catarrh. Indeed, the necessity for early treatment is abundantly shown, in the instance of catarrhal affection of the middle ear, by the extremely satisfactory termination of cases treated early in the disease, and the slight relief which but too often follows when the affection has been allowed to proceed for years unchecked. In such cases as the latter, the fact that considerable quantities of inspissated mucus have been found in the tympanic cavities first suggested the operation of making an incision into the tympanic membrane, and attempting the removal of mucus through the incision by passing a stream of air through the tympanum. This proceeding, with certain modifications afterwards introduced, is no doubt very useful in cases favourable for its employment, but it should be reserved for those which have defied the less severe means, and where there is unmistakable evidence of an obstruction to the passage of sound through the tympanum. Experiments with the tuning-fork, already referred to, give valuable evidence in this direction. Many operations have been suggested for the relief of tympanic affections, but none of them have stood the test of time. One of the causes of failure in all operative measures on the tympanic membrane is to be found in the fact that it is impossible to keep any opening in this structure from healing. That such attempts at relief should have been made is not surprising when it is remembered how intractable some of these cases are, how one of the forms of catarrh which affect the tympanum is slow and insidious in its progress, and sufficiently distinctive in its character, to have applied to it by general consent the term *proliferous progressive catarrh*.

3. Purulent Catarrh of the Tympanum.—Perforation.—The form of tympanitis in which

the effused products become purulent is an acute and generally an extremely painful affection. Usually the pus rapidly makes its escape from the tympanum into the external meatus, by a process of ulceration through the tympanic membrane, leaving as its result a perforation of this structure. In quite the early stage the affection may often be cut short by the free application of leeches in front of the tragus, followed by fomentations, but more often than not the membrane has given way before the patient comes under observation. If however the patient can be seen before this has happened, and if there is evidence of the tympanic cavity being in a state of acute inflammation, there should be no hesitation about making a free vertical incision in the posterior section of the membrane. The purulent secretion having thus obtained an exit, the incision will heal in a few days and the danger of septic infection be avoided. Should the patient come under notice shortly after the membrane has given way from ulceration, then if the tympanic cavity be emptied of the pus by the free use of Politzer's inflation and repeated syringing, the opening will often close, and leave very little, indeed sometimes hardly any, appreciable deafness. When a purulent discharge through the opening is allowed to go on unheeded for any length of time, it is the exception for the perforation to heal. This condition is constantly seen after scarlet fever, measles, or any of the exanthemata. A perforation of the tympanic membrane presents an infinite variety of aspects, from a small pinhole to nearly complete loss of the membrane, but there will always be a slight remaining external rim of membrane. This latter is perhaps the most frequent of all forms of perforation, and especially when the ulceration dates from an attack of scarlet fever. Although the handle of the malleus occasionally remains, it more usually comes away in these and other cases where the loss of tissue is very extensive. The head of this bone, however, may always be distinguished, unless there has been complete disorganisation of the tympanic cavity. Among other forms of perforation commonly met with may be mentioned those in which the anterior or posterior half of the membrane is left, and is bounded internally by the handle of the malleus; the so-called *reniform* perforation, where the lower part of the membrane is lost, and the umbo of the malleus indicates the position of the hilum of the 'kidney'; and the small, smooth-edged circular perforation which is common alike to all parts of the membrane. Occasionally, though not very often, the tympanic membrane is the seat of a double perforation. Similar variations in hearing accompany this condition, between slight deafness and total loss of hearing power. The size of the perforation affords no guide in this respect, extreme loss of hearing being met with when the perforation is very small, and very slight deafness where the loss of tissue has been most extensive; so that it may be unhesitatingly stated that the loss of the membrane is but in a very small degree the cause of the deafness in these cases, the disorganisation in the tympanic cavity mainly accounting for this. Such disorganisation is at times so complete (especially after scarlet fever) as to include the loss of all the ossicles, total deafness, and paralysis of the muscles supplied by the facial nerve. A very small perforation in the anterior and superior part of the membrane may from its position escape notice, but the diagnosis can be always verified by the facility

with which air may be made to pass through the opening, or the reverse, provided that the communication between the Eustachian tube and the tympanum is not closed by cicatricial tissue—a very rare condition when so little of the membrane has suffered ulceration.

One other form of perforation deserves mention, rather on account of its frequency than its importance, namely, perforation of Shrapnell's membrane, which is that part of the membrane proper lying just above the short process of the malleus. Ulceration of this small area, which is merely a protective membrane, may exist without any impairment to hearing so long as the remainder of the tympanic membrane is entire, so that it would appear to perform no function so far as a conducting medium is concerned, but it is important to bear in mind that when it is perforated the lesion is at times connected with exposed bone.

TREATMENT.—The treatment of purulent tympanitis and perforation will include assiduous cleanliness; keeping the Eustachian tube free from obstruction; and the use of local remedies. Among the most useful of these are weak solutions of alcohol, and insufflation of gallic acid or boric acid. As the condition of the ear improves under these measures, so will the hearing-power vastly increase when it has not been completely lost; but there still remains the oftentimes invaluable application of what is spoken of as the *artificial* membrane. Of all kinds, the best undoubtedly is the flattened pad of moistened cotton-wool, applied by the patient, every morning, with forceps constructed for the purpose. Until this be tried in each case it is impossible to say whether it will do good; but when it is useful—as it is in a large number of cases—by its help the patient will recover very good hearing, and this even when the perforation has existed for a period of many years. That its effects depend upon the support which it gives to the ossicles, thus re-establishing the normal pressure of the stapes upon the fenestra ovalis, has been unquestionably demonstrated.

4. Polypus.—One of the most frequent complications in cases of perforation of the tympanic membrane is polypus, a term employed to designate a fleshy tumour in the ear. Although polypi are occasionally present in the meatus independently of perforation, the most usual situation from which they arise is the lining membrane of the tympanum. Sometimes the exact point of origin is the edge of a perforation, and still more rarely the sides of the meatus. In size these growths vary from a small protrusion through a perforation, to a tumour which entirely fills the meatus and projects externally from the ear. In this latter instance the growth presents a very distinctive appearance, not unlike a raspberry. Sections of aural polypi hardened in chromic acid with few exceptions show their structure to be fibro-cellular, the fibrous element preponderating over the cellular in proportion to the age of the tumour.

But it should be remembered that the term 'polypus of the ear' is habitually used when the masses, small or great, are granulation-tissue springing from exposed bone within the tympanic cavity, and in such cases are of great significance, demanding very radical measures, which are mentioned later on in this article.

TREATMENT.—In all cases polypi should be removed, and the best instruments for this purpose are the rectangular 'ring' polypus-forceps, or, in the

case of a large growth, a Wilde's snare. The nature of the instrument used is not important and will be suggested to the surgeon by the appearance of the mass which has to be removed. Very often a scoop commonly employed to clear away dead bone is the most convenient instrument. Owing to their remarkable tendency to recur, removal is only the preliminary step in treatment. The principal part of this consists in their complete eradication by caustics. Of these the most efficacious and convenient is chloro-acetic acid, and later in the treatment nitrate of silver. The acid should be applied with great care by using it on the point of a probe defended by a small twist of cotton, and a convenient form of nitrate of silver is a bulb of the melted salt fused on to a probe of platinum wire. The caustic should be applied daily for some time after the polypus has been removed, and then less frequently. The treatment should also include the same scrupulous cleanliness and application of astringents, so desirable in the case of perforations. It is simply the want of attention to details that failure in the treatment of aural polypus may be ascribed.

COMPLICATIONS OF TYMPANIC DISEASE.—

(a) *Facial Paralysis*. This is due to inflammation around the portio dura in its passage through the aqueduct of Fallopius. When suppuration in the tympanic cavity, with caries of the bony canal, precedes or accompanies the loss of function in the nerve, recovery is hopeless; but when the paralysis follows a subacute catarrh of the tympanum, not ending in a perforation, as is sometimes the case, the paralysis in time disappears no less certainly than when it is dependent upon an affection of the nerve at a point after its exit from the temporal bone.

(b) *Pyæmia; Cerebral Abscess; and Meningitis*.—When the mastoid cells become the seat of inflammation, the pain, tenderness, and pitting on pressure over the mastoid process will at once suggest an early incision down to the bone; and it may be truly said that this is often delayed too long, and perhaps is never done too soon. Again, when the symptoms point definitely to pus within the mastoid cells, the bone should be pierced so as to make the external opening communicate freely with the cells. Relief given in this way will occasionally be the means of saving life, by preventing the absorption of poisonous material into the lateral sinus. Besides pyæmia thus induced, other fatal issues which suppuration in the middle ear frequently entails have their starting-point in the tympanic cavity, and in such instances cerebral abscess or meningitis may be the immediate cause of death. See BRAIN AND MENINGES, Septic Inflammation of.

Fatal terminations of this nature most frequently occur when cleanliness and local treatment of the ear have been neglected. Hence the necessity for such care.

But the fact that even under the most favourable conditions such events are possible, when there is a fistulous opening in the tympanic cavity, should induce caution with insurance-offices in accepting the lives of persons with this lesion, at the ordinary premiums. Fatal cases of this kind might seem in practice to be almost divisible into two classes—namely, those in which cerebral symptoms come on soon after the establishment of the perforation; and others where there has been a purulent discharge from the ear (that is, from the tympanum)

for many years before the advent of such symptoms. In this latter class must be included those cases in which the temporal bone has become the seat of caries; and it may be stated, subject to no exceptions, that whenever exposed bone can be detected by means of examination with a probe within the cavity of the tympanum, the subjects of this condition are always in a perilous state, and that at any time fatal symptoms may commence with a severe rigor. For this reason, even when exposed bone cannot be absolutely demonstrated in the way mentioned, the existence of bone-granulations where there is a perforation of the tympanic membrane should be regarded as a most serious complication. The same danger, though in a very much less degree, may be said to be present when dead bone can be detected in the mastoid process—in a less degree, because the outer table of the bone is often affected while the inner remains healthy. The dead bone then in the former position becomes exfoliated, and the external wound heals.

Since the last edition of this Dictionary was published the surgical treatment of Mastoid Disease and perforation in which symptoms of septicæmia have shown themselves has undergone a complete revolution. Where there is undoubted disease of the mastoid with pus within the antrum or mastoid cells, or where there are symptoms of septicæmia, what is termed the complete mastoid-operation is now performed. This consists in making a long curved incision behind the mastoid, laying this bone quite bare, pulling forward the flap thus formed as well as the external ear; drilling with a burr (turned by an electric motor) close to the external meatus till the antrum is reached, making this cavity continuous with the attic and tympanic cavity; scraping away all dead bone and thus effecting complete drainage, at the same time removing all diseased bone. Up to quite recently the difficulty has been that in many cases a certain amount of discharge has continued. Ballance has recently pointed out that at a second operation, conducted about two weeks afterwards, he has again pushed forward the flap and grafted skin over the surface of the cavity above named, and thus the complete and permanent cessation of the discharge has been ensured, and any possible septicæmia prevented.

The difficulties in selecting cases suitable for this operation are obvious, and we may formulate those in which the necessity exists as follows:

Firstly, undoubtedly in cases where septicæmia has commenced. Secondly, undoubtedly in cases where there is dead or carious bone in the tympanic cavity accompanied by ominous symptoms often repeated. Thirdly, wherever there is evidence of mastoid disease of long or short standing. Fourthly, in a certain proportion of cases where there is evidence of dead or diseased bone, but a very doubtful history of ominous symptoms. Fifthly, in a certain proportion of patients with intractable otorrhœa, in whom no bone-disease can be detected, and in whom no history of ominous symptoms can be obtained.

(c) *Malignant Disease*.—The seat of origin of the new growth is generally the lining membrane of the tympanic cavity, and a purulent discharge from this surface always precedes the appearance of the cancer. In its early stages cancer in this situation bears a strong resemblance to the ordinary forms of polypus.

III. INTERNAL EAR.—Apart from deafness due to local changes in the external or middle ear, the function of hearing is subject to impairment from causes which have their seat in the nervous structures of the ear; in other words, although the conduction of sound may be good, the perception of sound may be faulty. The inability to hear the vibrations of sound conveyed through the cranial bones, such as from a vibrating tuning-fork placed upon the vertex, is indicative of this condition. For the rest, the absence of tympanic disease, and the history of the case, must supply the evidence required for a diagnosis. Familiar examples of this nature are the deafness which often accompanies old age, or which is left after fevers when the middle ear has not suffered; the two forms of syphilitic nervous affection to be presently mentioned; the sudden and sometimes total loss of hearing which occasionally follows severe mental shock; the deafness after loud explosions near the ear, so common in artillerymen and naval men; and that which is caused by blows on the head and 'boxes on the ear.' An attack of mumps will sometimes leave behind an irremediable loss of hearing in one or both ears, unattended by any discoverable change in the tympanum. In a similar way weakly women occasionally become more or less deaf during their confinement, and this symptom becomes aggravated as each successive child is born. In connection with this subject, prolonged suckling may be mentioned as one of the numerous debilitating causes which undoubtedly aggravate the trouble of an already impaired ear.

Among the nervous affections of the auditory apparatus possessing certain characteristics which serve to distinguish it from others is the so-called *Mènière's disease*, an attack of which at times gives rise to symptoms which would be alarming if their true origin passed unrecognised. A patient who suffers in this way is seized with an attack of vertigo so severe that he not infrequently falls, and for some hours afterwards requires assistance in walking; occasionally vomiting succeeds the giddiness; and he recovers to find himself very deaf in one ear, with which previously he had heard well. Milder attacks of the same nature generally follow the first, and each one leaves the patient more deaf. Although with many writers it is a favourite theory that the seat of morbid changes in this disease is in the semi-circular canals, up to the present the point has not been satisfactorily determined. No treatment appears to exercise any influence upon the disease. *See* VERTIGO.

Syphilis.—The affections of the ear due to syphilitic disease demand separate consideration. They occur under the following varieties: Firstly, in the form of sores and warts in the external meatus, which yield to local treatment. Secondly, affections of the middle ear during the secondary ulceration of the throat, the treatment for which, beyond specific medicines, in no way differs from what is useful in the ordinary catarrh of the same parts. Thirdly, failure in hearing-power during the secondary stages, unattended with any change in the middle ear: this disappears under constitutional remedies. Fourthly, the loss of function in the auditory nerve, so commonly met with in the subjects of inherited syphilis. In this last variety the hearing-power begins to fail between five and fifteen years of age (seldom later in life), and proceeds to very great and often total deafness, the period between good hearing and the extreme point of deafness arrived at varying

from a few weeks to several years. From this cause children sometimes become in the course of a month or six weeks totally deaf, but such rapidity is exceptional. Experience has shown how powerless treatment is to arrest the progress of this affection, so that attention should be confined to preventing its subjects from becoming dumb, if they are attacked after they have acquired speech, and before they are likely to forget it, namely, from about four to seven years of age. This is best attained by teaching them lip-reading; and if they can read, by making them do so (*aloud*) several times each day. In this way a child will retain its recollection of language when otherwise speech would pass away.

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EAUX-BONNES, in the French Pyrenees.
Sulphur waters. *See* MINERAL WATERS.

EAUX-CHAUDES, in the French Pyrenees.
Sulphur waters. *See* MINERAL WATERS.

EBERTH'S BACILLUS.—A synonym for the *Bacillus typhosus*. *See* TYPHOID FEVER.

EBURNATION (*ebur*, ivory).—A state of bone-tissue in which it assumes the whiteness, smoothness, and hardness of ivory, in consequence of an increased deposit of calcareous matter. It occurs chiefly in rheumatoid arthritis. *See* RHEUMATOID ARTHRITIS.

ECBOLICS (*ἐκβολή*, abortion).—This name is given to the measures which produce abortion, or expulsion of the contents of the uterus. In moderate doses ecbolic drugs may act as emmenagogues. *See* EMMENAGOGUES.

ECCHYMOSIS (*ἐκ*, out of; and *χυμός*, juice). An extravasation of blood into the tissues, due either to injury or to disease. It presents at first a more or less blue or bluish-black appearance, which changes with age, passing through green to yellow. *See* EXTRAVASATION.

ECHINOCOCCUS (*ἐχίνος*, a hedgehog; and *κόκκος*, a grain or berry).—*See* ENTOZOA.

ECHOKINESIA (*ἠχώ*, echo; *κίνησις*, motion). The involuntary imitation of movements performed by another person.

ECHOLALIA (*ἠχώ*, echo; *λαλία*, speech).—The involuntary repetition of words spoken by another person.

ECLAMPSIA (*ἐκλάμπω*, I flash, I explode).—This term is now used as a synonym for convulsions, whatever may be their cause. *See* CONVULSIONS IN CHILDREN; and EPILEPSY.

ECSTASY (*ἐξίστημι*, I amaze).—**DEFINITION.**—The term 'ecstasy' has been applied to certain morbid states of the nervous system, in which the attention is occupied exclusively by one idea, and the cerebral control is in part withdrawn from the lower centres and those for certain reflex functions. These latter centres may be in a condition of inertia, or of insubordinate activity, presenting various disordered phenomena, for the most part motor.

DESCRIPTION.—The subjects of ecstatic phenomena are commonly of the female sex, or are men who lead celibate and ascetic lives. To such individuals they are in the present day almost confined.

In the middle ages, on several occasions, under special circumstances, an intense dominant emotion, with some attendant ecstatic manifestations, was known to spread widely by a sort of moral contagion.

Women who are the subjects of this morbid state are usually single, frequently present menstrual irregularities, and often distinct evidences of hysteria, of which the ecstatic condition may be but a part. The immediate cause of the attack is usually some repeated vivid emotion, commonly religious, sometimes one of fear. The direction taken by the motor or other phenomena of the ecstatic state is often very obviously determined by imitation. With this direction there is associated, in some cases, the assertion of supposed facts, which transcend the ordinary course of natural phenomena, and which have been proved, in many instances, to depend on intentional fraud.

As forms of ecstasy we have the condition of some religious enthusiasts, in whose dominant state all other mental processes are merged. Dreams and visions are determined by the ecstatic emotion and add to its intensity. Consciousness of the body may even be lost, so that all sensation is gone for a time; while the corporeal functions, including ingestion and egestion, are reduced to a minimum, and a little exaggeration may represent them as in complete abeyance. Hence the 'fasting girls' that have arisen in various countries, by whom 'stigmata'—marks, in the positions of the nails employed in crucifixion—are sometimes presented, probably by artificial production. In some hystero-epileptics a state of ecstasy—of rapt, intense emotion—forms part of the paroxysmal seizures, and then wild muscular spasms replace the tranquil repose of the more volitional ecstasies. Occasionally—when an intense emotion is shared by many persons—insubordinate muscular movements occur, of a rhythmic character, seen in the 'Jumpers' and 'Shakers' of the present day, and more strikingly in some of the dancing religious ceremonies of half-civilised races, and in the dancing epidemics of the Middle Ages. Such was the original dance of St. Vitus, in which the exciting emotion was religious; and such also the 'tarantella,' in which the excitant was terror at the supposed consequences of the bite of the tarantula, which the dance was intended to avert. The phenomena now manifested in the epidemics that have been described under the name of the 'Jumpers,' &c., merit, indeed, more detailed notice, since they have been observed in remarkable degree and conspicuous form in certain races in whom the process of brain-development concerned in what we term 'civilisation' is taking place. The conditions especially favourable for the development of these disorders appear to be generated by the close contact of such races with those in whom the process of evolution of the nervous system has been carried farther; and the less developed organisation is, as it were, strained by a connection which it is not strong enough to endure. At all periods a pathogenic influence has probably also been exerted by the excessive tension placed on some mental faculties by certain religious systems, but the effect is greater when the strain comes from without, and the nervous constitution is warped by the inequality between its own strength and that of the race with which it is not only in contact but attachment. The cerebral functions may be deformed by emotions that would be powerless to

disturb a brain whose functions were strengthened by equal development in all parts.

It is especially among the negro tribes in contact with more civilised white races that the most conspicuous forms of ecstatic disease have been observed. A condition described by Hammond under the term 'Myriachit' (Russian, *Miriatchit* = 'playing the fool') is almost identical with that met with in the so-called 'Jumpers.' The subjects of this condition have been met with chiefly in Siberia; both sexes are liable, but most sufferers are females. It occurs in epidemics, especially involving several members of the same family; although sporadic cases are often met with. Contact with a person who is the subject of this affection is often the exciting cause; but sometimes the malady is distinctly hereditary. Natives and acclimatised immigrants are exclusively attacked, and of them only children and persons of a low intellectual development. The characteristic of the disease is that its subjects are compelled to imitate any sound, gesture, grimace, or act, even if it gives rise to pain, anger, or remonstrance. As a rule the malady is chronic, but presents spontaneous remissions. The general health is unaffected, and the patient is not incapacitated from work. Thus the chief difference between this condition and most other ecstatic states is, that the subjects of Myriachit react only and simply to impulses entering through the afferent optic and auditory channels; and when an order is given they do not perform the acts, but simply repeat the words of the order.

There are many kindred affections of which 'Latah' (nervous or ticklish), met with in the Malays, is a good example. Sudden or particular sounds, and suggestions by word or pantomime suffice to throw the victim into a curious psychical state in which certain sounds or words are emitted, or movements performed which are beyond the control of the will. There does not, however, appear to be any tendency to contagion with consequent spread of the affection in epidemic form. Attempts to conceal their misfortune prove futile, as even an unexpected touch may evoke an explosion of obscene language totally irrelevant to the occasion.

Another of these affections is 'Ikota' which is met with among the Samoies, but which is said to occur only in married women, in whom a distasteful spectacle or an allusion to their misfortune will call forth inarticulate sounds or even induce a transitory paroxysm of mania.

The subjects of convulsive tic (*Gilles de la Tourette's disease*) manifest a psychical state akin to that met with in these other maladies; but in addition to the impulsive tendency to mimicry, both utterances and movements are commonly spontaneous and need no afferent impulse to evoke them.

The state of 'somnialesia,' or 'sleep-drunkenness,' is closely allied, psychically, to the conditions under discussion, although differing in its associations, by which it is connected more closely with epileptic mania than with the ecstatic disorders. The subjects of this condition perform some act, it may be even of violence or murder, on being suddenly awakened from sleep—the action being often prompted by a dream.

The close resemblance between ecstasy and some forms of hysteria has already been alluded to; but what is of still greater interest is the fact that suggestion plays a most important part in all the conditions that have here been discussed, and that all these conditions seem, as it were, to form links

in the chain which connects the dancing-epidemics of the Middle Ages with the phenomena of suggestion and hypnotism of the present day.

A remarkable case is quoted by Hammond, of a gentleman who would perform any act he was told to do by a person whispering into his ear while he was asleep.

TREATMENT.—It is rarely now that ecstatic manifestations have to be treated except as part of pronounced hysteria, and the treatment is that of the hysterical state which underlies the ecstasy. The measures of paramount importance are the substitution of a 'healthy moral atmosphere' for that under which the symptoms have arisen; and the exposure of actual fraud. Occasionally, even now, examples of solitary ecstasy come under observation. In these cases considerable care and tact are needed. Ecstasies are not amenable to the motives which influence most persons, and, if there is actual fraud, will sometimes die rather than be found out. In the case of 'fasting girls,' due observation of the body-weight during a short time will answer as well as, and is much safer than, a long exclusion of food. But the removal of the ecstatic to other surroundings is the most important step for both detection and cure.

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J. S. RISIEN RUSSELL.

ECTHYMA (ἐκθύω, I burst out).—A crusted eruption due to the action of pyogenic organisms. See IMPETIGO.

ECTOPIA (ἐκ, out of; and τόπος, a place).—An abnormal protrusion or displacement of a part; for example, *ectopia vesicæ*, protrusion of the bladder. See ORGANS. Displacement of.

ECTROPION } (ἐκ, out of; and τρέπω, I turn).
ECTROPIUM }

A condition in which the eyelid becomes everted, so that the conjunctival surface is exposed. See EYE, AND ITS APPENDAGES, Diseases of.

ECZEMA (ἐκζέω, I bubble up).—SYNON.: Fr. *Eczème*, *Ecéma*; Ger. *Eczem*, *Salzfluss*.

DEFINITION.—An acute, chronic or recurrent, superficial inflammatory disease of the skin, associated in its most typical forms with the presence of exudation either in the papillary or epidermic layers, and resulting in the formation of papules or vesicles; the latter, which quickly burst, discharge a gummy fluid with special characters and leave an excoriated weeping surface; the disease is always accompanied by more or less itching.

This definition is intended to embrace various forms of catarrhal skin-inflammation regarded by many recent writers as different diseases, and especially to include the condition termed Seborrhoeic Eczema, which constitutes the greater number of cases of 'eczema'—as the term is generally understood in England—which present themselves for treatment.

Eczema is by far the most common disease of the skin, its frequency, as estimated by various authors, ranging from one half to one quarter of all skin-diseases. As our knowledge progresses cases classed as eczema will doubtless tend to diminish in number.

ÆTIOLOGY.—*Influence of Age and Sex.*—Eczema occurs at all periods of life, from infancy to old age, in both of which it is commoner than in middle

life. The chief incidence of eczema is on the male sex in childhood and in middle life, while it is on the female sex from ten to twenty years of age and at the menopause. In old age the two sexes are equally affected. In childhood the seborrhoeic forms predominate; in middle life the weeping or dry and scaly types accompanied by gouty and dyspeptic symptoms are most frequent; while in advanced age the erythematous cases with much nervous disturbance are commonest.

Eczema must be regarded as the mode of reaction of the skin of certain individuals to various stimuli—mechanical, thermal, or chemical; but of the immediate or determining cause of this reaction we have no knowledge. We are therefore compelled to invoke the existence of a special vulnerability of the skin in such persons which renders them liable to the disease under influences which would not produce eczema in persons not so predisposed. This vulnerability of skin may be inherited, but in no other sense is the disease hereditary.

Ichthyotic skin is especially vulnerable to cold winds; and low grades of ichthyosis—which is the most hereditary of all cutaneous abnormalities—while of comparative frequency, often escape observation. Many so-called hereditary cases of eczema may be thus explained, and this explanation covers the statement of many older writers that the skin of eczematous patients is often excessively dry and deficient in subcutaneous fat.

Various irritants (croton oil, arnica, noxious substances used in divers trades) when applied to the healthy skin give rise to a *local dermatitis* the severity of which is proportional to the strength or duration of application of the irritant, the extent of which is exactly that over which the irritant is applied, and which ceases to spread and tends at once to recover on the discontinuance of the irritant. In a person predisposed to eczema, however, the severity of the skin-inflammation is out of proportion to the strength of the irritant; the disease transcends the limits of the local application and appears on distant parts of the body, symmetrical areas being often attacked; while its duration after discontinuance of the irritant is indefinite. This distinction between local dermatitis and true eczema, although easily established in many instances, is not always so, the class of cases called 'trade-eczemas' marking the border-line between the two.

Predisposing local or external causes.—Mechanical irritation by pressure, friction, handling of gritty substances (grocers, bricklayers), &c., is responsible for a certain number of cases, but irritation of chemical origin is of greater frequency and importance. Coarse soaps, containing an excess of free alkali and often made with impure fats, are a frequent cause of eczema, as are various dyes, alkalis, and acids, while even hard water is injurious, especially if too frequently used. To the eczematous skin sunlight is particularly noxious in virtue both of its thermal and chemical rays, and so to a less degree is artificial heat (stokers, cooks). Cold, especially when combined with wet and if the exposed parts are not carefully dried (washerwomen, barnmaids), is often a potent factor in the causation of eczema; and so is east wind, which is usually not only cold but is also laden with irritating particles of brine.

The presence of varicose veins, most common on

the legs of old people, gives rise to circulatory stagnation, and by thus diminishing the vitality of the skin renders it peculiarly vulnerable to irritants and, perhaps, to invasion by the micro-organisms which are found in such great abundance about the feet (Payne).

Intermediate between the local and general predisposing causes of eczema and partaking of the characters of both are the common conditions of Seborrhœa (Steatorrhœa) and Hyperidrosis. In both, changes may occur, probably under the influence of micro-organisms which result in the supervention of inflammatory phenomena upon what was previously a purely secretory disturbance. As the sweat has been proved to contain a considerable amount of fat, the changes in the two conditions are probably identical, and consist mainly in the formation of bodies of the fatty-acid series which act as chemical irritants. The eczema-producing qualities of sweat are exemplified by the dermatitis often resulting from a Turkish bath, by the so-called 'critical sweating' caused by various active hydropathic measures, and by the troublesome sweat-eczemas which so often prove a source of annoyance to Europeans living in the tropics. The morbid influence of decomposing sebum and sweat is further evidenced by eczema of the axillæ, flexures, perinæum, and interdigital regions of the feet and hands, and by the 'intertrigo' of folds of skin lying in apposition. The local origin—in part at least—of these various forms of eczema has a very important bearing upon their treatment.

Prominent among the *general predisposing causes of eczema* are digestive disturbances resulting in the circulation in the blood of toxic substances (auto-intoxication). The gastro-intestinal catarrh of children (due often to injudicious feeding), the chronic dyspepsia of adults with constipation and gastric dilatation (often associated with alcoholism or excessive tea-drinking), as well as anæmia, plethora, and obesity, all of which are common concomitants of eczema, come under this category. Persons of gouty habit or with gouty family antecedents are prone to suffer from eczema from slight causes, possibly by reflex disturbance from the intestine (Crocker); and in them the disease is particularly troublesome and obstinate; but in this limited sense only is the common statement true that 'gout gives rise to eczema.'

The influence of functional derangements of the nervous system is paramount in the etiology of many forms of eczema. There is no more fertile cause of acute eczema than mental shock, worry, anxiety, or grief; and no more common cause of its persistence and intractability than neurasthenia. Again eczema may be of reflex nervous origin from dentition, intestinal worms, and gastro-intestinal irritation, or from pregnancy, uterine or ovarian disease, or the various functional derangements incidental to the menopause. It may be associated with asthma, attacks of the two diseases sometimes alternating, and it is often apparently the immediate result of a chill.

In an extremely limited number of cases eczema may follow the distribution of peripheral sensory nerves, but the degenerative changes described in them by Colomiati and Leloir have not been confirmed by subsequent research.

The bacteriology of eczema.—This subject has recently been to a certain extent satisfactorily settled by the establishment of the fact that the agency of a living germ is not necessary for the production

of eczema. The vesicle typical of the disease is *amicrobic* (Török, Sabouraud, Brocq, Kreibich). Eczematous surfaces, however, soon become invaded by numerous micro-organisms, prominent among which are the clumps of cocci to which Unna has applied the name of *Morococchi*, and which he considers as the specific cause of eczema. Recent cultural researches (Sabouraud, Jadassohn, Galloway) have distinctly proved that '*Morococchi*' are merely an unusual form of *Staphylococcus aureus* or *albus*, the common forms of which are always present in abundance. The generally accepted view is that these staphylococci, as well as streptococci, are the causes of the persistence and local infectivity of eczema, and they are certainly responsible for the majority of impetiginous and other secondary changes which may ensue. In the seborrhœic and flexural types of eczema the agency of these micro-organisms is especially probable, and Payne regards 'the scalp, perinæum, and feet as bacterial nurseries where certainly many more than one species vegetate and from which they may spread to the neighbouring parts, giving rise to local inflammations or eczemas.' The truth of this conjecture is borne out by the result of antiseptic remedies and occlusive applications.

SYMPTOMS.—Eczema is remarkable for the varying appearances it assumes according to the acuteness of the inflammation, the stage of the disease, and the parts affected. It is markedly polymorphous and its various phases usually co-exist.

Acute eczema is sometimes ushered in by some malaise and slight constitutional disturbances, with local sensations of burning and itching. Soon some redness, swelling, tension, and infiltration of a patch of skin appear, the redness merging gradually into the colour of the surrounding parts. From changes in the upper epidermic layer the erythematous patch is often covered with minute scales. Soon increase of the hyperæmia, with infiltration of the papillary layer of the corium and thickening of the lower epidermic layers, results in the formation of papules of solid appearance but really containing fluid. Then the further spread of the infiltration invades the rete Malpighii, raising the upper epidermic layers to form vesicles. The contents of these are at first clear; soon, however, they become turbid owing to the presence of more or less numerous leucocytes. Should these increase greatly in number, the contents of the vesicles, which frequently coalesce, become distinctly pustular, but this is exceptional, unless the individual be strumous or cachectic. In a typical case the upper epidermic layers soon yield, either spontaneously, or as the result of the scratching of the patient, giving vent to a discharge usually termed 'serous.' It differs, however, from true serum and more closely resembles blood-plasma or the fluid which agglutinates the edges of a wound after the cessation of hæmorrhage. It possesses the familiar attribute of stiffening linen, and is rich in albumen and salts which render it acid and irritating, in contradistinction to serum (e.g. from an œdematous leg), which is a thin watery fluid, bland and unirritating.

When the vesicles burst the rete Malpighii is laid bare as an excoriated weeping surface studded with minute red points, corresponding to the exposed vascular tufts, and tiny bead-like drops of plasma exuded from them. The discharge, which is usually abundant, dries up, mixed with shed epithelial cells to form crusts, which, if scanty, are thin and pale

yellowish in colour. If the vesicular contents are abundant and rich in leucocytes, and bleeding occurs from the exposed capillaries, the crusts or scabs are thick, dark in colour, and more adherent, such conditions often obtaining in the eczema of strumous children.

To the various stages of eczema just briefly described the epithets *erythematousum*, *papulosum*, *vesiculosum*, *rubrum* (*madidans*), *crustosum*, and *impetiginosum* are commonly applied. Return to health is first evidenced by diminution in the amount of discharge and consequently of scale-formation. This is followed by extension of well-cornified epithelial cells from the edge over the excoriated surface. Even when this process is complete the disease may be far from cured. The skin may remain congested, infiltrated and indurated, while copious desquamation may continue, resulting in the formation of more or less extensive adherent scales (*E. squamosum*), and this condition may be a very persistent one.

Difficulties in the diagnosis of eczema arise mainly from (1) *arrest in its early stages*; (2) *persistence in its later stages*; or (3) *masking of the primary eruptive elements by secondary accidental lesions*. Thus, in old people the face and scalp are frequently the seat of a particularly obstinate form of eczema which shows itself merely as an erythema, sometimes with a little infiltration and some branny desquamation. The subjective symptoms of burning and itching are intolerable, probably from pressure on the sensory nerve-endings, and the objective signs are no doubt mainly due to the persistent rubbing and scratching of the patient. Acute eczema of the face in younger people is often accompanied by so much oedema of the skin and subcutaneous tissue, especially about the eyelids, as to simulate an erysipelas.

Again the arrest of the eczematous process in the papular stage results in a condition often misconstrued. Although the careful examination of a papule with a lens will usually reveal the presence of a little fluid, this method of diagnosis is often not available, as the itching which accompanies papular eczema is generally so excruciating that the tops are torn off the papules by the patient's scratching as soon as they are formed, generally with relief to the symptom. The resulting groups of blood-crusts papules are, however, very characteristic and serve to distinguish the condition from forms of lichen with which it is apt to be confounded.

The persistence of eczema in its later phases, especially on the palms and soles where the epidermis is thick, gives rise to marked hyperkeratosis, or thickening of the horny layer of the epidermis, the cells of which are immature, imperfectly cornified, and therefore abnormally adherent. The thickened epidermis cracks down to the rete Malpighii, causing deep painful fissures (*rhagades*) corresponding to the normal skin folds (*E. rimosum*).

Suppuration in eczema (*E. impetiginosum*, *pustulosum*) is, as has already been stated, exceptional, but may occur in strumous children and in persons greatly debilitated by other diseases. It is doubtless generally due to the agency of pyogenic cocci which often cause boils at the same time, but may occur independently of them; and the suppuration generally takes place around and in connection with hair-follicles and their sebaceous appendages (*E. folliculare*). The complication often persists after other eczematous changes have passed away, owing to the deep penetration into the skin of the causative agents.

On the legs, especially in old people, where the

veins are varicose or the arteries sclerosed, inflammation may extend deeply into the corium or even into the subcutaneous tissues, giving rise to great brawny hardening (*E. induratum*); and as a further result of the same process hypertrophy of the papillae may ensue, producing huge warty thickenings or elephantine enlargements (*E. hypertrophicum*, *E. verrucosum*), the ulceration of which may closely resemble epithelioma. These latter changes never occur in skin which is soft and pliable, e.g. the flexures.

Chronic eczema may not only result from the persistence of one of the stages of eczema, as already described, but often is the result of recurrences of acute or subacute attacks, a tendency to which is one of the most characteristic and troublesome features of the disease.

Constitutional Disturbances.—It is, as a rule, only in the most severe forms of eczema that the general health is markedly impaired; in them there may be some rise of temperature (1° – 2° F.), but generally the tongue remains clean, the appetite good, and the digestive functions intact, although occasionally there may be constipation. Albuminuria or any other abnormality noted in the urine must be regarded as purely accidental. Extreme itching, sometimes of maddening intensity, especially at night, often produces obstinate insomnia, and may cause much mental excitement, especially in persons of neurotic temperament, in whom the subjective symptoms are frequently out of all proportion to the objective phenomena present. In very acute cases smarting, burning, or pain may replace itching, but as the inflammatory symptoms subside itching always ensues.

Regional Distribution.—Although the skin of any part of the body is liable to be attacked by eczema, yet it may be said to have a preference for certain regions. Thus, it is common where skin and mucous membrane join (mouth, anus, vulva), where local causes play an important part in the perpetuation of the malady; it is common wherever hair is abundant, wherever sebaceous and sweat-glands are numerous (scalp, face, hands, feet), and wherever the skin is soft, thin, or thrown into contiguous folds, e.g. the flexures. Eczema is often symmetrical, accurate symmetry being a general feature of cases of nervous origin, but there are many exceptions to the rule.

Special types of Eczema.—*Nervous or true eczema* may be defined as eczema which occurs on previously healthy skin, and in which secondary changes are less prone to occur than in all other forms. It generally arises as the result of some mental shock in neurotic or gouty-neurotic persons. Its onset is sudden, and the accompanying subjective symptoms severe. It is often markedly symmetrical, showing a predilection for the extensor surfaces of the limbs; its lesions are often grouped, accurately delimited, and do not tend to peripheral extension. It frequently is arrested in the papular or vesicular phase, but is extremely liable to relapse. Under the heading must be included the 'acronervotic' eczema, common to women at the menopause or suffering from diseases of the pelvic organs, in which the hands are chiefly involved and co-existent tropho-neurotic changes in the nails (discolouration, pitting, splitting, hypertrophy) are of very frequent occurrence. In nervous eczema itching is usually extreme, and the disease is very capricious in response to treatment.

Seborrhœic Eczema is one of the commonest forms of the disease and is generally considered to be the result of eczema attacking a skin damaged or rendered vulnerable by pre-existent seborrhœa, which most writers regard as a purely secretory disorder, but which Unna—to whom we are indebted for the modern conception of seborrhœic eczema—considers as an inflammatory change and as the first step in the eczematous process. The disease usually begins on the scalp, which is the seat of scurf or 'pityriasis.' The hair is dry and often falls off in considerable quantity. Such a condition may become eczematous and spread at the margin, either as a crusty, scaly, or moist lesion, and thus involve the forehead, eyebrows, cheeks, ears, neck, præsternal regions, back, axillæ, bends of the elbows, hands, groins, genital regions, popliteal spaces, and interdigital regions of the toes. This is often termed the descending type of seborrhœic eczema. There is a tendency for the exudative forms to develop in the flexures, and other warm and moist parts; for ringed, scaly, or crusted forms, often resembling psoriasis, to develop on the outer aspects of the limbs and other dry parts. The scale or crust is usually of distinctly yellowish colour from admixture with fat. Centrifugal eczema, generally considered as seborrhœic in origin, often spreads over the trunk and limbs from eczema of the anus, perinæum, or scrotum. And frequently acute eczema starts from an old-standing chronic patch on the leg and spreads upwards (ascending eczema). It is in the seborrhœic forms of eczema that the morbid influence exerted by micro-organisms is most probable, but the fact has by no means been scientifically established. Itching is, as a rule, comparatively slight, as compared at least with nervous eczema, and the prognosis is much more favourable, as treatment is much more uniformly successful.

Sweat- or Flexural Eczema is closely allied to the foregoing type. It starts in any of the great flexures (axillæ, elbows, popliteal spaces, groins) or wherever adjacent folds of skin sodden by sweat rub against one another (pendulous mammæ, or abdominal folds). The changes which take place in the sweat and the fat which it contains are analogous to, if not identical with, those which occur in seborrhœa; so also are its manifestations and prognosis.

Follicular Eczema occurs almost exclusively on the outer sides of the forearms from a little below the elbows down to the wrists or backs of the hands, and on the legs over the calves and perineal regions, its symmetry being often remarkable. It begins as angry-looking, prominent, red papules surrounding hair-follicles, which form in groups, often circular, and extends by the development of similar lesions at the periphery forming gyrate, polycyclical figures (*E. circumscriptum*), the centres of which generally undergo resolution with desquamation and considerable pigmentation. Several such rings or patches usually co-exist, while, as a rule, the disease does not transgress the papulo-vesicular stage and discharge is not abundant; sometimes, however, profuse weeping occurs, resulting in diffuse patches of eczema 'madidans.' Itching is often extreme, and the condition is curiously obstinate to all forms of treatment, but sometimes yields with almost magic rapidity to the simplest emollient applications. It is doubtful whether the condition should be classified as an eczema at all,

its more obvious relationships being with sycosis, which is distinctly a coccogenetic disease. Persistent folliculitis is also a common sequela of otherwise cured eczema.

The HISTOPATHOLOGY of eczema is practically identical in these various forms, and our knowledge of it is chiefly owing to the elaborate researches of Unna. The principal changes take place in the epidermic layers. These are (1) *Parakeratosis*, or irregular cornification, the result of œdema, or excessive moisture, as the upper epidermic cells, instead of drying up into horny, non-nucleated cells with a deposit of kerato-hyalin granules, retain their moisture and therefore adhere together as masses which are thrown off in the form of scales; (2) *Acanthosis*, or multiplication of the prickle-cells of the Malpighian layer, resulting in great thickening, especially of the interpapillary processes, this condition especially characterising papular eczema; (3) *Spongy Metamorphosis* of the rete due to œdema, which after saturating the cells collects in the intervals between them, and, if in sufficient quantity, shows itself clinically as a vesicle. Changes also occur in the papillary layer, dilatation of the vessels being prominent in cases with much erythema, exudation around them in cases with much infiltration, and proliferation of connective-tissue in old-standing indurated eczema. The presence of all these factors in very various degrees confers upon eczema its Protean characters.

DIFFERENTIAL DIAGNOSIS.—Difficulties are most likely to arise in connection with the following diseases:—

Erysipelas of the face simulates acute eczema, but is preceded by rigors, and accompanied by high temperature, rapid pulse, headache, and often albuminuria; the edge of the patch is more sharply demarcated, and its surface more glazed than in eczema.

Syphilis.—The polymorphism of syphilitic eruptions may simulate eczema, but a consideration of all the features of any given case will usually clear up the doubt. The late palmar and plantar syphilide is usually unilateral and confined to the palm or sole; eczema usually attacks both palms or soles, and spreads up the arm or leg. Syphilides between the toes are attended by a peculiar fœtor.

Pustular syphilides of the scalp, beard, or other parts simulate impetiginous eczema, but are more circumscribed, often offensive, and leave scars. The rather rare minute lichenoid, follicular syphilide, often corymbose in arrangement, may be mistaken for follicular eczema in an early stage. The absence of itching is a striking characteristic of almost all syphilitic eruptions.

Scabies, especially when over-treated by sulphur or other irritating applications, may easily be mistaken for eczema. A careful study of the distribution of the disease, and examination with a lens of its seats of predilection (interdigital clefts, wrists, axillæ, mammæ, lower abdomen, penis, nates), will seldom fail to discover characteristic circular lesions; and should an acarus or its ova be obtained from a burrow, the microscope will clench the diagnosis.

Psoriasis in seborrhœic subjects very closely mimics some forms of dry seborrhœic eczema, so that Unna, reviving an old idea of Erasmus Wilson's, considers the two conditions as identical. Dry eczema and psoriasis involving the scalp alone are indeed sometimes indistinguishable. In a typical

psoriasis the lesions are more sharply defined, have a more raised margin, more abundant silvery scale, and a greater tendency to the formation of rings than those of eczema, while the localisation of psoriasis on the tips of the elbows and just below the knee-caps, if present, is very characteristic. Eczema has a general predilection for the flexor, psoriasis for the extensor surfaces of the limbs, and the lesions of psoriasis seldom itch so violently as those of eczema.

Sycosis of the beard and upper lip is a pustular folliculitis, a deep process resulting in the formation of papules and nodules, involving primarily the hair-follicles, and strictly confined to these hairy parts. Eczema of the same regions is a superficial process and is not confined to hairy parts. In sycosis the hairs can be easily detached by forceps, in eczema they are normally adherent. The two conditions, however, merge into one another.

Ringworm of the scalp, especially when diffuse and of old standing, is often mistaken for dry seborrheic eczema, with which it is often complicated. The amount of baldness produced by it, the presence of stumpy hairs, and the discovery of the fungus by microscopical examination will always settle the point.

Lupus erythematosus, in an early stage, if irregular in distribution, and especially if accompanied by marked seborrheic disorder, may require considerable care for its differentiation. The infiltration of the derma giving rise to some brawny hardness, and the patulous sebaceous ducts over the patch, generally serve, together with the general features of the case, to establish a firm diagnosis.

The lesions of *Impetigo* are pustular *ab initio*; if due to *Pediculi capitis*, the nits of which can always be found, they are generally most abundant in the occipital region; the disease always yields readily to properly directed local treatment.

Rosacea, complicated as it so often is by seborrhoea, is often misclassified eczema. Careful enquiry as to the history and general symptoms will almost always clear up the nature of the case.

Lichen planus is often mistaken for eczema, and *vice versa*. Its papules, which always persist as such, are, however, flat or umbilicated, shiny, grouped in lines, circles or irregular patches, and, if confluent into larger plaques, are of a characteristic purplish-brown tint. They are very common about the wrists, neck, and knees; and the mucous membrane of the mouth is often involved. Severe nervous disturbance frequently accompanies the disorder.

Prickly heat may result in a condition closely simulating eczema. Its sudden onset accompanied by excessive sweating, its extensive distribution, the bright red colour of its initial papulo-vesicular lesions, and the circumstances under which it arises all serve to distinguish it from an eczema.

Among the rarer diseases which may simulate eczema are:—

Mycosis fungoides in its early premycotic stage, *pityriasis rubra* (or general exfoliative dermatitis), *prurigo*, and the severer forms of chronic *urticaria*, *dermatitis herpetiformis*, *pemphigus foliaceus*, and *dermatitis repens*; for the points of differentiation reference must be made to the articles under these headings.

PROGNOSIS.—Eczema is generally a curable disease, but it is often obstinate and tends to recur. Generally speaking, cases in which the general

health is impaired or the nervous element predominates are of graver prognosis than those of seborrheic type. Fatal cases of eczema are extremely rare, but the supervention of acute generalised exfoliative dermatitis is an event the possibility of which should never be overlooked; it not infrequently is the result of over-stimulating or meddlesome treatment.

TREATMENT.—This should be both general and local, to each of which equal importance must be attached. Predisposed persons ought to adopt certain general hygienic precautions according to the determining cause of their eczema.

Diet.—All articles must be avoided which are difficult of digestion and keep up itching and scratching. Curries, pickles, condiments, highly salted meats, rich sauces, and sweets, pastry and cheese, all clearly come under this category, and so, in many cases, does coffee. Intercurrent dyspepsia must always be treated according to the form in which it presents itself. In children oatmeal and other gritty food-substances as well as potatoes are often specially deleterious; in them the dietetic rules should be on the same lines as for chronic urticaria.

Drink.—Alcohol, except in very limited quantities, undoubtedly does harm by causing congestion of the skin, in almost all cases of eczema, especially in acute and irritable gouty-neurotic cases. It is, however, of service in the pustular eczema of children, to whom fifteen to twenty drops of brandy in milk may be given two or three times a day; and a little sound wine may be indicated in the eczema of old or debilitated persons.

Washing and Baths.—In eczema of seborrheic type, especially when chronic, comparatively free use of water, and even of soap of good quality is not only permissible but obligatory for the removal of decomposing matters from the surface of the skin. The water used must not be 'hard'; thus, distilled or rain-water is always to be preferred. After using soap the part must be carefully dried and lubricated to counteract the excessive dryness produced by it. Soap ought never to be used to acute or weeping eczemas, as it macerates and carries away delicate young epidermis. When the cleansing of such surfaces is necessary—and it should be carried out as seldom as possible—thin gruel, gelatine, starch, or bran and water, weak carbolised oil, or decoction of marsh-mallow may be employed; while the yolk of egg is in common use for the scalp. In many cases the skin may be kept clean by dusting with starch or fuller's earth, but if there is discharge these substances are apt to cake.

The almost indiscriminate treatment of eczema by mineral baths has had its day. The baths themselves have usually anything but a beneficial effect upon the disease, and any improvement which may ensue upon residence at a spa must be attributed to the general improvement in health consequent upon the temperate and healthy mode of life there enjoined and usually followed. An exception must be made in favour of mild sulphur-baths (e.g. Harrogate, Strathpeffer, Schinznach, Luchon, Aix-la-Chapelle, &c.), which sometimes are of benefit in dry pruriginous eczema; their action, however, requires careful supervision. Sea-bathing is usually contra-indicated, but there are exceptions to the rule, especially in cases of neurotic origin.

Climate.—As a rule east winds which are cold

and heavily laden with brine do harm to eczema, but in tubercular and some neurotic cases residence at the seaside, where they naturally prevail, seems to be of real service. Warm climates generally aggravate moist eczemas, especially those of sweat-origin, but may benefit scaly cases.

Clothing should be warm and light, and woollen garments should be separated from the skin by silk or cotton underwear.

TREATMENT OF ACUTE ECZEMA.—In all very severe cases, in order as far as possible to obtain physiological rest to the skin, the patient must be confined to bed, in a room at an equable temperature of 60° to 65° F. The bedclothes should be light, and a surgical cradle, which prevents contact of them with the skin, is advantageous. The diet should be on the same lines as for acute febrile affections, and should consist of milk, beef-tea, and other easily assimilable liquid foods. Free purgation is universally considered to be beneficial, due regard being paid to the strength of the patient, and for this purpose the sulphates of magnesium and sodium are the drugs usually employed. A mixture containing acetate of ammonia and nitrous ether is generally useful, and to this five to ten drops of antimonial wine may be added if there is high arterial tension. When there is distinct evidence of gout, alkalis, iodide of potassium in small doses, and colchicum may be prescribed. Diuretics such as the acetate and citrate of potassium are often administered, but their utility is doubtful. *Locally*, all greasy substances and ointments, especially of a stimulating nature, must be avoided. Lotions must be used, and when burning and itching are severe and discharge copious, nothing is more beneficial than the *Liquor Plumbi Subacetatis Dilutus*, B.P., which may in some cases require to be diluted; it must be applied on soft linen rags and these kept constantly moist. During the night the dressing may be covered with gutta-percha tissue to prevent it getting dry. A solution of boric acid (gr. 5 to gr. 20 ad 3 j) is also much employed, but occasionally provokes much irritation. For localised patches 'black wash,' diluted if necessary, is often most useful. It is difficult to apply water-dressings to the head and face, and under these circumstances a powder consisting of fuller's earth, starch, and oxide of zinc is a useful application; a little boric or salicylic acid (2 per cent.) may be added.

Should the dressings stick to the eczematous surface or powders irritate, the *Linimentum Calcis* is a good substitute, especially if made with almond oil; it should be either applied on lint or simply painted on several times daily and covered with 'butter-cloth.' As inflammation subsides powders may be added to it, e.g. oxide of zinc, calamine, or carbonate of calcium or magnesium. In similar circumstances a very useful lotion is made by adding a drachm of *Liquor Plumbi Subacetatis Fortis* to an ounce of milk; the mixture ought to be made fresh every time the remedy is applied.

If scales and crusts accumulate they must be removed; this is best done by covering the part with strips of linen, rather than lint, soaked in oil or a weak solution of bicarbonate of sodium; or by the application of cold boric starch-poultices, which are made as follows: a teaspoonful of boric acid is added to two ounces of cold-water starch, and mixed with a little water; a pint of boiling water is then stirred in, and the jelly which results is spread on cloth in a layer about half an inch thick; when cool, a piece of

muslin is laid over it, and it is applied to the part.

TREATMENT OF SUB-ACUTE, RECURRENT, AND CHRONIC ECZEMA.—If the patient is suffering from gouty symptoms, dyspepsia, or constipation, treatment should be adapted to meet these conditions, the eczema being dealt with at the same time by local means. Freedom from worry or anxiety obtained by travelling abroad, residence at a health-resort, or change of occupation or interest is often of the greatest service.

The use of arsenic has of late years been to a large extent abandoned, and with justice, for it undoubtedly tends to aggravate subacute eczema and to favour relapses in many instances. In very dry, scaly cases, approaching psoriasis in character, it is, however, occasionally of value, especially if the patient is young and in good health. In children between the ages of two and five it is also serviceable. The routine practice of treating eczema in children with iron and cod-liver oil generally ends in failure, especially when the eczema is of an irritable kind. In the pustular form of eczema, however, iron is useful, and then arsenic may be combined with the *Vinum Ferri* or some other preparation of iron.

In all forms of eczema the greatest attention should be paid to the action of the bowels, and even when there is no constipation some increase in their natural action is beneficial. The kind of purgative to be used will depend on the age of the patient and other circumstances.

The local treatment of chronic eczema has for its objects the following indications: to soothe inflammation and diminish hyperemia, to stimulate chronic patches, to soak up discharge, to relieve itching, to supply deficient fat, to prevent contamination by micro-organisms, and to destroy these when their presence and morbidic action are suspected.

Generally speaking, erythematous eczema is best treated by lotions, sometimes containing bland powders (calamine, zinc oxide, &c.); by dusting powders (starch, oxide of zinc, carbonate of magnesium, prepared chalk, Emol-kelet, talc, &c.); or by protective varnishes such as Pick's *Linimentum Exsicicans* or Unna's *gelanthum*. The former is composed of 5 per cent. of glycerin and an equal proportion of tragacanth in 100 parts of water; the water and glycerin being gradually added to the tragacanth in a mortar. This makes a clear jelly which, if spread on the skin, forms a fine film, giving a cooling sensation. The addition of 1 per cent. of oil of Cade is often useful. *Gelanthum* is a similar preparation, but also contains glycerin and thymol. Greasy applications frequently disagree with erythematous eczema.

Popular eczema is usually obstinate to treatment. Lead-lotions and 'black wash' are often very serviceable, and a little tar may often be added to either with advantage, preferably the *Liquor Carbonis Picis* (B.P.), or the *Liquor Carbonis Detergens*. Ointments must be used with caution, such as those of zinc oxide, or oleate, boric-acid ointment, or the *Unguentum diachyli* made by mixing *Emplastrum Plumbi* (B.P.) with equal parts of olive oil at a gentle heat, to which oil of bergamot or lavender may be added to prevent decomposition.

Ointments may be rubbed in or applied on strips of cloth fixed by a light bandage and covered by gutta-percha tissue or similar impervious material.

Salve-muslins and plaster-muslins are preparations made by Beiersdorff of Hamburg, containing various

medicaments, and are practically very handy devices for the continuous application of ointments.

Lassar's paste is an excellent preparation, widely used, which consists of a mixture in equal parts of oxide of zinc, starch-powder, lanolin, and vaseline, to which 2 per cent. of salicylic acid may often be added with advantage. Pastes are generally preferable to ointments, and are made by adding starch, chalk, carbonate of magnesium, kaolin, terra silicea, or other absorbent powders, to ordinary ointments with a fatty basis. Rubbed over the skin they form a protective envelope, which may be covered with powder, waxed paper, or cotton-wool and a bandage. Cold creams are especially pleasant and cooling remedies, owing to their power of taking up water on the one hand and giving it off on the other. An excellent formula is \mathcal{R} Adipis Lane Anhydric. \mathfrak{z} j, Vaselini \mathfrak{z} ij, Aq. Calcis et Aq. Rosæ aa \mathfrak{z} iij. The Unguentum Aquæ Rosæ (B.P.) is also a capital preparation to which sulphur, salicylic acid, lead-salts, or tars may be added. Such creams ought to be applied in a thick layer.

Glyco-gelatine of zinc is the type of an occlusive dressing and may be used in papular eczema, where it frequently relieves itching, or in erythematous eczema, especially of the leg; or even in weeping eczema when the discharge is not abundant. It is composed as follows: \mathcal{R} Zinci oxidi et Gelatini aa \mathfrak{z} jss, Glycerini \mathfrak{z} ij, Aquæ destillatæ \mathfrak{z} iv. The gelatine and water must first be carefully mixed, the zinc oxide and glycerin being afterwards stirred into the mixture in a water-bath. On cooling a solid elastic mass is formed, which must be melted in a glue-pot or similar vessel for use, and applied freely with a stiff brush; the part should be dabbed with a pledget of absorbent wool before the glyco-gelatine quite dries, the result being a pliable coating which exerts a certain amount of compression. In hot weather less glycerin and more gelatine should be used. Sulphur and ichthyol may often be combined with the application with good results, but when a tarry preparation seems indicated, it is better to paint it on the skin first and to cover when dry with the glyco-gelatine.

Vesicular eczema usually calls for treatment by lotions and powders such as have been already described. It is of the greatest importance to remove accumulations of crusts or scabs, which can be best done by applying strips of lint soaked in oil, or by boric starch-poultices. As discharge lessens, pastes may be cautiously used and subsequently ointments. In some localised persistently discharging patches, occasional painting with a solution of picric acid (1 in 100) or of nitrate of silver (gr. v ad \mathfrak{z} j) is very efficacious.

Pustular eczema generally indicates the use of mild antiseptics, such as boric-acid lotion or weak ammoniated-mercury ointment (gr. v ad \mathfrak{z} j) constantly applied.

Chronic scaly eczema is best treated by ointments, which should be well rubbed in, the part being subsequently covered by soft linen thickly 'buttered' over with the remedy. Zinc oxide or boric acid ointment may be the basis with which mild mercurials, salicylic acid, or tar may be cautiously incorporated. In very obstinate infiltrated cases, a weak chrysarobin-ointment (gr. x ad \mathfrak{z} j) is often useful; and in others a chronic patch may be converted with advantage into an acute one by swabbing with caustic-potash solution (1-6) and subsequently healed by lead or other soothing lotions and ointments.

Local forms of eczema and their treatment can only be briefly alluded to.

Eczeema of the scalp is almost always seborrhœic. If discharge is abundant the hair should be cut quite short and scabs and crusts carefully removed. When there is much active inflammation lotions must be used until it has subsided: afterwards ointments may be applied. In very chronic scaly eczema of the scalp in adults, shampooing weekly, or more frequently, with Hebra's Spiritus Saponis Alkalinus is valuable. In young children the use of dusting powders and of zinc-ichthyol or zinc-salicylic salve-muslins may often be recommended, every precaution being taken to prevent scratching.

Eczeema of the face is often of erythematous, senile type. Greasy applications may disagree, and cooling lotions are almost always preferable; of these none is better than the familiar Calamine Lotion, to which a little Liquor Plumbi Subacetatis Fortis or weak solution of tar may be carefully added. When the hairy parts of the face are involved the treatment must be very like that in eczema of the scalp.

Eczema of the lips, angles of the mouth, and immediately surrounding parts is often extremely obstinate, although apparently trivial. It is generally connected with some disturbance of digestion, to which treatment must be mainly directed. If milder local measures fail, extremely good and persistent results sometimes follow the daily application of weak chrysarobin in gelanthum (2-5 per cent. cautiously increased).

Marginal eczema of the eyelids ('*tinca tarsi*') is common in strumous children, and treatment must be directed to the constitutional taint. Crusts must be removed by frequent bathing or the free application of any indifferent salve; the yellow oxide of mercury ointment (gr. iv ad \mathfrak{z} j) is then the most useful application, but obstinate cases may require occasional painting with caustic potash (gr. x ad \mathfrak{z} j) or nitrate of silver (1-2 per cent.).

In the axilla the difficulty of maintaining dressings in position may be overcome by the use of plaster-muslins or salve-muslins cut in narrow strips, or of bulky pads fixed with a bandage. In many instances pastes containing ichthyol (gr. xxx-lx ad \mathfrak{z} j) and resorcin (gr. x-xx ad \mathfrak{z} j) act with extraordinary rapidity and effect.

Eczeema of the scrotum frequently occurs in persons in a poor state of health, and is often accompanied by great swelling, much irritation and pain, and an offensive discharge. In severe cases rest in bed with nursing is essential, and soothing lotions (zinc, calamine, lead) are the most generally useful applications at first. To these very weak tar may be gradually added. Subsequently, perhaps, diachylon ointment is the most valuable remedy, but Lassar's paste with salicylic, boric, or carbolic acid is also of service.

Eczeema of the anus is sometimes a most distressing and inveterate disease. It is often of purely nervous origin, starting as a pruritus, the 'eczema' being entirely the result of the patient's scratching. Local causes must always first be looked for (e.g. hæmorrhoids, fissures, prolapse, and worms) and treated in the usual manner, if discovered. Digestive disturbances are often present and must be corrected. The most important point in local treatment is always to cleanse the part thoroughly, without the use of paper, directly after the bowels have acted.

In many cases water as hot as can be borne,

applied by means of a sponge for two or three minutes, affords great relief; and sometimes the daily use of soap and water is beneficial. The parts must then be carefully dried and an ointment immediately applied. Among the best are the calomel, the diluted red oxide of mercury, and the calamine ointments. Weak tarry or carbolic lotions, and an occasional painting with a solution of nitrate of silver (gr. x ad $\frac{3}{4}$ j) are often useful. Salicylic acid paste (gr. x-xxx ad $\frac{3}{4}$ j) may reduce the brawny infiltration so often present, and for the same purpose painting with a saturated solution of carbolic acid or a 10-per-cent. solution of caustic potash may be resorted to. In extreme cases the patient must be anæsthetised and the parts destroyed superficially with a Paquelin's thermo-cautery. In many cases, remarkably sudden and beneficial results accrue from change of air and scene. The internal administration of phenazone, phenacetin, or similar drugs in considerable doses is sometimes surprisingly efficacious.

Eczema of the palms and soles is often associated with seborrhoeic eczema elsewhere or with local sweat-disorder, and is characterised by hyperkeratosis and painful cracks especially at the lines of flexure. The hardened cuticle may be removed by the constant application of lint soaked in a weak solution of Liquor Potassæ, but the best application is Pick's Salicylic Plaster (5 per cent.) in strips, changed daily; this may be aided by rubbing in soft soap in the intervals of the dressing. When the thickened skin is removed weak salicylic pastes may be used, to which ichthyol is undoubtedly a useful addition; or tarry applications may be employed, of which none is probably better than the Liquor Picis Carbonis diluted according to the patient's idiosyncrasy.

ROBERT LIVEING. J. J. PRINGLE.

ECZEMA MARGINATUM.—See **TINEA TRICHOPHYTINA**; and **ERYTHRASMA**.

EFFUSION (*e*, out; and *fundō*, I pour).—The escape of a fluid from its natural channel or cavity into the substance of organs or the cellular tissue, or from free surfaces. As examples may be mentioned dropsy in its various forms, and effusions resulting from inflammation.

EGYPT, UPPER.—A very dry, tonic, winter climate. Mean winter temperature, 62°F. Season, October to March. See **CLIMATE**, Treatment of Disease by.

ELECTRIC CHOREA.—SYNON.: Dubini's Disease.—See **CHOREA**.

ELECTRICITY.—See **OCCUPATION-DISEASES**.

ELECTRICITY IN MEDICINE.—The purposes for which electricity is employed in medicine are various. It is used as a stimulant to excite muscular and nervous tissue which is the seat of paralysis or pain, and as a general systemic stimulant; its chemical or electrolytic action may be employed for causing destruction of tissue; and its thermal effects are used for hot-air baths, for heating appliances to take the place of poultices and hot-water bottles and in surgery for heating cauteries. The incandescent electric light has been adapted for the exploration of the bladder and of the antrum of Highmore, as well as of the throat and

other cavities of the body. Electricity is also used in medical practice for the production of ozone, and in the form of the arc-lamp for treating lupus and other skin-diseases, and for the general effects of bright light in the treatment of various general disorders. The use of the X-Rays or Röntgen Rays and the whole subject of skiagraphy will be dealt with in another article (see **RÖNTGEN RAYS**). Electricity is generally applied in the form of a current passed through the body or through a part of it, and this may be called 'current electricity' or electro-dynamic treatment to distinguish it from the uses of electricity in electrostatic treatment, where the patient is insulated and charged by connecting him to an electrostatic machine or other source of high-potential electricity. The technique of electrostatic treatment consists in the charging and discharging of the insulated patient. By variations in the polarity of the charging electromotive force and in the mode and rate of the charging and discharging, well-marked physiological effects can be produced, and these can be valuably employed in the treatment of some morbid conditions. As very high potentials are needed for these effects they are usually obtained from 'static machines' of large dimensions, the Wimshurst influence-machine being the best. The high-frequency coils of Tesla and D'Arsonval have also been applied of late years to the treatment of disease, and their mode of action is also mainly 'electrostatic' in its nature. Electrostatic treatment, formerly known under the names of Franklinic electricity, and frictional or statical electricity, was once much in vogue, and seems destined to take its place again as a useful therapeutic agent. Its chief field of usefulness is in functional nervous disorders, and in the relief of neuralgic and myalgic pains. It also seems to exercise a good effect upon the general health, particularly in old people.

Current electricity is applied in two chief forms, namely as a continuous current of steady flow, and as a rapidly varying or interrupted current like that of the induction-coil, or the periodically alternating current of certain dynamo-machines. In either case the physiological effect is due to the continual rapid variation of the current, and there are fundamental differences between the response of nerve and muscle to varying and to steady currents. On this account a battery for testing or treatment must include means of applying both, for sometimes one is needed and sometimes the other. The well-known rotating magneto-induction apparatus need only be mentioned to be condemned; it gives a rude form of alternating current, and is out of date, although improved magneto-machines are sometimes useful for providing alternating currents in places where suitable electric-light mains are not available.

Medical batteries for purposes of testing and treatment consist of two parts, one an induction-coil, and the other made up of a number of small cells coupled together in series. The chief requirements of a cell for medical purposes are that it shall need the minimum of attention, and shall not deteriorate when lying by unused. The form of 'Leclanché' cell, known as the 'dry cell,' is the best, and 25 or 30 is a convenient number. To facilitate the handling of the battery one also needs some means of bringing into the circuit any number of the cells at will, so that the magnitude of the current may be regulated. This is effected by a

moving pointer and a circle of studs connected to the cells, forming a contrivance known as a current-collector. A means of opening or closing the circuit at will, and of reversing the direction of the current without altering the position of the electrodes on the patient's body, is also needed. This is effected by means of a simple contrivance known as a commutator. A proper medical battery fitted with these accessories can be purchased for a sum ranging from ten pounds upwards, according to style and finish. For purposes of exact diagnosis, for physiological experiment, and for electrolytic operations a galvanometer is quite indispensable, as by its use alone are we able to say what strength of current is operating, because the resistance of the body varies considerably in the same and in different individuals, depending as it does very largely upon the degree of moisture present in the skin and the size of the electrodes employed, so that to say that a current from 'so many cells' has been used is not a good guide. The galvanometers used in medical practice measure the magnitude of the currents employed, and are calibrated to register in milliamperes, so that in recording we say that 'the muscle contracted with a current of so many milliamperes.'

When a battery is required for heating the wire of a cautery, or for lighting up an incandescent lamp of small size, a separate apparatus of a few elements of larger size is to be used. Four cells of a bichromate battery, or better still a storage-battery of four secondary cells, is a suitable outfit. These may be bought ready fitted with a regulating resistance to prevent over-heating and destruction of the lamp or cautery. Accumulators or storage-batteries are much the best when a means of re-charging them is available.

The essential and distinctive differences of the two forms of current—current of the cells, and current of the induction-coil—will now be described. The current of the cells is (1) *continuously evolved*; hence it is spoken of frequently as the 'continuous current'; it is also called the 'galvanic current' or the 'constant current.' (2) *It flows always in the same direction*, i.e. from the positive pole, which is in connection with the carbon-pole of the cell, through the outer circuit to the negative pole which is in connection with the zinc. The action of the two poles is somewhat different, and the direction of the current in the body, whether towards the nerve-centres or towards the periphery, has been found to exert an important effect upon the physiological and therapeutical results. (3) It has well-marked *chemical effects* at the poles (electrolysis), and in this way it may cause redness, inflammation, and even sloughing of the skin when the applications are too strong or too prolonged. It is therefore necessary to change the position of the electrodes on the body during long applications. These caustic effects are due to the electrical decomposition of the saline constituents of the body, acids being evolved at the positive pole and alkalis at the negative. The positive pole is called the *anode*, and the negative pole the *cathode*, and these terms are frequently employed.

The induction-coil current may be described as a rapid succession of separate impulses or momentary currents whose direction is constantly changing, so that, in using it, it is less necessary to distinguish between the poles. Its chemical, thermal, and electrolytic effects are not usually perceptible. It usually has greater electromotive force than the

battery-current, and therefore it overcomes the resistance of the body with greater ease. It causes the contraction of healthy muscle more readily than the battery-current, and the contraction which it produces takes the form of a tetanus, lasting as long as the current is applied, whereas the battery-current of normal strength causes a muscular contraction only at the moment of making or breaking the current and not during its continuance, and the stimulating effect of its two poles is different, as may be demonstrated by a simple experiment. If an electrode, provided with a key for opening and closing the circuit, be placed over a motor nerve-trunk or a muscle, while the other is on a remote part, we are able by means of the commutator and key to study the action of either pole on the nerves and muscles during the making and breaking of the current. With weak currents it is found that a contraction is produced only when the active electrode is negative (cathode), and only on closing the circuit. This is called the Cathodal Closure Contraction (C. C. C.). If the strength of the current be slightly increased, we get a contraction also when the active electrode is positive (anode) and the circuit is closed. This is the Anodal Closure Contraction (A. C. C.). The Opening Contractions (A. O. C. and C. O. C.) may also be seen if the strength of the currents is still further augmented.

The battery-current affects the brain and the nerves of special sense more readily than the induction-coil current. If it be applied in the neighbourhood of the eyes, flashes of light are seen, and blindness has resulted in one case from the incautious application of strong currents to the face. The gustatory nerve is affected in a similar way, and a metallic taste is perceived when the electrodes are placed on the cheek. If the electrodes are applied to the ears, noises are produced, and it is said that stimulation of the olfactory nerve will give rise to a peculiar smell.

MODES OF APPLICATION.—To apply currents of electricity we need to have conducting wires and electrodes attached to the battery. The best conductors are made of flexible stranded wires covered with silk or cotton insulation; they should have metal pieces or tags at both ends, so as to be readily connected to all kinds of batteries and electrodes. Electrodes vary much in design, and two chief forms are needed. One is a plate of flexible metal, such as tin or pewter, enclosed in a wash-leather sheath and applied to the back or chest, or under the hips. It is termed the 'indifferent' electrode, and serves mainly to complete the circuit through the patient. The other has the form of a disc of metal and is provided with a wooden handle. This is the 'active' electrode, by means of which the current is brought to the affected part. The active electrode, like the indifferent, must be covered with wash-leather to prevent injury to the skin. The use of sponges in sponge-holders as electrodes is to be condemned. The indifferent electrode should be roughly about ten square inches in area, while the active electrode should be of different sizes, ranging from one to four inches in diameter. Special electrodes of almost endless design have been made for reaching particular organs and regions, as the eye, ear, larynx, bladder, rectum, and uterus. A wire brush-electrode is sometimes useful for counter-irritant applications to the skin. The hand of the operator, who allows the current to pass through his own body to that of the patient, has also been used as an electrode.

If we wish to produce cutaneous effects chiefly, the skin and the active electrode may be nearly dry; but if we wish to influence the deeper structures, we must reduce the resistance of the epidermis by thoroughly moistening it and the electrodes with hot water. Sometimes it is advantageous to use a bath of water as the medium for conveying the current to and from the patient, and electrical applications in an arm-bath, or a foot-bath, or even in a large general bath for the whole body, are oftentimes very useful. In this method of applying currents the bath must be made of some non-conducting material, and the electrodes take the form of metal plates fixed at the ends of the bath.

There are two methods of applying electricity, known as general electrification and localised electrification. By the former method we pass the current through the entire body or a great part of it. Although its utility has only been fully appreciated during the last few years, it offers a therapeutic means which is of great value in diseases characterised by failing or defective nutrition, as, for instance, anæmia, debility, rheumatism, gout, general neuritis, &c. The electric bath, with alternating currents, is the simplest and best mode of applying general electrification. By the latter method, which we owe to Duchenne, we seek to influence special nerves, muscles, or organs, and to limit the action of the current strictly to these parts. If we wish to influence a muscle, we may do so by applying the electrode directly over the fibres of a muscle, or else we may stimulate the nerve supplying it, and so, indirectly, cause the muscle to contract. In testing muscles the active electrode is usually applied to the skin over the muscle itself, and there is a favourable point called the 'motor point' over all superficial muscles at which the stimulus can be applied most effectively. Neither of these methods is to be exclusively adhered to. Certain deep-lying muscles, such as the diaphragm and the supinator brevis, are capable of indirect stimulation only through their motor nerves. It will be found also that, in certain paralytic conditions, the muscles will not respond to stimulation applied through the nerves, though still contracting to the direct application of the current. In therapeutic applications of electrical currents it is often advisable to combine the electricity with a rhythmical exercise of the affected muscles, or the current itself may be caused to vary in a slow and rhythmic manner by some form of mechanical apparatus, which turns the current gradually off and on about once in two seconds.

DIAGNOSTIC AND THERAPEUTIC USES.—1. Diagnostic Uses of Electricity.—Electricity is of service for purposes of diagnosis, since by its means we are enabled to distinguish between paralysis due to (a) a lesion situated above the nucleus of origin of a muscle, and (b) paralysis from lesions in or below the nucleus of origin of the muscles tested. In paralysis due to lesions of the first class, we find (a) that reflex stimulation of the muscles is possible; (b) that the muscles undergo but little wasting; and (c) that the electrical reactions of the muscles are unchanged in character. Whenever the paralysed muscles are cut off from communication with their nerve-centres or nuclei of origin in the cord or brain, or when they arise from centres whose physiological activity has been destroyed by disease, we find (a) that reflex stimulation of the paralysed muscles is no longer possible; (b) that the paralysed muscles waste with remarkable rapidity; and (c) that the

electrical reaction of the muscles is profoundly altered. The change which takes place is spoken of as the 'reaction of degeneration,' and consists in a total loss of the contraction to the induction-coil and a change in the contraction to the battery-current, which becomes slow and sluggish instead of being a rapid twitch. The slow contraction further can be produced only by direct applications of the testing electrode to the muscle itself, for indirect stimulation through the motor nerve-trunk produces no contraction whatever. It sometimes happens further that a change takes place in the irritability of the muscles, so that the Anodal Closure Contraction (A. C. C.) soon becomes very marked, and equals or even surpasses in force the Cathodal Closure Contraction (C. C. C.). The Reaction of Degeneration is found (1) in cases of myelitis in the muscles whose nerves arise from the damaged part of the cord; (2) in so-called spinal paralysis, polio-myelitis, both of infants and adults; (3) in traumatic paralysis due to injury of the nerve-trunks; (4) in neuritis from disease, when the neuritis is severe enough to impair seriously the functions of the nerves.

Its occurrence is probably determined by the degeneration of the motor nerve or of the ganglion-cells of the anterior cornu from which the nerve arises.

In hemiplegia the irritability of the muscles remains unchanged, or there may be slight 'quantitative' increase of irritability. The determination of variations in the degree (increase or decrease) of irritability of nerves and muscles, apart from the reaction of degeneration, may often be made in various diseases, but no facts of prime diagnostic importance can be obtained from the observation of simple increase or decrease of irritability.

2. Therapeutic Uses of Electricity.—(a) *In Paralysis.*—The treatment of paralysis by means of electricity must be conducted rationally, and must not be applied indiscriminately to unsuitable cases. Progress in medical electricity has advanced along two lines, one based upon experimental physiology and the application of the facts observed to various morbid states, and the other based upon direct observation and experiment upon the sick. From the first method we can apply the observed fact that electricity is a stimulus to living tissues, by inquiring how stimulation may be made use of in disease. The fact that electricity is a stimulus is easy to perceive in the case of sensory and motor nerve and muscle, and it is not difficult to prove that it also acts as a stimulus to all living tissues, and the employment of electricity in disease is very largely an application of processes of stimulation.

Beard and Rockwell in the United States, and Debedat in France, have shown experimentally that in young animals the rate of growth can be accelerated by suitable electrification. The former daily treated two young puppies, and found that they grew faster than those of the same litter which were not so treated. Debedat in the same way proved an increase of weight in certain muscles of rabbits as compared with other untreated muscles in the same animals; the latter thus showed a local effect from localised applications of electricity, the former a general effect from general applications; and this division into general and local treatment is a useful one. Thus for general diseases one gives a general treatment, and for local diseases a local application. D'Arsonval by calorimetric observations and chemical analyses of the expired air has

established the fact that electrical currents increase the activity of the tissue-exchanges of the body. Capriati recently has shown by careful dynamometric researches that the application of electricity to the central nervous system in the form of a continuous current along the spine does undoubtedly increase the muscular power, and that applications to the muscles of a limb have the same effect. General electrification applied to cases of simple failure of nutrition, such as debility of any kind, produces a rapid improvement in the condition of the patient. In rickets, which is a comparatively simple form of defective nutrition, electrical applications have been shown by many observers to have a very high value. The more complex forms of defective nutrition, such as rheumatism, gout, and diabetes, are not yet completely controlled by electricity, but indications are not wanting that in time good results may be expected. Already electricity is doing something for these conditions. In the field of paralytic conditions there are certain strict limitations to the utility of electrical treatment, and it is here that electricity has lost credit quite undeservedly. When paralysis is due to an obstructive or destructive process in the brain or the spinal cord, or when it is due to progressive degeneration in the quality of those parts, then electricity is powerless to help. So, too, when paralysis is due to a breach of continuity in a nerve-trunk, electricity can do nothing until the surgeon has intervened and re-united the severed ends. In the latter class of cases electricity comes in quite properly afterwards to promote the recovery of functions which had fallen into abeyance; in the former the surgeon cannot help, neither can electricity. Therefore it is very important to insist that in paralysis there are cases suitable for electricity and cases which are unsuitable. Happily the number of those which are greatly benefited by electricity is a considerable one. To treat such diseases as spinal sclerosis, tabes dorsalis, progressive muscular atrophy, and so forth, is pure waste of time in the present position of our knowledge of electro-therapeutics. Treatment by electricity must be jealously reserved for such conditions as have been found to derive benefit from it: for example, infantile paralysis (so far as there may remain some living ganglion-cells in the nucleus of the muscle) and all forms of peripheral neuritis, whether due to injury or disease, whether the symptoms be pain or paralysis, for these diseases respond admirably to electricity when properly applied.

Again the large field of general electrification for all kinds of nutritive failure and disorder needs much more study, and has been greatly neglected through the prominence given to localised electrification since the time of Duchenne. The treatment of joint-affections and of skin-diseases presents also fields for study which are hardly yet opened up, largely through the mistaken idea that electricity is useful only in nervous disorders: an idea which has led to an enormous waste of time and energy upon such unpromising subjects as degenerative diseases of the brain and spinal cord. In the localised application of the electric current to paralysed muscles we should bear in mind the following rules:

(1) To employ induction-coil currents when simple stimulation is desired; if the muscles act readily to coil-currents, then these will suffice. If they do not, and the reaction of degeneration be present, it does not follow that only battery-currents are to be used. The production of a visible contraction is not a

measure of the useful effect, and the coil will be of service when it produces no contraction at all. In these cases it is to be supplemented by battery-currents made and broken by hand. The active electrode is to be moved over the moistened surface of the affected part.

(2) To employ the weakest currents which will cause muscular contraction, in order to avoid the risk of exhausting a muscle. Each muscle should be taken in turn and be made to contract several times in succession, and when the whole of the paralysed muscles have been done *seriatim* the process may be repeated. An application of this kind for ten minutes daily or every other day is usually sufficient.

(3) If the paralysis to the will remain absolute, and if the contractility of the muscles to coil-currents be perfect, we do no good by long-continued electrical treatment. This condition is often met with in hemiplegia. The patient is absolutely helpless on one side, although the paralysed muscles are in no degree wasted, and their irritability remains normal. Treatment for a few weeks is useful in such cases, and acts by the effect upon the brain of the peripheral stimulus, promoting the return of power in the parts which though not destroyed are functionally impaired.

(4) If the irritability to both forms of current has completely disappeared, we are not justified in persevering too long, or in holding out delusive hopes to the patient. Total loss of all reactions in a muscle is not very commonly met with except in old-standing cases of muscular atrophy, infantile paralysis, and severe injury (destruction) of nerve-trunks. (For the treatment of special forms of paralysis by electricity, the reader is referred to the appropriate sections of this work.)

(b) *In painful affections.*—The power of electricity to relieve pain is very great. The effect is often temporary at first, but after a few repetitions of the treatment the relief becomes permanent. In the treatment of painful affections electricity may act by counter-irritating effects simply, as is perhaps the case when the induction-coil currents are used, but this is not the only way nor is it the chief way in which pain can be relieved by electricity. Often-times electricity will give relief to neuralgic pains when treatment by drugs has failed, and in such cases we may suppose that it acts by a direct action on the nerves themselves. Both the electrostatic methods and those by electric currents are employed for the relief of pain, but the battery-current will be found the most generally useful. Some writers insist that the anode (positive pole) shall be applied to the painful spot, but as a rule there is but little difference between the actions of the two poles. The effects of battery-currents deserve to be tried in every case of troublesome neuralgia, and in cases of neuritis with pain, as for example in sciatica, in circumflex-neuralgia, and in brachial neuritis. Myalgic pains may be treated in the same way, and for these electrostatic treatment with sparks applied to the affected regions often acts well and rapidly.

In the treatment of spasmodic diseases, electricity is of limited utility.

In addition to the treatment of diseases of the nervous system electricity has been found useful in other disorders.

It has been employed with success in various skin-diseases. Rheumatic and gouty joints are benefited by the battery-current applied to the affected parts

either by means of pad-electrodes, or through the medium of a local electric bath, and the same treatment is of considerable value for the freeing of joints which have become stiffened by fibrous tissue as a result of past inflammation in or around them. Many of these phenomena depend probably upon vaso-motor changes produced by the battery-current, and it is probable that the effect of electricity in the relief of pain is to a certain extent dependent upon vaso-motor stimulation. There is scarcely a disease, from epilepsy to chilblains, in which it has not been alleged that electricity has been of use, but when judged by the test of practical utility electricity must be limited in its employment to those disorders in which its curative effects are at once more certain and more prompt than are those of other less troublesome modes of treatment. In obstetric medicine the induction-coil has been usefully employed for the arrest of *post-partum* hæmorrhage, and it has been largely practised for the treatment of uterine fibroids. Though useful in that condition it is not able to compete with more direct surgical methods, and has therefore not established itself as a recognised mode of treatment for fibro-myoma, at least in this country.

The chemical and thermal effects of galvanism are largely employed both in surgery and in medicine. Its thermal effect has been used for the heating of cauteries; and cauteries so heated have very obvious advantages over all other forms. The chemical effect of the negative pole has been used as a caustic for the destruction of tissues and tumours, particularly for nevi and in cosmetic surgery, for removing moles, superfluous hairs, and other small blemishes.

The general adoption of electric light in private houses has led to a considerable extension of the use of electricity by medical men. Most surgeons now use their electric-light mains for cautery and lighting instruments. Dental drills, saws &c. also are driven by electric motors operated in the same way. The direct application of the current of the main to the treatment of patients has given practitioners a new form of alternate current, called the sinusoidal current, which has decided advantages over the currents of the induction-coil, though not greatly differing from them in character. It is hardly necessary to add that before attempting to treat patients by applying to them the currents of electric lighting circuits there are certain elementary precautions to be taken in the way of regulating and reducing their strength from the 100 or 200 volts of the lighting supply to the much lower pressures which are needed in electro-therapeutics.

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ELEPHANTIASIS ARABUM (ἐλέφας, an elephant).—See FILARIASIS.

ELSTER, in Saxony.—Alkaline sulphated and chalybeate waters. See MINERAL WATERS.

EMACIATION (*emacio*, I make lean).—Wasting or loss of flesh. The term is applied both to the process of wasting and to the condition that results therefrom. See ATROPHY; and ATROPHY, GENERAL.

EMBOLISM (ἐμβολον, a plug).—SYNON.: Fr. *Embolie*; Ger. *Embolie*.

DEFINITION.—The mechanical lodgment in a vessel of some material, foreign to the normal

blood, which has been carried along in the course of the circulation.

Nature of emboli.—Emboli usually consist of portions of detached thrombus, i.e. of fibrinous clot which has been deposited in a vein or on the heart-valves. Softening thrombi are specially liable to become detached and be carried away. Less commonly other solid substances are carried along by the blood-stream,—fragments of heart-valves in infective endocarditis, pieces of tumours which have involved blood-vessels, liver-cells after traumatic injury, or even parasites. An embolus is not necessarily solid; fat, which is fluid at the body-temperature, may gain access to the circulation and lodge in the capillaries. The same is true of air. Emboli may also consist of minute bacterial masses.

Situation.—Foreign materials can only travel in the direction of the blood-stream, and can only be arrested in passing from a larger to a smaller vessel. Hence arteries and capillaries are the common sites of embolism; the only vein in which the needful conditions occur is the portal. The bifurcations of arteries offer special facilities for the arrest of emboli. The point at which a foreign mass lodges evidently depends upon its size: minute particles can be arrested only in the narrowest capillaries. A contorted cast of a vein may, however, block an artery of wider lumen than the vessel in which it originated. The source of an embolus is to be found by following back the blood-stream. Pulmonary emboli almost invariably arise from thrombi in the right heart or the systemic veins (see LUNGS, Hæmorrhage into). In the systemic arteries and capillaries they arise from clots in the left heart. Nevertheless, foreign particles may be so minute as to pass some capillaries. A septic fragment from a clot in a systemic vein may cause a metastatic abscess elsewhere than in the lung: epithelioma of the tongue may give rise to a secondary focus in the liver.

The term 'recurrent' embolism is applied to successive blockages in the same arterial area. An embolus, lodging at a point of bifurcation, may be broken up by the force of the current, and its fragments lodge further on; or small emboli may be followed by a larger one in the same district. 'Paradoxical' or 'crossed' embolism means the lodgment in a systemic artery of an embolus which has had its origin in a systemic vein or in the right heart, the pulmonary system having been short-circuited. Such cases are very rare: a patent foramen ovale has been present in the best authenticated instances. 'Retrograde' embolism means the transport of the embolus in a direction contrary to that of the normal current. It is a very rare occurrence, possible in veins only, and apparently due to temporary reflux of blood under special conditions.

Morbid Anatomy.—The embolus may not at first totally block the passage of blood. But even so, complete obstruction is usually soon produced by the consecutive thrombosis which occurs around it and which tends to spread back to the nearest arterial branch. In a recent case the embolic mass is easily recognisable, *post mortem*, by its obviously foreign characters and by the absence of any organic connection with the vessel-wall. Structural identity with its source, where this is traceable, can usually be established by microscopical examination. Where a longer period has elapsed before death, the recognition of the embolus is less easy, but a

longitudinal section of the blocked vessel may reveal the foreign mass embedded in more recent local thrombus. At times the diagnosis between thrombosis and embolism may be altogether obscured by the secondary changes.

Effects of Embolism.—These vary with the character of the embolus, as well as with its seat. If aseptic and non-irritating, the results are due solely to the mechanical block of the blood-stream; if septic and irritant, special local lesions will be superadded.

Aseptic or 'bland' embolism is the commoner form. The larger the vessel obstructed, the more serious, as a rule, are the results. The important factor is the readiness with which a collateral circulation can be established. Obstruction of the radial artery is less serious than that of many a smaller vessel, because of its free communication with the ulnar. *Terminal or end-arteries* are those devoid of arterial anastomoses; the territories which they supply have only capillary communications with adjacent areas: embolic obstruction of such arteries has grave results. Capillary embolism, unless multiple and widespread, is of little importance when aseptic.

When an artery is plugged, the current on the proximal side, back to the nearest collateral branch, is feeble or absent according to the degree of obstruction; the channel soon shrinks, and is more or less closed by consecutive thrombosis. The diverted blood seeks other channels. The principle upon which a collateral circulation is established, whether the obstruction depend on embolism or other cause, was first enunciated by von Recklinghausen. Certain of the branches of the blocked vessel, arising on the proximal side of the obstruction, almost always supply capillary areas contiguous with that from which the blood has been cut off. Such branches have at their disposal, at least by capillary anastomosis, the capillaries of the anæmic area, as well as their own. The resistance in them is lowered, and the current-rate increases; the blood, in short, follows the line of least resistance, and the size, and ultimately the structure, of the vessels which transmit it are modified in accordance with the volume and velocity of the stream. The effect of the original arterial block is thus counteracted by a rearrangement of the vessels. Such permanent compensation takes time, and the more strictly 'terminal' the artery obstructed, the more difficult is the compensation.

Deprivation of blood, unremedied, leads to tissue-death, and different tissues vary widely in their tolerance of such deprivation. Even temporary anæmia impairs functional activity, and if the affected organ be a vital one death may result. Embolism of a coronary artery, or even of one of its main branches, may thus cause sudden death (see MYOMALACIA). Some of the very highly specialised tissue-elements, for example, nerve-cells or renal epithelium, are so sensitive that they perish before the blood-supply can be restored (see BRAIN, Vessels of, Diseases of). This is not necessarily the case with the more lowly specialised connective tissues, whence embolism may lead to the phenomenon of 'partial necrosis,' i.e. death of the specialised, with survival and proliferation of the unspecialised tissue-elements: thus obstruction of a small branch of a coronary artery may lead to the formation of a fibroid patch in the heart. In some tissues, such as skin, bone, and

voluntary muscle, local death seems never to occur, partly because these tissues are not very sensitive, and partly because their arteries are not terminal.

If, as is commonly the case with terminal arteries, the anæmia resulting from embolic obstruction cannot be remedied in time to prevent the death of the tissues supplied, the resulting phenomena nevertheless vary in different situations.

(1) If the dead area be a superficial one, exposed to the action of putrefactive bacteria, gangrene may result, as after embolism of the main artery of a limb. (2) If the dead area be surrounded by living tissue, the process known as 'coagulation-necrosis' is apt to occur: fibrin is deposited from the lymph bathing the tissues, probably owing to the liberation of nucleo-proteid from the dead cells. The latter further swell up by imbibition of fluid, compressing the blood-vessels, and thus arises the *pale or anæmic infarct* commonly seen in embolism of the kidney—a firm, pale yellow, bloodless area, usually surrounded by a zone of congestion, and commonly wedge-shaped, from the mode of arterial distribution. (3) In other cases no such coagulation occurs, and the dead tissue becomes more fluid than natural, as in *embolic softening* of the brain. (4) Lastly, in certain situations, notably in the lung, spleen, and intestine, hæmorrhage may be added to the phenomena of local death, giving rise to *hæmorrhagic infarction*. The mechanism by which this occurs is as follows: the capillary blood-pressure in the territory of the blocked artery falls far below that in the adjacent areas; hence blood tends to pass into this territory from elsewhere, and it becomes much engorged, since there is not sufficient *vis a tergo* to propel it onwards into the veins. Cohnheim taught that the engorgement depended on reflux of blood from the veins. Litten brought forward experimental evidence against this view, and propounded the theory, now usually accepted, that the engorgement depends on afflux from collateral channels, through arterial or, more usually, capillary anastomoses. Something more than mere engorgement, however, occurs in hæmorrhagic infarction. The nutritional damage to the capillary walls renders them abnormally permeable, and a copious diapedesis of corpuscles takes place from the engorged vessels. Coagulation occurs, and a firm, dark-red, wedge-shaped mass is produced—the hæmorrhagic infarct—and this may be ultimately decolourised, as in the spleen. In any case, if the patient survive, the infarct, whether anæmic or hæmorrhagic, tends to undergo molecular disintegration and to be ultimately absorbed, leaving only a fibrous scar to mark its site—for the dead tissue excites around it a reactive fibrosis; calcification may occur in its remains.

Septic Embolism is a much more serious condition than bland embolism, for it produces all the mechanical effects of the latter, with, in addition, the local mischief wrought by the conveyed bacteria; these act, moreover, at a special advantage in a damaged area. The special local effects of the action of septic organisms in an embolus may be classed under three headings. (1) They may excite inflammatory softening in the wall of the blocked artery, and cause it to yield under the blood-pressure, forming an aneurysm. Such aneurysms are occasionally seen in infective endocarditis. (2) The softened wall may give way without any preliminary bulging, leading to local hæmorrhage. The subjects of infective endocarditis die, not uncommonly, of cerebral hæmorrhages thus produced. The cutaneous petechiæ and the retinal

hæmorrhages often seen in septicæmia are examples of a similar process on a smaller scale. (3) Local supuration may be set up where pyogenic organisms are conveyed by an embolus. Thus arise metastatic abscesses in the lungs in septic thrombosis of cerebral sinuses or other veins, and the visceral abscesses seen in pyæmia or infective endocarditis. See HEART, Diseases of.

Fat-Embolism is chiefly met with in the lung. It occurs after fracture of bones or traumatic injury of other tissues rich in fat. It is easily recognisable under the microscope if the lung be examined in the fresh condition, the refractile oil-globules being conspicuous objects in the pulmonary capillaries. In sections of the hardened lung staining with osmic acid renders them more conspicuous. It is probable that exaggerated importance has been attached to the condition. An aseptic oil-globule has almost no irritating properties, and it has been found by experimentally injecting oil into the vessels of animals that symptoms are frequently absent even though embolism is extensive. It is probably a very rare cause of death in man, though its occurrence is not uncommon. Septic fat-embolism is naturally a very serious and fortunately a rare occurrence.

Air-Embolism.—The entry of any considerable amount of air into a vein leads to speedy death, and the danger of this mishap is well recognised by surgeons in operations exposing the great veins. Should it occur, vascular obstruction by air-bubbles may undoubtedly be found *post mortem*—a true air-embolism. Yet the chief danger does not lie in this, but in the beating up of the mixture of air and blood into a froth upon which the right heart contracts ineffectively, whereby the circulation is brought to a stop. In cases which do not end fatally the air is soon reabsorbed.

TREATMENT.—Little can be said on the treatment of embolism, for the condition itself is irremediable by treatment. In the presence of any lesion liable to produce it, such as venous thrombosis, it is of the first importance to adopt such measures as will prevent its occurrence—the chief of which is perfect rest till sufficient time has elapsed for the organisation of the thrombus. Should it occur, all that can be done is to place the patient under the best conditions for the establishment of a collateral circulation. Threatened gangrene of a limb may be averted by rest and an elevated posture. The patient's strength must be supported by suitable food and stimulation in the hope that time may be gained for natural processes of compensation.

F. W. ANDREWES.

EMESIS (ἐμέω, I vomit).—A synonym for vomiting. See VOMITING.

EMETICS (ἐμέω, I vomit).—SYNON.: Fr. *Émétiques*; Ger. *Brechmittel*.

DEFINITION.—Agents that produce vomiting.

ENUMERATION.—Copious draughts of Lukewarm Water, Mustard, Sulphate of Zinc, Sulphate of Copper, Carbonate of Ammonium, Common Salt, Alum, Chamomile, Tartar Emetic, Ipecacuanha, and Apomorphine.

ACTION.—The act of vomiting consists in the simultaneous spasmodic contraction of the diaphragm and abdominal muscles, and relaxation of the cardiac orifice of the stomach, so that its contents are expelled. When the diaphragm and abdominal

muscles contract, but the cardiac orifice remains closed, so that the contents of the stomach cannot escape, the expulsive efforts are termed *retching*. The nervous centre which regulates these movements is situated in the medulla oblongata; and it may be excited either directly by the action upon it of drugs carried to it by the blood, or reflexly by irritation of various nerves. The drugs that act directly upon it have the same action, whether they are introduced immediately into the circulation or absorbed by the stomach. They may thus produce vomiting and evacuation of the stomach without being taken into the stomach at all, and on this account they are termed *indirect* emetics, although they act directly upon the vomiting centre. Such are ipecacuanha, apomorphine, and tartar emetic. Similarly the drugs that excite it reflexly are still termed *direct* emetics, because they are applied directly to the stomach. Such are the sulphates of zinc, copper, and aluminium; carbonate of ammonium; salt; mustard; and chamomile; which irritate the nerves of the stomach. In many cases the drugs which excite vomiting when injected into the blood are carried by it to the stomach and are eliminated by the gastric mucous membrane. During this process they irritate the stomach as much as, or more than, if they had been directly introduced into it. Tickling the fauces with a feather, or with the finger, also excites reflex vomiting, and may be adopted either alone, or in order to aid the action of other emetics. The terms *direct* and *indirect*, therefore, as applied to emetics, relate to the stomach, and not to the centre for vomiting.

Direct emetics, as they stimulate the nerves of the stomach only, have little action except that of simply exciting vomiting. The indirect emetics, which excite vomiting by their action on the medulla oblongata, act also on other parts of the nervous system, and cause secretion of saliva, secretion of mucus from the œsophagus, stomach, and bronchial tubes, and perspiration. They also cause much nausea, depression of the circulation, and loss of nervous and muscular power. Further, the vomiting they induce is more continuous and violent, and often expels the contents of the gall-bladder, causing part of the bile to flow into the stomach, and be thus evacuated.

USES.—Emetics are employed to remove the contents of the stomach under various circumstances. Firstly, when the food is causing irritation, and not undergoing proper digestion, or when bile is present in the stomach, as, for example, in dyspepsia or sick-headache; and in such cases large draughts of lukewarm water, of mustard and water, or of an infusion of chamomile are usually found beneficial. Secondly, in cases of poisoning; and here mustard, sulphate of zinc, and sulphate of copper are best, as they empty the stomach most quickly and effectually. Thirdly, to cause expulsion from the air-passages of false membrane in croup or diphtheria, or of secretions in bronchitis. For these purposes ipecacuanha is the emetic most frequently chosen; but if it do not act rapidly in croup, sulphate of zinc or sulphate of copper may be employed, or a teaspoonful of powdered alum and honey. In cases of either croup or bronchitis, where there is great depression of the circulation, carbonate of ammonium may be used with advantage, as it not only causes vomiting, but at the same time stimulates the circulation.

Caution should be exercised when giving emetics

to old people with atheromatous arteries, to persons suffering from hernia, and to pregnant women.

LAUDER BRUNTON.

EMMENAGOGUES (ἐμμηνα, the menses; and ἄγω, I move or expel).

DEFINITION.—Remedial agents to promote the menstrual flux.

ACTION.—The absence or decreased amount of the menstrual flow arises from very varied causes. It may depend upon anæmia, plethora, or some other blood-condition, or may be due to local disorders of the generative organs. Emmenagogues which act by altering the quantity or quality of the blood, or by giving tone to the nervous system, or by stimulating organs adjacent to the uterus, are known as *indirect* emmenagogues. Agents which stimulate the mucous membrane of the uterus are known as *direct* emmenagogues, but it must be remembered that some 'indirect' emmenagogues also exert a 'direct' action.

Many of the direct emmenagogues, when their action is pushed, cause powerful contraction of the uterus, leading to abortion, and are called *ecbolics*. Among these is ergot, the mode of action of which appears to be finally determined by Hemmeyer, who has shown that after section of the spinal cord ergot failed to produce any uterine contraction, even though its fibres were still readily excitable by the injection of ammonia into the uterine veins.

ENUMERATION.—*Indirect* emmenagogues include iron, strychnine, and other tonics, myrrh, warm hip-baths, leeches, and some purgatives such as aloes. Aloes probably stimulates the uterine mucous membrane as well as the large intestine.

The *direct* emmenagogues are savin, rue, cantharides, guaiacum, apiol, quinine, ergot, digitalis, tanacetin, pennyroyal, and the root of Gossypium herbaceum, possibly also permanganate of potassium. Of these drugs savin has a powerful action on the uterus. Its use is accompanied by grave dangers from its action on the nervous system. Rue acts similarly but less violently; myrrh is generally believed to exert more indirect than direct action; cantharides is a decided uterine stimulant. The action of permanganate of potassium is marked in some cases, though it does not appear to be an abortifacient.

USES.—Checked or retarded menstruation resulting from anæmia or general debility usually yields to indirect emmenagogues. If, however, the case prove more obstinate, one or more of the direct emmenagogues will often restore the suspended function. Should these fail, some mechanical impediment probably exists, or some altered physical condition of the uterus which no drugs can rectify.

SIDNEY PHILLIPS.

EMOLLIENTS (*emollio*, I soften).—**SYNON.** : Fr. *Emollients*; Ger. *Erweichende Mittel*.

DEFINITION.—Substances that soften and relax the parts to which they are applied.

ENUMERATION.—The principal emollient applications are:—Warm water, Steam, Poultices made of substances which retain heat and moisture, for example, crushed linseed, bread, bran, flour, oatmeal, and figs; Fatty substances, as linseed, olive, almond, and neat's-foot oil, lard, and suet; Spermæti, Wax; Soap Liniment and other liniments; Glycerin; Paraffin; and Wool-Fat. To these may be added such substances as do not properly

relax the tissues, but protect the surface from irritation, such as White of Egg, Gelatine, Isinglass, Collodion, and Cotton-wool.

ACTION.—Emollients relieve the tension and pain of inflamed parts by their action both upon the blood-vessels and upon the tissues themselves. They cause all the contractile tissues to relax and dilate, and thus, lessening pressure upon the nerves of the part, they relieve pain. They soften superficial parts by supplying them with either fat or moisture, and by increasing the supply of blood. In this way they prevent the skin from cracking after exposure to cold.

USES.—Fatty emollients are used to prevent the skin or mucous membranes from cracking; to prevent irritation or ulceration between parts constantly in contact, as on the limbs of children near the joints; to prevent bed-sores; to aid the healing of blisters; or in skin-diseases, such as eczema. They are also used, especially in the form of linimentum calcis, as applications in burns and scalds, for which purpose such substances as cotton-wool are likewise frequently employed. Mucilaginous substances are useful, when swallowed, to relieve pain and irritation in the throat, and to lessen irritable cough; and such substances as figs are employed to protect the intestines from injury by hard and pointed substances which have been swallowed. Warmth and moisture are applied in the form of antiseptic fomentations to the surface in pustules, boils, carbuncles, and deep-seated inflammation of the limbs, and in inflammation of the internal organs (see *POULTICE*). When the cuticle is lost they form a protective covering, under which the skin may heal, preventing the injurious consequences of friction from without. In the form of vapour they are useful in inflammation of the air-passages. See *INHALATIONS*, Therapeutic Uses of.

LAUDER BRUNTON.

EMPHYSEMA OF LUNGS.—See *LUNGS*, Emphysema of.

EMPHYSEMA, SUBCUTANEOUS (ἐν, in; and φῦσα, wind).—**SYNON.** : Fr. *Emphysème*; Ger. *Emphysem*; *Windgeschwulst*.

DEFINITION.—Emphysema denotes a distension of the areolar tissue of the body with air or other gases. It is called surgical, cellular, or subcutaneous, to distinguish it from vesicular emphysema of the lungs.

PATHOLOGY.—Subcutaneous emphysema is due to two causes: (1) an abnormal communication between an air-containing cavity and the areolar tissue; and (2) the formation of gas as a result of bacterial activity. The first cause is common, the second is rarely seen.

The most familiar example of the first cause is the fracture of a rib together with a simultaneous wound of the corresponding lung, or a punctured wound of the chest-wall, oblique in direction and traversing the tissues in such a way that the openings in the skin and pleura do not correspond. If the wound be closed so that air is unable to escape externally, it distends the subcutaneous tissue.

The mechanism of emphysema in the case of a wound of the lung either by a fractured rib or a stab is as follows:—as the chest-wall expands in inspiration, air escapes into the pleural cavity. In expiration, when the chest-wall collapses, air is unable to re-enter the lung from the pleural cavity on account

of the elastic recoil of the lung-tissue and its increased density. Air is therefore forced through the aperture in the parietal pleura into the subcutaneous tissue. With each successive respiratory movement more air is forced into the areolar tissue until the distension may affect the whole surface of the body. Pneumothorax is absent in most cases, either because the escape of air from the lung is limited or because the visceral and parietal layers of the pleura are adherent. Emphysema may also be due to disease of the lung such as rupture of a vomica or the bursting of a pulmonary abscess after the pleural layers have become adherent. A dangerous variety of emphysema is sub-pleural rupture of some of the air-vesicles during a violent expiratory effort. This is called *inter-lobular emphysema*, for the air escapes into the connective tissue of the lung, makes its way thence into the mediastina and the root of the neck. Other causes of emphysema in connection with the respiratory apparatus are fracture of the hyoid bone and of the laryngeal cartilages, wound of the larynx as in intubation, wound of the trachea as in 'cut-throat,' ulceration of the larynx, trachea, or bronchi. Fractures of the base of the skull involving the roof of the nose and the frontal, ethmoidal, and sphenoidal sinuses are complicated by emphysema, particularly in the orbit and the forehead. A fracture of the middle fossa of the skull passing across the tympanic cavity or involving the mastoid cells causes emphysema of the neck, and a fracture of the antrum gives rise to localised emphysema of the face. In injuries and diseases of the alimentary tract, e.g. after œsophagotomy or after perforation of the œsophagus, the third part of the duodenum, the posterior part of the cæcum or the rectum, air extravasates into the neighbouring connective tissue and thence into the areolar tissue beneath the skin.

Gas in the subcutaneous tissue not due to emphysema in the ordinary acceptation of the term is traceable to the action of bacteria. Prominent among those is the bacillus of malignant œdema which is found in acute spreading gangrene and causes emphysematous crackling in the decomposing tissues. Fraenkel has also isolated the *Bacillus phlegmones emphysematosæ*, and Wicklein the *Bacillus emphysematis maligni*. These are anaërobic bacilli, not identical with the bacillus of malignant œdema. *Post mortem*, gas forms in the internal organs, especially in the liver. According to Welch and Nuttall it is due to the *Bacillus aerogenes capsulatus*.

SYMPTOMS.—Extravasation of air may be limited to the part injured or diseased, or may be of great extent, causing extreme distension of the skin. When the swelling is limited it is slightly lobulated and elastic, pitting temporarily on pressure, which causes a characteristic fine crepitation, readily felt. On light percussion a superficial resonance can be obtained if the area affected be considerable. As a rule there is no redness of the skin unless decomposition is in progress. The position of an emphysematous swelling is a guide to the cause. Thus, emphysema of the orbit and forehead indicates fracture of the frontal sinuses. Emphysema of the upper part of the neck is due to injury or perforation of the larynx and trachea; emphysema appearing at the root of the neck arises from sub-pleural rupture of the pulmonary vesicles. Emphysema beginning in the chest-wall points to injury of the lung, but when it appears first in the flanks and spreads thence to the front of the abdomen it is generally due to

perforation of the intestine, except in the case of the rectum when the air extravasates into the perinæum, penis, and scrotum. In compound fractures the soft tissues may become emphysematous as the result of the shaking of the limb, or of the entrance of the bacillus of malignant œdema.

PROGRESS AND TERMINATIONS.—An extravasation into tissues from an aseptic source, such as the healthy lung, has no tendency to undergo decomposition, but when air has escaped from a pulmonary abscess or cavity, or from the bowel, considerable necrosis of tissue and inflammation occur. A notable example of this is seen in the sloughing of emphysematous tissue over a neglected strangulated hernia. When emphysema extends rapidly, especially if pneumothorax co-exist, dyspnoea soon sets in, and death from asphyxia may quickly follow.

TREATMENT.—A localised aseptic emphysema calls for no special treatment; pressure is useful in preventing its extension, and the air is quickly absorbed, although the possibility of suppuration should be borne in mind if there be an external wound. When the distension of subcutaneous tissue is so great as to seriously inconvenience the patient, multiple puncture of the skin with a needle is useful. If with a generally diffused emphysema there be pneumothorax and urgent dyspnoea, the wound in the chest-wall must be opened up, or if there be no wound the advisability of opening the pleural cavity must be seriously considered and prompt action taken.

A. H. TUBBY.

EMPIRICAL (ἐμπειρία, experience).—This term is applied to treatment founded on experience, but unexplained as contrasted with *rational*, which is founded on scientific reasoning.

EMPROSTHOTONOS (ἐμπροσθεν, forwards; and *τείνω*, I stretch).—A bending or drawing forwards of the body, due to tonic contraction of the muscles, observed in some cases of tetanic convulsions. See TETANUS.

EMPYEMA (ἐν, in; and *πύον*, pus).—A collection of pus within the cavity of the pleura. The term is occasionally used to denote a similar collection in a joint. See PLEURA, Diseases of.

EMS, in Germany.—Thermal muriated alkaline waters. See MINERAL WATERS.

ENAMEL, Workers in.—See OCCUPATION-DISEASES.

ENCEPHALITIS (ἐγκέφαλος, the brain).—Inflammation of the brain-substance. See various articles on BRAIN and MENINGES.

ENCEPHALOCLE (ἐγκέφαλος, the brain; and *κῆλη*, a tumour).—A hernial protrusion of a portion of the brain-substance through an opening in the skull, which may be either congenital or the result of accident, of surgical operation, or of disease.

ENCEPHALOID (ἐγκέφαλος, the brain).—A form of cancer, so named on account of its superficial resemblance to brain-tissue. See CANCER.

ENCHONDROMA (ἐν, in; and *χόνδρος*, cartilage).—A new-growth consisting of cartilaginous tissue. See TUMOURS.

ENCYSTED (ἐν, in; and κύστις, a bladder). Contained within a cyst. A term applied to new-growths, parasites, or collections of fluid thus enclosed, or limited by adhesions.

ENDARTERITIS (ἐνδον, within; and ἀρτηρία, an artery).—Inflammation of the internal coat of an artery. See ARTERIES, Diseases of; BRAIN, Syphilis of; and BRAIN, Vessels of, Diseases of.

ENDEMIC (ἐν, in; and δῆμος, a people).—This term is applied to diseases that prevail in particular localities or districts, and which are due to special aetiological conditions existing there. See EPI-DEMIC.

ENDERMIC MEDICATION (ἐν, in; and δέρμα, the skin).—Fr. *La Méthode Endermique*; Ger. *Intracutane Arzneiapplication*.

DEFINITION.—The method of using remedies either by rubbing them into the skin or by sprinkling them on a surface which has been previously denuded of its epidermis.

Endermic medication, once much practised, has almost, if not entirely, given place to hypodermic injection, (1) because the endermic method is the more painful; (2) because it is slower in action—thus, Trousseau found that twelve minutes were required for one-sixth of a grain of morphine endermically administered to take effect, whereas, hypodermically, the action of the same dose is observed in less than one minute; (3) because in many parts of the body endermic medication is totally inapplicable.

On the other hand, in a few cases, as for instance in localised pain, such as sciatica, and in paralysis of individual nerves, such as facial paralysis, this method may be employed with advantage, securing, as it does, the double benefit of counter-irritation by the use of the preliminary vesicant and the more or less direct and local action of the drug subsequently applied.

METHOD AND USES.—The epidermis should first be detached either by some ordinary vesicant, such as cantharides-plaster, or by the application of a cautery. The drug to be used is then laid on the raw surface in the form of powder rubbed up with starch, sugar, sulphate of sodium, or some other unirritating substance, paste made with water or thin mucilage, or ointment. In this way, neuralgia, sciatica, and other painful affections may be treated with the morphine-salts in doses of one-sixth of a grain and upwards; local paralyses and other nerve-troubles with strychnine in doses of one-fiftieth of a grain and upwards; and many other alkaloids may be used in the same way in various diseases.

A. S. CURRIE.

ENDOCARDITIS (ἐνδον, within; and καρδιά, the heart).—Inflammation of the lining membrane of the heart. See HEART, Inflammation of.

ENDOMETRITIS (ἐνδον, within; and μήτρα, the womb).—Inflammation of the lining membrane of the uterus. See UTERUS, Diseases of.

ENDOSCOPY (ἐνδον, within; σκοπέω, I look). The inspection of internal surfaces.—See the various articles on BLADDER, URETHRA, EYE, EAR, LARYNX, NOSE, &c.

ENEMA (ἐνέμημι, I inject).—SYNON.: Lave-ment; Clyster; Fr. *Clystère*; *Lavement*; Ger. *Klystier*.

DEFINITION.—An enema is a liquid injected by means of a suitable instrument into or through the rectum.

INSTRUMENTS.—Various instruments are used for the administration of enemata: 1. A simple elastic bottle with ivory or gum-elastic pipe, which has superseded the old bladder and pipe. 2. An india-rubber bottle with flexible tube at either end and double action, as in Higginson's instrument. 3. An ordinary piston-syringe, worked by the hand, which is either simple, or provided with a double action, so as to supply a continuous stream. 4. The hydraulic enema, which consists of a piece of india-rubber tubing about six feet long furnished with an ordinary ivory rectum-pipe at the one end, and a metal cone, or a screw-nozzle, at the other. It is well to place a soft rubber tube upon the end of the ivory nozzle to prevent any chance of injury to the mucous membrane from the hard ivory. The tube, being filled with the injection, has one end placed in the containing-reservoir, or connected to it by the screw; while a long rubber tube at the other end is introduced into the bowel. By careful management this tube may be introduced as far as the sigmoid flexure, and thus a quart or more of fluid can be passed into the colon. The vessel supplying the injection being placed on an elevation, the liquid gravitates into the bowel, filling the large intestine. When it is desirable to inject a large quantity the patient should lie first on the left side, then on the back, and lastly on the right side, to promote the filling of the intestine. In all cases care should be taken to prevent the injection of air into the bowel, and also to ascertain that the nozzle of the injecting pipe is free in or beyond the rectum, not in contact with the sphincter, nor thrust against the sacrum or into a hard fecal mass. The process of injection must be carried on slowly, with occasional pauses, otherwise premature contraction of the bowel with expulsion of the enema will result.

VARIETIES AND USES.—The chief varieties and uses of enemata are as follows:—

1. **Anthelmintic Enemata**.—To cure thread-worms injections of salt and water, or infusion of quassia, or from two to four drachms of spirit of turpentine suspended by yolk of egg in four ounces of water, are serviceable. A four-ounce enema of aloes or of asafetida may also be employed.

2. **Antispasmodic Enemata**.—Puerperal convulsions have been relieved by the injection of a solution of half a drachm or more of chloral hydrate. Injections of asafetida or of oil of rue are also given. Injections of warm water with two or three fluid drachms of sulphuric ether have sometimes relieved spasmodic invagination of the bowels. An enema of oil of turpentine or of asafetida will act well as a stimulant and carminative when the intestines are distended by gas.

3. **Astringent Enemata**.—These are used either to check diarrhoea, to arrest hemorrhage, or to remedy ulceration and mucous discharges. For the first of these purposes an enema of laudanum in thin starch is valuable. In cases of hæmorrhage from the bowels, as well as from the womb, injections of ice-cold water are frequently used. Cases of mucous colitis and of dysentery are often successfully treated with enemata of nitrate of silver (five grains to one pint of distilled water), of sulphate of zinc or alum (one or two grains to the ounce of water), or of sulphate of copper (one grain to the ounce of water).

4. **Emollient Enemata**.—Demulcents, such

as decoctions of starch, linseed, or barley; or pure linseed oil, are at times used with the object of imparting nourishment to the system, and of soothing an irritable mucous membrane.

5. Nutrient Enemata.—In cases of exhaustion, enemata of peptonised milk, beef-tea, and eggs beaten up are used. About four ounces should be given at once by means of a suitable syringe. When properly given, they will be retained and absorbed. Should the rectum become irritable, the irritability may be often lessened by adding a few drops of laudanum to each enema. The digestion and assimilation of nutrient enemata may be facilitated by the addition of preparations of pancreatin and pepsin. *See* PEPTONISED FOOD.

6. Sedative Enemata.—A sedative enema, as of laudanum, is often very useful in painful affections of the rectum and bladder. In uterine and ovarian irritation, warm-water enemata are very soothing.

7. Purgative Enemata.—These are used to overcome constipation. For this purpose—in the case of an adult—from one to two pints of fluid must be slowly pumped into the bowel. When the process is conducted gradually, stopping occasionally and making pressure on the anus if the injection threaten to come away, as much as four or five pints can be got into the bowel. The injection should be retained as long as possible, as thus a complete evacuation is ensured. As a general rule about a pint of liquid is enough for an adult; for an infant an ounce; for a child of four years, four to six ounces.

Composition.—Soap and water, gruel or thin starch mucilage with olive-oil, castor-oil, and sometimes oil alone, may be used. Sulphate of magnesium or aloes given per rectum is an efficient purgative. Glycerin-enemata (3i to ʒij) are now largely superseded by glycerin-suppositories.

The frequent use of very large injections is undesirable, lest undue distension result, with weakness and loss of tonicity in the bowel. The habitual use of injections washes away the mucus designed to lubricate the bowel.

JOHN C. THOROWGOOD.

ENGADINE, UPPER; in Switzerland.—A bracing mountain climate. Elevation of valley 5,000 to 6,000 feet. Season, June to September. Some parts are also open during the winter. *See* CLIMATE, Treatment of Disease by.

ENGLISH CHOLERA.—A synonym for simple cholera or choleraic diarrhoea. *See* CHOLERAIC DIARRHŒA; and DIARRHŒA.

ENGORGEMENT.—Overloading of the vessels, or of the heart, with blood. A synonym for congestion. *See* CIRCULATION, Disorders of.

ENOPHTHALMOS.—Retraction of the eye into the orbit. This condition is generally due to disease of the cervical sympathetic nerve.

ENTERALGIA (έντερον, the intestine; and άλγος, pain).—*SYNON.*: Enterodynia; *Neuralgia mesenterica vel mesaraica*; Colic. *See* COLIC; and INTESTINES, Neuroses of.

ENTERIC FEVER.—A synonym for typhoid fever. *See* TYPHOID FEVER.

ENTERITIS (έντερον, the bowel).—Inflammation of the intestines. *See* INTESTINES, Diseases of.

ENTEROCELE (έντερον, the bowel; and κήλη, a tumour).—A hernia, containing a portion of bowel. *See* HERNIA.

ENTEROLITH.—A concretion formed in some part of the intestines (*see* CONCRETIONS). Enteroliths are most commonly found in the cæcum and ascending colon, and, in the majority of cases, consist of firmly compressed, insoluble material, which has been swallowed as food or as medicine.

ENTEROPTOSIS (έντερον, the intestine; and πτώσις, a fall).—*SYNON.*: Splanchnoptosis; Glénard's Disease.

DESCRIPTION.—Enteroptosis, or falling of the abdominal viscera, is a condition which may affect individual viscera separately or the contents of the abdomen as a whole. Displacements of the kidney, liver, spleen, and stomach are dealt with elsewhere under the diseases of these organs, whether resulting from pathological changes in the viscera themselves, or from alterations in the relations of neighbouring parts.

Glénard's Disease, or general enteroptosis of such a degree as to demand attention, is much rarer than displacement of individual viscera. A slight degree of this condition is probably a common accompaniment of any considerable distension of the thorax, as in emphysema, and of relaxation of the abdominal walls such as follows pregnancy or other temporary cause of general abdominal enlargement.

ÆTIOLOGY.—The normal relation of the viscera to each other and to the parietes is maintained by the elastic pressure of the anterior abdominal wall, by suspensory bands of peritoneum, by the attachment of viscera to each other, and by the support afforded by blood-vessels. The whole abdominal contents may occupy a lower and more anterior position than normal, as a result of pressure from above, such as is produced by tight-lacing or the suspension of heavy clothing from a band around the waist. The action of gravity will produce a similar effect whenever the muscles of the anterior abdominal wall are permanently relaxed from want of tone or from previous over-stretching. General enteroptosis is therefore much commoner in, though not confined to, the female sex. It is sometimes associated with hernia and has been attributed to over-exertion and to straining in habitual constipation. The gradual traction exercised by peritoneal adhesions may in exceptional cases be responsible for a general displacement of the viscera.

PHYSICAL SIGNS.—In its slighter forms the condition can only be recognised when the patient is examined in the erect position. The patient is long-waisted; the upper part of the abdomen is flattened and its circumference small, while the lower part is prominent and perhaps pendulous. The edges of the liver and spleen may be felt below the costal margin. The kidneys may be palpable and freely movable. It is stated that the pancreas may be distinctly felt in a lower position than normal.

SYMPTOMS.—The symptoms are very variable and appear to depend more upon the patient's nervous organisation than upon the physical changes. In some cases the patient may be unaware of the existence of the condition even when it is well developed. In others there may be merely a sense of weight and dragging in the abdomen, which disappears as soon as the horizontal position is assumed. In the highly neurotic patients, in whom alone the

affection may assume a serious aspect, the symptoms may be of great variety and severity. The functions of any of the displaced viscera may be interfered with. Dyspepsia, vomiting, constipation, violent attacks of colic, and constant severe pain may reduce the patient to a condition of complete invalidism, of the bed-ridden, hysterical type. In these cases, as well as in patients who are not neurotic, urgent symptoms may occasionally result from torsion of the vessels supplying floating viscera, or from kinking of a ureter or some portion of the alimentary canal.

TREATMENT.—If the cause is pressure from above, this must be removed. Corsets must be forbidden and the clothing suspended from the shoulders. If the condition is due to a relaxed abdominal wall, attempts must be made to restore tone to the muscles by systematic exercises, friction, and electricity, the lost support being meanwhile temporarily replaced by a well-fitting abdominal belt. These measures failing, as they are likely to do in a highly neurotic patient, treatment by isolation and massage should be tried. As a last resource laparotomy may be advised, and may be successful by its effect upon the nervous system; by the surgeon's skill in fixing in their place some of the wandering viscera; or by the detection and division of adhesions which are retaining organs in an abnormal position.

LAURISTON E. SHAW.

ENTOPHYTE (ἐντός, within; and φυτόν, a plant). A plant parasitic in any part of the body. *Entophytic diseases* are diseases that are supposed to depend upon the growth of such plants, as, for example, fungus-foot. See MYCETOMA.

ENTOZOA (ἐντός, within; and ζῶον, an animal). Man, in common with other animals, vertebrate and invertebrate, is liable to entertain various forms of entozoa. In comparison with other vertebrates, even with members of his own order, Primates, the number known to infest man is singularly small. This is attributable in a large measure to the fact that his food is submitted to the process of cooking. The various entozoa found in the human subject are truly parasitic, and not examples of commensalism. A parasite inhabits a living organism, and obtains nourishment from its body. In commensalism creatures live within the bodies of larger animals, like parasites, but merely share the food, or live upon the refuse of the body of the host, not upon its juices and tissues. The more important entozoa found in man are:—

- I. *Cestoda*, Tapeworms.
- II. *Trematoda*, Flukes.
- III. *Nematoda*, Round-worms.
- IV. *Gregarinida*, Psorosperms.
- V. *Rhizopoda*, Amœba coli.

I. **Cestoda, or Tapeworms** (κεστός, a girdle).—Fr. *Cestoides*, *Ténia*; Ger. *Bandwurm*. Varieties:—

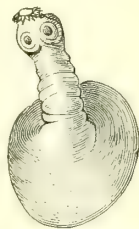
- a. *Tœnia solium*.
- b. *Tœnia mediocanellata* vel *saginata*.
- c. *Bothriocephalus latus*.
- d. *Tœnia cucumerina* vel *canina*.
- e. *Tœnia nana*.
- f. *Tœnia echinococcus*.

a. *Tœnia solium*.—This worm when fully developed may attain a length of 2 to 3 metres. The vertex or head, about the size of a small pin-head, is furnished with four suckers, a rostellum,

and twenty-six hooklets (fig. 3). A narrow neck succeeds the head, and at a short distance the segments begin to be visible. At first their breadth exceeds their length; but farther from the head they lengthen, until the mature segments are reached. These average 12 mm. in length and 5 mm. in width.

Each mature segment contains male and female generative organs. The genital openings are at the sides of the segments near the middle: the ovary presents seven to ten branches, each ending in a dendritic manner (fig. 5).

T. solium inhabits the small intestine of man, and is acquired by eating pork. Infection takes place in the following manner: The eggs, either free or contained in the segments (proglottides), are evacuated from the host, and are conveyed by means of food or drinking-water into the body of a pig or other animal. When the eggs reach the stomach of the pig the capsule is dissolved, and the liberated embryo makes its way into the wall of the intestines or is carried by the blood-current into other parts of the body. The young tapeworm, when it emerges from the egg, possesses six hooklets, and is known as the 'six-hooked embryo.' After settling in the tissues the embryo loses its hooklets and a cavity appears at the end opposite their attachment. From the wall of this cavity a head grows inwards. Around the parasite a thin fibrous capsule is formed. In this condition the parasite is known as a 'measle' or *Cysticercus cellulose* (κύστις, a bladder; and κέρκος, a tail) (fig. 1). Pork containing the cysticercus is said to be 'measled.'



The cysticerci are destroyed by thorough cooking. When a living cysticercus is introduced into the intestine, the head (scolex) fixes itself into the mucous membrane, the cyst-membrane is dissolved, a chain of segments or proglottides develop, and a tapeworm is the result.

The cysticercus occurs also in man, and is probably due to the introduction of the eggs of *T. solium* into the stomach. Cysticerci have been found in the aqueous and vitreous chambers of the eye, in the subcutaneous tissue, and in muscles. When encysted they retain their vitality for many years. The presence of these small bodies excites local irritation, inducing inflammatory thickening. In muscles and intermuscular tissue they occasionally cause swellings resembling tumours. In the brain, when numerous, they induce fatal results. A few may exist in the cortex of the cerebrum without producing any obvious effect, but in the neighbourhood of the medulla two cysticerci have been known to cause death. In the eye cysticerci have been detected by means of the ophthalmoscope. In this situation they present a striking and characteristic appearance.

b. *Tœnia mediocanellata* vel *saginata*.—This tapeworm differs from *T. solium* in several important particulars. When fully developed it may attain a length of 4 metres. The segments are thicker and wider than in *T. solium*; it has no rostellum or hooklets, but four suckers or proboscides (fig. 2). The mature segments have a larger

FIG. 1. —*Cysticercus (tœnia) cellulosus*, removed from the human eye; by Mackenzie, $\times 5$ diam. After Allen Thomson.

number of uterine diverticula, and they do not end dendritically (fig. 4). The cysticercus of this tapeworm infests the muscles of oxen. The tapeworm

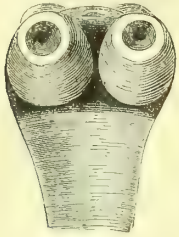


FIG. 2.—Unarmed Head of *Tania mediocanellata*, $\times 10$ diam. After G. Fritsch.



FIG. 3.—Armed Head of *Tania solium*, $\times 10$ diam. After G. Fritsch.

itself is acquired by man in consequence of eating infected beef. In the accompanying sketches the heads and proglottides of *Tania solium* and *Tania mediocanellata* are arranged side by side for comparison.

c. *Bothriocephalus latus*.—This is the largest tapeworm infesting man; when mature it attains a length of 5 to 8 metres, and contains two, three, and even four thousand segments, which are short and broad. The uterus has the form of a coiled ribbon, and the genital opening is near the middle of the ventral surface. The head is club-shaped and provided with two suckers.

This worm lives in the small intestine, and is met with in Switzerland and north-eastern Europe.

Fish are the intermediate host of this cestode: Braun has demonstrated its occurrence in the larval stage in the muscles and viscera of pike, trout, and turbot.

d. *Tania cucumerina* vel *canina*.—This is a small tapeworm about 3 decimetres in length, common in cats and dogs, rarely found in man. The head has a rostellum and about sixty hooklets arranged in four irregular rows. There are about 100 segments, of which the last 25 are mature. The intermediate host is the dog-louse, *Trichodectes canis*. This fact explains the large number of

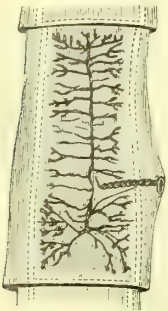


FIG. 4.—Proglottis of *Tania mediocanellata*, $\times 1\frac{1}{2}$ diam. After G. Fritsch.



FIG. 5.—Proglottis of *Tania solium*, $\times 1\frac{1}{2}$ diam. After G. Fritsch.

tania found in dogs not cleanly kept. The lice worry the dog, and it licks the irritated parts, and thus takes the lice with the cysticerci into the alimentary canal. Stroking dogs, or receiving their lingual caresses, is probably the means by which man is infected. It is noteworthy that children have furnished nearly all the specimens

of this worm which have been reported in the human subject.

e. *Tenia nana*.—This is a small tapeworm about 20 mm. in length, and consisting of about 150 broad segments. The head has four suckers, a rostellum, and circlet of twenty-two to twenty-eight hooklets. It is very rarely entertained in the alimentary canal of man.

SYMPTOMS.—The presence of tapeworms is indicated by a great variety of symptoms. Among the more important may be mentioned interference with the digestive process, irritability, and restlessness at night. Anæmia, headache, and vertigo are not uncommon. Irritation about the anus is sometimes a source of annoyance. Reflex phenomena, such as chorea, convulsions, or epileptiform seizures, insanity, mania, strabismus, and other untoward conditions, have been known to coincide with the presence of tapeworms in the alimentary canal, and to disappear when the worms have been expelled. Many other grave phenomena have been attributed to their presence. It is equally true that hundreds of individuals entertain tapeworms and suffer no inconvenience in consequence, nay, even are unaware of their guests until apprised of the fact by the appearance of two or three feet of cestode segments passed *per anum*. The presence of the *Bothriocephalus latus* is often associated with a form of anæmia identical with *pernicious anæmia*. See PERNICIOUS ANÆMIA; and BLOOD, Morbid conditions of.

TREATMENT.—Many drugs have been recommended, such as turpentine, koussou, kamala, areca-nut, and other anthelmintics. The liquid extract of male fern is the most reliable drug. The patient, if adult, should take a dose of castor oil at night, a drachm of extract early next morning on an empty stomach, to be followed in three hours by a small dose of castor oil. The fragments of worms should be examined in order to ascertain if the head has been expelled.

f. *Tania echinococcus* and *Hydatids* (*Hydatids*, a drop of water).—Fr. *Hydatide*; Ger. *Blasen-wurm*.—*Tania echinococcus* is especially interesting because its larval stages occur in man under the name of hydatid cysts.

The mature *tania* inhabits the intestine of dogs, jackals, and wolves. These animals are infected by feeding on the flesh of pigs containing the cysticerci. It is about 4 mm. in length, and consists of only four segments, of which the terminal one alone is mature. Its head has four suckers, a rostellum, and two rows of hooklets, varying in number from fourteen to twenty-five in each row.

The eggs of this worm are introduced into the alimentary canal with the food, but more commonly with drinking-water. The six-hooked embryo, emerging from the egg, makes its way into the surrounding tissues, and is frequently carried along by the blood-current in the veins as an embolus, to find a resting-place in lungs, kidney, liver, omentum, or brain, and becomes gradually transformed into an hydatid cyst.



FIG. 6.—*Tania echinococcus*, $\times 10$

There is no complete agreement concerning the development of these cysts. At first they are small white dots, with thick, homogeneous, transparent capsules, with concentric lamination, enclosing coarsely granular contents (fig. 7). After the cyst

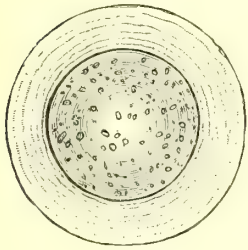


FIG. 7.—Hydatid of four weeks' growth, showing ectocyst and endocyst. $\times 60$. After Leuckart.

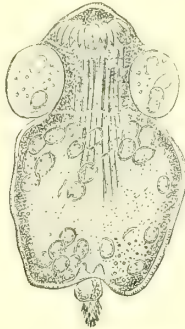


FIG. 8.—The so-called *Echinococcus* head, showing hooks, suckers, cilia, and corpuscles. $\times 250$ diam. After Huxley.

has been growing five months, has attained a diameter of half an inch, and is provided with an inner germinal layer, echinococcus-heads begin to bud inwards from its walls. Each of these heads, when fully formed, is about 0.3 mm. in length when fully distended, and represents the head and neck of an adult echinococcus (fig. 8). Each head is furnished with four suckers and an armed rostellum, the hooklets of which are very small. Numerous calcareous particles are lodged in its parenchyma. The rostellum with its hooks and suckers can be retracted and extended. In examining such specimens under the microscope it is usual to find them in the inverted or retracted condition shown in fig. 9. The heads or scolices are also formed within brood-capsules, and are throughout life believed to remain connected with the parenchyma

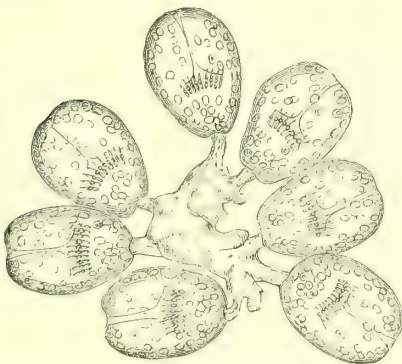


FIG. 9.—Group of *Echinococci*, with their hook-crowns inverted. $\times 180$ diam. After Busk.

of the main cyst. The walls of hydatids consist of two layers, an outer or cuticular and an inner layer or parenchyma. The cuticular layer is highly elastic and curls up when divided; the parenchyma consists of granular matter, cells, and muscle-fibre. The brood-capsules are developed from the paren-

chyma; each brood-capsule, like the parent-cyst, has two layers, an *outer* parenchymatous and an *inner* cuticular, thus reversing the conditions of the mother-cyst. Leuckart maintained that the scolices bud from the outer wall of the capsule; but when fully developed invaginate, so that what was formerly the internal cuticular surface of the head now becomes external. The mother-cyst grows larger as fresh brood-capsules are formed. Every echinococcus cyst does not develop brood-capsules, and may attain a large size yet remain sterile (*acephalocyst*). The presence of the cyst affects the surrounding structures, and leads to the formation of a fibrous capsule, sometimes of great thickness and usually very vascular. The brood-capsules, with their contained scolices, occur in clusters; each capsule is of the size of a No. 5 shot. Occasionally the capsules become much larger and form internal daughter-cysts attaining in some specimens the size of a Tangerine orange. Such daughter-cysts may develop cysts of their own, or granddaughter-cysts. When endogenous daughter-cysts are present, the mother-cyst is, as a rule, of very large size, and contains as many as two or three thousand daughter-cysts varying in size from a pea to an orange. Occasionally we find, in echinococcus cysts occurring in cattle, that daughter-cysts are formed from the cuticle and protrude beyond the *external* wall of the parent-cyst, and form brood-capsules. A few examples of this condition have been reported in the human subject.

Echinococcus multilocularis is a variety of hydatid, which has been described as affecting the liver. It forms a hard tumour made up of alveoli, separated by fibrous tissue. The alveoli are filled with gelatinous transparent material, stained at times with bile. Nearly all the reported cases have occurred in South Germany and Switzerland. The recognition of *E. multilocularis* is rendered more difficult by the circumstance that it sometimes ulcerates. This condition may easily be confounded with actinomycosis of the liver.

ANATOMICAL CHARACTERS IN MAN.—The human body frequently contains echinococcus cysts; there is scarcely an organ in which it has not been found, even in the medullary cavities of bones. Like the *cytticercus cellulose*, the embryo of *T. echinococcus* has its favourite situations: the former selects chiefly the intermuscular connective tissue and brain, the latter selects mainly the viscera, especially the liver. In more than half the total number of cases, the liver is the affected organ.

It is strange that, though the human liver is such a favourite viscus for hydatids, it is rare to find more than one cyst present in this organ at the same time, though in the liver of the pig they are frequently multiple. In man, one large cyst may be lodged in the liver, and twenty, thirty, or even a hundred occupy the omentum, mesentery, &c. This condition is, however, very uncommon.

SYMPTOMS.—The danger caused by these cysts arises mainly from the mechanical pressure they exercise on surrounding parts. Thus, in the brain or heart, a cyst the size of a hazel-nut gives rise to serious disturbance, but one as large as a man's head is often tolerated in the liver or pelvis. These cysts are often found *post mortem* in cases in which their presence had never been suspected. The parasite may die early in its career, and the cyst-wall calcify; hydatid membranes seem very prone to this change, the calcareous matter resembling very old

dried mortar. In the brain they simulate cerebral tumour, and induce death early, especially when growing near the pons or medulla. In the spinal cord they produce paraplegia. In the liver they may be accidentally ruptured, and cause death in a few hours from shock and hæmorrhage combined, or, later, from peritonitis. In some instances, after injury or meddlesome tapping, suppuration takes place with septic troubles, which the formation of pus entails. Cysts originating in the liver may perforate the diaphragm, and rupture into the pleura, or communicate with the lung and its bronchi. Occasionally they open into the stomach or bowel, the scolices being discharged *per anum*. They have been known to rupture into the bile-passages, the transit of the scolices causing biliary colic. Among the rarer sites of rupture may be mentioned the pericardium, the inferior vena cava, and the parietes of abdomen or thorax.

In the lung, pressure upon the air-cells or bronchi may cause gangrene and other troubles which follow bronchial obstruction. Hydatid cysts of the thyroid body are very rare; they usually rupture into the trachea and cause death by suffocation.

DIAGNOSIS.—The diagnosis of hydatid cysts, in organs other than the liver, is attended by very great difficulty. In the liver a hydatid cyst may be suspected when a tumour can be made out unaccompanied by pain, cachexia, high temperature, or wasting.

The conditions most likely to be confounded with hydatid cysts in the liver are abscess, distended gall-bladder, effusion in the right pleura, aneurysm of the abdominal aorta or hepatic artery, cancer of the liver, renal cysts, dilated stomach, pancreatic cyst, ovarian tumours, and dropsy due to closure of Winslow's foramen. See LIVER, Hydatid Disease of.

Although a person who has lived among dogs or in a pastoral country is especially liable to become infected, hydatids are often found in individuals who have never kept or lived with dogs.

When an abdominal or thoracic tumour is suspected to be a hydatid cyst, the nature of the swelling should be ascertained by puncturing it with a trocar, and drawing off some of the fluid. The characters which determine its nature are the following:—

The fluid is limpid, colourless (occasionally bile-stained when growing in the liver), and in specific gravity varies from 1004 to 1015. Free from albumen, unless any inflammatory effusion have occurred, it contains chloride of sodium, and is said to furnish traces of succinic acid, leucin, and tyrosin. The last three are present in quantities too small to be of any clinical value. The most positive signs are the presence of brood-capsules, scolices, hooklets, or the gelatinous, trembling, laminated membrane which forms the true wall of the cyst, and is in itself absolutely peculiar to hydatids.

It must also be remembered that hydatid cysts are occasionally sterile—that is, they contain no brood-capsules and furnish no hooklets. Such cysts frequently attain a large size, and seem to occur most frequently in the pelvis; they are as a rule mistaken for other forms of cysts.

TREATMENT.—The treatment of hydatid cysts is entirely surgical. When lodged in the omentum they are easily removed by abdominal section. In the pelvis they may be situated between the bladder and the rectum in the male, or burrow between the layers of the broad ligament in the female. These are better treated by incision and drainage. Hydatid

cysts in the kidney do not usually exceed the dimensions of an orange. They exhibit a marked tendency to discharge into the renal pelvis, the vesicles escaping by way of the urethra. Should the cyst attain such proportions as to give rise to a palpable tumour, it should be explored by a lumbar incision, and treated in the way recommended for the liver. Such cysts arising in the kidney have been known to open into the lung, the stomach, or the intestine. Intra-thoracic hydatids are best treated on the same surgical lines as empyema. In the subcutaneous tissues and orbit they are rarely diagnosed—indeed, these cysts in such situations can only be suspected; the suspicion is confirmed when the vesicles or portions of membranes escape during removal of the tumour. No tumour or cyst connected with the brain offers a more favourable prospect to surgeons than a hydatid cyst connected with the cerebral cortex. See LIVER, Hydatid Disease of.

II. Trematoda, or Flukes (τρίψα, a hole).—Fr. *Trématodes*; Ger. *Trematoden*; *Saugwürmer*. These forms have only one opening to the digestive cavity.

Varieties:—

a. *Distoma hepaticum*.

b. *Distoma lanceolatum*.

c. *Distoma hematobium* vel *Bilharzia hematobia*.

d. *Distoma Ringeri*.—See p. 415.

a., b. The first two are rarely seen, though they are common enough in the biliary passages of sheep and oxen. They are popularly known as flukes, and are the cause of the destructive disease known as 'the rot' of sheep. When flukes, with the exception of the *Bilharzia hematobia*, occur in man, they occupy the biliary passages, but are usually present in such limited number as to cause no serious disturbance of health.

c. *Bilharzia hematobia*, or *Distoma hematobium*, unlike other forms of flukes, has the sexes distinct. The male is 12–14 mm. long, and has a flattened body, which, at its posterior part, is rolled into a kind of tube, constituting the gynæcophoric canal. The female is almost cylindrical in form, and 16–

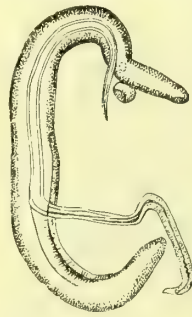


FIG. 10. — *Bilharzia hematobia*, male and female sexually combined. $\times 8$. After Küchenmeister.

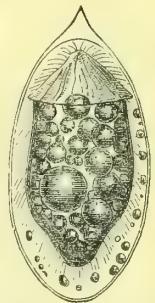


FIG. 11.—Ovum of *Bilharzia hematobia* with contained embryo and free sarcodengranules. $\times 234$.

19 mm. long, being lodged within the gynæcophoric canal (fig. 10). The mature worms live in the inferior vena cava, and the portal, splenic, mesenteric, vesical, and hæmorrhoidal veins of men and monkeys. The ova pass into the mucous membrane

of the bladder and intestine, probably escaping in consequence of the rupture of small vessels. Occasionally the ova may be found in the ureter and the pelvis of the kidney. The ova, often present in great numbers, are furnished with either a terminal or a lateral spine, the latter arrangement being commonest in the mesenteric forms.

The *Bilharzia* is common in Egypt, Abyssinia, the Cape, and Natal. The cercaria, as the larval forms are termed, abound in rivers and canals, and are especially common in the Nile. When taken into the alimentary canal the cercaria quickly work their way into the mucous membranes, gain access to blood-vessels (veins), and develop into mature flukes.

SYMPTOMS.—When lodged in the intestines they give rise to dysentery; in the bladder and ureters, to cystitis, hæmaturia, and vesical calculus. The hæmorrhage caused by their presence in the genito-urinary tract is known as ‘endemic hæmaturia.’ The eggs, while still within their host, may develop ciliated embryos. The disease may be suspected when hæmaturia occurs in persons coming from countries where the *Bilharzia* abounds: the diagnosis is confirmed by finding under the microscope the ova embedded in fragments and shreds of mucous membrane passed with the urine.

TREATMENT.—Means must be taken to allay the vesical irritation by such remedies as buchu, Uva ursi, hyoscyamus, and the like. No remedy is known that acts as a parasiticide to the *Bilharzia*. It is wise to remove the patient, if possible, from an infected locality. Cure is likely to be protracted, and as yet we are ignorant of the duration of life of the fluke.

III. Nematoda, or Round-worms (νήμα, a thread).—Fr. *Nématoides*; Ger. *Fadenwürmer*.

Varieties:—

- a. *Ascaris lumbricoides*.
- b. *Oxyuris vermicularis*.
- c. *Trichocephalus dispar*.
- d. *Ankylostoma duodenale*.
- e. *Fiilaria medinensis*.
- f. *Trichina spiralis*.
- g. *Fiilaria sanguinis hominis*.

Most of these parasites inhabit the alimentary canal, and when restricted to this region rarely give rise to dangerous symptoms, unless they be very numerous; but they are apt to migrate and invade other organs, and thus give rise to dangerous and even disastrous results. Their presence in the intestine is generally accompanied by extreme *eosinophilia* (see p. 157), the existence of which is often of material assistance in diagnosis.

a. *Ascaris lumbricoides*.—This, the common round or maw-worm, is of cylindrical shape, pointed at each end, and of a light brown colour. The female, as is usual among nematodes, exceeds the male in size. The length of the female varies from 25 to 40 cm. The male is distinguished by the presence of two chitinous spines near the caudal end of the body (fig. 12). This extremity is also bent and hook-like. The spines indicate the male genital orifice. In the female the genital pore is situated near the middle of the body. The ova are very numerous; when mature they possess a double shell surrounded by an albuminous coating, have considerable power of resistance, and are not killed by drying or freezing.

This worm lives chiefly in the small intestine, but may wander into the stomach, and even pass up the

œsophagus and be discharged through the mouth. An *ascaris* has been known to creep into the bile-duct and cause jaundice. In cases of perforation of the intestine in fatal cases of typhoid fever, these worms have been found lodged in the aperture.

We must not conclude from this that the worm caused the perforation; it is more reasonable to believe that when the accident happened the ascaris was passively lodged in the opening during the outward rush of the fluid contents of the bowel. It is possible that worms may occasionally perforate the intestinal wall and, getting into the subserous tissue, give rise to abscess. Such cases have been recorded. The wandering habit of ascarides is often fatal to them. Any foreign body in the intestine is sure to attract their attention and to form a sort of ‘worm-trap.’ Thus they have been strangled by metallic buttons, hooks and eyes, open-topped thimbles and the like (Cobbold).

SYMPTOMS.—It is unusual for an individual to harbour more than two or three round worms, and, as a rule, they rarely give rise to symptoms. When present in large numbers—a condition of things most common in children—diarrhœa, colic, nausea, vomiting, and convulsions may occur. Children have been known to pass in the course of a few years one hundred worms, and in one case five hundred and ten worms were voided by a child. Generally the passage of a worm by the anus or mouth is the first and only indication of its presence, unless the blood be examined (p. 157).

TREATMENT.—The most reliable drug is santonin, in the form of a powder: for children two to four grains, followed by a saline purge or castor oil; for adults five grains daily for three days, each dose to be followed by castor oil. Among other useful remedies we may mention aloes, scammony, jalap, calomel, kamala, sulphur, and turpentine.

b. *Oxyuris vermicularis* (Thread- or Seat-worm).—The thread-worm is an inhabitant of the colon, cæcum, appendix vermiformis, and adjacent parts of the ileum. The female is usually about 10 mm. and the male 4 mm. long. In addition to its greater size the female has a sharp-pointed tail (fig. 13), whereas in the male it is blunt and furnished with a spiculum. Immature oxyurides are frequently found in the appendix vermiformis, which may serve as a breeding-place for the parasites (Still).

Oxyurides are often present in large numbers,



FIG. 12. *Ascaris lumbricoides*, female with exserted spicules. Natural size. (Cobbold.)

and wander into the rectum. They often cause catarrh of the mucous membrane of the large intestine and appendix vermiformis, as well as great irritation in the neighbourhood of the anus. In

females they may cause great distress by creeping into the genital passages, especially during the night. The eggs of the oxyuris (fig. 14) are expelled with the fæces.

SYMPTOMS.—Among the local symptoms heat and irritation around the anus and nose are common. In children the presence of the worms is suggested by restlessness, nervous irritation, choreic symptoms, and convulsions. Signs suggesting such severe conditions as meningitis are by no means infrequent. The existence of thread-worms is easily verified by administering a mild purgative and examining the fæces. As a rule, they will be found in considerable numbers. In the case of children

is thicker, and contains the genital organs. In the male the caudal end is rolled into a spiral; near the extremity it is furnished with a spine (fig. 15).

The trichocephalus resides mainly in the cæcum. The early stages are passed in water or damp earth. The worm is very common in France: Davaine calculated that half the Parisians are infested by it. In England it is rare. Unless present in great quantities it is not often a source of trouble.

d. Ankylostoma duodenale.—This small worm is also known as *Sclerostoma duodenale*, and *Dochmius* or *Strongylus duodenalis*. Occasionally it is referred to as the *tunnel-worm*, on account of the disasters it caused among the men engaged in the St. Gotthard Tunnel. The female averages 12 mm. in length, the male is slightly smaller. The cephalic end is furnished with an oral capsule, which possesses a cleft covered by two chitinous lamellæ. The ventral lip has four curved teeth; the dorsal, two straight ones. The caudal end in the male has a three-lobed bursa and two spines (fig. 16). This worm infests the small intestine and bores its way into the mucous membrane. The effects of ankylostoma on the gut are very characteristic. The damaged spot is indicated by a small ecchymosis, with a central depressed white spot, in which the head of the worm lodged; occasionally it may be found still anchored to the mucous membrane. When present in large numbers these worms produce anæmia, sometimes called Egyptian chlorosis. The ankylostoma is very common in Egypt and Brazil. The ova are oval: they develop rapidly in muddy water and in mould, especially if these are mixed with fæces. Care should therefore always be taken to prevent contamination of the soil. The parasite gains access to the alimentary canal in drinking-water, and there attains maturity in five or six weeks.

SYMPTOMS.—The presence of this worm is generally indicated by the usual signs of anæmia, viz. pallor of the mucous membranes, weakness, palpitation, and faintness. The anæmia is due to the loss of blood consequent on the injuries inflicted by these parasites on the intestinal mucous membrane; and is characterised by eosinophilia (p. 157) and poikilocytosis. Intestinal colic, diarrhoea, and dropsical effusions sometimes occur.

TREATMENT.—The patient is kept on fluid diet for twenty-four hours; an aperient is then given in the evening, followed next morning by three doses (10 to 30 grains) of thymol in cachets at intervals of one hour. After the lapse of four or five hours another dose of aperient is given. The patient should remain in bed during the treatment, and no alcohol or other solvent of thymol be allowed.

e. Filaria medinensis, Dracunculus, or Guinea-worm.—This is a fine thread-like worm from 60 to 100 cm. in length, and about 2 mm. broad. The female only is known. The embryos (1 mm. in length) develop within the uterus. The intermediate



FIG. 13.—*Oxyuris vermicularis*, female. $\times 12$. After Leuckart.



FIG. 14.—Eggs of *Oxyuris vermicularis*, enclosing tadpole-shaped embryos. $\times 450$ diam.

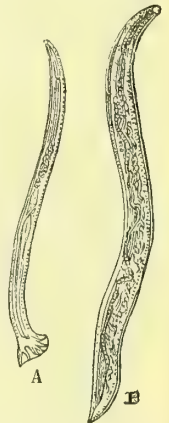


FIG. 16.—*Ankylostoma duodenale*; male (A), and female (B). $\times 5$ diam.

the diagnosis is often made by finding the worms on the sheets.

TREATMENT.—Brisk saline purgatives to expel the worms, and extreme cleanliness to prevent reinfection, are occasionally all that is required. Fruits and vegetables should be thoroughly cooked. In most cases such drugs as calomel, scammony, aloes, jalap, santonin, or asafetida should be given by the mouth, in conjunction with copious enemata of infusion of quassia, or salt and water. The application of dilute mercurial ointment to the anus will destroy the wandering parasites and prevent the infection of the vagina. When the appendix vermiformis is involved the parasites are extremely difficult to dislodge.

c. Trichocephalus dispar, or Whip-worm.—The male and female equal each other in length; as a



FIG. 15.—*Trichocephalus*; male (a), and female (b). Enlarged one-fourth.

rule they average 35 to 50 mm. The anterior part of these worms is fine and thread-like, the posterior

host is a minute crustacean (Cyclops), which inhabits drinking-water. While within the cyclops they are introduced into the human stomach, and then migrate into the subcutaneous tissues, where



FIG. 17.—*Filaria medinensis*. Reduced $\frac{1}{2}$.

they become mature. A common situation is the tissue beneath the skin of the legs and feet; the heel is a favourite situation.

The worm is confined to certain districts of Asia, Africa, and Brazil.

When it first lodges in the subcutaneous tissue it gives rise to little trouble; as it grows an abscess forms and gradually exposes the worm. Treatment consists in the injection of a few drops of a 1 per 1,000 solution of perchloride of mercury into the body of the worm, if this be protruding, or, by means of two or three punctures, into the tissues immediately in contact with it, if the skin of the host be intact (Emily).

f. Trichina spiralis.—The mature worm inhabits the intestinal canal. The female is 3 mm. long; the male is much smaller, and is further distinguished by presenting on the dorsal side of the tail two mammillary protuberances, which are turned towards the ventral aspect, and include between them four wart-like nodules (fig. 18). The young are

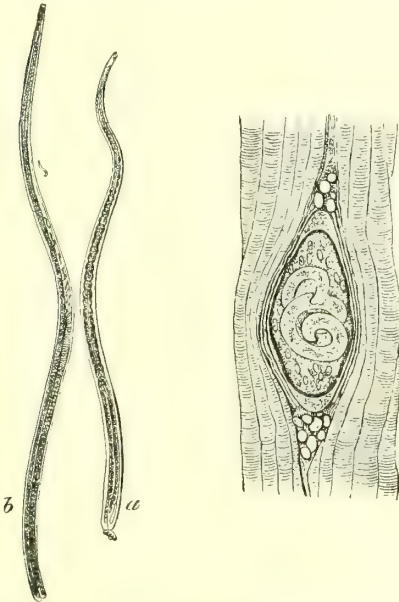


FIG. 18.—*Trichina spiralis*; male (a), and female (b). $\times 35$. Also nat. size.

FIG. 19.—Portion of human muscle, enclosing a single encapsuled trichina. $\times 75$. After Leuckart.

developed in the uterus, and are born in a free state. The immature trichina inhabits voluntary muscular fibre, where it is usually coiled into a spiral, embedded in granular material and surrounded by a cyst-wall, which frequently contains calcareous particles (fig. 19). These capsules are visible to the

naked eye as minute white specks. Each capsule usually contains one trichina, but two, three, and even five may be present.

The relation of the two forms is as follows. When a piece of muscle containing living trichinae is taken into the stomach, the capsules are digested and the worms set free. In two days and a half they become mature and pair. The embryos commence to be born on the eighth day; and the female may continue to bring forth worms for many days. The mature worm lives but a few weeks, but during this brief life may bring forth a thousand or thirteen hundred embryos. The young then seek for striated muscle-fibre, and migrate through the wall of the intestine and the subperitoneal tissue. A few may get into vessels and are distributed by the circulation. On gaining access to a muscle they penetrate the fasciculi, becoming encysted about three weeks after the original infection; the cyst is said to be in part due to a secretion furnished by the trichina. After a time the cyst-wall calcifies; should the trichina die, the cyst-contents also calcify. The duration of life of the worm in the muscle is very long, and it even survives for a time the death of its host. The trichinae are found most abundantly in the intercostal muscles, diaphragm, and laryngeal muscles. In the limb-muscles they are less abundant, and cluster near the union of the muscle with its tendon. The trichinae so accurately select the voluntary muscles that the pharyngeal constrictors and upper part of the oesophagus will lodge hundreds of them, while the rest of the tube contains none.

SYMPTOMS.—Human beings usually acquire the disease from eating trichinised pork. The symptoms may be divided into two stages: the first consists of gastro-intestinal disturbance due to the introduction of the parasite; the second corresponds to the migration of the worm, and is characterised by muscular troubles and febrile disturbance.

The gravity of the symptoms depends upon the number of trichinae taken. The initial gastro-intestinal catarrh commences about nine days after infection, and lasts usually about a week. The second stage develops as the intestinal catarrh subsides and is characterised by high fever and pain in the limbs and muscles, often mistaken for rheumatism. The muscles sometimes swell, become oedematous, and occasionally are partially paralysed. In this way aphonia, dysphagia, dyspnoea, trismus, and other serious symptoms may result. In grave cases delirium ensues, the limbs become flexed and paralysed, there is excessive diarrhoea, terminating in death. The duration of the second stage lasts four or five weeks. In fatal cases, death usually occurs about the third or fourth week.

At the end of five weeks the migration is usually complete, and the trichinae once encysted give rise to no further trouble. The diagnosis may sometimes be established during life by an examination of suspected articles of food, or by the detection of adult trichinae in the evacuations during the course of the disease. The most certain plan is to remove a fragment of affected muscle and examine it under the microscope. The muscular symptoms following the acute intestinal catarrh are however generally sufficient to establish the diagnosis.

TREATMENT.—If it can be at once ascertained that trichinised food has been taken into the stomach, an emetic should be administered without delay, followed by a brisk purgative; this treatment should be pursued for several days to expel the

worms before they become mature. Calomel alone, or combined with scammony or jalap, is recommended. Even when the young filariæ have begun their migrations, brisk cathartics still do good; but the patient's strength will require support during this critical stage by beef-tea, broth, eggs, milk, and the judicious administration of wine or brandy. As the patient becomes convalescent ordinary tonics will be needed.

Preventive treatment consists in the detection and rejection of infected pork rather than in increased care in cooking; since a temperature of 80° C., which is rarely reached in the interior of any large joint, is required to destroy the embryos. The disease is now rare except in America. Before the present stringent regulations obligatory at slaughter-houses in Germany, the disease was common in that country, where sausages made of uncooked ham are largely eaten.

g. Filaria sanguinis hominis.—This parasite and its embryo are separately described. See FILARIA-SIS.

IV. *Gregarinidæ* (*gregarius*, from *grex*, a flock).—Fr. *Grégariques*; Ger. *Gregarinen*.

Variety:—*Coccidium oviforme*. See PSOROSPERMOSIS.

V. *Rhizopoda* (ῥίζα, a root; and πούς, a foot).—Fr. *Rhizopodes*; Ger. *Rhizopoden*.

Variety:—*Amœba coli*.—See DYSENTERY.

J. BLAND SUTTON.

H. MONTAGUE MURRAY.

ENTOZoon FOLLICULORUM.—A synonym of the animalcule of the follicles of the skin, otherwise named *Acarus* (Simon), *Demodex* (Owen), and *Steatozoon folliculorum* (Erasmus Wilson). See ACARUS.

ENTROPION (ἐν, in; and ἑρπέω, I turn).—A morbid condition in which the eyelid is inverted, so that its free margin is directed towards the eye. See EYE, AND ITS APPENDAGES, Diseases of.

ENURESIS (ἐν, in; and οὐρέω, I pass the urine). Involuntary discharge or incontinence of urine. See MICTURITION, Disorders of.

EPHELIS.—SYNON.: Sunburn; Fr. *Ephélide*; Ger. *Sonnenflecken*.—This word is applied to pigmentary discolouration of the skin, of a brown, grey, or black colour, increased by the stimulus of light and heat, as of the sun's rays, or scorching by fire. Two principal varieties of the affection have been noted, namely, *Ephelis solaris* and *Ephelis ab igne*. See PIGMENTARY DISEASES OF THE SKIN.

EPHEMERAL FEVER (ἐπί, upon; and ἡμέρα, a day).—A mild form of milk-fever, so called on account of the rapidity with which it subsides, lasting not more than a day. See MILK-FEVER.

EPHIDROSIS (ἐπί, upon; and ἰδρώω, I sweat). A term signifying a state of sweating, and synonymous with hidrosis. See SUDORIPAROUS GLANDS.

EPIDEMIC (ἐπί, upon; and δῆμος, a people).

The word 'epidemic' is used in two senses: (1) As a *general term*, the word signifies 'common to, or affecting a whole people, or a great number in a community; prevalent; general' (Webster). It is in this sense that the word is used when it is applied to the mental, moral, and social phenomena, as for instance when we speak of 'epidemic suicide.'

(2) As a *technical term* having reference to disease, the word 'epidemic' is used in more than one sense; but all embody the idea of prevalence beyond that which is usual. In its substantive use it implies the existence of prevalence; in its adjectival use in connection with the word 'disease,' it implies the ability to assume prevalence. The employment of the word is not limited to diseases which are due to contagia. Thus Hecker included dancing mania among the epidemics of the Middle Ages, and Haeser uses the word in connection with ergotism and scurvy. Other writers would limit the use of the word to prevalence of a malady in a locality to which it is foreign, thus employing it in contradistinction to the word 'endemic.' Such limitation would prevent its employment in connection with a large prevalence of a disease which in smaller quantity is constantly present in the same locality, and which is due to conditions inherent in the locality or in its population; and this would be contrary to the general acceptance of the term. The essential idea of 'endemic' is the persistence of a disease in a locality or people as the result of local conditions; the essential idea of an 'epidemic' is exceptional amount of prevalence irrespective of the question whether the disease is in less degree constantly existent in the locality. The two words are not in complete contradistinction, but only in so far as they relate to magnitude of prevalence.

The amount of prevalence which may be properly deemed to constitute an epidemic is necessarily dependent on the difference between that amount and that which frequently or normally occurs, and therefore is dependent upon the effect which this prevalence has on the mind. Thus from two to three thousand persons, or more, frequently die in London in a single year from measles or whooping cough or diarrhoea, which are endemic. If the same number of deaths occurred from cholera, which is not endemic in this population, it would no doubt be spoken of as an epidemic.

It is this effect upon the mind, the terror which epidemics have inspired in the past, that has led to the wildest speculation as to their cause. The mysticism of former ages led to the search for explanations of the phenomena in extra-mundane conditions; and older writers tell of celestial portents, observed or believed to have been observed, which were currently accepted as presaging disaster. Later, 'portents' gave way to 'precursors,' and it was thought that the earlier epidemics of malignant cholera which invaded Europe were preceded by unusual prevalence of 'fevers' and diarrhoeal disease; but still later study has failed to find evidence of unusual behaviour of disease generally, preceding or accompanying a particular epidemic, such as would afford indication of the existence of an 'epidemic constitution' which rendered populations liable to attack. Experience of epidemic disease due to the contagia early demonstrated one mode of its distribution—the contact of the sick person with his fellows, or their contact with such articles as clothing with which he had been intimately associated. The existence of a contagium was thus assumed, but the ability of certain contagia to operate simultaneously upon numerous persons at long distances and over wide areas was not then capable of explanation. Difficulty undoubtedly resulted from the fact that communicable diseases of which the methods of diffusion were not all identical were not distinguished

from each other. Even in our own time the confusion between enteric fever and typhus led to perplexity; hence an ability to understand the phenomena of epidemics was dependent in the first instance upon a more precise knowledge of the nature of the maladies concerned.

The epidemics of cholera during the nineteenth century gave much opportunity for studying the causes of cholera under conditions particularly favourable for this purpose. The visitation of England by cholera in 1848 enabled the behaviour of the disease to be better appreciated than was possible in Eastern countries in which the disease is endemic; and as a result the important fact became manifest that water contaminated with cholera-dejecta possessed an enormous ability to cause that disease among persons who drank it. This one observation has done much to establish a method of inquiry which has been successfully utilised in subsequent investigations, and to dispel the mysticism which previously shrouded the study of epidemiology. In this connection the names of Snow and Simon will be held in grateful remembrance. In this instance water only was found to be the means of communication of the disease. Similar observation as to diphtheria and scarlet fever has not only included milk as a vehicle of contagion, but has raised larger questions as to the source of contamination, and has through the inquiries of W. H. Power and Klein taught that animal life other than human must be considered in seeking an explanation of epidemic disease in man. In these inquiries, methods of research widely different have been utilised. Inquiry in this field has in the main been based upon differentiation between the circumstances of those attacked by a disease and of those who have not been attacked, with eventual discovery by this means of a particular channel by which disease has been conveyed. All such inquiry has assumed the existence of a specific entity causing disease, but no further proof of its existence was obtainable by this method than that afforded by the evidence of its operation. Inquiry in the laboratory has been directed to the identification of this entity, to obtaining knowledge of its nature, of the manner in which it obtains entry to the animal body, of the conditions upon which the resistance of this body to its invasion depends, and of the means by which it can be communicated from one human body to another, with or without an intervening invasion of other animal bodies.

Other study of epidemic disease has had as its basis the consideration of statistics, the counting of the number of persons who have died from the particular cause, and (more recently) of the number of persons who have been attacked, so far as the symptoms to which this cause has given rise have enabled the disease to be identified. Study of this sort has shown that some epidemic diseases are seasonal in their prevalence; that while this ability to spread is undoubtedly affected by such circumstances as are incidental to man, some natural conditions have in paramount degree determined the prevalence of the disease, and its ability to spread beyond the limits of the area in which it is endemic and to manifest for a time its infective ability over wider areas. Along with regular variations in prevalence in each successive year, dependent apparently in their degree upon climatic conditions, which have been especially studied by Longstaff, are the larger variations over longer periods which

cause the greater epidemics or pandemics. These also maintain, as Whitelegge has shown, a tendency to rhythmical behaviour.

More recently, examination of the figures obtained by notification of infectious diseases in London has shown seasonal variation in age-incidence and seasonal variation in fatality (case-mortality); these facts support the view enunciated by Whitelegge that there is, from time to time, a change in the quality of the virus, enabling it to invade at one period more resistant persons than at another time, and to be more destructive of the individuals invaded at one time than another (*see PERIODICITY IN DISEASE*). The circumstances underlying these changes are not yet understood, but the more precise study of epidemic disease which is possible at the present time must increasingly add to our knowledge of its causes, and is already showing that for explanation of the behaviour of such disease, regard must be had to the changes to which the virus is subject owing to varying conditions, meteorological and telluric, and to passage through animals other than the human; moreover the channels of communication to man are not yet all known.

SHIRLEY F. MURPHY.

EPIDEMIC DROPSY.—**DESCRIPTION AND GEOGRAPHICAL DISTRIBUTION.**—In the cold season of 1877–78 an epidemic of a disease, whose most constant feature was dropsy, appeared in the southern suburbs of Calcutta. It died out as the hot weather set in, reappeared over an extended area in the cold months of 1878–79, and subsided in the hot season of 1879. A second recrudescence occurred in the cold season of 1879–80 over a wider area of the town and suburbs, and the disease finally disappeared in the hot season of 1880. An outbreak of the same kind appeared at Shillong on the Khasia Hills, in Assam, at Dacca and in some tea-gardens of South Sylhet, in 1878–79; and a similar epidemic broke out in the island of Mauritius in November 1878, and continued to prevail until April 1879, when it disappeared. No similar outbreak has been observed in Bengal or Mauritius before or since. Epidemics have occurred in some parts of tropical Asia and America and on ships trading in or with the tropics, of which dropsy was a prominent symptom, but these have been described as outbreaks of beriberi. *See BERIBERI.*

EPIDEMIOLOGY.—The Calcutta outbreak was the centre from which the others were exported. The exportation took place by means of travellers and labourers proceeding to tea-gardens and sugar-plantations. The disease restricted itself mainly to the classes among which it first appeared. Its power of diffusion was weak and progress slow, but personal intercourse was evidently the main instrument of extension. Social conditions seemed to have less influence than domestic in spreading the disease. Its subsidence in the hot and wet months and prevalence in the cold were well marked in Bengal, but seasonal changes did not affect its wax, continuance, and wane in Mauritius. Natives and half-castes were the exclusive sufferers, Europeans being exempt. Men were seized in larger proportion than women; children were susceptible, and among them sexual liability was equal. Distribution was unequal, particular houses or groups of houses showing an excess of cases. Insanitary conditions did not seem to determine incidence. Medical men, who had served in the Madras famine of 1876–77, recognised

the disease as similar to that which they had met with in the famine-tracts.

SYMPTOMS.—The appearance of the dropsy was usually preceded by fever, disturbance of stomach and bowels, burning and pricking of skin, and deep-seated pain in body and limbs. The pyrexia was mild and remittent in type, the temperature ranging from 99° to 101° F. In many cases an urticarial, scarlatinai, or morbilliform rash was observed. The lower extremities first swelled, and in bad cases anasarca of the trunk and upper extremities, seldom of the face, appeared. Effusion into serous cavities and oedema of the lungs sometimes occurred, giving rise to dyspnoea; and diarrhoea and mild dysentery were observed in some cases. Anæmia and emaciation were constant features. There was no enlargement of the spleen, and albuminuria was very rare. No anæsthesia, paralysis, or other symptom of multiple neuritis was observed. The illness was apt to be prolonged, and the death-rate was from 2 to 8 per cent. of attacks. Sudden death from cardiac and pulmonary complications sometimes took place.

DIAGNOSIS.—The only disease with which a dropsy of this kind occurring epidemically might be confounded is beriberi; but both epidemiology and symptomatology present numerous and striking points of difference. The bacteriology of neither disease has as yet been worked out.

TREATMENT.—Diaphoretics, diuretics, cardiac stimulants, and in later stages chalybeates and tonics, with careful dieting and nursing, were found to be the most satisfactory means of treatment.

KENNETH MACLEOD.

EPIDEMICS, Occurrence of.—See PERIODICITY IN DISEASE.

EPIDIDYMITIS (ἐπί, upon; and δίδυμος, a testicle).—Inflammation of the epididymis. See TESTES, Diseases of.

EPIGASTRIC PAIN.—The structures which normally occupy the epigastrium or 'pit of the stomach' are the greater part of the stomach, a small portion of the liver, and more deeply a part of the pancreas, the aorta giving off the coeliac axis and superior mesenteric branch, the inferior vena cava, the veins forming the commencement of the portal vein, the receptaculum chyli, and the solar plexus.

Abnormal sensations specially referred to the epigastrium are generally associated with disorders of the stomach (see STOMACH, Diseases of). Less frequently they may be associated with disorders of the abdominal wall and other parts just enumerated. Not infrequently they are accompanied by superficial or deep tenderness. An unpleasant sensation at the pit of the stomach accompanies nausea; while violent vomiting or retching causes considerable pain or aching in this region. Sometimes the pain is situated deeply, or shoots towards the back. This may depend upon disease affecting the posterior wall of the stomach, pancreatic disease, aneurysm, or other causes. A sensation of tension or dragging pain is sometimes experienced just below the ensiform cartilage in cases where the diaphragm is much displaced downwards, as from extreme emphysema, abundant accumulation of fluid or air in the pleura, or extensive pericardial effusion. A deep pain is not uncommonly referred to the epigastrium in cases of Addison's disease, and also in those of pernicious anæmia. Probably this is connected with the sympathetic plexuses. FREDERICK T. ROBERTS.

EPIGASTRIC PULSATION.—Pulsation is not uncommonly felt in the epigastrium. When situated at its upper part, just below the ensiform cartilage, it depends upon the heart, usually its right side, being due either to shortness of the sternum, displacement of the heart, or temporary distension or permanent enlargement of its right cavities. Very often a pulsation is felt and sometimes even seen, due to a pulsating aorta, especially in thin and anæmic subjects; and an impulse from this vessel may be transmitted through an enlarged pancreas, or through an abscess of the liver. Occasionally an impulse in the epigastrium is connected with an aneurysm, either of the aorta or of one of its branches. A pulsation in this region has also been attributed to regurgitation of blood from the right auricle into the inferior vena cava and hepatic veins, in cases of tricuspid incompetency. A murmur may sometimes be heard in the epigastrium. Usually this is a conducted cardiac murmur, but occasionally it depends on an aneurysm.

FREDERICK T. ROBERTS.

EPIGLOTTIS, Diseases of.—See LARYNX, Diseases of.

EPILEPSY (Greek, ἐπιληψία, ἐπίληψις, a seizure, from ἐπιλαμβάνειν, to seize upon).—**SYNON.**: Falling Evil, Falling Sickness; Lat. *Morbus sacer, morbus comitialis, morbus caducus*; Ger. *Fallsucht*; Fr. *Epilepsie*.

DEFINITION.—A disease characterised by recurring attacks of sudden brief disturbance of the cerebral functions, generally causing the patient to fall to the ground, whence the old English name. The effects of the disturbance of the brain vary. When intense it involves the motor centres and there is convulsion; when slighter there may be only a moment's unconsciousness, with or without a fall. In the slightest form there is only impairment of consciousness, without actual loss, but often attended by some sensory disturbance.

ÆTIOLOGY.—Epilepsy is a self-perpetuating disease, each attack leaving behind it a disposition to the occurrence of similar attacks, in consequence of the fact that all brain-activity has a residual effect, facilitating the recurrence of the same functional action. Hence, we must look to the exciting cause of the first fit for that of the disease. But often no exciting cause can be traced, and when an apparent cause can be ascertained it is often too trifling in character to be a sufficient explanation of so great a departure from the normal state. It is evident that the chief cause of the disease, in most cases, is an inherent disposition of the cerebral centres to the discharge of energy in abnormal form. The exciting cause, when one can be traced, acts only as does the spark which produces the explosion of gunpowder, the essential cause of which is the chemical composition of the explosive agent. The constitution of the brain is such as to make possible an instant energetic release of nerve-force in normal circumstances, and apparently a slight difference from the normal conditions permits its sudden liberation without adequate cause and in a form different from that which occurs in health. The conditions which predispose to the disease are therefore of chief importance in its causation.

Sex.—Females suffer rather more frequently than males, the proportion being as 13 to 12.

Heredity.—Nearly one half of the cases of epilepsy

in which the family history can be properly ascertained are found to present epilepsy or insanity in antecedents or collaterals. Other diseases of the nervous or general system are not met with in sufficient frequency to justify us in attaching weight to them as indications of a predisposition. But epilepsy and insanity seem to be interchangeable, and are met with in such frequency (considering that ascertainable family disease always falls short of the truth) as to justify the inference that in more than half the cases the disease is the result of a congenital disposition which is transmitted through the parents. Females present hereditary taint more frequently than males, the difference between them being about 6 per cent. Inheritance is more frequently from the mother's side than from the father's, but in the former females are in considerable excess of males, and in the latter case males constitute a considerable majority. Occasionally the number of members of a family who have suffered is large. Instances of five or six cases are not very rare, and the writer has met with eleven and even fourteen cases in one family. Hysteria and neuralgia do not occur with sufficient frequency in the families of epileptics, compared with others, to make them significant. Nor is there evidence that any morbid constitutional state, outside the nervous system, involves a disposition to epilepsy. The influence of syphilis seems to be exerted solely through the production of organic disease of the brain, and most cases of recurring convulsions due to it are thus produced. In a few cases of inherited syphilis the hindered general development has seemed to induce the disease.

Age.—Three quarters of the cases of epilepsy begin under twenty years of age; more than one quarter under ten; and nearly one half between ten and twenty. Only 15 per cent. begin between twenty and thirty, and only 6 per cent in the next ten years, and the percentage progressively lessens in each succeeding decade. No less than a quarter of the cases commence during the four years, twelve to sixteen inclusive. The excess of females is greatest in infancy and at puberty, but is distinct until thirty years of age, after which males are in excess.

Heredity has not much influence on the age at which epilepsy commences. The cases in which it can be traced are about 3 per cent. less between twenty and thirty, and 10 per cent. less in the cases over forty, than in those under twenty, but they occur up to quite late life.

Exciting Causes.—A reasonable exciting cause can be traced only in less than half the cases, rather more frequently in males than in females, but with equal frequency in the two sexes under ten.

Infantile Causes.—The cases commencing in infancy fall into three classes as regards causation. 1. In the majority of the cases the fits begin as 'dention convulsions,' as they are called, commencing as slight general attacks during the period of the first dentition and coinciding with the backwardness in teething and generally also in walking which indicates rickets. In some cases the attacks go on through childhood to adult life, increasing in severity; in others an interval of a few years occurs after the cessation of the infantile convulsions, which must be regarded nevertheless as exerting an influence disposing to the subsequent fits. In at least one tenth of the cases of epilepsy such a relation to these infantile convulsions, continuous or intermittent, can be traced. It is known that such

convulsions are really due to the effects produced upon the brain by the general retarding influence of rickets. The functional development of the lower reflex centres is more advanced than that of the higher cortical centres which should control them. The hindrance to development tells most on the centres which are least advanced, and so the effect is to induce an abnormal deficiency of control, a relative insubordination of the lower centres, which gives rise to the common phenomena of laryngismus stridulus and convulsions. This connection with rickets is of great importance because the latter is to a large extent preventable, and therefore a certain proportion of cases of epilepsy could be also prevented. These cases are distinguished by the slight and general character of the early convulsions. 2. In a much smaller class the initial convulsions in infancy are of great severity, often repeated, and either unilateral or beginning on one side, on which power is often lessened, sometimes considerably. In these cases the convulsions indicate the occurrence of a local cerebral lesion, due to injury, to local polio-encephalitis, to venous thrombosis, or even venous hæmorrhage, and often occur during conditions of general weakness, such as exhaustion from diarrhoea or an acute specific disease, or during whooping cough. Sometimes they come on in apparently healthy children during hot weather, like polio-myelitis. The convulsions often recur through life, but are distinguished by unilateral commencement, sometimes by limitation to one side, and often by traces of hemiplegia. These are cases of 'organic epilepsy.' 3. In a third class of cases there have generally been convulsions during the first two or three days of life, and birth was difficult, under conditions to make probable the existence of some cortical injury to the brain, such as hæmorrhage on the surface. Such cases are often attended by bilateral weakness or clumsiness of the limbs and by some mental defect.

Mental emotion.—Fright, some form of excitement, or intense anxiety, is alleged as the cause of the first fit in no less than one third of the cases in which the exciting cause can be ascertained. Of these, fright is by far the most common, a fact which is not surprising when we consider how potent is the influence of alarm on the motor centres of an animal, as manifested by the instant energetic movement to escape from danger, or the conspicuous tremor if no escape is possible. It seems to be more effective in females than males, especially after childhood. The first fit may occur immediately, or only after an interval of some hours or days. Hereditary predisposition often exists. Anxiety is effective chiefly in the second half of life, and is more frequently to be traced in males than females.

Traumatic causes.—A blow or fall on the head is not rarely the exciting cause of epilepsy which is apparently idiopathic, as well as of the organic form in which there are signs of local injury. Sometimes the first fit occurs immediately, more often after an interval which rarely exceeds a month. Exposure to the sun is a cause which is often alleged, but the cases are few in which it can reasonably be regarded as effective, and in most of these the exposure and first fit occurred in tropical climates.

Acute diseases may cause recurring convulsions by the mechanism of an organic lesion of the brain, probably thrombosis, of which such cases present

subsequent evidence. But in many cases the first convulsion, which led to the recurring attacks, occurred without any evidence of organic mischief, apparently from the influence of the toxic blood-state on the brain. In the majority of such cases the acute specific disease was scarlet fever, and this independently of dropsy or renal complications. This is a remarkable fact, taken in conjunction with the circumstance that scarlet fever may be followed by optic neuritis apart from any other cause than the blood-state. Influenza often intensifies the disease when it existed before, but it does not usually induce it.

Reflex causes are chiefly confined to the gastrointestinal system, e.g. an indigestible meal or the irritation of intestinal worms. Convulsions thus induced may recur and persist after the cessation of their exciting cause. In the cerebro-spinal nerves, irritation is very seldom effective. A few cases are on record in which a peripheral injury to a limb led to convulsions beginning in the part, but they are too rare to be of practical importance, especially when due account is taken of cases in which this apparent effect turned out to be only an excitant of convulsions really due to the influence of organic brain-disease.

Miscellaneous causes, which are met with in rare cases, need only be mentioned. Asphyxia is one which is occasionally distinct. Chronic alcoholism is certainly a cause of epilepsy, but presents the characteristic that the fits almost invariably follow a period of alcoholic excess. Other forms of blood-poisoning, from lead, from chronic renal disease, perhaps from tobacco, seem to be rare causes. Attacks are often ascribed to deranged menstruation; the common disturbance of the nervous system at the time of the 'periods' often induces an attack in persons otherwise predisposed, but there is no evidence that it has a definite causal relation to the disease. The frequency with which the disease commences at the time of puberty should be noted in this connection, because the hope constantly entertained that the disease, previously established, will cease when regular menstruation occurs, is wholly illusory.

SYMPTOMS.—The symptoms which manifest the paroxysmal disturbance of the brain differ very much in severity, and are variously designated 'fits,' 'attacks,' 'seizures,' 'faints,' 'turns,' 'sensations,' and, in America, 'spells.' The friends are sometimes anxious to conceal from the patient the nature of the disease, and carefully avoid the term 'fit,' which should therefore be avoided unless it is known that the patient is already familiar with it. The attacks may be divided into three classes, (1) severe, or 'major' attacks, the '*grand mal*' of the French, attended with tonic and clonic spasm; (2) slighter attacks, which may be termed medium, in which there is loss of consciousness and slight spasm, brief general rigidity, or local clonic spasm in cases of 'organic' epilepsy; (3) slight or minor attacks, the '*petit mal*' of the French, in which there is only loss of consciousness, and sometimes merely its impairment, attended by some characteristic sensation. In all forms with unconsciousness the loss may occur at the very onset, and the sufferer is then unaware of the attack until it is over. Often, however, consciousness is only lost after it has been influenced by the commencing process of 'discharge' in the brain, the perception of which constitutes the warning of the fit, or the

'aura' (a term dating from the time of the Greeks, when a vapour was supposed to ascend from the periphery and to produce the initial sensation).

Severe Attacks.—Precursory symptoms, or prodromata, are sometimes noted for a few days or hours before, sometimes consisting of the recurrence of the slightest form of minor attack, sometimes sudden starts, or a headache, or unnatural appetite, or some change in the aspect or manner which is recognised by the friends.

Warning.—The study of this is important because, when present, it indicates the part of the brain in which the process of discharge commences. In organic epilepsy, and in rare cases of idiopathic epilepsy in which there is local instability, the commencing fit produces spasm or sensation in one limb or a sensation referred to one of the special senses. In the attacks of the idiopathic form the sensations are more general in their character and relation, and are extremely variable. Some warning is present in more than half the cases of epilepsy.

Vertigo is one of the most common, and may consist in an apparent movement of the body or of external objects. But it should be observed that the term 'giddiness' is constantly employed by patients to designate a mere sense of mental confusion without any sensation corresponding to vertigo. The sensation, when definite, may be explained by the inequality of the discharge in the two hemispheres of the brain, since it may be accompanied or replaced by corresponding movement. A deviation of the head to one side is extremely common at the commencement of the fit, and sometimes occurs before the patient loses consciousness. It is occasionally accompanied by a visual aura, such as lights or objects before the eyes, which move towards the side to which the head turns, and the patient may seem compelled to follow them. The turning of the head may be attended by that of the whole body, and sometimes a patient will turn round two or three times before falling.

An epigastric sensation is another common warning, generally indescribable in character, and often ascending to the throat, where it causes a sense of choking, or to the head, and in either case quickly followed by loss of consciousness. Sometimes there is a sense of distinct nausea, and occasionally the sensation is actual pain at the epigastrium which usually remains there until consciousness is lost.

Cardiac and other sensations are less common. A sudden pain at the heart, or a sense of distress, or violent palpitation may be experienced. The sense of pulsation may become instantly general, or there may be a sudden feeling of a rushing from all parts of the frame upwards to the head, described as a 'rush of blood,' but purely sensory, without any evidence of vascular disturbance. Various other feelings of bilateral character are sometimes described, some passing upwards from the legs, others from the spine.

Cephalic sensations are not uncommon—sudden intense pain, a sudden sense of heaviness or fulness, and sometimes a peculiar sensation of something turning rapidly within the head which may be called 'cephalic vertigo.'

Psychical warnings are common, especially before minor attacks. It may be a sudden emotion, usually of fear, sometimes of horror, sometimes of intense consciousness of wrong-doing. In the cases in which fear is described, the patient may turn away or even

run away, before falling in the fit; and the same action is met with when there is no remembered warning. A peculiar ideational state is not uncommon, especially in slight attacks a feeling of being in a dream, often with an impression that the surroundings are unfamiliar, although really well-known, or a sensation of having been in the same circumstances at some preceding time. Rarely there is a sudden vague feeling of intense mental activity.

Special-sense warnings are also common. An olfactory aura is occasionally met with—a sudden momentary smell, generally unpleasant. A sense of taste proper is very rare, most of the warnings described as 'taste' being really of flavour, i.e. smell. In some cases of minor epilepsy there is smacking of the lips during the attack, sometimes following a conscious dreamy state, and Hughlings Jackson has described a case in which this was due to an organic lesion of the uncinatate convolution, to which the centre for smell is referred. Visual sensations are common. There may be an image of a ball of light or of sparks, often coloured, or a flash of light or colour. Yellow, red, and blue are the colours most commonly seen. Occasionally the sensation is more elaborate—a definite vision of a person or a scene. The object may seem to move towards one side, and the patient feel compelled to follow it, with resulting rotation of the head, both evidently due to an initial discharge in the opposite hemisphere. Loss of sight is an occasional aura, usually immediately followed by loss of consciousness. Less commonly there is an apparent diminution or increase in the size of objects actually seen, or of visionary spectra.

Auditory warnings are not infrequent. They vary extremely in character—hissing, rushing, whistling sounds, or sometimes a sudden explosive crash. The sound may be more elaborate, as that of bells ringing, and even a distinct musical note. Rarely there is a sound of spoken words. The sound heard may increase in loudness until consciousness is lost. More often, sounds actually heard seem to become fainter and inaudible, sometimes in association with corresponding dimness of sight. An auditory aura is often associated with vertigo, and with conscious deviation of the head, and even with a sense of compulsory rotation to the side to which the sound is referred—associations which are easily intelligible.

Convulsion.—In an attack of ordinary type there is at first general tonic spasm, fixing the limbs in an unnatural posture, the head turned to one side, the arms semi-flexed at the elbows, wrist-joints, and fingers, and the legs extended. The spasm, as it comes on, forces the air out of the thorax, and often simultaneously involves the larynx; a strange screaming cry results. The rigid tonic spasm is intense, and changes little during the time it lasts, half a minute or so, which always seems to the bystanders much longer. The fixation of the thorax arrests respiration, and the face becomes first congested and then livid. When the patient seems at the point of death, the tonic rigidity presents vibrations, at first so fine as only to be felt, afterwards greater and visible; and the change increases until remissions of spasm progress to intermissions, and the movements consist of violent general shock-like contractions, corresponding in seat with the chief tonic spasm. This form of convulsion effects an interchange of air in the lungs, and the cyanosis lessens, but saliva is frothed out between the lips,

often blood-stained in consequence of the tongue having been bitten. Occasionally the spasm is flexor in the lower limbs, and also in the arms. Rarely the arms are extended as well as the legs, usually in fits of moderate severity. The violence of the spasm may be so great as to cause subcoracoid dislocation of the shoulder. In rare cases the muscular contraction preponderates in the depressors of the lower jaw, and dislocation may occur. The spasm of the masseters may be so violent as to break the teeth. Biting of the tongue generally occurs during the clonic stage, the tongue being jerked towards the side most convulsed, and caught at the edge between the champing jaws, unless something is placed between the teeth to prevent their approximation. Rarely the tongue is bitten in 'medium' fits, which consist only of tonic spasm, being pushed to one side before the jaws are brought together. Various forms of convulsion are met with which deviate from the type, as for instance general fine tremor added to the tonic spasm, but not superseding it as does typical clonic spasm; prolonged tonic spasm, often with general flexion of the limbs, without succeeding clonic spasm; or general convulsion consisting only of clonic spasm. But all these atypical forms are rare in severe attacks.

The fits due to an old organic lesion commence by local spasm at the extremity of one limb, or in the face, which is generally clonic. It may remain local or become general, and in the latter case it usually changes, in severe attacks, to tonic spasm which runs the course already described. Occasionally it involves one side gradually, and ceases there when the other side becomes involved.

When the spasm is severe, the patient is usually unconscious and cannot afterwards remember the sensation which attended it. If the affection of consciousness is less, and a sensation can be afterwards recalled, it is usually greatly in excess of the spasm. When the arm is only slightly rigid and hanging by the side, it may feel as if it were twisted up and almost wrenched off. This is evidently the result of the cerebral process and not of the actual muscular contraction.

The pupils become quickly dilated in severe fits, but sometimes present afterwards a curious oscillatory contraction and dilatation. Reflex action is abolished after a severe fit for a minute or so; after this the deep reflexes are found to be excessive. In fits in which the head deviates strongly to one side, it is generally found that there is an excessive knee-jerk and foot-clonus on the side towards which the head turns, evidence of more intense convulsion on that side. Micturition is not uncommon during a fit, and is almost constant in some patients; in others it never occurs. This, together with the fact that the urine is often expelled with energy, suggests that it is not the simple result of unconsciousness, but is due to an extension of the spasm to the bladder. This is also true of the evacuation of the bowels, which is less common.

Vascular System.—The heart's action is seldom disturbed at the onset of an attack. Neither failure of the pulse nor pallor of the face has been perceived when an opportunity for observation has occurred. This is true of both slight and severe attacks. In several slight attacks the writer has been able to watch the condition of the retinal vessels, which was unchanged. As muscular spasm develops, the pulse becomes more frequent and strong, becoming feeble towards the close of the attack.

Excitation.—It is not common for epileptic attacks to be immediately induced by any exciting cause, but minor attacks are sometimes produced by a loud startling noise. A minor attack has even been known to be constantly excited by an unpleasant smell, or by sudden exposure to bright light. An instance of the latter was seen in a patient whose fits commenced by a blue light before the eyes, the result of initial discharge in the visual centre. In extremely rare cases, slight convulsive attacks may occur on first moving after rest, and so may minor attacks consisting of vertigo. In organic epilepsy there is sometimes a peculiar susceptibility to the attempt to obtain a 'deep reflex' (the knee-jerk, or foot-clonus) in the limb in which the attack begins. Anything resembling the induction of fits by stimulation of the skin, as in the guinea-pigs rendered epileptic by Brown-Séquard, is extremely rare in man.

Arrest.—The commencing fit may be arrested by a strong cutaneous impression. Thus in cases in which the convulsion, or a sensory aura, commences spontaneously in a limb, if a ligature can be promptly applied above the part in which the commencing fit is felt, the latter may cease at the ligature. A tape on the arm with a slip-knot often enables this to be speedily effected. The cutaneous impression seems to act on the sensory centres of the brain in such a manner as to cause the inhibition of the discharge in the related motor centre. The procedure (which has been employed for centuries) is effective even when the cause of the discharge is a tumour of the brain, a fact noted a hundred years ago, which precluded once for all the opinion that local commencement indicated a local peripheral cause of convulsion. The general convulsions of idiopathic epilepsy can seldom be arrested. Sometimes however they may be cut short by an energetic muscular movement, as by walking about the room, or by a strong sensory impression such as is caused by a mouthful of table salt, or even by 'smelling salts.' Energetic stimulation of the vascular system (as by nitrite of amyl) is also occasionally effective if the warning gives sufficient time.

Conditions after Attacks.—After a severe attack the patient usually goes to sleep for some hours, and may wake up well. He may quickly or slowly regain a normal state. If there is not sleep there is commonly severe headache for a time. For some hours after a convulsive fit the patient is feeble, and this muscular weakness is especially distinct when the attack has been unilateral. It then constitutes what has been called 'post-epileptic hemiplegia,' and is ascribed to the exhaustion of the nerve-elements by the discharge. Sometimes a similar weakness is met with when there is no convulsion, but only a sensory discharge, causing tingling in the limb. The loss of power is then transient and apparently due to inhibition of the motor centre by the discharge in the related sensory structures.

Vomiting sometimes follows a fit, immediately or after an interval. When it occurs before consciousness is regained it is a source of danger to life, because, if the patient is lying on his back, food brought up from the stomach may fall back into the larynx and cause suffocation. In such cases it is important to warn the friends always to place the patient on his side.

Automatic actions, having the aspect of volition, but evidently unconscious and not afterwards remembered, often follow slight or moderate attacks.

They are not seen after really severe attacks, which leave the whole system too exhausted for any manifestation of activity. A slight attack may be immediately followed by fumbling with the hands, undressing, or random remarks, which gradually cease as the patient regains a normal state. Sometimes the automatic action is more ordered and purposive. A patient may pocket any objects near him, and afterwards be astonished to find them in his possession. The remarks uttered are often such as to indicate a sense of resentment of what, no doubt, seems uncalled-for attention, and this tone is sometimes translated into action. A person who has assisted a patient out of the danger to which such an attack may have exposed him may be violently attacked. Other acts of violence occur; one woman threw her baby down a flight of stairs. Many crimes have certainly been committed in this post-epileptic stage. It seldom lasts long; after a quarter or half an hour the patient regains his usual condition. It occurs in only a minority of cases, but in some patients it is frequent, and naturally causes apprehension. It occasionally lasts longer, for several hours, but not often with violence. A carman, after a slight attack, drove his van from one end of London to the other, and found himself to his astonishment many miles from his destination. In these cases there is a peculiar mental state which cannot be afterwards revived. It is often difficult to believe that a patient is not in a natural mental condition except for some difference of manner which is recognised by those who know him.

Post-Epileptic Hysteria.—In patients who are of the age and sex which involve a tendency to hysteria, the post-epileptic action often takes the form of convulsions of hysterical character. There are the common co-ordinated movements, sometimes with opisthotonos, and wild action of the limbs, often a prolonged tremulous clonic movement; there is no apparent consciousness, but the aspect of the movements is such as might be caused by the will. They may continue for a quarter or half an hour or longer, unless cut short by the means which arrest such attacks, and are evidently due to the action of cerebral centres which are insubordinate in consequence of the loss of the control of the highest centres. Such hysteroid sequelæ are especially frequent after slight attacks of epilepsy, sometimes so slight as to be unnoticed. The patients who are subject to such recurring hysterical attacks, extending over many months or years, are generally found to have also slight attacks of minor epilepsy, without this sequela, and often to have had, at some time, epileptic attacks of more severe character. Those who are the subjects of this combined attack sometimes present pure hysteroid convulsions, which help to complicate the diagnosis. But if the probability of an epileptic element underlying the hysterical attacks be borne in mind, and the features of the seizures be carefully ascertained, there will seldom be real difficulty in the diagnosis. These attacks present the nearest approach met with in this country to the 'hystero-epilepsy' of the French. Here, pure hysterical attacks never present any close resemblance to the features of the true epileptic fit.

Turning on the face is an occasional post-epileptic action which is of considerable importance on account of its danger to life. It occurs when the fit is over, and cannot usually be associated with any tendency to turn at the onset of the attack. Its danger is due to the fact that it is common after

severe fits, as well as those of moderate severity, which occur during sleep, and that it involves the risk of suffocation—the post-epileptic coma being too profound to permit the asphyxia to rouse the sufferer. It may be safely said that, whenever a patient is found dead in bed with the face against the pillow, death has been due to epilepsy, although the existence of the disease may have been unknown.

Temperature.—A single fit has no appreciable effect on the temperature, but a succession, in the *status epilepticus*, may raise it several degrees, a symptom of serious significance. Under the same circumstances there may be considerable loss of body-weight. Minute extravasations in the face, from the vascular strain of severe tonic spasm, are sometimes produced, and occasionally a conjunctival vessel gives way. Hæmorrhages within the eye are unknown, and it is extremely rare for a fit to cause symptoms which suggest an intracranial extravasation on the surface of the brain.

Urine.—There is seldom any marked change in the urine after a fit. Even the amount of urea may not be notably increased. Albumen and sugar have been found for a short time in very rare cases—so rare as to be negligible.

Minor Attacks.—These differ very much in character. Many of their features have been already referred to, and only their chief characteristics need to be described.

The most common form is momentary loss of consciousness, which may return suddenly—so that even interrupted speech or action is continued—or gradually with some mental confusion, irrelevant remarks or brief fumbling movements of the hands. Sometimes this gradual recovery is attended with the automatic action, dangerous violence, or hysteroid manifestations which have been already described. When recovery is sudden the occurrence of the attack may be unknown to the patient, but when gradual there is usually some consciousness at least of the process of recovery. The unconsciousness may or may not be attended by muscular relaxation, causing the patient to fall to the ground. Thus, a gentleman, when dressing by his bath, had an attack which caused him to fall into the water, and as he did not recover consciousness until he had got out, he wondered to find himself wet. On the other hand, instead of muscular relaxation, the unconsciousness may be attended by slight tonic spasm, causing brief rigidity, and sometimes trifling quivering of the muscles. Such cases vary in severity, but as a rule approximate to the minor form of an attack. They have been referred to as ‘medium’ epilepsy, and it is convenient so to distinguish them, although they are not of sufficient importance to merit separate description. The tongue is not bitten in minor attacks, but urine is sometimes passed, especially in females.

The loss of consciousness in minor attacks is often sufficiently gradual to permit some sensation to attend it. It may present any of the varieties which have been mentioned as the warning of major attacks, but those which are most common are the epigastric sensation, a general sense of ‘rushing,’ or a sensation in the back, a sensation related to the special senses, either a discharge, or diminution of perception. Loss of sight often precedes a moment’s loss of consciousness. Smacking of the lips is common—although a remembered sensation of taste or of smell is comparatively rare.

The dreamy mental state already mentioned is common and is sometimes accompanied by movement of the lips and even by spitting, of which the patient remembers nothing. The attacks are often still slighter in character and consist only of momentary impairment of consciousness, often attended by some sensation. Of these, vertigo is common; it is brief, of moderate severity, and generally subjective only. Objective vertigo, a sense of movement in objects, is rare. It is most characteristic of an epileptic attack when it occurs at rest, when the patient is sitting still. But it may also occur when the patient is walking, and sometimes is caused by rising from the sitting posture. The epileptic nature of this might be doubted, but it occurs in those who are subject to severe attacks, and is similar to the vertigo which they experience when at rest.

The special sensations of minor epilepsy for the most part resemble those which attend severe attacks, but are less elaborate and consist only in a simple visual or auditory or olfactory sensation, or in simple loss of sight or of hearing. The two latter may be combined.

The variety of subjective sensations which attend minor attacks is too great for enumeration, and they are often almost too slight and brief for recognition. Before the sensation can be mentioned it has vanished. Such attacks are however as characteristic as those which are more pronounced. This trifling character is important because it prevents their occurrence being known to the friends, and they are often hardly noticed by the patient. It is necessary therefore to ask closely whether any strange momentary sensations of any character are felt. It is surprising how frequently these slight minor attacks can be ascertained, when the patient is thought to be absolutely free from them. Their importance is very great because, as long as they occur, there is a liability, almost amounting to a certainty, of the occurrence of severe attacks, if treatment is discontinued.

Automatic and hysteroid symptoms are common after minor attacks in some patients, while they never occur in others. Their features have been already described, but it may be mentioned how frequently slight automatic action, as fumbling with the hands, seems to be part of the attack. In some cases the patient, after a minor seizure, always utters some phrase. One patient always asked ‘What is the time?’ and took out his watch, never failing to do this—an indication of a sense of mental uncertainty. The occasional obscurity of the essential features of the minor attack is of special importance in connection with the cases in which the post-epileptic action takes the form of violence, because this may seem to an observer to be primary.

COMPLICATIONS.—*Mental Disturbance* is sometimes associated with epilepsy. It presents three important forms. The first is the paroxysmal excitement which immediately succeeds a seizure; it is usually slight or moderate in degree, often so slight as to be unnoticed, but it may amount to a condition of brief acute mania. A second form is more lasting mental derangement, usually of the nature of dementia, or subacute mania, which seems to replace attacks of epilepsy and ceases when these recur. A third form is a gradual mental failure supervening in cases of epilepsy of considerable duration, especially when the fits commence in early life. As a rule this mental

impairment is the result of imperfect cerebral nutrition, due to congenital influences of which the epilepsy is also a result, although it is often manifestly augmented by the effect of the fits on the brain. The practical relations of these forms are sufficiently obvious. It should be, however, mentioned that some mental dulness, mental depression, or loss of interest, or sometimes slight undue excitability, is frequently met with in cases in which epileptic fits are arrested, or considerably diminished, by treatment. It is apparently the result of slight disturbance of the cerebral action in consequence either of the agents employed or of the cessation of the sudden activity to which the brain has become accustomed and for which nerve-energy is elaborated. See EPILEPTIC INSANITY.

The Associations of epilepsy, apart from the mental disturbance just mentioned, are not numerous. Heart-disease may coincide with it, but, the wider the experience, the less evidence is there to connect the two. *Chorea* sometimes occurs in the subjects of epilepsy, and sometimes precedes the disease, but only in rare cases. The most significant relation between the two is presented by rare cases of chorea in which the choreic disturbance passes into a paroxysmal form which may be actually convulsive in character. The *thyroid gland* is not uncommonly enlarged in young women who suffer from epileptic fits, and may be temporarily increased in size after each attack. Increased frequency of the heart and increased general arterial pulsation are sometimes associated, but prominence of the eyeballs is very rare and the symptoms do not present a progressive character. *Megrim*, including paroxysmal headaches with or without sickness, and with or without sensory prodromata, often occurs in association with epilepsy. The common relation is that a patient has suffered from the headaches from early life, and that they become much less frequent or even cease when the epilepsy sets in. When the fits are arrested by treatment the headaches sometimes become more frequent. In rare cases the visual symptoms which precede the attack of megrim persist, in briefer form, as the warning of epileptic fits.

COURSE.—Epilepsy may begin by minor attacks occurring alone, sometimes during several years, which may then be succeeded by severe attacks. Or an initial severe attack may occur, followed after an interval of weeks or months by another, and by others at lessening intervals. In two-thirds of the cases in which a severe attack is the first symptom of the disease, another follows within a year. In developed epilepsy the interval between fits does not exceed a month in three quarters of the cases. An accurate monthly periodicity is often alleged, but seldom verified by careful observation. There is, however, an occasional tendency for the attacks to occur before or after the menstrual period, but this relation does not seem to be connected with any uterine derangement.

Status Epilepticus.—The fits of epilepsy are generally isolated, but sometimes occur in groups from two or three to ten or more, in the course of a few days, the groups being separated by intervals of weeks or months. Consciousness is usually recovered between the attacks, but sometimes a larger series of attacks occurs between which the patient has not time to regain consciousness. This constitutes the condition called 'status epilepticus.' The interval between the successive fits is so short

that the patient practically passes out of one into the other; the depression of the brain which results becomes more and more profound, and the condition is attended with considerable danger to life. The epileptic state may last for two or three days, with rapid loss of flesh, and a rise of temperature to 104°, 106° F., or even higher. The patient may die of exhaustion, or, the attacks ceasing, delirium may come on with other signs of meningitis, and death ensue. *Post mortem*, the chief changes found are alterations in the motor cells, and other signs of inflammation of the brain and membranes, both apparently secondary to the intense functional disturbance. The attacks may cease at any stage, and the patient gradually recover. It is difficult to discern any determining causes which induce the condition, except that it usually comes on when medicine is omitted which has failed to arrest the attacks and yet has apparently restrained them. Such cases emphasise the importance of not suddenly omitting treatment, even if the result of this is not altogether satisfactory.

Pregnancy and Parturition.—It is rare for epileptic women to have fits during child-birth. When one does occur it seems to involve no special danger. The influence of pregnancy differs very much. Many patients have no fits during pregnancy, and the immunity may continue through the period of lactation, to cease at its close. In other cases attacks continue but are less frequent. On the other hand the disease may begin during pregnancy, cease when it is over, to return during a subsequent pregnancy and afterwards persist.

Acute Febrile Diseases usually cause attacks to cease, and the patient remains free until the feverish period is over. But they return soon afterwards, and therefore, if treatment is discontinued, it should be resumed as soon as pyrexia ceases.

PATHOLOGY.—No pathological changes are known to be associated with idiopathic epilepsy. Those which are found after death in the status epilepticus are evidently secondary results of the severe functional disturbance of the brain. Alterations in certain parts, e.g. the cornu ammonis, have been thought to be related to the disease, but this has not been confirmed. The pathology of the disease has therefore to be inferred from facts of organic disease causing convulsions, and the features of commencing fits. The indications afforded by both classes of facts point to the cerebral cortex as the seat. Organic disease which causes convulsions is generally situated at the surface of the brain, and the initial symptoms of the fits correspond to the functions related to this region. Such convulsions are especially common when disease occupies the motor centres or their immediate proximity, and the attacks begin in the related part of the opposite side, but when severe may quickly become general. The subjective features of the attacks of idiopathic epilepsy have the same significance. The warning is often related to the special senses, and of a character which involves their origin in the highest centres. The psychical warnings have a similar import, and even vertigo is often so definite as to be susceptible of no other explanation than a simple inequality of the motor discharge on the two sides. Ever since Dr. Hughlings Jackson began his epoch-making studies of the modes of onset of attacks in their relation to organic disease, the conception of epilepsy as a disease of the cortex of the brain has been gaining ground until it is now dominant, the

older theories having disappeared in consequence of the absence of supporting facts, rather than by any positive disproof.

The minute scrutiny of the intimate processes in the nervous system presents us with many problems related to the nature of epilepsy, but they are for the most part beyond solution. The great fact of the disease is that there is an abnormal release of energy in the more severe attacks, abnormal in degree, in form, and especially in its occurrence without the usual exciting stimulus to which all normal action is due. With this we have to associate the fact that it is often due to some condition which is of congenital origin and often inherited from ancestors, while in other cases it is the result of the repetition of morbid activity which can be traced originally to an exciting cause. We have to ascribe it to the abnormal nutrition of the substance of the nerve-elements, whereby the tendency to activity is in excess of the restraint. This activity we must regard as the result of chemical processes occurring under the influence of life.

DIAGNOSIS.—Epilepsy presents many relations, and these involve corresponding diagnostic problems. Minor attacks are often mistaken for ordinary fainting, and the mistake is facilitated by the disinclination of friends to think they are more serious. But they are distinguished by their occurrence apart from the ordinary causes of syncope, especially when the patient is at rest, and exposed to no disturbing influences. They differ also in recurrence; no patient has a succession of ordinary faints through months, still less through years. They differ moreover in the suddenness of the loss of consciousness, and in its return being either quite sudden or through a transitional period of imperfect perception described as a 'dazed' condition, which is often attended by some automatic action. Moreover, a peculiar sensation of any kind, which is often constant at the onset of the attacks of minor epilepsy, never attends simple faints. These distinctions will generally suffice, especially if it be remembered that true faints are practically never mistaken for epilepsy, and that, if there is any room for doubt, the probability is great that the attacks are epileptic. Minor attacks of which vertigo is the subjective characteristic present other difficulties, because vertigo has many causes (see VERTIGO). The most important feature is a sense of mental obscurity in excess of that which would attend simple giddiness. Another point is the occurrence of epileptic vertigo when the patient is sitting still and quiet, but too much weight must not be laid on the converse. The greatest difficulty in diagnosis is presented by some cases of aural vertigo. In this the sense of giddiness usually continues for a time after the acute stage is over, the patient has other attacks of simple giddiness, and, on careful examination, signs can be found of labyrinthine disease—deafness, especially to audition through the bone, and tinnitus. But very sudden attacks may be attended by some impairment of consciousness. Aural vertigo is sometimes attended with vague unpleasant cephalic sensations, which may give rise to doubt; the subject of epileptic vertigo is generally free from such feelings.

The diagnosis of hysterical from epileptic attacks is sometimes a source of considerable difficulty. This is due to the frequency with which hysterical convulsion follows a minor attack of epilepsy, which may be so slight as to be unnoticed, or only seem

to be part of the hysterical fit. Such attacks, as already stated, are often termed 'hystero-epilepsy,' and they are the only form of attack at all corresponding to the name, which is met with in this country. But they are really epilepsy with an hysterical sequel. Any initial convulsion resembling epilepsy belongs to a true epileptic attack at the commencement. The continuous clonic spasm of the hysteroid attack bears no real resemblance to that of epilepsy. The difficulty chiefly arises from the common slightness of the initial epileptic attack which seems to run on into the symptoms of hysterical nature. Another important fact is that pure hysterical fits never go on during many months, still less during many years, at more or less regular intervals. It is also important to remember that tongue-biting and micturition are definite signs of an epileptic attack. Hysterical patients may bite the lip in a half-voluntary manner, but do not bite the tongue or the cheek. This is an important diagnostic sign, because its occurrence in only a few attacks is conclusive evidence that the essential disease is epilepsy. These facts assist a correct diagnosis, especially if it is remembered that patients with such duplex attacks almost always have, at other times, attacks of minor epilepsy pure and simple, and often have had such for many years before the onset of the complex attacks.

The distinction between organic and idiopathic epilepsy presents itself as a practical question, not in cases of active brain-disease, but only when there is reason to think that a slight lesion of the brain occurring years before has given rise to the convulsions. But the characteristic local onset is sometimes met with in idiopathic epilepsy, apparently from special instability at some one spot in the brain. In these cases the instability is sometimes bilateral, and the attacks begin for instance in the arm, sometimes on one side, sometimes on the other. Another point of weight is that in the idiopathic disease there is generally a family history of epilepsy. The distinction is chiefly of importance in regard to the question of operative treatment for the organic form, and because such acquired epilepsy has not the same tendency to be transmitted to children as the idiopathic inherited disease.

PROGNOSIS.—To forecast the future course of any given case of epilepsy is a task that is always difficult and often impossible. Of two cases apparently alike, one may yield to treatment quickly and permanently, and another may present no improvement however varied the measures employed. The shorter the duration of the disease, the better is the outlook, and *vice versa*, and the same is true of infrequency of attacks.

Heredity has not an unfavourable influence on the prognosis; arrest is obtained in as large a proportion of cases in which the disease is inherited as in others. Indeed, in the cases in which it is most distinctly acquired—those in which it has resulted from an organic lesion of the brain—the prognosis is distinctly worse than in others, the attacks being more difficult to arrest by treatment. In all cases it is true that there is no justification for anticipating spontaneous cessation of the attacks. It is often thought, in the case of girls, that the establishment of menstruation will have this effect, but the period of puberty is that at which epilepsy has most tendency to commence and least tendency to cease. When adult life has been attained, the disease does some-

times pass away, but so rarely that the effect cannot be looked for. On the contrary, many patients become epileptic in early life and remain so until old age. The prognosis is better the more the patient's conditions of life can be regulated, so as to secure freedom from the exciting causes of the attacks. But the great practical element in prognosis, which overshadows all others, is the influence of medicinal treatment on the disease. The cases are not rare in which appropriate treatment at once arrests all attacks, and if its influence is maintained for a sufficient time, it can be discontinued gradually, without recurrence. Unfortunately we are still without the means of distinguishing between the cases which will thus yield to treatment and those which will not. The observed effect of treatment is the only sure ground for prognosis.

The minor attacks of epilepsy resist treatment more than those attended by convulsion. When the latter are arrested, the former frequently persist and sometimes increase. The extent to which varied treatment has been adequately and perseveringly employed is also an important element in forming an opinion regarding the future. If it has failed, the prospect of a cure is small. Yet cases are often met with in which the past history would seem to justify only a gloomy forecast, but this is contradicted by the result of fresh measures. It must be remembered, moreover, that a patient's conditions of life may be altogether changed by improvement which falls far short of that which can be regarded as even an approximate cure.

TREATMENT.—In every case, the discovery and removal as far as possible of any causal influence are of primary importance. Intestinal irritation, apart from worms, may be effective, especially in children. Such substances as the skins of fruit, even of dried plums and currants, which are quite indigestible, should be avoided. The diet suitable in epilepsy depends more on its easily assimilable character than on its nature. Abstinence from meat has been recommended on theoretical grounds, but in a series of careful observations made by the writer on a number of patients, it was found that attacks were rather more numerous during the periods in which no meat was taken than in those in which it was given. Red meat may certainly be allowed once a day, but there is some reason to think that beef has sometimes a tendency to excite attacks and should be taken sparingly. Whether any alcoholic stimulant is taken habitually should depend on the state of the pulse and circulation. Most patients do not need one, and usually light wine should alone be allowed. When epileptic attacks have been excited by alcoholic excess, complete abstinence is essential. It is important that food should be eaten slowly; epileptics frequently have a tendency to eat fast. Those who are young often eat too much.

Constipation should be obviated by a daily laxative; a more natural condition is thus induced than if an aperient is taken only when obtrusively needed. Exercise of body and mind is desirable. Those who have passed the first half of childhood are the better for gentle mental work and not the worse for wisely moderated education, provided all pressure, as for examinations, is carefully avoided. At all ages mental and physical excitement should be avoided, especially hot rooms and crowded places; but it is often necessary to compromise and run a little risk, rather than distress a patient by too much exclusion from ordinary enjoyments. As part of

exercise, the question of cycling often presents itself. It is safe when patients have only nocturnal fits and no such minor attacks in the day as entail serious obscuration of consciousness; otherwise the risk of accident is too great. Some patients never have an attack of any kind when in active exercise.

While the correction of catamenial irregularity is desirable, its treatment has little influence on the course of the disease.

The question of marriage is often raised. In neither sex has marriage a definite influence on the disease. The tendency for children to suffer, sooner or later, is definite when epilepsy is due to inheritance and probably, but not certainly, when, without heredity, idiopathic epilepsy develops in early life, apart from a local lesion or conspicuous excitant. When fits are purely acquired, as the effect of injury or of some organic process, there is apparently no tendency to transmission. Even when the disease is inherited, the probabilities are against the affection of two or three children, but it is also against the escape of every one of six or more children. Of course a family history in both individuals who propose to marry involves a much stronger tendency to transmission, and such marriages should be deprecated in the interests of society and also of those concerned.

MEDICINAL TREATMENT.—The influence of bromides far transcends, in most cases, that of any other agent. The salt seems to undergo little decomposition in the system, and its effect apparently depends on its mere presence and some influence it exerts on the vital chemical action on which the discharge depends. This is more probable than that the bromine enters into the structure of the nerve-tissue, because the influence is so transient. Bromide has most influence on the motor structures: it frequently arrests entirely convulsive attacks, without influencing minor seizures, which consist in simple loss of consciousness. The various salts of bromine differ but little in their influence; what variation can be discerned seems difficult to associate with the amount of bromine, which is greater in the bromides of sodium and ammonium than in that of potassium, that is, if the official anhydrous salt be employed. The salt of strontium has been thought more effective, but its superiority is uncertain, and it is more poisonous than the bromide of potassium. Whatever is given, the dose or combination should be varied in the effort to find one which entirely prevents attacks. This should be continued, without intermission, until a habit of stability is established. Two years is the least in which this can be effected, and the gradual diminution of the dose should occupy another year. But as long as minor attacks persist, severe fits are sure to recur after cessation of treatment. Practitioners too often discontinue bromide prematurely on account of some slight inconvenience, or because they think it incompatible with the treatment of some incidental malady, which need only entail a temporary diminution. When attacks recur after premature discontinuance, it is usually more difficult again to arrest them. The daily dose should be 40 or 50 grains in two doses, or 60–90 in three doses. Unless this arrests the fits, it is seldom that attacks can be stopped by bromide only, and combinations are then more useful.

Bromide has two chief inconveniences: (1) Cerebral depression, or irritability, sometimes occurs, which may reach such a degree as to compel a dimi-

nution. It seems due to the repression of the tendency to discharge, for it may vanish after a severe fit, although the bromide is continued. In most cases it is only slight and of temporary inconvenience, passing away as the brain becomes accustomed to the new conditions. (2) Some patients are extremely prone to bromide-acne. In children an erythematous eruption may also occur, especially on the legs. The only preventive is the simultaneous administration of arsenic. Since this has been known, it is very rare to see the badly scarred faces so common before. The arsenic is generally well borne.

In many cases, bromide, while lessening the attacks, even so as to transform the patient's life, fails to arrest them; occasionally, it has little influence, and this, as already stated, is especially true of the minor attacks. It is often desirable to combine other agents with it, and sometimes to employ them alone. Bromide should rarely be suddenly discontinued. It may be effecting more than is apparent, and when stopped the patient may pass at once into a dangerous 'status epilepticus.'

Of adjuvant agents, one is borax, which the writer introduced in 1879. In inveterate cases, it is often conspicuously useful, but it seldom arrests attacks. From 5 to 15 grains may be given three times a day. The only inconveniences are transient dysenteric diarrhoea, and an eruption of psoriasis, soon ceasing if arsenic is administered. Zinc is an old remedy, especially useful for minor attacks. The bromide of zinc is not borne in adequate doses. The oxide, lactate, and citrate have been used; all are apt to cause nausea, but the oxide, 3 to 5 grains, in a coated pill, after food, answers best. Nitrate of silver, once in repute, cannot be given for long without discolouration of the skin, and patients were seen some years ago permanently stained and still epileptic.

Digitalis was a popular remedy for this disease in the West of England two hundred years ago. It certainly sometimes increases the influence of bromide, especially in nocturnal attacks, although it is of little use alone. This is also true of belladonna, which is most effective in minor seizures. These may sometimes be arrested by zinc and belladonna when bromide fails.

Hyoscine sometimes seems to augment the influence of bromide, and is conveniently given in the 1 per 1,000 solution of the hydrobromide, in a dose of two or three minims. The same is true of Indian hemp, which is useful in cases with much headache. Nitroglycerin has occasionally a marked effect, especially in cases associated with megrim, or in abnormal forms of minor attacks in young children. It is best given as Liquor Trinitrini in an acid mixture. To lessen depression from bromide, quinine or strychnine may be added, the mixture being acidified by hydrobromic or hydrochloric acid; or a grain of citrate of caffeine may be given with each dose. The use of opium as a preparation for a course of bromide has been advocated, but since practically abandoned. A careful trial in England many years ago showed that opium alone was useless.

The *status epilepticus* is seldom influenced by bromide unless its onset follows the omission of the medicine; which under these circumstances should be recommenced in full doses. Inhalations of chloroform sometimes arrest the fits. The hypodermic injection of morphine has been used with

success, but needs caution, on account of the danger that its effect may coincide with coma. Not more than $\frac{1}{12}$ of a grain should be given at once. This risk needs to be always kept in mind in the use of morphine by injection in epileptics. The effect of a severe fit soon after an injection may be fatal.

The most effective agent in the treatment of the *status* is hyoscine hydrobromate injected subcutaneously, $\frac{1}{200}$ gr. - $\frac{1}{100}$ gr., repeated every four or six hours for one or two days. If the temperature rises to above 104° F., it should be reduced by cold or iced baths, because the hyperpyrexia has been found to augment the destructive changes in the motor-cells of the cortex (Mott).

OPERATIVE TREATMENT.—The simple measure of the insertion of a seton in the neck was at one time often adopted, but has fallen into disuse. Yet it certainly had an influence in some cases, and deserves renewed trial. The only operation of practical importance is trephining the skull. Cases of active tumours do not come into the scope of this article, but the question often presents itself in cases of old cortical lesions, or in which the fits suggest such by their unilateral local commencement.

When there is reason to suspect depressed bone from injury, the operation should be performed. In other cases its advisability depends on the local limitation of the convulsions. When these frequently become general, and when (as is often the case) they have induced attacks of minor epilepsy, similar to those of the idiopathic disease, it is not likely that the removal of the damaged region will cause a permanent arrest of the attacks. It must be remembered also that the removal of the brain-tissue will cause for a time considerable palsy of the opposite side, and some of this is generally permanent. The remarkable effect of some operations for tumour has infused into the public mind an exaggerated and erroneous idea of what surgery can effect in these cases. Trephining has been employed in idiopathic epilepsy. It has even been known to arrest the attacks, just as a seton may do, but it is apparently only an energetic form of counter-irritation, with a prospect of success far too slender to justify its employment. W. R. GOWERS.

EPILEPTIC INSANITY.—SYNON. :—Fr. *Folie épileptique*; Ger. *Epileptisches Irresin*.

DEFINITION.—Mental disorder which is the outcome of the epileptic neurosis, and which is generally, though not invariably, accompanied by motor epilepsy.

ÆTIOLOGY.—The statements in the article EPILEPSY may be taken generally as applicable to epileptic insanity.

PATHOLOGY.—Our knowledge of the relation of epilepsy to structural lesion is still very imperfect; but it does not seem open to doubt that the changes associated with epileptic insanity have their seat in the cerebral cortex, and involve an altered nutrition of certain of the nerve-cells (see EPILEPSY). It seems probable that functional disturbance of the cells in the higher centres or psychic areas may be excited by radiation from the motor area in which an epileptic discharge originates; but in most cases there can be little doubt that the cells in the psychic areas are themselves the subject of structural change. In some cases, indeed, the psychic centres are the first affected, and the discharges may be, at least for a time, limited to these centres. In such cases the discharges are manifested by loss of consciousness,

hallucinations, or outbursts of mania unaccompanied by convulsions.

DESCRIPTION.—It is necessary to consider separately three aspects of the unsoundness of mind which is associated with epilepsy, according as it appears (1) as arrested or impeded mental development—*epileptic idiocy* and *epileptic imbecility*; (2) as progressive mental degeneration—*chronic epileptic insanity*; and (3) as transient mental disorder—*acute epileptic insanity* or *epileptic mania*. These three conditions are to be regarded as different phases and not as different kinds of epileptic insanity. They are intimately connected with one another, and occasionally they may all be illustrated in the progress of a single case. Thus epilepsy supervening during infancy or youth may so retard mental development as to produce imbecility; the subsequent periodical recurrence of epileptic fits during adult life may gradually pervert and deprave the already imperfect intelligence, causing it to pass through a phase characterised by emotional disorder, hallucinations, and delusions, and ending in the hebetude of complete dementia; this progress may occupy many years, and it may be interrupted by intercurrent outbursts of acute maniacal excitement. Though it is necessary, therefore, for the purpose of description to deal with each of the phases separately, their interdependence must not be overlooked.

1. Epileptic Idiocy and Epileptic Imbecility.—These terms are used to indicate different degrees of the same affection—a more or less complete arrest of mental development due to epilepsy. Those individuals in whom epilepsy supervenes in infancy, where there is no development of mind, or very little, are called idiots. Those in whom epilepsy commences after substantial progress has been made in mental growth, or in whom the epileptic seizures, though appearing early, do not recur frequently, and where there is considerable though defective mental development, are called imbeciles. The effect of the fits upon the mental condition of children differs greatly in different cases. The fits are usually followed by more or less mental retrogression. In many cases after a fit, and especially after a series of fits, the patients fall into a state of prostration and stupor of some duration. The educational training of the epileptic imbecile is seriously embarrassed by the occurrence of such attacks. A great part of what has previously been learnt seems to be wiped out by them, and requires to be learnt over again. In cases where the fits cease the evolution of the intellect may resume its course, but the mental organisation always remains damaged. It is estimated that about one third of the total number of cases of idiocy are due to epilepsy.

2. Chronic Epileptic Insanity.—In about two thirds of the cases of epilepsy there is an appreciable mental enfeeblement which gradually increases so long as the fits continue to recur. This progressive mental degeneration, though varying greatly in degree, is very uniform in type, and it is important to be able to recognise it. In the mildest cases there are usually to be detected lapses of memory, fits of absent-mindedness, and transient confusions of thought; and there is always more or less instability of mood and morbid irritability. The instability is the most characteristic mental feature of the epileptic. At one time he will be gloomy, despairing, conscious of his mental enfeeble-

ment, and lamenting its hopeless nature; at another time he will exhibit a surprising self-satisfaction, hopefulness, and confidence. Many epileptics are liable to fits of intensely passionate anger, occurring without apparent cause. The changes of mood often take place with a startling suddenness which is important as a diagnostic indication. In the fully developed chronic insanity of the epileptic the variableness of the mental condition may show itself in the intellectual as well as in the emotional state. A patient who usually exhibits confusion of thought, weak memory, and little power of attention may suddenly awake to a state of great mental activity, and show remarkable clearness and rapidity of thought, vivid memory, and great power of concentration. A common condition is to be habitually quarrelsome, argumentative, fond of teasing, insolent in demeanour, and given to capricious cruelty; yet patients will sometimes be found marvellously sweet in disposition, benevolent, affectionate, and considerate. In the advanced stages of the disorder the moral nature is generally deeply degraded, as shown in repulsive obsequiousness of demeanour, low cunning, treachery, and cruelty. The intellectual condition also passes through stages of degradation. Delusions of being injured by other persons fill the mind, and visual and auditory hallucinations occur. When these are present the patient, with his tendency to impulsive passion, is dangerous to persons near him. The last stage is marked by complete loss of moral sense and mental capacity. Nothing remains but the lower instincts in their most degraded form; and this is accompanied by a physical debasement which accords only too well with the mental condition.

3. Acute Epileptic Insanity—Epileptic Mania.—The term 'epileptic mania' is used to denote a peculiar automatic form of acute mania which sometimes follows epileptic seizures, and more rarely occurs in persons with the epileptic neurosis who have never had a convulsive attack—or at least who have not been known to have one. There is frequently some *transient mental perturbation* preceding or following a convulsive seizure which does not attain the intensity of mania, but which may be fitly alluded to here. The pre-paroxysmal phase of these perturbations may last for a few hours or a few days. They consist in a development of conditions which have been already described as characteristic of the early stage of chronic epileptic insanity. Sometimes there is morbid irritability, depression, or exaltation, sometimes confusion of thought or loss of memory. Immediately before the occurrence of a fit there is often some special idea which rivets the attention, the recollection of some particular scene, or a special hallucination such as a flame, a red colour, a spectre, or a peculiar odour. When there is a special idea, or special hallucination, it is usually the same which recurs at each seizure, and it generally has some relation to the circumstances in which the first fit occurred. After the convulsive fit has passed off, there is frequently a short period of confusion of thought, and lethargy. The condition known as *epileptic mania* is an outburst of maniacal excitement lasting from a few hours to several days. The general features which are distinctive of these attacks are the suddenness of their onset, the violence and abrupt rapidity of the acts of the patients, the painful and appalling nature of the ideas and hallucinations from which they suffer, the suddenness as well as

the completeness of the return to sanity, and the entire, or nearly entire, forgetfulness of what has happened during the course of the attacks. During an attack the impulsive acts may be of a suicidal but more frequently of a homicidal character, and this makes epileptic mania one of the most dangerous of all kinds of insanity. Sometimes the attack is characterised by furious maniacal excitement during its whole course, but there are also cases in which there is an apparent calmness of conduct. In a typical case of this kind the patient is irritable, suffers from a vague terror, seems impelled by a force, which he feels irresistible, to commit acts without conscious purpose. In this state the patients will walk long distances without object, steal or destroy articles in an unaccountable way, and will commit homicide or suicide with apparent deliberation. They seem insensible to everything which does not fall in with their dominant idea or impulse. This automatic action is a special characteristic of epileptic mania, both in the cases where there is violent excitement, and in those where there is an appearance of deliberate co-ordination of thought and act. The patients are as if in a waking dream; and when they awake suddenly to rational consciousness, they feel as if they had passed through a dream which they dimly conceive to have been of a distressing and frightful nature, but of whose details they have no recollection, or only a very vague one.

The relation of the mental disorder to the convulsive seizures is concisely given in the following sentences. When the fits are frequent, and continue through a course of years, they are generally associated with chronic epileptic insanity, though there are exceptional cases in which the mind is not seriously affected. When the fits are frequent, and are of the character of *petit mal*, the mental degeneration is usually more rapid and complete than when the motor symptoms are more severe, as in the *grand mal*. The calmer type of epileptic mania, called by Falret *petit mal intellectuel*, is most frequently associated with fits of the character of *petit mal* and with nocturnal fits; while the violent excitement, called by Falret *grand mal intellectuel*, is most frequently associated with the severe motor fits. Epileptic mania is most frequent after a fit or a series of fits, is less frequent before a fit, and least frequent in the intervening periods; it is specially apt to occur after a recrudescence of fits following a long period of freedom from them. It is also apt to occur after a rapid succession of abortive fits. It is rare in the first stages of epilepsy, most frequent in the middle stage, and rare after dementia has begun.

TREATMENT.—In the treatment of epileptic insanity everything which is useful in the treatment of epilepsy is to be included (*see* EPILEPSY). When pre-paroxysmal symptoms are observed, 20 grains of chloral hydrate should be given, and the patient put to bed. The same treatment is useful after a fit; and it may also arrest a fit, or series of fits, or ward off an attack of epileptic mania. Constipation and overloading of the stomach with food should be carefully avoided. There should be complete abstinence from alcohol; it is apt to bring on fits and also mania.

MEDICO-LEGAL RELATIONS.—Where responsibility for acts committed in what seems to have been an attack of epileptic mania is in question, the whole character of the mental symptoms and

the evidence of other epileptic conditions must be kept in view. The difficult cases are those in which there has been no violent excitement, and when the conduct has had the character of the calmer type of epileptic mania. Here it is important to bring clearly out the automatic character of the conduct. But the most important point is the existence of the fits. It may happen that the fits have not been recognised though they were really present. Nocturnal fits are the most likely to escape recognition. It must not be forgotten that they may have occurred without the knowledge of the patient himself. Careful inquiry should therefore be made for signs of their occurrence, such as biting of the tongue, of the lips, or of the cheek; spots of blood on the pillow; gathering of froth about the mouth on waking; unconscious emission of urine; peculiar fits of snoring; or the occurrence of numerous small ecchymoses under the skin of the neck or forehead, the last being a sign the importance of which was strongly insisted on by Trousseau. When no convulsive fits can be ascertained, evidence of vertigo, or of brief losses of consciousness occurring periodically or in series, should be looked for. If in the opinion of a medical witness an act of violence has been the result of epileptic insanity, it is his duty to show that not merely the act itself, but also the conduct and condition of the accused otherwise, are symptomatic of the epileptic condition. JOHN SIBBALD.

EPIPHORA (ἐπί, upon; and φέρω, I carry).—A flow of tears so persistent that they run down the cheek, due either to obstruction of the lacrymal duct or to excessive secretion.—*See* LACRYMAL APPARATUS, Diseases of.

EPISPADIAS (ἐπί, upon; and σπᾶω, I tear).—A malformation of the penis, in which the urethra opens on its upper surface.—*See* PENIS, Diseases of.

EPISPASTICS (ἐπί, upon; and σπᾶω, I draw). Substances which, when applied to the skin, are capable of producing a blister.—*See* COUNTER-IRRITANTS.

EPISTAXIS (ἐπί, upon; and στάζω, I drop). **SYNON.**: Fr. *Epistaxis*; Ger. *Nasenbluten*.

DEFINITION.—A flow of blood from the nose.

ÆTIOLOGY.—Epistaxis is a common phenomenon, occurring in at least fifteen per cent. of the population. It is more prevalent between the ages of ten and twenty-five, and after the age of fifty-five than at other ages; and is more frequent in men than in women. Blood may escape from the nasal vessels either by the rupture of an artery or by oozing from the veins and capillaries. If from an artery, traumatism and ulceration are the usual causes. If from veins or capillaries, either increased blood-pressure, or diminished resistance of the vessel-wall, or an alteration in the quality of the blood is responsible. Epistaxis may be described as primarily due to some local cause in the nose, and secondarily arising from an altered constitutional state.

The causes of epistaxis are (1) immediate, and (2) remote. (1) Of the immediate causes, traumatism is the most important. Under this heading may be grouped: blows on the nose, or fracture of the nasal bones; the introduction of foreign bodies, picking of the nose, and violent sneezing; fracture of the anterior or middle fossa of the skull,

and of the frontal sinuses. Among the various diseases of the nose giving rise to epistaxis are: hypertrophic rhinitis with separation of crusts, and ulceration and erosion of the mucous membrane due to tuberculosis, lupus, leprosy, syphilis, and diphtheria. Profuse hæmorrhage is frequently a symptom of new-growths, e.g. mucoid and fibroid polypi, angioma, sarcoma, and carcinoma. A sudden and profuse arterial hæmorrhage should always lead to a careful examination of the nasal and accessory cavities. A mild variety of epistaxis is met with in connection with adenoid growths of the nasopharynx.

(2) The remote causes are blood-diseases, such as leucæmia, scurvy, and purpura. Epistaxis often occurs in hæmophilia, and not infrequently in some of the infectious fevers, as in the early stages of enteric fever. Increased blood-pressure, whether due to excessive arterial tension or to venous obstruction, is relieved from time to time by profuse epistaxis. In renal disease, especially the cirrhotic variety with high arterial tension, hæmorrhage from various mucous membranes occurs, and profuse bleeding from the nose is not uncommon. Should a sudden epistaxis occur in an individual over thirty-five years of age without any local cause, a careful examination of the urine and heart should be made. When the venous circulation is interfered with, as in mitral stenosis and regurgitation, cirrhosis of the liver, emphysema, whooping cough, and tumours of the neck and abdomen, congested veins in the nose are especially prone to rupture. Acute yellow atrophy of the liver is characterised by hæmorrhages, especially from the nasal and buccal mucous membranes. If the atmospheric pressure be rapidly diminished, as in balloon-ascents, blood may pour from the nose. In both sexes at the age of puberty epistaxis is a common occurrence, and some authorities have spoken of nasal hæmorrhage occurring in girls at or preceding menstruation as vicarious in nature. Menstruation is also stated to have been replaced by epistaxis. But considerable caution should be exercised before accepting this explanation, since menstruation is often irregular and delayed without epistaxis resulting.

SYMPTOMS.—In people of plethoric habit and in those with high arterial tension, premonitory symptoms may be noticed, such as headache, giddiness, tinnitus aurium, pressure and fulness in the head. These symptoms disappear when the flow commences.

If the source be arterial, the blood is bright and the quantity profuse. Should the patient be in the recumbent position, blood may pass into the nasopharynx and escape from the other nostril, or may make its way into the larynx and be coughed up, thus simulating hæmoptysis, or be swallowed and afterwards be vomited, simulating in this way hæmatemesis. When a small quantity of blood is lost, in cases of high arterial tension or venous obstruction, the effect is a salutary one; but if the flow be very profuse and persistent, syncope may follow and blood enter the air-passages. When epistaxis is gradual and often repeated, anæmia results.

DIAGNOSIS.—The important point in severe cases is to determine the cause of bleeding and its origin. Careful anterior and posterior rhinoscopy is called for, and the bleeding points should be localised. To do this it is necessary to wash all clots out of the nose. The mucous membrane

around the bleeding point will then generally appear sodden and blood-stained. *See* NOSE, Examination of.

TREATMENT.—Epistaxis occurring in patients with high arterial tension and venous obstruction is a safety-valve. Provided that the loss of blood is not great and the flow ceases spontaneously, no interference is called for. If the loss of blood be continuous or considerable, remedial measures must be taken. If it be arterial hæmorrhage, a small plug of aseptic gauze is inserted into the nose, and digital pressure is applied externally. The application of an electric cautery is of value. If, despite these measures, the hæmorrhage continue, then the following means must be employed with all antiseptic precautions, for troublesome ozæna and pneumonia have occurred when these have been neglected, especially after plugging the nose. The simplest form of plug is Cooper Rose's tampon, which is a fiddle-shaped indiarubber bag capable of inflation after insertion into the nasal cavity. If this be not at hand, the nose may be packed with strips of aseptic gauze or a posterior nasal plug inserted by using a Bellocq's sound or a catheter and piece of silk. In no case should the plugs or strips be kept in for longer than twenty-four hours. They should then be removed, and the nostril if necessary repacked. Their removal often occasions considerable pain. Careful syringing of the nose with aseptic fluids for several days afterwards is essential, all force being carefully avoided. The indiscriminate injection of styptic fluids such as perchloride of iron is strongly to be deprecated. If used, they must be concentrated and carefully applied to the bleeding point. It is probable that the strictly local application of extract of suprarenal capsule will prove of service in epistaxis.

In slight cases the recumbent position and cold compresses to the nose, neck, and forehead will arrest the hæmorrhage; so, too, does the administration of half an ounce of vinegar in an ounce of water. Douching the face with cold water and raising the hands above the head is a favourite remedy. The successful treatment of epistaxis is mainly dependent upon an accurate diagnosis of its cause; and it frequently happens that nasal hæmorrhage is only a symptom of some constitutional condition calling for treatment.

A. H. TUBBY.

EPITHELIOMA (ἐπί, upon; and θηλή, the nipple, the word 'epithelium' being at first used for the tissue covering the nipple; and *oma*, a termination indicating a tumour).—**SYNON.**: Epithelial Cancer; Cancroid; Fr. *Épithélioma*; Ger. *Epithelkrebs*. *See* CANCER.

EPIZOA.—*See* ENTOZOA.

EPULIS (ἐπί, upon; and ὄδον, the gum).—*See* MOUTH, Diseases of.

EQUILIBRIUM.—This term is used to denote the equal balance of the different parts of the human frame, so that in sitting or standing or in the action of locomotion the trunk is kept in the erect position. Equilibrium in standing depends on keeping the centre of gravity of the whole body within the base-area of the feet, as any deviation outside this area will cause the whole body to fall over, unless the centre of gravity can be quickly restored. The muscles are the mechanism by which equilibrium

is preserved, and the muscles are kept informed of the position of the body by sensory impressions which come in from the periphery by the posterior nerve-roots, by visual impressions, and by impressions derived from the semicircular canals.

The muscles have been described as being in a state of tonic contraction, but this is certainly not true of all the muscles, and it is probable that there are two sets of muscles, one which fixes the joints, and the other which restrains and rectifies a faulty position. For instance, in the spinal column it is probable that the deep muscles, as the *spinalis dorsi*, the *semispinales colli et dorsi*, the *multifidus spinæ*, the *intertransversales* and *interspinales* act in a tonic manner in keeping the spine more or less rigid; but in the erect posture, where a person stands 'at attention,' the *erectores spinæ* and the *recti abdominis* are not contracted. These are the muscles which check and rectify any faulty position of the spine as a whole; for instance, if the spine is allowed to bend forwards, the *erectores spinæ* contract and stop the movement and bring the spine into the erect position again and then relax; also if the centre of gravity is allowed to fall behind the base line, so that the spine is extended backwards, the *recti abdominis* suddenly contract and regulate the movement and bring the spine into the erect position again. These are the muscles which counteract the influence of gravity, and in the erect posture the weight is so evenly adjusted that this position is maintained without either the *erectores spinæ* or *recti abdominis* contracting. So with the movements of the pelvis on the heads of the femora, it is probable, though it is difficult to prove it, that the deep muscles, as the *obturatorum*, *pyriformes*, and *quadrati*, act tonically in fixing the pelvis on the femora, but with regard to the large superficial muscles some act as restraining muscles. In the erect posture the *glutæus maximus* cannot be felt to be acting, neither can the hamstrings nor the extensor *quadriceps* and *sartorius*; but as soon as the pelvis and the trunk are allowed to sway forwards, the hamstrings instantly contract and counteract the effect of gravity, and if the trunk be allowed to incline backwards, the muscles on the front of the thigh contract and bring the body back to the erect posture. The extensors and flexors of the knee are not contracted in standing erect, as is well shown by the schoolboy trick of a smart blow behind the knees causing the person to fall down, owing to the extensors being relaxed—but they respectively contract if the person sways backwards or forwards. Coming down to the ankle, the muscles on the front of the leg and the calf-muscles are restraining muscles, and the tendons on the dorsum of the foot are not apparent unless the person sways backwards.

In disease equilibrium is lost from lesions on the sensory or motor side. (1) From loss of sensory impressions as in locomotor ataxy, where, the afferent tracts in the spinal cord being degenerated and the ingoing impressions retarded or stopped, the patient has only his vision and his semicircular canals to guide him; and therefore, when his eyes are closed, he sways to and fro, or falls over, owing to information not being given to the restraining muscles to act. Equilibrium may also be defective from faulty impressions of the relative position of the person to his surroundings due to disease of the semicircular canals, as in auditory vertigo. (2) On the motor side equilibrium is lost from a want of proper co-ordination of muscular movements, as in lesions of the

cerebellum and in cases of disseminated sclerosis; from paralysis of the restraining or fixing muscles, as in pseudo-hypertrophic paralysis or infantile paralysis (but it is extraordinary how much paralysis of the extensors of the knees may exist and yet the patient be able to stand and even walk); and from the slow action of the muscles, as in paralysis agitans.

Gait.—In relation to the preservation of equilibrium is the question of Gait. This may be altered from sensory or motor lesions. In sensory lesions of the spinal cord, as locomotor ataxy, the gait is characterised by an over-action and uncertainty of the movements of the legs, so that the feet are lifted too high, the toes are over-extended so that the soles of the feet are unduly visible, the heel is brought down with a stamp, and in turning round there is much staggering. On the motor side, the gait may be altered from simple paralysis of the different joints of the lower limb; in paralysis of the anterior tibial muscles the foot hangs down, and to clear the ground the hip has to be over-flexed, giving a high-stepping appearance; in paralysis of the extensors of the knee, the patient has to swing the leg forward by its weight, and in so doing the knee comes forward first, the foot is then planted on the ground, and the knee is thereby straightened and receives the weight of the body; while in paralysis of the *glutæus medius* and *minimus* the patient has a waddling gait from having to balance the weight of the body over the head of the femur as the other leg swings forward, owing to the fixation of the pelvis by these muscles being lost. Patients with weakness of the flexors and extensors of the hip-joints can manage to walk along the level, but they cannot go upstairs. If paralysis is combined with rigidity, as in spastic paraplegia or hemiplegia, the patient cannot raise the foot high enough to clear the ground, and it is therefore dragged along the ground or circumducted, or it is not lifted up high enough to prevent it scuffing. Among other forms of gait due to motor disablement are the inco-ordinate and cerebellar reel, in which the patient makes large excursions like a drunken man and falls over bodily without apparently any attempt to save himself; the slow gliding walk of the early stages of paralysis agitans, in which the patient seems to tumble on to each foot as it is put forward, and the short quick steps of the later stages of this disease, when he is unable, owing to the slowness of his movements, to get his feet in front of his centre of gravity. Lastly tremors such as those of disseminated sclerosis make the patient shake the whole body on each foot as it is put down; and the characteristic irregular gait of chorea, where the patient walks quickly, then suddenly stops, appearing to leave one leg behind, twists on one heel and then goes on again.

C. E. BEEVOR.

EQUINIA (*equus*, a horse).—A synonym for glanders. See GLANDERS.

ERB'S PARALYSIS.—See BRACHIAL PLEXUS, Lesions of.

ERETHISM (*ἐρεθίζω*, I irritate).—A condition of excitement or irritation, affecting either the whole system or a particular organ or tissue. The word has been especially applied to the condition of the body in the early stage of acute diseases, and also to that induced by the too free use of mercury (*mercurial erethism*). See MERCURY, Poisoning by.

ERGOTISM.—SYNON.: Morbus cerealis; Fr. *Ergotisme*; Ger. *Ergotismus*; *Kriebelkrankheit*.

DEFINITION.—A disease due to the action of ergot.

ÆTIOLOGY.—This disease derives its name from the fact that it is the result of the ingestion of ergot—the sclerotium of a fungus called *Claviceps purpurea*, which grows parasitically in the ear of the rye. In some seasons this form of blight affects the grain so extensively that ten per cent. of the meal may consist of ergot. The phenomena to be described as symptoms of ergotism have been regularly and exclusively traced to the use of articles of food made from rye-meal thus contaminated. The appearance and severity of the disease vary with the amount of ergot consumed. Children at the breast are never attacked. Ergotism has frequently broken out in well-marked epidemics, after unfavourable harvests. In ruder times it constituted a severe form of scourge; but now it usually occurs sporadically, or is limited to families or small communities.

ANATOMICAL CHARACTERS.—Ergotism is characterised by definite anatomical changes, mainly in connection with the vessels and the spinal cord. The arterioles are found *post mortem* to be contracted, and to be the seat of a peculiar hyaline degeneration of the intima, with thrombosis and diminution, or even complete disappearance or occlusion, of their lumen. Gangrene is the ultimate result of these morbid changes. The posterior columns of the spinal cord are the seat of a degenerative or sclerosing process, somewhat resembling the lesion found in tabes dorsalis.

SYMPTOMS.—Within a few days of the first ingestion of rye-meal contaminated with ergot, the ordinary phenomena of irritant poisoning are developed, namely, vomiting, diarrhoea, severe abdominal pains and cramps, and general depression—giddiness and headache being specially marked.

Along with the preceding, certain specific symptoms gradually make their appearance. The first and most characteristic of these is formication (a sensation as of insects running along the skin), attended by severe itching of the extremities. The other special senses, such as vision and hearing, may also become disordered. Occasional tearing pains occur in the limbs, and clonic spasms of the muscles. Ravenous hunger is said to be a striking symptom in some instances. The pulse is infrequent and small. Respiration is not markedly disturbed.

The remaining phenomena peculiar to ergotism are usually described as belonging to two forms, the *gangrenous* and the *spasmodic*, according as the circulation or the nervous system is chiefly affected.

(a) *Gangrenous ergotism.*—The toes, fingers, feet, ears, and nose are the parts most commonly attacked. The incipient discolouration, pain, and swelling are observed within a period of two days to three weeks from the commencement of the other toxic symptoms. See **GANGRENE**.

(b) *Spasmodic ergotism.*—The leading symptom of this form of the disease is the occurrence of severe intermittent cramps or painful spasms, specially affecting the lower extremities. These develop into tonic contraction of the muscles, with fixation of the limbs; and end perhaps in general convulsions, prostration, various kinds of mental derangement, unconsciousness, and death. Abortion does not appear to be of frequent occurrence.

COURSE, DURATION, and TERMINATIONS.—Many cases of ergotism are acute rather than chronic; but when gangrene appears, the course may be very protracted and variable. Spasmodic ergotism may last from two weeks to as many months. The mortality is said to have fallen from sixty to ten per cent. In a few cases, resolution occurs in affected extremities.

PATHOLOGY.—Beyond its effect as an irritant poison, the specific influence of ergot is mainly exerted upon the organs of circulation, the central nervous system, and the uterus. The different effects have now been satisfactorily traced to the actions of the four important organic constituents of the diseased grain. One of these, cornutine, an alkaloid, produces slowing of the cardiac rhythm by stimulation of the vagus at its central extremity. The contraction and hyaline change found in the arteries are due to the action, on the vaso-motor centre and the vascular walls respectively, of a second constituent, sphacelinic acid. The result of these effects on the heart and arteries is that the blood-pressure rises. The gangrene is readily accounted for by the vascular changes. The painful spasms, as well as the formication and other sensory disturbances, are the direct result of the action of the cornutine upon the spinal cord. The action of ergot upon the uterus is explained by some authorities as due to stimulation of the uterine centre in the cord by cornutine and sphacelinic acid. Other authorities maintain that it is set up by the local anæmia produced by the vascular contraction; while others, again, consider that ergot acts directly upon the muscular fibres of the organ.

DIAGNOSIS.—The occurrence of gangrene in a number of young and previously healthy persons, in countries where rye-meal is an article of food, should remove all difficulty from the diagnosis of ergotism. The spasmodic form of the disease can usually be distinguished from epidemic cerebro-spinal fever by the absence of pyrexia.

PROGNOSIS.—The prognosis depends chiefly upon the early recognition and removal of the cause. The probability of the escape of affected extremities may be estimated by the degree to which the gangrenous process has advanced.

TREATMENT.—The treatment of ergotism must commence with the removal of the cause. An attempt should then be made to hasten the elimination of the poison by the cautious administration of emetics and purgatives; to allay the symptoms of gastro-enteritis; and to support the strength of the patient by internal and external stimulants, such as alcohol, warmth, and friction. The treatment of less acute ergotism, including spasmodic cases, is mainly palliative. Efforts must be made to avert gangrene by careful local stimulation—by means, for example, of warm fomentations. If it make its appearance it should be treated surgically. See **GANGRENE**. J. MITCHELL BRUCE.

EROSION (*erodo*, I gnaw away).—A superficial destruction of tissue, caused especially by friction, pressure, corrosion, or certain forms of ulceration.

EROTOMANIA (*ἔρως*, love; and *μανία*, madness).—SYNON.: Love-melancholy; Satyriasis (in men); Nymphomania (in women); Fr. *Monomanie érotique*; Ger. *Liebeswuth*.—Insanity characterised by excessive sexual excitement; sometimes symptomatic of cerebral lesion, sometimes of disorder in the reproductive organs. See **INSANITY**.

ERRATIC (*erro*, I wander).—Wandering, shifting, or irregular. Applied to pains, eruptions on the skin, and other morbid phenomena, when they shift or move from place to place.

ERUCTION (*eructo*, I belch).—**DEFINITION.** The sudden escape or expulsion of gas from the stomach upwards, with or without an admixture of portions of liquid or solid food, or of gastric juice or other liquids. See FLATULENCE.

ERUPTION (*erumpo*, I burst forth).—This term is commonly applied to a pathological manifestation in the skin, more or less general; sometimes marked by colour, sometimes by prominence, but more frequently by both. When sudden and hyperæmic, a term derived from the efflorescence of a plant, namely, *exanthema*, is employed, as in the instance of the exanthematous eruptions—scarlet fever, rubeola, roseola, and variola. The term is equally applicable to less acute forms of dermatitis, such as urticaria, eczema, impetigo, ecthyma, acne, and furunculus; and is also used for still more chronic forms of disease, for example, psoriasis; and for outgrowths of the skin due to aberration of nutrition, as in the instances of warts and molluscous tumours. See DRUG-ERUPTIONS; OCCUPATION-DISEASES; and FEIGNED DISEASES.

ERYSIPELAS (*ἐρυθρός*, red; and *πέλλα*, skin).—**SYNON.**: Cutaneous Erysipelas; Fr. *Erysipèle*; Ger. *Erysipelas*.

DEFINITION AND DESCRIPTION.—A contagious disease of the skin, or mucous membrane, characterised by a spreading inflammation, which does not tend to suppurate, and which is associated with the presence, in the lymph-spaces and lymphatic vessels of the affected area, of a specific micro-organism, the *Streptococcus erysipelatis*. This definition is applicable to the simple cutaneous form of erysipelas, in which the nature of the infecting agent has been demonstrated, both in man and experimentally in animals. For this cutaneous affection the term 'erysipelas' should be reserved. It has however been extended, so as to include other inflammatory affections of a similar spreading nature, affecting different parts of the body, especially the subcutaneous tissue and the deeper planes of areolar tissue. Although in these situations the inflammation presents quite different characters, often running on to suppuration, yet these affections have for a long time been regarded clinically as erysipelas, and spoken of as erysipelatos inflammations. Hence some authors have described three or more varieties of erysipelas, the cutaneous, the subcutaneous or diffuse cellulitis, and the deep-seated inflammation of areolar tissue or phlegmonous erysipelas. Only the first of these will be discussed in the present article. The second and third are described in the articles on CELLULITIS and ABSCESS (*Diffuse Suppuration*) respectively.

ÆTIOLOGY AND PATHOLOGY.—It was not until 1882 that Fehleisen found in the lymph-spaces and lymphatic vessels of the skin, at the advancing edge of the inflammation in erysipelas, an organism growing in chains, or pairs, and belonging to the genus *Streptococcus*. He was able to grow the organism in pure culture on gelatine, and the inoculation of such a culture into the skin of the rabbit's ear produced an attack of typical erysipelas, the organism being again found in the inflammation so produced. Subsequent inoculations of the cultivated organism were made on the human subject, the

object being to influence the course of lupus, or cancer. Seven cases were thus tested by Fehleisen, and genuine cutaneous erysipelas followed in six; in the seventh case the patient had suffered from the disease a few months before. Other observations also tended to show that although one attack of the disease does confer a certain degree of immunity, yet it is only of a very limited duration, varying from one to three months, the patient afterwards showing in some few cases an increased tendency to contract the disease.

Clinically, in man, the incubation-period is from 24 hours to 3 days or more, while experimentally in animals Fehleisen found that the usual time was from 15 to 60 hours.

Thus, in the case of cutaneous erysipelas, the characteristics and life-history of the invading organism have been clearly established. Moreover, the identity of the so-called traumatic and idiopathic forms is now established.

In cellulitis and diffuse suppuration a streptococcus is also found, morphologically identical with that described by Fehleisen, and although differences in cultivation-characters were described by him, later observers agree that the organisms cannot be distinguished by reaction to reagents, or cultivation-characters.

With regard to inoculation-experiments, while Fehleisen noted differences in the appearance of the inflammation, according as the skin of the rabbit's ear had been inoculated with the *Streptococcus erysipelatis*, or that obtained from suppurating foci, later observers have failed to notice such differences. Lingelsheim and Fränkel claim to have produced typical erysipelas in the rabbit by the inoculation of a streptococcus derived from an abscess in man, and this observation has been confirmed by George Dean at the Jenner Institute, while Petrusky states that he has developed pure cutaneous erysipelas in man by the inoculation of a streptococcus derived from a case of suppurative peritonitis.

It was to the streptococcus found in these spreading subcutaneous inflammations that Rosenbach originally gave the name of *Streptococcus pyogenes*. Thus, while the question of the identity of this organism with that found in the cutaneous form of erysipelas is still an open one, it is well to remember that clinically the simple cutaneous disease differs widely from the subcutaneous inflammations.

Suppuration is generally absent in cutaneous erysipelas and frequent in the allied conditions. In cutaneous erysipelas streptococci are found only in the lymph-spaces and lymphatic vessels at the advancing margin of the rash, while in diffuse suppuration and cellulitis they can be demonstrated in the tissues and exudation-fluid of the whole of the inflamed area. In cutaneous erysipelas the organisms show little or no tendency to enter the blood-vessels locally, or to multiply in the blood-stream. An experiment of Fehleisen's on the rabbit's ear proves the absence of blood-infection. Having induced cutaneous erysipelas by the inoculation of a pure culture of the *Streptococcus erysipelatis*, he amputated the ear below the inflamed area, with Paquelin's cautery, with the effect of rapidly reducing the fever, and removing all constitutional symptoms. In cases of diffuse suppuration and cellulitis, on the other hand, blood-infection does occur, and pyæmia and septicæmia are not infrequent.

Further investigations are still needed to decide whether these differences in the behaviour of the

streptococci can be explained by varying degrees of virulence of one and the same organism, due to different sites of inoculation, and different degrees of resistance on the part of the invaded tissues, or whether the organisms in the several varieties of the inflammation are of distinct species. The suggested classification of the streptococci into long and short varieties has little or no value, owing to the easy conversion of one form into the other by suitable cultivation, and passage through different species of animals.

In the absence of any infection of the blood-stream by the organisms themselves in cutaneous erysipelas, the constitutional symptoms are due to the absorption, at the site of the inflamed area, of the products of the growth of the streptococcus. The writer has shown that in blood drawn from the inflamed area there is an increased tendency to the crystallisation of the hæmoglobin, as compared with blood drawn from the general circulation. This fact indicates increased local hæmolysis, probably the result of the action of the locally formed toxin on the tissue-cells and capillaries. Manfredi and Traversa have found that the products of the growth of the streptococcus outside the body, when injected into the circulation of animals, cause grave nervous phenomena, partly convulsive, partly paralytic in character.

While the *Streptococcus erysipelatis* does not ordinarily invade the blood-stream, it is possible that it may enter the circulation from the mucous surface of the respiratory, alimentary, or genital passages, possibly only after abrasion of the surface, and be carried by the blood-stream, without multiplying in it, to some part where from local injury, or other cause, it may find suitable conditions for growth. This seems the only possible explanation of those cases in which the disease occurs in different parts of the body without any wound. It is now, however, fully established that in the vast majority of cases of cutaneous erysipelas some abrasion of skin or mucous membrane serves as the point of inoculation.

Since the recognition of the specific organism of erysipelas, and the establishment of the local origin of the disease in the inflamed lymphatics of the skin, erysipelas is no longer classed among the acute specific fevers.

Predisposing Causes.—The causes which predispose to the occurrence of erysipelas are (1) those which favour the multiplication and dissemination of the organism outside the body, and (2) those which render the body a more suitable nidus for its growth.

1. Our knowledge of the life-history of the streptococcus outside the body is very incomplete. It grows readily at ordinary temperatures on gelatine and agar-agar. It has been found in potato-cultivations from the air of infected wards, the cultivations so obtained producing erysipelas in rabbits. There is little doubt, however, that it is usually conveyed, not by the air, but by direct contact, or by the hands or clothes of attendants.

All anti-hygienic surroundings, such as accumulation of decaying animal or vegetable matter, favour its growth. Thus the presence of a dust-heap near the window of a ward has been known to cause repeated outbreaks of erysipelas in the nearest bed. Dampness of floors, decomposing discharges from wounds, and dirty dressings all help the spread of the disease. Meteorological conditions are said to have an influence. Erysipelas is certainly most common in spring and late autumn, and least common

in the hotter and drier months. A wave of cold damp weather will sometimes be associated with an epidemic of the disease in the out-patient department of a hospital. Bad ventilation leads to a concentration of the poison, and hence to its easier dissemination.

2. Among the predisposing causes which render the patient more liable to take the disease, the most important is the presence of a wound or abrasion of skin or mucous membrane, which may serve as the point of inoculation. The existence of diabetes, chronic alcoholism, affections of the liver and of the kidneys undoubtedly render the subjects of such diseases less resistant to the growth of the organism. Personal hygiene is also influential, including bad or insufficient food, impure water, and lack of cleanliness. Age has apparently no influence, neither has sex.

A so-called constitutional predisposition to cutaneous erysipelas is occasionally met with, which is sometimes hereditary: the same patient suffers from repeated attacks of the disease, and so one attack is said to predispose to another. The pathological condition which underlies this peculiarity is not fully known. We have seen that the immunity conferred by one attack, experimentally produced, is only temporary, even in ordinary cases.

Erysipelas occasionally becomes epidemic, the type of the disease varying in different epidemics. We know little or nothing of the causes which render the disease epidemic.

MORBID ANATOMY.—The *post-mortem* appearances in erysipelas are not characteristic, and resemble those found in cases of other infective diseases associated with high fever. These are, in acute cases, early *post-mortem* staining of blood-vessels imperfect blood-coagulation, subserous petechæ, a swollen and soft spleen, and cloudy swelling of the tissues of the liver and kidneys. Locally, the bright red colouration of the inflamed area fades after death, leaving a faint yellowish tinge; the skin feels hard and inelastic; and the subcutaneous tissue contains an excess of serous fluid. Microscopical examination of the tissue of the affected area shows a large number of migrating leucocytes, lying in the spaces of the areolar tissue, among the fat-cells, and in the lumina of the smaller lymphatic vessels. The chains of streptococci found in the lymph-spaces of the skin at, or in front of, the advancing margin of the inflammation have been previously described.

The conditions associated with the various complications of erysipelas will also be present: thus a joint underlying the affected area will probably contain a turbid but not purulent effusion. The serous cavities are more rarely affected. In the case of a patient dying with pleural effusion during an attack of erysipelas following amputation of the breast on the same side, the turbid serum from the pleura contained large numbers of the *Streptococcus erysipelatis* in almost pure culture.

SYMPTOMS.—The incubation-period appears to vary from two to seven days. The constitutional symptoms sometimes precede the local. The invasion is marked by chilliness or rigor, or, in children, by convulsions, as well as by loss of appetite, nausea, headache, aching limbs, and general malaise—the usual symptoms of fever. If the process originates in a pre-existing wound, the patient is conscious of throbbing, tension, and discomfort in the part, and the granulating surface

assumes an unhealthy character. The temperature rapidly rises to 103° F. or higher, and usually within twenty-four hours the characteristic cutaneous inflammation becomes evident; this may, however, appear with the initial fever, or be delayed for two or three days. The nearest lymphatic glands are always swollen and tender.

The rash usually starts from some wound, abrasion, or granulating sore, frequently at the junction of the skin and mucous membrane, at the angle of the eye, or nose, or mouth, or from some place on the surface of the skin, excoriated by chronic purulent discharges from the ear or nose. It sometimes commences in the nasal or pharyngeal mucous membrane, and involves the skin secondarily. The inflamed skin is bright red in colour; the redness advances in all directions; and the advancing margin is sharply defined and slightly raised. The skin is swollen, and in situations where the subcutaneous areolar tissue is lax, as in the eyelid or scrotum, considerable œdema may occur. Frequently small vesicles arise, which may coalesce into large blebs, containing clear yellow, or occasionally blood-stained serum. When opened they dry up, scab over, and heal without ulceration, at any rate in the absence of further pyogenic infection.

The inflammation has but little tendency to run on into suppuration, except in parts such as the eyelid, where the œdema has been excessive and sufficient to produce local strangulation; and even here the question of a mixed infection must also be borne in mind. The fever continues as long as the redness keeps on spreading. The pulse is the best guide to the gravity of the case, and in serious attacks this soon becomes very frequent and feeble. The temperature may reach 106° F., and the diurnal variations are only slight.

Delirium is not uncommon. When present, it is due to the blood-change produced by the toxins of the disease, and not to meningitis, though this may occur rarely as a complication, especially in cases of erysipelas of the orbit, or when associated with fractures of the skull. The tongue is dry, foul, and in bad cases cracked and brown. There are sordes on the lips and teeth, and the urine frequently contains albumen.

The duration of erysipelas is very uncertain. The decline of the disease coincides with the cessation of the spread of the inflammation, together with a simultaneous fall of temperature. As the rash fades, the margin loses its distinct outline, and it frequently happens that the redness is spreading in one direction, and fading in another, at the same time. In some cases it may advance in a wave over the whole surface of the body and limbs, and though relapses frequently occur it would seem as though a partial local immunity of tissue is produced in certain areas of skin, over which the inflammation has passed, before a general immunity of the whole system has been attained. The rash, having appeared and thoroughly subsided in one area, does not as a rule reappear in that area again during the same attack of the disease, though the same area frequently becomes affected again by a subsequent attack, after the short period of immunity, which the disease confers, has passed away.

The subsidence of the rash is followed by desquamation of the cuticle, and frequently, in the scalp, by temporary loss of hair. When death occurs, it does so most frequently from exhaustion, or from grave blood-changes. It may be preceded by

delirium and coma, or by complications such as pleurisy or pneumonia.

When the disease attacks the mucous membrane of the pharynx or larynx, the constitutional symptoms resemble those of the cutaneous form, but the local symptoms present some peculiar features. The fauces and pharynx are bright red or dusky in colour, the soft palate and uvula are œdematous, and the lymphatic glands at the angle of the jaw markedly enlarged and tender. Extension of the disease to the glottis causes œdema with intense dyspnoea and liability to spasm of the larynx.

Erysipelas occasionally attacks the navel of newly born infants, and the vulva of weakly children; or the poison may infect the mucous membrane of the uterus or genital canal during the puerperal period, and thus produce a form of puerperal fever.

DIAGNOSIS.—When fully developed, erysipelas is easily recognised. In simple inflammation around a wound or abscess, the result of staphylococci or other infection, the characteristic invasion of erysipelas and the sharply defined margin are absent, while in simple erythema the temperature is not raised, and the eruption occurs in isolated patches.

PROGNOSIS.—This depends chiefly on the gravity of the general or constitutional symptoms. The pulse is, as a rule, a truer index of the patient's condition than the temperature. The following are bad signs:—A high temperature, violent delirium, excessive diarrhoea, early prostration, and a dry tongue with sordes. Old age, kidney-disease, and chronic alcoholism add largely to the risks of an attack. When the disease affects the larynx, the danger of the occurrence of œdema of the glottis is always present. In uncomplicated cases of erysipelas the death-rate is low.

TREATMENT.—To avoid the spread of the infection, the patient must be isolated—especially from other cases of wounds, and from puerperal women.

The treatment must be stimulating in character, in order to combat the exhausting effects of the disease. The bowels should be relieved at the commencement of the attack. One drug, Tincture of Perchloride of Iron in large and frequent doses, has been much used and has often been highly recommended, although in some cases it seems to exercise little influence on the spread of the inflammation. The diet must be as nourishing as possible. Beef-tea, eggs, and milk should be taken freely, while stimulants are often required, and occasionally must be given in large quantities.

Sero-Therapeutics.—Unfortunately the use of antitoxic serums in the treatment of streptococci infection does not at present rest on nearly so accurate an experimental or clinical basis as the use of an analogous serum in diphtheria.

The results of the injection of anti-streptococci serum vary. In some instances undoubted benefit is produced; in other cases little influence is noticed on the course of the disease. Unfortunately no separate statistics of the different forms of the disease are available. Some very severe cases of phlegmonous inflammation have been undoubtedly benefited, and some cases of puerperal fever show a rapid fall of temperature and recovery with its use. In some cases of diffuse inflammation of areolar tissue, or cellulitis, the use of the serum has apparently been followed by a localisation and limitation of a previously spreading inflammation, accompanied by a breaking down and suppuration of the central area.

It seems now established :—that a serum can be prepared which has a distinct antitoxic value when given to protect any animal against the poison of the particular strain of streptococcus used to immunise the animal from which the serum has been obtained ; but the same serum may be useless when used against a streptococcus of another race, which is morphologically identical, but which has been cultivated in a different soil. Hence, until the question of the identity of the streptococcus of cutaneous erysipelas and that found in diffuse cellulitis and puerperal fever (*Streptococcus pyogenes*) is experimentally settled, it is best to use, in each different form of the disease in man, a serum obtained from an animal immunised against the variety of organism associated with that particular form of the disease. Efforts are at present being made to produce experimentally a multivalent serum by immunising animals with several different strains. Unfortunately these distinct varieties of serums are not at present procurable by the practitioner. Further, from the marked differences shown by streptococci in their initial virulence, and also in their capacity for undergoing exalted virulence, by passage through different animals, it is possible that the slight physiological variation produced in the same streptococcus during its growth in any one animal may be sufficient to obliterate its racial peculiarity from an antitoxic point of view. If this be so, the varieties of antitoxic serums may be as numerous as the races of streptococci.

Thus in the absence of an accurate experimental knowledge, the treatment of erysipelas by anti-streptococcic serum, as at present prepared, is more or less an experiment. Fortunately the risks and disadvantages in case of failure are slight, while on the other hand the gain if the experiment succeeds is often considerable. Accurate records of the result in all cases are much to be desired.

LOCAL TREATMENT.—Warmth and avoidance of marked variations of temperature are essential. Cold is inadmissible ; it aggravates the inflammation, and tends to depress the tissues in their reaction against the poison of the disease. Hot fomentations or hot baths may be used if the situation of the part permit ; in other situations dry warmth may be used. The face and head may be enveloped in a mask of cotton wadding. Many local applications have been highly extolled in the treatment of erysipelas. These are :—painting the part with tincture of iodine, the application of antiseptic lotions or pastes, covering the affected area with a coat of white-lead paint, the local injection of a strong solution of carbolic acid around the margin of the inflamed area, and the scarification of the skin, and local application of the carbolic solution to the scarified surface. The drawing of a so-called limiting line in front of the advancing redness, with nitrate of silver or blistering fluid, has also been recommended. This procedure is based on the supposed blocking of the lymph-channels by leucocytes, in the area affected by the counter-irritation, thus rendering the vessels less pervious to the streptococcic invasion. Probably in actual practice the simplest measures are the best. The evacuation of the vesicles, and dusting the skin of the inflamed area with a non-irritating disinfecting powder, such as boric acid, oxide of zinc, and starch in equal parts, gives good results. The surface should be protected and kept warm by the wool-mask, and the limbs by dry wool or fomentations.

Erysipelas of the larynx and pharynx is best treated by an antiseptic and soothing lotion, applied locally, in the form of a spray. The Glycerinum acidi borici, diluted with water, forms a useful application ; or a solution of perchloride of iron may be locally applied. The constant sucking of ice is useful in relieving the distress, as well as the tendency to œdema of the glottis. If this complication occur, leeches may be applied externally, over the larynx. Should these measures fail and suffocation threaten, the aryteno-epiglottic folds may be carefully scarified, but tracheotomy will probably be necessary. C. J. BOND.

ERYSIPELAS, CURATIVE.—While attacks of intercurrent erysipelas have occasionally been known to exert a curative influence upon a number of diseases, notably lupus and syphilitic affections of the skin, such instances have been comparatively rare. The curative influence of erysipelas in tuberculosis was described at length by Mauriac as early as 1873. The similar influence of the erysipelas-toxins upon primary, secondary, and tertiary syphilitic lesions has been shown experimentally by Robert H. Greene of New York.

By far the most important, both from a clinical and experimental standpoint, is the action of erysipelas upon malignant tumours. Prior to Fehleisen's discovery of the specific micro-organism of erysipelas, in 1883, it was recognised, in a general way, that erysipelas had a certain beneficial action upon malignant tumours, and that, in some cases, this might be curative. These instances were, however, so few and isolated that no scientific attempts were made to determine the nature and limitations of this action. Fehleisen's experiments were the first step in this direction. His experiments upon animals, and later upon man, proved that erysipelas could be artificially produced by inoculations of the pure cultures of streptococcus of erysipelas. He inoculated 7 patients suffering from inoperable malignant tumours, but was able to produce erysipelas in only 6 cases. The results of these experiments were very striking. In one case, a fibro-sarcoma of the cheek with enlarged glands, the superficial nodules disappeared, and the enlarged glands diminished to one half their size. The other cases were inoperable carcinoma ; in one case, a twice-recurrent carcinoma of the breast, the tumour disappeared in one week, and there had been no recurrence at the end of six months. In a third case, an ulcerative carcinoma of the breast with enlarged glands, the latter disappeared entirely and the tumour was reduced in size one half. The remaining cases showed little change.

These remarkable results naturally led to further investigations, and Bruns in 1888 gave a detailed account of most of the cases up to that time. He discussed the effect of erysipelas not only upon carcinoma and sarcoma, but also upon lupus, keloid, and syphilitic lesions. He collected 14 cases of undoubted malignant disease in which erysipelas either occurred accidentally or was produced by inoculation. Of these 5 were sarcoma, 3 epithelioma, and 6 either carcinoma or sarcoma. Of the 5 cases of sarcoma 3 were fully and permanently cured. One of these cured cases was a multiple sarcoma of the head, and another a melanotic sarcoma of the breast.

In 1891 the writer of this article added 9 cases of sarcoma to the 5 collected by Bruns. In 3 of

these cases the erysipelas was artificially produced by the writer. The most important case was that of round-celled sarcoma of the neck with five recurrences in three years. At the last operation it was found impossible to remove the disease, and the condition was regarded as hopeless. Two weeks after the operation the patient had a severe attack of erysipelas, and shortly after this a second attack. The tumour quickly disappeared, and the patient regained his usual health, being alive and well seven years afterwards.

A study of this case led the writer to inoculate a series of cases with the streptococcus of erysipelas. The first of these was a recurrent spindle-celled sarcoma of the neck and tonsil. The tumour of the right tonsil was the size of a small hen's egg and almost completely blocked up the pharynx. The patient could swallow only liquids, and these with great difficulty; he was greatly emaciated. First agar-agar cultures were used by scarification without effect. Later hypodermic injections of fresh bouillon-cultures were made into the tumour of the neck. There was a rise of temperature to $100\cdot5^{\circ}$, associated with nausea, chilly sensations, and general malaise. The injections were repeated at intervals of three to four days for four months, and though no erysipelas was produced the rapidly growing tumour no longer increased, but showed some diminution in size. At the end of this time, by the use of a new and very virulent culture, a very severe attack of erysipelas was produced. Within two weeks the tumour of the neck had nearly disappeared, and the tumour of the tonsil had decreased in size two-thirds. The patient's general condition rapidly improved until he regained his normal health and strength. He was alive and well when last seen—five years later. The tumour of the tonsil had shown no tendency to increase in size and was apparently a mass of fibrous tissue, the malignant features having been destroyed. This method of treatment, namely repeated injections of living bouillon-cultures of *Streptococcus erysipelatis* introduced into the substance of the tumour, was employed in a series of 10 cases with the following results. In 2 cases of hopeless, inoperable recurrent sarcoma, the tumours disappeared, the patients remaining well upwards of three years after treatment. In 2 cases it was found impossible to produce erysipelas; in 2 cases death occurred as a result of the attack. The improvement which followed, even when no erysipelas was produced, led to the belief that a portion if not all of the curative influence might be due to the toxins rather than to the action of the germ itself. This view was still further confirmed by Spronck, of Utrecht, who reported 26 cases of carcinoma and sarcoma treated with a preparation of toxins of erysipelas prepared after the manner of Koch's tuberculin. Although Spronck did not obtain a single complete success, his results showed unmistakable evidence of the antagonistic and inhibitory influence of the toxins upon both sarcoma and carcinoma. Prior to the publication of Spronck's paper, the writer had experimented with bouillon-cultures of erysipelas heated to 100° C. Shortly afterwards experiments in a large number of cases, first with the erysipelas-toxins alone, and later in combination with the toxins of the *Bacillus prodigiosus*, furnished strong evidence that the curative action of the combined toxins was greatly superior to that of the erysipelas-toxins alone.

The preparation which has given best results is prepared in brief as follows: The *Streptococcus erysipelatis* is allowed to grow in bouillon for ten days to two weeks, and then the culture of *Bacillus prodigiosus* is added. The two cultures are then allowed to grow together for ten days, at the end of which time the germs are destroyed by heating to 58° C. The fluid is preserved in glass-stoppered bottles, a little thymol having been previously added. The preparation may be filtered by passing it through a Kitasato filter, which process makes it much weaker, the relative strength of the unfiltered to the filtered preparation being about 10:1. The filtered solution may be employed with greater safety in children or adults much reduced in strength. The toxins, to be of value, must be prepared from the most virulent cultures.

Since 1892 this method has been employed by the writer in upwards of 200 cases. Of 140 of these cases, 84 were round-celled sarcoma; 21 spindle-celled; 9 melanotic; 2 chondro-sarcoma; 12 sarcoma, diagnosed by microscopical examination, but the exact variety not stated; 6 inoperable tumours with a clinical diagnosis of sarcoma. Of the round-celled variety about 50 per cent. showed more or less improvement. In 2 cases only did the growth entirely disappear; in a third it nearly disappeared; a fourth was so much reduced in size that it was easily removed. All were well at the period of last observation—one to three and a half years after treatment. 10 out of the 21 spindle-celled cases disappeared entirely, and all the remainder showed marked improvement. Of the melanotic tumours no case showed more than slight improvement. In 24 cases partial or complete disappearance was observed; in 16 this was complete. Of these 8 were well three to six years; 9 from one and a half to three years; 4 from nine months to a year and a half; 3 died of recurrence which took place from seven months to three and a half years after treatment. Another case of very extensive spindle-celled sarcoma of the chest-wall after entire disappearance has just shown a recurrence on the opposite side, six years after treatment. These cases of recurrence demonstrate beyond question the correctness of the original diagnosis. Out of 35 cases of inoperable sarcoma reported by other surgeons and treated by the same method, in which the tumour completely or nearly disappeared, 10 were round-celled; 10 spindle-celled; 5 with the diagnosis of sarcoma resting on clinical signs only; 5 with the diagnosis based on clinical signs *plus* a history of recurrence after operation. Of these 35 cases, 26 disappeared completely; 2 others decreased so much that only an insignificant nodule was left which was easily excised, the patients remaining well from one to three years afterwards. Of the 10 cases of spindle-celled sarcoma 6 were well from two to nearly five years after treatment. Of the total of 35 cases 14 were well over two years; 6 over three years. In 2 cases death occurred during the third and fourth weeks of treatment, after the tumours (both round-celled sarcoma) had been entirely destroyed. In one case death was due to pyæmia of staphylococcal origin, the infection occurring at the site where the tumour had sloughed away. In the other case death resulted from septic absorption from too rapid necrosis of a very large round-celled sarcoma of the pelvis.

Risks of the Treatment.—With careful aseptic precautions and proper dosage the risks attending

the treatment are exceedingly small. The writer has had but two deaths in upwards of 200 cases, and these occurred five years ago. The initial dose should never be greater than one half-minim, and should be gradually increased according to the resulting reaction. It is not desirable to get a reaction-temperature higher than 102° or 103° F. If no improvement is apparent at the end of three to four weeks, the treatment should be discontinued. The toxins may be continued for years without injury to the patient, a fact that has been demonstrated in several cases.

Any explanation of the curative action of the streptococcus and its toxins upon sarcoma is extremely difficult, except on the theory that sarcoma is of micro-parasitic or infectious origin. The method is advised only in hopeless, inoperable cases of sarcoma, although there is reason to believe that repeated small doses could be used to advantage as a prophylactic measure after primary operations for carcinoma. The most promising field for the curative action of the toxins lies in their administration directly after all primary operations for sarcoma, instead of waiting for a recurrence.

If the toxins can, in a considerable number of cases, destroy and permanently cure large inoperable sarcomata, it is reasonable to suppose that their administration after operation will destroy the invisible portions left behind, and thus in a much larger number of cases prevent subsequent recurrence.

WILLIAM B. COLEY.

ERYTHEMA (ἐρύθημα, a flush or redness).—
SYNON.: Fr. *Erythème*; Ger. *Hautröthe*.

DEFINITION.—The term erythema is used in two senses:—(1) for a form of elementary lesion of the skin, characterised by dilatation of superficial blood-vessels and resulting redness or flushing; in this sense the word is synonymous with 'hyperæmia.' (2) as a quasi-generic term for certain conditions of the skin in which hyperæmia forms a prominent feature, e.g. *Erythema pernio*, *E. multiforme*, *E. nodosum*.

DESCRIPTION.—The redness produced by vascular dilatation is generally pinkish in colour; in very intense hyperæmia it may be dusky and bluish. The flush fades when pressure is made upon the skin, and reappears when it is removed. Erythema is thus distinguished from hæmorrhagic or purpuric eruptions. There is frequently an accompanying sensation of heat, and occasionally itching. Long-continued hyperæmia may result in desquamation of the skin; and other conditions, such as exudation of serum or of blood (*E. exudativum*), or formation of vesicles or wheals, may co-exist, but such phenomena are not erythematous in the exact sense of (1).

ÆTIOLOGY.—Dilatation of cutaneous vessels may be produced (1) by the action of vaso-dilator nerves upon the arterioles; (2) by paralysis of these vessels and of the capillaries. The former action may take place as a result either of reflex action in response to local irritation, or of stimulation of the nervous centres produced by general causes. Instances of the former are seen in the hyperæmia resulting from the application of heat, and of mechanical or chemical irritation to the surface: thus rubbing the skin produces redness (erythema), and the same effect may be due to a slight burn with a hot iron, to the action of the sun (*E. solare*) or of the X-Rays. Chemical irritants such as turpentine or mustard have a similar effect; and decomposing secretions in uncleanly persons are the cause of

cutaneous irritation (*E. intertrigo*), though in this case the process is seldom limited to a mere hyperæmia (see *INTERTRIGO*). General causes of erythema are seen at their simplest in ordinary flushing as the result of emotion: more permanent flushing of the face is often due to indigestion or to alcoholic excess. Among drugs which may cause erythematous eruptions are arsenic, belladonna, chloral hydrate, copaiba, cubebs, digitalis, iodides, boric acid, opium, quinine, salicylic acid, stramonium, strychnine, phenazone, and turpentine. The serum of other animals injected subcutaneously or into the vein often produces erythematous eruptions such as occur in treatment by antitoxin. The exanthemata of acute infective diseases are also erythematous in nature. Paralysis of the vessels of the skin due to fatigue after long-continued contraction is seen in cases of exposure to cold—frostbite and chilblains (*E. pernio*). The relations of erythema to inflammation of the skin (dermatitis) are difficult to define exactly. On the one hand many cases such as simple flushing are unquestionably non-inflammatory, and many of the toxic erythematous are equally 'simple' in nature. On the other hand erythema is often the first stage of inflammation, as in burns, and the association of processes such as exudation and hæmorrhage with the hyperæmia renders the distinction still more difficult. The more important cutaneous conditions forming the group to which the term 'erythema' is applied are (1) *Erythema multiforme*; (2) *Erythema nodosum*; (3) *Erythema induratum*; (4) *Erythema pernio*; (5) *Erythema ab igne*. There seems little reason to separate *Erythema nodosum* and *Erythema multiforme*; the former might well be included in the latter group of conditions; the last three affections are of an entirely different nature from the first two, and knowledge is rather hindered than advanced by grouping them with the erythematous. On the other hand the phenomena known as urticaria are closely allied to, if not identical with, those of *E. multiforme*.

Erythema multiforme.—SYNON.: *E. exudativum multiforme*; Polymorphous Erythema.—Under this name are grouped a number of conditions characterised by the existence of lesions in the skin consisting of localised reddish patches of varying shape, many of which subsequently develop exudation into the skin and subcutaneous tissue, and may give rise to bullæ or nodular enlargements. The affection runs a more or less definite course, and is often attended by symptoms of constitutional disturbance.

ÆTIOLOGY.—This eruption is most commonly seen in spring and autumn; it affects chiefly adolescents, and males are attacked more frequently than females. The exciting cause of the condition is probably the existence of some poisonous substance in the blood, due either to faulty ingesta or to auto-intoxication. Thus the erythema produced by the various poisons enumerated above is of the nature of erythema multiforme, as are also the rashes produced by antitoxins. The urticarial eruptions due to consumption of various kinds of molluscs and fish are also closely allied to this condition. In acute infective diseases, especially enteric fever, rashes of this nature may occur in addition to the typical eruptions, and the macular erythematous eruption met with in cases of septic absorption is well known. The administration of enemata is sometimes followed by erythematous eruptions, due probably to the absorption of poisonous products formed within the intestine owing to consti-

pation and resulting decomposition of the contents of the bowels. The condition known as peliosis rheumatica is closely allied to erythema multiforme, and the connection of erythema nodosum (*see below*) with rheumatism, though not beyond doubt, is generally accepted.

PATHOLOGY AND MOREID ANATOMY.—The condition consists especially in vaso-motor disturbances in the skin-area. The vessels in certain regions become dilated; exudation of fluid occurs; and occasionally blood-corpuscles escape into the tissues; numbers of escaped leucocytes are found lying along the vessels. The effused serum may accumulate in the interstices of the tissues, causing œdema, or may raise the upper layers of the skin and form vesicles. The latter may become purulent in some cases, either from secondary infection by pyogenic organisms or by emigration of leucocytes produced by the original poison. In some instances multiplication of the connective tissue-cells may occur, but it seems reasonable to consider that in such cases actual inflammation of the skin exists. When blood is effused, the usual changes take place in it (*see* EXTRAVASATION), and staining of the skin is seen like that well known in the later stages of an ordinary bruise.

CLINICAL CHARACTERS.—The eruption begins on the backs of the hands and feet, and frequently is found in these situations only. In some cases it extends upwards to the shoulders and hips, and in very rare cases is also found on the trunk. The lesions are frequently symmetrical in distribution. They consist in flattened papules, from the size of a pea to that of a bean, of a dark blue or brownish-red colour. They are surrounded on their first appearance by a red zone, which soon disappears, and the border of the papule then stands out in fuller relief. The mildest form of this disease consists in papules which disappear after a few days (*erythema papulatum*). Instead of thus disappearing the lesion may spread outwards from the edge, and flatten and become pale in the centre, thus forming a red ring, the condition being known as *erythema annulare*. While the first circle persists, a second ring may form around it, and thus two or more circles may co-exist, consisting of small papules or vesicles (*erythema iris*). Another stage may be reached by the enlarging circles meeting, and so forming segments of a circle, constituting the form known as *erythema gyratum vel marginatum*. In consequence of increased exudation erythema papulatum may assume the aspect of a wheal, forming *erythema urticatum* or *lichen urticatus*. This condition is attended with considerable itching; and, in consequence of scratching, the centre of the papular wheal is often covered by a minute bloody crust. The exudation may be sufficient to give the eruption the aspect of vesicles or bullæ, the so-called *erythema vesiculosum* and *erythema bullosum*.

At any of these stages the eruption may disappear. The sequelæ are slight pigmentation and desquamation.

The disease is accompanied by a slight feeling of burning, or by very slight itching. In the majority of cases constitutional symptoms are either absent, or are so insignificant as not to excite attention. There are, however, cases in which the symptoms are as marked as those which precede and accompany an eruptive fever. The prodromal symptoms are usually pain in the back, head, limbs, and joints, with gastric disturbance, and sometimes a congested

condition of the throat and fauces. In severe cases the prodromal period has been observed during four to six days; and among other symptoms, sneezing, bleeding at the nose, laryngitis, bronchitis, intense sweating, mental disturbance, and fever have been observed. All these symptoms may become aggravated during the period of eruption. In well-marked cases the disease may last from a fortnight to a month, but in exceptional circumstances the symptoms may be prolonged by fresh outbreaks of eruption. Simultaneously with the eruption on the skin, red spots may appear on the mouth and pharynx, from which the epithelium quickly disappears, leaving the parts raw and painful.

Hebra relates that in a woman who died while an eruption of *erythema gyratum* was on the skin, similar red rings were found in the small intestine.

Osler has recently given an analysis of 18 cases in which visceral lesions were associated with polymorphous erythema, in some of them serous or hæmorrhagic exudation being found in the walls of the stomach or bowels. In these cases gastrointestinal crises occurred, characterised by attacks of colic, either alone, or more frequently associated with vomiting and diarrhœa. Vomiting of blood and passage of blood in the stools were also met with.

Acute nephritis occurred in 6 of the cases, and chronic nephritis in one. There was hæmorrhage from the mucous surfaces in 6 of the cases, from the bowels in 5, from the nose and gums in one, from the stomach in 2, and from the kidneys in 2. There were cerebral symptoms in 2 cases, and pulmonary complications in one. In 4 cases purpura alone was noted. In the remaining 14 cases the lesions were characterised by erythema with exudation,—either urticaria or urticaria with purpura, acute circumscribed œdema, or the lesions of a typical erythema multiforme. Acute circumscribed œdema occurred in three cases, all in association with other exudative lesions. In 10 of the cases there were swellings of the joints or of the synovial sheaths or peri-articular tissues.

DIAGNOSIS.—In some cases the erythema is punctiform—*erythema scarlatiniforme*. It is distinguished from scarlet fever by the absence or very slight degree of fever. The fauces, although red, are not swollen. There is no characteristic appearance on the tongue; and the pulse is little affected. There are no sequelæ.

The mixed forms of the disease may simulate tinea circinata, but the absence of the spores of ringworm will make diagnosis possible. Seborrhœic eruptions may also assume the form of circular rings, but are generally more chronic in onset, and more scaly in character. Vesicular and pustular forms of eczema are distinguished by the weeping lesions which are nearly certain to be found co-existing in some parts, and the gradual onset and absence of constitutional disturbance. Limited lesions of vesicular character may closely simulate herpes; indeed, it is possible that the latter malady may be an allied condition.

PROGNOSIS.—In cases of erythema multiforme, occurring apart from recognised illnesses such as the acute specific fevers, the outlook is favourable, but relapses may occur. In the latter class of cases the course is that of the underlying disease.

TREATMENT.—If the cause of the attack be found to lie in the use of any drug or article of diet, this must be at once discontinued. The bowels are to

be kept acting freely, preferably by saline aperients, and such diaphoretic drugs as citrate of potassium may aid in eliminating toxic products by the kidneys. During the stage of fever and other constitutional symptoms, rest in bed is advisable. Intestinal disinfectants may be given in the form of salicylate of sodium or of bismuth, salol, and similar drugs. Local treatment must be directed to protecting the lesions from injury and subduing itching or pain. A simple dusting-powder of starch and oxide of zinc, rendered antiseptic by addition of boric acid, may be applied, and lead lotion or a weak solution of carbolic acid may be used as antipruritics.

Erythema nodosum.—This variety of erythema multiforme appears usually upon the legs, over the subcutaneous surface of the tibia. The condition may also affect the corresponding ulnar surface in the forearm, and other manifestations of erythema multiforme may co-exist in rare instances. The lesions consist of raised pink or reddish nodes, varying in size from that of a split pea to an area two inches in diameter. They are more or less abruptly elevated above the surrounding skin and are very tender on pressure.

The swellings are at first pale red with a yellowish tinge, later dark red, and finally livid; after they disappear they leave behind them a yellow pigmentation similar to that which follows a contusion. The number of swellings may vary from a very few on the lower extremities to successive crops on different parts of the limbs and trunk. In the latter case the feverish symptoms are well marked. The course of the disease is completed in from two to four weeks. The swellings never suppurate, never itch, are always painful, and the redness never spreads to the adjoining skin. This variety can occur in combination with the previously described forms of *erythema multiforme*. There is frequently a moderate degree of pyrexia along with loss of strength and appetite and a feeling of illness.

Erythema nodosum is found to be so frequently associated with rheumatic symptoms, such as arthritis, sour sweats, sore-throat, endocarditis, and even myocarditis, that it may be considered very probable that the affection is allied with the rheumatic diathesis. On the other hand septic conditions may present fever and joint-symptoms very similar to those of true rheumatism, and it is possible that erythema nodosum may be due to a distinct infective agent. Females are attacked very much more frequently than males, and the condition is rare after thirty.

A case has been reported in which phlebitis leading to embolism has complicated the disease.

In view of the possible connection with rheumatism, it is well to administer salicylate of sodium in these cases. The patient must be kept in bed, and warm fomentations may be applied to the shins, if the pain be severe. The bowels should be kept acting regularly. In after-treatment quinine and iron form a useful tonic.

Erythema induratum scrofulosorum (Bazin's disease).—See ERYTHEMA INDURATUM.

Erythema pernio, or Chilblain.—See CHILBLAIN.

Erythema ab igne (Ephelis ab igne).—This term is applied to the pigmentation met with on the skins of those who habitually sit with their legs exposed to the heat of the fire. The condition is not a true erythema except in its very first stage; later there is discolouration of the skin with the pro-

ducts of effused hæmoglobin. See SKIN, Pigmentary Diseases of.
G. THIN.

ERYTHEMA INDURATUM.—SYNON. : Bazin's Disease.—This term, unsuitable though it be, is retained to denote an eruption of not infrequent occurrence, characterised by the successive formation over months or years of indolent, painless nodosities, from a pea to a nut in size, commencing in the hypoderm, and either undergoing regression with atrophy, or gradually involving the superficial layers of the skin in an inflammatory process and discharging the necrosed or scanty puriform contents through a small opening, or ulcerating. In the gradual implication of the overlying tissues with the accompanying livid discolouration these nodosities closely simulate syphilitic gummata. By confluence plaques of infiltration may be formed, or ulcers of irregular shape or polycyclical outline may result. The distribution is notable, for the sites of predilection are the legs, especially below the bellies of the gastrocnemius muscles, and the tissues may be more or less diffusely swollen. Occasionally the nodosities may be seen on the thighs and, it is said, elsewhere. Moreover smaller nodules may arise on the backs of the fingers. The eruption is bilateral, with, at any rate, few exceptions.

Other points to note are the 'age' and 'sex' incidence, for females are especially attacked, and young subjects in late childhood and adolescence. Males and even middle-aged people may however be affected, and in the latter case a history of recurrence or recrudescence may sometimes be obtained.

There is considerable clinical evidence of association with tubercular complications, such as suppurating lymph-glands in the neck, and the subjects of the affection are those often denominated 'lymphatic' with acro-asphyxia and swollen lips. See SKIN, Tuberculosis of.

Histologically, the nodosities have been shown by Thibierge and Ravaut and the writer to possess a structure very suggestive of tuberculosis, while experimental inoculation in guinea-pigs gives rise to tuberculosis.

DIAGNOSIS.—Erythema nodosum with its painful, tender, actively inflamed nodules arranged chiefly on the shins in the long axis of the limb, and with the accompanying constitutional disturbance and definite course, can easily be excluded. The so-called scrofulo-tuberculous gummata of children have not the peculiar regional distribution of *E. induratum*, and a younger age-incidence. Thrombotic infiltrations may give rise to doubt, but a real difficulty arises in the case of syphilitic gummata, which frequently arise in the hypoderm of the legs, though almost invariably on one leg only.

PROGNOSIS.—This is good, for though the affection is often rebellious, and some tubercular focus seems probable, the subjects do not deteriorate in health as a rule; while the local virus is attenuated and does not infect the neighbouring skin as in *Lupus vulgaris*.

TREATMENT.—This is somewhat unsatisfactory, and cases are apt to prove very obstinate. The nodules may subside under rest in the horizontal position with careful bandaging, compression, and massage, but, if the tubercular nature of the lesion is confirmed, surgical interference may be desirable. As the subjects are those prone to tuberculosis, suitable constitutional treatment is also called for.

T. COLCOTT FOX.

ERYTHRASMA (ἐρυθρός, red).—This is a chronic, very slowly developing, recurrent, epiphytic disease of the skin, characterised by a very slight erythematous inflammation, which usually occupies the cruro-scrotal or axillary regions, but is capable of wider generalisation; produced by the presence in the corneous layer of a remarkably minute fungus—the *Microsporon minutissimum*.

DESCRIPTION.—Erythrasma is little known, and generally confounded with eczema marginatum. It is fairly common in men, but occasions little discomfort; it is less frequent in women, in whom it may be found about the axillary or perigenital regions. It is not met with in children.

The patches are rounded, not marginate or sensibly raised, from $\frac{3}{4}$ inch to $1\frac{1}{2}$ inches or upwards in diameter, uniformly roughened, and coloured yellowish-red or brownish. The pruritus and inflammation are very slight.

There are abundant delicate mycelia and conidia present, forming a rich network round the cells. The fungus requires staining and high magnification for its demonstration.

TREATMENT.—All parasiticide agents, such as iodine, resorcin, salicylic acid, and chrysarobin, capable of causing exfoliation, succeed in curing erythrasma. Relapses are apt to occur unless treatment is long-continued.

T. COLCOTT FOX.

ERYTHROMELALGIA (ἐρυθρός, red; μέλος, limb; ἄλγος, pain).—**SYNON.**: The red neuralgia; Terminal neuritis.

DEFINITION.—A condition of acute vaso-dilatation with intense burning pain and local elevation of temperature, usually affecting the peripheral parts of one or more of the extremities; either immediately brought about or aggravated when the involved part hangs down or is exposed to heat, and ameliorated when the limb is raised or exposed to cold.

Association with other diseases.—Erythromelalgia may occur in persons who do not present at any time other signs of disease, and for this reason it must be considered as a distinct clinical and morbid entity. Not infrequently, however, it appears as one of the symptoms of various diseases of the central and peripheral nervous system, tabes dorsalis and multiple sclerosis being the conditions more commonly associated. It may occur also in cases of myelitis, syringo-myelia, peripheral neuritis and dementia. Symptoms of functional disease of the nervous system have been present in some cases. Rarely it has been met with in cases of Raynaud's disease, severe osteo-arthritis, and myxœdema.

ÆTIOLOGY.—As an idiopathic condition erythromelalgia occurs after early middle age, being rather more frequent in men than in women; it has occasionally occurred in children; heredity seems to play no part in its causation.

Of the factors which can be placed in relation to the disease as causes, general debility after illness, peculiarities of occupation, and injury occur most frequently. Among the general debilitating conditions which have immediately preceded the appearance of erythromelalgia must be mentioned anæmic states, the puerperium, prolonged diarrhoea, malaria, gonorrhœa, and excessive fatigue. Occupations which involve prolonged strain upon any of the extremities, especially when the latter are exposed to extremes of heat and cold, are important

actors in some cases. Thus, the upper extremities have been affected in washerwomen, metal-polishers and alkali-workers who work with the hands exposed to extremes of temperature; and the lower extremities have been affected in persons who work in the standing position with the feet in water; in locomotive-drivers who stand upon the hot foot-plate of the engine, and in seamen; in bakers, postmen, and waiters. In more than one case the constant use of the small hammer was followed by erythromelalgia in the hand used. The disease has immediately followed the frequent use of very cold baths. Several cases are on record in which injury to one extremity (bullet-wounds, blows, &c.) has been followed directly by erythromelalgia, confined to the injured extremity. Such causal factors as those just enumerated are, however, far from constant, and in about half the reported cases of erythromelalgia, no exciting agent, either remote or direct, has been found.

SYMPTOMS.—The onset of the disease is characterised by the occurrence of intense burning pain, often described as like the application of a strong galvanic current to the skin, in some part of the body, usually the peripheral part of one of the extremities. The pain, which usually ceases after a time with rest and the horizontal position, recurs with increasing severity at irregular intervals, which become shorter and shorter, when the limb is exerted or is held in the dependent position. Shortly after the onset of the pain, the characteristic vaso-motor phenomena appear. The skin of the affected region gradually assumes an intensely livid purple-red colour, swells a little, so that it may pit slightly on pressure, becomes tense and shining, and is burning to the touch. The superficial veins stand out prominently, the whole area can be felt to pulsate, and the surface-temperature is raised often as much as two degrees above that of a corresponding non-affected area. The surface-temperature of the erythromelalgic skin is lowered when the limb is raised, and increased when this is in the dependent position, contrasting strikingly with the temperature of a normal limb, which is raised in the elevated position and lowered in the dependent position. The skin is often very hyperæsthetic, and intensely tender points may be present. Elevation of the affected part always causes amelioration of the pain and lessening of the vascular dilatation, whereas these are both increased when the limb is allowed to hang down. In severe cases an attack may at once be produced when the limb is allowed to hang down, and when the lower extremities are affected the sufferer may be entirely unable to stand. During the attack the application of warmth to the limb increases the pain, while cold greatly relieves it. Proximity of the affected limb to the fire, or even the warmth of the bed-clothes at night, will frequently induce an attack, and for this reason patients with this disease suffer less in the winter and are in the habit of sleeping with the affected parts uncovered. Thus one patient, whose hands were attacked, always slept with her hands clasped on the pillow above her head, avoiding both warmth and the dependent position.

During the attack localised sweating of the limb is common. In cases of long duration trophic changes, thinning of the skin, with brittle 'reeded' nails, may occur. In the idiopathic cases there are no signs of nervous affection. Though the patients resent deep pressure over the affected area, the

nerve-trunks are not tender. Anæsthesia is never present, and the reflexes, both superficial and deep, are normal or exaggerated to the degree usually met with in any painful limb. Except for the pain, which movement greatly increases, mobility is not interfered with and the muscles respond normally to electricity. The pain and vascular disturbance pass off with rest and the recumbent position after a few hours, to return again when the affected limb is exerted or placed in the dependent position. Sometimes between the attacks there is marked pallor of the affected region, but in certain cases of long duration a condition of permanent vaso-dilatation exists. In a few cases the characteristic pain has been present in other regions besides those showing the typical vascular disturbance, and in other instances vascular disturbances simulating erythromelalgia have occurred without any accompanying or preceding pain.

DISTRIBUTION.—The disease at its onset affects the periphery of one limb and is usually confined to one particular part of the hand or foot; subsequently it may spread so as to involve the whole periphery of a limb, and one or all of the other extremities may become affected, and in rare cases patches of erythromelalgia may appear on the trunk, face, neck, and buccal mucous membranes. The limits of erythromelalgic regions are nearly always sharply marked and correspond neither with the distribution of nerve-roots nor nerve-trunks.

Where erythromelalgia occurs in the course of diseases of the central nervous system, its clinical aspect does not differ from that described above, except that when occurring in connection with *tabes dorsalis* and *syringomyelia* pain may be absent. In some cases of *tabes dorsalis* each erythromelalgic attack has appeared simultaneously with a gastric crisis.

COURSE.—The milder forms of this disease and a few of the severe cases tend to a spontaneous cure. Many cases, however, while aided for a time by treatment, remain unchanged for years or gradually become worse. Where it is a symptom of disease of the central nervous system, erythromelalgia lessens or disappears when the nervous malady becomes far advanced.

PATHOLOGY.—Very few cases of this disease have been examined pathologically. Lesions have been found in the nervous system by Edinger (degeneration of the posterior nerve-roots corresponding with the affected region), and by Weir Mitchell and Spiller (degeneration of the peripheral nerves of the affected region). In several other cases the peripheral nerves have been excised and found normal. Most cases that have been examined have shown marked thickening of the middle coats of the small arteries of the affected region, but it is probable that such arterial change is a secondary result of some lesion of the nervous system. This inference is borne out by the experiments of Fraenkel and others, and by the common occurrence of such arterio-sclerosis in connection with neuritis, section of nerve-trunks, and *polio-myelitis*. From the occurrence of erythromelalgia in diseases of the central nervous system; from the fact that it has been followed in some cases by symptoms of central nervous disease, and from the inverse analogy, and occasional combination with *Raynaud's* disease, it is highly probable that the lesion is one of the nervous system. Its origin as a result of local injury, and the results of Weir Mitchell

and Spiller's recent investigation, suggest the peripheral nerves as the seat of the lesion, but in several other cases the peripheral nerves have been found normal, and the condition does not accompany the various known clinical forms of nerve-injury or neuritis. The precedence of pain in the attack, the effect of peripheral stimulation in inducing attacks, and Edinger's pathological result suggest a reflex vaso-motor disturbance with abnormal action of the vaso-motor centres, while the occurrence of erythromelalgia in cases of dementia, and its occasional patchy distribution in various parts of the body, point to a widely spread change affecting the various local vaso-motor centres in the central nervous system.

The present state of knowledge tends to the conclusion that erythromelalgia depends, as does also *Raynaud's* disease, upon a condition of instability, over-excitability, and abnormal function in local vaso-motor centres, which may be brought about in a variety of ways, i.e. by peripheral irritation, peripheral and central nerve-lesions, debility, and strain.

TREATMENT.—In early cases complete and prolonged rest in the horizontal position is essential, with careful attention to improvement of the general state of health and nutrition. Electrical treatment is useless in all severe cases—massage invariably aggravates the condition, and should not be employed. Amputation has been resorted to in cases where fingers and toes have been affected, but without benefit. Excision of the nerves supplying the affected region cured one case, but in another case fatal gangrene ensued. During the paroxysms the local application of cold affords most relief; morphine and alcohol are to be avoided.

Most cases are little amenable to treatment, but, after all remedies have failed, some of the severe cases have shown a tendency to spontaneous recovery.

JAMES COLLIER.

ESCHAROTICS (ἐσχάρια, a slough).—SYNON. : *Fr. Escharotiques*; *Ger. Aetzmittel*.

DEFINITION.—Escharotics are substances that completely destroy the tissues to which they are applied, and produce a slough. They are distinguished from other caustics simply by the greater intensity of their action.

ENUMERATION.—The chief escharotics are: Red-hot metal, Sulphuric Acid, Nitric Acid, Caustic Potash, Chloride of Antimony, Chloride of Zinc, Acid Nitrate of Mercury, Bromine, Chromic Acid, and Lime. Weaker caustics are—Nitrate of Silver, Sulphate of Copper, Sulphate of Zinc, Iodine, Carbolic Acid, Arsenious Acid, Sulphide of Arsenic, and Exsiccated Alum.

ACTION.—Escharotics combine with the tissues and destroy them. Around the part thus killed inflammation is set up, and the part is separated as a slough. Besides their local action, these agents act reflexly on other parts of the body through the nerves of the region to which they are applied.

USES.—Escharotics are employed, first, to destroy the virus in, and the tissues around, a poisoned wound, and thus prevent the absorption of the poison—for example, in bites by snakes or rabid animals, or in cases of inoculation with syphilis, or with micro-organisms in dissection or *post-mortem* wounds. Secondly, they are used to destroy unhealthy tissue, such as exuberant granulations, and to remove excrescences and morbid growths,

as warts, condylomata, nævi, polypi, hæmorrhoids, and cancer. T. LAUDER BRUNTON.

ESSENTIAL PARALYSIS.—A synonym for infantile spinal paralysis.—*See* PARALYSIS, INFANTILE.

ETHER, Uses of.—*See* ANÆSTHETICS.

ETHER-HABIT.—*See* HABITS.

ETHMOIDAL SINUSES.—*See* NOSE, Diseases of.

EUSTACHIAN TUBE, Diseases of.—*See* EAR, Diseases of.

EUTHANASIA (εὖ, well; θάνατος, death).

DEFINITION.—The measures by which we alleviate or seek to remove the mental and physical distress which often attends the approach of death.

IMPORTANCE OF EUTHANASIA.—To procure euthanasia is of great moment to the patient, to the patient's friends, and to the medical practitioner.

It is the duty, and should be the constant aim, of every thoughtful practitioner to acquire the art of successfully soothing the death-bed of his patient. He will thereby be enabled, when this is necessary, to relieve him of much of the attendant suffering, and to prevent many troubles individually of smaller moment, but collectively the source of much discomfort or distress. The friends' anxiety will be lightened by the relief afforded to the patient. Their own efforts in this direction are frequently unsuccessful; indeed may sometimes be prejudicial. The comfort of the dying is very often interfered with, and actual suffering induced, by the interference of well-meaning but inexperienced friends or relatives. They will sometimes question the action of the practitioner respecting the employment of a measure of which he may wish to avail himself, or they will administer food and stimulants to the patient in quantities which may be excessive or deficient or given at improper intervals. Death may thus be very often accelerated, and not without discomfort.

THE MORBID STATES CALLING FOR EUTHANASIA.—I. *Mental Condition.*—Whenever possible, the painful duty of communicating to his patient the hopelessness of his case should be relegated to the friends of the patient, who are always to be informed of the approach of death, in order that due attention may be paid to the affairs of the sick person, both spiritual and worldly. The practitioner should not hastily take on himself to tell him of the inevitable result of his illness. The patient may or may not desire to know the truth, and when it is communicated to him it is certain to affect his peace of mind in the one or in the other direction. Some welcome the announcement, while others are filled with dread. But it is a fact that the majority of persons seriously ill feel less on such matters than they would have felt in health. With many patients, too, not to know the truth respecting the danger which they are in, is itself a form of unhappiness which militates against the establishment of euthanasia. The practitioner should therefore most carefully study the individual case and the character of his patient. On the one hand he will exercise the greatest care not falsely to conceal their situation from the dying; on the other hand, he must keep in mind that a fatal termination is not always *certain*, and that in many cases

the last chance of an over-sensitive patient may be destroyed by the prospect of problematical dangers.

2. *Bodily Conditions.*—The physical states which specially call for euthanasia are—pain; dyspnoea, cardiac distress; exhausting cough; laryngeal distress; difficult expectoration; restlessness and sleeplessness; thirst; hiccough; abdominal distension; dropsy; bedsores; and exhausting discharges.

The removal of *pain* is essential for the promotion of euthanasia, and no other remedy is so successful for this purpose as opium or morphine cautiously administered. It is an adjunct of the greatest value to other measures, and, moreover, successfully combats the exhaustion and sinking sensations which, in certain cases, are a source of indescribable distress. Its anodyne effect is enhanced by its cardiac action. Its administration, however, often leads us into much fresh difficulty, by causing a dry tongue and distressing thirst; by arresting the excretions; in other instances by increasing the reluctance to take sustenance; or the presence of kidney-disease may prove a serious contra-indication to its employment.

The question as to the patient remaining out of bed supported by pillows in a suitable chair may arise when *dyspnoea* and *flatulence* are urgent; and will demand much judgment and care. By rendering moist the air of the bedroom with the vapour of hot water in a bronchitis-kettle placed upon a fire (not over *gas* or *spirit-lamp* contrivances, which only tend to vitiate the atmosphere of the sick-chamber), we can often insure the relief of pulmonary distress. Uremic dyspnoea will call for the administration of nitroglycerin, nitrite of amyl, or sulphuric ether.

Painful cardiac palpitation, and its attendant distress, are often relieved by the administration of antispasmodics along with iodide of potassium, a belladonna-plaster at the same time being applied over the præcordia. Digitalis, strophanthus, strychnine, or some of the other cardiac tonics, may also be resorted to hypodermically or otherwise.

One of the most distressing symptoms in the dying is the distressing *cough* of phthisis, which greatly exhausts the patient. It may be combated by judicious nursing; by an acid linctus, with or without the addition of a little morphine; by a combination of respiratory sedatives; by the frequent administration of small quantities of alcoholic stimulants; by hot or cold liquid nourishment; or by the sipping of iced lemon and water through a straw or glass tube. The *laryngeal distress* which frequently attends the final stage of phthisis will best be relieved by morphine-insufflations, cocaine-sprays, or recourse may be had to rectal alimentation. The loud, noisy, gurgling râles met with in the last phases of bronchitis and other conditions will call for change of posture and stimulants, although they are more distressing to the friends than a source of discomfort to the patient (*see* STERTOR). When the attempts at *expectoration* are difficult, measures must be employed which possess the power of diminishing the viscosity of the expectoration, or of stimulating the respiratory centre and the respiratory movements. *See* EXPECTORANTS.

The *restlessness* and tossing of the limbs, irregular sighing, with coincident *sleeplessness*,—the incapacity to go to sleep and stay asleep—which so often attend the last days of fatal illnesses, may sometimes be relieved by attention to the weight of the bedclothes, which can be conveniently reduced by employing a

suitable cradle. The skilful and judicious arrangement of the pillows and attention to the posture of the dying patient constantly tax the ingenuity and resource of the attendants. The temperature and ventilation of the sick-room are also of great importance in this respect, closeness and over-heating being zealously prevented, as well as crowding of the sick-chamber with anxious friends. The feet and legs ought to be frequently examined, and if chilled they should be warmed by a carefully applied foot-warmer. The unpleasant effects of cold sweats will also call for attention. The catheter will remove another often overlooked cause of inquietude—a distended bladder. By these and similar measures we may be able to counteract the cause or causes of insomnia, and thus secure for our patient a few precious hours of sleep.

Thirst is often a most distressing symptom. It may best be relieved by ice, or by teaspoonfuls of iced water or acidulated, unsweetened, cold tea or coffee. But in this connection let it be understood that we must be careful to avoid the popular mistake of forcing down liquids in cases where life is evidently fast ebbing away. Such a practice necessarily embarrasses respiration, and we have likewise to remember that at this time the act of deglutition very often is in abeyance. Death is occasionally accelerated by injudicious attempts to raise the patient, open his mouth, and administer food.

In the management of the dying we have always to contend with an enfeebled digestion. The *diet* in such cases should be one easy of assimilation. It not infrequently happens that the food given may be excessive or deficient in quantity, or that it is administered at irregular intervals, thereby causing much distress to the patient. Success in this direction depends solely on the tact of the nurse. While in acute disease nothing conduces more to success than definite instructions as to the amount and kind of the food to be given, and the hours for its administration, it is impossible and inadvisable to observe these rigid rules in the feeding of the dying. *See* PEPTONISED FOOD.

In conjunction with nourishment we can often remove much of the suffering by the judicious selection and administration of *alcoholic stimulants*. If the patient tires of one form of stimulant, another should be tried, provided we are careful to use only such as are easily digested and assimilated, and to give them in divided quantities. A combination of beef-tea, Liquor Strychninæ Hydrochloratis, and brandy is often very acceptable, especially in bad cardiac cases. Another stimulant of extreme service is ether, which particularly alleviates painful attacks of spasmodic breathing.

A sinapism to the epigastrium in conjunction with the internal use of a little ether and brandy will frequently remove that sometimes distressing symptom—hiccough. *See* HICCUGH.

Distressing *abdominal distension* must be relieved by the employment of turpentine stupes and by enemata containing some antispasmodic. *See* ENEMA.

The treatment of dropsy, abdominal distension, bed-sores, and exhausting discharges is dealt with in other parts of this work.

In addition to the conditions which call for euthanasia, it is clear that the severe dietetic restrictions which may have been enforced in such cases as Bright's disease and diabetes mellitus, ought to be reconsidered and removed when it becomes apparent that the patient's condition is absolutely hopeless.

The wishes and suggestions of the patient may generally be acquiesced in, and they will prove a useful guide to the practitioner as to the line of action to pursue. It is often the wisest course to humour cravings, especially when kind and gentle persuasion has failed to get the patient to waive his desire for them.

In addition to the means already mentioned, it need hardly be said that we still have all those measures which may be summed up in the expression *perfect nursing*, which implies carrying out many details as well as exercising sympathy and tact—a nurse not overdrilled; not a mere automaton who thinks too much of her 'uniform' and of her 'duty,' nor one who fails to cultivate that power of sympathy with which every nurse should be more or less endowed. Mental pains which may far exceed in their distressing results any purely bodily ailment can thus be easily alleviated. JOHN HAROLD.

EVACUANTS (*evacuo*, I empty).—SYNON.: Fr. *Evacuants*; Ger. *Ausleerende Mittel*.

DEFINITION.—Medicines used to produce some evacuation from the body.

ENUMERATION.—The chief evacuants are: Sternutatories, Expectorants, Sialogogues, Emetics, Cholagogues, Purgatives, Diaphoretics, and Diuretics. *See* the several articles upon these subjects.

EVIAN-LES-BAINS, in Savoy, France.—Alkaline waters. *See* MINERAL WATERS.

EXACERBATION (*exacerbo*, I aggravate).—Increase in the severity of the symptoms of a disease.

EXANTHEMA or **EXANTHEM** (ἐξ, out; and ἀνθῆ, I blossom).—SYNON.: Fr. *Exanthème*; Ger. *Ausschlag*. This term, formerly applied to any cutaneous eruption, is now restricted to the characteristic eruption of those specific infectious diseases which present this phenomenon.

EXANTHEMAT.—The specific fevers attended by characteristic eruption or exanthem, as typhus and typhoid fevers, small-pox, varicella, measles, rubella, scarlet fever. The term, which does not correspond to any natural division of the specific infectious diseases, appears to be passing out of use.

EXCITANTS (*excito*, I excite).—*See* STIMULANTS.

EXCORIATION (*ex*, from; and *corium*, the skin).—The superficial destruction of a portion of the skin or of the mucous membrane.

EXERCISE (*exerceo*, I drive about).—In medicine, this word is used in its more general and popular sense to signify active use of the muscles of the body, or of the parts thus called into play. Exercise may thus be defined as the ordered movement of one or more muscles, and of the structures upon which those muscles act. General exercise calls into activity the majority of the muscles of the trunk and limbs, and produces a notable influence upon all the chief functions of the body. In *special* muscular exercise, the activity is limited to particular muscles or groups of muscles. Exercise may be *voluntary*—when it is undertaken at the will of the subject, as in the normal act of walking; or *involuntary*, as in the action of the muscular

fibres of the heart, of the bowels, or of the pupil. It may be *active*—when it occurs in direct response to the normal nervous stimulus, however evoked; or *passive*, as when it results from the application of forces extraneous to the body of the subject—as in various forms of massage and ‘movement-cure,’ and from the appropriate application of some forms of electrical energy. And here it may be pointed out that, although the involuntary muscular tissue which plays so important a part in maintaining the essential life-processes of the body is not within the direct control of the individual’s volition, yet there is probably no involuntary muscle which may not be exercised, i.e. provoked to functional activity more or less sustained, by means of stimuli indirectly furnished by an active use of voluntary muscles suitably selected and adjusted.

It is therefore obvious, even from these brief considerations, that muscular exercise stands in many important relations to medicine. It is essential to the preservation of health (see PERSONAL HEALTH); and is often associated with the causation of disease. In certain disorders and cachexias it is an effective—not rarely an essential—means of treatment (see MOVEMENT, Therapeutical Uses of). Again, exercise is often abused: negatively, by its neglect; and positively, by being practised in excess; and such abuse in either direction may entail serious and far-reaching consequences. It is with this aspect of the subject only, the Abuse of Muscular Exercise, that the present article will deal.

In the first place it must be remembered that, from the moment of birth, muscular exercise duly proportioned to the powers of the individual—increasing with bodily development up to the prime of life, and thereafter gradually diminishing in amount and in severity in accordance with the waning vitality of the senescent organism—and duly apportioned to nutrition and to appropriate periods of rest, is an essential of sound health. In the healthy body the principal physiological results of properly regulated exercise may be summarised as follows:—increase in the frequency and volume of the *respirations*, with greater elimination of carbon dioxide (produced chiefly by oxidation of fats) and increased absorption of oxygen; increase in the force and frequency of the *heart’s action* (by some twenty beats per minute), with a subsequent fall in the pulse-rate, on the discontinuance of exertion, to about ten beats below the normal rate of rest. *Arterial blood-pressure* is increased during exercise, and for a short time after its cessation. The excretion of water by the *skin* is increased—even to double the amount excreted by the kidneys, and there is also a considerable elimination of chlorides and of fatty acids; hence the total amount of water in the body, and especially of that in the blood, is diminished by active exercise. The *kidneys* excrete a lessened amount of water and of chlorides: the nitrogenous constituents of the *urine* are slightly decreased during exercise, again rising during the succeeding period of rest—the net result being a slight increase in nitrogenous excretion. The *muscles* undergo an increase in bulk and firmness by regular exercise, representing a storage of nitrogenous tissue—owing to increased *appetite* (especially for fat and meat, as well as for water), while *digestion* is more rapidly and efficiently carried on. The total weight of the *aces* is slightly diminished.

The general result of these changes is an improvement and maintenance of nutrition.

Abuse of Exercise.—From the summary given above it will be understood that *deficient exercise*—implying lessened oxidation and a more feeble circulation—means less active tissue-change throughout the body. Fat and water accumulate in the organism to excess; appetite becomes impaired, digestion becomes less efficient, and constipation, with its secondary evils, including anæmia, tends to become chronic. The muscles themselves waste, growing softer, pale and flabby; and, as the muscular tissue of the heart-wall shares in these changes, that organ becomes more liable to dilatation—and even to rupture—under the strain of exertion, especially if this be suddenly or violently undertaken. The nutrition of the lungs becomes impaired, thus favouring the development of tuberculosis; and the malnutrition of the whole body tends to increase, or at least to be maintained at a low standard, by the mutual reinforcement of these degenerative changes in its tissues acting in a vicious circle.

Excessive Exercise produces results of which some are immediately recognisable. If very severe, or if persisted in despite the warnings of fatigue, other effects of a character still more serious are likely to be entailed. Locally, excessive exertion induces, first, exhaustion of the muscle; then diminished irritability; and finally leads to degeneration, so that an over-used muscle becomes smaller in bulk than normal. These local changes are the outcome of a denial to the tissue of those periods of rest which are requisite for the recruiting of its expended energy and for the proportionate elimination of its own waste-products. And what is true of the individual muscular fibre may be broadly predicated of the whole muscular system, were that subjected to exertion similarly prolonged and repeated without the necessary reliefs.

Exercise may be excessive by reason of its severe character, its too frequent repetition at short intervals, or its undue prolongation; but such terms are really only relative. There is no absolute standard by which to judge excess in exercise, because there is no fixed limit of the proper amount of exercise. This must vary in each case with the individual, and with his state of health and capacity for exertion at the time, as well as with the rate at which it is pursued—i.e. the time within which it is accomplished; while its effect on the organism is directly influenced by the external conditions under which it is practised—thus if active exercise be undertaken in heavy or too closely fitting garments, just before or just after a meal, or in an ill-ventilated room, its results are necessarily harmful. The only safe rule for general application is that which insists on the abandonment of the exercise directly moderate exertion has induced a definite feeling of fatigue, or signs of respiratory embarrassment—as when a cyclist begins to breathe with his mouth open. Again, if the fatigue following a day’s exercise be entirely removed by a good night’s rest, it may be assumed that the exertion has not been excessive; but if the sleeper awake feeling tired or exhausted, it is at least certain that he has trenched on his vital capital or reserve nutrition, and that his physiological expenditure has for the time being exceeded his receipts. If the warning given by fatigue be resisted, sleeplessness is apt to be induced, and this fatigue-insomnia is increased

by tea. Alcoholic beverages should be eschewed during exertion, since they tend to lessen the excretion of carbon dioxide. One of the earliest effects of excessive muscular exertion consists in laboured breathing, with occasional sighing respiration: perseverance beyond this point may produce hæmoptysis. If too frequently repeated, strenuous exertion may induce cardiac hypertrophy and dilatation, valvular disease, atheroma of the vessels, and—as the heart-muscle begins to degenerate—even rupture of that organ. After exercise continued for too long a time, the normal slowing of the heart's action during the period of subsequent rest may be marked by intermittence or replaced by irregularity of its action (palpitation). Exertion which is excessive in amount, or which involves muscular effort too frequent and concentrated in time, is indicated by great increase in the pulse-rate (120—140 per minute), and by disturbance of the cardiac rhythm. The practical application of these facts has a special bearing upon two important epochs in life. For the child, and especially for the adolescent and the young adult, it is a matter of vital gravity that his exercise—while practised regularly, in sufficient amount, and as much as possible in the open air—should be fairly apportioned in character and in duration to his physical capacity, and that it should be guarded by sufficient bodily rest and by adequate food. Thus in schools all pupils should be classed for the rougher games, according to their several physical conditions and capabilities—not by age or size or weight merely, for any such arbitrary standard of comparison may, if taken by itself, be quite delusive. The effects of exertion should be closely observed, and if necessary the pupil's physical classification should be accordingly modified. Any indication of distress or of circulatory or pulmonary embarrassment must be noted, and should forthwith ensure an appropriate alteration of the character and amount of the exercise allowed. Excessive competition in amusements involving either prolonged or very severe exertion should be rigorously discouraged; this applies particularly to such forms of exercise as school-runs and paperchases—which are free from objection if properly practised over moderate distances, gradually increased from a short minimum, by those whom previous examination has shown to be sound in wind and limb, and whose condition, as indicated by careful and repeated inspection, does not in any way suffer by a repetition of the exercise at moderate intervals. The practice of long-distance diving falls into a different category; it is easily and insidiously productive of most severe strain on the heart and lungs—the effects of which are sometimes developed with startling suddenness; and should very rarely if ever be practised by the growing lad. Rowing, again, is an excellent exercise when indulged in with reasonable precautions by healthy persons. But the exertion involved in a boat-race is severe—not on account of the total amount of muscular work accomplished, which is relatively small, but by reason of the concentration of the effort within as short a space of time as possible. The would-be oarsman should be safeguarded by a careful preliminary examination. The discovery, before or during practice for a race, of a dicrotic pulse, signs of commencing cardiac dilatation or hypertrophy, or the detection of a heaving or diffused impulse, especially if accompanied by a murmur audible along the left side of the sternum, or of any

marked abnormality of the urine, should be held to interdict all racing so long as they continue. Active exercise should be undertaken only in suitable pervious clothing, ensuring free movement of the chest and limbs. Thus, in military drill, for example, it is essential that the tunic be loose, especially across the chest and around the neck, and that belts and accoutrements be so arranged as not to hamper either muscular movements or the action of the heart and lungs. Exercise should be followed whenever possible by rapid bathing with vigorous friction of the skin. The immediate dangers of strenuous or prolonged exercise are much increased by the ingestion of insufficient water.

The healthy schoolboy at a properly conducted school ought always to be practically in training for such exertion as his ordinary pastimes call for. But the individual of maturer years, especially if he have been addicted to athletic exercises during earlier life, is beset by a more insidious danger in proportion as he drifts into a sedentary mode of living. A heart which, in common with the rest of the muscular system, has been moderately hypertrophied (increased in bulk and power) by the practice of active exercise, suffers in direct proportion to that hypertrophy if active exercise be for some time discontinued. If such an individual be suddenly called upon for severe or sustained effort, the results are apt to be more disastrous than if the same exertion be attempted by the man of originally less developed physique. Such individuals should never attempt a resumption of their former athletic efforts until a course of training, preceded and supervised by skilled medical examination, has prepared them for the enterprise by restoring them to adequate 'condition.' And every old athlete should remember that, in order to safeguard himself from the development of cardiac degeneration, it is more important for him than for the man with a less active record to practise continually some form of regular, active, muscular exercise in the open air. This, of course, must be appropriate in its nature to his age and powers; but, above all things, it must be undertaken regularly day by day, and not by spurts alternating with periods of muscular inactivity. Horse-exercise, bicycling, walking, and golf all afford suitable means of securing the desired result. Finally, it may be pointed out that many of those who have already begun to suffer from substituting a too sedentary life for one of active exercise are apt to be the subjects of commencing dilatation of the stomach. If this condition be overlooked or ignored, treatment is rarely satisfactory. It is important that such a person should not take active exercise for an hour before or after food; and that immediately after each meal he should lie down flat—not recline—on a firm level couch for not less than thirty minutes, so as to favour that movement of the contents of the stomach which is so necessary to proper digestion by avoiding the increased sagging of the greater curvature of the organ which gravity tends to aggravate in the sitting or erect posture.

C. E. SHELLY.

EXFOLIATION (*ex*, from; and *folium*, a leaf).—The separation of a portion of dead bone or cartilage from the living tissue, in the form of layers (*see* BONE, Diseases of). The term is also applied to the separation of a false membrane,

which has been mistaken for the whole mucous lining of the bladder or uterus. See BLADDER, Diseases of.

EXHAUSTION (*ex*, from; and *haurio*, I draw out).

DEFINITION.—Exhaustion consists in a failure to respond to stimulation. Exhaustion of muscle and nerve is brought about by excessive, quickly repeated, or continuous stimulation. It is favoured by cutting off, or by an alteration in the quality, of the blood-supply; by previous insufficient exercise of function; by exposure to extremes of temperature; by an insufficient supply of oxygen; by an excessive supply of carbonic acid; and by exposure to certain toxic agents.

Exhaustion may be *general* or *local*.

1. General Exhaustion.—General exhaustion is brought about by overwork, whether physical or mental, and especially by unremitting and monotonous duties which keep the same paths of action in a state of constant activity. It is not often, if ever, that general exhaustion is produced in a healthy man by mere physical labour, however great; but excessive mental labour, especially if it be monotonous, is certainly capable of even permanently damaging the nerve-centres. When in addition to hard mental work, which is performed voluntarily, some constant stimulus, which cannot be arrested, unceasingly works upon the brain, exhaustion quickly results; as when, for example, a man who is harassed by trying to earn sufficient for his family meets with some shock to his nervous system (such as a railway accident, the sudden death of a dear relative, or a severe pecuniary loss), which haunts him like a spectre day and night, robs him of his rest, and deprives him of his appetite. General exhaustion is favoured by all conditions which give rise to anæmia or faulty nutrition, such as hæmorrhage, prolonged pyrexia, inadequate diet, persistent morbid discharges, or venereal excess; by the retention in the tissues of the products of their activity, which is favoured by working in a foul atmosphere, or by derangement of the excretory functions; by exposure to extremes of temperature; and by a previous condition of excessive slothfulness. General exhaustion may be induced suddenly by physical causes, such as a severe injury (collapse from shock), or psychical causes, such as fright.

SYMPTOMS.—The symptoms of general exhaustion are: 1. Loss of sleeping power, persistent dreaming, talking in the sleep, and somnambulism. The patient may wake in the morning feeling totally unrefreshed. 2. Incapacity for work, and inability to seriously apply the mind to one subject for any length of time. 3. Headache, and a feeling of oppression in the head. 4. Languor and general lassitude. 5. A rapid feeble pulse. 6. An anxious expression of face; and a contracted and sluggish pupil. In addition to these we may get hypochondriasis and hysteria. Moreover the symptoms of more serious disease largely due to exhaustion may be superadded. Two instances have come within the writer's knowledge of transient hemiplegic symptoms having been induced by excessive application to literary work. The digestion is often deranged, and functional disturbance of the heart is common. Occasionally the urine is altered in quality, and may contain alkaline phosphates or sugar. More rarely it manifests excessive acidity.

2. Local Exhaustion.—Local exhaustion is the result of excessive local stimulation, and it is particularly liable to occur as a prominent symptom in patients who are suffering from general exhaustion. The failure of the heart so common among over-trained athletes (see EXERCISE); the loss of power in the rectum which results from the excessive use of purgatives; the failure of the uterus in cases of protracted labour; and the failure of the voluntary muscles which occurs in those professional ailments of which 'writer's cramp' is the type, may be taken as examples of local exhaustion. See OCCUPATION-DISEASES.

TREATMENT.—In the treatment of exhaustion the main indications are to lighten the labour, and obtain rest. In cases of general exhaustion it is often advisable to administer narcotics, such as opium, chloral hydrate, sulphonal, or bromide of potassium; and it will be generally found that, when once refreshing sleep has been established, the more aggravated symptoms will subside. Fresh air and a good diet are most necessary. Stimulants must be used with great caution, for it is clearly not desirable to goad the exhausted organs into further action, although it may be necessary to employ stimulants to give temporary power while the faculty of sleeping is being re-established. All causes of anæmia must be removed. When recovery is established, the patient must be encouraged to relieve the monotony of his life by some pursuit which should be, as it were, the complement of his ordinary occupation. Thus the headworker should endeavour to amuse himself in his leisure hours by gentle outdoor exercise, by music or painting, or by practising some handicraft. See DEBILITY; and FATIGUE.

G. V. POORE.

EXOMPHALOS (ἐξ, out; and ὀμφαλός, the navel).—A term applied to umbilical hernia. See HERNIA.

EXOPHTHALMIC GOITRE (ἐξ, out; ὀφθαλμός, the eye; and *guttur*, the throat).—**SYNON.**: Graves's Disease; Basedow's Disease; Fr. *Maladie de Graves*; *Goitre exophtalmique*; Ger. *Glotzaugen-kropf*; *Basedow'sche Krankheit*.

DESCRIPTION.—Typically this disorder is manifested by five leading symptoms: (i) general nervous instability, (ii) tachycardia with paroxysms of palpitation; (iii) enlargement of the thyroid gland; (iv) exophthalmos; and (v) a fine muscular tremor. It is the practice of some physicians to refer to this head cases of tachycardia without goitre or exophthalmos; but whatever may be the underlying pathology of such incomplete cases, they can by no legitimate licence of language be termed either Graves's Disease or Exophthalmic Goitre.

The *mental condition* of the victims is one of nervous expectancy, in which fearfulness has settled down, like a nightmare, on the spirit. A state of mental incoherence, happily termed 'chorea of ideas,' makes it very difficult to obtain from them a concise and consecutive history of their illness. Loss of flesh is often a marked feature.

Usually the patient's chief complaint is of the paroxysms of *palpitation*, which are excited under the slightest emotional influence; between the paroxysms the heart-beat remains rapid, commonly ranging from 100 to 120 beats per minute. It is remarkable how seldom, in the absence of compli-

cating endocarditis, the heart yields before the violent strain; indeed in most cases there is found clinically no perceptible enlargement of the heart.

The advent of the *goitre* is probably in point of time invariably subsequent to the cardiac disturbance, though the goitre now and again is the first symptom to attract the patient's notice. The enlargement usually involves the whole of the thyroid gland, but in rare cases may be confined to one lateral lobe or even to the isthmus: when both lateral lobes are affected, the right lobe is commonly larger than the left, such being the normal relation. At first the goitre is soft from vascular congestion and subject to variations in size, but later when true hyperplasia has occurred it is firm, and its size constant. Thrills and murmurs may be discerned over the goitre, particularly in the neighbourhood of the thyroidean arteries. The seeming absence of a goitre in some of the so-called incomplete cases may perhaps be due to the difficulty of detecting the minor degrees of enlargement. The microscopical changes in the thyroid gland are clear and characteristic. In the vesicles the lining epithelium has changed its cuboid form for the columnar type: it has also undergone such rapid proliferation that papillary puckers are thrown out into the vesicle to accommodate its increased dimension. Some of the vesicles are packed with shed epithelial cells. Within the vesicles the secretion is diminished in amount, and more mucinoid than in health. Along with these changes is found a great increase of newly formed tubular spaces lined by a single layer of cuboid epithelium. In cases of long standing there may be a marked development of connective tissue, and its encroachment on the glandular tissue may perhaps pave the way for the occasional passage of exophthalmic goitre into myxœdema.

The *protrusion of the eyeballs* gives to the face a scared and terrified expression: in many cases a clear band of sclerotic is visible above the iris due to retraction of the upper lid (Stellwag's sign) from spasm of Müller's muscle, and to this same cause may be ascribed the occasional absence of reflex blinking. The exophthalmos is often markedly increased during the paroxysms of palpitation: indeed actual dislocation from the orbit has occurred. The closed eyelids may not meet over the eyeballs, so that they become irritable and inflamed. Not infrequently the upper lid follows the downward movement of the eyeball in a halting lagging way (von Graefe's sign) instead of with a smooth simultaneous movement. Defective convergence for near objects is sometimes found, the attempt causing a sense of effort, but no diplopia. The two eyeballs are usually equally prominent, but in very rare cases unilateral exophthalmos is present: in this case the enlargement of the thyroid is usually more marked on the side of greater proptosis, but a crossed relation has also been described. At autopsy the amount of post-orbital fat has been found greatly increased: this would seem to be a result rather than a cause of the forward movement of the eyeballs, which may reasonably be referred to dilatation of the retro-bulbar vessels and retraction of the upper eyelid. The normal condition of the pupil is worthy of note in a condition in which the cervical sympathetic is at least under suspicion.

The trembling is generalised and is a *fine rhythmic tremor* that can be nearly always demonstrated, but most markedly during the periods of greatest heart-hurry; the handwriting may become so

unsteady that the patient may be unable even to sign her own name. Akin to the tremor is a feeling of giving way at the knees, of which patients often complain. Like the cardinal symptoms, the subsidiary symptoms all tell a tale of disturbed innervation. Sudden flushings are a common trouble, and paroxysms of profuse perspiration, that serve to lower the electrical resistance of the skin: this same vaso-motor derangement in the intestine is evidenced by intractable paroxysms of diarrhoea. Albuminuria is occasional, and its fugitive character would suggest a vaso-motor origin; glycosuria too is not uncommon, but some doubt has been cast on the identity of the reducing substance with sugar. To this same category may be referred the œdema that occurs in patches chiefly about the eyelids and face, but sometimes also over more extensive areas of the trunk and limbs. Sometimes the skin is the seat of a yellowish pigmentation, generalised or in patches. Unquestionably in some cases the pigment has been of arsenical origin.

PATHOLOGY.—There can be little doubt that the starting-point must be sought in the central nervous system. All observers are agreed upon the natural instability of the nervous system in the subjects of exophthalmic goitre. Its greater incidence is upon the female sex—the sex of more delicate emotional susceptibility—in the proportion of some 10 to 1. Moreover its common age of occurrence is adolescence, or early adult life, at the period of highest sexual activity, though exceptionally cases are observed in childhood and in advanced age. Family predisposition is occasional, but one may almost invariably elicit a history of well-marked nervousness, of some specialised neurosis such as asthma, or even of actual insanity in the parents; very commonly too the condition is linked to other nervous disorders such as chorea, epilepsy, hysteria, diabetes, and acromegaly. Lastly, the train of symptoms is almost invariably set in motion by sudden emotion or by prolonged worry. This direct dependence of the symptoms on emotional causes is an important argument against any theory that would assign the symptoms to a primary disease of the thyroid gland. Moreover the cardiac and arterial conditions of a thyroid-toxemia, experimentally induced, are the absolute antithesis of the cardiac and arterial conditions of exophthalmic goitre. The symptoms of the disorder are all found in embryo in the expression of the primal emotion of terror. In this connection the heart-hurry, the tremor, the starting of the eyeballs are so familiar as hardly to need mention, but the resemblance in detail is pointed by the concurrence of manifold subsidiary symptoms, such as perspirations, vomiting, and diarrhoea. *Cessante causâ, cessat et effectus*: in exophthalmic goitre the cause persists, so too does the effect: the idea of terror is imperative and persistent, so too is its expression. At first sight the enlargement of the thyroid seems to fall outside the similitude, for there is but little evidence of an even transitory enlargement of the thyroid under the influence of emotion. Yet the same sympathetic disturbance that lashes the heart into turbulent overaction, and dilates the retro-bulbar arteries, may likewise dilate the thyroidean arteries, and promote a vascular congestion that is the first step towards a true hyperplasia of the gland. It has been suggested that this hypertrophy is an effort of nature to minister to the diseased nervous system; for thyroidectomy and myxœdema have proved that the thyroid gland has some important influence on

the nutrition of the nervous system. If it be so, the effort at compensation is futile, for in spite of the histological evidence of heightened secretory purpose, and of great increase of secretory surface, there is a marked decrease of secreted substance. Histology thus declares emphatically against the theory of an initial thyroid-toxæmia: so too therapeutics, for in no single case has the introduction of thyroid-secretion into the system produced the typical mental symptoms; and we know that so given, it can and does replace the internal secretion of the gland in a state of vital activity, for the very reason that by such adventitious supply we are able to counteract the myxœdematous condition, which is due to an atrophy of the thyroid gland. At most the thyroid-hyperplasia may be responsible for some of the many subsidiary symptoms of the disease which we have already briefly mentioned.

COURSE.—While some cases are acute and run on rapidly to a fatal issue, the general tendency of the disease is to a chronic course. Some do undoubtedly recover, particularly in the class that commands the comforts of living; pregnancy now and then appears to exercise a favourable influence. Others run a steady downhill course, and death ensues from paroxysmal diarrhœa, from heart-failure, or from insanity. Occasionally a case ends in myxœdema. The size of the goitre and the prominence of the eyeballs are no criterion of the severity of the case, nor is the degree of cardiac disturbance, except in so far as it hastens cardiac failure. Cases in which mental derangement is extreme generally have an early and fatal termination (see *INSANITY IN SPECIAL DISEASES*). Rapid and progressive loss of weight and intractable anæmia are always signs of evil omen. In patients who recover completely or in whom the disease lapses into quiescence, the first sign of improvement is commonly a sustained diminution of the pulse-rate.

TREATMENT.—At present we have no remedy either in medicine or surgery by which to strike at the root of the morbid state. Those measures attain most success that promote tranquillity of mind and rest of body: change of scene, change of air, change of the daily habits of life have a foremost place in treatment. Much of the temporary benefit that is often derived from a stay in hospital is due to the complete bodily rest, the freedom of mind from the anxieties of everyday life, and the regular mode of living. Of drugs, those alone that minister to the general well-being, such as iron, arsenic, and strychnine, have stood the test of time: to this group also belongs supra-renal substance. Belladonna and thyroid-extract, both introduced on behalf of a faulty conception of the nature of the disease, even yet find devotees. Thymus-extract has been tried and found wanting, and its use has not served to strengthen the belief that the enlargement of the thymus, so often found *post mortem* in cases of exophthalmic goitre, is of the nature of a compensatory hypertrophy. Galvanism has its supporters on the Continent, but in this country has fallen into disrepute. Partial thyroidectomy has failed to justify its trial, and in more than one case the patient has died under the anæsthetic. More recently, complete excision of the whole cervical sympathetic has been undertaken on both sides of the neck: this in some cases is said to have resulted in recession of the eyeballs and shrinking of the thyroid gland, but has no influence on the

heart and the general nervous state. Surgery may scotch the disease, but can never kill it.

With this background of deep-seated nervous disorder, it is matter of small surprise that individual symptoms tend to be stubborn and even intractable. An ice-bag to the præcordium will sometimes soothe the tumult of the heart; a cold compress to the goitre will sometimes soothe the sense of suffocation. Diarrhœa will usually defy the most approved combinations of sedatives and astringents, and, though for the most part ceasing spontaneously and abruptly, is perhaps the most common cause of death.

RAYMOND CRAWFORD.

EXOSTOSIS (ἐξ, out of; and ὀστέον, a bone). A bony outgrowth from any part of the skeleton.—*See* BONE, Diseases of.

EXPECTANT ATTENTION.—An important mental state. *See* MESMERISM.

EXPECTORANTS (*ex*, out of; and *pectus*, the chest).—**SYNON.** : Fr. *Expectorants*; Ger. *Auswurfsbefördernde Mittel*.

DEFINITION.—Medicines which facilitate the removal of secretions from the air-passages.

ENUMERATION.—The leading expectorants are : (A) Ipecacuanha, Antimony, Apomorphine, and Iodide of Potassium; Chlorides of Potassium, Sodium, and Ammonium; and Inhalation of Steam. (B) Squill, Senega, Benzoïn, Benzoic Acid, Benzoate of Ammonium; Myrrh, Storax, Balsam of Tolu, Balsam of Peru, Ammoniacum, Galbanum, Asafetida, Anise, Fennel; Larch Bark, Tar, Terebene, Copaiba; Vapour of Chlorine, Iodine, Ammonia, Creosote, and Carbolic Acid. (C) Ammonia, Carbonate of Ammonium, Strychnine, Nux Vomica, and Belladonna.

ACTION.—The mode of action of expectorants is not well understood, and any explanation of it in the present state of our knowledge can only be regarded as provisional. Expectorants may be divided into two classes : (1) Those which modify the nature of the secretions from the respiratory passages; and (2) those which modify the respiratory movements by which the secretions are expelled. In considering the mode of action of the first class it must be remembered that the secretions from the respiratory passages depend, like many other secretions, on two factors, the *direct* influence of the *nerves* upon the secreting structures, and the *amount of blood* supplied to them. Each of these two factors may be influenced to a different extent by various drugs. As has already been said, the exact action of each cannot be determined at present, but the first class of expectorants may be subdivided into two divisions which are distinguished in the foregoing enumeration as A and B. The division A rather diminish than increase the activity of the circulation, and are therefore called *sedative* expectorants. The division B somewhat increase the circulation, and are called *stimulating* expectorants. Those comprised under C *stimulate the respiratory centre* in the medulla oblongata, and increase the respiratory movements.

USES.—*Sedative expectorants* (class A) are useful when there is congestion of the respiratory passages, with very scanty, tough expectoration, as in commencing bronchitis. In such circumstances, when dry rhonchi are heard abundantly, with few or no moist râles, the patient often coughs until quite exhausted, bringing up scarcely anything.

The administration of sedative expectorants renders the secretion from the respiratory passages more fluid, abundant, and easy to expectorate. When these expectorants do not succeed in ordinary doses, their action may be much assisted by the administration of a purgative, or, still better, by giving either ipecacuanha or tartar emetic in such a large dose as to produce sickness and vomiting. When the distress of the patient is great, the abstraction of a small quantity of blood by cupping or by venesection may give great relief. The inhalation of steam alone is also beneficial, and the air of the patient's chamber should be kept warm and moist.

Stimulating expectorants (class B) do more harm than good when administered in the conditions just described, but are beneficial when the acute symptoms have passed off. When this is the case, but the expectoration is tough and somewhat scanty, squill is a useful expectorant; but when the expectoration is abundant, benzoin, balsams, or ammoniacum would be preferable. In chronic bronchitis, inhalations of ammonia, chlorine, iodine, creosote, carbolic acid, eucalyptus, or pine-oil are useful. When the expectoration is fetid, chlorine, iodine, and carbolic acid inhalations are best.

The expectorants which act on the respiratory movements (class C) are useful in cases of debility, as they stimulate the respiratory nervous centre in the medulla oblongata, as well as assist the failing circulation. They may be advantageously combined with stimulating expectorants, such as squill or benzoin, according to the nature of the secretion.

T. LAUDER BRUNTON.

EXPECTORATION (*ex*, out of; and *pectus*, the chest).

DEFINITION.—This word specifies either the act of expelling anything from the chest, or the matter so expelled, which is also called *sputum* or *phlegm*. See COUGH; and SPUTUM, Examination of.

EXPLORATORY OPERATIONS.—See PERICARDIUM, PERITONEUM, PLEURA, SKULL, Exploration and Surgery of; and LUMBAR PUNCTURE.

EXPOSURE, Effects of.—See CHILL; INFECTION; FATIGUE; and RESUSCITATION.

EXSANGUINE (*ex*, without; and *sanguis*, blood).—Deprived of blood—bloodless.

EXTENSOR RESPONSE.—See BABINSKI'S SIGN.

EXTRA-UTERINE FŒTATION.—See PREGNANCY, Diseases and Disorders of.

EXTRAVASATION (*extra*, without; and *vasa*, vessels).—SYNON.: Fr. *Extravasation*; Ger. *Extravasat*.

DEFINITION.—Extravasation is the escape of any of the fluids of the body, normal or abnormal, from the vessel, cavity, or canal that naturally contains it, into the surrounding tissues. The result of the effusion is also called an *extravasation*.

Extravasation, being the effect of rupture or perforation of the walls of a hollow organ, may be due to injury, or weakness of the parietal structures, to morbid conditions of the blood, or to increase of internal pressure.

The fluids most frequently found extravasated are blood, urine, bile, the contents of the alimentary

canal, and certain constituents of morbid growths and fluid collections. The present observations will have reference only to extravasation of blood; other kinds of extravasation being described under the heads of the several organs involved.

Extravasation of Blood.—This may be produced by direct violence; by pressure of contained blood upon a diseased vessel-wall; or in certain morbid states of the blood (purpura, hæmophilia, &c.) owing to some unexplained conditions: possibly fatty degeneration of the capillaries here occurs and leads to their rupture.

When the effusion takes place into one of the serous sacs, it forms a collection of blood, variously named *hæmothorax*, *hæmatocele*, &c. If the subcutaneous, submucous, or other connective tissue, or the substance of an organ, be invaded, the effused blood finds its way between the elements, separating and compressing them; and there are formed what are described simply as *extravasations* of blood, or more definitely, according to their extent, *parenchymatous* or *interstitial hæmorrhages* or *apoplexies*, *suffusions*, *ecchymoses*, *petechiæ*, or *vibices*. The extravasated blood generally coagulates. After a time the blood-corpuscles break down, owing to degeneration of their stroma, and hæmoglobin is liberated. This becomes dissolved in the surrounding fluid and undergoes certain changes whereby it is broken up into hæmatoidin—an iron-free body—and other substances, including hæmosiderin and hydrated peroxide of iron. In small extravasations the pigment is rapidly absorbed by the lymphatics, part being subsequently excreted in the urine, and part deposited in adjacent lymphatic glands. In larger effusions the clot acts as an irritant, and a certain amount of fibrous tissue is formed round it. Thus there may result a cystic cavity containing either reddish-brown or colourless fluid, according to the extent to which the pigment has been absorbed. This condition is commonly met with in cases of cerebral hæmorrhage. In other cases connective-tissue cells penetrate into the clot, which is gradually absorbed and replaced by fibrous tissue. Crystals or amorphous masses of hæmatoidin are frequently found in such scar-tissue and in old cysts, thus marking the nature of the process which has occurred. The gradual absorption of the blood-pigments gives rise to the familiar series of colour-changes seen after bruising of superficial parts. The seat of effusion first appears of the colour of blood or its derivatives; but if much below the surface, the extravasation may not be visible for some days, after which time it appears of a bluish, greenish, or yellowish hue. A *hæmatoma* or collection of effused blood may become infected by pyogenic organisms and suppurate, giving rise to a collection of pus mixed with remains of blood-clot.

The *symptoms* of extravasation of blood are so various that they cannot be stated in general terms. If extensive and affecting vital parts, the effusion may be attended with shock, syncope, or death. Local pain is not common unless the extravasation be very large. The pressure of the effused blood upon the vessels, nerves, and muscles of the part—in a limb, for example—may produce paralysis, disturbance of sensibility, œdema, fall of temperature, and even gangrene. As a rule, however, extravasations of blood are limited in size, give rise of themselves to no serious symptoms, and readily disappear by absorption.

J. MITCHELL BRUCE.

EXTROVERSION (*extra*, outwards; and *verso*, I turn).—The eversion or turning inside out of a part, as the eyelids or bladder. In the bladder, extroversion is associated with that condition, usually congenital, in which the anterior wall of this organ and of the abdomen is deficient, and its posterior wall projects through the opening thus formed.

EXUDATION (*exudo*, I sweat).—The process by which certain of the elements of the blood pass through the walls of the blood-vessels into the surrounding tissues and cavities, as in inflammation. The term is also used to indicate the products of this process. See INFLAMMATION.

EYE, and its Appendages. Diseases of.
SYNON.: *Fr. Maladies des Yeux*; *Ger. Augenkrankheiten*.

The diseases of the eye will be considered in the following order:

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See also VISION, Disorders of; OPHTHALMOSCOPE; OPHTHALMOSCOPE IN MEDICINE; and PAIN IN VISCERAL DISEASE.

I. Diseases of the Conjunctiva.

1. *Inflammation*.—**SYNON.**: Conjunctivitis. The conjunctiva is exceedingly liable to inflammation, and its inflammations are commonly arranged in groups, which are sufficiently distinct in their typical examples, but are not separated by any definite boundary lines.

In their earlier stages inflammations of the conjunctiva possess many characters in common. They are attended by the four signs of inflammation—heat, redness, swelling, and pain; although the heat and the swelling are usually kept within limits by the discharge, which is often free, sometimes profuse, at first mucous, afterwards muco-purulent or truly purulent in character. If, however, the exudation be of a firmer consistence than usual, it not only produces a superficial discharge, but distends the meshes of the submucous tissue, elevates the conjunctiva from the sclerotic, and causes it to overlap the corneal margin as a swollen ridge: a condition known as *chemosis*. The redness depends, of course, on the degree of the congestion, which may or may not be sufficient to obliterate the intervacular meshes, and to produce a uniform colour; and the pain is not severe, except in cases of very dense subconjunctival swelling. From other forms of inflammation that of the conjunctiva is distinguished by certain negative characters. Except as a result of secondary changes, or as a mechanical effect of the presence of a film of turbid secretion, the transparency of the cornea is not affected, and the acuteness of vision is not impaired. The congestion is limited to the conjunctiva, and the distended vessels can be emptied for a moment by pressure through the lower lid, so as to reveal a glimpse of the white sclerotic underneath.

In mild cases conjunctivitis is an unimportant affection, but in its more severe varieties it is attended

by two sources of danger. During its acute stage it may produce partial or complete necrosis of the cornea, leading to great impairment of sight or to blindness; and in some chronic forms it often occasions great hypertrophy of the papillæ of the portion of the membrane which lines the lids. These papillæ may even become converted into shaggy or warty excrescences, which irritate the corneal surface, and cause the development of vessels beneath its epithelium. In time the papillæ dwindle, and the tissue round about them contracts, thus rendering the tarsal cartilage (especially that of the upper lids) incurved, and bringing the cilia to rest upon the surface of the eyeball. Great distress and permanent impairment of vision may be thus occasioned; and the contraction may be so slow as to deprive it of any manifest connection with the inflammation in which it had its origin.

The bacteriology of conjunctivitis has attracted much attention of recent years, and many attempts have been made to connect particular forms of the affection with particular microbes, but, except in the infantile variety, these attempts have to a large extent been defeated by the abundant bacterial growth invariably found in the secretions, and by the large number of species which are therein represented. On the whole it cannot be said that this department of inquiry has yet been sufficiently developed to afford any important aid in either the diagnosis or the treatment of the affections concerned; or even to constitute an assured basis for their better classification; and it is still permissible to say that the chief varieties of conjunctivitis are: (a) infantile; (b) simple or catarrhal; (c) contagious; and (d) diphtheritic.

(a) *Infantile Conjunctivitis*.—**SYNON.**: *Ophthalmia neonatorum*.

ÆTIOLOGY AND SYMPTOMS.—Infantile conjunctivitis is due in most cases to direct inoculation, during birth, with vaginal secretion containing the gonococcus. It commences on or about the third day, and passes rapidly into the purulent form. It is attended by considerable puffy swelling of the lids, and by profuse thick discharge, which soon dries upon the tarsal margins, and often causes them to cohere. If neglected or improperly treated, the disease often leads to sloughing of the cornea; but it may always be cured if it is seen while the cornea is still bright.

TREATMENT.—The treatment required is to wash away the discharge carefully and frequently; to apply an astringent lotion (the best is a solution of two grains of nitrate of silver to an ounce of distilled water) to the conjunctival surface every four hours, or less frequently when improvement is established; and to anoint the edges of the lids with simple ointment to prevent their agglutination. In cases of inherited syphilis mercurial inunction should be prescribed. If the infant be brought up by hand, its food must be carefully regulated; and if it be very feeble, it may take a little cod-liver oil, combined in an emulsion with two-minim doses of the liquid extract of cinchona. Where the mother's milk fails, it is often desirable to obtain a wet-nurse; but the local treatment is that which is chiefly important, and upon which, in most cases, entire reliance may be placed.

Proceeding, as it does, from a definite and well-understood source of infection, the prevention of ophthalmia neonatorum is even more important than its cure, and fortunately admits of easy and com-

plete accomplishment ; insomuch that the occurrence of the disease may now be mainly attributed to neglect of proper precautions. These were first definitely formulated by Professor Credé of Leipzig, who directed that, as soon as the head of the infant had passed the vulva, the face should be wiped clean, the eyelids separated, and a drop of solution of silver nitrate, of the strength of two per cent., allowed to fall from a glass rod upon the surface of the eyeball. The occasional slight reaction may be treated by cocaine and cold compresses ; and a much weaker solution, say of two grains to the ounce, would probably be equally efficacious. Where properly applied, as in some lying-in institutions, Credé's method is said to have reduced the proportion of cases to births from 19 to 0·2 per cent., and to have so much diminished the severity of the few occurring cases as to remove all danger of destruction of the cornea.

(b) *Simple or Catarrhal Conjunctivitis.*

ÆTIOLOGY AND SYMPTOMS.—This form of conjunctivitis is usually due to exposure to cold or to some chemical or mechanical irritant. Its most distinctive character is that the discharge is chiefly mucous, and has not much tendency to become purulent.

TREATMENT.—In every case the surface of the eyeball, and the lining membrane of the lids, should be examined for any foreign body which may be the cause of the trouble ; and if such should be found and removed, there will seldom be need for further treatment. If there be no foreign body, the two-grain solution of nitrate of silver, or of sulphate of zinc, may be placed within the conjunctival sac, by means of a quill or dropping-bottle, every two or three hours, and speedy recovery will usually be the result. In cases of very mild type, one or two applications of the astringent may be sufficient ; and these may be supplemented by the use, in the same manner, of a solution of cocaine with boric acid. A good formula for this purpose contains two grains of cocaine hydrochlorate, and four of boric acid, to the ounce of distilled water. During the treatment the eyes should have functional rest, and should be sheltered from external cold, from dust, and from glare.

(c) *Contagious Conjunctivitis.*—**SYNON.** : Purulent Ophthalmia.

ÆTIOLOGY AND SYMPTOMS.—In this form the discharge rapidly assumes a purulent character. This may happen when the simple form is aggravated by the state of the patient or by accidental circumstances ; or when the disease is produced by inoculation with the discharge from a similar case, or with contagious pus from the urethral or vaginal mucous membrane. The state of the patient which is most likely to promote the development of the contagious form is that in which the conjunctiva is beset with the granular semi-transparent bodies, formed by aggregations of lymph-corpuscles, which are known as 'trachoma,' as 'sago-grains,' or as follicular granulations. These bodies are commonly present in the eyelids of persons (especially of young persons) who are crowded together under insanitary conditions of living, as in barracks, camps, or badly regulated schools ; and their presence is not only liable to excite a special form of conjunctivitis, but also renders the conjunctivitis of simple irritation prone to assume a purulent character. It is impossible to say how soon the discharge of conjunctivitis becomes contagious, or capable of reproducing the disease in

others ; but its activity in this respect seems to bear some proportion to the intensity of the inflammation which produces it ; and the activity of gonorrhœal pus is probably greater than that of any conjunctival product. In the more intense forms of purulent ophthalmia there are great swelling of the lids and of the ocular conjunctiva, early chemosis, and a tendency to speedy sloughing of the cornea ; while the milder forms pass into the catarrhal by imperceptible gradations.

TREATMENT.—In the space here available, the treatment of contagious conjunctivitis cannot be described in detail. It mainly rests upon the action of local astringents, graduated in strength according to the severity and the stage of the disease. In severe cases the eyelids must, as far as possible, be everted, and the whole of the palpebral conjunctiva carefully touched with a stick composed of one part of nitrate of silver fused with four parts of nitrate of potassium. The caustic should be neutralised by a drop or two of a solution of common salt, applied by means of a camel's-hair pencil, before the lid is suffered to return into contact with the cornea ; and the cauterisation must be done carefully and with a light hand, so that the resulting eschar may include only the epithelium ; for, if the basement membrane be destroyed, there will be danger of subsequent adhesions between the eyelids and eyeball. The cauterisation should be repeated about every eight hours, or as soon as the eschar falls, and in the intervals, if the patient be awake, the conjunctiva should be gently sprayed or syringed every hour with a weak antiseptic lotion at a comfortable temperature. Diluted Condy's fluid, or solution (1 in 5,000) of mercuric chloride, or (15 per cent.) of boro-glyceride, is suitable for this purpose. The tension of chemosis may be diminished by radial incisions, outwards from the corneal margin, carried nearly down to the sclerotic. The strength must be supported by good diet and tonics ; and the nervous system calmed by anodynes. In milder cases the principle of treatment must be the same, but the local applications less severe ; and in chronic cases the local applications must be continued, after apparent recovery, so long as any residual thickening, capable of undergoing eventual contraction, can be found lurking in the palpebral folds.

(d) *Diphtheritic Conjunctivitis.*—This malady has chiefly been made known to us by the observations of German writers, and very few authentic instances have been recorded in this country. But it has from time to time prevailed extensively in Berlin, and might at any time make its appearance among ourselves. The writer has seen one remarkable instance in which the disease occurred with great severity in a child, whose eyes were bathed with milk as a domestic remedy for what seems to have been a mild ordinary conjunctivitis ; and the coincidence seems to have a special interest in relation to recent researches into the bacteriology of diphtheria.

SYMPTOMS.—Diphtheritic conjunctivitis is attended with great heat and pain, and with hard, brawny swelling of the eyelids ; but its most characteristic symptom is the infiltration of the sub-conjunctival tissue by effusion which does not form a pellicle upon the surface, but distends and fills the cavities of the areolar tissue. The result is to produce a pale, firm swelling of the conjunctiva, and a great tendency to rapid sloughing of the cornea. The subjects of the malady are mostly feeble and badly fed children, and the cases are

described as being almost hopeless unless early admitted into hospital.

TREATMENT.—The indications are chiefly to support the strength by suitable regimen; to apply ice or bags of freezing mixture to the lids during the hot stage; and to change the cold applications for hot ones as soon as the period of resolution, absorption, or repair can be said to have commenced. In the meantime iron and quinine should generally be administered internally.

2. *Pemphigus of the conjunctiva.*—A very formidable, but fortunately rare, variety of conjunctival disease appears to be a kind of pemphigus. See PEMPHIGUS.

ÆTIOLOGY AND SYMPTOMS.—In its early stage pemphigus of the conjunctiva displays itself by the formation of bullæ, which are of small size and inconspicuous, and are seldom followed by the raw surfaces commonly left elsewhere. This stage is followed by a slow process of conjunctival atrophy, which leads on to disturbance of the corneal epithelium, and ultimately to complete opacity. The conjunctival atrophy gradually obliterates the folds of the membrane, and renders the lids immovable upon the globe, frequently causing also inversion of cilia, which adds to the pre-existing irritation.

TREATMENT.—The only hope of dealing successfully with conjunctival pemphigus appears to depend upon early recognition and treatment of the general malady; for which purpose the chief reliance must be placed upon the preparations of arsenic, in combination with tonics and anodynes. When the period of atrophy has commenced, it is seldom, if ever, arrested. The apparent adhesions between the lids and the globe often suggest operative measures, but these are not recorded ever to have been successful. In one case, the writer dissected off the whole of the affected conjunctiva of the globe, and replaced it by transplanting that of a rabbit. The graft united well, and, for a time, the disease appeared to be arrested. In a few weeks, however, or as soon as the graft had become completely dependent upon the nutritive processes of the patient, the new structure became affected by atrophy, and the malady pursued its usual destructive course.

3. *Episcleritis.*—An affection which is not really conjunctival, being situated in the tissue between the conjunctival and the sclerotic, is that which has received the name of *episcleritis*, and which was formerly described as 'scleritis with inflammation of the insertion of a rectus muscle.'

SYMPTOMS.—It appears as an elevated patch of congestion, seated on the ocular surface near the corneal margin, most frequently on the temporal side. On close examination, the congestion, with the exception of a few dilated vessels, is seen to be sub-conjunctival, and to be attendant upon a circumscribed but not sharply defined swelling or thickening, which is adherent to the sclerotic, and which presents, in the interstices between the blood-vessels, an appearance as if it consisted of some new deposit, generally of a yellowish tint, external to that membrane. The swelling is indolent, chronic, and in itself generally painless, although it is sometimes accompanied by severe neuralgia. The subjects are most frequently women, especially such as are anæmic or otherwise out of condition. *Episcleritis* may last for months with little change, and it seems to be harmless as regards the other structures of the eye.

TREATMENT.—The writer has found *episcleritis*

resist all medication except the internal administration of mercury, to which it will often yield in the course of a short time. The best preparation for internal use is the perchloride, in doses not exceeding $\frac{1}{16}$ of a grain, which may usually be combined with five or ten minims of the tincture of perchloride of iron, and often with quinine. At the same time it is often desirable to sprinkle a little dry calomel over the swelling once in twenty-four hours; but this application is less important than the internal treatment, and should not be continued unless it is soon and distinctly beneficial. Recent Continental writers have advocated the sub-conjunctival injection of mercuric cyanide, combined with cocaine as a local anæsthetic in the manner described under *iritis*. When the congestion of the patch is considerable, the action of other remedies will often be promoted by the occasional application of a leech to the temple of the affected side.

4. *Hæmorrhage.*—Effusion of blood beneath the conjunctiva may occur spontaneously, but is generally traceable either to a direct injury or to violent exertion. Thus it may follow slight blows upon the eyeball, as from a twig or switch; or may be produced by a paroxysm of coughing, especially in pertussis; or may occur from the rupture of a vessel during parturition, or upon lifting a heavy weight. It is always unsightly. When traceable to any of the foregoing causes, the hæmorrhage is usually a matter of no moment; but when it happens during the night in young people, it should lead to a suspicion of nocturnal epilepsy, which has often been first discovered by its means. Moreover, in advanced life, more especially when occurring without manifest cause, it may point to arterial brittleness, of a kind which may indicate danger of a like hæmorrhage within the cranium. On these grounds it is a symptom which always calls for full inquiry into its causes, and which may sometimes afford warning of impending danger.

TREATMENT.—Absorption may be promoted by covering the closed lids by a compress moistened with a lotion of spirit and water, or of tincture of arnica and water if an appearance of more decided medication is desired.

II. Diseases of the Cornea.—Diseases of the cornea are often secondary to those of the conjunctiva, and may arise in their course as complications.

I. *Ulceration.*

ÆTIOLOGY AND SYMPTOMS.—To the present group belong the corneal ulcerations of purulent ophthalmia, whether infantile or of a later period; and also the ulcers which are produced by the friction of eyelids rendered rough by inflammation, or by the friction of eyelashes which have been turned inwards by distortion of the tarsal cartilage. When ulceration of the cornea occurs in the course of conjunctivitis, it invests the latter malady with a highly formidable character. The corneal tissue, once destroyed by ulceration, is not reproduced in its original transparency, but only as a more or less dense and opaque white cicatrix, which is both disfiguring to appearance and an impediment to vision. If the ulcer perforate, the iris almost necessarily becomes adherent to the cicatrix; and if the loss of substance be of large superficial extent, the resulting cicatrix is thin and feeble, so that it is rendered prominent by the pressure within the eye, producing the condition which has been called 'staphyloma,' and gradually elevating and distorting the surround-

ing portions of clear cornea. The first effect of the healing of a corneal ulcer is to flatten the natural curvature of the membrane ; but the secondary effect, if the cicatrix becomes prominent, may be to modify this curvature in various ways. Hence, even when a cicatrix of the cornea is surrounded by a still transparent annulus, behind which an artificial pupil may easily be made by the excision of a portion of the iris, the surgeon cannot predict with any certainty the quality of the vision which will be obtained, unless he is able, before operating, to determine the state of the corneal curvature. This is only possible when the margin of the pupil is so far free that it can be dilated with atropine sufficiently to render the fundus of the eye visible with the ophthalmoscope. When this can be done, any portion of cornea through which a clear view of the retinal vessels can be obtained by the surgeon will also afford clear vision of external objects to the patient ; but, if no such place can be discovered, a very cautious opinion should be given with regard to the benefit which may be hoped for from operation.

TREATMENT.—The importance of the cornea to the visual function renders it necessary that its integrity should be guarded with the greatest care. In any case of conjunctivitis, of even moderate severity, the cornea should be watched from day to day, and any appearance of turbidity about its central portion, or of elevation or irregularity of the epithelium at its margin, should lead to a reconsideration of the treatment. The former of these conditions is the ordinary precursor of sloughing ulcer ; the latter, of the extension to the cornea of an inflammatory process.

The general principles which govern the treatment of sloughing ulcer are, that the eye should be kept under the influence of eserine, which has a marked effect in checking the extension of ulceration by arresting the migration of white corpuscles ; that any astringents which may be applied to the conjunctiva should be prevented from coming into contact with the cornea ; that strength should be supported, pain relieved, and local nutrition stimulated by hot applications. When the ulcer continues to spread, its progress may often be arrested by diminishing the tension of the globe by the evacuation of the aqueous humour ; and this may be accomplished either by repeated paracentesis at the corneal margin, or by Saemisch's method of cutting through the base of the ulcer, and reopening the incision daily until a process of repair is established, or by the performance of iridectomy. Of these courses, the last-named is the most generally applicable. It not only produces the immediate effect which is desired, but it may also have the incidental advantage of establishing an artificial pupil at the side of the cicatrix.

The application of eserine is best effected by using a solution of the neutral sulphate, of the strength of four grains to the ounce of distilled water ; and a drop of this solution may be placed within the conjunctival sac twice or thrice daily.

The sloughing ulcers of the cornea which arise from causes other than conjunctivitis, as injury, or failure of nutrition, must be treated upon the principles which have already been laid down, but with constant reference to the liability of such ulcers to become the seats of bacterial infection, by which the healing process may be indefinitely delayed, or the extension of the ulcer indefinitely promoted. In such conditions, the ordinary principles of aseptic

surgery come into play, and the first indication is to cleanse and purify the ulcerated surface, and to protect it from further contamination. For this purpose it is generally important to define its precise boundaries, and this may be done by instilling into the eye a 2 per cent. solution of fluorescein in distilled water, by which the corneal tissue, wherever it has been deprived of its epithelium, will be stained of a bright green colour. The eye being effectively cocaineised, or, in severe cases or with troublesome patients, chloroform being administered, the entire surface of the ulcer should be cleansed, either by scraping with a sharp spoon or by the careful application of the heated wire of a fine electric cautery, and should be freely irrigated with a weak mercurial solution, say 1 in 5000 of the perchloride. The lids should then be closed, covered with a small piece of fine cambric lightly smeared with vaseline, padded with cotton wool, and the dressing sealed with a sufficient piece of isinglass-plaster. As a rule the dressing should not be removed for three or four days, when it will usually be found that the healing process has commenced, or even that it has been carried nearly to completion. Certain ulcers, however, will still require the use of local applications of a stimulating kind, among which dry calomel and other mercurials hold a prominent place. Such ulcers have usually a leash of vessels running from the corneal margin, and are often obstinately recurrent ; whence they are known as 'recurrent vascular ulcers.' They are usually connected with systemic derangement, and are often attended by photophobia. They leave scars of a size and opacity proportionate to their extent and depth ; and on this account it is desirable, whatever constitutional treatment may be required, to heal the ulcers themselves as speedily as possible, by the aid of local applications. In the early stages of the ulceration, eserine should be used ; but afterwards either dry calomel or a morsel of the unguentum hydrargyri oxidi flavi of the British Pharmacopœia. The application of any ointment to the eye is greatly facilitated by the use of a compressible tube, such as is used for artists' colours, from which a portion as large as a hempseed may be squeezed out upon the inner surface of the lower lid. Where there is much photophobia, the use of eserine is especially indicated, because, in addition to its action above mentioned, it produces contraction of the pupil, and thus gives comfort by lessening the quantity of light which is admitted into the eye. If the photophobia be very severe or intractable, it is often beneficial to divide the orbicularis muscle freely at the outer canthus, so as to diminish the pressure caused by its spasmodic contraction, which sometimes seems to be a chief cause of the irritability. After such an incision, a cold compress should be applied, and the patient kept in the dark for a few hours, when there will often be a marked alleviation of this distressing symptom, and a greatly increased general amenability to treatment.

2. *Inflammation.* — **SYNON.** : Keratitis. — In inflammation of the cornea presents three distinct types, the *suppurative*, the *vascular*, and the *interstitial*.

(a) *Suppurative Keratitis.* — Suppurative inflammation or abscess of the cornea seems to be essentially a phlegmon or boil of the corneal tissue, a portion of which dies, and is cast off in the form of a slough.

SYMPTOMS.—The abscess commences as a very tender grey spot in the cornea, surrounded by a zone

of turbidity, and accompanied by a good deal of ciliary neuralgia, as well as by a variable degree of lachrymation, conjunctival congestion, and intolerance of light. Under the influence of atropine, hot fomentations, and such constitutional treatment as the state of the patient may demand, the threatened suppuration is sometimes averted, and the turbidity clears away. More commonly pus is formed, and makes its way either externally, leaving an ordinary ulcer; or internally, producing the condition called *hypopyon*, in which there is pus in the anterior chamber. Sometimes it separates the corneal laminae, by gravitation, to a considerable extent before perforating them, and is then called *onyx*, from a resemblance to the lunula at the base of a finger-nail.

TREATMENT.—When suppuration is no longer doubtful, the best practice is to evacuate the abscess from within, by thrusting a cutting needle into the anterior chamber near the corneal margin, and then causing its point to penetrate the cavity of the abscess. The mingled pus and aqueous humour will escape as the needle is withdrawn, and its wound of entrance may be reopened once or twice daily by a probe, so as to insure the complete removal of all inflammatory products, until the healing process has made some way. If the abscess has burst internally before the case is seen, atropine should be applied without delay. If the pupil dilates fully, and the quantity of pus in the anterior chamber is small, the case may be left to the *vis medicatrix naturæ*, care being taken to enforce rest and to exclude noxious influences. If the quantity of pus is large, it should be let out by paracentesis; and if the atropine reveal adhesions of the iris, iridectomy should be performed. An abscess which has burst externally leaves simply an ulcer, generally with a disposition to heal readily, and requiring only such treatment as has already been described.

(b) *Vascular Keratitis.*—Vascular inflammation of the cornea is a severe and protracted malady, which usually leaves permanent opacity and impairment of sight.

SYMPTOMS.—It commences in typical cases, by the formation of two crescent-shaped patches of vascularity, one at the upper and the other at the lower portion of the cornea. The patches are formed by the development on the corneal surface of innumerable fine blood-vessels, so closely packed together that the interstices which separate them are scarcely discernible by the naked eye, and the affected parts present a uniform aspect of vivid redness. The crescents are somewhat elevated above the general corneal surface, and each crescent is bordered, along its concave or advancing edge, by a line of precursory epithelial turbidity. At the same time that the crescents increase in size, the borders of precursory turbidity are pushed before them; until at first these, and afterwards the crescents themselves, may meet and coalesce on the horizontal diameter of the cornea, so that its whole surface may become uniformly red. When this stage is reached, vision is almost abolished, but there may still be much intolerance of light. The corneal tissue is softened, and its margin is surrounded by a zone of sclerotic vascularity, which is visible through the congestion of the conjunctiva. As the inflammation subsides, the vascular crescents slowly recede from the centre of the cornea towards its circumference, and finally disappear, leaving behind them a dense opacity of an extremely obstinate character. A severe case of vascular keratitis generally affects both

eyes, and threatens a long period of actual blindness, followed by a long period of very imperfect vision. The worst examples of the malady are those which have been treated at the outset by astringent or irritating applications; and, even in the slighter forms, the malady is nearly always obstinate and protracted. As long, however, as the march of the vascular crescents, or their precursory turbidity, has not encroached upon the portion of cornea in front of the pupil, so long vision is not seriously jeopardised.

TREATMENT.—The great object of treatment is to arrest the new vascular development at a comparatively early stage; and for this purpose it is necessary to have recourse to eserine and sedatives locally, and to such constitutional treatment as the general condition may require. A solution of two grains of the neutral sulphate of eserine, with from two to four grains of hydrochlorate of cocaine, in an ounce of distilled water, should be dropped into the eye twice daily, and the closed lids should be frequently fomented with hot decoction of poppies, or with cold solution of extract of opium in water, according as one or the other temperature is the more agreeable to the feelings of the patient. The medicines most generally useful are those which appear to influence local nutrition through the central nervous system, such as the iodide and bromide of potassium, and the sulphate of quinine. These, if they are likely to exert a beneficial effect, generally do so speedily; and if the malady should be rapidly extending, it is prudent to reconsider the prescription without loss of time. In severe cases, especially when the lids are somewhat tumid and there is much photophobia, a leech may be applied with advantage, usually on the horizontal level of the outer canthus, and so far away from it as the growth of hair will allow, or blood may be taken more rapidly, by means of Heurteloup's artificial leech; and when, notwithstanding treatment, the malady pursues its course unchecked, a large iridectomy should be made without undue delay. The operation not only tends to arrest the vascular formation, but it also leaves a lateral pupil through which good sight may often be obtained long before the transparency of the central parts of the cornea is restored. In some cases, the writer has seen excellent results from peritomy; that is, from excising the annulus of conjunctival and subconjunctival tissue which immediately surrounds the cornea. The strip excised should be about 3 mm. in width; and the excision may sometimes be limited to the bases of the vascular crescents.

Development of vessels upon the cornea.—It is necessary carefully to distinguish vascular keratitis, properly so called, from that development of vessels upon the cornea which may occur in connection with the cicatrization of ulcers, or in consequence of the friction of lids left granular by conjunctivitis. In both these forms the new vessels are arborescent and irregular in their distribution, instead of being closely packed together; and they are not attended by the pink zone of circum-corneal congestion, which is never absent in true corneal inflammation.

The vessels which attend the formation of cicatrices generally dwindle in course of time; while those produced by granular lids consequent upon trachoma, —which, when they are very abundant and closely set, constitute the condition called *pannus*—will often disappear without direct treatment if the state of the lids themselves can be favourably modified by the

application of astringents or by other means. When trachoma-granulations are large and numerous, they may be treated by crushing them with forceps, by scraping, or by electrolysis; for which purpose they should be separately punctured by a platinum needle, connected with the negative pole of a galvanic battery. A weak current only is required, and it may be suffered to pass through each granulation for from ten to twenty seconds. In many cases the vascular network of pannus will be comparatively absent from the lower third of the cornea, so that sight may be much improved by an artificial pupil made in a downward direction. When pannus covers the whole cornea with a close vascular network, so that sight is almost destroyed, and when it resists milder treatment, it may sometimes be much improved by inoculation with the discharge of infantile purulent ophthalmia. The pus is inserted between the lids, and the artificial malady suffered to run its course unchecked, except by cleanliness and frequent bathing. When the discharge has ceased, the cornea will often clear in a surprising manner, and its abnormally vascular state protects it, to a very great degree, against the risk of sloughing. Still, this risk is by no means absent, and the treatment by inoculation should be regarded only as a last resource. A somewhat similar effect may be obtained by the inflammation excited by bathing the eyes with an infusion of the seeds of jequirity; but this method is scarcely less dangerous than that by inoculation.

(c) *Interstitial Keratitis*.—This is a chronic malady which is seen chiefly, or perhaps exclusively, in the subjects of inherited syphilis, who possess the peculiar teeth and *facies* which Hutchinson has shown to be characteristic of their inheritance. The disease was long described as a variety of 'strumous ophthalmia;' and, although the late Sir William Wilde pointed out how frequently it was associated with deafness, and also laid stress upon the value of perchloride of mercury in its treatment, its syphilitic character seems to have been first suspected by Hutchinson, who, having once obtained the clue, followed it with characteristic diligence until he arrived at a conclusive demonstration of the accuracy of his suspicion.

SYMPTOMS.—Interstitial keratitis commences as a slight cloudiness of the central portion of the cornea, with some roughening or irregularity of the epithelium. It extends from the centre towards the margin, and is liable to be attended, in different cases and at different stages of its course, by variable degrees of ciliary and corneal congestion, and intolerance of light. If neglected, or if treated by irritants, it is liable to assume the characters of vascular keratitis, and also to extend to the iris, in such instances often doing irreparable mischief. It is most common during childhood, but its appearance may be delayed until adolescence, or even until adult age; and, in such cases, usually appears to be determined by some general or local exciting cause, such as bodily illness, or direct injury. It attacks both eyes, one commonly later than the other, and its course is often protracted over months. When severe, it leaves some residual cloudiness of the cornea, and, even when mild, it is doubtful whether the cornea ever entirely regains the transparency of health. Still, when a case is seen and judiciously treated early, the prognosis may generally be a favourable one.

TREATMENT.—The treatment consists primarily in the avoidance of all irritants; the use of eserine

and local sedatives, especially cocaine; and the administration of perchloride of mercury, or of iodide of potassium, with or without iron or cod-liver oil. When they are not contra-indicated by any special circumstances, the perchloride of mercury and the oil are the remedies on which the greatest reliance may be placed. The earliest indications of photophobia, showing, as they do, that light is acting as an irritant, should be met by confinement to an almost darkened room, and by frequent bathing of the closed lids with cold water; but in fine weather daily exercise should be taken in the open air, the eyes being covered for a time with a black silk bandage and compresses of carded wool, so as to exclude light entirely. As soon as photophobia subsides, these precautions may be laid aside—the eserine and mercury being still continued, at least until the acute stage of the malady has entirely passed away. After this the absorption of residual opacity may be promoted by the application, once daily, of a small quantity of the ointment of the yellow oxide of mercury.

In cases which have been neglected, or aggravated by irritants in their early stages, and in which the phenomena of ordinary vascular keratitis become grafted upon the interstitial, it is generally desirable to perform iridectomy with as little delay as possible.

3. *Arcus Senilis*.—The condition thus named (although the adjective is not always appropriate) is fully described elsewhere (*see* ARCUS SENILIS). It may be distinguished from the peripheral zones of opacity, which are sometimes left after the subsidence of certain forms of keratitis, by the circumstance that arcus never extends quite to the margin of the cornea, but is always surrounded by an annulus of transparent tissue.

Besides the foregoing, there are other forms of corneal disease, of rare occurrence, which it would be beyond the limits of these pages to describe, but which must be treated on the same principles; and there is also the malformation known as 'conical cornea,' which falls wholly within the domain of surgery.

III. Diseases of the Sclerotic.—Diseases of the sclerotic, which were once regarded as a numerous and important group, have been reduced by recent investigations to comparatively insignificant proportions.

Inflammation.—**SYNON.**: Scleritis.—Excepting a narrow annulus immediately around the cornea, the sclerotic is almost non-vascular; and any real inflammation of its structure is almost confined to this particular region, where it seldom occurs excepting as a complication or as a part of some of the more severe forms of iritis or keratitis, especially when either of these affections extends to the ciliary body. In such cases we often see the sclerotic undergo inflammatory softening, as a result of which the ciliary region may be much altered in shape, yielding to the distension of the eyeball and to the traction of the recti muscles, and becoming distinctly elongated. Occasionally the sclerotic may be so much softened and thinned as to bulge into irregular prominences around the cornea, generally under the upper lid; and this condition is described as *sclerotic staphyloma*.

TREATMENT.—The inflammations thus arising call for no other treatment than that which is demanded by the more important inflammations of the cornea, the iris, or the ciliary body, with which they are associated; except that any evidence of yielding

of the sclerotic would be a reason in itself for the performance of iridectomy, in order to preserve the shape of the eyeball by diminishing its tension. It was once believed that the sclerotic, in its character of a fibrous membrane, was especially prone to gouty or rheumatic inflammation; and it is perhaps true, though certainly not proven, that the tendency of iritis or of keratitis to spread to the anterior sclerotic zone is more marked in persons of gouty or rheumatic diathesis than in others. The possibility is at any rate sufficient to require, in all these cases, an investigation of the tendency to lithic-acid formation, and the use of appropriate remedies when this tendency is discovered. But a large proportion of the examples of supposed gouty or rheumatic ophthalmia are nothing more than cases of the sub-acute or remittent form of glaucoma; and the pain associated with them is not really rheumatic but simply tensive. Vision has been irretrievably lost, in hundreds of instances, because a belief in the rheumatic character of these affections has interfered with the timely performance of iridectomy; and the proposal to adopt the epithet 'rheumatic,' in any form of eye-disease, is one which should be scrutinised very closely before it is accepted as a guide to practice.

IV. Diseases of the Iris.—In so far as they come into the province of the physician, diseases of the iris are not numerous, and are almost limited to the varieties of inflammation of that membrane.

1. Inflammation.—SYNON.: Iritis.—Iritis may be classified, according to its actual or supposed causes, as rheumatic or syphilitic; or according to the nature of the morbid process, as *plastic*, *serous*, or *suppurative*. The former classification often rests upon slender grounds, and the latter has the advantage of expressing facts rather than inferences.

(a) *Plastic Iritis.*—SYMPTOMS.—In plastic iritis the first symptom is some loss of the natural lustre of the surface of the iris, and of the clear definition of its fibres, together with some damping or alteration of its colour. These changes are attended by some impairment of vision, and are due to turbidity of the aqueous humour, so that they may be imitated more or less closely by turbidity of the cornea, and especially by disturbance of its epithelium. In iritis, however, they are associated with a diminished range and quickness of pupillary variation under variations of light; and, in a short time, with the effusion of plastic lymph, by which the margin of the pupil becomes tied down, here and there, to the surface of the anterior capsule of the crystalline lens. At the same time there is some congestion of the conjunctiva, and of the zone of fine vessels immediately around the cornea in the sclerotic. There is frequently more or less pain, especially towards night, but this is a very uncertain symptom. In severe cases, and especially in such as are clearly syphilitic in their character, the quantity of effused lymph may be considerable, so as to cover the pupil, while in mild cases it is only sufficient to fasten down the margin here and there. Iritis is sometimes a very insidious and seemingly slight affection, the real gravity and importance of which may be wholly overlooked by the patient.

TREATMENT.—The first principle of treatment is to prevent the formation of adhesions, or to break them if they have been formed; and for this purpose our main reliance must be placed upon the instillation of atropine. The anatomical structure of the eye is such that a moderately contracted

pupil is in contact with the lens-surface, while a fully dilated pupil is separated from it by a film of aqueous humour. Hence, as long as the pupil is contracted, any lymph which is effused tends to the immediate formation of adhesions; while, as soon as the pupil is dilated, the lymph diffuses itself harmlessly in the surrounding fluid, and no adhesions are produced. In ordinary circumstances, and in cases of only ordinary severity, the iritis then runs its course without inflicting any permanent injury, and vision is completely restored as soon as resolution has taken place. When, on the contrary, the pupil cannot be dilated, the lymph deposited in the area of the pupil forms an impediment to vision; and the adhesions themselves tend to render the iritis a recurrent affection, which is apt to return again and again until the eye is disorganised and destroyed.

The first principle of treatment is, therefore, to produce and maintain dilatation of the pupil; and for this purpose it is necessary to use some active mydriatic. The neutral sulphate of atropine is usually the most convenient agent for the purpose, and it should be used in a solution containing four grains to the ounce of distilled water. Of the pure watery solution, a drop should be carefully placed within the lids by a dropping-tube or quill, repeated in five minutes, and again in another five minutes, and this threefold application should be repeated three times a day. A mydriatic agent still more powerful and more rapid in its action than atropine is *duboisine*, which should be used in a watery solution, of the strength of four grains to the ounce; and it has less tendency than atropine to produce local irritation. The hydrochlorate of cocaine may be combined with either of the foregoing, and will not only assist their mydriatic action, but will also promote the comfort of the patient. If the pupil can be fully dilated in twenty-four hours, no other treatment will be necessary than to maintain the dilatation by using the mydriatic less frequently, and to protect the eye from being injured by exertion or by exposure to great variations of temperature or of light. If, on the contrary, after the use of atropine or duboisine for twenty-four hours, the pupil either remains contracted or dilates irregularly, showing that it is bound down here and there, it is necessary to have recourse to mercury without further delay, and to use one of the preparations, such as blue pill, calomel, or inunction with mercurial ointment, with which the effect of the medicine upon the system can be rapidly secured. Abadie of Paris, and some other continental authorities, have strongly recommended the employment of mercurial sub-conjunctival injections in cases of iritis, but the severe pain attending the method was at first a serious objection to its use, and the good results claimed for it have been disputed in many quarters. The objection arising from pain has to a great extent been removed by the practice, introduced by Abadie, of using a combined solution of cocaine and of mercuric cyanide, of which, if the conjunctiva be first cocaineised, as much as half a syringeful may be injected into the deeper sub-conjunctival tissues with no other inconvenience than a feeling of distension. Equal parts of the two solutions should be employed, the mercuric being of 2 per 1,000 and the cocaine of 2 per cent. The solutions should be recent, should not be mixed until the moment of injecting them, and asepsis should be carefully preserved. There is never any

occasion to carry the effect of the mercury farther than to the formation of a slight line upon the gums; and, in most cases, as soon as this line is perceptible, a notable amelioration of the eye-symptoms will be observed. It is desirable, however, that this degree of mercurial influence should be reached quickly, in order to cut short the disease as soon as possible; and when it has been reached, it will usually require to be maintained, for some days at least, by the administration of smaller and less frequent doses. In favourable cases, under the combined influence of mydriatics and mercury, the effused lymph will be absorbed, the adhesions broken through, and the eye restored to its original condition. In those of a less favourable character, the inflammation will, indeed, subside; but the adhesions will remain, and the pupil will be left permanently more or less crippled and distorted. Whether or not sight will be impaired will chiefly depend upon whether the effused lymph has formed a film or membrane across the pupillary opening. In the worst cases, notwithstanding treatment, the inflammation may extend to the ciliary body and choroid, and may produce functional destruction of the eye. This scarcely happens except when the iritis has been of great original severity, and when it has been neglected, or aggravated by irritants, in its early stages. Even the suspicion of iritis should absolutely preclude the use of the astringent applications on which we have mainly to rely in the treatment of the inflammations of the conjunctiva.

If an iritis be not seen until it has been three or four days in existence, so that the adhesions have had time to acquire a certain degree of firmness, it is not desirable to wait twenty-four hours before having recourse to mercury. The mineral should be employed without further delay, so that it may be abandoned if atropine should dilate the pupil, and may be pushed if dilatation cannot be produced.

While the atropine, or atropine and mercury, are being employed, the remainder of the treatment must be governed by general considerations. Rest of the other eye must be strictly enforced; local depletion may be practised whenever the congestion is considerable in degree; and such a regimen and mode of life must be prescribed as the patient can bear. Unless there be photophobia, it is seldom or never necessary to exclude light altogether from the eye; and, when photophobia is present, it is better to apply a protective bandage than to keep the patient in a dark room. The latter practically excludes him from cheerful companionship, and leaves him to dwell upon his troubles in darkness and solitude. Pain, if present notwithstanding the use of cocaine, should always be subdued, either by combining a sufficient dose of opium with the mercury, or by the subcutaneous injection of a solution of morphine. However the anodyne is administered, provision should be made for repeating it sufficiently often to produce and maintain the desired effect.

When recovery takes place leaving adhesions, these will, in the majority of cases, lead to a second attack of iritis, and this is almost always the predecessor of regular recurrence. In a few cases, however, the second attack does not follow; but, whenever it occurs, the tendency to future mischief should be stopped by surgical means, either by the detachment of the adhesions or by the performance of iridectomy.

(b) *Aplastic Iritis*.—The aplastic form of iritis

differs from the plastic in the greater quantity and more liquid condition of the effusion, which does not form adhesions, but distends the eyeball and compresses its contained structures. In a well-marked case the pupil is contracted and insensible to atropine (which in all probability is not absorbed); the aqueous humour is turbid; the iris is pushed back and its anterior surface appears concave; vision is greatly impaired; and the eyeball is perceptibly hardened to the touch.

TREATMENT.—Until the distension is relieved, no remedies will be effectual; and, when it is relieved, they generally cease to be needed. The treatment should be either by frequently repeated paracentesis of the anterior chamber or by a large iridectomy or sclerotomy, and the latter is generally to be preferred. Aplastic iritis occurs chiefly in persons of unhealthy or broken-down constitution; which may perhaps account for the unorganisable character of its products. As soon as the distension of the eyeball is relieved, the pupil is readily dilated, and the iritis soon subsides.

(c) *Suppurative Iritis*.—In a small number of cases, iritis assumes from the first a suppurative character, and leads to the formation of pus in the anterior chamber.

TREATMENT.—Such a condition calls for atropine; for stimulating and tonic medicines rather than for mercury; and for the evacuation of the pus by paracentesis if it is considerable in quantity, or if its presence appears to be a source of increased irritation.

(d) *Serous Iritis*.—This term, which would perhaps be more appropriately used for the aplastic form of iritis, has lately been applied to a form of disease which was described in old books as ‘aquocapsulitis,’ and in which the posterior surface of the cornea participates with the iris in the inflammation.

ETIOLOGY AND SYMPTOMS.—Serous iritis usually first attracts attention by occasioning a slight dimness of vision. There will often be no congestion, or scarcely any, but the movements of the pupil will be sluggish, and careful examination will discover one or more fine points of adhesion to the anterior capsule. But the most characteristic appearance of the disease is situated upon the lining membrane of the cornea, which will be found covered by a number of minute circular dots, ranged in a pyramidal outline. The base of the pyramid corresponds with the lower corneal margin; the apex encroaches more or less upon the pupillary region. As the disease progresses, the dotted pyramid increases in density and conspicuousness, and the pupillary adhesions increase in number and extent. The malady usually runs a very chronic course, and often leaves some permanent diminution of vision. In a certain proportion of cases it seems to be independent of any known cause; in others it follows some constitutional affection (the writer has seen a very severe case, in a young lady, consecutive to mumps); in others it is associated with constitutional syphilis. After injury to an eye, serous iritis sometimes appears in the other as the first stage of sympathetic ophthalmia.

TREATMENT.—This will include, in the first place, careful consideration of the state of the general health, and endeavours to correct whatever may be amiss. The eyes should be kept at rest, the pupils dilated but protected from bright light, and mercuric chloride, or iodide of potassium, or both, may be given in suitable doses, the efficacy of these remedies

being manifest in the non-syphilitic as well as in the syphilitic subjects of the disease. Where the adhesions are numerous, iridectomy may be required to relieve dragging. The cases are usually chronic, and many months may elapse before the eyes are restored to usefulness.

2. *Inflammation of the ciliary body and choroid.*—**SYNON.**: Cyclitis; Irido-choroiditis.—In some instances iritis is not confined to the membrane in which it originates, but spreads backwards to the ciliary body and the choroid. The most marked examples are those in which the original inflammation has been excited by morbid changes, resulting sometimes from disease, but more frequently from injury, in the opposite eye; and these cases are called *sympathetic ophthalmia*. They have been supposed to depend upon the propagation of peripheral irritation through a nervous centre, and to point to the presence of some nerve-irritation or functional failure, as the essential difference between the iritis which dies out as a localised affection, and that which spreads by continuity to the deeper parts of the eye. Recent observers have endeavoured to connect the phenomena with the presence and the migrations of microbes; but, so far, with no conclusive results. However occasioned, the issue of declared irido-cyclitis or irido-choroiditis is frequently disastrous; for the inflammation is always of a plastic character, and the effused lymph is scarcely ever absorbed in time, or with sufficient completeness, to prevent its contraction from inflicting profound injury upon the visual apparatus. The perceptive layer of the retina is not only in contact with the choroid, but the rods and cones derive the materials of their nutrition from the chorio-capillaris; and hence, as regards these delicate structures, an inflammation of the choroid, upon which they are directly dependent, is of far greater importance than an inflammation of the retina itself, which may be limited to the connective tissue of the fibre layer, and may leave the percipient elements almost unaffected.

SYMPTOMS.—The first sign of the extension backwards of iritis is usually furnished by tenderness of the ciliary zone, so that this region feels acutely the slightest touch of a probe, or of the end of a rolled paper spill, which is a more delicate instrument for testing the sensibility of the ocular surface. At the same time there is always a greater degree of impairment of vision than the iritis alone will explain, together with increased general congestion of the eye, and, in many cases, with an appearance of visible vessels on the iris.

TREATMENT.—The treatment to be pursued does not differ materially from that which is required by the more severe forms of simple iritis; and consists of depletion from the temple, generally by means of Heurteloup's leech; the application of atropine; the prompt and sustained administration of mercury, both internally and by inunction or injection; the control of pain; and the maintenance of strength. In sympathetic cases, the eye in which the mischief originated should be removed, even although the usefulness of this proceeding is somewhat doubtful when once the secondary affection is established. It is often necessary to continue the use of mercury for a long period, and it is chiefly when this has been done that some amount of vision is saved out of the wreck. The lens, in such cases, often becomes coated by lymph, and ultimately requires removal; and it may be necessary to perform iridectomy for

closure of the pupil more than once, in consequence of the tendency of the artificial opening to be drawn together again by contraction. On account of the severity of sympathetic ophthalmia, and of its generally unfavourable termination, it is a rule of practice to anticipate its occurrence, and to remove a diseased or injured eye which is likely to produce it, before the mischief has been done.

V. **Diseases of the Crystalline Lens** are separately treated of under the article CATARACT.

VI. **Glaucoma.**—In its modern signification, this word is applied to denote all the conditions produced by a morbid increase of tension within the eye—that is to say, by an excess of its contained fluids; and the different forms of the affection are mainly due to differences in the rate at which the tension increases.

SYMPTOMS.—If the increase be rapid, the loss of sight will be rapidly produced, and will be associated with other changes occasioned by sudden interference with the circulation and innervation, and by sudden stretching of the ocular tunics. When, on the other hand, the increase of tension is very gradual, so that the eye has time to become accustomed to the new conditions as they are produced, the symptoms often present a deceptive resemblance to simple atrophy; and these cases were at one time described as 'atrophy with excavation of the optic nerve.' In some of the more acute forms of increased tension, the pupil presents a clouded aspect of a greenish colour; and it was to cases of this class that the word glaucoma (from *γλαυκός*, sea-green) was originally applied, at a time when the pathology of the condition was not understood. When this pathology was rendered clear, and when it became known that the glaucoma of the ancients was in all essential respects identical with cases in which the most manifest symptoms were of a different kind, the word was retained as a convenient general term, to express states of disease to which it had no longer any apparent reference; and hence we still speak of glaucoma, although, in the great majority of the cases in which we do so, the green aspect of the pupil is conspicuous by its absence.

The normal tension of the eye appears to be maintained, in the face of continued internal secretion of fluid, mainly by the transudation of this fluid, at the so-called 'filtration-area,' in the angle formed between the cornea and the iris, into the plexus of veins contained in the canal of Schlemm. In order that this transudation may be free, it is necessary that the natural permeability of the tissues should be preserved, that the angle should be open, and that the access of fluid to the filtration-area should be unimpeded. In certain relations of the lens to the neighbouring structures, and, possibly, in consequence of increased bulk of the lens in advancing life, the periphery of the iris may be so pushed forward towards the cornea as to produce partial closure of the angle and impeded access of fluid to the filtration-area. Such conditions tend necessarily to increase; and they are liable to be increased still further by dilatation of the pupil, which implies thickening of the peripheral portion of the iris, and the occupancy, by this portion, of an abnormal amount of space. At a later period, as the tension continues to increase, the peripheral portion of the iris may even be applied to the margin of the cornea after the fashion of a valve, and transudation may be completely arrested.

The estimation of increased tension by the fingers is a matter which requires the *tactus eruditus*, and is best accomplished by directing the patient to close the eyes gently, and to cast them downwards. The tips of the two forefingers should then be placed upon the upper lid immediately below the margin of the orbit, and, when one finger has fixed the eye by holding it gently back against the orbital contents as far as it will recede, the other estimates the degree in which it may be dimpled by slight pressure. This degree varies to some extent in different persons within physiological limits, but a morbid increase of tension can scarcely be missed if it be looked for. Moreover, the diagnosis of glaucoma does not rest upon increased tension alone, but upon the combination of increased tension with decreasing sight. The rate of slow increase which may simulate atrophy, or of rapid increase which may simulate inflammation, is a matter of detail which should not be suffered to obscure the true nature of the morbid process.

The symptoms of glaucoma depend upon the more or less gradual interference with the ocular circulation, and upon the compression of the ocular nerves, by reason of increasing pressure within the eye. In consequence of the interference with the circulation, the blood enters and leaves the eyeball with difficulty. The first indication of this difficulty is afforded by the small veins which pierce the anterior portion of the sclerotic near the cornea, and course backwards beneath the conjunctiva towards the equator. These veins, which are invisible, or at least inconspicuous, in health, become distended and tortuous when the impediment to the venous outflow reaches a certain degree. At the same time, the ophthalmoscope will usually discover pulsation of the retinal vessels upon the optic disc, a pulsation first observed in the veins, and afterwards also in the arteries. It would seem that the contents of the veins are pushed back by the entering arterial current, and that the latter is only able to make good its way at the acme of the pulse-wave.

The symptoms which depend upon compression of the nerves will vary, of course, with the functions which these fulfil. Commencing with ordinary sensation, a moderate compression of the filaments derived from the fifth produces diminished sensibility of the corneal surface; while a pronounced increase of tension, especially if rapidly produced, occasions severe pain by the stretching of the ocular tunics. Proceeding to the motor filaments, compression of those derived from the third nerve produces paresis of the ciliary muscle and of the sphincter pupillæ; the former evidenced by rapidly increasing impairment of accommodation, the latter by sluggishness and dilatation of the pupil, the dilatation usually moderate in degree, but often causing the aperture to assume an elliptical outline, with its major axis horizontal. This pupillary dilatation is apt to be increased by the forward pressure of the crystalline lens; and, however produced, it tends, in the way already described, to occasion a still further increase of tension. The compression of the retina causes impairment of visual function, first manifested in the portions most remote from the central blood-supply; and hence taking the form of contraction of the field of vision, usually first displayed and most marked on the nasal side. Contemporaneously with this, central vision will be more or less reduced; and, at the same time, the increasing intra-ocular

pressure will occasion yielding from within wherever such yielding is possible, and hence chiefly over the area of the optic disc, the structures composing which will be to some extent pushed back out of the eye, leading to the formation of a 'cup' or excavation, which is one of the most conspicuous of the ophthalmoscopic signs of heightened tension. Finally, the general nutrition of the eyeball will suffer, in a manner which is often first displayed by the surface epithelium of the cornea, which is apt to become disturbed, irregular, or, as it were, 'steamy,' presenting an aspect somewhat as if it had been breathed upon, and constituting a cause of further serious impairment of the already diminished vision.

Such being the chief symptoms, one or more of which will be recognisable by examination in glaucoma, those complained of by the patient are often exceedingly characteristic. Glaucomatous tension is apt to increase in waves, separated by intervals in which it may decrease, or in which the structures pressed upon acquire some tolerance of the new conditions. We therefore often hear complaints of sudden 'clouds' or 'obscurations' of vision, which pass away after a few minutes or a few hours, and return at uncertain intervals. The intervening recovery in such cases is seldom complete; each 'cloud' usually leaving the sight permanently worse than before. The depth of the cloud, the period of its duration, and the presence or absence of pain, are conditions which will be governed by the extent and the rapidity with which tension has increased. When the cloud has passed away, we shall usually find diminution of central vision, contraction of the field, especially on the nasal side, and sluggishness of the rather large pupil. At the same time, it will often be found that the sensitiveness of the corneal surface is diminished; and the patient will often admit, on inquiry, that artificial lights of feeble intensity—candle-flames, for example—are frequently surrounded by coloured halos. But the combinations and the order of occurrence of symptoms will differ widely in different cases; and all that can be called essential, in order to constitute glaucoma, is a combination of impaired central vision and diminution of the field, with increased tension discoverable by the educated finger. When this combination is present, there should be no doubt as to the diagnosis, as there will certainly be no time to waste in inefficient treatment.

TREATMENT.—The treatment of glaucoma is entirely surgical, the affection being capable of arrest by iridectomy or sclerotomy in the great majority of cases. In the more acute forms, an operation, if performed sufficiently early, usually restores vision to its integrity; but the time during which this can be done is limited, and in the more chronic forms an operation will seldom do more than preserve what amount of sight is still retained. Hence it is important that treatment should not be delayed by any error of diagnosis; and the points to which attention should chiefly be directed, in any case of impairment of vision in which the question may arise, are increasing hardness of the eyeball as determined by palpation, and gradual contraction of the field of vision. Whenever the eye is becoming harder, and the circumferential extent of vision is narrowing in, the case is one in which an operation should be accomplished with as little delay as possible. At the same time, until the operation can be performed, a two-grain solution of eserine should be applied every four hours; this drug having

a marked effect in controlling and diminishing tension, and being apparently indebted for this power to its mechanical action upon the iris, which, by the contraction of the pupil, is thinned out and drawn away from the filtration-area. It is hardly necessary to add that in any case of actual or threatened glaucoma, the use of a mydriatic would be highly dangerous. The more acute cases of glaucoma are attended by much pain from the distension of the ocular tunics, and often by congestion and inflammation, the results of this distension; and such cases were at one time described as 'acute internal arthritic ophthalmia,' or by some similar name. Vision has been irretrievably lost, in hundreds of cases, by endeavours to control this form of glaucoma by medical treatment, to the neglect of operation, and the erroneous practice has been kept alive by the circumstance that some of the cases will undergo partial and temporary amendment. In such circumstances, however, the vision never rises to the degree of acuteness which existed prior to the attack, and the amendment is never more than temporary. Another increase of tension soon occurs, and, without surgical aid, blindness sooner or later closes the scene.

VII. Diseases of the Optic Nerve and Retina.

As shown by the ophthalmoscope, these diseases cover a very wide field of pathology. In order to understand them accurately, it is necessary to bear in mind the anatomy and relative arrangement of the affected tissues. The optic nerve in the orbit is invested by a double sheath, and the interval between the layers of this sheath, which is continuous with the sub-arachnoid space, terminates in a *cul-de-sac* towards the eyeball, the two layers becoming intimately united where they blend with the sclerotic. At this point, the opening in the sclerotic for the admission of the optic nerve is crossed by a film of perforated connective tissue, the *lamina cribrosa*, with which the sheaths of the nerve-fibres blend, or in which they are lost, so that the nerve anterior to the lamina consists of a bundle of unsheathed fibres, enveloping the central artery of the retina, which enters the eye with them, and the central vein of the retina, which passes out in the same position. On entering the eye the nerve-fibres bend round to form the anterior layer of the retina, which contains also the retinal blood-vessels almost to their ultimate divisions, together with some delicate connective tissue. The capillary circulation of the nerve itself is derived from the anterior cerebral artery, and is distinct, save for a very slight amount of anastomosis, from the capillary circulation of the sheath, which is fed by the arteries of the pia mater. It follows that hyperæmia of the sheath, or of the circle surrounding the nerve, may exist without hyperæmia of the proper nerve-tissue, and it has been supposed that fluid pressure in the intervaginal space may interpose an obstacle to the circulation in the vessels which pass through the sclerotic foramen, and may thus occasion dropsy of the termination of the nerve within the eye. Of the two foregoing conditions, either may undergo resolution harmlessly, or may produce such changes in or around the nerve as to occasion atrophy and loss of sight. Neither of them interferes with sight directly, because the circulation may be seriously disturbed by a degree of pressure which is insufficient to stop the conduction of impressions through the nerve-fibres. There is yet a third condition, properly called optic neuritis, or *neuritis descendens*, in which the capillary network of the

nerve itself participates in changes propagated downwards, and in which impairment of vision holds a very early place among the symptoms.

1. *Perineuritis*.—The true perineuritis, in which the unchanged nerve-disc is surrounded by a zone of high vascularity, is only seen as a result of meningitis, which may be either tubercular or due to other causes. It was at one time hoped by Bouchut, by whom this especial phenomenon was first described, that perineuritis might serve as a diagnostic sign in cases of doubtful meningeal inflammation; but this expectation has not been realised. The perineuritis itself has only been observed in cases the character of which was scarcely doubtful, and which, in most instances, have terminated fatally. Very probably, however, it would be found, if looked for, in those cases of exanthematous and other fevers which are attended by cerebral symptoms, followed, as a rule, after recovery by impairment of vision. In these we eventually find a partial nerve-atrophy, which does not lead on to complete blindness, but which does not appear to be susceptible of improvement; and this partial atrophy may no doubt be due to the pressure of perineural exudation during its contracting stage.

TREATMENT.—The treatment of perineuritis must generally be that of the affection in which it has its origin; but, in any fever in which this symptom had been detected, it would be a question whether mercury should not be cautiously administered for a considerable time after convalescence, in order to promote the absorption of any effused lymph by which the optic-nerve entrance might be constricted.

2. *Choked Disc*.—SYNON.: Dropsy of the Optic Nerve Entrance.—This is a condition chiefly seen in connection with intracranial tumours, whether syphilitic, tubercular, or of some other kind; and it almost invariably affects both eyes. It was formerly supposed to be due to the pressure of fluid driven down the intervaginal space around the nerve, and so constricting the latter at the terminal *cul-de-sac* as to impede the outflow of venous blood. This hypothesis is not now universally accepted, but the condition visible with the ophthalmoscope is one of distension and tortuosity of the retinal veins; arrest of the capillary circulation; impediment to the arterial inflow; dropsical effusion into the disc-tissues; and, sometimes, secondary inflammatory changes, such as effusion and cell-proliferation. All these changes may exist without impairment of vision, because they neither affect the perceptive layer of the retina, nor arrest the conduction through the nerve-fibres. At a later period, however, when any plastic elements in the exudation begin to undergo contraction, the interference with the circulation becomes more complete, the nerve-fibres themselves become compressed, and then failure of sight commonly occurs. At the same period, the nerve passes into a state of atrophy from the interference with its circulation; and these cases of nerve-atrophy were once sources of great perplexity to surgeons, and were frequently referred to causes which probably had very little to do with them. In consequence of the sight not being affected during the preliminary stage, the occurrence of this stage was for a long time absolutely overlooked, and was only discovered when physicians began to use the ophthalmoscope, as an ordinary instrument of diagnosis, in all cases of cerebral affection. It was then established that the

atrophy had been preceded by swelling of the disc and by obstruction of its circulation; and it was shown that the cases which had this history were often recognisable, after the swelling had passed away, by the tortuosity of the retinal veins which was left behind, and by the way in which these veins were lifted into bold curves at the margin of the disc; this alteration of their original course having been due to the swelling, and remaining after the swelling had subsided. The contraction of the effusion, and the consequent atrophy of the nerve-fibres and closure of the capillary vessels, would be likely to occur earlier in some parts of the disc than in others; and hence, at the time when commencing failure of sight first induced the patient to seek advice on account of it, the disc was commonly seen to be invaded by sectors of whiteness but to retain its vascularity, or perhaps more than its normal vascularity, in other parts; while, at the same time, the sight was first lost in those regions of the retina the fibres from which were first compressed. Hence it follows that a partial invasion of the disc by atrophic changes, and a partial invasion of the field of vision by blind portions, are among the earliest symptoms in cases of the class under consideration; and these symptoms were at one time referred rather to the nervous centres than to the retina or the disc itself, to changes in which they are now attributed. A not uncommon clinical history in such cases is that there has been constitutional syphilis, imperfectly treated and ultimately producing headache or other cerebral symptoms, which have probably called for the administration of iodide of potassium and have been relieved by it. Some weeks afterwards there is for the first time a complaint of failing sight; and then the ophthalmoscope reveals that the discs are passing into atrophy, that the retinal veins are lifted into prominent curves at the disc-margins, and that their further course over the retina is generally serpentine. In many cases, the recovery of the patient, as far as general health is concerned, leaves the precise character of the intracranial mischief doubtful; but, in fatal cases, a tumour is the morbid condition most frequently discovered. See OPHTHALMOSCOPE IN MEDICINE.

When the merely passive dropsical effusion into the disc becomes complicated with inflammatory changes, the sight begins to fail before signs of atrophy become manifest; and such cases are difficult to distinguish from those in which there is *primary or descending neuritis*. The blood-supply of the optic nerve being derived from the anterior cerebral artery, we may reasonably expect to find capillary engorgement of the nerve-substance of the disc in connection with arterial hyperæmia of the brain; and this capillary engorgement may pass into inflammation, either of local origin or by transmission downwards from above. In any case, if the first changes in the disc are of the character of neuritis rather than of obstruction, we see capillary or arterial hyperæmia of the nerve-substance rather than venous congestion; and effusion of plastic material upon the disc itself, with comparatively little prominence or disc-swelling, and with comparatively little extension over the disc-margins upon the surrounding retina. At the same time, even in the early stage of the affection, we find great impairment of sight, the conducting power of the nerves-fibres being seriously injured. Such cases are frequently syphilitic, and, unless the

absorption of the effusion should be quickly brought about by treatment, its contraction, like that of the effusion of obstruction, soon occasions atrophic changes. In these cases, however, the swelling having been absent or inconsiderable, the vessels do not show that elevation into bold curves at the disc-margin which has already been described; and the contraction being interstitial in the nerve-substance, and from the first affecting veins and arteries in an equal degree, the arterial inflow is diminished *pari passu* with the diminution of the vein-channels, and the latter vessels are seldom distended in such a manner as to render them distinctly varicose or tortuous. The ultimate result is a white disc, on which the arteries and veins have dwindled to threads, or at least are greatly reduced from their normal calibre.

We may therefore have three conditions which in their typical forms are tolerably distinct, but which are prone to run into one another by almost imperceptible gradations, and which may all lead on to atrophy and complete blindness: namely, perineuritis, neuritis, and choked disc. The liability to the last-mentioned condition should be carefully remembered by physicians, and should lead to careful ophthalmoscopic examination in all cases or obstinate headache or other cerebral symptoms of obscure origin, more especially in a patient with a syphilitic history.

TREATMENT.—It is manifest that the best hope of preventing ultimate blindness in persons in whom choked disc has occurred will be secured by the administration of medicines calculated to assist the absorption of the effusion, and by continuing these medicines, with comparatively small reference to the general symptoms, until the discs have cleared. The writer has seen such clearing occur, without loss of sight, in circumstances which rendered it almost certain that neglect of the disc-effusions would have been followed by blindness at no distant date. The same general rule will apply, of course, to the more directly inflammatory effusions of neuritis or perineuritis; and, when we find any one of the three conditions passing into atrophy, or when we find commencing atrophy in discs which show traces of past effusion, the principle of treatment is to endeavour to promote the absorption of any contracting material which may be the physical cause of the atrophy; and then, when this has been done, to seek to stimulate the nutrition of the nerve-fibres, and to assist them to recover from the shock which they have sustained. The mode of fulfilment of the first indication must depend mainly upon whether there is a history of syphilis, and, if so, upon the manner in which it has been treated. In the numerous cases in which a short course of mercury has been administered, enough to alleviate secondary symptoms, but wholly insufficient to eradicate the disease, it will generally be desirable to give iodide of potassium in full doses for a time, and to follow this by the prolonged administration of the perchloride of mercury, in the hope of really curing the patient. There are, in the writer's opinion, few things better worth remembering in therapeutics than that the iodide, immeasurably the most valuable drug which we possess as a remedy for a late syphilitic symptom, is, none the less, almost useless as a remedy for constitutional syphilis. It will remove the present symptom, speedily and often completely; but it can scarcely be said to have any tendency to prevent the recurrence of syphilitic sym-

ptoms at a future time, in the same or in some different form. For this purpose, the only trustworthy agent is mercury; and therefore, while the administration of the iodide for a sufficient time, and in sufficient quantities to test its power of doing good, will be enough in the cases in which syphilis is neither known nor suspected, the iodide must be followed by mercury whenever a history of syphilitic infection is either clear or highly probable. The second indication, to stimulate the nutrition of the nerve-fibres, is usually best accomplished by strychnine, given at such intervals and in such doses as to produce evidence of its constitutional effect before its administration is abandoned. It may perhaps be most effectually given by hypodermic injection; but this is a point which must be settled in accordance with the circumstances of the case in each individual instance. In cases of great disc-swelling with impairment or loss of sight, the writer has on four occasions incised the sheath of the optic nerve behind the eye, so as to give exit to any accumulation of fluid in the intervaginal space, or to release an inflamed nerve from the pressure of its unyielding covering. The results have been generally satisfactory; and in one of the patients, who had been totally blind for several days, vision was completely restored.

3. *Toxic Amblyopia. Toxic Central Neuritis.*—Much attention has been bestowed of late years upon a form of gradual failure of vision which first affects chiefly the central portions of the retina, and which appears to depend upon a chronic neuritis attacking originally the portion of the optic nerve-trunk which contains the fibres destined for the region of the macula lutea. This affection is often connected with the excessive use of the stronger forms of tobacco, as well as with the abuse of ardent spirits, or with both these conditions in combination. It is characterised by impairment of sight, and also of colour-sense, the impairment being at first limited to the central portion of the field, and the colour-sense often becoming changed before form-vision is very noticeably affected. It may extend, if neglected, over the whole of the nerve-fibres, and may ultimately lead to practical, but seldom to absolute, blindness, perception of light and of large objects being often retained. In order to test for central loss of colour-sense, it is necessary to be provided with small red and green objects which may be moved in succession over various parts of the field, and which, in typical cases, will appear much less bright in the centre than when they are held a little to one side of the direct line of vision.

TREATMENT.—This consists, firstly, in the absolute abandonment of tobacco and alcohol; secondly, in endeavours to improve the general health; thirdly, in the use of medicines, such as iodide of potassium and mercury, by which chronic inflammatory conditions are likely to be controlled; and, lastly, in stimulation of the weakened nerves by the careful employment of strychnine. A very careful prognosis should be given in the first instance; but, if improvement once commences, the case will usually terminate in recovery.

4. *Sclerosis of the Optic Nerve.*—Besides the consecutive forms of atrophy above enumerated, there is yet another of common occurrence, which is either a primary sclerosis of the optic nerve, or a sclerosis secondary to a similar affection of other parts of the nervous centres. This form of atrophy is not preceded by effusion, nor is it attended by any

marked decrease in the calibre of the central vessels of the retina, even when the capillary circulation of the disc has almost wholly disappeared. It is often seen in connection with disease of the spinal cord, as in locomotor ataxy; and also occurs in apparently healthy people, seemingly as a purely local affection.

Sclerosis is easily distinguished from the atrophies consecutive to effusion, whether active or passive, by the circumstance that the effusion, as it undergoes contraction, tends to render the nerve-tissues opaque as well as to bleach them, and thus leaves a disc-surface of an almost ivory whiteness and of uniform colour. In sclerosis, on the other hand, the nerve-tissue disappears to a great extent, and reveals the mottled surface, often of a bluish-white tint, of the lamina cribrosa. When this is plainly seen, and when, at the same time, the vessels are neither much diminished in calibre nor altered in their normal curvatures, sclerosis may be assumed to exist; and this form of atrophy may also be distinguished from that which is produced by the most chronic forms of glaucoma, by the circumstance that in the latter the vessels bend into the excavated disc at its margin, while in the former they pass over the margin in straight lines or nearly so. Chronic glaucoma would also usually be distinguished by the character of the failure of sight, which would be marked by the ordinary contraction of the field of vision, even when central vision was only a little impaired; and also by the gradual hardening of the eyeball, which would be present in glaucoma and absent in nerve-sclerosis. Still it cannot be denied that this particular diagnosis is not without its difficulties, and that in certain cases it has given rise to differences of opinion between men of large experience on all sides of the question at issue.

The diagnosis is important as well as difficult, since the mischief of glaucoma may admit of arrest by iridectomy or sclerotomy; so that to mistake chronic glaucoma for atrophy, and to neglect operation, may be to condemn the patient to unnecessary blindness. The opposite error can at least do no harm; and therefore, whenever a doubt really exists upon the point, the most proper course is to give the benefit of that doubt to the patient, and to advise the performance of an operation which cannot injure, and which may relieve him. The atrophy of sclerosis scarcely admits of treatment, but it may perhaps sometimes be delayed, or even prevented from becoming complete, by the administration of full doses of strychnine and iron.

5. *Atrophy from other causes.*—Besides the foregoing forms of atrophy, there is a variety which appears to be associated with chronic lead-poisoning, and in which the discs may acquire a peculiar grey or bluish tint; and the optic nerves may also undergo secondary wasting in consequence of other conditions presently to be mentioned, such as obstruction of the central artery by an embolus, or the long continuance of pigmentary retinitis.

6. *Retinal Hemorrhage.*—The chief disorders of the retinal circulation displayed by the ophthalmoscope are hæmorrhages, which may be attended by very different circumstances, and may present widely different characters.

a. *Single.*—When blood proceeds from one of the larger veins of the retina, which yield a considerable quantity, and which are situated immediately underneath the limiting membrane, the hæmorrhage usually spreads out over the fundus as a red patch of uniform colour and aspect, and vision is suddenly, and

sometimes almost totally, obscured. The writer has seen such bleeding occur from the yielding of a vessel during parturition; but this accident is extremely rare, and the large hæmorrhages in question are certainly more common in women at the period of cessation of the menstrual function than in any other circumstances. At this time, and when the health is not seriously affected, a favourable prognosis may be given with some confidence; for the blood will before long be absorbed, and restoration of vision, at least in a considerable degree, may be expected. The writer has once seen complete restoration to the normal standard, but this is an exceptional occurrence.

TREATMENT.—The only treatment necessary is to pay attention to the requirements of the general health, and to prescribe such diet, medicines, regimen, and habits as may tend to calm and equalise the circulation, and to prevent local congestions. The occurrence of sudden loss of sight in one eye will justify the suspicion of hæmorrhage; but the suspicion can only be converted into certainty by the ophthalmoscope.

b. Multiple.—A form of venous hæmorrhage which at first seems less formidable, because it is attended by a smaller degree of immediate interference with sight, but which calls for a less favourable prognosis, is that in which the hæmorrhages are multiple, often singly of small size, and scattered over the whole fundus of the eye. The appearances which they present differ, apparently in accordance with their precise position in the retina. If they proceed from vessels which are superficial, the blood is spread out, as in the last variety, in round or oval patches beneath the limiting membrane; but if the vessels lie a little deeper, and are fairly engaged in the fibre-layer, the blood will separate the fibres and find its way between them, forming flame-shaped or brush-like patches, which are often very numerous. Such multiple hæmorrhages are very slowly absorbed, and have a tendency to recur; so that they must always be regarded as placing the sight in serious jeopardy. They are often monocular, and they do not point to any definite disturbance of the general health. The only endeavour so to connect them with which the writer is acquainted was made by Hutchinson, who described some cases of flame-shaped hæmorrhage in persons all of whom he said were 'gouty'; but it will certainly be the experience of most practitioners that flame-shaped hæmorrhages occur in many patients who are not 'gouty' in the ordinary sense, and that they do not occur in vast numbers of those about the reality of whose gout there can be no question. The presence of multiple hæmorrhages is sometimes attended by a considerable degree of irritation, or even inflammation, in the tissues among which the blood has been effused; and this condition, in which the retina between the blood-spots may become opalescent or turbid, has been described as a form of retinitis—*retinitis apoplectica*. The element of inflammation, in such instances, is probably merely a reaction consequent upon the injury inflicted upon the tissues, and it cannot be inferred that the bleeding is itself the result of any inflammatory process.

TREATMENT.—In this, as in the former variety, there is no special indication for treatment, which must be confined to the correction of any manifest disorder of the general health, followed, in most instances, by the administration of iodides or bromides, as medicines calculated to assist in the

absorption and removal of the effused products. Any indication of a general hæmorrhagic tendency, or of a state allied to purpura or scurvy, would require, of course, full consideration and appropriate treatment. The extent of the ultimate injury to sight will usually depend upon the extent to which the perceptive elements of the retina have been compressed or disorganised, either by the bleeding itself, or by other changes consecutive to it.

c. Arterial.—Hæmorrhages which are distinctly arterial are not uncommon in the fundus of the eye, and can generally be distinguished without difficulty from the venous variety, not only by the colour of the effused blood, but also by the situation of the blood-patch, and by its manifest relations to a small arterial branch, which may often be seen to have dwindled or closed beyond the point at which it has given way. Arterial hæmorrhages are mostly multiple, but of small individual extent; and, when not in the immediate neighbourhood of the optic disc, are most commonly seen near the outer limits of the ophthalmoscopic field of view. They are generally attended by sufficient impairment of vision to occasion complaint, and thus to lead to their detection; but they seldom occasion blindness. They call for an examination of the urine for albumen, and failing any evidence of renal mischief, they are chiefly important as indications of a weakened and brittle state of the arterioles, likely to lead to intracranial hæmorrhage.

TREATMENT.—Arterial hæmorrhages into the retina point to the necessity of diminishing the strain upon the arterial coats, by such means as the avoidance of muscular exertion or mental emotion, and by seeking to diminish the quantity of the circulating blood by a diminution in the quantity of fluid ingested. Even when all precautions have been taken, arterial retinal hæmorrhages are frequently followed by apoplexy.

7. Embolism of the Central Artery of the Retina. Embolism of the central artery of the retina, or of one of its branches, is a condition of not infrequent occurrence.

SYMPTOMS.—When sudden blindness of one eye occurs in a person who is the subject of valvular disease of the heart, the diagnosis can scarcely be doubtful; but the ophthalmoscopic appearances will suffice to remove doubt if it should exist. The immediate effect of the sudden arrest of the arterial circulation of the retina is to render that membrane opaque and of a milky whiteness, except over the macula lutea, where the absence of connective tissue prevents any such change from being produced. Here, and here only, the original transparency is retained, and the colour of the choroid is still visible, with the result that the macula appears as a cherry-red spot in the midst of a white surface. When not concealed by the opacity, the larger veins of the retina are diminished in calibre and contents, and their blood is sometimes broken up into detached portions separated by interspaces. The arteries are empty, and are either invisible or traceable as white lines of fibrous tissue in the general milkiness of the fundus. The disc is usually bleached, but it will sometimes happen that its condition may be temporarily obscured by arterial hæmorrhage, occurring from some twig given off just below the seat of the embolus, and entering the eye independently.

The driving home of the embolus will throw upon such a twig the whole force of the circulatory *vis a*

tergo, and may thus rupture it—an occurrence of which the writer has seen several examples. The blood so effused is usually absorbed in a very few days, revealing the white disc and the collapsed arteries, and removing any uncertainty which might have existed with regard to the diagnosis. The opalescence of the retina also disappears before long, and then only the secondary nerve-atrophy and the disappearance of the arteries remain to disclose the nature of the original affection. Embolism seems to be a perfectly hopeless condition, because there is no anastomosis between the retinal and other vessels of a sufficient extent to maintain a collateral circulation. The writer has met with one instance in which only a sector of the field was affected, and with one in which embolism of a very small branch produced loss of sight over all the peripheral parts of the field, leaving central vision almost intact; but such cases are among the curiosities of ophthalmology, and complete and permanent loss of sight of the affected eye is the result which must always be anticipated.

8. *Retinitis*.—Retinitis is commonly described as occurring in three chief forms—the *albuminuric*, the *syphilitic*, and the *pigmentary*; but the writer is inclined to believe that only the last of these three is a genuine retinitis, and that in the others the inflammation, if it should exist, is merely a secondary consequence of the irritation produced by the presence of adventitious deposits.

a. *Albuminuric Retinitis*.—In the so-called albuminuric retinitis, the sequence of events lends support to the opinion that the renal disease is not an original affection, but a result of morbid changes which are common to the whole of the small arteries of the body.

SYMPTOMS.—In many cases of albuminuria, the sight is not affected from first to last, and the retinæ remain healthy. In some, the retinal changes precede the appearance of albumen in the urine; and, in the majority, the renal and retinal changes are coincident. The retinal changes are of two kinds: namely, arterial hæmorrhages, occurring in the fibre-layer, so that the blood-patches assume a fibrillated aspect with brush-like terminations; and the formation of white patches, either of cholesterine-deposit or of fatty degeneration, or of both combined, scattered irregularly over the fundus, but often grouped into a stellate figure around the macula lutea, and into an irregular ring around the disc. To these appearances are added, in some cases, those of swelling of the disc-margins with effusion into the retinal fibre-layer; and, when the last-named appearances are presented, there is always a far greater deterioration of sight than when they are absent. It is a matter of daily occurrence that the existence of renal disease is not suspected until impairment of sight leads to an ophthalmoscopic examination, and this to the discovery of the retinal changes.

TREATMENT.—The treatment of the renal maladies which produce albuminuria is in no way modified on account of the presence of a retinal complication; and the unfavourable prognosis which must generally be given as regards life throws into comparative insignificance the gradual failure of vision, which seldom proceeds to complete blindness.

b. *Syphilitic Retinitis*.—This is usually an incident of the most advanced stages of the disease, and is most frequently seen in persons who have been inadequately treated during the primary stage,

but who have for some months or even for a year or two been free from symptoms.

SYMPTOMS.—Dimness of sight is then complained of, and the retina is found to present scattered patches of very irregular outline, and of a filmy whitish aspect. Such patches may be more or less obscured by slight general turbidity of the retina itself, or of the vitreous body in its immediate vicinity, the latter condition being of itself highly suggestive of the nature of the malady.

TREATMENT.—The treatment must be greatly governed by the past history of the case, but may in most instances turn upon the use of iodide of potassium for the relief of the retinal troubles, followed by a sufficient course of mercury for the eradication of the syphilitic taint.

c. *Pigmentary Retinitis*.—This appears to be a true inflammation of the retina, or of the immediately subjacent choroid, differing from the foregoing affections in that it attacks the sensory elements, instead of the fibre-layer or the connective tissue of the membrane.

ÆTIOLOGY.—The subjects of pigmentary retinitis are of all ages, from nine or ten years to seventy; and, in some instances, the duration of the disease has been as much as twenty years, from the first appearance of the symptoms to their ultimate termination in blindness. As a rule, however, the patients are young adults, or persons not past middle age.

It is a remarkable feature of pigmentary retinitis that it almost invariably attacks more than one member of a family; and it has been said to be especially frequent in the offspring of marriages of consanguinity, but this statement is not borne out by English experience.

ANATOMICAL CHARACTERS.—From the extreme chronicity of its course, from its obstinacy, and from its peculiar anatomical distribution, pigmentary retinitis should probably be regarded, together with some forms of choroiditis, as having its analogies among some of the chronic diseases of the skin. It usually appears to commence in a narrow annulus near the equator of the eyeball, and gradually spreads inwards towards the optic disc; the tissues affected are the perceptive and pigmentary layers of the retina and the subjacent chorio-capillaris, which slowly become disorganised and matted together in one common degeneration. Coincidentally with the progress of the disease, pigment is deposited in the parts affected, and in the retina superficial to them, in the form of irregular lines and striations, and especially along the course of the main arterial branches. As the annulus of disease gradually closes in upon the macula, the optic disc undergoes atrophy, and the central vessels, both veins and arteries, dwindle in size.

SYMPTOMS.—The subjective symptoms are as characteristic as the ophthalmoscopic appearances. Over the region actually invaded, the sensory elements of the retina are destroyed and the power to receive visual impressions is lost. The fibre-layer not being implicated, the conduction of impressions from parts of the retina more peripheral than the disease may remain unaffected; and hence we may have a blind zone surrounding the centre of the field of vision, and surrounded itself by a zone still more external, in which dim vision is preserved. But the salient symptoms are two—the gradual contraction of the field of vision due to the progressive encroachments of the disease; and night-blindness,

due to the nerve-atrophy, which interferes with the conduction or perception of any but strong impressions. When these symptoms co-exist; when the field of vision is small and becoming gradually smaller; and when the patient, who can still see fairly in the daytime, can scarcely find his way about as dusk begins to fall, we may predict the ophthalmoscopic appearances with a very near approach to certainty. The optic disc will be unnaturally pale, and the fundus overstrewn, towards the periphery, with irregular black lines and stripes, of which it is possible that none may be visible within that portion of the field of the ophthalmoscope which includes the disc.

DIAGNOSIS.—Pigmentary retinitis may be mistaken for the most chronic form of glaucoma, on account of the contraction of the field of vision; but may be distinguished by the absence of high tension, by the night-blindness, and by the pigmentation of the retina. It may be mistaken for the atrophy of sclerosis, but only if the ophthalmoscopic examination be limited to the nerve-disc, to the neglect of the surrounding parts of the fundus.

TREATMENT.—In the treatment of a disease so essentially chronic, it is difficult to arrive at trustworthy evidence of the efficacy of a remedy, but the prolonged administration of iron is at least of a certain degree of utility in arresting the progress of the malady. The preparation employed is probably not material.

9. *Detachment of the Retina.*—**SYNON.**: Sub-retinal Dropsy.—This is a condition the causes of which have never been satisfactorily explained, but it has been attributed to the contraction of inflammatory effusions in the vitreous body, which have been supposed thus to exert traction on the retina.

The first symptom which attracts the attention of the patient is the loss of part, usually either the upper or the lower part, of the field of vision; and it is manifest that loss of the upper part of the field means detachment of the lower part of the retina, and *vice versa*. Detachment is sometimes produced by a blow or injury, but more frequently it occurs without any assignable cause, either local or constitutional. One or both eyes may be affected.

The diagnosis is rendered easy by the ophthalmoscope, which exhibits the detached portion as a sort of floating prominence, projecting into the interior of the eyeball, generally bluish-white in colour, and crossed by the retinal blood-vessels.

The prognosis is unfavourable in the majority of instances, and treatment is seldom effectual.

TREATMENT.—Cases have been recorded in which disappearance of the sub-retinal fluid, and restoration of vision, have followed prolonged confinement in the supine posture; and the occurrence of improvement after spontaneous rupture of the detached portion suggested to von Graefe the advisability of producing such a rupture by artificial means. Various operations have been undertaken for this purpose, and also for the evacuation of the sub-retinal fluid through a puncture in the outer tunics of the eye, and have in a few instances been partially successful; but the evidence in their favour hardly establishes more than that attempts of such a nature may be made. The writer once obtained an excellent result by puncturing a detached retina in an eye from which cataract had been removed eleven years before; but the puncture was greatly facilitated, and its risks were in a corresponding degree diminished, by the absence of the lens. Two

or three years later, however, the detachment returned and became complete. Detachment may be simulated, or may even be caused, by the growth of intra-ocular tumours, sarcomatous or gliomatous, which may demand the early removal of the eyeball. Such cases would usually be distinguished from simple detachment by increased hardness of the eyeball, which the morbid growth almost necessarily occasions, and which would be the more significant inasmuch as detachment alone is usually accompanied by diminished tension. It must nevertheless be remembered that normal tension does not absolutely preclude the presence of tumour; as there are well-authenticated cases in which the absorption of the ocular fluids has, for a time, precisely kept pace with the increase of the growth.

10. *Glioma.*—This name was given by Virchow to a malignant growth which has its origin in the neuroglia, or connective tissue of the nervous system, and which was formerly described as encephaloid cancer. When originating in the retina, it early produces loss of sight, and presently shows through the pupil as a substance of a primrose-yellow colour, by which the still transparent lens is pressed forward towards the cornea. It is chiefly a disease of childhood, and has been seen by the writer as early as the fifth week of infant life. It is liable to be mistaken by superficial observers for congenital or infantile cataract, an error which must be carefully guarded against, because the early and entire removal of the eye, together with as much of the optic nerve as can be reached, furnishes the only hope of preserving the life of the patient. When the operation is performed sufficiently early, it has in a few instances been completely successful, cases having been recorded in which no recurrence of the growth has happened after the lapse of years. In the majority, however, recurrence and death have terminated the history.

11. *Sarcoma.*—This differs from glioma in having its origin in the choroid, and in being of a darker colour, and sometimes pigmented or melanotic. It may only be discoverable with the ophthalmoscope. It is as malignant as glioma, and requires the same treatment.

VIII. Diseases of the Choroid.—Diseases of the choroid, recognisable by the ophthalmoscope, are almost limited to certain chronic forms of inflammation and of atrophy; for, in any acute choroiditis, there is always too much turbidity of the vitreous body to allow the state of the membrane to be seen.

Chronic Choroiditis.—The chronic forms of choroiditis are remarkable for leading to an undue formation, or to a great displacement, of the choroidal pigment; and to the ultimate complete wasting and disappearance of the portions of the choroid which are affected, so that over these portions there will ultimately be no choroid visible, and the ordinary red colour of the fundus will be replaced by the ivory whiteness of the inner surface of the sclerotic.

Chronic choroiditis may be divided into two chief varieties—the *disseminated* and the *diffused*. The *disseminated* occurs chiefly in children, and chiefly, perhaps exclusively, in those who are the subjects of inherited syphilis. It is seldom seen until its period of activity is passed. A child is brought on account of defective vision, which has probably existed from birth or from a time but little subsequent to it; and the ophthalmoscope displays a number of small white spots, with black borders,

scattered irregularly over the fundus of the eye. The white spots are patches of choroidal atrophy, and the black borders are rings of increased pigment-formation, by which the spots of inflammation, which must have been comparable to little pimples, have been surrounded.

TREATMENT.—Such cases admit of no treatment, except in the rare instances in which some active mischief may be detected, in the shape of small patches or spots in which effusion has not yet passed into atrophy, and in which such an antisymphilitic treatment should be employed as the state of the patient may otherwise permit or indicate.

Diffused choroiditis is more frequently an affection of adult age; and, although very frequently syphilitic, is not invariably so. It differs from the foregoing chiefly in the absence of any defined shape or precise limitation of the parts affected. In the early stages the choroid is seen to be troubled by congestion or effusion, and these conditions pass gradually into abnormal pigmentation and atrophy. The course of the disease may be very chronic and irregular, and different stages of it may be seen at the same time in different parts of the same eye.

The prognosis may in general be moderately favourable; for, although the choroiditis destroys the portion of retina immediately in front of it, its extension is capricious, and it may often be arrested in time to leave portions of the eye, and especially the central portions, unhurt. When it occurs in the vicinity of the macula lutea, so as to imperil central vision, it is much more formidable than when confined to the more peripheral parts of the membrane.

TREATMENT.—Whenever there is a history of syphilis, this must be taken as the clue to treatment; and, if no syphilis can be discovered, the chief reliance must be placed upon rest of the eyes, occasional depletion from the temples by Heurteloup's leech, counter-irritation by blisters or setons, and such internal medication as the general state of the patient may suggest.

IX. Diseases of the Vitreous Body.—Diseases of the vitreous body are as yet very imperfectly understood, and we know little more concerning them than that this substance is liable to become turbid in certain forms of acute general inflammation of the eye; and that it is sometimes rendered turbid, without inflammation, by the presence of floating films which may be readily seen by the ophthalmoscope, and which may be so numerous as to form a serious impediment to vision.

1. *Turbidity.*—Turbidity of the vitreous is very common in syphilitic cases; but the films referred to are seen when no syphilis can be suspected. Their number, and their free movements, show that the vitreous must in great measure have lost its natural semi-solid consistence, and have become fluid; but little or nothing is known of their actual pathology.

TREATMENT.—The most effectual treatment for flocculi in the vitreous is usually diaphoresis by the subcutaneous injection of from two to four minims of a 10 per cent. solution of nitrate of pilocarpine, which may be repeated on alternate days. Local counter-irritation with iodine may also be practised; and iodide of potassium be given internally in such doses as circumstances will allow.

2. *Muscae Volitantes.*—A phenomenon referred to the vitreous body is the appearance of the moving particles, or strings of beaded filaments, which are

commonly called *muscae volitantes*. True *muscae* are known by the negative character that the particles which produce them cannot be seen by the ophthalmoscope; and by the positive character that they never so intervene between the eye and an object, however small, as to exclude the latter from view. They are seen most readily against a white field, as a white wall or a white cloud, or in the illuminated field of a microscope when there is no object in view; and they float about with uncertain movements, but always a little out of the direct line of sight. They are occasioned by the filamentous framework of the vitreous body, and by the cell-nuclei or other irregularities upon the filaments. These bodies, without being opaque, yet differ in the precise degree of their transparency from the fluid which surrounds them; and hence they cast upon the retina shadows, which are then mentally projected outwards into space as floating objects. The projected shadows appear, of course, enormously larger than the microscopic specks which produce them, and the latter are wholly unimportant and of no morbid signification. *Muscae* may be discovered by any person by the simple expedient of looking through a very fine perforation in a metal disc at a well-lighted white surface—a white cloud, for example; and they are more conspicuous to some persons than to others, on account of the varying differences which may exist in different eyes, or in the same eyes at different times or under different conditions, between the index of refraction of the filaments and nuclei and that of the surrounding fluid. Moreover, by the operation of an obvious physical law, the more distant the particle from the retina, the larger will be its shadow upon that membrane, and the larger and more conspicuous will it appear. For this reason, and on account of the elongation of the myopic eyeball, *muscae* are usually more complained of by the short-sighted than by others. They are often sources of great uneasiness to patients; but, when once their true character is known, they may usually be disregarded as harmless appearances, the natural results of physiological structure. It is often important that the physician should be able to make their nature understood, in order that he may dissipate, once for all, the unfounded apprehensions which may be occasioned by their presence.

X. Diseases of the Eyelids.—The external surfaces of the eyelids, as parts of the common integument, are liable to all its diseases, and may thus participate in erysipelatous inflammation, in eruptions, and in the results of injury, besides becoming the seats of naevi, moles, warts, and other growths. Among the diseases special to the formation of the eyelids, the most important are the variations of shape to which they are subject, generally from the contraction of inflammatory exudations, but sometimes from perverted muscular action; the cystic tumours which are produced by obstruction of the orifices of Meibomian glands; the inflammation of the follicles of the eyelashes, or blepharitis; spasmodic closure, from abnormal muscular contraction; and either patency or passive closure, from paralysis. Many of these affections are distinctly surgical, and others are only parts or symptoms of more general disorders.

1. *Blepharitis.*—Blepharitis, or inflammation of the follicles of the eyelashes, has received a great variety of names from different writers, and is frequently known as *tinea tarsi*, or, in its more

advanced stage, as *lippitudo*. The disease consists essentially of an inflammation of the lining membrane of a hair-follicle from which an eyelash springs.

SYMPTOMS.—The first manifest symptoms are a small swelling close to the edge of the eyelid, generally of the upper lid; and the formation of a crust around the bases of the cilia which proceed from the swollen part. The swelling does not extend farther up the lid than to the breadth of about a line, but it soon spreads along the border until the whole length is involved, and it usually spreads also to the lower lid, manifestly in consequence of the contagious character of the discharge. If the crust is removed, and if the part from which it springs is magnified and carefully examined, it will be seen that the mouths of the follicles are somewhat open, no longer fitting closely to the issuing hairs, and, in a few moments, a clear fluid will be seen to exude, and speedily to dry into a crust or film, which covers the opening as if with a varnish. Many of the hairs in the affected follicles are loosened, and fall readily, or may be removed painlessly by slight traction. If the case be neglected, the follicles are destroyed as hair-bearing organs, so that the lost cilia are no longer reproduced; and the new tissue which constitutes the subcutaneous swelling of the lid-margin begins to undergo contraction, and in this way gradually everts the cartilage. The edges of the lids become red, swollen, and unsightly; the lachrymal puncta are so displaced outwards that they no longer take up the tears; the eyes have lost the protection of the lashes, and are exposed to irritation from atmospheric particles and other causes, so as to be especially prone to conjunctival and corneal inflammations; and these results are almost incurable. It is therefore very important that blepharitis should be effectually treated in its early stages, when, if only due care be taken in the selection and use of remedies, it is but a trivial affection.

TREATMENT.—The most essential part of the treatment is to remember that the secretion which forms the crust is of such a nature that it is not very easy of removal, and that, while it remains *in situ*, no remedies, however judiciously chosen, can obtain access to the parts really affected by the disease. The crust is composed partly of the already mentioned secretion from the inflamed surfaces, partly of the greasy secretion of the Meibomian glands; and it is the admixture of the latter which renders the crusts difficult of removal by water alone. A solution of bicarbonate of sodium of five grains to the ounce of warm water will remove them readily; and this solution should be applied in such a manner as to soak into the crusts and loosen them thoroughly before any attempt is made to detach them. As soon as they are detached, the surface beneath should be gently dried, and an astringent should be applied immediately, so that it may find its way down into the depths of the hair-follicles, and may reach the seat of the malady. The best astringent is generally the ointment of the precipitated yellow oxide of mercury, already recommended for the cure of ulcers of the cornea; and this may be applied to the affected part by the tip of a finger. If amendment does not speedily follow, it may be suspected that the crusts have been imperfectly removed, or the applications imperfectly made, and it will be well for the practitioner personally to superintend the process. When this has been done, if the affection continues obsti-

nate, some other astringent should be tried, and the nitrate of silver is among the best for this purpose. Amendment of the lid-margin may generally be quickly produced; but the disease will for a long time lurk in the depths of the follicles, and the treatment must be continued until all subcutaneous swelling has disappeared from the lid-margins. Unless this be done, speedy relapse is inevitable, the inflammation soon creeping out of the follicles again and recovering the ground of which it had been deprived. Such a result is constantly seen in hospital out-patients, in spite of all efforts to guard against it, and, in prescribing for blepharitis, it is always desirable to warn patients of the perseverance which will be required, and of the great importance of obtaining a radical cure. There can be no doubt that blepharitis is exceedingly contagious through the medium of its secretion, conveyed upon sponges, towels, or fingers, and this should be fully recognised whenever it attacks children who are attending a school. The name '*tinea tarsi*' may perhaps be taken as the expression of a belief that the disease is allied to *tinea tonsurans*, and that it is produced by the growth of a parasitic fungus. The writer is not aware of any sufficient ground for the adoption of this opinion.

2. *Entropium and Ectropium.*—Incurvation and excuvation of the eyelids may be looked upon as purely surgical maladies. The former exposes the eyes to injury from the irritation of inturned eyelashes (*trichiasis*); the latter from foreign bodies of various kinds.

TREATMENT.—The remedy for both, when any is practicable, must be usually sought in a surgical operation. An exception depends upon the fact that ectropium is sometimes produced by paralysis of the facial nerve, which renders the orbicularis muscle flaccid and powerless, and permits the lower lid to fall downwards under the influence of gravity. The cure of the general nerve-affection may restore the power of the muscle, and may in time lead to complete recovery of the natural position of the lid. In such cases, even if electricity does not form part of the general treatment of the paralysis, it may often be applied with benefit to the orbicularis.

3. *Blepharospasmus.*—This term is employed to denote an intermittent closure of the eyelids by involuntary action of the orbicularis in response to some concealed source of irritation; and is thus distinguished from the spasm which accompanies photophobia.

SYMPTOMS.—The spasm is most liable to occur in circumstances of mental excitement. The motor nerves appear, as a rule, to be merely the passive conductors of a reflected impulse, and the trouble seems usually to be dependent upon a morbid condition of the fifth nerve, or upon a source of irritation in some peripheral part from which a twig of the fifth nerve passes to the centre.

TREATMENT.—In the treatment of such cases, it is sometimes possible to find the twig which conveys the impression; that is to say, to discover a point where pressure, sufficiently firm to arrest conduction, will at once relax the spasm. Such points should be looked for at the supra-orbital notch, over the malar bone, and in any other situation suggested by special circumstances; and, if a point at which pressure will arrest the spasm is discovered, we learn at once by what branch of the fifth nerve, and therefore approximately from what region, the irritation is conveyed, and where its source is to be sought

for. If nothing can be discovered by careful examination, decayed teeth, accumulations of cerumen in the ears, and conjunctival granulations are possible conditions which should be looked for, and which should receive attention if they are found. When all other treatment has failed, the spasm has sometimes been stopped by subcutaneous section of a sensory nerve; and this may be practised hopefully if the spasm can be arrested by pressure on some definite spot, which must then serve for the guidance of the knife. If no such spot can be found, section of the supra-orbital nerve, and next of the subcutaneous malar, may be attempted; since neither of these is sufficiently important for its temporary dismemberment to be set against even the possibility of relief from a very distressing affection. In some cases, however, it would appear that the mischief must be central, and that no section of an afferent nerve can be useful. The division of the motor nerves of the orbicular muscles, if it could be successfully accomplished, would produce a paralysis even more injurious than the spasm; and the cases in which the latter is due to central irritation or other trouble, unless they can be relieved by medicine, and by the rectification of whatever may be manifestly wrong in the condition of the patient, offer very small hope of improvement. See FACIAL SPASM.

4. *Ptosis*.—Ptosis is a condition of permanent passive closure of an upper eyelid as a consequence of paralysis of its levator muscle, or it may happen in consequence of this muscle having been torn from its attachment to the tarsal cartilage, so that it can no longer modify the position of the lid.

SYMPTOMS.—Paralytic ptosis may be either partial or complete, according to the degree of the nerve-affection; and as the levator palpebræ is supplied by the third nerve, which supplies also the superior, the internal, and the inferior rectus, as well as the inferior oblique, the sphincter pupillæ, and the ciliary muscle, ptosis is usually accompanied by paralysis of one or more of these muscles. When they are all affected, the eyeball is turned outwards by the action of the external rectus, and is immovable in other directions excepting feebly by the superior oblique. The pupil is dilated, and the power of adjusting the eye for near vision is impaired or lost, although, when the lid is raised, near objects can still be distinctly seen by the aid of a convex lens. When all the muscles supplied by the third nerve are affected, the inference is that the cause of paralysis is acting upon the common

trunk of the nerve; and such a cause is not infrequently the presence of periosteal swelling at the sphenoidal fissure. If only some of the muscles are affected, the inference is that the cause of the paralysis is either limited to the central nuclei of origin of certain filaments, or else that it is situated anteriorly to the division of the main trunk into the branches which proceed to different parts; and it is conceivable that the limitations of the paralysis may point, with tolerable certainty, to the precise locality of the disorder.

The causes of ptosis, as of other paralytic affections of single cranial nerves, apart from injuries and the pressure of morbid growths, may almost be reduced to syphilis and to impaired nutrition of the centres, the latter usually connected with hard mental work and worry. In every case, evidence of syphilis should be carefully sought for; and, if found, should determine the nature of the treatment, as also of the prognosis, which, in such instances, may be generally favourable. In cases of the second class, where there is no evidence of syphilis, and where the symptoms point to general impairment of nervous energy, the administration of iodide of potassium, in combination with tonics, will sometimes be useful; but the main reliance must be placed upon rest, good living, and external surroundings favourable to the restoration of health.

5. *Diplopia*.—Double vision, although it has no proper relation to the subjects treated of in the present section, is yet so far allied to ptosis that, when occurring suddenly, it is almost always an effect of paralysis or of paresis either of the sixth nerve of one eye, supplying its external rectus, or of the branch of the third nerve which supplies its internal rectus. In the former case the affected eye will deviate inwards, and will have limited range of movement towards the outer canthus; while in the latter case these conditions will be reversed.

As regards the causes and treatment of these limited forms of paralysis, there is nothing to add to what has already been stated about ptosis. It is sometimes desirable, while the diplopia continues, to exclude the deviating eye from vision by a shade, an opaque spectacle-glass, or other suitable contrivance, on account of the vertigo and uncertainty of gait which may be occasioned by the double images.

See also EXOPHTHALMIC GOITRE; LACHRYMAL APPARATUS, Diseases of; LAGOPHTHALMOS; ORBIT, Diseases of; STRABISMUS; STYE; and VISION, Disorders of.

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F

FACIAL ATROPHY.—SYNON.: Facial hemiatrophy; *Hemiatrophia facialis progressiva*.

DEFINITION.—A wasting which is usually limited to one side of the face and which involves all the structures, although the change in the volume of the muscles is not due to a true atrophy.

ETIOLOGY.—Women are more commonly affected than men, and the atrophy usually begins before puberty. In most cases no cause can be assigned; in a few there has been hereditary transmission, and in others the condition has followed

one of the specific fevers, abscess in the region of the ear, erysipelas or tonsillitis, but trauma to the face is the most common exciting cause.

SYMPTOMS.—A white or yellowish-white patch appears on the cheek, chin, or forehead, and gradually spreads, the skin becoming parchment-like and often 'glossy'; all secretions are usually diminished, while hairs, if present, change colour and often fall off. The subcutaneous tissues waste, so that a local depression is formed, which is most obvious when the cheek is the part affected, and

atrophy of the orbital fat results in recession of the eyeball. As the disease progresses the bones and cartilages also atrophy, especially in the cases that begin before puberty. The teeth may be smaller, decayed, and loose on the affected side. There is no true atrophy of the muscles, although they become small in consequence of disappearance of their interstitial fat, a state of things that accounts for hemiatrophy of the tongue which occurs in some of the cases.

The muscles act voluntarily, and in response to electrical stimulation. Neuralgic pains may occur, but cutaneous sensibility is not blunted, and taste is not affected even when the tongue is involved.

The atrophy varies in distribution and in rate of progress. It may remain limited to the part in which it commences, or it may spread so as to involve the whole of one side of the face, when the two halves look as if they belong to two different individuals. More rarely the atrophy becomes bilateral, and in a few cases atrophic areas of skin have been present on the arm and back on the same side as the facial hemiatrophy.

MORBID ANATOMY AND PATHOLOGY.—Interstitial neuritis of the fifth nerve has been found in one case, a discovery that has led to the belief that the facial atrophy is induced by this change in the nerve. There are, however, objections to this view, and grounds for regarding the affection as the result of arrest of development.

DIAGNOSIS.—The condition is easily recognised, but has to be distinguished from asymmetry of the face of congenital origin, that due to infantile hemiplegia, the atrophy of the facial muscles due to a lesion of the seventh nucleus or nerve, sympathetic paralysis with consequent recession of the eyeball, and facial hemi-hypertrophy which causes the unaffected side of the face to appear small.

PROGNOSIS.—The atrophy is usually progressive, and produces permanent disfigurement.

TREATMENT.—Massage, facial gymnastics, and galvanism are indicated, as are tonics such as arsenic, iron, and strychnine. The deformity of the cheek may be lessened by a 'plumper' worn inside the mouth.

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disease, as caries; and sometimes to syphilis. Hæmorrhage into the sheath may also compress the nerve. Cold is the most common cause of palsy, but the exposure is often forgotten; it has been supposed to act by paralysing the peripheral nerve-twigs, but this is never the case in one-sided paralysis; in all cases lasting more than a few days, evidence of changed nutrition may be detected in the nerve-trunk as it emerges from the stylo-mastoid foramen by electrical stimulation. External cellulitis may extend into the canal. Tumours of the bone and fracture of the base may also damage the nerve as it passes through the bone. (2) Injury to the nerve outside the skull by blows, pressure by forceps in instrumental delivery, incised wounds (accidental or surgical), or parotid and other tumours, are occasional causes. (3) Within the skull the nerve may be damaged by meningitis, acute or chronic, and especially by syphilitic inflammation, or by pressure of neighbouring growths. The radicular fibres within the pons, or the nucleus beneath the fourth ventricle, may be damaged by hæmorrhage, softening, or by tumours involving that part.

Double facial paralysis is very rare, and is due to damage to both nerves at the base of the brain from meningitis or symmetrical syphilitic disease; to symmetrical otitis; or to an affection of the nuclei by disease of the pons, or to loss of function of the peripheral nerve-fibres in various forms of peripheral neuritis. It is probably by the latter agency that double facial paralysis sometimes results from toxic blood-states. These, with syphilis and diphtheria, are the most common antecedents of double facial palsy not due to demonstrable injury to the nerves. Disease of the nuclei affects both sides most frequently when it is degenerative in nature; it is then commonly partial, involving especially the lip-muscles in labio-glossal paralysis, or the orbicularis palpebrarum in rare cases of nuclear ophthalmoplegia. Lastly, it may be mentioned that the facial muscles (generally those in and about the lips) are occasionally involved in one form of juvenile idiopathic muscular atrophy, and an affection of the nerves may be thus simulated.

SYMPTOMS.—The onset of facial paralysis is usually rapid, when due to its common causes, including neuritis within the canal. It occupies from a few hours to three or four days in its development. It is found, for instance, one morning, in drinking, that the fluids run out of the side of the mouth; the face is noticed to be a little unsymmetrical; at night the eye cannot be completely closed, and next morning the paralysis is found to be complete. Rarely the onset is in a few minutes; probably the hæmorrhage into the nerve-sheath is the cause. When inflammation is excited by cold, the symptoms often commence within twenty-four hours of the exposure, and are rarely delayed for more than two or three days. They are usually preceded, accompanied, or sometimes followed, by some pain about the ear or side of the head.

In complete unilateral facial paralysis all the muscles on one side of the face are paralysed. At rest, the smooth forehead and lowered angle of the mouth are the chief indications, but on movement the difference between the two sides becomes very marked: the one half of the forehead moves alone in frowning or elevation of the eyebrow. The eyelids cannot be brought together, and in the attempt to close the eye, the globe is rolled upwards,

FACIAL PARALYSIS.—**SYNON.** : Paralysis of the Portio dura; Bell's Paralysis.

DEFINITION.—Paralysis of the muscles of the face, due to disease or injury of the nucleus or fibres of the seventh nerve.

Above the nucleus, the motor path for the face decussates, and joins that from the arm and leg; damage, therefore, in the pons (above the crossing), in the crus, internal capsule, or hemispheres, may produce facial paralysis, but does so usually as a part of hemiplegia. This paralysis, which is on the same side as that in the limbs, is partial only, affecting chiefly the muscles of especially unilateral use (as the zygomatici); and very little those of solely bilateral use, in the upper part of the face—e.g. the frontales. In this article paralysis from damage to the fibres or nucleus of the nerve itself will alone be considered.

ÆTIOLOGY.—(1) The most common causes of facial palsy are unilateral, and damage the nerve as it passes through the narrow canal in the temporal bone. There, the slightest inflammatory effusion will cause pressure on the nerve-fibres. Such neuritis may be due to exposure to cold (and is then often called 'rheumatic'); to contiguous bone-

so that only the sclerotic appears between the gaping lids; the patient then often fancies that the eye is shut. During sleep the eye remains open. In smiling, the lips may be displaced towards the healthy side, from the unopposed action of the opposite zygomatic muscles; the nostril of the affected side cannot be dilated; the upper lip cannot be raised; the cheek flaps loosely, from the relaxation of the buccinator; and, from the same cause, food accumulates between the jaws and the cheek. Whistling is impossible, from the paralysis of half of the orbicularis, and the lips cannot be approximated sufficiently even to permit a candle to be blown out. When the lesion is between the junction with the large petrosal and that with the chorda tympani nerves, or extends to this part, taste is partly or entirely lost in the front of the tongue. The loss of power of recognising acid and saline substances is most marked; but bitters and sweets are also not tasted in this part. Such loss is common in neuritis from cold, and shows that the inflammation has passed some distance up the canal. In rare instances, loss of taste has followed division of the nerve outside the skull, probably always because secondary inflammation has passed up the nerve. The palate is never paralysed from disease of the facial nerve. It is certain that the opposite statement rests on defective observation or interpretation. Oblivion of the uvula is common under normal conditions. Defect of movement, to be significant, must follow the course of any associated palsy of external muscles. The nerve-supply of the chief palatine muscle has been proved to be from the spinal accessory.

In some cases giddiness attends the onset of facial paralysis. In slight forms there may not be complete loss of power; but the defect is at first pretty equally distributed over all parts of the face.

In cases which recover, some return of power takes place in from a week to two months, and improvement is usually earliest in the upper part of the face; the power of frowning, winking, and closing the eye being soonest regained; that of moving the lips and mouth returning last. Even after several months of immobility, recovery may take place, but in these cases it is rarely complete, and a troublesome condition is apt to supervene: some of the muscles, especially the zygomatici, become shortened in late rigidity, and hence at rest the naso-labial furrow is deeper on the paralysed than on the healthy side, although the possible movement may be much slighter. This condition sometimes comes on rather rapidly; there is generally, in addition, an associated over-action of the upper and lower facial muscles, whereby, for instance, the orbicularis palpebrarum and the zygomatic and other muscles about the mouth act together in undue degree; in smiling, the eye shuts; and on closing the eye, the mouth is drawn upwards. Sometimes there are also spontaneous clonic contractions. This late rigidity and over-action may prove very troublesome and constitute a nuisance, especially to the young, not less than the original palsy, and more serious in that it is enduring. The writer has known it induce a good-looking girl to put an end to her life. If the interruption of the nerve is absolute and permanent, the muscles remain toneless, and no contracture supervenes.

The electrical condition of the muscles is very important. It is that always seen in paralysis from

nerve-lesion. The muscles, after a day or two of slightly increased irritability to both faradism and the slowly interrupted voltaic current, lose gradually their irritability to the former, retaining that to the latter, and even exhibiting to it increased irritability so that they act to a smaller number of cells than do those on the healthy side. The order of reaction to the two poles is often but not always changed, that to the anode being equal to the kathode or even occurring first. In the nerve, on the other hand, the irritability is lost to both forms of electricity, this loss proceeding *pari passu* with the degeneration which follows separation of the nerve from its nutrient centre. In slighter and more transient forms of facial paralysis, the change in irritability of muscle and nerve may be less; but even in most, which last only a few days, a slight change in irritability may be discovered. In the slightest cases (and in the earliest stage of more severe forms) there may be an increased irritability in the nerve, often most marked to the induced (faradic) 'shocks'—i.e. isolated momentary currents. Considerable change in irritability is proof of considerable disease of the nerve, and is thus of the highest prognostic importance, but it is not distinct until after a week; if absent at the end of a fortnight the disease is slight, and although the paralysis may still be complete, it will probably not last long.

DIAGNOSIS.—The diagnosis of facial paralysis (commonly so called—i.e. that due to disease of the nerve or its nucleus) is easy. It depends (1) on the implication of all the facial muscles, including those of bilateral use that escape in hemiplegia, because related to both cerebral hemispheres; (2) on the indication afforded by the electrical reactions that the nerve or its nucleus is diseased. The recognition of the place of the lesion is less easy. When within the pons, it is often associated with paralysis of the sixth nerve, or with hemiplegia of the opposite side from damage to the motor path to the limbs. At the base of the brain the auditory nerve is usually affected at the same time, but care must be taken not to mistake deafness due to ear-disease for an affection of the auditory nerve within the skull. When there are no other paralyses, the disease is probably within the bony canal. Deviation of the uvula is of no significance; affection of taste in the front of the tongue indicates disease within the canal. Special inquiry should be made for exposure to cold, ear-disease, syphilis, or a blow. Double facial paralysis, when due to symmetrical syphilitic disease, is usually associated with palsy of other nerves, since its cause is usually intracranial. When it is the result of peripheral neuritis there is usually also loss of power in the limbs.

PROGNOSIS.—The majority of cases of facial paralysis are due to disease of the nerve, and the prognosis depends on the evidence of the degree of damage to the fibres, especially on the indications afforded by an electrical examination, and also on the cause of the lesion. When this is progressive in nature, the interference with the nerve is necessarily persistent in duration. Thus, in facial paralysis due to a growth damaging the nerve, the prognosis must be grave. In syphilitic disease, on the other hand, it is good, provided that the duration of the damage to the nerve is short. This is also the case in paralysis from cold, in which, however, the evidence afforded by electricity is of special importance.

In double facial paralysis, the evidence of central

mischief renders the prognosis unfavourable, but recovery may be hoped for if the lesion is acute and the palsy incomplete. In syphilitic cases, the prognosis is, on the whole, good. In all cases of double facial palsy from peripheral neuritis, there is a similar affection of other parts, and the prognosis must be determined by the general character and course of the affection, and especially the degree to which the morbid blood-state is amenable to treatment.

TREATMENT.—The treatment of facial paralysis depends on the probable cause. When due to its common mechanism—neuritis from cold, hot fomentations to the side of the head and face should be employed in the early stage of the affection, followed by a blister behind the ear. A blister should never be applied in front of the ear, over the opening of the canal, because it will cause some subcutaneous cellulitis, and such cellulitis may actually cause facial neuritis by spreading to the nerve-sheath in the canal. The side of the head should be protected from cold. Internal treatment of neuritis must vary according to the cause. If this is syphilis, iodide of potassium in 10-gr. doses may be given; if exposure to cold, diuretics and small doses of mercury in the early stage, followed by tonics. Electricity to the muscles is needed if the damage to the nerve is so severe as to entail secondary degeneration and consequent changes in nutrition. If there is no change in irritability at the end of a fortnight, recovery will probably occur so soon as to make electrical treatment superfluous. If needed, the voltaic current should be used, slowly interrupted by a commutator, or by the positive or negative pole being moved over the individual muscles (according as they react more readily to the one or the other), the opposite pole being placed over the nerve. Only such strength should be employed as produces distinct muscular contraction. Although electricity probably does no more, it keeps up the nutrition of the muscular fibres while the nerve is recovering. Ultimate recovery is probably thus rendered more complete than without electrical treatment. The application can be made quite well by the patient, one electrode being kept over the place where the nerve divides, the other moved (1) across the forehead; (2) along the eyebrow; (3) along the lowered upper eyelid; (4) along the raised lower eyelid; (5) beside the nose to the upper lip; (6) along the upper lip; (7) along the lower lip; (8) from the zygoma to the angle of the mouth. In (3) and (4) the current should be weaker than in the other places. The late contraction which occurs in severe cases is but little influenced by treatment. Gentle elongation of the muscles may be practised, by a sort of facial massage, the fingers being drawn along the course of the muscles. Electrical applications should be stopped, since the stimulation of the sensory nerves may increase the late contraction by a reflex influence; its occurrence shows that a point has been reached when the influence of electricity for good can be but small.

In external mischief, and in intracranial disease, the treatment of the facial paralysis is usually subordinate to that of its cause.

The treatment of double facial paralysis presents no special points for consideration, except when it is part of multiple peripheral neuritis, the indications for which are described in a special article. *See* NEURITIS, MULTIPLE.

W. R. GOWERS.

FACIAL SPASM.—**SYNON.**: Mimic cramp; *Fr. Tic convulsif*. When affecting the eyelids, *Blepharospasm*; *Nictitation*.

DEFINITION.—Spasm, sometimes tonic but more often clonic, or both, involving some or all of the muscles supplied by the facial nerve, on one side or on both.

ÆTIOLOGY AND SYMPTOMS.—Spasm in the face may be part of a wider convulsive movement, as in epilepsy, hysteria, chorea, or torticollis, diseases dependent on central changes. That which begins in, and is limited to one side of the face, may be due to irritation of the trunk of the facial nerve by growths, by an aneurysm, or caries of the temporal bone, or to an actual lesion of the motor centre for the face in the cortex of the brain. Spasm due to disease of the nerve may follow facial paralysis (*see* FACIAL PARALYSIS). Much more frequent, however, are cases, unilateral or bilateral, in which the affection comes on gradually, chiefly in females in the second half of life, after depressing emotion. It is probable that such cases are due to deranged functional action of the cortical motor cells, through which, we may remember, emotion is so extensively expressed. In such cases the affection usually begins on one side, often in the orbicularis palpebrarum; and if it spreads to the other side, this muscle is first involved. In another group of cases, affections of the eye lead to spasmodic closure of the lids—*blepharospasm*. In other cases beginning in the eyelids, no local cause can be detected, as in the local clonic spasm affecting the orbicularis palpebrarum, known as ‘involuntary winking’ or *nictitation*, which seems to be a pathological development of the normal process. This is also a common seat of the spasm in the face that is part of ‘habit-spasm’ or ‘habit-chorea,’ but it has no connection with true chorea.

Facial spasm is painless. The spasm is usually increased by emotion, by voluntary movement, and by stimulation of the fifth nerve. Slight aural symptoms have been observed, referable to contraction of the stapedius muscle. Usually the movement remains limited to the face; sometimes it spreads to the muscles of mastication, of the neck, &c. The spasm, in typical cases, is both tonic and clonic, clonic paroxysms occurring from time to time, and being fixed by tonic contractions, or *vice versa*. All forms cease during sleep.

The only secondary form of facial spasm (besides that which supervenes on a paralysis of the nerve) is the clonic spasm in the frontales that is often met with in the form of ‘torticollis’ to which the writer has given the name of ‘retrocollic spasm.’ In this there are bilateral contractions in the muscles of the back of the neck, jerking the head backwards. These muscles are physiologically associated with the frontales, as may be seen when a person puts the head back to look up, and this association is reproduced in disease.

PROGNOSIS.—The prognosis of facial spasm is seldom good. If the cause can be discovered, it can rarely be removed, and the affection is generally most obstinate.

TREATMENT.—General tonics and nervine sedatives are the chief agents in the treatment of most forms of facial spasm. When there is evidence of direct irritation of the fifth or the facial nerve, counter-irritation by blisters may be tried. A careful search should be made for reflex causes, and if possible they should be removed, decayed teeth

being extracted, and neuralgia relieved, although the result is generally disappointing, if the spasm has long existed. When there are tender places in the course of the fifth nerve, pressure on which stops the spasm, the hypodermic injection of cocaine at these places should be employed. Morphine, used as a sedative, has been successful, but only after months of use, and its influence makes the remedy almost worse than the disease. Iron, quinine, and valerianate of zinc are the best general tonics. The writer has once known recovery to follow the administration of bromide, Indian hemp, and nux vomica; the case was characteristic, occurring late in life. The operation of nerve-stretching has often arrested the spasm for a time, and, in a few cases, permanently. It deserves a trial in severe cases, acting, perhaps, by the effect on the centres of the arrest of afferent impulses produced by the muscular contractions, or by the upward influence on the nucleus which has been proved to follow severe neuritis. Its effect might probably be augmented by the simultaneous use of sedatives, such as the injection of cocaine to lessen the influence of the fifth nerve on the facial centre. Where the affection springs from an habitual movement, facial gymnastics may be of service. A weak galvanic current, applied from the ear to the muscles, unbroken, has been recommended; but it rarely, if ever, effects a cure. The same is true of the application of galvanism to the sympathetic and to the back of the head. See EYE, AND ITS APPENDAGES, Diseases of.

W. R. GOWERS.

FÆCES, Examination of.—See STOOLS.

FÆCES, Involuntary Discharge of.—See DEFÆCATION, Disorders of.

FÆCES, Retention of.—See CONSTIPATION; DEFÆCATION, Disorders of; and INTESTINAL OBSTRUCTION.

FAINTING.—A popular synonym for syncope. See SYNCOPE.

FAINTNESS.—Faintness signifies a feeling of great weakness or exhaustion, as if the subject of it were about to faint.

FALLING SICKNESS.—A popular synonym for epilepsy. See EPILEPSY.

FALLOPIAN TUBES, Diseases of the.—The following morbid conditions of the Fallopian tubes will be considered here: (1) Abnormalities; (2) Hernia; (3) Inflammation (including *Perimetritis* and *Pyosalpinx*); (4) Tuberculosis; (5) Tumours.

For TUBAL PREGNANCY, see PREGNANCY, Diseases and Disorders of.

1. **Abnormalities.**—Accessory ostia and pedunculated tufts of fimbriæ are sometimes found near the outer end of the tube. In uterine malformations the tubes may be wanting, or imperforate, but with well-developed ostia and fimbriæ. Great elongation of the tubes results from the growth of parovarian cysts, while atrophy is produced by torsion or pressure.

2. **Hernia (*Salpingocele*).**—The tube usually accompanies an ovarian hernia, occasionally it is found alone in an inguinal or femoral sac. By displacement of the iliac peritoneum the tube may

be dragged into the inguinal canal without its ovary. Acute symptoms of strangulation will be caused by nipping or torsion of the herniated tube, needing relief by operation.

3. **Inflammation (*Salpingitis*).**—This is caused by (1) Sepsis following abortion or labour—by far the most common cause—or resulting from uterine operations, sloughing fibroids, uterine cancer, and the exanthemata; (2) Gonorrhœa; (3) Tuberculosis; and (4) Bowel-infection in rare instances. Gonorrhœal salpingitis is frequent, but usually less severe than that due to septic endometritis. The inflammatory changes in acute salpingitis are catarrhal or purulent in character. The tube becomes much swollen and tortuous, its mucosa acutely inflamed, and the muscular coat widely infiltrated. Thus the tissues are rendered so friable that the tube may be completely divided in tying a ligature round it. In chronic salpingitis, apart from dilatation of the tube, its lining membrane is found thickened and warty, or occasionally cystic from fusion of rugæ; and the muscular coat becomes sclerosed from interstitial fibrosis.

The effects of salpingitis are of great clinical importance and are due to extension of inflammation from the tube to the peritoneum and ovaries, or to obstruction and dilatation of its lumen.

(a) *Perimetritis.*—When the peritoneum becomes infected from the tube, a rapidly fatal peritonitis may result, but this is rare. Usually the reaction is limited to the pelvic peritoneum, and the lesion is then termed perimetritis. Adhesions are quickly formed around the end of the tube, uniting it to the ovary, broad ligament, and adjacent structures. Large collections of pus among these adhesions constitute a *perimetric abscess*, and if the exudation is serous it is termed *perimetric cystoma*. Such collections are generally found behind the broad ligaments between the tube, ovary, and adjacent bowel.

(b) *Oophoritis.*—Extension of inflammation to the ovary usually ends in adhesions or abscess. The exudation around the ovary which unites it to the thickened tube and broad ligament ultimately forms a tough capsule, which interferes with the rupture of follicles and may conduce to cystic changes in the ovary. When suppuration occurs, a large abscess with thick rugose lining membrane is formed in the substance of the ovary, or there may be multiple foci from suppuration of pre-existing cysts and dilated follicles. Purulent infection takes place through the apposed wall (rarely the ostium) of the inflamed tube. The term *tubo-ovarian abscess* is applied when an abscess in the ovary is in direct communication with a dilated tube.

(c) *Closure of tube.*—This is effected either by adhesions of the ostium to the adjacent parts (perimetric closure) or by retraction of the fimbriæ and gradual union of the thickened edges of the ostium (salpingitic closure). The uterine end becomes readily obstructed by swelling of the mucosa, or kinking of the tube. Thus the secretions are retained and cause dilatation of the ampullary segment into pyriform cysts known as *pyosalpinx*, *hydrosalpinx*, or *hematosalpinx* according to the nature of their contents.

(d) *Adhesions.*—Though in mild cases salpingitis may end in complete resolution, the process of cure generally involves the production of permanent fibrous adhesions around the uterine appendages, which may destroy the functions of the organs and induce sterility, or fix the uterus in severe retrover-

sion, or cause painful traction on the intestines and omentum.

(e) *Pyosalpinx*.—Pyosalpinx is a retort-shaped tumour produced by distension of the tube with pus, and due to septic, gonorrhœal or tubercular salpingitis. After closure of the abdominal ostium the dilated ampullary portion sinks behind the broad ligament, and arching round its ovary becomes adherent to the uterus and adjacent structures. The sac is lined by granulation-tissue, its muscular coat is deeply infiltrated with young inflammatory growth, and the serous surface is covered with fibrous adhesions from perimetritis. When distension has occurred rapidly the pyosalpinx may rupture and set up fatal peritonitis; more often it excites recurrent attacks of local peritonitis and gradually enlarges; or it discharges into the rectum, vagina, or bladder. The tuberculous pyosalpinx is usually large and remarkably quiescent. Its serous surface is often devoid of adhesions, the fimbriæ not destroyed, the wall ulcerated and thinned by distension, and the cavity filled with caseous pus and cholesterol. Though histological evidence of tuberculosis may have disappeared from the wall of the pyosalpinx, it will always be found in the undilated uterine segment.

(f) *Hydrosalpinx*.—Hydrosalpinx resembles pyosalpinx in general outline, but differs from it in thinness of wall and the watery nature of its contents. The mucosa is atrophied from distension but not completely destroyed, and the peritoneal adhesions are few. Hydrosalpinx is probably due to catarrh from mild salpingitis, the ostium being previously sealed by local peritonitis. The fluid is sterile and becomes absorbed by the peritoneum if the cyst bursts.

(g) *Hæmatosalpinx* may occur in hæmorrhagic salpingitis, especially if tubercular; but its chief cause is tubal gestation. It may also result from torsion of the pedicle of a distended tube, retention of menses, &c.

SYMPTOMS.—The symptoms of acute salpingitis are practically those of its complications. Given a septic or gonorrhœal endometritis, the existence of salpingitis is usually declared by the onset of perimetritis. In a case of moderate severity there will be acute pain and tenderness in the lower part of the abdomen and in the inguinal regions, indicating local peritonitis. Considerable pyrexia, rapid pulse, vomiting, and constitutional disturbance are also present. On vaginal examination apart from enlargement of the uterus there is at first little to be detected beyond fulness and excessive tenderness in the lateral fornices. But as the inflamed appendages sink into the lateral fossæ and the exudation increases, a definite swelling is felt behind the uterus in the posterior quarters of the pelvis.

When pus is rapidly formed there will be hectic fever, marked wasting, sweating, and leucocytosis, together with a tender fluctuating swelling in the pelvis. But suppuration is generally slow and is indicated by recurrent attacks of pelvic peritonitis or 'inflammation of the bowels,' due to leakage of pus into the peritoneum from a pyosalpinx or ovarian abscess.

The clinical signs of chronic salpingitis are usually pronounced. There is constant pelvic pain radiating down the thighs, relieved by lying down but increased by walking, defecation, and coitus. Menorrhagia, dysmenorrhœa, and sterility are also present. The menstrual pain usually precedes the flow by one or two days and continues throughout

the period; sterility results from obstruction of the tubes, or matting of the appendages by perimetritis. *Per vaginam* there is evidence of thickening in the lateral fornices, with retroversion and fixation of the uterus, or a fluctuating pelvic tumour is felt pushing the uterus forward.

The *diagnosis* of salpingitis in general rests upon the previous history of a septic abortion or prolonged vaginal discharge, with acute or chronic attacks of pelvic peritonitis, and the tangible evidence of a bilateral lesion in the site of the uterine appendages. Dilated tubes may frequently be recognised by their shape and position, but they cannot always be distinguished from small ovarian cysts and other pelvic tumours, nor can the nature of their contents be always inferred from the physical signs.

TREATMENT.—The treatment of acute salpingitis is that of the accompanying perimetritis, viz. prolonged rest in bed, hot vaginal douches, laxatives, and hot fomentations or counter-irritants to the lower part of the abdomen. It is hardly possible to over-estimate the value of rest for the relief of acute pelvic inflammation. Under its influence tenderness and pain subside, and the inflammatory swelling in the pelvis gradually gets smaller until it ceases to give trouble. A considerable proportion of cases however relapse and suffer from recurrent attacks of inflammation, or pass into the condition of chronic salpingitis. When the presence of pus is suspected, the swelling must be explored and the abscess drained either through the vagina or by abdominal section. The former route is chosen when the purulent collection bulges into the pouch of Douglas, and a large pyosalpinx may thus be efficiently drained. If the swelling is high up in the pelvis, as is usual with perimetric abscess, it will be more safely reached through the abdomen. Abdominal section will also be required for the removal of suppurating tubes and ovaries, but this operation is attended with many risks, such as general infection of peritoneum, hæmorrhage, laceration of bowel, and persistent sinus from infected ligatures. Hence it should not be performed until all acute inflammatory symptoms have subsided. When removal of both appendages is absolutely necessary on account of suppuration, it is generally better to include the uterus by supravaginal hysterectomy. In chronic salpingitis hot douches, counter-irritants, iodine, ichthyol, salines, and tonics usually afford relief; and the administration of mercury is often desirable. Curetting is required for persistent discharge from endometritis, and in some cases of painful retroversion with matted appendages separation of adhesions by laparotomy is justifiable.

4. Tuberculosis.—Tuberculosis is stated to be present in 10–15 per cent. of all cases of inflammatory disease affecting the uterine appendages. Infection is usually conveyed by the blood-stream from tuberculous foci in the lungs and other organs, but the lesion may be due to direct extension from tubercular peritonitis, or an adherent tubercular ulcer of the intestine, or from the uterus and lower genital tract infected from without. Though primary tuberculosis is undoubtedly rare, yet cases regarded clinically as ordinary salpingitis have not infrequently proved tubercular.

Miliary tuberculosis of the serous coat of the tube and mesosalpinx occurs as part of a general tuberculosis, or as a direct infection of the peritoneum from diseased tubes; in the latter instance the tubercles will be most numerous in the pelvic

region. In chronic tubercular peritonitis the broad ligaments and tubes become plastered to the front and sides of the pelvis by exudation, yet the tubes may show little or no disease, and the fimbriae are usually not obliterated. In miliary tuberculosis of the tube the mucous membrane is irregularly thickened and yellowish from the development of tubercles in its rugæ. The epithelial covering is intact and there is little caseation or inflammatory change. Caseous tubercle is the most common type, and the whole length of the tube is affected, or the lesion has a nodular distribution. There is now much thickening, the lumen is filled with caseous material, and the mucosa deeply ulcerated or replaced by tuberculous granulation-tissue. Miliary tubercles are found to a varying depth in the muscular coat, and the tube is surrounded by dense perimetric adhesions which are inflammatory in origin. In the fibroid stage there is an excessive development of fibrous tissue which represents a curative process. The tube becomes shrivelled up, the tubercles disappear, and the caseous material slowly undergoes cretification. As in other organs the effects of tubercular salpingitis depend much upon the associated inflammation. If the ostium becomes closed, the tube may be gradually converted into a large pyosalpinx; acute tuberculosis will also produce hæmatosalpinx. Besides extension to peritoneum or endometrium, the disease may end in chronic pelvic suppuration which proves fatal by prolonged discharge, fæcal fistulæ, or general tuberculosis.

SYMPTOMS.—While there are no characteristic symptoms of tubercular salpingitis, diagnosis is often possible upon general considerations. A history of tuberculosis and the physical signs of phthisis or tubercular peritonitis are very important; and conversely, absence of the common causes of salpingitis (sepsis and gonorrhœa) is significant. Tubal disease in the virgin is almost invariably tubercular. Menorrhagia and dysmenorrhœa are usually present, but if the patient is much exhausted by phthisis there will be amenorrhœa. Locally a dense nodular thickening is detected in the site of the uterine appendages, which differs from that of ordinary salpingitis chiefly in its chronicity. The disease may occur at any age, even in early childhood.

TREATMENT.—Removal of the diseased appendages is attended with considerable risks, because the dense adhesions and the friability of the bowel from the accompanying tubercular peritonitis predispose to fæcal fistula. Good results, however, follow simple abdominal incision and evacuation of fluid without removal of the tubes. If suppuration occurs, the abscess may be successfully drained through the vagina, but the quiescent tuberculous pyosalpinx above described is best treated by abdominal section. The uterus should be removed together with the adherent appendages if the operation is thereby simplified. When the pelvic lesion is of less importance than the condition of the lungs, the treatment should be entirely medical.

5. **Tumours.**—Tumours of the Fallopian tubes are very rare, papilloma and primary carcinoma being the only forms of clinical importance. Simple *polypii* occur as small outgrowths from the mucosa; they must be distinguished from *placental polypii* due to incomplete tubal abortion. *Lipomata* may develop in the subserous tissue and become pedunculated like the appendices of the colon; but they are not found in the wall of the tube. Under

the term *myoma* several formations have been included; some were displaced fibroids of the uterus, others were localised thickenings of the tube due to chronic salpingitis, or represented an imperfect cornu of the uterus. True encapsuled myomata are very rarely seen. Pedunculated *cysts* are sometimes found hanging from the outer end of tube, and the Hydatid of Morgagni may attain the size of a pear. Two cases of great distension of the tubes with true hydatid cysts have been recorded.

Papilloma is a very rare neoplasm which may follow long-standing salpingitis. The tube becomes distended with a sessile papillary growth, the secretion from which may cause ascites, or, if the abdominal ostium be closed, produce intermittent vaginal discharge of sero-sanguineous fluid. As in the urinary bladder, papilloma of the tube is closely related to villous carcinoma, though in several cases the growth has not recurred after removal.

In *primary carcinoma* the tube is also distended with a villous columnar-celled growth from the mucous membrane, and sanious watery or puriform fluid is periodically discharged into the uterus. The surface of the growth becomes necrotic, but its base invades the tube and ultimately extends to the ovary, peritoneum, uterus, and pelvic glands. This disease occurs in sterile women from 45 to 55, and causes attacks of severe abdominal pain followed by profuse watery discharge. A fixed hard nodular or partly cystic tumour is felt in one posterior quarter of the pelvis with signs of ascites and cachexia. In two-thirds of the recorded cases death has resulted from recurrence within a year of operation. Extension of carcinoma from ovary or uterus to the tube is occasionally found in advanced stages of the disease. Doubtful cases of *sarcoma* and *decidua malignum* of the tube are on record. J. H. TARGETT.

FALSE MEMBRANE.—An inflammatory exudation of a fibrinous character, which is deposited in layers, chiefly on mucous surfaces, and occasionally on abrasions of the skin. It is well exemplified in diphtheria and plastic bronchitis. See DIPHTHERIA.

FARADISATION, Uses of.—See ELECTRICITY IN MEDICINE.

FARCY.—See GLANDERS.

FASTING.—The manifestation of vital activity implies consumption of material; and unless the supply of material in the form of food is equivalent to the loss occurring, a progressive wasting of the body and failure of power must ensue.

PATHOLOGY.—To Chossat we are indebted for showing that the immediate cause of death from fasting is a reduction of the body-temperature. At first there is a gradual but not very extensive fall. Afterwards a more rapid decline occurs, until the reduction amounts to about 29° or 30° F. below the normal point, when death ensues. Chossat noticed that if, while in the state of torpor preceding death, the temperature of the animal experimented on was raised by exposure to artificial warmth, a restoration of consciousness and muscular power was induced; and some of his subjects of experiment which were thus rescued from impending death afterwards thoroughly revived on being supplied with food.

SYMPTOMS.—The most prominent symptoms arising from fasting are those due to the special sensations produced by the absence of food and fluid,

and those attributable to a decline of the physical and mental powers. In the first place there is great uneasiness in the epigastrium. This is followed by a sense of sinking in the same region, accompanied by insatiable thirst; and if fluid be persistently withheld as well as food, the thirst becomes the chief source of distress. The countenance assumes a pale and cadaverous appearance, and a look of wildness is presented about the eyes. Emaciation becomes more and more marked, and with it there is a decline of the bodily strength. There is also failure of the mental power. Stupidity may advance to imbecility, and a state of maniacal delirium frequently supervenes. Life terminates either calmly by gradually increasing torpidity, or, it may be, suddenly in a convulsive paroxysm.

DURATION OF LIFE.—The usual duration of life under complete absence of food and drink may be said to be from eight to ten days. The special circumstances existing may, however, exert a modifying influence, and from the nature of these the period may be either diminished or increased. A stout person, as may be readily understood, has a chance of living longer than a thin one, on account of the store of combustible material which may be drawn upon being larger. Exposure to cold in conjunction with starvation very much hastens death. The presence of moisture in the atmosphere favours the prolongation of life, by diminishing the exhalation of fluid from the body. It may be assumed to be owing to the existence of warmth and moisture that persons buried in mines or otherwise similarly placed have been known to live considerably beyond the ordinary period.

The duration of life may be considerably lengthened if drinking-water is available. Experiments on dogs show that life can be maintained in this way for five or six weeks.

The Welsh fasting girl, whose case caused so much sensational excitement in 1869, lived exactly eight days from the time she was placed under systematic inspection to solve the problem of whether she could exist, as had been alleged by her parents, for an indefinite period without food. It appears that during the first part of the time she was cheerful and exhibited nothing extraordinary. Later on it was found that she could not be kept warm, and ultimately she sank into a state of torpor from which she could not be roused, and which speedily terminated in death.

In the Troedyrhiw colliery near Pontypridd an inundation occurred in 1877, which led to the imprisonment of four men and a boy in one of the headings of the mine. The accident happened on Wednesday evening, the 11th of April. Efforts were at once made, by means of a cutting, to reach the chamber in which the imprisoned persons were confined, and to release them. This was not accomplished till the afternoon of Thursday, the 19th, when all were rescued alive and did well. They had been imprisoned in an atmosphere of compressed air nearly eight days, without food but within reach of water.

That a very small amount of food will suffice for sustaining life for a prolonged period is evidenced by what may be observed in some cases of constrictive disease affecting the œsophagus or cardiac orifice of the stomach.

TREATMENT.—Caution is required in the administration of food after prolonged fasting. Sudden transitions of all kinds are trying to the body; and,

instead of allowing the rescued sufferer to gratify his desire to eat and drink according to his inclination after several days' abstinence, the supply of both food and drink should at first be limited, and afterwards gradually increased. There is reason to believe that the non-observance of this rule has upon some occasions been followed by disastrous consequences, which a different plan might have averted.

F. W. PAVY.

FAT-EMBOLISM.—See EMBOLISM.

FAT-NECROSIS.—This term is applied to a peculiar change met with chiefly in the subperitoneal adipose tissue, but also occasionally elsewhere. Among the normal fat are seen small opaque white masses, the size of a hazel-nut, or smaller. They are sharply defined in outline, and the cells are seen microscopically to be opaque and granular, the contents being occasionally crystalline. The consistency of the nodules is firm, and they react like fat to heat and to osmic acid. The nature of the change which has taken place in the fat is still unknown. It has been referred with some probability to the action of the pancreatic secretion, since it is frequently found associated with affection of this gland. It has also been held to be due to the action of bacterial toxins, or to a primary necrosis, the result of overgrowth of adipose tissue. Rolleston has suggested that the condition may be a 'trophic' change due to disturbance of the abdominal sympathetic. W. CECIL BOSANQUET.

FATIGUE (*fatigo*, I weary).—Fatigue is the inability of an organ or of an individual to perform its functions normally, due to partial exhaustion from overwork.

Periods of functional activity invariably alternate with periods of repose, during which the waste caused by the exercise of function is repaired. Fatigue occurs directly we attempt to prolong the periods of tension at the expense of the periods of relaxation, and when we find patients pursuing their avocations too zealously we know that, if such offence be persisted in, fatigue will be followed by exhaustion, and that some form of 'break-down' will be the result.

General Fatigue.—This consists in a disinclination, followed, if rest be not obtained, by a disability to perform either mental or physical work, and this disability is first noticed in work requiring attention or sustained effort, and last in those acts which have become automatic.

Local Fatigue.—Local fatigue is a term generally reserved for the earliest signs of exhaustion in some particular muscle or group of muscles. Any one who has attempted to hold out a weight at arm's length knows the impossibility of continuing the effort for any length of time, and it is proverbially true that standing in one position is, to most people, far more tiring than walking, the reason being that in standing the muscles which support the body are subjected to a prolonged strain, while in walking we use the muscles on either side of the body alternately. The great increase of power which we obtain by using the muscles on each side of the body alternately would seem to be one of the chief reasons for the bilateral symmetry of the body. Not only is sustained effort a far more potent cause of fatigue than repeated effort, but we find that as fatigue supervenes, actions requiring sustained effort are the first

to fail: in this respect local fatigue resembles general fatigue. It is quite possible to exhaust a muscle by artificial stimulation; thus, if one of the small interosseal muscles be continuously faradised, it will be found that in time its power of contraction to any form of stimulus is temporarily abolished.

The symptoms of local fatigue are loss of power, tremor, cramp-like contraction, and dull, aching pain.

The treatment of fatigue in all its forms is *rest*, and if this be properly regulated exhaustion may be warded off and recovery rapidly ensue. See EXHAUSTION; and OCCUPATION-DISEASES.

G. V. POORE.

FATTY DEGENERATION.—SYNON.: Fr. *Dégénérescence graisseuse*; Ger. *Fettige Metamorphose*.—Fatty accumulation and fatty degeneration are in all probability due to distinct pathological processes. On the one hand certain tissues which normally possess the power of storing up fat, such as connective tissue and liver-cells, may contain an unusual amount of this substance, or the storage may take place in unusual parts of the body or under abnormal circumstances—fatty accumulation or infiltration. On the other hand, the actual protoplasm of cells may break down and give rise to fat as a product of disintegration—fatty degeneration or metamorphosis. Much valuable work upon the latter process was done by the late Sir Richard Quain, and later experiments by Voit appeared to show that in starving dogs, whose livers would not naturally contain fat, small doses of phosphorus produced not only an increase in the excretion of nitrogen, but the appearance of fat in the liver-cells. The action of this poison thus apparently led to direct conversion of some of the cell-protoplasm into fat. Recent experiments by Rosenfeld tend, however, to cast some doubt upon this cause at least of fatty degeneration. This observer found that by first starving dogs till most of the fat in their bodies had disappeared, and then feeding them upon sheep's tallow, he could cause this latter substance to take the place of the normal fat found in these animals. On administering to dogs thus treated small doses of phosphorus he found that the fat so produced in the liver-cells, usually regarded as due to breaking down of the protoplasm, was identical with the tallow stored elsewhere. He therefore concluded that the fatty liver produced by phosphorus-poisoning is due to storage of fat, which has been set free to circulate in the blood, and not to actual degeneration. These experiments need further confirmation, and meanwhile the classification usually adopted will here be followed. The process usually named fatty infiltration is better called fatty accumulation, since it is improbable that a merely passive process such as is indicated by the former expression can take place in a living cell. The fat formed in these cases is almost undoubtedly taken up by an active vital process.

Fatty accumulation or infiltration.—So long as the deposit of fat is confined to places in which it is normally found, it is impossible to draw any distinct line between normal and pathological accumulation. Very great accumulation of fat in the omentum or over the surface of the heart is evidently injurious, but the exact point at which it becomes so cannot be determined. When, however, the connective-tissue cells between the cardiac muscle-fibres take up fat it is certain that the change

must be regarded as pathological: the deposit has taken place in an abnormal locality. Fatty accumulation results from (1) Excess of food; (2) Diminished wear and tear of tissues, such as results from lack of exercise; (3) Defects in the aëration of the blood, as in anæmia or chronic disease of the lungs; (4) Hereditary tendency to storage of fat, certain families showing a distinct liability to develop obesity.

In the *liver* fatty accumulation occurs in connection with general obesity, but it is also a result of exhausting diseases, such as chronic phthisis or carcinoma, accompanied by general emaciation. In the latter cases it is due apparently to diminished oxidation of fat. The fatty liver is enlarged, pale, and flabby, with smooth surface and somewhat rounded edges: it tends to 'pit' on pressure with the finger. On cutting into it and washing the surface of the knife used, oil-globules may be seen floating on the water. Microscopically the cells are found to contain large drops of fat which distend the cells and may push the nucleus to one side. The periphery of individual lobules is the part in which the deposit of fat is most advanced. In the *muscles* the fat occupies the cells of the connective tissue lying between the fibres: the condition is frequently met with in lesions of the cortico-spinal motor neuron (upper-segment lesions), and in muscles which are unused owing to ankylosis of joints. In the *heart* the fat lying between the muscular elements is a cause of embarrassment to the organ and constitutes a condition of considerable gravity. This is the common form of 'fatty heart' met with in obese subjects. It is probably in this class of cases that most benefit may be derived from graduated exercises which tend to reduce the fatty deposit and restore the heart to a more normal condition.

Fatty degeneration.—In this case the fat found in the cells results from breaking down of their protoplasm, and indicates a diseased condition of the cells themselves. The causes which result in this degeneration are (1) Defective supply of nutriment to the cells; (2) Natural defect of vitality; (3) The action of certain poisons; (4) Disturbance of 'trophic' nervous influence.

(1) Defective supply of nutriment may result from occlusion of vessels supplying a part, as occurs in cases of disease (atheroma) of the coronary arteries of the heart, or of softening of the brain due to cerebral thrombosis. A relative deficiency of food-supply may occur if cells are overworked, metabolism being thus more rapid than the vessels can maintain. In profound anæmia the poverty of the blood appears to be sufficient to cause some fatty degeneration, but it is not impossible that in pernicious anæmia and allied conditions there may be poisons circulating in the blood which actually produce this effect. (2) In old-age the gradual death of cells is accompanied by fatty degeneration: the same change met with in the cells of rapidly growing tumours may be due either to natural deficiency of vitality in these growths or to inability of the vascular channels to keep up an adequate supply of nutriment. (3) Poisons which appear to cause fatty degeneration are phosphorus, arsenic, and alcohol, also the toxins formed by many varieties of bacteria, such as the *Bacillus tuberculosis* and *B. diphtheriæ*. A special acid substance secreted by the tubercle-bacillus gives rise to fatty degeneration and necrosis of the cells

in tubercular foci (*see* CASEATION). The acute fatty degeneration of the liver met with in acute Yellow Atrophy of this organ is very probably produced by some infective organism. (4) As a manifestation of nervous disturbance, fatty degeneration is met with in muscles paralysed by lesions of the spino-muscular neuron (lower-segment lesions), as in peripheral neuritis and anterior poliomyelitis.

The fat first appears in the cells as small particles which are stained black by osmic acid: these particles are minute and do not coalesce to form such large drops of fat as are met with in fatty accumulation. If the fat is dissolved out, the protoplasm of the cells appears granular. Cells which have undergone extreme fatty degeneration may appear as mere collections of granules ('granular cells'), the nucleus as well as the protoplasm having suffered disintegration. The tissues most frequently affected are the heart and voluntary muscles, the arteries and the liver. The epithelium of the tubules of the kidneys also undergoes this change as the result of chronic inflammation. In the *blood-vessels* fatty degeneration affects most frequently the intima of arteries, the affected patches appearing of a pale yellow colour, more opaque than neighbouring parts, distinguished from atheroma by their superficial character and by the ease with which they can be stripped off, leaving healthy tissues beneath. If the fatty cells break down, small erosions of the intima result. The middle coat of vessels may also undergo fatty degeneration, especially in connection with atheroma: capillaries may also be affected. In the voluntary muscles the condition is not of any great importance apart from the conditions which give rise to it, but in the *heart* fatty degeneration constitutes a very grave disorder. The change may be either local or general. The local or circumscribed form results from local defects of nutrition due to vascular occlusion. It is most frequently due to atheroma affecting the smaller branches of the coronary arteries, the narrowing or obliteration of these vessels affecting the particular areas of the heart to which they are distributed. Patches of fatty degeneration may be seen scattered as pale yellowish areas here and there throughout the myocardium. If any of these are extensive enough to involve the whole thickness of the cardiac wall, rupture or aneurysm of the heart may result. In less severe cases the fatty cells are gradually absorbed and replaced by a growth of fibrous tissue, constituting one form of 'fibroid heart' or 'myomalacia cordis.' In the general or diffuse form of fatty degeneration due to constitutional causes or toxæmia, the muscular fibres of the heart are affected throughout its whole substance, but the change is not equally advanced in all of them. The myocardium is soft and pale, and a peculiar mottled or striated appearance may be seen, especially in the columnæ carneæ, due to unequal involvement of groups of fibres. This appearance has been compared to the marking on a tabby cat or on a thrush's breast ('tabby-cat' or 'thrush-breast' striation). Sudden death from heart-failure may occur either in this or the previously described form of fatty heart. It is important to distinguish the two conditions, since active treatment suitable for the fatty accumulation would be very inadvisable in cases of true fatty degeneration. The causes of fatty degeneration of the *liver* have been already alluded to.

W. CECIL BOSANQUET.

FATUITY (*fatuus*, silly).—Mental imbecility. *See* IMBECILITY.

FAUCES, Diseases of.—*See* THROAT, Diseases of.

FAVUS.—*See* TINEA FAVOSA.

FEBRICULA (*febricula*, slight fever).—**SYNON.**: Fr. *febricule*; Ger. *Febricula*.

DEFINITION.—A febrile attack of not more than a few days' duration, not preceded by any invariable antecedent, and not attended by any definite organic lesion. The term 'febricula' includes a number of different forms of evanescent fever, the cause and nature of which are uncertain.

ÆTIOLOGY.—Cases of febricula may be thus classified:—

1. *Unrecognised modifications of known diseases.* Short febrile attacks occurring during epidemics of typhus, typhoid, or relapsing fevers, scarlatina, measles, rubella, and variola, have been described as febricula, although it is not improbable that these cases were really instances of the more serious epidemic disease prevalent at the time. It is possible that mild and unrecognised cases of tonsillitis, influenza, and rheumatic fever may also be thus diagnosed, and commencing endocarditis overlooked. It is moreover probable that many cases diagnosed in young children as febricula are due to slight septic absorption from inflamed post-nasal adenoids.

2. *Febrile attacks probably due to the introduction from without of bacteria or toxins, not yet identified, or to the action of chemical poisons formed in the tissues (auto-intoxication).*—The toxins of the many organisms which frequently obtain entrance to the respiratory and alimentary tracts may readily be absorbed by the mucous membrane, and thus cause passing symptoms. Slight febrile attacks, the nature of which is generally recognised, may occur in adults after prolonged exercise. Similar fatigue may in children cause febrile attacks even more readily, but the nature of these generally passes unnoticed. A slight febrile attack, accompanied by a rapidly disappearing erythematous eruption, sometimes follows the administration of rectal enemata, especially of soap and water.

SYMPTOMS.—Febricula is characterised by a rise of temperature, rarely exceeding 102.5° F. The access of fever may be gradual, or marked by slight rigors; and some or all of the common clinical symptoms of fever may be present in varying proportion and in greater or less degree, such as—general malaise; dry skin; frequent pulse, amounting to 100 or 120 per minute; increased frequency of respiration; furred tongue, thirst, loss of appetite, and nausea; constipation; scanty, high-coloured urine; and headache, intolerance of light, slight deafness, restlessness, sleeplessness, and slight delirium at night. An erythematous rash—general or local—is occasionally observed.

DIAGNOSIS.—The diagnosis of febricula rests upon the exclusion of all the other recognised kinds of fever. As a matter of practical diagnosis at the bedside, almost every disease attended by rise of temperature is now and then, at its outset, mistaken for febricula. The condition now known as glandular fever is readily distinguished by the accompanying enlargement of the lymphatic glands. *See* GLANDULAR FEVER.

PROGNOSIS.—The prognosis depends upon the degree and duration of the pyrexia, but is almost always favourable.

TREATMENT.—In the absence of any special indication, rest in bed until twenty-four hours after the temperature has fallen to normal, liquid food until the desire for solids returns, and, if constipation be a marked feature of the case, a moderate dose of some mild purgative, will be sufficient. It is, however, always prudent to remember that what seems to be febricula may be the beginning of some serious and perhaps highly infectious disease. Cooling drinks, such as citrate of potassium in effervescence, Liquor Ammonii Acetatis with a little spirit of nitrous ether; or dilute nitro-hydrochloric acid (one drachm to a pint of water) with some fresh lemon-juice added, may be given according as the one or the other is grateful to the patient. Any active treatment, except sponging the skin, is unnecessary and sometimes dangerous.

H. MONTAGUE MURRAY.

FEBRIFUGES (*febris*, a fever; and *fugo*, I drive away).—**SYNON.**: Antipyretics.

DEFINITION.—External applications or internal remedies which tend to lower the bodily temperature, when it has been abnormally raised by the processes of fever.

ENUMERATION.—The principal febrifuges are:—Cold Baths; Cold Affusion; Wet Pack; Tepid Baths; Alcohol; Diaphoretics; Salicylic Acid and its Salts, Quinine, Digitalis, Aconite; Chloral Hydrate; Guaiacol, Thymol, and Benzoic Acid; Salicin, Phenazone, Acetanilide, and Phenacetin; and Water and Diluents generally.

ACTION.—Following Professor Binz, we may divide febrifuges into two classes:—(1) those which withdraw heat directly; and (2) those which lessen its production.

1. In the first division we must give the foremost place to cold baths, the cold pack, and cold sponging, which powerfully abstract heat from the surface of the body, and rapidly cool down the blood. Diaphoretics and alcohol act more feebly in the same direction, by dilating the cutaneous arterioles, and thus allowing the mass of the circulating fluid to be effectually exposed to the chilling influence of the air. See COLD, Therapeutic Use of; and DIAPHORETICS.

2. Secondly, we have to consider those drugs which actually check the febrile condition itself, by diminishing the increased tissue-change which is taking place. We explain the antipyretic properties of quinine by their specific influence over the hæmatozoa on which malaria depends; and perhaps the progress of science may soon enable us to arrest other febrile processes by paralyzing or destroying their causes.

The free use of water tends to promote excretion and thus to remove the products of oxidation. Blisters possibly act by causing contraction of the vessels in the inflamed part.

USES.—Antipyretic treatment is not much adopted in this country as a matter of routine, holding as we do that temperatures raised within certain limits are not *per se* elements of danger, and that even although we may effectually cool down our patient, the progress of the disease may go on quite unchecked. But prolonged fever causes excessive tissue-change and fatty degeneration of the heart; and when the thermometer registers 105° F., and still tends up-

wards, we know that dangerous limits are reached, and that as a rule life is not long sustained above 107° F. It then becomes our duty to interfere; and this is best done by plunging our patient into a bath. The temperature of the water in the bath may be as low as 60° or as high as 80°; there is good evidence that a tepid bath of 85° to 95° may reduce the temperature as readily as a colder one, and according to some authorities it is preferable. When the temperature descends to within 4° or 5° of the normal we remove him to bed, remembering the dilatation of vessels which must follow the contracting effect of cold, and the consequent cooling process which must continue to go on. Here, as in all febrile conditions, the thermometer is our surest guide, and we must be directed by it as to when to resume the treatment, for frequent repetition may be needed, and on the Continent as many as 200 baths have been given in the course of a single illness. Along with this some physicians combine the use of large doses of quinine; but, notwithstanding the marked tolerance of the drug under pyrexial conditions, the danger of perilous depression from such free medication is no imaginary one. Apart from ague we find this drug most beneficial in fevers which owe their origin to septic poisoning. It must not be forgotten that quinine takes some hours to produce its maximum antipyretic effect. Digitalis is not a powerful antipyretic, and in large doses is too disturbing to the heart, and too apt to produce gastric derangement, to inspire much confidence. The influence of salicylic acid over acute rheumatism is remarkable, as it seldom fails to reduce temperature and relieve pain in forty-eight hours; but in other feverish conditions its beneficial action is by no means so well-marked. Salicin also has numerous advocates, who prefer it to the salicylates as being less depressing to the circulation. Aconite and diaphoretics are of undoubted service in aiding the defervescence of some of the minor febrile disorders.

Phenazone, Acetanilide, and Phenacetin are all rapidly acting antipyretics: their action is accompanied or followed by diaphoresis; the first is said to act on the thermogenetic centres; acetanilide and phenacetin are believed to act by lessening heat-production: they all appear to have an action on the blood itself, and are injurious in many pyrexial conditions. Guaiacol subcutaneously injected or taken by the mouth has been much lauded in the pyrexia of phthisis: thymol may produce dangerous collapse. Benzoic acid is a mild febrifuge.

R. FARQUHARSON.
SIDNEY PHILLIPS.

FEEDING, Forcible.—See FORCIBLE FEEDING.

FEIGNED DISEASES.—At any moment, and in any class of practice, we may be confronted by feigned disease, or by real disease or lesions induced by factitious means; and failure to differentiate the organic from the functional, or the genuine from the induced and fraudulent, may entail consequences both damaging to our reputation and often harmful to the patient. In the army and navy, in the public service generally, in insurance-practice, in connection with benefit-clubs, with railway compensation-cases, in schools, and in fact under any circumstances where advantages such as pensions or other gain may accompany disease, there will always be some unprincipled persons to whom

the *motive* will appeal. The greatest field for feigned disease is however found among criminals, either awaiting trial for murder or other grave crimes, or after conviction, the advantages attached to successful assumption of irresponsibility or grave disease being immediate and great. It is by no means always incumbent upon us to reveal either to the patient or his friends our discovery or suspicion, but it is of the highest importance that we should rapidly recognise the condition and act upon our knowledge with discretion and probity, a discretion which will dictate widely different action with differing circumstances.

The following classification will conduce to method in our study, while it will help us to arrive at a correct diagnosis by a process of exclusion and elimination.

1. Cases in which the patient, under some morbid impulse, involuntarily feigns disease.

2. Cases of voluntary exaggeration of real morbid symptoms; or of unreal symptoms or manifestations superadded to genuine ones, including deficient powers of subjective analysis.

3. Cases of deliberate and distinctly planned deceit and of factitious disease (*malingering*).

I. Diseases which are feigned not by the direct action of the patient's will, but through an inability to resist the promptings of an unstable and excitable nervous system, are discussed under the articles on HYSTERIA and NEURASTHENIA. Such cases are referred to here in order that it may be borne in mind that what looks like malingering may be Hysterical or 'Neuromimetic,' which appreciation will greatly affect the management of such a case and its development.

It is, moreover, sometimes impossible to decide whether the victim of a railway accident is a pure malingerer, or has become, through the fright and shock he experienced, a real neurasthenic; nor must we forget that a small ligamentous sprain of, or effusion into, a single minute joint of the vertebral column may give no *distinct* objective signs, but may produce a very real disablement. Again the absolute abeyance of energy and active intellectual capacity in a severe neurasthenic case may look very like grave organic cerebral disease or commencing insanity, but reference to the cause will often reveal the true nature of the case. The reflexes in neurasthenia may assume very capricious and varying characters, which may suggest organic nervous lesion; hence they must be observed closely and often, and the very fugitive nature of these phenomena will often relieve us from undue anxiety. Thus we must not diagnose our case from a lost patellar reflex or a slight ankle-clonus until we have watched these manifestations sufficiently critically to enable us to verify their real organic significance or to estimate properly their temporary occurrence after a nervous 'shock.'

II. In this group we must draw attention to the *motives* which may exist for a patient whose illness is, or has been, real to extend the period of his supposed incapacity for work by feigning graver or added symptoms. Thus it often happens that an epileptic tramp or beggar will increase the number of his fits for purposes of self-interest; or the insane patient will add feigned hallucinations to his real condition. Thus a criminal who was mentally affected and on the verge of pronounced insanity discovered that he was being specially observed with a view to his liberty being recommended as a

therapeutic measure. He did not know how nearly he approximated to the suspected diseased state, and feigned a condition much more pronounced than his real case was. His liberation had the effect of immediately restoring his mental balance, which would not have been the case had his *graver* symptoms been genuine. A diabetic patient will sometimes place cane-sugar in his urine if he finds his recovery is likely to cut short his stay in hospital. Real injuries such as fractures are not seldom purposely delayed in their recovery by ill-conditioned men who disturb and even remove the splints. Ulcers are denuded of the advancing margin of healing cuticle; a conjunctivitis is sometimes transformed into a keratitis, or even into a panophthalmitis by intentional irritation. A diarrhoea may be enhanced by purposely taking a pro-scribed diet. A man receiving weekly wages during incapacity following an injury may, by refusing to exercise his recently recovered member or through indolence by neglecting to submit himself to the prescribed massage and movements, induce a rigidity which further or even permanently incapacitates him. Close attention to the progress of cases will generally prevent our being misled, especially if, in examining a patient for a stated *subjective* pain of which we entertain some suspicion, we pretend to have found the pain, which he has localised at one spot, in a new but adjoining locality. Thus a man persistently complaining of an acute but anomalous pain in the left groin was easily induced by a special examination of the left iliac crest to assure his doctor that the new site was the '*only*' seat of his trouble; or a man refusing to return to work because of a lumbago will, if he be asked to bend down and touch the ground, make a great display of suffering, but may be seen to erect his trunk rapidly and without a wince if he thinks the examination is finished and the doctor acquiesces in the existence of this malady. The locality of a 'tender' vertebral spine will often shift if subjected to this test. In cases of 'dimness of vision' following a blow on the head or a fall, an ophthalmic examination will often detect an ametropic condition of one eye which careful enquiry may prove to have existed before the accident, and which only needs the proper lenses for good vision to be regained. This is important because 'affected sight' is so often pleaded by the lawyers as a basis for compensation after injury as pointing to brain-lesion. We must not forget that naturally deficient accommodation in *one eye or single* congenital amblyopia is often first *noticed* after an injury which has not produced it; nor that the lowered general tone following an injury may temporarily increase the deficiency. In connection with recovery from injuries of the extremities, early and persistent passive motion and massage are often necessary to obviate an undue extension of the period of convalescence, though the patient may strongly object to the treatment if he have anything to gain by a protracted disability.

Old-standing rheumatism, osteo-arthritis, gout, or *Dupuytren's Contraction* is often pleaded as due to a recent contusion or slight injury of the hand. A pulseless wrist was presented to the writer by a patient who wished to be thought gravely ill, and some alarm was experienced until a scar was found, resulting from an old wound, which had severed the radial artery—a fact afterwards admitted by the patient.

III. This group comprises all cases of deliberate

'malingering,' where the feigned disease is the expression of unmitigated imposture. In these cases there is always a motive, and the stronger the motive the deeper-laid will be the plan, and the more determined will be the effort to carry it out, in some cases actual death being risked with a gamester's courage. No station of life is exempt from instances of this form of deceit—if only the motive be strong enough. The most powerful motive is self-preservation; hence we see the most consummately devised schemes of simulated madness presented by persons under trial for murder. Moreover, to evade imprisonment or the more arduous and monotonous duties of military or naval life, the most ingenious devices are often adopted. Fraud upon accident-insurance offices is not infrequently attempted in this way. Superannuation from the public service is the motive at times. The escape from responsibility for grave immoral conduct has been the motive for marvellously simulated mental disease. Even young children have at times led practitioners astray by persistently prosecuted physical simulations. Probably a much larger proportion of 'epileptic' seizures—as seen among prisoners and mendicants—are instances of malingering than is generally supposed. Paralysis, neuritis, hæmoptysis, hæmatemesis, persistent vomitings, contractions of muscles or of limbs, blindness, deafness, otorrhœa, incontinence and retention of urine—these and an infinite variety of maladies have been feigned. Small stones have been introduced into the urethra and sand placed in the urine-chamber to simulate calculus; persistent pharyngeal ulceration has been caused by applications of nitric acid, as Semon has related; numerous small fragments of boiled chicken-bones have been removed from a sinus in the hand; rashes have been produced by cantharides and mustard-plasters (*see DERMATITIS VENENATA*); the heart's action and the pulse have been excited by violent muscular movements; thermometers have been rubbed in order to simulate a febrile state; strange and alarming œdemas have been produced and kept up for long periods by ligatures, and extemporised tourniquets have been used in attempts to blanch the limbs, or to waste them. An inexplicable chronic brawny œdema of a limb should always suggest caution in making a diagnosis. As more detailed instances the following may serve: A well-educated man was sentenced to a long term for a large fraud; on reception in prison he was reported to be acting strangely and to have drunk his urine and to have eaten his ordure; he was said to have a most melancholy manner and to be given to muttering constantly to himself. These symptoms all commenced directly he was convicted. Some marked inconsistencies were observed in his behaviour, and strong suspicions were aroused. The surgeon, upon first visiting him after these 'insane' manifestations, gave verbal orders in the man's hearing that 'as the patient had a preference for a special diet, and was so very insane, he should be so far humoured as to be allowed as much of that diet as he could himself produce, and any more from other sources he might ask for.' Beyond this he was apparently neglected. He did not again exhibit any dirty proclivity, and soon asked for the ordinary prison-diet, and offered to work properly.

A robust-looking man suffered from repeated convulsive seizures, accompanied, in each case, by the expulsion of urine. He was at first treated as a pronounced epileptic, but on being admitted to

a new hospital suspicion was aroused as to the genuineness of his case, especially as the man had no marks of past injuries on his limbs. In the hearing of the man the surgeon in charge described in detail to those around the bed certain grotesque and ridiculous phenomena which should follow after a definite interval if pressure were exerted on a special part of the vertex. The appropriate pressure was made, the seconds which followed were gravely counted by the watch, and at the appointed time the man exhibited the precise phenomena that had been described, ending by carrying out his conception of opisthotonos by travelling over the floor of the ward on his head and heels. He was put back to bed, and little notice was taken of him. No medicine and but little food arriving, the fits did not recur, nor did any unusual discharge of urine take place, the man leaving the hospital cured in about five days.

Possibly hypnotism might be used to dispel malingering in some cases, though, will-power being strong in clever impostors, they are unlikely to form good subjects for this influence.

A medical student insured simultaneously in several accident-companies, and after a short time sent in claims to all of them for compensation for a 'fractured leg.' He was visited by various medical officers, but it was not until a more than ordinarily determined surgeon slit open the plaster-of-Paris casing that the leg was found to be sound and to show no signs of fracture. The student had placed the casing on his own leg. A prisoner was admitted to hospital with a deep and profusely suppurating sinus extending for a distance of eight inches along the inner aspect of the lower leg. It was most offensive, and no treatment availed anything, until the surgeon found, deeply placed in the undermined tissues, a rough 'seton' formed of dirty lint. Proper precautions were taken and a plaster covering applied. The wound healed completely in a short time.

Though cured of one feigned malady, the perpetrators of these determined frauds generally attempt fresh kinds of imposture, though not always. A curious mimicry of left humeral dislocation and torticollis occurred in the writer's practice, where a man so deformed his deltoid by temporary contraction as to strongly suggest the lesion. But such power must be rare and peculiar.

Suicide is often feigned in prisons, and sometimes elsewhere. A notorious burglar of most violent and even homicidal habits was sentenced to a very long term; he commenced instantly to feign melancholia, and made what were considered pretended attempts to cut his throat with jagged mutton-bones; he also made a feigned attempt to hang himself with a rope made of his sheets. He was allowed to perceive that his behaviour was not regarded as indicative of insanity, but he then assumed a profound stuporous condition, refusing all food persistently until he began to emaciate, when he was fed by a tube with milk, arrowroot, and raw eggs. This went on for fourteen days; on the last day he struggled during the operation of feeding, and regurgitated a portion of the food into his pharynx, and then screaming and drawing a deep breath, some of the semi-liquid mixture passed into his bronchi; he had alarming symptoms and rapidly sank and died in three hours, with *post-mortem* signs of intense bronchial and pulmonary congestion. But the instant he had drawn the

material into his air-passages, he recognised his danger, and confessed that he had been all along feigning "in order to be transferred to an asylum as a lunatic;" he also rendered the surgeon and attendants every assistance in their efforts to save him. The last is an instance of the desperate lengths to which malingering is sometimes carried. A determined malingerer was detected in frauds so inimical to discipline that extreme measures of 'cure' were deemed necessary, and he was sentenced to the 'cat.' He received three strokes, and, as is not unusual from the shock, became pale; but to this condition was added an appearance of syncope. The surgeon arrested the flogging and examined the man, but being convinced that the syncope was assumed advised that the punishment should proceed. The man still lay limp and allowed his body to be passively shaken by the lash during two more strokes, presenting a ghastly appearance to the lay observers. At the third stroke after the recommencement of the punishment, he suddenly turned to the surgeon, and after calling him by some strong epithets bore the remainder of the flogging with a marked bravado. He never malingered again, but became a well-conducted cheerful man, and did not show any resentment towards the surgeon.

After falls and blows 'shock' is often complained of when no objective symptoms persist; and being considered a useful item in running up a claim after an accident, is often emphasised or assumed. It is therefore worth noting that the *facial expression* accompanying 'shock' is not easy to assume. Pallor, fibrillary muscular tremors of facial muscles, a lack of play in expression, sometimes alterations and oscillations of the pupils may be observed; and there is also frequently a loss of, or an exaggerated condition of, the deep reflexes. The hands tremble finely when held out horizontally, speech is usually slow, and a peculiarly plaintive sadness sometimes takes the place of the ordinary diversity of manner. Palpitation too is a very common symptom, as also undue emotional manifestations.

Sauvage has recorded the case of a young woman who feigned hæmatemesis by drinking bullock's blood in the slaughter-house *secretly*, and vomiting it *publicly*. Instances of hospital patients drinking the blood purloined from a venesection-vessel have occurred. The gums have been lacerated and the blood thus produced mixed with saliva and 'coughed' up to simulate hæmoptysis. Even children have soaked dyed rags in their urine so as to produce an alarming appearance of hæmaturia.

In actual suicide by hanging it is often most difficult to form an opinion as to the genuineness of the case, for it is quite certain that cases have occurred of suicide where the fatal issue was *not* intended. In prisons it is found that the feigned attempt is made at a moment when the warder's visit is expected, and usually the stool which supports the feigner is kicked away with an unnecessary noise which attracts attention, and there can be no doubt that many cases have occurred where the victim fully expected to be cut down and saved, thus preserving life and achieving a reputation for insanity at the same time; but emotional, passionate men will undoubtedly sometimes, in a sudden fit of uncontrollable temper, attempt suicide quite unpremeditatedly; hence it is hard to form a definite opinion on these fatal cases. The genuine and determined suicide, however, usually

destroys himself furtively, and chooses a time when he is unlikely to be disturbed. The intensely melancholy expression of the *true* would-be suicide, who has failed to carry out his terrible scheme, is usually very clearly indicative of the genuine attempt.

In the *detection of malingering* one great principle will best assist us. Induce a suspected impostor to overact his part, and watch keenly for inconsistencies. Preserve a completely impassive expression and manner, and when certain inconsistencies of signs and symptoms have been observed, conceal suspicions. Patience and judicious suggestion will generally be rewarded by such an overacted display as will corroborate our suspicions; although in some cases of feigned insanity the imposture has repeatedly baffled asylum-experts. Absolutely incompatible subjective statements of pain, and ridiculously inconsistent nervous, muscular, and mental exhibitions can generally be elicited in this way. Our next difficulty is to *cure* the malingered disease, and this is by far the greatest tax on our powers. We must now play upon the pride, the sense of shame, the honour and manhood of our 'patient.' A subtle scorn and irony of manner may do much; a distinct neglect of the case will do more; uncomfortably frequent tonic treatment by galvanism or faradism and by cold shower-baths, accompanied by low diet, suits many cases; and even painful tests guarded by a wise discretion in our own and the patient's interests may be needed. It is wise to induce the man to voluntarily relinquish his ill-conditioned actions rather than to proclaim our detection of his imposture; to let him perceive that his plan has failed; and even to allow him a golden bridge for retreat, rather than to signalise our victory by retributive punishment, or by disgracing him by public proclamation of his misdoings. To establish his *future* good conduct we need *his own will-power* to aid our treatment. But each individual case must be treated throughout on a plan suited to its special features. No stereotyped method will avail, but original and novel devices through their unexpectedness will generally carry success. In a word, a scientific knowledge of the symptoms and features of real disease is of course necessary in dealing with malingerers; but of still more import in detecting and curing them is a *very close habit of observation, never relaxed*; an ever-varying resourcefulness of device, and a large modicum of moral courage which will embolden the practitioner to form an opinion and hold by it in the face of great personal responsibility. The glory attached to the successful management of these cases is not great, and the strain upon the doctor is often very severe. Coincident and real disease or accident may arise and cast suspicion on the correctness of an absolutely true diagnosis; thus a ridiculous mental impostor may die of a deep-seated unsuspected aneurysm; a sham suicide may hang himself by mistaken calculation of his chosen time, or by mishap and so forth. It is well in the conduct of difficult cases to obtain the added protection of a second medical opinion. Above all, having found malingering we should look carefully for real disease, as the two are often co-existent. And when our carefully formed diagnosis is doubtful, we should always give the patient the benefit of the doubt. Finally, having detected and cured our malingerer, we must use great discretion in deciding when to reveal and when to be reticent about the facts of the case.

The actual revelation of our positive conviction, when called for, will often be most wisely made through a trusted friend of the detected impostor.

TENNYSON PATMORE.

FEIGNED ERUPTIONS.—See DERMATITIS VENERATA.

FESTER.—A superficial suppuration, resulting from inoculation of the skin with pyogenic organisms. See ABSCESS.

FEVER.—Fever is a general disturbance of metabolism, giving rise to several symptoms, the most striking of which is a rise in the temperature of the body. The other chief symptoms are loss of appetite, thirst, and disturbance of digestion and absorption, increase in the frequency of the pulse and respiration, diminution of secretions, nervous disturbances (e.g. headache, excitement), muscular disturbances (e.g. weakness), and general interference with the nutritive processes causing degenerations and wasting. These symptoms are partly due to the rise in temperature if high, but mainly to the toxic agent causing the fever.

The normal temperature.—See TEMPERATURE.

Fever or pyrexia.—Fever may be called slight if it does not exceed about 101° F., moderate about 103° F., severe if it exceeds these limits. If it reaches or exceeds 107.6° F. it is called hyperpyrexia. The temperatures compatible with life are generally placed between 93.2° and 107.6° F. Recovery from still higher temperatures has been observed. Recovery has occurred after a temperature of 113.9° in pneumonia, and in malaria after one of 114.8° . Teale records recovery in a case of severe internal injury after a temperature of over 122° had been more than once reached. High temperatures from infectious causes are more dangerous than those from non-infectious causes. Generally speaking recovery does not occur when high temperatures of 107° and 108° and upwards have lasted long. Fever generally shows diurnal variations somewhat similar to those of health, but not so regular; the temperature generally mounting in the evening to fall through the night, rising again towards morning to attain its maximum anew in the evening. A typical fever runs a more or less characteristic course naturally divisible into three stages: 1. The initial stage during which the temperature continues to rise (always preserving its diurnal variations) either rapidly or slowly. If the rise be rapid and accompanied by marked contraction of the cutaneous blood-vessels, a sensation of cold and a shivering-fit or *rigor* are likely to follow. During this stage the surface-temperature falls, and the rigor is a reflex induced by the cold, while the internal temperature rises. 2. It is succeeded by the *fastigium* or acme, during which the temperature, subject to the diurnal variations and occasional accidental fluctuations, is maintained at its highest point. It may last for days or even two or three weeks. The skin generally feels hot and dry, but its temperature is usually only a little greater than in health. The increased internal temperature does not usually produce sensible sweat. 3. The defervescence follows. It is sometimes short, when the fever is said to end by *crisis*, and sometimes protracted, when it is said to end by *lysis*. The temperature returns to normal, quickly and suddenly in the former, sometimes falling several

degrees within an hour, and gradually in the latter. This is associated with changes in the skin more favourable to the discharge of heat. The temperature of the skin rises, and a more or less abundant sweat-secretion occurs. There may be also free diuresis or diarrhoea. The increased action of the emunctories is often spoken of as a 'critical evacuation.' It frees the organism from waste and other products of the disturbed metabolism, bacterial toxins, &c.

Nutritional changes in Fever.—The metabolism of the body is profoundly disturbed chiefly through the action of the toxin which causes the fever. The mere increase of temperature (hyperthermia) has not much influence unless it reach a considerable degree. It may even be absent as in cholera, where great intestinal disturbances, similar to those of some fevers, may occur without any rise of temperature. The nutritional changes seen in fever may be thus arranged:—

1. *Changes in the urine.*—Passing over the ordinary well-known changes, there is an increase in urea, extractives, and phosphoric acid, and a diminution in chlorides. The nitrogen introduced through the food is reduced to a minimum, and yet the kidneys excrete much more (often more than twice as much) than they do on a similar diet in health. However much the increase is influenced by the rise of temperature, it is not caused by it. It begins before it, as observed in the hours preceding an attack of intermittent fever, and in the latent period of typhoid fever. Albumens, peptones, and albumoses may also appear in the urine.

2. *Changes in the respiration.*—The respiratory exchange is not materially altered in fever, the respiratory quotient $\frac{\text{CO}_2}{\text{O}}$ remaining the same.

3. *Disorders of secretion.*—All the secretions except the sweat are diminished, and even it is greatly disturbed. The saliva is scanty; the gastric juice is less abundant and has less digestive power, the hydrochloric acid failing and later the pepsin also; the bile is suppressed, and the intestinal secretions diminished.

4. *Degenerative tissue-changes.*—(a) The blood is generally more fluid and coagulates more slowly. Its albumen and respiratory capacity are diminished. Its urea and extractives are increased, the former often twice and the latter eight times. The red blood-corpuscles are diminished, and there is sometimes an abundant leucocytosis, due however to the cause of the fever and in no way dependent on the fever itself. (b) The muscles lose glycogen and weight, and may degenerate. (c) The amount of fat diminishes. (d) The kidneys are congested and may degenerate. They allow albumen to filter through. (e) The liver loses glycogen, and bile-secretion is diminished or suppressed. Functional troubles arise, e.g. ammonia is not transformed into urea, which is 40 times less toxic. Degenerations occur. (f) The heart suffers like the muscles. It loses energy, and other changes occur. (g) The body loses weight. This varies in amount with the cause of the fever, with the treatment, and with the diet. It sometimes begins early and increases until defervescence sets in, sometimes late as in typhoid fever, being scarcely noticeable before convalescence.

Production of fever.—The muscular and glandular degenerative changes are to be looked upon as chiefly due to the toxins circulating in the blood. Simple hyperthermia if pronounced can cause

granular and fatty changes, but not others. Destruction of tissue is obviously going on. The loss of nitrogen shows that albumen is being used up, not the free albumen but the fixed organic albumen of the tissues. The needs of the body compel it to feed upon its own proteids and fats, the nitrogen being discharged by the kidneys, and the carbon by the lungs. The process closely resembles that which occurs in starvation, and probably runs parallel with it at first, but later, owing to the continuance of the high temperature and defective nutrition, it may become exaggerated, finding the tissues now less stable and less able to resist the demands made upon them. This generates increased production of heat. Heat-production in health tends to exceed rather than fall short of the needs of the organism. This excess becomes more marked in fever, and if there be no increased loss, the temperature must rise. It is obvious that the temperature may rise either through (1) increased heat-production, (2) diminished loss, or (3) both combined. The first is under the control of the cerebral thermogenic centres, the second under that of a system of vaso-motor and secretory nerves connected with centres in the medulla. In health when an increase of heat occurs from any cause, the second mechanism at once comes into play, and by an increased loss maintains the equilibrium of temperature. In fever, on the other hand, when a similar increase occurs through the action of the thermogenic centres or otherwise, the nerves and centres which preside over, it may be, both mechanisms, but certainly over the second, become capable of only partially responding to abnormal impressions, and hence are no longer able to discharge their function efficiently. Thus in the initial stage of a fever which begins quickly, the skin often becomes pale from spasm of the cutaneous vessels, although even before this pallor is seen the internal temperature has been rising, as evidenced by the rectal temperature and the increased excretion of urea and carbon dioxide. The temperature continues to rise, partly owing to the increased production of heat, and partly owing to diminished loss from the skin causing hotter blood to return to the interior. The difference between the external and internal temperatures sometimes reaches even 18°F. The regulative mechanism fails to act, or acts only very imperfectly. The sensation of cold upon the skin reflexly stimulates the sensory nerves, and this, along with the hot internal blood stimulating the muscular centres, causes the rigor. The muscular spasms constituting the rigor produce heat which warms the skin, and the rigor ceases. It may easily be provoked anew by again exposing the skin to cold. The nature of the poison which starts the fever has much to do with the cutaneous vascular spasm, as it is more marked in some diseases, e.g. malaria, than others. After a time, generally when the acme is reached, the increased internal temperature acts upon the vaso-dilatator centres, causing a dilatation of cutaneous vessels and hence increased loss; but owing to its failure to act on the sweat centres, this increase is not great. Its effect is often seen by the falls which follow upon its being temporarily put in action. The continuance of the increased heat-production is due to the primary causal toxin circulating in the blood and affecting both the thermogenic centres and the metabolism of the tissues. When this toxic action ceases, the increased heat-production ends, the mechanism regulating loss becomes re-established, and the fever ceases.

Causation of fever.—All substances capable of acting on the heat-producing or regulating mechanism may cause fever. Their action will vary with the dose, mode of administration, and the subject. They may be divided into 3 groups, the inorganic, the organic, and the microbic. The first group has few members, e.g. the intra-venous injection of distilled water and the exhibition of phosphorus. The second group has many members, e.g. strychnine, eserine, cocaine, caffeine. If blood or serum or fibrin-ferment be injected into the circulation, fever results. Extracts of organs and the urine, particularly night-urine, have a similar effect. These may be called hetero-intoxications. In chlorosis there is often fever which can be explained by the gradual destruction of the red blood-cells. The same thing is seen to follow their rapid destruction in paroxysmal hæmatinuria. Again, severe fractures and contusions are sometimes followed by fever, caused by the absorption of the blood effused or of the debris of cells, for it is possible that all cells contain pyrogenetic substances. These are instances of auto-intoxication. Again, some of the albuminous products of digestion, e.g. albumose, especially deuto-albumose, have much pyrogenetic power; but the most frequent causes of fever are undoubtedly the toxins produced by bacteria. We need not stay to enquire if they are in any way similar to these digestive products, but it may be mentioned that a deuto-albumose has been extracted from tuberculin upon which its pyrogenetic property largely depends. Bacterial toxins differ from purely chemical toxins in generally showing an incubation-period and in acting usually in infinitesimally small doses.

Clinical types of fever.—(1) Continuous, in which the fever exists for some time at a fixed level. (2) Remittent, in which successive rises are followed by falls which do not reach the normal. (3) Intermittent, in which the falls do reach the normal. (4) Relapsing, in which the apyrexial periods are more prolonged. (5) Specific, due to the action of a special germ. These terms are not of much use. There are still other types which might be mentioned, such as hysterical and urethral fever, and they probably belong to the category of simple hyperthermias rather than to true fevers in which the pyrexia is caused by some poison circulating in the blood.

The rôle of the increased temperature and nutritive changes in fever.—The rise of temperature is the expression of the reaction of the organism to the cause, and is produced by its exerting its powers of defence against that cause. If excessive it is hurtful, as all reactions may prove, however good they may be in themselves. Severe simple hyperthermia is dangerous, and severe toxic hyperthermia is still more so; but slight or moderate pyrexia is not so. It increases phagocytosis and the bactericidal powers of the serum, and hence favours the destruction of microbes and the elimination of their toxins. Moderate pyrexia is therefore a good thing. Needless to say, no hard line can be drawn. Individual peculiarities must be studied, for what is moderate in one may be severe in another. The amount of general disturbance may be taken as a guide, but experience will prove the best guide of all. The nutrition is modified not only during the disease, but for a long time afterwards. Weak infants often become vigorous after febrile states, and increased nutritive power is often seen after typhoid fever.

The fundamental rôle of fever is then probably a useful and protective one.

The treatment of fever.—The number of therapeutical agents against fever at present before the profession is very large. They may be divided into two great groups :—I. Those which act upon the agents causing the fever. II. Those which act simply on the temperature. The first alone contains true remedies against fever. Unfortunately it includes but a small number, practically three : namely (1) quinine in malaria, (2) salicylic acid in acute rheumatism, and (3) the antitoxins of which the best known and most valuable up to the present is that of diphtheria. There are both hope and prospect that this rational class of remedies will be extended in the future. The second great group combats the temperature merely, and should generally be employed only when it has become severe. Moderate fevers do not scientifically require treatment unless the first group of remedies is applicable, for beneficial processes should not be checked. Among the best known members of the second group of remedies are antipyrine, phenacetin, antifebrin, and thallin (*see* ANTIPYRETICS). They all act in the same way, and lower the temperature by increasing cutaneous loss, both through radiation and sweat-secretion. Quinine and salicylic acid have a similar action, so have all quickly diffusible stimulants such as alcohol and ether, as well as all lotions, douches, cold spongings, the wet pack, rubbing the skin with ice, and baths. The cold bath is at once the most characteristic and powerful of the whole group. It is the method *par excellence* of combating the hyperpyrexia of rheumatic fever, and either it or some of its modifications may prove very valuable in many cases of pyrexia. The cold bath has proved of great service also in typhoid fever. It acts by first constricting the cutaneous blood-vessels and increasing the internal temperature; then follows reaction,—the cutaneous blood-vessels dilate, sweating occurs, and the internal temperature falls. Its effects may be continued by the subsequent administration of quinine, antipyrine, or any others of the antithermic remedies above mentioned. *See* TEMPERATURE; THERMOMETER; ANTIPYRETICS; COLD, Therapeutic uses of; FEBRIFUGES.

R. F. C. LEITH.

FIBRILLATION, Muscular. — A localised quivering or flickering of muscular fibres. *See* MOTILITY, Disorders of.

FIBRINOUS CONCRETION. — *See* CONCRETIONS.

FIBROID DEGENERATION. — A morbid change which consists in the substitution of a tissue somewhat resembling fibrous tissue for other structural elements; some pathologists consider this change to be of the nature of a degeneration. *See* DEGENERATION; and FIBROSIS.

FIBROID PHTHISIS.—A name given to certain cases of phthisis in which a considerable development of fibrous tissue occurs in the lungs. *See* PHTHISIS.

FIBROMA.—A form of tumour composed of fibrous tissue. *See* TUMOURS.

FIBROSIS. — *SYNON.* : Sclerosis, Cirrhosis, Chronic Interstitial Inflammation.

DEFINITION.—Until within the last few years the presence of an increased amount of fibrous connective

tissue within the different organs of the body when not referred to as a chronic ‘-itis’ has been described either as a ‘sclerosis’ or yet more commonly as a ‘cirrhosis.’ Of these latter terms, both are oblique and therefore unsatisfactory. Tissues the seat of increased development of connective tissue are, it is true, in general of a firmer consistence, but hardening, or *sclerosis* (σκληρός, hard), may be due to other causes. So too the great laying down of fibrous tissue in the liver may be associated with the development of a characteristic bright yellow appearance, or *cirrhosis*, of that organ (κίρρως, yellow); but a similar yellowing rarely accompanies the increased interstitial deposit of fibrous tissue in other organs. With regard to the term *chronic interstitial inflammation*, while in the majority of cases we recognise that the undue development of fibrous tissue is brought about by the reaction to irritation, we cannot be certain that such inflammation is the one sole efficient cause in every case; there are cases in which we must assume some other cause. Hence, even if it be open to the charge of being mongrel, the word *Fibrosis* recommends itself as the most direct, unassuming, and self-explanatory term to indicate and include all those conditions in which there is a relative increase in the amount of white fibrous connective-tissue in any organ or region of the body.

ANATOMICAL CHARACTERS.—Fibrosis may be either *localised* or *generalised*, either confined to one small portion of an organ or affecting an organ or tissue throughout its extent; it may take the place of cells of higher order, and, doing this, lead to no increase in the bulk of a tissue (*substitution- or replacement-fibrosis*), or there may be growth of connective tissue in excess so that the bulk of a tissue or organ is increased (*productive fibrosis*). It is not always easy to distinguish between these two forms, for, more especially where the new connective tissue is developed in consequence of frank inflammatory disturbances, that tissue as it grows old undergoes contraction, so that a productive fibrosis which in its early stages leads to increase in the size of a part may in later stages, with this contraction, bring about an actual shrinkage of an organ. In all cases, however, we recognise the presence of an increased amount of connective tissue in the affected part.

With reference to the origin of this connective tissue it may be stated as the general opinion, arrived at after many years of observation and controversy, that the new-formed fibrous tissue is developed from the fixed tissue-cells of the region in which it shows itself. There are, it is true, well-attested observations upon certain lower vertebrates which appear to prove clearly that in them, at times, wandering cells can come to rest in inflamed areas and gradually assume the characters of fibroblasts, or young connective-tissue cells, but this transformation has not been proved as yet in man or the higher vertebrates. On the contrary the appearance of nuclear division in the fixed cells at the boundary of inflamed areas, and the fact that new tissue is *always* formed in immediate connection with pre-existing tissue, prove clearly that it is from this pre-existing tissue and not from wandering cells, or leucocytes, that the new tissue in the main develops. But at the same time we are forced to recognise the existence of *Metaplasia*, i.e. the transformation, within certain limits, of one form of tissue into another. Muscle, tendon, yellow elastic-tissue, and the endothelium of vessels

and lymphatics are all, like white fibrous connective tissue, of mesoblastic origin; and evidence is accumulating that all of these may under abnormal conditions give rise to this last lowest and simplest form. If, for example in what appears to be the earliest stage of cirrhosis of the liver, we find an abundant accumulation of small round cells along the portal sheaths, and if in later stages these have given place to connective tissue, we must not conclude that there has been direct transformation from the one into the other. Experimental studies upon other regions have proved that these small round cells are in the main leucocytes which have been attracted to the part during the earlier stages of the inflammatory process; these at a later stage disappear, by migration, by breaking down, or by ingestion by the growing fibroblasts of the region. The vessels and framework of the liver are of mesoblastic origin, and it is from the mesoblastic cells of the organ, not from migrated leucocytes, and not from the hypoblastic glandular cells of the organ, that the new cirrhotic tissue originates. There is still much to be learnt concerning the new formation and the regeneration of tissues, and it is quite possible that within the next few years our views concerning these matters may be materially widened. The above represents what can be advanced with safety at the present time.

The stages of the development of this new fibrous tissue are those seen in granulation-tissue in general. The cells, at first round or oval with deeply staining nuclei and relatively abundant protoplasm, become oat-shaped, then spindle-shaped, the nucleus assuming a similar elongated form; terminal fine processes are given off, forming fibres, while lateral fibres are elaborated and separated off from the body of the cell. As these changes occur the protoplasm and the nucleus become more and more inconspicuous, until at last all that can be made out is a flattened elongated cell of extreme tenuity lying in a bundle of wavy fibrils. This process of fibril-formation under the governance of the nucleated cells may well be compared with the elaboration of the contractile substance of the muscle-fibre under the governance of the nucleus of the muscle-cell. It must, however, be acknowledged that these views concerning the development of connective-tissue fibres are not, as yet, beyond dispute. Some hold them to be of intercellular origin due to a form of coagulation of the matrix or ground-substance.

In not every case of fibrosis do we have this full development of white fibrous connective tissue; sometimes, as in certain forms of cirrhosis of the liver, the new tissue tends to be reticulated, or, as Councilman and those working with him have pointed out recently, there may be a relatively considerable development of elastic tissue. Further researches are necessary to show the relationship between the form of connective tissue which develops in certain cases and the tendency or lack of tendency to undergo contracture, as again the relationship between the newly developed tissue and the vessels.

In all cases of inflammatory fibrosis there is, in the earlier stages at least, what for this low form of tissue is a relatively abundant vascular supply: where contracture occurs, the vessels become narrowed and the blood-supply of the region materially diminished. This diminution, as well as the contraction of the new tissue and the pressure thereby induced, tells upon the higher cells of the tissue and leads to their atrophy. As they atrophy, their place is in part

taken by new fibrous tissue. There is thus a marked tendency, for interstitial fibroses especially, to assume a progressive and vicious character, to extend even after the original cause ceases to be in evidence.

ETIOLOGY.—In the majority of cases fibrosis would seem to be the result of a chronic inflammatory process—of a reaction to persistent if slight irritation, whether mechanical or bacterial in nature. But, as already indicated, we cannot in all cases recognise that we have to deal with the results of inflammation; stimuli so slight as to be within physiological limits, stimuli which can scarce be regarded as irritants or noxæ, may, it would seem, lead to fibrosis. The following forms of fibrosis may be distinguished according to their causation.

1. Inflammatory Fibroses.—**A. PRODUCTIVE.**—**1. Localised.** (a) Focal, e.g. the focal area of new connective-tissue growth induced by the presence of such micro-organisms as produce the more chronic forms of infectious granulomata (tubercles, gummata, &c.). (b) Capsular, forming around inert dead or foreign material, e.g. around old abscesses, necrotic areas, impacted bullets, inhaled particles of coal or other matter (pneumoconioses). In all these cases we clearly recognise the results of continued irritation of a low grade.

2. Serous.—The productive fibroses affecting serous surfaces secondary to inflammations, and whether localised or generalised tending to the development of organised adhesions.

3. Interstitial, Generalised.—Good examples of this form are the interstitial pneumonia following upon chronic pleurisy, and Hanot's (hypertrophic) cirrhosis of the liver (which the majority of observers regard as of infectious origin). The diffuse 'cirrhotic' changes in the liver, lungs, and kidneys secondary to, or accompanying, infectious granulomatous changes in these organs, as again those seen in the lungs in advanced cases of anthracosis &c., may also be here included. But, as already stated, while these conditions may be regarded as primarily of the productive type, there is, more especially in the later stages, a marked tendency to coincident replacement-fibrosis, the contraction of the new connective tissue leading to atrophy of the functional or nobler cells, these becoming replaced by fibrous tissue. And indeed, while in such cases the fibrosis is primarily of the productive type and the organs undergo primary enlargement, it is a matter of controversy at the present time whether conditions such as ordinary progressive cirrhosis of the liver originate as the result of a primary inflammation, with atrophy, of the parenchymatous elements of the organ, or of a primary perivascular inflammation affecting, for instance, the portal sheaths, the parenchyma becoming secondarily involved. Taking everything into consideration, it would appear correct to classify the important conditions of atrophic cirrhosis of the liver, chronic parenchymatous nephritis, and chronic indurative pancreatitis, as:

B. MIXED FIBROSES, in which the new tissue formation is in part a replacement, in part an overgrowth.

C. REPLACEMENT-FIBROSES.—**1. Cicatricial.**—Where there is breach of continuity with destruction of a certain number of cells, in the absence of infective agents, the new reparative connective tissue maintains a certain relationship to the amount of previous destruction, i.e. does not exceed it: there is replacement by tissue of a lower order.

2. Post-necrotic.—(i.) The same is true in cases

of complete and sudden necrosis of all the elements of a tissue, e.g. in the healing of a simple infarct.

(ii.) An identical process of fibroid substitution followed by contraction is seen in the organisation of non-suppurative thrombi: the 'dead' blood is replaced by connective tissue.

3. *Post-atrophic or Necrobiotic*.—Where there is gradual death of tissue-elements, from malnutrition or from loss of function, there is a similar replacement-fibrosis. This is well seen in secondary sclerosis of various tracts in the spinal cord, and here the exact substitution of fibrous tissue for white matter is peculiarly well marked. It is seen also in the interstitial changes which result from arterio-sclerosis. Chronic interstitial myocarditis and the so-called interstitial nephritis of arterio-sclerosis are mainly replacement-fibroses, secondary to malnutrition and atrophy of the higher elements of the affected tissues: i.e. the changes in these higher elements are primarily degenerative, the fibrosis (which as tending to be reparative may rightly be regarded as of inflammatory origin) is a secondary phenomenon.

In addition to these we have to recognise two other orders of fibrosis, which may be termed the non-inflammatory or functional, and the neoplastic.

II. *Non-inflammatory or Functional Fibroses*.—Of these the best marked examples are to be seen in cases of *lymphatic obstruction* followed by elephantiasis. The later stages of myxoedema with the resultant subcutaneous fibrous hyperplasia may possibly belong to the same category. In either case the overgrowth of the connective tissue cannot be regarded as inflammatory in origin; it is not in any sense a reaction to injury. Nor again can it be regarded as purely due to increased nutrition, due to increased bathing of the parts with lymph, for so far as experiments can teach, we find that increased nutrition alone does not lead to tissue-overgrowth. But by the increased amount of lymph in the affected parts undoubtedly the connective tissues are subjected to an increased strain; and it may well be that, just as with muscular activity there is increased growth of so dense a tissue as bone, particularly at the regions of muscular origin and insertion, so, here, increased pull upon or distensible force acting upon the connective tissues, when associated with adequate nutrition, acts as the effective cause in inducing hyperplasia of these tissues. The fibrosis (rarely extreme) which in the liver and elsewhere accompanies moderate grades of *venous congestion* (not being seen in extreme grades) is apparently of the same class, as possibly is the thickening of the *arterial intima* in ordinary arterio-sclerosis. In this last disease it is now generally admitted that the primary disturbance is a local weakening of the middle coat (see *ARTERIES, Diseases of*). As a result of the weakening and of the pressure within the artery, the wall tends to bulge outwards; in other words, with the bulging a strain is exerted upon the endothelium and intima in general, and now follows a proliferation of the intima with consequent localised fibroid thickening, which differs from ordinary granulation-tissue in being originally devoid of blood-vessels.

III. *Neoplastic Fibroses*.—*Fibromata*.—As these tumours are localised fibroses they have to be included in our consideration of this subject. The boundary-line between these and the products of inflammation is very obscure, or more truly would not seem to exist. Certain forms of localised fibromata, as, for instance, the true *cheloids*, owe their origin

to irritation or injury. We must admit that irritation is not here the only factor. Associated with it there is what, in the absence of fuller knowledge, we must describe as an idiosyncrasy on the part of the connective tissues of certain individuals to proliferate beyond physiological limits. In other tumours, the true *fibromata*, we have not as yet recognised the existence of a primary noxa. They may originate from simple connective tissue, or, as a study of uterine fibromata and fibromyomata seems clearly to indicate, certain forms originate from modified mesoblastic cells of higher order, which in the course of excessive proliferation revert to a simpler type.

J. G. ADAMI.

FICUS UNGUIUM (*figus*, a fig; *unguis*, a nail).

A disease of the posterior wall of the nail. See *NAILS, Diseases of*.

FIDERIS, in Switzerland.—Chalybeate waters. See *MINERAL WATERS*.

FIFTH NERVE, Diseases of.—The fifth or trifacial nerve (*nervus trigeminus*), the largest of the cranial nerves, arises by three roots: (a) an ascending sensory root whose fibres can be traced as far down as the upper cervical region; (b) a motor root situated in the pons Varolii, and (c) a descending root, arising from cells in the Sylvian grey matter at the level of the anterior corpora quadrigemina, which also contains motor fibres. The nerve reaches the surface of the brain by passing through the middle cerebellar peduncle, and there consists of a sensory and motor portion, the sensory fibres passing through the Gasserian ganglion, and being distributed to the face and a portion of the head. The motor portion, much the smaller, is physiologically independent of the ganglion, and supplies the pterygoid, masseter, buccinator, temporal and probably the tensor tympani, tensor palati, anterior belly of digastric, and mylohyoid muscles. The two first divisions—the ophthalmic and superior maxillary—are entirely sensory, and proceed from the ganglion. The third or inferior maxillary division proceeds also from the ganglion, and is joined by the motor portion after its emergence. Lesions of this nerve cause disorders of sensation, motion, and nutrition or secretion, according to the anatomical position and extent of the injury.

The affections of the fifth nerve may be considered in the following order:—

1. **Trifacial Neuralgia**.—Neuralgia may affect one or all of the three divisions of the nerve. It is fully described under the head of *tic-douloureux*. See *TIC-DOULOUREUX*.

2. **Trifacial Anaesthesia**.—Anaesthesia of the trigeminus, usually unilateral, may be dependent either upon (a) *central lesion* or upon (b) *peripheral lesion*.

(a) *Central lesion*.—Hemiplegia from hæmorrhage, softening from thrombosis, tumour, or other coarse affection of the intracranial nervous system, is usually accompanied by some anaesthesia in the district supplied by the trigeminus, arising probably from interference with the integrity of the fibres of origin of the nerve in their central course. The anaesthesia usually occurs on the same side of the body as the paralysis of motion, and therefore opposite to the seat of lesion. This is always the case when the lesion occupies a position above the pons Varolii. In disease of the pons Varolii, however, the loss of sensibility may involve both halves of the face,

although it usually affects the same side as that upon which the limbs are paralysed, and opposite that upon which the seventh and sixth nerves (when either or both of them are involved) are affected. In cases of apoplexy, the anæsthesia is usually very imperfect, and not sharply defined. It is short-lived, lasting from a few hours to days; but in certain cases it may continue, and even outlive the motor paralysis with which it is conjoined.

Intracranial tumours may produce more persistent anæsthesia, either by immediate destruction of sensory fibres or, indirectly, by the cerebral enlargement, due to their growth, causing compression of the fifth nerve as it traverses the floor of the skull. Anæsthesia in the district of the fifth nerve is sometimes noticed in cases of *tabes dorsalis*. In such circumstances it is usually bilateral and probably of nuclear origin.

(b) *Peripheral lesion*.—Anæsthesia dependent upon lesion of the trigeminus in its peripheral course is a symptom of serious moment, which it is important to distinguish from that of central origin; and this may be accomplished by noting the following points:—The degree of peripheral anæsthesia far exceeds that which obtains in cases owing their origin to a central cause. It is much more complete, and may involve, which the latter does not, trophic and vaso-motor complications. Its extent varies according as the trunk of the nerve, including the Gasserian ganglion, is involved; or only one or two of its branches. Should the main trunk be affected, there is more or less complete anæsthesia of one side of the face and part of the ear, conjunctiva, cornea, nostril, mouth, half the tongue, the gums of the same side, and a part of the palate. If the conjunctiva be touched with the finger, there is no reflex contraction of the eyelids. A glass from which the patient drinks seems to him as though it were broken, for he feels the material on the sound side and not on the affected side. The skin of the face is cool, and may be somewhat oedematous, and purplish in tint. After a few days, if the cause persists, and if care be not taken to prevent injury to cornea and conjunctiva, the eye on that side looks dry, glazed, and congested; the cornea becomes cloudy, and in time sloughs and perforates, the contents of the eyeball escaping to a varying extent, so that the organ is destroyed. There is dryness of the nostril on the affected side, and irritant substances applied to it fail to produce sneezing. Atrophy of the mucous membrane may in course of time lead to loss of smell on the affected side. Taste is lost on that side of the tongue, except sometimes at the base, which is supplied by fibres which run in the glosso-pharyngeal nerve. The salivary secretion is diminished. In time there may be bleeding from the gums and ulceration of the mucous membrane. Should the lesion exist upon one of the three divisions of the trigeminus, the anæsthesia will be found limited to the district supplied by that division.

The nature of the lesion must be determined by the examination of concomitant conditions. It may consist either in caries of the bone, neoplasm, aneurysm, or meningitis, acute or chronic. Whatever be the active cause by which the nerve is damaged, the effects will be the same: pressure upon, and disorganisation of the nerve-fibres will result in the disorders described—sensory, motor, trophic, and vaso-motor. In such circumstances, one or more of the other cranial nerves are usually affected coincidentally. In tubercular meningitis, the

fifth nerve is shown to be paralysed (along with others traversing the floor of the skull) by the conjunctivitis and keratitis so often present in advanced stages of the disease.

TREATMENT.—Syphilitic gummata on the floor of the skull, developed either in the membranes of the brain or in the nerve itself, are so frequently the cause of the disorganisation of the fifth nerve which gives rise to anæsthesia, that in all cases it is right—unless some other explanation is evident beyond all doubt—to bear in mind the possibility of such a cause, and to prescribe accordingly without delay. From 10 to 20 grains, or more, of iodide of potassium should be administered every four hours. Should there be a gumma pressing upon the trunk of the nerve, this treatment will probably have the effect of bringing about a rapid amelioration, and, in many cases, supposing it has been applied early enough, a complete cure. It is evident that, as regards other causes, there is no particular indication for treatment, which must be adapted to the special circumstances of the case.

3. **Trifacial Hyperalgesia.**—Hyperalgesia may accompany or precede neuralgia of the fifth nerve. It may also precede facial anæsthesia when this is due to neuritis. There are varieties in the degree of this hyperalgesia. It is sometimes so severe that the slightest touch occasions pain. The face cannot then be washed in the ordinary way, but the patient has to take a piece of sponge or wetted rag and cautiously dab the skin with it. Sometimes it is described as a feeling of soreness only when the hand is passed over the face. In either case the condition is accompanied by diminution of the tactile discrimination in the part. In *mimetic spasm* of the seventh nerve, there is often hyperalgesia in the region of one or more divisions of the fifth, and the lesion is then doubtless connected with the deep origin of this nerve. In blepharospasm it will often be found, if the face be carefully examined, that pressure with the finger at some point will check the spasm. Subcutaneous division of a twig of the fifth (or afferent nerve) at this point will often bring about a cure of the affection. The supra-orbital or subcutaneous malar are the nerves most commonly in fault.

Photophobia is referable to hyperalgesia of the branches distributed to the conjunctiva.

4. **Motor Disorders.**—Affections of the motor root of the fifth nerve are either (a) of a *spasmodic*; or (b) of a *paralytic* character.

(a) *Spasm*.—Spasm of the muscles supplied by the trigeminal nerve may be tonic or clonic. In trismus, or 'locked jaw,' the teeth are clenched together by the tonic contraction of the masticatory muscles, which can be felt tense to the touch. According as the muscles are involved generally, or only partially, the lower jaw will be fixed in a symmetrical position, or be pulled over to one side, or advanced or retracted. Clonic spasm of the same muscles is observed in various convulsive disorders; and slower movements of a horizontal character constitute the grinding of teeth sometimes indicative of cerebral disease.

Trismus may either be one symptom of tetanus (see TETANUS), or it may occur by itself, and then it either arises from cold or is of reflex origin, from irritation of the sensory portion of the nerve by decayed teeth, dentition, or disease of the jaw-bone. When its occurrence is epidemic, it is doubtless of infective origin. It may be due to the presence of

a foreign body, possibly of very small size, lodged in the cicatrix of a wound upon the face, or even in some distant part of the body. Irritation from worms is a possible cause. It is still more commonly hysterical. It is possible, though not proved, that it may be due to a lesion of the cerebral cortex.

TREATMENT. W—hen the condition arises from cold, the constant current should be applied to the contracted muscles. Any source of irritation, whether from decayed teeth, or otherwise, must be sought for, and, if possible, removed or remedied.

The removal of a foreign body will sometimes bring about an immediate cure. This failing, the hypodermic injection of morphine, in doses of gr. $\frac{1}{6}$, may be employed, and bromide of potassium given internally in doses of 20 grains. When the presence of worms is suspected, appropriate treatment must be employed. If the affection be hysterical, somewhat powerful faradic currents, directed to the muscles of the jaw, will scarcely ever fail to open the mouth and cure the ailment. Hysterical trismus cannot be mistaken for dislocation of the jaw, if it be remembered that in the latter accident the jaw is fixed with the mouth partly open.

(b) *Paralysis*.—Paralysis of the masticatory muscles is not very common, but may be observed sometimes in cases of bulbar paralysis, or it may accompany anesthesia of the face, and depend upon tumour, abscess, aneurysm, or some such coarse disease encroaching upon the trunk of the nerve within the cranium. To test the state of the muscles, the patient should be asked to move his jaw to and fro laterally, as well as in opening and shutting the mouth. Any irregularity of movement will be evident to the eye, and defective strength or absence of contraction in the affected muscles may be felt by placing a hand on each cheek, while the patient performs movements of mastication. In unilateral palsy on opening the mouth the point of the jaw is deviated towards the paralysed side. The affection is more often unilateral than bilateral. Its importance is bound up with that of the lesion which gives rise to it. There is a somewhat rare affection in which contraction of the masseters, temporals, and pterygoids is observed to be very feeble, and the jaw will drop from inability of the muscles to support it, not as a result of true paralysis, but rather from a rapid exhaustibility of the muscular system, so that the act of eating quickly occasions the appearance of this symptom. See MYASTHENIA GRAVIS.

As in peripheral affections of the sensory portion of the nerve, especial attention should be paid to the causation, and if this appear to depend upon a new-growth, the probability of its syphilitic character should be borne in mind, and iodide of potassium administered in doses of from 10 to 20 grains, or more. If these measures fail, the question of surgical operation may have to be entertained. See NEURALGIA; TIC-DOULOUREUX; and NERVOUS SYSTEM, Physical Examination of.

T. BUZZARD.

FILARIA (*filum*, a thread).—A genus of nematode worms, not very clearly defined, which contains a variety of thread-like parasites whose bodies are of uniform thickness throughout, and at least fifty times longer than broad. Under this head are often included several human parasites, such as the *Dracunculus*, or Guinea-worm (*Filaria medinensis*), the lung-strongylus (*F. bronchialis*), *F. vol-*

vulus, *F. loa*, *F. Bancrofti*, in addition to a variety of larval or sexually immature nematodes, whose genetic relations are only very imperfectly understood. In the latter category may be placed Bristowe and Rainey's entozoon (*F. trachealis*), and von Nordmann's eye-worm (*F. oculi-humani* or *F. lentis*); and the nematode hæmatozoa. It may be doubted if any of the above-mentioned parasites ought to be included in the genus *Filaria*, as understood by modern helminthologists, but, practically, it is still found convenient thus to speak of them. The *Dracunculus* will be found described under ENTOZOA; while the microscopic nematodes infesting the blood are considered in the next article. See FILARIASIS. PATRICK MANSON.

FILARIASIS.—This term is applied to a form of helminthiasis characterised by the presence, actual or past, of the embryos of certain nematodes in the blood.

The name *Filaria sanguinis hominis* was applied by Lewis to the embryo filaria which he discovered in human blood in 1872; and so long as no other similar parasite was found to have its habitat in the circulation, the name was appropriate. Recently, however, the writer has pointed out that the blood in certain tropical countries is liable to be infested by the embryos of three additional species of filaria; it therefore becomes necessary to modify Lewis's original name. Observation has shown that the filaria of Lewis appears in the blood only at night, disappearing from it during the day; whereas, of the recently discovered species, one of them appears in the blood during the day only, disappearing from it at night; the others, in those individuals in whom they occur, are present in the circulation both during the day and during the night. Taking these facts as a basis for nomenclature, the writer has named the four species—

Filaria diurna, *Filaria nocturna*, *Filaria persians*, *Filaria Demarquayi* (*F. Ozardi*?).

Methods of demonstrating embryo filariæ in the blood.—Blood is obtained in the usual way from a finger-tip; but in looking for filariæ, the respective habits of the different species must be borne in mind. Thus, if search is to be made for *Filaria diurna*, the blood should be drawn between the hours of 11 A.M. and 6 P.M.; if for *Filaria nocturna*, between 9 P.M. and 6 A.M.; if for *Filaria persians* or for *Filaria Demarquayi*, it may be drawn at any hour of the day or night.

To study the movements of the filariæ, ordinary preparations of freshly drawn blood must be used; but to warrant a positive opinion as to the absence or presence of embryos in the circulation, and for purposes of enumeration, a larger quantity of blood than can be dealt with rapidly in the ordinary way must be searched. The following is a good plan. Spread about half a drop of blood on a slide in a uniform layer over an area of one square inch and allow it to dry. The slide may be stained and examined at once, or stored away till such time as may suit the observer's convenience. All that is necessary to demonstrate the filariæ in such slides, no matter how old they are, is to immerse them for an hour or so in a weak solution of eosin, or a weak solution of fuchsin or other aniline dye (one drop of the saturated alcoholic solution in an ounce of water), then wash and mount them. So prepared, the filariæ and white blood-corpuscles are stained, while the colour of the red blood-corpuscles is dis-

charged. To demonstrate the sheath, shape, and structure of the filariæ, make a thin film of blood, dry, fix in absolute alcohol, and stain in weak hæmatoxylin.

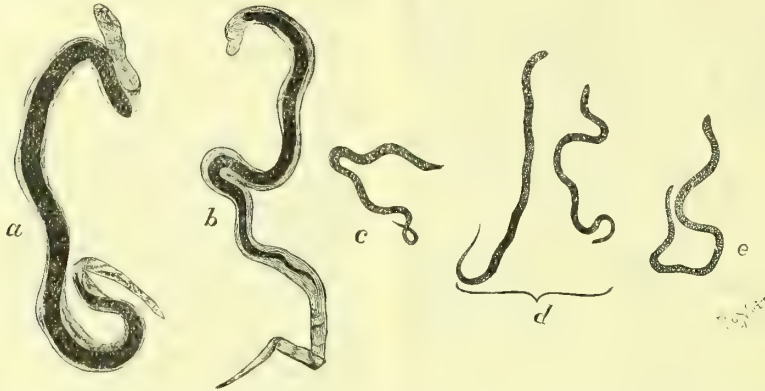
No matter how prepared, the slides ought to be scrutinised in the first instance with a very low objective—one inch is sufficient—and every part of them examined. A quick-travelling mechanical stage is of great assistance in this, and ensures thoroughness. When a filaria is found it can be centred and examined with a quarter-inch or higher objective ;

like an alimentary canal, or any definite internal structure, be made out.

As far as is at present known, the points of contrast in the species are as follows :—

1. *Filaria diurna* (fig. 1b).—The free hæmatozoal embryo of a species of filaria, the mature form of which is still unknown. Excepting that it enters the circulation during the day, this blood-worm resembles very closely in size, form, and habit the now familiar *Filaria nocturna*. The peculiarity in periodicity referred to is, doubtless, an adaptation

FIG. 1



- (a) *Filaria nocturna* × 300.—1. Measures $\frac{3}{16}$ in. by $\frac{1}{16}$ in., or thereabouts. 2. Is provided with a coarser sheath. 3. Caudal end tapers gradually for about one-fifth of the entire length of the animal, and terminates in a sharp, or nearly sharp point. 4. Cephalic end is rounded off, and has obscure oral movements of a pouting character. 5. Tongue-like organ relatively minute. 6. Appears in the blood at night, disappearing during the day. 7. Has a wriggling, but no locomotive, movement. 8. Most specimens have a well-marked granular-looking aggregation about the middle of the body.
- (b) *Filaria diurna* × 300. 1. Measures $\frac{3}{16}$ in. by $\frac{1}{16}$ in., or thereabouts. 2. Is provided with a very delicate sheath. 3. Caudal end tapers gradually for about one-fifth of the entire length of the animal, and terminates in a sharp, or nearly sharp, point. 4. Cephalic end is rounded off, and has distinct oral movements of a pouting character. 5. Tongue-like organ relatively minute. 6. Appears in the blood during the day, disappearing during the night. 7. Has a wriggling, but no locomotive, movement. 8. Most specimens have a faintly marked granular-looking aggregation about the middle of the body, but in none is it very well marked.
- (c) *Filaria Demarquayi*. × 300.—1. Measures $\frac{1}{16}$ in. by $\frac{1}{16}$ in., or thereabouts. 2. Has no sheath. 3. Caudal end tapers for about one-fifth of the entire length of the animal to a very sharp point. 4. Cephalic end rounded. 5. Tongue-like organ has not been observed. 6. Observes no periodicity. 7. Has a locomotive as well as wriggling movement.
- (d) *Filaria Ozwardi* (?) and *Filaria perstans* × 300. The *Filaria Ozwardi* in size and habits closely resembles *F. Demarquayi*.
- (e) *Filaria perstans* × 300. 1. Measures $\frac{1}{16}$ in. by $\frac{1}{16}$ in., or thereabouts. 2. Has no sheath. 3. Caudal end tapers more gradually for two-thirds of the entire length of the animal, and is abruptly truncated where it has tapered to about one-third of the diameter of the thickest part of the body. 4. Cephalic end is either conical or truncated, passing from one form to the other rapidly by a peculiar jerking, extruding and retracting, movement. 5. From time to time, a minute tongue-like organ is rapidly protruded and withdrawn at the extremity of the cephalic end. 6. Observes no periodicity. 7. Has a locomotive as well as a wriggling movement. 8. No granular appearance.

but it must not be forgotten that, in the first instance, only very low powers are suitable for searching.

The four species of embryo *Filaria sanguinis hominis* closely resemble each other. As seen in recently drawn blood, they are long, slender, gracefully formed, transparent, colourless, snake-shaped organisms, which for hours continue in a state of great activity, wriggling and twisting about on the slide like a wounded earthworm, or an eel on a hook. After a time movement gradually slows down ; but by a little management in preventing drying of the slide, the parasites can be kept alive for five or six days. In none of them can anything

to the habits of its intermediary host. There is some reason for supposing that *Filaria loa*—a parasite living in the connective tissues, and sometimes showing itself underneath the conjunctiva in negroes—is the mature form of *Filaria diurna* ; the geographical distribution of both, as far as our knowledge of this extends, is the same, and the undoubted embryo of *Filaria loa* resembles *Filaria diurna* in shape, and possibly in size ; and, moreover, a patient of the writer's, who at one time had a *Filaria loa* under his conjunctiva, later was found to have *Filaria diurna* in abundance in his blood. The parasite seems to be confined to the tropical zone of

Africa, more especially to the west coast (Southern Nigeria). Future observation is likely to show that a large proportion of the inhabitants of these districts are affected with this worm, and it is likely that in a proportion of instances some pathological condition may be traced to them. As yet our information on these points is much too fragmentary to warrant even speculation.

2. *Filaria nocturna* (fig. 1a).—The free hæmatozoal embryo of *Filaria Bancrofti*. We owe the first notice of this parasite to Demarquay, who, in 1863, found numerous specimens of what, considering the circumstances of the case, must have been *Filaria nocturna*, in the contents of a lymphous tumour of the thigh. Later, in 1869, Wucherer in Bahia, and, quite independently, in 1870, Lewis in Calcutta, found the same parasite in chylous urine. It was not, however, until 1872, when Lewis showed that the normal habitat of this embryo worm was

one or both ends, as may be observed in the wood-cut (fig. 2).'

As no sign of growth or of reproductive organs is distinguishable in this the hæmatozoal stage of the parasite, it is evident—and this was early recognised—that these blood-worms are the immature young of parasites which must exist in a sexually mature form elsewhere in the tissues of the human host, and in intimate relationship with the circulation. In 1877 Lewis found two living specimens—a male and a female—in the diseased tissues of a

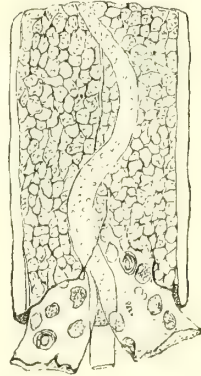


FIG. 4.—A portion of the mature *Filaria nocturna*, showing uterine tubules filled with ova in various stages of development; also the intestinal tube. $\times 100$.

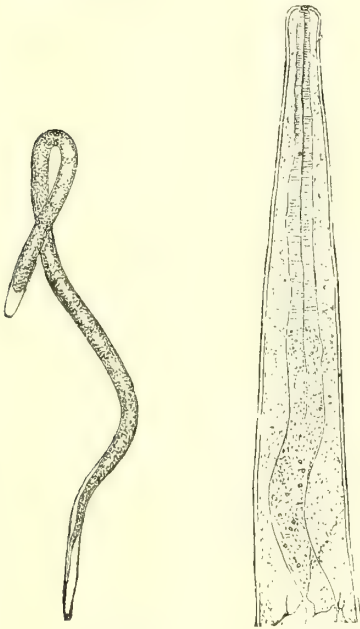


FIG. 2.—*Filaria nocturna*. $\times 300$. (From a micro-photograph.)

FIG. 3.—Anterior end of mature *Filaria nocturna*. $\times 100$.

the human blood, that the discovery excited much interest.

In the first issue of this Dictionary Lewis described the *Filaria sanguinis hominis nocturna* as follows: 'Its average length is $\frac{1}{75}$ " (= 0.34 mm.); its breadth $\frac{1}{3500}$ " (= 0.007 mm.), or about equal to the diameter of a red blood-corpuscle. It is enclosed in a transparent tubular sac, within which it can be seen to alternately contract and elongate itself. This sac is extremely delicate and translucent, and may sometimes, when the worm has shortened itself more than usual, be seen collapsed and folded like a ribbon, and the next moment be instantaneously straightened again, by the extension of the filaria to its ordinary length. After death the worm may occupy either the entire length of the tubular sac, or be so contracted as to leave the tube empty at

nævoid elephantiasis of the scrotum. Late in the previous year Bancroft, in Brisbane, found several mature females in a 'lymphatic' abscess of the arm, and also in 'an encysted hydrocele of the cord.' The latter were carefully examined by Cobbold, and the parasite was named by him *Filaria Bancrofti*. Since that time the mature *Filaria Bancrofti* has been found in India, China, Brazil, British Guiana, and, as an imported parasite, in England. It is a long thread-like worm, about the thickness of a horse-hair, of a white colour and firm consistence, and

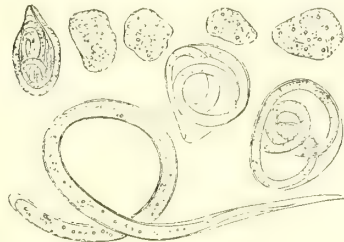


FIG. 5.—Ova and embryos of *Filaria nocturna*. $\times 300$

exhibits considerable activity, and a remarkable tendency to coil. Cobbold described the female as follows: 'Body capillary, smooth, uniform in thickness. Head with a simple circular mouth, destitute of papillæ (fig. 3). Neck narrow, about one third of the width of the body. Tail of female simple, bluntly pointed; reproductive outlet close to the head; anus immediately above the tip of the tail. Length of largest females $3\frac{1}{2}$ in., breadth $\frac{1}{50}$."' In the female the uterine tubes are packed with ova in all stages of development (figs. 4 and 5). Lewis

gives the measurement of these, in which the embryos are distinctly visible, as $\frac{1}{650}$ by $\frac{1}{750}$. In a fresh specimen of the filaria examined by the writer the largest ova gave slightly greater measurements, and he saw that towards the vaginal end of the uterine horns the embryos lay outstretched, and that in the vagina they had very much the appearance they have in the blood, proving that under normal circumstances the parasite is viviparous. The dimensions of the male worm, usually found in close association with one or more females, are considerably smaller (length 3·86 c.m., breadth 0·12 m.m.). The tail is generally arranged in a close spiral—like the tendril of a vine, the extreme tip being sharply incurved. The anus opens a short distance above the tip of the tail, and from it two chitinous spicules protrude, one of them being long, the other short.

It is now known that the parent-worms live together in the lymphatics; that the embryo while *in utero*, by dint of vigorous movements, stretches its chorionic envelope to form the long tubular sheath in which it lies extended, as we see it in the blood and lymph, and that, after this stretching of the chorion is complete, the embryo is borne into the lymph-stream, which carries it through the glands, along the thoracic duct, and thus into the blood. Under ordinary circumstances of health and habit the hæmatozoon cannot be found in the blood during the day, but at evening it appears there. At first only a very few can be found; as night advances their numbers gradually increase; about midnight they attain their maximum—in some cases as many as 500 may be found in a single drop; after this they gradually diminish up to about 8 or 9 A.M., when they disappear for the day. An attack of fever may disturb this periodicity, and Stephen Mackenzie has shown—and his observations have been abundantly confirmed—that by causing the subject of filaria infection to sleep during the day and keep awake during the night, the habit of the embryo parasite in his blood is correspondingly changed: it then appears, contrary to its normal habit, in the blood during the day, and disappears from it at night.

This singular periodicity is, apparently, an adaptation to the nocturnal habits of the female of particular species of mosquito, which imbibe with the blood the young filaria, and act as its intermediary hosts. Transferred to the stomach of the mosquito, the filaria first casts its chorionic envelope, and then bores its way into the thorax and thoracic muscles of the insect. Here it undergoes a remarkable metamorphosis, eventuating in its becoming possessed of an alimentary canal, a circumcaudal circle of three papillæ, and greatly increased size, power, and activity. This metamorphosis takes about sixteen days to complete. The filaria then quits the thoracic muscles and, travelling forward, lodges for a time in the head of the mosquito, finally passing into the proboscis, by which channel it is to be presumed it enters a human host when the insect next proceeds to feed on such.

GEOGRAPHICAL DISTRIBUTION.—*Filaria nocturna* has been found in most tropical and subtropical countries in which it has been properly looked for. Its exact geographical distribution as an endemic parasite is doubtless in keeping with the geographical distribution of the particular species of mosquito subserving its interests as intermediary host; but, as the filaria may live for many years in the tissues of its human host, it is frequently brought by

travellers from tropical and other countries to colder latitudes.

PATHOLOGICAL RELATIONS.—Chyluria, nævoid elephantiasis or lymph-scrutum, chylous dropsy of the tunica vaginalis (variously termed chylocele, lymphocele, galactocele), varicose and indurated inguinal glands, chylous ascites, recurring orchitis, certain kinds of abscess occurring in the limbs, especially in the thighs and groins, lymphangitis, and lymphatic fever, are almost invariably accompanied or preceded by the presence of the filaria in the blood, or in the lymph, or in both. The association is so constant and so intimate that in 99 cases out of 100 these diseases may be confidently attributed to the filaria, at all events when they are of tropical origin.

Tropical elephantiasis arabum is also attributed to the filaria, although the parasite is by no means always found in the fully developed cases. Theoretically, all elephantoid diseases are produced by some mechanical or other physical influence operating by obstructing or irritating the lymphatic vessels. In tropical elephantiasis it is maintained that this influence is the *Filaria nocturna*; and on the following grounds:—cases are on record of lymph-scrutum—a disease certainly attributable to the filaria—passing into true elephantiasis arabum; many cases of true elephantiasis arabum scroti have a history of antecedent lymph-scrutum; lymph-scrutum has supervened in flaps left after removal of an elephantiasis scroti by operation; elephantiasis of the leg has followed on the removal of a lymph-scrutum; lymph-scrutum and elephantiasis of the leg sometimes co-exist in the same individual; a case is on record of lymphorrhagia in an elephantoid leg co-existing with varicose inguinal glands and filariæ, and in which chyluria subsequently developed; an instance is recorded in which a son developed a lymph-scrutum, and the mother—living in the same house—an elephantiasis of the leg; where elephantiasis is endemic, there too chyluria and lymph-scrutum are found. It is unreasonable to suppose that the combinations and concurrences mentioned were, in every instance, simply coincidences; much more likely the same cause produced them all. This being proved to be the filaria in chyluria, lymph-scrutum, and varicose inguinal glands, it follows that it must be the filaria also that produces true tropical elephantiasis arabum. Furthermore, as far as is known, the geographical distribution of elephantiasis is co-extensive with that of the filaria.

Because the filaria is not always found in elephantiasis, it has been maintained by some that this parasite cannot be the cause of this disease. The fact is, the filaria is mortal and may die before its host; nevertheless, the ill it has wrought may not disappear at its death. It sometimes happens that in chyluria and in lymph-scrutum no filariæ can be found in the blood, urine, or lymph, although at one period they must have been present; sometimes it can be found in the lymph only, and not in the blood; and instances are on record in which the parasite disappeared from the blood and escaping lymph while the cases were under observation, yet the characteristic morbid discharges persisted. In pathology, as in other processes, a transient influence may produce a permanent lesion. It must be admitted, however, that the reason of the very frequent absence of the embryos of the filaria from the blood in developed elephantiasis is

not definitely known, and further observations bearing on this point are much wanted.

From the fact that in many countries where the filaria is endemic from 10 to 50 per cent. of the adult population harbour it, yet only a small proportion have any of the diseases enumerated, it is evident that the parasite does not necessarily give rise to disease. It would appear that so long as the parasite is healthy and normally situated it is innocuous. Should it die, it may act as a foreign body and cause abscess; the dead filaria has actually been found in lymphatic abscesses. But the precise way in which the parasite usually brings about pathological obstruction of the lymphatics is, as yet, to some extent a matter of conjecture. Of two ways in which it so acts there is good evidence. (1) The parent worm may cause lymphatic stasis by operating at a point high up the thoracic duct, either itself plugging that vessel, or giving rise to inflammatory thickening in or around its walls, ending in stenosis. (2) As mentioned, there are instances on record in which, though the filaria was present in the chylous urine, or in the lymph drawn from a lymph-scrotum, or from varicose inguinal glands—yet, after the most careful and prolonged search, it could not be discovered in the blood. It is evident that in these cases there must have been complete obstruction of a lymphatic area, otherwise the embryo filariae must have appeared in the blood, as in ordinary examples of filarial infection. The obstruction could not possibly have been in the thoracic duct, for, if complete in this situation, it must have speedily brought about the death of the patient. It is safe, therefore, to infer that it must have been somewhere on the distal side of the receptaculum chyli. In two such cases the ova of the parasite have been found in the lymph; and it is believed that in this circumstance we have the explanation of the site and character of the lymphatic obstruction in a large proportion of cases. Normally, the parasite is viviparous, and the long, slender, active embryo has no difficulty in finding its way along the lymphatics and through the glands into the circulation. But should the embryo be born prematurely, as in the two cases referred to, before the chorionic envelope has undergone the stretching-process already described—that is, should ova (seven or eight times the diameter of the outstretched young) be prematurely launched into the lymph-stream, they act as emboli, and, being very numerous, effectually plug the glands connected, directly or by anastomosis, with the lymphatic vessel in which the aborting parent lies. The location of the worm; the degree of obstruction, whether caused by embolism, pressure, or stenosis; pressure by enlarged glands interfering with the compensating drainage of the affected areas by the veins; traumatic irritation of the congested lymphatic area; the position of the part involved as regards dependence or elevation—a most important factor in elephantiasis; the health of the patient; and other circumstances, easily understood, will determine the site and exact character of the resulting disease.

Chyluria.—Fr. *Urine laiteuse*; Ger. *Chylurie*; *Milchsaftiger Harnabgang*.

The affection known as chylous or chylid urine has long remained a puzzle to physicians, not only on account of the very remarkable character assumed by the secretion, but also on account of the very erratic course which the disease runs. Scarcely any two persons affected with this malady give a similar

account of its mode of onset, of the duration of the attack, or of the symptoms and seasons of its occurrence.

DEFINITION.—A diseased condition, originating in the great majority of cases in tropical or sub-tropical climates, which manifests itself usually by a more or less milky appearance of the urine, modified at times by an admixture of a variable proportion of a substance resembling blood.

On standing, the fluid coagulates, so as to present the appearance of size. *Filaria nocturna* is generally found in the blood and urine of persons affected with the disease.

GEOGRAPHICAL DISTRIBUTION.—This is peculiar. The vast majority of cases are tropical or sub-tropical in their origin. Nevertheless, from time to time, cases crop up in the persons of individuals who have never been out of the temperate zones, or in any way exposed to tropical influences. Moreover, the disease is not equally distributed over the zone in which, in a general way, it may be said to be endemic. In most tropical countries it is of more or less frequent occurrence, but there are districts in these same countries which enjoy an immunity similar to that of temperate climates. The explanation of this apparent caprice in the distribution of chyluria is afforded by the peculiarity of the distribution of *Filaria nocturna*—its principal aetiological factor.

SYMPTOMS.—So far as is at present known, there are no premonitory symptoms of chyluria. Sometimes the only symptom is the milky condition of the urine—a condition which usually comes on very suddenly; generally, however, the patient complains of an uneasiness, scarcely amounting to pain, across the loins, along the ureters, over the bladder, or along the course of the urethra—especially towards the perinæum in the male. There is generally marked debility, with mental depression. Occasionally chylous discharges take place from various parts of the body—the axilla, the surface of the abdomen, the groin, and especially from the scrotum, in that condition of it which is known as elephantiasis lymphangiectodes (Bristowe), nævoid elephantiasis, varix lymphaticus or lymph-scrotum. The disease is also sometimes observed associated with true elephantiasis of the limbs and scrotum. It occurs at all ages, from childhood to extreme old age, and in about equal proportion between the sexes—perhaps more frequently in the female than in the male.

With regard to the *urine*, it presents, as already mentioned, a milky appearance, and frequently emits a strong milky or whey-like odour, which is made more evident by warmth. After standing a short time the fluid coagulates, so as to form a more or less semi-solid mass resembling blanc-mange. In the course of a few hours the clot contracts, and the urine is prone to rapid decomposition. In some cases the fluid presents a pink colour, from the admixture of coloured corpuscles like those of blood. Commonly the blood-like admixture, when present, is seen forming a shredly adherent coagulum at the bottom of the vessel after it has stood for some hours. Not infrequently the flow of urine is suddenly stopped during micturition by the blocking up temporarily of the urethra with clot. The specific gravity varies greatly—may range in the same individual from 1007 to over 1020. Shaken up with ether, the urine loses its milky aspect; and when nitric acid or heat is applied, a precipi-

tate almost invariably results. These characters, and the fact of the coagulability of the fluid, indicate the presence of fat, albumen, and fibrin, all of which are to be considered as abnormal constituents. The proportion, however, in which they exist in different individuals, and even in the same individual at different times, varies greatly. Dr. Beale's analyses show, that though a specimen of urine may contain at one time 1·39 per cent. of fat, another specimen, obtained a few hours later, from the same person, may contain none. In the majority of cases the fatty element is usually scanty in the morning before meals, and so are the other abnormal elements, unless exercise have been taken, or the circulation otherwise accelerated.

Microscopical Characters of the Urine.—*Filaria nocturna* may generally be detected in the urine. In making a search for the parasite, it is advisable to pick out one of the coagulated shreds found in the urine, transfer it to the glass slide by means of a forceps or pipette, and carefully tease the fragment before applying the cover-glass. A still better plan is to break up the coagulum in the urine with a glass rod as soon as it is formed, and then to search the sediment which, after an hour or two, collects at the bottom of the vessel in the same way as is customary in examining for 'casts.' As large a slide as is practicable ought to be examined, and a low power employed in the first instance, as it often happens that the filariae are present only in very small numbers, and might be readily overlooked if a small quantity of sediment were examined or a high power employed.

The periodicity characteristic of the filaria when in the blood is not observed by the parasite in the urine. It may be found in chylous urine passed during the day as well as in that passed during the night. In a certain, though small, proportion of cases the parasite cannot be found; but there is every reason to suppose that at one time, even in these instances, the filaria has been present. Cases are on record in which, after its presence had been fully ascertained, the parasite disappeared from the urine while the patient was under observation, the chyluria persisting. In the rare instances of chyluria originating outside the endemic area the filaria will, of course, not be found at any time. It is difficult to define the limits of this area, however, so that in every case of chyluria the parasite ought to be sought for.

The other leading microscopical character of the urine is the minutely molecular matter—fat in an emulsified condition—to which the fluid owes its opaline or milky aspect. There are also numerous white lymphoid corpuscles, together with red corpuscles, numerous or the reverse according to the degree of sanguinolence of the urine. Casts of the tubular structure of the kidney—indicative of organic disease of these organs—are seldom to be seen.

With regard to the *microscopical examination of the blood*, the writer has not observed that the corpuscles or serum presented any abnormality indicative of the presence of fatty matter in any form—the serum has seemed as clear and as free from molecular matter as normal blood. As far as his experience goes, the only feature worthy of special note in connection with microscopical examination of the blood in chyluria is the presence of the hæmatozoon already referred to. In consequence of the periodicity observed by the filaria in the blood, it is absolutely necessary that this examination be made

during the late evening, the night, or the early morning: some time between 9 P.M. and 6 A.M. is the best. Searched for at this time, and as recommended, the filaria is usually found in the blood; if it is absent from the urine it will not be found in the blood. If it is found in the blood in chyluric cases it will also be found in the urine; but it sometimes happens that, though the filaria is present in the urine, yet the most careful and prolonged examination may fail to detect it in the blood.

ANATOMICAL CHARACTERS.—The *post-mortem* examinations of persons who have died while affected with chyluria also testify to the freedom of the kidneys from disease. But, although no evidence of disease of the parenchyma of the kidneys has been discovered in connection with this disease, recent and carefully conducted *post-mortem* examinations have shown that the lymphatic system connected with the urinary tract may be seriously deranged. This is only what might have been expected from the frequency with which chyluria is associated with diseased conditions of the lymphatics in different parts of the body—notably of the scrotum and the inguinal glands. Stephen Mackenzie, Ponfick, Curnow, and the writer have recorded cases in which thickening and dilatation of the thoracic duct and marked varicosity of the pelvic and other lymphatics were found.

Multiplied observations, made during recent years, have made it more than probable that Carter's view of the pathology of chyluria is the correct one—namely, that somewhere in the urinary tract there is a varicose condition of the lymphatics, similar to that found in elephantiasis lymphangiectodes, varix lymphaticus, or lymph-scrotum—a disease, as pointed out, also commonly associated with the filaria, and very frequently with chyluria. Either the mature parasite itself, or its young, or its prematurely expelled ova, or stenotic conditions of the lymphatic trunks induced by some of these, act as a mechanical obstruction to the onward flow of the lymph, and thus induce a varicose condition of the lymphatics on the distal side of the seat of the obstruction. Rupture of the varicose vessels will then admit the lymph to the urine if the varicosity is anywhere in the urinary tract.

For diagnostic purposes, as well as to arrive at a correct idea of the pathology of chyluria, it is necessary to bear in mind that while chylous urine or its coagula, on the one hand, may be clear as the purest gelatine or, on the other hand, white like milk—in most cases from time to time, in others always—they may have a blood-like tinge, often of great intensity. This latter circumstance is likely to lead the unwary to a diagnosis of hæmaturia, and give rise to the idea that blood-vessels as well as lymph-vessels are in some way involved; all the more so as genuine hæmaturia is endemic and very prevalent in many of the countries—Egypt, for example—in which chyluria is also endemic (see ENTOZOA: *Bilharzia*). Some authors, misled by a false idea of the pathology of the disease, describe these cases of red chyluria under the name of hæmaturia, or hæmato-chyluria, assuming, and apparently being under the impression, that genuine blood from the blood-vessels is the cause of the red colour of the chylous clots in the urine. It is quite possible for the same individual to be the subject of chyluria and also of hæmaturia, and such a coincidence has been noted; but in an ordinary case of red-tinted chylous urine there is no reason to suppose

that the term 'chyluria,' applied to these cases, is unscientific, or that the characteristic pathological feature is derived from any set of vessels other than the lymphatics. In the first place, it is very unlikely that both sets of vessels—blood-vessels and lymphatics—could be similarly affected in the same individual and in the same organ; and, in the second place, it is well known that the contents of the thoracic duct, and of some of the larger lymphatics, and also lymph long stagnant in obstructed lymphatics, acquire a red tinge from the normal advance of the development in them of red blood-corpuscles. Hence, undoubtedly, the red colour so frequently seen in the coagula referred to. In the same way may be explained the absence, or gradations, of milkiness occasionally encountered. If the abnormal element in the urine be derived from vessels into which no chyle has regurgitated, if the vessels are filled with a chyle in which the fatty element is defective, either from peculiarities of diet or from its relation as regards time to the period of digestion, it will be less milky and more transparent and jelly-like. The differences in the analyses of chylous urines admit of a similar explanation.

Of the ætiological significance of the presence of the filaria in the circulation there can be no doubt—more especially when the number of observations recorded within the short period that has elapsed since attention has been drawn to its existence therein, is taken into consideration. These suggest more than a fortuitous connection; indeed, it might rather be said that chylous effusions may be considered as symptomatic of the parasitism.

As regards those cases of chyluria apparently unassociated with the filaria, we must bear in mind that anything which could give rise to constriction or blocking of the lymph-channels might act in the same way as the filaria does as regards producing lymphatic varicosity and rupture. Thus pressure by a tumour, constriction from inflammatory thickening in or about the walls of the thoracic duct or pelvic lymphatics, or stricture following ulceration of their walls, might, it is conceivable, bring about this state of varicosity leading to rupture and possibly to chyluria.

PROGNOSIS.—Persons have been known to suffer off and on from this affection for from one to fifty years. This would suggest that the malady usually runs a chronic course, which, as a rule, it doubtless does; on the other hand, patients apparently in fair health otherwise have been known to die very unexpectedly from no recognised acute disorder. With regard to the prospect of a cure, a very guarded opinion should be given, as the probability is that the complaint will return again and again so long as life lasts—even when the disease commences at a very early age, and often after a complete change of climate and avocation.

TREATMENT.—This has proved extremely unsatisfactory in almost all the cases recorded; in fact it cannot be distinctly stated that the course of the disease has been materially modified, much less cured, by any known remedy. Iodide of potassium has been tried in large doses, and in some cases appears to have been beneficial; in others the tincture of the perchloride of iron has seemed to be more successful. A decoction of the bark of *Rhizophora racemosa* (mangrove) has a reputation in Guiana, just as a decoction of the seed of *Nigella sativa* (used also as a condiment in curries) has in India. The latter remedy has, however, been

known to be powerless in mitigating the malady even in cases where on former occasions it had been resorted to with apparent success. Perhaps the most satisfactory results which have been published are those which have followed the administration of large doses of gallic acid—one or two drachms a day.

Large doses of benzoic acid given in glycerine have, in some hands, been followed by a cessation for a time of the chyluria. But from what we now know about the pathology of this disease, it is evident that rest in the recumbent position, with elevation of the pelvis, reduction of fluids, and complete avoidance of food containing fat, is by far the most important indication to be attended to in the treatment.

'Varicose' Inguinal Glands.—This is a troublesome and by no means uncommon result of filarial obstruction of the pelvic or abdominal lymphatic trunks. The condition is sometimes the only clinical evidence of filariasis. Frequently it concurs with chyluria, or with lymph-scrotum, or with both. Usually the glands, femoral and inguinal, of both sides are involved; more rarely only one groin, or one set of glands, is affected. There is a doughy, rounded and perhaps lobulated, semi-fluctuating swelling sometimes as large as the fist over Scarpa's triangle. The skin is freely movable over the mass, which, however, is firmly attached to the deeper structures. On lying down the swelling slowly subsides, to return on the erect position being assumed. There is only slight impulse on coughing.

These swellings are often mistaken for herniæ; a little care will enable the surgeon to arrive at a correct diagnosis. If there be any doubt, an examination of the blood at night will probably reveal filariæ; the cautious use of a fine hypodermic syringe by permitting abstraction of chylous or lymphous fluid—generally containing embryo filariæ, will remove all doubt. The axillary glands are sometimes, though much more rarely, similarly affected.

TREATMENT.—If these swellings give rise to much inconvenience, either from painful tension or from being the seat of frequently recurring attacks of inflammation, they should be excised; if, on the other hand, they cause no serious inconvenience, they had better be left alone, support by elastic bandaging being applied if deemed necessary.

Lymph-scrotum.—Cutaneous lymphatic varix of filarial origin may occur in any region, most commonly in the scrotum. Here it may or may not concur with a varicose condition of the lymphatics of the spermatic cord, with chylous dropsy of the tunica vaginalis, with chyluria, with varicose inguinal glands, or with elephantiasis of the leg. The surface of the scrotum is generally studded with innumerable dilated lymphatics; sometimes there are only one or two points of disease. Occasionally the varices become tense and rupture, either spontaneously or as the result of violence permitting the escape of enormous quantities of chyle, or of lymph. As a rule the scrotum though soft is increased in bulk and pendulous. Inflammation of the parts, often leading to abscess and accompanied by high fever, is a common and in some cases a regular periodic occurrence. In time this disease is apt to eventuate in the development of genuine elephantiasis scroti.

TREATMENT.—Free excision, leaving no flaps, is the only effective treatment.

Elephantiasis Arabum (ἐλέφας, an elephant).
SYNON.: Fr. *Éléphantiasis*; Ger. *Elephantiasis*.

This condition, although not generally in its

advanced phases associated with the presence of *Filaria nocturna* in the circulation, is believed to be the most common of the various lesions produced by this parasite. It is characterised by recurrence of febrile paroxysms attended by inflammation and progressive hypertrophy of the integument and areolar tissue, chiefly of the extremities and genital organs, by swelling of the lymphatic glands, enlargement and dilatation of the lymphatics, and in some cases by the co-existence of chyluria.

ETIOLOGY.—Elephantiasis is endemic in India, the Malayan peninsula, China, Egypt, Arabia, Polynesia, Africa, the West Indies, and parts of America, in fact wherever *Filaria nocturna* occurs. It occurs sporadically all over the globe, but these sporadic cases, although induced by obstruction of lymphatics, are not necessarily filarial in origin. Removal from the endemic area frequently checks the disease, while return there reproduces it. Elephantiasis affects both sexes and persons of all ages and conditions of life, but more men suffer from it than women. No race is exempt, but, doubtless owing to their habits of life affecting liability to filarial infections, it is much more frequent in dark than in fair races. It occurs at all ages, but is most common in adult and middle life, comparatively rarely beginning in young children or in the aged. Elephantiasis is not hereditary. Although Richards found that of 236 persons, 73 per cent. had one or both parents affected, this circumstance is no proof of heredity and depends on the fact that parents and children living under the same roof are equally exposed to filarial invasion. Various causes have been assigned for the disease. Air, water, food, and, as it is common near the sea-coast, eating fish, have been frequently credited with producing it. Certain forms of vegetation, and the geological formation of the soil, have also been regarded as predisposing and determining causes. Climate and locality, combined with bad living, hard work, and exposure of the feet and legs to injury in fishing and agricultural and similar work, are doubtless contributory causes.

ANATOMICAL CHARACTERS.—The hypertrophy of elephantiasis in most cases appears to be simply an increase in the natural elements of the part, the blood-vessels and lymphatics sharing in the growth. In other cases the lymphatics and lymph-spaces are most concerned, giving rise to a condition that has been described as *navoid elephantiasis*, in which the appearance is presented of a soft and fluctuating swelling, which when punctured gives issue to a white or pinkish fluid, very closely resembling chyle. The lymphatic glands also share in the enlargement. In other respects the progress of this variety is like that of the ordinary form of the disease.

Although *Filaria nocturna* is sometimes found in great numbers in the blood of persons suffering from elephantiasis, this is not usual; in fact, as compared with others in a filariated community, recent observations seem to prove that the subjects of elephantiasis show filariæ in the peripheral circulation less frequently than their neighbours. It would seem that in the development of solid elephantiasis the filaria is either killed or is cut off from the circulation.

SYMPTOMS.—The ordinary form in which elephantiasis presents itself is hypertrophy of the integument and areolar tissue of some part of the trunk or limbs, and notably of the legs and genital organs. The skin becomes enormously thickened

by hypertrophy of all the fibrous elements of its structure, attended by the deposit of a quantity of albuminous fluid in the interstices of the areolar tissue. The papillæ are prominent and much increased in size. The integument is formed into hard masses or folds, with a rugose condition of the surface, not unlike the appearance of an elephant's leg. The feet and toes are sometimes almost hidden, and the scrotum or labia form enormous outgrowths. The scrotum often attains great weight, and generally includes large hydroceles. Scrotal tumours have been removed weighing upwards of 100 lbs.

The onset of elephantiasis is frequently violent and attended with great suffering. There is high fever; intense pain in the lumbar region, groin, spermatic cords, and testes, which become swollen, while acute hydroceles form. These symptoms are often attended with sympathetic vomiting, nausea, and rapid erythematous swelling of the external parts; and, if the extremities be attacked, the swelling may be tense and painful, accompanied by much effusion into the areolar tissue. The surface of the integuments is much inflamed, and there is sometimes a serous discharge.

In some cases of elephantiasis the integuments are also the seat of a dilated and turgid condition of the lymphatic vessels, which during the periods of vascular excitement, when the febrile attacks occur, give way and discharge a lymphous fluid; in other cases the surface temporarily assumes an herpetic condition, which exudes an acrid and offensive serous fluid.

Elephantiasis not infrequently occurs without much or any obvious injury to, or disturbance of, the general health during the intervals between the febrile attacks, which in some cases are few and slight. Apart from malarial or other complication, the appetite, spirits, and strength are good, the functions are all normally performed, and the only inconvenience is that due to the size and weight of the outgrowth. Albuminuria, as well as chyluria, is occasionally present.

In some cases, after the outgrowth has attained a certain bulk, it ceases to grow altogether, or increases slowly and insidiously without febrile disturbance, and in such cases the general health remains good. But there is generally a tendency to recurrence of the fever once or twice a month, when the parts affected become tense, hot, painful, and swollen, and often discharge a serous or lymph-like fluid, which may be acrid and offensive. Some tumours, on the other hand, are very slightly, if at all, so affected, and remain perfectly dry. In all cases, however, some growth goes on; and even when, as occasionally happens, fever has ceased to recur, there may be a gradual, but slow and painless, increase of the hypertrophy. The greatest variety and uncertainty obtain in the duration and progress of the growth: sometimes it is very rapid, at other times it is slow, with intermissions of activity and indolence of development. The disease elsewhere than in the genitals, unless it be accompanied by exhaustion and debility, causes no failure in the generative powers in either sex.

COURSE, DURATION, AND TERMINATIONS.—According to Richards, the average duration of the disease, as deduced from the observation of 636 cases, was $1\frac{1}{2}$ years; and he notes that the earliest age was nine years, while the latest at which he observed it was eighty years. It appears from

this that the disease has little influence in shortening life.

TREATMENT.—Little has yet been done by constitutional treatment in cases of elephantiasis. Remedies useful during the febrile paroxysms have little power in checking the disease or in preventing its recurrence. Iodine, combined with quinine, arsenic, and iron, has been found useful. During the febrile state salines and diaphoretics are indicated. Opium may be necessary to relieve the intense pain which often accompanies the onset of the stage of excitement. When the febrile stage has passed, quinine is useful, which, if anemia exist, should be combined with iron. The local application of iodine, in such forms as the iodide of lead or biniodide of mercury ointment, has been thought useful; but as this is generally combined with elastic bandaging and the recumbent posture, the benefit can hardly be ascribed to the drug. Such measures, along with improved hygienic conditions, may no doubt control the progress of the disease and relieve suffering. No remedy, however, is so potent as change of climate, by removal from the endemic site of the disease. This, if effected in the earliest stages, may completely arrest the disease, and perhaps even disperse any incipient structural change. This has been observed in the rare cases in which elephantiasis occurs in Europeans, who, on returning to Europe, have after a time lost the disease, and almost, or entirely, all the hypertrophic changes that may have occurred. Natives of India improve if they leave the endemic area during the early stages, and go and reside in other and drier localities. The *modus operandi* of change to a cooler climate is not very apparent, unless it be that in such climates, owing to the parts being covered with clothing, and therefore less exposed to injuries, and thereby to secondary bacterial infections, the erysipeloid attacks are not so likely to be induced. The influence of cold on the skin and dartos-muscle, as well as on the general health, may also contribute to bring about the improvement which is very generally produced by residence in higher latitudes. When the hypertrophy is advanced, the paroxysms of fever are still liable to recur, even when the climate is changed, though with less violence.

Surgical treatment. when the hypertrophy is advanced, is often most successful in relieving the sufferer, not only of the local trouble, but also of the fever. Tumours of the genital organs, sometimes of enormous size, are now removed with complete success and comparatively small mortality. Before commencing the operation, especially in the case of a large scrotal tumour, it is well to drain it of blood by placing the patient on his back, and elevating the tumour on the abdomen for an hour or so, during which time pressure by a bandage (a modification of Esmarch's) may be tried, and cold (ice) may be applied. During the operation, the application of an elastic ligature round the neck of the tumour also prevents loss of blood.

The removal of a scrotal tumour is effected by incisions along the course of the cords and the dorsum penis. The cords, testicles, and penis are turned out by a few touches of the knife, and then reflected and held up on the abdomen, while the mass of the tumour is rapidly swept away by a few bold incisions in the perineum. The numerous venous and arterial bleeding points should then be arrested, and the wound dressed with anti-

septic applications. No attempt should be made to preserve flaps of integument either for the penis or testes. It is unnecessary, and almost certain to be followed by recurrence of the disease. The process of cicatrisation goes on rapidly, and in from two to four months all is closed in by cicatricial tissue, which gradually perfects itself, and has no liability to become the seat of a return of the disease.

In the endemic regions mosquito-bites should be carefully guarded against, and the same precautions adopted in this respect as for malaria (*see* MALARIA). The subject of filariasis should be regarded as a danger not only to his neighbours, but also, owing to liability to repeated auto-infection by mosquitoes, to himself.

3. *Filaria perstans* (fig. 1, *e*).—Hitherto this hæmatozoon has been found only in the blood of negroes from the West Coast of Africa, and in that of the Buck Indians of British Guiana: in the latter it is usually associated with *Filaria Demarquayi*. Twice it has been found in company with *Filaria diurna*.

Attention to the points of contrast between *Filaria perstans* and the other blood-worms—*Filaria diurna* and *Filaria nocturna*—as set forth in the table in p. 540, will lead to its recognition, and the fact that it is blunt-tailed readily distinguishes it from *Filaria Demarquayi*. In addition to what has been stated in this table, it may be mentioned that the body of this parasite is possessed in a high degree of powers of elongation and contraction, and that the measurements given in the table referred to are those of the animal when it is dead or moribund. When travelling actively among the blood-corpuses it sometimes so extends itself as to appear a mere thread, the elongation concurring with a corresponding attenuation; at other times, and when not actively travelling, it assumes the proportions given. A very striking habit which it has is that of coiling itself up like a rope, and remaining motionless in this attitude for a few seconds before resuming its usual movements. Another peculiarity worth mentioning is the result, apparently, of the locomotive habits of this parasite: not infrequently it is seen fixed to one spot on the slide, vainly endeavouring to break away, and constricted by a delicate rope of fibrin. It would seem that in moving about the slide it pushes the strings of fibrin in front of it; and at times becomes entangled in a rope of them. The blunt tail and absence of a sheath are very characteristic.

Filaria perstans is probably very common in the endemic regions: from 50 to 90 per cent. of the natives being affected. The writer has twice met with it in Europeans from West Africa.

The parental forms, male and female, have been found both by Daniels in natives of British Guiana and by the writer in a negro from the Congo. In length they closely approximate to *Filaria Bancrofti* and the parental form of *Filaria Demarquayi* (*Filaria Ossardi*); in diameter, however, both the female (.120 mm.) and the male (.06 mm.) are only about half the corresponding dimension of *Filaria Bancrofti*. The tail in both sexes terminates in a distinctive bifid or mitre-like appendage. Hitherto these worms have been found only in subperitoneal and subpericardial connective tissue, the sexes being in association.

That in certain cases it gives rise to pathological conditions is probable, but what these are we are

as yet ignorant. In many cases the parasite is associated with the peculiar disease called Negro Lethargy or the Sleeping Sickness of the Congo; on the other hand there is often no concomitant disease. There may be some grounds for associating it with one type of a peculiar African disease called Craw-craw, a kind of itch, apparently induced by a filaria-like parasite, and not the *Acarus scabiei*; as yet this supposition is little more than hypothetical.

The presence of a beak, the absence of a sheath, the marked locomotive habits, the extensibility and pliancy of its body, and also the non-observance of any periodicity in its entrances and exits from the circulation, suggest that the embryo *Filaria perstans* gains its freedom and enters its particular intermediary host by dint of its own efforts, and that it is not, as is the case with *Filaria nocturna* and probably *diurna*, dependent on the good offices of some species of mosquito or other bloodsucker.

4. *Filaria Demarquayi* (*F. Ozardi*?) (fig. 1, c and d) occurs in St. Vincent and St. Lucia, West Indies, in British Guiana, and in New Guinea. In size, locomotive habit, absence of sheath, and absence of periodicity it resembles *Filaria perstans*. It is readily distinguished from the latter, however, by the needle-like sharpness of its tail. Like *Filaria perstans*, though occasionally found in prodigious numbers, it is usually rather scantily represented in, at all events, the peripheral circulation—some four or five specimens occurring in a drop of blood. The female parental form—found by Daniels in British Guiana—is hardly distinguishable from *Filaria Bancrofti*, except, perhaps, by the fact that the tip of the tail is bulbous in contrast to the tapering and truncated tail of the latter; on the other hand, the males of the two species differ markedly in diameter, that of *Filaria Demarquayi* being twice that of *Filaria Bancrofti*. The head, too, is finer and more tapering in *Filaria Demarquayi*. Daniels found the mature worms in association in the sub-peritoneal connective tissue of the abdominal parietes. So far we are unable to attribute pathological effects to this parasite.

It would be well to bear in mind that until all the parental forms of the various *Filarie sanguinis* have been discovered there is no assurance that very similar embryos may not belong to distinct species.

PATRICK MANSON.

FILE-CUTTERS' DISEASE.—See OCCUPATION - DISEASES.

FINGERS, Clubbing of.—In this affection the terminal phalanges of the fingers become bulbous or club-shaped, and in extreme cases somewhat flattened laterally. The parts affected are the finger-pads and the tissues around the nail. The bones and joints are not affected. The nail becomes more curved towards the palm, and the skin over the root of the nail is tense and shiny, but otherwise unchanged. The bulbous fingers are livid or dusky when there is cyanosis, but not otherwise.

The toes may be similarly affected, but nearly always in a less degree. Kingston Fowler points out that where true clubbing is present the hard margin at the root of the nail can always be felt when the free edge is depressed, showing that the nail is raised from its bed.

The affection is usually insidious, but may develop rapidly. It is generally bilateral and symmetrical, and may occur in a variety of different diseases. It

is chiefly met with in bronchiectasis, chronic pulmonary tuberculosis with excavation, chronic empyema or pleurisy, chronic bronchitis and bronchial asthma, and congenital morbus cordis. It has also been observed in abscess of the lung and aortic aneurysm.

Several cases of unilateral clubbing in connection with subclavian aneurysm are on record. It has also been observed in a variety of other diseases, such as amyloid disease, cirrhosis of the liver, and purpura, and in one case mentioned by Samuel West clubbing was well marked in a perfectly healthy man.

The exact nature of the changes present is not known. Buhl attributes the condition to a fibrous thickening of the rete mucosum, and chronic venous congestion from obstruction to venous return is commonly given as the cause. But although this theory of causation may account for the unilateral cases it is, as West observes, inadequate; 'for in the first place clubbing is not oedema, it is hardly ever met with in association with oedema or in cases where cyanosis and central obstruction are considerable, as with heart-diseases other than congenital affections, with intra-thoracic tumours, &c.' Moreover it occurs under conditions where there is no obstruction to venous return, and is far more often absent than present even in those diseases with which it is most commonly associated. We must therefore conclude that the cause of the affection is at present unknown.

W. PASTEUR.

FISSURE (*findo*, I cleave).—A narrow and superficial crack or solution of continuity, observed on the skin and mucous membranes, especially near the line of junction of these structures, as on the lips and within the anus. See ANUS, Diseases of; and CHAPS.

FISTULA (*fistula*, a pipe).—A narrow track or canal leading from a free surface, and extending more or less deeply to some seat of local irritation; or it may be constituting an abnormal communication between two or more cavities, as in the case of vesico-vaginal or recto-vaginal fistula. See ABSCESS; and SINUS.

FISTULA IN ANO.—See RECTUM, Diseases of.

FIT.—A popular synonym for a sudden seizure characterised by loss or disturbance of consciousness from any cause, with or without convulsions (see CONVULSIONS; EPILEPSY; HYSTERIA; and SYNCOPE). The term is also applied to a sudden or acute seizure of certain diseases, such as gout, asthma, and ague.

FLATULENCE.—Flatulence is the presence of an excessive amount of gas in the alimentary canal. Carbon dioxide is the gas expelled in greatest quantity; but nitrogen, oxygen, marsh-gas, sulphuretted hydrogen, and hydrogen may also be found.

SOURCE OF GASES.—(1) *Carbon dioxide* may be formed in several ways. The most important of these is the decomposition of carbohydrates, the digestion of which is delayed in the stomach. Small quantities are not improbably formed during the production of lactic acid; but it is when the lactic acid is becoming converted into butyric acid that the largest amount of the gas is given off; it then acquires the odour characteristic of butyric acid. The action of the acid gastric juice on the carbonates

in the food and saliva and on the alkaline contents of the duodenum may give rise to the formation of gases in either stomach or intestines, as the case may be. A third source of carbon dioxide is from the blood. Some writers believe that large quantities of the gas may be given off, especially in neurotic persons, by a process which constitutes a kind of gastro-intestinal respiration; but the existence of such a mode of gaseous secretion is still doubtful.

(2) *Nitrogen* and *oxygen* are generally found in small quantities, and are derived from atmospheric air swallowed with the food and saliva. In neurotic persons irregular action of the pharynx and oesophagus may lead to the swallowing of large quantities of air which is subsequently eructated.

(3) *Marsh gas*, *sulphuretted hydrogen*, and *hydrogen* are invariably products of the decomposition of food, and when present are generally formed in the intestine, although they may regurgitate into the stomach. They are probably due to the putrefactive decomposition of meat, and to the action of butyric acid on cellulose. In some cases of neurotic intestinal flatulence, considerable quantities of these gases, especially of sulphuretted hydrogen, may be present. In many of the cases the flatulence is developed very rapidly and is obviously due to an emotional cause.

The above statements may be thus summarised. Carbohydrates are the chief source of flatulence, especially when the muscular movements of the alimentary tract are deficient, and when there is any permanent dilatation of the stomach or any other part of the canal. The composition of the large quantities of gas expelled in neurotic cases varies, and the exact mechanism of its production is unknown.

SYMPTOMS.—These depend upon the retention or expulsion of gas rather than on its formation. A sensation of fulness and varying degrees of discomfort may be experienced, or there may be actual distension of the stomach or intestines, giving rise to the physical signs ordinarily associated with these conditions. Irregular movements of the distended parts may cause epigastric pain and tenderness or abdominal colic and borborygmi, and the pressure exerted upon the heart may lead to palpitation and tachycardia. The site of the discomfort and the mode of passage of the flatus will in the majority of cases sufficiently determine whether the stomach or intestine is primarily at fault.

TREATMENT.—This can only be briefly summarised. For details the articles on **STOMACH** and **INTESTINES** should be consulted. The general principles of treatment are to reduce the carbohydrates of the diet to a minimum, and to improve the action of the muscular walls of the stomach and intestines. In the majority of cases more time and more teeth are required to render mastication efficient; fluids, especially at the commencement of a meal, should be avoided; sugar should be forbidden; potatoes and farinaceous foods should be taken sparingly; even wheat, preferably in the form of thin toast or biscuits, should only be permitted in small quantities, and at the beginning of a meal rather than at its close; vegetables containing much cellulose should be forbidden, especially if they cannot be obtained perfectly fresh; and all articles specially productive of flatulence in the individual case must be carefully ascertained and rigorously excluded. The so-called gastric and intestinal anti-

septics are sometimes valuable adjuvants. Among these may be mentioned sulpho-carbolate of sodium, the salts of bismuth—especially the subnitrate and salicylate—creosote, terebene, salol, and perhaps charcoal. Any tendency to constipation must be corrected.

When the presence of gas is causing pain or discomfort its expulsion may be facilitated by gentle kneading of the abdomen, and by the administration of one or more of those drugs grouped under the heading of *carminatives*. Neurotic flatulence is more difficult to remove. The treatment is similar to that needed in other neuroses. See **STOMACH**, **Neuroses of**.
H. MONTAGUE MURRAY.

FLEXION (*flecto*, I bend).—A bending. This term is applied either to the act of more or less forcible flexion, as in some methods of treatment, for example in the cure of aneurysm or the reduction of dislocations; or to the condition in which parts are bent, as the result of disease or of disorder, as when the limbs or certain internal organs are bent upon themselves. See **UTERUS**, Diseases of.

FLEXOR RESPONSE.—See **BABINSKI'S SIGN**.

FLINSBERG, in Silesia.—Chalybeate waters. See **MINERAL WATERS**.

FLOATING KIDNEY.—See **KIDNEYS**, Malposition of.

FLOCCITATION (*floccus*, a lock of wool).—Picking at the bed-clothes. A common symptom in typhoid fever and delirium tremens.

FLOODING.—A popular term for excessive discharge of blood from the womb. See **MENSTRUATION**, Disorders of; and **PREGNANCY**, Disorders of.

FLUCTUATION (*fluctus*, a wave).—A physical sign consisting in a wave-like or undulating sensation (*fluid thrill*). It is elicited by palpation with the one hand, while percussion is made with the fingers of the other; and is due to the presence of a fluid in a natural cavity, such as the peritoneum, or in an abnormal closed space, such as a cyst (see **PHYSICAL EXAMINATION**). The term 'fluctuation' as used by the surgeon has a somewhat different signification, being applied to the sensation of the presence of a fluid which may be felt when alternate pressure with the fingers is made, as over the seat of an abscess. See **ABSCESS**.

FLUKE.—See **ENTOZOA**.

FLUX.—A flow or excessive discharge from a mucous surface, through any of the natural passages, of serum, blood, mucus, pus, or the various secretions. As illustrations of fluxes may be mentioned salivation, bronchorrhœa, biliary flux, diarrhœa, dysentery or 'bloody flux,' cholera, and leucorrhœa or white flux (*fluor albus*).

FŒTUS, Diseases of the.—The incidence of morbid causes upon the fœtus produces morbid conditions which differ from the diseases of childhood and adult life only in degree, while the embryo when brought under similar influences becomes malformed or monstrous and shows conditions utterly unlike any that arise in post-natal life. At present we consider only the diseases of the fœtus, fœtal pathology. (For embryonic pathology, see **MONSTROSITIES**.)

The existence of the fœtus may be described as one of protected parasitism. It is life in a fluid medium of high and practically constant temperature, in the dark, and with almost complete protection from external violence and from microbic invasion. The peculiarities of the environment influence the development of maladies and give them distinctive features. Most of the organs of the fœtus are quiescent during intra-uterine life, but the heart and the placenta (which is in part a foetal organ although extra-corporeal) are very active and of supreme importance in the well-being of the unborn infant. It is through the placenta that foetal parasitism is established and maintained; consequently placental lesions are lethal or disease-producing in their effects upon the fœtus. The fœtus may suffer (1) from diseases and toxicological states transmitted to it from the parents, (2) from idiopathic maladies, (3) from the effects of traumatism, (4) from tumours, or (5) it may die *in utero* (foetal death).

1. Transmitted Diseases.—Many diseases from which the mother is suffering at the time of pregnancy may be transmitted to her fœtus *in utero*. The route is in most instances by the placenta, but in a few cases it is possible that the infection may pass into the liquor amnii, and thence, by the alimentary canal, into the foetal organism. The mechanism of the transmission of infection through the placenta is not yet fully understood, although much has been learned lately regarding the passage of microbes. It might be anticipated that the normal placenta would act as a barrier to bacteria and their toxins, and that some lesion of its structure would be necessary before their transmission became possible; but cases are on record in which the most careful scrutiny of the placenta showed no lesion either macroscopic or microscopic, and in which germs were nevertheless found in large numbers in the fœtus. Possibly chemical changes in the blood or in the placental tissue may explain these results. When the placental barrier has been passed the infection will travel to the fœtus by way of the umbilicus and so reach the liver and the right side of the heart before the other viscera; in this way may be explained the frequency of hepatic and cardiac lesions in the fœtus. It may be mentioned here incidentally that there is some evidence to show that the current of infection through the placenta may be occasionally reversed, and that morbid conditions in the fœtus may pass to the mother.

Among the diseases which may pass from mother to fœtus the *infectious fevers* occupy an important place. Small-pox is a case in point. When a pregnant woman takes variola it would appear that various consequences to her fœtus may follow: it may take small-pox and be born alive or dead with manifest signs of the disease (in any stage) upon it, or it may apparently be infected and pass through the various stages of the fever *in utero*, recovering perfectly, and be born later with the cicatrices upon it, or with scarcely any trace of them at all, for the presence of the liquor amnii prevents much pitting. In yet other cases the only indication that the fœtus has suffered with the mother may be immunity to vaccinia exhibited when the infant comes to be vaccinated. Some curious occurrences have been reported, such as infection of one fœtus in the case of twins, and the birth of a fœtus affected with small-pox when the mother herself has not taken the disease but has been exposed to the infection. In the latter case

it must be supposed that the mother had either a natural or an acquired immunity to variola, and that the infection passed through her blood to her unprotected fœtus. Vaccination of the pregnant woman does not always give immunity to the infant *in utero*, but it does so often enough to make it the right treatment to adopt in the case of all pregnant women during the prevalence of an epidemic of small-pox. Besides variola, other fevers may be transmitted from mother to fœtus. Measles and scarlet fever have both been observed, although with comparative rarity, for measles is uncommon during reproductive life, and scarlet fever does not often immediately precede the puerperium. Typhoid fever in the fœtus has only recently been recognised; it differs from the same disease in the adult in that intestinal and splenic lesions are rare, but it agrees in the presence of Eberth's typhoid-bacillus in the liver and blood, in the possibility of obtaining pure cultures from the organs, and in the presence of the agglutinating reaction of the blood-serum. Varicella, yellow fever, and even epidemic cerebro-spinal meningitis have all been recorded in the unborn infant. It would seem also that the hæmatozoon of malaria can at times pass the placenta, for foetal malaria has been observed; and the fœtus may have malaria of the same or of a different type from that of the mother. Foetal rheumatism has been noted. Other diseases than the fevers may pass to the fœtus. Foetal *syphilis* is the best known of the transmitted foetal maladies. It may pass from the mother to the fœtus, either by the placenta or perhaps directly through the ovum; and it may come from the father by the spermatozoon. It shows itself by osteochondritic changes in the long bones at the junction of the diaphysis with the epiphysis, in small-celled infiltration of various organs, especially the liver and spleen, and sometimes in pemphigus and other affections of the skin. Foetal *tuberculosis* is rare; it must be very far advanced and generalised in the mother before it can affect the fœtus, but when the bacilli have reached the foetal tissues they multiply there with great freedom. Owing to peculiarities in the foetal environment and to the fact that the infection reaches the fœtus through the umbilicus, it may not be easy always to recognise that the foetal malady is derived from the maternal. Thus in a case of maternal erysipelas, the fœtus was found to have died from streptococcic endocarditis; evidently the microbes had passed from the mother's skin into her vascular system, and through her placenta to the foetal heart, and become active on the cardiac valves. The occurrence of foetal diabetes in cases of maternal diabetes has been suspected from the discovery of sugar in the liquor amnii; but it is doubtful whether the conclusion is warranted.

Toxicological conditions as well as diseases may pass from mother to fœtus. It has recently been shown that alcohol introduced into the stomach of the pregnant woman or animal soon finds its way in large amount into the foetal blood and tissues; and it is well known that foetal death and prematurity of birth as well as congenital debility are common in the progeny of the female inebriate. Other poisons, such as lead and arsenic, may pass the placental barriers and may produce evil effects upon the unborn infant.

2. Idiopathic Diseases.—A large number of morbid foetal states must still be grouped under the unsatisfactory title of idiopathic. Prominent among them are the foetal diseases of the skin.

Fœtal *ichthyosis* may be met with in two forms, a grave type and a comparatively mild or benign type. The former is a most deforming hyperkeratosis and is practically never compatible with an extra-uterine life of more than a few days. Through an enormous proliferation of the epidermic layer of the skin the whole body of the fœtus is covered with numerous thick scales separated from each other by fissures and furrows, and the resulting condition has been compared to the skin of the alligator or to the dress of the harlequin. The mouth, eyes, and anus are in a state of ectropion on account of the surrounding hyperkeratosis. The mild or benign type is characterised by the presence of a more or less continuous coat of a collodion-like substance over the body, which soon after birth separates in the form of flakes like tissue-paper; sometimes it also is accompanied by some ectropion of the body-orifices. It is not fatal to life, and individuals suffering from it have been known to reach old age. It may be kept in check by means of alkaline baths and friction with glycerin or lanolin. A fœtal condition to which the name of *ichthyosis linearis neuropathica* has sometimes been given is better described as neurotic papilloma or as a variety of nævus than as a form of ichthyosis. It is characterised by the presence of papillary growths more or less localised and limited to the area of distribution of one or more of the cutaneous nerves. It generally affects one side of the body only. *Tylosis palmæ et plantæ* is another form of epidermic hypertrophy met with at birth; it is limited to the skin of the palms and soles, and is often very clearly hereditary. It has shown improvement under painting with a solution of salicylic acid in ether (five per cent.). Sometimes a fœtus is born alive with active desquamation of the cuticle, resembling the changes in the epidermis in maceration of the dead fœtus; this fœtal *keratolysis* may be due to post-maturity, to such fevers as measles or scarlet fever, to a mild degree of ichthyosis or sepsis, or to syphilis. It is to be distinguished alike from the physiological desquamation of the new-born and from the condition known as Ritter's disease, or dermatitis exfoliativa neonatorum. Little is known of the condition at birth of the individuals called hairy men, 'hommes-chiens,' &c. They suffer from generalised *hypertrichosis*, but whether the hair present is a persistent lanugo or a new formation after birth is not certain. The condition has in some cases been transmitted from father to son. The opposite condition, *hypotrichosis congenita*, is also met with; and both hairiness and congenital alopecia may be associated with dental defects. The various forms of *nævus* (pigmentary and vascular) are well-known congenital anomalies of the skin, and when small may occasionally be cured by vaccination on the affected region. *Absence of the skin* in localised areas, sometimes symmetrical, has been seen at birth; it is a real absence of the layers of the skin, and is now generally believed to be due to pressure of the amnion or to adhesion and subsequent tearing away of bands of the amnion which have formed between the skin of the fœtus and the innermost of the fœtal membranes. It is well to remember that *pemphigus* of a non-syphilitic nature may be met with in the fœtus at birth; this bullous formation may be due to sepsis or possibly to an hereditarily transmitted tendency to separation of the cuticle.

Morbid conditions of the subcutaneous tissue as

well as of the skin may occur in the fœtus. Of these the best known is *general fœtal anasarca* or *dropsy*. In it the subcutaneous tissue all over the body is dropsical, and to such a degree that the fœtus becomes truly monstrous in appearance; the body-cavities also, and especially the thorax and abdomen, are full of fluid, and the placenta is cedematous. The mother is not uncommonly dropsical also, and may suffer during pregnancy from renal disease, from anaemia, or from hepatic or cardiac trouble. Hydramnios is frequent, and the condition may affect several children of the same parents (*family prevalence*). General fœtal dropsy is probably a symptom of various morbid states rather than a distinct disease in itself; it may apparently be due to premature closure of the foramen ovale, absence of the thoracic duct, cystic kidneys, or fœtal leucæmia; in other instances the maternal condition is regarded as a sufficient explanation, the effect of backward pressure from the mother's vascular system causing dropsy first of the placenta and then of the fœtal tissues. It is specially common to meet with fœtal dropsy in the malformed twin, and particularly in the acardiac parasitic fœtus. *Congenital cystic elephantiasis* is a condition closely resembling general fœtal dropsy, especially that which occurs in the monstrous twin. In the subcutaneous tissue are numerous cysts with serous or curd-like contents. The whole body or only a special region (e.g. the head and neck) may be affected; the condition is due to a dilatation, or to a dilatation and occlusion of the lymphatic spaces and vessels. Both general fœtal dropsy and general cystic elephantiasis are incompatible with prolonged extra-uterine life. To other morbid states the name fœtal or *congenital elephantiasis* has been given. These are less fatal to life after birth. Among them is the firm thickening of the subcutaneous tissue and skin of one region of the body, such as the leg or arm, which sometimes is associated with the presence of amniotic bands, and sometimes with the occurrence of maternal lymphangitis and the passage of streptococci through the placenta. It may be accompanied by subcutaneous fibromata or by areas of nævus-like vascularity; and it may occasionally be exactly limited to one side of the body (*congenital hemihypertrophy*). It has been treated, sometimes with success, by elastic pressure, electricity, massage, and iodide of potassium.

Several diseases of the alimentary system have been met with in the fœtus. There may, for instance, be *congenital ascites*, a morbid state which has considerable obstetrical importance as a cause of great delay in labour, a delay which is sometimes so great as to necessitate the puncturing of the fœtal abdomen (justifiable fœticide). Hydramnios and malformations of the genito-urinary organs are fairly frequent concomitants. Unlike ascites in later life it is rarely due to obstruction in the portal circulation, and most commonly to pre-existent peritonitis. *Fœtal peritonitis*, however, is not always accompanied by effusion; when it is of the 'dry' type it is supposed to lead to malformations and delayed developments of the genital organs, and to the formation of adhesions between them and the intestines, thus predisposing to disturbances in later life (postponed effect of antenatal morbid states). The co-existence of fœtal peritonitis and *distension of the fœtal bladder* has been noticed several times; sometimes a valve or an occlusion has then been found in the course of the urethra, but as often no such obstruction has

been discovered. *Congenital obliteration of the bile-ducts* is another morbid state which has been ascribed to foetal peritonitis; it leads to jaundice of the new-born infant, and is probably always fatal a week or a fortnight after birth; it may show family prevalence. *Distension of the foetal ureters and hydro-nephrosis* occurs sometimes in association with distended bladder and sometimes quite apart from it; it may be traced to kinks or to valves in the ureters. *Congenital hypertrophy of the pylorus* may exist, and may lead to complete stenosis and death after birth with the symptoms of intestinal obstruction; but some of the less severe grades of hypertrophy may apparently be recovered from. *A congenital hypertrophy of the colon* and especially of the sigmoid flexure has been described, and to it has been ascribed one form at least of congenital constipation; but it seems more probable that the constipation has originated in some malformation or malposition of the bowel, and that the hypertrophy of the walls is secondary.

Idiopathic foetal maladies of the respiratory organs are very uncommon; but *septic pneumonia* has been met with at birth. It may have arisen from the transmission of streptococci through the placenta from the mother (then falling into the group of the transmitted maladies); or it may have been due to a sucking into the lungs of infected material while the foetus was on its way through the maternal passages during birth (intranatal infection).

It is difficult to separate the foetal *cardiac diseases* from the malformations, for it is common to find perforations of the inter-ventricular and inter-auricular septa and patent foramen ovale in association with the results of foetal endocarditis; indeed some regard the malformations as the consequences of the endocarditis. At the same time there can be separated the cases in which the heart is normally constituted but shows lesions of the lining membrane and valves resembling those which are commonly acquired in later life. To this group belong pulmonary and aortic stenosis and obliteration, with or without cardiac hypertrophy and dilatation; the right side being most affected. Foetal cardiac diseases and malformations are among the few antenatal maladies which have been diagnosed before the birth of the infant suffering from them. A foetal heart murmur has been detected by the careful auscultation of the maternal abdomen, and the diagnosis has been confirmed after birth. It has sometimes occurred that the murmur disappeared about ten days after birth, and it has been concluded that the lesion affected the ductus arteriosus or the foramen ovale and that the closing of these openings caused the disappearance of the murmur.

Foetal *bone-diseases* are very little understood, although a vast amount of investigation has taken place. Under the names foetal rickets, osteogenesis imperfecta, chondrodystrophia, and achondroplasia, conditions have been described in which there are commonly friability of the bones, generally of the long, but also sometimes of the flat, and either excessive or defective production of bone in the diaphysis or epiphyses. That the condition produced is rickets is more than doubtful, but true rickets is occasionally met with at birth. In what has been called achondroplasia the bones of the head and trunk are normal or nearly so, while those of the limbs are much stunted, giving rise to a form of dwarfism; this malady is not necessarily fatal to extra-uterine life—in fact an achondroplastic mother

has been known to give birth to an achondroplastic infant. See ACHONDROPLASIA.

3. **Results of traumatism.**—It may be safely concluded that the foetus does not often suffer from *traumatism* apart from the act of birth. Intranatal injuries from instruments, from the hands of the accoucheur, and even from the mother's pelvis are not uncommon, but these are not truly foetal. *Fractures* occur before birth, but are generally the result of unnatural friability of the bones, and the so-called congenital *dislocations* of the hip and knee, &c. are either produced in parturition or are due to some malformation. With regard to what have been called spontaneous *foetal amputations*, traumatism can only be invoked if we understand by it the slow influence upon the limb of a band of amnion or other constricting agency; these defects are more correctly looked upon as teratological or at any rate as on the borderland between the diseases and the malformations. At the same time it occasionally happens that gross maternal abdominal traumatism may determine foetal injuries, and cases have been reported in which a revolver bullet has penetrated the uterus and lodged in the foetal tissues.

4. **New-growths.**—The foetus has been known to suffer from practically every kind of *neoplasm* which may effect the adult, but well-authenticated cases of epithelioma are extremely rare. As might be expected, the tumours which are due to cyst-formation in embryonic ducts, which ought normally to disappear before birth, are commonly met with; under this heading come the various branchial, sacral, and coccygeal cysts, which are sometimes so large as seriously to impede labour. Teratomata or teratoid growths, such as the dermoids, are probably due to the inclusion of a twin embryo or to the displacement inwards of part of the epiblastic layer during the shutting in of the pleuro-peritoneal cavity; they are peculiar in that they contain an assortment of various tissues and even the recognisable rudiments of some organs. They are sometimes found attached to the sacral region or to the back higher up, and sometimes they are intra-abdominal (ovarian dermoids and *fetus in fetu*). Sarcomata of the kidney and rhabdomyomata of the vagina, heart, and sciatic nerve have been recorded among the foetal tumours. Small growths met with in front of the external ear are generally of the nature of fibro-chondromata, and are supposed to be connected with the rudiments of the branchial arches.

5. **Foetal death.**—The foetus may die *in utero*, and the causes of *intra-uterine death* are probably very numerous. They may be of the nature of maternal morbid states, such as acute febrile diseases (pneumonia, &c.), infectious fevers (variola, &c.), cardiac disease, nephritis, injuries, accidents, and syphilis; or they may be truly foetal maladies such as syphilis and fevers; or yet again they may be of the nature of paternal morbid states, of which little is known, but among which must be reckoned albuminuria, lead-poisoning, and syphilis. Diseases of the placenta are probably especially lethal to the unborn infant; they are often associated with pre-existing maternal endometritis and renal disease. Habitual death of the foetus has been described and may be associated with almost any of the above-named causes, but is doubtless specially connected with syphilis, alcoholism, renal disease, uterine displacements, and endometritis. If the foetus die between the third and fifth months of intra-uterine

life, the commonest *post-mortem* change is a drying up and shrivelling of the tissues without putrefaction (*mummification*), and if the dead fœtus be one of twins and the other keep on growing, then there is a flattening of the mummified one which is called in consequence the *fœtus compressus* or *papyraceus*. *Maceration* is the commonest result of foetal death after the fifth month; it is a non-putrefactive softening and liquefaction of all the foetal tissues; and it is accompanied by desquamation of the cuticle, by the formation of bullæ containing blood-stained serum on the skin, and by the presence of sanguinolent serum in all the body cavities, especially in the peritoneal and pleural sacs. True *putrefaction* is rare *in utero*, and probably is always due to rupture of the membranes and the entrance of germs into the uterus; it is accompanied by the formation of gases in the uterus (*physometra*), and by the signs of general blood-poisoning. *Calcification* of the dead fœtus apart from cases of tubal pregnancy is extremely rare, but has been noted. The *diagnosis* of intra-uterine death is a very difficult problem, for it must be based largely upon negative symptoms and signs, such as the absence of foetal movements, of the foetal heart-beat, and of continued increase in the size of the maternal abdomen. Other circumstances which may aid in diagnosis are retrogressive changes in the mammary glands, history of the previous occurrence of foetal death in the patient, the presence of some grave or acute maternal disease, the disappearance of the purple discolouration of the vulva and vagina, a feeling of weight and cold in the pelvis, and possibly the supervention of maternal acetonuria, or the passing off of albuminuria when it has previously existed. The birth of a dead fœtus is not specially delayed or dangerous, possibly because the tissues are softened, and because the involution of the uterus and the shrivelling up of the placenta have already occurred, and so shut off the ordinary route by which septic infection reaches the mother.

Since it is a difficult matter to diagnose the death of the fœtus, it follows that it is still more difficult to ascertain the existence of foetal diseases while the subject of them is still *in utero*. The occurrence of diseases, and especially of transmissible diseases, in the mother ought to make the medical man suspect their possible presence in the fœtus; the existence of hydramnios (which can be diagnosed) nearly always indicates a foetal disease or a monstrosity, but it is not possible to determine which; and the previous occurrence of disease in earlier pregnancies in the same mother should make their recurrence a thing to be looked for, since many foetal maladies show a tendency to repetition in subsequent pregnancies.

J. W. BALLANTYNE.

FOLIE A DEUX.—**SYNON.**: Communicated Insanity.—It is rare for the sane to become affected with mental disorder as a result of contact with the insane. Nurses and attendants in asylums are not specially liable to become insane. Under certain conditions, however, constant association with an insane person seems to lead to the development of insanity in one previously sane. The person who is secondarily affected is usually of neurotic or insane inheritance, or somewhat feeble-minded. Women are more easily affected than men, so that most commonly a daughter is found to adopt the delusions of her mother, or a wife those of her husband, though occasionally the reverse is the case. The older patient usually affects the younger, and the stronger

in will the weaker, the second patient having generally been for a long time intimately associated with and under the influence of the first. Delusions of persecution are those most frequently thus conveyed, particularly delusions of conspiracy to deprive the patients of fancied rights, of poisoning, or of plots to injure in various ways, especially if there appears to be some slight ground for the suspicion, and where the conduct and ideas of the first patient are not at first sight markedly insane. Occasionally delusions of demoniacal possession or of poverty are transmitted in this way. One patient may persuade the other to commit suicide simultaneously. The insanity of the second patient is usually of less intensity and more curable than that of the first, though similar in other respects. Although most commonly there is only one person secondarily affected, yet instances are recorded of three or four or a whole family adopting the delusions of the first patient.

The more acute forms of mental disorder such as acute mania or acute melancholia are not as a rule transmitted from one person to another, though it is not uncommon for an individual to break down mentally while nursing an insane relative in an acute attack and thus suffering from intense anxiety in addition to physical exhaustion. Instances of a number of people becoming insane from the same cause are seen in epidemics of insanity, of which many have been recorded. Occasionally a patient in an asylum induces another to adopt his delusion, though this is rare. Twins sometimes become insane at the same time, but not necessarily as the result of communication. The practical application of these facts is that it is dangerous for people to nurse their insane relatives, and that if one person becomes insane from association with another who is already insane they must be separately treated.

R. PERCY SMITH.

FOLIE CIRCULAIRE (Fr.).—This term is applied by the French psychological physicians to a variety of insanity characterised by alternations of excitement and depression. The patient passes through an attack of mania of perhaps an ordinary character, but when he appears to have recovered he sinks into melancholia, and thence emerges again to become maniacal and excited. The duration of each stage may vary from weeks to months. Sometimes one state will follow the other immediately: in other cases a period of convalescence will intervene, during which the patient appears well, and can hardly be considered insane. Yet the prognosis is extremely unfavourable in all such cases; and it is of great importance, in estimating the extent of recovery of a patient, that it should be clearly ascertained that the attack is not one of a series following one another in the manner mentioned.

G. F. BLANDFORD.

FOMENTATION (*foveo*, I keep warm).—**SYNON.**: Fr. *Fomentation*; Ger. *Bähung*.

DEFINITION.—Fomentation is the application, to the surface of the body, of flannels, cloths, or sponges moistened with hot water, either pure, or containing some medicinal substance in solution.

ACTION.—The action of a simple fomentation is the same as that of a poultice. By its warmth and moisture it tends to relax the muscular fibres of the skin and soften the cuticle, thus relieving tension, and diminishing pain and irritation. In the early

stages of inflammation it favours resolution, by maintaining the temperature, and promoting active circulation through the area which has suffered from the injurious influence, which has started the process. In the later stages it promotes and hastens suppuration, by causing dilatation of the vessels, and encouraging exudation. A fomentation is superior to a poultice in lightness and cleanliness, but unless care be taken it loses its heat more quickly. This disadvantage may be overcome by covering the fomentation with a thick layer of cotton-wool.

APPLICATION AND USES.—A fomentation is thus applied: A stout piece of towelling, known as a 'wringer,' with a stick attached to each end, is put into a basin. A piece of coarse flannel, sufficiently large to cover the affected part when folded into four layers, is placed on the wringer, and boiling water is poured upon it. It is then lifted from the basin by means of the sticks, which are twisted rapidly in opposite directions, as much water as possible being squeezed out of the flannel.

This is then placed on the affected part and covered with a large piece of oiled silk or indiarubber sheeting, overlapping the flannel by at least one inch on each side. Over this may be placed a thick layer of cotton-wool, and a bandage. If the flannel be not squeezed sufficiently dry it will wet the bed or clothing. If not sufficiently covered with oiled silk and wool it soon becomes cold. Whatever means may be taken to retain the heat of a fomentation, it can be kept above the temperature of the body only for a few minutes. If, therefore, the full effect of fomentation is desired to be obtained, the flannels must be changed every twenty minutes. In many parts a sponge, or a piece of spongio-piline, wrung out of boiling water, forms a most convenient form of fomentation. When the fore-arm or hand is affected, a bath of hot water may be substituted for fomentations. The temperature of the water must be maintained by the repeated addition of small quantities of boiling water.

Fomentations are especially useful in all cases of erysipelas and diffuse cellulitis, and in boils. In peritonitis they are borne more easily than poultices, on account of their greater lightness. Whenever they can be employed they are superior to poultices, on account of their cleanliness. When used to cover discharging wounds or abscesses, a small dressing of boric lint or antiseptic gauze should be wrung out in hot water and placed over the wound to absorb the discharge, and changed frequently when the outer fomenting flannel or lint is renewed. In many cases it is advisable to make the entire fomentation of boric lint. In this way medicated fomentations may be applied with great relief to inflamed or suppurating wounds without fouling the cloths or flannels. Should they become contaminated they may be boiled in the water before using.

VARIETIES.—If it is desired to add some slight counter-irritation to the warmth and moisture, the fomentation may be sprinkled with turpentine before it is applied. This forms the ordinary *turpentine stupe*. The sedative action of the fomentation may be increased by sprinkling it with laudanum. The ordinary poppy-fomentation is often used with the same intention. It is thus prepared: Half a pound of poppy-heads with the seeds taken out is boiled for ten minutes in four pints of water, and the liquid then strained off. The decoction is kept warm over a fire, and the flannels dipped in it and applied as before described about every half-hour.

The term 'dry fomentation' is sometimes applied to bags of hot salt, bran, or chamomile-flowers; or pieces of flannel toasted before a fire and laid on hot. These often give relief in cases of intestinal, renal, or biliary colic.

MARCUS BECK.
C. J. BOND.

FOMITES (*fomes*, touchwood, tinder).—Substances capable of retaining contagium-particles, and thus of being the means of propagating any infectious disease. The most important fomites are bed-clothes, bedding, woollen garments, carpets, curtains, and letters. See INFECTION.

FOOD.—See ALIMENT; and DIET.

FOOD, POISONOUS.—Under certain conditions, various articles of diet may become possessed of poisonous properties. This may arise from a variety of causes, in addition to the introduction of known and specific poisons. Articles of food may become more or less poisonous from the following causes:—

1. *Flesh* may contain some poisonous substance administered as a drug or eaten as a food.

2. Poisonous substances may be derived from the *vessels* in which the food has been kept: for example, tinned provisions may become contaminated with tin derived from the vessel or solder; beer and cider which have stood in leaden pipes may become contaminated with lead.

3. Poisonous substances may be added by way of adulteration, as in the case of the colouring of preserved peas and olives with copper-salts.

4. Certain *kinds of foods* may develop poisons even in the fresh state: for example, meat and some kinds of fish, as mussels, salmon, and sardines.

5. Food may become poisonous from the development of poisons (such as ptomaines or alkaloidal poisons, and toxalbumoses or proteid poisons) as the result of fermentative or putrefactive changes in the albuminous constituents, produced by the agency of ferments.

6. Food may be poisonous from the presence of the germs or spores of certain specific or parasitic diseases, namely, enteric fever, diphtheria, and scarlet fever, tuberculosis, trichinosis, actinomycosis, hydatid-disease, and anthrax. Disease may be conveyed to man in this way, or the germs or spores of diseases may obtain access to food from exposure to sewer-air. See ENTOMOZOA.

7. The flesh of animals may be poisonous from the animals having fed on noxious or poisonous plants; and under this head may be classed poisonous honey which bees have gathered from poisonous plants.

Sound and unsound meat.—The obvious characteristics of good sound fresh meat are that its colour is red; it is marbled in appearance; firm and elastic to the touch; possessing a slight but not unpleasant odour, which is especially detected when a clean knife is thrust into the meat and withdrawn; and when exposed to the air for a day or two, it should neither become dry on the surface, nor wet, nor sodden. Sound meat is slightly acid to litmus paper; unsound meat may be neutral or alkaline. With commencing putrefaction the colour of the meat becomes pale, and the smell disagreeable, and later the meat softens in parts and turns green.

Poisonous Meat.—Cases of meat-poisoning have occurred from the ingestion of the following

foods: boiled ham, baked pork, boiled and salted pork, sausages, tinned pigs' tongues, roast beef, brawn, veal-pie, pork-pie, beef-pie, American ham, tinned ox-tongue, chicken-broth.

The symptoms exhibited are those of more or less severe gastro-intestinal disturbance, with those indicating various degrees of disturbance of the nervous system. The first symptoms of illness usually set in somewhat suddenly, at a varying period after the eating of the poisonous food; they consist of nausea, vomiting, abdominal pain, and diarrhoea (the stools being generally of a very offensive character), accompanied with a sense of faintness, muscular weakness, and feeling of prostration, which is sometimes very severe. Rigors may or may not occur. These symptoms are generally followed by fever and headache (which is often intense), and great thirst.

If the illness progresses, however, other nervous disturbances may be observed, such as cramps, muscular twitches, disturbances of vision, dilatation of the pupils, drowsiness, and occasional coma. The appearances observed in the organs of the body after death occurs are inflammatory, hemorrhagic, or destructive changes in the stomach and intestines; engorgement of the lung-tissues with blood; and inflammatory or destructive changes in the liver and kidneys. These phenomena must be regarded not merely as the result of local irritation, but of a general disease resembling in some of its effects the ordinary specific fevers.

This point was insisted on by the late George Buchanan in one of his Reports to the Local Government Board, in which he says 'that the phenomena which were spoken of as food poisoning are claiming on ever-growing evidence to be regarded as true infective diseases, as much so as was scarlet fever or tuberculosis; that they have not been generally admitted into this rank arises, first, from the circumstance that some of them have seemed to be wanting in the incubation period, and, secondly, because they are rarely recognised as being transmissible from person to person; while in the Middlesborough epidemic of 1888 we found suggestion of disease bacteria operating alternately through the atmosphere and through infection of food material by them.'

The Middlesborough epidemic that prevailed during the early part of 1888, and which resulted in 490 deaths during the year, in a population of 98,000, was an epidemic of pleuro-pneumonia due, at all events in part, to the consumption of American bacon, made by soaking in water, and then only slightly drying, salted pork imported from America. In this bacon there was discovered a bacillus which was capable of producing a specific general fever, the special characteristic of which was a pleuro-pneumonia.

From investigations of the cases of food-poisoning that have occurred with the various kinds of meat mentioned above, the following inferences may be drawn, viz.:—

1. In food rendered poisonous by keeping we find one or both of two things, namely, a living microscopic organism, and an organic chemical poison (ptomaine, toxalbumose, or toxin).

2. The material that is in all probability immediately operative in the production of morbid phenomena is the chemical poison, which is probably a product of the action of the micro-organism on the albuminous constituents of the food.

3. The micro-organism, provided its surroundings are favourable to its growth and activity, may produce its own special chemical poison from the material which affords its nourishment either outside the body or within it.

4. Both the micro-organism that produces the chemical poison in an infected food, and the chemical poison itself, may be evanescent; as on the one hand the micro-organism may be killed by its own products, and on the other the chemical poison may undergo destructive changes, so that the infected food, poisonous when eaten at one time, may fail to be poisonous when eaten at a later period.

5. In many cases of food-poisoning the incubation-period has been traced; in others it has been less obvious, and in some there is practically none. The symptoms which arise after the incubation-period are probably due to the operation within the body of the micro-organism, and the symptoms produced without an incubation-period (i.e. from half an hour to a few hours after taking the food) may probably be due to the operation of the organic chemical poison previously produced in the food. This inference is a fair one, since the micro-organism would require time, as in the other specific infections, for its growth and cultivation in the body, and for the formation of its poisonous chemical product; whereas the chemical poison previously produced in the article of food would operate more speedily, the rapidity of its operation being in proportion to its toxic nature, the amount taken, and the individual peculiarities of the recipient.

6. Of some of the animal foods mentioned as producing poisonous effects, Ballard has tabulated thirteen instances, in which the food was, or consisted largely of—

Pig's meat of one kind or another in 9 instances	
Butcher's meat (kind not stated) „ 2 „	
Veal „ „ „ „ „ 1 instance	
Beef „ „ „ „ „ 1 „	
Total . . .	13

This is probably a fair representation of the relative frequency with which swine's flesh gives rise to specific diseases of the kinds referred to, as compared with animal food from other sources. In connection with these forms of food-poisoning, the poisonous effect is not necessarily always due to ptomaines or toxalbumoses or toxins produced by a putrefactive decomposition of the article of food consumed. In some cases the symptoms and death are due to a true infection, extremely virulent bacilli having been found in articles of food, and also in the viscera of individuals who have died from eating such articles.

Some of these bacilli, as Klein showed in the Portsmouth pie-poisoning case, may not be pathogenic on inoculation, though when taken by the mouth or in cultures they may produce a chemical poison, which, received into the alimentary canal, produces illness and death, if in sufficient quantity.

The toxicogenic powers of these bacteria are largely influenced by the conditions under which they develop. The most important of these conditions are—

(a) The nature of the food infecting the body; (b) the temperature; (c) the amount of oxygen supplied; (d) the time which elapses between the infection and the consumption of the food.

The poisonous properties of some kinds of meat

and fish are due in some instances to the fact that the germs which they contain grow practically without any air-supply (as was in all probability the case in connection with the tinned-salmon poisoning case investigated in 1891 by the writer, and the tinned-sardine poisoning case investigated in 1892 by Stevenson). In such cases, in all probability, the contents of the tin were not sterilised, and, after sealing, the germs within the tin continued to grow anaerobically and elaborated a chemical poison.

On the other hand, several cases have been recorded in which canned meats were not poisonous when first opened, but soon became so on exposure of their contents to the air. In such cases, the meat in all probability becomes first infected after the opening of the can.

With regard to the nature of the chemical poisons formed in articles of food as a result of infection with pathogenic bacteria, three classes may be described:—

1. The ptomaines, or putrefactive animal alkaloids. See PTOMAINES.

2. The toxalbumoses or poisonous proteids, produced by bacterial agency. See ALBUMOSES.

3. The toxins or poisons of uncertain composition, also doubtless produced by bacterial agency.

PREVENTIVE AND CURATIVE MEASURES.—Good cooking, namely, exposure to a sufficiently high temperature for a sufficiently long time, is undoubtedly the best treatment—short of absolute destruction—of unsound and diseased meat. Smoking meat is less effective than cooking; salting, as a rule, is more effective than smoking, but there is evidence to show that smoking may merely hold the life of micro-organisms in suspense; for instance in the conversion of American salted pork into American hams, a specific germ (a bacillus) has been known to retain its harmful properties. The best precaution of all is cleanliness. Factories where articles of meat are prepared and tinned should be well ventilated, well lighted and clean; and the incursions of ground-air, sewer-air, or putrid emanations of any kind should be rigidly prevented. Kitchens and pantries should also be similarly cared for.

With regard to the curative measures for the results of eating poisonous food, these must be guided by general principles. The gastro-intestinal and nervous symptoms are to be treated, and the powers of the patient sustained until the poisonous matter is removed by excretion.

In many cases of meat- and fish-poisoning the following prescription for adults is a useful one, administered every three or four hours until the effects of the poison have passed away: Solution of the perchloride of mercury m xx ; iodide of potassium gr. v; chloral hydrate gr. v; carbolic acid gr. j; aromatic spirit of ammonia m xx ; chloroform water ad ʒj ; one dose.

In addition to the different forms of meat already described as producing poisonous symptoms, the following articles of food have also been known to produce toxic effects occasionally.

(a) *Tinned articles of food.*—Salmon, sardines, anchovies, ox-tongue, pigs' tongues, meat, cherries, apples, and peas.

Tinned cherries and apples have been known to produce poisonous effects from soluble tin-salts contained in them, produced in all probability by the malic acid present in the juice exerting a galvanic action upon the solder of the tins, carrying some of the tin into solution as a malate of tin (see

four cases of tinned-cherry poisoning investigated by the writer: *Brit. Med. Journ.*, April 12, 1890). Tinned and bottled peas sometimes contain copper-salts which have been added for the purpose of colouring them.

(b) *Fish.*—Cases of poisoning by fish, crustacea, and the various shell-fish of our islands are not infrequently met with. Generally it is the ingestion of crabs, lobsters, and mussels which produces such results. Symptoms of gastro-intestinal disturbance and nettle-rash are usual, but occasionally fatal results ensue from the ingestion of mussels. In the case of poisonous mussels, Brieger has shown the toxic effects to be due to a ptomaine which he has named mytilotoxine, and which is doubtless produced within the mussels by bacterial agency, the bacteria most probably gaining access to the mussels through the medium of sewage-polluted water—since it has been found that mussels gathered on shores polluted by sewage are not infrequently poisonous in their effects, and contain the toxic ptomaine; whereas, if laid for a few months in the open sea, they soon cease to contain it and lose their poisonous properties.

(c) *Milk.*—This important article of diet may produce symptoms of poisoning or disease in many ways: (1) If acid from lactic acid fermentation (due to the presence of the *Bacillus acid lactici*), it is frequently productive of flatulence, sickness, and diarrhoea in children. (2) Milk may contain the germs of typhoid fever, scarlet fever, diphtheria, and cholera. The germs of typhoid fever may gain access to the milk, either in water polluted by typhoid stools having been added to the milk or used to cleanse the milk cans, or by exposure of the milk to sewer-air. The germs of scarlet fever may be conveyed into the milk from the hands of a milker suffering from scarlet fever, or from cows suffering from a disease identical with, or closely resembling, human scarlet fever. Diphtheria is possibly conveyed from the cows themselves, and cholera through the medium of contaminated water. (3) Tubercle-bacilli may gain access to the milk from the tuberculous udders of cows, and if the milk is unboiled it is in this way easy to account for the high mortality of young children from tubercular ulceration of the intestines and tabes mesenterica. (4) Milk may be contaminated from the animal suffering from foot-and-mouth disease (so-called vesicular eczema of the mouth and interdigital spaces of the feet); if there are vesicles on the teats of cows suffering from this disease the virus may get into the milk, and a person consuming that milk may be attacked with fever, vesicular eruption on the throat and lips, and swelling of the lymphatic glands of the neck.

(d) *Cheese.*—In cheese that has undergone a peculiar fermentation a poison has been discovered by Vaughan, which is a ptomaine named tyrotoxinon (diazobenzene butyrate). This ptomaine is intensely poisonous, producing nausea, dryness of the mouth and fauces, a sense of constriction in the throat, vomiting, diarrhoea, and great nervous prostration. The symptoms usually pass off after the lapse of a few hours, but may end in death from collapse. Cheese containing this poison is not necessarily altered in appearance or taste. Butter and cream, as well as cheese, have given rise to ptomaine-poisoning.

(e) *Vegetables.*—Vegetables may become poisonous either from the development of poisonous matter produced by putrefactive changes, or from the addition of poisons, as in the colouring of peas with

copper, or from the growth within them of fungi. For instance, actinomycosis, a disease which occasionally occurs in cattle and man, is now regarded as due to eating raw barley or other cereals upon which the ray-fungus has grown. See ACTINOMYCOSIS; ERGOTISM; and MUSHROOMS, Poisoning by.

(f) *Water*.—Drinking-water may produce poisonous effects, either from the presence of specific micro-organisms (generally from pollution of the water with sewage), or from contamination of the water with metallic poisons, such as lead and copper.

(g) *Beer*.—Beer may be rendered poisonous by the presence of arsenic, as in the widespread epidemic of arsenical poisoning that occurred in Manchester, Liverpool, and other Midland towns in 1900. The arsenic in the beer was derived from impure sulphuric acid, which had been used, by one particular firm, in the manufacture of the brewing-sugar.

ARTHUR P. LUFF.

FORAMEN OVALE, Patency of.—See HEART, Malformations of.

FORCIBLE FEEDING.—Forcible feeding is frequently resorted to when a patient from some cause or other does not take food, and is commonly used in the treatment of insanity.

Melancholia is the most common type of mental disorder in which food is refused. The maniac may have also to be fed in this way. In cases of delirium it is most important not to put off forcible feeding too long, as fatal exhaustion may rapidly supervene. Sordes about the lips and mouth, and a dry furred tongue with mental disorder in no way contra-indicate liberal diet, and are often the warning note that forcible feeding must be resorted to if a patient is refusing food.

METHODS.—The various means of artificial feeding at our disposal are: (1) Feeding-cup; (2) nurse feeding patient by means of a spoon, as with a child; (3) nasal tube with funnel attached; (4) large œsophageal tube with funnel attached; (5) rectal feeding. We need not refer to the first two forms, and rectal feeding is of little value in the treatment of the insane.

Feeding by nasal tube is done by passing a long soft red rubber tube (size No. 11–12), well lubricated with oil, through the nose into the œsophagus. It will usually be found that one side of the nose allows a passage more readily than the other owing to deflection of the nasal septum. The *advantages* of nasal feeding are that fewer nurses are required to hold the patient, and that no gag is wanted, therefore the risk of injury to teeth is avoided. Regurgitation or vomiting is more difficult to effect, and less liable to occur spontaneously than when the tube is introduced through the mouth. The *disadvantages* are more numerous, but none are of a serious character. Nasal feeding takes a longer time than œsophageal feeding. The tube being smaller is easily blocked up by mucus or thick food. If a patient is shouting during the passing of the tube it readily goes into the mouth or larynx: in the latter case the accident is quickly recognised by the stridor which it causes. Nasal feeding, if long continued, may lead to ulceration of the mucous membrane of the nose.

Feeding by the œsophageal tube.—With this method a gag is required, unless a patient has lost all his teeth. The serrated surface of the gag must be protected by means of rubber tubing. If there

is any difficulty in inserting the gag, the handle of a spoon may be used to separate the teeth. In using a gag great care must be exercised not to force the mouth open too rapidly against the powerful muscles of the jaw, otherwise the jaw may be dislocated or even fractured. The tube (No. 24–28) is then dipped into the food to lubricate it, and passed to the back of the pharynx; when the patient swallows, the tube is carried down into the œsophagus, and is then easily pushed into the stomach. The *advantages* of this method of feeding are, the rapidity with which the meal is given, and the readiness with which drugs like sulphonal can be administered. The *disadvantages* are that more assistance is required than with nasal feeding; that a gag must be used; that a patient with a small pharynx may become very cyanosed during the feeding, and that regurgitation can be easily effected by the patient. If a patient vomits fluids into the mouth, by the side of the tube, the tube and gag must be at once withdrawn to allow of the mouth being emptied, otherwise the food may be drawn into the air-passages and asphyxia result. When the requisite food has been given, the tube should be withdrawn during inspiration, as it is then less likely to produce vomiting. In the majority of cases nasal feeding should be recommended as the easiest and safest method.

PROCEDURE.—The recumbent position upon a mattress is the one commonly used, provided there are sufficient attendants to overcome easily any resistance, as violent struggling is a fruitful source of bruises, and even of broken bones. The head must be held firmly, care being exercised not to injure the ears. If an œsophageal tube is being used the nurse holding the head can also hold the gag in position. On no account must any attendant be allowed to kneel upon any part of the patient. The legs can be fixed by a long towel placed above the knees, the ends of the towel can be knelt on, or otherwise held firmly. Some authorities prefer to seat the patient in an arm-chair and fix his limbs to the arms and legs of the chair.

The food must be of a more or less fluid form and should consist of milk (3 pints daily), eggs (4 to 6 daily), soups, vegetable extracts, stimulants and salt. Peptonised foods are often useful. Each meal should be usually rather more than a pint in quantity. Patients should be fed three or four times a day, and if in a very weak state as often as every four hours.

MAURICE CRAIG.

FOREIGN BODIES.—See CONCRETIONS.

FORMICATION (*formica*, an ant).—An abnormal subjective sensation referred to the skin, which is described as of a ‘creeping’ character, and as resembling the crawling of ants upon the surface. See SENSATION, Disorders of.

FOURTH NERVE, Diseases of.—Morbid states of the fourth nerve are shown by *spasm* or *paralysis* of the superior oblique muscle, which it supplies.

Spasm.—Little is known of over-action of this muscle, except that through it is produced rotatory nystagmus when the quick movement of the upper end of the vertical axis is inwards. But it is then only the channel through which central disturbance is manifested.

Paralysis.—Paralysis is not uncommon. Its usual causes are inflammation of the nerve-sheath from

cold; hæmorrhage into the nerve; syphilitic affections of the nerve or of its sheath; cerebral tumours, &c., pressing on or injuring the nerve at its origin from the valve of Viessens, or in its course; aneurysm; exostoses or growths in the orbit; and degeneration of the nucleus, in common with the nuclei of the other nerves of the ocular muscles, from which it is occasionally paralysed in *tabes dorsalis*, in 'progressive external ophthalmoplegia,' in the rare acute inflammation of the nuclei, and in sudden lesions in this part of the mid-brain. It has also been known to be involved in the rare affection of the muscles of the eyeball in diphtheritic paralysis.

SYMPTOMS.—Even in complete paralysis of the superior oblique muscle there is little obvious deviation of the affected eye. Movement downwards is, however, defective, and therefore diplopia exists when the eye is moved below a line which runs obliquely downwards from the healthy to the paralysed side, through the point of mid-fixation. The uncertainty which results from 'defective projection' of the image formed by the affected eye occurs chiefly in looking down, and in the corresponding downward position of the head. It is common for the first discovery of the defect to be that the patient becomes giddy when he goes downstairs, and sees two flights of stairs before him instead of one. The chief visible defect in movement of the affected eye (examined alone) is downwards and inwards, because it is when the eyeball is moved in this direction that the superior oblique has most influence on the vertical position of the eyeball. In the direct downward movement there is slight convergent strabismus, because the inferior rectus lacks the counteraction to its inward traction, so that its tendency to rotate the globe (lower end of vertical axis inwards) is also unopposed and perceptible. The diplopia which exists when both eyes look down is homonymous—that is, the image formed by the affected eye is on the same side as that eye. The left eye (if affected) being higher than the right, its image (the left) appears lower than the right image. The action of the superior oblique being to move the upper end of the vertical axis of the eye inwards, there is, in its paralysis, an abnormal divergence of the upper ends of the vertical axes, and the double images (being always inverted) will converge; their upper ends being nearer together than the lower. This is due to the obliquity of the false image (e.g. the left), and this obliquity is greatest when the eyeball is moved to the left and downwards, because in this position the rotatory power of the superior oblique is greater, and the obliquity is least in looking inwards and downwards. Thus the convergence of the images is greatest when the difference in height is least, and *vice versâ*. When the paralysis of the superior oblique has existed for some time, a secondary contraction of the inferior oblique may cause crossed diplopia on looking upwards.

TREATMENT.—The treatment of paralysis of the fourth nerve is in the main that of its cause. When due, as it very commonly is, to syphilis, iodide of potassium in full doses, with or without mercury, is necessary. Small doses of mercury, with quinine, salicin, or iodide of iron, are also useful for rheumatic paralysis. Blisters to the temple are beneficial in the early stages. A little good, but not much, has been said to be produced by applying the constant current from the eyelid to the forehead, a few cells only being used. The direct stimulation of the muscle is impracticable.

W. R. GOWERS.

FRAGILITAS OSSIUM.—A diseased condition of the bones, in which they are extremely fragile, so that they are liable to fracture from very slight causes. See BONE, Diseases of.

FRAMBESIA, or GRANULOMA TROPICUM.—**SYNON.** : (Africa and West Indies) Yaws Fr. and Ger. *Pian*; Span. and Portug. *Bubas*.

DEFINITION.—A specific infectious chronic disease of various tropical countries, communicated by contact of the virus with a breach of the surface of the skin, and characterised by an eruption of squamous patches developing through a papular stage into diagnostic yellow encrusted granulation-tumours, which usually terminate in resolution without destruction of tissue.

GEOGRAPHICAL DISTRIBUTION.—The disease is endemic in tropical Africa and America and in certain of the Polynesian islands, and it is asserted to be prevalent in Ceylon, Malaya, and other parts of the Eastern tropics. A great deal of confusion has been created by assuming the identity of the disease with syphilis, tuberculosis of the skin, 'oriental sores,' and other similar ulcerative maladies that occur in the tropics. *Frambesia*, however, is essentially a non-ulcerative malady, the ulcerations sometimes seen in affected persons being the result of concomitant disease. In places where yaws is endemic it occasionally becomes epidemic. In several of the West Indian islands it has been necessary to establish special yaws-hospitals and to enact repressive laws to stay the ravages of the disease.

ÆTIOLOGY.—In most instances yaws is spread by the direct contact of the healthy with the diseased; but the contagium may be carried by flies and other insects, and it moreover clings to huts in which affected persons have lived in poverty and squalor. The disease is inoculable, and certain of the African and Polynesian races inoculate their children so as to secure immunity in adult age. The contagium is undoubtedly a microbe, and there is reason to believe that it is a micrococcus—having definite characteristics under culture—which is invariably found in the affected tissues. There is no racial immunity, the fact that most cases are seen among the dark-skinned races being due to their economic and hygienic conditions. Diet has no influence in the origination or spread of the disease—which cannot be transmitted by the parent to the offspring. No age exempts from the operation of the contagium, but the great majority of cases occur in children between five and fifteen years, the disease being hardly ever seen in early infancy. From an analysis of over 3,000 cases of yaws admitted into the special West Indian hospitals it was found that males and females were attacked in the proportion of three to two.

PATHOLOGY.—The raised squamæ and the papulæ are simply earlier and undeveloped forms of the granuloma, their bulk being composed chiefly of granulation-cells usually surrounding colonies of micrococci. On examining a section of a small granuloma under a low magnifying power it is seen that the papillæ increase in size from the circumference to the centre, and that they and all the deeper layers of the skin are infiltrated with masses of granulation-cells. At the summit of the tumour, the epidermis is absent, its place being taken by a thick crust of inspissated yellow secretion which dips down between the greatly enlarged papillæ. No

suppuration is found except in those cases in which the tumours have been subjected to much irritation. When the disease pursues its normal course to a cure the granulation-cells and the microbes gradually disappear, the papillæ decrease in size, and the skin—which for a time is maculated—resumes its natural appearance, no cicatrix being left to indicate the site of the tumour. During the height of the disease micrococci may be found in the absorbent glands and in the fibrous structures of the body. It would appear that the granulation-cells which form the bulk of the cutaneous tumours are simply barriers to cut off the microbes and to prevent the diffusion of their toxins.

SYMPTOMS.—The period of incubation is usually from ten to fifteen days, but it may be prolonged to six weeks. Frequently the onset of the eruption is marked by no apparent ill-health; but malaise, itching, pains of a rheumatic character, and fever may precede the eruption and last for a week or more—the pains persisting or recurring at intervals throughout the attack. The skin becomes harsh and dry, and round whitish raised patches of furfuraceous desquamation are seen about the body and limbs. These patches may appear as a general eruption, or they may be small and few, or by coalescence they may involve very large areas. Later on papule will form in some of the squamæ; and after a time, depressed yellowish spots of inspissated secretion will be seen at the summits of the papules. The papule increases in size in all directions and then forms the granuloma or so-called ‘tubercle’—which appears as a tumour growing out of the skin and covered at its flat or rounded summit with a cheesy-looking crust which at first is moist and bright yellow, but later on becomes dry and brownish like the soiled rind of cheese. The tumours vary in size from an eighth of an inch to several inches in diameter, and they are usually round or ovoid in shape. Rarely the crusts are absent, and then a pinkish tuberculated mass is seen. The granulomata spring from the true skin, but they may extend to mucous membrane, when they form about the nostrils, mouth, or anus. The last two orifices, by auto-inoculation, may become encircled with coalesced tumours which are devoid of crusts in consequence of the constant moisture and friction to which they are subjected. The granulomata in mild cases may be solitary, or they may appear in successive crops all over the surface of the skin. The eruption is often symmetrical and is oftenest seen on the exposed portions of the body. An examination of 100 successive hospital patients showed that it appeared on the face and head in 49 cases, on the trunk in 20 cases, on the genitals in 16 cases, on the perinæum in 20 cases, on the upper extremities in 29 cases, and on the lower extremities in 70 cases. Although the squama and papula are earlier stages of the fully formed eruption, many of them may persist throughout the attack, or appear and remain in their aborted forms at any time during its progress. Ordinarily in from a few weeks to two or three months the tumours shrink in size and the crusts become drier and thinner, until finally they fall off and disclose a whitish macula which rapidly becomes dark brown or black and persists for a considerable time. No cicatrix or puckering of the skin is left, and when the macule fades away there is nothing whatever to indicate the former site of the tumour. The duration of the disease is variable; the tumours may disappear under

appropriate treatment in from two to four months, but they may last for many months or even longer. Successive crops of the eruption may be evolved during the course of the disease, its relapsing nature being at times most marked. The lymphatic glands are frequently enlarged and tender, and the femoral glands may become enormously increased in size. Relapses occur in about 5 per cent. of the cases, and they usually take place within a period of six months after apparent cure. One attack usually produces immunity from a subsequent one; but, in a small proportion of cases, the immunity is lost and the manifestations of the second attack differ in no way from the first, no matter what time may have passed between them. In cachectic persons and in those suffering from malarial disease, ankylostomiasis, leprosy, syphilis, and tuberculosis, the granulomata often break down into intractable ulcers which at first may be covered with yaws-crusts. When the disease is concomitant with tuberculosis or tertiary syphilis, the ulceration at times spreads and attacks the deeper structures, so that all the tissues down to bone may be successively destroyed. Such cases are often seen in the West Indies; and, until recently, the ulceration was invariably ascribed to yaws. Should, however, ulceration supervene on yaws, it is due to intercurrent disease; and, therefore, its cause must be sought for and dealt with.

TREATMENT.—Yaws tends to spontaneous cure when the patients are placed under proper hygienic conditions, and many persons recover who bathe frequently in the sea and even drink sea-water. Indeed any interference with the course of the disease is likely to do more harm than good, and the chief aim of the medical man should be to strengthen the constitution. In the early stages remedies are recommended that will maintain a vigorous circulation in the skin, so as to favour the efflorescence of the eruption. When the full eruption has lasted several weeks, medicines may be given to cause the absorption of the cutaneous tumours; and iodide of potassium, arsenic, or perchloride of mercury may be administered with benefit. Should mercury be given, however, the utmost care must be taken not to produce salivation—which would probably result in disastrous consequences. Should there be any intercurrent disease, its cure should be essayed before the yaws is treated, otherwise the case may become an incurable one. As yaws is highly contagious, every effort should be made to isolate affected persons; and houses, clothes, bedding &c. used by the sick should be destroyed or thoroughly disinfected afterwards. It has been found that the infection often clings to the wretched huts of the native races most subject to attacks of the disease, and at times it may be necessary to demolish these hovels in order to safeguard the public health.

H. A. ALFORD NICHOLLS.

FRANCE, South of.—The eastern part (Mediterranean coast) is dry and bracing, with a very clear atmosphere. The chief resorts in it are CANNES, MENTONE, HYÈRES, and NICE. The western part is moist and mild but variable, the principal places in it being ARCAÇON, BIARRITZ, and PAU. See CLIMATE, Treatment of Disease by.

FRANZENSBAD, in Austria.—Alkaline sulphated waters. See MINERAL WATERS.

FRECKLES.—SYNON. : *Lentiginæ*; *lenticulæ*; Fr. *Ephélides*; Ger. *Sommersprossen*, *Sommerflecken*.—A freckle is an acquired increased pigmentary discolouration of a susceptible skin, involving a definite circumscribed roundish or irregular area of the rete Malpighii, in which melanin is deposited. It has received its Latin name from a resemblance in colour, figure, and size to a lentil. It varies in tint from yellow to olive; and is met with on the exposed parts of the skin, particularly the root of the nose, cheeks, neck, and dorsum of hands, and occasionally on the covered parts of the body ('cold freckles'), as the chest, back, and inner aspects of thighs. It is usually found in children and women in whom the skin is sensitive and delicate, and has obtained its German synonyms from its greater frequency in the summer season, when the affected areas are exposed to the chemical influence of the solar actinic rays.

Freckles are usually seen after the first decade (not in infants and seldom under the age of eight). They have been seen in mulattoes. Both sexes are equally liable, and freckles are specially common in those of fair complexion and with red hair. The lesions may be few or numerous, they sometimes coalesce, often persist through life, and are unaccompanied by subjective sensations.

The treatment of freckles is that of other cutaneous pigmentary affections. A favourite lotion is one of one grain of perchloride of mercury added to one ounce of Almond-mixture. Citric acid is sometimes useful. Meddlesome interference is the worst form of treatment. See SKIN, PIGMENTARY Diseases of; XERODERMA PIGMENTOSUM; and CHLOASMA.

JOHN HAROLD.

FRÉMISSEMENT CATAIRE (*frémissement*, purring; *cataire*, connected with a cat).—A physical sign felt on applying the hand over the region of the heart or great vessels in certain morbid conditions, and compared to the sensation conveyed to the hand by the purring of a cat. This sign is more commonly known as 'thrill' or 'purring tremor.' See PHYSICAL EXAMINATION.

FREMITUS (*fremitus*, a low noise).—A group of physical signs elicited by placing the hand over the respiratory organs while the patient speaks (*vocal fremitus*), or coughs (*tussive fremitus*); or in certain morbid conditions when the patient simply breathes (*rhonchal* and *friction-fremitus*). A fremitus may sometimes also be felt over the cardiac region in connection with the movements of the heart, when the surfaces of the pericardium are much roughened. Another form of fremitus is a peculiar sensation called *hydatid fremitus*, which may be elicited by a special mode of percussion over hydatid tumours in some cases. See PHYSICAL EXAMINATION.

FRICTION.—SYNON. : Rubbing; Fr. *Friction*; Ger. *Reibung*.

DEFINITION.—By friction we mean surface-rubbing, as distinguished from *shampooing*, *medical rubbing*, or *massage*, a process of manipulation by which deep pressure is made upon the muscles.

Friction is employed over the surface of a limb, or over the trunk, for a variety of purposes. It is especially useful when the circulation is enfeebled, either by the external application of cold, amounting, when in a severe degree, to frostbite, or in cases of paralysis. The effect is still further increased by the use of

various stimulating liniments, especially when it is desirable to excite counter-irritation over a large cutaneous surface, for the relief of congestion or inflammation of internal organs.

Friction is also employed in medicine to facilitate the absorption, and introduction into the system of various remedial agents applied externally. See ENDERMIC MEDICATION; and INUNCTION.

FRICTION-FREMITUS.—The form of fremitus produced by the rubbing together of surfaces roughened by various morbid conditions, as of the pleura in breathing, or of the pericardium from the movements of the heart. See FREMITUS; and PHYSICAL EXAMINATION.

FRICTION-SOUND.—A physical sign, heard on auscultation, and due to the rubbing against each other of serous surfaces that have lost their natural smoothness and moistness from any cause. See PHYSICAL EXAMINATION.

FRIEDREICH'S ATAXIA.—SYNON. : Hereditary Family Ataxy; Postero-lateral Sclerosis of the Spinal Cord; Diffuse Sclerosis of the Spinal Cord and Bulb.

This is a disease apt to show itself in several members of the same family, at periods varying between early childhood and the twenty-fifth year. It is about equally common in males and in females. It is not usually an hereditary disease in the strict sense of the term; it is rather a 'family disease,' showing itself in several children of the same parents, although neither they nor their parents may have had the disease. It is true that in rare cases one or more members of consecutive generations have been affected, but this is altogether exceptional, as owing to the early development of the disease marriage has rarely been possible either for male or female so affected.

NATURE AND PATHOLOGY.—When first recognised, this disease was regarded as a form of locomotor ataxy; but, as Charcot and Bourneville pointed out, some of the cases at least are more closely related to 'disseminated sclerosis.' Friedreich's disease is, in fact, a malady, as it were, intermediate between these two affections, having important alliances with each, though, for the most part, the clinical alliance is closer between it and disseminated sclerosis than between it and locomotor ataxy.

The disease is dependent upon the development of areas of sclerosis in the posterior and in the antero-lateral columns of the cord, as well as in some parts of the grey matter, more especially in the 'posterior vesicular tract' of Clarke; together with an extension of such morbid processes to the bulb, so as to implicate the cells of the post-pyramidal nuclei, of the hypoglossal nuclei, and other parts. The disease has, therefore, likewise been named 'Diffuse sclerosis of the spinal cord and bulb.' The posterior columns are most extensively involved from the lumbar region upwards, the crossed pyramidal tract and the direct cerebellar tract are also often very completely degenerated, while portions of the anterior columns and other portions of the lateral columns are more variously affected in different cases. The posterior nerve-roots are little if at all affected. The sclerosis is said to begin by an over-growth of neuroglia as a primary event rather than with primary atrophy of the nervous fibres themselves

followed by overgrowth of the neuroglia. Whether this be so or not, it is undoubtedly true that in some cases an altogether excessive amount of well-developed connective tissue has been met with in the affected regions of the cord in this disease.

SYMPTOMS AND COURSE.—Generally commencing without any special exciting cause, and mostly during childhood or adolescence, though occasionally later, up to the twenty-fifth year (and only very rarely later than this), the malady shows itself first by the occurrence of a gradually progressive weakness in one or both lower extremities, together with an unsteady or uncertain rather than a distinctly ataxic gait. The legs are often placed wide apart, and the walk is not unlike that of a drunken man or a patient with some form of cerebellar disease. By slow degrees the weakness increases, till standing becomes very difficult even with assistance, and at last impossible. The weakness and uncertainty soon extend to the upper extremities, which also become uncertain and tremulous during the execution of movements, as in touching the tip of the nose with the forefinger, or in picking up small objects. Occasionally this inco-ordination is not exaggerated by closure of the eyes. The uncertainty of movement is not nearly so exaggerated as that met with in the 'intentional tremor' of a well-marked case of disseminated sclerosis.

Later on, the trunk and the head become the seat of oscillations, which are exaggerated when the patient sits up or executes any movement, but, on the other hand, cease when he is completely at rest in the recumbent position.

The knee-jerks are usually soon lost, though in rare cases they have been rather exaggerated and associated with ankle-clonus. The plantar reflex persists, and is accompanied by well-marked dorsiflexion of the toes. In a case now under the writer's care, of three years' duration, the plantar reflex is very notably exaggerated, the slightest touch on the sole sufficing to produce rapid and well-marked dorsiflexion of the foot and toes.

As the disease becomes developed, sooner or later in different cases, two very important changes show themselves with considerable constancy, which are of great value from the point of view of diagnosis. The earlier of these to appear is a peculiar deformity of the foot (*pēs cavus*), which seems to be shortened and bent antero-posteriorly, the instep being much hollowed, while the toes are extended at the metatarso-phalangeal articulations (with prominence of the extensor tendons) and flexed distally.

The other change, of almost equal significance, is the development of a well-marked lateral curvature of the spine.

In males a condition of impotence supervenes; and in females the menstruation becomes exceedingly irregular. These signs are not long in showing themselves when we have to do with patients past the age of puberty.

After some years the tongue partakes in the trembling, and speech becomes hesitating, thick, and at last scarcely intelligible. Ultimately the tongue may become completely paralysed and motionless.

Nystagmus of a slow horizontal character is apt to show itself, on the occasion of voluntary movements of the eyeballs, though this is generally not of so well marked a type as that met with in disseminated sclerosis. There is no actual paralysis of ocular muscles. The pupils react to light and accommodation, and sight is usually good.

During the final stages of the disease there is often

more or less complete paralysis of all the limbs—usually associated with a generalised muscular wasting. At this period, also, the limbs are occasionally affected with cramps or transitory contractures.

Again, it is only during the later or final stages of the disease that affections of sensibility are prone to show themselves—and that, principally, in the form of anæsthesia of the lower extremities, sometimes so complete as to involve the joints and muscles as well as the skin. In the early stages there is little or no affection of sensibility of any kind. Rarely, pains are complained of, but they are usually slight and not of the lightning-like or lancinating type. The special senses and the intellect usually remain unaltered. The sphincters are not affected; and there is no tendency for bed-sores to occur.

Sudden attacks of vertigo are apt to supervene (and that quite irrespective of the position of the patient at the time); or towards the close of the disease actual apoplectic attacks may occur, to one of which the patient may succumb. Such an attack is said to be of the following type: 'It is characterised by a rapid but incomplete loss of consciousness, by resolution of all the limbs and a generalised anæsthesia, by a considerably impeded respiration which is of a jerking and noisy type, by a tumultuous action of the heart and great frequency of pulse (130), together with a notable elevation of temperature' (Brousse).

The development of the disease is often extremely slow, but always fatally progressive. It may last from five to thirty years.

DIAGNOSIS.—As already indicated, the disease has to be distinguished from disseminated sclerosis, and from locomotor ataxy. Such difficulties of diagnosis must, however, present themselves principally in regard to the first case of Friedreich's disease that happens to show itself in a family. If the medical attendant knows that already one or more of the patient's brothers and sisters have become affected in a similar manner, more than half the difficulty of diagnosis is at once got rid of; since several cases of ordinary locomotor ataxy or of disseminated sclerosis in the same family must be regarded as events of extreme rarity.

It should, however, be borne in mind that in Friedreich's disease (as well as in locomotor ataxy, and disseminated sclerosis) much variation exists in different cases, in the rapidity and in the order of development of the several signs and symptoms. A similar variability exists in regard to the relative development of these several signs in different cases—the result being that only a generic similarity is likely to exist between any two or three consecutive cases of this disease that may chance to present themselves to the same observer.

Supposing, however, that we have to do with a solitary case of disease, then the question of age becomes of first importance. If the patient should be under twenty years of age, the chances would be decidedly against locomotor ataxy, and in favour of the case being one either of Friedreich's disease or of disseminated sclerosis; while the indications would be still further against locomotor ataxy, if there should be an absence of the peculiar pains characterising this latter disease, or of other affections of sensibility.

The points, in detail, which must be taken into consideration for the diagnosis of these three diseases from one another will now be given in parallel columns.

LOCOMOTOR ATAXY.	DISSEMINATED SCLEROSIS.	FRIEDREICH'S DISEASE.
Rarely before twentieth year.	Often before twentieth year.	Commonly before twentieth year.
Affections of sensibility early and constant. Lancinating pains. Sight and hearing often affected.	Affections of sensibility often absent. No pains. Sight and hearing sometimes affected.	Affections of sensibility generally absent. No pains.
Double vision, and Argyll-Robertson pupil present.	Double vision frequent, and Argyll-Robertson pupil absent.	Double vision, and Argyll-Robertson pupil absent.
No nystagmus.	Nystagmus very common; often early and well marked.	Nystagmus frequent; often late and slight.
No oscillations of head and trunk.	Oscillations of head and trunk frequent.	Oscillations of head and trunk very common.
Knee-jerk absent; no ankle-clonus. No rigidities.	Knee-jerk commonly exaggerated, and clonus present. Rigidities common.	Knee-jerk usually absent, and no clonus. Rigidities rare.
No speech-defects.	Difficulties in speech.	Difficulties in speech.
Simple inco-ordination in arms common.	Tremors of arms, on movement, gradually becoming choreic in range.	Tremors of arms exaggerated by movement.
Gait ataxic.	Gait spastic.	Gait reeling and unsteady, of cerebellar type.
Mental disturbance rare.	Mental disturbance frequent.	No mental disturbance.
No deformity of feet or spinal curvature.	No deformity of feet or spinal curvature.	Pes cavus and lateral curvature of spine.

PROGNOSIS AND TREATMENT.—The disease is not amenable to any special line of treatment. On the other hand, it is one which is comparatively little dangerous to life, even though the sufferers may be helpless and bed-ridden for many years. Patients so affected may live for prolonged periods, if due care be taken to maintain their general health.

H. CHARLTON BASTIAN.

FRIEDRICHSHALL, in Germany.—Sulphated waters. *See* MINERAL WATERS.

FRONTAL SINUSES.—*See* NOSE, Diseases of.

FROSTBITE.—**DEFINITION.**—The result, varying in severity from temporary local anæmia up to gangrene, of exposure of a part to great cold.

ÆTIOLOGY.—The parts most liable to frostbite are the toes, feet, fingers, hands, nose, and ears—all because of their position, which involves special exposure, and a blood-supply readily affected by external influences. Most cases occur among men in the prime of life: firstly because their occupations involve special exposure; next because a drunken sleep is often the cause of prolonged exposure; and lastly, because in weakly individuals the general effects of exposure to cold are more often directly fatal.

GENERAL EFFECTS.—These vary with the intensity of the cold, the protection of the part, the general state of the patient, and other conditions. Tissues may be frozen to insensitiveness and yet recover perfectly; but if kept frozen, death of the affected part supervenes—more rapidly than when it is simply deprived of its blood-supply. If the part be frozen until it is absolutely dead, subsequent thawing will not be followed by the re-entry of blood; and the part will gradually undergo the changes characteristic of dry gangrene.

If actual death of the part has not occurred, the blood re-enters, and an inflammation—at first aseptic—ensues. Free exudation rapidly takes place, the part swells and shows all the signs of impaired circulation, vesicles with red or purple contents rise, and finally, if the part be seriously damaged, circulation completely ceases and moist gangrene is established—either limited and superficial, or involving a considerable part of a limb. In these cases the inflammation generally becomes septic from the growth of organisms from the skin into the damaged parts, and much tissue that might otherwise have been saved is destroyed. General infection may ensue.

SIGNS AND SYMPTOMS.—Usually the first sign is redness of a part coupled with some pain; this is followed, more or less quickly, by purplish lividity with diminished sensibility. Next, congelation takes place, and the part becomes white, hard, and absolutely insensitive. Then the freezing extends to the deeper parts until the part is literally 'frozen to death.' Signs of returning circulation, however slow, are the first signs of recovery; swelling, and often vesication, follow. Persistent pallor or a red or purple colour, which is not discharged by pressure, is a sign of ill-omen. A part which becomes cherry-red is said by von Bergmann to be doomed: the only doubt is how deep the moist gangrene which it precedes will extend.

In parts which do not die after frostbite, chronic ulceration and other trophic lesions are common for a long time after the injury. Thawing of a frozen part is acutely painful.

TREATMENT.—This needs exceptional patience and judgment: safe progress is always slow. For the general treatment of the frostbitten *see* COLD, EFFECTS OF.

The patient must be kept lying in a cold room of which the temperature is very slowly raised. The

affected part should be somewhat elevated and gently rubbed with snow or bathed with cold water. Next friction with vaselined hands may be used; and, finally, the part should be wrapped in cotton-wool, the rubbing being practised more or less frequently over an area much greater than that of the part apparently affected. Should swelling appear as the circulation is resumed, elevation must be increased until the vertical position is reached. Should vesicles be formed and the onset of moist gangrene be threatened, the part should be thoroughly cleansed, as if for an operation, wrapped in cotton-wool, and well raised.

Limited sloughing or intercurrent cellulitis should be treated in the usual way (*see* CELLULITIS). Should the gangrene be so extensive as to necessitate amputation, this should be performed, with the most careful asepsis, as soon as the line of demarcation is evident. A septic inflammation of the stump so often led to fresh sloughing that surgeons used to postpone amputation until the dead part was almost separated.

Impaired circulation and its results persisting after frostbite should be treated by attention to the general health and especially to the heart; by exercise, massage, and warm clothing.

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STANLEY BOYD.

FUMIGATION (*fumigo*, I smoke).—SYNON. : *Fr. Fumigation*; *Ger. Beräucherung*.

DEFINITION.—A mode of employing certain medicinal agents, capable of being volatilised by heat, the vapour being allowed to escape into an apartment, or to come into contact with articles of clothing and other objects, for purposes of disinfection; or being allowed to act upon the surface of the body for therapeutic purposes. The chief agents which are thus used are sulphur and mercury.

MODE OF APPLICATION AND USES.—*Sulphur*. The mode of using sulphur as a disinfectant is explained in the article DISINFECTION.

Mercury.—Mercurial fumigation, general or local, has long been employed in the treatment of syphilis, and many different preparations of mercury have been used in this way.

General mercurial fumigation is now usually effected by exposing the body to the fumes of calomel mixed with steam, the steam being added with the twofold object of inducing gentle perspiration, and of rendering the mercurial fumes less irritating to the respiratory passages. In the form of mercurial vapour-bath recommended by the late Henry Lee, the apparatus consists of a spirit-lamp which is enclosed in a case of wire-gauze, on the top of which is a small plate surrounded by a porcelain trough. One ounce of water is poured into the trough, and the lamp is then lighted. When the water begins to boil, from 20 to 60 grains of resublimed calomel are spread on the plate, and the apparatus is placed

between the patient's legs as he sits on a chair undressed, but surrounded by a blanket, or better by a moleskin cloak long enough to reach the ground. The cloak is tied round the neck, but kept away from the rest of the body by a cane hoop. If it be thought desirable that the vapour should be inhaled, the slit of the cloak may be opened slightly from time to time. The duration of each bath should not be longer than from fifteen to twenty minutes. The patient should not be left alone during the bath, as it sometimes causes faintness. When the calomel is exhausted, he goes to bed wearing the cloak as a nightdress. When any of the various bath-boxes or cabinets is used for fumigation the cloak of course is not required. The bath may be repeated every night, or less often, according to the case and the effect produced. The state of the gums must be closely watched, and the dose of calomel regulated according to circumstances.

General mercurial fumigation has been employed in all stages of syphilis, and it may be tried whenever mercury is not well borne by the stomach. But in the writer's opinion it is chiefly indicated in certain cases of wide-spread eruption which resist ordinary methods of treatment. *See* SYPHILIS.

Local mercurial fumigation has been found serviceable in treating obstinate affections of the skin and mucous membrane, in the later as well as in the earlier stages of syphilis. From 3 to 5 grains of calomel is the usual quantity for each fumigation.

ARTHUR COOPER.

FUNCTIONAL DISEASES.—A class of diseases in which no anatomical or structural change can be detected to account for their presence.

FUNGI, Diseases due to.—*See* PARASITES, VEGETABLE; MUSHROOMS, Poisoning by; and BACTERIA.

FUNGOID (*fungus*, a mushroom).—A term applied to superficial granulations and morbid growths, especially those of a malignant nature, when they sprout rapidly and assume an appearance somewhat like a mushroom. *See* CANCER; and ULCER AND ULCERATION.

FUNGUS-DISEASE OF INDIA.—*See* MYCE-TOMA.

FURFUR

FURFURACEOUS } (*furfur*, bran). — The

term *furfuraceous* is applied to conditions in which the epidermis is shed in the form of bran-like scales. It is especially applicable to such cutaneous diseases as pityriasis, psoriasis, and ichthyosis. Scurf and dandruff may be regarded as synonyms of furfur.

FURUNCULUS (Lat. a little rascal, dimin. of *fur*, a thief).—A synonym for boil. *See* BOIL.

G

GADFLY.—The popular name for a genus of insects whose larvæ infest man and the lower animals. See CESTRUS.

GAIT.—See EQUILIBRIUM.

GALACTAGOGUES (γάλα, milk; and ἄγω, I move).

DEFINITION.—Agencies which increase the secretion of the mammary gland.

ENUMERATION.—The most common galactagogues are: Mental Emotions; Local Nervous Stimulation; Warmth; good Food; Alcohol; Jaborandi; the fresh leaves of the Castor-Oil plant; and Tonics.

ACTION.—The maternal feelings, as well as the reflex stimulation of the infant's lips, act in developing the functions of the breast. Warmth and good diet also play their part in the process. Alcohol in the form of malt-liquors, and malt-extract, are useful adjuncts; and so are such tonics as iron, which counteract in some measure the severe drain on the constitutional resources. Jaborandi is a galactagogue of doubtful utility. A poultice made of the fresh leaves of the castor-oil plant, aided by teaspoonful doses of a fluid extract prepared from the same, is said to have a stimulating influence on the mammary secretion. The administration of the phosphates internally improves the quality of the milk in nursing women.

ROBERT FARQUHARSON.
SIDNEY PHILLIPS.

GALACTHIDROSIS (γάλα, milk; and ἰδρῶς, perspiration).—A term signifying milky perspiration. See SUDORIPAROUS GLANDS, Disorders of.

GALACTORRHŒA (γάλα, milk; and ῥέω, I flow).—An excessive flow of milk. See LACTATION, Disorders of.

GALL-BLADDER AND GALL-DUCTS, Diseases of.—A. BILE-PASSAGES.—

1. **Simple Catarrh.**—This common disorder is marked by an increase of the mucus in the bile-passages, and as the bile is secreted at a very low pressure the result is to retard and, if the catarrh be sufficiently great, to obstruct the flow of bile. Retardation in the flow of bile thus brought about is the cause of the condition termed 'biliousness,' evidenced by slight yellowness of the conjunctivæ; obstruction, more or less complete, is the cause of the commonest form of jaundice—'catarrhal jaundice.'

The catarrh may affect any of the bile-ducts from their commencement down to the opening of the common bile-duct into the duodenum. Its origin is referred by most observers to an extension of catarrh from the duodenum upwards into the common duct ('duodenal catarrh'); but a more common origin, in the writer's opinion, is extension from the intra-hepatic bile-ducts downwards ('excretory catarrh'). However produced, its commonest cause is the same, namely, irritation of the lining membrane of either duodenum or bile-ducts by the products of disordered digestion.

SYMPTOMS.—The symptoms are mainly those of the accompanying digestive disturbances, with

uneasiness over the liver or stomach, to which is superadded a yellowish discolouration of conjunctivæ and skin, with pale stools and presence of bile in the urine.

Catarrh of the bile-ducts—in many cases to a much higher degree—is also a common accompaniment of certain infective disorders: e.g. typhoid fever, pneumonia, malaria, and sporadic forms of enteric catarrh.

The symptoms in such cases are the same as the above, with, in addition, more general constitutional disturbance—including always some degree of fever. Even the simplest forms of biliary catarrh ('catarrhal jaundice') are often marked at the outset by some slight rise of temperature—a fact which suggests that even in their case infective influences in addition to those connected with digestive disturbances are not improbably at work.

There are no constant changes observable in the liver during life. In general, it is slightly enlarged. The duration of the symptoms is very variable, but averages from four to six weeks.

DIAGNOSIS.—The chief difficulty is in determining to what cause the catarrh is due, whether to some temporary disturbance of digestion, or temporary toxic influences (e.g. drain-poisons, as in butchers, sewer-men, &c.), or to a more permanent cause, e.g. the presence of organic disease or of gall-stones. The antecedent history of the case, the age of the patient, the absence of emaciation, will help in distinguishing the simpler forms from those connected with cancer, while the freedom from pain and the absence of any history of gall-stones will help in excluding gall-stones. In many cases the diagnosis is only made clear by the subsequent progress of the case. If due to simple irritation from digestive or toxic disturbances, the catarrh passes off completely; if due to organic disease, or if it be connected with the presence of bile-sand or gall-stones, it will persist or soon recur.

TREATMENT.—The chief indications are (1) to allay or remove any gastric catarrh; (2) to promote healthy conditions in the intestinal canal; and (3) to promote an increased flow of bile, with the object of flushing out the biliary system.

The first indication is met by removing all causes of gastric catarrh, and by appropriate use of alkalis (bicarbonate of sodium) or acids (nitro-hydrochloric); the second, by the administration of mercurial antiseptics (calomel, Blue Pill, or perchloride of mercury) with or without salines; and the third by allowing the patient to drink freely of mineral waters, of which the best are Vichy and Carlsbad, or simple water between meals or shortly before. These measures are always successful in cases of sub-acute catarrh, the result of indigestion. The task of treatment is more difficult when the catarrh is chronic, depending upon long-standing habits as regards eating, drinking, and insufficient exercise. It is the more important to remove this chronic catarrh, inasmuch as its long persistence is liable to favour the formation of bile-sand and eventually of gall-stones. See JAUNDICE.

2. **Infective Catarrh (Cholangitis).**—This affection is more serious than that just considered. It is the result of actual infection of the bile-passages

with micro-organisms, the chief being the *Bacillus coli communis* and the ordinary pyogenic organisms. It is an affection liable to complicate all septic processes occurring in the liver or in the portal veins, and it is specially liable to occur as a complication of gall-stones, whether these be situated in the gall-bladder or in the lower portion of the common bile-duct.

MORBID ANATOMY.—The changes induced are in the first instance those of catarrh, with accompanying lymphangitis; in severe cases the infection not only spreads along the lumen of the bile-ducts, causing desquamation of the epithelium, but also involves their walls and the neighbouring tissues. On section of the liver the bile-ducts present the appearance of small abscess-cavities. Not infrequently, the neighbouring branches of the portal veins are also involved by extension of the infective process with resulting pylephlebitis.

SYMPTOMS.—The chief symptom is *fever*, generally accompanied by rigors. In severe cases the fever assumes an intermittent character, the temperature frequently ranging as high as 104° to 105° F. The whole character of the attack closely resembles that of an attack of ague. These attacks may occur off and on over a period of years; they are always accompanied by more or less marked jaundice, which generally disappears between the attacks. In the first instance, they are probably the result of lymphangitis, but when they become severe and continuous, they denote the supervention of suppuration in the bile-passages—a very dangerous condition, causing death by acute toxæmia and exhaustion.

DIAGNOSIS.—The disease rarely occurs as an independent condition, but is usually preceded by a history of gall-stones or of long-standing biliary obstruction, the result of malignant disease. The supervention of fever of a remittent type in the course of these affections is always suggestive of suppuration.

TREATMENT.—The most urgent indication is to remove the cause, and as this in the great majority of cases is the presence of gall-stones, the risk of infective cholangitis is an urgent indication for surgical operation. In the early stages this may be carried out with every prospect of success, but when suppuration has ensued the chances are much less favourable. The gall-bladder should be opened and its contents removed; while free drainage should be established and maintained until the bile passes freely into the duodenum and the patient is free from fever.

3. **Cancer.**—Cancer of the bile-ducts, like cancer of the gall-bladder, is an occasional complication of gall-stones. It is generally of the cylindrical-celled variety. The most common site is the common bile-duct, either at its termination or at its point of junction with the cystic duct. The symptoms are those of slowly developing and persistent jaundice. The diagnosis cannot be made with certainty during life.

4. **Stenosis of the Common Bile-duct.**—This is a condition which may result from disease of the bile-duct itself, such as cancer, gall-stones, or cicatricial contraction resulting from the passage of gall-stones; or it may result from pressure of new-growths in the liver, glands, and, most commonly of all, in the head of the pancreas. The result is chronic and persistent jaundice with great dilatation of the bile-ducts.

B. DISEASES OF GALL-BLADDER.—The affections of the gall-bladder resemble in their mode of causation and general character those of the bile-ducts just considered—with, however, two differences: (1) that all the effects are greatly intensified, and (2) that they do not cause jaundice except by secondary implication of the ducts.

1. **Catarrh.**—This condition is very common, and always accompanies catarrh of the bile-ducts, although it may also exist as an independent condition. The temporary sojourn of the bile in the gall-bladder intensifies the irritant action of any toxic products it may contain; and when infection occurs, it also affords a favourable opportunity for the micro-organisms to develop their pathogenic properties. The degree of catarrh varies from slight congestion with an increased flow of mucus, to an oedematous and swollen condition of the wall of the gall-bladder.

CAUSES.—These are of three kinds: (1) *congestive*, such as are so frequently met with in the passive congestion of heart-disease; (2) *digestive*, including the various conditions due to disturbances of gastro-intestinal digestion—the commonest causes of catarrh of the gall-bladder; and (3) *toxic*, the last comprising the various effects on the wall of the gall-bladder exerted by the products of infective diseases, such as typhoid fever, pneumonia, diphtheria, influenza, and septic diseases generally.

SYMPTOMS.—The effects of the catarrh so produced are in all cases: an increase of mucus especially rich in cholesterol; increased desquamation of epithelium; and the tendency to the formation of small calculi, from precipitation of bile-pigment in an insoluble form (bilirubin-calcium). These generally form the nuclei around which the cholesterol in the mucus collects, and ultimately precipitates to form gall-stones. The conditions described may exist for many years without giving rise to any symptoms, or at most only to a sense of discomfort in the region of the gall-bladder in connection with the attacks of indigestion which so commonly mark the history of these cases. They only become marked when gall-stones are actually formed. *See GALL-STONES.*

TREATMENT.—This is similar to that already referred to in the case of simple catarrh of the bile-passages.

2. **Infective Catarrh.**—This is a more important condition than the one just described, and commoner than was formerly supposed. Normal bile has no direct antiseptic properties; but in health it is quite sterile. When, however, catarrh of the bile-passages is present, micro-organisms may readily enter, either from the blood or from the duodenum, and the bile thus become infected. This is of little consequence in the case of the common bile-duct, since this is periodically flushed with bile. The matter is different, however, with the gall-bladder, where the bile stagnates for a time, and in which organisms may remain, as observations show, for long periods. Infective catarrh so produced is one of the commonest causes of gall-stones. If the conditions be favourable, e.g. the temporary blockage of the cystic duct by tenacious ropy mucus or by small gall-stones, suppuration may occur (*suppurative cholecystitis* and *empyema of the gall-bladder*).

SYMPTOMS.—These are in the first instance as indefinite as those of simple catarrh; but unlike the latter they always tend to become more and

more marked. They include pain, slight fever, uneasy feelings over the region of the gall-bladder, varied from time to time by more painful attacks almost resembling those of biliary colic. These attacks are due to painful contractions of the wall of the gall-bladder.

TREATMENT.—Whenever the infective nature of catarrh of the gall-bladder is recognised, an early operation is advisable, in order to save patients from all the pain and dangers connected with the subsequent formation of gall-stones. On operating in such cases, nothing may be found in the gall-bladder but thick ropy mucus; but removal of this and of the underlying condition causing it completely frees the patient from his attacks.

The medical treatment of such attacks should be the administration of sedatives, the application of counter-irritation, free drinking of hot water; subsequently to the actual attack, the removal of all causes, and the correction of all habits, as to eating, drinking, and exercise, which favour gastric disturbance and stagnation of bile in the gall-bladder. These measures undoubtedly serve in many cases to arrest the catarrh at least for a time; probably, in not a few cases, to arrest it permanently. But there is always the liability that even if arrested the catarrh has led to the formation of one or more gall-stones which remain a permanent legacy, and become a cause of future trouble. See GALL-STONES.

3. Suppuration of the Gall-Bladder.—This condition is a result of the infective catarrh of the gall-bladder above referred to. Its occurrence is usually determined by the combination of (1) obstruction of the cystic duct by thick mucus, bile-sand, or gall-stone; and (2) the inclusion within the gall-bladder of a large number, or virulent strain of organisms. As in the case of other suppurative conditions, the effects vary according to the dose and the resistance. When the contents of the gall-bladder are muco-pus rather than pus, the condition is termed a *suppurative catarrh*; when pus is present the term '*empyema of the gall-bladder*' is used. When in a third, and fortunately rarer group, the infection has a fulminating character almost from the very outset, causing acute ulceration, gangrene, and rapidly spreading peritonitis, it is known as *phlegmonous cholecystitis*. In the last the walls of the gall-bladder become infiltrated with pus.

SYMPTOMS.—The symptoms are those of a septic infection, localised to the region of the gall-bladder. They include high fever, constitutional disturbance, local tenderness over the region of the gall-bladder, with enlargement and tenderness of the liver; and in cases of suppurative catarrh and empyema, the development of a distinct tumour.

TREATMENT.—This consists in opening the gall-bladder, evacuating the contents, and establishing free drainage externally until the bile passes freely into the duodenum.

4. Cancer of the Gall-bladder.—This is either *secondary* to cancer of the liver, or occurs as a *primary* growth. The latter is usually of the columnar-celled variety. In the great majority of cases it is associated with the presence of gall-stones. According to Schröder, as many as 14 per cent. of all cases of gall-stones suffer at the same time from cancer of the biliary passage. The association of the two should always be borne in mind in cases of long-standing gall-stones. It furnishes one of the

reasons why gall-stones in middle-aged and elderly people should, whenever possible, be removed by surgical means.

WILLIAM HUNTER.

GALL-STONES.—SYNON.: Hepatic Calculi; Cholelithiasis; Fr. *Calculs biliaires*; Ger. *Gallensteine*.

MODE OF ORIGIN.—This condition is the result of catarrh of the bile-passages, and of two changes in the bile thereby produced: (1) increase of cholesterolin at first contained in the mucus and afterwards precipitated in solid amorphous or crystalline form; and (2) the precipitation of pigment of the bile in an insoluble form combined with calcium. These changes may occur in any portion of the bile-passages; but they are specially liable to occur in any part where time is given for mucus to collect. Hence the commonest site of gall-stone formation is the gall-bladder. The pressure at which the bile is secreted is so low that the rapidity with which it flows is greatly influenced by (1) the amount of resistance it meets in the long line of narrow passages through which it flows, so that an increase of mucus hardly appreciable in itself may nevertheless considerably retard the flow of bile, and (2) the freedom of movement of the diaphragm. Free movement of the diaphragm by compressing the liver is probably one of the most important factors in promoting expulsion of bile from the gall-bladder, and any cause which interferes with such movement, such as tight lacing, pregnancy, and sedentary habits, correspondingly tends to interfere with the proper emptying of the gall-bladder, and favours the occurrence of catarrh.

ÆTIOLOGY.—*Age.*—Gall-stones are exceedingly rare in infancy and childhood, but become more and more common as age advances. *Sex.*—Women are five times more subject than men. This greater liability of women is connected with pregnancy, tight lacing, laxity of abdominal walls, and diminished movements of diaphragm. *Sedentary habits.*—These are a factor generally associated with advancing age, and seen in the case of inmates of lunatic asylums. *Heredity and gout* are regarded as factors by some observers. From all these considerations it is clear that anything favouring stagnation of bile in the gall-bladder predisposes to the formation of gall-stones. But mere stagnation of bile alone without other factors will not induce the precipitation of cholesterolin and bile-pigment. To give rise to this, a diseased condition of the mucous membrane is necessary. The *cholesterin* of gall-stones is a product of the secretion of the mucous membrane and glands of the gall-bladder and of the larger bile-ducts, or of the disintegration of their epithelium. Its formation in the form of viscous masses by the mucous membrane of the gall-bladder can be actually observed. The other chief constituent of gall-stones is *bilirubin-calcium*, also a product of catarrhal conditions of the mucous membrane of the bile-passages, its precipitation being determined by the presence of albumen derived from degenerating epithelium or inflammation of the gall-bladder. The conditions leading to the presence of these two chief constituents of gall-stones are thus essentially the same, and such as specially prevail in catarrhal conditions of the bile-passages and gall-bladder. Hence it is that the formation of gall-stones is liable to complicate nearly all affections of these passages marked by catarrh. See GALL-DUCTS.

Causes of Catarrh.—Three chief factors are to be recognised:—(1) *Passive Congestion.*—This is

best seen in chronic heart-disease. (2) *Chronic Indigestion*.—The catarrh caused by the excretion of irritant substances with the bile affects in the first instance the smaller ducts, but always extends to the gall-bladder. It leads to the formation of the small bilirubin-calcium concretions, or so-called bile-sand not infrequently found in the bile-ducts and gall-bladder. These concretions constitute the nuclei of most gall-stones. (3) *Infection of the gall-bladder*.—This is by far the most potent cause, and is favoured by temporary stagnation of bile. The most common organisms found are the pyogenic organisms, the *Bacillus coli communis* and the *Bacillus typhosus*. The infection usually occurs through the bile-ducts; but, in the case of infective diseases, it probably also occurs through the blood. This has been shown with certainty to be the case with the *B. typhosus*; and a fact of great importance is that the infection may persist in the gall-bladder for periods of months or even of years after it has disappeared from every other organ of the body. Evidence as to the importance of typhoid-infection as a cause of gall-stones is steadily increasing year by year. In no fewer than nineteen out of twenty-two cases of enteric fever, the *B. typhosus* was found in the contents of the gall-bladder—in fifteen of these cases in pure culture (Chiari). In a considerable number of cases the first attack of gall-stones has followed shortly after an attack of enteric fever. A temporary infection of this kind may, without causing any symptoms, excite a catarrhal condition of the gall-bladder sufficient to produce one or more gall-stones; and even when it passes off, the latter are left as the legacy of the infection. In such a case, the stones may remain quiescent for years without causing any symptoms; but they are liable to excite trouble at any time, their mere presence serving to keep up a certain degree of catarrh in the gall-bladder.

A still more important danger is that, in the event of any subsequent infection, the conditions are more favourable to its action. The catarrh induced is of a more severe character, more gall-stones are formed; and irregular spasms of the gall-bladder (the cause of the severe pain in such cases) are induced. Some of the stones may be forced into the cystic duct (causing biliary colic) and thence into the common duct (causing the jaundice following the biliary colic). Independently of such changes, the inflammatory catarrh excites inflammation in and around the walls of the gall-bladder; or it may cause the various suppurative conditions in the gall-bladder already described (suppurative catarrh, empyema of the gall-bladder, suppurative and phlegmonous cholecystitis).

CHARACTERS OF GALL-STONES.—Gall-stones vary in size from fine gravel to masses 4 or 5 inches in diameter, and in number from one to many hundreds; in the case of those found in the gall-bladder, they present a characteristic faceted appearance from mutual pressure. On section, they usually present a dark central portion—the so-called nucleus of bilirubin-calcium, around which is the body of the stone consisting chiefly of cholesterol. In most cases this cholesterol is amorphous, in certain cases it is crystalline. The stones are generally more or less bile-stained; but if formed in a gall-bladder or any other cul-de-sac cut off from the bile, their colour is pearly white. The stones formed in different portions of the biliary system have certain distinguishing features; those formed within the liver are small and dark, con-

sisting of pure bilirubin-calcium; those within the gall-bladder are usually numerous, faceted, and of mixed character, consisting chiefly of cholesterol; while those found within the common duct are usually large, round, or oval, and either single or in groups of two or three, consisting largely of bilirubin-calcium.

SYMPTOMS.—The symptoms associated with gall-stones may be described under three headings:—(1) those connected with their formation; (2) those connected with their passage through the cystic duct ('gall-stone colic'), or through the common bile-duct ('gall-stone jaundice'); and (3) those connected with various complications resulting from the presence or passage of gall-stones (choolangitis, suppurative cholecystitis, empyema of gall-bladder, formation of biliary fistula betwixt the gall-bladder and adjacent parts—duodenum, stomach, colon, abdominal walls—and intestinal obstruction).

1. The symptoms connected with the formation of gall-stones are always vague and often absent. They include a sense of discomfort in the right hypochondrium experienced from time to time in connection with attacks of indigestion, and generally referred to such attacks. The associated catarrh of the bile-ducts is generally manifested by more or less 'biliousness.' So that the early history is usually one of recurrent attacks of indigestion. When these become marked, the local discomfort becomes more intense; and the patient has attacks of spasmodic pain and distress due to painful contractions of the wall of the gall-bladder. Such attacks probably mark the passage of tough mucus or bile-sand through the cystic duct. They may last off and on over a period of many years.

2. The most characteristic symptoms are those connected with the passage of gall-stones, viz. *biliary colic*. The attack—which may be preceded by more or less dull pain in the region of the liver—usually sets in suddenly with agonising pain in the right hypochondrium and epigastrium, shooting up to the right shoulder, so intense in character that the patient rolls about in agony. The pain is generally accompanied by rigors, vomiting, profuse sweating, and great feebleness of pulse. The region of the liver is tender, and the muscles over it rigid. The attack varies in duration, sometimes lasting only for a few hours, sometimes for several days. It ceases when the stone escapes from the cystic duct into the common bile-duct. Its onset is generally followed in the course of 24 hours by more or less jaundice—sometimes intense if the stone is large and becomes impacted in the duodenal end of the common bile-duct; but generally slight and transient. The gall-stones may subsequently be found in the stools on breaking up these in water and then filtering them through a fine sieve. Similar attacks may occur from time to time, so long as gall-stones continue to pass.

3. The effects of biliary colic do not always pass off in the way above described. The stone, instead of passing, may become impacted: either in the cystic duct: or if it escape from the latter, in the common bile-duct:—

(1) *Impaction in the Cystic Duct*.—The effects are (a) a dilatation of the gall-bladder by fluid of a clear mucoid character (*hydrops of gall-bladder*), the dilatation sometimes giving rise to a large tumour projecting downwards from the under surface of the liver; (b) *suppurative cholecystitis*, or empyema of

gall-bladder with all its symptoms; (c) *chronic inflammation and cicatricial induration* of parts around gall-bladder, and cystic duct; occasioning local discomfort and pain.

(2) *Impaction in the Common Duct*.—The stone is usually arrested just at the orifice of the common duct, and may thus give rise to (a) more or less complete *jaundice*, according to the completeness of the obstruction. If the stone remains movable, the jaundice may be slight or even absent. Intense jaundice in such cases is due to obstruction from catarrhal swelling around the stone. (b) *Cholangitis*.—The catarrh may in the first instance be non-infective; but is very liable to become infective from the entrance of organisms into the common duct. A condition of infective cholangitis is then set up, characterised by ague-like paroxysms, chills, fevers, sweatings, jaundice of varying intensity, and recurrent attacks of pain. These symptoms may continue off and on for years, but they are always dangerous; since the condition is liable to give rise to suppurative cholangitis with septicæmia. *See GALL-DUCTS*.

(3) *Biliary fistula* are not uncommon. *See BILIARY FISTULA*.

(4) *Obstruction of the bowel* is a rarer complication of gall-stones. *See INTESTINAL OBSTRUCTION*.

TREATMENT.—(a) *During the attack* of biliary colic, the patient should be kept under morphine administered hypodermically, in doses of a quarter of a grain; or, if the attack be very severe, should be placed under chloroform. Great relief is given by a hot bath, or by hot fomentations over the liver. Milder attacks are relieved by small doses of *Tinctura chloroformi* et *morphinæ composita* or by preparations of opium—as the liquid extract—by the mouth. The patient should drink freely of warm water.

(b) *Between the attacks*, careful dieting and regular exercise are of the utmost importance. Laxatives, and the use of mineral waters, especially those of Vichy and Carlsbad, should be enjoined.

The object of the treatment is (a) to remove all causes of catarrh of the stomach and of the bile-ducts; and (b) to flush the biliary system as freely as possible.

In the removal of catarrh, great advantage can be obtained from use of alkalis, salts of bismuth, and bitter tonics, especially nitro-hydrochloric acid. To promote a free flow of bile, water should be drunk freely—not with food, but *between meals*. By active exercise, assisted by home gymnastics, or massage of the abdomen, free movements of the diaphragm may be stimulated. As regards drugs, the most valuable are salicylate of sodium (15 grain doses), sulphate of sodium, and phosphate of sodium (1 to 2 drachms daily), with occasional small doses of a mercurial pill.

A great number of remedies for gall-stones have been proposed with the idea of dissolving the stones, but none of them are efficacious. The best known is Durande's—which consists of three parts of ether and two parts of turpentine—10 to 20 minims enclosed in capsules three times a day. Olive oil has also been recommended for the same purpose.

SURGICAL TREATMENT.—The operation of opening the gall-bladder and removing stones is attended with remarkable success; and in the early stages, before complications have set in, is unattended by any difficulties.

The following are the indications for operative treatment, given by Mayo Robson:—(1) Frequently recurring biliary colic without jaundice, with or without enlargement of the gall-bladder. (2) Enlargement of the gall-bladder without jaundice, even if unaccompanied by great pain. (3) Persistent jaundice ushered in by pain, and where recurring pains, with or without ague-like paroxysms, render it probable that the cause is a gall-stone in the common duct. (4) Empyema of the gall-bladder. (5) Peritonitis starting in the right hypochondrium. (6) Abscess around the gall-bladder or bile-ducts, whether in the liver, or under, or over it. (7) Some cases where, although the gall-stones may have passed, adhesions remain and prove a source of trouble. (8) A mucous or biliary fistula. (9) Certain cases of jaundice with distended gall-bladder dependent on some obstruction in the common duct; but in such cases the increased risk must be borne in mind, as malignant disease may be the cause of the obstruction. WILLIAM HUNTER.

GALLOPING CONSUMPTION.—A popular name for pulmonary tuberculosis when it runs an acute course. *See PHTHISIS*.

GALVANISM, Uses of.—*See ELECTRICITY IN MEDICINE*.

GANGLION (γάγγλιον, a hard swelling).—The term has been applied to three somewhat different affections: namely (1) the *simple* ganglion or cystic tumour found in connection with the sheath of a tendon; (2) the *compound* or *diffuse* ganglion, which consists of a chronic effusion into the common sheath of a group of tendons, giving rise to a fluctuating swelling which may contain melon-seed bodies; and (3) enlargements of the *bursæ mucosæ* which may or may not communicate with the synovial cavity of a neighbouring joint. *See BURSÆ, Diseases of*.

1. Simple Ganglion.—The simple ganglion forms a rounded tumour occasionally lobulated, in the immediate neighbourhood of some tendon. The most common situation is the dorsum of the hand or wrist, but the swelling may also be found on the dorsum of the foot, the palm of the hand at the root of a finger, and behind the outer and inner malleoli. The tumour varies in size from a pea to a pigeon's egg. It may fluctuate distinctly or be so tense as to seem solid. It is not adherent to the skin or to the tendon to the sheath of which it is attached. It is painless, but may give rise to a sense of weakness in the affected part. The wall is composed of a more or less delicate fibrous tissue, lined by an imperfect layer of endothelial cells. Its contents are usually semi-solid, like clear jelly, but are sometimes fluid. The fluid resembles that secreted by the synovial membrane of the sheath of the tendon. The tumour probably arises in most cases by a hernial protrusion from the sheath of a tendon, the neck of which gradually contracts and finally closes, and this gives rise to a cyst in connection with the sheath. It is also said that a ganglion may originate in the dilatation of pre-existing small follicles or sub-synovial crypts, and also that it may occasionally arise as a cyst of entirely new formation.

TREATMENT.—Painting with iodine or counter-irritants is uncertain in result, and generally useless. Forcible rupture of the cyst-wall by a blow, or by steady pressure, with the accompanying

subcutaneous diffusion of the contents, sometimes effects a cure. The best treatment is to puncture the cyst with a sterilised needle or tenotomy-knife, lightly scarifying the interior of the sac, and squeezing out the contents; pressure should then be applied. If this method should fail, the ganglion may be treated by open incision, and the cyst-wall dissected away from the tendon and its sheath.

2. The Compound Ganglion.—This disease is nearly always found in connection with the sheath of either the common flexors or extensors of the fingers. It may consist of a simple effusion into the sheath, forming an hour-glass-shaped swelling, the constriction being caused by the annular ligament. The tumour may contain melon-seed bodies. It is the presence of these little bodies which gives rise to the sense of crackling experienced in manipulation of the swelling. They are smooth and oval in shape, and of a pearly white colour; are almost cartilaginous in consistence; and are composed of imperfectly developed fibrous tissue with an appearance of concentric lamination. Their origin is somewhat uncertain. They have been supposed to arise from the hypertrophy, and subsequent detachment by the movements of the tendons, of the fringes found in synovial membranes with possibly subsequent fibrinous deposit from the contained fluid. It should, however, be remembered that such bodies are not peculiar to the compound ganglion. They are also found in distended bursæ, especially that over the patella, where no movement of tendons can occur and where the lining membrane is not a fully developed synovial structure. In this case they probably arise from small accidental hæmorrhages and owe their characteristic shape and consistence to the friction of the opposing surfaces of the sac in which they lie.

ÆTIOLOGY.—It is now well known that the disease known as compound ganglion of the wrist is tubercular in origin, and is really a chronic tubercular teno-synovitis. In advanced cases the tendons themselves are invaded by the advancing tubercular jelly-like tissue, which resembles that found in similar affections of the synovial membranes of joints. In some cases the condition is associated with tubercular disease of the carpus.

TREATMENT.—The treatment must follow the lines of the treatment of tubercular disease of synovial membranes elsewhere. Injections of iodoform-emulsion in glycerine may be tried, but early radical operation is probably the best.

Under strict aseptic conditions, the skin over the swelling should be incised and the flaps reflected, thoroughly exposing the whole of the cyst; this should then be carefully laid open from end to end and the contents evacuated. Any tendons that are implicated in the cyst-wall, or are invaded by the tuberculous tissue, are freed and carefully cleaned by dissection; and the whole of the walls of the sac with all tuberculous tissue are cut away. The resulting wound is carefully closed, firm pressure applied, and the limb immobilised on a splint.

The results of such treatment, as regards the movements of the tendons and usefulness of the limb, are good, if aseptic primary union take place, and if passive movements of the fingers are early carried out. Many of these patients, however, eventually develop other manifestations of tubercular disease.

MARCUS BECK.
C. J. BOND.

GANGRENE (*γρᾶνω*, I gnaw or corrode).—**SYNON.**: Mortification; Fr. *Gangrène*; Ger. *der Brand*.

DEFINITION OF TERMS.—Gangrene is a clinical term implying death of a considerable mass; it implies 'molar' death of the tissues, as opposed to the 'molecular' death which characterises ulceration. Necrosis also means death of tissue, and pathologically is applied equally to a cell and to a limb: clinically its use is limited to death of hard tissues (bone and cartilage), and the dead part is called a 'sequestrum.' When death is confined to soft parts, the term 'sloughing' is used, and the product is called a 'slough.' 'Mortification' and 'Sphacelation' are applied equally to death of all tissues, hard or soft.

CLASSIFICATION.—*Clinically* cases of gangrene are usually classified according to their cause (so far as this is known), e.g. inflammatory, thrombotic, or embolic gangrene; or according to some striking factor in the ætiology, e.g. diabetic gangrene. But a practically important division—into 'dry' and 'moist' gangrene—is based upon the dryness of the dead part—a matter much influenced by accidental circumstances.

ÆTIOLOGY.—**I. General Causes.**—These are, as a rule, pre-disposing. They embrace all depressing circumstances and especially such as induce weak action of the heart. Among them may be mentioned insufficient and bad food; exposure, exhaustion, loss of blood; cardiac disease; extreme anemia. Glycosuria and albuminuria seem to render the tissues more liable to pyogenic infection and its results.

2. Local and Exciting Causes.—These destroy cell-life either *directly*, e.g. heat; or *indirectly* by cutting off nourishment. The same cause may act either directly or indirectly. The exact mode of action of some causes is unknown, e.g. injury to the central nervous system.

1. Injury.—*Mechanical Violence*—cutting, crushing, tearing—may directly destroy the life of a part; or it may injure the vessels of a limb, causing such an immediate obstruction to the circulation, or such an extensive extravasation into the tissues, or later on thrombosis of such important trunks, that indirect gangrene of more or less of the part beyond results. Ligature of an artery in its continuity may cause gangrene, if the collateral circulation cannot be established. Circular constriction may at once stop all circulation, as in a tightly strangulated hernia or the ligature of a pedicle; or it may cause only venous obstruction at first, great swelling and possibly sloughing occurring later on—as in ordinary paraphimosis or slightly strangulated hernia. Constant pressure, rendering a part anæmic and interfering with the activity of its cells, is a chief factor in the causation of bedsores, &c. The direct effect of caustic chemicals and of heat and cold need only be referred to here. See **HEAT** and **COLD**.

2. Inflammation.—All inflammations resulting in gangrene are infective, and pyococci are the usual cause. They induce gangrene primarily by the irritant action of the micro-organisms on the tissues; secondarily by circulatory disturbance and œdema. A slight cellulitis in a strongly predisposed patient—e.g. of the leg in a patient with dropsy—may lead to extensive sloughing. The more intense the cellulitis, the less need for predisposition. Passing up the scale of intensity we reach the 'acute spreading,' 'fulminating,' or 'emphysematous' gangrene, due to the *B. œdematis aërobis*, the *B.*

emphysematosus or the B. œdematis maligni, which may destroy a limb in three or four days. There is also a group of cases characterised by rapidly spreading ulceration with the formation of membrane upon the surface, viz. true diphtheria of wounds, phagedænic ulceration, and hospital gangrene, in the last two of which only cocci have been recognised. See CANCRUM ORIS.

3. Vascular Obstruction. (a) *Arterial.*—*Spreading arterial thrombosis*, excited usually by the changes in atheromatous vessels, is a very common cause of gangrene in old people and in those whose vessels degenerate prematurely. The term 'senile gangrene' was synonymous with thrombotic gangrene from degenerate vessels; but it has lost all exactness of meaning since it has been shown that many cases of dry gangrene in the old are inflammatory, being doubtless favoured by the presence of degenerate and especially of thrombosed vessels.

In *Endarteritis obliterans*, described by Felix v. Winiwarter, the thickening of the intima leads to great diminution and even obliteration of the lumen of the arteries of a part. No cause is discoverable, the typical patients being young and free from syphilis. The disease is rare.

Embolism not uncommonly obstructs arteries and may thus give rise to gangrene. See EMBOLISM.

(b) *Venous.*—So free is the anastomosis of veins that it is extremely rare to meet with gangrene as a result of venous thrombosis, however extensive.

4. Nervous Lesions.—Motor paralysis, as in anterior poliomyelitis, leads to malnutrition, and this predisposes the part to ulceration from irritation, and to sloughing from pressure. But the strongest evidence of the influence exercised by the nervous system in favouring gangrene is afforded by cases of sudden crushes, hæmorrhages into or inflammation of the spinal cord, or of some wounds and other acute lesions of a cerebral hemisphere, in which large bedsores develop with great rapidity, and in spite of all precautions. In paraplegia the slough is sacral, in hemiplegia retro-trochanteric—i.e. not in a part specially exposed to pressure. The sloughing is the result of an inflammation, and the share in its production to be attributed to the nerve-lesion is uncertain.

In Symmetrical Gangrene or Raynaud's Disease the most commonly accepted view of the ætiology is that spasm of the arterioles leads to gangrene of the affected parts; and that the spasm is of nervous origin. The stimulus inducing it and its mode of action are unknown. See RAYNAUD'S DISEASE.

5. Drugs.—*Ergot* when taken in bread made from rye infected with the *Claviceps purpurea*, which sometimes constitutes one tenth of the meal, gives rise to gangrene of toes, feet, fingers, nose, and ears. Its action is probably complex, as is the constitution of the drug. By one or other of its constituents, it slows the heart and somewhat weakens the pulse, causes contraction and even occlusion by thrombosis of arterioles together with a hyaline degeneration of the intima, produces violent cramps and the usual symptoms of irritant poisoning. Gangrene from this cause is far more common in men than in women for some unexplained reason—perhaps, because they take more than their share of the bread. The worst epidemics occurred when famine forced the people to eat the diseased rye so soon as it was garnered, and there-

fore when the ergot was fresh. It is somewhat astonishing that gangrene from the clinical use of ergot should be almost unknown, considering the freedom with which it is prescribed, and the periods during which it is exhibited (see ERGOTISM). *Carbolic acid* applied as a lotion has in rare instances caused gangrene of a finger or other small part. *Mercury* sometimes excites a sloughing stomatitis.

The ætiology of gangrene is often mixed. Thus, rarely, gangrene of a limb occurs during or just after an acute fever—especially typhoid—either from thrombosis or from embolism. It is obvious that the cardiac weakness and general depression induced by the illness must play an important part in determining the gangrene. Again 'diabetic gangrene' is often spoken of. In this disease, gangrene and inflammatory complications do not occur before early middle age, and they are not met with in acute or 'malignant' cases. The presence of excess of sugar in the fluids of the body apparently favours the growth of pyococci, i.e. favours infection; on the other hand the nutrition and resistance of the tissues are probably prejudicially affected thereby. Certain it is that an arterial fibrosis and endarteritis deformans may be found in young subjects, and that glycosuria often induces a chronic peripheral neuritis. Gangrene in diabetes is usually inflammatory, excited by injury; it is sometimes fairly rapid and painful, and it then depends chiefly on arterial degeneration; sometimes it is slow and painless, when it depends chiefly upon the peripheral neuritis (Godlee).

SIGNS OF GANGRENE.—For a few hours and sometimes for much longer it may be impossible to be sure whether a part is going to live or die. The signs of death are: 1. Change of colour—to waxy pallor, red and white mottling—and finally lividity: all these may be seen in Raynaud's disease, and maintained for considerable periods, recovery occurring ultimately. 2. Cessation of all function, including warmth, sensation, motion, pulsation, and secretion. Warmth may, however, be artificially maintained—although the part is dead; and even movement may be produced through the action of long tendons, as in the case of a dead toe. 3. Drying and shrivelling of a part—'mummification'—or putrefaction of it are positive signs of death.

Mummification is favoured: by gradual arterial obstruction, the venous path being open; by elevation; by slow progress, loss of epidermis, exposure to cool dry air, and by the absence of fat, muscle, and fluid effusions in the part affected.

Moistness and putrefaction are favoured: by sudden arterial obstruction (e.g. embolism), the heart being able to drive blood into the anæmic area, but unable to secure its return by the veins; by venous obstruction; by rapid progress; by warmth, poulticing, and other obstacles to evaporation; by much fat, muscle, or effusion of fluid.

By mummification, sepsis and the absorption of irritant and toxic bodies are reduced to a minimum; all occur very freely in moist gangrene. The promotion of drying is therefore very important. Sepsis is not a necessary consequence of gangrene, as is shown by the result of non-infective embolism of an end-artery in a viscus: but it is usually difficult to sterilise and protect from infection a large portion of a limb.

Whether a dead part dries or remains moist depends upon accidental circumstances to a suffi-

cient extent to prevent the constant association of a given cause with either the moist or the dry form. Moreover all grades between the two extremes are seen. It may nevertheless be said that injuries of all kinds, inflammations, sudden arterial obstructions, and venous obstructions, usually cause moist gangrene; and that gradual obstruction of arteries is the great cause of dry gangrene (thrombosis, endarteritis obliterans).

APPEARANCE OF PART.—(1) *In dry gangrene.*—A dull red or purple patch appears, usually upon a toe, without marked swelling; it slowly spreads, the skin becoming dry, wrinkled, brown or black, and opaque. A toe in this condition has been somewhat aptly compared to a 'sucked-out grape skin.' Throughout the progress of the case it is fairly clear where dead and living tissues meet, the living showing a band of redness of moderate intensity due to absorption of irritants from the dead (and moist) layer in contact with them. A more or less offensive odour is usually present.

(2) *In moist gangrene* the appearances vary much more than in dry. Usually the part is markedly swollen as the result of effusion from inflammation, from venous obstruction, or from mere lagging of blood. Even if at first bright red from acute inflammation, the part becomes blue and livid from retarded circulation as gangrene approaches. The epidermis is often raised by sero-sanguineous effusion either in bullæ or over wide areas. When the epidermis is brushed off, the derma appears moist, dull red, black, greenish or ashy grey—the latter colours being associated with more or less advanced putrefaction, which is further evidenced by a highly offensive odour and emphysematous crackling. This may not be discoverable in tense parts; and is most marked when due to one of the bacilli casually concerned in emphysematous gangrene. The spread of moist gangrene is usually more rapid than that of dry, and it is often impossible to be certain where dead and living tissues meet: this, however, soon becomes evident if spread ceases by the development of a 'line of demarcation,' i.e. of inflammation in the living tissues in contact with the dead.

The appearances of parts destroyed by mechanical violence, by heat, by cold, or by various chemicals, vary much with the physical effects of these causes, e.g. crushing, charring, or straining a part.

GENERAL SYMPTOMS.—These are complex, being made up of symptoms connected with the particular part affected, of others due to the cause of the gangrene, and of others again arising from changes in the gangrenous part.

The mere aseptic death of many parts hardly affects the general well-being; but death of any part of course implies cessation of the functions of that part—be they of great or little importance. Some causes of gangrene are painful and produce shock when the pain is sudden and severe, exhaustion when it is long-continued. Gangrene from slowly spreading thrombosis ('senile') or from endarteritis obliterans is often accompanied by constant and great pain, and is usually preceded by signs of impaired circulation—coldness, numbness, formation and painful cramps—warnings which should never be neglected in old people. The moist gangrenes are more acute, and pain as a rule is not great; embolism is perhaps the most painful cause.

Next to the part affected and its mass, its infection with septic or other germs is the point of chief im-

portance. There is a great difference under this heading between dry and moist forms. In the latter all the symptoms of acute or chronic septic absorption are, as a rule, marked, and they often lead to a fatal issue; in the former they are slight and often local only, so that health is preserved for long periods.

Arrest of Gangrene.—Many forms, e.g. from injury, do not tend to spread. Inflammatory gangrene is the type of spreading gangrenes; it may supervene upon other forms when the gangrenous parts become septic. Thrombotic gangrene and other forms due to progressive obliteration of arteries also tend to spread. When the causes cease to act and the gangrene is checked a 'line of demarcation' becomes evident between the dead tissues (moist or dry, septic or aseptic), on the one hand, and the more or less inflamed living tissues, on the other. The irritation of the living by the dead leads to the formation of granulation-tissue which absorbs the connections between the living and the dead until—after weeks, months, or even years—the dead part is freed and comes away. It is then found that the deeper structures of a limb have retained their vitality to a greater distance from the centre than the more superficial; so, should healing occur, it is with the formation of a typical 'conical stump.' In septic cases the eroding granulation-tissue is confined to the living tissues; but an aseptic slough may be completely infiltrated and absorbed; or, if too large for this, cast off without suppuration. During the process of separation of septic parts, hæmorrhage may occur; it is specially frequent in phagedænic ulceration and hospital gangrene.

PROGNOSIS.—The general condition, the situation and extent of the gangrene, its tendency to spread, and the point at which amputation must be done to remove the dead part, must be considered.

TREATMENT.—*General.*—In depressed patients the importance of careful feeding, stimulants in moderation, and warmth is obvious. Pain must be met by anodynes; and in chronic thrombotic gangrene a good deal depends upon whether opium is or is not well borne in cases in which amputation is not allowable. Bed-sores must always be carefully guarded against. If diabetes is present, suitable dietetic and other treatment should at once be instituted.

Local.—Something in the way of prevention may often be effected by removal of pressure, division of constricting bands, or incisions to evacuate effusions containing irritants and causing tension.

It may be stated, generally, that in cases of gangrene due to injuries, so soon as it is evident how much is gangrenous, amputation should be done; so, also in gangrene from embolism. These cases do not tend to spread, but do tend to putrefy: and every precaution against this must be taken.

In acute spreading gangrene (inflammatory) with no tendency to arrest, early and high amputation affords the only chance of life, but is only too often unsuccessful, owing to local recurrence.

In chronic, slowly spreading, thrombotic cases, often starting afresh after apparent arrest, the patient may be saved much prolonged suffering by amputation well above the gangrenous part. It was advised that in gangrene reaching to the middle of the foot amputation should be done at the knee; but the tendency of late has been to amputate through the leg. With regard to the height at

which amputation should be done : in cases of simple injury, it is necessary to see that the tissues of the flaps are sufficiently supplied with vessels to be viable. Where the gangrenous part is septic it is at least equally important to avoid infected tissues in the flaps ; for any inflammation endangers the vitality of injured tissues. In virulent spreading inflammations leading to gangrene, the micro-organisms are carried up the limb for long distances by the lymphatics ; so, to avoid recurrence of gangrene in the flaps, it is desirable to avoid the presence in them of any oedema, though success may follow an amputation with oedematous flaps. In chronic thrombotic cases, recurrence of gangrene may be due to the use in the flaps of tissues too poorly supplied with blood to live : this is a valid reason for operating high up. But, too often, fresh gangrene is the result of an inflammation due to infection at the operation. Given efficient asepsis, therefore, the circulation in the tissues, determined by eye and touch, must fix the height at which an amputation will be safe. In Raynaud's disease, early amputation should never be done, such surprising recoveries occur ; in ergotism, too, a line of demarcation should be allowed to form.

While waiting to learn the extent of a gangrene, or in limited cases in which there is no question of amputation, the suspected part, and even that beyond it, should be cleaned as for operation and dressed with gauze and wool, as after operation, to prevent putrefaction.

If putrefaction of a dead part has set in, and it cannot be completely removed, an endeavour to control the putrefaction, to combat local irritation and general sepsis, and to promote speedy separation of the part, must be made. Remove as much dead tissue as possible : score dead parts freely to allow fluid to escape : fill the cuts and cover moist surfaces with boric acid, acetate of aluminium, aristol or other powder ; apply a dressing of cyanide gauze and wool, and leave the part exposed to the air. On no account use poultices. Boric or mercuric fomentations well wrung out will often keep sloughs fairly dry and sweet, and they quicken granulation. They must be often changed, the materials being washed and boiled before use again.

In cases of gangrene associated with diabetes, amputation should be done in painful cases spreading quickly in spite of diet and codeine (e.g. a quarter of an inch *per diem*). These cases are dependent chiefly on arterial degeneration. In slow painless cases there is no need to hurry, or to operate high up, trimming up being often sufficient. A tendency to coma is a reason for amputating rather than a contra-indication. Actually comatose patients have recovered after amputation and lived for years (König).
STANLEY BOYD.

GARGLE (γαργαρίζω, I wash the throat).—SYNON. : Fr. *Gargarisme* ; Ger. *Gurgelmittel*.

DEFINITION.—Gargles are liquids employed for the production of local effects on the throat and pharynx.

MODE OF GARGLING.—Gargling consists in taking about a tablespoonful of the gargle into the mouth, throwing back the head, and agitating the liquid by the air expelled through the larynx. With some persons the gargle goes little beyond the uvula and base of the tongue ; but if the head be thrown well back, the fluid can be made to pass into the cavity of the pharynx, and may even reach the

larynx and vocal cords. The tension of the muscles, in thus throwing back the head, is apt to provoke efforts at deglutition, so that sometimes small portions of the gargle may be swallowed ; and occasionally the effort terminates abruptly in the patient jerking his head forwards, and expelling the gargle forcibly through the nose.

Guinier, of Cauterets, has demonstrated a method of gargling the laryngeal cavity. His instructions are as follows : (1) to raise the head slightly ; (2) to open the mouth moderately ; (3) to protrude the chin and lower jaw ; (4) to emit the sound of the double vowel æ. These four movements open largely the back of the mouth, lift the velum palati and uvula, separate the base of the tongue from the posterior wall, and allow the liquid used for gargling to gravitate into the cavity of the larynx. One expiratory act is the only respiratory movement that is now possible, deglutition is under control, and the gargle bathes the pharynx and supraglottic portion of the larynx. When it is desirable to gargle the back of the pharynx and posterior nares, the patient must take a mouthful of the gargle, and use it as he lies flat on his back with his tongue drawn forward.

USES AND COMPOSITION.—The use of gargles is contra-indicated in parenchymatous inflammations of the tonsils, and in all cases where movement of the fauces causes severe pain ; and where, as in some persons, an inability exists to retain liquid beyond the anterior pillars of the fauces : for these it is more convenient to apply the fluid to the fauces and pharynx, either by injection, or in the form of a medicated spray, or else by aid of a brush or sponge.

Gargles vary in composition according to their object. If prescribed with the view of exercising the muscles of the soft palate and pharynx, and thereby increasing their tone, cold or iced water is generally sufficient. If with the view of reducing and allaying local inflammatory conditions of the throat, solutions of chlorate or nitrate of potassium or of borax or sodium bicarbonate (two drachms to eight ounces), or of solution of acetate of ammonium (one part in four), combined with infusion of linseed, thin gruel, or water, and used warm, are the best. When it is desirable to excite the mucous membrane and neighbouring glands to further secretion and thus reduce local congestion, stimulant gargles are useful, such as the tinctures of capsicum (half a drachm to eight ounces), arnica, myrrh, pyrethrum, and Eucalyptus rostrata (two drachms to eight ounces) ; this class of gargles often relieving the deafness caused by obstruction of the Eustachian tubes, by increasing the pharyngeal secretion. To check excessive secretion, astringents are advisable in the form of the salts of iron and zinc, iron-alum or alum (forty grains to eight ounces), tannin (half a drachm to eight ounces), or infusion of rhatany. This infusion with addition of rose-water forms a valuable astringent gargle and has no deleterious action on the teeth. If we require to check ulceration, or to dilute and purify foul or putrid secretions from the throat and tonsils, antiseptic gargles will answer the purpose, composed of one or two drachms of liquor potassii permanganatis to a pint of water, or one drachm of solution of chlorinated soda to half a pint of water, or sulphurous acid one part to four, or glycerin of carbolic acid (half an ounce to eight ounces). In cases of syphilitic sore-throat, a gargle of perchloride of mercury (three grains to eight ounces) has been recommended. In many cases combinations of

different kinds of gargles are beneficial. Water, barley-water, rose or orange-flower water sweetened with a little honey, glycerin, or syrup, constitute the bases of most gargles.

JOHN C. THOROWGOOD.

GASTEIN, in the Austrian Alps.—Simple thermal waters. See MINERAL WATERS.

GASTRALGIA (γαστήρ, the stomach; and ἄλγος, pain).—**SYNON.**: Gastrodynia; Fr. *Gastralgie*; Ger. *Gastralgie*; *Magenschmerz*.—Paroxysmal pain connected with the stomach, and usually of a neuralgic type. See STOMACH, Neuroses of.

GASTRECTASIS (ἐκτασις, stretching).—See STOMACH, Dilatation of.

GASTRIC FEVER.—A popular name for any febrile condition attended with prominent abdominal symptoms; as well as for typhoid fever. See TYPHOID FEVER.

GASTRIC ULCER.—See STOMACH, Ulceration of.

GASTRITIS.—Inflammation of the Stomach. See STOMACH, Inflammation of.

GASTRODYNIA (ᾠδύνη, pain).—Pain connected with the stomach. As generally used the term is synonymous with *gastralgia*. See STOMACH, Neuroses of.

GASTRO-ENTERIC (ἐντέρον, the intestine).—This term is applied to those morbid states in which the stomach and intestines are simultaneously affected. See STOMACH; and INTESTINES.

GASTROPTOSIS (πτῶσις, falling).—See STOMACH, Displacement of.

GASTRORRHOEA (ῥέω, I flow).—A vague term used to denote any excessive secretion of fluid by the mucous membrane of the stomach.

GENERAL PARALYSIS OF THE INSANE. **SYNON.**: General Paresis; Paretic Dementia; Dementia Paralytica; Fr. *Paralyse générale des aliénés*; Ger. *Allgemeine Paralyse*.

DEFINITION.—A disease of the nervous system especially affecting the brain; characterised clinically by progressive generalised loss of power and sensation, associated with general failure of nutrition and progressive mental deterioration ending in dementia; though usually presenting for a time on the one hand exaltation and expansive ideas, or on the other depression with hypochondriacal or melancholic ideas; almost invariably fatal and presenting *post-mortem* evidence of organic changes in the brain and spinal cord and their membranes.

CAUSES.—(1) *Sex.*—The male sex is more frequently affected than the female in the proportion of about 4 to 1. Below the age of twenty-five, however, the influence of sex is not so marked, and in general paralysis occurring in children the two sexes are equally affected. (2) *Age.*—The disease is most common between the ages of thirty and fifty-five years, though well-marked instances of it are met with in childhood and also in advanced age. (3) *Heredit.*—Insane inheritance is less frequently found than in other forms of insanity. Direct inheritance of general paralysis is found in some

cases; and of especial importance is the fact that a history of both parental syphilis and general paralysis is often obtained in juvenile cases, with evidence of inherited syphilis in the patient or other members of the family. The same association may also be found in cases where the patient is no longer 'juvenile,' but breaks down in young adult life, sometimes with no other apparent cause than an inheritance of syphilis and general paralysis. A parental history of alcoholism, epilepsy, 'softening of the brain,' hemiplegia, or 'paralysis' is not infrequent. (4) *Marriage.*—The greater number of general paralytics are married. (5) *The occupations* of general paralytics are those involving much strain, emotional excitement, exposure, and overwork with insufficient rest for repair. The lower classes are more liable to general paralysis than the upper. Pecuniary difficulties and adverse circumstances are frequently the immediate cause of the disease, but are generally associated with such predisposing causes as heredity, syphilis, or alcoholic excess. A previous history of mental disorder is rare. (6) *Head-injuries.*—There may be a history of head-injury many years before, followed by alteration of character or by alcoholism or syphilis. In some cases there is a history of a recent head-injury, but on investigation it is generally found that symptoms of general paralysis have preceded the injury, or that the latter has been due to a fall resulting from a slight seizure, and that there are other causes which might lead to the development of general paralysis. (7) *Dwellers in towns*, especially where syphilis and alcoholism are rife, are more liable to be affected than those living in the country. The Celtic races, as long as they avoid towns, syphilis, and alcoholism, seem to be free from the disease. (8) *Syphilis.*—The aetiological importance of syphilis in general paralysis is now generally recognised, though observers are not yet agreed as to its frequency or as to whether general paralysis invariably implies syphilitic infection either congenital or acquired. The more closely the history of cases is enquired into, the more frequently can a history of syphilis be obtained; and in the 'juvenile' cases there is always, and in adults occasionally, to be found evidence of congenital syphilis either in the patient or other members of the family. Syphilis appears to act in the great majority of cases as a predisposing rather than an exciting cause, the nutrition of the cortex being altered so that degenerative changes readily take place from other poisons or from mental or physical stress. (9) *Alcoholic Excess.*—A history of alcoholism is found very frequently in general paralysis, and is considered by some observers to be a more important cause than syphilis. Alcoholic excess is sometimes a symptom of the onset of the disease. (10) *Sexual Excess.*—General paralysis occurs most commonly in those who have led fast and irregular lives and in whom sexual excess has been combined with the two preceding causes. In other cases, as for example in the case of old men marrying a second time to partners much younger than themselves, this appears to be immediately responsible for the disease where no other cause can be traced. Sexual excess in the early stage may be mistaken for the cause of the disease. (11) *Sunstroke* is often credited with the production of general paralysis, but in most cases an early seizure has been mistaken for a sunstroke, and even in those cases where there has really been 'coup de soleil' there will be found to be other causes likely to produce

general paralysis. (12) *Lead-poisoning* in some cases gives rise to symptoms closely resembling general paralysis, but it is doubtful if it can produce the disease apart from other causes such as syphilis and alcohol. It is rare to find only one cause unless it be in the cases occurring early in life, where there is nothing else than congenital syphilis to account for the disease. The most common combination is the male sex, the prime of life, a previous history of syphilis, and either alcoholism, sexual excess, or great physical or mental stress.

SYMPTOMS.—The disease is usually described as occurring in 'stages,' but these divisions are arbitrary and useful only for purposes of description, there being often no dividing line between one stage and another.

The essence of the disease from the mental side is a progressive dementia, and from the physical side a gradually increasing paralysis, at first affecting the finer movements of speech and handwriting, the pupillary reactions and co-ordinate movements, and later involving general loss of power in the limbs and loss of control over the bladder and rectum.

First Stage.—This may be held to include the period from the first appearance of some mental or physical disorder up to the establishment of chronic mental failure. Alteration of character is very common before there is noticeable intellectual or bodily failure. Thus the earliest indications of loss of control and of impending intellectual ruin may be some act which brings the patient into conflict with the law. Theft, sexual crimes, drunkenness, incendiarism, exposure of the person, may occur thus in those who have previously been well-conducted. Failure of memory and of intellectual power and business-capacity appears early, the highest acquirements being affected, especially if they are of a nature requiring skill and dexterity. The emotional state is one of instability, varying from one of depression with sense of impending illness and tendency to weep at trifles, to a gay hilarious condition with sense of well-being and reckless benevolence. Outbursts of violent passion may occur, passing off quickly or leading to sudden violence. Fortunes may be squandered, or reckless speculation indulged in by those without means. The patient is ever changing his wishes and intentions, and there may be confusion of thought or 'absence of mind' passing into stupor and silence. Severe frontal or vertical headache, fullness or singing noises in the head, and vertigo are common. There may be insomnia with nocturnal restlessness, or, on the contrary, excessive drowsiness during the day.

Sooner or later definite mental alienation appears, and may occur in various forms. (1) A great many cases simply show from the first a progressively increasing dementia, with slightly marked or transient depression or excitement. The memory for recent events fails, as also do the business-capacity, judgment, and power of abstract thought. The will becomes weakened and the patient becomes childish. With this is loss of cleanliness and of tidiness in dress. The patient, though manifestly failing, expresses himself as being 'quite well,' or 'all right,' but without necessarily any exuberant exaltation. In other cases there is torpor and silence with resistance, sluggish circulation, and loss of facial expression. Control over bladder and rectum is lost early, and general enfeeblement develops rapidly. (2) The next group contains those cases marked by expansive delirium or maniacal excitement. The exalted

self-feeling which may exist in the earliest stage becomes more marked, and develops into boundless exuberance of spirits with general excitement, reckless expenditure, and extravagant delusions as to wealth, power, and position, changing from day to day and hour to hour. With this may be great violence or homicidal attacks, absurd self-decoration, noisiness, sleeplessness, destructiveness, and loss of flesh. In some cases the amount of exaltation is great but the general excitement moderate, while in others maniacal excitement preponderates, with the enunciation of exalted delusions from time to time. In the latter group the excitement is often extreme and marked by extreme destructiveness, filthy habits, refusal of food, and profound exhaustion, from which it appears that the patient cannot possibly recover. Incoherence becomes extreme, and the condition may pass into chattering delirium with fragmentary references to exalted ideas. In other cases the state is one of silent excitement. (3) Cases marked chiefly by depression. In some patients depression exists throughout the disease without any development of excitement or exaltation; in others it exists as an early stage, and after a time is replaced by these. The depression may be marked by delusions of wickedness, perdition, ruin or conspiracy, or hypochondriacal delusions as to the state of the viscera, or by stupor. In some cases delusions of persecution are found, resembling at first sight the organised delusions met with in chronic delusional insanity. Depression in general paralysis may be accompanied by refusal of food and suicidal attempts, as in functional melancholia, but there is more evidence of dementia. Loss of memory is frequent, and there may be isolated extravagant ideas associated with the depression. Occasionally micromania (delusion of belittlement) and 'necromimesis' (simulation of death) are met with. (4) In another group the mental disorder is characterised by alternating excitement and depression—general paralysis of the double form.

Whatever be the mode of onset, dementia is the goal to which the disease leads, and some evidence of it is found in all varieties.

The *physical signs* of the disease are of the utmost importance in forming a diagnosis, and should always be carefully sought for. They may occur very early and before the mental disorder is obvious, or may not be marked till later, and in some cases they are masked by the mental condition at first.

The facial movements, especially if there be excitement, are apt to be excessive and inco-ordinate. The act of speaking or showing the teeth is accompanied by twitchings or tremors of the lips and cheek-muscles or elevation of the eyebrows. The expression may be one of gaiety or of depression or of stolid indifference, but as the disease progresses expression is lost. Sucking, or smacking, or chewing movements of the lips and jaw are frequent. Occasionally facial paralysis is found.

The oculo-motor signs are of great importance. Occasionally ptosis or strabismus is met with, but chiefly in the early stages. The pupils are commonly unequal and irregular in outline, and the light-reflex is lost or sluggish, especially in the larger pupil, while the reaction to accommodation still persists, to be lost later on in the disease. In other cases, especially those beginning with tabetic symptoms, there is minute contraction of the pupils, with loss of light-reflex and loss of dilatation on shading or on peripheral stimulation. Failure of consensual

action of the pupils and of dilatation on peripheral stimulation is often a very early sign. The tongue shows fine tremor and coarse jerky movements on protrusion, and is often pressed tightly on the lower lip, as if for support. Affection of speech may be an early symptom or may not be marked till the disease has made some progress. It is characterised by tremor, increased effort, slowness, and stammering or stumbling over long or difficult words. It becomes more noticeable with fatigue, and often resembles in its slurred and slovenly nature the speech of a drunken man. Occasionally there is aphasia. The handwriting may be affected quite early, or later, as in the case of speech. It exhibits tremor, slowness, reversion to the childish type, separation of letters, omission of terminal letters or syllables or of entire words, or sometimes their repetition, and a general untidiness. It may be quite illegible. The hands show clumsiness in other movements requiring dexterity and delicate co-ordination, and tremor when they are held out with the fingers separated. The gait is commonly free and active in the earliest stages, and some of these patients are at first constantly walking. Sooner or later there is, however, clumsiness and unsteadiness, with a tendency to trip over obstacles and inability to walk along a straight line. In a certain proportion of cases there is ataxic gait with other symptoms of tabes dorsalis for some years before the onset of mental disorder. Temporary loss of power in one or other leg may appear early as the result of a seizure. The knee-jerks may be normal, but in a large proportion of cases are exaggerated. In others they may be absent either with or without ataxic gait, while occasionally one knee-jerk is lost and the other exaggerated. There may be defective colour-vision or defect of sight, sometimes associated with atrophy of the optic discs, defect of smell and diminution of cutaneous sensibility; but these are more common in the later stages of the disease. Occasionally a convulsive seizure, or a series of them, marks the onset of the disease, though these are much more frequent later on. Sexual power may be excessive at first, but more commonly sexual desire is increased but sexual power lost. At the end of the first stage there may be a complete remission of the mental symptoms, especially if the onset has been marked by excitement or depression, and to a less degree of the physical signs. Remissions may last for some months and may be of varying degrees of completeness.

Second stage.—The second stage may follow a remission or may be inseparable from the first, being merely marked by confirmed chronic mental failure. Dementia is now a marked feature, the memory for remote as well as recent events becomes unreliable or is lost. Thought and reasoning-power are much enfeebled; the patient lives in a world of his own, is wrapt up in his delusive ideas, and occupies himself in childish ways. In other cases there may be persistent unreasoning excitement with destructiveness and dirty habits; or hypochondriacal depression may replace the former exaltation. The emotional state becomes more uniform and apathetic, volition is enfeebled, and the patient is commonly placid, easily led, and spending a great deal of his time sitting unoccupied in an arm-chair. The special senses become more blunted, and analgesia may lead to injuries being overlooked. Bruises form easily and may suppurate: hæmatoma auris is often met with.

The physical signs previously referred to become more marked, the appetite may be voracious and the manner of eating gluttonous, so that the food may be bolted, and as a result of this and of diminished pharyngeal sensitiveness portions of it may pass into the larynx. Incontinence of urine (sometimes due to the overflow from a distended bladder) is more common than at first, and the patient needs constant attention to keep him clean. The general nutrition fails, so that whereas there was undue fatness at the end of the first stage or during remission there is now gradual loss of flesh and wasting of muscles. The temperature is slightly above normal. The skin becomes coarse and greasy, and there may be herpetic eruptions. Seizures of various kinds are now more apt to appear, and these may be epileptiform, apoplectiform, paralytic, or quasi-syncopeal in nature. Paralysis following seizures may be temporary or permanent, and the dementia is always more marked afterwards. Life may be cut short by a seizure in this stage.

Third Stage.—The third stage is marked by the extreme of mental and physical ruin. The patient is confined to bed and leads a purely vegetative existence, having to be fed, washed, and attended to, in every way. The mind is a blank, relatives are not recognised, and no attention is paid to surroundings. Speech is lost or reduced to meaningless noises. Sensation, emotion, and volition disappear. There is complete paralysis, the muscles waste, and contraction of limbs appears and often becomes extreme. Unless great care be taken as to cleanliness, bed-sores may form, and the bones become extremely brittle. Constant grinding of the teeth, even to erosion of the jaws, is a common feature. Finally death supervenes, from failure of the heart, from gradually increasing coma, from a convulsive seizure, or from some pulmonary, vesical, or intestinal complication.

DURATION.—The average duration of life from the onset of symptoms is two years, but some cases are cut short in a few weeks or months, while others live ten years or longer.

PROGNOSIS.—This disease is almost invariably fatal, but cases have been recorded in which the symptoms have become arrested, the patient remaining demented but the physical signs becoming stationary. Recovery has been reported in a very few cases, but generally such cases are examples of a long remission of symptoms.

DIAGNOSIS.—In the early stages the disease is frequently overlooked, care not being taken to examine into the physical as well as the mental state; and in many cases time alone will show the real nature of the disease. Alteration of character and failure of capacity for business in a man of adult life, especially where there is a history of syphilis, should lead to a careful physical examination. From ordinary acute mania or melancholia or other functional disorders the diagnosis is made by the physical signs. The occurrence of a fit early in the disease is a very significant sign. Epilepsy is not followed by the development of general paralysis, but, as the latter sometimes begins with a series of epileptic seizures, there may be a wrong diagnosis: the occurrence of the physical signs and the progress of the case will clear up the doubt. In senile cases there may be a difficulty in deciding between general paralysis and ordinary senile dementia. In both there may be tremor, general weakness, excitement, eroticism, loss of memory, convulsive seizures, and

progressive decay of body and mind. In senile dementia, although aphasia is not uncommon, there is not the characteristic affection of speech and handwriting of general paralysis, the pupils usually still react to light and are not unequal, and there is a greater liability to localised paralyses or hemiplegia, the result of thrombosis or cerebral hæmorrhage. Intra-cranial tumours sometimes give rise to symptoms resembling the demented form of general paralysis. The speech is, however, not characteristic, and the occurrence of vomiting and optic neuritis with local convulsive attacks and paralyses should clear up the diagnosis. Acute mania from alcoholic excess is often associated with great exaltation, but there is less liability to affection of the pupils, knee-jerks, and speech. Tremor, though common, is finer, and there is less inco-ordination than in general paralysis. There is more difficulty in cases of chronic alcoholism where there are persistent tremor and enfeeblement of mind with perhaps inequality of pupils. In such cases the disease does not progress during efficient treatment as does general paralysis. Hallucinations and delusions of persecution are more common, and there are often other evidences of chronic alcoholism, while the speech is not characteristically affected. Bromide of potassium and chloral hydrate if taken for long periods may give rise to delirium with much tremor, passing off with the cessation of the drug. Syphilitic disease of the brain or meninges may simulate general paralysis, but there is a greater likelihood of headache, optic neuritis, and local paralyses, while expansive delirium is as a rule absent. Antisyphilitic remedies may rapidly lead to recovery, whereas general paralysis seems to be unaffected by them. Although locomotor ataxy may precede the development of general paralysis, ataxy may in other cases be associated merely with functional mental disorder without development of affection of speech and handwriting and without dementia. The term 'pseudo-general paralysis' has been applied to cases of alcoholic or syphilitic origin in which there are tremors, inequality of pupils, local paralyses, and mental disorder. In some of these cases the disease eventually assumes the characteristics of general paralysis, namely progressive dementia and progressive paralysis, while in others there is recovery or arrest. In deciding as to the nature of any given case the whole of the mental and physical symptoms and the course of the disease must be taken into account.

PATHOLOGY.—The skull-cap is usually thickened, and the diploe obliterated. The dura mater is thickened and adherent to the bone, and may show hæmorrhagic pachymeningitis either on its inner or outer surface. The deposit may vary from a thin rust-coloured fibrinous layer to a large clot or a cyst contained in a fibrinous membrane covering one or both hemispheres. The pia-arachnoid is thickened, sodden, and gelatinous, and shows milky patches, especially over the frontal and parietal regions. Subarachnoid fluid is found in excess, where there is wasting of the convolutions. The pia-mater is unduly vascular, and is generally adherent in patches to the summits of the convolutions in the parietal and frontal regions, which become torn in the attempt to strip it. Sometimes the adhesion is so intimate that it is impossible to remove the pia-mater.

The brain generally is wasted, more especially in the frontal and parietal regions, and the substance

is soft and sodden. There may be localised softening or small hæmorrhages in the grey or white matter. The grey matter shows irregular vascularity or anæmia. The ventricles are dilated with excess of fluid, and the lining membrane is granular in appearance.

The spinal membranes may show similar changes to the cerebral. The substance of the cord is soft, and the cord as a whole frequently wasted. On section there may be evidence of degenerative changes in the lateral or posterior columns.

Microscopically, degenerative changes are met with in the cortical neurons, especially in the large pyramidal ones. The changes are met with in all degrees from commencing chromatolysis to complete destruction of the cell-body, separation from its processes, and granular disintegration.

The tangential and radial fibres of the cortex disappear. The small arteries are dilated and often distended with blood, their coats thickened, and the perivascular sheath full of exuded leucocytes. Syphilitic arteritis, and fatty and hyaline changes in the vessel-walls may be found. Ford Robertson considers proliferation of the cellular elements of the capillaries, or hyaline and granular changes in their walls, to be an essential change in general paralysis.

The neuroglia is increased, and especially in the outer layer of the cortex there is a diffuse matting of the processes of 'spider-cells.' The latter are also found scattered through the cortex in connection with the sheaths of vessels, or in contact with shrunken and degenerate nerve-cells. In the cord similar degenerative changes are found in the cells of the anterior cornua, and sclerotic changes in the lateral or posterior columns. Much controversy has raged in the past over the question as to whether the changes found in the central nervous system are evidence of a primary inflammatory process (meningo-encephalitis) with secondary degenerative changes, or a primary degeneration of the nervous tissues with secondary changes in the vascular fibrous and neuroglial structures. Mott advocates the view that the disease is essentially a primary degeneration of the cortical neurons with secondary changes due to the irritation of the products of degeneration. This theory of a primary degenerative change is more generally received than that of a primary inflammation. Another theory is that some toxin, either from syphilitic infection or some other poison, or arising from stress of the nervous system, leads to changes in the cerebral capillaries, and subsequent degeneration of the nerve-cells.

Some of the symptoms, such as the convulsive seizures, the fluctuating conditions of excitement and depression, the remissions, fatty changes in muscle, and profound affection of general nutrition, suggest an intoxication; and Mott holds that these symptoms in the advanced stages of the disease are due to such toxins as choline and neurine resulting from nervous degeneration and circulating in the blood. The condition at the commencement of the disease is however uncertain. General paralysis does not appear to be a tertiary syphilitic disease, as the effects of antisyphilitic treatment are nil, and although a history of syphilis is found in a majority of the cases, there are still examples in which the degenerative change appears to be entirely due to other causes of nervous decay. See INSANITY, Pathology of; and PACHYMENINGITIS.

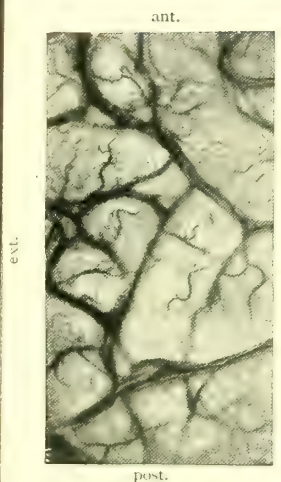


FIG. 1.—NORMAL PIA-ARACHNOID.



FIG. 2.—PIA-ARACHNOID OF GENERAL PARALYSIS.

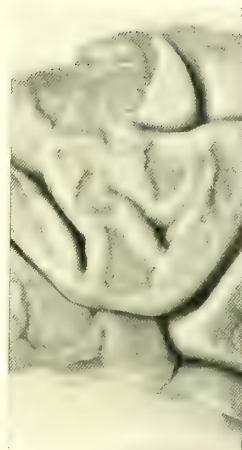


FIG. 3.—DECORTICATION OF GENERAL PARALYSIS.

The opaque and thickened pia-arachnoid of General Paralysis, illustrated in Fig. 2, is usually present over the fronto-parietal region of the brain, but may be almost universal. The worm-eaten cortex of General Paralysis after removal of the pia, shown in Fig. 3, is usually most marked where pia-arachnoid is in contact with pia-arachnoid, namely, in the mid-line below the level of the falx cerebri. In this region the adjacent portions of the pia become granular and finally adhere, and decortication may occur here alone in early cases. In Fig. 3 the erosions are limited to the summits of the convolutions, where the pia mater is most adherent; they are of course artefacts. Possibly the normal movements of the cerebrum, together with the stagnation of the abnormal cerebro-spinal fluid, which probably occurs here, may be largely responsible for this. In more advanced cases decortication may be wide-spread, but is especially prone to occur from *post-mortem* changes.

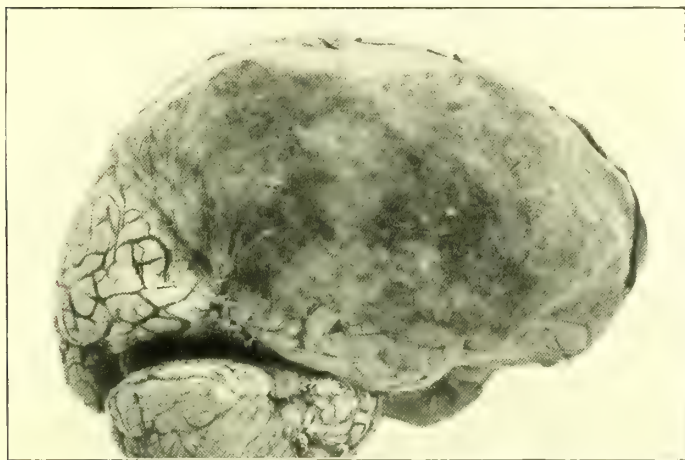


FIG. 4.—SUB-DURAL DEPOSIT. PACHYMENINGITIS HÆMORRHAGICA.

Hereditary delusional insanity with dementia, of 10 years' duration. Age 59 years. A week before death patient gradually became semi-comatose, and continued so till 8 hours before death. During the week his habits were defective and he spoke only twice. No convulsions and no paralysis. 8 hours before death he became comatose. Right pupil 1.5 mm., left 6 mm. Temperature rose rapidly to 105° 8'. At autopsy dura mater was natural but tightly stretched. On right hemisphere was the old hemorrhagic cyst shown in figure, which contained partly organised blood-clot, and was readily separable from dura and pia. On the left side a similar condition existed, but a recent hæmorrhage had occurred in the deposit, amounting to half a pound of recent clot and blood. The left hemisphere was extremely compressed. See p. 985.

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TREATMENT.—At present there is no curative treatment for general paralysis. In the large majority of cases the patient can be properly cared for only in an institution for mental diseases. In a few of the cases characterised by progressive dementia with no excitement or depression the patient can be treated in a private house or a hospital, but even in such cases there is the possibility of the sudden onset of excitement or of symptoms which need the arrangements of an asylum. In the very earliest stage, when perhaps there would be a possibility of arresting the disease, the patient will not as a rule submit to any treatment, and his mental condition is not so disordered as to justify his detention legally. In cases which come under notice very early, complete bodily and mental rest with abstinence from alcohol and from sexual or other excitement should be enforced. Where there is a history of syphilis, or when the disease begins with local paralyses, anti-syphilitic treatment should be pursued, but the results are most disappointing. General paralytics in all stages need most careful supervision to prevent injury to themselves or others, and to guard against the accidents which may arise from convulsive seizures, the passage of food into the larynx, or distension of the bladder. In consequence of brittleness of the bones, great care must be taken in handling the patients, especially if there be excitement. Alcohol should as a rule be avoided, and the bowels should never be allowed to become confined. Fits should be treated on the principles of general medicine. Trephining of the skull and drainage of the subarachnoid fluid has been resorted to under the idea that the symptoms are due to pressure on the brain resulting from its accumulation. In some cases a remission of symptoms has followed this treatment, but the disease has not been arrested. There seems no doubt that the increase of subarachnoid fluid is secondary to brain-wasting and not its cause. The treatment of excitement or depression should be conducted as in other maniacal or melancholic conditions. In the advanced stages the most scrupulous attention must be paid to cleanliness, or bed-sores may rapidly form.

R. PERCY SMITH.

GERMAN MEASLES.—See RUBELLA.

GERMS.—See BACTERIA; and INFECTION.

GEYSERS, The, (1) in Sonoma County, California, U.S.A. Hot springs, with alum, iron, and sulphur; (2) in Wyoming Territory, U.S.A. Thermal waters. See MINERAL WATERS.

GIDDINESS (Sax. *gidig*, turning round).—A synonym for vertigo. See VERTIGO.

GISSHÜBEL, in Bohemia.—Acidulous alkaline table water. See MINERAL WATERS.

GINGIVITIS (*gingivæ*, the gums).—Inflammation of the gums. See MOUTH, Diseases of.

GLANDERS (*glans*, an acorn).—SYNON.: Equinia; Fr. *Morve*; Ger. *Rotz*. Its associated condition is named Farcy; Fr. *Farcin*; Ger. *Wurm*; *Hautwurm*.

DEFINITION.—A contagious febrile disease (associated with the presence of a specific micro-organism, the *Bacillus mallei*); occurring primarily

in the horse, ass, and mule; but transmissible from them to man (generally by inoculation of an abraded surface of the skin); characterised by inflammatory lesions of the nasal and respiratory mucous membranes, and of the lymphatic vessels and glands, with general pyrexia, pains in the joints and muscles, and great prostration, usually accompanied by a pustular cutaneous eruption.

The local manifestations vary in their order of appearance and comparative severity. In cases where the nasal and respiratory mucous membranes are earliest or most severely affected, the disease is designated 'glanders'; but when the lymphatic system first and especially suffers, the term 'farcy' is used. But the two sets of symptoms are usually associated; and whichever may appear first, the other, as a rule, follows. Moreover, it has been shown that the same virus may give rise to either set of symptoms, or to both; and it would seem that the order of their appearance is determined in great measure by the mode of infection and also by the condition and constitution of the recipient.

ÆTIOLOGY.—Glanders is a disease dependent upon a specific micro-organism—the *Bacillus mallei*—which was discovered by Schutz and Loeffler in 1882. The causal relationship of this bacillus to the disease was firmly established by means of experimental inoculations on horses and other animals; and in 1885 Weichselbaum succeeded in isolating the bacillus from a pustule in an acute case of human glanders.

The natural disease is fairly common among horses, asses, and mules. Some carnivorous animals such as lions and tigers are stated to have contracted the disease as a result of feeding upon infected flesh. Oxen and swine, however, are immune. Among the smaller animals, dogs, cats, guinea-pigs, ferrets, moles, and field-mice easily succumb to experimental inoculation with the specific micro-organism, the last named in about three days; rabbits are only slightly susceptible; rats and white mice are immune.

Glanders in the human subject is a comparatively rare disease and is usually met with in males who are more or less constantly employed among horses, and who are therefore liable from time to time to come in contact with diseased animals or the morbid discharges from them, e.g. ostlers, grooms, and knackers.

BACTERIOLOGY.—The *Bacillus mallei* is a facultative anaerobic, non-liquefying, non-motile, non-sporing bacillus, measuring from 0.2 to 0.4 μ in breadth, by 1.5 to 3 μ in length; usually single, but sometimes united in pairs or growing out into long filaments. Individual bacilli, when stained, often have a characteristic beaded appearance due to small aggregations of protoplasm having retained more of the dye than the intervening portions. It stains well with the usual aniline dyes if the preparations are made from young cultures; but with difficulty if the preparation is derived from the morbid tissues or fluids, or if old cultures are used. It is decolourised when treated by Gram's method.

Upon artificial media it grows best when cultivated at the body-temperature (gelatine-cultures incubated at 20° C. yielding but a scanty brownish growth). On glycerine-agar, small whitish transparent colonies develop within twenty-four hours; these after a few days become opaque, cream-coloured, or brownish, and may attain a diameter of 7 mm. On blood-serum, a yellowish-brown

opaque slimy growth forms. In bouillon, its growth is rapid, the medium showing at first a uniform turbidity, but later on a sticky ropy deposit is noticed. Usually from the third day onwards the indol-reaction can be obtained in broth-cultivations. In litmus-milk, its growth is accompanied by the formation of acid, while the medium may or may not be coagulated. The most characteristic growth, however, takes place upon the surface of potato. Upon this medium a moist, slimy, yellowish opaque growth appears in from 36 to 72 hours, resembling honey in appearance; after a time this becomes deeper in colour, and at the end of about a week is reddish-brown, and the substance of the potato has acquired a slate-coloured or greenish hue.

The resistance of the *Bacillus mallei* to antiseptics, heat, desiccation, and direct sunlight is but slight when tested under laboratory conditions; still great care is needed in disinfecting stables &c., as in such situations the virus may remain active for some months.

Mallein.—Mallein is a fluid extract of such of the soluble products of the growth of the *Bacillus mallei* as are unaffected by heat, and is an extremely valuable aid in the diagnosis of equine glanders. The mode of its preparation is somewhat similar to that of tuberculin.

A virulent strain of the glanders-bacillus is grown in glycerine-veal-broth in flat shallow flasks—in order to insure sufficient supply of oxygen—at a temperature of 37° C. for about four weeks. The cultures are then killed, either by exposure to streaming steam (100° C.) for a period of twenty minutes on each of three successive days, or by one exposure to steam under pressure (115° C.) in the autoclave. The turbid broth is next filtered through a porcelain filter—a process which removes all the dead bodies of the bacilli and yields a clear brownish filtrate, containing only the soluble portions of the organisms and the products of their growth. The filtrate is evaporated to about half its bulk, *in vacuo* at a low temperature, and 0·5 per cent. phenol added as a preservative. If the filtrate be still further reduced in bulk, and then thrown into a large excess of alcohol, a precipitate is formed which contains the active principle of mallein. This, after careful drying over calcium chloride, &c., is known as 'dry' mallein.

The use of mallein as a means of diagnosis is restricted to animals, and here its value has been amply demonstrated. The method of its employment may be summed up as follows: The temperature of the suspected animal is taken and noted, then 1 c.c. of mallein, or 0·05 gram. of the dry preparation (previously dissolved in a small quantity of sterile water) is injected subcutaneously in the neck, strict aseptic precautions being observed. In the case of a glandered animal the injection will be followed by (a) a local reaction, and (b) a general reaction evidenced by a rise of temperature. (a) A large, hot, tense, painful oedematous swelling appears at the seat of injection in the course of a few hours. This gradually increases in size, reaches its maximum in about twenty-four hours, when it measures at least four inches in diameter, persists for several days, and does not entirely disappear for eight or ten days. (b) The temperature, which should be observed at four-hourly intervals, begins to rise about four hours after injection, and usually reaches its maximum in from twelve to

fifteen hours. The elevation amounts to 1·5°, 2°, 3°, or even 4° C. above the mean normal temperature. (In the horse the average normal temperature is 37·9° C.) On the other hand, if the animal is not suffering from glanders, the local oedema which appears some hours after injection is slight in amount, soon subsides, and has entirely disappeared within twenty-four hours; and the rise of temperature rarely exceeds 0·5° C., although it may reach 1° C.

Mode of Infection.—Glanders in man is practically always contracted by direct inoculation of virus from a diseased animal. Cases have been recorded of transmission of the disease from man to man, but these are extremely rare.

The infective material is present in the secretions from the nose, in the pus of the glanders-nodules, and rarely in the circulating blood. The *Bacillus mallei* is sometimes eliminated by the kidneys and appears in the urine. The disease may result from the bite of a glandered animal, but usually the virus is derived from the secretion of its nasal mucous membrane, through some pre-existing cut or abrasion of the skin or mucous membrane. Accidental inoculation of the cultivated bacilli has also been recorded among laboratory workers. Experiments by Babes and Nocard have proved conclusively that infection may take place through sound skin, or the uninjured mucous membrane of the eye, nose, or mouth; while the sad death of von Hofman from acute glanders proves that absorption may take place through the mucous membrane of the respiratory tract as the result of inhalation of the virus.

Incubation-period.—The period of incubation varies from one to eight days, but in exceptional cases three weeks or even more may elapse before the constitutional symptoms manifest themselves. The duration of the disease, as well as the order of development of the local affections, varies greatly in different cases. Hence the classification, on the one hand, into *acute*, *subacute*, and *chronic glanders*; and, on the other, into *acute* and *chronic glanders*, and *acute* and *chronic farcy*, but no sharp lines of distinction can be drawn between any of these classes.

SYMPTOMS.—1. *General*.—The earliest symptoms are a sense of general discomfort, fatigue, prostration, and chilliness, with headache, and obscure pains in the muscles and joints. As the disease advances, these pains become more severe, and simulate those of rheumatism. Pyrexia, at first but slight, rapidly becomes established. The pulse is quickened and sometimes full, the skin hot and dry, the tongue foul, the urine scanty and high-coloured; and the patient suffers much from restlessness, sleeplessness, and loss of appetite, often with obstinate constipation. Sometimes the feverishness is intermittent, but more frequently continued, or intermittent at very irregular intervals. Still later, rigors occur—more severe than those which may have occurred at an earlier period—followed by profuse sour perspirations and clamminess of the skin; the pulse becomes very rapid, weak, and compressible; diarrhoea, with fetid stools, succeeds the constipation; the thirst is excessive; respiration becomes more and more difficult and laboured; low delirium, with tremors, is followed by coma; and death ensues from exhaustion. In the acute form, the disease runs its course in an average period of about sixteen days; some cases have terminated fatally within a week, others have been prolonged

for four weeks. In the less acute form the duration may be from six weeks to two months. In the chronic form, in which all the symptoms are less severe, the duration may extend over several months, and in some cases recovery ultimately takes place.

2. *Local*.—Local lesions, varying somewhat in different cases, present themselves in association with the constitutional symptoms.

The wound or abrasion through which the virus has been introduced, or the spot at which it has been applied (for the wound may have healed), becomes inflamed, tense, painful, and surrounded by spreading redness. An ulcer appears, enlarges, assumes an unhealthy corroded, chancroid aspect, and discharges dirty, sanious, and often offensive matters. The lymphatic vessels of the part become inflamed, and present a knotted, cord-like, and subsequently an irregularly nodulated condition—the *farcy-buds*.

The glands are infiltrated and enlarged, and the whole part is swollen and oedematous. The lymphatic glands and vessels of other parts subsequently become affected, but not perhaps to the same extent as in the horse. Resolution and absorption sometimes take place to a greater or less extent; but much more frequently suppuration of low type follows, accompanied by abscess-formation, and the production of foul ulcerating cavities, with hard irregular edges, and fistulae.

At a variable period in the course of the disease—from within forty-eight hours to the end of three or four weeks—a characteristic eruption appears on the skin, especially of the face, chest, and abdomen. This first shows itself as irregularly scattered collections of red spots, 'which are very small and resemble flea-bites, but soon acquire a papular elevation, subsequently rising above the level of the surface, like small shot, and assuming a yellowish colour. They lie each in a kind of hole in the corium, as if the latter had been punched out' (Virchow). They appear to be due to the deposit of some neoplastic material, which subsequently softens and breaks down. By-and-by they become vesicular (some say they are vesicular from the first), and then rapidly sero-purulent, with inflamed livid bases. The surrounding skin is red and swollen, as in erysipelas. When close together, these pustules become confluent, and give rise to irregular ulcerated surfaces, with soft brownish sloughy coating. Large collections of similar deposit in the subcutaneous tissue give rise to hard painful boil-like formations, which break down, and lead to extensive sloughing of the skin and deeper structures, with thick dirty white or sanguineous offensive discharge.

The first mucous membranes involved—especially that of the nose—are sooner or later affected by specific inflammation and ulceration. When the nose is affected (as is always the case in that form of the disease called glanders, either primarily or secondarily), there is a discharge of comparatively thin colourless 'catarrhal' mucus. This soon becomes thicker and coloured; and there is considerable pain, heat, redness, and oedematous swelling about the nose itself, and the adjacent parts of the face. Ultimately the discharge becomes thick, sticky, tenacious, and semi-purulent, of a dirty yellowish or brownish colour, and often stained with blood. In all cases probably there is ulceration of the mucous membrane, following tubercle-like deposits in it; and the ulceration

sometimes extends so deeply as to lead to perforation of the septum, or partial destruction of the turbinated or palatine bones. The ulceration is often limited to the upper part of the nose; and the mucous membrane of the frontal and other sinuses is liable to be similarly affected.

Bronchial catarrh, with rhonchi heard all over the chest, accompanied by severe cough with profuse expectoration, indicates the implication of other parts of the respiratory mucous membrane. The conjunctiva and the mucous membrane of the mouth, gums, fauces, and especially the tonsils, are often affected to a serious extent; so also is the larynx—hoarseness, pain, and difficulty in speaking resulting therefrom. When the lymphatic and cutaneous systems suffer first and most severely, the nasal mucous membrane is not affected until towards the termination of the case; and in some instances death has occurred before the affection of the nose has appeared.

ANATOMICAL CHARACTERS.—The pathological lesions found on *post-mortem* examination are such as might be anticipated from the signs and symptoms manifested during life. To these, however, may be added fluidity of the blood; softness and rottenness of the muscles, with hæmorrhagic abscesses in them (considered by Billroth as characteristic); patches of grey hepatisation in the lungs, or lobular pneumonia; abscesses in the parotid, submaxillary, and cervical glands; and nodules, hard or softening down, in the liver, spleen, and membranes of the brain.

The *Bacillus mallei* may be found in the nodules (especially in such as are comparatively recent), in the mucous membranes, skin, liver, spleen. It has also been found in the blood and various morbid secretions.

DIAGNOSIS.—The diagnosis of glanders in man may be difficult in the early stage, particularly if the history is defective and no external wound appears. But when the disease is fully developed, the signs and symptoms, taken in conjunction with the occupation and history of the sufferer, are sufficiently characteristic. In some rare instances, however, in which the constitutional symptoms have been slight, and the local manifestations have developed slowly, great difficulty has arisen, and the true nature of the disease has not been recognised until after death. Rheumatism, typhoid fever, and pyæmia, and, in the more chronic cases, syphilis and tuberculosis, are the diseases with which it is said that glanders may possibly be confounded. The demonstration of the characteristic bacilli may be regarded as conclusive; they should therefore always be sought for in the morbid secretions and discharges.

The serum-diagnosis of glanders, upon lines similar to those which have given such excellent results in enteric fever, has been attempted, but the results obtained have been too inconsistent to be of any real value.

In doubtful cases some of the *materies morbi* may be injected into the peritoneal cavity of a male guinea-pig. If the material contains the *Bacillus mallei*, the scrotum becomes tense, red, and shining, and purulent inflammation of the tunica vaginalis testis occurs; and if the animal is killed at about the fourth day after inoculation, the bacillus can be easily demonstrated in the pus present between the layers of this membrane.

PROGNOSIS.—The prognosis in glanders is

always extremely grave. On examining the records of 245 cases (including both the acute and chronic forms) of glanders occurring in the human subject, one finds that no fewer than 208 have terminated fatally—that is, a mortality of practically 85 per cent. Of the acute cases, only two or three recoveries have been noted; but on the other hand, Bollinger collected a series of 34 chronic cases, which showed a mortality of only 50 per cent.

It would appear that the more slowly the symptoms develop, and the less their general severity, the greater is the chance of ultimate recovery—a chance which increases with every day the patient survives.

TREATMENT.—Locally any suspicious wound should be freely cauterised or excised, as soon as attention is directed to it. All abscesses and collections of morbid secretions should, as far as possible, be freely incised, and their contents thoroughly evacuated. The resulting cavities and fistulae should be frequently and thoroughly washed out with germicide and disinfectant solutions—perchloride of mercury, chloride of zinc, sulphate of copper, or carbolic acid in safe dilution; and antiseptic dressings should be applied. The operator and those who dress the wounds should wear india-rubber gloves. Inhalations of iodine, or carbolic-acid vapours or sprays, should be frequently used, and the nasal passages and cavities should be thoroughly syringed from time to time with disinfectant solutions, such as dilute carbolic acid (1 in 40) or perchloride of mercury (1 in 1000).

The constitutional treatment should be supporting, stimulating, and soothing, and varied from time to time according to the indications. Quinine in large doses, and perchloride of iron with or without perchloride of mercury, may be useful. But at present, although many drugs have been tried, none has been found having any marked specific effect on the course of the disease, unless indeed exception be made in favour of mercury. Gold reports a case successfully treated by prolonged mercurial inunction. JOHN W. H. EYRE.

GLANDULAR FEVER.—Glandular fever is an acute infectious disease, without any rash, characterised by acute adenitis affecting especially the cervical, but also other groups of lymphatic glands without any constant lesion of the mucous or cutaneous areas from which the lymphatics are derived.

ÆTIOLOGY.—The disease occurs usually in small epidemics affecting the children of a single household, rarely in wider epidemics among the children of a large district. Infants occasionally suffer, adults seldom. It is, however, probable that few children between the ages of two and fourteen years, if exposed to the infection, escape. The lymphatic glands most often and earliest affected are those beneath and to the front of the sternomastoid muscle. The infection is believed to find entrance (1) through the tonsils and pharynx either without producing any local lesion, or at most some slight redness; or (2) through the intestines. Koplik has suggested that the acceptance of this latter alternative might afford an explanation of the fact that the cervical glands on the left side are those first, most severely, and most constantly affected, since the infective agent might spread to them by continuity from the thoracic

duct. The posterior cervical, the inguinal, and the mesenteric groups of glands may also be affected.

SYMPTOMS.—After a period of incubation varying from five to, perhaps, fifteen days, but usually of six or seven days, the child suddenly becomes ill, and is noticed to hold its head stiffly. The temperature is found to be raised (101° – 103° F.); there is nausea, perhaps vomiting and constipation; and the tongue is furred. There is pain on swallowing at about the level of the cricoid cartilage, but no condition of pharynx or tonsils to account for the dysphagia. On palpation of the neck some tenderness will almost always be elicited in the anterior triangle, usually on the left side, and movement of the head causes pain, often very acute. On the second or third day the enlargement of the cervical glands becomes conspicuous, forming an oval swelling beneath and to the front of the sterno-mastoid muscle, usually on the left side, uniform to the eye but commonly resolved on palpation into two or three masses. The glands are tender, often extremely tender, so that the pressure even of the bedclothes is dreaded. The fever about this time reaches its maximum, which may be 104° F. or even more. The pain and tenderness in the glands first affected diminish rather rapidly, but the decrease in bulk is much slower. The glands on the opposite side of the neck may then be similarly affected, and later the inguinal glands, the affection of each succeeding group being as a rule attended by a fresh rise of temperature. The mesenteric glands are affected in one third or one half of the cases; and the mediastinal probably in some, causing paroxysmal cough. The spleen is enlarged in about half the cases. Except in the mildest attacks there is obstinate constipation, which, when convalescence sets in, gives way to diarrhoea of green mucous material. The duration of the fever is uncertain, depending to a great extent on the number of the groups of glands affected; the average may be taken to be about sixteen days. Convalescence is often prolonged, the child being left, on the subsidence of the acute symptoms, anæmic and debilitated. Nephritis, sometimes with hæmaturia, is the most serious, but a rare complication. Epistaxis may occur and be troublesome, and transient synovitis has been observed. Severe mucous diarrhoea occurring about the time of defervescence and continuing during the early part of convalescence is the commonest complication.

DIAGNOSIS.—Except in the presence of a definite epidemic, this can only be made by excluding tonsillitis, pharyngitis, and other local lesions. In severe cases, with marked abdominal pain and tenderness and high temperature, dusky face, and mental dulness, the symptoms resemble those of the early stage of typhoid fever, but the appearance of swelling of the cervical glands will usually clear up the diagnosis on the second or third day of illness. The situation of the swelling will serve to distinguish the disease from mumps, the term usually applied to it by the laity. In presence of epidemic plague it would seem that a diagnosis must depend on the result of bacteriological examination.

PROGNOSIS.—This is good except in the rare cases in which nephritis occurs, when its gravity will depend on the severity of that complication. The glands seldom or never suppurate.

TREATMENT.—Treatment does not cut short the disease. Fomentations with or without belladonna ease the local pain without preventing the affection

of other groups of glands. Antipyrine or, in young children preferably, quinine may be given to reduce the temperature and relieve the malaise during the first few days. A dose of castor oil may be given at the onset, but drastic purgatives should be avoided. Small doses of calomel ($\frac{1}{12}$ to $\frac{1}{8}$ grain) twice or thrice a day for five or six days appear to be useful. The child should be kept in bed and given a light diet. During convalescence, iron and cod-liver oil should be prescribed so soon as the diarrhoea has subsided, and after the first fortnight change of air is to be recommended, but the child should be warmly clothed, especially about the abdomen.

DAWSON WILLIAMS.

GLASS-WORKERS.—See OCCUPATION-DISEASES.

GLAUCOMA (γλαυκός, sea-green).—In its modern acceptation, this word is used to include all the conditions, whether acute or chronic, primary or secondary, which are produced by heightened tension or increased fluid-pressure within the eyeball. The word was originally applied only to those cases of heightened tension in which there is a greenish opaque appearance behind the pupil. See EYE, AND ITS APPENDAGES, Diseases of.

GLEET.—SYNON.: Fr. *Goutte militaire*; Ger. *Nachtripper*.

DEFINITION.—A urethral discharge, milky and viscid, or clear and waterlike, appearing as a drop at the meatus urinarius or as shreds floating in the urine, composed of leucocytes, epithelial cells of various forms, and crystals of phosphates and urates.

ÆTIOLOGY.—In most cases of gleet, though some chronic urethral discharges arise from masturbation and excessive venery, a clap has preceded the discharge by some months or years. In some 10 per cent. of the cases, microscopical examination or cultivation-experiments will reveal the presence of the gonococcus; in the remainder only staphylococci or streptococci may be found.

SYMPTOMS.—In many cases the discharge may escape notice for a time without really disappearing altogether, and again become plentiful without obvious cause. Pain is either absent, or a slight tickling or smarting at the parts inflamed is felt during micturition or at other times. But these symptoms vary so greatly in different patients that little reliance can be placed on them for indicating the seat of the inflammation. Nevertheless, when pain is described as felt in the loins, thighs, or anus, the prostatic portion is generally affected. If the urine is voided in three portions, and the first portion is stocked with floating shreds, while the second and third portions are clear, the discharge is probably formed in front of the prostate. If there be a clear second portion, and a few shreds appear in the third portion, that being also fairly clear, some of the discharge is prostatic. If the third portion is turbid, the uvula of the bladder is inflamed.

Physical signs.—When seeking for the cause and locality of a gleet, the first thing is to palpate the urethra from the meatus to the perinæum to exclude lacunar and perineal abscesses, hard chancre and gumma of the urethra; then it is well to divide the examination of the urethra into two parts: the pendulous and bulbous portions being examined first, the membranous and prostatic portions afterwards.

The precise situations of the lesions in the penile portion are ascertained by passing along the urethra a bullet-sound or bougie (No. 25 or 26 of the French scale), or Otis's urethrometer, if the meatus be too small to admit a bullet large enough to expand the passage to its full extent. As the bullet reaches an inflamed spot a slight resistance is noted, and the patient feels pain when the instrument passes over the inflamed or rigid area. Both resistance and pain cease when the area is left behind. Should the inflammation have existed long enough to produce fibrous bands, the onward passage of the bullet-sound is impeded, or arrested if the bands be too short to permit the bullet to slip past. A smaller bullet must be taken, or the bulb of the urethrometer must be contracted until the obstruction can be passed. By this means the amount to which the normal capacity of the urethra has been lessened is learned. The bullet-bougie is not usually passed farther than the bulb, though it may be pushed to the bladder by a little manœuvring. A tolerably accurate diagnosis can thus be made, though it is always advantageous, and sometimes indispensable, to complete the examination by ocular inspection. This is done by the endoscope; by its means the condition of the obstructing patches may be more precisely ascertained, and, also, the absence or presence of inflammation of the glands of Littre and lacunæ of Morgagni, or of the numerous ducts which open into the membranous portions of the canal. See BLADDER, Examination of.

The conditions causing gleet are: (1) Chronic inflammation of areas of the mucous membrane; (2) Granular patches; (3) Warts; (4) Ulcers; (5) Inflammation of glands and sinuses; (6) Chronic catarrh of the mucous surface and ducts of the prostate; (7) Chronic prostatitis.

1. Chronic inflammation of the mucous membrane.—In this, the most common form of gleet, the interior of the urethra, naturally rosy-pink when not inflamed, is dull red or purplish-red in patches or streaks. It must not be forgotten that the mucous membrane of the urethra, especially of the penile portion, after several attacks of inflammation, may assume a greyish or slatey-red hue, or a brownish stain from absorbed pigment, when the colouring matter of the blood has escaped into the tissues. Here and there viscid grey flocculi of mucus adhere to the inflamed surfaces. The red areas are less elastic than the unaffected parts, and in course of time may develop into bands of stricture-tissue, which more or less diminish the expansile power of the urethra. Common positions for these inflamed areas are the pars cavernosa at two or three inches from the meatus, or at four to six inches from it—i.e. in the bulbous portion of the urethra. But the membranous and prostatic portions are also the seats of chronic inflammation in gleet. In long-standing cases white unyielding areas are disposed among the patches of inflammation. These may remain or disappear. When they are left, some obstruction is caused to the expansion of the urethra—that is, more or less stricture. Usually several months are needed for the patches of inflammation to develop into fibrous bands.

2. Granular patches.—These are excessively common in any part of the penile and bulbous portions of the urethra, while they are not rare in the membranous and prostatic portions. They develop on the areas of chronic inflammation,

especially if these patches have been excoriated or ulcerated; and it is rare not to find them in a case of six months' duration on the third and fourth inches from the meatus. The bullet-bougie reveals their presence by a slight resistance to the passage of the sound, and by a smarting pain felt while the bullet passes over them. When the sound is withdrawn, it is often marked with a little bloody mucus. If the endoscope is then inserted, the granular patches appear as bright carmine-red areas of uneven or velvety aspect, readily bleeding if the instrument be roughly pushed over them. Here and there they are flecked with little dots of greyish mucus, which is very adherent, and which protects the granulation from the action of astringents. In course of time the granulations may form white fibrous scars, or may subside and leave the membrane pale and smooth, but supple as before. Their duration is very long and uncertain, and their cure is tedious.

3. **Warts.**—Warts, which are most commonly situated just within the meatus, may stud the whole length of the urethra. Near the meatus they are arboriform; lower down, sessile, or only slightly pedunculated. They are also frequent in the membranous and prostatic portions. Sometimes a single sessile wart in this position will be the sole cause of a gleet which is troublesome to the patient through its obstinacy, otherwise most harmless.

4. **Ulcers.**—Ulcers are not uncommon in the early stages of chronic urethritis. They appear at any part of the canal, and are indicated by smarting felt at a particular point of the passage during micturition, or when the bullet-sound passes over them. They can be diagnosed with certainty only by the endoscope (*see* BLADDER, Examination of). When seen, they appear as minute, round, shallow excoriations, with sharply defined edges and yellow floor. If left untreated, these shallow erosions may heal and contract into thin and somewhat prominent layers of scar-tissue, disposed sometimes longitudinally, more often obliquely or transversely across the urethra. When fully matured they constitute the 'bridle stricture.'

In persons who have tough well-formed scars of stricture-tissue, ulceration of some parts of this scar-tissue is not uncommon. These ulcers form again and again, and are best treated by thorough dilatation of the strictures, and washing with mild astringent injections.

5. **Follicular sinuses.**—These sinuses, inflamed during acute gonorrhœa, often secrete discharge long after the gonorrhœa has ended. In the anterior urethra generally only a few such crypts are chronically inflamed, but in the prostatic portion many of them secrete a thin turbid fluid. This form of discharge may be considered to be caused by chronic prostatitis, and it will be described farther on. In cases of hypospadias, at the extreme end of the urethra, close to the margin of the meatus in the glans penis, there is in many persons on each side of the orifice a crypt three-quarters of an inch long. These crypts often continue to discharge pus long after the gonorrhœa has subsided elsewhere. Other but shorter sinuses open into the interior of the normal urethra. These follicular sinuses do not form indurated patches in the substance of the urethra; hence they never cause stricture. Temporary narrowing may, however, result from the distension of an inflamed and suppurating sub-mucous follicle, which before the evacuation of its contents may project on the wall of the urethra. The dis-

charge from such a cavity is much more copious than from a simple follicle, and subsides very slowly, though this end is usually reached if there be no stricture in front of the suppurating follicle to impede the easy flow of the urine past the follicle. Usually the bulbous and membranous parts of the urethra are the slowest to recover. One of the commonest causes of discharge is the presence of a lacunar or follicular abscess in the floor of the urethra within an inch of the meatus: this abscess generally discharges into the urethra, but it often discharges externally as well; it is the so-called 'frænal abscess,' and may be so small and so obscured by the firmness of the frænum as to be overlooked by casual observers.

6. **Chronic catarrh.**—In chronic catarrh of the prostatic urethra, with a history of preceding gonorrhœa, there may be a scanty milky-white discharge seen at the meatus when several hours have elapsed after micturition, or the orifice may be sealed by the dry discharge, or there may be only shreds or flocculi in the urine. In addition to the slight discharge, the patient may complain of occasional great increase of it after hard riding, or dancing, or free indulgence in wine, but this does not always happen. The shreds consist mainly of pus-cells held together by mucus; there are also present cells of pavement-epithelium and transitional forms, granular cells, crystals of urates, &c. They have no characteristic value, as similarly formed shreds are washed from granular patches placed in the anterior urethra. Fine filamentous shreds are formed in the folds of the mucous membrane while the urethra is at rest. They are also formed as casts of the prostatic ducts when inflammation has extended into the body of the prostate. These casts are generally built up more of epithelial and granular cells, and less of pus-cells, than those formed in the urethra. The prostatic origin of these shreds is shown by the fact that urine drawn through a catheter from the bladder is bright, clear, and free from shreds. When the mucous membrane of the prostate is exclusively, or principally, affected, the symptoms are very indefinite, and are often limited to the scanty viscid discharge. Hence no diagnosis can be positive without an examination of the urethra throughout its length.

To examine the urethra with a sound, a No. 25 or 26 of the French scale should be used. If a sound of this calibre will not enter the meatus, it is better to cocaineise the mucous membrane within the meatus and nick the orifice at once to a sufficient size; for while the meatus is contracted, the cure of prostatic gleet is almost hopeless. The sound is then passed slowly and gently along the urethra. No pain is felt until the prostate is reached, when a hot or scalding pain is felt, which ceases directly the instrument is withdrawn. The pain is the same whether a curved sound or flexible bougie be used. When the prostate is swollen there is more resistance, but the pain is less scalding to the patient.

If the urethra between the triangular ligament and the orifice into the bladder be inspected, several changes from healthy conditions may be found. The mucous membrane in health is pale pink, or bluish-pink in colour, faintly marked with longitudinal carmine lines, which of course seem to radiate from the centre of the lumen, owing to the funnel-shaped contraction of that part of the urethra which is exposed to view by the endoscope.

When there is chronic inflammation, and the

surface has not become indurated, it is of deep carmine hue, closely beset with small eminences which give it a strawberry-like appearance, a resemblance often enhanced by the minute flocculi of pus which adhere to the surface among the prominent granulations, and simulate the yellowish seeds of the ripe strawberry. These vascular excrescences are at first general over the mucous membrane; but, as time goes on, they subside, and are represented by a few papillary tufts or excoriations, which become permanent if not treated. The congested surface bleeds readily, so it must be well mopped, using tincture of hamamelis, if the bleeding is obstinate, before inspecting it or applying other astringents to the surface. Most commonly, in chronic catarrh of the urethra there are excoriations, sometimes granulations, and, rarely, pedunculated warts. If the inflammation has lasted several months, the veru montanum is often shrunken and white, and the orifices of the ejaculatory ducts are invisible or show as dark greyish dots.

7. Chronic prostatitis.—If there be general congestion of the organ, and not merely superficial catarrh, the symptoms are more pronounced. In such cases there is usually some pain, which is variously described. Sometimes it consists of a sensation of heat extending along the whole urethra, often radiating to the buttocks, but felt most after micturition. In other persons there is often dull pain in the perinaeum, a sense of weight or fullness of the rectum, rather worse by night than by day; aching above the pubes is often complained of. Micturition is called for twice or more by night. By day it is not increased in frequency. When micturition is attempted, the urine is often slow to come, and usually a few drops dribble off after the stream ceases. Walking fatigues easily, and brings on the sense of fullness in the rectum. The finger in the rectum generally finds some swelling and slight tenderness of the prostate. A sound traverses the urethra without causing pain till the bulbo-membranous portion is reached: the instrument is then grasped for a few seconds, to pass on again to the neck of the bladder: here again pain is felt, and resistance is encountered, but this ceases suddenly as the sound enters the bladder, though the pain, of a pressing or squeezing character, still remains. As the instrument is withdrawn, it is expelled rapidly until it is beyond the bulb, where it lies quietly enough and all pain ceases. When, however, the prostatic mucous membrane is also much congested, the pain attending the passage of a sound is, as already mentioned, hot or burning.

TREATMENT.—1. *Inflamed areas.*—When the pain caused by the passage of the exploring sound is acute, the resistance small, and the discharge white and thick, the condition is mainly one of congestion, which is benefited by instillations at the places where the pain is felt. A suitable solution is one of ten to twenty grains of nitrate of silver to the ounce of water, injected by means of a Guyon's bullet-catheter and syringe. This may be repeated every three or four days, while an astringent injection of alum and sulphate of zinc is used in the intervals. If there be grip or hitch as the bullet passes along the urethra, the use of a No. 25 or No. 26 (millimetric scale) steel sound, or even a larger one, twice weekly is requisite. When no grip exists, the last remains of the discharge can sometimes be dried up by using soluble bougies at

night containing chloride or sulphate of zinc in suspension (*'porte-remède Reynal'*) for ten or fourteen nights. Much may be done with injections by the patient himself when the inflammation is limited to that part of the urethra which extends in front of the triangular ligament. When the disease has extended behind that limit, very few patients can apply an injection to that portion, and the treatment must be carried out wholly by the surgeon. The injections ordinarily used for gonorrhœa are not sufficiently strong to affect the deeper parts of the mucous membrane, and solutions having a strength of four or five grains of alum, as much of sulphate of zinc, and of one fourth or one half grain of sulphate of copper to the ounce of water, are needful in this chronic inflammation of small areas.

When congestion has produced induration, or even contraction which does not yield to gradual dilatation, the fibrous band should be divided by a urethrotome of suitable shape, until the urethrometer, expanded to the largest size in which it moves freely along the unaffected parts, travels without hitch or grip past the contracted patches. Valuable aid can be obtained by means of the endoscope, through which the inflamed patches can be attacked with mops, used under the control of the eyesight, soaked in aqueous solutions of the nitrate of silver containing 10 to 20 per cent. of the salt. Indeed, many otherwise intractable affections of the urethra are easily managed if treated endoscopically. See URETHRA, Diseases of.

2. *Granular patches.*—The treatment of granular patches is tedious and uncertain without the endoscope. Something may be done by instillations of solutions of nitrate of silver of fifteen or twenty grains to the ounce of water, while the patient employs a strong injection between the sittings. But the precise application of stronger caustics through the endoscope is far more sure and satisfactory. Brushing the granulations with a 20-per-cent. solution of nitrate of silver, or with a 1-per-cent. solution of perchloride of mercury in alcohol, after scraping their surfaces with a curette, causes these granulations to disappear after four or five applications, and to leave no trace of their presence. The pain of this method is inconsiderable, and need not interfere with the patient's ordinary avocations. Careful dilatation of the contractions is also most necessary during the application of the caustics. In treating granular patches, the writers have abandoned the use of soluble bougies and medicated bougies of all kinds, on account of their feeble and uncertain action.

3. *Warts* yield only to topical treatment. When very small, they may be destroyed by pressing a pencil of cupric sulphate against them. They may be nipped off with scissors through the endoscopic tube. If arboriform and pedunculated, the galvanic cautery is the most effective treatment. It is well to cocaineise the urethra by injecting into it ten minims of a 10-per-cent. solution of hydrochlorate of cocaine, and allowing five minutes to elapse for absorption, before passing the tube of the endoscope through which the topical application is made.

4. *Ulcers* are usually cured by light touches of the copper-sulphate pencil, and by dilating any stricture which may be present.

5. *Follicular sinuses.*—These often continue to secrete pus, which, escaping into the urethra, forms shreds, which appear in the urine. The best treatment is mopping with an astringent solution (10 per

cent. solution of silver nitrate), or pressing into the follicle a fine point of cupric sulphate. After this application, the patient should use a strong injection to wash the sinus freely for some days. In time the sinuses cease to secrete discharge even if not treated. But before this cessation takes place, the sinus may become distended and break again, commonly into the urethra as before; rarely the collection of matter may penetrate into the erectile tissue and through the fibrous tissue to the skin, and so reach the surface of the body. When this threatens, the abscess should be promptly incised; it usually closes after evacuation without further trouble.

6. *Chronic catarrh* of the prostatic urethra is little benefited by general treatment or by drugs. Abstinence from stimulants, violent exercise, and sexual excitement is requisite; and when the secretion of pus is abundant, the specifics for catarrh, such as copaiba, cubebs, and buchu, are beneficial. But when there is little more than a shreddy discharge in the urine the proper treatment is topical. The remedies are local instillations, by an Ulzmann's curved catheter-syringe, of solutions of ten, fifteen, or twenty grains of nitrate of silver to the ounce of distilled water directly on to the inflamed surfaces, made at intervals of five or seven days, the length of time being decided by the amount of reaction caused by the instillation. All soreness and increase of discharge which follows the caustic application should subside before another is applied. Usually several such instillations, with solutions increasing in strength, must be made to obtain a cure.

The use of the endoscope renders the treatment more certain and expeditious, as in treating similar patches in the anterior portion of the urethra. By this means the inflamed areas can be distinctly seen, and the remedy applied directly to them. Useful astringents in which to dip the mops are tincture of iodine, solution of perchloride of iron of the British Pharmacopœia, of mercuric chloride (1 per cent. in spirit), or of silver nitrate (10 or 20 per cent. in water). These applications must be repeated, like the instillations, at intervals of a week or more, according to the amount of irritation they set up. Often, under most favourable conditions, the cure is slow and tedious, but it is not the less certain, and may always be attained with patience.

7. For *chronic prostatitis*, when accompanying catarrh of the prostatic urethra, the treatment required is that needful for prostatitis. The general treatment is here of great value, including moderate exercise without fatigue, careful restriction of the diet to unstimulating food, tonics if required, removal of constipation, regular bathing in tepid or cold baths, occupation for the mind without lascivious or erotic excitement, and, when all congestion has been thus removed, cold injections into the rectum. Also at this stage the passage of full-sized steel sounds, and instillations of solutions of silver nitrate into the previously cocainised prostatic urethra, are among the most effective remedies for this troublesome ailment. Before caustic injections are used it is well to empty the bladder of urine, and to inject eight or ten ounces of some bland fluid, such as cold boiled water, or cold saturated aqueous solution of boric acid, with which the patient may flush out the urethra after the caustic solution has taken effect. This precaution prevents much of the soreness which invariably follows the use of strong astringents in this region. The patient should also keep his bed, or at least his room, for a couple of days after each cauterisation,

to lessen the probability of an acute inflammation of the prostate or of the uvula vesicæ following the injection, of which there is always some risk.

CHARLES STONHAM.
CHARLES GIBBS.

GLEICHENBERG, in Austria.—Muriated alkaline waters. See MINERAL WATERS.

GLÉNARD'S DISEASE.—See ENTEROPTOSIS.

GLIOSIS.—See SYRINGOMYELIA.

GLOBULINURIA.—Albuminous urines almost always contain both serum-albumen and paraglobulin. The relative amounts of these two proteids vary widely in different urines, but only very exceptionally is one or other of them practically wanting. In some cases examined by Czátary the proportion of paraglobulin to serum-albumen was found to be lower in the urine than in the blood and serous effusions of the same patients. This relatively small excretion of globulin is very probably due, as Halliburton has suggested, to the larger size of its molecules, which opposes an obstacle to their passage into the urine. If this be so, a high proportion of globulin should indicate a more severe renal lesion.

The clinical significance of variations observed is not yet fully made out, but it would appear that in cases in which the kidneys are the seats of lardaceous disease the proportion of paraglobulin tends to be unusually high, whereas Czátary found the lowest relative amount in cases of granular kidney, and of the passive renal congestion of cardiac disease. Fibrinogen, which is also a member of the globulin-family, is probably an occasional constituent of urine, and in a remarkable case recorded by Byrom Bramwell and Noël Paton a globulin was abundantly present which was deposited in crystalline form when the urine was allowed to stand.

A rough notion of the amount of globulin present may be obtained by noting the degree of turbidity produced when the urine is allowed to fall, drop by drop, into distilled water, but for more accurate determinations advantage is taken of the precipitation of globulin by saturation with magnesium sulphate, after the urine has been rendered neutral or faintly alkaline.

A. E. GARROD.

GLOBUS HYSTERICUS (*globus*, a ball; *hystericus*, connected with hysteria).—SYNON.: Fr. *Globe hystérique*; Ger. *Hysterische Kugel*.—A subjective sensation experienced by hysterical patients, as of choking, or of a ball rising in the throat. See HYSTERIA.

GLOSSALGIA (γλῶσσα, the tongue; and ἄλγος, pain).—Pain in the tongue. See TONGUE, Diseases of.

GLOSSITIS (γλῶσσα, the tongue).—Inflammation of the tongue. See TONGUE, Diseases of.

GLOSSO-PHARYNGEAL NERVE, Diseases of.—The glosso-pharyngeal nerve is distributed to the back of the tongue and the soft palate, the upper portion of the pharynx, the Eustachian tube, and the tympanum; and also to the stylo-pharyngeus, the middle constrictor of the pharynx, the levator palati, and the zygus uvulæ muscles. But the nerve is connected with the fifth, facial, and pneumogastric nerves, and with the pharyngeal plexus; it is

certain that many of its terminal branches merely distribute fibres from these other nerves; and the early ideas of the function of the glosso-pharyngeal, founded on its terminal distribution, are widely erroneous. Although its twigs go to the structures that subserve taste on the back of the tongue, it is certain that these, with those for common sensation in the same part and in the fauces, come from the fifth nerve; and that the fibres that innervate the palatine muscles are derived from the spinal accessory.

Paralysis.—Very little is known of the precise effects of paralysis limited to the glosso-pharyngeal nerve, since it is rarely paralysed alone. The middle constrictor of the pharynx is, however, paralysed, and the mucous membrane of the pharynx is rendered insensitive. From their position, the fibres of origin are commonly damaged in conjunction with fibres of the hypoglossal, spinal accessory, and pneumogastric nerves. The common causes of disease in this situation are meningitis, syphilitic and other growths, and bone-diseases. The nerve may also be paralysed from disease of its nucleus of origin in the medulla oblongata, and then commonly suffers in association with the nerves to the larynx, the tongue, and often the lips (see LABIO-GLOSSO-LARYNGEAL PARALYSIS). The sensory part of the nerve may then escape, and only the motor part be paralysed, the chief effect of which is paralysis of the pharynx. The causes of paralysis from disease of the nucleus are slow degeneration, softening, hæmorrhage, and the effects of diphtheria.

Spasm.—Nothing is known of separate spasm in the muscles supplied by the glosso-pharyngeal nerve. In conjunction with the other motor and sensory nerves to the pharynx, it takes part in the production of the spasm of hydrophobia, and in some hysterical phenomena.

TREATMENT.—The treatment of disorders of the glosso-pharyngeal nerve is never special, but always that of the cause, and is sufficiently described in the several articles which deal with the above-mentioned ætiological conditions. W. R. GOWERS.

GLOSSO-PHARYNGEAL PARALYSIS.—A synonym for labio-glosso-laryngeal paralysis. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

GLOSSY SKIN.—See NEURITIS, MULTIPLE.

GLOTTIS, Diseases of.—See LARYNX, Diseases of.

GLYCOSURIA (γλυκός, sweet; and οὐρον, urine).—A condition of urine in which sugar is present; generally used as a synonym for diabetes mellitus. See DIABETES MELLITUS.

GLYCURONIC ACID.—Glycuronic acid is a substance allied to the carbohydrates, and represented by the formula $C_6H_{10}O_6[COH-(CH.OH)_4-COOH]$. It occurs as an abnormal constituent of urine, as the result of ingestion of certain chemical substances, chief among which are camphor, chloral hydrate, butyl chloral hydrate, morphine-salts, nitro-benzol, and some of the quinine-derivatives. After anæsthesia from chloroform (but not ether) its presence can be detected in the urine of the patient.

Glycuronic acid in urine is liable to be mistaken for grape-sugar (dextrose), since it is capable of reducing alkaline copper-solutions (Fehling's test)

(see DIABETES MELLITUS; and LIFE-ASSURANCE). Dextrose can, however, be readily distinguished from glycuronic acid by its behaviour with yeast, glycuronic acid yielding a negative result when so treated. When pure it is not crystalline. It is readily soluble in water, much less so in absolute alcohol, and very sparingly in ether. Its pure solutions are dextro-rotatory, but less so than dextrose.

JOHN HAROLD.

GOITRE.—SYNON.: Bronchocele; Thyrocele; Derbyshire Neck; Fr. *Goitre*; Ger. *Kropf*; *Struma*.

DEFINITION.—A term in general use for enlargement of the thyroid gland. Here we shall only consider simple hypertrophy or parenchymatous enlargement, adenoma, cysts, and the various combinations of these, with or without fibrosis, which are commonly included under the name of goitre. Other enlargements will be considered elsewhere. See EXOPHTHALMIC GOITRE; THYROID GLAND, Diseases of.

ÆTIOLOGY.—(a) *General.*—The ætiology of goitre varies according to the nature of the enlargement. Simple hypertrophy occurs not infrequently in both anæmic and pregnant women, and about the time of puberty in both sexes. In such cases the enlargement probably occurs in the first instance in response to a demand for an increased supply of the secretion of the gland. Of the exciting cause of adenomatous and cystic goitre, we know nothing; the cause of endemic goitre will be considered later.

(b) *Sex.*—Women in this country are much more liable than men to suffer from all forms of goitre, just as they are more prone to develop other diseases of the thyroid gland. The proportion between the two sexes varies in different parts of England from seven up to forty-four to one. It has been suggested that this is on account of women being more frequently water-drinkers, for in India it has been noticed that both sexes suffer alike. Besides there appears to be some association between the enlarged gland and the uterine functions.

(c) *Age.*—Congenital goitre occurs but rarely. During the time of puberty the disease frequently first attracts attention, although it may occur at any time of life up to fifty years of age.

(d) *Heredity.*—In some families goitre is hereditary; in others, it is only apparently so owing to different members of the family living in a district where goitre is endemic.

(e) *Locality.*—Endemic goitre, a special form of parenchymatous enlargement which is liable to undergo cystic degeneration, occurs in certain districts, and is evidently due to some localised cause. Most frequently these districts are mountainous regions with deep valleys in which goitre is prevalent. In England goitre is endemic in the upper valleys of the Pennine Range, as far south as the Peak in Derbyshire—hence the name Derbyshire Neck—and also in the Cotswold Hills. Among the continental countries in which it prevails are France, mostly in Savoy; Germany, mostly in the Black Forest; Austria, mostly in Styria; Northern Italy, mostly about the Alps, as at Aosta. In Switzerland goitre is very prevalent, in some parts of the Cantons of Friburg and Berne Bircher has found as many as 80 per cent. of the recruits to be goitrous. In Asia goitre is endemic

in the Himalayas, in the Altai Mountains in Siberia, and in the hilly districts of China. Goitre is, however, not only indigenous to mountainous districts, but occurs also in open country as in the plains of Lombardy, and in the Indian Punjaub.

(f) *Water-supply*.—Of the various local conditions present in goitrous districts it is now evident that goitre is due in a large number of the cases to an impurity in the drinking-water. Snow-water was formerly considered to be the cause, but this opinion is met by the statement that goitre does not exist in Greenland or Lapland, and that it is prevalent in Sumatra where there is never any snow. The influence of drinking-water, or more strictly speaking of some impurity in the water, in producing goitre is shown by the following facts. The migration of a healthy family into a goitrous district has been followed by the development of the disease in several members of it; whereas removal from such a district has prevented the further development of the disease in a family already affected. A change in the water-supply has been followed by a rapid diminution in the number of cases of goitre. Thus in Bozel in Savoy out of a population of 1,472 in the year 1848 no less than 900 were goitrous and 109 cretins. A fresh supply of water was then obtained, in consequence of which both goitre and cretinism diminished so rapidly that in 1864 there were only 39 cases of goitre and 58 cretins. On the other hand an actual epidemic of goitre has followed the introduction of a new supply of 'infected' water into a village previously free from the disease, and has also occurred in a regiment of young soldiers when moved into a goitrous district. Drinking the water of certain wells on the Continent is well known to produce goitre and is intentionally and successfully taken for this purpose by men who wish to escape military service.

This peculiar property of certain waters has been attributed to iron pyrites, copper pyrites, sulphate or carbonate of calcium or carbonate of magnesium in the water according to the geological formation of the district. There is, however, no positive evidence to show that any of them can produce goitre. Whatever the active agent may be, its activity is destroyed by heat, as the water is rendered harmless by boiling. This important fact renders it probable that some living micro-organism present in the water may be the actual cause of endemic goitre. Further than this we cannot at present go, as no such micro-organism has yet been isolated.

ANATOMICAL CHARACTERS.—In simple hypertrophy of the thyroid gland, the enlargement is uniform, retaining the shape and consistence of the normal gland. In endemic goitre there is at first general uniform enlargement. At a later stage multiple cysts may develop, formed from the normal follicles of the gland by their distension with colloid material, the epithelial lining degenerating as the cysts increase in size. Adjacent follicles may become fused, and so some of the cysts may reach a considerable size. The cyst-walls are formed by the inter-lobular septa and the capsule of the gland. There may be a great increase of fibrous tissue in some cases, so that goitres of great size occur composed of gland-tissue, fibrous tissue, and cysts—fibro-cystic goitres. Apart from simple hypertrophy the great majority of goitres which occur in this country are due to the growth of adenomata in the gland. An adenoma of the thyroid may be single or multiple. Its structure is similar to that of the

gland itself, and it is provided with a distinct capsule. Confined at first to one part of the gland, it may gradually extend into other parts till the enlargement which was localised at first may in its later clinical features closely resemble a general parenchymatous enlargement. Such adenomata often become cystic, and in this way the single cysts of the thyroid gland which are of frequent occurrence are formed. Like the adenomata from which they have developed, these cysts can be separated from the gland and shelled out complete. Their walls may be thick, and the contents are brownish in colour, and as Victor Horsley points out they have a tendency to contract adhesions to surrounding parts. Hæmorrhages into the substance of an adenoma, and especially into a cystic adenoma, are not uncommon.

SYMPTOMS AND COMPLICATIONS.—In slight cases a swelling in the thyroïdal region which moves upward during deglutition, and a sense of fulness in the neck, are the only symptoms present. The character of the swelling varies according to the nature of the goitre. In simple hypertrophy there is a smooth firm enlargement of the whole gland, which retains its original shape. If such a parenchymatous goitre becomes cystic or fibrous, the shape and consistency become irregular and vary according to the size and situation of the cysts and the amount of fibrous tissue which is developed. An adenoma forms a smooth well-defined firm tumour, which may be accompanied by some general enlargement of the gland as well. A cyst forms a smooth globular swelling, in which fluctuation may be felt. In order to detect this sign it is most important to palpate the tumour with four fingers at once, as advised by Horsley, so as to steady it from every direction; even then it is sometimes difficult to distinguish between an adenoma and a cyst. In such cases an exploratory puncture may be made in order to ascertain if a cyst is present. The superficial veins in the front of the neck are often enlarged and prominent over a goitre, and an adenoma or cyst of one lobe may cause considerable displacement of the larynx, trachea, and blood-vessels in the neck. Various secondary symptoms may be caused by a goitre by pressure on surrounding parts. Added to a sense of fulness there may be a feeling of dragging or constriction about the throat. More serious symptoms than these are, however, sometimes present, namely dysphagia and dyspnœa. The former rarely occurs except when the tumour has reached an immense size or when it presses on the œsophagus, as happens when the lateral lobes meet behind the gullet. Dyspnœa is a more common symptom which occurs in both a persistent and paroxysmal form. A dyspnœa of gradual onset is the more common, and may be caused by the pressure of a goitre on the front or side of the trachea. The trachea may also be compressed by an extension of the goitre behind it or by an adenoma extending down into the mediastinum and forming a substernal goitre. Dyspnœa may be due even to pressure on the recurrent laryngeal nerves causing paralysis of the abductors of the vocal cords. All goitres are liable to temporary enlargement, especially during menstruation and pregnancy, such enlargement being accompanied by an increase in any pre-existing dyspnœa. In addition to this persistent dyspnœa, severe paroxysmal attacks occur in cases of large goitre. These attacks come on suddenly, most

frequently during the night, causing great distress, and lasting from five to fifteen or even thirty minutes, after which they pass off. Death may, however, occur suddenly during the attack. The voice in cases of large goitre of long duration is often hoarse and toneless. Some cases of endemic goitre are associated with cretinism, which so frequently develops in the children of goitrous parents. The association of cretinism with a goitre indicates that an advanced stage of goitrous degeneration of the thyroid gland has been reached, in consequence of which it can no longer supply an adequate amount of its secretion (*see* CRETINISM). In some cases of goitre symptoms of exophthalmic goitre supervene (*see* EXOPHTHALMIC GOITRE). Hæmorrhage takes place, not uncommonly into a cyst or even into the substance of an adenoma, causing a rapid enlargement of the goitre with pain from the increased tension. Absorption of the blood gradually takes place, and the goitre diminishes again in size. A goitre may become acutely inflamed during the course of an acute disease or as the result of an injury. It then becomes swollen and tender, and there is pain in the neck with fever. If the goitre is already large, there may be great dyspnoea and cyanosis. The attack may terminate by resolution, or by suppuration, and the formation of one or more abscesses.

DIAGNOSIS.—A simple hypertrophy of the thyroid gland can be recognised by its situation, shape, uniform consistence, and upward movement when the patient swallows; an adenoma by the localised swelling, and its smooth, rounded, and well-defined outline. The presence of a cystic goitre can be ascertained by the fluctuation within its walls, care being taken to fix the tumour during palpation, as already described. Any doubt can be cleared up by passing a hollow needle into it. The fluid thus withdrawn may be mixed with blood, and when a cyst has been emptied it often happens that blood will flow freely from the walls of the cyst till it is filled.

The diseases which simulate goitre are malignant disease of the thyroid gland; calculus embedded in its substance; lymphadenoma; aneurysm; and fatty and other tumours of the neck. A leading point in making a diagnosis is to ascertain, by directing the patient to swallow, whether the swelling is attached to the trachea. In cases of malignant disease and lymphadenoma, the history and general condition of the patient will give an important clue. It must be remembered that malignant disease may develop in a previously simple goitre. When a goitre closely overlies the carotid artery, it may simulate an aneurysm, owing to the forcible pulsation conveyed through its substance.

PROGNOSIS.—The prognosis in goitre is favourable. The occurrence of death from suffocation, due solely to the effects of a goitrous enlargement, is rare. It occurs in those countries where little treatment is tried, and where the goitres attain an immense size. The disease is more likely to endanger life when the goitre passes behind the trachea or behind the sternum. Simple hypertrophy of the gland in young adults is most amenable to medical treatment, while adenomatous and cystic goitres can, as a rule, be easily removed by an operation. Goitres have been known to disappear without any treatment. This result may be due to the removal of the affected person from

endemic influences; or it may happen when the enlargement has arisen during pregnancy.

TREATMENT.—In the treatment of endemic goitre the general rules requiring attention are removal of the patient from the goitrous district, or complete abstinence from the drinking-water, unless it has been previously boiled; also from wearing anything tight round the neck, carrying weights, and ascending hills.

As simple hypertrophy of the thyroid gland in adolescents generally occurs in response to a physiological demand for an increased supply of its secretion, a marked diminution in size can be brought about by supplying the secretion from an external source. In other forms of parenchymatous goitre this line of treatment is also useful.

This treatment is readily carried out by giving extract of thyroid. Dry thyroid substance (Thyroideum Siccum, B.P.) may be given in one to three grain doses in a powder, pill, or tablet, or liquid extract of thyroid (Liquor Thyroidei, B.P.) in doses of five, ten, or fifteen minims in water twice or thrice daily. The frequency of the pulse should be recorded daily, so that the dose may be diminished if any undue acceleration beyond ten or twenty beats a minute should occur. By this method of treatment the circumference of the neck in one case was reduced from $16\frac{1}{2}$ in. to $13\frac{3}{4}$ in. in three weeks. When the full advantage of the treatment has been obtained, it is advisable to continue a single daily dose of the remedy for several weeks or even a couple of months, to prevent a return of the former enlargement. Iodine has long been used both internally and externally in the treatment of goitre, its efficiency being greatest in simple hypertrophy and in the earlier stages of endemic goitre. It has been shown by Baumann that thyroidal secretion contains iodine in an organic combination, *thyroidin*, so that the action of iodine or its combinations in the treatment of goitre may be to a certain extent supplemental and so analogous to that of thyroid extract. Five to ten grains of potassium iodide in solution, or five minims of tincture of iodine in water, or from one half to two grains of iodoform in a pill may be given three times a day. In simple hypertrophy blistering with liquor epispasticus, painting the surface with tincture of iodine, and inunction of iodine ointment, or of a mixture of one part of it with two or three of potassium iodide ointment, are useful methods of local treatment. Biniodide of mercury ointment smeared over the enlargement, the patient being then made to sit with his neck exposed to the rays of the sun for many hours, has proved an effectual remedy in India; this treatment is useful in this country even without the assistance of the rays of a tropical sun. Formerly, injections of iodine were not infrequently employed in the treatment of parenchymatous goitre, and in some cases with beneficial results. Twenty or thirty minims of a 1-in-12 solution of iodine in absolute alcohol were slowly injected, under local anaesthesia, twice a week into the substance of the goitre, care being taken to avoid transfixing the trachea or any blood-vessel with the needle of the hypodermic syringe which was usually employed. This treatment is not free from danger, as no less than sixteen cases in which death occurred as a result of the injections have been collected by Heymann. Victor Horsley has shown experimentally that death in such cases is due to the passage of the injection-fluid directly into a large blood-

vessel. This treatment often fails, and is therefore not to be recommended.

An adenoma, if it is small, and causes no inconvenience, may require no treatment, unless the patient wishes to be rid of it. If it is increasing in size, or causes inconvenience by pressure on the surrounding parts, or dyspnoea by compression of the trachea, it should be removed. If a cystic adenoma has been distended once or twice by hæmorrhages, removal is usually advisable. An adenoma can be readily removed by shelling the tumour out of the gland, which is thus left behind entire. The operation is a straightforward one, and the wound heals in a week.

In cystic goitre surgical treatment is also required. In cystic degeneration of a parenchymatous goitre the cysts are usually multiple, so that it is necessary to remove one lobe of the gland, and in some cases even the whole gland. As, however, total thyroidectomy is followed by the development of secondary myxoedema, this operation should, except in the case of malignant goitre, be avoided, if possible. When total extirpation is really necessary, the onset of myxoedema can now be prevented by giving thyroid extract, say, ten minims of liquor thyroidei, each night for the rest of the patient's life. In the case of a single cyst or cystic adenoma, the cyst should be enucleated just in the same way as an adenoma, from which it has developed. The older methods of treating thyroidal cysts by tapping, by incision and drainage, or by injections of perchloride of iron, should be no longer employed, as they are both risky and inefficient.

PUGIN THORNTON.

GEORGE R. MURRAY.

GONAGRA (γόνα, the knee; and ἄγρα, a seizure). An attack of gout in the knee. See **GOUT**.

GONARTHROSIS (γόνα, the knee; and ἄρθρον, a joint).—Inflammation of the knee-joint. See **JOINTS**, Diseases of.

GONORRHŒA (γονή, seed; and ῥέω, I flow). **SYNON.**: Clap; Blenorrhagia; Fr. *Chaudefisse*; Ger. *Tripper*.

DEFINITION.—A contagious purulent inflammation, affecting primarily, in men, the urethral, and in women the lower vaginal and vulvar mucous membrane. Occasionally the conjunctival and rectal mucous membranes, to which the nasal has been added on doubtful evidence, are attacked by gonorrhœa. Certain joint-affections, endocarditis and other lesions, to be subsequently mentioned, may also complicate the disease. See **GONORRHŒAL ARTHRITIS**.

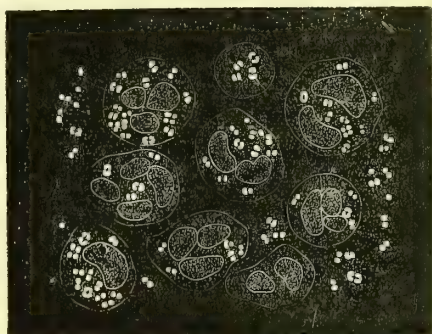
ÆTIOLOGY.—True gonorrhœal inflammation is due to inoculation of a specific coccus, the *Gonococcus* of Neisser, usually by direct contagion from a surface already the seat of gonorrhœa. Membranes covered by pavement-epithelium, and possessing a rich lymphatic supply, are the most readily affected; those covered by columnar epithelium, e.g. the rectum, are not so liable to become diseased; while the mucous membrane of the mouth, stomach, and air-passages appears to be exempt. Healthy mucous membranes are as liable to contract the disease as are those the seat of some morbid change.

In women the arrangement of the genital mucous membrane favours the retention of secretions coming in contact with it. Among other causes favouring the development of gonorrhœa may be

mentioned the presence of gleet, alcoholic excess, and previous attacks. Men with long or moist foreskins, in first attempts at copulation, and with slight hypospadias, are specially liable to contract gonorrhœa.

INCUBATION PERIOD.—The symptoms of gonorrhœa are not usually manifested until four or five days after infection: presumably the contagion needs this time for multiplication and development before it is capable of exciting inflammation. According to circumstances, the incubative stage may be shortened or prolonged, ranging from two to eight or ten days. As a general rule, the shorter the incubation-stage the more severe is the disease. The period of incubation in the case of gonorrhœal conjunctivitis is shorter than that of urethritis, varying from six to seventy-two hours, the average time being about forty-eight hours.

MORPHOLOGY.—Gonococci are ovoid in shape, and average 0.7μ in the long diameter by 0.5μ in the short; they are arranged as diplococci and tetrads, i.e. in pairs, and groups of four; contiguous cocci having their adjacent sides flattened or even slightly concave, each element then resembling a



Micrococci of Gonorrhœa in the discharge from a case of acute gonorrhœa. The cocci are seen mostly as pairs or groups of four, and are mainly in the protoplasm of the pus-cells. $\times 850$.

coffee-bean or kidney in shape. Some observers have described a capsule surrounding the gonococcus, but none have yet succeeded in demonstrating this structure in preparations made from the pus.

In gonorrhœal pus, the cocci usually form colonies within the substance of the pus-cells and are arranged in pairs, fours, and their multiples; their presence has also been noted within the cell-nuclei; sometimes, and especially in severe cases, the gonococcus multiplies freely in the liquor puris.

STAINING REACTIONS.—The gonococcus stains readily with all the usual aniline dyes, especially with gentian-violet and fuchsin, and is as readily decolourised; it does not retain the stain when treated by Gram's method, this feature at once differentiating it from the ordinary pyogenic cocci.

BIOLOGY.—The gonococcus is a non-motile, facultative anaerobic, highly parasitic coccus, reproducing itself by fission only, the lines of fission lying at right angles to those of the parent-cocci. It can be cultivated upon artificial media outside the body, but it is essential that human serum (blood-serum, pleuritic effusion, &c.) should enter into the composition of the medium, no growth

whatever taking place when cultivations are made upon the ordinary media of the laboratory.

Development of the gonococcus only takes place within a small range of temperature, growth ceasing below 25°C . and above 40°C .—the optimum temperature being undoubtedly 37°C . Its thermal death-point is 45°C .

The resistance of the gonococcus to the action of weak germicidal fluids, direct sunlight, and desiccation (when spread in thin layers) is extremely slight; the specific properties of gonorrhœal pus being destroyed by exposure to a temperature of 60°C . for ten minutes. In like manner its vitality in artificial cultures is small, and it is necessary to transplant at intervals of not more than a week, in order to ensure vigorous growth. On the other hand, the virulence, or rather the pathogenic properties of the gonococcus do not appear to be affected by these frequent transplantings, for Wertheimer has produced a typical gonorrhœa by inoculating gonococci of the thirtieth generation.

CULTURAL REACTIONS.—The media employed for the cultivation of the gonococcus are (a) equal parts of human blood-serum, and 2 per cent. peptone-agar (Wertheimer), or (b) a mixture of hydrocele-fluid—or pleuritic fluid—and 2 per cent. peptone-glucose-agar, in the proportion of 1:2 (Heiman).

De Christmas has recently advocated the use of rabbit's blood-serum, but this does not give such good results as the above-mentioned media. To isolate the gonococcus the method suggested by Abel is reliable and convenient, and may be thus briefly described. Three plates (Petri dishes) are poured with ordinary 2 per cent. peptone-agar, and as soon as set, but while still hot, a drop of blood taken with all aseptic precautions from the finger or lobe of the ear is deposited by means of a sterile pipette on the centre of the agar-surface of each plate. By means of a sterile brush or platinum loop, a minute quantity of gonorrhœal pus is mixed with the drop of blood on the first plate and then smeared all over the surface of the agar. The moistened loop or brush is transferred quickly to the second plate, and the process repeated, and finally the third plate is inoculated in like manner. After incubation at 37°C . for twenty-four to thirty-six hours, colonies of the gonococcus may be picked off the second or third plate, and subcultivated upon one or other of the serum-agars.

Serum-agar 'Plate' or 'Slant' Cultures.—Growth of the gonococcus appears at twenty-four hours as discrete, raised, translucent colonies, resembling minute drops of water; these gradually increase in size during the next two or three days, attaining a diameter of perhaps 1 to 2 mm. The colonies then show irregular scalloped edges, and finely granular shining surface, and are greyish white or faint buff in colour.

Colonies in the depths of the medium are spherical or oval when young, but later on become irregular in outline, and sometimes throw out short processes.

Microscopical preparations from growths upon artificial media show cocci similar to those observed in gonorrhœal pus, but the tetrad form is distinctly more common, and if the growth is more than two days old, involution-forms are the rule rather than the exception.

PATHOGENESIS.—The ordinary laboratory animals are immune to experimental infection with

the gonococcus, whether inoculated into the urethra, vagina, conjunctiva, or elsewhere; occasionally, however, a purulent non-fatal peritonitis is produced in mice and guinea-pigs, as the result of intra-peritoneal injection of serum-agar cultures of the gonococcus. De Christmas, too, states that he has obtained a toxin from cultures of this organism, which proves fatal when large doses are injected intravenously into the rabbit.

Inoculations of the gonococcus into the healthy human urethra, on the other hand, almost invariably yield positive results.

Mode of invasion.—The gonococcus penetrates and multiplies in the epithelial cells, and advances into the underlying lymphatic spaces. Here it rapidly proliferates, exciting inflammation and exudation, the migrated leucocytes containing the microbes. The process advancing, serous effusion passes into the substance of the corpus spongiosum, to the glands and lacunæ of the urethra, to the erectile tissue of the corpora cavernosa, &c., and lights up inflammatory mischief in the parts affected. The facility with which the gonococci pass along the lymphatics explains why certain portions of the urogenital canal, e.g. the navicular fossa, bulb, neck of the bladder, and epididymis, are the parts most commonly affected. Moreover, the microbes, aggregated into masses, may be carried away in the lymphatic and blood-currents as infective emboli: this would explain the occurrence of gonorrhœal rheumatism, endocarditis, &c.

Adopting the theory of phagocytosis, it may be supposed that the white cells, and others acting as phagocytes, are hostile to the development of the gonococcus, and this may serve, in some measure, perhaps, to explain the fact that different persons and organs are not equally affected by the gonorrhœal poison; in certain persons or organs their innate strength of resistance to infective poisons may be sufficient to destroy the microbes or arrest their development. When the proliferation of the poison takes place mainly in the pus-cells, the microbe has but little power, and consequently the course of the disease is mild; but if the organisms develop freely in the liquor puris the course is usually severe, and specially marked by involvement of lymphatic structures, giving rise to lymphangitis and bubo.

Still there are certain predisposing causes which may exert an influence on the development of the poison, and so augment or modify the severity of the disease.

First, there is no doubt that the urethral mucous membrane differs greatly in its susceptibility to inflammation in different individuals. It is, moreover, asserted that both sexes grow accustomed to the contagious secretions of the same individual, so that they neither infect nor are infected by their usual companions, while they readily receive disease from, or impart it to, strangers.

Gonorrhœa differs so considerably in the two sexes, owing to the difference in the seat of the affection, that it will be advisable to describe it in the male and female separately.

(a) **Gonorrhœa in the Male.**—**ANATOMICAL CHARACTERS.**—The fossa navicularis, rich in follicles and their ducts, is the primary seat of gonorrhœa in the male. The organism passing into the ducts remains in security during the period of its development and multiplication, and penetrates into the deeper tissues and lymphatic network. As

the disease progresses it gradually passes backwards by direct extension, but often pauses at the bulb or membranous part of the urethra. In more severe cases the disease may extend to the submucous tissue, the prostatic urethra and prostate, neck of the bladder, epididymis, bladder, and kidneys. Pyelitis and nephritis are less often the result of direct extension of the inflammatory process; more commonly they are the result of sympathetic irritation, especially in cases where the neck of the bladder is involved.

In the acute stage of gonorrhœa, the mucous membrane, where inflamed, will be deeply congested and of a bright red colour; it is swollen, and the epithelium on the surface proliferates and desquamates. A thick, creamy, purulent discharge soon makes its appearance. The dorsal lymphatics are often found enlarged and tender, and the inguinal glands are not infrequently involved and may suppurate. The glands are more likely to suppurate in cases in which the foreskin and glans are involved in the inflammation. As the disease subsides, the inflammation dies away first in those parts first attacked; the redness will then be less marked, and is often punctiform and patchy. When the disease subsides, the products of inflammation are absorbed by the lymphatics.

In more severe cases the surface-epithelium may be destroyed, and small ulcers and erosions formed; these may subsequently form granular patches (*see GLEET*), and may ultimately give rise to stricture. This necrosis of tissue very rarely extends beyond the submucous tissue. The ulcers may heal without producing any ill effects. If, however, they become chronic, fibrous tissue develops in their bases and gradually tends to contract; in other cases granulations spring up, and may develop into warty growths, similar to those seen round the corona.

The existence of different varieties of intensity of the inflammation has given rise to the use of distinctive names; thus: (1) In some cases several layers of epithelial cells are raised into small eminences by serous effusion; these burst and leave small erosions—*phlyctenular gonorrhœa*. (2) The inflammatory lymph, entangling leucocytes in its meshes, may form a continuous, more or less tough, membrane on the surface, which can be peeled off in flakes—*diphtheritic gonorrhœa*. (3) If the discharge is copious, and mixed with much serum and mucus, the case is sometimes spoken of as *catarrhal*.

SYMPTOMS.—Clinically, gonorrhœa may be conveniently divided into three stages corresponding with the development, period of vital activity, and destruction of the poison.

During the *first or incubative stage* there is some tickling and itching at the meatus and tip of the penis, and slight scalding on micturition. A feeling of heat, tension, and weight in the testicles and perineum is often complained of. Nocturnal erections and emissions are not uncommon, and the sexual appetite is increased. Micturition is more frequent than usual. These symptoms may be so slight as to be unnoticed by the patient, and only come on during the latter half of the incubative stage.

In the *second or acute inflammatory stage* the signs are pronounced. The lips of the meatus are swollen and red, the urethra is tender to the touch, and the corpus spongiosum is swollen and engorged. The discharge soon makes its appearance; at first it is sero-purulent and scanty, but in a very short

time it becomes greenish-yellow, thick, creamy, and copious. Micturition is frequent and painful; in consequence of the swelling of the urethral walls, the stream of urine is diminished in size, or it may escape in drops. The pain and scalding are often severe, especially if raw patches are present in the passage, or if the urine is concentrated and highly acid. Swelling and tenderness of the dorsal lymphatics are common, with enlargement of the inguinal glands (sometimes but not usually going on to supuration).

In more severe cases the whole penis becomes tender and swollen, and the subcutaneous veins engorged. As the disease spreads to the erectile tissue of the corpus spongiosum, it becomes infiltrated with inflammatory exudation, and the affected parts are no longer able to be distended with blood. Consequently, erections, which are very troublesome at night, are only partial, and the straining and tightening of the inflamed parts, by the distension of the uninfamed, produces severe pain, and alteration of the shape of the erect penis—chordee. In some cases copious hæmorrhage occurs from the distended parts. Small quantities of blood, giving a red tinge to the discharge, may also come from ulcerated and eroded areas of the inflamed mucous surface.

The effect of the disease on the patient's general health is marked in serious cases; he becomes pale, weak, and thin.

Peri-urethral swellings, giving the urethra a beaded feeling, are by no means uncommon, especially at the fossa navicularis and bulb. They are due to follicular inflammation, and to exudation into the spongy tissue round the urethra. If this be very acute, peri-urethral abscess may result.

When the disease spreads to the membranous-prostatic urethra and neck of the bladder, there is almost constant desire to micturate, with great pain and spasm following the act; there is also pain on defecation and in sitting down. Towards the end of micturition a few drops of pus and blood may escape. The discharge is less copious, and the urine often turbid from admixture with mucus, which forms a plentiful deposit. From inflammatory congestion of the passage micturition may be difficult, the urine dribbling away. In other cases the irritation in the passage excites spasm of the compressor urethræ muscle, resulting in complete retention. When the disease has extended so far back there is distinct heat and pain in the perineum; if the patient have an emission of semen, which is likely on account of the inflammatory irritation of the veru montanum and ejaculatory ducts, the act will be painful, as if something had been torn in the perineum. The semen, instead of escaping in jets, gradually trickles away, and is often mixed with and followed by a few drops of blood. In cases of gonorrhœa, fever may or may not be present; it is usually slight and transient, unless the disease be complicated. The duration of this second stage varies according to the severity of the disease, the treatment adopted, and the care of the patient himself. Usually the acute stage passes off gradually in from three to six weeks. The symptoms are, *ceteris paribus*, more severe in a first attack, but subsequent ones are more likely to become chronic.

After a varying time the acute inflammatory stage of gonorrhœa passes into the *third*. The symptoms may gradually abate, the pain and discharge disappearing, and the disease terminate. In other cases the discharge becomes less in quantity,

more watery, and of a sero-purulent nature, the other symptoms disappearing. Lastly, all the signs may subside with the exception of a thin whitish discharge, especially noticeable in the morning. In such cases the disease persists in the patchy form, and constitutes gleet. See GLEET.

During the third stage the acute symptoms may at any time be lighted up again if the treatment be abandoned, or in consequence of injudicious conduct on the part of the patient.

DIAGNOSIS.—Acute urethritis is not invariably gonorrhœal; and it may be of great importance, especially in cases likely to lead to legal proceedings, to determine whether the urethral mischief be gonorrhœal or not. Much credence must not be placed on the patient's statements. Certain complications are said only to follow gonorrhœa, and no other form of urethritis; but this is not so clearly established as to be of certain diagnostic value. In cases where a definite opinion as to the venereal nature of the case is necessary, the pus should be examined for the gonococcus.

For this purpose cover-slip films of the pus should be prepared in the usual way and fixed by passing three times through the flame of a Bunsen's burner. One preparation should be stained with methylene blue, and a second by Gram's method, and counterstained with fuchsin (weak aqueous solution). A $\frac{1}{10}$ -inch oil-immersion lens is necessary for the microscopical examination.

The determination of the identity of the gonococcus should be based upon the three leading characteristics of the organism, viz. (1) the presence of colonies (made up of varying numbers of diplococci and tetrads) within the substance of the pus-cells; (2) the peculiar coffee-bean or reniform shape of the individual elements; (3) the inability of these cocci to retain the stain when treated by Gram's method; and only such cocci as conform exactly and completely to these criteria should be identified as gonococci.

The two first points can usually be best demonstrated in the methylene-blue preparation. In the counterstained Gram, the gonococci will be seen to have yielded up the blue-black colour resulting from the combination of the gentian violet and iodine, and to have taken the fuchsin counterstain.

In cases of importance, whether medico-legal or otherwise, it is necessary to confirm the diagnosis by cultivating the gonococcus upon serum-agar, and also by demonstrating the inability of the suspected organism to grow upon the ordinary laboratory-media.

Urethritis may be easily distinguished from urethral chancre; but it must be remembered that both conditions may be present. Urethral chancre is nearly always situated just within the meatus, never more than one inch from it; the ulcerated surface can be seen if the lips of the urethra be separated, and a short aural speculum introduced.

The discharge from a chancre is not creamy, but shreddy, and the pain during micturition is stinging and limited to the raw surface. Occasionally a slight muco-purulent discharge, without pain or much swelling, is present during the initial stage of syphilis, but it subsides spontaneously in one or two weeks, and is accompanied by the indurated sore, and succeeded by secondary symptoms.

In balanoposthitis (which may or may not be venereal in origin) there need be no urethral discharge. This can be ascertained by thoroughly

syringing away all the pus from under the prepuce, and then squeezing the penis to see if pus escapes at the meatus. See BALANITIS.

Prostatic perineal (and lacunar) abscesses may discharge into the urethra. The commonest of these is the so-called frænal lacunar abscess. In such conditions the discharge, profuse on one occasion, at once subsides to a scanty gleet. These abscesses may be diagnosed by the history of the case, and a careful examination of the patient, but it must be borne in mind that they occasionally complicate gonorrhœa.

PROGNOSIS.—The prognosis of gonorrhœa is usually favourable if proper precautions be taken early. But in spite of attention and care, the disease is sometimes very severe, especially in young lads of lymphatic temperament, or in men of nervous irritable constitution. Gonorrhœa is said also to be liable to run a severe course in persons who suffer from acne. It is more likely to become chronic after repeated attacks, and in those who suffer from stricture or have a narrow meatus.

Various complications, to be afterwards referred to, may arise in the course of the disease, and certain sequelæ follow it. In rare cases gonorrhœa terminates fatally by general pyæmia, or more frequently may lead to joint-affections of more or less severity.

TREATMENT.—The so-called *abortive* treatment of gonorrhœa has, on account of its non-success and attendant risks, been practically abandoned, and calls for very short notice here. It consists in the internal administration of large doses of copaiba or cubebs, or of injections of strong antiseptics: the former cause marked constitutional disturbance, the latter aggravate the disease, and both very seldom do any good at all. Another method of rapid cure may be mentioned. If the case can be treated in the first day or two after incubation there is a slight hope of reducing the length and severity of the inflammation by washing out the urethra by the following method: the bladder is filled with warm boric-acid solution through a catheter; then the patient micturates slowly, and as he does so he distends the urethra by squeezing the meatus. Another method is to pass a small catheter and fill the bladder: the patient then evacuates the bladder along the urethra external to the catheter, producing a continuous stream of boric-acid solution.

It is necessary that the patient should remain in bed under this treatment, the injection being administered twice daily. It must, however, be remembered that this treatment is liable to produce complications, such as inflammation of the bladder or of the testes, and is often unsuccessful. Therefore, the writers regard this treatment as justifiable only in exceptional cases; and then only under the strictest antiseptic precautions. In the early stages of acute gonorrhœa, rest, if the patient can take it, is important. The bowels should be opened and kept acting by the use of saline aperients, and the diet should be low and of a non-irritating description. All alcohol, especially malt-liquors, highly seasoned dishes, great exercise, and sexual excitement should be rigorously avoided. Warm baths and strict cleanliness of the parts, with support of the penis and testes, are useful.

All discharges should be allowed to drip into a bag with cotton-wool at the bottom: warn the patient not to infect his eyes and not to squeeze the urethra. The diuretic salts of potassium, with tincture of hyoscyamus, are also useful, to diminish the

acidity of the urine, and allay the smarting and pain during micturition; the same may, if excessive, be relieved by immersing the penis in iced water during micturition. In extreme cases, cocaine may be injected before micturition. As long as there is much smarting and signs of acute inflammatory congestion, strong astringent injections should be avoided, but relief is obtained by washing out the urethra with tepid water after micturition. An eighth of a grain of permanganate of zinc to one fluid ounce of distilled water forms an injection which may generally be employed in the earliest stage. When the acute stage has passed, the continuance of the congestion is shortened by astringent and antiseptic injections, and the internal administration of copaiba, sandal oil, or cubebs. Injections should not be strong at first—2 grs. of sulphate of zinc to the ounce being sufficient. The patient should be directed to pass his water, and then wash out the passage with a little tepid water; after that the injection should be used, and kept in the urethra for two minutes. Many substances are used as injections, and it is difficult to say in a given case which will suit best. Sulphate of zinc, grs. 2 to 3j, or mixed with acetate of lead; chloride of zinc, gr. $\frac{1}{4}$ -gr. 1 to 3j; sulphocarbonate of zinc, grs. 2 to 3j; permanganate of zinc, gr. $\frac{1}{4}$ to 3j, are all useful. Protargol, alum, acetate of lead, hydrastis, and extract of belladonna may also be used. Extract of belladonna should not be used with permanganates, as they form an explosive mixture. The mercuric chloride in solutions of various weakness, 1 : 3000 or 1 : 2000 is used on theoretical grounds by some; but in the writers' experience they are most uncertain, and more often excite than allay irritation in the urethra.

Whatever injection is chosen, it must be used judiciously, and care taken that it does not aggravate the condition it is intended to benefit. As a rule, the injection should be made at least three times a day by the patient; if the membrano-prostatic urethra be involved it may be necessary for the surgeon to apply astringents himself (see GLEET). Bougies of iodoform and eucalyptus are highly thought of by some; they should be introduced at night. The writers have not seen any great benefit follow their use. If the injections increase the discharge, or heighten the soreness, they should be abandoned, and soothing internal remedies alone given. The chief internal specifics are copaiba, sandal oil, and cubebs; infusion of uva ursi, Gurjun balsam, tolu, turpentine, benzoic acid, and salol. They are often beneficial, but are apt to cause dyspepsia; and, in the case of copaiba, sandal wood oil, and Gurjun balsam, congestion of the kidneys, especially in the later stages of the disease. These remedies should be given thrice daily for several weeks. In the later stages the preparations of iron in combination with quinine are often useful.

Chordee is frequently a very troublesome symptom, for, in addition to the pain it causes, the patient's rest is broken, and consequently his health weakened. Perhaps the most useful remedy is a suppository at night of opium or belladonna in combination with camphor (1 gr. and 3 grs.); bromides, chloral hydrate, cannabis indica, and lupulin are also useful; while the erection can be immediately stopped by the application of cold water. In some cases a hot bath just before going to bed, with the use of the suppository mentioned, will prevent chordee. When gonorrhoea becomes chronic, ac-

companied by a gleet discharge, further treatment may be necessary. See GLEET; and CHORDEE.

COMPLICATIONS AND SEQUELÆ.—Various complications may arise during the progress of gonorrhoeal urethritis; many of these are referred to in special articles, to which the reader is referred for information.

1. *Balanitis* and *Balanoposthitis*.—See BALANITIS.

2. *Phimosis* and *Paraphimosis*.—See PENIS, Diseases of.

3. *Retention of urine* may occur in consequence of swelling of the mucous membrane, and muscular spasm closing the passage at the membrano-prostatic portion; this is specially likely to occur if there is inflammation of the neck of the bladder. Under the influence of warm baths, saline aperients, rest, and morphine suppositories, the congestion is lessened, and the temporary retention is usually relieved. It causes great pain and anxiety when present. Gonorrhoea affecting a patient who is the subject of stricture may, by causing acute congestion of the narrowed portion of the urethra, temporarily occlude the passage and cause retention. The treatment is the same as given above; in both cases it may be necessary to pass a fine soft instrument in order to afford immediate relief. In the case of stricture further treatment will be necessary when the gonorrhoea is cured. See MICTURITION, Disorders of.

4. *Lymphangitis* and *Bubo*.—See BUBO; and LYMPHATIC SYSTEM, Diseases of.

5. *Hæmorrhage from the urethra*, if profuse and continuous, is probably due to rupture of the corpus spongiosum during erection. A few drops of blood may come away with the discharge or after micturition; this may be due to rupture of the congested vessels, or to the presence of a granular patch, and needs no special treatment. If hæmorrhage is continuous, ice will be serviceable in arresting it, and hamamelis may be injected. In some cases a catheter may be passed, and the penis compressed by a bandage. See HÆMORRHAGE, p. 621.

6. *Lacunar abscesses* may occur anywhere in the urethra from inflammation of the lacunæ or follicles: the commonest is in the floor of the fossa navicularis, forming the 'so-called' frænal abscess. Peri-urethral abscess in gonorrhoea is produced by the extension of a lacunar abscess or by inflammation of Cowper's gland. These abscesses may cause acute symptoms, but are often chronic in their progress. The pus may escape into the urethra, externally through the skin, or both ways; in the latter case a urinary fistula is formed, which will, however, usually heal in time. When peri-urethral abscess is diagnosed, it should be freely opened; if the abscess has originated in a distended follicle, and has burst into the urethra, it may be treated successfully by laying it open, through the endoscopic tube, with a canaliculus-knife.

7. *Inflammation of the neck of the bladder*, often erroneously spoken of as cystitis, is due to extension of the disease to the membrano-prostatic urethra. Cystitis itself is a rare complication. This complication does not usually come on until the second or third week; it causes much pain and spasm, and has a very depressing effect on the patient's general health.

Inflammation of the neck of the bladder causes great frequency of micturition, with pain during,

and scalding at the end of, the act, caused by muscular spasm. A few drops of blood usually escape after the urine, with a little muco-pus. The usual gonorrhoeal discharge ceases almost entirely during the attack, but usually returns when the inflammation of the neck of the bladder subsides. Relapses are common.

Low diet, warm baths, rest, and anodynes will prove of great value in this complication.

When the bladder itself is affected, the disease usually attacks the base, and may spread to the kidneys. The inflammation may be acute or chronic, and may attack the mucous membrane only, or the whole thickness of the wall (parenchymatous cystitis). See BLADDER, Diseases of.

8. *Prostatitis and prostatic abscess.*—Prostatitis is a severe complication of gonorrhoea. It is due to the spread of the disease to the uvula vesicæ, and along the prostatic ducts to the body of the gland. It may be excited by injudicious treatment, wet and cold, or venereal excitement during gonorrhoea. This disease may run on to suppuration, or may become chronic.

In the acute form the gland is swollen and tender, causing painful and difficult micturition, and sometimes retention. There is a sense of weight, heat, and fulness in the perineum and above the pubes, with rectal irritation and pain on defecation.

Should suppuration occur, the local and constitutional symptoms increase. Eventually the abscess bursts, commonly into the urethra, more rarely into the rectum or bladder or on to the perineum. The treatment is the same as that described for inflammation of the neck of the bladder, combined with the passage of a catheter if retention should occur, and the evacuation of pus as soon as it has been diagnosed. Such abscesses are best opened through the perineum; should they burst into the urethra they will usually heal spontaneously; if not, a perineal opening must be made. See PROSTATE, Diseases of.

9. *Epididymitis and Orchitis.*—Inflammation of the epididymis is the most frequent complication of gonorrhoea. The disease is usually unilateral, but in a small proportion (3·5 per cent.) of cases both sides are affected. The spread of gonorrhoea to the epididymis is much more commonly seen among hospital out-patients than among private patients, doubtless because the latter take more care of themselves. The epididymis is not affected until the urethritis has spread backwards to the membranoprosthetic portion—i.e. towards the end of the third week. See TESTES, Diseases of.

10. *Inflammation of the rectal mucous membrane* occasionally occurs, more especially in women. It is due to direct inoculation of the discharge. There is smarting and burning pain, much increased on defecation. The mucous surface is bright red, and may be granular and ulcerated. A yellowish discharge escapes *per anum*. Thorough cleanliness, with the use of astringent lotions, will effect a cure.

11. *Gonorrhoeal conjunctivitis* is a very grave complication, especially if not seen early, as it may destroy the whole eye. The patient should be warned that no discharge from the urethra must be allowed to get near the eye. See EYE, AND ITS APPENDAGES, Diseases of.

12. *Scleritis and iritis* occasionally occur in the course of gonorrhoea, but are very rare. See EYE, AND ITS APPENDAGES, Diseases of.

13. *Inflammation of the joints, fasciæ, and great nerves, &c.* See GONORRHOEAL ARTHRITIS.

14. *Stricture* may occur as a sequela. It usually follows granular urethritis, resulting from gonorrhoea. Some months or even years elapse before the stricture is established. See URETHRA, Diseases of; and GLEET.

15. *Warts* may occur on the glans penis or in the urethra. See GLEET; PENIS, Diseases of; WARTS.

16. *Endocarditis* may also occur, and probably future observations will show that this complication is less rare than is at present imagined.

(b) *Gonorrhoea in the Female—Vaginitis.*

MORBID ANATOMY.—In the female, gonorrhoea first attacks the lower part of the vagina, and may spread to the vulva and urethra, or upwards to the upper part of the vagina and the uterus; and, by continuous extension, to the Fallopian tubes, peritoneum, and ovaries in one direction, and to the bladder, ureters, and pelves of the kidneys in another direction, besides attacking the joints, fasciæ, and eyes, as in the male subject. The disease may become chronic, and be accompanied by complications to be afterwards mentioned.

In the first stage the mucous membrane is congested, swollen, and tender; the parts are drier than natural, in consequence of a diminution of the natural secretions; but in a few hours there is a thin transparent mucous discharge, which quickly becomes sero-purulent, creamy, and copious. The discharge when fully established has often a greenish tinge, and is offensive and highly irritating. The affected mucous membrane is often studded with little eminences due to enlargement of the papillæ (vaginitis granulosa). In more severe cases patches of erosion may be seen, especially if the disease spreads to the os uteri. Owing to the irritating nature of the vaginal discharge the vulva becomes inflamed, swollen, and excoriated. The glands of Bartholin not infrequently become involved, and suppurate. The inflammation becomes chronic in from six to ten or twelve days, the pain, swelling, and congestion subsiding, and the discharge, now muco-purulent, remaining plentiful. In the chronic form, the upper part of the vagina, the *cul de sac*, and the neck of the uterus are the parts specially involved. They are of a brighter colour than the rest of the mucous membrane, and secrete the discharge. Not infrequently, when the inflammation has ceased in the vagina, pus can still be squeezed from the meatus urinarius or some of the crypts opening round that orifice, if the finger be drawn forwards along the under-surface of the urethra.

SYMPTOMS.—In the acute stage there are heat, itching, and swelling of the parts, smarting on making water, aching pains in the back and loins, with great tenderness on examination. The bright red colour of the congested mucous membrane, together with the offensive greenish purulent discharge from the surface, is sufficient evidence of vaginitis, but is not sufficient to determine its specific origin. As the disease becomes more chronic the acute symptoms pass away, but there is still a plentiful discharge.

DIAGNOSIS.—The diagnosis of vaginitis and vulvitis is easy, but it is sometimes impossible to speak with certainty as to its gonorrhoeal origin. The actual source of the discharge should be ascertained, since copious yellow irritating discharge may come from the cervix uteri, independently of any

pre-existing vaginitis. If a positive opinion as to the gonorrhœal nature of the case is required, the gonococcus must be sought for in the discharge. If there is purulent urethritis, the contagious nature of the case is satisfactorily determined, as irritating uterine discharges do not affect the urethra.

PROGNOSIS.—If assiduously treated, so that extension to the vaginal *cul de sac* and cervix uteri is prevented, the disease has a duration of about three weeks. If neglected, it may become chronic and prove very intractable, lasting sometimes for many months; this is especially likely to be so if the disease has spread to the cervix uteri and endometrium, or to the urethra. Although many complications may arise during the progress of the disease, but few are commonly of immediate danger. The length of time that the discharge remains contagious is most uncertain. Probably any discharge, however scanty and serous it may have become, may cause disease if increased by accidental irritation.

TREATMENT.—In the *acute* stages of vaginitis the treatment consists in allaying irritation by rest in bed; warm baths; frequent injections of warm water, solution of boric acid, or weak astringents—such as weak solutions of acetate of lead, of alum, &c.; and the administration of saline aperients. If the pain be great, a little laudanum may be added to the warm-water injections. The diet should be low, all sources of irritation should be removed, and the habits and health of the patient regulated. When the acute stage has subsided, astringent injections should be used somewhat stronger than those employed at first. The vulva should be protected from contact with the discharge by vaseline, and the labia should be separated by a piece of lint wetted with lead-lotion.

In the *chronic* forms of the disease the strength of the astringent injections must be increased. The most useful are perchloride of mercury, the sulphate, chloride, and sulpho-carbolate of zinc, tannic acid, and liquor carbonis detergens. In the intervals between the injections, tampons saturated with the astringent lotions may be introduced into the vagina, and alum or tannin may be applied to the deeper parts of the canal by means of the speculum. The injections must be efficiently done, after washing away the discharge with warm water, so that the whole mucous surface of the vagina, especially that of the *cul de sac*, is thoroughly laved. Astringent vaginal suppositories are also useful. If in chronic cases there are patches of erosion, similar to those met with in cases of gleet in the male, they should be mopped with a 20-per-cent. solution of silver nitrate.

Internal remedies are useless unless the urethra is affected, when they have a certain value.

COMPLICATIONS.—*Vulvitis* is one of the earliest and commonest complications of gonorrhœal vaginitis. The labia, nymphæ, and clitoris are red and swollen, and secrete a copious yellow foetid discharge. The nymphæ often project between the swollen labia, and the parts may be excoriated in consequence of destruction of the surface epithelium, and sometimes from ulceration of the mucous follicles. There is considerable pruritus. Usually, if the parts are kept clean and free from irritation, the inflammation subsides in a few days. Warm baths and sedative lotions are useful to allay the irritation, and the labia should be separated by a piece of lint kept moist with lead-and-opium lotion.

Urethritis is the most constant accompaniment

of gonorrhœa; rarely so acute as to cause much irritation, it may produce severe suffering. It begins with itching and smarting at the meatus, which is red and swollen. A purulent or muco-purulent discharge oozes from the passage unless the patient has just micturated; even then a little discharge can be found in the mouths of the follicles which open close to the meatus. This discharge is very persistent, and is probably a source of contagion long after the disease is cured elsewhere. The treatment of urethritis consists of frequent warm baths, astringent injections, and copaiba internally. Obstinate chronic discharges may be arrested by caustic solutions, carefully applied.

Abscess of the glands of Bartholin is not uncommon in gonorrhœa. There is a painful swelling, which may attain the size and somewhat the shape of a bantam's egg, lying in the labium majus, in which fluctuation can be detected early. The abscess may not empty itself through the duct, but into the cellular tissue of the labium, where a large collection may form before it opens superficially. The treatment consists in free incision as soon as fluctuation is felt. Often a troublesome fistula is left, which, alternately closing and opening, causes fresh accumulation and fresh discharge of pus from time to time. This is best treated by free incision and drainage.

Bubo is rare in the female, but may occur when the urethra is involved.

Acute inflammation of the cervix and os uteri is a frequent consequence of gonorrhœa. The neck of the uterus is swollen, red, and often excoriated about the os, whence a copious discharge issues, at first clear and viscid, then purulent. This subsides to a thin muco-pus, and either soon ceases or more commonly passes into chronic catarrhal discharge from the glands of the cervix, which lasts an indefinite time and long retains its contagious quality. Chronic cervical and corporeal endometritis, associated with a thick viscid discharge in the former and a thinner discharge in the latter, may result from gonorrhœal infection. In the acute forms, complete rest, warm baths, with astringent applications and saline aperients, may arrest the disease. In the chronic stage the treatment is that of uterine catarrh. See UTERUS, Diseases of.

Among the rarer and more dangerous complications of gonorrhœa in the female may be mentioned metritis, perimetritis, salpingitis, pyosalpinx, hydrosalpinx, ovaritis, and peritonitis. Pyæmic infection may occur through the vaginal veins.

Certain complications, more general in their nature, may also occur as in the male sex. Gonorrhœal arthritis is less common in women than in men.

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GONORRHOEAL ARTHRITIS.—SYNON.: Gonorrhœal Rheumatism, Urethral Arthritis; Fr. *Arthrite Blennorrhagique*; Ger. *Tripperrheumatismus*.

DEFINITION.—An infective inflammation of the joints and associated structures, occurring in the subjects of inflammatory discharge from the genito-urinary mucous membranes.

ÆTIOLOGY.—This disease, as its name implies, is generally referable to the presence of gonorrhœa. Occasionally it appears to originate in other kinds of inflammatory discharge connected with the urethra, such as gouty or traumatic urethritis or

gleet; while, on the other hand, it has been observed as a complication of gonorrhoeal ophthalmia without urethritis. It is much more common in men than in women, apparently from the more thorough cleansing of the female urethra during urination. An affection of the joints which strongly resembles it is found in connection with chronic uterine disease, and in the puerperal state; but so-called 'chronic uterine disease' in women is unquestionably very often gonorrhoeal. Either a rheumatic or a gouty history is frequently to be traced in the patient. Previous attacks powerfully predispose to the return of the disease on the recurrence of urethritis, even in a mild form. The observation has also been made that persons who have suffered from the disease are frequently found to be, and to have been, peculiarly susceptible to urethral inflammation; excessive sexual intercourse being regularly followed by discharge in such individuals. It is practically certain, however, that these excesses, this predisposition, and this 'susceptibility' are all to be accounted for by the actual presence of persistent gleet. In other instances the husband is re-infected by the wife; and successive attacks of urethritis and gonorrhoeal arthritis in the husband, along with leucorrhoea, chronic uterine disease, and tubal, ovarian, and peritoneal disease in the wife, may be referable to a single remote infection. See GONORRHOEA.

Among exciting circumstances, the most important appear to be injury of a joint, such as a sprain; a drinking-bout; and chill during the course of gonorrhoea.

ANATOMICAL CHARACTERS.—In recent cases of this disease the structures connected with one or more of the articulations are acutely inflamed. The cavity contains a variable amount of serous effusion according to its form and size; the knee, for example, being considerably distended, while the digital joints are more moderately enlarged. The various component parts are hyperæmic and swollen; and the peri-articular structures full or even cedematous. In more advanced cases the joints are found to contain either sero-purulent or purulent material; the cartilages may be eroded; and finally the articulations may become completely disorganised or ankylosed. The gonococcus has been found in the intra-articular effusion. The cardiac structures are very rarely affected. The eye may present the ordinary appearances of catarrhal (not gonorrhoeal) conjunctivitis.

SYMPTOMS.—The disease, as ordinarily observed, commences at any period in the course of gonorrhoea—very frequently within a week of its appearance, but possibly not until it has degenerated into a slight gleet, or apparently disappeared. The patient is probably first aware of pain in the loins, or of swelling and pain in the soles of the feet, and very shortly these symptoms involve the ankles. In other instances the knees or wrist-joints suddenly become painful, tender, and swollen—possibly after strain or exertion. At the same time the patient is feverish, suffering from malaise and anorexia; the tongue becomes foul; and the pain, broken sleep, helplessness, gonorrhoea, and general illness give rise to restlessness and depression. Along with, perhaps even before, the articular symptoms, conjunctivitis sets in, affecting one or both eyes; although of a well-marked catarrhal kind, it usually passes off in a few days with little or no treatment.

The physical signs connected with the joints are

generally well marked, the parts being hyperæmic and much swollen, both from intra-articular effusion and from exudation around. The amount of oedema of the dorsum of the hand or foot when the neighbouring joints are involved by gonorrhoeal arthritis, and of the upper part of the subcutaneous surface of the tibia when the knee-joint is affected, is often remarkable. The severity of the pain varies much. At times it is great, preventing sleep, especially as it is usually aggravated at night; in other instances it is extremely slight, and the condition is then more chronic in character. The pain is 'gnawing' or 'aching;' according to some patients, it is more severe before the swelling appears, according to others it is aggravated by the swelling. An important feature of the pains in gonorrhoeal arthritis is that in some cases they are not limited to the joints, but involve the fibrous structures, especially of the loins, the plantar and palmar fasciæ, the tendo Achillis, and the sheaths of nerves such as the great sciatic. The muscles, or their aponeuroses, also appear to suffer; the fleshy parts of the arm, forearm, neck and thigh, as well as the loins, suffering especially. Frequently, indeed, the patient declares that the pains are universal. Stiffness is also felt, particularly when a joint or limb has been kept long in one position. Tenderness varies much, like the other phenomena, being exquisite in some instances and entirely absent in others. Portions of the tendon-sheaths may be found swollen and tender.

The heart and pericardium are very rarely involved. See HEART, Inflammation of: 2. Ulcerative Endocarditis.

Such are the usual characters presented by a case of gonorrhoeal arthritis within the first week of its appearance. Under favourable circumstances the symptoms may decline; but in the majority of instances one joint after another is invaded by the morbid process, while those already attacked either slowly recover or continue affected, the disease becoming more extensive and protracted for several weeks or even months. Cases are met with in which all the joints of one or more limbs are simultaneously affected, and have been so affected for ten to sixteen weeks; certain of the articulations being but recently invaded, while others are slowly recovering from the attack with which the disease commenced. In this manner every joint in the body may be invaded, including the temporo-maxillary, the sterno-clavicular articulation, and the spinal column. The second toe is a favourite seat of the disease.

The class of case just described constitutes the most severe form of gonorrhoeal arthritis. Happily, in most instances the disease is much milder, only one or two joints being affected, and the process either ending with a sharp painful burst of acute synovitis, or lapsing into a state of chronic intra-articular effusion, with neither tenderness nor pain.

When the disease is protracted, either in one or in many joints, the constitutional symptoms lose their acute character. There is little or no pyrexia; the appetite is fair; and the patient may even go about his work. But the health is gradually impaired, the patient being debilitated and depressed; in the most severe cases he may be completely crippled, unable to stand, write, or feed himself, and reduced to a condition of general helplessness and wretchedness.

COURSE, DURATION, AND TERMINATIONS.—The variable course of gonorrhoeal arthritis has

been already sufficiently indicated. The duration of the disease is quite indefinite, varying from a few days to many months. The most unfavourable termination of the disease is ankylosis of the joints, with hopeless crippling; but this is rare. It never proves fatal directly. The disease, as already stated, is very liable to return. The urethritis is contagious, and the writer has seen husband and wife suffering together from the disease.

PATHOLOGY.—Opinion is still divided upon the essential nature of gonorrhoeal arthritis. Three leading views may be mentioned. (1) It is believed by some pathologists that 'gonorrhoeal rheumatism' is nothing more than acute or subacute rheumatism, associated with gonorrhoea or other similar discharge. (2) Most authorities now regard the disease as one of several results which may arise from absorption of the products of the gonococcus, the seat of the primary infection being usually the urethra (*see* GONORRHOEA). (3) The third view is perhaps not inconsistent with either of the other two. It represents gonorrhoeal arthritis as a trophic or nutritive disorder, due to reflex disturbance; the products of the urethral inflammation affecting primarily certain centres in the spinal cord and brain, and the altered condition of these giving rise to the articular changes.

According to both the pyemic and the trophic or reflex theories of gonorrhoeal arthritis, the joint-affection may originate in a purulent discharge from any mucous surface, the urethra included. Thus joint-disease has occasionally been observed in association with dysentery, and with chronic bronchitis. It is beside the purpose of the present article to enter into a discussion of these theories.

DIAGNOSIS.—The diagnosis of this disease turns upon the existence of a urethral discharge in association with articular inflammation. The occurrence of the latter in young male subjects should always arouse the suspicions of the practitioner as to the presence of gonorrhoea; and he ought at once to ascertain, by careful inspection, the state of the urethra, never accepting the patient's statement on the subject. In some instances there may be but a trace of gleet; or the history of a recent gonorrhoea alone may remain. In doubtful cases the urine must be examined with particular care. It should be passed in the presence of the practitioner, and the first fluid ounce of it collected in a separate vessel and examined for products of urethritis that may be washed out, including muco-purulent threads from the follicles. A history of one or more similar attacks in connection with gonorrhoea will confirm the diagnosis. Ophthalmia in association with sub-acute articular symptoms ought immediately to suggest gonorrhoeal arthritis. Suspicion of the infective nature of the arthritis is sometimes first roused by the failure of salicylates to give relief.

PROGNOSIS.—The prognosis is generally favourable. In young healthy subjects, under careful treatment, the disease will probably subside shortly. It will, however, prove protracted and obstinate under the opposite circumstances. Further, unless cure be complete, the future is likely to prove a history of recurrences and accumulating miseries. Gonorrhoeal arthritis increases in severity in subsequent attacks, and the prognosis is correspondingly more unfavourable. Another point which should be seriously impressed upon the patient, by way of warning, is that the risk of the recurrence

of arthritis also increases with each exposure to gonorrhoeal infection.

TREATMENT.—The treatment of gonorrhoeal arthritis is still unsatisfactory. While some practitioners endeavour to check the urethral inflammation as speedily as possible, others strive to encourage the discharge. The former plan is unquestionably the more rational and the more successful. The treatment of the gonorrhoea or gleet must be commenced at once and persevered with assiduously. *See* GONORRHOEA; and GLEET.

At the same time, the attention of the practitioner will be anxiously directed to the joints. If the local symptoms be severe, absolute rest is necessary, the patient being confined to bed, and the affected limb protected with a splint in such a way that applications can be made to the joints. Anodynes may be called for at first, such as poultices, fomentations—simple or opiated—or an application composed of equal parts of extract of belladonna and glycerine. In some instances leeches may be tried; blisters in others, where there is either great pain, unrelieved by anodynes, or persistent effusion. In some acute cases with little pain or general disturbance, strapping may be sufficient—particularly with 'Scott's Dressing'; and in a more chronic form of the disease friction of the joint and associated parts, and moderate passive and active exercise, may effect a cure.

Internal treatment must be pursued simultaneously. In acute cases free purgation should be insured at first, and this should be followed by a course of alkaline salts, either alone or in combination with quinine. Treatment specifically directed to the gonorrhoea is often required. If the disease persist, potassium iodide should be given, combined with alkalis or with iron, according to circumstances; iron alone suits other patients well. Phenacetin and salicylates appear to be beneficial in some instances. In other cases mercurials effect a cure, especially if there be a syphilitic taint, which is not uncommonly the case. The diet must be carefully regulated in the different stages: at first 'low' and without stimulants, afterwards generous. In very chronic cases of gonorrhoeal arthritis with threatening ankylosis, the patient should be sent, if possible, to a warm watering-place, and be subjected to a thorough course of treatment both externally and internally. Sea-voyages very successfully fulfil the different indications for treatment in obstinate cases. *See* RHEUMATOID ARTHRITIS.

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GONORRHOEAL RHEUMATISM.—*See* GONORRHOEAL ARTHRITIS.

GOOSE-SKIN.—A condition of the skin in which this structure is rough and wrinkled, like that of the plucked goose. It is of a transient character, being due to contraction of the muscular fibres of the skin, producing wrinkling of the integuments, and prominence of the hair-follicles—horripilation; and is observed as the result of the direct application of cold, or of a shock, and in the early stages of fevers.

GOUNDOU.—*See* ANAKHRE.

GOUT (*gutta*, a drop).—**SYNON.**: Podagra, Chiragra, Gonagra (when the disease affects the foot, hand, or knee respectively); *Fr. Goutte*; *Ger. Gicht*. The name *gout* is supposed to have originated

in the idea of the dropping of a morbid fluid into the joints, and is of very ancient date.

DEFINITION.—Gout has been usually regarded as a general or constitutional disease, associated with uric acid, the complaint being commonly looked upon as a manifestation of the so-called *uric-acid diathesis*. Gout is characterised ordinarily by a peculiar inflammation of the joints—*articular or regular gout*, attended by the deposit of urate of sodium in their structures, affecting especially the smaller joints, and at first more particularly the metatarso-phalangeal articulation of the great toe, but afterwards extending to other joints. Similar deposits of urate may occur in other tissues—*tophaceous gout*; and certain organs of the body are liable to become the seat of functional disorders, or of pathological changes, during the progress of the disease—*non-articular or irregular gout*; while it is also usually attended with general symptoms. Gout in the early part of its course is in the large majority of cases an acute affection, occurring in periodic attacks; but subsequently it tends to become more or less chronic and permanent, though even then generally presenting exacerbations from time to time.

ÆTIOLOGY.—1. Cases of gout are practically due to one or more of three morbid influences—(a) hereditary transmission; (b) certain errors in regard to food and drink, often associated with deficient exercise; and (c) impregnation of the system with lead.

(a) *Hereditary transmission.*—Gout, once established, may be transmitted through several generations, even when every endeavour is made to eradicate it; and if the malady is intensified by pernicious habits, it is likely to become a permanent legacy. In more than half the cases an hereditary taint can be distinctly traced. Gout appears to be more readily transmitted by the female than the male line. According to Hutchinson, the diathesis becomes stronger in the parent with advancing years, and consequently the disease shows itself with greater frequency, and in more marked form, in the younger than in the older members of a gouty family; and should gout have arisen *de novo* in the parent, children born before the first attack may be free from the disease. Hereditary influence may be so powerful that the complaint arises without any other obvious cause; but most commonly it is aided by more or less indulgence in certain injurious habits to be presently mentioned. Gout sometimes misses a generation (*ataavism*). This is usually due to the fact that, in the generation free from the complaint, every precaution is taken to avoid causes which tend to originate a gouty paroxysm. The disease may be transmitted from a gouty grandfather through a healthy daughter to her son. The hereditary nature of gout is shown in the age at which the disease reveals itself. Should the predisposition be powerful, the complaint may appear in very early life; and the younger the subject who is attacked, the more likely is there to be an hereditary taint. Hutchinson is of opinion that many obscure joint-affections in young persons are due to hereditary gout.

(b) *Errors relating to food, drink, and exercise.*—Gout is originated *de novo*, in consequence of certain errors affecting the diet and habits; or an inherited tendency to the disease is thus considerably aggravated. In general terms these errors may be summed up as excessive eating, especially of parti-

cular articles of food; undue indulgence in alcoholic drinks; and indolent habits, with deficient exercise.

The gouty diathesis is chiefly promoted by foods which are rich in nitrogenous elements, and especially lean meat. Beef is believed by many to be particularly baneful. Undue consumption of sugar and starchy foods has also been regarded as aiding materially in the development of gout.

The relation of alcoholism to the gouty diathesis is abundantly proved by everyday experience. The more potent wines have the greatest influence, and port wine has proverbially been regarded as the most injurious of all. Champagne, burgundy, madeira, sherry, and marsala are also undoubtedly capable of developing or aggravating gout. Hock, sauterne, moselle, and light claret seem to be least injurious; but even these, if indulged in to excess, may in course of time set up, or at any rate intensify, the gouty diathesis. Strong malt-liquors stand next to wines as originators of gout. Spirits are comparatively feeble in their power of producing the disease, and this is the explanation usually given of its infrequency in those countries or districts where drinks of this class are chiefly used, such as Scotland and Newcastle-on-Tyne, where whisky is the common beverage. Rum is said to form an exception to this statement. Cider and perry may unquestionably set up gout, especially when sweet and not properly fermented. Excessive indulgence in a mixture of alcoholic drinks is probably more deleterious than if one is adhered to. The quality of wines has much to do with their tendency to induce gout. Factitious wines, and others of inferior quality, as well as those which are very sweet, or which contain much tannin, are most liable to produce this complaint. Drinks which cause a marked diuretic action are less injurious than those which have but little action of this kind.

Deficient exercise undoubtedly promotes the development of gout in many cases. Persons who follow sedentary occupations, or who live indolent lives, are most liable to the disease; and not a few become gouty because improvement in their circumstances enables them to 'keep a carriage,' and they are thus deprived of the exercise which they were previously accustomed to take.

(c) Another group of cases of gout are those which occur in connection with *impregnation of the system with lead*. Garrod found that, among his hospital-patients, 33 per cent. of those suffering from gout had been subjected to the influence of lead in their various occupations; and the association is now generally acknowledged. This metal does not appear, however, to originate the gouty diathesis, unless aided by indulgence in alcoholic drinks, though the amount of the latter consumed is usually far less than would alone account for the condition. Gouty persons are remarkably susceptible to the influence of lead; and when this metal is given to such subjects for medicinal purposes, it is very liable to bring on a severe attack of acute gout. This intimate relation between gout and saturnine poisoning is not always seen. 'Workmen from the south develop it in the north of England. The natives of the north, though equally exposed, seldom become gouty even when the kidneys are affected' (Oliver). The reason for this difference is unknown.

2. *Predisposing causes.*—(a) *Age.*—Distinct gouty attacks in a large majority of cases make

their first appearance in persons between thirty and thirty-five or forty years old. Those which occur under thirty are, with few exceptions, more or less hereditary. Well-marked gout is exceedingly rare under twenty. Lorimer found that saturnine gout appears at an earlier age than non-saturnine. The complaint is usually declared before fifty, and first attacks are progressively less frequent after this period of life. It is quite exceptional for gout to commence after sixty-five, but it may begin even in extreme old age.

(b) *Sex*.—Males are far more commonly the subjects of well-marked gout than females. This is mainly accounted for by the difference in the habits of the two sexes. In some measure it may depend on the occurrence of menstruation in females, which is supposed to act as a safety-valve. In women gout is more likely to appear after the cessation of this function. When strongly hereditary, gout may, however, develop even in young females.

(c) *Bodily conformation and temperament* have been credited with a predisposing influence in relation to gout, persons of a corpulent plethoric habit of body being supposed to be most subject to this disease. These conditions are often produced by the very habits which originate gout, and many persons who are gouty by inheritance do not present any of these characteristics. Gout is not uncommon in individuals of a nervous temperament, thin and wiry in frame; and these are said to be more subject to the irregular and asthenic forms of the disease.

(d) *Social position and occupation* influence the occurrence of gout, although generally indirectly. Formerly the complaint was met with almost entirely among the aristocracy. Now, however, it is common enough among the middle classes, chiefly those who are in affluent circumstances; while it is very prevalent among butlers, coachmen, butchers, publicans and barmen, coal-heavers, porters, haircutters, and painters. Conditions of life which involve excessive mental fatigue or prolonged anxiety seem to predispose to gout. Duckworth is of opinion that *sexual indulgence* in early life is a potent cause.

(e) *Climates* which are cold or temperate, and especially at the same time damp and changeable, present by far the greatest number of cases of gout, and in most tropical countries this complaint is unknown. In the south of Europe it is far less frequent than in this country. Gout is much more prevalent in England than in Scotland or Ireland; it is uncommon in the north of England.

3. *Causes of a gouty attack*.—An acute paroxysm of gout may come on without any evident *exciting cause* whatever, especially if the disease is strongly hereditary, or has been long established. Under these circumstances outbreaks of the complaint seem to become habitual at certain seasons, or to arise from very slight causes, which are less and less obvious as the case progresses. Often, however, some distinct exciting cause can be made out. The most important are eating or drinking too much, either on some particular occasion, or habitually for a longer or shorter period, until at last a fit of gout terminates the indulgence; indigestible articles of food; constipation; undue physical work or exertion; exposure to cold or wet, or sudden suppression of perspiration; excessive mental work, worry, or anxiety; emotional causes, sudden, powerful, or

depressing, such as joy, rage, fright, or grief; sexual indulgence; hæmorrhage, acute illness, or other like debilitating causes; and injury. The injudicious use of certain medicines, such as preparations of lead or iron, may excite a gouty paroxysm. The implication of a particular joint may be due to a strain or injury, which may be very slight, such as the pressure of a tight boot, or the toe being trodden upon. Injury to the knee has caused that articulation to be first affected. As *predisposing causes* of acute gouty attacks, climate and season are highly important. Undoubtedly gouty paroxysms may often be averted by residence in a warm climate, either permanently or during the colder seasons of the year. Early attacks are most frequent in late winter or spring, and the first paroxysm occurs especially towards the end of January or beginning of February. Later on, a fit of gout may take place also in the autumn; and subsequently the intervals become shorter and more irregular. The occupation of an individual may predispose to the occurrence of gout in particular joints; thus butlers have it in the feet, coachmen and washerwomen in the hands.

PATHOLOGY.—The pathology of gout is imperfectly known. Many diverse views concerning its nature are held: the most important of these will now be indicated.

1. Gout is by the large majority of writers regarded as a constitutional disease, and it is customary to speak of the *gouty diathesis*, of which the local changes are but a manifestation. Further, it is believed by most authorities that a person may be gouty, and yet be free from any of the special lesions usually associated with the condition.

It is generally assumed that gout has an intimate relation with the presence in the system of *uric acid*, which is regarded as the morbid agent. As Duckworth expresses it—‘No uric acid, no gout.’ In its pronounced form gout is characterised by the deposit of biurate of sodium in connection with the joints and other structures, which distinguishes it from all other complaints. As to the exact relationship between gout and uric acid, there is much diversity of opinion. Many attribute the disorder to excess of this acid in the blood and tissues. Others pay more regard to the exact conditions under which it exists, and to its solubility. Gout is often spoken of as a mere manifestation of the *uric-acid diathesis*. William Roberts drew attention to the fact that an excess of uric acid in the blood (*lithæmia*), though only an exaggeration of a normal condition, is always pathological. He was of opinion that there are different causes for such excess, and believed that the gouty diathesis and plumbism are radically distinct, differing in all respects except in their tendency to uratic deposits. He further considered it quite possible that precipitation of the crystalline urate may occur not only in the form of coarse masses, but also in the form of minute stars or detached crystals, which can only be detected with the microscope; and that these microscopic deposits, either in the substance of organs or in the blood itself, may account for the visceral neuroses, thrombosis and embolism, and the occasional sudden death, which are encountered in gout, and may explain some very obscure incidents in its history.

In all probability uric acid exists in the blood and in the intercellular fluid mainly as a quadriurate of sodium. This salt is soluble in blood-serum and has no toxic action, but is a very unstable

body, and is soon converted into the biurate, which is very sparingly soluble in that medium, and is readily deposited from it in a crystalline form. Luff describes two forms of biurate, the gelatinous and the crystalline, the former being first produced from the quadriurate. It is five times more soluble in blood-serum than the crystalline variety, and is therefore not precipitated. The gelatinous modification is afterwards converted, either slowly or rapidly, according to various conditions, into the crystalline compound. Uric acid is being constantly formed in the system, but under ordinary circumstances is either destroyed or is eliminated by the kidneys.

The main views held to explain the lithæmic condition and the presence of urates in the tissues may be summarised as follows:—

(a) *Excessive formation.*—It appears reasonable to suppose that certain of the recognised causes of gout must lead to an absolute increase in the amount of uric acid formed in the system. Ebstein is of opinion that in the gouty this acid forms in perverse localities, as in muscles and the marrow of bones. Haig is opposed to the view of excessive formation, and attributes any excess of uric acid present in the body simply to its introduction in the food.

(b) *Undue retention.*—According to Garrod, such retention is due to defective action of the kidneys, either temporary or permanent, and when these organs are in a state of advanced organic disease, their excretory functions must obviously be interfered with considerably. William Roberts thought it probable that the defective power of the kidneys to eliminate uric acid is due to diminished alkalinity of the blood, rendering it less soluble. Haig supports the view that the retention of uric acid in the blood is the result of deficient alkalinity of the blood, which leads to gradual accumulation of the acid in certain parts. According to Garrod it appears that uric acid is attracted in different degrees by different structures, and tends to be held back by, and accumulate in, the liver and spleen, and in fibrous tissues, especially those of the joints, probably because these structures are acid, or less alkaline than the rest of the tissues and fluids of the body. Luff believes that under normal conditions uric acid is formed in the kidneys alone, and he ascribes accumulation of urate in the blood to deficient secretion by these organs, and consequent absorption from them. He affirms from his experiments that the solubility of uric acid in the blood is not affected by diminished alkalinity due to the addition of an organic acid, nor does it hasten deposition of biurate or diminish its solubility.

(c) *Special views.*—Latham advocates the hepatic origin of gout, but considers that 'imperfect metabolism of glycocine is the primary and essential defect.' According to Ebstein, the primary factor in gout is nutritive tissue-disturbance, a necrotic change taking place in the affected structures, which is followed by deposit of urates.

The relation of the nervous system to gout deserves special notice. Various writers on the subject have maintained that such a connection exists. Dyce Duckworth has brought forward evidence in favour of the view that gout is a 'neuro-humoral' disease. He maintains that there is a functional disorder of a definite tract of the nervous system, which he is inclined to localise in some part of the medulla oblongata.

While the pathological relation between uric acid and gout is acknowledged by most authorities, there are some who altogether dispute the connection, and maintain that this acid is an inert innocuous substance. One view which has been advocated during recent years demands special notice, namely, that which refers gouty phenomena to the effects of the alloxur-bases (xanthin, hypoxanthin, &c.) which are regarded as the *materia peccans*. Experiments have been made which are said to show the injurious effect of these bases on the kidneys. Alloxuric substances (uric acid and bases) are believed by many, adopting the theory of Horbaczewsky, to be derived from nuclein, being the ultimate products of the breaking down of the material in the body which is contained in tissues and cells, and probably mainly from that in leucocytes. According to Kolisch, as long as the uric-acid-forming organs are intact, especially the kidneys, the bulk of the products of nuclein-metabolism are excreted as uric acid. If these organs are diseased less acid is formed, and there is a corresponding increase in the production of xanthin-bases. He believes that in gout a constitutional tendency to the breaking down of the nuclein is inherited, but it is only when the renal functions become impaired from any cause that grave manifestations of the diathesis make their appearance. Croftan is of opinion that normally all the nuclein is converted into uric acid, which is innocuous and non-toxic, and that a perversion of the normal function leads to the formation of alloxur-bases, which are toxic. He considers that in a uratic subject there is a tendency to disintegrate a quantity of nuclein far in excess of the quantity normally disintegrated—in short, that nuclein-metabolism is excessive.

With regard to the local manifestations of gout, the joints on which most stress falls are generally affected first. Indeed, as already stated, Ebstein maintains that a nutritive disturbance of the affected tissue is the primary change, leading ultimately to its necrosis, and that uratic crystallisation is secondary, not occurring before complete death of the damaged texture. He looks upon sodium urate as a directly poisonous irritant wherever deposited, the injurious effects varying according to the quantity and concentration of the deposit, and the vulnerability of the special tissue involved, firm textures resisting better than those of looser character.

Klemperer considers that the deposition of urates in the tissues is due to a special chemical affinity of necrotic areas for uric acid, the necrosis being due to an unknown cause. Mordhorst, in discussing the deposition of urates in the tissues of gouty patients, dissents entirely from Ebstein's view. He states that in a saturated solution a urate is thrown down in the form of minute spherules which grow by accretion. He holds that under unfavourable conditions the fluids of the tissues liable to gouty inflammation may become neutral or even acid in reaction, and the granular urate, which is always first deposited, or even sodium biurate itself, may consequently undergo decomposition with the formation of crystals of free uric acid, which may block even the larger lymphatics of the part, and so set up an acute gouty inflammation. Since, however, the acidity of the affected tissues can only be temporary, the free uric acid will be reconverted into granular urate when alkalinity is restored, and ultimately into sodium biurate. According to this view the inflammatory process may be regarded as

curative, since it causes an increased flow of alkaline blood through the part, and encourages osmotic interchange between the focus of inflammation and the neighbouring capillaries.

While acute articular gout is almost invariably regarded as inflammatory, George Balfour has advanced an altogether different view, and affirms that from beginning to end of a gouty paroxysm there is neither sign nor symptom of inflammation. He maintains that its phenomena are readily and only explicable on the supposition of a stasis (or block) in the capillaries of the affected joint. This is accompanied by the formation of an anæmic area in and around the joint, and naturally the veins connected with this area of negative pressure get filled by reflux from the contiguous valveless veins. Plasma slowly accumulates within this area, the corpuscular elements also escape by diapedesis into the tissue-interspaces, and the red corpuscles readily part with their colouring matter. The blood-plasma contains urates, which are not very soluble, and when it accumulates within the anæmic area surrounding a gouty joint, the salts slowly crystallise out and get left behind when the serous part of the fluid is reabsorbed. Another view which has been brought forward as to the nature of an acute attack of gout is that the inflammation is not in the articulation at all, but that there is a neuritis in the surrounding tissues.

ANATOMICAL CHARACTERS.—The results of *post-mortem* examinations show that even in the very earliest period a deposit of urates takes place in the tissues of the joint; and as the attacks are repeated again and again, the deposit increases, until at last it may form considerable masses, and infiltrate extensively all the structures entering into the formation of the joint. The articulation then becomes permanently enlarged and distorted, while the ligaments are thickened and more or less stiff or even quite rigid, until ultimately complete ankylosis may be produced. The deposit seems to commence in the substance of the cartilage covering the ends of the bones, starting near its superficial or free surface, and gradually extending more deeply, though for a time a thin layer of cartilage lies between it and the cavity of the joint. This deposit at first forms a whitish opacity, but as it becomes more abundant it encrusts the cartilages, and also the inner surface of the ligaments, and the surfaces of fibro-cartilages where these exist. More or less extensive spots or patches become in time distinctly visible, and even the entire surfaces of the bones forming a joint may be covered with a chalky-looking substance. 'The appearance of articular cartilage in which simple deposit of urates has taken place exactly resembles that which would result from smearing or splashing the surface with fresh white paint' (Duckworth). The synovial membrane may also present white points, but the synovial fringes at their margins seem to escape, on account of their vascularity. In the larger articulations synovial fluid may be thickened, and may even contain separate crystals or tufts of the urate. Subsequently the ligaments and adjoining structures are infiltrated, and it is to this cause that the stiffness or rigidity of gouty joints is mainly due. Distinct masses of deposit may, however, form, which also interfere with movement. They are known as *tophi* or *chalk-stones*. In course of time the tissues covering a gouty joint may be destroyed, including the skin, the chalky-looking substance

being thus exposed, and unhealthy suppuration and ulceration set up, or even gangrene. Very rarely suppuration takes place in a gouty joint itself, or hæmorrhage may occur.

The opaque white substance characteristic of articular gout consists of fine crystals, in the form of needles or prisms. They are chiefly arranged in minute clusters, radiating from a centre; and in the cartilages they form a more or less compact network. Chemical examination shows that they are chiefly composed of biurate of sodium.

The metatarso-phalangeal articulation of the great toe is the one primarily attacked in the large majority of cases of gout; and this joint, on one or both sides, may alone present any change. As usually seen, however, the disease has progressively involved many joints. In the feet it may implicate all the articulations, but it is a curious fact that the tarso-metatarsal and the phalangeal joints of the great toe generally escape, or are but little affected. Similarly, all the joints of the hands and fingers are often involved, but the metacarpo-phalangeal joint of the forefinger is one of the most commonly attacked. The gouty change not uncommonly extends to the larger joints, more especially those of the leg, but the shoulder and hip-joints are very rarely implicated. In exceptional cases other articulations are found involved, such as the temporo-maxillary, those of the spinal column, of the pelvis, or even of the larynx in rare instances. According to some writers fibrillation and erosion of the cartilages take place before the biurate of sodium is deposited.

Coming now to other structures, deposits of urate may occur in various parts of the body, sometimes abundant and widely spread, in connection with bursæ, tendons and aponeuroses, sheaths or the substance of muscles, the sclerotic coat of the eye, the cartilages of the external ear, eyelids, nose, or larynx, under the skin, or rarely in the cerebral and spinal meninges. Urates have also been found in concretions on the aortic and mitral valves, and in plates upon the arch of the aorta. More or less serous effusion may be present in bursæ which are the seat of uratic deposits. The exact composition of these tophi varies, and they may contain calcium urate, oxalate, or phosphate, as well as sodium chloride and organic matter, in addition to sodium urate.

As regards bone, the periosteum is often affected, and some writers have described a deposit of urate in bone itself; but Garrod has not found evidence of its having originated in this tissue. He considers that the periosteal formations sometimes acquire sufficient size to press on the osseous tissue, and to cause its absorption.

The *kidneys* may be affected at a very early period, even when there have been little or no external manifestations of the complaint. In the first instance a deposit of sodium bi-urate takes place, probably within the renal tubuli, which afterwards involves their walls and penetrates to the intertubular tissue. This is seen in the form of white streaks in the course of the tubuli, and of white points at the extremities of the papillæ. The deposit goes on increasing, and a chronic inflammatory process is set up, ending ultimately in the production of a variety of 'granular contracted kidney,' known as the 'gouty kidney' (see KIDNEY, Diseases of). Other morbid states in connection with the urinary organs observed in some cases of

gout are the presence of gravel or calculi consisting of uric acid, urates, or oxalate of calcium; pyelitis; chronic cystitis; and urethritis.

In the course of gout other morbid changes often arise. Among these are: changes in the pharyngeal structures; congestion, catarrh, or more serious inflammation of some part of the alimentary canal; affections of the teeth; congestion or catarrh of the air-passages, chronic bronchitis, or dry bronchial catarrh, emphysema, and, some think, pneumonia; congestion of the liver, fatty disease, perhaps cirrhosis, and gall-stones; meningitis, neuritis, perineuritis, cerebral hæmorrhage; cardiac changes, including chronic valvulitis and degeneration of the valves, and hypertrophy, followed by degeneration of the cardiac walls, or primary degeneration associated with disease of the coronary arteries; atheromatous changes in the vessels, hypertrophy of the muscular coat of the small arteries, or arterio-capillary fibrosis; various diseases of the skin, such as erythema, urticaria, eczema, psoriasis, a peculiar macular eruption, as well as changes in the nails; iritis, hæmorrhagic retinitis, and other conditions of the eyes; and affections of the ear. How far some of these conditions can be directly attributed to the gouty diathesis, or are merely the result of the same causes which have induced this diathesis, may be fairly disputed. It is worthy of remark that acute inflammation in connection with the heart is not met with in cases of gout, and this has been attributed to the great vascularity of the cardiac tissues.

The principal change in the blood is the excess of uric acid, which can be found especially during the acute paroxysms. The other changes are unimportant. See BLOOD, Morbid Conditions of.

Uric acid has been obtained from the fluid contained in the blebs produced by blisters; from inflammatory serous effusions; from synovial fluid; from dropsical accumulations, such as ascites; and from the subarachnoid fluid.

CLINICAL HISTORY AND SYMPTOMS. — The clinical history of gout is a very varied one, and the symptoms observed in different cases which are regarded as of a gouty nature are exceedingly numerous and diverse. The custom of looking upon every acute illness, particularly inflammation of organs, occurring in gouty subjects, as being due to the gouty condition is unjustifiable, though it may be acknowledged that gout does often modify their clinical history.

1. Premonitory Symptoms. — Suspicious twinges or uncomfortable sensations about the toes or fingers are commonly noticed from time to time before the first actual fit of gout occurs. In a large number of cases, however, no definite premonitory symptoms immediately precede the first gouty paroxysm; but in connection with subsequent paroxysms prodromata are usually marked, so that confirmed gouty patients can predict when an attack is imminent. The most obvious of these are digestive and hepatic disorders, attended with abundant flatulence and eructations, heartburn, acidity, and constipation or diarrhœa, with unhealthy stools, the tongue being often large, flabby, and much furred; palpitation or uncomfortable sensations about the heart; catarrh of the throat and respiratory passages, tonsillitis, violent fits of sneezing, or asthmatic attacks; derangements of the nervous and muscular systems, indicated by a liability to headache, giddiness, attacks of megrim, noises in

the ears, disorders of vision, irritability of temper and fretfulness or lowness of spirits, languor, impairment of mental vigour and intellectual hebetude, heaviness or drowsiness, sleep, however, being restless, disturbed, and attended with unpleasant dreams, peculiar grinding of the teeth, sometimes associated with somnambulism, numbness or tingling in the limbs, especially in the fingers or toes, neuralgia or neuritis in various parts, twitchings, startings in the limbs, muscular cramps, especially in the calves of the legs, or possibly even convulsions, lumbago and other forms of muscular rheumatism; profuse perspirations; certain skin-affections; and changes in the urine. This excretion usually tends to become high-coloured and deficient in quantity, and to deposit urates abundantly, or even crystals of uric acid. In advanced cases of gout, however, the urine may be pale, watery, and clear immediately before an acute attack supervenes. Some patients are warned of the approach of a gouty fit by feeling unusually well, both physically and mentally.

2. Acute Articular Gout. — The first acute fit of gout comes on as a rule during the night, usually between 2 and 5 A.M. The patient is aroused from sleep by uneasiness or pain, generally referred to the ball of the great toe on one side, and the joint is found to be inflamed, the inflammation increasing in intensity, until it usually becomes extremely severe. In some instances the corresponding joints on both sides are attacked simultaneously, in rapid succession, or alternately. It must not be forgotten that gout may start in any of the smaller joints of the foot or hand, or even in the middle-sized joints, especially the knee or ankle. It has been affirmed that, next to the great toe, the metacarpo-phalangeal joint of the index-finger is most liable to be first attacked. Exceptional cases have come under the writer's notice, in which the disease implicated several joints at a very early period of its course, the feet, however, being free.

Severity of the pain is certainly a striking feature in the majority of cases of acute gout, especially in early attacks. Directly the foot is affected, any attempt to stand causes much suffering, and the pain speedily increases, until it becomes very intense, or almost unbearable. It is described in different cases as burning, throbbing, aching, tearing, plunging, boring, or piercing. The pain prevents sleep during the night, but towards morning it tends to diminish, and during the day there is usually comparative ease, an exacerbation again taking place as evening approaches, which goes on increasing towards night. Tenderness is very marked, and is often so exquisite that the patient dreads to be touched, and cannot bear the least movement or jarring of the affected part, or sometimes even the weight of the bedclothes, or the slightest shaking of the room. The objective signs of inflammation in connection with a gouty joint also soon become very prominent as a rule.

These are marked redness, which may be very deep and sometimes tends to duskiness or lividity, while the veins are enlarged and turgid; considerable local heat, as revealed to the touch and the clinical thermometer; and much swelling, the skin covering the part assuming a tense and shining appearance. When several joints in the foot or hand are affected, diffused redness and swelling are noticed. The tumefaction is due to effusion not

only into the interior of the articulation, but also into the surrounding tissues, subcutaneous oedema being a marked feature in connection with gouty inflammation. This can be better appreciated when the acute symptoms subside, so that pressure can be borne, which shows the pitting characteristic of oedema, and this may hold on for some time. However intense the objective signs of inflammation may be, it is an extremely rare event for acute gout to end in suppuration. As they subside, marked desquamation of the skin usually takes place. As the swelling increases, the subjective sensations generally diminish in severity; but during the progress towards recovery, intense itching is apt to supervene.

An acute attack of gout is almost always attended with more or less *general* symptoms; but it is an important fact that their severity depends upon the extent and intensity of the local manifestations of the disease, and upon the accompanying symptoms. Chills or even actual rigors may be felt at the outset, followed by febrile phenomena, sometimes slight, in other cases marked, the pyrexia being as a rule strictly secondary or symptomatic. The skin feels hot, and usually perspires, but not profusely; the temperature is moderately raised, presenting no definite variations, though marked remissions are generally observed towards morning; the pulse is increased in frequency; and the digestive organs are much disordered, as evidenced by anorexia, thirst, thickly furred tongue, offensive breath, and constipation. The urine is generally deficient in quantity, and may be very scanty, high-coloured, and concentrated; its acidity is increased; and on standing, an abundant deposit of amorphous urates often forms, varying in colour according to circumstances, being pale-buff, yellowish-red, dark or brick-red, or intense pink if the fever is high. According to the most recent researches the excretion of uric acid is increased during the course of an attack of acute gout, especially towards its close; and there is also said to be an excess of the alloxur-bases. The patient is usually exceedingly restless, and cannot lie with comfort in any position; sleep is much disturbed or altogether prevented; and cramps of the calves of the legs or of other muscles may still further aggravate the suffering. All these symptoms tend to increase the constitutional disorder. The temper is generally very irritable, and may even be violent.

The duration of the first fit of gout varies. It usually ranges from four or five days to a week or ten days, but may last two or three weeks or more, there being then commonly intermissions or remissions, and several joints being involved in succession. The termination of the gouty paroxysm may be attended with critical phenomena, such as free perspiration, diarrhoea, or a very abundant discharge of urates. After the attack the patient may not recover his previous health for some time; but not uncommonly he feels better than before. As a rule, the affected joints are apparently quite restored after early attacks of gout; but more or less stiffness or even deformity may remain. Oedema may also persist for a considerable time, especially if the inflammatory condition has been prolonged.

One of the characteristic features of gout is the tendency which the paroxysms exhibit to recurrence with increasing frequency. In not a few instances the second fit does not occur until an interval of two or three years or more has elapsed, but in most this is not prolonged beyond a year. The same interval

may be noticed between the next few paroxysms, but as the disease progresses they return twice a year, then more frequently, and at last become more or less constant. At the same time the mischief extends as regards the joints. It may be limited to the great toe for some time, but in successive fits spreads to the other articulations of the foot, to the hands, the ankles and knees, the wrists and elbows, very rarely to the hips and shoulders. In short, gout tends in time to involve nearly all the joints indiscriminately, and several may be implicated during a fit. Moreover, those articulations which are repeatedly attacked become more and more disabled and deformed. The symptoms in connection with a particular joint tend to diminish in intensity the more often it is affected. As additional articulations become involved, however, the general symptoms often increase in severity, and the patient does not recover in the intervals. The duration of the attacks becomes longer as their frequency increases. The rapidity of the progress of gout is very different in different cases; and the time taken to produce permanent mischief in the joints varies considerably.

In feeble persons the subjective and objective symptoms of a gouty paroxysm may be comparatively slight, but the ultimate effects upon the joints are often much worse. In some instances articular gout develops very insidiously, without any acute attacks. The pain in connection with a particular joint depends considerably on its structure, being usually much more marked if its ligaments, or the parts around, are rigid and unyielding. Previous injury or disease affecting a joint may likewise modify the symptoms.

3. **Chronic Articular Gout.**—In course of time more and more of the joints become permanently and obviously affected, a condition of chronic articular gout being established. The permanent changes are indicated by the articulations becoming enlarged, deformed, and irregular in shape, often presenting nodulations or bulgings, which may attain a large size. They are also stiff and crippled in their movements, at last becoming quite immovable and rigid; and being either permanently flexed, extended, or sometimes even bent backwards. The interference with movement and the deformity do not bear any necessary proportion to each other, the one or the other predominating according to the mode in which the uratic deposit has taken place. The more this infiltrates the ligaments and surrounding tendons, the greater becomes the impediment to movement. The fingers are particularly liable to be much altered in chronic gout. Tophaceous nodules are often formed in connection with the joints known as 'Heberden's nodes.' A common condition is said to be stiff flexion of the metacarpo-phalangeal and second phalangeal joints of a finger, with over-extension of the first phalangeal joint, so that its knuckle presents a deep hollow. Deflexion of the fingers to the ulnar side of the fore-arm is observed in some gouty cases. The toes are also sometimes distorted towards the outer side of the foot.

Gouty concretions in connection with joints, by their mere mechanical and irritative effects, are liable to cause damage to the adjoining structures. They may be seen stretching or shining through the skin, and causing it to assume a bloodless appearance, or, on the other hand, rendering it congested and bluish, the veins also being enlarged. Ulti-

mately the entrance of pyogenic cocci may produce an abscess around a concretion ; or more frequently the skin may merely give way from the continued pressure. Thus the so-called *chalk-stones* are exposed, and may come away either in a liquid form or as solid particles or masses ; occasionally there is a free discharge of pus as well. Ulcers are left, of an unhealthy and atonic character, and usually presenting no disposition to heal. There may be a number of these ulcers in the same individual, on the hands and feet.

When bursæ are involved, much deformity is produced. They are easily felt, usually presenting a combination of hardness and fluctuation, due to the presence of both concretions and fluid in the bursal cavity. These signs are chiefly noticed in the bursa over the olecranon. Abscesses may also form in connection with these deposits, and the latter may thus be completely got rid of, the abscess subsequently healing rapidly.

In cases of chronic articular gout the general system tends to become permanently affected. The patients are generally more or less feeble ; they may be thin and pale or sallow-looking ; or plethoric, but with flabby tissues, and presenting signs of languid circulation, with enlarged capillaries about the face. They often suffer from disorders of digestion and other symptoms already described ; but not uncommonly, as gout assumes a more chronic form, patients feel better, becoming habituated to their condition. It is remarkable that those suffering from numerous gouty abscesses often exhibit but little constitutional disturbance.

The urine in chronic gout generally becomes abundant, watery and pale, of low specific gravity, and deficient in solid ingredients. Very different statements are made as to the amount of uric acid and alloxur-bases discharged. Some writers affirm that they are increased ; others that they are diminished ; and still others that there is no constant relation, either as regards the total amount, or in the proportion between uric acid and xanthin-bases. This last view is probably correct. Deposits of urates in urine are not often observed in cases of advanced chronic gout, except before an acute exacerbation. Glycosuria is not uncommon in gouty persons, and there may be distinct diabetes mellitus. Oxaluria is also frequent.

4. Irregular Gout.—The clinical phenomena which are recognised as irregular manifestations of gout may assume either an *acute* or *chronic* form. They may be observed in persons who are distinctly subject to articular gout ; or in those in whom the disease is not so obviously revealed. Moreover, their intensity is often in an inverse ratio to that of the joint-affection, and the two classes of symptoms may exhibit a remarkable tendency to alternation, when the articular symptoms are prominent those connected with other parts being slight or absent, and *vice versa*. Acute symptoms are particularly liable to arise when, from any cause, during the progress of a gouty fit the joint-inflammation is checked suddenly or rapidly—*retrocedent gout*. At other times internal symptoms seem to be due to a want of development of the external phenomena—*suppressed gout* ; and when the latter appear, the former subside.

It must suffice to indicate here the general nature of the symptoms of irregular gout. The *acute* symptoms are mainly associated with the digestive apparatus, the circulatory system, the respiratory

organs, or the nervous system. In connection with the *digestive apparatus*, acute dysphagia may occur, attended with spasm of the pharynx and œsophagus. Acute inflammation of the salivary glands is of rare occurrence. The most important symptoms belonging to this group are those due to gastric disturbance. This may be of the nature of severe cramp or gastralgia, characterised by a sudden, intense, spasmodic pain in the epigastrium, relieved by pressure, and accompanied by a sense of great weight and oppression ; the patient presenting an aspect of much suffering, distress, and anxiety ; or being even more or less collapsed and prostrated. In other cases the symptoms are those of acute gastritis, bilious vomiting being prominent. Intestinal colic, or even muco-enteritis, may also occur in connection with gout. Hepatic disorder is common. The *circulatory system* is not uncommonly implicated. The heart is liable to be disturbed in its action during the attacks of gastralgia, but this disturbance may also be observed independently. There may be severe palpitation, the action of the heart being very rapid, irregular, or even intermittent, accompanied by most unpleasant sensations over the cardiac region, præcordial anxiety, often a feeling of oppression or constriction, dyspnoea or a sense of suffocation, and much distress, anxiety, and dread of death ; the pulse tends to be weak and small, or may be irregular ; and sometimes the attacks are attended by signs of collapse. In other instances the cardiac disorder is evidenced by very feeble or slow action, with a tendency to syncope. Again, there may be all the phenomena of a severe anginal attack (*see* ANGINA PECTORIS). Sansom, in discussing the effect of influenza on the gouty condition, expresses the opinion that the most important association of the post-influenzal irregular heart is with gout. Irregular gout affecting the *respiratory system* is mainly indicated by asthmatic paroxysms. In some cases there is a marked liability to acute catarrh of the air-passages, and crystals of uric acid have been found in the expectoration. Bronchitis is more obstinate in gouty subjects. Pulmonary congestion is also supposed to be a manifestation of retrocedent gout in some cases ; and gouty pneumonia has been described. In connection with the *nervous system* gout may give rise to attacks of severe headache or vertigo ; delirium or even acute mania (*see* INSANITY IN SPECIAL DISEASES) ; epileptiform or convulsive fits ; cerebral or spinal meningitis ; acute neuralgia, either external or internal, and especially sciatica, probably due to neuritis ; or severe muscular cramps. Apoplexy from cerebral hæmorrhage has been attributed to suppressed or retrocedent gout, but this can only occur if the vessels of the brain are diseased. Cerebral congestion might give rise to a temporary apoplectic attack. Among the acute manifestations of irregular gout may be further mentioned certain skin-affections, as eczema, erythema, or urticaria ; affections of mucous membranes, such as the conjunctivæ and lachrymal passages ; functional renal disorder, with albuminuria, or irritability of the bladder ; and external local signs of inflammation, associated with uratic deposit.

Many of the more *chronic* symptoms associated with gout have already been pointed out, when speaking of its premonitory symptoms, and only certain special phenomena need be alluded to here. Chronic skin-diseases are of frequent occurrence, namely, psoriasis, chronic eczema, pruritus—

either local or general—and acne. These may alternate very distinctly with articular gout. The nails are sometimes peculiar, being coarse and fibrous, striated and fluted, or lined vertically. Their substance may grow thick and brittle. In some gouty subjects daily paroxysms of heat and redness of the nose, attended with severe itching and irritation, cause considerable annoyance or distress. Violent sneezing fits are common. Many of these individuals are also liable to chronic catarrh, affecting the throat and the air-passages; and special appearances of the throat, tongue, and teeth have been described. In course of time gouty patients often become permanently asthmatic, the lungs being emphysematous, and dry bronchial catarrh being established. Gravel or urinary calculus may give rise to symptoms; or those indicative of chronic urethritis or cystitis may be present; especially in persons advanced in years. Permanent disorders of sensation, or slight local paralysis, may result from chronic changes involving particular nerves. Gouty persons are usually very sensitive to pain. Certain affections of the eyes have been attributed to gout, and among them a form of gouty iritis, described by Hutchinson as coming on insidiously, and almost painlessly, and ending in destruction of the eye. Changes in the external auditory meatus may also be associated with this complaint.

With regard to *tophi*, these can be seen or felt, provided they are superficial. Those connected with the helix of the external ear are most common; but they may also be looked for in the sclerotic or eyelids, in the nose, and under the skin, in the region of tendinous aponeuroses, especially in the leg or thigh. Tophi are originally liquid, and if one is punctured at an early period, an opalescent or milky fluid escapes, which on microscopical examination is found to contain an abundance of delicate acicular crystals; subsequently they become more consistent, and ultimately quite solid and hard, being then made up entirely of these crystals, which are closely aggregated together and interlaced. Taking the ear as an illustration, at first a small elevation appears under the skin of the helix, like a vesicle, having a soft feel. This gradually hardens until finally a little bead-like or pearl-like body is formed, presenting a whitish colour as seen through the skin. In course of time the cutaneous covering may be destroyed, leaving the little concretion exposed; or this may even become detached and removed, so that only a small depression is left.

5. Symptoms due to Chronic Organic Diseases.—In addition to what has been stated under the preceding heading, it is only necessary to refer here to the renal and cardio-vascular changes which are associated with gout. Disease of the kidneys is indicated mainly by the changes in the urine, other symptoms of chronic Bright's disease being often very obscure. The cardiac lesions are revealed by their respective physical signs; and there may be symptoms, first of excessive cardiac action from hypertrophy, and subsequently of cardiac failure, as well as those of particular valvular lesions. The vascular changes are discovered on examination of the arteries; and by their effects upon the circulation. It is impossible to describe within the limits of this article the numerous symptoms resulting from the various chronic organic diseases which may develop during the progress of gout, and which in many cases ulti-

mately produce a very complicated clinical picture. See HEART; ARTERIES; and KIDNEYS.

DIAGNOSIS.—A distinct gouty diathesis or tendency may be present, which it is important to recognise, while the joints are quite free from any apparent mischief. In most cases, however, the diagnosis has to deal with the nature of an articular affection, and to determine whether this is gouty or not. The chief diseases from which gout has thus to be distinguished are acute or chronic rheumatism, rheumatoid arthritis, and possibly gonorrhoeal synovitis. The data upon which a conclusion has usually to be formed with regard to a first attack are the presence or absence of an hereditary tendency to gout, as well as its intensity; the age and sex of the patient; the social position, occupation, and previous habits; the presence or absence of any obvious cause for the attack, or of premonitory symptoms; the localisation and characters of the joint-affection; the general symptoms; the character of the urine; the duration of the illness; and the absence of any cardiac complication.

The diagnosis in subsequent attacks is generally easier. If the metatarso-phalangeal joint of the great toe is alone inflamed several times in succession, or even if only the smaller joints of the feet and hands are implicated, the diagnosis of gout is tolerably certain. Then the presence of the permanent articular changes induced by gout, and of tophi in other parts, is of material assistance. Moreover, the urine may give evidence of renal mischief. In very doubtful cases it might be desirable to raise a blister, or even to take a little blood from the patient, and endeavour to obtain crystals of uric acid from the serum. The Röntgen rays may afford decided help, tophi being more transparent than the bony outgrowths found in rheumatoid arthritis.

In distinguishing gout from acute rheumatism, the absence of any hereditary tendency to gout and the existence of a family predisposition to rheumatism are important facts. Moreover rheumatism occurs most frequently for the first time in early life, and is not uncommon even in young children. Though more common among males, it often attacks females. It is not favoured by the habits which generate or promote gout, and affects all classes of persons, but especially those who from their occupation are liable to be exposed to cold and wet. The joints involved are the middle-sized or the larger ones, several of which are generally implicated in succession during the illness, the rheumatic inflammation exhibiting an erratic character; the local symptoms tend to be less severe than in gout; there is less marked oedema about the joints, and no enlargement of the veins, or subsequent desquamation. It must not be overlooked, however, that gout may attack the middle-sized joints. Pyrexia is high as a rule in acute rheumatism, and is often quite out of proportion to the extent of the articular affection; while profuse acid perspiration is almost always a prominent phenomenon. In addition to other differences, chronic rheumatism is at once distinguished from gout by the entire absence of uratic deposits.

Rheumatoid arthritis is usually met with in females between twenty and forty years old. There is neither hereditary taint, nor a history of such habits as generate gout, but, on the contrary, the patients are generally poor, hard-worked, and badly fed; all joints seem to be equally liable to be affected, both large and small, and the local sym-

ptoms are not of a very acute character, though the pain may be very severe, but they tend to continue for a long period. The general symptoms are those of debility and anæmia; and Spender has drawn attention to certain early symptoms, especially cold hands and feet, constant dampness of the hands, pigmentation of the skin, and a rapid and compressible pulse. Rheumatoid arthritis is a disease which tends to progress, involving joint after joint, but it presents no periodicity in its attacks, and often advances without any intermission, as a subacute or chronic disease. Ultimately it often causes much deformity and crippling of the articulations, which creak and grate on movement, but this results from a very different pathological change from that which takes place in gout, for there is not the slightest deposit of urates, either in the joints or elsewhere; nor can any uric acid be obtained from the serum, even in the most extreme cases. The urine presents no special characters; and the kidneys are not diseased.

Gonorrhœal arthritis is distinguished from gout by the age of the patient; the distribution of the joint-affection; the absence of any tendency to recurrence, except in association with a fresh urethral discharge; and the different nature of the morbid changes, tophi never being formed.

As exceptional points bearing upon the diagnosis of articular gout, the following may be mentioned. It has happened that pyæmia beginning in the great toe has been mistaken for gout, but the progress of the case would soon clear up any doubt under such circumstances. Again, articular inflammation from injury might resemble gout; and, moreover, it must be borne in mind that such an injury may really set up gouty inflammation for the first time, so that the joint may not recover properly. In some individuals the ends of the phalanges of the fingers are enlarged, especially the terminal ones, and cause nodulations—*nodi digitorum*—which resemble those of gout, and are by some regarded as being of a gouty nature.

In any gouty case the detection of the organic diseases liable to be set up in its course is of great moment in diagnosis, especially renal and cardiovascular changes; and also the association with their proper cause of catarrhal affections, skin-diseases, and other complaints, when these are really of a gouty nature.

PROGNOSIS.—The first point relating to the prognosis of gout which calls for notice refers to the immediate dangers in any particular case. A simple acute attack of articular gout rarely, if ever, kills the patient. When, however, internal organs are implicated, the matter becomes much more serious, and a fatal result may occur, so that the prognosis must be a guarded one under such circumstances. The danger becomes much greater if the complaint has been long established, and if the kidneys or other important organs have become structurally diseased. Indeed, these diseases of organs are often themselves immediately dangerous. Again, any acute inflammation occurring in a confirmed gouty subject is the more serious on this account; and the same remark applies to injuries and shocks of all kinds.

In the next place, the future of a gouty patient has to be considered, as regards the prevention of subsequent attacks. It must always be recognised that gout is a recurrent affection, and complete immunity can never be guaranteed, when once the

complaint has declared itself. At the same time not a few cases have occurred in which there has been but one attack. In giving an opinion on this point, the prognosis in any individual case will depend upon:—1. The degree of hereditary tendency to gout. 2. The age of the patient; for the earlier the period at which the disease begins, the less hopeful is the prospect of a cure. 3. The time the complaint has lasted from its commencement, and the frequency and duration of the gouty fits. If gout has become established, and especially if distinct tophi have formed, it is quite impossible to eradicate it. 4. The habits, mode of living, and occupation of the patient. It is only when the patient is prepared to adhere strictly to proper rules of living that a cure can be hoped for. Those who in their occupation are tempted to drink much, or who are exposed to cold or wet, are much less likely to be cured. It may be remarked here that gouty subjects are less able than others to resist exposure.

Another point bearing upon prognosis refers to the duration of life in gouty persons. If the disease comes on late in life, and the paroxysms occur only at long intervals, while the organs are free from any organic mischief, gout may not appreciably shorten life, and the patients may even enjoy good health up to an extreme old age, provided they are sufficiently careful in their mode of living, and no accidental complications arise. Chronic gout does, however, unquestionably tend to shorten the duration of life, to a greater or less degree in proportion to its severity, and more especially to the indications present that the kidneys, heart, arteries, or other important structures are organically diseased. This tendency is important in relation to life-insurance.

It has been affirmed that gout is a protection against certain other diseases, such as phthisis and diabetes mellitus, but this statement does not rest on any scientific data.

TREATMENT.—Although there are certain well-defined principles applicable to the treatment of gout in its various phases, every case must be considered on its own merits. It will be convenient to discuss this part of the subject under certain general headings, premising that the administration of medicines is often the least important part of the treatment, and that the habits of life of the patient always need thorough supervision in all their details.

1. Preventive and Curative Treatment.

Individuals who have a marked hereditary predisposition to the complaint may, by careful attention to their mode of living, do much to diminish the severity of the disease in later life and to prevent even its appearance. In all cases the willing co-operation of the patient is essential to success.

(a) *Diet.*—Moderation in the quantity of food is the first point to be attended to in the treatment of the gouty diathesis. It is not necessary or desirable to restrict persons who are gouty to a very low diet, especially if they are in any way weak, but an amount sufficient for proper nourishment in each individual case may be allowed, and at no meal should the stomach be uncomfortably filled. The meals must be taken at regular times, and not hurriedly. Very late dinners, as well as suppers, should be prohibited, but it suits many persons better to dine at half-past six or seven o'clock than at midday. The nature of the food is highly

important. It is quite unnecessary, as some recommend, to restrict the patient absolutely to a vegetable diet, but a due proportion of animal and vegetable aliments should be allowed. At the same time, in persons who have any tendency to gout, an essential part of the treatment often consists in diminishing the amount of meat which they consume, and they should not take it more than once a day. Indeed, the aim must be to reduce all kinds of proteid food, whether animal or vegetable, to such an amount as the system can satisfactorily dispose of, with due regard to the proper nutrition and strength of the body. As regards the kinds of animal food which are suitable for gouty subjects, white fish, chicken or fowl, game, tripe, and mutton are the best forms. Sweetbread is generally recommended, but it has been recently laid down that sweetbread and similar tissues rich in nuclein should be rigorously excluded. Soups had better be avoided, or should only be taken in very small quantity. Tender and underdone beef may be taken in moderation from time to time, if it is readily digested. Pork or veal, dried and salted meats, and rich dishes of all kinds, should be avoided. Lightly boiled or poached eggs may be permitted. Some authorities regard fats and milk as important elements in the diet of gouty patients, but this is a matter of personal experience. Vegetables which contain much woody fibre, or which create flatulence, must be eschewed; and certain articles such as rhubarb, tomatoes, especially when cooked, asparagus, and sorrel, are often injurious to the gouty. Those who are subject to gout should either abstain altogether from, or only take a very limited quantity of, sugar and saccharine articles of diet. Hence, although digestible fruits may often be taken with advantage in moderation, those which are very sweet must be used with particular caution. Stewed and baked fruits often agree well, but fruit-tarts, and, indeed, pastry of all kinds, should be interdicted. The juice of oranges or lemons is considered beneficial for gouty persons. Starchy foods are permissible in moderation. It may be laid down as a rule to be invariably followed, that gouty patients should limit themselves to simple meals, and not indulge in a number of 'courses;' and that they should avoid condiments and salt, as well as everything which is in their case indigestible.

(b) *Drink*.—No rules can be laid down applicable to all cases, but there are certain broad principles which have to be borne in mind. An abundance of good and pure drinking-water is to be commended, but it should be taken mainly apart from meals. It is a good plan for the subjects of gout to take a tumblerful of either cold or hot water before retiring to rest at night. Effervescent potass- or lithia-water may be substituted for ordinary water with advantage, but not soda-water. One or other of the natural 'table-waters' may also be allowed if desired. Tea and coffee may be taken in moderation, provided they do not disagree. With reference to alcoholic drinks, in a considerable number of instances one of the first objects in the treatment of the gouty diathesis should be the regulation of the use of this class of beverages. This indication is obvious enough when the condition is evidently due mainly to intemperance; but even when the patient is temperate, it may be that in his case the amount consumed needs to be reduced, or total abstinence

enforced, especially if there should be a strong hereditary predisposition to gout, or if the complaint appears in early life. Some patients are undoubtedly better if they abstain entirely from stimulants; others, however, can take those of a proper kind, and of good quality, in strict moderation, with advantage. It may be laid down as a general rule that malt-liquors and all stronger wines are injurious, and should be interdicted. Those which are most suitable are sound light claret, hock, moselle, chablis, or sauterne. A small quantity of good dry sherry suits some gouty patients very well. A little brandy, well diluted, often agrees better than any other kind of alcoholic liquor; in other cases whisky or gin answers best. Whatever stimulant is selected, it should only be taken at meal-times. Persons who are distinctly gouty should avoid excess on every occasion; and even if they do not invariably abstain from alcohol, they may find it beneficial to do so for limited periods, especially if there is a tendency to the development of gouty symptoms. It may be mentioned here that the treatment of selected cases of gout by an exclusive meat-diet, with an abundance of hot water, has been advocated.

(c) *General hygiene*.—Sedentary habits must be combated, whether due to occupation or indolence; and it must be insisted upon that a due amount of outdoor exercise is taken daily, although prolonged exertion, tending to cause fatigue and exhaustion, must be avoided. Walking and horse-exercise are highly beneficial, especially in the case of those who live rather too freely. Even carriage exercise is useful, so that the patient may have the benefit of the fresh air. For those who cannot have much regular exercise, cycling, lawn-tennis, fencing or single-stick, golf, and the like may be serviceable. As bearing upon the prevention of an acute fit of gout, it is an undoubted fact that some individuals who are subject to the disease seem to be able to ward it off by walking, riding, or adopting some other form of prolonged and violent exercise on the first intimation of a threatened attack. Gouty persons should rise early in the morning and avoid late hours. They should be relieved from excessive mental labour, or any great strain upon the mental faculties, but especially from worry and anxiety of all kinds. Attention must be paid to the cutaneous functions, the surface of the body being also protected from the injurious effects of cold. Warm clothing should be worn, in keeping with the weather, and those who can bear it should wear flannel next the skin. With regard to baths, many gouty persons are decidedly the better for using a cold or tepid bath every morning, followed by energetic friction; in other cases the employment of the warm bath at intervals, or of the Turkish bath, answers best. A course of treatment in a hydropathic establishment from time to time, under due medical supervision, is sometimes decidedly beneficial. Climate demands attention, whenever the circumstances of the patient allow a choice to be made. It may be advisable for gouty subjects to reside permanently in some warm and equable climate, or at any rate during the winter and early spring. In this way attacks may often be warded off, and the disease thus prevented from making progress. Those who are obliged to remain in England during the inclement seasons should avoid exposure to wet and cold, as well as sudden changes of temperature, and night-air. The bedroom should

be warm and well ventilated ; and in cold weather it is desirable to keep a fire burning during the night. Unduly heated and badly ventilated rooms, as well as crowded places of public resort, must be avoided.

(d) *Medicinal treatment.*—There can be no doubt but that the judicious use of certain medicines may assist materially in warding off or mitigating the gouty condition, and in preventing the occurrence of acute paroxysms. The digestive functions demand particular attention, and medicines which help these functions are often of the greatest service, if required. A course of alkalis or acids, according to the indications in each case, may prove most serviceable, combined with some simple bitter infusion or tincture, but acids must be employed with caution. Salts of potassium and lithium are used for special purposes in gout, but how they act is a matter of controversy, and cannot be discussed here. The bicarbonate of potassium, carbonate of lithium, or the citrates are the best salts to administer. They should be taken well diluted, and on an empty stomach. Magnesia or its carbonate may be given with advantage if there is much acidity, and if the bowels are habitually constipated. Saline aperients are often of great service, and they may be usefully administered in small doses, freely diluted, and regularly repeated, in order that they may act on the kidneys. In many cases other aperients may be employed at intervals with advantage, but strong purgatives must be used with particular caution. An occasional dose of blue-pill, calomel, or other cholagogues may be decidedly beneficial.

(e) *Mineral waters and baths.*—Certain mineral waters are of great value in the treatment of the gouty diathesis, when employed under proper medical supervision, and patients will often use them when they will not undergo a course of regular medicinal treatment ; while the water thus taken internally is itself of service. Space will not permit any lengthy discussion of this subject here, and it must suffice to mention that the kind of mineral water that suits one patient may be highly injurious to another. These waters are employed both internally and in the form of baths. Those chiefly used in gouty conditions are the waters of Bath, Buxton, Cheltenham, Clifton, Harrogate, Leamington, Malvern, Woodhall Spa, and Droitwich, in England ; Strathpeffer and Moffat in Scotland ; Llandrindod, Llangamarch, and Llanwrtyd in Wales ; Lisdoonvarna and Lucan in Ireland ; and Carlsbad, Marienbad, Vichy, Wiesbaden, Baden-Baden, Ems, Royat, Aix-la-Chapelle, Aix-les-Bains, Evian-les-Bains, Contrexéville, Dax, La Bourboule, Luchon, Kreuznach, Naheim, Ischl, Friedrichshall, Hunyadi János, Pullna, Schlangenbad, Teplitz, Seidlitz, Homburg, Kissingen, Rubinat, Wildbad, Ragatz, Gastein, Elster, Tarasp, Apollinaris, and other continental waters. Some of these may be taken regularly or at intervals, for the purposes which they respectively fulfil ; or, if circumstances permit, a systematic course of treatment from time to time, at certain of the places mentioned, may be recommended. See MINERAL WATERS.

2. *Treatment of Acute Gout.*—When a fit of acute articular gout sets in, our objects should be to shorten the attack ; to restore the affected parts as nearly as possible to their normal condition ; and to relieve symptoms. In the first place, particular attention must be paid to the *diet*. The aim should be to make this as low as is compatible with the

condition of the patient, especially if the fit is of a very acute type. In young and strong patients the diet should at first consist of milk, farinaceous articles, and abundance of water, barley-water, or toast-and-water. Those who are advanced in years, weak, or broken-down in health, or who have long suffered from gout, require a more nutritious diet, but it should be easily digestible, consisting of beef-tea and good soups, milk, and eggs beaten up, the quantities being regulated by the requirements of each case. As the symptoms subside, the food should be gradually improved, white fish, fowl, and meat being allowed in succession, but in strict moderation. If possible all kinds of alcoholic stimulants should be interdicted, but it may not be judicious to cut them off entirely in some cases, either on account of the previous habits or present condition of the patient, and then it is best to give a definite quantity of brandy or whisky, well diluted, with the food. For those who cannot take spirits, a little good hock or sauterne answers well. Under exceptional circumstances the writer has given a moderate quantity of good champagne even in acute gout with decided advantage.

As regards *medicinal* agents, colchicum has long held a most prominent place in the treatment of acute gout, and is commonly regarded as a specific. There can be no doubt as to the influence of this drug in relieving the symptoms and shortening the paroxysm of gout. Its effects must be watched, however, for it does not agree in every case. It has been alleged that colchicum renders the patient more liable to subsequent attacks of gout, but for this notion there does not seem to be any real foundation. The tincture or wine of colchicum may be given in doses of ten to twenty or even twenty-five minims every four or six hours, and either of these preparations may be combined with the citrate or bicarbonate of potassium or with a salt of lithium. Garrod considers that guaiaicum, though less potent than colchicum, has considerable power in relieving gouty inflammation and may be given when fever is absent.

Salicylates, especially salicylate of sodium, have been much recommended in the treatment of acute gout, and may sometimes be advantageously combined with the agents just mentioned. They certainly have proved useful in the writer's experience. Another class of drugs employed are phenazone, acetanilide, and allied agents, but they are not to be recommended. A group of remedies, much advocated of recent years on account of their remarkable solvent powers over uric acid outside the body, consists of piperazine, lysidine, lycetol, and allied agents. They have, however, by no means fulfilled the expectations formed with regard to them in the treatment of gout, and in the writer's experience, as well as in that of many other observers, have signally failed. Among special treatments inhalation of oxygen has been advocated in the treatment of acute gout.

It is necessary to keep the bowels acting freely by means of suitable aperients, and saline purgatives are of considerable value for this purpose. Other aperients, such as compound rhubarb pill, colocynth, resin or tincture of podophyllum, calomel or blue-pill, may be employed in appropriate cases. Diluents may be given freely, in order to promote the action of the kidneys ; and if the cutaneous functions appear to be defective, some mild diaphoretic may be administered, or it may even be desirable to employ the hot-air or vapour-bath.

Medicines may be needed for the relief of symptoms, especially pain and sleeplessness, for which Dover's powder or other preparations of opium, belladonna, chloral hydrate, sulphonal, or bromides may be indicated, but they must be used with discretion. In very severe cases the hypodermic injection of morphine is of much service.

Local treatment.—The affected parts in acute gout should be kept entirely at rest, and placed in a comfortable position, supported by pillows, and either horizontal or elevated, according to the feelings of the patient. In ordinary cases it is sufficient to wrap up the joints in flannel, or to surround them with cotton-wool completely covered with oil-silk or gutta-percha sheeting, according to Garrod's method. If the pain is considerable, other local applications might be used, of which most efficient are warm fomentations, to which tincture of opium or belladonna may be added, poppy-fomentations, lead-and-opium lotion, localised steaming, belladonna liniment, tincture of aconite, or a solution containing morphine and atropine. Some practitioners prefer evaporating lotions containing alcohol, ether, or chloroform; others warm-water compresses. A blister in the neighbourhood of a gouty joint may be of service, if the attack be asthenic, and also should effusion or much stiffness persist. During recovery, benefit may be derived from careful friction with some stimulating liniment, application of iodine, shampooing, gentle passive movements, douching with salt and water, or the application of a light bandage or elastic support, should there be a tendency to permanent thickening and stiffness, or to œdema and enlargement of the veins.

Cases of acute articular gout have been successfully treated by the Tallerman hot-air baths, and these are certainly worthy of more extensive trial. It is affirmed that subacute gout has been treated successfully by massage and galvanism combined.

The acute forms of *irregular gout* must be treated according to their nature, and here it must suffice to offer a few general remarks on the subject. If serious internal symptoms arise, which are distinctly of a gouty nature, and especially if they occur as retrocedent phenomena, it is important to try to excite inflammation in the joints, by means of local heat, friction, and sinapisms. Colchicum may be of service in the non-articular, as well as in the articular form of gout. In painful affections opium or other anodynes are called for; and frequently the administration of alcohol and other stimulants is indicated, with antispasmodics, such as ammonia, ethers, camphor, musk, or belladonna, especially when the stomach or heart is affected. In conditions attended with signs of much depression or collapse, external heat may be applied over the body, or sinapisms to the limbs and over the cardiac region. In the treatment of inflammatory diseases associated with gout much care is required, especially in resorting to depletory measures.

3. Treatment of Chronic Gout.—When gout becomes an established chronic disease, the same general rules of treatment are to be observed as in the prevention or attempted cure of the complaint. Similar medicinal remedies are also indicated, lithium-salts being particularly valuable, and they may even aid in removing gouty deposits; but others may be added to the list, which are suitable in different cases. Colchicum is often of much service, taken habitually or from time to

time, in the form of the extract at night, or a few minims of the tincture or wine two or three times a day, combined with other medicines. Among the many therapeutic agents recommended in the treatment of chronic gout under different circumstances may be mentioned benzoic acid or benzoate of ammonium, phosphate of ammonium or of sodium, iodide of potassium, bromide of potassium, carbonate of aluminium, lime-juice, guaiacum, ammoniacum, and tonics, especially quinine, tincture or infusion of cinchona, ferruginous preparations, and arsenic. Undoubtedly most of these are of use in appropriate cases of chronic gout, to serve their special purposes, but they must be used judiciously, especially iron. Garrod strongly advocates the administration of guaiacum in chronic gout, and for the purpose of averting acute attacks. If it causes intestinal irritation, he gives serpentary as a substitute. Symptoms connected with various organs frequently call for attention, and must be treated by appropriate remedies. Should diarrhoea set in, it must not be hastily arrested, as this may be a mode of relief to the system. With regard to the local conditions in chronic gout, the prolonged use of some of the mineral waters previously mentioned, both internally and in the form of baths, such as those of Aix-la-Chapelle, Aix-les-Bains, Homburg, and Baden-Baden, or definite treatment in various spas, not forgetting Droitwich in this country, may succeed in removing to some extent deposits of urates, and in diminishing stiffness and thickening of joints. For these purposes local measures may also be of service provided the morbid changes are not too far advanced, namely, occasional blistering or application of iodine; the prolonged use of wet bandages; friction with liniments; shampooing and passive movements; or systematic compression by means of some non-irritating plaster. Solutions of alkalis or alkaline carbonates, and especially of carbonate of lithium, have been kept applied to gouty joints and other parts for a long time, under the belief that deposits of urates may be thus dissolved. Massage, electricity, and the modern hot-air treatment have their advocates. Superficial uratic deposits should not be interfered with unless they become troublesome, when it may be desirable to puncture the skin and let the contents out. The propriety of removing large masses by operation may come up for consideration, but this should only be attempted if there is every probability that they can be entirely removed without any serious difficulty, and if the patient is in a fit state for the operation. When abscesses or ulcers form, they come under the treatment of the surgeon; but it may be observed that simple aseptic dressings usually answer best in these cases, and they may sometimes be advantageously dressed with a solution of carbonate of potassium or lithium. Under any treatment it is by no means an easy matter to induce lesions of this kind to heal in cases of chronic gout.

The treatment of the various chronic organic diseases which are liable to arise in the course of gout must always be kept in mind, but the reader is referred to other appropriate articles for a consideration of this part of the subject.

FREDERICK T. ROBERTS.

GRAIN-POISONING.—See ACTINOMYCOSIS; ERGOTISM; and PELLAGRA.

GRAM'S STAIN for BACTERIA.—*See* BACTERIA, p. 132.

GRAND MAL (Fr.).—A term applied to epilepsy when it assumes the form of a severe convulsive attack. *See* EPILEPSY.

GRANULAR DEGENERATION.—*See* DEGENERATION.

GRANULAR KIDNEY.—A morbid condition of the kidney, in which this organ is the seat of fibroid change, and as a result becomes contracted, hard, and granular. *See* KIDNEYS.

GRANULATION (*granulum*, a little grain).—The term 'granulations' is applied to small vascular prominences, consisting of embryonic tissue, growing on the surface of wounds or healing ulcers, by which the reparative process is carried on—whence the expression 'healing by granulation.' *See* WOUNDS, Healing of.

GRAVEL.—**DEFINITION.**—The deposit in and escape from the urinary passages of gritty particles.

VARIETIES.—Gravel may be composed of (1) uric acid and its compounds; (2) oxalate of lime; (3) phosphate of lime; or (4) the triple phosphate of ammonium and magnesium. By far the most common form of gravel is uric acid. This, owing chiefly to its great insolubility, is frequently deposited in the kidney and bladder, and is seen in the newly passed urine in the form of the well-known reddish-brown crystals, often described as resembling grains of cayenne-pepper. The supernatant urine is generally clear, rather dark in colour, and of a distinctly acid reaction. *See* OXALIC-ACID DIATHESIS; PHOSPHATIC DIATHESIS; and URIC-ACID DIATHESIS.

SYMPTOMS.—The passage of *uric-acid* crystals or gravel frequently causes no subjective symptoms, and is consistent with perfect health. Sometimes, however, it gives rise to, or is accompanied by, both general and local disturbance of function. The general symptoms are those of dyspepsia. Locally, there is dull aching in the lumbar region, not increased by movement; frequent micturition; a sense of heat and irritation at the neck of the bladder and along the urethra, especially during and after voiding water; and sometimes the appearance of a faint cloud of mucus or a slight tinge of blood in the urine.

TREATMENT.—From what has been said, it follows that the most important points in the treatment of gravel are strict limitation as to the *quantity*

of food; the avoidance of highly seasoned, very rich, or sweet dishes; the preference for vegetable rather than for animal food; abundant exercise in country air; and abstention from or the very sparing use of alcoholic liquors. Medicinally there may be given diuretics to increase the quantity of the urine, and facilitate the escape of gravel; pure water, alkalis, and alkaline waters freely diluted, to prevent the precipitation of uric acid; and saline aperients and saline waters, to promote digestion, and assist in ensuring the free action of the liver and alimentary canal. *See* CONCRETIONS. W. CADGE.

GRAVES'S DISEASE.—*See* EXOPHTHALMIC GOITRE.

GREEN SICKNESS.—A popular synonym for chlorosis, applied on account of the greenish colour of the skin sometimes present in that disease. *See* CHLOROSIS.

GRINDERS' ROT.—*See* PNEUMOCONIOSIS.

GRIPPE (Fr.).—A French synonym for influenza. *See* INFLUENZA.

GROWTH, Disorders of.—*See* ATROPHY; HYPERTROPHY; and MONSTROSITIES.

GRUTUM.—*See* MILIUM.

GUINEA-WORM.—*See* ENTOZOA.

GUMBOIL.—*See* MOUTH, Diseases of.

GUMMA (Lat., gum).—A growth occurring in syphilis, so named on account of its supposed resemblance to gum. *See* SYPHILIS.

GUMS, Diseases of.—*See* MOUTH, Diseases of.

GURGLING.—A physical sign heard on auscultation of the chest or abdomen in certain conditions, due to the movement of gas and fluid within a cavity, whether normal or abnormal. A gurgling sensation may also be felt at times in the intestines, as over the cæcum in typhoid fever. *See* PHYSICAL EXAMINATION.

GUTTA ROSEA (*gutta*, a drop; *rosea*, rosy).—A synonym for acne rosacea. *See* ACNE ROSACEA.

GYMNASTICS (*γυμνός*, naked).—*See* EXERCISE.

GYNÆCOLOGY (*γυνή*, a woman; and *λόγος*, a word).—This term signifies the study of the diseases peculiar to women. *See* UTERUS; OVARIES; FALLOPIAN TUBES; MENSES; &c.

H

HABITS.—**SYNON.**: Drug-habits. — **DEFINITION.**—The conditions produced by the continuous self-administration of certain drugs on account of the pleasurable sensations at first produced by them. After a time the drug is continued not so much in order to obtain its pleasurable effects, which become less and less marked, as to avoid the depression and unpleasant sense of want which follow its discontinuance.

The following 'habits' will be considered: Arsenic; Cannabis Indica; Chloral Hydrate; Chloroform; Cocaine; Ether; Morphine; and Paraldehyde. For Sulphonal, Trional, and Antipyrine *see* SULPHONALISM.

Arsenic-habit.—Excepting in the arsenic mines of Cornwall the drug is not often taken in this country as a habit. In other cases the habit generally originates during the treatment of some skin-

affection or other chronic disorder. In Styria whole families take arsenic to counteract the fatigue of mountain-climbing.

Habitués will take four to eight grains or more for a dose without ill-effects, while these are brought on by minute doses in other persons. Among those who can take the drug with impunity the effects produced are exhilaration without intoxication, a sallow, waxlike complexion, and some depression in the interval between the doses. For the symptoms of chronic arsenical poisoning see p. 99.

TREATMENT.—In mild cases, and when the drug has not been taken for any length of time, it should be at once withdrawn; but in old-standing cases the dose should be gradually reduced; for a fatal issue has followed the sudden discontinuance of the drug.

Cannabis Indica-habit.—This drug is largely used in India, Egypt, and other Eastern countries. It is chewed, smoked, and taken with alcoholic liquors. The preparations of the drug used are Bhang, Haschisch, and Ganji.

When symptoms arise they take the form of dyspeptic troubles, anæmia, wasting, and mental enfeeblement, and may terminate in chronic insanity and death.

The *treatment* is conducted on the same principles as in the case of the Morphine-habit (p. 611).

Chloral-Hydrate habit.—This is commenced almost invariably on account of insomnia, and continued in order that sleep may be obtained and the physical distress caused by the discontinuance of the drug avoided. The *dose* is a matter of idiosyncrasy. Some can take forty grains or more nightly without ill-effect; others are highly susceptible to the action of the drug. The amount taken is not as a rule continually progressive. The largest recorded daily dose was 300 grains.

SYMPTOMS.—There is produced a dull, apathetic, listless condition, with intellectual foginess, feeble will-power, and a muddled mental state, sometimes with hallucinations. The heart's action becomes feeble; the pulse rapid, weak, and soft; and the extremities cold. Digestive troubles and dyspnoea are complained of. There is muscular weakness, chiefly affecting the lower extremities, which may proceed to such an extreme degree that a condition resembling paraplegia may be produced. As a result of the vasomotor disturbance the skin is liable to urticaria, diffuse inflammatory erythema, and bullous eruptions; ulceration of the nails may occur. Albuminuria and a spurious glycosuria may be found (see GLYCURONIC ACID). If the drug be suddenly discontinued there is not the same amount of depression as is produced by a sudden cessation of the Cocaine- or the Morphine-habit; but in a case with well-marked symptoms the sufferings endured from sudden inhibition of the drug are severe.

The *Prognosis* is more favourable than in the Cocaine- or the Morphine-habit.

TREATMENT.—The patient should be placed in bed in a warm room. The drug should be reduced rapidly, and altogether abandoned in less than a week. In a mild case immediate withdrawal can be practised. If cardiac tonics are needed, strychnine, digitalis, or sparteine may be given in ordinary doses. Alcohol should not be employed; for, although it is usually taken by those who have the chloral-hydrate habit, it is not well borne, and may produce dangerous depression. Hot coffee should

be given freely; and caffeine, with or without phenacetin or antipyrine, will be found of service for the headaches, neuralgias, and flushes that may distress the patient. Massage and the hot pack will assist in combating insomnia, and a change of scene and air will be beneficial.

Chloroform-habit.—Chloroform may be inhaled for pain or out of curiosity. The *dose* is generally unknown, the amount being rarely measured. A pint a day has been used. Fatal termination from an overdose is not uncommon.

SYMPTOMS.—The effects produced by the drug are at first everything that is delightful. A series of brilliant images and gorgeous pictures fill the imagination, each vision more beautiful than the last. After a time the pleasurable sensations fail to appear, and the victim experiences nothing but terrible nightmares. The general symptoms are nausea, flatulence, diarrhoea, enfeeblement of the heart, coldness of the extremities, and other conditions due to sluggish circulation and lowered vitality, together with mental anxiety and nervous depression.

TREATMENT.—This should be similar to that adopted for the chloral-hydrate habit; but the chloroform should be stopped at once.

Cocaine-habit.—Cocaine is taken as snuff, in wine, hypodermically, or by mouth to ease pain and distress, and to allay hunger and fatigue; the drug is continued at first for pleasure, and later because the unappeased desire causes suffering. Among the cases which have come under the notice of the writer the daily *dose* ranged from 5 to 50 grains. A daily dose of 90 gr. has been recorded.

SYMPTOMS.—At first the effects are wholly agreeable. They consist of a rapid relief from pain and worries, a sense of elation, new life, vigour, and increased mental power; but the mental and bodily collapse supervenes far more speedily than in addition to morphine.

A little later, the main symptoms are loss of weight, dyspepsia, anorexia—though occasionally the appetite is ravenous—constipation, restlessness, a peculiar inability to attend for more than a few minutes at a time to any one occupation, and talkativeness, which, together with dilatation, and insensitiveness of the pupils, follows on a full dose. Sleeplessness and intense depression occur if the accustomed dose is not obtainable.

Later on, after a full dose, there will still be intellectual exaltation, brilliancy of discourse, and a loss of the sense of time, followed shortly by a dull, moody, dissatisfied, and limp mental condition—symptoms indicating the evanescent character of the intoxication. To escape the recurrence of the depression the drug is taken more frequently; and in a few months symptoms of profound degeneration and debility will appear. The face becomes cadaveric, the eyes sunken, the body wasted; there is trembling, nervousness, and insomnia; the memory is deficient, and there may be distressing attacks of dyspnoea and palpitation, noises in the ears, hallucinations, muscular twitchings, tonic or clonic spasms, or even epileptiform convulsions which may prove fatal. Impotence is the rule, but it may be accompanied by easily roused sexual excitement. Albuminuria may be present. There is itching of the skin, and the sufferer may lacerate his skin not only on account of the irritation, but to try to rid himself of insects (the 'Cocaine bug,' as it is termed in America) which he fancies are crawling about his skin. A stage is sometimes reached when

there is a fear of being alone, yet a distrust of all companions; and a melancholic or maniacal condition may arise, which may continue for some time after the drug has been abandoned.

When the cocaine-habit is suspected, the body should be examined for needle-marks and abscess-scars, which are similar to those found in morphine-habitués. A stage of want can be produced in a few hours, as advised under the morphine-habit, and the urine can be analysed for cocaine.

PROGNOSIS.—Cocaine, being the more attractive drug, is not so readily given up as morphine, but the conditions affecting the prognosis are identical.

TREATMENT.—The reduction of the daily dose should be spread over 2 to 20 days, according to the symptoms and the severity of the case. Bromides may be given on lines laid down under 'Morphine-habit.' Strychnine should be given: as hypnotics, morphine and paraldehyde are useful. For detailed treatment, the restlessness, neuralgias, and other symptoms, see 'Morphine-habit.'

Ether-habit.—Ether prepared from methylated spirit is generally used. The habit is met with in the north of Ireland. A dose 1 to 4 drachms or more is taken neat, preceded and followed by a draught of water.

SYMPTOMS.—Intoxication of a quarrelsome type rapidly appears; coma and subsequent sobriety follow, all in a period of about 2 hours. The process can be, and often is, repeated several times during one day. As a rule there are no ill-effects. In chronic cases the symptoms are similar to those produced by chronic alcoholism.

The treatment is the same as that for Alcoholism (see p. 42).

Morphine-habit.—**SYNON.**: Morphinomania; Opium-habit; Fr. *Morphinomanie*; Ger. *Morphiumsucht*.

The habit of taking morphine or opium is usually commenced for the relief of pain or insomnia. Opium-smoking is due to curiosity or the example of others. Laudanum-drinking is sometimes resorted to as an antidote to ague or rheumatism. The dose of morphine partaken of varies from 1 to 100 grains daily; of solid opium to over 100 grains daily. One pint of laudanum has been known to be taken daily.

SYMPTOMS.—The symptoms vary according to idiosyncrasy: no apparent ill-effects occur in some persons who take large doses for a length of time; in others, pronounced effects arise in a few weeks or months.

The face becomes pale or sallow; there is a peculiar aged appearance and greyness; the features are pinched and the figure bent; while emaciation and general aspect suggest malignant cachexia. The pupils are contracted after a dose: in the stage of want they may appear normal, dilated, or unequal, with defective accommodation. The conjunctivæ are at times congested, and the eyes sunken and glassy, with a dreamy, 'far-away' expression. After a large dose there may be huskiness of voice. The teeth become loose and decayed. The hair becomes grey and falls off. Although at first, and especially after a dose, there is increased mental activity and intellectual brilliancy, the memory becomes impaired, particularly for recent events. The appetite is at one time insatiable, at another absent. Constipation is the rule, but diarrhœa and a chronic dysenteric condition may arise, especially in the stage of want. Sexual desire is at first in-

creased, later absent. In females there is amenorrhœa: the pregnant woman is liable to abort. The virile power and the menstrual flow usually reappear shortly after the drug has been discontinued. Albuminuria may be present and may continue many weeks after the drug has been discontinued.

Sleep is fitful and uncertain. Lassitude and restlessness alternate with wakefulness and activity. Neuralgias occur, and a condition resembling ague may be experienced. Moral as well as physical degeneration ensues. The truthful, upright man may become unreliable and utterly untruthful. Indeed, the untruthfulness of drug-inebriates generally is proverbial. There may be hallucinations and delusions, and a state resembling *petit mal* may arise, during which criminal acts may be performed. Apart from this condition, it is not unusual for resort to be had to lying, cheating, and stealing, if the drug be otherwise unobtainable. As an aid to diagnosis the body should be examined for needle-marks (indurated and often discoloured patches, mostly circular), which, in old-standing cases of hypodermic injection, will cover the body. Scars of abscesses of various shapes will probably be found in chronic cases. These latter are not nearly so frequently found among patients who employ morphine in some form which requires warming before use. If this examination be not permitted, a stage of want may perhaps be brought about by keeping watch on the patient's movements for a few hours, and by stopping all possible channels of supply; but the watcher must be acutely observant, as the majority of drug-takers resort to innumerable devices to keep their habit secret and to guard against deprivation. The urine may be examined by the iodic-acid test. Morphine may be found in the urine 5 to 8 days after the drug has been discontinued; if found after this time it is certain that the patient is still taking the drug. Morphine may also be found in the feces. In cases where the drug is not being taken by the mouth or hypodermically it may be smoked in an ordinary tobacco-pipe or mixed up with tobacco in a cigarette, or introduced *per rectum*. See URINE, morbid conditions of.

The symptoms of sudden withdrawal indicate the intensity of the suffering. A stage of want is evinced by extreme restlessness, neuralgia and insomnia. If the withdrawal be continued, the secreting glands become abnormally active as shown by running at the eyes and nose, gastritis, diarrhœa, vomiting, cramps, and excruciating pains.

In 6 to 12 hours a condition resembling delirium tremens with hallucinations may arise, or even an acutely maniacal state which may persist for 48 hours. Sudden collapse and death have been known to occur.

PROGNOSIS.—The longer the drug-habit has lasted the more likelihood is there of a relapse. The daily amount of drug taken does not affect the ultimate result; the habit of short duration and large dose is more curable than that of long duration and small dose.

If the cause of the original habit be not removed, or if it subsequently return, it is probable that the habit will also be resumed.

TREATMENT.—The aim of treatment should be to reduce the drug rapidly and with as little suffering to the patient as possible. As immediate withdrawal entails violent suffering, some danger, a tedious convalescence, and results no more satis-

factory than those obtained by less heroic methods, this plan should only be employed for mild cases, in the young and strong, where the daily amount (if injected) is less than 5 grains.

Rapid withdrawal is advocated by Mattison of New York, and is an excellent procedure. Briefly, it is carried out as follows: with or without previous administration of bromides, reduce the morphine at once to $\frac{1}{4}$ or $\frac{1}{5}$ of the usual daily allowance, and in every case administer the drug by the mouth. Give bromide of sodium (30 grains) twice daily. Each day increase the daily doses of bromide by 20 grains, until on the ninth day two doses of 100 grains each are given. Gradually lessen the opiate till on the eighth to the tenth day it is entirely abandoned. On the day its regular use is discontinued, give at night $\frac{1}{4}$ of former (i.e. before treatment commenced) entire daily amount of morphine, on the next night $\frac{2}{5}$ of this dose, and on the following night $\frac{1}{3}$ of the first night's dose. With each of these doses give a maximum dose of bromide. Twenty-four hours after the last dose of opiate, put patient in bed and keep him there two to four days. Any other symptoms must be treated as they arise. A subsequent dose or two of morphine (by the mouth) may be required for insomnia if other remedies fail. The treatment must to some extent vary with each case. The writer has sometimes employed far larger doses of bromide with advantage.

The *slow method*, extending over weeks or months, may be employed if, owing to organic disease, rapid reduction of the opiate is considered unsuitable. Oscar Jennings of Paris has demonstrated with the aid of sphygmographic pulse-tracings that digitalis, sparteine, and trinitrin have a very similar effect on the pulse of a morphine-habitué to that brought about by a dose of morphine. At the commencement of the treatment he reduces morphine by about $\frac{1}{4}$ of the customary daily quantity, and then by 1 grain or less each day. He gives digitalis as soon as the heart becomes sluggish. When a daily dose of about 2 grains of morphine is reached, for every sixth of a grain reduced (in the case of syringe-users), double that quantity is given by the mouth or rectum. By this means the syringe is shortly abandoned. The internal doses are then gradually reduced to zero.

Whatever method is adopted, it will be found almost impossible to keep satisfactory control over a patient in his own house, and if this be attempted, the result is generally discouraging to the medical attendant, the patient, and the friends. Wherever the treatment be carried out, the attendants must be ever on the alert against deception.

Every patient who wishes to be cured of his habit must be prepared to endure some suffering. Hare truly says, 'When a patient goes through the withdrawal without suffering, you need not flatter yourself that it is on account of your treatment; it is because he has a secret supply of his drug.' We must therefore expect to be called on to minister to a variety of ailments, the chief among which will be restlessness, insomnia, neuralgias, and digestive troubles. For restlessness the hammock, as first recommended by Jennings, is useful; for the insomnia and neuralgias various remedies are required. Both in the morphine- and the cocaine-habit, in the early stages of treatment, chloral-hydrate is apt to produce excitement instead of repose. It can be used in the later stages. Hot-air baths, massage,

and electricity will be at times beneficial for all the above symptoms. For digestive troubles soda and bismuth, rhubarb and soda, capsicum, hot-water enemata, hot fomentations, and especially milk-diet will be found of service. If stimulants are required, caffeine, kola, and ammonia should be employed—not alcohol. The writer has never found it necessary to administer alcohol to any case of drug-habit. Too often the drug-inebriate has been an alcohol-inebriate, and in any case he is readily converted into one. Every drug-patient should be warned against the use of alcohol. Tonics, especially strychnine, quinine, and iron, will be required. A sea-voyage under supervision, and perhaps a prolonged rest from all work, may be indicated when active treatment is suspended.

Paraldehyde-habit.—Employed for insomnia and continued on account of the sleeplessness and depression which may follow the disuse of the drug. The dose taken after several months' habit varies from one to six ounces daily.

SYMPTOMS.—The principal symptoms are loss of flesh, digestive troubles, the odour of the drug in the breath, palpitation and irregularity of heart, muscular tremors and inco-ordination, and insomnia. A condition resembling delirium tremens may be present.

TREATMENT.—This consists in the immediate withdrawal of the drug. Sleep must, however, be secured. For this purpose, bromide of potassium should be given freely. Five drachms or more may be required before bed-time. At bed-time chloral hydrate should be given in a 20- to 25-grain dose, followed, if required, by a smaller dose three hours later with a drachm of bromide of potassium. Sp. Ammon. Aromat. in 2-drachm doses should be given with each large dose of bromide, and two or three times during the night if the patient wakes and requires a stimulant. On the following day bromide of potassium may be required in 10- to 20-grain doses with Sp. Ammon. Aromat. \mathfrak{zss} . every two hours; and perhaps Pot. Brom. \mathfrak{zj} – \mathfrak{zj} . with chloral hydrate at night. Later a tonic treatment of strychnine and quinine will be required. Alcohol should not be given.

Sulphonal, Trional, Antipyrine.—With any of these drugs, 'habit' may arise, and the symptoms produced are very similar. See SULPHONALISM.

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HABITS, Personal.—See PERSONAL HEALTH.

HABIT-SPASM.—See OCCUPATION-DISEASES.

HÆMACYTOMETER.—See BLOOD, Examination of (p. 150).

HÆMATEMESIS (αἷμα, blood; and ἔμεω, I vomit).—SYNON.: Fr. *Hématémèse*; Ger. *Blutbrechen*. Vomiting of blood. See HÆMORRHAGE.

HÆMATHIDROSIS (αἷμα, blood; and ἰδρῶς, sweat).—Blood-stained Sweat. See SUDORIPAROUS GLANDS, Disorders of.

HÆMATIN ($C_{76}H_{46}N_4FeO_8$).—Hæmatin may be obtained from red blood-corpuscles by treatment with acids or with strong alkalis.

It is an amorphous dark-brown powder, insoluble in water, alcohol, or ether, but readily soluble in caustic alkalis, in sulphuric or acetic acids, and in chloroform. The acid and alkaline solutions each

give a characteristic spectrum. A solution gives a characteristic absorption-band in the spectrum, different from those produced by hæmoglobin. It forms a green solution when boiled with caustic potash. See SPECTROSCOPE IN MEDICINE.

HÆMATINURIA.—See HEMOGLOBINURIA.

HÆMATOBIUM (αἷμα, blood; βίος, life).—Living in the blood. See ENTOZOA; and MALARIA.

HÆMATOCELE (αἷμα, blood; and κήλη, a tumour).—SYNON.: Fr. *Hématocèle*; Ger. *Blutgeschwulst*.

DEFINITION.—A swelling occasioned by effusion of blood into the sac of the tunica vaginalis, or into a cyst connected with the testicle.

ÆTIOLOGY AND SYMPTOMS.—The extravasation of blood in hæmatocele may take place in a healthy state of the parts, or it may succeed or be combined with hydrocele. In both cases it may be occasioned by a blow, or by violent efforts made in straining, especially in old persons, or when the blood-vessels are diseased. It may happen also from the accidental wound of a vessel in tapping a hydrocele; or, without any such wound, a hæmatocele may supervene if the walls of the sac are very vascular, and a vessel, lacking the pressure of the hydrocele-fluid, gives way. The blood effused, if small in quantity, mixes with the fluid of the hydrocele, and occasions slight enlargement without disturbance. If it be large in quantity, coagula are formed; inflammation is excited in the tunica vaginalis; and plastic exudation occurs on its inner surface, sometimes forming layers, and rendering the sac extremely dense and firm.

The testicle preserves the same relation to the remainder of the tumour as in hydrocele, being situated at its posterior part. Its position, however, is liable to the same alterations which occur in hydrocele, but they are very difficult of detection, owing to the great thickening of the parts.

DIAGNOSIS.—A hæmatocele may be distinguished from a hydrocele by the absence of transparency; the obscure character of the fluctuation; the heavy feel of the tumour when balanced in the hand; the sudden and accidental mode of its occurrence, and the discolouration of the scrotum by infiltration with blood. In old chronic cases in which the tunica vaginalis and its envelopes have become much thickened and indurated, the tumour possesses so firm a character, and feels so heavy and solid, that it is very liable to be mistaken for a chronic enlargement of the testicle; and, more especially, for a malignant tumour, with which it is sometimes associated. The diagnosis is at all times difficult, and in some instances cannot be satisfactorily made out by the most experienced hands until an incision has been made. The records of surgery furnish many cases in which castration has been performed owing to a mistaken diagnosis. In all doubtful cases a trocar should be introduced, or, better still, an incision should be made before any serious operation, such as castration, is undertaken.

TREATMENT.—When a hæmatocele succeeds a hydrocele, the tinged fluid may be removed by tapping, but a radical cure of the hydrocele is now so easily effected, that few patients decline the slight risk the operation involves (see HYDROCELE). Even when inflammation arises, if the sac be tapped

and tension removed, and the patient be kept at rest, with ice applied to the part, the inflammation may subside. When, however, the blood effused is large in quantity, and when the inflammation is acute and threatens suppuration, the tumour should at once be freely laid open by incision, its contents evacuated, and the tunica vaginalis reflexa cut away. This must be done with care, so as to avoid wounding the testicle. Chronic hæmatocele with a very thickened sac must be treated in the same way. The practitioner must bear in mind that the testicle is sometimes situated in front, as in cases of inversion, and is then very liable to injury in the operation of incision, and even in tapping.

Encysted Hæmatocele.—Encysted hæmatocele implies an effusion of blood in the sac of an encysted hydrocele. The treatment is the same as that required for ordinary hæmatocele.

Hæmatocele of the Cord.—Blood may also be effused into the areolar tissue of the spermatic cord, constituting *diffused* hæmatocele of the cord; or into a cyst in the cord, constituting *encysted* hæmatocele of the cord. Such cases are very rare.

In diffused hæmatocele of the cord which persists or threatens to suppurate, incision and drainage may be required. An encysted hæmatocele should be dissected out and, if necessary, the inguinal canal repaired with buried sutures.

T. B. CURLING. C. B. LOCKWOOD.

HÆMATOCELE, Parenchymatous.—This term has been used to designate an effusion of blood into the testicular substance. It is nearly always caused by an injury, and is attended by acute pain. The withdrawal of the blood by means of an aspirator, or trocar and cannula, gives relief. The blood-extravasation may also take place into the epididymis.

C. B. LOCKWOOD.

HÆMATOCELE, Pelvic.—See PELVIC HÆMORRHAGE.

HÆMATO-CHYLURIA.—See FILARIASIS, p. 544.

HÆMATOCOLPOS.—See PELVIC HÆMORRHAGE.

HÆMATOIDIN.—This substance is one of the forms of iron-free hæmatin: it occurs naturally as rhombic prisms or needles of a red or greenish-red colour—a fact which shows that the colour of hæmoglobin is not dependent on the iron. It is of considerable pathological interest, being frequently found in old clots, and in the cavities of ruptured Graafian follicles. It is the cause also of the staining so often seen in the neighbourhood of extravasations of blood, varying from lemon-yellow to reddish-black.

HÆMATOMA (αἱματώω, I fill with blood).—A collection of extravasated blood forming a definite swelling. The contained blood may have undergone certain changes. See CEPHALHÆMATOMA; HÆMATOMA AURIS; PELVIC HÆMORRHAGE; and TUMOURS.

HÆMATOMA AURIS (αἱματώω, I fill with blood; auris, of the ear).—SYNON.: The Insane Ear; Fr. *Othématome*; *Hématome de l'Oreille des Aliénés*; Ger. *Othämatoma*; *Ohrblutgeschwulst von Geisteskranken*.

DEFINITION.—An affection of the auricle,

which occurs almost if not quite exclusively in the insane, and consists in the effusion of blood or bloody serum between the cartilage and its perichondrium, to such an extent as to form a distinct tumour.

ÆTIOLOGY.—In most of the few cases of hæmatoma auris which have been published to show that this disease may occur in the sane, the description given of the patients rather points to their insanity than otherwise. It is most common in cases of general paralysis and mania (acute and chronic), but also occurs in melancholia, dementia, and idiocy. It is about four times as frequent in men as in women; and more often affects the left ear than the right. Sometimes both ears are affected, but seldom at the same time. There would seem to be, in many or all of the insane, a morbid condition of the vessels or other tissues of the auricle, which predisposes to the occurrence of hæmatoma. If this condition be present to a sufficient degree, the disease may arise spontaneously; in other cases a very slight injury may be sufficient to cause it; while in others very considerable violence is necessary for its production.

SYMPTOMS AND COURSE.—The disease first makes itself evident by the appearance of a swelling of about the size of a horse-bean; this is almost always upon the anterior surface of the pinna, and usually in the neighbourhood of the fossa of the antihelix. The skin over the tumour is generally of a reddish or bluish-red colour, but may be unaltered at first; the temperature of the ear is sensibly raised; the swelling is very painful and tender; there is no extravasation of blood from the cutaneous vessels; and the tumour is not œdematous. At this stage, the effusion which has taken place between the cartilage and its perichondrium consists of dark-red fluid blood. In rare cases the swelling does not increase further; the inflammatory symptoms subside after about a week; absorption gradually takes place; and only a slight thickening remains. More usually the tumour increases and may attain the size of a hen's egg; it becomes tense, elastic, distinctly fluctuating, and is often hot and of a bright red colour. Its prominent anterior wall, consisting of skin, cellular tissue, and perichondrium, is felt to be thinner and less resisting than the posterior, which contains the ear-cartilage. In certain cases, however, owing to the brittle cartilage having split up and portions of it having adhered to either wall, both walls present irregularly alternating characters. The time which hæmatoma takes to attain its largest size varies from a week to a month; it then generally involves the whole of the concha, occluding the external auditory meatus; the folds of the auricle are lost, with the exception of the helix (which appears as a band running round the tumour) and the dependent lobule. The weight of the tumour causes the whole ear to fall somewhat forwards and outwards. Sometimes, especially in the presence of constant or repeated irritation, the inflammatory stage may last many weeks, and the deformity which always results from the affection is thereby greatly increased. Unless subjected to violence, it very rarely happens that the tumour opens spontaneously, although its tense and inflamed appearance often seems to indicate that such an occurrence is imminent. If rupture does take place, suppuration ensues; portions of cartilage come away; the cavity closes very slowly; and

great deformity results. The most common course is for the inflammatory symptoms gradually to subside. The anterior wall becomes firmer owing to a new deposit of cartilage upon its inner surface; the sense of fluctuation is gradually lost; and the tumour slowly diminishes in size, often yielding a somewhat doughy sensation to the touch. Occasionally, at this stage, some gaseous contents have been observed in the cavity. The colour of the skin over the tumour becomes gradually more dusky; it then passes into yellow and, later on, into an unnatural pallor. As the fluid contents become absorbed, the tumour becomes harder and smaller; folds again appear in the auricle, but do not correspond to the original ones; and the pinna remains permanently thickened, puckered, and often nodular.

ANATOMICAL CHARACTERS.—Many of these have been given above in explanation of symptoms, and do not require to be repeated. A shrivelled auricle, which has previously been affected by hæmatoma, presents, on section, two distinct layers of cartilage: these are of varying thickness, and separated from each other by vascular fibrous tissue, which often contains within it other small isolated plates of cartilage and sometimes also small portions of bone. The two layers of cartilage have been developed upon the inner surfaces of the perichondrium. The loose portions of cartilage and bone which are occasionally seen are developed from the fibrous tissue. It used to be supposed that the bone (which is soft, vascular, and contains well-developed Haversian systems) resulted from ossification of the ear-cartilage; but the writer has shown that this is not the case.

PROGNOSIS.—The local affection is in no way dangerous, but it always leaves behind it a permanent characteristic deformity of the auricle. The sense of hearing is only affected by the occlusion of the auditory meatus; but this condition very rarely persists after the acute stage. The occurrence of hæmatoma auris influences the prognosis of the mental disease unfavourably, but does not necessarily indicate the approach of a fatal termination to the case.

TREATMENT.—Protection of the part from injury is usually all that is necessary. Cooling applications are useful when the inflammation is excessive. The tumour should not be opened; nor should a portion of the anterior wall be removed, as has been recommended; these procedures almost certainly lead to suppuration, owing to the extreme difficulty of keeping the wound aseptic in an insane patient. It is useless to empty the cavity by aspiration, as it fills again with great rapidity. Treatment by pressure is so painful that the patient will not submit to it.

C. SPENCER COBBOLD.

HÆMATOMETRA.—See PELVIC HÆMORRHAGE.

HÆMATOMYELIA.—Hæmorrhage into the spinal cord. See SPINAL CORD, Diseases of.

HÆMATO-MYELO-PORUS.—A form of hæmorrhage into the spinal cord described by van Gieson. See SPINAL CORD, Diseases of.

HÆMATOPORPHYRINURIA.—Hæmatoporphyrin ($C_{16}H_{18}N_2O_3$) is an iron-free derivative of

hæmatin, isomeric with bilirubin. It is present in minute amount in normal urine, and in larger, but still small, quantities in many morbid urines, which nevertheless show no striking peculiarity of tint. In the condition usually known as hæmatoporphyrinuria, the urine contains this pigment in considerably larger amount and has a dark portwine colour. This colour is only in very small measure due to the hæmatoporphyrin present, and mainly to other abnormal pigments of which very little is as yet known. The condition usually forms one of a group of toxic symptoms, which follow the taking of sulphonal, or even trional, for longer or shorter periods. Occasionally, but rarely, similar urine is passed by patients who have not taken these drugs.

In normal and even in ordinary morbid urines it is seldom possible to detect hæmatoporphyrin without the employment of one of the several methods of extraction, but in the dark red urines some of the bands of the so-called 'alkaline' spectrum or of the 'metallic' spectrum can usually be more or less clearly seen on direct spectroscopic examination. Only when a mineral acid is added does the 'acid' spectrum appear.—See SPECTROSCOPE IN MEDICINE: Coloured plate.

The occurrence of hæmatoporphyrinuria in patients taking sulphonal is of serious import, and calls for the immediate discontinuance of the drug. The administration of alkalis in such cases has been recommended, and should be tried. In cases in which no sulphonal has been taken hæmatoporphyrinuria is not usually a grave symptom.

A. E. GARROD.

HÆMATORRHACHIS.—Hæmorrhage into the spinal canal on either side of the dura mater. See MENINGES, SPINAL.

HÆMATOSALPINX.—See PELVIC HÆMORRHAGE.

HÆMATOTHORAX (αἷμα, blood; and θώραξ, the chest).—An extravasation of blood into the pleural cavity. See PLEURA, Diseases of.

HÆMATOZOA (αἷμα, blood; and ζῶον, an animal).—This term is applied to all kinds of animal parasites dwelling in the blood and blood-vessels. See ENTOZOA; FILARIASIS; MALARIAL DISEASE.

HÆMATURIA (αἷμα, blood; and, οὔρον, urine). SYNON.: Fr. *Hématurie*; Ger. *Blutharnen*. Blood in the urine. See HÆMORRHAGE.

HÆMIC MURMUR.—A murmur connected with an abnormal condition of the blood, as in anæmia. See ANÆMIA; HEART, FUNCTIONAL DISEASES OF; and PHYSICAL EXAMINATION.

HÆMIN (C₂₂H₃₀N₄FeO₄HCl).—Hæmin, which may be prepared from dried hæmoglobin by treatment with glacial acetic acid, in the presence of an alkaline chloride, is a hydrochloride of hæmatin. It crystallises tolerably readily in needle-shaped prisms or rhombic plates, and thus becomes an easy means of detecting the presence of blood in stains of a doubtful nature. The crystals are insoluble in water, alcohol, chloroform, and ether, and soluble in caustic alkalis and boiling hydrochloric and acetic acids. See HÆMORRHAGE (Hæmaturia).

HÆMOCYTOMETER.—See BLOOD, Examination of, p. 150.

HÆMOGLOBIN (αἷμα, blood; and *globus*, a ball).—The colouring matter of the blood. See HÆMATIN; HÆMATOIDIN; HÆMATOPORPHYRINURIA; HÆMIN; and HÆMOSIDERIN.

HÆMOGLOBINOMETER.—See BLOOD, Examination of, p. 152.

HÆMOGLOBINURIA (Hæmoglobin; and οὔρον, the urine).—SYNON.: Hæmatinuria.

DEFINITION.—A morbid condition of the urine resulting from disintegration of blood-corpuscles, characterised by change of colour of the excretion, in consequence of the presence of hæmoglobin and débris of red corpuscles; sometimes paroxysmal; sometimes a result of the action of poisons; and sometimes associated with other more dangerous symptoms, and tending to a fatal result.

ÆTIOLOGY.—Hæmoglobinuria is not referable to any structural change in the kidneys. It is a result of morbid action in the blood, consisting essentially in the disintegration of the red corpuscles whereby the hæmoglobin is set free. It has been observed in the course of fevers, particularly ague and scarlatina, also as a result of insolation, and, in the paroxysmal cases, as a result of exposure to cold, and occasionally after muscular exertion. Hæmoglobinuria has also been described as a conspicuous symptom of a disease attacking newly born infants. In the outbreak described by Winckel of Dresden, in 1879, twenty-three cases occurred in quick succession, and of these nineteen proved fatal. This was evidently due to the action of a specific poison existing in the Maternity Hospital at the time. Cohnheim also showed that hæmoglobinuria may occur as a result of severe, but not speedily fatal, burns. Transfusion of the blood of one kind of animal into the veins of another suffices to produce it. Moreover, many poisons have been found to induce the condition. By far the most important of these is chlorate of potassium. In the case of children, one to two drachms in the twenty-four hours is a dangerous quantity; in the adult, three drachms or upwards. Next to it, but at a long interval, rank carbolic, hydrochloric, sulphuric, and pyrogallic acids, and the inhalation of arseniuretted hydrogen gas. In what way we are to explain the action of these various substances upon the blood-discs is as yet far from clear, but the fact that they do so act is fully established.

MORBID ANATOMY.—The kidneys are of a deep chocolate colour throughout, with radiating striae of a darker tint. The sections show under a low power dark lines corresponding to the tubules, and spots marking the Malpighian bodies. With a higher power one makes out that the tubules are blocked with granular pigment, and that this is discharge from the Malpighian tufts. There are sometimes alterations in the spleen and in the bones, and hæmorrhages into various tissues.

SYMPTOMS.—The chief clinical features of hæmoglobinuria are those referable to the urine. It is dark like porter, or like the sediment of port wine; sometimes brown like coffee, or almost black. This colour is due mainly to blood-pigment, and usually few, if any, blood-corpuscles are present. Pigmented tube-casts and oxalate-of-lime crystals are often present in large numbers. The urine yields

a coagulum on heating, or on the addition of nitric acid. Examined spectroscopically, it shows the characteristic absorption-bands of oxyhæmoglobin and methæmoglobin. Otherwise the patient shows tokens of general discomfort : gastro-intestinal disturbance ; jaundice more or less pronounced ; sometimes enlargement of the spleen, and subcutaneous hæmorrhages ; weakness of the heart ; and tendency to hypostatic congestion in cases of a bad type, with other symptoms of depression or even collapse.

DIAGNOSIS.—The different forms of hæmoglobinuria have to be diagnosed from each other, and from certain diseases which resemble the paroxysmal forms. See HÆMOGLOBINURIA, PAROXYSMAL ; and BLACKWATER FEVER.

PROGNOSIS.—Cases of this kind rarely terminate unfavourably except when hæmoglobinuria is merely one of many symptoms in connection with blood-poisoning.

T. GRAINGER STEWART.

A. E. GARROD.

HÆMOGLOBINURIA, PAROXYSMAL. —

SYNON. : Paroxysmal Hæmatinuria.

DEFINITION.—A paroxysmal affection of the system ; manifesting itself by changes in the urine, and particularly the discharge of hæmoglobin ; caused sometimes by syphilis or malaria, and sometimes by other conditions not yet determined ; unassociated with any anatomical change as yet recognised ; and characterised by the occasional occurrence of constitutional disturbance, with discharge of dark, blood-stained urine.

ÆTIOLOGY.—The more recent researches point to syphilis as the most important of the extrinsic causes of the disease, and assign to malaria a less important share in its causation than it was at one time supposed to have. By far the most important cause of the paroxysm is exposure to cold : in a few instances the attacks have followed exertion. Among intrinsic causes, sex is evidently important, for the disease is almost confined to males. It may occur in children, and is apt to persist during a period of years.

As in other forms of hæmoglobinuria, the symptom is traceable to disintegration of red corpuscles within the circulating fluid, but the manner in which this is brought about still remains very obscure. That such disintegration may occur locally has been proved experimentally by Ehrlich. He tied a ligature round the finger of a patient who was liable to the disease, and then chilled the congested part by immersing it for a quarter of an hour in ice-cold water, afterwards transferring it for a like period to tepid water. In this patient it was found that many corpuscles were broken down, while no such effect was produced elsewhere in the body or in healthy people. Chvostek has shown that the ligature alone suffices to bring about this result, and also that cold alone does not cause an undue hæmolysis in the blood of such patients outside the body. On the other hand their blood is disintegrated with undue ease by mechanical influences such as shaking in a test-tube and also by the action of carbon dioxide. Murri and others regard the vaso-motor disturbances which accompany the attack as playing an equally important part with the abnormal blood-condition in causing the hæmoglobinuria.

The attack is attended by a conspicuous fall in the number of red corpuscles, but the loss is quickly repaired. The morphological blood-changes are not very characteristic ; there is an absence of the

tendency to rouleaux-formation, and some observers lay stress upon the presence of decolourised red corpuscles (*Blutschatten*) which they regard as a characteristic feature. During the attack the serum contains free hæmoglobin and may have a pink tint. The few recorded *post-mortem* examinations have not revealed any very striking changes, but the presence of some interstitial change in the kidneys suggests that the undue calls upon these organs during the paroxysms may not be quite free from ill effects upon them.

SYMPTOMS.—This form of hæmoglobinuria is paroxysmal, but not periodic. It may commence in childhood or during adult life. The recurrence of the attacks is determined by exposure to the exciting cause. The paroxysm may commence abruptly without any premonitory symptom, but is more commonly ushered in by a feeling of uneasiness in the loins and limbs, by shivering, and general chilliness. Sometimes it is preceded by furred tongue, and other symptoms of gastric catarrh ; and sometimes albuminuria precedes by a few hours the occurrence of hæmoglobinuria. Some degree of vaso-motor disturbance, evidenced by coldness of the extremities and often by 'deadness' and pallor of the fingers, usually accompanies the paroxysm, and the attack may be followed by a slight degree of jaundice. The more abrupt attacks frequently terminate by the discharge of the peculiar urine, the uneasy feelings having subsided before it occurs ; and the next urine is normal, or nearly so. In some cases albuminuria precedes or lingers for a time after the discolouration has passed off, and sometimes it replaces the hæmoglobinuria. After an initial fall during the exposure the temperature rapidly rises to 101° or 103° F. and as rapidly falls to normal again when the attack subsides.

The characters of the urine are very peculiar. Its colour is like porter, or like that of muddy port-wine ; its specific gravity ranges from 1015 to 1035 ; it is acid, or faintly alkaline ; it is highly albuminous ; sometimes it contains excess of urea. It throws down a copious sediment, which contains very few or no blood-corpuscles, but an immense amount of granular blood-pigment, with numerous tube-casts—hyaline or epithelial, often loaded or coated with amorphous granular matter, and with minute crystals of oxalate of lime. The colour is not due to blood-corpuscles, nor to hæmatin, but as a rule to a mixture of oxy- and methæmoglobin. The absorption-band in red of methæmoglobin is usually clearly visible when the urine is examined with the spectroscope (see SPECTROSCOPE IN MEDICINE: Coloured plate). In some cases the urine is less affected, being merely albuminous, and not depositing pigment.

A very interesting point in the clinical history of hæmoglobinuria is its relationship to Raynaud's disease. The relationship is neither constant nor exclusive, and the precise link which connects them is as yet not clearly determined. Still the clinical fact remains, that when the one disease exists the other is not infrequently present, and, as has been already mentioned, some vaso-motor spasm is present during most paroxysms of hæmoglobinuria.

DIAGNOSIS.—The only diseases with which intermittent hæmoglobinuria is likely to be confounded are hæmaturia, and renal calculus or gravel. From the former it is distinguished by the abundance of the blood-pigment and the extreme rarity of blood-

corpuscles; from the latter, by the short duration of the attacks, the presence of the characteristic deposit, with the fact that the pains affect both loins, not merely one. It is sometimes important to distinguish the milder forms, in which merely albuminuria occurs, from congestion or from commencing inflammatory Bright's disease. It is not always possible to distinguish these during the early hours of the attack; but the amount of general disturbance, the state of the tongue, the slight jaundice, the suddenness of the onset, and the absence of dropsy, generally suffice to make the diagnosis clear.

PROGNOSIS.—The prognosis is good in paroxysmal hæmoglobinuria, as to the individual paroxysm. The tendency to the disease is also not infrequently got rid of. It has not proved fatal in any case.

TREATMENT.—With a view to preventing the attacks, individuals who are subject to paroxysmal hæmoglobinuria should carefully avoid exposure to cold and over-fatigue of every kind. They should take abundance of good and easily digested food, should avoid free use of alcohol, and should clothe warmly. As the paroxysm is spontaneously recovered from, little need be done, excepting with the view of alleviating the discomfort of the patient. Chvostek found that nitrite of amyl administered at an early stage tended to cut short or avert the paroxysm, but Mannaberg and Donath failed to obtain this result. The patient should go to bed and be kept warm, and have abundance of warm drinks. In respect of diminishing or removing the tendency to the malady, various remedies have been found useful, among which may be mentioned quinine, tincture of cinchona, iron, arsenic, and chloride of ammonium. Antisyphilitic treatment by means of mercury has also been found useful, and has even brought about a cure in some cases.

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HÆMOGLOBINURIC FEVER.—See BLACK-WATER FEVER.

HÆMO-PERICARDIUM (αἷμα, blood; περί, about; and καρδιά, the heart).—An extravasation of blood into the sac of the pericardium. See PERICARDIUM, Diseases of.

HÆMOPHILIA (αἷμα, blood; and φίλος, predisposition for).—SYNON.: Hæmorrhagic Diathesis; Bleeders; Fr. *Hémophilie*; Ger. *Bluterkrankheit*.

DEFINITION.—A congenital disease, often hereditary, characterised by a tendency to immoderate bleedings, whether spontaneous or traumatic, and to obstinate swellings of the joints.

ÆTIOLOGY.—Men are far more liable than women to this disease, the proportion being about eleven to one. Women who suffer from hæmophilia show much less typical specimens of the disease than men, and rarely die from hæmorrhage, although floodings and profuse menstruation are common.

The best-ascertained cause of hæmophilia is hereditary predisposition. No other cause is known with anything like certainty. The origin of the disease *de novo* has not been definitely proved. In a family of bleeders, the disease descends as a rule to the boys through the mothers, the women remaining quite healthy and apparently free from all disease. In this way it may persist through seven generations,

as in the Appleton-Swayne family. The fathers do not seem to transmit the disease to their sons: at least, instances of this are rare. The women of bleeder families are remarkably fertile. Some have thought hæmophilia to be more common in Germany, but this is probably owing to the greater attention paid to the disease in that country. Cases have been met with in the Indian Archipelago, North America, the Scandinavian kingdoms, and elsewhere. The disease is not limited to the Aryan races, as the Jews, a Semitic people, are singularly liable to it.

ANATOMICAL CHARACTERS.—No morbid appearances have yet been found after death with any constancy. The blood-vessels are apparently unaltered. The condition of the blood has not yet been properly investigated. The statement that the blood-platelets are diminished in number or altogether absent also lacks confirmation. It has been suggested that there is a marked increase in the mass of the blood, and it has been repeatedly found that there is an increase in the number of red blood-corpuscles. It would be important to ascertain whether the number of white corpuscles is increased or diminished, and whether they are altered in character. The swellings of the joints appear in some cases to be due to the extravasation of blood within the articulation. At other times it may apparently represent an irritation of the bone-marrow.

SYMPTOMS.—The first signs of hæmophilia are commonly seen during the first year of life; but sometimes they are delayed until the beginning of the second dentition. Cases on record of a much later appearance of the first symptoms are not trustworthy. It is very rare for bleedings to be noticed at birth. There is nothing about the subjects of hæmophilia, when not suffering from bleeding, to distinguish them from ordinary persons. They look well; and nothing amiss can be discovered by physical examination in the chest or abdomen.

There are three well-marked *degrees* of hæmophilia. The first is the most typical and characteristic, in which there is a tendency to every kind of hæmorrhage, traumatic or spontaneous, interstitial or superficial. The swelling of the joints is well-marked. This degree is scarcely ever seen in women; but it is the most common among men. In the second degree, spontaneous hæmorrhages from the mucous membranes only are present. The third degree, in which the tendency is little marked, is seen only among the women of bleeder families; and shows itself only by spontaneous ecchymoses.

Hæmorrhage.—Spontaneous bleedings are sometimes preceded by symptoms of unusual fulness and plethora. The mucous membranes supply the blood in this case; in childhood bleeding from the nose is the most common, and also the most fatal, although bleedings from the bowel, mouth, or chest may also occur. There has been only one known instance of death from hæmaturia. The traumatic bleedings vary much in intensity, even in the same individual. Death has followed division of the frenum of the tongue, vaccination, leeching, and the extraction of a tooth. This last is a very common cause of death. If abscesses be opened, furious bleeding commonly takes place; and the same occurs if a blood-tumour or extravasation of blood be interfered with.

The bleeding is nearly always capillary, and may kill in a few hours or after some weeks. The

quantity of blood lost is sometimes enormous. After the bleeding the patients are extremely anæmic; and this state may last for months.

Besides superficial bleedings, interstitial hæmorrhages, ecchymoses, and blood-tumours may be observed, whether spontaneous or traumatic. A bruise which a healthy person would not feel may fill the connective tissue of a limb with blood; or the bleeding may be circumscribed, and form a tumour instead.

Swelling of the joints.—This chiefly affects the larger joints, the knee being most commonly attacked. The pain may pass from one joint to another and finally settle in the knee. The joint, most commonly after some injury, becomes swollen and painful, and apparently filled with fluid; there is fever; and this state may last for many weeks. Relapses are very apt to occur during convalescence.

A case of hæmophilia of one kidney has been reported, and the removal of the affected organ led to cure (Senator). As a rule the disease lasts throughout life, but may become less marked with advancing years. Some bleeders may reach old age and ultimately die of disease unconnected with their hæmorrhagic diathesis.

DIAGNOSIS.—The diagnosis of hæmophilia is often easy. If from early infancy a boy have suffered repeatedly from abundant bleeding—especially traumatic—and from joint-affections, there can be no doubt of the diagnosis. It is made more certain by the existence of hereditary predisposition. In women the diagnosis must be made with more care, as they are subject to hæmorrhagic disorder which first appears about puberty, but which is not hereditary. See MENSES, Disorders of.

PROGNOSIS.—The prognosis is not so serious with respect to life as was formerly thought.

TREATMENT.—The treatment of hæmophilia includes almost the whole subject of the treatment of hæmorrhage. Surgical interference in the way of the ligature of arteries is, however, contra-indicated in connection with hæmophilia, as the hæmorrhage in such cases is almost exclusively capillary. For the same reason, the administration of ergot, with a view to bringing about a contraction of the arterioles, would not be likely to prove of service. Treatment ought to be directed entirely to bringing about an increase (*a*) in the rapidity of coagulation, and (*b*) in the firmness of the resulting clot. In our present ignorance on the subject of the pathology of hæmophilia, treatment must take cognisance of every factor which is known to influence the coagulability of the blood. The facts that have been ascertained in connection with this subject are as follows: A defect in coagulability may depend on one or other of the following conditions:—

A Defect in the Inorganic Substratum of Fibrin.—This may consist in a diminution of the amount of available calcium-salts in the blood. Defective coagulation arising from this cause is to be met by the administration of lime-salts, preferably calcium chloride (grs. x–xx). Owing to the disagreeable taste and deliquescent nature of the salt, it is best administered in the form of a varnished pill or an enema. A one-per-cent. solution of calcium chloride may also be applied as a local styptic. Stronger solutions are not to be recommended, as coagulation is retarded after a certain percentage of calcium has been exceeded. In view of the fact that the action of the lime-salt is not instantaneous, the solution must be applied on a tampon

in such a manner as to hinder the flowing away of the blood with which it comes in contact. In all cases the clots obtained by the addition of lime to the blood are characterised by an extraordinary firmness. The coagulative efficacy of a solution of lime-salts can be increased by the addition of fibrin-ferment. This last can be obtained by extracting washed fibrin with water.

A Defect in the Organic Substratum of Fibrin.—There is little doubt that the coagulation of the blood is brought about by the combination of an element of plasma-fibrinogen with a fibrinoplastic element derived from the breaking down of the white corpuscles. This fibrinoplastic element consists of the substance which is known as Woolridge's tissue- or cell-fibrinogen. A diminution of the available tissue-fibrinogen in the blood leads to a condition of diminished coagulability. Its addition to the blood in all cases is followed by an immediate increase of coagulability, and an increased formation of fibrin. In view of this fact a local application of tissue-fibrinogen would appear to be indicated when hæmorrhage in hæmophilia does not yield to the application of lime, or lime and fibrin-ferment. Tissue-fibrinogen may be obtained in the following manner: Procure the thymus gland (the chest-sweetbread) or testicle of a calf, lamb, or other animal. Mince it fine in a mincing-machine, and extract with, say, twenty times its volume of water for ten minutes to twenty-four hours. Strain through fine muslin or through flannel, and add to each pint of the strained infusion 1 ounce of diluted or 1 fluid drachm of strong acetic acid (B.P.). A precipitate of tissue-fibrinogen forms almost immediately. Allow it to settle, and get rid of the supernatant fluid by decantation, or, better, collect the precipitate on a filter-paper. In either case dissolve the tissue-fibrinogen thus obtained by the addition of a few drops of alkali (dilute carbonate or bicarbonate of sodium). Apply the somewhat viscid solution on a tampon of cotton-wool to the bleeding point. Escharotics such as ferric chloride are seldom found to be of any service in bleeding in hæmophilia.

In cases of severe hæmorrhage, transfusion may be necessary. See SALINE SOLUTION, INFUSION OF; and TRANSFUSION.

GENERAL TREATMENT OF THE MORBID CONDITION.—It is impossible in our present ignorance of the pathology of hæmophilia to lay down a scientific method of treatment. It will, however, be evident that any treatment directed towards rendering the blood more coagulable would be indicated. Thus the administration of lime-salts might prove useful. Further, the means which are at our disposal for increasing the number of white corpuscles in the blood might be employed, with a view of increasing the amount of the fibrinoplastic element, for we have seen that tissue- or cell-fibrinogen is contained in the white corpuscles. While awaiting further information on this subject, the following therapeutic agents might be brought into requisition: (*a*) The administration of aromatic substances, such as camphor and turpentine. The administration of these substances increases the number of white corpuscles in the blood (Binz), and they are employed apparently with success in the treatment of internal hæmorrhages. (*b*) The administration of pilocarpine (Horbaczewski), which increases the number of circulating white corpuscles. (*c*) The administration of nuclein (Horbaczewski),

or nuclein-containing substances such as tissue-fibrinogen (Wright), for these substances increase the number of white corpuscles in the blood. They are available in the form of yeast, sweetbreads, lamb's fry, and kidneys. It has, however, not yet been ascertained whether these cell-substances lose their efficacy on boiling. The subcutaneous inoculation of 100–200 cubic centimetres of a sterilised 1–2½-per-cent. solution of gelatine (of the quality used for bacteriological purposes) has recently been favourably reported on.

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HÆMOPTYSIS (αἷμα, blood; and πτύω, I spit).—SYNON.: Fr. *Hémoptysie*; Ger. *Bluthusten*. Hæmorrhage from the larynx, trachea or lungs. See HÆMORRHAGE.

HÆMORRHAGE (αἷμα, blood; and ῥήγνυμι, I burst forth).—SYNON.: Fr. *Hémorrhagie*; Ger. *Blutfluss*.

DEFINITION.—The escape of blood from any part usually containing it.

This article will deal generally with those cases of hæmorrhage which result in the expulsion of blood from one of the natural passages opening on the outside of the body, viz. (1) Nose (see EPISTAXIS), (2) Mouth (Hæmoptysis and Hæmatemesis), (3) Ear (Otorrhagia), (4) Anus (see INTESTINES, HÆMORRHAGE from; and MELÆNA), (5) Urethra (Hæmaturia), (6) Vagina (see METRORRHAGIA). Hæmoptysis, Hæmatemesis, and Hæmaturia are considered in detail at the end of the present article.

ÆTIOLOGY.—The causes of hæmorrhage thus defined may be placed in three groups.—1. *General causes.*—a. Uniformly distributed disease of the vessels or defects in the composition of the blood may lead to hæmorrhage. This group includes *leucocythemia*, *pernicious anemia*, *hemophilia*, *scurvy*, and *purpura*. These diseases may give rise to hæmorrhage from any of the sites just mentioned. To these may be added *yellow fever* and *acute yellow atrophy of the liver*, which may cause hæmorrhage into the cavity of the stomach and consequently hæmatemesis.

b. Sudden and extreme increase in the venous pressure may cause rupture of indifferently supported vessels. In this way epistaxis or otorrhagia may be caused by a paroxysm of *whooping cough*.

2. *Local causes.*—a. Injury of the vessel or vessels from which the hæmorrhage occurs is often the responsible cause. Thus, a *broken rib* may give rise to hæmoptysis from damage to the vessels of the subjacent lung; a *calculus* in the pelvis of the kidney may cause hæmaturia; and a *swallowed fish-bone* or *corrosive poison* may produce hæmatemesis.

b. Disease of the vessel-wall may so weaken it that it gives way under the ordinary blood-pressure. Among the diseases weakening the walls of the vessels, *tubercular arteritis* stands first as the principal cause of hæmoptysis in phthisis. Necrosis of tissue depending on the direct action of other bacterial products, or on vascular occlusion—conditions occurring in *suppuration*, *syphilis*, and *enteric fever*—may also lead to destruction of part of a vessel-wall before the lumen is closed by thrombosis. *Local arterio-sclerosis* may lead to rupture of the aorta, and the invasion of the lung by a *new-growth* may cause hæmoptysis from rupture of the vessels of the

tumour. c. Increased local intra-vascular pressure may lead to the rupture of the affected vessel. Thus, the increased pressure in the portal vein in cases of portal *cirrhosis* of the liver may cause copious hæmorrhage from œsophageal varices, themselves a result of the earlier stages of the disease, and so give rise to hæmatemesis. The engorgement of the pulmonary vessels in *mitral stenosis* is frequently attended by hæmoptysis.

3. *Miscellaneous causes.*—It is necessary to mention the possibilities of *feigned hæmorrhage* as well as the so-called *vicarious hæmorrhage* believed by some to take the place of arrested menstruation. Moreover, blood may be subsequently mixed with a discharge and give rise to erroneous conclusions, as in the case of hæmaturia due to admixture with blood from the vagina. See LIFE ASSURANCE.

CHARACTERS.—The appearance of the blood will vary with the amount and source of the hæmorrhage, with the time which has elapsed since its escape from the vessels, with the place in which it may have been retained, and with the substances intermixed with it. In general terms, blood from an artery is bright, and blood from a vein dark; but this distinction is often of little value, since blood exposed to air will soon become bright red, whatever its source. A copious hæmorrhage from a small aneurysm in a tuberculous lung will present precisely the same appearance as a similar amount of blood from the dilated veins at the lower end of the œsophagus in a case of cirrhosis of the liver. The blood from a small hæmorrhage is more likely to be retained within the cavities of the body, and therefore to show signs of decomposition when expelled. Consequently, large hæmorrhages present the greatest difficulties in diagnosis, so far as the appearance of the blood is concerned; and blood altered so as almost to resemble coffee-grounds is generally derived from some part in which it can be easily retained, and in which chemical changes readily occur, e.g. from the stomach rather than from the lung. For similar reasons a small amount of bright blood passed *per anum* is generally derived from the lower end of the rectum, and a large amount of dark tarry blood from the stomach or duodenum. The substances intermingled with the blood will often afford assistance in the diagnosis of its origin. Recognisable particles of food are generally derived from the stomach, though if a meal have been taken a short time previously, a few fragments might have collected in, and be ejected from, the mouth. Bronchitic or phthisical sputa mixed with blood expectorated the day after a hæmorrhage afford certain evidence of its origin.

DIAGNOSIS.—The diagnosis of the exact site of the hæmorrhage depends upon the consideration of four items of evidence. 1. *The history of the patient.*—This may reveal the occurrence of injury or the existence of disease sufficient to give rise to the hæmorrhage. 2. *The phenomena attending the expulsion of the blood.*—Coughing or vomiting will at once suggest the origin of the hæmorrhage, though vomited blood, unless in large quantity, may have been previously swallowed. 3. *The characters of the blood.*—These have already been considered. 4. *The physical examination of the patient.*—This is only permissible to a very limited extent, but mere observation, palpation, and auscultation may suffice to discover a thoracic aneurysm, a tuberculous lung, or a cirrhotic liver.

EFFECTS.—A small hæmorrhage from the nose,

from the rectum, or even from the lung may be followed by a sense of relief and give rise to no unpleasant effects. The rapid loss of a large quantity of blood is attended by pallor, sudden fall of temperature, collapse, rapid breathing, syncope, and, just before death, convulsions. A second large hæmorrhage within twenty-four hours is generally fatal. At the end of this time regeneration of the blood proceeds rapidly, and the danger, therefore, speedily diminishes. Repeated hæmorrhages may lead to persistent anæmia. See BLOOD, Morbid Conditions of.

If the hæmorrhage depend on any of the general causes, on local increase of the blood-pressure, or on injuries, it will tend to cease gradually as the intravascular pressure diminishes. If, however, it depend on the rupture of a softened part of an arterial wall, or on an ulcerated opening in a vein, it will often increase, continue, or recur at short intervals.

TREATMENT.—Healing takes place primarily by thrombosis, and later on by proliferative inflammation of the affected vessel. To produce this, absolute rest—physical, emotional, and mental—and a low blood-pressure are essential. The faintness following a hæmorrhage is itself beneficial, and stimulants should never be given with a view to removing it, although the patient may thereby be temporarily gratified. The posture to be recommended will vary with the seat of the hæmorrhage, but the administration of morphine is a safe procedure in nearly all cases of severe hæmorrhage. Styptics may be employed if there is any probability of the drug reaching the affected part. Ergot and its derivatives should never be given if the hæmorrhage is due, as it usually is, to diseased vessels or increased intravascular pressure, but may be useful in capillary hæmorrhage or when a muscular organ, like the uterus, is the seat of the hæmorrhage. It is sometimes necessary to adopt measures calculated to increase the coagulability of the blood. See HÆMOPHILIA.

Hæmatemesis.—**SOURCES.**—Vomited blood may be derived from vessels in the œsophagus, stomach, or duodenum; or from the rupture of an aneurysm through the wall of one of these organs.

ÆTIOLOGY. 1. *General.*—Any of the *hæmorrhagic diseases of the blood* enumerated above may give rise to hæmatemesis, copious or slight, but no general increase in the venous pressure, however sudden, is likely to cause it.

2. *Local.*—Injury of the vessels may be due to penetrating *wounds* from the outside or inside (fish-bone), or *corrosive fluids*. Blood derived from these sources is small in amount and dark in colour. *Prolonged retching* may cause rupture of a small vessel and give rise to the vomiting of a small amount of bright blood. An *aneurysm* of the abdominal aorta or of one of its upper branches may occasionally rupture into the stomach, and if this occur the vomited blood will be abundant and bright. Among the diseases of the vessel-walls, *acute ulceration* of stomach or duodenum must be placed first. A branch of the splenic, coronary, or superior pyloric artery may be opened, and the resulting hæmorrhage be proportionately severe; in such case the blood is bright and sometimes frothy. Occasionally in acute ulceration and almost invariably in chronic ulceration or in the ulceration of a *malignant growth* only small vessels are affected

and but little blood is effused, and this is not ejected until it has undergone considerable change and become of coffee-ground appearance. The hæmatemesis so frequently complicating portal cirrhosis of the liver is generally copious, and the blood bright, though derived from the ulceration and rupture of the dilated venous anastomoses at the lower end of the œsophagus, and often passing into the stomach before being expelled from the mouth. If blood, escaping into the œsophagus, either from ruptured varicose veins or from an aneurysm of the descending thoracic aorta, be expelled at once, it is voided easily without cough or vomiting.

3. *Accidental.*—Blood derived from the nose, gums, larynx, lungs, and elsewhere may be swallowed and subsequently vomited. It is then more or less discoloured and rarely exceeds a few ounces.

DIAGNOSIS.—When the blood is much altered by the action of the other gastric contents, the appearance it presents may be closely simulated by food, bile, medicines containing iron, and red wine, if these have been detained in the stomach for a few hours. In doubtful cases a filtered solution of the vomit in caustic potash should be prepared and examined by means of a pocket-spectroscope (see SPECTROSCOPE IN MEDICINE); or the *hæmin-test* may be employed (p. 622). The history of the case and the reddish-brown colour of the vomit are, however, in most cases, a sufficient guide. Cough may accompany hæmatemesis.

PROGNOSIS.—First attacks of hæmatemesis are not generally fatal unless due to rupture of an aneurysm, or to implication of the aorta or of any large arterial trunks supplying the stomach. As the exact local condition in ulceration can never be ascertained no definite prognosis can ever be given. The recurrence of hæmorrhage very seriously affects the prognosis.

TREATMENT.—If the hæmorrhage is severe, half a grain of morphine should be given by hypodermic injection; and absolute rest in the recumbent posture be maintained for six hours, no movement of any kind being permitted. At the end of that time rectal feeding, by suppositories, may be commenced, but nothing, not even ice, must be given by the mouth; although, to allay thirst, the tongue may be painted with lemon-juice and a little glycerine, and a few ounces of warm water may be injected into the rectum. If the collapse is serious, ammonia may be gently sniffed as a stimulant, or half an ounce of brandy given *per rectum*. Styptics are of doubtful benefit. Fifteen-grain doses of gallic acid or 4 grains of the *Pilula Plumbi* c. Opio may be given by the mouth. When the hæmorrhage clearly depends on cirrhosis of the liver, styptics are of most use, while morphine is often unnecessary. In these cases calomel followed by a saline purge should be given after an interval of two days. Cautious feeding by the mouth may be recommenced at the end of three days, except in acute ulceration, when it should be deferred until ten days have elapsed.

Hæmoptysis.—**SOURCES.**—The laryngeal, bronchial, and pulmonary blood-vessels, or an aneurysm bursting into the air-passages from without.

CAUSES.—1. *General.*—Hæmoptysis from blood-diseases is usually slight, the blood being generally derived from the mucous membrane of the larynx, trachea, or bronchial tubes.

2. *Local*.—Blood escaping from the lungs is chiefly due to local changes. *Injury*, such as a broken rib pushed inwards, may damage the vessels of the lung and give rise to hæmoptysis without producing pneumothorax or hæmorthorax. Inhaled foreign bodies, especially if provided with sharp edges or points, may damage the mucous membrane of the air-passages and cause hæmoptysis. An aneurysm of the transverse or descending aorta may burst into the air-passages and cause fatal hæmorrhage, often preceded by slight hæmoptysis for a few days.

Disease of the vessels leading to rupture is usually tubercular. (1) Tubercular arteritis may affect the smaller branches of the bronchial or pulmonary arteries and cause such weakening of the walls that the ordinary pressure, especially during coughing, suffices to cause rupture of the diseased vessel. The lumen of the vessel is often much diminished before rupture occurs, thrombosis readily follows the local fall in blood-pressure, and consequently the hæmorrhage is often slight. This is the chief cause of hæmorrhage in early phthisis. (2) In the necrosis and caseation of tissue which leads to the formation of a tubercular cavity, two changes leading to hæmorrhage may affect the principal vessels traversing the affected area. (a) The cavity may arise in the immediate neighbourhood of an arterial branch of medium size, and the wall may be inflamed and weakened at the outset, dilating under the intra-vascular pressure so as to form an aneurysm, the walls of which are in immediate contact with those of the cavity, which it fills. Sooner or later, as the cavity enlarges, the wall of the aneurysm will give way, and the blood be ultimately discharged through any connection that may exist between the cavity and the air-passages. This is the principal cause of severe hæmoptysis in late phthisis. (b) In a few cases the walls of large arterial trunks traversing a large chronic cavity may undergo ulceration and rupture before their lumen is entirely obliterated. In such cases severe hæmorrhage may follow. Laryngeal hæmorrhage is generally due to ulceration—acute or malignant. In both cases it may be slight or severe. Hæmorrhage is very rare in tubercular ulceration of the larynx. In this group must be placed the hæmoptysis—slight in the early stages, severe and often fatal in the later—of malignant growths arising in or invading the lung; also the hæmorrhage due to the presence of a hydatid cyst, the *Distoma Ringeri*, bronchiectatic ulceration, and gangrene; for although hæmoptysis may be the first sign of gangrene, it generally occurs during the later stages and is then not infrequently fatal.

Increased intra-vascular pressure.—This may co-exist with any of the preceding conditions and facilitate rupture of the affected vessels. In most cases, however, it acts upon vessels uninjured save by the interference with their nutrition dependent upon passive congestion. This is the principal cause of the slight hæmoptysis which may follow severe coughing in bronchitis, and of the attacks of more severe bleeding that rarely complicate asthma; but it is best illustrated by the more copious hæmorrhage liable to occur in mitral stenosis. In this disease the vessels of the lungs are not infrequently permanently over-distended and the circulation slowed, so that thrombosis and infarction are common occurrences (see LUNG, Hæmorrhage into). Local variations in blood-pressure are thus easily induced, rupture of vessels is frequent, and hæmo-

pysis results. In hæmoptysis of moderate severity in young persons the diagnosis generally lies between tubercular arteritis and the increased intra-venous pressure of mitral stenosis.

EFFECTS.—The immediate effects will vary with the extent and recurrence of the hæmorrhage (p. 619). Suffocation may result from hæmorrhage into the air-passages: and even if this be averted inhalation of the blood may be followed by a fatal attack of acute pneumonia, or possibly by a spread of tuberculosis.

DIAGNOSIS and PROGNOSIS.—These depend mainly upon considerations already mentioned (p. 619). The onset of fever in hæmoptysis adds much to the gravity of the prognosis.

TREATMENT.—If the hæmorrhage be small in amount, merely tinging the expectoration, a few days' rest should be enjoined, a light diet be ordered, and a mild aperient be given. As soon as the disease causing the hæmorrhage has been ascertained, measures for its relief must be undertaken. If a tubercular origin be suspected, though without clear evidence, open-air life in a dry sunny climate suited to the taste, physique, and means of the patient should be insisted upon, until, at any rate, the possible development of tubercular changes can be excluded.

If the hæmorrhage be sufficiently severe to give rise to symptoms, the patient must be kept absolutely still in bed with the shoulders well raised, and the body efficiently and comfortably supported. No movement should be permitted, and no conversation allowed. The patient should be reassured, but the visits of friends must be reduced to a minimum. Hot-bottles should be applied to the extremities, and a hypodermic injection of morphine (gr. $\frac{1}{2}$) should be given to aid the completeness of the rest and to prevent cough. A saline aperient should be given. For three to six hours no food should be allowed, although small pieces of ice may be placed in the mouth. Subsequently cold fluid food may be administered in small quantities every two hours. In the large majority of cases this is all the treatment that is necessary or indeed useful. If syncope be feared, the inhalation of ammonia is often beneficial, provided it does not excite cough; stimulants in small quantity may be needed. Styptics are useless, and ergot is harmful as it increases the blood-pressure without contracting the diseased vessel which is too damaged to react to its influence. The inhalation of turpentine and the application of an ice-bag over the chest are procedures sanctioned by usage but of no certain benefit; indeed the ice-bag is probably prejudicial, since it causes contraction of the uninjured parts of the lung. When the danger of recurring hæmorrhage has ceased the secondary anemia and the original disorder must be carefully treated.

Hæmaturia.—The term hæmaturia is used whenever red blood-corpuscles are found in the urine; if only hæmoglobin be present the term is not applicable. See HÆMOGLOBINURIA.

SOURCES.—Blood in the urine may be derived from the kidney or its pelvis, from the ureter, bladder, prostate, or urethra, or, in the case of the female, it may be due to the admixture of blood with the urine after the latter has left the urethra. Blood may also be mixed with the urine fraudulently.

1. *General Causes*.—The hæmorrhagic diseases previously mentioned only rarely cause hæmaturia.

When this does occur the blood is probably derived from the pelvis of the kidney or from the bladder. The toxic or malignant forms of the acute specific fevers may also give rise to a similar condition of the urine.

2. *Local Causes.*—*Renal Hæmaturia.*—When the blood is derived from the kidney it is intimately mixed with the urine. If the hæmorrhage is a large one the resulting blood may be indistinguishable from pure blood; if it is slight a dirty smoky tint is imparted to the urine, and in some instances a reddish-brown precipitate is deposited on standing. Blood-casts are most readily found when the hæmorrhage is slight. (a) Slight transitory renal hæmaturia may follow the administration of cantharides, turpentine, cubebæ, copaiba, potassium nitrate, quinine, phenol, and hydrochloric acid, if these are given in toxic doses. (b) In acute inflammatory diseases of the kidney a moderate amount of blood and numerous blood-casts are generally found at some stage of the disease. When the acute condition supervenes upon chronic Bright's disease other forms of casts will be found. (c) In cases of renal tumour and of tumour or calculus in the pelvis of the kidney, ruptured vessels may cause large amounts of blood to appear at irregular intervals, especially after slight injury. (d) In renal embolism sudden hæmaturia is common, but the quantity of blood is small and the condition soon disappears. (e) In renal tuberculosis there may be sufficient blood to produce smoky urine, but in the majority of cases it exists only as a microscopical deposit. (f) Parasites, such as the cystic stage of the *Tænia echinococcus* (hydatid), hydronephrosis, and traumatism are occasionally causes of renal hæmaturia. (g) Passive congestion from pressure on the renal veins is a rare but possible cause of hæmaturia.

Vesical Hæmaturia.—Blood derived from the mucous membrane of the bladder is less immediately mixed with the urine and is generally passed in largest quantity towards the end of micturition. Not infrequently it is passed as blood-clots. The commonest causes of vesical hæmaturia are villous tumours and calculus. In both these conditions the hæmorrhage is due to the mechanical rupture of small vessels. Rupture of varicose veins is a less frequent cause. Slighter degrees of bleeding may be due to acute cystitis, passive congestion, tubercular ulceration, or the presence of the *Bilharzia hæmatobia*. In the latter case, ova with lateral or terminal spike will be readily found in the deposit obtained from the urine. See BLADDER, Diseases of; and ENTOMOZOA.

Urethral hæmaturia.—When the blood is derived from the urethra it is always expelled at the beginning and occasionally also at the end of the act of micturition, is often clotted, and is never intimately mixed with the urine. Its chief causes are inflammation and ulceration, direct injury, or the effects of the passage of a calculus. See URETHRA, Diseases of.

Accidental hæmaturia.—This may occur during menstruation or from the admixture of any uterine or vaginal discharge containing blood. The blood is rarely intimately mixed with the urine, and when it is, is generally of a brighter colour, which arouses suspicion as to its origin. If malingering is suspected the urine should be passed under observation or withdrawn by a catheter.

RECOGNITION.—Whenever the urine contains a

large amount of blood, the appearance is sufficiently characteristic, but when the amount is only sufficient to impart a smoky tint to the urine, microscopical and chemical examination is essential.

I. The *microscope* furnishes the most satisfactory test, as it is the only means by which the presence of blood-corpuscles can be ascertained. The urine should be allowed to stand for some hours, or the deposit be obtained by means of a centrifuge. The corpuscles will be globular or crenated according to the specific gravity of the urine; but their colour is often so pale that it fails to characterise them. They may be distinguished from pus-corpuscles, vegetable spores, and fragments of dumb-bell crystals by characters which can readily be seen in the Plates illustrating the article on Urinary Deposits.

The chemical examination comprises three tests, of which the first, which is most generally employed, is least reliable.

II. This is known as Almen's or the *Guaiacum-test*. Two or three drops of a freshly prepared simple tincture of guaiacum are added to about a drachm of urine, and the mixture shaken. Upon this is then poured about half the quantity of a 15-per-cent. solution of peroxide of hydrogen in ether (ozonic ether). If blood be present, the lower portion of the ozonic ether gradually becomes blue. This reaction is given by saliva, iodide of potassium, and other substances, and is not therefore wholly trustworthy.

III. The *Hæmin-test* is more reliable. The urine should be rendered alkaline by the addition of liquor potassæ and then boiled. A portion of the reddish-brown deposit which forms (*Heller's Test*) is placed on a glass slide and dried. A drop of *glacial* acetic acid and a small crystal of common salt are then added; the slide is next briskly warmed over a spirit lamp, and then set aside to cool. On microscopical examination hæmin-crystals may be found.

IV. A *pocket-spectroscope* furnishes a readier and no less reliable test. The reddish-brown precipitate described in the last test is placed in a test-tube, and shaken up in a little warm water. On placing the tube between the prism and the daylight, the characteristic absorption-bands will be seen. See SPECTROSCOPE IN MEDICINE.

DIAGNOSIS.—This rarely depends upon the character of the urine, although the microscopical examination of the deposit may reveal important evidence. The quantity of blood contained in the urine, the degree of admixture, and the microscopical examination of the deposit may be sufficient to suggest the cause, but a consideration of the symptoms and a careful examination of the patient are essential in forming a reliable diagnosis.

TREATMENT.—In the majority of cases the hæmaturia needs no treatment, and in those cases in which a large amount of blood is lost, absolute rest in bed and the administration of aperients are the only measures to be resorted to apart from the treatment of the diseases primarily responsible. For the treatment of these see the various articles on the Kidney, Bladder, and Urethra.

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HÆMORRHAGIC (αἷμα, blood; and ῥήγνυμι, I burst forth).—Associated with hæmorrhage. The word is applied to certain inflammatory products or to effusions when they contain blood, as in *hæmorrhagic peritonitis*; and to varieties of certain diseases in

which extravasations or hæmorrhages from free surfaces occur, for example *hæmorrhagic small-pox*, *hæmorrhagic measles*, and *hæmorrhagic purpura*.

HÆMORRHAGIC DIATHESIS.—See HÆMO-PHILIA.

HÆMORRHOIDS (αἷμα, blood; and ῥέω, I flow).—SYNON.: Piles; Fr. *Hémorrhoides*; Ger. *Hämorrhoiden*.

DESCRIPTION.—The hæmorrhoidal vessels, and especially the veins distributed to the lower part of the rectum, are very liable to become dilated and varicose, giving rise to a disease termed *hæmorrhoids* or *piles*. When the plexus beneath the mucous membrane within the external sphincter is thus affected, the hæmorrhoids are said to be *internal*. When the veins beneath the integuments outside the muscle are enlarged, the hæmorrhoids are called *external*. Internal and external hæmorrhoids very frequently co-exist.

External Hæmorrhoids.—We may distinguish two kinds of external piles—(1) the *venous*; and (2) the *cutaneous*.

(1) The *venous tumour* consists of a softish elevation of the skin near the margin of the anus, of a rounded form, and a livid or slightly blue tinge. On cutting into it, we find it is formed by a dilated vein or veins, which after inflammation will be occupied by a dark-coloured coagulum.

(2) The *cutaneous excrescence* consists of a flattened prolongation of skin, due to hypertrophy of the cutaneous layers, and is commonly pedunculated. It is generally the result of the first form—a projecting skin-tag left after absorption of the coagulum having undergone some hypertrophy. Often there is only a single broad flat excrescence at the side of the anus, but sometimes there are two—one on each side; and occasionally there are several encircling the anus. They are apt to become inflamed and oedematous. Similar excrescences occur as the result of irritating discharges from the bowel, and are common in stricture and chronic ulceration of the rectum.

Internal Hæmorrhoids.—Internal piles may be divided into three classes—(1) the *venous*; (2) the *columnar*; (3) the *nevoid*.

(1) The *venous pile* resembles the external venous pile, except that it is covered by mucous membrane and is within the sphincter.

(2) The *columnar pile* consists essentially of hypertrophy of the folds of mucous membrane which surround the anal opening—the pillars of Glisson. They have a red, almost vermilion, colour, an elongated form, and contain within them one of the descending parallel branches of the superior hæmorrhoidal artery (Hamilton). This pile is very vascular, bleeds copiously, and is made up of hypertrophied submucous tissue with many dilated arteries and veins.

(3) The *nevoid pile* is bright red, spongy and villous, and has been well compared in aspect to a strawberry. In structure it resembles a capillary nævus. It may bleed almost continuously.

Internal piles seldom attract attention until they have become so developed as to protrude at the anus in defecation. They then exhibit a remarkable diversity of appearance, according to their number, size, and condition. The protrusion may consist of only one large pile, found usually toward the perinæum, especially in women. More commonly there

are three distinct prominent growths, differing in size, one at each side of the anus, and a third in front—the last, the perineal, being the largest. In old cases they may be more numerous—as many as four or five. The distinction between them is commonly well-marked, but not always, for the piles sometimes merge into each other, so that the protrusion forms nearly a circular prominence. The aspect of extruded piles depends much upon their condition, whether congested, inflamed, or constricted by the sphincter. In an inactive state, and in a relaxed condition of the sphincter, they form softish tumours of a granular appearance, presenting just at the orifice of the anus; but when protruded and congested, they constitute large tense tumid swellings, of a deep red colour and smooth surface, which readily bleed. When hæmorrhoids are of large size, the integuments at the margin of the anus become everted, and form a broad band girding the base of the tumours. The skin thus everted is liable to be mistaken for external piles, and to be excised in operations—an error very likely to be followed by serious contraction of the anus.

ÆTIOLOGY.—Hæmorrhoids are a disease of middle and advanced age. They rarely occur before puberty; but very few persons in after-life altogether escape them. All circumstances which determine blood to the rectum, or which impede its return from the pelvis, tend to produce this disease. There is in many persons a natural predisposition to the complaint, which may be hereditary. But a predisposition is most frequently acquired by sedentary habits, indulgences at table, and neglect of the bowels. Hæmorrhoids, though a common disease in both sexes, occur more frequently in males than in females. Few women bear children without becoming in some degree affected by them; but the urinary and genital disorders of the other sex, combined with freer habits of living, leading to congestion of the liver, are still more fertile sources of piles. It must be remembered that piles may be merely a symptom in certain cardiac, hepatic, or uterine diseases.

SYMPTOMS.—The symptoms produced both by external and internal piles vary greatly in different subjects and in different stages of the complaint.

External piles cause a feeling of heat and tingling at the anus. A costive motion is followed by a burning sensation, and the excrescence becomes swollen and tender on pressure, so as to render sitting uneasy. This congested state of the pile may pass off; or it may lead to inflammation accompanied with considerable enlargement of the hæmorrhoid, forming an oval tumour, red, tense, and extremely tender. In such case phlebitis has ensued, and the pile-cavity is occupied by a coagulum. The inflammation may subside or go on to suppuration. When the matter is discharged, the clot of blood escapes with it, the abscess closes, and the dilated vein is usually obliterated, the pile being reduced to a small flap of integument. External piles rarely give rise to bleeding.

Internal piles, when slight, may exist for years, causing little inconvenience besides slight bleeding after a costive motion; with occasionally a feeling of fulness, heat, and itching just within the anus. If only small, they protrude slightly with the mucous membrane in defecation, returning afterwards within the sphincter. When of large size, the piles always protrude at stool, and require to be replaced, the patient usually pushing them up with his fingers. In a lax state of the sphincters, and in a loose

hypertrophied condition of the mucous membrane from which they spring, hæmorrhoids come down, even when the patient stands or walks about, so as to prove exceedingly troublesome, and to interfere with his taking walking exercise. In consequence of the irritation from pressure and friction to which the protruding piles are liable, their mucous surface becomes tumid and abraded, and furnishes a free mucous discharge tinged with blood, which soils the linen. They are often so sore that the patient is obliged to keep the recumbent posture, the pressure in sitting causing more or less uneasiness.

Persons subject to piles frequently suffer no inconvenience from them until irritated by an unusually costive motion, or by a smart purgative; or when, under the excitement of alcohol, the growths become congested and inflamed, and cause spasm of the sphincter muscle. Then they have what is termed an 'attack of piles'—that is to say, they experience a sensation of heat, weight, and fulness just within the rectum, followed by considerable pain at stool, and sometimes irritation about the bladder. These symptoms, which are often attended with febrile disturbance, arise from inflammation and swelling of the piles, which afterwards subside, but seldom without leaving some enlargement of the growths. Anæmia may follow upon continued bleeding; and reflex pains in the back, groin, and genitals are often complained of.

Strangulation.—When internal piles of some size protrude at the anus and are not returned, they are liable to be constricted and strangulated by the external sphincter. The contracted muscle impedes the return of blood, and occasions inflammatory swelling of the piles, which may become strangulated and gangrenous. In this way hæmorrhoids of large size have been known to slough off, the patient being relieved of the annoying complaint by a sort of natural process. An occurrence of this kind is attended with a good deal of pain and suffering, but is free from danger. In general, the extremities only of one or two of the larger growths perish, and the patients, though experiencing relief, are by no means cured of the disease.

Hæmorrhage.—One of the most common symptoms of internal hæmorrhoids—indeed that from which the name of the complaint is derived—is hæmorrhage, which occurs when the bowels are evacuated. The bleeding varies greatly in amount. Sometimes the motions are merely tinged with a few drops of blood; in other instances the quantity lost is considerable, several ounces being voided at stool. The bleeding may be irregular, occurring only after costive motions, or in certain states of health; or it may take place daily, going on even within the bowel, and producing the usual symptoms of derangement from continued losses of blood. The character of the bleeding also varies with the structural character of the pile, being, however, usually venous. There are persons who are liable to discharges of blood from the hæmorrhoidal veins, either at regular periods, or when, from good living or want of exercise, some plethora is induced. In these cases from three to six ounces of blood, or even more, pass away at stool, following the evacuation; and the blood which is voided is of a dark colour and evidently venous. Such discharges must not be rashly interfered with. They relieve congestion of the liver and kidneys, help to ward off attacks of gout, and prevent fits of apoplexy; so that in certain of these plethoric persons they are

rightly regarded as safety-valves. The bleeding from internal piles is undoubtedly in some cases arterial. Sometimes the blood may be observed to escape from the pile in jets. This is not evidence that it is arterial, but may be due to a regurgitant stream of blood forced through a rent in a vein by the pressure of the abdominal muscles. That bleeding from piles is always good for the health is quite a mistaken notion; and it is important that the practitioner should distinguish the bleeding taking place as a consequence of local disease, from that which arises from plethora, congestion of internal organs, or such a disease as cirrhosis of the liver.

TREATMENT.—When piles are small and cause but little inconvenience, the treatment is very simple. Persons with this complaint should take stimulants in great moderation, if at all; and in most instances they would do well to abstain entirely from alcohol. Many individuals never suffer from piles except after taking a glass of spirit-and-water, or a few glasses of wine. Such persons should become water-drinkers. Active exercise in the open air should be taken daily; and the patient should avoid sitting too long at the desk, because it is by prolonged sedentary occupation and the neglect of the rules of health that hæmorrhoidal complaints are encouraged. Chairs with cane seats are to be recommended. The bowels must be carefully regulated, so as to avoid hard and costive motions, as well as too frequent action. Irritating the rectum by active and repeated purging is more hurtful even than constipation. In regulating the bowels, a simple enema of half a pint of cold water, injected after breakfast, answers most admirably, and is more efficacious than purgatives. If aperients be needed, preference may be given to confection of senna, cascara, or the compound liquorice powder. Carlsbad salts, or the foreign mineral waters—the Pullna, the Friedrichshall, or the Hunyadi János—taken in the morning fasting, answer well with many persons, and ensure a comfortable relief. The diet must be simple and moderate, and the habits of the patient regular. The relief afforded by this palliative treatment is often remarkable.

Ordinary bleeding may be met by injections of cold water, or by some astringent injection or ointment; among these may be named a solution of sulphate of iron (10 grs. to ℥j), or preparations of tannic acid or rhatany. When the bleeding is copious, injections are not so successful, and operative treatment often becomes necessary. As useful local applications may be mentioned, the ointment of galls and opium, a combination of morphine and tannic acid, an ointment containing menthol, and injections of hazeline, or the local application of hazeline after each motion.

External piles when large and troublesome, and internal piles when they protrude at stool, and are not easily reduced, or are subject to inflammation, ulceration, and frequent bleeding, can be removed only by operation. It is seldom desirable to operate upon piles developed during pregnancy, or upon hæmorrhoids occurring in the course of organic disease of the liver.

T. B. CURLING. FREDERICK TREVES.

HÆMOSIDERIN (αἷμα, blood; and σίδηρος, iron).—A generic name including all iron-containing pigments derived from hæmoglobin in the tissues. Hæmosiderin gives the ordinary reactions of iron,

and is found in any part where excessive destruction of blood-corpuscles has taken place. Like hæmatoidin, with which it is frequently mixed, it may exist in both granular and crystalline forms. See ANÆMIA, PERNICIOUS.

HÆMOSTATICS (αἷμα, blood; and στατός, stopped).

DEFINITION.—Internal remedies and local applications which arrest hæmorrhage.

ENUMERATION.—The chief hæmostatics are: Ligature; Pressure; Rest; Cold; Hot Water; the Actual Cautery; Astringents; the whole class of Styptic drugs; and Calcium Chloride and Nuclein, which have been shown to increase the coagulability of the blood. See HÆMOPHILIA.

ACTIONS and USES.—When taken in their widest sense, it is evident that hæmostatics must include all the various means which have been devised to stop bleeding. Externally we must vary our plan of treatment according to circumstances. No surgical principles are better founded than those which enjoin us to tie a wounded artery, and to apply pressure to a vein; and for the absolute arrest of hæmorrhage from any readily accessible part a most powerful aid has been provided in Esmarch's elastic bandage. When the bleeding depends on general capillary oozing, the application of ice may often prove effectual; and where this fails, recourse must be had to some of the numerous articles of the Pharmacopœia, already referred to, which possess styptic properties (see STYPTICS). An example of the successful application of a hæmostatic is the arrest of uterine hæmorrhage by means of injections of perchloride of iron, of hot water, or of ice-cold water. In the case of undue hæmorrhage from a leech-bite, if milder remedies, such as pressure, do not succeed, we may apply the solid nitrate of silver, or include the bleeding point in a loop of twisted suture.

Absolute rest is essential for the successful treatment of hæmorrhage; and the regulation of the diet and of the bowels is equally to be attended to. For the details of treatment in each particular form of hæmorrhage, the reader is referred to HÆMORRHAGE; HÆMOPHILIA; MELÆNA; and the diseases in which these conditions occur.

ROBERT FARQUHARSON.
SIDNEY PHILLIPS.

HÆMOTHORAX.—See PLEURA, Diseases of.

HAIR.—The diseases to which the hair is liable may be divided into two chief groups: those of nutrition and growth, including atrophic, hypertrophic, and pigmentary changes; and those associated with inflammation of the follicles, and the presence of parasites. The diseases comprised in the latter group will be found described under their respective heads, so that in this place we have to deal only with the first group.

DISEASES INVOLVING THE NUTRITION AND GROWTH OF THE HAIR.—Most of the alterations in the growth and colour of the hair are atrophic in their nature. The more common forms of atrophy produce various kinds of baldness; and in addition we have greyness or loss of the natural colour of the hair, and certain structural alterations, such as those met with in trichorrhæxis nodosa; these latter alterations, though not simple atrophies, yet belong to the atrophic group.

Alopecia or Baldness.—Baldness is of two kinds—*congenital* and *acquired*. *Congenital* alopecia is very rare; it is a malformation rather than a disease, and is generally associated with a defective development of the teeth. This condition is permanent.

Acquired baldness arises from many different causes. It may result (1) from *various forms of disease*; (2) from simple *defective nutrition and growth*; or (3) from *atrophic senile changes*.

(1) *Baldness from various forms of disease.*—Different diseases produce baldness in different ways, and they may be conveniently divided into four groups: acute febrile diseases; follicular inflammations; nervous affections of the skin; and parasitic diseases.

The baldness which follows *fevers* is always temporary, though often extreme. There is apparently an almost sudden death of the hair at the time of the fever, especially when there is long-continued high temperature, and this is followed by its falling off some two or three months afterwards. Parturition and some other illnesses besides fevers produce a similar result, though generally more slowly and persistently. The falling off of the hair which accompanies secondary syphilis often occurs without any visible eruption on the scalp.

Folliculitis, or inflammation of the hair-follicles, is one of the commonest causes of baldness. We meet with it in seborrhœa capitis, in sycosis, and in various pustular eruptions involving the hair-follicles. When produced in this way, alopecia may be temporary or permanent, according to the nature or the severity of the disease. All diseases of the skin which leave scars or obliterate the follicles lead to permanent alopecia of the parts affected. Erythematous lupus, scleroderma, and some forms of syphilis, are examples. It is especially the forms of alopecia resulting from folliculitis which are so much aggravated by the stimulating applications almost universally recommended by hairdressers, without regard to the cause of the malady.

The changes in the *nervous system* which produce baldness may be general, as in some cases of shock; or local, as in alopecia areata. Falling of the hair, which sometimes follows a blow or other injury to the nerves, belongs to this class. See ALOPECIA AREATA.

The *parasitic diseases* which lead to partial baldness are tinea tonsurans and favus, which are fully described under their proper headings.

(2) *Baldness from defective nutrition.*—Premature baldness from defective nutrition differs little from senile alopecia, and is often hereditary. The thinning of the hair in myxœdema is an example of this variety.

(3) *Senile Baldness.*—This differs from the preceding variety in degree only.

The treatment of this kind of alopecia, as might be expected, is not very satisfactory. Some good, however, may be done in the early stages by stimulating remedies. One of the best is a lotion consisting of vinegar of cantharides, ʒvj.; glycerin, ʒj.; spirit of rosemary, ʒij.; rectified spirit, ʒv. This should be dabbed on the scalp daily with a small sponge. Frequent and excessive sweating is certainly an exciting cause of premature baldness in those predisposed to the malady; hence its frequent occurrence in Europeans who live in tropical climates. See BALDNESS.

Pigmentary Changes.—Canities, or greyness, results from the deficient development of the natural pigment of the hair. It may be congenital or acquired. The extreme form of congenital canities is met with in albinos, in whom there is an absence of pigment in the skin and choroid as well as in the hair. In an irregular form it sometimes exists as a congenital white lock on the scalp or face, the surrounding hair being of a normal colour.

As an acquired alteration in the hair, canities is well known as a simple senile change. Premature greyness, not differing essentially from the senile change, is often met with in those suffering from any chronic disease, such as phthisis; it is also more or less hereditary. In all these cases the greyness is symmetrical. In an asymmetrical form it is met with in several diseases of the skin, especially in area, leucoderma, and chronic neuralgia of the scalp. The distribution of the grey hair and the history of the case will serve as a means of differential diagnosis.

In many cases of canities treatment is obviously useless. In those cases which admit of rational treatment, cantharides is by far the best local stimulant, as it has a tendency to excite the natural development of colouring matter, as may be often seen in the skin where a blister has been applied. Arsenic is usually the best form of tonic for the same reason.

Excessive growth.—Downy and almost invisible hairs may grow into strong and visible ones, but there is never any increase in their number; the great increase in their size sometimes leads to the erroneous belief that the actual number is greater than before. Sometimes the old hair is held in the follicle after the new hair has grown up, but this is not a true increase of numbers. An excessive growth of hair is not uncommon, and is a source of annoyance when it occurs on the face in women. *See* DEPILATORIES.

Structural changes.—*Trichoclasia*, or *trichorrhexis nodosa*, is the most important and best-known change in the structure of the hair. To the naked eye the hairs appear to be marked with two, three, or more small bulging spots, somewhat resembling nits. The hairs very easily break at these points, which, examined under the microscope, seem to consist of spindle-shaped swellings of the shaft of the hair. In a more advanced stage this swelling partially bursts near its most distended part, and finally the hair breaks with a ragged fracture, so that the divided hair has the appearance of two brushes, the bristles of which are interlocked. When the separation is complete, a stumpy hair is left with a frayed or brush-like free extremity. A variety of this disease is sometimes met with in which a smooth fracture takes place, not at the spindle-shaped swellings, but in the intermediate parts of the hair. *Trichorrhexis* is chiefly met with in the hairs of the face, especially those of the chin.

Lepothrix.—Although this curious affection is a concretion rather than a structural change of the hair, yet it has that appearance to the naked eye, and may be conveniently referred to under this head. The hairs affected with this disease are those of the axillæ and the scrotum. The concretions, seen under the microscope, have usually a rounded and nodulated form apparently surrounding the shaft of the hair, but not symmetrically. Sometimes the concretion extends continuously for some distance along the shaft of the hair, giving it a feather-like appear-

ance. The hairs themselves are abnormally brittle. These curious concretions are associated with the presence of a short bacillus which penetrates, and grows beneath, the cortical scales of the hair-shaft. The disease is very difficult to eradicate. Shaving and antiseptic applications are the two therapeutic agents generally employed.

ROBERT LIVEING.

HAIR-BALLS.—*See* CONCRETIONS.

HALL, in Austria.—Common salt waters, with iodine. *See* MINERAL WATERS.

HALLUCINATION (*hallucinator*, I blunder).—A false perception of an organ of sense, for which there is no external cause or origin (*see* ILLUSION); as when a man in total darkness thinks he sees an object. Hallucinations of all the senses occur, the most frequent being those of sight and hearing. They may be found in persons not insane, but indicate a disordered state of brain.

HAMMAM-MELOUAN, in Algiers.—Thermal muriated saline waters. *See* MINERAL WATERS.

HAMMAM-MESKOUTINE, in Algiers (the *Aque Tibilitine* of the Romans).—Highly thermal saline springs, weakly mineralised. *See* MINERAL WATERS.

HAMMAM-R'HIRA, in Algiers.—A winter resort for invalids. Highly thermal waters. *See* MINERAL WATERS.

HAMMERMAN'S PALSY.—*See* OCCUPATION-DISEASES.

HANGING, Death by.—Hanging is the effect of suspension of the body by the neck by means of a ligature or noose, the constricting force being the weight of the body, wholly or in part, or the weight multiplied by the distance through which the body falls. The mode of death varies according to these circumstances. With a long drop, the method now usually employed in judicial hanging, and particularly if the knot is under the chin, death is not infrequently due to fracture, or displacement, of the cervical vertebræ, and injury to the medulla oblongata. Death may also occur, without such anatomical lesion of the cervical vertebræ, from shock or syncope, or, as it is termed by Casper, neuro-paralysis.

When death does not occur in either of these ways, it is the result of asphyxia from occlusion of the air-passages, or rather of asphyxia in combination with coma, caused by compression of the cerebral blood-vessels. Though compression of the carotid arteries and jugular veins may be maintained for a considerable time without a fatal result, if the trachea is open below the point of constriction—whereas death speedily ensues if the air-passages are also occluded—yet death may result from the disturbance of the cerebral circulation alone; and the two causes operate conjointly in every case, in varying proportions.

PHENOMENA.—When death is not instantaneous, as in cases of injury to the medulla, or from neuro-paralysis, convulsive movements of the type seen in asphyxia may continue for some minutes after suspension, and the heart may continue to beat for a considerable period after all other vital movements have ceased.

Subjects who have been partially hanged have described various sensations, more or less pleasurable, similar to those of cerebral congestion and narcotic stupor.

POST-MORTEM APPEARANCES.—The appearances found after death by hanging are not uniform or constant; and there is no single sign invariably present diagnostic of this mode of death. Indicative of suspension, but not necessarily of death so caused, is the mark of the cord on the neck. Usually it is above the hyoid, passing obliquely upwards behind the ears, and losing itself in the occiput. But the position may vary according to the tightness of the noose before suspension, or the position of the head and direction of the pressure. It is generally single, but if the cord should have been twisted twice round the neck, two marks may be found, one circular and the other oblique. The characters of the mark differ somewhat according to the texture and thickness of the ligature. Usually it is a shallow groove or furrow, of a whitish or brownish hue and parchmenty consistence, occasionally abraded, rarely ecchymosed; but it may have livid edges or a chocolate tint. The appearances may vary in different parts of the same mark. The subcutaneous cellular tissue is compressed and silvery. Occasionally minute extravasations are seen in the deeper layers of the skin. The middle and internal coats of the carotids are sometimes lacerated; and where the momentum has been great, lacerations of the cervical muscles, hæmorrhage in their vicinity, fracture of the larynx, rupture of the thyro-hyoid ligaments and fracture of the hyoid bone, and fracture or dislocation of the cervical vertebræ, with injury to the medulla and effusion into the spinal canal, have been found. All the appearances usually found in the neck in cases of hanging may be produced by suspension of the dead body, especially if the legs are pulled forcibly downward.

The face is sometimes, but not commonly, distorted and expressive of suffering. Usually it is placid and pale, though if the body have hung for some time, it becomes very livid. The eyes are sometimes very prominent, and the pupils are usually dilated. Frothy mucus may be found at the mouth and nostrils. The tongue is pressed against the teeth and indented, or it may be clenched between the jaws. The base of the tongue is injected. The hands are often tightly clenched, the nails even being driven into the palms. Erection, or semi-erection, of the penis in men, with expulsion of semen or prostatic fluid, and vascular turgescence of the genitals in females, with sanguinolent effusion, are occasionally observed. Expulsion of the contents of the bladder and rectum is likewise common. The condition of the brain varies. Congestion of the meninges is sometimes pronounced, at other times not very marked. The mucous membrane of the larynx and trachea is congested, and mucous froth is present. The lungs are at times pale and distended; at other times collapsed. The condition of the heart and venous system characteristic of asphyxia is common. Marked redness of the mucous membrane of the stomach, simulating irritant poisoning, has been occasionally noted. The determination of the fact of death by hanging depends on a consideration of these various phenomena, and the absence of other causes of death.

ACCIDENT, SUICIDE, OR HOMICIDE?—Hanging is rarely homicidal. It signifies great disproportion of strength between the assailant and the victim;

and therefore, in the absence of this condition, there will be injuries indicative of a struggle. Apart from collateral circumstances, homicide can only be argued from the presence of such injuries as could not have been self-inflicted or caused accidentally during the act of suspension. Occasionally hanging is accidental, as in foolish experiments and insane imitation. It is not necessary that the body should be entirely off the ground to cause death by hanging. Many instances are recorded of suicide by hanging in most extraordinary positions calculated to throw the greater part of the body-weight on the noose.

TREATMENT.—This is rarely called for, except in accidents or attempted suicide. The body must be cut down, and artificial respiration employed. Venesection may be had recourse to for relieving cerebral congestion. *See* **ARTIFICIAL RESPIRATION**; and **RESUSCITATION**. **D. FERRIER.**

HANOT'S DISEASE.—*See* **LIVER**, Cirrhosis of (Hypertrophic Biliary Cirrhosis).

HARROGATE, in Yorkshire.—Saline, chalybeate, and sulphur waters. *See* **MINERAL WATERS**.

HASTINGS, on the South-East Coast of Sussex.—A mild climate. Mean winter-temperature, 40° F. Exposed to the east, but sheltered from the north. *See* **CLIMATE**, Treatment of Disease by.

HAT-MAKER'S DISEASE.—*See* **OCCUPATION-DISEASES**.

HAUT MAL (Fr.).—A synonym for epilepsy gravior. *See* **EPILEPSY**.

HAY-FEVER.—**SYNON.** : *Catarrhus astivus*; Bostock's Catarrh; Hay Asthma; Fr. *Asthme d'été*; Ger. *Frühsonnencatarrh*.

DEFINITION.—A catarrhal affection of the mucous membrane of the eyes, nose, mouth, pharynx, larynx, and bronchi, accompanied by dyspnoea; induced in persons predisposed to it by the action of the pollen of various plants, chiefly of the Graminae; prevalent during the hay-season, but subsiding at its close; and varying in severity according to certain atmospheric conditions, and the amount of pollen present in the air.

ÆTIOLOGY.—Hay-fever exists in Europe and North America, and it is especially common in England, where the number of cases is annually double that of any other country. The Anglo-Saxon race appears more liable to it than other races. It prevails more among men than women, probably because the former are most exposed to the atmosphere; and inhabitants of towns visiting the country are more liable to attack than the country-people themselves. J. N. Mackenzie of Baltimore assigns hay-fever to an exalted condition of the nasal erectile tissue, and especially the portion covering the posterior end of the inferior turbinated bone and the septum immediately opposite, rendering it more sensitive to the action of pollen and other irritants; and he maintains that destruction of this erectile tissue by the galvano-cautery cures the disease. Turbinctomy has been followed by complete relief (Lockard).

The disorder has been ascribed by some writers to the sun's heat in the summer months, also to certain odours, vegetable and animal; but the experiments of Blackley show it to be due to the

specific influence on certain mucous membranes of the pollen-grains of the following natural orders of plants: Ranunculaceæ, Papaveraceæ, Fumariaceæ, Crucifereæ, Violaceæ, Caryophyllaceæ, Geraniaceæ, Leguminosæ, Umbellifereæ, Rosaceæ, Liliaceæ, Compositæ, Graminaceæ, and others, both exotic and native. Different kinds of pollen were applied to the mucous membrane of (1) the nares, (2) larynx, trachea, bronchial tubes (by inhalation), (3) conjunctivæ, (4) tongue, lips, and fauces; and in all these cases it produced the symptoms of hay-fever, the pollen of grasses being most potent. Among these, *Secale cereale*, or rye, exercised most marked effects, though greater influence is generally attributed to *Anthoxanthum odoratum*.

It has been found that large quantities of pollen float in the air during the summer months; and that the number of cases of hay-fever depends on the amount present, which increases in warm, damp weather, decreases when it is very dry and hot, and often nearly disappears after heavy rain. Cold weather reduces the number of sufferers by checking the inflorescence of plants. The higher strata of the atmosphere appear to contain more than that immediately overlying the soil; and Blackley found the greatest number of pollen-particles at between 1,000 and 1,500 feet above the earth's surface, whither they are probably carried by aerial currents. The number of pollen-grains present reaches its maximum in June, when Blackley collected 880 in a day on a square centimetre of glass. The size and forms of the pollen-grains vary greatly in the different species, but this does not seem to influence their action, which appears to depend on the pollen-sac absorbing moisture from the contiguous mucous membrane and bursting, when the minute granules it contains are thus extruded, and cause irritation.

SYMPTOMS.—An attack of hay-fever generally occurs without any premonitory disturbance, immediately on the application of the pollen to the mucous surfaces—for instance, when the person enters a hay-field. The first symptoms are itching of the parts with which the particles come in contact, beginning with the hard palate and fauces, and then extending to the nostrils, eyes, and face, though, if the wind be strong, the eyes may be first attacked. The catarrhal stage follows, marked by violent fits of sneezing, and running from the eyes and nose, with occasional pains in the head and in the frontal sinuses; then the submucous tissue of the nares swells; and in a short time both nostrils become blocked and impervious to air. A change to the recumbent position, however, if the patient lies on one side, will often re-open the uppermost nostril, while the other, as the result of gravity, becomes still more occluded. The sneezing will continue without fresh application of pollen, as at night, when the subsidence of the swelling restores, or even exaggerates, the sensibility of the Schneiderian membrane. The *ala nasi* become red and inflamed, and occasionally bleed. The discharge after this becomes less in quantity, inspissated and puriform, and finally subsides. In the eyes, the swelling of the submucous tissue causes closure of the, lacrymal canals and nasal ducts; and considerable injection of the conjunctival capillaries is apparent. Sometimes, but rarely, oedema of the eyelids follows. Similar to the nose-symptoms are those occurring in the throat, some swelling taking place in the pharynx, which gives rise to partial closure of the Eustachian tubes, and hence to a certain degree of

deafness. Slight feverishness is occasionally present, the pulse quickening to 100, and the temperature slightly rising; but in a large number of cases pyrexia is entirely absent. The changes which take place in the mucous membrane of the air-passages give rise to asthmatic symptoms, such as tightness of the chest, difficult and wheezy breathing, with prolonged expiration, and some dry cough, followed, at the close of the attack, by expectoration.

The catarrhal symptoms are, however, more characteristic than the asthmatic, which are not invariably present.

The liability to attack lasts generally from three to four weeks in summer, but its duration depends on the presence of the exciting cause, which, if not removed, may cause the malady to last for months. A fall of rain will diminish the disorder by clearing the air of pollen; exercise, which increases the number of respirations and, therefore, of pollen-grains inspired, will render it more severe; while each attack makes the individual more susceptible to this subtle influence, and consequently augments the probability of other seizures. As a rule, hay-fever has no complications, and passes away completely on the removal of the exciting cause. Constant recurrence of the attacks has been noticed to lead to deafness, owing to catarrh of the Eustachian tubes.

DIAGNOSIS.—The diagnosis of hay-fever from other affections is easy, as the fact of the catarrhal symptoms occurring only in summer separates it from an ordinary 'cold in the head;' while their combination with dyspnœa in hay-fever prevents it being mistaken for spasmodic asthma arising from other causes, in which there is usually no catarrh.

PROGNOSIS.—The prognosis is favourable if the patient be removed from the exciting cause, as the asthmatic symptoms seldom, if ever, lead to pulmonary emphysema, or to any permanent change in the bronchi.

TREATMENT.—The most obvious course in the treatment of hay-fever, but not always the most easy one, is to avoid exposure to pollen. Blackley notices that a small amount of this material might exist without giving rise to hay-fever, but if ten particles of pollen were detected on the glass slide exposed to the air for twenty-four hours, symptoms were sure to appear in persons liable to it. Sufferers from this complaint should avoid hay-fields, hay-ricks, and much exposure and exertion in the country during the hay-season, and should remain to a great extent within doors; but, where circumstances admit, change to the seaside is highly desirable, and it frequently effects a speedy cure. Even on the coast some care must be taken to select a locality free from vegetation; for if the wind blows from the land, and hay-grass be flowering at the time, an attack may be induced. Choice should be made of a seaside place backed by high cliffs, and where the prevailing winds are from the sea. Another method of avoiding attacks is by sea-voyages or short yachting cruises, undertaken during the hay-season, and many sufferers enjoy complete exemption in this way. Of other localities, high mountain stations, where there is more grazing than hay-growing, and closely inhabited cities with few parks or grassy squares, are to be preferred. Carbolised cotton-wool and other available respirators are sometimes used with advantage.

The medicinal treatment consists, first, in combating the general predisposition to the complaint

by tonic measures; and, secondly, in allaying the local irritation.

The first object is best achieved by shower-baths, and by such tonics as iron, quinine, nux vomica, sulphate of zinc, and arsenic. Lotions of subacetate of lead or sulphate of zinc applied to the eyes and inner surface of the nostrils give some relief; but the writer has found the most successful results from brushing the interior of the nostrils and pharynx with a solution of hydrochlorate of cocaine (20 per cent.) with a curved brush. Various sprays are exceedingly useful, such as those of solutions of carbolic acid (eight grains to an ounce), sulphurous acid (equal parts with water), sulphate of quinine (two grains, with acid, to an ounce), and tannic acid (four grains to an ounce); but the best of all is a solution of cocaine (8 to 10 per cent.) which reduces the sensibility of the parts affected. The spray may be applied, with proper precautions, to all the irritated surfaces—eyes, nose, throat, and larynx—with great relief.

C. THEODORE WILLIAMS.

HEADACHE.—SYNON.: Cephalgia; Fr. *Douleur de tête*; Ger. *Kopfschmerz*.—Pain or uneasiness in the head is very variable in its nature, and is produced by a great number of causes. It is present at some period or other in the course of most acute, and many chronic diseases; and may be associated or not with organic change in the brain, or in other organs of the body. See PAIN IN VISCERAL DISEASE.

SYMPTOMS.—Headache presents many varieties. It may be slight or most intense; superficial or deep-seated. It may be more or less confined to particular parts, as the forehead, the temples, the occiput, or vertex. Sometimes the pain is limited to one spot, producing the sensation as if a nail were being driven into the head, when it is called *clavus*. It may extend over one side of the head, as in hemicrania, or *megrin*; or be generally diffused. Headache presents every variety of character—dull, sharp, cutting, &c. Its accession may be sudden or gradual; and the paroxysms may be of the shortest possible duration, or may extend over hours, days, or months. The pain may be simple, or associated with various perverted sensations, such as giddiness, tingling in the limbs, disordered hearing, or disturbances of vision.

VARIETIES.—For practical purposes headaches may be arranged in the following order:—

1. *Structural headache*, or headache dependent upon disease within the cranium.

2. *Congestive headache*.

3. *Nervous or sick headache*—hemicrania or *megrin*.

4. *Toxæmic headache*.

1. *Structural headache*.—This may be due to any of the many forms of disease of the brain, or of its membranes, such as meningitis, cerebral softening, abscess of the brain, and cerebral tumour; or it may be premonitory of cerebral softening. It is nevertheless often wanting in these disorders, and the locality of the pain, when present, by no means corresponds with that of the lesion. As a rule, the pain of organic disease is fixed and habitual, though sometimes, as in abscess or cancerous tumour, it may be of an intermittent character. If there be sickness associated with it, the sickness occurs without any apparent gastric disorder, and the pain continues after the sickness ceases. Stooeping, and

even the recumbent posture, aggravate the pain, while it is lessened by elevating the head. If organic disease be suspected, the collateral symptoms must be carefully scrutinised and weighed. It rarely happens that organic disease needs to be inferred from pain alone.

2. *Congestive headache*.—Many forms of headache depend upon a greater or less degree of congestion of the vessels of the brain; the congestion being either active or passive.

Active congestion may be caused by hypertrophy of the left ventricle of the heart, general plethora, catamenial irregularities, mental or emotional excitement, and other conditions. The pain in such cases is of an obtuse character, affecting the whole or a part of the head, particularly the forehead and occiput. It is accompanied by a sense of pulsation in the ears, flushed face, glittering eyes, and giddiness on stooping.

Passive congestion may be produced by dyspnoea, by asthma, by valvular disease of the heart, or by disordered action of the liver, bowels, and skin; it may be an after-effect of drunkenness; or may result from any cause which can produce a state of debility in the vessels of the brain, such as general anæmia, exhaustion from fatigue, loss of blood, leucorrhœa, or that following over-excitement, mental exertion, or bodily fatigue—causes which all favour congestion. When the headache is induced by anæmia, debility, or indigestion, the pain occurs generally across the forehead or at the top of the head.

3. *Nervous or sick headache, hemicrania or megrim*.—This disorder is discussed in a separate article. See *MEGRIM*.

4. *Toxæmic headache*.—The headache which attends all fevers and inflammatory disorders, though due in some measure to cerebral congestion, is chiefly caused by the action of the blood, altered in character and elevated in temperature, on the nervous elements of the brain. In uræmia likewise the headache, which frequently precedes or accompanies the other symptoms pointing to the existence of renal disease, results from the morbid condition of the blood. So also, in some persons, breathing the impure air of a crowded room, or the products of the combustion of coal-gas, will, by the imperfect decarbonisation of the blood, speedily produce a headache.

DIAGNOSIS.—Besides the above varieties of headache, pain about the head external to the brain may be produced by *rheumatic* affections of the scalp, with tenderness of the skin and rheumatism in other parts; by *syphilitic* affections of the periosteum or bone; by *inflammation* of the scalp, commencing erysipelas, &c.; and by trigeminal and other varieties of *neuralgia*. Headache may be discriminated from neuralgia by its mode of accession; by the generally longer duration of the attack; and by the more complete intermissions.

TREATMENT.—The treatment of headache must necessarily depend upon the peculiarities of each individual case. In organic or toxæmic headache, the disease with which it is associated, and not the symptom itself, will of course be the object of consideration, and the treatment will be found discussed in connection with these special morbid states. The same remark applies to many forms of congestive headache, such as those produced by disease of the heart, asthma, &c. If catamenial irregularities or

uterine disorders are the exciting cause, these must be treated by appropriate measures. If anemia or debility be present, then, in the intervals between the paroxysms, iron in some form, either alone or in combination with quinine or some vegetable bitter, must be given. During the paroxysms a little sal volatile, a cup of soup or strong tea or coffee, or some weak alcoholic stimulant, may be of service. Where anemia is not a prominent symptom, or when the disorder assumes a periodical or intermittent character, quinine alone, in doses of two or three grains twice or three times a day, may be given; and if this fails to afford relief, arsenic is often of signal service. Great benefit is frequently afforded in the latter cases by the careful administration of alcohol or other alcoholic stimulant during the paroxysm. Except when the headache is associated with general plethora or active congestion, strong purgatives are to be avoided, and the bowels are to be regulated by the mildest aperient which will answer the purpose. The patient's habits and mode of life must also be strictly regulated; and care must be enjoined as to diet, sleep, clothing, and exercise, especially if the headache be associated with dyspepsia. If dyspeptic symptoms are prominent, or if the pain be connected with a gouty diathesis, then these disorders must be treated with their appropriate remedies.

If during the paroxysms the head be hot and the face flushed, warm or cold lotions, iced water, or eau-de-Cologne may be applied; a warm douching may be useful in some cases. Occasionally in severe attacks a few leeches may be placed on the temples or behind the ears with advantage, or a blister to the nape of the neck, but never if the face be pale and the pulse feeble. Compression of the temporal arteries with a pad, sustained pressure around the head, or holding the arms high above the head, will sometimes relieve severe congestive headache.

The treatment of sick headache is discussed under the article MEGRIM. P. W. LATHAM.

HEAD-NODDING OF INFANTS.—**DESCRIPTION.**—Head-nodding, or Spasmus Nutans, is a functional disorder of infancy, characterised by rhythmical movements of the head, which are usually associated with nystagmus.

ÆTIOLOGY.—It begins almost always during the latter half of the first year; two cases have been recorded in which it began as early as the sixth week, and it has begun as late as the twentieth month. Girls are affected rather more often than boys. A very striking seasonal relation is noticeable: of seventy-six published cases seventy began in the six months from October to March, and of these no less than forty-seven began in December or January. It occurs much more frequently in town than in the country, and in the poor than in the rich.

The causation of this disorder is obscure. Rickets is present in many cases, but often only in very slight degree, and it may be entirely absent. Debilitating diseases, such as measles, whooping-cough, or gastro-enteritis, have preceded the onset in some cases, and have been followed by relapses in others. In at least 9 per cent. of the cases head-nodding has followed shortly after a blow on the head from a fall. Dentition has certainly aggravated the movements in some cases, and in others they have subsided after the eruption of a troublesome tooth; but the condition may be present

before the period of dentition. Many of the infants thus affected live in dark rooms, and it has been suggested that, as in miner's nystagmus, which is also occasionally associated with nodding movements of the head, deficiency of light may be an important factor.

SYMPTOMS.—The first symptom is usually the curious movement of the head, which may be rotary as in negation, nodding as in assent, or lateral. The rotary movement is the most common, but is often combined with some nodding or lateral movement; nodding alone is rare. The movement is rhythmical and monotonous, varying in rate from about 30 to 120 per minute. As long as the infant is in the erect position it may be continuous, or may occur only at short intervals; it ceases in the recumbent position, and when the infant's attention is attracted; covering the eyes also stops it. The movement is perfectly easy, not jerky, and seems to cause the infant no discomfort.

Generally within a few days after the onset of the head-movement, sometimes before it, nystagmus in one or both eyes appears; this may be vertical, horizontal, or rotary, and may even have a different direction in the two eyes. If not limited to one eye, it is usually more marked in one than in the other. When horizontal in both eyes it is often convergent not conjugate. It is always a very fine, rapid nystagmus (3-4 per second); it may be continuous or may occur only when the child looks in certain directions. It is increased, or indeed may only be elicited, by fixing the head or by attracting the child's gaze to some object. Hippus and convergent strabismus have occasionally been observed. In looking at objects these infants often put the head on one side, or throw it back, or look out of the corners of their eyes in an odd fashion.

In several cases transient attacks of unconsciousness, like *petit mal*, have been noticed.

PROGNOSIS.—After a few weeks or months the head-movements gradually disappear, and shortly afterwards the nystagmus also ceases. The symptoms rarely persist beyond the age of eighteen months. There seems to be no tendency to epilepsy afterwards, nor is the intellect impaired.

DIAGNOSIS.—Mentally deficient children sometimes show irregular movements of the head which may be distinguished by their occurrence at a later age, and sometimes by their less rhythmical character. A to-and-fro swaying of the trunk is occasionally seen in the second and later years; in some cases it is a harmless habit, in others it is a form of masturbation, while in others again it appears to be epileptic (eclampsia nutans). The age, the involvement of the whole trunk, and in some cases its more purposive character, or the association of epileptic manifestations, may serve to distinguish it. Congenital head-nodding and nystagmus, which may be hereditary and persist through life, can only be distinguished by the history and subsequent course.

PATHOLOGY.—It seems certain that the condition is functional, but nothing further is known of its pathology. There are points which suggest that it is cortical in origin, and it may well be that the imperfect education of the cortical centres at this age favours such a disturbance.

TREATMENT.—Bromides seem to have some slight effect in diminishing the movements; phenazone has also seemed useful; but it is doubtful whether recovery is hastened by the use of drugs.

The infant should live in a well-lighted room, and any predisposing cause, such as rickets, must be treated with appropriate remedies.

G. F. STILL.

HEALING SPRINGS, in Bath County, Virginia, U.S.A.—Thermal waters. See MINERAL WATERS.

HEALTH.—See PERSONAL HEALTH; and PUBLIC HEALTH.

HEALTH-RESORTS.—See CLIMATE; CLIMATE, Treatment of Disease by; and BATHS.

HEARING, Disorders of.—These disorders may be grouped under three classes, namely: (a) *Partial or complete loss of hearing, or deafness*; (b) *Exalted hearing* (so called); (c) *Perverted hearing or tinnitus*. They may be due to various conditions quite independent of any actual disease of the auditory apparatus, and only such causes of disordered hearing will be considered in the present article as are not due to changes in the conducting portion of the ear, which can be demonstrated by the different methods of examination, or to recognised affections of the nervous apparatus connected with hearing. These will be found discussed under the article EAR, Diseases of.

(a) *Partial or complete loss of hearing.*—Perhaps the deafness due to accumulations of cerumen, which so frequently interfere for a time with the hearing of persons whose ears are free from disease, should be regarded as disordered hearing, rather than as a symptom of a pathological condition. As nothing more energetic than careful syringing is required to remove such obstructions, it will be sufficient to observe that in this proceeding the nozzle of the syringe should be directed along the roof of the external canal. Among a very large number of people with the organs of hearing in an apparently healthy state, some few will be found upon whom, throughout their lives, certain notes produce no response. They will not, for example, be able to hear the sounds made by grasshoppers, or the singing of some birds—the call of a partridge, for instance, and in most persons, as age advances, the very high notes are lost. To prove this, it is only necessary to blow one of Galton's whistles in a room full of people, when a considerable proportion of the assembly will fail to catch the high notes, which are distinctly heard by the rest; and although this failure is also noticeable in many nervous affections, all other sounds will perhaps be heard quite normally by these individuals. Emotional influences play a very large part in the destruction or suspension of hearing, and this is especially observable in the case of women. The unexpected sight of a dead husband, hearing of the death of a dear friend, the proposal of a severe surgical operation on a relative, a quarrel, an alarm of thieves, and witnessing a carriage-accident, have each within the knowledge of the writer been followed by intense and sudden deafness, which has only been partially recovered from. The same effect has been noticed in men who have been subjected to prolonged mental strain, in connection with literary work, or during commercial crises. Complete loss of hearing, extending over several months, was on one occasion followed by perfect hearing in a girl of fifteen, on the first appearance of menstruation. The temporary effect of quinine and salicylic acid on the hearing is well

known, but when quinine has been administered in large doses, and for a long period, this special sense is not unusually injured permanently. Among the diseases which often induce a lasting deafness, without any perceptible local change in the conducting portion of the ear, may be included mumps, diphtheria, and many other infective diseases; for although in many instances the middle ear often suffers, this is not always the case, and the immediate cause of the deafness must be sought in the products of inflammation which have been left within the cranium. The same explanation is probably also the correct one in those instances where children lose for ever all hearing power after cerebral excitement or congestion. Habitual and obstinate constipation is sometimes attended with loss of hearing, which returns after the action of purgative medicine. A clot of blood within the cranium, while causing hemiplegia of the opposite side, may destroy the hearing on the same side as the effusion; and a case is on record in which closure of the cerebro-spinal foramen gave rise to this symptom.

(b) *Exalted hearing.*—What is termed 'exalted hearing' will generally, on careful examination, be found to be not so much a definite change in the capacity of the hearing apparatus to receive impressions, as an inability on the part of the patient to receive such impressions without an undue effect on the nervous centres being produced. Thus in many inflammatory states of the brain or its membranes this symptom is often a prominent one. It is also not uncommonly met with in hysterical and nervous persons.

(c) *Perverted hearing.*—Attendant on most of the above examples, and closely allied to deafness, is the often persistent tinnitus; but there are conditions in which this distressing symptom is the chief and solitary trouble. Thus tinnitus, with a feeling of pulsation in the ear, is occasionally the first warning of an intracranial aneurysm; while a furious tinnitus and the hearing of strange noises sometimes precede an attack of acute mania. Patients who have been the subjects of malarial fevers and sunstroke often complain of tinnitus; and as in all cases of disease of the ears, when present, it is the most intractable of symptoms, so it is when the ears have not been the seat of any malady or injury.

TREATMENT.—Since all the above states of disordered hearing may strictly be said to be due to causes which are in themselves abnormalities of one part or another of the organism, it is to these that the treatment will naturally be directed rather than to alterations in hearing which in truth are merely symptoms. See EAR, Diseases of; and TINNITUS.

W. B. DALBY.

HEART, Diseases of.—The diseases of the heart are arranged as follows:

Abscess in walls of. See Inflammation of walls, p. 665.

Aneurysm of walls, p. 632; of valves, p. 663.

Atrophy, p. 633.

Dilatation, p. 633.

Displacement, p. 636.

Endocarditis, p. 650.

Failure. See Dilatation.

Fatty accumulation, p. 639.

Fatty degeneration, p. 639.

Fibroid disease, p. 641.

Functional disorders, p. 642.

Granular degeneration, p. 645.
 Hæmorrhage into walls, p. 645.
 Hydatid disease, p. 645.
 Hypertrophy of, p. 646.
 Inflammation of lining membrane, p. 650.
 Inflammation of walls, p. 655.
 Malformations, p. 656.
 Morbid growths, p. 660.
 Orifices, diseases of, p. 663.
 Palpitation of, p. 642.
 Passive congestion of, p. 660.
 Rupture, p. 661.
 Syphilis, p. 661.
 Thrombosis, p. 662.
 Tuberculosis, p. 663.
 Valves, diseases of, p. 663.
 Wounds, p. 672.

The following articles appear in their alphabetical position in other parts of the book : CIRCULATION, Disorders of; INSANITY IN SPECIAL DISEASES; MYOMALACIA; PERICARDIUM, Diseases of; and PAIN IN VISCERAL DISEASE.

HEART, Abscess of.—See **HEART, Inflammation of Walls of.**

HEART, Aneurysm of.—**DEFINITION.**—A simple depression or a sacculus formed in the walls of the heart and communicating by an orifice, varying in size, with one or more of its cavities.

ÆTIOLOGY AND PATHOLOGY.—Sacs connected with the cavities of the heart have occasionally been found at birth. They must be regarded as congenital malformations. The essential condition which leads to acquired aneurysm of the heart is a change in a portion of the myocardium, by which the resisting-power of the affected part against the pressure of the blood within the cavity is diminished. Under such circumstances a simple depression, corresponding to the weakened spot, may be first formed on the inner surface of the heart. This gradually extends through the cardiac wall towards the external surface, where the resistance becomes less, and where a pouch or sac is then formed, communicating with the cavity of the heart, by a wide or narrow neck. Weakening of the heart's wall may be due either to inflammatory processes, including syphilis and tuberculosis, or to necrosis of the muscular elements, following upon obstruction of the blood-supply.

(a) *Inflammation* affecting the endocardium may extend to the substance of the heart and lead to softening or ulceration : both conditions have been found in connection with aneurysm. In pyæmic states abscesses may form in the walls of the heart, and softening of tubercular masses may also occur there ; cases are recorded in which, the contents of the sac thus formed having been discharged into the circulation, the cavity became converted into an aneurysmal pouch. Syphilitic deposits in the myocardium may result in the formation of patches of fibrous tissue which are liable to yield before the pressure of blood and form aneurysms.

(b) *Degenerative changes* in the muscle-fibres, due to blocking of small branches of the coronary arteries, are also the cause of fibrosis affecting portions of the myocardium. The muscle-fibres undergo fatty changes and are absorbed, their place being taken by a new growth of scar-tissue. Stretching of this fibrous tissue may give rise to aneurysm.

Cardiac aneurysm occurs with much greater frequency among males than among females (45 : 17.

Quain). It may occur at any period of life, acute inflammation being more often the cause in younger persons, degenerative changes in older subjects.

MORBID ANATOMY.—On laying open the pericardium in cases in which aneurysm of the heart exists, adhesions which are more or less universal, or which may be limited to the seat of the disease, are very frequently found. The heart itself is generally enlarged ; and where the aneurysm projects externally it is altered in shape—so much so in some instances that the organ looks like a double heart. The sac may project from the walls as a rounded or conical tumour ; or may assume the appearance of an elongated sac winding round the base of the aorta. Again, nothing abnormal may be observed until the heart is laid open, when a depression or an opening may be discovered in the walls of the ventricle, or in the septum. In rare instances more than a single pouch is formed : there may be several, each communicating with the cavity of the heart by a separate opening, or all by a common neck. The size of the tumour may vary from that of a bean to that of an average-sized cocoa-nut. The opening leading into the pouch may be the widest part of the sac, the aneurysm being a mere depression like a watch-glass or half an egg, or there may exist a constricted or defined neck, leading to a tumour bulging from the walls. The size of the opening may vary from a couple of inches across, to one capable of admitting only a probe. The neck is, in a few cases, described as hard and cartilaginous ; in others, as being smooth and regular, or jagged and irregular. The wall of the aneurysm may consist of practically unaltered heart-substance or of fibrous tissue without any trace of muscular elements. All transition-stages between these extremes may be met with. The thickness of the wall varies from that of a sheet of paper to a quarter of an inch or even more. The two layers of pericardium are frequently adherent over the aneurysm, thus tending to strengthen the sac. The aneurysmal cavity in the majority of cases is lined by smooth membrane ; but in a few instances, apparently of acute formation, the walls may consist of muscular fibres, torn and separated by the blood which has been extravasated among them. The contents of the sac are generally in the form of blood-clots or laminated fibrin, the outermost layers of which may be more or less organised and adherent to the wall.

SEAT.—In the great majority of cases the left ventricle is the part affected (52 out of 56 cases. Quain). Aneurysms of the ventricular septum affecting the 'undefended space' stand on a somewhat different footing from others, as they are probably due partly to developmental defect and partly to endocarditis.

SYMPTOMS.—Usually cardiac aneurysms are only discovered after death, having given rise to no definite symptoms during life. In a few cases symptoms, such as pain, dyspnoea, cyanosis, palpitation, and irregularity of the pulse, may be present, and may be accompanied by a murmur. It is thus evident that the symptoms of aneurysm are such as may exist in other lesions of the heart ; and it is extremely doubtful whether we have at our command the means of diagnosing the existence of this condition.

TERMINATIONS.—Death may result from the disturbance of the heart's action, induced by the presence and the extent of the disease ; from the aneurysm opening into the pericardium ; or from its

burrowing in the wall of the heart, and opening into a cavity of the organ other than that from which it originated. Lastly, one or two cases are recorded in which the progress of the disease had apparently been arrested by the walls of the sac becoming indurated or calcified.

DIAGNOSIS, PROGNOSIS, and TREATMENT.—Since it is probably impossible with our present means of diagnosis to ascertain the existence of a cardiac aneurysm, the question of prognosis does not arise practically, nor is treatment possible. It is well, however, to bear in mind that in cases of heart-disease in which the influence of syphilis cannot be excluded it may often be advisable to try the effect of full doses of iodide of potassium. The benefit of this salt is proved in other forms of aneurysm, and it might also prove serviceable in the cardiac variety. Apart from this, treatment could only be directed in individual cases to reducing arterial resistance, combating any signs of cardiac failure which may be present, and mitigating pain and distress.

R. QUAIN.

W. CECIL BOSANQUET.

HEART, Apoplexy of.—See HEART, Hæmorrhage into the Walls of.

HEART, Atrophy of.

DEFINITION.—Diminution in the size and weight of the heart, without any qualitative change in its structure.

ÆTIOLOGY.—Atrophy of the heart occurs as part of general wasting, provided that the wasting is not the result of any disease which throws additional work upon that organ. The most marked cases of cardiac atrophy are met with in slowly growing carcinomata. In one case of cancer of the pylorus, the heart weighed only $3\frac{1}{8}$ ozs. In chronic pulmonary tuberculosis the diminution in the weight of the heart is less marked, and in nearly half the cases the heart is the normal, or more than the normal, size. This result may be due to the obstruction to the circulation through the lungs. The heart also atrophies when the demands upon it are diminished. Thus amputation at the hip-joint in a young subject is followed by appreciable diminution in the size of the organ.

MORBID ANATOMY.—The heart is uniformly diminished in size as regards both its walls and its cavities. The muscular tissue generally wastes more than the fibrous tissue, and the pericardium may therefore present an opaque and slightly puckered appearance. For the same reason the consistence of the walls is often slightly increased and the section darker than normal. The muscular fibres are small and contain an excess of pigment. Fatty degeneration frequently accompanies atrophy. The co-existence of this change will modify the appearances. See HEART, Fatty Degeneration of.

SYMPTOMS.—Since the size of the heart is proportionate to the demands made upon it there will be no symptoms of cardiac failure unless fatty degeneration be also present. The physical signs are a slightly diminished præcordial dulness, a feeble but not diffused impulse, an apex-beat slightly within and above the usual situation, and possibly a diminished area over which the sounds are audible.

DIAGNOSIS.—From a consideration of the etiology of the condition it will be seen that this is a matter of no importance. It depends upon the existence of a lessened necessity for work, and upon the re-

cognition of the physical signs just given. Emphysema and other causes of diminished cardiac dulness and weakened cardiac impulse must be excluded.

TREATMENT.—The treatment of atrophy of the heart is the treatment of the disease upon which it depends.

H. MONTAGUE MURRAY.

HEART, Calcification of.—See HEART, Degenerations of.

HEART, Cancer of.—See HEART, Morbid Growths in.

HEART, Dilatation of.—**DEFINITION.**—In dilatation of the heart there is uniform enlargement of one or more of the heart's cavities. Dilatation is usually associated with hypertrophy.

ÆTIOLOGY.—The occurrence of dilatation implies that the walls of the heart which yield are too weak to resist successfully the internal pressure to which they are exposed. This defective relation may be due either to actual enfeeblement of the walls of the heart; or to excessive blood-pressure, which even the healthily constituted walls are unable to withstand. The enfeeblement of the walls may be a consequence of acute or chronic degeneration, or may be inherent but unconnected with visible textural disease. The excessive blood-pressure may be dependent on actual obstruction to the circulation; on overfilling; or on undue rapidity of action, which (other things being equal) implies an unwonted expenditure of force. As a matter of fact, however, dilatation and hypertrophy are generally if not always associated; and the processes by which these combined conditions are attained are more complicated than the foregoing statement might lead one to suppose. It will be convenient, therefore, to consider certain cases *seriatim*.

1. In obstructive disease at the aortic orifice; in general stricture of the minute systemic arteries, such as occurs in connection with contracted granular kidneys; and indeed in all cases in which resistance is offered to the free discharge of blood from the left ventricle, progressive hypertrophy of the walls of that ventricle takes place. But the hypertrophy may be complicated even from the beginning with dilatation. The hypertrophy, at any rate at first, is compensatory, and may be taken as a measure of the excess of resistance which the heart is called upon to overcome. The dilatation, however, is not strictly compensatory, and is probably to be regarded as a measure of the inability of the walls to cope fully with the extra work required of them. It is, moreover, obvious that the occurrence of dilatation, by increasing the area of resistance to the intra-ventricular blood-pressure, increases *pro tanto* the muscular effort requisite for the propulsion of the blood into the aorta; and by enlarging the capacity of the ventricular cavity and consequently the amount of blood to be discharged from it, on that account also throws additional labour on the muscular walls of the ventricle. Thus hypertrophy and dilatation react on one another; and hypertrophy, which probably at first compensates for the mechanical obstacle to the discharge of the normal contents of the ventricle, tends, later on, to compensate not only for this but for the virtual disability of the heart which associated dilatation entails.

2. In regurgitant disease at the aortic orifice, hypertrophy and dilatation of the left ventricle also

take place. But in this case, while the hypertrophy probably reaches a higher degree of development than in simple obstruction, dilatation preponderates from first to last; and the ventricle attains larger dimensions than in perhaps any other form of disease. In this case there is no impediment to the escape of blood through the aortic orifice, and therefore *primâ facie* no need for compensatory hypertrophy. There is no doubt that here hypertrophy follows dilatation. The immediate effects of regurgitation are, that during diastole the ventricle becomes more rapidly and completely filled with blood than it does under other circumstances; that hence the subsequent contraction of the auricle tends to distend it unnaturally with blood—*dilatation from overfilling*; and that during the whole of this period the walls have also to sustain the backward pressure of the systemic arterial blood. The result is that, on the principles above enunciated, the walls of the ventricle have to encounter a larger area of pressure, and to expel a larger amount of blood, than natural, and hence are called upon to make excessive effort, and hypertrophy ensues. Thus dilatation from overfilling causes hypertrophy; and both, acting continuously, promote the progressive increase in the capacity of the ventricular cavity and in the thickness of the ventricular walls.

In both cases, but more especially in the latter of them, the ventricle ere long fails to discharge its contents sufficiently into the aorta at each contraction, and the retention of this residual blood becomes an important factor in promoting dilatation—*dilatation with failure*; *residual dilatation*.

3. The effects of continued violent action of the heart, whether caused by nervous influence or by muscular effort, are much the same as those of obstructive disease. For both increased rapidity of contraction and increased amount of blood pouring into the heart, to be expelled at each beat (other things being equal), imply increased expenditure of force; and the persistence of either or both of these conditions causes, therefore, the supervention of hypertrophy and dilatation.

4. The above discussion relates especially to dilatation and hypertrophy of the left ventricle. But, *mutatis mutandis*, it applies with equal force to dilatation and hypertrophy of the other sections of the heart. Thus, in mitral valve-disease, the left auricle undergoes hypertrophy and dilatation—dilatation from overfilling preponderating in regurgitant disease of the valve, hypertrophy preponderating in obstructive disease.

5. In pulmonary valve-disease the right ventricle becomes hypertrophied and dilated—the dilatation being greatest where there is pulmonary regurgitation, the hypertrophy being greatest where the disease is obstructive.

6. In tricuspid valve-disease the right auricle suffers, becoming chiefly dilated in the presence of tricuspid regurgitation, chiefly hypertrophied when there is obstruction. And thus, also, just as when the systemic circulation is impeded the left side of the heart suffers, so when the pulmonary circulation is obstructed, the right side of the heart undergoes enlargement.

In all these cases, therefore, hypertrophy and dilatation seem to result concurrently; but whether the one or the other condition preponderates, depends partly on the particular nature of the cause to which the hypertrophy and dilatation are due,

partly on the inherent strength or weakness of the cardiac walls. In all cases, too, the other cavities of the heart, besides that primarily and directly implicated, suffer, according to their position, from the effects of the greater or less work which sooner or later is cast upon them. It may be particularly pointed out that cavities situated behind the one primarily implicated tend sooner or later to share in its hypertrophy and dilatation; that in cases of incompetence of either of the auriculo-ventricular orifices the corresponding ventricle, in consequence of receiving more than its due amount of blood, and of thus having more work thrown upon it, undergoes dilatation and hypertrophy; and that in obstructive disease of these orifices the ventricles tend to become atrophied from having less work to do.

Acute dilatation which occurs temporarily in acute febrile disorders, such as typhus, is due mainly to enfeeblement of the cardiac walls, but in scarlet fever is partly referable to high arterial tension. See SCARLET FEVER; and RHEUMATISM, ACUTE.

ANATOMICAL CHARACTERS.—In pure dilatation of the heart, the cardiac walls must be thinner than natural. It is a question, however, whether, excepting in the cases of partial dilatation or aneurysm and acute failure, dilatation ever takes place independently of hypertrophy; for even as regards the auricles, where dilatation with attenuation is chiefly observed, there is reason to believe that the attenuation is not commensurate with the extension which accompanies it, and consequently that the total bulk of muscular tissue is increased. When dilatation is associated with no apparent change in the thickness of the walls, hypertrophy is of course present.

It must be mentioned, however, that it is often very difficult to determine on *post-mortem* examination the true relation between the thickness of the cardiac walls and the capacity of the cardiac cavities; for their apparent relation is largely dependent on the condition of the cavities as to systole or diastole at the moment of death; and on the state of the heart as to cadaveric changes at the time of *post-mortem* examination.

The form which the heart assumes in dilatation is the same as that which it assumes in hypertrophy; and indeed, as the two conditions are probably always associated, it is needless to endeavour to establish any distinction between them in this respect. If the dilatation be general, the form of the heart remains unchanged, but its size is uniformly augmented. If the left ventricle be mainly affected, the heart appears not only enlarged but elongated, the left ventricle taking more than its due share in the formation of the cardiac apex. If the right ventricle be specially implicated, the heart becomes enlarged in its transverse diameter; it is more rounded in its contour as seen from the front than it should be; and its apex is obtuse, and either bifid, from the fact that the apices of both ventricles take an equal share in the formation of the cardiac apex, or formed wholly by the right ventricle. If the auricles be dilated, they constitute large masses on both sides of the root of the aorta and pulmonary artery.

The walls of the dilated heart vary not only in thickness, but also in quality. Thus they may be preternaturally firm or preternaturally soft; they may be healthy in structure, or may present more or less degenerative change.

CONSEQUENCES OF DILATATION.—Dilatation of the ventricles, especially if it be considerable, is apt to disarrange the mechanism of the auriculo-ventricular valves. Even in mere temporary distension of the right ventricle a kind of safety-valve action of the tricuspid valve takes place, in consequence of which regurgitation of blood occurs from the ventricle into the auricle. Moreover, established dilatation of the right or left ventricle is liable to be attended by persistent regurgitation of blood through the corresponding auriculo-ventricular orifice. The defaulting valve under these circumstances has a natural aspect; but careful examination shows either that the orifice has undergone dilatation in company with the ventricle—the valve itself presenting no corresponding increase; or that there is a want of relation between the size of the muscular papillares and chordæ tendinæ on the one hand, and the capacity of the ventricle on the other, which interferes with the due closure of the valve.

It is obvious that if regurgitation becomes established, the usual consequences of regurgitation will presently ensue—namely, in connection with affection of the left side of the heart, dilatation and hypertrophy of the left auricle, and subsequently congestion of the lungs and pulmonary apoplexy; and in connection with affection of the right side of the heart, dilatation and hypertrophy of the right auricle, fulness of the systemic veins, anasarca, nutmeg liver, and congested, indurated kidneys. It is also obvious that, even if no regurgitant condition be developed, dilatation of the heart, which implies feebleness of heart and imperfect circulation, must ultimately induce the ordinary remote consequences of heart-disease. A further consequence of dilatation and other cardiac affections attended by feeble circulation is the formation of thrombi during life, both in the heart itself and in other parts of the vascular system. Dilatation of the left auricle may cause compression of the left bronchus.

SYMPTOMS.—Since dilatation of the heart rarely, if ever, exists alone, but is associated with hypertrophy, valve-disease, degenerations, and other conditions, it is almost impossible to make any definite statement with regard to the signs and symptoms by which its presence may be recognised. Still, there is no doubt that dilatation is one of the most important factors of heart-disease, clinically considered; and that its supervention materially affects the patient's condition and prospect of life. Dilatation, as a rule, implies weakness, and over-distension of the implicated cavities with blood, which probably never becomes sufficiently expelled.

The *physical signs* of dilatation are necessarily in many respects the same as those of hypertrophy. The præcordial dulness is increased in area—the extent and form of this area, and the situation of the apex-beat, being determined by the general size of the heart, and the relative dimensions of its component parts. In proportion, however, as residual dilatation preponderates over hypertrophy, the impulse of the heart becomes weak, and possibly to some extent diffused. In extreme dilatation, as in extreme weakness from other causes, the sounds of the heart, and especially the first sound, are enfeebled. And it may be asserted that generally the tendency of dilatation is to shorten the first sound, and to give it the characters of the second sound. It has, nevertheless, been observed over and over again that it is in the concurrence of

hypertrophy and dilatation that the cardiac sounds attain their greatest intensity. The feebleness of the heart's action in residual dilatation is generally attended before long by more or less irregularity; and even in the absence of valve-disease, a mitral or tricuspid systolic murmur, implying regurgitation, is apt to be established.

The *symptoms* of dilatation are to a large extent those of cardiac obstruction, and more especially of mitral disease. The patient complains of weight, oppression, or uneasiness in the cardiac region, with probably a sense of fluttering there, and of a tendency to sighing respiration. He becomes short-breathed, and may have extreme dyspnoea. His face is apt to become livid; his extremities cold and blue; and his pulse weak and irregular. Dilatation of the systemic veins arises sooner or later; and subsequently general anasarca, commencing in dependent parts, serous effusions, epigastric pulsation, pulsation of the veins in the neck, and of the liver, together with the other usual consequences of cardiac failure. The chief of these are—congestion of the lungs, with pulmonary apoplexy, cough, and expectoration of blood; congestion, enlargement and tenderness of the liver, with jaundice; and congestion of the kidneys, attended by the discharge of scanty, high-coloured, heavy urine, containing albumen and possibly blood. Other symptoms, referable to the nervous and digestive organs, which need not be enumerated here, are also liable to supervene.

The symptoms will vary, of course, according as the left or the right ventricle is mainly affected. In the former case we are liable to have at first irregularity and feebleness of pulse with tendency to faint; then pulmonary complications; and at a later period, symptoms referable to the systemic venous circulation. The latter case is one of considerable interest; because in a large number of instances it is, in its most marked form, a consequence of emphysema of the lungs, or of other analogous conditions, and moreover is apt to come on very rapidly. Under these circumstances, there is necessarily much dyspnoea, but the systemic venous and capillary systems speedily become overloaded; extreme cyanosis often develops rapidly; and, before long, all the other symptoms referable to disease of the right side of the heart become established: namely, pulsation of the veins in the neck, and of the liver; epigastric pulsation, general anasarca, dropsy of the serous cavities, with perhaps petechial extravasations, jaundice from nutmeg liver, and albuminuria from congestion of the kidneys.

PROGNOSIS.—There is no doubt that some degree of dilatation of the heart, and more especially of the right ventricle, may arise either from over-exertion, or from functional disturbances, and in connection with pulmonary disorders. But such dilatation is for the most part temporary or remediable; and only by continuance of its cause does it become established and a matter of serious importance. Dilatation from over-filling, as of the left ventricle in aortic incompetence, is a permanent condition and not necessarily of evil omen. In the same way there is no doubt that the residual dilatation which comes on in the course of structural disease of the heart or lungs, or of other structural diseases which influence the action of the heart, is remediable within certain limits by due attention to the conditions under which it arises. Nevertheless

it is certain that the presence of dilatation with failure of the heart in connection with other diseases, more especially those of the heart itself, lungs, or kidneys, is a grave source of danger; and that in the great majority of cases it is of fatal omen, aggravating the patient's distress, and hastening his death.

TREATMENT.—The treatment of dilatation of the heart merges in that of the other cardiac conditions with which it is associated, and in that of other diseases in the course of which it may have supervened. It may be stated, generally, however, that the treatment is that of cardiac debility, and of distension of the heart with blood. See HEART, Valves of, Diseases of.

The chief indications, therefore, are rest of mind and body; avoidance of exposure to cold and wet; the exhibition of ample, nutritious, and readily digestible solid food; due attention to the action of the bowels, kidneys, and skin; and the employment of medicines likely to regulate and strengthen the action of the heart. For the last purpose digitalis in sufficient doses at short intervals is universally acknowledged to be of great value. It is desirable to combine the digitalis with other diuretics, such as potassium acetate and scopolium, until the anasarca has been removed, and afterwards with iron or some vegetable tonic. Ammonia and other diffusible stimulants are often called for to relieve urgent distress, and are of great service. In cases where there is much lividity, and evidence of stagnation of blood in the right side of the heart, removal of blood by venesection or leeching will often prove of great service.

When the dilatation is due to pulmonary disease, this of course requires primary and especial treatment.

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HEART, Displacements of.—Besides the displacements of the heart that occur as the result of developmental errors or of disease, there are certain changes of position which this organ undergoes in health. The most important of these physiological displacements of the heart are—first, its vertical movements in respiration; and, second, the alterations in its situation corresponding with changes in the bodily posture. The present article, however, will deal only with abnormal displacements of the heart.

I. Congenital Displacements.—Such misplacements may be either within the cavity of the thorax, or external to it.

1. Of the *internal* malpositions—*ectopia cordis intrathoracica* or *ectocardia intrathoracica*—the most common is that to which the term *dextrocardia* has been applied. In this the heart is in a very similar position on the right side of the chest to that which it should occupy on the left. This condition may co-exist with transposition of the other viscera of the body, or it may occur alone. When the heart is misplaced, the aorta generally follows an irregular course, crossing the right bronchus and passing down to the right side of the bodies of the vertebrae; and the right carotid and subclavian arteries are given off as separate vessels, while the brachiocephalic trunk is situated on the left side. In some instances, however, the vessels at the arch are not transposed; while in others the aorta, after passing over the right bronchus, crosses the spine and follows its usual course to the left of the bodies of the ver-

tebrae. In cases of transposition the heart itself may be well formed; or it may be very imperfectly developed.

In another kind of misplacement, *mesocardia*, the heart is situated more in the median line than natural—a position which it occupies in the fetus at the earlier periods. Cases have also been recorded in which the organ occupied a transverse and an antero-posterior direction.

2. Of the *external* misplacements, those in which the heart is situated externally to the thoracic cavity—*ectopia* or *ectocardia extrathoracica*—the most common is that in which, from deficiency of some part of the sternum, the organ lies in front of the chest—*ectopia cordis* or *ectocardia pectoralis*. In other cases, from deficiency in some portion of the diaphragm, the heart is placed in the abdomen, either lying in the peritoneal cavity, or, if the integuments are partially defective, in a sac in the præcordia—*ectopia cordis* or *ectocardia abdominalis*. In a third form the heart lies at the root of the neck—*ectopia cordis* or *ectocardia cephalica*.

SYMPTOMS, DURATION, AND TERMINATIONS.—When the heart is well formed, its malposition within the thorax does not necessarily cause such interference with its functions as to be productive of symptoms, or materially to curtail the duration of life. Indeed, cases are on record in which the heart and other viscera have been transposed in persons who had never presented any signs of disorder of the circulation, and who lived to very advanced ages. When, however, the organ is also defective, and especially when the displacement is external to the thoracic cavity, life is usually only of short duration—though some remarkable cases of external displacements are on record, in which the patients survived to advanced ages.

II. Acquired Displacements.—**ÆTIOLOGY.** The causes of acquired displacement of the heart may be arranged in two classes—namely (1) conditions that exert *pressure*, and (2) conditions that exercise *traction*, upon the heart.

1. The heart is *pressed* or pushed out of position by effusions of fluid—inflammatory, dropsical, or bloody—into either pleural cavity; by pneumothorax of either side; by intrathoracic tumours—whether mediastinal (including aneurysm and abscess), pulmonary, or parietal; by hypertrophic emphysema, or other causes of enlargement of the lungs; by extensive pneumonic consolidation; or by abundant pericardial effusion of any kind. Certain conditions of the abdominal contents produce a similar effect—for example: gaseous distension of the stomach and intestines; enlargement of the liver and other solid organs; abdominal tumours of all kinds; the pregnant uterus; and ascites, when considerable. Hernia of the abdominal viscera through the diaphragm, and abscesses in relation with the diaphragm, also cause displacement of the heart.

2. The heart suffers *traction*, or is drawn out of position, during absorption of pleuritic effusion with imperfect expansion of the lung, on either side; by the contraction of pleuro-pericardial adhesions; of pulmonary cirrhosis, or of cavities in phthisis; in collapse of either lung from pressure on the main bronchus; and in some forms of deformity of the chest from curvature of the spine.

MECHANISM OF DISPLACEMENT.—When the displacing force belongs to the first or pressure-class, it acts against the surface of the pericardium and heart that is opposed to it, and presses or pushes it

a tergo, away from its own seat, in the direction of least resistance. Thus the heart is, speaking broadly, pushed towards the left by effusion into the right pleural cavity; towards the right by similar disease on the left side; downwards by tumours in the region of the base; and upwards by gaseous distension of the stomach.

On the other hand, when the displacing force is of the nature of traction, the heart is drawn *a fronte*, that is, towards the seat of the force. Thus, when a cavity in a tuberculous lung is contracting, the pericardium and heart, as well as the walls of the chest, are displaced towards the healing area. It must be observed, however, that in this class of cases, actual traction, in the strict sense, is rare, and that the ordinary displacing force is, in reality, the atmospheric pressure; the heart and the other organs being 'sucked' towards the potential vacuum, in the same way as water is 'drawn' into a syringe. Still, in a very small number of cases, the pericardium does actually become involved in a healing process in the lungs; and it and the heart are dragged towards the cicatrix.

Besides the displacing force, there are at work in dislocation of the heart certain other agencies, which contribute to the result, whether their effect be to increase or to diminish that of the chief cause:—

(a) The weight of the heart manifestly favours displacement in different directions, according to the posture. Thus, in the erect posture, it favours downward and limits upward displacement. However, the effect of the weight of the heart is comparatively insignificant, and may be practically disregarded.

(b) The resistance, positive or negative, of neighbouring parts must be taken into account. The heart when disturbed from its position will move in the direction of least resistance. Thus it cannot be displaced to any extent either forwards or backwards, but is moved with comparative ease towards either pleural cavity. The resistance inferiorly is greater under the right half of the diaphragm than under the left. On the other hand, the resistance around may become negative; for example, in left pleural effusion the corresponding half of the diaphragm is pushed downwards, and the accompanying downward displacement of the cardiac apex is thus increased.

(c) The heart is attached at its root; and, speaking broadly, this is a fixed point, at the right and upper extremity of the long cardiac axis. This attachment will limit and otherwise modify displacements of the heart in all directions, especially downwards. Round this point as a centre, and with the long axis as the radius, the apex of the heart would describe an arc of a circle, cutting the surface of the chest in the left axilla, the left submammary region and the epigastrium.

(d) The tendency that the heart has to rotate or roll on one or other of its axes is also affected by its attachment at the root. If the heart lay free in the pericardial cavity, there would be no limit to such rotation under the influence of pressure or of traction. The base being fixed, rotation is greatly limited, and does not occur to any extent except around the longitudinal axis; the left ventricle, for example, being rotated more forwards or more backwards, as the case may be. Rotation round the transverse-horizontal and the antero-posterior-horizontal axes is very limited.

ANATOMICAL CHARACTERS AND EFFECTS. — The

only essential change that the heart is found to have undergone in displacement is an alteration of its relations to the surrounding parts. The softer parts of the cardiac wall, however, such as the auricles, are sometimes compressed to a moderate degree. The pericardium is partly dislocated and partly stretched. The great vessels at the base of the heart and at the root of the neck may be elongated, shortened, twisted or bent, according to the particular form of displacement; and the circulation within them impeded. The neighbouring organs are variously displaced and compressed. One of the effects often seen after displacement is permanent fixation of the pericardium and heart in their new position, for example, in the pleural cavity, on the disappearance of the original cause.

The effects of displacement of the heart upon its functions differ greatly in the two classes of dislocation to which we have referred:—

In displacement due to *pressure*, the heart is compressed between the displacing force and the resistance in other directions, and the dislocation is generally rapid. Fortunately, in most cases of such displacement the resistance is slight; and the heart, if healthy, suffers little or no real compression of its substance or cavities between the two forces, the mobile and compressible lung especially yielding before it. But if the heart be diseased—and especially if its walls be weak, degenerated or dilated—moderate compression, as in flatulence, may cause embarrassment of the cardiac action and even fatal paralysis; and the rapidity or even suddenness with which displacement generally occurs when due to pressure—for example, in pneumothorax—is another and perhaps the principal cause of such embarrassment.

On the other hand, when the heart is *drawn* out of its normal situation towards a tubercular cavity, or towards either pleural cavity from which an inflammatory effusion is being absorbed, the displacement occurs, not because there is want of space, but because there is excess of space, within the thorax. The process is also very gradual. The effects, therefore, upon the functional activity of the heart may be said to be few, though the unusual pulsation may be a source of inconvenience, and even of anxiety, to the patient. In very rare cases, the heart and pericardium, when thus displaced, may be involved in the fibrotic process going on in the lung or pleura, and the adhesions thus established may ultimately interfere with the cardiac action.

SYMPTOMS.—In displacement of the heart, special symptoms are frequently slight or altogether wanting; or they are inseparable from the symptoms of the original cause. This may be said to be almost invariably the case when the displacement is due to gradual traction, as in phthisis. In the pressure class of cases, on the contrary, there are frequently developed, and that rapidly or suddenly, symptoms due to compression of the heart, such as a sense of distress, stifling, and pain over the præcordia or at the epigastrium, or even true angina; dyspnoea, perhaps amounting to orthopnoea; palpitation; blueness of the surface; and irregularity and feebleness of the pulse. When the displacement is due to upward pressure from gaseous distension of the stomach and intestines, the above symptoms may be associated with flatulence or 'spasms,' and are relieved by the erect posture, eructation, vomiting, and the administration of carminative and absorbent remedies. If this condition be not removed within

a short time, it may become aggravated, pass into a state of collapse, and end in death.

VARIETIES AND PHYSICAL SIGNS.—The varieties of cardiac displacement, according to the direction in which the dislocation occurs, may, for clinical purposes, be said to be as follows: towards the *left*, towards the *right*, *downwards*, *upwards*, *backwards*, and *forwards*. It must be observed, however, that this is only a broad general classification, and that the heart is very rarely displaced in an absolutely horizontal, or in an absolutely vertical plane. The exact direction taken in each variety will now be described, as well as its special causes, and the physical signs by which it may be recognised.

1. Displacement towards the Left.—This, the commonest variety of marked cardiac dislocation, is most frequently caused by contraction of the left lung from any of the conditions already enumerated, and by effusions into the right pleural cavity. Right pneumothorax, and tumours connected with the right side of the chest, with the mediastinum, or with the right lobe of the liver, are less common conditions leading to the same result. The distance towards the left to which the heart is dislocated varies, the extreme limit being probably the anterior axillary line. During its progress towards the left, the heart is rotated around its longitudinal axis, so that the right ventricle is more exposed anteriorly; and the apex is moved, at first somewhat downwards, and afterwards upwards.

The visible and palpable impulse is found to the left of its normal situation, and either lower or higher than it, or on the same level with it, according to the degree of displacement. In some cases due to contraction of the left lung, the impulse may be found in any one or in all of the left intercostal spaces from the base to the apex of the heart, and of different rhythm in the different spaces. If the displaced heart be the seat of valvular disease, a thrill may be felt in an unusual situation, for example, in the left axilla. The area of percussion-dulness is altered in outline, being invaded on the right side either by the dulness due to effusion there, or by resonance due to pneumothorax or to encroachment of the right lung-border; while it is either transposed towards the left axilla, or blended with unnatural dulness over the left lung. The cardiac sounds are reduced in loudness over the normal præcordia, while they are unnaturally loud towards the left axilla. Structural murmurs if present are similarly affected, as regards the seat of their greatest intensity and the lines of their conduction. A systolic murmur may be developed at the base of the heart from distortion of the great vessels.

2. Displacement towards the Right.—This form of dislocation of the heart is the result of effusion into the left pleural cavity; of contracting processes connected with the right lung or pleura; of left pneumothorax; and of tumours of the left side of the chest or in the mediastinum. The heart may be displaced towards the right side until an impulse is found in the axillary region, but the true apex at the same time is transferred only as far as the epigastrium. During its lateral movement, the heart is rotated on its longitudinal axis in such a manner that the left ventricle is more exposed.

The physical signs correspond closely with those enumerated under left displacement—the two sides being, of course, exactly reversed. The cardiac impulse is most frequently transferred to the epigas-

trium. New areas of pulsation are developed in the region of the right nipple, and in the second and third right interspaces, close to the sternum; they indicate the displaced position of the right auricle, if præ systolic, or of the aorta, if systolic and followed by palpable shock in diastole. The description of the auscultatory phenomena, as regards both sounds and murmurs, does not require to be repeated.

3. Displacement Downwards.—This is an exceedingly common form of cardiac displacement, though seldom extreme in degree. It is the constant result of hypertrophic emphysema of the lungs; and may also be caused by the downward pressure of tumours at the base of the heart, such as aneurysm, and by collapse of the stomach and intestines. Displacement of the heart downwards is limited by the diaphragm, and by the attachment of the pericardium and great vessels at the root of the heart. At the same time the apex may either move somewhat towards the left in its descent if the downward pressure be uniform, as in emphysema; or it may ascend somewhat towards the left if the pressure be exerted chiefly upon the base.

The ordinary apex-beat is generally quite imperceptible in this form of displacement, on account of enlargement of the lungs; or it is greatly weakened, and situated in the sixth left space, or lower, to the left of its normal position. A new area of systolic pulsation is perceptible in the epigastrium, generally well marked, and connected with the right ventricle. The præcordial dulness is usually completely replaced by pulmonary resonance; or, more rarely, confused by the dulness of some form of mediastinal tumour. The cardiac sounds are feeble, or absent, over their usual seat; and are heard, instead, over the epigastric triangle and the lower left cartilages.

4. Displacement Upwards.—The many abdominal causes of this form have been already mentioned; so have the symptoms due to compression of the heart which characterise it when so produced. The heart, as a whole, is moved upwards in the chest, and at the same time the apex passes more or less towards the left, and the right ventricle may become somewhat more exposed anteriorly.

The cardiac impulse is elevated until it is found on the nipple-level, or even higher; or it is lost, along with the area of percussion-dulness, behind the inferior border of the left lung. The sounds of the heart are transposed upwards, and weakened. The displacement of the cardiac apex towards the left axilla in pericardial effusion is described elsewhere. See PERICARDIUM, Diseases of.

5. Displacement Backwards.—This variety of displacement of the heart is very uncommon; and when it does occur, is generally referable either to abundant pericardial effusion, or to backward curvature of the spine (kyphosis) in the dorsal region. A certain amount of backward displacement is, however, not very rare in extensive excavation of the left lung, in association with other forms of dislocation. The base of the heart is then the part most transposed into the left paraspinal groove, and the apex is tilted somewhat forwards as well as elevated.

The physical signs of backward displacement are those of the cause of the malposition rather than any that can be referred to the condition itself.

6. Displacement Forwards.—Displacement forwards is also very rare, although it is frequently simulated by bulging of the præcordia in enlarge-

ment of the heart. The chief cause of it is the presence of a tumour in the mediastinum—especially aneurysm of the descending aorta, or enlargement of the bronchial glands. The amount of actual transposition is necessarily exceedingly small, the anterior border of the lungs being compressed or pushed aside, but the further progress of the heart forwards arrested by the anterior wall of the chest.

The physical signs are, therefore, increase of the area and strength of pulsation and of percussion-dulness over the præcordia, bulging of the same in young subjects, and increased loudness of the cardiac sounds in that situation.

7. Complex Displacements.—It has already been indicated that displacements of the heart, strictly speaking, occur almost without exception in more than one of the directions described; and they may all, therefore, be said to be generally more or less complex. Dislocation at once upwards and towards either side is especially common, as the result of contracting processes in the apex of the lung.

DIAGNOSIS.—After the full account that has been given of the several forms of displacement of the heart, there ought to be no great difficulty in diagnosing them from each other, as well as from the conditions which simulate them. These must be carefully remembered. The chief of them are: (1) physiological displacements, already referred to; (2) cardiac enlargement, especially when attended with bulging of the præcordia; (3) pulsating tumours of the chest and abdomen, particularly aneurysm of the aorta; (4) adhesion of the pericardium; and (5) atrophy of the lungs from any cause.

TREATMENT.—The rational treatment of displacement of the heart would be to remove its cause; but when the cause is of the nature of traction, treatment is very rarely called for, even if it were possible. In displacement due to pressure, on the contrary, treatment is often urgently indicated, perfectly practicable, and highly successful by attention to the cause. The unpleasant sensation of pulsation complained of in some instances of displacement—for example, in phthisis—is frequently relieved by an assurance on the part of the physician that the palpitation is of no import; and by the application of a simple plaster, containing iron, belladonna, or opium. J. MITCHELL BRUCE.

HEART, Embolism of.—See **EMBOLISM**; **HEART, Thrombosis of**; and **PULMONARY ARTERY, Embolism of**.

HEART, Failure of.—See **HEART, Dilatation of**.

HEART, Fatty Accumulation on.—**SYNON.** : Fr. *Hypertrophie graisseuse du Cœur*; Ger. *Fettige Infiltration des Herzens*.

DEFINITION.—The growth of fat on the surface and in the substance of the heart, in quantity sufficient to interfere with its functions, and thus to constitute a disease.

ETIOLOGY.—Whenever excessive quantities of fat are deposited in the subcutaneous tissue and other usual sites, the sub-epicardial tissue is also likely to become the seat of an excessive accumulation (see **OBESITY**). It is therefore a disease of late middle life, the majority of cases being over fifty years of age.

MORBID ANATOMY.—The accumulation is always most marked in the places where fat is normally

present—i.e. the track of the coronary arteries and their branches; thence it spreads until, in extreme cases, the muscular walls of the right ventricle may be entirely concealed by a thick layer of fat, though the left ventricle is rarely, if ever, completely covered. When the fat has accumulated to this extent the walls of the heart lose all appearance of muscular structure, looking like a thick layer of adipose tissue. Near the surface, on microscopical examination, but few muscular fibres can be seen, and the very wide intervals between them are occupied by fat-cells (see fig. 1). Proceeding inwards, the fat-cells become fewer, and finally disappear before the endocardium is reached. It is worthy of note that the fibres, though surrounded by fat, may still retain their normal structure. Fatty degeneration, however, often co-exists with fatty accumulation.

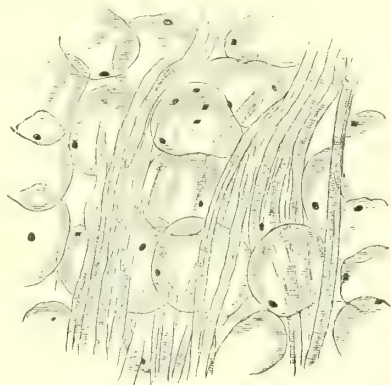


FIG. 1.—Fatty growth in the substance of the heart. $\times 400$ diam.

SYMPTOMS.—The fat accumulated on the heart and in its substance has no action unless present in sufficient quantity to affect the nutrition and therefore the contraction of the muscular fibres among which it lies. The feeble circulation and easily induced dyspnoea often found in obese persons are perhaps occasionally attributable to the presence of the fat over the heart; but in the majority of cases the presence of the fat is an indication of the diminished functional activity of the heart rather than the cause of it. A heart affected in this way is more prone to rupture.

The presence of fatty accumulation on the heart may be suspected in an obese person when the pulse is small and weak, when the first sound of the heart is feeble and the impulse weak, and when the extent of dulness on percussion is increased. It is probable, however, that in many of these cases fatty degeneration of the muscular fibres is also present.

TREATMENT.—The treatment of fatty accumulation on the heart is identical with that of its cause, obesity. See **OBESITY**.

If the symptoms above mentioned are present, especial care must be taken not to put any excessive work upon the heart, but by tonics, passive movements, massage, and the promotion of free excretion to aid in the restoration of the normal strength of the organ. H. MONTAGUE MURRAY.

HEART, Fatty Degeneration of.—**SYNON.** : Fr. *Dégénérescence graisseuse du Cœur*; Ger. *Fettige Metamorphose des Herzens*.

DEFINITION.—A pathological process in which the muscular fibres of the heart are converted into a granular fatty matter.

ÆTIOLOGY AND PATHOLOGY.—The process by which this change takes place, as well as the circumstances under which it occurs, is discussed under the head of FATTY DEGENERATION.

This change is best seen in cases in which the orifices of the coronary arteries are contracted or where there is thickening or calcification of one of the trunks or of the branches of these vessels as a result of syphilis, strain, chronic nephritis and, other causes of arterial degeneration (*see* ARTERIO-SCLEROSIS). The fatty change is found to occur in the more *diffused* or *general* form in those diseases in which the vital powers are lowered, as in chronic cachectic disease, pernicious anæmia, poisoning by phosphorus and chronic alcoholism, and after loss of blood. In the acute specific fevers and septic diseases the tissue of the heart also undergoes this change. A more or less diffused form of fatty degeneration takes place in cases of enlarged heart, since these enlarged hearts require a larger supply of the materials for nutrition than can be furnished to them by the coronary arteries, which in such cases are frequently themselves diseased, both at their origin and in their course. The disease also occurs after delivery in some instances, the heart having been enlarged during pregnancy; and certain cases of sudden death after parturition are thus explained. The disease is more frequent in males, in the proportion of nearly two to one.

ANATOMICAL CHARACTERS.—In fatty degeneration the heart is enlarged in about two-thirds of the cases. Not infrequently it is simply dilated; but it may be of average size, or even below the normal. The colour of the heart's substance is pale; generally it is of a yellowish-brown or buff, or muddy pink colour. This discolouration is seen in spots or patches; and though the whole heart may be pale, the spots, being still paler when seen beneath the endocardium, give the tissue a mottled look. The same appearance may be observed beneath the pericardium, and in the substance of the heart. With the progress of disease the spots run together, giving portions of the walls a uniform buff-coloured character, while the rest of the organ retains its ordinary aspect. The consistence varies from that of mere flabbiness or softness, to such a condition as permits of the tissue being torn like wet brown-paper. The organ then feels like a piece of wet chamois-leather, or a wet glove. In other cases the heart retains in appearance much of its ordinary colour, but the tissue breaks down on pressure, as does a lung consolidated by pneumonia. This is a state which occurs more frequently in hearts which are hypertrophied.

All parts of the heart are subject to fatty degeneration, but not equally so. It is most frequently found in the left ventricle; next in the right ventricle; then in the right auricle; and least frequently in the left auricle. It is generally more evident in the columnæ carneæ, and in the inner layers of the muscular walls, than elsewhere.

The *microscopical characters* of this disease are described in the article on FATTY DEGENERATION. The effects on the heart of fatty degeneration include *rupture* (in twenty-five out of sixty-eight cases); and partial rupture, leading to effusion of blood into the walls ('*cardiac apoplexy*'), cyst-formation, and false aneurysm of the heart. The last

may also form by simple yielding of a portion of the softened cardiac wall. The involvement of the columnæ carneæ sometimes leads to imperfect action of the valves; but valvular disease itself is not often present in this connection, and when the valves are affected it is chiefly the aortic valves that are involved.

SYMPTOMS AND DIAGNOSIS.—There is no doubt that many cases occur in which fatty degeneration is found in the heart after death, without its presence during life having been suspected, particularly in exhausting disease of which it simply forms a part. In such cases the requirements of the system may not be out of proportion to the powers of the heart; death then comes on slowly and insidiously without attention being attracted to the state of this organ. On the other hand, when the heart suffers from some local cause, such as disease of the coronary arteries, while the system generally maintains its powers more or less fully, the balance between it and the heart is lost, and evidence of the change that has occurred in the central organ of circulation can be traced without difficulty. Among the symptoms of the disease we then observe faintness and syncope, disordered respiration, pain in the region of the heart, disturbed pulsation, drowsiness, and coma. The patient complains in the earlier stages of being easily exhausted, particularly by mounting ascents; he says he feels faint on reaching the top of a flight of stairs; though not giddy, he feels he must fall; though not breathless or fainting, he sighs deeply and seeks the air. Any unusual excitement, or a heated or close atmosphere, produces the like effects. At the same time there is often experienced an uncomfortable sensation of choking or fulness in the chest. In the intervals the patient may be fairly well.

Of the effects of fatty degeneration upon the functions of the heart, the most prominent are those which exhibit the weakness of the organ. *Coma*, preceded or not by giddiness, is a striking symptom. Adams of Dublin mentioned as many as twenty attacks in one of his cases of fatty heart; and Quain noticed the occurrence of even more frequent seizures. The term '*cardiac syncope*' may be well applied to the condition of faintness which is frequently found in connection with fatty heart. In some cases the feeling of syncope amounts to little more than a simple sense of faintness—that the patient must fall if he do not lay hold of something. In other instances it is accompanied by a feeling of impending death; and such patients do frequently die. Death may result in such cases from cardiac failure, as indicated after death by a flabby heart containing blood in the left ventricle; or, where there is a less amount of degeneration, death may result from irregular action or spasm, and the ventricles may be found empty.

Præcordial pain is also an effect of fatty degeneration. It may occur independently of syncope; its association with syncope constitutes what is called *syncope anginosa*. *See* ANGINA PECTORIS.

The *respiration* is always affected in cases of fatty degeneration, the result being either simple breathlessness, even on slight exertion, or Cheyne-Stokes respiration. In some instances the disturbance is represented as a sense of choking or suffocation; the person feels as if he were breathing through a sponge. In some instances it is so slight as scarcely to be regarded; in others so severe that the smallest effort, particularly in mounting ascents,

is most painful. In Cheyne-Stokes respiration there is usually no change in the heart's action; but during the phase of dyspnoea, the heart's action has been observed to become remarkably irregular.

As the disease advances, the attacks, of whatever kind, become more frequent and severe, and often disturb and distress the patient at night. The temper becomes irritable; the expression frequently appears anxious, and the countenance sallow. Copious perspiration from very slight causes, coldness of the extremities and swelling of the ankles, are among the further incidents of the disease. The pulse is generally affected; but its characters depend upon the part of the heart affected, and on the extent and degree of the disease. Intermittence and irregularity are the more frequent alterations; weakness is another; and infrequency—often remarkable—is a third. Rapid pulse, more especially when it increases with age, is a symptom deserving attention in the diagnosis of fatty degeneration. The irregularity may be constant, or it may appear and disappear: it will be present during the slight disturbances above described, disappear altogether when the patient is in tolerable health, and return as the effect of any depressing cause—a striking observation, because the cause is insufficient to affect a sound heart. Still, the frequency with which the pulse is affected in cases of simple dyspepsia, more especially along with the gouty diathesis, should not be overlooked in the diagnosis of fatty heart.

It was formerly thought that the presence of *arcus senilis* was characteristic of this disease, but this view is now generally discarded. See *ARCUS SENILIS*.

As the disease progresses the symptoms become more marked; the various effects of feeble and languid circulation further show themselves; angina may perhaps become fully developed; or the patient may be cut off suddenly by one or other of the effects connected either immediately or remotely with the lesion itself. Of 83 cases of 'fatty disease' of the heart collected by Quain, 68 had died suddenly.

The *physical signs* that characterise fatty disease are neither many nor marked. They are—a feeble præcordial impulse, proportionate to the extent and the degree of the disease; a feeble muffled first sound, under like conditions, sometimes scarcely audible. When the heart is enlarged, the impulse, though weak, will be found extended, and so likewise will be the dulness on percussion. A murmur may be present, from degeneration of the columnæ carneæ, as suggested by Rokitsansky. The second sound is often distinct and clearly accentuated, as compared with the first.

DIAGNOSIS.—When the heart is hypertrophied or dilated only, the presence of fatty degeneration is more difficult of diagnosis by its physical signs. We must then seek to trace how far the usually well-marked signs of hypertrophy of the organ are modified by those we have described as being present in, and characteristic of, fatty degeneration. The same observations will apply to dilatation. The symptoms and signs of fatty degeneration might be confused with like phenomena originating in disordered state of other organs, such as the lungs or liver.

PROGRESS, DURATION, AND TERMINATIONS.—It is impossible to determine the duration of a disease the date of origin of which is in most cases very obscure. Still there are grounds for

believing that persons with a certain amount of degenerated tissue in their hearts have lived for many years. On the other hand, death has occurred from fatty degeneration of the heart, determined *post mortem*, in which the entire absence of symptoms until a few months before the fatal event justified the opinion that the duration of the disease had not much exceeded that period. When fatty degeneration occurs as the result of phosphorus-poisoning or of certain exhausting diseases, the progress of the change, which can then be determined, is rapid. In such cases, the morbid process is not confined to the heart alone, and therefore death, when it occurs, cannot well be attributed solely to the condition of this organ.

Death is frequently sudden, the proportion being as five to one compared with other modes of death from fatty disease of the heart. The immediate modes of death in these cases are those already alluded to, namely, by syncope, angina, coma, and rupture of the heart; the first and last of these contributing nearly the whole number of those who die suddenly.

PROGNOSIS.—In cases where the disease depends on toxic causes, as in phosphorus-poisoning, and in cases where it is of the nature of involution—for example, after parturition—there is good ground for believing that, the cause being removed, the effect will cease, and a fairly healthy condition of the organ be restored. On the other hand, when the coronary arteries are obstructed, or when nutrition generally is impaired, the prognosis must be in the highest degree unfavourable, more especially so if in the latter case the patient cannot be placed in a condition by which this degenerative tendency may be counteracted.

TREATMENT.—The treatment consists in the adoption of all the measures calculated to improve the general health—such as pure fresh air, wholesome food and temperance, together with moderate exercise (either driving, riding, or walking), if it can be accomplished without causing pain or breathlessness. Everything which might tend to lay stress on the heart's action, such as walking uphill or making efforts, or any mental excitement, should be avoided. With reference to drugs, such tonics as can best be tolerated by the patient should be given, especially iron and strychnine. Special attention must be paid to the condition of the excretory organs, such as the kidneys and liver, which are liable to become congested when the cardiac action is feeble. In cases of actual syncope, in addition to the administration of the usual stimulants, galvanism applied from the nape to the præcordia by the interrupted current has in a few instances been useful.

It must also be remembered, with reference to the administration of anæsthetics, that chloroform has an especially depressing effect on the action of the heart; and that when its power is enfeebled by fatty degeneration, a very small quantity of this anæsthetic, which would have little or no effect on a healthy heart, may prove fatal.

RICHARD QUAIN.

J. MITCHELL BRUCE.

HEART, Fibroid Disease of.—**SYNON.** : Chronic Myocarditis.

DEFINITION.—A morbid condition in which some of the muscular fibres of the walls of the heart are replaced by fibrous tissue.

ÆTIOLOGY.—Fibroid disease of the heart is met with most frequently in middle-aged male subjects. The disease is supposed to be occasionally nothing more than an extension between the muscular bundles of a chronic process that has commenced as endocarditis or pericarditis. The cause of this, which is generally rheumatism, is then regarded as the cause of the fibroid growth; but more probably the serous inflammation is the result of the fibroid change. Fibrosis is sometimes the consequence of acute interstitial myocarditis. In a considerable number of cases of fibroid disease, the change is syphilitic. In other instances it is associated with degenerative changes in the vessels, myomalacia cordis, or infarction. A general increase of fibrous tissue in the myocardium may also be the result of prolonged congestion of the coronary veins (*see* HEART, Congestion of). Very frequently no cause can be discovered.

ANATOMICAL CHARACTERS.—Fibroid disease of the heart occurs most frequently in the walls of the ventricles. In rare instances there is a uniform increase of fibrous tissue between the muscular fibres throughout the organ. In other cases the disease appears as a local thickening of the connective tissue underneath an opacity of the endocardium or of the pericardium, whence septa run outwards or inwards between the muscular bundles. Most frequently, however, it presents the appearance of a *fibrous patch*, generally situated near the apex of the heart, replacing the muscular substance throughout its whole thickness, and, in some cases, even involving a considerable portion of one ventricle. Smaller patches, nodules, scars, and streaks may be found in the deeper parts of the myocardium. The apices of the papillary muscles, again, may become fibroid, especially in chronic valvular disease. Polypoid *tumours*, composed of fibrous tissue, have been met with on the endocardial surface of the heart, more especially projecting from the wall of the left auricle. Possibly the detachment of such a polypus may be one mode of origin of the 'fibrinous balls' or 'concretions' occasionally found lying free in the auricular cavities. Fibroid formations due to *syphilis* are described in the article on HEART, Syphilis of.

Microscopically the appearances are seen to be due to the presence of dense fibrous tissue. *See* FIBROSIS.

If a large portion of the wall of one cavity is fibrotic, irregular patchy dilatation of the chamber ensues. Localised fibrosis, especially if it commences beneath the endocardium, gives rise to chronic aneurysm of the heart, by the yielding of the diseased area to the intra-ventricular pressure (*see* HEART, Aneurysm of). Deeper or more limited patches or lines of cirrhosis cause irregularity or puckering of the cardiac walls; and valvular insufficiency may result from this, or from fibrosis and functional disturbance of the papillary muscles. Lastly, fibroid disease occasionally involves the conus arteriosus in an annular form, giving rise to constriction and the formation of so-called 'cardiac stenosis.'

SYMPTOMS.—The symptoms of fibroid disease of the heart vary greatly. When the fibrosis is very limited, few symptoms are present. In a majority of the cases in which a considerable portion of the cardiac wall has been found diseased, the symptoms were described as those of failure or dilatation of the heart, namely, dyspnoea on exertion; præ-

cordial pain or distress; occasional palpitation; a small, weak, or irregular pulse; dropsy; and visceral complications. As a rule, no endocardial murmur had been present; but fibrosis of the papillary muscles had sometimes given rise to signs of incompetence of the auriculo-ventricular valves. *See* also HEART, Aneurysm of; and HEART, Syphilis of.

COURSE AND TERMINATIONS.—The course of fibroid disease of the heart is generally chronic, although urgent symptoms may be observed a short time before death. Attacks of pain, palpitation, and dyspnoea may occur and subside long before the fatal illness. The development of cardiac aneurysm, and its possible terminations, will modify the course of the disease. Sudden death may occur, with or without previous cardiac symptoms, and must be regarded as a special mode of termination of fibroid disease of the heart. Otherwise the cases generally end by pulmonary complications, dropsy, and exhaustion.

DIAGNOSIS.—Fibroid disease of the myocardium has to be diagnosed from chronic valvular disease; from enlargements due to extracardiac causes, such as renal disease, gout, or emphysema; and from fatty degeneration. Its existence is suggested by a history or other evidence of syphilis. Under all circumstances, an accurate diagnosis is extremely difficult, if not impossible. The presence of a murmur does not exclude fibrosis, as the valves may become secondarily involved; and valvular disease is not always attended by a murmur. The other cardiac lesions mentioned must be excluded in the ordinary way.

PROGNOSIS.—When fibroid disease of the heart is accompanied by symptoms sufficient to establish a diagnosis, the prognosis is unfavourable, although it is not necessarily rapidly fatal.

TREATMENT.—This consists in relieving and supporting the heart by every possible means, especially rest and cardiac stimulants, such as alcohol and ether. Potassium iodide may be given with benefit in some cases, particularly if there be a history of syphilis. Digitalis will have to be administered with great circumspection.

J. MITCHELL BRUCE.

HEART, Functional Disorders of.—**DEFINITION.**—A disturbance in the functions of the heart, with or without pain, having origin in causes other than inflammation or organic diseases of the heart itself.

Functional disorders may co-exist with organic disease, but only functional disorder, uncomplicated with organic lesion, will be considered in this article.

ÆTIOLOGY.—To estimate the *immediate* or *proximate* cause of functional disturbance of the heart, regard must be had to its structure, innervation, and blood-supply. Weak muscular fibre, apart from degenerations by disease, becomes a predisposing cause of feeble and irregular action. Interference with the functions of the cardiac ganglia, the vagus nerve, and the nerves and ganglia of the sympathetic system may so modify the action of the heart as to cause deficiencies of power of every variety and extent. Any abnormal condition of the blood-supply, whether in quantity or quality, shows itself by disturbance in the functions of the heart. The causes of functional disorder may be classified thus: (1) Those conditions acting through or upon the nervous system, such as the general exhaustion of

the nervous system; all forms of reflex irritation arising from dyspepsia or diseases of lungs, liver, kidneys, uterus, and other viscera; venereal excesses; mental excitement, shock, or distress; protracted, violent, and unusual physical exertion, especially cycling, running, and rowing; acute fevers; chronic diarrhoea; various articles of diet, as tea and coffee; alcohol and tobacco in excess; many medicines, as aconite, belladonna, and digitalis; fasting and prolonged exposure to cold; and injuries, notably blows on the epigastrium. (2) Those conditions acting upon the general blood-supply of the body, and consequently affecting the special blood-supply of the heart, such as the turgid and plethoric states of gross feeders, depraved states caused by bad and deficient diet, and all forms of blood-disorder, as anaemia, gout, and scurvy.

Females are on the whole more subject to functional disorders of the heart than males. These disorders most usually occur between puberty and the age of thirty, but they may be met with at any period of life.

SYMPTOMS.—In the simplest form of functional disorder there is a morbid consciousness of the heart's action. The patient complains of throbbing, fluttering and palpitation, to which in some cases may be added throbbing of the vessels in the neck, noises in the ears, and flushing of the face. In some of these cases examination may show a healthy and normally acting heart. In other cases, there is rapidity of action with or without irregularity, and to the symptoms mentioned may be added heat and pain in the head, with a sensation of a whizzing, or rushing upwards of the sounds of the heart; dimness of vision, with photophobia; and a tendency to syncope, and to clammy perspirations, with cold shivering; and after the attack a large amount of pale urine may be voided. In some rare cases the voluntary muscles, more especially of the lower extremities, may refuse to act, so that the gait becomes tottering, or the patient grasps adjacent objects to steady himself, yet there is neither paralysis nor vertigo. The irregular form of nervous palpitating heart is often associated with anaemia or that general enfeeblement of the body-powers denoted by the term neurasthenia.

Sometimes the principal symptoms are anxiety and lowness of spirits, with mental and bodily incapacity for exertion; flatulent dyspepsia, with cold clammy extremities; anorexia, or perverted appetite; exhaustion, with tendency to faintness; and there may be a sensation of præcordial pain. Males are more subject to this form of functional disorder than females, and it chiefly occurs in persons having an abnormally small and feeble heart, or in whom a state of general nervous debility is superimposed.

When the distinctive feature of functional disorder is rhythmical error, and this is appreciable to the patient, the special symptom is that of extreme anxiety, even to the fear of impending death. Such forms of disorder occur in those having weak hearts without other evidence of disease; in the dyspeptic; in those subject to gout, especially if an attack is impending; and in those whose habits and occupations involve exhaustion of the nervous system.

Functional disorders of the heart are not infrequently associated with pain. This may be confined to the præcordia, especially the apex region or right border of sternum, but, like the pain of true angina pectoris, it often radiates over the left side or to the neck, axilla, and down the left arm. It may

be more or less paroxysmal, sharp, or cutting, or persistent, dull, and heavy. The pain is very frequently associated with flatulence, and is relieved in such cases by the remedies appropriate to this condition. Pain is not proportional to the intensity of the other symptoms. It may be absent altogether when palpitation is extreme, and may be persistent while other symptoms are in abeyance. In some rare cases, pain may be the indication of urgent, or even of fatal, disorder; this is evidenced by the well-known effects of a severe blow in the epigastrium, or of sudden mental shock. These are illustrations of purely functional angina pectoris; but for the most part this symptom has its origin in structural lesion of the heart itself. See *ANGINA PECTORIS*.

PHYSICAL SIGNS.—The physical signs referable to the cardiac action may be conveniently separated into the following groups, although in practice they will be found associated with each other: (1) *Increased or diminished impulse*; (2) *rhythmical disturbance*, with intermissions; (3) *increased or diminished frequency*, the rhythm and force being normal; (4) *haemic murmurs*.

1. An *increased impulse* may be the only evidence of disorder, or, more frequently, it may be complicated by some rhythmical disturbance.

On palpation the impulse is felt in two, three, or more interspaces, and to the untutored hand may suggest the existence of a true fremitus. On auscultation, the sounds are more difficult of appreciation than in simple palpitation; they are loud and clear, and sometimes so exaggerated and pronounced as to be audible both in the mammary and epigastric regions. When there is an unusual amount of irregular functional excitement in systole, there may be occasionally heard, as a passing, but not persistent occurrence, a reduplication of the second sound.

In cases where the *impulse is diminished*, the special characters are somewhat negative; the sounds being feeble, but otherwise normal, except in those cases in which some irregularity of action, or sharpness of the sounds, is present.

2. *Errors in rhythmical action* may be classified under the heads of irregularity and intermittence. The irregularity may be in the force or in the frequency of the beats. Every variety of irregular frequency may occur. Sometimes there appears to be a kind of order in rhythmical disorders—that is, short series of varying irregularities may regularly succeed each other. True intermittence is not so frequent as irregularity; but when intermittence does occur, it is generally associated with irregularity. These disturbances may be only momentary or of long duration, slight or considerable; but, however this may be, their character is determined by the irregularity in the recurrence of the systole, and the consequent prolongation or retardation of the period of intermission in the systole.

The physical examination reveals the nature of the irregularity. The sounds generally are intensified, the first sound being sometimes heightened to the extent of a sharp knock; while the second, save in intensity, is not materially altered.

Occasionally there is met with an appreciable rhythmical disturbance in the pulse, which is not found to exist in the heart—*false intermittence*. The heart only indicates irregularity of power; and as there is occasional failure of force in the already weak systolic contractions, the impulse is not communicated to the artery at the wrist. These false

intermissions accompanying irregularity most frequently occur when the heart is oppressed by flatus in the neighbouring viscera, or is excited by the injudicious use of tea or tobacco. The sensation communicated to the patient is that of a disagreeable flutter or 'tumbling over' of the heart.

Rhythmical irregularity occasionally appears as a normal condition, having a lifelong existence. Some cases are marked by an extension of the pause, with unsteadiness of the systolic impulse; others by its apparent extinction, so that there exists a rapidity of beats defying all analysis—tachycardia; but tachycardia also occurs either as an habitual state, or in paroxysms. See TACHYCARDIA.

3. Functional disorder occasionally assumes the form of either *increased or diminished frequency*, while the force and rhythm remain normal. Each of these conditions may be congenital and proper to the individual, or may be the result of abnormal influences. The functionally fast beat is generally induced by other diseases, as fever, diabetes, tuberculosis, &c., and is indicative of injury to normal innervation. The slow and drawing beat is generally met with where the nerve-power is healthy, but the heart itself is weak or fatty; or there is a perverted innervation under the influence of digitalis, aconite, or injury to the ganglionic system—a blow in the epigastrium offering a familiar example. See BRADYCARDIA.

4. *Inorganic murmurs* are frequently heard in functional disorders of the heart, but especially in those cases of hæmic disorder where the systolic impulse is increased, and there is rhythmical irregularity. These hæmic murmurs have the special characters usually attached to such sounds. They are systolic, basic, loudest in the second left intercostal space, and are not conducted in the course of the great vessels. There is, as a rule, no apex-murmur; but at the apex, synchronously with the basic murmur, the first sound is clearly defined, with a loud ringing second sound. The murmurs vary in character: sometimes they are hard and rough, sometimes soft and blowing. The seat is in the pulmonary artery, and, as a rule, they are accompanied with palpitation: this palpitation may be persistent, though the murmurs are not. The murmurs are generally associated with the venous hum to be heard in the jugular veins. Sometimes in hæmic cases a peculiar scratchy sound is present to the left of the sternum simulating pericardial friction, and giving rise to the erroneous diagnosis of pericarditis. See PERICARDIUM, Diseases of.

In other cases, functional murmurs are of exocardial origin, and to some of these the term *cardio-pulmonary* has been applied. Cardio-pulmonary murmurs are generally short, whiffing, systolic murmurs, audible only at or near the apex when the heart's action is excited. They are usually more marked at the end of inspiration when the lung is distended, and are weaker or disappear during expiration. They are probably due to the sudden compression of the lung by the forcible cardiac impulse. Their limited and localised position, their association with excited cardiac action, and their varying intensity with the phases of respiration are important points in their diagnosis. Apical systolic murmurs are also met with when the heart is displaced by pleural effusion, by pulmonary disease, or by thoracic deformities. The exact mode of production of these murmurs, which may be called *displacement murmurs*, is

uncertain. Systolic murmurs of peculiar cavernous quality may be audible over pulmonary cavities when these are in close anatomical relation with the heart.

COMPLICATIONS AND SEQUELÆ.—The several functional disorders of the heart are often complicated with other diseases—many external to the heart, and some of the heart itself. The more prominent of the former are disorders of the nervous system and of the blood. The diseases of the heart with which functional disorder may be complicated are mainly degenerations of the walls and valvular lesions. In all these cases the amount and urgency of the functional disturbance are no indication of the urgency of the disease with which it may be complicated.

DIAGNOSIS.—In order to make a correct diagnosis, the first consideration is to ascertain the entire absence of structural disease; or, should it be present, whether it be adequate to cause the full amount of the symptoms exhibited. If the heart have its normal position and dimensions, and if the sounds, though weak, be natural in tone and quality, it may be concluded that the cause is functional only, in the absence of any abnormal physical disease. The same may be said of rhythmical irregularities, except when arising for the first time in an old person. When the symptoms are associated with hæmic murmurs, the character of the murmur, its seat, and its persistency must be considered in connection with the absence or the presence of blood-disease. It must be always borne in mind that the absence of the physical signs of disease is not always conclusive of there being no structural lesion, for lesions do not always yield evidence of their existence. In doubtful cases, repeated examination will generally lead the experienced observer to a correct diagnosis.

PROGNOSIS.—The prognosis of functional disorders of the heart, for the most part, is favourable. Functional rhythmical irregularity, as a rule, does not indicate danger, but it may do so if associated with some obscure structural lesion. Nevertheless, cases of simple functional disorder, so severe as apparently to indicate an immediately fatal termination, prove, for the most part, manageable, and result in a restoration to health. The freedom of the heart from all agitation and other indications of disease, before and after an attack, is due to its being a normal and uninjured organ; and though liable during an attack to the morbid influences of spasms and congestive loading, it still may be a healthy organ. This holds whether the periods of disorder be short or prolonged, occurring rarely or frequently recurring. They are distressing but not dangerous. Irregular action may persist for years, and, in some cases, has lasted from early life to extreme old age.

TREATMENT.—The treatment of functional disorders of the heart should have reference primarily to the removal or cure of the exciting causes and, secondly, to the mitigation of the symptoms.

Treatment during the paroxysms.—In cases of simple palpitation the remedies which usually prove most successful are antispasmodics and carminatives. Among these may be mentioned sal volatile, spirit of ether, foetid spirit of ammonia, compound tincture of lavender, compound spirit of horse-radish, tincture of sumbul, and peppermint. Digitalis is recommended by many authorities for the relief not only of ordinary palpitation, but also of paroxysmal

tachycardia. In many cases of functional rapidity or irregularity it proves useless. Belladonna will sometimes relieve when digitalis fails, especially in cases of rapid and intermittent cardiac action. It may either be used internally in ten-minim doses of the tincture, or applied externally to the præcordial region in the form of plaster, or liniment sprinkled on impermeable piline. In cases where sleeplessness and distress accompany the rapid action, opium sometimes proves more useful than any other remedy; but it is a drug which must be used with caution. Where there are signs of failing heart, strychnine and caffeine may be given either by the mouth or hypodermically. In cases where the functional disorder arises from indiscretions in food and drink, a dose of calomel or blue-pill followed by a saline aperient is indicated. It is hardly necessary to say that in the more severe cases the patient must be kept in bed at rest in the horizontal position. Alcohol, although useful in exceptional cases, is a dangerous remedy. If it relieves, the patient is apt to fly to it on the appearance of the slightest signs of heart-disorder; and we have met with cases where the effects of alcoholism had become far more serious than the original disorder for which the stimulant had been prescribed.

Treatment between the paroxysms.—The paroxysm being allayed, it is then well to correct any disordered condition which may probably have been its exciting cause. Dyspepsia is to be relieved, the liver is to be set right, the uterine functions are to be restored to regularity, loaded bowels emptied, plethora subdued, anemia cured, exhaustion compensated for, and debility counteracted. The overworked must seek renovation in travel and cheerful recreation. In persons prone to these disorders many precautions should be observed. The food should be moderate in quantity and unstimulating in quality; tea, coffee, alcohol, and tobacco should be eschewed; and exercise should be unfatiguing, and chiefly taken in the open air. Hot, crowded, and ill-ventilated rooms, and the postponement of sleep by late hours, should be especially avoided. A careful mental discipline should be observed; and this must be sought in a healthy exercise of the brain. As all the varieties of functional disorder of the heart are peculiarly under the influence of a morbid will, it is important that the medical attendant should generally encourage and cheer; and as soon as careful investigation has satisfied the requirements of a just prognosis, further investigations by the stethoscope should be avoided.

THOMAS SHAPTEL.

HECTOR MACKENZIE.

HEART, Granular Degeneration of.—

SYNON.: Parenchymatous Degeneration; Cloudy Swelling.

ÆTIOLOGY AND MORBID ANATOMY.—See DEGENERATION.

SYMPTOMS.—Inasmuch as parenchymatous degeneration of the heart is usually but a complication of some acute specific disease, the condition of the patient is one of great febrile prostration with cardiac asthenia. The physical signs, which are regarded as more distinct evidence than the symptoms of the condition of the heart, include feebleness, advancing to complete absence, of the apical impulse, or more rarely palpitation; and progressive weakening, and finally loss, of the first sound. The pulse corresponds with the feeble condition of

the heart; indeed, in some cases it is imperceptible although associated with cardiac palpitation.

PROGNOSIS.—The existence of granular degeneration of the heart adds seriously to the gravity of a case of fever. The danger increases with the rate and weakness of the pulse, and with the feebleness of the cardiac impulse and first sound. The re-appearance of the first sound under observation justifies a favourable prognosis.

TREATMENT.—The existence of this condition is to be regarded as an important indication for the use of alcoholic and other cardiac stimulants.

J. MITCHELL BRUCE.

HEART, Hæmorrhage into the Walls of.—

SYNON.: Cardiac Apoplexy.

ÆTIOLOGY AND PATHOLOGY.—Rupture of the heart is the most frequent origin of hæmorrhage into the walls of the organ. The blood in these cases may be derived either from the cavity of the ventricle, or, more rarely, from a coronary vessel ('cardiac apoplexy'). In both classes fatty degeneration of the muscular fibres is generally present. The coronary arteries are also a source of hæmorrhage in rupture of a coronary aneurysm, in ulceration of their walls, and in embolism or thrombosis leading to infarction. The formation of acute aneurysm of the heart is necessarily attended by hæmorrhage (see HEART, Aneurysm of). Ecchymoses in the walls of the heart are met with in granular degeneration of the heart; in fatty degeneration due to poisoning by phosphorus or arsenic, in the so-called 'hæmorrhagic' diseases, such as purpura and scurvy, and especially in cardiac and pulmonary disease.

In non-fatal hæmorrhage into the walls of the heart, the blood undergoes the changes usual in extravasations, and gives rise to collections of pigment-particles between the muscular fibres, to blood-cysts, or to collections of puriform matter. See EXTRAVASATION.

Hæmorrhage into the myocardium possesses no special clinical relations.

J. MITCHELL BRUCE.

HEART, Hydatid disease of.—This is a rare disease; yet, according to Cobbold, 3·5 per cent. of all cases of hydatids in man occur in the heart.

ANATOMICAL CHARACTERS.—Hydatid cysts of the heart are either simple or multiple, the latter being the more common. They are situated in the myocardium of either side of the heart, and project into the pericardial sac or one of the cavities in the form of a cyst. In other cases the parasite undergoes degeneration and remains quiescent, its existence being unsuspected. Hydatids may rupture or be dislodged from their seat in the cardiac wall—either inwards or outwards or in both directions at once. In the first event, the parasite or its contents or fragments become impacted in the cardiac cavities or orifices, or give rise to embolism of the great vessels or of a distant artery. Rupture into the pericardial sac causes pericarditis; rupture both internally and externally has given rise to hæmato-pericardium.

Hydatids of the heart are frequently associated with the same disease in other viscera. See ENTOZOA.

SYMPTOMS.—In several cases of this disease, the subjects have died suddenly. These persons were not known to have suffered previously from sym-

ptoms referable to the heart. In other instances the ordinary phenomena of chronic cardiac disease were present, including endocardial murmurs; but it is not certain that these were in every case due to the presence of the hydatids in the heart. A sudden fatal termination will result from internal rupture and embolism, or from hæmato-pericardium. The disease does not appear ever to have been suspected during life. Cardiac symptoms and signs, or sudden death, occurring in an individual known to be suffering from hydatids of other viscera, would suggest that the heart was possibly also affected.

The disease cannot be said to have any special interest therapeutically.

J. MITCHELL BRUCE.

HEART, Hypertrophy of.—SYNON.: Fr. *Hypertrophie du Cœur*; Ger. *Hypertrophie des Herzens*.

DEFINITION.—An excessive uniform growth of the tissues of which the heart is composed, the result of which is an abnormal increase in the weight and bulk of the organ.

ANATOMICAL CHARACTERS.—The most important anatomical change in cardiac hypertrophy is an increase of the proper muscular tissue of the heart. There may be hypertrophy of only one part of the cardiac walls, abnormal thinning being found in other parts. The organ may be greatly enlarged from general dilatation, without any notable thickening of the walls; but the capacity of the chambers, and especially the gross weight of the organ, should always be carefully noted in estimating the degree of hypertrophy, as there may be a greatly augmented extent of wall, although its actual thickness seems normal. Simple hypertrophy is very frequently the first condition, preceding hypertrophy with dilatation. See HEART, Dilatation of.

The increase of weight and bulk may be due to (1) augmentation of the size of the muscular fibrillæ, which may become more than double that of the normal; (2) the formation of new muscular fibres; (3) increased development of blood-vessels; (4) augmentation of connective tissue; and (5) fatty accumulation. In the course of time the hypertrophied tissue may unduly press upon the blood-vessels and contribute to induce degeneration, while the continuance or increase of the primary cause of obstruction may lead to dilatation of the cavity (auricle or ventricle), the work of which is obstructed.

GENERAL CONSIDERATIONS.—The weight of the heart of the infant at birth becomes more than doubled at the age of two years. Between seven and fourteen years of age it is about eight times the weight at birth; from fourteen to twenty the increase is such that the weight is again nearly doubled. There is, speaking generally, progressive augmentation even to old age, due more to the augmentation in the weight of the great vessels within the pericardium than to that of the heart-substance itself.

The average weight of the female being less than that of the male, the heart of the former is of course less. It will be readily seen that, as the weight of the heart varies with that of the individual, one cannot express with exactitude any figure as the normal weight. We may roughly say that the heart of the adult should weigh about 10 oz. One that weighs 12 oz. is hypertrophied. The weight of an hypertrophied heart may vary between this and the

enormous figure of 60 oz., which has been recorded; not uncommonly it reaches 27 to 30 oz.

VARIETIES OF HYPERTROPHY.—Bertin in 1811 described three forms, which most succeeding writers have referred to, namely: (1) *simple hypertrophy*, in which the parietes of the compartments are thickened, the cavities retaining their natural dimensions; (2) *hypertrophy with dilatation* (*eccentric hypertrophy*), in which the cavities are increased in capacity, while the parietes are either of natural or of augmented thickness; (3) the so-called *concentric hypertrophy*, or *hypertrophy with diminished cavities*, in which new material was supposed to be added, chiefly in the interior of the ventricular walls. It is, however, now generally agreed that *concentric hypertrophy* of the ventricles has no real existence. The appearances which have given origin to the term are due, in the great majority of instances, to the occurrence of *rigor mortis* in the muscle of the ventricle, which has caused firm contraction, and thus diminution, to almost obliteration, of the cavity. If a heart presenting such signs be soaked for some hours in warm water a gentle dilatation with the finger will restore the ventricle to its normal dimensions. The left ventricle may simulate concentric hypertrophy in some cases of mitral stenosis when the disease has developed in early childhood. The ventricle has not received its normal blood-contents, and development has proceeded disproportionately in the right chambers and the left auricle. The cavity of the left ventricle has thus been left relatively small till the time of death. In like manner, in congenital stenosis of the pulmonary artery the *right* ventricle, hypertrophied in intra-uterine life, may so continue, and its cavity never become dilated to the normal extent. The ventricles are much more frequently hypertrophied than the auricles, and the left ventricle more frequently than the right.

I. Hypertrophy of the left ventricle.—**ANATOMICAL CHARACTERS.**—The whole heart appears to be elongated, and the apex-portion of the left ventricle especially prolonged. The walls of the left ventricle and the inter-ventricular septum are thicker and firmer than in the normal; the septum may bulge into the right ventricle, diminishing its capacity and even producing actual obstruction of its *conus* (Coats). In transverse section the thickening and bulging of the septum are very manifest, the right ventricular cavity presenting the appearance of a crescentic appendage to the left chamber.

ÆTIOLOGY AND PATHOLOGY.—Hypertrophy of the left ventricle occurs under many different conditions.

1. Quain attached considerable importance to such *nervous* causes as prolonged mental excitement or strain, and those emotional conditions which produce frequent palpitation (see HEART, Functional Disorders of). It is, however, only in a small proportion of cases that a heart which beats for long periods at an abnormal rate of rapidity becomes hypertrophied. The correct view seems to be that for the production of hypertrophy of the left ventricle in cases of nervous excitation there must be an additional factor besides rapidity—either a thickening of the arteries, or a vaso-motor irritation causing contraction of their muscular walls, whereby the intra-arterial tension is increased. If the blood-pressure be at or below the normal, the rapidly beating heart does not tend to hypertrophy.

2. Among *mechanical* or *physical* causes are all those obstructive conditions to be specially examined

presently. Violent and long-continued athletic or other exercises, which notably accelerate the contractions of the heart, or produce excessive blood-pressure, may be mentioned here. It has been pointed out that great muscular exertion with the arms is specially prone to cause hypertrophy, as in the case of hammer-men. Prolonged working in a bent or constrained position is also mentioned as a cause.

In some callings and conditions in which muscular effort is severe, hypertrophy of the left ventricle is prone to occur—e.g. dwellers in hilly regions, especially those accustomed to carry heavy loads (Münzinger, Allbutt), stonemasons, slaughterers (Leyden). In many cases it is probable that the two factors just mentioned—nervous excitation and muscular overstrain—combine to produce the hypertrophy. So may be best explained the signs and symptoms in the cases of ‘irritable heart’ of soldiers during the American Civil War (Da Costa), those in our own Foot Guards who carried heavy accoutrements (Myers), and those in the Franco-German war (Fraentzel) (*see EXERCISE*). The excitement of a soldier’s life co-operates as a cause with the muscular effort which may be excessive and either prolonged or violent and fitful. It is a significant observation of Hirsch that there has been a notable increase in the proportion of heart-diseases in a community at times of great political and social excitement.

3. *Diseases of the valvular apparatus*—aortic and mitral—induce hypertrophy of the left ventricle by causing an obstruction to the outflow of blood, or an abnormal blood-pressure within the cavity from regurgitation. (a) *Stenosis of the aortic orifice* is a common cause of hypertrophy of the left ventricle. The opening is not only narrowed, but is also rendered more rigid, and thus increased force is necessary to propel the blood. Along with the valvular lesion there may also be, especially in advanced life, a loss of elasticity and a roughening of the inner coat of the aorta from degenerative changes—conditions which increase the mechanical strain upon the left ventricle. (b) *Aortic regurgitation* often induces so great enlargement of the left ventricle, from hypertrophy and dilatation, that the heart in such cases merits the name *cor bovinum*. The back-flow of blood increases the intra-ventricular pressure, tends to dilate the cavity, and calls forth augmented efforts of ventricular contraction. In some cases of injury and rupture of the aortic valves it has been possible approximately to estimate the rate of hypertrophy. In two cases, related by Stone, of injury to these valves by blows upon the chest, assuming that the heart was healthy previous to the injury, there must have been an increase in weight of nearly an ounce a week during the four or five weeks that elapsed before death. (c) *Mitral regurgitation*. Insufficiency of the valve at the left auriculo-ventricular orifice of necessity causes the left ventricle to be subject to abnormal pressure, with the result that it becomes hypertrophied as well as dilated. In pure *Mitral Stenosis*, on the other hand, the left ventricle is of normal dimensions or is smaller than the normal. If the left ventricle at any *post-mortem* examination in cases of this disease be seen to be hypertrophied, there is some superadded cause for such hypertrophy—e.g. arteriosclerosis, or pericardial adhesions.

4. *Aneurysm of the aorta*.—There has been much difference of opinion on the question whether aneurysm of the thoracic aorta is a cause of hyper-

trophy of the left ventricle. The divergence is capable of explanation. When the situation of the aneurysm is at the commencement of the aorta, where it involves the semilunar valves, hypertrophy is the rule. When the aneurysm is so situated as not to involve the valves, hypertrophy is a rare exception. This is proved to demonstration by the following figures, deduced from the records of the London Hospital. Of forty-one cases of aneurysm of the thoracic aorta in which the valves were *not* implicated, enlargement of the left ventricle was evidenced in only nine instances, and in three of these the hypertrophy was recorded as only slight. In forty-one cases in which the valves were *involved*, hypertrophy and dilatation were manifested in thirty-five. The writer has seen *post mortem* many cases in which, though a huge aneurysmal sac was observed on the thoracic aorta well above the valves, the heart was small.

5. *Renal disease*.—Hypertrophy of the left ventricle may occur in several forms of renal disease. Of these the ‘granular contracted kidney’ is a very important cause, the hypertrophy being often of the purest type, without dilatation. This change is the result of the great increase of blood-tension produced by the resistance offered to the blood in the small arteries—probably also in the capillaries—throughout the whole body, as well as in the kidneys.

6. *Pregnancy*.—It has been shown by many observers that the left ventricle tends to become hypertrophied and the arterial tension augmented during pregnancy. The hypertrophy may continue for a considerable portion of the period of suckling, and then the heart, like the uterus, may undergo a process of involution. According to Duroziez, the ventricle tends, after many pregnancies, to remain hypertrophied.

PHYSICAL SIGNS OF HYPERTROPHY OF THE LEFT VENTRICLE.—In simple hypertrophy (e.g. when occurring in athletic persons, or in relation with chronic renal disease in the earlier periods) the wall of the chest on *inspection* may be observed in the neighbourhood of the apex to be raised with each ventricular systole. Pulsation, also, of the carotid arteries in the neck may be observed, such pulsation being rather prolonged, and not sudden (as in aortic regurgitation), unless there be concurrent fever. On *palpation* the fingers are lifted by the apex, the impulse of which is prolonged and heaving. The duration of the heaving impulse is in proportion to the degree of hypertrophy. The impulse is often strong enough to move the bed-clothes visibly, and even to raise the head of the auscultator by the impact against the stethoscope. Of course a larger portion than usual of the heart’s surface impinges against the chest-wall. The outline of the apex-portion of the heart, as obtained by *percussion*, is acutely triangular, its acute angle pointing downwards and to the left. This becomes broader and more rounded as dilatation preponderates over hypertrophy. In simple hypertrophy the heaving apex may be in the sixth or seventh, rarely in the eighth, left intercostal space; but in dilatation with hypertrophy the area downwards and towards the left axilla is greatly increased. On *auscultation* the first sound at the apex is found to be dull and prolonged. It is the sound of muscular contraction that the ear in the greatest degree appreciates, the element of valve-tension being muffled by the thick wall of the ventricle. Over

the second right costal cartilage the second sound is loud, and may have a ringing character; it may sometimes be the only one of the two normal sounds audible. In this situation it is much louder than over the second left interspace (the commencement of the pulmonary artery) unless the ventricular hypertrophy is accompanied by mitral insufficiency. If, with evidence of powerful left ventricle and firm arteries, the aortic second sound is not loud, but dull and ill-pronounced, there is strong probability that the aortic cusps are swollen or thickened from disease. In later stages of hypertrophy, when the muscular tone of the left ventricle is impaired, the sounds of the heart may have a triple rhythm, resembling the cantering of a horse—the *bruit de galop*. This is especially observed in the hypertrophy associated with renal disease, and in the hypertrophy with dilatation accompanying gastro-hepatic derangement.

The *radial pulse* is felt to be prolonged; the artery is firm and resisting, and its coats may be thickened. In the hypertrophy resulting from muscular overstrain the pulse is large and full, the first wave is prolonged, and there is no diastolic. In chronic renal disease, as the arterial coats become thickened the amplitude of the pulse is manifested in less degree, the vessel being felt hard, resisting, and full between the beats. In some cases the fullest force exerted by the finger may fail to obliterate the pulse entirely, and on the distal side of the point compressed the wall of the artery may be felt cylindrical and firm. In the hypertrophy associated with aortic stenosis the pulse is somewhat small and gradual both in its rise and fall. In that manifested with aortic incompetency it is sudden and powerful in its impact against the finger, but rapidly receding, so that the artery seems empty between the beats—it is the collapsing or 'water-hammer' pulse.

SYMPTOMS OF CARDIAC HYPERTROPHY.—It must not be forgotten that simple hypertrophy may exist without producing symptoms which attract the attention of the patient, and that there is a natural tendency to some degree of cardiac hypertrophy with the advance of age. *Dyspnœa*.—In moderate hypertrophy without complication, there is usually easy and natural breathing when the patient's body and mind are at rest. But mental excitement or bodily effort at once induces more or less of temporary dyspnœa. In some cases the due expansion of the lungs may be mechanically impeded by the increased volume of the heart. In eccentric hypertrophy with dilatation, more especially when the right cavities are thus affected, pulmonary congestion and œdema are usually present, and then marked dyspnœa is a prominent and distressing symptom. *Cough*.—In simple hypertrophy there may be an occasional dry, irritating cough, and in young plethoric women a wheezing cough may be complained of. In right-side enlargements, when pulmonary obstruction and dropsical effusions supervene, cough is, in most cases, a very frequent and painful addition to the other sources of discomfort to the patient. *Hæmoptysis and other hæmorrhages*.—Hæmoptysis from capillary engorgement is common, being generally active and sudden. In hypertrophy of the left ventricle there is often active distension, and sometimes rupture, of the branches of the bronchial arteries. The cerebral arteries are also specially liable to give way. Epistaxis may also be due to cardiac enlargement. *Palpitation* is a

common symptom in all organic diseases of the heart, and is often very marked in cardiac enlargements. The least excitement, bodily or mental, may induce a greater or less degree of this symptom. Especially in hypertrophy with dilatation, most distressing paroxysms of palpitation are apt to occur from time to time. Besides bodily and mental excitement, other conditions, such as indigestion, flatulence, or an overloaded stomach, readily call forth this symptom. When there is much dilatation, the palpitation may be irregular and intermittent, and is then more particularly a very alarming symptom. It must be remembered, however, that palpitation of the heart often occurs in the absence of any structural heart-disease.

The *renal and urinary* derangements are very important. In the hypertrophy of the left ventricle, associated with chronic Bright's disease, there will be found the copious excretion of a pale urine of low specific gravity. Albumen may be found in small quantity, and may sometimes be absent. In the hypertrophy associated with gastro-hepatic disturbance, the urine will be scanty, of high specific gravity, often thick with urates, containing much urea, and dark with the colouring-matter of the urine, of the bile, or of both.

Certain *cerebral symptoms* ought to be mentioned in connection with the other more direct signs of cardiac hypertrophy. A feeling of fullness, or perhaps of throbbing, may be felt in the head after great muscular exertion or mental excitement. In many cases there may be headache, ringing in the ears, vertigo, *muscæ volitantes*, and disturbing dreams.

II. Hypertrophy of the right ventricle.—

The capacity of the right ventricle is generally greater than that of the left—according to recent researches in the proportions of about seven to six. The walls are from one third to one half the thickness of those of the left.

In the great majority of cases the right ventricle becomes hypertrophied only in association with a morbid change in the left, and when such hypertrophy occurs it is nearly always accompanied by dilatation.

The right ventricular wall may be thickened to the extent of half an inch or even an inch, instead of the normal eighth of an inch; its greatest thickness is at its base. Thus the wall of the hypertrophied right ventricle may be as thick as that of the normal left ventricle. The columnæ carneæ of the right ventricle are even more liable to hypertrophy than the wall. In dilatation with hypertrophy the columnæ carneæ become stretched and attenuated. The substance of the hypertrophied left ventricle can generally be torn with ease, while that of the hypertrophied right ventricle is usually tough and leathery.

ÆTIOLOGY AND PATHOLOGY.—The right ventricle may be observed in a state of simple hypertrophy in—

1. *Congenital anomalies of the heart*.—Chief of these is stenosis of the pulmonary artery, which in the majority of cases results from foetal endocarditis. The obstruction may be below the valves, the conus of the right ventricle being narrowed. In each case the right ventricle becomes hypertrophied in consequence of the obstruction in front, and there is no dilatation of the cavity. See HEART, Malformations of.

2. In *emphysema, fibrosis, and consolidated or*

compressed conditions of the lungs, the impediment to the pulmonary circulation induces hypertrophy with dilatation of the right ventricle.

3. In the subjects of *curvature of the spine* the capacity of the thorax is nearly always diminished; the lungs are emphysematous, and often suffer other pathological changes. The two branches of the pulmonary artery may be unequal, owing to the differences of development of the two lungs. The right ventricle is dilated as well as hypertrophied.

4. *Endocarditis affecting the tricuspid valve*.—It is rarely that endocarditis attacks the valvular apparatus of the right side of the heart without that of the left side participating in still greater degree, except in intra-uterine life. In some cases (and these not very infrequent) the result of endocarditis is stenosis of the tricuspid orifice, in which case the right ventricle tends to hypertrophy, probably on account of the co-existing mitral stenosis, while the right auricle becomes greatly dilated.

5. The most common cause of hypertrophy with dilatation of the right ventricle is *valvular disease of the left side of the heart*. Whether there be mitral stenosis or regurgitation, the consequences are hypertrophy of the right ventricle and dilatation on account of the persistent overstrain.

SIGNS AND SYMPTOMS OF HYPERTROPHY OF THE RIGHT VENTRICLE.—In enlargements of this ventricle, *inspection* may reveal a rounded smoothness of the epigastrium, with perhaps some bulging of the ensiform and lower left costal cartilages. The præcordial impulse may be seen to be very diffused, extending towards the tip of the ensiform cartilage. Facial lividity is frequently seen; and jugular pulsation may be observed when there is tricuspid regurgitation. *Palpation* over the lower part of the sternum detects an impulse, which feels as if immediately under the hand, and usually lacks the heaving character of the impulse of an hypertrophied left ventricle. Epigastric pulsation is often very pronounced. The liver-pulsation in such cases may result either from venous regurgitation or from right systolic action exerted through the diaphragm. On *percussion*, the inferior line of dulness is found to extend lower down and farther towards the right than normal, sometimes reaching an inch or more beyond the right sternal edge. On *auscultation*, the second sound over the pulmonary valves is found to be louder than over the aortic. The accentuation of the pulmonary second sound, however, is not so marked as in cases of well-compensated stenosis or insufficiency at the mitral orifice. The aortic second sound is weak, and though palpation may detect a forcible impulse at the apex of the heart, the radial pulse may be felt small and contracted. In some cases a triple sound—the *bruit de galop*—may be heard over the right ventricle.

The *radial pulse* in hypertrophy of the right ventricle is usually small and resistant. There is a difficulty in the passage of blood through the pulmonary circuit, and the supply to the left chambers is reduced in amount; moreover, the blood is imperfectly oxygenated and, containing an undue proportion of carbon dioxide, it stimulates the vaso-motor centre and augments the intra-arterial tension.

III. Hypertrophy of both ventricles.—**COMPLICATIONS AND SEQUELÆ.**—Simple hypertrophy of the heart may go on quietly for a long time, just balancing the obstructive influence, and

giving rise to no other form of disease. But when dilatation co-exists with hypertrophy, palpitation, dyspnoea, venous congestion, and serous effusions are the ordinary results. Diseased conditions of the arteries may occur simultaneously, or may be induced by the long-continued additional strain put upon them by an hypertrophied heart. Cerebral hæmorrhage often occurs in connection with an hypertrophied left ventricle, as in Bright's disease; although there are always perhaps other factors, besides the mere excessive propulsive power of the heart, in the production of apoplexy. Pulmonary and general congestion and oedema are the usual attendants of mitral lesions with right-side enlargements. Pulmonary hæmorrhagic infarction (the so-called 'pulmonary apoplexy') generally results from greatly lowered pressure, due to plugging or other cause, in some branch of the pulmonary artery, and takes place in connection with right-heart enlargement. Sanguineous exudation in the tract of the bronchial mucous membrane may occur in left-heart hypertrophy. Persons suffering from cardiac hypertrophy are apt to be gravely affected by acute febrile diseases, because the resultant acceleration of the heart's action increases the embarrassment of the organ. Hypertrophied cardiac walls are peculiarly liable to the fatty degenerative changes described elsewhere. See HEART, Fatty Degeneration of.

IV. Hypertrophy of the auricles.—Hypertrophy of the *right* auricle is scarcely ever met with except in association with dilatation of the cavity. Simple hypertrophy is only possible in the case of tricuspid stenosis occurring in infant life. Hypertrophy and dilatation are marked in cases of mitral disease, both regurgitant and obstructive. The hypertrophy is in greater degree in the obstructive lesion. In both, but especially the latter, the auricle may be sufficiently powerful to cause a back-flow into the venous channels by its systole, and thus a venous pulsation, presystolic in time, in the veins of the neck. Hypertrophy of the *left* auricle occurs in greatest degree in mitral stenosis. The writer found, in a boy of nine, a left auricle varying in thickness from one eighth to one quarter of an inch—i.e. the thickness in some parts being nearly that of the right ventricle of an adult. In some cases—in children—the hypertrophied auricle may be observed to cause a distinct presystolic pulsation in the third left intercostal space at the outer portion of the cardiac dulness. It has been considered by many observers that the earliest and most characteristic change in the left auricle in mitral stenosis is dilatation. Samway, from a review of the *post-mortem* records of 70 cases at Guy's Hospital, found that in pronounced mitral stenosis the characteristic modification of the left auricle is not dilatation but hypertrophy. In the later stages of the disease dilatation occurs, and the muscular wall becomes thin. An increase of dulness to the left over the situation of the left auricle is often manifested on percussion in cases of mitral stenosis.

V. Hypertrophy of the whole heart.—This is manifested in the highest degree in cases of combined aortic and mitral disease; either when a rheumatic lesion has induced mitral and aortic regurgitation coincidentally, or when the initial lesion has been aortic regurgitation, and mitral inadequacy has resulted from the abnormal intra-ventricular pressure. In mitral disease, however, when the aortic valves are intact, the hypertrophy and dilatation of both ventricles may be very considerable.

Differences of opinion have been expressed as to the influence of *pericardial adhesions* in determining hypertrophy of the heart. Pericarditis is a potent cause of rapid hypertrophy during the period of childhood. A heart presenting signs of almost universal pericardial adhesion will be often found to evidence a far greater degree of hypertrophy than could be ascribed to any co-existing valvular imperfection. When the period of heart-growth has ceased, in fully adult life, the enlargement is less marked.

PATHOLOGY.—True muscular hypertrophy of the heart is compensatory or conservative. The heart obeys the law that muscle tends to increase in force and in development when an obstacle is imposed to its normal working (*see* HYPERTROPHY; and HEART, Valves and Orifices of, Diseases of). We have just seen what the obstacles are in hypertrophy of the different parts of the heart. Some obstacles are direct, such as stenoses; and by hypertrophy the heart is enabled to overcome deficient valvular action or obstruction to the blood-current, the increased peripheral resistance of Bright's disease and of emphysema. Other lesions are indirect or relative obstacles, particularly valvular incompetence, which permits over-filling of the cavities, and demands a corresponding increase of vigour in the systolic discharge, which leads to hypertrophy.

DIAGNOSIS.—An extended area of dullness, displacement of the apex-beat downwards and to the left, and a comparatively slow, heaving systolic action, with augmented force of impulse, are the chief diagnostic physical signs of cardiac hypertrophy. In *young and thin people* the last of these signs may seem to be present, but the accompanying conditions readily exclude hypertrophy, especially the non-extension of the cardiac dullness. An *emphysematous left lung* may mask hypertrophy; and contraction of the lung might suggest its presence to a superficial observer. In *pericardial effusion* the triangular shape of the area of dullness, with the apex of the triangle upwards, is a distinctive feature; there might also be the history of an acute disease, with lancing pain, dyspnoea or suffocative sensations, and other symptoms not found in mere enlargement of the heart. *Pleuritic effusion* or *aneurysm* would be still more readily discriminated. The differential diagnosis between left-heart and right-heart enlargements has been sufficiently discussed in speaking of the symptoms and signs. *Dilatation*, as distinguished from hypertrophy, is characterised by the feebleness and diffuseness of the præcordial impulse, which may even be quite imperceptible; and by the general signs and symptoms of a feeble circulation.

PROGNOSIS.—Simple, uncomplicated hypertrophy, as in the young and in athletes, is not incompatible with long life, if the cause be removed in time. According to the extent and degree of complication, whether in the form of valvular lesions or co-existent pulmonary disease, the prognosis will be unfavourable. When the cardiac change is itself producing secondary lesions, as degeneration of the arterial coats, when dilatation is advancing, and when there is Bright's disease, the prognosis becomes very unfavourable.

TREATMENT.—Hypertrophy being in itself a conservative change, protective from worse results, the primary object is to remove, if possible, the cause of the hypertrophy. To aim merely at reducing the hypertrophy, irrespectively of its cause,

as by lowering the nutrition, would favour the more serious evil of dilatation. All mental and bodily exertion which excites the circulation must be scrupulously avoided. Alcoholic stimulants should be taken sparingly, and no more wine allowed than such as may seem to benefit digestion. The diet should be carefully selected, nitrogenous food being generally necessary, though great care must be exercised to avoid excess in this direction. The digestive organs must be sedulously looked after, not only because good nutrition is very important, but also because flatulence and dyspepsia directly embarrass the heart's action. Mild saline and aloetic aperients with alteratives should be given. Diuretics will be necessary if there is a tendency to dropsy, and in all cases great attention must be paid to the removal of congestion when it affects important organs, and the restoration of their functions when affected, more especially of the liver and the kidneys. When there is great excess of cardiac action, direct cardiac sedatives, as aconite, diluted hydrocyanic acid, conium, and perhaps the local application of ice, may be called for. When there is dilatation or feebleness of the muscular substance along with the hypertrophy, iron and digitalis are the chief remedial drugs. A. E. SANSOM.

HEART, Inflammation of Lining Membrane of.—**SYNON.**: Endocarditis; Fr. *Endocardite*; Ger. *Endocarditis*.

Three forms of endocarditis are commonly described: (1) Simple endocarditis; (2) Ulcerative, or malignant, endocarditis; and (3) Chronic endocarditis. Only the first two will be discussed here. Chronic endocarditis is referred to under the head of HEART, Valves and Orifices of, Diseases of, p. 663.

Simple Endocarditis.—**ÆTIOLOGY.**—Endocarditis generally occurs in association with acute rheumatism and chorea; less frequently with other acute specific febrile diseases, such as scarlet fever, pyæmia, and septicæmia—including puerperal fever; more rarely with typhoid fever, variola, measles, and tonsillitis. It may be observed in the course of pregnancy, and after parturition, in acute and chronic Bright's disease, and in syphilis. Injuries of the heart, such as strain or rupture of the valves, may also lead to it; and local endocarditis is frequently the result of friction of one part of the endocardium on another during the cardiac revolution, as, for example, by growths from the walls or valves.

The cause of endocarditis is an irritant circulating in the blood. In many cases this is known to be an organism; in others it is probably a toxin or some other chemical irritant. See p. 652.

Age is an important predisposing factor in the ætiology of acute endocarditis, the occurrence of which as a complication of acute rheumatism is certainly most frequent in young subjects, and declines as age advances. Women are more subject to rheumatic endocarditis than men.

The localisation of the endocardial inflammation appears to be determined chiefly by pressure and tension, rather than by any peculiarity of the membrane itself, or of the blood in contact with it. Thus the left ventricle is almost the sole seat of the disease in the adult, and the right ventricle in the fœtus; while endocarditis is rarely seen beyond the boundaries of the valves, that is, the parts most subjected to strain. In the same way, chronic endo-

carditis is usually due to increased pressure within the heart, as in chronic Bright's disease, and in conditions that entail prolonged severe strain upon the valves during exertion. A similar cause is at work in pregnancy.

ANATOMICAL CHARACTERS.—Inflammation of the endocardium affects chiefly the valves and the chordæ tendinæ, and especially the lines of contact or the surfaces of the valves exposed to the force of the blood-current. The lines or points of contact of the valves present smooth, soft, warty projections, known as 'vegetations,' due to the formation of smooth thrombi upon small, necrosed, and generally swollen, areas of the endocardium. As the process advances, the affected areas become more opaque and firm, as well as larger. When the endocarditis has gone thus far, resolution is probably rare. The most common result is what is known as 'chronic valvular disease.' The affected parts are left opaque, puckered, and thickened by growth of connective tissue. As a consequence of these changes, the valves may become much altered in size and shape, and the ostia contracted and irregular, so that the mutual adaptation of the parts is greatly disturbed. Other results of endocarditis are not uncommon, such as adhesions between the neighbouring structures, and ossification or calcification of the new connective tissue. Laceration of the valves, rupture of the chordæ tendinæ, ulceration, suppuration, the detachment of vegetations, and the formation of aneurysm are rare events in this variety of the disease.

Microscopically, the formation of laminated thrombi is the most prominent change. These are formed on small areas of endocardium believed to have undergone necrosis, as their nuclei fail to stain. In some cases slight exudation may lead to swelling of the endocardium, and to the appearance of polymorphonuclear leucocytes at the margin of the affected areas.

The ultimate effects of these changes upon the functions of the valves and their appendages are described in the article **HEART, Valves and Orifices of, Diseases of.**

SYMPTOMS.—The symptoms of endocarditis are inseparable from the symptoms of the disease with which it is associated, and the diagnosis of it is made almost entirely from the presence of physical signs. Thus fever probably precedes the advent of endocarditis in every case; and it cannot be said that the simple uncomplicated disease in any respect affects either the pyrexia or any other element of the same. Local symptoms are almost equally rare, unless the endocarditis leads to serious lesion of the cardiac valves. As long as these remain sound, and the disease is acute and does not involve deeper structures, pain in the heart, præcordial distress, syncope, shortness of breath, and other symptoms of heart-disease cannot be said to occur at all frequently in endocarditis. The cardiac contractions are necessarily increased in frequency; and palpitation and dyspnoea may occur on movement. It is otherwise when the inflammation has lasted so long as to render the valves incompetent, or to obstruct the orifices; or when the myocardium is attacked and dilatation ensues. The symptoms just enumerated then make their appearance. In children, rheumatic endocarditis is peculiarly latent, while dangerously progressive or recurrent. See **RHEUMATISM, ACUTE.**

Physical Signs.—The physical signs of acute endocarditis are—increased extent and frequency, with

variable strength, of the visible and palpable impulse; moderate increase in the area of præcordial dullness, and various alterations in the cardiac sounds. At the beginning of endocarditis, the first sound at the (left) apex is frequently heard prolonged and hollow or muffled; and, as the process advances, this alteration of character may gradually pass into a murmur, which is at first indistinct but afterwards well-formed. If the aortic valves are affected, the second sound may similarly lose its characters, become dull, and finally be converted into, or be complicated with, a murmur. The most frequent murmur in acute endocarditis is mitral systolic; aortic murmurs are decidedly less common; and mitral præ systolic murmur is very rare. Various murmurs, inorganic or organic, may also appear and disappear during the course of the disease.

COMPLICATIONS.—Endocarditis is itself always a complication of the diseases previously mentioned. Myocarditis and pericarditis may be correctly regarded as complications of endocarditis when the inflammation begins in the lining membrane of the heart. According to some authorities, clots may form in the heart in endocarditis, and give rise to very urgent symptoms (see **HEART, Thrombosis of**). Embolism may arise from detachment of fragments of coagula or vegetations. Congestion or inflammation of the lungs and pleuræ frequently occurs in association with endocarditis, and so may albuminuria.

COURSE, TERMINATIONS AND SEQUELÆ.—The course of simple endocarditis varies with the course of the original disease with which it is associated, as well as with the complications. If acute rheumatism be quickly checked, inflammation of the endocardium will be also arrested; but the two diseases may recur together. In a considerable number of cases, however, endocarditis results in permanent valvular lesions.

Simple endocarditis is very rarely immediately fatal, but, being by far the most common cause of valvular disease of the heart, it leads indirectly to much suffering, and in such cases ultimately to death.

DIAGNOSIS.—The diagnosis of endocarditis depends upon the discovery of the development of an endocardial *bruit*, due to structural changes, during the course of one of the diseases already named. From functional murmurs the bruits of valvular inflammation may be diagnosed—first, by their locality, which is most frequently the mitral area; secondly, by their time, diastolic or præ systolic murmurs being always structural; and, thirdly, by their association with pericardial friction. The special characters of functional murmurs are described elsewhere (see **HEART, Functional Disorders of**). Chronic valvular disease may be diagnosed from acute endocarditis by the presence of cardiac enlargement; of cardiac failure—especially of pain and dyspnoea; of some special forms of valvular disease—mitral stenosis, for instance, being very rarely an acute lesion; and of visceral complications. Much more difficult of diagnosis is acute endocarditis occurring in the course of chronic valvular disease. Change in the character of the murmur, if this have been previously known, may lead to the suspicion of fresh inflammation, but cannot positively establish the diagnosis of its existence. Acute endocarditis is most frequently overlooked when the primary disease is 'latent'—particularly rheumatism in children.

PROGNOSIS.—The immediate prognosis of acute endocarditis is generally favourable, and may be estimated by the absence of local symptoms. The remote prognosis, on the other hand, as regards both life and health, is bad, inasmuch as endocarditis so frequently ends in chronic valvular disease. A feeble, soft, and smooth murmur is more likely to disappear than a loud, well-defined bruit. The probability of the disappearance of diastolic basic murmurs is very small; it may be best estimated by the absence of the effects produced by aortic incompetence upon the heart and vessels. Murmurs referable to endocarditis disappear more often in young subjects than in adults.

TREATMENT.—The treatment of acute endocarditis has to be discussed under three heads—preventive, immediate, and subsequent.

Preventive treatment.—When a patient is suffering from any disease which may become complicated with endocarditis, every means must be adopted to prevent its occurrence. Thus, in acute rheumatism it is all-important to check at once the intensity of the disease by recourse to one or other of the salicyl-compounds or other means; for experience shows that endocarditis, when it does occur in acute rheumatism, generally makes its appearance within the first week. Again, the duration of the primary disease must be curtailed if possible, inasmuch as endocarditis, although it generally appears early, may possibly occur at any period of the disease. Another point of equal importance in the prevention of endocarditis is diminution of the cardiac activity. We have seen that the pressure within the heart is an important factor in the causation of endocarditis; and this pressure must be reduced by diminishing the work to be done by the heart without lowering the cardiac power. Rest in the recumbent posture must therefore be enforced—an end which, except in children, is usually already secured by the presence of acute rheumatism of the joints. Excitement of every kind has to be strictly interdicted. The personal comfort of the patient must be zealously attended to, and pain relieved, so that restlessness and irritability may be avoided; and for this purpose carefully selected anodynes may be necessary. Stimulants must, if possible, be avoided; the bowels be regularly and fully moved; and the skin be kept moist.

Treatment during an attack.—When endocarditis has actually made its appearance, the various means just insisted upon must be in no way relaxed. Rest, mental as well as physical, is still of the first importance. The medicinal treatment of the original disease—especially of acute rheumatism—must be persevered in. Local applications to the præcordia, such as poultices or leeching in cases of sthenic inflammation, are often of great service. The administration of stimulants will require the greatest care; excitement of the heart, on the one hand, being avoided, and, on the other hand, digitalis, ammonia, or alcohol being employed if symptoms of cardiac distress supervene.

Treatment after an attack.—When the primary disease has subsided, and convalescence has commenced, the physician must not forget the state of the endocardium which is still the seat of active changes directed to the repair of the damaged valves. Instead of urging the patient to sit up and walk about we must recommend a very gradual return to exercise, and the most jealous avoidance of actual exertion. There can be no question that, at

this stage, rest for several weeks is of more importance than medicinal treatment. At the same time various tonic and other remedies should be employed.

Ulcerative Endocarditis.—**SYNON.** : Malignant Endocarditis; Diphtheritic Endocarditis; Fr. *Endocardite Ulcéreuse*; Ger. *Infectiose, Maligne Endocarditis*. This form of endocarditis is invariably due to infection by pathogenic bacteria.

ÆTIOLOGY.—Ulcerative endocarditis occurs most frequently between the ages of twenty and forty, but is not uncommon in children. It is more often observed in females than in males. Previous valvular disease predisposes to it, and consequently the left side of the heart is more frequently affected than the right.

The primary lesion, through which the micro-organisms enter the blood-stream, may be so slight as to escape observation; it may be situated in any portion of the alimentary, respiratory, or genito-urinary tracts, or may be a wound of the skin.

In many instances the endocarditis is but one feature of a general septic or pyæmic condition, and may then be associated with and dependent upon puerperal thrombosis, appendicitis, pyelophlebitis, acute osteomyelitis, otitis media, meningitis, gonorrhœa, lobar pneumonia, or other acute specific disease.

BACTERIOLOGY.—It is only reasonable to suppose that any of the pathogenic bacteria are capable of producing this disease, and in point of fact no less than twelve different micro-organisms have been recorded as associated causally with cases of infective endocarditis. An analysis of cases, however, shows that the most important in point of frequency are the *Streptococcus pyogenes longus*, the *Staphylococcus aureus*, the *Diplococcus pneumoniae*, and the *Gonococcus*; while the *Bacillus tuberculosis*, *Pneumo-bacillus*, *Bacillus pyocyaneus*, and *Bacillus typhosus* are also occasionally the responsible agents.

PATHOLOGY.—In infective, as in simple, endocarditis the pathological changes are limited at first to the endocardium on the margins of the valves at the seats of maximum contact during closure. The endothelial and subendothelial layers at these points are invaded by micro-organisms, which may be derived from the blood in the cardiac cavities themselves, or from the small blood-vessels of a pre-existing valvular lesion. The affected cells undergo necrotic changes, and over the necrosed area a thrombus is deposited, consisting primarily of a layer of blood-platelets, but soon converted into a 'vegetation,' by the addition of leucocytes and fibrin. Active proliferation of the cells of the subendothelial layer now takes place, leucocytes from adjacent blood-vessels infiltrate both necrosed tissue and thrombus, and if the effort at repair is effective the vegetation is gradually replaced by granulation-tissue, and finally converted into fibrous tissue. On the other hand, this cellular infiltration may result in the formation of minute abscesses in the substance of the vegetation, or, if the necrosis is the more active process, erosion and ulceration ensue, followed by perforation or complete disintegration of portions of the valve; and the resulting fragments of degenerated tissue, perhaps conveying micro-organisms, are thrown into the blood-stream to give rise to multiple emboli in distant parts of the body.

ANATOMICAL CHARACTERS.—The appearances

in malignant endocarditis are so far variable as to suggest their grouping into two types of the disease—the *ulcerative* and the *vegetative*.

In the *ulcerative* form, the endocardium presents small areas where the proper tissue has disappeared, leaving behind it shallow ragged erosions. This change occurs most frequently in connection with the mitral valves and opening, including the auricular aspect of the curtains and the line of attachment of the valves—particularly the two commissures of the segments and the anterior aspect of the anterior segment; also the aortic cusps; parts of the septum and general endocardial surface; and the papillary muscles and chordæ tendinæ. The endocardium of the right ventricle is relatively more frequently involved in malignant than in simple endocarditis.

The earliest stage of the morbid process may sometimes be observed as small yellowish opacities of the endocardium. But as a rule the surface is found already eroded, presenting a small, irregular, oval, circular, or linear ulcer; its ragged grey base covered with a loose friable thrombus; its edges fringed with vegetations, swollen, indurated, and undermined. Such an ulcer increases but slowly in area, but it readily advances into the depth of the tissue. A variety of secondary changes may then occur—acute valvular aneurysm, complete perforation of the valves, abundant vegetative or fungating growths, and suppuration, which ends in acute cardiac aneurysm or in complete perforation of the parietes.

The *vegetative* form of malignant endocarditis is readily recognised *post mortem* by a prominent and possibly extensive growth of polypoidal vegetations, in connection usually with the auriculo-ventricular opening, the neighbouring surface of the left auricle, and the auricular aspect of the mitral segments. Less frequently the aortic valves are involved. In either situation the vegetations form a large mass of soft growth, almost choking the mitral or aortic opening, while portions of it must have floated and waved in the blood-current, and by their contact or impact during the cardiac movements have injured the neighbouring or opposed surfaces of the endocardium, there setting up fresh disease of the same kind. The surfaces of these vegetations are sometimes calcified.

In both forms of malignant endocarditis it is obvious that during life portions of the diseased endocardium and thrombi, as well as their fluid products, whether on a small or on a large scale, must have been readily detached or washed off, and carried as emboli into other parts of the circulation.

In addition to the lesions just described, the heart may present a variety of morbid changes. It is in at least 75 per cent. of cases (Sansom) the seat of chronic valvular disease and associated enlargement, sometimes of congenital malformation. The myocardium may be swollen and in a state of granular degeneration like other visceral structures; or it presents a number of small embolic, hemorrhagic, inflamed, or fattily degenerated areas. Pericarditis is sometimes associated.

Embolism is one of the essential accompaniments of ulcerative endocarditis. It occurs most often in the spleen, kidneys, and heart, but may be almost universal. In the vegetative form of the disease the emboli may be so large as to be arrested even in the abdominal aorta; the tibial, brachial, and other vessels of the limbs are often occluded, as well as the splenic, renal, mesenteric, cerebral, and a

variety of arteries in other parts of the body. In the purely ulcerative type of the disease the emboli are much smaller, and their effects are only recognised *post mortem* by infarcts in various conditions of change. These secondary foci often proceed to abscess-formation—suppurative meningitis is fairly common. The skin and mucous membranes, the serous membranes, the vessel-walls, and the retina are also favourite seats of minute embolism and hæmorrhage. The lungs are specially affected in some cases; pulmonary embolism and infarction accompany ulcerative endocarditis of the right chambers; in other instances acute pneumonia appears to be the primary lesion, in which the endocarditis of the left chambers of the heart has originated. The local effects of infective embolism occur very extensively in ulcerative endocarditis, namely, hæmorrhage, softening, and acute aneurysm ending in fatal rupture. See EMBOLISM.

SYMPTOMS.—In its clinical characters malignant endocarditis is either (a) an *acute*, severe, and fatal malady with markedly typhoidal features; or (b) a disease of *sub-acute*, *possibly chronic*, course, pyæmic in character, occasionally ending in recovery. These two forms call for separate description.

(a) **Acute Typhoidal Malignant Endocarditis.**—In this variety of the disease, the patient is seized with a rigor when apparently in good health, or during convalescence from an acute illness—particularly rheumatism; in other instances the invasion is more gradual. Pyrexia and the other phenomena of severe fever are quickly developed, and the patient complains of malaise, debility, and cardiac distress; but for the first two days the medical attendant probably fails to make a complete diagnosis. By the end of this time, the patient's condition having become steadily worse, the cardiac sounds are found to be impure; they shortly become murmurish; and probably on the next day an endocardial murmur is developed, systolic or diastolic, still changeful in its characters. The general symptoms now begin to assume a typhoid type. The patient lies helplessly on his back; the temperature rises to 104°; the tongue, at first furred, becomes dry and brown; sordes form; the respiration becomes hurried, possibly with thoracic pains and hæmorrhagic sputa; and somnolence sets in, broken by low muttering delirium, jactitation, and subsultus. The spleen is enlarged and tender, suggesting infarction. The bowels may be relaxed. The skin is visited by heavy sweats, and may become covered with a peculiar petechial eruption. The urine, passed naturally or incontinently, or withdrawn with the catheter, is found to contain blood, albumen, and occasionally micro-organisms. The ophthalmoscope reveals retinal and other intra-ocular hæmorrhages. Bleeding takes place from the bowel. Less frequent symptoms are parotitis, arthritis, pericarditis, and jaundice. Meningitis, associated with headache and delirium, and ending in coma, is more common, and gives a new or different aspect to the case.

Malignant endocarditis of this severe type may be said always to terminate in death after a duration of less than fourteen days; it has been known to end fatally in two days. The course which the disease pursues is one of steadily increasing gravity, broken only by equally ominous complications in the form of meningitis, hæmorrhages, or sudden paralysis referable to cerebral hæmorrhage. Very rarely the disease passes into the subacute or chronic form.

The pyrexia belongs to the continued type, the maximum rise being 104° or 105° F., with a daily remission of about one degree. In some instances it may not exceed 101° , or may even keep close to normal. With the fever may be associated repeated rigors, followed by increased prostration and stupor. The physical signs connected with the heart have already been referred to. The endocardial murmur is usually systolic and mitral. It develops under observation, changes in loudness and quality day by day, and may be reinforced by a second murmur over another part of the præcordia. If traced to the right side of the heart, it is of special significance. Occasionally the murmur is absent, suggesting a parietal lesion. The radial pulse presents all the characters of the typhoid state with increasing prostration, rather than those of any particular form of valvular disease. The so-called cutaneous 'eruption' consists of fine reddish spots, each with a paler centre representing the seat of a minute embolus. The signs of pleurisy, pulmonary infarction or gangrene, or even of pyo-pneumothorax, are sometimes to be determined.

(b) **Subacute and Chronic Malignant Endocarditis.**—This variety of the disease bears but little superficial resemblance in its clinical characters to the severe form just described. Its general features are those of subacute or chronic cardiac disease accompanied by pyrexia and multiple embolism. Heart-disease is the central fact, to which are superadded the phenomena of general infection. There is no prostration until the last; no typhoid aspect. The patient is anæmic, sallow, poorly, and he wastes steadily; but he sits up in bed, reads, engages in conversation intelligently and cheerfully, and may even resent strict treatment. The physical signs connected with the heart consist mainly of a well-developed endocardial murmur, possibly referable to a previous valvular lesion, but changing its characters from time to time under observation. It is usually systolic and mitral; occasionally aortic systolic or diastolic, or mitral præ-systolic; and it may be suddenly altered or complicated by the development of a fresh lesion in the heart, such as rupture or perforation. The pulse varies greatly.

The type of pyrexia in fully developed cases of malignant endocarditis of subacute course is markedly remittent, or even intermittent. Three, four, or even six degrees F. of a rise and fall (between 98° and 102° to 104°) may be recorded daily for weeks on end. In other instances the disturbance of temperature takes the form of irregular bouts of pyrexia complicating the downward course of a severe case of valvular disease. Some patients suffer from repeated rigors, unexpected and irregular, followed by profuse sweats and prostration.

The other phenomena of general infection are mainly significant of embolism—pain and the proper physical signs of infarction of the spleen and lungs, blood in the urine, loss of pulsation in the arteries of the limbs, the development of acute aneurysm with subsequent rupture, and even gangrene of the extremities. This element of the disease gives it a peculiarly uncertain and protean character, its course being, for instance, suddenly interrupted by the development of severe hæmorrhage into the peritoneal cavity, or possibly into the brain, with paralysis and coma ending in rapid death.

The duration of this disease is almost indefinite. It has extended from six or eight to twelve or sixteen weeks—in one case eighteen months. The

progress is very striking. The patient steadily but almost imperceptibly loses strength, flesh, and colour. With very few exceptions, the disease terminates in death, and such patients as have recovered have been left with permanent heart-disease.

DIAGNOSIS.—The acute form has to be diagnosed from typhoid fever, acute tuberculosis, and other infective diseases belonging to the same type. The diagnosis usually turns on its association with some primary disease, and on the result of a physical examination of the heart and the correct interpretation of any murmur that may be discovered. When no murmur is developed, ulcerative endocarditis may escape recognition. Repeated rigors, the type of pyrexia, the character of the spots, and the occurrence of retinal hæmorrhages will further serve to exclude typhoid fever and tuberculosis. When delirium and other cerebral symptoms are urgent, ulcerative endocarditis has been mistaken for 'cerebral rheumatism' and simple meningitis.

The subacute and chronic forms are specially liable to be confounded with a number of diseases which present a similar type of pyrexia, particularly the many pathological conditions which underlie obscure persistent pyrexia, such as latent tuberculosis; lymphadenoma; chronic suppuration in connection with the liver, prostate, bowel, kidney, internal ear, lymphatic glands, and connective tissues; and also syphilis. The existence and changeable character of the murmur, the sudden embolisms, and the absence of other local signs, all indicate the cardiac seat of the disease. From malaria it can be distinguished by the history, by the absence of periodicity in the rigors and fever, as well as by the cardiac murmur.

Some of the gravest complications of this form may, for a moment, be misleading, such as violent headache ending in coma, referable to the development and rupture of embolic intracranial aneurysm. Experience teaches us to be prepared for every possible variety of symptom in a disease of which a striking feature is visceral embolism.

The examination of blood taken during life is frequently of assistance in diagnosis, by showing the presence of micro-organisms. Great care must be exercised in sterilising the skin, instruments, &c., and as the bacteria are never present in large numbers in the peripheral circulation, several cubic centimetres of blood should be collected and many cultivations prepared. It may be necessary to withdraw the blood from a vein by means of a sterile syringe, especially in cases of suspected gonococcal infection, when the blood should be mixed with melted agar-agar (cooled down to about 40° C.) and plate-preparations poured from the mixture, before leaving the bedside.

PROGNOSIS.—The prognosis was formerly hopeless. Almost the only chance of recovery lies in the efficacy of the anti-toxin treatment described below.

TREATMENT.—The gravity of acute ulcerative endocarditis impresses on us the importance of preventive treatment. Dr. Sansom urges the necessity of protecting the subjects of chronic valvular lesions against zymotic diseases, and of attending to the treatment of suppuration with special care. In some instances the primary disease or source of infection can be dealt with remedially, and the attempt should always be made to do so. The mouth, for example, should be cleansed several

times daily ; and it is an absolute rule of practice to secure at least one complete evacuation of the bowels in twenty-four hours.

The most useful and by far the most promising method of treatment for ulcerative endocarditis consists in serum-injections (*see* ANTITOXINS ; and SERUM-THERAPEUTICS). A serum indicated by the results of bacteriological investigation of the blood should be administered freely until its antitoxic action has been proved or disproved ; and in the latter event other serums, or even other brands of the same serum, should be tried.

Many drugs have been given in this disease, unhappily with little benefit, including mercurials, quinine and other antipyretics, and the essential oils of eucalyptus, turpentine, and their allies. In a few instances of the acute form success appears to have followed the use of sodium sulphocarbolate (30-grain doses four times a day), and quinine (15-grain doses three times a day with a free allowance of alcohol).

The case must be managed on the general principles applicable to all acute specific fevers. The best possible nursing, abundance of food, and a sufficient supply of stimulants will be provided. Fresh air is essential. If the disease be traced to defective sanitation, it may be advisable to take the risk involved in changing the room, or even the house, in which the patient is lying. Indeed, in every case it is advisable to move the patient from the room in which he has been ill for some time.

J. MITCHELL BRUCE.

HEART, Inflammation of Walls of. —

SYNON. : Myocarditis ; Carditis ; Fr. *Myocardite* ; Ger. *Myocarditis*.

This disease may be either acute or chronic ; but the latter form, which is attended by the formation of fibrous tissue in the myocardium, is described under the head of HEART, Fibroid Disease of.

Suppurative Inflammation.—Abscess of the heart has been most frequently observed in cases of pyæmia following acute necrosis of bone or diffuse periostitis ; less frequently after phlebitis, chronic or acute arthritis, urethral stricture, chronic abscess, and cancerous ulceration. It occurs most frequently in cases following injury to a bone or a joint in boys. In older subjects it has generally been associated with pyæmia secondary to one or other of the diseases just mentioned. The pyogenic organisms may reach the myocardium by way of the coronary vessels or directly from an infective ulcer in the endocardium.

ANATOMICAL CHARACTERS.—The left ventricle—towards the base and in the papillary muscles—is most commonly affected. Pericarditis co-exists in the great majority of cases, and very frequently endocardial inflammation as well. Pyæmic foci are generally multiple, and vary in size from a pea to a pin's head. They appear at first as small, slightly elevated, yellowish or buff-coloured softened patches, projecting either on the external or on the internal surface of the heart, and covered with inflammatory deposit. On section, these patches either present an appearance of diffused yellowish softening, or contain one or more collections of dark dirty puriform matter, with ragged ill-defined boundaries, as if formed by destruction of the discoloured tissue around. When due to extension from an endocardial ulcer there may be a single abscess, often of larger size than those just described.

Microscopically examined ; the yellowish patches prove to be portions of the myocardium which are infiltrated with pus and consist of necrosed or degenerated muscular tissue and various bacteria. The several stages of the pyæmic process have been found side by side in some cases ; and infective emboli have been discovered in the branches of the coronary arteries, where they served as the foci of the abscesses. The walls of the heart are sometimes in a condition of softening throughout. Abscess of the heart occasionally bursts ; and the contents make their way either into the left ventricle—producing cardiac thrombosis and aneurysm, and perhaps giving rise to further embolism and pyæmic disease—or into the pericardial sac.

SYMPTOMS.—Whatever the symptoms of pyæmic abscess of the heart may be, they are obscured by the general symptoms of the primary disease, and by the local symptoms and signs of pericarditis. Thus, the patients are described as presenting a febrile, typhoid, or pyæmic appearance, an anxious look, dyspnoea, and præcordial pains ; and pericardial friction has generally been discovered over the heart. Rigors have been observed in some cases ; and a peculiar pustular eruption on the skin in other cases. Delirium probably occurs more frequently than in ordinary cases of pyæmia, but may be referable to the accompanying pericarditis. The *physical signs* found in these cases are chiefly those of acute pericarditis. Sometimes an endocardial bellows-murmur may be heard, due either to valvular lesion, or to the formation of an acute aneurysm of the cardiac wall.

The majority of cases of abscess of the heart prove fatal by asthenia ; but the other terminations of abscess mentioned above will be attended by their respective symptoms, and the possibility of sudden death is especially to be noted.

Parenchymatous Inflammation.—This term is used to designate a form of acute myocarditis associated with acute endocarditis and pericarditis, and depending upon the same causes, the most frequent being acute rheumatism. In a small proportion of cases, rheumatic myocarditis appears to occur independently of inflammation of the lining or of the covering membrane. It has been observed most frequently in males, and before the twenty-fifth year of life. Exposure to cold, severe exertion, and local injury are mentioned among exciting causes, but with questionable correctness.

ANATOMICAL CHARACTERS.—The ordinary form of the disease is characterised by the appearance of leucocytes between the muscular fibres of the heart. In one class of cases the inflammation is moderate in intensity and confined to one or more layers of muscle underlying the endocardium or the pericardium, which is also inflamed ; in another class of cases the inflammation is more diffuse, affecting the muscular walls generally.

The myocardium, as it is seen through its inflamed covering, appears of a mottled opaque buffy colour, and is somewhat swollen and softened. The microscopical characters consist chiefly in the appearance of micro-organisms, leucocytes, and inflammatory effusion in the peri-vascular intermuscular connective tissue ; swelling, opacity, coagulation-necrosis, rupture and fatty degeneration of the muscular fibres ; and the ordinary inflammatory changes of the vessels of the part. Beyond this stage, unless the case prove fatal, the diffused form of myocarditis passes into a chronic condition ; and it ends either in

fibroid disease with a moderate amount of atrophy, by development of the inflammatory products and compression of the affected fibres; in fatty degeneration; in calcification; or in cardiac aneurysm.

In all forms of interstitial myocarditis the left ventricle is most frequently the seat of inflammation.

SYMPTOMS.—The principal symptoms of acute rheumatic myocarditis are restlessness and urgent dyspnoea; severe pain and distress referred to the præcordia; and palpitation, which gradually passes into irregularity and greatly increased frequency, and finally into complete cardiac failure. The pulse corresponds. The countenance is anxious, and either pale or cyanosed. The mind is fearful and distressed at first; and delirium frequently supervenes before death, especially in young subjects. Vomiting is not uncommon. The *physical signs* are generally associated with those of endo- and pericarditis. When uncomplicated, they may be described as consisting of violence of the cardiac impulse at first, which rapidly loses in strength and regularity while it increases in frequency; increased area of præcordial dullness; and short sharp sounds, afterwards becoming duller and more feeble. When these symptoms and signs make their appearance, they generally run their course rapidly, and end in death. In a small number of cases they as rapidly disappear.

COMPLICATIONS.—The complications of acute myocarditis have already been sufficiently indicated, including, first, ætiologically, pericarditis, endocarditis, acute rheumatism, and other causes of these forms of inflammation; and secondly, pathologically, rupture of the cardiac walls or valves, acute cardiac thrombosis, aneurysm, hæmato-pericardium, embolism, and septicæmia.

COURSE AND TERMINATIONS.—The course of acute interstitial myocarditis, as already stated, is generally rapid, extending from a few hours to eight days in different cases. Death occurs, in the great majority of cases, from the effects of cardiac failure, if the inflammation be extensive or proceed to supuration. The formation of acute aneurysm by internal rupture, the production of pericarditis by external rupture, and other complications will variously modify the progress and termination of cardiac abscess. Simultaneous rupture both externally and internally causes sudden death.

DIAGNOSIS.—The diagnosis of acute myocarditis is extremely difficult. Occurring in connection with acute rheumatism, it has to be distinguished from endo- and pericarditis. The absence of murmur and of the characteristic signs of pericarditis, along with symptoms of cardiac failure and severe local phenomena, such as pain, distress, dyspnoea, and finally collapse, should generally serve to establish the diagnosis of inflammation of the walls of the heart. It cannot be said that cardiac abscess has ever yet been diagnosed; but the careful consideration of all the points in the case, including the sudden development of a murmur indicative of rupture of a portion of the wall, or of a valve, may hereafter ensure greater success. In the event of the development of the last-named sign, and of septicæmia or embolism, cardiac supuration would have to be carefully diagnosed from ulcerative endocarditis, with which it is often associated. In children, the delirium of acute myocarditis has to be distinguished from that of acute meningeal inflammation, an object which may be effected by the careful observation

of the signs and symptoms connected with the heart.

The diagnosis of pyæmic abscess must largely depend on the discovery of some source of infection.

PROGNOSIS.—The prognosis of myocarditis, when it is either so extensive or so intense as to give rise to unequivocal symptoms, is extremely unfavourable.

TREATMENT.—The two principal indications of treatment in acute inflammation of the substance of the heart are to rest and sustain that organ, and to relieve the pain and distress. Local anodynes, especially in the form of the preparations of belladonna and poultices, and stimulating counter-irritants, such as mustard poultices, will conduce to fulfil the second indication. Such relief is essential, if rest is to be secured. The patient must be spared the very smallest exertion. Food must be given in small quantities, and be easily digestible and highly nutritious; the bowels must be kept open; and the flow of urine should be as free as possible. Alcoholic stimulants will be urgently called for, but must be employed with great discrimination; and palpitation, as much as weakness of the impulse, is to be regarded as an indication of the necessity for these. At the same time, digitalis, ammonia, and other cardiac stimulants should be given cautiously, so as to sustain the cardiac action.

J. MITCHELL BRUCE.

HEART, Malformations of.—**SYNON.** : Congenital heart-disease; Morbus Cœruleus; Fr. *Affections congénitales du Cœur*; Ger. *Missbildungen des Herzens*; *Angeborenen Herzerkrankheiten*.

DEFINITION.—Defects in the structure of the heart due to arrest or perversion of the normal process of development.

Development of the Heart.—The heart develops from a single tube, formed by the fusion of the two original symmetrically placed blood-vessels, found in the early developing embryo. This tube receives at its hinder end the two large omphalo-mesenteric veins, and terminates anteriorly in the aortic bulb, which divides into two branches, the two primitive aortæ. The originally straight tube soon becomes bent upon itself in the form of a U, so that the portion into which the veins enter is brought into apposition to the origin of the aortæ. Constrictions appear in the tube, partially dividing it into four compartments—the hindmost, into which the veins enter, being the sinus venosus, the next the auricular portion, the third the ventricular cavity, and the fourth the aortic bulb. The auricular and ventricular portions enlarge, but are not in their early stages complete cavities, the interior being occupied by a sponge-work of muscular fibres, some of which ultimately disappear, while others take part in the formation of the septa or persist as columnæ carneæ and musculi papillares. In order that the double circulation of mammalian animals may be established, it is necessary that the single tube should be divided longitudinally into a double series of chambers. The process by which this is brought about is somewhat complicated. The orifice leading from the auricular into the ventricular cavity becomes elongated into the form of a slit: two out-growths from the heart-wall appear, situated one on each side of this slit about the middle of its length, and these meet and fuse with one another, thus dividing the orifice into two separate channels. A similar out-growth takes place from the hinder

(upper*) part of the auricular cavity; and this, growing forwards (downwards) as a septum, meets the partition formed in the auriculo-ventricular orifice and fuses with it. In the same way a partition grows backwards (upwards) from the apex of the ventricular cavity and coalesces with the others near their point of fusion. In like manner, by means of longitudinal ridges which arise along the walls and ultimately fuse together, the single aortic bulb is divided into two vessels, the aorta and the pulmonary artery, the line of division becoming, by torsion of the tube, somewhat spiral, so that the pulmonary artery which is connected with the right ventricle below crosses the aorta to lie on its left side above. This last septum coalesces with the others at its hinder (lower) end, sending a short prolongation forwards (downwards) to meet and complete the ventricular partition. The formation of the foramen ovale in the auricular septum is a secondary process: a number of small holes appear in the septum and coalesce so as to form a single channel. An endocardial fold grows over this aperture and at first acts as a valve: subsequently it becomes attached along the margin of the foramen and entirely closes it after the birth of the child. The ductus arteriosus—the communication between the aorta and the pulmonary artery—becomes obliterated before the end of the third week of extra-uterine life.

Varieties of Malformation.—Defects may occur in the formation of any of the septa above mentioned, or in the development of the valves at the orifices of the great vessels. These vessels are occasionally transposed, so that each communicates with the opposite ventricle to that with which it is normally connected. The foetal channels—the foramen ovale and the ductus arteriosus—may also fail to close, as they normally do, after birth. It is to be remarked that this last class of defects cannot rightly be described as congenital, since the failure in development occurs after the child is born. In the majority of cases several malformations co-exist in affected hearts, one abnormality counteracting to some extent the disturbance in the circulation brought about by the other.

A. Defects in the Primary Septa:

1. In rare cases *absence of all the septa* is met with, and the heart remains a single tube divided into auricle, ventricle, and aortic bulb. The condition produced is similar to that normally existing in fishes.

2. *Absence or Defect of the Ventricular Septum.* Complete absence of the septum between the ventricles is occasionally found, the heart possessing two auricles and one ventricle, as in the frog. Partial absence of this septum is not uncommon, the most usual seat of the defect being the portion separating the upper parts of the ventricles—the so-called ‘undefended space.’ In the hearts of children, and less easily in those of adults, it may be seen that this portion of the septum is thin and membranous, not being strengthened by muscular fibres, as is the rest of the partition. Occasionally the weakness of this part of the septum is shown by the formation of an ‘aneurysm of the undefended space,’ projecting into the cavity of one of the ventricles. Rarely other parts of the septum may be absent, allowing

communication between the two ventricles. If the extreme upper portion of this septum be wanting, a passage may even be left open between the origins of the aorta and the pulmonary artery.

3. *Defects in the Auricular Septum* (apart from patency of the foramen ovale) are occasionally met with. The commonest situation of such defect is the anterior (lower) portion of the septum, which is formed last, and normally fuses with the septum of the auriculo-ventricular orifice.

4. *Defects in the Septum of the Aortic Bulb.*—Complete absence of the arterial septum may be found in connection with defect in the ventricular partition. The more common malformations are caused by irregularities in its position. Thus the lumen of the pulmonary artery may be so far encroached upon as to leave this vessel very narrow (*stenosis*) or quite impervious (*atresia*), the aorta being consequently much larger than usual, and connected in many cases with both ventricles. Conversely, but much more rarely, the aorta may be narrow or obliterated and the pulmonary artery unusually large. Another defect that is occasionally found is the connection of the aorta with the right ventricle, and of the pulmonary artery with the left (*transposition of vessels*).

B. *Defects in the Valves* at the orifices of the great vessels are not infrequently found. Thus, instead of three semilunar valves only two may be present, or a single membrane with a central perforation may represent the valves. Such fusion is probably the result of foetal endocarditis. In other cases additional valves are formed, so that four or even five pockets exist, but the additional segments generally remain rudimentary. Small congenital perforations of the valves are not very uncommon.

C. (1) *Patency of the foramen ovale.*—It is not very rare to find *post mortem*, even in individuals who have shown no symptoms of cardiac disease, a small aperture, corresponding to part of the foramen ovale, by which communication between the two auricles is produced. Larger defects, permitting free interchange of blood between the right and left hearts, are generally accompanied by the usual signs of congenital malformation, and are often associated with other cardiac deformities.

(2) *Patency of the ductus arteriosus* is also most frequently found in connection with other varieties of congenital defect.

D. *Formation of additional septa* is not a common abnormality. The appearance of an imperfect second septum in the right ventricle has been described, the aorta also arising partly from this ventricle. The condition resembles that normally found in the turtle, in which animal there are two aortic ventricles and one pulmonary.

E. *Fissure of apex.*—The slight depression sometimes found corresponding to the division between the two ventricles at the apex of the heart may be exaggerated, so that the apex of the organ appears bifid or mitre-shaped. A similar condition is said to occur normally in the dugong.

F. *Complete absence of the heart* (acardia) is met with in foetal monstrosities. It is, of course, incompatible with extra-uterine life.

COMBINATIONS OF DEFECTS.—Stenosis of the pulmonary artery, one of the commonest malformations, affords a good instance of the interdependence of cardiac anomalies. Since the blood cannot reach the lungs in sufficient quantity through the narrow

* The expressions ‘forwards’ and ‘backwards’ refer to the position of the original cardiac tube which lies longitudinally in the embryo: the words in brackets express the relations existing in the fully formed heart in the upright position.

trunk, an additional supply must be brought from elsewhere. For this purpose the ductus arteriosus generally remains patent, so that the aorta may supply the defect. At the same time the pressure existing in the right heart, owing to the narrowness of its arterial outlet, needs relief, which is afforded either by continued patency of the foramen ovale, or by a defect in the ventricular septum, situated generally at the undefended space. Stricture of the trunk of the aorta, either at its origin or, as more frequently happens, close to its junction with the ductus arteriosus, is similarly remedied by the patency of this latter vessel. In the event of total atresia of either great vessel occurring, the ventricle from which it arises atrophies to a great extent from want of use. In cases of stenosis, on the other hand, the ventricle hypertrophies and dilates behind the obstruction. In many hearts in which the pulmonary artery is stenosed, the aorta is correspondingly enlarged and communicates with both ventricles, thus receiving an additional quantity of blood and relieving the pressure in the right heart.

ÆTIOLOGY.—Two distinct, or partially distinct, causes appear to be at work in the production of cardiac malformation. It may be due, on the one hand, to some defect in the developmental energy of the embryonic cells, similar to that whereby defects are produced in other parts; the nature of this failure of energy is not clearly understood. On the other hand, endocarditis, identical with that occurring in post-natal life, may affect the heart of the foetus while still unborn, and may cause perversion of the process of development. Thus inflammation of the endocardium within the trunk, or at the orifice, of the pulmonary artery may cause stenosis or obliteration of this vessel; while endocarditis affecting the undefended space may cause weakening of this part and lead either to the formation of an aneurysm or to actual defect in the septum at this point. It must be admitted, however, that the rheumatic poison, circulating in the blood of the mother, may not only cause endocarditis in the foetus, but may be not altogether unconnected with certain cases which appear to be due to unexplained errors of development. It has been found in experiments upon fertilised eggs, that not only such external interference as incubating them in abnormal positions or varnishing parts of the exterior may produce defects in the development of the chicks; but that the injection of poisons, such as the toxins of bacteria, may have the same result. It is therefore highly probable that many congenital abnormalities may really be due to toxins in the mother's blood, which have poisoned the growing cells of the embryo and caused them to develop irregularly or defectively. The rheumatic poison is a very probable agent of this nature, as are also the poisons of the acute specific fevers and other diseases. It is known indeed that many of these may cause death of the foetus and consequent abortion; a smaller dose may act only on individual cells and impair their subsequent development. The 'selective' action of certain toxins upon particular tissues would conceivably hold good in the embryonic stage of these tissues, and the rheumatic poison which affects the endocardium in extra-uterine life might act on the same tissue before birth. When once any primary defect has been produced, the resulting disturbance in the blood-pressure within the cavities of the heart will tend to cause secondary malformations, such as patency of the foramen ovale

or defect in the ventricular septum, by which some degree of compensation is effected.

CLINICAL PHENOMENA.—The subjects of congenital defects of the heart are, as a rule, weak and sickly. They are handicapped in the race of life and succumb readily to adverse conditions. Thus they are very susceptible to cold, and prone to develop bronchitis and tuberculosis of the lungs. Many infants suffering from this form of malady perish from convulsions. The disturbance produced in the circulation of the blood by the various lesions results in the appearance of murmurs, which present sometimes very characteristic features. They are generally systolic in time, but may be diastolic, or may not correspond exactly with either sound in the ordinary cardiac cycle. They are generally loud and harsh in quality, and are audible over a great part of the præcordium, or even over the whole chest.

Three special phenomena are associated with cardiac malformations—cyanosis, clubbing of the fingers, and concentration of the blood. *Cyanosis*, or blueness of the face and extremities, is in many cases a very marked feature. It may exist from the moment of birth, the term 'morbus cæruleus' being applied to the condition presented by infants so affected. In other cases it is slight and may be only manifested on exertion. The nose, ears, and lips in severe cases are of a deep blue, or almost black, colour, while the rest of the face and the hands and feet are livid and dusky. The circulation in the parts appears slow and defective. No entirely satisfactory explanation has been given of this phenomenon. It has been held (1) that it is due to mixture of venous and arterial blood; but this explanation cannot be maintained in face of the facts that cyanosis may be present in cases in which no communication between the right and left hearts is found, and absent in others where free admixture of blood exists. (2) Deficient aëration of the blood owing to defective circulation through the lungs is possibly a factor in some cases, but cannot sufficiently explain all. (3) Venous congestion, owing to defective action of the heart and consequent over-filling of the veins, is also a probable cause of some at least of these cases, while in addition to this (4) the concentrated condition of the blood causes any defect of oxygenation, and any tendency to stagnation in superficial parts, to be manifested by a deeper colour than would be produced under normal conditions.

Clubbing of the fingers and toes is usually present along with cyanosis, and absent when this is not found. The cause of its appearance is also doubtful, but it is probably due to congestion of the extremities, arising from defective circulation and leading to increased formation of connective tissue. The nose in some cases is also bulbous. See p. 548.

Concentration of the blood may reach a very considerable degree. Thus the specific gravity may rise to 1070 or 1080, and the number of red corpuscles may be augmented to over eight millions in each cubic millimetre, the hæmoglobin being increased by as much as one tenth of the normal amount (110 per cent.). It has been suggested that this increased richness of the blood is due to diminished wear and tear of the corpuscles, owing to defective circulation, especially through the lungs. Little is known, however, at present of the life-history of the blood-corpuscles under normal conditions, and the whole subject awaits further elucidation.

In many cases of congenital heart-disease none of these characteristic phenomena are present.

DIAGNOSIS.—Two points have to be determined in relation to congenital deformity of the heart: (1) the existence of a malformation, and (2) the nature of the lesion. With regard to the former point, it necessarily happens, owing to the tendency of these conditions to shorten life, that the patients in whom the diagnosis has to be made are generally children. If the typical signs—cyanosis, clubbing, and cardiac murmurs—are present, the diagnosis of congenital malformation is easily made. If, however, these are absent, great difficulty may arise. The presence of a cardiac murmur in a very young infant is very suggestive of congenital defect. In rather older children the co-existence of a loud murmur with a præcordial impulse which is either feeble or normal in strength usually points to the existence of malformation, since the various forms of acquired disease rapidly produce in children hypertrophy of the heart, and consequently an increased force of contraction. The increase in the size of the heart, due to post-natal endocarditis, generally results also in some bulging of the præcordial region, which is absent in congenital cases. Congenital stenosis of the aorta—a rare condition—may, however, give rise to hypertrophy of the left ventricle, and narrowing of the pulmonary artery is accompanied by enlargement of the right side of the heart. The murmurs caused by congenital disease of the heart are more frequently basic than apical, and in the majority of cases are systolic in time; they are frequently very loud and harsh—out of proportion to the enlargement of the heart and force of the impulse—and may be audible all over the præcordia and, indeed, all over the thorax. It must be borne in mind, however, that in anæmic children hæmic murmurs at the base of the heart are not at all uncommon; these, however, are not accompanied by a thrill, which may, or may not, be present in cases of malformation. A weak pulmonary closure-sound along with an apical murmur is suggestive of cardiac deformity, the pulmonary sound in children being normally accentuated.

In the majority of cases it is not possible to determine the exact nature of the deformity present. As already mentioned the commonest malformation of the heart consists in a *narrowing of the trunk of the pulmonary artery*, generally combined with a compensatory enlargement of the aorta and patency of the foramen ovale. This condition is typically accompanied by cyanosis and by a loud systolic murmur, audible over a wide region, and having its point of maximum intensity in the second left intercostal space near to the border of the sternum. Signs of enlargement of the right side of the heart may be present, but the pulmonary closure sound is not accentuated, and may be unduly weak. *Patency of the foramen ovale* may give rise to no physical signs whatever in slight cases: in others it may be accompanied by cyanosis and by the presence of a murmur, which is generally systolic in time, but may occupy any position in the cardiac cycle. *Defects in the ventricular septum* are said to give rise to loud systolic murmurs without any accompanying thrill. A basic murmur going along with a well-marked thrill, in a heart which shows no sign of hypertrophy, may be indicative of *persistency of the ductus arteriosus*. The thrill is often very prolonged in these cases, lasting well into the diastolic period. The murmur may

be found to begin distinctly after the systolic impulse. Cyanosis and clubbing of the fingers are often absent in cases of this deformity. *Congenital stenosis of the aorta* is marked by similar signs to those which accompany the condition when it results from post-natal disease. The left ventricle is hypertrophied and a systolic murmur, well conducted into the vessels of the neck, is audible. A thrill may or may not be present. In those cases in which obliteration of the vessel takes place near to its junction with the ductus arteriosus—a condition generally treated as a cardiac malformation—signs of the formation of a collateral circulation may be evidenced by the presence of dilated subcutaneous arterial trunks. In cases of *transposition of the aorta and pulmonary artery* no murmur is likely to be audible, but the second sound of the heart is much exaggerated in the pulmonary region, and cyanosis is generally well marked.

PROGNOSIS.—Speaking generally the outlook in all cases of congenital heart-disease is bad, the great majority of patients thus affected failing to reach adult life. In particular instances any forecast of the probable course of the case must be guided rather by the general features manifested than by any reliance upon a power of ascertaining the exact nature of the lesion: the degree of cyanosis of the face and extremities, the amount of dyspnoea occurring on any exertion, and the presence or absence of associated pulmonary disease constitute the main guides available for forming an opinion. The circumstances of the patient must also be taken into account, since the subjects of cardiac malformations need most careful attention and the most favourable surroundings to enable them to survive. With regard to the different varieties of defect, it appears that patency of the foramen ovale is the least serious, since small degrees of this malformation are compatible with the attainment of advanced age. Defects in the ventricular septum come next in order of severity, life being occasionally prolonged to the adult period. Cases of pulmonary stenosis may also rarely reach adult age, but the majority die in childhood. Patients suffering from total atresia of this artery, or from transposition of the aorta and pulmonary artery, seldom live many months. Fœtuses, the subjects of atresia of the aorta, are seldom viable, and stenosis of this artery, if congenital and affecting the orifice of the vessel, is generally rapidly fatal. Stenosis occurring at the junction of the ductus arteriosus is less unfavourable, and is even compatible with the attainment of adult life. Rupture of the aorta may be the ultimate cause of death in such cases. Tuberculosis occurring in patients suffering from cardiac deformity is likely to progress rapidly to a fatal issue.

TREATMENT.—Present knowledge of the causation of congenital deformities is not sufficient to indicate any definite prophylactic measures to be taken by the pregnant woman. It is necessary to guard carefully against infectious diseases, and to have immediate recourse to treatment should any symptoms of rheumatism be manifested. The importance of observing with additional care during pregnancy all the ordinary rules of personal hygiene should be impressed upon the future mother. In children who are the subjects of congenital heart-disease nothing can be done to remedy the deformity. Treatment must be directed to making the surroundings of the patient as favourable as possible. Cold is particularly to be guarded against. Cloth-

ing should be warm and suited to the season of the year. It may be advisable to spend the winter in some warm climate. Unnecessary exertion of all kinds is to be avoided, the occurrence of dyspnoea giving warning in individual cases of the attainment in this respect of the limit which should not be overstepped. The risk of tuberculosis should be minimised as far as possible by maintaining a continual supply of fresh air—without chilling the patient—and by seeing that the food is plentiful and digestible. Residence should, if possible, be in a warm dry locality, upon a gravelly soil. The existence of a family tendency to rheumatism makes all the above precautions even more urgently necessary. Failure of the heart, when it occurs, must be treated on ordinary lines: there is no contra-indication to the use of digitalis and the other cardiac stimulants in these cases. The slightest symptoms of bronchitis must be at once recognised and the condition treated. It is scarcely necessary to insist that the subjects of congenital deformity of the heart should be advised not to marry. The slighter degrees of malformation are not necessarily incompatible with occupations involving no muscular exertion and no special liability to exposure to cold and wet. W. CECIL BOSANQUET.

HEART, Morbid Growths in.—The morbid growths that have been met with in the heart may be thus enumerated in the order of their frequency: (1) Secondary tumours, especially carcinoma; (2) Primary sarcoma and lympho-sarcoma; (3) Non-malignant tumours; and (4) Cysts.

Malignant Disease.—Malignant disease of the heart is very rare, and is generally secondary. Occasionally the heart becomes involved by continuity, the lungs and mediastinum having been primarily affected. Cases have occurred at all periods of life, from infancy to old age; but at least one half of the subjects have been in the middle period of life. The disease is most frequent in males.

ANATOMICAL CHARACTERS.—Carcinoma, epithelioma, and sarcoma have all been found in the heart in different instances. The right side appears to be the more frequently invaded; but the disease is generally multiple. The morbid growth more often presents itself upon either of the surfaces of the heart, rather than in its substance. Encephaloid is the most common; epithelioma by far the most rare. The extent of cardiac wall involved by the growth is sometimes great. When the nodules project externally, they are associated with pericarditis, either local or general. Similarly, prominent nodules in the interior of the heart may cause local endocarditis and thrombosis; in other instances the valvular structures are so involved that incompetence results. In very rare cases the degeneration and softening of the new tissue may lead to rupture of the heart.

SYMPTOMS.—In the great majority of cases of malignant disease of the heart no symptoms are present. Very rarely the subject of the disease suffers from severe præcordial pain, possibly anginal, dyspnoea, palpitation, and vomiting; and death may occur suddenly. In cancer of the heart spreading from the mediastinum or lungs, dyspnoea, cough, and pain are necessarily frequent symptoms. Tenderness on percussion over the præcordia (in association with local pain), pericardial friction, and endocardial murmurs due to involvement of the valvular

apparatus in the new-growth are the only physical signs that have been specially observed.

The disease naturally ends in death, perhaps suddenly.

Malignant disease of the heart has probably never been diagnosed. The appearance of true cardiac pain, or of any of the physical signs just mentioned, in the course of a case of cancer, would suggest that the heart might be involved secondarily.

TREATMENT.—The treatment of malignant disease of the heart is limited to the relief of distress.

Non-Malignant Tumours.—These are among the very rarest of morbid appearances in connection with the heart. *Myxomata* have been recorded as instances of this class of diseases. *Lipomata*, *myxomata*, and *fibromata* have been described.

Cysts.—The occurrence of true cysts in the myocardium (hydatids, abscesses, hæmatomata, and softening gummatous being excluded) is doubtful.

Actinomycosis is very rare, and may simulate malignant tumours. It is generally secondary to primary infection of the lung.

J. MITCHELL BRUCE.

HEART, Palpitation of.—**SYNON.** : Fr. *Palpitation du Cœur*; Ger. *Herzklopfen*.

DEFINITION.—Abnormal movement of the heart, without appreciable structural lesion; the movement, frequent or tumultuous, varying in tone and force. See HEART, Functional Disorders of.

HEART, Passive Congestion of.—The coronary veins, like the veins of other parts, are subject to engorgement when the flow of the blood from them into the right auricle is interrupted. The most common cause of this interruption is dilatation with distension of the cavities of the right side of the heart, which conditions are themselves usually due either to emphysema or to valvular disease of the heart. Disease of the trunks of the coronary veins and pressure upon them may be regarded as less frequent causes of cardiac congestion.

Post mortem, congestion of the heart is recognised, when recent, by fulness of the veins on the surface of the organ; by œdema of the loose connective tissue at the base; and by ecchymosis of the pericardium and endocardium. The pericardial sac contains some serous or sero-sanguinolent effusion; and the mouth of the coronary sinus may be found to be dilated. When the congestion is slight, gradually developed, and of long standing, the venous fulness gives rise to an increased formation of connective tissue in the walls of the heart, which become, in consequence, tough and indurated; while the dilatation of the cavities, with which the congestion is associated, is rendered permanent by the same cause. When divided with the knife, the cardiac walls do not fall inwards; their substance feels like a piece of leather; and the section has a smooth homogeneous appearance. Microscopically, the connective tissue seems to be increased in quantity; and the muscular fibres are in a condition of granular, fatty, and pigmentary degeneration.

Congestion of the heart should never be overlooked in estimating the effects on the circulation of passive congestion, which must impair the functional activity of the heart in the same way as it impairs that of the liver, stomach, and kidneys.

J. MITCHELL BRUCE.

HEART, Rupture of.—The heart is liable to rupture from external injuries, and from causes acting from within. The latter are called spontaneous ruptures, and these only will be considered here.

ÆTIOLOGY.—Intrinsic rupture of the heart may be said never to occur spontaneously when the walls are healthy. The following are the diseased conditions of the heart's walls that *predispose* to rupture:—Myomalacia cordis; hydatids, new-growths, and hæmorrhage in the walls; abscess, ulceration, and fatty degeneration. Of 100 recorded cases of rupture, Quain found the heart fatty in 77. Of the same 100 cases 63 were over sixty years of age; and of 98 cases 54 were males. The *exciting* cause of rupture of the heart is usually excitement or effort; but it may occur during rest or the ordinary avocations of life.

ANATOMICAL CHARACTERS.—In 76 per cent. of cases the left ventricle was the *seat* of the rupture; in 43 per cent. the anterior wall; the right ventricle was ruptured in 13 per cent.; the right auricle in 7 per cent.; the left auricle in 2 per cent.; the septum in 4 per cent.

In rupture of the heart the torn part presents various characters. In *complete* rupture the opening may hardly admit a probe, or it may be several inches in length; it may be longer externally or internally; single or multiple. In *incomplete* rupture the injury may be confined to the internal surface, or to the external surface, or it may occur in the substance of the walls. The edges of the rent are ragged, irregular, from tearing or splitting of the muscular fibres, and sometimes ecchymosed; or the parts may appear ulcerated or perforated. The pericardium generally contains blood-clot and serum.

MECHANISM.—When the walls of the heart are softened by degeneration, or are very thin, as is sometimes the case in the auricles or the right ventricle, they may give way before the pressure to which they are exposed during muscular efforts or strains, or even in the ordinary action of the organ. A part of the wall of the heart is then torn across by the contraction of the healthy fibres among which the tear occurs. Or again, when the walls of the heart are thick, it may be that the outer surface, being strained over the contents of a distended ventricle, as would be the outer surface of an overbent hoop, gives way, tears, and the opening gradually extends from without inwards. Thus rupture is more frequent in the left than in the right ventricle, and its explanation is to be found in the fact that it is also more frequently the seat of fatty degeneration. Further, if hæmorrhage takes place into a softened spot in the substance of the heart, the hæmorrhagic area may yield either externally or internally, and give rise to rupture.

SYMPTOMS.—The symptoms of rupture of the heart may be described as (1) premonitory, and (2) immediate. The former indicate a diseased condition of the organ—breathlessness on exertion, palpitation, more or less irregularity of pulse and faintness. These symptoms may be so slight as hardly to attract attention; or so severe as to cause intense suffering. In the majority of cases no mention is made of preceding symptoms, and death is sudden.

The occurrence of the lesion itself, when the patient lives long enough to describe his sensations, is always marked by intense præcordial pain, distress in breathing, restlessness, rapid and irregular pulse, faintness, pallor, coldness of the skin, sometimes vomiting, and loss of consciousness and convulsions.

If life be prolonged beyond a few minutes, there may be slight intermission in the progress of these symptoms. The duration of the attack itself varies remarkably. In 71 out of 100 cases, death occurred within one or two minutes. One patient, however, lived eight days, 1 six days, 1 three days, 5 two days, 3 nearly twenty-four hours, and 19 various times between five minutes and twelve hours.

The *physical signs*, so far as they can be ascertained, are diminished impulse; muffled, distant, or imperfectly developed sounds; and a weak and intermittent pulse.

COURSE AND TERMINATIONS.—The progress of the fatal malady depends much upon the seat of the rupture, the size of the opening, and the rapidity with which the extension of the laceration takes place. When the septum is torn, there is no external hæmorrhage, and life is prolonged until the patient dies from disturbance in the functions of such an important organ as the heart. The progress of the symptoms is also influenced by the direction and course of the rupture. If the torn fibres overlap from the inside or from the outside, the injury penetrates slowly through the cardiac wall, and the fatal progress is also slow.

PROGNOSIS.—As far as is known, rupture of the heart is always fatal. Still it is possible that such an accident, owing to the small size of the opening, its incomplete character, and its occlusion by a coagulum, might not end in death.

TREATMENT.—Little can be done in the way of treatment of rupture of the heart. The patient's sufferings must be relieved by a hypodermic injection of morphine, or by the use of sedatives or antispasmodics. Perfect rest should be maintained if possible.

RICHARD QUAIN.

J. MITCHELL BRUCE.

HEART, Syphilis of.—Syphilitic disease of the walls of the heart is comparatively rare. The congenital as well as the acquired form of the disease has been met with.

ANATOMICAL CHARACTERS.—This morbid condition presents two leading forms. *Gummata* of the heart appear as pale yellow patches in the cardiac wall, or are found on section as yellowish nodules. They present a variety of appearances, according to their age. When young, they are firm or even fibrous, elastic and homogeneous; creak on section; and are very slightly succulent: when older, they become soft and cheesy. In either form the masses are not isolated, but pass continuously into the myocardium, either directly or through the medium of soft vascular connective tissue, so that they were originally described in this country as 'infiltrations' or 'fibrinous deposits.' The superjacent endocardium or pericardium is vascularised and dull in the early stage of the nodules; opaque and thickened in the more advanced stage. The masses or nodules occur in various numbers in different instances, but are generally multiple. They may be found in any part of the heart. Gummata most frequently become caseous in the centre, as described; and they may then soften more completely and discharge inwards, leading to acute cardiac aneurysm and ulcer of the wall; but more frequently the cheesy products are in a great measure absorbed, leaving a puckered fibroid patch behind.

The second form of syphilitic disease of the myocardium is the *fibroid patch*. This is sometimes

well-defined and localised, and in such instances it represents the stage of full development of an area of ordinary syphilitic interstitial inflammation. In other specimens the fibroid patches appear as irregular masses of indurated fibrous tissue, occupying part of the wall of the heart, and sending septa into the depth of the myocardium, while the endocardium and pericardium that correspond to them are opaque, thickened, and puckered. The syphilitic nature of such patches may be determined by the presence of specific lesions in other viscera; and doubtless they may originate in incomplete local or temporary invasion of the coronary branches by syphilitic arteritis.

An intermediate form of the disease is one in which the outer zone of the gumma has undergone development into fibrous tissue, and the caseous centre remains.

The microscopical characters of syphilitic growths do not require to be described here. In the heart, the primary seat of the disease is the intermuscular tissue. The muscular fibres lying imbedded in the gummatous products or in the fibroid growth are either healthy in appearance, or fattily degenerated and broken up.

Syphilitic endarteritis also affects the coronary vessels or their branches, and may give rise to infarction of the walls of the heart, softening, or fibrosis as described above.

Among the occasional effects of syphilitic disease of the myocardium are chronic aneurysm of the walls; distortion of the orifices and of the valves and their appendages; and, more frequently, adhesion of the pericardial surfaces. Some of the other viscera present, as a rule, evidence of syphilis.

SYMPTOMS.—The subjects of syphilis of the walls of the heart may, from a clinical point of view, be divided into three classes. The first class of patients suffer from some one or other of the ordinary symptoms of chronic cardiac disease, such as dyspnoea, cardiac distress, palpitation, pulmonary complications, and general dropsy; while the physical signs are those of cardiac enlargement, and possibly of valvular incompetence. Præcordial uneasiness, syncope attacks, and remarkable infrequency of the pulse have been prominent features in several recorded cases.

The second class of subjects of this disease die suddenly, after few if any complaints referable to the heart.

The third class die of syphilitic marasmus, and may or may not present some evidence—by physical signs or otherwise—that the heart is not sound.

In many of the cases other symptoms of visceral syphilis have been prominent, for example, phenomena connected with the nervous system.

DIAGNOSIS.—Well-defined symptoms and physical signs connected with the heart, particularly those just mentioned, occurring in a syphilitic subject at a comparatively early age for degenerative lesions, would furnish considerable grounds for the diagnosis of specific cardiac disease, in the absence of other more probable causes—such as a history of endocarditis, strain, or Bright's disease. Success of specific treatment supports the diagnosis.

PROGNOSIS.—If such a diagnosis were positively made, the prognosis would be by no means favourable in spite of the hope that the condition might be successfully controlled by specific treatment, the coronary arteries being frequently involved and the nutrition of the myocardium seriously threatened.

The liability to sudden death must not be overlooked.

TREATMENT.—Anti-syphilitic remedies, especially potassium iodide, should be freely tried, along with other cardiac measures indicated on general principles. See also HEART, Valves and Orifices of, Diseases of.

J. MITCHELL BRUCE.

HEART, Thrombosis of.—**SYNON.**: Heart-clotting; *Fr. Thrombose Cardiaque*; *Ger. Gerinnungen im Herzen*; *Herzpolypen*.

DEFINITION.—Coagulation of the blood within the cavities of the heart during life.

ÆTIOLOGY.—Thrombosis of the heart is principally determined by local arrest of the movements of the blood within its cavities. The peculiar saccular condition of the extremities of the auricular appendages, and the trabecular arrangement of the columnæ carneæ of the ventricles, as well as the distance of the same parts from the main blood-currents, determine the favourite localisation of the thrombosis. Roughening of the endocardium and the presence of micro-organisms are also causes of cardiac thrombosis, which might be considered less common than those already mentioned, unless vegetations, the fibrinous coagula of endocarditis, be regarded as thrombi, which, in the strict sense of the term, they are. Certain conditions of the blood contribute to the occurrence of cardiac thrombosis. Finally, thrombi once formed tend to promote the further progress of the condition. See THROMBOSIS.

ANATOMICAL CHARACTERS.—Coagula found within the heart are of two kinds, which have been termed *active* and *passive*, according as they are formed during life, or at or after death, respectively; and the characters of the former, with which alone we are here concerned, cannot be understood until those of the latter have been briefly described.

Passive coagula are found in the heart in most necropsies, occupying the track of the principal blood-currents. Frequently they appear as black or red blood-clots, occupying the auricles principally, and moulded in their cavities. In other cases they take the form of masses of firm whitish fibrin, cleaving with some tenacity to the endocardium, but not truly adherent; matted with the chordæ tendinæ and columnæ carneæ; and projecting some distance into the pulmonary artery. Or, again, passive coagula may be a combination of the two previous forms, the upper part (according to the position of the body) being decolourised or fibrinous, the deeper part resembling more an ordinary blood-clot. In certain cases these passive clots are peculiar. In phthisis and other diseases proving fatal by very slow exhaustion, they are remarkably firm and fibrinous, and closely matted among the chordæ tendinæ—appearances which seem to indicate that coagulation was slowly proceeding for some time before the heart finally ceased to beat. In anæmia they are jelly-like and translucent. In leuchæmia they are soft and creamy in appearance, and yield, when broken up, a puriform fluid. In the acute exanthemata these passive clots are soft and friable; and in many cases of these and of other forms of acute disease and of sudden death, no coagula are found in the heart, which contains only fluid blood.

Active coagula—the result of thrombosis of the heart—are, on the contrary, situated in the saccular appendages of the auricles, at the apices of the ventricles, and in the recesses behind and between

the columnæ carneæ—in other words, as far as possible from the track of active blood-currents. In these situations they may be seen projecting in the form of fleshy knobs or globes, with their free surface smooth and rounded. Their deep surface is adherent to the endocardium, from which, however, it can generally be separated without much difficulty, leaving behind it a discoloured mark. If the thrombus be incised, it will be found to be laminated in structure, somewhat after the fashion of an onion, the colour of the section being greyish-brown or yellowish, with irregular patches of red and black. In most instances the centre is less firm than the periphery; it usually consists of a sanious puriform fluid.

If the process of thrombosis have been proceeding for some time, these formations may extend in all directions, embrace the columnæ carneæ, coalesce in front of them, and finally may fill up a considerable portion of one, or even of more than one, cavity. The thrombi are generally friable; but sometimes they gain in firmness by the deposit of lime-salts within them; and at other times it is possible that they become detached and form into the 'fibrinous balls' which have been found lying free in the cavities of the auricles. Cardiac thrombi may, in part at least, be reabsorbed. They frequently give way during life; and portions of them, as well as of their puriform contents, are conveyed into the circulation, causing embolism and pyæmia.

SYMPTOMS.—The clinical phenomena associated with cardiac thrombosis may be described as those of the last stage of chronic disease of the heart. Præcordial distress and restlessness; irregularity and feebleness of the pulse; œdema and coldness of the extremities; pulmonary congestion, infarction and œdema; dulness of expression, and sopor, broken by low weak delirium; with other symptoms, as well as with the signs of cardiac failure and insufficient emptying of the cavities in systole—all these phenomena are associated with the process of active coagulation within the heart. These phenomena are not directly referable to the thrombosis. All that can be said is, that in such a case thrombosis is probably going on and increasing the embarrassment and the gravity of the condition. An unusual degree of progressive cyanosis appears in some instances. The symptoms of arterial embolism may suddenly make their appearance from detachment of particles of the clots; and, if the puriform contents find their way into the circulation, septicæmia may result.

Cardiac embolism.—The dislodgment *en masse* of a large venous thrombus, and the impaction of the same, or of a 'fibrinous ball,' in one of the orifices of the heart have frequently caused rapid death. See PULMONARY VESSELS, Diseases of.

DIAGNOSIS.—In the presence of the very serious conditions with which cardiac thrombosis is usually associated, the question of its existence is not a point of great importance.

TREATMENT.—The treatment of cardiac thrombosis consists in the treatment of its cause; and nothing is demanded or can be done for the former which is not indicated for the relief of the latter.

The treatment of *embolism* of the pulmonary artery is described separately.

J. MITCHELL BRUCE.

HEART, Tuberculosis of.—Independently of the pericardium, the heart itself is believed to be

rarely the seat of tuberculosis. Grey miliary tubercles have been found in the connective tissue of the wall of the heart in acute general tuberculosis. Tubercle-bacilli occasionally occur on endocardial ulcers. In other instances the 'tubercle' has been of the yellow or cheesy kind, in the form of small nodules lying at various depths in the muscular tissue beneath the pericardium; the latter also being frequently affected, as well as the lungs, intestines, and other organs.

There appears to be no evidence that tuberculosis of the myocardium gives rise to definite symptoms, or that it can be recognised during life.

J. MITCHELL BRUCE.

HEART, Valves of, Aneurysm of.—**DEFINITION.**—A valvular aneurysm is a circumscribed pouching or sacculation of one of the valve-segments.

DESCRIPTION.—Two forms of aneurysm of the valves of the heart are met with. In the one, the whole thickness of the valve is dilated by the blood-pressure so as to form a pouch; in the other, one of the lamellæ being ulcerated by endocarditis, the blood pushes the remaining lamella before it to form a sac. The second form, which is sometimes called 'acute valvular aneurysm,' occurs most commonly in ulcerative endocarditis. Valvular aneurysms vary in size from a pea up to a pigeon's egg. The orifice is almost invariably towards the side of greatest blood-pressure—those on the mitral valve opening towards the left ventricle, those on the aortic valves towards the aorta. They are usually rounded in shape, but may have irregular prolongations between the lamellæ of the valves. Valvular aneurysms are sometimes multiple. The valves of the right side of the heart are seldom affected. The mitral valves are the seat of the larger aneurysms, and are twice as often aneurysmal as the aortic valves.

Valvular aneurysms terminate commonly by early rupture, giving rise to perforation and consequent insufficiency of the valve, and often leading to considerable laceration. Rupture occurs more rapidly in aneurysm of the aortic valves. Mitral aneurysms occasionally become chronic, and filled with solid blood-clot; and aneurysms of the aortic valves may more rarely be found in this condition.

SYMPTOMS.—When seated on the mitral valve, aneurysms usually give rise to no signs until the perforation and laceration of the valve suddenly develop the murmur of mitral insufficiency. An aneurysm of one of the aortic segments may cause a soft systolic murmur over the valves, which one day, as the sac ruptures, is supplemented by a diastolic murmur, and accompanied by the symptoms of aortic regurgitation. The phenomena of this accident are similar to those of sudden rupture of an aortic valve.

B. WALTER FOSTER.

HEART, Valves and Orifices of, Diseases of.—**CLASSIFICATION.**—Two different forms of disorder of the circulation—obstruction and regurgitation—may result mechanically from disease of the valves and orifices of the heart. Valvular disease, on the one hand, is said to be *obstructive* when narrowing of an orifice (*stenosis*) presents an obstacle to the passage of the blood-current. On the other hand, when the blood regurgitates or flows back through an orifice, in consequence of imperfect closure (*insufficiency*) of the valves, due either to

valvular changes or to widening of the orifice, the condition is called *regurgitation*.

Aneurysm of the valves of the heart is discussed separately. See HEART, Valves of, Aneurysm of.

ÆTIOLOGY.—Each of the orifices of the heart may be affected with one or both forms of disease, but the frequency with which they are severally attacked varies. The results of organic disease are chiefly met with in the left side of the heart, and are due to local inflammation (endocarditis) and its consequences, or to chronic degenerative changes, such as atheroma. In adult life the valves of the left side are more frequently affected than those of the right, because they have to bear a much greater pressure; but in fetal life, when the pressure is reversed, the right valves suffer more. Endocarditis is commonly of rheumatic origin, and attacks the mitral more frequently than the aortic valves; the former having to sustain the full force of the ventricular systole, while the latter only bear the force of the aortic recoil. In addition to rheumatic fever, the chief diseases which tend to develop endocarditis are pyæmia, septicæmia, and the acute infective fevers. Valvular disease may be met with in chorea, but in this connection is probably of rheumatic origin. Gonorrhœal infection is occasionally followed by endocarditis. The aortic valves and orifice are more commonly affected than the mitral by chronic endarteritis extending from the aorta, the chief causes of which are gout, old age, syphilis, chronic renal disease, strain, and the abuse of alcohol. These facts explain why mitral affections (commonly rheumatic) occur mostly in early life, and aortic affections in later life. Valvular lesions are more common in men than in women, owing to their more laborious occupations. Direct rupture of a valve owing to a sudden strain may occur. This is most frequently met with in the aortic valves, but similar accidents may occur to the mitral valve and its tendinous cords. Valvular defects are also due in some cases to congenital malformations. See HEART, MALFORMATIONS OF.

ANATOMICAL CHARACTERS.—The pathological changes in the valves and orifices of the heart, which cause valvular defects, are mostly the results of acute or chronic endocarditis. In the acute form, the valvular lesion is caused by the growth of vegetations which interfere with the action of the valve-segments; or by softening and ulceration of the valve-structure, which lead to valvular aneurysm and perforation, or to loss of substance and consequent insufficiency. The more chronic forms of inflammation produce thickening of the valves from overgrowth of the connective tissue, with subsequent retraction from shrinking of the newly formed fibrous elements; or adhesion of the valve-segments causing stenosis.

Aortic stenosis, when not due to congenital malformation, is generally the result of thickening and calcareous degeneration of the valves, or of deformity of the valves from vegetative growths which obstruct the free passage of the blood from the ventricle. Sometimes it is due to adhesion of the valves preventing their elevation, and causing them to form a diaphragm with a narrow central aperture. More rarely it is caused by thickening and contraction of the fibrous ring of the aortic orifice, gradually involving the valves; or by endocardial thickening producing contraction immediately beneath the aortic ring.

Mitral stenosis results most frequently from

thickening and rigidity of the valves, which contract all round the mitral orifice, so as to narrow the outlet, and form a diaphragm between the auricle and the ventricle. This diaphragm is in some cases funnel-shaped, while in others the mitral orifice is transformed into a button-hole aperture, or narrowed to the size of a goose-quill. The tendinous cords of the valve are shortened, and their muscles thickened. In some cases the valves are smooth and thin; in others they are thickened, studded with vegetations, rough and calcareous. This latter state may cause stenosis, without any funnel-formation, as may also fibrinous clots or polypii obstructing the orifice. In many cases of mitral stenosis the valves are also insufficient. Mitral stenosis is mostly observed in young females, as a result of mild rheumatic attacks or chorea. It is sometimes called congenital in cases in which it can be referred to no acute illness.

Aortic insufficiency sometimes depends on dilatation of the aortic orifice, due to softening of the aortic coats, with little or no change in the valves, which are incapable of closing the enlarged orifice. This may be called *relative* insufficiency. Vegetations, thickening, retraction, calcareous degeneration, adhesions, perforations, loss of substance, and rupture of the valve-segments by effort are all causes of aortic insufficiency. In rupture of the valves, a full description of which was first given by Sir Richard Quain (*Edin. Monthly Journ.* 1846), the valve-segment is torn from its angle of attachment, and its free edge retroverted towards the ventricle. This accident happens most frequently in cases where the valves were previously diseased by chronic inflammatory changes connected with the strain of very laborious occupations, and in such cases further laceration may subsequently occur.

Mitral insufficiency is due to thickening, retraction, or deformity from vegetations, of the valve-curtains; adhesion of the curtains to each other or to the ventricular wall; and calcareous degeneration. In some instances one of the valves is perforated or torn; and sometimes the tendinous cords are shortened and thickened, or ruptured as the result of degeneration, preventing the normal action of the valve-curtains. In rarer cases associated with dilated ventricle, the papillary muscles are so weakened by degeneration that they can no longer aid in the closure of the orifice. Dilatation of the left auriculo-ventricular orifice is also a cause of *relative* mitral insufficiency. This form is unconnected with structural valve-changes, and is due to defective muscular contraction, such as occurs in anæmia, typhoid fever, and the failing systole of the later stages of aortic insufficiency.

Valvular defects on the right side of the heart are due to similar changes. They arise chiefly during fetal life, when the right cavities have to bear greater pressure. In adult life these defects are generally associated with diseases of the lungs, which cause increased tension in the right cavities, and lead to their dilatation.

Combined valvular lesions are not infrequent. The most common are stenosis and insufficiency of the aortic valves, along with the same morbid changes of the mitral valves. Endocarditis of the mitral orifice may spread directly to the aortic valves by way of the intervening surface of endocardium. In the last stages of both forms of aortic valvulodisease, the mitral valve becomes insufficient, either from chronic endocarditis, or from dilatation of the

ventricle and of the auriculo-ventricular orifice. Mitral stenosis is not infrequently associated with aortic insufficiency, but is more commonly connected with some degree of narrowing at the aortic orifice. Tricuspid insufficiency is usually met with in the last stages of diseases of the left heart and of some forms of pulmonary affection; and tricuspid stenosis as a congenital malformation is met with in connection with a similar narrowing of the mitral orifice.

EFFECTS.—Valvular diseases of the heart produce a series of morbid phenomena, which are connected together by a necessary sequence. Each and every form of valvular defect impairs the perfection of the heart as a pumping machine, and disturbs the normal relations between the contents of the arteries and of the veins. In front of the lesion there is less blood; behind it there is more. In aortic valvular diseases, the first effects are increase of the blood-pressure in the left ventricle, and lessened blood-pressure in the aorta; next, from the difficulty which the left auricle has in emptying all its contents into an over-full ventricle, there is produced increased pressure in the left auricle and pulmonary veins. Lesions of the mitral valve cause similar results: first, increased pressure in the left auricle, less pressure in the left ventricle, and consequently lessened pressure in the aorta, with a gradual increase of pressure extending from the left auricle to the pulmonary veins. Aortic affections thus act first on the arterial, and secondly on the pulmonary circulation; while mitral lesions affect the pulmonary vessels more immediately. The final results of the two forms are, however, identical, and may be stated in the form of a law—namely, that *all valvular diseases of the heart tend to lessen the quantity of blood in the arterial system, and to produce increased fulness and stasis in the veins*. From the action of this law, various associated visceral disorders follow as consequences. These disorders, however, vary greatly in the period of their occurrence, and in the intensity of their manifestations. This variation is due to the more or less perfect way in which the original valvular defect has been compensated for, by changes in the power of the cardiac muscle and in the capacity of the cardiac cavities. These changes often suffice to maintain fairly the normal balance between the arterial and venous contents, thus *compensating* for the valve lesion; and the process by which this is effected demands careful consideration.

Compensation.—Compensation is effected differently, according to the form of disease. It may be stated generally, that it consists in hypertrophy of the cavity immediately behind the defect. Now hypertrophy means increased contractile power, and consequently better filling of the arteries, and increased arterial tension. Thus it makes up for the valvular incompetency, which tends to lessen arterial tension. When the increased power of the ventricle exactly balances the effects of the valvular mischief, the compensation is complete.

In *aortic stenosis*, hypertrophy of the left ventricle is the mode in which compensation is effected; the obstacle to the blood-current is overcome by the increased muscular power.

In *aortic insufficiency* there is some dilatation of the ventricle as the primary result of the lesion, accompanied by hypertrophy of the muscular wall. A sufficient excess of blood is thrown into the aorta at each systole to allow for the regurgitation during

each diastole; and thus the balance is maintained, though not always equally.

In *mitral lesions* the left auricle is dilated as the primary consequence of the condition of the valves; hypertrophy follows, but is generally insufficient to prevent increased fulness of the pulmonary veins. This impedes the circulation in the lungs; and increased tension in the pulmonary artery soon begets the necessary hypertrophy of the right ventricle. It is by means of this increased power of the right ventricle that the blood is driven through the lungs in spite of the defect in the left heart, and pulmonary stasis is prevented; and the blood entering the left auricle under greater *vis à tergo*, the compensation of the valvular defect is effected. The compensation, from the nature of the means on which it depends, is manifestly less perfect than in aortic lesions.

On the *right side of the heart* similar modes of compensation are observed.

The basis of the salutary changes just described is increased cardiac nutrition; and, consequently, a free coronary circulation is a necessity. Conditions which interfere with this prevent compensation, and so diminish the duration of life. Of the causes of failure of compensation in cases in which this condition has once been established, the most important is over-exertion, causing strain upon the heart. Affections of the lungs such as bronchitis, by throwing extra work upon the right ventricle, may have a similar effect. Acute diseases or starvation, by interfering with the general nutrition, may also cause a damaged heart to fail. Wherever the compensation begins to fail, dilatation of the cavities and vessels behind the lesion commences. This may, however, be checked, and the power of the heart restored for a time. Sooner or later, however, changes in the nutrition of the cardiac muscle, in the vessels, and in the general nutrition, bring on failure of compensation. The cardinal symptom in such cases is weakened contractile power of the heart (*asthénie*, Beau). In this state, the cavity chiefly affected has no longer power to expel its contents fully into the vessels, and consequently becomes gradually and increasingly distended. Failing compensation in aortic valve-disease manifests itself by dilatation of the left ventricle, and the development of secondary mitral insufficiency. Similar retro-dilatation marks the failure in mitral cases, only here it is the right ventricle which dilates, and tricuspid insufficiency and general venous stasis are added to the pre-existing pulmonary engorgement.

Visceral complications.—The most important of the associated disorders of chronic valvular disease, depending on defective contraction of the heart, are the visceral congestions.

In the *lungs*, the habitual engorgement of mitral disease produces a hyper-secretion of mucus and a state of chronic catarrh. The blood-vessels also undergo changes from the excessive intravascular pressure, and become dilated, varicose, and atheromatous; hence oedema and hæmorrhage arise. In mitral stenosis especially, grave and frequent attacks of hæmorrhage, with laceration of the pulmonary substance, are liable to occur, as well as other chronic changes. See LUNGS, Hyperæmia of; Brown Induration of; and Hæmorrhage into.

In the *liver*, venous stasis results from the obstruction to the passage of blood from the hepatic veins into the inferior vena cava. Passive congestion ensues, and 'nutmeg liver' results (see LIVER,

NUTMEG). The passive congestion in the liver, as in the lung, causes catarrh of the tubes, and may thus be productive of slight jaundice. Among other symptoms associated with the hepatic congestion may be mentioned hæmorrhoids and epistaxis.

The *spleen* is a very easily distended organ, and suffers like the liver, but frequently before it; and this may partly account for the pain in the early stages, which is complained of beneath the left ribs. In long-standing cases the spleen becomes tougher, and the capsule opaque and thickened; while hæmorrhagic infarcts are common.

From the hepatic congestion there naturally follows distension of all the other radicles of the portal vein; hence the congestion and chronic catarrh of the *stomach* and *intestines*, which impede digestion and assimilation, and so reinforce the other causes producing the cachexia of chronic valvular disease.

The function of the *kidneys* is more or less disordered. The first stage of general circulatory trouble is lessened arterial tension; this makes itself felt in lessened pressure in the Malpighian tufts, and is manifested by scanty, dense, high-coloured urine. Later on the venous stasis in the kidney leads to the transudation of serum, and consequently albumen appears in the urine. Long-continued venous congestion ends in structural changes, which, as elsewhere, consist in connective-tissue hyperplasia and degenerative (rarely fatty) changes in the tubules. These renal changes sometimes add uræmia to the patient's ailments.

In the *brain* decided alterations are not found, except when a detached vegetation produces embolism and its special phenomena. The brain-substance is, however, generally oedematous, and the membranes are thickened. Delirium is an occasional symptom in heart-disease, and when present to any great degree is of evil import. The blood-vessels of the general circulation are frequently affected with atheroma in hypertrophy of the left ventricle, and it is these degenerative changes, as well as embolic aneurysm, which favour the occurrence of apoplexy.

General dropsy.—The mechanical impediments to the circulation which produce these several visceral congestions, also manifest themselves in the general dropsy which is common in the last stages of heart-disease. The dropsy begins as a puffiness of the ankles, especially the left, at bedtime. The general venous stasis, thus first indicated, advances slowly and surely, if not checked, to general anasarca, and even to dropsy of the serous cavities. The increased venous tension, and the hydræmia of blood-deterioration, are the causes of this serous transudation, which shows itself first in the feet, the most dependent portions of the body, where the pressure of the blood-column is naturally greatest. The horizontal posture, by distributing the pressure, is sufficient at first to disperse the oedema of the ankles. General anasarca is much more frequent in mitral disease, especially the regurgitant form, than in aortic lesions.

In some cases a solid form of oedema is observed. This occurs mostly in the last stages of valvular affections, and is due to thrombosis of venous trunks, in which, the circulation being much impeded, coagulation easily takes place. The termination of the external jugular veins is a common site for such thrombosis; and the left innominate vein, from its transverse position, and from its emptying into the superior vena cava, almost at right angles to the current, is, in the writer's experience, more com-

monly obstructed than the right. Solid oedema is consequently seen more frequently on the left side of the head and neck and in the left arm, than on the right side.

SYMPTOMS.—The earliest symptoms of failing compensation are breathlessness and palpitation on slight exertion, and in mitral cases frequently the appearance of slight oedema about the feet and ankles.

Palpitation is intimately related to the state of cardiac nutrition and innervation, and has no special connection with any form of valvular disease. Cardiac pain, varying in intensity from mere uneasiness to the agony of angina, is most common in aortic cases, and is associated with endarteritis, particularly at the root of the aorta, or with dilatation of the left ventricle, or is a neuralgia of the cardiac plexus. In mitral affections, pain arises from over-distension of the left auricle, and its pressure on neighbouring parts, and later on from dilatation of the right ventricle. Dyspnœa may be present in any form of valvular disease, but it is often absent from the earlier history of aortic cases, while some dyspnœa is always present with mitral lesions. This difference is due to the absence of pulmonary congestion in aortic disease, while it is more or less present from the first in mitral affections. The dyspnœa is a breathlessness rather than a difficulty of breathing. It is panting and gasping in its character, with acceleration of the rate. It is aggravated by any movement, and often compels the patient to sit upright (orthopnœa). Headache, vertigo, night-terrors, and sleeplessness are other symptoms, which depend on disordered cerebral circulation. Sleeplessness is one of the most distressing of all symptoms, and can only be relieved when the dyspnœa is lessened. Other more special symptoms will be found in certain cases, and will be traceable to the disturbances in the circulation, which the particular form of valvular disease has engendered.

Physical signs.—The physical signs associated with valvular affections may be said to be—firstly, those of alteration in the size of the heart; and, secondly, those of mechanical disorders of the circulation; together with one or more endocardial murmurs. The persistence of a murmur is the cardinal sign, and if the murmur be either diastolic or præ systolic in its rhythm, it is of absolute value. A systolic murmur, especially at the base of the heart, may be associated with anæmia (hæmic murmur), but in such a case there is no cardiac hypertrophy, as indicated by increased cardiac dulness, though there is often nervous over-action of the heart. There is also no accentuation of the pulmonary second sound, inasmuch as there is no extra-fulness of the pulmonary blood-vessels from obstructed circulation. The pulse in anæmia is generally quick, ample, and compressible, but, withal, jerky; while with an organic systolic murmur it is generally slow, rising gradually under the finger, and not very compressible (aortic stenosis), or small, irregular, and unequal (mitral insufficiency). The clinical methods of investigating valvular lesions are mainly inspection, palpation, percussion, and auscultation. The signs of each form of valve-disease are stated below; and on these the special diagnosis rests.

DIAGNOSIS.—In *Aortic stenosis* there is often some prominence of the præcordial region, and a steady forcible impulse is perceived below and to the left of its normal position. A thrill, systolic

in time, may often be felt at the base of the heart. On auscultation, a loud, frequently rough, rasping, sometimes musical, murmur is heard with the first sound at mid-sternum, and also at the second right intercostal space. The murmur, commencing with the first sound, extends to the succeeding second sound, which is often not very distinct. In relative stenosis with a dilated aorta, the murmur is softer, and the second sound sometimes louder than normal. The murmur of aortic stenosis is audible all over the upper part of the thorax, especially on the right side; is conducted along the great vessels to the left vertebral groove, and it may be even to the lower dorsal vertebrae; and is occasionally heard at the apex of the heart. The pulse is regular, slow, retarded by the narrowing of the aortic orifice, and slowly developed under the finger. The sphygmogram (see fig. 1) shows the line of ascent to be oblique or broken, instead of nearly vertical; the summit is generally blunt; and the line of descent

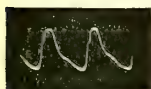


FIG. 1.—Pulse-tracing in Aortic Stenosis.

shows small or no secondary waves, and ill-developed dicrotism. Aortic stenosis when moderate, requiring only hypertrophy of the left ventricle for its compensation, is often very perfectly remedied by this change, and produces little or no disorder of the circulation.

This is also true of cases in which roughness of the valves or vegetations are the cause of even a loud murmur. The amount of hypertrophy of the left ventricle is the surest guide to the gravity of the condition. When the stenosis is very great, epileptiform and syncopal attacks may occur, and lead to sudden death. When the compensation fails, the mitral valve often yields from dilatation of the left ventricle and degeneration of the papillary muscles. Then the pulmonary second sound becomes accentuated; the pulmonary circulation is embarrassed; and dyspnoea, bronchial catarrh, pulmonary hæmorrhage, œdema, and cyanosis supervene.

In *Aortic insufficiency*, inspection discovers a forcible and diffused impulse, the apex-beat being lower than natural, sometimes as low as the seventh intercostal space, and outside the nipple-line. The præcordial region may be bulging from the enlargement of the heart; pulsation may be seen in the upper intercostal spaces at the right edge of the sternum; and a thrill may sometimes be felt there, and occasionally as low down as the heart's apex. The great vessels of the neck pulsate visibly. The area of cardiac dulness is increased in all directions, but mainly vertically. On auscultation, a murmur is heard, replacing and following the second sound, of a blowing or hissing character, rarely rough, and lessening in loudness towards its end. It is usually loudest at mid-sternum and in the second right intercostal space; and it is conducted upwards to the right clavicle, but mainly downwards to the xiphoid cartilage. It is not heard at the back of the chest. It may be conducted to the apex of the heart rather than to the ensiform cartilage; and this occurs, in the writer's opinion, when the posterior or mitral segment of the aortic valves is the incompetent one, as the regurgitant current then falls on the mitral valve, and the murmur is thus conducted to the apex. The second sound may be wholly lost at the base of the heart, being replaced by the murmur; but in some cases it is audible—this being due either to normal closure of

one or two aortic segments, or to the propagated pulmonary second sound. If audible in the carotids, the second sound is aortic, and is of some value as indicating an incomplete defect of the valves. The second sound is often audible at the apex. The first sound at the base is almost always modified, being generally murmurish and often obscured by a systolic murmur, due to slight obstruction from thickening of the valve-segments, or to relative stenosis and the vigour of the ventricular systole. In some cases there is no distinct first sound audible at base or apex; its absence being possibly due to noiseless closure of the mitral valve by the intra-ventricular blood-pressure before the systole occurs. Thus in cases of free regurgitation, both first and second sound may be absent over the left heart; but on listening over the femoral artery with a binaural stethoscope lightly applied, the two sounds of the heart can sometimes be heard. In the majority of cases the insufficiency is no doubt asso-

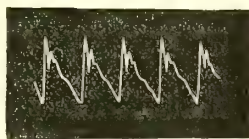


FIG. 2.—Pulse-tracing in Aortic Insufficiency.

ciated with some stenosis, and the murmur is double—a short rough systolic portion, with a softer, longer, and more hissing diastolic portion. This double murmur might be well called the *up-and-down* murmur of aortic valve-disease: the two descriptive words indicating the length, and, to some extent, the characters of its component parts.

The signs connected with the pulse in aortic insufficiency are very significant. As the pulsations of the aorta are visible to the right of the sternum, so the arteries often beat visibly all over the body, even to the radial, temporal, and dorsal arteries of the foot. The ophthalmoscope has shown the same phenomenon in the central artery of the fundus oculi. This remarkable movement of the arteries is due to two causes—firstly, to the hypertrophy and dilatation of the left ventricle, which throws an excessive quantity of blood into the vessels at each systole; and, secondly, to the sudden collapse of the arteries, due to the aortic regurgitation. The arterial recoil during the ventricular diastole is not opposed, as in health, by the resistance of the perfectly closed aortic valves, and, consequently, the blood-column is not sustained, and the arteries collapse. These 'locomotive' features in the pulse are generally increased by elevating the arm. The pulse is sudden, short, large, regular, rapidly collapsing, and vibratory. The sphygmographic tracing brings out these characters: the line of ascent is vertical and lofty; the summit is sharp and pointed; the line of descent falls rapidly, and is broken by a series of secondary waves due to vibratory conditions, but has an ill-developed dicrotism. The post-dicrotic portion of the tracing falls rapidly, from the absence of a sustained blood-column. The longer and more oblique this portion of the tracing, *cæteris paribus*, the less copious the regurgitation (see fig. 2).

Aortic insufficiency often lasts for many years without producing any obvious disturbance of the systemic and pulmonary circulations; hence the absence of dyspnoea and œdema. The hypertrophy and dilatation of the left ventricle, which form the

compensation, suffice to prevent ill effects. At each systole the dilated ventricle throws sufficient blood into the aorta to allow for the reflux, and to maintain a fair arterial tension. Thus the compensation is perfect. In many cases, however, if the reflux is free, the coronary arteries, which are partly filled by the arterial recoil, are deprived of the full force of the blood-wave, and the nutrition of the heart consequently suffers. This is the great source of failing compensation, and the malnutrition of the cardiac muscle soon leads to dilatation of the ventricle, secondary mitral insufficiency, and atrophy.

When the hypertrophy is excessive (over-compensation), as it is in some cases, there are flushings of the head and face, headache, vertigo, and violent arterial action all over the body.

In *Mitral stenosis* there is rarely any prominence of the præcordial region; and in its earlier stages neither increase of the cardiac dullness, nor alteration in the position of the impulse. The impulse, when regular, is fairly distinct, but it is often very irregular, and is associated with a thrill, which precedes, runs up to, and terminates in the impulse. In advanced cases, the area of cardiac dullness is increased laterally; the impulse is diffused, and may be seen in the epigastrium; this indicates hypertrophy of the right ventricle, which sometimes obscures the left ventricular impulse and gives rise to epigastric and hepatic pulsation. The left auricular systole may occasionally be noticed, if the chest be thin, in the third left intercostal space. The sounds heard on auscultation in this lesion vary. The pathognomonic sign is a murmur preceding the systole, and ending with its commencement. This is best called the *præstolic* murmur (also 'auricular systolic'); and is produced when the contracting auricle forces blood under high pressure through a narrowed orifice into the ventricle. The first portion of the blood passes from the auricle into the ventricle noiselessly; and it is only when a stream of higher tension is forced into it by the true auricular systole that the murmur is developed. The murmur is, therefore, short in most instances, occupying the last part of the diastole; it runs up to the first sound, and ends abruptly in it. The position in which the murmur is best heard in its typical præstolic form is at the apex-beat of the heart or at a point between this and the left border of the sternum. It is strictly localised and not conducted in any direction.

This murmur is soft and puffing, but may be harsh. It commonly fills only the last part of the diastolic period. In some cases, with greater roughening and deformity of the valves, the murmur is longer, rougher, more rolling or grinding, and ends abruptly with the first sound, which is very flapping in tone, and might easily be mistaken for the second sound conducted. The careful observation of the impulse or of the carotid pulse with the finger, while auscultating, is necessary in order to avoid the error. Mitral stenosis develops three murmurs under different conditions of the valves: (1) A short softish puff (sounding like *vool*) immediately preceding the first sound; (2) a short rough grinding murmur (*rrrr*); and (3) a long murmur filling nearly all the diastole, often rough, but varying in tone in some cases. These several murmurs increase in intensity up to, and end abruptly with, the first sound. The soft, hissing, diastolic murmur, occasionally heard at the apex, which lessens in in-

tensity, and ends before the first sound, is a conducted aortic (or pulmonary) murmur.

A special peculiarity of the præstolic murmur is its variability: it is the only organic murmur which disappears and reappears as the cardiac conditions change. For instance, a murmur, inaudible while the patient is at rest, is developed by a little exercise; or, again, an irregular tumultuous action of the heart masks all murmur, which becomes distinct as the heart steadies down under the action of digitalis. In other cases there is no distinct murmur, but only a slightly prolonged and rough or grinding first sound, while the murmur is often lost as the heart-failure advances. In cases of mitral stenosis there is accentuation of the pulmonary second sound, from the greatly increased tension in the lesser circulation; there may also be a doubling or reduplication of the second sound at the base. This reduplication is a sign of great value in cases in which the prolonged or grinding first sound is the only sign. The reduplication of the second sound is due to a want of synchronous closure of the pulmonary and aortic valves, from their altered relative tension. A doubling of the first sound is sometimes noticed, probably due to retarded closure of the mitral valve, from lessened fullness of the left ventricle.

The rhythm of the heart is frequently greatly disordered in mitral stenosis, and also in mitral insufficiency. A few beats occur regularly, or nearly so, and then a series of very small hurried contractions follows, to be again succeeded by stronger and better pulsations. These irregularities are referable to the varying charges of blood on which the ventricle contracts. The over-distended right cavities and the left auricle contract rapidly to expel their contents, but the narrowed mitral orifice does not allow a full charge to pass into the ventricle; the diastole is too short for this purpose; and the wave of contraction passes on to the ventricle from the auricle, producing a series of small ineffective pulse-beats, each representing a small charge sent into the aorta. When the series of ineffective contractions ceases, the next diastole is longer; and the succeeding systole sends, as the sphygmograph shows, a fuller charge into the arteries. During the small irregular beats the præstolic murmur is often indistinguishable, but it is again heard with the succeeding slower and more effective beats. This irregularity in the heart's rhythm, however, is not present in all cases of mitral stenosis. In some

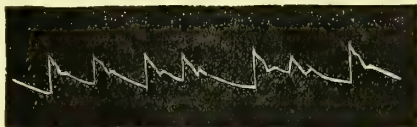


FIG. 3.—Pulse-tracing in Mitral Stenosis.

instances the heart's action and the pulse are regular. The sphygmograph in such cases records a small pulse of low tension, with a little inequality in the volume of the beats; this inequality is often increased by exercise. In other cases the pulse-tracing is small, irregular, and unequal in its pulsations, and marked by true and false intermissions (see fig. 3).

In the earlier stages of mitral stenosis the face may be pale, and the congestive symptoms which mark mitral insufficiency are absent till the later stages. This form of valvular lesion gives rise more

commonly than mitral insufficiency to hæmorrhagic infarction in the lungs; but in other respects the pulmonary and systemic circulations suffer in the same way as described in the other form of mitral disease. The subjects of this defect are often ill-developed and stunted in growth.

In *Mitral insufficiency* inspection discovers some slight prominence of the præcordial region, with increased impulse, the apex often beating to the left of the nipple line. The area of cardiac dulness is augmented mainly in a lateral direction, from the hypertrophy of the right ventricle. On auscultation, a murmur is heard along with or, more often, replacing the first sound, and following it; loudest at the apex; loud along the left edge of the heart; but absent or not so distinct over the right heart and at the base. The murmur is propagated towards the left axilla, and is audible in most cases in the left vertebral groove near to the angle of the scapula. If not audible posteriorly the murmur signifies regurgitation through a narrowed but patent mitral orifice, and may indicate mitral stenosis, in the absence of either a præstolic or diastolic murmur. The mitral systolic murmur associated with dilated left ventricle or enfeebled muscle is not usually heard posteriorly. The murmur is usually loud, blowing, and distinct in its character, keeping the same tone throughout. The true first sound is generally obscured by it, but in some cases may be heard through it, and is then due to the partial closure of the mitral, or to the action of the right auriculo-ventricular valves. The pulmonary second sound is commonly accentuated. The pulmonary first sound has in some cases a murmurish character, probably due to hypertrophy and dilatation of the right ventricle, and relative stenosis of the pulmonary orifice.

The radial pulse in cases of fairly perfect compensation is regular, but quick, small, weak, and easily compressible; and the sphygmograph shows low tension, and an inequality in the size of the pulsations. In cases of less perfect compensation,



FIG. 4.—Pulse-tracing in Mitral Insufficiency.

it becomes irregular and intermittent. In all cases the pulse-beat is weak in comparison with the vigour of the ventricular systole (see fig. 4).

There is no unusual fullness of the superficial veins in the earlier stages of mitral insufficiency. Later, when the right cavities become over-distended, the veins of the neck become full and may even pulsate. This is very distinct when the tricuspid valve gives way. In all cases slight exertion is sufficient to induce dyspnoea; and there is an ever-present tendency to bronchial catarrh, from the congested state of the lungs. When compensation fails, the murmur becomes less distinct; the heart's action is rapid, irregular, and tumultuous; the accentuation of the pulmonary second sound is lost; dyspnoea becomes orthopnoea; and cyanosis, œdema, and hæmorrhagic infarction of the lungs, with general and visceral dropsy, close the case.

Many cases of mitral regurgitation obtain fairly perfect compensation; but the disease, like mitral stenosis, of necessity entails some dyspnoea on exer-

tion, and keeps up constantly an engorged state of the pulmonary vessels.

Valvular affections of the right heart, arising from disease, are uncommon. Those of the *pulmonary valves* are rare; cases are on record, however, of pulmonary stenosis, and a few of pulmonary insufficiency. In the first case the systolic murmur is loud and superficial, and is heard loudest at the third left costal cartilage close to the sternum, and in the second left intercostal space; it is not usually conducted across the sternum, nor upwards to the right clavicle, as is an aortic murmur. A diastolic pulmonary murmur is soft and blowing; and is heard loudest in the same situations, and downwards towards the ensiform cartilage.

The *tricuspid valves* are more frequently affected. Tricuspid insufficiency is indeed a common sequel of disease of the left side of the heart. Structural changes in these valves are, however, rare. Tricuspid insufficiency does not always produce a murmur; when present this is soft and short, and is heard nearer the middle line than a mitral murmur, at the base of the ensiform cartilage. The pulsations of the cervical veins may indicate the lesion when the murmur is absent.

Tricuspid stenosis causes a præstolic murmur, harsh in character, loudest at the base of the ensiform cartilage and towards the left edge of the sternum, not propagated towards the left heart, and not audible at the back of the chest, though faintly conducted along the sternum to the base of the heart. A præstolic thrill may be present. Mitral stenosis has been observed in association with this lesion, and two præstolic murmurs may be made out in such cases.

The physical signs and the diagnosis of *combined valve-lesions* remain to be described. The mitral and aortic valves may each be affected with stenosis and insufficiency, from a single attack of endocarditis, or from one lesion arising as a consequence of the other. The double aortic murmur, already described, indicates the aortic combination; but it must be always remembered that the systolic murmur in these cases may exist with little or no actual stenosis.

In the double form of mitral disease, either defect may exist alone at first, and afterwards be associated with the signs of the second. In some cases the præstolic murmur may fail to be heard, and a systolic murmur may alone be audible; in other cases, there is a prolonged apex-murmur which slightly changes tone; in other cases, again, a short grinding præstolic murmur is followed, occasionally at an interval, by a soft, blowing, systolic one. The combination of aortic with mitral disease may be recognised by the presence of their special murmurs.

PROGNOSIS.—It is very difficult to state general rules of prognosis in valvular affections of the heart, as so much depends on the peculiarities of each case. There are, nevertheless, certain broad rules. As regards origin, rheumatic inflammation is less serious than degenerative change, which occurs later in life, and is necessarily progressive. Accidental rupture is the gravest form of origin. The valve affected is also a prognostic element; but any attempt to arrange cases in order, according to the seat of the valvular defect, must be open to so many exceptions that it must not be too much trusted. Speaking generally, however, aortic regurgitation constitutes the gravest form of lesion owing to the

liability of the patient to the occurrence of sudden death. Pure aortic stenosis with good ventricular hypertrophy is perhaps the least unfavourable form of valvular defect. Mitral regurgitation is also compatible with the attainment of advanced age, but stenosis of this valve is usually progressive, and the outlook in such cases is grave. For purposes of life-insurance the presence of any valvular defect prevents the subject of it from being accounted a 'good life.' It is probable, however, that some adults with compensated mitral regurgitation might safely be accepted at increased rates.

Aortic cases are often free for years from any grave symptoms. The presence of the second sound over the carotids is favourable. The pulse-tracing also affords valuable aid in prognosis, as it gives, by the size of the dicrotic wave and the obliquity of the line of descent, a rough measure of the amount of insufficiency. There is much more risk of sudden death in aortic cases than in mitral.

In mitral lesions the dangers arise from the pulmonary complications; embolism is more common than in aortic affections. Mitral cases can be rescued from asystole more frequently, and die of advanced cardiac cachexia, generally with dropsy. Under favourable conditions of life, requiring little physical exertion and causing no emotional excitement, both forms of mitral disease are compatible with many years of life. When they are conjoined, the prognosis is more unfavourable. In cases of sudden insufficiency, produced by rupture of the valve-curtains or of the tendinous cords, death may occur very rapidly from the disturbance of the circulation.

The whole question of prognosis turns principally on the state of the myocardium. So long as this is sound, compensation may be maintained; the moment degeneration sets in, cardiac failure and all its evil train of symptoms come on. Such failure coming on gradually, without any previous overstrain of the heart, is always most grave. Each successive attack becomes graver, and the visceral congestions which accompany it more stubborn. Albuminuria is a marked index of the gravity of the congestion, and is serious in proportion to the frequency with which it has occurred. In some cases a copious flow of limpid urine is a very grave symptom. Dropsy of the extremities, and more especially of the cavities, is bad, as indicating failure in the peripheral circulation. Next to the cardiac muscle, the state of the peripheral vessels is most important; thus, petechiæ are bad signs; while atheroma and other conditions, such as febrile attacks, add to the danger by interfering with the circulation. The general nutrition of the patient suffering from valvular disease also enters into the prognostic problem. There is a 'cachexia' proper to the end of heart-cases, which is due to the gradual deterioration of the nutritive fluids by the long-continued visceral congestions which hinder assimilation and excretion. Blood is less perfectly made and less perfectly purified; hence the steady deterioration of the general condition, which is always of evil import as regards duration of life. In the poor, the prognosis, as to duration, is not favourable owing to the difficulty of obtaining the needful amount of rest and of nourishing food; but in the well-to-do, all observers see many cases extending over a great number of years.

TREATMENT.—Valvular affections of the heart, whether the result of rheumatic inflammation or of

degenerative change, are, as a rule, incurable. Some few cases of rheumatic origin lose the signs of valvular disease, and are practically restored by the after-processes (for example, contraction) in the inflamed valve; and some few cases also of mitral insufficiency, associated with dilated left ventricle and softened muscle, are cured by treatment. These exceptions are, however, few; and as we cannot repair the valve-mischief, in the vast majority of cases our treatment must be directed to aid the compensatory hypertrophy, and to check the development of the consequences of the defect. The maintenance of the nutrition of the substance of the heart is, therefore, the main object of treatment; just as the state of the nutrition of the heart is the key to the prognosis. On this account the general regimen of heart-cases is very important. In cases in which good compensation is present, no treatment directed to the cardiac condition alone is necessary.

Prophylaxis.—Since valvular disease is generally the result of rheumatism occurring in childhood, it is important to watch carefully for indications of this disease and to treat it effectually. Slight pains in the limbs—frequently muscular—should not be neglected: such so-called 'growing pains' necessitate a careful examination of the heart, and if the slightest murmur be detected complete rest in bed should be insisted upon. Salicylate of sodium should be given in all cases of rheumatic pains in children. See RHEUMATISM.

General regimen.—The diet should in all cases of valvular disease of the heart be unstimulating but sustaining, consisting of a good proportion of albuminous food (meat, eggs, poultry, and fish), with wine in moderate quantity, and some chalybeate water. There should be no unnecessary excitement of the heart, either by exercise or emotion. All athletics and violent efforts should be avoided. In boys, football and all forms of racing must be forbidden. Cricket and gentler outdoor games may be permitted in well-compensated cases, and gentle horse-exercise is often beneficial. Bicycling upon level ground is admissible, but up-hill this form of exercise is carefully to be avoided. In aortic cases, steady exercise without strain is beneficial. The residence should be so situated as to avoid the necessity of exertion, sudden changes of temperature, cold, and damp. The chief object of the regimen should be to prevent anæmia; hence plenty of fresh air is essential. Tobacco is injurious. In early life over-exertion and exposure to cold—in adult life, emotional, sexual, and dietetic excesses are the chief dangers. The propriety of marriage must be considered in each case on its merits. Women, as a rule, should not marry; when affected with mitral disease they are often barren. To men marriage is more generally permissible.

Medicinal treatment.—The therapeutical treatment varies according to the stage of the cardiac disease. The mechanical defect of a valve first makes itself felt by shortness of breath, sometimes by palpitation and præcordial pain; these symptoms pass away when compensation is effected, but till then require treatment. In cases of mitral disease, tincture of digitalis (m v doses) relieves the palpitation; spirits of chloroform is also a useful adjunct. In aortic cases, ether and other diffusible stimulants, small doses of opium and belladonna, with the local application of belladonna to the præcordial region, are valuable remedies.

The præcordial pain, mostly retro-sternal, may, when severe, require a few leeches or cupping, but generally yields to mild counter-irritants, such as turpentine, iodine, or mustard. Internally the bromides are useful; when the pain occurs paroxysmally, ethereal preparations and ammonia act well.

When the compensatory changes in the heart are effected, the palpitation and pain decline, and the chief indication is to keep up the nutrition of the heart by the hygienic rules above given, and by the administration of preparations of iron, combined with arsenic, strychnine, quinine, and mineral acids. Chalybeate waters are also useful adjuncts. The syrup, infusion, and tincture of the *Prunus virginica* are preparations of value in some cases after the use of digitalis. The secretions should be carefully watched, and the bowels opened freely every day, so as to avoid straining, and to relieve the portal circulation. The quantity of urine should be daily noted, as it is a capital index of the state of arterial tension. Patients in whom the most perfect compensation exists are, nevertheless, in a state of unstable equilibrium, for an exaggeration of a physiological act or emotion may disturb the balance of the circulation. In most cases the compensation breaks down sooner or later. The pulmonary congestion soon manifests itself by bronchial catarrh, which requires expectorants in various combinations, while friction, poultices, and counter-irritation are applied to the chest-walls.

For the general visceral congestions our chief remedies are, firstly, diuretics; and, failing these, hydragogue cathartics. Of diuretics, the salts of potassium, digitalis, squill, broom, chimaphila, spirit of nitrous ether, and juniper are the most useful. The hydragogue cathartics, which relieve the over-distended portal vessels primarily, and the general circulation secondarily, are also most valuable: of these the compound powders of scammony and jalap in 20- to 40-grain doses; bitartrate of potassium in electuary, one or two drachms, every morning; sulphate of magnesium; and senna are the most trustworthy. By the judicious use of an occasional purgative, and the administration of a suitable diuretic, aided by cupping, poultices; and sometimes a small blister over the loins, combined with rest and stimulants, the worst cases of dropsy from cardiac failure are often saved.

For the dyspnoea and the insomnia, two of the worst symptoms, we have a remedy of great power in the subcutaneous injection of morphia in doses of one-sixth of a grain upwards. This remedy acts often like a charm, and may be used even in the worst cases of both mitral and aortic disease, but always with caution. Albumen in the urine does not necessarily contra-indicate its use. In some cases chloral hydrate and bromide of potassium, alone or in combination, are valuable remedies for the insomnia; and chloralamide in doses of 20-40 grs. is a safe drug which does not cause cardiac depression. Sulphonal and paraldehyde are also useful; but they should be given cautiously. The bromides may be prescribed with other sedatives, such as nitroglycerin, for the dyspnoea. The compressed-air bath also relieves the last symptom.

Dropsy, like the visceral congestions with which it is associated, requires the use of diuretics and hydragogue cathartics. When these fail, the swollen limbs may be punctured with benefit. Continued friction of the limbs, by stimulating the vessels, will often cause considerable anasarca to disappear. The

drug on which main reliance must be placed when general dropsy supervenes is digitalis. It is *par excellence* the cardiac tonic. Convallaria, strophanthus, casca, caffeine, and Adonis vernalis are other drugs having similar action—strengthening, but slowing, the heart-beat. They are all less trustworthy than digitalis. Caffeine is a good diuretic, and so occasionally is convallaria, even when digitalis has failed. Strophanthus is generally the most useful.

Digitalis has so great a share in the therapeutics of heart-disease, and a knowledge of its action is so important, that it must be discussed separately and last. Whatever views may be held as to its physiological action, its greatest triumphs are seen clinically in the treatment of valvular disease, when cyanosis, distended jugular veins, dyspnoea, congested viscera, dropsical limbs, scanty urine, tumultuous heart-action, and a frequent, irregular pulse, indicate failing compensation. This assemblage of symptoms is mostly seen in mitral cases, and it is precisely in this class that the drug is most valuable. Under its use 'the pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognises its master and beats a sullen retreat.' In mitral stenosis these good results are due not only to the increased vigour given by the drug to the contractile power of the heart, but also to the fact that by its slowing action the diastolic period of each revolution is lengthened, and the time thus increased during which the distended auricle can force its contents through the narrowed mitral orifice into the left ventricle. Digitalis here not only obtains a better filling of the ventricle, but a more effective discharge of its contents when filled: and thus, under its use, beat by beat, the general and pulmonary venous congestion is relieved. In mitral insufficiency it is almost equally potent. In both forms certain of its good effects would seem to be due to some influence, probably through the pneumogastric nerves, in producing contraction of the pulmonary blood-vessels. It is perhaps this property which makes it valuable in pulmonary hæmorrhages, independent of heart-disease.

In aortic valvular diseases digitalis is not so valuable a remedy. In these cases the assemblage of symptoms mentioned above is not met with, except sometimes in the later stages, when the mitral valve is secondarily affected, and the case is not one of pure aortic disease. In these compound cases the drug is valuable, especially in combination with ether and ammonia. In aortic insufficiency alone, the slowing action of the digitalis produces evil by increasing the length of the diastolic period of each revolution, during which the regurgitation takes place. The force it may give to the systole is no gain in the face of this slowing action, inasmuch as the aortic recoil gains in the same proportion as the ventricular systole, and thus forces blood back into the ventricle with increased vigour during the lengthened diastole. It is important in aortic insufficiency to maintain the frequency of the cardiac action; hence these cases are so constantly the better for carefully regulated bodily activity; and so, when the toning effects of digitalis are required, it should always be given in combination with ether and ammonia, to keep up quick action of the heart, and to prevent the vertigo and syncope which may otherwise occur. When there is excessive hypertrophy in cases of

aortic insufficiency, digitalis is useful sometimes in quieting palpitation, reducing excessive frequency, and lessening headache and vascular excitement. Caffeine given with senega, aconite in small doses, and Veratrum viride also relieve these symptoms; but a few drops of nitrite of amyl inhaled from cotton-wool, or nitroglycerin tablets, are more rapidly and surely beneficial. In many cases of aortic disease potassium iodide in doses of 3 to 5 grains is a valuable remedy. In aortic stenosis digitalis is rarely required. The simple mode of compensation makes these cases require little treatment. Digitalis is sometimes useful in combination with stimulants to give vigour to the myocardium, and check the tendency to dilatation. If it slows the action of the heart notably, its effect becomes hurtful. Nux vomica often prevents this.

In combined valvular lesions, the predominant lesion must be the guide in the use of digitalis; but it may be given advantageously whenever the general signs of venous stasis are present. The diuretic power of the drug is one of the best tests of its beneficial action. Relying on this test, the writer often gives digitalis for weeks, nay, months at a time, and obtains improvement in the nutrition of the heart which lasts long after its discontinuance. Digitalis effects this improvement by increasing the vigour of the coronary circulation, and thus builds up new heart-muscle to compensate a valvular defect. In aortic cases the nutrition of the muscle must be kept up by iron, arsenic, quinine, strychnine, and stimulants.

The preparations of digitalis which may be used are, the powder in $\frac{1}{2}$ to 1 grain; the tincture in \mathfrak{m} v to xv; and the infusion in \mathfrak{z} j to \mathfrak{z} vj doses. As a diuretic in cases of dropsy, the old combination of squill, digitalis, and blue-pill is invaluable.

B. WALTER FOSTER.

HEART, Wounds of.—SYNON.: Fr. *Blessures du Cœur*; Ger. *Herzwunden*.

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—Wounds of the heart may be punctured, incised, or lacerated; and inflicted with a variety of weapons or other sharp bodies, as well as with projectiles, especially bullets. Traumatic ruptures and contusions form another considerable class of injuries of the heart, which chiefly result from falls, crushing accidents (for example, being 'run over'), kicks and blows. Injuries of the heart due to the entrance of foreign bodies, such as a needle or a bone, have been inflicted in some rare cases from the interior of the œsophagus or stomach; the most remarkable case of this kind being one of a wound of the pericardium from behind by the point of a sword which a juggler had attempted to 'swallow.'

Post mortem, the chest-wall generally presents evidence of the wound that has been inflicted. The pericardium rarely escapes injury. Its sac is found to contain blood in recent cases, or effused lymph or pus when life has been preserved for a few days or more. The walls of the heart at the seat of injury present different appearances, according to the precise nature of the lesion. Punctured and incised wounds may be of all sizes; may take either a direct or an oblique direction through the muscular fibres; and are generally penetrating. Bullet-wounds cut away a portion of the heart, whether at the borders or from the thickness of the organ. Traumatic ruptures present special characters (*see* HEART, Rupture of). In all the varieties of injury

of the heart, the wound is found plugged with blood-clot, the edges being either infiltrated or ecchymosed and torn. In cases that do not prove rapidly fatal, the usual signs of inflammation, or healing, and even cicatrisation, are found in the heart; or aneurysm of the cardiac walls may be developed as a result of the latter. The valves and their appendages are frequently incised or otherwise injured. In some cases a portion of the weapon, projectile, or foreign body may be found in the heart. The ventricles—and especially the right ventricle—are the parts of the heart most commonly injured. The great vessels, lungs, and the arteries of the chest-wall may also be wounded in different instances.

It must be observed that a blow over the heart has proved instantly fatal without leaving any lesion discoverable *post mortem*.

From these results it is evident that wounds of the heart may prove rapidly fatal by loss of blood, by compression of the heart resulting from hæmorrhage into the pericardium, or by shock; while, at a later stage, pericarditis, myocarditis, and secondary hæmorrhage may be expected to supervene.

SYMPTOMS, COURSE, AND TERMINATIONS.—In about one third of recorded cases of injury of the heart, either death is immediate—fainting, convulsions, and the other symptoms of syncope, as well as those of shock, being the prominent phenomena, along with external hæmorrhage; or the patient drops dead after a few moments, during which time he may have undergone considerable exertion. In a second class of cases, the symptoms of syncope or of shock occur immediately, but death does not ensue at once. The patient then lies in a state either of unconsciousness or of complete prostration. In the latter event he complains of a sense of great debility, præcordial oppression, dyspnoea, and suffocation; the surface is cold, pallid, and trembling; vomiting may occur; and there is usually hæmorrhage from the region of the heart. Death occurs after minutes or hours, either from exhaustion, due chiefly to continued or repeated loss of blood, or from compression of the heart. In a third series of cases the course is more protracted. The patient, after suffering from the symptoms just enumerated, but in a less degree, passes through the various phases of constitutional disturbance commonly observed in severe wounds, complicated, however, with pericarditis, myocarditis, and repeated hæmorrhage; and dies of exhaustion after an illness of weeks or months. Lastly, in a small proportion of cases, the patient survives the various accidents and complications just described, and the wound of the heart heals; but it sometimes happens that signs of aneurysm of the cardiac walls, or of incompetence of the valvular apparatus, are developed as the result of the lesion.

DIAGNOSIS.—Wounds of the heart can usually be diagnosed without difficulty by the situation of the external injury and the severity of the symptoms. Similar symptoms may, however, follow injuries of the great vessels in the neighbourhood of the heart, or of the arteries of the walls of the chest, if the hæmorrhage be profuse. Still, unless there be almost complete certainty that a vessel within reach is the only seat of the bleeding, the diagnosis should be left undecided, and all dangerous interference avoided.

PROGNOSIS.—Injury of the heart is generally to be regarded as certain to end in death; but it should not be forgotten that as many as 12 per cent.

of recorded cases are said to have recovered. Some very remarkable instances have occurred of recovery after very severe injury to this organ. The prognosis may be broadly estimated by the severity of the immediate symptoms. Traumatic rupture of the heart is said to have invariably proved fatal.

TREATMENT.—In wounds of the heart, the hæmorrhage must be at once arrested by the usual surgical means; immediate death must be prevented by cautious stimulation; and the patient must be kept in such a condition that, while life is preserved, the danger of inflammatory reaction in the region of the heart, and of fresh hæmorrhage, is reduced to a minimum. It is on this account that restorative measures are to be guardedly employed at first, and stimulants resorted to in the further progress of the case only when urgently indicated. Absolute rest of body and mind is indispensable. Nervine and cardiac sedatives, such as morphine, potassium bromide, chloral hydrate, and belladonna, may be of great service when used with judgment. J. MITCHELL BRUCE.

HEARTBURN.—**SYNON.** : Cardialgia.—Heartburn is a hot or scalding sensation, usually referred to the cardiac orifice of the stomach, but in some cases diffused over the whole abdomen. It is a marked symptom of many forms of indigestion, and is frequently accompanied by eructations of a very acid character: the fluid rejected from the stomach produces a sensation of scalding in the throat and œsophagus. See **DIGESTION, Disorders of**; **FLATULENCE**; and **STOMACH, Diseases of**.

HEAT, Effects of Severe or Extreme.—**(A) Constitutional Effects of Severe Heat acting generally.**—The constitutional or general effects of exposure of the whole body to high temperatures vary with the source and degree of heat, the slowness or rapidity of transition from lower temperatures, and the length of exposure, as well as with the age, constitution, habit, and health of the body; and they are liable to be more or less modified or obscured by various concomitant circumstances and conditions, such as the hygrometric state, and the purity or impurity of the atmosphere.

The range of temperature within the limits of which life can be maintained appears to be greater in the case of man than in that of most of the lower animals, in virtue of greater power of accommodation to external influences, without undue elevation or lowering of the temperature of the body. But in every case any such combination of external circumstances as causes the temperature of the body to rise 10° to 15° above the normal standard speedily proves fatal.

1. **Artificial heat.**—Numerous observations and experiments show that in *dry air* exposure to very high temperatures can be borne, during periods varying with circumstances, without danger or even serious inconvenience, the temperature of the body being kept down within safe limits by evaporation from the surface and from the lungs. Glass-workers, metal-founders, gas-stokers, engineers in steamboats—especially in the tropics—bakers' oven-builders, and others constantly carry on their work in temperatures of from 120° to 160° F. or even higher—to say nothing of the blasts of radiant heat to which some are from time to time exposed. In *moist air*, evaporation from the surface and its cooling influence being diminished or prevented, much lower tem-

peratures speedily become insupportable. Berger was unable to remain in a vapour-bath the temperature of which had risen from 106° to 120° F., although he had easily borne a temperature of 230° F. in dry air for five minutes. When the natural loss of heat is prevented by the moisture of a hot atmosphere—as in a vapour-bath, or by immersion in a hot-water bath, or otherwise—the temperature of the body rises. It has been found, taken in the rectum, to rise as high as 103° in eight minutes in a vapour-bath of 127° ; and in a hot-water bath of 104° to 111° the temperature taken in the mouth has been found to rise to 101.6° .

Fleming found that exposure in the Turkish bath during an hour to a temperature commencing at 170° F. and gradually lowered to about 130° F. caused the temperature of his body (taken by a specially devised thermometer in the mouth) to rise rapidly during the first ten minutes from a normal average of 97.65° to 99.2° F. (a rise of 1.55° F.); and then more slowly until the end of fifty minutes, when the highest point, 101.3° F. (a rise of 3.65° F.), was reached. His pulse rose during the first ten minutes from 78 to 91 beats in the minute, and like the temperature attained its maximum, 115, at the end of fifty minutes. His breathing first diminished in rapidity from 22.5 to 20.8, and then increased to 25.4 in actual rapidity, but maintained a diminished ratio to the pulse. The arterial tension seemed to be increased by the greater rapidity of the heart's action, combined with the engorged state of the capillary circulation. The quantity of material eliminated during the hour amounted to forty-four ounces. The proportion of chlorides in the urine passed after the bath (3.65 in the 1,000) was little more than half that in the sweat (6.05) collected during the bath, and much less than in the urine previously passed (5.68). The proportion of urea in the urine was slightly increased, and the sweat contained 1.55 in the 1,000.

The effects felt by those exposed to great heat vary with the temperature, the length of exposure, and collateral circumstances, such as an impure atmosphere and physical exertion, &c. A sensation of warmth, at first agreeable, is succeeded by one of oppressive and then painful heat, until this is again relieved by the establishment of copious evaporation from the surface. Pleasant stimulation, and some degree of excitement of the nervous and muscular systems, are quickly followed by languor, lassitude, listlessness, feelings of exhaustion, indisposition to mental effort or muscular exertion, dizziness, tendency to sleep, faintness, and unconsciousness, sometimes accompanied or preceded by convulsions. If relief is not afforded death ensues. If timely relief is afforded, more or less speedy and complete recovery may be brought about.

If the symptoms have been severe and persistent, or if the sufferer has been exposed to repeated attacks, permanent damage to the health, and especially to the nervous system, almost certainly results, in spite of apparent temporary recovery. In many cases general debility and deterioration, in some cardiac troubles, and in others insanity, have been recorded as the persistent after-effects. Symptoms and conditions closely or exactly resembling those of sunstroke may be produced by exposure to an artificially heated atmosphere, or to blasts of radiant heat from fires or furnaces. See **SUNSTROKE**.

Bernard, Delaroché, and others have shown that

animals exposed to temperatures of from 130° or lower to 150° F. and upwards quickly die.

The *post-mortem* examination of animals so killed showed that the heart had entirely ceased to beat a few seconds after death, and that neither it nor the muscular coat of the intestines could by any means be stimulated to contract again. The muscular fibres of the heart examined microscopically appeared rigid and coagulated (Kühne and Ranvier). Further, the blood in both arteries and veins was dark-coloured. *Rigor mortis* set in almost directly after death, and general decomposition very speedily commenced. Apart from these changes no lesions sufficient to explain the cause of death can be seen. The proteids of the nervous and muscular systems begin to undergo coagulation at 116·6° F. (47° C.), or even at a considerably lower temperature, if the exposure is prolonged. Such a coagulation in the nerve-cells and muscle-fibres would cause chemical and physical changes incompatible with life.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances met with in the human subject in cases in which death has been attributed to the general effects of heat have not been uniform, probably because there has been in most cases simultaneous exposure to other deleterious influences. It is probable that the changes leading to the fatal issue are microscopic, and are to be found chiefly in the nervous system. Recent observations by Goldscheider and Flateau, Marinesco, and Mott have shown that changes can be seen in the structure of the nerve-cells of animals and men who have died with hyperpyrexia. Nissl-bodies, composed of a nucleo-albumen, are not visible in such cases.

TREATMENT.—The *immediate treatment* to be adopted in the case of those suffering from exposure of the whole body to heat consists essentially in removal into a cooler and purer atmosphere; quiet rest in the recumbent position; fanning; cool or even cold affusions or sponging, especially over the head and spine—the effect, however, being carefully watched; and the administration of cool or lukewarm fluid in small quantity at a time, with some stimulant. Copious draughts of cold water in a highly heated state of body are liable to give rise to dangerous or even fatal results. Bleeding is not to be recommended.

The *after-treatment* must be conducted on general principles, and determined by the condition of the patient, and the indications afforded in the particular case.

2. Climatic and Solar Heat.—The effects of climatic heat experienced on transition from temperate to tropical regions are as follows: 1st. The average temperature of the body rises 5° to 1° F. according to Davy and Crombie, or to a somewhat greater extent according to others. The daily fluctuations of bodily temperature in health in India correspond to those in England. The normal temperature of native Indians is about half a degree higher than that of Europeans (Crombie). 2nd. The pulse is quickened according to most observers, but Rattray says this is incorrect. 3rd. The breathing becomes slower and less deep, falling from about sixteen to about twelve or thirteen per minute. Less carbonic acid and less water are exhaled. 4th. The skin acts much more freely, its excretion being increased by about 24 per cent. (Rattray). The continued hyperæmia and over-action of the skin, however, are liable to be followed by congestion and obstruction of the sweat-

ducts, giving rise to 'prickly heat.' 5th. The urine is lessened in quantity, and the urea and chlorides are diminished proportionately to the temperature as the latter rises from 70° F. to 90° F. 6th. The appetite, especially for animal food, is diminished, and the digestive powers seem lowered. 7th. Indisposition to exertion and a sense of exhaustion of mind and body are experienced in degrees varying with circumstances; the depressing effects being most felt when the heat is not only great but continuous day and night, and when the atmosphere is moist.

The effects of the radiant heat of the sun, as distinguished from those of atmospheric heat, are discussed in the article on **SUNBURN**.

Exposure to the direct rays of the sun, or to great or continued heat in the shade, especially under unfavourable atmospheric and general conditions, may give rise to heat-fever, heat-apoplexy, or one or other of the forms of sunstroke. See **SUNSTROKE**.

(B) Local Effects of Severe Artificial Heat: Burns and Scalds.—The local effects of heat vary with the degree, the length of exposure, the medium of application, and the part acted upon. *Burns* result from 'dry,' *Scalds* from 'moist' heat.

SIGNS AND SYMPTOMS.—(1) A comparatively slight degree of heat causes redness, tingling, pain, and tenderness, which soon subside. Desquamation of the epidermis may follow, but no permanent trace of injury is left. (2) A higher degree causes severe burning pain, and great redness of surface, followed by effusion of serum beneath the cuticle (vesication). Complete restoration without scar is usually effected. (3) Still higher degrees of heat or longer exposure cause intense pain, and immediate destruction of the true skin to a greater or less depth. Permanent scarring results. (4) Violent heat and prolonged exposure cause death and charring of the skin and structures beneath it down even to the bone. Loss of parts and more or less serious deformity and scarring necessarily result.

Only too frequently, in spite of such precautions as it is possible to take, the sloughs of burns become infected, pus forms, and sometimes widespread and destructive cellulitis develops. But suppuration is no necessary result of a burn.

The separation of sloughs, and the processes of repair after severe burns, take place slowly; and as a rule the patient suffers much more acutely, and during a much longer period, than after other forms of injury involving equally extensive destruction of tissue.

The *constitutional symptoms and effects* associated with burns and scalds vary with the superficial extent and situation, rather than with the depth of the injury. Thus an extensive burn or scald over the abdomen affecting only the skin is much more likely to prove fatal than charring part of an extremity; and burns over the front of the abdomen or chest are more likely to prove fatal than those of similar extent on the back. If half the surface of the body is even superficially affected the sufferer rarely recovers. Children suffer more than adults, and old age is unfavourable.

In severe cases death may result from shock, either immediately on receipt of the injury, or after a period of from two or three to forty-eight hours or more. During such time the sufferer remains in a state of collapse or prostration, with pale face, low temperature (due chiefly to decreased produc-

tion, partly to increased loss, of heat when epidermis has been widely removed), cool breath, small frequent pulse, dry tongue, and scanty urine; sometimes the patient is apathetic, at other times excitable and restless; thirst is common, vomiting occasional, convulsions or delirium rare. Hæmoglobinuria may be found. *Post-mortem* examination shows congestion of the viscera, especially the brain. The kidneys may have a reddish-brown colour, largely due to blood-pigment in the tubules. During life red corpuscles altered in form and even broken up have been seen; and after death the blood has often been described as thick, and thrombosis of vessels in many organs has been found.

More than fifty per cent. of the fatal cases occur within forty-eight hours, and the majority seem to be rightly attributed to *shock*. But there may be many contributory causes of greater or less importance. Thus Sonnenburg thinks some almost immediate deaths may be due to over-heating of blood and consequent paralysis of the heart. Other possible factors are: carbon dioxide; destruction of red corpuscles and accumulation of solution of hæmoglobin, forming a fluid which is destructive of leucocytes, thus setting free fibrin-ferment (leading to thrombosis) and other less-known toxic substances; and the accumulation in the blood of toxic materials which should be removed by kidneys and skin.

In about forty-eight hours, more or less, the stage of reaction sets in. The patient revives, and some degree of general pyrexia becomes manifest. Too often it is of septic type, and is accompanied by inflammation and suppuration of the burnt part. An acute septicæmia with erythematous or scarlatiniform rashes is not uncommon in children. The tendency to congestion of the abdominal and thoracic viscera persists, and sometimes runs on to actual inflammation (septic or toxic?). But the most singular lesion of this period is ulceration of the duodenum between the pylorus and the papilla of Vater. The presence of such an ulcer may be indicated by epigastric pain after food, and perhaps hæmatemesis or mælena. Often perforation into the peritoneum or fatal bleeding from an eroded artery occurs without warning. The complication is rare and not necessarily fatal. Embolism of a small artery and autodigestion is a suggested explanation of the occurrence, but not of the localisation of the ulcer. William Hunter produced similar ulcers in dogs by the intra-venous injection of toluylene-diamine, and suggests that in burns the cause may be a toxin eliminated with the bile. During this period, and from the causes indicated, death occurs in about 30 per cent. of the fatal cases.

In a fortnight or so, as a rule, acute sepsis will have subsided, many sloughs will have separated, granulation and suppuration will have become established, and healing will gradually take place. But large sloughs take weeks to separate, and where the deep fascia is opened serious cellulitis may cause much trouble and chronic fever; or the patient may sink, worn out by suffering, and exhausted by the profuse discharge from the suppurating surface, or by persistent diarrhoea, accompanied or not by blood in the motions. Sometimes the kidneys are affected, and blood and blood-casts may be found in the urine. Pyæmia, erysipelas, or tetanus may occur and cause death; but there would not appear to be any special liability to these diseases after burns or scalds.

TREATMENT.—1. *The Relief of Pain.*—It is said that the pain of slight burns may be relieved by plunging the part into cold water, or into hot water, or by holding it in front of a fire. Elevation of the part, gentle compression with some soft material, and uniform warm temperature act best. In more serious cases suffering should be relieved by morphine hypodermically.

2. *The Dressing.*—When hyperæmia only is induced protection of the part by a bandage over a thin layer of wool or gauze is sufficient. When vesicles rise, they should be somewhat freely opened and their contents evacuated; but the epidermis should not be removed, as it forms the best protection to the subjacent rete. Careful handling is necessary to avoid denudation of raw surfaces. Clothes should be cut rather than pulled off, and in bad cases their charred remains are best floated off in a hot bath.

When once vesication has been caused, the possibility of infection arises. The first object of the dressing is to prevent this: it must absorb any discharge, prevent its decomposition, and be comfortable. Some recommend that the burnt area shall be gently washed with a feeble antiseptic, such as boric acid or dilute carbolic lotion. If the parts have not been sterilised by heat, they will not be by such applications. In limited cases, some attention may be paid to the cleansing of skin around the injured area.

The early dressings, especially of burns destroying only the surface of the skin, are often excessively painful, so that anaesthesia is necessary in children and may be so in adults. The less frequent the dressing, therefore, the better. Ointments and oily preparations absorb nothing, do not mix with discharges, and do not prevent their decomposition. Such dressings only cover the burns, protect them from mechanical irritation, and peel off more or less easily.

The choice of dressing lies really between a 'dry' dressing and one kept moist by a covering of waterproof tissue. The former is the better if it can be successfully worked. It should consist either of simple sterilised gauze or of double cyanide gauze wrung as dry as possible out of 1:2000 perchloride of mercury lotion. Either should be applied several layers thick, and should be covered with a good layer of salicylic or other wool and fixed with a bandage. Such a dressing absorbs discharges and prevents decomposition of them chiefly by dryness due to ready evaporation. If the discharge is at all free it is as well to place deep in the gauze a layer of some powder, such as aristol, or equal parts of powdered boric acid, zinc oxide, and kaolin. Under this treatment thin superficial sloughs are dried and gradually cast off, granulation and healing taking place beneath them. So long as the patient is comfortable and the temperature normal, such a dressing may be left. When it is necessary to change it, the wool and as much gauze as can be removed without pain is taken off: the rest is thoroughly soaked with warm sterile saline solution from a syringe or in a bath.

Another good form of dry dressing is the following. In cases in which the whole thickness of the skin is not destroyed, lint soaked in a saturated solution of picric acid may be uniformly applied to the burnt surface, after opening all vesicles, and allowed to dry on under a layer of cotton wool.

Sometimes, however, infection of the sloughs

occurs, pus accumulates beneath them and does not escape readily into the dry dressing, but accumulates in contact with the living tissues. Pain and fever result. If removal or partial removal of the slough is practicable, it may yet be possible to use a dry dressing, or to revert to it after short employment of a moist dressing.

The moist dressing consists either of several layers of gauze or of two or three of boric lint wrung very dry out of boric lotion or a dilute mercury lotion. This is covered by a layer of oiled silk projecting half an inch beyond the lint in all directions; over this are placed a layer of wool and a bandage. This dressing quickens the separation of sloughs, facilitates the escape of discharges, and prevents their decomposition; but to accomplish these results the dressing must be changed once, twice, or more frequently during the twenty-four hours, and this may be a painful process.

3. *Removal of Dead Parts.*—Parts of limbs which have been completely charred, or which have lost so much skin that recovery with usefulness is impossible, should be removed by amputation as soon as shock permits. But when there is doubt as to the completeness of destruction of the skin, as is often the case in both burns and scalds, an antiseptic dressing must be applied until the evidence on this point is clear. Sloughs should be removed if discharge is bagging beneath them.

4. *The Prevention of Deformity.*—The more slowly a granulating sore heals, the more deeply does the granulation-tissue penetrate, and the greater is the resulting contraction. Speedy healing should, therefore, be aimed at in all cases of wide destruction of the epidermis by Thiersch's method of skin-grafting. An affected limb must be kept upon a splint until healing is complete, occasional opportunities for performing the movements of the fixed joints being allowed. It is said that the permanent warm bath diminishes contraction. For the remedying of deformities due to burns, plastic operations are often necessary.

5. *The Treatment of Shock.*—In extensive burns, even though superficial, the immediate danger to life is great. It must be met by keeping the patient in a bath at about 105° F., by the free infusion of saline solution into veins or subcutaneous tissue, the hypodermic injection of strychnine, and the administration of hot milk and coffee or of hot brandy and water. See SALINE SOLUTIONS, Infusion of.

(C) *Local Effects of Severe Solar Heat: Sunburn.* See SUNBURN.

M. S. PEMBREY. STANLEY BOYD.

HEAT, Therapeutic Use of.—Heat is employed in the treatment of disease as a general or local stimulant, a caustic, or a counter-irritant; and that in the form either of *dry* or of *moist* heat. See BATHS; COLLAPSE; CHOLERA; COLIC; COUNTER-IRRITANTS; FOMENTATIONS; POULTICES; and RESUSCITATION.

HEAT-STROKE.—A synonym for sunstroke. See SUNSTROKE.

HECTIC FEVER. See FEVER; and TEMPERATURE.

HELMINTHIASIS (ἐλμινς, a worm).—Disease characterised by the presence of worms.

HELMINTHS (ἐλμινς, a worm).—See ENTOZOA.

HELOUAN, near Cairo, Egypt.—Thermal sulphurous muriated saline waters. See MINERAL WATERS.

HEMERALOPIA.—DEFINITION. — Day-blindness: a disorder of vision in which objects cannot be seen well or comfortably in ordinary daylight or by strong artificial light, but are seen more clearly or more comfortably in shade or in twilight.

Most modern authors use the term in the opposite sense of *night-blindness*, regarding it as derived from *ἡμέρα*, the *day*, and *ὤψ*, the *eye* or *sight*. In this work it has, from the first, been used in the sense given above, the *al* being regarded as representing *alpha privativum*, or as a contraction of *ἀλαός*, *blind*. This meaning has since been adopted in the *Nomenclature of Diseases* of the Royal College of Physicians. It has long been used as the antonym of *Nyctalopia*. See NYCTALOPIA.

CAUSES.—Day blindness is a symptom rather than an idiopathic affection, and is sometimes the effect of mere intolerance of light (photophobia), dependent upon irritation or inflammation of the cornea, &c. The various causes may be grouped as follows:—

1. Opacity in some of the refractive media, such as central nebula or leucoma of the cornea, or circumscribed opacities occupying the pupillary region of the lens or the vitreous. In such cases, vision is obscured when the pupil is contracted, but becomes clearer when the pupil dilates in shade. 2. Irritation or inflammation of the parts of the eye innervated by the sensory fibres of the fifth nerve—the lids, the conjunctiva, the cornea, or the iris; neuro-pathic affections of these structures; and reflex irritation from nose, teeth, uterus, &c. 3. Hyperæsthesia of the retina, which may be due to reflex irritation, or (rarely) to inflammation of the retina, the optic nerve, or the choroid, to insufficient pigmentation of the ocular fundus as in albinism, to dazzling from coloboma of the iris or preternatural dilatation of the iris from any cause, to exposure to bright lights or glare of any kind, to prolonged seclusion in darkness or dimly lighted places. A transient physiological form of hemeralopia occurs whenever the eyes suddenly pass from darkness into bright light.

TREATMENT.—The treatment of hemeralopia should follow the special indications. Impairment of sight due to central opacity in any of the refractive media may often be relieved by dilatation of the pupil with a weak solution (gr. $\frac{1}{20}$ to gr. $\frac{1}{2}$ to an ounce) of atropine or other mydriatic, or by making an artificial pupil. Inflammations of the various structures of the eye and reflex irritations are to be treated by appropriate means. The eyes should be protected by a shade or by neutral-tinted glasses. The general treatment must be adapted to the associated local and constitutional conditions.

JOHN TWEEDY.

HEMIANÆSTHESIA (ἡμι-, half; ἀ-, priv.; and αἰσθάνομαι, I feel).—Paralysis of sensation, affecting one side of the body. See SENSATION, Disorders of.

HEMIANALGESIA (ἡμι-, half; ἀ-, priv.; and ἄλγος, pain).—Insensibility to painful impressions, affecting one side of the body. See SENSATION, Disorders of.

HEMIANOPIA (ἡμι-, half; ἀ-, priv.; and ὤψ, sight).—The term 'hemiopia' is now generally

taken to mean loss of perceptive power in one lateral or vertical half of the *retina*; while by hemianopia is understood obscuration of vision on one lateral or vertical half of the *visual field*. Thus, owing to the fact that rays of light cross within the eye, right lateral hemianopia is produced where left lateral hemiopia exists, as a result of intracranial disease of one or other kind. See NERVOUS SYSTEM, Examination of.

It is well known that in the lower vertebrate animals the optic nerves undergo a complete decussation at the optic chiasma, so that all impressions from each eye go to the opposite optic lobe. Some authorities believe that a similarly complete decussation of the optic nerve-fibres takes place at the chiasma in man; but the majority adopt the view that at the optic chiasma there occurs a semi-decussation only. It seems quite possible that these discrepancies in doctrine may be in part dependent upon the fact that the course of the optic fibres is not the same in all cases. There may, in fact, be variations in the degree of completeness of the decussation of the optic nerve-fibres at the chiasma, just as, from the researches of Flechsig, we now know that not infrequently considerable variations occur in the amount of decussation of motor fibres in the anterior pyramids of the medulla.

The effects resulting from congenital or early blindness of one eye supply us with some information bearing upon this point. Where a semi-decussation exists, the effects of the atrophy of the optic fibres proceeding from the affected eye should, beyond the chiasma, be pretty equally distributed, so that, though in such a case the two optic nerves will be very dissimilar in size, the optic tracts ought to be of equal bulk; and this is what is most commonly met with. On the other hand, it is undoubtedly the fact that in some of such cases the optic tract on the opposite side is found to be notably atrophied, while that on the same side as the atrophied optic nerve presents a healthy appearance—signs which betoken a pretty complete decussation of the optic fibres at the chiasma.

Different forms of Hemianopia and their causes.—In the cases where the common arrangement obtains—that is, the semi-decussation—it seems to be the inner fibres that cross one another, while the outer fibres of each nerve are continued on into the outer part of the corresponding optic tract. The result of this arrangement would be that the fibres from the right half of each retina would proceed to the right optic tract, while those from the left side of each retina would go to the left optic tract; so that if the image of any object should fall simultaneously either on the right side of the two retinæ, or on the left side of the two retinæ, the impressions in either case will be transmitted along one and the same tractus opticus—the right or the left, as the case may be. Therefore it seems clear that, in all cases in which the semi-decussation of the optic fibres obtains, any intracranial lesion which either destroys, seriously presses upon, or disturbs the nutrition of the optic tract on one side, will give rise to an opposite *lateral hemianopia*. If the decussation had chanced to be total, there would, on the contrary, have been produced a ‘crossed amblyopia.’

The optic tract is apt to be involved in some part of its course between the chiasma and the geniculate bodies by different kinds of lesions. Thus, we may have tumours originating in parts of the tract itself; or the tract may become involved secondarily, or

pressed upon, by some tumour or hæmorrhage occurring in the *crus cerebri* or in the posterior tubercle (pulvinar) of the thalamus, or by a tumour originating in the inner and under part of the temporal lobe, or from the bone itself in the middle fossa of the skull.

Two other, much rarer, forms of hemianopia require to be mentioned, which are produced by lesions affecting different parts of the chiasma. One of these is double *temporal hemianopia*, which may be produced by a lesion pressing upon the anterior median portion of the chiasma. Any lesion capable of doing this would probably be a tumour, and it would produce the same effects whether there is a total or a semi-decussation in the chiasma. The lesion would involve the decussating fibres that come from the inner half of the retina on the two sides, and thus would lead to double temporal hemianopia. The other form is known as *nasal hemianopia*, and where semi-decussation of the optic fibres exists it would be produced by a tumour or an aneurysm causing an injurious amount of pressure upon either outer angle of the chiasma, since there would then be paralysis of the fibres emanating from the outer part of the retina, and consequently the suppression of the inner part of the visual field of the corresponding eye. It is only with extreme rarity that this form of hemianopia is double.

We have now to turn to another side of the question. It is natural to ask whether the lateral hemianopia that develops as a consequence of disease of the optic tract will also result from a lesion which involves the optic fibres beyond the *corpora geniculata*—that is, in their deep intracerebral course. This was the view put forward by von Graefe in 1860, and one which was very commonly accepted till it was challenged by Charcot some few years since. Relying apparently upon the paucity of cases on record in which lateral hemianopia had resulted from lesions of the cerebral hemispheres themselves, and upon the many cases that had come under his own observation in which ‘crossed amblyopia’ had been produced by functional or structural lesions involving the posterior or sensory segment of the internal capsule, he came to the theoretical conclusion that all the optic nerve-fibres from each eye went to the opposite cerebral hemisphere, owing to the supposed fact that those fibres which had not decussated at the chiasma did decussate somewhere in the *corpora quadrigemina*. These views he embodied in a well-known diagram. The subsequent progress of knowledge has, however, not been compatible with this theory. Thus Munk’s original statement has been confirmed by Schäfer and S. Brown, to the effect that removal of one occipital lobe in the monkey produces, not crossed amblyopia, but opposite lateral hemianopia. Again, Seguin has shown that opposite lateral hemianopia is more especially produced by lesions in that inner portion of the occipital lobe known as the ‘cuneus,’ a region supplied by a rather large branch of the posterior cerebral artery which passes along the calcarine fissure. Still it would appear that lesions anywhere in the posterior parietal, temporal, or occipital region of the hemisphere, when they implicate the ‘optic radiations’ of Gratiolet (i.e. fibres passing backwards from the *corpora quadrigemina* and *geniculata* to the occipital cortex), are also capable of producing an opposite hemianopia in man of a permanent character. In the lower animals (monkey, dog, &c.), for the production of a similar defect the removal of the occi-

pital lobe must be complete, and even then the defect is of a comparatively brief duration—two or three weeks at most.

There is at present an apparent contradiction between these well-attested facts, as to the production of lateral hemianopia from disease of the occipital lobe (implying as they do that the semi-decussation persists even to the cortical termini of the optic fibres), and the fact of the existence of a 'crossed amblyopia' in association with functional or structural disease of the hinder portion of the internal capsule. A solution of this difficulty cannot as yet be readily found. Ferrier still maintains that crossed amblyopia is produced by destruction of one angular gyrus. He asserts, indeed, that 'the angular gyri are more particularly the centres for clear vision, each mainly for the eye of the opposite side.'

DIAGNOSIS.—Under this head reference has to be made to different sides of the problem. The first point to be considered is the mode of recognition of the different varieties of hemianopia. In some cases, where the defect is well marked, and the patient is intelligent, he will himself call the observer's attention to it; very frequently, however, such defects are only discovered by being specially looked for. This subject will be referred to under **VISION, Defects of**. Here it must suffice to say that for all accurate observation an instrument named a 'perimeter' is required, by the aid of which the exact nature and extent of the limitation of the visual field can be discovered; but, for a first rough examination, the following simple method will suffice for the detection of any great narrowing of the visual field in particular directions. The patient stands with his back to the light, and, covering one eye, looks steadily from a distance of eighteen inches at the nose or eye of the observer, who then moves his forefinger about in different parts of the field of vision, and notes any part where the patient says it is invisible or badly seen. Anything like a hemianopic defect, of either variety, may be easily appreciated in this manner.

The fact of the existence of hemianopia having thus been established, we must next seek to determine the site of the lesion by which it has been caused. The problems of regional and of pathological diagnosis are in these cases often intimately related to one another. With respect to the latter problem some hints have already been given.

For purposes of regional diagnosis the first thing is to determine whether the case before us belongs to one of the other of two main categories into which cases of hemianopia are divisible—that is, whether it is a case of '*tract*' or of '*central hemianopia*'; and this may be determined by a delicate test first used by Wernicke. This test is based upon the fact that, in the case of lesions in the primary optic centres of one side (the corpus geniculatum and one anterior quadrigeminal body), or of their afferent fibres, there is an interference not only with visual impressions, but also with the path for impressions that excite pupillary contractions through these oculo-motor centres; whereas in the case of deeper lesions, situated in parts of the cerebral hemisphere involving either the optic radiations or the occipital cortex, the pupillary reflex would not be at all interfered with. Thus in cases of '*tract hemianopia*' there exists what has been termed hemiopic pupillary inaction; while in cases of '*central hemianopia*' this sign does not exist. The sign itself is

sought for by throwing a pencil of light obliquely (at an angle of 40° – 60°), by means of an ophthalmoscope mirror, on to the blind side of the retinae, so as to see whether contraction of the pupil is, or is not, produced. If carefully performed, no reaction of the pupil occurs in cases of '*tract hemianopia*'—that is, where the defect is due either to disease of the afferent fibres, or of the primary optic centre itself. But where the hemianopia is caused by disease in the cerebral hemisphere itself (*central variety*) the contraction of the pupil occurs equally well whether the light be thrown upon the blind or upon the unaffected half of the retina.

From the point of view of a regional diagnosis, we may enumerate four fairly marked types of '*central hemianopia*'.

1. There are cases in which hemianopia exists alone, or as the most prominent symptom, or in association with certain general signs indicative of the existence of tumor cerebri. Such are the cases where the occipital lobe is affected in or about the region of the cuneus.

2. Cases in which hemianopia co-exists with hemianæsthesia and choreiform or ataxic movements of one half of the body, without marked hemiplegia. These are probably due to a lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule, on the side of the brain opposite to the dark half-fields and the hemianæsthesia.

3. Cases of hemianopia with complete hemiplegia and hemianæsthesia are probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

4. Cases of hemianopia with aphasia and hemiplegia, with or without hemianæsthesia, are probably due to an extensive superficial lesion of the area supplied by the left middle cerebral artery, such as may be brought about by its plugging either from thrombosis or embolism.

In all these cases the pupillary reaction would not be interfered with, and there would be no atrophy of the optic nerves unless as a sequence of choked disc. In addition to these forms, five types of '*tract-hemianopia*' may be enumerated.

1. The first type of tract-hemianopia would be caused by lesions in the primary optic centres on one side (the corpus geniculatum and one anterior quadrigeminal body). Little or nothing is known of lesions thus limited, which are excessively rare. Still, such parts may be involved primarily or secondarily, by a tumour; when among a varying group of symptoms we might have, as results of the lesion in the parts specified, lateral hemianopia, hemiopic pupillary inaction, together with early atrophy of the optic nerves.

2. In other cases where disease involves the optic tract and the crus cerebri, we may have hemianopia co-existing with some form of cross-paralysis. A probable combination would be paralysis of the third, fourth, and sixth cranial nerves on the side of the lesion (some one or all of these nerves), together with partial hemiplegia (without anæsthesia), and hemianopia on the opposite side. In some cases choked discs would appear early, without interfering with the hemiopic symptoms until optic atrophy with complete blindness had become established.

3. In cases where the optic tract is involved more anteriorly, other symptoms co-existing with the hemianopia may be very vague and uncertain;

but the hemiopic pupillary inaction should be marked, and there should be early partial atrophy of the optic nerves. These latter signs suffice to distinguish this group of cases from those belonging to the first category of 'central hemianopia,' with which otherwise they might be confounded.

4. Cases of bitemporal hemianopia due to lesions pressing upon the anterior part of the chiasma.

5. Cases of nasal hemianopia, mostly single, due to a lesion involving the outer part of the chiasma.

The only other forms of monocular hemianopia are also very rare, and are due to lesions of the optic nerve itself in front of the chiasma.

Other very rare forms of hemianopia affect the superior or inferior, rather than the lateral, segments of the field of vision. As Seguin points out, when these forms exist with a very irregular demarcation-line, they are probably still more peripheral in their origin and due to embolism or thrombosis of the central artery of the retina.

In another class of cases, instead of a definite hemiopic defect of sight, we meet with partial, quadrant- or sector-like defects in the upper or lower halves of the visual fields. Little or nothing is known as to the causation of such defects. They might conceivably be caused by disease in certain limited portions of the occipital cortex, or of some only of the fibres constituting the optic radiations. Again, they might be due to a localised damage to some of the fibres of one crus or one optic nerve; or to degeneration of the retina caused by the plugging of some one branch of its central artery.

PROGNOSIS AND TREATMENT.—Nothing special can be said under these heads concerning the hemianopia itself. Both the prognosis and the treatment of this defect are absolutely dependent upon the causative conditions, and the nature and severity of the collateral symptoms with which it is associated. It may in some cases not be dependent upon an actual organic lesion, but be one among other indications of mere functional defect. We must, in fact, always seek to mitigate or remove the underlying causal conditions, and upon our chance of being able to do this the prognosis in any given case entirely depends.

H. CHARLTON BASTIAN.

HEMICRANIA (*ἡμι-*, half; and *κρανίον*, the head).—Pain limited to one side of the head. The term is, however, generally used as synonymous with *migrain*. See *MEGRIM*.

HEMIOPIA
HEMIOPSIA } (*ἡμι-*, half; and *ὥψ*, sight). -

A loss of perceptive power in one lateral or vertical half of the retina. See *HEMIANOPIA*; and *VISION*, Disorders of.

HEMIPLEGIA (*ἡμι-*, half; and *πλῆσσω*, I strike).—Paralysis of motion of one side of the body; sometimes applied to loss both of motion and of sensation. See *PARALYSIS*; *BRAIN*, *Hæmorrhage* in the; *BRAIN*, *Softening* of; *BRAIN*, *Tumours* of, &c.

HEPATALGIA (*ἥπαρ*, the liver; and *ἄλγος*, pain).—Strictly this word signifies pain in connection with the liver. It has, however, been specially applied to a supposed neuralgic pain referred to this organ, coming on in paroxysms, and said to be of a severe character in some instances, so as to simulate hepatic colic. Whether there is any such

affection is exceedingly doubtful; and probably in cases of supposed hepatalgia the neuralgia is either superficial, or there is some tangible but undiscovered cause for the pain, connected with the hepatic apparatus or some neighbouring structure. See *PAIN IN VISCERAL DISEASE: Coloured Plates*.

FREDERICK T. ROBERTS.

HEPATISATION (*ἥπαρ*, the liver).—A term applied to the condition produced by acute inflammation of the lung, in which the pulmonary substance becomes solid and friable, somewhat resembling the liver in its physical characters. See *LUNGS*, *Inflammation* of.

HEPATITIS (*ἥπαρ*, the liver). Inflammation of the liver. See *LIVER*, *Inflammation* of.

HEPATOCELE (*ἥπαρ*, the liver; and *κῆλη*, a tumour). Hernia of the liver. See *Liver*, *Displacements* of.

HEPATOPTOSIS.—See *LIVER*, *Malformations* of.

HERCULESBAD (HERCULES-FURDO), in Hungary.—Thermal sulphur and muriated saline waters. See *MINERAL WATERS*.

HEREDITARY ATAXIA.—Hereditary Cerebellar Ataxia. See *FRIEDREICH'S DISEASE*.

HEREDITY.—By heredity is meant that special property through which the characters and qualities of organisms are transmitted to their descendants throughout successive generations, so that the offspring in their main features resemble their parents. The transmission of parental characters to offspring is a subject for scientific inquiry. It has long been known that a young organism is derived from an ovum formed within the body of the female parent, so that a descent from the mother and a direct continuity through the egg with her structure was apparent. But it is within a comparatively recent period that the direct participation of the male parent in the production of the offspring has been traced. The older writers thought that the semen evolved a vapour or spirit, which influenced the ovum, or that the fluid part of the semen penetrated the vitelline membrane, and induced germinative changes, which resulted in the production of a young organism, without the male contributing any definite structure in the process. The discovery, in 1842, by Martin Barry, of the penetration of the vitelline membrane by the spermatozoon put the subject on a more definite basis, and the researches of Bütschli, Auerbach, Föl, Strasburger, Flemming, Hertwig, E. van Beneden, and others, have given greater precision and completeness to our conceptions of the process of impregnation. It has now been demonstrated that the young organism arises within the egg by the incorporation, or conjugation, of an extremely minute particle derived from the male parent with an almost equally minute particle derived from the female parent. The particle derived from the male is the head of the spermatozoon, which, after it has penetrated within the ovum, is called the male pronucleus. The particle derived from the female is a portion of the germinal vesicle, which has remained included within the egg, and is called the female pronucleus. The male and female pronuclei gradually approximate, touch, and then become incorporated with each other, and

form what is called the segmentation-nucleus. The segmentation-nucleus consists of chromatin-fibres and nucleoplasm, which are derived both from the nucleus of a male sperm-cell, i.e. the head of a spermatozoon, and from the nucleus of a female germ-cell, i.e. the germinal vesicle. The segmentation-nucleus is therefore a composite or hermaphrodite nucleus, and consists of matter derived from both parents. This composite nucleus then rapidly divides and subdivides within the ovum, so as to produce daughter-nuclei, and the protoplasm of the yolk undergoes a corresponding process of division. Each of the daughter-nuclei becomes surrounded by a layer of protoplasm, and numerous new cells are produced. The nucleus of each of these new cells contains chromatin-fibres derived from the segmentation-nucleus, and through it from the corresponding fibres of the nucleus of the male sperm-cell and of the female germ-cell. It is believed that the male and female chromatin-fibres enter in equal proportions into the structure of the nuclei derived by division from the segmentation-nucleus, so that each of the nuclei of the new cells, like the segmentation-nucleus, is composite or hermaphrodite, i.e. represents both parents. The cells produced by the division of the segmentation-nucleus and its envelope of yolk-protoplasm form the blastodermic membrane. In man and the higher organisms this membrane consists of three layers of cells, named epiblast, mesoblast, and hypoblast. From these layers, by a continuous process of division and subdivision of the nuclei of their constituent cells, accompanied by a differentiation of the protoplasm, all the tissues and organs of the body are descended. As the nuclei of all the cells in the body are derived by continuous descent from the nucleated cells of the blastoderm, and as these again are the descendants of the segmentation-nucleus, one may infer that the nucleus of each cell is composite or hermaphrodite and represents both parents.

The changes which take place in a nucleus during its division constitute the process of *karyokinesis* or *karyomitosis*. They consist at first in an enlargement and rearrangement of the chromatin-fibres of the nucleus, and then in a splitting of each fibre into two daughter-threads, so that each daughter-thread divides itself equally between the two daughter-nuclei which are formed by the splitting of the parent-nucleus. As this process goes on throughout the whole period of cell-multiplication, there is an actual transmission of structure from the segmentation-nucleus to the nuclei of all the cells derived both directly and indirectly from it. Now, as this descent in structure is in all probability associated with a power of transmitting properties, and as the segmentation-nucleus consists of material derived from both parents, a structural medium is provided for the transmission of the characters of the parents to their offspring, and the phenomena of heredity may be said to rest on a physical basis.

The material which forms the structure of the male and female pronuclei and the segmentation-nucleus has been termed by Weismann the *germ-plasm*. He believes that in each individual produced by sexual generation, a portion of the germ-plasm derived from both parents is not employed in the construction of the nuclei of the cells and tissues of the soma or personal structure of the individual, but is set aside unchanged for the formation of the germ-cells of the succeeding generation. According

to this theory there is a direct continuity of germ-plasm from one generation to another, which acts as the conveyor of hereditary characters. Weismann maintains that the germ-plasm is not modified by the habits and mode of life of the individual, and he conceives it to be transmitted from one generation to another uninfluenced by the conditions of life in which the individual is placed. He assumes that the reproductive cells which contain the germ-plasm are not acted on by the organs and tissues of the body in which they are situated, except in so far as they may be affected by general conditions of growth and nutrition. Hence, he concludes that characters acquired by individuals during their lifetime from any external cause, which modify the persona or soma, cannot be transmitted to their offspring.

Several arguments may, however, be advanced in opposition to Weismann's conclusion, which seems to require for its confirmation that the germ-plasm of the reproductive cells should, from the commencement of the development of the embryo, be isolated from the cells from which the other organs of the body are derived. But both reproductive cells and somatic cells are formed in the fertilised ovum by the segmentation of the nucleus, and the reproductive cells in man and other vertebrates do not appear as isolated organs until after the rudiments of all the great organic systems have been mapped out. Hence, before they are set aside as germ-cells or sperm-cells, the germ-plasm has apparently been in a stage of diffusion, and under precisely the same influences as those which in the embryo affect the formative cells of the body generally, so that the conditions which would secure the germ-plasm and the somatic cells from mutual interaction are not complied with.

Further, there is experimental evidence to show that in placental mammals an interchange of material takes place during gestation in opposite directions from foetus to mother, as well as from mother to foetus. A character derived by a foetus by descent from its male parent may thus be acquired by the mother from the foetus, and influence the germ-plasm of her reproductive cells so as to be transmitted to her future offspring, even though they may not have the same male parent. Hence, it is an axiom with those engaged in the breeding of particular kinds of stock, that, if they wish to keep the strain pure, the mother must not at any time be allowed to beget offspring by sires of another blood.

The hereditary transmission of characters from parents to offspring is not limited to such as are normal and physiological, and of service in the perpetuation of the species and of the race. Characters which are of no service, and, indeed, are detrimental to the individuals in whom they occur, may also be transmitted, and it is from this point of view that the subject of Heredity requires to be considered in this place. The study of those modifications in the structure of the body, which pathologists group together under the name of Congenital Malformations, has furnished numerous illustrations of the descent in families of variations from the normal structure. Familiar examples are furnished in the presence of supernumerary digits on the hands or feet in some families, or a diminution in their number in others. But modifications or variations in structure that can be transmitted from parents to offspring are by no means limited to changes which can be recog-

nised by the naked eye. They are sometimes so minute as to be determined rather by the modifications which they occasion in the functions of an organ than by the ready recognition of structural variations. One might adduce as an example Daltonism, or colour-blindness, which has distinctly been shown to be hereditary, and which is due apparently to a minute structural defect in the development of the retina, or the optic nerve, or the brain itself, occurring in particular families.

But there are certain diseases which occur in some families more than in others, and which are regarded as hereditary. Thus there is a strong tendency to the production of cataract in successive generations, and in other families an equally strong predisposition to the hæmorrhagic diathesis. When we speak of tendencies, susceptibilities, proclivities, or predisposition to the transmission of characters, whether they be those of health or of disease, we employ terms which undoubtedly have a certain vagueness. We are quite unable to recognise, even with the highest powers of the microscope, any structural difference in the germ-plasm in different persons which would enable us to say that in one family a particular feature should have one form, in another family a different form; or that in one family there should be a tendency or susceptibility to one kind of disease, while another family should display a special capacity for transmitting another form of disease. We can only determine that such a sequence will take place by tracing the life-history of the individuals belonging to these families. Though, as we have said, these terms have a certain vagueness, and are wanting in scientific precision, yet it is not the less true that they express a something of the importance of which we are all conscious, though we may not be able to formulate it in a precise definition. WILLIAM TURNER.

HERMAPHRODITE (*Ἑρμῆς*, Mercury; and, *Ἀρροδῖτη*, Venus).—A term applied to an individual in whom the formation of the sexual organs is such as to give rise to the impression that both the male and the female organs are present. See MONSTROSITIES.

HERNIA (*hernia*, a rupture).—SYNON. : Fr. *Hernie*; Ger. *Bruch*.

DEFINITION.—By a hernia is meant the protrusion of an organ from the cavity in which it is normally confined. The intestine is the part which most commonly suffers from this form of displacement, and consequently the term 'hernia,' without any qualifying epithet, usually means protrusion of some part of the gut from the abdominal cavity, and is equivalent to the popular expression 'a rupture.' Other organs to which the term is applied are the brain (*hernia cerebri*), the lung (*hernia pulmonis*), and the testicle (*hernia testis*). All the abdominal organs, with the exception of the pancreas, have at one time or another been found among the contents of hernial protrusions. In the present article the forms of hernia to which the abdominal viscera are liable are alone considered.

CAUSATION.—In order to establish the treatment of hernia on a scientific basis, it is necessary to study the mechanical factors that are responsible for its production. The form of the abdomen remains normal only so long as there is a proper relationship between the intra-abdominal tension and the resistance offered by the abdominal wall. If the

intra-abdominal tension be increased to an extent beyond that which can be borne by the muscular and tendinous surroundings, a protrusion of a portion of the contents results in the situation of least resistance. For instance, in infancy the intra-abdominal tension may be rendered excessive by the presence of gas in the intestines resulting from indigestion. In addition to the gas there is often present a variable quantity of fluid in the peritoneal cavity resulting from the same cause. This produces in the young infant a general distension of the abdominal wall, and a progressive yielding of the recently cicatrised umbilicus, forming an umbilical hernia. Fluid and bowel may be forced singly or together into the inguinal canal forming inguinal hernia or hydrocele in the male or female. A distinct ventral hernia may occasionally result from the same cause, the fibrous tissues between the recti muscles yielding under great strain. Later, in vigorous life, the intra-abdominal tension may be raised to such a pitch by the violent contraction of the abdominal wall that some of the viscera may be driven along the inguinal canal, the escaping structures gradually acquiring a peritoneal covering or occupying a still unobliterated process of peritoneum. This may occur suddenly, as in lifting a heavy weight, or in a violent effort of coughing, or straining to expel feces; or it may result gradually from a succession of such efforts. On this account certain occupations render the labourer particularly liable to this accident. Later in life the intra-abdominal tension may be greatly increased by the abundant formation of fat in and about the viscera, while the resisting-capacity of the wall is diminished in a corresponding manner by its deposit in and between the muscular planes.

In the female intra-abdominal tension is increased very rapidly by the pregnant uterus or by tumours in connection with the ovary and uterus. In this sex the inguinal canal is much more secure than in the male, while the femoral canal is much less so.

The resisting power of a portion of the abdominal wall may be reduced by some injury to it, such as an incision, and the resulting cicatrix may yield under the strain to which it is habitually exposed.

In considering the treatment of any variety of hernia the surgeon must therefore consider how he can best reduce the intra-abdominal tension, and how he can most effectually restore to the abdominal wall its original integrity and resisting-power. A hernia may have existed for so long that, to effect a cure, recourse must be had to operative interference in order to restore to the defective portion of the abdominal wall its normal structure and formation. Even after this has been effected satisfactorily the surgeon must see that the same mechanical conditions which originally produced the rupture do not continue to exist after his operation; or if they must still do so, as in the labourer, chronic bronchitic, &c., he should supplement the resisting-capacity of the abdominal wall by means of an efficient support or truss.

In the case of a hernia the contents of which can be reduced within the abdominal cavity, it is necessary not only to adopt such measures as will tend to reduce the intra-abdominal tension and increase the resisting-power of the abdominal walls, but also to maintain such constant pressure upon the aperture of exit from the abdomen, or to place opposite it such a rigid resistance, as will effectually prevent any subsequent escape of the abdominal

contents through it. The sac and its coverings exist only as long as the pressure which the intestines exert upon them continues in action. On removing this pressure by preventing the escape of the intra-abdominal contents, the sac gradually shrinks up and disappears. It is usually supposed that such a change only takes place during early life, and few surgeons expect to effect what is called the 'cure' of a hernia in an adult; indeed, it is usual to order that the truss be removed at night when the patient adopts the recumbent posture, and that it be replaced before the erect posture is resumed. This is wrong both in principle and in fact. The great difficulty that the surgeon experiences in the treatment of hernia is in obtaining a properly fitting and efficient truss. The time and skill required for the manufacture and perfect fitting of a truss are considerable, and the latter is particularly difficult to obtain. Again, the circumstances and surroundings of the patient are frequently such that it is impossible to count on the bowel being retained permanently within the abdomen, and in such cases a so-called 'radical cure' should be performed. In the case of umbilical and femoral hernia excision of the sac or more or less complete closure of the aperture of communication is effected. No cure of an inguinal hernia in the male can be properly called 'radical,' since a channel which is occupied by the spermatic cord must of necessity be left. The only real form of radical cure of inguinal hernia is that associated with the removal of the testicle and the complete closure of the external abdominal ring.

Classification.—The most practical classification of hernial protrusions is based upon the usual division of the abdomen into three regions, namely, the epigastrium, mesogastrium, and hypogastrium.

1. Protrusions in the epigastric region are very rare. They are (a) *diaphragmatic* and (b) *epigastric*.

(a) *Diaphragmatic* hernia is due either to relaxation or to laceration of the muscular tissue of the diaphragm. In some cases congenital deficiency of the muscle is the primary cause; in others the natural openings in it become dilated. Signs of this hernia are very obscure, but when the protrusion depends upon the laceration of the muscle, the occurrence of a recent injury may excite suspicion, if associated with abnormal physical signs in the thorax.

(b) *Epigastric* hernia escapes at the region bounded by the cartilages of the false ribs on either side of the linea alba. It is very rare, and, as the abdominal orifice of the sac is usually large, the hernia is easily reduced.

2. The hernie in the mesogastrium are (a) *ventral*, (b) *umbilical*, and (c) *lumbar*.

(a) The term *ventral* is given to any hernial protrusions escaping through abnormal openings in the walls of the abdomen to which no special name is given. They are seen in the region of the linea alba, either above the umbilicus, or, more frequently, below it; in the line of the linea semilunaris; and even over the muscular portions of the walls. Commonly of traumatic origin, their nature is clearly shown by the ready manner in which the protruded viscus can be pressed back into the abdomen. They sometimes ensue upon the weakening of the walls after distension, or upon the loss of tissue following an abscess.

(b) *Umbilical* hernia is met with at all ages and

in both sexes. It forms a tumour at the site of the umbilicus in the first instance, and gradually descends over the linea alba as its bulk increases. Very soon after birth this variety of hernia appears. The protrusion takes place at the umbilical ring, and as it pushes the peritoneum before it, an acquired hernial sac is formed. To prevent, therefore, the development of the sac in infancy, and to assist the closure of the ring in the linea alba, a slightly convex disc of cork, enclosed in washleather, should be strapped over the umbilical aperture. The prognosis of infantile umbilical hernia is favourable, for the aperture closes with age, and the tissues continue firm.

In adult life this kind of hernia is frequent in fat individuals. The tumour often acquires enormous proportions. Its contents consist of small intestine and omentum, with not uncommonly a portion of the transverse colon. Accumulations of fecal matter therein often give rise to obstruction, and the symptoms arising in consequence of this state more or less resemble those of strangulated small intestine. A correct diagnosis of the condition may usually, however, be arrived at from the history of the attack, the comparative mildness of the malady, and the prompt alleviation of the symptoms produced by exciting an action of the bowels. The contents of this form of hernia, when of long standing, often become adherent or bound by bands to the sac, in which state they remain permanently irreducible.

When the protruded viscus can be entirely reduced within the abdominal cavity, a suitable well-fitting truss should be worn constantly; if irreducible, one adapted to the circumstances of the case must be used.

(c) *Lumbar* hernia takes place in the loins. It is a very rare variety, and usually occurs as the result of an injury.

3. The hernie in the hypogastrium are the most numerous and the most common. They include: (a) *inguino-scrotal* or *inguino-labial*, above Poupart's ligament; (b) *femoral*, below Poupart's ligament; (c) protrusions through the apertures of the pelvis in front, beneath the horizontal ramus of the pubes—*obturator*; (d) beneath the arch of the pubes—*perineal*, *puddental*, *vaginal*; and (e) behind, through the ischiatic notch—*ischiatic*.

(a) *Inguinal* hernia is seen at all ages and in both sexes. The following varieties are described: the *oblique* or *external*; and the *direct* or *internal*. In the first, the orifice of the sac is external to the course of the internal epigastric artery; in the second it is internal to this vessel. When the protrusion forming an inguinal hernia does not descend below the inguinal canal it is termed a *bubonocoele*; but when it occupies the scrotum or labium it forms an *inguino-scrotal* or *inguino-labial* hernia. The essential difference between the inguinal hernia of youth and that of middle age lies in the constitution of the sac which encloses the protrusion. From infancy to early adult life, protruding viscera escape from the abdomen into a serous sheath continuous with the parietal peritoneum, namely, the vaginal process of that membrane which extends into the scrotum or labium. In middle life and afterwards, the parietal peritoneum is thrust through apertures or weak points in the abdominal walls by the protruding viscus. In this way two distinct kinds of hernial sac are formed, the first being due to a congenital defect, the second

to a mechanical and acquired cause. In practice, it is very important to bear these distinctions in mind. In the first kind a truss is applied to prevent the passage of the viscus into the sheath, in the hope that by this means its walls may unite and its orifice contract—in fact, to assist nature in accomplishing that condition, the failure of which permits the protrusion to take place. But, in the second, a truss is used to prevent the protruding viscus pushing the peritoneum before it, and so forming for itself a sac; if the development of the sac be thus arrested, there can be no hernial protrusion.

Inguinal herniæ occupy the inguinal canal, and are therefore in relation with the spermatic cord in the male, and the round ligament in the female. They escape from the canal through the external abdominal ring into the scrotum or labium. The neck of the tumour is, therefore, always above Poupart's ligament, and to the inner side of the external pillar of the external abdominal ring. This anatomical fact constitutes the main distinction between inguinal and femoral hernia.

(b) *Femoral* hernia forms a tumour at the inner and upper part of the thigh, immediately below the pubic attachments of Poupart's ligament. These structures are in immediate relation with the neck of the sac. The protrusion escapes at the femoral aperture, the site of the entrance of the lymphatic vessels of the thigh into the abdominal cavity. The neck of the sac is, therefore, to the inner side of the sheath of the femoral vessels; although, in proportion to the bulk of the tumour, its body may overlies it, and even extend upwards above Poupart's ligament, and outwards towards the crest of the ilium. The sac of a femoral hernia is always an acquired formation: hence arises the importance of wearing a truss after observing the slightest indication of a femoral protrusion; for if the yielding, relaxed parietal peritoneum be supported at the crural aperture by a well-adjusted pad, a visceral hernia must be avoided, as there will be no sac into which it can escape. In other words, arrest the development of the sac and there can be no hernia.

To discriminate between a femoral and an inguinal hernia, place the index-finger upon the spinous process of the pubes; if the neck of the tumour is to its outer side, and the whole length of Poupart's ligament can be traced above it, a femoral hernia exists; should precisely the reverse conditions be ascertained, the tumour will be due to an inguinal protrusion. Another method of diagnosis is the direct and careful examination of the site of the femoral aperture: if it is clearly and distinctly tangible and well-defined, it cannot be occupied by a hernial protrusion.

Femoral hernia is most common in the adult female. It has been developed before ten years of age; is rare between that age and twenty; but is very frequent in persons between twenty and forty years old. Prolific women are more frequently the subjects of this hernia than the single and sterile.

(c) *Obturator* hernia escapes from the pelvis through the thyroid foramen, and traverses the canal normally occupied by the obturator nerve and vessels. It is rarely met with. A fulness, rather than a tumour, is produced at the inner or pubic region of the thigh, beneath the pectineus muscle, accompanied by a peculiar numbness and pain, which may be traced to the distribution of the filaments of the obturator nerve. The lives of patients have been lost in consequence of overlooking these

herniæ, the cause of death being only ascertained *post mortem*.

(d) *Perineal*, *puddental*, *vaginal*, and (e) *ischiatric* herniæ are very rarely seen. The name assigned to each indicates the locality in which the tumour is formed; and for a special description of them the reader must be referred to monographs on the subject of this article.

RESULTS AND TREATMENT.—We must next, as briefly as possible, describe generally the various morbid conditions which the hernia itself may undergo, and the means by which serious consequences arising from such conditions may be averted.

All hernial protrusions are either *reducible* or *irreducible*—that is, they can either be restored to their normal situation, or they are permanently confined to the region in which they are protruded. The treatment of all reducible herniæ consists in the employment of means to prevent the escape of the protrusion. To effect this object various kinds of bandages or *trusses* have been devised. Each kind of hernia requires its special form of truss, and every individual should, as far as practicable, obtain a truss well fitted to his or her configuration. The essentials of a good truss consist in the spring having sufficient power to support the hernia and prevent its escape, while it should not be so strong as to injure the structures about the abdominal rings. The pad should be firm, of a shape suitable to the case, and of a size not inconvenient to the wearer. In ordering a truss the practitioner should bear in mind that it is necessary to state the circumference of the body two inches below the iliac crests, with the measuring tape so arranged that it lies over the seat of the hernia. Details as to whether the hernia is right, left, or double, and an accurate description of the size of the tumour should be supplied. Further, it should be stated whether it is of the femoral, scrotal, or inguinal variety, and instructions should be sent specifying whether strong, medium, or weak pressure is required.

The irreducibility of a hernia depends on its bulk, adhesions, and special anatomical conditions. Under such circumstances special bandages must be employed.

But other much more important morbid states of the protruded viscus than the above cause impediments to the reduction of a hernia, namely: (1) *constriction* by the tissues around the orifice of the sac; (2) *accumulation* of fecal matter in the protruded viscus; (3) *inflammation* of the hernia; and (4) *strangulation*, when a part of the alimentary canal forms the hernia.

1. Those herniæ, for example the inguinal, which pass through openings in the muscular walls of the abdomen, are liable to *constriction* from contraction of the muscular tissue. Inguinal herniæ of long standing and more than ordinary bulk are very prone to become irreducible in consequence of muscular contraction. If the patient be placed under the full influence of chloroform, the abdominal muscles become relaxed, and the hernia is quickly reduced.

2. Hernial protrusions formed of large intestine, such as occur at the umbilicus, frequently become irreducible from *fecal accumulations*. In these cases enemata, and even purgative medicines cautiously prescribed, frequently relieve the symptoms.

3. *Inflammation* excited in an omental protrusion may cause temporary and even permanent irreducibility. Local and constitutional symptoms of a

rather severe type sometimes attend such cases. The usual methods adopted to induce resolution must be employed.

4. A hernia is described as *strangulated* when subject to a constriction which at first impedes, and sooner or later arrests, the circulation of the blood in its capillary vessels. The passage of the intestinal contents is necessarily stopped. A patient the subject of this state remains in the greatest danger to life so long as the exciting cause, the constriction of the bowel, exists. Hour by hour that danger increases; and, although rare instances of recovery might be quoted after the continuance of strangulation for many hours, the majority of patients die because the intestine was not liberated early enough. The local and constitutional symptoms are strikingly characteristic. Very frequently the first symptom is vomiting, unaccompanied by any alvine evacuation. The vomiting continues, and is excited by ingesta. This state is probably due to mere obstruction of the alimentary canal, but it ought always to excite the anxious solicitude of the medical attendant to ascertain whether the patient has any outward signs of a hernial tumour. He must examine those regions at which protrusions commonly occur, and never rest content with the statements of the sufferer. At first the pulse is not affected in a very marked way, but as vomiting continues the heart beats more rapidly, while the pulse becomes weaker and contracted. The surface, especially that of the extremities, becomes cold; the countenance aged and anxious; the visage, lips, and hands shrivelled and bluish; the prostration extreme. The tumour is painful when touched, and it may have increased in size and become tense. All these facts indicate progressive morbid changes in the tissues of the strangulated bowel, as well as in that part of the alimentary canal above the hernia. Besides the mere act of vomiting all ingesta, the characters of the fluid vomited must be carefully noted. Usually, at first, it is the food last swallowed, more or less digested and mingled with bile; in the second stage it becomes yellowish or greenish, and at last it is stercoraceous—that is, offensive to smell, of a brownish colour and frothy, and often in great quantity.

To understand the pathological phenomena which may occur in strangulated hernia, it must be remembered that a hernia may be developed on a single occasion, and this is especially the case when bowel is driven into an unobliterated vaginal process. But more commonly the production of a 'rupture' is a gradual one, the bowel entering the canal and proceeding along it, and finally passing out through the external abdominal ring into the scrotum. In either of these circumstances the contents of the hernia, whether bowel or omentum or both, are liable to become strangulated, and the mode in which the bowel reacts to the strangulation varies in a marked manner in the two conditions. The variation depends on the character of the constriction. In a hernia into the unobliterated peritoneal process, the constriction results from the presence of a sharp hard diaphragm, which corresponds to the position of the internal abdominal ring, and represents the mode in which the vaginal process of peritoneum is being normally obliterated at this point. In the slowly acquired variety, the constricting medium usually corresponds to the junction of the sac with the abdominal cavity, called the neck of the sac, and is broad and smooth. In the case of

the sharp constriction, the period of congestion of the strangulated knuckle consequent on the interference with the backflow of blood from it is brief, for the reason that the mucous and muscular coats of the bowel become thinned by the pressure of the sharp peritoneal edge upon them, and in this way a space is provided which allows the passage of blood along the veins. The mucous and muscular coats ulcerate rapidly along the line of compression, and nothing but the peritoneal coat which readily perforates may remain, while the appearance of the loop of bowel enclosed in the sac may present no abnormal degree of congestion. In such cases the operator should be careful to withdraw the bowel from the abdomen for a sufficient distance to enable him to examine the line of constriction, and if he finds the muscular and mucous coats destroyed to a dangerous extent he can invaginate the line of ulceration by a series of Lembert's sutures. On the other hand, when the constriction is broad the obstacle to the backflow of blood not only does not diminish, but is steadily increased, owing to the engorgement and inflammation of the strangulated portion. Such a loop presents quite a different appearance from the former, being swollen, thick, and leathery, and most livid at the portion furthest from the constriction. The bowel at the seat of strangulation presents no such ulcerative changes as those just described, but looks fairly normal, its nutrition being hardly if at all impaired by the pressure it has been subjected to. In this condition, if the engorgement be excessive, the whole loop must be excised, and continuity of the bowel above and below the constriction established by whatever means the operator prefers. The only treatment of these urgent symptoms consists in the liberation of the bowel by surgical means. In the meantime palliatives may be employed, opium administered by the mouth, enemata injected, local applications of ice used, and gentle taxis applied.

The surgical treatment of strangulated hernia resolves itself into two methods—taxis and operation. *Taxis* is the term applied to the judicious application of external pressure to the contents of the hernial sac for the purpose of returning the tumour into the abdomen. Taxis undoubtedly may obviate operation, and therefore should be considered as a first method of treatment in these cases; the practitioner, however, should always remember that an essential condition in the performance of taxis is that it should be done with the utmost gentleness, otherwise there is a very serious danger of the wall of the bowel being injured and bruised with subsequent inflammation. It has been advised that the following positions should be observed in performing taxis. The pelvis should be elevated and the thighs flexed in inguinal hernia; the thighs should be flexed and slightly rotated inward in femoral hernia; and in umbilical hernia the legs should be drawn up for the purpose of relaxing the muscles of the abdominal wall. Some success has been achieved by applying one teaspoonful of ether over the hernial tumour every twenty minutes, covering with compresses during the interval; after about three applications or even less, the tumour loses its tightness, diminishes in size, and may slip spontaneously into the abdominal cavity. It is important that the parts around the tumour should be well protected by copious smearing with olive oil. Taxis should not be undertaken if there are any signs of inflammation of the superficial tissue; where taxis has been

attempted before ; where the condition has lasted for many hours ; or where it is possible that intestinal gangrene has been set up. In those cases in which reduction of the tumour has been successfully accomplished by means of taxis, but no alleviation of the symptoms has been produced, then among other possibilities the question whether what is called 'reduction *en masse*' has occurred must be taken into consideration. Three chief forms of this accident have been described : (1) where rupture of the sac has occurred through which the contents have been forced ; (2) where the strangulated bowel has been reduced into a loculus of a bilocular sac ; (3) where the sac itself with the contained strangulated bowel has been returned into the abdominal cavity. The only safe means of arriving at a definite conclusion under these circumstances is an exploratory operation.

The desirability or otherwise of giving a general anæsthetic in the treatment of strangulated hernia by taxis must be decided after consideration of the facts in each individual case, but it should be clearly arranged before the patient is rendered unconscious that operative interference should be undertaken in case the effort to return the strangulated bowel into the abdominal cavity is ineffectual.

W. ARBUTHNOT LANE.

HERNIA CEREBRI.—See SKULL, Diseases and Deformities of.

HERPES FACIALIS.—SYNON.: Herpesfebrilis ; Herpes labialis.

The term herpes facialis is applied to a small group, or groups, of vesicles on a red base. The eruption is associated with some swelling of the skin or mucous membrane, and with a sensation of very slight pain and stiffness of the part affected. It is most commonly met with on the lips, but occasionally on the alæ of the nose, the ear, or the cheek. It is also not uncommon on the buccal mucous membrane, and on that of the soft palate and uvula. It is generally, but by no means always, confined to one side, and this is more observable on the face or ear than on the lips, where it often extends to both sides of the middle line. The eruption is frequently associated with catarrh of the air-passages, and, though quite unimportant, is apt to recur. No treatment is required, as the eruption disappears in a few days.

ROBERT LIVEING.

HERPES PROGENITALIS.—This disease differs but little from herpes facialis except in the part affected ; it is, however, very apt to be troublesome, especially when irritated by rubbing or injudicious treatment. The eruption is met with in both sexes, but is more common in the male than in the female. In men it appears on the prepuce or glans penis ; in women on the labia or adjacent mucous membrane. When irritated, it is apt to form small ulcers, which are difficult to heal, and which lead to enlargement of the inguinal glands. Under these circumstances herpes has often been mistaken for syphilis.

TREATMENT.—This should be always of the most soothing kind, such as the application of water dressing or a very weak boric acid or lead lotion, with mild aperients.

ROBERT LIVEING.

HERPES ZOSTER (ἑρπας, I creep ; ζωστήρ, a girdle). SYNON.: Zona ; Shingles ; Fr. *Dartre* ; Ger. *Herpes* ; *Bläschenflechte*.

DEFINITION.—An acute vesicular eruption running round one half of the body, following a course determined by the structure of the nervous system and not by that of the skin.

ÆTIOLOGY.—Both sexes are equally liable to the disease. It most commonly occurs between the ages of three and twenty, with a greater proneness to appear between four and thirteen. Children below one year of age are not commonly attacked, but a case has been recorded in an infant four days old. The aged are usually supposed to be more liable to the disease, but this belief probably arises from the extreme severity of the disease in old and debilitated persons, and the intensity of the pain which it frequently leaves behind it.

In the majority of cases zoster arises without known cause. Cold is sometimes invoked to explain its origin. Like other acute specific diseases it begins suddenly with more or less fever, and after a variable period the characteristic lesion appears. It runs a definite course, tends to occur in epidemics, and second attacks are rare. Although persons apparently in perfect health are frequently attacked, those whose resistance is weakened by some intercurrent disease are more prone to suffer from zoster. Thus many of the children attacked have recently suffered from whooping-cough, measles, diarrhoea, or some similar ailment. Pregnant women are especially susceptible, and zoster may follow operations on the pelvic organs. Phthisis is also a well-marked predisposing factor. Patients who are taking arsenic also seem to be more prone to herpes. Attacks of zoster are frequently attributed to mental causes, such as fear or sorrow ; but these influences would appear to act, if they act at all, only by rendering the patient more prone to attack.

Diseases of the nervous system undoubtedly render the patient more liable to herpes zoster. Thus it is extremely common in general paralysis of the insane. But in some nervous diseases zoster is apparently a direct symptom of the pathological changes that underlie the disease. This is the case in myelitis, spinal caries, pachymeningitis, cerebrospinal meningitis, and tabes dorsalis. In all these affections zoster may arise in consequence of implication of the posterior root-ganglion in the morbid process. In such cases the eruption is best spoken of as 'symptomatic,' as opposed to 'spontaneous' zoster.

MORBID ANATOMY.—A vesicle of zoster at its height is a unilocular cavity, the floor of which consists of naked papillæ. Incomplete partitions are formed by altered epithelial cells which retain their attachment to the roof of the vesicle. The cavity is filled with swollen epithelial cells of all ages which have lost their prickles, and with leucocytes ; occasionally certain protozoa-like bodies are seen, which have been shown to be formed from altered epithelial cells. The papillæ beneath the vesicle are filled with small round inflammatory cells, and their vessels are engorged. If death takes place in the acute stage, one of the posterior root-ganglia will be found to be intensely inflamed. The vessels will be engorged, and hæmorrhage will probably have occurred into the substance of the ganglion at one or more spots. Should the lesion be severe and death take place at a later stage, the affected area in the ganglion is found to be converted into dense fibrous tissue, in which neither nerve-cells nor nerve-fibres can be seen.

SYMPTOMS.—The onset of the rash is frequently

preceded by slight malaise, especially in children. The child lies about by day and tosses restlessly at night. It may vomit or refuse food. Adults may suffer from shivering similar to that which accompanies the onset of acute lobar pneumonia, but of less intensity. During this period the temperature is generally raised somewhat; in adults as a rule it does not exceed 100°F. , but in children it may occasionally reach 102°F. Pain is usually present from the first: it is shooting in character, and worse at night. It occupies the area subsequently covered by the eruption. With the appearance of the rash the temperature usually falls, while the pain remains; but this is by no means always the case. Thus, the rise of temperature lasts from three to five days, and this period may be considered the duration of the acute disease. The rash, on the other hand, may either begin to make its appearance a few hours after the onset of the attack or may be delayed until the acute general symptoms are past. Most commonly it begins to appear about the third or fourth day of the illness. If it comes out early it does not usually reach its full extent for two or three days, whereas if it is delayed it may attain its full development in twelve hours.

It occupies areas on the trunk and limbs which represent the distribution of nerve-fibres that enter a single posterior root-ganglion. These areas are approximately shown in the figures illustrating the article on PAIN IN VISCERAL DISEASE.

From the time when the rash first makes its appearance it spreads with varying rapidity. In each affected area three spots usually exist at which the branches of the nerve come to the surface (the posterior primary, and the lateral and anterior branches of the anterior primary division). It is with these points as centres that the eruption first makes its appearance: from these it spreads along the distribution of the main branches, and may invade the territory supplied by even the finest nerve-twigs, until the whole area is occupied by vesicles or raised erythema. The most severe incidence of the eruption always falls on the skin in the neighbourhood of the central points, and it is over these that, in the severest cases, huge bullæ or small gangrenous patches may appear. But the large majority of cases do not run so severe a course, and in some the whole eruption consists only of a few vesicles scattered around one or more of the maximum spots. On the palm and on the sole of the foot vesicles are rare, and, when present, tend to be of small size, very little raised and not surrounded by erythema. Vesicles on the fingers, the back of the hand, the toes, and the dorsum of the foot are not usually severe.

The rash may consist, from the first, of vesicles, containing clear fluid, surrounded by more or less erythema; but more commonly it first shows itself as an erythema upon which vesicles rapidly arise. In very severe cases the vesicles may contain blood-stained fluid. Sometimes these rapidly break down, giving place to shallow ulcers. In all such cases subsequent scarring is profound and much after-pain is likely to result. In most cases the contents of the clear vesicles become turbid, and, possibly in connection with this pus-formation, the lymphatic glands that drain the affected area of skin are usually enlarged and frequently somewhat tender. About the fifth to the tenth day after their first appearance the vesicles begin to dry up and form scabs. Under these scabs the eruption

heals, and at the end of six weeks nothing is to be seen beyond some freshly healed scars. If the eruption has been severe the scars may be large, irregular, and white, closely resembling those of a superficial burn.

During the period when the rash is fully out, the pain becomes more local. It may be little more than a painful itching or it may become of an agonising burning character. Usually the pain dies away when the rash begins to heal, but in elderly patients it may last for weeks, months, or even years after the scars are formed. This severe after-pain is relatively more common where zoster has affected the ophthalmic division of the trigeminal nerve, and is always associated with considerable scarring.

Second attacks are extremely rare, and in no recorded case has a second attack coincided in distribution with the first. Two simultaneous eruptions at different levels have been known to occur.

Zoster of the Head and Neck.—Within the territory of the first or ophthalmic division of the trigeminal nerve, zoster, exactly resembling in course that seen on the body, is a fairly common occurrence. The area occupied by the eruption may extend over the whole or part of the ophthalmic division of the nerve, and may involve the side of the nose as far as the upper border of the ala nasi. This implication of the side of the nose is of great importance, for it is in these cases that the eye is affected. The conjunctiva becomes red and injected, and ulcers may appear on the cornea. Iritis may also occur, and it is therefore always wise in these cases to keep the pupil well under the influence of atropine. Panophthalmitis resulting in loss of the eye has also been described.

Herpetic eruptions about the lips and nose or on the cheeks are common in febrile states, but such eruptions bear no relation to herpes zoster. They are usually bilateral, notoriously prone to recur, are not preceded or accompanied by neuralgic pain, and do not follow any definite nerve-course. But true zoster of the face occurs, and may be distributed over either the second or third division of the trigeminal nerve.

The tongue may also be the site of an herpetic eruption, but the vesicles rapidly break and give rise to shallow ulcers. These ulcers are usually found on the upper surface and tip of the tongue. This form of eruption is probably more frequently a form of herpes febrilis than of true zoster.

COMPLICATIONS.—*Cutaneous.*—The rash usually heals and leaves scars within a few weeks; but occasionally gangrenous sores may appear and last for many weeks or even months. The eruption seems to be quite uninfluenced by the simultaneous existence of other cutaneous lesions. Thus it is not uncommon in patients suffering from psoriasis, owing probably to the almost universal exhibition of arsenic in this disease, and in such cases it shows no deviation from its usual course and distribution.

Motor.—Occasionally, though rarely, zoster is accompanied by motor paralysis. Thus zoster of the areas of the arm may be associated with temporary weakness of the whole arm or of the hand, and that of the cervical areas may be accompanied by facial paralysis of the usual peripheral type. Ptosis and ophthalmoplegia externa and interna may be associated symptoms of zoster appearing in the distribution of the ophthalmic division of the trigeminal nerve.

TREATMENT.—No treatment is effectual in aborting the eruption, but when the rash has appeared it may be dusted with a powder consisting of starch (ζij), oxide of zinc (ζj), and camphor-powder (gr. 15 to 45); to these may be added, if there is much pain, gr. 15 of powdered opium. If preferred, an ointment may be applied consisting of boric-acid ointment (ζj) and cocaine (gr. 5).

For the after-pain no treatment but morphine is really effectual. It is, however, always well to try phenazone in 10-grain doses three times a day. General tonics, such as arsenic and quinine, are indicated; for patients who suffer severely from this after-pain are always debilitated.

HENRY HEAD.

HETEROLOGOUS (ἕτερος, other; and λόγος, nature).—A word used to characterise any morbid product, whether fluid or solid, which is different in composition or structure from the normal fluids or solids of the body.

HETEROMORPHOUS (ἕτερος, other; and μορφή, form).—Applied to new-formations which are different in form and structure from the normal tissues.

HETEROTOPOUS (ἕτερος, other; and τόπος, a place). Misplaced. A term applied to the appearance either of a normal tissue in an unnatural situation—for example, of hairs on mucous surfaces; or of morbid growths in unusual places—for instance, of epithelioma in nervous tissue.

HICCUP or HICCOUGH.—SYNON.: *Singultus*; Fr. *Hoquet*; Ger. *der Schlucken*.

DESCRIPTION.—Hiccup, according to physiologists, is a sudden spasmodic descent of the diaphragm accompanied by a spasmodic closure of the glottis, the characteristic noise being caused by the incoming column of air striking against the partially closed glottis. The assumption of a spasmodic closure of the glottis in hiccup seems scarcely warrantable. Normally the descent of the diaphragm in each respiratory act is accompanied by a contraction of the posterior crico-arytenoid muscles, which causes an outward rotation of the arytenoid cartilages, and a dilatation of the glottic aperture. The diaphragmatic and the laryngeal acts keep time together, and in health the rhythm of sixteen or eighteen to the minute is maintained. If, however, the diaphragm give a sudden descending jerk irrespective of any respiratory need, as is the case in hiccup, and this jerk occur at a time when the dilators of the glottis are not acting, a noise will be produced by the rush of air through the insufficiently widened glottic aperture. It seems certainly possible to account for the noise of hiccup by the mere fact of the descent of the diaphragm occurring when the glottis is not properly open. The noise is not a constant phenomenon, and during an attack of hiccup it never occurs during ordinary inspiration, or without the spasmodic action of the diaphragm, although the latter phenomenon may occur without the former.

ÆTIOLOGY AND DIAGNOSIS.—Hiccup may be produced by any irritation of the phrenic nerve—its origin, its course, or the ultimate twigs which are distributed to the under-surface of the diaphragm. Undue distension of the stomach, by being overfilled with food or drink, or by an accumulation of wind due to faulty digestion, is the most common cause of hiccup. Its occurrence from

this cause is far more common in children than in adults. Convulsions and muscular spasms generally are more easily caused in the young, and hiccup in this respect follows the ordinary rule. Hiccup is produced by direct or by reflex irritation. With many persons the introduction of hot spiced or peppery foods into the stomach immediately produces hiccup, and the writer knows one or two persons in whom hiccup is produced by the passage of hot fluids through the pharynx. It is a frequent symptom in peritonitis when the peritoneal covering of the diaphragm becomes affected. It sometimes occurs in cases of cancer of the stomach; occasionally, perhaps, from over-distension of the organ, but more often from an extension of the cancerous disease to the peritoneal surface of the stomach. It is occasionally a troublesome symptom during convalescence in cholera, and is often accompanied by eructations of wind, and sometimes by vomiting. If hiccup occur with any persistency in the course of typhoid fever, it is often an indication of perforation and the onset of general peritonitis. Although most frequently a symptom of gastric or abdominal disturbance, hiccup occasionally occurs as a true neurosis. It may accompany hydrocephalus or meningitis, and is then due probably to an implication of the cerebral origin of the phrenic nerve. It also occurs in uræmia and acute gout. Cases of obstinate hiccuping in hysterical subjects have been recorded, and certain cases of paroxysmal hiccup have been regarded as instances of modified epilepsy.

TREATMENT.—The treatment of hiccup will depend upon the cause. An emetic to empty the stomach, or a stimulant to increase its natural peristaltic action, will often give relief. If we can manage to produce a forcible action of the diaphragm, we may often succeed in curing it, as it were, of the trick of spasmodic action. Attempts to count a hundred without drawing breath, or to hold the breath for a minute, are familiar remedies for hiccup; and, by producing a feeling of suffocation, and necessitating a violent descent of the diaphragm, they are often successful. Warm applications or counter-irritation applied to the diaphragmatic region or over the cervical spine, may occasionally give relief. Pressure upon the trunk of the phrenic nerve, by means of the finger applied over the scalenus anticus muscle, is said also to have given relief occasionally in obstinate cases. Among the drugs which have been recommended for the relief of hiccup are chloroform (administered internally), either alone or combined with opium; camphor in the form of a spirit solution, in doses of twenty drops and upwards; valerianate of zinc, belladonna, bromide of potassium, trinitrin, musk, or antacids; and, in very severe cases, morphine administered hypodermically.

G. V. POORE.

HIDRADENITIS. — See SUDORIPAROUS GLANDS.

HIDROCYSTOMA. — See SUDORIPAROUS GLANDS.

HIPPURIA (ἵππος, a horse; and οὐρον, urine).—The condition of the urine in which it contains hippuric acid in excess. See URINE, Morbid Conditions of.

HIPPUS (ἵππος, a horse).—Rapid spasmodic variation in the size of the pupils apart from the action of light or of accommodation. See p. 630.

HISTRIONIC SPASM (*histrion*, an actor).—A synonym for facial spasm, so called on account of the contortions of the face to which this affection gives rise. *See* FACIAL SPASM.

HOARSENESS (Sax. *has*, having a rough voice).—Roughness of the voice, due to disease or disorder connected with the larynx. *See* VOICE, Disorders of.

HOBNAIL LIVER.—A name given to a cirrhotic liver, when it presents small prominences on its surface, somewhat resembling hobnails. *See* LIVER, Cirrhosis of.

HODGKIN'S DISEASE.—A synonym for lymphadenoma. *See* LYMPHADENOMA.

HOMBURG, in Germany.—Common salt waters. *See* MINERAL WATERS.

HOMICIDAL INSANITY.—*See* CRIME, Irresponsibility for; and INSANITY, Impulsive.

HOMOLOGOUS (*ὁμός*, like; and *λόγος*, relation).—In pathology this term is applied to new-growths presenting the same structure as normal tissues, such as fatty or fibrous tumours.

HOOPING-COUGH.—*See* WHOOPING-COUGH.

HORDEOLUM (*hordeum*, a barleycorn).—A synonym for sty. *See* STYE.

HORNS.—SYNON.: Cornu Cutaneum; Fr. *Corne de la peau*; Ger. *Hauthorn*.

DEFINITION.—Horns are circumscribed painless epidermic formations of irregular masses of keratin.

DESCRIPTION.—Horns are usually single, and rarely grow before the age of forty, though they have been met with in infancy. They vary in shape and size, being elongated, conical, curved, or twisted. They may grow on any part of the body, but are most commonly met with on the scalp, forehead, temples, and nose. Their surface is rugose, and they vary in colour, being either grey, brown, yellow, or black. In many instances horns arise from sebaceous cysts; they sometimes arise in warts (a horn may be regarded as an overgrown wart) and they may be the starting-point of a malignant growth. Unna regards them as papillary and medullated keratomata.

TREATMENT.—Radical treatment consists of excision of the horn including its base, so as to guard against its reproduction. If thought desirable, the latter may be dabbed with a solution of chloride of zinc.

JOHN HAROLD.

HOT AIR, Treatment by.—*See* BATHS; GOUT; RHEUMATISM, Chronic; and RHEUMATOID ARTHRITIS.

HOT SPRINGS, in Bath County, Virginia, U.S.A.—Thermal waters. *See* MINERAL WATERS.

HOT SPRINGS, in Garland County, Arkansas, U.S.A.—Thermal waters. *See* MINERAL WATERS.

HUM, VENOUS.—A continuous murmur heard in the veins, generally observed in cases of anæmia. *See* ANÆMIA; and PHYSICAL EXAMINATION.

HUNTINGDON'S CHOREA.—*See* CHOREA.

HUNYADI JANOS, in Hungary.—Sulphated waters. *See* MINERAL WATERS.

HUTCHINSON'S TEETH.—*See* TEETH, Diseases of.

HYALINE DEGENERATION.—*See* DEGENERATIONS.

HYDATIDS.—*See* ENTOZOA.

HYDATIDS, UTERINE.—*See* MOLE; MOLAR PREGNANCY.

HYDRÆMIA (*ὑδρᾱ*, water; and *αἷμα*, the blood). A watery condition of the blood. *See* ANÆMIA; and BLOOD, Morbid Conditions of.

HYDRAGOGUES (*ὑδρᾱ*, water; and *ἄγω*, I drive).—SYNON.: Fr. *Hydragogues*; Ger. *Wasser-treibende Mittel*.

DEFINITION.—Purgative medicines which cause a copious watery discharge.

ENUMERATION.—Hydragogue purgatives include:—Bitartrate of Potassium, Buckthorn, Colchicum, Colocynth, Croton Oil, Elaterin, Gamboge, Hellebore, Jalap, Podophyllum Resin, and Scammony. The two most important are Compound Jalap Powder and Elaterin.

ACTION.—It has been supposed by some writers that the action of the drugs included in the present class is due only to the increased peristaltic action which they produce. This, however, is not the case, as certainly some, if not all, of those just enumerated induce a free secretion of fluid by the intestinal mucous membrane, while at the same time they stimulate the peristaltic action of the bowel, and cause the evacuation of this watery fluid.

USES.—Hydragogues are chiefly employed for the removal of fluid from the body, in cases either of general anasarca or of dropsical effusion in serous cavities. They may be employed to assist the action of the kidneys when this is insufficient; and it has been observed not infrequently, when the secretion of urine has previously been deficient, that it becomes greatly increased after a free discharge of fluid has taken place from the bowels, in consequence of the administration of a hydragogue cathartic. *See* PURGATIVES.

T. LAUDER BRUNTON.

HYDRARGYRIASIS (*hydrargyrum*, mercury). The state produced by the introduction of mercury into the system. *See* MERCURY, Poisoning by.

HYDRARTHROSIS (*ὑδρᾱ*, water; and *ἄρθρον*, a joint).—Effusion of serous fluid into a joint. *See* JOINTS, Diseases of.

HYDROA.—SYNON.: *Dermatitis Herpetiformis*; *Pemphigus pruriginosus*; *Herpes gestationis*; *Herpes circinatus bullosus*.—This disease is characterised by a peculiarly polymorphic eruption. The type is erythematobullous, the vesicles, bullæ, or more rarely pustules tending to group, or be marginal, on the reddened area. Successive crops appear at irregular intervals for an indefinite period, the appearance of each being ushered in by an aggravation of the itching, a symptom always present to a certain degree, but diminishing on their full evolu-

tion. In distribution, partiality is evinced for the neighbourhood of the articulations, for the sacral region, and for covered parts generally, though the disease is not restricted to such. The herpetiform arrangement does not always prevail in the case of children, in whom the lesions may assume an isolated disposition.

COURSE.—Though occasionally acute in its inception, it runs an essentially chronic course as a rule, continual relapses taking place, or recurrences after a period of complete immunity. It is remarkable how well the health is maintained, when the extent of blistered and inflamed surface, and the restlessness due to the pruritus, are considered. The lesions are succeeded by crusts, which are hard and angular, and by stains and pigmentation; but permanent scars are rare. It may occur at all ages, may last many years, and while it usually terminates spontaneously, it may end fatally. The term *Herpes gestationis* has been given to it when, as sometimes happens, it appears during pregnancy or after delivery. Once having appeared, it is apt to reassert itself in subsequent pregnancies, though the sequence may not always be continuous. The vesicles are loculated and form in the substance of the rete mucosum, which is oedematous. They are firm and tense, arising between the cells, not within them, by ballooning, and the clear fluid with which they are filled contains a large percentage of eosinophile globules. No well-defined pathogenic microbe has been isolated by any method of culture hitherto tried. Peripharyngeal parenchymatous neuritis has been observed.

PATHOLOGY.—As to its pathology, the view is pretty widely held that the phenomena are due to toxins derived from absorption into the blood of excrementitious matters, or bodies arising from imperfect metabolism. These determine the cutaneous manifestations either directly or through the medium of the nervous system. There is often diminished excretion of urea preceding an outburst of the eruption, and it is believed that some toxic substance, normally eliminated in the urine, is retained in the blood. Though the toxin has not yet been identified, there are circumstances which go to support this view. Thus the administration of iodide of potassium at once intensifies the eruption, or induces an attack if latent. It is accepted that a bullous form of iodine-eruption, which much resembles it, is specially apt to appear when that drug is given in nephritis, or whenever the kidneys act inefficiently. A highly nitrogenous diet has seemed to accentuate the disease in some instances.

DIAGNOSIS.—To a superficial observer it most resembles *scabies*, but it exhibits no contagious properties; there are no burrows; the localisation is different; and the itching is not markedly worse at night. From *pemphigus vulgaris*, however, the distinction is not in all cases easy. Still in *pemphigus* there is hardly polymorphism, the bullæ are distributed irregularly, itchiness is only occasionally aggressive, while the constitutional symptoms are much more grave. Most instances of so-called urticaria bullosa, if not a mere accidental occurrence on some wheals, ought rightly to be regarded as dermatitis herpetiformis. *Erythema multiforme* is limited in duration, seldom itches, and bullæ are casual features. *Impetigo herpetiformis*, if not of pyæmic origin, may be a severe pustular eruption.

TREATMENT.—Arsenic exerts in most cases, though not invariably, a distinct controlling effect,

and during its administration the attacks are often abortive or less annoying. It should be pushed to the limits of tolerance, and persevered in for prolonged periods. Phenacetin has been suggested should arsenic fail. Pricking the blebs relieves the pruritus probably by diminishing tension. Painting the affected areas with a solution of ichthyol in water or calamine lotion in the proportion of one-sixth to one-fourth is most generally useful. Inunction with sulphur-ointment has also proved beneficial. Baths of permanganate of potassium are soothing, if not directly curative. The diet ought to include vegetables, farinaceous matter, and fish, but only a small quantity of meat. The action of the kidneys should be promoted by diluents or mild diuretics.

W. ALLAN JAMIESON.

HYDROCELE (ὕδωρ, water; and κήλη, a tumour).—**SYNON.**: Fr. *Hydrocele*; Ger. *Wasserbruch*.

DEFINITION.—A swelling produced by a collection of fluid in connection with (A) the testicle, or (B) the spermatic cord.

A. HYDROCELE OF THE TESTICLE.—The principal forms of hydrocele of the testicle are (1) *vaginal*, (2) *congenital*, and (3) the *encysted*; (4) *chylous*, (5) *bilobular*, and (6) *diverticular* hydrocele will also be noticed.

1. Vaginal Hydrocele.—**DESCRIPTION.**—This is a chronic dropsical effusion into the sac of the tunica vaginalis. The fluid is a pale-yellowish serum, which in old cases is often loaded with cholesterolin. The quantity varies, but seldom exceeds twenty ounces. As many as six quarts have been evacuated. The testicle is usually situated at the back part and rather below the centre of the sac; but its position may be altered by adhesions; and, in cases of congenital inversion, the testicle is in front of the sac. In old hydroceles the sac is often greatly thickened by deposition of lymph on the tunica vaginalis, and its conversion into fibrous tissue, which is sometimes the seat of calcareous deposits. Vaginal hydrocele is generally single, but often occurs on both sides. It forms an oval or pyriform swelling, which fluctuates; has a smooth, even surface; and commences at the lower part of the scrotum, very gradually and without pain. When examined by transmitted light it is found to be translucent, except at the back part, where the testicle is situated. Owing to the tunica vaginalis remaining unobliterated for some distance along the cord, the swelling occasionally assumes an elongated form, and extends up towards the inguinal canal. It is then known as *infantile hydrocele*. A hydrocele sometimes varies in size, becoming larger and more tense during the day than when the patient first rises in the morning.

ETIOLOGY.—Hydrocele is a common disease, especially in warm climates; and occurs at all periods of life, but is most common in middle age.

DIAGNOSIS.—The circumstances—that the swelling commences below; that the spermatic cord can be detected above the tumour; that the testicle cannot be felt; and that the tumour receives no impulse on coughing, and does not vary in size on pressure—are signs distinguishing a hydrocele from an inguinal rupture. When the sac is much thickened, so as to obscure fluctuation, and prevent the passage of rays of light, the tumour may be mistaken for a hæmatocele, or disease of the testicle, and the diagnosis is difficult.

TREATMENT.—In *Infants*, vaginal hydrocele generally disappears under the application of weak tincture of iodine or, as this may irritate the skin, of some evaporating lotion. Acupuncture, causing the fluid to escape into the areolar tissue of the scrotum, is the only operation that is required.

In the *adult* external remedies are of no use. It is usual to resort at once to operative treatment, *palliative* or *radical*. The *palliative* operation consists in puncturing the tumour with a trocar and cannula, and evacuating the fluid accumulated in the tunica vaginalis. The hydrocele usually returns in the course of two or three months, and then the operation must be repeated, or the patient must undergo *radical* treatment. This may be effected by incision or excision of the sac; or by injection of the sac with a stimulating fluid. The last is the plan most commonly resorted to, though an increasing number of surgeons now advocate primary excision on account of the liability to recurrence after iodine-injections.

The fluids which have been found most useful are: (1) the Edinburgh tincture of iodine; (2) a solution of perchloride of mercury—I grain to an ounce of water; and (3) carbolic acid and glycerin in varying proportions.

When iodine is to be used the patient should be placed in the recumbent posture, as faintness is not infrequent, and a sickening pain running up into the groin and lumbar regions is usually complained of. This may be avoided by injecting 10 m of a four-per-cent. solution of cocaine as soon as the hydrocele-fluid has been evacuated. A few minutes after the injection of the cocaine about a drachm of the solution of iodine is injected into the sac and there retained.

The injection of carbolic acid was introduced by Levis of Philadelphia, and is preferred by many surgeons, as being less painful and more efficient than iodine. Levis injects about a drachm of a solution composed of ʒj of carbolic-acid crystals, with the addition of 5 per cent. of glycerine or water. A sense of warmth is produced by the injection, which is quickly followed by a decided numbness, and the patient is at once able to walk about and to attend to his ordinary duties. No rest is necessary until inflammatory action sets in, which is usually after the lapse of twenty-four hours. It is said to be more certain in producing the necessary amount of inflammation for effecting a cure than iodine, and it appears to be less painful. One of the writers prefers the injection of a corrosive-sublimate solution, 1 in 500, as being more certain and less painful even than the carbolic acid. After the fluid in the sac has been withdrawn, an ounce or two of the solution is injected, and care is taken to ensure that it is brought into contact with every part of the serous sac before it is allowed to escape. To make quite sure that this is the case, the injection may be repeated. It will be noticed that when mercury is used it is simply as a wash, and is not left in, as is the case with both iodine and carbolic acid.

When employing the injection-treatment one precaution is especially necessary—the surgeon should see that the cannula has been passed well into the sac, or some of the fluid used as an injection may find its way into the areolar tissue through the slit (about half an inch long) in the side of the cannula. This accident was followed, in one instance of corrosive-sublimate injection, by salivation.

As a rule, after this method of treatment, no con-

finement to bed is necessary; but a suspensory bandage is advisable. A slight inflammation is set up in the sac, causing a rapid return of the swelling, which, however, gradually subsides until the patient is cured.

Sacs with thick walls are not suitable cases for the injection-treatment. Here excision is the proper treatment, though it must not be forgotten that even after the removal of the whole of the parietal layer of the serous membrane recurrence of the hydrocele has been recorded.

2. Congenital Hydrocele.—In children, the original communication between the cavities of the peritoneum and of the tunica vaginalis sometimes fails to be obliterated; and fluid accumulated in the sac constitutes the variety termed congenital hydrocele. The communication is usually small in size.

DIAGNOSIS.—Congenital hydrocele is easily distinguished from ordinary hydrocele by the absence of a defined boundary on the upper part of the tumour; by the impulse received on coughing; and by pressure causing the disappearance of the swelling, and rendering the testicle perceptible. It is distinguished from a reducible hernia by the fluctuation and transparency of the swelling; by the absence of gurgling accompanying its disappearance on pressure; and by the slow return of the swelling on the patient assuming the erect posture.

TREATMENT.—Congenital hydrocele is usually cured by the gentle pressure of a truss on the inguinal canal, so as to occasion obliteration of the neck of the sac, after which the fluid usually becomes absorbed. Should this fail, excision with ligature of the neck of the sac must be carried out.

3. Encysted Hydrocele.—In this form of hydrocele fluid is effused into an adventitious cyst distinct from the vaginal sac, developed in the areolar tissue beneath the visceral portion of the tunica vaginalis investing the head of the epididymis. There may be two or even more such cysts. As the cyst enlarges, the epididymis becomes flattened and displaced to one side, while the testicle is found either in front or at the bottom of the cyst. The fluid contained in the sac differs from that of vaginal hydrocele in being less in quantity, perfectly limpid and colourless, and nearly free from albumen. The fluid sometimes contains spermatozoa in great abundance, rendering it opaque and milky-looking. Their presence may be owing to the rupture of one of the tubes of the epididymis, and the escape of semen into the sac of the hydrocele. More probably, the spermatozoa-containing cysts are due to dilatation of one or more of the vasa efferentia in consequence of stricture; and those that contain limpid fluid may be developed from the organ of Giraldès.

DIAGNOSIS.—An encysted hydrocele is distinguished from vaginal hydrocele by the position of the testicle at the bottom of the tumour; and generally by the colourless character of the fluid evacuated.

TREATMENT.—When large in size, so as to be inconvenient, encysted hydrocele may be treated in the same way as vaginal hydrocele, and injection is attended with the same success.

4. Chylous Hydrocele.—**SYNON.** : Chylocele; Galactocoele; Liparocoele; *Fr. Hydrocèle laiteuse ou graisseuse.*

DESCRIPTION.—Chylous hydrocele may be bilateral or unilateral. It is not translucent, but it otherwise resembles an ordinary hydrocele. The

sease is one of the manifestations of the presence of the *Filaria sanguinis hominis*. See FILARIASIS.

TREATMENT.—Tapping is merely palliative, as the fluid soon re-accumulates. In one case, at the first tapping the fluid was of the ordinary serous nature, but this result appears to be unique. Injection with solution of iodine is usually successful. If it fail, incision, with drainage, or excision of the sac may be performed.

5. Bilobular Hydrocele.—SYNON.: *Hydrocele en bissac* (French surgeons).

DESCRIPTION.—This disease may be altogether outside, or partly inside and partly outside, the abdomen. The formation of the former variety is easily explicable. The *processus vaginalis* may be closed completely at the internal abdominal ring, and partially at its connection with the *tunica vaginalis propria*, the intervening part remaining open. In such a condition a hydrocele develop, it will form two sacs, continuous internally, and marked at their junction externally by a constriction, the depth of which will vary inversely with the size of the opening between the sacs. It will be a combination of a *funicular* and a *vaginal* hydrocele, with a communication between the sacs. The diagnosis cannot present any difficulty, and the symptoms, possible results, and treatment will not differ from those of an ordinary hydrocele of the *tunica vaginalis propria*.

For the formation of the other variety, where one sac is inside and the other outside the abdomen, two hypotheses are possible. It may result from some congenital malformation of the *processus vaginalis*, or this process may be closed at the internal abdominal ring, and, if a hydrocele subsequently develop, a diverticulum may be formed at the upper part of the process, and experience less resistance to its extension in the subperitoneal tissue than in the inguinal canal or the *tunica vaginalis communis*.

This form of bilobular hydrocele has been observed at birth, and the great majority of recorded cases have been in patients under forty years of age; but it may develop or become manifest at a later period. Some cases have been complicated with retention of the testicle within the abdomen or in the inguinal canal, but in others the gland has been in its normal position. The intra-abdominal sac may vary greatly in size. In one case it was not larger than a hazelnut; in another it extended above the level of the umbilicus and beyond the median line of the abdomen. It is usually situated between the peritoneum and the iliac fascia, but in one case it was between the peritoneum and the *fascia transversalis*. The external sac may also vary in size. Sometimes it is small, and limited to the *processus vaginalis*; but when the *tunica vaginalis propria* is also involved, the sac may be very large. When the contents of the sac are serous, the patient does not experience any greater inconvenience than with an ordinary hydrocele. But there appears to be in these cases a great tendency to the formation of a hæmatocele, and the intimate relation of the internal sac with the peritoneum may then become very important.

DIAGNOSIS.—When the intra-abdominal sac is large and causes a projection of the abdominal wall, attention cannot fail to be directed to it, and pressure over the abdominal swelling will produce increased tension of the external sac, and *vice versa*. But if the intra-abdominal sac be small, or lodged deeply in the iliac fossa, or extend into the pelvis,

it may escape notice; but the reducibility, partial or complete, of the contents of the external sac and the *immediate* re-appearance of the swelling when the pressure has been removed, ought to suggest the possibility of an intra-abdominal sac. Careful palpation, when the patient has been anaesthetised, will detect some fulness in the iliac fossa; and if the sac has extended into the pelvis, digital exploration through the rectum will aid the diagnosis.

TREATMENT.—The treatment will depend upon the size of the communication between the sacs, the nature of their contents, and their walls. If the communication be free, the contents serous, and the walls thin, tapping the external sac will empty the internal sac also, and the injection-treatment will probably effect a cure. But if the communication be very narrow, tortuous, or valvular, if extravasation of blood has occurred, or if the walls of the sacs be thick, operative treatment varying with the nature of the case will be requisite.

6. Diverticular Hydrocele.—This variety of hydrocele is also bilobular, but is altogether scrotal. It was first described by Béraud in 1856, who showed by dissection that the *tunica vaginalis propria* sometimes has very small diverticula. If a hydrocele form, one of these diverticula may become larger and protrude through some accidental rupture or inherently weak part of the *tunica vaginalis communis*. This protrusion might subsequently become so distended as to form the chief part of the swelling.

The *diagnosis* of this condition during life must be very uncertain. The form might be unusual, and the coverings would be very thin, so that translucency would be very marked. If the hydrocele be not tense, it is said that pressure will convey to the hand of the observer a sensation of fluid passing from the diverticulum into the parent sac. It is practically unimportant, for the *treatment* is similar to that of ordinary hydrocele.

B. HYDROCELE OF THE CORD.—Hydrocele occurs in the spermatic cord in two forms—*diffused* and *encysted*.

The *diffused*, which is very rare, is simply an oedema of the areolar tissue of the cord. When inconvenient, owing to its size, antiseptic incision and drainage should be undertaken.

Encysted hydrocele of the cord arises from a collection of fluid in the unobliterated funicular process of peritoneum, which is carried down in the natural transition of the testicle. It produces a small swelling in the cord, of an oval form, above and distinct from the testicle, more or less transparent, and quite movable. The swelling, when small, is of no importance; and it seldom requires treatment. When large it may be treated as though it were an ordinary hydrocele. See TESTES, Diseases of.

C. Hydrocele of a Hernial Sac is sometimes classed with hydrocele of the cord.

Where no definite history of a rupture is to be obtained, it may be difficult to distinguish this form from an encysted hydrocele of the cord, especially if the hernial sac is closed at its neck by adhesions or by a piece of omentum. An exploratory incision will probably clear up the diagnosis, when should it prove to be a hernial sac it should be ligatured at its neck and excised as in a radical operation for hernia.

JEREMIAH MCCARTHY.
F. SWINFORD EDWARDS.

HYDROCEPHALUS, Acute (ὕδωρ, water; and κεφαλή, the head).—A synonym for tubercular meningitis. See MENINGES, CEREBRAL, Diseases of.

HYDROCEPHALUS, Chronic. — SYNON. : Water on the Brain; *Hydrops Capitis*; Fr. *Hydrocéphale*; *Hydropisie du Cerveau*; Ger. *Der Wasserskopf*; *Hirnwassersucht*.

DEFINITION.—A gradual accumulation of serous fluid within the lateral and third ventricles of the brain, causing them to become more or less distended, and the head enlarged; occurring principally in infants or very young children; and leading to restlessness, irritability, or convulsions, followed by dulness, drowsiness, motor weakness, or actual paralysis, together with failure of mind and of the special senses.

The essential condition in this malady is the intra-ventricular effusion. The cases in which the fluid has been found outside the brain and within the arachnoid sac are, in all probability, merely examples of the disease in which, intra-ventricular effusion having previously been well marked, the distended corpus callosum, or, it may be, the floor of the third ventricle, has given way, and allowed the fluid to pass beneath the arachnoid. The so-called extra-ventricular form of the disease is therefore, in the majority of cases, merely a secondary and altogether accidental condition.

As a sequel of a large arachnoid hæmorrhage, serous fluid may also be found within the arachnoid cavity; this, however, is a condition which has no real title to be mentioned under the head of chronic hydrocephalus, as some writers have done. And the same remark applies to those accumulations of serous fluid which take place beneath the arachnoid, as a sequel of wasting or atrophy of the cerebral hemispheres, one or both. The collection of fluid in such cases is to be regarded as a simple sequel of the atrophy, and is of itself unproductive of morbid symptoms.

ÆTIOLOGY.—Two principal groups of causes are described. In certain cases, formerly known as 'essential,' the affection is believed to be due to inflammation of the lining membrane of the ventricles. In other patients, however, this affection is distinctly secondary or *symptomatic*, and then may be caused by one or other of two principal sets of conditions. Thus (1) it is often occasioned, as writers of the last century pointed out, by the pressure of scrofulous or other tumours upon the 'straight sinus,' producing mechanical congestion of the great veins of Galen as well as of their radicles on the walls of the lateral ventricles, and, as a consequence, an increasing dropsical condition of the ventricles themselves. Or else it may result from another mechanical cause, namely, closure of the foramen of Majendie and other minute apertures in the membranes closing in the fourth ventricle as a sequel of some form of meningitis. (2) Much more rarely it is said to be produced as a sequel of an attack of acute hydrocephalus. This latter mode of origin is admitted by some authors, and denied by others. It is at least a possible mode of origin, although one which it is difficult to establish with certainty. By far the largest percentage of cases probably belong to the first set of the 'symptomatic' category.

The disease is sometimes congenital, and may be so far developed during uterine life as to cause

great difficulties in parturition—frequently necessitating the sacrifice of the life of the child. At other times the enlargement of the head begins to show itself soon after birth, or at some period before the end of the first or second year. Or it may reveal itself later still in childhood; much more rarely during adolescence; and more rarely still in adult life.

Congenital 'microcephaly' must not be confounded with hydrocephalus. It is true that in certain small-headed infants, having the cranium malformed and the sutures ossified, an excess of fluid may be found within the head; but the fluid in these cases is situated outside the atrophied brain, and not within the ventricles. The two conditions are, in fact, totally opposite in nature.

ANATOMICAL CHARACTERS.—Three different states, in regard to size of head, have been described as existing in this affection:—(1) where the head is smaller than natural; (2) where the head is of natural size; and (3) where the head is more or less considerably enlarged.

Those of the first category ought not to be included at all. They are the cases of 'microcephalism' above referred to. Those of the second category could never be diagnosed with any degree of positiveness during life; and it may, indeed, be questioned whether such cases exist to any large extent, except as more or less transitory stages of instances of the disease pertaining to the third of the above categories.

Even the cases in which the head is distinctly enlarged differ among themselves, since in some of them (a) both sutures and fontanelles are widely open; while in others (b) the sutures, and perhaps the fontanelles, are completely closed. It seems probable that the latter may represent conditions into which some of the former pass, when the disease lapses into a chronic and stationary condition.

Owing to the separation of the cranial bones in young infants, this disease, when it occurs in them, soon becomes associated with an actual enlargement of the head, which increases rapidly. In consequence of the distending pressure from within, caused by the increasing size and fulness of the ventricles, the bones entering into the formation of the vault of the cranium become separated from one another, though the bones of the face remain unaltered. The frontal, parietal, the superior part of the occipital, and a small part of the squamous portion of the temporal bones become expanded and thinner than natural, at the same time that they are separated from one another—especially in the regions of the anterior and posterior fontanelles, and of the sagittal suture.

The cranial bones are often very thin, but occasionally they may be unusually thick throughout—even in young children.

The circumference of the head, even of a young child, may in hydrocephalus easily reach 24 to 30 inches, or more. The size attained by the head in certain cases has been comparatively enormous; thus, in an altogether exceptional case, recorded by Cruikshank, it is said to have measured, in a child sixteen months old, no less than 52 inches in circumference, and the amount of fluid contained within the cranium was found to weigh as much as twenty-seven pounds.

The fluid is generally slightly albuminous; possesses some saline constituents; and has a specific

gravity ranging from 1006 to 1014. Its composition agrees pretty closely with that of dropsical fluids generally.

In the great majority of cases, as already stated, the fluid is contained within the more or less distended lateral and third ventricles of the brain. The upper and lateral parts of the cerebral hemispheres, as well as the corpus callosum, become thinned and distended, so as to resemble a mere bag, the walls of which are represented externally by pale unfolded and much-flattened convolitional matter, and internally—next the fluid itself—by the lining membrane of the ventricles. This latter has become much thicker and tougher than natural; it may also be more or less granular on the surface; and often shows an increased number of distended vessels. These appearances are, however, not to be taken as an indication of the inflammatory origin of the malady, as some observers seem to suppose. They may be found, as the writer has seen, well masked, in cases where the effusion and distension have been the result of a mere mechanical congestion, produced by pressure upon the commencement of the straight sinus, owing to a tumour in the middle lobe of the cerebellum. On the other hand the writer has examined a case, in which the most careful search revealed nothing that could have produced mechanical congestion, and in which there was, moreover, no sign of anything like an inflammatory condition of the walls of the greatly distended lateral ventricles.

The shape of the ventricles and of the compressed ganglia about the base is, of course, greatly altered. The foramen of Munro may be half an inch or more in diameter. The optic and olfactory tracts and lobes are also often much altered by pressure.

The brain-substance may be even tougher than natural, because the long-continued mechanical congestion, which exists in so many cases, favours the overgrowth of the neuroglia. The brain of a hydrocephalic child, after the fluid has been evacuated, commonly weighs more than the brain of a healthy child of the same age.

In those cases in which during life the fluid has escaped from the ventricles through a rupture in the corpus callosum, the brain has been found more or less flattened and collapsed in the lower part of the enlarged cranium, while the escaped fluid occupies the arachnoid cavity above it.

SYMPTOMS.—Chronic hydrocephalus begins to manifest itself in various ways, and also, as above stated, at various ages. The great bulk of the cases are either congenital, or commence before the fifth month. But, in certain rare instances, the head may begin to enlarge long after the union of the sutures, in early adult life, or even beyond middle age.

As to modes of commencement, at least four, pretty distinct from one another, may be encountered. (1) The disease may be ushered in by a period of fretfulness and irritability, with or without the occurrence of convulsions and strabismus, before any enlargement of the head is detected. Or (2) slow enlargement of the head may be noticed as the first event. In some cases, this enlargement not only sets in, but may continue for months, till notable increase in size has taken place, and yet the child may exhibit no morbid symptom whatever. The writer has seen a well-marked instance of this in a child whose head had been enlarging for eighteen months (the process beginning when

it was a year old), and in whom, though the head was twenty-four inches in circumference, no other morbid signs or symptoms presented themselves.

(3) Chronic hydrocephalus may supervene in a child after a fall, through the intermediation of cerebellar disease. The writer had under his care a little girl four years old, who, after falling from a table and striking the occiput severely, suffered for from twelve to eighteen months from symptoms indicative of cerebellar disease, after which the head began to enlarge, and hydrocephalus became the apparently dominant condition. Complete blindness ensued, then convulsions set in, and in the midst of one of these the patient died. A tumour of the middle lobe of the cerebellum was found, *plus* all the signs of a well-marked hydrocephalus. (4) The disease may occur as a sequel of an attack resembling acute hydrocephalus (tubercular meningitis); that is to say, a child appears to suffer for a time from what is regarded as tubercular meningitis; the symptoms then undergo some mitigation; they become more or less chronic; and ultimately the head begins to enlarge, as in chronic hydrocephalus. There is some doubt about the real nature of these cases. The initial symptoms may not in reality have been those of tubercular meningitis. The chronic disease and its symptoms may occasionally be initiated in an acute manner. See MENINGITIS, POSTERIOR BASIC.

It may be easily imagined that the subsequent course of the symptoms in persons suffering from chronic hydrocephalus, beginning in these various ways, may also be subject to great variations.

As the head enlarges, or as the pressure within increases, sensations of weight or pain may be experienced. The child may show increased fretfulness and irritability; or its manner may become more dull and heavy than natural. At other times there is no noticeable change in these respects.

In the 'symptomatic' forms associated with tumours, there is apt to be vomiting of a very obstinate and paroxysmal character, together with continuous pain, marked by exacerbations. Convulsions, either unilateral or general, may also occur, as well as paralysis of one or other of the ocular muscles. In such cases, too, in comparatively early stages, ophthalmoscopic examination will frequently reveal optic neuritis, which has a tendency to go on to white atrophy, with the production of more or less complete blindness.

In later stages of the disease mental action becomes increasingly impaired; there is loss of memory, dulness, and a great tendency to sleep during the day. There may be marked weakness or actual paralysis of limbs. Children affected to this extent often keep to the recumbent position, having at last no power of sitting up, or even of raising their head from the pillow. Where the enlargement becomes extreme the weight of the head is so great that it cannot be maintained in the upright position. It has to be supported by the hand or some artificial prop; or else the child does not attempt to rise from the recumbent position. A sense of fluctuation is often recognisable. The forehead becomes prominent and overhanging, while the eyeballs are depressed; and as the face remains unaltered or even becomes emaciated, it seems altogether unnaturally small, and thus contributes to produce a most characteristic appearance ('*facies hydrocephalica*'), which is often intensified by the old-looking, and more or less blank, expressionless

aspect of the face. The appetite sometimes remains good; at other times it becomes much impaired, and a gradual emaciation ensues. Blindness, more rarely deafness, loss of smell, and impairment of other senses tend gradually to reveal themselves after a time.

COMPLICATIONS.—In all cases where the hydrocephalus is itself symptomatic of some primary intracranial disease, interfering with the proper return of blood from the ventricles and central portions of the brain, the symptoms resulting from this latter state of things are necessarily complicated with others immediately produced by the original morbid condition. Hence the very great variations encountered in the grouping of symptoms in different cases.

DIAGNOSIS.—Where the head becomes distinctly enlarged, with widely separated sutures and open fontanelles, there can be scarcely any room for doubt about the diagnosis. But before the head is distinctly enlarged, the diagnosis of chronic hydrocephalus with any degree of certainty is impossible. In many cases also where the head is only slightly enlarged, and the sutures are not opened, it may be very difficult, for a time, to pronounce an opinion as to whether or not an infant or young child is hydrocephalic. Natural variations in the size of the head are considerable; and it may also be enlarged from rickets, or from that very rare condition known as 'hypertrophy of the brain.' Even great thickenings of the bones of the head have occasionally given rise to uncertainties in regard to diagnosis. But in all these cases, in order to enable the practitioner to arrive at a trustworthy opinion, the particular form of the head has to be considered, together with the sum total of the various symptoms which may have preceded or accompanied its increase.

Whether we have to do with an instance of 'essential' or of 'symptomatic' hydrocephalus in any particular case often cannot be settled; but in others it can be decided by the existence of symptoms distinctly pointing to the presence of an intracranial new-growth.

COURSE AND TERMINATIONS.—Hydrocephalus often proves fatal in the course of a few months; or it may be less rapid, entailing death only after a year or two. Its progress is variable, however. Remissions and stationary conditions are apt to occur, with occasionally distinct exacerbations of all the symptoms.

Occasionally one of these stationary conditions becomes prolonged, and the individual may live for years. Some hydrocephalic subjects have subsequently lived on to the age of twenty, thirty, or even forty years. In a few exceptional cases a cure seems to have been effected, either naturally or under the influence of remedial agencies.

Death may take place in convulsions; from slow exhaustion with emaciation; or from intercurrent pneumonia or some other acute disease.

TREATMENT.—Very little, unfortunately, can be done, in the majority of cases, to produce decided or lasting improvement. This is especially so in those instances—only too numerous—in which the hydrocephalus is due to some scrofulous or other tumour interfering with the return of blood from the ventricles.

Blistering the scalp, with mercurial inunctions, formerly much lauded, may do a great deal more harm than good; and the same may be said in

reference to pressure of the enlarged head by strapping or bandages. This latter is a barbarously coarse method of treatment, which has happily fallen into disuse. Blistering may do good in some cases, but it should be cautiously employed.

The general health of the child must be maintained as much as possible, by the aid, if necessary, of tonics and cod-liver oil. Purgation and diuretics may also be had recourse to. Iodide of potassium may be given internally in gradually increasing doses, as even young children bear this remedy well. Bromide of potassium will also help, perhaps, to mitigate vomiting and convulsions, when these are urgent symptoms.

It may be worth while in suitable cases to try the effect of greatly diminishing the amount of fluids taken, so as to reduce the fulness of the vascular system. The writer has had reason to believe that this method is well worthy of being tried, where other means have failed, and where there is any chance of being able to carry it out.

Puncture of the head and withdrawal of small quantities of the fluid with various precautions has been occasionally practised, but with no satisfactory results. Attempts have recently been made to secure drainage of the lateral ventricles in cases of hydrocephalus by establishing a permanent channel between these cavities and the subarachnoid space. The pressure of fluid within the latter is found to be constantly equal to that within the cerebral veins, this equality being due to the even balance of secretion and absorption. If, therefore, such a channel could be kept open, a constant equable drainage of the ventricle would theoretically result. For this purpose bundles of strands of catgut and metal tubes have been introduced and left *in situ*, one end communicating with the ventricle, the other lying in the subarachnoid space, so that fluid could pass freely along them. The results obtained, however, have so far been disappointing. Surprising diminution has indeed occurred at times in the size of the head, but no permanent cure has been effected, and death has occurred in spite of some temporary amelioration of symptoms. This is probably due to two causes: on the one hand the softness of the cerebral substance tends sooner or later to block any channel made, and thus to prevent drainage; on the other, these cases are generally treated at so late a period that fatal mischief to the brain has probably resulted before relief has been afforded.

H. CHARLTON BASTIAN.

HYDROMETRA (ὕδωρ, water; and μήτρα, the womb).—Dropsy of the womb. See UTERUS, Diseases of.

HYDRONEPHROSIS (ὕδωρ, water; and νεφρός, the kidney).

DEFINITION.—A disease (usually chronic) of the kidney, generally produced by obstruction of the ureter. The obstruction leads to dilatation of the renal pelvis, and frequently of the ureter itself, the condition of the latter depending on the site of the obstruction. The dilatation and distension not only involves the pelvis and the calices of the kidney, but also the renal substance which is stretched out in the form of a layer of varying thickness. In the extreme forms of the disease one kidney only is affected, in the milder forms both may be involved. In some varieties of hydronephrosis the distension of the organ is intermittent,

and in rare instances the intermittence is, in women, associated with the menstrual period. The distension of the kidney frequently causes the appearance of a tumour of considerable size, and in the intermittent varieties of the disease the partial or complete disappearance of this tumour may occur from time to time. The fluid distending the tumour is not normal urine, as the percentage of solids and of urea in it is very low; albumen in small quantity is often present, and in some cases blood.

ETIOLOGY.—Hydronephrosis is most frequently dependent on obstruction of the ureter produced by the impaction of calculi or as a result of pressure caused by uterine, ovarian, and other pelvic growths, but especially by carcinoma of the uterus. Obstruction by calculi may be due to their impaction either at the junction of the pelvis of the kidney with the ureter or at the opening of the ureter into the bladder. Impaction of a calculus does not always lead to hydronephrosis; in some cases it may cause complete suppression of urine. Chronic peritonitis, pelvic peritonitis, and cellulitis may also produce obstruction of the ureter and hydronephrosis. Congenital anomalies, such as preternatural narrowness of the ureter or the implantation of the ureter at an acute angle into the pelvis of the kidney, not infrequently cause hydronephrosis. Movable kidney in its more extreme forms may also give rise to this condition, especially where great mobility of the organ is associated with congenital narrowing of the ureter. In rare instances anomalies of the renal artery may cause hydronephrosis on account of the pressure exerted by the anomalous artery on the ureter. Intermittent hydronephrosis is more especially associated with calculus, movable kidney, and pelvic peritonitis. Hydronephrosis recurring with the menstrual period is sometimes seen as a sequel of pelvic peritonitis or of calculous obstruction, and is apparently due to the catamenial turgescence of the pelvic organs aggravating, to a sufficient extent to cause hydronephrosis, some slight obstruction due to one or other of these pre-existent causes. In rare instances a slight degree of hydronephrosis may be dependent on such a remote cause as the obstruction due to phimosis.

Hydronephrosis is sometimes congenital and dependent on atresia of the ureters produced during fetal life; such congenital hydronephrosis may produce so great an enlargement of the kidney as to cause difficulty in labour.

MORBID ANATOMY.—In the slighter forms there is dilatation of the pelvis and calices of the kidney with some flattening of the papillae. In the more extreme forms the kidney presents the appearance of a bag, the interior of which is imperfectly divided by a number of septa which owe their origin to the fibrous walls of the calices. The renal tissue is stretched out as a layer of varying thickness, at times no more than an eighth of an inch. In some cases the pelvis of the kidney is distended to the size of an orange; in others the distension of the pelvis and the ureter is more uniform. In the slighter forms the principal alteration in the histology of the kidney is that the cells lining the tubules become hyaline and glass-like, losing their characteristic granular structure. In the more severe forms considerable desquamation of the epithelium occurs, and the interstitial tissue between the tubules becomes increased in amount. In old-standing cases the wall of the hydronephrotic sac is very largely fibrous.

SYMPTOMS.—Where the disease is partial and double, e.g. where it occurs as a complication of carcinoma uteri, it may be entirely overlooked unless special attention is directed to the probability of its occurrence. Frequently its presence is only suspected owing to the secretion of a dilute urine, and then palpation in the loins may reveal the presence of enlarged kidneys. In other forms, where the hydronephrosis is more complete but unilateral, the patient may come under observation owing to the presence of a swelling in the loin, which presents usually the typical characters of a renal tumour. In many such cases no other symptoms are presented even where the tumour is of large size. In others pain is a prominent symptom, and this is more especially the case in the intermittent varieties associated with movable kidney, calculus, &c. The pain is usually lumbar and abdominal, and does not radiate into the groin or down the thigh; it is often accompanied by nausea, vomiting, faintness, and in some cases, even where suppuration of the contents of the sac does not occur, by considerable fever. The sudden disappearance of the pain and subsidence of the tumour, accompanied by an increased flow of urine, are most characteristic symptoms of the intermittent form of the disease. The renal tumour produced by hydronephrosis has rounded outlines, and is sometimes said to be lobulated. Fluctuation can often be detected by palpation in the abdomen and the loin.

Hydronephrosis when double causes death from uræmia, and this occurs even when the obstruction is partial so as to produce only moderate distension of the kidneys. Unilateral hydronephrosis may cause serious symptoms from pressure on other organs, as, for instance, the bowel.

DIAGNOSIS.—The differential diagnosis of unilateral hydronephrosis has to be made from splenic, ovarian, and hepatic tumours, from pancreatic cysts, and from hydatids of the liver and of the peritoneum. Where the hydronephrosis is of large size it may be confounded with localised ascites. The distinction between hydronephrosis and pyonephrosis is sometimes difficult, as fever is not an invariable accompaniment of the latter, and may be present in the former.

PROGNOSIS.—Prognosis depends essentially on the cause of the hydronephrosis. Where the condition is a bilateral one, as in uterine carcinoma, prognosis is most serious. Where the disease is unilateral, the prognosis depends on the condition of the opposite kidney; and provided the hydronephrosis does not cause severe pain, and the opposite kidney is sound, the disease may cause comparatively little inconvenience. In some cases, however, severe pain is present owing to great or rapid distension of the kidney.

TREATMENT.—In cases of unilateral hydronephrosis surgical treatment is generally required. Drainage of the kidney is usually resorted to, but in many cases nephrectomy is ultimately required, as the obstruction to the ureter may be such that it cannot be relieved, and thus a sinus discharging urine is left as a result of a draining operation. Where the hydronephrosis results from calculous obstruction surgical interference may be successful in removing the obstruction. In bilateral hydronephrosis little can be done, and the condition is a most serious complication of the primary disease causing it.

JOHN ROSE BRADFORD.

HYDROPATHY (ὕδωρ, water; and πάθος, a disease).—A synonym for hydrotherapeutics. *See* HYDROTHERAPEUTICS.

HYDROPERICARDIUM (ὕδωρ, water; περί, about; and καρδία, the heart).—An accumulation of dropsical fluid in the pericardium. *See* PERICARDIUM, Diseases of.

HYDROPHOBIA (ὕδωρ, water; and φόβος, fear). **SYNON.**: Rabies; Fr. *Lyssa*; la Rage; Ger. *Hundswuth*; *Wasserscheu*.

DESCRIPTION.—Hydrophobia is an acute infective malady of the central nervous system. The specific virus, the exact nature of which is undetermined, is found abundantly in the central nervous system and more especially in the medulla.

The disease is one common to man and many of the lower animals, and it is customary to restrict the term hydrophobia to the disease in man, and rabies to that in animals. The term 'hydrophobia' is, however, not strictly accurate, as there is not necessarily any fear of water during the height of the disease, but only great difficulty in swallowing liquids; the attempts to swallow bring about attacks of convulsions, and not uncommonly the mere sight of the water or other substance provokes similar attacks.

ÆTIOLOGY.—The disease is never spontaneous, but is usually communicated through the bite of a rabid animal and the consequent inoculation with the virus that is present in the saliva. In the great majority of cases the disease is communicated to man by the bites of rabid dogs, occasionally of cats, and in rare instances of foxes and wolves. The prevalence of rabies in Russia is due largely to the frequency of wolf-bites. Hydrophobia is more apt to follow bites on exposed parts of the body such as the face and hands. Instances are on record where the disease has occurred as the result of the hand being licked by a rabid animal, and then the infection must be dependent upon the presence of some abrasion. Deep lacerated wounds are more liable to be followed by the disease than superficial bites involving only the skin and subcutaneous tissue. The bites from rabid cats are more dangerous than those from dogs, owing to the tendency of the cat to 'fly' at and bite exposed surfaces such as the face and hands. Wolf-bites are dangerous owing to their severe and lacerated nature. It has been asserted that the disease may arise from the bite of a healthy dog, but this is not the case; the mistake has arisen from the fact that a dog may communicate the disease while suffering from the initial manifestations of the illness, and before the characteristic symptoms have been developed (*see* **RABIES**). A considerable proportion of persons who are bitten by rabid dogs do not develop hydrophobia, and it has been estimated that only from 16 to 25 per cent. of persons so bitten develop the disease. Other authorities state that when no preventive measures are adopted half or even two-thirds of those bitten escape. There is great difficulty in determining this question with accuracy owing to the difficulty of obtaining evidence that the dog or other animal reputed to be rabid was really so. The virus of rabies is not only present in the fresh saliva, but the latter when dried retains its virulence for some days (*see* **RABIES**). Hydrophobia has in rare instances been acquired through a wound received during the *post-mortem* dissection of a rabid animal.

The *incubation-period* of hydrophobia in man is usually six weeks; it has been asserted that it may be as short as three weeks, and in exceptional instances incubation-periods of twelve or even of eighteen months have been described. It is more than probable that in the latter instances some fallacy was introduced: either that the disease developed was not hydrophobia, or else that a fresh inoculation has occurred without the patient's cognisance. It is quite possible for hydrophobia to be communicated to the human subject by the lick of a dog suffering from the disease as mentioned above, or even, as in a case mentioned by Gowers, by the unloosening with the teeth of a knot on a rope with which a rabid dog had been tied. Such modes of infection as this make it probable that in the cases with long incubation-period there has been really a fresh infection.

MORBID ANATOMY.—The most characteristic naked-eye features in *post-mortem* examination are congestion in the fauces, larynx, trachea, and stomach, and fluidity of the blood. The salivary glands sometimes show obvious signs of congestion, and in some cases congestion of the central nervous system, and more especially of the medulla, is visible to the naked eye. A number of observers, more especially Clifford Allbutt and Gowers, have described an accumulation of leucocytes around the vessels and the infiltration of the adjacent tissue of the bulb with similar cells. This change has a special distribution, as it is most marked in the region of the medulla, more especially in the lower part of the fourth ventricle in the vicinity of the respiratory centre. Gowers described the change as most intense in the hypoglossal, glosso-pharyngeal, and vagal nuclei. Similar conditions are present in the cerebral convolutions and also in the spinal cord. There is little or no affection of the upper part of the medulla, cerebellum, or basal ganglia. In addition to the above changes minute extravasations of blood may occur, and the ganglion-cells of the parts affected have been described as undergoing granular degeneration. Babes also describes hyaline degeneration of the vessels of the spinal cord and brain; he states that in some cases actual obliteration of the vessels may thus be produced. The salivary glands may also show evidence of inflammation on microscopical examination.

SYMPTOMS.—During the incubation-period there are usually no symptoms, and the wound heals without producing any signs of general disturbance. Towards the end of the period of incubation, the site of the wound may become irritable, and tingling and itching often occur. Doubtless in some instances this is dependent on the fact that the patient's attention is directed to the bite, but in other cases these sensory symptoms may be very marked and too pronounced to be thus explained. During the latter part of the incubation-period the patient's mental condition may also become altered, and depression, restlessness, and nervousness become marked. These latter symptoms, however, are perhaps more accurately described as the initial symptoms of the actual malady. In most instances the outbreak of the disease is not accompanied by any local phenomena in the wound; in others the sensory symptoms described above become more marked so that actual pain is experienced, and it has been stated that the wound may break open. The invasion in man is usually accompanied by considerable mental depression along with some

tightness or other unpleasant sensation about the throat, and sleep becomes disturbed. There is often marked pallor, slight and intermittent delirium may be present; and the patient complains greatly of thirst. Very soon, however, there is distinct difficulty in swallowing, especially of liquids, and the attempt to swallow causes spasmodic contraction of the muscles of the throat so that the fluid may be expelled from the mouth. Very soon the spasm is not confined to the muscles of deglutition but involves also those of respiration, occasioning what has been called a 'catch in the breath.' The violence of the respiratory spasm rapidly increases so that in a very few hours what was merely a catch in the breath becomes a violent inspiratory effort. Later still the spasm is no longer limited to the muscles of respiration but becomes more widely spread, so that general convulsive attacks recur at every attempt to swallow.

During the initial stage of the disease, while the spasms are still limited, the mental anxiety and dread of the patient increase, although consciousness is retained, and the patient may be quite rational and able to talk, the interference with respiration being only slight, and causing a mere temporary interruption of his speech. The restlessness and agitation, however, rapidly increase and produce a condition of extreme dread and fearfulness, which is one of the most characteristic features of hydrophobia. The extreme dread may only exist intermittently, but more usually it continues and undergoes an increase during the paroxysmal seizures. It is stated that during the paroxysm the frenzy of the patient is such that he may attempt to bite those round about him, but in the few cases of hydrophobia that the writer has seen this has never occurred. Consciousness is usually retained in the intervals between the paroxysms. The convulsive attacks in the fully established disease are not only produced by attempts to swallow, but may be excited by the mere sight of water or food, or even by any other slight visual or auditory stimulus, such as the light falling on the bed or a person entering the sick room. Vomiting may occur, and is sometimes an early symptom. There is usually some pyrexia at the onset of the disease, the temperature being raised two or three degrees. In exceptional instances it may rise as high as 105° F. Albumen and sugar may be present in the urine, and priapism is occasionally a marked symptom. The saliva frequently accumulates about the mouth, and the difficulty of getting rid of the accumulated mucus, owing to the partial palsy of the respiratory and other muscles, leads to a peculiar cough, which is the ground for the statement that the patient may 'bark like a dog.' Similarly the frothing at the mouth is dependent on the saliva and stringy mucus adhering to the lips. The duration of the disease in man after the onset of symptoms is usually four days or less. Cases lasting six, eight, and even ten days have been described. Death commonly occurs from exhaustion or from asphyxia, due to palsy of the respiratory mechanism. Some cases are said to die from cardiac failure, and exceptionally general paralysis like that seen in rabies occurs.

VARIETIES.—Although the above description applies to the great majority of cases, in some the mental symptoms, such as restlessness and furious excitement, may occur early in the course of the disease, before the characteristic features of the malady can be detected, and may thus produce a

clinical picture resembling acute mania. In others, but more rarely, a condition similar to paralytic rabies (*see RABIES*) is produced, that is to say, an ascending paralysis involving first the extremities and finally the muscles of respiration. It is probable that paralytic rabies has occurred in man, and most frequently as the result of the bite of a rabid cat. In ordinary hydrophobia death occurs prior to the onset of marked paralysis, but in these paralytic cases the condition is not preceded by any of the ordinary convulsive symptoms.

PATHOLOGY.—The virus of rabies is probably dependent upon the activity of some micro-organism, although the existence of such an organism has not been clearly established. The disease exhibits some resemblances to tetanus, in which it has been shown that the principal phenomena of the malady are produced by the toxic substances elaborated by the specific bacillus. The most remarkable fact about the virus of rabies is the power that it possesses of increasing in the nervous system. In the earlier stages of the malady the virus is present most abundantly in the medulla, but before death it is found throughout the nervous system. It is, however, not limited to this, but is also found in the saliva (especially, it is said, in the parotid saliva) and also in the secretions of the lacrymal, mammary, and pancreatic glands. Most observers are agreed that it is not present in the blood or in the tissues of the body generally, nor in any of the excretions or secretions except those mentioned. The virus is also found in the peripheral nerves, and may be present in the supra-renal bodies. There is some evidence tending to show that the virus inoculated by a bite reaches the central nervous system through the nerves, and it is perhaps for this reason that lacerated wounds involving nerves are more liable to be followed by the disease. It is possible that the virus may be conveyed from the peripheral nerves to the central nervous system through the lymphatics. The action of the virus is of such a character as first of all to increase the functional activity of certain of the bulbar centres, and more especially to exaggerate their reflex excitability, in a manner somewhat analogous to the action of strychnine. This is especially seen in the fact that the spasm produced first of all by attempts at swallowing may, instead of being localised to the muscles of deglutition, spread to other groups, and so produce a general convulsive seizure. Further, the reflex excitability is so increased that these convulsions may be brought about by other and slighter causes, such as visual and auditory stimuli. In this respect the action of the virus of rabies possesses a certain resemblance to that of strychnine, though in the case of the latter substance the main effects are exerted on the reflex functions of the cord, whereas in the case of the former the primary effect is exerted on the bulbar centres. The excitatory effect produced by the virus of rabies is, however, an initial one, and is followed by a condition of depressed activity of these centres and of those in the spinal cord, resulting in palsy. The action of the virus of rabies, however, is not limited to the bulbar and spinal centres, since it produces excitatory effects also on the cortical centres, in this way causing mental excitement, delirium, and even mania.

DIAGNOSIS.—The clinical diagnosis of hydrophobia does not usually present very great difficulties, owing to the peculiar character of the

respiratory spasm. Further the mental phenomena, and especially the great dread that is so characteristic of the malady, afford useful aids, and in a number of cases the history of a bite can be obtained. Hystero-epilepsy, mania, and tetanus are the diseases which are most liable to be confounded with the ordinary form of hydrophobia in man. From tetanus it can usually be distinguished not only by the characteristic respiratory spasm, but also by its long incubation-period, and by the absence of trismus and of that persistent rigidity which is seen in tetanus. A mistake is only liable to arise if too much stress is laid on convulsive seizures, since these are often very well marked in cases of hydrophobia. In some cases considerable difficulty may be experienced in distinguishing between hydrophobia and acute mania, and here again stress must be laid on the presence of the respiratory spasm and the history of a bite. In some cases, perhaps, clinical differentiation between the two diseases is impossible, and in rare instances it has been only by experimental inoculation after death that cases of supposed mania have been proved to be cases of hydrophobia. The difficulty perhaps that presents itself most often is to distinguish between hydrophobia and functional disturbance of the nature of hystero-epilepsy, to which the term 'lyssophobia' has been applied. This is a condition which is apt to arise in persons who have been bitten by a dog and who have a great dread of hydrophobia. In lyssophobia difficulty of swallowing may be present, but it is of the nature of globus hystericus, and is accompanied by hysterical convulsions; true respiratory spasm, such as is seen in hydrophobia, is not present. It is probable that hydrophobia is more often mistaken for mere functional disturbance than *vice versa*. In the spurious form the symptoms soon subside with the application of ordinary remedies directed to soothing the patient's mental excitement. It is stated that difficulties in differentiating between hydrophobia and certain organic brain-diseases accompanied by convulsions and delirium may sometimes arise.

PROGNOSIS.—Hydrophobia is looked upon as an almost invariably fatal disease, although it is stated that recovery has, in very exceptional instances, occurred in man. There must, however, always remain a certain amount of doubt as to the exact nature of these cases. When the symptoms of the disease are once fully established, the patient usually dies within four days.

TREATMENT.—The most essential point is unquestionably the prevention of hydrophobia by adequate measures directed to prevent the occurrence of rabies. See **RABIES**.

When the patient has been bitten by an animal suspected of being rabid, the animal should be isolated with efficient precautions to prevent danger to others, since observation of the phenomena exhibited by the animal will in a very few days determine with certainty the question whether it is or is not rabid. If the suspected animal be immediately killed, as is often the custom, this may involve the delay of three weeks or more in determining with certainty the existence or non-existence of rabies. The circulation should be temporarily arrested in the part bitten by compression above the seat of the bite, and the wound should be thoroughly washed as soon as possible, and efficiently cauterised with strong nitric acid

or with a strong antiseptic such as carbolic acid. Nitrate of silver may be used for the purpose, but is not such an efficient caustic as nitric acid. The actual cautery may also be used, and it is probable that excision of the part bitten is beneficial. Cauterisation without arrest of the circulation is not very efficient. The wound may be sucked if there are no wounds or abrasions on the lips or in the mouth of the person performing this act; the mouth should at once be thoroughly washed out with some efficient antiseptic. It is usually stated that the disease does not ensue if efficient cauterisation with temporary arrest of the circulation be immediately carried out after the bite, and if the wound is a simple one. If the animal that has inflicted the bite is definitely known to be suffering from rabies, the patient should undergo the prophylactic treatment of Pasteur with as little delay as possible. It is very unwise for the treatment to be delayed until experimental evidence is obtained of the existence of rabies in the animal, as this may involve a delay of three weeks, and the Pasteur treatment requires a fortnight for its performance.

In the *Pasteur treatment* an emulsion prepared from the desiccated spinal cord of a rabid rabbit is injected subcutaneously, and the treatment is based on the fact that desiccation causes a progressive diminution in the activity of the virus present in the central nervous system. On the first day of the treatment an emulsion of the spinal cord that has been desiccated for fourteen days is employed; on the second day a similar emulsion prepared from a cord desiccated for twelve days is given, and so on, until on the fifteenth day of the treatment an emulsion of a spinal cord that has only been desiccated for three days is injected. A large body of facts have been published in the 'Annales de l'Institut Pasteur,' showing that by this mode of treatment the number of cases of hydrophobia occurring in man from the bites of rabid animals has been diminished to less than one per cent. The treatment to be efficacious should be begun within a week of the infliction of the bite.

Various attempts have been made to prepare an antitoxic serum by injecting the attenuated virus of rabies into animals, and there is a considerable amount of evidence that under these circumstances the blood of sheep and other animals develops an antitoxic substance, and that a protective serum may in this manner be prepared. Although this has been shown to possess valuable properties, it has not been used in the prophylactic treatment to the same extent as the Pasteur method.

When the disease has declared itself in the human subject little can be done except to relieve the more distressing symptoms. A large number of remedies have been vaunted as capable of curing the disease, the most notorious of these being the well-known vapour-bath treatment. This has been shown to be entirely useless in the genuine disease, the reported cures being in all probability cases of so-called lyssophobia. Chloroform, morphine, bromide of potassium, and chloral hydrate may all be used to diminish the violence of the convulsions and to allay the sufferings of the patient. Curare has also been employed in this disease, and it has apparently been successful in a small number of instances. This drug is tolerated in large quantities in this disease, and may be given in doses of from one sixteenth to one third of a grain, repeated every quarter or half hour, until the severity of the

paroxysms is lessened (Gowers). There is considerable risk of producing in this way such a degree of muscular palsy as to necessitate recourse to artificial respiration. The patient should be placed in a darkened room and absolute quiet maintained, since sudden noises, the presence of strangers, or light, may all produce convulsions. As much nourishment as possible should be given, and owing to the difficulty in swallowing it should be administered *per rectum*. Tracheotomy is probably useless. Those in contact with the patient should take care that in his struggles and furious excitement they are not bitten, and should disinfect the face or hands from any contamination by the saliva. A bite inflicted by a person suffering from hydrophobia should be treated in the same manner as one from a rabid animal. The danger, however, is small, as there is no record of the disease having been communicated from one human being to another.

JOHN ROSE BRADFORD.

HYDROPS (ὑδῶρ, water).—A collection of drop-sical fluid in any part of the body. See DROPSY.

HYDRORRHACHIS (ὑδῶρ, water; and ῥάχις, the spine).—A collection of fluid in the spinal canal. The term is commonly used as a synonym for spina bifida. See SPINA BIFIDA.

HYDROSALPINX (ὑδῶρ, water; and σάλπιγξ, a tube).—A local collection of serous fluid in a Fallopian tube. See FALLOPIAN TUBES.

HYDROTHERAPEUTICS (ὑδῶρ, water; and θεραπεύω, I treat).—SYNON.: Water-cure; Hydro-pathy; Fr. *Hydrothérapiutique*; Ger. *Wasser-heilkunde*.

This article contains (1) notes on the internal use of water, and on the more common hydrotherapeutic procedures; and (2) a consideration of the morbid conditions suitable for hydrotherapeutic treatment.

Internal Therapeutic Use of Water, and the more Common External Hydrotherapeutic Procedures.—The dietetic necessity of water is well known; life cannot exist without it; all our tissues contain an indispensable proportion of water; we constantly lose a large amount by respiration, and by all excretions; all the internal functions of tissue-change are dependent on a certain quantity of water. This want is supplied by the solid and fluid food which we take, water included; while temporary excess of supply leads to increased discharge by the excretions, and temporary deficiency to a diminution of the water of the excretions. An increased ingestion of water further leads, for a time at least, to an increased removal of the products of retrogressive tissue-change; the tissues and the blood itself are, so to speak, washed out by it; and, as the consequence of the increased removal of the used-up material, the body is enabled to take in a larger amount of new substance, and hence we observe not rarely increase of weight as the effect of plentiful water-drinking, if not carried to excess as regards quantity and time; the secretions of the urine, bile, saliva, and pancreatic juice appear to be increased by the abundant internal use of water, as well as the perspiration; though the latter to some degree requires the concomitant influence of high external temperature or bodily exercise.

Water has also an important share in all internal

courses of mineral waters. Used by itself, it can exercise some good influence in cases of gout and gravel, in hæmorrhoidal complaints, imperfect secretion of bile, and constipation from sluggish peristaltic action. As, however, excessive water-drinking, according to Priessnitz's original plan, is apt to cause dyspeptic troubles, water is now, in general, used internally only either for dietetic purposes, or to assist in other courses of treatment.

The *external use* of cold water admits of a very great variety of applications, and a corresponding variety of effects on the body. The two main effects of the different forms of cold baths are *abstraction of heat*, with its further influences on the functions of the body; and *stimulation of the cutaneous nerves*, and through these of the nerve-centres. The two effects are usually combined, but in some forms of bath the stimulation or the *exciting effect* preponderates; in others the abstraction of heat, with its *calming or depressing influence*. Hence the different forms of baths, or rather hydrotherapeutic procedures, may be divided into *stimulating and calming*, but it is to be borne in mind that there is no strict line of distinction. With this limitation we may regard as *stimulating*—the full cold bath of short duration, the stimulating action of which is increased by motion of the water, be it natural or artificially imparted; the rapid wash-down, either by means of a large sponge, or a wet sheet, with or without friction; the spouting of the back, and the pail-douche; the needle-bath or circular shower-bath; the different forms of the rain-bath, and the usual shower-bath; the great variety of other douches; and the running or flowing sitz-bath. The immediate effects of these stimulating forms, in a constitution endowed with a certain amount of reactive power, are exhilaration, increased activity of circulation and muscular force, and improved appetite and digestive power. By altering the duration of the bath and the temperature of the water, the effects may be considerably modified, and thus adapted to different conditions. In adult as well as young persons, most beneficial effects are often produced by the combination of warm baths with cold douches on the spine.

The more *calming* forms are—the wet sheet-envelope, entire or partial; the impermeable wet compresses; the full cold bath of long duration and without motion; the sitz-, the shallow, and foot-baths without motion; and the full bath of higher temperature. Depression through abstraction of heat exceeds the stimulation in these forms: diminution of nervous irritability, of sensation and mental activity, and of the frequency of the pulse and energy of circulation; a feeling of lassitude; and a tendency to sleep, are the principal effects. These forms can, however, be modified, and the effects vary in proportion. Thus the wet sheet-envelope allows ample variation by using warm or cold water, by using the sheet dripping or wrung out, by making it fit tightly round the neck, by moving it to and fro, by frequently changing it, &c. The calming and stimulating form may be further combined by using, first, the wet sheet-envelope, or the woollen blanket-envelope, for a sufficient period to produce perspiration; and then a more or less cold bath or shower-bath of short duration. The physician has, indeed, infinite varieties of application at his disposal, to be used according to necessity.

Powerful and most important hydrotherapeutic helps are the different vapour- and hot-air baths (Russian, Roman, Turkish), combined with douches and baths of various temperatures. These kinds of baths are, however, treated of in another article. See BATHS.

A plain nourishing diet, without or with only a limited amount of stimulants; outdoor exercise in proportion to the strength of the individual; and in some cases massage, in others active or passive gymnastics, are likewise to be regarded as valuable adjuncts in the hydrotherapeutic treatment of chronic diseases; for massage and muscular exercise mean not only increased action, oxidation, excretion, and development of muscle, but also increased general circulation and respiration, increased inhalation of oxygen, and increased production of heat, so necessary in the cold water-cure. There is also no reason whatever why suitable pharmaceutical remedies should not be combined with the water-cure treatment—a method which, as already mentioned, is frequently adopted in the best establishments.

Therapeutic Effects, and Morbid Conditions suitable for Hydrotherapeutic Treatment.—The principal results of well-adapted courses of cold-water treatment are: improved nutrition and action of the skin; increased tone of the nerve-centres; regulation of the circulation; amelioration of the sanguification and nutrition; and acceleration of the retrogressive tissue-changes. It is essential for such successful results that the organism be able to stand a certain amount of abstraction of heat; that it be capable of more or less energetic reaction; and that the digestive and assimilative organs be able to take up a fair amount of nourishing material, which is required by the increased demand on the body.

Acute febrile diseases.—Among the oldest therapeutic uses of the cold bath, though it has only lately been more extensively revived, is the employment of different forms of cold baths in acute febrile diseases, attended by a high degree of pyrexia. The moderately cold, or the cooled-down bath, is the form principally employed; but cold effusions, the shower-bath, the wet envelope frequently changed, cold compresses, the application of ice, washing with iced water, and iced enemata, are likewise applicable; and the liberal internal allowance of cold water forms an important part of the dietetic management of this class of diseases. See FEBRIFUGES.

Typhoid fever.—Typhoid fever is the disease in which this treatment, with numerous modifications, has been most generally adopted. As soon as the temperature of the patient reaches $102^{\circ}2'$ to 103° F., he is placed in a bath of about 90° F., and the temperature is gradually cooled down, by the addition of cold water, to 80° or 60° F., according to the patient's power of reaction. The patient is kept in the bath generally from 10 to 15 or 20 minutes, when slight shivering often manifests itself. The patient's temperature, measured in the rectum, is usually reduced by this procedure about $1\frac{1}{2}^{\circ}$ to 5° F., not immediately, but within the first hour after the bath. As often as the temperature may again reach $102^{\circ}2'$ to 103° , the patient is again placed in the bath. Thus, during the height of the pyrexia three to five baths may be required in twenty-four hours, while later on about two are usually sufficient, and often only one. Instead of the bath gradually

cooled down, a bath of a temperature between 60° and 90° F. may be given, according to the condition of the individual patient. The frequent and careful use of the thermometer is an essential element in this method of treatment, which may be, as it often is, advantageously combined with the administration of alcohol, quinine, and other remedies. The earlier the baths are commenced, the greater seems to be their influence in mitigating the severity of the disease and its sequelæ, and in shortening its duration. It is claimed that the mortality is considerably less with this than with the expectant or any of the other usual modes of treatment.

Hyperpyrexia.—A still bolder use may be made of the cold-water treatment in those rarer cases of hyperpyrexia occasionally occurring in the course of rheumatic fever, when the temperature rises to 108° F. and more; and where very cold and prolonged baths, the application of ice-bags, &c., appear to be the only means of saving life. The same treatment is likely to be useful in some forms of sunstroke; and also in some cases of hyperpyrexia arising from other acute disease than rheumatic fever.

Scarlet fever.—In scarlet fever the writer has found warmer baths (80° to 98° F.) more generally applicable than quite cold or cooled-down baths, though in cases attended by a high degree of pyrexia and brain-symptoms these are preferable.

Hectic fever.—In hectic fever, connected with various chronic diseases, the effect of hydrotherapeutic treatment is less decided, and not yet sufficiently tested.

Digestive derangements of the most different kind, associated with *sluggish venous circulation* in the abdominal organs—conditions which may be grouped together under the term *abdominal venosity*, tendency to hæmorrhoids, to hypochondriasis, &c.—are often the objects of the water-cure, which may be useful by stimulating the physical and psychical energy of the nervous system, as well as the nutrition and tissue-change, by invigorating the skin. Habitual constipation from this cause is often relieved by the hydrotherapeutic belt. In this class of cases the common salt waters and the alkaline sulphated waters are more frequently used, and are often preferable; they may, however, be advantageously combined with judicious hydrotherapeutic treatment.

Chronic metallic poisoning may be treated in some cases with equal benefit, if there is sufficient reactive power, at cold-water establishments, as at the thermal sulphur and simple thermal spas. The external hydrotherapeutic procedures, aiming at increased perspiration and tissue-change, are in this class aided by the abundant internal use of water, in order to wash out the tissues, and especially the liver.

Skin-weakness or atony of the skin is often the cause of frequently recurring attacks of diarrhœa with neuralgic pains, of tendency to catarrh of the respiratory mucous membrane, and of rheumatism. Gently stimulating hydrotherapeutic appliances, with gradually increasing energy, are here mostly useful, unless, as in impeded convalescence, the reactive power is so reduced that the gaseous thermal salt-baths and mountain-air are preferable, while in other cases sea-air and sea-baths are successful competitors of the water-cure. See SEA-AIR; SEA-BATHS; and SEA-VOYAGES.

Hysteria.—In hysteria and hysterical affections the water-cure has obtained many good results, not by the internal use of water, but by the milder forms of baths. Functional hyperæsthesia and anæsthesia, hemicrania, spinal irritation, intercostal neuralgia, and other forms of neuralgia depending on imperfect nutrition and tissue-change, are likewise often benefited.

Organic diseases of the nerve-centres are not suitable for treatment in cold-water establishments, excepting occasionally for palliative purposes.

Rheumatism and gout.—In muscular rheumatism the original supporters of the water-cure considered their plan as infallible, but this is by no means the case. The diaphoretic methods, namely, the woollen blanket-pack and the wet sheet-envelope, often prove useful; but we know also of many failures even in good establishments. The exposure to all weathers during the cure ought certainly not to be imitated by such invalids, and the access of cold air to the wet body should be more carefully avoided than is often the case. The course must not be prolonged too much at one time, but may be repeated after an interval of months, which may be spent with advantage at sheltered seaside localities, at moderate elevations, with the help of pine-leaf baths, or at one of the gaseous thermal saline spas.

Rheumatic and gouty swellings of joints require great care in their management. The enfeebled invalid is rarely a fit object for the ordinary water-cure; but the stimulating local compress, more or less impermeable, is a useful element in the treatment of such cases.

Milder cases of *gout* may expect benefit from the usual hydrotherapeutic treatment, in so far as it aims at increased retrogressive tissue-change, and invigoration of the nervous system, especially if this treatment is associated with great moderation in the use of stimulants, and also of food in general; but local packing not rarely causes fits of gout. The more serious forms of gout are too much complicated with various defects of constitution to encourage us in recommending cold-water treatment.

Chronic affections of the skin.—In some diseases of this kind, such as prurigo, urticaria, eczema, and local perspirations, a more or less modified hydrotherapeutic treatment is an important adjuvant.

Syphilis.—The favourable results obtained in syphilis have greatly contributed to the reputation of the water-cure; but the latter is only an excellent adjuvant to medicinal treatment in these cases, in the same way as the sulphur-waters are; and many of the cures of so-called *lues* may be regarded as cures of *mercurialism*.

Catamenial irregularities are not rarely treated at hydrotherapeutic establishments. Profuse menstruation is often checked by the regular use of the cold hip-bath of short duration, namely, three to five minutes; in insufficient menses, on the other hand, warm hip-baths of ten to fifteen minutes' duration are frequently useful, combined in some cases with the wet sheet-envelope; and dysmenorrhœa is likewise occasionally treated with advantage by the partial wet sheet-envelope.

This list of morbid conditions which may be more or less benefited might easily be increased. This is not astonishing if it is considered that hydrotherapeutic treatment can be infinitely modified and adapted to the powers of the constitution; and that it may be assisted by varying hygienic, climatic,

dietetic, and medicinal influences. For we must reiterate that there does not exist any antagonism between hydrotherapeutic and other rational treatment, the former being, in fact, only part of the latter. Hence, however, it is also evident that the treatment in well-arranged hydrotherapeutic establishments ought to be under the guidance of the most intelligent physicians, just as is the case at all the best spas. Indeed the physician to such an establishment ought to be of a high class, possessing in a more than usual degree the gift of recognising all the individual peculiarities of the constitution, especially the amount of reacting power, adapting the principal remedy to every individual case, and combining other elements of treatment with hydrotherapeutic management wherever this is necessary. In the same way as we demand in suitable cases the administration of other remedies together with water-treatment in hydrotherapeutic establishments, so we must also express a wish that, apart from such establishments, hydrotherapeutic elements should be more generally combined with the usual medical treatment. For this purpose it is to be desired that well-conducted establishments should be in or near large towns, in order that persons following their usual occupations might undergo certain kinds of treatment at such establishments, or that attendants from such establishments might be sent to the houses of invalids.

HERMANN WEBER.

HYDROTHORAX (ὕδωρ, water; and θώραξ, the chest).—Dropsy of the pleura. See PLEURA, Diseases of.

HYÈRES, France.—Town three miles from the sea. Mean winter temperature, 55° F. Well sheltered; little rainfall. See CLIMATE, Treatment of Disease by.

HYGIENE (ὁγίεια, health).—The science and art which relate to the preservation of health. See PERSONAL HEALTH; and PUBLIC HEALTH.

HYGROMA, CYSTIC.—See CYSTIC HYGROMA.

HYMEN, IMPERFORATE.—See MENSTRUATION, Disorders of.

HYPÆSTHESIA (ὕπoς, under; and αἴσθησις, sensation).—Diminished sensibility of a part. See SENSATION, Disorders of.

HYPERÆMIA (ὕπερ, over or excessive; and αἷμα, blood).—Excess of blood in a part. See CIRCULATION, Disorders of.

HYPERÆSTHESIA (ὕπερ, over; and αἴσθησις, sensation).—Increased sensibility of a part. See SENSATION, Disorders of.

HYPERALGESIA (ὕπερ, over; and ἄλγος, pain).—Undue sensibility of a part to painful impressions. See SENSATION, Disorders of.

HYPERIDROSIS (ὕπερ, excessive; and ἰδρῶς, sweat).—Excessive perspiration; also termed *hidrosis*, *ephidrosis*, and *sudatoria*. See SUDORIPAROUS GLANDS, Diseases of.

HYPERINOSIS (ὕπερ, over; and ἵς, ἰνός, flesh).—Excess of fibrin in the blood. See BLOOD, Morbid Conditions of.

HYPERMETROPIA (ὕπερμετρος, beyond measure; and ὥψ, sight).—A congenital or acquired error of refraction of the eye, in which, owing to low refractive power of the dioptric media, or too little convexity of the refracting surfaces, or unnatural shortness of the antero-posterior axis of the eyeball, parallel rays of light do not, while the accommodation is in repose, converge to a focus on the layer of rods and cones of the retina, as in the normal or emmetropic eye, but to an imaginary point somewhere behind. It is the opposite of myopia, and is sometimes called hyperopia or hyperpresbyopia. See VISION, Disorders of.

HYPEROPIA (ὕπερ, above; and ὥψ, sight).—See HYPERMETROPIA.

HYPERPLASIA (ὕπερ, over; and πλάσσω, I mould or form).—An excessive growth of normal tissue-elements, which may lead to hypertrophy, or to the formation of distinct tumours. See HYPERTROPHY; and TUMOURS.

HYPERPRESBYOPIA (ὕπερ, above; and πρέσβυς, old; and ὥψ, sight).—See HYPERMETROPIA.

HYPERPYREXIA (ὕπερ, excessive; and πυρεξία, fever).—Excessive pyrexia. See FEVER; TEMPERATURE; and HYDROTHERAPEUTICS.

HYPERTRICHOSIS.—See HAIR.

HYPERTROPHY (ὕπερ, over; and τροφή, nourishment).

DEFINITION.—The word 'hypertrophy' signifies excessive nourishment, but is in practice used to designate the result of excessive nourishment—that is, excessive growth. It must be carefully distinguished from mere enlargement, and only spoken of where there is a real increase in a part, or, at least, in a tissue, without alteration of quality. Hypertrophy may be *general* or *partial*.

I. General Hypertrophy.—General hypertrophy, though a remarkable condition, is of little practical importance. It is known only in those individuals of enormous size who are called 'giants.' The production of giants depends on causes entirely unknown, since it is noticeable that this condition commonly affects only one in a family, and is in its most conspicuous forms not hereditary. Giants are usually of feeble constitution, and deficient in procreative power. The name *macrosomatia* has been given to a condition equally unexplained, in which the whole body becomes enlarged in a monstrous degree. This condition has been observed to be in some instances congenital, or, at least, to begin in very early life. True general hypertrophy does not appear to be capable of being produced by any artificial means, since excessive feeding either produces hypertrophy almost confined to one tissue—namely, fat—or else fails to produce any enlargement at all.

II. Partial Hypertrophy.—By this is meant (a) excessive increase of any part or tissue of the body during the period of natural growth, either in intra-uterine or extra-uterine life; or (b) increase of a part or tissue already completely formed. According to this distinction hypertrophy may be classified as either (1) *congenital* or (2) *acquired*.

I. CONGENITAL HYPERTROPHY.—Congenital hypertrophy is that condition in which some part of the body begins from the first to grow so rapidly as

to attain a size far beyond the normal. This condition has been seen to affect one side of the body, or one limb only, which thus becomes much larger than its fellow on the other side. Such a condition might be in theory difficult to distinguish from atrophy of the opposite side, or of the other limb—that is, from hemiatrophy (see ATROPHY); but in general the hypertrophic side is so far beyond the normal size as to prevent ambiguity. One remarkable case is on record in which one leg and arm assumed the proportions of those of a giant, while the other remained unaltered. Sometimes a congenital hypertrophy occurs without this unilateral character, as in the case reported by Curling of a girl aged fifteen, who had several fingers of both hands enlarged in an extraordinary degree without any assignable cause, the equality of the two sides being nevertheless preserved. The whole head, or one side of the head, may become abnormally enlarged. Such instances, although unexplained, must, it would seem, be put into the same class as the gigantic growth of the whole of the body. Hypertrophy of special tissues is also sometimes congenital, as of the skin in Ichthyosis. Congenital hypertrophy of the hairs is known as Hypertrichosis; that of the nails as Hyperonychchia. Congenital overgrowth of bones is also observed, forming congenital Hyperostosis, or, as affecting the face, Leontiasis ossea. Hypertrophies similar to those here called congenital may occur, though rarely, in adult life. The enlarged part is found to be highly vascular, to have an increased temperature, and to preserve its normal proportions. The cause is in these cases equally unknown.

2. ACQUIRED HYPERTROPHY.—Acquired hypertrophies may be classified as follows:—

(a) *Recuperative*, consequent on increased work.
(b) *Defensive*, arising from pressure or inflammation.

(c) *Nutritive*, from increased supplies of food.
(d) *Apparently spontaneous* or of unknown cause.

(a) *Recuperative*.—Some organs and tissues are so constructed that increased functional activity causes hypertrophy. This is especially the case with the muscles. It is a matter of familiar observation that voluntary muscles increase in size when much employed, as is seen in the often-mentioned arm of the blacksmith or the leg of the ballet-dancer. In order to produce this increase, the exercise must be of a certain degree of intensity, but not excessive. It must be frequently repeated, with intervals of rest; and at the same time the nutrition of the whole body must be good. In the absence of these conditions, exercise is more likely to produce wasting. The explanation of this familiar process is still obscure.

Hypertrophy of the heart occurs in cases where that organ is made to work at a higher tension than the normal, and this higher tension can only result from increased resistance to the flow of blood, either at one of the orifices of the heart, or in the peripheral vessels. Hence the conditions most commonly giving rise to it are valvular disease, especially stenosis; and obstruction of the arteries, either by the thickening of their walls, or by contraction of their muscular coats. The right side of the heart will also become hypertrophied when any condition whatever hinders the passage of blood through the lungs. Disease of the kidneys is a frequent cause of hypertrophy of the left ventricle of the heart, though in what way is still a matter of discussion.

It is only quite clear that the kidney-disease in some way increases the resistance in the smaller arteries and capillaries of the body generally.

Hypertrophy of the smooth or involuntary muscular fibres also occurs whenever that tissue has to contract for a long period under a higher tension than the normal. Thus the walls of the bladder become thickened in cases where, from obstruction of the passages, the evacuation of urine is effected with more difficulty, and under a higher pressure, than usual. In the same way the walls of the stomach, the œsophagus, and the intestines become hypertrophied in cases of obstruction to the passage of food through the alimentary canal.

The explanation commonly given of these cases of hypertrophy in the contractile organs is a teleological one, that in consequence of obstruction the organ *has* to contract with greater force than usual, and thus becomes hypertrophied in the same manner as a voluntary muscle which is frequently exercised. It is, however, clear that this so-called explanation does not account for the connection between obstruction and more powerful contraction. The only explanation that can be given is that in these cases pressure or tension on the organ itself is the stimulus to contraction, and that the force of contraction appears to depend roughly upon the strength of the tension which produces it.

There is less evidence that nervous or glandular structures undergo hypertrophy in consequence of their increased use; but if one kidney be destroyed the other is generally found enlarged; and some authorities believe in an increase in the size of the brain from mental activity.

(b) *Defensive hypertrophy* is the natural reaction of certain tissues against slight injury or irritation, whereby they become increased and serve the purpose of protection. But if the injury be excessive, atrophy or destruction of tissue results.

Pressure, which is a mild form of irritation, produces, when moderate and intermittent, hypertrophy of the external integument; but some regions of the skin are much more affected by this cause than others. It is difficult to draw the line between this and inflammation.

Inflammation does not cause hypertrophy of organs generally, but only of the connective tissues. In most tissues, e.g. muscular, nervous, glandular, the only result of inflammation is atrophy or destruction, with loss of functional power. But it seems to be a general law that moderate inflammation, chronic or intermittent, causes connective tissue to increase. Purely fibrous structures, such as serous membranes, become thickened by chronic or repeated inflammation of moderate intensity, especially if supuration be not produced, but this increase is not always permanent. In periostitis the formation of new bony tissue causes the increase to be permanent, and thus hypertrophy of bone is a frequent consequence of inflammation. The increase of connective tissue from irritation is obviously a conservative or defensive process, placing a barrier to the extension of injurious agents, and forming a capsule round foreign bodies.

Increase of connective tissue is also constantly present in chronic interstitial inflammation of solid organs, where indeed it is often impossible to draw the line between inflammation and fibrous overgrowth, and the term Inflammatory Hypertrophy has been used. These changes are produced by some irritation or injury, or result from wasting of

the other (parenchymatous) elements of the organ. In consequence of this atrophy of the other tissues there is likely to be rather a diminution than an increase in the size of the organ or part generally.

(c) *Nutritive hypertrophy*.—It must be regarded as doubtful whether increased nourishment alone is capable of producing enlargement of any part of the body. It certainly does not necessarily do so, as is shown by the case of experimental hyperæmia, produced, for instance, by section of the cervical sympathetic nerve, in one side of the face and head of an animal, when hypertrophy is only a rare and occasional consequence. When, however, the increased supply of nutrition in the form of blood is combined with some irritation or functional stimulus, we often find hypertrophy result. Thus, for instance, reflex hyperæmia of the skin of the face, or blushing, which is produced by numerous internal causes, such as gastric or uterine derangement, may subsist for years and reach a very high degree without altering the nutrition of the part. But if there should be in addition some disturbance or inflammation of the glands of the skin, we have the condition called *acne rosacea*, in which hypertrophy is an important element. Almost the only instance that can be quoted of hypertrophy from increase of blood-supply alone is that of the corpus luteum during pregnancy, when the ovary participates in the functional hyperæmia of the uterus. The well-known experiment of Hunter should also not be forgotten, in which he transplanted the spur of a cock from its foot to its head, and found it to increase in size.

Special nutrition.—It is well known that abundant supplies of amylaceous and fatty food, especially with a full allowance of water, cause increase of the adipose tissue. It is also said that small doses of phosphorus favour the growth of bone in young animals. But there is no other instance in which it is known that any special food causes hypertrophy of any special tissue.

(d) *Apparently spontaneous or physiological hypertrophy*.—Physiological hypertrophies form an important class. One of the best instances is that of the enlargement of the uterus during pregnancy. This enlargement is clearly not the consequence of hyperæmia alone, nor of increase of the functional activity in the muscular walls, though both these conditions are present; but must proceed from some direct physiological stimulus like that which determines the growth of the embryo itself. The hypertrophy affects all parts of the organ—its mucous and serous coats, as well as the muscular walls. Enlargement of the mammæ appears to arise from similar causes; and it is even probable that swelling of the thyroid gland may, through some obscure connection with the sexual organs, be caused in the same way. Some instances of hypertrophy we cannot in any way explain, such as the apparently spontaneous enlargements of the tonsils, spleen, and thymus gland which are sometimes observed. *Acromegaly* is a form of partial hypertrophy. A peculiar hypertrophy, with deformity of the extremities, associated with pulmonary disease, has been called by Marie ‘pulmonary hypertrophic osteoarthropathy.’ See *ACROMEGALY*.

Process of Hypertrophy.—It has been a question whether hypertrophy depends upon the increase in the size of the minute elements of an organ, or only on increase of their number. There can be no doubt that the former change often

occurs. Thus, in the pregnant uterus the muscular fibres have been found from seven to eleven times as long as natural, and from twice to seven times as wide. In a remarkable case of enlargement of the nerves, described by the late Dr. Moxon, the nerve-fibres were found to be on an average three times and some of them even forty times as large as normal. When enlargement of one kidney takes place as a consequence of destruction of the other, the tubules and Malpighian tufts are found greatly increased in size. In hypertrophy of the heart, the muscular bundles are found to be thickened, though the fibrillæ are unchanged; but in most cases multiplication of the tissue-elements is the chief cause of the increase in size. To this latter process Virchow gives the name of *hyperplasia*, and it is important to remember that, though constantly occurring in hypertrophy of organs, it does not necessarily lead to the latter change.

'False' Hypertrophy.—The term *false* hypertrophy is sometimes used for a process in which an organ becomes outwardly increased in size, owing to the deposition within it of some foreign material, or to mere distension, as in a fatty liver, an emphysematous lung, or a hydrocephalic brain. The disease called 'Duchenne's pseudo-hypertrophic paralysis' is another instance. But the term is hardly needed, except as a caution.

TREATMENT.—It is obvious that no general rules can be laid down for the treatment of hypertrophy. When it is connected with increased functional activity, it is usually a favourable rather than a hurtful condition, though in some cases it may appear that the hypertrophy more than compensates the deficiency or irregularity by which it is produced. But even if this be so, the cure of excessive hypertrophy is not within our powers. The utmost that can be done is to endeavour, if possible, to check the process by which the hypertrophy is produced. Functional hypertrophy, however, which has resulted from some obstacle or undue resistance, may entirely subside when that resistance is removed. Thus if the uterus have enlarged around a fibrous tumour, it may regain its normal bulk when the tumour is removed; and we sometimes see a sensible diminution in the size of an hypertrophied heart when the derangements which produced it no longer act. Moreover, hypertrophy may be completely reduced by a general lowering of the nutrition of the body. Thus, in early stages of pulmonary phthisis, the heart may be hypertrophied; but when death occurs in a late period of the disease, the organ is rarely found enlarged, and is even wasted; though according to Peacock's tables, less so than in other wasting diseases.

J. F. PAYNE.

HYPINOSIS (ὑπός, under; and ἴς, *ivós*, flesh).—Deficiency of fibrin in the blood. See BLOOD, Morbid Conditions of.

HYPNOTICS (ὑπνος, sleep).—Measures or agents employed to induce sleep. See NARCOTICS.

HYPNOTISM (ὑπνος, sleep).—A synonym for Braidism. See BRAIDISM; and MESMERISM.

HYPOCHONDRIASIS (ὑπό, under; and χόνδρος, a cartilage).—SYNON.: Fr. *Hypochondrie*; Ger. *Hypochondrie*.

DESCRIPTION.—The term 'hypochondriasis' is derived from an ancient hypothesis that the sym-

ptoms of this disorder were due to perturbations of natural force generated in the liver and pylorus, to which idea the frequent prevalence of flatulence probably conduced. The condition thus called is really a disease of the nervous system. It is a form of mental unsoundness closely allied to melancholia, of which, indeed, it often forms the initial stage. It is characterised by a morbid anxiety, either without any, or having only very slight foundations, relative to the state of physical health. The patient thinks about his health unduly, observing himself with restless care, examining especially the characteristics of his secretions, translating into evidence of progressive organic mischief every trivial departure from perfect action of his organs, and becoming more and more absorbed in precautions against the malady with which he believes himself affected. Nothing that happens tends to the side of reassurance. If his sleep be disturbed, the symptom may be portentous, he thinks, of brain-softening; if it be sound, the patient, instead of being comforted, fears apoplexy. Constipation of the bowels signifies obstruction; a slight diarrhoea implies coming exhaustion. Everything which he reads or hears in reference to disease the patient applies to his own case, examining himself on every point thus presented to his mind, and rarely failing to find something which dovetails with symptoms of his own. For he recognises only the points of resemblance; the features of difference are unconsciously ignored. In some cases the patient is constant in referring his troubles to one particular organ. Year after year his story is the same: it is his stomach, liver, brain, or some other organ, which is in fault; but always the same. In other instances there is a vacillation quite as remarkable. Routed, perhaps, by the convincing arguments of his adviser, he is forced to yield the position which he had assumed, but only to take an equally strong one in reference to some other part of his frame. These diversities strongly recall the fixed and shifting delusions of insanity. The patient is prone to wander from one doctor to another, often carrying with him a bundle of prescriptions and a long written list of questions, which must receive categorical answers. Apparently satisfied at the time, he speedily recollects some point upon which he has not received assurance, and this, he conceives, vitiates the whole of the explanation and advice which have been given to him, and he is plunged again into his previous state of anxiety and doubt. Where circumstances do not involve forced labour for existence, the patient passes his time in chasing his health, which is always contriving to elude his grasp. If he holds an appointment, he will resign it in order to have full opportunity for studying himself; and, his occupation once gone, he finds too late that it was his best friend, and he then ascribes to his forced idleness all the ills which had induced him to seek retirement.

In many cases the most careful examination can discover no signs of disease, and the patient wears the aspect of health; or there may be a worn, anxious look. In others there may be, especially in the digestive organs, slight deviations from perfect integrity, which explain some of the symptoms, but not the exaggerated apprehensions to which they give rise.

Hypochondriasis is a chronic disorder. It may continue, and this perhaps most frequently, as a

harmless peculiarity attached to a life which is not perceptibly shortened in length, though often sadly diminished in utility and happiness, by its symptoms. Or there may be an improvement practically amounting to a cure, which will endure for a longer or shorter period. In the decline of life, however, there is very apt to be a return of symptoms, which are often then much exacerbated. Or hypochondriasis may pass into true melancholia, and then the bodily health, previously the constant object of solicitude, improves wonderfully. Indeed, nothing more is heard about it.

ÆTIOLOGY.—Hypochondriasis is very much more common in the male than in the female sex. The period of life most prone to it is from 20 to 40 years of age. It is apt to occur in those who inherit a tendency to insanity, and the disease in its own peculiar form is often hereditary. Excesses of various kinds, especially on the side of the sexual system, will precipitate the appearance and intensify the symptoms of the affection, but it is doubtful whether they can altogether originate it. The same may be said of gout, which is apt to be associated with the condition, and to complicate its symptoms. Depressing moral circumstances also are not without influence in determining an onset of hypochondriasis in those predisposed to it. This is especially true of mental strain. The frequent occurrence of some deviation from healthy condition in the liver, stomach, or bowels, which is noted in these cases, would suggest that, probably through an interruption to the perfect nutrition of the body, diseases of these viscera bear their part in the causation of hypochondriasis. The intercurrent occurrence of internal hæmorrhoids with bleeding is very common, and this would manifestly tend to keep up, if it did not originate, the disease. Stricture and chronic ulcer of the intestines are occasionally associations which probably also influence the appearance of hypochondriasis.

ANATOMICAL CHARACTERS.—There are no anatomical characters peculiar to the disease.

DIAGNOSIS.—When careful examination, which must never be omitted, has disproved, so far as is possible, the existence of organic disease tending to produce the symptoms described by the patient, it sometimes becomes a question whether the case is one of hypochondriasis or of melancholia.

In the former there is no tendency to suicide; on the contrary, a strong desire to live pervades the sufferer's mind, and impels him to endless search for the cure of his ailments. He delights in consulting medical men and entering into the minutest details which he thinks can aid them in helping him. Up to a certain point his story is frequently characterised by a logical accuracy, which fails him, however, in some point of great importance, by which the conclusions are invalidated. The melancholic patient, on the other hand, is often suicidal and always despairs of any relief to his condition, the description of which, as given by him, is confused, frequently incoherent, and unintelligible. *See* MELANCHOLIA.

PROGNOSIS.—Early and marked hypochondriasis occurring in a person with a strong hereditary taint of insanity, without any definite cause of mental depression, is of ill omen. Such a case very often drifts into pronounced melancholia.

The prognosis is more favourable, perhaps, the less strongly marked the hereditary predisposition and the more evident and adequate the immediate

causes to which the patient has been exposed, the most potent of which are sexual or alcoholic excesses, mental strain or shock, or the sudden change from a life of activity to one of forced, and, as the sequel shows, uncongenial leisure.

TREATMENT.—Moral treatment alone is of any influence in a large majority of cases. When there is, however, manifest anæmia, a history of syphilis, evidences of gouty mal-assimilation, accumulation of fæces, catarrh of the intestinal canal, or hæmorrhoids, the therapeutics proper to these conditions should be employed. Alcoholic stimulants should be avoided. Travel, especially under judicious companionship, and the encouragement of regular, definite, and useful employment for the attention and the physical powers, are the most potent means of treatment, by which the disease may be often much ameliorated, and sometimes cured. Ridicule of the patient's sufferings will rarely or never be of service, but at the same time a habit of prescribing for all the symptoms as they arise must be avoided.

T. BUZZARD.

HYPOTHERMIC MEDICATION (*ὕπὸ*, under; *δέρμα*, the skin).—**SYNON.** : Fr. *La méthode hypodermique*; Ger. *Hypodermatische Injection der Arzneimittel*.

DEFINITION.—The introduction, by means of a sharp-pointed hollow needle, of various drugs into the subcutaneous cellular tissue.

METHOD OF APPLICATION.—The needle, which must be sharp and smooth at its point, should always be *sterilised* either by holding it in the flame of a spirit-lamp for a few seconds, or by immersing it in boiling water. It is a good plan both before and after use to fill and empty the syringe several times with a hot solution of one in twenty carbolic acid. If a hypodermic tablet is used, it should be dissolved in a syringeful of sterilised water; if, on the other hand, a ready-made solution of the drug is used, the dose to be administered should be accurately gauged by moving down the button to the required mark on the piston. Both methods guard against the possibility of overdosage. A fold of skin is pinched up, or otherwise stretched, between the left thumb and index-finger, the needle is pushed firmly and sharply into the cellular tissue, and the barrel of the syringe being steadied between the right index and middle fingers, the fluid is slowly injected by pressure with the right thumb on the end of the piston. This method is generally painless or nearly so. The proceeding is modified in certain cases. Thus, ergotin should be injected deeply into the muscular tissue; and, in some instances, cocaine is injected into the cutis vera (*see* ANÆSTHESIA, LOCAL). *Local ill-effects*, such as pain, abscess, bleeding, and ecchymosis, are almost always avoidable with proper precautions. Except in some rare instances, only parts with abundant subjacent cellular tissue should be selected. Such parts as the face, forehead, and ears, as well as the neighbourhood of large vessels, are to be avoided. *General ill-effects* may arise from injections, especially of narcotics, in diabetics, alcoholics, and the subjects of renal disease. *Solutions* should be made with vehicles which fulfil the following conditions: (1) They should have a high solvent power. (Some drugs, however, can only be administered in a state of suspension.) (2) They should be permanent, not deteriorating by keeping. (3) They should be non-irritating. Carbolic acid or boric acid may often

be added to the solution with advantage. Paroleine is the best vehicle for the injection of carbolic acid, eucalyptol, iodoform, and turpentine. Solutions should be kept in glass-stoppered bottles, or hermetically sealed glass capsules. Soluble tablets are both convenient and portable.

INSTRUMENTS.—For general purposes a simply constructed and accurately graduated syringe with a capacity of twenty minims should be used. It should be adapted to an aseptic metal case with two or more needles, and a supply of silver wires to keep their canals clear. The needles, which should be sharp and smooth at the point, may be made of nickled steel or gilded platinum. The weakest part of the needle is just at the shoulder, and this should always be carefully examined before use. By an ingenious contrivance the needle may be passed through the shoulder before the latter is screwed on to the nozzle of the syringe, and any defect is thus quickly and easily remedied. Drying and consequent shrinking of the piston-packing may be obviated by a metal cap which is applied to the nozzle after using the syringe. An instrument not in frequent use may easily be kept in good condition by occasionally washing it out with hot carbolic water. Where the barrel consists of glass, the metal parts should never be attached by cement. Some makers manufacture the syringe entirely of metal. The minims are best marked on the *piston*, which should have a screw-thread on which a button runs. In this way the dose can be accurately gauged and fixed before the injection is made. The piston, which should have a simple sliding, not a screw-action, is terminated at the top by a flat knob which is pressed by the thumb when the injection is made. In some cases, as in the injection of alcohol, ether and saline solutions, syringes of larger capacity may be used to avoid multiple injections. From time to time the instrument should be boiled.

ADVANTAGES.—These are obvious, but the method has its limitations. The hypodermic injection of such drugs as morphine and cocaine should always be under the direct control of the practitioner, and ought never to be entrusted to the patient or the friends. As compared with other modes of drug-administration, the advantages of the hypodermic may be briefly summed up as follows: (1) Greater rapidity of absorption and more speedy effect as in the relief of pain, and the production of vomiting, or rapid stimulation; (2) a strictly local effect as in the injection of cocaine or β -eucaine (see **ANÆSTHESIA, LOCAL**); (3) an efficient method of administration when other methods are impossible—e.g. cases of vomiting, nausea, and other conditions due to an irritable or diseased stomach, cases of dysphagia, and certain cases of delirium, mania, or coma; and (4) less risk of a cumulative effect (Eulenburg).

REMEDIES USED HYPODERMICALLY, AND DISEASES FOR WHICH THEY ARE EMPLOYED:—

Acidum Benzoicum.—Benzoic acid, 10 grains; alcohol, to 112 minims. *Dose*, from 8 to 16 minims. (The solution should be slightly warmed before use.) *Used* as a respiratory stimulant in the collapse of acute fevers, and in uræmia.

Acidum Carbolicum.—Pure carbolic acid, 1 or 2 grains; paroleine, or water, to 112 minims. *Dose*, 8, 16, or 24 minims. *Used* in erysipelas, malignant pustule, and other infective diseases.

Aconitina.—The B.P. preparation should be employed. Aconitine, 1 grain; dilute sulphuric acid,

q.s.; distilled water, to $\frac{1}{2}$ ounce. (Martindale.) *Dose*, 1 or 2 minims. The first dose should not exceed $\frac{1}{100}$ grain. It has been *used* in various forms of neuralgia, especially of the trigeminal, and in various other diseases, but in most cases the use of the Tinct. Aconiti *per os* is preferable.

Æther.—Sulphuric ether is the best form for hypodermic use. It may be employed alone, in combination with alcohol, or with camphor (1 in 10). *Used* in cases of collapse, whether arising from injury or disease.

Alcohol, in doses of 30 to 60 minims, in the form of whisky or brandy, may be *used* for the same purposes as ether, by deep injection.

Ammonia, Liquor.—*Dose*, 2 to 5 minims hypodermically or intravenously. Of great value in syncope from any cause, and in threatened death from embolism. See **SALINE SOLUTIONS**.

Amyl Nitris.—Nitrite of amyl, 1 part; alcohol, 9 parts. *Dose*, 8 to 12 minims. *Used* in threatened death during chloroform anæsthesia, in strychnine-poisoning, and, especially, in angina pectoris.

Antipyrine, in doses of from 4 to 5 grains, cautiously increased, in fresh aqueous solution. *Used* in various neuralgias and to relieve the pains of tabes dorsalis. It should be injected along the course of the hyper-sensitive nerve. The first effect is painful, but the resulting anæsthesia often lasts for several days.

Apomorphine Hydrochloridum.—*Dose*, 5 to 10 minims of the freshly prepared B.P. solution, or tablets $\frac{1}{15}$ to $\frac{1}{10}$ grain. An over-dose may kill by causing profuse bronchial secretion (Lauder Brunton). *Used* as a prompt emetic.

Aqua.—Iced water has been used hypodermically with some success for the relief of pain (e.g. sciatica) and to reduce pyrexia. Hot water has been also used for localised pain over a limited area. But the most important method is that which is known as *Hypodermoclysis*—the '*injections massives*' of French authors. Sterilised water containing some saline, or salines, in weak solution is employed at a temperature of 100° F. The following is a useful formula: Sodium chloride, 1 drachm; boiled water, 1 pint. One to three pints of this solution may be injected 3 or 4 times daily. The apparatus required consists of a glass receiver from the lower end of which a tube leads to a fine trocar. The skin at the point of puncture (the lumbar, gluteal, scapular, or submammary region) having been sterilised, and the apparatus also rendered aseptic, the needle is pushed steadily and quickly into the cellular tissue, and the fluid is allowed to flow in slowly and evenly, the rate of influx being regulated by the height at which the receiver is placed. Diffusion and absorption may be facilitated by occasional gentle massage. When the needle is withdrawn collodion is applied to the puncture. Direct injection into the veins has been recommended and practised (see **SALINE SOLUTIONS**), but, as a general rule, the hypodermic method is preferable on account of its absolute safety.

Hypodermoclysis is invaluable in various forms of hæmorrhage, in shock, and in septic conditions of the blood. It is thus used in uræmia, septicæmia, and diabetic coma; also in the collapse of cholera and in peritonitis. In cases of urgency, when the apparatus is not at hand, rectal saline injections may be used in the first instance. Intraperitoneal saline injections have also been used successfully.

Arsenium and its preparations.—Great care is

necessary to prevent local irritation. Fowler's solution, 1 part; distilled water, 2 parts, may be employed. *Used* in various nervous and cutaneous diseases and in septic conditions. Recommended by Jaccoud and others in tubercular affections. See SODII CACODYLAS, under this article.

Atropina.—Neutral sulphate of atropine, 1 grain; distilled water, to 2 ounces 160 minims. *Dose*, 8 minims ($\frac{1}{150}$ grain). The solution must be freshly prepared. Tablets $\frac{1}{150}$, $\frac{1}{100}$, $\frac{1}{60}$ grain, dissolved in hot water, are useful. *Used* as an antisudorific and antispasmodic; also as an analgesic, especially in combination with morphine.

Auri Chloridum.—*Dose*, $\frac{1}{35}$ grain in 20 minims of distilled water. *Used* in some syphilitic affections, also to cure (?) the alcohol-habit.

Caffeina.—Caffeine, 20 grains; sodium salicylate, $17\frac{1}{2}$ grains; distilled water, to 1 drachm (3 minims = 1 grain). *Dose*, 1 to 6 minims (Martindale and Westcott). *Used* in various nerve-pains, chronic alcoholism, cardiac dropsy, and in poisoning by alcohol, opium, and cocaine.

Calabarina, *Physostigmatis Extractum*. *Calabarina*, *Extract of Calabar Bean*.—The latter is best adapted for hypodermic use. Extract of Calabar bean, 10 grains; rectified spirit, 10 minims; rub together till smooth, and add gum acacia, 10 grains; mix, and add gradually distilled water to half an ounce. *Dose*, 3 to 12 minims. *Eserine* salicylate, $\frac{1}{100}$ grain, may also be employed. *Used* in tetanus, strychnine-poisoning, and enuresis.

Chloroformum.—*Dose*, 10 to 20 minims. Rarely *used* as a substitute for morphine in various painful affections.

Cocaina.—*Dose*, 2 to 5 minims of the B.P. solution, or tabloids of $\frac{1}{6}$, $\frac{1}{4}$, or $\frac{1}{2}$ grain. Chiefly *used* to produce local anaesthesia, either alone or in combination with morphine according to Schleich's method (see ANÆSTHESIA, LOCAL). Cocaine has also been used for the relief of megrim, hemicrania, and other nervous affections, as well as to overcome the craving for morphine and alcohol. It must be used with caution. See HABITS.

Codeina.—The phosphate may be used in *doses* of $\frac{1}{8}$ to $\frac{1}{4}$ grain in 20 minims of distilled water. *Used* mostly in diabetes mellitus.

Conina.—The hydrobromate in *doses* of $\frac{1}{20}$ to $\frac{1}{5}$ grain has rarely been *used* in various spasmodic nervous affections.

Curara.—Best administered hypodermically and in *doses* of $\frac{1}{12}$ grain. *Used* in tetanus and hydrophobia.

Digitalinum.—*Dose*, $\frac{1}{100}$ grain in 20 minims of distilled water. *Used* very exceptionally hypodermically.

Ergotininum.—Ergotinine citrate in *doses* of $\frac{1}{200}$ to $\frac{1}{100}$ grain in hot aqueous solution may be used, or 3 to 10 minims of the Injectio Ergotæ Hypodermica, B.P., may be given. *Used* in uterine hæmorrhage, hæmoptysis, and other hæmorrhages. It should be injected deeply.

B-Eucaine Hydrochloride.—A synthetic substance closely allied to cocaine, is soluble 1 in 20 of water. It is less toxic than cocaine, and does not affect the pupils. *Used* to produce local anaesthesia. See ANÆSTHESIA, LOCAL.

Ferri Ammonio-citras.—*Dose*, 2 grains and upwards. Rarely *used* in anaemia.

Gelatinum.—Has been used with success in some cases of aneurysm and hæmophilia. Its use is dangerous when there is kidney trouble. *Dose*,

six to eight ounces of a sterilised solution injected into the gluteal region twice or thrice weekly. See ANEURYSM.

Hydrargyrum.—*Mercury* and its salts. Mercury should only be used hypodermically in very exceptional cases, e.g. in grave and rapidly progressive syphilitic visceral lesions, and to overcome obstinate local syphilitic affections. It must always be avoided in dyscrasic conditions, as well as in renal disease, diabetes mellitus, and atheroma. Bacelli's method may be used of injecting 20 minims of a 1 per cent. solution of the cyanide every day or every other day till the desired effect is produced, or the plan of Lang of Vienna, which is claimed to be unirritating, may be adopted. Lang uses a 50 per cent. mixture of metallic mercury with fat and oil, which he calls 'grey oil.' The mixture is warmed to a temperature of 23° or 24° C. before use, and 0.05 c.c. (= 0.04 gramme of mercury) is injected twice weekly till improvement sets in, and then every 5 or 8 days until there is apparent cure. After this, two injections at intervals of 10 to 14 days are given as supplementary treatment. Eight to twelve injections in all generally suffice.

Hyoscina and its salts.—The hydrobromide in *doses* of $\frac{1}{200}$ to $\frac{1}{75}$ or $\frac{1}{50}$ grain in aqueous solution should be employed. *Used* to quiet excitement and induce sleep, especially in maniacal cases, in puerperal mania, and in the insomnia due to worry. It is only temporarily useful in paralysis agitans. It may be used as an antispasmodic and antisudorific. It is not an analgesic.

Morphine Tartras.—Other salts may be used, but this is the least irritating. *Dose* of the Injectio Morphine Hypodermica, B.P., 2 to 5 minims. Morphine may be combined with cocaine (see ANÆSTHESIA, LOCAL) or with atropine. It is used for the relief of pain, to induce sleep, to arrest nervous vomiting, and in the treatment of some forms of diabetes mellitus. It is unsafe in structural disease of the kidneys. Great care must be taken to avoid inducing the morphine-habit. See MORPHINE-HABIT, p. 611.

Muscarina is similar in its action and uses to pilocarpine, over which it possesses no advantage.

Nitroglycerinum or *Trinitrin*.—Tablets of $\frac{1}{250}$ grain dissolved in distilled water; to be injected deeply. Of great value in anginal attacks, especially when these are due to double aortic disease. In these cases where there is insomnia and feeble cardiac action a combination of trinitrin $\frac{1}{250}$ grain, morphine $\frac{1}{4}$ grain, and strychnine $\frac{1}{10}$ grain, affords great relief. Trinitrin is also used in some forms of asthma and in sea-sickness.

Pilocarpine Nitras.—*Dose*, $\frac{1}{10}$ up to $\frac{1}{2}$ grain. *Used* as a powerful diaphoretic.

Quinina.—The hydrobromide or the hydrochloride may be used in *doses* of $\frac{1}{2}$ to 1 grain dissolved in hot water 20 minims.

Sodii Cacodylas.—This is obtained from Cacodyl, one of the series of compounds formed by arsenic with alcohol-radicles. Cacodylate of sodium, which contains 55 per cent. of arsenious acid, is best administered hypodermically, and in this way it has a much wider range of action than arsenic. Further, its use is not attended with pain, and it is less likely to disturb the system. Gauthier recommends the following formula: Sodium cacodylate, 6.40 grammes; carbolised alcohol, 10 drops; and distilled water, 100 drachms. This mixture should be boiled for a few moments, and then made up to

100 drachms with distilled water. Each cubic centimetre (16 minims) contains 5 centigrammes ($\frac{3}{8}$ grain) of cacodylic acid. One dose is given every day for 8 or 10 days, and then an interval of 8 or 10 days is allowed. The drug is very useful in tubercular diseases, in some cases of diabetes mellitus, in exophthalmic goitre, and even in leucæmia. It is useful also as a palliative in cancer of the stomach. It must not be used where there is hæmorrhage.

Sparteine Sulphate.—Dose, $\frac{1}{10}$ to $\frac{1}{2}$ grain. A useful substitute for digitalis as a cardiac tonic.

Strophanthin is very rarely used as a cardiac stimulant and tonic.

Strychnina.—The hydrochloride in doses ranging from $\frac{1}{100}$ to $\frac{1}{30}$ grain in aqueous solution is in more or less constant hypodermic use. It is an invaluable respiratory and vaso-motor stimulant. Hence it is of great value in threatened cardiac failure, especially when due to acute pulmonary disease. It is useful in failing respiratory action due to opium-poisoning. It is of great value in various forms of paralysis.

Stypticin or Cotarine Hydrochloride is obtained by the oxidation of narcotine. It is useful in various forms of uterine hæmorrhage, but it has no influence on bleeding due to neoplasms. The dose is three grains deeply injected into the gluteal muscles, to obtain an immediate effect.

Water.—See AQUA, p. 706.

ANDREW S. CURRIE.

HYPODERMOCLYSIS (*ὕπό; δέρμα; κλύσις*, a drenching).—**SYNON.**: Fr. *injections massives*.—The hypodermic injection of artificial serum or saline solutions. See HYPODERMIC MEDICATION: *Aqua*. See also SALINE SOLUTIONS.

HYPOGLOSSAL NERVE, Disorders of.—The hypoglossal nerve is the motor nerve for the tongue, and for most of the other muscles which are attached to the hyoid bone, the exceptions being the stylo-hyoid, the mylo-hyoid, and the middle constrictor of the pharynx. It also supplies the sterno-thyroid muscle.

1. Paralysis.—Paralysis of this nerve is shown chiefly by the resulting interference with the movement of the tongue—'glossoplegia.'

ÆTIOLOGY.—The hypoglossal nerve may be damaged in any part of its course by the growth of tumours, even outside the skull; but is most commonly affected at its origin from the medulla, by pressure, meningitis, syphilitic processes, or by caries of the upper cervical vertebræ. The tongue is also paralysed by disease of the nucleus of origin of the hypoglossal fibres, but its paralysis is then associated with that of the lips, and commonly also of the palate, pharynx, and glottis (see LABIO-GLOSSO-LARYNGEAL PARALYSIS). Such paralysis may be sudden, from local softening, or gradual, from nuclear degeneration. The latter form is often part of progressive muscular atrophy, due to a similar affection of the motor structures in the spinal cord. Disease of the motor tract above the nucleus also causes paralysis of the tongue, together with the face, arm, and leg of the same side. The tongue then deviates towards the paralysed side. In disease of the left hemisphere, with aphasia, there is a curious inability to protrude the tongue: this is absent in disease of the right hemisphere. Bilateral glossoplegia commonly results from disease of the nucleus or its neighbourhood, rarely

from symmetrical disease of the lower part of the motor region of the cortex in what has been termed 'pseudo-bulbar paralysis.' Unilateral paralysis, when isolated, is generally due to disease of the fibres of the nerve within or outside the medulla.

SYMPTOMS.—In unilateral paralysis, the tongue at rest is in its normal position in the mouth, but its root is higher up on the paralysed than on the normal side, in consequence of the loss of the tonic, or voluntary, contraction of the posterior fibres of the hyo-glossus. Within the mouth, the tongue is moved freely to the healthy side, but is not moved to the paralysed side. When protruded, it deviates towards the paralysed side, because the protrusion is the result of the action of the fibres of the genio-glossus, and the tongue is pushed over towards the weaker side. In bilateral paralysis the tongue lies in the mouth behind the teeth, and cannot be protruded. If the loss of power is complete, the tongue cannot be projected over the lower teeth. It is broad and flabby, if there is no atrophy, and sometimes when atrophy is associated with fatty overgrowth. When there is wasting, as is generally the case when the nerve or nucleus is diseased, the tongue is shrunken and its surface is wrinkled, on one side or both. The faradic irritability of the muscular fibres is then lost. In unilateral paralysis, articulation and deglutition are little impaired. The pronunciation of labials and the production of falsetto notes may, however, be difficult. In bilateral paralysis, articulation is much impaired. Phonation, however, is perfect, unless the larynx is also paralysed. Mastication is interfered with, because the food cannot be moved about in the mouth. Deglutition is also impaired, because the food cannot be rolled into the fauces; and soft foods, when they reach the pharynx, may be driven again into the mouth, in consequence of the absence of the natural supporting movement of the tongue. Taste is not primarily affected, but may be somewhat dulled, because the patient is unable to move sapid substances over the surface of the tongue.

DIAGNOSIS.—The position of the lesion is indicated by the associations of the paralysis. If the disease is in the motor tract above the nucleus (pons, crus, or hemisphere), there is hemiplegic weakness, partial or complete, on the side of the paralysis of the tongue. In disease of the nucleus the paralysis is commonly bilateral, is associated with paralysis of the lips and throat, and there is usually wasting. Disease of the fibres of origin within the medulla from a small area of softening may be associated with paralysis of the opposite limbs, so that the tongue deviates from the paralysed side instead of towards it. When the disease is at the surface of the medulla, the paralysis is commonly associated with that of the corresponding half of the palate and vocal cord, from disease of the adjacent spinal accessory nerve. In all these cases there is commonly wasting, but it may be slight. The diagnosis of the pathological cause of the paralysis rests on the course of the affection, and on the presence of any causal and associated condition. The chief sources of error in diagnosis arise from the fact that in hysteria the tongue is often voluntarily but persistently protruded towards one side; the nature of the deviation is recognised by its extreme degree and by the freedom of movement within the mouth. In facial paralysis, with lateral deviation of the orifice of the mouth, the tongue may be protruded, consensually, in the middle of

this, and therefore to one side of the middle line of the face, and thus may seem to deviate. A knowledge of the fact will prevent error.

PROGNOSIS.—This is usually unfavourable, on account of the gravity of the disease which damages the nerve or centre. Even in syphilitic cases, recovery is often incomplete unless early and energetic treatment can be adopted.

TREATMENT.—The treatment of paralysis of the hypoglossal nerve is that of the causal disease. Tonics, counter-irritation, iodide of potassium and mercury, with the application of electricity to the tongue if there is reason to anticipate recovery of some nerve-fibres, are the most important remedies to be employed, according to the aetiological indication. The most convenient method of applying electricity is by means of a tongue-depressor in a wooden handle, the blade being insulated by a coating of sealing-wax where it comes in contact with the lips.

2. **Spasm.**—Spasm in the parts supplied by the hypoglossal nerve is rare. The tongue participates in the convulsive movements in epilepsy, is jerked between the champing jaws, and thus becomes bitten. It is sometimes the seat of persistent tonic spasm in hysteria. Cases have been met with in which the tongue is affected with a 'functional spasm' in speaking, analogous to 'writer's cramp,' but these are so rare as scarcely to need detailed description.

W. R. GOWERS.

HYPOSPADIAS (*ὑπό*, under; and *σπᾶω*, I draw, or tear).—A malformation of the penis in which the orifice of the urethra is underneath or behind the glans. See PENIS, Diseases of.

HYPOSTASIS (*ὑπό*, under; and *στάσις*, standing, settlement).—This term is applied to that condition of the veins of a part which consists in an overfulness, with a diminution in the rate of flow of the contained fluid, caused by a failure in the propelling forces of the circulation.

HYPOTONUS.—See NERVOUS SYSTEM, Examination of.

HYSTERIA (*ὑστέρα*, the womb).—**SYNON.** : Fr. *Hystérie*; Ger. *Hysterie*.

INTRODUCTION.—'Hysteria' is a term the etymology of which is misleading, and had best, therefore, be disregarded. It is often improperly applied to cases of simple malingering, and others which do not admit of ready explanation. Its use is best restricted to a condition of the nervous system fairly defined, but the intimate pathology of which is not known, characterised by the occurrence of convulsive seizures, and by departures from normal function of various organs, leading to very numerous and often perplexing symptoms. These are apt to simulate those commonly arising from definite alterations of structure, but differ from the latter in the fact that they may often, even when at their worst, be removed instantaneously, usually under the influence of strong emotion. It would seem that there is a disturbed or congenitally defective condition of the cerebral substance, involving in all cases the highest nervous centres, and in various examples extending more or less also to some of those which preside over automatic phenomena. Partial or complete suspension of inhibitory influence would appear to be the most patent result of the condition, whatever it be; and this is re-

cognised as well in regard to the mental as to the more evidently physical processes belonging to cerebral function. A laugh which cannot be checked, but continues until tears flow or the limbs become convulsed, is a typical example of such a suspension of control, and, if studied, throws light upon the nature of a considerable portion of the phenomena of hysteria. The jerking expirations of laughter arise from excitation of the respiratory centre; and when this excitation, uncontrolled by higher centres, acquires an abnormal strength, it extends to other parts of the medulla oblongata and spinal cord, and produces general convulsions. It overflows, as it were, into other nervous centres, which in health would receive none of the exciting impulse. Between the lowest (automatic) functions of the cerebro-spinal nervous system and the highest (psychical) there is an ever-increasingly complex system of excito-motor processes, which may be in part or wholly under the pathological influence, whatever it be. Hence the *bizarre* character of the hysterical phenomena, and the circumstance that the symptoms always include modifications of those processes which underlie the mental faculties. The suspension of the power of control possessed by the higher centres explains the irregular movements, spasms, and convulsions. In hysteria, hyperæsthesia and pain would appear to be dependent upon such a molecular change being initiated in the sensory ganglionic centres as is ordinarily propagated from the periphery. Hysterical paralysis, on the other hand, signifies that the power of the higher centres in liberating movements is in abeyance. In hysterical anaesthesia it is probably feeling or sensory perception, and not the function of the more immediate sensory apparatus, which is in abeyance, while the reflex actions which result from excitation of sensory nerves may be performed in an orderly manner. A patient may work a needle with fingers which can be touched or pricked without the act being felt. Tactile impressions are conveyed to the ganglionic centres by the afferent nerves, and excite the action of efferent nerves so that the muscles are contracted. What is wanting is the participation of those higher centres in which consciousness runs parallel to this physiological action.

ÆTIOLOGY.—*Predisposing causes.*—It is probable that a state of more or less imperfect development of the higher nervous centres, of congenital origin, very frequently, if not always, underlies the various circumstances which apparently conduce to the hysterical condition.

The female sex is much more prone than the male to the affection, which usually occurs between the ages of fifteen and thirty, and most frequently of all between fifteen and twenty. Luxury; ill-directed education, and unhappy surroundings; celibacy where not of choice, but enforced by circumstances; unfortunate marriages; alcoholism; premature cessation of ovulation; and long-continued trouble—all predispose to hysteria. A somewhat frequent antecedent is a long and wearisome nursing of a sick relation, with much broken rest. The disorder is only exceptionally found in women suffering from diseases of the genital organs, and its relation to uterine and ovarian disturbance is probably neither more nor less than that which obtains in other neuroses. Exception must be made in the case of prostitutes affected with venereal disorders, who are very prone to hysteria. In this class, however, the condition is complicated by the physical and moral

influences to which their life subjects them, and among these alcohol frequently occupies a very important place. Like epilepsy, megrim, and some forms of insanity, hysteria is prone to be intensified at the catamenial period.

The occurrence of hysteria (although comparatively rarely) in males is sufficient of itself to disprove the uterine theory of causation.

Determining causes.—These include painful impressions; long fasting; strong emotions; imitation; and shock to the nervous system, physical or moral.

SYMPTOMS AND DIAGNOSIS.—In the limited space in which it is necessary that the subject of hysteria should be treated, it will be best to describe together some of the most frequent forms which the neurosis takes, and the principles upon which a diagnosis can be made. Hysteria produces symptoms which may be referred to every function of the body. For consideration they may be roughly classed in the following groups, it being understood that all may occur either coincidentally or in succession: (1) *Mental*; (2) *Sensory*; (3) *Motor*; (4) *Circulatory*; and (5) *Visceral*.

1. *Mental*.—The intelligence may be apparently of good quality, the patient evincing sometimes remarkable quickness of apprehension; but, carefully tested, it is found to be wanting in the essentials of the highest class of mental power. The memory may be good, but judgment is weak, and the ability to concentrate the attention for any length of time upon a subject is absent. So also regard for accuracy, and the energy necessary to ensure it in any work that is undertaken, are deficient. The emotions are excited with undue readiness, and when aroused are incapable of control. Tears are occasioned not only by pathetic ideas but by ridiculous subjects, and peals of laughter may incongruously greet some tragic announcement. Or the converse may take place: the ordinary signs of emotion may be absent, and replaced by an attack of syncope, convulsion, pain, or paralysis. Perhaps more constant than other phenomena in hysteria is a pronounced desire for the sympathy and interest of others. This is evidently only one of the most characteristic qualities of femininity, uncontrolled by the action of the higher nervous centres, which in a healthy state keep it in subjection. There is very frequently not only a deficient regard for truthfulness, but a proneness to active deception and dishonesty. So common is this, that the various phases of hysteria are often assumed to be simple examples of voluntary simulation, and the title of disease refused to the condition. But it seems more reasonable to refer the symptom to impairment of the highly complex nervous processes which form the physiological side of the moral faculties.

2. *Sensory*.—Pain, hyperæsthesia, and anaesthesia occur with perhaps equal frequency. The diagnosis of the hysterical origin of such alterations of sensibility is effected partly by excluding, so far as is possible, the presence of other causes, and partly by consideration of any accompanying or antecedent peculiarities of manner and conduct. Hysterical *pain*, where it is associated with some evident local change, is found to be greatly in excess of that which would ordinarily accompany the observed cause. Where pain or hyperæsthesia is complained of in situations and of a character which would commonly point to some existing inflammation, it is necessary, by examination of the pulse and tempera-

ture, to exclude such a condition. Hysterical pain is apt to cease suddenly when the attention is diverted, and to be increased by inquiry and sympathy. Some of the most common seats of pain and tenderness are the following:—

(a) The lower part of the side of the chest (usually the left) simulating intercostal neuralgia, but distinguished from it by the tenderness being widespread, superficial, and not confined to certain points. Pressure here will sometimes occasion disturbances of respiration and circulation.

(b) Some of the vertebral spines, usually in the cervical and upper dorsal region. From the error of mistaking this for commencing disease of the vertebræ numbers of young women have been confined to a couch for months and years, and their health permanently damaged. The points of diagnosis are the patient's antecedents: there is often a history of aphonia, or paralysis, or hysterical fits. Or it may happen that, long after the pain has been first complained of, the patient has been seen to take a prodigious amount of exercise *on some one occasion* without complaint. A very much slighter pressure, too, causes pain than is at all usual in vertebral caries. It has to be carefully remembered, however, that a patient affected with vertebral disease may also be hysterical.

(c) Acute pain in a joint, occurring usually some little time after a slight injury and giving rise to suspicion of inflammation, but distinguished from this by the fact that after a few days of great pain the joint does not feel hot to the touch, and is not swollen, and that the thermometer shows no rise of temperature. The pain is more easily excited, too, by touching the skin than by pressing the articulatory surfaces against each other. See **JOINTS, Diseases of**; and **PAIN IN VISCERAL DISEASE**.

It is necessary to remember that in locomotor ataxy there may be exquisite pains (of a shooting character) having their seat in a joint or its neighbourhood, and accompanied by some localised hyperæsthesia of the skin. The disease rarely affects young females, but it may do so, and the condition is then liable to be mistaken for hysteria. See **TABES DORSALIS**.

(d) Tenderness of the mamma or darting pains through its substance, recalling those of scirrhus. The absence of any lump, and the effect of engaging attention, will serve to ensure the distinction.

(e) Pain in the head of very severe character, 'like a nail being driven into the skull' (*clavus*). This is probably neuralgic, and is by no means confined to the hysterical. There is also a more diffused pain, described as of great violence and exceedingly obstinate. This pain is sometimes suggestive of cerebral tumour, from which, however, it may often be distinguished by the fact that the ophthalmoscope shows no optic neuritis, that there is no vomiting, and that localised paralysis is absent. But very great caution is necessary in coming to a conclusion that severe and long-continued pain in the head is hysterical. Errors of diagnosis in this direction are very common indeed. And here it may be well to say that in an accurate knowledge of the characteristics of the disease supposed to be simulated lies the only safety as regards the diagnosis of hysteria. Nor must it be forgotten that persons with serious organic disease are frequently affected also with hysterical symptoms.

(f) Epigastric tenderness. Careful pressure will often show that the tenderness is at the origin of the

recti abdominis muscles, and not in the stomach. But there is sometimes pain in the stomach itself, and this may be associated with disgust for food or depraved appetite.

(g) Tenderness in one or other iliac region, deep pressure upon which will sometimes evolve hysterical symptoms, and also in some cases of hysterical convulsions will check the paroxysm.

Anæsthesia and *analgesia* are apt to be found sometimes on both sides, but much more frequently in one half of the patient's body, parted off from the other by the median line, and thus involving apparently half the head, face, and trunk, as well as the upper and lower extremities, though it may be in different degrees of intensity. It not seldom happens that the patient is herself unaware of the existence of this insensibility until examination has disclosed it. The loss of sensibility sometimes affects also the special senses; and smell, taste, hearing, sight, and the perception of colour, may each or all be lost on one side. It is frequently associated with tenderness in the iliac region of the same side; or it may be limited to a small patch. The sense of touch often remains while painful impressions and those of temperature cannot be perceived. The *anæsthesia* may be confined to the surface, or involve as well the deeper structures, into which pins may be stuck without evoking signs of pain. Accompanying the *analgesia* it is often seen that the pin-prick employed to test the condition fails to draw blood on the affected side, while readily doing so in the opposite limb. The left conjunctiva is often the seat of *anæsthesia*, so that it may be touched or even rubbed without any reflex movements of the eyelids being excited. So also the pharynx may be tickled without exciting the ordinary spasmodic contraction, and the epiglottis touched by the finger without inconvenience. A very frequent symptom is loss of reflex contraction when the sole of the foot is tickled with a feather or pointed instrument. This is sometimes complete; in other cases prolonged titillation will evoke a plantar reflex of greater or less extent. Where this absence of (or extremely lowered) plantar reflex occurs in a person who has previously been highly susceptible to tickling of the foot-sole, and common sensation is still preserved, it is a very important symptom of hysteria. Such affections of cutaneous sensibility or of reflex action may have to be looked for, as they are often unsuspected by the patient herself. As regards diagnosis, the existence of peripheral nerve-lesions may be excluded by the absence of trophic disturbance or change in the electrical reaction of the muscles. The condition is not likely to be confounded with hemiplegia, unless perhaps when it has immediately followed a convulsive attack, and is accompanied by apparent loss of power in the limbs. Examination of the patient and her history will suffice for the diagnosis.

The other special senses also may be disordered in hysteria. There may be intolerance of light, subjective sense of taste or smell, roaring noises in the ears; or, conversely, loss of sight (either in half of both eyes, or in one eye), loss of smell, or taste, or hearing. Or there may be feelings as of a limb or other part being enormously enlarged, of the body being confined in a stiff case, of the feet being drawn up by strings under them, of 'pins and needles' around the waist, or of numbness and coldness in one half of the body (commonly the left). It may be said generally of the disorders of sensa-

tion that they are capricious in their appearance, coming and going as they would not did they depend upon organic disease (this recurrence is especially significant); that they are very apt to ensue upon some moral shock or convulsive seizure; and that careful examination will prove them to be unaccompanied by such other symptoms as would be likely to be present did they depend upon the organic alteration which they simulate.

3. *Motor*.—The principal motor symptoms in hysteria are local spasm, more or less general convulsion, and paralysis. A common symptom of hysteria is the *globus hystericus*. A lump like an egg appears to the patient to arise from the epigastrium, and, travelling upwards to the throat, causes a sensation of choking, and is often accompanied by an outburst of tears.

Spasm affecting some of the various muscles concerned in the respiratory acts gives rise to a great variety of symptoms highly characteristic of hysteria. There may be cough of a peculiarly sharp, ringing character, constant except during sleep, unaccompanied by expectoration, strongly influenced by moral causes. A little observation will show that the cough does not occur when the patient is quite alone and apparently no one within earshot; but, on the other hand, is greatly intensified by inquiry and solicitude. It ceases during sleep. Auscultation of the chest shows no departure from the normal state. Sometimes, instead of cough, a loud expiratory sound is produced, of most discordant character, resembling, perhaps, a railway whistle, the quacking of a duck, or the barking of a dog; and this may take place irregularly, or may be marked by a curiously distinct periodicity. Or there may be rapid, deep whooping inspirations, with signs of suffocation. Occasionally with the hysterical cough there is a hypersecretion of mucus; and if, as often happens, there is also disturbance of digestive functions and consequent tendency to emaciation, and at the same time such constriction of the air-passages as gives rise to sibilant râles, a *primâ facie* resemblance to phthisis is presented, which can only be distinguished by prolonged observation, aided by the stethoscope and thermometer. Laughing and crying are very frequent forms of expiratory spasm. Yawning, hiccough, and sneezing are also met with. *Clonic spasm* of muscles, especially of those moving the head and shoulders, or back, is not uncommon; or one of the muscles of the thigh may be so affected, and the apparent pulsation caused by the rhythmical contractions give rise to a suspicion of aneurysm.

Tonic spasm of one or more muscles of a limb is still more frequent. It is often very obstinate, and after enduring for months or years may suddenly resolve without any permanent alteration being left behind.

Contracture of a limb thus produced may continue during sleep, and even resist the influence of chloroform inhalation, unless this is pushed to its full extent. Should one of the abdominal muscles be thus affected, an abdominal tumour is produced, which may be mistaken for some growth in the cavity; and if the pulsations of the aorta should be communicated to it, a strong *primâ facie* resemblance to abdominal aneurysm is caused. The best mode of diagnosis is by faradisation, which, if persevered in for several minutes, will exhaust the muscular contractility and cure the ailment if it be of this kind. If it is not a 'phantom tumour' of this

description, but a genuine growth, the muscle will be contracted by the current, and it may then be possible to feel the tumour as something evidently distinct from the muscle.

Convulsive seizures are of common occurrence, and are usually preceded by a sense of suffocation, difficulty of swallowing, pain in the belly or stomach, headache, vertigo, or some indescribable sensation in one of the extremities. There is often a cry as of one being choked, unlike the peculiar wailing shriek which may usher in the epileptic seizure. Usually there is not the extreme suddenness of attack which characterises epilepsy, but the patient may be manifestly struggling against the seizure for a small but appreciable interval. When at last she falls, she does not usually do so with violence enough to receive severe injury, and positions of danger are generally avoided. The epileptic not infrequently falls into the fire, the hysterical patient never. The spasms of muscles which succeed are often tetanic in character, and sometimes wear an aspect of design—the patient grips articles with her hands or teeth. The face may be more or less red. In epilepsy it is usually first pale and then livid.

There is often more or less complete opisthotonos, which is usually absent in epilepsy. It has been doubted whether consciousness is ever completely lost in an hysterical fit; but though for the most part perhaps it is retained during the attack, there are certainly cases in which it is to a great extent in abeyance. It is characteristic of hysteria that, however rapid and violent the contortions, the patient usually avoids inflicting any serious injury upon herself. During the attack gesticulations and language are apt to be used which may be reproachful, or marked by an amatory character as regards some bystander, such as is calculated to cause him considerable embarrassment. There may be a single convulsive seizure, terminated by a fit of weeping and the passage of a large quantity of almost colourless urine of low specific gravity. Or there may be a succession of attacks extending sometimes over several hours. The tongue is not bitten. As a rule, the hysterical patient rapidly returns to her ordinary condition after the outburst is completed, and fails to show the heaviness and tendency to deep sleep which is characteristic of epilepsy. Where there is during many hours a long-continued succession of fits with brief intervals of immunity, considerable doubt may arise as to the nature of the attacks, because a very similar, numerous, and rapid recurrence of fits sometimes takes place in true epilepsy. We are indebted to Charcot for the observation that while in the case of the epileptic seizures of this kind the temperature is observed to rise greatly (attaining, for instance, a height of 105° F.), no such great increase is noted when the fits are of hysterical origin—a slight elevation only occurring. The variety seen in the character of hysterical convulsions suggests that the pathological influence involves the nervous centres, sometimes more and sometimes less extensively; giving rise to various kinds and degrees of muscular movements, from those of a highly co-ordinated or quasi-voluntary kind, down to those of a simply tetanic form. These latter would appear to indicate either that control of the reflex function of the spinal cord is temporarily suspended, or that the cerebellar influence, as Hughlings Jackson suggests, is being allowed to have full play, owing to some peculiar condition of the cerebrum interfering for a time with its normal

power of antagonism. But it must be borne in mind that the hysterical patient may, like others, become epileptic, and that there is nothing on the other hand to prevent the chronic epileptic from betraying symptoms of hysteria. Indeed this is not at all infrequent in the female epileptic. Such mixed cases are often difficult of diagnosis, and it is usually only a prolonged observation which succeeds in distinguishing the nature of the condition. Movements which somewhat resemble those of chorea are occasionally met with, but their character and the surrounding circumstances usually make it easy to distinguish them.

Paralysis may affect any of the limbs in hysteria, but paraplegia is the more usual form. Hemiplegia is comparatively rare. The muscles retain their nutrition. Where the condition has existed for a considerable period there is often at first a slight loss of irritability to induced currents, but after a very few applications this becomes normal. At first, too, considerable electro-cutaneous and electro-muscular insensibility may be present. If the form of paralysis be hemiplegic, the face is not affected; if paraplegic, the sphincters are not paralysed, and although an apparent incontinence of urine sometimes occurs, there is no cystitis, and the urine does not become ammoniacal. There is never any bed-sore. If the arm be the limb affected, and the examiner, after flexing it slightly at the elbow-joint, leaves go, it will sometimes remain in the flexed position, which it would not do in a flaccid organic hemiplegia. It is noteworthy that, in half the cases of hysterical paralysis, there is no history of antecedent convulsions. Cutaneous anaesthesia of the extremities will give rise sometimes to a pseudo-paralysis, and muscular anaesthesia may cause symptoms of an ataxic character. These may be distinguished from the result of organic change by careful examination. Hysterical speechlessness may be distinguished from aphasia by the patient being able to write down with great facility the wishes she is unable to express in speech; and from localised paralysis of the tongue, by her being perfectly able to protrude the organ and to swallow.

4. *Circulatory*.—There may be syncope which will simulate dying. After an indescribable sensation at the heart—a fulness, or stifling feeling—the pulse becomes almost imperceptible, the patient is speechless, and, for periods varying in length, is apparently in a most precarious condition, recovery taking place after prolonged sighing. Or there may be tumultuous action of the heart. The abdominal aorta (and sometimes also other arteries) is occasionally the seat of powerful pulsations, which are visible in their effects upon the abdominal wall and strongly suggest the existence of aneurysm. The capillary circulation may be deranged in the two directions of hyperæmia and ischæmia. In the former there is a patch of redness of the skin, accompanied by a feeling of burning and tenderness; in the latter, which is especially seen in conjunction with analgesia, the skin is pale and no bleeding follows the pricks of a pin. In a recorded case, pressure upon a tender spinous process checked the radial pulse for a time.

5. *Visceral*.—Vomiting is sometimes a very obstinate symptom, all food taken being speedily ejected, the condition lasting a surprisingly long time, often for many months, sometimes for years, usually without so much prostration as might be expected, but nevertheless with great loss of weight.

Or there may be such an active aversion from food as renders it very difficult to support nutrition; or a depraved appetite may cause substances to be swallowed which have no nutritious property. In the belly there is frequently a hyper-secretion of gas with spasm of the bowels, causing borborygmi and noisy eructations. Intestinal gas may be imprisoned between two points of spasmodic contraction of the intestine, giving origin to a tumour capable of being moved about in the abdominal cavity, and of sudden resolution. These 'balloons,' as they have been called, are probably sometimes mistaken for tumours of the spleen, kidney, or other organ. It is not uncommon to have retention of urine, the bladder becoming greatly distended, but contracting at once and expelling its contents if the patient be placed in a hip-bath, and a bucket of cold water thrown over the pelvis. In other cases there is an unduly frequent desire to empty the bladder, and there may be some apparent incontinence. The secretion of urine may be suppressed almost entirely (but this is very rare), the little urine that is passed containing an unusual proportion of urea, which is also found in the vomiting accompanying this condition.

There is sometimes very obstinate constipation, extending over weeks or even months, and giving rise to enormous impaction of fæces—occasionally also there is diarrhoea.

Cases occur in which symptoms of cerebro-spinal sclerosis of the insular or disseminated form, especially paresis of one or other limb in turn, after persisting perhaps for many months in a young woman, rapidly or even suddenly disappear. This circumstance is often supposed to show that the case was one of hysteria. It is probable, however, that the view still generally held that a shifting of loss of power from one limb to another is really characteristic of hysteria is quite an error. The hysterical woman who has lost all power in her legs will, it is true, very often later on (while still paraplegic) lose the power of one arm (usually the left), but in the writer's experience she is not prone to lose the power in a limb, then recover it, and then lose it in another. The idea of this shifting of powerlessness being strongly suggestive of hysteria has probably arisen from the mistakes in diagnosing as hysteria cases of insular sclerosis, which must have been continually occurring before the latter disease had been differentiated. So in regard to vision. The hysterical patient will become quite blind of one eye, while the patient affected with insular sclerosis will only describe obscurity of vision in one eye which *gradually* becomes more pronounced. It is doubtful if the sight is ever entirely lost. The writer cannot call to mind, since he has been better acquainted with insular sclerosis, any case of simple hysteria in which first one eye lost some amount of vision for a time, and recovered, and afterwards the other eye behaved in a similar fashion. This is not at all uncommon (is indeed frequently the case) in the course of insular sclerosis. The marked and almost constant occurrence of hysterical symptoms in the earlier stages of insular sclerosis often tends to obscure the diagnosis, and a very guarded prognosis becomes necessary. It has been common to class such cases with those of hysterical paralysis; but it is probable that they are examples of insular sclerosis recovering only for a time, as is characteristic of the disease. Many cases of insular cerebro-spinal sclerosis are still erroneously diagnosed as hysteria. See MULTIPLE SCLEROSIS.

SEQUELÆ.—Hysterical symptoms sometimes pass into those of mania, melancholia, and occasionally also of dementia.

PROGNOSIS.—This is favourable as regards life, death from hysteria being very rare. Recovery for a time is common enough, but too often there is a return of the disease, the symptoms being usually of a different kind. Some patients will run through almost every conceivable phase of the disorder in turn. As a rule there is a tendency to cessation of the disease after the climacteric period. It occasionally happens, however, that the disease is continued into an advanced period of life.

TREATMENT.—If Medicine were in a position to regulate the mode of life, food, education, and especially the selection for propagation of the species, it is probable that in succeeding generations hysteria would become more and more rare in the race. In many cases it can do but little for the individual. Intercurrent maladies must of course receive the treatment proper to them. Where anæmia is present, much good may often be done by iron and arsenic. States of malnutrition tend to precipitate and intensify hysterical symptoms; and to remedy these is often to do much for the concomitant nervous disorder.

But probably the greatest amount of benefit which can be brought to bear upon the hysterical patient is through her surroundings. A girl who has not spoken above a whisper for months while at home, will often recover her natural tone of voice in a week if placed under the judicious discipline of strangers. This is a well-known circumstance, and the fact has tended very much to the belief that hysteria is simply vicious simulation. Such an inference is unjust.

That an altered relation of the ganglionic nerve-cells to the blood-supply forms at least a part of the pathology of hysteria appears probable from the effects of fasting in provoking hysterical outbursts, and the influence of food and stimulants in postponing them. Ammonia inhaled by the nostrils is a well-known and valuable agent for the purpose. Alcohol should be avoided altogether, as there is great danger of excess.

It is through the sensory nerves that the most rapid influence is brought to bear upon the hysterical condition. Thus cutaneous anesthesia and hyperæsthesia may often be rapidly cured by the application of strong induced currents to the affected portion of skin; aphonia by acting in a similar way upon the skin covering the larynx. Paralysis of the limbs is in many cases quickly cured by the same means.

Hysterical convulsions may almost always be cut short by douching the patient very freely indeed with cold water. This should be poured from a height upon the face. For a few seconds there is no perceptible effect, then the breathing becomes gasping, and the patient seeks by moving away to avoid any further application. It often happens that the remembrance of this treatment serves to prevent a repetition of convulsions, but it would be wrong to conclude from this that the proceedings of the patient had been voluntary. The effect of the cold douche is to create, through the medium of the cutaneous nerves, a sudden change in the character of the blood-circulation, which may well influence the state of the ganglionic nerve-centres. The supposition seems fair that to remember the shock is to have a weak excitement of the nerve-

centres which were strongly excited by the application.

Bromide of potassium, which is of such value in the *grand mal* of epilepsy, has no influence in preventing hysterical convulsions. In a doubtful case the exhibition of this drug is therefore useful for purposes of diagnosis. Valerian (the powder or tincture) has an unquestionable effect in the convulsive and spasmodic symptoms of hysteria, little or none probably upon the paralytic phases. Asafetida by enema is useful in tympanites and colic of hysterical origin. Small doses of strychnine and opium are useful in relieving some of the distressing feelings complained of by hysterical patients.

Dr. Weir Mitchell, of Philadelphia, has introduced a mode of systematic treatment of hysteria and nerve-prostration, which has lent important aid to our means of dealing with cases of this kind. The treatment essentially consists in the complete isolation of the patient, who lies in bed and is fed and attended by an intelligent nurse. Under the influence of massage of the muscles of the trunk and extremities, conducted twice a day for an hour or more, the patient becomes able to take large quantities of milk (from 60 to 100 oz. in twenty-four hours), besides three full meals of highly nutritious food. By these means the nutrition of the body undergoes a remarkable improvement, one or two stone of weight being often added; the muscles become firm, the skin soft and elastic; the complexion assumes the hue of health; the patient regains natural sleep if this has been lost; while various morbid symptoms—pains, local tenderness, contracture, loss of power in the limbs—disappear. It is especially in cases where signs of emaciation have been present that this mode of treatment may achieve extraordinary success.

It is often a question whether the hysterical should marry. Where the disorder is slight and the general health is good, marriage may be advised, supposing that the prospects of a happy union are favourable. But in very severe cases, and especially when there is also a strong neurotic history in the family, it should be discountenanced. Nothing but harm can be expected from the strain of domestic cares upon a congenitally defective nervous system.

T. BUZZARD.

HYSTERIA, TOXIC.—See LEAD, Poisoning by.

HYSTERICAL INSANITY.—Almost every variety of insanity may present in certain patients features which are commonly known and termed 'hysterical.' Melancholic individuals will be afflicted with hysterical paraplegia or other paralyses. Some will become cataleptic or apparently unconscious. Others will display all the phenomena of hystero-epilepsy. Not infrequently do we see a violent outburst of acute mania culminating and subsiding in a brief period of time, resembling in this an ordinary attack of 'hysterics.' It may be doubtful, however, whether hysterical insanity should be looked upon as a special variety of the malady. It seems more correct to look upon it as insanity occurring in hysterical patients, and characterised by the phenomena peculiar to them. We may expect sudden changes of symptoms, sudden improvements, and sudden relapses.

PROGNOSIS.—The prognosis is unfavourable, as this form of insanity is found in patients of an unstable nervous organisation, prone to frequent derangement. Even if recovery takes place, attacks are not unlikely to occur subsequently.

TREATMENT.—Such persons above all others require moral treatment. Medical treatment should be directed towards the improvement of the general health rather than the removal of special symptoms.

G. F. BLANDFORD.

HYSTERO-EPILEPSY.—SYNON.: Fr. *Hystéro-épilepsie*; *Hystérie Epileptiforme*; *La Grande Hystérie*; Ger. *Hystero-epilepsie*.

DEFINITION.—This term has been so loosely applied that it is incapable of strict definition. It is to the French school, and especially to the late Professor Charcot of Paris, that we owe its application to cases of the convulsive form of hysteria displaying an amount of severity such as is rarely witnessed in this country. The term is sometimes allowed to cover any or all of the more marked symptoms of hysteria, whether sensory, motor, or visceral. In England its etymology has led to its being applied to a class of cases, essentially epileptic in origin, but characterised by convulsions and other motor phenomena of a 'purposive' aspect. Under these circumstances it is only necessary to refer to the articles on EPILEPSY and HYSTERIA for further information.

T. BUZZARD.

I

ICE, Therapeutic use of.—See COLD, Therapeutic use of.

ICHTHYOSIS (ἰχθῦς, a fish-scale).—SYNON.: Fish-skin Disease; Fr. *Ichthyose*; Ger. *Fischschuppenausschlag*.

DEFINITION.—A disease which has obtained its name from the division of the cuticle into polygonal plates somewhat like the scales of the fish, although no overlapping exists. The skin is dry, rigid, rough, and greyish-green, often of the hue of the upper surface of the turbot; and the cuticle exfoliates, in some places as dust, in others as thin shining laminæ like mica or bran.

ÆTIOLOGY.—Ichthyosis may be regarded as a defective development of the skin, usually, however, appearing some months after birth. In rare instances it has arisen in adults. Males are affected twenty times as often as females. Heredity sometimes obtains. The degree of its manifestation may depend on various circumstances, especially those relating to scanty food and want of cleanliness. It may be regarded as endemic in the Molucca Islands.

ANATOMICAL CHARACTERS.—The cuticle is abnormally copious; the fibrous tissue of the derma is condensed; the papillæ are enlarged and lengthened, and are sometimes apt to bleed on

slight injury; the areolar layer is lax and fatless, and the whole integument wants succulence and elasticity. Further, the cuticle is hard and brittle,—the inorganic matter, mainly phosphates and silica, being increased fourfold—and cracks along the lines of motion or wrinkles of the skin. The fragments are powdery upon the neck, the front of the trunk, and the flexures of the joints, angular and prominent on their extensor aspects, and smooth and polyhedral on the rest of the limbs. The follicles are filled with dry epithelium and sebum, which in places may concrete and add to the thickness of the crust. The skin as a whole forms coarse wrinkles, and, from the laxness of the subcutaneous areolar tissue, moves freely over the fascia. The oily, and sometimes the aqueous, secretions are scanty; an unpleasant odour is often exhaled; and the transparency and lustre of the healthy skin are wanting. The hair is coarse and brittle. The health of the patient is usually good, but cardiac hypertrophy and asthma have been noted. The cold of winter is felt severely, and death often results from pulmonary complaints.

MODIFICATIONS.—Ichthyosis varies according to site. On the limbs it is most symmetrical, and the scales are largest; on the hands and feet the cuticle is horny, and there are deep wrinkles; and on the face the detached edges of the plates cause great roughness, and the complexion is altered to a brick-red. Varieties are also produced by the amount of sebum, which may form either thick scales or projecting spines. Such modified forms have suggested several synonyms. Thus, when dryness of the skin is conspicuous it has been termed *xerodermia*—but this may progress to a more severe form. When the network of lines bounding the scales is regular and widely stretched, from the tightness of the whole integument, the name 'harlequin skin' has been applied, such instances being usually congenital and the birth premature. When the smooth surface has a mother-of-pearl polish it has been called *ichthyosis naecea*. When the concreted epidermic and sebaceous substances resemble the scales of reptiles, the term *ichthyosis serpentina* is applied, although the monitor is the reptile whose skin it is most like; and finally, the variety in which long spines appear is designated *ichthyosis hystrix*, the 'porcupine disease.' Malformations of the eyelids, ears, and fingers have been frequently found in ichthyotic subjects. The disease has been known to disappear after eruptive fevers, and after mercurial salivation. Palmar and plantar keratosis is closely allied, but it has no relation to Darier's disease, which has been proved to be microbic. See p. 362.

TREATMENT.—The principles of treatment are as follows: First, we must promote an improved nutrition of the body by the use of a generous diet, including fresh fruits, and excluding oatmeal and wholemeal. Cod-liver oil, arsenic, iron, or other tonics are indicated. Secondly, it is necessary to remove the excess of epidermic matter and sordes, which is best effected, when the disease is limited, by resorcin or salicylic acid; the mixture of salicylic acid, tincture of Indian hemp, and flexible collodion sold as 'corn-solvent' is useful. When the disease is extensive, ablutions with soft water and soft soap, and especially the Turkish bath and shampooing, are beneficial. Thirdly, we have to stimulate the circulation and innervation of the skin by friction and inunction with such substances as lanoline, cod-liver oil, or cacao-butter mixed with

glycerine. The salve-mulls and super-fatted soaps, as used by Unna, give great relief in localised cases. Injection of pilocarpine has been used to increase sweating and thus soften the cuticle.

E. D. MAPOTHER.

ICTERUS (*ικτῆρ*, a weasel; with yellow eyes). A synonym for jaundice. See JAUNDICE.

ICTUS SOLIS (*ictus*, a stroke; *sol*, the sun). A synonym for sunstroke. See SUNSTROKE.

IDAHO HOT SPRINGS, in Clear Creek County, Colorado, U.S.A.—Thermal waters. See MINERAL WATERS.

IDIOCY (*ιδιώτης*, a private individual, a person holding no public position; hence a layman or unskilled person, an ignorant clumsy fellow).—**SYNON.**: Imbecility, Feeble-mindedness; **Fr.** *Idiotie, Idiotisme, Démence innée*; **Ger.** *Blödsinn, Idiotie, Idiotismus, Narrheit*.

DEFINITION.—Mental deficiency occurring during infancy and the early periods of life and dependent upon imperfect development of the brain, whether due to transmitted qualities, injury, malnutrition, or disease.

NOMENCLATURE.—Popular usage associates the name 'idiot' with the pronounced and more degraded types, while the term 'imbecile' has come to be used of those cases above the lowest grade, and 'feeble-minded' of a higher level still, though each of these terms has been and is still used by some, and especially by American writers, of the whole group. Legally, idiot and imbecile are used as synonymous terms in the Idiots Act 1886, as opposed to lunatic, though they are included under the term 'lunatic' in the Lunacy Act 1890.

The term 'feeble-minded' does not appear on the Statute Book, but the Elementary Education (Defective and Epileptic Children) Act 1899 provides for children who, not being imbecile and not being merely dull or backward, are defective, that is to say, children who, by reason of mental or physical defect, are incapable of receiving proper benefit from the instruction in the ordinary public elementary schools, but not in special classes or schools for such children. The mentally dull and backward children owe their condition to a vicious environment after birth in respect to nutrition (short of disease), home care, and education, and do not come under our definition.

DESCRIPTION.—It may be premised that we have to deal with a great variety of cases traceable to many causes, one outcome of which has been that the centres in the brain which form the substratum of mind have suffered during their development. Hence arises a defect in mental capacity, ranging in extent from a slight departure from the normal to that condition of profound *amentia*, in which only the organic functions persist and the unfortunate subject perceives nothing, knows nothing, does nothing.

The cardinal symptom is the mental condition; as a rule the whole range of conscious states is impaired, but in some cases the intellectual power, in others the moral sense, is more particularly affected. Owing, however, to the intimate relation of the nerve-centres to each other and the close dependence of the body on the nervous system, both as regards function and nutrition, we find that the mental defect is commonly accompanied by profound physical

alterations. As it is impossible to construct a type that shall satisfactorily represent such varied conditions, the following statement of the more striking abnormalities found in different cases, either singly or combined, will serve for a general description.

The height and weight of mentally defective children are less than normal, but the difference is less on the average in girls than in boys. The skin is often coarse, deficient in elasticity and tone, and shows thickening of the subcutaneous tissue. In some cases it is dry and branny, in others very greasy from excessive secretion of sebum; in others, again, perspiration is excessive at the expense of the secretion of urine. Molluscous appendages are very common, and extreme obesity is occasionally seen. A general and striking character is met with in the teeth, which are often incomplete in number, poorly developed, much coated with tartar, and loose, very often crowded and irregular. The eruption of the teeth is much delayed, and the gums frequently call for attention. The muscular system, in addition to the paralyses and contractures due to specific nerve-lesions, commonly shows a general lack of tone due to deficient cerebral activity, though the reverse condition is sometimes seen owing to general loss of control. The record written in the bones is permanent and more explicit, and is often a clue to the period of onset and the nature of the case. They are sometimes very brittle, and the osseous deformities are very striking. Those of the head have naturally attracted most attention. It is now generally agreed that the natural growth of the skull depends upon the healthy development of the brain, and that cerebral conditions are more likely to cause than be caused by the malformations seen in the skull. Among these are heads too small, too large, or asymmetrical; skulls short from back to front, round and high; with overhanging or retreating forehead, or very small in the occipital region. Frequent and significant among the anomalies are strongly marked ridges in the lines of the sutures—especially in the interfrontal, which produces a ‘prow-shaped’ cranium; and in the sagittal when the skull is called ‘scaphocephalic’ from the likeness of the ridge to a boat’s keel; this is specially marked when, as sometimes happens, the head is also flattened on either side of the ridge. A similar ridge is also found over the coronal suture. This condition appears to depend on delayed ossification; while the occasional obliteration of the sutures points to its premature completion. On the other hand the lines of the sutures may be marked by shallow furrows, while the centres of ossification are the site of pronounced bosses. In cases with hydrocephalus the usual cranial conditions are found (see HYDROCEPHALUS). A general enlargement of the cranium, especially in the vertical direction, is associated with hypertrophy of the brain, and the extreme cases of microcephaly, with receding forehead and a circumference of 17 inches or less, usually present a brain cortex of extreme simplicity as regards convolutions, or a porencephalous condition.

Side by side with these cranial anomalies we find malformations of the bones of the face, and more especially of the jaws. These so-called ‘developmental neuroses’ appear to depend upon errors of brain-development occurring early in intra-uterine life. The chief points are general smallness of the face, narrow and high palate, V-shaped or saddle-shaped dental arch, enlarged alveolar processes,

receding lower jaw, orbits very close together or very wide apart, and facial asymmetry.

The circulation is generally feeble, rendering the patient liable to chilblains, and the processes of repair slow. The temperature of the body is commonly subnormal. The digestive system is very liable to be deranged by defective mastication, and great care is necessary in the preparation of the food and in the exclusion of unsuitable articles of diet. Constipation readily occurs from defective innervation, and changes of temperature and errors of diet readily produce diarrhoea. Congenital hernia is common. The respiratory movements are wanting in vigour and are often not under voluntary control; hence the assistance they afford to the circulation is very feeble, and these subjects are very liable to succumb to pulmonary affections; further their low resisting powers render them very prone to contract tuberculosis if placed in unfavourable circumstances, as on clay-soil, or in ill-ventilated rooms. Obstruction to respiration is sometimes caused by nasopharyngeal adenoids, which produce characteristic changes in the face and chest, and by disturbing the cerebral circulation or causing deafness further hamper the intelligence.

The sexual organs are often imperfectly developed, phimosi, undescended testis, and undeveloped ovary being common; puberty is generally delayed. The ears are often defective in their conformation, and their situation abnormal or asymmetrical.

While the presence of one or other of these morphological faults is commonly found, this is not always the case; on the other hand, many of them are met with in mentally normal individuals.

Turning now to the nervous system, we find that examples of disordered function are very common and highly significant. Generally speaking, there is a want of tone and vigour, as seen in the inability to retain the saliva and in the difficulty some patients have in standing. All reactions tend to be slow and movements slovenly, and there is often some inco-ordination with a loss of finer movements. Thus those who are able to write rarely hold the pen in a facile manner; walking is generally acquired late, and there is difficulty in following objects by moving the eyes only. The art of cutting up and conveying food to the mouth is learnt slowly and with difficulty; this is also true of putting on and fastening the clothes, and performing ablutions.

In some patients movement is almost wanting, in others it is continual and sometimes rapid. Prominent among the abnormal movements are those due to epilepsy, and the ataxia, tremor, and athetosis associated with cerebro-spinal degenerations.

In addition to these a number of striking automatic and impulsive movements are met with, such as swaying to and fro, spinning round and round, walking on the toes, twiddling the fingers in front of and near the eyes, grinding the teeth, beating the head and face, and making grimaces and rhythmic noises. These are characteristic of low-grade cases. Right-handedness is far less universal than among normal persons. The name ‘microkinesis’ has been applied to small slow rhythmic movements often seen in fingers. The control of bladder and rectum is often defective, the fault lying, as a rule, with the higher centres in the brain.

Speech is very commonly affected, and is often

absent or nearly so. The most usual defects are a dysarthria and a lack of musical quality and proper pitch, with perhaps a 'sing-song' rhythm. It is far more usual for these conditions to depend upon mal-development of the higher brain-centres representing the speech-organs than upon any malformation of the organs themselves, which, unless very marked, rarely causes trouble in cases where the higher centres are healthy. In some instances the absence of speech probably depends upon a condition of the highest centres which precludes the formation of ideas, and so the need for language; even gestures may then be wanting. Speech is usually acquired very late, and often remains infantile in character or merely automatic or echolalic. At the sudden mental breakdown which marks the onset of some cases speech may be lost completely or nearly so, and when the mental impairment is severe the power of speech is rarely re-acquired. Stuttering and stammering occur in all their usual forms among the higher grades of idiocy, rarely in the lower. The presence or absence of speech cannot be taken as a measure of the intelligence. Its absence is a serious bar to progress; and this is especially the case when the difficulty lies in the central processes by which words are perceived as having meaning. A high degree of intelligence is compatible with simple motor or sensory aphasia. Deaf-mutism is rarely found combined with idiocy, and when so found cannot be dealt with by the oral method of teaching. See DUMBNESS.

Vision is very often impaired by such defects of the eye as hypermetropia, irregular astigmatism, congenital cataract, strabismus, and nystagmus; and visual perception also seems to be blunted in many by reason of central nervous defects. A large proportion are born blind, a few become so in the course of development. The sense of taste is often defective, sometimes perverted so that repulsive things are eaten. Medicines are as a rule readily administered. Smell is in a few cases extremely acute, though usually it is below normal. Hearing, when affected, is generally so from central causes, as seen in cases combined with athetosis, or from naso-pharyngeal obstruction. General tactile sensibility seems to be obtuse, or to be acute in unusual places only, as the head or tongue. This accounts for the tendency to utilise the tips of the fingers much less than the normal child does, and the clumsy manner of holding a pen or a needle. Some delight in gentle stimuli, others in head-beating. Pain is often borne with wonderful callousness or is not felt as pain. Thus one sees analgesia to the knife in opening an abscess or to a burn; in other cases there is hyperæsthesia, or extreme irritability, so that gravel may be rubbed on the skin to give relief till it is quite raw. Even the organic reflexes seem to be sluggish, as shown by the frequent absence of cough in phthisis, and its general ineffectiveness in pulmonary affections. The vaso-motor system also reacts slowly to external changes, rendering the patient liable to take cold readily.

Whatever the accompanying anomalies of structure, nutrition, or sensori-motor reaction, the essential factor is the mental condition as directly expressed in language, habits, instincts, conduct, attention, volition, and the emotions. It is by the meaning rather than the manner of the movements by which the mind expresses itself that any case must be ultimately gauged. Some of these points will now be considered.

Perhaps the best index to the general mental aptitude is the intensity and persistence of the attention, and for educational purposes this forms the most useful basis of classification. Where sense-impressions fail to arouse percepts or even reflexes, attention is extremely low or absent, and instruction is impossible; where perception is present but unaccompanied by intelligent interest and pleasure it is still low, and renders training slow and difficult. A well-developed voluntary attention to either external conditions or existing ideas is hardly ever found. On the other hand, the attention may be preoccupied with organic sensations, or concentrated on a narrow intellectual avenue at the expense of all others. The rousing of a healthy attention by repeated stimuli likely to interest is the basis of training (see CHILDREN, TRAINING OF). It is owing to inefficient attention that spontaneous play is so rarely found; this trait leads the normal child to leave the idiotic severely alone, and the theoretical advantage claimed to be derived from association with ordinary children is generally found in practice to be non-existent or even a negative quantity. Imitation is common in its lower reflex forms, less so as a spontaneous well-directed function. Memory is proportionate to the strength and duration of sense-impressions and the attention to them. Where special interest is aroused and attention developed special memory-aptitudes are acquired, often greatly in excess of the general level of mental capacity. A similar one-sided skill may be acquired for manual work, drawing, music, or calculation, and extreme acuteness of vision has been cultivated by the habit of looking for and picking up every shining particle seen on the floor.

Pleasure is very generally aroused in these cases by music; this does not as a rule indicate any special æsthetic sense; it is sounds and rhythm which attract, even though discordant, jarring, and monotonous. Simple airs are readily learnt and reproduced by many, and sometimes there is exceptional power in this direction. Some defective children take a delight in long and uncommon words and great precision of language, others are extremely loquacious; but as a rule the vocabulary is limited to emotional expressions and the most ordinary requirements of concrete daily life. The practice of speaking of the 'self' in the third instead of the first person is very usual. Excessive eating is common, though some would starve if the food were not placed in their mouths. The sexual instinct is feeble, sometimes wanting, perverted, or exaggerated.

Owing to the rudimentary nature of the ideas of which they are capable, the mentally deficient, with few exceptions, live entirely in the present, have no ambitions, regrets, or anxieties, suffer little pain at the loss of near relatives, and are untroubled by their own condition if they recognise it. They have, as a rule, no true religious sentiment, and their ideas about the unseen world are of the most primitive and crude nature. They are easily led, and tend to lean upon others, and they readily make themselves at home in fresh surroundings. They have few social bonds among themselves. They are, as a rule, frank, docile, affectionate, and timid, but in a few cases sullen, spiteful, deceitful, obstinate, or passionate.

Classification.—In the classification of a symptomatic group such as this, it is difficult to escape a cross-division. Thus classes have been made based

partly on anatomical conditions, partly on the supposed cause, mode of onset, and leading symptom; the whole group has been divided into apathetic and excitable idiots from the point of view of the emotions or temperament, and the powers of attention and speech have been suggested as a basis for grouping the various grades. The most generally useful plan is to take a biological basis for the main divisions and to group the cases under these in accordance with such ætiological and pathological factors as we are able to ascertain. All cases may be regarded as originating either in a transmitted tendency already present in the fertilised ovum or in a defect acquired by it subsequent to fertilisation. The transmitted tendency may be realised or the defect acquired either before the onset of labour when the case is *congenital*, or later when it may be called *developmental*. Of the acquired cases some are *accidental*, that is, due to conditions of an adventitious nature occurring somewhat suddenly; the rest may be described broadly as *nutritional*, that is, dependent on causes of a more intrinsic nature and affecting the nervous functions in a more gradual manner.

For convenience of description, the following types have been distinguished: Microcephalic; Hydrocephalic; Hypertrophic; Paralytic; Epileptic; Eclamptic; Toxic; Traumatic; Post-Febrile or Inflammatory; Syphilitic; Athetotic; Mongolian; Cretinous (*a*) Sporadic, (*b*) Endemic.

The majority of cases are congenital, and as a rule present some of the physical stigmata already described; the developmental minority may manifest an average intelligence through infancy, and even up to puberty, but break down mentally generally during one of the critical developmental epochs, birth, first and second dentition, and puberty. Speech may be lost for a time or permanently, and there is definite retrogression in mental power. They may possess the crested skull, deformed palate, &c. The accidental cases are the fewest in number: these individuals may be quite free from physical defect and brilliant mentally, and then the intellectual functions may be suddenly completely destroyed without hope of recovery, as, for example, by an attack of meningitis or other injury to the brain.

The *Mongolian Type* forms so well-marked and distinct a variety that it deserves special description. The growth is always stunted, the head and face round and small, with a small retreating lower jaw and an absence of prominent features; the skin is dry, coarse, and readily irritated; the hair is sparse and straight, and is liable to alopecia. The ears are small and closely set, the hands short, broad and spade-like, the nails and teeth ill-nourished, the opening for the eyes too small and obliquely set downwards and inwards, giving the almond-eyed appearance. It is further diminished by a fold of skin over the internal canthus, the epicanthic fold. The tongue is long and fissured transversely, and the lips also are fissured. The children tend to adopt the tailor's posture when sitting. Speech is defective, stuttering and indistinct. They are mimics, obstinate and fairly educable, but show widely differing degrees of intelligence. They are often the late-born children in a family or come of phthisical stock, and have been described as 'unfinished children.' They do not respond to thyroid-feeding as cretins do.

Cretinism is myxœdema occurring in early life and

dependent on absence of the thyroid gland. The mental condition is thus secondary. Cretins show great stunting and deformity, marked myxœdematous thickening of the subcutaneous tissue, a pendulous abdomen, supraclavicular tumours, and a muddy complexion. They respond rapidly to thyroid-extract both mentally and physically. Sporadic cretins are very rare as compared with Mongols, and do not amount to one per cent. of all cases.

ÆTIOLOGY.—In the production of idiocy two or more factors may be working together to the same end, either of which alone might have been insufficient. It thus becomes difficult sometimes to determine the principal or even any cause. In few families is the history quite free from suspicion of taint, and so attention may be directed to this where the influence of maternal or other conditions, such as masturbation, is more directly responsible. On the other hand an unstable nervous inheritance may not be recognised and the blame ascribed to a harmless fall.

The following are the most important conditions responsible for mental defect:

A. TRANSMITTED: Phthisis; Insanity (including Feeble-Mindedness); Epilepsy; Neuroses such as Asthma, Deafness, Chorea, Nervousness, &c.; Constitutional Debility.

B. ACQUIRED: (*a*) *Nutritional*.—Maternal Ill-health, e.g. Vomiting, Uterine Hæmorrhage, &c.; Maternal Nervous Shock or Strong Mental Impression; Maternal Intellectual Overstrain; Maternal Accident; Maternal Intemperance; Maternal Syphilis (unimportant); Premature Birth; Sporadic and Endemic Cretinism.

(*b*) *Accidental*.—Prolonged Labour with Suspended Animation; Injury during Parturition; Eclampsia; Insolation; Cranial Injury (rare); Severe Mental Shock; Cerebral Hæmorrhage and Sclerosis; Hydrocephalus; Inflammation of Brain or Meninges; Specific Fevers; Drugs (rare).

Epilepsy is present in about 20 per cent. of the cases. The Mongolian type accounts for about 10 per cent., and these children are rarely epileptic. From 5 per cent. to 10 per cent. are probably to be ascribed to one of the causes classed as accidental. Of the remainder the great majority are congenital, and include a considerable number of children born prematurely. Syphilis is of little importance as a cause, and far more cases are produced by prolonged pressure during delivery with suspended animation than by injury from the use of the forceps, which, on the contrary, helps to prevent these conditions. Twenty-five per cent. of mentally defective children are first-born, and this does not seem to be due to the smallness of the families. The children born at the end of a long family also seem to be frequently affected, but twins are not specially liable.

As regards the influence of the intermarriage or blood-relations there is nothing to show that in-breeding is in itself harmful; it is so only when the stock shows a taint which would be accentuated by in-breeding, and perhaps eradicated by crossing with another and healthy strain. Even crossed unions are exposed to similar risks, and idiocy will appear in the progeny of apparently the most unblemished families. The number of cases which are the offspring of consanguineous marriages is about 7 per cent.

From this review it would appear that the prevention of idiocy must depend very largely upon guarding as far as possible against all conditions

likely to affect injuriously the nutrition of the embryo and child, upon precluding absolutely the direct transmission of mental defects by idiots becoming parents, and upon the avoidance of marriages where there are neuroses on both sides likely to lead to a degenerate offspring.

PROGNOSIS.—Broadly speaking the developmental cases are more disappointing than the congenital, while the accidental cases are the most unsatisfactory of all. This largely depends on the natural tendency to expect more progress where the child presents regular features, a fine complexion, and a well-developed frame; whereas many of the congenital cases, though ungainly in movements and of unattractive appearance, are far more susceptible of improvement. The damage to the brain in the accidental group may leave total mental shipwreck in a body possessed of fine physical endowments. Epilepsy is a great bar to educational progress, and the paralysed cases are often very hopeless. The prospect of life is less than in normal children, especially in the epileptic and Mongolian groups. In the congenital cases the actual mental capacity must be our guide as to the future, especially the power and extent of speech and the character of the attention. Great and lasting improvement, both physical and mental, can be looked for in Cretinism so long as thyroid-feeding is maintained.

PATHOLOGY.—The following are some of the pathological conditions found in the brain: (1) hypertrophy; (2) excessive firmness of the brain-substance; (3) hydrocephalus; (4) porencephaly; (5) sclerosis following hæmorrhage; (6) abnormal simplicity of the convolutions; (7) absence of important parts; (8) old inflammatory conditions; and (9) absence or degeneration of the nerve-cells and fibres.

TREATMENT.—The treatment of idiocy consists of a judicious combination of medical, physical, intellectual, and moral agencies. It is necessary to remember that underlying the mental processes is the brain-action on which they depend. The function of the brain is closely dependent on the proper performance of the nutritive processes, which must therefore be given as free play as possible. This means that the patient must be kept in the highest possible state of health by avoiding errors of hygiene, such as cold, ill-ventilated rooms, clay-soil; and by care that the dietary is liberal, nutritious, simple, and easily digested, the meals being at suitable intervals. Regular habits of life should be cultivated, and any disorders which may occur should be corrected by appropriate medical remedies. Thus, circumcision is often desirable, and adenoid vegetations obstructing the naso-pharynx may need removal. The teeth and eyes call for special attention. Sporadic cretinism is rapidly improved by the continued administration of thyroid-preparations; once a week will often suffice in the later stages. Epilepsy demands treatment by special attention to the food and its digestion, by keeping the bowels regular (the use of an enema being sometimes necessary and most valuable), by medicinal treatment which is found suitable to the case (the bromides being as a rule most useful, though at times harmful), and by general measures for safeguarding the subject during the fits, such as a readily opened shirt, pillows stuffed with a material giving easy access to the air, &c. A regular and full allowance of sleep, and as much open-air life as possible, must be secured. Care should be taken that damp

shoes and clothes are promptly changed. The early signs of illness must be quickly observed, such as loss of appetite and abnormal body-temperature. Tonics are occasionally required, and the syrup of the hypophosphites is useful for this purpose. Alcohol is as a rule undesirable; milk should be freely used.

Medicines should not be given in food. Cleanliness should be scrupulously attended to, and daily baths are often desirable; they can rarely be given quite cold. Night-socks are often necessary in winter. In some cases regular general massage, frequent feeding, and rest in bed are necessary to promote nutrition which is suffering from restlessness and excitement. Massage is often of value in cultivating the usefulness of muscles which are too little employed. Orthopædic instruments are needed in many cases of deformity, surgical procedures being used with caution owing to the difficulties of the after-treatment. Suitable exercises and a mid-day rest on a backboard should be recommended when there is tendency to curvature of the spine, and a poroplastic jacket may also be necessary. Operative measures on the skull in the hope of relieving pressure on the brain are not to be recommended, as experience shows that they are of no use.

Having secured as far as possible the general well-being of the body, we have to devote our attention to the functions of the nervous system, the higher levels of which form the substratum of mental action. We proceed from the purely physical sensorimotor reactions upwards, from simple sense-stimuli to compounded sensations; from simple reflex automatic and rhythmic movements to co-ordinated, varied, and adapted movements; from large, coarse reactions to smaller and more delicate. This is done by choosing simple stimuli, and by their frequent repetition, such as those of touch, colour, shape, weight, or heat, by graduated movement-exercises and by simple forms of play. In this way it is sought to rouse the attention and fix it by interest. Thus by slow stages the pupil may be taught as a lesson to dress and undress himself, to use spoon, knife, and fork, to tie and button his clothes. It may be necessary to teach walking with a 'go-cart.' By degrees impressions from being merely felt come to be perceived, and perceptions become united to form ideas. Here, again, the progress is from simple to complex, from concrete to abstract. At this stage the methods of the Kindergarten become useful. Ideas of number and arithmetic are a great stumbling-block, though reading and writing are frequently acquired. Imitative drawing can be taught, but drawing as a mode of expression or as an art is almost unknown. Even in a low state of intelligence simple moral appreciations or habits are formed, and wise guidance is here very important, especially in the early periods. The child must not be spoiled, but should be disciplined to obey, and wrong-doing should always lead to some loss of pleasure. Physical punishment is unnecessary and useless; the love and approval of the teacher is the greatest reward, and its withdrawal the most effectual punishment. Punishments should not be such as to interfere with hygiene, and the appropriate methods can only be learned from the study of the individual child.

Among the *matériel* and occupations useful in school at different stages are the following:—Bean-bags; balls; bricks; hammer and nails; tying-

blocks, buttoning-sleeves; beads; groups of simple objects in which colour, number, form, and quality are illustrated and associated; paper for folding, cutting, and plaiting; sand and clay for moulding; dolls for dressing; paints and brushes; coloured chalks and chequered books; scales and weights; model money and materials for the shop-lesson; string for winding, knotting, netting; woolwork and needlework; rush-plaiting, basket-making; slöjd; fretwork; carpentering; simple gardening; simple painting and printing; bagatelle, croquet, and other simple games; drill with music, bar-bells, and dumb-bells; singing, piano, dancing, and theatricals.

The educative treatment of the mentally deficient should be begun at the earliest possible moment. Experience does not show that rapid and marked improvement is to be looked for at special periods such as the seventh and fourteenth years, as is sometimes stated; rather are these to be regarded as periods of danger to the developing mind. Speaking generally, the chief desiderata are to recognise the presence of abnormal defect as early as possible, to rescue the subject from the isolation in which his difference from normal children tends to place him; to provide, if possible, the society of other children on the same mental plane, by whom he may be regarded as one of themselves, and among whom life may seem less difficult and hopeless, and success within his reach. The life should be ordered, but varied, providing at once manifold impressions and incentives to activity, and the restraints of organised society; in this way such self-dependence and self-respect as is possible will be fostered, and the highest standard of life compatible with the mental and physical conditions maintained.

R. LANGDON-DOWN.

IDIOPATHIC (*idios*, peculiar; and *páthos*, a disease).—A term sometimes applied to a morbid condition when the antecedents giving rise to it are entirely unknown.

IDIOPATHIC MUSCULAR ATROPHY.—*See* MYOPATHY.

IDIOSYNCRASY (*idios*, peculiar; *σύνγκρασις*, mixture or constitution).—In medical language this term denotes a peculiarity of reaction, exhibited by an individual, towards certain mental or physical conditions.

Mental or nervous idiosyncrasies may be seen in response to many varieties of stimuli. For example, vision may be the channel of affection. Syncope is produced in some persons by the sight of blood; or, as is related by Prochaska, swooning may invariably occur on the sight of beetroot. As regards the sense of smell, some people are distressfully affected, in both bodily and mental ways, by the exhalations from certain animals, the cat in particular; in others, horror and fainting are induced by the odour of roses or of apples. And so on through the rest of the senses.

In another group of idiosyncrasies the higher nervous centres play no part, the phenomena being of reflex production through the spinal centres, or being due to direct poisoning of the system or of organs. Among foods or drugs swallowed, among gases or dusts inhaled, among substances brought into contact with the skin, many, harmless to the majority of men, are for this or that individual irritants or poisons. For instance, eggs, honey, sugar,

or fish may produce in certain persons gastric pain, nausea, or vomiting; strawberries may act as a most virulent poison, producing symptoms of intense nervous shock; convulsive spasms may be excited by the smell of musk or civet; asthma by the inhalation of the powder of ipecacuanha; urticaria by the eating of shell-fish, or even by the application of the yolk of egg to the skin.

Idiosyncrasies exhibited with regard to particular drugs have a special interest for the medical man. They may be of a qualitative nature, as in the production of unusual symptoms with dangerous or fatal results by anæsthetics, or in iodism; or of a quantitative nature, as in the case of opium and belladonna, minute doses of which will poison some persons, while doses of them, large enough to destroy a dozen average individuals, may be taken by one here and there with impunity.

Idiosyncrasies may be set up or modified by varying conditions of life and health. Transient oddities of susceptibility may arise in pregnancy, hysteria, and madness. These only differ in their transitory character from other idiosyncrasies, and indicate temporary modifications of reactive sensibility. Intolerance of opium may arise in some morbid states the nature of which is fairly known, for example, in renal disease, which causes defective elimination of this poison; tolerance of, or comparative indifference to, the same drug may be attained by its constant use. Age, habits, and state of body may each and all modify the reaction of any individual towards his surroundings, and may change his behaviour under the influence of drugs.

Imaginary idiosyncrasies.—Persons are not infrequently met with who declare that particular foods and medicines disagree with them. It may often be found, on investigation, that the assertion is incorrect. The obstacles offered to effective treatment by such fancies are sometimes considerable, but when the idea of their existence has been proved in any case to be unfounded, it is generally possible to evade them by tact, or to dissipate them by firmness.

WILLIAM M. ORD.

IKOTA.—*See* ECSTASY.

ILEUM, Diseases of.—*See* INTESTINES, Diseases of.

ILEUS (*εἰλέω*, I twist).—A synonym for an intestinal obstruction. *See* INTESTINAL OBSTRUCTION.

ILLUSION.—A false or mistaken perception of one of the senses, as when a person sees or hears something, and takes it to be something else. Illusions may occur in the sane as well as in the insane. *See* HALLUCINATION.

IMBECILITY.—*See* DEMENTIA; and IDIOCY.

IMMUNITY (*immunis*, exempt).

DEFINITION.—Immunity to disease may be defined as a condition of the animal body, natural or acquired, which renders it resistant to the invasion of one or more infective disorders.

GENERAL CONSIDERATIONS.—An animal or man in such a condition is said to be protected against the particular disease from which it is immune. All the disorders in which the question of immunity occurs are infective, that is, are capable of being transmitted from one animal to another by means of a contagium vivum, virus, or

primary infective agent. The question of immunity is closely bound up with the study of this primary infective agent, with its morphology, its physiological life-processes, and the modes by which it produces its pathological effects. *See* BACTERIA.

The infective agent is known in the following infective disorders occurring in man : Tuberculosis, anthrax, diphtheria, glanders, tetanus, relapsing fever, erysipelas, pus-infection, pneumonia, influenza, actinomycosis, typhoid fever, cholera, gonorrhoea, plague, Malta-fever, and malaria ; and it is in connection with some of these disorders that the question of immunity has been worked out experimentally.

In other infective disorders, such as scarlet fever, measles, small-pox, syphilis, and typhus, the infective agent is unknown, so that the nature of immunity to them is at present unsolved.

The physiological life-processes of the pathogenic micro-organisms are important to the question of immunity under two headings. For their development and growth these infective agents require a suitable medium, which, in any individual disease, is formed for them or by them from the fluids of the body. In the interstitial fluids of the tissues, and in fibrin and proteids exuded from the blood in pathological effusions, these infective agents find suitable media for rapid development and growth, and in such media they produce poisonous chemical substances, which, circulating in the body, are the cause of the symptoms, both general and specific, of infective disorders.

The poisonous products which pathogenic micro-organisms form are divisible into two classes. In one the poison is the result of the breaking up of the proteid substances dissolved in the medium in which the micro-organism lives, resulting in the formation of albumoses, and, in some cases, of non-proteid substances containing nitrogen. The other class includes substances present either in the bodies of the bacillus, or excreted by them into the surrounding medium. These bodies are frequently referred to as the *toxins*. The toxins vary greatly as regards their physiological action, and their sensitiveness to external conditions. *See* TOXINS.

In connection with immunity two points have to be considered regarding the infective agent : (1) Its degree of virulence or attenuation ; and (2) the localities in which it grows in the body in natural disease ; and with these are associated its mode of distribution, and its effect in an animal after experimental inoculation into the circulation, under the skin, or into one of the cavities of the body.

1. *Degree of Virulence or Attenuation of the Infective Agent.*—Artificial cultures of pathogenic bacteria tend to lose their virulence in the majority of instances, so that a culture, even if it has once been virulent, will, after keeping, fail to kill an animal in a short time, taking three or four more days to produce a fatal result ; and this attenuation occurs, even though the bacterium preserves its cultural characteristics. During the process of loss of virulence some bacteria undergo partial degeneration, as, for example, those of cholera and diphtheria. A virulent bacterium may also be attenuated by various methods, as, for example, keeping it at a temperature of 45° C. to 50° C. for several hours. In some cases attenuation results

from the passage of a continuous current of air through the liquid in which the bacterium is growing, and in others, by the addition of antiseptics to a medium ; both carbolic acid and trichloride of iodine have been used for this purpose. A weak virus may, in some instances, be intensified or made virulent again. The chief method by which this is done is that of passing the bacterium through a series of susceptible animals, one animal being inoculated from the one just dead. After a series has been done, an extraordinary degree of virulence of the micro-organism may be obtained, as, for example, in the cases of the vibrio of cholera, of the typhoid-bacillus, the *Bacillus coli communis*, and the pneumococcus. Another method is to inject, at the same time as the bacterium, the chemical products of another micro-organism. Thus the typhoid-bacillus has been intensified by injecting a weak culture into the peritoneal cavity, and at the same time, subcutaneously, the chemical products of the streptococcus or the *Bacillus prodigiosus*. In this case it may be said that these chemical products lower the resistance of the body, and so enable the typhoid-bacillus to grow. The infective agent which produces individual cases of disease may, in some instances, be weak, and in others virulent, and it seems probable that, for maintaining the virulence of a pathogenic micro-organism, the living body is essential.

2. *Localities in which the Infective Agent occurs in the Body.*—Three classes may be distinguished :

(a) In one the infective agent distributes itself throughout the tissues of the body. Anthrax is a type of this class : as, wherever inoculated, the bacillus passes into the fluids and tissues of the body, and is there found at death. Glanders is another example. (b) In a second class the infective agent becomes limited to the seat of inoculation, or, when it enters the body, its growth results in the formation of different lesions in the tissues. Examples of this are diphtheria and tetanus, tuberculosis and pus-infection. (c) In a third class the infective agent is chiefly found in the formed elements of the body. Thus, in leprosy, the bacillus is confined almost entirely to the interior of the cells of the leprous nodule ; in malaria, the plasmodium is found inside the red blood-corpuscle.

Immunity may be : (1) Natural ; or (2) Acquired.

1. *Natural immunity.*—Natural immunity includes those cases in which man or any animal does not contract in the ordinary course of events a particular infective disorder, as well as those cases (some of which are now well known) in which it is found difficult, and, in fact, impossible, to infect an animal with a particular disease. Such cases as these are the best examples of natural immunity. The injection, for example, of a virulent infective agent into one animal will not kill it, while a similar or even smaller dose into an animal of another species will with certainty be fatal. Warm-blooded animals, therefore, including man, may, from this point of view, be divided into two classes—those which are *susceptible* to a particular infective disorder, and those which are *refractory*.

The discussion of natural immunity in man includes many other factors besides the condition of health of the tissues of the body, such as exposure to infection, susceptibility to disease according to race, sex, and age, and the influence of climate and temperature. Man is subject to certain infective diseases (e.g. typhoid fever, cholera, scarlet fever, measles,

diphtheria, &c.) which are not prevalent in animals. Animals (domestic and farm) are subject to certain diseases (e.g. swine-plague, contagious pleuropneumonia, distemper, black leg, &c.) which do not affect mankind. Lastly, there are certain infective diseases from which both mankind and animals suffer, viz. tuberculosis, anthrax, hydrophobia, glanders and farcy, pyæmia and pus-infection, tetanus and plague. In many of these diseases the primary infective agent is unknown; the question of immunity in them must therefore remain practically open. In others, however, the virus is known, and in some well studied. Special interest attaches to the question of immunity in those diseases which are natural in both man and animals, or which are natural in man and which are infective to animals by means of inoculation of the virus, although these may not suffer from them in the natural course of events. We have in such diseases opportunities for experimentally testing the conditions producing immunity. *Tuberculosis*, of which the primary infective agent is the *Bacillus tuberculosis*, is a natural disease in man, cattle, and pigs. Goats, sheep, horses, and dogs are relatively immune to it. It is difficult, although not impossible, to produce tuberculosis in dogs by the injection of the tubercular virus: positive results are obtained with very large doses injected into the peritoneal cavity. *Anthrax*, a natural disease in oxen, sheep, and in man, is observed also in pigs, goats, and horses; but experiment has shown that adult white rats, Algerian sheep, dogs, and pigeons are difficult to infect, and are in fact refractory to the disease. To *diphtheria*—a natural disease in man—rabbits, guinea-pigs, dogs, cats, monkeys, and cows are susceptible; while rats and mice are refractory, being naturally immune to the *Bacillus diphtheriæ*. To *cholera* no animal can be said to be susceptible, although the intensified *Vibrio cholerae asiaticæ* (Koch) is fatal to dogs and guinea-pigs, but is not so active in rabbits. In many instances this natural immunity, as tested by inoculation, is not an absolute quantity: it is, in fact, a quality possessed in varying degree by the several animals called refractory to a disease. Thus dogs may be regarded as practically immune to the *Bacillus tuberculosis*, even large doses inoculated subcutaneously producing no effect. To the *Bacillus tetani* they are immune to a less degree: a large dose subcutaneously will be fatal, while a small dose will produce a mild illness ending in complete recovery. Adult white rats are refractory to the *Bacillus anthracis* only in a minor degree. If a large number be inoculated with a virulent virus, less than half will die of the disease, the remainder showing only a passing illness or no illness at all. Pigeons, too, can be killed with the bacillus, although many survive and show a lesion at the site of inoculation. It is asked, What is the reason of this immunity—what physiological difference is there between the cow and the dog, that the former is very susceptible to tuberculosis, the latter eminently refractory? or between the French, Russian, or English sheep and the Algerian, or between the young rat and the adult white rat, that the former acquire anthrax and the latter not? or that a well-bred pig is susceptible to swine-erysipelas and a mongrel refractory? The investigation of these facts is the point of the question under discussion, and has led to the discovery of important facts bearing on immunity to disease.

2. *Acquired immunity*.—In man there is evidence to show that one attack of an infective disorder protects the individual against subsequent attacks; but the truth in the matter is difficult to ascertain. There is no doubt that an attack of small-pox is protective, as the results of the now obsolete inoculation showed. It is, however, an assumption that one attack in every case of an individual disease confers immunity. There is, indeed, some evidence to show that an attack of erysipelas or of tuberculosis not only does not confer immunity, but actually predisposes to subsequent attacks. But this point of view of the subject is so hedged by difficulties that little good would come from a discussion of it. It will be best, therefore, to pass on to the question of immunity acquired experimentally. The basis of most of the experiments performed is that an attack of a disease protects against a subsequent attack, and that, although the first attack may be mild, it must not be too mild, or no protection is afforded. It will not be too much to say that all recent experiments in immunity have been based on the idea of the protection afforded by vaccination against small-pox. See SMALL-POX; and VACCINATION.

The factors in immunity and in infection are three in number: (1) the degree of virulence of the infective agent; (2) the dose of the poison; and (3) the degree of resistance of the body to the invasion of the infective agent. The body has natural defences against the invasion of micro-organisms, but these means of defence are not effective against a certain (large) dose of the poison, nor with very virulent bacteria. The natural defences of the body are: (1) an unabraded and healthy skin and mucous membrane; (2) the presence of hydrochloric acid in the gastric juice, which inhibits the growth of bacteria in the stomach, and is inimical to the growth of some pathogenic organisms, for example, the *cholera-vibrio*; (3) the natural antagonistic action of certain substances present in the liquids and cells of the body.

The natural resistance of the body may be increased by the production of artificial immunity, which may be brought about in three different ways: by using an attenuated living virus; by treatment with the chemical products of the virus; or by the injection of the blood-serum of animals made immune by one or other of the first two methods. The first two methods are practically the same, inasmuch as the bacterium acts by means of its chemical products, and these two methods are frequently referred to as *preventive inoculation*.

Preventive Inoculation.—1. Immunity may be conferred by the injection of an attenuated virus, as was shown by Pasteur in the case of anthrax. The bacillus was attenuated, and two kinds of vaccine were made. The *premier vaccin* is the weaker, and is injected subcutaneously in sheep, being followed, after an interval, by the injection of the more powerful *deuxième vaccin*. After a time the animal is found immune against anthrax; that is, the inoculation of a virulent culture of the bacillus produces no disease. Similar results were obtained with the bacillus of fowl-cholera and with the attenuated cultures of the bacilli of tetanus and of diphtheria.

2. *Immunity conferred by the Chemical Products of the Virus*.—The production of immunity by the attenuated living virus is now mainly of academic interest, since it is found that the results can be

better gauged if the chemical products of the bacterium are used instead. Thus it has been found that the chemical products of the anthrax-bacillus and of the *Bacillus pyocyaneus* can confer immunity when injected into an animal. In the case of the cholera-vibrio and the typhoid-bacillus the bodies of the micro-organism are used for injection, as in these cases the poison exists chiefly in the bacterium itself. A similar procedure is pursued with plague. In the case of the bacilli of diphtheria and tetanus the toxin is excreted into the culture-medium, and may be obtained of a high degree of virulence. Gradually increasing doses injected into the horse produce a high degree of immunity. Similar results have been obtained with certain non-bacterial poisons, namely, snake-venom and two vegetable poisons, abrin and ricin, obtained respectively from the seeds of the *Abrus precatorius* and of the castor-oil plant. The repeated injection of very small doses of these poisons into animals results in the production of a very high degree of immunity against the poison.

The Blood and Tissues in Immunity.—The study of artificial immunity produced in the manner just described has brought to light two facts: (1) The immunity is specific, that is, an animal rendered immune against diphtheria is not immune against tetanus, and *vice versa*. So with the other diseases, and even with the closely allied poisons, abrin and ricin; an animal immune against the former is not immune against the latter. (2) There exist, in the blood and tissues of these artificially immune animals, substances which are antagonistic to the infective agent or its poison. These are the immunising substances, and may be found in large quantities in the blood-serum and certain of the excretions of the body. These immunising bodies may be referred to as *anti-substances* or *anti-bodies*, and are of different natures.

The chief groups of these bodies are: (1) Antitoxins and antiferments; (2) Antimicrobial substances; (3) Agglutinins; (4) Bacteriolysins.

The *antitoxins* are substances which completely counteract the effect of the toxins. Thus, the antitoxin of diphtheria, when mixed with the toxin outside the body, prevents its physiological action, and, indeed, actually combines with it. Injected into the body after the toxin, antitoxin will counteract its effect, if the interval between the injections is not too long. A previous injection of antitoxin will confer immunity against both the toxin and the bacillus. The *antimicrobial substances* protect only against the living infective agent, and not against its poison. The *agglutinins* are substances which cause the clumping together of the bacilli when these are mixed with it, and the *bacteriolysins* are bodies which cause the dissolution of the bacteria.

See ANTITOXINS.

These substances are produced in the body as the result of the reaction of the tissues to the chemical products of the bacterium. They are all specific substances, and have an action only on the special bacterium concerned, or its poison. The existence of these bodies in the blood and tissues of animals rendered artificially immune throws great light on the process of immunity, which, from this point of view, must be considered to be due to the presence in the body of substances which are antagonistic both to the bacterium and its poison. Whether natural immunity to disease is due to the presence of such antibacterial substances is a question which

is not yet settled, and the idiosyncrasies which are shown in the invasion of infective disease in various classes of animals have not been completely explained by the researches as yet performed. In some cases, where disease occurs naturally in animals and not in man, or *vice versa*, there is only a partial immunity. Although no animal suffers naturally from cholera, yet the intensified vibrio of cholera is fatal to dogs and guinea-pigs; thus, there may be immunity against the natural disease, and not an immunity against inoculation. There are, however, instances of natural inoculation-immunity. Thus dogs are highly refractory to the inoculation of tuberculosis, pigeons also are immune to the inoculation of anthrax, and mice are refractory to the diphtheria-toxin. It may, in the future, be found that specific antibacterial substances are present in such conditions, but account must be taken of the existence of phagocytosis, and though this cannot be held, as it was formerly, completely to explain the phenomenon of naturally acquired immunity, yet the fact that certain living cells of the body do ingest living bacteria, and may destroy them, is an important one in considering immunity. *See PHAGOCYTOSIS.*

Immunity and Cure of Disease.—The question of immunity is closely connected with that of the cure of infective disease. Immunity is the successful resistance of the body against the invasion of an infective disease; cure is the prevention of further progress of the disease after the symptoms have already appeared. Many cases of infective disease get well, even if no specific treatment is employed, and the natural subsidence of an infective disease may, as the result of modern research, be reasonably ascribed to the production of immunity, and more particularly to the formation of antibacterial substances, as the result of the reaction of the body against the action of the bacterial poison. That such substances are formed in the body during the course of a disease is shown by the presence of agglutinins in the blood of patients suffering from cholera or typhoid fever. Moreover, the injection of antitoxins after the disease has begun in some instances leads to a cure of the disease, that is, to a cessation of the growth of the bacterium. Thus, in diphtheria, more particularly in the early stage, the injection of the antitoxic serum stops the spread of the membrane, and so relieves the symptoms of the disease. It, moreover, neutralises in the body any of the bacterial toxin which has not yet combined with the tissues. In tetanus a similar result may be obtained, although the effect is not so marked. Whether, in cases of diphtheria treated by antitoxin, the patient survives or not, depends on the amount of toxin which has combined with the tissues previously to the injection of the antitoxin. If a fatal dose is already in combination with the tissues, no treatment by antitoxin can prevent a fatal result, although it hinders the further progress of the active disease. Cases of tetanus are not so amenable to treatment by the antitoxin, owing to the firm combination of the toxin with the tissues, and to the usually late period at which the patient comes under treatment.

SIDNEY MARTIN.

IMPERATIVE IDEAS.—*See INSANITY.*

IMPETIGINODES.—Impetiginous; that is, having the character of impetigo; hence, eczema impetiginodes. *See IMPETIGO.*

IMPETIGO.—Two diseases are described under this title: *Impetigo contagiosa* and *Impetigo herpetiformis*; but the term is generally limited to the first of these.

Impetigo Contagiosa.—**CLINICAL FEATURES.** The disease appears as slightly erythematous raised points on the skin, which rapidly become vesicular. These lesions may remain small in size, or by coalescence produce bullæ. The vesicles and bullæ are very superficial, often imperfect and covered by the upper layers of epithelium only. They at first contain almost clear serum, but rapidly become sero-purulent and rupture, and the collapsed roof of the vesicle, along with coagulated exudation, pus, and desquamated epithelium, give rise to the crust which is the characteristic feature of the disease. The crusts are irregular in shape, though more or less rounded in outline, and, unless subject to undue irritation, are surrounded by no more than the faintest margin of erythema. In the ordinary course of the lesion the crust dries in a few days, falls off, and leaves a reddened surface, which rapidly loses its pigmentation, and heals without the formation of any scar. The discharge from each patch is contagious, so that the disease tends to spread at the margin, and to give rise to new foci in the neighbourhood, until large areas of the skin may become infected. Neighbouring lymphatic glands readily enlarge and, unless care is taken, suppurate. The onset of the disease may be marked by febrile phenomena, especially in the debilitated, and in cases of extensive and rapid infection. As a rule, however, the general symptoms are very slight.

The disease is most prevalent in children, but is frequently seen in adults, especially those in charge of affected children. It appears most commonly on the parts of the body most exposed to contagion, especially the face and the scalp, but is also common on the fingers, hands, arms, and legs. From these situations the virus may be conveyed by the fingers or clothing to any part of the body. Epidemic outbreaks of the disease occasionally occur in crowded localities and schools. The disease usually runs a mild course, but in debilitated children symptoms of septic absorption may arise, and the individual lesions assume an ulcerated and gangrenous type. These cases may end fatally.

Another form, showing itself as primary pustules situated around the follicles of the skin, is known as the 'Impetigo of Bockhart.' This form of the disease is supposed to have a follicular origin, and to be characterised by a greater tendency to destruction of the skin.

PATHOLOGY.—The disease may be considered as the characteristic example of the effects of the inoculation of pyogenic organisms on a susceptible skin. In the lesions of the disease many forms of bacteria have been discovered, but the most important are the cocci of suppuration, namely the *Staphylococcus pyogenes aureus*, the *S. pyogenes albus*, and the *Streptococcus pyogenes* (see **PYOGENIC BACTERIA**). Usually more than one of these species of bacteria are present, but the organisms most commonly found are the staphylococci, and especially the *Staphylococcus aureus*. In addition to these, white cocci with mildly virulent characters of the type of the *Staphylococcus epidermidis albus* (*S. cutis communis*) are usually present, but this organism cannot yet be definitely distinguished from the *Staphylococcus pyogenes albus*. The *Bacillus pyocyaneus* and the *B. coli communis* have also

been isolated. Sabouraud and some other observers state that in the vesicular stage of impetigo, while the contained serum is clear, the *Streptococcus pyogenes* is invariably present, and no other organism; that secondary infection of the lesions by staphylococci occurs at an early stage; and that these staphylococci grow rapidly and obscure the streptococci present. The same investigators hold that the primary pustular lesions in the type of impetigo described by Bockhart contain staphylococci from the commencement, especially the *Staphylococcus aureus*. The results of these investigations are held to support the hypothesis that the ordinary type of impetigo is the immediate result of the inoculation of the *Streptococcus pyogenes*, the disease being subsequently complicated by secondary infection with staphylococci; whereas impetigo with primary pustular lesions (Bockhart) is due to the primary inoculation of staphylococci, especially the *Staphylococcus pyogenes aureus*.

In this way epidemics of the disease may be easily explained as well as varieties such as that known as 'Football Impetigo.' It is still uncertain whether these lesions can arise by the direct inoculation of organisms from the outside, whether they must be derived from individuals similarly affected, or whether the bacteria already existing in the skin may under certain conditions acquire sufficient virulence to produce the disease.

TREATMENT.—The purulent crusts must first of all be removed. This is best done by softening them with warm antiseptic washes or fomentations. In the case of the scalp, if necessary, the hair must be cut short. Any pediculi present on the body or in the clothing must be destroyed. Careful washing with soap and water may be employed, followed by the use of boric acid in the form of lotions or of fomentations. After removal of the crusts, dressings must be used so as to render the skin as aseptic as possible. One of the most efficient methods is by the use of mercurial ointments, and of these the Ung. Hydrarg. Nitrat. Dilut. is probably the best. The ointment should be used in the first instance spread on lint and applied to the surface; subsequently, as the lesions begin to heal, it may be gently rubbed into the skin. The dressings should be renewed twice a day, or as often as may be necessary to prevent the crust re-forming.

Many other forms of treatment by means of antiseptic remedies may be devised, such as by carbolic acid, perchloride of mercury, ammoniated mercury, and β -naphthol. In the case of debilitated patients, care must be taken to improve the general health by cleanliness, good hygienic conditions, and a generous diet.

Impetigo herpetiformis.—This name has been applied by Hebra and Kaposi to a condition in which groups of pustules appear on the skin, in recurrent outbreaks, attended by severe constitutional symptoms, such as rigors and high fever. Its course seems to be invariably fatal, but its pathology and aetiology are unknown. By some observers the disease is considered to be of virulently septic origin, while others have included it in the category of dermatitis herpetiformis (see **HYDROA**). This form of disease does not seem to have been observed by British dermatologists.

JAMES GALLOWAY.

IMPOTENCY (*in*, not; and *potens*, capable).—**SYNON.**: Fr. *Impuissance*; Ger. *Impotenz*.

DEFINITION.—Impotency may be defined as incapacity in the male for copulation, and is to be distinguished from sterility, that is, incapacity for procreation. Cases of impregnation without penetration have been recorded, but, as a rule, impotency involves sterility. A man may be sterile without being impotent.

Impotency may be complete or partial, permanent or temporary.

CAUSES.—Deformity, congenital or acquired, of the external genitals may produce impotency. Extreme degrees of epispadias or hypospadias, or a permanent rudimentary condition of the penis, may cause it. Elephantiasis of the penis or scrotum, a large hydrocele of the tunica vaginalis, a large irreducible scrotal hernia, or tumours, benign or malignant, of the penis may mechanically hinder penetration. Cicatrices, resulting from wounds or disease of the penis, a rigidly contracted frenum præputii, or even a permanently adherent prepuce may cause such distortion of the organ during erection as to render copulation impossible.

Usually, however, impotency is of nervous origin, but may be classified as *psychical*, *irritable*, and *paralytic*.

Psychical impotency results from the undue pre-dominance of the cerebral inhibiting centres. In newly married men, who have previously led chaste lives, it may be induced by want of self-confidence. In such cases a tonic may be prescribed, and the patient be directed to abstain from all attempts at intercourse while under treatment, but encouraging assurances will do more in effecting a cure than any sort of medical treatment. In cases where excessive venery or masturbation has been practised, there may be diminished excitability of the lumbar centre; but this condition is usually of very brief duration. Impotency may be experienced in attempted intercourse with one person and not with others.

Irritable impotency is the result of undue excitability of the nervous centres. It occurs after habitual masturbation or excessive venery. The semen is ejaculated before penetration has been effected, and the erection speedily subsides.

Paralytic impotency may be due to injury or disease of the nervous system. Diseases and injuries of the spinal cord, producing paraplegia, necessarily prevent active copulation; but if the spinal lesion be above the erection-centre, erection and connection might be possible, the man being a passive agent. Cases are reported where even impregnation has occurred in such conditions.

Some drugs are credited with producing impotence. Arsenic, when taken for a long time, has, in some instances, produced this effect, but virility is regained when the drug is discontinued. Opium-eating and excessive indulgence in tobacco have been accredited with similar effects. Diuretics, as the nitrate of potassium and bicarbonate of sodium, are well known to act as anaphrodisiacs.

Virility is more or less affected by constitutional diseases. Few complaints have greater influence in impairing the generative functions than those of the kidney. In irritative dyspepsia, with deposits in the urine of earthy phosphates or oxalate of lime, there is generally some inability. In diabetes, gout, and chronic hepatic diseases the reproductive organs are weak and often quite inactive, but may recover tone as health is restored.

Impotency sometimes occurs in middle life with-

out any obvious cause. Such persons have been observed to grow sleek and corpulent, to have a scanty beard, and to be indisposed to active muscular exertion. In general they evince no unhappiness at their altered condition. In atonic impotency, the external organs afford indications of the want of power. Not only are the testicles soft and flaccid, from the absence of blood in the vessels and sperm in the tubes, but the penis is small and shrivelled, and the glans relaxed.

TREATMENT.—In an atonic state of the organs, in which the erections are feeble, unstable, and insufficient, ten to fifteen minims of the tincture of cantharides may be given every three or four hours for a short time before the occasion arises for the exercise of the sexual functions. Diluted phosphoric acid, phosphate of iron, strychnine, and ergot of rye are remedies which may be given in impotency. The conditions to which these aphrodisiac remedies are chiefly applicable is when the intromittent organ is but feebly excited, and does not maintain the physical state necessary for penetration, during the period of congress. Such torpidity may exist in persons in whom desires are at times strongly felt, and the functions of the testicles properly performed. In these cases, also in timid persons, and in others whose organs are inexcitable from long disuse, stimulating treatment may conduce to success, and insure confidence for the future. But these remedies exert no influence in a constitutional apathy of the sexual functions. They have rarely, also, more than a temporary effect; and in persons advanced in life, when the parts, having fulfilled their office, are experiencing their natural decline, they operate injuriously, and tend to produce congestion of the prostate and local disease. In those cases in which the sexual organs are weakened or prematurely exhausted by excess, they are likewise hurtful, as well as fruitless. After such abuses a period of repose is required; and by the avoidance of all sources of excitement, and by diet and remedies adapted to invigorate the body, such as the preparations of iron, a gradual restoration of the procreative functions may be hoped for. Electricity is a remedy sometimes employed in impotency, but is rarely of much service. See STERILITY IN THE MALE.

IMPULSE (*impello*, I thrust forwards).—A sensation of a stroke communicated to the hand, for example, by the action of the heart or by the pulsation of an aneurysm; or by the sudden movement of a fluid when agitated in any way (see PHYSICAL EXAMINATION). The term is also employed in connection with a mental condition in insanity. See INSANITY, Varieties of.

IMPULSIVE INSANITY.—See INSANITY, Varieties of.

INCARCERATION (*in*, in; and *carcer*, a prison).—That condition of hernia in which it cannot be reduced, on account of obstruction at the neck of the sac or from some other cause. See HERNIA.

INCOHERENCE (*in*, not; *con*, together; and *hæreo*, I stick).—Inconsecutive or 'wandering' thought, as expressed in speech. See CONSCIOUSNESS, Disorders of.

INCOMPETENCE (*in*, not; and *competo*, I meet accurately).—In its general sense this term

signifies inability of a part to perform its functions. It is mainly applied to imperfection in the closing apparatus of an orifice, such as the valves of the heart (insufficiency) or the pylorus. See HEART, Valves and Orifices of, Diseases of.

INCOMPRESSIBLE.—Incapable of perceptibly yielding to pressure. Usually applied to the pulse. See PULSE.

INCONTINENCE (*in*, not; and *contineo*, I hold).—In medical language incontinence signifies inability to retain the urine or fæces, so that they are discharged involuntarily. See DEFÆCATION, Disorders of; and MICTURITION, Disorders of.

INCRUSTATIONS.—See CONCRETIONS.

INCUBATION (*incubo*, I hatch).—The *period of incubation* is the stage which intervenes between the reception of an infection into the organism and the first manifestation of the disease. Such an interval must occur in every infectious disorder, but the term is not usually employed except in relation to the acute specific diseases in which a more or less well defined period of incubation is observed.

A knowledge of the ordinary duration and of the extreme limits of the period of incubation may be of some assistance in diagnosis when the facts as to exposure are known, but its chief importance lies in the power which it confers of preventing the spread of disease in a community. For example, in a well-managed boarding school, the medical officer has information as to the infectious diseases from which each pupil has previously suffered; and, on the occurrence of a case of such a disease, he can ascertain with approximate accuracy the pupils with whom the patient has been in close relation, and, among these, those who have not suffered from the disease in question. Such information, though not absolutely trustworthy, is of value as indicating the zone of danger. Such 'contacts' should be kept under observation for the period of incubation of the disease, since the immediate recognition of the earliest symptoms of an attack may enable the medical officer to prevent new cases from becoming fresh foci of infection. The medical officer should also know when the disease begins to be infectious, whether, that is to say, its infection is easily conveyed during the period of incubation, and in the very earliest stages or development of the symptoms. For example, in the case of measles, which is a typical example of a specific disease infectious at the very earliest stage, the first case will probably not be recognised and isolated until the patient, in an infectious state, has been in contact with his school-fellows for at least twenty-four hours, and not improbably for a good deal longer. The number of 'contacts' will therefore in the ordinary course of school-life be considerable, and some of them will already be in the first, the second, or even the third day of incubation when the first case is discovered. On the other hand, in a disease such as scarlet fever, which is less infectious in the early stage, and in which, moreover, the sudden onset usually leads at once to a complaint of illness and incapacity for exertion, isolation can generally be effected at an early date, and the number of 'contacts' is less. The system of isolating 'contacts' has been carried out on a large scale in dealing

with plague in India, and has apparently been among the most effective of the preventive measures taken. When the patient is convalescent it becomes the duty of the medical adviser to determine when the risk of infection has ceased, and when therefore the convalescent person may be released from isolation. The answer to this question is very much complicated by the fact that infection may be retained in clothes and furniture, and that the disinfection of these is only effectual when skilfully carried out with suitable apparatus (see DISINFECTION). The pathological processes which occur in the body during the period of incubation are discussed in the articles on BACTERIA; IMMUNITY; and INFECTION.

The duration of the incubation-period is not constant, although in some diseases, as in small-pox, the variations are within very narrow limits. The circumstances which may account for these differences between individuals are variations in the resistance of the individual, the virulence of the virus, and the dose of the virus. (i.) There is little evidence that any idiosyncrasy of the individual influences the period of incubation; the general condition of health appears to determine the result of the conflict between the infective agent and the germicidal powers of the organism, but not, to any constant or considerable extent, the duration of that struggle. When an infective agent is once admitted, one of two things usually happens: either it is destroyed by the cells and fluids of the body, or it increases and produces an attack of the disease. There is some evidence, however, showing that the infective agent may occasionally remain latent for considerable periods, inhibited in its growth, but not destroyed; in such cases the incubation-period will be proportionately prolonged. Any circumstance which causes a lowering, local or general, of the resistance of the tissues may subsequently permit the growth of the infective agent. The onset of diphtheria may in this way be determined by a common cold, that of pneumonia by a blow, and, according to Broadbent, that of typhoid fever by a preceding influenza. (ii.) There is very little direct evidence concerning the effect of variations in the virulence of the virus. On the whole it seems probable in the case of typhoid fever, scarlet fever, and diphtheria that the more virulent the type of the disease the shorter will be the period of incubation. (iii.) The importance of the dose of the virus varies a good deal in different diseases, and is greatest in typhoid fever and diphtheria.

The further question whether some of the sequelæ of certain of these diseases, as, for instance, the discharge from the nose or ears after scarlatina or diphtheria complicated by rhinitis or otitis, may not continue to be infective long after the acute stage of the disease itself is past, is one that cannot be answered with confidence at the present time. In the case of scarlet fever, Simpson has suggested that these discharges, though not themselves due to a specific cause, may become the vehicle for the infective agent if the patient be retained in hospital in association with persons suffering from the disease in its acute stage. It is impossible to lay down any hard and fast rule as to the duration of the infection. Every case must be judged on its merits, having regard to all the surrounding circumstances. All that can be done is to indicate in a general way a minimum period during which a patient must be considered to remain a source of danger to susceptible

persons. This period may certainly be very much exceeded in some cases.

As a general rule the patient presents no symptoms during the period of incubation, and considers himself to be in his usual health. Some feeling of indisposition or fatigue may be experienced in cer-

tain instances, and is perhaps the rule in typhoid fever, but in diseases characterised by a sudden onset, of which scarlet fever is a good example, the patient may feel and seem perfectly well until seized with the headache, vomiting, or other symptom which is the first manifestation of the disease.

Disease.	Incubation Period.			Period of Isolation of Patient (Minimum).	Period of Observation of 'Contacts.'
	Usual.	Minimum.	Maximum.		
Cholera . .	4 to 5 days	a few hours	6 days	Until evacuations are free from the specific microbe	7 days
Dengue . .	4 days	a few hours	5 days	—	1 week
Diphtheria . .	2 days	—	7 days	For 4 weeks at least, or until sore throat (of any kind) or mucous discharge have ceased and the B. diphtheriæ is proved to be absent 'not less than 3 days after the discontinuance of local antiseptic applications'	12 days
Enteric fever . .	10 to 14 days	8 days	23 days	To end of 2nd week of convalescence. Urine must not contain B. typhosus	3 to 4 weeks
Influenza . .	3 days	1 day	5 days	Until convalescence is complete, about 10 days	6 days
Measles . .	14 days	7 days	18 days	Until desquamation and cough have ceased, or 3 weeks at least after rash	18 days if free from coryza
Mumps . .	21 days	14 days	25 days	Until all swelling has subsided, 4 weeks at least	25 days
Plague . .	3 to 5 days	12 hours	10 days	One month, or until faces and urine and discharges, if any, are free from B. pestis	14 days
Pneumonia . .	? 2 days	a few hours	6 days	—	1 week
Rubella . .	14 days	5 days	21 days	Until desquamation has ceased, 2 to 3 weeks altogether	3 weeks
Scarlet fever . .	2 days	1 day	7 days	Until desquamation has ceased and throat is well, 6 to 8 weeks	2 weeks
Typhus fever . .	12 days	—	—	3 to 4 weeks	2 weeks
Varicella . .	14 days	13 days	19 days	Until all scabs have fallen off	18 days
Variola . .	12 days	8 days	15 days	Until desquamation has ceased and all scabs have fallen off	12 days
Whooping-cough	8 days	2 days	12 days	Five weeks from beginning of whooping, or until spasmodic cough and whoop have ceased, for 2 weeks	2 to 3 weeks
Yellow fever . .	3 to 4 days	Less than 24 hours	7 days	—	8 days

DAWSON WILLIAMS.

INDIARUBBER-MAKERS.—See OCCUPATION-DISEASES.

INDICANURIA.—Two precursors of indigo are met with in urine, viz. indoxyl-sulphuric and indoxyl-glycuronic acids. The so-called urinary indican is an idoxyl-sulphate, and must be distinguished from the indican of plants, which is a glucoside. Indoxyl-glycuronic acid is a less stable substance, and by its decomposition indigo-blue is sometimes spontaneously formed, especially in alkaline urines, and is deposited as an amorphous or crystalline sediment or floats as a scum. In rare instances it has been met with as a constituent of calculi.

In normal human urine the amount of indican present is very small, but in morbid conditions the quantity is often conspicuously increased. Urines rich in indican sometimes have a deep-brown colour, which has been ascribed to higher oxidation-products of indoxyl. Suspended indigo-blue may give to the urine a bluish tint.

When the urine is boiled with hydrochloric acid and an oxidising agent, such as a few drops of a solution of bleaching-powder, indigo-blue is formed and varying amounts of indigo-red; these pigments are readily extracted by chloroform, which acquires a blue or purple colour and shows a characteristic spectrum.

Indoxyl is formed by the oxidation of indol, and the urinary indoxyl-compounds have their origin in the decomposition of proteids by the intestinal bacteria. When such proteid decomposition is in-

creased, or when the conditions are unusually favourable to the absorption of its products, as when there is obstruction, especially of the small intestine, the excretion of indican is increased. On the other hand, indican has been found to be absent from the urine of new-born infants in the earliest days of life, before the intestines have become the seat of bacterial activity.

Clinically, gastric dilatation, ulcer and new-growths, peritonitis, intestinal obstruction, wasting diseases, such as tuberculosis, and enteric and some other fevers, are among the morbid conditions which are most apt to be attended by an increased excretion of indoxyl-compounds in the urine.

A. E. GARROD.

INDIGESTION.—Difficulty in digestion. See DIGESTION, Disorders of.

INEBRIATES, Detention of.—See DIPSO-MANIA.

INFANTILE CONVULSIONS.—See CONVULSIONS IN CHILDREN.

INFANTILE PARALYSIS.—See PARALYSIS, INFANTILE.

INFANTILE SPASTIC PARAPLEGIA.—See CEREBRAL DIPLEGIA.

INFANTS, Diseases of.—There are few disorders which can be said to be peculiar to infancy and childhood. The diseases to which children are liable are, as a rule, those which attack older per-

sons, and present the same pathological characters. But disease as it occurs in children does yet require especial study, for the symptoms by which it is accompanied often differ widely from those met with in adults.

GENERAL CHARACTERS.—The most striking peculiarity of childhood is the marked excitability of the nervous system. A fragment of indigestible food, for example, may produce high fever, or alarming agitation, and even throw the child into convulsions; a slight irritation of the larynx may produce severe spasm, and simulate for the time the symptoms of diphtheria. The beginning of acute disease is almost invariably accompanied by profound general disturbance; but disturbance as profound may be excited by the simplest functional disorder, so that the severity of the symptoms is no guide at all to the severity of the lesion with which we have to deal. In all cases, therefore, it is of importance if possible, to pick out the local symptoms—those, namely, which point to mischief of any special organ—and separate them from others which are expressive merely of the general distress. Such local symptoms are the cough, rapid breathing, and active nares which point to acute lung-disease; the squinting and immobility of pupils which are so characteristic of cerebral affections; and the peculiar jerking movement of the legs, which, combined with hardness of the abdominal muscles, betrays the existence of colic. Such local symptoms are not, however, always to be discovered, and even if present may not furnish trustworthy indications; for so close is the connection in the young child of distant organs with one another that the organ from which the more definite symptoms appear to arise may not be the organ which is actually the seat of disease. The two organs which are most frequently found to present these deceptive manifestations are the stomach and the brain. Vomiting is a common symptom at the beginning of every acute disease, and in many children any casual disturbance is apt to be attended by it. In some cases of pneumonia, again, notably those in which the inflammation is seated at the apex of the lung, headache, vertigo, delirium, and stupor may be so marked that the ordinary symptoms of the disease are completely obscured, and the case is mistaken for one of meningitis. Again, the violent nocturnal delirium so often excited by the irritation of worms in the alimentary canal must be within the experience of all.

The nervous excitability of children, and its influence upon the system generally, are well illustrated by the high temperature noticed in many children on the first evening after admission into the wards of a hospital. The elevation varies in degree in different subjects; but if the patient be not a mere infant, it is usually over 100° , although the complaint be one not ordinarily attended by pyrexia.

Perhaps, however, the most familiar instance of the impressibility of the nervous system is seen in the case of convulsions. A 'fit' in the child has a very different meaning from a similar attack in the adult. In the latter it is usually evidence of a grave centric lesion, and its occurrence occasions the greatest anxiety. In the child, on the contrary, it is a common expression of the perturbation of the nervous system, set up in response to some excentric irritation, and often, as in the case of the onset of acute disease, is analogous to the rigor which ushers in an acute attack in older persons. Sometimes, it is

true, convulsions are produced in the child, as in the adult, by severe cerebral disease; but in such cases the fits are frequently repeated, and are succeeded by rigidity, paralysis, and other signs of centric irritation. See CONVULSIONS IN CHILDREN.

The impressibility of the nervous system is increased by causes which produce a sudden depression of strength, such as a bad attack of diarrhoea, or loss of blood; and in one chronic disease—rickets—the nervous irritability is very great. The effect of chronic wasting upon the child is, however, usually to produce an opposite result; and under the long-continued influence of enfeebling disease the excitability of the nervous system becomes gradually less and less manifest, until it finally disappears almost entirely. It is of importance to the practitioner to bear this fact in mind, for in a child much reduced by chronic illness, the presence of an intercurrent acute complication—such as inflammation of the lung—may be indicated by very few symptoms, the system having become almost insensible to nervous impressions.

Another peculiarity which strikes the attention of any one accustomed only to disease as it occurs in the adult is the vast preponderance in infantile disorders of mere disturbance of function, and the disastrous consequences which may ensue from such derangements. Infants rapidly part with their heat, and are easily chilled. They are therefore excessively sensitive to changes of temperature. A catarrh is a common ailment in the young child, and is attended by various dangers according to the part of the mucous tract which is affected by it. Gastric catarrh with violent and repeated vomiting, and intestinal catarrh with uncontrollable diarrhoea, are answerable for a large proportion of the deaths among young children during the warmer months. In less severe cases the gradual failure in nutrition which results is a common cause of wasting; such cases may end fatally. In the autumn and winter the bronchial mucous membrane is more frequently attacked. However apparently slight may be the catarrh, a weakly infant is always exposed to the danger of collapse of a considerable portion of the lung; and such an interference with the respiratory function is often a cause of sudden death.

It is in consequence of this frequency of functional derangements, and their dangerous character, that *post-mortem* examinations in infants so often fail to show any appearances explanatory of the cause of death.

CLINICAL EXAMINATION.—The clinical examination of young children requires tact and patience. The patient cannot himself describe his symptoms, but all necessary information can be gained from the parents. Mothers are, as a rule, good observers, and allowing for their natural anxiety and a slight tendency to exaggeration, their statements can usually be relied upon. We can thus learn the previous state of the child, the exact date at which his symptoms began, and the order in which they appeared. Infants should be always stripped for examination, so that the whole body may be exposed to view. Before, however, ordering the removal of the clothes, we should be careful to satisfy ourselves upon certain points which can only be properly observed while the child is in repose. Thus, in order to count the pulse and respiration, perfect quiet is indispensable, for the least movement quickens the heart's action, and alters the rapidity of the breathing. At the same time the

London Hospital.

HOW TO BRING UP INFANTS.

1. Keep them warm. Let the clothing be warm (*i.e.*, flannel or woollen), and not tight. Children should wear stockings nearly up to the knees, and frocks well up to the neck, with long sleeves. Give them plenty of fresh air; take them out whenever the weather is fine. Wash the child all over with warm water and soap daily. If possible let the child sleep in a cot by itself. Open the windows at least twice a day.

FOOD.

2. If the mother has plenty of breast-milk, the child should *not have any other food whatever*, until it is seven months old. It must be suckled every two hours during the first month, and the interval gradually increased, so that at the end of three months the child is suckled every three hours. *Too frequent suckling is a common cause of the sickness of infants.*

3. If the mother has only a little milk, let the child have it; and in addition milk mixed as directed in Rule 4. Except when ordered by a doctor, *do not on any account give the child any baked flour, arrowroot, corn flour, biscuits, tops and bottoms, or any so-called "infants' food" before it is seven months old.* It is a good plan, if the mother has only a little breast-milk, that she should drink a cupful of cow's milk half-an-hour before suckling.

4. If the child must be brought up entirely by hand, it should be fed out of a bottle with cow's milk warmed and diluted. Fresh cow's milk must be used: during the first month it must be diluted with at least twice as much water or, better still, with lime water or barley water* in the same proportion, if the milk be of good quality. A small quantity of *white* sugar must be added to each bottle. As the child gets older, the proportion of milk must be gradually increased. The milk should always be boiled before being used. This is especially important in the case of infants under three months old, and in summer.

* Barley water is prepared as follows:—Two tea-spoonsful of pearl barley to a pint of cold water, boiled down to two-thirds of a pint, and then strained through muslin. A fresh quantity must be prepared at least twice a day.

5. If cow's milk cannot be digested after a fair trial, condensed milk may be given—one tea-spoonful to four or five table-spoonful of water which has been boiled, *but after a month another attempt should be made with fresh cow's milk.*

6. The best kind of bottle is the old fashioned straight one with an indiarubber teat, and without any tube at all. It is quite impossible to prevent corks and tubes from becoming foul and turning the milk sour. When the child has finished its meal the bottle must be rinsed, and placed in clean water until again required. Once a day it should be carefully cleaned with hot water in which a little soda has been dissolved, and then rinsed with water and a few tea-leaves. On no account must the milk that remains, after the child has finished, be used again. A fresh quantity must be prepared for each time of feeding.

7. When the child has reached the age of eight months, it may have one or two meals a day of milk thickened with one or other of the following malted foods, viz.: Allen and Hanbury's, Mellin's and Savory and Moore's. Or Chapman's entire wheaten flour may be used: to prepare this, make a tea-spoonful of the flour into a paste with cold water, pour half a pint of boiling water on it, and let it simmer for twenty minutes, constantly stirring; then add it to the milk, which must be warmed. The other meals should be of milk only. The amount of these foods used may be increased as the child grows older. As a rule, the child should be gradually weaned at ten months' old. At eight months the child may have broth or beef-tea, in addition to the milk. At ten months give fat bacon, the yolk of an egg lightly boiled or beaten up, or a little milk pudding. At eighteen months, give a little meat every day scraped or pounded into soft pulp; or a little mashed potato with gravy. Cheese, beer, or spirits should never be given to children.

**** IT IS A GREAT MISTAKE TO GIVE CHILDREN UNDER TWO YEARS OLD "JUST WHAT THE PARENTS HAVE." IT IS A GREAT MISTAKE TO GIVE THEM TEA INSTEAD OF MILK. ON NO ACCOUNT GIVE BABIES TEETHING POWDERS AND SOOTHING SYRUPS.**

temperature can be taken by the thermometer in the rectum. The whole body should then be examined for spots or swellings; the condition of the skin noted—whether dry or moist; the fontanelles examined; and the state of the abdomen ascertained, including the size of the liver and spleen. If the child cry at the time, we mark the character of the voice, for hoarseness is an early sign of congenital syphilis.

In the physical examination of the chest in a child, it is important: to percuss the two sides at the same period of the respiratory movement, that is, during expiration or during inspiration; to strike gently with *two* fingers, for by this means a larger volume of sound is brought out, and slight dulness is more easily detected; to use a stethoscope instead of the unassisted ear, in order to limit the area listened to; and to manage so that the child's mouth be open during auscultation, so as to hinder the transmission of sounds from the throat. In an infant the back is best examined by placing the child on the nurse's left shoulder, with his left arm round her neck. If the chin be now depressed by the nurse's hand on the child's head, the muscles of both shoulders are relaxed. The front and sides of the chest can be examined as the infant lies on his back. We must remember that the breath-sounds, especially that of inspiration, are of a more blowing quality in the child than they are in the adult; and that there is naturally less resonance at the right base, on account of the proportionately greater size of the liver.

At the end of the examination the mouth should be looked at for signs of aphthæ or thrush; and the condition of the gums and naso-pharynx should be ascertained. Lastly, the throat is to be inspected—depressing the tongue with the handle of a spoon. If there be disorder of the digestive apparatus, such as sickness, constipation, or diarrhoea, it must not be forgotten to examine the discharges carefully. The urine should not be overlooked.

TREATMENT.—In all cases, especially if the patient be an infant, it is important to inquire into the hygienic and dietetic arrangements to which the child is subjected. Children, as a rule, respond well to treatment. This may be explained partly by the large proportion of mere functional derangements in the illnesses to which they are subject, and partly by the state of constant change through which the body is passing; growth and development are active in organs, and the tendency is to repair. The term 'treatment,' however, includes far more than the mere giving of physic. A complete change in all the influences acting upon the patient—a reconstruction of the dietary, and a reformation in the hygienic arrangements, especially with regard to air, light, and clothing—will often prove of immense service, and be of far more value than actual drug-giving in furthering the recovery of the child.

In the treatment of acute illness we must remember that young children cannot bear lowering measures; but we must not therefore rush to the opposite extreme, for unless suffering from temporary exhaustion, they are far from being benefited by profuse stimulation. In the beginning of acute inflammatory diseases stimulants are injurious. Even in chronic ailments, such as rickets, where a certain amount of alcohol is often of service, wine should be given with caution, and its effects upon the digestion carefully watched; it can only be given with

advantage so long as it improves the appetite, and increases the digestive power.

With regard to medicines little need be said in this place. It may be remarked that, on account of the tendency to acid dyspepsia in all children, alkalis are of especial service; and that they should be always combined with an aromatic, on account of the value of the latter in stimulating the alimentary mucous membrane, and relieving the flatulence and other painful consequences of indigestion. It is important also to remember that children are wonderfully tolerant of certain drugs, while they bear others very badly. Belladonna may be given to infants and children in comparatively large doses. They are also more tolerant of arsenic than their elders. To the action of opium, however, they are excessively susceptible, and the drug should be given—to infants especially—with extreme caution.

EUSTACE SMITH.

INFANTS, Feeding of.—See **DIET**; and **ATROPHY**.

INFARCT (*infarcio*, I cram in).—An infarct is a firm wedge-shaped patch of tissue, found in certain organs as the effect of arterial embolism, or a marked diminution (more than 75 per cent.) of the pressure in the supplying artery. See **EMBOLISM**.

INFECTION.—Before the nature of infective processes was understood and when, owing to the difference in the length of incubation-periods and the different results of infection, there appeared to be some difference in the method of transmission of infective disease from patient to patient, it was natural that an attempt should be made to distinguish between those diseases in which there was direct transmission from patient to patient and those in which the transmission was indirect. Again, direct transmission was supposed to be either by direct contact, in which case the disease was spoken of as being 'contagious,' or through the atmosphere, when it was said to be 'infectious.' But since it is now known that, in all infective diseases, there is an infective element which can multiply, sometimes outside the body, and usually inside, it is better to include all these diseases in one great group in which the *materies morbi* can be transmitted by 'contagion,' the principal factor being the multiplying *materies morbi* which, transmitted, directly or indirectly, from patient to patient and finding suitable conditions for its development, is capable of growing and of setting up a specific infective disease. It must be insisted on at the outset that although most of the infective diseases are transmissible from one individual to another—and this group is gradually absorbing all the infective diseases—there are a few which up to the present have not been demonstrated as being transmissible, or as being transmissible only through an intermediate host. For example, malaria and relapsing fever are sometimes said to be non-transmissible. That they do not pass directly from patient to patient may be readily enough accepted, but it is now recognised in the case of malaria, and it is possible that a similar statement holds good also of relapsing fever, that on the parasite being introduced into the blood directly from individual to individual, or being first taken by the mosquito into its digestive cavity, and later, after certain developmental changes, being injected through the proboscis into the blood of the human subject, the disease is again induced;

thus is furnished an instance of a supposed non-transmissible disease affording an example of a typically transmissible one.

In the article 'Bacteria' it is stated that the infective agent in disease is almost invariably a lowly developed micro-organism, a vegetable or an animal blood-parasite. For the present we may leave out of account the larger animal parasites, which, however, in so far as they multiply and give rise to certain lesions, may be looked upon as infective agents. Some of these disease-producing bacteria grow readily outside the body, flourish at a temperature considerably below that of the human blood, subsist on nutrient media outside the body, and retain their vitality under conditions varying in some cases widely from those met with in the animal organism. Such bacteria, after making their way from the patient, may remain alive for considerable periods lying in wait ready to invade another patient and so set up an attack of the disease with which they are causally associated. On the other hand certain disease-producing organisms are endowed merely with the power of existing and multiplying in the fluids and tissues of the living body. They can scarcely exist at any other than the blood-temperature, and may be so constituted that they are either unable to obtain nutriment from dead tissues, or are rapidly killed off by other organisms with which they have to compete. Here the *materies morbi* must be passed on directly from patient to patient if it is to remain alive and active. Once this process is set a-going the infective organism appears to become more and more distinctly parasitic, and so more and more capable of invading the higher organism, of multiplying therein, and of setting up the special fermentative and disease-producing processes with which they are associated. An organism that can live outside the body may of course give rise to a transmissible disease either directly or indirectly, the *materies morbi* remaining outside—sometimes for very considerable periods—but where this disease-producing organism is strictly parasitic and cannot live outside the body, indirect transmissibility, though not unknown, is comparatively rare. The more saprophytic a disease-producing organism is, that is, the more it is able to live outside the body, the more important becomes indirect transmission; while in the case of the strictly parasitic organisms, indirect transmissibility plays a comparatively unimportant part. The facultative saprophytes (those bacteria that can live outside the body) which produce disease vary enormously as regards resistance to external agencies, a variation that may be noticed even in organisms of the same species. For example, the *Bacillus anthracis*, when placed under specially favourable conditions, multiplies very rapidly. When inoculated into the tissues of an animal and thence into the blood, it undergoes rapid multiplication by a process of fission, and in a very short time invades the whole of the fluids of the body. It will be found, however, that in this stage the bacillus is very easily killed. If a drop of the blood of the affected animal be transferred to another animal, the disease is reproduced with great certainty and rapidity. If, however, the animal after it succumbs be buried at once, these bacteria die out and are no longer a source of danger, since the bacteria kept within the body do not develop spores and are comparatively easily killed. If, however, the blood from an animal affected with

anthrax be allowed to escape and come in contact with the air, especially on a porous surface kept fairly moist, as on the surface of damp ground, the contained bacilli rapidly undergo such change that it is with the utmost difficulty they are destroyed, for resisting spores are formed which may be dried, heated, and even treated with certain antiseptics with impunity, and still remain alive, ready to develop into active bacilli as soon as the conditions for their growth again become favourable. They are not killed when they are buried, and, for long periods, remain capable of setting up an infection. In the one case the transmissibility must be direct, in the other case it must necessarily be indirect. The old term of 'contagion' then may be discarded with advantage.

One of the essential features of an infective agent is that it must be capable of multiplication in the fluids and tissues of the animal body, and the more this property is developed, the more dangerous becomes the organism as an infective agent. It is evident that in infection we are dealing with a process of insemination, but all the seed that is sown does not necessarily bear fruit; several factors must be favourable before a disease can be set up even by a disease-producing organism. Among these factors may be considered the following:—*The number of organisms introduced.* It has been found, for example, that a certain number of even virulent anthrax-bacilli may be introduced directly into the circulation of an animal without setting up any specific infective disease, the bactericidal power of healthy blood appearing to be quite sufficient to destroy a small number of bacteria, with the result that no infection takes place. *The virulence of the organism* also plays a part in determining this infection; a much smaller number of a virulent organism being sufficient to bring about what only a much larger number of less virulent organisms can achieve. In the case of the poison-producing organisms, such as the bacilli of tetanus and diphtheria, the poison plays a most important part in determining whether infection takes place or not. The micro-organisms introduced alone may be able to make no headway in the tissues; they are unable to multiply, and are readily destroyed, and no infective process results. If, however, along with the micro-organisms a certain amount of their specific toxin be introduced, the tissues or the fluids of the body with their bactericidal powers may be so reduced or interfered with that they are unable to repel the invasion of the bacteria, and an infective process is set up. *Impaired tissue-vitality*, brought about through whatever agency—want of nutrition, impaired excretion, impaired vitality of any kind through mechanical injury, cold, heat, toxins, or chemical irritants, whether general or local—is also of importance as predisposing to infective diseases from the fact that fewer and less virulent organisms can make good their position in the tissues under these conditions than when the vitality of the tissues is unimpaired (see BACTERIA, p. 133). In this connection must be considered the effect of the invasion of the tissues by two or more organisms. *Mixed infections* are in most instances far more serious in their results than are simple infections, and a disease, which under ordinary circumstances might run a comparatively mild course, may in the presence of the effects of a superadded organism become most dangerous. For example, in 'pre-antitoxin days' it was recognised

that if diphtheria was superadded to a case of scarlet fever, of however mild a type, it very frequently ended fatally, the mortality among such cases being very high indeed. That this was due, in great measure, to the mixed infection is evident from the fact that the mortality among such cases was very much higher than the mortality among cases either of simple scarlet fever or of diphtheria in the same epidemic. We have further evidence of the importance of this cumulative action in the success of the treatment of such cases with antitoxic serum, in which the diphtheria-factor is practically eliminated, the anti-toxin neutralising the diphtheria-toxin. As such cases come under observation at an early period, the treatment can be thoroughly carried out, and the mortality falls to a fraction of what it was in the 'pre-antitoxin period.' This is not an isolated instance. When the *Streptococcus erysipellatis* of a comparatively mild type is inoculated along with the *Bacillus prodigiosus*, which by itself is almost innocuous, a most virulent type of erysipelas may be induced; again, it is a matter of common observation that the *Bacillus tetani*, when reinforced by certain innocuous chemical substances or by certain non-pathogenetic organisms, may set up a most virulent form of tetanus.

Of other predispositions to infection may be mentioned *inflammatory conditions* which appear to act in a very interesting fashion. During an epidemic of cholera, only those patients contract the disease who first suffer from some slight disturbance of the alimentary tract; even when the cholera-bacilli are present in the lower part of the alimentary canal, there may be no attack of cholera unless some inflammation with its accompanying serous exudation be set up. Hueppe and Wood have pointed out that, for the production of considerable quantities of the cholera-poison, the bacillus requires a certain proportion of what may be termed crude albuminous material. This may be supplied in the form of egg-albumen or in the serous constituents of the blood. How does this bear on cholera? The cholera-bacillus in the alimentary canal may remain latent, as it were, for a considerable period, producing a comparatively small amount of poison; it has been so weakened by its passage through the stomach, and the medium on which it can grow is so little favourable, that the toxin is formed in insufficient quantities to do the patient much harm. When, however, from any cause there is irritation of the gastro-intestinal tract, interference with the secretion of acid by the stomach, or the pouring out of serous fluid as the result of an inflammatory process, the conditions immediately favour the production of toxin, and an attack of cholera is the result. There is good reason to assume that in the case both of the typhoid-bacillus and of the cholera-bacillus, the more virulent it is the more nearly it approaches the obligate parasitic condition, i.e. although it grows readily and produces a large quantity of poison within the body, it is much more easily killed outside the body; while, on the other hand, the more saprophytic in character it becomes, that is, the more it resists external influences and the more it becomes modified so that it can lead an existence outside the body, the less virulent are its characters as a parasite and an infective agent. The diphtheria-bacillus also may remain inactive for very considerable periods in the healthy throat; it is only when, as the result of inflammation, there

is an exudation of serous fluid that it is capable of multiplying rapidly, of producing large quantities of toxin from the crude albumen, and so of setting up a characteristic diphtherial infection and intoxication.

This question of predisposition to infection crops up in connection with almost every one of the infective diseases. The tissue-parasites, such as the bacilli of tuberculosis and leprosy, and the streptococci and staphylococci appear to be controlled by the tissues, the general nutrition rather than any special tissue-insusceptibility apparently being the most important factor. In the case of the exanthemata, however, this general nutrition, though, no doubt, playing a part, is comparatively unimportant as compared with special immunity or special predisposition. See IMMUNITY.

It must be borne in mind that even after bacteria—taking them as an example—have gained a footing in the tissues, a considerable period may ensue before they have multiplied sufficiently and have produced their toxin in sufficient quantities to produce any local or constitutional effects in the patient (see INCUBATION). It must be remembered that disease-producing organisms within certain limits always breed true, though they may vary greatly in virulence, in powers of growth, and in power of producing toxins; on the other hand, they may give rise to somewhat different results according to the tissues in which they grow and the paths by which they enter the body. As an example of this may be taken the different forms of tuberculosis, all of them produced by the tubercle-bacillus, but varying enormously in their course and results. In one patient the tubercle-bacillus by its presence may give rise to the formation, first, of comparatively normal granulation-tissue, this gradually developing into fibrous tissue, as in chronic fibroid phthisis; the bacilli in this instance appearing to invade the tissues by the lymphatics. In another case bacilli in exactly the same position may give rise to granulation-tissue much more rapid and luxuriant in its growth, ultimately undergoing caseous degeneration. Again, the tubercular processes resulting from the invasion of an epithelial surface, as in tubercular catarrhal pneumonia, may be essentially different, as regards naked-eye appearances, from chronic fibroid tuberculosis; and when the tubercle-bacilli make their way into the blood-vascular system, miliary tubercles scattered through the whole of the organs of the body—tubercles, which at first sight appear to have little in common with the large caseous tubercular masses found in the lung, liver, or lymphatic glands—result. In certain forms of infection the multiplication of the *materies morbi* takes place not so much in the tissues as in certain cavities, ducts, or channels, or in superficial wounds. It is only when the bacteria in these positions can produce toxins that they can play any important part as infective agents. Indeed it would perhaps be better to speak of them as toxin-producing agents. This has already been referred to in connection with cholera and diphtheria, and the same statements are true of the tetanus-bacillus. The diphtheria-bacillus may, in very severe cases, make its way from the lesion of the throat into distant parts of the body, but in the case of cholera the bacillus appears up to the time of death of the patient to remain in or near the alimentary tract or, in the case of tetanus, in the superficial wound.

Even disease-producing organisms may, as we

have seen in the case of the cholera-bacillus, lead a saprophytic existence in the cavities of the body. The pneumococcus is often found in the secretions of the buccal and respiratory mucous membranes of patients who are suffering from none of the toxic or infective effects usually associated with this organism as a parasite, while it is well known that certain of the pus-producing organisms are regularly present in the tonsils and in the appendages of the skin.

The alimentary canal being a cavity in communication with the outer world may contain in one or other of its divisions almost any organisms usually found outside the body. They may remain simply as saprophytic organisms, though, by the aid of their toxins, or through the weakening of the protective epithelium, they may gradually make their way into the tissues, there becoming parasitic and infective in the true sense of the word; in this they are invariably aided by the pouring out of serous fluids in which they become more and more parasitic and are enabled to produce their specific poisons.

Infection, therefore, may commence with intoxication, and it also results in intoxication. An infection may be local, in which case, as in tetanus and diphtheria, we have the infective agent multiplying at the point of invasion of the organism and sending out toxins to various parts of the body, giving rise to special changes in the central nerve-cells as in tetanus, or in the muscles and nerves as in diphtheria; while in both cases the poison, if present in large quantities, may act directly upon the secretory cells of the liver, kidney, &c. In other cases the infective agent spreads along the lymphatics as in erysipelas, the streptococcus found in this disease gradually passing from point to point, being rapidly destroyed, however, by the tissues which react to the stimulation set up by the advancing micro-organism. Or, again, the infective material may be carried to distant parts of the body by the blood- or lymph-streams as in abscess-formation, the infective agent becoming impacted in a capillary, and setting up a metastatic abscess.

As the local results of infection we may have inflammation, as seen in the dilatation of the blood-vessels, the accumulation of leucocytes, and proliferation of the connective-tissue cells, or, if the process be more acute, fibrinous exudation or death of the tissue resulting from the action of the poisonous substances plus the altered circulatory and mechanical conditions; in abscess-formation peptonising enzymes aid materially in bringing about the destruction of the tissues.

In addition to these special changes are certain general changes, some already mentioned, which result from the action of bacterial poisons, and appear to be due to the action of some substance, common to bacteria, described by Buchner, Centanni, and others as a protein, and found, for example, in pyogenic bacteria, and in the bacilli of anthrax, typhoid fever, and tetanus. When injected it is capable of setting up fever, diarrhoea, leucocytosis, and nutritive, respiratory, and vascular changes. These general changes then are induced by a non-specific bacterial substance similar in certain respects to those initiating local changes.

An infective disease may be endemic, in which case it may be taken for granted that the infective organism is distinctly saprophytic. In such cases the conditions must be exceedingly favourable to the continued existence of the bacillus outside the human or animal body; but in most instances it

appears to be essential that there should be a host to whom, now and again, the parasite may resort in order that it may retain its parasitic habit. There cannot, of course, be any evidence to show that there is any persistence of a disease-producing organism in a purely saprophytic form, without its coming at some time or other to inhabit a higher animal organism.

In the case of epidemics it may be assumed that transmissibility is practically direct from individual to individual, especially where no special predisposition appears to be necessary. Epidemics may result from the rapid distribution of the *materies morbi* from a centre in which it is endemic. This is especially the case in water-borne diseases and when, through starvation or fatigue, large bodies of people, as in times of warfare, are peculiarly susceptible to infection; or, finally, there may be epidemics of diseases from cases of which the virus is readily transmitted indirectly, especially where hygienic and nutritive conditions are bad, as in an outbreak of plague.

In order to deal efficiently with disease a knowledge of the method of infection and the nature of the disease-producing organism must be obtained at the very outset. In glanders, tuberculosis, diphtheria, plague, and the pyogenic affections, as well possibly as measles, variola, scarlatina, and leprosy, the exciting organisms can probably live for a time outside the body, that is, they may be saprophytic organisms. Here then it is necessary to prevent not only direct infection, but also the possibility of these organisms returning to their saprophytic existence. In the case of small-pox and scarlet fever, this can be fairly readily carried out, as in these direct transmission plays a much more important part than indirect. If the patients are kept isolated during the period when the infective organisms are given off, and if disinfection is carefully carried out, there is little danger of the infective agent being passed on to other patients. In tuberculosis, diphtheria, plague, and, to a less degree, glanders, something more than mere isolation is necessary. Building up of the tissues, air, light, and improvement of the general hygienic conditions, all play a great part in preventing infection; and this is especially the case in diseases in which predisposition is an important factor.

Those diseases which result from the invasion of such facultative parasites as the bacilli of typhoid fever, cholera, anthrax, &c., are not to be stamped out by isolation merely. The supply of the infective agent must be cut off, and this must be done while the organism can be readily destroyed, that is, immediately it comes from the patient. But, beyond this, the primary or common source of infection should be found, and measures taken to eradicate it.

As regards those fermentation-processes set up in the alimentary canal—say, by saprophytic organisms which have the power of producing irritant or toxic substances, but which have no power of invading the tissues—little need here be said, except to point out that under certain conditions these may prepare the ground for the action of such disease-producing germs as the cholera- or typhoid-bacillus.

It has been proved experimentally that the channels by which the infective agents enter the system vary very considerably in different cases, and that an organism which inhaled might give rise to a very severe attack of a disease would, as in the case of

small-pox, when introduced subcutaneously set up a comparatively mild attack. There are numerous similar infective agents. For example, the bacillus of Quarter-III, when injected into the subcutaneous tissue, gives rise to an attack of the disease, while, injected into the blood-vessels, it produces little or no effect. Further, a much larger dose of *Bacillus pyocyaneus* can be given subcutaneously than either intra-peritoneally or intra-venously. The typhoid-bacillus, on the other hand, is better borne when injected intra-venously than intra-peritoneally, while an animal will bear an intra-peritoneal injection of a larger quantity of the diphtheria-bacillus than it will bear subcutaneously.

As regards the protective action of the gastric juice, it may be mentioned that the acid contained therein affords very efficient protection against the invasion of the lower regions of the alimentary tract by the tubercle-bacillus or the cholera-bacillus. Tuberculosis of the intestinal tract, where the gastric secretion remains normal, appears to be almost unknown; while Koch found that it was necessary to neutralise this acid in the stomach of the guinea-pig before there was any possibility of the cholera-bacillus passing through the stomach into the intestinal canal. Even when it passed this portal, it met another of the protective agencies, the peristaltic action of the intestine, which, keeping the contents of the intestine with the contained cholera-bacilli constantly in motion, prevented the development of the toxins, with the result that no cholera was produced. There was no multiplication of the cholera-bacillus unless this movement was paralysed by the exhibition of morphia.

GERMAN SIMS WOODHEAD.

INFILTRATION (*in*, into; and *filtrō*, I strain through felt).—See DEGENERATION; and CONCRETIONS.

INFLAMMATION (*inflammō*, I set on fire).—SYNON.: Fr. *Inflammation*; Ger. *Entzündung*.

DEFINITION.—Very numerous definitions have been given of inflammation. The most generally received has been that attributed to Celsus, which gives the four marks of inflammation as *rubor, tumor, calor, dolor*; but this appears to have been really due to Erasistratus, who, according to Galen, first gave precision to the conception of simple burning, as understood by the older Greek physicians, and applied the name *φλεγμονή* (previously synonymous with *φλόγωσις*) to a swelling, which had also the characters of heat, pain, throbbing, and resistance. The definition was thus based upon the notion of *swelling*, and would hardly have taken this particular form had it been derived from superficial inflammations. Although the four 'cardinal' signs may still be recognised in what we call inflammation, this definition is now inadequate. Many attempts have been made to arrive at one which shall be more complete; and at the present day very various definitions are given by different pathologists.

The most popular definition is based upon the cause, viz. that inflammation is a series of changes produced in a part by injury which may be mechanical, physical, chemical, or due to living organisms; provided the damage produced does not entirely destroy the vitality of the part. This is a definition of the cause rather than of the process, and is logically defective since it excludes certain consequences of injury such as hemorrhage and the results of simple section of

vessels, nerves, &c. Some (as Adami) limit the definition to the 'local attempt at repair of injury,' which others object to as involving the teleological idea of purpose or intention. Others would include only injuries produced by micro-organisms which are the chief causes of the diseases called inflammations in the human body. Some exclude the process of repair from inflammation proper; others (as Ziegler) would define the process as *local tissue-degeneration* produced by injury, combined with *exudation* and followed sooner or later by *regeneration*. It would be really more correct to speak of inflammations than of inflammation; since the changes produced by different specific injuries differ so much that it is difficult to define any one form which can be called simple inflammation. In the face of these conflicting definitions, Thoma (with whom the present writer agrees) has proposed to drop the term 'inflammation' altogether, as involving merely a traditional conception, and to speak only of injury and the reaction produced by it; the process of repair being considered separately. As, however, the term 'inflammation' is certainly likely to be still used, it cannot be at once dropped, but we may understand that it means nothing more than certain consequences of injury. These consequences appear to be: (1) damage of cells or tissues; (2) a defensive reaction on the part of the organism which has the effect of removing the injurious agent, or of neutralising wholly or completely its effects; and (3) the process of repair which may or may not be regarded as actually a part of the same process. These three factors will have to be considered separately.

1. *Damage or injury.*—Admitting that an injury of some kind is always the starting-point of the processes called inflammation, we must regard the other changes as secondary to this. In all animals with a complete blood-circulation, injury to any vascular part sets up disturbances of the nervous and vascular mechanism, resulting in hyperæmia, which in slight injuries may be the only visible effect. In severer injuries they always accompany the other changes, and hence are an integral part of the process. Bearing this in mind, a gradation may be traced in the effects of injury. A very slight injury to the skin, for instance, by friction, heat, or a chemical irritant, produces transitory hyperæmia; a more severe or more lasting injury sets up inflammation (blister or eczema); one still more intense, necrosis or sloughing. The same gradation may be seen in the after-effects of cold, which, according to its duration or intensity, may lead to mere heat and throbbing, or to inflammation (chilblains or mild frostbite), or to actual gangrene. Even in apparently spontaneous inflammations a cause of injury may always be traced, such, for instance, as a diffusible virus, or the presence of micro-organisms and their products. Again, disturbances of circulation and innervation, as in the secondary inflammations of circulatory and nervous diseases, or a mere lowered state of nutrition, as in various chronic cachexias, are regarded as causes of inflammation; but in such cases there is generally a combination of injuries, the presence of some noxious substance coinciding with nervous, vascular, or nutritional disturbance. Frequently it appears that the lowered state of nutrition thus induced permits micro-organisms to exert their pathogenic effects, which the resistance of healthy tissues would otherwise inhibit.

Damage of cells or tissues shows various degrees up to and including total necrosis. In cells the chief signs are cloudiness, or cloudy swelling of the protoplasm; and indistinctness of the nuclei which are imperfectly coloured by staining reagents.

Fibres of connective-tissue swell up and lose their elasticity; muscle-fibres lose their striation and the nuclei become indistinct; nerve-fibres pass into fatty degeneration. These changes were called by Virchow 'parenchymatous inflammation,' but are not now regarded as constituting inflammation unless accompanied by other changes.

2. *The defensive reaction* includes (at least in the higher animals) hyperæmia and exudation. The exudate not only acts mechanically, but by its chemical constituents has an inhibitory action on micro-organisms; and it also assists nutrition in the process of repair. Some, with Metschnikoff, attribute a special defensive function to certain leucocytes. The new formation of connective tissue is also a defensive reaction, forming a barrier against the penetration of foreign substances into the organism, or surrounding them so as to render them inert.

3. The process of repair consists in the new formation of tissue-elements to replace those destroyed. It is seen in epithelial, muscular, and nervous elements, but most conspicuously in connective-tissue. The newly formed connective-tissue not only constitutes a defensive barrier, but replaces other tissues which have less power of repair, thus forming a scar.

Since animals are so constructed that injuries short of death always call forth some attempt at repair, we find that along with damage there is always some sign of restoration. The combination of these two factors constitutes inflammation, distinguishing it from necrosis on the one hand, and simple hyperplasia on the other. Accordingly a part of the inflammatory process may be regarded as conservative, tending to compensate the disturbance produced by injury. Hence inflammation often accompanies repair, but can hardly be considered as a necessary part of the latter process. The truth appears to be that in 'clean' or aseptic wounds, however extensive, there is little damage to the tissues. Damage is caused by the secondary injuries resulting from the entrance of bacteria or irritating substances, and when this occurs there is inflammation. In this case the delayed healing which follows cannot be attributed to the original injury, nor, strictly speaking, to inflammation, but to the secondary complications which produce the inflammation, and which this process actually tends to compensate and remove.

These secondary processes may be seen in operation simultaneously in any inflamed—that is injured—part; but the damage to the tissue is evidently the primary change, which calls forth the other processes. In studying the effects of injury in their simplest form, that is, in the lower animals without a blood-circulation, it is evidently right to begin with the tissue-changes; but since in the higher animals the vascular changes are indissolubly associated with these, and are the most conspicuous, it will be more convenient to begin with them. We shall, therefore, consider separately: (1) the vascular processes—hyperæmia and exudation; (2) the changes in the tissues.

I. DIRECT OBSERVATION OF THE VASCULAR PROCESSES IN INFLAMMATION.—When the mesen-

tery or tongue of a frog is drawn out and placed under the microscope, the contact of the air soon determines inflammation. The earliest change seen in the vessels of a part thus exposed is dilatation, first of the arteries, then of the veins, the capillaries being little affected. The dilatation of the vessels is accompanied at first by acceleration of the blood-stream, most noticeable in the arteries; but the acceleration does not last more than half an hour or an hour, and it then gives place to retardation, which continues as long as the inflammation lasts. The 'primary acceleration' is, however, sometimes absent or too short in its duration to be noticeable. So far, the process is doubtless the same as in active hyperæmia produced by local irritation. Whether the first dilatation is due to direct paralysis of the muscular walls of the arteries, or to a reflex action passing through the spinal cord, or to an inhibitory action passing through nerve-ganglia in the arterial walls, is uncertain. There is, however, no necessary connection with the spinal cord, since dilatation may take place when the part is disconnected from the great nerve-centres.

From this point begin the phenomena peculiar to inflammation. The dilatation of arteries may go on increasing for ten or twelve hours, till they have twice their original diameter, and pulsation becomes very prominent in them. The capillaries look as if gorged with corpuscles, forming a quasi-solid mass. The blood-current in all the vessels becomes slower and slower, till it is almost stagnant. This condition has been called *stasis*; but it is better to reserve this name for an absolute stagnation, which is no necessary part of inflammation, but is produced in experimental inflammations by local causes, chiefly, probably, by drying up of the tissues. In true inflammation the amount of blood passing through the part is always greatly increased, as may be seen by the fuller current in the veins leading from it. At the same time certain peculiarities are observed in the behaviour of the red corpuscles and leucocytes in the veins. In ordinary conditions of the circulation, the central part only of the vein (as of the artery) is occupied by the corpuscles, which move on mingled together. But during retardation the corpuscles, especially the leucocytes, spread over the marginal portion of the vein usually free from them, and the leucocytes begin to drag along the walls of the vein, as if adherent, till at length they form a layer lining the wall of the vessel, while the red corpuscles are carried on by the current. In the capillaries, this marginal layer is never perfectly established, though leucocytes may be seen momentarily adhering to, or moving slowly along, the walls. In the arteries, no such process is observed, except (according to Cohnheim) for a moment during the diastole of the pulse. This *marginal position* of corpuscles is a purely physical phenomenon, depending upon retarded motion. It has even been imitated experimentally in the case of particles moving in a fluid where the particles have a lower specific gravity than the fluid (Hamilton). When this *marginal position* of leucocytes is established, begins the process which, observed long ago by Waller, and less clearly though earlier by William Addison, was re-observed and brought into notice by Cohnheim. In the words of the last-named observer: 'On the outer contour of the wall of a vessel, usually a vein, in which the marginal layer of leucocytes is well

developed, sometimes first in a capillary, is seen a small projection which enlarges in length and breadth, and becomes a roundish colourless lump. This again enlarges, puts out new pointed projections, and gradually withdraws itself from the wall of the vessel, till it is attached to it only by a long narrow stem. Finally this attachment also is broken, and we see a colourless contractile body with one long process and several shorter, with one or several nuclei—in fact, a *leucocyte*.⁷ The same process is going on meanwhile at other points of the veins and capillaries, till at length, either quickly or slowly, the outer surface of all the visible veins becomes covered with several rows of leucocytes, while their interior shows the same appearance as before. That the leucocytes seen outside were formerly inside the veins, having simply passed through the walls, admits of no reasonable doubt, though it is often difficult for the eye to seize the precise moment of passage. In the arteries nothing of the kind is seen. In the capillaries the emigration of leucocytes is very evident, and accompanied by the passage of red corpuscles also through the walls, which does not take place in the veins proper. It is also clear that the passage of corpuscles takes place at the lines of junction of the cells constituting the capillary wall. It has been shown that the blood-plaques or platelets emigrate from the vessels under the same circumstances.

Accompanying the extravasation of the blood-corpuscles there is always a copious exudation of fluid, which goes far beyond physiological limits. The fluid also differs from the ordinary physiological transudation and more resembles blood-plasma. Aided by this and by their own spontaneous movements, the leucocytes are carried far and wide into the tissues, till the whole field, that is, the whole mesentery, is so crowded with them that nothing else can be seen. The red corpuscles, on the other hand, remain more closely in the neighbourhood of the vessels. When the exudation, carrying with it the corpuscles, and containing, as it does, coagulable material, reaches a surface, it forms the layer of inflammatory lymph or 'false membrane,' seen in inflammations of serous surfaces. The tissue, at the same time, becomes more or less infiltrated with lymphoid corpuscles, producing the condition known as 'small-celled infiltration.'

Cause of retardation and cell-emigration.—The processes just described mark inflammation off sharply from simple hyperæmia. There is little doubt that the essential factor in their production is a change in the constitution of the vascular wall, though it is possible that this may be connected with a change in the tissues outside, especially with a loss of elasticity, which may be traced in all injured tissues—as may be seen, for instance, in the production of a 'wheal' by a blow on the skin. The same change, while it retards the passage of blood, makes the vascular wall permeable to the corpuscles, and favours exudation. What the change is we cannot say, but it seems probable that it is the same as that which the walls of vessels undergo when inadequately nourished. Two classes of experiments throw light on this point. It has been found by Ryneck that fluids other than blood, such as milk, pass with greater difficulty than usual through the vessels of an irritated part; and that stagnation can be produced in the vessels of a frog, which is kept alive by the circulation of salt-solution instead of blood in its vessels. Cohnheim has

also shown that a state of the vascular walls similar to that which may be presumed to exist in inflammation may be produced by shutting off the blood from the vessels for a certain time. He put a ligature round the tongue of a frog, and observed the vessels after the ligature was cut. If the blood had been excluded for twelve to twenty-four hours only, the vessels on the return of the blood passed into a condition of simple hyperæmia; but if longer, stagnation, marginal position of leucocytes, and extravasation of corpuscles were observed, and these phenomena were more marked the longer the ligature had remained, provided it was not long enough entirely to destroy the vitality of the part. From these experiments we must conclude that the cause of the stagnation and its attendant phenomena is not in the blood or the cells, but in the walls of the vessels, and that the change in these is of the nature of degeneration.

2. CHANGES OF THE TISSUE-ELEMENTS.—*Tissue-changes in non-vascular parts.*—If the centre of a frog's cornea be touched with a solution of chloride of zinc, the nearest corneal corpuscles are destroyed. No naked-eye change follows, but in three days karyokinetic changes may be observed in the surrounding corpuscles. By this process repair is completed. If, however, the anterior corneal lamina be destroyed, or if a considerable area of the cornea be damaged, the cornea becomes turbid from the presence of large numbers of migratory leucocytes brought to the spot by the lymphatics. Some of these migrated cells may, perhaps, assist in forming the repair-tissue.

Tissue-changes in vascular parts.—In the omentum of mammalia, changes occur during inflammation in which unbiassed observation can see nothing else than cell-division, growth, and germination. There is indeed reason to believe that such appearances are found normally, as evidence merely of growth, but the inflammatory changes are distinguished from the normal by their greater rapidity and luxuriance. In fibrous connective tissue similar processes have long been observed, and, since attention was drawn to them by Virchow, have been regarded till lately as showing, in the clearest manner, that the fixed cells of the tissue germinate and produce new elements. This 'proliferation of connective tissue' is an obvious fact, but its importance is chiefly, if not entirely, in relation to tissue-formation, and not in the production of lymphoid or pus-cells, often ascribed to it. Around inflamed parts the connective tissue is often found infiltrated with lymphoid cells, the origin of which was formerly set down to proliferation, but which are chiefly leucocytes derived from the veins and capillaries of the inflamed part. The possibility, however, of the production of some lymphoid cells by proliferation of the fixed elements cannot be denied.

There is also reason to believe that the leucocytes emigrated from the vessels may further divide and 'proliferate.' Stricker and Klein have both observed actual cell-division take place under the microscope; so that some of the new cells may be regarded as the descendants of emigrated corpuscles. We may then compare the small or lymphoid cells of inflamed parts to the population of a colony, where most are emigrants, or the descendants of emigrants, but some few trace their descent from the aboriginal inhabitants.

Activity of the Corpuscles outside the Vessels—Phagocytosis.—The lymphoid corpuscles derived

from the blood-vessels show great activity and probably discharge important functions after they have left the vessels. They certainly bear a part in the mechanism of defence. One evident function which they perform is that of absorbing and removing injurious foreign particles from the seat of inflammation. Molecules of fat and pigment, and fragments of dead cells and tissues are taken up by them. When charged with fatty molecules they are the *granular corpuscles* once considered a special sign of inflammation. They also absorb bacteria, both dead and living. According to Metschnikoff there is a perpetual combat between the cells and the bacteria, and the name 'Phagocytes' has been given to those which have this power of absorption and destruction; the whole process being called 'Phagocytosis.' See PHAGOCYTOSIS.

Chemiotaxis.—The movements of these cells have been shown to be remarkably influenced by chemical substances dissolved in the serum which surrounds them, as well as by solid particles. Finely powdered copper and mercury attract the migratory cells, while gold and silver are inactive; but the most powerful attraction is exerted by chemical products derived from bacteria, or bacterial proteins. The protein from *B. pyocyaneus*, according to Büchner, acts even in a dilution of 1 in 3,000. Casein, gelatin, and certain albuminates act in the same way. This attractive power is called *positive chemiotaxis*. On the other hand ammonia, urea, leucin, and other substances are found to repel the wandering cells, exerting a *negative chemiotaxis*. Some of these facts explain the attraction of migratory corpuscles to an injured—i.e. inflamed—part, and also the action of these corpuscles in antagonising the effects of injury.

The importance of the processes of phagocytosis and chemiotaxis is that they indicate a direct action of the cells—chiefly blood- and lymph-corpuscles, but also some mesoblastic tissue-cells—against the injuries which constitute inflammation.

As it is chiefly bacteria and bacterial products which call forth these activities, we may conclude that their function is chiefly in relation to these organisms. The utility of these processes appears to be as a means of defence against the invasion of bacteria, and against the resulting infection. See IMMUNITY.

Changes in the tissue-elements.—These always show the combination of retrograde or degenerative with progressive or germinative changes, characteristic of inflammation, sometimes the one, sometimes the other, predominating, according to the severity of the injury and the nature of the tissue.

In *connective tissue* the retrograde changes consist in swelling and softening of the fibres, which may end in liquefaction, especially when there is suppuration. The progressive changes consist in proliferation and formation of new cells for the purpose of repair. Special histological methods generally permit us to distinguish these elements from migratory lymphoid corpuscles. Nuclear changes (karyokinesis) may often be traced in inflamed parts by proper methods, and are the most certain evidence of cell-proliferation, but are by no means peculiar to inflammation (see CELL). Tissue-repair begins with the formation of so-called epithelioid cells or fibroblasts, which sometimes enlarge into giant-cells. The nuclei of these cells become the nuclei of the new tissue-elements, the fibres of which are probably formed by splitting up of the protoplasm of the cells. Outgrowths from

the surrounding blood-vessels run into the new tissue and vascularise it. The whole process is thus one of continuous growth, quite independent of the migratory lymphoid cells, and of the hyperæmia and exudation, except in so far that the latter help by affording a more abundant supply of nourishment. The relation of the lymphoid cells to repair is thus very obscure.

Muscular tissue becomes granular and loses its striation under the influence of inflammation, and sometimes undergoes another peculiar form of degeneration. But at the same time the muscle-nuclei (unless the tissue be absolutely killed) show signs of growth and proliferation, by which in the end new fibres are produced and the tissue regenerated.

Nerve-fibres undergo rapid disintegration when inflamed; but the nuclei preserve their vitality, and are capable of producing new nerve-tubes, as is seen after section of nerves.

Ganglionic nervous tissue never shows any signs of regeneration after being inflamed, and is probably killed by comparatively slight and transient inflammations, though this tissue is very rarely thus affected.

Epithelium on mucous surfaces shows very characteristic changes. A large number of elements die and are shed off, so that the desquamation of epithelium is far more rapid than under normal conditions. Many cells show excessive mucous transformation and—in chronic inflammations—much fatty degeneration. There is also a copious formation of new epithelial cells, produced, as in normal conditions, apparently from the basement membrane.

Glandular epithelium, when inflamed, shows the characteristic change called granular degeneration or cloudy swelling (see DEGENERATION), which has been also called parenchymatous inflammation, and is evidence of the simplest kind of injury to the cells. Severe injuries cause fatty degeneration and breaking down, as seen in various kinds of poisoning. There is probably always a regeneration of gland-cells, but this is often traced with great difficulty.

The above are the tissue-changes usually met with in ordinary inflammations. When the inflammation takes the form of *suppuration*, destruction of tissue is far more complete, and repair is prevented, at least temporarily.

PRODUCTS AND RESULTS.—All inflammatory products result either from exudation or from new-growth. The exudative products are serum, mucus, and fibrin, the latter of which, in combination with leucocytes, forms the so-called 'inflammatory lymph.' New-growth takes place from the vessels in the form of vascular connective tissue, of which granulations are a special form. With regard to exudations, no clear line can be drawn, at any stage of the process, between serous and fibrinous exudation or inflammatory lymph. But the production of pus seems to require some special irritant or morbid factor.

Exudations.—Serous and mucous exudations can only be regarded as products of inflammation when excessive. The fluids formed in inflammations of serous cavities differ from those produced in passive exudations (or dropsies), in containing more fibrin and more albumen. But inflammatory exudations vary in this respect, and are sometimes scarcely to be distinguished from simple serous effusions. The

aids poured out on serous surfaces in acute inflammation always coagulate, and even in chronic cases are often shown to be capable of coagulation. On mucous surfaces, on the contrary, the exudation does not as a rule coagulate. That this is owing in some way to the action of the epithelium seems most probable, whether it is that filtration through epithelium alters the composition of the fluid, or whether the living epithelium prevents coagulation in the same way as the endothelium of the vessels prevents coagulation of the circulating blood. When, however, the epithelium is removed, a fibrinous layer may be produced on the exposed sub-mucous surface; and the same result seems to follow the application of very powerful irritants, as in croupous inflammations. Mucous exudations contain mucin, as well as serum-albumen, in variable proportions.

Fibrinous exudations, in coagulating, entangle whatever leucocytes may be either extruded with the exudation or present in the tissues. The properties of the coagulated mass differ according to the proportion of corpuscles. These differences do not necessarily show any corresponding differences in the composition of the blood, but depend upon the facility with which the corpuscles leave the vessels, and probably on the state of nutrition of the latter. The product called *inflammatory lymph* consists of coagulated fibrin entangling leucocytes, the two constituents being in varying proportions. The fibrin does not differ from that of blood-clot; and may therefore be formed in the same way by a reaction between the exuded constituents of blood. But it is not a constant product of inflammation, and hence has been thought to owe its production to local causes—that is, to reaction between the tissues and the exudation. Thus fibrin is formed on serous surfaces where one of the fibrin-constituents is normally found, and in fibrous tissue where there are similar chemical constituents; but, as stated above, not generally on inflamed mucous surfaces, or in epithelial structures. But since it is possible that in these cases the exudation becomes altered by filtration through the tissues, there is no reason to doubt that fibrin, or both its chemical constituents, may be exuded from the vessels. The inflammatory fibrin itself is never *organised*, but often becomes *replaced* by a vascular connective tissue which grows into it, and causes its absorption. This is the process formerly spoken of as ‘organisation of lymph.’

Pus.—Pus is inflammatory exudation in which the corpuscles greatly predominate, and the intermediate substance is liquid. It is thus difficult to draw a line between pus and some forms of inflammatory lymph, but the former does not contain fibrin, nor does it coagulate spontaneously. For the characters of pus and the exciting causes of suppuration reference may be made to the articles upon **ABSCESS** and **PYOGENIC ORGANISMS**.

Granulation-Tissue.—When a part has been destroyed by inflammation, the lost tissue is replaced by the preliminary formation of a peculiar structure, consisting of a highly vascular connective-tissue framework, containing an excessive number of leucocytes, and also epithelioid or formative cells, with occasional giant-cells. See **WOUNDS**, **Healing of**.

VARIETIES.—The most striking differences between different kinds of inflammation are those depending upon the differences of tissues, and of the situations in which it occurs.

1. *Catarrhal.*—On mucous membranes the exudation is mucous, does not coagulate, and contains only detached epithelial cells, with scattered leucocytes; the process tends to become chronic; but the effect on the body as a whole is less marked than in other forms. The term catarrhal inflammation is with less propriety transferred to inflammations of the skin, the lung, and some glandular organs, in which desquamation of epithelial cells is a prominent feature.

If catarrhal inflammation is very severe it becomes purulent, and the exudation consists chiefly of pus, little or no mucus being produced. This purulent catarrh is especially characteristic of specific inflammations of the mucous surfaces, such as virulent conjunctivitis or gonorrhoea.

2. *Croupous or fibrinous.*—Croupous inflammation is that form in which a coagulable exudation is formed upon a mucous surface. In *diphtheritic* inflammation there is, besides a membranous exudation, some necrosis of the mucous membrane. In these forms the tendency is to acute, not chronic, disease; and the general symptoms are strongly marked. The name ‘croupous’ has also been transferred to certain inflammations of the lung (‘croupous pneumonia’) and of the kidney, but (in the latter case especially) with doubtful propriety.

The *fibrinous* form may be regarded as the normal or usual form of inflammation of serous membranes and fibrous connective tissue.

On serous surfaces the lowest degree of inflammation is seen in a serous exudation, hardly to be distinguished from simple dropsy; but there is no clear line between this and a coagulable exudation, or fibrinous inflammation. This too, if still more severe, may become purulent; and, as we see in the pericardium or pleura, a purulent may succeed to a fibrinous inflammation. Finally, vascular connective tissue, forming adhesions, is generally produced. In areolar connective tissue, and in the interstitial tissue of various organs, the same stages may be distinguished, known as *inflammatory oedema*, which occurs near a focus of acute inflammation; *inflammatory hardening*, such as precedes the formation of an abscess; and, finally, either abscess itself or purulent infiltration—the two forms of suppuration in connective tissue.

3. *Parenchymatous.*—The name ‘parenchymatous inflammation’ has been given to the changes occurring in the special tissues of organs independent of their connective-tissue framework. In contradistinction to this, inflammation of the connective-tissue framework is termed *interstitial inflammation*.

4. *Phlegmonous.*—Phlegmonous inflammation is the same as *acute interstitial inflammation*, ending in the formation of abscess.

5. *Indurative.*—Indurative inflammation is that in which new connective tissue is produced in the interior of organs. This is *chronic interstitial inflammation*.

6. *Scrofulous.*—Scrofulous inflammation has been described as that type which occurs in cachectic persons, whose tissues are easily injured and heal slowly. But there can be little doubt that all scrofulous inflammations are really tubercular. *Strumous* is a word better forgotten, being synonymous with scrofulous, and liable to be misunderstood.

7. *Infective.*—Infective inflammations are those

produced by the passage into the blood of infective organisms, derived from some previously existing inflammation. Pyæmia and inflammations associated with the puerperal state are instances.

8. *Chronic*.—Most inflammations have at first a typical course, reaching their acme and then declining. If the decline is not followed by resolution they become chronic. Others, again, show from the first the character they all along maintain. Chronic inflammations are usually distinguished by the persistence of that condition of the vessels which permits exudation and cell-emigration, with less hyperæmia and general fever than in the acute form. On mucous surfaces the chronic form differs little from the acute, except in these two respects. In serous membranes chronic inflammation produces fibrous adhesions, with little or no liquid exudation. In the interstitial tissue of solid organs a large amount of new connective tissue is produced by chronic inflammation, which first compresses the special elements, causing them to waste, and then contracts in bulk, so that the organ becomes atrophied, and usually harder and more fibrous. This is the process called 'fibroid degeneration,' as in cirrhosis of the liver. 'Chronic parenchymatous inflammations' are simple degenerations, as seen in the kidney.

TERMINATIONS.—The most favourable termination of inflammation is what is called *resolution*, in which the vascular phenomena and tissue-changes decline together, and pass away without leaving any tangible material result. Even in the apparently most perfect cases of resolution, there is little doubt that products of exudation remain when the vascular changes have subsided, and are slowly removed by the lymphatics. Other so-called terminations are *necrosis*, or total death of the part; and partial destruction by suppurative or *ulceration*. But if any loss of substance occurs, the inflammatory process cannot be regarded as at an end till the loss is wholly or partially restored by newly formed connective tissue. Where there is no destruction of tissue, but only masses of liquid or solid exudation, the inflammation is not, strictly speaking, resolved till these are removed. Very frequently an acute passes into a chronic inflammation.

CONSEQUENCES.—1. *Local consequences*.—If an inflamed part does not simply return to its original state, *atrophy* is the most common consequence. *Hypertrophy* can hardly be said to occur in the part actually inflamed, though it may in adjoining parts, as we see in enlargement of bone from periostitis. False hypertrophy from new formation of connective tissue is common; but, as such tissue contracts, the final result is atrophy. The hardness of this tissue causes *induration* to be put down among the consequences of inflammation.

2. *General consequences*.—The effect of inflammation on the whole body is to produce the condition of *fever*, which is discussed in another part of this work (see FEVER). It is most probable that some fever-producing or *pyrogenic* substance passes into the blood from every inflamed part, and causes increased tissue-change, with consequent increased production of heat through the whole body; or else exerts a special action on the heat-centres. Several substances having a pyrogenic property have been isolated. They are either animal ferments (enzymes) or albumoses, produced by the action of bacteria on proteid substances. The degree in which local inflammations cause fever

varies, and does not appear to depend wholly on the mass or the intensity of the inflammation, though both these conditions are partly concerned. Acute inflammations produce more fever than chronic; those of connective-tissue more than those of mucous surfaces; and, most of all, those which end in suppuration. In infective inflammations the fever is generally high, but not to be attributed to the local inflammation, being a concurrent effect of the same cause—the infective poison. Besides general fever, the special conditions called *pyæmia* and *septicæmia* may be consequences of primary inflammation; but only when certain special causes are at work. See FEVER; and PYÆMIA.

ÆTIOLOGY.—All inflammations are caused by some injury—either mechanical, as by actual violence; or physical, as changes of temperature; or chemical, by powerfully acting substances, as acids, alkalis, and many more; or by micro-organisms. A most important secondary factor is the condition of the body, whether under- or over-nourished, or in some other way wrong; and this may probably be still more closely defined as the condition of the blood-vessels. Many parts of the body, as the skin and the stomach, are constantly exposed to injury, but do not become inflamed except from some internal cause; and, therefore, a change of nutrition may be the apparent or immediate cause. It is also clear that certain inflammations, as herpes zoster, are determined by disturbance of the nerves; and it is very probable that similar nervous disturbances cause other local inflammations. Besides these, there are certain specific causes, namely, infective or specific poisons, which, when introduced into the blood, produce local inflammations. Many local inflammations, external and internal, appear to arise spontaneously, neither irritation nor fault of nutrition being easily traced. But in these, as in others, it is probable that some infective cause is at work, or else that there is a gradual accumulation in the blood of some irritant substance, the effects of which are only seen when the nutrition of the part is lowered. Such, for instance, are the secondary inflammations which occur in chronic diseases (see BRIGHT'S DISEASE). It is also probable, and in some cases proved, that other inflammations which were at one time thought spontaneous, are really secondary, depending upon some previous local inflammation, even without what is called actual pyæmia. Thus the number of such apparently spontaneous inflammations is gradually lessening.

TREATMENT.—Doubtless the most satisfactory method of treating inflammation is by removing its cause. This cause being in so many cases the action of bacteria, antiseptic or bactericidal substances are often the most powerful agencies which can be used in treating inflammations, if they can be brought in contact with the pathogenic organisms. Many of the agents used in the treatment of local inflammations really act as bactericides. The treatment of gonorrhœa is a familiar instance. But these methods of treatment are *specific*, and cannot be brought under general rules; and are not here discussed. Treatment of inflammation proper is only called for when the inflammatory or defensive reaction is so much in excess as itself to constitute an injury. We shall divide this subject into the treatment of *directly accessible* (chiefly external) inflammations; and that of *indirectly accessible*

internal) inflammations. Treatment will also differ according as the inflammation is acute or chronic ; and also according to the constitution of the patient.

(a) *Directly accessible*.—Directly accessible inflammation in an early stage may be treated by local blood-letting, and by astringents. The benefit of local blood-letting in an early stage is undoubted, and it is probably due to its relieving the condition of stagnation, and permitting freer circulation of blood in the part. Of astringents the type is *cold*. When the vascular disturbance, that is, hyperæmia, is great, and the general fever high, cold, produced either by ice or evaporation, is generally the best treatment. In the case of mechanical injuries, for instance, it may be regarded as an ascertained fact that if any injured part be kept cold during the period of reaction, the inflammation is less severe. When the condition of inflammatory exudation is set up, the effect of cold is less marked ; and if a temporary benefit is produced, the condition after the application of cold is stopped may be as bad as ever, or perhaps worse. Cold is also a powerful nervous sedative, and reduces the nervous irritability of inflamed parts. An effect like that of cold is produced by solutions of certain *metallic salts*, especially those of lead, zinc, silver, and bismuth. These salts furnish the most certain and direct means of treating an inflammation in parts actually accessible to their action. Hence they are used in superficial inflammations of the mucous membranes, such as the digestive mucous membrane and the conjunctiva, and of the skin. These mineral (and also vegetable) astringents act more potently on the exudative processes of inflammation than on the vascular disturbance. Hence their activity is most valuable when that of cold ends ; and they have a striking effect on chronic inflammations which are unaffected by cold. Even *pressure* may be useful as an astringent, as we see in strapping a testicle or an inflamed joint.

In the treatment of even acute inflammations, the precisely opposite application, that of *heat*, is sometimes valuable. There are instances (such as the cure of a whitlow by plunging the finger into very hot water) where the sudden application of heat seems to cut an inflammation short. Probably this is only when the cause is some infective micro-organism. Short of this, heat doubtless increases the activity of most inflammatory processes. But heat combined with *moisture* is the type of an *emollient*, by which the substance of inflamed tissues is relaxed, the blood-vessels dilated, the sense of tension and nervous irritation removed ; and though exudative processes are probably encouraged, the *mechanical* condition of inflammatory stagnation is relieved, and resolution is thus hastened. When pus is forming, there is little doubt that heat and moisture (in the form of poultices and fomentations) hasten the process, and increase the amount of pus formed ; but since the increased production of pus is of no consequence, and it is more important to hasten the process, this may be the right treatment for suppuration. When pus is once formed, the same treatment is useful in guiding it in the direction in which it is least hurtful. Finally, it may be beneficial to apply heat and moisture superficially, to relieve deep-seated organs, by stimulating the vascular and lymphatic circulation through the skin. Thus, in applying poultices for pneumonia we do not make the lung or even the pleura hotter,

but relieve the overloaded blood-vessels and lymphatics.

Further, in treating all superficial inflammations we must guard against anything which increases the injury. Thus, in some skin-diseases, as herpes zoster, secondary inflammation is prevented by an artificial covering of collodion. The benefits of mechanical rest in treating injuries of the limbs need not be more than mentioned ; nor that of physiological rest, wherever it can be obtained, in threatened or existing inflammation of any active organ whatever—for example, the brain, the stomach, or the kidney.

Diet is also of great importance. There is little doubt that the intensity of all inflammations is lessened by greatly diminishing the food taken. In some acute inflammations, especially fevers, we may pursue a different system, with the view of saving the patient's strength ; but it is possible that in the reaction against the starving process, 'the feeding of fevers' may be in the present day sometimes carried too far.

In the treatment of *chronic inflammation* in accessible parts, the first aim will be to bring the tissues and vascular walls into a healthy state. Here we find that metallic and vegetable astringents are most useful. But it may, with the same object, be well to draw more blood into the part, in order that the vascular wall may be better nourished. This is effected by *stimulants*, which are of well-known efficacy in chronic inflammation. Some of these agents are the same as astringents, but in a more concentrated form—nitrate of silver, sulphate of copper, &c. ; others, like iodine and cantharides, are irritants. The local action of certain substances which may be called *aromatics* in healing some kinds of chronic inflammation is most remarkable. Thus, tar, either from wood or from coal, and similar bodies are used in chronic inflammations of the skin ; copaiba, cubebs, sandal-wood, &c., when excreted by the urine, act beneficially in chronic cystitis and urethritis. Benzoin, the tars, copaiba, tolu, and many other aromatic and resinous compounds, when excreted by the lungs, act in the same way on chronic bronchitis. The *rationale* of the action of these drugs is difficult to understand, but they are all oxidisable substances which undergo slow oxidation in the air, often accompanied by an ozonising action. They are also in varying degrees antiseptic or parasiticide. Again, it may happen that it is best to sweep the old tissue away, and allow new vessels to be formed, which will probably have healthier walls. This is effected by destructive *caustics*, as nitrate of silver, potassa fusa, chloride of zinc, or even the actual cautery.

(b) *Indirectly accessible*.—The treatment of indirectly accessible inflammations, or, what amounts to the same thing, the treatment by internal (general) methods even of directly accessible inflammations, is much less satisfactory than the local.

The first indication is to give the part actual rest, mechanical or physiological. The next is to consider if there is any way of reducing the intensity either of the local reaction, or of the fever. Of such means the chief are general blood-letting ; the general application of cold ; and certain particular drugs, such as mercurials, antimonials, purgatives, digitalis, aconite, quinine, and a number more. For the treatment of inflammations of special parts, the articles on these subjects must be consulted, as well as the articles on BLOOD, Abstraction of ;

COUNTER-IRRITANTS ; and HEAT, Therapeutic Use of.

The general treatment of chronic inflammation is based upon the principle that chronic inflammation is, in other words, *imperfect repair*. It is in cachectic persons, or persons with an inherited proclivity (perhaps not yet manifest) to cachectic diseases, that inflammations most tend to become chronic. Hence, the first rule is to improve the nutrition. Many patients with chronic inflammations get well at once when placed in good quarters, and on good food, provided that the digestive functions are first attended to. Next in importance come nutrient tonics, of which cod-liver oil is the chief. There are few chronic inflammations in which it does not do good. Iron is very often valuable ; and, if it fails or is contra-indicated, arsenic may be employed. In chronic inflammations of fibrous tissues, iodide of potassium has a real value, not easily explained.

Finally, a most important means of treating indirectly accessible inflammations must be mentioned, namely, that by *counter-irritants*, or setting up a rival inflammation. In order to relieve an inflammation, for instance, of the knee-joint, we produce a superficial inflammation of the skin. This is most used in chronic, but applies to some acute inflammations also. Various explanations have been given of the undoubted efficacy of this treatment. Some believe the action is transmitted through the nerves ; others that blood is drawn away ; others that the lymphatics are stimulated. The writer's belief is that in the most marked cases of benefit from counter-irritation there is a continuity of the tissue between the inflamed organ and the part where the counter-irritant is applied, and that the action may sometimes consist in drawing away blood ; but more generally the benefit results from setting up currents of plasma through the lymphatics and the connective-tissue spaces. It should be noted that in some inflammations where oedema is a marked feature (e.g. epididymitis), simple puncture has an unquestionable efficacy which may perhaps be explained in the same way. The substances used for counter-irritation are either vesicants or rubefacients, such as cantharides, ammonia, mustard, or iodine. Dry heat at different temperatures may produce the effect of either of these classes. When redness is produced on the skin, it does not follow that hyperæmia alone results. In fact, the desquamation often shows that a low form of inflammation has been established.

J. F. PAYNE.

INFLATION (*inflō*, I blow into).—A term applied, therapeutically, to the method of blowing air or gas into any hollow space. It is employed particularly in connection with the lungs in the process of artificial respiration (*see* ARTIFICIAL RESPIRATION). It is also used for the purpose of dilating the bowel in cases of obstruction, and for purposes of diagnosis in doubtful renal tumours. *See* INTES-TINAL OBSTRUCTION.

INFLUENZA.—SYNON. : Fr. *La Grippe* ; Ger. *Influenza*.

Influenza is an acute specific infectious disease characterised by fever, catarrhal affections of the respiratory and digestive systems, and nervous disorder of various types.

OCCURRENCE.—Numerous pandemics appear to

have occurred in the eighteenth century, and three at least in the nineteenth, beginning in 1837, 1847, and 1889 respectively. A pandemic is generally succeeded by epidemics, and by endemic prevalence in some areas. The disease is said to be endemic in China and in the eastern parts of the Russian Empire, and the pandemic of 1889 would seem to have had its origin in Central Asia (Bokhara).

ÆTIOLOGY.—The history of the pandemic of 1889 has proved that influenza is an infectious disorder disseminated by human intercourse. A specific bacillus was discovered by Pfeiffer in 1892 in the bronchial secretions, in the peribronchial tissues, and in the pleura. It has occasionally been found in the blood during the febrile stage, and, after death, in many organs, as, for instance, in the lungs in pneumonia, in the brain and meninges in meningitis and encephalo-meningitis, in the secretions of the tonsils in pharyngo-tonsillitis, in the accessory cavities of the nose, in the middle ear, &c.

Many of the symptoms and some of the sequelæ, including peripheral neuritis, are in all probability due to toxins produced by the bacillus in the body. The first symptoms of the disease usually develop from two to four days after exposure to infection, and the patient is capable of disseminating the disease throughout the whole course of his attack from the time of onset of the earliest symptoms, until convalescence has been sufficiently established to enable him to resume his ordinary avocations, it may be, even in mild cases, a week or ten days after the commencement of his illness. An attack of influenza does not confer any appreciable immunity, but it is a mistake to assert that one attack predisposes to another. The fact appears to be that the immunity is of short duration.

BACTERIOLOGY.—The *Bacillus influenzae* is a minute rod, 1.5 μ in length by 0.3 μ in breadth, and occurs principally in the sputum, arranged in pairs, or more rarely, in short chains of three or four elements. The bacillus does not stain readily with the usual dyes ; and is completely decolourised when treated by Gram's method. Prolonged staining (12 to 24 hours) in very dilute carbolie fuchsin gives the best results. The organism stains most deeply at the poles, and thereby often resembles a diplococcus. The B. influenzae is a non-motile, non-spore, obligate aërobie, and requires for its successful cultivation the presence of serum, blood, or solutions of hæmoglobin, in its nutrient media. It can only be grown at or near the body-temperature (30° C. to 43° C. ; Optimum, 37° C. ; Thermal Death-point, 60° C.). The duration of life of the bacillus in artificial cultures is limited to three or four days : dried cultures are dead within 24 hours. The B. influenzae is readily killed by desiccation or exposure to direct sunlight. The toxin of the bacillus has not yet been separated.

Cultural Characters.—Upon 'Blood-agar,' small discrete, transparent, raised colonies, resembling drops of water, appear at 18 to 24 hours ; they do not increase in size with age, although the centres sometimes become yellowish-brown in colour. In serum-broth small whitish flakes appear on the surface in 24 hours, but soon sink to the bottom of the tube, and form a flocculent deposit, leaving the supernatant fluid clear. The bacillus is pathogenic for the monkey, and probably for the rabbit also.

MORBID ANATOMY.—The morbid changes found after death from influenza are of a very varied character, and are in the main to be attributed to the

visceral lesions by which death has been brought about. Those due to the generalised disease are of a character common to all forms of acute infective disease—namely, parenchymatous degeneration of the liver, kidneys, and spleen, of the muscular substance of the heart, and of the minute vessels. The last-named may lead to capillary hæmorrhages into the viscera or nervous centres. The spleen may be much enlarged and almost diffuent. In the pneumonia of influenza the influenza-bacillus may be present alone or associated with the *Streptococcus pyogenes longus*, but the *Diplococcus pneumoniae* is present in the majority of cases. In the bronchial sputum these two microbes have also been found, while in the secretion of the nasal cavities the *Staphylococcus aureus* and *albus* and the *Bacillus capsulatus* of Friedländer have also been detected.

Morbidity and Mortality.—The proportion of a population which suffers from the disease at the beginning of a pandemic is high. In 1889–90 it was estimated that in St. Petersburg two-thirds, in Berlin one-third, in London nearly one-seventh of the population was attacked. Smeë estimated that in the years mentioned 8 per cent. of the total population of Europe suffered an attack of the disease, and a very large proportion of those who escaped then have suffered during epidemics which have occurred subsequently. The mortality attributed directly to influenza is low, and has been stated to range from 0·25 to 1 per cent. of persons attacked, but epidemic prevalence of influenza is always attended by a great rise in the general death-rate, which may be thrice the average. On analysis this rise is found to be due to an increase in the number of deaths attributed to bronchitis, bronchopneumonia, pneumonia, and other respiratory diseases, to diseases of the circulatory system, and to an unusual number of deaths among old people. It may be added here that influenza unquestionably tends to diminish the birth-rate. This influence may be in part due to its causing abortion.

SYMPTOMS.—The symptoms produced by influenza, the complications which may occur during its course, and the disorders by which it may be followed vary so greatly in individual cases that it is impossible to give any single picture of the disease. Three main types are usually recognised: the respiratory, the gastro-intestinal, and the nervous, to which ought, perhaps, to be added the febrile (Osler). This classification is not only convenient for purposes of description, but also corresponds to a natural character of the disease, as is shown by the occurrence of epidemics in which the majority of the cases are of one or other type. But it must not at the same time be recognised that this rule is far from being absolute, that cases of any type may occur during any epidemic, that the several types may be represented in the same household at the same time, and that the same patient may suffer from a combination of them, though this is not usual. It may, indeed, be said with truth that there is no organ or tissue of the body which may not suffer during an attack of influenza, though as a rule the brunt falls upon one system—respiratory, digestive, or nervous.

Respiratory Type.—The mildest form of this type, 'simple catarrhal fever' (Peacock), may be considered to be the most characteristic form of the disease; the symptoms to be described are seldom wanting at the beginning of an attack of any form of the disease, and those observed in other types

might, with much justice, be described as complications or sequelæ of the simple catarrhal type. This is true at least of first attacks; in subsequent attacks these early catarrhal symptoms may be little marked, though they are seldom together absent. The onset is usually sudden. The patient feels chilly, perhaps shivers; the surface is pale, and there is a general feeling of discomfort with, perhaps, headache, and ill-defined pains in the limbs. The mouth and nose are dry, and there is some oppression of breathing and a short dry cough. These symptoms are soon succeeded, generally in a few hours, by febrile reaction; the temperature rises to 101°, 102°, or even 104° F., the pulse is rapid and soft, the respiration is hurried, the skin is hot, dry, and flushed. The flush may amount to erythema, and be followed by desquamation. This is especially to be observed on the face, chest, and upper extremities. Within twenty-four hours, as a rule, the conjunctivæ become injected, the throat red and sore, and a thin watery discharge begins shortly to run from the eyes, and to be expelled by the cough which continues frequent; it often recurs in paroxysms which in children may suggest the onset of whooping cough. The respiration is more or less hurried, but physical examination of the chest reveals nothing beyond some harshness of respiration, with perhaps a few sibili or rhonchi on forced inspiration. The tongue has a white fur usually confined to the central parts, leaving the tip and edges red and irritable; there is thirst, almost complete loss of appetite, and, as a rule, constipation. The urine is scanty and high-coloured. The patient is restless and may be mildly delirious at night. During the day some patients are restless and excited, eager to do their usual work, if of an intellectual kind; others are listless and depressed; and all are very quickly fatigued. Epistaxis is not uncommon. After an interval of one or two days the temperature declines, the skin becomes moist, the conjunctival injection passes away, the secretion from the respiratory passages becomes opaque or muco-purulent, the tongue becomes clean, the appetite returns, and the patient is convalescent. The duration of the whole attack is on an average three to five days, but it may be extended to seven or ten days. The symptoms, then, of this form do not differ in any characteristic way from those of severe coryza or 'common cold,' and bacteriological investigations appear to confirm the opinion that during epidemic or endemic prevalence of influenza, some illnesses are attributed to it, which are in fact due to pyogenic infection (streptococci). As a rule, however, the frontal headache and the pain in the eyes and limbs are more marked in influenza than in coryza, and as a rule, also, the degree of physical and mental depression left after the fever has passed is much greater. Some patients are liable for weeks or months to profuse perspirations on slight exertion or at night, and others only very slowly regain their appetite and strength, so that the standard of general health remains 'below par' for a long period.

In more severe cases of this type symptoms and physical signs of bronchitis or pneumonia become prominent from an early stage, and the duration and course of the disease are determined by the progress of these complications. There is nothing characteristic about the bronchitis, unless it be that in some cases the expectoration is very copious, at first thin and clear, but containing later purulent

masses; Pfeiffer considers greenish-yellow nummular expectoration as characteristic, but it cannot be very common. In many cases, probably in a majority, the bronchitis is not specific, the catarrh produced by the specific infection opening the way to the ordinary infection of bronchitis. The severity of the bronchitis differs very greatly; there is seldom any danger except in old persons, or the subjects of chronic bronchitis. Pneumonia, of one or other form, is the most common of the serious complications of influenza, and is the main cause of the large mortality directly attributable to the disease. Very different opinions have been expressed as to the true nature of this complication. The fact appears to be that its pathology differs essentially in different cases. In some cases the inflammation of the lung is due to the specific infection; in others influenza is merely the determining cause of croupous or catarrhal pneumonia produced by the ordinary infective agents of those disorders. True specific influenzal pneumonia is lobular, and produced, as some think, by extension from the bronchi, but this sequence of events is not always to be discerned. It may, especially in the old, creep about from one part of the lung to another, or from one lung to the other, so that the physical signs vary in extent and situation almost from day to day. The duration of such cases is indefinite: apparent recovery may be followed again and again by relapse, and the patient's strength may thus be exhausted rather by the duration than by the extent or the severity of the pulmonary inflammation. Such cases may be seen also in young children; but in adolescents and adults the type is usually sthenic, and a whole lobe may be rapidly involved. Even in such cases the course is usually prolonged and irregular, and there is seldom any distinct crisis. The symptoms are not characteristic, the sputum is only rarely rusty or blood-stained, and usually becomes muco-purulent at an early stage. In favourable cases, after some ten or twelve days, the lung begins to resolve, the expectoration becomes more copious and purulent, and the patient is left in a depressed state to a long convalescence. In some cases the prominent symptoms are those of pleurisy, but as a rule in such cases they are succeeded by or combined with those of localised pneumonia, and much effusion into the pleural cavity is unusual. Lastly, influenza is very apt to become complicated by acute croupous pneumonia, the symptoms of the initial disorder running on into those of the latter. *See LUNGS, Inflammation of.*

Gastro-intestinal Type.—This type has been observed frequently during some of the epidemics which have occurred since 1889, but on the whole less often than the respiratory or the nervous. The onset is usually sudden, and to the symptoms observed in the milder cases of the respiratory type are added pain in the epigastrium, tenderness in various parts of the abdomen, nausea or vomiting, a coated tongue, and great depression, with often a dusky flushing of the face and chest. Epistaxis is not uncommon; enlargement of the spleen occurs in many cases, and is in proportion to the intensity of the fever. The tongue presents at first a uniform white fur, which often has a characteristic 'porcelain' appearance. This clears in patches on the second or third day, leaving the tongue red and irritable, or it is succeeded by a thin irregular brown coating accompanied by dryness. Mean-

while, diarrhoea has usually commenced. It is often accompanied by much painful colic, and the stools are loose and contain much mucus. The duration of such attacks is very various. In some cases the symptoms, with care and suitable diet, pass away in a few days; in others the temperature remains a degree or two above the normal, anorexia is complete, and thirst becomes troublesome. The patient complains much of abdominal pain, often of headache, and is sometimes mildly delirious at night, so that the condition simulates enteric fever. The conjunctivæ are injected, and along with the skin present a sub-icteric tint; in some epidemics well-marked jaundice has been common. In other cases, again, the fever subsides after two or three days, and appetite returns to some extent, but the digestive powers are much enfeebled, the stools continue loose and light-coloured, or if formed are pale and offensive, and of low specific gravity. This condition may continue for weeks, and the least indiscretion in diet may cause a relapse after apparent recovery. Cases are sometimes met with in which the most prominent symptoms at the onset are fever, bilious vomiting, constipation, and pain in the right iliac fossa, so that the case presents a great resemblance to appendicitis. It is, indeed, possible that the appendix is actually involved, and some would explain the increased frequency of appendicitis at the present time by the theory that it is a complication or sequela of influenza.

Nervous Type.—This type was exceedingly common during the last pandemic, and is frequently encountered in the epidemics which have succeeded it. Individual cases vary very much in severity, but in an average example the patient after a slight chill is seized with pain in the eyeballs, frontal headache, and pains in the back and limbs; the backache is in many cases very severe, and has been compared by women who have borne children to that experienced during childbirth. The temperature rises rapidly, reaching its maximum, in a large proportion of cases, within twenty-four hours; the rise is generally limited to 2° or 3° , but temperatures of 104° F., and even higher, have been noted. As the temperature rises the pulse becomes more frequent. It is soft, often feeble, and some cyanosis is not uncommon during the febrile period. As this passes away various disturbances of the cardiac rhythm, due apparently to affection of the nervous apparatus of the heart, may be observed. The most characteristic is a remarkably slow rate (50 or less). In other cases the heart becomes irregular or intermittent, and there may be attacks of palpitation, which cause the patient much alarm and distress. In cases presenting either form of disturbance of cardiac rhythm syncope may occur, and may be fatal. Attacks resembling angina pectoris have also been observed, and sudden death may be brought about by myocardial degeneration. The duration of the febrile period varies from one to nine or ten days, but is usually three, four, or five days. The maximum is, as a rule, only maintained for a few hours. The fall is sometimes rapid: a distinct crisis, accompanied by profuse sweating, occurs, and the temperature declines to the normal, or even lower, in twelve hours. In other cases the fever has a remittent character, with regular daily fluctuations by which the temperature returns to the normal in two or three days.

Though it is usual to observe a well-marked rise

of temperature, it is important to recognise that no increase in the temperature may ever be detected, although the other symptoms of the attack may be fairly well marked and the sequelæ serious. During the acute stage the patient experiences great depression and lassitude—symptoms which persist after the pain and pyrexia have disappeared, and are often aggravated by insomnia; in some cases convalescence is prolonged for weeks, or even months, and occasionally well-defined melancholia develops. The prostration is accompanied by anæmia.

Among the more severe cases of this type two clinical forms may be distinguished, the comatose and the delirious (Judson Bury). In the former the patient, with or without recognised symptoms characteristic of the onset of influenza, gradually becomes drowsy and apathetic; he answers questions with difficulty, and in a few days becomes comatose. Recovery may occur, but a fatal termination is, according to Bury, more common. Examination of the brain, *post mortem*, may reveal nothing abnormal, or there may be congestion of its surface, or a purulent meningitis, with or without encephalitis, which is usually hæmorrhagic. In cases that recover, and in those in which no morbid changes are found *post mortem*, it must be assumed that the symptoms were produced by cerebral toxæmia, or that any definite lesions which existed were slight or temporary. The meningitis may be secondary to suppuration in the middle ear, or nasal cavities, but may occur independently. Cerebral abscess, when it occurs as a complication of influenza, would appear to be almost invariably secondary to otitis. In the other severe form, the delirious restlessness, irritability, delirium, and even mania are the essential features, or delirium may alternate with a melancholic condition. Spinal meningitis may accompany the cerebral meningitis, but has been known to occur alone also.

Of the nervous disorders which may occur after an attack of influenza, but are perhaps particularly prone to ensue on the nervous type, it can only be said that they include inflammation, degeneration, or functional derangement of every part of the nervous system. Thus in relation with the brain we may have cerebral hæmorrhage, embolism, and thrombosis of arteries, veins or sinuses, meningitis, or encephalitis, dementia, mania, melancholia, hysteria, epilepsy, catalepsy, and neurasthenia. In relation to the spinal cord we may find myelitis or degeneration of any of its various tracts, and disseminated hæmorrhagic myelitis has also been recorded. Finally, to quote Bury again, 'we may have neuralgia or neuritis in the territory of nearly every cerebral or spinal nerve as well as the different forms of multiple neuritis.' Peripheral neuritis may come on days, or even weeks, after the acute symptoms of the febrile stage have ceased. There is pain in the affected limbs, with weakness of certain muscles, dyæsthesia, with reflexes at first increased but afterwards diminished, though never, it is said, abolished. All four limbs may be involved, or two on the same side, or one only. The muscles most often affected are, in the upper limb the scapulo-humeral, in the lower the anterior muscles of the leg and the flexors of the knee. In severe cases the muscles waste rapidly, and may present the reaction of degeneration. In other cases symptoms of disorder of the bulbar nuclei or of their nerves occur with or without derangement

of spinal nerves. The symptoms resemble those due to diphtheria, but are much less regular in their distribution. Paralysis of any of the external ocular muscles may occur either alone or in various combinations. Paralysis of accommodation or transitory dilatation of one pupil may be observed either as an isolated phenomenon or as a part of a more wide-spread affection. Affections of the optic nerve are rare; the most frequent is retro-bulbar neuritis, but papillitis and neuro-retinitis have been observed. Pharyngeal and laryngeal palsies have also occurred, but are uncommon. Loss of the sense of smell and impairment of taste are not uncommon sequelæ, and may persist for long periods. The prognosis in post-influenzal neuritis is good on the whole, even in cases in which the paralytic symptoms are extensive, and the atrophy well-marked; but recovery may be very tedious.

Febrile Type.—A febrile type may conveniently be recognised to contain those cases in which the patient suffers from a febrile paroxysm or from remittent or continued fever with the usual concomitant symptoms, but without catarrh or localised nervous symptoms. In one form of this type, which is perhaps more common in children than in adults, the patient complains of malaise, and the temperature is found to be 101° or 102° ; the thermometer rises rapidly to 103° , 104° , or even 105° , and the patient suffers from headache and is drowsy. In severe cases the drowsiness may deepen to stupor. As a rule the temperature falls as rapidly as it rose, and the whole attack may be over in twenty-four hours, leaving perhaps some depression for a few days. In other cases the temperature, without attaining so high a point, remains for many days or some weeks either of the continuous or remittent type. Cases of the former kind may simulate enteric fever, and it is important to remember that influenza may be immediately succeeded, without defervescence, by enteric fever, the disturbance produced by the former having so diminished the resisting power that the infective agent of the latter, previously present in the body or in the environment, is enabled to develop.

COMPLICATIONS AND SEQUELÆ.—Many of the systemic disorders already described might be regarded as complications of the primary fever, but bacteriological evidence appears to confirm the view, founded on clinical observation, that they are more justly considered to be symptoms of the disease and produced directly by the specific virus. At the same time it must be recognised that an attack of influenza may have a most injurious effect upon chronic maladies, so that pulmonary tuberculosis, for instance, which had become quiescent, may again become active, or that the way may be opened to secondary infections producing for instance otitis, suppurative rhinitis or pharyngitis, or greatly aggravating a tendency to chronic bronchitis. The list might be indefinitely extended. Attention will only be directed to a few of the more serious and frequent complications and sequelæ to which reference has not already been made.

Insanity is said to be a frequent sequela of influenza. See INSANITY IN SPECIAL DISEASES.

Albuminuria is observed in a considerable number of cases. It may come on during the acute attack, and endure for several weeks or months. There may be merely a trace of albumen at an early stage, with a microscopical amount of blood, and hyaline casts containing a few blood-cells or granules,

or small granular casts, in the centrifuge-deposit. In other cases acute degeneration of the kidney, acute nephritis with in some instances involvement of the glomeruli, or acute hæmorrhagic nephritis occurs. Chronic diffuse nephritis is occasionally a sequel of influenza, and cases of so-called functional albuminuria are aggravated.

Arthritic pains are common during the acute attack, and may be accompanied by tenderness and swellings. These affections are commonly spoken of as rheumatic, but it may be doubted whether they have any relation to rheumatism, though pericarditis and endocarditis have been noted as occasional complications of influenza. Phlebitis, especially of the smaller veins of the foot and leg, is not rare as a sequel of influenza, and occurs perhaps especially in persons of a gouty diathesis.

The *cutaneous* lesions which have been attributed to influenza are numerous. Reference has already been made to the erythematous rash sometimes observed at the onset; occasionally a fine papular rash has been seen at the same period. Herpetic eruptions affecting the lips or other regions occur with considerable frequency. Purpura has also been observed. The erythematous and papular rashes are usually succeeded by fine desquamation, but occasionally a remarkable desquamation in large sheets has been observed.

Stomatitis may complicate the respiratory or gastro-intestinal form and cause a great deal of distress. It is usually of the so-called follicular variety, the primary lesion being quickly followed by ulceration which may extend until large areas are continuously involved.

RELAPSES AND REPEATED ATTACKS.—It has been estimated that a relapse occurs in 10 per cent. of all cases; it is at least very common. After four to six days of freedom from fever the temperature again rises and all the symptoms recur. As many as three such relapses have occurred. Second attacks are very common, and it is certain that the immunity produced by an attack lasts only a short time. It has sometimes been observed that a person who has suffered from one type of this disease during one epidemic suffers from another during the next, even though the prevalent form may be of a different type; but no rule can be stated.

DIAGNOSIS.—Except during the prevalence of a well-marked epidemic the diagnosis of influenza may be exceedingly difficult, and must usually be reached by a process of exclusion. A slight attack, particularly if of the respiratory type, may pass almost unobserved, the symptoms being attributed to 'a common cold,' but its true nature may be made apparent by the mental and nervous depression which succeeds.

In the early stage of an attack of the catarrhal type, the coryza, headache, and fever may, especially if there be discrete erythema of the face and chest, lead to a suspicion of measles; in other cases where pharyngitis is a prominent symptom and erythema is extensive, the case may for a short time present a considerable resemblance to scarlet fever, but in either a few hours' observation will usually make clear the diagnosis from measles or scarlet fever. Occasionally pharyngitis due to influenza is attended by false membrane on the tonsils. This is usually limited to the mouths of the follicles, at least at first, and is then semitransparent and easily detached. Bacteriological examination showing the absence of the Klebs-Löffler

bacillus will confirm the diagnosis. The resemblance which cases of the gastro-intestinal type may present to enteric fever has already been noted. The absence of characteristic stools, of the enteric exanthem, and later on of Widal's reaction, will usually allow a diagnosis to be made.

PROGNOSIS.—The prognosis in childhood and maturity of cases uncomplicated by pneumonia is always good. In aged persons, in those prematurely old, especially if the subjects of emphysema and chronic bronchitis or alcoholism, the prognosis is more serious, as in them complications, in the one instance great aggravation of the bronchitis, in the other cardiac failure, are apt to develop. In any case the prognosis must depend to a very great extent upon the early enforcement of suitable treatment.

TREATMENT.—The most important point in the treatment of influenza is to induce the patient to recognise that the affection may easily become serious, and to persuade him to remain in bed during the acute stage; to this some would add two days in bed after the temperature has become normal, and the precaution is a wise one, at least in persons who are past middle life or prone to bronchitis. It is often difficult to induce a vigorous adult to give up work early enough, as at the onset there is commonly a good deal of excitement and restlessness. The advice should, however, be strongly enforced, for a patient who fights against the disease in its early stage is more likely to suffer from severe pulmonary complications, while a too early return to work after the subsidence of acute symptoms or neglect of precautions against cold and over-exertion during convalescence may lead to relapse or to various pulmonary or nervous sequelæ.

The best routine remedy for the relief of the muscular and joint-pains and backache, with which the disease so commonly commences, is probably sodium salicylate, though phenazone or phenacetin is useful, and perhaps preferable when headache is severe. If there is nausea the drug selected should be given in an effervescent mixture. Quinine may also be prescribed with advantage in this stage, as well as later on in cases in which the temperature becomes or continues elevated or of intermittent type. In attacks of moderate severity the pains in the back and limbs diminish, or disappear after the patient has grown warm in bed, and reaction may be encouraged by hot drinks. When restlessness or insomnia persists after relief of pain, bromides may be prescribed, and in weakly individuals ammonium bromide should be selected. In such persons and in children depressing remedies should be avoided. A great number of other drugs have been highly praised from time to time, among which sodium benzoate and carbonate, cinnamon, carbolic acid, and phenocoll hydrochlorate may be mentioned, but none appear to have come into general use.

It is inadvisable to give a purgative in all cases at the commencement of treatment, though there need be no hesitation in doing so if there be constipation. Calomel followed by a saline draught, and in weakly persons and young children castor oil, are the best drugs to use for this purpose. Where the symptoms point to an attack of the gastro-intestinal form it is wiser as a rule to eschew purgatives and to prescribe a simple enema or at most a moderate dose of castor oil. The diarrhoea characteristic of this form is often very difficult to treat, and for some

weeks any solid food taken into the stomach is followed by painful intestinal peristalsis and an evacuation, even though the appetite may have been in great measure regained. The administration of digestive ferments, of acids and alkalis, of bismuth, or of sedatives commonly has little effect, and the only effective treatment is to direct the abdomen and thighs to be warmly covered and to reduce the diet to broths, soups, and milk thickened with corn-flour or fine oatmeal and spiced. As soon as these can be taken without subsequent discomfort, additions may carefully be made to the diet, the patient's sensations being the best guide.

When convalescence is established tonics should be given, of which the best is strychnine, and when depression is marked the amount prescribed should be increased until a full dose is taken. The diet should be simple and nutritious, and when the weather and social conditions are favourable change of air should be recommended, but not otherwise, as rest, quiet, warmth, and ease are among the chief necessities.

It is not proposed to discuss here the treatment of the pulmonary and nervous affections accompanying or following influenza, as this may be best considered in the articles on BRONCHITIS; LUNGS, INFLAMMATION OF; NEURITIS, &c.

The *prophylaxis* of influenza when the disease has become thoroughly established in a community is difficult. If the first case in a household can, in the earliest stage, be isolated by being confined to a room with a nurse in attendance, the dissemination of the infection to other members of the household will probably be prevented; and if this system could be applied generally throughout a community much might be done to prevent the development of an epidemic. But in practice the earliest cases are rarely isolated, either because the patients consider that they are suffering from 'a common cold' or because they refuse to remain in bed, or even to refrain from going about their ordinary business. Much may however be done to protect old and delicate persons from risk of exposure to infection.

DAWSON WILLIAMS.

INFUSION.—See SALINE SOLUTION, Infusion of.

INHALATIONS, Therapeutic Uses of (*inhalo*, I breathe in).—Inhalation is a method of applying remedial agents to the respiratory tract, whereby these substances in a gaseous or atomised form are brought into contact with the mucous membrane of the nose, mouth, pharynx, larynx, and bronchi, and may even penetrate to the epithelium of the air-cells. The examination of the lungs of colliers, grinders, and others engaged in dusty occupations has shown that the inhaled dust can be detected in the lung-tissue, where it induces chronic pneumonia. The experiments at the Académie de Médecine proved that medical sprays are equally penetrating.

METHODS.—The modes of inhalation vary with the drug used, depending mainly on the temperature at which it volatilises, and also on the medicinal effects aimed at.

Chloroform, ether, bichloride of methylene, and nitrite of amyl evaporate at ordinary temperatures, and only need to be diluted with air to be safely inhaled. Calomel and sulphur are sublimed at high temperatures in special apparatus; but the majority of drugs are best vaporised through the medium of

hot water or steam, or reduced to fine spray by passing compressed air through their solutions.

Many forms of inhaler are in use, but in selecting one for warm inhalations the requisites are—(1) that it can be used without difficulty by the patient; (2) that a temperature of 130° to 150° F. can be steadily maintained; (3) that the steam be thoroughly impregnated with the medicament; and (4) that the inhaling-tube be fitted to the nostrils as well as to the mouth, so as to ensure a sufficient supply of the inhaled vapour. Lee's steam-draught inhaler is especially useful for projecting a full current of steam into the throat and air-passages. The force and temperature are alike controlled by the sliding valve on the inhaling-pipe. When no inhaler is at hand, a jug with a wide mouth, and half filled with hot water, may be used, a towel being placed round between the mouth and nose of the patient and the opening of the jug, to prevent the escape of the vapour into the air.

VARIETIES AND USES.—Inhalations are employed chiefly in diseases of the pharynx, larynx, and air-passages, and may be classed as *sedative*, *stimulant*, and *antiseptic*.

Sedative.—Steam is soothing to the throat, when this part is dry, inflamed, or irritable. In incipient laryngitis and croup, as well as in irritable bronchitis, the inspiration of steam from specially arranged kettles is very grateful, the moist vapour promoting secretion and expectoration.

Jets of steam are used in hospitals and bathing establishments as vehicles for the inhaled drugs, and are directed into the patient's mouth.

A sedative inhalation made of one part of chloroform and two or three of rectified spirit—of which one teaspoonful may be added to a pint of water, at 60° to 100° F.—is much commended in hay-fever and laryngeal spasm. It must be remembered that chloroform is very powerful when given with steam as an inhalation. One drop of chloroform in a pint of water at 150° may produce giddiness.

Stimulant.—Stimulating moist inhalations can be prepared with various volatile oils. Oils of pine and of cubebs are useful stimulants in cases where there is much secretion from the throat and air-tubes. Thus 2 drachms of oil of pine or oil of cubebs may be mixed with 60 grains of light carbonate of magnesium in 3 ounces of water; and of this mixture 1 drachm may be used in a pint of water at each inhalation. One drachm of the compound tincture of benzoin, in half a pint of hot water, is often useful in chronic bronchitis and laryngitis. Oil of turpentine or the Vapor Olei Pini Sylvestris of the Pharmacopœia makes an excellent stimulant inhalation in cases of dilated bronchi.

Antiseptic.—Antiseptic inhalations are used where the object is to correct a fetid secretion, as well as to stimulate the secreting membrane to fresh action. In fetid bronchorrhœa, in gangrene of the lung, in fetid abscess, and in pyo-pneumothorax, benefit is derived from a vapour of creosote or of iodine, or from glycerine of carbolic acid (2 to 3 drachms to a pint of boiling water), or again from oil of thymol, prepared like the other essential-oil vapours, with light magnesia (60 grains to 3 ounces of hot water). Carbolic-acid vapour can be well diffused in a room by mixing the acid with water in Lee's steam-draught inhaler, or by dropping the acid on a hot metallic plate. Creosote or eucalyptus oil, mixed with an equal quantity of ethylic alcohol, is often employed in the form of drops (10 to 20 drops

at a time) on a small sponge placed in a perforated zinc respirator. The respirator thus charged is worn by the patient for one hour morning and evening, with a view to improving the state of the lungs. After a free morning expectoration the respirator-inhaler should be used, and instruction given to inspire deeply by the mouth and expire through the nostrils with the mouth closed.

Antispasmodic.—The fumes of burning nitre-paper are employed as inhalations in cases of pure spasmodic asthma uncomplicated with bronchitis. The paper—prepared by soaking white blotting-paper in solution of nitrate of potassium (30 to 40 grains in an ounce of water)—is sometimes washed over with tincture of benzoin, and this, in certain cases, may be an improvement; but in ordinary forms of asthma the nitre-paper, burnt till the patient is enveloped in smoke, will usually relieve the asthmatic paroxysm. The fume of a grain of powdered opium volatilised on hot metal has been praised as a remedy to cut short nasal catarrh; as well as smoking solid opium in the Chinese fashion through a pipe, in spasmodic asthma. Cigarettes, pastilles, and powders containing stramonium, belladonna, and other antispasmodics, are well known as remedies in spasmodic asthma.

Inhalations of atomised spray have of late years come into deserved repute as valuable aids to the treatment of chronic diseases of the throat and lungs. The principle of the hand-ball and steam-spray atomisers is that, if two capillary tubes are placed at a certain angle to each other, one dipping into a fluid, while through the other a stream of air is driven by heat or compression, a vacuum is formed in the first, causing the liquid to pass out in the form of fine spray. In using these instruments, the operator should seek to blow the spray into the patient's throat at the time when an inspiration is being taken, as thus the spray will obtain a free entrance through the larynx into the trachea. It is doubtful if much spray enters the air-tubes; some certainly does, as has been stated above, but the cold sprays do not appear to afford so much relief to affections of the lungs as the warm inhalations. In cases of tumidity of the larynx, a spray containing 10 grains of alum to 1 ounce of distilled water may be used. In place of alum, 2 grains of sulphate of iron, 5 grains of sulphate of zinc, or 5 grains of dry perchloride of iron, in 1 ounce of water, may be employed. For antiseptic purposes, 5 minims of sulphurous acid, or 1 minim of carbolic acid, to 1 ounce of water, or a like quantity of solution of iodine, may be used. In putrid sore-throat and diphtheria the writer has seen excellent results from iodine, either inhaled in vapour or applied in solution. As a styptic and hæmostatic spray 50 to 200 grains of tannic acid are employed, dissolved in 10 ounces of water, but for relaxed throat a weaker solution is useful. For sedative purposes a solution of bromide of ammonium, or one containing half a grain of acetate of morphine to 1 ounce of water, may be employed. Diluted ipecacuanha-wine spray is said to be very efficacious in relieving the dyspnoea of chronic bronchitis and emphysema. This spray in a few instances may induce vomiting, but this accident may be obviated by diluting the wine with a considerable proportion of water. All these spray-solutions should be filtered through paper, otherwise they may contain small particles which clog the aperture of the atomiser. At the Continental spas it is usual to

medicate large chambers by means of sprays and vapours, in which patients can sit for hours breathing the artificial atmospheres; and in this way various mineral waters, such as those of La Bourboule, Aix-le-Bains, and Cauterets, are locally applied.

An inhaler should give a cloud of fine spray. This can be ensured by driving the spray against the sides of a strong glass globe with such violence that it is broken into very fine particles, and these can be deeply inhaled through the mouthpiece without causing irritation, and often with very great comfort to the patient.

Insufflation is a method of applying powders to the larynx. A vulcanite tube curved at a suitable angle is carried over the tongue to the laryngeal aperture, and then the powder which has been introduced through a small opening covered by a slide in the tube, is blown into the larynx and trachea. The blowing is effected sometimes by an elastic ball at the end of the tube, at other times by the mouth of the operator. In laryngeal phthisis $\frac{1}{20}$ to $\frac{1}{4}$ of a grain of hydrochlorate of morphine, mixed with sugar of milk, may thus be blown into the larynx, and often with great comfort to the patient. A grain of iodoform and a grain of boric acid are often added to the morphine powder.

JOHN C. THOROWGOOD.

INOCULATION (*in*, into; and *oculus*, a bud, a graft).—As a medical term, inoculation is either an operative procedure or an accidental occurrence, by means of which infective materials are brought into direct contact with the minute vessels of the skin or of a mucous membrane, or with those of the subcutaneous or submucous tissue, so that they are readily and speedily absorbed. See INFECTION; IMMUNITY; VACCINATION; HYDROPHOBIA; PLAGUE; and TYPHOID FEVER.

INSANITY.—SYNON.: Fr. *Folie*; Ger. *Geisteskrankheit*; *Geistesstörung*.

The articles arranged under this heading comprise the following:

I. General Considerations regarding ætiology, symptoms, diagnosis, prognosis, and treatment, p. 747.

II. Pathology and Morbid Anatomy of Insanity, p. 751.

III. Developmental Insanity, p. 757.

IV. Insanity of Pregnancy, the Puerperal Period, and Lactation, p. 758.

V. Senile Insanity, p. 761.

VI. Delusional Insanity, including Paranoia and Monomania, p. 763.

VII. Imperative Ideas, p. 765.

VIII. Impulsive Insanity, p. 765.

IX. Mental Stupor and Catatonia, p. 766.

X. Moral Insanity, p. 767.

XI. Insanity in Special Diseases, that is, Insanity occurring in the course of ordinary Medical Diseases, p. 768.

The following is a list of articles relating to this subject arranged under their respective headings in other parts of the Dictionary: ALCOHOLISM; CATAPLESY; CHILDREN, TRAINING OF; CIVIL INCAPACITY; CLIMACTERIC INSANITY; CONSCIOUSNESS, Disorders of; CRETINISM; CRIME, IRRESPONSIBILITY FOR; DEMENTIA; DIPSOMANIA; EPILEPTIC INSANITY; FEIGNED DISEASES; FOLIE CIRCULAIRE; FOLIE À DEUX; FORCIBLE

FEEDING; GENERAL PARALYSIS OF THE INSANE; HYSTERICAL INSANITY; IDIOCY; LUCID INTERVALS; LUNACY, LAW OF; MANIA; MELANCHOLIA.

I. General Considerations.—For the healthy function of the brain it is necessary that all the component portions shall be in a sound condition working harmoniously as a perfect whole. The grey and the white matter, the blood-vessels, lymphatics, and neuroglia all play important parts, and must be free from defect. They must not be stunted or arrested in development, the blood must be of healthy quality, not too much or too little, not contaminated by alcohol or other poisons. The brain-cells must not be overworked or damaged by blows, heat, or organic lesions. For the *sana mens* there must be the *sanum cerebrum*.

ETIOLOGY.—**I. Predisposing causes.**—**Heredity.**—The one great predisposing cause of insanity is an inherited tendency to neurotic disorder. Though all writers recognise this tendency, they vary much with regard to the importance to be attached to it and in the statistics they give, one observer attributing ten per cent. of cases to this cause, another no less than ninety. The discrepancy arises from the fact that some take into account insanity only and not other neurotic disease such as epilepsy or dipsomania: one man reckons only those whose insanity is inherited in the direct line from father or mother, while another looks not only to the direct line, but also to the collateral branches. The difficulties of the investigation are enormous when we go back even to three generations. The history of two grandfathers and two grandmothers is hard to ascertain with anything like exactness, but to go further back and examine the records of four great-grandfathers and four great-grandmothers is well-nigh hopeless. No accurate information can be got by questioning, for such questions are evaded, or a direct negative is given, whatever be the facts. Only from our own observation can we form conclusions, and this is limited to a few generations. In families where insanity is known to exist, many of the members are sane and remain sane all their lives. We require to know why some escape and others break down, not only in insanity but in epilepsy, hysteria, hypochondria, neuralgia, dipsomania, or other neurosis. The tendency is not eliminated by those who themselves escape insanity, for by reason of *atavism* or *latency* it may reappear in the children of parents who have shown no symptoms. This has a grave bearing on the marriage of cousins or other persons in whose family insanity has developed. It is essential that all the predisposed should marry healthy partners whose family history is good, and who themselves are free from neurotic disorder or threatenings thereof.

In many persons the inherited predisposition or tendency is manifested not by insanity but by eccentricity of various kinds, so that they differ from ordinary members of society and are badly adjusted to their environment, not so completely out of harmony as to require restraint, but always on the verge of becoming so. They may go through life in this condition or break down, eccentricity merging into insanity. They form the large class of borderland cases, and are often extremely difficult to deal with. Many drink, and their mental state is ascribed to drink; but frequently the drinking-habit is an inherited neurosis. They all display

the predominance of the egoistic or subject-consciousness which is such a marked feature both in the insane and those in the borderland. Some evince an intense selfishness shown in penurious habits, a desire to amass wealth for which they stint themselves and families. If anything goes wrong with their affairs and they experience a loss however trifling, they deplore their misfortunes, say they are ruined, and become melancholic. Others live in a state of constant suspicion, suspect everything and every one, their medical man, their clergyman, or lawyer. They think their tradesmen are trying to cheat them, and will listen to all the gossip of servants or the like. A third group make life miserable by intense jealousy; even the young will display this egoism, and are jealous of any one who comes between them and their friends. In married life incredible jealousy is found in those who pass as sane. Men and women make their lives intolerable without the faintest ground. Other people live in a state of perpetual fear and apprehension, take a gloomy view of everything, and continually think that something amiss is going to happen. Others are hypochondriacs, spending their lives in the vain search for remedies for imaginary ailments, neglecting their business, family, and friends.

Some of these eccentrics drift into insanity, but many persons develop symptoms of the disorder without any previous peculiarity or warning. The time of life is held to be accountable for it in certain cases, and there has been described an insanity of *puberty* and *adolescence* which above all is due to hereditary taint (see INSANITY, DEVELOPMENTAL). The period between 20 and 30 is far more productive of insanity than that preceding, but between 30 and 40 the liability is greatest, for this is the time of the highest development and specialisation of the brain-centres, a time when an inherited weakness may assert itself, and when also an *acquired* insanity may arise from overstrain, alcohol, or syphilis, which at an earlier age do not play so important a part. Yet the insanity of this period has received no name. As age advances the insane cases are less frequent, but the *climacteric* is a time of danger (see CLIMACTERIC INSANITY). Old age has also much to do with the causation of insanity, but if we eliminate the cases of mere mental decay and degeneration, we shall find that heredity is often the chief cause, and in many has produced previous attacks (see INSANITY, SENILE). Heredity is a very important factor in the production of insanity during pregnancy, the puerperal period, and lactation. See INSANITY OF PREGNANCY, the PUERPERAL PERIOD, and LACTATION.

Sex.—That females preponderate in our asylums is certainly owing to the lesser mortality, for they are much less liable to fatal brain-disease. Probably the difference in the number of the two sexes who become insane is not very material, but statistics help us little, as only those of new cases can determine the question.

Age.—The time of life at which most insanity occurs is that of maternity, between thirty and forty years of age; it then decreases with each decennial period. The time of the highest development of the brain-centres is that in which they are most prone to disorder, the time when an inherited weakness is most likely to manifest itself, and when the last and least organised structures are most liable to disturbance and loss of equilibrium.

II. Exciting Causes.—These have been divided into *physical* and *moral*, and by *moral* is meant *mental*. The *physical* are, in the opinion of many, toxins either derived from without or generated within the body. Such toxins as those of rheumatism or gout are easily recognisable, and the relationship between them and insanity is unquestionable. Disease of the kidneys is known to be connected with brain-disorder and causes uræmic convulsions and uræmic coma; diabetes may end in coma, and disturbance of the liver may permit toxins from the intestinal canal to reach and affect the brain. So also the toxins of fevers, of typhoid fever or scarlatina, of influenza or pneumonia may light up insanity in some, and ordinary delirium in others. What the connection may be between the uterus and the brain is a disputed point, and it may be doubted whether puerperal insanity is always due to sepsis as some hold, while other toxins depending on disease or disorder of bodily organs have yet to be defined. Besides these autotoxins from within there is a large mass of insanity caused by poisons from without which is due to the vices of civilisation and is preventable.

Alcohol.—Alcoholism probably plays a larger part in the causation of insanity and an insane inheritance than any other factor, and this is the reason why cerebral disorders increase among the lower rather than the upper classes. See ALCOHOLISM.

Syphilis.—Among the preventable causes of insanity syphilis holds a prominent place, and its importance is emphasised daily by the researches of pathologists. General paralysis of the insane is mainly due to syphilis, acquired or inherited. Many other brain-disorders and lesions of the cerebral vessels have their origin in syphilitic disease.

Lead, bang, opium, cocaine, &c.—Among the preventable causes of insanity must be ranked lead-poisoning, such cases occurring principally in manufacturing districts (see LEAD-POISONING). Many recover, but some drift into dementia, having gone through various stages of mania or melancholia. In the East or wherever Orientals are gathered together bang, hashish, or Indian hemp is a fertile source of insanity. The Superintendent of the asylum at Cairo reports that in 41 per cent. of his male patients bang alone or combined with alcohol caused the disease. Drugs like opium, morphine, cocaine, chloral hydrate, and other hypnotics may bring about a habit which amounts to a disease, but if the poison is removed actual insanity is rarely found.

In the search after toxins we must not forget that there are other and equally potent causes. These have been called *moral*, but a better name for them is *mental causes*. They bring about insanity, not by toxins but by disturbance of the brain-circulation and vessels, by alteration of the blood-supply, not in the quality but in the quantity. We know that mental excitement, even when pleasurable, prevents sleep by producing hyperæmia. In health this quickly subsides, but if long-continued worry and overstrain render this sleeplessness constant, chemical changes are set up and the brain-cells are damaged by fatigue as those of the spinal cord are by overwork and strain. We cannot disregard the importance of such hyperæmia leading to congestion and the accumulation of the products of waste.

Sudden fright or shock may bring about rapidly the changes which are slowly caused by long-con-

tinued strain. How the circulation is affected by such events is shown by the fact that occasionally sudden shock will produce cerebral hæmorrhage.

Stroke and traumatism.—Many break down in India and are said to have had sunstroke. A better name is heatstroke, for the cause of the insanity is exposure to the heat of the hot season, night and day, and consequent pneumonia. *Traumatic* insanity may be due to a blow causing organic change, or to shock. In the latter case hereditary predisposition is almost certain to be found.

Epilepsy.—A considerable amount of insanity is due to epilepsy. See EPILEPTIC INSANITY.

SYMPTOMS.—The first symptom of insanity is an *alteration* in the feelings or conduct of the individual. He feels that something is amiss, as in truth there is. There is a defective condition of his nerve-force and a reduction of the highest mental centres, but this he does not realise, and proceeds to account for his feelings by external causes. He imagines that something has been put in his food, or, feeling himself to be changed, thinks that every one is looking at him or conspiring against him. The stage of *alteration* may be long or short, and during it his conduct and feeling may be changed, but in the great majority of cases it does not last long without *delusions* which arise out of, and represent, the feeling experienced. Delusions are not the first symptom of insanity; they are preceded by the altered feelings, so that when we find delusions it is probable that the disorder has lasted for some time, and on inquiry we hear that he has complained of his head, that his nights have been sleepless, his appetite impaired, and his digestive apparatus disordered. The first stage of insanity in most patients is one of depression. This at first may be slight, and may pass off with rest and treatment, or it may increase till it becomes a grave melancholia with suicidal tendency requiring active measures. In a certain number the depression is succeeded by exaltation and excitement. This may point to serious cerebral mischief and commencing general paralysis. The reduction here is to a greater depth than in the melancholic, and the prognosis is less favourable, even if there be no symptoms of general paralysis.

Generally speaking, then, we find delusions present in insane persons; and it is to be remembered that delusions always concern the patient personally. Mistaken beliefs, beliefs in mesmerism, spiritualism, or ghosts, mistaken beliefs or popular fallacies about health, these are not insane delusions unless the person who holds them believes that they concern him, and make him act in some insane way. Those most commonly found are delusions that there is something amiss with the patient. Either he has some loathsome disease, as leprosy or syphilis, or he is poisoned by something put in his food or drink, or he is very wicked and going to be tried and punished or tormented or burnt. Many, especially women, are troubled by fears that they have been so wicked as to destroy their hope of salvation in the life to come, and many think not that they are wicked but that others are plotting and conspiring to ruin them, to rob them of money and character, and to accuse them of every kind of possible crime. This is the well-known *monomania of persecution*. On the other hand where there is exaltation of ideas and excitement and hyperæmia of brain, the patient will think himself immensely rich, strong, and of high rank. He will invent machines or paint pictures to bring him unbounded wealth. He will be

restless, incessantly moving about to carry out his projects, and requiring legal restraint to keep him quiet. These exalted ideas are common in general paralysis, but they are also found in ordinary mania.

Hallucinations of the senses are also frequently found in the insane, especially those of *sight* and *hearing*. The former occur in the acute rather than in the chronic stages of insanity, and pass away with the acute period, as in delirium tremens. But hallucinations of hearing 'voices' are met with in both the acute and chronic insane, and may remain when all other symptoms have subsided, or if they vanish they are apt to reappear at any time. Such patients are a dangerous class, often incurable, and under the influence or command of the voices may commit crimes or suicide.

Insane acts.—Although in the great majority of insane patients delusions are found, yet the insanity of some is shown by insane conduct without apparent delusion. This class often presents great difficulties when restraint is needed, for it may be alleged that the act proceeds not from insanity but from vice. There may be such acts as indecent exposure, fantastic dress, solitary living or peculiar habits, dipsomania, homicide or suicide, self-mutilation, wasting or squandering of property or stealing. This will be discussed under MORAL INSANITY and IMPULSIVE INSANITY. The patients who commit insane acts do them chiefly under the influence of delusions, or they may not have arrived at the stage of delusion. Stealing, for example, is common at a very early period in general paralysis. Others may have lost their delusions but not wholly recovered, while patients may show the first signs of dementia, senile or epileptic, by foolish and insane conduct.

DIAGNOSIS.—A great number of patients who come before the physician for treatment present no difficulties of diagnosis. A man in acute mania or acute melancholia may be difficult to treat, but diagnosis is easy; and in cases of suspected general paralysis there may be no doubt about the insanity, although there may be as to the precise nature of the disease. Diagnosis is difficult in cases of so-called *partial insanity* where a patient with perhaps fixed delusions is able to conceal or deny them, and to talk rationally on ordinary topics. Information has to be gained from friends, and according to their views and inclination they will magnify or minimise the symptoms they have to describe, and frequently obstruct rather than aid the inquiry. The patient is said to have delusions; it is important to ascertain accurately whether that which is alleged is a delusion or a fact. If it is a delusion, is it an insane delusion and one which justifies the signing of a certificate? Also, does the patient hold the delusion at the time of the examination? Frequently we hear of a delusion held perhaps a month previously which has afterwards passed away. Where delusions concerning conspiracy or poison prevail, inquiry concerning the patient's health will often elicit them, and questions about the family or friends may bring to light delusions of conspiracy and persecution. Where there is exaltation and reckless expenditure it is often difficult to come to a decision. The alleged lunatic may spend more than he ought, yet not so much as to make it an undoubtedly insane act, and we may not be able to get at any delusion. Here a decided change may be noticed, a change in habits and feelings, which very often is the surest mark of insanity. The difficulties of dia-

gnosis arise chiefly in cases where the unsoundness of mind is of a negative rather than a positive character, an insufficiency of mental power rather than an aberration. Medical men are frequently called upon to say whether a person of weak mind, who is not a lunatic in the ordinary sense of the word, is or is not capable of taking care of himself and his affairs. Such questions often arise in forensic contests, and counsel argue that because there are no delusions nor the symptoms of ordinary insanity, therefore the alleged lunatic cannot be held to be of unsound mind. One class of these persons is that of the weak-minded or imbecile who have been deficient from birth or childhood. Here the difficulty is to determine the degree of the defect. They are not idiots, but something perhaps not far removed. They are often wicked and vicious, and it may be necessary to protect their persons as well as their property. They constitute a portion of the *morally insane*, may have a certain amount of education, an excellent memory, and are sharp and cunning in many ways. But they, notwithstanding, may have no idea of the value of money beyond that of half a crown, and know nothing of capital, interest, or income, being obviously at the mercy of any designing man or woman. There are others whose imbecility does not date from childhood, but has been caused by attacks of insanity, apoplexy, epilepsy, or drink. Here the difficulty is lessened, inasmuch as we have a standard of comparison in the patient's former condition which we have not in the congenital imbecile. A marked symptom is loss of memory, a common result of epilepsy and drink. But this also is a question of degree; and many can tell us all about their childhood and school-days who know nothing of what happened yesterday, and if they have made a will are not certain of it, and can tell us nothing of its purport. There is also the dementia of old age, often very painful to deal with if it takes the form of vicious or extravagant habits. Here we must compare the former life of the patient with his present, and we shall probably recognise the change which is so often the best proof of insanity. The difficulty in all these classes is greater if we have not known the individual previously. Then our comparison is with a former condition, only known by hearsay, which will be influenced by the friends according to the direction they wish our opinion to take. This unsoundness of mind is observed in persons whose insanity is not marked by delusions but by conduct, and much of it depends on hearsay, for many insane acts, even attempts at suicide and homicide, are not witnessed by the medical attendant but narrated to him. The alleged lunatic must be questioned upon them, and if he justifies them in an insane manner, the diagnosis is easy. If he denies them, further inquiry may be necessary. Perhaps the most difficult of all are cases of dipsomania. There is no law which enables us to subject inebriates to compulsory restraint. They cannot be placed under certificates unless there is evidence of insanity. In delirium tremens there often is such evidence, but it is not wise to send such patients to an asylum, as in a week or less the disorder will come to a favourable or unfavourable conclusion.

PROGNOSIS.—The first question asked by the friends of a patient is, will he get well? If the malady is recent and acute and is not General Paralysis, the answer will be favourable, always

provided that proper treatment be adopted. An asylum may be an obvious necessity, but often this is violently resisted, and it is certain that many curable cases are converted into chronic by improper treatment. If adequate measures are employed, a large proportion of recent cases will recover, even when very acute. No insanity is more acute than the delirious mania of puerperal women, yet the recoveries from this in asylums are 80 per cent. The percentage of recoveries from acute delirious mania in males is not so high, but the majority get well at any rate in the first attack. The prognosis here is whether the patient will live or die. If he lives he will almost certainly recover. The principal points to be observed are the pulse, the tongue, and the temperature. A rapid pulse, a tongue brown or black and dry, and a high temperature are unfavourable. Acute delirious melancholia with obstinate refusal of food is a very formidable disorder, and, as it frequently occurs in patients whose health is already broken, the prognosis is bad. But melancholia of a less acute type is curable beyond any other form of insanity, and this after a long period of depression. Such patients may have the most torturing delusions, the most suicidal tendencies, may continue in this condition for years, and then get quite well. And this is to be borne in mind if legal provisions have to be made. We are constantly asked whether a family history of insanity will prevent a patient's recovery. It certainly does not in the first attack, as is shown by the large percentage of recoveries in puerperal insanity, where hereditary predisposition is generally the cause. The insanity of young persons is mainly due to this, but a large number recover. Such, however, are apt to have subsequent attacks, and many become chronic lunatics later in life. With the exception of the melancholic an unfavourable prognosis must be given in all cases where the duration of the disorder is at all prolonged. All writers are agreed that early recognition and early treatment conduce to recovery. If we are consulted about patients whose insanity has lasted a twelvemonth or longer, the prognosis is bad, especially where the symptoms at first slight have grown by degrees till they have become severe and formidable; those who have hallucinations of hearing of a twelvemonth's duration are unlikely to lose them. Epileptic insanity is unfavourable, because the epilepsy must have been frequent to cause the mental disturbance, and is not likely to cease. Alcoholic insanity may or may not be cured according to its nature and degree. Acute insanity following a bout of drinking may pass off, and as in delirium tremens the patient will recover entirely, but the dementia from long-continued inebriety depending upon changes in the brain-cells and centres is most unfavourable, as are any organic changes produced by syphilis. All recurrence, whether after long or short intervals, is bad. Some patients have alternate days, others alternating attacks of mania and melancholia, with perhaps a short interval of sanity between. Many have recurring attacks of the same kind. The prognosis is unfavourable in all such cases.

TREATMENT.—There are two classes of the insane requiring treatment: those who suffer from acute and curable insanity, and those whose malady is chronic and incurable, who must be kept under more or less restraint for the term of their lives. We are not now concerned with the latter. When

dealing with the former, the first question is, where and by whom is the patient to be treated? It may be laid down as a rule to which there are but few exceptions that an insane person cannot be properly treated at home. If the insanity is not a mere transitory outburst but a prolonged attack with delusions and excitement, maniacal or melancholic, home is the worst place. Removal to fresh surroundings is an imperative necessity, and the sight of near relatives is, generally speaking, as prejudicial as that of the home. Many must be removed to an asylum, some because their means admit of no other course, others because they are too dangerous to be kept in an ordinary house. But there are patients who can be treated successfully out of an institution, and if they are young or are earning their livelihood in a profession or post of responsibility, it is much to their advantage to escape the stigma of a lunatic asylum. Among these are:—

(a) Cases of acute delirious mania, puerperal or other, which often run a short and regular course, and may be treated in a private house if suitable rooms can be provided—cool, airy, and quiet, with windows and fire guarded, under the care of experienced and sufficient attendants. If any relatives are in the house, the patient should see them rarely or not at all.

(b) Melancholic patients whose malady is not acute, who take their food, are clean and orderly, and able to take outdoor exercise, may be treated in private houses other than their own. A friend or relative may be of the party, but strangers are a valuable adjunct. Most melancholics are suicidal, yet adequate vigilance should prevent this. Many are able even to travel, and derive great benefit from constant change of scene.

(c) Young patients suffering from 'acute dementia' or, as it has been called, stupor with dementia (*see DEMENTIA*), need not be sent to an asylum, and may even be treated at home under an experienced and judicious attendant who will carry out the directions of the medical man.

There are some, however, who have a much better chance of recovery in an asylum, and whose malady is likely to become chronic if this treatment is not adopted.

(d) The melancholia of many patients is too acute for residence in a private house. They are on the look-out all day and all night for opportunity of suicide by windows, stairs, or other means; they require forcible feeding, strip off their clothes, and will not lie on a bed. All such demand the appliances and safeguards of an asylum.

(e) There are patients suffering from acute mania, not acute delirium, but conscious mania, who, knowing well what they are about, are noisy and violent, even homicidal, mischievous and quarrelsome, always complaining of the attendants, wilfully wet and dirty. This form of insanity is generally of some duration, the patients require much exercise, but are not fit to walk beyond asylum-grounds, and demand the authority and supervision of an asylum-superintendent, for they cannot be left to attendants only.

(f) Very few general paralytics can be treated out of an asylum. If dementia commences very early and the patients are quiet and tractable, some may be managed in a private house if the friends greatly desire it, but the grandiose exaltation of the majority, the impatience of control and reckless violence, make an asylum necessary. Once

there, they are the happiest members of the community.

(g) There are some whose malady, not being very acute, has been called *partial insanity* or *monomania*. Sometimes it is a form of melancholia marked by suspicion, aversion, delusions of persecution, and general alteration of feeling and conduct. Above all, it is characterised by the most intense egoism. This is the condition of many of the chronic insane, but occasionally the disorder though similar in many respects is recent and curable, and for this an asylum is the most effectual remedy. Because he is not acutely insane or dangerous the friends shrink from such a step, but placed in a family or private house, a patient of this kind becomes the centre and focus of the whole of his surroundings, and his egoistic ideas are fostered accordingly. If he is melancholic, never was man so unhappy, so God-forsaken, so tormented; those about him must assure him fifty times a day that it is not so, and be for ever encouraging his luxury of woe. If he is not melancholic, but the opposite, he never ceases to accuse and revile those about him, and strives to make their lives as uncomfortable as he can. Place one of these in an asylum, where, instead of being the one important unit, he becomes, say, the hundredth part of the whole community, occupying not the entire attention of those who have charge of him, but only a very small part, his self-importance is greatly chilled, especially as his ideas and complaints are totally disregarded by the other ninety-nine inmates. Many thus recover, often without medical treatment of any kind. The asylum carries with it a moral treatment not to be found elsewhere.

With regard to medicinal treatment little need here be said. The medicines appropriate to each form of insanity will be mentioned under the different heads. The chief medicines required are tonics and sedatives, and among the former the most valuable are the preparations of iron, either alone or in combination with arsenic or strychnine. Constipation, which is almost always a trouble in melancholia, must be combated by such drugs as cascara, given at regular intervals, with or without enemata, while massage of the abdomen is often useful. Of sedatives and hypnotics there is a long series. Besides opium and the bromides there are chloral hydrate and chloralamide, sulphonal and trional, hyoscine and hyoscyamine, and paraldehyde, probably the safest of all. Opium is especially beneficial in melancholia, while the bromides are more suited to excited and exalted patients. Chloralamide is preferable to chloral hydrate, trional to sulphonal; hyoscine and hyoscyamine are dangerous medicines, and are rapidly going out of favour. The objection to paraldehyde is its nauseous taste, but it is a safe narcotic with little unpleasant after-result, and may be administered without risk in the majority of cases. The treatment of insanity by the administration of animal extracts, lately so much advocated, has been very carefully carried out at the Royal Asylum, Morningside. Easterbrook has published a detailed account of the patients there treated by various extracts, especially thyroid, and came to the conclusion that these substances produce a general tonic effect upon cell-metabolism. A certain number of patients recovered under large doses of thyroid where previous treatment had been well tried but failed, yet no one recovered from ordinary intractable insanity under small or moderate doses.

In a hundred cases of insanity which were not hopeless there were twelve recoveries.

Good and abundant food is most necessary in the treatment of the insane. A large number, especially the melancholic, refuse food, but must not be allowed to remain long without it. Either by the nose or by the mouth, they must be fed; it is useless to wait in the hope that hunger will induce them to eat, for starvation does not cause hunger in such persons, but on the contrary, adds to their weakness, bodily and mental. When they find out that they will be fed by force, they frequently give in and take their food. G. F. BLANDFORD.

II. Insanity, Pathology of.—During the last six years the pathology of insanity has made very rapid and remarkable advances, which have been coincident with, and in a large measure dependent upon, corresponding advances in our knowledge of the normal structure and physiology of the nervous system. It has also been very greatly assisted in its progress by the precise knowledge that has been gained regarding the behaviour of the nerve-cell under pathological conditions, through the brilliant series of experimental researches carried out by Lugaro, Marinesco, van Gehuchten and others. During this period, moreover, the modern doctrine of the toxic basis of insanity has taken definite shape and steadily grown in importance. The very numerous structural alterations that can be demonstrated by modern methods of investigation in the brains of persons who have died insane are to-day recognised to be, with few exceptions, the result of the action of various toxins, some of which are introduced from without, but most of which are generated within the body itself, either in consequence of perverted functional activity of various organs and tissues, or by micro-organisms.

We shall not here attempt to frame any classification of the pathological varieties of insanity, since the present position of knowledge does not permit of this being done with accuracy. The ultimate classification will be one in which the so-called mental diseases are simply placed in their proper relationship to other diseases of the nervous system; they certainly will not collectively form a special subdivision.

ÆTIOLOGY AND PATHOGENESIS.—It is now very generally recognised that there are two great factors in the pathogenesis of a very large majority of cases of insanity—(1) an inherent weakness of individual organisation, and (2) certain exciting or determining causes. The essential nature of the inherent weakness of individual organisation which predisposes to the occurrence of insanity is as yet very imperfectly understood. That such a predisposition does, however, exist in many individuals, and that it tends to be transmitted from generation to generation, are facts too clearly proved to admit of dispute at the present day. There is conclusive experimental evidence that the nerve-cells of lower animals vary within considerable limits in different individuals of the same species in regard to their vulnerability to certain toxic agents. Extensive lesions of essentially the same character as those produced by such toxic agents in lower animals can be demonstrated in the brains of persons who have died during an attack of acute insanity, in the pathogenesis of which it is now certain that toxins play an essential part. On the other hand, there are the strongest grounds for believing that the same toxins may act

upon the nerve-cells of other persons without producing either similar lesions or corresponding symptoms. There is, therefore, reason to regard predisposition to insanity as determined by a comparatively feeble resisting power of the brain-cells to the action of certain toxins, or by an instability of cerebral tissue.

The exciting or determining causes of insanity are very variously enumerated and classified. But, with very few possible exceptions, they are all capable of being regarded as consisting in the action of toxic substances upon the cerebral tissues. These toxic substances may conveniently be classified as follows:—

(1) Those that are introduced from without, such as alcohol, morphine, lead-salts, cocaine, &c. This form of toxic action is often distinguished by the term *hetero-intoxication*.

(2) Those that are generated in the tissues of the body itself, in the course, or as a remote result, of various infective and non-infective diseases, such as syphilis, influenza, rheumatism, &c.; or in consequence of perverted functional activity of various organs such as the liver, kidneys, thyroid gland, &c.; or in association with other disorders of metabolism, such as those determined by living in bad hygienic conditions, and by various forms of mental shock and over-strain. To such toxic action the term *auto-intoxication* is commonly applied.

(3) Those that arise in the contents of the alimentary tract in consequence of functional derangements of the digestive organs, and are absorbed into the system, producing the condition generally referred to as *auto-intoxication from the gastro-intestinal canal*.

(4) Those that are developed by the action of micro-organisms which have gained admission through the alimentary tract into the blood-stream, producing the condition known as *auto-infection*.

It has long been generally recognised that certain forms of insanity are determined by hetero-intoxication. Perhaps the most obvious example of the kind is alcoholic insanity. It is only comparatively recently, however, that the vast importance of auto-intoxication as a cause of insanity has been adequately realised. As above indicated auto-intoxication has numerous different forms. In many instances it is initiated by the action of micro-organisms, that is to say, by infection, but the primary action of the specific toxins is soon reinforced, and indeed often entirely replaced by various secondary intoxications. Other forms of auto-intoxication have no special relationship to organismal infection, but arise merely from the disordered functional action of various glandular organs and tissues.

The all-important rôle of intoxications and infections in the pathogenesis of nervous diseases will, perhaps, be better understood from the following conclusions recently formulated by D'Abundo and Agostini, whose views may fairly be regarded as representing the consensus of the modern authoritative opinion upon the subject:—

(1) In the pathogenesis of nervous diseases in general, infections and intoxications are the most frequent, conspicuous, and active element, and this at all periods of life, intra- and extra-uterine.

(2) An infective-toxic heredity (e.g. syphilis, alcoholism, &c.) favours in the descendants the development of infective-toxic nervous diseases with typical lesions.

(3) Infections and intoxications in the parents,

or in the mother during gestation, very often produce in the foetus a most marked retardation in the process of myelinisation in the different systems of nervous connection.

(4) Some of the degenerative neuroses are to be regarded as due to defective cerebral and spinal organisation, arising from intra-uterine toxic pathological processes that have been cured.

(5) Infections and intoxications of the nervous system favour the development of secondary intoxications which feed, reinforce, and complicate the clinical phenomena, and together produce the forms of disease due to poly-intoxication.

(6) The action of infective-toxic agents can display itself in any part of the nervous system, leading to peripheral or central, systemic or disseminated localisation, and resulting in the acute or chronic neuro-psychoses.

(7) Mental confusion is merely the most frequent clinical type of infective-toxic action; other psychopathic types may also have a toxic origin.

(8) Acute delirium may be regarded as a clinical manifestation caused by various infective-toxic agents.

(9) Recent researches upon the ætiology of general paralysis of the insane greatly strengthen the theory of its infective-toxic origin.

(10) The clinical manifestations of infections and intoxications of the nervous system are the resultant of more or less profound nutritive disturbances, which at certain stages are capable of arrest, even when the symptomatology is such as to make us doubt the possibility of recovery.

We shall briefly indicate the probable relationship of these various forms of intoxication to some of the more important clinical varieties of insanity.

There are undoubtedly certain cases of acute insanity that are brought on by over-exertion of brain-function (idiopathic insanity). It seems very improbable that the cerebral disorder in these cases is the direct result of the excessive functional activity of the cortical neurons, because it is certain not only that these elements have great powers of recuperation after fatigue, but also that their functional activity becomes practically suspended long before an irreparable state of exhaustion is reached. The morbid cerebral changes which develop in consequence of such over-exertion of function are probably to be explained by certain secondary changes. The continuous or frequently recurring excitation or irritation of the nerve-cells induces cerebral hyperæmia, which from various causes tends to be maintained for an indefinite time; it is possible that the function of the vaso-motor nerves becomes overstrained. A chronic sub-inflammatory condition is produced; the metabolism of the nerve-cells becomes disordered; and waste products, abnormal in kind or in quality, are discharged into the cerebral lymph-channels. Hence the tissues forming the walls of these channels undergo proliferative and degenerative changes and at the same time tend to become slightly infiltrated by leucocytes. More or less serious obstruction to the passage of the lymph naturally results. The continuations of these channels in the Pacchionian villi and dura mater (the whole extent of which is now known to transmit cerebro-spinal fluid to the neighbouring veins) likewise tend to become in some degree obstructed through irritation and consequent proliferation and degeneration of the tissues forming their walls. The outflow of cerebral lymph is thus

seriously interfered with, and the nerve-cells are bathed by a fluid which is in a morbid condition, being loaded with the products of waste. Hence, especially if hyperæmia is long maintained, the nervous disorder tends to become intensified and may progress even to a fatal issue. In other cases the morbid processes gradually subside, and repair takes place.

There can be little doubt that such hyperæmia and secondary local auto-intoxication are important factors in the pathogenesis of all cases of acute insanity, and also in that of most forms of chronic insanity. But there are probably many cases of acute insanity in which the primary cause is itself of a purely toxic character. Numerous observations have been recorded of the occurrence of various micro-organisms in the blood of persons suffering from acute delirious mania. For the most part, however, these organisms have been simply the common pathogenic bacteria associated with supuration, and it is doubtful if their presence in the blood can be regarded as representing more than a secondary auto-infection. This at least is the view maintained by Ceni and Ferrari, who have very carefully investigated the whole question. There is much greater reason for the belief that the special bacillus isolated by Bianchi and Piccinino is the determining cause of one form of acute delirium, although absolute proof is still wanting. The remarkable cures that have been obtained by Marro and others by washing out the stomachs of patients suffering from acute mania, as well as other evidence bearing upon the question, would seem to indicate that many cases of this disease are determined by auto-intoxication from the gastrointestinal canal. It is also maintained by some authorities that certain cases are the result of conditions of simple auto-intoxication, arising in ways not as yet understood. The delirium frequently associated with many severe febrile diseases is plainly of toxic origin, although in such cases there is doubtless at the same time diminished resisting power on the part of the cortical nerve-cells.

Acute alcoholic insanity is clearly determined chiefly by the direct action of alcohol upon the cortical nerve-cells. In chronic alcoholic insanity we have to recognise, in addition to the same factor, a progressive secondary intoxication resulting from organic changes in various organs, more especially the kidneys, liver, and stomach. Although the precise nature of normal senile involution is at present in dispute, there can be no doubt that, in its later stages at least, senile insanity is essentially the result of auto-intoxication dependent chiefly upon organic changes in the abdominal and thoracic organs.

General paralysis of the insane is now clearly recognised to be a toxic disease. *See* GENERAL PARALYSIS OF THE INSANE.

The relation of disorders of the cerebral circulation to the different forms of insanity is certainly as yet in many respects very obscure. We are inclined to believe, however, that such disorders have much greater importance than is at present generally attributed to them, at least by neurologists in Great Britain. The comparative disregard in which they are now held as factors in the pathogenesis of insanity is doubtless largely due to the influence of the writings of Leonard Hill, who on the ground of numerous experimental observations has formulated certain

conclusions with which much of the older teaching, founded mainly upon clinical observation, is clearly incompatible. Hill maintains that the quantity of blood within the cranium is practically invariable (the Monro-Kellie doctrine), and that therefore cerebral arterial hyperæmia can only imply an increased flow of blood through the brain, not a greater distension of vessels; and that cerebral venous congestion necessarily implies compression of arterioles and capillaries, and therefore arterial anæmia. He also maintains that there is no local mechanism by which the blood-supply to the brain is regulated, and that 'the cerebral circulation is controlled by the vaso-motor centre acting on the splanchnic area.'

With regard to the first of these conclusions, it seems to us that Hill has been led into error by neglecting to take into account the displaceable lymph within the substance of the brain itself, and by failing to recognise the elasticity of the vessels and other cerebral tissues. When these factors are taken into consideration, it becomes evident that the quantity of blood within the brain is capable of undergoing important variations through inverse variations in the quantity of the lymph within the brain; and that vascular distension is not limited, as Hill maintains, by the rigid walls of the skull and the 'incompressible brain-substance,' but by the contractile and elastic coats of the vessels themselves.

With regard to the second conclusion, we maintain that Hill's experiments are by no means decisive, and that there is strong evidence in support of the existence of a local mechanism for the regulation of the cerebral blood-supply. Vaso-motor nerves in the pial arterioles have now been clearly demonstrated, and it is impossible to believe that they can have no functional importance. Moreover, prior to publication of Hill's work, Cavazzani, using methods certainly no less trustworthy, obtained experimental evidence of vaso-motor action upon the cerebral vessels, and more recently Spina has recorded similar observations. Lately, also, Acquisto and Pusateri, and D'Abundo have demonstrated at least the anatomical basis for a dural reflex, concerned with the regulation of the calibre of the pial arterioles.

Hence we maintain that both active and passive congestions, essentially similar in their nature and effects to those that occur in other organs, do occur in the brain, and that, although the subject is still invested with much obscurity, there are the strongest grounds for believing that there is a local mechanism for the regulation of the cerebral blood-supply, disorder of which is often an important factor in the pathogenesis of the various forms of insanity.

Normal mental action has clearly been proved to be accompanied by cerebral hyperæmia. We maintain that this is a true quantitative hyperæmia, and that it does not consist merely in an increased rate of blood-flow. As already indicated, functional cerebral hyperæmia is apt to pass into pathological hyperæmia, if the excitation or emotion that brings it about is unduly prolonged, and it is probable that idiopathic insanity (or the insanity which follows on the action of so-called 'moral causes,' such as grief, worry, anxiety, and prolonged study) is initiated in this way. In other forms of acute insanity, there is certainly as a rule, and probably always, similar active congestion. In senile mania,

active hyperæmia appears to affect certain areas specially, its incidence and distribution being in large part determined by organic changes in the vessel-walls. In general paralysis, there is constantly well-marked active congestion of the brain, which probably specially affects the cortex. The view, which has recently gained much credence, that in this disease there is a condition of cerebral anæmia, consequent upon venous congestion, is, we believe, entirely erroneous. True cerebral anæmia, consisting in a diminution in the quantity of blood within the brain, and not merely in retardation of the blood-flow, is probably an important factor in the pathogenesis of many cases of chronic melancholia. It has now been clearly established that states of mental excitement are in general associated with low arterial tension, and that, on the other hand, states of acute mental depression are very commonly accompanied by the opposite condition of high tension.

PATHOLOGICAL ANATOMY.—*Congenital Abnormalities of the Brain.*—Slight structural abnormalities of the brain are unquestionably far more common in the insane than in the mentally sound. They are especially frequent in cases of idiocy, congenital imbecility, and epileptic insanity. Some of the more gross congenital defects are quite incompatible with normal intellectual development, while many others have only rarely been observed in persons who were mentally sound. Some of the more important abnormalities that may be observed are the following: atypical arrangement of convolutions and sulci, microgyria, absence of the corpus callosum, imperfect separation of the cerebral hemispheres, heterotopia of the grey substance, porencephaly and microcephaly.

Focal Lesions of the Brain.—Localised gross lesions of the brain are certainly of very considerable importance in the pathogenesis of insanity. They are present in from 25 to 30 per cent. of cases examined after death. They consist chiefly of softenings of vascular origin, among which are to be included recent and old hemorrhagic, embolic, and atrophic softenings. The last, which are by far the most common, result from gradual occlusion of arterial branches and capillaries, owing to morbid changes in their walls, more especially endarteritis deformans and hyaline-fibroid degeneration. They occur especially in senile insanity and are generally multiple. They affect the cortex chiefly. Other gross lesions that may not infrequently be observed in the brains of the insane include primary and secondary tumours, tubercular and gummatous nodules, and sclerotic areas.

Morbid Conditions of the Skull.—These are essentially of two classes, namely (1) variations in form and size overstepping the limits of the normal, and (2) changes in the structure of the bones, evidenced especially by abnormalities in thickness, consistence, and histological characters.

The skulls of the insane in this country have not yet been sufficiently studied by strict craniological methods to permit of precise conclusions being formed regarding the frequency and significance of the slighter morbid conditions of the former class.

Changes in the thickness, texture, &c., of the bones of the skull are extremely common in the insane. The calvarium, as a rule, suffers more than the base. General or local thickening is exceedingly common. Statistics compiled by different observers show the condition to be present in from 25 to

50 per cent. of all cases. It is sometimes accompanied by the development of large irregular bosses, or of more or less numerous small excrescences on the inner aspect. The bone is usually at the same time condensed, but in some instances it is softened. The highest degrees of thickening occur in senile insanity, but the largest proportion of thickened crania is found in cases of general paralysis. Atrophic thinning is comparatively rare. It is observed mostly in cases of senile insanity. Osteosclerosis, or condensation of the bone, which is usually accompanied by thickening, is seen in about 30 per cent. of cases. The condition is practically a constant one in general paralysis. Osteoporosis, or general softening of the bone, occurs in about 5 per cent. of cases. It is mostly observed in the senile insane. The weight of the cranial bones is greatly increased in most cases of general paralysis, and also in many of epilepsy.

Morbid Conditions of the Dura Mater.—One of the most common morbid conditions of the dura in cases of insanity is an undue degree of adhesion to the calvarium. It is present, according to the statistics of different observers, in from 15 to 40 per cent. of cases. Senile insanity, epileptic insanity, and general paralysis furnish the largest proportion of examples. Morbid adhesion to the pia-arachnoid is comparatively rare. Thickening of the dura is exceedingly common, but is generally slight in degree and somewhat difficult to recognise. Atrophic thinning is occasionally to be observed. Bony formations, most commonly situated in the falx cerebri, but sometimes near the outer aspect of the membrane, occur with considerable frequency. In all cases of long-standing insanity, the dura mater also undergoes other chronic alterations which, for the most part, are recognisable only on microscopical examination. They consist in general or local proliferation and degeneration of the endothelial cells of the inner surface and of the walls of the perivascular canals. As already stated, such changes cause obstruction to the outflow of cerebrospinal fluid. They also, however, in chronic forms of insanity, have another important consequence, namely, obliteration of large numbers of the superficial dural capillaries by compression. Following upon the obliteration of these original vessels, new capillaries shoot out upon the surface of the dura. They tend to give way before the normal blood-pressure during the last few days of life, owing to fatty and other changes in their walls. It is in this way that most of the subdural 'false membranes,' so common in the insane, are developed. They consist essentially of blood-clot which, however, is very commonly in process of vascularisation. Other subdural false membranes are formed from blood which has escaped from a cerebral arteriole or vein. See MENINGITIS (PACHYMENINGITIS HÆMORRHAGICA INTERNA).

Morbid Conditions of the Pia-arachnoid.—The most important morbid change to which the pia-arachnoid is subject in insanity is one which manifests itself as thickening, milkiness, and opacity, accompanied generally by granularity of the surface. It is a constant condition in all cases of long standing. It is seen in its most extreme degrees in general paralysis and senile insanity. The corresponding tissue-changes consist of general hyperplasia of the connective tissues and proliferation and degeneration of the endothelial cells lining the lymph-spaces. The granulations on the surface

are composed of proliferated endothelial cells. In advanced general paralysis, there is usually, in addition, well-marked infiltration of the tissues with the leucocytes, indicating an acute inflammatory process. Whether or not, apart from such leucocytic infiltration, we regard the condition as inflammatory will depend upon what we mean by inflammation (*see* INFLAMMATION). We have already insisted upon the great importance of conditions of general toxæmia in the causation of insanity, and have explained how secondary local auto-intoxication is set up by morbid metabolism in the cerebral tissues. We believe that both of these conditions contribute to the production of the structural alterations that occur in the pia-arachnoid, dura mater, and skull. The toxins, which are added to the lymph in consequence of the altered metabolism in the brain itself, are probably, however, more important in this connection. The tissues of the pia-arachnoid, dura mater, and skull are bathed by a lymph which is in varying degrees unsuitable for their healthy nutrition. Hence they undergo the morbid alterations which we have briefly described.

Morbid Condition of the Cerebral Vessels.—The morbid changes that affect the cerebral vessels in insanity are of great importance, but are of so numerous and complicated a nature that we can do little more than name them here (*see* BRAIN, Vessels of, Diseases of). Two forms of granular yellow pigmentary deposit are very commonly to be observed in great abundance in the adventitia of the intracerebral vessels. One represents a pathological increase in the quantity of a normal constituent. It is generally of a light-yellow colour. The other, which has a reddish-yellow tint, consists of hæmorrhage, and is always indicative of antecedent hæmorrhage. Hyaline degeneration chiefly affects the adventitia. It occasionally occurs as an acute condition, but is more common in its chronic form of the hyaline-fibroid degeneration of Gull and Sutton. This change is very often a well-marked one in various forms of chronic insanity. It specially tends to implicate the capillaries of the first layer of cortex. Endarteritis deformans or atheroma occurs in its most severe form in some cases of senile insanity. Acute periarteritis occurs in the advanced stages of general paralysis. Colloid degeneration and calcification have been described, but are certainly very rare. The condition known as '*état criblé*,' consisting in the development of spaces at the side of the larger vessels in the white substance, is common in all atrophied brains, especially in those of the senile insane.

Morbid Conditions of the Neuroglia and Mesoglia. In all forms of insanity in which there is chronic irritation in the brain, or dead tissue to be replaced, the neuroglia-cells undergo hyperplasia. This condition is especially common in the outermost layer of the cortex, and in the white matter. It is generally present throughout extensive areas of the cortex in general paralysis. Localised areas of sclerosis are exceedingly common in the aged insane and in chronic alcoholic insanity. Ventricular granulations, which are almost constantly present in general paralysis and senile insanity, are essentially localised overgrowths of neuroglia.

Morbid Conditions of the Nerve-cells.—Although many points regarding the normal structure of the nerve-cell are still in dispute or undecided, the following brief description, which applies specially

to the larger cortical nerve-cells, probably fairly represents the consensus of opinion upon the subject among the best authorities at the present day. The nucleus is generally placed about the centre of the cell-body. It has a distinct limiting membrane and contains a reticulum and granules, and a rounded nucleolus, generally centrally situated. All of these constituents are acidophile in reaction, excepting from two to four small particles adhering to the nucleolus, which are basiphile: these represent the whole of the chromatin in the nerve-cell. The cell-body has various shapes, but is commonly pyramidal. It extends on the one hand into three or four long dendritic processes, the protoplasmic processes or dendrites, which, except close to the cell-body, are clothed with numerous short filiform or pyriform processes, the gemmulæ; and on the other into a single delicate smooth process, the axis-cylinder process or axon, which at some little distance from the cell-body begins to give off at right angles some finer branches, termed collaterals. The main fibre may become the axis-cylinder of a medullated fibre, or run a comparatively short course as a non-medullated fibre. It terminates in close relationship to another nerve-cell, either as a peri-dendritic or a peri-cellular arborisation. The basis of the cell-protoplasm consists of minute fibres, which for the most part course independently in the processes, but form a true reticulum round the nucleus. The fibrils of the axis-cylinder process arise from this peri-nuclear reticulum. In the cell-body the spaces of the reticulum are filled up by a homogeneous or faintly granular substance which has a strong affinity for certain dyes, such as toluidin blue, and which in preparations by certain methods appears in the form of minute aggregations, the Nissl-bodies, or chromophile elements of the nerve-cell. The bodies of the larger cells contain numerous minute channels which open on the one hand into a peri-nuclear space (bridged, however, by the fibrils, which are attached to the nucleus), and on the other upon the surface. Near the base of the cell, there is generally in the adult a small collection of a pale-yellow substance, the nerve-cell pigment. Various forms of reticula at the surface of the cell have been described by different observers, but their significance is still somewhat doubtful.

Although the neuron-theory, according to which each nerve-cell, together with its processes, forms an anatomically and physiologically separate element, has in recent years been boldly attacked, it is still maintained and defended by the great majority of authorities. It is now very generally believed that the nervous impulses are received by the dendrites (in some instances by the cell-body) and discharged by way of the axon (*law of dynamic polarisation*). It has been clearly established by recent observations that the chromophile substance is utilised during the functional activity of the cell. It has been maintained by some that the protoplasmic processes exhibit amoeboid movements, which have an important action in the breaking and making of connections with other cells, and that during sleep these processes are retracted. This theory has been strongly opposed by Lugaro, who, on the basis of a series of experimental observations of his own, has advanced another hypothesis, which seems to us to be much more credible. He maintains that only the gemmulæ exhibit contractility, and that not retraction but general expansion of

these appendages, implying multiplication of contacts and wide diffusion and consequent weakening of stimuli, is their characteristic attitude during sleep. In conditions of fatigue their contractility is greatly diminished; after rest their capacity to contract is fully restored. During psychical activity the gemmulæ undergo continuous movements of retraction and expansion, whereby contacts with the terminations of the axis-cylinder processes of other cells are broken and made.

Lugaro, who has undoubtedly done more than any other observer to advance our knowledge of the nerve-cell in normal and pathological conditions, has recently put forward another hypothesis which is of special interest in relation to the pathology of insanity. He states this hypothesis as follows: Two distinct elaborations of external impressions occur in the nervous centres, one inter-neuronic, at the terminations of the afferent fibres, and the other intra-neuronic, between the wave conveyed by the dendrites and the dynamical processes that develop in the interior of the cell-body; the first corresponds to the phenomena of consciousness or perception (*fenomeni di conoscenza*), the second to the affective states. Flechsig has lately divided the brain into sensori-motor and associative or intellectual areas, and has attributed certain special functional values to various portions of the latter. If his views upon this subject could be accepted as thoroughly established, they would form a highly important physiological basis for the explanation of many morbid phenomena exhibited by the insane. But it happens that at the present moment they are being attacked in their very foundations by several other high authorities, and therefore it is necessary for us to await the issue of the controversy before deciding whether they can be utilised in pathology or not.

The experimental pathology of the nerve-cell is now an extremely rich and elaborate department of neurological science. It may be said that two separate types of morbid change have been distinguished, namely, primary and secondary degeneration. The former results from the direct action of a toxin upon the cell, or from an equivalent disturbance of nutrition; the latter follows injury to the axon. The characters of primary degeneration differ greatly according to the toxin employed and the intensity and duration of its action. But in general the alteration consists first in disintegration and dissolution of the chromophile bodies (*chromatolysis*), then in disintegration of the achromatic fibrils, displacement and disintegration of the nucleus, and death of the whole element. If the toxic action ceases before the first stage is passed, recovery may take place. In secondary degeneration the changes represent a definite reaction which runs its course and terminates either in recovery or in the death of the cell. Chromatolysis begins around the cone of origin of the axis-cylinder process and gradually spreads to the rest of the cell; the fibrils and nucleus may subsequently be involved as in primary degeneration. There is considerable difference of opinion as to the conditions which influence the intensity of the reaction and determine death or recovery of the cell. Experimental pathology has further taught us that many of the cellular alterations which can be recognised in the human brain after death are really the result of terminal auto-intoxications and other conditions arising shortly before or even after death, and that we must, as far as possible, look behind such alterations

in order to see the true pathological picture of the disease from which the patient has suffered. Thus it has been demonstrated that hyperpyrexia of short duration, and long-continued pyrexia, can produce a general, more or less complete chromatolysis of the cortical and spinal nerve-cells. It has also been shown that slight chromatolytic changes are produced by inanition, which, however, are probably chiefly of auto-toxic origin. Anæmia and prolonged insomnia have also been demonstrated to be possible causes of chromatolytic and other changes in the cells. Experimental observations have further shown the necessity of caution in correlating various deformities of the protoplasmic prolongations and their gemmulæ with morbid phenomena in the human subject. There can be no question that pathological alterations in these appendages of the cortical nerve-cells have great importance in various forms of insanity, but the histological methods at present available do not serve to distinguish them clearly from other alterations which simulate them, and which, it has been clearly proved, may be caused by terminal auto-intoxication, *post-mortem* change, the fixing fluids themselves, as well as by other still obscure causes. Lastly, it has been shown that the nerve-cells are very rapidly affected by certain *post-mortem* alterations, which it is therefore absolutely necessary to understand and to be able to discount, in all studies in neuro-histology upon the human subject.

Primary and secondary degeneration are the only diseases of the fully developed nerve-cell as yet clearly distinguished. The various types of morbid change that may be observed may occur in either of these forms of disease. We can only enumerate the more important of them here, namely, chromatolysis, achromatolysis, intense colourability of the achromatic substance, pigmentary degeneration, fatty degeneration, vacuolation, varicose atrophy, varicose hypertrophy of the axis-cylinder process, displacement of the nucleus and homogeneous degeneration of the nucleus. The one disease of the nerve-cell which does not clearly fall within the groups of primary and secondary degeneration is developmental arrest. Various degrees of this condition can be recognised in the cerebral cortex in many cases of idiocy and congenital imbecility.

When we endeavour to apply this knowledge in the study of the morbid alterations in the nerve-cells of the human brain in cases of insanity, we are met by endless difficulties. We recognise at once that most of the changes which up to a few years ago were regarded as the basis of the morbid mental phenomena have little to do with these phenomena, being for the most part initiated during the last few days of life or after death. Even yet the definite facts that can be said to be established are very few in number. We have in addition, however, a number of 'working hypotheses' which greatly help us to visualise what is going on in the cortical nerve-cells in various forms of insanity. We shall conclude by briefly indicating some of the more important of these facts and hypotheses.

The special phenomena of the various forms of insanity depend not upon different forms of nerve-cell lesion, but upon the nature and intensity of the toxic or other condition causing the primary or secondary degeneration, the localisation of the morbid action and the special reactive qualities of the individual nervous system. In cases of acute mania and acute melancholia dying in the course of

the disease, from 25 to 50 per cent. of the cortical nerve-cells show distinct degeneration of primary type. This percentage is much in excess of that to be found in other toxic diseases in which there have not been corresponding cerebral symptoms. Moreover, the lesions are much more severe, and it can be satisfactorily proved that large numbers of the cells have disintegrated and disappeared. In cases of dementia, secondary to attacks of acute insanity, it can be shown that a considerable proportion, in some instances fully 50 per cent., of the cortical nerve-cells has entirely disappeared. In senile insanity there is a slow degeneration of the cerebral nerve-cells, for the most part of a primary character; its severity far exceeds that of the similar changes associated with normal senile involution. The lesion is essentially diffuse in its distribution, but at the same time it is generally more advanced in some areas than in others. Extensive complete destruction of cells takes place, both in association with and apart from their involvement in local softenings of vascular origin. In general paralysis there is also a diffuse degenerative change in the nerve-cells of the brain, chiefly of primary type. The morbid process is, as a rule, much more acute than in senile insanity, and the destruction of cells is greater. In chronic alcoholic insanity there is always a very considerable loss of nerve-cells, while many of those that remain present changes of a primary or secondary character. In polyneuritic psychosis and other conditions of 'primary mental confusion,' Ballet and Faure, as well as other later observers, have found secondary degenerative changes in the giant and large pyramidal nerve-cells of the cortex, comparable to those that occur in the cells of the anterior horn of the spinal cord in peripheral neuritis. In all of these forms of insanity there are, in association with the degenerative changes in the cell-bodies, extensive lesions of their prolongations, that is to say, of the medullated nerve-fibres of the brain. Severe change in the one necessarily implies corresponding alteration in the other; the two being merely parts of the same issue-element.

If we accept Lugaro's theory of the functional contractility of the gemmule, we may regard the insomnia so commonly associated with insanity as dependent upon an irritable condition of the cortical neurons, in consequence of which these minute appendages of the protoplasmic processes continue to contract in response to the slightest stimulus, instead of remaining in their position of rest. On the other hand we may conceive of stupor as dependent upon an abnormal condition of the neurons, in consequence of which the contractility of the gemmule is more or less completely abolished. The hypothesis of the distinctive functional values of the inter-neuronic and intra-neuronic elaborations, the first portion of which, it seems to us, may now be accepted as a postulate, chiefly serves to emphasise what we have long insisted upon, namely, the paramount importance of the nervous connections, or the established relations of one neuron with another. It is in virtue of such connections, elaborated throughout the years of growth and education, that the marvellous machinery of the organ of the mind is gradually put together, and it is by their dissolution that this machine breaks down, in other words, that the mental powers become disorganised. In accordance with the second portion of this hypothesis, we may conceive of states of simple mania

and melancholia as dependent upon abnormal metabolism within the substance of the nerve-cells, and can understand how they are compatible with unimpaired action of some of the purely intellectual faculties, the integrity of which is dependent upon the maintenance of the mutual relations of groups of neurons.

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III. Developmental Insanity, strictly speaking, should include idiocy, congenital imbecility, and the greater part of epileptic insanity. But those conditions will be dealt with in separate articles.

SYMPTOMS.—The most typical and the most common developmental psychosis is unquestionably that which is liable to occur from puberty up to the age of twenty-five, and in 72 per cent. of the cases it takes the form of an attack of mania of a sharp and relapsing character, though in some cases it assumes a melancholic form. Only 32 per cent. of the cases occur between puberty and twenty-one, the remaining 68 per cent. occurring in the four years from twenty-one to twenty-five. Contrary to the common opinion, puberty and the three years following it, that is, the fourteenth, fifteenth, sixteenth, and seventeenth years, produce only 10 per cent. of the pubescent and adolescent insanity. A typical case of adolescent mania, once seen, is not soon forgotten. A young woman with a markedly insane heredity grows up till she is twenty-three without any neuroses, except that she remains long girlish and undeveloped in form. She then, without any sufficient exciting cause, exhibits for a few weeks a slight lowness of spirits, inattention and want of interest in her work and surroundings. This gradually passes off, and within another few weeks there appears a precisely opposite mental and emotional condition of liveliness and buoyancy of spirits. With this there is an unsettledness of conduct, a want of moral balance, a change of likings, a disregard of conventionalities and many unmaidenly ways exhibited before friends or strangers of the opposite sex. Sleep gets shortened, and a change of expression in face and eye is seen. This mild exaltation of cortical action soon passes into a more marked mania. The patient talks continuously and with little coherence of ideas; she sings loudly, dresses grotesquely, and becomes 'cheeky' to an outrageous degree, setting at naught parental and scholastic authority. She is boastful, offensively forward, immodest, impossible to please, and very perverse. She turns night into day, is irregular in her eating and in all her habits, is quarrelsome, blaming her guardians and companions for all her peculiarities of conduct, and is unable to get on with any one for more than a few hours at a time. Then comes a time when this 'simple mania' becomes really 'acute' in its character, and the patient screams, becomes incoherent in speech, tears her clothes, fights, and neglects the common decencies. She is then flushed in the face, and further altered in expression; her eyes are brilliant and fierce in look; and she cannot take care of herself in any way as to food or natural functions. During this time her temperature rises to 99.5° or even higher, her perspiration is offensive, and menstruation is temporarily suspended. She loses weight rapidly and soon looks haggard and exhausted. In a few weeks this acutely maniacal condition begins to abate, and is succeeded by what seems a near

approach to recovery of mental power and control, but with some confusion that may go on to stupor. Every one about her is hopeful and is intensely relieved to think the end of the attack is come. But this turns out not to be so, for about the time the next menstrual period is due the patient becomes mentally excited again, and goes through much the same sequence of symptoms as have been described. These periodic recurrences occur again and again until either a real and final recovery takes place after a year or so, or she sinks into secondary dementia—that most terrible of all mental states, where the hopes and fears of life, its joys and sorrows, its duties, capacities, and aspirations have disappeared for ever, and mind in the higher sense has died, while the bodily life may go on for many years. About 60 per cent. of the cases of adolescent mania recover, and 30 per cent. sink into dementia.

PERIODICITY.—The explanation of the remissional periodic character of the symptoms is that it is a morbid expression of the great law of generative and sexual periodicity. In the excited period there is always a strong sexual factor in the emotions, ideas, and speech, and the practice of masturbation is very common. Bouts of excessive sexual intercourse are sometimes indulged in, unsuitable marriages contracted, and sexual crimes committed during the early part of the exalted phase.

STUPOR AND MELANCHOLIA.—The symptoms in every individual case are of course not precisely on the clinical lines just sketched. We have in some cases delusions of suspicion and irritability, taking the place of the simple mania. In others the condition of confusion or complete stupor, or temporary mental obliteration, the 'acute dementia,' 'primary dementia,' 'dementia attonita,' of other writers, occurs as a developmental insanity, the patient becoming absolutely devoid of volition, standing still, not being able to speak or act, and looking as if the facial muscles were all thrown out of action, while the heart's action becomes very feeble. The pulse is weak and thready, the extremities cold and blue, chilblains forming on the hands and feet. In some cases ordinary melancholia occurs often with suicidal symptoms, and a tendency to periodicity in its incidence like the mania. In a very few cases a gradual weakness of mind occurs (*dementia præcox*).

Lesser Developmental Psychoses.—There are a series of lesser mental, volitional, moral, and 'conduct' changes, short of technical insanity, that are liable to occur in adolescents of both sexes derived from neurotic and insane stocks. These changes are often as distressing to deal with and more difficult to treat, while they are far more liable to be misunderstood than ordinary insanity. The chief of such conditions are stupidity or lethargy, asocial or anti-social state, causeless aversion to relatives or unreasonable defiance of authority, morbid egotism or delusional antipathies, visionary schemings or frothy religionism, and adolescent criminal acts, sexual perversions being common, as might have been expected. 'Mad' adventures, insane idealisms, and unhealthy attempts to produce original forms of literature or poetry are common enough among educated neurotics at this age. The absence of balance, common sense, practical or possible aims in life, and sobriety of conduct or thought generally is characteristic of such people, many of whom thus

show that they are true 'degenerates.' The writer regards all such as examples of the evolution of the impulse of reproduction—that organic instinct which is only second to the love of life in importance—upsetting the cortical condition and working of brains strongly predisposed to insanity or to the higher neuroses through heredity.

TREATMENT.—The most successful treatment is founded on physiological considerations. Feed well with all sorts of non-stimulating diet, such as eggs, farinaceous and fatty diet, and fruits. Give custards made of milk and eggs in large quantities. Exercise, work and amusements in the fresh air, and cold bathing are essential. The writer has no sort of belief in the treatment by confinement to bed lately recommended by some authors for such disorders. Cod-liver oil, maltine, and the hypophosphites are most useful. If sedatives are used a judicious mixture of the bromides and sulphonal (Potas. Bromid. gr. xv. and Sulphonal gr. x. three times a day) will be found useful. When patients begin to gain weight they are commonly safe—except those who are getting demented. About 60 per cent. recover. Recovery is accompanied, in addition to a gain in weight and fatness, by an expansion of the form and of the mammæ in women, and by the usual signs of completed physiological manhood in the other sex, such as the full growth of the sexual hair and beard. Many of the cases of adolescent mania relapse afterwards through child-bearing, moral upsets, alcohol, or senility.

T. S. CLOUSTON.

IV. Insanity of Pregnancy, the Puerperal Period, and Lactation.

1. Insanity of Pregnancy.—Insanity of pregnancy is rare, and accounts for about 1 per cent. of women admitted to asylums. The cases may be considered in two groups: (1) those occurring in the early months, associated with general disturbance of the circulation and nutrition, and (2) those occurring during the later months, associated with the increasing worry and discomforts of advancing pregnancy. In the first group the disorder may pass off with gradual adaptation of the nervous system to the altered conditions; while in the second the insanity is likely to continue after confinement as a puerperal insanity.

CAUSES.—(1) Inheritance of neuroses or insanity; (2) previous attacks of mental disorder, either before marriage or in connection with some previous pregnancy or confinement; (3) a first pregnancy, especially if associated with much vomiting, neuralgia, or sleeplessness; (4) moral causes, such as illegitimacy or desertion by the husband; (5) cardiac, renal, or other organic disease; (6) alcoholism or other toxic state; (7) early general paralysis of the insane.

FORM OF MENTAL DISORDER.—Most commonly the disorder is of the melancholic or stuporous type, but maniacal excitement is not rare. In the melancholic forms there may be apathy, loss of energy, and inability to fulfil ordinary duties, with self-accusation and suicidal desire, on the one hand, or agitation with hypochondriacal misinterpretation of the discomforts of pregnancy on the other. Hallucinations may be present, and suspicions of poisoning may lead to refusal of food. Aversion to the husband or to others may lead to violent assaults. Maniacal excitement may be of the delirious, the hysterical, or the ordinary acute maniacal type.

Occasionally chronic delusional insanity arises during pregnancy, and in a few cases the patient becomes weak-minded without any marked acute psychosis, and remains demented even in a first pregnancy.

PROGNOSIS.—This is, on the whole, good, especially in the early months, if the patient be removed from home and treated by rest till the nervous system has adapted itself to the new conditions. In cases occurring in the later months recovery does not take place till after confinement. The prognosis as to the mental life of the child is not a favourable one, and congenital mental defect or nervous instability is likely to result.

TREATMENT.—In mild cases rest under skilled nursing, with liberal feeding, removal from all household worries, attention to the bowels and sleep, and administration of tonics may effect recovery. In severe cases treatment in an asylum is necessary. In the administration of drugs regard must be had to the possible effect on the nervous system of the child. Induction of premature labour does not seem justifiable in the early months, as the disorder may pass off quickly; and as in cases occurring later delivery at the natural term does not cut the disorder short, there is no reason to suppose its artificial production would be beneficial, while it would add an additional risk to life.

2. Puerperal Insanity.—Insanity following childbirth is found in between 5 and 6 per cent. of the women admitted to asylums. Many of the milder and shorter cases of this nature never reach the wards of an asylum, but are successfully treated elsewhere.

CAUSES.—(1) An inheritance of neuroses or insanity. Some observers place this as high as 50 per cent. In many cases puerperal insanity is the expression of the presence of the neuropathic diathesis, and its subjects are 'degenerates' whose nervous system gives way first at the puerperal epoch. Occasionally a mother and her daughter may each suffer from puerperal insanity. (2) A first confinement, especially towards the end of the reproductive period, when the pelvis and soft parts have become unyielding. (3) Some complication attending labour, such as the use of forceps or anaesthetics, delayed labour, *post-partum* hemorrhage, ruptured perinaeum, or any cause leading to septic poisoning. The latter may be slight or severe. In some cases no doubt the mental disorder is of the nature of a toxic delirium, while in others it precedes or is merely concurrent with septic absorption, and might have occurred independently of it. Although primiparae are more likely than others to sustain injuries during parturition, the cases of puerperal insanity in which there was the most severe septicæmia were, in my experience, not primiparae. (4) Moral causes. These may be present in many cases, even where there is no inheritance of insanity and no evidence of septic complications. Any great strain or anxiety or emotional shock may upset the balance at a time when the nervous system has passed through the stress of delivery, and should have the opportunity for rest and recuperation. The birth of an illegitimate child, the death of a near relative, of the husband, or of the child, sudden fright, the apprehension of the pains and perils of confinement, excitement from officious and gossiping visitors, desertion or ill-treatment by a brutal husband, or the worry of an inefficient nurse may, for example, act in this

way. (5) Previous attacks of insanity either before marriage, or after former confinements. (6) Conditions leading to maternal ill-health during pregnancy, defective general nutrition, nervous exhaustion from frequent child-bearing or prolonged lactation, organic diseases, or alcoholism.

ONSET.—In some cases insanity has existed before confinement, and continues subsequently. In other cases it may begin immediately after delivery, and in a still larger proportion during some part of the first week. Other cases begin at varying periods up to six weeks or two months after parturition. The line drawn between the later puerperal insanity and the insanity of lactation is quite an arbitrary one, and there are many cases to which the term 'puerpero-lactational' might be applied.

FORMS OF MENTAL DISORDER.—In the main the cases fall within the two great groups of maniacal excitement and melancholic depression. Of those occurring within the first fortnight after delivery, the great majority are delirious or maniacal, while those occurring later are chiefly, but not exclusively, melancholic. A transient frenzy of excitement or delirium may occur during delivery, or immediately after, and may pass off very quickly. The mental disorder at this period is marked by its sudden onset, its extreme uncontrollable violence, its transitoriness, and the profound affection of consciousness which may be associated with an impulsive homicidal or suicidal act. Again, within the first few days after delivery a short, but very acute, delirium with rise of temperature, great restlessness and violence, hallucinations, rambling incoherence, and profound affection of consciousness may occur. Memory of the attack may be entirely lost. Transitory cases of this nature do not necessarily find their way into asylums.

Of cases of longer duration there are several varieties. Puerperal Mania is the commonest form. Within a few days after delivery there is sleeplessness, slight depression, loss of interest in the child, querulousness, and dislike to the husband and nurse. Maniacal excitement then rapidly supervenes. The cheeks become flushed, the eyes bright, the pupils dilated, the tongue dry, the pulse rapid and soft, and the temperature slightly raised. The excitement increases with noisiness, shouting, singing, and incoherent conversation which is often obscene. There is constant restlessness with struggling, fighting, and a tendency to strip off clothes, to impulsively strike those about her or the child, or even to injure herself. Relatives may not be identified, the attention cannot be arrested, and consciousness is profoundly affected. Hallucinations and delusions are frequent, and judgment is entirely at fault. Food is commonly refused, and there is loss of control over the bladder and rectum. Sleep is either entirely absent, or comes in short snatches from which the patient wakes to continue her ravings. The urine may be temporarily albuminous, or may in some cases contain sugar. The milk may be suppressed or may be secreted so plentifully that the breasts become tense and painful, abscess ultimately forming. The lochial discharge may be normal, absent, or offensive. The excitement may last a few days, weeks, or months, and is apt to recur periodically. After a time it may be replaced by depression with agitation, delusions of perdition, self-accusation, and suicidal attempts; or by stupor, silence, and apathy, but with eventual recovery, as the phy-

sical health is re-established. Menstruation is absent during the attack, and there may be a long period of delayed convalescence with amenorrhœa, eroticism, and impulsiveness.

A second variety is the Acute Delirious type. In such cases the confusion and loss of orientation may become extreme, and there is constant delirious chatter with sleeplessness, dry brown tongue, rapid and feeble pulse, and tendency to pass into the typhoid state with fatal result. In these cases the temperature is irregularly raised, not infrequently with rigors, offensive lochia, pelvic inflammation, abscess in the breast, pulmonary congestion or consolidation. In a third group the excitement is more of the Hysterical type with eroticism and less profound loss of consciousness. The fourth variety is acute Confusional Insanity, occurring chiefly in patients who have been much exhausted at confinement or have been in a low state of health previously. There is dreamy confusion, inability to answer questions or to grasp the environment, hallucinations, restlessness without excitement, aimless wandering about, refusal of food from inability to grasp its nature, and general resistance. Delusions are not as a rule marked. There is loss of control over the emunctories. These cases improve rapidly with treatment, but if neglected may pass, on the one hand, into a low delirious state, or into stupor and dementia.

Puerperal Melancholia may begin almost immediately after delivery, but most commonly more than a fortnight after. The depression may be of the religious type, with self-accusation of crimes or wickedness and delusions of perdition. Complaint of being 'unworthy,' or of having lost natural affection, or of having brought ruin on the family, is common. Occasionally the depression is hypochondriacal in type, with delusions about the pelvic viscera of being 'unnatural' or of being 'not human.' In another group there is terror at imaginary impending harm, dread of arrest, or fear lest the child be starved or injured. Delusions as to the child's health, such as that it is deformed or black, may lead to intense disgust with it and infanticide. In other cases there is great depression with apathy, loss of interest, and stupor. There may be either silence and downcast expression with loss of energy, or, on the other hand, restless agitation with constant noisy bewailing. In all these cases there is great risk of suicide, and frequent attempts are made. Sleeplessness, refusal of food, and personal neglect are constant, as are also amenorrhœa and cessation of the secretion of milk.

Chronic delusional insanity with organised ideas of persecution may originate in the puerperal state, the prognosis being then very unfavourable. In rare cases General Paralysis begins at this period.

PROGNOSIS.—About 80 per cent. of cases recover. Acute cases often recover quickly, and most of the maniacal cases are convalescent within 3 or 6 months. Patients suffering from melancholia are not placed under care as early as those with excitement, unless suicidal or homicidal symptoms appear early, and therefore take longer to recover. The prognosis is favourable so long as there is amenorrhœa and imperfect restoration of the general health, but bad when menstruation has been re-established and the general health restored without corresponding mental improvement. Death may occur in septic or acutely delirious cases or where prolonged maniacal excitement has led to exhaustion; although

some of the most severe cases with septic complications recover perfectly if treatment is undertaken promptly. The prognosis in the event of further confinements is commonly unfavourable, some women becoming insane after each labour.

TREATMENT.—Rest in bed during the early stages is of the utmost importance. Perfect quiet and freedom from all suggestion of home-worries are essential. The patient must be removed from home; or, if this is not feasible, the husband and child must be removed, and the patient herself efficiently nursed. The child should be at once weaned, and never, on any consideration, be left in the patient's care. She may neglect it so that it dies, or may kill it accidentally in excitement, or as the result of delusions or hallucinations, or in a sudden impulse. If left alone she may even destroy herself during maniacal excitement, without any previous indication of suicidal tendency. The physical state must be carefully ascertained, especially the condition of the uterus, the lochia, the breasts, and the temperature; for the possibility of septic absorption must not be overlooked. In the cases beginning early after confinement the uterus or vagina should be washed out, but in the later cases this is not necessary unless there is evidence of local disease. The patient must be fed liberally. If solid food is refused or inadvisable, abundant liquid food must be administered—if necessary, by the nasal or œsophageal tube (*see* FORCIBLE FEEDING). The bowels must be regulated; a brisk purge at the onset is often beneficial. In cases of profound exhaustion or delirious excitement stimulants are needed. Sleeplessness must be treated as in other cases of acute mental disorder, but food is more important than narcotics. The milder and transitory cases may be treated out of an asylum, but in all severe cases such care is necessary, and the earlier the patient is put under control, the earlier is recovery likely to follow. During convalescence tonics and emmenagogues are useful. The amenorrhœa, however, usually passes off as the general health improves. After recovery a period of complete rest is needed, and the risk of pregnancy should be avoided.

3. Insanity of Lactation.—It has been pointed out (*see* PUERPERAL INSANITY) that the borderline between puerperal and lactational insanity is an artificial one, and is usually fixed at six weeks or two months after confinement. It is, however, impossible to say that mental disorder, becoming evident three months after delivery and when lactation has become established, is not due in some measure to the remote effects of child-birth. In cases occurring in the earlier months of lactation both factors have to be borne in mind, while in those occurring later the exhausting effect of prolonged lactation is the principal exciting cause.

CAUSES.—(1) Inheritance of neuroses or of insanity; (2) Previous attacks of mental disorder, especially puerperal or lactational; (3) Defective general nutrition, as in women of the poorer classes, exhausted by frequent child-bearing and prolonged suckling.

FORMS OF MENTAL DISORDER.—The onset is generally gradual. At the beginning there is neurasthenia with vertical headache, sense of fatigue, loss of flesh, loss of energy, sleeplessness, anæmia, and constipation. Mental depression supervenes and soon passes into well-marked melancholia. In the experience of the writer, this is the

predominant form of disorder; but in many cases the depression is followed by maniacal excitement. This sequence is partly a matter of idiosyncrasy, and partly dependent on the amount of bodily exhaustion which is always present in these cases.

Melancholia from lactation is commonly marked by self-accusation of wickedness or neglect, dread of being 'lost' or ruined, and strong determination to commit suicide. The mother who dreads a horrible future may kill her child and herself to escape from its possibilities. One patient of the writer's heard voices, saying to her 'Why don't you kill the baby and your husband?' Hallucinations of the senses, especially of hearing, are common, and may influence the conduct. In some cases there is stupor with absolute refusal of food and impulsive suicidal attempts; in others, agitation and resistance to all attention.

Maniacal excitement is of the ordinary acute maniacal type with violence, destructiveness, confusion of thought, and incoherence. The acute delirious type is uncommon, and there is rarely a rise of temperature.

In a small proportion of cases chronic delusional insanity begins during lactation, and dementia may result, especially where there has been a long period of treatment at home, or the child has still been nursed long after exhaustion has set in.

The milk may cease to be secreted before or at the time of the onset of depression. Amenorrhœa is common in lactational insanity, and is dependent on the co-existing anæmia.

PROGNOSIS.—Recovery takes place in 75 to 80 per cent. of cases. There is much less danger to life than in puerperal cases, but the possibility of suicide must not be overlooked.

TREATMENT.—Anæmia and malnutrition being constant symptoms, tonics are needed in most cases as well as abundant feeding. In cases of melancholia especial attention must be directed to the bowels. Narcotics should, if possible, be avoided, and chloral hydrate and sulphonal should rarely be used. Sleep is more likely to follow gentle exercise, feeding at night, and the judicious use of stimulants. The immediate cessation of the exhausting drain of lactation is imperative, and the patient must be separated from her husband and child lest she kill them. The extremely suicidal nature of the melancholic cases renders treatment in an asylum almost always necessary; and this procedure is equally advisable in maniacal cases, as the form of the disorder never disappears rapidly, as happens in some puerperal cases.

R. PERCY SMITH.

V. Senile Insanity.—Senile mental decay is most likely to occur in persons of neurotic or insane stock, and to be accelerated in its onset by constant undue stress of work, or by the effects of alcohol, syphilis, or head-injury. In the great majority of cases the period from 60 to 65 years is that in which it first appears. In women the mental disorders of the climacteric, sometimes ending in dementia, mark the period of involution as occurring somewhat earlier than in men.

Insanity may occur in old age, and yet not be true senile insanity. Recurrent insanity dating from the period of adolescence may continue to recur in old age with intervals of comparative health, and recovery from such attacks may occur even in old age. In other cases senile decay may

be the immediate cause of mental disorder in patients who have suffered from insanity at other epochs or crises, as, for example, after parturition or at the climacteric. Previous mental disorder may have occurred in this way as much as thirty or forty years before, the patient having recovered and remained well till the onset of senility. Mental disorder is also met with in old age in association with cerebral lesions due to occlusion or rupture of blood-vessels, or in uræmia and other toxic states. It is impossible to distinguish from senile insanity all those cases due to some organic cerebral change, since the essential feature in senile decay is gradual failure of brain-nutrition with wasting and degeneration of its substance in association with rigidity of arteries and impoverished cerebral blood-supply. The term 'senile insanity' should, however, be limited as far as possible to mental disorders occurring for the first time in old age apart from gross cerebral lesions. Dementia is not by any means the only form of disorder met with at this epoch. Senile insanity is often erroneously spoken of as due to 'softening of the brain' and as being always incurable. There are, however, functional mental disorders in old age from which the patient may recover, as well as others dependent on permanent atrophic changes which end in dementia. The various forms will now be considered.

1. Melancholia.—Mental depression in old age may be the precursor of dementia, the initial stage of a maniacal attack, or the commencement of a definite melancholia. The earliest symptoms are generally those of fatigue or exhaustion. Loss of vigour and lack of application to the usual occupation is experienced, everything becomes a burden, thought is attended by painful effort, and attention cannot be concentrated or sustained. Sleeplessness and continuous anxiety concerning the trivial details of daily life follow, accompanied by loss of appetite, distaste for food, dyspepsia, constipation, and loss of flesh. The patient becomes irritable, especially in the home-circle, and, though needing rest, is unable or unwilling to cease from work. He accuses himself of being lazy, and tries to force himself to do more. Injudicious friends tell him it is 'nothing,' and advise him not to give way. He has noises in the head or headache, dreads impending evil, doubts his conduct or motives, and shows indecision and forgetfulness, loss of interest, increasing gloominess, and thinks of suicide. The disorder may remain as a 'simple melancholia,' passing off with rest, removal of the causes of stress, and treatment directed to the restoration of general health. In more severe cases delusions of wickedness, of being an unpardoned sinner or murderer, or of having caused disaster to his relatives or the whole world, are present. Long past sins, errors, or sexual irregularities are dwelt on, and their importance exaggerated. The attention fails, no work is possible, and either there is silence and complete lack of energy, or agitation and loud lamentations as to his state. Delusions of possession by the devil, and hallucinations of hearing and of other senses are common. The memory may not be markedly affected, but as a result of loss of attention recent events are not recorded as in health. Such patients may be able to give a clear account of their financial or domestic relationships though unable to direct their affairs. Sleep is always much disturbed, and the depression and agitation are worse at night or in the early morning.

Suicidal desire and attempts are very common, food is often refused, and there is neglect of personal cleanliness or of tidiness in dress. The patient wastes, his tongue is foul, his bowels confined, and his pulse hard and tense. In other cases, the delusions concern mainly the patient's financial and social position rather than his moral condition. He believes he is ruined and has dragged all his family with him, that he owes money which he can never repay, that he is a fraudulent bankrupt, that he has 'no money and no clothes' in spite of ocular demonstration to the contrary. Desire for death is common, and sometimes there is a wish to kill those dependent on him in order to relieve them of the impending ruin. Food is refused because it cannot be paid for.

Hypochondriacal melancholia is also common in old age. The patient may believe he is decomposing and giving rise to offensive vapours, or that he is dying or dead; or his complaints may be limited to the state of his brain, his viscera, or his sexual organs. He may feel that his brain is split or separated from the cord, that it is 'sobbing and moaning' or that there is a 'roaring pain' in it. There may be a feeling of suffocation or delusion that the heart has 'gone.' The abdominal viscera may seem diseased, the food 'will not pass,' there is 'no inside,' the bowels are 'lost' or 'completely obstructed.' Patients with delusions of intestinal obstruction are extremely suicidal and obstinately refuse food. The diminution of sexual power and desire associated with old age may be looked upon as a 'terrible disaster;' and there may be delusions as to the size, structure, and form of his penis, testes, urethra, or bladder. Such patients are often extremely suicidal and prefer death to the thought that they may have lost sexual power. Hypochondriacal melancholia is much less curable in old age than the before-mentioned form. In some cases the grouping of ideas of depression is not strictly limited to one variety, but, for example, hypochondriacal ideas are combined with delusions of perdition or ruin. In other cases depression is associated with suspicion of the motives of others, delusions of unfaithfulness, irritability, querulousness, and hostility. Such patients are likely to develop fixed delusions of persecution.

2. Delusional Insanity (Senile Paranoia).—This may occur primarily in old age and may exist in the depressed or exalted form, the latter being the less common. Delusions of persecution may develop slowly without marked depression, with ill-defined dread of injury to prospects, position, family or life, commonly with hallucinations of hearing. In old women delusions of sexual persecution may arise in association with vulgar or vaginal irritation. There may be belief that rape is attempted at night, or that plots are laid against the patient's chastity. With this there may be delusions of being drugged or chloroformed, of being watched through holes in the wall or through floors, of being X-rayed or of having electric shocks. The prognosis in these cases is very unfavourable.

3. Mania.—Mania may occur in old age as a functional disorder and be recovered from, or it may be the forerunner of senile dementia, or be associated with organic disease of the brain. Usually there is a short period of depression with sleeplessness and then gradually increasing irritability, outbursts of violent temper, resentment of opposition, eroticism, exalted self-feeling, and moral perversion.

In this state there may be sexual crimes subjecting the patient to criminal prosecution, or merely lasciviousness, obscene conversation and acts. Offers of marriage may be made to young girls or to designing women. The exaltation may show itself in impossible schemes for making money, and as a result of opposition to unreasonable schemes or expenditure. The patient may alter his will and leave his property to those who have no claim to it. There is vacillation, indecision, impulsive violence, confusion of thought, and lapses of memory. There is often great difficulty in treating a patient in this state, as the disorder may remain as 'simple mania,' and it may be very difficult to certify him as insane. With enforced rest and care, however, this condition may pass off, or it may recur, or pass into more definite 'acute mania' with excitement, noisiness, violence, destructiveness, incoherence, exalted delusions as to wealth, strength, and prospects, and dirty habits. Occasionally the disorder is of the confusional type, and in others extreme delirious excitement with complete loss of orientation, rise of temperature, and profound exhaustion ends rapidly in death. In all cases of senile mania the memory is more or less affected, in some only as a loss for the events of the attack, in others, especially those in whom the disease comes on in later years, it is permanently affected for both recent and remote events. There is far more risk to life, and danger of permanent damage to mind, in senile mania than in senile melancholia, especially in cases with marked arterial or cardiac degeneration, chronic renal disease, or gross cerebral disease. Senile mania may be recovered from, but there is almost always inability to resume business, and if there be not loss or weakening of memory there may be emotional instability, irritability, and perhaps loss of moral sense.

4. Dementia.—Dementia may arise primarily or be preceded by functional mental disorder. Its development may be spread over many years, mind and body failing together, or, in the course of a few weeks, it may end in profound decay of mind, general weakness, failure of nutrition, and death either from general asthenia or some local complication such as cerebral hæmorrhage or thrombosis, pulmonary hypostasis and pneumonia, cardiac failure, cystitis, or diarrhoea. In some the memory fails gradually, the patient passing comfortably into dotage, needing constant care but giving no trouble; while in others there is so much disturbance of conduct that constant restlessness, childishness, loss of sense of decency, impatience of control, and attempts to transact business for which there is no mental power, render care in an asylum not only advisable but necessary. The emotions are blunted, or if there is a momentary disturbance the effect passes away as the cause is forgotten. Hallucinations are rare in uncomplicated senile dementia.

The greatest failure is shown in the intellectual faculties. The memory for recent events is especially affected. Daily events make no impression, or are forgotten in a few hours or days. Localities or individuals make no impression, the patient sometimes not recognising his own family or house, nor even remembering his name, the names or number of his children, the extent of his means, and the people who ought to benefit by his will. All conception of time is lost, and those long dead are spoken of as if alive. Habits of cleanliness disappear, the patient perhaps exposing himself

almost unconsciously, or passing urine or feces in his clothes or in public, or indulging, apparently intentionally, in filthy habits. He may be easily led, and give away his money or sign documents at the instance of unscrupulous people, or may, on the contrary, be querulous, obstinate, and self-willed. Sleep is often disturbed by nocturnal restlessness or talking, and by a desire to get up. The appetite is usually good, the patient taking childish pleasure in his food. The handwriting may merely show senile tremor, or be absolutely unintelligible; it often gives evidence of the great loss of memory. Although sexual power is much diminished or lost, there is often in the early stage increased sexual desire.

The progressive nervous degeneration is shown by tremulousness, feebleness of grasp, and unsteadiness of gait, with eventual loss of power of walking even without definite paralysis. Eventually the patient becomes bedridden, his sphincters cease to act, swallowing becomes more difficult, general wasting is marked, the heart becomes feeble, and convulsive seizures may occur and close the scene.

In some cases the symptoms simulate general paralysis, but there is less tendency to loss of pupillary light-reflex, less characteristic affection of speech and handwriting, and greater liability to hemiplegia, than in that disease. True general paralysis may, however, occur in old age.

PATHOLOGY.—There is general wasting of the brain-substance with increase of subarachnoid fluid compensatory to the wasting. To the naked eye the cortex appears thin, pale or yellowish, and has lost its characteristic striation. Arterial changes are always marked. Extreme atheroma of the larger cerebral vessels, as also military aneurysms, are often found, as well as hæmorrhages and thrombosis. Microscopically there is seen to be extensive arterio-sclerosis or hyaline-fibroid degeneration. The neurons show degenerative changes of the primary type very widely spread, and in advanced cases some of them may have disappeared. There is also hypertrophy of the neuroglia throughout the cortex, and especially in the neighbourhood of the degenerated nerve-cells. Even if there be no previous evidence of a large hæmorrhage or patch of softening, the microscope will often reveal small extravasations and minute areas of softening. The dura-mater is often adherent to the calvarium and may show hæmatoma (*see* PACHYMENINGITIS HÆMORRHAGICA) on its inner surface. The pia-arachnoid is thick, milky, and opaque in patches, but strips readily from the convolutions. Degenerative changes are commonly found in other organs, especially the heart and kidneys.

TREATMENT.—Many of the milder cases of senile dementia can of course be treated at home if the means permit. The patient should at first be kept in bed so as to secure as much rest as possible and avoid further exhaustion of strength, and to relieve arterial tension and permit of the treatment of any local or general disease. Treatment must be directed to the elimination of any toxins, such, for example, as are associated with constipation, uræmia, excess in nitrogenous diet, or in alcohol, and to the complete cessation of mental work. The diet must be light, but nourishing and abundant. Regard must always be had to the question of testamentary capacity.

R. PERCY SMITH.

VI. Delusional Insanity.—**SYNON.** : Monomania, Paranoia.—The terms at the head of this section comprehend abnormal mental conditions which have puzzled every student of mental medicine. It is not every insane person suffering from delusions that can be described as suffering from delusional insanity. Persons so affected are either those who have sense-perceptions different from the sane, and most varied in their manifestations; or those who have fixed ideas which are not removed by argument, and which generally have some direct relationship to the preservation of the individual himself, or his environmental existence in society. Essentially the condition is chronic, and restricted to a fixed delusion upon one subject, but it is erroneous to think that the victim is of sound judgment and healthy feeling on all others. If there be one symptom which is characteristic of this condition, it is the presence of hallucinations—sense-impressions not excited by external stimuli—and mostly those of hearing. Together with sense-impressions wrongly interpreted (illusions) they combine to develop and form all possible varieties of delusions, and although delusions connote an impairment of the highest faculty of reason, in these cases there is generally no marked mental weakness. On the contrary there is much shrewdness and pertinacity, volition is strong, and the emotions are well under control, but during a considerable part of the progress of the disease, the patient is necessarily a source of considerable danger to himself and others. Much of the violence and homicide which is perpetrated by the insane before legal restraint is imposed is caused by this class, an important factor from the medico-legal point of view.

We prefer the title Delusional Insanity to its synonyms, since it gives us the most important characteristic fact established by actual observation. Monomania was the term applied to this form of mental aberration by Esquirol, who considered it to be in the main an exaltation; when the fixed delusion was accompanied with depression, he gave it the title of ‘lypomania,’ thus confusing it with some forms of melancholia. English alienists have mostly preferred Delusional Insanity, while Paranoia (*παρά, beyond; and νόσος, to understand*) has been accepted by continental authorities in Europe and America. The term Paranoia has been applied generally to all chronic cases of insanity, whether primary or secondary; and it has been made to include all borderland cases, eccentrics, cranks, grumblers, letter-writers, and crazy persons of all sorts, more especially those who avow their belief in symbolism and allegorical expressions and figures. Persons evincing unfounded and unreasonable credulity, those who believe in fetichism and who embrace mystic and occult dogmas have been brought into the same class, the reason being that an anachronism is looked upon as a morbid character; that a belief in alchemy, or in the power of magnetism and electricity, which was compatible with high intellectual ability several centuries back, is now a sign of atavistic degeneracy; that the acts of chronic lunatics are reversions to earlier conditions of the human race; and that the delusions of persecution so frequent in this class are a survival of struggles for existence which were necessary in ancestral days and lawless times, but which are now only an indication of perversion and degeneration. The real nature of the conditions included

in the term 'paranoia' is much disputed. Mickle states that all cases do not belong to the great group of hereditary neuroses. He describes the following divisions of paranoia: A, Depressed—(a) persecutory, (b) hypochondriacal, (c) querulous; B, Expansive—(a) ambitious, (b) religious, (c) erotic. Our own opinion is that this form—for which the term 'paranoia' should not be used—is of all others that in which heredity plays the chief part, having a tendency to make the disease apparent during one or other of the somatic crises through which the life of an individual proceeds, e.g. childhood, puberty, adolescence, the puerperal, climacteric, or senile epoch, there being a constitutional tendency to mental disorder of a delusional type. Although there is less emotional disturbance in this form than in any other variety of insanity, it is incorrect to state that there is none; for every action and every thought has a distinct fundamental feeling-tone of pleasure or pain, and the egoistic feelings which so predominate in these cases obtain such an ascendancy over the intellectual life that the personality becomes completely changed.

Having advocated the term Delusional Insanity, we include in this class all those who suffer from a chronic form of insanity, whether hereditary or acquired, whose delusions—dominating the whole mind—appear to be fixed and systematised, and in whom there is not the characteristic emotional exaltation of mania nor the self-depreciation of melancholia.

In the *hereditary form*, the delusions may occur at an early age, or at any period of life, and may evolve into a transformation of the intellect, the will, or the emotions and sensations. It is this group which gives rise to the so-called monomanias of the various faculties, impulses, and obsessions. Not infrequently there is among this class considerable eccentricity or ability amounting even to genius, or there may be flagrant moral depravity and a total lack of ethical feeling. In these cases the delusions may be polymorphic, but there is, as a rule, a freedom from hallucinations, and many make temporary recoveries.

In the *acquired form*, which is less common, the disease manifests itself during the best period of adult life (35–45). The manifestation is preceded by a period of incubation, lasting possibly for years, during which there is a morbid timidity, hesitancy, and uncertainty, and the onset is rarely abrupt; for there are no psychical or physical stigmata, and usually no impulses or obsessions. The delusions are systematised and the consciousness is confused and obscured through the multiplicity of the hallucinations, the patient explaining these as due to external intervention. The disease is chronic and progressive, but there may be lucid intervals, during which the person may infect his friends, or, not infrequently in asylums, those about him, upon whom he is able to impose his personality—*délire double*. The termination is generally a slightly marked dementia.

The varieties of delusional insanity are characterised in the main by either: (1) exaltation, or (2) ideas of persecution, and the delusions are based upon hallucinations or illusions of the senses. The hallucinations have been regarded as having their situation in the cortical centres, and are possibly due to an abnormal erethism of the cortex, and automatic excitation under unnatural conditions giving rise to a nervous discharge as if provoked by a peripheral

impression. The senses affected are usually the auditory, not infrequently also the visual and tactile—those upon which the most highly differentiated experiences depend, being the most used and most markedly evolved of the senses. This excitation reacts upon the higher psychical centres with all the reality of normal sensation, giving rise to painful delusions. Occasionally also the converse is true, and the hallucination is awakened in the perception-centre through the influence of a higher psychical region, so that the idea or a thought may awaken in a sensory centre a representative symbol, such, for example, as an auditory verbal image, followed afterwards by hallucinations of hearing. It frequently happens that patients will complain of humming, buzzing, or complex sounds, before the actual words develop, and one of the most common explanations offered by those affected when projecting these sensations outwardly, is that 'they' speak so low, or make sounds which are indistinct, coming from above or below, by day or by night. An 'echo' is often complained of by patients, 'their thoughts are being read and reverberated like an echo'; and acts committed by them are believed to be auditorily described and reproduced. A patient informs me that it is useless his writing to me, as what he writes is immediately repeated by 'them' and precipitated into my presence before his own statement is finished. While reading the newspaper one patient is accosted by unfriendly voices, and a duologue results between the voices (auditory lobe, lower centre) and herself (frontal lobe, higher centre). Sometimes the sensations may appear as internal voices, excitation of the auditory centre being propagated to the motor word-centre in physiological connection with it, giving rise to *motor verbal* or *psychic hallucinations*, the patient feeling the words being uttered in her own throat and mouth.

Sensory disturbances are not uncommon, and patients may be seen with weary persistence almost the whole day brushing off imaginary filth, fanning themselves from chemicals, or covering different parts of their bodies from powders, or objectionable liquids which are being thrown, blown, or flicked at and over them, to their intense annoyance and disgust, and always, so far as they know, without cause. Olfactory and gustatory hallucinations are infrequent. The unusual experience and secret character of these all-powerful and unceasing annoyances cause them to be explained by the immediate power of great natural forces, such as magnetism, electricity, waves of matter, hypnotism, telephones, and microbes, and to be carried out by powerful unrecognised agencies, such as devils, anarchists, murderers, witches, freemasons, &c. The patient (whose personality becomes completely altered, owing to these newly recurring sensory troubles and false ideas, contributed by the different morbid senses to consciousness) changes from passive self-defence, during which he endured his tormentors in silence, to active hostility (persecuted persecutors). It is at this period, and probably before legal restraint has been enforced, that the sufferer carries about his person lethal weapons, which he may use criminally against inoffensive persons; and it is at this stage that the reason, led by the influence of hallucinations of the senses, deduces both sensible and improbable conclusions side by side (partial insanity). It is not surprising that irritation from an unceasing round of imaginary outrages should cause

these unhappy persons frequently to change their environment (migrating mania of Foville), to write to conspicuous personages, or commit crimes to 'invoke justice.' Not surprising is it either that the victims of this form of insanity should occasionally end their miserable lives by suicide. A patient, a literary man under the delusion of continuous persecution, moved to a solitary spot on the bank of the Medway, whence again he moved to London, and in the middle of the night clambered along the parapet of his hotel, entering an adjoining house, where, anticipating a safe retreat, he was overtaken by the police, and brought up on a charge of burglary.

After an interval of unceasing annoyance for varying periods, and having been the imaginary target for every conceivable form of active hostility, the patient begins to look upon himself as the hero of an unjustifiable and irrepressible persecution—always suffering and never escaping—and he becomes by slow gradations a self-exalted martyr. In this transition stage from persecution to exaltation, during which his insanity appears to be recast, he appeals to mystic explanations to describe the great things that happen to him, he coins new words to express his sensations, he invents weird signs to free himself from the persecution of unseen agencies, and he draws symbols of compasses, skulls, books, stars, angles, diamonds, &c., to exorcise spirits or remove witchcraft and sorcery. Then follows a worn-out neurasthenic stage of exhaustion, when there may be mental torpor, which he explains by accusing unknown persons of stealing his brains or subjecting his thoughts and torturing his body.

This type of insanity, composed of several analogous forms of psychosis, is not very common in asylums, for out of 2500 cases (1450 females, 1050 males) in Claybury, delusions of persecution were only markedly present in less than 20 per cent. of the females and 16 per cent. of the males, and exaltation with systematised delusions in 9 per cent. of the women and 6 per cent. of the men.

ROBERT JONES.

VII. Imperative Ideas.—SYNON.: Fr. *Obsessions mentales*; Ger. *Zwangsvorstellungen*.

DEFINITION.—A pathological state in which the mind is dominated by one idea, compelling the patient to do something or avoid something.

Mental besetments or obsessions are very significant symptoms, occurring, as they always do, in neurotic subjects. They vary greatly in character, and though apparently trivial at first may subsequently assume great importance. Many are of a simple character: one person is obliged to count the gas-lamps or the numbers of the houses; another must tread the pavement carefully, avoiding the joints or stepping on them; another must touch certain things, like Dr. Johnson, who touched the posts as he walked the streets. Some must spell the names over shops and go back to see if they have spelled them right. Others are dominated by various fears: they cannot look down from a window, a cliff, or other high place without a desire to throw themselves down; or cannot sit in a crowded room unless they are close to a door; or are in constant dread of touching or having touched something dirty or infectious, and spend their lives in washing their clothes or hands. This is often carried to extremes, the sufferer fearing not merely dirt but disease, such as syphilis, or defilement of

his food, so that he will eat nothing except what he cooks himself. Such ideas overwhelming a man's whole life, though perhaps absurd at first, render him quite incapable of taking care of himself or his affairs.

Though these besetments vary greatly, they may roughly be divided into those characterised by a desire or *impulse* to do something absurd or foolish; and those arising from a *dread* or doubt—a doubt as to whether the patient has done right, a desire to do it over again, or a dread that something is going to befall him. The most trivial matters assume the greatest importance and render life not worth living. If we look for the very beginnings of such obsessions we find them in the *tricks* which some display—tricks with the hands, tricks with the feet, tricks of gesture, walk, or dress; or in fear of the dark, fear of robbers, ghosts, and the like. All such persons, whether adults or children, are neurotic, many displaying an insane diathesis which fully explains the phenomena. No such obsessions, however slight or absurd, should be neglected. At the outset they may be overcome; later they gain the mastery. All tricks in children should be sternly repressed, and their fears should be judiciously treated, not by ridicule but by example and encouragement. The bringing up of such children is of great importance, and no pains should be spared to repair their degenerated constitution. If the tricks and besetments do not begin until adult life, they equally demand attention. At first a patient may overcome them, and should be urged in every way to resist the impulse and to put aside the fear; and, according to the material to be worked on, we may succeed or not. Sometimes the idea may change, and in place of one fear another may arise, and unfortunately in many cases there is an advance from slight to graver forms, and the patient is possessed by impulses of a violent kind, or fears which render him dangerous to himself or others.

Mickle points out that the best description of some forms of obsession is to be found in the pages of *Lavengro*, by George Borrow, who, he believes, under the guise of different personages in the romance described his own case.

Not long ago a lady from the fear of contamination would take no food but what she ate outside the house or cooked herself; and washed her arms and hands with carbolic acid till they were a mass of sores. Clouston narrates the case of a medical man who placed himself under restraint because he was haunted by an impulse to commit murder or suicide. All such should be carefully observed and recorded, as they throw great light on criminal cases and on the evidence to be given in courts of law. See IMPULSIVE INSANITY.

G. F. BLANDFORD.

VIII. Impulsive Insanity.—Violent acts are committed under an insane impulse by many patients whose insanity is plain and acknowledged. They may be done under the influence of delusions or hallucinations; but the term *impulsive insanity* is commonly applied to a disorder manifested, not by delusions and similar symptoms, but by acts of violence to which a patient is driven by blind, uncontrollable, and morbid impulse, whereby the will and the reason are overpowered for a longer or shorter time. These are for the most part acts of *suicide* or *homicide*; and in connection with the

latter, great controversy has arisen as to the responsibility of persons committing them. Here, as in moral insanity, there are no delusions; frequently no change will have been detected in the individual prior to the act, nor will there be observers of it. And it is a fact that the impulse may be satisfied and exploded in the act, and having thus found a vent may be felt no longer, at any rate for a time.

Impulsive acts of violence are committed continually in asylums, and many patients on recovery have related the history of the impulsive feeling and their difficulty or, it may be, their failure to control it. A medical man placed himself in Morningside Asylum, being haunted by a constant impulse to commit homicide or suicide which he feared he could no longer resist. He had been overworked, but with rest and treatment recovered, returned to his hard practice, and relapsed. A youth nearly murdered his mother from homicidal impulse which he vainly tried to resist. He had been peculiar from twelve years of age, and noticed as such by his fellow clerks. Such insane impulses may exist without apparent symptoms, and without discoverable delusion or hallucination. They are akin to the imperative ideas or obsessions which beset some people (*see IMPERATIVE IDEAS*). Such tricks in the neurotic may develop till the *obsession* becomes a *possession* and rules the whole life. Thus may these persons advance to insanity, their higher centres losing control more and more, and these absurd and harmless but imperative ideas becoming delusions or leading to insane acts.

Many acts of violence are committed through delusion or hallucination, though this may not be recognisable without much searching, or not till after a long time. Orange mentions a woman who murdered her child and was acquitted as insane, the act being looked upon as one of sudden uncontrollable impulse. But at Broadmoor she confessed that she did not want to live and remembered that she thought it would be the right thing to kill the children before she killed herself. The act was committed under a delusion. A large number of insane and apparently impulsive acts arise from hallucinations of hearing, 'voices' as they are called. Many a murder has been committed, the victims being perfect strangers, because the lunatic has heard a 'voice' calling him filthy names or commanding him to kill. These 'voices' become a terrible persecution and cause intense anger against the unknown persecutors. And if on some occasion the patient fancies that he hears them from a chance bystander, he may murder him not from an uncontrollable impulse, but from the delusion. Yet the hearing of such voices is often carefully concealed and may require close search.

The medical witness will in the case of homicide carefully examine and pay special attention to the following points:—

(a) The nature and character of the act must be noted. The presence or absence of motive may often assist us. When the victim is near of kin and dearly loved, suspicion of insanity will at once arise. When, on the other hand, it is a perfect stranger, never before seen, where there has been no previous meeting or quarrel, the same suspicion will arise. The method of the act may guide us somewhat, but not much. There may be premeditation, though generally there is not.

(b) The demeanour of the prisoner after the act may assist us. Were there, or were there not,

attempts to conceal it, or to escape detection and arrest, and, if so, what was the nature of them? What was said in explanation? It sometimes happens that there is complete unconsciousness or forgetfulness of what has occurred, and we may then strongly suspect the presence of epilepsy.

(c) We must closely inquire into the family history, and shall often find that, in cases of impulsive homicide, the family of the accused has suffered with insanity. And where this is so, we may also find that from youth the accused has been deficient and weak in intellect, or odd and eccentric. The weak-minded, in fact, may be grouped in a special class of homicides. As there is a weak-minded moral insanity, so there is a weak-minded impulsive homicidal insanity, the sufferers from which have not infrequently been hanged, their insanity not having been sufficiently marked to absolve them from legal responsibility. Fits in childhood may contribute to this state. And throughout, at the age of puberty, or in adult life, there may be slight but sure indications of the insanity that has been inherited, which are displayed in an impulse to mischief, homicide or suicide, even as in others the tendency is shown in ordinary attacks of insanity.

(d) We must look very closely for symptoms of a history of epilepsy. Such indications as nocturnal micturition or a bitten tongue may guide us to the truth; while in acknowledged epileptics it may happen that the homicidal attack takes the place of the ordinary convulsion, and without the occurrence of the latter there may be a period of unconsciousness and unconscious action lasting, perhaps, for days. *See EPILEPTIC INSANITY*.

The occurrence of one homicidal attack of a strange or anomalous character may make us fear its recurrence; and when we have to examine a criminal who has committed one act of this kind, it is important to inquire whether he has ever at any former time done any sudden act of violence of a similar description. For this reason such patients should not be released from an asylum, except under special precautions.

G. F. BLANDFORD.

IX. Mental Stupor and Catatonia (*κατά, down; τένος, tension*).—Stupor is a state in which the outward signs of mentation are in abeyance.

ÆTIOLOGY.—This is the same as in other forms of insanity. Among the stresses most likely to produce this condition are mental or moral shock, and sexual excess in an unstable individual.

CLASSIFICATION.—There are three forms of stupor recognisable: (1) Anergic Stupor; (2) Delusional Stupor, or Melancholia Attonita; and (3) Secondary Stupor. The terms 'primary dementia' and 'acute dementia' are sometimes used for certain cases of anergic stupor. These terms are misleading because they erroneously bring these curable conditions into the category of true dementia which is permanent weak-mindedness and an incurable condition.

SYMPTOMS.—For brevity and convenience we shall take the symptoms of *anergic* stupor and *delusional* stupor together, merely pointing out the distinction between them, for not uncommonly the two forms are observed to interchange in the same patient. The mental symptoms are to a large extent negative in character. The patient stands or sits unoccupied with a vacant expression, and he

takes no apparent heed of his surroundings. There is marked mutism, and saliva not uncommonly dribbles from the mouth. The pupils are dilated. Food is refused, and when placed in the patient's mouth may remain there for a long time unmasticated. The extremities are cold and blue and frequently œdematous. The pulse is feeble, and respiration is slow and shallow. The evacuations are passed involuntarily, and the patient usually suffers from constipation. Nutrition fails rapidly as in many other forms of acute mental disorder. The hair and nails become brittle, pustules and small superficial abscesses may occur, indicating nutritional change in the skin. There is amenorrhœa in the female. At night the patient may remain quietly in bed, but sleep is usually very deficient. The memory is said to vary in the two conditions. In the anergic form the memory is almost absent, so that on recovery the patient remembers little or nothing of his illness; on the other hand with the delusional type memory may be fairly accurate. Delusions may or may not be present in the anergic form, but they form an important symptom in melancholic stupor. Probably the condition of the patient is one in which his mind is preoccupied and dominated by some delusion which causes him to look upon those about him as his enemies and persecutors. In anergic stupor the patient, though dependent on others to look after him, is unresistive and apathetic; he can be dressed, fed, and attended to without difficulty: on the other hand a case of melancholic stupor will usually resist everything that is done for him.

In *secondary stupor* or post-maniacal stupor the symptoms closely resemble those already described, but differ in that the stuporose condition is more transitory and usually less severe. In these cases the patient can usually be aroused, and often be persuaded to speak; in fact in its milder form the condition is purely one of lethargy and apathy.

Stoddart supports the above classification of stupor, which is based on mental symptoms, by his observation of certain physical signs in these conditions. Delusional stupor (*Melancholia attonita*) in common with other forms of melancholia is characterised by a form of rigidity which mostly affects the muscles of the trunk and larger joints. Cases of post-maniacal stupor or secondary stupor show a form of anæsthesia which affects the limbs, especially at their peripheral parts. In anergic stupor the above-mentioned rigidity is wanting, but in its stead we find either the cataleptic condition known as *flexibilitas cerea* or complete flaccidity. Similarly the anæsthesia described above is wanting in anergic stupor; these patients do not react to painful stimuli, but this is not due to true analgesia, since the patients on their recovery recall vividly the details of the investigation.

Catatonia.—This disorder was first described by Kahlbaum in 1874. It is a form of insanity by no means common in Great Britain. It is divided into stages. During the first few weeks the condition is one of ordinary depression, followed sooner or later by excitement. This excitement is shown either by a period of acute mania or by a state of agitated melancholia. The second stage is ultimately followed by a condition which is marked by immobility with rigidity (*attonita*); this is the characteristic stage of the disorder. There is now absence of all spontaneous movement, and with this more or less complete immobility. Passive

movements are met by a powerful resistance (*negativism*). This rigid immobility not infrequently is from time to time interrupted by movements incessantly repeated in an automatic manner (*stereotyped movements*). Throughout this stage mutism is a characteristic symptom, but this mutism may be broken by periods during which the patient repeats words or phrases in a monotonous and automatic manner (*verbigeration*). The other symptoms of catatonia resemble those found in ordinary stupor. Vaso-motor disturbances are common, respiration is usually shallow and slow. There is not infrequently refusal of food. If the patient does not recover he generally passes on into a condition of ordinary secondary dementia, but in addition the symptoms of stereotyped movement and verbigeration as a rule persist.

PROGNOSIS.—The prognosis of anergic stupor is fairly good, although in a limited number of cases the recovery is delayed for as long as three years. In the delusional type of the disorder and catatonia, the chances of recovery are not so hopeful. The peculiar liability to phthisis must never be lost sight of in dealing with all forms of stupor. Cases of secondary stupor usually get well: in fact this condition is commonly a stage on the road to recovery.

TREATMENT.—The treatment of all forms of stupor must, during the early stages, be stimulating and supporting. Food must be generous and plentiful; and if nourishment is refused artificial feeding must be resorted to without delay. Constipation must be relieved, and the bowels regulated. In some cases massage and passive exercises have proved beneficial: in others cold or tepid shower-baths may be used with advantage, but these must not be persisted in if there is no good reaction following the bath. Turkish baths of short duration may be also tried. General tonics should be prescribed, and, if necessary, stimulants. Maltine and cod-liver oil should be given once or twice a day if there is loss of weight. MAURICE CRAIG.

X. Moral Insanity.—SYNON.: Fr. *Manie sans Délire; Folie Raisonnée; Monomanie Affective*; Ger. *Gemüthswahnsinn*.

Under the names of *moral insanity*, *emotional insanity*, *impulsive insanity*, *affective insanity*, has been described the disorder of certain patients, which is manifested by insane actions and conduct rather than by insane ideas, delusions, or hallucinations. Such persons are sometimes said to be of whole and perfect intellect, though unsound in the moral and emotional part of their brain. They come under the notice of medical men not so much for purposes of treatment as for diagnosis. Their conduct being chiefly displayed in foolish or violent acts, they require to be restrained, and the question arises, Is this conduct badness or madness? Are they responsible for it or not?

Objections have been made to the term *moral insanity*, and it has been proposed to call it *insanity without delusion*, inasmuch as it does not constitute a defined and well-marked disease, like acute delirium or general paralysis. Every patient may at one time or other be 'morally insane'—that is, may not have reached the stage of delusions, or may have recovered from it, and every patient may commit 'impulsive' acts of violence, whether his insanity is displayed in other ways or not.

By *moral insanity* is to be understood a disorder of mind shown by an entire change of character

and habits; by extraordinary acts and conduct—extravagance or parsimony; false assertions and false views concerning those nearest and dearest—but without absolute delusion. Such a change may be noticed following any of the ordinary causes of insanity. It may follow epileptic or apoplectic seizures, or may be seen after a period of drinking. Its approach is gradual, as a rule, rather than sudden; and the extraordinary character of the acts may not at first be so marked as subsequently. Friends wonder that a man should say this or that, or should do things so foreign to his nature and habits, but some time may elapse before they can convince themselves that such conduct is the result of disease; and the acts may be such that many will look upon them even to the last as signs merely of depravity. Such insanity, of course, varies in degree. When it is well marked, and the conduct is outrageous, there will be no difficulty in the diagnosis. But it may be less marked, and may consist of false and malevolent assertions concerning people, even the nearest; or of little plots and traps to annoy others, in which great ingenuity and cunning may be displayed. And there will be the greatest plausibility in the story by which all such acts and all other acts will be explained away and excused. It would seem sometimes as if a universal badness had taken possession of the individual, yet a badness so inexplicable that it can only be looked upon as madness. Where we can ascertain that this condition of things is something which has come over the patient, being formerly absent, and that he is altogether changed, we may suspect insanity. But much examination and opportunity for examination may be needful before we can sign a certificate, for such people are often very acute, and quite on the alert. They have no scruples about falsehood, and will deny or justify everything with which they are taxed; and where the insanity is manifested in conduct, the medical man may never be a witness of it, and is obliged to receive on hearsay that which the patient strenuously denies. Careful inquiry, however, will probably reveal the origin and cause of the change. There may have been a period, though short, of acute insanity—as acute mania or melancholia—which passed away and left this as a permanent condition; or it may be the precursor of a more advanced stage of the disorder, one marked by the ordinary symptoms, as delusions and hallucinations. If the change has been rapid and progressive, if the sufferer has become more and more outrageous and eccentric, it is likely that in a short time unmistakable insanity will be displayed; but some cases progress slowly, and steps for restraint have to be taken before anything like delusion is to be found. It may be necessary to prevent a man from squandering all his property—a common symptom in this variety—or from wandering from home and absenting himself no one knows where, or keeping low company. And when a man previously quite sober suddenly takes to drinking, the question may arise whether this is not the effect of insanity. Great difficulty may be found in proving the latter, but unquestionably it is often the case. Here, however, if the habit is indulged in, the patient will most likely get rapidly worse, and then restraint will be more easily enforced. An alteration in habits and conduct is not infrequently found in the aged, and may be manifested by drinking or debauchery or general emotional change. Here a

comparison with the former life of the individual may assist the diagnosis. See SENILE INSANITY.

Moral insanity may be the precursor of general paralysis; it may also be the sequel and result of a more severe insanity; it may be the outcome of apoplexy, or of a blow or other damage to the brain. It may be one of the alternating states of the so-called *folie circulaire*. Here a period of depression alternates with one of excitement, gaiety, self-glorification, or irascibility, and the latter may be one closely resembling that usually called moral insanity, and evidenced by exaggerated conduct and absurd acts. It may follow a simple attack of epilepsy, or may be the precursor of such attacks, being a part of that epileptic condition known as masked epilepsy. See EPILEPTIC INSANITY.

The one constant and marked feature of this insanity is the absence of delusion; but we are not on this account to argue that the intellect is sound. There is often great acuteness and cunning displayed by such patients, yet along with the cunning there may be the most silly and foolish conduct. Often there is great acuteness shown by those who have delusions, but because of the latter we say their intellect is disordered. Yet it is proof of disorder of the intellect if a patient spends his capital as though it were income, defends and justifies the most outrageous acts, and cannot be made to see that they are outrageous. Close examination will probably reveal the fact that there is very considerable intellectual lesion in these cases. There is a want of the power of attention and concentration of ideas on a subject. A patient commences a story of his grievances, and in two minutes is far away from his theme, and is boasting of his virtues or conduct, and no amount of bringing back will enable him to give a definite and succinct account of what he has to complain of. Such rambling is a marked symptom of this insanity, and a strong indication of a weakened intellect.

There is one more form of moral insanity which is, perhaps, the hardest of all to diagnose and estimate. It is the congenital moral defect occasionally met with in persons who have been from birth odd and peculiar, and incapable of acting and behaving like other people. They can hardly be called idiots or imbeciles, for they may exhibit a considerable amount of intellect and even genius in certain special directions. We shall generally find that they are the offspring of parents strongly tainted with insanity, epilepsy, or alcoholism, and many in childhood are the subjects of fits, chorea, or other neuroses. They are incapable of being instructed like other boys and girls; are often frightfully cruel towards animals, or their brothers and sisters; and seem utterly incapable of telling the truth or understanding why they should do so. The offspring of intemperance and poverty, and often lacking the special training which is essential, they become the chronic inmates of prisons, and it is most difficult both for medical and other prison officials to say how far they are responsible, and how far not. See IDIOTCY.

G. F. BLANDFORD.

XI. Insanity in special Diseases.

Brain, Tumours of the.—Brain-tumours in some cases affect the mind to a considerable extent. The symptoms depend on the parts of the brain involved. Generally with advancing pressure due to growths within the skull, sluggishness, indolence,

loss of will-power, and general disregard of the surroundings and of conventional duties are noticed, along with somnolence, disregard of cleanliness, and irritability. It has been noted by Wilks that patients with brain-tumours and coarse brain-disease often have their hands placed over the genitalia. Optic neuritis and implication of cranial nerves assist diagnosis. See BRAIN, TUMOURS OF.

Cholera.—Few cases are admitted into asylums during epidemics of cholera, but there are many cases of acute mental disorder associated with the disease. Acute mania may arise or simple melancholia, often of the stuporose or hypochondriacal type. Delusional insanity with ideas of persecution has also been described, and permanent mental weakness has been seen by the writer following an attack of cholera. In most cases where the disease itself has passed the patient recovers from the mental sequelæ.

Chorea.—Chorea has been shown by Gowers to occur more in neurotic families than in others. In nearly all cases there is more or less marked mental weakness, which depends on the duration of the disease, the severity of the movements, and the physical health of the subject. Age too has a distinct influence: the younger the child the greater the danger of permanent mental defect. The best treatment is time and healthy surroundings, with organised gymnastics; sea-air and simple diet are also useful. In some of the older cases of chorea a delirious state may arise, and this may pass into true delirious mania of the most dangerous type. Death generally occurs in such cases; the only hope lies in the administration of abundant fluid food with stimulants. As an after-effect of the delirium there is great tendency to some mental weakness which is allied to the weakness of neurasthenia, and needs rather rest than stimulation.

Diabetes.—Diabetes is common in neurotic families; thus insane or highly neurotic parents often have diabetic offspring, and diabetic parentage may lead to neurosis in the next generation. With diabetes there may be acute mania, but it is more common to meet with mental weakness and melancholia of a hypochondriacal type. In some diabetics, when mental disorder appears, the sugar disappears from the urine, at least for a time. Diabetes not infrequently occurs in general paralytics. In these the sugar may disappear for a time, but returns when the patient passes into dementia. In insanity associated with diabetes treatment must be directed to the latter disease.

Disseminated sclerosis may give rise to symptoms of mental disorder resembling in many ways the symptoms of general paralysis of the insane, especially that form which has been called 'adolescent' or 'developmental general paralysis.' The chief characteristics which distinguish disseminated sclerosis are the absence of syphilitic history, the peculiar staccato speech, the rhythmic tremor of the upper extremities, and the irregular paralysis of certain groups of muscles.

Exophthalmic Goitre.—The true relationship of this disease to insanity is not fully understood, but we recognise first the occurrence of Graves's disease in neurotic families; next, that it may definitely affect the nature of the symptoms; and finally, that there are a few cases in which the mental and bodily disorders are directly connected. It is noteworthy that only one or two of the characteristic symptoms of exophthalmic goitre may be

present along with mental disorder, as in some cases of general paralysis; and also that the mental and bodily symptoms may be present together in recurring mental illnesses. Owing to the peculiar aspect produced, young people may think that they are noticed and spoken about by others. This may lead to melancholia or to delusional insanity. In some cases acute enlargement of the thyroid gland, with accelerated pulse and prominent eyeballs, is associated with restless, sleepless activity, which passes into acute mania of either the hysterical or the delirious type. Such cases not infrequently die of some intercurrent trouble, such as diarrhœa. The treatment must depend on the symptoms of the general disease: feeding, rest, and sedatives, such as belladonna and hyosine, seem to act best.

Gout.—In many cases associated with the onset of acute attacks of gout there is some marked intellectual change. In some there is depression, while in others there is unusual intelligence and brilliancy. Suicidal tendencies develop in some persons before the attack. In many cases of so-called 'suppressed gout' there is a considerable amount of mental depression, generally of a hypochondriacal type. Some patients believe they are dying, or that they have cancer or some certain cause of death. Others have vague but most serious mental depression; and in some of the most determined suicidal cases suppressed gout is the exciting cause. Patients may be relieved from mental depression immediately on the appearance of articular gout. In such cases purgatives and free use of Turkish baths are the best methods of treatment. Occasionally during gout acute mania, often of the delirious type, may arise. This is what used to be called 'Metastasis of gout to the brain.' In these cases the prognosis is very grave, and the treatment must be the free administration of stimulants and chloral hydrate.

Heart-disease.—Intellectual capacity is related both to the brain and to the circulation; and, as might be expected, irregularities of blood-supply may be associated with irregularity of cerebral function. There is, however, no definite connection traceable between forms of heart-disease and mental disorders. It appears to be true that with mitral insufficiency there is commonly a tendency to mental depression (Dickson). The writer has frequently met with a true melancholia with suicidal tendencies in patients with valvular incompetence. Restlessness, with mental exaltation, may be found in cases with aortic trouble, and great hypertrophy of the left ventricle. Temporary rapidity of the heart's action is met with in some patients with mania, and the pulse is generally slow in stuporose states.

Influenza and other Febrile diseases.—Soon after the first of the recent epidemics the writer described influenza as a frequent exciting cause of mental disorder, and this has been confirmed by general experience. Influenza may set up various forms of insanity which may occur at various periods of life; there is no specific form deserving the name of influenzal insanity. Influenza may be the predisposing or the exciting cause of an attack. It acts most prejudicially in those of neurotic stock, those who are already degenerating, and those who have suffered from previous nervous illnesses; hence it specially affects the intemperate. Probably the influenzal poison acts directly on the brain; thus it may relieve existing insanity or modify the attack.

It produces mental depression, and the various forms of melancholia more frequently than mania; it may, however, start acute delirious mania, and there is serious danger of this developing when the attack of influenza is associated with childbirth. Greater danger to the nervous system apparently arises from frequently repeated attacks than from the existence of a high degree of fever. This form of disease frequently leads to suicide; it may start delusional insanity, with ideas of persecution in the organically neurotic and in the drunken, and may thus lead to crimes depending on the delusions. Its treatment should be chiefly expectant, and the patient should be placed in healthy and invigorating surroundings. Sea-air is very useful, as is also cold fresh air in mountains during the summer. Free administration of alcoholic stimulants is beneficial, and for the sleeplessness which is one of the prominent symptoms alcohol, chloral hydrate, and paraldehyde are among the most useful remedies.

What has been said of influenza holds good almost exactly of the disorders of mind following other fevers. They affect chiefly those who are of neurotic or of degenerating stock, or who by alcoholic or other excesses have induced in themselves a condition of instability. Any fever accompanied by delirium may start delirious mania, or in predisposed subjects may set up any other form of insanity to which the patient is liable. The height of the fever has some influence, but this too depends on the neurotic tendency of the patient. Prolonged high temperature leads in these cases to more or less complete and enduring dementia. Such attacks may occur with measles, and several have been described as following mumps. Pneumonia may start neuroses, and, in the writer's experience, scarlet fever is particularly dangerous in this relationship. Small-pox is also dangerous during the acute stage, and later the disfigurement may lead to a morbid self-consciousness which tends to develop ideas of suspicion, ending in delusional insanity. Typhus, with its acute onset and high fever occurring in young persons, has a very powerful effect in upsetting the mental balance. In very many cases the disorder is slight and temporary, so that the only evidence of it is loss of recent memory, general irritability, slight tendency to hallucinations and emotional weakness. Fevers occurring in the acutely insane modify the symptoms for a time, but do not as a rule check them. In cases of subacute mental disorder, or where the acute stage has passed, fevers may lead to complete cure; in chronic cases the fever may produce temporary relief; but as a rule, when the fever has subsided the mental symptoms recur. In patients subject to recurring mania the fever may start an outbreak.

Malaria.—There are several conditions under which insanity occurs with malarial symptoms. With the febrile stage there may be restlessness, sleeplessness, depression, and later acute mania of the most dangerous delirious type. The acute symptoms may recur at regular intervals associated with the febrile stage of the disorder; in these cases the headache, the full arteries, and the congested face mark a difference from simple mania. The mania may suddenly break out in malarial patients, but in such cases alcohol, syphilis, or exhaustion may have been an associated condition. After the passing off of the acute symptoms of malaria there may appear mental disorder of various forms. These cases as a rule belong to the unstable or neurotic class, and

among them stupor is more frequent than mania. In some patients malaria leaves behind it some mental disorder or nervous disease; thus sleeplessness or neuralgia may occur, or definite delusions of a limited nature, which may or may not be associated with hallucinations. Though quinine may be tried in all these cases it is common to find that it fails to do good; and prolonged courses of arsenic with baths and general hygienic treatment will have to be tried.

Myxedema.—It is noteworthy that with defect or disease of the thyroid gland mental deficiency is noticed in certain cretinoid idiots, who assume an aspect like that of the myxedematous adult. In the latter, with the increase of the symptoms of myxedema, there may be dulled perception, slow reaction, and consequent mental confusion. Patients thus misunderstand what is said and what is taking place near them, and may believe that things are done to annoy them. Hence ideas of plots, conspiracies, and the like arise. In some cases this may be followed by or associated with exaltation of ideas, the patient believing himself to be royal or great. There is slow progressive loss of will-power and of control, but most of all progressive loss of memory and of power of taking interest in surroundings; this leads on to dementia, in which state the patient may live long. The writer has met with a fairly large number of elderly women who, with progressive loss of mental power and with the presence of some delusions, have many of the symptoms of myxedema, though in a modified degree—a myxedematous degeneration rather than myxedema. In all cases with any of the symptoms of this disease it is well to try the effect of thyroid-treatment; but this must be done with care, for some of these patients, though apparently tolerant of large doses, yet after its continued use fail in general health and appetite. After administering the drug for a fortnight it is well to discontinue it for a few days, or even for a week or more, before giving it again, thus allowing short intervals between the periods of treatment.

Pellagra.—This disease is rarely met with in England, and is supposed to occur from eating diseased maize. It rarely runs its course without the appearance of some mental symptoms, and is often the starting-point of one or other form of mental disorder. It causes marked melancholic symptoms, with general sluggishness of thought, feeling, and reaction. It leads in some cases to delusional insanity, with hallucinations of the senses, but more often passes into temporary stupor or partial dementia. Suicide is common in these cases, and it has been said there is special tendency towards death by drowning. In some cases the disease has much the aspect of general paralysis of the insane, but there is little or no affection of speech, and the patients rarely show any exaltation of ideas. Change of diet and of surroundings leads to recovery in most of the cases, but in some permanent mental weakness arises.

Phthisis.—Clouston was one of the earliest to point out the special connection of phthisis with insanity. It had long been recognised that an undue number of deaths from phthisis occurred in asylums. This is probably due chiefly to direct contagion, but it appears that the neurotic afford a specially suitable nidus for the development of this disease. In phthisical insanity the whole aspect of the pulmonary disease changes. The patients com-

plain little of cough, expectoration, and the usual troubles; instead of being buoyant they are generally disagreeable and complaining, readily believing that persons about them annoy, worry, or interfere with them. They often refuse their food, and say poison is in it. It is still doubtful at what stage of phthisis the mental disorder generally occurs. Some think that it is most common in the earlier stage—that of consolidation of the lung, and it certainly is true that often with the development of active bodily signs of phthisis the mental disorder passes off. There is a form of alternation, the mind being better when the lung-disease is making progress, and worse when the pulmonary symptoms are dormant. The new modes of treatment seem to be the best for patients who are both insane and phthisical—change, healthy surroundings, fresh air, and simple living. The hotter climates do not suit these patients so well as colder regions.

Sunstroke.—Great difference of opinion has arisen as to the part played by insolation in producing insanity, but it appears that though a great deal of mental disorder really depending on alcohol, syphilis, or other causes of degeneration has been attributed to sunstroke, yet a certain number of cases of insanity actually depend on this cause. Langdon Down believed that a number of children of Europeans who were born or passed some time in the tropics became idiots or imbeciles as the direct result of heat or sunstroke; such patients frequently had fits and became restless, troublesome, and without moral sense. Hyslop thinks that a certain number of the insane become so as a direct result of the sun. The difficulty is to decide between general paralysis, alcoholic insanity, certain forms of dementia and syphilitic brain-disease, and effects of the sun. In true insanity depending on insolation the symptoms are generally those of progressive dementia, resembling general paralysis; but instead of developing, these symptoms remain stationary for years or even pass off more or less completely. It is possible that many cases of cured general paralysis have really been of the type described. In some of the cases due to insolation the mind remains feeble, but efficient, for many years. No special treatment can be followed: rest, freedom from alcohol, and avoidance of the sun's rays and of heat are the chief indications.

G. H. SAVAGE.

INSENSIBILITY (*in*, not; and *sentio*, I perceive).—This word signifies either loss of consciousness, or merely loss of sensation in a particular part of the body. See CONSCIOUSNESS, Disorders of; and SENSATION, Disorders of.

INSOMNIA (*in*, not; and *sonnus*, sleep).—Want of sleep, or sleeplessness. See SLEEP, Disorders of.

INSPECTION (*inspicio*, I look upon).—See PHYSICAL EXAMINATION; and NECROPSY.

INSTALLATION (*in*, into; and *stilla*, a drop).—The method of applying remedies to a part in the form of drops. Installation is chiefly employed in connection with the eye.

INSUFFLATION (*in*, into; and *sufflo*, I blow).—This term is applied to a method of applying remedies in the form of powder to the throat

and respiratory passages, by blowing them through a tube into these parts. See INHALATIONS, Therapeutic Uses of.

INTEGUMENTS, Diseases of.—See SKIN, Diseases of.

INTEMPERANCE, Effects of.—See ALCOHOLISM.

INTERCOSTAL NEURALGIA.—Any of the dorsal nerves may be the seat of neuralgia, not differing materially in its symptoms from neuralgia affecting other mixed nerves, but especially important from a diagnostic point of view. The pains are paroxysmal; usually affect the region of distribution of the anterior division of one or two of the dorsal nerves; and are confined to one side, most frequently the left.

ÆTIOLOGY.—The female sex, neurotic heritage, and weak general health predispose to intercostal neuralgia. As determining causes may be mentioned blows; the action of cold; local injury to the nerves from the growth of thoracic aneurysm or tumour; and disease of the vertebrae. Exhaustion from over-suckling, menorrhagia, or leucorrhœa; irritation from cracked nipples; and pregnancy, are all occasional but important causes of this form of neuralgia. The pain met with in the chest in early cases of phthisis is not infrequently due to intercostal neuralgia. It is probable that, in a large proportion of cases, the neuralgic pain is due to neuritis of one or more intercostal nerves.

SYMPTOMS.—Pain is complained of at some part of one side of the thorax or abdomen, most often in the region innervated by the sixth, seventh, eighth, and ninth intercostal nerves, and much more frequently in the front or side than behind. It is occasionally found in the axilla and inner side of the arm. The pain may be intermittent, occurring in paroxysms, varying in number from a recurrence every few minutes to only two or three such during the twenty-four hours; or there may be persistent pain of a dull character, interrupted at varying intervals by darts of a very sharp kind, which may sometimes be referred with precision to the course of the neighbouring nerve. The pain is described as 'tearing,' or resembling such injuries as a 'stab of a knife,' or 'horing with a red-hot iron.' The acts of coughing and sneezing, as well as any rapid movements of the body, are apt to increase the distress, but the pain is also independent of these disturbances, and will attack without any such provocation. The pain is sometimes more of a wearing than acute character, and the rest will often be destroyed by it. Painful points are sometimes to be discovered in the following situations: 1. Over a spinous process corresponding to the emergence of the affected dorsal nerve from the intervertebral foramen. 2. At the side of the chest or abdomen, where the lateral branch becomes subcutaneous. 3. Near the sternum or at the margin of the rectus abdominalis muscle, at any part down to the pubes, where the termination of the nerve supplies the skin. The skin in the neighbourhood of the tender points is sometimes so hyperæsthetic that the pressure of the clothes is painful. In epileptics and other highly neurotic patients, intercostal neuralgia is often associated with palpitation of the heart, and the pain is usually referred in a vague manner to that organ. Close examination will show that it is in the chest-wall, and tender points may generally

be discovered. The affection is not accompanied by fever. The paroxysms of pain may produce fainting and vomiting. They often cause dyspnoea, with an anxious expression of face, from the inability to draw a full breath without starting the pain.

COMPLICATIONS.—Intercostal neuralgia is sometimes accompanied by herpes zoster. The pain usually precedes the appearance of the eruption, but it is occasionally coincident only, and sometimes comes after it; more often than not it outlasts the eruption, possibly for a long period. In certain cases actual pain lasts but a few days, but is succeeded by an intolerable itching, which is described as being under, not in, the skin. This sensation is said to be felt less in walking than when at rest.

Not infrequently neuralgia of some other nerves, either at a distance, as the fifth, or anatomically near, as the brachial plexus, occurs as a complication of intercostal neuralgia. This is especially likely in cases happening in the period of bodily decay. It is then, too, that the affection may occasionally be accompanied by attacks of angina pectoris.

DIAGNOSIS.—Absence of pyrexia, as shown by the use of the thermometer; the intermittence of pain, and its occurrence irrespective of respiratory movements, although liable to be precipitated by them; and the results of physical examination, serve to distinguish intercostal neuralgia from pleurisy, a condition with which it is very apt to be confounded, on account of resemblance in the stabbing character of the pain. It should be remembered, however, that exceptionally no rise in temperature may accompany the painful stage of pleurisy.

From muscular rheumatism it may be discriminated by the presence of the small and characteristic tender points; by tenderness of the spinous processes on pressure; and by the pain being found not to be dependent upon movements. The same features serve to distinguish it from myalgia, especially that form which often comes from long-continued use in an unaccustomed manner of some muscle attached to the ribs, as when a person unused to carpentering handles the saw energetically for a long time.

Physical examination and the presence of pyrexia will preserve from the error of confounding the dull pain often noted in pneumonia with that of intercostal neuralgia.

Pains of a stabbing, plunging, or electric-shock-like character are often experienced in the intercostal spaces in the course of tabes, and it is important not to confound this disease with a simple attack of intercostal neuralgia. The distinguishing points are the occurrence of similar pains coincidently or alternately in other parts of the body, especially in the lower extremities; the absence of patellar tendon-reflex; and the characteristic gait (if present)—all of which mark tabes. *See* TABES DORSALIS.

PROGNOSIS.—As in the case of other forms of neuralgia, that of the intercostal nerves can hardly be said to be attended by danger, though it must be allowed that in some very rare instances the severity of the pain appears to have actually destroyed life. It is apt, however, to be of troublesome duration, dependent on the cause, lasting for periods of weeks or months, and liable to recurrence.

TREATMENT.—Such should be made in other branches of the same nerve, and in the distribution of neighbouring nerves, for any source of irritation

which it is possible to remove. Constipation should be treated by three grains of Blue Pill, followed by some Friedrichshall or Hunyadi János water, repeated on two or three occasions. Quinine should be given in doses of from five to ten grains twice a day; and if there should be a state of anæmia, iron should be added. Phenazone in doses of from ten to twenty grains may be given two or three times a day, if necessary. Exposure to cold and damp must be avoided; while the surroundings generally should be favourable to improving the nutrition and tone. If the pain be very acute, and sleep prevented, morphine may be injected hypodermically in the neighbourhood of the affected nerve, commencing with a dose of a twelfth of a grain, and increasing this, if necessary, to a quarter of a grain in the twenty-four hours. This dose should not be repeated, however; and it is better to be satisfied with a repetition of the smaller dose, if required. Small blisters (size of half a crown) may be applied to the neighbourhood of the spinal column, near the point of emergence of the affected nerve, one succeeding another as it heals. The continuous voltaic current, from about ten to twenty cells, Leclanché or Stöhrer, may be applied, one sponge on the spine and the other upon the painful points in turn. *See* NEURALGIA; and HERPES ZOSTER.

T. BUZZARD.

INTERLOBULAR (*inter*, between; and *lobulus*, a little lobe).—Situated in the tissue between the lobules of any organ. A good illustration is *interlobular emphysema*, in which air occupies the tissues between the lobules of the lungs.

INTERNAL CAPSULE.—The internal capsule and adjacent ganglia—optic thalamus and corpus striatum—are especially liable to lesion from thrombosis and rupture of their blood-vessels. These are furnished principally by the middle cerebral artery, which in the first part of its course sends off numerous straight twigs, which sink into the anterior perforated space, and supply the corpus striatum and the adjacent part of the optic thalamus. Owing to their position, and direction as regards the main current, they are easily ruptured or blocked up, and owing to their being of the nature of 'end arteries,' and almost destitute of anastomoses with other cerebral arteries, thrombosis rapidly leads to softening of the regions which they nourish.

SYMPTOMS.—The symptoms of lesion of the internal capsule may be divided into three groups or stages.

First stage.—To this stage belong the symptoms usually accompanying an apoplectic seizure (apoplexy), as also the loss or diminution of sensation on the opposite side of the body, which sometimes occurs in consequence of pressure on, or functional interference with, the sensory tracts of the internal capsule. There is also to be observed complete paralysis of voluntary movement on the opposite side of the body, occasionally varied by convulsive spasms of the paralysed side, and conjugate deviation of the head and eyes towards the sound side. This last symptom is due to the centres for the head and eyes of the opposite hemisphere suddenly losing their antagonists. The temperature of the paralysed side is, as a rule, higher than that of the sound side. The total paralysis and flaccidity of the opposite side of the body, and conjugate deviation of the head and eyes, are transient symptoms, lasting from a few hours to a day or two.

Second stage.—This stage includes those symptoms which continue for a variable period, after those depending on the suddenness and disturbing effect of the lesion have passed off. They constitute the common type of hemiplegia or paralysis of voluntary motion on the side opposite the lesion. The face, arm, and leg, and to a certain extent the thoracic and abdominal muscles on one side of the body, are affected. The paralysis does not affect all these parts equally. As a general rule it may be stated that those movements are most affected which are most independent of those of the opposite side, and which are most complex and delicate. Hence the movements of the hand and arm are more affected than those of the face or leg, owing to the fact that these are more commonly exercised in associated or alternating action with those of the other side. The facial paralysis is most marked in the lower facial region. The orbicularis palpebrarum is more or less paretic, but never paralysed to the extent which occurs in Bell's or peripheral facial paralysis, depending on lesion of the seventh nerve. The angle of the mouth on the paralysed side hangs lower, and the tongue deviates slightly to the paralysed side. The weakness of the facial muscles is best brought out when the patient smiles or tries to whistle. The face then becomes drawn to the sound side. While some volitional control may have been acquired over the leg, the hand and arm remain perfectly motionless.

In the process of resolution the leg recovers before the arm, and as a rule the recovery proceeds from the proximal to the distal end of the limb, the movements of the shoulder and hip being regained before those of the hand or foot. The flexors regain their power before the extensors.

The reflexes upon the paralysed side are altered: thus the knee-jerk and other tendon-reflexes are increased, an ankle-clonus is obtained, and the plantar reflex is of the extensor type.

The faradic contractility of the muscles is unimpaired; occasionally it is increased rather than diminished. The muscles do not undergo atrophy except by disuse. The temperature of the paralysed limbs, which at first is usually increased, is generally found to be lower than that of the sound side, to the extent of a degree, more or less.

Recovery may take place from all the symptoms of this stage, within a period varying from weeks to months, or the patient may pass into the third stage.

Third stage.—The special symptoms of this stage are the occurrence of what is termed 'late rigidity' in the paralysed limbs, a condition of evil import. This rigidity shows itself most frequently in the arm, but it is common enough in both limbs. The rigidity affects the flexors more particularly, and causes the limb to assume a position in which flexion predominates. It is variable in degree, and at the commencement is capable of being overcome. At first, also, it is remittent, tending to give way when the patient abstains from volitional efforts or from excitement, and seems almost gone on waking from sleep or when the patient yawns or stretches himself. Gradually it assumes a more intense form, and the limb becomes permanently fixed and rigid.

After death, this condition is found to coincide with descending sclerosis of the motor tracts of the brain and spinal cord. The degeneration proceeds from the seat of lesion downwards through the

crus, pons, and pyramid of the same side, and then across to the posterior part of the lateral column of the spinal cord on the paralysed side. Frequently, also, a similar track of degeneration is found on the inner aspect of the anterior column of the spinal cord, and occasionally also in the lateral column on the same side as the brain-lesion.

Even during the rigid stage there is, as a rule, no trophic degeneration of the muscles or annihilation of faradic contractility, though the muscles waste from disuse unless artificially stimulated. But in some rare instances, as Charcot has shown, the secondary degeneration invades the anterior cornua of the spinal cord, in which case amyotrophy or trophic degeneration of the muscles ensues.

There is no recovery from this condition.

Variations and complications.—Though hemiplegia of the opposite side, without affection of sensation, is the type of disease of the internal capsule, certain variations from the above type are not uncommonly observed.

When sensation is permanently affected along with voluntary motion, we have reason to regard the lesion as implicating the posterior part of the internal capsule and the optic thalamus, an occurrence by no means rare. Co-existing with hemianæsthesia is usually homonymous hemianopsia from interference with the adjacent optic radiations. When, however, the lesion causes rupture of the fibres forming the anterior two-thirds of the posterior limb of the internal capsule, the hemiplegia is most marked and most enduring. It is this lesion only which gives rise to secondary degeneration of the motor tracts and permanent rigidity.

Cases are on record in which lesions of the internal capsule have given rise, not to complete hemiplegia of the opposite side, but to monoplegia, such as paralysis of the face or of one or other limb. These monoplegiæ are due to limited lesions of the internal capsule, which the researches of Franck and Pitres, Beevor and Horsley, and others have demonstrated, contains the motor tracts corresponding to the differentiated centres of the cortex.

The arrangement of the tracts in the internal capsule is briefly as follows. In the fore part of the anterior limb are the corticofugal and corticopetal fibres of the frontal lobe. In order from before backwards there come the fibres for the conjugate movement of the eyes, the mouth and tongue, then the face, the upper limb, and the lower limb. Posteriorly is the combined sensory tract extending as far as the hind end of the lenticular nucleus.

D. FERRIER.

W. A. TURNER.

INTERNAL EAR, Diseases of.—*See* EAR, Diseases of.

INTERSTITIAL (*inter*, between; and *sto*, I stand).—Relating to the interstices of an organ. The term is applied to the tissue which exists between the characteristic elements of any organ, namely, to some form of connective tissue.

INTERTRIGO (*inter*, between; and *tero*, I rub).—**SYNON.**: Erythema intertrigo; Eczema intertrigo.—**DEFINITION.**—A slight erythematous inflammation of the skin occurring in the hollows or folds of the integument, where two surfaces lie in apposition.

ÆTIOLOGY.—The cause of intertrigo is not, as might be implied by its name, *friction alone*; but

rather moisture and heat associated with contact and pressure, acting on a sensitive skin. In certain situations the amount of inflammation is liable to be aggravated by the addition of irritant discharges, such as excessive perspiration, decomposed urine, and fecal matter.

Intertrigo is common in corpulent persons, in those in whom there is free activity of the sudoriparous glands, and in infants from inattention to the napkins. In infants it is seen in the perinæum, extending from the anal fossa behind to the groins in front, and likewise in any other of the deep folds of the integument, as the folds of the neck. Among adults, in addition to these situations it occurs in the axillæ, in the groove beneath the mammae, between the thighs, in the genital regions, scrotal and labio-femoral clefts, the cleft between the nates, and in the flexures of joints; between the glands and the prepuce; at the vulva, and in obese women over the iliac crests. It is well to keep in mind the possibility of inherited syphilis.

The term intertrigo points to a rubbing together or chafing, fretting or galling of the skin by friction, and no doubt friction may have some share in producing the inflammation; but it is also certain that intertrigo results most frequently from irritation caused by the heat and moisture of the part.

DESCRIPTION.—Intertrigo very rarely remains at the erythematous stage, having a natural tendency to run on to exudation, and to be further complicated with excoriations and chaps. In this condition it becomes an eczema (eczema intertrigo), and is very properly treated as such. Many cases beginning as a simple intertrigo drift into a distressing pruriginous eczema, and not infrequently a mild phase of eczema seborrhoicum is the starting-point. Heat, tenderness, and frequently pruritus are the usual sensations.

TREATMENT.—The removal of the cause is the first indication to be attended to in the treatment of intertrigo. This may be effected by keeping the folds of skin apart by thin folds of absorbent borated gauze or silk. The parts should be kept scrupulously clean, dry, and as cool as possible, and dusted with any impalpable unirritating desiccative powder, as fuller's earth, French chalk, kaolin, starch, zinc oleate, zinc oxide, boric acid, or lycopodium. Various combinations of these may be tried, and a little powdered camphor may be added for its antipruritic effect. The 'caking' of starch-containing powders is a great objection to their use. Pruritus must be combated by appropriate measures, and decomposition of secretion guarded against. Where powder is unsuitable, a lotion of lime-water thickened with oxide of zinc will be found useful; or a weak lactate of lead lotion may be tried; or zinc ointment should be kept constantly applied. Where there is much exudation, it is desirable, as in eczema, to avoid abluion and ring the changes with powders, stiff ointments, or zinc-gelatin paste or creamy lotions according to the changing conditions of the affection.

JOHN HAROLD.

INTESTINAL OBSTRUCTION.—**SYNON.** : *Ileus*; *Fr.* *Occlusion Intestinale*; *Ger.* *Darm-verschlussung*.

DEFINITION.—Under this term are included all those cases in which the contents of the intestinal canal are obstructed in their onward passage, by conditions occurring within the abdomen or pelvis. Cases in which obstruction is due to conditions

affecting bowel protruding through one of the external openings are included under the head of **HERNIA**.

The subject of intestinal obstruction will be best treated by first discussing *the condition in general*, and afterwards *its different varieties* in detail.

ÆTIOLOGY AND PATHOLOGY.—The causes of intestinal obstruction may be enumerated as follows, arranged as nearly as possible according to the acuteness and urgency of the symptoms and the imminence of danger to life to which they give rise. The relative frequency with which they occur may be approximately estimated by the numbers appended, representing the results of an analysis of 1,839 fatal cases.

1. *Strangulation by Bands or in Apertures* (440).

(a) By peritoneal bands, the results of previous inflammatory mischief, either under such bands, or by loops or knots, or in buttonhole-like slits, or by kinking caused by traction, or by the margins of slits and rings produced by the adhesion of organs to one another, or to some part of the parietes (219). (b) By the omentum or mesentery forming bands or slits. (c) By diverticula or diverticular cords, due to the persistence of the vitelline duct (Meckel's) or of the omphalo-mesenteric vessels, free or attached (75). (d) By the appendix vermiformis (42). A Fallopiian tube adherent to the neighbouring peritoneum is another cause of strangulation by a normal structure abnormally attached. (e) By the margins of peritoneal pouches (retro-peritoneal hernia, hernia through the foramen of Winslow, and other forms of internal hernia) (39). (f) By bands resulting from inflammation about the necks of old herniæ. Another instance which must be remembered is the band which may be formed by the pedicle of an ovarian cyst.

2. *Volvulus*.—In some cases the intestine is obstructed by being twisted on its mesenteric axis or even on its own axis. In others, the lumen of the sigmoid flexure may be closed by its being twined round a loop of ileum (106).

3. *Intussusception or Invagination* (537).

4. *Impaction of Gall-stones* (51).

5. *Contractions*.—Under this term—the 'contraction' of Fagge, the 'compression and traction' of Bristowe—are included those cases in which the bowel is gradually obstructed by matting together of its coils. This may be due to knitting together of one or more coils by peritonitis, originating, for example, in inflammation of the mesenteric glands. Other conditions are given below.

6. *Stricture*.—(a) Cicatricial, from injury or ulceration; (b) new-growths (373).

7. *Compression*.—By new-growths, hydatids, &c., outside the bowel (66).

8. *Impaction of Foreign Bodies, or Intestinal Concretions* (78).

9. *Impaction of Fæces. Confirmed Constipation* (78).

10. *Congenital Malformations*.

SEATS.—Some parts of the intestinal tract are very much more frequently affected by certain particular causes. Thus, acute obstruction by bands most frequently affects the small intestine; volvulus, the sigmoid flexure. Intussusception most frequently involves the cæcum and colon, and, next, the ileum. Constrictions due to new-growths are most common in the large intestine, especially in its lower portion. Impaction of gall-stones, with very few exceptions, occurs in the lower part of the

ileum. Contractions and traction affect the small intestine more frequently than the large.

SYMPTOMS AND SIGNS.—The symptoms and physical signs of intestinal obstruction are, as a rule, sufficiently constant and characteristic to establish the general diagnosis. But it is usually very difficult, and often impossible, to determine without exploration the differential diagnosis.

The symptoms and signs common to intestinal obstruction will be first given, and then, more briefly, those specially characteristic of each form.

1. *Pain.*—Cases of acute obstruction by a band, volvulus, many intussusceptions, or impaction of a gall-stone, are usually signalled by acute pain, often 'doubling the patient up,' and sometimes producing faintness and collapse. Where there has been a period of incarceration, as in some cases of obstruction by bands or in slits, pain supervenes somewhat more gradually, though later it becomes equally severe. In stricture of the large intestine there is, in the earlier stages, comparatively little pain. Again, in obstruction of the large intestine by impaction of feces, the patient complains merely of fullness, weight, and discomfort.

At the onset the pain is doubtless due to the injury immediately inflicted on the serous and mucous coats, and is very speedily increased and maintained by the effects of the congestion of the blood-vessels on the nerves. This pain will obviously be more severe when the small intestine, with its more intimate association with the nerve-centres and its larger vascular supply, is affected. But it must always be remembered that the severity of the pain depends on the abruptness and completeness of the obstruction, and the extent to which the intestine is involved, rather than upon the variety and cause of the obstruction. Somewhat later comes the pain associated with distension of the bowel itself; this is increased at intervals by paroxysmal exacerbations of acute suffering, which accompany the futile peristaltic efforts of the intestine. Instances of this pain are only too well seen in patients with thin abdomens, in whom attacks of peristalsis, especially when the muscular coat has become hypertrophied, recur after the taking of food, examination of the abdomen, the administration of an enema, &c. Other causes of pain are traction upon inflamed parts, especially when ulceration is beginning to set in; and, at a later stage, enteritis and peritonitis.

With regard to the constant or spasmodic nature of the pain—when the obstruction is complete the pain is constant, with periodic exacerbations; but with an incomplete obstruction it is likely to be intermittent. An important point to consider is how far the pain affords any guide as to the situation of the obstruction. In the great majority of cases of acute intestinal obstruction, wherever may be the seat of the mischief, the pain is referred to the neighbourhood of the umbilicus (*see PAIN IN VISCERAL DISEASE*). Thus, in a large number of cases the site of the pain is of little value as a guide. But there are certainly some cases, especially those of acute obstruction by bands, or sudden strangulation in a slit, in which the pain is referred to a region other than the umbilical. Where the pain is accompanied by the occasional uprising of a coil of intestine anywhere, this spot will very likely be a guide to the seat of the mischief. The readiness with which pain may be masked by preparations of opium, and the dangerously fallacious appearance

of improvement which may follow; the way in which the distinct localised suffering of obstruction may be merged and lost in the more general and diffused pain of peritonitis; and, finally, the cessation of pain which may occur at the close, when the nervous system is dulled by impending death—need only be alluded to here.

2. *Tenderness.*—Both in acute and chronic obstruction, tenderness is usually absent until peritonitis has supervened.

3. *Vomiting.*—This is a very constant symptom of intestinal obstruction. It may commence almost simultaneously with the occurrence of the obstruction, in which case it is due to the shock of the injury inflicted. This vomiting, and its characteristic features—first the contents of the stomach, then bilious fluid, and lastly, liquid more or less feculent—have been differently explained. It is undecided how far the obstruction causes the usual peristalsis to be reversed (anti-peristalsis), or how far, aided by the distension of the bowel above and the glandular secretion poured out, it converts the usual passage of intestinal contents into two currents—one, peripheral, in the natural direction up to the point of obstruction, and the other central or axial, which pours the intestinal contents into the stomach. As a general rule, the higher the seat of obstruction, and the more acute the cause, the more severe is the sickness, and the more frequently does it recur. When the obstruction is high up in the small intestine, the vomit is usually bilious and offensively sour, but without the foetor of feces. When vomit has been retained for any time, as when the patient is under the influence of opiates, it may have had time to decompose and to acquire a feculent odour (Treves). Again, true stercoraceous vomit may come from the lower ileum as well as from the colon, for the contents of the lower ileum may, in the natural state, have all the characters of feces. Moreover, great distension of the intestine may render the ileo-cæcal valve inefficient to prevent regurgitation from the large into the small intestine.

4. *Constipation.*—This may be absolute from the first, even to the passage of flatus, in acute complete intestinal obstruction, as in that due to bands, apertures, volvulus, or gall-stone.

In other cases, where the strangulation is less abrupt and severe, or where the intestine by some peristaltic movements frees itself for a time, fecal evacuations may be passed for a while; and a careful inspection of these, especially as to presence of bile, consistency, feculent odour, &c., may give valuable information as to the degree and seat of the obstruction. In intussusceptions, especially in the more chronic cases, the bowel is rarely altogether impermeable at first, though later it becomes so from the effect of inflammatory swelling. In such cases there is a period in which small quantities of fecal matter mixed with blood and mucus are passed, frequently with much tenesmus; later on blood and mucus only are passed. In strictures of the bowel, constipation comes about comparatively slowly, being here replaced by irregular actions, diarrhoea alternating with constipation, the motions being sometimes altered in shape and size, with passage of blood, &c. Later on, constipation may be rendered absolute by impaction of some undigested article of diet, or by a twist or kink of the bowel above the obstruction.

First among the causes of constipation comes occlusion of the bowel. Another very important

one is reflex nerve-action. Thus, in cases of acute strangulation, the constipation is often absolute from the very commencement, although the obstruction may be in the small intestine, and much fecal matter may still be lodged below the point of occlusion. Again, constipation is very usual in those cases of partial obstruction of the bowel where a segment is suddenly and severely nipped. This is well observed, as a rule, in Littre's hernia, where only a part of the circumference of the bowel is involved in the strangulation. Another cause of constipation which must be remembered is paralysis and exhaustion of the intestinal wall.

The following fallacies must be borne in mind. Even after complete occlusion has occurred there may remain in the bowel, below the seat of obstruction, some portion of its contents; and the secretions of the mucous membrane being added, the evacuation of these by natural effort, aided by enemata, may give rise to the false idea that the occlusion is not complete, or that relief has been obtained. Again, the bubbling away of an enema must not be mistaken for the passage of flatus. Finally, false hopes are sometimes raised by the passage of a stool shortly before death. If this has come from above the obstruction, it may be due to some sloughing which has taken place or relaxation of spasm.

5. *Aspect and condition of the abdomen: Distension, swelling, meteorism.*—The degree of distension and its rate of onset vary with the cause and site of the obstruction, the amount of vomiting, the food taken, &c. Thus, in acute obstruction of the lower part of the ileum, distension comes on rapidly and severely; but an even more rapid and a severer distension is seen to follow on one form of obstruction of the large intestine, namely, volvulus. In obstruction high up in the ileum or jejunum, the epigastric and upper regions appear distended, and the lower ones sunk. But in trying to diagnose the site of the obstruction from the distension the greatest caution must be maintained. 'There is a difference in the shape of the abdomen when distended, according as the arch of the colon is below or above the seat of obstruction. In the former case the belly is rounded, projecting well forwards, but with comparatively little fulness of the lateral and lumbar region. In the latter case it is more broad, and, if the hand be placed on the patient's loins as he lies in bed, a feeling of resistance is experienced which is wanting when the small intestine is alone distended' (Fagge). But the same writer gives us the following cautions in interpreting signs. Absence of fulness in the course of the colon is no absolute proof that the disease is high up in the intestine. Again, the presence of fulness in the course of the colon is no proof that the seat of the obstruction is in the large intestine. For in more than one instance a prominence of the epigastrium and the appearance of a large horizontal coil have been due, not to the transverse colon, but to the ileum, dilated until it rivalled the colon itself. The presence of meteorism is, when rapid and severe, most dangerous and distressing.

Apart from the above distension from accumulation of intestinal contents, certain localised swellings in the abdomen may be occasionally recognised. Thus, intussusception commonly gives rise to an elongated sausage-shaped swelling lying in the course of the large intestine. Stricture of the sigmoid flexure may sometimes be felt in a patient

with a thin, relaxed abdominal wall, as an indistinct localised thickening. Other instances of tumour are very rarely met with. The fat-loaded abdominal wall usually present in a sufferer from impacted gall-stone often prevents this cause of obstruction being felt by palpation. A localised swelling will be of more value as a guide if pain has also been referred definitely to this same particular spot. A number of coils of small intestine, matted together by adhesion, may form a kind of tumour, and localised dullness on percussion has been caused by collapsed coils of small intestine grouped together below an obstruction (Treves).

6. *Peristalsis.*—The fact that the movements of the intestine can be clearly recognised in some cases may be of much help in the diagnosis. One coil may be seen rising up and becoming prominent, and then sinking down and giving place to another, and sometimes waves of action, as it were, seem to pass along a considerable length of bowel. Such movements recur at irregular intervals, and are accompanied by gurgling noises (borborygmi) and sensations, and by exacerbation of suffering. This peristalsis, as Fagge pointed out, is characteristic of the chronic varieties of obstruction, in which the coats of the bowel have undergone hypertrophy. It also indicates an absence of peritonitis. While peristaltic movements are much more often seen in the small than in the large intestine, they may sometimes be observed in the latter. Fagge also pointed out, as worthy of especial note, that the large intestine, when distended, does not continue to lie horizontally across the upper part of the abdomen, but bends downwards, and may form a broad loop, lying vertically and (with the dilated ascending and descending colon) filling the whole front of the abdomen. On the other hand, the coils of the ileum are, under similar circumstances, generally arranged transversely; and, as these coils are often quite as broad as the transverse colon, the uppermost one, lying horizontally just below the ribs, may easily be mistaken for that part of the large intestine.

7. *Diminution of Urine.*—In acute obstruction the urine is diminished in quantity. This fact has been differently explained. Some writers consider that it points to a site of obstruction high up in the jejunum, the area for the absorption of fluid being thus much diminished. Others attribute it to the urgency of the vomiting which occurs in acute obstruction high up. Others again argue that this symptom is merely one of the phenomena of collapse, and will occur in all forms of intestinal obstruction, whatever their seat, in which collapse is present; and as collapse is often present in obstruction of the small intestine, while it seldom occurs in that of the large bowel, suppression or diminution of urine is common in the former, and rare in the latter, condition.

8. *General Aspect and Symptoms.*—In cases of acute obstruction, often from the first, and in the later stages of both acute and chronic obstruction, the general aspect of the patient is more or less characteristic. A condition of collapse frequently occurs; and when this appears early it is due to the shock of the injury, and is usually proportionate to the nerves and the amount of intestine involved, being more marked in the case of the small than of the large intestine; and, above all, to the abruptness and severity of the obstruction. The countenance is expressive of anxiety and distress; the eyes

are sunk, often with dark circles round them, the nose pinched, the cheeks hollow, the lips pale or purplish, and the complexion faintly livid; the general surface is cool or cold (the ears, forehead, nose, and feet first becoming cold), and often covered with a clammy perspiration. Although the mental faculties are as a rule undisturbed, there is a disposition to torpor, from which the sufferer is from time to time aroused by exacerbations of pain, or recurrence of vomiting. The pulse is frequent and small, and towards the end becomes thready. The temperature is rarely above normal unless peritonitis sets in; usually it is normal or subnormal, sometimes falling to a marked extent. Sometimes there is more or less dyspnoea, which may be explained in part by pressure upwards of the diaphragm by the distended bowel below, and the latter cause often gives rise to distressing hiccough. The voice varies, being feeble and whispering, or strong and full quite up to the end. The tongue becomes red, and later dry and brown. Towards the close there is often a combined odour of offensive sweetness and decomposition in the breath. The patient suffers from severe and constant thirst, which often he fears to assuage lest vomiting should be provoked.

In most cases the course is one of progressive emaciation, weakness, and exhaustion, the end being often preceded by peritonitis.

COURSE, COMPLICATIONS, AND TERMINATIONS. In the acute forms of intestinal obstruction the prospect of recovery is extremely slight; perhaps, in the large majority of cases, it would be just to say that there is none at all. And here a word must be said as to those extremely rare instances of spontaneous recovery which have occurred from time to time under every form of acute obstruction, volvulus only excepted. Very much of the value of these cases has been lost by their after-histories not being published, chances thus being lost of our knowing in what cases of really mechanical obstruction spontaneous recovery is really possible. In the second place there is strong reason to believe that in several of these cases the diagnosis has been at fault, the real cause of mischief being peritonitis, e.g. starting from the cæcum or its appendix, or one originating in tubercular mischief, an enteritis, and not mechanical obstruction.

In the more chronic forms of obstruction, with the exception of those depending upon impaction of feces, and some others, perhaps (as chronic intussusception), death, though considerably longer delayed, ensues sooner or later, in spite of temporary relief afforded by operative measures or in other ways.

In cases of acute strangulation, in which the occlusion has been sudden and complete from the first, and in which timely relief has not been obtained, the average duration has been found to be from three to six days. In the cases reported as fatal within a few hours, it is possible that the case has been one of perforation of the intestine or stomach, rather than one of obstruction of the bowels (Fagge). Where the case is one of incarceration at first, followed by actual strangulation, death may not occur until after the lapse of eight or ten days. In cases of constriction or compression in which complete occlusion has come about gradually, or even has not been finally established, the duration of life varies greatly according to the complications which arise, and it may be prolonged for weeks or even months. So, too, in cases of stric-

ture of the large intestine, where the patients are of average intelligence, when they avail themselves regularly of medical supervision, and are amenable to the directions given, especially in the matter of diet, the end may be deferred for one and a half or two years or more.

The *complications and accidents* that are liable to occur are: Peritonitis starting from the seat of the lesion, and more or less rapidly spreading and becoming general; enteritis; ulceration and perforation of the bowel; sloughing of the strangulated or intussuscepted portions; hæmorrhage into the peritoneal cavity or into the bowel; sudden asphyxia or broncho-pneumonia, due to the entrance of vomit into the air-passages; absorption of the products of decomposition or of the breaking down of a growth; gradual asphyxia from the interference with respiration by abdominal distension; and syncope from cardiac depression and cerebral anæmia.

DIAGNOSIS.—Nowhere does successful treatment depend more intimately on correctness of diagnosis; nowhere is a correct diagnosis more invariably difficult; nowhere is it so often impossible. We can only hope to arrive at it by careful attention to the following points, and by striving to keep before us a mental picture of the different hidden possibilities which may lie at the root of the mischief.

The following questions must be considered: I. *Is the obstruction acute or chronic?* II. *Where is the obstruction situated?* III. *What are the best methods of investigation?* IV. *What are the diseases most likely to be mistaken for intestinal obstruction?* V. *Is the case one favourable or unfavourable for operation?* This last question will be considered later under **TREATMENT**.

I. *Is the obstruction acute or chronic?* The symptoms given as distinctive of the two groups are often misleading when applied to individual cases.

(1) Under **acute obstruction** would be included the following: (a) Strangulation by bands or through apertures; (b) volvulus of the colon; (c) acute intussusception; (d) impaction of a gall-stone in the small intestine.

SYMPTOMS.—Pain, whether agonising or colicky, sets in suddenly at a definite time, being generally referred to the neighbourhood of the umbilicus. It is usually paroxysmal at first, but tends to become continuous. It may have followed an unwise meal, going to work, some straining effort, active purgation, or a blow. Vomiting is early and severe—first the contents of the stomach—then bilious, excited by any effort to take food—and ultimately brownish, foul-smelling, or more completely stercoraceous. Constipation is absolute, even to the passage of flatus, though small scybalous masses may come away from a point below the obstruction after enemata or straining efforts of the patient. The abdomen soon becomes distended, but its form presents little that is really distinctive of one form of obstruction rather than another; this being largely due to the facility with which one or two distended coils may be displaced into areas where they conceal the proper contents, and simulate their distension. The urine is diminished or suppressed. The collapse, the state of the tongue, pulse, and temperature are as already stated.

(2) Under **chronic obstruction** would come: (a) Stricture of the large intestine; (b) contractions or adhesions obstructing the intestine (usually small) by bending, dragging on, narrowing, or matting up

one or more coils; (c) stricture of the small intestine after ulceration—traumatic, tubercular, or malignant; (d) chronic intussusception; (e) obstruction by compression of growths from outside; (f) faecal impaction.

SYMPTOMS.—In chronic obstruction these are characterised by their slow irregular development. The pain comes on at first at long intervals, corresponding to troublesome attacks of constipation, and is put down to 'attacks of wind,' colic, or indigestion. Even late in the case fixed pain is often absent; but pain occurs in paroxysms which correspond to attacks of peristalsis, showing the writhing movements of intestine, with muscular coats hypertrophied and as yet not stilled by peritonitis. Constipation is for some time the chief trouble: the bowels never act satisfactorily without aperients. Constipation alternates with diarrhoea; or broken up or narrowed bits, unsatisfactory alike in form and bulk, are passed with difficulty, accompanied by much flatus and some loose motion. After one or more attacks of obstruction more or less complete, the contracted lumen of the bowel may be absolutely blocked. Vomiting is often long deferred, and the patient may keep comparatively well (with clean tongue, sleeping well, and taking food), even when his condition has become one of great danger.

While the above description applies to well-marked acute and chronic obstruction respectively, the two forms do not always fall into such distinct groups. Further, the above terms do not always correspond to obstruction of the small and obstruction of the large intestine. All the symptoms of chronic obstruction, lasting over six months, may be presented by a case of obstruction of the ileum from cancerous puckering of its mesentery; while, on the other hand, every symptom of the acutest obstruction of the small intestine, ending fatally in four days, may be shown by a volvulus of the sigmoid (Fagge). Again, the greatest difficulty in deciding between acute and chronic obstruction may be caused by a patient, whose age suggests malignant stricture of the large intestine, and in whom absolute constipation, a greatly distended belly, and evident peristalsis call loudly for relief, definitely declaring that the constipation is merely a matter of a few days, and denying absolutely any previous attacks of constipation or obstruction. Finally, there is a third class in which an attack of acute obstruction supervenes upon symptoms indicating a chronic obstruction. Such condition most frequently occurs in patients with malignant stricture, or with coils matted together by adhesions, with chronic intussusception, or gradual occlusion of the lumen of the bowel by a growth within or outside its walls, the actual obstruction being brought about by some sudden blocking, as by a faecal mass, or by kinking or bending of the bowel above. The chief help in the diagnosis is given by examination of the patient's history. There will usually be an account of symptoms pointing to chronic obstruction, with previous less severe attacks.

II. Where is the obstruction situated?—It may be said at once that a case of chronic obstruction is usually one of the following: (a) Stricture in the rectum, sigmoid flexure, splenic or hepatic flexure; (b) stricture of the small intestine from mischief within its lumen, or from matting, kinking, or dragging on it by adhesions, &c., outside; (c) chronic intussusception; (d) faecal impaction. Of the above, ob-

struction of some point in the left side of the arch of the large intestine is by far the most frequent.

With regard to acute obstruction, usually in the small intestine, it must be confessed that it is very frequently quite impossible to go beyond localising it in the small intestine. It has been shown that the urgency of the chief symptoms is not related to the seat of the obstruction, but to its severity. No doubt obstruction high up in the small intestine will in many cases be accompanied by earlier vomiting, but less likely to become faeculent. Distension of the abdomen will be less in a case like the above than in one where the obstruction is low down in the ileum. The numerous fallacies which may beset us, depending on the amount of food taken, the influence of drugs, the erratic manner in which one or two coils may cause distension of an area at some distance from their proper habitat, must be borne in mind. It will be wiser to be content in acute obstruction with determining whether it is in the small intestine, as by a band; in the large, as in volvulus; or in both, as an acute intussusception—and to remember that all beyond may be blind guesswork.

III. What are the best methods of investigation?

A correct diagnosis can often only be arrived at by most careful attention to the following points. Trivial as some may seem, all or any may be of the utmost importance; and as cases of acute obstruction may vary every hour, repeated careful examinations may be needful to throw light on a condition which was quite obscure a few hours before.

(a) *The History.*—The value of this is often nil, but the smallest point may sometimes be of the greatest importance. Thus (i) *previous peritonitis* may lead to a suspicion of strangulation by a band, or by multiple adhesions. The history of pelvic mischief, especially in women, or of tubercular inflammation may be of the utmost value. (ii) *Injury*, such as a kick from a horse, may have led to a rupture and subsequent hole in the omentum, thus causing later on a seat of strangulation; or to obstruction from cicatrisation of some bruised loop of intestine. (iii) *An imprudent meal* of heavy and indigestible food may determine the occurrence of obstruction, by the sudden formation of gas and turbulent peristalsis in cases of peritoneal adhesions, or stricture, by causing twisting or kinking. (iv) *Old-standing hernia.* Adhesions formed about the neck of an inguinal hernia may strangle a loop of small intestine quite independent of the hernia.

(b) *The Symptoms.*—These are next to be investigated. The symptoms characteristic of acute and chronic obstruction respectively, and their fallacies, have been already given.

(c) *Physical Examination.*—The most careful and thorough examination of the abdomen by palpation and percussion, and by exploration of the rings, the rectum, and, if need be, the vagina, should be instituted at the earliest practicable period, before general abdominal distension or the supervention of complications has obscured the indications first presented. The form of the abdomen is to be first studied. Then, any fulness which remains localised, especially if accompanied by abiding pain in the same place, any limited dulness, any repeated upspringing of one or more coils at the same place (especially if it is noticed that borborygmi are arrested here), may all be of value in helping to localise the seat of obstruction. In some cases it may be desirable to administer an anæsthetic, to facilitate the examination by relaxing the abdominal

muscles, and to save additional suffering. But this step may be dangerous if vomiting is easily excited. With regard to the rings, the regions of umbilical, inguinal, femoral, and even obturator hernia should be carefully scrutinised. Not only may the modesty of a patient have led to the concealment of an ordinary hernia, but possibly a small knuckle may be tightly strangled in the neighbourhood of the internal ring, or in the canal, or the lumen of a piece of intestine may be partially closed in a tiny femoral hernia. As to the need of a rectal examination, it is only too certain that even nowadays this method of examination is still too much neglected, and thus intussusceptions are sometimes overlooked until it is too late to reduce them by insufflation; and cases of malignant stricture are allowed to go on, and treated as indigestion, flatulence, or constipation. The introduction of the whole hand into the rectum has been rightly given up of late years; the information it gives is very limited, and only obtained at grave risk. Exploration *per vaginam* may afford additional information.

(d) *Use of Enemata and the Long Tube.*—These methods are usually as useless in diagnosis as in treatment. Further, their use is often accompanied by danger, and may be misleading also. Finally, they frequently weaken and distress the already sufficiently handicapped patient. The following dangers and fallacies accompany their use: (i) An obstruction may be pervious to fluid from below and not from above (Fagge). In such a case the fluid pumped through may easily add to the accumulation above this point. (ii) When the abdomen is distended, the administration of large enemata must add to the misery of the patient. (iii) Further, the passage of an enema tube and the amount that can be injected may be misleading also. Thus, it is well known that the tube may be stopped by a fold of mucous membrane; or it may double on itself, thus leading to the belief that no obstruction exists within reach. Again, it is obvious that the sources of fallacy—leakage, admission of air, the varying capacity of the large bowel in different patients, &c.—are numerous. The use of enemata will again be alluded to under TREATMENT, p. 780.

IV. *What are the diseases most likely to be mistaken for intestinal obstruction?*—Of these, the most important in frequency and gravity is peritonitis—especially that form due to mischief in the cæcum or the appendix, or a peritonitis of tubercular origin. Pain, vomiting, constipation, and tenderness may all be present and lead to the greatest difficulty in diagnosis. The points most likely to help are the following: (1) *The previous history.* Thus, though the onset may appear to be sudden, there will very likely have been attacks of 'stomach-ache,' irregular action of the bowels, and tenderness in the right iliac region. (2) *The temperature.* This is usually high in acute peritonitis; but, unless the case is seen early, it may be, as in intestinal obstruction, inclined to subnormal if prostration or collapse have set in. (3) *The vomiting* is usually less urgent in peritonitis, and more rarely feculent. (4) *The constipation* is less absolute. (5) There is usually diffused tenderness and distension in peritonitis. (6) There is no peristalsis. (7) *The abdomen* in peritonitis is often 'smooth, firm, and barrel-like' (Hutchinson). Other conditions occasionally simulating intestinal obstruction are enteritis, e.g. from eating tinned foods, and ptomaine poisoning; acute hæmorrhagic pancreatitis: here

the suddenness of the onset, the rapid collapse, and perhaps the presence of a median deep-seated epigastric swelling, may suggest the cause; thrombosis of the mesenteric artery is another condition which, by the abdominal pain, vomiting, and tympanites, simulates obstruction, but here diarrhoea and blood-tinged stools are not unknown; lead-colic, again, has to be remembered as a condition bringing about an arrest of action but not mechanical obstruction, and to be distinguished by the history and progress of the case.

TREATMENT.—This will be divided into A. Non-Operative, and B. Operative. But while the first will always maintain its place in cases where an operation is refused, where the condition of the patient or his surroundings and those of the medical man do not admit of its performance, it cannot be too strongly laid down that the only hope in acute intestinal obstruction lies in operation, both for clearing up the diagnosis, and for relieving the condition which will otherwise certainly prove fatal. But while the adoption of non-operative treatment in acute intestinal obstruction is almost certainly doomed to failure, it does not therefore follow that operative interference will meet with a large amount of success. Any candid, reflective mind, which has made itself acquainted with the facts of the case, will admit that the same brilliant success which modern surgery has obtained in several regions of the abdomen has not extended to intestinal obstruction, especially the acute variety. The reasons are not far to seek. We have to deal here with conditions often most difficult to find, with conditions that quickly produce irreparable mischief, conditions which early exhaust the patient's strength, and in the exploring and removing of which grave shock is, of necessity, often produced. The diagnosis of acute intestinal obstruction having been made, the practitioner will do well to put both sides before the patient and his friends, the uselessness of palliative treatment, the risks of operative interference, and the fact that these risks are much increased by every hour's delay. If the facts of the case permit of one of those varieties more favourable for operation being diagnosed, such as a Meckel's diverticulum, a single band, or an early intussusception, the practitioner will be justified in strongly urging operative interference. When the case is more obscure, he can only put both sides fairly, and leave the decision to others.

A. NON-OPERATIVE TREATMENT.—I. *Purgatives.*—As a rule almost without exception, in acute or subacute cases, the use of all kinds of purgatives is to be emphatically condemned. They aggravate symptoms; while they are liable to light up quiescent into acute trouble, and convert chronic mischief into acute. Threatening sloughing or perforation may also be rendered inevitable. It is only where all urgent symptoms have subsided, or, in more chronic cases, where there is no evidence of peristalsis or of peritonitis, that mild laxatives, especially some salines, often prove of great value, but great caution is necessary in their administration. These points, together with the use of stronger aperients in obstruction from fecal impaction, will be alluded to later.

2. *Sedatives.*—Opium and belladonna have been given far too much as a matter of routine, as in the usual combination of opium gr. $\frac{1}{2}$ -gr. 1, and green extract of belladonna gr. $\frac{1}{6}$ -gr. $\frac{1}{4}$, every three or

four hours. But it must be remembered (1) that our knowledge of the action of these drugs on the inhibitory and accelerating nerves of the intestine is by no means complete. (2) That we are here especially in the dark. Thus, if we could tell the nature of the obstruction, how the coils lay, and which of them were capable of usefully directed peristaltic action, we might perhaps employ belladonna in large doses. In other cases, where ulceration might be threatening, opium alone would be employed. (3) That while opium undoubtedly allays the pain, checks violent and hurtful peristaltic movements of the intestines, dulls the acuteness of the patient's mind constantly alive to his anxieties and distress, while it promotes sleep and thus husband his strength, it is an entire mistake to speak of opium by the often-quoted phrase 'as our sheet-anchor' in intestinal obstruction. It may dangerously modify symptoms and signs; thus, a patient dosed with opium may be quite unaware that the tenderness of his abdomen is increasing. It may apparently so improve the condition that an operation, later on proved beyond a shadow of a doubt to have been the patient's only hope, is put off till it is too late. In other words, while symptoms are lessened, the condition of the intestine may be hourly getting worse. (4) Adult patients often show remarkable susceptibility to belladonna; and the excitement which may follow an over-use of this drug in these cases may be most harmful to the patient. (5) If opium and belladonna must be given we should be content with the smallest possible doses, especially of the former. In other words, the patient must be judiciously prescribed for, and not drugged. Later on, when operation is declined or set aside, it may be needful to give opium in larger doses, especially if peritonitis be present. (6) There is a certain class of adult cases of intestinal obstruction in which belladonna, in much larger doses than are usually tried, has been most successful. Thus, in more chronic cases, one or two grains of the green extract given every hour, till from five to fourteen grains have been taken, have been followed by relief.

The subcutaneous injection of morphine and atropine in the smallest possible doses is to be preferred to the routine treatment by opium and belladonna. Their administration by suppository is the least satisfactory of all methods in cases of acute obstruction. In a few very acute and painful cases the inhalation of an anæsthetic may be asked for; it must always be remembered that this course, while temporarily relieving suffering, and perhaps enabling the case to be further cleared up, has certain inherent dangers of its own.

3. *Diet*.—Sufficient care is still but seldom taken in this simple matter, and the patient is allowed to add needlessly to his misery and danger. In acute obstruction, whenever the diagnosis is suspected or established, it is much best to cease feeding altogether by the mouth, and to trust to nutrient enemata. The small intestine and the stomach itself are liable to be gradually filled up by an ascending column of fluid, and vomiting is thus constantly impending; and when it has already set in, it is only made worse by giving food by the mouth. As a rule, nothing whatever should be given by the mouth save a little ice in small pieces. Nutrient enemata may be made on some such plan as—milk $\mathfrak{z}\mathfrak{j}$, brandy \mathfrak{zss} ., yolk of one egg, with liquor pancreaticus (B.P.) $\mathfrak{z}\mathfrak{j}$, or a few drops of

diluted hydrochloric acid and a little pepsin—the whole being gently warmed, but not cooked. Simple enemata of strong beef-tea, only just liquid, may be given every two, three, or four hours. After, or in place of either, a nutrient zymised suppository may be inserted. Before an enema is given, it must be ascertained that the rectum is empty. Thirst is best met by the guarded use of ice, the frequent moistening of the patient's lips, or the injection of sterilised water into the connective tissue of the axillæ. Large liquid enemata sometimes relieve thirst markedly, but the objections to these have already been given. When it is clear from the failing pulse, the falling temperature, the cold extremities, the dry tongue, and most fetid breath that trusting to nutrient enemata is not sufficient, limited quantities of champagne, brandy and soda-water, or some kind of meat-juice must be given by the mouth.

4. *Enemata*.—In intussusception and fæcal impaction, and in cases of stricture with a fæcal block above, large enemata may be valuable; but with these exceptions, and especially in all acute cases of obstruction, they are very rarely useful.

5. *Local applications*.—Material relief is often given by the assiduous use of quite hot flannel, wrung out of a 2 per cent. creolin-solution. This, which soothes with its heat and helps to sterilise the skin before operation, should be regularly renewed. Poultices are too heavy, and quickly get cold. Turpentine is sometimes applied on hot flannels. It has the objections of frequently slightly blistering the skin, thus rendering it tender for examination, and perhaps interfering later on with the incision of an operation. In a few cases the patient experiences greater relief from local cold, as by cracked ice applied at frequent intervals between two layers of flannel tacked together with a few stitches.

6. *Electricity*.—This is occasionally of service in fæcal impaction.

7. *Washing out the stomach*.—This step, recommended by Kussmaul, gives much relief in some cases where the stomach is distended with fluid. Where, however, fluids have been properly restricted from the first, it will not be required.

B. OPERATIVE TREATMENT.

Surgical Operations.—These include—(1) *Abdominal section*; (2) *Formation of an artificial anus*; (3) *Puncture*. Colotomy will be referred to under the treatment of chronic obstruction.

1. *Abdominal Section*.—A few most important points, many of them fallacies still unrecognised, first call for attention: (1) Acute and chronic obstructions do not always fall into distinct groups. (2) Chronic obstruction often puts on an acute appearance. (3) The demand frequently put forward at the present day that cases of acute intestinal obstruction require surgical treatment is a fair one, but for the reasons given above it is a mistake to expect a very large proportion of successes, even after early operations for intestinal obstruction. (4) Many leading surgeons have held strongly that the chief cause of the great mortality after operations for intestinal obstruction has been in their having been put off too long. While this is no doubt a chief cause, it is not the only one. It is a mistake to group all cases of acute intestinal obstruction together as all equally suitable for operation. *Post-mortem* evidence shows that a distinct proportion are from the first hopeless of relief by opera-

tion, others becoming hopeless so early in the case as to render any operation probably futile. Thus, with regard to operative treatment, cases of acute intestinal obstruction may be divided into these three groups: (a) *Cases probably hopeless from the first*—e.g. some cases of severe volvulus of sigmoid and colon; some cases of acute intussusception in infants; and cases of complicated snaring or matting, as in peritonitis started about the female pelvic organs, or in inflammation around mesenteric glands. (b) *Cases in which there is hope of doing good by operation*—e.g. cases of band, especially the single band in young subjects; some cases of intussusception, and of internal hernia. The question of benefit from operation in acute obstruction is intimately bound up with these two points: (1) the impossibility of making an accurate diagnosis of the nature or the site of the obstruction in many cases; and (2) the peculiar structure of the parts; thus, the intimate association of the intestine with the nervous system, the weakness and easy paralysis of its propelling power, and the readiness with which it is strangled, &c. Finally, among the conditions which handicap the explorer are (a) the tightness with which his hand is gripped by the abdominal wall here, not stretched as by a large ovarian tumour; (b) the way in which distended coils of intestine crowd up and get in the way; (c) the risk of fatally disturbing parts already known to be damaged; (d) the condition of the patient. A skilled surgeon should be called in as early as possible, as it is only fair that he should see the case early and throughout. When an operation is decided upon, the surgeon will be in a far better position than when called in, as too often happens, late in the case. When an operation is to be performed, the pulse should be fair, the temperature not falling, the abdomen not much distended, and with sufficient peristalsis to make it probable that there is little or no peritonitis. On the other hand, the operation should not be performed, as it too often is, on the merest chance of relief, when the abdomen is enormously tympanic, when the temperature and pulse are falling, and the patient passing into irrecoverable collapse.

The Operation.—Only the chief points can be noted here. The bladder being emptied, and the abdominal wall sterilised, the patient, with his extremities warmly clad, is placed on a water-bed filled with hot water. Everything that can possibly come within the field of operation must be scrupulously cleansed before the operation, and kept aseptic. An anæsthetic having been given, the abdomen is quickly opened along the linea alba below the umbilicus; and all hæmorrhage being arrested, two or three fingers are introduced, and the following possible sites of strangulation first looked to: (1) the cæcum, its condition of distension or emptiness telling whether the obstruction is below or above it. (2) The inner aspects of the femoral, inguinal, and obturator apertures, to make sure that no tiny hernia exists, imperceptible from the outside. (3) The brim of the pelvis, as bands of omentum are often fixed hereabouts, and also because, in women, local peritonitis, originating in the uterus or its appendages, is not infrequently the source of the obstruction. If the above search with two or three fingers or the whole hand fails, and it often will when distension is present, embarrassing the fingers in their movements and obscuring the relation of parts, one or two loops

which lie nearest the wound should be scrutinised and followed in the direction of increasing congestion and distension, thus leading to the obstruction. If this prove fruitless, the most distended portion of the intestine must be drawn out (under cover of sterilised towels) bit by bit from the upper part of the wound and passed in again, after examination, into the lower angle, in such a way that at no time are more than five or six inches of intestine exposed. After drawing out and replacing some feet of intestine in this way, it is probable that, owing to the increasing congestion or resistance, the surgeon will reach the obstruction. An assistant should hold the coil from which the surgeon starts under a hot sponge, to prevent the same ground being traversed a second time. If after five to ten minutes' search the mischief is not found, the surgeon must decide between making an artificial anus and closing the wound, and enlarging the latter and allowing all the coils to prolapse under hot sterilised towels, frequently renewed. The decision must turn on the condition of the patient and the surroundings of the surgeon. On the one hand it is only taking this bold step which will, in a large number of cases, bring to light the cause of the obstruction. On the other, if the coils be distended, it will be impossible to get them back without draining them and closing the opening. The needful manipulations necessarily produce much shock, and perhaps inflict serious damage. When the obstruction is found, it must be dealt with according to its nature. Thus an opening (as in the mesentery) must be stretched with the finger-nail and the bowel withdrawn; omental bands, if not torn through, divided between two chromic-gut ligatures; any diverticular band brought up into view, and divided with similar precautions, or, if the patient's condition admits of it, resected and the cut ends cleansed and closed. Resection of gangrenous intestine, and intestinal anastomosis, may have to be resorted to. The best modes of dealing with volvulus, intussusception, and gall-stone, are given under their respective headings. It remains to allude very briefly to two most important steps, the omission of which, owing to the hurry at the close of an operation for intestinal obstruction, often renders what has been done futile. The first is that it is absolutely useless to leave the abdomen full of distended intestine. This will inevitably prove fatal, partly by intestinal toxæmia, partly by pressure on the diaphragm, and cardiac collapse. Its removal is imperatively needed, either by making an opening in the intestine, draining this and then closing the opening, or by the formation of an artificial anus. The other step is the thorough cleansing of the peritoneal sac when this has been contaminated, either by sponges or by flushing with a hot solution of boiled water, introduced using an irrigating tube. In these cases a Keith's tube should be introduced into the pelvis and sucked out regularly.

2. Formation of an Artificial Anus; Enterotomy.

This is done under the following conditions: When the patient's condition does not admit of any searching for the cause of the trouble, when the operator cannot detect the site of obstruction, or finds that he can neither deal with this nor perform resection, or intestinal anastomosis. The artificial anus is best made by tying in a Paul's tube by means of a purse-string suture. The coil should first be brought well outside the abdomen, and emptied with gentle pressure before the suture is inserted. Another

means, when a Paul's tube is not forthcoming, is to bring out a coil, fix it by sutures, and then open it by a trocar and cannula passed through sterilised thin indiarubber sheeting, so that liquid fæces do not flood the wound, &c. Nélaton's operation is usually adopted. Making an artificial anus is merely palliative; and should only be resorted to when other steps fail or are impracticable, and when it is certain that the obstruction is low down in the small intestine. The chief drawback to this method lies in the extreme annoyance the patient suffers afterwards from the continual escape of liquid fæces, and the certainty of marasmus if the opening in the intestine be high up.

3. *Puncture of a Distended Coil by Trocar and Cannula.*—This step should be abandoned. While in a very few cases puncture has apparently proved efficient, there is no doubt whatever that the number of cases in which this method has been fruitlessly employed, and which have never been published, is infinitely greater. The chief risks are: (a) Not hitting the right coil; thus one (though distended) may be hit so far from the obstruction as to give no real relief. In not a few the bowel below the obstruction may be punctured (Treves). (b) The puncture may give rise to no real relief, but to increased distress. (c) The puncture may be followed by fatal leakage into the peritoneal sac.

VARIETIES.—1. *Strangulation by Bands or in Apertures.*—As some at least of these depend upon the results of previous inflammation, it is important to inquire minutely into the early history.

ANATOMICAL CHARACTERS.—The bands causing strangulation may be adhesions resulting from peritonitis (omental), a persistent Meckel's diverticulum, or some normal structure abnormally attached. In any case it is the small intestine, and usually the end of the ileum, which is specially liable to become thus strangulated. A band or bands, sometimes rounded, sometimes flattened, may stretch from one part of the mesentery or omentum to another, or, oftener, may be attached by one end to the omentum or mesentery, and by the other to the bowel (only exceptionally to the large intestine), or to the abdominal wall or pelvis. Much more rarely are they found passing from one part of the bowel to another. The broader adhesions sometimes present slits in which portions of bowel may be caught. Similarly, ruptures or slits may occur in the omentum or mesentery, or even in very rare instances in the suspensory ligament of the liver, or broad ligament of the uterus. The lower attachments of the omental bands are usually to the parietes or viscera, to the brim of the pelvis, or to near the inner orifice of one of the rings. Of the diverticular bands, the most important is the one called 'true,' which is due to a persistent or partially obliterated vitelline duct. This band is single, and is attached at one end to the ileum from one to three feet from its termination; its other end may be free or attached in the vicinity of the umbilicus. Its structure may be like the intestine, or cord-like. The intestine may be strangled beneath it, when the band is adherent at both extremities, or snared and knotted up by it, in a much more complicated way, when its distal end is free. Other congenital bands may arise from the omphalo-mesenteric vessels. Strangulation may, much more rarely, take place in various forms of internal hernia (hernia into the foramen of Winslow, or into peritoneal pouches, mesocolic, duodeno-jejunal, or retro-peritoneal).

SYMPTOMS.—These are similar to those already described under Acute Obstruction. Strangulation of the small intestine by the above-mentioned bands or apertures is most commonly met with in early adult life, the average age being from twenty to forty. One sex does not appear more liable than the other, except in the case of strangulation by diverticula, which has been observed about twice as often in the male as in the female subject. The more youthful the patient, the more will a true diverticulum suggest itself. But strangulation is not unknown in much older patients, as shown by the subjects of herniæ and omental bands.

TREATMENT.—The surgeon may resort to abdominal section with much hope. As to the date of interference, the earlier the better, before the patient's strength is lowered by vomiting, and the operation rendered additionally difficult by distension.

II. *Volvulus.*—The pathology of this has been already given (p. 774).

SYMPTOMS.—In addition to the usual symptoms of Acute Obstruction, there are certain special points which may aid in the diagnosis. The patients are usually adults; and constipation has most likely been of long standing and habitual. The occurrence in insane patients, though the insanity probably only points to neglected bowels, has been sufficiently frequent to be noteworthy. The symptoms are urgent (with the exception, perhaps, of vomiting, which may be less marked than in other forms of acute obstruction), and may end rapidly in death. But the point which is likely to be distinctive of volvulus is the urgent distension of the abdomen, often due to one or more huge coils of large intestine.

TREATMENT.—Operative steps, taken early, give the only hope here; otherwise the huge distension, and the early stage at which inflammatory changes set in, rapidly bring about a fatal result. The distended sigmoid should be exposed and its contents evacuated by a trocar or aspirator passed through sterilised thin indiarubber sheeting so that liquid fæces do not contaminate the wound. If needful, the bowel must be incised and emptied, every care being taken not to infect the peritoneum. As soon as the distension is materially lessened, the twisted loop should be brought outside, the twist undone, the opening closed and the bowel replaced. If tympanites persist, the opening should be made into an artificial anus, or a right-sided colotomy performed, to relieve the cæcum. This has been done as a primary operation with complete success in a few cases. But owing to the rapidity with which gangrene attacks a volvulus, the treatment first given is much to be preferred.

III. *Intussusception or Invagination.*—This form of obstruction is most important, owing to its frequency in early life, its rapid fatality in many cases, and the number of brilliant successes which have followed insufflation and abdominal section.

ÆTIOLOGY.—Intussusception is the most common variety of obstruction to which children are liable, about one-fourth of the cases on record having occurred during the first year, and more than one half during the first seven years of life. In early life and childhood it is more frequent in males. Intussusception would appear to be brought about by some irregular peristaltic action of one part of the intestine, perhaps conjoined with inaction of another part. Sometimes it follows severe purging,

diarrhoea, or violent straining, sometimes the presence of worms, or a polypoid growth, especially in the lower ileum. In a few cases it has followed movements or strain, especially during the dandling and dancing of an infant upwards and downwards in the arms.

ANATOMICAL CHARACTERS.—In ordinary cases a transverse section will show three rings of bowel, and a longitudinal one three layers of bowel on each side. The external of these three layers is called the *intussusciens* or sheath, the middle the returning layer, and the innermost the entering layer, these last two, taken together, forming the *intussusceptum*. Of these layers, the outer and middle have mucous surfaces in mutual contact, and the middle and inner have serous coats thus opposed. The increase of the intussusception takes place by the entering and middle layers moving in together, and dragging in the outer one after them. Thus, while the inner bend between the entering and the middle layers remains the foremost part of the intussusception, the outer bend between the middle and outer layers is constantly changing. Between the middle and inner layers the mesentery or mesocolon or both, as the case may be, are drawn in. This fact determines, from the first, peculiarities in the shape of the intussusception; and, later on, the compression of the vessels brings about congestion, ecchymosis, swelling, and sloughing. Thus the traction on the mesentery, often considerable as the invagination increases, causes a curving of the cylinder of the intussusceptum, and to a less degree that of the sheath, towards the root of the mesentery. It also may pull the axis of the intussusceptum and its terminal orifice out of that of its sheath, and nearer the mesenteric border of the intestine, the orifice of the end of the intussusceptum becoming also, by the traction, slit-like. Intussusceptions occur much more frequently in some parts than in others. Thus, when the small intestine, especially the ileum and lower jejunum, is involved, it is spoken of as the *enteric* variety (30 per cent.). In the ileo-cæcal region two forms are found: (*a*) the *ileo-cæcal proper*, in which the ileum and cæcum, preceded by the valve which forms the most advanced point of the intussusceptum, pass into the colon. This variety forms 44 per cent.; it may attain great size, the valve reaching as far as the anus, and being then liable to be mistaken by careless observers for a prolapsus recti. (*b*) In the other, *ileo-colic* (8 per cent.), the lower part of the ileum is prolapsed through the ileo-cæcal valve. The *colic* variety (18 per cent.) may occur anywhere in the large intestine, but is most frequent in the descending colon and sigmoid flexure.

SYMPTOMS.—In *acute* intussusception the onset is sudden; pain is an early symptom, and of a colicky, gripping character, but not as a rule so agonising as that which may be produced by a band. Like tenderness, it may here be a guide to the site of the intussusception. Vomiting is not so urgent as in strangulation by bands or other conditions. And the same is true of constipation: this is rarely absolute; usually it is replaced by a diarrhoea, at first fecal, then fecal mingled with blood-stained mucus, the admixture of fecal matter becoming less or ultimately absent altogether. Severe tenesmus and straining recur at frequent intervals: and these, with the absence of sleep, and the inability to take food, soon exhaust the strength of the little patients. On examination of the abdomen, the intussusception-

mass can be distinctly felt as a firm, cylindrical, sausage-like swelling, in all cases of ileo-cæcal intussusception. In these cases it may traverse the right flank, the upper part of the abdomen, the left flank, and even be felt *per anum*, or seen projecting. With the above symptoms are present, to a varying degree, those of shock and peritonitis.

Under the name of *chronic intussusception* are included those cases of intussusception which have a duration of one or more months. These are rare, but their extreme importance is shown in the words of Treves—that no form of intestinal obstruction offers so many difficulties in the way of its recognition, and no form has been the subject of more error in diagnosis. This form appears most frequently in adult males. Its course is most irregular. The onset is sudden only in about one-third of the cases—a point, when present, of much value in the diagnosis of this from other forms of chronic obstruction. Pain, seldom severe, may be entirely absent for long intervals, these becoming shorter as the disease advances. Vomiting is only marked in about half the cases; it is feculent only in about 7 per cent. The action of the bowels varies greatly. As a rule they are irregular, more often with a tendency to diarrhoea than towards constipation. In about 50 per cent. a bloody discharge from the anus occurs. Distension of the abdomen is slight, and may be absent save during attacks of temporary obstruction. A tumour is to be felt in about half the cases. The general condition is one of wasting and anæmia, the patient not infrequently dying of marasmus.

COURSE AND PROGRESS.—(1) *Acute*. Unless relief is obtained, the case will end fatally from shock and exhaustion within three to five days in young children, or in infants in a much shorter time, or within a week or ten days in older children; in adults between the second and third week. These cases lead up to the second class of *subacute* cases, in which life may be prolonged for three or four weeks. (2) The case may be more or less *chronic*, and terminate in death only after a period of several weeks or months, from wasting and exhaustion or peritonitis and enteritis, with or without perforation. Lastly, recovery may take place after sloughing, separation, and evacuation of the invaginated bowel. This last event, of great interest, but by no means necessarily curative, is extremely rare in children under two. It has occurred proportionately in 20 per cent. of ileo-cæcal, in 28 per cent. of colic, and in 6 per cent. of enteric cases. Its most frequent period of occurrence is from the eleventh day to the period between the third and fourth week (Leichtenstein). Briefly stated, it is brought about by adhesions forming between the invaginated and receiving portions of the bowel, and death of the intussusceptum from strangulation of its blood-supply. In most of the recorded cases it has been some portion of the small intestine that has come away; in some the cæcum and its appendix with portions of the colon. Sometimes the sloughed intestine is evacuated in shreds, at other times in its entirety. While this result is usually met with in acute cases, it may occur in chronic ones that end acutely. The signs are great fetor of the stools, and gangrenous shreds of intestine found in them. It has been already said that this process can by no means be relied on to save life. The giving way of the adhesions, and fecal extravasation, chronic diarrhoea, hemorrhage,

pyæmia, and, later on, a stricture, may prove fatal.

TREATMENT.—Acute intussusception, especially in the child or infant, demands prompt and active measures. Opium and belladonna should be given at once and sufficiently to check undue peristaltic action and relieve pain. The abdomen should be kept covered with hot fomentations; and the amount of nourishment given by the mouth should be most strictly limited. In young children especially, owing to the rapidity with which their strength runs down, and in all cases, so as to ensure a condition which still admits of replacement, reduction should be attempted as early as possible. Inflation should be preferred to injection as somewhat less risky. The child being placed fully under an anæsthetic, and its pelvis and lower limbs somewhat raised, the nozzle of a Lund's inflator, a rectal enema-tube, or a full-sized catheter well smeared with vaseline, attached by tubing to a bellows, is carefully passed into the bowel. The nates being securely pressed around the tube, air is steadily pumped into the colon, while the surgeon keeps one hand on the abdomen, not only to prevent over-distension, but also to watch for any receding of the tumour towards the cæcal region. With regard to the amount of force used, replacement of the bowel can usually be effected only by considerable distension of the whole colon, and this requires a good deal of rather forcible pumping to complete it. This is especially the case with regard to the last few amounts of air sent in (Goodhart). Inflation failing, if the condition of the patient admit of it, more powerful means may be made use of by connecting the rectal tube with an irrigator placed not higher than three feet above the bed, a more equable and forcible distension being thus obtained (Goodhart). Goodhart points out that the last method entails a greater risk of rupture of the bowel, but that the end justifies the means, considering the great danger of these cases. If the above methods fail, abdominal section should at once be resorted to in suitable cases. The following points are here important: (a) *The age.* In infants under a year, unless reduction is early tried and quickly successful, the prognosis is very grave. Even at this early age there is an increasing tendency on the part of hospital surgeons to resort to operation early. It is felt that too much importance has been attached to the cases published in which inflation has met with success; that these cases in reality bear a very small proportion to those which have been treated in the same way unsuccessfully. Again, it is felt, and rightly, that valuable time is often wasted in repeated attempts at inflation often unsuccessful in reducing the intussusception completely. Finally, the increasing amount of success which attends operative interference shows that, though the vitality of these little patients is small, intussusception is favourable for operation, in that it is one of the very few forms of acute intestinal obstruction in which the condition can be accurately diagnosed, its site definitely located, and the amount of manipulation and interference is therefore brief and slight when the surgeon is asked to operate early. (b) *The condition of the patient as to collapse, &c.* (c) *The duration of the case.* In the majority, especially in children, the tendency of the condition is to strangulation and not simply incarceration, and while the rapidity of the strangulation varies a good deal, the chances of inflation or injection are small, unless in

recent cases. (d) It is thus of the utmost importance to decide *whether the bowel is strangulated or incarcerated.* Hutchinson points out that the severity of the symptoms will be helpful here, namely, the urgency of the vomiting, the degree of the constipation, the character of any stools passed, and, above all, as utterly incompatible with gangrene, advance of the tumour onwards.

Only the chief points in the operation can be given here. The peritoneal cavity being opened by a median incision with the umbilicus for its centre, the intussusception is found. For a few minutes careful pressure should be made on this to diminish the oedema and inflammatory swelling, before any attempts at reduction are made (Senn). The wound being sufficiently enlarged so as to admit of two fingers of each hand, and the small intestine packed away, if the mass cannot be hooked up into the wound it must be reduced *in situ*. This can be done, if there are no adhesions, either by traction upwards on the upper part of the invagination; or, as thought better by Hutchinson, by finding the lower part, holding the ensheathing part so that it cannot be drawn into constricting folds, and pressing out the contained bowel by gentle squeezing movements between the finger and thumb. These movements must be continued, and the finger and thumb gradually shifted upwards along the gut till every atom of the mass is reduced; this being often made known by the appearance of the vermiform appendix. Every care must be adopted to finish the operation as speedily as possible, and every precaution taken against shock. The chief points in the after-treatment are the application of warmth, and the administration of milk, brandy, and laudanum. If reduction be found impracticable enterectomy is permissible.

IV. Impaction of Gall-stones.—Gall-stones large enough to block the gut may enter the intestine after a process of inflammatory adhesion and ulceration between the gall-bladder and the duodenum or colon. In such cases, though there is usually a previous history of more or less suffering in the hypochondriac region (which may assist in the diagnosis), those paroxysms of pain and the jaundice which accompany the passage of gall-stones down the duct may not have been experienced. Obstruction from this cause is comparatively rare. It is met with far more frequently in the female than in the male, and with very few exceptions after late middle life. The patients are very often obese. As a rule the obstructing gall-stone is single. The most common seat of impaction is the lower ileum, and next to this the jejunum.

SYMPTOMS.—These are usually sudden and acute; death from enteritis conjoined with those of acute obstruction occurring, as a rule, in about five days, perhaps earlier, as the result of shock. Recovery rarely takes place.

DIAGNOSIS.—This is aided by the sex, age, and previous history of the patient; the acuteness of the symptoms; and perhaps by the recognition of a more or less distinct hard lump corresponding to the obstructing gall-stone.

TREATMENT.—If a very short trial of palliative treatment is futile, abdominal section is here the patient's only chance; though, owing to the usual age, habits, and condition of the tissues, the step must always be a very anxious one. The stone having been found, it may be dealt with—(1) By trying to pass it on into the large bowel. (2) By

intra-intestinal crushing, either between the fingers or by flat-bladed forceps guarded with indiarubber tube. (3) This may be facilitated by Tait's suggestion of puncturing the stone with a needle, passed obliquely. (4) The loop being drawn outside, the stone may be extracted and the opening most carefully closed. If the bowel immediately around the calculus be inflamed, the stone should be pushed into a healthy portion before extraction.

V. Contractions.—In this class may be included cases due to adhesions, kinking or compressing the gut, or matting together several coils; and those where mischief, inflammatory or malignant, has closed the lumen by traction, or narrowed it—as when the mesentery puckers from old tubercular or malignant mischief. These cases are among the gravest and the most hopeless of intestinal obstructions; they affect the small more frequently than the large intestine; and are often preceded by old peritonitis.

SYMPTOMS.—Owing to the rarity of stricture of the small intestine, this group practically comprises all cases of chronic obstruction of that part of the bowel (Fagge). The attacks are often repeated, and the final one is in reality acute supervening on previous chronic mischief. Vomiting and constipation will for a long time be irregular and uncertain. Visible peristalsis, and colicky attacks of pain with loud rumblings, will probably be marked features.

TREATMENT.—For some time this must be palliative with a view of preventing obstruction. Abdominal section is here especially likely to be disappointing, owing to the complicated nature of the obstructing agency, the fact that there may be more than one seat of obstruction, and the intimacy with which one or more coils of intestine are matted to each other or to adjacent viscera. Formation of an artificial anus (*vide supra*), and still more colotomy where the large intestine is affected, may give relief. When the opening is made in the small intestine, it must be low down, or miasmus will follow.

VI. Obstruction from Stricture.—(a) *Simple cicatricial stenoses.* These may result from the effects of dysenteric, tubercular, or syphilitic ulceration of considerable extent. The dysenteric are most frequent in the lower part of the large intestine, the tubercular in the lower ileum and about the ileo-cæcal valve, and the syphilitic in the rectum. Rarer instances of stenosis occur after injury, the sloughing off of an intussusception, the strangulation of hernia, or the ulceration produced by temporarily impacted gall-stones, fecal masses, or foreign bodies.

(b) *New-growths.*—A more exact pathology has practically narrowed down these to one kind, namely, cylindrical epithelioma. Very rarely innocent growths—e.g. fibromata or adenomata—may cause more or less obstruction, usually by becoming polypoid. The locality attacked is in the very great majority of cases on the left side. Cases occur in about equal proportions in the rectum and sigmoid flexure: then about half as frequently in the left colon and splenic flexure; the remainder being met with in the ascending colon and hepatic flexure.

SYMPTOMS.—In the earlier stages these are often by no means marked. Disordered, irregular, and unsatisfactory action of the bowels, and general discomfort, with intervals of comparative ease, constitute the earliest indications. At this time, owing to the forgetting or deferring of an examination, the cases are far too frequently treated as instances of flatulence, indigestion, and constipation. Later on,

the symptoms of obstruction become more pronounced. In most cases abdominal distension, accompanied by fetid eructations and pain, comes on, varying in degree from time to time. The pain is usually distinguished by its paroxysmal character, and its relation to unwise feeding. Nausea is an early symptom; but vomiting usually appears late, and usually only becomes feculent in the final attack of obstruction. The motions are always unsatisfactory, the bowels not acting without aperients, loose stools often alternating with scybala, or broken-up bits with slime and mucus coming away. Troublesome, teasing tenesmus, causing the patient repeatedly to seek relief from the sensations of an imperfectly emptied bowel, is often present when the stricture is low down. Peristaltic movements of the intestines, accompanied by colicky pain and rumbling borborygmi, become increasingly frequent and manifest as the muscular coat hypertrophies. As the case progresses, the general condition of the patient, the increasing emaciation and cachexia, and the failure of treatment, all point to the malignant nature of the case.

COURSE AND TERMINATIONS.—The course of all such cases, though very variable in duration, is progressively unfavourable, and sooner or later death supervenes, usually from peritonitis, with or without perforation, or from exhaustion by prolonged suffering and possibly suppuration. Absolute occlusion, as a rule, comes about slowly. In some cases it is never completely established; in others, it occurs suddenly, from impaction of hardened feces or undigested food. It is worthy of note that, even in cases in which the seat of constriction is in the sigmoid flexure or rectum, the greatest evidence of stress due to the fecal accumulation, distension, ulceration, and perforation, is often found in the cæcum.

TREATMENT.—From the very first, as soon as there is reason to suspect the presence of a stricture, the most careful attention to diet, and the regular administration of such medicines as will favour soft semi-solid motions, should be insisted on. In other words, if the misery of death by obstruction is to be warded off the patients must pay the strictest attention to their in-take and their out-put. If these points are attended to, the patients may be kept, for the year or two which the disease takes to run its course, in comparative ease; but it is extraordinary how frequently and at what cost these most obvious precautions are still neglected. Patients, the subjects of stricture, should be warned that they are living on the brink of a precipice. As a rule, carelessness in diet brings its own penalty, and most patients willingly acknowledge the relief which such a dietary as the following gives:—soups; milk, bread and milk, milk and oatmeal, arrowroot; poached or whipped-up eggs, a few oysters, a little vegetable (especially those which leave a small residue), breadcrumbs, yolks of eggs, with plenty of gravy, or well puréed with milk; a little underdone meat, well pounded or shredded. In the early stages, and later on in the intervals of relief from threatening obstruction, aperients should be given regularly. There is nothing better than an ounce of castor-oil twice a week, and some laxative daily, such as cascara sagrada tablets, compound liquorice powder, a pill containing aloes, compound extract of colocynth and oil of juniper—one of these may be administered overnight, or Hunyadi János, or a similar water, is given in the morning. Where

obstruction is actually threatening, a laxative such as castor oil or sulphate of magnesium may, if retained, still give great relief if combined with a little tincture of opium, and aided by enemata of castor oil. But where obstruction is actually present, the case must be treated on the usual lines, a sedative here replacing laxative treatment.

When the above treatment has failed, or when the case is seen too late and obstruction is present, lumbar colotomy (on the right or left side, according to the indications afforded) is, on the whole, the safest operation, and should not be too long deferred. Where there is not much distension, and the operator has skilled assistance, it will be better to open the abdomen in the middle line, locate the growth, bring this outside, and fix it over a glass rod or a bougie, and open the bowel by tying in a Paul's tube (*vide supra*). Later on the growth may in favourable cases be removed.

VII. Obstruction from Compression from without.—Various viscera, enlarged and displaced, especially the uterus and ovaries, and in rarer instances the spleen, the kidney, or even a distended bladder; tuberculous or cancerous glands, tumours of the omentum, growths from one or other part of the abdominal or pelvic parietes, hydatid cysts, &c., may so compress a neighbouring portion of bowel as to lead to obstruction.

DIAGNOSIS.—Careful examination, including that *per vaginam* and *per anum*, together with the history of the case, consideration of the collateral signs and symptoms, and in some cases, as where the swelling is cystic, an exploratory tapping or aspiration, will generally suffice to establish, approximately at any rate, the existence of this form of obstruction.

TREATMENT.—This consists, firstly, in the removal of the cause, if practicable; secondly, where this is impossible, in relieving the obstruction. By altering the position of the body, the displaced viscera may sometimes be moved so as no longer to compress the bowel; tumours—ovarian, uterine, renal, or hydatid—may be dealt with by operation. Where this is impossible, manipulation or copious enemata may be employed, or sedatives may be tried, in the hope that the bowel may, under their influence, release itself. But if none of these measures should be applicable or successful, and if the symptoms of obstruction be severe, resort to colotomy, or the making of an artificial anus by abdominal section, may become needful.

VIII. Obstruction from Impaction of Foreign Bodies, Intestinal Concretions (Enteroliths), &c.

(a) Foreign bodies in bulk, such as bones, coins, &c., occasionally find their way into the intestines. In a considerable proportion of cases they are evacuated *per anum* without much inconvenience; in some cases they give rise to enteritis and various other intestinal troubles; in some rare cases they lead to more or less complete occlusion, with acute or subacute symptoms.

(b) Foreign bodies, as hair, &c.; skins, seeds, and stones of fruit (the husks of cereals, and oats especially), the curds of milk in young children, and some medicinal substances, as magnesia, chalk, iron oxide, &c., taken over a long interval, may accumulate and give rise to more or less complete obstruction. Such masses constitute the large proportion of the so-called 'intestinal concretions' in the human subject.

(c) Hard concretions (enteroliths), consisting for the most part of phosphates of lime and magnesium, with organic material, and having usually as a nucleus some foreign body or hardened fæces, have rarely been met with.

Intestinal concretions are most frequently found in the cæcum or rectum; much more rarely in the ileum. They are slowly formed, and rarely bring about intestinal obstruction. *See* CONCRETIONS.

TREATMENT.—As a rule, purgatives can only do mischief. Sedatives, combined with laxatives, favour the gradual onward passage of the foreign body. If absolute impaction has clearly taken place, and the symptoms are urgent, abdominal section and extraction may be imperative.

IX. Obstruction from Impaction of Fæces. Prolonged constipation may lead to definite obstruction by impaction of fæcal masses, conjoined with paralysis and inaction of the bowel from distension, and contraction of the empty portion below. Sometimes the occlusion is rendered more absolute and irremediable by the doubling or dragging down of the bowel by the weight of its contents. The seat of the obstructing fæcal mass is usually the sigmoid flexure or the rectum; but great accumulation and its effects—distension, ulceration, perforation—are often most manifested in the cæcum. This cause of obstruction is most frequently met with in women of sedentary habits, after middle life, and especially among hypochondriacs or lunatics.

SYMPTOMS.—These are characterised by their chronicity; complete occlusion, as a rule, coming about slowly. There is little or no actual pain during the earlier stages; and even during the later stages, in the absence of complications, it rarely becomes acute. Vomiting is altogether absent at first; in the later stages there may be much nausea, accompanied with foul evacuations, and, towards the last, fæculent vomiting. Absolute constipation is slowly established, and then may last for two or three months. In a few cases the extraordinary periods of seven, eight, or even nine months have been reached without relief (Treves). Before the constipation has thus become absolute, it has very likely been interrupted by attacks of diarrhoea, due to catarrh of the intestine above the impaction, and giving very imperfect relief. The fæcal masses can sometimes be felt on examination of the abdomen, or they may be concealed by the presence of fat or flatulent distension.

COURSE.—In a considerable proportion of these cases relief may be afforded by appropriate treatment. In some, death ensues from gradual exhaustion, from peritonitis after ulceration and perforation, or from acute obstruction owing to the bowel becoming suddenly blocked or acutely kinked.

TREATMENT.—For this *see* CONSTIPATION.

In the extremest cases, while colotomy is to be looked on as the very last resort, the practitioner should avail himself of this rather than allow his patient to die of over-distension and ulceration of the intestine. When relief has been obtained, the greatest care as to diet and after-management is necessary, in order to prevent that recurrence of trouble to which the patient remains liable. *See* CONSTIPATION.

X. Obstruction from Congenital Malformation.—Constriction or occlusion of this kind is very rarely met with in the duodenum at or about

the entrance of the common bile-duct, or about the junction of the duodenum with the jejunum; and in some cases has appeared to depend upon valve-like folds of mucous membrane, resembling enlarged or confluent valvulæ conniventes. The lower portion of the ileum, near the ileo-cæcal valve, or about the junction with the omphalo-mesenteric duct, appears most likely to be so affected. The colon (and almost exclusively the sigmoid flexure) is the part of the bowel most frequently constricted by the effects of foetal peritonitis, but instances are very rare.

All such cases are of pathological interest rather than of practical importance. Vomiting of meconium, absence of proper evacuation, straining, convulsions, and evidence of more or less severe suffering, are followed by speedy death, though in some rare instances life has been prolonged for weeks or even months. No treatment can avail, and surgical operations can only hasten death, or at best succeed in prolonging misery.

Very much more common, and somewhat more hopeful, are those cases in which there is congenital defect of the lower part of the rectum or anus, or both. They may be divided into—(a) imperforate anus; (b) anus in the natural position, but the rectum deficient.

TREATMENT.—Immediate relief may often be afforded by surgical operations, and in some instances more or less permanent good results have been obtained, and by persevering management maintained; but survival to adolescence or adult age has seldom ensued. See RECTUM, Diseases of.

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INTESTINAL WORMS.—See ENTOZOA.

INTESTINES, Diseases of.—SYNON.: Fr. *Maladies de l'Intestine*; Ger. *Krankheiten des Darmes*.

The various morbid states exhibited by the intestines are attributable to (1) the effect of irritants introduced into the alimentary canal or developed therein in the course of perverted digestive action; (2) the invasion of parasites, microbic and other; (3) the influence of an unsuitable environment such as extremes of heat or cold; (4) functional disturbances of other systems, nervous, circulatory or excretory, or the conditions of general malnutrition which may be thereby brought about; or (5) lastly, to circumstances not as yet recognised. The influence of age and sex in the production of intestinal disease is largely determined by the extent to which these factors favour the incidence of the foregoing conditions. See PAIN in VISCERAL DISEASE.

The several diseased conditions of the intestines will be discussed in the following alphabetical order:—(1) Abscess in Walls; (2) Actinomycosis; (3) Amyloid Disease; (4) Anthrax; (5) Atony; (6) Atrophy; (7) Casts of; (8) Catarrh; (9) Concretions in; (10) Congestion; (11) Contraction; (12) Dilatation; (13) Gangrene; (14) Hæmorrhage from; (15) Hypertrophy; (16) Inflammation; (17) Malformations and Malpositions; (18) Neuroses; (19) New-Growths; (20) Paralysis; (21) Parasites; (22) Perforation and Rupture; (23) Spasm; (24) Syphilitic Disease; (25) Tubercular Disease; (26) Ulceration; (27) Vascular Changes.

1. Intestines, Abscess in Walls of.—See Inflammation of, p. 790.

2. Intestines, Actinomycosis of.—See ACTINOMYCOSIS.

3. Intestines, Amyloid Disease of. ANATOMICAL CHARACTERS.—See AMYLOID DISEASE.

SYMPTOMS.—The most prominent symptoms which this condition gives rise to, so far as the alimentary canal is concerned, are diarrhœa and hæmorrhage. Since the other important viscera are always simultaneously affected, other symptoms co-exist. The diarrhœa is rather characterised by fluidity than undue frequency of the stools, though the latter does occur; the evacuations often contain altered blood. It is rare to find either pain or tenderness; and the diarrhœa when once established rarely ceases. Hæmorrhage from the surface of the mucous membrane, independently of any ulceration, may occur from rupture of the diseased vessels.

TREATMENT.—Enemata of starch and opium are useful, though their effect is but temporary. Recovery, even from an advanced state, may follow if the cause, such as a suppurating joint, can be completely removed.

4. Intestines, Anthrax of.—See PUSTULE, Malignant.

5. Intestines, Atony of.—See NEUROSES of, p. 796.

6. Intestines, Atrophy of.—A general atrophy of the intestines accompanies a wasting of the entire body from any serious cause of malnutrition, such as starvation, where these organs are estimated in fatal cases to lose 42 per cent. of their weight, becoming extremely thin and transparent.

Intestinal catarrh may lead to atrophy of the bowels, even to an extreme degree. It may follow on a catarrh which is acute—this being frequent in children—or on the subacute or chronic forms. Certain parts of the canal are more liable to be affected than others, the cæcum, ascending colon, and lower end of ileum being the regions most commonly attacked. The change is almost restricted to the mucosa, the villi of the small intestine especially suffering, the thickness of the coat being reduced to one-fifth the normal in extreme cases, together with complete disappearance of Lieberkuhn's glands. The entire layer is replaced by connective tissue containing a few round cells, and presents a smooth appearance, with irregular thickenings at places; there is none of the pigmentation so constantly seen in the fibroid change of chronic catarrh; the muscular tissue, even of the mucosa, and the lymphoid follicles are unaffected. Nothnagel has also described areas of the muscular coat congenitally atrophied.

The symptoms referable to this condition, when the small intestine is involved, are those of general malnutrition from deficient absorption of the digested foods, and this may be so severe as to prove fatal, especially in infants and children. When the change is limited to the colon, there may be nothing beyond a slight diarrhœa, with bulky pulaceous offensive stools free from mucus. No treatment is specially available for this condition.

7. Intestines, Casts of.—See CASTS, p. 254.

8. Intestines, Catarrh of.—See p. 790.

9. Intestines, Concretions in.—SYNON.: Enteroliths; Bæzoars.—These are commonly met

with in the colon, cæcum, and appendix, which offer greater facilities of lodgment, and are liable to cause enteritis, ulceration, and perforation, or varying degrees of obstruction. See CONCRETIONS; STOOLS; and INTESTINAL OBSTRUCTION, p. 774.

10. Intestines, Congestion of.—See p. 804.

11. Intestines, Contraction of.—The calibre of the intestinal canal may be diminished by the pressure of tumours; by structural changes in the walls, such as cicatrices; or by displacements of portions of the bowel in invagination, &c. Such causes of stricture are more properly described under intestinal obstruction. See INTESTINAL OBSTRUCTION.

The term 'contraction' may be applied to that state of shrinking which the gut is liable to present below the seat of any permanent stricture.

Congenital malformations, producing contraction of the canal, even to complete occlusion, may be met with.

12. Intestines, Dilatation of.—The normal diameter of the small intestine may be taken as $1\frac{1}{2}$ inch throughout; and that of the large intestine as gradually diminishing from $2\frac{1}{2}$ inches at the cæcum to $1\frac{1}{2}$ inch at the upper part of the rectum. But the canal is evidently capable of distension much beyond these limits, as may be recognised when large accumulations of flatus or fæces occur. Such conditions, however, may disappear after death, the bowel returning to its proper capacity. These dilatations, therefore, may be regarded as temporary.

Other forms of distension of a more permanent nature are frequently observed.

Cases are sometimes met with in which extreme distension of the stomach, small intestine, and colon, either separately or together, occurs without any obvious cause, but perhaps associated with constipation. These have been provisionally termed *idiopathic*, and compared to similar affections of the œsophagus and stomach. The condition has been met with in children even at birth, or within the first three years of life, complicating diseases unconnected with the gastro-intestinal canal, such as broncho-pneumonia, or posterior basic meningitis, as well as in adults, and usually runs an acute and fatal course. In one case the colon was uniformly distended to a diameter of 6 to 8 inches; and in another the average diameter of the small intestine was twice the normal, the stomach also sharing in the distension; the person had been a large eater, and was extremely fat. The sigmoid flexure appears to be very prone to this distension.

In the greater number of cases the dilatation is attributable to the existence of some stricture in the course of the canal; and inasmuch as a persistent obstruction is usually located somewhere in the large intestine, it is the colon which most frequently suffers, and this may be so distended as practically to obliterate the ileo-cæcal valve. The mere accumulation and retention of the contents above the obstruction is doubtless one factor in causing the distension; but a diminished resisting power on the part of the gut probably co-exists, brought about by malnutrition of its textures. The muscular coat of the dilated portions is usually hypertrophied, while the mucous membrane is thinned and peculiarly liable to ulceration, the decomposing contents furnishing an exciting cause.

Localised bulgings or sacculations of the bowel may be met with, sometimes involving all the coats, and at others as diverticula formed by protrusions of the mucous membrane between the muscular fibres covered only by peritoneum. These 'false or distension-diverticula' are commonly found in old people suffering from chronic constipation or some condition causing general distension of the intestines; they are single or multiple, occasionally occurring in great numbers, and globular in shape; are most frequent in the colon, where they extend into the appendices epiploicæ, and occur less often in the small intestine along the mesenteric border. Inflammation from the irritation of fæcal or foreign matter is liable to be set up in them, leading to perforation into the peritoneum, or very rarely to communication with the bladder.

The existence of any extreme dilatation may be recognised by inspection or manipulation of the abdomen, especially if the parietes be thin and wasted, as they frequently are in such cases. Tympanites is present to a variable degree, uniformly distending the abdomen or causing asymmetrical swelling. Fæcal vomiting may occur in connection with the existence of a dilated intestine, but this is rather to be attributed to the primary obstructing cause, in the symptoms of which the few indications peculiar to this condition are merged. In extreme cases, the movements of the diaphragm may be interfered with, and the heart's action impaired even fatally.

Paralysis or atony from wasting or degeneration of the muscular coats, by diminishing the resistance of the bowel, allows of its distension. This is well exemplified in the extreme dilatation from flatus which so frequently accompanies acute peritonitis; and is comparable to certain cases of gastro-ectasis of a temporary character. See TYMPANITES.

13. Intestines, Gangrene of.

ÆTIOLOGY.—The immediate cause of the absolute death of a portion of the intestine is the complete arrest of the flow of blood through the part affected. This obstruction may be produced by:—

(i.) Embolism of the superior mesenteric artery. Several cases of this condition have been recorded.

(ii.) Thrombosis of the mesenteric veins. The perfect stasis induced by this cause is of very rare occurrence, but it has been seen to follow invasion of the portal vein by malignant disease, and associated with thrombosis of the femoral vein in the puerperal state.

(iii.) Detachment of the mesentery close to the intestine will be followed by gangrene of the part which is thus deprived of its blood-supply.

(iv.) Local constrictions of the bowel. This is by far the commonest class of causes of gangrene, and is the probable sequel of an unreduced strangulated hernia, an invagination (intussusception) or ileus. In these states the vessels are pressed upon, owing to the altered position of the gut, which, with the continuously increasing pressure of the œdema that follows the venous obstruction, leads to complete stasis.

(v.) The more gradual obstruction to the blood-flow, from constriction of the vessels by diseases of their walls, leads to sloughing, and tends to occur in amyloid disease of the intestines.

(vi.) Sloughing also occurs as a sequel of the long-continued pressure of hard fæces, or of the inflam-

matory state, when the process is of such intensity that complete cessation of the circulation takes place in localised spots, usually affecting the mucous membrane only, though occasionally penetrating deeper, ulcers remaining after separation of the sloughs. Owing to this cause is the frequently met gangrene of the vermiform appendix.

ANATOMICAL CHARACTERS.—From the nature of the intestinal tissues, the gangrene which is met with is of the moist variety. The portion of bowel which is affected is at first of an intense red colour, gradually increasing to purple, and even to black. The extreme congestion of the vessels leads to effusion of blood into the tissues, which are uniformly coloured; decomposition rapidly takes place in the stagnant blood, and the products acted on by the sulphuretted hydrogen of the intestines become black, all traces of red colour being soon lost. Meanwhile the mucous membrane and muscular coats are swollen and sodden by the serum and blood with which they are infiltrated; and a dark, black to ash-grey, soft, pulpy mass is finally thrown off from the healthy tissue. The extent of substance which may undergo this necrosis and be separated is extremely variable, from a mere slough of half an inch in diameter or smaller, to portions of bowel several feet in length.

SYMPTOMS.—The occurrence of symptoms whereby gangrene of the bowels can be diagnosed is scarcely to be expected. The signs for the most part resolve themselves into those of the cause, whether that be a plugging of the mesenteric vessels, or a localised enteritis. Extreme fetor of the stools may suggest its presence, but can afford no indication of the extent or depth of bowel involved, which are all-important data for prognosis, and to some degree for treatment. It is not until the sphacelus has been passed *per anum*, or until signs of ulceration are manifest, that the positive existence of gangrene can be ascertained. A very few hours suffice to produce this condition when once the cause is established; and since it cannot be either arrested or cured, the separation of the slough is to be desired, although fatal hæmorrhage or perforation may be associated with this process. Intestinal gangrene is always to be regarded as grave, though recovery not infrequently follows sloughing of considerable tracts when confined to the mucous membrane.

TREATMENT.—The circumstances associated with this morbid process as a rule preclude any treatment being specially directed towards it. If there be reason to believe that the entire thickness of the intestine is gangrenous, operative proceedings should be at once resorted to. Considerable success has followed resection of the dead part of the bowel, and union of the segments by sutures (*enterorrhaphy*).

14. Intestines, Hæmorrhage from.—An escape of blood from the intestines is a sign of certain morbid conditions rather than an actual disease itself; hence the cause of the hæmorrhage must be sought for.

ÆTIOLOGY.—The causes of intestinal hæmorrhage may be thus indicated:—

(a) *Increased blood-pressure*: Extreme congestion from such states as heart-disease, portal obstruction, embolism or thrombosis of the mesenteric vessels, intussusception, &c.

(b) *Affections of the intestinal walls*: Injuries of

the bowels; ulceration; vascular growths; hæmorrhoids; amyloid disease of the walls.

(γ) *Primary morbid blood-states or deterioration of vessels*: Purpura hæmorrhagica; hæmophilia; scurvy; leucocythæmia; yellow fever and severe intermittent and remittent fevers.

(δ) *Occasional causes*: Bleeding into the bowel from the stomach; rupture of an aneurysm into the intestine.

The mere enumeration of the causes must here suffice. It is obvious that the relative frequency of these conditions differs considerably, and in many cases the cause is at once apparent, while occasionally the source of the blood may be more obscure. It would seem from statistics that intestinal hæmorrhage is of more frequent occurrence in males, as gastric hæmorrhage is more common in women; the latter fact being explained by the greater liability of females to ulcer of the stomach, as the former appears to be by the preponderance of males suffering from the determining causes of hæmorrhage, such as liver-disease.

SYMPTOMS.—Associated with the symptoms special to the loss of blood, which are in the main similar to bleeding from any other organ, there are the signs and symptoms of the causal disease. The extent of the hæmorrhage will necessarily largely determine the symptoms, many bleedings being so trivial as to give rise to no appreciable effects, and in extreme cases the loss being so great and sudden as to lead to rapid collapse and death. Between these extremes all degrees of anæmia, faintness, pallor, giddiness, and failing pulse may be observed. A sensation as of a warm fluid flowing into the abdomen is occasionally complained of, but otherwise hæmorrhage in this situation is seldom possessed of characteristic features. Abdominal pain may accompany intestinal hæmorrhage, and is specially severe in embolism of the mesenteric arteries, but this symptom is not to be attributed to the bleeding, both being associated manifestations of a common cause. The occurrence of the above-mentioned indications in the course of a disease liable to lead to this condition would point to hæmorrhage, especially if there be a fall in temperature from a previous pyrexial state.

Occasionally the escape of blood is beneficial. This is particularly the case where the cause is a congestion of the intestinal tract, with or without hæmorrhoids. Thereby the fulness of the bowels is relieved, and a more equable circulation is established. In some cases of typhoid fever, contrary to what might be supposed, improvement has been noticed to follow a moderate loss of blood (Trousseau).

Except in such cases as when the effusion of blood is so excessive that death takes place before any escapes from the bowel, intestinal hæmorrhage reveals itself sooner or later in the character of the evacuations. If the cause be situated immediately within the anus, or the blood be sufficient in amount to escape alteration, then the red colour is retained. The hæmatin is readily affected by the sulphuretted hydrogen in the canal, and converted into a blackened material, sulphide of iron being formed, which stains the fæces; or a black tarry substance is evacuated, being the altered clotted blood (*see MELÆNA*). When the blood has undergone this change, the source of it is in the stomach or first part of the duodenum; blood from the colon—where it is usually due to ulceration—being passed adherent

to the fæces. The position of the source, the quantity, and the duration of its stay in the canal, largely determine the extent of alteration in the blood, and its degree of admixture with the fæces.

DIAGNOSIS.—The history of the case; the condition of the patient; and the character of the voided blood (*see* *MELÆNA*; and *STOOLS*) are the points upon which a diagnosis of the cause of intestinal hæmorrhage is to be based.

PROGNOSIS.—The amount of blood evacuated is not a sure guide to forming an opinion of the result. It is difficult to estimate the actual quantity lost, since much may be retained in the bowel. The general condition of the patient, especially the state of the pulse, is of far more importance; while allowance must be made for the nature of the cause, not forgetting the occasional favourable import of a flux.

TREATMENT.—In a certain number of cases bleeding from the bowel is uncontrollable; in others it is capable of cure; while in a third group it is rather to be encouraged. When arrest of the hæmorrhage is desired, rest, both general and local, is essential; the patient should be maintained in the recumbent position, as thereby the liability to syncope is averted; and the canal is to be kept quiet by abstinence from food, and the free use of opium, to prevent peristalsis.

The active treatment is to be directed to withdrawing the blood as much as possible from the affected region, by means of heat, sinapisms, dry-cupping, &c.; and to the application of styptics to the bleeding surface, or the administration of such remedies as arrest bleeding after their absorption into the blood. The most effective agents given by the mouth are turpentine in a full dose of 40 to 60 drops, followed by half-drachm doses every three hours; and the *Pilula Plumbi cum Opio* (B.P.) gr. v., every four or six hours. Tannic acid and the vegetable astringents are usually too slow in their action to be of much avail. Bitartrate of potassium in two-drachm doses is of much benefit in arresting the bleeding of piles; for which purpose also, as well as for vicarious hæmorrhage, or the flux of passive congestion from the lower bowel, the writer has found frequently repeated doses (m v. to m viii.) of tincture of hamamelis most efficacious. Probably the most reliable remedy is the *Injectio Ergotæ hypodermica* in five-minim doses, repeated if needful. Should the source of the hæmorrhage be within the range of local application by rectal injections, the most useful agents for this purpose are turpentine one to three drachms, with four to eight ounces of mucilage of starch; equal parts of tincture of perchloride of iron and water; or tincture of hamamelis, a drachm to three ounces of water, as an enema. Such astringents as tannic acid, opium, or hamamelin may be conveniently administered in the form of suppositories.

When the hæmorrhage is distinctly the result of engorged vessels, its occurrence should not be checked, provided it be not excessive. Sulphate of magnesium in full doses, with a few minims of diluted sulphuric acid, is then of great service.

The giving of stimulants is a procedure that involves careful judgment. While undoubtedly the tendency of loss of blood is to produce death by syncope, it is also true that faintness itself favours the cessation of the bleeding, and, so far as a general direction can be given, stimulants should be avoided, unless there be reason to fear, from the condition of the patient, character of the pulse, &c.,

that the syncope is grave. Short of that, alcohol, by temporarily increasing the heart's power, increases the bleeding.

Transfusion of blood or of 'normal saline' solution should be resorted to in extreme cases. *See* *SALINE SOLUTION*.

15. Intestines, Hypertrophy of.—This is always of local occurrence, a general hypertrophy, involving the entire length of the bowel, being practically unknown.

In chronic enteritis the mucous, submucous, and even muscular coats are apt to become much thickened, and though this is partly due to an excessive formation of connective tissue, there is also some actual hyperplasia of the normal structures.

In portions of the intestines above an obstruction, a true hypertrophy of the gut, particularly of the muscular layers, is to be found; and, as already said, this is usually associated with dilatation of the tube. Marked hypertrophy, especially of the longitudinal muscular bands of the colon, has been noticed in those cases of so-called idiopathic dilatation where no obvious obstruction exists. The pathogeny of this condition is obscure, for it does not appear probable that it precedes the dilatation, as in the heart, but rather that it is developed coincidentally.

It is rare for this condition to be other than inferred during life; it often gives rise to no symptoms and calls for no treatment; and when established is rather of the nature of a compensatory state. The congenital cases are, however, generally fatal, but relief is sometimes afforded by right-sided colotomy.

16. Intestines, Inflammation of.—*SYNON.*: Enteritis; Fr. *Entérite*; Ger. *Darmentzündung*.

Under this term are included all those structural changes in the mucous membrane of the intestinal tract which primarily follow the application of an abnormal irritant, provided that the irritant be not of sufficient intensity to produce absolute destruction of tissue. Such changes will involve more or less all the tissue-elements of the mucous membrane, and may extend to the muscular, or even the peritoneal coat. The inflammatory process may present considerable variety in type. The simplest form, to which the term 'catarrh' may be applied, passes, by almost insensible gradations, from the tissue-changes met with in the course of normal digestion, to a distinct condition of disease. To this there may be superadded certain specific characters due either to the nature of the cause, or to the predisposition of the tissue affected, or to both (*see* *INFLAMMATION*). There are thus differences in the severity with which enteritis may occur; but in all cases the essential characters of inflammation are present, which may be regarded as the results of the irritant *plus* the efforts at repair on the part of the affected tissue.

The morbid process may affect the intestine throughout the greater part of its length, either in common with or independently of the stomach—*general enteritis*; or it may be distinctly limited to certain parts of the canal—*local enteritis*, including duodenitis, typhlitis, colitis, and proctitis. As a rule, the term *enteritis* is restricted to inflammation of the small intestines, while *colitis* denotes a similar affection of the large bowel.

In respect to duration and intensity, enteritis may be *acute* or *chronic*.

(A) **Acute Enteritis.**—Acute enteritis is meant

to include all those cases where the essential features of an inflammation are present, varying in severity from a simple catarrh or muco-enteritis, to those graver forms possessed of special features, such as suppuration and ulceration. The more severe cases, especially in children, are sometimes called simple or English cholera, or *cholera infantum*.

ÆTIOLOGY.—*Predisposing causes.*—(a) Age especially predisposes to enteritis; for although it may occur at any period of life, infants and children during the period of dentition are peculiarly susceptible.

(b) The season of the year appears to exercise an influence, for during the summer and early autumn this disease is certainly much more frequent; and particularly so when there is extreme difference between day and night temperatures, or when the heat is associated with much drought.

(c) Occasionally this malady would appear to be epidemic.

(d) Certain mental and emotional states undoubtedly confer a liability to the occurrence of symptoms which are practically indistinguishable from those of a catarrhal enteritis.

(e) Conditions of general ill-health and marasmus, especially in children, predispose to intestinal catarrh.

Exciting causes.—1. Irritating ingesta of the most varied kind, such as abnormal, ill-cooked, or improperly digested food, toxic products of digestion, and irritant drugs or poisons, often cause enteritis, though not so frequently as in the corresponding affection of the stomach. Of these, improper food is by far the most common, especially during the first year of life. Among the most important of the causal agents of this group are the alkaloids or ptomaines and certain albumoses which result from the putrefactive decomposition of proteids either before ingestion or afterwards. These substances appear to be the determining factor in certain severe, and often fatal, forms of enteritis (see FOOD-POISONING). Certain irritants, such as corrosive sublimate, used as lotions or injections, have been known to produce enteritis—even fatal—without direct introduction into the alimentary canal.

Specific forms of intestinal inflammation are determined by various micro-organisms and their products, and to such causes may be referred cholera, dysentery, typhoid fever, and probably many cases of 'summer diarrhoea' or cholera infantum, all of which are primarily varieties of enteritis. Some of the normal micro-organisms of the intestinal canal appear under certain conditions of their environment to be able to assume pathogenetic powers (see *BACILLUS COLI COMMUNIS*). Many cases of food-poisoning have been known to be due to the *B. enteritidis* and *B. botulinus*, with which meat may be infected though itself giving no evidence of putrefaction or unsoundness.

2. Exposure to cold may be followed by inflammation of the intestines, as it may be by inflammation of the lungs, kidneys, or pleura. See CHILL.

The occurrence of inflammation and ulceration of the duodenum which occasionally follows extensive superficial burns cannot be altogether explained by the hyperæmia of the intestines, which is said to follow the superficial injury; but whether the cause be embolic, or the elimination by the bile of some irritant poison, is unknown.

3. Wounds, new-growths, volvulus, intussuscep-

tion, herniæ, impaction of feces, gall-stones, and parasites, will lead to enteritis.

4. Inflammation of neighbouring parts may involve the intestines by extension, as from the stomach, peritoneum, or bile-ducts.

5. An inflammatory state of the intestinal mucous membrane occasionally accompanies or follows the exanthemata; and it sometimes complicates septicæmia, particularly when this is of puerperal origin.

ANATOMICAL CHARACTERS.—It probably never occurs that the whole length of intestine is the seat of inflammation, and it is not often that even the entire small intestine is so affected. It is far more frequent to find certain tracts, of a few inches or a few feet, involved. Speaking generally, the colon, cæcum, rectum, duodenum, ileum, and jejunum are attacked, as regards frequency, in the order named. In some situations special features are present, but the essential characters of inflammation always exist, whatever be the site.

Owing to the physical properties of the intestinal tissues, and the rapid onset of softening and putrefactive changes, the appearances seen after death by no means necessarily correspond to what actually exists during life. Thus, the hyperæmic state of the mucous membrane, with the increased redness, varying from a more intense pink than normal up to a deep dark red, may leave but a trace *post mortem*, the vessels having become considerably emptied from the constriction of the vessels in *rigor mortis*. An increased vascularity, however, is one of the important features of the state under consideration, and it may sometimes be so intense as to lead to capillary rupture and formation of petechiæ in the mucous membrane. The tissue-elements of the gut, as a result of the irritant causing the inflammation, with the accompaniment of an increased vascular supply, undergo changes in their appearance and behaviour. Thus the epithelial cells are in a state of cloudy swelling, and leucocytes transude into the tissues from the vessels. Should the inflammation assume a suppurative character, collections of pus (abscess) may form in the thickness of the intestine, and ultimately burst on the mucous surface or into the peritoneal cavity. In all cases there is some cedema of the intestinal walls from serous effusion, and the free surface of the membrane is covered with a glairy mucus, containing leucocytes, and frequently crystals of triple phosphate. The epithelium of the follicles of Lieberkühn becomes extremely granular, and proliferates extensively, with the frequent result of blocking up the lumen of the gland, which thus becomes very prominent; or the epithelium may loosen and fall out, leaving well-defined empty pits, particularly if the examination of the mucous membrane be delayed long after death. The solitary and agminated glands are invariably much swollen, and very often the process of inflammation is most intense in their vicinity. Occasionally the mesenteric glands are similarly affected.

How this inflammation may terminate very much depends on the cause and extent; it may subside, and the bowel gradually assume its normal characters with no impairment of function; it may lapse into a chronic state; or it may pass on into ulceration, or even sloughing and gangrene.

To these various degrees and varieties of the inflammatory state different terms have been applied.

Catarrhal Enteritis (muco-enteritis).—The essential characters of this are an excessive mucous secretion, with desquamation of the epithelium. The submucosa may be infiltrated with leucocytes, but it usually remains normal, as also the muscular coat, unless the morbid condition be prolonged. Complete recovery generally takes place, but atrophy with considerable destruction of villi and Lieberkühn's glands, leaving the surface smooth and bare, not infrequently follows, especially in children. There is often some superficial erosion or simple ulceration. This form of inflammation involves great lengths of the canal, and is more common in the colon or ileum, less so higher up. See COLITIS.

Suppurative.—This term denotes a more severe form of inflammation, in which there is a formation of pus in the submucosa which may permeate all the coats. This is a very rare condition, and affects only a short distance of the bowel, which is deep red and distended, and frequently contains a sanious fluid.

A form of enteritis to which the term 'croupous' or 'diphtheritic' has been applied is of occasional occurrence in the small intestine, and rather more frequently in the colon. It is characterised by the formation of membranous patches of varying extent which consist of the necrosed mucous membrane containing a fibrinous exudation. It denotes a very severe form of inflammation, and the neighbouring bowel is swollen and purple, and tends to become gangrenous. See p. 321.

The term *dysenteric* is indifferently applied to more than one form of enteritis or colitis. The writer thinks that it had better be limited to that form of inflammation due to the specific organism of dysentery. See DYSENTERY; and COLITIS.

SYMPTOMS.—That a considerable variation is met with in the kind and severity of the symptoms presented in cases of enteritis, is only to be expected when the great difference in degree and extent of morbid change that is met with is remembered, as well as the great variety of causes; and in a very large number of cases the symptoms are quite out of proportion to the appearances found *post mortem*. Whereas one patient may suffer from an attack of intestinal catarrh, with but a trifling array of symptoms, another may succumb within a few days. There is no one symptom or even group of symptoms that is absolutely characteristic of the disease; even the general condition of the patient is not constant. In the milder forms of intestinal catarrh there may be slight pyrexia, with thirst and quickened pulse, but these symptoms may be scarcely noticeable, and the actual existence of the condition is often assumed, without anything like proof; while, on the other hand, in the severe suppurative enteritis they are extreme, and the patient is in a state of considerable prostration.

The following symptoms, more or less marked, occur in different cases.

Stools.—Diarrhœa is in some respects the most constant symptom, though, when the affection is limited to the higher part of the canal, and space is given for the re-absorption of the excessive exudation, it may not only be wanting, but there may be actual constipation. The lower down the bowel is inflamed, the greater the liability to diarrhœa, which hence becomes a marked character of colitis and proctitis. In the severe forms of the affection complete constipation may be due to the paralysis

of the inflamed bowel, arresting the peristalsis, and allowing of the accumulation of the intestinal contents above the lesion; this is obviously more complete when the enteritis is associated with any state producing mechanical obstruction, such as intussusception or ileus.

The character of the evacuations is very variable. As a rule they are semi-fluid when diarrhœa exists; or they may consist chiefly of an almost clear liquid with a few feculent flakes; but, when time has permitted a partial re-absorption of the fluid portion, the stools become more consistent, and in cases of enteritis which are chiefly due to fecal accumulation, solid hard masses are passed.

Mucus, in greater or less quantity, is constantly present—being especially abundant in affections of the large intestine and rectum, when it is often discharged as complete tubular casts of the bowel. In catarrhal affections of the small intestine, the mucus may only occur in microscopic particles, and when considerable in amount and intimately mixed with the fæces, generally comes from the first part of the colon; motions consisting of almost pure mucus come from below the splenic flexure.

Blood is not usual except in the severer forms, unless there be ulceration or hæmorrhoids; and pus is seldom noticed unless the rectum be inflamed. Quantities of intestinal epithelium, crystals of triple phosphates, and micro-organisms, especially the *Bacillus coli*, may be detected by the microscope.

Owing to the imperfect performance of digestion or absorption, the motions are liable to contain many abnormal constituents—as fat, when the duodenum and upper part of the jejunum are involved, or even masses of food scarcely changed; and the altered characters of the intestinal contents, with the products of decomposition, are in themselves most effective in maintaining a diarrhœa. As a rule, the discharges are paler than normal, or may be even colourless; the greenish tint so often seen in the enteritis of children is due either to a pigment formed by a bacillus, or to biliverdin; in the latter case it is considered as indicating some abnormal alkaline change in the intestine, whereby the biliverdin has become altered, though the stool itself as passed is acid in reaction. Unaltered bile-pigment in the fæces is abnormal; its presence indicates catarrh high up in the small intestine, and is always associated with fluid evacuations from increased peristalsis of the ileum and colon. The odour is usually extremely offensive or even putrid, especially the white or greyish putty-like masses passed by children; though sometimes, when the evacuations are very liquid and colourless, smell may be altogether absent. See STOOLS.

Owing to a large production of gases, discharges of flatus are of very frequent occurrence, but unless there be actual obstruction, tympanitic distension of the abdomen is not usual.

Vomiting, except in the severe forms, is not a common symptom of enteritis, unless the stomach be involved, and it is relatively more frequent in children than in adults. Short of actual vomiting, nausea is frequently complained of.

Pain and Tenderness.—Pain in itself is a most uncertain symptom, being scarcely noticeable in the milder forms of catarrh; while in colitis, the colicky, griping pains, which may or may not be relieved by pressure, are characteristic. Still more is this the case when the rectum is affected, when the straining and tenesmus constitute one of the

most distressing symptoms of the malady. When the peritoneum is involved, the pain and tenderness are marked and characteristic. Both may be generally diffused over the abdomen, or may be local in character, as over the cæcum; in a large number of cases the pain is referred to the umbilical region.

General Symptoms.—Among the more general symptoms, or those associated with inflammation of special regions, are the phenomena of the febrile state. The temperature may reach 102° F., or even higher; or in some cases it may be scarcely elevated. The appetite may be unaffected, especially if the upper part of the tract be free from the disease; while there may be complete anorexia when the reverse is the case. Thirst is of usual occurrence, and it becomes very marked when the evacuations are abundant and fluid. The tongue indicates rather the general state of the patient, and is a less reliable index of the actual state of the intestinal mucous membrane than in corresponding affections of the stomach. It may be dry, red, irritable, and glazed; or coated with a thick fur, with the edges and papillæ bright and prominent; in milder cases it is often unaffected. The urine may contain a large amount of indican, indicative of albuminous putrefaction in the intestines. The character of the pulse varies with the general state. If the pyrexia be extreme, there is the usual dry skin and concentrated urine, with a tendency towards the production of the typhoid state, which usually is reached in fatal cases. In many cases the prostration is excessive, though the mind is usually unaffected to the end, and very often there is a marked irritability of temper. Headache is of rare occurrence in enteritis. A persistent hiccough is met with sometimes. In children the disease rapidly leads to a condition of emaciation and collapse. The child lies in a languid, almost torpid, state, with the skin of the abdomen intensely hot and dry; while the extremities are cold and blue, the face is pinched, and the body generally appears shrunken. Frequently this state is interrupted by attacks of convulsions, especially if dentition be in progress; or the child is extremely fretful, and maintains an almost constant, short, feeble cry, evidently accompanied by pain.

When the disease affects the duodenum, jaundice, due to closure of the bile-duct, very often occurs. The intimate nervous relation between the rectum and base of the bladder explains the frequency of micturition so commonly associated with proctitis.

In cases of so-called 'ptomaine-poisoning' the symptoms supervene within a few hours after the meal, and run an extremely severe course, frequently terminating in death, although a great variety is to be noticed among different individuals exposed to the same cause. In addition to the evidences of a virulent gastro-enteritis, such as vomiting, diarrhoea, bloody stools, abdominal cramps, pyrexia, and later, prostration and collapse, certain nervous phenomena—convulsions, tetanic spasms, paralysis, and coma—are frequently present in varying degree.

DIAGNOSIS.—The variability and oftentimes vagueness of the symptoms frequently admit of a diagnosis of enteritis being made only by a process of exclusion. The history of improper feeding, whether temporary or prolonged, often suggests the nature of the disease; though it cannot be denied that all rules of a rational dietary are frequently

violated with apparent impunity both by children and adults.

Diarrhoea alone can by no means be taken necessarily to indicate the existence of intestinal inflammation, and the same may be said of constipation, pain, vomiting, and other single symptoms. It is rather to a group of symptoms, with the previous history, that the observer must look. The character of the stools, as already described, often indicates the region of gut affected; and the existence of extreme tenderness and pain, with a hard quick pulse, and the characteristic decubitus, point to the involvement of the peritoneum, which an exacerbation of temperature tends to confirm. The distinctive features of typhlitis, colitis, and proctitis are elsewhere described sufficiently to form material for diagnosis in most cases. To distinguish between inflammation of the jejunum and ileum is usually impossible, nor, practically, is it a matter of importance. The history of the case, the course of the temperature, and the characteristic rash and headache, as well as Widal's reaction, should serve to separate typhoid fever from acute enteritis, which is sometimes mistaken for it.

PROGNOSIS.—A simple intestinal catarrh occurring in a healthy subject certainly tends, after a few days, to complete recovery; but occurring, as it frequently does, in persons in ill-health, it is far more liable to pass into an obstinate chronic condition. In children, the opinion should be very guarded; for while in a large number of cases perfect recovery follows removal of cause and suitable treatment, others, for no very apparent reason, will, in spite of everything, progress to a fatal termination; and this is, of course, more likely to be the result where the child is weakly or the subject of other disease. The mildest diarrhoea in children suffering from athrepsia quickly becomes serious, killing as it were from rapidly supervening shock; the onset of so-called summer diarrhoea during convalescence from such diseases as whooping-cough or pneumonia is also of grave import; and in true cholera infantum, hyperpyrexia, great prostration, and especially uncontrollable vomiting, are almost surely fatal indications. Enteritis in different degrees of severity constitutes one of the most important causes, if not the most important cause, of infantile mortality.

In the severer forms of enteritis, as they affect adults, opinion must be guided by the nature of the cause, and the general state of the patient. Recognising that the unfavourable tendencies of the disease are towards extreme prostration, to perforation with fatal collapse, or to chronic ulceration—according as these conditions are threatened, so may the prognosis be fairly made. The duration of extreme cases rarely extends beyond a few days, when, if death do not occur from cardiac failure or pulmonary complication, the symptoms abate, and recovery, with oftentimes a tedious convalescence, follows, or a chronic condition of disease is established.

TREATMENT.—Although great variety exists in the degree of severity of the symptoms of acute enteritis, and a corresponding difference obtains in the treatment to be pursued, yet certain general principles may be first laid down, and the more special details adapted to certain conditions afterwards indicated. Inasmuch as the disease is one where the organs concerned with the preparation of the food are those mainly at fault, every effort should be made to minimise the bodily waste. This is best attained by keeping the patient in bed, which also offers

the additional advantage of providing a uniform warmth.

As regards diet, in the greater number of cases of acute intestinal inflammation the appetite is much impaired, even to complete anorexia. Provided that the person attacked have been previously in good health, no harm is done by complete abstinence from food for twenty-four or even forty-eight hours. This gives a much better chance of rest to the intestine, and a better opportunity for the removal of any irritant ingesta which may have been the cause of the inflammation. The thirst during this period may be relieved by ice-cold water, with or without a little lemon-juice. It must not be forgotten that, with the mucous membrane and its glands inflamed, the conditions of normal digestion and absorption are materially interfered with, and articles of diet that ordinarily are most nutritious and easily digested may and do become, under these altered circumstances, positively harmful. The aim in feeding the patient should be to give those materials which require the least digestion, and, being most quickly absorbed, leave the smallest amount of indigestible residue. If the stomach be implicated—and it is rare that it is not so—meat-foods are badly borne. Instead of the proteid constituents being digested, they undergo putrefactive decomposition, and thus add fresh irritants to the canal lower down. If, however, the stomach be tolerably free, then meat-essences, made thin and allowed to stand till cold, may be given. The nausea or vomiting which is usually present is more easily overcome by giving the nourishment cold; and a few drops of lemon-juice are of great service if added to the beef-tea. Milk is very uncertain in the way it is tolerated by such patients. Occasionally it is impossible to give it, the vomiting or diarrhoea being increased by it; but equal parts of milk and soda-water may constitute sufficient nourishment to last for several days in extreme cases, and may be well borne. Lime-water may be substituted for the soda-water, but, as a rule, effervescing fluids are more grateful, koumiss being often of great service. The milk should be sterilised and as free as possible from cream, for fats in all forms are to be avoided, since the products of their decomposition are extremely irritating. Beef-tea or chicken-broth made with milk in place of water may be tried with advantage. Farinaceous substances, if given at all, should be administered only in small quantities at a time; a remark which equally applies to all other food. Thinly made Benger's food or Plasmon is often most useful. Nutrient enemata are of value in some cases.

A very great deal may be done for the patient with drugs, both in the relief of symptoms and in aiding the cure.

It is seldom advisable to check the diarrhoea in acute enteritis; and an aperient to begin with, unless any evidence exist of peritonitis, is a rational treatment; thereby the irritant, whatever it may be, is removed, and a better chance for recovery is given. Improper food is so commonly the cause, that the majority of cases are benefited by a preliminary purgation. Probably the best aperients in this case are castor oil, and calomel in doses of one to four grains according to age, but it may be necessary to follow up the latter after a few hours with a quickly acting saline aperient. If the inflammation be confined to the colon, where, as already said, accumulations of feces are the common cause, copious

simple enemata, repeated every six or eight hours, are of great advantage; and this plan may be pursued in conjunction with the aperient given by the mouth. The object is to clear out the alimentary canal; and provided that this has been done, abstinence from food for twelve hours, and some bismuth in an effervescing form, are frequently sufficient in milder cases to put the sufferer on the road to cure. The writer places great reliance on bismuth, either in the form of solution with an effervescing citrate of potassium, and three or four minims of diluted hydrocyanic acid; or granular effervescing lime-juice and bismuth. The nausea and vomiting are best relieved by this treatment. For the pain, poultices or poppy-head fomentations, or the internal administration of opium, may be very effective. Should there be peritonitis, the opium must be increased in amount, with a view to giving complete rest to the bowel. Several leeches applied to the anus, to relieve the hyperemia of the intestinal tract, are occasionally necessary in extreme cases. When the attack is distinctly attributable to cold, a profuse sweating induced by hot baths, and ten grains of Dover's powder, is often of great benefit. In those cases where, from the duration, character of stools, and previous treatment, there is reason to believe that the irritating causes are got rid of, the diarrhoea may then require special treatment, especially when the patient has been in ill-health, or is constitutionally debilitated. A powder, consisting of Dover's powder 5 grains, and carbonate of bismuth 10 grains, given every six hours, is very efficacious. Vegetable astringents are frequently used for the same purpose.

When the more acute symptoms have subsided, the bismuth may be still continued, and later on given with a vegetable bitter, such as calumba. Ten to fifteen minims of the diluted hydrochloric acid in an ounce of water much assist recovery of the digestive power of the stomach, for which purpose also pepsin is valuable.

In consequence of the great liability to a second attack which this disease engenders, avoidance of well-known harmful articles of diet, and the use of warm clothing or flannel belts, are demanded as a prophylaxis.

In infants and young children the great liability to collapse, often rapidly fatal, must be borne in mind. Stimulants in some form are almost a necessity. The following prescription may be employed: *Liquoris Bismuthi et Ammonii Citratis* mj-ij, *Spiritus Ammoniae Aromatici* mj-v, *Tinctura Cardamomi Composita* mj-v; *Aqua* ʒj-ʒij—according to age. Brandy in small quantities is often the means of saving life in these cases. When the collapse is not threatening, one or two drops of the official solution of corrosive sublimate, with half a fluid-drachm of syrup, and a fluid-drachm and a half of water, every two or three hours, may be of great service. It is a more convenient mode of giving mercury than in the form of Grey powder. But in one form or another the writer believes mercury to be of prime necessity. Corrections of diet on the lines indicated above are of course essential. Hot baths and other means to keep the child warm must be employed.

In the severer cases, which are lapsing into the typhoid state, the general principles for that condition must be followed; but, except in such a state, alcoholic stimulants are rarely called for.

Inasmuch as there is good reason to believe that

many cases of intestinal inflammation, especially those of an epidemic character, are due to the activity of micro-organisms, the treatment of the disease on antiseptic principles has naturally been suggested. The problem has been to find a germicide which should pass to the ileum and colon without itself undergoing decomposition in the mouth, stomach, or upper part of the intestine, and should at the same time be harmless to the patient. Many are the agents which have been proposed, and with varying success. Few, if any, are superior to calomel or corrosive sublimate, or the solid preparations of bismuth. Thymol and salol are occasionally useful, but these latter, like resorcin, salicylic acid, salicylate and benzoate of sodium, salicylate of bismuth and cerium, the sulpho-carbolates of sodium or zinc, creosote, naphthalin, iodoform, and peppermint, are far more efficacious in checking fermentation in the stomach and upper bowel. Whichever remedy be employed, it should be given in frequently repeated small doses according to age. Irrigation of the colon by copious enemata of water, containing small proportions of one or other of the above-mentioned substances, is an effective method of intestinal antiseptics.

(B) Chronic Enteritis.

ÆTIOLOGY.—1. A certain proportion of the acute cases become chronic, the original cause persisting.

2. Those conditions which lead to a chronic state of congestion of the intestinal tract will thereby so affect the constitution of the tissues, with a consequent disturbance of function, as to constitute a chronic inflammation. The most important of these conditions is obstruction, either at the right side of the heart, or affecting the portal circulation in the liver.

3. Chronic enteritis is the occasional accompaniment of some general chronic disease, such as Bright's disease, when deteriorated blood may be regarded as leading to a chronic inflammation.

4. Residence in tropical climates is a not infrequent cause of lasting inflammatory disease of the bowels.

ANATOMICAL CHARACTERS.—The intestinal mucous membrane, when it has been the seat of chronic inflammation, is generally thickened, tough, and of a grey colour, from a deposition of pigment, due to the chronic congestion. The epithelial cells are cloudy and ill-defined, and there is a round-celled infiltration of the mucous and submucous layers passing into the stage of connective tissue; hence the thickness and toughness. This fibrous hyperplasia is often localised as polypoid elevations with intervening areas of atrophic mucous membrane. The lymphoid follicles are prominent and hard; the intestinal glands are frequently blocked with cells and secretion, and form minute solid perceptible masses, or are atrophied or even cystic; and the villi of the small intestine are shrunken and stunted. The surface of the membrane is more or less covered with a viscid glairy mucus, containing pus and imperfectly formed epithelial cells; not infrequently such mucus may be voided in the form of membranous-looking shreds or even complete casts of the tube, and this is particularly the case in the pellicular form of colitis (*see* CASTS). Sometimes the muscular coat is thickened from formation of connective tissue. As a rule, therefore, the bowel is increased in thickness; but in children it not infrequently happens that a chronic enteritis is associated with an atrophy of all the coats and the con-

tained glands, the tube being much thinned and parchment-like, and of a slaty tint. It is unusual for a chronic inflammation of the intestine to exist in adults without coincident ulceration, which is often most extensive; but in children the disease may proceed to a fatal termination, and show no such condition after death.

SYMPTOMS.—It is not always easy to say exactly when an acute case has lapsed into a chronic state, very much the same symptoms being continued. In such affections of the small intestine, the diarrhoea may be wholly wanting, and the bowels may be very confined. This is due to the diminished peristalsis, from impaired irritability of the muscular coat. When, however, ulceration is extreme, and especially if it be the colon or rectum that is mainly affected, chronic diarrhoea is an invariable symptom. The remarks made on the character of the stools in acute enteritis are equally applicable to the chronic state, with the addition, that solid and liquid evacuations frequently alternate. Lasting as the disease often does for many months or even years, a general impairment of nutrition results. The functions concerned in the elaboration of the food, as well as that by which the digested products are absorbed, are necessarily perverted. The marasmus is speedily noticed in infants and children, whose growing tissues less readily withstand malnutrition. Apart from the general ill-health produced, the mental qualities become affected, so that the intellect may become dulled and sluggish, the temper irritable, and the patient may fall into a condition of marked hypochondriasis; this is particularly liable to be the case when the colon is the seat of the disease. The emaciated appearance; the dirty muddy complexion; complicated often with a short dry cough, dependent on reflex irritation from the stomach, frequently lead a superficial observer to suspect the existence of phthisis. *See also* COLITIS.

PROGNOSIS.—Chronic enteritis almost invariably tends towards a fatal termination, though this may be long delayed. The general nutrition becomes more and more deranged; and death from inanition finally terminates an existence of prolonged suffering and discomfort.

TREATMENT.—Owing to the unfavourable tendency of this disease, the treatment can be rarely more than palliative. The debilitating and wearying character of the malady emphatically calls for good feeding. The diet should be abundant and nutritious. During the exacerbations of membranous enteritis, the giving of food is often difficult owing to the vomiting and pain produced; in such cases koumiss is of great value. When the disease affects the large intestine, the ordinary digestive changes in the food have taken place, and the contents of the canal reach the colon in the normal semi-fluid condition; in this state they may be passed; but owing to the impaired movements of the affected bowel the feces are apt to accumulate, and constipation results. This should be guarded against by simple enemata, and the soothing effect of injections of warm water only is often very marked. Distinct benefit has been known to follow hydrotherapeutic treatment, especially if accompanied by free rectal injection. In those cases where the enteritis is a sequel of a congestion of the intestine, the treatment must be directed to relieve if possible the cause of that congestion. Since this is usually some such intractable condition

as Bright's disease, cardiac dilatation, or cirrhosis of the liver, attention should be directed to the relief of these affections.

Tonics—such as quinine, iron, bark, with sea-air—are of undoubted benefit; and, so far as possible, causes of mental worry should be removed. Massage of the abdomen and gentle movements specially directed to the exercise of the abdominal muscles are most beneficial.

17. Intestines, Malformations and Malpositions of.—These may be (a) *congenital*; or (b) *acquired*.

(a) *Congenital*.—Though seldom of much clinical importance, congenital malformations of the intestines are often of great interest from a developmental point of view. The malformation may be of the nature of an *excess*. Thus certain parts of the canal—duodenum, colon, and appendix vermiformis—have very rarely been found double. The commonest of all these malformations is a diverticulum of the ileum, which may arise from the free margin of the ileum from one to three feet above the ileo-cæcal valve. The cæcal extremity of the process (which is occasionally hammer-shaped) may be connected with the umbilicus by a thin fibrous cord, showing it to be an unobliterated portion of the vitelline duct, and is commonly known as *Meckel's diverticulum*. It varies in length from half an inch to six inches, or even more; its structure is exactly that of the ileum; and it has been found the seat of typhoid ulceration, or of perforation from the irritation of foreign bodies that have become lodged therein. In very exceptional cases diverticula of similar structure have been found protruding from the duodenum, just above the opening of the bile-duct, or from the jejunum; they may even be multiple, but they are not connected with the umbilicus. The vermiform appendix may vary from half to twice the natural size.

Deficiencies of development may affect the whole alimentary canal, or only certain parts. Andral records a case where only a straight tube joined the rectum and oesophagus. The ileum may open upon an ectopic bladder. The rectum may end in a cloaca common to the urino-genital organs, or in either the bladder, urethra, or vagina. The bowel may terminate in a closed extremity anywhere between the brim of the pelvis (the rectum being generally represented by a fibrous cord, though even this may be wanting) and the anal region immediately beneath the skin; the anal pouch, which develops from without inwards, is in the latter case absolutely wanting; and all degrees between this and a pouch that has just failed to establish a junction with the rectum may be met with, producing the lesion known as *imperforate anus*. The valvæ conniventes are sometimes wanting, or very imperfect, over varying areas of the small intestines. Congenital constrictions of different parts of the canal are occasionally met with—in the duodenum, either close above the opening of the common bile-duct or at the junction with the jejunum; in the lower end of the ileum, where some abnormality in the closure of the vitelline duct appears to be the cause; or in the sigmoid flexure. Such constrictions may be multiple and of very short extent, the canal being much dilated above, and extremely narrowed and shrunken below. The ileo-cæcal orifice has been seen contracted to the diameter of a

small cedar-pencil. The cause of these lesions is very obscure, though they may be occasionally accounted for by the existence of prominent valve-like folds of the mucous membrane.

Among congenital malpositions may be mentioned complete transposition of the viscera, the cæcum and ascending colon being on the left side, and the sigmoid flexure and descending colon on the right, the liver, spleen, stomach, &c., sharing in the change. Certain parts only of the intestinal canal may occupy an abnormal situation, as in the various congenital herniæ; or the displacement may be due to unusual length of the mesenteries, the cæcum and sigmoid flexure being the parts that present the most usual malpositions from this cause. Thus the cæcum may occupy the left hypochondrium or left iliac fossa, or be found in the pelvic cavity, and other parts of the canal show corresponding changes of place. The sigmoid flexure has been seen lying to the right side of the left kidney, which was situated immediately below the bifurcation of the aorta. Similar displacements are referable to adhesions, determined by intra-uterine peritonitis, which is frequently associated with syphilis.

(b) *Acquired*.—The acquired malformations include the dilatations and contractions that are associated with stenosis; and the adhesions and abnormal communications established by ulceration and peritonitis, elsewhere referred to.

Hernial protrusions of the mucous membrane through the outer coats, often very numerous, and varying in size from a pin's head to a walnut, have been seen in the colon, and less often in the small intestine, sometimes extending into the appendices epiploicæ. They are liable to become developed in cases of long-standing constipation.

The malpositions which the intestines may come to present from changes set up after birth are so variable as scarcely to admit of classification. Hernia, both external and internal, volvulus, and intussusception are among the well-recognised displacements; but there is scarcely any limit to the changes in position which the traction and pressure of tumours and the effects of peritonitis may produce. See **INTESTINAL OBSTRUCTION**; **HERNIA**; and **ENTEROPTOSIS**.

When any symptoms are produced by malformation or malposition of the intestines they are usually those of obstruction, and can only be dealt with surgically. See **INTESTINAL OBSTRUCTION**; and **ANUS, Diseases of**.

18. Intestines, Neuroses of.—Disturbances of the sensory, motor, and secretory functions of the intestine when of primary nervous causation constitute the intestinal neuroses.

1. *Sensory*.—Under normal conditions the several activities of the canal are performed without giving rise to any conscious sensation, nor does excitation of the mucous membrane by mechanical, thermal, or chemical stimuli give rise to pain. Stimulation, however, of the central end of a divided splanchnic nerve does cause pain, doubtless due to the contained myelinated fibres of the afferent spinal roots. As compared with the stomach, the intestine is certainly less liable to be the seat of painful affections, as witness the difference in cases of ulceration of the respective viscera. Nevertheless, severe enteritis, obstruction, and new-growths are frequently, and indeed usually, asso-

ciated with some pain. Further, when the peritoneum becomes involved the liability to pain is much increased. The commonest form of intestinal pain is that denominated *colic*, in which the idea of a painful muscular spasm is involved (see COLIC). Whether any lesion limited to the mucous membrane is capable of exciting painful impressions is uncertain, and equally obscure is the existence of a true neuralgia of the intestines (enteralgia), although the occurrence of pain in the bowels, often severe, is a well-recognised symptom in the course of hysteria, neurasthenia, hypochondriasis, and sometimes also in such definite maladies as *tabes dorsalis*. In all such cases the symptoms are identical with those of colic, and the treatment is the same.

2. *Motor*.—Paralysis of the bowels. A paresis of the intestinal movements may be brought about by causes acting (1) through the *nervous system*; or (2) through imperfection of the *muscular tissue*.

(a) *Nervous*.—Cases are recorded in which the intrinsic ganglia and nerves of the intestinal muscular coat have been the seat of degeneration, but the symptoms dependent thereon are doubtful, beyond atrophy of the muscular coat. Certain lesions of the brain are accompanied by symptoms of intestinal paralysis, but with no hitherto recognised regularity, and it is assumed that such lesions act by interfering with the function of the vagi. It is doubtful how far disease of the spinal cord produces actual paralysis of the intestines, though constipation may result, a circumstance that may be explained by assuming an interference with the centre that controls defecation. See DEFÆCATION, Disorders of.

(b) *Muscular*.—The irritability of the muscular tissue may be much weakened by degeneration (cloudy, fatty, or amyloid). Inflammation of the mucous or serous coat, especially the latter, is liable to determine granular change in the muscular fibres, which, aided by a co-existent œdema, largely impairs the contractile power of the tissue. Chronic congestion of the bowel leads to the same result. The irritability is also liable to be lessened by habitual constipation, as also from the over-stimulation of too powerful and too frequent purgative medicines taken for the relief thereof; and the muscular fibres of a much-dilated portion of the bowel are apt to become paralysed from distension and stretching. The general want of tone that the muscular and nervous systems manifest subsequent to debilitating diseases, or from want of food, hysteria, and other conditions, also finds expression in the alimentary canal, in diminished peristaltic action.

Certain astringent drugs produce their effect possibly by diminishing the excitability of the nervous system, as appears to be the case with opium. Lead, which would seem to cause both paralysis and spasm of the muscular coat, may act on the nerve-terminals or on the muscular fibres.

The prominent symptom of intestinal paralysis is constipation, though other signs of obstruction, such as vomiting and meteorism, may be super-added. See CONSTIPATION; and INTESTINAL OBSTRUCTION.

Treatment is, as a rule, directed to the primary cause, but great benefit has followed the application of electricity to the abdominal parietes. Massage applied on systematic principles is of undoubted service.

Increased peristalsis; spasm.—The irregular and forcible movements of the bowels, usually accom-

panied with pain, are known as *colic*. See COLIC.

The specially painful spasm of the anal sphincters and lower portion of the rectum, termed *tenesmus*, is usually associated with ulceration and other lesions in that locality.

An over-excitible condition of the intestinal neuro-muscular apparatus may show itself as an exaggerated peristalsis producing diarrhoea. Such a state is prone to occur in neurotic subjects or in emotional states. In some persons the muscular irritability is so much increased that the mere taking of food is sufficient to induce an action of the bowels. The intestinal crises of *tabes dorsalis* are extreme cases of this class.

The bromides, arsenic, and salicylate of bismuth are the most useful drugs in this condition.

(c) *Secretory*.—Although nothing is positively known as to the nervous control over the secretion by the intestine, experiments would indicate that under certain states of nerve-stimulation the flow may be much increased; and clinically in most of these conditions of 'nervous diarrhoea' there is, along with the exaggerated peristalsis, an excessive amount of fluid poured out into the canal, as evidenced by the watery motions. The mucous discharges from the bowel, and mucous casts, which characterise the so-called 'mucous colitis,' are more probably of the nature of secretory neuroses than inflammatory (catarrhal) in origin. See COLITIS, MUCOUS.

19. *Intestines, New Growths of*.—These may, for the present purpose, most conveniently be divided into *malignant* and *non-malignant*—a clinical distinction irrespective of their minute structure.

(a) *Malignant growths*.—ÆTIOLOGY.—Malignant growths of the intestine, as in other situations, while not wholly unknown in the earlier periods of life, are rarely met with before the age of forty, and oftener after fifty. Statistics show a slight preponderance of occurrence in males. From an examination of 9,000 fatal cases of cancer, the relative frequency of intestinal cancer to that of all other organs was found to be as 1 to 25 (Tanchou).

Cancer of the intestine is nearly always primary, and very frequently runs its course without any secondary formations elsewhere. Occasionally the bowels are affected by extension from neighbouring parts, and this is especially liable to be the case in the rectum, when the uterus or vagina is the seat of the disease, and in the duodenum, which may become involved in an extension from the pancreas, liver, gall-bladder, or stomach. Very rarely small nodules are found in the solitary and agminated glands secondary to carcinoma existing elsewhere.

ANATOMICAL CHARACTERS.—Malignant disease may occur at any spot throughout the entire length of both small and large intestine, but is infinitely more often to be met with at certain special parts, notably the rectum, sigmoid flexure, cæcum, colon generally, and duodenum near the opening of the bile-duct; the jejunum and ileum being rarely affected. It has been estimated that in 80 per cent. of the cases of intestinal cancer the rectum is affected; in 11·5 the colon; in 4·2 the cæcum, appendix, and ileo-cæcal valve; and in 4·3 the small intestine. There is undoubtedly a predilection for those spots where any delay may occur in the passage of the intestinal contents, such as the

flexures of the large intestine, especially the sigmoid.

The greater number of intestinal growths included in the category of malignant are of the epitheliomatous type, especially the cylindrical variety, which may undergo colloid change; while the scirrhous and encephaloid are much rarer. As a rule, malignant growths commence in the mucosa and submucosa, frequently in Lieberkühn's or Brunner's glands, and then gradually involve the other tissues. *See* CANCER.

The mesenteric glands are generally affected, though often but slightly. Secondary infection is much less common than in cancer of other organs; when present, the liver and peritoneum are the usual seats. The inguinal lymphatic glands are occasionally enlarged when the sigmoid flexure is the seat of the growth.

Following the general course of these neoplasms when found elsewhere, they may undergo degeneration and ulceration, thus suffering a diminution in bulk at one spot while they extend in other directions. In the course of their development they may set up adhesions between the bowel and other parts; and two or more coils of intestine may be thus involved, and fistulous communications established between them. The colloid form is the most liable to invade the peritoneum, where large gelatinous masses may be developed.

The new-formed tissue may constitute an irregular mass of very variable size and extent, of a nodulated or of a villous appearance, perhaps partially ulcerated, and extending into the passage of the canal, producing an obstruction; or it may develop in an annular manner, involving the whole circumference of the bowel. The obstruction produced in the latter case may be extreme, even to narrowing the lumen of the tube to barely the size of a probe. The intestine above the growth may become enormously dilated while below it is contracted. The extent of obstruction may be altered by partial destruction of the new-growth by sloughing, though the subsequent cicatrices that may result will again constrict the gut.

SYMPTOMS.—For a varying time before this disease definitely asserts itself, the patient may complain of vague dyspeptic symptoms; loss of appetite; a sense of uneasiness in the abdomen, not amounting to pain, and usually increased after meals; and marked irregularity in the action of the bowels, with or without flatulent distension. There is an increasing tendency to constipation, for which the patient often takes large quantities of aperients of a drastic and cathartic character, which one by one lose their effect. The persistence and gradual increase of these symptoms, especially if there be any loss of flesh, is very significant, and should excite suspicion. Sooner or later, according to the duration of the case, the usual cachexia is established; and in the greater number of cases the patient rapidly emaciates, especially towards the end, though in cases of very short duration the wasting may not be so excessive. The emaciation depends not only on the general perversion of nutrition caused by the development of the cancer, but also on the direct influence it exerts on organs concerned in the digestion and absorption of nutriment; hence the higher up in the bowel the growth is situated the sooner is the loss of flesh determined.

The local signs and symptoms referable to the

new-growth itself are very variable in their occurrence, and often are singularly slight in comparison with the gravity of the cause. Thus pain may be completely wanting, and perhaps there is but little tenderness on pressure; when present the pain is usually of a dull character, and quite localised; although it may attain a severe degree.

The indications of the tumour produced by the new-growth are very uncertain, being often little more than an ill-defined fullness in one region, scarcely even amounting to that in the annular form of cancerous stricture; at other times presenting a distinct hard irregular mass of variable size. This last quality, being partly dependent on fæces, is very characteristic. Should the growth happen to be situated over the aorta or iliac arteries, an indistinct pulsation may be communicated to it. The percussion-note over the tumour is usually imperfectly tympanitic, from the existence of coils of intestines between it and the abdominal wall, the thickness of which will of necessity considerably modify the signs of the existence of the growth. The mass may present all degrees from free mobility to complete fixity, dependent on the nature of its seat, and also on the existence of adhesions to neighbouring parts. By pressure on veins or on nerves, œdema and pain of the lower extremities may be caused.

Symptoms of intestinal obstruction are rarely wanting, and may be the first for which the patient seeks relief. Vomiting, constipation of increasing severity, with signs of intestinal distension above the lesion, are among the most constant. The character of the vomit will largely depend on the duration and situation of the lesion, and the actual degree of obstruction. When the jejunum or lower part of the duodenum is the seat of the growth, the vomiting is more copious and constantly bile-stained, the bowel and stomach above the stricture sharing in the distension. When the obstruction is lower down, in the large intestine, the vomiting may be slight, and consist of little more than the gastric secretions, with such food as is taken, only becoming stercoraceous when the obstruction becomes complete. Occasional diarrhoea, determined by the chronic enteritis which exists, may alternate with the constipation; and rupture of the intestines, especially the cæcum, has been met with as a result of the atrophy and ulceration of the over-distended, chronically inflamed bowel. A moderate degree of pyrexia is not infrequent, due probably to septic absorption from the ulcerated growth, or to an associated enteritis. As in other forms of intestinal obstruction, the amount of urine voided is likely to be very much diminished when the stricture is high up, and the vomiting is excessive. *See* **INTESTINAL OBSTRUCTION.**

The stools may be characteristic of the obstruction, consisting of small separate masses, frequently hard and round, or flattened or in thin cylinders ('pipe-stem') if the growth be near the anus; they may be mixed with sloughed-off portions of the new-growth, or with blood that has escaped from the ulcerated surface. *See* **INTESTINE, HÆMORRHAGE FROM.**

If the peritoneum be involved, peritonitis may arise; and ascites, often considerable, is usually developed with colloid cancer. Superadded to these symptoms will be those caused by the morbid condition of any other organ that may be affected, such as the liver, bladder, or uterus.

COURSE AND TERMINATIONS.—Malignant disease of the intestines in the majority of cases progresses continuously. It is difficult to state even an average duration, owing to the insidious onset and vagueness of the first symptoms; but the greater number of cases do not go beyond twelve to eighteen months from the time when the disease is clearly established, while some may be fatal in a few weeks, and a few may last for several years.

Death may result as the direct consequence of the cachexia; or from complete intestinal obstruction, hæmorrhage, peritonitis, or other effects of the growth.

DIAGNOSIS.—An exact diagnosis is often not to be made, and the nature of the case remains throughout uncertain, if not actually as to the existence of a malignant growth, at least as to the seat of it. The insidious and ill-defined character of the earliest symptoms presents nothing diagnostic, though their progressive character and resistance to treatment would cause a suspicion, especially in a person over middle age, and in whom a gradual even though slight loss of weight is noticed. Even in the later stages, the symptoms are almost identical with those of chronic enteritis, which co-exists with the new-growth to a greater or less extent; and in the not infrequent cases in which a tumour is not to be felt, or is uncertain in its indications, the diagnosis becomes extremely difficult.

Supposing that the existence of an abdominal tumour be clearly ascertained, it is not always easy to determine its connection with the intestine, since the variability in position, in mobility, and in size (due to the accumulation, or the reverse, of fæces) precludes any diagnostic sign, although this very variability is regarded by some as almost indicative of intestinal cancer. In distinguishing between an intestinal tumour and one connected with the liver, gall-bladder, pancreas, kidney, mesenteric glands, uterus, abdominal wall, or the inflammatory thickening following a perityphlitis, an aneurysm, or a simple fecal accumulation, the history of the case, age, progressive nature of the condition, existence of tumour, signs of obstruction, and character of the stools, are the points to be considered in forming a diagnosis. Any one or even two of these points might equally indicate other lesions, but, taken collectively, they will usually justify the formation of an opinion. A rectal cancer, which is accessible to the touch or even to inspection, need offer no difficulty, but it is otherwise where it is a question of distinguishing a duodenal growth from one strictly limited to the pylorus. The vomiting in the latter case is more persistent than in the former, and there is a greater liability to hæmatemesis; but these are most uncertain signs, as also is the existence of jaundice, which often complicates the duodenal affection, from the greater chance of its involving or pressing on the bile-duct. A firm epigastric tumour, felt close to the margin of the thorax, and associated with distinct dyspeptic symptoms, may also be due to primary cancer of the head of the pancreas; in such case the symptoms of obstruction are less marked at first, but the growth will probably involve the bowel in its progress, and cause stenosis; owing to the destruction of the gland-tissue, the pancreatic juice is not secreted, and undigested fat may be found in the stools. Similar difficulties may surround the investigation of a tumour situated in the right iliac fossa. The importance of completely emptying the bowels by enemata, before finally

forming a diagnosis, cannot be too emphatically insisted on, since the signs, and in many respects the symptoms, of intestinal cancer may be simulated by a fecal accumulation due to simple atony of the colon. The anæsthetisation of the patient is also frequently necessary before the exact situation, or indeed the existence, of a growth can be determined.

TREATMENT.—The extremely rare cases of reputed natural cure of malignant disease of the intestines, brought about by sloughing of the growth and subsequent cicatrization of its site, afford no hope of our being able to imitate the process artificially, and the medical treatment remains at the best symptomatic and palliative.

The diet should be so arranged as to contain the minimum of indigestible residue, and permit the chief digestion and absorption to take place in the stomach, if it be the upper part of the tube that is affected. But in the majority of such cases the utmost disinclination for food exists, even apart from any vomiting or pain which its ingestion may produce, and hence, whatever the directions, the patient in the later stages practically takes nothing. The anorexia is frequently as marked even when the mischief is seated in the colon, and the area for digestion and absorption is uninterfered with.

In the earlier stages it may be advisable to insist upon as much nutritious food as possible being taken, either by the mouth or in the form of enema or suppository, so as to offer the most prolonged resistance to the inevitable end; but at the same time there is no slight reason for thinking that the same course favours the development of the new-growth. Preparations of iron may be given.

The symptoms dependent upon the obstruction we can do very little to relieve short of operation. Only the mildest aperients are permissible, to combat the constipation, and these should be salines with the object of rendering the fæces as fluid as possible; while the vomiting is as a rule uncontrollable, and, indeed, is often a relief.

Hæmorrhage may require special attention, the tincture of hamamelis being one of the most effective styptics. The pain may be so severe as to necessitate free administration of morphine subcutaneously; in other cases belladonna is of value in alleviating the local discomfort, and acts favourably by allaying any spasm.

The operative treatment of malignant disease of the intestine is described in the article **INTESTINAL OBSTRUCTION**.

(b) *Non-malignant growths.*—Owing to the variety of tissues that enter into the formation of the intestine, no less than to the origin of these tissues from two of the three primary layers of the blastoderm, the new-growths that may develop in connection with it are exceedingly numerous.

VARIETIES.—1. *Fibromata.*—These growths, which are developed from the connective tissue of the submucous coat, are usually of small size, frequently pedunculated, though sometimes appearing as sessile flattened nodules, of half the size of a pea, projecting into the canal. The smaller ones may be scattered throughout the length of the bowel, while the larger ones (up to the size of a walnut) are fewer in number or single, and are usually found in the rectum. They present the ordinary microscopical characters of fibrous tissue.

2. *Lipomata.*—Pedunculated growths of adipose tissue, springing from the submucous coat, are not

of uncommon occurrence in any part of the intestines.

3. *Myomata*.—Very rarely small growths are met with, chiefly composed of non-striated muscle-cells, with a variable amount of connective tissue.

4. *Vascular tumours*.—*Angiomata*.—Vascular growths are sometimes found, of an erectile character, similar to nævi of the skin.

5. *Mucous growths*.—It is to these growths that the term 'polypi' is generally applied. They essentially consist of the tissues of the mucous membrane, though differing in their vascularity, and also in their glandular elements. When these latter are excessive in amount, they are liable to present characters which connect them with malignant forms of new-growth, especially if their surface assume a villous formation. Polypi are not limited to any one part of the canal, though undoubtedly they are most common in the rectum. They are occasionally multiple, 170 have been seen in the lower half of the colon in a single case; and they have been met with at all ages.

6. *Lymphoid growths*.—*Lymphadenomata*.—Neoplasms corresponding in structure with the solitary or agminated glands, or with the lymphoid layer of the submucosa, are met with in association with these normal constituents of the intestinal wall, and quite independently of leucocythæmia. Nodular formations and diffuse infiltrations of similar structure have been met with throughout the stomach and intestines in Hodgkins's disease (lymphadenoma), but though giving rise to large masses, they produce no obstruction.

7. *Sarcomata*.—Small nodules of this type of neoplasm have been described as occurring in very rare instances in the intestine.

8. *Cysts*.—These have been rarely met with, and the contrast to their comparative frequency in the uterine mucous membrane is remarkable. Several instances are on record of congenital dermoid cysts which have been found attached to the rectum or sigmoid flexure, probably originating from the neuroenteric canal.

Cystic enlargement of the vermiform appendix, or of diverticula of the intestine, may follow on closure of their communication with the lumen of the bowels, and accumulation of fluid in their interior—*entero-cystomata*.

EFFECTS AND SYMPTOMS.—As a rule, the growths mentioned above present very little interest, unless they be situated just within the rectum and accessible to digital examination, for it is seldom that they are of sufficient size to form tumours which can be felt in the abdomen. They cannot be diagnosed, though they may give rise to certain symptoms, as, for instance, hæmorrhage from the vascular polypi and erectile tumours, or partial obstruction if they attain any size; but such symptoms are not diagnostic. One of their most striking effects appears to be the liability that the polypoid forms occurring in the small intestine have of inducing serious intussusception, from interfering with the due progress of peristalsis. Prolapse of the rectum is similarly found to be occasionally due to polypi.

TREATMENT.—No treatment can be attempted, beyond that of the symptoms which may arise; or the removal of growths within reach of the anus, or very exceptionally by laparotomy.

20. *Intestines, Paralysis of*.—See Neuroses of, p. 796.

21. *Intestines, Parasites of*.—The intestinal canal from within a very few hours after birth, to judge from the fæces, swarms with numerous forms of microbes, which, having gained entrance with the food and air, find in the canal suitable conditions for their development; although many, including numerous pathogenic forms, are destroyed or rendered inert by the acid of the gastric juice. Our present knowledge would lead to the belief that many of the species have no effect upon the human body, while others take an active share in intestinal digestion, as effective though apparently superfluous aids to the pancreatic secretion. How far any of these saprophytes, which may be regarded as normal inhabitants of the canal, may under certain conditions of the contents or excessive development, &c., become responsible for symptoms of disease (pathogenic), is not known with certainty, though it appears most probable such is the case. But it is well recognised that certain specific diseases, such as tuberculosis, cholera, typhoid fever, &c., are connected with specific organisms which, gaining admission with the ingesta to the alimentary canal, produce the characteristic symptoms of these several diseases.

Besides these obviously pathogenic organisms, there are numerous others which are more or less constantly found; of these *Bacillus coli communis* is by far the commonest (see p. 121). Most of these are anaerobic saprophytes, but their liability to become pathogenic in certain conditions of their environment has been mentioned. Among other more commonly met with forms are *Bacillus lactis aërogenes*, *Briege's bacillus*, *Bienstock's bacillus*, *Bacillus pyocyaneus*, *Staphylococcus pyogenes*, *Streptococci*, and various *Vibrios* and *Spirilla*. Some of these are frequently met with in the evacuations of patients suffering from different forms of enteritis; and it is probable that the poisonous substances which these organisms form from the intestinal contents, especially the albuminous, take a large share in the causation of the disease, or at least that many of the symptoms are to be referred to the ptomaines which are absorbed. But our present knowledge does not enable us to associate with each of the multiform diarrhoeal conditions which occur its own specific causal organism, as we can do in the case of cholera or typhoid fever.

Actinomycosis has been met with in the intestine, where it forms raised whitish patches, setting up inflammation, and proceeding to abscess, which may burst into the peritoneum or present at the integuments. As the disease spreads, some healing may take place. See p. 22.

The *Bacillus anthracis* also occurs in the intestine, causing swollen oedematous patches of the mucous membrane, which are intensely congested and rapidly slough. The intestinal lesion may be primary, or it may be part of a general infection. See PUSTULE, MALIGNANT.

A few species of the yeast-fungi or saccharomycetes, such as *torule*, and of the hyphomycetes or moulds, such as the *Oidium albicans* or 'thrush,' occasionally develop in the intestines. See STOOLS.

Various species of animal parasites, which mostly obtain their nourishment from the contents of the canal, are met with in the intestines. The majority of these organisms, with the symptoms they give rise to and their treatment, are described under ENTOMOZOA.

22. Intestines, Perforation and Rupture of.—**ÆTIOLOGY.**—The causes of perforation or rupture of the intestines may be arranged thus : (1) *External injuries*, such as blows, being run over, &c., which, though more liable to rupture the solid abdominal viscera, frequently cause laceration of the intestines, especially the ileum or jejunum.

(2) *Corrosive poisons*, when swallowed in any considerable amount, may destroy not only the walls of the stomach, but also those of the upper part of the intestines.

(3) *Extreme distension by flatus or feces* above the site of a constriction may cause the bowel to burst. It is the cæcum which usually gives way in chronic obstruction in the colon or rectum.

(4) *Ulcerations*, pre-eminently the so-called peptic ulceration, and less commonly typhoid, tubercular, and catarrhal ulceration, may lead to intestinal perforation. See APPENDICITIS.

(5) *Perforations* may be produced *ab extra*, by the bursting of abscesses or aneurysms into the canal.

SYMPTOMS.—The most striking symptoms which perforations of the bowel present are a sudden attack of severe abdominal pain, followed by vomiting and collapse; and it is a noticeable fact that rupture of the hollow abdominal viscera is more liable to induce this condition than a similar lesion of such organs as the liver or spleen. Rigors are of occasional occurrence at the onset, and the pulse is quickened, with sometimes a rise in temperature.

Should the patient live twenty-four hours after the establishment of a perforation, signs of peritonitis will assert themselves—severe abdominal pain and tenderness, pyrexia, vomiting, and other symptoms. Supposing that the perforation follow an ulceration in the course of a previously high temperature, such as enteric fever, there is sometimes, though not always, a sudden and considerable fall in the body-heat; this, with the acute pain, may be the first indication that perforation has taken place.

Perforation or rupture of the intestines usually proves fatal within forty-eight hours of its occurrence, although cases are recorded which have lasted for weeks; very rarely recovery has taken place. Occasionally a localised abscess may result.

There are no reliable signs whereby rupture of the stomach may be distinguished from that of the intestines.

TREATMENT.—Rest is of primary importance, in regard both to the whole body and the bowels themselves. This object is best attained by the free use of opium, commencing with a grain, and repeating it in a few hours until its influence is fully established. It is also desirable to stop all food, except an occasional teaspoonful of meat-essence; to give ice to suck; to administer nutrient enemata, and brandy and ether subcutaneously if the collapse be profound; and to apply warmth to the extremities. Resection of the ruptured portion of the bowel, with suturing of the divided ends, and washing out of the peritoneum, has been performed with success, and should be attempted in all cases seen soon after perforation has taken place.

23. Intestines, Spasm of.—See p. 797.

24. Intestines, Syphilitic Disease of.—The intestinal canal is rarely the seat of the specific lesions of syphilis, except at the lower end of the rectum, and margin of the anus. Small gummata

have been found in the submucous tissue of various parts of the bowel, and sometimes the ulcers to which these growths give rise by their degeneration and breaking down; radiating fibrous thickenings and cicatrices of the mucous membrane have also been seen in syphilitic subjects, sufficient even to cause obstruction. It is doubtful whether there be any specific ulceration of the intestine which is not preceded by gummata, although small ulcers do occur in new-born children, the subjects of congenital syphilis.

Syphilitic ulceration and stricture of the rectum are not of infrequent occurrence, the former especially in women. See RECTUM, Diseases of.

25. Intestines, Tubercular Disease of.—

ÆTIOLOGY.—As a primary disease, tuberculosis very rarely attacks the intestines in adults, though it is of very frequent occurrence in children as part of a general tuberculosis, or possibly from the consumption of milk from tuberculous cows. In adults, on the contrary, tubercular disease of the intestines is very commonly developed secondary to a similar affection of the lungs, due probably to the swallowing of the sputum.

ANATOMICAL CHARACTERS.—The submucous layer and the peritoneal coat are the structures in which the tubercle originates; in the former situation it especially favours the ileum and cæcum, although it may develop throughout the entire length of the tube, while the peritoneal tubercle is about equally distributed. The mesenteric glands are always considerably involved. The rarity with which the stomach is affected by tubercle is in marked contrast to the frequency of the intestinal lesion.

In cases of acute tuberculosis in children, death may take place before any changes in the tubercular formations have taken place, and countless grey granulations, from the size of a pin's head to bodies quite microscopic, are to be found in the submucosa, and in the solitary and agminated glands. Later on, however, these non-vascular new-growths coalesce, and form distinct masses, which from lack of nutrition undergo caseous degeneration and break down, thus forming the tubercular ulcers. The ulcers tend to spread, and rarely to heal, and while they may be at first limited to the glands, they invade the adjacent mucous membrane, spreading especially in a direction round the bowel, their extension being preceded by the development of fresh tubercles, chiefly along the course of the main blood-vessels. Large masses of the mucous surface may be thus destroyed, leaving a ragged, flocculent surface, formed of the muscular fibres, or even of deeper structures, which lesions rarely proceed to perforation into the peritoneal cavity, adhesive peritonitis having established attachments to adjacent parts. The thickened, congested, irregular edges of the ulcers, with miliary tubercles close to the margin and on the corresponding peritoneal surface, are very distinctive.

SYMPTOMS.—Until ulceration be established, there will be no symptoms of tubercular disease referable to the intestinal canal; and even when this stage is reached, there are no special symptoms.

TREATMENT.—Little can be done for intestinal tuberculosis. The course of the disease is almost invariably to a fatal end, and it is very rare for healing and cicatrization to take place. The neces-

sity for feeding the patient is almost contra-indicated by the existence of a destroyed digesting and absorbing surface, whereby the food becomes a positive irritant. Such nourishment as is taken should therefore be in the most digestible and concentrated form, that as much as possible may be taken up from the stomach and upper part of the intestine. Starch and opium enemata may do a little to check the diarrhoea, but their efficacy is soon lost. Hæmorrhage, should it set in, is scarcely amenable to treatment, though astringent enemata may be of some use, combined with the internal administration of acetate of lead and opium. No treatment has as yet been effectual in arresting the spread of intestinal tuberculosis.

26. Intestines, Ulceration of.—Ulceration of the intestinal wall, from one cause or another, is of extremely common occurrence. The morbid processes involved in the production of the ulcers are in all cases essentially the same, namely, a molecular death and disintegration of the tissue, leaving a solution in continuity, of varying extent and depth.

VARIETIES: (1) Primary inflammatory ulcers.—Any enteritis, whether of the mildest catarrhal character, or of a specific type such as diphtheritic, dysenteric, or typhoid, may lead to ulceration of the bowels. As a rule, the more severe the cause of the inflammation, the greater the liability to this complication; and the same holds in respect of any intestinal catarrh, developed in the course of any serious state, such as typhus fever or Bright's disease. The ulcer may appear either as a small abrasion of the epithelial layer, which gradually extends and deepens until the whole mucosa is involved; or the first indication may be a thin glairy pellicle, adherent to the mucous membrane which in time is thrown off, leaving a breach in the subjacent tissue. In other cases the destructive process commences in the thickness of the bowel, either from the rupture of small collections of inflammatory products, resulting from an enteritis, or from inflammation of the lymphoid follicles. The escape of these products into the tube leaves behind an ulcer.

These lesions may be found anywhere throughout the bowels, although they are perhaps more frequent in the large than in the small intestine; and one form of follicular ulceration, associated with the specific poison of dysentery, is almost limited to the former situation. The colon is also the seat of a very extensive simple ulcerative colitis, in which the mucosa is destroyed over large areas, but which does not commence in the follicles (*see COLITIS*). At those places where any delay is likely to arise in the passage of the fæces—the cæcum, sigmoid flexure, and rectum—and at those spots which are most prominent, such as the edges of the valvulæ conniventes, and the sacculi of the colon, where an enteritis is most likely to be produced, there will be the probable site of inflammatory ulcers. The specific ulcers of dysentery and typhoid fever are described elsewhere. In cholera, ulceration of the lower end of the ileum and colon is sometimes met with.

(2) Ulcers resulting from the separation of necrosed tissue.—The process of molecular disintegration which takes place along the line between living and dead tissue, resulting in the separation of a slough and the leaving of an ulcer, takes place in the intestines as elsewhere. The causes leading to the death of circumscribed areas of tissue are various.

Sometimes the vitality of a portion of the mucous membrane is destroyed by degeneration, such as the lardaceous, and an ulcer marks the spot of the removed patch, which usually occurs in the ileum or colon. More frequently, as in Bright's disease, obstructions of the portal system, or plugging of the mesenteric arteries, the local death is induced by capillary hæmorrhage or a cessation of blood-flow through a limited area. Under such circumstances the solvent power of the digestive juices may be exerted on the non-living tissues, which are thus removed, and an ulcer is left. To such ulcers the term *peptic* has been applied, and identical lesions are met with in the stomach. They almost invariably occur in the first part of the duodenum, above the point of entrance of the alkaline bile and pancreatic juice, although very rarely they have been seen in the jejunum. Ulcers of this character appear to be occasionally connected with large superficial burns, but how the relationship is established is not known. It is a singular fact in regard to them, that they are three times more common in men than in women, which is quite the reverse of what obtains in the stomach, although the relative frequency of gastric and duodenal ulcers is estimated as thirty to one.

(3) Ulceration of new-growths.—Almost any neoplasm of the intestinal wall may ulcerate, though as a rule the more rapidly developed forms are the more liable.

CHARACTERS.—The appearances presented by the various ulcers differ with the cause and the duration.

They may be single, as is generally the case with the duodenal ulcers; or innumerable, as the follicular ulcers of the colon. Typhoid and tubercular ulcers are as a rule multiple, and are most numerous at the lower end of the ileum, where the agminated glands are most abundant. Occasionally large surfaces of the mucous membrane are destroyed, with here and there small isolated spots of the membrane left, due to the spread and coalescence of many separately arising ulcers. In dysentery and chronic tubercular ulceration this is especially liable to happen. Many of the catarrhal and follicular ulcers are extremely small, not more than a line in diameter.

The peptic ulcers are distinguished by their very definite, 'clean-punched' appearance; the edges are slightly sloping, and but very little, if at all, thickened; while the mucous membrane immediately adjacent has a perfectly healthy appearance. In most of the other varieties the edges are thickened, irregular, and shaggy, frequently excavated and undermined, the ulcerative process extending beneath the mucous membrane, which gradually dies and sloughs away as its nutrition is cut off. Dependent upon the depth and course of the ulcer will be the nature of its base, which may be formed of the muscular coat, of the peritoneum much thickened, or of adjacent structures with which adhesion has been established, such as the liver or abdominal wall. The floor of ulcerated tubercular deposits and malignant new-growths usually presents small nodules which are being developed coincidently with the ulceration. The buff or ash-grey pigmented sloughs, partially separated, give a characteristic appearance to the old-standing ulcers of dysentery and tuberculosis. The tubercular and typhoid ulcers of Peyer's patches present a certain difference in the direction in which they extend: though at first both are limited to the patch, the former tend to spread in an annular manner, whereas the latter have usually

their long axis corresponding to the length of the bowel. This difference depends rather on the duration of the ulcer than on any specific distinction due to the two diseases; for the more acute typhoid lesion rarely spreads much beyond the area of the patch, which is in the long axis of the bowel, while the chronic tubercular ulcer follows the distribution of the lymphoid tissue outside the patches, and particularly along the course of the blood-vessels and lymphatics.

COURSE.—The course of an intestinal ulcer may be acute or chronic, lasting a few days or for years. Some of the simple ulcers of an acute intestinal catarrh belong to the former group; while the ulceration that accompanies chronic enteritis may be of indefinite duration.

The acute forms may either heal or go on to perforation; in the former case their existence can only be inferred, and catarrhal and follicular ulcers belong to this category. The peptic and typhoid ulcers are those most liable to perforate the bowel. The peptic duodenal ulcers appear to be more prone to perforate than the corresponding lesion in the stomach; but the results of perforation are said to be more likely to be localised in the former case. It is also stated that the ulcer of the duodenum following a burn less often perforates than cicatrises, and thus may lead to obstruction. Occasionally the perforation may lead to a communication between one coil of intestine and another, without any rupture into the peritoneal cavity. In chronic ulcers, where no adhesion or communication takes place, the base is thickened by a new-formed connective tissue, which is developed as fast as, or even faster than, the destructive process proceeds, and hence the intestinal wall adjacent to and involved in such ulcers is usually much thickened and indurated. Short of actual perforation or adhesion to other parts, the site of the ulceration most frequently is marked on the external surface of the bowel by a subacute peritonitis, which may produce a partial matting together of the intestines.

In the course of the healing of the larger ulcers, by the formation of a contracting cicatricial tissue, the gut may be considerably constricted, and a formidable obstruction established. But this does not necessarily follow even large ulcers, for stricture of the intestine does not follow the ulceration in typhoid fever; and the depth of the destruction would seem to influence this result. When the superficial portion of the mucous membrane only is destroyed very little contraction follows, but when the deeper parts of the wall are involved the subsequently developed cicatrix tends to shrink considerably.

SYMPTOMS.—The greatest diversity is met with in the symptoms of intestinal ulceration, and few, if any, can be regarded as characteristic. The lesion may occur without producing any symptoms; or those that do exist may be determined by the course of, or by the conditions associated with, the ulceration; or, lastly, the results of this condition, such as perforation, may entirely obscure the actual ulceration itself: it frequently happens therefore that the existence of an ulcer is not recognised. Nor may the severity of the symptoms be taken as a measure of the extent of the ulceration, for the most marked pain, tenderness, diarrhoea, and other symptoms may be produced by an area of typhoid ulceration that heals; while a perforating duodenal ulcer may give scarcely any indication of its exist-

ence, until within a few hours of a fatal ending. This course appears to be very characteristic of duodenal ulcers. The writer has recorded a case of a young man who was suddenly attacked with symptoms exactly resembling those of intestinal colic, after constipation of a week's duration; there was no vomiting and no tenderness, the pain being relieved by pressure on the abdomen. Collapse set in, and death resulted in less than twenty-four hours from the commencement of the attack. The necropsy showed a perforating duodenal ulcer. Such a case is not singular, and the fatal event may be preceded, as in this case, by nothing beyond an occasional feeling of discomfort at the epigastrium, not serious enough to call for advice or treatment.

Such symptoms as diarrhoea, vomiting, pain, tenderness, and pyrexia are as much dependent on a co-existent enteritis or new-growth, as they are upon the ulcer. Doubtless the exposed surface of an ulcer offers the opportunity for irritation resulting in increased peristalsis, but this is not of necessity, for constipation may be present. Where the area of ulceration is extensive, the absorbing surface is by so much diminished, and thus while the general nutrition suffers, the unabsorbed products of digestion are liable to decompose and induce diarrhoea. Vomiting may arise from a duodenal ulcer, but not always, and when present it may be due to peritonitis; icterus may also complicate an ulcer in this situation, by involving the opening of the bile-duct, or by extension of the duodenal catarrh. Pain may be absent or quite insignificant, unless the rectum be the seat of the disease, when the pain and tenesmus are excruciating.

The passage of blood in the stools, especially if bright, is probably an indication of ulceration low down, but it does not always occur, and it may be due to hæmorrhoids or general venous congestion from portal obstruction. Mucus is more commonly present, as discrete masses of the size of a hemp-seed or in larger quantities. *See* STOOLES; and MELENA.

DIAGNOSIS.—From what has been said, the formation of a diagnosis of intestinal ulceration is frequently impossible, and an ulcer is assumed rather than proved to exist. In enteric fever, ulceration is taken for granted as existing, though no special symptoms may indicate its presence. But if a severe and persistent diarrhoea, with liquid evacuations containing perhaps blood and shreds of mucus, and much pain and tenderness over the abdomen, supervene in a case of pulmonary tuberculosis, it is a fair inference to assume ulceration of the intestines, a diagnosis that would be confirmed by the discovery of the specific bacillus in the stools. In dysentery, as in enteric fever, the ulceration is a specific part of the disease, and the diarrhoea, pain, and characteristic stools are in this case directly dependent upon the ulceration. In distinguishing between gastric and duodenal ulcer the deferred occurrence of pain after taking food, even to three or four hours and being unrelieved by vomiting, is characteristic of the latter lesion, as well as the site of the pain being rather in the right hypochondrium, and less marked at the xiphoid cartilage and in the dorsal region, which are the frequent situations of the pain of gastric ulcer; the digestion is also much less disturbed in the duodenal affection, and hæmatemesis is much less common, being replaced by melena. The greater frequency among males has been mentioned. It is not infre-

quently difficult to distinguish this condition from gall-stone colic, the situation and character of the pain in both cases being very similar, but the duration of the pain in the latter is usually shorter and more paroxysmal, and there are usually signs of enlarged liver, distended gall-bladder, or jaundice.

PROGNOSIS.—This largely depends upon the cause. Except through perforation or fatal hæmorrhage by erosion of vessels, death does not take place from the ulceration itself. But a tubercular ulcer is not to be expected to heal, and it may by its development hasten the end of a phthisical patient. The prognosis of typhoid ulceration will almost entirely be founded on the general state of the patient, since the indications of the ulceration itself may be so slight. The ulceration of malignant new-growths may be of actual benefit, by removing portions of the mass, and so diminishing obstruction. In all cases the liability to subsequent stenosis must be remembered; and the impaired health of body and mind in chronic ulceration may continue throughout life.

TREATMENT.—Since ulcers of the intestine are inaccessible to direct treatment, little can be done for them apart from the general conditions which they may complicate, or the treatment of the symptoms to which they may give rise.

Recognising that an ulcer, when it exists, may lead to perforation, the object will be to avoid all undue movements of the intestines, and hence aperients are forbidden, and opiates are indicated. The astringents that are likely to be used for the diarrhoea or hæmorrhage may exert a local action on the lesion, and for that purpose bismuth, chalk, and the vegetable astringents, ergot or turpentine, are recommended. Intestinal antiseptics, such as salol, are often of benefit by diminishing the putrefactive decompositions which are likely to exist. Rest is probably the only element of treatment that can affect the ulcerative process directly; while any improvement of the general condition will necessarily favour the healing—objects which can be best accomplished by the use of diet of the most bland description, or of nutrient enemata.

27. Intestines, Vascular changes in.—The conditions herein included are known as *active hyperæmia* when the excess of blood is primarily at least on the arterial side of the capillaries; and *passive congestion* where the fulness is caused by some obstruction to the venous flow.

It should be remembered that even within the limits of health a considerable variation is met with in the degree of vascularity of the alimentary canal. The fluctuating periods of activity and rest undergone by the tube are associated of necessity with alternations of comparative hyperæmia and anæmia, as during the digestion of a meal or during fasting. It is impossible, therefore, to draw any line beyond which the vascular fulness can be said to be abnormal; as it is equally impossible to say exactly where hyperæmia and normal gland-change end, and catarrh begins. These states, on the border line between health and disease, stop short of producing recognisable tissue-change, and are of transient duration.

ANATOMICAL CHARACTERS.—The appearances seen *post mortem* are far from being always indicative of what existed during life. For an extreme arterial fulness may completely disappear after death, from contraction of the vessels; while venous

engorgement more or less completely remains. Submucous petechiæ and hæmorrhagic erosions are commonly to be met with in this condition.

ÆTIOLOGY.—The causes of intestinal *hyperæmia* are as follows: (a) Mechanical and chemical irritants, foreign bodies, and poisonous drugs. Spices and highly seasoned food and alcohol may bring about an abnormal degree of hyperæmia of the whole or part of the canal. These causes act locally and directly upon the vessels and are scarcely distinguished from the mildest degree of inflammation.

(b) Vaso-motor paralysis of the splanchnic area. If from any cause the normal tone of the mesenteric vessels is diminished, by inhibition or removal of the tonic influence excited by the sympathetic, the vessels dilate and hyperæmia ensues. It is in this way that diarrhoea following certain emotional states may be explained. The intimate relation which has been shown experimentally to exist between the splanchnic nerves and the vaso-motor system generally, but especially with the cardiac innervation by means of the 'depressor nerve,' whereby any considerable peripheral resistance in the systemic capillary area which impedes the action of the heart is compensated for by a dilatation of the mesenteric vessels, renders it probable that an undue hyperæmia of the intestines is of very frequent occurrence.

(γ) Among less frequent causes of intestinal plethora are, the suppression of habitual discharges—menstrual, &c.; and the removal of pressure, as of ascitic fluid.

The causes of *passive congestion* are the following:

(a) A general congestion of the entire intestinal tract will be produced by any of those causes which lead to universal congestion of the tissues, as obstructive heart-disease. Pressure by tumours or other conditions on the inferior vena cava above the liver, or on the portal vein, will bring about the same result. So also will any obstruction to the portal circulation in the liver. This is by far the commonest cause of intestinal congestion, since cirrhosis of the liver, however produced, directly tends to it.

(b) A congestion of a portion of the tube occurs when any obstruction exists to the venous flow of that part, as is marked in cases of invagination and strangulation of the bowel.

The rarer conditions of embolism of branches of the mesenteric arteries, or thrombosis of the veins, will induce intense congestion of the region supplied by the occluded vessels. The resulting infarction is somewhat smaller than the actual extent fed by the vessels, owing to the free anastomosis at the periphery of the area. Sloughing and ulceration of the mucous membrane, or extensive gangrene of all the coats, is very liable to follow on this condition.

Both these conditions of engorgement are essential features of the vascular changes comprised in inflammation.

SYMPTOMS AND EFFECTS.—These conditions may of themselves give no evidence of their existence, although an excessive and sudden hyperæmia of the splanchnic area will cause a fall in the general blood-pressure with symptoms of fainting. Overfulness, just as anæmia, of the intestinal vessels leads to increased peristalsis, probably due to the deficiency of oxygen and excess of carbonic acid which attend each of these conditions.

From hyperæmia, there is an increased secretion

of mucus and other intestinal fluids, often more watery than normal, which with an increased peristalsis, induced by the same irritant that led to hyperæmia, produces a diarrhoea. Provided this functional over-activity be limited to the production of the normal secretions of the part, and increased healthy action only, the condition of hyperæmia is not exceeded; but the passage into catarrh is easy, and unmarked by any abrupt lines.

Profuse bleeding, often accompanied by severe abdominal pain, usually follows plugging of the mesenteric vessels. The more complete the obstruction to the flow, the greater will be the pressure in the veins, and so will be produced first an œdema of the mucous membrane and entire thickness of the bowel, and later an escape of fluid into the canal itself and into the peritoneal cavity, the latter being more marked.

TREATMENT.—It is seldom that these conditions are such as call for treatment. The hyperæmia is usually of a transient nature; and the cause of congestion is generally irremovable. Aperients, such as jalap and gamboge, are sometimes beneficial.

The treatment of hæmorrhage has been considered; but this and diarrhoea, when due to congestion, are, unless excessive, often beneficial, and should not be checked. W. H. ALLCHIN.

INTRA-THORACIC TUMOURS.—See **MEDIASTINUM**, Diseases of; and **THORACIC ANEURYSM**.

INTUSSUSCEPTION (*intus*, within; and *suscipio*, I receive).—A form of intestinal obstruction, in which one portion of the bowel passes into another portion. See **INTESTINAL OBSTRUCTION**.

INUCTION (*in*, on; and *unguo*, I anoint).—**SYNON.**: Anointing.—This is a method of inducing absorption of certain substances by placing them in contact with, or rubbing them into, the skin. The local application of liniments to cause counter-irritation, to relieve pain, or for any other purpose, is sometimes included in the connotation of this term.

Mercury is often administered by this method both for syphilis and for tubercular peritonitis, especially in children. Half a drachm of mercurial ointment should be smeared upon the front of the abdomen, and the part covered with a flannel binder. Every second night the child should be washed and the application repeated. In the case of adults inunction is but rarely employed in England. H. MONTAGUE MURRAY.

INVAGINATION (*in*, in; and *vagina*, a sheath). A synonym for intussusception. See **INTUSSUSCEPTION**; and **INTESTINAL OBSTRUCTION**.

IODISM.—**DEFINITION.**—Iodism is the term within which we include a variety of painful and inconvenient effects, following, under certain circumstances, the administration of iodine and its salts, but more especially the iodide of potassium.

The effects produced by iodide of potassium may be classified as follows:—

1. *On the nervous system.*—Mental depression and diminution of muscular energy are not infrequently noted in patients taking iodide of potassium; while neuralgia, tinnitus aurium, and convulsive movements have also been described. It is possible that the potassium is here the active agent.

2. *On mucous membranes.*—Much mucous irritation is occasionally observed; conjunctivitis, lachrymation, sneezing and running from the nose, frontal headache, and puffy swelling of the eyelids, closely simulating coryza, being the most common symptoms of iodism, and sometimes following a single small or moderate dose. Pharyngeal congestion, irritable redness of gums and tongue, and œdema of the glottis have also been described. These symptoms are doubtless due to the iodine.

3. *On the skin.*—The eruptions produced by iodide of potassium appear under several forms. Erythema has been observed; and small round petechial spots, situated between the knees and ankles. These do not as a rule cause any inconvenience, and are usually accidentally discovered; but Stephen Mackenzie records the case of an infant of five months old, suffering from hereditary syphilis, who died of purpura after taking two and a half grains of iodide of potassium in a single dose. See **DRUG-ERUPTIONS**.

A pustular eruption resembling acne, and sometimes so profuse as to simulate smallpox, is not very uncommon. Large projecting nodules may appear on the face, or a copious crop of bullæ, which occasionally burst and become converted into fungoid growths, varying in size from a pea to a shilling. All these rashes are more likely to be produced by small than by large doses, and it is said that the frequent use of soap and the simultaneous administration of arsenic may prevent them.

4. *On the nutritive and glandular systems.*—Patients taking iodide of potassium sometimes complain of nausea, anorexia, and a bitter taste in the mouth; but where cachectic symptoms supervene, indicated by rapid emaciation, nervous palpitation, insomnia, and hypochondriasis, a ravenous desire for food has been observed. Vomiting, diarrhoea, and diuresis have also been described. Salivation is not an uncommon symptom. A time-honoured accusation against iodine is its supposed tendency to cause atrophy of the mammae and testicles; of this, fortunately, there is no real proof, the disappearance of the testicle, which occasionally accompanies the absorption of inflammatory products in its substance, being sometimes unjustly attributed to the treatment pursued. There is no doubt, however, of the anaphrodisiac effect of potassium iodide, and it is believed that permanent loss of sexual power may result from its long-continued use.

Fortunately for the reputation of one of our most useful drugs, the graver symptoms of iodism are decidedly rare. Iodine is often badly borne where the kidneys are diseased, and, as Trousseau pointed out, in exophthalmic goitre.

PATHOLOGY.—The only reason for the occurrence of most cases of iodism seems to be an individual peculiarity on the part of the patient. But now and then a more plausible explanation may be given, when we find cardiac or renal disease coinciding with the pustular rash of iodide of potassium. Retarded capillary circulation would naturally detain the drug within the blood; while the blocking of its usual means of exit from the system might be supposed to throw the onus of elimination on the glandular structures of the skin. Hence has been derived the plausible theory that iodine-acne is produced by direct local stimulation of the sebaceous structures; but, however true this may be in the slighter cases, Thin's careful examination of the skin of a patient suffering from a bullous rash has shown

the true pathological condition to be one of rupture of blood-vessels at certain localised points, with blocking by coagula, and escape of some of the constituents of the blood into the surrounding tissues. The sebaceous elements and sweat-glands were quite unaffected, and he believes the iodic papule, the so-called acne, the bulla, and the purpuric spot, represent different stages of vascular injury.

TREATMENT.—Coryza, or any skin-eruption, suddenly occurring in a patient taking iodide of potassium, ought to be looked upon with suspicion, and treated by the immediate suspension of the drug. The slighter varieties of iodism are by no means uncommon, and may appear after a single small dose; but although ammonia has been confidently vaunted as a specific against iodism, experience has been unable to confirm this, and we should place more faith in encouraging prompt elimination by very free dilution of the remedy, which is best taken on an empty stomach about half an hour before meals. Sulphanilic acid prevents or arrests iodism (Ehrlich), and so also does atropine (Binz).

ROBERT FARQUHARSON.
SIDNEY PHILLIPS.

IRIDOPLEGIA.—Paralysis of the iris. *See* PUPILS.

IRITIS.—Inflammation of the iris. *See* EYE, AND ITS APPENDAGES, Diseases of.

IRON-WORKERS.—*See* PNEUMOCONIOSES.

IRRIGATION (*irrigo*, I water).—A method of

applying cold water as a therapeutical agent, which consists in causing it to fall drop by drop, or in a continuous stream, on one spot. *See* COLD, Therapeutic Use of.

IRRITABILITY (*irrito*, I provoke).—In physiology this word signifies the power of responding to a stimulus, as exemplified by the contractility of muscular tissue. In medicine irritability implies an undue excitability of an organ or tissue, such as of the stomach or bladder.

ISCHÆMIA (*ἰσχω*, I restrain; and *αἷμα*, the blood).—Deficiency of blood in a part, short of complete cessation of the circulation: partial anæmia. *See* CIRCULATION, Disorders of.

ISCHL, in the Salzkammergut, Austria.—A sheltered, bracing, mild, rather moist climate. Altitude, 1,560 feet. Thermal common saline baths. *See* CLIMATE, Treatment of Disease by; and MINERAL WATERS.

ISCHURIA (*ἰσχω*, I restrain; and *οὐρον*, the urine).—This word properly signifies the arrest of the secretion of urine (*see* URINE, Suppression of). It is also applied to mere retention of urine.

ISSUES.—*See* COUNTER-IRRITANTS.

ITCH.—A popular name for scabies. *See* SCABIES.

ITCHING.—*See* PRURITUS.

J

JACKSONIAN EPILEPSY.—Epileptiform convulsions characterised by a constant local onset; a limited range, tending gradually to spread; a partial or complete retention of consciousness during the earlier attacks, and often until the whole of one side is involved; and temporary weakness of the affected muscles after each attack. The convulsions are due to irritation of the motor area of the cerebral cortex.

JACTITATION (*jactatio*, a tossing about of the body, or marked restlessness).—This is a condition mostly associated with certain severe febrile diseases, but also with some nervous affections, with severe pericarditis, or as a sequel of copious uterine or other hæmorrhages. A restlessness amounting to jactitation may likewise be met with in some patients, when suffering from severe or long-continued pain. It must not be confounded with certain forms of chorea, in which a somewhat similar tossing about of the body may be encountered. The absence of pain and of marked febrile disturbance, together with the history of the patient, will enable the latter condition to be readily distinguished.

JAPANESE RIVER-FEVER.—**SYNON.**: Shima Mushi.—An acute disease limited to the island of Nippon. It occurs during harvest, is confined to those engaged in handling the crops, is not infectious, and is characterised by fever, lymphadenitis,

cutaneous eschars—followed by ulceration, a dark-red papular eruption, bronchitis, conjunctivitis, and enlargement of the spleen. The disease lasts about three weeks, and is generally followed by recovery (85 per cent.). It is believed to be due to a form of acarus resembling the ‘harvest-bug.’

JAUNDICE.—**SYNON.**: Icterus; Fr. *Ictère*; *Jaunisse*; Ger. *Gelbsucht*.

DEFINITION.—Jaundice may be defined as a yellowness of the integuments and conjunctivæ, and of the tissues and the secretions generally, from impregnation with bile-pigment.

ÆTIOLOGY AND PATHOLOGY.—All cases of jaundice may be referred to one of two classes:—

I. Cases in which there is obvious mechanical impediment to the flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages, and thence absorbed into the blood (*Obstructive Jaundice*).

II. Cases in which there is no obvious impediment to the flow of bile in the larger ducts, but obstruction in the small intra-hepatic bile-ducts, associated with and dependent upon blood-disorder (*Toxicæmic Jaundice*).

Obstructive Jaundice.—When there is any obstruction to the flow of bile through the hepatic or common duct, the way in which jaundice arises is sufficiently clear. The bile-ducts and the gall-bladder become distended with bile, which is ab-

sorbed into the blood mainly by the lymphatics. If the hepatic duct of a dog be ligatured, and the animal killed after two hours, the lymphatics in the walls of the bile-ducts are seen to be distended with yellow fluid; the fluid in the thoracic duct is also yellow, and so likewise are the intervening lymphatic glands. In patients also who die of obstruction of the bile-duct, the lymphatics of the liver are often found to contain bile. On the other hand, two hours after ligature of the common duct, the serum of blood taken from the hepatic vein contains much more bile-pigment than that of blood taken from the jugular vein, which shows that in cases of obstruction of the bile-duct, bile is also directly absorbed by the veins.

To explain those cases of jaundice in which no obvious obstruction exists, several hypotheses have been promulgated.

1. *Suppression of the Hepatic Functions.*—It was formerly supposed that the function of the liver was merely to separate the elements of bile which were already formed in the blood; and that when anything interfered with the function of the liver, the elements of bile accumulated in the blood, and the result was jaundice of the skin and other tissues. This view has, however, no real basis; and all later observations show that the liver, not the blood, is the seat of formation of bile-pigment, and that jaundice by suppression, as thus defined, is an impossibility.

Suppression of function ('jaundice by suppression') is, however, supposed to be evidenced by two other classes of facts:—(a) *Suppression of bile-secretion* with the formation of pigments other than bile-pigments. There is no conclusive evidence of any such mode of causation of jaundice. In certain conditions pigments other than bile-pigment may be formed, and may in certain cases produce some discolouration, but this is totally distinct from jaundice. In many cases of jaundice evidence of altered activity of liver-cells is forthcoming, e.g. diminished secretion of bile and increased formation of bile-pigments; but such changes cannot be regarded as denoting 'suppression' of biliary function. On the contrary, in the larger number of such cases the most marked feature is an increased formation of bile-pigment—evidence, therefore, of increased activity rather than of suppression of function. (b) *Suppression of metabolic function*, as evidenced by diminished formation of urea with appearance of leucine and tyrosine in the urine. On this point, hitherto greatly relied upon as denoting a suppression of the function of the liver, there is, as one of the writers (H.) has shown, an almost complete absence of evidence. Thus, in phosphorus-poisoning the excretion of urea remains good up to the very last. In other words, there is no evidence of suppression of liver-function as regards urea-formation, any more than there is suppression of bile-formation.

It is thus exceedingly doubtful whether this 'suppression of function,' apart from actual destruction of liver-cells, plays any part in producing jaundice. In severe toxic conditions, such as *acute yellow atrophy of the liver*, a very extensive destruction of liver-cells occurs, with a corresponding loss of function; but the jaundice in such cases is not due to the 'suppression of function,' but to antecedent and concomitant changes affecting the epithelium of the smaller bile-ducts, causing increased viscosity of bile and obstruction.

2. *Hæmatogenous origin of bile-pigment* ('Hæmatogenous Jaundice').—There is no evidence whatever to support this mode of origin of jaundice. A hæmatogenous origin of bile-pigment sufficient in degree to cause jaundice does not occur. The liver-cell, not the blood, is the seat of formation of bile-pigment both in health and disease.

3. *Increased secretion of bile with excessive absorption from the intestine* ('Jaundice of Polycholia').—Many forms of jaundice, especially those produced by poisons, are marked at one stage or other by increased flow of bile and increased excretion of bile-pigment consequent upon the increased destruction of blood they occasion. There is no conclusive evidence that jaundice may result in such cases from increased absorption of bile from the intestine. The jaundice so often met with in such cases is the result of absorption of bile from the bile-ducts in the way presently to be described.

4. *Deranged Innervation* ('Jaundice of Emotion').—There is no evidence that this is a factor in the production of jaundice, although it is commonly credited with being an important one. It is, however, possible that strong mental emotion may cause spasm and reversed peristalsis of the involuntary muscular fibre of the bile-duct and intestine at a time when the bile is flowing.

5. *Toxic and Hæmolytic Factors.*—All the evidence goes to show that there are two important factors in producing those forms of jaundice unconnected with obvious obstruction, namely (a) increased destruction of blood with increased supply of hæmoglobin to the liver, and (b) action of poisons. These factors usually co-operate, for the most common cause of increased destruction of blood is the action of poisons. These two factors are not of equal importance. The occurrence of jaundice is determined more by the character of the poison than by the amount of blood-destruction. The most intense jaundice may be produced by poisons that cause but little, or only a moderate, destruction of blood, such as phosphorus or toluylenediamin. Most of the severe forms of jaundice met with in disease—'icterus gravis,' 'malignant jaundice,' 'acute yellow atrophy of liver'—are of this character, and illustrate this point. On the other hand, intense destruction of blood may be attended with little or no jaundice: for example, the hæmoglobinuria induced by the experimental injection of water, glycerine, or arseniuretted hydrogen, paroxysmal hæmoglobinuria, and pernicious anæmia.

Toxæmic jaundice is the result of absorption, just as simple obstructive jaundice is. It is caused by changes in the epithelium of the smaller bile-ducts, and increased viscosity of bile produced thereby. The jaundice is thus in every sense hepatogenous. The chief of the changes referred to are: (a) increased formation of bile-pigment, with (b) increased viscosity of bile, consequent on catarrh of the smaller bile-ducts. The increased viscosity retards temporarily the flow of bile along the bile-passages, and for a time may arrest this flow altogether, causing complete obstruction with absorption of bile. The cause of this intra-hepatic obstruction, as has been shown by one of the writers (H.), for the most notable of jaundice-producing poisons, namely, toluylenediamin, is a catarrh of the bile-ducts extending from above downwards (*descending catarrh*), produced by the excretion of the poison through the bile (*excretory catarrh*).

Simple and Toxicæmic Jaundice.—Instead, then, of the two varieties of jaundice formerly described, one *hepatogenous* or *obstructive*, the other *hæmatogenous* or *non-obstructive*, it is necessary now to recognise all jaundice as *hepatogenous* and *obstructive*—the result of absorption of bile—the cause of the obstruction being either simple and mechanical (*Simple Jaundice*), or due to increased viscosity of bile consequent upon action of poisons upon the blood, and their excretion through the liver (*Toxicæmic Jaundice*). The latter form of jaundice, although hepatogenous and obstructive, differs from the former in its origin, being primarily caused by the action of toxic agencies, hence the title ‘toxicæmic.’ This title at once emphasises the *toxic* origin of the blood-change, and the general *toxic* character of the symptoms usually found in association with this variety of jaundice. That is to say, it occurs in toxic conditions generally, in pyæmia, remittent fever, malaria, yellow fever, snake-poisoning, &c. The jaundice in all such cases has certain *pathological characters*: (1) It appears to be independent of any obstruction to the flow of bile in the larger ducts—the obstruction, as we have seen, being in all cases intrahepatic. (2) It is associated at one time or another with the presence of bile in the stools, often with an excessive formation of bile (*polycholia*). (3) The bile-acids may be diminished in quantity—this feature being a common one in jaundice caused by poisons.

We may now proceed to enumerate the different causes in tabular form:—

A. Jaundice from Mechanical Obstruction of the Bile-duct (‘Obstructive Jaundice’).

1. *Obstruction by foreign bodies within the duct*—Gall-stones and inspissated bile; Hydatids and Distomata; Foreign bodies from the intestines.

2. *Obstruction by inflammatory tumefaction of the duodenum, or of the lining membrane of the duct, with exudation into its interior.*

3. *Obstruction by stricture or obliteration of the duct*—Congenital deficiency of the duct; Stricture from perihepatitis; Closure of the orifice of the duct in consequence of an ulcer in the duodenum; Stricture from cicatrization of ulcers in the bile-ducts; Spasmodic stricture.

4. *Obstruction by tumours closing the orifice of the duct, or growing in its interior.*

5. *Obstruction by pressure on the duct from without, by—*Tumours projecting from the liver itself; Enlarged glands in the fissure of the liver; Tumours of the stomach, duodenum, pancreas, kidney, or omentum; Abdominal aneurysm; Accumulation of feces in the bowels; Pregnant uterus; Ovarian and uterine tumours.

B. Jaundice due to Intra-hepatic Obstruction of the Bile-ducts, caused by Action of Poisons on the Blood (‘Toxicæmic Jaundice’).

1. *The poisons of various specific diseases*—Yellow fever; Malarial fever; Relapsing fever; Typhus; Enteric fever; Scarletina; Icterus gravis; Acute atrophy of the liver; Pyæmia.

2. *Animal poisons*—e.g. Snake-poison.

3. *Mineral poisons*—e.g. Phosphorus; Mercury; Copper; Antimony; Arsenic; Chloroform and Ether.

SYMPTOMS.—From what has been stated in the preceding section, it is obvious that jaundice is not

a disease, but is a symptom of many different diseases. There are, however, certain phenomena connected with jaundice, independent of its cause, which deserve to be mentioned.

1. *Discolouration of Skin.*—Next to the liver itself, the skin is the tissue of the body which becomes most deeply jaundiced; but before it becomes affected, a yellow tint is usually observed in the conjunctivæ. There must be a certain concentration of bile-pigment to produce a yellow colour of the skin; in the slighter and more temporary cases, the conjunctivæ only may be affected. Jaundice of both skin and conjunctivæ is usually observed within twenty-four hours of closure of the duct.

The colour of the skin varies from a pale sulphur- or lemon-yellow, through a citron-yellow, to a deep olive or bronzed hue. The tint varies according to the cause and its duration. In obstructive jaundice it is light at first, and increases in depth the longer the disease lasts; although in advanced cases, as already stated, the colour sometimes becomes pale, not from the obstruction yielding, but from the tissue of the liver becoming destroyed and very little bile being secreted. In obstructive jaundice the depth of tint often varies from day to day, not from any variation in the degree of obstruction, but according to the amount of bile secreted by the liver, and the eliminative activity of the kidneys. It is well to remember that what is called ‘black jaundice’ may result from any cause of obstruction—from gall-stone as well as from cancer. In these cases the greenish or almost black hue is due to the absorbed bile-pigment being vitiated and dark, or to the visage being also darkened from imperfect aëration of the blood, the dark colour resulting from a mingling of the lividity with the colour of bile. In toxicæmic jaundice the discolouration is usually less intense; it is moreover of a more temporary character, rapidly disappearing if the cause be removed, and is generally associated with grave constitutional disturbances, as is set forth in the following sections. The colour also varies with the age, the natural complexion, and the amount of fat in the individual. It is deeper in the old, the wrinkled, and the dark-complexioned, than in young persons of fair complexion, and with plenty of fat. Lastly, it is important to remember that the colour often remains in the skin for some time after the cause of the jaundice has been removed, and that then its departure may be expedited by diaphoretics and warm baths.

2. *The secretions* are tinged with bile-pigment, but some much more so than others. This is notably the case with the urine, by which the greater part of the bile-pigment is eliminated from the body, and which acquires a saffron-yellow, greenish-brown, or brownish-black hue, according to the amount of pigment which it contains. The urine usually becomes yellow before the skin, or even the conjunctivæ; and when the cause of the jaundice is transient, it may happen that the whole of the pigment is eliminated by the urine, without the skin becoming jaundiced. On the other hand, when once the skin has become yellow, it may remain so for some time after bile-pigment has nearly disappeared from the urine.

Other secretions may contain bile-pigment as well as the urine. The cutaneous glands sometimes eliminate it in such quantity as to stain the linen yellow, but the amount discharged in this way is never great. Instances have been recorded where

the secretion of the mammary glands has been tinged with bile-pigment, but they are not common. Still rarer instances have been noticed where the saliva or the tears have been tinged. It is not a little remarkable that bile-pigment is not eliminated in cases of jaundice by the mucous membrane of the respiratory passages or of the digestive tube. This is a matter of some practical importance, for, were it otherwise, the stools might contain bile-pigment even when there was complete obstruction of the bile-duct. Still, when either of these mucous membranes is inflamed, and throws off an albuminous or fibrinous exudation, the altered secretions may contain bile-pigment. Thus, when pneumonia co-exists with jaundice, there is often bile-pigment in the sputa, which may be distinguished by the nitric-acid test from the greenish or yellow colour often presented by pneumonic sputa owing to changes in the blood-pigment independent of bile. Indeed, in cases of jaundice bile-pigment may be detected in inflammatory exudations, as in the serum of a blister, before it appears either in the skin or even in the urine. It is probable that those rare cases where the saliva has been noticed to be yellow admit of a similar explanation; in many of them there has been mercurial salivation, a condition in which the saliva is not normal, but contains much albumen.

3. *A bitter taste* is not infrequently complained of by persons who are the subjects of jaundice. It may denote the presence in the blood of the biliary acids, for taurocholic acid is intensely bitter. It is at all events not due to bile-pigments, which are tasteless. Moreover, it is a common symptom in biliary derangements where there is no jaundice.

4. *Derangements of digestion*, such as flatulence, constipation, and an altered character of the motions, may be due to the absence of bile from the motions. Bile is an antiseptic, though a very feeble one, and when it is absent the intestinal contents undergo fermentation, gases accumulate in the bowels, the fæces become putrid, and from the absence of bile and the presence of fat they present a pale-drab or clay colour. Bile is also the natural stimulant of the peristaltic action of the gut, and consequently, when the supply is cut off, the bowels are usually constipated; but in some cases the putrid fæces act as an irritant and excite diarrhoea. In those cases of jaundice where there is no obstruction of the common bile-duct, the motions may be but little altered.

When bile does not enter the bowel, the digestion of fat is interfered with. Jaundiced patients dislike fat, and do not assimilate it, and the fatty matter in the ingesta may sometimes be detected in the stools. Hence, whatever be the cause of obstruction of the bile-duct, the nutrition of the body suffers: the emaciation may be slow, but it is progressive, until all the fat disappears, and then the weight of the body may remain stationary for many months. With the emaciation there is always more or less muscular debility.

5. *Pruritus*, without any cutaneous eruption, is a very obstinate and distressing symptom in many cases of jaundice. It is usually worse at night, and, by preventing sleep, may wear out the patient. It is chiefly observed in cases of obstructive jaundice. It is not due to the presence of bile-pigment in the blood, for in some cases it precedes the jaundice, and in others it comes and goes during the persistence of the jaundice. Moreover, in many

cases of jaundice it is absent throughout, while it is not uncommon in biliary derangements where there is no jaundice.

6. *Cutaneous eruptions*.—Urticaria, lichen, and boils or carbuncles are occasionally observed in connection with jaundice; and likewise that remarkable affection of the skin known as *xanthoma*, *xanthelasma*, or *vittiligoidea*. See XANTHELASMA.

7. The *temperature* is not altered in jaundice, except when this occurs as a complication of some acute febrile disease, or when there is inflammatory action in the liver itself.

8. *Slowness of pulse*.—A common result of non-febrile jaundice is retardation of the heart's action, and diminution of arterial tension. The pulse may fall to 50, 40, or even 20, and sometimes it is also irregular. This slowness of pulse is particularly noticeable when the patient is recumbent. When there has been antecedent pyrexia, the pulse usually falls on the supervention of jaundice. Slowness and irregularity of the pulse are chiefly observed in jaundice from obstruction of the bile-duct, and particularly in those common cases known as catarrhal jaundice; and accordingly they are not unfavourable symptoms, as might have been supposed. So far as the writers' experience goes, patients with this symptom invariably recover. It has not yet been explained why this condition of circulation should be present in some cases of jaundice, and absent in others. Some experiments of Röhrig have shown that the salts of the biliary acids paralyse the heart, and retard its action, while bile-pigment has no such effect. It has been shown by Wickham Legg and others that the slowing of the heart is due to the action of cholalic acid on the cardiac ganglia. Slowness of the pulse, therefore, in jaundice may indicate the presence in the blood of unchanged biliary acids; but so far there are no observations to show that bile-acids are present in the urine in these more than in other cases of jaundice. See p. 171.

9. *Hæmorrhages*.—In many cases of toxicæmic jaundice hæmorrhages take place from the various mucous membranes and into the substance of the skin. They may also occur in cases of obstructive jaundice of long standing, from any cause, when the secreting tissue of the liver has in a great measure disappeared.

10. *Xanthops* or *Yellow vision*.—In rare cases of jaundice, all white objects appear yellow to the patient. The administration of santonin internally has also sometimes been followed by yellow vision, which has ceased as soon as the colouring-matter has been eliminated by the kidneys. This fact, as well as the observation that in several cases of jaundice with xanthops the conjunctival vessels have been preternaturally distended with blood, has led to the belief that the symptom is due to a tinging with bile-pigment of the humours of the eye. On the other hand, the circumstances that the xanthops may intermit, without any change in the jaundice; that it is usually absent when there is intense jaundice of the cornea and other tissues of the eye; and the statement that it may occur in typhus fever, and in certain derangements of vision, such as night-blindness, when there is no jaundice, have led most authorities to consider it, as appears to have been proved in regard to the effects of santonin, as due to an affection of the visual nervous apparatus.

11. *Cerebral symptoms and the Typhoid state*. Patients with jaundice are often irritable in their

temper and hypochondriacal; and occasionally they are attacked with acute delirium, stupor, coma, convulsions, muscular tremors, subsultus, carphology, paralysis of the sphincters, a dry and brown tongue, and other indications of the typhoid state. These symptoms are most common in cases of toxæmic jaundice, but they also occur in cases of obstruction, usually of long standing, where all or the greater part of the secreting tissue of the liver has been destroyed. Different opinions are held as to their cause. After death no lesion is found of the brain or its membranes, and they are, therefore, most probably due to some alteration in the circulating blood. There is ample proof that the blood of the human subject may be saturated with bile for months, or even years, without any cerebral symptoms resulting.

The cerebral symptoms in jaundice are often most severe when the jaundice is slight, and they may occur in diseases of the liver when there is no jaundice. They are best explained by the knowledge which we now possess of the function performed by the liver in disintegrating albuminous matter into less complex substances, such as urea and uric acid, which are eliminated by the kidneys. When this function of the liver is arrested or seriously impaired, as in acute atrophy, metabolism is impaired; leucine and tyrosine and other toxic substances, with which we are imperfectly acquainted, accumulate in the blood and tissues.

DIAGNOSIS.—There is rarely much difficulty in the diagnosis of jaundice, but it is well to remember that certain conditions are sometimes mistaken for it. Such are chlorosis, pernicious anæmia, hæmoglobinuria, the anæmic aspect resulting from organic visceral disease (and particularly from contracted kidneys), from cancer, from exposure to malaria, from Addison's disease, or from lead-poisoning; an undue amount of sub-conjunctival fat, or an unusually dark colour of the ordinary urinary pigment, or the presence in the urine of abnormal pigments, such as those of *santonin*, *turmeric*, *rhubarb*, &c. In every case where there is the slightest doubt, it will be removed by resorting to the nitric-acid test for bile-pigment in the urine. If this gives no result, the case is not one of jaundice.

It is a more difficult matter to determine the cause of the jaundice, and yet this should invariably be the aim of the medical attendant, before forming a prognosis or proceeding to treatment.

1. In the first place it is always well to determine whether the jaundice be of the obstructive or toxæmic type. In *obstructive jaundice* the stools contain no bile-pigment, and are therefore clay-coloured. In *toxæmic jaundice* the stools almost invariably contain bile, because the obstruction is rarely complete, and it is always accompanied by an increased destruction of blood and an increase of bile-pigments. The rule is not without exceptions, and there are several sources of fallacy. The jaundice usually persists for some time after the duct has become pervious, and thus bilious motions may co-exist with jaundice which has resulted from obstruction; or, if the motions be thin and watery, they may appear to contain bile from the admixture of jaundiced urine; or, not infrequently, when the bile-duct is quite impervious, the motions are of a brownish tinge, owing to the presence of altered blood, which may closely resemble dark bile. Iron, bismuth, and charcoal also colour the motions. A tumour corresponding to the region of the gall-

bladder will favour the view that jaundice is due to obstruction of the bile-duct. Lastly, jaundice which persists and is yet slight is most probably independent of obstruction, for jaundice from persistent obstruction speedily becomes intense.

2. It is always important to note the mode of commencement of jaundice. That which appears suddenly in a person whose previous health has been good is most probably the result of obstruction of the duct by a foreign body, or it has a toxic origin. The former cause will be distinguished by biliary colic, vomiting, and clay-coloured stools, the latter by some degree of fever and constitutional disturbance. On the other hand, jaundice coming on slowly, but ultimately becoming intense, with clay-coloured stools, points to pressure on the duct from without, or to a growth in its interior.

3. A history of previous attacks of jaundice of a similar nature is in favour of a catarrhal origin, or of gall-stones. See GALL-BLADDER AND BILE-DUCTS; and GALL-STONES.

4. Pain in severe paroxysms concurring with jaundice points generally to gall-stones or cancer; more rarely to hydatids, or to an aneurysm of the hepatic artery. Cancer is distinguished from gall-stones by there being usually a history of failing health and emaciation before either the pain or the jaundice.

5. Jaundice concurring with enlargement of the liver is most probably due to cancer or cirrhosis; more rarely to pyæmic abscesses, or to amyloid liver with large glands in the portal fissure.

6. Jaundice concurring with ascites points to cancer or cirrhosis. The diagnosis of the latter will usually be assisted by the physiognomy, the slowness of the jaundice, the previous habits, and a history of alcoholic dyspepsia; while in cancer there are often darting pains, and the jaundice is usually intense.

7. Jaundice concurring with pyrexia is either secondary to some acute febrile disease; or is due to suppurative pyelophlebitis, a suppurating hydatid cyst opening into a bile-duct, or inflammation of the bile-ducts. Temporary pyrexia may also occur during the passage of a gall-stone.

8. Cerebral symptoms associated with jaundice suggest acute atrophy of the liver, poisoning by phosphorus, pneumonia, or some specific fever.

9. Jaundice in a young person, preceded by symptoms of gastric catarrh, is most probably catarrhal.

TREATMENT.—There is no special treatment for jaundice. In a considerable proportion of cases, however, it is impossible at once to determine the precise nature of the causal disease, and pending an exact diagnosis we are obliged to adopt some provisional treatment. In view of the possibly dangerous character of the disease, the patient should be confined to bed for a few days. When, after a few days, no serious symptoms present themselves, and a catarrhal condition of the larger ducts is suspected, moderate exercise may be allowed even in the open air. The clothing should be warm, but light. The food should be simple and nutritious. There is no need to rigidly exclude fatty and saccharine matter, but no excess should be allowed, and pastry should be avoided. Alcoholic beverages, especially fermented liquors, should be withheld. In some cases of long-continued jaundice considerable quantities of flesh are required, and are digested, to supply the fatty matter lost to the system by the

absence of bile in the intestine. Milk is usually well borne, and supplies fat in an emulsified form. The patient should be encouraged to drink freely warm fluids or the natural alkaline waters, as those of Vichy, as the kidneys are the chief eliminators of bile. Warm baths should be used, and occasionally the Turkish bath is of service. At the commencement of an attack a dose of calomel or blue-pill (gr. ij.) in combination with colocynth or rhubarb pill, followed by a saline aperient draught on the following morning, may be administered. Mercurials should be cautiously used as long as an uncertainty as to the exact nature of the case remains, but the evil effects of mercury have been exaggerated, and it has been determined that it diminishes, and not increases, the secretion of bile. An alkaline and bitter stomachic is often of much service, especially when the tongue is coated; 20 grains of bicarbonate of potassium or sodium, with 20 minims each of aromatic spirit of ammonia and spirit of chloroform, 5 minims of tincture of nux vomica, in an ounce of some bitter infusion, as calumba, gentian, cascarilla, or chiretta, recently prepared, may be given three times a day between meals. In some cases, when the tongue is clean, or has been cleaned by an alkali, acids appear to do good; 10 to 20 minims of diluted nitro-hydrochloric acid in an ounce of a bitter infusion should be given half an hour before meals, and taraxacum may be usefully added—20 grains of the extract, or a drachm of the succus. Aperients are in many cases required, and in most cases useful. They have the effect of lessening portal congestion, and replace the natural purgative quality of the bile. Saline aperients as a rule answer best. A drachm of Carlsbad salt in a tumblerful of warm water, or half a tumblerful of Carlsbad water, to which some hot water has been added, should be taken before breakfast, and its efficacy is increased if it is *sipped* instead of being taken at a draught. Another useful saline aperient is phosphate of sodium, a drachm three times a day in a bitter infusion. In some cases vegetable aperients answer, when colocynth, aloes, rhubarb, or cascara may be given. When diarrhoea is present, salicylate of bismuth (gr. x.), or oil of turpentine (mx. to xx.) in capsules, on an empty stomach, should be tried. When jaundice is not due to an insuperable obstruction, euonymin, iridin, podophyllum resin, and other hepatic stimulants, are sometimes useful; and when there is gastro-duodenal catarrh, ipecacuanha is often of much service. The following is a good formula: \mathcal{R} Euonymin gr. jss., iridin gr. j., ipecacuanhæ gr. $\frac{1}{2}$, pilulæ colocynthidis et hyoscyami gr. ij.; ft. pil. ; one to be taken every or every other night. For flatulence, which often is troublesome, carbohc acid, creosote, menthol, and turpentine are useful. The following pill may be employed: \mathcal{R} Aloin gr. $\frac{1}{2}$, acidi carbolici (seu menthol) gr. j., extracti taraxaci gr. ij.; to be taken night and morning. Nitrate of pilocarpine is sometimes of service in relieving the pruritus often present. One-sixth of a grain should be given subcutaneously once or twice a day. For the same symptom warm baths at night, followed by the use of a flesh-brush, are sometimes serviceable. Bicarbonate of sodium, with bromide of potassium or belladonna, sometimes affords relief. The following lotion may also relieve the itching: Diluted hydrocyanic acid $\mathfrak{z}\text{j}$., borax $\mathfrak{z}\text{j}$., water to $\mathfrak{z}\text{viij}$. When these measures fail, and the night's rest is broken, an opiate may be required. Ox-gall is

sometimes useful in supplying the place of the deficient bile. The Fel Bovinum Purificatum (recently prepared) may be given in five- or ten-grain doses in coated pills, three times a day, two hours after meals. Daily enemata of one to two quarts of water at 60° to 90° F., to be retained as long as possible, are extolled by German physicians, especially in catarrhal jaundice. It cannot be too strongly impressed that, while using these measures, the physician's aim should be to establish the exact nature of the jaundice, when the correct treatment will be more clearly indicated.

CHARLES MURCHISON.
WILLIAM HUNTER.

JEJUNUM, Diseases of.—See **INTESTINES, Diseases of.**

JIGGER.—A popular term employed to designate the sand-worm or sand-flea. See **CHIGOE.**

JOINTS, Diseases of.—The diseases of joints will be considered as follows: I. GENERAL CONSIDERATIONS; II. LOCAL DISEASES: (1) Cartilages, Diseases of; (2) Congenital Dislocation; (3) Immobility; (4) Inflammation, Acute, of the Synovial Membrane; (5) Inflammation, Chronic, of the same; (6) Gonorrhœal Inflammation; (7) Gouty Inflammation; (8) Tubercular Inflammation; (9) Loose Cartilages in joints; (10) Nervous Affections; (11) Rheumatoid or Osteo-arthritis, Chronic; (12) Serous Effusions; and (13) Syphilitic Disease.

I. GENERAL CONSIDERATIONS.—Diseases of the joints are classified according to the structure primarily or chiefly involved. They may commence in the synovial membrane, in the bone, or in the cartilage. Primary disease of the ligaments is rare, and is not clinically demonstrable. No form of joint-disease remains long confined to one tissue, so that when the disease is of some duration it will be found to implicate, more or less, every element of the joint-apparatus. In this article the diseases of joints will first be generally discussed; and the individual diseases will then be considered separately.

ETIOLOGY AND PATHOLOGY.—The larger articulations, those in constant use, and more especially the joints of the lower extremity, are the most frequently diseased. Thus the knee is more often the seat of disease than any other joint; the hip-joint comes next in order; and then the ankle and elbow. All kinds of joint-diseases are frequent in children and young persons. The first year of life appears, however, to be nearly exempt from these affections, and during the second year they are comparatively rare, perhaps because movement and risk of injury are at that period at a minimum. Acute arthritis, however, is occasionally witnessed during the first year of life—during even the first six months. It is unconnected with syphilis or injury; very sudden in its appearance, and rapid in course; dangerous to life; and destructive to the articular ends of the bones by suppurative disorganisation. The causes of joint-disease in general are connected either with disordered nutrition, in which case it usually assumes an inflammatory type; or with disordered function. The latter may depend on the former, or be unconnected with it. Again, the cause may be local in its origin or arise from some constitutional defect. Among the *exciting* causes, injury is by far the most frequent. This being often slight, and not followed by any imme-

diate consequences, the connection is frequently overlooked. A blow, or a fall against the edge of a table or down stairs, may readily bruise the synovial membrane in such exposed joints as the knee or elbow, without causing any external sign. A slight hæmorrhage takes place into the synovial cavity or the sub-synovial areolar tissue, and serous effusion may speedily supervene; in this manner a common variety of acute or traumatic serous synovitis is produced. But although injury is a most fertile cause of joint-disease, the articulations may sustain most severe injury without becoming inflamed. It is rare to find any serious consequences result from dislocation; the joint usually perfectly recovering itself. Penetrating wounds of the joints are always serious injuries; they often occasion acute synovitis, and, if septic changes occur, are followed inevitably by suppuration in the articulation, and danger both to the limb and life of the individual. Fractures often implicate the joint-surfaces, and prove a frequent source of stiff-joint. Plastic synovitis may be thus set up, causing adhesions; or suppuration takes place; or the callus formed for the repair of the fracture may interfere with the joint-motion. Gunshot wounds of joints often produce the severest form of inflammation, suppuration being the usual result. When joint-disease follows an injury, it is usually confined to one joint; but when joint-disease originates from constitutional causes, more than one joint is often affected; or when only one, the constitutional nature of the cause is manifested in diseased conditions present elsewhere, or by traces of inflammation in other joints due to the same cause. The development of tuberculosis in the synovial membrane and bone is a very frequent cause of chronic joint-disease.

Joint-inflammations are of common occurrence in all kinds of fever; and also as sequelæ of the exanthemata. The great frequency of polyarticular serous synovitis in acute rheumatism is well known, as also in purpura and hæmophilia, where it is complicated with blood-extravasations. In pyæmia the joints are frequently the seat of sero-purulent and purulent effusions; as they also occasionally are in scarlatina. Puerperal synovitis is a variety of the pyæmic. In typhus monarticular arthritis is frequently met with, and the hip is the joint oftenest affected. Endocarditis and polyarthritis are very often associated together, and the endocarditis may precede and give rise to the joint-disorder by embolism. The fact that multiple joint-affectations are met with both in pyæmia and in rheumatism suggests a connection, but what its nature may be is not clear. Although in articular rheumatism pus-formation is rare, we sometimes witness joint-sup-
puration in such cases; while pyæmia and metastatic abscess may originate from ulcerative endocarditis. In chronic synovitis, affecting two or more joints, the heart should always be examined, for traces of endocarditis will sometimes be found. In the exanthemata, typhus, and diphtheria, metastases in the shape of joint-inflammations more or less frequently take place. Joint-inflammation is of frequent occurrence in dysentery. With gonorrhœa a form of arthritis is associated which is called 'gonorrhœal.' Syphilis in the later stages frequently attacks a joint, the knee by preference, syphilitic deposits taking place in the bone or the sub-synovial connective tissue, but synovial effusion is not common. A suppurative inflammation of

the ends of the bone is not rare in children the subjects of inherited syphilis. In gout the joint-structures are affected; as a rule the peri-synovial tissue becomes inflamed owing to deposits of urates. Similar deposits also occur in cartilages of encrustation. The great toe is most often affected, but the other tarsal, digital, and larger joints are frequently diseased.

Some ill-understood form of vaso-motor or trophic irritation appears to occasion arthritis in locomotor ataxy. Effusion into the joint is preceded by pain; and the knee and shoulder joints are those generally affected. In some cases of the disease changes similar to those in rheumatoid arthritis have been observed, and generally in the knee, shoulder, elbow, or hip. They occur early in the disease; arise suddenly; are often monarticular; and not rarely give rise to dislocation, especially in the shoulder. These characters distinguish the disease from ordinary rheumatoid arthritis. Severe inflammation of the joints of the paralysed limbs has been observed in cases of hemiplegia. The occurrence of joint-disorder, usually synovial inflammation, is frequent in chronic disease of the spine; and it also occasionally happens in acute softening in the form of suppurative arthritis. In both cases the knee is most frequently affected.

ANATOMICAL CHARACTERS.—Joint-disease may begin as an inflammation of the synovial membrane, of the bone, or of the cartilage. Fibrous tissue having but slight tendency to inflame, it is improbable that primary disease affecting the ligaments can be otherwise than most exceptional; but these textures very soon become secondarily affected, from their intimate connection with the synovial membrane. The synovial membrane is perhaps more ready to inflame than any other tissue in the body, and in many joints it is much exposed to injury from without, while excessive joint-movement alone is sometimes sufficient to excite synovitis. Primary disease of the bone comes next in order of frequency. Cartilage is least likely to take on primary disease (*see p. 251*). Each of these tissues, however, becomes speedily affected by disease which has invaded or commenced in the other.

SYMPTOMS AND DIAGNOSIS.—The local symptoms of joint-disease have reference to *impairment of function and change in form*; together with *pain*, both local and sympathetic; and certain *physical signs*.

Impaired function.—Usually this is great in proportion to the natural mobility and importance of the joint, and most evident in the extremities. The earliest symptom in hip-joint-disease is a slight limp or halt, while in other joints mere stiffness occurs; the full range of movement is simply curtailed, before actual pain or swelling takes place. The position of maximum relaxation—namely, that intermediate between flexion and extension—is commonly assumed by diseased joints. Even in the early stages of disease, the interference with movement is often very great, amounting to a sort of vital ankylosis, produced by the action of the muscles, whose tension prevents the joint-surfaces moving upon each other—an effort to avert pain. This form of ankylosis disappears during necrosis. Muscular or vital ankylosis must be distinguished from the rigidity produced by structural changes. Both synovial effusion and peri-synovial infiltration mechanically hinder free joint-movement.

Changes in form.—Changes in form are due to

the alterations in shape and texture of the joint-structures, and to effusions within its cavity. These changes may be best appreciated by careful measurements, and a comparison with the opposite joint. The practitioner is thus better able to diagnose the special character of the swelling, whether it be due to synovial effusion, and confined to the limits of the capsule, causing it to bulge at the least protected parts; or to chronic thickening of the synovial membrane, recognised on palpation by its elasticity and more general diffusion; or to disease of the bone and periosteum, when the swelling is deep-seated and hard. By accurate comparison a fluid collection outside the joint, either an abscess or a bursal tumour, may be distinguished from intra-articular swelling.

Pain.—The character of the pain is an important symptom in diseases of the joints. In acute synovitis it is severe and lancinating. In bone-inflammation it is a dull aching pain, with marked local tenderness, liable to periodic exacerbations of an intense kind. Often the pain is of a shooting, starting character, wakening the sufferer from sleep. The pain is of this character and most severe in sub-articular osteitis. Pyæmic suppuration and chronic synovitis are generally painless.

Physical signs.—When one hand is laid flat upon a diseased joint while the other moves it, certain sensations or sounds are often distinguishable. A peculiar soft crepitation, due to the presence of blood-clot, must not be mistaken for the rougher sensations which adhesions afford, the friction-sounds of movable bodies within the joint, or the grating of exposed bone. The rubbing of one granulating-surface upon another may be likened to that of two pieces of velvet. Abnormal movements, such as lateral motion in a ginglymoid joint, usually imply extensive joint-disorder. Displacement or partial dislocation, and alteration in form of the joint-surfaces, occur as the disease progresses. A notable increase of local heat may be felt in all inflamed joints. When fistulous tracks exist around a diseased joint, they do not often, on being probed, afford direct evidence of the condition of the joint, but they generally prove the existence of articular suppuration, and disease of the bone.

COMPLICATIONS AND SEQUELÆ.—The complications which occur in joint-disease are generally connected with long-continued suppuration. Amyloid degeneration of the viscera is pretty certain to be present when suppuration has existed for a year or more in young people; less certainly in adults. Hætic fever, tuberculosis, or pyæmia may occur at any period. In the absence or failure of treatment, the patient, should he survive, will suffer from contraction, deformity, and imperfect growth of the limb, together with more or less complete loss of function.

PROGNOSIS.—The prognosis in diseases of the joints will depend on many circumstances, and must be considered both as regards life and as regards function. First, with respect to *life*, the gravity of joint-diseases increases with the size of the joint affected. They are more serious in the lower than in the upper limb. Pyæmia is comparatively rare in acute joint-suppurations—why, it is impossible to say. When pus escapes from the interior of a joint into the surrounding tissues, pyæmia may occur. A continuous high temperature, or a large evening increase associated with hectic, is a bad sign; the exhaustion, which depends on profuse

suppuration with its attendant hectic fever, amyloid degeneration, and tuberculosis, are the most frequent causes of death in joint-disease. The prognosis as regards *function* is often difficult to determine. After an attack of simple acute or sub-acute serous synovitis, recovery is generally complete. Joint-function is usually absolutely lost after suppuration of traumatic origin, recovery being quite exceptional. When the suppuration is of a pyæmic nature and the patient survives, the effusion may become absorbed, and the joint-motion be preserved or ankylosis ensue. Chronic synovitis with thickening of the sub-synovial tissue, due to infiltration with granulation-material, can seldom be cured except by operation, especially after suppuration has taken place. If recovery should take place, the joint-function is lost, and deformity is always present. Increased mobility—‘flail’ joint—is a very rare sequel of joint-disease. It is occasionally seen in the shoulder, and in the knee.

TREATMENT.—The treatment of diseases of the joints must be directed to preserve the functions of the limb. Of the first and greatest importance among remedial measures is *rest*, which is best secured by fixation of the joint and limb in an appropriate apparatus. This is of cardinal importance to a diseased articulation, just as motion is a necessity for a healthy one. Immobilisation should not be continued longer than necessary; it will sometimes seriously damage even a previously healthy joint, immobilised for instance on account of fracture of the limb; and a continuance of rest after all diseased action has subsided often exerts a prejudicial influence. Rest, however, should be continued so long as swelling, pain, and increased temperature persist.

Position.—The means adopted to secure immobility must be also utilised to obtain the best available position for the future function of the part, should ankylosis become inevitable. In the ankle the foot should be maintained at a right angle; the hip and knee must be kept extended in the axis of the body; the elbow is generally flexed to a right angle, the position in which the limb is most useful. Splints of various forms are used, and we possess in plaster-of-Paris and starch ready and invaluable means of producing an apparatus which gives uniform and equable support of a simple and very perfect kind.

Extension exerts a beneficial influence, as well by immobilising the joint as by its power to remove contraction and deformity. It relieves pain and abates the symptoms rather by keeping the joint at rest, and changing the surfaces of contact, than by any actual separation of the joint-surfaces. A much greater weight than a patient could tolerate would have to be used before any such separation could occur. Extension often even increases the intra-articular pressure. By straightening the limb it removes the joint from its position of maximum relaxation, and puts the skin and tendons on the flexor aspect on the stretch, and alters the mutual accommodation of the joint-surfaces.

As there is almost invariably an increase of temperature in the affected joint, the application of *cold*, by means of ice-bags or coils of cold-water tubing, is indicated. Cold acts most beneficially in all acute, and many subacute inflammations. Even in deep-seated joints like the hip, it will often soothe the pain and abate the symptoms, but it is more applicable to the superficial joints. Cold is

both anæsthetic and prophylactic in its action. The sensations of the patient in respect of the continuance of cold applications must be consulted. In most cases they are grateful. In chronic joint-affections when an acute attack supervenes, threatening suppurative, cold should also be applied. Cold applications may in some cases be continued for weeks or months with advantage. When the acute symptoms have passed off, and it is desirable to encourage lymphatic activity and absorption, the cold must be discontinued, and compression, together with friction and warmth, substituted. Cold is not applicable to purely chronic cases without much pain or tenderness.

In some instances of acute and subacute arthritis *local depletion* by means of leeches or scarification is very useful, and this may be combined with hot *fomentations* in cases where cold is not well borne. In chronic inflammation of the bone the *actual cautery* sometimes procures immediate abatement of the pain, and, after a time, the subsidence of the inflammation. The 'button' cautery may be used, or, still better, linear cauterisation, over the most sensitive points. Or an *issue* may be employed instead.

Compression by strapping, or with a thick layer of cotton-wool and a tightly applied bandage over it, is applicable to the chronic stages of joint-disease. It must be discontinued if it occasion pain. It is better calculated to remove fluid effusions than the plastic material poured out into the peri-synovial tissue. For these cases the more continuous compression of a properly applied elastic bandage will prove more efficient; or the strapping known as 'Scott's dressing.'

Massage is a most valuable local means for the dispersion of chronic swellings of joints. It both removes the results of diseased action in the joint, and helps to restore its function. It is well suited to disperse serous effusions when the acute stage is over; for cases of plastic synovitis it is also useful, but not for cases of the type known as *tumor albus*. It produces a diminution of the sensibility of the part, and a local increase of temperature, and the lymphatics are stimulated to increased activity. There are several modes of employing massage. The first is centripetal stroking with the palm of the hand from the periphery of the affected part towards the centre of the body, called *effleurage*, one hand following the other in immediate succession. The amount of pressure varies with the circumstances of the case. This will readily disperse fluid effusions both of blood and serum. Friction-massage is another useful method, and is practised by pressing the palm firmly upon the surface, and then rotating it. This plan may be combined alternately with the last method, massage proper—*pétrissage*—which is done by raising up the soft parts vertically from the bone with both hands, and compressing them, always in a centripetal direction. See MESSAGE.

Forcible movements.—Forcible movements, which break down adhesions, are often most useful in cases of stiff joint arising after protracted immobilisation, as after fracture in the vicinity of a joint, or after a severe sprain. Pain will be relieved in this way, and mobility restored in some instances, in a degree quite remarkable. They are less applicable in cases of fibrous ankylosis.

Constitutional treatment.—Where any general taint exists, this must be dealt with at the same

time. A tendency to tuberculosis must be met by iron, tonics, good food, and pure air. Gout, syphilis, or rheumatism must, when present as a diathesis, be appropriately treated.

Operative treatment.—Puncture alone, or combined with antiseptic washing-out of the articulation, may often be performed with advantage, to evacuate the fluid in a distended joint, or to diagnose the presence of pus and evacuate it when suppurative has taken place. Sufficiently free incisions, however, and the insertion of drainage-tubes, are generally to be preferred in cases of joint-suppurative; and with these should be combined the washing-out the joint-cavity with a three- or five-per-cent. solution of carbolic acid, or other effective antiseptic. It has been proposed to substitute free incisions and drainage for excision of the joint, in certain chronic forms of disease, such as white-swellings, but excision, or arthrectomy, is probably in most respects preferable. Excision is practised for chronic joint-disease not amenable to other means; it is not a substitute for amputation, but is intended to obviate its necessity. Subperiosteal resection, where practicable, possesses many advantages, especially in cases of traumatic origin. The attachments of the muscles and tendons, and the cellular interspaces between them, are thus left undisturbed. The chances of peri-synovial suppurative are diminished, and the bleeding is reduced to a minimum. There is more complete bony reproduction of the joint-surfaces, and in young persons a new joint very similar to the normal is in some instances formed, while in all cases there is a probability of better subsequent function and position. The operation thus performed requires time and skill. It is scarcely applicable to the knee or even the hip, and is unsuited for cases of chronic synovial disease, where it is of the last importance to excise all the diseased granulation-material. The after-treatment of excisions is of great importance. Plaster-of-Paris bandages supply one of the most useful means of immobilisation, especially in those cases where ankylosis is sought for, as in the knee; and the splint should be unchanged, if possible, for four or five weeks. In the elbow, shoulder, and wrist, where mobility is the end aimed at, passive movement should be commenced as soon as the condition of the wound admits of it—namely, in about a week or ten days. Galvanism must be used at a later period to restore the wasted muscular apparatus. Arthrectomy aims at the removal of all the diseased tissue, yet stops short of a formal excision in which healthy parts may be removed.

Amputation is only performed as a last resort. It is very rarely needed for joint-diseases in the upper limb, except perhaps the wrist, when the hand is permanently crippled. In the lower limb, amputation must be performed in those cases in which the patient has lost all strength and healing power, from the drain of a long-continued discharge. It is advisable where amyloid degeneration or incipient tuberculosis exists, or in any case, in short, in which the power of the patient is inadequate to furnish the amount of repair required in the expectant form of treatment, or in case of excision, always a more serious operation than mere amputation. Amputation should also be adopted in those cases in which the local disease, especially of the bone, is too extensive to admit of a good functional result after excision. In the very young

formal excision is undesirable, since the epiphyses are almost of necessity sacrificed, and the growth of the limb checked. Resection in some joints is practised to avert ankylosis, or to restore the lost function of the joint, as in the shoulder, elbow, and wrist. Various congenital and other deformities of the joints may be removed by osteotomy of the bones concerned.

1. Joints, Cartilages of, Diseases of.—See CARTILAGE, Diseases of; BONE, Diseases of; and following sections.

2. Joints, Congenital Dislocation of.—DESCRIPTION.—This is a curious and ill-understood affection of the joints. It is almost exclusively confined to the female sex. The hip-joint is nearly always the one affected, and the displacement is generally double. It has often been erroneously mistaken for morbus coxae, and treated accordingly.

It probably occurs in early foetal life from defective formation of the joint-surfaces. It is not discovered until the child begins to walk, which it generally only commences to do at a late period. When the hips are the joints affected, the gait is accompanied by a most ungainly swaying of the body from side to side like the waddling of a duck. Great improvement may be effected if the dislocation be reduced and the head of the bone kept in the socket by fixing the limb and pelvis with plaster in a suitable position.

3. Joints, Immobility of.—SYNON.: Ankylosis; Fr. *Ankylose*; Gr. *Gelenkverwachsung*.

ÆTIOLOGY.—This condition may be due to changes in the structures of the articulation constituting a *true ankylosis*; or in those surrounding the joint, a *false ankylosis*. It may be fibrous and incomplete, or bony and complete. False or spurious ankylosis—extra-articular—may depend on muscular spasm or rigidity; on cicatricial contractions; on paralytic or spasmodic affections; or upon prolonged disuse of the joint. It is often difficult, even under chloroform, to distinguish the presence of absolute bony ankylosis, as the fibrous form may be so strong and extensive as almost wholly to prevent movement. The two varieties are but degrees of the same process. Both may result from previous inflammatory changes in the joint, either of the nature of plastic synovitis, or of granulations springing from the bone and other joint-tissues, becoming further organised. Fibro-cartilaginous ankylosis is a common form in young persons. In time it usually becomes converted into true bony ankylosis.

The marked ankylosis which tonic spasm and rigidity of the muscles produce in the early stages of some joint-diseases, as in the hip-joint and knee, may be called *vital* or *physiological*. It is induced by an effort to avert pain; it disappears entirely during necrosis. A joint may become stiff and ankylosed by long fixation on account of some injury or disease elsewhere, especially if it be retained in a flexed position, as the muscles of the flexor side actually shorten when their points of origin and insertion permanently approach one another. The same thing may also happen in the muscular atrophy due to paralysis; in which the cartilages and bones atrophy at the same time.

TREATMENT.—The treatment of diseases of joints should, in all cases where it is possible, be prophylactic against the occurrence of ankylosis. When this has taken place in an incomplete degree, an attempt to restore the function of the limb must

be made, by breaking down the adhesions by forcible or gradual extension, by passive motion, by massage, and by tenotomy of the tense tendons. Excision is indicated to restore motion in complete ankylosis of such joints as the wrist, the elbow, and the shoulder. In other cases of complete ankylosis, especially in the lower limb, surgical interference should be confined to an attempt to rectify a faulty position either by tenotomy, extension, forcible straightening, excision, or osteotomy. Amputation can only be needed in extreme and otherwise irremediable deformity.

4. Joints, Acute Inflammation of the Synovial Membrane of.—SYNON.: Acute Synovitis; Fr. *Synovite Aiguë*; Ger. *Acute Synovitis*.

This is one of the most common of all joint-affections. Probably in half the total number of chronic joint-diseases the synovial membrane is first affected. The synovial membrane is very rich in vessels and cells; and much exposed to injury, and to the effects of movement. An inflammation beginning at one point soon spreads over the whole synovial sac.

DESCRIPTION.—Acute synovitis is a very common result of injury. It also occurs in rheumatism, gout, pyæmia, and other diseases. It may be serous, sero-fibrinous, or purulent. Serous synovitis is the simplest and most common variety. Even a slight external injury is often sufficient to produce it; a sprain of the joint, or excessive movement, may cause it, as well as the constitutional disorders already mentioned. The knee is very often affected, from its exposed position, and the large area of the serous membrane lining it. The synovial membrane becomes injected and thickened, with effusion into the sub-synovial tissue. The natural secretion is increased in quantity, and many cells are shed into the joint-cavity, the capsule becoming swollen, tense, fluctuating, or elastic. The least protected parts bulge, from the pressure of the effused fluid, and the normal contour of the joint is lost. It is usually semiflexed. A severe, burning, cutting pain is experienced in it. It is exceedingly sensitive to pressure, and painful on the slightest movement. There is usually considerable fever. When the inflammation is more intense, the synovitis becomes sero-fibrinous. Flakes of lymph are mingled with the synovia; layers of false membrane cover the synovial membrane, which is considerably thickened and dull-red in colour; and the constitutional disturbance is greater. Finally, it is but a short step from this to suppurative synovitis. If the irritant cause continue its action, the leucocytes filling the meshes of the synovial membrane are shed in larger quantity; the fluid becomes turbid and puriform; the fever and local symptoms increase very much in severity; the external parts become implicated in the inflammation within; and suppuration or abscess is the result.

TREATMENT.—The treatment of the first two stages of synovitis is directed primarily to check the progress of the inflammation; and then to procure resolution, and absorption of the effused fluid. Fortunately the synovial membrane possesses very active absorbent powers; and early and efficient treatment, conjoined with removal of the source of irritation, will generally ensure a cure, with complete restoration of function. The chief means are cold applications; immobilisation till

the acute stage is over ; and then compression and friction, or in suitable cases counter-irritation. Gout, rheumatism, or other diathesis, must be appropriately treated at the same time.

In those cases in which the inflammation has persisted for some time, and plastic effusion has taken place on the surface and in the substance of the synovial tissue, the joint will remain for a long time stiff and thickened, and its function impaired, after all acute symptoms have subsided ; while in those cases where the inflammation has continued long enough to invade the other joint-structures, a perfect cure may not be possible.

When suppuration occurs, the joint must be dealt with as any other abscess-cavity. Free incisions must be made into it, and it should be thoroughly washed out with some antiseptic solution, free subsequent drainage being provided for. In the more favourable cases, ankylosis in a convenient position will be obtained. Recovery of function is very rare. In other cases the suppuration continues ; the cartilages become necrosed and detached ; the bone becomes exposed and carious ; and either excision or amputation must be performed to save the patient's limb or life.

5. Joints, Chronic Inflammation of the Synovial Membrane of.—SYNON. : Chronic Synovitis.

Chronic synovitis may arise as the sequel of the acute disease ; or, as is more frequent, it may depend on some constitutional dyscrasia, or at least some continuously acting irritant, although in the first instance it is generally excited by an accidental injury.

VARIETIES.—There are three chief varieties of this disease which are often co-existent, namely : *pannus-synovitis*, *granulation-synovitis*, and *papillomatous synovitis*.

a. *Pannus-Synovitis*.—In this variety a delicate membrane will be found, stretching from the inflamed and thickened synovial margins more or less over the surface of the cartilage, to which it may be in whole or in part adherent. This change may result simply from prolonged rest.

b. *Granulation-Synovitis*.—The condition formerly described under this name is, in the large majority of cases, of tubercular origin. See section 8.

c. *Papillomatous Synovitis*.—SYNON. : Fimbriated disease of the synovial membrane ; Papilloma, or Papillary Fibroma of the synovial membrane ; Ger. *Gelenkscotten*.

This is a peculiar form of joint-disease dependent on chronic synovitis, in which numerous pedunculated bodies, cylindrical or fusiform, varying in size from a pin's head to a large pea, project from the membrane, generally near the margin of the cartilage, or may cover the entire surface. These bodies are identical in minute structure with the synovial fringes. Some become detached, and fall into the cavity of the joint. There may be dozens or hundreds of these bodies present in one joint. Occasionally they contain cartilage-cells, or osseous particles. The joints of the lower limb are most often affected.

TREATMENT.—The disease is scarcely remediable except by arthrectomy or excision of the joint, which may be practised in those cases in which there is serious loss of function.

6. Joints, Gonorrhœal Inflammation of. See GONORRHEAL ARTHRITIS.

7. Joints, Gouty Inflammation of.—See GOUT.

8. Joints, Tubercular Inflammation of.—This is most frequent in the knee- and hip-joints. Almost all the cases described as *tumor albus* are cases of tubercular disease.

ANATOMICAL CHARACTERS.—This disease generally commences in the synovial membrane, which becomes thickened, and by degrees converted into a semi-gelatinous mass of granulation-tissue, yellowish-white or pink in colour. Or the disease may originate in osteomyelitis of the end of the bone, the inflammatory process being essentially chronic and dependent upon the presence in the tissues of miliary tubercle. After the disease has existed for some time, it is difficult to determine in what tissue it may have originated, and it is of little clinical importance to do so, for in any case the later stages of the malady present similar features. The cartilages are encroached upon from their margins and from their deep surfaces by the granulations, springing from the synovial membrane and the articular lamella, while active changes occur simultaneously in their substance, similar to those already described. The ligaments soften, and all the structures of the joint become involved. Frequently larger or smaller masses of necrosed bone will be found in the cancellated structure, and the granulations have a great tendency to suppurate. In the thickened synovial membrane, and also in the ends of the bones, miliary bodies, identical with tubercles, may very frequently be detected.

SYMPTOMS.—In tubercular inflammation of the synovial membrane the joint is uniformly swollen, tense, elastic, with a white glistening surface, and enlarged veins shining through the skin. The patient can usually move about until suppuration has taken place, as the pain is never very severe in the intervals of the acute attacks of inflammation which supervene from time to time. Enlargement of the lymphatic glands, or marks of strumous ulceration elsewhere, are seldom wanting ; while sooner or later a large proportion of the individuals affected by this form of joint-disease show signs of general tuberculosis. Sometimes this state precedes, but generally it follows, the local joint-affection. When the disease begins in the bone the localised tenderness and deep-seated thickening, conjoined with the general symptoms, may reveal the nature of the process before the more general invasion of all the joint-elements.

TREATMENT.—This must be mainly directed to improving the patient's general condition. Any local treatment, short of a complete removal of the diseased structures, is not of the least use when the joint has become disorganised, as before described ; and some form of excision should be performed before the viscera become implicated. If other organs be involved, or the local disease be too extensive, then amputation becomes imperative. When not removed, fresh foci of suppuration form ; the patient becomes more and more exhausted ; or some intercurrent disease sets in. It is rare for spontaneous cure to happen.

9. Joints, Loose Cartilages in.—SYNON. : Fr. *Corps Flottants Articulaires* ; Ger. *Gelenkmäuse*.

DESCRIPTION.—'Loose cartilages' in joints may originate either from chronic inflammation or from traumatic causes. They may be single or multiple. The knee-joint is most frequently affected, and in it

the most serious symptoms are produced. These bodies may be fibrous, fatty, cartilaginous, or partly ossified. They may be produced from polypoid growths springing from the synovial membrane in certain forms of chronic synovitis, and in arthritis deformans; and they are then usually of the fibrous or osteoid variety. Free bodies consisting of fat are rare, and are derived from the subsynovial fatty tissue, often produced in a fashion analogous to the appendices epiploicæ of the great intestine.

The cartilaginous and osteoid formations are the largest and most important varieties of these bodies; hence the common term 'loose cartilage.' Portions of the joint-surface may sometimes become detached, as the consequence of injury, or by a process of quiet necrosis. They thus become loose in the joint. It is stated, but the fact is doubtful, that some of these bodies may obtain nourishment from the surrounding synovial fluid, and that cartilage and even bone can be developed in them subsequently to their detachment.

SYMPTOMS AND DIAGNOSIS.—The symptoms of loose cartilages in a joint vary very much. In some instances these bodies cause no inconvenience. In others they produce repeated attacks of excruciating pain, followed by synovitis, laying the patient up for weeks; while in the most severe cases the limb may become almost useless. When the knee is the joint affected, the patient experiences great insecurity in walking, the loose body from time to time becoming wedged between the joint-surfaces. The joint is thus 'locked.' The patient may suddenly fall, or faint with pain; an attack of synovitis follows; and with a frequent repetition of this process joint-disorganisation may finally result. The prognosis as regards function is always bad.

TREATMENT.—The treatment of loose cartilages may be either *palliative* or *radical*.

The *palliative* method consists in applying support to the joint; limiting its movements; and fixing the loose body in some synovial pouch where it cannot interfere with the articular surfaces.

The *radical* method consists in excising the body—an operation, with few exceptions, almost exclusively practised upon the knee-joint. The body may be removed by a free direct incision into the joint, and squeezing the body through the wound at once. Or the indirect manner of operating may be adopted. This consists in subcutaneously incising the capsule of the joint with a long narrow-bladed knife introduced at some distance from the articulation; forcing the body through this incision into the cellular tissue outside; and then closing the small external puncture in the skin. Three or four weeks later the 'cartilage' may be removed by a superficial incision, or left undisturbed, when it often becomes absorbed.

In appreciating the comparative value of these two plans, it may be said that the former has hitherto proved more uniformly successful *quoad* extracting the body, but that it has been more dangerous to limb and life—a danger, however, which antiseptic precautions reduce to a minimum. The indirect method has been attended by a considerable number of failures in the extraction of the loose cartilage, especially if it be pedunculated; but it has hitherto proved a less dangerous operation. The extremity should be immobilised afterwards for two or three weeks.

When some dozens of these bodies are present in a joint, many of them free, many attached, excision

of the articulation is often the only remedy. This is a severe measure, and not to be lightly undertaken, in the joints of the lower limb at all events.

10. Joints. Nervous Affections of.—**SYNON.**: Hysterical Joint; Neuralgia of Joints; Arthralgia; Fr. *Arthralgie Hystérique*; Ger. *Gelenkneurose*.

DESCRIPTION.—*Hysterical affections* present symptoms simulating real joint-disease so closely that the most energetic therapeutic measures have often been resorted to, though in vain, for their cure. Prolonged immobilisation, blistering, the actual cautery, resection, and even amputation, have been practised upon joints in which there was not a trace of organic disease.

The existence of hysterical affections of joints is denied by some; but assuredly they do occur; and most often in young women, well-to-do in life, with disordered catamenia. The same thing occurs, but less frequently, in young men. The disease is not witnessed under the age of puberty. The hip and knee are the joints principally complained of—most frequently the latter. An all-important feature of an hysterical joint is that, while the local symptoms may be intense, the general symptoms are either absent, or in no sort of proportion to the local.

A special character of this disease is that deep pressure is often less painful than superficial pressure; and that the pain and tenderness are vague, shift from one point to another, and will disappear at a given spot when the patient's attention is directed elsewhere. There is pain on movement, but of an indefinite character, and not so limited or localised as in real disease. Nocturnal startings do not occur; the patient may enjoy uninterrupted sleep for hours. There is never a continuous rise of temperature, either general or local; the correlation of the symptoms is not the usual one; the function of the joint is much more interfered with than the other features of disease present would appear to justify. There is an exaggerated fear of examination; and the *facies hysterica* is often well marked. There may be thickening around the joint, and even marked synovial effusion into it; but these conditions are passive in character, and generally due to the treatment employed. The limb is wasted and consequently weak, but never to the same extent as in real joint-disease. Exacerbations occur at the menstrual period. A careful inquiry should be made into the history and antecedents of the case. An examination under chloroform will often afford important evidence; and the patient's symptoms will be improved afterwards by the movements then practised on the joint.

Neuralgic pain in the articulations may arise under different circumstances. It may be the referred pain, unattended by local lesion, which is so frequent in the knee in cases of hip-joint disease. Neuralgic pains in various joints are observed in the preliminary or early stages of chronic myelitis. In the first stage of locomotor ataxy the knee may be affected by severe neuralgia when the disease is low down in the cord; or the shoulders when it is at a higher point. Lastly, so-called neuralgia of a joint may really indicate some obscure lesion, as chronic inflammation of the bones entering into the formation of the articulation.

TREATMENT.—The methodical exercise of an *hysterical joint* is as plainly indicated as rest is imperative in a case of organic disease. The bowels should be regulated, as also the menstrual flow.

Asafetida, iron, and quinine are most important remedies; and healthy mental and moral influences are valuable adjuncts. 'Get up and walk' is a good prescription in many such cases. Very careful and repeated examination should always be made, to exclude any possible form of chronic inflammation, before pronouncing a joint to be hysterical. It must not be forgotten, however, that after slight injuries which produce some inflammatory symptoms, those of hysterical joint may supervene, and persist long after all traces of organic disease have disappeared.

The treatment of *neuralgia* connected with a joint will necessarily vary with its cause. See NEURALGIA.

II. Joints, Chronic Rheumatoid Arthritis of.—SYNON.: Osteo-arthritis; Fr. *Arthrite avec Usure des Cartilages*; *Arthrite Sèche*; Ger. *Alters-abschleifung*.

ETIOLOGY AND PATHOLOGY.—The number of names that have been applied to this disease betrays the obscurity enveloping its pathology. In nature it is, however, essentially a senile degeneration, accompanied by chronic inflammation; and is, in part, perhaps, the result of wear and tear of the joint. It is most common in hard-working people, exposed to the influence of wet and cold, and in the aged. One or many joints may be affected; generally the fingers, the toes, the hip, and the knee. It may be set up by injury, such as a sprain, dislocation, or fracture; or it may arise without known cause. It is difficult to say which of the tissues is primarily at fault, but sooner or later all become involved. The synovial membrane inflames; papillary outgrowths form upon it; the cartilage swells; and the ends of the bones enlarge. After a time the quantity of synovial fluid diminishes; the joint-friction increases; the cartilages are rubbed away at the surfaces of contact; and finally the bone itself, which becomes denser by interstitial deposit, disappears. The surface is eburnated, and marked with striæ produced by friction; while deposits of new bone, which may often be felt externally, form around the margins of the joints, so that the area of its surfaces becomes greatly increased.

SYMPTOMS.—The symptoms of chronic rheumatoid arthritis chiefly consist in constant pain, of a dull aching character, and worse at night. Motion becomes more and more difficult and painful as the disease advances; but ankylosis never occurs. Rough crepitus is felt both by the patient and the surgeon when the joint is moved. See RHEUMATOID ARTHRITIS.

TREATMENT.—The treatment of osteo-arthritis can only be palliative. It consists in the use of warm douches and other warm applications, and the administration of iodide of potassium internally. The disease is incurable.

Charcot's Disease.—This is a peculiar form of osteo-arthritis, affecting usually the larger joints, such as the knee, hip, and shoulder, and occurring in connection with *tabes dorsalis* and *syringomyelia*.

In many instances the changes in the joint are similar to those in osteo-arthritis, while in others there is much more rapid and extensive destruction of bone. Besides the destructive changes which occur in the joint, there is often abundant prolifera-

tion and ossification of the cartilage-margins, so that new irregularly formed masses of bone are found, it may be to as great or even a greater extent than in osteo-arthritis.

SYMPTOMS.—The malady occurs as a rule somewhat early in the progress of a case of *tabes*. The onset is rapid, the synovial effusion greater than in osteo-arthritis, and the wasting and wearing away of the cartilage and bones much quicker.

The swelling generally occurs in the affected articulation quite suddenly and without pain; there is usually no pyrexia or local evidence of inflammation, but the joint may be red and oedematous. Most commonly the knee is affected. The general symptoms of inco-ordination may be strongly marked or altogether absent. It is well to carefully look for them in all cases of extensive chronic enlargement of a joint in the adult. See *TABES DORSALIS*.

TREATMENT.—The treatment consists in giving the affected joint rest, and in the employment of suitable remedies to control the progress of the nervous trouble. The prognosis in most cases is very unfavorable.

12. Joints, Serous Effusion into.—SYNON.: *Hydrops Articulii*; *Hydarthrosis*; Fr. *Hydarthrose*; Ger. *Gelenkwassersucht*.

This is a form of chronic serous synovitis, in which there are no obvious inflammatory symptoms. The joint sometimes becomes greatly distended; the ligaments are stretched; and in consequence there is a sensation of tension and feebleness in the articulation. The knee and elbow are most frequently attacked; and the disease is often associated with a gouty or rheumatic diathesis, or with rheumatoid arthritis. It is very difficult to cure. The joint may be punctured and the fluid drawn off; or, still better, it may be injected and thoroughly washed out with iodine (equal parts of the tincture and water), or with a carbolic acid ($2\frac{1}{2}$ to 5 per cent.) solution. Relapses, however, are common.

13. Joints, Syphilitic Disease of.—This disease may originate in children, in the form of a suppurative osteitis at the junction of the epiphysis and diaphysis. Other signs of congenital syphilis will help to establish the diagnosis. The disease runs a rapid course, and the joint is frequently destroyed. In the adult a chronic plastic synovitis, due to gummatous infiltration of the peri-synovial tissue, or of the bone and periosteum, is the more common form. There is very little fluid effusion within the joint, but considerable impairment of mobility is produced by the thickening outside it. The progress of the disease is slow and painless, except at night or on motion. The history of the case; the presence of traces of syphilis elsewhere; and the effects of treatment, will help in establishing the diagnosis. The internal use of mercury and iodide of potassium, combined with local pressure by means of strapping with mercurial plaster, speedily effects a marked improvement and cure.

WILLIAM MAC CORMAC.

JUGULAR VEINS. See PHYSICAL EXAMINATION; THORACIC ANEURYSM; MEDIASTINUM, Diseases of, &c.

K

KALA-AZAR (Sans. *Kala*, black or deadly; Assamese (?) *Azar*, sickness).—Kala-Azar, or the black disease of the Garo Hills, is epidemic in Assam and in the country between the Brahmaputra and the Garo Hills. It takes the form of a chronic and relapsing infective fever, causing profound and progressive anæmia, wasting, and dropsy. It usually affects several members of the same household, and has a high mortality.

SYMPTOMS.—Ross divides the various manifestations of the disease into three stages, viz.: (1) that of recurrent attacks of high fever with rapid enlargement of the liver and spleen; (2) that of great enlargement of these organs with constant low fever; and (3) that of 'cachexia.'

1. The early stages cannot be distinguished from ordinary malaria. The fever may be first remittent, and afterwards intermittent. Rigors are frequent or absent. In many cases the fever may cease for long intervals, and then be followed by relapses; in others, the preliminary febrile attacks are followed by slight continuous fever, with occasional exacerbations. The subsequent course of the affection usually follows one of two distinct types: one accompanied by general wasting with enlargement of the spleen and liver, and the other by the same symptoms together with dropsy of the legs and abdomen; the latter is the rarer variety. Anæmia and emaciation set in early, the feet become œdematous, and the skin assumes a leaden hue.

2. During the second stage the enlargement and tenderness of the liver and spleen persist, with constant low fever, relieved by occasional exacerbations. The diurnal temperature-curve remains constant, and the amplitude is not great. The anæmia remains well-marked; emaciation is not always extreme. Ascites is nearly always present, and sometimes dropsy of the feet and face. Epistaxis is common. Towards the end of this stage, if the patient survive, the continued enlargement of the organs ceases, tenderness disappears, and the fever declines.

3. The commencement of this stage marks the point after which recovery is impossible. The fever may become changed into a subnormal temperature. The ascites increases, though the dropsy of the feet lessens. The anæmia is not very extreme. Inter-current affections of the lungs or bowels may end the case, or death may result from asthenia.

DURATION.—According to Rogers the usual duration is from four to nine months, the extremes being two months to three years. Ross puts the first stage at one to two months only, and the beginning of the third stage at nine months or a year from the commencement of the illness.

MORBID ANATOMY.—There is great *wasting* of the extremities, with *ascites* and sometimes *dropsy* of the feet and legs. The *skin* is dark. The chief changes are found in the *liver*, which may be twice its normal size, harder than usual, and showing an excess of interlobular tissue. The *spleen* is also enlarged, averaging 2½ lbs. (21 cases, Rogers). The *supra-renal capsules* are normal. The *yellow marrow* is converted into red, is softer than usual, and free from the normal small spicule of bone. The *heart* is in a state of granular and fatty degene-

ration. The *small intestine* is thinned and pigmented.

ÆTIOLOGY AND PATHOLOGY.—There has been much controversy concerning the causation and nature of this disease, but only two views need be mentioned here. The first is that the disease is a form of ankylostomiasis, the other that it is a manifestation of malarial disease. There is no doubt that the large proportion of patients affected by this disease also suffer from ankylostomiasis; but that this is the causative condition is rendered extremely improbable by the facts that 67 per cent. of the presumably healthy inhabitants in affected districts suffer from ankylostomiasis; that fever is uncommon in the latter disease; that the death-rate is much lower; and that the anæmia differs in the two diseases. In kala-azar the sp. gr. of the blood is but little reduced, there is generally leucopenia (see p. 156), and the colour-index of the red corpuscles is about normal.

The malarial theory of the disease is maintained by Rogers, who believes it to be an exceptionally virulent form, and by Ross, who regards it as ordinary malaria. The disease certainly occurs in malarious districts, and the symptoms and pigmentation are identical with those of malarial fever; but, on the other hand, the death-rate is high; the symptoms are less constant; the disease is probably communicable from the sick to the healthy, and quinine is powerless in its treatment. The disease is endemic in places where the inhabitants suffer largely from both ankylostomiasis and malaria, and it is possible that in kala-azar there is some additional source of infection not yet discovered.

COMMUNICABILITY.—Kala-azar spreads along the lines of traffic. People living in a house in which other cases are present, or visiting friends sick of the disease, are especially liable to contract the disorder, but only if they reside for some time with infected persons. Ross holds that the disease is only communicable by the mosquito as in ordinary malaria.

TREATMENT.—In consequence of the large number of cases in which ankylostomiasis exists the dejecta of all patients in localities where ankylostomata are present should be examined for the ova of these worms as a routine duty (see ENTOZOA). If the parasites of malaria are present give quinine in large doses, but when they have disappeared, only small doses, the patient now requiring tonics, and nourishing food, especially milk. These small doses of quinine should be given for weeks or months continuously after an attack of malarial fever.

If the spleen is much enlarged the continued use of quinine and sulphate of sodium is indicated.

Preventive Measures.—Bearing in mind that the disease is probably a communicable one, Rogers recommended: (1) that all newly imported coolies should be placed in fresh huts, and not allowed to enter old infected ones. (2) In slightly infected huts all infected persons, together with their households, should be moved out into separate segregation-lines, and the huts which they had inhabited should be burnt and not rebuilt. (3) In badly infected lines all the healthy people should be moved out during the dry cold weather, when infection is at a

minimum, and placed in new lines, while the infected persons and their households should be segregated, and the infected lines abandoned.

As far as tea-gardens are concerned, this practice has been attended with success. Thus in a certain tea-garden in which 200 coolies were lost in the course of four years from the disease, new huts, a quarter of a mile from the old ones, were built, the old ones burnt down, and the coolies moved into the new ones. The epidemic ceased. In another case all the new coolies were placed in new huts and no new case occurred, whereas cases arose among the old coolies in the old lines.

ANDREW DUNCAN.

KAPOSI'S DISEASE.—See XERODERMIA PIGMENTOSA.

KATATONIA (κατά, down; τόνος, tension).—See INSANITY (Mental Stupor).

KELOID.—See CHELOID.

KERATINISATION.—The name applied by Unna to the process by which pigmentation occurs in xeroderma and ichthyosis, believed by him to consist of a partial loss of water and oxygen, and a consequent relative increase of sulphur.

KERATITIS (κέρας, a horn, the cornea).—Inflammation of the cornea. See EYE, AND ITS APPENDAGES, Diseases of.

KERATODERMIA.—See TYLOSIS.

KERATO-HYALIN.—The substance, the exact nature of which is unknown, deposited in the cells of the deep Malpighian layer of the skin as they become changed into horny cells. See KERATOSIS.

KERATOSIS.—The process by which the soft cells of the Malpighian layer of the epidermis are converted into horny cells, a process accompanied by the deposition of kerato-hyalin in their interior.

KERION (κηρίον, a honeycomb).—SYNON.: *Tinea Kerion*; *Kerion Celsi*.—A term applied to a pustular folliculitis of a scalp affected with ringworm. The inflamed area occurs in the form of one or several blotches of a deep red colour, prominent, and dotted over with yellow spots—the apertures of the empty hair-follicles, which exude a copious muco-purulent fluid. The yellow spots are converted into tumid hollows of distended follicles by the swelling of the inflamed skin around them. Another feature of the disease is the actual destruction of the hair-follicles, and the subsequent baldness of the affected part. The production of an 'artificial kerion' by the application of croton oil to the scalp is one of the methods of treatment of tinea tonsurans. See TINEA TRICHOPHYTINA.

JOHN HAROLD.

KERNIG'S SIGN.—A temporary rigidity occurring in the flexors of the knee which can be induced in all cases of spinal meningitis by flexing the thigh to a right angle. In this position the leg cannot be fully extended upon the thigh.

KIDNEY, Diseases of the.—The diseases of the kidney will be considered in the following order:—

Abscess, p. 821.

Amyloid Disease, p. 821.

Anomalies, p. 822.

Atrophy, p. 823.

Consecutive Inflammation, p. 823.

Cystic Disease, p. 827.

Embolism, p. 828.

Fatty Disease, p. 829.

Granular Kidney, p. 829.

Hæmorrhage, p. 831.

Hydatid Disease of, p. 831.

Hyperæmia, p. 832.

Hypertrophy, p. 833.

Inflammation of, p. 833.

Inflammation of Pelvis of, p. 834.

Malformations, p. 822.

Malpositions, p. 835.

Morbid Growths, p. 836.

Parasites, p. 838.

Syphilis of, p. 838.

Thrombosis of, p. 838.

Tuberculosis of, p. 838.

The following is a list of articles on cognate subjects which appear in their alphabetical position in other parts of the Dictionary:—ACTINOMYCOSIS; ALBUMINURIA; ALBUMOSURIA; BRIGHT'S DISEASE; CONCRETIONS; CHYLURIA; DIABETES MELLITUS; DIURETICS; DROPSY; HÆMATURIA; HÆMOGLOBINURIA; LITHONTRIPTICS; NEPHRALGIA; OXALIC-ACID DIATHESIS; PAIN IN VISCERAL DISEASE; PERINEPHRITIS; PHOSPHATURIA; POLYURIA; RENAL CALCULUS; URÆMIA; URINARY DEPOSITS; URINE, Morbid Conditions of.

GENERAL CONSIDERATIONS.—The effects produced by renal disease may generally be classified under three heads: (1) the local effects produced in the kidney itself; (2) the changes produced in the composition of the urine; and (3) certain remote effects produced on the body at large.

1. *The local effects*, such as the production of a tumour or the development of cysts, or the presence of one or more of the varieties of renal pain, will be discussed under the headings of the individual diseases.

2. *Changes in the Urine.*—The urinary changes produced by renal diseases will be discussed in the article URINE, Morbid Conditions of.

3. *Effects produced by Renal Disease on the Body generally.*—These may be classified as follows:

(1) *Nutritional.*—The nutrition of the body generally suffers in renal disease, partly owing to the interference with digestion and absorption, but apparently also indirectly owing to an action of the renal tissue on the metabolism of the body generally, inasmuch as the experimental removal of a large amount of the renal tissue is followed by a condition of extreme wasting accompanied by the excretion of a copious urine containing an increased quantity of urea. In renal disease wasting is often a prominent feature, quite out of proportion to any gastric disturbance, nausea, vomiting, or diarrhoea produced by the malady. In granular kidney and other destructive diseases of the renal substance wasting is a prominent feature. Further the temperature of the body is often subnormal in renal disease.

(2) *Resisting power.*—The resistance of the tissues to microbic infection in renal disease is undoubtedly diminished, and this is one of the reasons why such patients are so liable to secondary inflammations. The most common secondary infections in renal diseases are pericarditis, pleurisy, peritonitis, and pneumonia.

(3) *Dropsy*.—Dropsy is a prominent feature in some forms of renal disease, but is not present in all, being more especially characteristic of Bright's disease in its acute form and in one form of chronic Bright's disease, the so-called large white kidney. It is less frequently seen in the granular kidney, the contracted white kidney, and cystic kidney. Patients with renal disease may suffer not only from renal dropsy but also from cardiac dropsy owing to the development of secondary cardiac complications. Dropsy in renal disease is invariably accompanied by a diminution in the quantity of urine excreted; but this is not its cause, as is shown by the fact that complete suppression may occur in a number of renal diseases without the development of dropsy. Therefore it is quite possible that, even in acute Bright's disease and chronic Bright's disease where dropsy is such a prominent feature, this symptom does not owe its origin to the mere diminution in the quantity of urine excreted. It is more probable that the scanty excretion of urine is the result of the dropsy than its cause. Renal dropsy is characterised by involving the subcutaneous tissues, especially the scrotum, the sacrum, and the subcutaneous tissues of the face. Where it is large in amount there is also dropsy in the serous cavities and even the solid organs, more especially the lungs and the brain. The dropsical fluid in renal disease contains only a small amount of proteid matter, often not more than 1 per cent., but large quantities of nitrogenous extractives, especially urea.

(4) *Cardio-vascular lesions*.—In many renal diseases the heart, especially the left ventricle, undergoes hypertrophy, and the arteries, not only of the kidney but of the body generally, are thickened by changes in all their coats. The sub-endothelial coat presents the most characteristic changes, owing to a very great increase in the amount of elastic tissue and the development of loose fibrous tissue between the elastic laminae. It is not unusual for the inner coat to equal in thickness the middle and the outer. The thickening of the inner coat is not uniform, and frequently involves only one portion of the circumference of a vessel. The larger arteries have their adventitia increased in amount, and the medium-sized arteries, in addition to presenting the changes described above, may have a well-marked increase in their muscular coat. The changes in the inner coat are often most marked in the vessels of the kidney itself, but considerable variations are found in the degree of the degeneration in the renal vessels and in those of the body generally. These arterial lesions are limited to cases of chronic renal disease, and are best marked in chronic Bright's disease and in the granular kidney.

The degree of development of cardiac hypertrophy and of the arterial lesions is not entirely dependent on the diminution in size of the kidney, as extreme forms of contracted white kidney and even of the granular kidney are seen where the cardiac hypertrophy is small in amount and the arterial lesions less developed than in other cases where the renal lesion is less marked.

Speaking generally the arterial lesions are such as may be correlated with the high pressure. Some pathologists have thought that the arterial lesions are primary and that the cardiac hypertrophy is induced secondarily to them, others that the cardiac hypertrophy is primary and that the arterial lesions are such as would be produced as a result of the

increased arterial pressure. Arterial lesions may often be detected on ophthalmoscopic examination by the undue rigidity and thickness of the retinal arteries. These arterial lesions are not seen usually in such destructive diseases as cystic kidney and hydronephrosis, notwithstanding that the quantity of renal tissue is greatly diminished. This affords a further reason for thinking that their production is not entirely dependent on mere destruction of renal tissue.

(5) *Uræmia* is the name given to the toxic state liable to supervene in almost all forms of destructive renal disease, and is considered elsewhere. See URÆMIA.

KIDNEY, Abscess of.—Suppuration in the substance of the kidney may exist in the form of a number of diffused abscesses of small size, or as a single abscess of larger size. The former condition is most usually seen in so-called 'surgical' kidney (see KIDNEY, Consecutive Inflammation of), the latter as the result of infective embolism. Infective embolism may, however, give rise to multiple abscesses of small size as in malignant endocarditis. Where a single abscess is present without known cause, it is generally the result of infection from some other part of the body. An abscess in the kidney may either rupture into the pelvis and so be spontaneously evacuated, or it may perforate the capsule and form a perinephric abscess. See PERINEPHRITIS.

Renal abscess may be suspected and diagnosed if, some days after the occurrence of a rigor and renal pain, a considerable quantity of pus suddenly appears in the urine. Cases of single abscess of the kidney which rupture into the renal pelvis frequently recover completely. Where the abscesses are multiple the prognosis is more serious owing to the underlying condition.

KIDNEY, Amyloid Disease of.—**ÆTIOLOGY AND MORBID ANATOMY.**—See AMYLOID DISEASE.

SYMPTOMS.—The onset of the disease is gradual, and frequently the first manifestation of the disorder is the passage of an increased quantity of urine. The urine is of low specific gravity, and it is stated that at the actual onset of the disease it is free from albumen. Soon, however, albumen is present in variable amount, but usually where the quantity of urine is greatly increased the percentage amount of albumen is small, although greater than that seen in the granular kidney. One hundred ounces of urine may be secreted in the twenty-four hours, and cases have been described where two hundred ounces have been passed. In such cases the quantity of urea secreted in the twenty-four hours is not markedly diminished; the urine contains but few casts, and these are usually hyaline. In another form of the disease the clinical picture is quite different; dropsy is a marked feature, often resembling in its distribution the cardiac form of dropsy more closely than that seen in Bright's disease. Cases of amyloid disease, however, may be seen where the general subcutaneous dropsy is marked in amount and very similar in distribution to that of Bright's disease. In such cases the quantity of urine is diminished, and, although its specific gravity may still remain low and its colour pale, such urine may contain large quantities of albumen. All gradations are seen between cases where a copious urine containing but a trace of

albumen is secreted and those in which the amount of urine is small but loaded with albumen. In the latter form of the disease granular and hyaline casts are more abundant, and in some instances casts giving the amyloid reaction with iodine have been detected. The duration of both forms of the disease is uncertain, as it is so largely dependent on the course of the original malady producing it, but the latter variety may last for as long as two years, and the more chronic for six. The other symptoms produced may be largely masked by those dependent on the original malady, but anæmia and wasting are usually marked features, and uræmia is by no means uncommon as a fatal complication. The fatal issue is more dependent on the original disease than on the amyloid degeneration of the kidneys produced by it. In some instances changes in the fundus oculi similar to those seen in chronic Bright's disease occur.

DIAGNOSIS.—The condition often resembles one of the various forms of Bright's disease. The existence either of polyuria with slight albuminuria, or of scanty urine with marked albuminuria and dropsy, without any signs of cardiac enlargement or arterial thickening or degeneration, is most suggestive of amyloid disease, especially if the spleen and the liver be enlarged. In cases of pyelitis the existence of marked albuminuria out of proportion to the amount of pus in the urine is very suggestive of the existence of this complication. It is important to recognise the presence of amyloid degeneration in cases of pyelitis, as the existence of this renal complication may materially influence the prognosis and the results of any operative interference for the relief of the pyelitis. It is not uncommon for fatal uræmia to occur as a result of surgical exploration when the kidneys are affected with amyloid disease as a complication of pyelitis.

The prognosis depends very largely on that of the disease causing this complication. In some instances the successful treatment of the underlying malady is followed by the disappearance of the amyloid disease, and in nearly every case by its arrest.

TREATMENT.—The treatment is similar in most respects to that of Bright's disease, except that, unless complications such as uræmia or dropsy are present, the disease requires a more liberal diet than that ordered in Bright's disease, as it is essential, owing to the existence of a debilitating malady such as suppuration or bone-disease, that a nourishing diet should be given. Further, the amyloid disease, unless very far advanced, is not one which greatly interferes with the excretory powers of the kidney, and there is not therefore the reason for diminishing the nitrogenous ingesta as is so often necessary in other renal diseases. *See* AMYLOID DISEASE.

KIDNEY, Anomalies of.—Kidneys may present three kinds of anomaly: in number, in form, or in position, although two or more of these are frequently combined. *See* KIDNEYS, Malpositions of.

Anomalies in Number.—Both kidneys may be absent during foetal life, but as this condition is incompatible with the maintenance of life, it is only of developmental interest. One or more supernumerary kidneys may be present, but this anomaly is very rare. One kidney only may be present. This may depend on the fusion of two originally

distinct organs, or on the absence or atrophy of the second. If one organ is congenitally absent, no traces of the renal vessels or of the ureter are found on that side; but if it has undergone atrophy during foetal or early extra-uterine life, a remnant of varying size is always present. In the congenital cases the atrophied kidney is frequently found to have been developed in an anomalous manner, so that the glomeruli or the convoluted tubules may be absent. When the atrophy has resulted from pathological changes occurring after birth, the renal artery may be found occluded or the ureter obstructed; and the atrophied kidney will contain remains of altered convoluted tubules and glomeruli, together with a large amount of fibrous tissue. Where the atrophy is of congenital origin, the single kidney that is present is often considerably above the weight of two normal kidneys. In some cases these single kidneys may weigh as much as a pound. The presence of a single large kidney is of no great clinical importance, except that it materially increases the gravity of certain renal diseases such as calculus; such kidneys are, moreover, not uncommonly affected by Bright's disease. In some instances the existence of a single large normal kidney in a patient complaining of abdominal symptoms has been erroneously diagnosed as a renal tumour.

A single kidney resulting from the fusion of two primary separate organs will be considered under anomalies in form.

Anomalies of the renal vessels, especially of the arteries, are very common, and are of some importance to the surgeon. They may lead to the production of hydronephrosis, by causing obstruction to the ureter. Anomalies of the ureter may also lead to the production of hydronephrosis. *See* HYDRONEPHROSIS.

Anomalies in Form.—The commonest malformation of the kidney is for the *lobulation*, which is so characteristic of the foetal kidney, to be more marked in the adult than usual. In cases in which the lobulation is extremely well marked the organ is often malformed in other ways, so as to be considerably longer than normal. It is not uncommon for one extremity of the kidney, especially the lower, to show marked lobulation, and in some instances a portion of the lower extremity may be partially separated from the remaining kidney substance. The *horse-shoe kidney* is also not uncommonly met with. In this condition the two kidneys are united by a bridge of renal tissue, usually joining their two lower extremities. The kidney generally is ill-shapen, and lobulation is well marked. In cases of horse-shoe kidney the arrangement of vessels is anomalous, a number of arteries entering the concave margin of the kidney and also the anterior surface. The ureters usually pass down over the front of the kidney. In some instances the horse-shoe kidney is really spurious, the bond of union being composed of fibrous tissue.

Other instances of malformation of the kidney are afforded by the union of the two kidneys end to end, producing the so-called 'sigmoid kidney.' In very rare instances the two kidneys are fused by their surfaces, forming what has been called the 'disc-shaped kidney.' In both these rare malformations the two ureters are found on one side of the body, but they are distinguished from the condition of double ureter by the fact that one crosses to the opposite side, and they both open in the normal positions in the bladder.

Malformations of the ureter and renal pelvis are not uncommon. A double ureter may sometimes exist, but more often an apparently double ureter is really the result of the non-union of two of the calices. The pelvis of the kidney may be uncommonly large and join a normal or unnaturally small ureter. Cases are sometimes seen in which the ureters and the pelvis of the kidney are dilated and the kidney-substance itself malformed, the appearances being such as to resemble those of hydronephrosis. Some such instances are dependent on congenital obstructions in the lower part of the urinary tract and even on phimosis, but instances occur where no cause for the dilatation of the pelvis and ureters can be found.

Malformations are of no great clinical importance except in so far that certain of them predispose to disease, e.g. anomalies of the pelvis and ureters to hydronephrosis, and dilatation of the ureter to pyelo-nephritis. The presence of such anomalies as a horse-shoe kidney or single fused kidney may lead to errors of diagnosis, from the detection of a mass which is erroneously interpreted as a new formation. Further, in rare instances, a horse-shoe kidney has exerted sufficient pressure on the vena cava to cause thrombosis of this vessel.

KIDNEY, Atrophy of.—This term is best restricted to a condition where the diminution in the size of the kidney is the only morbid process. The extreme forms of atrophy of the kidney involve only one organ. In such cases the kidney may still retain its normal shape, but may not be more than half an inch in its longest diameter. All intermediate sizes between this and the normal may be found.

At least three forms of atrophy may, therefore, be recognised: (1) of developmental origin; (2) from arterial occlusion; (3) from obstruction of the ureter. The atrophy is often of congenital origin, and associated with some anomaly in development (*see above*). The renal artery in some cases is exceedingly narrow, either throughout its whole course or at one spot only, and atrophy thus seems to result from diminution in its calibre. Obstruction of the ureter will also account for some cases, as there is experimental evidence to show that if the hydronephrosis produced by obstruction of the ureter be drained, a very perfect form of atrophy of the kidney results.

The tubules in cases of atrophy of the kidney frequently contain epithelium, which has undergone hyaline degeneration, and is of glass-like appearance. A similar change in the epithelium is seen when the atrophy is produced experimentally by ligation of the ureter.

If the term 'atrophy' be used in its more general sense, atrophy of the kidney is seen in hydronephrosis, in the granular kidney, in the contracted form of Bright's disease, and occasionally even in the amyloid kidney. In all these conditions the diminution in the size of the organ is largely dependent on the contraction and collapse of great numbers of the renal tubules.

Partial atrophy of the kidney is a term applied to the diminution in the size of the organ which follows embolism—the absorption of a necrotic infarct often causing a considerable diminution in the size of the organ accompanied by much scarring.

JOHN ROSE BRADFORD.

KIDNEY, Consecutive Inflammation of.—Under this term are included the various patho-

logical conditions which may affect the kidney as the result of disease or injury of the lower parts of the urinary tract. The very inappropriate name 'Surgical Kidney' is very frequently applied to that form of consecutive inflammation of the kidneys in which scattered foci of suppuration are found in one or both organs.

PATHOLOGY.—Diseases of the lower urinary tract may react upon the kidneys in two important ways: (1) by obstructing the passage of the urine, and so causing abnormal tension in the whole urinary tract above the obstruction, and (2) by the extension of infective processes to the kidneys from the bladder. Further, it is possible that certain phenomena occasionally accompanying affections of the lower parts of the urinary tract may result from reflex disturbances of the renal circulation.

1. Obstruction to the passage of urine may occur in the ureters from congenital malformation, from impaction of a calculus, or from the pressure of a tumour. It may occur at the vesical orifice of the ureter from thickening of the wall of the bladder in hypertrophy, from thickening and induration of the submucous tissue and swelling of the mucous membrane in chronic cystitis, or from the growth of a tumour around the orifice. Obstruction to the passage of urine from the ureters into the bladder also results from any condition in which the bladder is unable completely to empty itself, as in hypertrophy of the prostate and in certain forms of paralysis. Stone in the bladder causes obstruction at the orifices of the ureters by the chronic cystitis to which it gives rise, while stricture of the urethra acts in the same way and by occasioning muscular hypertrophy of the bladder-wall.

In the large majority of cases, whatever be the exact nature of the obstruction, the valvular orifices of the ureters do not become incompetent, and the only force concerned in the dilatation of the ureter and pelvis of the kidney is the force of the renal secretion. Exceptionally, however, if the bladder is thin and dilated, or the vesical orifice of the ureter is enlarged by ulceration, regurgitation of urine from the bladder into the ureters may occur.

2. The acute inflammation of the kidneys which so frequently determines the fatal issue in diseases and injuries of the lower urinary tract is due to the infection of the kidneys by pathogenic micro-organisms. This renal infection is in the large majority of cases preceded by the occurrence of cystitis, and one or more of the organisms associated with this condition are present in the urine. Of these organisms, the most important, especially in so far as the secondary renal infection is concerned, is the *Bacillus coli communis*. But other pyogenic organisms may be present, and very frequently the infection is a mixed one (*see PYOGENIC BACTERIA*). The pyogenic organisms are often associated with others, such as the *Micrococcus ureæ*, causing decomposition of urea with ammoniacal change in the urine. This condition of the urine is often present, but the organisms which cause it are not to be regarded as essential elements in the secondary infection of the kidneys. It is undoubtedly true that ammoniacal urine may be found in the pelvis of the kidney, but secondary nephritis may occur in cases in which the urine has throughout been acid in reaction.

The most common route by which the infection of the kidney takes place is the lumen of the ureter. From what has been said above it will be

seen that it is only in exceptional cases that the infection is due to actual regurgitation of infected urine from the bladder into the ureter; in the absence of such regurgitation invasion by micro-organisms is doubtless favoured by the existence of catarrhal inflammation of the pelvis of the kidney and of the ureter, in consequence of which threads of mucus may occupy the vesical orifice of the ureter; along these the organisms find their way to the upper urinary passages, finally extending into the substance of the kidneys, where they may be demonstrated in the renal tubules.

Secondly, the infection of the kidneys from the lower urinary tract may occur by way of the lymphatics. Lindsay Steven long ago demonstrated by injection the continuity of the lymphatics of the bladder with those of the ureter, and these again with lymphatics in the superficial part of the cortex of the kidney and beneath the capsule; and in a fatal case of cystitis he demonstrated the presence of micro-organisms in the whole length of this lymphatic tract. Such a mode of infection may serve to explain certain cases of secondary suppurative nephritis not accompanied by pyelitis.

Thirdly, suppuration in the kidney secondary to an infective process in the lower urinary tract may result from a blood-infection, and thus be pyæmic in nature. Such a renal infection is in no way different from that which may follow infective processes not occurring primarily in the urinary tract, and will not be further considered here.

Certain phenomena occasionally arising as complications of various injuries and diseases of the lower urinary tract were at one time attributed to reflex disturbances of the renal circulation. Among the phenomena especially liable to occur after such procedures as the passage of a catheter or the dilatation of a stricture, which have been explained in this way, are: (1) fatal suppression of urine associated with intense congestion of the kidneys; (2) temporary arrest of the secretion of the kidneys followed by hæmaturia; and (3) the occurrence of rigors. Although the possibility of the occurrence of a reflex disturbance of the renal circulation in such cases has not been disproved, there is increasing evidence in support of the view that these rapidly developed and often fatal complications are the result of a general infection or toxæmia. As a striking instance of such a rapid infection, may be mentioned a case recorded by Albarran. A young man, upon whom internal urethrotomy had been performed for stricture of the urethra, had a violent rigor one hour and a half after the operation, the temperature rising to 104.4° F. Further rigors occurred, and the patient died twelve hours after the operation. The *Bacillus coli communis* was found in the blood, in the whole length of the urinary tract, and in the different organs. The congested state of the kidneys may in such cases be the result of a general toxæmia, and not, as formerly supposed, of a simple reflex disturbance.

ANATOMICAL CHARACTERS.—The consecutive inflammations of the kidneys are met with in four forms.

I. Chronic interstitial inflammation, followed by absorption of the medullary portion, and later by stretching and thinning of the cortex, without pyelitis.—This condition is the uncomplicated effect of obstruction to the free flow of urine, and of the consequent increased urinary pressure. It is

most frequently met with in cases of pressure on the ureter from without. In diseases of the bladder and urethra it is almost always complicated by an acute attack of interstitial inflammation, which is the immediate cause of death, and which more or less conceals the appearances about to be described. In the early stage there is slight dilatation of the ureter and pelvis of the kidney. The kidney itself is increased in size; the capsule separates without difficulty, but may leave the surface somewhat wanting in its natural smoothness. The venous stars on the surface are often clearly marked, the cortical substance being of a pale pinkish white or sometimes yellowish colour. On section the cortex is found to be wider than natural. The medullary portion is usually pale, like the cortex, but the large veins at the cortico-medullary junction are often distended with blood. The kidney-substance is tougher than natural. The Malpighian bodies can usually be clearly seen, sometimes as red dots. Microscopical examination shows an overgrowth of the interstitial connective tissue, which, especially around the Malpighian bodies, is richly cellular. The epithelium shows no change. The next stage observed is commencing absorption of the medullary portion; the ureter, pelvis, and calices become still more distended; the papillæ are first flattened, and then the pyramids become hollowed out. This is a process of pure absorption, there being no ulceration. The cavity formed by the dilated calyx, and the hollow left by the disappearance of the pyramid, are lined by a continuous smooth layer of opaque white mucous membrane. In the final stages the cortex in its turn becomes thinned and stretched, until at last the whole kidney may be dilated into a large sac, one side of which is smooth, being formed by the thickened walls of the dilated pelvis, the other deeply sacculated, each sacculus corresponding to a lobe of the kidney. On this side the wall is formed of the thinned and stretched cortex, sometimes no thicker than a shilling, to which the capsule, now thickened and opaque, is firmly adherent. In the later stages the microscope shows the same abundant small-cell infiltration, with the development of a greater or less amount of fibroid tissue. The Malpighian bodies show marked changes. The capsules, instead of being delicate and membranous in structure, become greatly thickened, apparently by dense fibroid tissue formed around them in concentric layers. As this change progresses the vessels may become strangulated and finally obliterated; the glomerulus then shrivels, and is represented by a circular body almost homogeneous in the centre, but marked by a few curved lines indicating the situation of the obliterated vascular tufts. Around this centre is a concentrically laminated layer, formed by the thickened capsule. Even in the most advanced stages the epithelium of the convoluted tubules shows remarkably little change beyond being somewhat flattened.

If at any stage obstruction to the free flow of the urine be removed, the process ceases. The new tissue between the tubules undergoes development into dense fibroid tissue, the process being accompanied by great contraction. The kidney, from being increased in size, may thus become much smaller than natural, excessively tough and puckered, and irregular in form. If the distension have reached the most extreme stage before the primary disease is relieved, the kidney may finally be repre-

sented merely by a small nodule of dense fibroid tissue.

II. *Acute diffuse interstitial nephritis without suppuration*.—In this variety both kidneys are usually affected. The kidney is increased in size, and the surrounding fat is sometimes oedematous and adherent to the capsule. When removed, the capsule separates without difficulty, but often leaves the surface coarse; it is somewhat opaque, and often marked with ramifying vessels. The surface is usually of a pale, yellowish-white colour, often mottled with dark red, or in some cases the red may greatly predominate. The mottling often corresponds to the bases of the lobules of the gland, some of which are paler than others, in consequence of the more advanced condition of the interstitial inflammation. On section, the cortex presents the same colour and mottled appearance as the surface, and is evidently swollen. The pyramids may be pale, but are often dark red, contrasting strongly with the paler cortex. The Malpighian bodies are usually clearly visible, and may show on the cut surface as red dots. The consistence of the kidney is unnaturally soft, unless previously to the acute attack it has been indurated by the chronic process first described. The pelvis may be merely dilated, and its mucous membrane opaque, but more commonly it is marked by ramifying vessels, and presents evidence of chronic congestion, in the form of pigmentation, thickening, and induration. In other cases it is intensely injected, sometimes covered with a membranous exudation mixed with phosphatic deposit, and the urine it contains may be foul and ammoniacal. In consequence of the pallor of the kidney sometimes met with as a result of the emptying of the vessels after death, this form of kidney may, without the microscope, be confounded with the large white or the fatty kidney.

On microscopical examination the following conditions are found. Between the tubules is a very abundant cellular exudation, especially around the Malpighian corpuscles. So far the condition is merely an intensification of that described as resulting from increased urinary pressure. The change is not uniform; in one part the renal structure may appear almost normal, and close by the new cells may be heaped up to such an extent as nearly to conceal the tubules. In the pyramidal portion a similar condition is met with. The renal epithelium throughout is slightly more cloudy than natural, and somewhat swollen, but the nuclei of the cells are readily to be seen in sections prepared in the ordinary way. The adhesion of the epithelium to the membrana propria is somewhat lessened, so that unless considerable care be taken it will wash out in preparing the specimen. Fibrinous casts may be seen here and there in the tubules; and occasionally small round cells, resembling those outside the tubules, may be seen within them.

III. *Acute interstitial nephritis with scattered points of suppuration*.—*Suppurative nephritis or pyelo-nephritis*.—This form of inflammation of the kidney is the usual cause of death in fatal cases of disease of the bladder or urethra, in which infection of the contents of the bladder has occurred. It thus comes to be one of the most common fatal complications in cases of injury or disease of the spinal cord, with paralysis of the bladder. The naked-eye appearances are the following: The surrounding fat may be oedematous and unnaturally adherent to the capsule. The whole kidney is considerably swollen,

and its substance soft. The capsule is opaque and thickened, and marked by fine ramiform injection. It separates easily, but tears the kidney-substance in so doing. As it peels off, yellowish-white spots, surrounded by a red zone, come into view. Some of these are minute drops of pus escaping from the small abscesses as the capsule is stripped off, others are on the point of breaking down into pus, but are still solid. These abscesses are grouped together in areas corresponding to the bases of the lobes of the kidney. If the veins can be recognised, the abscesses will often be seen to correspond to the points at which the interlobular veins appear on the surface. On section, the cortex is seen to present much the same appearance as in the last form of kidney, but in addition yellowish streaks are seen passing from the points of suppuration deeply into the cortex, and often into the medullary portion. These streaks correspond to the course of the interlobular vessels. They differ from embolic infarcts in their great length compared with their breadth. The pelvis is usually in a condition of most intense inflammation; the mucous membrane is often covered with a layer of exudation mixed with phosphates, and the contents, composed of urine, blood, and mucus, are in some cases foul and ammoniacal. Cases, however, do occur in which a similar condition of suppuration in the kidney is met with without pyelitis. In these it is probable that the pyogenic organisms reached the kidney by the lymphatics.

Occasionally the kidney is surrounded by a large abscess, arising from a perforation of the pelvis. More frequently one of the superficial abscesses in the cortex bursts beneath the capsule, and gives rise to a large collection of pus separating the capsule from the kidney.

The microscope shows the small-cell infiltration between the tubules in a still more intense form than in the varieties before described. In the areas of suppuration the kidney-substance has entirely disappeared, and its place is occupied by leucocytes packed closely together. In the central parts of these accumulations of small round cells the intercellular substance has softened, and the formation of pus has taken place. The amount of general interstitial change varies considerably; sometimes between the areas of suppuration the kidney-substance is almost healthy, in other cases there is a very marked general interstitial inflammation. The epithelium shows the same changes as in the last variety.

The micro-organisms which cause these acute inflammations of the kidney are found chiefly in the renal tubules, and in many instances form dense masses completely plugging the straight tubules and even extending into the convoluted tubes of the cortex. The organisms are also found in the lymph-spaces between the tubules in the pale streaks reaching from the medulla to the cortex along the course of the interlobular vessels. In pyæmia the organisms are very commonly seen plugging the looping vessels of the glomeruli, and occasionally some have found their way into the tubules, but in the acute renal inflammations belonging to the group under consideration the organisms are not found in the blood-vessels unless the direct infection of the kidney from the lumen or lymphatics of the ureter is complicated by an indirect infection through the blood-stream.

IV. *The cicatricial kidney*.—This is the result of recovery from one of the preceding conditions—probably only from one or both of the first two, as,

if the disease reach the stage of suppuration, the patient is hardly likely to survive. The kidney is shrunken, irregular in form, and marked by deep cicatrices. The substance is excessively tough, and the capsule firmly adherent. Small cysts may be scattered through its substance, which are supposed to result from strangulation of the tubules. The microscope shows a great excess of dense fibroid intertubular substance, and numerous obliterated glomeruli.

The varieties of kidney here described may be combined in various ways, and the naked-eye and microscopic appearances are modified accordingly. It is in fact more usual than not to find that the acute inflammatory affections occur in a kidney already more or less altered as the result of simple increase of tension. Thus a dilated kidney may suffer from acute diffuse interstitial inflammation, with or without suppuration; or a cicatricial kidney may, from a return of the primary disease, again suffer from an acute attack.

SYMPTOMS.—*Simple chronic interstitial nephritis*, with dilatation of the kidney from increased urinary pressure, gives rise to but few symptoms, and is very difficult to recognise. The most important signs are, that the quantity of urine secreted is increased, and its specific gravity lowered. The whole urine passed in twenty-four hours should be collected, and the specific gravity taken. There may be a trace of albumen, or it may be entirely absent. A few hyaline casts may be present, but they are by no means constant. The exact state of the urine is often concealed by the mucus, blood, and pus from the lower urinary tract. It is surprising how much urine is secreted by a kidney which is reduced to a mere sac, with no pyramids and a cortex no thicker than a shilling. In some cases the distended kidney may be recognised by palpation, but this is not common. It is not accompanied by hypertrophy of the heart, nor by marked increase of the arterial tension.

Although in itself not productive of definitive symptoms, this form of chronic renal disease is of great practical importance as a predisposing cause of such serious complications as rigors and suppression of urine after any operative interference upon the lower urinary tract.

Subacute interstitial nephritis gives rise to more marked symptoms. It runs an irregular course, often lasting for weeks or even months, and terminating either in recovery, or in a final acute attack with suppuration. If the disease arise as the direct result of some operation on the lower urinary tract, its commencement is usually marked by a rigor; in other cases it comes on more gradually, with frequent chills but no actual rigor. The temperature is high at night, reaching 101° to 102° F., but it falls towards morning, so that if it be only taken at that time, the elevation may be completely overlooked. The patient becomes weak and languid, and emaciates rapidly. He loses appetite, and there may be nausea or occasional vomiting. There may be diarrhoea, but this is by no means constant. The mucous membrane of the mouth becomes clammy, and the tongue foul, with a tendency to dryness. In severe cases the tongue becomes dry and brown, and sordes form on the teeth and lips. The skin is usually moist and clammy, and there is not the dryness so frequently met with in other forms of renal disease. There is no oedema. In rare instances the swollen kidney

may be felt by palpation in the loin, and tenderness may be elicited on deep pressure. The patient may complain of pain in the lumbar region, but this symptom is of little value, as it is often absent, and may arise from many other causes than renal disease. The pulse presents nothing characteristic. The patient frequently sinks into a drowsy state, somewhat resembling the effect of an over-dose of opium; but true coma is rarely, if ever, present, and convulsions never occur. The urine is passed in fair quantity, often in excess of the normal amount. The amount of albumen is never very great, but it is usually difficult to estimate accurately how much is renal, and how much is derived from blood or pus from the lower urinary tract. Microscopical examination may show hyaline casts, or occasionally pus-casts; renal epithelium is also frequently met with; but all microscopical examination is rendered difficult by the presence of mucus and pus from the lower urinary tract. If the primary disease be either removed or relieved by treatment, the symptoms gradually subside; if not, they remain without much change till the patient gradually dies exhausted, or an acute attack, rapidly leading to suppuration of the kidney, puts an end to the case.

Acute interstitial nephritis with suppuration most frequently forms the fatal termination of the variety of disease just described, but it may occur without any previous symptoms. The invasion is marked by a severe rigor, often occurring within a few hours of some operation on the lower urinary tract. The rigor is accompanied by great elevation of temperature, and followed by profuse perspiration, during which the temperature falls, perhaps below normal; but it soon rises again, and remains slightly raised, with evening exacerbations. The rigor may be repeated during the progress of the case at irregular intervals. The general symptoms resemble in every respect those just described as indicative of subacute interstitial nephritis, but they are increased in intensity. The strength rapidly fails, there is great emaciation, the pulse becomes feeble, and the tongue 'like a piece of broiled ham.' There may be occasional vomiting and diarrhoea. As the fatal termination approaches, the temperature falls often considerably below normal, the skin becomes cold and clammy, and the patient sinks into a drowsy condition, seldom deepening into actual coma. Although the patient is often said to be dying of 'uræmia,' there are none of the uræmic symptoms observed in acute Bright's disease. There are no convulsions or actual coma, and no oedema. The urine is usually so foul as to defy accurate examination, either chemically or by the microscope. It is secreted in fair quantity to the end of the case. Pus and blood are always found in it, but it is impossible to say whether they come from the kidney or from the lower urinary tract. Renal epithelium and pus-casts are occasionally met with.

DIAGNOSIS.—As already stated, the existence of the more chronic secondary renal inflammations can be determined only by a careful examination of the urine. In the acute forms, especially when accompanied by suppuration in the kidney, the general symptoms may in themselves be indistinguishable from those occurring in many other forms of septic infection or toxæmia. As a rule, however, their association with septic conditions of the lower urinary tract serves to indicate their nature. Severe cystitis without renal infection

may, however, cause marked general symptoms, and the examination of the urine, except by the detection of pus-casts, will not serve to show whether or not the kidneys are involved. The effect of treatment may, however, in such cases indicate whether the symptoms are the result of the cystitis alone. Catheterisation of the ureters and the separate examination of the urine secreted by each kidney has been used as a means of diagnosis, but for many reasons the practical utility of this method must be very limited in cases of this nature.

PROGNOSIS.—This depends, in the chronic or subacute form, entirely upon the possibility of removing or relieving the primary disease. After suppuration has commenced in the kidney, the chance of recovery is very remote.

TREATMENT.—The most essential element of the treatment is to remove the cause if possible, but the act of doing so is seldom unaccompanied by the danger of increasing the disease, involving, as it often does, severe operations upon the urinary organs, as lithotomy, lithotripsy, or internal or external urethrotomy. These operations would of course, if possible, be avoided if the renal symptoms were at all marked. The fatal termination being in almost all cases associated with cystitis and extension of the inflammatory process to the pelvis of the kidney, it is needless to point out that our best hope of preventing consecutive renal inflammation lies in the prevention of cystitis by scrupulous attention to cleanliness in the instruments used. This cannot be too much insisted upon in the management of cases of paralysis of the bladder from injury or disease of the spinal cord. The catheters used should, if possible, be of soft red rubber, which is not damaged by boiling or by solutions of carbolic acid. Paraffinum Liquidum is the best lubricant and can be efficiently sterilised by heating in a test-tube; and before any instrument is passed the meatus should be cleansed with a weak mercurial lotion.

If cystitis occur this must be treated without delay, in order, if possible, to restore the healthy condition before exhaustion has taken place. The most important element in the treatment is the daily irrigation of the bladder with a non-irritating antiseptic, such as a boiled solution of boric acid. *See* **BLADDER**, Diseases of.

If the symptoms of subacute interstitial nephritis are present, the patient will frequently derive much benefit from a pure milk diet. At the same time small doses of opium seem to promote the action of the skin, and so to relieve the kidney without producing the dangerous effects so much to be feared in Bright's disease. The action of the skin may at the same time be still further promoted by vapour-baths. The bowels should be kept freely open. Counter-irritation, either by dry-cupping or mustard-poultices over the loins, followed by hot fomentations, is frequently of use. When the symptoms of the acute form are well marked, operations to relieve the cause only hasten the fatal event. By careful nursing, and the treatment above described, the symptoms may be so far reduced in intensity as to render an operation for the removal of the cause justifiable.

MARCUS BECK.

RAYMOND JOHNSON.

KIDNEY, Cystic Disease of.—The presence of cysts in the kidney may co-exist with other lesions, or they may constitute the sole morbid

change. In the former case the cysts are usually few in number, small in size, and scattered. Where cystic disease is the only morbid change present the formation of cysts may be so widespread that but little appearance of renal tissue is left, although even in these cases a considerable amount exists in a compressed form between the cysts.

I. Scattered single cysts varying in size from a pin's head upwards are not uncommonly seen in the cortex of an apparently healthy kidney. They are much more abundant, however, in the granular kidney and in the contracted form of Bright's disease (*see* **BRIGHT'S DISEASE**). In a few instances of granular kidney the formation of cysts is more widespread, so that a score or more may be present in a single kidney. In these cases the size of the cysts varies considerably. They are most marked in the cortex and frequently project from the surface, their walls being thin and their contents a clear, yellowish, albuminous fluid, though in some cases this has undergone a colloid or gelatinous degeneration. Haemorrhage into the cysts occasionally takes place; the contents of these will be discoloured with altered blood. The cysts present in granular kidney may in exceptional cases be very numerous. They are never arranged with any great regularity, and while a large number may be found in one kidney, only a few may be present in its fellow. These cysts, however large and numerous, are essentially similar to the microscopic cysts found in granular kidney and in chronic Bright's disease, which owe their origin to dilatation of portions of the renal tubules. The larger cysts also owe their origin to the dilatation of the renal tubules, although in the fluid they contain, as in that of a hydronephrosis, but small quantities of urea are present. These cysts, especially when large, are lined by a layer of cubical or slightly flattened epithelium, the cells of which are usually hyaline. The presence of these cysts in granular kidney is a matter of no clinical importance. Occasionally thick-walled single large cysts, the size of an orange, are found. Usually such cysts involve one or other extremity of the kidney, and the renal tissue is spread out in an irregular manner so that one wall of the cyst, usually the anterior, is thinner and contains less renal substance than the posterior. The presence of these large single cysts is not usually accompanied by any other morbid condition of the kidney except that it may be malformed. These cysts are also supposed to owe their origin to obstruction of the renal tubules, but it is possible that some cases are dependent on the formation of a cyst as the result of external violence causing a rupture of the renal substance, although, speaking generally, wounds of the kidney heal readily. These cysts are of no great clinical importance except that the cyst may be of sufficient size to be recognised by abdominal palpation, and may be confounded with an enlarged gall-bladder or with some other variety of renal tumour.

Rare forms of cystic disease are seen involving the medulla of the kidney, the cysts being more irregular in their shape than in their distribution, and it is probable that some of these cases are adenomata of the kidney undergoing cystic degeneration.

II. The term '*cystic disease*' is generally limited to a condition in which the whole kidney consists of a congeries of cysts of varying size. Notwithstanding the enormous development of cysts the organ

retains its outline, but otherwise presents the appearance of a large bunch of grapes. Two forms of this general cystic disease may at any rate be recognised, one in which the condition is congenital, the other in which the morbid process is only detected in adult life or in the aged. In both forms of the disease both kidneys are usually affected, although cases have been described where the disease was unilateral, or at any rate where it was much more advanced on the one side than on the other. In the congenital form the kidneys are of very varied size, in some cases not more than double or treble the normal, in others the increase is so great as to produce difficulty in labour. The congenital form has been supposed to be due to dilatation of the straight tubules as a result of inflammation brought about by presence of uric acid, the atresia of the straight tubules leading to the development of retention-cysts. Another view of the disease is that the cyst-formation occurs in the persistent remains of the Wolffian bodies. Malformation of the kidney and of the lower urinary tract is often associated with the presence of congenital cystic disease; and malformations in other parts of the body, such as the presence of supernumerary fingers and toes, have been described.

In the variety of the disease seen in adults, the kidneys are greatly increased in size, consisting of a mass of cysts varying from one-eighth to three-quarters of an inch in diameter. The cysts are entirely distinct from one another, and their contents cannot be emptied by pressure. The fluid in the cysts is usually amber-coloured, containing traces of urea and a variable amount of proteid matter. In some cases the colour of the fluid is altered by the presence of extravasated blood. All distinction between the cortex and the medulla is lost, the kidney apparently consisting of a uniform mass of cysts of irregular size; but on section the renal tissue is found in considerable quantities between the walls of the contiguous cysts. In some cases this renal tissue is normal in appearance, in others it presents changes similar to those seen in the granular kidney.

Small cysts may also be present in the mucous membrane of the pelvis of the kidney, and it is not uncommon for cysts to be present in other organs of the body, especially the liver, the brain, and sometimes the thymus.

In cases of cystic disease the heart and the arteries may show changes similar to those seen in cases of granular kidney; but this is not invariably the case, and it is probable that, when present, these vascular changes are dependent on the fact that the remaining renal tissue spread out between the cysts has undergone the same changes as those seen in the granular kidney.

The pathology of general cystic disease in adults is obscure. Some have thought that these cases are either really congenital and have remained latent for a number of years, in some cases throughout life, or else that their mode of origin is similar. Others have thought that the disease is of the nature of a growth, more especially owing to the fact that cysts are found in other organs such as the liver and brain. It is unquestionable that extensive cystic disease of some kind may result from the presence of adenomata.

SYMPTOMS.—In many cases no symptoms at all are produced, death being dependent on some other morbid process. It is probable that the disease

always runs a long latent course, symptoms occurring more or less suddenly, and sometimes only towards the end. In some cases the first suspicion of the presence of cystic disease is the occurrence of acute uræmia. More usually, however, before the onset of uræmia other symptoms have been present, such as loss of strength, emaciation, and pain in the back. Hæmaturia, often profuse, is not uncommon. These symptoms, together with the presence of bilateral tumours and the secretion of an abundant pale urine containing traces of albumen, are the most characteristic features of the disease. In one case observed by the writer the condition was diagnosed four years before the onset of fatal uræmia. In some instances the renal tumour produces varying degrees of intestinal obstruction, as a result of pressure on the colon. Cystic kidney commonly causes death by producing uræmia; this is usually of the acute type, and epileptiform fits and coma are frequent. In some cases, however, latent uræmia similar to that seen in calculous obstruction is produced, and in these cases death may occur quite suddenly from respiratory failure.

DIAGNOSIS.—Abdominal palpation usually readily reveals the existence of this condition; but difficulties may present themselves where the disease is unilateral or more developed on one side than on the other. One of the characteristic features of the tumour is its lobulation. The disease may be confounded with hydatids; and in some cases, owing to the presence of hæmaturia, malignant disease of the kidney may be diagnosed.

PROGNOSIS.—The prognosis of the disease is very uncertain, and depends mainly on whether symptoms are present. If these are of the uræmic type, life is not usually prolonged for more than a few months, and death may ensue very rapidly.

TREATMENT.—The fully developed disease is beyond cure, and all that can be done is to treat such cases hygienically and dietetically, like cases of Bright's disease. Where the disease is unilateral the affected kidney may be removed, but such cases are very rare, and nephrectomy should not be performed where there is evidence of bilateral disease, firstly, because in such cases operative interference may bring about fatal uræmia, and, secondly, because the kidney, although extensively diseased, may still perform its functions, though to a limited extent.

KIDNEY, Embolism of.—**ÆTIOLOGY.**—Infarction of the kidney may follow the detachment of clots or vegetations from the interior of the heart or large arteries, or of fragments of calcareous matter from atheromatous ulcers in the aorta. Microscopic emboli may be due to the lodgment of micro-organisms in the kidney. The arrangement of the arteries in the kidney is such as to lead inevitably to infarction if any of them become occluded. Results somewhat similar occasionally follow thrombosis of the renal artery. See p. 838.

Renal embolism is therefore a common consequence of thrombosis in the left auricular appendix or in the left ventricle, and of valvular disease of the heart, especially when due to malignant endocarditis. In rare instances the embolus may consist of a piece of a clot detached from the interior of an aortic aneurysm. Usually one or more of the interlobular arteries of the kidney becomes blocked, but in some cases the embolus is of large size and blocks one of the main divisions, or even the entire renal artery.

MORBID ANATOMY.—The results of renal infarction depend chiefly on the nature of the embolus. See **EMBOLISM**.

In cases of chronic heart-disease extensive and repeated embolism may occur, leading to the formation of a considerable number of large infarcts in the kidney; and their subsequent absorption leads to great deformity and puckering of the organs.

SYMPTOMS.—These are frequently so trivial as to escape observation. The most characteristic symptoms are the sudden occurrence of pain, together with albuminuria, the extent of the latter depending a good deal on the extent and character of the embolism. In a considerable number of cases sudden hæmaturia occurs, but it is not such an invariable accompaniment as albuminuria. The pain is usually of moderate severity, and is felt in the back and loin. It is not so definite in its localisation as that seen in renal colic, but may be confined definitely to one side. The albuminuria and the hæmaturia are both characterised by their sudden appearance and equally sudden disappearance. The hæmaturia is usually only sufficient to impart a smoky colour to the urine; copious hæmaturia is rare.

In some cases the occurrence of renal embolism is first revealed by the occurrence of sudden pyuria. This is necessarily confined to septic cases. The pyuria also is characterised by its sudden appearance and disappearance, and is dependent on a renal abscess rupturing into the renal pelvis and being discharged with the urine. In septic cases the onset of renal embolism is marked by the occurrence of rigors and high fever.

DIAGNOSIS.—Renal infarction has to be distinguished especially from passive congestion of the kidney and from acute Bright's disease. The principal difficulties in diagnosis arise in cardiac cases. Renal embolism is usually recognised by the suddenness of onset and transitory duration of its main symptoms, and by the presence of an underlying disease capable of causing it. Absence of renal dropsy is often a means of distinguishing infarction from the onset of an acute inflammation.

PROGNOSIS.—The prognosis depends entirely on the cause. In simple morbus cordis it is not in itself a serious condition; but in septic cases it is serious, in that it affords a clue to the existence of a graver underlying disease.

TREATMENT.—There is no special treatment for this condition, but it is essential to separate it from acute nephritis, inasmuch as it does not require the treatment necessary for that affection.

KIDNEY, Fatty Disease of.—Fatty disease of the kidney consists of a simple fatty degeneration of the epithelial elements of the kidney, and is a condition which is not characterised by any recognised symptoms. It is usually part of a widespread fatty degeneration involving also the heart and liver. See **FATTY DEGENERATION**.

MORBID ANATOMY.—The kidneys are usually normal in size, their surface is smooth, the capsule strips readily. There is no congestion. The surface of the organ is paler than naturally and has a greasy appearance, and on section the cortex is also pale and yellowish. On microscopical examination the epithelial cells show the well-known features of fatty degeneration. The Malpighian bodies, the vessels, and the stroma are not affected.

SYMPTOMS.—It is said that the quantity of urine is apt to be diminished. Albuminuria is not usually

present, but it has been asserted that small quantities of fat are present in the urine; this, however, may also be seen in health.

DIAGNOSIS.—The condition can only be suspected from the existence of causes capable of causing fatty degeneration and from the presence of fatty degeneration in other organs, and more especially the liver.

PROGNOSIS.—The involvement of the kidneys is not known to affect materially the prognosis of the general disease, the involvement of the other organs, especially the heart, being a more important factor in the case.

TREATMENT.—The treatment is that of the underlying condition.

KIDNEY, Granular.—The granular kidney presents some resemblances to that variety of Bright's disease described as the contracted white kidney (p. 217), inasmuch as in both the cortex is greatly diminished in thickness, and owing to the degeneration of the epithelial structures in the kidney and the overgrowth of fibrous tissue the surface of the organ in both instances is granular.

At least two forms of the true granular kidney may be recognised, one where the morbid process is mainly limited to the kidney itself, and the other where the renal changes are accompanied by very extensive degeneration of the arterial system; but in both forms the renal arteries are profoundly affected.

ÆTIOLOGY.—In many cases the ætiology of granular kidney is really that of arterial degeneration. In such cases, alcoholism, syphilis, heredity, and perhaps laborious occupations, play a part. The disease affects men more often than women, and is especially common after middle life, being rare under twenty-five, and uncommon before forty. It may also be produced by the action of toxic substances, especially lead and alcohol, and perhaps the poison of gout. The disease is said to be associated with excessive eating and also with prolonged mental anxiety.

MORBID ANATOMY.—The kidneys are usually smaller than normal, but great variations in size are seen; in many cases in which the disease is associated with extensive arterial degeneration, the kidneys are not so shrunken as in other cases in which the general arterial lesions are less developed. The weight of an affected kidney may be reduced to two ounces, but usually it is between three and four. The diminution in size is often greater than the diminution in weight, owing to the great overgrowth of fibrous tissue. The surface of the organ varies considerably in colour; in some cases it is of a uniform red colour, in others mottled. The surface is coarsely granular, and for this reason the term 'raspberry kidney' has been applied to it. The capsule is adherent, moderately thickened, and tears the renal substance as it is stripped off. The cortex is extremely narrow, frequently measuring not more than one-sixteenth of an inch. The medulla is deeply congested and the fat in the hilum abundant. The cortex may contain cysts varying in size from a pin's head to a cherry; in some cases great numbers of these cysts are found, in others only one or two. The cysts are filled with a yellowish albuminous fluid containing small quantities of urea. On section, the vessels stand out prominently, their walls being markedly and irregularly thickened. The vessels in other parts of the body show signs

of degeneration, such as a loss of elasticity and great thickening of the inner coat, dependent on an overgrowth of fibrous tissue in the subendothelial layers. Military aneurysms may be found in the cerebral vessels. On microscopical examination the kidney shows a general, but irregularly distributed, overgrowth of fibrous tissue, specially well marked round the glomeruli. Many of the latter are atrophied, the capillary tufts having undergone hyaline degeneration; in others there is a development of fibrous tissue in the capillary tuft itself. A few normal glomeruli may be found, and others in which the capillary tuft shows signs of cellular and fibrous overgrowth without any great thickening of the capsule. The circulation in the glomeruli is greatly interfered with, partly because of the fibrosis of the glomerular chamber and of the tuft, and partly owing to the thickening of the endothelial coat of the arteries. The tubules in many areas of the kidney are apparently replaced by fibrous tissue, the epithelial cells having disappeared and the walls of the tubules remaining collapsed. In many parts of the cortex the tubules are dilated, and in these the epithelium consists of cubical hyaline cells; in other parts the epithelium has undergone granular and fatty degeneration. The tubules of the medulla are less affected than those of the cortex. The vessels of the kidney show considerable changes in all the coats; peri-arteritis is present to a considerable extent, but the inner coat is especially thickened, by the formation of fibrous and elastic tissue in the subendothelial layer, so that this coat often equals in thickness all the other coats of the vessel together. The middle coat also may be thickened by the formation of an excess of muscular tissue. The interstitial tissue throughout the kidney is commonly infiltrated with small round cells. The heart is hypertrophied; the degree of hypertrophy varies with, and is probably dependent on, the extent of the general arterial degeneration. The amount of cardiac hypertrophy bears no relation to the degree of atrophy of the kidney. Hypertrophy affects the left side of the heart especially, but in any case in which the enlargement of the heart is at all considerable, the right side is also affected. The cardiac hypertrophy is in great part dependent on an overgrowth of muscular tissue; in some exceptional cases, an irregular overgrowth of fibrous tissue may occur.

SYMPTOMS.—In a considerable proportion of cases the disease is absolutely latent, death occurring from some complication, and is only revealed at the autopsy. Thus, in one case, fatal cerebral hæmorrhage, and in another uræmic phenomena, often of extreme violence, may be the first indications of the existence of the malady. More usually the patient comes under observation presenting signs of a general failure of health such as weakness and emaciation, together with some headache and perhaps such cardio-vascular symptoms as palpitation and shortness of breath. Such patients are found to pass eighty or one hundred ounces of clear, pale urine having a specific gravity of 1010, and containing a small quantity of albumen. Albumoses may be present in the urine in addition to serum-albumen and serum-globulin. In another group of cases the early symptoms are of a cardiac type, such as shortness of breath, palpitation, præcordial pain, and œdema of the lungs and lower extremities. In this way the disease presents symptoms similar to those seen in mitral regurgitation, and it is not uncommon

for cases of granular kidney with secondary cardiac failure to be looked upon clinically as cases of mitral regurgitation with renal congestion.

The most frequent type of the disease is that in which the patient presents general vague symptoms of ill-health together with symptoms referable to the disturbance of the circulatory system. In some instances the general symptoms of ill-health are so vague as to present a close resemblance to those seen in neurasthenia. The malady has generally advanced to a very considerable extent before it produces symptoms sufficient to lead to its recognition.

Hæmorrhage may also occur as an early symptom. This most frequently takes the form of epistaxis. Cerebral hæmorrhage is likely to occur in cases in which the morbid lesions in the kidney are less marked than those in the general arterial system. Hæmorrhage into the retina is not uncommon, and may be the first symptom. In rare instances copious hæmaturia occurs, and it is probable that in some of these cases the hæmorrhage really comes from the mucous membrane and pelvis of the kidney, and is not due to acute inflammation. Purpura is a frequent complication.

The general wasting seen in granular kidney is sometimes as extreme as that seen in malignant disease, and is far more obvious than the emaciation accompanying other forms of renal disease where dropsy is present and masks it.

The urine in cases of granular kidney is usually albuminous, though in most instances the quantity of albumen present is not more than a trace. The quantity of albumen, however, varies at different times, and at some stages may be absent. This is most frequently observed in the early stages of the disease, but sometimes the urine may remain free from albumen during the fatal illness, and this may be so even when the patient dies from uræmia.

Albumoses in traces are frequently present, and a few instances have been described where these substances have been present in very large quantities. In some cases the albumosuria is dependent on the co-existence of bone-disease or of inflammatory processes in some other part of the body, or even of ovarian cysts. There are, however, cases of uncomplicated granular kidney in which large quantities of albumoses may be secreted by the urine. It has been supposed that in these cases the alimentary canal is the source of the albumoses, and that these are absorbed and remain unchanged owing to some anomaly in the processes of digestion and nutrition. See ALBUMOSURIA.

The heart in cases of granular kidney usually shows distinct signs of hypertrophy, often considerable in amount, and in not a few cases the hypertrophy is further complicated by the co-existence of mitral regurgitation owing to dilatation. The physical signs of hypertrophy may be more or less completely masked by the presence of emphysema, the latter being a common complication of this form of renal disease. In addition to the usual physical signs of cardiac hypertrophy, the heart-sounds are often considerably altered, the first sound being muffled, reduplicated, or presenting the well-known *bruit de galop*. The aortic second sound is markedly accentuated. The pulse shows the characteristic features of high tension, and in many cases in addition to this the arteries yield evidence of degenerative processes, and the radial arteries roll under the finger like pieces of whipcord. Marked structural changes in the vessels, sufficient

to be recognised clinically by the finger, are, however, by no means invariable, and are not so characteristic of granular kidney as the presence of a high-tension pulse. The arterial degeneration may often be detected from the examination of the fundus oculi owing to the rigid and thickened condition of the arteries; and it is not uncommon to see extreme congestion of one or more of the retinal veins produced by the pressure of a rigid artery crossing a vein. Papillitis, retinitis, retinal hæmorrhages, and all the features which characterise albuminuric retinitis, are frequently present in this disease.

DIAGNOSIS.—When uræmia occurs in a latent case, especially if it take the epileptiform type, the disease may be confounded with epilepsy. This can usually be excluded by an examination of the fundus oculi.

In the cases in which vague general symptoms only are present the disease may be confounded with neurasthenia or some more or less trivial gastric affection. Whenever symptoms of this character occur in middle-aged men, granular kidney should be suspected, and the state of the urine, the vessels, and the fundus oculi carefully examined.

In the cardiac type of the disease considerable difficulties present themselves in the differential diagnosis between granular kidney complicated by a failing heart and primary morbus cordis complicated by congestion of the kidney. Although in the latter case the urine is usually scanty, high-coloured, and contains a considerable quantity of albumen, cases are sometimes seen in which the percentage of albumen is small, resembling the condition present in granular kidney. Further, when granular kidney is complicated with a failing heart and dropsy, the amount of urine secreted is necessarily diminished. The condition of the vessels, the degree of tension, and the state of the fundus oculi furnish the most reliable evidence in making a differential diagnosis between the two conditions.

Granular kidney has been confounded with cerebral and cerebellar tumour. In all cases of suspected granular kidney it is essential that the urine should be repeatedly examined, as the absence of albumen based on a single examination is of little or no value in excluding the disease.

PROGNOSIS.—This disease must be regarded as an incurable malady, although patients suffering from it may in some instances live for many years, and even suffer comparatively little inconvenience. They are always, however, in imminent danger of succumbing to some complication, such as cerebral hæmorrhage or fatal uræmia.

In cases of the cardiac type the prognosis is always very serious, and also in those cases in which inflammatory complications, such as pericarditis, pneumonia, or pleurisy, are present.

TREATMENT.—The first essential in the treatment of granular kidney is to protect the patient as far as possible from strain, and especially from strain of his vascular system, as any sudden exertion may, owing to the serious arterial disease present, lead to the occurrence of cerebral hæmorrhage. For this reason, among others, care should be taken, when prescribing purgatives for these patients, that none are ordered that cause any severe griping or straining at stool, and it is probable that saline purgatives are the most efficient and suitable drugs in these cases.

The high tension should, if possible, be relieved. Warm baths and saline purgatives are efficient for this purpose, together with the regulation of the diet and the avoidance of stimulants. The only drugs which possess an efficient action in lowering tension are the nitrites and nitro-glycerin. Nitrite of amyl produces the most transitory effects, and is not very suitable. Nitro-glycerin and erythrol tetra-nitrate are more useful, owing to the fact that the effects produced are more lasting. Often, however, owing to the anatomical changes in the blood-vessels, these drugs produce but little relief. Venesection may be used for the same purpose in cases of extreme high tension associated with uræmic symptoms or even with cardiac distress, and it is notorious that a spontaneous hæmorrhage such as epistaxis often relieves these patients considerably. The best effects in the treatment of the high tension are usually obtained by promoting free action of the skin, free purgation with salines and careful dieting. The diet should be light, and these patients should be especially warned against eating large quantities of meat and highly seasoned foods. Alcoholic stimulants are harmful, and even such substances as tea and coffee should be taken in moderation owing to their stimulant action. It is impossible, however, in all cases to cut off alcoholic stimulants; they may be necessary, for instance, in some cases of the cardiac form of the disease. Any anæmia that may be present should be treated with iron and arsenic. Whenever cardiac failure has supervened, and the patient presents symptoms of mitral disease, the case must be treated on the usual lines, although such drugs as digitalis, strophanthus, strychnine, and cardiac tonics generally, causing increase of the blood-pressure, must be used with caution, since the heart is already failing as a result of increased arterial pressure, and all these drugs tend to increase arterial pressure. At the same time it must be remembered that their beneficial action on the heart often outweighs their effects on the arteries.

Wherever the presence of dropsy requires incision or puncture for its relief, extreme care must be taken to avoid septic complications, since granular kidney, like other renal diseases, leads to a diminished resistance on the part of the tissues. Residence in a warm equable climate during the winter months is as important in the treatment of granular kidney as it is in that of Bright's disease.

KIDNEY, Hæmorrhage in connection with.—Hæmorrhage may occur into the substance of the kidney as a result of traumatism, as a result of embolism, of acute inflammation, and from the presence of growths.

Submucous hæmorrhages in the renal pelvis are seen in cases of purpura, leucocythæmia, and granular kidney, and it is possible that the profuse hæmaturia sometimes seen in these diseases is dependent on hæmorrhage in this situation. See p. 621.

KIDNEY, Hydatid Disease of.—Hydatid disease involving the kidneys is rare, both liver and lung being more commonly attacked. The left kidney is rather more frequently affected than the right, and the cyst usually arises in the substance of the organ. In enlarging the cyst usually reaches the pelvis of the kidney. In rare instances it has enlarged upwards so as to perforate the lungs. There is no record of a cyst having burst into the

peritoneum. It is not uncommon for the cyst to cease growing. In other instances suppuration may occur as in other organs. When a hydatid cyst ruptures into the pelvis of the kidney the hooklets and daughter-cysts are passed in the urine, and may give rise to attacks of renal colic, but the cyst may reach a very large size before it ruptures. The discharge of the hooklets and daughter-cysts in the urine usually takes place paroxysmally, and may go on for many years. See *ENTOZOA*.

A tumour resembling a hydronephrosis may be produced, but the disease can scarcely be diagnosed unless hooklets are found in the urine or there is clear evidence of hydatid disease elsewhere. When hooklets are present in the urine the parent hydatid cyst is not necessarily in the kidney; it may be in the retroperitoneal tissue, but the combination of hooklets in the urine with attacks of renal colic is very suggestive of hydatid of the kidney.

The *prognosis* is on the whole favourable, owing to the fact that the great majority of these cysts, if they rupture, rupture into the pelvis and their contents are discharged.

The *treatment* is similar to that of hydatid disease involving other parts.

KIDNEY, Hyperæmia of.—Two varieties of congestion of the kidney are seen: (1) active congestion; and (2) passive congestion.

In congestion of the kidney the primary changes occur in the blood-vessels of the organ, but these may lead to secondary results in the kidney, especially if long-continued or severe.

ÆTIOLOGY.—*Active congestion* of the kidney is closely associated with nephritis, and is usually produced by the action of toxic substances on the organ. It is thought, however, that it may arise under other conditions, and more especially by reflex effects produced through the nervous system. Many poisons, such as cantharides, turpentine, carbolic acid, and mineral acids, produce acute congestion of the kidney. The poisons of scarlet fever and of other acute specific fevers produce the same result. Although many poisons, mineral and organic, produce acute congestion, their action is rarely limited to the blood-vessels. Some poisons, such as cantharides, produce marked effects on the blood-vessels; others, such as heavy metals, produce their main effects on the renal cells. Acute congestion of toxic origin is separated with difficulty from nephritis and Bright's disease.

Acute renal congestion produced reflexly through the nervous system is sometimes seen as a sequel to irritation of the lower urinary tract, as by the passage of a catheter. This rarely occurs where the kidneys are perfectly healthy, but it is liable to supervene if advanced renal lesions exist. It has been thought that congestion of the kidney may arise in some cases of excessive cardiac hypertrophy, e.g. aortic disease.

Passive congestion of the kidneys may be seen whenever the circulation through the venous system is interfered with. It is most commonly met with in cases of cardiac or of pulmonary disease, in which the right side of the heart is either primarily or secondarily involved. It may also be due to the pressure upon the vena cava, caused by diseases of the pericardium, abdominal tumours, or hepatic cirrhosis. The renal veins may be pressed upon by growths or aneurysms, and in rare instances thrombosis of the renal veins may occur as a result

of marasmus or of septic processes. Thrombosis of the renal vein may occur as a result of these causes when the kidneys are healthy; but it is also seen in certain renal diseases, especially the granular kidney and malignant disease. In rare instances the renal veins become thrombosed as a result of a phlebitis spreading from other parts, especially the pelvic veins.

MORBID ANATOMY.—In *active congestion* the kidneys are generally of normal size, or larger than normal, and in some very acute forms the distension may be such as to cause rupture of the capsule. The capsule strips off readily, leaving a smooth but congested surface. On section the vessels are found engorged, and the distended glomeruli can frequently be seen as bright red spots. Streaks of extravasation may also be visible here and there, which on microscopic examination are seen to be dependent on hæmorrhage into and between the tubules. The changes are more marked in the cortex than in the medulla. Microscopically degenerative changes may be found in the renal cells, and both the tubules and the interstitial tissue show the presence of extravasated blood. In *passive congestion* the anatomical changes depend very much on the duration of the disease. In the acute forms dependent on thrombosis of the renal veins the congestion of the kidney-tissue is extreme, and is especially marked at the junction of the cortex and the medulla. On microscopical examination blood is found freely extravasated in and between the tubules, and the cells of the kidney may have undergone complete necrosis. In the more chronic forms the kidney is enlarged, red and often tough, the capsule may be adherent, while, on being stripped, it leaves an uneven surface, although in the earlier stages the surface of the organ may be smooth. The congestion is always well marked in the medullary portion of the kidney, and the Malpighian bodies are not generally so distended as in active congestion. On microscopical examination the veins are found distended with blood, and extravasations into the interstitial tissue are especially marked in the medullary region. Where the congestion is at all severe the tubules themselves will also contain blood. The interstitial tissue is increased in amount, and the basement membrane of the tubules is thicker than normal. In many tubules the cells have lost their characteristic structure and have become hyaline and glass-like. In many cases it is difficult to distinguish with certainty between the results produced by mere passive congestion and those dependent on the co-existence of slighter forms of nephritis.

SYMPTOMS.—In *active congestion* true symptoms are frequently absent, and the condition is only revealed by an examination of the urine. In others the patient experiences some pain in the back, and this in exceptional circumstances may be severe. The urine is diminished in amount and contains albumen in variable quantity, large amounts sometimes being present. Blood is also frequently present, usually in small amount, but in some forms of acute congestion produced by toxic agencies, such as turpentine and cantharides, the amount of blood present may be large. Hyaline, granular, and blood-casts also occur. In the more severe forms of acute congestion suppression of urine may occur, but dropsy is absent and forms one of the means of distinguishing between acute congestion and acute Bright's disease.

In *passive congestion* the urine is diminished in quantity, often dark in colour, and contains albumen in varying amount, from a mere trace to a sixth or even a third of its bulk. Hyaline and granular casts are also present, and it is not uncommon for blood even in considerable quantity to be present in the urine. In many cases of passive congestion it is difficult to exclude other causes, especially toxic ones, and this may account for some of the higher grades of albuminuria seen, for instance, in pregnancy. In most cases of passive congestion the amount of albumen in the urine is small.

DIAGNOSIS.—*Active congestion* has to be separated from Bright's disease and from infarction of the kidney. It is sharply marked off from the severe forms of Bright's disease by the absence of dropsy and also by the rapid manner in which the condition subsides and health returns.

Passive congestion has also to be separated from Bright's disease. The difficulties that present themselves occur mainly in cardiac cases, in which it may be difficult to determine whether we have to deal with a primary cardiac case, with secondary congestion of the kidney, or a primary renal case with secondary cardiac failure. In passive congestion and in Bright's disease the urine may be scanty, but in passive congestion it is usually of high specific gravity, whereas in Bright's disease, even if scanty in amount, the specific gravity is usually low. This statement, however, is not always accurate, and cases may be seen of congested kidney dependent on heart-disease where the urine is of low specific gravity; these, however, are exceptional. In passive congestion, although blood may be present even in some abundance, epithelial casts do not occur, the only casts present being hyaline and granular.

PROGNOSIS.—*Active congestion* of the kidney frequently subsides even when dependent on toxic causes, such as turpentine and cantharides; reflex active congestion when occurring in diseased kidneys is, however, extremely serious. The prognosis of *passive congestion* must necessarily depend almost entirely on its cause. It is usually a serious complication of morbus cordis and a sign of plethora of the venous system, although extreme anasarca may occur in cardiac cases without signs of marked congestion of the kidney, and conversely cases are seen where the renal congestion is apparently greater than the congestion of the vessels generally. Passive congestion in cardiac disease affects the prognosis unfavourably, owing to the extreme difficulty of promoting diuresis in such cases.

TREATMENT.—It is customary in *active congestion* to apply dry cupping or hot fomentations over the loins, and in this way the pain may be relieved. Measures promoting the activity of the skin and of the bowels must be used, and the diet must be reduced to a minimum in order to spare the kidneys as much as possible. In the more severe forms the malady requires treatment identical with that of acute Bright's disease.

In *passive congestion* treatment must be directed to the cause of the condition, and many such cases improve greatly under the administration of digitalis or caffeine, drugs which increase the activity of the kidney. Digitalis is especially of service owing to the fact that it produces its diuretic effects by improving the general circulation. Saline purgatives are often of great service, and the

activity of the skin should be promoted by hot-air and warm baths.

KIDNEY, Hypertrophy of.—The term 'hypertrophy of the kidney' is usually applied to a condition in which the organs are increased in size without the presence of any gross morbid lesions. It has been much disputed whether the increase in the dimensions of the organ is dependent on an increase in the size of the normal elements of the kidney, or whether it is dependent on an increase in their number. Experimental evidence has been adduced, more especially by Tuffier, to show that after removal of portions of the kidney new formation of renal elements takes place, and Tuffier considers that by progressive partial nephrectomies it is possible to remove an amount of kidney equal to the whole of that normally present, and yet for kidney-tissue to be found *post mortem*. This conclusion, however, has not been accepted by other authorities, and most observers are agreed that no such formation of new elements occurs after the removal of portions of one kidney. The removal of one kidney in animals or in man is usually, but not invariably, followed by a great increase in the size of its fellow; and in cases of congenital atrophy or want of development of one kidney, it is not uncommon for the other to more than equal the total weight and size of two normal kidneys. See KIDNEY, Atrophy of.

The removal of one kidney is more likely to be followed by an increase in size in the other when the organ is removed in the young. Unilateral extirpation of the kidney in old animals is not followed by any hypertrophic enlargement of its fellow, and it is well known that in cases of malignant disease in the human subject involving one kidney the opposite organ is frequently not enlarged. Most observers are agreed that in cases of so-called hypertrophy of the kidney the elements are somewhat enlarged. Hypertrophy of the kidneys in the human subject may involve one or both organs. Bilateral hypertrophy often occurs in diabetes mellitus, and, although in some cases of diabetes the kidneys present evidences of Bright's disease, it is not uncommon for both organs to be considerably enlarged without the presence of any other morbid lesion.

Unilateral hypertrophy, however, is much more common, and this is seen in cases of congenital absence of one organ, in cases of unilateral atrophy, and also as a result of unilateral disease. Where unilateral hypertrophy occurs it is not uncommon for the organ to be more than double the size and weight of two kidneys. The main importance of hypertrophy of the kidney, apart from its theoretical interest, rests in the fact that such a kidney may be mistaken for a renal tumour, and several instances are on record where an exploratory laparotomy has been done under this impression. Further, although the presence of a single hypertrophied kidney is compatible with prolonged life, many instances having been recorded in patients over eighty years of age, yet such kidneys often become involved in pathological processes, more especially calculous disease and Bright's disease. Calculous disease is especially formidable in such cases. Hypertrophy of the kidney as such produces no clinical symptoms.

KIDNEY, Inflammation of.—The term *nephritis* is sometimes applied to the initial stages of

Bright's disease and more especially to acute Bright's disease. It is better, however, to restrict it to the changes produced as a result of the action of certain specific poisons and more especially those produced during the height of certain infective diseases, such as scarlet fever, pneumonia, typhoid fever, and diphtheria. Three groups of renal poisons may be recognised. The first is represented by the action of cantharidin and probably by the virus of scarlet fever. The second consists of certain metallic salts. A third group consists of certain vegetable and animal toxic substances, such as abrin, ricin, serpent-venom, and bodies present in the blood of some animals, e.g. the eel. The first group produce their main effects on the glomeruli and give rise to what has been spoken of as a glomerulonephritis. The second group produce a coagulation-necrosis of the cells of the convoluted tubules. The organic poisons of the third group cause degeneration of the epithelial structures of the kidney differing in some respects from a coagulation-necrosis. In many respects the effects seen in nephritis are similar to changes produced in some stages of Bright's disease, and even to those seen in the more severe forms of acute congestion.

Bright's disease is looked upon by some as merely a more severe or more prolonged nephritis. The nephritis seen during the incidence of acute diseases may be described as transitory, and is the cause of the so-called febrile albuminuria seen during the height of these diseases. Some maladies that produce transitory nephritis during their height may lead to the production of Bright's disease later. Thus in pneumonia and scarlet fever transitory nephritis is very common during the height of the disease, and Bright's disease may arise during convalescence; but the two conditions do not necessarily merge into one another.

The transitory nephritis of these diseases is characterised clinically by a moderate and transient albuminuria unaccompanied by the more serious effects of Bright's disease, such as dropsy. In many cases it is probable that the lesions present in the kidney are scattered and partial in their distribution. In the more serious forms of nephritis seen as the result of the action of such poisons as cantharides or turpentine, hæmaturia and more or less complete suppression of urine may occur. In the great majority of cases the condition is temporary and the albuminuria soon subsides and the patient completely regains health. It is possible that in some such cases these forms of nephritis lay the foundation for permanent renal disease.

In the slighter forms no special treatment is called for, but in the more serious forms similar measures to those adopted in acute Bright's disease are required. *See* BRIGHT'S DISEASE.

KIDNEY, Inflammation of Pelvis of.—Pyelitis is an inflammation—acute or chronic—of the pelvis of the kidney. Pyelitis may exist by itself, or it may be part of a more widespread affection of the genito-urinary tract, such as cystitis on the one hand and suppuration of the kidney on the other hand.

ÆTIOLOGY.—Pyelitis may be caused by infection from the lower urinary tract, or by the irritation of toxic substances excreted by the kidney. It is possible that the infective agent may sometimes reach the pelvis of the kidney directly from the blood-stream without being excreted by the kidney.

In the great majority of instances, however, the infection reaches the pelvis of the kidney either from above or from below. Predisposing causes to pyelitis are afforded not only by the stagnation of urine in the renal pelvis as a result of obstruction caused by the presence of calculi, but also by dilatation of the ureter either of congenital origin or dependent on some former obstruction of the lower part. Dilatation of the ureter is a most important predisposing cause in cases of pyelitis dependent on infection from below. Pyelitis may complicate cystitis, as for instance in gonorrhœa, with little or no distension of the ureter; but it is in cases of enlarged prostate and urethral stricture complicated by dilatation of the bladder and ureters that pyelitis is most frequently seen (*see* KIDNEY, CONSECUTIVE INFLAMMATION OF). Micro-organisms are also excreted by the kidney, and in their passage down the urinary tract they may set up inflammatory mischief either in the kidney itself or in the pelvis; in this way a descending pyelitis may be produced. There are a number of acute diseases such as typhoid fever and acute pneumonia, in which slight pyelitis may be present, and a number of poisons, especially turpentine and cantharides, may cause pyelitis. Other causes of pyelitis are the presence of calculi and tubercular disease in the pelvis of the kidney. The majority of cases of pyelitis are the result of sepsis, calculi, or tubercular disease. It is said that exposure to cold may lead to pyelitis, but this is very doubtful.

MORBID ANATOMY.—The mucous membrane of the pelvis of the kidney is congested, coated with mucus, and often greyish in colour. Numerous punctiform submucous hæmorrhages are frequently present and may cause necrosis of the overlying epithelium and so produce ulcers. The renal pelvis may contain varying quantities of pus. If the disease is of any duration, the pelvis of the kidney will be dilated and its walls thickened, and, in some cases, covered by a kind of false membrane. In most cases, both the kidney and the ureter present marked changes, the latter being thickened and often dilated and its lumen more or less obstructed, partly by the swelling of the mucous membrane and partly by the accumulation of inflammatory products and blood. The changes in the kidney present considerable variety. In simple pyelitis little or no change is observed except that the apices of the renal pyramids may be somewhat flattened. The pyelitis is frequently complicated by the presence of pyelonephritis or by pyonephrosis; in the former case areas of suppuration of varying size are found extending between the pyramids of the kidney into the cortex, and there are often numerous small abscesses in the cortex beneath the capsule. In pyonephrosis the kidney undergoes distension similar to that seen in hydronephrosis, but the renal tissue presents many inflammatory changes. The distension may be so great that the organ remains simply as a bag containing pus. In other cases where the distension is not so great, areas of the medullary portion of the kidney, more or less considerable in size, show necrotic changes. When pyonephrosis ensues as a complication to pyelitis, the capsule of the kidney is often greatly thickened, and adhesions may be formed between the kidney and surrounding organs.

SYMPTOMS.—The symptoms produced by pyelitis depend not only on the severity of the disease, but also very largely on its cause. In simple pyelitis, unassociated with the presence of calculi or

local tuberculosis, pain of moderate severity is felt in the loin; but in many cases this is not sufficiently severe to be called more than uneasiness. Frequency of micturition is often present, and a marked feature even when there is no evidence of the presence of cystitis. The urine is acid, and contains a variable amount of pus. Hæmaturia is not generally a marked feature, unless the pyelitis is due to local irritation (calculi or tuberculosis). The amount of albumen in the urine is small, and is dependent on the presence of the pus. Albuminuria out of proportion to the quantity of pus present is a sign of the involvement of the kidneys by some other morbid process, most commonly amyloid disease. Fever is usually present, and its course exceedingly irregular. In the more severe forms the temperature may be high and rigors may occur; while attacks simulating those of renal colic may be produced by the dislodgment of inflammatory products and by the passage of blood-clots through the ureter. There is usually tenderness on deep pressure over the affected kidney. When pyonephrosis ensues the urine frequently remains free from pus, since the production of the pyonephrosis involves the obstruction of the ureter. Where pyelonephritis is present as a complication or accompaniment of pyelitis the patients are much more profoundly ill; they become wasted, and assume a cachectic appearance and suffer from hectic fever. Where pyonephrosis develops, the tumour formed by the distended kidney may often be recognised. Pyelitis may be unilateral or bilateral. The former is seen in some cases of calculus and tubercular pyelitis, the latter more often where the pyelitis is a sequel of cystitis. In calculous pyelitis pain is a more prominent feature than in the simple variety, and such patients often present a history of the passage of calculi or gravel. Hæmorrhage is also often profuse in cases due to calculi. In tubercular pyelitis the discharge of pus is usually abundant and that of blood scanty, although cases have been described in which the onset of tubercular pyelitis was marked by profuse hæmaturia. In tubercular pyelitis tubercle-bacilli may be found in the urine, either by microscopical examination of the centrifuged deposit, or else by the inoculation of susceptible animals with the suspected urine. The latter is on the whole the more reliable method, owing to the difficulties occasionally experienced in distinguishing between the *Bacillus tuberculosis* and the *smegma-bacillus*.

DIAGNOSIS.—The diagnosis of pyelitis is usually readily made in uncomplicated cases by examination of the urine. Some difficulty may be experienced in determining whether the disease is unilateral or bilateral, and most frequently this has to be determined by the situation of the pain and tenderness. In some cases, and more especially in the female, cystoscopic examination of the bladder and catheterisation of the ureters may afford certain evidence as to the situation of the disease. Difficulties sometimes present themselves in distinguishing between cystitis and pyelitis, since, although the urine is generally alkaline in cases of cystitis and acid in pyelitis, this is not invariable, and some forms of cystitis occur with acid urine, and pyelitis complicating cystitis may be accompanied by an alkaline urine. The diagnosis has usually to be based on the presence of renal pain and tenderness. It is often a matter of great importance to determine whether pyelonephritis is present. Where this is the case the patient is usually more ill, more

wasted, and suffers from more characteristic hectic fever than where pyelitis alone is present. The urine in pyelonephritis is often abundant and of low specific gravity. Calculous is to be distinguished from tubercular pyelitis by the history, by the greater tendency to hæmaturia, and often by its longer duration. Calculous pyelitis is a malady which may exist for many years without seriously interfering with the general health. The most reliable means, however, of distinguishing between the two is by determining the presence or absence of tubercle-bacilli in the urine.

PROGNOSIS.—The prognosis of pyelitis depends on its cause. In the slighter forms dependent on the presence of gravel this is not unfavourable. The trivial pyelitis accompanying acute specific fevers is also of no great importance, except that it may lay the foundation for subsequent calculous disease. Cases of septic pyelitis and of tubercular pyelitis are grave. Pyelitis complicating gonorrhœa may even be fatal by setting up pyæmia; but many of these septic varieties of pyelitis can be successfully treated by operative interference.

TREATMENT.—Many forms of pyelitis require surgical treatment, such as the removal of stones or the draining of the distended kidney, or even in some cases the passage of a long catheter up the ureter and the washing out of the renal pelvis. The slighter forms associated with the presence of gravel may often be greatly benefited by medical treatment. The diet should be light, and all stimulating drinks should be avoided. The patient should take large quantities of barley-water, of infusion of buchu, or of linseed-tea. Alkalis, such as carbonate or citrate of potassium, often relieve the urgency of micturition; and *uva ursi* and infusion of buchu have been recommended for the same purpose. Mild counter-irritation may be applied to the loins to relieve the pain.

KIDNEY, Malpositions of.—Malpositions may be congenital or acquired. Further, the kidney may be fixed in its anomalous position, or the displacement may be accompanied by mobility. The most important congenital malpositions of the kidney are those in which the organ is situated (1) over the sacro-iliac synchondrosis; (2) in the concavity of the sacrum; or (3) in the iliac fossa. Such displacements usually involve only one kidney. Malposition of the kidney is often associated with other congenital anomalies, especially with anomalies in the development of the large intestine, and in its relation to the peritoneum. In some instances imperforate anus is associated with malposition of the kidney. The most frequent of the 'fixed' displacements is that over the sacro-iliac synchondrosis. A fixed displacement of the kidney is of some clinical importance, since (1) it may be mistaken for a tumour, as in the case of a kidney occupying the concavity of the sacrum and palpable *per rectum*; and (2) the displaced organ may become enlarged and tender, as a result of kinking of the renal vein. In this way considerable pain may be produced, and the condition be erroneously diagnosed as inflammatory, as in a case in which a swollen kidney, situated in the iliac fossa, was diagnosed as inflammation of the appendix.

Displacements with mobility are usually known as *movable kidney*. Two varieties of movable kidney are generally recognised: one where the mobility of the organ is slightly greater than normal,

so that it descends more during the normal respiratory movements; the other, where the unnatural mobility is far greater, so that the organ can be moved about freely in the abdominal cavity; to the latter variety the name of floating kidney has sometimes been applied. The normal kidneys move considerably during respiration, but not to an extent sufficient to allow the hand to be inserted above the kidney. Some authorities consider that if more than the lower third can be felt during inspiration the term 'movable kidney' should be applied to the condition. In the slighter forms of movable kidney the organ maintains its normal relations to the peritoneum, but in the more severe forms, or 'floating kidney,' the peritoneum passes behind the kidney to a variable extent, forming a kind of mesonephros. It is probable that the formation of this spurious mesonephros is generally congenital, but it may also be acquired as a result of unnatural mobility of the organ. The kidney normally is kept in its position partly by the pressure of the adjacent viscera and the tonic contraction of the abdominal muscles. In addition to these the most important factors are the bands of fibrous tissue which pass through the fatty bed in which the kidney lies, and also the reflections of the peritoneum from the kidney to the adjacent organs. The most important of the latter is the reflection passing from the anterior surface of the kidney to the duodenum.

ÆTIOLOGY.—Movable kidney is more common in women than in men, and the right kidney is more frequently affected than the left. The slighter forms of movable kidney are extremely prevalent in women, but such a condition apart from the production of symptoms is scarcely to be looked upon as pathological. Predisposing causes are distension of the abdomen and laxity of the abdominal walls, arising either as a result of the distension, or else due to impaired nutrition. In some instances the condition has developed as a result of tight-lacing. In many it has followed traumatism, either direct injury to the loin or more often displacement of the organ resulting from a shock or jar produced by jumping.

MORBID ANATOMY.—The kidney may be found in its normal situation, but owing to looseness of attachments it may be more freely movable behind the peritoneum. In the more serious forms the spurious mesonephros will allow the kidney to be moved about freely in the abdominal cavity. The kidney itself does not usually present any marked changes, but in some instances of extreme mobility where kinking of the vein or of the ureter has resulted, the results of these conditions may be visible (see **HYDRONEPHROSIS**; and **KIDNEY**, Congestion of). In some cases, changes in other organs are found, e.g. dilatation of the stomach, which apparently is produced by the traction exerted on the duodenum by the displaced organ through the peritoneal reflections passing from the kidney to the duodenum. The common bile-duct may also be dragged upon and partially obstructed in this way.

SYMPTOMS.—In a large number of cases no symptoms are produced, and the condition is recognised accidentally during examination of the abdomen for other purposes. In a certain number of cases, however, the patients seek advice with more or less characteristic symptoms, and in a small proportion of cases the effects produced may be severe. One of the most constant symptoms is

pain in the abdomen and loin, which occasionally resembles that of renal colic. In these cases, there may be nausea and vomiting with tenderness in the abdomen and loin. Abdominal distension may occur, and intestinal obstruction be simulated. Neurasthenic symptoms are often present even in cases where the pain and other effects produced are not severe, and instances occur in which no other signs than those of movable kidney are seen in patients suffering from very severe neurasthenia. In such cases wasting is often a marked phenomenon. In a small proportion of cases of movable kidney, hæmaturia occurs, accompanied by severe pain, and it is supposed that in such cases kinking of the renal vein has resulted from the unnatural mobility of the organ. Transient jaundice is rarely met with; gastric symptoms are accounted for by dilatation of the stomach. Intermittent hydronephrosis is one of the most important of the more serious effects produced by this condition.

DIAGNOSIS.—The condition is usually readily recognised owing to the fact that a large number of patients suffering from it are emaciated and palpation of the abdomen readily reveals the extreme mobility of the organ. Pressure on the kidney often causes nausea and faintness. The condition may be overlooked where the patient presents only gastric symptoms, and difficulties may arise in the differential diagnosis between some forms of movable kidney and calculus, as both may produce paroxysmal pain and hæmaturia. Although neurasthenia is often associated with movable kidney, the latter is not often the sole cause of the former, and the neurasthenia may persist in all its severity after the successful relief of the movable kidney.

PROGNOSIS.—In the vast majority of cases the condition is not serious, but the pain may be of sufficient severity to interfere with the normal avocations of the patient. Where hydronephrosis is produced more or less serious effects may result.

TREATMENT.—When no symptoms are present no treatment is required. When symptoms are present, relief can, in the majority of cases, be given by the application of a suitable belt or pad, and by measures directed to the improvement of the general health, and especially the tone of the abdominal muscles. Massage and Weir-Mitchell treatment may be of service, but massage is sometimes harmful, and the mobility increased by it. Surgical treatment may be required if the pain be severe and constant, especially if hydronephrosis have resulted. In such cases nephrotomy, nephrorrhaphy or even nephrectomy may be required.

KIDNEY, Morbid Growths of.—Both simple and malignant growths occur in the kidney. The former are of no great clinical importance and include adenomata, fibromata, lipomata, angiomata, and myxomata. In addition to these, gummata, and the growths peculiar to leucocythæmia and lymphadenoma, are not uncommon.

Adenoma is the most common simple growth affecting the kidney, and is usually no larger than a split pea. It may be found in healthy kidneys, but more often in granular kidneys. In some instances it may reach a large size and may be bilateral. An isolated fibroma is sometimes seen in the medullary portion of the kidney, and in rare instances multiple fibromata occur in the capsule

associated with fibromata in other parts, especially the ovary.

The malignant growths commonly affecting the kidney are sarcoma, carcinoma, and malignant adenoma. In addition to these, columnar epithelioma is met with, and even squamous epithelioma, occurring as a primary growth, has been described.

Renal sarcomata are usually of the round- or spindle-celled variety; in a few rare instances alveolar sarcoma has occurred. Sarcomata are met with most frequently in early life, and in a considerable number of the cases the tumour contains striated muscle-fibres; to these malignant sarcomatous tumours the name of rhabdomyoma has been applied. This form of tumour is common in cases of congenital sarcoma, but is also met with in sarcoma occurring in young children. Melanotic sarcoma in the form of secondary growths also occurs in the kidney, and may be accompanied by melanuria. *See* PIGMENTATION; and URINE, Morbid Conditions of.

In most cases of carcinomata affecting the kidney the growth is of the encephaloid variety; scirrhus is only rarely met with. In many instances the encephaloid growth undergoes colloid or other form of degeneration; the growth is usually exceedingly soft and friable.

In a considerable proportion of cases of malignant diseases of the kidney the growth really begins in what is spoken of as a suprarenal 'rest.' These 'rests' are situated in the cortex of the kidney beneath the capsule, and the growths arising in them resemble in their structure that of a suprarenal body, but, owing to the presence of considerable quantities of fat, this may be overlooked. These growths often reach a considerable size, and they may either infiltrate or compress the neighbouring substance of the kidney. These tumours are accompanied by secondary growths, similar in structure, in other parts of the body. The structure of a growth arising from a suprarenal 'rest' resembles that of malignant adenoma more than that of sarcoma. In many cases its clinical course is relatively slow.

Villous tumours of the mucous membrane of the calices and of the renal pelvis sometimes occur, and may reach a very large size.

In about half the cases of malignant growths involving the kidney, the tumour remains confined within the capsule of the organ, even when the growth has reached a very large size; and in this way a more or less general enlargement of the kidney is produced. In the rest of the cases the growth perforates the capsule. The growth often spreads along the renal vein and may produce thrombosis of this vein or of the vena cava. Secondary deposits occur, especially in the lungs, the liver, and in the lumbar glands.

SYMPTOMS.—The more benign tumours of the kidney usually give rise to no symptoms. Cases of adenoma of the kidney have been described in which, owing to extensive cystic degeneration of the growth, effects have been produced resembling both clinically and histologically those seen in cystic disease of the kidney.

Pain, hæmaturia, and the presence of a tumour form the characteristic clinical features of malignant disease of the kidney. The pain is usually felt in the loin, and is liable to undergo exacerbations simulating those of renal colic. In some cases these attacks of pain are dependent on the passage of blood-clot or of fragments of the growth.

Hæmaturia occurs in about one half the cases and may be very profuse; it is usually intermittent. In rare instances fragments of the growth have been detected in the urine. In most cases a renal tumour can be detected, and presents the usual features. It is not unusual for the tumour to be of sufficient size to be visible through the abdominal parietes, and in such cases the superficial veins of the abdomen may be unnaturally distended owing to the interference with the circulation through the vena cava. The tumour presents the ordinary features of a renal tumour. It is limited to one side of the abdomen, has a rounded outline, and moves but slightly with respiration. The presence of the colon crossing the tumour can often be determined either by palpation or by percussion. When the disease is fully established, the characteristic asthenia and cachexia are produced especially in the case of carcinoma. Intestinal obstruction may result from pressure on the colon or from the growth invading some portion of the intestine. In rare instances the disease may spread and involve the vertebral column and produce paraplegia. Thrombosis of the vena cava may lead to œdema of the legs. Ascites may also occur if the peritoneum be invaded.

DIAGNOSIS.—The diagnosis is usually based on the presence of a renal tumour together with hæmaturia and in many cases cachexia. If hæmaturia is absent, the renal tumour may be mistaken for a splenic tumour, for enlarged abdominal glands, for perinephric abscess, or for certain tumours of the liver and ovary. Splenic tumours usually present a sharp edge and notch, and are more movable with respiration than renal tumours; and it is exceptional for the colon to pass in front of a splenic tumour. In some cases a splenic tumour is vertical instead of oblique, and then the difficulty of distinguishing between the two is greater. Even when both hæmaturia and a renal tumour are present, errors may be made, as in one instance where hæmorrhage occurred into the sac of a hydro-nephrotic kidney, producing a solid renal tumour and hæmaturia. Where a tumour cannot be felt the difficulties are considerably greater, as profuse hæmorrhage like that seen in malignant disease may occur with renal calculus and renal tuberculosis, and even in some cases of granular kidney. *See* PHYSICAL EXAMINATION.

PROGNOSIS.—The average duration of carcinoma of the kidney is a year, but in exceptional cases life may be prolonged for several years. In sarcoma the course of the disease is still more rapid.

TREATMENT.—Medical treatment is purely palliative. Morphine is given to relieve the pain. The bowels require especial attention owing to the constipation resulting from the pressure on the descending colon.

If the tumour has its origin in a suprarenal 'rest,' surgical interference is often successful; the removal of the whole kidney is not always necessary, for, in some cases, the growth can be shelled out.

In cases of carcinoma and sarcoma the prospects of a cure by nephrectomy are not so favourable. The results of removal of sarcoma are rather better than those of carcinoma, but recurrence is frequent.

Owing to the doubt that must exist in a large number of cases as to the actual nature of the morbid growth present, an exploratory laparotomy is generally advisable.

KIDNEY, Parasites of.—The parasites which have been described as existing in the human kidneys are hydatids (*see* p. 831). *Strongylus gigas*, *Pentastoma denticulatum*, *Bilharzia hæmatobia*; also the larval form of the *Filaria sanguinis hominis*, which inhabits the lymphatic vessels, either of the kidneys themselves, or of some lower part of the urinary tract.

TREATMENT.—In the treatment of patients affected with *Bilharzia hæmatobia*, the internal use of oil of turpentine is recommended. It is stated that, when the bladder is affected, vesical injections of iodide of potassium, twenty or thirty grains dissolved in tepid water, repeated every second or third day, have been found useful. *See* ENTOZOA; CHYLURIA; and KIDNEY, Hydatid-Disease of.

KIDNEY, Syphilitic Affections of.—Syphilis, like other acute specific diseases, may cause or predispose to certain affections of the kidney. Bright's disease is not uncommon as a complication during the early stages of syphilis. In this respect the influence of syphilis is comparable to that of scarlet fever and other acute diseases, since it may not only lead to the production of Bright's disease, but may also produce a transient albuminuria. When Bright's disease ensues as a sequel to syphilitic infection, the disease runs the same course as when due to other causes. Amyloid degeneration of the kidney is a common result of syphilis, and is sometimes erroneously diagnosed as Bright's disease (*see* BRIGHT'S DISEASE). In rare instances gummata are formed in the kidney, but there is no means of diagnosing their presence.

KIDNEY, Thrombosis of.—Thrombosis of the renal vessels is a rare lesion: venous thrombosis is more frequent than arterial. Thrombosis of the renal vein may occur primarily during the course of debilitating diseases, e.g. diphtheria. It is more usual as a result of extension into the renal veins of thrombi arising in the inferior vena cava. In such cases the thrombosis may start in the veins of the pelvis or of the lower limb, and may gradually involve the vena cava until it reaches the point of entry of the renal veins. Such renal thrombosis is seen in cases of primary septic pelvic diseases, after operations such as ovariectomy or the removal of uterine 'fibroids,' or following the septic complications of childbirth. In rare instances thrombosis spreading from the pelvic veins to the vena cava and renal veins is seen after abdominal operations involving the ligature of pelvic veins without any clear evidence of the presence of septic complications. Thrombosis of the renal veins may also complicate certain renal diseases, especially malignant disease of the kidney and granular contracted kidney. Thrombosis of the renal veins leads to extreme passive congestion of the kidneys and, if bilateral, to complete suppression of urine; but in many instances only one renal vein is involved.

Thrombosis of the renal artery or its branches occurs in very rare instances as a result of the presence of endarteritis. In such cases effects very similar to those seen in embolism are produced, more or less extensive areas of the cortex of the kidney undergoing necrosis. In one instance, recorded by the writer, bilateral thrombosis of the interlobular arteries occurred, producing complete necrosis of the cortex of both kidneys.

KIDNEY, Tuberculosis of.—Renal tuberculosis exists in two forms. (1) In one the organ contains scattered miliary tubercles, and the renal infection is part of a general tuberculosis. In such cases the renal lesion does not develop to any great extent, and the tubercles are always of small size. They are most abundant in the cortex in the vicinity of the capsule, and are usually of a yellow tint. A few scattered tubercles may be found in the medulla. In exceptional instances the individual foci may reach the size of a split pea. In this form of the disease the infecting agent reaches the kidney through the blood-stream, and the bacilli are arrested in the capillary tufts and in the interstitial tissue between the tubules. Bacilli may be present in the urine in small numbers. Miliary tuberculosis of the kidney produces no symptoms, and is of no clinical importance, the lesions in other parts, especially in the lungs and meninges, overshadowing those in the kidney.

(2) In the other form of tuberculosis of the kidney the disease is more localised, but the tubercular foci are more extensive. One or both kidneys may be involved, and no other organ may be affected. More frequently, however, the local tuberculosis is associated with similar disease in other parts of the body, especially of the genito-urinary tract. This is peculiarly the case in males; the epididymis, bladder, vesiculæ seminales, and prostate being most frequently involved. It is to this form of the malady that the expression tuberculous kidney is generally applied.

ÆTIOLOGY.—Tubercular disease of the kidney is more common in children and young people, but many instances are met with in the earlier years of adult life, and the disease is more common in men than in women. In tubercular disease of the genito-urinary tract it is rare for the lower portion of the tract to be alone affected; and, for this among other reasons, it is probable that in many cases the tubercular infection of the kidney is primary, and the involvement of the lower urinary tract, such as the bladder or prostate or ureter, is secondary. In a certain proportion of cases variously estimated by different authorities, the renal lesion is consecutive to a primary focus of tuberculosis in the prostate, epididymis, or some other part of the genito-urinary tract. In some instances the malady spreads by direct continuity from the bladder to the kidney along the ureter, the latter being thickened along its whole course, and its mucous membrane ulcerated. In other cases it would seem that the infection reaches the kidney from the lower urinary tract in a similar manner to that seen in pyelonephritis, namely by the lymphatics. All writers are agreed that in a certain proportion of cases, estimated by some to be as high as 40 per cent., the renal lesion in cases of tuberculosis of the genito-urinary tract is the primary one. For these reasons the co-existence of tubercular lesions in the kidney and genito-urinary tract is frequent.

MORBID ANATOMY.—The renal lesions in tuberculous kidney are very various. In most instances extensive areas undergoing caseation are found in the cortex of the organ involving the medulla by direct extension. These masses are somewhat conical in section and roughly resemble infarcts. The caseation that the central parts undergo leads to the formation of cavities with shreddy walls. The number and size of these areas present great variation. Their contents may undergo calcifica-

tion. In most instances the mucous membrane of the pelvis of the kidney is also affected and contains tubercles. As a result of their caseation, ulcers of varying size are formed. The pelvic mucous membrane generally is greatly thickened, and this may be sufficient in amount to cause obstruction to the exit of urine, and thus lead to the formation of a pyonephrosis. The ureter may also become blocked by the detachment of fragments of the tuberculous tissue, or in some instances by the thickening produced by tubercular changes in its wall at some distance from the pelvis of the kidney. In this way also a hydro- or pyonephrosis may be produced. Occasionally fibroid changes take place, leading to the atrophy of the kidney and the encapsulation of the tuberculous tissue contained in it. Even where the disease is primary in the kidney, other organs soon become affected, such as the abdominal glands, the lungs, and occasionally the intestines.

SYMPTOMS.—The deposit of tubercle in the kidney may lead to moderate enlargement of the organ quite apart from the development of such complications as pyo- or hydronephrosis, and in such instances a renal tumour may be detected. More often the enlargement of the kidney is slight in amount, and on palpation pain and tenderness are more frequently detected than the presence of any notable enlargement of the organ. The symptoms of pyelitis are usually present, owing to the frequency with which the tubercular changes involve the renal pelvis. The symptoms of tubercular pyelitis resemble in many respects those of calculous pyelitis; but the frequency of micturition is often extreme, and not so definitely related to movement. Thus it may be quite as marked by night as by day. Pain is commonly present, and is usually constant in the lumbar region. Exacerbations may occur from time to time which may be due to the temporary blocking of the ureter by fragments of the tuberculous tissue. Pus in variable amount is present in the urine. The amount of albumen is usually related to the quantity of pus present, but it is not uncommon for amyloid disease of the kidney to exist as a complication, and in such cases the albuminuria is greater. Hematuria is often present during some period of the illness, and is usually scanty in amount; but it is not very exceptional for profuse hematuria to occur from time to time. The general effects that are usually seen in tuberculosis of other organs, such as emaciation, sweating, and pyrexia, are commonly present. Examination of the urine will frequently reveal the presence of tubercle-bacilli, and their presence may not only be detected by the ordinary methods of staining, but also by the inoculation of susceptible animals with the suspected urine. In cases of doubt the latter is the better procedure, as difficulties are sometimes experienced in distinguishing with accuracy between the *Bacillus tuberculosis* and the *Smegma-bacillus*. Death may be due to uræmia, or sometimes to suppression of urine; but in a large proportion of cases it is dependent on the extension of the disease to other parts, more especially the lungs and the intestine.

DIAGNOSIS.—In most instances the disease will present itself with the symptoms of pyelitis, and the diagnosis may be made by the detection of the bacilli in the urine, or by the evidence of tuberculosis of the genito-urinary tract, such as the epididymis, prostate, and, in women, the thickening of the ureter. In cases where blocking of the ureter

has led to distension of the kidney some difficulty may be experienced in distinguishing between simple pyonephrosis and one complicating tubercular disease.

PROGNOSIS.—The prognosis is very grave, although where the disease is strictly unilateral success has followed the removal of the affected kidney. Catheterisation of the ureters may afford useful evidence as to the presence of unilateral disease. Where the malady is fully established, life is not usually prolonged for more than a few months; but cases have been known to live as long as three years after the nature of the disease has been recognised.

TREATMENT.—Where there is clear evidence of the disease being limited to one kidney nephrectomy is advisable, but in other cases treatment can only be directed to the relief of the pain and the promotion of the general health, such as are employed in the treatment of tuberculosis elsewhere.

JOHN ROSE BRADFORD.

KINÆSTHESIS (κινέω, I move; and αἴσθησις, sensation).—The sense by which we appreciate the character and extent of voluntary movements.

KINETOPLASMA.—The granules in a motor-cell which are stained by basic dyes, and often known as a chromophile or chromatic substance.

KING'S EVIL.—A popular name for scrofula, originating in a belief formerly held that the disease could be cured by the king's touch. See SCROFULA.

KISSINGEN, in Bavaria.—Common salt waters. See MINERAL WATERS.

KLEBS-LÖFFLER BACILLUS.—A synonym for the *Bacillus diphtheriæ*. See DIPHTHERIA.

KLEPTOMANIA.—Insanity characterised by an irresistible impulse to steal. See INSANITY, Impulsive; and CRIME, Irresponsibility for.

KOPF-TETANUS.—A local manifestation of tetanus occurring in cases in which the face is injured, and attended by trismus and facial paralysis. Later on the general phenomena of the disease supervene.

KOPLIK'S SPOTS.—Small red spots, with pale blue centres, sometimes observed on the mucous membrane of the mouth during the pre-eruptive stage of measles. See MEASLES.

KRA-KRA.—SYNON.: *Craw-craw*.—A word used by negroes to denote any form of skin-eruption characterised by much local inflammation, and especially by the presence of pustules.

KRAUROSIS (κραῖρος, dry).—An atrophic condition of the female generative organs, accompanied by intense itching of the affected parts.

KREUZNACH, in Germany.—Common salt waters containing iodine. See MINERAL WATERS.

KROUOMANIA (κρούω, I shake).—This term is sometimes applied to co-ordinated movements, rhythmically repeated, and found principally in 'habit-spasms' in feeble-minded children.

KYPHOSIS (κυφός, bent).—A synonym for angular deformity of the spine. See SPINE, Diseases of.

L

LABIO-GLOSSO-LARYNGEAL PARALYSIS

(*labium*, a lip; *γλῶσσα*, the tongue; and *λάρυγξ*, the throat).—There are two forms of this disease, which have to be considered separately, namely (A) a **chronic** form; and (B) a **sudden** form—the latter is often termed ‘acute,’ but is, as a rule, sudden in onset. (C) A true **acute** (not sudden) variety is met with in rare instances. A fourth variety, **pseudo-bulbar** paralysis, entirely different in seat, needs to be distinguished, but will be afterwards described.

(A) **Chronic Labio - Glosso - Laryngeal Paralysis.**—SYNON.: Bulbar Palsy; Duchenne's Paralysis; Fr. *Paralysie Glosso-labio-laryngée*; Ger. *Progressive Bulbarparalyse*.

DEFINITION.—A progressive symmetrical paralysis of the lips and adjacent facial muscles, of the tongue, pharynx, and sometimes also of the larynx; with or without conspicuous wasting; and often associated with muscular atrophy elsewhere.

ETIOLOGY.—Of the causes of the affection little is known. It is a disease of later life, being almost unknown under forty. Exceptional cases have been met with in early adult life, and even in late childhood. In the latter instances several members of a family have been known to suffer, and a congenital tendency to early failure of vitality in the structures concerned may reasonably be assumed. In the common form, males are affected more frequently than females. The disease, as such, does not appear to be inherited, but in many cases there is a family history of other affections of the central nervous system. Of immediate causes, exposure to cold, mental anxiety, and defective nourishment have been supposed to exert an influence.

ANATOMICAL CHARACTERS.—When, as is commonly the case, there is wasting of the paralysed parts, their muscles are reduced in size, and present changes similar to those met with in the limbs in chronic spinal atrophy. Their substance is pale; fatty tissue may be in excess; and the muscular fibres often, but not always, present granular degeneration. They are frequently narrowed, and the tissue between them may be increased in quantity, and may contain pigmentary and other products of degeneration, and an increased number of nuclei. When there is no apparent wasting, this may be merely because the interstitial fat is increased so as to compensate for the reduction in bulk of the muscular fibres. Rarely the structure of the muscles presents little alteration. The motor nerve-fibres are grey, translucent, and, under the microscope, degenerated. Their nuclei of origin in the medulla oblongata are also diseased. The motor cells are shrunken and atrophied; their processes are lost; and the intermediate tissue is degenerated. The hypoglossal nucleus is examined most readily, and the change in it is striking, as well as in the nucleus of the spinal accessory (palate and larynx); changes may often also be seen in the nuclei of the glosso-pharyngeal, the vagus, and the ‘nucleus ambiguus.’ The anatomical alteration which underlies the affection of the lips has not yet been detected. There is still uncertainty regarding the precise origin of this part of the facial nerve. Most of its fibres diverge from the middle line, close

to which the hypoglossal nucleus is situated, and some at least pass towards the lower part of the column of cells, which, above, gives origin to the motor fibres of the fifth nerve. It is certain, however, that many fibres of the facial descend towards the level of the hypoglossal nucleus. Further, the physiological association of the movements of the tongue and lips is most close. We cannot narrow the tongue without contracting the orbicularis. Hence it is certain that the nucleus for the lower facial muscles and the hypoglossal nucleus, whether blended, contiguous, or distant, are closely connected and are alike predisposed to disease, so that this connected part of the facial nerve suffers in the same manner as the hypoglossal. In the same manner the fibres for the orbicularis palpebrarum suffer with those for the ocular muscles. The fibres passing to the nuclei from the cerebral hemispheres may also be degenerated, and it is probable that these are alone affected in the cases in which there is no wasting in the muscles. When there is muscular atrophy in the limbs, a corresponding degeneration may be found in the anterior cornua of the spinal cord, often conjoined with sclerosis in the lateral columns, and in the anterior pyramids in the medulla. There is every reason to believe that, in the chronic form, the atrophic changes in the nerve-elements are the primary alteration.

In very rare cases, no morbid appearance has been observed in the medulla. Some of these may have been cases of peripheral neuritis, local and degenerative. In others, in which even the nerve-endings were normal, the degeneration of cerebro-nuclear fibres may have escaped observation, or some toxic influence may have abolished function without causing destructive degeneration.

SYMPTOMS.—The symptoms have the distribution indicated by the name given to the disease by Trousseau, the affected parts being the lips, tongue, throat, and larynx. They are, so to speak, arranged about the tongue as a centre. It is in this organ that the earliest symptoms commonly present themselves, as a trifling indistinctness of speech, due to an imperfect articulation of those sounds in which the tongue is most concerned—especially the dental and palatine explosive sounds, in which the tongue has to be pressed against the teeth or hard palate, *t, d, k, g* (hard), &c. The tongue can be still protruded, although perhaps not quite so far as normal. The lips then become weak, and sounds in which the lips are concerned are imperfectly articulated. The vowels *o* and *oo*, in the pronunciation of which the orbicularis contracts so as to protrude the lips, cannot be well sounded. The lips are not brought together so perfectly, or separated so promptly, as in health, and the labial explosives, *b* and *p*, become *f*, and so does the labial resonant *m*. Whistling is impossible. The lower part of the face loses its expression, the lips are habitually separated, and the saliva cannot be perfectly retained. The difficulty in articulation is soon increased by the weakness of the palate, which ceases to shut off the nasal cavity, so that a nasal resonance accompanies sounds from which it should be absent. The paralysis of the tongue increases until the organ can no longer be protruded. Deglutition becomes impaired, partly

from the weakness of the tongue, but in part also from that of the constrictors of the pharynx. The soft palate ultimately hangs motionless, or, if it can be raised a little in an attempt to phonate, during the act of swallowing does not close the posterior nares, so that liquids regurgitate into the nose. Food is apt to lodge in the upper part of the pharynx, and crumbs or liquid to get into the larynx. The laryngeal muscles subsequently become weak, and the glottis cannot be closed. The vocal cords may be equally affected, or one may be paralysed in greater degree than the other. Coughing is necessarily imperfect: air is driven through the larynx, but there is no sudden opening of a previously closed glottis, and hence no explosive cough. In proportion as the glottis is paralysed, phonation is interfered with, but the ability to produce some sound is rarely altogether lost. As the disease progresses, the speech becomes almost unintelligible, being reduced to unarticulated and scarcely modulated vocal sound. It is to be noted, however, that for a long time the habitual articulation is rarely the best possible. Words can be distinctly articulated by a deliberate effort which are scarcely at all articulated in ordinary speech. The saliva can neither be swallowed nor retained within the mouth, and is constantly dribbling over the lower lip, below which the patient has to hold a handkerchief continually. It has been thought that the quantity of saliva is increased, but the evidence of this is insufficient. The condition of the tongue varies much in different cases. In some it is large, broad, flabby, and soft to the touch throughout. In others it is conspicuously wasted, and covered with wrinkles and furrows from the shrinking. In some cases the lips retain their normal size; in others they are distinctly thinner than normal. This striking contrast between different cases (already referred to) led Duchenne to distinguish two varieties, the *atrophic* and the *paralytic*, but in some of the cases of the latter class it is merely the increased interstitial fat that prevents wasting from being conspicuous during life. In the affected muscles the electrical irritability is usually little changed: they still contract to the faradic current, even when the atrophy is conspicuous. Sometimes there is an indication of the reaction of degeneration, in undue readiness of contraction to the anodal (positive) closure, or this reaction may be well marked, faradic irritability being much lessened. Other muscles of the head are rarely affected. Those in the upper part of the face always escape, and the zygomatic muscles, with the elevators of the upper lip, are so little affected that their unopposed contracture produces a peculiarly deep and characteristic naso-labial furrow. In many cases muscular atrophy in the limbs, in greater or less degree, is associated. So, too, in cases of ordinary muscular atrophy, commencing in the limbs, the lips, tongue, and throat are often affected towards the end of the case, in the same manner as in the primary form, but in less degree.

Death, in labio-glossal paralysis, is usually the result of asthenia, due, it may be, in part, to the difficulty in deglutition, or it is the result of chronic lung-disease produced by the repeated entrance of particles of food into the air-passages. Sometimes the patient dies in a paroxysm of coughing, occasioned by an ineffectual attempt to swallow liquids or saliva.

DIAGNOSIS.—Before labio-glossal paralysis was well known, the difficulty in swallowing was as-

cribed to a chronic inflammation of the fauces, but this mistake is now scarcely possible. Yet the writer has known it to be mistaken for cancer of the throat. The symptoms have to be distinguished from those due to other diseases of the medulla, and to disease elsewhere. Many sudden lesions, in the region affected in this disease, may cause similar symptoms, but these are distinguished by their onset. Compression of the medulla may also give rise to symptoms of similar distribution, but these are commonly unilateral, or one side suffers before, and more than, the other; and they are often accompanied by a preponderant affection of the muscular part of the spinal accessory nerve, and by great weakness, without wasting, in the limbs. In 'pseudo-bulbar paralysis,' movements of the tongue are impaired by disease of the cerebral hemispheres. These movements are represented in the lower part of each ascending frontal convolution. When this region is diseased on one side, the loss is soon compensated for by the centre in the opposite hemisphere, but a symmetrical bilateral lesion in this situation may cause complete paralysis of the tongue and other parts affected in the disease now described. Such paralysis is acute in onset so far as its complete degree is concerned, and therefore is chiefly liable to be mistaken for the sudden form, presently to be described.

PROGNOSIS.—The prognosis is always grave. The chronic disease consists in a slow degeneration of the nerve-elements, the effects of which often lead to death in about a year from the onset. Although, in some cases, a temporary arrest may be obtained, and occasionally the malady, after causing a considerable degree of disability, is arrested for several years, it is doubtful whether, in any instance of this form, definite improvement has been produced. The prospect of prolongation of the state of arrest, if this occurs, depends very much on the amount of care the patient can secure. The older the patient, the less is the likelihood of arrest.

TREATMENT.—The degenerative tendency which underlies chronic labio-glossal-laryngeal paralysis is usually beyond the reach of remedies. It is essentially a senile failure of nutrition, local and premature, and as little amenable as are other manifestations of senile decay. Therapeutical efforts must be chiefly directed to the endeavour to retard it, by securing freedom from any cause that can be traced, especially from depressing emotion, overwork and the like, by attention to hygiene, improving the general health, and by the administration of nerve tonics, quinine, strychnine, arsenic, nitrate of silver, and the like. Strychnine is certainly most useful when given hypodermically, one injection being given daily, in any convenient place. The nitrate of strychnine is the salt for the purpose; $\frac{1}{50}$ gr. may be gradually increased to $\frac{1}{20}$ gr. It is less effective than in progressive muscular atrophy, probably because the latter often occurs earlier in life.

Electricity may be tried, although it is generally unsuccessful. Erb has recommended the application of the voltaic current through the throat, the positive electrode being placed on the back of the neck, the negative stroked down the side of the pharynx externally, and such a strength being employed as shall produce reflex movements of deglutition; but it is difficult to discern any influence. Change of air is desirable in the early

stage, and rest is imperative. The patient should be discouraged from ineffectual attempts to talk. Food must be carefully regulated, easily digestible varieties being reduced to a semi-solid condition. In the later stages, should swallowing be impossible, nourishment must be administered by an oesophageal tube, or, what is better, by a catheter introduced through the mouth or nose, through which liquid food is given by means of a funnel.

(B) **Sudden Labio-Glosso-Laryngeal Paralysis.**—**SYNON.** : Acute or Apoplectiform Bulbar Paralysis.

DEFINITION.—Paralysis of similar distribution to that of the chronic form, with or without conspicuous wasting, of sudden onset. It is always due to a vascular lesion, usually thrombosis.

ÆTIOLOGY.—The causes of this affection are for the most part those which lead to sudden lesions elsewhere in the brain, especially degenerations of vessels, atheroma, and syphilitic disease. Very rarely injuries have been the apparent cause. It is a disease chiefly of late life.

ANATOMICAL CHARACTERS.—Little is known of the morbid changes in cases of sudden onset which have recovered with persistent symptoms. In cases which have died rapidly, foci of softening in the medulla have been found; and there is reason to believe that such softening, from vascular occlusion, is the general cause of the affection. Usually there is disease of one or both vertebral arteries, and closure of one or more branches which come off from the diseased part. Sometimes the diseased vertebral is much larger than the other, and its branches have taken the larger share in the blood-supply to the nuclei. Probably a small hæmorrhage may also give rise to it, but an extravasation here is seldom survived. The frequent symmetry of the symptoms in the sudden form, and their limitation to the parts which are affected in the chronic disease, show the close relation of the central structures, owing to which they are all affected by the one lesion: this is probably closure of one of the central branches. The wasting in some of the cases of this variety is very much less than that which follows an acute lesion of motor grey matter elsewhere. It is possible that the ascending fibres from the nuclei concerned pass up close to the middle line, in a situation in which all may be damaged by a single lesion.

SYMPTOMS.—The onset is sudden, often preceded by headache and giddiness, rarely attended by loss of consciousness. The patient suddenly finds a difficulty in swallowing and in articulation, with inability to protrude the tongue. The symptoms commonly attain their maximum in the course of a few hours or days. Respiratory disturbances may be present—cough, dyspnœa, and hiccup. Convulsions and weakness in the limbs, sometimes with tingling, occasionally occur at the onset of severe cases. Many, which present these symptoms, die in the course of a few hours. In those which recover, paralysis of the tongue, lips, throat, and larynx may remain, resembling that which characterises the chronic form, and there may or may not be conspicuous wasting. In many cases, however, the symptoms are incomplete, some part escapes, or the bilateral symmetry is imperfect. This we should expect from the more random character of the lesion. The disease differs from the chronic form also in 'not being progressive.

Unless a fresh sudden attack occurs, the patient may remain in the same condition for a considerable time, and may even improve. It is rare, however, for improvement to be considerable. A stationary condition of considerable palsy sometimes continues for months, rarely for a year or more. It is more common for other slight attacks to occur as other branches of the diseased artery become occluded.

DIAGNOSIS.—The diagnosis of the acute form of labio-glosso-laryngeal paralysis calls for little remark. It must be remembered that the symptoms may deviate from the type more than in the chronic variety, as the lesion sometimes produces irregular effects. It is chiefly liable to be confounded with the impairment of movements of the palate and tongue sometimes left after double hemiplegia, from which the history suffices to distinguish it, the two attacks of hemiplegia usually occurring at different times, and the hemiplegic symptoms being pronounced. The rare symmetrical affection of the cortical centres for the tongue, mentioned above, is also not simultaneous on the two sides, and it is associated with at least transient hemiplegic weakness much more obtrusive than that which accompanies the bulbar affection.

PROGNOSIS.—If the patient recovers from the immediate effects of the lesion, the prognosis of the paralysis of the lips, tongue, and other parts is better than in the chronic form, inasmuch as recovery of slightly damaged structures may lead to some restoration of power. The prognosis is also better if there is any reason to ascribe the mischief to syphilitic disease. Nevertheless, in some acute cases the paralysis remains in its initial degree, although even in these there is not the tendency to increase which is seen in the chronic variety.

TREATMENT.—Any causal indication must be carefully sought for in acute labio-glosso-laryngeal paralysis, and treated, especially evidence of syphilis. In other conditions the treatment is that for the vascular disease which is its cause, and which has been already described. Electrical treatment has little influence, nor can we anticipate the possible good from the hypodermic injection of strychnine which is occasionally obtained in the chronic form. The remarks regarding diet and feeding in the chronic form are equally applicable to the acute variety.

(C) **Acute Myelitic Bulbar Paralysis.**—**SYNON.** : Myelitis Bulbi.

In rare cases, symptoms of bulbar paralysis, as above described, come on in the acute (not sudden) manner characteristic of central inflammations, and depend on this process in the grey nuclei which are the seat of degeneration in the chronic form. Such cases, when the lesion is limited to this region, are too rare to make it necessary to do more than mention their occurrence. The process is analogous to spinal polio-myelitis, and the principles of its treatment are the same. It is more common for such an onset of bulbar symptoms to follow those of an ascending inflammation of the cord. They may also occur as the final stage in the mysterious disease, 'acute ascending paralysis,' in which it is probable that a toxic agent in the blood acts on certain parts of the nerve-centres and abolishes their function.

(D) **Pseudo-Bulbar Paralysis.**—The fact has been already mentioned that bilateral lesions of the cerebral hemispheres may cause symptoms

closely resembling those produced by disease of the nuclei of the medulla. This effect is due to the fact that the muscles paralysed, being bilateral in use, are represented in both hemispheres, and can be innervated from either; hence compensation by the other side prevents serious paralysis from a unilateral lesion; but if disease afterwards occurs in the part affecting the compensation, paralysis results such as is caused by disease of the medulla, or rather (since there is no wasting) by disease of the fibres from the cortex to the bulbar nuclei, homologous with those of the pyramidal tracts. The lesions causing pseudo-bulbar paralysis are usually in the cortical centres, rarely in the internal capsule, or in the outer parts of the lenticular nuclei, where they probably implicate the fibres in the white substance from the centres in the lower part of the motor region related to the lips, tongue, &c. The lesions may be of any kind, but are generally sudden and due to vascular disease causing symmetrical thrombosis. Hemiplegia usually attends the onset, and constitutes a distinguishing feature of these cases.

W. R. GOWERS.

LACRYMAL APPARATUS, Diseases of (*lacrima*, a tear).

The lacrymal apparatus consists of the gland, with its excretory ducts; and of the puncta, the canaliculi, the lacrymal sac, and the nasal duct, through which the secretion is conveyed into the nose. The diseases of this apparatus are almost limited, with the exception of growths affecting the gland itself (*see* ORBIT, Diseases of), to the excessive secretion of tears, and to impediments to their escape into the nose. To excessive secretion, or to impeded outflow, the common term *epiphora* has been applied; but the great majority of cases of epiphora are due to the latter of the two causes.

Epiphora.—*Excessive secretion of tears* is, in some cases, an affection for which it is not possible to discover an adequate cause, and it may perhaps be sometimes due to the prolonged operation of emotional influences. In most instances, however, it is associated with some kind or degree of conjunctival irritation, and is to be regarded only as a reflex phenomenon hence arising. It is well known that any temporary or accidental irritation, such as may arise from the intrusion of a foreign body into the conjunctival sac, is apt to be followed by a copious secretion of tears, which assist in dislodging the offender; and irritations of a more chronic kind, produced by congestion or irregularity of the lining membrane of the lids, may have a similar effect.

Impediments to the escape of the tears, causing them to collect in the conjunctival sac, or even to flow over the cheek, may depend upon displacement of the puncta, so that these apertures are no longer applied to the conjunctival surface, from which they normally remove superabundant moisture by capillary attraction. Such displacements affect chiefly the punctum of the lower lid; and may be consequent either upon conjunctival swelling, by which the lid is pushed away from the eye, or upon paralysis or weakness of the orbicularis muscle, which allows the lid to fall by the action of gravity. In some cases epiphora will depend upon obliteration or occlusion of the puncta. The former condition is incurable; the latter may be produced by plugs of inspissated mucus, which may be removed by the careful employment of a probe.

But the most ordinary cause of obstruction is stricture of the nasal duct, in which condition the tears are arrested a little below the sac, and the sac consequently becomes over-distended. In such cases the sac can be seen and felt as a small lump situated just beneath the *tendo oculi*. When pressure is made upon this lump, a fluid, consisting of tears mixed with more or less mucus or muco-pus, will regurgitate into the eye, and the lump itself will disappear. The danger in such cases is that the continued distension of the sac and the intrusion of bacteria will in time excite inflammation of the lining membrane, leading to the formation of pus, and this to an opening upon the cheek, producing what is called a *lacrymal fistula*. Such an opening never heals until the duct is again pervious, and it is liable to undergo periodic attacks of unsightly inflammation.

TREATMENT.—In all cases of lacrymal hypersecretion, the first thing to be done is to search under the lids for any concealed foreign body which may be lurking there. If none be detected, examination must be made for conditions likely to be irritating; and they are to be treated, if they exist, by mild astringent or other suitable local applications. There is probably no medicine which can be said to exert any positive effect in diminishing the amount of the lacrymal secretion.

If the displacement of the lid can be cured by treatment addressed to its causes, the tears will usually return to their accustomed channel. If the displacement be incurable, as happens in some cases of paralysis of the *portio dura*, or of chronic ectropion, the patient may often be relieved by slitting open the canaliculus as far as the caruncle, so as to carry back the aperture to the secretion which it is designed to remove.

The treatment of stricture of the nasal duct can often be only palliative. The patient should acquire the habit of emptying the distended sac by finger-pressure many times a day, and of wiping away the fluid; while, to diminish the irritation of the mucous membrane, a drop of any mild astringent lotion may be applied to the inner corner of the conjunctiva two or three times a day, immediately after such pressure has been made. Perhaps the lotion most generally suitable for this purpose is a solution of acetate of lead in distilled water, of a strength not exceeding three grains to the fluid ounce.

When a radical cure is desired, the canaliculus must be slit up, and the patency of the duct restored by the passage of probes through the stricture. If fistula has already formed, or even if the sac is the seat of an abscess, no other plan is available; but for the necessary details the reader is referred to works on ophthalmic surgery.

R. BRUDENELL CARTER.

LACTATION, Disorders of (*lacto*, I give suck).—**SYNON.**: *Fr. Troubles de la Lactation*; *Ger. Störungen der Milchdrüsen*.—The disorders of lactation are numerous. Sometimes the quantity of the lacteal secretion is excessively small and quite inadequate for the support of the child. At other times it is so abundant that the milk will flow from one nipple as the infant is sucking the other; and when the child is removed from the breast, the secretion continues from both sides. The term *agalactia* is applied to the former, and *galactorrhœa* to the latter condition.

1. **Agalactia** (ἀ, priv. ; γάλα, milk).—Agalactia signifies either a total suppression of the mammary secretion, or a very scanty supply. It results generally from anæmia and general debility.

The treatment should be directed towards improving the health of the patient as much as possible, by a generous and judicious diet, and tonics, particularly those containing iron. Recent physiological observations show that a proteid diet has a marked effect in increasing the secretion of milk. Certain drugs have been employed as galactagogues, and, it has been said, with benefit. The leaves of the castor-oil plant, boiled, have been used as a local application—the liquid for fomentation, and the leaves as a poultice ; and a strong decoction of the same plant has been given as a drink. It is doubtful, however, whether such remedies are efficacious. See GALACTAGOGUES.

2. **Galactorrhœa** (γάλα, milk ; ῥέω, I flow). This occurs in two forms. In the one the composition of the milk is normal, but the quantity excessive ; in the other form the increase in the bulk of the secretion is due to a preponderance of the watery part of the fluid.

The chief remedies employed to reduce the amount of the mammary secretion are belladonna and iodide of potassium. Belladonna is employed as an outward application, as well as administered internally. The extract rubbed up with glycerine may be spread on lint, and thus applied to the breasts, or the Emplastrum belladonnæ may be used. The child should not be put to the breast too frequently. If the excessive secretion continue for any length of time, great emaciation may result ; and to this condition the term *mammary diabetes* has been applied. Under such circumstances lactation should be entirely stopped as soon as possible. Strapping the breasts tightly immediately after they have been emptied is of use. Every care must be taken to avoid the formation of a mammary abscess ; and if the breasts get hard and knotty a breast-pump should be employed to free the tubes.

3. **Depressed Nipples**.—Depressed nipples are generally produced by the pressure of stays. If this condition be observed during pregnancy, periodic attempts should be made to draw the nipples out by means of a glass nipple-shield, to which an india-rubber tube and teat is attached.

4. **Fissures and Excoriations of the Nipples**.—These often lead to abscess, and it is said that they may sometimes end in malignant disease. To avoid the occurrence of these lesions, astringents should be applied to the nipples during pregnancy, in order to harden them. Eau-de-Cologne and water, brandy and water, or a weak solution of tannin, may be employed for this purpose. Or the nipple may be anointed with pure vaseline, lanoline, or cacao-butter, and gently manipulated between the finger and thumb. This is highly recommended by some.

Sometimes an abrasion on the surface forms an ulcer or a crack at some part of the nipple, most frequently at its base, which gives rise to great pain during suckling. The remedies for these cracks are astringent applications, such as tannin, flexible collodion, a weak solution of nitrate of silver or of carbolic acid. Care should be taken to sponge these away before the infant is again put to the breast ; and a nipple-shield with an india-rubber teat will be found of great service.

For abscess and other morbid conditions of the

mammary gland supervening during lactation, see BREAST, Diseases of ; and NIPPLE, Diseases of. CLEMENT GODSON.

LAGOPHTHALMOS (λαγώς, a hare ; and ὀφθαλμός, the eye).—This term is derived from an old supposition that the hare sleeps with its eyes open ; and is applied to a condition in which there is inability to close one or both eyes. Lagophthalmos may be due to paralysis of the orbicularis muscle, in which case it will be attended by falling of the lower lid, and will generally be associated with paralysis of other muscles supplied by the seventh nerve (see FACIAL PARALYSIS) ; to the contraction of cicatrices ; to spasm of the upper eyelid (see THIRD NERVE, Diseases of) ; or possibly to congenital malformation of the lids. In paralytic lagophthalmos, the treatment is that of facial paralysis. Where there is contraction or deformity, each case must be considered on its own merits, with regard to the possibility of obtaining relief from a surgical operation. R. BRUDENELL CARTER.

LALLING.—The term used to characterise the speech of children before they have learned to pronounce their words quite intelligibly and such defects of speech as may resemble this (Kussmaul).

LANDRY'S PARALYSIS.—See SPINAL CORD, Diseases of—Acute Ascending Paralysis.

LANGEN - SCHWALBACH, in Nassau.—Chalybeate waters. See MINERAL WATERS.

LARDACEOUS DISEASE (*lardum*, bacon).—A synonym for amyloid disease, which is so called from the supposed resemblance of the cut surface of an affected organ to raw bacon. See AMYLOID DISEASE.

LARYNGEAL PHTHISIS.—A term applied to laryngeal tuberculosis. See LARYNX, Diseases of ; and PHTHISIS.

LARYNGISMUS STRIDULUS (*larynx*, the windpipe ; *stridor*, a noise).—A form of obstructed breathing, attended by a peculiar stridor or crowing sound during inspiration, and dependent on spasm of the muscles of the glottis. See LARYNX, Diseases of.

LARYNX, Clinical Examination of the.—Successful examination of the larynx depends, like all other objective investigations, upon practice and suitable appliances. The first essential is a suitable light, placed upon one side of and behind the patient's head, so that it can be reflected into the throat from a concave mirror of 15" focal length adjusted over the eye of the observer, on the same side as the light, and in such manner that, while he gets clear vision through the hole in the centre, his eye is protected from the glare of the lamp. A bull's-eye lantern with a black chimney is generally used in this country, though on the Continent an ordinary Argand gas-burner with clear glass chimney is preferred. But as the quantity of light required is in direct proportion to the illumination of the room, it follows that the darker the latter the more easily will the interior of the larynx be revealed. A candle in a dark room will often be more successful than the lime-light in a chamber brilliantly lighted. The design of the reflecting mirror is not of much consequence, though most specialists prefer one or

other form of the spectacle frame, if fitting truly, as it is more quickly placed in position, while its pressure is less irksome than the kind adjusted with a head-band. In any case the central orifice must be so placed directly in front of the eye that, without any turning of the head to one side, perfect vision is obtained. By closing each eye in succession, with the reflector in position, it may be at once ascertained whether both eyes are directed upon the region under examination, thus securing the advantage of double vision.

The laryngoscope itself consists in the small pharyngeal mirror, in which the view of the larynx is obtained while the light is thrown downwards by the same means. The mirror should be adjusted to the stem at an angle of about 120° ; but the stem should be sufficiently flexible to enable the angle to be altered at pleasure. Three or four sizes should be in use, the smaller generally being preferable, partly because they can be placed further upwards and backwards under the uvula, and partly because, in that they do not irritate so large a surface, they produce less tendency to reflex action. The latter is the real difficulty in making an examination, and only practice will enable the observer so to place his instrument as to produce the minimum amount of irritation. No rules will help the beginner very much. Suffice it to say that, after drawing the patient's tongue out by holding it between the finger and thumb of the left hand, and thereby keeping the head fixed in one position, slightly tilted backwards, the laryngoscope must be placed as far back as possible, doing so gently and firmly and without touching the back of the tongue, the aim being to lift upwards the tip of the uvula with the further edge of the mirror, so that the latter may be thrust as far back as possible. The mirror must be held as nearly horizontally as is consistent with obtaining the reflection from its surface, this being often facilitated by tilting the patient's head further upwards. In consequence of the importance of being able to alter the inclination of the head, it is obviously a mistake to allow the patient to hold the tongue out himself; indeed this should be done only when the observer's second hand is needed for making an application to the larynx, or for inserting an instrument. Occasionally even with the utmost skill and patience it is impossible to obtain a view in the manner described, chiefly in consequence of the elevation of the back of the tongue. In this case it will sometimes prove of great help to depress the tongue with a tongue depressor instead of pulling it out: thus a view may be obtained of the laryngeal structures when the patient utters the sound 'A' or 'Ah,' though the view thus obtained is seldom more than momentary. In other cases the difficulty arises from an exaggerated degree of irritability of the pharynx, so that, the moment tongue or pharynx is touched, contraction of the constrictors takes place, rendering a view of the structures below impossible. Practice will often overcome this difficulty, especially if the patient presents himself with an empty stomach. Brushing the pharynx with a 5 or 10 per cent. solution of cocaine is often recommended, although the extreme discomfort thereby induced often intensifies the patient's nervousness, while at the same time it lessens the local sensitiveness. Consequently a local anæsthetic should be used only as a last resource. The most difficult part of the larynx to discover is the anterior commissure and

anterior wall of the trachea. Assistance is gained by instructing the patient to phonate the sound *E* rather than *A*, though, with tongue held out of the mouth, a consummation of the attempt is not possible. At any rate the epiglottis is thereby raised and brought forwards and the glottis more completely exposed. Further help is sometimes gained by increasing the backward inclination of the head and then making the plane of the laryngeal mirror more horizontal with the plane of the glottis. Often by making a patient laugh or cry the inspection of the anterior commissure is facilitated.

During an examination every structure capable of being thus revealed must be passed in review, and frequently, especially with an expert, every point of importance can be grasped in a momentary examination. The beginner, however, must expect to find several inspections necessary before he has mastered all the points of importance; at the same time he must remember that the more often he makes his examination at the same sitting the more irritable the patient's throat will become. The vocal cords will always be the landmarks from which we estimate our success in obtaining a proper view, their normal pearly white, or, in adult males, pink colour, making them very conspicuous; but where they are so greatly swollen and injected as to make their nature difficult of recognition, it is sometimes troublesome to find contiguous structures accurately. Again, the supra-glottic regions may be so much swollen as to conceal the true cords, a condition which adds to our difficulties. Besides the true cords, the false cords or ventricular bands, the aryænoïd cartilages, the aryæno-epiglottic folds, and the epiglottis must be passed in review. Nor must it be forgotten that the base of the tongue and lower portions of the pharynx are as fully revealed by the laryngoscope as the larynx proper, and that their omission from examination often betrays the inexperience of the laryngoscopist.

GREVILLE MACDONALD.

LARYNX, Diseases of.—The principal diseases and disorders which affect the larynx may be conveniently enumerated and described in the following order:

1. Disorders of Circulation, p. 845.
2. Inflammation, p. 846.
3. Leprosy, p. 848.
4. Lupus, p. 848.
5. Nervous Affections, p. 848.
6. Spasm, p. 850.
7. Syphilis, p. 850.
8. Tuberculosis, p. 851.
9. Tumours, p. 853. See also CROUP; DIPHTHERIA; PAIN IN VISCERAL DISEASE; and TRACHEA, Diseases of.

1. Larynx, Disorders of Circulation of.—*Anæmia* of the larynx does not exist as a separate disease. Like pallor of the gums, it is described as one of the symptoms of incipient phthisis.

Congestion of the larynx requires a passing notice. Hyperæmia of some portion of the mucous lining of the larynx results from over-exertion of the voice, exposure to cold, the action of irritants, or any cause obstructing the circulation through the larynx; and it may be consecutive to laryngitis. It causes more or less persistent hoarseness, and a sense of discomfort in the throat; and it excites what is commonly called a 'tickling cough.' Congestion is clinically inseparable from laryngitis.

TREATMENT.—Treatment of congestion of the larynx is unnecessary, beyond resting the voice, if the congestion is merely temporary. If more persistent, astringents should be applied locally, as in cases of chronic laryngitis.

2. **Larynx, Inflammation of.**—The varieties of laryngitis treated of here will be considered under the following headings: (a) **Acute**—including i. *Catarrhal*; ii. *Phlegmonous*. (b) **Chronic**.

(a) i. **Acute Catarrhal Laryngitis.**—SYNON.: *Laryngitis Catarrhalis*; *Cynanche Laryngea*; Fr. *Laryngite Aiguë Catarrhale*; Ger. *Kehlkopfentzündung*.

DEFINITION.—Acute catarrhal inflammation of the mucous membrane of the larynx.

ETIOLOGY.—Laryngitis is more common among males than females, and among children than adults. The most frequent exciting cause is exposure to cold, but the disease may often be attributed to sudden changes of temperature, a damp or irritating atmosphere, the inhalation of acid vapours or gases, excessive use of the voice, and to the abuse of alcohol, hot drinks, and spices. It may be due to accidental attempts at swallowing boiling water. The catarrhal process may be propagated from the pharynx or nose. It occurs also as a complication of exanthematous fevers—especially of measles. Sedentary occupations, and anything that depresses the vitality of the body, may predispose to laryngitis.

ANATOMICAL CHARACTERS.—Acute laryngitis is characterised by the changes common to catarrhal inflammation of other mucous membranes—hyperæmia, swelling, and increased secretion. Small superficial erosions may occasionally occur, especially at the posterior extremities of the vocal cords, but ulceration does not extend below the epithelial layer.

SYMPTOMS.—A mild attack of laryngitis commences with hoarseness, irritable cough, and a sense of tickling and soreness about the larynx, without any febrile disturbance. Subsequently the voice may be completely lost. In severe cases the patient becomes restless and anxious; experiences a feeling of constriction about the rima; complains of sore-throat in swallowing; and points to the larynx as the seat of pain. The breathing is altered, having more or less the characteristics of laryngeal obstruction, namely, the long-drawn hissing inspiration, prolonged expiration of the same character, but with less of the sibilant sound, and diminution or complete abolition of any pause between successive respiratory efforts. The voice also is altered, becoming husky and uncertain, deeper in tone, and croaking in quality. The patient likewise suffers from cough, of the same character as the voice; from the imperfect closure of the vocal cords it wants sharpness, is husky, and sometimes is accompanied by a hollow, clanging sound, constituting what is commonly called the *croupy cough*. The expectoration varies according as the inflammation involves the larynx alone, or extends to the trachea and bronchi. In the former case it is usually clear, thin, tenacious mucus, which is *hawked* rather than coughed up, mixed with the saliva, which is freely secreted but not swallowed. Sometimes the expectoration contains harder pellets, secreted in the ventricles of the larynx; while, if the trachea and bronchi are involved, the usual thick expectoration of bronchitis is also coughed up. Deglutition is painful and difficult, owing to implica-

tion of the aryteno-epiglottic folds and epiglottis; yet the pharynx, on inspection, shows only a little redness, quite inadequate to account for the dysphagia. Accompanying these symptoms there is a rapid pulse, and slight rise of temperature, though the tongue may be moist, and other indications of inflammatory fever slight.

The laryngoscope reveals congestion of the larynx, which varies much in extent and degree. In slight cases the reddening may be confined to special parts, like the epiglottis and vocal cords, without any appreciable swelling of the affected structures. The cords usually exhibit changes, which account sufficiently for the hoarseness. During attempts at phonation they are often seen to be imperfectly approximated, being separated by an elliptical fissure, the result of paresis of their internal tensor muscles, the thyro-arytenoidei interni. Less frequently the posterior part of the glottis remains open during phonation, forming a triangular space with the apex directed forwards, a condition depending on paresis of the arytenoideus muscle. These pareses of the cords are probably due to inflammatory implication of their neuro-muscular apparatus. Protrusion of the swollen inter-arytenoid fold between the posterior ends of the cords, preventing their due adduction, is an occasional source of hoarseness. At other times no definite loss of movement can be recognised, and here the hoarseness is to be ascribed to alterations in the tension and vibration of the cords produced by the catarrh. In the severe type the whole larynx is congested, and the aryteno-epiglottic folds, epiglottis, and ventricular bands are swollen so as to hide the vocal cords more or less.

In most instances of the mild form, recovery ensues in a few days.

If the case progress untowardly, the restlessness and anxiety increase; suffocative paroxysms occur and recur with increasing frequency; the patient's whole attention appears concentrated on the effort of breathing; he dreads to speak or swallow, and if obliged to say anything, he takes first a laboured inspiration, and then, with a straining effort, brings out what little voice is left. Gradually, as the aëration of the blood becomes more imperfect, drowsiness comes on; the eyes, staring in the previous stage, are half closed by the drooping lids; the face, bathed in perspiration, becomes livid; and death occurs, probably in a paroxysm of suffocative dyspnoea.

Acute laryngitis occurring in young children, sometimes termed *spasmodic laryngitis* or *false croup*, differs somewhat from the description just given. These differences depend on the small size of the child's larynx, and on the tendency to spasm incidental to this age. Without any previous illness beyond, possibly, a slight cough and hoarseness, the child is suddenly seized at night with a barking cough, noisy stridulous inspiration, and more or less urgent dyspnoea. The suffocative seizure seldom lasts more than a few minutes; but it may recur the same night, or on successive nights, the child being generally free from all symptoms, except slight cough and hoarseness, during the daytime. These attacks are mainly due to spasm of the sphincter muscles of the larynx; but in all probability they are also partly the result of drying of the secretions accumulated during sleep, and consequent narrowing of the glottis. Paroxysmal dyspnoea of the same nature, and due to the same cause, may develop as a complication of the exanthemata, especially measles.

DIAGNOSIS.—Acute laryngitis is apt to be confounded with that form of inflammation which is characterised by the formation of a false membrane in the larynx, namely, croup or laryngeal diphtheria. If practicable, a laryngoscopic examination, showing the absence of false membrane, is the most certain means of diagnosing simple from membranous laryngitis; but even where perfect casts of the trachea, bronchi, and primary bronchia are expectorated, the larynx may be absolutely free from false membrane, and simply inflamed. The absence of diphtheritic membrane in the fauces, of acrid excoriating discharge from the nostrils, and of glandular swellings, as well as the mode of accession of the symptoms, would lead us to regard the case as simple laryngitis; while the presence of any of these symptoms, or the prevalence of an epidemic of diphtheria, would lead us to suspect false membrane. Even after due consideration of these points, and of the character of the voice, cough, &c., in certain cases, we are unable to say whether we have to deal with simple or with membranous laryngitis. The acute laryngitis or false croup of children is further distinguished from membranous croup by the sudden accession of the symptoms at night, and by the progress of the case—true croup or diphtheria increasing in intensity, while, from the first sudden onset of the symptoms, false croup diminishes, unless complicated with lobular pneumonia or other severe disease. In many cases bacteriological examination of the secretions from the larynx or fauces will alone enable us to exclude diphtheria. From laryngismus stridulus, acute laryngitis is distinguished by the presence of slight pyrexia and other indications of primary inflammatory affection of the larynx and air-passages; by the absence of indications of any other affection of the nervous system, or of a tendency to convulsions; by the usual occurrence of the attack in the night only; by the slighter affection of the breathing; and by the frequent *croupy cough*, which is the prominent symptom, and which is wanting in laryngismus.

PROGNOSIS.—In mild attacks of laryngitis all the symptoms exist only in a slight degree, and disappear in a few days under simple treatment. Such is the ordinary course of the false croup of children. In adults laryngitis may occur in any degree between the mildest form and that in which, the symptoms being all most intense, it may prove fatal in a day or two, or even in a few hours. The danger depends in great measure on the amount of swelling present. Although mild laryngitis usually terminates favourably in a few days, it is necessary that every case should be sedulously watched, as at any period dangerous symptoms may set in and prove rapidly fatal. The disease is most deadly in the young. Acute may pass on to chronic laryngitis.

TREATMENT.—As soon as the first indications of even slight laryngitis are observed, the patient should be confined to a warm room, at a temperature of 65° F., a kettle being kept boiling to moisten the atmosphere. Talking must be forbidden, in order to ensure as much rest to the larynx as possible. The diet should be chiefly liquid, and no stimulants are required. As a rule, no further treatment is necessary; but, if the cough be troublesome, we may prescribe a saline diaphoretic draught, with small doses of morphine, and 4 or 5 minims of antimonial wine every three or four hours. The cough and sense of dryness, or soreness of the throat, are generally relieved by the

inhalation of simple steam, or by the vapour of compound tincture of benzoin, one drachm being added to a pint of hot water. In every case a regular evacuation of the bowels is to be secured. If swelling of the larynx ensue, the patient must be directed to suck ice constantly. Should the dyspnoea become urgent, tracheotomy must be performed at once. With due care in the performance of the operation and after-treatment of the patient, in cases of simple laryngitis, tracheotomy is almost always successful, but it should not be resorted to unless the case is urgent.

Simple cases of false croup in children are usually relieved by warm poultices to the throat. An emetic may be administered in the comparatively rare instances in which dyspnoea depends on insufficient removal of secretions from the larynx.

ii. **Phlegmonous Laryngitis.**—**SYNON.** : *Œdema Glottidis*; Œdema of the Larynx; Œdematous Laryngitis; Fr. *Laryngite Œdémateuse*; Ger. *Kehlkopfödem*.

DEFINITION.—Inflammatory exudation into the submucous tissue of the larynx of serous, sero-purulent, or sero-fibrinous fluid.

ÆTIOLOGY.—Phlegmonous laryngitis occasionally arises as a primary disease, and is then to be regarded as a symptom of septic infection; thus in rare instances it seems to have been the earliest manifestation of erysipelas.

Mechanical injuries, and the direct action of boiling water, corrosive poisons, and foreign bodies are less uncommon causes. Phlegmonous laryngitis may also develop in the course of specific fevers, such as measles, scarlatina, small-pox, typhoid fever, diphtheria, pyæmia, and in renal disease.

In most cases, however, the laryngeal affection is the result of extension of disease from neighbouring parts, pharyngitis, whether dependent on erysipelas or other causes, being the most common antecedent.

Inflammatory affections of the cervical connective tissue, and perichondritis, or deep ulceration of the larynx, associated with carcinoma, syphilis, or tuberculosis, are also occasional causes of phlegmonous laryngitis.

ANATOMICAL CHARACTERS.—The mucous surface presents a dull red or livid colour, and is greatly swollen, owing to infiltration of the submucosa. A certain amount of pus is generally diffused through the exudation, but it is rare for a circumscribed abscess to form. The process usually attacks first the aryæno-epiglottic folds and the epiglottis, and it may extend thence to the ventricles and other parts of the larynx, the vocal cords themselves being seldom affected. The submucous tissue below the vocal cords, lining the cricoid cartilage, may very occasionally be the sole seat of the disease.

SYMPTOMS.—The symptoms indicative of severe laryngitis exist in an increased degree in this affection, the swelling of the epiglottis interfering especially with deglutition, and that of the aryæno-epiglottic and ventricular bands causing very dangerous dyspnoea. By examination with the finger and, far better, by the laryngoscope, we observe further the physical condition caused by the swelling. The epiglottis is congested, misshapen, rounded like a chestnut, or having its two sides swollen so as to resemble two mucous bladders pressed together in the middle line; and, unless this hides the rest of the larynx, the aryæno-epiglottic folds will be recognised as two long rounded

swellings, passing from before back, and nearly meeting in the centre, the ventricular bands being visible only when there is little swelling above. The prognosis in such cases is extremely grave.

TREATMENT.—The patient should suck small pieces of ice, leeches might be applied over the larynx, and an attempt may be made to reduce the swelling by scarification with a curved laryngeal bistoury, but in most instances tracheotomy is required.

(b) **Chronic Laryngitis.** — **DEFINITION.** Chronic inflammation of the mucous membrane of the larynx.

ÆTIOLOGY.—Chronic laryngitis is often, but not invariably, a sequel of acute laryngitis, and is due to the same causes. Chronic affections of the nose and pharynx frequently co-exist, and seem in some instances to be the starting-point of the malady.

ANATOMICAL CHARACTERS.—The mucous membrane of the larynx is hyperæmic, and may be slightly swollen, the vessels being often dilated in patches. The morbid process may extend in rare cases to the submucosa, producing diffuse connective-tissue thickening, or small warty growths, especially on the vocal cords—‘*chorditis tuberosa*’ of Türk. Marked enlargement of the veins on the epiglottis is occasionally observed, and is probably the result of the chronic catarrh.

SYMPTOMS.—The symptoms of chronic laryngitis are hoarseness or aphonia after slight exertion of the voice; a hacking cough, with but little of the brassy, laryngeal character; either no expectoration, or only a little tenacious mucus; and a sense of dryness and tickling, with slight pain, in the throat.

The laryngoscope shows a varying amount of congestion, either general or local, but the hyperæmia is seldom so pronounced as in acute laryngitis. The cords are usually affected, presenting a pinkish, slightly thickened appearance.

Mucous secretion is often seen sticking to the inter-arytenoid fold or to the anterior angle of the glottis. In a special variety of the disease, known as ‘*laryngitis sicca*,’ the secretion is apt to dry and form adherent scales or crusts, in consequence of some modification of its composition. Epithelial erosions are not uncommon, but ulceration never occurs. Pareses of the vocal cords often ensue, giving rise to changes in the glottis, as described under Acute Laryngitis.

In the rare instances in which thickening invades the subglottic region, stenosis commonly ensues, and obstructive dyspnoea becomes a prominent symptom.

DIAGNOSIS.—In cases of chronic disease of the larynx a laryngoscopic examination is indispensable. Without the laryngoscopic diagnosis and treatment can be only guess-work; with it, the appearances described above are clearly recognised. Where chronic laryngitis is persistent, the possibility of tuberculosis must be kept in mind, and repeated examination of the lungs and sputum should be made. In elderly or middle-aged patients a localised swelling and congestion of one of the vocal cords, especially if associated with impaired mobility, should arouse the suspicion of carcinoma.

PROGNOSIS.—Chronic catarrhal laryngitis may subside spontaneously, but is usually persistent unless properly treated.

TREATMENT.—Any imprudence in diet or hygiene must be corrected, pungent condiments or

spices, very hot drinks, alcohol in excess, smoky or dusty rooms, being avoided, and the voice rested as much as possible. About every other day the larynx should be brushed out with an astringent lotion. In mild cases a solution of chloride of zinc, 30 grains to the ounce, will suffice; but if this fail, a more powerful astringent, nitrate of silver 10 to 40 grains to the ounce, should be employed. Such applications should not be continued for more than two or three weeks at a time.

Inhalations of chloride of ammonium, pine-oil, and creosote are recommended by some physicians. In some obstinate cases the waters of Ems, Reichenhall, Marienbad, Aix-les-Bains, and other spas are believed to yield good results. Attention must be paid to the general health, and to the state of the digestive organs.

3. **Larynx, Leprosy of.**—**ÆTIOLOGY.**—The causes are identical with those of leprosy in other situations. Leprosy of the larynx may be associated with a similar affection of the buccal mucous membrane, the tongue, and the palate.

ANATOMICAL CHARACTERS.—The lesions at first consist of a nodular thickening of the epiglottis and aryæno-epiglottic folds, which may be succeeded by very chronic ulceration and cicatrization. Ultimately the disease may invade other parts, and cause stenosis of the larynx.

SYMPTOMS, COMPLICATIONS, AND DIAGNOSIS. The symptoms of laryngeal leprosy are those of chronic inflammation; and, if the nodules ulcerate, the ulceration is very slow, and not of the destructive character of syphilis. As leprosy of the larynx occurs only when the disease of the skin has been long established, its diagnosis is clear. It usually exists for a long time, even years, before it ulcerates; and even after this it is very slow in its progress, and not fatal.

TREATMENT.—As in other forms of leprosy, treatment is almost useless. Tracheotomy must be performed if stenosis occur. *See* LEPROSY.

4. **Larynx, Lupus of.** — **ÆTIOLOGY.** — Although in all probability a form of tuberculosis, this condition has characteristic clinical features. The larynx may be primarily affected.

ANATOMICAL CHARACTERS.—Small nodules appear on the epiglottis, and subsequently on the aryæno-epiglottic folds and other parts of the larynx, associated sooner or later with thickening and occasionally with ulceration and scarring of the mucous membrane.

SYMPTOMS, COMPLICATIONS, AND DIAGNOSIS. Lupus of the larynx may give no definite symptoms, even when pronounced lesions exist, and doubtless on this account this affection of the larynx is often overlooked. In other cases the symptoms are those of chronic inflammation. Lupus is distinguished from syphilis and ordinary tuberculosis by its prevailing nodular character, and by the rarity of ulceration. It lasts for many years.

TREATMENT.—Lupus of the larynx must be treated constitutionally in the same way as other forms of tuberculosis, and locally by caustics or the galvanic cautery. Lactic acid, applied as in other cases of laryngeal tuberculosis, is of service.

5. **Larynx, Nervous Affections of.**—(1) **SENSORY DISORDERS OF THE LARYNX.**—*Hyperæsthesia* of the laryngeal mucous membrane is a usual accompaniment of inflammation, acute and chronic, and is a not infrequent symptom of hysteria, hypochondriasis, and neurasthenia. *Anæsthesia* occurs

in cases where the superior laryngeal nerve or its centre is affected, and especially as a sequel of diphtheria. It is also an occasional symptom of bulbar paralysis and hysteria, and is said to accompany epileptic fits. *Hemianæsthesia* may result from tumours of the base of the skull. *Paresthesia*, in which the larynx is the seat of various perverted and unaccountable sensations, is not an uncommon manifestation in anæmic, hysterical, and hypochondriacal patients.

(2) MOTOR DISORDERS OF THE LARYNX.—
(a) *Spasm* is considered in a separate section. See 6. Larynx, Spasm of.

(b) *Paralysis* of the larynx is of great variety. It will be best discussed, first, under the head of the *nerves* affected; and, secondly, with respect to the individual *muscles* paralysed.

i. *Paralysis of the Nerves*.—*Paralysis of the Superior Laryngeal Nerve*.—In this rare condition, depending either on diphtheria or bulbar paralysis, the interior of the larynx is quite insensitive to the introduction of a probe; the epiglottis lies motionless against the base of the tongue, owing to paralysis of its depressors—the thyro-epiglottic and aryteno-epiglottic muscles; and the voice is said to be rough and low-pitched, from palsy of the crico-thyroid muscles. The glottis is represented by a wavy line, in consequence of paralysis of the external tensors. The immobility of the epiglottis and the anæsthesia are a source of great danger, as food is likely to enter the larynx and lead to pulmonary complications. Food must be administered by the œsophageal tube in such cases. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

Paralysis of the Recurrent Laryngeal Nerve.—Complete paralysis is generally due to pressure of an aneurysm of the aorta or other intrathoracic tumour, or of a goitre, on the nerve after its origin from the vagus, but the same effect may follow degenerative or destructive lesions of the motor fibres in any part of their course from the medulla oblongata down to their peripheral termination. Bilateral paralysis, in the very rare instances in which it has been observed, was the result of carcinoma of the œsophagus, double aneurysm of the aorta and innominate artery, tumours of the base of the brain, extensive pericardial effusion, and, lastly, pressure on the trunk of one pneumogastric nerve, for example, by enlarged bronchial glands. The symptoms of bilateral paralysis are aphonia, inability to cough, and slight stridor. Slight dyspnoea may be induced by attempts to speak. The vocal cords are seen lying motionless and relaxed, in a position midway between abduction and adduction. In unilateral paralysis, which is the usual form and is more common on the left side, one cord alone occupies the position just described, but, as the healthy cord crosses over the middle line during phonation to meet its palsied fellow, there is no aphonia, though the voice is generally weak and uncertain.

Hitherto complete paralysis alone has been considered; but paralysis depending on structural disease of the fibres or nuclei of the recurrent laryngeal nerves is far more often incomplete. In these circumstances the fibres supplying the abductor muscles are the first to suffer. In course of time the adductors may become affected also, but paralysis of these muscles is seldom complete. This fact is attributed to a greater vulnerability of the abductor nerve-fibres than of those supplying

their antagonists, the adductors. Semon believes that the abductor muscles themselves are less resistant than the adductors.

ii. *Paralysis of Individual Muscles*.—*Bilateral Abductor Paralysis*.—The cause may be either central—as in tabes dorsalis, bulbar paralysis, syphilitic or other intracranial disease; or peripheral—lesions of both recurrent or pneumogastric nerves, pressure on the trunk of one vagus, or degenerative affections of the abductor muscles, as described under the preceding heading. The symptoms are severe inspiratory dyspnoea and stridor, and weakness or slight hoarseness of the voice. With the laryngoscope the cords are found lying close together, being separated by a mere chink. On phonation they approximate as usual, but during deep inspiration no divergence takes place, and a slight movement of adduction is generally observed. This action is attributed either to the effects of suction, or to a perverted innervation of the unopposed adductors. Bilateral paralysis may cause fatal suffocative attacks, and, if persistent, necessitates the performance of tracheotomy.

Abductor paralysis may be simulated by mechanical fixation from ankylosis or arthritis of the crico-arytenoid joints, or by cicatricial or inflammatory changes in their neighbourhood. Diagnosis is often difficult in such cases, but in the latter condition there is usually some distortion of parts, such as contraction, irregularity, or swelling about the arytenoid cartilages, which is not the case in paralysis.

Unilateral Abductor Paralysis.—This condition is generally attributable to peripheral causes, and especially to compression of the left recurrent laryngeal or vagus nerve within the thorax by an aneurysm or other tumour. Palsy of the right abductor is an occasional result of chronic disease of the apex of the right lung.

The affected vocal cord occupies the median position; but if the adductor becomes involved, the cord recedes from the middle line and takes up a situation between that of abduction and adduction. The symptoms of unilateral paralysis are slight hoarseness and dysphonia, but dyspnoea and stridor do not occur except on exertion.

Paralysis of the Adductors.—Paralysis of these muscles is nearly always bilateral, and is probably the result of a central nervous affection of a functional nature, occurring in hysterical, neurotic, or anæmic subjects. It is doubtful whether gross nervous disease ever leads to isolated paralysis of the adductors. The patient, generally a female, becomes suddenly aphonic, but the reflex acts of sneezing and cough are still attended by the usual sound. Attempts to speak occasion a slight degree of dyspnoea, due to the escape of breath through the widely open glottis. The laryngoscope shows that the cords are abducted normally, but do not approach one another during efforts to phonate.

Paralysis of the Internal Tensors.—The internal tensors or thyro-arytenoidei interni may be paralysed, on one or both sides, as a consequence of laryngitis or of hysteria. According as the affection is bilateral or unilateral, there is aphonia or mere hoarseness.

During phonation the vocal cords are separated by an oval or elliptical space, and appear to be very thin and narrow, owing in all probability to deficient tension.

Paralysis of the External Tensors, the crico-

thyroid muscles, is unknown, apart from the results of paralysis of the superior laryngeal nerve.

Paralysis of the Arytenoides.—This is also commonly a result of hysteria or of laryngeal catarrh, and gives rise to hoarseness or aphonia. During phonation the cords meet in front, but are separated posteriorly by a triangular gap.

TREATMENT.—If the paralysed condition depend on serious disease outside the larynx, local treatment is of little avail. When syphilis or aneurysm is suspected, iodide of potassium should be prescribed freely. In cases of adductor paralysis, anæmia and the hysterical state must be suitably treated, and intra- or extra-laryngeal faradisation may be tried, but sudden recovery often occurs without any treatment. See PNEUMOGASTRIC NERVE, Diseases of.

6. Larynx, Spasm of.—In the description of other diseases of the larynx, this condition has been referred to as a frequent complication. Spasm causes the urgent symptoms when the larynx is irritated by the lodgment of a foreign body or the like. It may result from pressure on, or disease of, the pneumogastric or recurrent laryngeal nerve; from hysteria; or as a manifestation of a more general affection of the nervous system, in which case it constitutes the following special malady.

Laryngismus Stridulus.—SYNON.: Child-Crowing; Spasmodic Croup; False Croup: Fr. *Spasme de la Glotte*; *Pseudo-croup Nerveux*; Ger. *Kehlkopfkrampf*.

DEFINITION.—*Laryngismus stridulus* is characterised by short or more prolonged accessions of suffocation; depending on tonic spasm of the adductor muscles of the larynx, and usually of the diaphragm and other respiratory muscles, causing closure of the glottis and a sudden arrest of inspiration; and ending in a shrill crowing sound, as the inspiratory act is resumed and concluded. It is purely a nervous disease; is unaccompanied by any inflammatory affection of the larynx or air-passages; and is often associated with other convulsive affections.

ÆTIOLOGY.—Anything causing excessive reflex irritability, rickets, chronic hydrocephalus and other organic affections of the brain or medulla oblongata, predispose to this convulsive affection. It is sometimes associated with the irritation of teething. A sudden fright, irritation of the larynx by the accidental entrance of food, or some such slight agitation as that caused by a child being tossed in the air, may excite the attack.

SYMPTOMS.—With or without premonitory indications of a tendency to convulsive affection, such as drawing-in of the thumbs and great toes, or clenching of the hands; often during sleep, and with no evident exciting cause, or at any time in the day; a child is suddenly attacked with difficult breathing, inspiration being accompanied by the crowing sound characteristic of laryngeal spasm. This may continue for some time, and then gradually subside. The spasm may be short, or it may be longer and more intense, inspiration being proportionately difficult. It may be complete, and the act of inspiration cease entirely, until, just as death seems imminent, the spasm relaxes, and with a crowing inspiration breathing is re-established. In the worst cases, and sometimes in the first attack, death does actually occur.

DIAGNOSIS.—The diagnosis of this disease from laryngitis is considered under Acute Laryngitis.

The symptoms caused by a foreign body lodged in the larynx closely simulate laryngismus stridulus. The nature of the case is decided by its history; and, unless the age of the patient precludes it, by a laryngoscopic examination.

PROGNOSIS.—The milder forms of laryngismus stridulus yield to suitable treatment, and disappear as improvement takes place in the condition inducing the attack. Severer forms, if not fatal in a first, may be so in a subsequent attack. When the spasm depends on some incurable organic change in the nervous system, the case is of course hopeless from the first.

TREATMENT.—Attention to the diet and general management of the child, regular bathing, and the administration of remedies suitable to correct faults of digestion, are necessary (see RICKETS). The persevering use of bromide of potassium has been found beneficial; and chloral hydrate is of undoubted value. For the immediate treatment of the spasmodic attack, prompt immersion in a warm bath, the administration of an emetic, or the use of an anæsthetic vapour or amyl nitrite may be resorted to; and should breathing not be re-established as the spasm ceases, dashing cold water on the face and chest, friction, application of strong ammonia or vinegar to the nostrils, and especially artificial respiration, must be adopted, with the object of restoring the respiratory function. Tracheotomy may be requisite, but the practitioner is, unfortunately, seldom present when the indications for the operation arise.

7. Larynx, Syphilis of.—Syphilis affects the larynx differently according to the stage of the disease at which the organ is attacked. Erythematous maculæ, raised mucous patches or condylomata, and superficial ulceration, like that seen in the fauces and pharynx, are described as secondary lesions, but are seldom actually observed, as opportunities for making a laryngoscopic examination are not often afforded. Mucous patches, represented by small greyish flat elevations, and congestion, are the commonest manifestations at this period of the disease. These patches generally disappear without leaving any trace of their existence.

The larynx is more seriously affected in advanced stages of syphilis. The lesions, consisting of diffuse infiltration, nodular gummatous deposits, or ulceration, are especially prone to attack the epiglottis and vocal cords, and in a less degree the ventricular bands and posterior wall.

The special proclivity of the epiglottis to syphilitic disease has been attributed to direct infection from the pharynx, it being an undoubted fact that syphilis generally involves the pharynx together with the larynx. Syphilitic ulceration may be superficial or deep. Deep ulcers, which are often of gummatous origin, are sharply cut, and extend rapidly. As a result, extensive destruction of the epiglottis, suppurative perichondritis, and necrosis of the various cartilages, often ensue. External fistulæ are occasionally thus produced. Perichondritis at times develops without previous ulceration. Cicatrices are almost an invariable sequel of deep ulceration, and are a great source of danger, owing to the contraction and stenosis which they occasion. Curious puckering of different parts is commonly produced, at times associated with small polypoid excrescences of the mucous membrane. These last, unlike gummata, show no tendency to ulcerate, and may gradually disappear. Adhesion and webbing

of the cords, adhesion of the epiglottis to the walls of the pharynx, fixation of the vocal cords in various positions, sometimes simulating bilateral abductor palsy, and depending then on fibrous changes around the crico-arytenoid articulations—these are some of the many effects of syphilitic cicatrisation.

In *hereditary* syphilis, lesions of the larynx generally occur within the first few months after birth, but occasionally the larynx is not attacked before the age of puberty is reached. The early manifestations of the disease in hereditary cases are represented by congestion and mucous patches; at a later period they are not to be distinguished from those which occur in the advanced stage of acquired syphilis. Deep ulceration in any case may be associated with extensive oedematous swelling, and hæmorrhage from perforation of large vessels is an occasional occurrence.

SYMPTOMS.—Secondary syphilitic affections cause hoarseness and sometimes loss of voice, but there is usually neither pain, cough, fever, nor dyspnoea. The more serious tertiary affections may give rise to all the symptoms caused by tubercular ulceration, but pain and dysphagia are rarely experienced except when the epiglottis is extensively ulcerated, and even then these symptoms are less severe than in tubercular cases, and the constitutional symptoms are those of syphilis, as distinguished from consumption.

DIAGNOSIS.—Secondary syphilitic affections of the larynx are easily recognised with the laryngoscope, and there are usually other indications of the constitutional taint. The diagnosis of the disease, in its later stages, must be based on the appearance of the lesions above mentioned, combined with manifestations of syphilis in other parts of the body, especially of the skin and pharynx. In the absence of constitutional symptoms, the diagnosis may present great difficulties, especially as regards tuberculous. This question will be discussed in the next section.

It is sometimes not altogether easy to distinguish syphilitic from carcinomatous ulceration, but in the latter disease the appearance of a growth is more pronounced, ulceration is less rapid, and pain is more often present. Moreover, the vocal cords and ventricular bands are the favourite seats of malignant growths, the epiglottis and other parts being seldom primarily affected.

The good results following the use of iodide of potassium will often assist in arriving at a correct diagnosis, but this test is open to a fallacy, as carcinoma and syphilis may co-exist.

PROGNOSIS.—The prognosis in cases of laryngeal syphilis must vary according to its form. In early cases, and where ulceration is superficial, the prognosis is very favourable, but in advanced stages the prospects of the patient are more uncertain; for, although the progress of ulceration can almost invariably be arrested by treatment, subsequent cicatrisation may cause dangerous stenosis. A knowledge of the complications incidental to deep ulceration should induce us always to give a guarded opinion.

TREATMENT.—Local treatment is not necessary for the secondary syphilitic affections of the larynx, and we must trust to mercurial inunction and other constitutional remedies. Syphilitic ulceration of the larynx requires persevering treatment with large doses of iodide of potassium; and where it does not yield to this remedy, appropriately combined with quinine, cod-liver oil, &c., mercury must be

employed, the best method being by mercurial inunction or the hypodermic injection of the perchloride. A combination of mercury and iodide of potassium is often useful. The local application of a solution consisting of iodine 10 grs., iodide of potassium 100 grs., glycerine 1 oz., is sometimes of service in the case of syphilitic ulcers, in addition to the above treatment. When the dyspnoea is dangerous, tracheotomy must not be delayed. It will usually prove successful, although the destruction of tissue and cicatrisation may be such as to necessitate the permanent wearing of the tube. Operations for the division of cicatricial bands and adhesions seldom give satisfactory results, owing to the tendency of the parts to unite again. Systematic dilatation, by means of Schröter's hollow vulcanite bougies, may be tried in such cases.

8. Larynx, Tuberculosis of. — **SYNON.:** Laryngeal Phthisis; *Fr. Phthisie Laryngée*; *Ger. Kehlkopf-tuberculose.*

ÆTIOLOGY.—Laryngeal tuberculosis is almost invariably secondary to pulmonary phthisis, but in a few instances a necropsy has demonstrated the existence of tubercular disease in the larynx without any affection of the lung. In the former case the larynx is either directly inoculated with infective sputum derived from the lung, which is the usual method, or the tubercular virus is conveyed to the larynx by the blood. Where the larynx is primarily affected, we may suppose that the infective agent, the tubercle-bacillus, is inhaled, and effects a lodgment here without reaching the lung.

ANATOMICAL CHARACTERS.—In the larynx, as in other mucous membranes, the tubercular process may be considered under two heads—infiltration or deposit, and ulceration; but inasmuch as the necrotic element predominates over the fibrous or indurative, the tendency is ultimately towards ulceration. The degree of infiltration varies greatly, now giving rise to massive swelling of parts like the epiglottis and aryteno-epiglottic folds, where the submucous tissue is loosely arranged, at other times merely extending to the subepithelial layer. In the latter case, owing to the early development of ulceration, a stage of infiltration cannot generally be seen to precede loss of substance, though from analogy there is reason to believe that this is invariably the case. Definite tubercular tumours are occasionally met with.

The parts most prone to tubercular disease may be thus enumerated, in the order of frequency: the vocal cords and inter-arytenoid fold in about equal proportions, the aryteno-epiglottic folds, epiglottis, and, lastly, the ventricular bands. Extension of the infiltration and ulceration to the deeper parts may lead to suppurative or adhesive perichondritis, especially of the arytenoid cartilages. Suppurative perichondritis causes necrosis, and at times extrusion of the cartilage. Oedema is a frequent complication of extensive tubercular lesions. It is unnecessary in the present article to describe the manifold changes that may be produced in different cases.

SYMPTOMS.—The symptoms of this affection are those of an aggravated laryngitis: hoarseness or aphonia, irritable cough, difficulty or pain in swallowing when the epiglottis or aryteno-epiglottic folds are involved, and inspiratory dyspnoea and stridor if stenosis be present. Pain shooting to the ear is occasionally complained of, and is probably referred from the sensory nerve of the larynx, the

superior laryngeal, to the auricular branch of the vagus. The constitutional and local symptoms of pulmonary phthisis, which are present in varying degrees, add much to the patient's distress. In early cases the laryngoscope reveals small shallow ulcers or pale granulations on the vocal cords, particularly their posterior ends, fleshy swelling of one or both cords, and prominence of the inter-arytænoïd fold, often with an irregular papillary margin. Swelling of one or both sides of the epiglottis, aryteno-epiglottic folds, and ventricular bands, and deep ulceration may be observed later on. The ulcers present either a greyish-yellow or pinkish granular base, and minute yellowish nodules are occasionally developed in their neighbourhood. In certain instances the vocal cords are split longitudinally into terrace-like, reddish, ulcerated ridges, so that each cord seems to be composed of several individual segments. Small tubercular tumours, of a greyish-pink colour, may occasionally be seen springing from the cords, ventricles, ventricular bands, or other parts. When the epiglottis is much swollen or hangs backwards, it may be impossible to obtain a view of the rest of the larynx. A similar difficulty is sometimes experienced when the larynx is coated with a copious viscid secretion. The colour of the larynx varies greatly: generally speaking, a greyish-pink or pale yellowish tint prevails, contrasting often with localised patches of congestion in the vicinity of ulcers or infiltrations. But this colour, which is very characteristic, is not invariably met with, and general congestion may exist. Impaired mobility of the cords is very common; it mostly depends on mechanical fixation of the arytenoid cartilages from surrounding infiltration. At times the vocal cords occupy the median position, simulating bilateral abductor paralysis, and giving rise to severe stenosis. Neuropathic paralysis also occurs. Thus one cord may be motionless in the cadaveric position, midway between that of adduction and abduction. On the right side this condition may occasionally be the result of implication of the recurrent laryngeal nerve in pleuritic thickening of the apex of the right lung; whereas the left recurrent may be compressed by enlarged bronchial glands as it winds round the arch of the aorta. Imperfect closure of the glottis depending on bilateral paresis of the adductors, as in cases of functional or hysterical paralysis, is a more common appearance. Lastly, in advanced cases of phthisis, aphonia may be unassociated with any defect of movement, the feeble action of the respiratory muscles being insufficient to cause the requisite vibration of the cords. In other instances paralysis is probably of myopathic origin.

DIAGNOSIS.—Chronic laryngitis is sometimes one of the earliest symptoms of phthisis, and therefore should always direct attention to the state of the lungs. The diagnosis of tubercular disease rests on the detection of localised infiltration or ulceration of certain parts of the larynx, in association with physical signs of pulmonary tuberculosis. Syphilis is the disease with which it is most likely to be confounded, and the difficulty is increased by the fact that the two affections may co-exist; some authors even assert that syphilitic ulceration predisposes to tuberculosis. Pronounced pallor of the larynx, swelling of the inter-arytænoïd folds, with papillary excrescences, fleshy swelling of one cord, chronic ulcers of the cords, and a pale swollen condition of the aryteno-epiglottic folds and epiglottis,

are very characteristic of tuberculosis. On the other hand, marked congestion, rapidly extending ulceration of the cords or epiglottis, and cicatricial contraction, would be in favour of syphilis. Nevertheless, in many cases the laryngoscopic appearances are quite inconclusive; and, should examination of the lungs yield a negative or equivocal result, the detection of the tubercle-bacillus in the sputum will alone establish the diagnosis. Tuberculosis can seldom be mistaken for a malignant growth, but the diagnosis will be considered under the latter disease.

PROGNOSIS.—Laryngeal tuberculosis is rarely cured, though spontaneous cicatrization undoubtedly occurs in exceptional cases. The progress of the disease can often be temporarily checked, and at times is arrested by suitable treatment. As a rule, however, the prognosis is most unfavourable. Infiltration of the epiglottis and aryteno-epiglottic folds has a most ominous significance, owing to the interference with deglutition thereby entailed.

TREATMENT.—The constitutional treatment is discussed in the article on PHTHISIS; consequently the local treatment alone will be considered here. Lactic acid is the most efficacious remedy at present known. The writer has seen several cases where permanent cicatrization of tubercular ulcers has followed the systematic application of this remedy. The larynx must first be well brushed out with a 20-per-cent. solution of cocaine; and then, after an interval of three to five minutes, a 50-per-cent. solution of the acid should be thoroughly applied on a small piece of cotton wool, fixed to the screw of a laryngeal holder. The strength of the solution should be increased till saturation is reached, and the treatment should be continued for ten days to a fortnight, the applications being made every other day, or in some cases daily. It is extremely important to paint the larynx thoroughly with cocaine two or three times before using the acid, otherwise the application is very painful. If, notwithstanding this precaution, pain be experienced, a weak spray of cocaine to the larynx may be trusted to give relief. The selection of fit cases is a matter of great moment. Real benefit is only to be expected where localised ulceration is unassociated with much submucous thickening. Where deep infiltration exists, radical treatment is out of the question.

A solution of menthol in olive oil, 20 per cent., painted on the larynx, gives much relief to pain and dysphagia, and is said to promote healing of ulcers; but in the last respect it cannot be compared with lactic acid. Insufflation of a powder consisting of boric acid and iodoform equal parts, with cocaine or morphine $\frac{1}{6}$ to $\frac{1}{3}$ of a grain, has also been much praised, but its effects are only palliative.

Painful deglutition can generally be relieved by spraying the throat, a few minutes before food is taken, with a 2- to 5-per-cent. solution of cocaine. A lozenge, containing $\frac{1}{10}$ to $\frac{1}{6}$ of a grain of cocaine, also gives relief in less severe cases. Insufflation of morphine, $\frac{1}{6}$ of a grain, with a little starch-powder or sugar of milk, produces less complete analgesia, but the effect lasts rather longer. Much relief may also be obtained from insufflation of 'new orthoform.'

In cases where liquids enter the larynx during drinking, as sometimes happens when the epiglottis is destroyed, Wolfenden recommends that the patient should drink through a tube, lying flat on

his face. Thickened drinks can often be taken better than ordinary fluids in such cases. Laryngeal cough may be mitigated by a cocaine-spray, the use of menthol or oil of peppermint in an oro-nasal respirator, insufflations of morphine, or by sucking ice.

Tracheotomy is only to be performed when marked stenosis is produced; but this operation is seldom required, and its effects on the pulmonary disease are very unfavourable, on account of the difficulty of expectoration which is experienced when the action of the glottis is abolished by the introduction of a tracheal cannula.

9. Larynx, Tumours of.—(a) **Benign.** SYNON.: Polypi, or Growths of the Larynx; Fr. *Tumeurs, Kystes, et Polypes du Larynx*; Ger. *Kehlkopfpolypen*.

ETIOLOGY.—The invention and use of the laryngoscope, leading to accurate diagnosis, has established the fact that tumours of the larynx are of much more frequent occurrence than was formerly supposed. They are most common in adult males, but they occur in either sex, and at any age from infancy upwards. Inflammatory attacks, syphilis, and anything leading to habitual congestion of the larynx, are said to favour their development.

ANATOMICAL CHARACTERS.—Growths of various kinds occur in the larynx, papillomata being much the commonest. These present the appearance of a sessile warty or mulberry-like growth, attached generally to the anterior part of the vocal cords, though they may spring from almost any part of the larynx. Papillomata vary in size from a millet-seed to a walnut, and may be single or multiple. They sometimes show a tendency to recur when removed. Fibromata, the next commonest form of innocent tumour, may be sessile or pedunculated; their surface is usually smooth or slightly lobulated, but in size and localisation they closely resemble papillomata.

All other benign growths are very rare, and it must suffice to enumerate myxoma, lipoma, enchondroma, adenoma, angioma, and cystic tumour.

SYMPTOMS.—Benign tumours may cause little inconvenience; but, owing to the fact that the commoner varieties generally affect the vocal cords, some modification of the voice is almost a constant symptom. A dry spasmodic or croupy cough is sometimes excited. The breathing is but little affected until the growth attains some size, when dyspnoea will set in, at first only on exertion, occasionally spasmodic; as the growth increases it becomes constant, and at last it may prove fatal if the disease be unrelieved. Tumours attached below the vocal cords are rare; when they exist, and are large enough to interfere with the breathing, expiration is as noisy and difficult as inspiration. These growths are usually painless. They may be so situated as to interfere with swallowing, but this is not usual. In addition to these symptoms, the growth can be seen with the aid of the laryngoscope, and felt with the laryngeal probe or sound, and sometimes with the finger. Occasionally portions of a papilloma are expelled by coughing.

DIAGNOSIS.—A certain diagnosis can only be arrived at by examination of the larynx. The laryngoscope will often, but not invariably, enable us to distinguish a papilloma from a fibroma. The diagnosis of a benign from a malignant growth is sometimes no easy matter, though the absence of ulceration, congestion, or infiltration, which cha-

racterises an innocent tumour, is rare in malignant disease. The fact that simple growths hardly ever originate from the inter-arytenoid or aryteno-epiglottic folds will help to distinguish these from the polypoid excrescences that often fringe the margin of tubercular or syphilitic ulcers in these regions.

PROGNOSIS.—The importance of these growths varies with their situation and rate of increase. A few months have sufficed for the growth of tumours from their origin to their attaining a size sufficient to threaten suffocation; in other cases they may exist for years without giving rise to any symptom beyond dysphonia or aphonia. As soon as a growth causes dyspnoea it has become dangerous. The disease must practically be regarded as incurable, except by operation or other local treatment.

TREATMENT.—A small stationary fibroma or other tumour, giving rise to but little inconvenience, requires no treatment. All other tumours must be removed by operation. Removal may be effected by instruments introduced into the larynx from above with the aid of the laryngoscope; or, where the disease is very extensive, an artificial opening into the larynx (thyrotomy or division of both thyroid and cricoid) may be necessary, the growth or growths being removed through this opening. This is the only feasible method of operation in the case of children. If the growth is removed *per vias naturales*, it may be crushed through at its base by a properly constructed *éraseur*; it may be seized and torn off by forceps; or it may be cut off by knives or scissors. The particular operation, and the instruments to be used, must be determined by the circumstances of the case. The use of a cocaine-spray renders such manipulations much easier. The statement which has been advanced, that the irritation of endo-laryngeal operations may induce the transformation of innocent into malignant growths, has been conclusively disproved.

Pachydermia Laryngis.—In this affection local thickening of the epithelium and subepithelial tissue gives rise to the appearance of small growths. Pachydermia attacks the vocal cords as a rule, but the inter-arytenoid fold at times is involved. In the former position the disease appears as a flattish papillomatous growth on the processus vocalis. When the change is symmetrical, as is often the case, the outgrowth on one side commonly fits into a corresponding shallow depression on the surface of the opposite growth. This appearance is believed to be the result of pressure.

Hoarseness and slight alterations of the voice may soon attract the patient's notice, or there may be no symptoms for some time. The diagnosis is generally easy when the vocal cords are affected. Carcinoma is the only disease that is likely to give trouble. But in carcinoma the growth is unilateral, except in advanced stages, and partial fixation of the cord occurs early, whereas in pachydermia the disease tends to become symmetrical, and the movements of the cords are seldom impaired. The progress of the two affections is very different. Carcinoma is progressive; pachydermia is more or less stationary and may completely disappear, though it may recur subsequently.

Pachydermia of the inter-arytenoid fold may closely simulate tuberculosis, but examination of the lungs and sputum will help to establish the diagnosis.

As regards treatment, the continued administra-

tion of iodide of potassium is believed by Semon and others to give good results. In obstinate cases the cautious use of the galvanic cautery has occasionally been successful.

(b) **Malignant.**—Malignant tumours of the larynx may be primary or secondary. Primary growths are far less rare than was formerly thought, carcinoma being much more often met with than sarcoma. Epithelioma is the commonest form of carcinoma, the medullary and scirrhous varieties occurring less frequently. Carcinoma develops as a 'cauliflower' or nodular growth from the vocal cords or ventricular bands, though occasionally other parts are first affected. The tumour, at first localised, tends gradually to involve the whole of one side or more of the larynx. Owing to the infiltrating nature of the disease, the mobility of the corresponding vocal cord is often interfered with. Ulceration is a common occurrence, and may be followed by oedema, hæmorrhage, perichondritis, and necrosis of the cartilages. Carcinoma sometimes shows little tendency to infect the glands and other parts of the body, and sarcoma is even less liable to spread.

SYMPTOMS.—Hoarseness or aphonia is usually the first symptom, but dysphagia and dyspnoea may arise later on. Pain is more pronounced in this than in any other affection of the larynx, and may shoot to the ear. Ulceration is sometimes attended with great fetor of the breath.

DIAGNOSIS.—The laryngoscopic appearances are seldom characteristic, often closely resembling those presented by innocent growths, especially papillomata. The diagnostic importance of fixation of the corresponding vocal cord has been pointed out by Semon. Microscopical evidence of carcinoma or sarcoma may sometimes be detected in small portions of the tumour, removed by the laryngeal forceps, and is then of course conclusive.

The diagnosis of syphilis from malignant disease has been already discussed. Tubercular lesions seldom present the appearance of a localised growth, and careful examination of the lungs and sputum will generally decide the matter. The age of the patient is of some importance, carcinoma being seldom observed before the age of forty.

PROGNOSIS.—The prognosis is unfavourable in all forms of carcinoma, but progress is least rapid in the scirrhous type. Sarcoma of the larynx seems to extend still more slowly.

TREATMENT.—Partial resection of the larynx has been successful in a few cases of early localised malignant disease, and is the only radical method that seems at present justifiable. Removal of a cancerous larynx was first practised by Billroth, and has since been performed by several Continental surgeons. The results of the operation have, however, been so unfavourable that it cannot be recommended. The obstruction to respiration may demand the performance of tracheotomy; and if the lower part of the trachea is free from disease, the operation will prolong life. Extirpation of a malignant growth by an endo-laryngeal operation should not be attempted.

Pain and dysphagia must be alleviated by the application of cocaine or morphine, as in cases of tuberculosis.

PERCY KIDD.

LATAH.—See ECSTASY.

LATERAL SCLEROSIS.—See SPINAL CORD.

LATHYRISM.—A condition somewhat analogous to ergotism and pellagra, believed to be due to a diet containing the *Lathyrus sativus* and *Lathyrus cicera*, two species of the chickpea, without a sufficiency of ordinary food.

The symptoms are muscular weakness, stiffness, and tremor, especially of the lower extremities, resulting in a peculiar ataxic gait; sensations of 'creeping' and various other paræsthesiæ; exaggerated deep reflexes, and occasionally disorders of the sexual reflex and of micturition and of defæcation. The symptoms develop fully in the course of a month, and disappear gradually if the cause be removed.

LAVAGE (Fr.).—SYNON.: Ger. *Magenauspülung*.

DEFINITION.—A method of washing out the stomach, introduced by Kussmaul.

DESCRIPTION.—The following apparatus is necessary:—(1) A soft flexible india-rubber stomach-tube, about twenty inches in length; (2) a large-sized glass funnel; (3) a yard of india-rubber tubing; and (4) some suitable lubricating material.

One end of the india-rubber tubing is fitted over the end of the glass funnel, and the other is attached to the stomach-tube by the insertion of a small piece of glass tubing, so that the observer can determine when the irrigation is complete by noticing that the returning fluid is quite clear. Two good-sized lateral oval openings should be made in the stomach-tube close to its rounded distal end.

Having moistened the tube with warm water, the practitioner introduces it into the pharynx with his right hand, and gently presses it down, while the patient, who is preferably in the sitting posture, performs the act of swallowing.

To wash out the stomach it is necessary to fill the receiver with fluid, to raise it to the level of the patient's head, and then to add more fluid until the whole of the apparatus is filled. A siphon-action should now be established by depressing the funnel, and the stomach is thus emptied. By repeatedly filling and emptying it as described, it can be thoroughly washed out. In withdrawing the tube it is advisable to pinch it so as to prevent reflux into the larynx. The repugnance some patients have to the use of the tube is generally soon overcome, and with a little practice a patient can acquire sufficient manipulative skill to introduce the tube and wash out his own stomach.

The fluid to be employed in lavage may be either lukewarm water; 1-per-cent. salt-solution; 3- to 5-per-cent. solution of bicarbonate of sodium; 3-per-cent. solution of either boric acid or borax; Vichy Water; or any innocuous fluid possessing antiseptic properties.

In the beginning, lavage may be resorted to once or twice daily, but less frequently as the case improves.

USES.—Lavage is of great service in removing fermenting gastric contents, in cleansing the gastric mucous membrane, and in stimulating the activity of the gastric glands in cases of chronic dyspepsia, especially when associated with excessive secretion of mucus. By its employment we may also increase peristaltic action, and strengthen the enfeebled gastric muscular fibres in cases of insufficiency or atony.

Among the diseases in which lavage has been

successfully employed may be mentioned gastric dilatation ; chronic gastritis ; and intractable cases of chronic dyspepsia. See STOMACH, Diseases of.

It should be remembered that there are certain possible dangers in connection with lavage. The chief of these is injury to the œsophagus or to the gastric walls, with hæmorrhage or perforation. In very rare instances fatal syncope has attended its use. Lavage should never be resorted to in cases of recent hæmatemesis, thoracic aneurysm, gastric ulcer, or advanced pulmonary and cardiovascular disease. The gastric contents should always be examined after they are withdrawn. See STOMACH, Examination of. JOHN HAROLD.

LEAD, Poisoning by.—SYNON. : Plumbism, Saturnism ; Fr. *Intoxication Saturnine* ; Ger. *Bleivergiftung*.

ÆTIOLOGY.—Pure metallic lead has probably no injurious action on the system, but it readily oxidises and forms salts, all of which are poisonous. The fume of the molten metal is composed of the oxide and sulphate of lead, and is so destructive to vegetable and animal life that at lead-smelting works very long flues and high chimneys are built for its removal. The length of the flue allows of the dust being deposited and recovered. Cleaning out of the flue is a dangerous process, and workmen are only allowed inside two hours at a time.

Although lead-salts are undoubtedly poisonous, they are not actively so ; hence *acute* lead-poisoning is extremely rare. A few cases of mild irritant-poisoning have occurred due to the ingestion of large doses of acetate of lead, and cases of acute saturnine encephalopathy have been produced by diachylon (plaster) taken as an abortifacient.

It is with the *chronic* form of poisoning that we have principally to deal ; and as lead is widely employed in the arts and manufactures, and food and drink are frequently contaminated by it, this form of poisoning, often very insidious in its development, may arise under various, and at times unexpected, circumstances.

1. Metallic Lead.—In Great Britain lead-poisoning can scarcely be said to occur among lead-miners, but in Australia where the ore exists largely as a carbonate, plumbism of even the worst type is not unknown. Men who smelt the ore and convert it into pig-lead, workers in lead or its alloys, such as plumbers, solderers, type-founders, and compositors, suffer. Saturnine poisoning too occurs among fishmongers from the metallic coating on their counters, in leather-cutters working on a leaden slab, and in file-makers. Young people engaged in placing leaden capsules on the necks of bottles containing spices &c. have suffered. Ill-defined and sometimes serious symptoms have followed the use of tea wrapped in leaden foil, of snuff similarly enveloped, of canned foods and fruits owing to the action of the juices upon the solder, and of articles of drink contaminated by the employment of shot for cleaning bottles.

Drinking-water is a frequent source of lead-poisoning. Several families in a town or district may be affected at the same time owing to the water having taken up lead in its transit through leaden pipes or its storage in leaden cisterns. Pure water, even distilled water, acts upon lead in the presence of air, and a slightly soluble oxide of lead is formed. Carbonic acid, absorbed by the water from the atmosphere, precipitates part of the oxide as oxy-

carbonate, while the remainder is kept in solution by the free carbonic acid. It is difficult to obtain an absolutely pure drinking-water. It usually contains a trace of organic impurity. The presence of nitrates and nitrites is particularly dangerous, not only on account of their possible origin from sewage, but because their presence enables the water to dissolve more lead. Sulphates, phosphates, carbonates, and silicates in water form insoluble compounds with lead, and becoming deposited on the interior of the pipe form a protective covering. Yet even in these cases the presence of carbonic acid in the water may convert part of an insoluble lead-salt into one that is soluble. Peaty waters have a strong 'plumbo-solvency' as a rule. This has been attributed to the presence of an acid arising from vegetable decomposition. It is probable that the action of water upon lead is more than one of simple chemistry. Power attributed the 'plumbo-solvency' of peaty waters to the presence of two varieties of micro-organisms, and in a series of investigations on drinking-water carried out by Bolam, Mack, and the writer, it was found that while the solvency of water upon lead was influenced by the weather and the character of the salts dissolved in the water, it was frequently proportional to the number of micro-organisms contained therein. Water capable of producing this effect upon lead will to a considerable extent lose this power if filtered through layers of sand and magnesian limestone. It is desirable to change these filtering beds frequently. For the conduit of drinking-water into houses and its storage, galvanised iron and glass pipes have been recommended, and slate cisterns. These, however, are not without danger, for in the Colonies, where in consequence of long periods of drought the water has to be stored in galvanised iron cisterns, epidemics of plumbism have occurred owing to the molten zinc in which the iron plates were dipped having contained lead. Besides, even when cisterns are made of slate the slabs may be joined together by means of red or white lead.

Oxides of Lead.—Persons employed in the manufacture of red-lead, those who handle it or inhale the metallic dust frequently suffer. The writer has seen numerous severe cases of lead-poisoning in workmen engaged in the manufacture of the large galvanic batteries used for telegraph purposes, also in coloured-glass-makers, glass-polishers, and in dip-pers of cheap earthenware.

Salts of Lead.—The most important is the carbonate or white lead. The white-lead factories and the potteries have supplied the largest number of cases of plumbism. In Great Britain preference is shown by house-painters for lead carbonate made by the old Dutch process. The workpeople who empty the white beds in the factory, also those who wash and grind the white lead, frequently suffer from saturnine poisoning, but the part of the Dutch method of manufacture that is most dangerous is the emptying of the stoves or chambers wherein the washed carbonate of lead is dried. Although the stoves are not allowed to be emptied until the temperature has fallen, yet the inhalation of the very fine dust that rises from the shelves will in a few days, if this work is continued, be followed by serious, sometimes by fatal, results. This remark applies with equal force to certain departments of pottery-manufacture. A considerable proportion of earthenware manufactured in this country is

dipped in a glaze that contains a large percentage of white lead; hence the men who dip the ware, those who brush it afterwards or place it in the ovens, majolica-paintresses and litho-transfer-makers are especially prone to suffer from lead-poisoning. Plumbers, colour-mixers, and dye-workers who use chromate of lead run a similar risk. Women who wash the clothes of painters incur to some extent a like danger, as also those persons who use certain hair-dyes and cosmetics. See OCCUPATION-DISEASES.

Lead gains an entrance into the system by the alimentary canal, the respiratory passages, and the skin. Certain people exhibit an idiosyncrasy to lead, some being more readily affected by it than others. Women are more rapidly brought under the influence of lead than men, and are more seriously affected by it. Alcoholism, gout, and poverty predispose to plumbism.

SYMPTOMS.—Sooner or later after the introduction of lead into the system the individual gradually loses colour, his face becomes expressionless, and the skin exhibits a dull earthy hue. This is sometimes spoken of as *Saturnine cachexia*. The patient complains of an unpleasant taste in the mouth, and his breath is fetid. On attempting to show his teeth, which are usually discoloured, there is often considerable tremor of the muscles of the upper lip; the gums may be ulcerated, and at their margin close to the teeth there is usually seen a bluish or violet line, better marked as a rule on the lower than the upper gum, and not present where the teeth are absent. Ulceration and retraction of the gums cause the teeth in front to look elongated. This blue line is a valuable sign, not so much of active lead-poisoning as of the existence of lead in the tissues. The line is due to a deposition of particles of lead sulphide in the interior of large phagocytic cells lying in the layers beneath the epithelial covering of the gums; occasionally it also occurs in the epithelial cells themselves, and is usually most marked in the infiltrating cells present in large numbers at those points where the ulceration of the gum is most active.

Lead Colic.—**SYNON.**: *Colica Saturnina*; Fr. *Colique de Plomb*; Ger. *Bleikolik*.—One of the earliest symptoms complained of is pain in the abdomen, usually of a very severe character, and referred to the neighbourhood of the umbilicus. Pain may develop suddenly, and be so severe that the patient rolls about in agony. In most instances it is aggravated by pressure; in others, pain is relieved by pressure. It is often unilateral, worse in one half of the abdomen than the other. Pain, too, is often elicited in the neck by firm pressure applied along the course of the vagus. This pain is more acute on the side corresponding to that on which the abdominal pain is most severe. The abdominal wall is usually firm and retracted. There may be vomiting which fails to bring relief. Usually during the attack, and for some time previously, the bowels are obstinately constipated, but occasionally there is diarrhoea. In the attack the pulse may be hard and firm, showing heightened arterial tension, but when the pain is severe and continuous it is often extremely feeble and quick. There is distaste for food. The urine is extremely scanty, not more than six to eight ounces being passed daily. If it is a first attack of colic and the patient is young, the urine is generally free from albumen. Sleep is secured with difficulty. There

is a restlessness of body apparently in excess of that caused by the abdominal pain. In a few days, three to five under treatment, the severity of the colic subsides. Lead colic has been mistaken for appendicitis and *vice versa*.

Lead Palsy.—**SYNON.**: Fr. *Paralysie Saturnine*; Ger. *Bleilähmung*.—Following upon colic, but sometimes even without it, there develops motor paralysis. Wrist-drop is the commonest form of loss of power, and in this, as in all instances where paralysis is due to toxæmia, it is double, although it is frequently found worse on one side than the other. Muscular fatigue, the nature of patient's occupation, and the amount of local exposure determine to some extent, but not absolutely, the localisation of the loss of power. Wrist-drop occurs apart from working in lead, as for example by drinking contaminated water. It is symmetrical as well as bilateral. Usually it develops slowly, but the writer has known it occur in an hour or two. It is sometimes preceded by a sense of numbness and tingling. The loss of power is usually first observed in the common extensors of the fingers, and subsequently in those of the index, the little finger, thumb, the radial extensors of the wrist, ulnar, and short supinator. These muscles are not all equally involved, nor always in the order here mentioned. Paralysis of the extensors of the wrists, for example, may be more complete than that of the fingers. There may be too very pronounced paralysis of the wrists and fingers, and yet the patient may be able to extend his index finger. He may be unable to separate his fingers owing to paralysis of the dorsal interossei. The hand falls at a right angle to the forearm: it is flaccid and cannot be raised voluntarily. Any effort made to do so is followed by a considerable amount of muscular tremor, and by the unopposed flexor muscles being thrown into a state of contraction accompanied by irregular movements of such fingers as are not paralysed. While the distal phalanges of the fingers cannot be extended by the patient, he may succeed in raising them if the proximal are supported by the hand of the examiner. It is interesting to note that in wrist-drop the supinator longus, although innervated like the extensors by the musculo-spiral nerve, is not paralysed. This is the commonest form of saturnine palsy, but as other groups of muscles may be affected, it is customary to speak of types of lead-paralysis. In the *brachial* or *Duchenne-Erb* type the muscles involved are the deltoid, biceps, brachialis anticus, and supinator longus. This form of paralysis sometimes succeeds, at other times precedes, and is independent of wrist-drop. The arm becomes flaccid, and hangs powerless by the side in a semi-prone position. Elevation and rotation of the arm outwards are impossible, also flexion and supination of the forearm. In the *Aran-Duchenne* type the phenomena resemble those observed in progressive muscular atrophy. Not only are the thenar, hypothenar, and interosseous muscles of the hand paralysed, but they are also atrophied to a considerable extent; the interosseous grooves are deepened, and the hand presents a 'bird-claw' or a '*main en griffe*' appearance. Since this form of paralysis affects principally file-makers, who are in the habit of tightly gripping the chisel with the fingers of the left hand, it is thought that muscular strain plays a part in localising the paralysis. Bernhardt has published two cases of this form of

paralysis occurring in the right hand of house-painters, where the only muscular strain was that connected with the use of the brush. The muscles affected in the Aran-Duchenne type of saturnine paralysis do not exhibit marked tremor or fibrillation, nor, as a rule, does the lesion advance quickly; in these respects it differs from progressive muscular atrophy, which it otherwise closely resembles.

In lead-palsy the loss of muscular power is not confined to the arms and forearms; the muscles of the legs and trunk may become affected. In some of the writer's cases the muscles of the thighs, pelvis, and trunk were paralysed, and yet, despite this widespread implication of the muscular system, complete recovery took place. In the form of lead paralysis known as *peroneal*, which is usually consecutive to loss of power in the upper extremity, the common extensors of the toes and big toe are affected, while the muscles higher up the leg escape. There is ankle-drop. The foot falls in an inward direction, so that the patient tends to bear his weight upon the external border of the foot. It is seldom that the muscles of the face are affected, but those of the eyeball may be. Sensation, as a rule, is undisturbed, but the writer has observed analgesia. In saturnine paralysis the affected muscles when grasped may be tender, but the pain is never so acute as in alcoholic paralysis. The rapidity with which the palsied muscles atrophy causes recovery as a rule to be slow. Occasionally there is very marked tremor as well as atrophy of the affected muscles. The knee-jerks are often absent. Electrical contractility disappears in the paralysed muscles. It may even precede the loss of voluntary power, and be followed by the reaction of degeneration.

In the absence of a history of exposure to lead, and where no blue line is present in the gums, the diagnosis of saturnine paralysis is sometimes difficult. It might be mistaken for that caused by alcohol or arsenic. Saturnine paralysis usually appears first in the wrists and hands, while alcoholic and arsenical affect the legs below the knee. The muscles and nerves, too, are less tender. In progressive muscular atrophy the paralysis is secondary to the wasting, and the fibrillary tremor is usually of a finer character, and on the whole it is better marked in progressive muscular atrophy.

Other Phenomena.—Sometimes the symptoms of plumbism in female lead-workers assume an hysterical character. Beyond a few apparently insignificant nervous symptoms, usually of an exalted nature, the patient may not seem ill, and yet within the space of two days she may be dead. This form of toxic hysteria is often rapidly fatal. Lead has a particular selection for nervous tissues. White-lead workers often feel ill in the morning—they have headache and feel nervous. Occasionally, when in the factory, a worker, who, a short time previously, complained of severe headache, is observed suddenly to fall to the ground in a state of insensibility and is convulsed. The coma continues and the convulsions keep recurring, in one of which the patient perhaps dies, never having regained consciousness, or the epileptiform seizures pass off only for the patient to find, when he has recovered consciousness, that he has lost his eyesight. Vision may thus be lost temporarily or permanently even at a very early age. The writer has seen several young people, whose ages varied from 18 to 25 years, and who had

worked only a few months in a white-lead factory or a pottery, rendered blind for life. Neuro-retinitis may occur without previous epileptiform seizures. In addition to changes in the optic disc, ophthalmoscopic examination may reveal hæmorrhages in the retina. Acute delirium running on to mania may develop. The symptoms may resemble those of acute meningitis. There may be hemiplegia and aphasia. When the system has been exposed for a lengthened period to the influence of lead, albumen is generally found in the urine. Some authors attribute the epileptiform seizures and blindness to the diseased condition of the kidneys, but in the experience of the writer, while these are frequently associated, fatal saturnine encephalopathy may occur without albumen ever being present in the urine. The association of lead-poisoning and gout is well known. The one predisposes to the other. Women who are the subjects of lead-intoxication often suffer from menorrhagia, seldom amenorrhœa, and, if pregnant, very frequently miscarry. Lead is a strong ecboic. It is almost impossible for a pregnant female lead-worker, who may even not have any symptoms of plumbism, to continue following her occupation without aborting. Miscarriages follow in rapid succession, and many female lead-workers only succeed in bringing forth a living child at full term by withdrawing entirely from their work as soon as they know they are pregnant.

ANATOMICAL CHARACTERS.—A patient may die in acute saturnine encephalopathy, and the autopsy reveal very little naked-eye change. The brain may be dry and shrunken, or its veins congested; the tissue may be pale and watery as in uræmia, or there may be signs of meningitis if the disease has been of long standing. In chronic cases of lead-poisoning the kidneys are contracted, and, on microscopical examination, show a marked increase of the interstitial tissue. This change has come to be regarded as typical of all cases of lead-poisoning. This is scarcely quite correct. In the more acute cases the epithelial cells of the tubules are affected, and apart from the microscopical demonstration of this circumstance, there is also the fact that even in chronic lead-poisoning, although the kidney is contracted and its capsule adherent, the renal tissue is pale, indicating that the kidney was previously enlarged and pale. Owing to the intemperate habits of many lead-workers who die in infirmaries, it is sometimes difficult to say how much of the structural changes in the kidney is due to alcoholism and how much to plumbism. Upon the hæmoglobin of the red blood-cells lead is known to exercise an injurious influence. The action may be direct or indirect through its operation upon the marrow of the bones. Hence arise the anæmia and cachexia observed in the subjects of lead-poisoning—an anæmia which may be persistent. The iron of the broken-up hæmoglobin is eliminated by the skin and urine, and, after death, is found in excess in the liver.

Opinion is divided as to whether the paralysis caused by lead is due to peripheral neuritis or to changes in the central nervous system. While in the atrophied muscles the transverse striæ are found to have become diminished and the connective tissue increased, the nerves frequently exhibit signs of inflammation, viz. thickening of the sheath and increase of the connective tissue-elements, alterations of the medullary sheath, and atrophy of the axis-cylinder. In the subacute cases time has not been

partly by its action on the heart, partly by its dilating effect on the blood-vessels of the splanchnic area.

W. D. HALLIBURTON.

LEECHING.—The local abstraction of blood by means of leeches. See BLOOD, Abstraction of.

LENK, in Switzerland.—Sulphur-waters and climatic health-resort. See MINERAL WATERS.

LENS, Diseases of.—See CATARACT.

LENTIGO.—A synonym for freckle. See FRECKLES.

LEONTIASIS OSSEA.—See HYPERTROPHY, pp. 169 and 702.

LEPOTHRIX (λεπίς or λέπος, a scale; and θρίξ, a hair).—**DEFINITION.**—A term applied to a hair in which there is loosening and partial detachment of the overlapping edges of the scales of its cuticle.

Such hairs are usually met with in the axilla and scrotum; they are dull, dry, and lustreless. Diffuse or nodular masses of concretion encrust irregularly and adhere firmly to the hair. Glasgow Pattison and others have demonstrated the bacterial origin of this incrustation. The concretion on the hairs of the axilla is occasionally of a red colour (see SUDORIPAROUS GLANDS, Diseases of). The affected part should be shaved at frequent intervals and thoroughly cleansed by soap and water and parasiticide preparations.

JOHN HAROLD.

LEPROMA.—See LEPROSY.

LEPROSY (λεπρός, scaly).—**SYNON.** : Lepra; Elephantiasis Græcorum; Fr. *Lèpre*; Ger. *Der Aussatz*.

DEFINITION.—A specific disease, the result of infection by the *Bacillus lepræ*, running a chronic course and tending to a fatal issue. Its presence is characterised by granulomatous infiltration of the skin and mucous membranes, of certain nerve-trunks, and of the viscera, inducing functional irregularities and trophic changes in the affected parts.

GEOGRAPHICAL DISTRIBUTION.—It may be laid down as a general rule that lepers increase in numbers as the equator is approached from either pole. Although isolated cases may occur in any country of *Europe*, leprosy is practically confined, so far as that continent is concerned, to Western Norway, certain districts of the Baltic littoral, Southern Russia, Greece, and Turkey, and several of the islands of the Mediterranean, more especially Cyprus and Crete.

Asia.—From the coasts of Syria on the Mediterranean, right across Asia to the China Sea, leprosy is prevalent, and in many countries amounts to a scourge. Asia Minor, Persia, Turkestan, India, Burmah, and Southern China are mostly infected, the last-mentioned perhaps the most deeply of all. In the more northern latitudes of Asia, Siberia, Mongolia, Manchuria, Corea, and China north of the Yangtze, leprosy is rare or wholly unknown. In Russian Siberia there are one or two leper-settlements, but no general infection of the community. In Northern China and in Manchuria leprosy is not indigenous, and even in Corea the majority of lepers are Japanese. In India leprosy widely and generally prevails. The Annamese are extensively tainted, the dwellers in Cochín-China, Cambodia, and Siam less so.

The Islands of the Pacific.—Leprosy has been known in Japan from early times, and at the present day the disease is very prevalent. In almost every island in the Pacific infection by leprosy has followed the advent of the Chinese labourers.

The Islands of the Indian Ocean.—Ceylon and the Andaman Islands, Madagascar and Mauritius are all leprosy, infected probably from India; while Java, Sumatra, and Penang appear to have been infected by the Chinese.

Australasia.—The Chinese are accredited with introducing leprosy into Australia, and at the present time the majority of lepers are Chinese. Among the Maoris in New Zealand, however, leprosy would appear to be indigenous.

Africa.—Egypt has from the earliest recorded times been the seat of leprosy, and throughout the length and breadth of the African continent no people yet met with has been found free from the disease. Along the Mediterranean shores of Africa, in Morocco, in the Canary, Madeira, and St. Helena Islands, and wherever Europeans have penetrated on the East and West Coasts of the Dark Continent, leprosy is found to prevail. In South Africa so wide-spread is leprosy that a leper-asylum has been instituted at Robben Island, adjacent to Cape Town.

America.—In Canada, leprosy has occurred among Norwegian immigrants ever since they first settled in the country; the disease has, however, gained no marked hold on the inhabitants. In the United States the main seat of leprosy is among the Chinese on the Pacific slope, but among negroes and a few whites, mostly Norwegians, the disease also prevails. In Mexico and Central America lepers are to be seen in all the principal cities. In the West Indian Islands leprosy is frequently met with, chiefly among negroes. In South America, the Guiana States show the maximum of infection; and lepers are met with in every part of the continent except, it is stated, Chili and Bolivia.

LEPROSY AS IT AFFECTS RACES.—At the present moment the Teutonic stock, if we except the Norwegians, seem to be freer from leprosy both in Europe and North America than the Latin races whether they reside in Europe or South America. The whole Indo-European family has, however, at one time or other been leprosy, although at the present day the disease is abating to the westward, but still continues rife to the eastward, especially in Persia and India. The Mongolian family is by no means universally leprosy, the disease being confined mostly to Japan, Southern China, and places apparently infected from these sources.

ETIOLOGY.—Although leprosy can occur independently of any geological, geographical, or climatic conditions, there can be no doubt that a combination of overcrowding, poverty, and unwholesome food renders the individual especially susceptible to leprosy infection. The specific cause, however, is in all probability the *Bacillus lepræ*. Yet, although the bacillus so named is always associated with leprosy, our knowledge of the part it plays in leprosy is still incomplete. How leprosy is communicated from one person to another is unknown, and the disease has never been communicated to the lower animals.

Transmission by contagion.—The possibility of transmission by contagion is still in dispute. *In favour* of such a mode of transmission it may be urged:—that infected immigrants have contami-

nated leprosy-free communities; that leprosy can be communicated from husband to wife, from wife to husband, and from parent to offspring; that leprosy prevails in communities dwelling together in the close contact engendered by overcrowding; that isolation of lepers in an asylum or village is followed by a lessening, and it may be a disappearance, of the disease in the country in which such a step is adopted; and that leprosy has probably been communicated by both inoculation and vaccination. *Against* these arguments it may be maintained that most of the above contentions are open to some other explanation, e.g. the apparent origin of the disease among a reputedly leprosy-free people may be based on an inaccurate medical history of such community, while the cases of inoculation and of reputed communication of the disease by vaccination are too few to draw a definite conclusion from.

Heredity.—It is 'not proven' that leprosy is diffused by hereditary transmission. There is no leprosy physiognomy or physical trait to be noticed in the children of lepers; and when children, born of leprosy parents, are brought up (or suckled) by a leprosy mother, contagion cannot be excluded.

Sexual intercourse.—A single coitus is accredited by many men with the origin of their leprosy infection, but the evidence as to this method of acquiring leprosy is altogether faulty.

Food.—Jonathan Hutchinson maintains that uncooked and rotten fish is the medium by which leprosy is acquired. There is, however, no direct proof of this theory, which is based chiefly on the fact that leprosy disappeared from England (and elsewhere) as the food of the people improved. The scarcity of *salt* or of *rice*, to which some authorities ascribe leprosy, is probably only indicative of a general lack of food.

Vaccination.—Vaccination is not a factor of any great importance. Since vaccination was introduced into Canton, leprosy has actually diminished; and where there were previously two leper-villages, now only one exists. Among Europeans in leprosy countries calf-lymph, or lymph imported from Europe, is mostly used, so that the chance of white children contracting leprosy is infinitesimal. The question as to the presence of the *Bacillus lepræ* in vaccine-lymph obtained from leprosy persons has been tested in Trinidad and in India, where twenty-seven and ninety-three observations respectively were made on this subject, and, except in two doubtful cases, no bacilli were found.

The Bacillus Lepræ.—The parasite appertaining to leprosy may be demonstrated in the 'juice' expressed from a leprosy patch, in the discharge from a leprosy ulcer, and in sections of leprosy tissue. To obtain the 'juice,' a leprosy nodule is compressed between the blades of sinus- or nasal-forceps. The surface of the compressed part is then lightly pricked by multiple punctures, when a clear lymph-like fluid will quickly exude. Another plan of obtaining the 'juice' is to apply a small blister over a leprosy nodule. A smear of the juice on a cover-glass when dried and stained by Ziehl's or Ehrlich's method is first decolourised in 25 per cent. nitric acid for three minutes, then washed in 60 per cent. alcohol, and afterwards in distilled water. The specimen is then dried and mounted in xylol-balsam. When sections of tissue are examined, the same stains may be employed, but the preparation must be left in the stain for twenty minutes. After decolouration the preparation is dehydrated by absolute alcohol,

then immersed in oil of cloves and mounted in xylol-balsam. When thus prepared, the *Bacillus lepræ* will be found to present the appearance of a straight rod, measuring on an average 6μ by 3μ , and, if a contrast stain of methylene blue has been applied after decolouration by the acid, exhibiting bright red points in a blue setting. Vacuoles or uncoloured areas are observable in the stained bacilli; this appearance may, however, indicate the presence of stained 'granules' between unstained protoplasmic material. Spores are stated by some observers to be present, and others consider the parasite motile. These appearances vary with the stain employed; an aniline dye colouring the envelope so as to obscure the contents, whereas with such reagents as iodine, borax-methylene-blue, and osmic acid, the 'granules' colour deeply and the envelope is more transparent.

The *Bacillus lepræ* closely resembles the *Bacillus tuberculosis* in appearance and in behaviour. The differences and similarities are as follows: (1) The *Bacillus lepræ* is somewhat shorter, straighter, more uniform, and stains more readily than the *Bacillus tuberculosis*. (2) Cultivation and inoculation experiments with the *Bacillus lepræ* have all failed. (3) The bacilli in leprosy are found in groups or zooglææ, whether met with within the cells or in the lymphatics; whereas in tuberculosis the bacilli are found in comparatively small numbers, and in more scattered positions. (4) Leprosy selects the skin and nerves for attack, whereas tuberculosis specially affects the lungs and serous membranes.

INCUBATION OF LEPROSY.—There is neither clinical nor experimental evidence whereby even an approximate conclusion can be arrived at concerning the incubation-period of leprosy. Three to fifteen years have been suggested, but on quite unsatisfactory premises. Taking three years as the shortest period, it would explain how, disallowing heredity, leprosy is not met with in the children of lepers until the third year. According to Hoegh as many as twenty-seven years have passed between the time of presumed exposure and the invasion of the disease.

PREMONITORY SYMPTOMS.—There are no signs by which the subsequent development of the ordinary manifestations of leprosy can be foretold, although certain prodromata have been described. The principal of these are attacks of fever, weakness, hyperidrosis, dyspepsia and hepatic derangements, epistaxis, headache, vertigo, rheumatic pains, and various nervous affections such as hyperæsthesia and 'pins and needles.'

VARIETIES.—Although leprosy is a single disease, of a specific character, it presents a marked variability in its clinical features. So definitely do these types persist, that they have been regarded as independent morbid states. The types, however, in many instances blend, and what is termed 'mixed' leprosy results: a condition which tends to negative the idea of independency of the so-called nodular (or tuberculated) and the anæsthetic (or non-tuberculated) types of the disease.

Nodular Leprosy.—*SYNON.*: *Lepa Nodosa*, *L. Tuberculosa*, *L. Tuberculata*; Hypertrophic leprosy.

Prodromal phenomena may be pronounced, slightly marked, or practically absent. When present, especially in children, they consist of languor, drowsiness, profuse sweatings, a febrile state, epistaxis, enlarged glands, and change in the features

and in the character of the skin generally. It is not, however, until the characteristic cutaneous or nervous lesions gradually supervene that a person is known to be leprous.

The eruption.—On the face or ears, on the extremities, and less frequently on the trunk, erythematous-looking patches of various sizes appear during, or immediately after, a febrile attack. The patches, when first seen, consist of mere congestive areas, but the colour deepens until a reddish, bronzed, or coppery tint prevails. These coloured areas are thickened, slightly raised above the surrounding skin, and endowed with a primary hyperæsthesia which gives place, it may be speedily, to a state of impaired sensation. An approach at symmetry in the patches is usual, the two sides of the face or opposite limbs showing simultaneous eruptions, the former usually anticipating the latter. These leprous patches vary in area from the size of a shilling to several inches in diameter. With the subsidence of the feverishness the patient's general condition improves, the blotches on the skin fade and sometimes completely disappear. Traces, however, of the cutaneous lesions may remain in the form of a brownish tint here and there, and of a hardening, though but rarely of an elevation, of the skin. The subsidence of the primary eruption may be so complete as to give rise to a hope of a permanent cure, or to a belief that the diagnosis of leprosy was premature. In the course of a few weeks or months, however, there occurs another febrile attack associated with or followed by a fresh crop of discoloured patches not necessarily affecting the same areas of skin. These may again more or less completely fade away, or may leave patches which present a deep pigmentation and a hard infiltrated base. The second attack will be followed by others at irregular intervals, but it may not be until several such 'leprous storms' have come and gone that a permanent nodular thickening is left.

The second stage of nodular leprosy dates from the appearance of local deposits, which exist as raised masses of infiltration or 'nodules.' These nodular or tuberculated excrescences may be the first intimation that the illness is leprosy; for it occasionally happens that the febrile attacks and early cutaneous changes are unobserved or but slightly pronounced. The infiltrated patch, termed the *leproma*, may appear on the site of a previous macula or altogether independent of it. In consistency the lepromatous crop presents elevations which are at first firm and somewhat elastic. Their size varies, an infiltrated patch may cover an extensive area of skin, and the 'tubercles' may vary in size from that of a pea to a walnut, or even a pigeon's or hen's egg; the colour in white people is at first pink, changing to a deeper red, and finally to a dirty brown; in black races the leproma presents at first clear, rather transparent-looking, papules, which when pricked do not exude serum. The tumours may be discrete, aggregated, or they may coalesce. The skin over them looks coarse and greasy, and, when the elevations are decided, devoid of pain. Anæsthesia may obtain at first, but this usually gives way to normal sensation. Once more the disease may apparently subside, the lepromata becoming practically absorbed. On the other hand, several nodules may remain until another febrile attack ensues, when a fresh deposit takes place in the same or in previously unaffected regions of the skin. As crop succeeds crop with

recurrent attacks of fever, the skin of the face becomes permanently affected, and takes on the 'leonine' aspect so typical of nodular leprosy, a condition due to cutaneous and subcutaneous infiltration. Thickened masses cover the malar bones; the skin of the forehead protrudes, with deep lines between corresponding to the natural folds of the skin; the nose broadens; the eyelids look puffy; the chin appears hypertrophied; the ears enlarge, the nodules affecting the lobule and helix. On the limbs lepromatous nodules appear, especially on the backs of the hands and wrists and the outer aspects of the forearms. The dorsal aspects of the feet are favourite sites, but in this region they are more flattened as a rule than are the nodules on the hands. On any part of the limbs or trunk hyperplasia of the surface-tissues may develop with more or less symmetry, although the lepromatous eruptions on the back, on the front of the chest and abdomen, on the neck, on the palms and soles, are seldom pronounced. The skin over the shins, knees, and elbows tends to become harsh and scaly. Of the penile tissues the prepuce is most frequently attacked. Even the male nipple may become thickened. Although the hair of the scalp seldom falls, the hair elsewhere drops off, the eyebrows being early affected and lost. Coincidentally with the loss of hair the nails are lost or altered in appearance, remaining for a time as horny stumps.

Special Senses.—The eye usually shows destructive changes. Beginning usually with the infiltration of the conjunctiva, the leprous material may implicate the cornea, the iris, the ciliary body, the choroid, and the retina, usually in the order given. The eye may be, during the invasion, the seat of severe pain for a time, and finally the whole organ may be reduced to a mass of disorganised structures with total loss of sight. In some cases the eye bulks larger than normal, so that it is impossible for the eyelids to close. The nose is early attacked, the 'stiffness' of the nasal passages showing a hyperplasia of the mucous membrane; ultimately the cutaneous tissue thickens, the nose appears flattened, and the cartilages give way. The external ear exhibits thickened tissue almost from the onset of the disease, pendulous masses depending from the lobule, and the whole ear becomes stiffened and indurated. Hearing, however, although impaired at times, usually remains fairly good throughout the life of the leper.

The *mucous membranes.*—Deposits occur on the insides of the cheeks, on the fauces, in the epiglottidean folds, the vocal cords, and about the larynx, giving rise to huskiness of voice and changes in intonation.

The subsequent development of the nodules in leprosy varies: (1) the nodules may disappear after the subsidence of a febrile attack, leaving a cutaneous stain or deformity; (2) they may remain stationary or increase but slowly in size; (3) they may increase in size rapidly with each successive outburst of fever, the most recent adding to their bulk with each attack, the older becoming harder, denser, and smaller; (4) the nodules usually ulcerate sooner or later. These ulcers may heal, leaving cicatricial deformities; or they may extend and coalesce, so that a large area of a limb is involved, the granulations on the ulcer bleeding readily. Occasionally the ulcers become phagedenic, the parts attacked being rapidly destroyed.

Along with the local manifestations of leprosy,

constitutional symptoms co-exist. The febrile attacks are the most pronounced evidence of the general effect of the presence of the *B. lepræ*. The occurrence of fever is irregular as to its periods of recrudescence, the intervals between attacks varying from weeks to many months. The initial outbursts are usually shorter and sharper than those in the later stages, when the rise of temperature is more irregular and continues longer, rigors, sweating, and a morning and evening temperature of about 100° F. to 103° F. respectively being usual. During the apyrexial intervals the temperature remains continuously subnormal. Dyspepsia is a common feature of the ailment. The bodily strength remains for a time unimpaired, but gradually the vigour languishes, and in the later stages slight exertion is followed by exhaustion and prostration. A cough, at first laryngeal in origin, harasses the patient when the disease obtains a firm hold, and, finally, pulmonary tuberculosis usually supervenes and leads to a fatal issue. Instead of, or in addition to, pulmonary lesions, visceral changes, especially in the kidneys, are common features of the last stages of the leper's life. The average duration of life in nodular leprosy after the onset of the disease is from four to eight years. In the badly-cared-for two or three years may be the limit; but in the better-off class of lepers eight to twelve years of life may be expected.

B. Nerve-Leprosy. — *SYNON.* : Anæsthetic, Smooth, Non-tuberculated, Atrophic, Mutilating, or Dry Leprosy.

PRODROMATA.—Febrile attacks play little or no part in the precursory stage of anæsthetic leprosy, but in the course of the nerve-trunks and in their terminations pains of an indefinite character prevail, which at this stage of the ailment are usually put down to rheumatism or neuralgia. Occasional shooting pains in the nerves, with tenderness when the trunk is pressed upon, and a hyperæsthesia, or, it may be, a partial anæsthesia of certain patches of the skin, indicate some perversion of nerve-function. In the prodromal stage, also, weakness and even wasting of the muscles of the forearm may occur before it is possible to recognise leprosy as the cause.

Eruption.—Twelve months of prodromal symptoms may elapse before cutaneous signs develop; and as the skin-affections and the changes in the nerves and the general signs and symptoms alter as they progress, it is convenient to describe them under three stages:

The first or eruptive stage is characterised by the appearance of an eruption, by nerve-changes, and by commencing atrophy. The eruption consists of spots or blotchy discolouration in the skin. So indefinite is the alteration in the colour of the skin in the white and yellow races, that it is only in a particularly favourable light, or sometimes when viewed at some little distance from the patient, that the eruption can be seen at all. A pale or light yellow tinge in the fairer races, and a bright yellow hue in the darker races, appears on the skin in small discrete areas, possessing usually a circular outline. The spots seldom exceed an inch in diameter, they are only partially, if at all, anæsthetic, and are accompanied by itching or occasionally a burning sensation, especially when rubbed. No elevation attends the spots of eruption, and the margins merge into the surrounding skin. It is on the back, the extensor aspects of the upper or lower limbs, and less frequently on the face, that the

eruption first appears; the spots are for the most part symmetrical, and they frequently appear to develop along the course of a nerve-trunk. Concomitant with the eruption, changes can be made out in the nerve-trunks; and if attention is drawn to the nerves prior to the spots appearing, the tenderness and the increase in size in the nerve-trunks are found to have preceded the discolouration of the skin. Anæsthesia, however, is rare at this stage; but on the affected skin the secretion of sweat is arrested, tactile sensibility is partially in abeyance, and the muscular power is impaired, as evidenced by tremor, weakness, and feebleness of grasp. The first stage of nerve-leprosy may last for one, two, or more years.

The second or developing stage.—The affected patches of skin during this period of the disease show a serpiginous tendency, their edges being raised and scaly, and their surfaces becoming anæsthetic and wrinkled. The pallor or even whiteness of the centres of the patches is marked, the hair over them also becomes white before falling, and the secretion of sweat is completely suspended. Anæsthesia becomes a prominent feature; not only do the patches lose sensation, but the anæsthesia may extend on the upper extremities as high as the elbows and arms, and in the lower extremities to the knees and thighs. So complete is the anæsthesia that neither pricking nor pinching, heat nor cold, elicit any response; and the skin being devoid of feeling, accidental burns are common, causing wide and deep cicatrices on occasions. Bullæ appear on different parts of the body and limbs; they are usually quite small in the first stage of the disease, but later they may be of large size, several inches in diameter, appearing singly and quite suddenly. It is during the bullous eruption that fever and enlargement of lymphatic glands are met with in this variety of leprosy. The bullæ rupture a few days after formation, discharging a serous material which crusts and then falls off, leaving a pale, sharply defined, and anæsthetic patch. The coalescence of adjacent bullæ accounts for the rapid and serpiginous advance of anæsthetic patches on the skin. As time advances, the anæsthesia becomes deeper, the muscular strength wanes, the finger-pulps shrivel, the flexor tendons of the fingers (especially the fourth and fifth) contract, and the whole hand becomes deformed. The stages of change in the hand are usually as follows: Diminution of tactile sensibility, muscular weakness, contraction of tendons, wasting of the hand-muscles generally, atrophy of subcutaneous tissues, shrinking of the skin, absorption of the phalanges and dwindling of the hand. The nails become hard, contracted, clawed and pegged; the whole or part of a finger may drop off; or a dead piece of bone may be shed, and the nail may adhere to the second phalanx or to mere stumps projecting from the knuckles. Changes in the feet may also run more or less concurrently with those of the hand, perforating ulcers, œdema, and 'spontaneous amputation' of the toes by a process resembling that met with in *ainhum*, being common. With these changes the leper becomes weaker, thinner, and more apathetic; the nerve-trunks become thicker; and mutilation in the face, ears, and limbs proceeds apace. The patient's temperature is as a rule subnormal; or, while that of the extremities is much below normal, the trunk-temperature may be slightly raised.

The second stage may last for 6, 10, or more years.

The third or permanent stage.—A marked feature of this stage is the development of muscular paralysis. The third and seventh cranial nerves become affected, causing paralysis of the ocular and facial muscles; the affection attacking one or both sides. The muscles of the limbs are likewise paralysed, a fact specially noted during walking, the feet being lifted high and the limbs being swung or dragged along. Spontaneous amputation of the limbs may extend beyond the fingers and toes, and the wrist or ankle become the seat of separation; the part beyond the line of demarcation taking on a form of mummification in the upper extremity, but more often a moist form of gangrene in the lower extremity. During the third stage, the advance of the disease may be for a time arrested; and even a seeming accession of strength and sexual power may re-appear, and may continue for a time. The length of life of a leper suffering from anæsthetic leprosy is indefinite. Some lepers, if well cared for, may live for 20 to 30 or more years after developing the disease.

C. Mixed Leprosy.—When, in addition to the development of nodules in the skin and tissues, anæsthesia and thickening of the nerve-trunks occur, with some mutilation, the variety of leprosy is said to be ‘mixed.’

ANATOMICAL CHANGES.—The *Bacillus lepræ*, although the determining factor in leprosy changes, is not found generally distributed throughout the body. The presence of the bacillus in the blood has been both asserted and denied. In nerve-leprosy the bacilli are rarely found in the anæsthetic patches, in the fluid obtained by blisters over these patches, or in the pus flowing from the ulcers.

A section of a *cutaneous nodule* of some standing shows the epidermis to be attenuated, the true skin thickened and firmer than usual, and the subcutaneous tissues the seat of a diffuse infiltration. The walls of the blood-vessels, the nerve-sheaths, and the intraneural connective tissues are increased in bulk by the presence of a semi-gelatinous or lardaceous substance. The several glands of the skin, the papillæ, and the hair-follicles disappear in nodules of long standing. Microscopical examination of the nodules shows a marked infiltration of cells massed around the blood-vessels, the lymphatics, and the nerves. Giant-cells, laden with bacilli, may attain a size three or four times larger than a leucocyte. Bacilli are, in addition, often found in dense masses (globi) believed to be sections of lymphatic vessels stuffed with bacilli which have caused the coagulation of the lymph.

Neither in the sections of erythematous maculæ nor in those of anæsthetic patches are the bacilli usually met with, a diffuse fibroid degeneration with incomplete obliteration of the structures peculiar to the skin being the change most apparent in the latter. The nodules of the mucous membrane resemble in a general way those seen in the skin; they are, however, as might be expected, of a softer consistence.

The nerve-trunks in leprosy may attain a size three or four times larger than the normal. The swellings on the course of a nerve are not uniform, and the enlargements when cut into may appear of a pinkish, grey, or yellowish colour. The bundles of nerves are separated by the new-growth, the

nerve-tissue appears of an ashen-grey colour, and both the peri- and endo-neurium are infiltrated. The neurilemma itself and the axis-cylinder may degenerate, when the nerve becomes reduced to a mere fibrous cord. The nerves most frequently attacked are the ulnar, the median, the peroneal, the posterior tibial, and the branches of the superficial cervical plexus. Lesions of the spinal cord, such as sclerosis, meningitis, and changes in the posterior cornua, have been recorded in lepers, but they would seem to be exceptional, if indeed they are not altogether independent of leprosy infiltration. The liver, the spleen, the testes, and other organs may show typical leprosy cells in their tissues, although there are no macroscopical changes.

DIAGNOSIS.—The eruption or the development of anæsthetic patches on the skin is usually the first suspicious feature indicating a possibility of the disease being leprosy; and the discovery of the *Bacillus lepræ* in the ‘juices’ of the subcutaneous tissues or in the sheath of a nerve serves to clinch the diagnosis. Among a leprosy-infected people, syphilis, tuberculosis, ainhum, various forms of skin-diseases such as erythema, psoriasis, leucoderma, keloid, and several of the parasitic ailments of the skin are the more common diseases mistaken for leprosy. In regard to skin-affections their presence is apt to obscure the fact that leprosy is present; almost all the poorer lepers suffer from general scabies, and the macular patches are apt to be overlooked or altogether obscured by the eruptions due to scratching. When, however, anæsthesia is present, and the hair falls off; when, after a hypodermic injection of pilocarpine, patches of skin are found not to perspire; when the trunks of the nerves are found tender and thickened, and when the *Bacillus lepræ* is met with in the fluid of bullæ, &c., the diagnosis is established beyond doubt.

PROGNOSIS.—Although cases of leprosy are recorded, and perhaps proved, to have been ‘spontaneously’ cured, leprosy must be regarded generally as a disease from which recovery is impossible. The prognosis for nodular leprosy is highly unfavourable, and a fatal issue may be expected during any period from two to eight or ten years from the onset of the disease. An acute case may occasionally destroy life in a year or two, but it is more common for some intercurrent or super-vening ailment to develop, and carry off the patient. Bronchitis, pneumonia, pulmonary tuberculosis, or laryngeal constriction may attack the respiratory passages; Bright’s disease may hasten the end; or an intestinal flux, frequently of a dysenteric nature, may be the immediate cause of death.

A sufferer from nerve-leprosy is, however, in quite a different position from one afflicted with the nodular form of the disease; and the recorded cases of recovery are invariably of this variety of the disease. In these cases the activity of the bacillus seems to subside, and although the patient is left mutilated in various parts, the disease may cease to progress, and a long life of twenty to thirty or more years subsequent to the outbreak of the disease may be granted to the patient.

TREATMENT.—*Isolation.*—Although complete isolation of the leper is impossible, yet a modified system of segregation has, especially in Norway, seemed to do good. In India and China, leper-villages are plentiful, but they imply segregation in

a very limited sense, the lepers being free to come and go, beg in the streets, or sell the wares they have themselves manufactured. The general consensus of opinion is against lepers being allowed to beg or trade; more especially is this the case in the ulcerative stage of the disease, when the discharges from their skins, and it may be their respiratory and digestive tracts, are swarming with bacilli. It would seem, therefore, advisable that lepers should be isolated in asylums. Children born of lepers should be immediately removed from their surroundings and reared apart.

Hygienic and dietetic treatment.—A sufficiency of clothing and food, and good hygienic surroundings, serve to prolong the life of a person who has contracted leprosy. Cleanliness, avoidance of fatigue, and an open-air life, all aid materially in alleviating the virulence of the disease. So well recognised is this fact that many believe that, as in tuberculosis, so in leprosy, open-air treatment and all the dietetic and hygienic care that is bestowed with marked good in that disease will also alleviate, if not actually stay the progress of leprosy. In some hospitals the lepers are advised to come to the hospital twice yearly, when they are well fed, clothed and housed, and given cod-liver oil, iron, and other tonics with marked benefit. Under such treatment lepers improve temporarily, and it would appear that life may be prolonged indefinitely.

Medicinal.—Of the many 'cures' for leprosy which have been introduced none are in any sense specific. 'Chaulmoogra oil' and its active principle (*gynocardic acid*), taken internally in doses of from 10 to 40 minims of the oil thrice daily, and applied externally over the affected parts of the skin and joints, has some reputation as a curative agent. The oil may be introduced hypodermically. Gurjun oil, at one time highly spoken of, is now almost neglected. It is significant that in the district in which gurjun oil is produced—the Muar district of Johore—leprosy prevails widely. Cowti oil has been tried in Bombay. What is known as the Beaupterthuy treatment consists in supplying good food and general hygienic measures, in applying as a caustic the oil of the cashew nut, and in administering mercury and alkalis. Temporary improvement has followed this treatment. Arsenic in gradually increasing doses, perchloride of mercury by the mouth or hypodermically ($\frac{1}{2}$ grain weekly, Crocker), iodide of potassium in increasing doses, salicylate of sodium, salol, chlorate of potassium in doses of from 80 to 100 grains daily, have each and all been tried more or less extensively with seemingly good temporary effects. It must be remembered, however, that a leper under medicinal treatment is usually also under improved dietetic and hygienic surroundings, and it is difficult to differentiate between the beneficial effects of the purely medicinal and the hygienic measures. Unna has employed what may be termed 'reducing' remedies. The ointment he recommends for the body is: R Chrysarobin, 5 per cent.; salicylic acid, 2 per cent.; ichthyol, 5 per cent.; made with lanolin, or other vehicle, into an ointment for the skin and rubbed over the affected part once or twice daily. When the ointment is intended for the face pyrogallol is substituted for the chrysarobin.

Other remedies employed in leprosy are thyroid gland, methylene blue, aristol, euophen, naphthol, and resorcin, &c. Anti-venene has been tried in leprosy with apparent benefit. Leprine, an ex-

tract prepared from leprosy tissues, has also been employed, with indifferent results; and the hot-bath treatment of the Japanese has little to recommend it.

When for any reason a *surgical operation* is deemed necessary or expedient in a person suffering from leprosy, it may be unhesitatingly undertaken. Wounds in lepers heal readily. The removal of dead bone, amputation, nerve-stretching, the opening up of sinuses, operations on the eye, and tracheotomy, may all be hopefully undertaken for the relief of leper-patients whenever such operations are required. Perforating ulcers of the feet, a common ailment in leprosy, are perhaps best dealt with by passing a bistoury from the ulcer in the sole right through to the dorsum of the foot and carrying the knife forwards to emerge at the cleft between the toes; the wound thus made usually granulates up readily.

ROBERT LIVEING.

JAMES CANTLIE.

LEPTOMENINGITIS (λεπτός, delicate or thin; and *meningitis*).—A term signifying inflammation of the pia mater. See MENINGES, Diseases of.

LEPTOTHRIX (λεπτός, delicate or slender; and *θρίξ*, a filament or hair).—*Leptothrix buccalis* is a name assigned by Robin to certain vegetable parasites or minute filaments, which can be recognised, by means of the microscope, among the epithelial scales of the tongue or other parts of the mouth; and especially between the teeth, or in the hollows of decayed teeth. They occur in healthy persons, as well as in the sick, and have in this situation really no pathological signification.

Dermatologists also employ the term 'leptothrix' to indicate a morbid thinness and weakness of the hair.

LESION (λῆδο, I hurt).—This word originally signified a hurt or an injury; but its use is now extended to comprehend all organic changes of a morbid character, affecting an organ or tissue.

LETHARGY (λήθη, oblivion; and ἀργία, idleness).—A disorder of consciousness, which consists of prolonged and profound sleep, from which the patient may be momentarily aroused, but into which he falls off again immediately. One form of it corresponds with the final stage of hypnotism. See CONSCIOUSNESS, Disorders of; TRANCE; INSANITY (Mental Stupor); and NEGRO LETHARGY.

LEUCINE (λευκός, white).—SYNON.: Fr. *Leucine*; Ger. *Leucin*.

Leucine or amido-caproic acid is a decomposition-product of the complex proteid molecules, when these are acted upon by strong acids or alkalis, or when they are submitted to the prolonged action of the pancreatic ferment trypsin. It is a product of intestinal digestion, but does not occur normally in the fæces or urine.

It is now regarded as an antecedent of urea in the decomposition of proteids; the transformation in all probability occurring in the liver.

CHARACTERS.—Pure leucine is soluble in water, acids, and alkalis, slightly in alcohol, insoluble in ether. It sublimes without decomposition.

Microscopically, leucine, as it occurs in the urine, appears as oil-like, highly refracting, laminated, crystalline, globular masses, obscurely radiating; but, when pure, leucine forms white, refractile,

glistening, flat crystals (*see* URINARY DEPOSITS, *Coloured Plate*), having an unctuous feel. The microscopical test is not a thoroughly reliable one; hence we must have recourse to the following method: Free the urine of albuminous substances, and precipitate it with basic lead acetate; filter, and pass sulphuretted hydrogen gas through the filtrate to remove the excess of lead; filter, and evaporate the filtrate to dryness. Extract the residue with boiling alcohol; filter, and evaporate the filtrate to a syrupy consistence, when, if present, leucine will crystallise out.

Chemically, leucine is to be detected by the following reaction: A little urine mixed with nitric acid is carefully evaporated to dryness on platinum foil; a nearly imperceptible colourless residue is left. If caustic soda is added to this residue, and heat applied, the leucine will be dissolved, and according to its degree of purity the solution thus formed will be either colourless or of a yellowish-brown colour; and on concentrating the fluid by heat on platinum foil, an oily drop is formed, which rolls about on the surface of the foil (*Scherer's test*).

PATHOLOGICAL SIGNIFICANCE.—Leucine has been detected in nearly all the tissues of the body, more especially in the glandular tissues. It is found in the liver, kidneys, pancreas, the thymus, thyroid, salivary, and lymphatic glands, and in the brain. Leucine and Tyrosine have been detected in the urine only in cases of acute yellow atrophy of the liver, and in the atrophied hepatic cells in the same disease (*see* LIVER, Atrophy of, Acute Yellow). Tyrosine is invariably found associated with leucine. *See* TYROSINE.

JOHN HAROLD.

LEUCOCYTES.—*See* BLOOD, Morbid Conditions of.

LEUCOCYTHÆMIA (λευκός, white; κύτος, a cell, and αἷμα, blood).—**SYNON.**—*Leucæmia* (Virchow); *Fr. Leucocythémie*; *Ger. Leukämie*.

DEFINITION.—A disease, characterised by a considerable and persistent increase in the number of white corpuscles in the blood, associated with enlargement of the spleen and disease of the medulla of the bones, or with enlargement of the lymphatic glands.

Two varieties of the disease are now recognised: (1) *Spleno-medullary* (lieno-myelogenic) *leucocythæmia*, *myelocythæmia*, or *myelæmia*; characterised by enlargement of the spleen, by changes in the medulla of the bones, and by the presence of blood in which the increase of the white corpuscles is due chiefly to the unwonted presence of myelocytes, and to enormous numbers of polymorpho-nuclear granular leucocytes, of which an important proportion are eosinophile; and (2) *lymphatic leucocythæmia*, *lymphocythæmia*, or *lymphæmia*; characterised by enlargement of the lymphatic glands, and by blood in which the very numerous leucocytes consist almost entirely of lymphocytes (*p. 156*). It must be admitted that the two groups do not stand sharply apart from one another, since there may occur on the one hand cases of lymphocythæmia without any enlargement of the glands, and on the other cases of enlargement of the spleen and glands with changes in the bone-marrow, and blood containing large numbers of myelocytes, lymphocytes, and polymorpho-nuclear leucocytes;

but it is on the whole convenient to consider these groups separately. The spleno-medullary form is very much more frequent, and will be first described.

1. Spleno-medullary Leucocythæmia.

ÆTIOLOGY.—This variety is more common in men than in women. It may occur at any period of life, being most frequent between thirty and forty years of age. Deficient hygienic conditions, the disturbances of pregnancy, and the climacteric in women have been thought to have some influence. Gowers found that in women it was most frequent between forty and fifty; and that some cases began during pregnancy, and others soon after it. Malarial fever has been an antecedent in many cases; but a causal connection is uncertain. Race, heredity, and social position have no marked relation to the occurrence of the disease.

ANATOMICAL CHARACTERS.—*Blood.*—The blood is paler than normal, and may be greyish-red in colour. In extreme cases coagulation is imperfect; a grumous chocolate-brown mass results. After defibrination three layers form:—red corpuscles, white corpuscles, and serum. Under the microscope the white corpuscles are seen to be in great excess; instead of two or three in the field, there may be several hundreds. Enumeration (*p. 150*) shows that not only are the leucocytes increased, but the red corpuscles are diminished to such an extent that the total number of corpuscles is less than the normal. Instead of 5,000,000 per cubic millimetre there may be only 2,500,000, or even 1,000,000. On the other hand the leucocytes may be increased from the normal (7,000 to 10,000) to 600,000 per cubic millimetre, though the number very rarely rises over 300,000, and may be less. The proportion of the white to the red corpuscles may be 1:20, 1:10, or possibly 1:1; but the higher proportions are always largely due to the marked diminution of the red corpuscles, as well as to the increase of the leucocytes. In recording observations, the number per cubic millimetre of both red cells and leucocytes should always be stated (*p. 151*).

The further examination and enumeration of the leucocytes must be made with the help of blood-films (*p. 153*). The following changes are then observed (*see Coloured Plate, p. 156*). Of the leucocytes present (1) from thirty to fifty per cent. are so-called *myelocytes*, large cells, possessing a single large nucleus staining faintly, and fine oxyphile granules in the protoplasm. These cells are not found in normal blood, but are present in normal bone-marrow. (2) The *polymorpho-nuclear finely granular oxyphile cells*, forming the bulk of the leucocytes (70–80 per cent.) in normal blood, are enormously increased in number, although owing to the presence of the other varieties they may now only form 50 per cent. (3) *Eosinophile cells* are increased very nearly in proportion to the general increase of the leucocytes—that is, while they are more numerous, their percentage is not very different from what it is in health. Some of the coarsely granular eosinophile cells resemble in the shape of their nucleus the characters of myelocytes. (4) Sometimes cells whose coarse granules stain with basic dyes are seen (*mast-cells*). (5) The *lymphocytes* are relatively diminished from the 15 or 20 per cent. of normal blood to 7 or even 3 per cent. The red corpuscles may present slight differences in size and in shape (*poikilocytosis*). In addition, there are nearly always *nucleated red*

corpuscles (normoblasts) in considerable number. The fall in hæmoglobin is greater than the fall in red corpuscles, so that the colour-index (p. 152) of each red corpuscle is less than normal. The specific gravity of the blood is reduced from 1055, the mean in health, to an average of 1042, the change being due to an increased proportion of water, from 790 parts per 1000 in health to 840 in leucocythæmia. The fat and fibrin are increased as the iron is diminished. An interesting fact is the great increase of the xanthin-bases, which are believed to result from the destruction of the leucocytes. Lactic, formic, and succinic acids have also been found, and the alkalinity of the blood is slightly reduced. The octahedral Charcot-Leyden crystals have been found in the blood, and in many organs after death. They are not peculiar to this disease. See CHARCOT-LEYDEN CRYSTALS, p. 272.

Organs.—The *spleen* is always enlarged. Instead of the normal 6 to 10 ounces, it generally weighs 5 or 6 pounds; sometimes it is as little as 1 pound; and in one case it reached 18 pounds. It is uniformly enlarged, and maintains its normal shape. The capsule is thickened in places, and the organ is more or less adherent to the abdominal wall, diaphragm, or adjacent viscera. On section it often has a brownish rather than a red colour, and is homogeneous or marked with paler lines, due to thickened trabeculae. It is smooth, hard, and dry. Not infrequently there are large conical infarcts, triangular on section, yellow in colour or caseous if old, red if recent. The change in the spleen is one of great increase of the splenic pulp and of the trabecular tissue, the pulp being densely packed with leucocytes of the same kind as those found in the blood; the Malpighian corpuscles are usually inconspicuous.

The *bone-marrow* is either soft and yellow, like pus, or more commonly firm and pink. In either case, the fat of the normal adult marrow is replaced by a cellular substance, in which are found myelocytes, nucleated red corpuscles of various sizes, cells containing degenerating red cells, and giant-cells. These changes may be found in all the bones. It is most marked in those which possess most spongy tissue, as the ribs and vertebrae, but also occurs in the long bones. The compact tissue may be thinned and the bones enlarged or perforated (Mosler) in consequence. The lymphatic glands are not commonly enlarged in the spleno-medullary form; and, if enlargement does occur, only small groups of glands are involved. In such cases the periphery of the glands may present leucocytes similar to those found in the blood, and differing from the lymphocytes in the centre of the gland.

The *liver* may attain a weight of 5 or 6 pounds. It is uniformly enlarged, pale and smooth, presenting no obvious change in structure to the naked eye; though on close examination it is seen that the portal tracts are wider than normal, and the microscope shows that they are infiltrated with leucocytes, while the capillaries of the organ throughout are distended by similar cells. Fatty degeneration and atrophy of the liver-cells are also common. Hemorrhage may occur under the capsule of the organ.

The *kidneys* present very similar changes, slight if any enlargement, moderate pallor, more or less infiltration with leucocytes, and occasionally hemorrhage under the capsule, under the pelvic mucous membrane, or into the tubules or glomeruli. The renal epithelium may be granular or fatty.

In the *heart* there is often fatty degeneration of the muscular fibres, and both endocarditis and pericarditis may be among the terminal events of the disease.

Similarly the *lungs* may present the changes of bronchitis or oedema; and the pleural cavities frequently contain dropsical or inflammatory exudation.

In the *brain* hæmorrhages constitute the most frequent change, and are sometimes of sufficient size to be the immediate cause of death. Rarely diffuse sclerotic changes and scattered areas of acute inflammation have been found in the brain and spinal cord. Hæmorrhages are also found in the *retina*, in the *ear*, and under the *skin*. The former can be seen during life by the ophthalmoscope, and are accompanied by white spots and streaks, due to accumulation of leucocytes in the tissues and along the lines of the vessels, as well as by a marked enlargement and tortuosity of the retinal veins. The condition has been described as *leucocythæmic retinitis*.

SYMPTOMS.—The onset of spleno-medullary leucocythæmia is often very insidious. The patient suffers quite unaccountably from languor, weakness, loss of flesh, and perhaps shortness of breath. This may go on for some months, until at last the increased size of the abdomen leads to an examination, at which is revealed an enlarged spleen, already perhaps occupying half the abdomen. The early symptoms may include some sense of distension or pain in the splenic region, slight swelling of the feet, and even decided pallor; but it is very important to observe that the face often retains a good colour—that is, there is no obvious anaemia for a long time after the spleen is greatly enlarged, even when the blood presents in a marked degree the characteristic changes. In many recorded cases the early stages of the changes in the blood and spleen have not been observed. When discovered the spleen is thus often of large size, reaching three or more inches below the umbilicus, and within two inches of the middle line. It is distinguished by its sharp edge and one or two pronounced notches. When larger it may extend below to Poupart's ligament, and even into the pelvis so as to be perceptible by vaginal examination (Spencer Wells); in front it may reach and cross the middle line, the lower end extending for three or four inches obliquely beyond the umbilicus. This curved position of the organ is determined by its vascular attachment to the coeliac axis. The surface is smooth, firm, and sometimes slightly tender. Occasionally a peritoneal rub is felt or heard over it, and rarely a vascular murmur may be detected with the stethoscope. The presence of the enlarged spleen causes considerable enlargement of the abdomen, and often some pressure on the thoracic viscera. In an earlier stage or in a less marked case the spleen may only reach a few inches below the left costal margin. The edge of the liver is often two or three inches below the right costal margin; it is smooth, and moderately firm. The condition of the medulla of the bones is often not manifest during life; sometimes the sternum or the shaft of a long bone is tender to pressure or percussion.

The temperature is often, but not always, raised; and periods of considerable pyrexia may alternate with others in which there is little fever. The cases in which there is most pyrexia are usually the

most rapid. The pulse if little affected in early stages becomes rapid later. The patient may remain for several months, or even a year or two, with but little change, only slowly becoming weaker, paler, and thinner, with increasing size of the abdomen. But eventually, and in a few cases much more rapidly than is above indicated, serious circulatory difficulties supervene. The heart's action becomes enfeebled, the pulse is rapid and weak, palpitation occurs, the impulse may be displaced, and a systolic murmur is heard at the pulmonary cartilage, or even at the heart's apex. Dropsical effusions are common, the feet become edematous, and ascites or pleural effusion is recognised. Epistaxis often occurs comparatively early, but other hæmorrhages take place towards the end, from the bowels, stomach, lungs, or uterus, and into the joints and the connective and subcutaneous tissues. As already stated, they are found *post mortem* in many of the viscera. The hæmorrhagic tendency is such that injuries may give rise to serious loss of blood; the extraction of a tooth or a puncture for paracentesis has led to death; and excision of the spleen has more than once proved quickly fatal from this cause. In addition to the dyspnoea, which results partly from the anæmia, partly from the enfeeblement of the heart, and partly from the great size of the abdominal viscera, the patient may be short of breath from the occurrence of bronchitis, of œdema of the lungs, or of pleural effusion. For like reasons cough is of frequent occurrence. The functions of the stomach are interfered with by pressure, and indigestion and vomiting occur, as well as diarrhoea, which may be accompanied by hæmorrhage. The urine is acid, of high specific gravity; the urea is not much altered in amount, but there is an increase of uric acid and of the xanthin-bases. Albumen is not commonly present, except towards the end of the disease. In women menstruation is often arrested, but menorrhagia or metrorrhagia may occur. In men persistent priapism has sometimes been observed. The ophthalmoscopic appearances of the fundus of the eye have already been described; if the region of the yellow spot is involved vision will be affected. Similarly deafness has sometimes resulted from hæmorrhage into the structures of the ear.

The *nervous system* is not often much affected beyond the production of languor, weakness, and perhaps mental depression. Delirium and coma are occasional terminal events, and more rarely apoplexy, paralysis of the facial or other cranial nerve, or sudden deafness, results from the hæmorrhage or leucocytic accumulations already described.

DURATION.—Cases of spleno-medullary leucocythæmia have a duration of from about six months to seven years, the average of sixty-three cases collected by Gowers being two years; but the difficulty of knowing the date of the actual commencement must be remembered.

CAUSE OF DEATH.—The immediate causes of death in leucocythæmia are loss of blood from the nose or bowels, pneumonia, pleurisy, œdema of the lungs, cerebral hæmorrhage, cardiac dilatation and exhaustion.

PATHOLOGY.—The pathology of leucocythæmia is still obscure. It is certain that it is not primarily and solely a disease of the spleen. There are good reasons for believing (Muir) that the myelocytes give origin by division to the polymorpho-nuclear

granular leucocytes; and the unusual occurrence of the former in the blood, and the increase of the latter, point to an undue proliferation and growth of the marrow-cells, that is, to a disease of the marrow, as the primary cause of spleno-medullary leucocythæmia. The difficulty in this case is undoubtedly to account for the enormous size of the spleen. It may be due to the accumulation of leucocytes in great numbers, partly for the purpose of their destruction in the spleen; and to consequent tissue-change of a hypertrophic kind, such as thickening of trabeculæ and vascular development. But the relations between the size of the spleen and the amount of leucocythæmia, especially in the early stages, have to be explained. It may be that, where the spleen is enlarged with a slight degree of leucæmia, the spleen is accommodating most of the leucocytes that are poured into the blood; and that, as the spleen becomes more and more overloaded, the corpuscles must accumulate in the blood-stream.

There is absolutely nothing in the ætiological factors which helps to explain the lesion of the bone-marrow. The relations of ague to the spleen might be such that the spleen after ague was less active as a destroyer of leucocytes; but that would not account for the primary activity of the cells of the bone-marrow.

The relation of the disease to *microbes* is of course of the greatest interest, but remains unsettled. Fernie, Kelsch, and Vaillard found short rods, and others have found streptococci, and staphylococci. Löwit more recently has discovered in the corpuscles organisms of the nature of a sporozoon, to which he has given the name of *Hæmaphysa leucæmiæ magna*. He always finds it in the peripheral vessels in the medullary form of leucæmia, while in the lymphatic form he finds another parasite of a similar character. These observations appear to want confirmation, and strong doubts have been thrown upon them by Hirschfeld, Tobias, and Bloch.

DIAGNOSIS.—The diagnosis of spleno-medullary leucocythæmia rests on the enlargement of the spleen, and on excess in the blood of granular leucocytes and myelocytes. In all cases of splenic enlargement the blood must be carefully examined (p. 149), otherwise cases of *splenic anæmia* may be mistaken for leucocythæmia. If the leucocytes are in considerable excess, a fact which may be ascertained by simply examining a drop of blood under a high power, splenic anæmia is highly improbable; but the true test of spleno-medullary leucocythæmia is not so much the number of leucocytes as the existence of *myelocytes*, and the increased quantity of polymorpho-nuclear and eosinophile cells. Myelocytes may be present in early stages when the number of leucocytes is no greater than in an ordinary leucocytosis (p. 156). The special character of the leucocytes assists in the diagnosis of this form of the disease from the lymphatic variety, in which the lymphocytes form nearly the whole of the leucocytic increase.

It must be definitely made out that the tumour is the spleen. It is remarkable that an enlarged spleen in women has often been mistaken for an *ovarian tumour*. An ovarian tumour large enough to reach the costal margin is central in position, while the history will generally show that it began at the lower part of the abdomen. It is also important to bear in mind that leucocythæmia does

not at all stages produce marked pallor of the face; the patient may have a good colour when the spleen is large, and leucocyt hæmia pronounced.

PROGNOSIS.—The prognosis of this disease is bad. Improvement not infrequently takes place under treatment; the spleen may diminish remarkably in size, and the leucocytes may fall in number nearly to the normal. Similar changes even take place spontaneously, but they are generally temporary, and in every case a relapse follows and eventually death ensues. It is difficult in an early stage to estimate the future duration of the illness. Neither age, sex, nor apparent causation affords any help. The size of the spleen affords little information; but some idea may be gained by a consideration of the extent to which the leucocytes are increased, and the red corpuscles are diminished; the greater deviation from the normal being the more serious. Hæmorrhages are of grave augury, but epistaxis less than other forms. Cardiac dilatation, œdema, and albuminuria indicate an advanced condition.

TREATMENT.—Many drugs have been tried in this disease, but arsenic is the only one which has proved serviceable in any large number of cases. Under its use in some cases, the spleen has diminished in size, and the number of the leucocytes has fallen to nearly normal; but the benefit is rarely more than temporary. The drug may be given in increasing doses, up to 30, 60, or even 90 minims of the liquor arsenicalis daily. Iron, quinine, iodides, bromides, and mercury are practically useless. Mosler advised injection of Fowler's solution into the spleen; provided that the organ be firm, dense, and close to the abdominal wall, that there be no hæmorrhage manifested, and no high degree of cachexia. Temporary improvement has also been obtained by the inhalation of oxygen to the extent of 30 or more litres daily (Kimberger); and under this treatment combined with the administration of arsenic, remarkable improvement took place in a case under the writer's care; but it has not always succeeded. Excision of the spleen has been attempted, and has been invariably fatal—in most cases from uncontrollable hæmorrhage. Further trial of it does not, at present, seem justifiable; and if it is not the primary seat of the disease there is little reason for the operation. Similarly, though quinine, cold affusion, ergotin, and voltaic electricity may be employed to reduce the size of the spleen, whether it be a post-malarial condition, or a part of anæmia, or leucæmia, the course of the disease is little likely to be affected thereby.

In all cases one may make the attempt to postpone the inevitable end by reasonable care of the patient, by avoidance of physical and mental strain, by judicious diet, by maintaining a normal action of the bowels, and by protection from cold. Special symptoms may require treatment. Hæmorrhage must be checked by the usual methods, and solid perchloride of iron may be applied to accessible places. For splenic pain, counter-irritation, sedative liniments, and hypodermic injection of morphia may be used. For œdema or other indications of cardiac weakness, digitalis or liquor strychninae hydrochloratis may be employed.

II. Lymphatic Leucocyt hæmia.—The *ætiology* of this form of the disease is even less obvious than that of the spleno-medullary form. The cases are more rare, and present so many differences among themselves that classification is difficult.

Recently attention has been drawn (Fraenkel) to a group of very acute cases which appear to present distinctive features. As antecedent conditions he mentions anæmia, pregnancy, injury, and certain infectious diseases, especially influenza. The ages of his patients were from 13 to 34; but one of five cases recorded by Rose Bradford was 58 years old and another 7 years.

ANATOMICAL CHARACTERS.—The blood in the lymphatic forms is characterised by the predominance of those so-called uninucleated cells, with a non-granular protoplasm, which have gone by the name of lymphocytes, and have hence secured for this condition the name of lymphæmia, or lymphocyt hæmia. Ehrlich distinguished two kinds of these cells: one a small cell, not larger than a red corpuscle, with a large nucleus and a very small amount of protoplasm (lymphocyte); the other two or three times as large as a red cell, with a single nucleus and a large amount of surrounding protoplasm (large uninuclear). In several of the acute cases, it is the latter cells which have formed the bulk of the leucocytes. Hayem and Lion place no reliance upon the size of these corpuscles as a means of distinction, but divide them into those which are of small size, opaque, and staining deeply with reagents; and those which are translucent, stain feebly, and vary from the size of a red corpuscle to two or three times larger. The former only are the true lymphocytes; and, as they record cases of leucæmia in which the latter are predominant, the justice of the term 'lymphocyt hæmia' is by them held to be doubtful. In any case we have here cells which have an undivided round or oval nucleus, and do not contain granules. The diseased blood further contains usually some polymorpho-nuclear cells (2 to 5 per cent. of the total leucocytes), a very few eosinophiles, and occasionally a few myelocytes. The red corpuscles are deficient, falling often to 3,000,000, or less; nucleated red cells are rare. *Post mortem*, the blood may be of a brown, café-au-lait colour. The glands over the whole of the body may be enlarged, and present conditions not essentially different from those described under Lymphadenoma. The retroperitoneal, mesenteric, and intrathoracic glands are affected as much as those readily accessible during life. The glands are generally separate from one another, but sometimes matted together. They are rather soft, and whitish or pink in colour on section; they may present hæmorrhage in their substance. The spleen is moderately enlarged, but rarely reaches the size that is common in the spleno-medullary form; the liver is also enlarged. The medulla of the bones presents often the lymphoid transformation already described. The thymus is occasionally enlarged, and found persistent even in adults. The tonsils, the lymphoid follicles of the pharynx, and the solitary glands of the stomach and intestine are enlarged; or a more diffuse infiltration may occur as in the gums, which may ulcerate—so-called 'leucocyt hæmic stomatitis.' In addition there may be new-growths of lymphadenoid structure in various parts of the body (*see* LYMPHADENOMA), and, microscopically, all the infiltrated organs contain leucocytes of the uninucleated form; it is especially to be noted that in the affected bone-marrow these lymphoid corpuscles predominate.

SYMPTOMS.—The chronic form of the disease presents a general resemblance, so far as the

symptoms are concerned, to spleno-medullary leucocythæmia. The symptoms arise somewhat insidiously: the lymphatic glands enlarge in different parts of the body, the spleen and the liver undergo moderate enlargement, the patient becomes anæmic, and suffers from attacks of fever; and in course of time occur complications of hæmorrhage, albuminuria, passive effusions, and cardiac failure, similar to those seen in the other variety.

In the acute cases, to which reference has already been made, the duration is from 20 days to 8 or 9 weeks, or rarely as long as 16 weeks. The disease begins insidiously with general weakness and malaise, or it may be with some pains in the spleen and joints. The external lymphatic glands may then become enlarged, but they are not always very prominent. There is a slight or moderate enlargement of the spleen and of the liver, and the bones are sometimes tender. A prominent feature in many of the cases has been a severe form of stomatitis with sloughing and gangrene of the gums, but (in Rose Bradford's cases at least) without any implication of the subjacent periosteum or bone. With all this there is febrile reaction, a high degree of anæmia, and hæmorrhages from the gums, under the skin, and from the alimentary canal in the form of mælena.

PATHOLOGY.—The pathology is still under discussion. The association of enlargement of the lymphatic glands and of the other blood-forming organs with the new-growth of adenoid tissue throughout the body and with the condition of the blood suggests a process analogous to that of new-growth in all the lymphatic structures, in contrast with the spleno-medullary form in which the bone-marrow is thought to be primarily at fault. But this view has to be harmonised with our knowledge of Hodgkin's disease or lymphadenoma, in which there is growth of all the lymphatic structures without leuchæmia. Conversely a case is reported (Pappenheim) of lymphatic leuchæmia, or at any rate, of lymphocythæmia, without any swelling of the lymph-glands; here the bone-marrow was affected, and Pappenheim is led to the suggestion that even in the lymphatic forms the bone-marrow may be the primary seat of the disease.

Löwit's discovery of another organism, *Hæmaphysa leuchæmie vivax*, in the blood-forming organs has not yet been confirmed.

Fraenkel thought from the inflammation and sloughing of the gums that infection probably took place through the mouth, but in Rose Bradford's cases the gums appeared to be affected late in the illness.

DIAGNOSIS.—As in the other form the diagnosis can always be determined by a careful examination of the blood with an enumeration of the corpuscles. The blood should be examined in all cases presenting numerous glandular enlargements, and in acute cases, otherwise unexplained, beginning with stomatitis, gingivitis, and cutaneous hæmorrhages.

PROGNOSIS.—This is uniformly bad.

TREATMENT.—This can only be conducted on the same lines as above mentioned for the spleno-medullary form.

FREDERICK TAYLOR.

LEUCOCYTOSIS (λευκός, white; and κύτος, a cell).—A condition of the blood, in which certain of the white corpuscles are moderately increased in number. See BLOOD, Morbid Conditions of.

LEUCODERMIA (λευκός, white; and δέρμα, the skin).—White or achromatous integument. See PIGMENTARY DISEASES OF THE SKIN; and LEUCOPATHIA.

LEUCOMA (λευκός, white).—A white opacity of the cornea, generally the result of inflammation or ulceration of that structure. See EYE, AND ITS APPENDAGES.

LEUCOMAINES (λευκάμα, whiteness).—A term applied to a class of alkaloids resulting from the normal metabolism of the animal proteid tissues without the intervention of any bacterial agency. To the class of alkaloids resulting from the putrefaction of tissue the term *ptomaines* is given. See PTOMAINES.

LEUCOPATHIA (λευκός, white; and πάθος, a disease).—SYNON.: Albinism, Achroma, Leucoderma, Leucasmus. See ALBINISM; and PIGMENTARY SKIN-DISEASES.

LEUCOPLAKIA.—See TONGUE, Diseases of.

LEUCORRHŒA (λευκός, white; and ῥέω, I flow).—SYNON.: Fr. *Leucorrhée*; Ger. *Weisser Fluss*; Lat. *Fluor Albus*; Pop. 'The Whites'; 'White Discharge.'

DEFINITION.—A non-hæmorrhagic discharge, of pale colour, escaping from the female genital fissure.

ÆTIOLOGY.—Leucorrhœa is a symptom rather than a distinct disease; and may result from any of the morbid processes that lead to hypersecretion from the genital mucous surfaces, or from the glands opening upon them, whether the mucous membranes be injured or entire. It is, however, a source of much discomfort and deterioration of health, and so demands special treatment.

SYMPTOMS.—Leucorrhœa presents several distinct varieties according to the seat of its cause; and the symptoms of each variety require separate consideration.

1. **Vulvar Leucorrhœa.**—In this variety a glairy viscid secretion is found bathing the apposed surfaces of the pudenda, stiffening into a crust on the surface of the labia majora or on the insides of the thighs, and sometimes glueing the lips more or less firmly together at their margins. It is usually derived from the muciparous glands covering the internal surfaces of the labia majora and the nymphæ; but in cases of special eruptions and general vulvitis it may come from the vestibular surface; and in still rarer cases it is poured out from the glands of Bartholin. Vulvar leucorrhœa is met with at any period of life, but is most common in the young, *infantile leucorrhœa* almost always being of this variety. In cases of gonorrhœal infection in the female, the vulva is usually the seat of a profuse discharge which is apt to become purulent, but it is rarely confined to this situation, spreading both into the urethra, and upwards into the higher spheres of the genital mucosa.

2. **Vaginal Leucorrhœa.**—The discharge in cases of vaginal leucorrhœa is most frequently white in appearance, of acid reaction, and due to a secretion from the general surface of the vaginal mucous membrane. Its whiteness is found, on microscopical examination, to be owing to the presence of quantities of scaly epithelial cells, many of which are crowded with fatty particles, while others have been quite broken up in consequence of the fatty degene-

ration. Sometimes the discharge has a more yellowish tint, and then it is found to contain quantities of pus-cells among the epithelial scales. In the former group of cases we have to do with a simple catarrhal condition of the vaginal mucosa; in the latter there are red granulation-like spots scattered over the membrane, which has here lost its epithelial covering. Vaginal leucorrhœa is a complaint to which women are specially liable during the reproductive period. The catarrhal form is extremely common in young married females; while the other form occurs rather about the menopause, or, if occurring earlier, is complicated with some of the other varieties of leucorrhœa. Apart from specific causes, it may be brought on by sexual excesses; by the presence of a foreign body, such as a pessary; by a displaced uterus; by a chill; or by any condition that interferes with the circulation in the pelvis. In a large proportion of cases it is secondary to the next variety of leucorrhœa.

3. **Cervical Leucorrhœa.**—The discharge that comes from the canal of the cervix uteri is transparent, like unboiled white of egg, very tenacious, and of alkaline reaction. It may still present these characters as it escapes from the pudenda; but it generally becomes somewhat clouded as it passes through the vaginal canal, and gets acted upon by the acid secretion from the vaginal walls. Independently of this change in the vagina, it is sometimes found already more or less opaque as it lies within the cervical canal, and may be seen of a yellowish or greenish or reddish tint in various cases. The clear cervical leucorrhœa is seen under the microscope to be made up of a viscid magma, having entangled in it large numbers of columnar epithelial cells, which have a tendency to arrange themselves in rows. These are easily seen to be the ciliated epithelial cells that cover the normal mucous membrane, but deprived for the most part of their cilia. They are accompanied by smaller rounded cells like mucous corpuscles or wandering cells, partly derived from the interior of the crypts, and partly shed from the general surface from which the epithelium has been removed. In almost all cases some of the epithelial cells and mucous corpuscles are charged with fatty particles, and surrounded with granules, resulting from the breaking down of some of their number. The more turbid fluid, the more the cells are found to have undergone such degeneration; and where the discharge is profuse, fluid, and of yellowish colour, it has more the characters of a purulent fluid in which the relatively few cylindrical cells are changed in form, becoming oval or rounded, and nearly all reduced to a compound granular mass. The more deeply tinted discharges owe their discolouration to the admixture of blood, the red corpuscles of which can easily be recognised. Apart from the leucorrhœas of specific origin, this is the commonest of all the varieties. It may be found in females of any age, but specially affects women during the reproductive period, and more especially those who have been mothers. We can understand the special liability of the cervix to catarrhal affections, when we remember that all intra-uterine discharges pass through and may irritate it; that it is exposed to damage during the transit of the fœtus in parturition; that vaginal affections easily pass into it by continuity of structure; and that it may readily be injured by foreign bodies in the vaginal canal, or even by fretting of its orifice against the vaginal wall in

cases of displacement or excessive mobility. It is, besides, a favourite site for the lodgement of gonococci in cases of 'latent gonorrhœa.'

4. **Intra-uterine Leucorrhœa.**—Here also the discharge is transparent, like white of egg, and alkaline in its reaction, but it is more fluid than the secretion from the cervical canal, and may escape as a clear liquid from the genital fissure. In cases of long standing, more particularly where there exists some organic disease in the uterine parietes, the fluid becomes turbid, purulent, and more frequently than in any other variety of leucorrhœa tinged with blood, even alternating with irregular discharges of blood. Under the microscope we see many cylindrical epithelial cells, not infrequently ciliated, along with groups of smaller cells, partly cylindrical, partly rounded, that have been discharged from the uterine follicles, all embedded in a mucous fluid. Where the discharge is more turbid, the epithelial cells are seen to be undergoing fatty degeneration, and to be accompanied by wandering cells, pus-globules, and crowds of free fatty particles. This uterine leucorrhœa may be found at any period of life, but as an independent affection it is found almost exclusively in virgins or young married women, or in women who are ceasing, or have ceased, to menstruate. In the last-named class of cases the cervix is often atrophied, and its orifices narrowed; and the intra-uterine secretion may accumulate for a time, and be expelled with some degree of suffering, and having a fœtor that may raise a suspicion of cancer. Most frequently it is found associated with cervical leucorrhœa, the endo-cervical affection having passed up to the endometrium, or, more rarely, *vice versa*. Perhaps the most frequent form of it is found in women who are subject to a leucorrhœal discharge before or after the menstrual periods; and in the cases of amenorrhœa where a pale discharge escapes at the usual menstrual periods, this has its source in the interior of the uterus proper.

5. **Tubal Leucorrhœa.**—Doubtless some small portion of the fluid that escapes in certain cases of leucorrhœa is furnished by the Fallopian tubes; but despite the elaborate attempts of Hennig and others to establish a distinction between it and the other varieties, it remains rather as a subject of pathological interest than of clinical importance, and need not occupy us further here.

DIAGNOSIS.—The statements of a patient in regard to a leucorrhœal discharge cannot be relied on in establishing a diagnosis as to its source. If it be white and flaky we may judge that it is vaginal; if more transparent, and escaping in half-coagulated flocculi, we may conclude that it is cervical; while a clear and more continuous and fluid discharge would be more justly referred to the uterus proper. But it is never safe to trust merely to the appearance of the discharge as it escapes from the vulva, for it may have become modified as it lay in or traversed some part of the canal, or may be compounded of fluids derived from different surfaces. The seat of the discharge must therefore be exposed. In the vulvar variety it suffices to separate the labia and occasionally to expose the navicular fossa and the orifices of the Bartholinian ducts, by passing the finger into the anus. The vaginal form of leucorrhœa requires for its detection the use of a speculum, duck-bill or tubular; and the cervical, one of these or a bivalve speculum. For the diagnosis of intra-uterine leucorrhœa it is sometimes helpful to remove

some of the fluid for microscopical examination by means of a fine syringe. Unless a clear history of infection can be obtained, or the presence of gonococci verified by the microscope and culture, it is almost impossible to establish a distinction between a gonorrhœal discharge and the simpler catarrhal leucorrhœa. In the former there is a very notable tendency to spread through all the contiguous mucous surfaces; and while the vulva is likely to be predominantly affected in acute attacks, in chronic cases the germs lodge more frequently in the cervix or in the Fallopian tubes.

TREATMENT.—In instituting our treatment of leucorrhœa it is of the first importance to have in view the *constitutional* condition of the patient; to use means to counteract any diathetic tendency—tubercular, strumous, or syphilitic; and to raise as far as possible the general standard of the patient's health, by the administration of tonics and the enforcement of a suitable diet and regimen. It is partly in this way that a change of residence is often useful; and in making a change, it is well for the patient to go to some of the spas, such as Ems or Kissingen, the waters of which are helpful in reducing congestions and catarrhs of the pelvic viscera. In young women of relaxed habit of body, it may be enough to prescribe quinine and iron or arsenic, and the daily use of a cold sponge bath; and in infantile leucorrhœa, cod-liver oil and iron should be administered.

In the great majority of cases of leucorrhœa some kind of *local* treatment becomes an absolute necessity. Sometimes it is enough to pay strict attention to cleanliness, washing the pudendal surfaces with a soft sponge, or syringing the vaginal canal with tepid water; and even when astringent applications are to be made, the surfaces should first be subjected to a detergent stream of water. Where there is marked congestion of the uterus it is best to make the injections with hot water, and to keep the stream passing through the vagina for at least five minutes at a time; the immediate relaxation of the blood-vessels and hyperemia being followed by contraction of their walls, which favours the cessation of the discharge. The astringents most serviceable for checking *vulvar* and *vaginal* leucorrhœas are alum, aluminated iron, acetate of lead, sulphate of copper, sulphate of zinc, borax, and infusions of oak-bark, matico, and other vegetables charged with tannin. They are best applied in the form of an injection with a Higginson's syringe, having a vaginal nozzle attached to it; or of a douche through a long india-rubber tube, with a stop-cock, for regulating the flow, fitted close to a glass vaginal nozzle, and the other extremity opening into a wide receptacle, or fitted to a filter or pitcher into which the fluid is poured. Where there is a difficulty in using the injection, and where it is desirable to keep up a more prolonged application of the medicament, it may be introduced into the vagina in the form of pessaries made with cacao-butter or with gelatine. Topical applications to the canal of the cervix and cavity of the uterus ought always to be made through the speculum, and without such applications it is a hopeless task to undertake the cure of *cervical* leucorrhœa. Here, more concentrated or more powerful astringents or escharotics become necessary. Nitrate of silver in the form of a stick of caustic is easily applied, but its repeated application will lead to mischief. Zinc-alum, dried sulphate of zinc, sulphate of copper, perchloride of iron, or

tannin may be introduced, in the form of rods or arrows made with starch and gum. If a uterine sound or stilette be dipped in water and a thin film of cotton wadding wrapped round the point to the length of about two inches, the adherent mucus can be cleared away, and the same or another sound mounted with wadding can be charged with fuming nitric acid, the acid nitrate of mercury, strong carbolic acid, a solution of perchloride of iron, tincture of iodine, or iodised phenol, and carried through the speculum along the cervical canal. In *intra-uterine* leucorrhœa it becomes necessary to carry the application right up in the same way to the interior of the uterus. It is usually best to begin with one of the stronger liquids, apply it a few days after a menstrual period, and follow it up with applications of iodine. So long as the stilette or sound with the dry wadding passes easily through the os internum, it is usually necessary to continue from time to time the intra-uterine application.

ALEXANDER RUSSELL SIMPSON.

LEUKERBAD (LOËCHE), in Switzerland.—Thermal earthy waters. See MINERAL WATERS.

LEVICO, in the Trientino, Austria.—Arsenical sulphate-of-iron waters. See MINERAL WATERS.

LEYDEN'S CRYSTALS.—See CHARCOT-LEYDEN'S CRYSTALS.

LICE, Diseases due to.—See PEDICULUS.

LICHEN (λεῖχην—secondary signification—an eruption).—This term was used by the old writers to signify a large group of diseases usually distinguished by the appearance of a papular eruption. A more correct knowledge of their ætiology has, however, resulted in the reclassification of many of these diseases. Thus, Lichen urticatus is now relegated to the true urticarial group; while Lichen simplex and Lichen agrius are known to be stages in the development of eczematous and other diseases. There remains only the disease now usually termed Lichen planus. Two or three other conditions in common parlance still retain the name, and will be referred to later.

Lichen planus (SYNON.: *Lichen ruber*) is a disease characterised by an eruption of small papules, caused by the presence of infiltrating cells of inflammatory character in the upper layers of the cutis, with certain changes in the epidermis and neighbouring structures. The papules develop slowly, and the infiltrating cells show no tendency to rapid degeneration or to supuration. In consequence, the disease is usually of long duration, and is limited in distribution. Occasionally its course is acute, and the eruption universal. The papules when first seen are minute, slightly raised, sharply defined, and separate one from the other. As they increase in size (up to about 5 millimetres in diameter) their angular outline, just noticeable in the earlier stage, becomes more marked; thus, in the fully developed discrete papule, the shape is distinctly polygonal. The top of the papule as a rule becomes flat, and frequently shows a smooth polished surface, with a small depression corresponding to a hair-follicle or sweat-duct. When this is the case the mouth of the hair-follicle becomes filled with a plug of epithelium, which often projects as a conical peg from the surface of the papule. By this means the

papule of the disease occasionally has an acuminate shape, and in certain stages and certain cases the acuminate variety of papule is common. In course of time the epithelial plug in the follicle falls out, and a central depression is left, which is a very characteristic feature of the plane papule. These acuminate papules serve to emphasise the close relationship between lichen planus and the rare disease known as *Pityriasis rubra pilaris* (Devergie) in this country, and as *Lichen ruber acuminatus* by German observers.

The colour may be scarcely different from the surrounding skin, but usually the papules are a characteristic tint of purple or red, which varies with the acuteness of the process and the natural complexion of the patient. The eruption may appear on any part of the body, but is often especially marked on the flexor surfaces of the forearm and wrist, in the neighbourhood of the knee, and on the shin. Early and slight manifestations of the disease may be specially easily identified where the skin is fine and free of hair. The cutaneous papules tend to become capped with a fine scale of desquamating epithelium, and this feature of scalliness becomes more evident as the disease becomes chronic, and as the papules coalesce to form patches.

The eruption is frequently seen on the buccal mucous membrane, producing greyish-white points and streaks on the inner surface of the cheeks and lips, and leucoplakia of the tongue.

The subjective symptoms vary widely. The discomfort, if any, is generally slight; but sometimes the sensations of burning, itching, and tingling may be so severe as to threaten the life or mental condition of the sufferer.

The disease is rare before the age of twenty. Chronic lesions may be found in extreme old age. It seems to affect both sexes fairly equally. It is supposed to show a special preference for persons of the educated and leisured classes, and for those with neurotic tendencies.

The cause of lichen planus is uncertain. Some regard it as of toxæmic, others of nervous origin.

Three varieties may be recognised:—

1. *Acute lichen planus*.—The disease occasionally occurs as an acute eruption, appearing rapidly over the whole surface of the body. Along with the eruption of the characteristic papules there is a considerable amount of general erythema. The papules may coalesce to form small rounded patches or rings, but do not give rise to sheets or thickened patches of disease. The erythema soon disappears and the papules flatten out and are absorbed, leaving slightly pigmented spots, which in their turn also vanish. The whole course of such an attack may last for three or four months; local patches of the eruption, especially on the extremities, remaining for a little longer. This type of the disease appears to occur most commonly in young persons, and, in spite of the rapidity of appearance and large amount of the eruption, the discomfort may be very slight. The pruritus and irritation of the skin depend greatly on the general health and nervous condition of the patient.

2. *Chronic lichen planus*.—The majority of the cases run a more chronic course. Certain areas of the body are specially liable to become affected, such as the forearms, the thighs, the legs, the back, and abdomen, but usually, in addition, a number of papules are disseminated over the body,

though these may be so insignificant as readily to escape notice. In the areas specially selected by the eruption in any individual case the lesions tend to coalesce, and thus to give rise to patches of eruption sometimes reaching several inches in diameter. In such cases the purple or livid areas of the disease, slightly raised above the surface, traversed by the natural lines of 'skin-cleavage' exaggerated by the eruption, and covered by numerous fine greyish-white scales, are very characteristic. In other cases the eruption appears in ringed or circinate figures, while in others a distinctly linear distribution is observed. The lines of eruption are frequently the lines of pressure of garments, &c., or of injury to the skin by scratching.

3. *Lichen planus verrucosus*.—When the disease is markedly localised the coalescence of the chronic papules, the thickening of the cutis, and the increase of the horny epithelium give rise to the variety of the disease known as *hypertrophic lichen planus* or *lichen planus verrucosus*. This form is usually found on the extremities, especially on the legs. The peculiarly tinted dry, thickened, and scaly patches of eruption are easily recognisable. There may be observed in nearly every case outlying individual papules of the eruption presenting the characteristic flat-topped appearance; these serve to distinguish the disease in case of doubt.

The disease occasionally becomes widely *diffused*, and is then difficult to distinguish without critical examination from severe cases of general psoriasis, and shares with this disease the unfortunate liability to degenerate into one of the secondary forms of general exfoliative dermatitis. The disappearance of the eruption is followed by pigmentation, sometimes so dense as to give rise to a slate-brown or sepiæ discolouration of the affected part, even in cases in which no arsenic has been administered. The pigmentation gradually disappears, but may remain for years after the lesions have vanished; this is one of the most characteristic features of the disease. It is not very uncommon to find atrophic points and patches as the sequel of an eruption of lichen planus.

A form of disease probably allied to the diffuse form of lichen planus, showing areas of erythema differing in intensity with papular lesions, has been described under the title of *Lichen variegatus* (*Parakeratosis variegata*, Unna), and is under investigation at the present time.

HISTOLOGY.—The most noticeable lesion occurs in the cutis, and consists of dense collections of mesoblastic cells occupying chiefly the upper papillary layers. These cells are about the size of leucocytes, and consist of a single nucleus, with a small surrounding border of protoplasm. They degenerate and disappear slowly, and may possibly be converted into ordinary connective-tissue cells. Along with these cells are others resembling leucocytes, showing a tendency to fragmentation of nuclei and rapid disintegration of the cell. The cellular infiltration is densest in a group of papillæ immediately underlying the centre of the papule, and extends in gradually diminishing lines and streaks along the cutaneous vessels into the surrounding cutis. The lymph-spaces are widened, and there is a certain amount of dilatation of the blood-vessels with consequent œdema. The changes in the epithelium are not so marked, and consist chiefly of a tendency to overgrowth of the cells of the rete mucosum. The stratum granulosum is as

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well defined or even thicker than normal, and in the chronic varieties the horny layers are also thicker, producing the hyperkeratosis so characteristic in lichen planus verrucosus. As a result of the thickening of the cutis, and the formation of a firm overlying horny layer of epithelium, the surface of the papule becomes flat, and develops the polished surface of the typical papules of lichen planus.

PROGNOSIS.—The prognosis of cases of lichen planus is nearly always good. In the acute cases recovery is often rapid, and in the course of a few weeks pigmentation only is left in the positions of lesions. In the chronic and localised variety on the other hand, especially of the verrucose type, the duration of circumscribed patches is indefinite.

TREATMENT.—*Local.*—The first indication is to allay local irritation. For this purpose anti-pruritic remedies, such as carbolic acid, resorcin, &c., as local applications are useful. The combination of mercury with anti-pruritics of this class is often advisable, as this drug appears to promote the absorption of the lesions. In acute forms of the disease the use of lotions of the glycerine of the subacetate of lead, 5 per cent. to 10 per cent., with 1 per cent. to 2 per cent. of added carbolic acid, are useful in the first stage of treatment. Later, when the disease becomes localised, the use of an ointment or paste containing the remedies indicated, either as an inunction or as a covering to the patches of disease, is of more value. For this purpose the following ointment may be used: Carbolic acid, 4 parts; perchloride of mercury, $\frac{3}{4}$ –1 part; zinc ointment, to 100 parts. If much scaliness is present the use of salicylic acid or resorcin is to be recommended. Other remedies of service are the liquor picis carbonis, oil of cade, oleum rusci, the salts of mercury such as ammoniated mercury, and sulphur. When the condition of lichen verrucosus is attained, more severe local measures must be used to remove the horny epithelium. For this purpose thorough washing of the patches with a lotion of soft soap and alcohol, or the application of strong ointments or plasters of salicylic acid, should be employed. After the removal of the epithelium, milder treatment by means of the remedies already mentioned may be adopted. In the most chronic and warty patches much benefit is obtained by light curetting or scarification, or even by gentle cauterisation of the patches.

Internal treatment is often necessary, and should in nearly all cases be of tonic character. Change of air and relief from wearing occupations are to be advised. Such persons benefit from the judicious use of specially prepared foods, digestive tonics, cod-liver oil, and iron. Phenazone and other allied remedies are of value in the treatment of patients suffering from nervous excitement and irritability of the skin. Two drugs have been much used on account of their supposed specific effect on the disease, namely, arsenic and mercury. Of these, arsenic is of little value. On the other hand, the judicious use of mercury in the form of small and frequent doses of the perchloride seems to have a distinct effect, both in the cure of acute cases, and in aiding the resolution of the chronic lesions.

Lichen Circumscriptus.—A name applied to a circinate and papular eruption occurring on the trunk. It is one of the forms assumed by seborrhoea corporis or dermatitis seborrhoica.

Lichen Pilaris.—The name applied to a papular eruption affecting the hair-follicles, especially

in children, the lesions of which are frequently grouped and surmounted by horny spines of considerable length. The term *L. spinulosus* is also applied to this condition. It is a rare disease, and should not be confused with the slightly inflammatory hyperkeratosis of hair-follicles common on the external surfaces of the arms and thighs, especially in those suffering from slight xeroderma, to which the name has occasionally been given, and which is also known as keratosis pilaris.

Lichen Urticatus.—A common eruption in childhood, and probably the result of urticaria.

JAMES GALLOWAY.

Lichen Scrofulosus.—**SYNON.** : Lichen Scrofulosorum.—The name lichen scrofulosus has been given to an eruption of somewhat infrequent occurrence, characterised by the evolution of numerous indolent miliary papules, due to a folliculitis of little intensity, disseminated or usually with a strong tendency to group in clusters, which may be arranged in crescents or circles. The eruption may be copious or scanty, and then easily escapes recognition. The favoured sites are the flanks and sides of the abdomen, but other parts of the trunk, and even the limbs, may be involved. The colour of the papules is reddish from the inflammatory congestion, but when the latter subsides a tawny hue results, or some temporary pigmentation or slight atrophy is left. Well-formed papules are more or less rounded or conical, but in involution become flat-topped. It should be noted also that, like other follicular papules, tiny spines may project; that occasionally the composite character of the patches may be obscured by a degree of scaling; and that some acneiform papules may be intermixed with the simple papules. The eruption is somewhat capricious, appearing suddenly, and sometimes disappearing rapidly when the patient is placed under favourable conditions. It is an affection for the most part of childhood.

The association of this eruption with tubercular lesions of various kinds is very striking. Histological research discloses an architecture which is extremely suggestive of tuberculosis, and the presence of tubercle-bacilli has been noted. A local reaction has been observed after tuberculin-injections, and there is some evidence that inoculation of the papules into animals may produce tuberculosis. Such evidence has rather displaced the old theory that the eruption was the outcome of some cachexia, which was suggested by the multitude of papules, the benignity of the eruption, the absence of any local infective progression, and the capricious behaviour of the eruption. There is still some difference of opinion as to whether the eruption is due to the presence *in loco* of tubercle-bacilli of feeble infective power or to the circulation of toxins in the blood from some tuberculous focus.

DIAGNOSIS.—This eruption has to be distinguished from the other minute papular eruptions developed in connection with the follicles, but the combination of symptoms set forth will generally serve to make the distinction. Three eruptions may be specially mentioned: (1) a rare affection of childhood—the Lichen spinulosus of Devergie—in which a few symmetrical clusters of follicular papules are localised on various points of predilection, such as the hips, shoulders, and back of the neck; (2) the disseminated follicular papules of 'eczema seborrhoicum'; and (3) the papular clus-

tered eruption of acquired syphilis, which is very rare in childhood.

TREATMENT.—This must be directed to the promotion of the general nutrition of the patient by such dietetic, hygienic, and medicinal or surgical measures as will serve to cure or remove any tuberculous foci and prevent infection. Cod-liver oil internally and as inunctions has been especially recommended.

T. COLCOTT FOX.

LIEBENSTEIN, in Saxe - Meiningen.—Chalybeate waters. See MINERAL WATERS.

LIENTERIC (λεῖος, smooth; and ἔντερος, the intestine).—A form of diarrhoea in which the stools contain much undigested food, in consequence of its having passed rapidly along the alimentary canal. See DIARRHŒA; and STOOLS.

LIFE-ASSURANCE.—The great extension of life-assurance of late years has placed the solution of important questions involving large financial operations within the range of the daily work of the medical practitioner. It is important, therefore, that his duties and the nature of the knowledge required of him should be defined. We shall dwell briefly on the nature of life-assurance, and point out the rules which should guide the medical adviser of a life-assurance office in forming an opinion. We shall then consider the principal agents which modify the duration of human life, and their bearing on the questions submitted to us. Life-assurance is a contract of indemnity. The amount of the indemnity to be paid on death is fixed by each individual according to his means or wishes, and the company engages to pay the sum when life fails or under other conditions, in consideration of an annual or other payment or premium so long as the contract lasts.

Irrespective altogether of health, the risk undertaken by the company is greater or less according to the variation in two factors: (1) the age of the life to be assured; (2) the duration of the term of assurance. The premium required for an assurance on a life aged forty is greater than that for a life aged twenty; and again the premium for an assurance over the years of life from forty to sixty is greater than that for an assurance from forty to fifty only. As the premium is calculated on the assumption that the proposer has a chance of surviving to the extreme limit of life, it is obvious that the company ought to insure at the ordinary rate of premium such lives only as have the best chance of attaining to old age, or, as they are called, *first-class lives*. Hence lives coming under any of the following classes must be discarded from the first-class standard:—

1. Lives not possessed of sufficient vital power to afford them the possibility of attaining old age. This will include persons who have shown a tendency to certain diseases which will develop later on in life, as gout, rheumatism, &c.

2. Lives whose family history discloses a tendency to early death.

3. Lives whose place of residence or occupation has a more than usual effect on life, or involves peculiar hazards.

Generally, lives which fall short of the highest standard in respect of one out of these three conditions may often be accepted, but if they fail of the highest level in more than one condition, they must be excluded. The ideal, therefore, which the medical examiner should set before himself as

constituting a first-class life, is the exclusion of these three conditions.

The tables of *expectation of life* put before medical examiners, which are deduced from the Institute of Actuaries' HM tables, and are based on the mortality of healthy males whose lives had been assured with twenty British life-offices, are often misunderstood. Expectation of life indicates the average number of years which is lived by all persons of a common age, from that age up to the extremity of life. The total number of years that will in the aggregate be lived by a group of persons of the same age, divided by the number in the group, gives the 'expectation.'

The medical examiner is for the time being the retained adviser of the office which employs him. Any medical man may decline to act for a company, but, if he has accepted a fee, he is bound to consider their interests as of primary importance, and his relations with his patient as secondary. He should not, therefore, allow himself to be swayed by motives of personal friendship, or because he is or has been the ordinary attendant of the proposer. Generally speaking, it is not judicious to employ the ordinary medical attendant of a proposer to report for an office, but it is often necessary to obtain his evidence confidentially, and, unless he decline to act, he is bound not to withhold any information he may have regarding the health, habits, or family history of the life. Every examiner should be aware that his report and opinion are necessarily submitted to the physician of the company, and thus a greater accuracy in the use of medical terms will often be observed.

The examiner will have an interview, always private, with the proposer, who should not be accompanied by any one. The wife may ask that her husband or friend shall be present, but this is not advisable. Persons of either sex are always more frank when alone with their doctor. There may indeed be ailments kept secret even from a wife or husband, or former events in life bearing on health, which would not be revealed in the presence of a third party. It is scarcely necessary to advise that the utmost courtesy and quietness of manner should be observed, and the proposer placed at his ease as much as possible. A deterrent inquisitive manner is apt to give rise to prevarications or concealment in the replies given.

An inquiry into the functions of the body is often resented when put abruptly, but the writer has never failed to obtain all necessary information by a quiet questioning. He is in favour, however, of direct questions, plainly spoken out, especially with women; but *many* questions of the kind are to be avoided.

Regarding temperance, it is generally useless to put the question directly to a proposer. His estimate of what constitutes excess may not be yours. His opinion of his own habits may be comparative as regards his neighbour, and he may be a dram-drinker (the worst form of excess) yet never 'get drunk'! It is well to approach the subject with inquiry as to his hours, habits, neighbourhood, and society, and thus elicit both his favourite drink, and how often it is taken.

The proposer being placed in a good light, the examiner will notice his general build, corpulence or otherwise. The face and hands will especially occupy him. The face may indicate intemperance, sensual habits, or nervous excitability. It should

be neither pale nor flushed, free from blotches, and the nose not unduly red. The drunkard's nose, protuberant, granulated, and rosy at the end, is often associated with a blear-eyed state of the eyelids. There are some forms of skin-affection which simulate it, but the practised eye will discriminate the chronic effects of alcohol which, by impairing the elasticity of the capillaries, has given rise to the nose here indicated. A flushed state of the mæ may lead us to suspect phthisis; while redness of the cheeks composed of permanently dilated blood-vessels, together with a velvety integument exuding sebaceous matters, always caused Brinton to suspect kidney-disease.

The hand affords evidence of various kinds, and its form, colour, and nails should always be regarded. We may thus detect gouty thickening; or, from the clubbed state of the fingers and incurved nails, be enabled to detect chronic phthisis, or old pleuritic or pericardial disease. The tongue should be examined, not only as regards its being furred, fissured, moist, or dry, but also for ulcers or growths, which, if present, should always be examined by pressure of the fingers. The throat may also give evidence of present or old syphilitic ulceration. The gums may present the blue line of lead-poisoning, or the dark-red line of phthisis. The gait, nervousness of manner, twitching of muscles, inability to walk straight, peculiarities of the eye-balls and facial muscles, or other symptoms, will betray some deep-seated affection of the nerve-centres, and the patellar reflex should be examined if any suspicions arise. Tremblings or shakiness of handwriting may indicate alcoholism, or abuse of tea or of smoking.

The teeth may be deficient or carious, and their condition is often a good test of general health. The notched teeth of syphilis should always be looked for. Finally, the whole aspect of a man should be noted, as conveying that he is robust, hearty, and vigorous, well developed in muscle of chest and limbs, or puny and weak, with loose tissues and flabby muscles.

The chief duties of the medical examiner are to ascertain with precision:—

(1) The *family history* of proposer; (2) his *past history*; (3) his *present state* of health; and (4) his *habits*.

1. The *family history* is to include not only his parents, brothers and sisters, and grandparents, but the health and ages at death both of them and their descendants collateral to proposer—that is, of uncles, aunts, and first-cousins. These facts need not all be recorded in the report paper supplied by the office, but their elucidation often enables the examiner to form an opinion as to the eligibility of a life where any taint, as consumption, gout, or cancer, exists in one generation. In case of deceased parents, brothers, and sisters, the exact causes of death should be given. If these are unknown it is important to state any facts concerning the symptoms and duration of the fatal illness which may enable an approximately accurate conclusion to be drawn.

2. The *past history* of a proposer will occupy us with much care. He is bound to tell us whether he has ever had any serious ailment; but, as memory is fallacious, the direct questions which are found in every form of medical report issued by the offices are to be put. It will be convenient then to ask in detail whether he has had symptoms of diseases

affecting the chest, head, and abdomen, or any disorder of a general kind, as fevers; and young men should be asked plainly about syphilis.

Inquiry should be made as to former residence in an unhealthy climate, or at any special place on account of defective health; also whether proposer has had any medical attendant, and, if so, for what.

3. The *present state* of health is then to be ascertained, and a careful examination made of chest and abdomen. We cannot insist too strongly on the necessity for *uncovering the chest*. Without this, percussion cannot be practised, the expansion, the movements, and alterations in the walls, caused by congenital malformation or by former disease, as pleurisy and phthisis, must escape notice. The heart-sounds cannot be estimated through the dress; and we have known many instances in which morbid conditions were overlooked because the proposer was so examined. There are heart-murmurs and lung-sounds which can be simulated or disguised by a well-starched shirt. For the examination of the *abdomen* the waistband should be unloosed, and palpation and percussion practised, by which the existence of enlargement of the liver or spleen, or of tumours, may be ascertained: for this purpose the reclining posture should be adopted.

4. By the *habits* of the proposer we understand his general mode of living, and his opportunities for exercise, but above all his temperance or excess in the use of stimulants. The average quantity of stimulants taken daily should be written down from the reply of proposer on the document which he has to sign. The testimony of friends is always asked on this point, but the replies are generally fallacious, and often consist in a 'Yes' or 'No' to the queries sent to them. In proposals important as to amount, a written statement as to the friends' estimate of the habits of a proposer should be obtained in the form of a letter, and not on a printed form.

5. *Age*.—Different ages predispose to particular diseases. Respiratory diseases prevail most from puberty to twenty-five, and this is the age when phthisis is most fatal. It has been reckoned that at forty half the danger from this disease is over, and three-fourths at fifty years of age; and the rule seems a fair one. An heredity to the development of phthisis at a somewhat advanced age—say after forty—may, however, exist, and prove an actual danger to life. The liability to certain diseases, as gout, apoplexy, degeneration of organs and blood-vessels, and also urinary affections, increases after forty, and it is at and after middle life that the temptation to excess in diet and stimulants, with a diminished desire for exercise, leads the way to slow organic alterations. Persons who attain to great age are generally spare, and have in almost all instances been frugal in their habits, and active in mind and body. The decay of muscle and of organs is precipitated by want of use, and the due exercise of all faculties of mind or body is conducive to longevity. It is good to compare the apparent with the actual age of a proposer. As a general rule, when he is really older than he looks, his expectation of life surpasses the average, and when he has aged beyond his years the risk is thereby so far increased.

6. *Occupations*.—The life which is protected from the vicissitudes of fortune by a fair provision for daily wants, which has occupation for both mind and body, without undue strain or the necessity for

hurry, and which has daily exercise in open air, combined with a moderate amount of sedentary work—is undoubtedly the best risk. The clergy are perhaps the best lives of all callings, and the statistics of the clerical offices bear out this statement. Teachers, heads of schools, lawyers, and physicians probably come next. The 'business' class, which includes merchants, stockbrokers, bankers, manufacturers, directors of companies, &c., is fairly healthy, and stands among the first on the list, but the examiner will do well to remember the anxieties incident to all these callings.

There are certain classes exposed to manifest dangers, for which special rates are demanded by all offices. Thus the publican is charged 1*l.* per cent. extra for occupation, a sum which appears to be scarcely sufficient.

7. *Heredity*.—Heredity plays an important part in the life-history of every individual, as there can be no doubt that physical, mental, and moral characteristics are often transmitted from ancestors. The whole constitution may not be so communicated; and parents may be mere transmitters along a chain, their offspring representing an earlier ancestor. The examiner will therefore inquire both about parents and grandparents. In tracing the effects of transmitted disease we should find how far others of the same generation, as brothers, sisters, and cousins, may have been affected by it. We have thus a gauge of the intensity of the transmission. Longevity, which implies the perfection of the whole animal system, is no doubt hereditary in some families. If both parents have been the subject of the same disease, the heredity is intensified in the offspring. A disposition to zymotic diseases is marked in certain families. The tendency to insanity is intensified by successive inheritance; that to syphilis is lost in the repetition of inheritance. Phthisis and cancer become intensified by inheritance, and appear earlier in each generation. Double inheritance (both parents) induces an earlier and a more rapid form of disease. Heredity appears to influence the sexes equally. *Forms* of disease run in families: in some the consumptive taint is shown in acute tuberculosis, in others the most chronic form of fibroid phthisis prevails. The *mother's* transmitting influence seems greater than the *father's*, and it is common to all hereditary diseases for the mother to transmit to sons and the father to daughters. *Family phthisis* appears at an earlier age as we descend the genealogical tree; thus, the grandfather may have it at sixty, his sons at forty, and his grandsons in early life.

Cancer is hereditary in one-third to one-seventh of the cases. It is most prevalent between forty and sixty. Encephaloid cancer is met with earlier; scirrhus cancer from forty to fifty; epithelial cancer later. The risks of hereditary cancer therefore increase with age after a certain point, while those from phthisis diminish. The rule must be to reject the issue of two cancerous parents. The heredity of *rheumatism* and *gout* is incontestable. Hereditary gout appears early, often at eighteen to twenty; acquired gout appears about forty. If both parents, or even one, have had several acute attacks of rheumatism, there is great likelihood of transmission to the child. *Diabetes* is allied to gout and rheumatism, and comes after the latter in frequency of heredity. *Heart-disease* claims the same heredity as gout and rheumatism; and while the acute form in the parent is apt to be repre-

sented in the child, it is not to be forgotten also that such parents commonly transmit a tendency to slow thickenings of the valves to their children, who may never suffer from the acute form. The many instances of valvular lesions met with in practice, where the individual has had no acute attacks, may often be thus accounted for. If one parent have had gout or rheumatism severely, the proposer being in perfect health, the heredity may be overlooked; but if both parents have had these affections the life should be declined, or a large addition made to the premium.

It has been found that *asthma* was hereditary in fourteen cases of thirty-five, and in seven it was paternal and direct. The most frequent period of development was in youth, and next in old age. If both parents were asthmatic, an addition should be made to the premium. *Albuminuria* may be regarded as only transmissible when the result of gout or alcoholism in the parent.

Intemperance must be considered as hereditary in a high degree. It runs in families, who not only inherit the nervous constitution which drunken parents transmit, but that peculiar temperament which flies to stimulants as a resource on any emergency of life. The *craving* for drink, a want which alcohol can alone supply, as it stops for a time the waste of nerve-power, is often derived from the excesses of drunken parents.

Still further, the children of drunkards inherit various neuroses, such as hysteria, epilepsy, mania, ataxy, and different forms of paralysis; and a child begotten during drunkenness early falls a victim to either phthisis, or some deep-seated disease of the nervous system. Intemperance is perhaps the most formidable enemy to the safe assurance of life, and even ranks before phthisis in its deadly effects on the system. Organic ailments are by it originated, and organic weakness crystallised into disease. The degenerations of age are anticipated and precipitated by alcohol, and the dram-drinker is sure to have a shortened life. The man who carries his drink well and is 'never drunk' is in the greatest danger. Small doses of stimulants throughout the day, ended by a somewhat larger one at night, leave the system always charged with alcohol, and the excretory organs are continuously under its influence. The most searching inquiry should therefore be made as to the quantity taken each day, and the frequency of the dose. The medical examiner must be thrown on his own tact to discover from certain well-known indications whether the proposer is a drinker. Among these are the flushed face and nose, the tremulous tongue or hand, the relaxed skin, the eye, and the manner over-rapid and nervous, or subdued and sullen. Of all classes of the intemperate the habitual dram-drinker is the worst.

Diseases of the nervous system are eminently heritable, as epilepsy, general paralysis, and mania. Of 321 epileptics, one-third had epileptic parents. The children of such become diseased early, but it may be considered that after forty the liability is exhausted.

Insanity is commonly hereditary: about one in three or four has had a parent insane. The issue of one insane parent might be accepted if the age be 35 or 40, and the proposer free from any nervous affection.

PERSONAL EXAMINATION.—The medical examiner should ascertain the condition of the chief

organs of the body, especially the heart and great vessels, the lungs, and the kidneys.

Heart.—The size of the heart and the strength or weakness of its impulse should be ascertained. *Intermission* of heart-beat and pulse, if unaccompanied by other irregularities of rhythm, may not be of grave importance. If it be only a loss of beat once in twenty or thirty, it may be regarded as harmless, or referable only to an alteration in the innervation of the heart. It may not be always present, may be indeed absent for days or weeks, or be suspended during a feverish attack, or present only during some trivial alteration of digestion. *Irregularity* of heart-action is of different import. The heart will repeat a number of rapid beats, then pause, and lose one or several successively. The sounds are confused in character and run into one another, the impulse is diminished, and the interval lost or irregular. Feebleness is the predominant character. A heaving and displaced heart-beat is a common result of hypertrophy. The danger to life arises from the consecutive events of hypertrophy, dilatation of the heart's chambers, and valvular incompetence.

Murmurs may be audible over any of the valvular regions of the heart, and accompany either the first or second sound or both. The *characters* of a murmur—its softness, flowing, harsh or rasping sound—are *per se* no indications of its value or pathological import: a harsh or a loud murmur is often of a less serious nature than one more distant and less pronounced, as the power of the ventricle behind the obstruction is generally the cause of its loudness, and a moderate degree of hypertrophy is a compensating agent. If there be obstruction at an orifice, all symptoms of heart-disorder may be held in abeyance for years if only there is power to drive the blood through the obstructed valve. *Ephemeral* murmurs often give rise to doubts as to the safety of assurance—such as occur in the left subclavian region, when firmly pressed by the stethoscope, or in certain positions of the person examined, or in that portion of heart which is covered by the lung. They may be heard on deep inspiration only, and are temporary in character. They are not to be regarded as prejudicial to life (*see* HEART, Functional Diseases of). The *anæmic* murmur, soft, systolic, and blowing, is heard over the first part of the aorta or pulmonary artery. It is occasionally heard over the mitral region. It is generally associated with curable conditions; but it is not to be forgotten that fatty degeneration of the walls of the heart has been found in cases of prolonged anæmia. The rule is to defer such cases for a month or two.

The lesions most important to consider are those of the aortic and mitral valves.

Aortic murmurs may be single or double. The single murmur is probably obstructive, the valve being thickened and warty, and the coats of the vessel diseased.

In *aortic regurgitation*, recognised by a diastolic murmur, the greatest degree of ventricular hypertrophy occurs, and the heart attains an enormous size in compensatory efforts to maintain the systemic circulation against gravity, the open valves permitting the return of a large portion of the blood into the ventricle. The carotids are seen throbbing, and the radials present the well-known character caused by sudden recession of the blood-current called the water-hammer pulse. This form of

disease is among the most fatal of heart-affections, and is entirely uninsurable, the elements of durability being wanting.

Mitral murmurs.—It is now a well-ascertained fact that persons in great number having a marked mitral murmur may live to advanced age without presenting any symptoms, and therefore without the effects of heart-disease recognised as injurious to life; and of this the best medical authorities have recorded numerous instances. The first downward step in the fatal event of mitral disease is when the cavities of the heart begin to yield to the pressure of the regurgitated blood, and dilatation follows (*see* HEART, Dilatation of). The series of events is mechanical; and it may be stated that, so long as the muscular integrity of the heart's walls is maintained, the subject of the affection may enjoy fair health with ordinary precautions. The dangers to such a life would probably arise after middle life, at the period when degenerations of structure commonly commence. The rule of assurance-companies has till lately been to reject all such lives as present any form of mitral disease; but if a case present itself with murmur only, without great enlargement of the heart; if the ventricular contraction be strong and the murmur well pronounced; and if, in addition, it can be shown that such condition must have prevailed for a long time—say, from the date of one attack of rheumatic fever years previously; if the proposer has his other organs healthy and does not exceed thirty-five years of age, then it is safe to accept such a life with a considerable addition to the premium (10 to 15 years), or by securing that all the premiums should be paid up in a short term of years—say, eight or ten.

The most dangerous heart-cases are those indicating failure of muscular power, and aortic cases are more perilous than mitral. The presence of albumen in the urine should always be looked for in connection with disease of the heart. *See* p. 670.

Pulse.—The frequency of the healthy pulse varies with the constitution of the nervous system, and the posture, sitting or standing, of the individual. Its healthy beat might average 60 to 70; but a large range should be allowed for temperament and emotional causes, the very fact of its being examined giving rise to acceleration. The physician will therefore give time for its subsidence, or make a fresh examination on another day. A sustained frequent pulse may indicate fever, phthisis, or exophthalmic goitre. The fulness or incompressibility of a pulse, and a character of wiriness under the fingers, should be noticed. The latter may indicate sclerotic changes in the vessels, when the condition of the temporal and other arteries should also be examined.

Chest.—A careful examination of the lungs should be made by palpation, percussion, and auscultation, the dress being removed, and measurements taken by a tape at the level of the sixth rib. These vary in different individuals, and from 33 to 38 inches has been given by different observers as the average in adult males. The difference between extreme inspiration and expiration should not be less than two inches at the level of the nipple. It may be taken that the *mobility* of the chest is of more importance than its size. Attention should be paid to any undue flattening of the chest-walls, such as may have been caused by phthisis or old effusions.

Phthisis.—According to the Registrar-General,

one death in every eight is due to this cause. If the physical signs of phthisis are present the life cannot be considered insurable, except for very short periods, and even then on terms which would be rarely accepted by a proposer. However, as every condition of health has its insurance-price if we could appreciate its possible future with anything like accuracy, it is well to remember that phthisis is sometimes very chronic. Of these, fibroid phthisis and a well-defined single cavity in one lung are illustrations; but, if it were possible to accept them for the purpose of insurance, their chronicity should have been already shown by a long previous history, and the actual state of proposer should be without either fever or much wasting.

Hæmoptysis.—There is no question more frequently before the medical examiner than the influence of blood-spitting on the value of life. We have met with the following varieties:—

(a) After a run or strain in athletics, as rowing or lifting weights, a mouthful of fluid blood has been brought up, a few clots some hours, or next day, after; and then a total cessation of the symptom occurs, without any subsequent disorder of health.

(b) The patient having had cold or cough for a week or two, a 'few streaks' of blood mixed with phlegm have been brought up, or a spoonful of red blood in the morning on waking, and the symptom has recurred after an interval of days or weeks.

(c) Without acknowledged disorder of health, or cough, a robust or, it may be, a spare person may bring up a mouthful of fluid blood, dark, or it may be in clots, for a day or two, when it ceases, without any symptom of illness, but probably will return, some weeks or months afterwards.

(d) A copious blood-spitting, a cupful or pint, fluid at first, then clotted, possibly repeated in a day or two, with or without any manifest disorder of health.

Such are the most common descriptions of blood-spitting which are given us orally, or reach us as medical reports from examiners. As we are dealing with reports only, we do not mention the symptoms, such as bodily temperature, which should guide us if we had the opportunity of seeing the patient at the time.

(a) is the usual story of hæmoptysis from strain or injury, and may occur without any subsequent ill-health. It is common among the young and robust. Should an interval of months have elapsed since the occurrence, and the health remain good, and the absence of any physical signs of lung- or heart-disease be verified by examination, such lives may be accepted, with a small addition to the premium, especially if the applicant have passed thirty years of age.

(b) is the characteristic of commencing phthisis, and grave disorders of general health, as well as physical signs of lung-disease, will soon become manifest. The life is plainly not insurable.

(c) is probably of cardiac origin, or due to hæmophilia. That from heart-lesion is met with in two very distinct forms. The more severe is commonly due to mitral disease, especially stenosis, and occurs late in the history of disorders dependent on that cause; and evidence of venous congestion, and of dilated heart-chambers, will have generally been in existence long before a hæmorrhage into the lung takes place.

(d) is of not infrequent occurrence. A person

commonly under thirty-five will have brought up blood from the mouth on various occasions, fluid, but more commonly dark or clotted, and have but little disturbance of health. There is commonly no cough, nor any expectoration, and only slight dyspnoea may be complained of. The most careful examination will detect nothing wrong in the lungs, and the subjects have commonly large and well-made chests. There is no rise of temperature at or after the attack, and the nutrition of the body is perfect. The writer has examined very many such cases, and in all the only physical sign was a slight click with the systole at the apex or lower end of the sternum. He has watched many of them over a series of years, and found no more serious symptom than here stated. There is little doubt that they are chiefly due to mitral imperfection, and, as they may eventuate in serious disease, are not eligible for ordinary life-assurance. It might, however, be possible to accept some of them for very short periods at an advanced premium. See HÆMOPHTYSIS, p. 620.

Emphysema.—Proposers often present the physical signs of emphysema, especially at the bases of the lungs. The proposer may or may not be the subject of fits of asthma, but the breath is always short on exertion. The co-existence of bronchitis shown by *râles* of the dry, sonorous, or mucous character, most marked with expiration, is often found, and eventually few advanced cases remain unassociated with heart-failure. Cases of this kind, excepting those which present heart-symptoms, may be accepted with an addition to the premium, the amount of which will vary with the mildness or severity and the rarity or frequency of the attacks, and the degree of loss of elasticity in the lungs. The liabilities increase with age, and the premiums should be paid up before the proposer is much beyond middle life. A short occasional attack of asthma, without the characteristic physical signs, may not materially vitiate a life.

A liability to *bronchial attacks* manifests a delicacy of the air-passages, and is often observed in persons of a phthisical, gouty, or syphilitic family. Chronic bronchitis in the elderly is commonly found in emphysematous cases, and the gradual supervention of changes in the air-vesicles and chambers of the heart renders such lives precarious. They are, however, occasionally accepted for a short term, and with a considerable addition to the premium.

Pleural effusions, if existing, would prevent the acceptance of a life, but neither a contracted side from absorption of a former effusion, nor the marks of paracentesis for the evacuation of the fluid, if the recovery has been complete, and the lung has descended to nearly its former level, should forbid acceptance if the general health be good. It must be remembered, however, that many cases of pleural effusions are of tubercular origin.

Digestive tract.—The tongue and fauces should in all cases be inspected. The salivary glands should be free from hardness or enlargements. The fauces should be examined for marks of former ulcerations. The nostrils should be free from obstruction, and the voice not nasal. The stomach should be examined by palpation and percussion, as stricture of the pylorus, or dilatation, may be present, and if found would justify rejection of the life. The *liver* should not come below the ribs, and a breadth of about four inches represents the normal dulness from the lower ribs upwards. Inequalities on

the surface of the liver may mean cancer or cirrhosis or hydatids, and their presence would justify exclusion. Mesenteric tumours, fibroid, scrofulous, or malignant, are not admissible. Hardness in the right iliac fossa, especially with a history of former *appendicitis*, is also inadmissible. A history of recent attacks of *appendicitis* justifies the addition of an increased premium and in some cases the rejection of the life, unless the appendix has in the meanwhile been removed. Any of the following affections, if known to have existed, should be regarded with suspicion: *Hæmatemesis*, gastralgia with vomiting, hepatic colic, hydatid cysts, obstruction of the bowels, typhlitis or perityphlitis, and ascites. There are cases of *hæmatemesis*, generally in the female, or the result of strain or direct injury, which might be accepted, but only after years of perfect health have elapsed. *Jaundice* of former occurrence, if the attacks have been slight, would not forbid acceptance. *Biliary colic*, if the attacks have been severe or recent, would prohibit acceptance; but a former attack, with subsequent years of good health, need not imply rejection.

Dysentery which may have occurred in a hot climate, from which the proposer has been absent for some years, with good present health, need not disqualify, but a considerable extra premium will be required if the proposer returns to a hot climate.

All disorders of the *rectum*, except hæmorrhoids of a simple kind, are serious. Cancer, abscess, or the syphilomata, may be suspected if symptoms of pain or obstruction can be elicited. *Hæmorrhoids* need not disqualify unless they be of a severe form, or have recurred after operation, or presently call for surgical interference.

Genito-urinary organs.—Disorders of these organs are among the most important which affect the value of lives. They are often very insidious, as persons in a state of disease may present the appearance of perfect health. It is notorious that a man may have glycosuria or albuminuria, and neither know it himself nor exhibit in his appearance any evidence of the disorder. It is therefore imperatively necessary that the urine should be tested chemically in every case. This rule was formerly not adhered to, and many offices suffered in consequence; but it has now become a general practice, which has in some instances to be supplemented by a microscopical examination.

Hæmaturia of recent occurrence and *stone in the bladder* would forbid acceptance. *Dysuria* from stricture or enlarged prostate is an unfavourable condition, but there are mild cases of stricture which may be accepted with an addition.

Syphilis.—Syphilis affects the duration of life considerably, and is to be regarded not only from the length of time which has elapsed since the last attack and its cure, but from the nature and degree of the symptoms. The late Berkeley Hill believed that the curable cases get well in two years; the incurable may last for an indefinite time. For two years a person may expect a return of the eruption. Tertiary affections generally appear about five years after the primary affection. The rules for assurance may be thus stated: During the actual existence of any syphilitic disorder the proposal should be postponed. A man who has had syphilis in only the ordinary cutaneous and superficial form, and has been properly treated for it, may be accepted after a year has elapsed without symptoms. If he have had syphilis in the tertiary forms, he should

only be accepted if two years have elapsed since the latest symptom, and then with a small addition to the premium. Those who have had visceral syphilis affecting the brain, cord, liver, lungs, kidneys, &c., should be rejected.

Albuminuria is found in several chronic affections of the kidney, and in acute nephritis. It is also present in several acute inflammatory affections, with which we are not now concerned. Its presence should always be looked for. It is usually more copious after food and exercise, and its appearance in the urine is often intermittent, being absent at times for hours or even days. Acute albuminuria from exposure to cold and wet, and that which follows scarlatina, may disappear without leaving any organic change in the kidney, but in certain cases it recurs. If, however, some years have elapsed since its occurrence, and the proposer have enjoyed perfect health, the case may be accepted. In chronic cases, it is most commonly the result of some form of Bright's disease. There is, however, no doubt that albuminuria occurs in many persons without any other symptoms of disordered health, and may so continue for years. Concerning the importance of this there is much difference of opinion (see ALBUMINURIA). We should advise the following rules for practice:—

Albuminuria, formerly existing as a result of acute nephritis or scarlatina, with a return to ordinary diet and an interval of perfect health for years, need not disqualify a proposer. Cases of albuminuria existing, and known to have existed for years, in persons of otherwise perfect health and good family history, may be assured for a short period—say, of five years—with an addition to the premium, and may come up for examination again at the end of that period. Cases of albuminuria with any of the symptoms of *chronic Bright's disease*, with any cardiac, gouty, or rheumatic complication, or any suspicion of intemperate habits, should be declined. For the tests for albumen, see ALBUMINURIA. *Casts* in the urine confirm the diagnosis of organic disease of the kidneys.

Glycosuria.—Sugar may be present in the urine apart from diabetes mellitus, of which all the symptoms may be wanting. Glycosuria is often a temporary condition, and may disappear leaving no ill effects; but it is always to be regarded with suspicion, and its presence alone should forbid the acceptance of a life. It is common in obese persons, gross feeders, and in the gouty habit; and its subjects may have no suspicion of its existence, and feel in perfect health. The urine should therefore be always examined for sugar. Diabetes mellitus is a permanent condition, and the mortality from it increases with age up to sixty or seventy years. Males are twice as liable as females. It is difficult to estimate its duration, as dietetic measures can much retard its progress; but the prognosis, on the whole, is bad, as the liability to cerebral affections, to phthisis, carbuncle, and other fatal complications is very great. Its subjects, therefore, must be excluded from life-assurance. Glycosuria will forbid the acceptance of a proposer while it exists; but if there be no gout, and if all traces of sugar have disappeared for years, a calculated addition might be made to the premium (see GLYCURONIC ACID). In glycosuria the quantity of urea is generally increased. A high specific gravity, even above 1030, is no evidence of sugar in the urine, unless a chemical examination have

proved its existence. For the tests for dextrose, or grape-sugar, see *DIABETES MELLITUS*.

The excess of *phosphates* in the urine is generally temporary, and often due to anxiety and waste of nerve-power. It is recognised by a cloudiness on boiling, which is entirely cleared by the addition of a drop or two of nitric or acetic acid. It forms no obstacle to the acceptance of a life.

The presence of alkaptonuria implies no additional risk. See *ALKAPTONURIA*.

Disease of the *testicle*, tubercular or malignant, disqualifies for assurance. *Hydrocele* and *varicocele* may *per se* be regarded as harmless.

Female organs.—Metritis, pelvic inflammation, and ovarian disease, are doubtful cases for acceptance. Women who have had repeated abortions, puerperal hæmorrhages, eclampsia, or a necessity for obstetrical operations, are not eligible unless the menopause has been favourably passed. During a *first pregnancy*, it is well to defer assurance till after a safe confinement. Lactation is no bar to acceptance. The average duration of life in women is longer than in men, and by latest returns is shown to be increasingly so.

Nervous system.—Diseases of this system, as cerebral softening, locomotor ataxy, hemiplegia, paraplegia, and aphasia, disqualify a proposer. Local paralysis of the motor muscles of the eyeball, and ptosis, unless congenital, would lead to suspicion of brain-disease, and would not be acceptable. *Paralysis of the facial nerve* (Bell's), when purely local, and free from complications with disease of the ear and disorder of the fifth or other nerve, need not disqualify. *Paralysis* from lead-poisoning is always serious, and the liability to diabetes and deep disorders of the nervous system should not be forgotten. A recognition of the blue line on the gums would lead us to decline the life. *Neuralgias* of branches of the fifth nerve may not disqualify unless they be associated with cerebral symptoms, or be the result of syphilis or exostosis. *Vertigo* of a severe kind, associated with symptoms of *Ménière's* disease, would disqualify; and even its more transient forms, if often repeated or associated with gout, may be a bar to acceptance. *Trembling of the hands* may arise from organic disease or from the abuse of tea, tobacco, or alcohol. If from the latter cause, we may expect the tongue to be also tremulous. Although it may be temporary, its presence should cause grave suspicion as to habits.

The more severe forms of *epilepsy* must forbid acceptance, unless ten or fifteen years have elapsed since the last attack, with an interval of perfect health, and even then an addition should be made to the premium. There is a mild form appearing in early life, which can generally be traced to such causes as overwork or too close application to study, and where the heredity is absent; if years have elapsed without its recurrence, and the proposer has passed thirty and has engaged in the active work of life without symptoms of nervous disorder, and is strictly temperate, the life may be accepted with an addition of five to seven years.

Height and weight.—The relative proportion of height and weight is important in estimating the probabilities of future disease in a proposer. In estimating over-fatness, if the chest and shoulders be large and the abdomen moderate in size, the condition is less important than if the abdomen were large and pendulous. In stout persons, therefore, the circumference of chest, abdomen, and

limbs should be given, and flabbiness or firmness of tissue is to be considered, while habits as regards exercise, food, and stimulants should be carefully inquired after. The *dangers* of obesity are weakness of the heart or ultimate fatty degeneration of that organ. In a large proportion of these cases, fatty degeneration has affected the liver, and atheromatous alterations take place in the coats of the arteries, which lead to their ultimate rupture and consequent hæmorrhages. Obese persons are also worse subjects for fevers and accidents than are the spare and muscular, their power of recovery being lowered from feebleness of circulation. For these reasons the very obese are scarcely subjects for life assurance, although occasionally, if their habits be moderate, they may be accepted with an addition. On the other hand, the existence of excessive *leanness*, which may be the result of phthisis, diabetes, or various other wasting disorders, demands a careful examination. Most companies have tables of relative heights and weights, but a wide margin must be left for individual variations. One is appended which the writer has found sufficiently accurate:—

Height in inches.	Weight.		Height in inches.	Weight.	
	st.	lbs.		st.	lbs.
63·0	8	13	70·0	12	4
63·5	9	2	70·5	12	8
64·0	9	5	71·0	12	11
64·5	9	8	71·5	13	1
65·0	9	11	72·0	13	5
65·5	10	1	72·5	13	9
66·0	10	4	73·0	13	13
66·5	10	7	73·5	14	3
67·0	10	10	74·0	14	7
67·5	10	14	74·5	14	11
68·0	11	3	75·0	15	1
68·5	11	7	75·5	15	5
69·0	11	10	76·0	15	9
69·5	12	0			

Claims.—On the death of an assured life a claim is sent in to the office by the legal owners of the policy. This is accompanied by a certificate from the registrar of the district in which death occurred, and in some cases from the medical attendant. The practitioner who has to certify the cause of death cannot be too definite in his statement, nor too careful to avoid the use of terms which express only the symptoms and not the disease which proved fatal. Thus *dropsy* is common to diseases of the heart, liver, kidney, and other organs, and should not be employed; and for the same reason *hæmorrhage* and *diarrhœa*, which are common to many affections, should not be used. *Gastritis*, when certified as a cause of death, should always excite suspicion. The writer has known it used to conceal irritant poisoning and intemperance. A sudden attack of persistent vomiting with diarrhœa, leading within a week or two to a fatal issue, should not be received without careful inquiries as to the assumed cause of death. *Syncope*, which may represent the final stage of many diseases, should not be used. *Childbirth* is often used to cover other affections, such as phthisis, and should be limited to incidents proving fatal at or soon after a confinement.

We have also occasionally to consider whether the death of a proposer has been due to diseases

which must have existed at the time when the life was accepted, and therefore whether the statements made in the proposal as to the previous state of health and habits of a proposer were strictly true. If not true, the claim could not be maintained. There is here scope for all the acumen of the medical adviser of an Insurance Office.

It is believed that the practising physician will find in this article sufficient data to guide him in the selection or rejection of most lives offered for assurance. It is not possible to lay down specific rules for the amount of additions to the premium which it will occasionally become the duty of the medical examiner to advise. He may either rate a life at so many years older, or adopt some of the other methods by which the company may be secured against loss—as paying up all premiums within a given number of years, or deducting a portion of the sum to be paid by the company if death occur within a certain time, or making the sum payable at a certain age (endowment). Although on all these questions of finance the medical adviser can only recommend, his opinion will be highly useful to the company, with whom the ultimate decision will rest.

JAMES E. POLLOCK.

LIGHT, Therapeutic Effects of.—See SKIN, Tuberculosis of.

LIGHTNING, Effects of.—SYNON.: Fr. *Les Effets de la Foudre*; Ger. *Blitzschlag*.

The effects produced by lightning differ only in degree from those produced by the discharge of static electricity, generated in the laboratory. With a Leyden jar of sufficient size a small animal may be killed, and in larger animals the effects of shock and local injury may be produced. By lightning a person may be killed outright, and a *post-mortem* examination may reveal no lesion whatever. The mode of death in these cases seems to be by the shock to the brain and nervous system generally. Effects not distinguishable from ordinary concussion of the brain may be observed, and the person struck may remain insensible, with slow respiration, scarcely perceptible pulse, and dilated pupils, for periods varying from a few minutes to more than an hour. This may be followed by complete recovery; or there may remain paralysis of the limbs, usually the lower, or occasionally derangements of the special senses—blindness, a metallic taste in the mouth, noises in the ears, and an odour in the nose. The brain may be more or less permanently affected, and delirium, mania, and loss of memory may occur as results of the lightning-stroke.

Various objective phenomena have also been observed. The electricity on its way through the body may produce a number of mechanical effects. Wounds like those produced by a blunt stabbing instrument may mark the points of entry and of exit; bones have even been broken, the *membrana tympani* has been ruptured, and internal viscera have suffered in a similar way. Patches of erythema, urticaria, superficial ecchymoses, and scorplings of the surface having a curious tree-like and branched arrangement, have all been described; and this last phenomenon has apparently given rise to the assertion that delineations of trees standing in the neighbourhood of the accident have been traced photographically on the body of the victim. Lightning is apt to be attracted by any metal worn about the body. Watch-chains are frequently broken

and fused, and by the intense heating of these metallic conductors the clothing has been set on fire. Watches have been broken and partially fused, and have forcibly burst through the pockets in which they were contained. Steel articles, such as pocket-knives, have been rendered magnetic. The clothing is sometimes burnt and torn to a great extent, and strong boots have been found burst open, or thrown off the feet to a distance, or nails in the soles have been driven out of them.

The remote effects of lightning are due to the mechanical injuries produced by it. Permanent paralyses may result from injury to the nerves; and inflammatory action may be set up by the injury inflicted on internal or external parts. One case is recorded in which the whole of the hair on the head and body, as well as the nails of both hands, came off after a lightning-stroke. It has been asserted that *rigor mortis* does not occur in persons killed by lightning, and that the blood remains fluid for a very long time after death, but neither of these facts has been substantiated.

TREATMENT.—The treatment of those who have been struck by lightning consists in first rousing and keeping up the respiration and circulation. The cold douche is often of great value; and this, combined with friction of the limbs, warmth to the extremities, and the administration of stimulants, either by the mouth or in the form of enemata, would seem to be the measure best calculated to restore the suspended animation. Secondly, special injuries must be subsequently treated according to their nature. See RESUSCITATION.

G. V. POORE.

LIPIK (LIPPIK), in Slavonia.—Thermal mineral saline waters, with iodine. See MINERAL WATERS.

LIPOMA (λίπος, fat).—A fatty tumour. See TUMOURS.

LIPPITUDO.—Marginal blepharitis. See EYE, AND ITS APPENDAGES.

LIPPSRINGE and INSELBAD, in Germany.—Earthy waters. See MINERAL WATERS.

LISBON, West Coast of Portugal.—Warm, moist climate, with very variable temperature. Mean temperature in winter, 54° F. Prevailing winds, N.E.—S.E. in spring. See CLIMATE, Treatment of Disease by.

LISDOONVARNA, in Ireland.—Sulphur-waters. See MINERAL WATERS.

LITHÆMIA (λίθος, a stone; and αἷμα, the blood).—See URIC ACID DIATHESIS.

LITHONTRIPTICS (λίθος, a stone; and τριβω, I wear away).—SYNON.: Fr. *Lithontriptiques*; Ger. *Steinauflösende Mittel*.

DEFINITION.—Lithontriptics are therapeutical measures used for the purpose of dissolving calculi in the urinary tract.

ENUMERATION.—The chief lithontriptics are: Water, Potassium, Lithium, Borax, Phosphate of Sodium, Soap, Lime-water, Nitric Acid, Phosphoric Acid, Hydrochloric Acid, Sulphuric Acid, Piperazine, and Mineral Waters, such as those of Contrexéville and Wildungen.

ACTION.—Lithontriptics dissolve stones in various ways. Some of them possess a simple sol-

vent action, as in the case of water. Others unite with the calculi so as to form a more soluble compound, as in the case of the union of potassium or lithium with the uric acid of a calculus, producing urate of potassium or lithium, which is more soluble than uric acid itself. In the case of phosphatic calculi, diluted nitric acid combines with the bases of which they are composed, to form a more soluble compound.

USES.—Lithontriptics may be employed for the purpose of dissolving calculi either in the kidney or in the bladder. They may either be taken internally, so as to act upon the calculi through the medium of the urine; or be injected directly into the bladder. This latter treatment can only be adopted in the case of a vesical calculus, and is inapplicable in the case of a renal calculus. The most useful of all lithontriptics is water, especially distilled water. When this is taken in large quantities, the urine becomes very dilute, and small calculi may be partially dissolved, so as to be reduced in size and ejected through the natural passages. If the calculus is composed of uric acid, salts of potassium or lithium are the best remedies for internal administration, the urates of these bases being more soluble than the urate of sodium. Piperazine is a solvent of uric acid, twelve times as powerful as lithium carbonate. It has been used as a lithontriptic both internally and as a local injection into the bladder. In the case of phosphatic calculi, acid remedies are employed instead of alkaline; but it is exceedingly difficult to render the urine acid by means of acids given by the mouth, unless they are administered in quantities likely to derange the digestion. In place of mineral acids, benzoic acid and benzoate of ammonium have been employed, as benzoic acid passes out of the body in the form of hippuric acid, giving an acid reaction to the urine. On account of this difficulty, acids have been directly injected into the bladder, in order to act directly upon the stone; for which purpose nitric acid, largely diluted, is the one which has been most generally employed. This procedure, however, is now rarely had recourse to, as it is much easier to crush the stone by mechanical means. The two most important springs for the treatment of renal or vesical calculi are Contrexéville and Wildungen. The waters of both of these places contain carbonate of iron, which appears to aid their action.

T. LAUDER BRUNTON.

LITHOPÆDION.—See CONCRETIONS.

LITHURIA (λίθος, a stone; and οὐρον, the urine). A condition in which a deposit of uric acid or urates takes place in the urine. See LIVER, Functional Diseases of; URIC-ACID DIATHESIS and URIC-ACID CALCULUS; and URINE, Morbid Conditions of.

LITTLE'S DISEASE.—See CEREBRAL PLEGIA.

LIVER, Diseases of.—The diseases of the liver will be considered in the following order:

Abscess, p. 882.

Acute Yellow Atrophy, p. 885.

Amyloid Disease, p. 886.

Chronic Atrophy, p. 887.

Cirrhosis, p. 887.

Cysts, p. 890.

Enlargements, p. 891.

Fatty Disease, p. 891.

Functional Disorders, p. 892.

Gangrene, p. 893.

Hydatid Disease, p. 893.

Hyperæmia, Passive (Nutmeg), p. 895.

Inflammation and Active Hyperæmia, p. 896.

Malformations, p. 898.

Malpositions, p. 899.

Morbid Growths, p. 899.

Syphilitic Disease, p. 901.

Tuberculosis, p. 903.

The following articles will be found in their alphabetical position in other parts of the Dictionary: ACTINOMYCOSIS; BILE, Disorders of; BILIARY FISTULA; BILIOUS; CHOLAGOGUES; CHOLESTERIN; CONCRETIONS; GALL-BLADDER AND BILE-DUCTS, Diseases of; GALL-STONES; HÆMORRHAGE (including HÆMATEMESIS); PAIN IN VISCERAL DISEASE; PHYSICAL EXAMINATION; PORTAL OBSTRUCTION; PORTAL THROMBOSIS.

LIVER, Abscess of.—SYNON.: Hepatic Abscess; Fr. *Abscès du Foie*; Ger. *Leberabscess*.

Abscesses of the liver are divisible into two classes: (1) tropical abscess; (2) other forms of pyogenic infection, including those resulting from pyæmic conditions, especially such as arise from typhlitis and surgical operations or affections about the rectum; suppurating hydatid cysts and other parasites; traumatism, especially blows on the liver; gall-stones; and pylephlebitis. Tropical abscess will be alone considered in this article. Suppuration complicating other conditions is considered elsewhere. See pp. 565, &c.

Tropical Abscess of the Liver.—ÆTIOLOGY.—The association of abscess of the liver with dysentery has long been recognised, and the more the subject is investigated the more constant is this association found to be. So much is this the case that some recent writers incline to regard the terms 'tropical abscess' and 'dysenteric abscess' of the liver as synonymous. It may safely be asserted that in 75 per cent. of cases of hepatic abscess a history of some degree of dysentery—not necessarily of a very urgent character—can be elicited. It must be recognised, however, that in this association the order of events is not always the same: usually the dysentery is antecedent to the hepatitis; sometimes the diseases appear to be co-incident; more rarely the hepatitis appears to precede the dysentery; and in not a few cases the clinical manifestations of the two conditions alternate. The advocates of the ætiological identity of these diseases suggest that in a proportion of cases in which dysentery is not a feature either before, during, or after the occurrence of abscess, the pathological drama has not been played out. They argue that, just as dysentery may exist without hepatitis, hepatitis may exist without dysentery. Whatever may be the exact relationship between these diseases—a relationship succinctly expressed by the term 'hepatic dysentery'—it is certainly a very intimate one, and one which should always be borne in mind in the diagnosis of hepatic abscess, as well as in the prognosis of dysentery; and it should also be borne in mind that the abscess may not develop till months after the dysentery has been recovered from, and the patient perhaps has left the endemic area.

The most potent circumstance in predisposing to hepatic abscess is residence in a hot climate, especially if the heat is combined with humidity and, possibly, malaria. There are exceptions, but, speaking generally, liability to this form of abscess increases *pari passu* with these conditions. Intemperance in eating and drinking; lazy, luxurious habits; bad food; hardship and exposure, such as are incidental to a soldier's life in time of war; hot, close, and overcrowded barracks; and insanitary conditions in general have a marked though predisposing influence. Men are more subject to it than women, adults than youths; children seem to enjoy an almost absolute immunity. Race has undoubtedly some influence, but how it acts—whether in consequence of peculiarities of habit, acclimatisation, or specific racial idiosyncrasies—has not been adequately determined. It is certain that liver-abscess is much more common in Europeans residing in hot climates than in the ordinary natives, but natives who adopt the habits of Europeans as regards alcohol and rich living are said to approach the European in liability to the disease.

Among the circumstances which determine the formation of abscess in persons predisposed are, above all, chills of the surface such as arise from rapid alternations of heat and cold, from sitting in draughts in wet clothes, from bathing in cold water, or sleeping without adequate clothing. A blow on the liver, a bout of drinking, or a surfeit, is often the starting-point of the disease.

ANATOMICAL CHARACTERS AND PATHOLOGY. Tropical abscess of the liver may be single or multiple, and it may vary in size from a minute collection of pus to an enormous sac containing many pints of fluid, and occupying a large extent of the liver. When a section of a liver in a state of incipient suppuration is made, one or more circular patches, greyish-yellow or otherwise altered in colour, and surrounded by an area of congestion, are revealed. Abscess is formed by the breaking down of one of these patches, liquefaction commencing in the centre. Frequently two or more neighbouring patches coalesce. When the abscess has attained any size its walls are found to be very irregular, their inner surface being coated usually with a yellowish tenacious purulent material lying on a thin zone of more or less infiltrated liver-tissue—the abscess-sac—which in its turn is surrounded by a hyperæmic zone; beyond this the liver-tissue may be fairly healthy. Around and, so to speak, in the walls of the principal focus of suppuration a number of minute abscesses may form, and it is by the bursting of these into the main cavity, and the necrosis of pieces of intervening tissue, as well as by the slower process of purulent and molecular disintegration of its walls, that the cavity enlarges. The contents vary considerably: usually they are of a chocolate-brown colour, and have a thick gummy consistence tending to become gelatinous on cooling. The odour too is peculiar. Not infrequently lumps of necrosed liver-tissue or clots of blood are found floating in the fluid, which may also be streaked with blood, bile, or a yellow mucoid-looking pus of a paler colour. Occasionally the pus is more fluid, and may have the appearance of ordinary pus; rarely it is thin, watery, or grumous. It frequently contains a variety of micro-organisms, including the colon-bacillus, but in not a few instances the bacteria of pus are absent. It is further characterised in a certain proportion of

cases by the presence of a protozoal organism—the *Amœba coli*. See DYSENTERY, p. 426.

When the abscess is single its most usual situation is towards the back part of the right lobe, but it may occupy the left lobe or any other part of the gland. When abscesses are numerous, they are scattered throughout the liver, especially near the peritoneal surfaces.

Death may occur before the abscess ruptures; rarely it becomes encysted or absorbed; usually it makes its way to the surface of the organ and, after adhesions have been formed between opposing serous surfaces, opens on the surface of the body or into some of the neighbouring viscera. The most usual direction in which rupture occurs is into the right lung, next in frequency the intestinal tract, less frequently the surface of the body, and rarely into the pleural or peritoneal cavities or into the pericardium, gall-bladder, inferior vena cava, or pelvis of the right kidney. Rupture into the pericardium or inferior vena cava is necessarily and speedily fatal, but recovery has followed surgical interference after the rupture of a hepatic abscess into the peritoneum. Recovery may sometimes speedily ensue after rupture of the abscess, especially if it opens into the lung or intestinal canal; but in this event, in the majority of instances, the necrotic process is not arrested, and the patient sinks after perhaps a long illness. Recovery after surgical operation, even in the case of very large abscess, is often remarkably rapid, a great cavity of many ounces capacity healing up perhaps in a week or two.

Of the intimate pathology of hepatic abscess little, if anything, is known. By the discovery of the *Amœba coli* in connection with this disease and dysentery, the question seems to have entered on a promising field (see pp. 426, 428). It has been shown that the amœba occurs not only in the hepatic discharges, but also in the tissues surrounding hepatic abscess. That it is not invariably pathogenic is proved by its being sometimes present in the normal feces of apparently healthy individuals.

With reference to the association of dysentery and hepatic abscess, it ought to be mentioned that in some epidemics of dysentery abscess is of more frequent occurrence than in others; and this has been remarked, not only in epidemics in the tropics, but in epidemics occurring in Europe. In many of the latter hepatic abscess is almost unknown, but in others it is not so rare, as in the Dublin epidemic of 1818. Manifestly there is a specific element common to certain types of dysentery and hepatic abscess; but what this is, and how it operates, has not been determined. At one time it was held that the abscess was caused by the absorption of pus by the portal vein from the dysenteric ulcers, and from suppurating phlebitis which was supposed to be present in the intestinal radicles of that vessel. But no such phlebitis was demonstrated; and, moreover, it has been contended that as in certain instances the hepatitis may precede the dysentery, it therefore could not be caused by the latter. Further, as hepatic abscess is by no means a frequent sequela of gastric ulcer, of typhoid fever, or of tubercular or malignant ulceration of the intestine, the ulceration of the bowel in dysentery cannot *per se* be regarded as the cause of tropical abscess of the liver.

SYMPTOMS.—Sometimes these are fairly acute,

but in the great majority of cases they are of an extremely subdued and insidious character, so that sometimes the first intimation of the presence of abscess in the liver is its rupture into the lungs or intestine, and a discharge of the peculiar chocolate-coloured pus. Very often the symptoms amount to little more than feelings of ill-health, vague uneasiness about the region of the liver, and a state of low irregular feverishness. Temperature in the morning may be but slightly, if at all, above the normal, rising during the day to 101° or 102° ; or there may be spells of complete apyrexia or, occasionally, of high fever. Profuse sweating, particularly during sleep, is generally very marked. Slight feelings of chilliness occur from time to time, and in a few cases sharp rigors are met with; but too much importance must not be attached to their absence. The complexion may be muddy, and the sclerotics slightly yellow; the urine, though often loaded and dark, is seldom icteric; marked jaundice is rare. The temper is generally very irritable, and the spirits depressed. The tongue is sometimes coated; at other times it is clean. Appetite is usually poor; but occasionally it is fairly good. Vomiting may occur, especially if the left lobe of the liver is involved. The bowels may be confined; generally they are loose or irregular, often dysenteric. A short dry cough and some dyspnoea are not uncommon. The decubitus is usually dorsal or on the right side. In some cases pain of a cutting character in the region of the liver is a distressing symptom, and is regarded as evidence of perihepatitis, and the formation of adhesions between the peritoneal surfaces. In other cases the pain is of a very subdued type—merely a sense of weight and uneasiness; but it is usually intensified by pressure or percussion or smart compression, and it is often observed that percussion over the lower part of the abdomen may cause pain not where the blow falls, but in the region of the liver. Firm pressure on the liver often gives rise to a feeling of nausea or faintness. Pain in the right shoulder or scapula is one of the classic symptoms of hepatic abscess, and is regarded as an indication that the mischief is in the neighbourhood of the diaphragm. The abdomen is generally on inspection seen to be motionless and full. The liver is enlarged, but not always to a great extent; the enlargement may be upwards, a more significant sign than when it is enlarged in a downward direction. There may be a general bulging of the right hypochondrium, and obliteration of some part of one or two intercostal spaces, with a degree of œdema or even redness of the integuments. In wasted patients with normal livers the epigastrium should be concave when they are lying down, but in abscess of the liver it is often flat or even bulging, and this is more apparent when the patient is made to stand up; this appearance, with fever and tenderness, is always a suspicious symptom. Friction or crepitation may be heard at some part of the base of the right lung; the presence of these indicates proximity of the abscess to the diaphragm; and if, in addition, the *Amœba coli* is found in the sputum, rupture of the abscess into the lung is impending. When this occurs, a thick, gummy, chocolate-brown pus—quite pathognomonic in appearance, and yielding the reactions of bile when tested chemically, and exhibiting granular and degenerated liver-cells under the microscope—is brought up. In consequence of the persistent coughing after the abscess has opened and partially

emptied, there may be much hæmorrhage from its walls, and a very bloody sputum; so that the great bulk of the expectoration may be blood, with here and there only a streak or pellet of the chocolate-brown pus. This appearance is very apt to deceive the unwary, and to lead to a diagnosis of pulmonary hæmoptysis. The stethoscope may detect approximately the point of entrance of the abscess into the lung, and occasionally it reveals peculiar and very loud bruits, synchronous with the pulse somewhere about the upper border of the hepatic area, and sometimes of a to-and-fro character. These bruits probably arise from a to-and-fro play of air out of and into a partially empty but uncollapsed abscess-sac through a narrow sinus, and are produced by the jog communicated to the inflated abscess-sac by the systole of the ventricles. Should the abscess open into the bowel, there may be one or two loose stools of a similar chocolate-brown character; and in a case of suspected abscess of the liver the physician ought always to bear this in mind, and inspect any loose motions reported to him. Rupture into the stomach may be followed by vomiting; rupture into the pericardium or peritoneum, by sudden collapse.

DIAGNOSIS.—This is often a matter of great difficulty, even to the most experienced, and at times only to be settled by the development of events, or the use of the aspirator. An excellent rule for the practitioner in countries in which hepatic abscess is endemic is as follows: Whenever persistent ill-health is associated with a low degree of fever, and symptoms—no matter how trifling—referred to the abdomen, which cannot be readily accounted for, suspect abscess of the liver, and examine the organ daily with this possibility in view, and inquire carefully for a history of dysentery. It must be borne in mind, too, that the dysentery may have been very trifling, called perhaps diarrhoea, and that it may have occurred several months before the health began to break down. The significance of enlargement of the liver, localised hepatic swelling, pain, tenderness, a fixed and motionless abdomen, rigors, and a hectic temperature following dysentery can hardly be misunderstood. But it is seldom that in the early and more hopeful stages of abscess the indications are so unequivocal; indeed it may be, and often is, entirely overlooked, and not discovered until it has burst into lung or bowel, or until it is found on the *post-mortem* table. The presence of pneumonic crepitus about the base of the right lung in liver-cases is often misinterpreted; rightly understood, it is a most valuable confirmatory sign, especially if it is limited to a small patch. Sometimes the periodicity of the fever, the severity of the rigors, and regular procession of pyrexia and diaphoresis in hepatic abscess, lead to a diagnosis of ague; but the impotence of quinine to cure the fever, and the absence of the *Plasmodium malarie* in the blood, and of splenic enlargement—which is nearly always present when the liver is affected in malarial hepatitis, which rarely ends in abscess—ought to awake suspicion. When in a malarial subject there is a history of dysentery, and both liver and spleen are enlarged, diagnosis in the early stage of hepatic abscess is nearly impossible without the aid of the aspirator. In any case where grave doubt exists, where fever is persistent, and the patient is manifestly losing ground, exploration with a medium-sized aspirator-needle must not be delayed. This trifling operation, if attended with

any risk, is infinitely less dangerous than procrastination. With proper precautions it is not a dangerous proceeding; so far from doing harm it has often a markedly curative effect in cases of hepatitis, even when there is well-marked local bulging. As an encouragement to early recourse to the aspirating needle, it ought to be borne in mind that hectic fever may occur in hepatitis before the formation of pus, and even in cases in which pus never does form; and the whole trouble which otherwise might have ended in abscess may rapidly subside after exploratory needling. In exploring the liver with the aspirator, in the absence of definite localising indications, the needle ought to be inserted in the first instance about the seventh or eighth interspace in the anterior axillary line; if pus is not found here, a second and third puncture should be made, the needle being driven in to its full extent if necessary, in front just below the ribs a little to the inside of the nipple-line, and again behind in a line with the angle of the scapula well below the edge of the lung. *See PARACENTESIS.*

PROGNOSIS.—It is impossible in the present state of medical knowledge to say how dysenteric hepatitis will terminate, whether in resolution or abscess. Should the latter form, provided it is single and opened soon and effectually drained, the prognosis is fairly good; quite 50 per cent. recover. If left too long or allowed to open of itself, the prognosis is bad, though by no means hopeless. If two or more abscesses are present, the chances of recovery are proportionately diminished; and if there are many suppurating points scattered through the liver, death is inevitable. If after operation the temperature quickly becomes normal and keeps so, prognosis is favourable; but if, on the other hand, after operation there is a distinct evening rise and a continuance of the hectic fever, the worst may be apprehended.

TREATMENT.—Hepatitis threatening abscess ought to be treated by absolute rest in bed, low diet, and, in the absence of diarrhoea or dysentery, saline aperients, such as sodium sulphate. The right side ought to be covered with a large hot frequently renewed poultice, a foot in breadth and three feet in length, which should pass from beyond the middle line behind to beyond the middle line in front. If there is dysentery, a thirty-grain dose of ipecacuanha should be given, and repeated as in ordinary dysentery (*see DYSENTERY*). In the absence of intestinal trouble, chloride of ammonium in twenty-grain doses every six hours is sometimes exhibited with advantage. Quinine is indicated in malarial complications.

Symptoms not improving, exploration with the aspirator must not be too long delayed. If pus is found the abscess should be opened forthwith, thoroughly drained, and treated on Listerian principles. It may be remarked that in no surgical affection is it more necessary to observe the great surgical principles of early opening, free drainage, and perfect asepsis. By these means the mortality in this affection has of late years been enormously reduced, and a diagnosis of liver-abscess is now no longer to be regarded, as it was in former days, as almost tantamount to sentence of death. The opinion of the physician is sometimes asked as to the propriety of making an external opening in the case of an abscess discharging through the lungs or intestinal canal, or from an inefficient sinus in the walls of the chest or abdomen. A good rule to observe in such

a case is this: If the patient is improving, fever absent, and discharge lessening, do not counsel interference; if, on the other hand, hectic fever persists, the discharge increases or does not diminish materially, and the patient continues to lose ground, advise operation. If there be doubt about which course to advise, recommend change of air, which has sometimes a wonderful influence in wavering cases in inducing healing. Should fever and ill-health persist after the abscess has been freely drained, and the drain, though effective, give vent to very little discharge, suspect the presence of a second abscess, and advise further exploration, and—if pus is found—further operation. Recovery has followed after several abscesses have been so treated in the same individual. If the temperature keeps up after all abscesses have been opened, and discharge from the drainage tube is profuse, it is an indication that the necrotic process is still going on in the liver; the prognosis is then most unfavourable. Gangrene sometimes sets in around the operation-wound, and is almost invariably fatal.

When pus has formed, the diet ought to be fairly liberal; but overfeeding and overstimulation are under all circumstances to be carefully avoided.

PATRICK MANSON.

LIVER, Acute Yellow Atrophy of.—**SYNON.**: *Fr. Atrophie Jaune Aiguë du Foie; Ictère Grave; Ger. Acute Atrophie der Leber.*

DEFINITION.—An extremely acute inflammation of the liver, due to some unknown poison.

ÆTIOLOGY.—Acute yellow atrophy is a rare disease. It may occur at any period of life, but is most prevalent between the ages of 20 and 30. It is twice as common among women as among men; pregnant women are especially liable to it (*see PREGNANCY, Diseases of*). Emotional disturbances, such as grief and trouble, and bad hygienic conditions, have been thought by some to predispose to this disease. It is occasionally preceded by syphilis and acute alcoholism, and may supervene upon cirrhosis of the liver. It was originally attributed to phosphorus-poisoning, which causes a closely allied condition.

ANATOMICAL CHARACTERS.—The appearances are those of an acute necrosis, combined with slight reactive inflammation. The liver is small: in extreme cases it may weigh as little as nineteen or even thirteen ounces. It decreases in all diameters, but often irregularly. The capsule is wrinkled, and the organ is limp and flabby, not unlike collapsed lung (Rolleston). On section the outlines of the lobules are almost, if not quite, lost, and the surface is of a bright yellow-ochre colour broken by reddened patches. The yellow represents the early and acute stage of the disease; the red represents the later changes, in which most of the liver-cells have disappeared, the colour being partly due to distension of blood-vessels. By these signs the liver may be readily distinguished from that in phosphorus-poisoning, in which condition the liver is large and contains large quantities of fat. The liver in acute yellow atrophy contains very little fat. The gall-bladder and bile-ducts are either empty or contain a grey mucus. Under the microscope, the liver-cells are seen to be shrunken, granular, and bile-stained. Around both intra- and interlobular veins are a number of round cells, probably leucocytes. Charcot-Leyden crystals, as

well as crystals of leucine and tyrosine, have been found, especially when the liver has been preserved for some time. Double parallel rows of cubical cells, such as are seen in some forms of cirrhosis, are generally present.

Bacteria have been observed in the liver, but their pathological significance is doubtful. The spleen is enlarged and soft in the great majority of cases. The stomach and alimentary canal present dark red or tarry contents, the outcome of hæmorrhage; the tubular glands of the stomach are filled with fatty degenerated epithelium. The muscular tissue of the heart shows likewise granular or fatty degeneration; and the tubules of the kidneys are filled with epithelium in various stages of fatty degeneration.

SYMPTOMS.—Acute yellow atrophy is commonly preceded for some days or weeks by a simple jaundice, in which nothing peculiar can be made out. Delirium and convulsions then suddenly set in, followed by deep coma, stertorous breathing, dilated pupils, and increased jaundice. During the first part of the disease the pulse is natural or reduced in frequency, but with the appearance of the convulsions and delirium it rises to 120 or 130, becoming very feeble. The urine is normal in quantity, allowing for the vomiting, and bile-stained, and usually contains leucine and tyrosine. In some cases it is necessary to evaporate the urine to a concentrated condition to detect these. The amount of urea excreted is small (*see* LEUCINE; and TYROSINE). There is almost always constipation, the stools being at first pale, afterwards black from admixture of blood. Vomiting is very constantly present; at the end of the disease the vomit consists of a black coffee-ground matter. The tongue becomes dry and tremulous. The right hypochondriac and epigastric regions are painful and tender. The liver, at first tender and natural in size, or even larger than natural, decreases daily in dimensions, so that at last percussion may give no liver-dullness at all—the softened condition of the liver allowing it to become folded on itself, and the intestines rising up to take its place. With the decrease of the liver, the spleen increases in size. A hæmorrhagic diathesis likewise sets in, as shown by petechiæ on the skin, epistaxis, hæmatemesis, and melæna. The temperature is commonly low, until just before death.

DIAGNOSIS.—The prodromal stage cannot be distinguished from catarrhal jaundice. When the graver symptoms develop the possibility of phosphorus-poisoning must be considered. In the latter disease, the history, the early onset and severe symptoms of gastric irritation, the persistence of the enlarged liver, and the continued excretion of a fair quantity of urea are the points which will best ensure its recognition. Slight disturbances of temperature are not sufficiently characteristic to be of any diagnostic importance.

PROGNOSIS.—The prognosis is extremely bad; probably no cases recover.

TREATMENT.—The treatment must be conducted upon general principles. Intestinal antiseptics, e.g. salol, salicylate of bismuth, β -naphthol, have been recommended. The diet should be limited to milk, diluted, if preferred, with soda-water.

H. MONTAGUE MURRAY.

LIVER, Amyloid Disease of.—**SYNON.**: Albuminoid or Waxy Liver; Lardaceous Liver; Fr.

Dégénérescence Amyloïde du Foie; Ger. *Amyloïde Entartung der Leber*.

ÆTIOLOGY.—The ætiology of amyloid disease of the liver is the same as that of amyloid disease elsewhere. *See* AMYLOID DISEASE.

ANATOMICAL CHARACTERS.—The liver has its normal shape; is more or less enlarged, sometimes to such an extent as to fill the greater part of the abdominal cavity; it has a clean sharp edge, and is firm, resistant, and inelastic, with a smooth glistening surface. The organ cuts like bacon, hence the name 'lardaceous.' The cut surface is a translucent grey, like gelatine or boiled sago, or, in advanced cases, a peculiar subdued red, like that of raw ham; but sometimes it is yellowish or speckled, the liver being anæmic and often fatty also. From the incised veins a little pale blood usually oozes. The characteristic staining-reactions are described elsewhere (*see* AMYLOID DISEASE). The structures invaded by the new material have, in a section examined microscopically, a lustrous, transparent, and somewhat swollen appearance. When the entire lobule is affected, the aspect is homogeneous. The appearance of an amyloid liver may be modified by the co-existence of fatty change, or cirrhosis, or syphilitic disease. The spleen is generally, the kidneys not infrequently, and the intestine and other structures sometimes implicated.

SYMPTOMS.—Palpation, in marked cases of amyloid disease of the liver, will readily detect a large hard resistant tumour, having the normal outlines of this organ; the smoothness of its surface; and the extent to which it encroaches upon the abdominal cavity. Pressure does not elicit any tenderness, nor is there usually any pain; at most, in advanced cases, there is only a sense of tension and fulness, as in other hepatic enlargements. The painless nature of the tumour is distinctive. The disease does not interfere with the portal circulation, and does not therefore directly cause ascites. When this occurs, it is the result of general cachexia, induced by the constitutional malady, and perhaps by associated renal complication. The dropsy generally affects the legs in the first instance, and afterwards the serous cavities, and is not a prominent symptom unless the kidneys are implicated (*see* KIDNEY, Amyloid Disease of). There is no jaundice; or if this occur—a rare event—it is from pressure on the bile-duct externally by enlarged lymphatic glands. The evacuations are, however, frequently of a pale yellow, and at times of a clayey, colour, which may be accounted for by the extensive impairment of secreting structure, and the consequent secretion of a poor colourless bile. Vomiting, without other indications of gastric derangement, and diarrhœa, are symptoms not uncommon in advanced cases, and are no doubt due to the implication in the disease of the vessels and villi of the stomach and intestines.

DIAGNOSIS.—The peculiar features of the enlargement, its painless character, the concurrence of the usual ætiological factors, especially if the spleen and kidneys be involved, will distinguish an amyloid liver from other kinds of hepatic enlargement. If there be associated cirrhosis or syphilitic disease, the diagnosis will be difficult.

PROGNOSIS AND DURATION.—The disease may run on for months or even years. It may disappear if the process that caused it can be arrested, as is sometimes the case with chronic suppuration. But it generally proves fatal, either by intercurrent

affections, or by anæmia, general dropsy, and, exhaustion, such result being more rapidly determined when the kidneys are involved.

TREATMENT.—Surgical measures, directed towards the closing of sinuses, or the stoppage of any discharge that may be present, are of the first importance. But there are many cases—in advanced phthisis, for example, where the position of the disease is a bar to such treatment, or in syphilis—where the only treatment that can avail must be directed especially to the associated cachexia. Whether this be syphilitic or tubercular, the preparations of iodine are indicated: the iodide of potassium, the tincture of iodine, or, where the anæmia is marked, iodine in combination with iron. The syrup of iodide of iron in drachm-doses, three times a day, has proved useful, if not in reducing the tumour, at least in improving the general condition of the patient. The iodine mineral springs, as Woodhall Spa, Kreuznach, Adelheidsquelle, &c., are indicated. The baths of Aix-la-Chapelle, Ems, and Weilbach have each had their supporters in the treatment of this malady. Chloride of ammonium, in ten to twenty grain doses, three times a day, continued for some time, has been found to be efficacious in reducing large hard livers. The general therapeutical indications are pure air, plain nourishing diet, and adequate protection of the skin by warm clothing and other measures.

JAMES F. GOODHART.

LIVER, Atrophy of, Chronic.—A small liver is seen in many wasting diseases, and in old age. The liver shrinks, becoming tougher in consistence, but rarely granular on the surface. The cut surface is dark red or pale brown; the acini are either invisible, or else smaller than natural. The increased toughness is due to the atrophy of the liver-cells, and the resulting condensation of the connective-tissue of the organ.

LIVER, Cirrhosis of.—**SYNON.**: Interstitial Hepatitis; Fr. *Cirrhose du Foie*; Ger. *Chronische interstielle Leberentzündung*.

The term 'cirrhosis' was originally employed by Laennec, who considered that the liver was full of a yellow (*κίττός*) new growth.

Under this heading two forms of cirrhosis of the liver must be dealt with: (i.) *Common or portal cirrhosis* and (ii.) *Biliary cirrhosis*. A third variety, *Interlobular cirrhosis*, is described under syphilitic disease of the liver.

Portal Cirrhosis.—**SYNON.**: Common, atrophic, or multilobular cirrhosis; Hobnailed liver; Chronic interstitial hepatitis; Gin-drinker's liver.

DEFINITION.—A chronic disease, characterised by dyspepsia, hæmatemesis, ascites, little or no jaundice, debility, and a semicomatose state before death. The morbid changes are a multilobular fibrosis of the liver and degeneration of the liver-cells.

ÆTIOLOGY.—The morbid process is set up by poisons reaching the liver, chiefly, though not entirely, by the channel of the portal vein. *Chronic alcoholism* is such a common feature in the history of patients with cirrhosis that it might at first sight appear that the direct action of alcohol on the liver produced cirrhosis. Experiments on animals, however, while proving that alcohol is a protoplasmic poison and induces fatty degeneration in the liver-cells, do not support the view that, *per se*, it leads

to hepatic fibrosis. The action of alcohol is probably indirect. Gastro-intestinal catarrh is the first step; this gives rise to the formation of poisons in the alimentary tract, which, when carried to the liver, cause cirrhosis. *Dyspeptic* cirrhosis has been described and referred to the action of fatty acids, such as acetic, butyric, valeric, and may be set up by stimulating articles of diet, such as spices, curries, and sauces. The association of alcoholism with cirrhosis has been thought, but without any convincing evidence, to depend on bodies other than alcohol contained in wine, whisky, &c., such as sulphate of potassium (Lancereux) and amyl-alcohol.

In some instances cirrhosis may be due to the effects of poisons generated in the course of the *specific fevers*—scarlet fever, measles, typhoid, &c. *Malaria* has been assigned as a cause, but here the causal relationship is very doubtful. It is probable that micro-organisms absorbed from the bowel may give rise to cirrhosis (Adami), but convincing proof is not yet forthcoming.

The poison of syphilis, especially in young children, may be looked upon as so preparing the soil that the factors ordinarily inducing cirrhosis work at a great advantage, and a 'parasymphilitic' cirrhosis results. It has recently been suggested that poisons manufactured in the spleen in severe splenic anæmia (Banti's disease) and other infections may, when carried to the liver, cause cirrhosis.

Sex and age.—It is commoner in men than in women, in the proportion of $5\frac{1}{2}$ to 2. The average age at death is about forty-eight years; it may, however, occur in young children.

MORBID ANATOMY.—The size of the liver is very variable: it may be smaller than natural, and justify the term 'atrophic'; in such instances it may only weigh thirty ounces. On the other hand it may be enlarged to twice its normal size. Sometimes, but by no means always, large cirrhotic livers show extensive fatty change (*fatty cirrhosis*). A large (non-fatty) cirrhotic liver is often associated with latency of symptoms. The larger cirrhotic livers are less knobby or hobnailed than the smaller ones. In some instances the 'hobnails' show hyperplasia, and are so exaggerated that the condition is called *nodular cirrhosis* or *cirrhosis with multiple adenoma*. A further change in the 'adenomata' may result in the development of carcinoma—primary carcinoma with cirrhosis. This is a very rare event. The surface of the cirrhotic liver is irregular, granular, or hobnailed; the capsule is somewhat thickened and opaque, and may be united to the diaphragm by adhesions which are markedly vascular. The 'hobnails' are lobules of liver-cells squeezed into prominence by the fibrous tissue contracting around them, and are often yellow from fatty change and bile-staining, thus justifying Laennec's term 'cirrhosis.'

As a rule the liver is uniformly affected, but sometimes the left lobe is in a more advanced state than the rest. On section the organ is much firmer than natural, and is seen to be divided up by grey, slightly gelatinous-looking strands of fibrous tissue into small compartments of varying size. Microscopically fibrous tissue in various stages of development and containing elastic fibres spreads out from the portal canals to form meshes of different sizes, and encloses a varying number of hepatic lobules. The condition is therefore called multilobular cirrhosis, in contrast to unilobular cirrhosis

(*vide* Biliary Cirrhosis), where the tendency is for each lobule to be separated from its fellows by a distinct fibrous sheath. In the fibrous tissue small round cells are present when the process is advancing. Columns of deeply staining small cubical cells—the so-called new bile-ducts—are frequently seen in the fibrous tissue. Their nature has given rise to a good deal of discussion. The liver-cells are atrophied, and frequently show fatty change; pigment-granules are sometimes present, especially in malarial cases. Compensatory hyperplasia of the liver-cells may occur, and probably plays an important part in nodular cirrhosis and in the formation of the so-called new bile-ducts.

The gall-bladder and bile-ducts are usually healthy, but small bilirubin-calcium calculi are not infrequent in the ducts. The branches of the portal vein are compressed inside the liver, while its trunk usually shows some slight thickening. An important compensatory mechanism, by which the blood of the portal vein can get into the inferior and superior venæ cavæ, is provided by dilatation of the normal anastomoses between the portal vein and the general systemic veins. The more important of these anastomoses are (a) the communications between the œsophageal and the gastric veins; (b) between the superior hæmorrhoidal and the other hæmorrhoidal veins; (c) the veins of Sappey (paromphalic) in the falciform ligament of the liver and the veins of the abdominal wall around the umbilicus; and (d) widespread anastomoses between the veins of the peritoneum, duodenum, colon, and those of the abdominal parietes. The first two of these anastomoses may give rise to œsophageal varices and to hæmorrhoids respectively.

The spleen is enlarged in cirrhosis, not from backward pressure alone but also from a toxic condition of the blood, combined with passive engorgement. The stomach and intestines show signs of chronic catarrh; the heart is flabby, and may present fatty degeneration.

Two important though not necessarily associated lesions are tuberculosis, especially of the lungs, and renal disease.

CLINICAL COURSE AND SYMPTOMS.—The onset is often preceded by long-standing dyspepsia and by symptoms of chronic alcoholism. The face may be muddy and sallow, the venules dilated to form 'stigmata,' and the conjunctivæ slightly icteric; well-marked jaundice is not common. The tension of the pulse is low, the temperature, as a rule, normal, and the tongue furred. At this stage the liver will be found to be enlarged and tender, while the spleen is palpably enlarged. The urine is high-coloured, and is usually free from albumen. The disease may be divided into the early or pre-ascitic stage, and the late or ascitic stage. Hæmatemesis usually occurs a considerable time before ascites develops, and only exceptionally after ascites has made its appearance. It may come on suddenly and without warning, or be preceded by malaise or a debauch. The amount of blood vomited is large, and though it may give rise to severe anæmia, hæmatemesis is rarely fatal. It is followed by melæna. It may be due to ulceration of a varicose œsophageal vein, and may then be repeated—this is the condition found in most of those rare cases where hæmatemesis in cirrhosis is fatal—to gastritis, or to minute erosions or breaches of surface of the gastric mucosa. As a rule, after recovery from the hæmatemesis the patient continues in much the same

state as before for a considerable time, and may even, if fortunate enough to change his habits, regain good health, the disease becoming compensated for or latent.

In most cases, however, ascites develops after a varying interval; its appearance is often preceded, and its onset marked, by a flatulent condition of the abdomen. Since ascites occurs late in the disease, the patient usually is somewhat wasted and has lost strength and vigour. The onset of ascites is usually gradual; its rapid development may be due to thrombosis of the trunk of the portal vein—an occasional complication of cirrhosis. Ascites may interfere with a correct estimate of the size of the liver and spleen, and may be either preceded or followed by œdema of the feet. It is often said that the liver is larger in the early stages, and subsequently contracts and becomes smaller; variations in size undoubtedly occur, but depend more on congestion and its relief than on fibrosis and its subsequent cicatricial contraction. The abdominal wall is stretched, somewhat thin, and may show lineæ albicantes, while dilated veins (*caput Medusæ*) around the umbilicus are usually present. As the ascitic effusion increases the heart is pushed up, an apical or basic systolic murmur being not uncommon; while the thorax is encroached upon so that respiration is interfered with. Under these conditions paracentesis should be performed without delay. In cirrhosis, uncomplicated by chronic peritonitis, paracentesis is rarely required more than once or twice: the ascites may then cease to collect or even pass away; but the patient rapidly fails, becoming drowsy and exhibiting toxic symptoms due to hepatic inadequacy or, in other words, to the liver failing to arrest poisons derived from the alimentary canal (*see* ASCITES). There may be low delirium or semi-coma, and occasionally convulsions. In this stage hæmorrhages into the skin and bleeding from the gums and other mucous membranes may occur; epistaxis is not infrequent. Sometimes death is due to diarrhœa, hæmatemesis, or melæna, but usually it is preceded by increasing weakness and coma.

COMPLICATIONS.—Tuberculosis is frequently associated with cirrhosis; phthisis may indeed sometimes throw the symptoms of cirrhosis completely into the shade. Tubercular peritonitis is another complication that may occur. Owing to diminished resistance, acute infections, such as erysipelas, pericarditis, infective endocarditis, and peritonitis, may supervene; or occasionally an acute infection may attack the liver itself and give rise to the symptoms of icterus gravis. In very rare instances primary carcinoma supervenes in a cirrhotic liver—primary carcinoma with cirrhosis; the symptoms are the same as in cirrhosis.

Thrombosis of the portal vein occurs in a small proportion of cases and shows itself by the rapid development of ascites. Right-sided pleural effusion is not uncommon, and may be due to the spread of inflammation, or possibly infection, from the liver; sometimes the effusion is blood-stained, and is then associated with tuberculosis. Marked cardiac failure may occur and obscure the existence of cirrhosis. Some degree of alcoholic neuritis may also co-exist.

DIAGNOSIS.—The history of alcoholism, hæmatemesis not manifestly due to other causes (*see* p. 620), with splenic and possibly hepatic enlargement, followed by ascites, are the important points in arriving at a diagnosis of cirrhosis. The association of hæmat-

emesis and ascites strongly suggests cirrhosis. The chief conditions from which a differential diagnosis is necessary are syphilitic disease of the liver, malignant disease, and perihepatitis and chronic peritonitis. Signs of syphilis should always be looked for carefully, and it is a good rule to give iodides in cases thought to be cirrhosis, since in gummatous disease of the liver all external signs of syphilis may be wanting. In carcinoma of the liver it may be impossible to be certain until the ascitic fluid is drawn off, and the liver can be carefully examined. In chronic peritonitis and perihepatitis ascites collects again and again after tapping, which it does not always do in cirrhosis; moreover, there may be signs of arterio-sclerosis, granular kidney, or adherent pericardium, conditions which may be responsible for chronic peritonitis. Hæmatemesis is rare in the conditions, which, by inducing ascites, may be confused with cirrhosis. *See ASCITES.*

PROGNOSIS.—In the early stage and when hæmatemesis has occurred, strict obedience to treatment and the avoidance of alcohol and of stimulating diet may be followed by long latency or even apparent cure of the disease. Much therefore depends on the patient's power of will; while his state of nutrition is of considerable prognostic importance. When ascites has developed the outlook is extremely grave, and most cases die within a few months after being tapped once or twice. Many of the reported cures in cases at this stage are probably examples of syphilitic disease. The maintenance of a free secretion of urine and of a healthy state of the kidneys is in favour of the patient.

TREATMENT.—In the early stage strict abstinence from alcohol is essential, and care should be taken not to give tinctures or spirituous solutions of drugs. The diet should be mild and be free from spicy or stimulating articles of food. Milk, fish, and simple puddings should be taken. The bowels should be kept open and any tendency to dyspepsia checked by careful dieting and appropriate drugs, such as bismuth, bicarbonate of sodium, bitter tonics, dilute acids, &c., according to its character. Exercise and fresh air should be enjoined. Benefit will accrue from a 'course' at Carlsbad, Vichy, or Marienbad, or the Carlsbad waters and 'course' can be taken in this country. Hæmatemesis must be treated by rest, rectal feeding, and opium. Styptics may be given by the mouth, if the hæmatemesis is repeated. Two days after hæmatemesis a saline purge and a blue-pill should be given to clear the blood out of the bowel. Iodide of potassium combined with iodides of sodium and ammonium may with advantage be given both in the early and in the later stages of the disease, on the chance that the disease is really syphilitic, but no drug can remove the fully formed fibrous tissue. The object of treatment is thus to prevent any increase in the morbid change and to allow compensatory changes—hyperplasia of the liver-cells, and the increased anastomoses between the portal vein and the general systemic veins—to develop. It is, therefore, important to keep up the patient's strength and nutrition by tonics.

In the late or ascitic stage free, but not too vigorous, action of the bowels should be promoted by salines and small doses of blue-pill or calomel. Diuretics are useful, especially in the form of the pill containing mercury, squills, and digitalis (Addison's pill); citrate of caffeine may be tried. Copiba is a good diuretic, but care must be taken that it does not upset digestion. Ascites should be tapped when any

embarrassment of respiration, pain, or hæmatemesis occurs, and should not be long postponed; Southey's tubes should be used, and the abdomen be subsequently compressed by a binder or many-tailed bandage. Continued or permanent drainage has not proved successful. The surgical treatment of promoting vascular adhesions between the liver and the abdominal walls and so increasing the compensatory collateral circulation has been employed of late years with some success.

In the ascitic stage a milk-diet must be adhered to; it has the advantages of minimising fermentation and acting as a diuretic.

When toxic symptoms have developed diuretics and purgatives should be continued; hæmorrhages should be met by chloride of calcium, and cardiac failure counteracted by hypodermic injections of strychnine. Considerable benefit follows intravenous transfusion. The writer has on several occasions seen a semi-comatose patient restored to consciousness for some weeks by this means (*see SALINE SOLUTION, INFUSION OF*). When in the last stage of weakness alcoholic stimulants are often given; gin is probably the least harmful, but strychnine hypodermically should be given a trial first.

Biliary Cirrhosis.—This condition will be dealt with under the two heads of (1) Hypertrophic Biliary Cirrhosis; (2) Obstructive Biliary Cirrhosis.

1. Hypertrophic Biliary Cirrhosis.—**SYNON.**: Hypertrophic cirrhosis with chronic jaundice; Hanot's disease.

DEFINITION.—A disease characterised by chronic jaundice, attacks of fever, enlargement of the liver and spleen, and usually by its occurrence in young persons. There is no gross obstruction of the large bile-ducts. Histologically the cirrhosis is more unilobular than in common or portal cirrhosis.

NATURE OF THE DISEASE.—There are different types of this disease, and transitional forms between it and portal cirrhosis occur. Moreover portal cirrhosis may become implanted on it in the late stages, and gives rise to a mixed cirrhosis. Discussion has taken place as to whether it starts as an ascending inflammation of the bile-ducts, the infection spreading from the duodenum, or whether it is a descending cholangitis due to a poison brought to the liver by the hepatic artery; the former is the more probable view.

ÆTIOLOGY.—Males between the ages of 20 and 30 are perhaps most often affected, but children are comparatively frequently attacked. A peculiar and very fatal form of biliary cirrhosis in Brahmin infants has been observed in Calcutta. Several members of the same family may manifest the disease and so suggest some common infective agent. Among children the sexes seem to be equally affected.

There is no proof that alcohol, malaria, or syphilis plays any special part in its causation; it has been noted to follow some infectious diseases such as enteric fever (Boisset), and there is reason to believe that the infection may be water-borne (Boix). The presence of fever, leucocytosis, splenic enlargement often preceding the hepatic change, and glandular enlargement, not only in the portal fissure, but sometimes in other parts of the body, make it probable that the change in the liver is the local manifestation of a general infection. A microbic origin, though probable, has not yet been proved.

MORBID ANATOMY.—The liver is uniformly and considerably enlarged and may weigh from 80 ounces to 8 pounds; except for adhesions, the surface is fairly smooth. On section the liver is firm, of a dark-green colour, and granular. The large bile-ducts, the gall-bladder, the portal vein, and the hepatic artery are healthy.

Microscopically, each individual lobule of the liver is seen to be surrounded by delicate connective tissue (*unilobular cirrhosis*); in places the fibrillar connective tissue invades the lobules (*intercellular cirrhosis*).

The connective tissue is much more fibrillar than in portal cirrhosis, but is more intimately related to the liver-cells; it contains elastic fibres, but not to the same degree as in portal cirrhosis.

In most cases where the disease has existed a considerable time there is in addition portal cirrhosis; this is a secondary lesion, and is probably due to poisons manufactured in the spleen reaching the liver by the portal vein.

The small bile-ducts show proliferation and some fibrosis around them, while the bile-capillaries often contain plugs of inspissated bile. The appearance known as new bile-ducts is prominent, but has no special significance, inasmuch as it is seen in common cirrhosis, gumma, and other destructive lesions of the liver. The cells of the liver are much better preserved than in portal cirrhosis: this is correlated with the prolonged course of the disease. The spleen is much enlarged and weighs from 15 to 40 ounces, or even more, sometimes weighing more than the liver. Adhesions around it are frequently met with. The lymphatic glands in the portal fissure are enlarged, pigmented, and oedematous; exceptionally the glands elsewhere, as in the axilla, groin, &c., are also enlarged. The alimentary canal is, as a rule, free from signs of catarrh.

CLINICAL FEATURES.—The onset is gradual with some malaise, but jaundice is probably the first thing noticed; this is permanent, and though it varies from time to time is progressive on the whole. It never becomes so deep as the 'black' jaundice of malignant disease, since, as shown by the presence of bile in the fæces, the obstruction, which is due to cholangitis of the small intrahepatic bile-ducts, is not absolute. The abdomen enlarges from the increase in size of the spleen and liver. The spleen may be found to be enlarged before the liver, and is much bigger than in portal cirrhosis. It is more prone to excessive enlargement in young children, probably from the fact that the capsule is more distensible than in later life. It may be larger than the liver (*Hypersplenomegalic biliary Cirrhosis*). The liver is very considerably enlarged, smooth and tender. There is no enlargement of the gall-bladder. There is generally an absence of the enlarged subcutaneous veins seen on the abdomen in the common cirrhosis; and ascites either does not occur or only shows itself to a slight degree late in the disease, and is then due to a secondary multilobular cirrhosis. The urine contains bile, and is somewhat diminished in amount. The blood may show leucocytosis. There is a poor state of nutrition, growth is interfered with, and in long-standing cases clubbing of the fingers may occur. The disease is very chronic, and may last seven or more years. From time to time exacerbations in the jaundice together with abdominal pain are met with. Small hæmorrhages and epistaxis are seen,

but copious hæmatemesis is infrequent. Death may be precipitated by acute infections, such as erysipelas, pneumonia, and icterus gravis; in the last event the liver shows the lesions of acute atrophy superimposed on the chronic lesion. Death may occur from increasing weakness, or from toxæmia deepening into coma.

DIAGNOSIS.—The important points in the recognition of the disease are chronic jaundice with bile in the stools, the enlarged and smooth liver, the considerable splenic enlargement, and the absence of ascites and enlarged subcutaneous veins on the abdominal wall. In this way it is distinguished from portal cirrhosis; but it must be admitted that transitional forms occur, and that hypertrophic biliary cirrhosis may towards the end come to resemble portal cirrhosis. It differs from gall-stone obstruction in the absence of typical colic, and in the considerable enlargement of the liver and especially of the spleen. From obstructive jaundice it differs in that bile is present in the fæces. Some, rather unusual, cases of syphilitic disease of the liver resemble it, but outward signs of syphilis should put the practitioner on his guard. Between prolonged cases of infective jaundice and hypertrophic biliary cirrhosis there is very little if any difference except in the degree, duration, and result.

PROGNOSIS.—Though the disease runs a course of years, it is eventually fatal. The average duration is about five years; but during its course the patient is often able to do work.

TREATMENT.—The patient should be placed in the best hygienic conditions, take a light and simple diet, especially milk, and abstain from alcohol. The bowels should be carefully regulated, and auto-intoxication prevented by calomel, salol, or other intestinal antiseptics. A course at Carlsbad, Vichy, or Marienbad should be tried. In short the treatment is much on the same lines as those of portal cirrhosis, to which the reader is referred.

ii. **Obstructive Biliary Cirrhosis** was described by Charcot and Gombault as a fibrosis spreading out from the bile-ducts as a result of obstruction to the outflow of bile. More recent experiments and observations, however, have shown that aseptic biliary obstruction *per se* only gives rise to local necrosis and atrophy of the liver-cells, as in compression of the common bile-duct in cancer of the pancreas; though when infection of the ducts is added to obstruction, as in some cases of gall-stone-obstruction, pericholangitic fibrosis may result.

Cases undoubtedly occur in which biliary obstruction is found to be associated with fibrosis of the liver; but it is undesirable to recognise obstructive biliary cirrhosis as a special morbid entity, inasmuch as (1) the change is not the result of biliary obstruction pure and simple, and (2) the clinical aspect of such cases is usually rather that of obstruction of the common bile-duct than of cirrhosis of the liver.

H. D. ROLLESTON.

LIVER, Cysts of.—Cysts other than hydatid are occasionally met with in the liver, and may reach a large size. They are mostly simple and appear to be of the nature of retention-cysts; but there is a cystic disease of the liver in which small cysts up to half an inch or an inch in diameter are scattered through the liver and likely to be

associated with a similar disease of the kidney, the exact nature of which is at present far from certain. It is of pathological rather than of clinical interest. Dermoid cysts are rare.

LIVER, Enlargements of.—ANATOMICAL RELATIONS.—The normal hepatic dullness extends upwards—in front, to about the sixth rib; laterally, in the mid-axillary region, to the eighth rib; and by the side of the spine, to the eleventh rib. The lower border of the liver corresponds in the nipple line to the lower border of the ribs; and the dullness behind merges into that caused by the right kidney. The left lobe of the liver extends across the epigastrium to the left of the mesial line; the dull sound caused by its upper border merging in that produced by the heart. The upper part of the convexity of the liver rises to a little more than an inch above the sixth rib, the lung dipping down in front, and giving rise to a modified percussion-sound; but for practical clinical investigation it is better to take the line of absolute dullness. The extent of the normal hepatic dullness from above downwards in the right mammary line is nearly four inches, and at the side about four inches and a half. In the middle line in front it extends from the base of the ensiform cartilage to about two fingers' breadth below its point. It should be remembered that the limits of the liver present, compatibly with health, considerable variation; that the organ is relatively larger in early than in adult life; that it is depressed in inspiration, and ascends in expiration; that it is somewhat lower down in the erect than in the recumbent position; and that there is temporary distension during digestion. See PHYSICAL EXAMINATION, *Plate*.

DIAGNOSIS.—There are various sources of fallacy which may lead to an erroneous conclusion as to the size of the liver. Thus, an intestine distended with flatus may get in front of the anterior border of the organ, and lead to the supposition that there is contraction, when the contrary is the case. When there is ascites to any extent, it is difficult to make out the boundaries of the liver. In this case, however, by placing the patient on the left side, so as to let the fluid gravitate in this direction, a diagnosis may often be effected; also, by suddenly pressing the finger down below the ribs, and thus displacing the fluid, one may sometimes detect the enlarged organ. A rigid right rectus muscle is liable to be taken for a tumour; to obviate this source of fallacy the patient should lie on his back with his thighs drawn up, and his attention should be diverted by conversation while the examination is being made. Sources of fallacy may exist in the liver itself, as in malformations or malpositions of the organ; or they may be outside it, either in the abdomen or chest. Malignant disease of the stomach, omentum, or pancreas; a kidney greatly enlarged or unusually movable; or fecal accumulations in the colon, may be mistaken for hepatic enlargement. The following considerations will assist in arriving at a correct diagnosis—(a) enlargements of the liver, however much they may extend beyond, generally occupy the normal site of the organ, and however irregular the surface, the usual outline may be traced; (b) such enlargements usually follow the movements of the diaphragm in full respiration. Effusion into the right pleura may be mistaken for enlarged liver, especially as this organ may be depressed by it, and so appear to extend beyond

its limits in the downward as well as in the upward direction. In pleuritic effusion, however, the dullness on percussion will vary with the position of the patient, and the upper line of dullness will in effusion be straight, or possibly a little convex downwards—in hepatic enlargement or subdiaphragmatic abscess, convex upwards. Pleuritic effusion and hepatic enlargement may, however, co-exist. Pneumothorax, emphysema of the right lung, thoracic tumours, and even extreme pericardial effusion, may depress the liver, and affect the diagnosis. See SUBDIAPHRAGMATIC ABSCESS; and ENTEROPTOSIS.

The principal enlargements of the liver are associated with the following diseases of the organ: Hyperemia or congestion; obstruction of the bile-ducts; abscess; hydatid disease; fatty infiltration; hypertrophic cirrhosis; amyloid disease; malignant growths; syphilitic disease; leucocythæmia; actinomycosis; and cystic disease. These morbid conditions of the liver will be found described under the appropriate headings. Some might be disposed to treat of hypertrophy of the liver as an enlargement of the organ, but it hardly comes within the range of practical medicine, for it but rarely occurs as a complementary restitution of new tissue in the left lobe in response to destruction of large tracts of liver-substance in the right lobe.

JAMES F. GOODHART.

LIVER, Fatty Disease of.—DEFINITION.—A disease attended by painless enlargement and diminished consistence of the liver; due to the presence of a large quantity of fat or oil in the secreting structure; and occurring in connection with phthisis and other wasting diseases, or in persons of luxurious and indolent habits, in whom there is usually an abundant development of fat in the tissues and other organs.

ÆTIOLOGY.—Fatty liver may combine *fatty degeneration* and *fatty accumulation*. Fatty degeneration is met with in association with other hepatic diseases, as acute atrophy, amyloid disease, cancer, and phosphorus-poisoning. It is with fatty infiltration that we are here more particularly concerned. The fat may come either from within or from without the body. The former case is illustrated when the greater part of the fat of the tissues and organs is absorbed, as in the emaciation of advanced phthisis. A fatty liver exists in about one third of the cases of this disease, and is met with much more frequently in females than in males. Fatty infiltration of the liver also occurs in connection with other wasting diseases, and is not infrequent in patients who have been long bedridden. When fat is introduced from without, the affection of the liver is associated with development of fat in other organs and in the tissues generally. Persons thus affected are usually given to undue indulgence in eating and drinking: to eating not only too much food, but food rich in oil and fat, and drinking beer, or other alcoholic beverages, to excess. Want of exercise of mind and body, a heated atmosphere, and luxurious habits, materially assist in determining the affection. In illustration of this cause may be adduced the oft-cited experiments of Magendie, who induced very fatty livers in dogs by feeding them exclusively on butter; and also the production of the *foie gras* in geese, by penning them up in a heated atmosphere and cramming them.

ANATOMICAL CHARACTERS.—In fatty disease the liver is more or less enlarged, but seldom to any great extent; the surface is smooth; the borders are rounded; the substance pits on pressure; and the organ is either of pale yellow or drab colour, or, when partially affected, has a mottled appearance. A portion placed in water floats, showing a diminished specific gravity. On cutting into the organ the knife is greased; and a greasy stain is imparted to blotting-paper applied to the cut surface. Under the microscope in slighter grades, fat-granules and globules are seen to be limited to the outer zone of the lobules in the vicinity of the portal vessels; but in advanced cases every cell will be found to be filled either with separate globules, or with a single large drop of fat. In less extensive infiltration the liver may show a mottled surface, the fat being collected in the outer or portal zone, sometimes in the intermediate or arterial zone, the area of the hepatic veins being free. Fat in limited quantity is always present in the human liver, so that the term 'fatty' can only be applied when it is in excess.

SYMPTOMS.—In the lesser grades of fatty liver there are no distinctive symptoms, either objective or subjective. When the affection is more pronounced, percussion will indicate more or less enlargement, usually in the downward direction; and palpation may detect a rounded border and diminished consistence, and will, at any rate, determine that the organ is not unduly hard, has no irregularity of surface, and does not differ materially in shape from the healthy liver. There is seldom, if ever, any pain—at most, in marked cases, a sense of tension and of uneasiness on lying on the left side. Jaundice is a rare event; and ascites and enlargement of the spleen cannot be classed as symptoms of the disease. In cases of fatty infiltration, dependent on luxurious habits, as regards diet, &c., there is usually more or less development of fat in other organs, as well as in the omentum and subcutaneous cellular tissue. In cases where the liver is much enlarged, and there is much abdominal fat, the upward pressure may interfere with the action of the diaphragm, and cause, especially after meals, embarrassment of breathing. Advanced cases may be associated with irregularity, generally sluggishness, of the bowels; more or less dyspepsia; and loss of appetite; though these symptoms are not characteristic of the condition.

DIAGNOSIS.—The enlargement of the liver, with preservation of its normal shape, without hardness or irregularity; the absence of pain, jaundice, ascites, or enlargement of the spleen; and its association either with the emaciation of phthisis or other wasting diseases, or with the habits of the gourmand and general development of fat in the body, will usually enable us to distinguish fatty from other hepatic diseases.

PROGNOSIS.—The prognosis of fatty disease of the liver is affected by the associated condition of the patient, and will, of course, be unfavourable in phthisis.

TREATMENT.—The general therapeutical indications in fatty liver resulting from luxurious habits of living, point to reform in the direction of diet, air, and exercise. Rich, oily, and fatty articles of food are to be avoided; while sugar and starch should only be taken in small quantities. Beer, in all forms, is objectionable, and so also is every other form of alcohol, except, perhaps, small quantities of

light Bordeaux with meals. Exercise, either on foot or horseback, should be had recourse to daily, but must be regulated according to the soundness of the heart and circulation. Free exposure to pure air, and avoidance of heated rooms, are desirable. The functions of the skin must be promoted by adequate clothing, and by the use of the bath, or by sponging with soap and warm water. The bowels must be attended to, and dyspepsia met by antacids and vegetable bitters. The Carlsbad waters—the warm Sprudel especially—are indicated, being supposed to act upon the redundant fat.

JAMES F. GOODHART.

LIVER, Functional Disorders of.—**DESCRIPTION.**—The liver being the largest gland in the body has a number of extremely important functions connected with the processes of assimilation, nutrition, and metabolism.

The satisfactory discharge of these functions may be impeded or interfered with by a number of different causes and with very various results. A condition of hepatic inadequacy or insufficiency, usually partial and not manifesting itself equally on all the functional activities of the organ, is thus of frequent occurrence. The conception of functional disease of the liver implies (1) that there is no structural change in the organ, and (2) that there is no cause for the disturbance of function except in an inherent want of energy and power in the organ, in other words, that the functional disturbance is primary in the liver and not secondary to morbid processes elsewhere.

As far as our knowledge goes at present there is little proof that primary functional disease, as above indicated, forms a definite morbid entity. It is true that a multitude of symptoms and diseases have in the past been referred to this cause with the result that the lay mind has become imbued with and attracted by the vague conception of functional disorders of the liver; it is undoubtedly true that in practice the vast majority of cases formerly so described admit of more intelligible explanation. The nature of the essential factor may vary: thus (1) in some instances it is temporary morbid change in the liver, such as congestion, cloudy swelling and parenchymatous degeneration of the liver-cells, or transient inflammation; (2) in many cases the liver suffers from the action of toxic bodies manufactured in the intestines or elsewhere; (3) sometimes there is definite structural change, as, for example, in the early stages of cirrhosis or inflammation of the bile-ducts; (4) while in other instances, conditions either due to general perversion of metabolism, such as lithæmia and gout, or arising elsewhere as gastro-intestinal dyspepsia, have been regarded as the manifestations of a primary hepatic inadequacy.

The consideration of the subject will be facilitated by briefly referring to the following important functions of the liver.

The glycogenic function of the liver may be considered as comprising (a) the storing up, in the form of glycogen, of sugar derived from the alimentary canal, and (b) the formation and supply of sugar to the economy. The liver may fail in stopping sugar brought to it, and alimentary glycosuria will result, or the sugar-forming function may run riot and diabetes mellitus develop. The mechanism by which the glycogenic function of the liver is disturbed is a question of much interest and is discussed elsewhere. See DIABETES MELLITUS.

In the metabolism of *proteids* the liver plays an important rôle in supervising the formation of urea. It has been generally assumed that disturbance of this function of the liver results in the production of uric acid at the expense of urea; Murchison indeed based his well-known description of lithæmia (*see URIC-ACID DIATHESIS*) on the supposition that from inherent defect in the liver its functions are disturbed by insufficient causes, and that as a consequence uric acid was poured out in larger quantities into the circulation. That disturbance of hepatic function may play some part in excessive production of uric acid is very probable; but it must be remembered that uric acid may be manufactured elsewhere in the body from nuclein, and possibly also in the kidney. Uricæmia should therefore be regarded as the outcome of a general perversion of proteid metabolism, and not as solely due to disordered hepatic activity. Further, disturbance of the proteid metabolism in the liver is in many instances so manifestly due to structural changes, or to morbid processes, such as dyspepsia and auto-intoxications elsewhere, that it is unsafe to assume a primary disturbance of this function as a common event.

The secretion of bile by the liver was formerly thought to be diminished by a 'torpid' state of the organ in the condition known as *biliousness*. Diminution of the flow of bile into the intestine depends in such cases on catarrhal swelling of the mucous membrane of the bile-ducts. In some instances there may be an incipient catarrhal jaundice from the spread of inflammation from the duodenum to the common bile-duct; while in other cases there is probably catarrh of the small intra-hepatic ducts, set up by irritating and toxic bodies absorbed from the intestinal tract, and often associated with congestion of the liver. It is quite in accord with this explanation that the most satisfactory treatment for this disorder is directed to the intestine, such as Blue-Pill and Mistura Sennæ Composita, and not to stimulation of the liver. *See GALL-BLADDER and BILE-DUCTS; and JAUNDICE.*

By its *antitoxic function* the liver arrests poisons reaching it from the alimentary canal, and prevents their passage into the general circulation. In doing so it may suffer damage, as is seen in an extreme degree in phosphorus-poisoning; and as a result this antitoxic or protective function fails and poisons escape into the blood. Many of the symptoms popularly ascribed to 'torpid liver,' such as headache, drowsiness, irritability and depression, *muscæ volitantes*, and bitter taste in the mouth, are due to the flooding of the circulation with toxic bodies. In well-marked structural change in the liver, as in acute atrophy and in the later stages of cirrhosis, the failure of the antitoxic function of the liver results in extreme hepatic toxæmia or cholemia, which has many resemblances to uræmia. *See JAUNDICE.*

The foregoing remarks have shown that functional disorders of the liver are practically either manifestations of definite structural change in the liver and bile-ducts or secondary to morbid processes elsewhere.

The symptoms which have, many of them, been already mentioned in the sections on the functions of the liver may now be briefly summarised as follows: Distaste for food, dyspepsia, and flatulence are due to gastro-intestinal disturbance, set up by poisonous or unsuitable food. The icteric tint of

the conjunctivæ, the muddy complexion, and the diminished quantity of bile in the stools are accounted for by slight catarrhal obstruction to the outflow of bile from the liver. Piles, the feeling of weight in the right hypochondrium, and the disordered action of the bowels are due to congestion of the liver, brought about by the advent of food-products either poisonous in quality or excessive in quantity. This state of hepatic congestion is readily set up in patients who have suffered from malarial fevers in hot climates, such as India. Headache, giddiness, irritability of temper, malaise, other nervous symptoms, and the bitter taste in the mouth are due to poisons absorbed from the alimentary tract getting into the general circulation from failure of the antitoxic function of the liver. *See BILE, Disorders of.*

The treatment of secondary functional disturbance is to discover and treat the primary cause. Often it is to be found in the alimentary canal, and can be removed by blue-pill and senna-draught. Mercury drives out the bile from the gall-bladder and ducts, and by sluicing out the common bile-duct will have a beneficial effect on catarrh of that duct. Purgation removes the products of fermentation and prevents auto-intoxication, while at the same time it reduces the hepatic congestion.

Plenty of water should be taken, so as to stimulate the action of the kidneys, while a milk-diet free from irritating or stimulating ingredients should be prescribed. Alcohol should be rigidly tabooed while the symptoms last, and only taken sparingly in the intervals. The symptoms due to congestion, early cirrhosis, &c., must be treated on the lines laid down elsewhere. After the symptoms have subsided fairly active exercise, plenty of fresh air, and a carefully regulated diet should be enjoined. As pointed out above, the rational treatment includes that of the primary cause. *See DIGESTION; STOMACH; and the other articles on the LIVER.*

H. D. ROLLESTON.

LIVER, Gangrene of.—*See LIVER, Abscess of; and Inflammation, Acute, of.*

LIVER, Hydatid Disease of.—**ANATOMICAL CHARACTERS.**—The liver is the organ most frequently affected with hydatid disease. There is usually but one cyst, but there may be two, three, or more; and the size of the cyst may vary from that of a pea to that of a child's head. The cysts may exist in either lobe of the liver, but they are more frequent in the right; and they may be attached to the upper or under surface, or project from the border, or lie buried in the substance of the gland. This is more or less modified in form, and increased in size, according to the magnitude and site of the cyst. When the cyst is small and deep-seated, there will be no appreciable change in the liver, and the disease may be latent for years. When, however, the cyst is very large, it, with the liver, constitutes a tumour, which may encroach upon the thorax, and also fill a great part of the abdomen. Pressure of the cyst may induce atrophy of a portion of the liver, but, at times, hypertrophy is the result. The bile-ducts have occasionally been found to be obliterated, or a communication to have been effected between them and the cysts. When at the surface of the organ, the cysts as they enlarge may induce inflammation and thickening of the peritoneum, and adhesion to neighbouring structures.

SYMPTOMS.—A hydatid cyst, when sufficiently large and near the surface, generally exhibits itself as a tumour of variable size, situated either in the right hypochondrium or in the epigastric region; evenly globular in its early stages; firm, resisting, yet elastic, and, at times, with a sensation of fluctuation. Briançon and Piorry noticed a vibration or trembling—*hydatid fremitus*—which is sometimes felt when the surface is compressed gently by three fingers of the left hand, and sharp percussion made with the right hand over the middle finger. If the tumour is situated behind the liver, it will, as it develops, push this organ forwards, flatten it, and increase the area of dullness. The tumour may last for a considerable time, and go on increasing to some extent, and yet the patient may remain free from constitutional disturbance, perform all his functions well, and keep in good condition as regards flesh and strength. When, however, it has attained a very large size, it will give rise to various symptoms—to a feeling of tightness and distension; if it press upwards, to embarrassed breathing, cough, possibly more or less dullness, with diminished respiration, at the base of the right lung, and palpitation; if upon the abdominal viscera, to interference with their functions. Pain is not generally present, but in some cases there is a gnawing pain, either at the epigastrium or extending forwards from the lumbar region. (Edema of the lower extremities may occur when the tumour presses upon the inferior vena cava; or ascites may result from a similar effect on the portal vein.

DIAGNOSIS.—Hydatid tumour of the liver is not always easily diagnosed; but the characteristic features already noticed, and the maintenance (in many cases up to an advanced stage) of a good state of health, will generally point to its nature. Abscess of the liver will be distinguished by local and remote pain; rapidity of onset; the frequent antecedence or co-existence of dysentery; and severe constitutional symptoms, such as hectic fever, rigors, &c. It must, however, be remembered that hydatid cysts are liable to become inflamed and to suppurate, when the diagnosis will not be so readily made; but even so, the suppuration of a hydatid cyst is less likely to be accompanied by the acute paroxysmal fever so characteristic, if present, of abscess of the liver. Cancer of the liver will generally be marked by irregularity of surface, and often a peculiar hardness and heaviness; the presence of pain; the cachectic aspect; loss of flesh; and the rapidity of progress. Aneurysm of the abdominal aorta may form an epigastric tumour, of even spherical shape; but the pulsations, frequently very forcible, coupled probably with a bruit, audible along the course of the vessel before and behind, will determine the diagnosis. The site, the pyriform shape and the uniform size, the greater mobility, and possibly the accompaniment of jaundice, will distinguish from hydatid disease the tumour caused by a distended gall-bladder. Frerichs believed that hydatid disease of the liver is more frequently confounded with localised pleuritic effusion at the base of the chest than with any other affection. He remarks that the same signs—dullness on percussion, absence of vocal thrill, intercostal fluctuation—would be present in both cases. He rests the diagnosis on the fact that the line of dullness would present a curve which would look upwards in the one case, downwards in the other. To this sign, of uncertain presence, may be added

that the vocal vibration and resonance, although perhaps somewhat lessened, are not usually damped by pressure from below the diaphragm to the extent that is effected by fluid in the pleura.

TERMINATIONS AND PROGNOSIS.—Hydatid tumour of the liver may last for years, and be compatible with an average state of health; or, at an early or advanced period of its existence, it may terminate in one of the following ways: 1. It may, from its bulk and position, press upon and interfere with the functions of different organs. Pressure on the large venous trunks may induce ascites and dropsy of the lower extremities; pressure upon the stomach and intestinal canal may obstruct functions connected with the assimilation of food, and induce failure of flesh and strength, and ultimately death from exhaustion. 2. The tumour may contract adhesions with the diaphragm; ulcerative action through this may be set up, and either (a) discharge of the contents of the sac may take place into the pleura, and fatal pleuritis result; or (b) further adhesions and ulceration may effect communication with the lung, when hæmoptysis or pneumonic symptoms ensue, and the contents of the sac, mixed with blood or the products of inflammation, are expectorated. The disease is sometimes cured by this means. 3. A rare result is adhesion to, and ulceration into, the pericardium, with escape of contents, and rapidly fatal results. 4. Adhesion may be effected with some part of the alimentary canal, and the contents of the sac be discharged by vomiting or by stool. 5. Rupture of the sac may be caused by a blow or otherwise; the contents be discharged into the peritoneum; and fatal peritonitis result. 6. The tumour may contract adhesions with the parietes, point externally, and be opened or effect an opening by natural process, inflammation and suppuration having been previously set up in the sac. 7. Budd and Frerichs notice a possible cure from the obliteration of the sac by the formation within it of a putty-like matter, the combined result of degeneration of the cyst and its contents, and of inflammation outside the parent-cyst. These conditions are often seen *post mortem*, and are frequently associated with calcareous changes in the cyst-wall, as well as with the deposition of brown or orange biliary pigment. So generally is pigment of this kind present in various parts of these cured hydatids, that there is good ground for supposing that the rupture of minute bile-ducts into the cyst or its wall is no uncommon thing; and that, either by killing the parasite or by effecting slow inflammatory changes in the bed of the cyst, it leads to a spontaneous cure in no inconsiderable number of cases. As an illustration, although a rare one, of the extent to which the slow inflammatory changes may proceed under favouring conditions, there is a specimen in the museum of Guy's Hospital where the hydatid is surrounded by, and had no doubt determined, an enormous growth of gummatous material. 8. Communication may be effected between a hydatid cyst and one of the larger bile-ducts, and then the result will usually be fatal, although there are one or two cases recorded of recovery. 9. Similar cysts may be formed in other parts or organs of the body. 10. As a possible rare event may be mentioned communication of the sac with the ascending vena cava, escape of the contents into this, transfer of the contents to the right side of the heart, impaction in the pulmonary artery, and fatal asphyxia.

TREATMENT.—So long as hydatid tumours produce no distressing symptoms and do not affect the function of any organs, there is no pressing cause for interference. But as some of the inconvenient or dangerous symptoms mentioned in the last paragraph are likely sooner or later to occur, and as, owing to the improvements in modern surgery, interference with these cysts is now far less dangerous than it used to be, it is generally recommended that they should be treated surgically without much delay. The special forms of inconvenience which would make operation necessary are the size of the cyst and the presence of adhesions to, or mechanical interference with, surrounding parts. Any of these may cause disturbance of the functions of neighbouring viscera or induce ascites from pressure on the portal vein. The symptoms which are indicative of approaching danger are those of acute or chronic suppuration, or an obvious extension of the cyst upwards, rupture into the pleura being an occasional and extremely dangerous complication, and one which, if not actually fatal, is likely to leave a completely collapsed lung and an incurable pneumothorax.

It was formerly recommended that hydatids of the liver should be punctured with a fine trocar and cannula; and the reports of Australian and other surgeons render it certain that a number of cases may be cured in this way. There are, however, certain dangers in connection with this method of treatment which appear to be unavoidable, especially those arising from leakage of the contents of the cyst into the peritoneal cavity. It is also recognised that by no means all the cases can be cured in this way. It is therefore usually recommended in this country that the cyst be drained or removed without recourse to previous tapping. In a case where a cyst of some size impinges on the abdominal wall, the operation consists in making an incision over the most prominent part of the tumour, or, if possible, at a somewhat higher level. The cyst-wall is grasped with volsella and the needle of an aspirator is plunged into it. As the fluid is drawn off the cyst collapses and its walls can be drawn into the wound. At this period the wall may be freely incised, and the daughter-cysts together with the endocyst removed. The cyst-wall is then stitched to the skin of the abdominal wall and a drainage tube inserted. In the case of smaller cysts it may be necessary to perform the operation in two stages: in the first, the tumour is exposed and means are adopted to ensure the presence of adhesions; in the second the cyst is opened and treated as in the case of the larger cyst. Occasionally it is found possible to dissect out the whole cyst; and this is especially the case if the wall has become calcified. Under these circumstances this is the only treatment to pursue, and it usually presents no great difficulties.

Treatment by free incision and drainage is not altogether free from danger. Serious hæmorrhage may occur, but this is rare. The greatest risk is in connection with the possible occurrence of septic changes after the operation. If this should happen when the cyst is large, the patient's life will be in danger. It is therefore of the greatest importance that throughout the treatment the strictest attention should be paid to all the antiseptic arrangements. When operating it is important to recollect that hydatid cysts may project upwards at least as far as the second rib in front; and that cysts, not

necessarily of large size, may be found in the right iliac fossa or touching the spleen in the left hypochondrium.

If it be decided to adopt the method of simple puncture, the least dangerous method is to employ the aspirator, but if this be done it is very important only to exhaust a small quantity of air from the bottle before the needle is inserted, and afterwards to work the pump very slowly, otherwise the daughter-cysts will be sure to be drawn against the eye of the cannula, and the escape of the fluid thereby prevented. Such methods of treatment as the injection of the cyst with some stimulating fluid, or the gradual opening of it by the application of caustics, are now obsolete.

Treatment by medicinal agents administered internally has proved entirely useless.

JAMES F. GOODHART.
R. J. GODLEE.

LIVER. Hyperæmia (Passive) of.—SYNON.: Passive Congestion of the Liver; Nutmeg Liver.

DEFINITION.—Uniform enlargement of the liver, with preservation of its normal shape; caused by over-distension with blood, the result of mechanical obstruction to the return of blood to the heart. It may be attended by a sense of fulness and oppression in the right hypochondriac and epigastric regions, and often by slight jaundice.

Passive congestion is due to interference with the return of blood from the liver through the hepatic vein and inferior vena cava to the heart. Such interference may be due immediately to dilatation of the right heart, with affection of the tricuspid valve; to obstruction to the circulation in the course of the pulmonary artery, caused by different diseases of the lungs; or, farther on, in the line of the circulation, to disease of the mitral or aortic valves. The affections of the lungs which interfere with the pulmonary circulation are either acute, as pneumonia, and then the hepatic hyperemia may pass off with the disease; or they are chronic, as emphysema and fibroid disease, and then the hyperemia will persist. Passive congestion of the liver may result from direct obstruction to the flow of blood by the pressure on the inferior vena cava of aneurysmal or other tumours, or of the cicatricial tissue formed as a result of mediastino-pericarditis. Mere weakness of the heart's action often keeps up a certain amount of passive congestion.

ANATOMICAL CHARACTERS.—A hyperæmic liver is increased in size about equally in all directions; its resistance is also increased; its peritoneal investment appears distended and shining. On making an incision blood oozes out freely, and the cut surface is dark red—either uniformly so, or spotted with intervening lighter spaces. The central vessels of the lobules—the hepatic veins—are engorged; and when persistently so, as in disease of the mitral valve, the cut surface gives the characteristic appearance to which the term *nutmeg liver* has been applied. The dark centres contrast with the pale circumference of the lobules, the light and dark parts being clearly defined, but varying according to the section; the surface gives an appearance which resembles closely that of a cut nutmeg. The central dark spots result from distension of the hepatic veins, and deposition of bile-pigment in the adjacent hepatic cells, which are more or less atrophied by pressure; the lighter spaces correspond to the circumference of the lobules, the light colour

being emphasised, in some cases, by the presence of fat in the cells at the circumference. In addition to the atrophy of the cells by pressure of dilated hepatic veins, there may be hypertrophy of the interlobular connective tissue, with lymphoid bodies scattered through it. The so-called chronic congestion of the Anglo-Indian is described on p. 897.

SYMPTOMS AND SEQUELÆ.—In slight cases of hyperæmia the liver does not extend much beyond its normal limits, but it may perhaps be felt below the borders of the ribs and across the epigastrium. In severe cases, and especially in passive hyperæmia from obstructed circulation, the organ often attains a considerable size, and is found, on percussion, to extend upwards into the mammary region, downwards nearly to the umbilicus, and across into the left hypochondrium. Its resistance is generally increased, and, in old-standing cases, such as the local condition occurring as the effect of tight-lacing, it may even be hard enough to create the suspicion of malignant disease. There is often, especially in acute cases, tenderness on pressure. The patients do not complain of pain, but of a sense of oppression and fullness in the right hypochondriac and epigastric regions, and of uneasiness from the pressure of clothes, or on lying on the left side. In the most acute cases there is marked functional and general disturbance; a furred tongue, nausea, vomiting at times of bile, bilious diarrhoea, sallowness of complexion, or some amount of jaundice. In certain cases the bile seems to be peculiarly acrid, and causes much griping and distress as it passes downwards, and smarting as it is voided. The urine is high-coloured, and loaded with urates. The patients often complain of headache, are irritable and depressed in spirits, and feel languid and drowsy. When jaundice is present in any marked degree, there is probably catarrh and transient obstruction of the smaller bile-ducts.

There are symptoms indicative of the pulmonary or cardiac affection on which it depends. The complexion is more or less dusky, and there is a certain amount of lividity mixed with the jaundiced hue. The liver may be found to vary in size in accordance with the variations in the conditions which give rise to it. It may sometimes pulsate. Ascites may now become a prominent symptom, while the general dropsy resulting from associated heart-affection may be yielding to treatment.

DIAGNOSIS.—It is only when the congestion is chronic, and the liver hard and resistant, that an erroneous diagnosis may be made; but the previous history of the case, the discovery of a sufficient cause, and the associated general symptoms, will lead to a right conclusion. It might be mistaken for albuminoid disease, and indeed the liver may be really undergoing this degeneration as well.

PROGNOSIS.—This will depend upon the cause, and as it is largely due to disease in the chest, it will be influenced by the nature and stage of such disease.

TREATMENT.—If, as is often the case, the cause is irremediable, treatment can only be palliative. This should include rest; restriction to a bland fluid diet; and the free action of the bowels by a dose of calomel, followed after a few hours by a saline aperient, either in the form of a draught, or of one of the more active mineral waters. A single sufficient dose of calomel, four or five grains, will often rapidly relieve attendant gastric irritation and vomiting, and ensure a free downward discharge

of bile. Should there be much tenderness on pressure over the liver, the application of sinapisms or turpentine stupes, followed by hot poultices, will be beneficial. The portal system must be kept relieved by a dose of Pullna or Friedrichshall water, or by a saline draught, every or every other morning, preceded the previous night by some mercurial preparation, if the secretion seem to require this. Resin of podophyllum may be advantageously substituted for mercury in some cases, but it is uncertain in its action. Its griping effect is counteracted by the addition of a little extract of hyoscyamus or half a grain of extract of cannabis indica, and its action will be quickened by adding a little compound colocynth pill. Other remedies too appear to be sometimes useful, such as taraxacum (aperient and alterative); combined, in subjects of gouty habit, whose urine is more or less charged with lithates, with bicarbonate of potassium, or, in other cases, with diluted nitro-hydrochloric acid. This acid, in torpidity and chronic enlargement of the liver, is one of our most effective remedies. It appears to act by altering and promoting the biliary secretion, and by improving the tone of the digestive organs. It may be used both internally and externally. Ten to twenty drops of the diluted acid of the Pharmacopœia may be given two or three times daily, combined with taraxacum, quinine, or other drug, according to indications. The external use of the acid has been advantageously had recourse to at the Seamen's Hospital for years, either in the form of compress over the abdomen, or by sponging the surface of the body. The fluid is prepared by adding eight ounces of the diluted acid of the Pharmacopœia to a gallon of water at about 98° F. Earthenware or wooden vessels should be used; and all sponges and towels must be well washed in cold water after use, or they will be destroyed by the acid. At times the external use of the acid causes purging; and sometimes severe irritation of the skin. Chloride of ammonium and iodide of potassium must be mentioned as drugs that have been found serviceable in reducing livers enlarged by chronic congestion.

In the hyperæmia resulting from disease of the mitral or aortic valves, or from chronic pulmonary changes, it is sufficient to remark that the chest-symptoms will often be most effectually relieved by treatment directed especially to the liver.

Much benefit will result in many cases of hyperæmia from a course of mineral waters at one of the German or English spas, Marienbad, the springs of Carlsbad, Kissingen, Cheltenham, Harrogate, &c. A more detailed notice of the different spas and waters in relation to hepatic affections will be found under other articles. See LIVER, Functional Disorders of; and MINERAL WATERS.

JAMES F. GOODHART.

LIVER, Inflammation of (including ACTIVE HYPERÆMIA).—SYNON.: Hepatitis; Acute Congestion of the Liver; Fr. *Hépatite Aiguë*; Ger. *Acute Leberentzündung*.

In this article hepatitis and acute congestion of the liver will be treated as one disease, suppurative hepatitis being dealt with separately (see p. 882). It may be said that hepatitis is only an advanced stage of acute hyperæmia or congestion, and this is probably what is meant when cases are returned, as they often are in Army returns, as hepatitis.

Active hyperæmia of the liver is divided into two

varieties, acute and chronic, the latter form, the so-called 'tropical liver,' being due to a persistence of the causes which lead to the acute condition.

ÆTIOLOGY.—Though far from unknown in cold countries, acute congestion of the liver is especially common among Europeans who have migrated to the tropics, and there continue their old habits of life under very different climatic conditions. There is also a form of hyperæmia of the liver which is purely physiological, occurring especially in Europeans near the beginning of their life in the tropics. It is due to the diminished lung-metabolism, the great heat, the less active habits (induced by the heat), and the rich food, for the appetite is stimulated rather than reduced on first arrival in a hot country. This state of physiological hyperæmia may remain stationary, but is very prone to pass into a pathological state from some slight exciting cause, such as a cold bath, a wetting, the fitful pulling of a punkah during sleep, a sudden storm at night, a surfeit of food, an excess of wine taken at a dinner-party, or a chill, especially after polo or tennis. Such causes can produce a very acute congestion or even a hepatitis. If such exciting causes are repeated, or if a patient persists in dietetic or alcoholic excess, or does not take sufficient regular exercise, this state passes gradually into that of chronic engorgement.

Another cause which renders the affection common in warm countries is the influence of repeated attacks of malarial fevers. All attacks of malarial fever lead to the congestion of the internal organs, especially of the liver and spleen, and if these attacks are frequently repeated and not properly treated, they result in a well-marked form of hepatic cirrhosis, ultimately followed by dropsy and all its attendant evils. See *MALARIA*.

The following figures show how much more common hepatitis and hyperæmia of the liver are among Europeans in India than among the natives, thereby also showing the influence of the dietetic and other climatic conditions as factors in their causation. In ten years (1889-99) there were 12,461 admissions for hepatitis and congestion of the liver in the British Army in India, of which the average strength was 68,950, while in the Native Army (average strength 127,000) there were only 1,826 admissions for these complaints; that is, while the British ratio was 18 per thousand, it was only 1·4 among the native sepoys. As regards the influence of alcohol, while there is no doubt that alcoholics suffer largely from hepatic congestion, moderate drinkers and even total abstainers are by no means exempt from this disease.

The life of the British soldier in the long Indian day is in itself conducive to such attacks—parades at early dawn, work over by 9 A.M., confinement in a crowded barrack, with the hot air pouring in through the too often open door, the feebly pulled punkha, the heavy midday meal, the long hours spent lying on a bed, and the evening parade, followed by great thirst, too often quenched by large draughts of beer. There can, however, be no doubt that the frequency of these affections in India has been much reduced during recent years. Bryden's statistics for hepatitis and liver-abscess in the years 1871 to 1876 gave a ratio of admissions to hospital of 55 per thousand; whereas the figures for the same affections for the past ten years (1890-99) show a very much reduced ratio, viz. only 18 per thousand. This change for the better is to be attributed to the improvements in the habits of Euro-

peans in India during the past quarter of a century. The Anglo-Indian of to-day is, as a rule, a temperate man, and probably consumes in a day much less alcohol than the average city-man at home. His chief drink now is whisky, well diluted with aerated water and a block of ice; light German beer, light French or Italian wines, very different in potency from the brandy and heavy ports and sherries of a generation ago.

The same influences have been at work in amending the surroundings of the British soldier in India. More attention is paid to the cooking and the quality of his food; the coffee-shop has to a large extent replaced the canteen; rational amusements are now provided which supply him with regular exercise; and the barracks are airy and better ventilated, and provided with punkhas. The Army Temperance Association has made great strides among the rank and file. Nearly every regiment in India has its own 'soda-water' machine, and aerated waters are sold to the soldiers at an almost nominal cost. That these hepatic affections are much rarer among women and children (European) in India is no doubt due to their quiet indoor life and their lesser indulgence in excess of food and alcohol.

No one has yet given a satisfactory explanation of the lesser prevalence of liver-diseases among Europeans in the West Indies. The British civilian or soldier does not change his habits when he goes to the West rather than the East Indies, yet there seems to be little doubt that liver-affections are much less frequent there than in the East Indies. It may be that the oceanic climate of the West Indian islands has some influence; the sea-breezes temper the great heat; and in no part of the West Indies is the heat so fierce as that of the Punjab, Central India, or Madras.

In the Army returns many cases are returned as hepatitis which are either acute hyperæmia or an incipient stage of hepatic suppuration. Some writers of the French school who carry the dysenteric origin of liver-abscess too far, and ignore its comparatively rare occurrence among natives, among whom dysentery is so common, see a 'congested state of the liver' in every autopsy on a case of dysentery. This the present writer cannot admit, nor can he agree with Kelsch and Keiner in tracing every congestion of the liver to either dysentery or malaria.

ANATOMICAL CHARACTERS.—The liver is uniformly enlarged, and its capsule distended and shining. On incision blood, usually of a deep red colour, oozes out freely. The hyperæmia is not limited to the centre of the lobule. Microscopically the lobules may show signs of fatty change.

SYMPTOMS.—The symptoms vary within wide limits, especially with the degree of acuteness. In slight congestion a smart attack of diarrhœa, with copious dark-coloured stools, is nature's own method of relief, and often the only important symptom. In the more severe cases there is probably pain in the side, and a feeling of weight or fulness in the hepatic region, felt especially when lying on the left side. The degree of general disturbance varies much. The tongue is furred, or has a brown line down its centre, and is indented at the edges. A bitter taste in the mouth is often complained of, the urine is scanty and high-coloured, and the bowels are constipated or irregular. There may be one or two degrees of pyrexia. A pain in the right shoulder, headache, and depression of spirits are

common, and drowsiness after meals is sure to be noted. In the still more acute cases (those usually called hepatitis) all the symptoms are more pronounced, the temperature rises to 102° F. or thereabouts, palpitations are not uncommon, and the patient may complain of having had bad dreams.

In the chronic form, the so-called 'tropical engorgement of the liver,' the liver is uniformly enlarged and painful on pressure. The superficial epigastric veins are often dilated (indicative of portal obstruction), the patient probably has piles, and, in some cases, the hepatic facies is well marked. The patient is either of the thin and sallow type or is fat and flabby, the eyes have dark rims round them, the sclerotics are yellow, the complexion is of an earthy muddy type, and on the malar eminences and nose there may be enlarged and tortuous veins. The hepatic pain is persistent and pronounced, local spots of tenderness may be made out at the costal border, especially at the cartilages of the eighth or ninth ribs. Another painful spot may be found a few inches below the angle of the right scapula. The bowels are costive or irregular, the patient is mentally depressed, irritable, or even stupid, the tongue is usually much coated, the appetite is capricious, the patient being seldom capable of enjoying a good breakfast, but often able to indulge freely at other meals. Sleep is fitful and generally bad. Nausea is often complained of in the morning, piles are generally present—a 'fit of the piles' often giving temporary relief to the general symptoms. A permanently moist condition of, or an itching at, the anus is a very common complaint. Irregular slight rises of temperature may occur at night. The circulation is not good, and there may be chilliness or coldness of the legs. Headache, giddiness, and dyspeptic symptoms make up the clinical picture. The irritability of the patient is proverbial.

DIAGNOSIS.—The diagnosis is generally easy, the pain in the hepatic region, the enlargement of the liver, the intestinal disturbance, and the state of the tongue, generally sufficing to make it clear. In all cases attended with fever the possibility of an abscess of the liver must not be forgotten, and in some such cases time alone will clear the matter up. Pneumonia at the base of the right lung and pleurisy have been mistaken for acute hyperæmia and *vice versa*. Intercoastal neuralgia has been diagnosed in some cases of hepatic congestion, and inflammation of the gall-bladder has been mistaken for it. In perihepatitis there is usually some neighbouring organ affected, and in any case the pain in perihepatitis is usually very acute and localised. The fever at first is often looked upon as malarial, and treated as such. The possibility of abscess must ever be borne in mind, and careful inquiry made as to a previous history of former or recent dysentery.

The chronic form of congestion or 'tropical engorgement' is seldom mistaken for anything except gout, which may also be present. The early stages of malignant disease may be mistaken for mere congestion.

PROGNOSIS.—The prognosis of acute uncomplicated congestion is good. Out of 12,419 cases admitted into military hospitals in India only 42 died, a case mortality of only .3 per cent. The prognosis of acute attacks depends much on the patients themselves, and their ability to make the necessary changes in their habits of life; that of chronic congestion is not so good, since, though few

succumb to it directly, many go from bad to worse and die of some intercurrent disease or remain permanent invalids.

TREATMENT.—The lines both for prevention and treatment are indicated in the section on ætiology, viz. moderation in eating and drinking, and avoidance of chills. Regular exercise is essential, and for middle-aged men no game is better than golf, which can nearly always be had in the tropics. The public have a firm belief in a supposed necessity for violent exercise, to 'shake up the liver,' as they say, but an inflamed liver, like any other organ when inflamed, needs rest. A morning ride is excellent, but the same cannot be said for a violent game of polo followed by sedentary work in an office for the rest of the week.

For an *acute attack* rest in bed, abstention from alcohol, and light diet are essential; hot curried dishes and excessive tobacco-smoking are to be avoided. As regards drugs, the indication is to relieve the overloaded portal system by free saline purges. For this purpose Carlsbad Sprudel salts, Apenta, Hunyadi János, or other mineral waters are prescribed, to be taken in the early morning; the time-honoured blue-pill and colocynth at night is often invaluable. Formerly abstraction of blood by means of leeches was much in vogue, and was undoubtedly effective. Twenty grains of chloride of ammonium is still frequently prescribed. For *local treatment* the hepatic region may be painted over with the tincture of iodine, or a mustard-plaster may be applied, the latter often giving prompt relief. In *malarial hepatitis* treatment must be directed to the liver and spleen. The so-called 'spleen-mixtures' in India generally contain aperient salines with quinine and iron.

The chronic condition of tropical engorgement needs careful treatment. Rigid moderation above all is essential. The diet and daily supply of alcohol must be cut down with a rigid hand. For acute exacerbations the treatment must be as is indicated above for acute attacks. The 'hydropathic' belt should be worn for ten days or so, or till the skin gets red and raw. It can be made from a piece of swansdown cloth one foot broad, and long enough to go round the waist. It is applied round the waist over the hepatic region, covered with a piece of waterproof sheeting, and kept in its place by a broad bandage. Nitro-hydrochloric acid is often prescribed in these cases, but it is doubtful if it is of use in cases associated with intestinal catarrh. Attacks of diarrhoea are best treated by bicarbonate of sodium, and salol, or small doses of mercury and chalk. Bismuth and pepsin are useful, but astringents are generally harmful.

Patients suffering from tropical engorgement of the liver should be sent to Europe as soon as possible, and should undoubtedly undergo a course of treatment at Carlsbad. In the cold weather in India an artificial course of Carlsbad salts can be tried. A change to Kashmir will often be possible for those who cannot afford to get to Europe, or even the hill-stations in the dry weather may be tried. If the patient is a good sailor, a short sea-trip is an excellent change. If, however, the chronic engorgement is of long standing, every effort should be made by the patient to undergo the full course of treatment at Carlsbad.

W. J. BUCHANAN.

LIVER, Malformations of.—Abnormalities in the form of the liver are not common, and are

more often acquired than congenital. The following are some of the most frequently observed malformations that are *congenital*, and due to some original defect: A more or less quadrangular liver; a rounded liver; reduced proportions or total absence of left lobe; prolongation of the left lobe in the form of a narrow tongue-like process towards the region of the spleen; abnormal grooving of the surfaces of the liver; extreme depth of normal fissures. Another occasional variety of hepatic malformation consists in extensive lobulation, and the existence of one or more additional small lobes—a condition met with in the livers of rodent animals. *Acquired* malformation may be due to compression of the organ by tight-lacing, straps, and other means. In cases of hepatic malformation caused by tight-lacing, the upper part of the right lobe may be traversed from side to side, either by a single very deep furrow, or by two or more shallow depressions with elevated transverse folds of liver-tissue between them. In those instances in which the constriction has been so applied as to 'fold up' the liver, and to increase its convexity from before backwards, a single deep transverse furrow is often formed on the under surface of the right lobe. The hepatic tissue in the furrows atrophies; the fibrous tissue is often increased, especially just under the capsule.

A furrowed condition of the upper surface of the liver is often observed, in which elevated folds of hepatic structure, with corresponding depressions, take a direction from before backwards. This furrowing occurs in cases of obstructed respiration, and is held to be due to the pressure of the lower ribs on the liver, which organ is more or less fixed by depression of the diaphragm and by the strongly contracted abdominal muscles.

In 1888 Riedel directed attention to a partial malformation of the liver occasionally observed in association with cholelithiasis and a distended gall-bladder. In this condition the right lobe of the liver is extended downwards in the form of an attenuated tongue-shaped process (*floating lobe*).

W. JOHNSON SMITH.

LIVER, Malpositions of.—Abnormalities in the position of the liver are much less rare than abnormalities in its form. The more frequent forms of *congenital* displacement include—Lateral transposition, the liver being found on the left instead of the right side of the abdomen; eventration, the organ being exposed in front of the abdomen of a fetus; the presence of more or less of the liver in the chest, through congenital deficiency of the diaphragm. In *acquired* displacement the liver may be either depressed or elevated, some rotation of the organ on its transverse axis taking place in an opposite direction in each case. Depression may be caused by pressure from above, as by effusion in the right pleural cavity, and probably to some slight extent by considerable pericardial effusion, or cardiac hypertrophy. Elevation of the liver, which takes place more frequently, may be due to pregnancy, ascites, or the presence of some large abdominal tumour. Curvature of the spine, whether lateral or angular, usually gives rise to some change in the position of the liver. In Pott's disease the organ is often forced downwards towards the crest of the right ilium.

By tight lacing both the position and the form of the liver may be altered. The organ may be forced

downwards, and at the same time so twisted on its transverse axis that its convex surface looks directly forwards, and its concave surface directly backwards. See LIVER, Malformations of.

A liver quite free from structural disease may, under certain conditions of rare occurrence, be capable of considerable downward displacement within the abdominal cavity (*hepatoptosis*). This displacement of the liver is invariably associated with some deformity, the organ, according to Treves, being flattened out, 'so that the anterior surface is elongated at the expense of the superior surface, and the inferior surface at the expense of the posterior.' There is much difference of opinion as to the aetiology of this condition. It occurs in most instances in women who have had many children, or who have been weakened by much hard work, and is probably due to a relaxed state of the abdominal walls, associated with elongation of the suspensory ligaments of the liver. See ENTEROPTOSIS.

W. JOHNSON SMITH.

LIVER, Morbid Growths of.—Hydatid disease, cysts of various kinds, actinomycosis (p. 22), syphilitic gummata, angiomas, adenomata, and malignant disease in its various forms are each and all to be met with. Some of these are described under their own special headings; of the others, cavernous angioma need not detain us, as it has no clinical significance. It is found *post mortem* in the form of isolated patches of spongy naevoid tissue in the substance of the liver. Adenomata, in like manner, are small and rare, and might perhaps be better designated as an erratic localised lobular hypertrophy of the liver. They are of no practical importance.

Malignant Disease.—Cancerous or sarcomatous growths may occur in the liver either primarily or secondary to similar growths elsewhere.

ÆTIOLOGY.—Hepatic cancer is rare in early life. Habits of life or over-indulgence in the use of spirituous liquors do not seem to play any part in determining the malady. The influence of hereditary tendency to cancer must not, however, be overlooked, and it has been contended of late years that locality has an influence in bringing it about. Malignant disease of the liver and gall-bladder is primary in about one fourth of the cases; and in about one half of the remainder the primary disease is seated in structures connected with the portal system. Of such as *originate* in the liver, the disease in the larger number commences in the gall-bladder or the bile-ducts, and is of the nature of cylinder-celled cancer. Some few, at any rate, have appeared to be the direct outcome of the local irritation of a gall-stone.

ANATOMICAL CHARACTERS.—Malignant disease of the liver may be either carcinoma or sarcoma, the former being either hard or soft (scirrhous or medullary), the latter round-celled and occasionally melanotic. Colloid degeneration is rare. Cancer may occur as an infiltration of the liver-tissue, when large masses of the liver are uniformly affected, and little or no irregularity of surface results. It usually, however, occurs in circumscribed masses, smaller nodules or larger protuberances, varying in size, to use a familiar comparison, from that of a pea to that of a child's head. These masses are more or less numerous, and usually distinct; or they may encroach upon one another, and coalesce. When near the surface, they give rise to the marked irre-

gularity which is very characteristic of the disease ; and, when large and numerous, cause considerable increase in the size of the liver. The masses on the surface are sometimes flattened, and have a central depression (umbilication) which has been designated as 'cancer navel.' The cut surface is either white, or reddish-white, or darker red when blood has been recently extravasated, or of varying colour from altered blood-pigment, when the extravasation of blood has been of longer date. The portal and hepatic veins are sometimes invaded by the cancer.

SYMPTOMS.—In the earlier stage of this disease, the symptoms are indefinite. When far advanced, however, a prominent irregular swelling may be seen, raising the abdominal parietes, and occupying often a large portion of the abdominal cavity. Lesser grades of the disease may be detected by palpation and percussion. The liver will be found to extend more or less beyond its normal limits ; to be hard and resisting ; and, in a large proportion of cases, irregular. In a few cases, however, when the disease is of an infiltrating form, the surface will perhaps be smooth throughout. At times there is no enlargement of the organ, and the portion affected lies under the ribs, so that physical examination does not help us. There is often tenderness on pressure, especially when the peritoneal coat is inflamed. Usually, but not always, there is pain in the liver itself ; sometimes merely a feeling of tightness and fulness ; at other times a gnawing, aching pain ; and some patients have described the pain as 'burning.' There is frequently also pain shooting back to the spine, over the sacrum, or about the angle of the right scapula. A sensation as of a cord drawn round the right hypochondrium has been complained of. There is sometimes pain radiating down to the lower part of the abdomen ; and occasionally wandering pains in the extremities and body generally are complained of. When the stomach is intact, there may be no material disturbance of its functions, but usually derangement is manifested by loss of appetite, nausea, vomiting, and other symptoms, which will be intensified if the stomach is implicated in the disease. The bowels are, as a rule, constipated in the earlier stage, but toward the close there is often more or less looseness. Jaundice occurs in nearly one half of the cases of malignant disease of the liver, and is due to compression of the bile-ducts by cancerous masses within the organ, or by an enlarged lymphatic gland in the portal fissure. When once established it is permanent, and the colour of the patient varies, being pale yellow, or deep olive-yellow, or greenish, or sometimes of the dark hue which has given rise to the term 'black jaundice.' The stools in such cases are white or clayey in appearance ; and the urine deep-coloured from bile pigment. The condition of the urine, when there is no jaundice, is variable ; in the earlier stages of the disease it is generally scanty and pigmented, and loaded with urates ; in the last stage, copious, pale, and deficient in urea. This condition is due to the utter failure of digestive and nutritive power. Ascites is present in more than half of the cases, and is due either to compression of the portal vessels, or to implication of the peritoneum. Sometimes the large size of the tumour, especially if ascites be present, may cause much pressure upwards, and give rise to distressing chest-symptoms, such as embarrassed breathing or palpitation. Hæmorrhage not infrequently occurs

in advanced cases. The blood may come from the stomach or bowels, and be due to portal obstruction ; or may be of passive character, as in scurvy or purpura ; or the bleeding may take place beneath the skin. In the latter case the hæmorrhage is accompanied, according to Frerichs's experience, by intense jaundice, and usually by somnolence and delirium. The complexion of patients suffering from the disease under consideration, when there is no jaundice, is usually sallow, anæmic, earth-coloured. There is, in a large majority of cases, progressive and, towards the close, often extreme emaciation. Generally there is no fever, but it may be even considerable and of a hectic type, when the cancerous development goes on rapidly. When the disease of the liver is secondary to, and complicated with, cancerous affections of other organs, as the stomach—which occurs in a considerable proportion of cases—pancreas, uterus, or mammary gland, symptoms may exist indicating such complications, but need not be specially dealt with here.

DIAGNOSIS.—When hepatic cancer is somewhat advanced, and the liver large and irregular on its surface, the diagnosis will be easily effected. In the early stage, on the contrary, and throughout some cases in which the liver is not perceptibly enlarged, one must be cautious in giving a hasty or too decided opinion. Inherited tendency to cancer ; the age of the patient ; in women the period of 'change of life ;' a sallow earthy aspect ; progressive emaciation ; and pain in the right hypochondrium, point with fair probability to the disease. But nearly the same conditions and symptoms may be associated with aggravated hypochondriasis, or chronic catarrh of bile-ducts ; and in the latter case the difficulty of diagnosis is increased, as gall-stones are often present when there is cancer. Permanent closure of the bile-duct from other causes gives rise to persistent jaundice and other symptoms, as in the case of closure by pressure from a cancerous mass, but the emaciation and loss of strength are then seldom so rapid. Enlargement of the liver from malignant disease may be confounded with the following hepatic enlargements and malignant tumours : 1. Albuminoid or lardaceous disease. In this affection, the perfectly smooth surface, with preservation of normal shape of the liver, and absence of that peculiar hardness and pain in the tumour and of jaundice, will be sufficiently distinctive, accompanied as it should be by associated indications in other organs (*see* AMYLOID DISEASE). 2. In many cases of cirrhosis, as also of malignant disease, the liver is enlarged and its surface uneven, and in both diseases there is great resemblance in the aspect and general cachectic state of the patient, and similar disturbance of gastric and hepatic function. In cancer, however, the ascites is generally but slight, and the liver continues to increase, and is marked by large hard nodules and protuberances. In cancer the skin is often moist ; in cirrhosis it is harsh and dry. Intemperance is not an element in the ætiology of cancer, as it is in cirrhosis. In the latter disease, as also in lardaceous liver, the spleen is frequently enlarged. 3. Hydatid tumour is to be distinguished from a localised cancerous mass by the presence of more or less distinct fluctuation ; and the absence of pain, and of serious functional and constitutional symptoms. 4. A tumour caused by hepatic abscess might perhaps give evidence of fluctuation ; at any rate it would probably be associated with or

consecutive to dysentery; and is often attended by rigors, hectic fever, and characteristic shoulder-tip pain. 5. Malformations and malpositions of the liver have been mistaken for cancerous enlargement, especially in females about the period of 'change of life.' 6. A largely distended gall-bladder has been mistaken for a cancerous projection from the liver, but the smooth oval swelling, and the site of the enlargement, are for the most part distinctive. 7. Cancer of the omentum would present a movable tumour, separable, probably, from the liver by a slight area of tympanic resonance. 8. Cancerous deposits in the left lobe of the liver may be readily mistaken for cancerous affections of the stomach. The following points will assist in diagnosis: (a) Percussion even in cases of great thickening of the walls of the stomach gives a tolerably clear tympanic sound; in cancer of the left lobe of the liver, the sound is much more deadened, and is only somewhat tympanic on stronger percussion-stroke; (b) careful examination of the liver, and of the stomach, when full and when empty, will also lead to a correct conclusion; (c) even when the liver and stomach are both affected, careful examination may often make out the boundaries of disease in each. 9. Malignant tumour of the right lobe may be mistaken for enlargement of the right kidney; in the latter case percussion will generally reveal a tympanic zone between the hepatic dulness and the tumour, owing to the presence of intestine between the kidney and liver. The hepatic tumour is also distinguished from this and other abdominal tumours by its following the movements of the diaphragm in respiration. But when the renal enlargement is very great, diagnosis is not easy. 10. Malignant disease of the ascending or transverse colon will constitute a movable and generally somewhat tympanic swelling; and fecal accumulations in the colon may be removed, but not always readily, by aperients and injections. Percussion, too, will often elicit a resonant space between the enlarged intestine and the liver.

PROGNOSIS.—The prognosis is always unfavourable. Emaciation and loss of strength are usually progressive. The disease, when once fully pronounced, runs its course rapidly, the fatal termination being seldom deferred beyond a year. Scirrhus has usually a longer duration than medullary cancer.

TREATMENT.—This can be but palliative, and directed to rendering the inevitably fatal course as smooth as possible by relieving distressing symptoms. Remedies which in other hepatic affections are valuable, such as cholagogues, or mineral waters, are here useless, if not worse. The diet should be plain and nourishing; and the moderate use of wine and alcohol is not contra-indicated, as in other disorders of the liver. Serious gastric and other derangements must be met by appropriate remedies; it being always borne in mind that we have to soothe the patient, and not add to his distress by the exhibition of nauseous drugs. The administration of ox-gall is sometimes useful in the jaundice that is so common, and the liquor pancreaticus is also worth trial as an aid to the defective digestion of fats. For the relief of pain, the various preparations of opium are indicated, and, as a rule, morphine acts the best: it may be administered either internally, or by the hypodermic method, and must be repeated when pain demands it. Local applications over the liver, as poultices,

spongiopiline, &c., with tincture of opium sprinkled over the surface, are useful, especially when the peritoneal coat is inflamed. Tapping should not be had recourse to for the relief of ascites, unless this becomes so great as to interfere by upward pressure with the functions of the lungs or heart. The fluid soon re-accumulates, and the effect of the operation is to hasten the fatal termination.

JAMES F. GOODHART.

LIVER, Nutmeg.—SYNON.: Fr. *Foie Noix de Muscade*; Ger. *Muskatnussleber*.—See LIVER, Hyperemia of, Passive.

LIVER, Syphilis of.—A. **Hereditary Syphilis.**—SYNON.: Congenital syphilis; Pericellular, monocellular, intercellular cirrhosis.—In infants affected with hereditary syphilis the liver is much more often affected than in the acquired disease; this is in favour of the view that the poison arrives from the placenta by the umbilical vein, which it will be remembered supplies blood to the liver, rather than that the ovum is directly infected by a syphilitic spermatozoon.

MORBID ANATOMY.—To the naked eye in the slighter cases the liver is little, if at all, altered; in more marked instances it may be brown, yellow, or even of a violet hue. It is enlarged, and may show adhesions on the surface. On section it is tougher than in health, and marbled, with loss of the normal lobulation. Small miliary spots, minute granulomata, are generally visible; in exceptional instances genuine gummata or localised areas of fibrosis have been present. Histologically the change is that characteristic of secondary syphilis, a diffuse small-celled infiltration between the liver-cells, and contrasts with the localised lesions of tertiary syphilis. This intercellular small-cell infiltration is accompanied by degenerative changes in the hepatic cells, and may develop into varying degrees of fibrous tissue. The small spots resembling miliary tubercles are collections of small round cells (syphilomata). Very considerable variations may occur in the lesions actually found in congenital hepatic syphilis; Hudela described as many as seven different types.

CLINICAL FEATURES.—Enlargement of the liver and spleen in a child with other evidences of hereditary syphilis justifies the diagnosis. The liver is smooth, firm, and tender, while its size is a rough indication of the severity of the disease. Very exceptionally a definite tumour can be felt. Jaundice and ascites are rare.

TERMINATIONS.—When death occurs in infants with congenital syphilitic disease of the liver, it is due to marasmus and not to the direct effects of the hepatic lesion. Inasmuch as it is a secondary lesion, intercellular cirrhosis is curable and reacts well to mercury, and in most cases that survive the liver probably recovers completely. As a late result of untreated hereditary hepatic syphilis tertiary manifestations may develop. This *delayed hereditary syphilitic* disease shows itself about puberty or even later. The liver may be much scarred by cicatrices and deformed by gummata, while extensive lardaceous disease may in addition be present. In other words, it is the same as the tertiary lesions of acquired syphilis. It is probable that, as a result of intercellular cirrhosis that has undergone resolution, the liver is left in a condition of more or less lowered resistance. If the causes of ordinary portal cirrhosis then arise, the liver falls a more ready prey, and what may be called *parasyphilitic cirrhosis*

results. Multilobular cirrhosis in children, formerly the subjects of intercellular cirrhosis, is thus analogous to the parasymphilitic lesions of acquired syphilis, tabes dorsalis, and general paralysis of the insane. Probably many of the cases of multilobular cirrhosis in young children are of this nature.

TREATMENT.—This is that of hereditary syphilis, i.e. mercury either by inunction or by the mouth. Mercurial ointment should be rubbed into the skin of the axilla or groin daily for the first three months, then it should be intermitted for a week, and later for two weeks at a time, and should be continued into the second year when it should be practised for one month in every three. Mercurial inunction may be carried out by placing the ointment under the child's binder and letting it work in, but this is less satisfactory than the first-named method. Inunction is more effective than giving hydrargyrum c. cretâ by the mouth, since the latter may set up diarrhoea. To a child under two months of age half a grain should be given twice a day and the dose gradually increased. The treatment of delayed or tardive hereditary syphilis is that of tertiary syphilitic disease of the liver, while that of parasymphilitic cirrhosis is the same as in portal cirrhosis.

B. Acquired Syphilis.—*In the secondary stage* jaundice is sometimes seen synchronously with the roseola; it yields to mercury and not to the ordinary remedies for catarrhal jaundice, and is therefore probably directly related to syphilitic infection. It has been referred to various causes, such as a change in the mucous membrane of the ducts analogous to that of the cutaneous roseola, to pressure exerted by enlarged glands in the portal fissure, or to a general toxic change in the liver-substance leading to catarrhal changes in the minute bile-ducts. The jaundice is benign and must be distinguished from that of acute yellow atrophy which in some rare instances has been known to attack the subjects of secondary syphilis. It appears that women are more disposed to this benign syphilitic jaundice than men.

Intercellular cirrhosis has generally been considered to be pathognomonic of hereditary syphilis, but it seems almost certain that it also occurs in the secondary stage of the acquired disease, though opportunities for investigation of this point are infrequent.

In the tertiary stage the liver may show gummata and cicatrices, while lardaceous disease is a well-recognised sequela of syphilis. Syphilitic change in the liver is much commoner than in any other internal organ, but it is not nearly so frequent as in hereditary syphilis. Men are more often affected than women, and it has been thought that local factors such as traumatism, alcoholism, or past affections of the liver such as jaundice may determine syphilitic lesions. Gummata are usually multiple, and are commoner in the larger right lobe and on the anterior surface. They are not uncommon near the falciform ligament, and may set up local perihepatitis or adhesions to the diaphragm. By contraction of cicatrices and gummata the liver may become lobulated, deformed, and extensively scarred; and parts of the organ may be practically destroyed. Usually the lesions are localised and the intervening liver-tissue is fairly healthy, but the organ may be widely lardaceous or there may be localised lardaceous change around a gumma. On section a gumma has a dead-white appearance and is dry and slightly friable. Cicatrices depress the surface of

the liver and invade it for a short distance. Gummatous change may give rise to very considerable enlargement of the liver, especially when combined with lardaceous change.

In its earliest stage a gumma is a mass of pink granulation-tissue; as the result of impaired vitality the central part dies and from fatty degeneration becomes caseous. Around the caseous centre giant-cells are seen, while further out the granulation-tissue organises into a fibrous capsule; the arteries in this area show endarteritis obliterans. This fibrous capsule invades the adjacent lobules, which are, moreover, occupied by a small-cell infiltration. In young gummata the fibrous capsule is but slightly developed, while in old ones the capsule is thick, contracts, and may contain calcareous material. As a result of contraction and absorption a gumma may shrivel into a cicatrix perhaps containing a central core of caseous material. Cicatrices may be the relics of gummata, or may arise from organisation of syphilitic granulation-tissue without any preliminary caseation. Now that prolonged suppuration is less common, syphilis is the more frequent cause of lardaceous disease of the liver. See AMYLOID DISEASE.

CLINICAL FEATURES.—Gummata and especially cicatrices not uncommonly are quite latent and give rise to no symptoms. The clinical manifestations may be postponed for many years after the primary infections, though a fair proportion of cases give rise to trouble within three years of infection. Very considerable variation exists in the clinical picture produced by syphilitic disease of the liver. In many cases portal cirrhosis or chronic peritonitis is imitated; probably many reputed cures of cirrhosis with ascites are of this nature. Hæmatemesis is rare and digestion is much less disturbed than in cirrhosis; the subcutaneous abdominal veins may be enlarged; and pain, tenderness, and uneasiness in the right hypochondrium may depend on syphilitic inflammation of the capsule of the liver. In other instances gummatous disease of the liver simulates malignant disease; a definite tumour or tumours in an enlarged liver are found, and may be associated with ascites or less commonly with jaundice. In the absence of ascites and other signs, a large gumma may resemble a deeply seated hydatid cyst. In rare instances a large gumma is associated with fever, and may then suggest intrahepatic suppuration, malaria, or even tuberculosis or typhoid fever. Exceptionally a gumma involving the bile-ducts in the portal fissure closely simulates gall-stones; the diagnosis is very difficult, and in the absence of any evidence of syphilis impossible. When gummata are combined with lardaceous disease their presence may be easily overlooked.

The *diagnosis* depends on evidence of past syphilis or on the relief of the symptoms after antisymphilitic treatment. Even in the entire absence of any proof of syphilis, antisymphilitic treatment should always be tried in cases suggesting portal cirrhosis or chronic peritonitis, and in hepatic tumours of doubtful origin; and in some of these improvement will decide the diagnosis. It must be remembered that cicatrices cannot be removed by treatment, and that the failure of antisymphilitic treatment does not necessarily and absolutely disprove syphilis. The *prognosis* of syphilitic disease of the liver is, when treated, fairly good and far more hopeful than in the conditions—cirrhosis, chronic peritonitis, malignant disease—that may imitate it.

TREATMENT should consist in adequate doses of iodides combined with mercury. The iodides of potassium, sodium, and ammonium should be given at first so that 20 grains of iodide salts are taken in the twenty-four hours; this should be increased until 80 or 90 grains are taken in the twenty-four hours. Mercury may be given in the form of hydrargyrum c. cretâ combined with powdered opium to prevent diarrhœa, or the liquor hydrargyri perchloridi may be combined with the iodides. When gummata develop rapidly and early after infection, intra-muscular injections of soluble mercurial salts may be employed. Recently large gummata have been partially removed with good results in promoting absorption under medical treatment.

H. D. ROLLESTON.

LIVER, Tubercular Disease of.—Tuberculosis may affect the liver (1) as part of a general acute infection (*acute miliary tuberculosis*); (2) as a local disease spreading from, and limited to the neighbourhood of, the bile-ducts (*tubercular cholangitis*); and (3) as single limited foci of 'conglomerate tubercle.' Only the second variety has any clinical interest. It may give rise to attacks resembling biliary colic without jaundice. It does not cause ascites, and rarely produces any local symptoms or signs.

LIVER-FLUKE.—A common name for the *fasciola*. See ENTOZOA.

LLANDRINDOD, in Radnorshire, South Wales.—Saline, sulphated, and chalybeate waters. See MINERAL WATERS.

LOBULAR (*lobulus*, a little lobe).—Of or belonging to a lobule. A term generally applied to morbid conditions affecting individual lobules of organs which are thus constituted, such as *lobular pneumonia*, *lobular pulmonary collapse*, and *lobular hepatitis*.

LOCK-JAW.—A popular synonym for tetanus. See TETANUS.

LOCOMOTOR ATAXY (*locus*, a place; and *κινεω*, I move; *ἀ*, priv., and *τάξις*, order).—See TABES DORSALIS.

LOECHE-LES-BAINS (LEUKERBAD), in Switzerland.—Thermal calcic waters and climatic health resort. See MINERAL WATERS.

LORDOSIS (*λорδός*, bent).—A term applied to abnormal curvature of the spine forwards. It is found chiefly in the lumbar region as an increase of the natural curvature; but it may also occur in the dorsal and cervical regions. See SPINE, Diseases of.

LORETO, in the Romagna in Italy.—Saline waters. See MINERAL WATERS.

LOS ANGELES, the capital of southern California. A health-resort, 110 miles from the Pacific coast, in a well-sheltered fertile valley at an elevation of 371 feet. Famous for its orange and lemon groves. Pasadena, a suburb, lies 500 feet higher, and is well supplied with hotel accommodation. Climate mild, but not marked by the degree of equability of temperature that characterises the Pacific stations. Mean temperature 61° F.; mean daily range 24° F.; and rainfall 15 inches.

LUCCA, in Tuscany.—Thermal waters containing sulphate of calcium with iron. See MINERAL WATERS.

LUCID INTERVALS.—No better definition of this state has been given than that of Lord Thurlow, who calls it 'an interval in which the mind, having thrown off the disease, had recovered its general habit.' It must be regarded as extremely unlikely that a perfect restoration to reason has taken place in the course of any long-continued insanity, without full opportunity having been afforded of testing its nature. The law more readily recognises the restoration of the mind to a state of civil capacity such as will render testamentary acts valid, than such temporary recovery as would restore responsibility for crime. If a civil act be rationally performed, the law accepts that as *prima facie* proof of the capacity of the agent; but juries very seldom convict the accused of a crime if insanity is proved to have existed within a short period of its commission. JOHN SIBBALD.

LUHATSCHOWITZ, in Moravia.—Muriated alkaline waters. See MINERAL WATERS.

LUMBAGO (*lumbi*, the loins).—SYNON.: Fr. *Lumbago*; Ger. *Lendenweh*.—Muscular rheumatism affecting the muscles and fasciæ of the lumbar region. See RHEUMATISM, MUSCULAR.

LUMBAR ABSCESS.—An abscess, generally of spinal origin, occupying the lumbar region, or pointing in the loin external to the erector spinæ. See PSOAS ABSCESS.

LUMBAR ANÆSTHESIA.—Professor Bier at Kiel and Monsieur Tuffier in Paris have lately performed many operations under the local analgesia produced by injecting 1-4 centigrams of a freshly prepared and sterilised solution of 1 in 200 hydrochlorate of cocaine into the lumbar subarachnoid space. The method has not been adopted, to any great extent, in England; but it may possibly prove useful when the operator is single-handed, especially in extraperitoneal and thoracic operations. Vomiting occurs so often during the period of analgesia that it is not adapted for the more serious intraperitoneal operations. See ANÆSTHESIA, LOCAL. D'ARCY POWER.

LUMBAR PUNCTURE.—DESCRIPTION.—The operation of puncture of the spinal canal in cases of meningitis may be employed in adults, but is usually performed in children. It consists in withdrawing the cerebro-spinal fluid from the subarachnoid space which is punctured between the laminae of the third and fourth lumbar vertebrae. A fine trocar and cannula are well boiled, and the skin of the patient is rendered sterile. The child is then placed upon its right side with the thighs so bent that it is curled up and the vertebral column is well bowed. The spine of the fourth lumbar vertebra is then found either by counting downwards from the last dorsal vertebra, to which the twelfth rib is attached, or by dropping a perpendicular upon the bed from the highest point of the crest of the ilium, since this line crosses the upper border of the spine of the fourth lumbar vertebra. This point is selected because in a child at birth the spinal cord only reaches as low as the third lumbar vertebra, while in an adult the lower limit of the cord is level with the lower border of the first lumbar vertebra. The

exact point of puncture may be marked with a drop of iodine, and local anæsthesia may be produced by ethyl chloride, but this is not often necessary. The trocar and cannula are plunged through the skin immediately to one side of the spine of the third lumbar vertebra and on a level with its lower border. It is pushed on boldly until the point of the trocar strikes the bone—the lower border of the lamina. The handle of the trocar is then directed upwards so that its point passes downwards over the lamina, and it is pushed onwards until a grating sensation is felt, caused by the trocar passing through the ligamentum subflavum. The trocar is withdrawn as soon as the cannula has fairly entered the subarachnoid space, and the cerebro-spinal fluid issues either in a stream or drop by drop. The quantity of fluid which escapes varies greatly: it is often only a few drops, but the average amount is from 4 to 12 drachms. It flows more rapidly when the patient is raised than when he is recumbent, and the rate often varies synchronously with the respiratory rhythm. The cannula is withdrawn as soon as sufficient fluid has been collected, the puncture is sealed with a film of cotton-wool soaked in collodion, and a pad and bandage are applied. The operation is trivial, and the writer has never seen any harm result from it, though clumsy operators have broken their needles against the laminæ, and a few cases of death within 24 hours of the puncture have been recorded. The surgeon at first is a little apt to under-estimate the thickness of the erector spinæ, and his needle thus fails to penetrate the canal; his labour is then in vain, and no fluid escapes.

USES.—Lumbar puncture is more or less useful for diagnostic purposes in cases of meningeal hæmorrhage, hydrocephalus, acute serous, sero-fibrinous, purulent, and tubercular meningitis, but it does not seem to have any remedial or curative value. The cerebro-spinal fluid obtained by lumbar puncture in these affections, as well as in the obscure nervous disorders secondary to infective diseases in children, which are now grouped together provisionally under the term 'meningism,' should always be examined histologically, by inoculation into animals, and by plate-cultivation. Reliance should never be placed upon a single method or upon one inoculation.

D'ARCY POWER.

LUMBRICUS.—By many practitioners this term is still employed to designate the large round worm (*Ascaris lumbricoides*). The title is entirely a misnomer, for, notwithstanding its general resemblance to ordinary earth-worms, its organisation is totally different. Occasionally, in practice, patients seek to deceive the medical attendant, by placing one or more earth-worms in the night-stool or chamber-pot. See ENTOZOA.

LUNACY, Law of.—Medical men and lawyers look at insanity from somewhat different points of view. A medical man applies his mind to its study so as to ascertain how far he may infer from the evidences of mental action the existence of morbid conditions which he may hope to alleviate or remove. It is therefore his duty to be acquainted with such symptoms as give the earliest indication of the approach or development of these morbid processes. For it is during their initial stages that he may most successfully intervene with the resources of his art, to check their progress or to ward them off. He therefore identifies with the existence of disease every deviation from the healthy

mental standard which indicates the necessity for medical treatment or advice. A lawyer, on the other hand, takes note of insanity only in so far as it affects the safety of person or the preservation of property. The question which he seeks to determine is whether a person is justly responsible for certain acts which he has committed, or is competent to perform certain acts which he may be called upon to perform. It is evident, therefore, that the legal view of insanity must naturally be much more limited than the medical. 'A lawyer, when speaking of insanity,' says Sir James Stephen, 'means conduct of a certain character; a physician means a certain disease, one of the effects of which is to produce such conduct;' and though this has been adversely criticised, it seems to show correctly the directions in which the two views diverge. They might perhaps be as fairly indicated in other words if we say that the lawyer has to deal with the nature or quality of certain acts, while the physician has to deal with the condition of certain persons. No satisfactory general definition of legal insanity which would suit all cases ever has been given, or ever can be given. In the earlier ages of our legal system none but the most violent or degraded cases of insanity were recognised. Bracton in the thirteenth century defined a madman as 'one who does not understand what he is doing, and, wanting mind and reason, differs little from brutes.' Sir Edward Coke, though he recognises different classes, according as the insanity is congenital, permanent, or temporary, only admits that a person is insane when he is *non compos mentis*, or has wholly lost his memory and understanding. Sir Matthew Hale, in the seventeenth century, was the first to recognise the existence of less extreme forms of insanity. 'Some persons,' he said, 'that have a competent use of reason in respect of some subjects, are yet under a *particular dementia* in respect of some particular discourses, subjects, or applications; or else it is partial in respect of degrees, and does not excuse persons who commit capital acts in this state.' He also said that it is 'very difficult to define the invisible line that divides perfect and partial insanity,' and 'that most persons that are felons of themselves or others, are under a degree of partial insanity when they commit these offences.' The recognition of these gradations reaching even to the mutual overlapping of crime and insanity, indicates as much breadth of view as could be expected at a time when the very judge who recognised them passed sentence of death on persons convicted of witchcraft. We cannot doubt, indeed, that at that period the ignorance of the nature of insanity was such that many lunatics were executed for this offence. The recognition of the necessity of taking legal account of degrees of insanity less severe than 'furious madness' or idiocy may be said to have commenced at the beginning of the nineteenth-century. The medical practitioner requires to be acquainted with the present legal relations of insanity chiefly from three points of view: 1. Where a person suffers from such unsoundness of mind that it is necessary for his welfare, or the safety of the public, that his liberty should be restricted by his being placed in an asylum or subjected to similar restraint. 2. Where a person suffers from such unsoundness of mind that he is incapable of managing himself or his affairs. 3. Where irresponsibility for crime, on account of insanity, is pleaded in a

court of law. The subject is only dealt with in this article from the first of these points of view. The other two aspects of the subject are treated in the articles on CIVIL INCAPACITY, and CRIME, Irresponsibility for.

The lunacy statutes differ slightly in the three divisions of the kingdom. It will therefore be necessary, after describing what is required under the law in England, to show where its requirements differ from those which exist in Scotland and Ireland. The details to be given here will only include what is necessary for the information of the general practitioner. Any one who intends to devote himself specially to the treatment of the insane, or to receive one or more persons of unsound mind into his house, will find the regulations with which he must comply fully described in works specially devoted to the subject. When a person living in his own home is under treatment for insanity, the medical attendant is justified by the common law in adopting any measures of restraint which may be necessary for safety or the proper treatment of the malady. This has been decided by the courts of law in recent cases. If, however, it is proposed to place the patient in an asylum, or under the charge of any person who is to derive profit either directly or indirectly from the proceeding, it is necessary that certain forms should be carefully observed.

In the case of a *Chancery lunatic*, the patient may be taken charge of by his 'Committee of the Person,' or he may be received into an asylum or other institution for lunatics, or into a private house, on an order by the committee. Such an order must, however, have annexed to it an official copy of the appointment of the committee. If no committee has been appointed, an order by a Master in Lunacy is sufficient authority for the detention of the lunatic.

In the case of *other private patients*, it is necessary, under the Lunacy Acts of 1890 and 1891, to have a *reception order* made by the 'judicial authority.' The judicial authority under the Act is either a judge of county courts, a stipendiary magistrate, a metropolitan police magistrate, or a Justice of the Peace or chairman of a board of guardians duly and specially appointed for such duty. This order is obtained by private application upon a *petition*, accompanied by a *statement of particulars* and by two medical *certificates*. These must be made in the forms prescribed by statute.*

THE PETITION.—This document must be signed, if possible, by the husband or wife, or by a relative, and, if not, it must contain the reasons for its being signed by another person, and a statement of the connection of that person with the patient. The person signing the petition must be at least twenty-one years of age, and must have seen the patient within fourteen days of the date of presenting the petition. If a petition for a Reception Order has been previously presented in regard to the patient, and has been dismissed, the facts relating thereto must be stated in the petition; and there must be presented along with it a copy, to be obtained from the Commissioners in Lunacy, of the statement received by the Commissioners of the reasons for such dismissal. If one of the medical certificates

is not given by the usual medical attendant on the patient, the reason for this must be stated in the petition; and if an urgency order (hereafter described) has been made in regard to the patient, it must be referred to in the petition. No member of the managing committee of a hospital can sign the petition for the reception of a patient into that hospital.

THE MEDICAL CERTIFICATES.—One of the medical certificates must, whenever practicable, be by the usual medical attendant on the patient. Each certificate must be on a separate sheet of paper, and each person who signs a certificate must have separately examined the patient not more than seven clear days before the presentation of the petition, and must be a person registered under the Medical Act, 1858, and in actual practice. Certain persons are disqualified for signing either of these certificates or the certificate required in support of an urgency order. These are: The petitioner, or the person signing the urgency order, or the husband or wife, father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, partner, or assistant of such petitioner or person. The statute also forbids the following persons from signing certificates: The manager of the institution into which the patient is to be received; the person who is to have charge of the patient as a single patient; any person interested in the payments on account of the patient; any regular medical attendant in the institution; or the husband or wife, father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, or the partner or assistant of any of these persons. Again, neither of the persons signing the certificates can be the father or father-in-law, mother or mother-in-law, son or son-in-law, daughter or daughter-in-law, brother or brother-in-law, sister or sister-in-law, or the partner or assistant of the other of them. And no member of the managing committee of a hospital can sign a certificate in the case of a patient to be received into that hospital.

THE RECEPTION ORDER.—The judicial authority may, upon the presentation of the petition and certificates, make the reception order forthwith; or, if the certificates are not regarded as satisfactory, or for any other reason, he may appoint a time, not more than seven days after the presentation of the petition, for its consideration, and may, if he thinks necessary, visit the patient. The petition is then considered in private, and no one except the petitioner, the alleged lunatic (unless the judicial authority otherwise order), any one appointed by the alleged lunatic for that purpose, and the persons signing the medical certificates—all of whom are bound to keep secret the matters disclosed—can, without leave of the judicial authority, be present. A further adjournment for not more than fourteen days may then be made for further inquiry if the judicial authority shall think fit, after which he either grants the order or dismisses the petition. A reception order is sufficient authority for the person signing the petition, or some one authorised by him, to take the patient and convey him to the place mentioned in the order; but it does not continue in force unless the patient is received in the asylum or house within seven days of its date, except when the execution of the order

* These forms can be obtained at any law stationer's; but, as they are frequently wanted with the least possible delay, we mention the name of Messrs. Shaw & Sons, 6 Fetter Lane, London, E.C., as in the habit of supplying them.

has been suspended by reason of a medical certificate that the lunatic is not in a fit state for removal—in which case the order continues in force for fourteen days, unless a subsequent certificate is given that the patient is fit for removal, and in that case the order remains in force only for three days after the date of that certificate.

THE URGENCY ORDER.—If the necessity for placing a patient in an asylum or house is regarded as urgent, an order, called an 'urgency order,' may be signed as in the case of the petition by a husband or wife, or by a relative; and, if signed by another person, the reason for this must be stated, and also the connection of that person with the patient. The person signing must be at least twenty-one years of age, and must have seen the patient within two days of the date of the order. This order must be accompanied by a statement of particulars similar to the statement of particulars which is required to accompany the petition, and by a medical certificate stating that it is expedient for the welfare of the patient, or for the public safety, that the patient should be forthwith placed under care and treatment. The certifier in this case must have seen the patient not more than two days before his reception. An urgency order is sufficient authority for a person, authorised by the persons making the order, taking the patient and conveying him to the place mentioned in the order, and it remains in force for his detention for seven days from the date of the order; or, if a petition has been presented, it remains in force till the petition has been disposed of. An urgency order may be made either before or after a petition has been presented; but, if made before, it must be referred to in the petition, and, if made after, a copy of it must be sent forthwith to the judicial authority to whom the petition has been presented.

The information contained in the foregoing paragraphs indicates sufficiently for the purposes of the general practitioner the steps which require to be taken for placing a person of unsound mind under care and treatment. It is of the greatest importance, however, in filling up the several forms, that the most scrupulous attention should be given to the marginal notes which are attached to the forms. Great care must be taken to ensure that the instructions contained in these marginal notes are complied with in every, even the smallest, detail. For it may happen that what may appear to many to be a trifling deviation from the instructions, will render the document invalid, and thus entail much inconvenience and distress. In regard to the medical certificates, it should be kept in view that the persons who sign them must arrive at opinions in regard to two questions which are quite distinct. Each certifier has first to determine whether the patient is of unsound mind, and, next, whether it would be proper to place him under detention. In stating the facts, it must be borne in mind that they must be such as will appear to the judicial authority making the reception order sufficient evidence of insanity. Great care must be taken to state the facts both intelligently and accurately. There must be sufficient in the facts observed by the medical man himself to justify the opinion to which he certifies. A certificate founded solely on information obtained from others is invalid, and such information should be regarded by the certifier as useful merely to corroborate his opinion. It must also be kept in view that the opinion must be

directly deducible from examination of the patient on the particular day and at the particular place specified. The statement of facts observed should therefore contain at least one statement of a fact, or combination of facts, which could not be made in regard to a person of sound mind. A frequent error is the stating of the facts in such an imperfect manner that, though they may have been as a matter of fact sufficient evidence of insanity, the manner in which they are recorded makes them appear to be insufficient. It is sometimes stated, for example, that a patient 'believes himself to be possessed of great wealth'—without adding that this is an erroneous belief. Another kind of failure to state the facts intelligently is illustrated by an actual instance where they were given thus: 'His appearance, manner, mode of speaking, as well as his conduct.' These were probably quite adequate to prove to the certifier that the patient was insane, but it is obvious that they afforded no proof to those who merely read the statement.

If a lunatic, who is not a pauper, and not wandering at large, is not under proper care and control, or is cruelly treated or neglected by any relative or other person having the care or charge of him, information should be given to a constable of the district or a relieving officer or overseer of the parish, who is bound within three days to take steps, prescribed by statute, to have him placed in an institution for lunatics.

The duties performed by medical men and others in connection with the placing of patients under care and treatment have been frequently the source of great trouble and expense, from legal proceedings taken against them by patients or others who have felt themselves aggrieved. In order to afford protection against such action, when improperly taken, it is provided in the Lunacy Act of 1890 that a petitioner or certifier 'shall not be liable to any civil or criminal proceedings, whether on the ground of want of jurisdiction or on any other ground, if such person has acted in good faith and with reasonable care.' If such proceedings are taken, they 'may, upon summary application to the High Court of Justice or a judge thereof, be stayed upon such terms as to costs and otherwise as the court or judge may think fit, if the court or judge is satisfied that there is no reasonable ground for alleging want of good faith or reasonable care.' Even under the protection afforded by this enactment, it will be understood that both expense and trouble may be occasioned by vexatious proceedings, as the steps necessary to get proceedings stayed by the High Court may involve considerable outlay.

Any one who makes a wilful misstatement of any material fact in any petition, statement of particulars, or medical certificate, is guilty of a misdemeanour; but no prosecution for such misdemeanour can take place except by order of the Commissioners in Lunacy, or by the direction of the Attorney-General or the Director of Public Prosecutions.

A private patient may be discharged from the asylum or house in which he has been detained on the written authority of the person on whose petition the reception order was made, or, if this person is dead or incapable, by the person who made the last payment on account of the patient, or by the husband, or wife, or next-of-kin. If a patient should die while under detention, it is necessary to

give notice of the death to the coroner and to the Commissioners in Lunacy.

In the case of *pauper lunatics*, the procedure is somewhat different from that required for private patients. Any one aware of the existence of an insane pauper in a parish ought, if the case is a proper one for treatment in an institution for lunatics, to give notice to the relieving officer or the overseer. When a district medical officer under the poor-law becomes aware of such a circumstance, it becomes his statutory duty to give this notice in writing within three days after obtaining such knowledge. The relieving officer or overseer may then take steps to have the patient placed in an institution for lunatics upon one medical certificate, accompanied by the order of a justice of the peace, if the justice is satisfied that the alleged pauper is either in receipt of relief, or in such circumstances as to require relief for his proper care.

In order to place either a private or a pauper patient in an asylum in *Scotland*, a petition, accompanied by a statement and two medical certificates, has to be presented to the sheriff.* In the case of a private patient, the person signing the petition must state the degree of kinship or other relation in which he stands to the patient. In the case of a pauper, the petition must be signed by the inspector of the poor. In either case, if there be reasonable ground for so doing, the patient may be placed in the asylum on what is called a 'certificate of emergency,' signed by one medical man. If, however, the order of the sheriff is not obtained within three days thereafter, the patient must be discharged. In the case of a patient placed for profit in a private dwelling-house in *Scotland*, the fact must be reported to the General Board of Lunacy for *Scotland*, and the sanction of the Board obtained.

The procedure required for placing a patient in an asylum in *Ireland* resembles that which is required in *England*. For admission to a private asylum, an order and two medical certificates must be filled up and signed, subject to regulations resembling those already described as enforced in *England*; but the facts indicating insanity do not require to be stated in the certificates.* Pauper patients are placed in district asylums, and are admitted to these institutions on application being made at the asylum of the district in which the patient resides. The necessary form is obtained at the asylum. It consists of (1) a declaration to be made before a magistrate that the patient is insane and destitute, and has no friend able or willing to pay for his board in an asylum; and to this is annexed a statement descriptive of the patient; (2) a certificate by a magistrate, and a clergyman or poor-law guardian, in corroboration of the declaration; and (3) a medical certificate of insanity. When these forms have been filled up, it is necessary to wait until it is notified to some of the friends of the lunatic that there is room for him at the asylum. The procedure specially designed for the committal of dangerous lunatics is, however, frequently adopted in placing paupers in asylums; but this is a course which ought to be avoided, and which the medical practitioner ought specially to discourage. According to this procedure, the patient requires to be apprehended by the police, and brought

before two Justices of the Peace. These call to their aid the medical officer of the dispensary district, and either discharge the patient or order his removal to the asylum. Patients who are not destitute, but whose friends are unable to pay the rates of board charged in private asylums, are received into district asylums at low rates, upon application being made at the asylum in a similar manner to that already described for paupers. The chief difference between the two forms is that, in the case of patients not destitute, the medical certificate requires to be signed by two medical men instead of only by one.

JOHN SIBBALD.

LUNATIC (*luna*, the moon).—SYNON.: *Fr. Lunatique*; *Ger. Mondsüchtig*.—A designation given to persons suffering from mental disorder, because such subjects were formerly believed to be peculiarly affected by lunar influences. The term is used popularly as synonymous with 'insane.' In medical literature it is seldom employed, but the legal relations of the word are important. The adjective 'lunatic' is also used to signify that the object with which it is associated is connected with insanity, as *lunatic asylum*. See *INSANITY*.

LUNGS, Diseases of.—The diseases of the lungs will be dealt with in the following order:

Abscess, p. 908.
Active Hyperæmia, p. 908.
Amyloid Disease, p. 909.
Apoplexy, p. 909.
Atrophy, p. 909.
Brown Induration, p. 909.
Cancer, p. 909.
Cirrhosis, p. 909.
Collapse, p. 909.
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* The regular printed forms for *Scotland* may be obtained from Messrs. T. & A. Constable, 11 Thistle Street, *Edinburgh*; and those for *Ireland* from Messrs. Falconer, 53 Sackville Street (O'Connell Street), *Dublin*.

LUNGS, Abscess of.—**DEFINITION.** Circumscribed suppurative of the pulmonary tissue.

ÆTIOLOGY.—Pulmonary suppuration occurs in many different conditions. These will be considered under the following headings: (1) A pneumonic lobe may disintegrate and become riddled with innumerable small pus-containing cavities large enough to give rise to suggestive physical signs, and yielding a characteristic expectoration; or the lobe may become gangrenous or break down at one or more spots into definite abscesses. (2) Pulmonary tuberculosis may in the same way result in one part of the lung becoming riddled with similar cavities; or a large cavity or a circumscribed cavity of limited size may be formed. The last not infrequently occurs at the base of the lung. (3) Chronic non-tubercular cavities are met with in various situations, the causation of which is uncertain. (4) Local ulceration in bronchiectasis may lead to the formation of cavities lined altogether or in part by granulation-tissue; such cavities may be described as abscesses. (5) Pulmonary embolism, even if aseptic in origin, may result in an abscess, but suppuration is usually an indication that the patient is the subject of embolic pyæmia. Abscesses in pulmonary embolism are generally small, multiple, and situated on the surface of the lung. (6) Injuries of the lung, whether resulting from external injury or inspiration of foreign bodies, may give rise to abscesses. (7) Suppuration of a hydatid cyst may cause abscess (see p. 916). (8) Abscess of the lung may result from the bursting into it of an abscess connected with some external structure, such as mediastinal abscess, suppurating bronchial gland, empyema, sub-diaphragmatic abscess, abscess from carious rib or spine, and secondary suppuration of a malignant growth of the œsophagus.

SYMPTOMS AND PHYSICAL SIGNS.—These vary with the origin and distribution of the suppuration. Their character depends primarily upon whether or not the abscess communicates with the bronchus. In the former case there will be cough and expectoration; in the latter there may be only such physical signs as are caused by a localised collection of fluid together with the general symptoms of suppuration, but often the physical signs are very slight or absent.

The expectoration may vary very much in appearance. It may have the appearance of pure pus, or be dark and chocolate-coloured. It may consist of pus mixed with mucus or saliva, which, in the case of bronchiectasis and some other conditions, will, on standing, separate into three layers. It may be free from odour or have a faint smell, or be as foul and offensive as it is possible to imagine. When the abscess is secondary to suppuration originating elsewhere, the expectoration may contain evidence of its origin, such as bile, necrosed liver-tissue, calcareous nodules, stomach-contents, portions of bone, &c. It is therefore of great importance to examine the expectoration very critically.

If the abscess be small, and does not open into a bronchus, the physical signs are often very equivocal. If it be large and has not opened into a bronchus, the physical signs will approximate more or less closely to those of an empyema, or still more closely to those of hydatid of the lung. After the abscess has established a communication with the bronchus, the physical signs of a cavity should from time to time, if not permanently, be apparent.

The physical signs of the abscess are often ob-

scured by those of the morbid condition of the lung which preceded it. See BRONCHIECTASIS.

PROGNOSIS AND TREATMENT.—The treatment of bronchiectatic abscesses is most unsatisfactory. Even if a large cavity be opened, only partial relief is obtained, as the other cavities continue to discharge. The treatment of basic tubercular cavities is on the whole satisfactory. The most promising cases for surgical interference are the chronic non-tubercular ones. The cure of an abscess may be effected by spontaneous expectoration or by surgical interference. If the spontaneous discharge be inefficient, as shown by the persistence of the general signs of suppuration, or when the abscess does not communicate with the air-passages, surgical interference should be considered. When a local abscess of the lung is suspected, exploration should be made with the aspirator. On no account should a simple exploring trocar be employed unless it has been ascertained in some way that adhesions are present. Otherwise a pneumothorax may result which, apart from the subsequent and often fatal issue, will modify the physical signs, and possibly result in the displacement of the abscess. When the presence of pus has been ascertained a portion of the chest-wall should be removed by taking away two or three inches of two or more ribs, taking care not to injure the pleura. A circle of silk or catgut sutures is then passed with a 'whole-curved' needle deep into the substance of the lung, thus attaching it to the parietal pleura. The parietal pleura and the abscess are then to be incised. As a rule this is safely accomplished by means of a narrow bistoury and subsequent dilatation of the wound by means of dressing forceps; but as there is a risk of serious hæmorrhage it has been recommended to make the incision by means of the actual cautery, and this plan has certain advantages, although the charring of the tissues renders it difficult to appreciate the condition of the lung with the finger, and makes it more likely that secondary hæmorrhage will occur at a later period. Should the hæmorrhage at the time of the operation be severe, the wound must be plugged, but it will be found, as in the case of other vascular viscera, that the bleeding has a tendency to stop spontaneously. A large drainage-tube is to be inserted into the cavity. Irrigation of the cavity is not permissible.

The treatment of pulmonary cavities by inhalations (p. 745) and intralaryngeal injections has been much practised, and, in cases which are not amenable to surgical interference, should be tried; but the results have not been as successful as it was at one time hoped they would be. R. J. GODLEE.

LUNGS, Active Hyperæmia of.—This term is employed to denote widely differing conditions.

1. The vessels in those parts of the lung immediately surrounding a patch of acute inflammation are dilated, and permit the passage of a larger amount of blood than under normal conditions, just as the corresponding vessels do in the case of any other similar foci elsewhere. These circumferential areas may be said to be in a condition of *active congestion*. It is moreover possible that the resonance over them may be slightly impaired.

2. Over-action of the right ventricle may conceivably lead to the presence of an excess of blood in the lung, though such a condition must always be temporary. It is, however, probable that excess

of blood in the vessels of the lung, when due to altered cardiac action, is always the result of defective action of the left side, or of paralytic conditions of the vessels, and is therefore more correctly described as *passive* or *mechanical*, as the case may be.

3. The term 'active hyperemia' is also occasionally applied to the distension of the bronchial and pulmonary vessels which follows the inhalation of hot air and other irritants. This condition cannot, however, be differentiated from true inflammation.

H. MONTAGUE MURRAY.

LUNGS, Amyloid Disease of.—In advanced cases of amyloid disease the lung-tissues may present more or less of this morbid change. See AMYLOID DISEASE.

LUNGS, Apoplexy of.—A synonym for extravasation of blood into the lungs. See LUNGS, Hæmorrhage into.

LUNGS, Atrophy of.—SYNON.: Senile Emphysema; Fr. *Atrophie du Poumon*; Ger. *Lungenatrophie*. See LUNGS, Emphysema of.

LUNGS, Brown Induration of.—See LUNGS, Passive Hyperæmia of.

LUNGS, Cancer of.—See LUNGS, Morbid Growths of.

LUNGS, Cirrhosis of.—A synonym for chronic pneumonia. See LUNGS, Inflammation of; D. Chronic Pneumonia.

LUNGS, Collapse of.—SYNON.: Apneumotosis; Fr. *Affaissement Pulmonaire*; Ger. *Lungen collapsus*.

DEFINITION.—Simple diminution in size of the whole or of a part of a lung, with reduction of the volume of the contained air; caused by interference with its free entrance in inspiration.

ÆTIOLOGY.—The causes of collapse of the lung are either *intrinsic* or *extrinsic*; and frequently the two classes of causes are combined. The *intrinsic* causes present actual obstruction of the respiratory passages, and include all diseases of the larynx, trachea, and bronchi attended by inspiratory dyspnoea, whether due to the pressure of external tumours, to affections of the passages themselves, or to the presence of inflammatory products, blood, or foreign bodies within them. To this class of causes belongs the collapse of the lung which is apt to follow infantile bronchitis, when the tubes become obstructed, and there is no power to expectorate. All causes that interfere with respiratory efficiency favour the occurrence of the condition named. A plug of mucus may be drawn, in inspiration, deeper and deeper into the bronchial tubes, which it obstructs, and, acting like a 'ball plug,' allows the expulsion of air in expiration, but interferes with inspiration; the air not being replaced, apneumotosis is developed; and as there is no air behind the plug of mucus, cough is powerless to expel it. In children bronchial inflammation is exceedingly common; and, the smaller tubes being proportionately smaller in the child than in the adult, the danger of collapse is increased. When children under five years of age die of bronchitis and allied affections, apneumotosis is almost invariably present; and 25 per cent. of the total mortality of infants may be safely set down to this cause. Partial collapse of the lung may follow pressure

on the respiratory passages (see MEDIASTINUM, Diseases of). The *extrinsic* causes of pulmonary collapse are certain conditions of the walls of the chest, which diminish the force of the inspiratory act, such as paralysis or debility of the inspiratory muscles, and softness of their bony attachments. Muscular paralysis is seen in injuries to the spinal cord. Debility of the respiratory muscles may often be observed before death. Collapse of the lung, apart from pleural effusion and pneumothorax, is only rarely met with in adult life, when great prostration occurs in the course of fever, and respiration is impeded by pulmonary congestion. Associated as it is with softness and weakness of the ribs, rickets is one of the most frequent causes of collapse of the lungs. The action of the inspiratory muscles may be still further interfered with by abdominal distension, or by the binding up of the abdomen of the infant with tight bandages. The danger of collapse is lessened when the ribs have gained firmness and fixity, and when, raised by the respiratory muscles, the thoracic cavity is enlarged, and the lungs are consequently expanded. Collapse resulting from admission of air to the pleural cavity is described under LUNGS, Compression of.

ANATOMICAL CHARACTERS.—The whole of one lung or of one lobe may be affected, but the condition is generally limited to individual lobules or small parts of the lung, the affected lobules being abruptly separated from those adjoining. As a rule, several patches of collapse occur in each lung, having a darker colour and more depressed surface than the healthy parts. The lower margin of the left lower lobe is most frequently affected. The collapsed portions of lung are tough and leathery in consistency; they resist pressure, are non-crepitant, are smooth on section, and sink in water. The bronchi are filled with mucous fluid; there is an entire absence of air in the collapsed parts. On inflation, the affected portion assumes a natural appearance, unless considerable congestion exists; whereas in pneumonia inflation cannot restore the lung to its natural appearance. In pneumonia pleurisy is rarely absent; but in collapse the pleura is invariably healthy.

SYMPTOMS.—The symptoms of collapse of the lung vary greatly with the cause, rapidity, and extent of the morbid condition. In severe cases, for example, in the collapse that follows bronchitis in very young subjects, the symptoms are peculiar. There are great prostration, debility, restlessness, and sleeplessness. The temperature falls; the surface becomes cold, blue, or dusky; the eyes become shrunken; and the pulse is quick and small. There is a constant feeble whining cry. Respiration is very quick and shallow, as high as 70 to 80 or even 100 per minute. The rhythm is changed, the interval being between inspiration and expiration, instead of after expiration. There is no pain as in pleurisy. The cough is constant and impotent; is often followed by a cry of impatience; and differs much from the suffocative cough of bronchitis.

On examining the chest the lower part is found retracted and diminished in diameter. The intercostal spaces sink in inspiration, and move outwards slightly in expiration. When the collapse is extensive, there are dulness and resistance on percussion, unless the affected lobules are interspersed among the healthy ones. The respiratory murmur is lost over the affected parts; though conducted breath-

sounds, of a bronchial character, and rhonchi are generally audible almost universally. In the simpler cases of collapse of the lungs, such as occur in pertussis during the severe fits of coughing, some of these symptoms and signs may be suddenly developed, and again speedily disappear.

DIAGNOSIS.—Apneumatosiis may be distinguished from croupous pneumonia by the comparative rarity of the latter disease in infancy; and by absence of the great heat of skin, and of fine crepitation on auscultation. From extensive miliary tuberculosis it is diagnosed by the absence of advancing symptoms of constitutional disorder, though the two conditions may co-exist. In pleurisy, the dulness on percussion, and the absence of respiratory sounds at the base, are much more marked than in apneumatosiis. Congenital collapse, or *atelectasis*, is a condition which has to be distinguished from infantile collapse or *apneumatosiis*. Readily separable by symptoms, these two conditions may be indistinguishable by physical signs. In atelectasis the lung retains, in whole or in part, its foetal condition, nature having failed to establish respiration and fit the child for its new mode of existence. In apneumatosiis the once permeable lungs cease to admit air, and thus death from apnoea occurs without any apparent structural change being discoverable, save that the respiratory organs bear the appearance of foetal, unexpanded lungs.

PROGNOSIS.—The prognosis in collapse of the lung is favourable if the affection is recent, and the child healthy, with fair muscular power, and under favourable hygienic conditions. On the contrary, the disease is generally fatal if it involve a considerable extent of lung, especially if it supervene on atelectasis. Death usually occurs from slow asphyxia, the effect being the same as if the size of the lung were reduced by the removal of the affected parts. As much as half of the entire lungs has been found involved, thus fully accounting for the quickened respiration, the distress, and the dyspnoea, and for the bloodlessness and extreme pallor, with cold blue extremities. The fatality of whooping-cough in infants is mainly due to the ready collapse of the lungs, especially when the child is badly nourished and breathing impure air. The natural course of the disease is from bad to worse: more lung is involved each day; and death occurs after two or three weeks from slow asphyxia. If collapse follows acute bronchitis, death often ensues rapidly; but if recovery takes place, the lungs are slow to regain activity, and the seeds of future mischief may remain. After an attack of pneumonia, complete absence of breath-sounds may exist for a time, and then suddenly—after a blow, shock, or violent cough—air enters the collapsed portion of lung, and the respiratory sounds assume a normal character.

TREATMENT.—Slight counter-irritation, by means of stimulating embrocations, is useful. An emetic of ipecacuanha will help to remove accumulation if the patient is not too weak. Expectoration may be promoted by small doses of similar drugs (*see* EXPECTORANTS). When the lungs are extensively involved, vital power must be kept up by the help of ammonia, perchloride or phosphate of iron, port-wine, beef-tea, and other stimulants; and the food must be selected so as to be digestible by the stomach of the infant.

E. SYMES THOMPSON.

LUNGS, Compression of.—SYNON.: Fr. *Compression du Poumon*; Ger. *Lungen-compression*.

DEFINITION.—Diminution in size of the whole or of a part of a lung, associated with reduction of the volume of the contained air; caused by pressure on the pleural surface.

ÆTIOLOGY.—Compression of the lung may arise in the course of numerous diseases or injuries affecting the chest; the compressing influence being either gaseous, liquid, or solid. The first effect of any of the causes is to permit *collapse*; only when the action of the elastic tissue of the lung ceases does actual *compression* begin.

First, the admission of *air* to the pleura from without through a perforating wound, as from a sword or bayonet thrust; or from within, as by rupture of an air-cell, or the extension of pulmonary ulceration through the pleura, produces in either case compression. If no previous pleurisy has existed and no free opening is present, the compression is complete; but if, on the other hand, long-standing pleurisy has caused adhesion; compression cannot take place, or will be but partial.

Secondly, compression may arise from the presence of *fluid*, such as pleuritic effusion, acute or chronic; passive non-inflammatory effusion, as in hydrothorax; or blood, as in hæmorthorax.

Thirdly, compression of the lung by *solids* is seen in the case of various tumours of the chest, whether originating in the mediastinal structures, in the lungs, or in the thoracic parietes.

In the fourth class of cases compression of the lung is the result of the enlargement of neighbouring parts, other than the thoracic viscera; and especially of the abdomen, as in ascites, and tumours of the liver, spleen, or ovaries.

ANATOMICAL CHARACTERS.—Compression of the lung may be either general or local, complete or partial. A lung compressed by pleuritic effusion is found to be reduced in volume, non-crepitant, dense, but not quite insusceptible of inflation. The blood is coagulated in the affected lobes, the clot being often decolourised and adherent to the walls of the vessels, many of which are impervious, or altogether obliterated; while the pervious vessels and the air-cells of the adjacent parts are distended, and emphysema is produced. In other cases the compressed lung proves to be anæmic, tough, and dry.

In cases of slow recovery from chronic empyema the lung is often found bound down and thickened. The portion of the thoracic cavity vacated by the shrunken lung is occupied by the displaced heart, and sometimes by the extension of the sound lung across the middle line.

SYMPTOMS AND PHYSICAL SIGNS.—The symptoms of compression of the lung vary greatly in accordance with its causes, the rapidity of onset, and the extent and degree of compression. If pleuritic effusion be very rapid the dyspnoea may be exceedingly urgent. After perforation of the pleura with sudden collapse of the lung there also occur acute pain, dry cough, and painful spasms of the intercostal muscles. The pulse is frequent, feeble, and often irregular. Symptoms, more or less acute, of inflammation may follow. In other instances the symptoms are those of hydrothorax or of intrathoracic tumour.

The physical signs of compression of the lung are chiefly those of the associated cause, such as pneu-

mothorax, pleurisy, hydrothorax, or intrathoracic tumour; and partly certain phenomena characteristic of the physical condition of the lung itself. The latter vary considerably with the degree and extent of compression, but they may be described in general terms as follows: Either increased clearness of the percussion-sound over the area of compressed lung, with tubular or rarely even tympanitic quality, especially in children, or in extreme cases of compression complete loss of resonance; indefinite, weak, but occasionally rather blowing or tubular respiratory sound, sometimes mixed with scanty, dry, subcrepitant rhonchus; and exaggerated loudness and ringing quality of vocal resonance. A further description of these symptoms and signs will be found under the headings of the various causes of compression referred to.

DIAGNOSIS.—The diagnosis of compression of the lung is in general simply the diagnosis of the condition on which it depends.

PROGNOSIS.—The prognosis depends on the cause of the compression. Thus in pneumothorax it is unfavourable, though recovery may take place. In hydrothorax the ultimate prognosis is unfavourable, as it is usually an evidence of formidable, if not incurable, organic disease. In pleurisy, if the effusion has been rapid, met by prompt treatment, and uncomplicated with tuberculosis, complete recovery may take place without much compression of lung or distortion of chest; but pleurisy is too frequently the first incident in the development of phthisis. In empyema the prognosis is more favourable than in pneumothorax or hydrothorax.

TREATMENT.—Little need be said as to the treatment of lung-compression. It resolves itself into that of the primary or causative diseases. Bearing in mind the injury done to the lung by compression, efforts should be made to relieve the lung before it has been irremediably bound down. The early adoption of paracentesis thoracis is the most practical means of gaining this end in pleuritic effusion. Remedies calculated to remove effusion and thus relieve the lung should be given, remembering that the more speedy the relief given to the lung the more complete will be the cure. Suitable movements of the chest might be ordered subsequently, with the view of promoting expansion of its walls and of the lung. Nor must a mention of the value of elevated health-resorts, the Engadine for instance, be omitted as a means of promoting the expansion of a lung which has been compressed.

E. SYMES THOMPSON.

LUNGS, Congestion of.—See LUNGS, Hyperæmia of.

LUNGS, Consumption of.—See PHTHISIS.

LUNGS, Embolism of.—See LUNGS, Hæmorrhage into.

LUNGS, Emphysema of.—SYNON.: Fr. *Emphysème du Poumon*; Ger. *Lungenemphysem*.

DEFINITION.—An excess of air in the lungs, whether due to a dilated condition of the air-sacs, or to the presence of air in the interlobular tissue.

VARIETIES.—There are two forms of emphysema of the lungs, namely: A. *Vesicular Emphysema*. B. *Interlobular Emphysema*.

A. **Vesicular Emphysema.**—Vesicular pulmonary emphysema exists in three forms, namely, (1) *partial lobular*; (2) *lobular*; and (3) *lobar*. The last form involves the whole of a lobe, or the

whole of one or both lungs. The first form is rarely seen alone, but is generally associated with the second form, which is very common, and is found in connection with diseases, such as bronchitis, which are attended with violent or long-standing cough. The third form is by far the most important, and will be more especially referred to in the present article. It more frequently attacks both lungs than one, and the lower as well as the upper lobes. It is a serious malady, and sometimes destroys life at an early period. Its features are characteristic: the lung-substance has a peculiar doughy feel; pits on pressure; is wanting in healthy crepitation; and has a pale pink colour very closely resembling that of a calf's lung. It has been described as 'large-lunged vesicular emphysema.'

ETIOLOGY.—*Determining causes and mechanism.*—With reference to the determining causes of emphysema, there are two theories, namely, the inspiratory theory, and the expiratory theory. On the first view the dilatation and rupture of the air-sacs are accounted for by the over-distension of the lungs in inspiration. On the second view these changes are considered to be caused by the strain to which the lung-tissue is subjected in violent expiratory efforts, especially the act of coughing. It has been thought by others that emphysema must be looked upon as a complementary lesion, arising in consequence of the over-distension to which the healthy portions of the lungs are subjected in cases of pulmonary collapse. Without entering into any critical examination of the theories as to the mechanical causes of emphysema, it may perhaps be sufficient to say that there can be little, if any, doubt that the lobular forms of the disease are mainly produced by expiratory efforts, such as violent cough, or blowing wind-instruments. They have their seat in those parts of the lungs which become most distended by such acts. With regard to the lobar form of the disease, however, this explanation of its mechanism does not suffice. In this affection the inspiratory power is that which distends the lungs. The pulmonary tissue has lost a portion of its elasticity, it yields to distension, and no longer reacts perfectly when the distending power ceases. Further distension follows; reaction diminishes still more; until at length in some instances the lungs become greatly enlarged.

In senile cases the loss of elasticity of the chest-walls aids in preventing the pulmonary reaction.

ANATOMICAL CHARACTERS.—In the early stages of emphysema there is simply a dilatation of the air-sacs; an increase in the size of the alveoli; and a diminution in the extent of the alveolar walls, which, yielding with the distending cavities, become partially obliterated. As the disease progresses, the air-sacs become more distended and the walls of the alveoli sometimes completely obliterated, so that the air-sacs are quite smooth, instead of honey-combed. Then follows perforation of the air-sacs—at first slight, here and there an oval opening being discoverable; afterwards the openings become larger and more numerous. The subsequent progress of the disease is attended with further distension of the air-sacs, and rupture of the fibres of their walls. The openings thus caused coalesce, until at length the walls are simply represented by membranous shreds, and even large vesicles may form. These changes, varying in degree, characterise all the forms of emphysema. In the lobar form, however, perforation takes place to a much greater

extent, relatively to the amount of dilatation, than in the lobular or partial lobular form.

The emphysematous lung is anæmic; and its blood-vessels become widely separated, and often ruptured and atrophied. The bronchial tubes are sometimes dilated, especially in old-standing cases, and in these there is frequently found an increased development of the circular muscular fibres.

There is a form of lobar emphysema which is met with in old age, and which differs in some respects from that already described. The lungs are not so large; they are universally distended, however, to a greater or less extent; and they present a somewhat atrophied appearance. The alterations, of which they are the seat, are probably the result of those changes which age produces in the chest-walls, impairing their elasticity. This loss of elasticity may also affect the lung-tissue.

SYMPTOMS.—A constant and gradually increasing dyspnoea is one of the most important and most frequent of the symptoms of emphysema. Bronchitis, and therefore cough, with expectoration, is generally present. Hæmoptysis is rare, and, when it does occur, is slight. The patient is usually free from pain, but complains of a feeling of oppression, or a 'smothering in the chest.' In severe cases of lobar emphysema this last symptom and the dyspnoea are often the only circumstances which attract the attention of the sufferer to his malady. In other instances, however, and especially when the disease is only partial, a close examination will elicit the fact that there have been bronchitic symptoms. Few cases of emphysema exist for any length of time without the occurrence of asthmatic seizures. In advanced cases the aspect is peculiar. The countenance is dusky, leaden, and puffy. The nostrils are dilated, and expand widely on inspiration, while the angles of the mouth are drawn down. The voice is feeble. The whole body has a cachectic appearance, and is sometimes much wasted. General dropsy often ensues.

The lungs being the seat of general expansion, the thorax is kept abnormally distended. Thus it can undergo but little enlargement at each inspiration. As there is no impediment to the passage of air to the air-sacs, inspiration is accomplished rapidly. Not so, however, with expiration. The lung-tissue has in great measure lost its elasticity, and reacts slowly after distension; and this results in laboured, slow, and ineffectual efforts to expel the air. Further, as the lungs are more or less riddled with perforations, their aerating surface is diminished, and this necessarily causes dyspnoea whenever any increased demand is made on the respiratory function. The quantity of blood circulating through the lungs, even from the earliest stages of the affection, is also diminished; and the destruction of the capillary vessels, which ensues when the disease is more developed, further decreases the vascularity of the pulmonary tissue. Hence its pale, anæmic appearance after death, a circumstance which serves to explain how rarely it is the seat of pneumonic inflammation.

PHYSICAL SIGNS.—Among the most important of the physical signs of emphysema are the following: The upper part of the chest and the clavicles are prominent; the neck seems shortened; the fossæ above the clavicles are deepened; there is increased curvature of the dorsal spine; and the sternum is arched. The gait is stooping; the ribs are prominent; and the intercostal spaces are de-

pressed. There is indeed a general increase in the size of the chest, usually most marked at the upper part. These are the features of the disease when it is extensive. If partial, or confined to one lung or part of a lung, the prominence of the chest exists on one side only, and the other symptoms and signs are less marked. The movements of the chest in respiration are peculiar. The breathing is for the most part superior thoracic, but the chest is not much expanded on inspiration, for the lungs are already inordinately distended. The lower end of the sternum and the lower ribs are drawn in during inspiration. In some cases during inspiration there is marked protrusion of the abdomen. The respiration presents other features. The inspiration is short and quick, and is followed by a prolonged and often wheezy expiration. Coughing is performed feebly, and expectoration is attended with difficulty. Percussion and auscultation elicit important diagnostic marks of the disease. When it is general there is increased, and in some instances almost tympanitic, resonance over the whole of the chest, most marked towards the apices of the lungs, and along their anterior borders; and in partial cases almost confined to these spots, or to one side. The præcordial region is generally resonant, owing to the distended lungs coming between the heart and the wall of the chest; and the cardiac impulse can often be felt beneath the lower end of the sternum. The respiratory murmur is faint, and characterised by peculiarities which a knowledge of the anatomical condition of the lungs and of the chest-walls enables us to explain. The inspiratory murmur is short, and is followed by a prolonged expiratory murmur. This latter is unlike the sound heard in any other affection, and is, in fact, pathognomonic of emphysema. In some advanced cases the respiratory sounds are scarcely audible, if the bronchial tubes are free from mucus, and no spasm exists. A *râle* is often heard in emphysema, but it is not a 'dry *râle*.' It is probably produced in the finest bronchial tubes, and is a modification of the sub-crepitant *râle* of bronchitis. Although valuable in aiding diagnosis when present, yet, from its frequent absence and the difficulty of distinguishing it from the ordinary sub-crepitant *râle*, it loses much of its diagnostic import.

COMPLICATIONS AND SEQUELÆ.—Bronchitis is one of the most frequent of the diseases associated with emphysema of the lungs. It is rare for the latter affection to exist for any length of time without the supervention of the former. Bronchitis presents some peculiarities when it affects an emphysematous lung. It is rather the result of congestion than of inflammation. It often attacks the finer bronchial tubes; and, when severe, is attended with profuse secretion—a circumstance which, coupled with the fact that expectoration is less easily accomplished than when the lungs are healthy, seriously complicates the affection, and increases the danger of death from asphyxia. The inflammation sometimes runs on very rapidly, and copious purulent or puriform expectoration occurs. Even when this is the case, an examination of the tubes after death may reveal but little vascularity of the mucous membrane. These severe bronchitic attacks are very apt to be attended by the formation of fibrinous clots in the heart and the large vessels arising therefrom. Bronchitis, in a subacute or chronic form, is a very constant cause of winter cough in emphysematous patients.

Asthma, occurring with greater or less severity, is a frequent attendant on emphysema. The attacks come on for the most part during the night, and may possibly be due to the congestion of the lungs which takes place during sleep, or when the body is long in the recumbent posture. This congestion probably sets up an irritation, which gives rise to reflex spasm of the bronchial muscular fibres.

Secondary affections of the heart are constantly met with in advanced cases of emphysema. Many pathologists have believed that the right cavities alone become affected; but more recent observations have shown that the cardiac disease is not confined to one side. There is, in extensive emphysema, a general hypertrophy of the heart, with dilatation of all the cavities, especially of the ventricles. But hypertrophy is not the only change which takes place—valvular disease is frequently found. The deposits and thickening which occur about the valves are no doubt secondary to the changes in the muscular walls, and must be attributed to the general malnutrition produced by the disease. It is not difficult to understand how it happens that in emphysema there is general cardiac hypertrophy. The impediment which exists to the circulation of the blood through the lungs necessarily gives rise to an overloaded state of the right side of the heart; hence result increased action of the right cavities, and hypertrophy of their walls. Again, the overloaded state of the venous system, and the consequent impediment to the capillary and arterial circulation, call for increased action of the left ventricle; and this is followed by its dilatation and thickening. There exists also another cause, which probably has some influence in producing this cardiac hypertrophy, namely the altered position of the heart. This organ is pushed downwards, and its impulse is often felt strongly in the epigastrium. The position of the ventricles is therefore changed, and the direction of the axis of their cavities is altered with reference to that of their great vessels. This must lead to embarrassment of the circulation.

As a consequence of the changes in the heart and venous system in emphysema, dropsy often results. Many cases go on for a long time without any dropsical symptoms, while in others there is only slight oedema of the legs. In advanced cases, however, there is frequently general dropsy complicated with albuminuria.

General emphysema is attended in its progress by symptoms of cachexia and anæmia. In some cases there is much wasting of the muscular system, even before dropsical effusions occur. Further, the patients often have a sallow and anæmic appearance, not unlike that met with in renal and other serious organic diseases. There has been an impression that emphysema and phthisis are incompatible diseases, but recent researches have shown that this view is not correct. Indeed, in most cases of death from phthisis, patches of emphysema, involving the whole or part of a lobule, are met with; and doubtless have been produced by the fits of coughing so common in the disease. But the great question is whether tubercular deposit ever takes place in lungs which are the seat of lobar emphysema; and this question must be answered in the affirmative, although the concurrence of the two diseases is uncommon. Pneumonic consolidation is very rare in an emphysematous lung.

Pleurisy not infrequently exists in connection with emphysema; pleuritic adhesions being often

found after death. The occurrence of pleurisy must, however, be considered as an accidental circumstance. In the most extensive cases of emphysema pleuritic adhesions may not be found.

PATHOLOGY.—When the disease is partial, and has followed or is attended by bronchitis, or some other affection in which there has been violent or long-standing cough, the emphysema may be the result of mechanical violence, without pre-existing degeneration of the lung-tissue. When, however, it is of the lobar form, degeneration is probably the primary step in the affection. The facts which tend to confirm this view are: (1) the insidious manner in which the disease sometimes comes on, and the development which it attains, without any previous history of violent or long-standing cough; (2) the frequency with which it attacks the whole of both lungs; and (3) its hereditary character. The exact nature of the degeneration has not been satisfactorily made out. Fatty matter has been found in a few instances, but not in all cases. The degeneration is probably one primarily involving the elastic fibres and other structures of the walls of the air-sacs. Whatever be the nature of the degeneration, there can be no doubt that lobar emphysema is a malady resulting from some form of malnutrition of the lung-tissue. There is reason to believe, too, that this form of emphysema is sometimes associated with gout.

TREATMENT.—This must be referred to under two heads, namely (1) the treatment of *the disease*; and (2) that of *the secondary affections*, which follow or are associated with it.

1. *Treatment of the Disease.*—It can scarcely be expected that, when once perforation and rupture of the air-sacs have taken place, the normal condition of the lung can be restored. But, while we admit this, it is by no means implied that the disease is beyond control. That condition of lung-tissue which precedes the perforations—the simple distension of the air-sacs—admits of great amelioration, and further degenerative changes may be, if not prevented, at least much retarded.

All measures which tend to invigorate the system, to give tone to the heart, and to improve the condition of the blood, should be resorted to. Among the remedies for internal administration the most useful is iron. It should be given in small and continued doses. Quinine is valuable, as are also the various bitters and other remedies for dyspepsia, from which emphysematous patients often suffer. Cod-liver oil is very useful in some cases. Strychnine has been recommended with the view of improving the tone of the muscular fibres of the bronchial tubes. It has not been found useful in this respect; nor need we wonder at this, for the disease is one primarily of the air-sacs, and not of the bronchial tubes, and, if the muscles of the latter are secondarily affected, it is rather with spasm than paralysis. Small doses of strychnine given for dyspeptic symptoms may be useful. In some cases iodide of potassium has apparently been beneficial. Probably these cases have been associated with gout.

Breathing compressed air has been strongly advocated, and no doubt it has afforded in some cases decided temporary relief, and good effects are said to have followed its use. See AIR, Therapeutic Use of.

The regulation of the diet, and the general management, are most important. The diet should

be nourishing, and a moderate amount of stimulants should be allowed. The food should be easy of digestion, and nutritious in proportion to its bulk. The stomach should never be overloaded, as that condition will give rise to dyspnoea. Errors of diet must be avoided, and the functions of the bowels should be carefully regulated. Another point is to give the lungs as little work as possible, and to let the patient breathe a pure air. All violent exercise, or physical exertion of any kind, must be strictly prohibited; moderate exercise is, however, to be recommended. Moderate walking, yachting, carriage-exercise, riding at a quiet pace and on an easy horse, are important adjuvants in the general treatment of emphysema. The condition of the skin should be carefully looked to; warm clothing should be constantly worn; and the greatest care should be taken to ward off bronchial inflammation. Residence during the winter in a warm and dry climate is to be recommended.

2. *Treatment of Secondary Affections.*—Among the most important of the affections secondary to emphysema is bronchitis. No depressing measures should be used in this disease, but such as will promote expectoration, and check the secretion of the bronchial tubes, if, as is very frequently the case, this be excessive. Ammonia, the various stimulating expectorants, and iron are the most valuable remedies, together with moderate counter-irritation (*see BRONCHITIS, Diseases of*). The dyspnoea attendant on emphysema admits only of palliative treatment, but is often greatly improved by the observance of the rules laid down for the general management of the disease. The dyspnoea is always increased by the presence of bronchitis; by the stomach or bowels being overloaded; and by the general over-distension of the venous system, which necessarily ensues as a consequence of the impediment to the flow of blood through the lungs. Care should be taken to prevent any flatulent distension of the stomach or intestines, and to keep up a good action of the bowels, liver, and kidneys. For the relief of the asthma which frequently exists in connection with emphysema, full doses of iodide of potassium are often useful. Stramonium may also be smoked, and other measures beneficial in ordinary spasmodic asthma may be tried (*see ASTHMA*). In reference to the treatment of the dropsical symptoms, which follow as a secondary consequence of emphysema, the reader is referred to the articles DROPSY; and HEART, Dilatation of.

B. *Interlobular Emphysema.*—This condition, in which the excess of air in the lungs is contained, not in the air-sacs and alveoli, but in the connective tissue between the lobules, is described under the head of EMPHYSEMA, SUBCUTANEOUS, to which article the reader is referred.

A. T. H. WATERS.
T. R. BRADSHAW.

LUNGS, Gangrene of.—SYNON.: Fr. *Gangrène du Poumon*; Ger. *Lungenbrand*.

DEFINITION.—Death of a portion of the substance of the lungs.

Gangrene of the lungs is of two kinds, namely, (1) *diffused*, in which the whole of one lobe or lung is affected; and (2) *circumscribed*, in which a portion only of a lobe undergoes gangrenous change.

ÆTIOLOGY.—Gangrene is sometimes a result of acute pneumonia, or of the local action of other toxins. It has been known to follow the inhalation

of noxious gases. Its occurrence is favoured by extreme constitutional depression, or by pressure interfering with the circulation and nutrition of the lung. An aneurysm or mediastinal growth, pressing upon the main arterial, venous, and nervous trunks at the root of the lung, is perhaps the most common antecedent of diffused gangrene; whereas the more circumscribed form of the disease is a sequela of acute and limited pneumonia, cancer, bronchiectasis, or rapid phthisis in a debilitated subject. In pulmonary apoplexy and other operations on the mouth septic infection may lead to gangrene, the gangrene being limited to the portion of lung involved in the original extravasation of blood. Pneumonia caused by a foreign body in the air-passages is apt to run on to gangrene. The diffused gangrene that occurs in drunkards and lunatics, in measles, small-pox, and typhus, evidences excessive nerve-prostration and loss of nutritive power. In children, gangrene of the lung sometimes follows cancrum oris, as well as the eruptive fevers.

ANATOMICAL CHARACTERS.—The colour of a gangrenous lung is dark, dirty olive, or greenish-brown. It is moist or even wet; and either of the consistence of engorged lung, or softer and more diffident. The odour is that of external gangrene or decomposed flesh, and is distinctive during life, rendering the room in which the patient lies horribly offensive. When scattered patches of gangrene occur, there is often in one part a solid mass of greenish lung-tissue, and in another a central sloughy or gangrenous cavity, surrounded by a broad rim of soft infiltrated lung. The seat of circumscribed gangrene is usually the periphery of the lung and the lower lobes. If a bronchus open into the gangrenous patch, inflammation of the bronchial membrane is set up. In rare cases the pleura is involved, and pyo-pneumothorax is induced. Sometimes the pulmonary arteries are found plugged, and more often the bronchial arteries. In those rare cases in which recovery takes place, interstitial pneumonia is set up, leading to encapsulation of the gangrenous spot; the sloughs are ejected; and cicatrization follows, as in pulmonary abscess. *See LUNG, Abscess of.*

In diffuse gangrene the whole of one lung is sometimes involved. The pulmonary tissue is then converted into a black putrid substance, saturated with blackish purulent fluid; or the gangrenous part merges gradually into oedematous or hepatized tissue.

Embolism, arising from the introduction of septic matter into the veins, and leading to abscess in various organs, may follow either form of gangrene. Secondary gangrenous change is frequently met with in other parts of the same or in the opposite lung.

SYMPTOMS.—It is seldom possible to diagnose gangrene of the lung until the purulent discharge reaches a bronchus and is ejected. Then the sputa are found to separate speedily into layers—a superficial froth, a liquid middle layer, and a lower sediment. The smell of the sputa and breath is excessively fetid. The dyspnoea and prostration are usually great. The physical signs are those of softening and excavation, percussion being either dull or tympanitic, and loose crepitation being soon replaced by gurgling and perhaps amphoric breathing. The passage of the circumscribed into the diffused form may be traced by watching the physical signs.

DIAGNOSIS.—Suppurative ulceration of the bron-

chial cartilages gives rise to great foetor of breath. Sometimes a gangrenous odour in the breath occurs when the putrefactive change is limited to the secretions; and it may be present also in pyo-pneumothorax with internal fistula. These several diseases must be excluded by a careful estimation of the history and the physical signs. See BREATH, The.

PROGNOSIS.—The prognosis of pulmonary gangrene is hopeless in the diffused form; and is doubtful, even when the symptoms or signs point to a limitation of the mischief.

TREATMENT.—Every attempt must be made to support the strength by nourishing food and stimulants; bark and ammonia, quinine and acids, iron and cod-liver oil being indicated. The inhalation of creosote or carbolic acid in spray may be tried, or of turpentine on hot water (p. 745).

When a gangrenous abscess of the lung exists, and it is evident that the passage of foetid matters through the bronchi is setting up dangerous irritation, leading to exhausting discharge, or threatening to poison the system, the question of draining the gangrenous cavity should be entertained. The introduction of a drainage-tube sometimes affords immediate relief in such cases. The foetor of breath ceases; the offensive secretions, being no longer locked up in the lung, lose their putrescent character; and the relief to the constitution is great. An accurate diagnosis is in such cases essential, the danger of the operation being somewhat enhanced if the abscess is at a distance from the chest-wall, and if the lung is not adherent to the costal pleura. For a detailed account of the necessary operation see LUNG, Abscess of.

Besides the general treatment which co-existing disease may require, special attention must be given to the removal of the gangrenous odour from the atmosphere of the room, from the sputa, and from the patient, who is apt to exhale from the skin a similar odour to that given off in the breath. Sulphurous acid, carbolic acid, or chlorinated lime fulfils the first indication; Condy's fluid the second; and the latter may also, when diluted, form a useful wash or gargle. See INHALATIONS.

E. SYMES THOMPSON.

LUNGS, Hæmorrhage into.—SYNON.: Extravasation of Blood into the Lungs; Pulmonary Apoplexy; Fr. *Hémorrhagie du Poumon*; Ger. *Lungenblutung*.

ÆTIOLOGY AND PATHOLOGY.—In the article HÆMORRHAGE will be found enumerated the causes which lead to pulmonary hæmorrhage. In the present article only those extravasations of blood into the lungs are included which do not depend upon direct injury to the organ, or upon exposure and rupture of vessels in the course of destructive disease affecting it.

Hæmorrhage into the substance of the lungs may be *diffuse*, *punctiform*, or *circumscribed*.

(1) *Diffuse pulmonary apoplexy.*—The diffuse extravasation of blood into the lungs is an extremely rare condition. Some cases have been recorded, however, in which it has arisen from primary disease of a branch of the pulmonary artery. The lung-tissue is broken down by the hæmorrhage into it; and the patient soon succumbs.

(2) *Punctiform hæmorrhage*, and (3) *circumscribed or nodular pulmonary apoplexy*, are not of uncommon occurrence, and are attendant upon the same morbid conditions of the lung. Mitral disease

—mitral stenosis especially, but also mitral regurgitation—is the chief remote cause of these two forms of pulmonary apoplexy.

In the so-called 'hæmorrhagic diseases of the blood,' punctiform hæmorrhage in the lungs is possible; but petechiæ more frequently affect the pleural surface or bronchial mucous membrane (see HÆMORRHAGE). Mechanical congestion of the lungs, from the above-mentioned forms of heart-disease, is by far the most common condition upon which this minute and interstitial form of hæmorrhage supervenes, giving rise to no additional symptoms, but causing considerable and peculiar pigmentation of the lung. The interstitially thickened lungs acquire a brownish tint, from the absorption of the blood-spots, leaving hæmatin behind; and the appearance has given rise to the term *brown induration* of the lungs. See LUNGS, Hyperæmia of.

Nodular or circumscribed pulmonary apoplexy is often associated with the petechial hæmorrhage just described, and, like it, mostly supervenes upon the mechanical congestion of the lungs arising from heart-disease. There are two ways in which this form of hæmorrhage may be produced. The first way is by rupture of capillaries or small veins under the heightened pressure of the pulmonary circulation. An effusion of blood thus occurs, which fills up one or more lobules, and coagulates to form the dark firm consolidations so characteristic of the lesion. Or a branch of the pulmonary artery becomes obstructed by an embolus, for instance, by a fragment of coagulum conveyed from the right auricle, or from one of the systemic veins (phlebitis, varicose veins); and its territory becomes at once filled with extravasated blood. See EMBOLISM.

ANATOMICAL CHARACTERS.—A lung that is the seat of this form of hæmorrhage is usually toughened and heavy. Some hard, and more or less square, flat surfaces may be felt and seen raised above the general surface of the lung, which has shrunk below their level. The pleura covering such patches is darkened in colour, and presents flakes or granulations of lymph, impairing its translucency and smoothness. On making a vertical section through one of the surfaces, it is found to form the base of a more or less conical mass, which has a firm section like damson-cheese, and is sharply defined from the surrounding tissue. In its axis is seen a branch of the pulmonary artery, occupied by partially altered clot. There is usually some staining of the pulmonary tissue immediately surrounding the apoplectic nodule, from imbibition. Such hæmorrhagic nodules vary greatly in number and size; there are usually several in each lung, of about the size of a walnut; but one may occupy a whole lobe. They also vary in appearance according to the date of their occurrence; their colour, at first that of dark blood-clot, passes through pale chocolate to yellowish-red or pale yellow, as the colouring matter becomes gradually absorbed. The whole extravasation may be gradually and completely absorbed, leaving the restored lung but little damaged; or a shrunken fibrinous deposit or blood-cyst, with surrounding induration, more rarely a calcareous centre, may mark the site of former hæmorrhage. It should be added that these extravasations, although generally near the pleural surface of the lung, are not always so, but may occur deeply in its substance; they occur more frequently in the right lung, and generally in the upper lobe.

SYMPTOMS.—Amid the symptoms attendant upon the conditions leading to pulmonary apoplexy, it is difficult to single out any diagnostic of this special lesion. An exacerbation of dyspnoea already terrible enough, or a sudden failure of pulse, may perhaps be noted. Dark scanty hæmoptysis is, however, the pathognomonic sign, the frothy mucous expectoration containing some streaks, or small clots, of dark coagulated blood. Some circumscribed patches of dulness, with bronchial breathing and neighbouring crepitation, may perhaps be made out, especially in the mammary and mid-axillary regions.

PROGNOSIS AND TREATMENT.—These lesions are among those which often close the scene in the heart-disease to which they are accessory; and are therefore irremediable. Sometimes when, from any cause, their occurrence appears to have been hurried forward; when the lividity is great, the dyspnoea urgent, and yet the disease is not of long duration; wet-cupping or bleeding from the arm will certainly give temporary relief, and perhaps avert immediate danger.

R. DOUGLAS POWELL.

LUNGS, Hydatids of.—**SYNON.** : Fr. *Kystes Hydatiques du Poumon*; Ger. *Lungenechinococcus*.

DEFINITION.—A disease due to the presence of hydatids in the lungs.

ÆTIOLOGY.—Hydatid cysts in the lungs rarely occur in this country as a primary disease of these organs, but they are not infrequently met with as an extension of disease from the liver.

In Australia, where the affection is very common, it is sometimes met with in the lung without any other organs being affected. Of 100 cases, the liver was the organ affected in 70, the lung in 12.

ANATOMICAL CHARACTERS.—Single sacs or acephalocysts (p. 461) are by far most usual, varying in size from a pigeon's egg to a man's fist. Sometimes the upper and sometimes the lower lobes are the seat of the cysts.

If the parent-sac be destroyed by inflammation and consequent suppuration, a communication is established between the cavity and the bronchi, through which the daughter-cysts may be ejected. Not infrequently the pulmonary sac communicates with a similar sac in the liver. It is natural to infer in such cases that the disease originated in the liver. Indeed, it has been stated that primary hydatid of the lung is unknown.

SYMPTOMS AND PHYSICAL SIGNS.—Hydatids may exist in the lung for a considerable time without giving rise to any noticeable symptoms; but as the tumour enlarges and presses upon the surrounding tissues, hæmoptysis occurs, as also bronchitis, pneumonia, or even gangrene. Sometimes the cysts perforate the pleura and cause pneumothorax.

If the site of the tumour be superficial, altered breath-sounds and percussion-note may be observed; but if it be deeply seated, the physical signs may escape detection. If bronchitis or pneumonia be set up, the signs and symptoms of these disorders mask those of the originating disease. Often the symptoms are like those of rapid phthisis, namely, cough, muco-purulent expectoration, hæmoptysis, night-sweats, and emaciation. The meaning of these symptoms is apparently confirmed by the physical signs, namely, dulness on percussion; absence of breath-sounds, or prolonged expiratory murmur; and, when the cysts burst, gurgling and pectoriloquy. Unless the daughter-cysts or hook-

lets are expectorated, there is nothing to point unmistakably to the nature of the disease. When perforation of the diaphragm occurs, hepatic symptoms or those of pleurisy supervene. The patient looks anxious; the features are collapsed; the skin is clammy and livid; the extremities are cold; incessant paroxysmal cough occurs, with vomiting; and by degrees sallowness and jaundice make their appearance. Symptoms of acute pneumonia may occur—of consolidation, followed by excavation; the expectoration being at first rusty, then bile-tinged, muco-purulent, and fetid, and containing, besides shreds of lung-tissue, entire cysts or portions of them.

DIAGNOSIS.—It may be difficult to distinguish a large hydatid cyst from pleuritic effusion, as the lung may be displaced, the chest bulges, and the intercostal spaces become prominent and fluctuating. The rounded outline of the dull space, the absence of acute symptoms, the history of gradual onset, the absence of ægophony and of alteration of physical signs on change of posture, will guide the decision; and an exploratory puncture, which gives exit to a clear saline non-albuminous fluid, containing possibly hooklets or fragments of cysts, will confirm the diagnosis. The conduction of the heart-sounds and impulse, and the tense unyielding condition of the bulged side on palpation, may lead to the suspicion of mediastinal tumour; but in the case of hyatids there is seldom any visible venous engorgement, or laryngeal or cesophageal pressure-sign, as in aneurysmal or other mediastinal growths. There is, moreover, generally a freedom from cachexia or constitutional disturbance. In circumscribed abscess the neighbouring lung is rarely so free from disease as in hydatid.

PROGNOSIS.—Although the symptoms may be so severe as to threaten immediate death from suffocation, recovery occurs in at least half the cases in which hydatid disease begins in the lung, and one third of those in which it spreads from the liver. If the cyst is allowed to burst of itself, recovery takes place in from 30 to 40 per cent. of cases. But the mortality is greatly reduced by early operation.

TREATMENT.—The surgical treatment of hydatid of the lung should be undertaken as soon as the disease is diagnosed because the risks involved in delay are greater than those dependent on surgical interference. If left untreated the patient is liable to (1) suffocation from rupture of the cyst into a bronchus; or (2) a probably incurable hydro-pneumothorax from rupture of the pleura. It is seldom permissible to *aspirate* a pulmonary hydatid which has not suppurated, because this procedure has more than once been followed by the simultaneous rupture of the cyst into a large bronchus, and the consequent suffocation of the patient from the rapid flow of large quantities of hydatid fluid into the air-passages. The position of the cyst having been localised a free opening should be made by removing two or three inches of two or possibly three ribs, great care being taken not to open the pleura. Fully curved needles threaded with silk or catgut should then be passed through the pleura into the subjacent lung in a continuous series round the margin of the circle of pleura exposed. It is possible if the cyst be thin that the sutures may cut through and the fluid may leak out alongside them, but this does not involve any serious risk, whereas opening the pleural cavity without thus stitching up the lung may result in the production of a pneumothorax.

If the cyst be old and thick-walled there is no chance of this happening, nor would it occur if lung-tissue intervene between the hydatid and the surface. As soon as the cyst or the lung is fixed to the parietal pleura it may be opened by a free incision and drained like any other pulmonary abscess. This treatment is especially applicable to those cases in which suppuration has occurred. Convalescence is likely to be slow. The operation may, if desired, be performed in two stages.

It must be remembered that hydatids of the lung are not infrequently multiple, and that at least in the experience of one of the present writers (R. J. G.) it is not very unusual for tuberculosis to occur in a lung which has been operated on for hydatids.

E. SYMES THOMPSON.

R. J. GODLEE.

LUNGS, Hyperæmia of, Passive.—Passive hyperæmia of the lungs may be conveniently divided into two varieties: (1) that occurring in debilitated subjects and due to general causes (*Hypostatic Congestion*); and (2) that resulting from obvious mechanical obstruction, such as mitral stenosis (*Mechanical Congestion*), and leading to Brown Induration.

Hypostatic Congestion.—**ÆTIOLOGY.**—Passive or hypostatic congestion of the lung affects the most dependent parts. Failure of heart-power and dilatation of the pulmonary capillaries are the chief causes of this condition. In states of exhaustion from low fevers, especially typhus and typhoid; after severe surgical operations; in extreme old age; or towards the end of prostrating illness, this failure of heart-power, and consequent stagnation of blood in the lower parts of the lung, usually the bases, is apt to supervene, and is one of the common modes of death. An altered condition of the blood, due to the presence of toxins, is possibly a contributing cause of hypostatic congestion. The site of the congestion is largely influenced by gravitation.

ANATOMICAL CHARACTERS.—Passive hyperæmia almost always affects the bases of both lungs, although often not in an equal degree, the difference depending mainly upon the position of the patient during the last days of life. The affected lung is dark-coloured, and engorged with dark blood. Its tissue is more or less deeply stained with blood, oedematous, and is less crepitant than natural, tearing also more readily than natural under the pressure of the finger. If thoroughly washed, however, in a gentle stream of water, the lung-texture will be found to be but little altered. This condition very readily passes into a low form of pneumonia, and thus portions of the lung may be found consolidated, having much the appearance and consistence on section of a congested spleen (splenification, hypostatic pneumonia). The bronchial tubes and pleura are affected by *post-mortem* staining.

SYMPTOMS.—The symptoms of this form of congestion of the lungs are lividity, especially of the lips and extremities; and quickened shallow breathing; superadded to those of extreme prostration. Dullness on percussion, with enfeebled breathing and moist crepitant *râle*, due to oedema, are found over the bases of both lungs, but in greatest extent on that side to which the patient has been inclining.

TREATMENT.—The treatment consists in vigorously supporting the patient by alcoholic stimulants

frequently administered, and with nutritious food. Nutritive enemata are often of great value. In all exhausting diseases this condition should be anticipated, and warded off if possible by timely support and stimulants, and by frequently turning the patient from one side to the other. Of medicines, ammonia, ether, strychnine, bark, quinine, and oxygen-inhalations are of the greatest value.

Mechanical Congestion.—**ÆTIOLOGY.**—The obstruction may be at the mitral orifice, as in mitral stenosis or regurgitation; or in the left ventricle, when this cavity is dilated and imperfectly emptied, as in the advanced stages of constrictive or regurgitant aortic disease. Whether there be an absolute narrowing of the blood-channel between the pulmonary and systemic circulations—that is, at the mitral orifice or at the commencement of the aorta; or whether, from enlargement of the mitral orifice or from disease or injury of its valve, regurgitation be permitted, so that each contraction of the right ventricle is met and opposed, more or less, by a counter-rush of blood from the left ventricle—in any case, and still more in the combination of two or more of these causes, it is clear that the pulmonary circulation can only go on at an increased pressure by the contraction of the right ventricle becoming more vigorous; and that hyperæmia must result from the damming back of the blood through the pulmonary veins.

Of the causes named, mitral constriction is that which leads most readily to obstructive hyperæmia of the lungs.

ANATOMICAL CHARACTERS.—Obstructive hyperæmia affects the whole of both lungs. The result of the heightened blood-pressure from increased force of injection into the lungs, to overcome an impediment to the escape of blood from them, is most felt in the pulmonary capillaries. These capillaries gradually become lengthened, tortuous, and dilated even to three times their normal dimensions; the fibrous tissue is increased; the thickened and tortuous capillaries intrude upon the air-spaces; and the elasticity of the lungs is diminished. Sometimes minute hæmorrhages take place into the parenchyma of the lung; sometimes larger escapes of blood fill the alveoli of circumscribed patches (*pulmonary apoplexy*). The total result of the intimate changes described is a uniform increase in the size and weight of the lungs, with an increased density and toughness. On section, the lungs are found to be more pigmented and solid-looking than natural, sometimes of a brownish hue (*brown induration*); they are, however, crepitant throughout, except here and there, where they may present fibrous foci or firm, dark, damson-cheese-like patches, fading to brown-red, representing various results and stages of hæmorrhage. There may be some oedema present. The pulmonary arteries and veins are enlarged and congested; and the bronchial mucous membrane is usually the seat of chronic catarrh. Patches of atheroma are frequently to be seen in the larger branches of the pulmonary artery.

SYMPTOMS.—Dyspnoea and cough, both brought on or increased by effort, with palpitation, and oppression or tightness, usually referred to the epigastrium, are the most constant symptoms of obstructive hyperæmia of the lungs. Patients suffering from this condition have repeated attacks of bronchial catarrh, and hæmoptysis is of common occurrence. The hæmoptysis may be considerable, but

more usually the expectoration is streaked with blood or contains small dark coagula.

This form of hyperæmia commonly occurs before middle life, during the usual period of mitral heart-disease. The signs of heart-disease, and most often of constriction of the mitral valve, are present. The subjects of this affection are often undersized and badly nourished: the pigeon-breasted type of chest being common, especially in those cases in which the disease manifests itself early in life. A small, frequent pulse; more or less lividity of lips; and other signs and symptoms of the cardiac disease, of which the pulmonary condition is the consequence, are to be observed. A fine inspiratory crepitant *râle* over the lungs may be heard. During the repeated bronchial catarrhs, with increased pulmonary hyperæmia, to which such patients are especially prone, all symptoms are much aggravated.

DIAGNOSIS.—The existence of obstructive heart-disease suggests the presence of corresponding hyperæmia of the lungs. The fine crepitant *râle* and the hæmorrhagic symptoms and signs more positively point in the same direction.

PROGNOSIS.—The prognosis rests chiefly upon the heart-condition present. Increasing frequency of catarrhal complications, and especially of hæmoptysis, shows the turning of the balance against the patient. The condition may in favourable cases continue for years, especially when dependent upon simple constriction of the mitral valve.

TREATMENT.—The treatment is essentially that of the heart-disease; with the avoidance of all causes which quicken respiration, and which tend to produce catarrhs, to which these patients are so especially prone. R. DOUGLAS POWELL.

LUNGS, Hypertrophy of.—This term is applied to enlargement of the lungs with increased functional power, met with as a compensatory affection of one lung, or of a portion of one lung, to make up for more or less loss of pulmonary tissue by disease.

The dimensions of the enlarged part will be shown by an extension of the normal physical signs. In this way the change can be distinguished from that occurring in emphysema.

LUNGS, Induration of.—See LUNGS, Inflammation of; and Passive Hyperæmia of.

LUNGS, Infarction of.—See LUNGS, Hæmorrhage into.

LUNGS, Infiltrations of.—See PNEUMONIOSIS.

LUNGS, Inflammation of.—SYNON.: Pneumonia; Fr. *Pneumonie*; Ger. *Lungenentzündung*.

DEFINITION.—The term 'pneumonia' has been employed simply to designate inflammation of the lung-tissue. Inflammatory processes in the lungs, however, occur under such diverse circumstances, and are accompanied by such diverse clinical phenomena and histological changes, that 'pneumonia' used in this sense includes widely different diseases.

VARIETIES.—Pneumonias are divisible into the following varieties: A. **Acute Pneumonia**, including *secondary pneumonia*; B. **Broncho-Catarrhal, or Lobular Pneumonia**; and C. **Chronic or Interstitial Pneumonia**. In ad-

dition to these there are those pulmonary inflammations which lead to the formation of *abscess*.

There are certain other forms of lung-consolidation which have sometimes been described as pneumonic, but which are, for the most part, non-inflammatory in their nature, and will, therefore, be only briefly alluded to in the present article. These are: (1) that condition of collapse and hyperæmia, mainly due to weak inspiratory power, feeble circulation, and gravitation, which is so common in the more dependent portions of the lungs in many acute and chronic diseases. (2) Consolidations of the lung resulting from mechanical congestion and embolism, such as are met with in certain diseases of the heart.

The several varieties of pulmonary inflammation must now be considered separately.

Pulmonary abscess is separately described. See LUNG, Abscess of.

A. Acute Pneumonia.—SYNON.: Fr. *Pneumonie Aiguë*; Ger. *Croupöse Pneumonie*.—This is pneumonia *par excellence*. It is sometimes termed *croupous pneumonia*, from the supposed resemblance of the histological process to that of croup. It is also known as *lobar pneumonia*, inasmuch as a large area of the lung is usually involved in the inflammation.

DEFINITION.—Pneumonia may be described generally as an acute infective disease, characterised clinically by sudden onset, severe febrile symptoms, cough, expectoration, and dyspnoea; by the physical signs of pulmonary consolidation; and by a rapid abatement of the general symptoms between the fourth and tenth days. Anatomically it is characterised by an acute inflammation of the lung-tissue, and by the accumulation of the inflammatory products within the alveoli, which products consist, in the main, of a fibrinous exudation and leucocytes.

ÆTIOLOGY.—*Age.*—No age is exempt from pneumonia. It is met with between the ages of one and five years. Here, however, it is liable to be confounded with broncho-pneumonia and with collapse of the lung, so that the results of statistics are less reliable at this than in the subsequent periods of life. It may be stated, notwithstanding, that pneumonia is less common during infancy than has been generally supposed, and that among the pneumonias which are so frequent during this period of life the broncho-catarrhal forms preponderate. After the age of five years the liability to pneumonia diminishes, but it again becomes exceedingly frequent between the ages of twenty and fifty, during which period the liability to the disease reaches its maximum. It is also quite common in old age.

Sex.—In adults more males than females suffer; probably owing to the former being more exposed to atmospheric influences. In early life this difference does not obtain.

Social position, &c.—Pneumonia is more common among the poor and badly fed, and in those whose occupation necessitates an irregular mode of life and great exposure, than among the upper classes of society.

Constitution and health.—Those who are constitutionally weak, and those whose vitality has been impaired by intemperance, insufficient food, anxiety, overstrain, or other causes, are more prone to the disease than the strong and vigorous.

Climatic influences.—Conditions of weather and climate are probably the most important of all known agencies in the causation of pneumonia, and

'catching cold' is the most common exciting cause of the disease. The influence of cold and damp in increasing the liability to acute inflammatory diseases of the chest is well known. This influence is marked in pneumonia, although to a less extent than in bronchitis. Pneumonia is more common in temperate climates than in those regions which are characterised by great heat or extreme cold. Climates and seasons which are liable to sudden changes of temperature, and winds from the north and north-east, appear to be especially favourable to the development of this disease.

Epidemic influences and contagion.—Pneumonia sometimes prevails epidemically; it is occasionally endemic in a house; and evidence tends to show that under certain circumstances, at present unknown, it may be communicated from one person to another. Such facts point to a micro-organism as the causative factor in the production of the disease.

Septic causes.—Sewer-gas emanations appear to play some part in the causation of certain cases of pneumonia. Such cases are said to differ somewhat in type from ordinary pneumonia, and have been termed *pythogenic*.

Previous diseases.—Pneumonia, as is well known, often occurs in those who are the subjects of other disease. It is impossible to speak with certainty as to the relation which subsists between the pneumonia and the disease in the course of which it supervenes. In some cases it may be merely an accidental complication; while in others the previous disease may exercise more or less influence in the causation of the pneumonia (see *Secondary Pneumonia*).

Exciting causes.—In many cases of pneumonia evidence of the existence of any exciting cause is entirely wanting. Of discoverable causes, that which is most common is a sudden chill, or, less frequently, more prolonged exposure to cold or damp. Excluding cold, no conditions can be mentioned which have any marked influence in determining the disease.

BACTERIOLOGY.—Acute lobar pneumonia is an acute specific fever, resulting from the invasion of the lung-tissue by the *Diplococcus pneumoniae*, and its multiplication therein. The organism is present in the exudation filling the lung alveoli, in the sputum, and in the morbid products of such other lesions as may complicate the primary disease, e.g. pleurisy, pericarditis. It is, however, but rarely present in the blood of the general circulation.

Secondary pneumonia is frequently a true lobar pneumonia, presenting the typical appearances of that disease in its acute form, and characterised by the presence of the *Diplococcus pneumoniae* in the exudates. The pneumococcus is present in the saliva of some five or ten per cent. of normal individuals, and it seems probable that in many cases the primary disease simply acts by producing those conditions requisite to enable the saprophytic pneumococcus to invade the lung-tissues of its host and initiate its specific lesions.

Other organisms, such as the *Pneumobacillus* of Friedländer, the *Streptococcus pyogenes longus*, the pyogenic staphylococci, &c., however, act in many cases as the causal agents in the production of the secondary pneumonia, which is then often lobular rather than lobar in type.

The pneumococcus was first isolated by Pasteur in 1880 from the saliva of a case of hydrophobia,

and afterwards from the saliva of healthy individuals. Sternberg demonstrated its presence in the sputa and exudates of cases of pneumonia, although for a long time he was under the impression that the coccus was identical with the organism Friedländer had isolated from similar situations. The honour of pointing out the causal relationship of the coccus to pneumonia belongs to Talamon, while Fraenkel in 1885 completed the chain of evidence necessary to establish the specificity of the pneumococcus.

The *Diplococcus pneumoniae* when observed in sputum, exudation, &c., consists of oval or lanceolate-shaped bodies arranged in pairs or short chains, the individual elements measuring from 0.5 to 0.75 μ in length, and a little less in breadth. Each diplococcus or chain is surrounded by a clear hyaline capsule, composed of a substance closely allied to mucin, which can only be clearly demonstrated by special staining methods. In cultivations of the pneumococcus upon artificial media the cocci are spherical, and although favouring the diplococcus-form frequently occur in chains of varying length, and the capsule is invariably absent. Although under certain conditions the pneumococcus produces resistant forms, these cannot be regarded as true spores.

The pneumococcus is well stained by all the usual aniline dyes, and also by Gram's method (p. 132).

Cultivation.—A really virulent strain of the pneumococcus is only capable of development when cultivated at a temperature approaching that of the body—its range being from 28° C. to 42° C., the optimum 37.5° C.; and then only upon media which are distinctly alkaline to litmus. Its vitality is feeble, cultures in fluid media dying in a few days, those upon solid media in from seven to ten days; upon blood-agar—i.e. nutrient agar upon the slanted surface of which sterile blood has been smeared—however, the coccus will retain its vitality for about two months. The virulence is quickly lost when the organism is cultivated entirely outside the living body.

The pneumococcus grows best upon artificial media when freely supplied with oxygen; it is, however, a facultative anaërobe, and capable of life and growth in an atmosphere devoid of oxygen.

When cultivated in *bouillon* the pneumococcus soon produces a uniform turbidity of the medium; multiplication ceases after 36 or 48 hours, and the medium again becomes clear, owing to the subsidence of the cocci. Growth upon the surface of *agar* appears in 24 hours in the form of delicate transparent spherical colonies resembling minute drops of liquid upon the medium; these later on become more opaque, though they do not increase in size. A smear-culture upon *blood-agar* grows as a thin translucent layer, or as discrete semi-transparent raised colonies, which subsequently become opaque, or take on a buff-coloured tint. Upon the surface of *inspissated blood-serum* the growth occurs in the form of minute jelly-like transparent drops, which in time tend to become greyish-white and opaque. In *litmus-milk* a luxuriant growth takes place, accompanied by the production of an acid reaction, and usually resulting in the coagulation of the casein. No growth takes place upon the surface of potato. Upon gelatine incubated at the room-temperature no growth appears, but if 20 per cent. gelatine is employed and incubated at 37° C. small white

spherical colonies become visible in about 36 hours; these remain discrete, and do not liquefy the medium.

Pathogenesis.—Rabbits and mice are extremely susceptible to experimental inoculation with the *Diplococcus pneumoniae*, whether introduced subcutaneously, intravenously, or into a serous cavity; guinea-pigs are also susceptible, though in a less degree. When highly virulent cultivations are employed death results in from 12 hours to 4 days, from acute septicæmia. *Post mortem*, local reaction is absent or but little marked; with the exception of splenic hypertrophy there are no obvious visceral lesions, but the blood of the general circulation, and of the various organs, is literally crowded with capsulated pneumococci. If less virulent cultures are used the local reaction is more marked, and may consist of extensive fibrinous infiltrations; or pyogenic processes may be initiated, followed, when the subcutaneous tissue is the seat of inoculation, by abscess-formation. After the evacuation of the contents of such an abscess the animal may recover, and will then be found to have acquired a more or less complete immunity towards the pneumococcus.

Cats, rats, sheep, and dogs are highly resistant to experimental infection, and in fatal cases but few cocci are to be detected in the blood-stream; if intrapulmonary injection is practised, pneumonia and pleurisy are produced, and the inoculation is but rarely attended by a fatal result. In so far as his susceptibility to the pneumococcus is concerned, man must be placed in the same category as sheep and dogs.

Fowls and pigeons are immune to the pneumococcus.

ANATOMICAL CHARACTERS.—The changes occurring in the lungs in pneumonia are commonly described as consisting of three stages:—

1. *Stage of engorgement.*—This is the stage of inflammatory hyperæmia and œdema, and it is characterised microscopically by overfulness and slight tortuosity of the pulmonary capillaries, and by swelling of the alveolar epithelium. The lung is of a dark red colour; it is heavier and less crepitant than natural; it pits on pressure; and its cut surface yields a reddish, frothy, tenacious liquid.

2. *Red hepatisation.*—Here there is an exudation of liquor sanguinis and blood-corpuscles. The exuded liquids coagulate within the alveoli and terminal bronchioles, the coagulum enclosing numerous white and a few red blood-corpuscles. The alveolar epithelium is swollen and granular. The lung is now much heavier than in the preceding stage, and is increased in size, so as to be often marked by the ribs. It is quite solid; sinks in water; and cannot be artificially inflated. It is remarkably friable, breaking down with a soft granular fracture. The cut surface has a markedly granular appearance, seen especially when the tissue is torn, and due to the plugs of coagulated exudation-matter which fill the alveoli. The colour is of a dark reddish-brown, often here and there passing into grey. This admixture with grey sometimes gives a marbled appearance. The pleura covering the solid lung always participates more or less in the inflammatory process. It is opaque, hyperæmic, and coated with lymph.

3. *Grey hepatisation.*—This stage is characterised by a continuance of the process of inflammatory

cell-emigration, and by more marked changes in the epithelium. The white blood-corpuscles continue to escape from the vessels, and the alveolar epithelium becomes more swollen and granular. The alveoli thus become more completely filled with young cell-forms, so that the fibrinous exudation is no longer visible as an independent material. The fibrinous exudation now disintegrates, and the young cells rapidly undergo fatty metamorphosis. The alveolar walls themselves, with few exceptions, remain throughout the process unaltered; although very occasionally, when this stage is unusually advanced, they may be found here and there partially destroyed. Owing to these changes, the reddish-brown colour of the lung becomes altered to a greyish or yellowish white. The granular appearance is much less marked; the solid tissue is much softer and more pulpy in consistence; and a puriform liquid exudes from the cut surface of the organ. This stage, when advanced, has been termed 'suppuration or purulent infiltration' of the lung.

Although these three stages of the pneumonic process have been described as succeeding one another in orderly succession, it must be remembered that each stage does not occur simultaneously throughout the whole of the affected area of the lung. The changes advance irregularly, so that while one portion of the lung is in the stage of red hepatisation, another may be in the grey stage—hence the mottled, marbled appearance of the consolidation. The rapidity with which the several stages succeed one another is also subjected to marked variations. In some cases the pneumonic consolidation very rapidly becomes grey, while in others the time occupied in the transition is much longer. These differences will be again alluded to when considering the clinical history of the disease.

Terminations.—The natural and almost invariable termination of the histological process is in *resolution*—the lung gradually returning to its normal condition. This is effected by the fatty and mucoid degeneration, and consequent liquefaction, of the inflammatory products which have accumulated within the alveoli. As the liquefaction proceeds, the circulation in the alveolar walls is gradually restored; the softened products are removed by absorption, and to a much less extent by expectoration; and the lung ultimately regains its normal characters. The other exceptional modes of termination in *gangrene*, *abscess*, and *chronic pneumonia* will be alluded to subsequently.

Site.—The local lesion in pneumonia is in the majority of cases limited to one lung. When double, one lung is usually involved before the other. The right lung is more commonly affected than the left. The part of the lung usually involved is the lower lobe (about 75 per cent.). The consolidation tends to extend upwards and the whole lung may be involved. Pneumonia of the upper lobes is more frequently double than basic disease. It is quite rare for the pneumonic process to commence in two different portions of the lung. When the consolidation is met with in both lungs, or commences in the upper and middle lobes, the pneumonia is often a secondary affection, and has supervened in one whose health has been previously injured, as by alcohol; and such distributions of the local lesion should always make the physician look carefully for evidence of pre-existing disease.

SYMPTOMS.—The incubation-period of pneu-

monia is probably very short. The onset is in the majority of cases sudden, not being accompanied by any premonitory symptoms. Much less frequently, certain premonitory symptoms precede the more severe phenomena which characterise the invasion of the disease. These symptoms include general malaise, headache, chilliness, pains in the back, and loss of appetite.

Invasion.—The invasion in adults is, in most cases, announced by a rigor, much less frequently by vomiting. This rigor is more marked in pneumonia than in almost any other disease. It is usually single, and is rarely repeated, either at the commencement or in the course of the illness. In old subjects the rigor is very frequently absent, and in children its place is often taken by convulsions or vomiting. The rigors or other phenomena marking the invasion of the disease, together with the attendant pyrexia, are usually quickly followed by symptoms pointing to the lung-affection. These symptoms commonly supervene in the course of from twelve to twenty-four hours, although in exceptional cases not until after the lapse of two or three days. The earliest of them are pain in the side, dyspnoea, and cough. These more local symptoms, together with the pyrexia, acceleration of pulse, thirst, and prostration, gradually develop up to the second day of the disease, by which time (and sometimes before this) the pulmonary lesion is usually sufficiently advanced to yield unequivocal physical signs. The general aspect and symptoms of the patient are now tolerably characteristic. The flushed and sometimes dusky face, anxious expression, hurried shallow breathing, hot skin, rapid pulse, short frequent cough, and marked prostration, supervening quickly upon the well-marked initial rigor, indicate pretty clearly the nature of the disease. An herpetic eruption on the lip is very usual and characteristic.

Pain.—The pain in the side, which is increased by deep inspiration and by cough, usually corresponds in situation with that of the affected lung, although it is occasionally experienced in other parts. This symptom may occur coincidently with the rigor, although it more commonly succeeds it. In quite exceptional cases it precedes it, being the first symptom noticed. The pain is due to the implication of the pleura in the inflammatory process; and its severity consequently varies considerably according to the extent and situation of the pulmonary lesion. When this is confined to the central portions of the lung, pain may be entirely absent.

Respiratory phenomena.—Increased frequency of respiration, dyspnoea, and cough, are early and prominent symptoms. The respiration—usually regular—ranges from 30 to 50, and in children reaches even to 70, while the pulse may be only from 90 to 120. This perversion in the pulse-respiration ratio is important in diagnosis. The breathing is shallow; inspiration is abrupt; and, when the pain in the side is severe, respiration is sometimes irregular. The accelerated respiration is accompanied by marked expansion of the *ala nasi*, and by more or less dyspnoea. There is, however, no definite relation between the last-named symptom and the frequency of the respiratory act. Owing to the pain, and to the frequency and difficulty of breathing, speech may be interfered with, and is often rendered exceedingly difficult. Cough is an almost constant symptom, except in the very old. It is short and hacking, rarely paroxysmal like that of bronchitis. It is

usually in the early stages attended by severe pain in the side, so that the patient endeavours to repress it. The cough is usually attended by expectoration; but in children and the old expectoration is often absent, and occasionally such is the case at other periods of life, even throughout the whole course of the illness. The sputa of pneumonia are very characteristic. They are viscid, glairy, and remarkably tenacious, so that they cling to the mouth of the patient, and adhere closely to the sides of the vessel containing them. In colour they present various shades of red, brown, and yellow, owing to the admixture of blood. The appearance so well known as 'rusty' is that most commonly met with. Sometimes they are much more diffuent, and of a dark purple colour, somewhat resembling prune-juice. The characteristic sputa are usually met with on the first or second day of the disease. The amount expectorated is small, and sometimes the pneumonic is associated with a frothy catarrhal sputum. Occasionally a profuse hæmoptysis is an initial symptom. During the period of resolution the sputa become less viscid, and more catarrhal in character, and they usually contain small particles of black pigment. The histological elements met with in the sputa are leucocytes, red blood-cells, pneumococci, and altered epithelium from the alveoli and air-passages; and towards the decline of the disease, fat-granules, pigment, and occasionally fibrinous masses, which are casts of the alveoli and terminal bronchioles.

Pulse.—The pulse in adults usually ranges from 90 to 120, and may be even more frequent. It is commonly much more rapid in children, and less so in the old. In the early stage of the disease it is sometimes full and strong, but soon becomes smaller and easily compressible. It may be irregular, intermittent, or dicrotic. The smallness of the pulse is probably due partly to diminished cardiac power, and partly to the diminished amount of blood which is propelled from the left ventricle, owing to the overloading of the right cardiac cavities which results from the obstructed circulation in the lungs.

Pyrexia.—The pyrexia of pneumonia is continuous, with slight morning remissions and evening exacerbations. The temperature rises very suddenly, with the invasion of the disease, to from 102° to 105° F.; and this high temperature is usually maintained until the period of crisis. This sudden rise and maintenance of a high temperature are very characteristic. The amount of elevation varies in different cases. As a rule, it does not exceed about 104° or 105° F., but temperatures of 107° have been known to terminate favourably. In fatal cases it may reach 109° shortly before death. The maximum temperature is usually met with on the second or third day of the disease, but it occasionally occurs immediately before the crisis. The daily variations are usually as follows: The temperature is lowest about 7 or 8 A.M. In the forenoon, or somewhat later, it commences to rise, and attains its maximum in the early evening. It then falls, but a slight exacerbation occasionally occurs again at midnight, after which it gradually falls. The difference between the highest and lowest temperatures is usually not more than 1° F. The pyrexia runs, for the most part, a uniform course until the period of crisis, when the temperature falls rapidly, in the manner to be hereafter described; but in exceptional cases the course of the fever is more irregular.

Nervous system.—Headache, restlessness, and sleeplessness are almost always prominent symptoms in pneumonia. Slight delirium is also common, especially towards evening, when the pyrexia is at its maximum. Sometimes the delirium is more marked and violent. It constitutes a more prominent symptom in the old, and in the debilitated and intemperate. In drunkards it is constantly present, and here it often assumes the character of delirium tremens. Convulsions are common in children, especially at the period of invasion. They are rare in the adult. These nervous symptoms are sometimes so prominent, especially in drunkards and young children, as to mask the nature of the disease.

Digestive organs.—The symptoms of pneumonia referable to the digestive system are similar to those met with in other severe febrile diseases. There is thirst, with loss of appetite. The tongue is more or less thickly coated with a white fur, and tends in severe cases to become dry and brown. Herpes usually makes its appearance about the lips, and sometimes on other parts, from the second to the fourth day of the disease. Vomiting, which is a common symptom of invasion, is an occasional complication, as is also diarrhoea; constipation, however, is the rule. Sometimes there is slight jaundice.

Urine.—The quantity of urine is considerably diminished, and its specific gravity increased, so that abundant urates are deposited. The excretion of urea is greatly increased, and it may amount to as much as seventy-five grammes in the twenty-four hours. The uric acid is likewise augmented. The inorganic salts, especially the chlorides, are much diminished, and during the height of the disease the chloride of sodium may entirely disappear. This diminution is probably due to abundant exudation of salts into the pneumonic lung. Slight temporary albuminuria is perhaps more common in pneumonia than in almost any other acute febrile affection. The amount is usually in direct proportion to the severity of the disease. Bile-pigment is occasionally met with.

Blood.—The fibrin-elements are increased, and in most cases there is considerable leucocytosis. The leucocytes may be 15,000 to 50,000 per c.mm. This leucocytosis disappears with crisis.

PHYSICAL SIGNS.—The earliest physical signs of pneumonia are usually discoverable within forty-eight hours of the invasion of the disease. They often appear within twelve or twenty-four hours; but occasionally, when the local lesion is deeply seated, nothing abnormal is to be detected until the third or fourth day. It will be well to describe them in the order in which they commonly make their appearance. The time occupied in their evolution will vary according to the rapidity with which the several stages of the pneumonic process succeed one another; and it must be remembered that only some of them are usually observed in practice. In a large number of cases, bronchial breathing and some impaired resonance (the two signs most frequently met with) are the only abnormal signs discoverable.

The earliest abnormal physical signs are due to the pain caused by the movement of the affected side; to the hyperæmia of the pulmonary capillaries; and to the commencing exudation into the air-vesicles. The respiratory movements of the side are more or less impaired. This is partly owing to pain, and partly to diminished elasticity of

the lung-tissue. The breath-sounds are usually somewhat weak and harsh, but not distant; although, as stated by Stokes, they are occasionally in the earliest stage harsher and louder than natural. Percussion during this stage is usually not markedly altered. The resonance, however, in the upper portions of the chest is sometimes appreciably tympanitic in quality. The vocal fremitus is increased. The most important sign, however, of the congestive stage is *fine crepitation*. This râle consists of a number of fine, dry, crackling sounds, following one another in rapid succession, which were aptly compared by the late C. J. B. Williams to the sounds produced by rubbing the hair between the fingers close to the ear. It occurs during the later period of this stage, when the process of exudation from the pulmonary capillaries is commencing to take place. This râle is inspiratory; it is intensified by deep inspiration and also by cough, and it is sometimes necessary to make the patient cough in order to elicit it. A similar râle is often heard with deep inspiration in portions of the lung which have been imperfectly expanded. Such imperfect expansion is common in the posterior parts of the lungs of patients who have been confined to bed from acute or chronic disease, and in whom, owing to muscular weakness, inspiration is incompletely performed. The râle produced under these circumstances is distinguished from pneumonic crepitation inasmuch as it completely disappears after a few deep inspirations; whereas the pneumonic râle, when once established, persists until the consolidation of the lung is tolerably complete.

The physical signs of the stage of hepatisation are due to the more or less complete consolidation of the lung. The fine crepitation which characterised the later periods of the preceding stage may continue during the process of consolidation, but ceases as the filling of the air-vesicles becomes complete; although it may often still be heard at the confines of the more firmly consolidated lung. It frequently happens, however, when the consolidation is rapidly induced, that no crepitation is heard throughout the course of the disease until the period of resolution. The situation of the cardiac impulse is not altered. Vocal fremitus, when obtainable, is usually increased. To this general rule, however, there are exceptions, and it not infrequently happens that it is unaltered, and it may even be completely absent. This diminution in the vocal fremitus is sometimes due to the blocking of the smaller bronchi with the inflammatory exudation, but more frequently it appears to result from an accumulation of mucus. In the latter case it may sometimes be restored by cough. Coincidentally with the increase of vocal fremitus there is usually increased vocal resonance, and sometimes whispering pectoriloquy. The percussion-sound now is much more deficient in tone, and there is also an increase in the sense of resistance, but neither the dulness nor the resistance is so marked as in pleural effusion. In basic disease, percussion under the clavicle often yields a distinctly amphoric note, while the lower portions of the chest may be almost absolutely dull. The auscultatory sign of this stage is bronchial breathing. This is usually remarkably superficial, high-pitched, and metallic in quality (tubular breathing). Sometimes, however, it is less metallic and softer (diffused blowing—Walshe). These respiratory phenomena, like the vocal resonance and fremitus,

may be absent over larger or smaller areas of the consolidated lung, owing to the obstruction of the bronchi by catarrhal secretion. The slight pleurisy which constantly accompanies the pneumonia is rarely susceptible of physical demonstration during this stage. This is probably owing to the immobility of the solid lung. During the period of resolution, as expansive power returns, friction-sounds are occasionally audible.

Resolution usually commences in those portions of the lung which were the last to become consolidated. The most important and the earliest of the signs of resolution is the return of crepitation. The crepitation, however, differs from that met with in the earlier stages of the disease. It is larger, coarser, and more liquid in character—*redux crepitation*; and its liquid character gradually increases until it may become distinctly bubbling. When resolution is very rapid, *redux crepitation* may be absent. The bronchial breathing now loses its metallic ringing quality; the percussion-dulness gradually disappears; and the respiration regains to a great extent its normal characters.

The commencement of resolution and of the improvement in the physical signs occasionally takes place coincidently with the establishment of crisis; but more commonly it is not observed until from twenty-four to forty-eight hours after the temperature has reached the normal standard. The time occupied in the completion of resolution varies. Sometimes all physical signs almost completely disappear in twenty-four hours. Usually, however, resolution is less rapid, and marked signs of consolidation remain for periods varying from two or three days to two weeks. A slight amount of dulness and some weakness of respiration often persist at the posterior and inferior portions of the lung for even still longer periods. This is especially the case if the pneumonia is complicated with pleurisy. When marked signs of consolidation exist after the third week, there always exists more or less probability that the pneumonic process may become chronic.

COURSE AND TERMINATIONS.—The symptoms which have been described continue with often increasing severity up to about the end of the first week, sometimes longer, when there is usually an abrupt subsidence of the pyrexia—*crisis*, and the disease generally terminates in *recovery*. In other cases *death* occurs either before or after the crisis. The disease may also terminate in *gangrene* of the lung; in *pulmonary abscess*; or in *chronic pneumonia*. These several modes of termination must be considered separately.

Complete recovery.—This is the most common termination of pneumonia in young and healthy adults, and the improvement usually begins with the *crisis*. The time at which this crisis takes place, as indicated by the sudden fall of temperature, varies from the third to the twelfth day. In the majority of cases it is on the fifth, sixth, or seventh day; occasionally as early as the third day; and sometimes it is prolonged into the middle of the second week.

Crisis is the most characteristic feature of pneumonia. Its supervention is sometimes indicated by a change in the pulse, which becomes softer, and somewhat irregular in force and rhythm. The most marked phenomenon attending it is the abrupt fall of the body-temperature. This fall usually begins late in the afternoon or evening; and the

temperature very often reaches the normal standard in from sixteen to twenty-four hours. Sometimes the fall is even more rapid—in from five to ten hours. The morning remission and evening exacerbation occur during the period of defervescence. The temperature not infrequently falls 1° or 2° F. below normal, and may remain so for two or three days. Occasionally a marked increase in the pyrexia is observed immediately before the commencement of defervescence.

With the fall of temperature all the symptoms rapidly improve. The skin becomes moist and often perspires profusely. The amount of urine increases. The respiration falls in frequency; and, to a less extent, the pulse. The cough becomes looser, and the expectoration more copious. The sputa gradually lose their tenacity and rusty colour, and become more bronchitic in character; they are now usually mingled with more or less black pigment. An improvement in the physical signs is sometimes observed at the same time; more commonly, however, this does not take place till one or two days later. The patient often falls into a deep sleep, and on waking, with the exception of great weakness, declares himself pretty well, and begins to ask for food. In some cases, however, the amount of prostration following the crisis is so great that the return to health is more gradual; and a condition of collapse may ensue, which often terminates in death. There is a liability to diarrhoea at this period which it is all-important to bear in mind.

In the majority of cases pneumonia terminates abruptly in the manner above described. Sometimes, however, the temperature falls more gradually—by *lysis*; and recovery is more protracted. Defervescence may not be complete till the end of the second week. The critical fall of temperature is occasionally interrupted by more or less marked exacerbations, due either to the implication of fresh portions of the lung, or to the supervention of one of the complications to be hereafter alluded to.

In some cases, after the occurrence of crisis, there is a slight return of fever of a hectic type. This irregular fever may last three or four days. Its persistence should always suggest a pleural complication (see **COMPLICATIONS**, p. 924). Lastly, a distinct relapse may occur; but this is very rare, and the relapse is in most cases shorter and less severe than the primary attack.

Death.—When pneumonia terminates fatally, it usually does so towards the end of the first, or quite at the beginning of the second week, and often *after crisis*. The great fatality of the disease is mainly due to the attendant toxæmia; hence cardiac failure is a much more important danger than apnoea. The danger from direct interference with the respiratory function increases with the extent of lung involved, and it is consequently greater in double than in unilateral disease. Failure of cardiac power is undoubtedly the most important element of danger. There are several conditions in the disease which tend to interfere with the circulation. First and foremost of these is the toxæmia. This, which at present we have no means of combating, is often intense; and the consequent interference with metabolism and damage to tissue is increased by the accompanying high body-temperature. Then, again, the obstruction to the circulation in the lung by throwing increased work on the right heart augments the difficulty.

Such being the modes by which pneumonia tends to destroy life, it will be readily understood that the earliest and most important signs of unfavourable augury are on the side of the circulation. The pulse becomes more frequent, small, irregular, and often dicrotic. The frequency of the respiration, the dyspnoea, and the cyanosis increase. The cough becomes feeble and ineffectual. Owing to the engorgement and failure of power of the right ventricle, general pulmonary oedema usually supervenes, so that moist *râles* are audible at both bases. The extremities become cold, and there is often profuse perspiration. The mind wanders, and a condition of partial coma supervenes before the close. In some cases a rapid rise of temperature takes place before the fatal termination, while in others there may be a considerable fall in the thermometer. In those exceptional cases in which pneumonia tends to become more or less chronic, death may occur during the third or fourth week. Death may also result from the complications.

Gangrene.—This is quite rare. It is most common in chronic drunkards, and in those of debilitated constitution. Its occurrence appears to be due partly to blocking of vessels, and partly to the local influence of some special form of septic infection. It is usually limited to a small area of the pneumonic lung; and is either diffuse or circumscribed. It commonly supervenes late in the disease; and the most reliable signs of its occurrence are marked foetor of the breath and the expectoration, and great prostration. Portions of lung-tissue are occasionally found in the sputa. Gangrene is almost invariably, but not necessarily, fatal. *See* LUNGS, Gangrene of.

Abscess.—This is also very rare. *See* LUNGS, Abscess of.

Chronic Pneumonia.—Acute pneumonia in very exceptional cases becomes chronic, and leads to induration of the lung. *See* CHRONIC PNEUMONIA.

VARIETIES.—The clinical phenomena of pneumonia are occasionally somewhat different from those which have been described. Variations are met with resulting from differences in the individual, and in the ætiological factors concerned in the disease.

In the aged pneumonia sometimes runs a latent course. Invasion may be unattended by rigor or other prominent symptoms. The elevation of temperature is much less than in adults. Cough, expectoration, pain, and dyspnoea may be completely wanting. If cough be present, the sputa often do not present the rusty tinge, but are simply transparent or muco-purulent. Slight pyrexia, with some increased frequency of breathing and prostration, may be the only symptoms present. This latent course of pneumonia it is important to bear in mind, as it indicates the necessity of making a most careful physical examination of the chest in all febrile illnesses of the aged.

In young children the disease more frequently affects the upper lobe (apical pneumonia); and nervous symptoms are often obtrusive, simulating meningitis. When pneumonia occurs in those who are debilitated by previous disease, by the abuse of alcohol, or by other causes, the phenomena of invasion are usually not pronounced, and symptoms of intense prostration may occur early. The initial rigor and pain in the side are often observed; but cough is slight, and the expectoration, instead of the rusty-brown tint, may present a dirty-brown or

prune-juice appearance. Various symptoms of an asthenic type soon become prominent; the most important of which are low delirium, alternating with stupor; tremors; and paralysis of the sphincters. The tongue is brown and dry; sordes form on the teeth; the pulse is exceedingly rapid and feeble; and there are often slight jaundice and albuminuria. Death usually supervenes some time during the second week of the disease. After death the lung is usually found to be less firmly consolidated and less granular than in sthenic forms of pneumonia; and the stage of grey hepatisation in some cases is exceedingly advanced.

Secondary Pneumonia.—Pneumonia occurring in the course of other diseases usually differs somewhat from the primary disease. The changes in the lung may be precisely similar, but in many cases the consolidation is less dense and fibrinous, and epithelial changes constitute a more or less prominent feature. Secondary pneumonia occurs chiefly in the acute specific fevers—typhoid and influenza; in acute rheumatism, Bright's disease, and in many other acute and chronic diseases. It is a frequent terminal event in many diseases.

The symptoms are usually less pronounced than in primary pneumonia, and they are often masked by those of the pre-existing disease. Latency may be so marked that the pneumonia is overlooked, and its recognition in most cases depends upon careful examination of the lungs.

Hypostatic Pneumonia.—Allusion may be made here to the consolidations so often met with at the bases and more dependent portions of the lungs in the course of both acute and chronic disease, which are due mainly to mechanical causes, and have been termed *hypostatic pneumonia*. They consist, in the main, of collapse, hyperæmia, and oedema of the lung-tissue, resulting from weak inspiratory power, feeble circulation, and gravitation. The consolidation thus mechanically induced is increased by exudation from the vessels due to the damage to their walls caused by the blood-stasis. The diagnosis between this consolidation and a secondary pneumonia is sometimes impossible.

COMPLICATIONS.—The complications of pneumonia are, for the most part, due to the invasion of other tissues than the lung by the *Diplococcus pneumoniae*.

Pleurisy.—Pleurisy of slight intensity, and unaccompanied by effusion, is, as already stated, almost invariably met with in pneumonia over those portions of the lung which are consolidated. This is natural to the disease, and cannot be regarded as a complication. Pleurisy of greater intensity, and attended by a sero-fibrinous effusion, occurs in from 5 to 15 per cent. of the cases (*pleuro-pneumonia*). Signs of effusion are not usually discoverable before the third or fourth day of the disease. The amount of liquid varies with the extent of the lung-consolidation. When this is considerable, involving nearly the whole lung, there is but little room for effusion. The supervention of such pleurisy does not commonly materially modify the course of the disease. It may, however, protract the period of defervescence. Its influence in interfering with the disappearance of the physical signs has been already alluded to.

Empyema.—A suppurative pleurisy, occurring usually at the termination of the disease, often after crisis, is not infrequent; and a persistence or recurrence of the pyrexia should always suggest it.

Bronchitis.—This is also a common complication, especially in the aged and in young children. Many cases, however, which have been described as pneumonia, associated with bronchitis, have doubtless been cases of broncho-pneumonia. The bronchitis almost invariably affects both lungs. Its supervenition is attended by an increase in the cough and in the amount of expectoration. When it involves the smaller tubes it constitutes a serious complication.

Pericarditis.—This is not uncommon. It may result from the direct extension of the inflammatory process from the pleura. When extensive, it is a grave complication, and greatly increases the mortality.

Endocarditis.—This occasionally occurs. Osler found it in 10 per cent. of his cases.

Meningitis.—This is a rare complication. It appears in most cases to be caused by the pneumococcus.

Jaundice.—A slight yellowish tinge of the conjunctiva, and even of the skin, is not infrequent in pneumonia, and has no clinical significance. Much more extensive jaundice is also occasionally met with, which appears usually to be due to duodenal catarrh, and is attended by gastric symptoms; a non-obstructive jaundice sometimes occurs.

Relapse in pneumonia is exceedingly rare. Recurrence after an interval of months or years is more common than in any other acute disease except erysipelas.

DIAGNOSIS.—The diagnosis of pneumonia, usually easy, is sometimes attended with difficulty. Although it cannot be made with absolute certainty prior to the appearance of physical signs of pulmonary consolidation, in the majority of cases a very confident opinion can be formed as to the nature of the illness in the earliest stages, before such signs are discoverable. The phenomena which are of the most diagnostic value at this early stage of the disease are the pyrexia, the increased frequency of respiration, the altered pulse-respiration ratio, the pain in the side, and the cough. The sudden and rapid rise of the body-temperature, which usually reaches its maximum in forty-eight hours, is very characteristic; such a rapid attainment and maintenance of a high temperature being more common in pneumonia than in any other disease. When physical signs of pulmonary consolidation are discoverable, which they usually are within forty-eight hours, the diagnosis becomes certain.

Difficulties in diagnosis may arise in those cases in which the local process in its earlier stages is deeply seated. Here characteristic physical signs may be wanting for four or five days, during which time some doubt may exist as to the nature of the disease. In the aged the disease so often runs a latent course that its existence may be easily overlooked unless a careful examination be made of the chest. Then, again, in the intemperate its nature may be masked by delirium and other nervous symptoms.

In children, apical pneumonia with cerebral symptoms may simulate meningitis; and a pleuritic effusion, owing to the frequent prominence of bronchial breathing, may be mistaken for pneumonic consolidation.

The diagnosis of *secondary* pneumonia occasionally presents some difficulty. Owing to the frequent absence of marked symptoms the disease may be readily overlooked unless careful examination is made of the lungs.

The diagnosis of pneumonia from other diseases

of the lungs is rarely difficult. The disease with which it is most liable to be confounded is pleurisy; and when a pleural effusion complicates pneumonia, some care may be required to avoid error. That somewhat rare variety of acute pulmonary tuberculosis, in which a large area of lung becomes rapidly consolidated, may be mistaken for pneumonia. Here, however, although the whole lung may be involved, the disease usually commences in the upper lobes, so that the abnormal physical signs are more marked at the apex. The onset of the disease also is commonly much less sudden, and its course is more protracted. Examination of the sputum for tubercle-bacilli may, however, be necessary to establish diagnosis. It may be stated generally that, in all acute consolidations of the lung, a protracted course of the pyrexia, and the occurrence of marked exacerbations and of remissions of the fever at irregular intervals, afford grounds for the supposition that the consolidation is tubercular.

The diagnosis of pneumonia from broncho-pneumonia and collapse of the lung will be considered when treating of broncho-pneumonia. See Broncho-Pneumonia, p. 930.

PROGNOSIS.—Pneumonia is one of the most fatal of the acute diseases, and in few is it more difficult to make a reliable forecast. The danger is due mainly to the attendant toxæmia, much less to the extent of lung affected.

Of all the circumstances which influence prognosis, that which is perhaps the most important is the state of the general health. In those whose vitality has been damaged by previous disease, by the abuse of alcohol, by privation, or by mode of life, pneumonia is exceedingly dangerous. The prognosis is, however, especially grave in those injured by the long-continued abuse of alcohol. Pneumonia occurring in those who are the subjects of pre-existing disease—*secondary* pneumonia—is more dangerous than primary; and that occurring in chronic Bright's disease is especially fatal.

Of equal importance is the age. In healthy children the mortality from pneumonia is comparatively small. The fatality formerly ascribed to the disease at this period of life was probably owing in great measure to the inclusion of cases of broncho-pneumonia in the statistics; the latter disease being exceedingly fatal. Healthy young adults rarely die; after the age of thirty the mortality increases considerably; and in the old pneumonia is an exceedingly fatal disease.

Pneumonia is more fatal in females than in males, the mortality being in the proportion of three to two. Pregnancy renders the disease more dangerous.

The danger of pneumonia increases somewhat with the extent of lung involved. It is, however, the implication of both lungs which renders the prognosis especially grave. With regard to the situation of the consolidation, it may, perhaps, be stated generally that pneumonia commencing in the upper lobes is rather more serious than basic disease. This difference does not obtain in the case of children. The gradual extension of the consolidation late in the disease, and the spreading of the inflammatory process from one centre to another, constitute elements of gravity. When resolution is much protracted, the fact that the disease in very exceptional cases terminates in an indurative consolidation of the lung is not to be forgotten.

The mortality of pneumonia is greatly increased by the existence of complications. The prognostic importance of these has already been considered.

Of symptoms, the most important are those indicating failure of the circulation. A pulse which in the adult is persistently over 120, and in the child over 140, is of grave significance. Marked irregularity in force and rhythm is also unfavourable, especially in the young. Weakness of the first sound of the heart is another indication of failure. Variations in the respiration are of less import. Extreme quickness of breathing, marked dyspnoea, and cyanosis are not uncommon in cases which terminate in recovery; at the same time, such symptoms must have more or less unfavourable significance. Sputa of a dark prune-juice colour are of somewhat evil augury; as is also an abundant liquid puriform expectoration. The indications derived from the pyrexia are of less prognostic value in pneumonia than in most acute diseases. A temperature of 105° or 106° F. does not in itself indicate danger. Greater elevation is grave. In many fatal cases the temperature never attains 102°. The significance of a protracted defervescence has already been alluded to. On the side of the nervous system, it is to be remembered that slight delirium is not uncommon; but when it is marked, and especially when it occurs late in the disease and prevents sleep, it is most grave. It is of greater significance in adults than in children. Tremors and a tendency to coma are also unfavourable. A dry, brown tongue is unfavourable, especially when associated with only a moderate degree of pyrexia. Gastric catarrh and diarrhoea add to the danger; and the liability to diarrhoea at the period of crisis, and the frequency with which it then endangers life, should be ever remembered. Tympanites is very unfavourable. A slight amount of jaundice is not of ill omen, but the appearance of much albumen in the urine early in the disease is undoubtedly of bad import.

TREATMENT.—In considering the treatment of pneumonia, it is of the utmost importance to bear in mind the true nature of the disease. All rational and successful therapeutics must be based upon the recognition of the fact that it is not simply a local affection of the lung which we wish to influence. The 'heroic' methods of treatment by venesection, tartar emetic, &c., so much in vogue in the past, had for their object the controlling or cutting short of a simple pulmonary inflammation. Such methods have now been abandoned, and with our increasing knowledge of the bacteriology of the disease we are looking with confidence to the discovery of specific remedies which may secure immunity or successfully combat the local inflammation and the attendant toxæmia. An anti-pneumococcus serum is already being used with results that justify further trial. Washbourn recommends the injection of 20 c.c. of this serum twice daily. See SERUM-THERAPEUTICS.

Failing specific remedies our object must be, as in other specific fevers, to conduct the disease to a favourable termination. If we cannot arrest its progress, we can often do very much both to maintain the strength of the patient, and to modify those elements in the disease which tend to destroy life.

The modes by which pneumonia tends to destroy life have been already considered—that damage to the tissues generally which obtains in all acute specific diseases; and direct interference with the

respiratory function due to the lung-consolidation. Of these, the former is the more important, and impaired cardiac power is its most serious consequence. The natural course of the disease is also to be remembered. In the strong and robust, pneumonia usually terminates in recovery. It is in those who are debilitated by age, privation, mode of life, abuse of alcohol, or pre-existing disease, that such great mortality attends it. It is a question of the intensity of the disease on the one hand, and of the resisting power of the individual on the other. Such considerations as these indicate the importance not only of doing all that is possible to husband and support the strength of the patient, but also of not interfering too actively with the disease, unless circumstances arise which, if uninfluenced by treatment, would tend to prove rapidly fatal.

Such being the general principles which should guide us in the treatment of pneumonia, the manner in which they are to be best carried out may now be indicated.

Everything should be done to husband the strength of the patient, and the services of efficient nurses are all-important towards the attainment of this object. Perfect rest must be enjoined, and all unnecessary speaking be forbidden. The patient should be kept in bed, be lightly covered, and be sponged freely three or four times daily with cold or tepid water. The room should be large and airy; and the temperature about 60° to 62° F. It should be well ventilated: a plentiful supply of fresh air is most important, and, although due care should be exercised in the ventilation, there is not the same necessity to keep the patient scrupulously protected from draughts as in the treatment of acute bronchitis.

The diet should be carefully regulated, nutritious and easily digestible, consisting of milk, milk with egg, beef-tea, mutton- or chicken-broth, meat-essences, and similar articles, given in varying quantities and at varying intervals, according to the condition of the patient. Water should be allowed liberally to relieve thirst and promote perspiration. With the object of stimulating the secretion of saliva and promoting the appetite, it is well to keep the mouth cleansed with glycerine and lemon-juice. Some acid and bitter drink, as one made with hydrochloric acid and orange-peel, may also be given.

In the earliest stage of the disease it is usually wise to give a small dose of calomel, or of blue pill and colocynth, to be followed in five or six hours by a saline aperient. The exhibition of purgatives, however, requires great care, as they occasionally set up a catarrhal condition of the intestine, and consequent diarrhoea, which may prove an element of danger, especially near the period of crisis. It is important, therefore, except on the first day of the illness, to procure all necessary evacuation of the bowels, by a small dose either of castor oil, or of colocynth and hyoscyamus, or by simple enemata. With the object of promoting elimination by the skin and kidneys, some citrate or acetate of ammonia with citrate of potassium should be given three or four times daily.

On the first day of the disease when the symptoms of invasion are accompanied by much pain, anxiety, and restlessness, morphine used with care and judgment is often of the utmost value. Morphine ($\frac{1}{4}$ grain with, if necessary, strychnine $\frac{1}{30}$ grain) may be given hypodermically.

There are two circumstances which often tend greatly to interfere with the satisfactory progress of pneumonia—the pain in the side, and sleeplessness. Pain in the side is often troublesome during the first two days. It may usually be relieved by the application of large hot linseed poultices, or hot fomentations, or by the use of an ice-bag. If these means do not succeed, a small blister (2 by 2 inches), or three or four leeches, may be applied to the seat of the pain, and the hot applications then renewed. In exceptional cases small doses of morphine hypodermically may be necessary to relieve this symptom. Cough is only harmful by increasing the pain, and rarely calls for interference. Sleeplessness is always a more or less troublesome symptom, and yet to secure a sufficient amount of sleep is obviously of primary importance. A careful nurse, keeping the room quiet and darkened, and attending to the comfort of the patient; the relief of pain by the measures above indicated; and sponging with tepid water the last thing at night, are often sufficient to secure some interrupted sleep. Should it be necessary, some hypnotic must be given: bromide of potassium and hyoscyamus, sulphonal, chloralamide, Tinct. Chloroformi et Morphinæ Co., are all useful, and, if they fail, morphine guarded by atropine and strychnine may be administered *sub cutem*. Chloral hydrate is usually contra-indicated, on account of its depressing effect upon the circulation.

A large number of cases of pneumonia terminate in health without the necessity of any further interference on the part of the physician than has been described. Frequently, on the other hand, circumstances arise indicative of danger, which require to be met by more active treatment.

Pyrexia is sometimes excessive, but in attempting to interfere with it, it must be remembered that it is natural to the disease, and only when excessive does it become harmful. A temperature of 104° is, in itself, of no unfavourable import. Frequent sponging with tepid or cold water, by cooling the patient, and promoting rest and sleep, is always desirable. The frequent, or more or less continuous, application of an ice-bag to the affected side, as recommended by Lees, is another means of refrigeration often useful. According to this physician it controls not only the temperature but also the lung-inflammation. It usually relieves pain and increases comfort. If the patient likes it, it is wise to make use of it. In children, an ice-poultice is preferable to the bag.

Hyperpyrexia is occasionally met with. A temperature of 105° calls for a more assiduous employment of the refrigerating treatment already alluded to; and in those exceptional cases in which the temperature reaches 106° or 107° a cold bath at 75° or 80° may be necessary. Antipyretic drugs are dangerous in pneumonia; but quinine in 5-grain doses, given in cachets, is sometimes of undoubted use, although it may not materially influence the temperature-chart. High temperature in pneumonia always calls for the free exhibition of alcohol.

Symptoms indicative of danger are especially likely to arise in those whose vitality is impaired by age, habits, or other causes, and, in such, special care and watchfulness should be exercised in anticipation of any unfavourable symptom. The chief source of danger, as already stated, is failure of cardiac power, and consequently all symptoms of such failure must be carefully watched for.

Any sign of cardiac failure will in the first place suggest the advisability of administering alcohol. It may be stated generally that a pulse of over 120 calls for the employment of stimulants. Brandy appears in most cases to answer best. The amount administered must depend upon its effects; and although in most cases from four to eight ounces in the twenty-four hours will be sufficient, if the asthenia persist it may be given in larger quantities. The disease is one of comparatively short duration, and there is perhaps no other acute illness in which the free exhibition of alcohol, when indicated, is more likely to be advantageous.

The free inhalation of oxygen is also desirable under these circumstances. It not only relieves the embarrassed respiration, but tends to prevent heart-failure. Oxygen should always be available in a severe case, and it should be used early. It sometimes also promotes sleep, which in these cases is obviously of primary importance.

Of drugs, the most useful is strychnine. This is best given in full doses at rather long intervals—liq. strych. hydrochlor. m iij to v , by the mouth or hypodermically every six or eight hours. Ammonium carbonate in full doses (gr. v to gr. x) is also useful. Digitalis, although indicated by a rapid and failing heart, is of doubtful value.

The special danger at, and immediately after, crisis calls for the assiduous employment of the above treatment at this period, and indicates the importance of not relinquishing it too soon after the critical fall of temperature.

Interference with respiration from extensive consolidation of the lung is too often an important source of danger; and the consequent increased work thrown on the right ventricle tends to increase the heart-weakness. Symptoms of cardiac are thus usually associated with those of respiratory failure; and in dealing with the latter, we must depend mainly upon the means already described as best calculated to help the heart. The free use of strychnine and oxygen, which stimulate not only the heart but the respiratory centre, may often avert death.

Venesection is well known to relieve the dyspnoea of pneumonia. It diminishes the pulmonary congestion and unloads the distended right heart. It must, however, be borne in mind that the relief is merely temporary, and that the loss of blood must tend more or less to weaken the patient, and hence to favour that condition of asthenia which is of all things the most to be feared. Bleeding is certainly only to be thought of when in the *early* stage of the illness the dyspnoea is urgent, and when at the same time the pulse is strong and full; and it should not exceed the removal of eight or ten ounces of blood. Such cases are certainly not common.

Of the complications, delirium sometimes calls for treatment. Active delirium is not common in pneumonia, except in those who have been intemperate. When marked, it is always indicative of danger. In its management the practitioner must be guided by the general condition of the patient; but, speaking generally, it calls for the free exhibition of alcohol, and the external application of cold. Cold, applied in some of the ways already indicated, is of especial value in these cases, not only reducing the temperature, but quieting the patient and often producing refreshing sleep. The influence of an experienced nurse is all-important. Bromides are often useful, and it may be necessary

to give morphine hypodermically. Hyoscine ($\frac{1}{200}$ grain) may be used with caution (p. 707).

Of the management of other complications occurring in the course of pneumonia there is nothing special to be remarked; they must be treated on general principles. The existence of bronchial catarrh often requires small doses of ipecacuanha with carbonate of ammonium and salines. Diarrhoea and gastric symptoms are to be met by careful dieting, chalk, bismuth, and, if necessary, other astringent remedies. In pneumonia, as in other acute diseases, the administration of an opiate enema is an efficient and safe means of checking diarrhoea. Pericarditis rarely admits of any special interference. The persistence of pyrexia or its recurrence after crisis may call for exploration of the pleura, and an empyema may require treatment. See PLEURA, Diseases of.

Convalescence.—During the period which immediately succeeds the crisis, the utmost care is required to support the patient, and to prevent any serious amount of prostration, which at this time so often supervenes. Stimulants are usually required for some days after the temperature has attained the normal standard. Convalescence in most cases is quickly established. Solid diet is soon desired, and may be safely given. Tonics—such as quinine, iron, and cod-liver oil—and change of air are useful in assisting the restoration to health.

B. Broncho-Pneumonia.—**SYNON.**: Catarrhal Pneumonia; Lobular Pneumonia; Fr. *Pneumonie Lobulaire*; *Broncho-pneumonie*; Ger. *Bronchopneumonie*.

DEFINITION.—Broncho-, catarrhal, or lobular pneumonia is inflammation of the lung, due to an irritant entering and spreading by the bronchi. The irritant usually causes, in the first place, inflammation of the bronchial mucous membrane, which spreads to the bronchioles and alveoli. This frequent association of the pulmonary inflammation with bronchial catarrh explains the terms *broncho- and catarrhal*, which are applied to this form of pneumonia. The pulmonary inflammation is commonly limited to scattered groups of air-vesicles (*lobular*), but sometimes it causes large areas of consolidation (*lobar*). The inflammatory products which fill the alveoli consist principally of cells, derived from the epithelium of the alveoli, and from the bronchial mucous membrane; exudation and emigration play a much less prominent part than in ordinary pneumonia.

ETIOLOGY.—The most important group of cases of broncho-pneumonia are those associated with a more or less general bronchitis, the bronchial usually preceding the pulmonary inflammation. Whatever causes inflammation of the bronchial mucous membrane may be a cause of broncho-pneumonia, and when the inflammation involves the smallest bronchi, its extension to some of the alveoli is almost invariable.

Simple non-specific bronchitis is frequently followed by broncho-pneumonia, especially in childhood and in old age. All those conditions which favour the occurrence of such bronchitis must therefore be enumerated among the causes of this form of pneumonia. Of these conditions it will be sufficient to mention here the marked influence of cold and damp. It is, however, the specific forms of bronchitis, associated with measles, whooping-cough, diphtheria, and influenza, which are the most liable to be followed by broncho-pneumonia.

It occurs less frequently in scarlatina, small-pox, and typhoid fever.

All conditions which tend to impair the general health and strength favour the occurrence of broncho-pneumonia. The weakly and debilitated suffer most. Bad air, insufficient food, and rickets are important predisposing causes.

Broncho-pneumonia is most common during the first four years of life—the period when bronchial catarrh, measles, and whooping-cough are so frequent. It is also common in old age. In young adults it is comparatively rare. Sex has no influence.

Another group of cases of broncho-pneumonia are those due to the inhalation of more definite irritants (aspiration-pneumonia): (1) dust, particles of steel, iron, stone, coal, &c., gaining access to the air-passages may cause inflammation of the bronchi and subsequently of the alveoli; (2) irritant gases, e.g. ether-pneumonia; (3) micro-organisms. Under this head the bacillus of tuberculosis is the most important—a tubercular broncho-pneumonia being the principal lesion in chronic pulmonary tuberculosis. Septic blood and pus may be sucked into the air-passages in diseases of, or during operations on, the nose, mouth, pharynx, and trachea. The contents of an empyema or of a bronchiectatic cavity may be aspirated; as may also blood after a pulmonary hemorrhage. Lastly, septic organisms conveyed with particles of food or saliva may enter the air-passages when the glottis is insensitive or paralysed. The pneumonia in these cases is usually more localised than in the preceding group, and there is less attendant general bronchitis. When the irritant is septic, suppuration and even gangrene are liable to occur.

BACTERIOLOGY.—To enumerate the micro-organisms that at various times have been regarded as specific for this condition would be to catalogue the pathogenic bacteria, including pathogenic moulds. In point of fact no one organism has been found associated with broncho-pneumonia with sufficient constancy to warrant belief in its specificity, but, on the contrary, it would appear that the invasion of the lung-tissue by any one or more of the pathogenic bacteria may, under certain conditions as yet but little understood, result in the production of the disease.

The organisms most frequently associated with this condition are certainly the pyogenic streptococci and staphylococci, and the pneumobacillus.

ANATOMICAL CHARACTERS.—The appearances presented by the lungs after death from broncho-pneumonia vary. The bronchi always exhibit signs of more or less bronchial catarrh. This may involve the whole of the bronchial mucous membrane, but it is usually most marked in the smaller tubes. These are found containing a thick, tenacious, and often puriform secretion, which is occasionally here and there drier or inspissated. The mucous membrane of these tubes is more or less softened, swollen, red, and thickened, and often presents irregular superficial erosions. Owing to this inflammatory swelling the tubes stand out prominently on section of the lung. Cylindrical dilatations of the tubes are also frequently met with.

The lung-tissue itself exhibits, associated in various degrees, collapse, congestion, œdema, emphysema, and pneumonic consolidation. The bluish, non-crepitant, smooth, depressed portions of col-

lapse, which become darker and more friable with age, are usually most abundant in the lower lobes and margins of the lungs. The collapse sometimes involves the whole of one lobe, but more commonly it is limited to much smaller areas of the lung. When scattered and limited in its distribution there is usually more or less emphysema of the intervening portions of the lung; when collapse is very extensive in the lower lobes emphysema is most marked in the upper.

Those portions of the lung in which the pneumonic process has supervened appear most commonly as scattered patches and nodules of consolidation, varying in size from a small pea to a hazel-nut. These are usually more or less conical in shape, with their bases towards the pleura, which membrane is generally unaffected over them, though it may be coated with lymph. On section the patches are commonly ill-defined and pass insensibly into the surrounding tissue, which is variously altered by congestion, collapse, and emphysema. They are of a reddish-grey colour, slightly elevated, smooth, or faintly granular, and soft and friable in consistence. As they increase in size, they may become confluent, and thus are produced large tracts of consolidation. In a more advanced stage, the nodular and more diffuse consolidation becomes paler, firmer, and drier, and resembles in colour the greyish-yellow hepatisation of acute pneumonia. The cut ends of dilated bronchi, filled with pus, are occasionally seen in the centres of the pneumonic nodules. The pneumonic process often involves the collapsed portions of lung, which then become more opaque and swollen. In septic broncho-pneumonia the pneumonic patches often suppurate, and sometimes contain sloughs of lung-tissue.

Microscopical characters. — When examined microscopically, the pneumonic patches are seen to consist of an accumulation within the alveoli of a gelatinous mucoid-looking substance, leucocytes, epithelial elements, and red blood-corpuscles. The epithelial elements are swollen and granular. In some cases much of this accumulation is precisely similar to that contained in the smaller bronchi, and it is evidently the inflammatory and richly cellular bronchial secretion which has been inhaled. The relative proportion of epithelium and leucocytes varies considerably in different cases, the latter being more numerous in acute, and especially in septic cases.

The subsequent changes which take place in the lungs vary. When the disease does not end in death, resolution is the most common termination. The contents of the alveoli undergo fatty degeneration, and are removed by expectoration and absorption, the lung gradually regaining its normal characters. This process, however, is less readily effected than in the consolidation of acute pneumonia; and it sometimes occupies such a lengthened period, that some thickening of the bronchial and alveolar walls and dilatation of the smaller bronchi remain. In still more chronic cases the fibroid thickening is much more marked, and a considerable amount of irregularly distributed, pigmented induration and bronchial dilatation may be produced. Caseation occurs only in tubercular cases.

SYMPTOMS.—The symptoms of broncho-pneumonia vary according to the rapidity with which the disease is developed, the extent of lung in-

volved, and the nature of the disease in the course of which it supervenes.

The more severe and most common forms of the disease are met with in early childhood in association with simple bronchitis, and the specific bronchitis of measles and whooping-cough. In these cases the symptoms and dangers are, in the main, those of the capillary bronchitis which precedes the pneumonic process, and it is sometimes impossible to distinguish between them. Frequent cough, accelerated respiration, slight action of the nares, and other symptoms of severe bronchitis, with a little pyrexia, precede for a varying length of time the symptoms due to the implication of the lung-tissue. The earliest symptoms of the pneumonic process are by no means well-defined, and consequently the time at which the lung becomes involved cannot be fixed with certainty. Rigors, convulsion, and vomiting are but rarely observed. Usually an increase in the acceleration of the respiration or in the dyspnoea, and a marked rise of temperature, are the first signs of the pulmonary implication. The breathing becomes more rapid, and commonly causes much distress, the child tossing about and being exceedingly restless. The dyspnoea is more marked at some times than at others, and is occasionally more or less distinctly paroxysmal. The respiration is superficial, inspiration being short, and the expansion of the thorax imperfect. There is marked action of the accessory respiratory muscles, and the upper portions of the thorax are raised, while the lower and the soft parts are retracted during the inspiratory act. The action of the nares is very pronounced.

An increase in any pyrexia which attended the pre-existing bronchitis is, with few exceptions, observed as the lungs become involved, and such increase is to be regarded as one of the most valuable indications of the existence of a pneumonic process. The maximum temperature of acute simple bronchitis, even in the child, is seldom higher than 102° F., whereas that of the secondary pneumonic process is often 104° or 105°. This increase usually occurs more or less gradually; there is rarely the sudden rise of temperature met with in acute pneumonia. Unlike the temperature of this disease also, the fever of broncho-pneumonia runs no definite course. It varies with the extent of the lung-implication, and with the rapidity with which this implication is effected. There is no regular diurnal variation; the remissions and exacerbations are often considerable; and they occur at irregular times, the temperature being sometimes higher in the morning than at night. The cough, which before the implication of the lung was paroxysmal in character, gradually becomes less and less so, and it now often causes much pain to the patient. The sputa, when present, are bronchitic in character, usually tenacious, and occasionally streaked with blood; as, however, expectoration rarely occurs in the child, they are not often seen. The pulse is much increased in frequency, in children under five years often being 150. It may in the earliest stage of the disease be moderately full and strong, but it quickly becomes soft, small and feeble. In addition to the above, there are often symptoms referable to the digestive organs. Of these diarrhoea is the most important. This is quite frequent, especially when the disease follows measles. It is very readily induced by medicines

and by improper feeding ; and as it greatly weakens the patient, it is important that this liability to diarrhoea should be kept in mind. Vomiting, as already stated, is very rare as an initial symptom ; it is, however, common in the course of the disease, especially as a result of cough, the bronchial secretion, together with the contents of the stomach, often being expelled.

As the bronchitis and the implication of the lungs increase, the breathing becomes still more rapid and superficial ; the dyspnoea is more marked ; the expression is anxious ; the face is pale ; and symptoms of carbonic-acid poisoning become evident. Strength now fails ; the face and lips become cyanotic ; and the extreme restlessness gives place to apathy and a semi-comatose condition, which is interrupted from time to time by ineffectual efforts to cough. With the rapid failure of strength and increasing cyanosis, cough almost ceases ; the pulse becomes exceedingly feeble ; and the child, often extremely emaciated, may die exhausted, and in a condition of more or less profound coma. Sometimes death occurs suddenly during a paroxysm of cough, or with convulsions.

In those cases in which capillary bronchitis constitutes a less prominent feature of the illness, the symptoms are usually less severe—the pyrexia is less marked and its onset more gradual ; the pneumonic process often supervenes later ; and the course of the disease is more protracted. A slight and markedly irregular pyrexia, increasing difficulty of breathing, with loss of strength and flesh, may continue for months, and the child ultimately die, or recover with more or less damaged lungs. In these more chronic forms of the disease, which are perhaps most common after whooping-cough, the pneumonic process is often less disseminated, and involves larger areas of the lung—sometimes a whole lobe.

When broncho-pneumonia occurs in adults and in the aged, the symptoms are for the most part much less pronounced than in the child. In strong adults the disease is perhaps most common after diphtheria, and here the pulmonary symptoms may be well-marked ; but in the debilitated, and especially in the old, the course of the disease is more latent, some pyrexia, slight cough and dyspnoea, and marked debility being the principal symptoms observable.

In the aged and feeble, broncho-pneumonia is frequently associated with that form of lung-consolidation which results from weak inspiratory power, feeble circulation, and gravitation ; and which consists mainly of collapse, hyperæmia, and cedema of the lung-tissue (hypostatic pneumonia). This hypostatic consolidation may exist quite independently of bronchial catarrh ; but when such catarrh occurs in the aged and feeble, gravitation often determines the supervention of the pneumonic process, which under such circumstances is consequently not infrequently unilateral.

When the more acute varieties of broncho-pneumonia terminate fatally, they usually do so from the tenth to the fourteenth day of the disease ; and in infants death may occur much earlier. Death, unlike that from acute pneumonia, is mainly due to the interference with the respiratory function, and, to less extent, to cardiac failure. The interference with the respiration is usually much greater than in acute pneumonia, for, in addition to the diminution of the respiratory area, due to the pul-

monary consolidation and collapse, there is the much more important cause of interference—namely, the impediment to the entrance of air, caused by the swelling of the bronchial mucous membrane, and the accumulation of secretion in the smaller bronchi. Over-distension and failure of the right heart occur here as in ordinary pneumonia. In the more chronic forms of broncho-pneumonia death may not occur for some months, and then it results as much from general failure of strength as from interference with the respiratory function.

When the disease does not terminate in death, improvement in the symptoms is usually gradual. The temperature falls slowly, several days, and occasionally some weeks, being occupied in the completion of defervescence ; and this gradual decline is often interrupted by more or less marked and frequent exacerbations and remissions of the fever. The cough and dyspnoea diminish, and the appetite gradually returns ; but restoration to health is often protracted ; and the child remains for some time liable to repetition of the bronchial symptoms. In some cases, however, the disease clears up much more rapidly, and in three or four days all signs of consolidation may disappear.

Sometimes recovery is not complete, and the disease leads to some induration of the lung, and dilatation of the bronchi. Emphysema and pulmonary tuberculosis are occasional sequelæ.

Physical signs.—The physical signs of broncho-pneumonia are in the main those of capillary bronchitis. Imperfect expansion of the thorax, elevation of the upper portions, and recession of the lower, during the inspiratory act ; rhonchi and moist râles, audible over both sides ; and the absence of any marked alterations in percussion-resonance, are the principal signs observable, not only in the earlier stages, but throughout the whole course of the disease. The recession of the chest-walls is increased by the collapse. The pulmonary implication is indicated rather by the symptoms—increase in the pyrexia and in the dyspnoea—than by any marked alteration in the physical signs. The difficulty of detecting the lung-consolidation is due to its usually being limited, in the earlier stages, at all events, to small areas, which are surrounded by healthy or emphysematous lung, so that resonance on percussion is but little impaired. It is only when these small areas have coalesced into larger areas of consolidation, that any marked alterations in percussion-resonance are discoverable. The impaired resonance due to collapse is not to be distinguished from that due to pneumonic consolidation ; and, inasmuch as the collapse is so often symmetrical, involving both bases posteriorly, the difficulty of appreciating it is increased. Much more valuable aid in physical diagnosis is in most cases to be obtained from auscultation. Over those portions of the lung where consolidation has taken place, the moist bronchitic râles tend to assume a somewhat metallic quality ; they also become finer, though not so fine as true pneumonic crepitation ; and they are more superficial. The detection of these superficial, somewhat metallic fine moist râles, heard with inspiration, and not dispelled by cough, over small areas of the lungs, especially at the posterior bases, is a most valuable and often the only physical sign of the pulmonary implication. If larger areas become consolidated, there may be in addition some tubular breathing, and some impairment of resonance on percussion ; and when a

whole lobe is involved the physical signs become increasingly pronounced, and in a young child a bronchophonic cry is often marked on auscultation.

COMPLICATIONS.—Pleurisy is less common than in acute pneumonia. Slight inflammation of the pleura is, however, usually found *post mortem* over those portions of the lung which are consolidated. Pleuritic effusion is rare. Intestinal catarrh is an important and common complication. The liability to this in the child, and the mechanical congestion resulting from the obstructed pulmonary circulation, must be borne in mind in explaining its frequency.

DIAGNOSIS.—The diagnosis of broncho-pneumonia is occasionally difficult. This difficulty is mainly owing to the co-existence of capillary bronchitis. The recognition of the pulmonary implication in its earlier stages is often impossible. The increased pyrexia and frequency of respiration are the symptoms of the most diagnostic value. Owing to the small areas of lung involved, any alteration in the physical signs of the capillary bronchitis may be entirely wanting. The occurrence of extensive collapse in the earlier stages gives more marked physical signs of consolidation, and hence renders the diagnosis more easy. It is almost impossible to diagnose certainly, either by symptoms or by physical signs, between the collapse and the pneumonic consolidation. This, however, is of but little practical importance, inasmuch as the collapse is usually associated with, and often the immediate precursor of, the pneumonic process.

The diagnosis of broncho-pneumonia from acute pneumonia may occasionally be difficult in those cases of the former disease in which an extensive area of the lung has become consolidated. The history of the case, and especially the course of the pyrexia, will usually suffice to distinguish them.

The greatest difficulty is in connection with tuberculosis—to distinguish between a simple and a tubercular broncho-pneumonia is often possible only after prolonged observation. In the more protracted cases so common in children after the age of infancy, the possibility of tuberculosis must always be borne in mind. A persistence of the physical signs and of some irregular pyrexia is always suspicious. Examination of the upper lobes for any sign of softening; and of the sputum for tubercle-bacilli and elastic tissue may help the diagnosis. In young children who do not expectorate sputum may sometimes be obtained by the exhibition of an emetic.

PROGNOSIS.—The two circumstances which have an especial influence upon prognosis in broncho-pneumonia are the age of the patient, and the general health. Before puberty, the younger the patient the graver the prognosis. In children under five years, the mortality is probably about 20 per cent. The disease is especially fatal in weakly children, in those debilitated by previous illness, and when occurring in measles and diphtheria. The existence of rickets materially increases the gravity of prognosis. The danger also increases greatly with the extent of lung involved, much more so than is the case in acute pneumonia. Of the value of the several symptoms as influencing prognosis, after the description which has been given of the disease and of the modes in which it tends to cause death, it is hardly necessary to speak further. Symptoms of imperfect aëration of blood, lividity, emphysema, cessation of cough, together with a distended and failing right heart, are those most to be feared.

In protracted cases the possibility of tuberculosis must be remembered.

TREATMENT.—In the treatment of broncho-pneumonia, it is important to bear in mind—first, that the disease is generally associated with capillary bronchitis, and with more or less pulmonary collapse; second, that its occurrence is especially favoured by everything that weakens the patient; and, third, that it tends to destroy life principally by interfering with the function of respiration, which interference necessarily increases with the consequent weakening of the respiratory power. Such being the facts, it is obvious that the main object of treatment will be, first, to control the bronchitis, and endeavour so to modify it as to prevent the occurrence of collapse; and, secondly, to support as much as possible the strength of the patient.

It would be out of place in the present article to enter into a detailed description of the management of acute bronchitis (*see* BRONCHI, Diseases of). It will be sufficient to indicate the more important means of controlling the disease.

The patient should be kept in a warm room, about 65° F., the temperature never being allowed to fall below 62°. The room should be well but carefully ventilated; and protection from draughts is important—much more so than in the treatment of acute pneumonia. It is also advisable to keep the air moist by means of a steam-kettle, as the exhalation of water from the lungs is thus diminished, and the bronchial secretion consequently rendered less tenacious, and more easily removed by cough. A little carbolic acid (1 in 60) or tincture of benzoin may be added to the water. A tent to enclose the steam is sometimes advisable, but as this tends to interfere with the supply of oxygen and to weaken the patient, it must be used with caution. The diet, which must be regulated according to the age of the patient, should be nutritious and easily digestible, the importance of supporting the strength being kept in mind. When the disease follows measles, the liability to gastro-intestinal catarrh must not be forgotten. The chest should be covered with a cotton-wool jacket, and either a lightly made linseed-and-mustard poultice, or some stimulating liniment sprinkled on flannel, be applied alternately front and back.

Medicinally, small doses of ipecacuanha with salines, and carbonate of ammonium, should be administered frequently. Evidence of accumulation of the secretion from weakness or ineffectual cough calls for increased doses of the ammonia; and under these circumstances small doses of Apomorphine Hydrochlor. may often be added with advantage—for a child gr. $\frac{3}{30}$ to gr. $\frac{1}{30}$. An occasional emetic often materially relieves the patient: for a child, Vin. Ipecac. $\frac{3}{4}$, for an adult Ammon. Carb. gr. xxx. given in hot water every fifteen minutes until vomiting occurs. When cough is paroxysmal and causes much distress, belladonna in full doses is often of great benefit. The exhibition of opiates obviously requires great caution, and in children they are contra-indicated.

Signs of heart-failure call for the exhibition of brandy, and children bear stimulation well. An infant may begin with from five to ten drops every two or three hours. If with failure the right heart is distended, the jugular veins full, and much dyspnoea, the question of leeching or venesection should be considered. Leeches over the sternum are often useful: in the child three or four may be

applied. Strychnine is also most valuable, stimulating both the heart and the respiratory centre. It is best given hypodermically (Liq. Strych. Hydrochlor. m.j.) for a child every three or four hours. Oxygen is less useful than in acute pneumonia.

The external application of cold is sometimes useful in the acute forms of the disease so common in early life. This not only reduces the temperature, but appears to be especially valuable in increasing the depth and force of respiration, and thus in preventing the occurrence of collapse. Its utility has been strongly advocated by both Bartels and Ziemssen, who recommend the frequently renewed application of cold wet compresses to the chest. The use of tepid baths, with cold affusions, is a more efficient way of obtaining the same result. The child is placed in a bath of from 85° to 90° F., and, while immersed, the head, back, and chest are quickly sponged with cold water. This repeated occasionally, with due precautions, is often followed by reduction of temperature, a diminution in the frequency and an increase in the depth of the respirations, and other signs of improvement. The application of ice-bags or ice-poultices to the chest sometimes yields good results. The effects produced by the cold require in all cases to be carefully watched, and any depression caused should be met by the timely exhibition of stimulants.

In the more chronic forms of broncho-pneumonia these active methods of treatment are but rarely called for. Here attention to strength and nutrition is most important, and small doses of cod-liver oil in the later stages, even before the complete disappearance of the pyrexia, are often useful. When the disease leads to induration of the lungs and dilatation of the bronchi, the treatment resolves itself into that of chronic pneumonia.

Convalescence, it must be remembered, is usually slow, and there is a tendency to relapse. Great care is consequently requisite during this period. All causes of catarrh must be carefully guarded against; and the restoration to health assisted by nutritious diet, cod-liver oil, and preparations of iron. A change of air is especially valuable.

C. Chronic Pneumonia.—**SYN.**: Interstitial Pneumonia; Cirrhosis of the Lung; Fr. *Pneumonie Interstitielle*; Ger. *Lungencirrhose*.

DEFINITION.—Chronic pneumonia is a comparatively rare disease, characterised by a gradual increase in the connective tissue of the lung, which leads to an induration of the pulmonary texture, and to progressive obliteration of the alveolar cavities. It is commonly associated with catarrh and dilatation of the bronchi, and often with ulceration of the bronchial walls, and excavation of the indurated lung. Cough, expectoration—often abundant, but varying with the bronchial catarrh—dyspnoea, gradual impairment of nutrition, and occasional accessions of slight pyrexia, are the most prominent clinical phenomena accompanying the disease, which runs an exceedingly chronic course, often subject to long periods of quiescence, but tending to terminate fatally in from five to fifteen years.

ETIOLOGY AND PATHOLOGY.—In the large majority of cases chronic pneumonia is the result of some antecedent inflammation of the bronchi, alveoli, or pleura, although cases are sometimes met with in which no history of any such antecedent affection is discoverable. It may be stated generally that all inflammatory processes in the lungs, as in other organs, which become chronic, lead to

an increase of the connective-tissue elements, and consequently to fibroid induration of the organ. In the lungs by far the most common cause of such induration is chronic pulmonary tuberculosis. In all cases of phthisis, excepting those which are the most acute, there is more or less fibroid growth; and the extent of this growth is, for the most part, in direct proportion to the chronicity of the disease. Those forms of tuberculosis which are the most chronic, and in which the fibrosis reaches its maximum, have been termed 'fibroid phthisis.' Such cases, it must be admitted, are somewhat closely allied to some forms of chronic pneumonia. The two diseases, however, differ pathologically in this respect—that, whereas much of the pulmonary consolidation of tuberculosis tends to undergo molecular death and caseation, that of chronic pneumonia exhibits no such tendency; but any destruction and excavation of the indurated lung which may take place is due to secondary inflammation and ulceration commencing in the bronchial walls. In considering the pathology of chronic pneumonia, therefore, it is necessary to exclude the fibrosis due to chronic pulmonary tuberculosis.

The chief causes of chronic pneumonia are:

(1) *Acute pneumonia.*—The pulmonary consolidation of acute pneumonia almost invariably undergoes complete resolution. This resolution is usually effected rapidly, in from seven to fourteen days. In very exceptional cases, however, the process is so protracted as to lead to some thickening of the walls of the alveoli, and sometimes to organisation of their contents. This indurated hepatisation differs but little in its physical characters from ordinary red and grey hepatisation; it is simply somewhat firmer, more resistant, and less granular. It is probable that this small amount of induration commencing in the alveolar walls may gradually increase, so as ultimately to give rise to that extensive fibrosis of the lung which constitutes what is usually known as chronic pneumonia.

(2) *Broncho-pneumonia.*—Broncho-pneumonia is a more frequent cause of the disease than the preceding (Wilson Fox). The greater liability of this form of pneumonia to lead to pulmonary induration is to be accounted for partly by its longer duration and greater tendency to become chronic, and partly by the existence of bronchial dilatation with which it is so often associated. The accompanying collapse, so frequent in children, is also an important factor in the causation of the fibrosis. That bronchial dilatation is favourable to an indurative pneumonic process has been especially insisted upon by the late Wilson Fox. Dilatation of the bronchi is exceedingly common in the simple and specific bronchitis of childhood, and especially in that associated with whooping-cough; it is also a direct result of pulmonary fibrosis. In whatever way originating, its existence favours the persistence of the catarrhal and pneumonic processes. The removal of secretion is rendered more difficult; the retained secretion tends to increase and keep up the irritative process, both in the dilated bronchi and also in the pulmonary alveoli; and this persistence of the bronchial and pulmonary inflammation leads to fibroid thickening of the bronchial and alveolar walls. In this way more or less disseminated patches of indurative consolidation are produced, which, as the process goes on, gradually increase, so that ultimately they may involve large areas of the lung. The progressive tendency of the

process may be partly due to the fact, already stated, that pulmonary fibrosis is a cause of bronchial dilatation, so that fibrosis once established, by inducing further dilatation of the bronchi, favours the extension of the bronchial and pulmonary induration.

(3) *Pleurisy*.—Pleurisy in exceptional cases leads to the development of a chronic pneumonia. It appears to be in those cases of pleurisy which are more or less chronic, and in which the lung remains long collapsed from the effusion, that such a result is most liable to occur. The induration of the lung thus induced is sometimes, however, exceedingly partial, consisting merely in some increase of the interlobular connective tissue originating and extending inwards as dense bands from the thickened visceral pleura. In other cases pleurisy gives rise to a much more general fibrosis.

(4) *Inhalation of solid irritating particles*.—This, which occurs in miners, potters, stonemasons, grinders, &c., is the cause of the fibrosis of the lungs so common among persons so employed. The continuous irritation of the inhaled particles induces a bronchial and alveolar inflammation, and ultimately a progressive fibrosis, which, gradually extending, may involve large areas or even the whole of the lungs. These cases often become tuberculous. *See PNEUMOCONIOSES*.

(5) *Atelectasis*.—Collapse of the lung which has existed for some time may lead to more or less fibrosis of the affected area.

(6) *Syphilis*.—A diffuse interstitial pneumonia is an occasional result of congenital syphilis.

ANATOMICAL CHARACTERS.—The histological changes met with in the lungs in chronic pneumonia may be described generally as consisting in the development of a fibrous tissue from the walls of the alveoli, from those of the bronchi, and from the interlobular connective tissue; which new growth, as it increases, and from its tendency to contract, gradually replaces and obliterates the alveolar structure. The character of these changes, however, varies somewhat according to the inflammatory antecedents in which they originate. When the result of acute pneumonia, the primary, and usually the principal, change takes place in the walls of the alveoli, although ultimately the interlobular tissue is involved. The alveolar walls become thickened by the growth of a small-celled tissue, which, in its earlier stages, usually contains new blood-vessels. The alveolar cavities which are not obliterated are either empty, or contain exudation-products and a few epithelial cells. In some cases the intra-alveolar exudation becomes organised.

When secondary to ordinary broncho-pneumonia, or to that induced by the inhalation of solid irritating particles, the new fibroid growth also originates principally from the alveolar walls. Here, however, in the earlier stages it is less uniform, and the peri-bronchial and interlobular connective tissues play a more prominent part in the process. The new peri-bronchial tissue invades the walls of the adjacent alveoli, and materially increases the fibrosis.

In the chronic pneumonia resulting from pleurisy, the change, as already stated, is usually more localised, consisting in the development of dense fibrous bands passing inwards from the thickened pleura. These are developed from the interlobular tissue. In other cases the fibrosis is more general.

In whichever of the pulmonary structures the new fibroid growth originates, all the connective tissue

of the lung may ultimately become involved, and as it increases the alveolar cavities may be completely obliterated.

The macroscopical appearances of the lung vary with the extent of the fibroid change. In the earliest stages of the induration resulting from acute pneumonia, where there is merely a slight thickening of the walls of the alveoli, the consolidation very much resembles that of red or grey hepatitis. It differs in being firmer and less friable in consistence, and is somewhat less granular. In the later stages, and in all cases where the fibrosis is extensive and general, the appearances presented by the lung are very characteristic. The organ is diminished in size, dense, firm, fibrous, in parts almost cartilaginous in consistence. The cut surface is smooth; and the large amount of irregularly distributed black pigment usually present gives to it a peculiar grey, marbled appearance. Numerous dilated bronchi traverse it in all directions.

The bronchi are almost invariably found dilated in those portions of the lung where the induration is advanced. This dilatation is often very considerable, the dilated tubes forming cavities, which may occupy a large portion of the indurated lung. The walls of the tubes are much thickened, and the mucous membrane is often ulcerated. This secondary inflammation and ulceration of the bronchi, which occurs especially in the dilated portions, is induced by the irritating and often putrid secretion which they contain. It may extend into and involve the indurated lung, and so lead to more or less excavation. The mucous membrane sometimes sloughs, and the gangrenous process may involve the lung. The cavities so common in these lungs are in the main, however, dilated bronchi. *See BRONCHI, Diseases of*.

The pleura of the affected lung, except in the earliest stages of the disease, is much thickened and adherent.

Site.—Chronic pneumonia is in the majority of cases unilateral. The whole lung may be involved or only a portion. In the latter case the base is much more commonly affected than the apex. When it is due to the inhalation of irritating solid particles, both lungs are usually implicated.

SYMPTOMS.—In the earlier stages of chronic pneumonia the symptoms are often very obscure, and it is not uncommon to meet with advanced and extensive fibrosis in which the lung-affection must presumably have been of much longer duration than the symptoms accompanying it. In some few cases the symptoms are directly continuous with those of some more acute pulmonary inflammation—an acute or a broncho-pneumonia. Under these circumstances, a prolongation of some of the phenomena of the original disease may indicate the superposition of the pulmonary fibrosis. The pyrexia does not entirely disappear. The cough persists, as does also some increase in the frequency of the respiration and pulse; and the patient, instead of improving, gradually loses strength and flesh. At the same time the physical signs of the pulmonary consolidation remain, and may gradually give place to those of pulmonary induration. Where chronic pneumonia is secondary to pleurisy, a continuous sequence in the symptoms is even less commonly observed. When it is the result of the inhalation of irritating solid particles, the symptoms of bronchial catarrh are predominant. *See PNEUMOCONIOSES*.

When the fibrosis is fully established the symptoms are usually more pronounced. They vary considerably, however, with the extent of lung involved, the situation of the consolidation, and especially according to the presence or absence of bronchial catarrh. When bronchial catarrh is absent, and the disease is quiescent, a considerable area, or even the whole of the lung, may be involved without producing any marked pulmonary symptoms; and slight dyspnoea and cough, with some general impairment of nutrition and failure of strength, may be almost the only phenomena present. Such quiescence and immunity from symptoms, however, although common in the course of the disease, are rarely observed over lengthened periods.

With the existence of catarrh of the bronchi, much more marked pulmonary symptoms are usually observable. Inflammation of the bronchi is especially favoured by their dilatation, and it is almost invariably present, to a greater or less extent, during the course of the disease. With it is usually associated activity of the indurating process. This dilatation of the bronchi, and secondary inflammation and ulceration of their walls, are most important factors in accounting for the symptoms. The course of the disease now often simulates that of chronic fibroid tuberculosis, but it is for the most part more chronic, less regularly progressive, and more frequently interrupted by periods of quiescence. The dyspnoea is now more marked, and cough becomes a troublesome symptom. The cough may be more or less constant, and it is usually attended by expectoration. Its characters vary, however, according to the situation and the extent of bronchiectasis. When, as is most frequent, the lower lobe of the lung is involved, the secretion accumulates, and its removal by expectoration is exceedingly difficult. Under these circumstances the cough is often violent and paroxysmal (*see BRONCHI, Diseases of*). The sputum may be simply mucopurulent, but when there is much bronchiectasis, owing to its accumulation and retention in the tubes, it often becomes septic, is of a greyish or greenish-black colour, and more or less fetid. This putrid secretion not only intensifies the inflammation in the bronchiectatic cavities where it originates, but is often conveyed by aspiration to other parts of the same, or the opposite lung, and so originates foci of broncho-pneumonia, which materially hasten the progress of the disease. Hæmoptysis is not infrequent, but it is usually small in quantity, and is in most cases due to ulceration of the bronchial walls.

Pyrexia is usually present to a greater or less extent in the course of chronic pneumonia. The fever, however, is exceedingly irregular, and there are often long periods of perfect immunity. During the pyrexial periods the maximum evening temperature is rarely more than 101° or 102° F., and it may be only 100° . The morning temperature is often normal. The pyrexia appears in most cases to be due to inflammation and ulceration of the bronchi. It is not infrequently the result of a supervening tuberculosis.

With the progress of the disease the patient gradually emaciates; the fingers become clubbed; digestion is impaired, and diarrhoea is often present. Dropsy is a common symptom, although it is rarely extensive, and is, for the most part, confined to the lower extremities. It appears in most cases to be due to the anæmia and impeded pulmonary circula-

tion. The pulmonary obstruction may also give rise to some enlargement of the right side of the heart, and cyanosis. Amyloid disease of the viscera is occasionally met with. Death usually results from the general failure of strength, or from some intercurrent affection of the opposite lung.

Physical signs.—In the earliest stage of chronic pneumonia, when it is the result of a more acute pneumonic process, the physical signs are, in the main, those met with during the acute consolidation. It is the persistence of the signs of the pulmonary consolidation that indicates a supervening fibrosis. Dulness on percussion; increased vocal fremitus; bronchial breathing; and the existence of *râles*, which are larger, moister, and more metallic in quality than those of fine crepitation, are observable during this stage. When the induration is fully established, the physical signs are those of contraction and consolidation of a whole or a portion of the lung, with usually those of more or less bronchiectasis. The retraction is well marked, and commonly affects the whole side, although when the lung is not universally involved it may be more limited. Expansion is deficient, or completely absent. The heart is displaced towards the affected side; the diaphragm and the abdominal viscera are drawn up; and the opposite lung may encroach considerably across the middle line in front. Percussion is hard, wooden, and high-pitched, sometimes more or less amphoric. If there be much pleural thickening, vocal fremitus may be diminished or absent, but vocal resonance is always increased. The respiratory sounds will vary according to the extent of the bronchial dilatation and excavation, and the amount of secretion. They are for the most part bronchial; usually large and loud; and often distinctly cavernous. When there is much secretion in the dilated bronchi, high-pitched bubbling *râles* are heard, which are often amphoric and cavernous. These may be audible only after cough. The opposite lung is usually enlarged and hyper-resonant, and the respiratory movement exaggerated.

DIAGNOSIS.—The diagnosis of chronic pneumonia rests mainly on the physical signs. The disease with which it is most liable to be confounded is chronic fibroid tuberculosis. The physical condition of the lungs and the symptoms of the two diseases are sometimes deceptively similar. In those quite exceptional cases in which chronic pneumonia involves only the upper lobe, the situation of the consolidation is very greatly in favour of its tubercular nature. This probability is infinitely increased if the other lung be affected. In unilateral basic disease, and in induration of the whole of one lung, the other lung being healthy, the tubercular nature of the disease is much less probable; although the occasional supervention of a tuberculosis in these cases, and also the possibility of a primary basic tuberculosis, must be borne in mind. Repeated examinations of the sputum for the *Bacillus tuberculosis* is usually the only means of settling the diagnosis.

PROGNOSIS.—Chronic pneumonia, when it involves a considerable area of the lung, usually tends ultimately to terminate in death, although under favourable circumstances life may be prolonged for many years. When the disease is limited, and remains quiescent, the general health and duration of life may sometimes be but little affected. The most important element in the prognosis is the con-

dition of the bronchi. The existence of bronchial dilatation, as evidenced by profuse, and often foetid, expectoration, is always unfavourable, as it not only weakens the patient, but is usually attended by extension of the induration, and may ultimately lead, in the dilated tubes, to ulceration of the bronchial walls and surrounding tissue, and occasionally to gangrene. Pyrexia, as another evidence of inflammation of the bronchi and indurated lung, is likewise unfavourable, as is also hæmoptysis. The latter indicates deep ulceration, and it may in exceptional cases endanger life. The general condition of the patient must also be taken into account in making a prognosis. Failure of strength and of digestive power, diarrhoea, and dropsy, are all of unfavourable augury.

TREATMENT.—In considering the treatment of chronic pneumonia, it is in the first place important to bear in mind that the usual origin of the disease is some more acute pulmonary inflammation. Hence the necessity for the most careful management and supervision of such inflammations in their later stages, with the object of procuring, if possible, a complete resolution of the pneumonic products.

When the fibrosis of the lung is established, it is hardly necessary to remark that the new growth is incapable of removal, and by treatment we can only hope to influence the extension of the disease, and control the bronchial catarrh with which it is so frequently associated. The frequency and gravity of bronchial catarrh has been already insisted upon; and its management, in the majority of cases, constitutes by far the most important element in the treatment. Our object must be to prevent and control it; to promote expectoration, and to prevent that decomposition of the secretion which is so apt to result from its retention. Here the question of climate will necessarily present itself, and very much may usually be done by residence at some suitable station. One not subject to vicissitudes of temperature, and at the same time dry and moderately bracing, is most likely to be beneficial. The patient should be warmly clad, and everything should be done, by means of diet and medicine, to improve the general health. Cod-liver oil and iron are often useful for this purpose. If an attack of acute bronchial catarrh supervenes, it should be treated at once, and the importance of quickly controlling it should not be forgotten.

In the treatment of the more chronic catarrhal process which is so often associated with profuse secretion, much may usually be gained by the use of stimulating and antiseptic inhalations, of which creosote, eucalyptus, and carbolic acid are, perhaps, the most generally useful. These not only tend to diminish the amount of secretion, and to prevent sepsis, but induce coughing, and so assist in its evacuation. These substances may be administered internally with the same object. Counter-irritation, especially painting with iodine, appears sometimes to be serviceable. Gastric disturbance, diarrhoea, hæmoptysis, &c., must be treated, as they arise, on general principles.

In cases of localised basic disease, when other treatment fails, the question of paracentesis and artificial drainage of a large bronchiectatic cavity might be entertained.

T. HENRY GREEN.

(Bacteriology) JOHN EYRE.

LUNGS, Inflation of.—This term is applied to two conditions. It sometimes signifies that condition in which the lungs are acutely and temporarily distended more or less with air, as in some cases of bronchitis, a condition which is usually called 'acute emphysema.' It does not give rise to any definite symptoms; but it can be made out by physical examination, the signs being those indicating distension of the lungs. No treatment, other than that of its cause, is needed.

'Inflation' is also a term applied to intentional expansion of the lungs with air, as carried out in certain methods of artificial respiration.

FREDERICK T. ROBERTS.

LUNGS, Malformations of.—There are no malformations of the lungs which can be regarded as of much importance from a clinical point of view. As anatomical peculiarities, the shape of these organs, or the arrangement of their lobes, may be abnormal.

LUNGS, Malpositions of.—These are rare. The most important malposition of the lung is that known as *hernia*, in which a portion of the organ projects into the neck, or through some part of the chest-walls, or through the diaphragm into the abdominal cavity. If the hernia passes towards the surface of the body it may be made out clinically, being indicated by a soft and compressible swelling under the skin, localised, resonant on percussion, and rendered more prominent by a cough. Pulmonary symptoms are usually entirely absent. It is practically impossible to detect a hernia of the lungs through the diaphragm.

FREDERICK T. ROBERTS.

LUNGS, Morbid Growths in.—Neoplastic growths in the lung are so frequently either secondary to similar growths in the mediastinum, or in the case of primary growths, mainly diagnosed on account of the invasion of parts outside the lung itself, that it is convenient to consider all forms of new-growth occurring within the thorax under **MEDIASTINUM, Diseases of.**

LUNGS, Œdema of.—**SYNON.:** Fr. *Œdème du Poutmon*; Ger. *Lungenödem*.

DEFINITION.—Infiltration of the pulmonary tissue with serous fluid.

The serous fluid is effused from the pulmonary capillaries into the pulmonary connective tissue, and into the alveolar and bronchial spaces.

ÆTIOLOGY.—The causes of this exudation are manifold, but of two sorts:—(a) *Disordered circulation:* (1) active congestion, attendant upon inflammatory conditions of the lungs and bronchi; (2) hypostatic congestion; (3) mechanical congestion—in heart-disease, emphysema, or pressure upon the pulmonary veins; (4) want of tone of vessels after inflammatory conditions, as pneumonia or bronchitis, or pressure upon the vagus nerve or pulmonary plexus; (5) afflux of blood to the lungs in croup, and during the asthmatic paroxysms determined by the ineffectual efforts at inspiration. (b) *Morbid conditions of the blood:* in albuminuria, and to a less degree in other diseases in which the condition of the blood is altered or impaired—for example, scurvy, purpura, anæmia, hydræmia—the lungs partaking of the general disposition to dropsy.

ANATOMICAL CHARACTERS.—In cases of œdema pulmonum, the lungs are usually large, filling the

thoracic cavity, and sometimes indented by the ribs. They are heavy; their pleural surfaces are wet; and the pleural cavities contain an excess of serum. Both lungs are as a rule affected, their lower and most dependent portions chiefly; and one lung, on the side to which the patient has last inclined, is more highly oedematous than the other. The higher the degree of oedema the less crepitant the lung, and the more distinctly the surface pits on pressure. A portion cut from a simply oedematous lung will, however, almost always float in water; but at the base of the lung there is usually some collapse in addition to the oedema, and a portion removed therefrom sinks. On section the lung exudes abundant thin serum, and more or less frothy fluid, with which the bronchial tubes are also occupied. On first making a section the succulent tissue will break down easily under the finger; but after the excess of fluid has been squeezed out, the lung feels toughened. The fluid is almost entirely confined to the alveolar spaces and parenchyma proper, as distinguished from the interlobular septa of the lung. Oedema may be found at any portion of the lung—at the apex, for instance, determined there by the inflammatory process. The transition between oedema and inflammatory consolidation is very gradual. Oedema is also very apt to pass into, or to be complicated with, a certain degree of inflammation. The degree of friability, and of compressibility, and the application of the water-test, are the readiest methods of distinguishing between the two. If a portion of oedematous lung be examined under the microscope the alveoli are found to contain more or less numerous large granular cells, but these are never so numerous as to occupy entirely the alveoli.

SYMPTOMS.—The symptoms of oedema of the lungs are—in addition to those of the disease which has produced it—dyspnoea, which may amount to orthopnoea; troublesome ‘retching’ cough; and difficult, yet tolerably abundant, frothy, serous expectoration. The percussion-note is deadened at both bases, although the dullness is usually more extensive at one base than the other; the vocal fremitus is diminished; the respiratory murmur is enfeebled or lost; and a fine bubbling crepitation is heard.

DIAGNOSIS.—The diagnosis of pulmonary oedema is not usually difficult. The absence of pleuritic pains and of fever, and the double-sidedness of the disease, together with the absence of any true bronchial breathing or ægophony, will exclude pneumonia or pleurisy. The presence of dullness will also distinguish the condition from simple capillary bronchitis, with a certain degree of which, however, it is often combined. The general condition of the patient, and the presence or absence of those diseases or circumstances which are known to produce oedema of the lungs, must be carefully taken into consideration. If, for instance, after an asthmatic paroxysm we hear some fine bubbling *râles* over the bases of the lungs, and find the patient expectorating an unusual quantity of frothy serous fluid, we may suspect pulmonary oedema rather than bronchitis.

PROGNOSIS.—The prognosis in oedema of the lungs depends mainly upon the general or local conditions with which it is associated. It is of very grave purport in chronic Bright’s disease, or in heart-disease. It is also a grave complication in chronic bronchitis, showing failure of heart-power.

It is, however, often a transient and unimportant condition when it succeeds to acute chest-affections, as pneumonia or bronchitis, or to asthma. As a complication of acute chest-affections, it is rarely recognised clinically.

TREATMENT.—Kidney- or heart-disease, if present, will mainly determine the exact treatment. If there be failure of cardiac power, ether, ammonia, and alcoholic stimulants are required; and if the heart’s action continues hurried or irregular, digitalis is especially indicated. Hydragogue purgatives should be administered, according to the strength of the patient. Diuretics are useful in some cases, especially the vegetable diuretics, such as digitalis, juniper, and scopolarium, as also spirit of nitrous ether; and the same is to be said of diaphoretics—for example, acetate of ammonium, warmth, hot-air baths. Moderate stimulation and support must be kept up. When we suspect a loss of tone of vessels, as after bronchitis or pneumonia and in anæmic states, perchloride of iron with some mineral acid is to be recommended.

Locally, poultices are to be applied to the chest, with sufficient mustard to produce redness. Dry-cupping will often give great relief (p. 358). Blisters should be avoided. In all cases rest in bed or on a couch is necessary. R. DOUGLAS POWELL.

LUNG, Perforation of.—**SYNON.** : The term *pneumothorax* is almost equivalent.

DEFINITION.—The formation of an opening through the pulmonary pleura, communicating with the interior of the lung.

ÆTIOLOGY.—Perforation of the lung may arise in many ways. Its causes may be classified under the four following headings:

1. *Penetrating Wounds*; for example, gun-shot wound, punctured wound, or laceration by a broken rib.
2. *Rupture of the Lung*; as sometimes occurs during violent expiratory efforts, e.g. in whooping-cough, parturition, &c.
3. *Diseases affecting the Pleura or neighbouring structures*; such as empyema, hepatic abscess or hydatid, or suppuration of the bronchial glands.
4. *Diseases affecting the Lung itself*; for instance, tuberculosis, emphysema, gangrene, hydatids, or cancer.

Of all the causes of perforation of the lung, tuberculosis is infinitely the most common. It is the rule in phthisis for pleuritic adhesions to form *pari passu* with the pulmonary lesion, and these adhesions are usually very firm and difficult to break down. In neither respect, however, does this rule always hold good. In some rare cases in the earliest stage of the disease a small tubercular nodule situated immediately under the pleura softens, and the pleura gives way. Again, at any stage of the disease an outlying tubercular mass, situated below the point to which the pleural adhesions have extended, may soften and rupture into the pleural cavity.

In the more acute cases of phthisis there is often a singular indisposition to the formation of pleural adhesions. The pulmonary pleura becomes covered with a thin, smooth, translucent layer of lymph, shining through which can be seen at several points opaque yellow spots. These spots are found to correspond with underlying caseous masses, by which the pleura has been undermined and deprived of its vascular supply. Pneumothorax has its most frequent

origin in rupture of the pleura at one of these yellow points.

Finally, sinuses are sometimes found leading from old cavities within the lung to the pleural surface. Occasionally these sinuses, the pleura being adherent, penetrate through the thoracic wall and point externally. In other cases they may open into the opposite pleural cavity.

ANATOMICAL CHARACTERS.—In all cases of pneumothorax, the affected lung is collapsed. In cases of old standing the collapse may be so complete, and the lungs so covered by the products of exudation, that the organ can be found only with difficulty. The opening may have closed, and is often difficult to discern. It may consist of a small slit, communicating with a cavity by a slanting sinus, so as to form a complete valve; or it may be of considerable size, and communicate widely with a cavity or bronchus. All degrees of patency between these two extremes occur. The position of the opening is very variable; it is most commonly situated somewhere on the lateral or convex side of the lung. The rupture is almost always into the pleural cavity on the same side. It may, however, take place into the opposite pleural cavity, through the mediastinal fold of pleura. The pleura is inflamed, and covered with lymph; and its cavity contains air and a greater or less quantity of purulent fluid. The heart is displaced, unless in some rare cases it be held by a strong adhesion. Some years ago the writer tested the degree of air-pressure present in ten cases of pneumothorax by means of a water-pressure gauge. In two cases it was *nil*; in one case it was equal to 1·25 inch; in two cases 2 inches; in one case 3·75 inches; in two cases 4 inches; in one case 5·3 inches; and in a double case it equalled 3·5 inches in one pleura, and 2·7 in the other. The gas effused approximates in composition to that of expired air, containing from 8 to 16 per cent. of carbonic acid. Sometimes sulphuretted hydrogen also is found in fetid cases.

SYMPTOMS AND SIGNS.—The symptoms and signs of perforation of the pleura are those of pneumothorax, followed by hydro- or pyo-pneumothorax. At the moment of attack sudden acute pain is felt in the chest, at the seat of rupture, and is immediately followed by great dyspnoea and shock. In a well-marked case the expression of face is peculiarly agonised and terror-stricken; the extremities are cold; damp sweats break out; the pulse is quick and small; and the respirations are exceedingly rapid. The position of the patient is that of orthopnoea, with an inclination forwards, and to the sound side; it is, however, frequently changed in the endeavour to gain breath. The voice is feeble and whispering. The urgency of the shock and dyspnoea depends upon the amount of useful lung suddenly disabled. If the patient survive the attack, after two or three days fever of a hectic character, with sweats, supervenes. In some cases, however, the symptoms of pneumothorax come on very insidiously.

Physical Signs.—The physical signs are very characteristic. There is enlargement of the side affected, and effacement or bulging of the intercostal spaces. The heart is displaced towards the sound side. The percussion-note is hyper-resonant or tympanitic over the side affected, except where (at the apex) the lung may perhaps be still adherent; and on auscultation either no respiration at all is audible, or amphoric breathing of a peculiar charac-

ter may be heard at one or more points, sometimes accompanied with the characteristic metallic tinkle. A peculiar metallic echo is heard if the patient coughs. If, while the ear is applied, a coin placed on the diseased side is struck with another coin, a characteristic bell-note is heard. The vocal fremitus is diminished or lost. At a later stage, when more or less effusion has taken place, the signs of air and fluid in the pleural cavity present themselves—namely, dullness below and hyper-resonance above—in varying proportions, and shifting in relative position with the posture of the patient. If the amount of fluid be moderate, a splash or succussion-sound may be elicited. This sound may be audible to the ear applied to the chest, or to bystanders. If the fluid effusion be considerable, intercostal fluctuation may be felt; and this fluctuation gives to the finger, on percussion at the level of junction of air and fluid, a peculiar sensation of thrill. The position usually assumed by the patient now is with the head raised, and leaning towards the diseased side.

DIAGNOSIS.—The diagnosis of perforation of the lung is to be made from other diseases; and also with respect to the probable nature of the opening, and the degree of pressure present. If the three essential signs of pneumothorax be remembered—namely, displacement of heart, tympanitic percussion-note, and either absence of respiration or amphoric breathing—there can scarcely be any difficulty in making the diagnosis. It cannot be confounded with a bilateral disease like emphysema. The shifting resonance and dullness, the succussion-splash, with perhaps metallic tinkle and amphoric breath-sound, are signs abundantly sufficient to distinguish hydro-pneumothorax from ordinary empyema. It is sometimes very difficult to distinguish between a *localised* pneumothorax and a large thin-walled cavity in the lung, the signs being almost identical. Respecting the nature of the opening, whether valvular or free, careful auscultation will usually gain the desired information. If amphoric breathing be well-marked, it may be assumed that the opening is a free and tolerably direct one; if, on the other hand, no respiratory sound be audible, the communication with the pleura is indirect and more or less completely valvular. In the latter case the pressure-symptoms become more urgent.

PROGNOSIS.—Of course the prognosis in every case of tubercular pneumothorax is necessarily very grave, but by no means equally grave in all cases. The following considerations will guide to a correct prognosis: (a) *Nature of opening.* If the communication with the pleura be valvular, signified by the entire absence of breath-sound, and the increasing urgency of dyspnoea, the patient will die in a few hours unless relieved by paracentesis. (b) *State of the opposite lung.* If the effusion of air have occurred on the side least affected by previous disease, the case is correspondingly hopeless. If, on the other hand, we know that the lung now collapsed was previously much diseased, and if the other lung be but little affected, the duration of life may not be greatly shortened by the accident. Life is then gradually extinguished by hectic fever, and progressive disease in the opposite lung. It is by no means impossible, and probably happens more frequently than is supposed, that the opening in the pleura may close, the air become absorbed, and the case converted into one of simple empyema. In pneumothorax arising from accidental wound or injury to the lung, the prognosis depends upon the

visceral injury. The air in the pleura is absorbed with considerable readiness.

TREATMENT.—In all cases in which death is threatened by asphyxia, in consequence of air accumulating in the pleura, paracentesis with a fine trocar must be performed. This will in such cases give great relief, and may be repeated if necessary. There is a tendency for an opening, at first completely valvular, to become at a later period more patent, or possibly to close, so that it is better to operate when necessary with a fine trocar than to make a permanent opening. Rest to the affected side should be secured, as far as possible, by the application of a broad piece of strapping extending round the side to beyond the middle line in front and behind. The shock and dyspnoea are best treated by opium in repeated small doses. Stimulants may also be necessary, but opium is far more useful.

R. DOUGLAS POWELL.

LUNG, Rupture of.—Rupture of the lung is an extremely rare occurrence. Cases of so-called rupture of the lung from external violence are, for the most part, really produced by perforation or laceration of the pleura by a fractured rib. It is said that rupture of the lung may occur in whooping-cough. See LUNG, Perforation of.

LUNGS, Syphilitic Disease of.—Even in advanced cases of acquired syphilis pulmonary lesions are rare. The lungs are more often involved in congenital syphilis.

ANATOMICAL CHARACTERS.—*Gummata* constitute the most certain and unquestionable lesions of a syphilitic nature in the lungs, but they are rare. When present, they vary in number from one to many. In the latter case they are disseminated, but are stated to have a predilection for the deeper parts of the organs. In size these growths usually vary from that of a pea to a walnut, but may reach the dimensions of a large egg. They are generally well-defined, rounded in shape, and often surrounded with a fibrous capsule. In their early condition gummata in the lungs appear on section greyish or brownish-red, homogeneous, firm, and dryish in consistence. Subsequently they tend to degenerate, becoming more or less caseous, yellow, and less consistent; and they may even break down in the centre, so as to form cavities; while irregular strands of fibrous tissue may radiate into the surrounding tissue. The structure of these gummata corresponds to that of similar formations elsewhere. See SYPHILIS.

There is some reason to believe that a variety of *chronic interstitial pneumonia* is in exceptional instances due to syphilis. The result is a fibroid infiltration of the pulmonary tissue, which in its general and microscopic characters cannot be distinguished from a similar condition due to interstitial pneumonia from other causes, but the new tissue is said to be more vascular in its early stages. The affected parts are much indurated; and any bronchi which are implicated tend to become more or less dilated. The morbid condition may be distributed in various parts of the lungs, but appears to have a preference for their bases and the vicinity of their roots. It frequently originates at the surface, and penetrates thence into the interior of the lungs in the form of fibrous bands, the pleura being generally thickened and adherent, and superficial puckering and depressions being visible. In other

instances the fibrous tissue spreads from the root of the lung. Syphilitic fibroid infiltration has no tendency to caseation; but it may become the seat of ulceration or gangrene.

In connection with congenital syphilis, a peculiar condition has been described as affecting the lungs in new-born or very young infants (*syphilitic pneumonia, white pneumonia*). It assumes a more or less diffuse or infiltrated arrangement, is of variable extent, and may involve one or both organs. One lung may be affected throughout, while the other is quite free from disease. The more obvious characters are as follows: The pleura is usually unaffected. The lung is enlarged, and may be in a state of full expansion, so that its surface is marked by the ribs; it feels remarkably heavy; and at the seat of the disease is dense, firm, hard, and usually resistant, not breaking down under pressure. On section it presents a white or yellowish-white colour, being more or less bloodless; is uniform and smooth; and little or no fluid can be expressed or scraped from the cut surface. Careful examination reveals minute bands of fibrous tissue running in all directions. Microscopically the change seems to consist mainly in thickening of the alveolar walls and minute bronchi, with proliferation and desquamation. The vessels also become thickened and ultimately obliterated.

A syphilitic bronchitis and broncho-pneumonia is also described.

SYMPTOMS.—In the majority of cases syphilitic lesions in the lungs have only been discovered after death, no symptoms having occurred during life to draw attention to these organs; or they have been obscured by symptoms affecting other parts. Physical examination might possibly reveal the presence of gummata, as evidenced by localised dulness, bronchial breathing, increased vocal fremitus and resonance, and other signs of consolidation. The most significant signs, however, are those indicating marked induration of the lung from fibroid infiltration, especially if unilateral, and confined to the base or middle portion of the organ. In course of time signs of cavities might become evident, due to breaking down of gummata or to dilated bronchi. The general symptoms are those of constitutional syphilis, combined with those of phthisis. There is but little or no pyrexia accompanying the pulmonary lesions; and the progress of the case is essentially chronic. The effects of treatment may be of peculiar significance in the diagnosis of syphilitic disease of the lungs. The absence of tubercle-bacilli is considered an important element in distinguishing syphilitic disease from tubercular phthisis; the latter may, however, be grafted on syphilitic lesions.

TREATMENT.—Should syphilitic disease of the lungs be recognised or suspected, the appropriate treatment in most cases is to administer iodide of potassium freely and continuously. In some instances a mercurial course of treatment answers best; or perchloride of mercury might be combined with the iodide. It may be necessary to employ internal remedies or local applications for the relief of pulmonary symptoms. Cod-liver oil and tonics may be given with advantage for the amelioration of the general condition, in cases where such medicines are needed. The usual measures for the treatment of phthisical cases, as regards diet, hygiene, climate, open-air, &c., must of course be duly recognised, and adopted in suitable cases.

FREDERICK T. ROBERTS.

LUNGS, Tuberculosis of.—This may occur as part of a general infection (see TUBERCULOSIS), or as a local affection of the lungs. See PHTHISIS.

LUPUS ERYTHEMATOSUS. — SYNON. : *Erythème centrifuge* (Biett); *E. atrophicans* (Malcolm Morris); *Ulerythema centrifugum* (ὀβλή, cicatrix), Unna; *Granuloma erythematosum* (Jadassohn).

DESCRIPTION.—Lupus Erythematosus is a cutaneous inflammatory eruption of a special type, characterised by the evolution of more or less rounded, often rather tender, erythema-like macules or discs, or somewhat raised and infiltrated congestive patches, which tend to spread excentrically to a limited extent, and to leave a notable atrophy in their wake. The colour will vary from that of active congestion to cyanosis, according to the activity of the process, the site of the lesions, and the state of the circulation. If the congestion be removed by pressure a degree of cellular accumulation is often disclosed, sometimes of a citron tint and varying in different cases with the duration of the lesion and the depth to which the skin is involved.

The eruption evolves by a crop or crops of lesions, or by single lesions at intervals, and the tendency to evolve may cease at any time or be renewed. Exceptionally superficial and transient, especially in the acute cases, the lesions, as a general rule, tend to become permanent and fixed, or remain quiescent with periods of threatened activity under the influence of various excitants. The sites of predilection are characteristic, viz. the nose, the cheeks, and other parts of the face, the ears, the scalp, the backs of the hands, less commonly the forearms and feet, and rarely the palms. Any of these sites may be affected exclusively or in combination. By extension and coalescence a large part of the so-called 'flush-area' of the face may be covered in a 'bathing' distribution, which is sometimes seen in lupus vulgaris. Bilateral distribution and symmetry are the rule. A rare but interesting phenomenon bearing on the pathogeny is the occurrence of an acute exanthematic outburst on the sites of predilection or involving even the trunk, associated sometimes with high fever and toxæmic symptoms, and even ending in death from pleuro-pneumonia or tuberculosis.

Various *modifications* are brought about by the degree of congestion, of cellular accumulation, of scaliness, of atrophy, &c. There is not the tendency to ulceration or warty overgrowth, however, seen in lupus vulgaris. A telangiectatic variety may be met with, due to extreme vascularity and dilatation of the vessels, and another interesting and striking phase, usually seen on the face, is due to the special implication of the sebaceous apparatus, causing an orange-rind appearance, or the formation of a crust, with prolongations into the dilated ducts (*L. seborrhagicus, sebaceus, acnéique*; *Herpès crétacé*, Devergie).

ÆTIOLOGY.—Lupus erythematosus is not hereditary, not contagious, and not auto-inoculable. Very rarely commencing in childhood, or even adolescence, it is not infrequent in the adult—about thirty years on an average—and females are especially prone to it. It tends to die out in old people. The exciting cause is a passing one, as a rule, but may be continued or recurrent. It is probably not directly due to external excitants, such as sunlight,

heat, and cold, or to reflex causes, but a specific eruption of internal origin. Possibly, however, the localisation is determined by vaso-motor influence. It is admitted that the eruption is prone to occur in persons with a poor circulation, without much stamina, and perhaps enfeebled by disordered menstrual functions or otherwise, and in the subjects of tuberculosis, especially of the neck-glands. It is said to be more common in the colder northern climates. The name is derived from a certain clinical similarity of some cases to lupus vulgaris, especially on the face. Most authorities, however, consider lupus erythematosus and lupus vulgaris distinct affections, and they point out the striking morphological distinctions of typical cases, the extension by continuity, the absence of auto-infection, the usual multiplicity of patches, the special regional distribution with frequency of scalp-implication, the symmetry, the age-incidence, the different histology, the infrequent reaction to tuberculin, the absence of tubercle-bacilli, the fruitless inoculations, &c. Some authorities, however, still affirm the intimate association with tuberculosis, either regarding lupus erythematosus as a special attenuated form of tuberculosis, or as connected with it in some way as yet unexplained. The marked frequency of a family history of tuberculosis, of symptoms of past or present tuberculosis, such as enlarged glands or 'tuberculides,' and of ultimate death from tuberculosis are insisted on; although in some cases the autopsy fails to disclose any trace of such lesions. If the intimate connection with tuberculosis be admitted the toxic theory perhaps affords the most probable explanation, or the dissemination of organisms of little virulence.

HISTOLOGY.—There is a chronic inflammatory infiltration commencing about the blood-vessels, with dilatation of the lymph-channels, often thrombosis, and a secondary atrophy of the collagenous tissue and breaking down of some cells. The epithelium is, on the other hand, dry and hyperkeratotic. The inflammation does not necessarily pick out specially the plexuses of the pilosebaceous apparatus, though it may do so.

DIAGNOSIS.—Obviously *L. erythematosus* may simulate various congestive and inflammatory patches, especially about the face, but the eruption usually is more persistent, and is accompanied by atrophy of the skin. In the absence of the latter the observer can only be suspicious of persistent patches about the face. From chilblains it is distinguished by persistence through summer and winter, the covering white dry cuticle, and tendency to atrophy as distinguished from scarring. The diagnosis of an unusually infiltrated *L. erythematosus* from an erythematoid *L. vulgaris* is sometimes a very delicate matter, especially if the related lymphatic glands are involved. Acute outbursts when confluent may simulate erysipelas on the head and face, and, when disseminated over the face and upper extremities, and perhaps trunk, erythema multiforme for a time. On the scalp deep-seated redness gives place to markedly bald atrophic areas, with, for a time, a red punctuation of the follicles.

PROGNOSIS.—In view of the class of patient apt to be affected with this eruption, and of its possible association with tuberculosis, the outcome is not entirely free from anxiety. For the rest, the eruption is one of the most intractable with which we have to deal, though it may disappear spontaneously

or remain quiescent, or on occasion be removed by treatment.

TREATMENT.—There is no specific internal or external treatment, and the refractory nature of the eruption is evidenced by the long list of remedies and methods put forward. A nice handling and experience are necessary.

The *internal remedies* in vogue may be classified as (1) those directed to the sustenance and building up of the health generally; (2) resolvent, as preparations of mercury and iodine; (3) vaso-motor, as quinine in large doses, salicin, salicylate of sodium, ichthyol, ergot, alkalis, and ammonium carbonate; (4) empirical, as arsenic and phosphorus.

Local treatment is of prime importance, and must vary according to the conditions present. All exposure to external irritation must be avoided. It is a rule not to irritate active patches, but to apply soothing and drying remedies; under such treatment the eruption sometimes disappears or remains quiescent and comparatively inconspicuous, and with this condition of affairs some patients are content. Among such remedies may be mentioned zinc oxide, calamine, bismuth oxychloride, terra silicea, starch, acetate of lead, &c., used in powder-form, such as Unna's pulvis cuticolor (zinci oxidi, boli rubræ, boli albæ, āā 2'0; magnes. carb. 3'0; amyli oryzæ, 10'0); calamine lotion, lead lotion, black wash, Dühring's zinc-sulphide lotion (zinc sulphate, potassium sulphide, āā gr. xxx; alcohol, 3 iij; rose-water, 3 iijss), and Boeck's liniment. When the eruption is quiescent or fixed we may proceed to try more powerful astringents or resolvents of different strengths, in the form of pigment, plaster, or paste. As examples may be mentioned oleate of mercury (3-5 per cent.), daily frictions with flannel dipped in Hebra's spiritus saponatus kalinus (2 parts soft soap in 1 of methylated spirit), to which tar may be added; emplastrum hydrargyri, the French *sparadrap rouge*; Unna's zinc-sulphur-paste, with the addition of ichthyol or cinnabar. The compressing effect of collodion is often useful, and to it many drugs may be added, as 10 per cent. ichthyol or 10-20 per cent. soft soap. Oxidised pyrogallol and chrysarobin are also recommended by Unna. A strong treatment at night is often usefully balanced by a soothing day-treatment.

Should these means prove unsuccessful and the patient demand further treatment, we are left with a choice of destructive remedies, viz. caustics, such as pure carbolic acid, ethylate of sodium, strong preparations of resorcin and pyrogallol, Schutz's arsenical treatment, acid nitrate of mercury, delicate puncturing or stroking with a fine cautery, multiple scarification. It must be remembered, however, that these measures produce scarring in addition to the atrophy left by the disease, and such scarring may be more disfiguring than the eruption partially concealed by appropriate cosmetics.

T. COLCOTT FOX.

LUPUS VULGARIS.—See SKIN, Tuberculosis of.

LYMPH (*lymp̄ha*, water, lymph).—Physiologically, lymph signifies the fluid which circulates in the lymphatic system. Pathologically, the term is still occasionally and incorrectly applied to the coagulable exudation which escapes from the vessels in inflammation. The name 'vaccine lymph,' or 'lymph,' is also given to the fluid con-

tained in the vaccine-vesicle. See INFLAMMATION; and VACCINIA.

LYMPH-SCROTUM.—See FILARIASIS.

LYMPHADENITIS (*lymp̄ha*, lymph; and *adenitis*, inflammation of a gland).—Inflammation of lymphatic glands. See LYMPHATIC GLANDS, Diseases of.

LYMPHADENOMA.—SYNON.: Hodgkin's Disease; Anæmia Lymphatica (Wilks); Fr. *Adénie* (Trousseau); *Lymphadénie* (Ranvill); Ger. *Pseudo-leukämie* (Wanderlich).

DEFINITION.—A disease characterised by more or less widely spread enlargement of the lymphatic glands, frequently accompanied by enlargement of the spleen, and progressive anæmia.

ÆTIOLOGY.—Very little is known of its causation. It occurs at all ages, but is more frequent in early and late adult life, being relatively uncommon between the ages of forty and fifty. Though intemperance, mental depression, insufficient food, over-exertion, exposure to cold, parturition, or febrile diseases, have occasionally been noted as antecedents, no one of these has occurred sufficiently often to be regarded as an adequate factor in its causation. The exciting cause which has been noted most frequently is some local irritation, as of a decayed tooth, discharge from the ear, sore throat, inflammation of the lachrymal sac or eczema. In these cases the glands nearest the source of irritation first enlarged, and then more distant ones were affected.

ANATOMICAL CHARACTERS.—The several groups of glands are affected in the following order of frequency, beginning with those most commonly diseased: cervical, axillary, inguinal, retro-peritoneal, bronchial, mediastinal, mesenteric. Subsidiary adjacent glands are often enlarged together with the chief groups; and nodular growths, similar to enlarged glands, arise in the course of the lymphatics in places in which the existence of glands is not usually recognised, so that continuous chains of nodules connect the various groups. The size attained by the glands in lymphadenoma varies from that of a bean to that of a hen's egg. At first the individual glands are separate and movable one on another. Ultimately they often unite to form a conglomerate mass, in consequence, in most cases, of the perforation of the capsules of the glands by growth, which may also invade adjacent parts. The cervical glands are usually enlarged in both the anterior and posterior triangles; and the submaxillary glands may encircle the neck beneath the lower jaw. They may press on the trachea or larynx, displace the latter, compress the internal jugular vein, and cause paralysis of the recurrent laryngeal nerve. The occipital glands are usually also enlarged. The axillary glands often form a mass of very large size, and prolongations may extend beneath the pectoral muscle. The glands in the anterior mediastinum frequently suffer, and the growth may extend to adjacent structures, such as the pericardium, which may be perforated. The thymus may be involved, secondarily or primarily, or may escape. The bronchial glands are diseased more frequently than the cardiac glands, and the trachea and bronchi may be pressed upon, or the lung invaded. The retro-peritoneal glands often form a mass of large size, which may surround and compress the solar plexus, causing symptoms similar

to those of Addison's disease. Enlargement of the mesenteric glands is neither common nor considerable. The inguinal group is frequently diseased, and the femoral vessels and crural nerves may be thereby compressed.

The consistence of the enlarged glands may be either soft or very hard. Usually the longer the enlargement has existed, the firmer are the glands. Their section is more uniform than in health. The colour is yellowish or whitish-grey. In the firmer glands dense tracts of fibrous tissue are seen to pass in different directions. Rarely the follicles have a different appearance, being opaque and yellowish from fatty degeneration, while the septa are white and conspicuous, from fibroid thickening. Caseation is, however, rare, and when it occurs is commonly confined to one or two glands. The softer glands yield a juice on scraping; the firmer glands yield no juice. In the former, the only histological change is an enormous increase in the cellular elements—the lymph-corpuscles of the reticulum; but the relations of the septa and follicles often remain normal. Sometimes the cell-growth invades the septa, which become split up and disappear; and it may even, in a similar manner, perforate the capsule. The firmer glands present much fibrous tissue, which may be confined to the septa, or invade also the delicate network in the substance of the gland; and then the cells gradually disappear, and the whole substance of the gland may be transformed into a fibrous mass. The tracts of fibrous tissue may have under the microscope a peculiar vitreous aspect, especially around the arteries.

The spleen is diseased in at least four-fifths of the cases, usually in consequence of disseminated growths, often irregular in shape, arising from the Malpighian corpuscles, yellowish or greyish-white, rarely caseating, and usually corresponding in consistence with the glands in the same case and resembling them in structure. The splenic pulp may be normal in quantity, or may be compressed and atrophied. In some cases it is increased in quantity, and this increase may even be the sole change. The size attained, in the cases of nodular growths, is not great, the weight being from ten to thirty ounces. When the splenic pulp is increased, the size attained is rather greater. In the latter case the enlargement is uniform, while it may be irregular when there are growths. The medulla of bones has been found, in rare cases, to present a change similar to that met with in splenic leucocythæmia and pernicious anæmia. Collections of adenoid tissue elsewhere often undergo changes similar to that of the lymphatic glands. The tonsils, the mucous membrane of the pharynx, the œsophagus, the stomach, and the large and small intestines, may all be the seat of growths, originating in the follicular glands, and sometimes ulcerating. The liver is often the seat of scattered lymphoid growths, usually minute, varying in size from a pin's head to a small pea. They occupy the interlobular spaces. Rarely larger nodular growths are found. In other cases the liver is simply congested. Similar minute growths are often found in the kidneys, chiefly in the cortex; and these organs may also be the seat of parenchymatous degeneration. The peritoneum may be inflamed over enlarged glands, or may be the seat of growths. Growths have also been found in the testicles; and frequently in the lungs, where they may break down and form cavities.

SYMPTOMS.—The most important symptoms of lymphadenoma are due to the enlarged glands and to the altered blood-state. The earliest symptoms are commonly due to enlargement of external glands, especially the cervical. When the internal glands are primarily affected, pain and pressure-signs may precede other symptoms. Occasionally the signs of anæmia precede those of the local change; and, in rare instances, irregular febrile disturbance may occur before the glandular enlargement. The affected glands are smooth, and present, at first, a peculiar mobility, which may disappear when they become adherent, and constitute an irregular lobular tumour of some size. They are usually painless, except during periods of rapid growth. A diminution in size has been observed before death. The enlargement of the cervical glands may cause the neck to equal, or even exceed, the head in circumference. The pressure on the veins may cause symptoms of passive cerebral congestion. The larynx may be displaced; and the movements of the lower jaw may be interfered with. Pressure on the trachea, by the glands in the neck and in the posterior mediastinum, may cause dyspnoea and even death by suffocation; that on the pharynx and œsophagus may obstruct deglutition, and cause death by starvation. The enlargement of the axillary and inguinal glands may interfere with the movement of the limbs, and impede the circulation. Various and serious pressure-effects result from the enlargement of the thoracic and abdominal glands, obstruction in veins, pressure on nerves, &c. The enlargement of the spleen can usually be readily felt. Earlier or later anæmia becomes pronounced. The red corpuscles may be reduced to 60, 50, or 40 per cent.; the hæmoglobin is diminished to a still greater extent (chlorotic type); and in severe cases poikilocytes and nucleated red cells (normoblasts) are seen. The leucocytes are generally not increased, but a moderate leucocytosis occasionally occurs, the white cells being mostly of the polymorphonuclear kind (*see* p. 156). The liver may be enlarged from the disseminated growths, and from congestion. Jaundice only occurs from the pressure of enlarged portal glands upon the bile-ducts. Ascites may be due to similar pressure, or to the blood-state, being then part of general dropsy. The function of the kidneys is rarely affected. Stomatitis, sometimes ulcerating, results from the lymphoid growth in the mucous membrane; and a similar change in the stomach causes interference with digestion and vomiting—symptoms which are increased by the anæmia. Slight dyspnoea results from the blood-state, while intense difficulty of breathing, and even actual suffocation, may occur from the pressure of enlarged glands on the trachea or bronchi. The functions of the nervous system are variously deranged by the ill-nourished blood. Towards the end there may be convulsions, delirium, and coma. Pyrexia is a frequent, but not invariable, symptom. It is almost always present in early life, much less common at advanced ages. The temperature may be considerably raised, even when the glandular enlargement is slight; the elevation varies from two to six degrees, and may be continuous, or with daily remissions, or periods of considerable elevation may alternate with periods in which it is only slightly raised.

COMPLICATIONS.—The pressure-effects of the enlarged glands, already mentioned, are sometimes so considerable as to give rise to complications,

such as thrombosis in vessels, pleural and pericardial effusions, and bronzing of the skin from disease of the solar plexus. Intercurrent affections occasionally met with are Bright's disease, pneumonia, fatty degeneration of the heart and liver, erysipelas, pemphigus and boils.

COURSE AND DURATION.—Lymphadenoma may remain local for a long time, even years, affecting one group of glands only, and subsequently slowly becoming general. When the general enlargement of glands is established, the disease rarely lasts more than two years. It usually terminates fatally by asthenia; but not rarely by some secondary effect of the morbid process, as asphyxia, starvation, or diarrhoea; or by a complication, especially by pneumonia.

PATHOLOGY.—The changes in the glands in lymphadenoma resemble, in the early stage, those which result from simple irritation. The nature of the disease is still very uncertain. It has often been compared with malignant disease, but in our ignorance of the nature of the latter the full value of the comparison is not obtained. The probability of its being in some way infective is very great. This view is supported by the frequency with which the first appearance of glandular enlargement follows a local irritation in its immediate neighbourhood, and by the manner in which the lesion remains for long periods confined to one group of glands, and afterwards rapidly becomes generalised not only in glands but in all lymphoid structures. Microscopical and bacteriological researches have not, however, as yet yielded any final results. The resemblance between some cases of lymphatic leucocythæmia and cases of Hodgkin's disease is very close in every point except the condition of the blood, and some authorities, regarding leucocythæmia as only an accident in the history of lymphadenoma, would include lymphatic leucocythæmia under Hodgkin's disease. But it is more common to accept, as the feature which distinguishes Hodgkin's disease from leucocythæmia, the absence of any leucocytosis, other than the moderate increase of polymorphonuclear cells, which occurs in inflammatory and other febrile conditions. See p. 156.

DIAGNOSIS.—The early stage of lymphadenoma is generally the most difficult to recognise with certainty: that is, when one group of glands is alone affected, remains stationary for weeks or months, and is unaccompanied by a sufficiently pronounced anæmia. Tubercular enlargement is generally confined to a single group of glands; it occurs in young people, and the glands caseate and suppurate; whereas the glands of lymphadenoma rarely caseate, and then probably form a secondary tubercular infection. Cancer may resemble lymphadenoma; the glands harden earlier; and secondary deposits are not so likely to take place in other glands as in internal organs. Syphilis sometimes causes a general enlargement of the external glands, which, combined with some anæmia, may suggest Hodgkin's disease. The glands are generally smaller and the condition more quickly generalised in the former disease.

PROGNOSIS.—When lymphadenoma is widely spread, or the local growths are considerable in size, a fatal termination is almost certain. The younger the patient and the better the preceding health, the longer is the duration of the disease. The consistence of the glands has little prognostic value. The softer they are, the more rapid is the

course of the disease; but, on the other hand, if it is influenced by remedial agents, the soft glands can be restored to a better functional condition than the hard. The prognosis is worse the more profound the anæmia. Elevation of temperature as a rule indicates a rapid course, but to this there are some striking exceptions.

TREATMENT.—The possibly infecting influence of the primary glandular enlargements has led to their extirpation. Where other glands have been involved, or the spleen has been enlarged, the operation has done no good; and, in such cases, surgical interference is only justified by impending death from local pressure. But where the affection has been confined to one group of glands, the progress of the disease has been retarded by their removal, and in some cases the malady has even been cured. An operation should not be performed if the proportion of red corpuscles is less than 60 per cent. of the normal. Other methods of local treatment have been employed, with some benefit, especially rubbing and shampooing, the alternate application of heat and cold, compression, and blistering. Of internal remedies arsenic is incomparably the most potent. It should be pushed to the largest doses the patient can bear, such as mxv . of liquor arsenicalis three times daily. It often causes some pain in the glands, followed by their diminution in size, and even, in a few recorded cases, by their complete disappearance. Phosphorus has been given in the disease, but it is far less useful than arsenic. Cod-liver oil may be used when there is any evidence of a scrofulous diathesis. Iron, useless alone, has sometimes appeared to do good when given in conjunction with other remedies. Of late years the animal extracts have been employed, but with no material success. Bone-marrow of the ox, from the fact that the marrow is sometimes involved in Hodgkin's disease, and from its occasional value in pernicious anæmia, may be tried in quantities of one ounce daily. Some assistance may be derived from change of air, general tonics, and careful dieting.

W. R. GOWERS.

FREDERICK TAYLOR.

LYMPHANGIECTASIS (*lymp̄ha*, lymph; and *angiectasis*, vascular dilatation).—Lymphatic varix, or varicose dilatation of lymphatic vessels. See LYMPHATIC VESSELS, Diseases of.

LYMPHANGITIS (*lymphanḡeion*, a lymphatic vessel).—Inflammation of lymphatic vessels. See LYMPHATIC VESSELS, Diseases of.

LYMPHATIC GLANDS, Diseases of.—I. Acute Inflammation. — **SYNON.**: *Lymphadenitis*.

ÆTIOLOGY.—*Strain*, as in lifting weights, or overwalking, is sometimes alleged as the cause of inguinal lymphadenitis, and no point of infection is discoverable. It is difficult to see how strain can prejudicially affect these glands, and the effect can be only predisposing to suppurative inflammation. Possibly infection through the blood may occur.

Deposit of *organisms*, usually pyococci, in the lymph-channels of the gland is the commonest cause. As a rule the organisms are arrested in the peripheral spaces into which the afferent lymphatics open. An infected lesion is almost always discoverable in the lymph-area supplying the gland. The specific unknown virus of the *chancroid* and the *Bacillus pestis* are other causes.

MORBID ANATOMY.—An acutely inflamed gland is considerably swollen; its capsule is tense and purplish. Its surface on section bulges, is of purplish tint, less translucent than natural, and may show hæmorrhages; its substance is more friable and more succulent than natural. If suppuration is beginning, one or more yellowish points will be seen, usually in the cortex; these increase in size, run together, and ultimately fill the capsule which adheres to surrounding parts. Infection of the tissues around such a gland may occur early or late, and a periadenitic abscess be formed. Into the cavity of this a gland may project.

SYMPTOMS.—These are pain and the rapid swelling of one or more glands, the outline of which soon becomes indefinite owing to periadenitic infiltration and possibly suppuration. If pus forms the skin thins, reddens and bursts, as over any other 'pointing' abscess.

DIAGNOSIS.—After the swollen gland has ceased to be felt, this depends on the signs of an acute inflammation starting in the situation of a gland. The discovery of a primary focus of infection affords additional evidence.

TREATMENT.—This consists in giving rest and applying fomentations to the gland, and in disinfection and suitable treatment of the focus whence infection started. If pus forms a free and early incision should be made, the opening be lightly plugged with gauze, and the fomentations continued. If the patient is anaesthetised, removal of the shell of a suppurating gland will hasten healing. A gland in the centre or wall of a periadenitic abscess should be removed. The quickest way to deal with long persisting sinuses after glandular abscess is to slit up the channels and to remove the remains of the glands to which they lead. If densely fibrous, the walls of the sinuses should be dissected out or cut quite through in two or three lines.

2. Chronic Lymphadenitis.—**ÆTIOLOGY.**—

(1) Slight chronic irritation, such as proximity to a focus of chronic suppuration, renders glands somewhat large, hard and pigmented. (2) Syphilis in all its stages causes glandular enlargements—hard, discrete, rarely reaching any size or causing trouble in the primary or secondary stage; but in the tertiary, gummata of mediastinal and other internal glands may rarely form masses which produce serious pressure-symptoms (*see* BRONCHIAL GLANDS; and MEDIASTINUM, Diseases of). (3) Infection with tubercle-bacilli is the commonest cause of chronic lymphadenitis. In infancy the bronchial and mesenteric glands are most commonly affected, the virus usually reaching them from the pulmonary and intestinal mucosæ respectively. In childhood and later life the cervical glands are much more often affected than the other superficial glands. Carious teeth, tonsils, eczema and sores about the scalp and face and the middle ear, are all suspected as sources whence the bacilli have reached the glands; but in the great majority of cases no history of any lesion likely to have been the source of infection can be obtained. Either the bacilli enter through the mucous membrane without exciting any local trouble; or, having entered at some distant place, are carried by the blood to the glands.

MORBID ANATOMY.—Tuberculous glands are moderately enlarged; one the size of a walnut is exceptional. The connective tissue round about them is usually dense, and the connection between

it and the capsule of the gland closer than normal (*periadenitis*). On section, the surface of some glands becomes markedly convex, is of pale pink colour and softer than normal. Microscopically these glands show early fatty degeneration of the giant and other cells. To the naked eye caseation in smaller or larger patches is usually visible, and often the whole gland right up to the capsule is converted into a friable, opaque, pale-yellow mass. This often softens into a thick white cream or a thin curdy pus which slowly makes its way to the surface. Calcareous granules, or considerable masses of calcified cheesy material, are not rare. Sometimes the tissues round the gland become infected and a periadenitic abscess results.

SYMPTOMS.—Not uncommonly tuberculous glands swell up somewhat acutely at the start and then shrink, but do not disappear. In the great majority of cases, however, the enlargement begins insidiously and the glands are discovered by accident. Their course is very variable and is much influenced by the general health. Under favourable conditions, many swollen tuberculous glands, even of considerable size, subside and may disappear; but an intercurrent illness will often lead to rapid increase. For years small, hard, discrete glands may stud one or both sides of a neck; then, without obvious cause or in connection with some depression of health, one or more glands enlarge, soften, lose their outline owing to œdema and infiltration of surrounding tissues, and give rise to pain. Slowly the skin reddens, darkens and thins over it, and finally the abscess bursts, leaving one or more sinuses surrounded by thin blue edges. After months or years, during which dense fibroid thickening of the surrounding tissues occurs, and after more or fewer temporary cessations of discharge and closure of the orifices, healing takes place by a prominent red puckered and unsightly scar. Only one gland may be affected, and it may never suppurate; or one or both sides of the neck may be deformed by the rounded lobulated masses. They may appear also in one or both axillæ, and much more rarely in the groins. In some cases the tendency is towards caseation and drying; in others abscess after abscess forms. Other manifestations of tuberculosis may be present; but a considerable majority of cases of enlarged glands fail to produce pulmonary or general tuberculosis.

DIAGNOSIS.—It is impossible in many cases of slight chronic enlargement of the lymphatic glands to ascertain definitely whether they are due to tubercular or some less progressive form of infection. A history and other signs of tuberculosis, slow course, limitation to one region, tendency to soften, tendency to become fixed by periadenitis, absence of marked and progressive anæmia, of tendency to lethargy, of wasting and of fever, are all in favour of tubercular infection as opposed to lymphadenoma. A somewhat rapid course, a tendency to spread from gland to gland, and from one group to others, considerable enlargement of glands without any signs of softening or of fixation by periadenitis, marked anæmia, possibly leucocythæmia, tendency to lethargy, loss of flesh, and evening rise of temperature, are in favour of lymphadenoma. But the diagnosis between tubercular infection and lymphadenoma is impossible at first. Microscopical examination of a removed gland is useful. *See* LYMPHADENOMA.

A rapidly growing tumour starting in glands, spreading quickly to other glands but not to other

groups, soon becoming fixed by infiltration of surrounding parts, adhesion of individual glands to each other with rounded lobulation of the general surface of the mass, signs of great vascularity, the occurrence of secondary growths, and rapid failure of general health, point to lymphosarcoma.

TREATMENT.—In all cases of chronically enlarged glands, general hygienic treatment is of the first importance. This consists in residence as far as possible in the open air and in a dry climate, and in the thorough ventilation of all rooms both by day and night. As a rule the windows should be always kept open, draughts being prevented by sheets of perforated zinc, lowered shutters, or some other mechanical contrivance. Woollen garments should be worn next the skin. All milk should be boiled or efficiently sterilised, and drinking-water filtered through porcelain or boiled. When the cervical glands are affected the mouth should be cleansed twice daily with a weak solution of formalin (1:500), boric acid (1:20), or chinosol (1:1200). In tubercular cases, whether operated upon or not, it may be necessary to keep patients under exceptionally favourable circumstances for very long periods in order to overcome the tendency to recurrence. In many cases such general treatment, coupled with attention to all possible primary foci, is sufficient. But when the best hygienic circumstances procurable for a given patient have failed to check the progress of the disease, removal of the affected glands and of the neighbouring apparently healthy glands for some distance around should not be postponed. The skin should never be allowed to redden, much less to thin or burst, over a gland. Very extensive operations upon one or both sides of the neck, in the course of which all the great vessels and nerves come to light, and which last two hours or even longer, are well borne, and danger to life is extremely slight. In a considerable majority of the cases dealt with by thorough operation a cure seems to be effected. In many cases more than one operation is necessary to effect this; but every now and again recurrence follows recurrence, the freest operating proving of no avail. In all cases the best hygienic conditions must be obtained and continued for months or years. It is worthy of note that incisions placed in or parallel to the creases in the skin of the neck leave scars which are much less prominent than those of wounds in the long axis of the neck; also, that it is possible to remove glands from a wide area by burrowing from a small incision. But in proportion as access is impeded does recurrence appear more probable. When sinuses are present the incisions should be planned to excise the sinuses themselves, their orifices (including all thin blue undermined skin), and the glands to which they lead. No operation is likely to succeed unless attention is paid to the last point. Often the cheesy gland responsible for the trouble communicates by means of a small easily overlooked hole in the deep fascia with the cavity which itself seems to be the whole trouble, and to be adequately dealt with by erosion; or, again, sinuses low in the neck may spring from a suprahyoid gland. All tubercular granulation-tissue lining the sinus should be removed; and rapid healing is more likely to occur if fibrous sinus-walls and gland-capsules are dissected out. When for any reason tuberculous tissue cannot be cut out, the sharp spoon must be used freely, and phenol should be applied freely to the

infected tissues left by the spoon. In such cases plugging the wound with iodoform gauze is the best mode of dressing. During operations such as these disfiguring scars, the result of Nature's cures, can often be excised and replaced by fine lines.

3. Pigmentation.—Lymphatic glands (e.g. bronchial) may become black from deposit of dark particles (*see PNEUMOCOINOSIS*). Blood-pigment is deposited in them from all serious contusions, the granules often lining the lymph-path. After tattooing some of the pigments used may be found in the nearest glands.

4. Fatty metamorphosis.—According to Stiles and some other observers many glands in the axilla become changed under ordinary conditions to masses of fat having the capsule and shape of a gland with blood-vessels entering at the hilum. Under various stimuli, e.g. cancer mammae, the fat goes and the ordinary structure of a lymph-gland reappears.

5. Amyloid disease.—This is occasionally met with in lymph-glands, rendering them pale, homogeneous, waxy, and translucent. They may be of normal size and firm, or enlarged and softer. Such glands sometimes press on structures in the portal fissure.

6. Varicose lymphatic glands (Manson).—These are seen chiefly in the groin as a result of obstruction of the thoracic duct. *See FILARIASIS*.

7. New growths.—Occasionally a single gland enlarges without marked change of structure. There are no signs of inflammation; no other glands become affected, and no general symptoms are produced. Such a swelling is sometimes called a *lymphoma*. In acute cases of *lymphadenoma* and *leucocythemia* the glands may enlarge rapidly, retaining the lymphoid structure. Many glands may be affected, and many groups may be involved. *Sarcomata* of all kinds, including *melanotic*, are said to have arisen in lymphatic glands. They pursue their ordinary course but tend to infect the lymphatic glands nearest to those in which the growth started. *Sarcomata* as a rule do not produce secondary glandular growths, but *sarcomata* of the testis, fascia lata and tonsil are marked exceptions to this rule. All *cancers* tend to produce secondary glandular growths before spreading by the blood-stream. Glands so infected are generally very hard and fixed; but when secondary to epitheliomata they not uncommonly soften, and cavities form containing a fluid like pus in which the microscope shows plenty of deformed and obviously multiplying epithelial cells.

TREATMENT.—The only treatment is removal. The advisability of this will depend upon the extent of the disease and other considerations.

STANLEY BOYD.

LYMPHATIC VESSELS, Diseases of.—As it is impossible to dissociate the diseases of the primary lymph-spaces and of the smallest lymph-channels from those of the connective tissue (e.g. erysipelas, cellulitis) in which they lie, only the diseases of the larger lymphatic vessels, with distinct walls, will here be considered. For Injuries and Diseases of the Thoracic Duct *see* THORACIC DUCT.

1. Acute Inflammation.—*Acute Lymphangitis.*—The manifestations of inflammation are seen much less frequently in the lymphatic vessels than in the glands; owing to the inability of particulate

irritants to excite inflammation until they come to rest, and to the much greater frequency with which they are deposited in the labyrinth of the lymphatic glands, than in the relatively straight channels of the vessels.

ETIOLOGY.—In the enormous majority of cases lymphangitis arises in connection with some obvious focus of infection—a wound or ulcer, an abscess (open or closed), or a patch of erysipelas—whence micro-organisms (usually pyococci, see ABSCESS; and PYOGENIC BACTERIA) have entered the lymph-channels. Arrested by some more or less accidental circumstances, they soon excite irritation and thrombosis. Rarely, the source of absorption is not evident; it may have been a mere unnoticed prick, without any subsequent local reaction. This is specially seen in dissecting and *post-mortem* rooms. The pathogeny of the lymphangitis and cellulitis which precede the development of tropical elephantiasis is not yet certain. In some cases, after subsidence of the inflammation, an indurated patch has remained in which a living adult female filaria has been found; but the subsidence of the inflammation may be held to show that the filaria alone did not excite it. Lymph-stasis seems to predispose to lymphangitis; the writer has seen six attacks of lymphangitis of a slightly swollen arm occur within a few months of a thorough clearing out of the axilla for cancer. The filaria may cause lymph-stasis, and thus facilitate ordinary infection. Lymphangitis by extension from inflamed surrounding tissues doubtless occurs, but is not recognisable clinically.

SYMPTOMS.—At first pain and tenderness are complained of along the course of the affected lymphatics; soon a broader or narrower red streak appears, or there may be several such streaks, indicating the line or lines of the inflamed vessels. These streaks do not necessarily begin at the point of infection. They are red, raised, firm, tender, and irregular in outline; and are due to perilymphatic hyperæmia and exudation. The glands to which they lead are inflamed.

The temperature rises sharply at the outset, and a rigor is not uncommon. The fever usually subsides in three or four days, but some cases run a prolonged course and, exceptionally, supuration may occur at one or more points along the track of the lymphatics.

The deep lymphatics are probably as liable as the superficial to inflammation, but the trouble cannot be certainly diagnosed from pain, tenderness, fever and swollen glands—the only symptoms. See CELLULITIS.

TREATMENT.—(1) Whenever possible, the source of absorption should be disinfected with phenol. Thorough drainage should be secured, and suitable local treatment adopted. (2) General and local rest with elevation of the affected part must be obtained. (3) Hot antiseptic fomentations should be applied. Painting the streaks with glycerin and belladonna may be beneficial. Some surgeons prefer ice to fomentations. Abscesses must be opened early. Should chronic oedema or thickening of tissues remain, douching, massage and bandaging are the appropriate remedies, though too often unsuccessful.

2. Chronic Lymphangitis.

A chronic *lymphangitis obliterans* is described as playing an important part in cases of lymphoedema and elephantiasis of unknown aetiology arising in the temperate zone.

Syphilitic lymphangitis is of little importance. It is best known and typified by the thick cord of the dorsal lymphatics, not uncommonly felt above a hard chancre on the penis.

Tubercular lymphangitis of subcutaneous vessels is somewhat rare. Slowly, during months, small lentil-like nodules form in the course of the lymphatics above some tubercular ulcer—obviously at points where the bacilli, taken up from the primary lesion, become arrested. At first these nodules cause no general symptoms and no local trouble. Gradually increasing, they infiltrate the surrounding tissues and skin, soften, burst and leave a tubercular ulcer. Early in the case the glands to which the lymphatics lead will be found enlarged.

TREATMENT.—*Syphilitic lymphangitis* needs no special treatment. In *tubercular lymphangitis* the primary focus must be excised or otherwise suitably treated. The enlarged glands and all subcutaneous nodules must be removed. As these may continue to develop for weeks after removal of the primary focus, the patient requires watching for some time. Suitable general treatment is indicated.

3. Dilatation of Lymphatic Vessels.—

SYNON. : *Lymphangiectasis*; *Lymphangiooma*.

ETIOLOGY.—1. A lymphatic vessel having more than usual to carry dilates. This frequently happens, especially in acute inflammations. Lassar showed that no swelling of a dog's foot occurred after dipping in hot water, until the main lymphatics were delivering more than eight times the normal quantity. Such dilatation causes no characteristic signs, and recovery is as a rule complete. 2. The usual cause of dilatation is obstruction, but so free is the intercommunication of the lymphatic vessels and of the lymph-spaces in the tissues that the obstruction must be very extensive, or affect trunks of the first magnitude, to produce any marked effect. The best examples of lymphatic varix are seen in lymph-scrotum and other results of filariasis (see FILARIASIS). Often in the scrotum, more rarely in the leg, small vesicles, scattered or thickly set, occur, consisting of fluid and raised epidermis; these are varicose lymphatics. When pricked or ruptured many ounces of chyle or lymph may escape (*lymphorrhagia*), the flow lasting hours, or even days, and tending to exhaust the patient. From such fistule and slighter abrasions it is probable that ordinary infections start and induce the attacks of lymphangitis and cellulitis, which seem to convert the 'lymph-scrotum,' or merely oedematous leg, into a solid elephantoid mass. But if we may judge from some of the elephantoid legs which develop in this country, inflammatory attacks do not seem essential to this change, though they doubtless quicken the thickening of the tissues, and produce the most extreme degrees of induration. In elephantoid limbs, such as occur in this country, the inner coats of both arteries and veins are much thickened and the lumina diminished; many of the lymphatic trunks are obliterated. The smaller lymphatics and lymph-spaces are of course dilated.

In spite of the frequency and freedom with which glands are removed for tubercular and malignant disease, it is unusual to see oedema result: probably because a collateral circulation was opened up gradually as the glands, subsequently removed, became blocked. When oedema does occur, it is difficult to eliminate venous thrombosis. Removal of the femoral glands, and especially of the gland and tissue in the crural canal, has seemed to the writer

very liable to produce lymph-œdema. In such cases varicose lymphatics are rarely seen. Collections of lymph may form at points where the subcutaneous tissue is loose, e.g. over the olecranon process, accompanied by many of the signs of inflammation; but an incision evacuates only an orange fluid, and a lymph-fistula may be established. The connective tissue of a part which is the seat of lymph-œdema hypertrophies slowly, and the epithelium thickens and becomes coarse.

The obstruction caused by even the most extensive tubercular affection and caseation of the glands of a part seems never to cause lymph-œdema. In malignant disease œdema, e.g. of the arm in cancer of the breast, is common, but compression of the axillary vein is probably always the chief factor in its causation. The effect of plugging the glands and the lymphatics of the skin and subcutaneous tissue with cancer-cells must, however, be considerable, in proportion as the obstruction is widespread.

In certain congenital affections, of which macroglossia and macrocheilia are the best known, the part affected is enlarged, and its connective tissue is increased. Dilated lymphatics may be visible to the naked eye. In macroglossia the surface of the tongue is coarse and warty from enlarged papillæ: microscopical examination will show them to be occupied largely by dilated lymphatics. Proof of physical obstruction to the return of lymph in these cases is usually lacking. By some pathologists these and similar formations are regarded as tumours—*cavernous lymphangioma*. In more than one instance macroglossia has been associated with *cystic hygroma* of the neck. See CYSTIC LYMPHANGIOMA.

There are also recorded a few cases resembling diffuse hypertrophy or excessive development of the subcutaneous tissue, always congenital, but sometimes growing faster than the rest of the limb. On removing the mass, numerous small openings have been visible in the fat, and under the microscope the structure of a cavernous lymphangioma has been revealed. These 'tumours' are vague in outline, are not compressible, like nævi, and show none of the bluish or purplish tint of hæmangioma; they closely resemble diffuse lipomata.

TREATMENT.—In the slighter forms, elevation, massage, and bandaging are the remedies; in the more severe, excision of parts, with careful suture both of the deep tissues and of the skin to prevent leakage and promote union, thus avoiding a subsequent fistula. Asepsis is most important, as there is a predisposition to infection. In the most severe cases removal of the part may be necessary to rid the patient of an encumbrance.

For the treatment of cystic hygromata see CYSTIC LYMPHANGIOMATA. STANLEY BOYD.

LYMPHOCYTES.—See BLOOD, Morbid Conditions of.

LYMPHOCYTHÆMIA.—See LEUCOCYTHÆMIA.

LYMPHOMA.—See LYMPHADENOMA.

LYMPHORRHAGIA or **LYMPHORRHŒA** (*lymphæ*, lymph, from *λύφω*, water; and *ῥήγνυμι*, I burst forth, or *ῥέω*, I flow).

DEFINITION.—These terms literally signify a flow of lymph, but they are used to indicate an abnormal discharge from any part of the absorbent system, whether it be of lymph or of chyle.

ÆTIOLOGY AND PATHOLOGY.—Lymphorrhagia may be due to a wound affecting either the thoracic duct, the larger lymphatic trunks, or the glands. A discharge of lymph has in rare instances followed even a slight wound, particularly in the neighbourhood of joints, and this has been attributed to a constitutional defect—a *lymphorrhagic diathesis*, corresponding to the hæmorrhagic diathesis. Idiopathic lymphorrhœa is almost always the result of dilatation of one or more vessels, which ultimately rupture. They are often greatly distended before they give way. See FILARIASIS.

SYMPTOMS AND EFFECTS.—Should an escape of lymph take place upon any part of the surface of the body, it differs much in its quantity and characters in different cases. It may be less than an ounce, or amount to five and even ten pounds within the twenty-four hours; while in the same case its quantity is liable to variation from time to time, and the flow has even been known to assume a periodic character, increasing during the period of digestion. In traumatic cases the discharge either presents the ordinary appearance of lymph, being clear and limpid, or it is mixed more or less with blood or with inflammatory products. When rupture takes place spontaneously after dilatation of the vessels, the fluid is more like chyle, being more or less milky and white, from the presence of particles of fat, but its characters are liable to alter from time to time. It contains a variable quantity of fibrinogenous elements, and is proportionately disposed to coagulate spontaneously. Internal lymphorrhagia is attended with different results. In the case of the intestines and urinary organs the fluid is discharged with the fæces and urine respectively, in the former case being supposed to give rise to fatty stools, and in the latter to chyluria. Bradley attributed some cases of effusion into serous cavities, such as certain forms of hydrocele, hydrocephalus, pleuritic effusion, and ascites, to a lymphorrhagia into the respective cavities; and the writer and other observers have met with cases of ascites which seemed to support this view. Fatal peritonitis has resulted from the entrance of chyle into the peritoneum, owing to the rupture of a dilated receptaculum chyli. The escape of lymph or chyle out of the system tends to affect the general health, and, if it is in large amount, this is likely to lead to marked emaciation, debility, and anæmia.

TREATMENT.—In external lymphorrhagia all that can be done is to check the flow of lymph by pressure of bandages and the application of astringents. In cases where it takes place into internal passages, tincture of perchloride of iron in full doses may be of service.

FREDERICK T. ROBERTS.

LYPEMANIA (λύπη, grief; and *μανία*, madness).—The name applied by Esquirol to the form of insanity characterised by mental depression, usually called melancholy. See MELANCHOLIA.

LYSIS (λύω, I dissolve).—This word is now generally applied to the gradual decline of fever. See FEVER.

LYSSOPHOBIA (λύσσα, madness; φόβος, fear). A term applied to a combination of symptoms presenting superficial resemblance to hydrophobia. See HYDROPHOBIA.

M

MACROCHEILIA (*μακρός*, great; and *χείλος*, the lip).—A condition, usually congenital, in which the lips are hypertrophied.

MACROCYTE (*μακρός*, great; and *κύτος*, a hollow).—A form of large red blood-corpuscles met with in some kinds of anæmia. *See* BLOOD, Morbid Conditions of.

MACRODACTYLIA (*μακρός*, great; and *δάκτυλος*, a finger).—Hypertrophy of one or more fingers, either congenital or developing in childhood.

MACROGLOSSIA (*μακρός*, great; and *γλῶσσα*, the tongue).—Fr. *Macroglossie*; Ger. *Zungenverfall*.

The term 'macroglossia' is applied to an enlargement of the tongue, which sometimes goes to the extent of protrusion of the organ from the mouth. This affection seems to depend on dilatation of the lymphatics, with lymph-stasis, leading to hyperplasia of the connective and lymphoid tissues. It is sometimes associated with idiocy or imbecility. The cause of the lymphangiectasis, which is probably the fundamental condition, is undecided. *See also* TONGUE, Diseases of.

MACROSOMATIA (*μακρός*, great; and *σῶμα*, the body).—Fr. *Macrosomatie*; Ger. *Riesenwuchs*.—A condition in which the whole body becomes enlarged in a monstrous degree. *See* HYPERTROPHY.

MACROSTOMIA (*μακρός*, great; and *στόμα*, the mouth).—A congenital enlargement of the mouth, due to imperfect closure of the mandibular fissure upon one or both sides. It is a rare deformity, and is generally accompanied by malformation of the auricle, by an accessory tragus, or by a mandibular tubercle. Excessive closure of the mandibular fissures may also occur, leading to *microstoma*.

MACULÆ (*macula*, a spot or stain).—SYNON. : Fr. *Macules*; Ger. *Flecke*.

Maculæ are spots or small stained areas due to localised pigmentation, hæmorrhage, or inflammatory exudation, in or beneath the skin. Sunburn, freckles, bronzed and melasmic spots, and the stains left on the skin after the dispersion of certain cutaneous eruptions, such as psoriasis, acne, lichen planus, syphilis, and leprosy, are examples of maculæ.

MADEIRA, North Atlantic Ocean.—Moist, mild, equable climate; absence of dust; well protected.—Mean temperature 61° F. *See* CLIMATE, Treatment of Disease by.

MADNESS.—*See* INSANITY.

MADURA-FOOT.—A synonym for fungus-foot of India. *See* MYCETOMA.

MAGNETISM, ANIMAL.—This name was formerly applied to an imaginary new force or principle, supposed to be akin to magnetism, and to be in operation when individuals were 'mesmerised.' This hypothetical new force was thought

to be called into play by the mesmeriser; and it was deemed to be by virtue of its influence that the will, thoughts, and actions of the 'medium,' or person mesmerised are capable of being modified in the so-called mesmeric trance or sleep. This view as to the nature of the causal conditions is now regarded as altogether erroneous and devoid of all foundation in fact, although certain remarkable effects may unquestionably be produced on many persons (by so called 'mesmeric passes,' by concentration of attention associated with some strain of ocular muscles, by attention to a series of weak monotonous sensations, or other related means), owing to the induction in such persons, under physiological conditions, of some at present imperfectly understood state or modification of cerebral activity (*see* MESMERISM). This state is now generally spoken of as the 'hypnotic condition,' 'hypnotic sleep,' or 'hypnotism'; or more rarely as 'induced somnambulism.' On the other hand, when such a state is induced, as a therapeutic means or agency, it has been spoken of as 'Braidism' (*see* BRAIDISM).

H. CHARLTON BASTIAN.

MALACOSTEON (*μαλακός*, soft; and *ὀστέον*, a bone).—*See* MOLLITIES OSSIUM.

MALAGA, in South of Spain.—Dry, mild, bracing, equable climate. Mean temperature in winter, 55° F. Winds: N.W. (*Terra*), dry and dusty; E. (*Levante*), cold and damp. Drainage and accommodation defective. *See* CLIMATE, Treatment of Disease by.

MALAISE (Fr.).—SYNON. : Indisposition; Ger. *Misbefinden*.—During the onset of acute fevers, in the course of mild digestive disturbances, and in other conditions, the patient complains of a vague ill-definable feeling of illness, accompanied by languor and listlessness. This condition is known as *Malaise*.

MALARIAL DISEASE (Ital. *mala aria*, bad air): comprising MALARIAL FEVER and MALARIAL CACHEXIA.—SYNON. : Malaria; Paludism, Marsh - Fever, Jungle - Fever, Periodic Fever, Paroxysmal Fever.

DEFINITION.—A disease caused by unicellular parasites of the red blood-corpuscles.

VARIETIES: (1) Intermittent Fever or Ague, comprising Quartan Fever and Milder Tertian Fever; and (2) Remittent or Estivo-Autumnal Fever.

SUMMARY.—The malady is characterised at the onset by a specific rhythmical fever; by destruction of red corpuscles; and by deposition of altered hæmoglobin in certain tissues. Later there is a tendency to frequent relapses; and to the establishment of a specific cachexia, of which the principal symptoms are melanosis, anæmia, and enlargement of the spleen.

The parasites are of at least three species, and belong to a group of Protozoa, of which other species are found in many vertebrates. They occupy and destroy the red corpuscles; convert the hæmoglobin into melanin; multiply indefinitely in the blood by simultaneous sporulation, which occurs

at regular periods of one, two, or three days (according to the species); produce rhythmical accessions of fever by the liberation of a toxin at the moment of sporulation; and sometimes continue to infect the patient for years. On ingestion by gnats of the genus *Anopheles*, they undergo further development in the insects; from these their spores may pass through the salivary duct into healthy human beings.

Quinine is a specific remedy at certain stages of the disease.

HISTORY.—The ancients were able to distinguish the rhythmical fevers, and to recognise their various types and their connection with the presence of stagnant water. Early in the eighteenth century Lancisi further emphasised this connection, and appears even to have suspected gnats of being associated with the disease. About the same time the discovery that cinchona bark cures the rhythmical fevers enabled physicians clearly to demonstrate the difference between these and fevers of other types; and in 1753 Torti, assisted by this discovery, first established with accuracy the symptomatology of the malarial fevers. Nearly a century later Meckel laid the foundations of our knowledge of the pathological anatomy by his discovery of minute black granules, called *melanin* or *malarial pigment*, in the blood and spleen—a discovery confirmed later by Virchow, Heschl, and Frerichs. In 1854 Planer suggested, and in 1874 Arnstein showed, that the melanin originates in the blood and is afterwards deposited in the spleen and other tissues; thus reversing the theory formerly held.

In 1880 Laveran recognised that the melanin is produced within unicellular parasites, which occupy the corpuscles and convert their hæmoglobin into this substance; and described all the various forms under which the parasites are found in human beings. In 1885 and subsequently, Golgi established the facts that the organisms multiply by simultaneous sporulation; that the accessions of fever occur at the moment of sporulation; and that different types of fever are associated with different varieties of parasites. These observations were confirmed by Marchiafava and Celli and Canalis, who also made a careful study of the parasite of remittent fever; while a number of other observers repeated and amplified all these discoveries in many parts of the world. The structure of the parasites was carefully studied, and similar organisms were found in various lower animals. Gerhardt (1880), followed by many others, produced infection in healthy persons by injection of blood from diseased subjects; and many observers, especially Laveran, Kelsch and Kiener and Bignami, carefully examined the pathological anatomy.

In 1883 King elaborated a theory that malarial disease is communicated by the bites of gnats. In the following year Laveran briefly enunciated a similar hypothesis. In 1894 Manson propounded the induction that certain forms of the parasites (gametocytes) are destined for further development in gnats; and the gnat-theory was also accepted by Koch, Bignami, and others, on various grounds. In 1897 MacCallum proved the sexual nature of the gametocytes. In 1897 and 1898 the writer, following Laveran's and Manson's inductions, ascertained the life-history of this group of organisms in gnats, found the mode of infection by inoculating a number of healthy birds by the bite of infected gnats, detected two hosts of the parasite

of remittent fever in India, and studied the habits of the insects there. These observations were almost immediately confirmed and amplified by Koch and Daniels. Bignami, Grassi and Bastianelli found the parasites in various species of anopheles in Italy, and claimed to have succeeded in infecting men. Subsequently Koch, Daniels, the writer, and other observers found the alternative hosts, and studied their habits in relation to malaria, and the modes of prevention, in various parts of the world. Daniels and Koch have also noted the great prevalence of the disease among native children in the tropics, and they, Celli, and others have studied the subject of immunity. Manson has infected healthy persons in London by means of gnats brought from Italy, and has proved experimentally that it is possible to remain free from the disease by living in a mosquito-proof dwelling.

EPIDEMIOLOGY.—1. *Distribution.*—Malarial Disease is essentially a malady of warm climates. If it occurs in temperate climates, the season of fresh infections is the summer or the autumn. As a rule its prevalence increases the nearer we approach to the equator. Broadly speaking, it exists in almost all warm countries; though, everywhere, some areas are much more malarious than others, this law often applying even to localities a few miles apart. Tropical Africa, India, the Indian Archipelago, Southern China, Central America, and the shores of the Mediterranean may be cited as being the most generally malarious regions. But the disease exists to a small extent much farther north. For example, it was formerly present in Scotland, and still occurs in Holland. Strict investigation is now showing it to abound among indigenous populations which were formerly thought to be comparatively exempt; and, in fact, the physician may expect to meet it wherever the local conditions are favourable. The total amount of sickness and mortality due to it cannot be exactly computed, but must be enormous.

Malarial Disease is essentially also an *endemic* disease: it adheres to certain localities. At the same time it is apt to increase and diminish both in prevalence and severity within an endemic area; and also to travel slowly beyond endemic areas into others where it was formerly little known.

2. *Conditions.*—Warmth, stagnant surface-water, rank vegetation, poor dwellings, and the proximity of many infected persons, are the conditions under which infection is most likely to occur. From these it follows that level or depressed ground, unabsorbent soil, rains, forests, marshes, and native villages are particularly favourable to the disease. These conditions may be general, as on the coasts of Africa; or local, as in mountain valleys, oases in the desert, or villages in the hollows of open savannahs. In the latter cases, the slopes of the hills, the desert, or the savannahs themselves may be comparatively healthy. On the other hand, cold, dry soil and atmosphere, altitude, steep surface, open country, well-built houses and cities, and a healthy population are opposed to the disease; while well-contained lakes and rivers, and cultivated vegetation are not particularly favourable to it. It must be added, however, that many localities where all malarious conditions seem to exist are singularly free from the disease.

3. *Mode of dissemination.*—Conclusive microscopical and experimental researches demonstrate that the parasites complete the cycle of their

existence in gnats of the genus *Anopheles*, which carry them from diseased to healthy persons; and that the habits of the insects—their prevalence in warm countries, their tendency to breed in surface-pools of water, and their predilection for rank vegetation—go far to explain the phenomena which were formerly attributed (without any strict, much less experimental, evidence) to a telluric influence.

Gnats, or as they are called in the tropics *mosquitoes*, belong to a family of dipterous insects named Culicidæ—a family which contains about 12 genera and 250 species. The *larvæ* and *pupæ* are aquatic. The adults possess a suctorial proboscis and scaled wings. The commonest genera are *Culex* and *Anopheles*. In the former the female has short palpi, in the latter long palpi. According to many observers there are other differences between these genera, which, though they may not always hold good, are generally very useful to the observer. Thus the *larvæ* of *Culex* have a long breathing-tube at the tail, by which they hang suspended, head downward, from the surface of the water; while those of *Anopheles* possess no elongated breathing-tube and float flat on the surface. The former *larvæ* may be found in almost any stagnant water in tubs, pots, cisterns, drains, &c.; while those of *Anopheles* prefer pools on the ground, hollows in rocks, sluggish runnels of water, and sometimes even ponds, tanks, rice-fields, and the margins of streams. The adults of *Culex* generally have a thin proboscis and a clumsy thorax, and rest on a wall with the tail almost touching it; the adults of *Anopheles* have a proboscis which appears thicker, and a straight elegant body, and rest with the tail pointed away from the wall at a varying angle. *Anopheles*, moreover, generally possess several dark marks on the anterior margin of the wings, while the wings of *Culex* are more frequently plain. The males of both genera can be distinguished by their feathered antennæ. As a rule, only the females feed on blood. Both sexes also feed on the juices of fruit. The insects may live for months, haunting houses close to the breeding-pools, or biting cattle and birds, and laying their eggs every few days. They hibernate in temperate climates.

In malarious localities a considerable percentage of *Anopheles* caught in the dark corners of infected houses are found to contain the parasites. The insects seem to become infected, as a rule, from native children, of whom, according to the observations of Daniels, Koch, and others, sometimes over 50 per cent. may harbour parasites. Stephens and Christophers observe that *Anopheles* congregate round persons camping in bush far from human habitations, and that many even of such gnats have blasts in their salivary glands. In short, all recent investigations demonstrate how easily infection must take place in localities where both *Anopheles* and cases of malaria abound.

It has not yet been absolutely demonstrated (*a*) that other kinds of gnats besides *Anopheles* do not carry the human parasites; (*b*) that the insects do not acquire the parasites from each other, or from other animals as well as from man; or (*c*) that there may not be some other route for infection. Up to the present, however, no facts which will justify any of these hypotheses have been established, and the simple observation that *Anopheles* carry the parasites from diseased to healthy persons appears to be quite sufficient to account for the dissemination

of the disease. At all events, this seems certainly to be by far the commonest way by which infection is produced.

PARASITOLOGY.—The parasites of malaria belong to a group called the Hæmamebiidæ. This group is generally placed among the Sporozoa, an order of the Protozoa or Unicellular animals. The Hæmamebiidæ contain at least nine species, of which three are found in man, one in monkeys, three in bats, and two in birds. The writer divides the group into two genera, one of which includes the quartan and tertian parasites of man and all the parasites of birds, bats, and monkeys; while the other contains the æstivo-autumnal parasite. The life-history of the human parasites is as follows:—

The youngest organisms are found as small amœboid bodies in or on the red blood-corpuscles. Each consists of a small nucleus (chromatosome) and surrounding bioplasm. The *amœbule* grow in size and convert the hæmoglobin of the containing corpuscle into melanin. They reach maturity in one, two, or three days, according to the species; and then become either (*a*) sporocytes, or (*b*) gametocytes. Among those destined to be sporocytes, the nucleus divides into a variable number of segments—each segment collecting some of the bioplasm round it and becoming a spore. When finally mature, the organism ruptures the cellular host and scatters its spores, together with a small residual mass containing its melanin, into the liquor sanguinis. The spores attack fresh corpuscles, and the melanin is engulfed by the phagocytes of the host. This constitutes the *asexual* or *fever-producing* cycle of the parasites.

The amœbulæ which develop into *gametocytes* show no division of the nuclei within man. These bodies are, in fact, sexual forms, male and female, and belong to the *sexual* or *mosquito-cycle* of the parasites. They circulate unchanged in the blood, awaiting ingestion by the appropriate second host, *Anopheles*. As soon as they are drawn into the stomach-cavity of this insect they undertake their sexual functions. They burst from the enclosing corpuscle and swell slightly. The male gametocyte now emits four or more active motile filaments, called *microgametes* and formerly called *flagella*. These are, in fact, spermatozoa. They are generally emitted from 10 to 30 minutes after the blood was drawn by the insect (they are also often emitted in an ordinary fresh microscopical specimen). After struggling for a few minutes, one or more of them break away from the parent male cell, and, after wandering about in the liquor sanguinis, enters one of the female gametocytes, fertilising it. This process has been actually witnessed by MacCallum. The fertilised female gametocyte is called a *zygote*. After a time it becomes elongated and motile, and commences to work its way through the mass of blood in the insect's stomach. Arrived at the parietes, it passes through them and fixes itself on the outer surface of the stomach. Here it commences to grow rapidly, still, however, containing its original granules of melanin. The nucleus undergoes numerous subdivisions. After at least a week, the zygote, still fixed motionless in this position, is mature. It is then found to consist of a capsule full of many hundreds of filamentous spindle-shaped bodies called *blasts* or (incorrectly) *sporozoites*. The capsule now ruptures, pouring the blasts into the body-cavity of the insect. The

blasts are carried all through the tissues by the circulation, until they arrive at the *salivary gland*, when they penetrate into the *salivary cells*. From these they pass into the *salivary duct*. This duct practically opens at the extremity of the central stylet or lancet of the mosquito's proboscis—the salivary secretion being destined to pass into the wound made by the puncture. With this secretion the blasts enter into the circulation of a fresh host, and set up a malarial infection in him, as proved by numerous experiments made by the writer, Daniels, and Koch, on birds, and by Bignami, Bastianelli, and Manson on men.

The three human species all possess this life-history, but differ in minor particulars, which enable us to distinguish them with ease.

The *Quartan Parasite* (*Hæmameba* or *Plasmodium malarie*—figs. 1-7) occupies a small or medium-sized corpuscle. Its amœboid movements are rather slow. Its melanin consists of rather coarse dark-brown granules. It produces about 8 spores. Its life-cycle lasts 72 hours. All stages can be found in the finger-blood. The gametocytes are shaped roughly like the sporocytes before the spores are formed.

The *Tertian Parasite* (*Hæmameba* or *Plasmodium vivax*—figs. 8-13) occupies a large, sometimes pale, corpuscle. Its amœboid movements are very active, the half-grown amœba being of a ragged shape, and often looking like several separate amœbæ. Its melanin consists of very fine granules, which are generally of a light-brown colour. It produces about 15-20 or more spores. Its life-cycle lasts 48 hours. All stages can be found in the finger-blood. The gametocytes are shaped like the sporocytes, but have a coarser pigment.

The *Æstivo-Autumnal Parasite* (*Laverania* or *Plasmodium præcox*—figs. 14-48) occupies a medium-sized corpuscle. Its amœboid movements are very active when it is young. The smallest forms often contain no melanin at all, but frequently take the appearance of a little ring inside the corpuscle. Later, the amœbæ form two or three masses of black pigment and retire to the internal organs. The sporocytes (which are seldom found in the peripheral blood) are rather small and produce numerous small spores. The life-cycle lasts 24 hours in the æstivo-autumnal quotidian parasite, and 48 hours in the tertian variety. Only the youngest amœbæ and the gametocytes are found in the finger-blood. The gametocytes have a special shape—that of a crescent with a mass of black pigment-granules in the centre. In the male, the granules lie separate; in the female they lie close together, often in the form of a ring. Shortly after the blood is drawn many of the crescents become oval and then spherical, and some produce flagella (spermatozoa) in from 10-30 minutes.

It should be noted that in all the species the gametocytes are not invariably present. In the quartan and tertian species they generally appear during the paroxysm, remain for a day or two, and then vanish. In the æstivo-autumnal they do not appear until the first series of paroxysms has lasted about one week; but may then remain for several weeks. In old cases of æstivo-autumnal fever, however, or in cinchonised patients, they may not be produced at all; though when they are once produced, quinine does not much affect them.

The development of all the species in mosquitoes is practically the same (figs. 35-48); but the young

tertian zygotes can generally be distinguished by their fine, light-brown pigment.

MODE OF INFECTION.—Infection is caused by the bites of gnats which contain blasts; these gnats have hitherto been found to belong exclusively to the genus *Anopheles*. Bignami states that the bite of a single infected gnat can infect man. Judging from dissections of the insects it is probable that a few thousands of the blasts suffice to produce this effect.

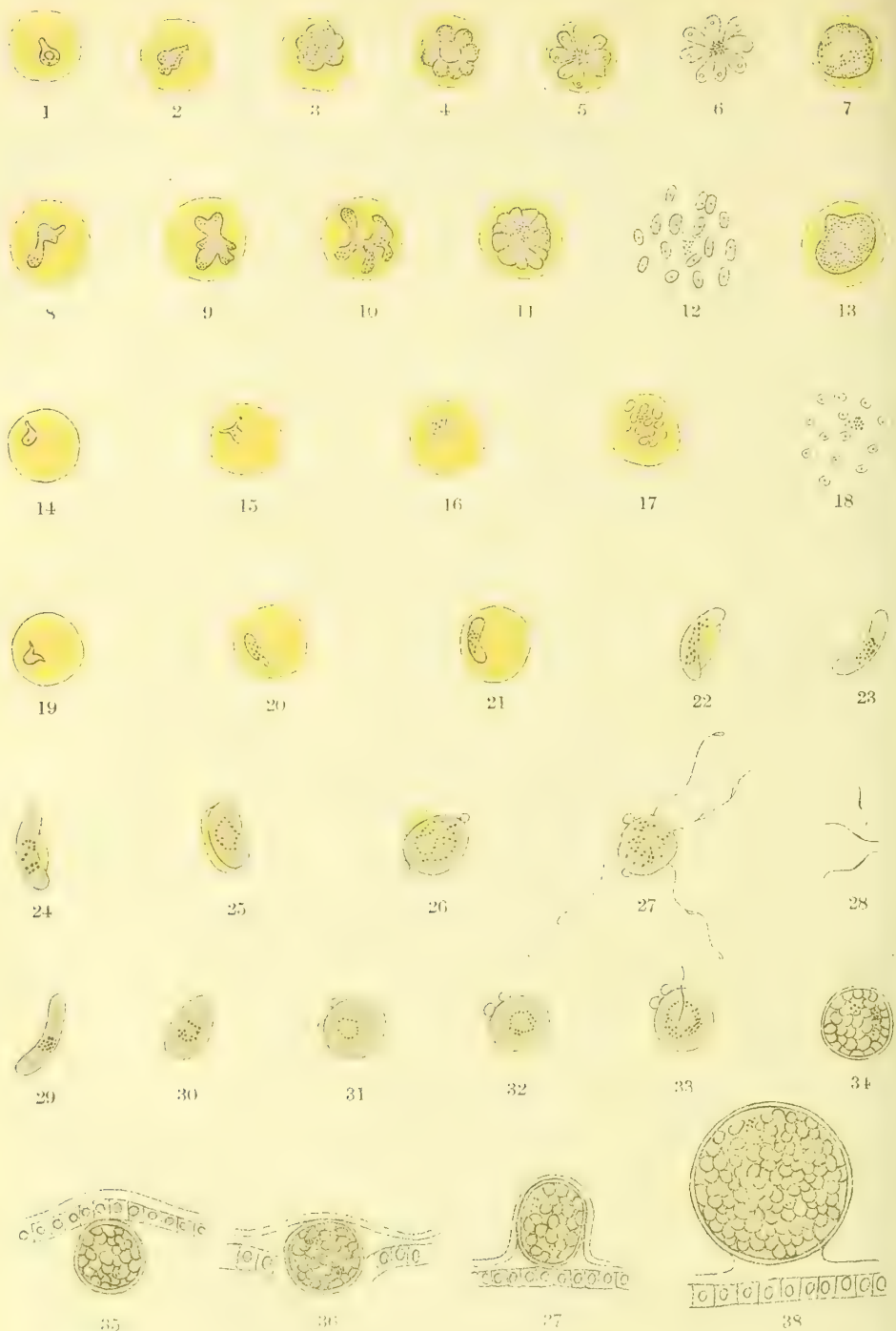
The question whether there may not be another route for infection has not yet been definitely settled; but, at all events, no other has actually been found.

NUMBER OF PARASITES REQUIRED TO PRODUCE FEVER.—It is a general experience that when fever is present the parasites can be found in the blood by a skilled observer. They may be extremely numerous: it is certainly not uncommon to find as many as one parasite to every fifty corpuscles. We may roughly estimate that, when fever is present, at least one corpuscle in 100,000 contains a parasite. Physiologists calculate that in the whole body of a healthy man of 150 lbs. weight there are about 25 billion red corpuscles. If one in 100,000 of these contains a parasite, there must evidently be about 250 million parasites in the whole body. This then must be approximately the least number of organisms required to produce fever.

In this estimate the gametocytes, which do not cause fever, need not be considered.

PATHOGENESIS.—To explain (a) the fever, and (b) the excessive destruction of corpuscles, which follow the sporulation of a horde of parasites, some observers have supposed that the newly produced spores irritate the thermic centres. Against this view may be set the fact that a large dose of quinine given before the paroxysm will destroy vast numbers of spores (as proved by subsequent microscopical examination), but may not modify the impending fever in the smallest degree—though it may entirely avert the *next* attack. Again, the excessive destruction of corpuscles seems to exclude the idea that the only action of the parasites on the system is a mechanical action. The theory of a pyrogenic and hæmolytic toxin (which may just possibly be connected with the freshly liberated melanin) appears to be the only theory capable of explaining the facts of the case. At the same time it must be understood that no toxin has as yet been actually isolated in malarial blood.

PATHOLOGICAL ANATOMY.—The pathological anatomy of the paroxysm depends upon (1) the deposition of melanin in certain tissues; (2) the deposition of the detritus of red corpuscles and of hæmoglobin; and (3) the congregation of æstivo-autumnal parasites in certain sites. As the paroxysms succeed each other, the melanin not only continues to be deposited but passes out of the capillaries and tends to be eliminated by the connective tissue and lymphatics. At the same time another pigment, the *yellow pigment*, is precipitated in the liver, spleen, bone-marrow, and kidneys; and the precipitation of this pigment continues after the subsidence of the parasitic invasion, and lasts during the cachectic anæmia which follows. It consists of granules of a pale or of a bright yellow colour, very small, or up to about 6μ in diameter, and of a square or angular shape. In the liver it occurs in the hepatic cells, often round the central vein, whereas the black pigment is found only in the connective tissue. In the spleen it is found in cells of the pulp; in the

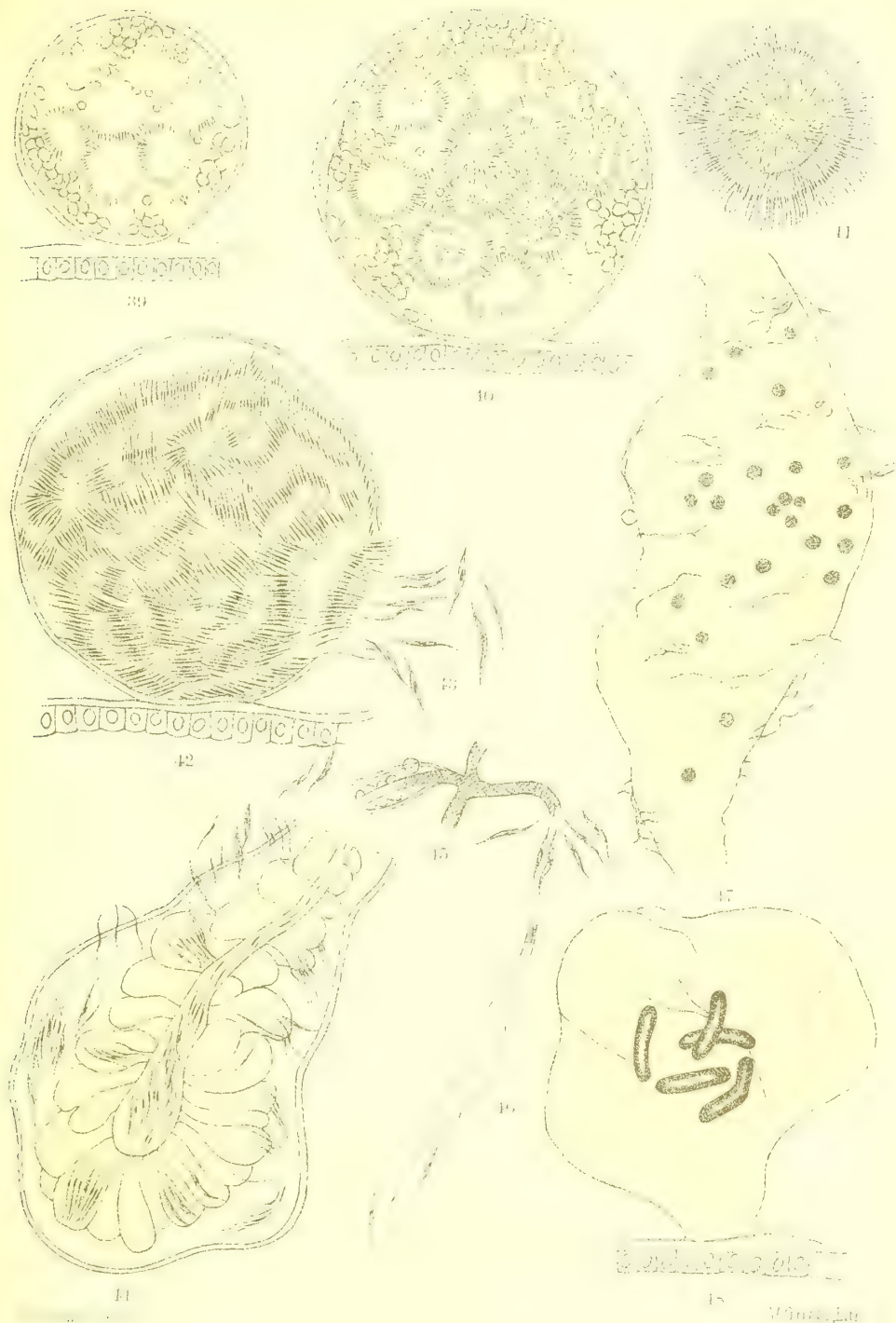


Figs. 1-7, Quartan Parasites; 1-3, *amœbulæ*; 4-6, *sporocytes*; 7, *gametocyte*.

.. 8-13, Tertian Parasite; 8-10, *amœbulæ*; 11, *sporocyte*; 12, *free spores*; 13, *gametocyte*.

.. 14-23, Estivo-Autumnal Parasite in Man; 14-17, *development of sporocyte*; 18, *free spores*; 19-21, *development of gametocyte (crescent)*; 22, *male crescent*; 23, *female crescent*.

.. 24-34, Development of Estivo-Autumnal gametocytes (crescents) in stomach-cavity of *Anopheles*; 24-26, *changes in male crescent*; 27, *escape of spermatozoa (flagellate body)*; 28, *free spermatozoa*; 29-30, *changes in female crescent*; 33, *fertilisation of female crescent by spermatozoon*; 34, *zygote*.



Figs. 35-48. *Estivo-Autumnal Parasite* in tissues of *Anopheles*; 35-37, *zygote perforating stomach-wall*; 38, *zygote attached to outer wall of stomach and growing in size*; 39, 40, *zygotes dividing into meres*; 41, *single mere consisting of blastophore carrying blasts*; 42, *mature zygote full of blasts*; 43, *free blasts*; 44, *blasts in salivary gland*; 45, *blasts in salivary duct*; 46, *blasts issuing from point of stylet*; 47, *stomach studded with mature zygotes*; 48, *capsule of zygote with five "black spores."*

kidneys in the epithelium of the tubules. It blackens with age (Bignami). Though both melanin and the yellow pigment contain iron, only the latter gives the iron-reactions with potassium ferrocyanide and hydrochloric acid, or with ammonium sulphide (p. 57). The black pigment is soluble in alkalis (e.g. ammonium sulphide), but insoluble in acids; the yellow pigment in both. Further study on all these points is required. The yellow pigment is found also in essential anæmias and even in ankylostomiasis (Daniels), and is reasonably thought to be derived from the detritus or dissolved hæmoglobin of the red corpuscles. It is probably eliminated in the bile.

Macroscopically, we have during the paroxysms pigmentation and sometimes hyperæmia of the spleen, liver, brain and meninges, marrow, and kidneys. The liver is reddish brown, enlarged and softened; the spleen enlarged, softened, and almost black. In chronic disease, the spleen may be much enlarged; its colour is variable, depending upon the degree of melanosis; adhesions are generally present. The liver also may be much enlarged, with adhesions; the colour depends upon the relative amounts of the two pigments; the lobules are marked by melanosis of their periphery. As true cachexia sets in, melanosis vanishes, leaving the previous hyperplasias. Finally atrophy and amyoid degeneration are recorded.

CLINICAL PHENOMENA.—1. *Incubation-Period.* While it is certain from experiment that the malarial parasites cause infection after entry into the human circulation, the immediate changes which they undergo at that moment have not been ascertained. It may reasonably be held, however, that they shortly enter the corpuscles and become amœbulae; and that these amœbulae next commence to reproduce themselves in the ordinary manner—gradually becoming more and more numerous until, after the lapse of a definite interval, they become numerous enough to cause illness.

The actual incubation-period has been determined (a) by infection-experiments with gnats; (b) by similar experiments with inoculation of malarial blood; and (c) by observation of persons who have been taken ill some time after spending a single night in a malarious locality. The period may be taken as being from about six to twenty days.

There are cases in which the progress of the invasion appears to be checked for a long time after infection, probably by the use of quinine as a prophylactic, or by a cool external temperature. In such cases the fever may suddenly burst forth when the patient abandons the quinine, or enters a hot climate. Instances of fever occurring a few hours after infection are apocryphal.

2. *Prodromata.*—When the number of parasites mounts up toward the number requisite to cause fever, certain premonitory symptoms may be observed. These are, briefly, aching in the bones; dull pains in the head, liver, and spleen; chilliness; and often slight rises of temperature for a day or two before the typical paroxysm occurs.

3. *The Malarial Paroxysm.*—When the parasites have multiplied up to or beyond the fever-point the catastrophe occurs. At this point the organisms, occupying the corpuscles in vast numbers, have simultaneously grown to their full dimensions, and have divided each into a number of spores. Next, almost simultaneously, the containing corpuscles of

all the parasites are ruptured; the spores, the melanin, and the toxin escape into the liquor sanguinis; and the paroxysm or ague-fit commences. This consists of the three well-known stages—the cold stage, the hot stage, and the sweating stage.

Cold stage or rigor.—The patient experiences a curious sensation of chilliness. The fugitive pains in the body increase. A sudden nausea often sets in. The chilliness rapidly augments; and if the patient rises, or if a breeze blows upon him, he begins to shiver, and then to shake all over. He is obliged to fling himself on his bed, and in the hottest weather calls for bed-clothes to be heaped over him. His teeth chatter, he lies huddled up, his lips and nails are blue, his countenance is anxious, and frequently he vomits several times. If we take his temperature, however, we find that, far from being below normal, it is already actually some degrees above it. The pulse is small and a little quicker, the respiration hurried; the urea, chlorides, and total amount of urine increased; phosphates diminished; pupils dilated; and headache, horripilation, and vertigo often present. Thayer found this stage in 97 per cent. out of 332 cases. It is more pronounced in the quartan and tertian varieties; less marked and often entirely absent in the æstivo-autumnal. The stage lasts from a few minutes to about an hour.

Hot stage.—The temperature of the blood continues to rise, and may reach its maximum about two hours after the onset of the ague-fit. As it rises, the rigor gives place to flushes of heat, until the patient now feels as hot as he formerly felt cold. Headache, vomiting, vertigo increase; the skin is flushed, the conjunctivæ injected; the pulse rapid and full; pains in the bones, cough, urticaria, jactitation, epistaxis, diarrhoea, and even delirium or drowsiness, and coma may be present. The urine becomes more scanty and high-coloured, and frequently contains albumen. The temperature often reaches 106° F. or more. Enlargement of the spleen is demonstrable in a large percentage of even recent infections; and anæmia can be already detected on counting the corpuscles. This stage is almost invariably present. It may last from one to many hours. It is much longer in the æstivo-autumnal infection than in quartan and tertian.

Sweating stage.—Beads of perspiration now begin to form on the lips, forehead, and breast. The temperature falls rapidly. The pulse becomes slower. Profuse sweating, with relief from most of the symptoms, sets in. Phosphates in the urine are augmented, the excretion of urea still remaining above the normal. The temperature continues to fall until it often goes several degrees below normal. The sweating then ceases, and the enlargement of the spleen rapidly diminishes; but more or less anæmia remains. The length of this stage may be put down at two to four hours.

The patient now feels almost recovered. The paroxysm has ceased, and the *intermission* begins.

The parasites.—If the blood be examined every hour during the ague-fit the following changes in the parasites will be observed. At the beginning of the rigor a number of full-grown organisms, each practically consisting of a cluster of spores, can be seen; and also a few very young amœbulae attached to the corpuscles, and apparently recently escaped from some of the full-grown parasites just referred to. As the paroxysm advances the mature parasites rapidly diminish in number as they break up into

free spores; while at the same time the amœbulæ become more numerous. Finally, about the height of the fever, no more mature parasites are found—all of them having broken up by this time; but the blood now contains numerous amœbulæ, each attached to or ensconced within its cellular host. During the sweating stage and all through the following intermission, the organisms grow in size, until they in their turn reach maturity and occasion a fresh paroxysm.

With the quartan and tertian parasites, all these changes can easily be followed by examining blood taken from the skin at intervals; but with the æstivo-autumnal species this is not possible, because when the amœbulæ reach a certain stage of growth they vanish from the peripheral circulation, so that the sporocytes can only be found in blood taken from the inner organs, e.g. by acupuncture of the spleen.

Besides the sporocytes and amœbulæ, we often find gametocytes—sometimes in very large numbers. These, having no marked pathological significance, do not concern us at present.

Destruction of parasites.—As each sporocyte produces from 8 to 30 spores, according to the species, the total number of the invading organisms would increase indefinitely were it not for some resistant principle in the host. We are forced to conclude that some such principle exists, but we do not yet fully understand the mechanism. If a patient be kept at rest in bed the phenomenon of *spontaneous recuperation* often occurs; the parasites rapidly decrease in numbers, and an expected paroxysm fails to appear. Phagocytosis does not suffice to explain such cases, and it is more likely that the blood fabricates some chemical agent inimical to the invaders. Quinine, if given in sufficient doses, has the same effect. The observer must never imagine, however, because the parasites have become too few for the moment to be readily found, that they have disappeared altogether.

Disposal of melanin.—The residual masses of melanin left after the dispersion of the spores are quickly taken up by (a) the wandering phagocytes of the blood, and (b) the fixed phagocytes of the vascular endothelium. The former are principally the large mononuclear leucocytes, and, to a less degree, the polynuclear neutrophils. The fixed phagocytes are endothelial cells of the spleen, cerebrum, liver, and bone-marrow. Thus every paroxysm is followed by the appearance of melanin in a vast number of cells, especially in the spleen and the splenic vein. Pigmented leucocytes begin to appear in the peripheral blood shortly after the commencement of the paroxysm.

Leucocytes during the paroxysm.—These are diminished in number in ordinary attacks. In pernicious attacks, however, they may be largely increased in number—though this is now generally attributed to some complication. *Relatively*, the mononuclear cells are increased, and the other varieties diminished, at least after the crisis.

The red corpuscles.—A certain number of these are destroyed by the breaking up of the sporocytes which they contain; but counts made before and after the paroxysm show that a large number must be destroyed by some other agency in addition. It is therefore supposed that the toxin liberated by the sporocytes is hæmolytic as well as pyrogenic. During severe paroxysms one or two million corpuscles, or even more, may disappear in every

cubic millimetre of blood. This hæmolysis is more marked in earlier than in later attacks.

Not only are the corpuscles reduced in number, but they also often lose much of their hæmoglobin. The total amount of hæmoglobin may be reduced by 50 per cent. after several paroxysms, and reparation is slow.

Other blood-changes.—The density of the blood decreases with the decrease in the number of corpuscles (Viola). The total quantity of blood is diminished (Kelsch). The isotonicity of the corpuscles is increased owing to the destruction of the less resistant cells (Viola).

The urine.—The amount of urine excreted has not been sufficiently investigated to be considered here in detail. The specific gravity tends to be increased. The colour is usually dark, especially during the fever. The phosphates decrease during the fever and increase during defervescence and during the intermission. Potassium is in excess of sodium; both are more abundantly excreted during the paroxysm than after it. The chlorides are in excess during the first stage of the paroxysm, and diminish at the crisis and afterwards. Iron is considerably increased, especially after the attack. The amount of urea is increased; it is greatest during the first stage, and least after defervescence. The uric acid seems to be unaffected. Albuminuria, hæmaturia, and glycosuria occur in some cases. Experiments on the toxicity are not conclusive.

Other facts.—The tongue is generally small, red, and clean during the first paroxysm; later it may become coated, especially in severe cases. The bowels tend to be constipated. A marked increase in the area of splenic dullness can generally be detected, even after the first paroxysm. Splenic, hepatic, or epigastric tenderness is often present. Marked pallor, indicative of the rapid hæmolysis, frequently occurs after the attack.

Modifications in the paroxysm.—The paroxysm as described above is subject to modifications which may be divided for clinical convenience into (a) normal, and (b) abnormal modifications. The most important of the first are the modifications due to the species of parasite concerned.

The quartan paroxysm.—This lasts about 9 hours. The premonitory symptoms are generally slight. The rigor is usually severe and distressing, and is seldom absent. The fever is sharp, rising to 105° or 106° F., or even more. The sweating is profuse and gives rapid relief.

The tertian paroxysm lasts about 11 hours, and is very like the quartan. The rigor is perhaps less severe, the fever more so, secondary symptoms such as vomiting, headache, and pains in the bones being perhaps more marked.

The æstivo-autumnal quotidian paroxysm is generally short, lasting 6 to 12 hours (Marchiafava and Bignami). Rigor is very often slight or absent; but the secondary symptoms are apt to be very severe.

The æstivo-autumnal tertian paroxysm is the gravest and longest of all. It lasts from 24 to 40 hours (Marchiafava and Bignami). The temperature-curve differs much from that of the other varieties. There is a rapid and high rise, followed by sustained fever, and then by a temporary fall with some amelioration of the symptoms—called the *pseudo-crisis*. But the fever now increases again, rising still higher than before; and, lastly, the true crisis sets in. The secondary symptoms are most severe

in this variety; pernicious attacks most common; and, perhaps, rigor most often absent.

Other normal modifications.—It must always be remembered that the paroxysm is likely to be most prolonged and severe in *earlier attacks*, to whatever species of parasite they are due. In such cases the *prolongation* is often caused by the fact that as a rule in recent infections the parasites do not sporulate so simultaneously as they do later. At the same time the severity of the symptoms is greater, probably because the patient, who is as yet unaccustomed to the disease, is more sensitive to the intoxication. Hence it often happens, even in certain cases of tertian or quartan, that the paroxysm is so prolonged that before it is well over the succeeding paroxysm has already commenced, giving rise to a *remittent* or *subintant* fever.

On the other hand, in old infections just the opposite state of things prevails: the parasites tend to sporulate smartly together; and the system, better inured to the toxin, suffers less severely from its effects. This is frequently seen in natives of malarious districts, especially in native children. In such the paroxysm is often scarcely apparent: the adult lies down for half an hour, and the child continues to play undisturbed.

Other points to be remembered are that the severity of the paroxysm depends *ceteris paribus* (a) on the number of sporocytes present, and (b) on the species—the æstivo-autumnal being the most virulent.

4. *Pernicious Paroxysms.*—In addition to the variations of the paroxysm just given, which may be looked upon as being more or less normal incidents of the disease, certain exceptional forms, of great importance to the physician, are sometimes met with. These forms, which show considerable clinical variety, are collectively known as *pernicious attacks*. As their name implies, they are dangerous to the patient; but, fortunately, they are comparatively rare. They occur chiefly in very malarious localities and seasons; and among those who have already suffered from a few attacks but have not yet become habituated to the poison, especially among those who have been exposed to hardships and have not been adequately treated. As a rule, pernicious attacks are associated with very large numbers of parasites—not necessarily found in the peripheral blood, but present in the body as a whole, or in certain organs.

A remarkable fact about such paroxysms is that they are found almost entirely in connection with the æstivo-autumnal parasites, especially the tertian variety. As already mentioned, these parasites, as soon as they grow to any considerable size, tend to disappear from the peripheral circulation and to congregate in the spleen, liver, bone-marrow, brain, or digestive tract (this statement does not include the gametocytes of this species). Hence, if the total number of organisms is very large, we are apt to find, in addition to the ordinary symptoms of the paroxysm, certain grave symptoms referable to the organ or organs most attacked. Such cases are also known as *comitate* or *complicated attacks*.

Simple or Hyperpyrexial Pernicious Paroxysm.—This name should be reserved for cases in which the symptoms are referable simply to superintoxication without implication of any special organ. Death is threatened or occurs as the result of hyperpyrexia, accompanied by the usual symptoms.

Cerebral Pernicious Attacks.—These are referable to the accumulation of immense numbers of æstivo-autumnal parasites in the cerebral and meningeal capillaries. Sections often show the vessels crowded with mature sporocytes, leading to stasis and hyperæmia. The principal clinical forms produced are as follows. The *comatose* form; headache and stupor followed by coma; pupils contracted; respiration slow—occasionally Cheyne-Stokes breathing; sometimes spasms; reactions often entirely abolished. Death is frequent, and the cases are very apt to be mistaken for sunstroke. Fever is not necessarily high. Other forms are indicated by their names—*ataxic*, *tetanic*, and *delirious* forms. *Fits* are common in children during the paroxysm. *Bulbar paresis*, *hemiplegia*, *paraplegia*, and *amblyopia*, are all known to occur, but if death does not intervene, all are completely or nearly transient. Cerebral and retinal *punctiform hæmorrhages* are not uncommon. *Meningeal* and *choreal* types are known. In all a fatal result may occur as a result of secondary lesions, some days after the parasites have been reduced by quinine.

Gastro-intestinal type.—The capillaries of the digestive tract are often the seat of a parasitic stasis, leading to symptoms referable to this tract. The most important form is the *choleraic*, which closely simulates cholera. The stools, however, do not become entirely choleraic, though death may occur. *Dysenteric*, *diarrheal*, and *melanic* attacks are also known; but the observer must be on his guard against mere concurrent affections in such cases.

Bilious type.—In this there is polycholia of a hæmatogenous order, derived from excess of hæmoglobin thrown into the liquor sanguinis during the paroxysm. These cases are characterised by jaundice, dark stools and urine, vomiting, high fever, rapid blood-destruction, death under coma, or slow recovery.

Algid type.—Though numerous parasites and all the other symptoms of the paroxysm are present the fever may be almost entirely absent—and this not as a result of habitude. The pulse may be almost lost, and collapse follows. The pathogeny is not known.

Other types are the *sudorific* type, in which the sweating stage is followed by collapse; the *syncopal* type, in which fainting occurs; the *gastralgic* type, associated with sharp epigastric pain and vomiting; the *hæmorrhagic* type, with epistaxis, hæmoptysis, &c.; lastly, the *pneumonic* type, simulating pneumonia but not consisting of a concurrent pneumonic attack; there is painful cough and bloody sputum, with a moderately dull note over the lung.

Post-malarial fever.—This is a name given to a continued fever, which is not directly due to the parasites, but which sometimes sets in after a grave malarial paroxysm. It is most probably produced by secondary lesions caused by the organisms during the paroxysm. It is not amenable to quinine; and may last many days. It is remarkable that during its course the number of corpuscles may continue to fall, and even death may occur, though for many days the parasites may have disappeared as the result of previous cinchonisation. Needless to say, the observer must show reserve in giving this name to cases in which the organisms are not found at once.

5. *Procession of Paroxysms.*—We have confined our attention hitherto to the *single* paroxysm and its

modifications, because it constitutes, so to speak, the *pathological unit* of an acute malarial infection. The sequence of events consists of a series of these units disposed in a more or less regular order. The paroxysm tends to recur every one, two, or three days, according to the species of the pathogenic organism. But at the same time this type is apt to be modified by several circumstances which it is necessary now to examine.

Single Infections.—A single infection is one in which each paroxysm is due to the progeny of the parasites which caused the previous paroxysm. Thus in a *single quartan* infection, if a first generation of parasites caused fever on the first day of a month, the next paroxysm would be caused by their progeny on the fourth day of the month; the third paroxysm by the third generation on the seventh day; and so on—there being no paroxysms between these dates. Similarly in a *single tertian* infection (whether the parasites are the ordinary or the *æstivo-autumnal tertian* parasites) the paroxysms occur every other day. Lastly, in a *single quotidian* infection, one paroxysm occurs every day.

Double Infections.—Here we have two series of parasites of the same species sporulating at different times. Thus in a *double quartan* infection, while one series causes paroxysms on the first, fourth, and seventh of a month, another series may be causing paroxysms on the second, fifth, and eighth days, the third, sixth, and ninth days being fever-free. Or perhaps the second generation may sporulate on the third, sixth, and ninth days, the second, fifth, and eighth days now being fever-free. Similarly a *double tertian* shows paroxysms not only on the first, third, and fifth days, but also on the second, fourth, and sixth days; while a *double quotidian* may have one series of paroxysms every morning, and another every evening.

Lastly a *triple infection* is one in which there are three series of paroxysms due to the same species of parasite. Thus in a *triple quartan* the patient has one series of attacks on the first, fourth, and seventh days; a second series on the second, fifth, and eighth days; and a third on the third, sixth, and ninth days.

Hence it is apparent that a quotidian fever is not necessarily always caused by the quotidian parasite. It may also be due to a double tertian or a triple quartan infection. It is remarkable, however, that there is often a notable difference between quotidian fevers due to the different species, because, with the quartan and tertian parasites one series of paroxysms is *stronger* than another; so that the type is still visible on the temperature-chart by a higher fever on every other day with the tertian parasite, and on every third day with the quartan parasite.

Remittent, Subintrans, or Subcontinuous Fevers. Other circumstances which influence the fever curve are the normal modifications to which the paroxysm is subject. The most important of these is the prolongation of the paroxysm due (a) to infection by the *æstivo-autumnal tertian* parasites, or (b) to sporulation not being simultaneous, or (c) to want of habituation in the patient. In such cases the paroxysms are apt to run into each other; a second begins before the first has ceased; and we obtain what is called a malarial remittent fever.

This point should be carefully noted. A remittent fever is not fundamentally different from an intermittent fever; it is merely due to the coales-

cence of attacks, with absence of the usual intermission. It is always apt to occur during the first days of fever due to any species of parasite. It is, however, especially liable to occur in the *æstivo-autumnal tertian* infection, because of the ordinary length of the paroxysm in such cases; and also in the *æstivo-autumnal quotidian*, because of the short life-cycle of this parasite and its tendency to sporulate irregularly. Thus remittent fever is generally, though not always, associated with the *æstivo-autumnal* parasites.

Another source of variation in the chart is the *anticipation or retardation* of sporulation which sometimes occurs.

Lastly, in grave or complicated cases, post-malarial or other secondary fevers obscure the periodicity of the attacks. In all these cases, however, the observer should remember that the beginning of a fresh paroxysm is generally marked clinically by an access of fever, often accompanied by chills or rigors, and microscopically by the appearance of young parasites in the corpuscles.

6. *Rallies and Relapses.*—After a few weeks of fever, even without treatment, a *rally* usually occurs. The fever, perhaps at first remittent, becomes intermittent; then, in complex infections, first one and then another series of paroxysms cease, the chart being modified accordingly. Reparation of the blood takes place; the fever-producing forms of the parasite disappear; the temperature stands below normal; and the patient thinks himself recovered and resumes his avocations. This improvement may last for days, weeks, or months; but if treatment has not been continued, and especially if the climate is tropical, a relapse is almost sure to happen. All the old symptoms recur, and the parasites are found again. After this relapse has lasted some time, there is a second rally, followed perhaps by a second relapse; and rallies and relapses alternate, sometimes during years. On the other hand, permanent recovery, either spontaneous or the result of treatment, or death may intervene at any moment.

Examination of the blood generally shows that the fever-producing forms of the parasites are absent during the rallies and present during the relapses; but the gametocytes, especially of the *æstivo-autumnal* parasites (called *creascent*s) may be found also during the rallies. It has been suggested that the organisms enter upon some latent phase during the rallies; but the simplest theory (and one which is supported by a study of the parasites of birds) is that they only diminish so much in number as to become at once insufficient to cause fever and difficult to find in the small quantities of blood usually examined. We can readily understand how even a few scores of parasites may keep the infection alive for years without being numerous enough to produce any symptoms whatever.

The relapses are precipitated by exposure, fatigue, chills, indigestion, and intercurrent affections, by anything, in short, which tends to reduce the vitality of the host. Nothing is commoner in the tropics than to see bands of native workmen or soldiers attacked by epidemics of fever immediately after commencing some severe labour. Such cases are often only relapses among persons already infected. Owing to the same facts even intelligent persons often ascribe their fever to chills.

In malarious localities many apparent relapses may really be due to reinfection; and our present knowledge shows that, if *Anopheles* be present, a

patient may become reinfected even from himself. On the other hand, true relapses certainly occur in persons long removed from malarious areas. Such cases are found with all the species of parasites: the quartan is especially obstinate.

7. *Chronic Malaria*.—The condition just described is known as chronic malarial disease; and consists of a continued infection in which the parasites occasionally become numerous enough to produce fever, together with the development of certain important secondary symptoms. As the disease progresses, three series of phenomena manifest themselves: (*a*) the parasites tend to become less and less numerous; (*b*) their toxin produces less and less effect; and (*c*) the accumulated effect on the system of successive paroxysms becomes more and more marked—producing the secondary symptoms.

These consist principally of *anæmia*, *melanosis*, *siderosis*, enlargement of the spleen and liver, and perhaps a secondary fever. The *anæmia* is due partly to the blood-destruction which takes place in the paroxysm, and in part probably to the state of the organs. During the earlier paroxysms, each attack is followed by a sensible reduction of corpuscles; but this reduction tends to become less and less with each relapse; and finally a constant oligocythæmia, standing at 1,000,000 corpuscles per c.mm. or even less, is arrived at. Grave paroxysms precipitate this result. The oligocythæmia is associated with nucleated normoblasts and leucopenia, in which the large mononuclear cells are said to be relatively above the normal in number. True pernicious *anæmia* with megaloblasts is certainly found, especially among pregnant women and the aged (Marchiafava and Bignami). The *melanosis*, due to the constant deposition of black pigment in the organs, grows more intense as the infection advances, but tends to be eliminated as the invasion subsides. On the other hand, *siderosis* (deposition of yellow pigment) increases as the invasion subsides. The *spleen*, sensibly enlarged even after the first paroxysm, increases with each relapse, until it may reach almost to the crest of the ilium and beyond the umbilicus. The *liver* shows a similar enlargement, extending several inches below the costal margin. Both organs may be indurated, and may be tender on pressure, or may even be the seat of a dull aching. In cases of grave, and especially of tender, tumours, the writer has found in India a condition of *cachectic fever* of a continued type, oscillating within a few degrees above normal, not accompanied by parasites even in the splenic blood, and not amenable to quinine. He is inclined to attribute it to the state of the organs; to which Kelsch and Kiener refer their *fièvre symptomatique*, which is probably the same thing. Debility, emaciation, cardiac bruits, epistaxis, darkening of the skin, occasional icterus, diarrhoea, constipation, dyspepsia, and, later, œdema of the feet and face, and ascites, fill in the outline.

It is important to note, however, that in many cases, especially among well-to-do or habituated persons, secondary symptoms scarcely appear, chronic malaria finding expression only in occasional paroxysms occurring at long intervals. It also seems that enlargement of the liver and spleen depends upon local and racial conditions as well as upon malaria. Thus the writer has not found it in one or two severe and lasting endemics. Rogers gives observations tending to show that splenic tumour may depend on impurity of water-supply.

Daniels shows that while a very large percentage of negro and Indian children possess splenic tumour in British Guiana, the former recover from it first. It is probable that various conditions or complications, such as habitude, climate, poverty, food, diarrhoea, dysentery, and helminthiasis, modify the secondary symptoms in many ways.

8. *Malarial Cachexia*.—As the disease progresses further, the parasites disappear entirely in many cases, even the melanin being eliminated from the spleen. This is shown by examination of splenic blood during life; and Daniels (confirmed by the writer) shows that a considerable percentage of greatly enlarged spleens and livers found at autopsies contain no melanin at all. In such cases, however, the secondary symptoms—anæmia, splenic and hepatic tumours, &c.—are very apt to remain after the extinction of the invasion; and we obtain the final condition of malarial cachexia.

In early cachexias it is probable that the secondary or cachectic fever may be present; but later on, especially as the splenic tumour becomes indurated and then decreases, the temperature falls to sub-normal, and a state of extreme debility, with any or all of the symptoms noted above, remains. Death may occur from asthenia, or more commonly from complications, especially from pneumonia during the cold season, and diarrhoea and dysentery during rainy seasons. On the other hand, complete recovery may set in even at this stage, especially in children and well-fed and well-treated persons.

9. *Sequelæ*.—Owing to the length of the chronic malarial infection, it is always difficult to say whether a sequelæ is a resulting or an intercurrent affection. Paresis, paralysis, aphasia, ataxia, and chorea, sometimes follow grave paroxysms. The pathogeny of nervous sequelæ is traced chiefly to cerebral or cerebellar punctiform hæmorrhages. *Ulcerative enteritis*, *acute and chronic nephritis*, *albuminuria*, and *glycosuria* are fairly well established sequelæ both of parasitic and cachectic malaria. *Cirrhosis* of the liver is given by Kelsch but disputed by others. *Punctiform hæmorrhages* of the retina and *deafness* are sometimes found. *Rupture* of the *spleen* from slight injuries is far from uncommon. Most important sequelæ, if they can be called so, are *poverty* owing to the inability to work, and perhaps a proneness to *helminthiasis*. Generally speaking, malarial disease has no phlegmonous tendency. (See also p. 770.)

10. *Complications*.—The condition of things which has been called *typho-malarial fever* should be looked upon as being a mere concurrence of the two diseases. Typhoid fever like other disease (e.g. gonorrhoea) is apt to bring on relapses of malarial fever. A more important and very fatal complication is that of *pneumonia* in malarial cachectics. *Dysentery* and *diarrhoea* are almost as frequent and fatal associations in the tropics. The writer is of opinion that *ankylostomes*, *round-worms*, *whip-worms*, *amæbe*, and *cercomonads*, all tend to be more common in poor malarial cachectics. *Insolation*, *tropical hepatitis*, and *abscess* are often associated with all stages of the disease. The physician should remember (*a*) that intercurrent infections predispose to malarial relapses, and (*b*) that the reduced vitality due to chronic malaria seems to predispose to intercurrent infections.

DIAGNOSIS.—The physician may be required, not only to distinguish the disease at any stage from other affections, but to determine the species of

the parasite, the severity and progress of the invasion, or the presence of post-malarial and cachectic fevers.

During the first paroxysms a prompt and definite diagnosis cannot always be made on clinical grounds alone—at least in early cases. In many instances the first paroxysms have a subinfrant character; the fever is remittent and approaches the continued type; and the secondary symptoms have not yet developed. Hence, as it is bad treatment to delay the exhibition of quinine until the rhythmical quality of the fever declares itself, immediate examination of the blood is often demanded. In the majority of cases this clinches the diagnosis at once by revealing the presence (a) of the parasites, and (b) of pigmented leucocytes. In the first one or two paroxysms, however, both parasites and melaniferous leucocytes may be still too scarce to be easily detected. In such cases we are often guided by the leucopenia and large number of mononuclear cells after the crisis (Christophers and Stephens). The same condition may, however, occur in typhoid fever.

In *somewhat older infections* both parasites and pigment and the periodical nature of the fever become almost invariably manifest. To find the first with certainty, however, the physician must be a practised observer; he must be able to detect the small amœbule of the æstivo-autumnal parasites; he should search the blood when the temperature is rising; and may have to search it more than once. As regards the fever, although the classical curves may not be established, a sudden drop to below normal (e.g. to 97° F.) is very indicative of malarial fever, if powerful antipyretics have not been given.

Still later the occurrence of relapses and the appearance of splenic and hepatic tumours and of the dusky complexion are additional aids to diagnosis. In *old cases*, when the parasites are absent or too scanty to be found, we must rely upon the secondary symptoms and the character of the anæmia. Ankylostomiasis is very apt to be taken for malarial cachexia. In early cases of the former the ova of the worms are to be found; in those of the latter, splenic and hepatic tumours exist. Advanced cases of both, however, may often exhibit little difference, except perhaps in the deeper anæmia of ankylostomiasis.

The curative effect of quinine in malaria is always a valuable aid to diagnosis. Post-malarial and cachectic fevers are distinguished by their continued type, by the absence of fever-producing parasites, and by resistance to quinine. Examination of the splenic or hepatic blood is demanded in some cases.

DETECTION.—The microscope must have a one-twelfth oil-immersion lens, and a sub-stage condenser. Use the flat mirror. Do not place too near the window. Employ as much light as is consistent with good definition—pigment shows best in a bright light. To cleanse glasses in water will suffice. New cover-glasses must be soaked in acid and then washed. Rub the finger dry and clean before pricking. Prick the insensitive skin above the nail. The blood must come easily. Take up on the cover-glass a drop the size of the head of an ordinary pin. Wipe the slide. Drop the charged cover-glass upon it from the height of half an inch. Examine fields where the corpuscles lie fairly separate but are not crushed; for the larger parasites thicker fields will suffice. Do not waste time over bad fields or specimens. The student must make

himself familiar with vacuolated and crenated red corpuscles, dirt and flaws in the glass, blood-plates, &c., by studying his own blood. Vacuoles have a thick refractive margin, appear to alter in size with change of focus, and often quiver within the corpuscle—things never seen with amœbulæ. Ring-shaped and eye-shaped vacuoles are sometimes seen, but these are seldom quite circular like amœbulæ. If *only* small amœbulæ, or only those with crescents, are present, the case is æstivo-autumnal. If larger pigmented forms, still enclosed in corpuscles, are numerous, it is quartan or tertian. In this case, if the infected corpuscle is small, we have a quartan; if large, a tertian case. Crescents which have become spherical may be mistaken for large quartan and tertian organisms, but the former are not enclosed in corpuscles. Examine at the beginning of the paroxysm. A parasite will generally be found in one in ten fields; sometimes several occur in one field.

To *dissect mosquitoes*, kill with chloroform and examine fresh. Pass a needle through the thorax and hold down the insect in a drop of water placed on a glass slide. With another needle separate the last two tail-segments. Pull out the stomach, intestine, Malpighian tubes, and ovaries. Examine the stomach for zygotes, first with low and then with high power. They will be seen as small circles containing melanin when young; as large spheres later. For the salivary glands use fairly strong salt-solution and tease out the scales near the neck. The blasts will appear like snips of thread in the body-fluid or in the cells of the gland. Insects gorged with blood should be kept alive for a day or two until the stomach has emptied itself. They need water and bananas to be kept living for long.

Staining is not necessary for diagnosis, and cannot be discussed here. Almost any method suffices merely to colour the parasites, both in man and in the gnat; but in order to distinguish the nucleus the Romanowsky method should be employed, and the reader must study the monographs on malaria.

To examine *splenic* or *hepatic* tissues during life insert a rather large hypodermic needle (without the syringe), the patient holding his breath. Leave *in situ* for about 60 seconds, during which the needle fills with blood. Withdraw and blow out the blood and tissue cells upon a slide. Parasites and black and yellow pigments can easily be found. The writer has never observed any real inconvenience produced.

Since the life of the whole group or horde of organisms coincides exactly with the life of any one of the individuals constituting it, it will readily be understood that in a single infection by the quartan parasite sporulation occurs only every 72 hours, that is, every third day; in the tertian it occurs every other day; in the æstivo-autumnal every day or every other day according to the variety.

In the case of double and treble infections there are two or three distinct hordes of parasites of the same kind in the same host—distinct, because at a given moment they present different stages of growth. Thus, when one horde is sporulating, another is still growing, and will sporulate tomorrow or the next day according to the species. Similarly we often see *mixed infections*—that is, cases in which hordes of two or even three different kinds of parasites exist in the same host. In all these cases each horde continues to propagate inde-

pendently of any others which may be present along with it.

The parasites pass the greater part of their existence safely ensconced within the corpuscles and engaged simply in growing. Their only effect during this period is to consume and destroy the individual corpuscles in which they happen to lie. But when the moment comes for them to rupture the corpuscles and scatter their spores in the liquor sanguinis, other and more important phenomena assert themselves. Together with its spores, each parasite liberates a few black granules of melanin—the altered hæmoglobin which it contains. This is ultimately taken up by the phagocytes of the blood, and finally finds its way to the spleen and other organs, which in the course of time it renders melanotic. At the same moment, moreover, each parasite liberates a minute quantity of some toxic substance. If the whole horde of parasites sporulating at the moment be a large one, the total amount of toxin liberated must also be large; and if this be sufficiently large, it produces the fever and other symptoms which are characteristic of a typical attack of malarial fever. Hence it follows that the attacks of malarial fever coincide in time with the sporulation of each successive horde of parasites; and, since this occurs at regular intervals of one, two, or three days, according to the species of parasite concerned, the successive attacks of fever must show a similar rhythmical periodicity—which is, in fact, the distinctive peculiarity of malarial fever.

PROGNOSIS.—This is much better in temperate than in hot climates; and in those who take quinine well, who can afford good food and removal from malarious localities, and who belong to habituated races. Pernicious attacks are certainly much less common with the quartan and tertian parasites; on the other hand, these parasites often produce very obstinate infections in hot climates. Nearly all infections *can* be stamped out if taken in time. Recovery is often complete even from very severe cachexias. Pernicious attacks are very fatal—especially the cerebral and choleraic forms, particularly when occurring after considerable anæmia has been already established, or in old people. Pneumonia, chronic dysentery, and diarrhoea are grave complications. Amyloid degeneration necessitates a bad prognosis. As a rule the prognosis is good in malarial disease if the patient can and will follow the directions of his medical adviser.

TREATMENT. 1. Preventive.—*Personal.*—In all malarious localities mosquito-curtains should be scrupulously used during sleep. They should be entirely free from the smallest holes, rents, or apertures; should be carefully tucked under the mattress or bedding; and should be entered with precautions to prevent the simultaneous entry of gnats. A punkah over the curtains adds to the safety and comfort of the sleeper; and punkahs should be used in the days and evenings as much as possible. *Culicifuges* are not to be trusted.

Quinine is a very good prophylactic. The writer prefers 5 to 10 grains daily before breakfast. Others recommend 15 grains or so twice a week. It is certain that infection can be largely postponed, if not entirely prevented, even in the most malarious districts, by persons who employ such measures with care and common-sense.

Domestic.—Punkahs, fixed wire-gauze screens to the windows, whitewashed walls, and removal of

all stagnant water from tubs, flower-pots, and depressions in the ground are called for.

Barracks, gaols, hospitals, and rest-houses should always be carefully attended to in these respects. Patients suffering from either acute or chronic malaria should be invariably protected by nets.

Segregation.—Europeans generally contract the disease through the agency of *Anopheles* from natives—especially from native children—living in their vicinity. In all malarious tropical towns the Europeans should live in a separate quarter. The houses should be large airy structures, the rooms being whitewashed or painted white, and provided with a white ceiling. Surrounding bush should be cleared as far as possible—though a few large trees may be left.

Municipal measures.—In all towns possessing sanitary agencies, the whole area should, if possible, be gradually drained and levelled so as to remove all pools where *Anopheles* may breed. Agents should also be employed to destroy larvæ by oil or tar, or by overturning vessels of stagnant water. In the writer's opinion these measures will be likely to protect a larger number of people at less cost than measures of personal or domestic hygiene alone can do. Koch recommends treating a whole population with quinine; this may be practicable in certain isolated communities.

The best rule is to adopt as many measures as are possible under local conditions.

Exemption.—A certain degree of exemption is certainly often acquired by those who have passed through a long malarial infection. Thus a large percentage of native children suffer from the disease in all its stages; but as they grow older they throw it off; and, though they occasionally suffer later from fever, they do not suffer much. Hence, adult natives often seem to escape where Europeans contract grave infections. No means of conferring protective properties is known.

2. Curative.—Quinine is a specific against the propagation of the parasites within the blood; but it must be employed in a skilful manner. The object is not to reduce a given attack of fever, but to rid the system of the parasites entirely. Immediately after a definite diagnosis is obtained, the drug must be given *promptly* and in *sufficient doses*, and must be *continued for months*. The writer considers it a grave mistake to withhold it until the temperature falls below normal; though, perhaps, *excessive* doses should not be employed when there is high fever. The most effective time for administration is about an hour before the commencement of an expected paroxysm; but in the early remittent stages quinine should be given thrice daily. As a rule, the daily amount for an adult need scarcely *exceed* 30 grains, distributed according to the physician's discretion. The amount of 'singing in the ears' is a good test of the degree to which the system has been saturated. A useful rule is just to maintain a considerable degree of singing in the ears while the parasites or pigmented leucocytes are sufficiently numerous to be detected, and for a week afterwards. In a hot climate the parasites appear to be much more resistant to quinine than in a temperate one. After the attack or relapse has passed off for a week, the dose may be reduced to 10 grains daily before breakfast, and 5 grains again in the evening—to be continued for another two weeks. Then, gradually, further reductions may be made. Manson recommends 15 grains in

three doses once a week. The susceptibility of the patient must be a guide. The drug must not be entirely abandoned for at least three months after an attack.

The writer always recommends quinine to be given in acid solution just before a meal, the first mouthful of food being swallowed just after the medicine. This removes the taste at once, and mitigates the irritant effect on the stomach; while singing in the ears often announces absorption within half an hour. Pills and capsules are apt to become insoluble in damp tropical climates. Rectal or hypodermic administration is often necessary: in the former case, up to 20 grains or more thrice daily; in the latter, up to 10 grains thrice daily. The physician should always keep special tabloids of quinine, and a good syringe for hypodermic use (see HYPODERMIC MEDICATION). If he possesses only the sulphate, this should be dissolved by adding half its weight of tartaric acid with the requisite amount of boiled water. The syringe and needle should also be boiled; and the injection made into muscle.

In *chronic malaria* it is not necessary to press quinine unless there is evidence to show that the parasites are still present. Small doses suffice at this stage; but on the other hand, close attention must be given to the secondary symptoms. Arsenic, iron, and sodium sulphate and bicarbonate are undoubtedly beneficial. Starved cachectics require careful feeding. At all stages removal to a temperate climate, sea-voyages, or courses of medicinal waters are to be recommended.

Subsidiary symptomatic treatment is not to be neglected; but details must be left to the physician. Purgatives and also opium are often most useful. Methylene blue has been rather discredited. Warburg's tincture is valuable (dose about one table-spoonful). Antifebrin and phenacetin are much abused, and the writer even thinks that they may be harmful if given before the crisis, or too frequently. In hyperpyrexia tepid baths are better. A full knowledge of the pathology is the first essential of treatment; and the physician is not justified in neglecting the logical use of quinine for popular nostrums. In malarious localities the patient must invariably sleep in well-arranged mosquito-curtains, to avoid infecting others and re-infecting himself. Quinine must be well pushed in pernicious attacks. Entozoa should be expelled.

See also BLACKWATER FEVER, p. 143.

RONALD ROSS.

MALFORMATIONS (*male*, amiss; and *formo*, I fashion).—SYNON.: Fr. *Malformations*; Ger. *Missbildungen*. See MONSTROSITIES.

MALIGNANT DISEASES.—This term is applied, first, to tumours which, without any natural limitation, gradually involve the surrounding tissues, reproduce themselves in distant parts, and, if unremoved, inevitably lead to the destruction of life; and, secondly, to certain varieties of fevers and other acute affections, such as typhoid fever, scarlet fever, small-pox, and cholera, which present peculiarly grave and aggravated symptoms, and generally end in death.

MALIGNANT PUSTULE.—See PUSTULE, MALIGNANT.

MALINGERING.—See FEIGNED DISEASES.

MALLEIN.—See GLANDERS.

MALTA.—A warm, moist, and variable winter climate. See CLIMATE, Treatment of Disease by.

MALTA FEVER.—SYNON.: *Febris undulans*; Mediterranean Fever; Gastric Remittent, and Bilious Remittent Fever; Rock-Fever (Gibraltar); Neapolitan Fever, &c.

DEFINITION.—An endemic disease of long duration, characterised by fever, enlarged spleen, profuse perspiration, constipation; by almost invariable relapses: accompanied by pains of a rheumatic or neuralgic character, and sometimes swelling of joints or orchitis; and characterised in fatal cases by enlargement and softening of the spleen, no swelling or ulceration of Peyer's patches, and the constant occurrence in various organs of a species of micrococcus.

GEOGRAPHICAL DISTRIBUTION.—Formerly this fever was believed to be confined to the shores and islands of the Mediterranean, where it is widely distributed; recent investigations tend to show that it occurs in other places—India, Brazil, Cuba, Puerto Rico, Hong-Kong, Manila, and even in England.

ÆTIOLOGY.—Malta fever is probably due to the introduction into the system of a specific micro-organism (*Micrococcus melitensis*). This assertion is based upon the following facts: (1) This micrococcus is found in the organs of every fatal case. (2) This bacterium can be readily cultivated outside the body. (3) Inoculations of pure cultivations into monkeys have caused fever and death. (4) From the organs of these monkeys the identical micrococcus has been abundantly obtained. (5) Inoculations of cultures have been followed by the disease in man. (6) The serum of the subjects of Malta fever, in high degrees of dilution, causes agglutination and sedimentation of cultures of *Micrococcus melitensis*. As to how, under natural conditions, this micrococcus gains entrance to the organism nothing definite is known. There is no evidence that Malta fever is communicated directly from individual to individual. One attack of Malta fever probably confers immunity against a second.

Age and Sex.—This disease chiefly affects young persons between the ages of ten and thirty; less frequently under ten, and from thirty to fifty; and very rarely above fifty. It does not seem to occur among infants, or if it does it is not recognised. Sex has little influence, but the complaint is more common among men than women.

Station in life.—Malta fever attacks the well-to-do classes, living in large well-ventilated houses, probably in as large a proportion as it does the poor in their more crowded dwellings.

Months and Seasons.—The summer is the season of the greatest prevalence of the disease, and, in the Mediterranean at all events, most cases occur in the month of July. A marked diminution takes place in autumn, and it is rare in the winter and spring months.

INCUBATION.—It is impossible to say definitely how long the period of incubation is, but the writer would put it approximately at ten days.

CLINICAL DESCRIPTION.—*Early Symptoms.* For the first week or ten days sleeplessness and headache are complained of, which may be mild or severe; the appetite is absent; there is nausea, sometimes vomiting, and a feeling of weight and tenderness in the epigastric region; constipation is

the rule, diarrhœa the exception. The spleen and liver are enlarged, and both may be tender on pressure. Tympanites is uncommon, but may occur, as also may gurgling in the iliac fossa. During this time almost invariably a slight cough with scanty expectoration is developed, and on examination the breathing at the bases is found to be unsatisfactory, harsh, and creaking in character, with now and then a moist crepitation. There is no eruption, but the patient suffers from a most profuse perspiration, and a more or less abundant crop of sudamina is developed. There may be a little delirium at night during this time, but this is rare, and is so slight as scarcely to call for remark. Unless there be headache or severe pain in the lumbar region, the patient for the first week or two usually professes that he suffers very little; at the end of this period the headache and acute symptoms usually disappear, and the long and monotonous period of the fever begins, a period which seems interminable alike to medical attendant and patient. The patient's aspect is natural but listless; his tongue is clean; he has a wish for solid food, which must often be denied; and his bowels require the stimulus of an aperient or enema for evacuation.

Later symptoms.—The profuse perspiration still continues, and day after day the patient becomes weaker and loses weight, until he has scarcely power to stagger a few yards. During this period the temperature often ranges high. The patient sleeps moderately well, has no delirium or restlessness, is uncomplaining, and takes without any ill effect a large supply of fluid food and stimulants. The only variation in his condition is afforded by a rheumatic affection of the joints; one day the knee is found red, swollen, and intensely painful on being touched, a few days afterwards a wrist or ankle may be attacked. Sometimes almost all the large joints in the body are affected in this way, or there may be intercostal neuralgia, sciatica, or an inflamed and swollen testicle. Thus many weeks may pass, but at last the temperature comes down to the normal, and the patient enters on a long and tedious convalescence.

Temperature.—The chief characteristic in regard to the temperature-curve in this fever is its irregularity. The type varies from the continued to the intermittent: one case is almost continuous throughout, another almost intermittent; some cases begin with a markedly intermittent type, and pass into the continued, while others begin as continued and pass into the intermittent. Some severe cases show a long irregular elevation of temperature, only reaching normal limits about the ninetieth day. The temperature-curve, as a rule, rises high, reaching 104°, 105°, and even 106° F.

DURATION.—Patients with Malta fever show an average stay in hospital of nearly ninety days. The length of the fever may vary from fifteen days to as many weeks or more.

DIAGNOSIS.—Apart from their respective serum-reactions, a severe rapidly fatal case of Malta fever cannot be distinguished from a similar case of typhoid except by *post-mortem* examination, when the absence or presence of a specific anatomical lesion in the small intestine at once separates the two kinds. If, as many hold, these fevers are caused by the entrance into the body of specific micro-organisms, then it is evident that the most rational and scientific method of classifying them would be by the identification of the parasite peculiar to each. In all cases

of Malta fever there is found a minute round or oval bacterium—the specific micrococcus; whereas in typhoid there is found a much larger rod-shaped micro-organism—the typhoid-bacillus. Principally through the labours of Professor Wright, a valuable aid in the diagnosis of Malta fever has been discovered in the agglutinating and sedimenting power of the serum of patients suffering, or who have lately suffered, from the disease on cultures of *Micrococcus melitensis* (see SERUM-REACTIONS). In the case of this bacterium the specific reaction is obtainable at an earlier stage (second or third day) of the disease, and with much higher dilutions (1 : 100 or more) than is the case with the corresponding test for typhoid fever. The specific property of the serum persists for many months—perhaps years. Ordinary cases of Malta fever can be distinguished from typhoid by their long duration, the tendency to constipation, the absence of a specific eruption, and the much smaller rate of mortality, which does not exceed 2 per cent.

TREATMENT.—This is a specific fever, and no drug at present known has any power of modifying its course. The sulphate and salicylate of quinine, Warburg's tincture, eucalyptus, calomel, salicylic acid or the salicylates, carbolic acid, and other drugs have been tried again and again for this purpose, but without any good result. Medicinal treatment must therefore be directed to mitigate severe symptoms. At the beginning of the fever phenazone is often found useful to combat severe headache and sleeplessness. For the constipation a mild aperient or simple enema will very often prove necessary. For the neuralgic and articular pains, hypodermic injection of morphine, and the liniments of aconite, opium, and belladonna will be prescribed. Treatment must therefore be principally directed to keeping up the patient's strength by judicious dieting, and, when required, by stimulants, and by attention to ordinary hygienic principles. Removal of the patient from the affected area does not cut short the course of the fever; but, as in many other diseases, complete restoration to health will certainly be hastened by change of climate.

DAVID BRUCE.

MAMMARY CONCRETIONS AND CALCULI.—See CONCRETIONS.

MAMMARY GLAND, Diseases of.—See BREAST, Diseases of.

MANIA (*μανία*, fury, madness).—SYNON.: Fr. *Manie Suraigue*; *Délire Aiguë*; *Fureur*; Ger. *Tobsucht*; *Wuth*.

Under the term 'mania' very distinct disorders or degrees of disorder have been described, which we shall speak of as *Acute Delirious Mania*; *Acute Mania*; and *Mania*.

I. Acute Delirious Mania.—An outburst of delirious mania may take place after very few and very short premonitory symptoms. Quite suddenly, after a few days or even hours, the patient will display the most violent excitement, which may as suddenly subside, or run a well-marked course of a few weeks; and if it does not terminate fatally will gradually decline, recovery usually taking place. Such an attack may have its origin in some sudden mental shock, as the death of a friend, a violent quarrel, a disappointment or suddenly announced misfortune; or it may arise in the course or decline of an acute disease, as pneumonia or measles.

It may also come on during rheumatism, or after great fatigue, an epileptic seizure, or childbirth. A theory lately propounded that this disorder is due to autotox poisoning is not at present supported by any evidence.

We cannot tell at first whether the attack will be transient or prolonged. We may try to cut it short by a brisk purgative, and by such medicines as chloral hydrate and bromide of potassium, a subcutaneous injection of hydrobromate of hyosine, or a full dose of paraldehyde; and these not infrequently answer the purpose. Sleep is procured, and perfect recovery may take place in a few days. There are patients whose organisation is so unstable that it is thrown off its balance by a cause perhaps trifling, but which produces a tremendous nerve-discharge and complete disturbance of the whole mental functions. But so transient may this be, that one sleep restores the normal equilibrium, and the patient is cured. This condition in females is often called hysterical—*hysterical mania*. There is no special connection between it and the uterine functions, and it is better to retain the name 'hysterical mania' for a variety to which it may be more appropriately given.

The delirium, however, does not always terminate quickly. If sleep becomes less and less, the mind more and more confused, and quiet and lucid intervals rarer, we may be sure that the attack will be serious and prolonged, and that careful and efficient nursing for some time will be necessary. Where a quiet and airy room can be provided, and a patient's means are sufficient to allow him an adequate staff of attendants, an asylum is not indispensable. He will not require to take exercise in a garden; he will not be dangerous, as some are, to himself or others, though he may be violent and excited. He may be noisy, however, and so not able to remain unless the house is detached. The room should be lofty and cool, the windows protected and darkened; all furniture must be removed, and the bed made on mattresses placed on the floor, for he will not lie on a bedstead, and attempts to keep him there will end in bruises or more serious injury. Clothes will be torn off; but if the weather is very hot, as is so often the case during these attacks, this will be of little consequence. If it is cold, a strong suit laced up the back may be put on, and underneath it the requisite body-clothes; or a blanket may be placed round the patient, and fastened up the back.

These patients are in incessant motion, singing, shouting, and talking in a string of incoherent utterances, often repeating the same sentence again and again, or a snatch of a song or text, or a rhyme of their own composition. As a rule they are not violent, and do not attack those about them, though they may resist that which is done for them. They may be hilarious and full of glee and mischief, which is a good sign; or terror-stricken, with visions of horrible objects, which is unfavourable. They are wet and dirty; and the urine will be high-coloured and often retained for a long period. We shall derive valuable information if we are able to take the temperature; but often that is a difficult task. A high temperature is a bad sign; and so is a rapid pulse if it continues persistently when a patient has not been using violent exertion for some time. The tongue will often become thickly coated, dry, and brown. If it does not, but remains moist and comparatively clean, this is of good omen.

PROGNOSIS.—The prognosis in these cases is upon the whole favourable. The terminations are almost always either recovery or death. The patients are mostly young persons, who recover unless weakened by previous attacks, other disease, or childbirth. Many of the fatal cases, in the writer's experience, have been complicated by tuberculosis.

TREATMENT.—Sleep in the attacks now under consideration is generally absent, sometimes for many days. Women can last longer without sleep than men, and die much less frequently in acute delirium. If sleep does not come the patient dies, and our great effort must be to promote sleep by various methods. The first question will be whether we are to give drugs to accomplish this; and, if so, what drugs. Opium must not be given; it will not procure sleep, whether given by the mouth or subcutaneously. It may produce a slight narcotism for half an hour or so, and, if we increase the dose, will cause narcotic poisoning and death; but in the height of the attack it will not procure sleep. Chloral hydrate we may try in combination with bromide of potassium, giving half-drachm doses of each, and watching the effect. In most cases sleep of longer or shorter duration will be caused by these drugs; and although it may be short, it may be sufficient to save the patient's life, and enable him to battle successfully with the disorder. In the writer's experience many more of these acutely delirious patients died before the introduction of chloral hydrate than since. Yet it must not be given in enormous or repeated doses, and a considerable interval should elapse between them. It may be administered easily in stout or ale, and often in wine. We may also give paraldehyde, or that powerful but somewhat dangerous drug, hyosine. Such drugs as these are not to be administered frequently, or at regular intervals. We wish to procure sleep enough to prevent the patient from dying by exhaustion; but in this very acute form no medicine is likely to produce more than a short sleep, and is more likely to do this if not given too frequently, and less likely to produce bad effects. Macleod has recently advocated the administration of large doses of the bromides—two drachms every two hours during the day till an ounce is given, and on the second day a similar amount. This produces a deep sleep, lasting some days, from which the patient must be roused and fed at regular intervals.

Next to sleep the most important matter is food. To enable the sufferer to withstand the exhaustion, which is the cause of death when a case ends fatally, he must be fed frequently and liberally, and, if necessary, by force (*see* FORCIBLE FEEDING). These patients rarely refuse food, but require careful coaxing and feeding; and a skilful attendant will give something every two or three hours—minced meat and vegetables, or bread and milk, beef-tea, eggs, and the like. Brandy often produces great excitement at the onset and height of an attack, and stout or ale is more suitable and more likely to bring about sleep. We may give also plenty of lemonade, barley-water, and such drinks, if there be great heat and thirst.

Although this unconscious or semi-conscious delirium may continue for many days, yet in almost every case the violence and excitement are paroxysmal, with intervals of comparative calm, even if there be no sleep. Judicious attendants will avail themselves of these quiet intervals to administer

food, and keep the patient in the recumbent posture, thus ensuring rest, instead of letting him be continually on his legs wandering about the room, and so exhausting his strength. And when held down quietly, with cold cloths applied to the head, or his face fanned by the nurse, he is not unlikely to drop off to sleep. Cold to the head may be applied, because it is soothing and grateful to the sufferer, though it is a question whether the circulation in the brain is most affected thereby.

The bowels may be kept open by a dose of calomel administered in the food, or by half-a-grain of resin of podophyllum. Active purgation is inadmissible except at the very outset, and enemata cannot easily be given in the violent stages. It used to be the fashion to apply blisters to the nape of the neck or calves. This is most inadvisable, for such parts may become very sore, owing to the restlessness of the patient, and thus deprive him of sleep. Neither is it necessary to cut all the hair off, which in the case of women may be a very grievous matter. If very long it may be shortened without being cut close to the head.

II. Acute Mania.—Quite different from the unconscious raving of maniacal delirium is the conscious but violent excitement to which we give the name of *acute mania*. The former is a disorder dangerous to life, running a rapid course to death or amendment in a week or two. The latter may go on for weeks or months with little danger to life, but with excitement so troublesome that the sufferers require the restraint and discipline of an asylum. Though most insane, full of delusions and outrageous habits of every kind, they know what they are about, and are all the more mischievous in consequence. They can take every advantage of an opportunity, and know how to exasperate those about them. They generally eat well, and sleep indifferently, but sufficiently to support life; and their bodily health often remains wonderfully good, considering what they go through. They will destroy clothes, windows, bedding, and deny or justify all they have done. The termination is not usually fatal, unless the health gives way through some other disease. The patients generally recover gradually, or sink into chronic mania or dementia.

PROGNOSIS.—The prognosis in cases of acute mania will depend upon circumstances. (1) The number and duration of the attacks are important. In a first attack the prognosis is favourable. If recent, we may have hopes, even if there have been preceding attacks of a like character. (2) If the patient is not of advanced age or of broken health, the prognosis is favourable. (3) If the mania consists of violent, turbulent conduct, rather than of fixed delusions, as is frequently the case, there is more hope. If the patient hears voices, the prognosis is bad. If there are delusions which impel him to refuse food, and he does so persistently and violently, it may be difficult to give sufficient nourishment, and he may sink from exhaustion, or become a chronic maniac.

TREATMENT.—Patients suffering from this form of mania do not require, like the last, to be kept in one room; on the contrary, they should take plenty of exercise in the open air. This will promote sleep more than drugs, though we may give an occasional dose of chloral hydrate, or bromide of potassium, or paraldehyde. Such medicines, however, should be given only to procure sleep, not to

allay excitement; we may also try sulphonal or hyosine. In many cases, however, sleep procured by drugs appears to prolong the attack, and, where there is no danger to life, it is better to omit them, and to let natural sleep come after the fatigue of exercise. Plenty of food is required, for the waste is great.

Patients of this kind are not to be cured or even kept without discipline and moral treatment; and great tact, firmness, and patience are required for their management. They may be very dangerous and spiteful, will know how to provoke attendants, and how to take them unawares if off their guard. Moral treatment will be far more efficacious than drugs, but it can only be carried out in an asylum. Patients in this condition, if kept in private houses, must be rendered quiet by drugs; but there is great fear lest by this method the disease, instead of being cured, may be converted into a chronic and incurable mania.

III. Mania.—A great variety of cases are grouped under this name, arising from various causes, but alike in the fact that they are marked by excitement rather than depression, by exaltation or wrath, but not by gloom. Excitement and noisy and irrational conduct characterise some, but most patients present delusions coinciding with their temper and bodily condition when in health. Almost always this form of insanity is marked by delusions, if it lasts long enough; but sometimes a short burst of excitement—a transitory mania—may pass away without the stage of delusion being reached, or there may be throughout outrageous conduct or extravagance without delusion.

The diagnosis of an ordinary case of mania is not difficult. The prognosis must depend on the cause; the age of the patient; the character of the delusions, if there be any; the occurrence or non-occurrence of previous attacks, and their history. Attacks of mania are frequently recurrent, and may be repeated again and again through a long life; recovery may take place on each occasion, or the disorder may at last turn into chronic mania or dementia. The period of excitement in many cases is followed by one of depression, and these may alternate with great regularity for twenty or thirty years; and even when the patient is sunk into hopeless dementia the period of excitement may occur as regularly as before the mental powers had given way.

TREATMENT.—Of the treatment of these cases a great deal cannot be said. The majority will require the care and vigilance to be found in an asylum, at any rate during the excited stage. The intervening or rational period will often be prolonged advantageously by removal from the asylum; and when this is the case there will be frequently found less reluctance to return to it when the necessity arises, and instances are not uncommon of patients even themselves seeking its shelter.

G. F. BLANDFORD.

MANIPULATION (*manus*, the hand).—The investigation and the treating of disease by the use of the hands. See PHYSICAL EXAMINATION; FRIC-TION; MASSAGE; and SHAMPOOING.

MANITOU SPRINGS, in Colorado, is famous for its mineral springs and as a high-altitude station, being situate 6,370 feet above the sea-level, in a sheltered valley of the Rocky Mountains at the foot of Pike's Peak. The climate is that of Colorado

Springs, six miles distant (*see* COLORADO SPRINGS), and the mineral springs consist of: (1) alkaline and saline, useful in affections of the kidney and liver; and (2) chalybeate alkali, of repute in uterine disease. The climate has been found valuable in the treatment of chronic phthisis. The neighbourhood of the great tracts of Manitou and Estes Parks, 1,000 to 1,200 feet higher than Manitou Springs, being available for camping-out, is an additional attraction.

MARASMUS (*μαρῖνω*, I grow lean).—A synonym for general wasting. *See* ATROPHY, GENERAL.

MARIENBAD, in Bohemia.—Alkaline sulphated waters and mud-baths. *See* MINERAL WATERS.

MARSH FEVER. - *See* MALARIAL DISEASE.

MASSAGE.—*SYNON.*: Medical Rubbing; Mechanotherapy; *Fr. Massage*; *Ger. Massiren*.

DEFINITION.—A series of mechanical movements, best executed by the hands of the operator, affecting not only the skin, but also the deeper structures of the body.

MODES OF ACTION.—Massage acts in the following ways: (*a*) it quickens the flow of the fluids (blood, lymph, chyle, and others); (*b*) it increases secretion and excretion; (*c*) it excites muscular action.

METHODS.—The movements in massage are of several kinds: (1) Stroking, or *effleurage*; (2) Pressure, or *pétrissage*; (3) Percussion, or *tapotement*; (4) Vibrations; (5) Passive movements; (6) Active or Swedish movements; (7) Medical gymnastics.

Stroking is performed by lightly drawing the hand in one direction over the surface of the part: on the head from the vertex, and on the spine from the neck, downwards; on the limbs, from the extremities towards the trunk. When *friction* is employed, greater pressure is made, and the hand is moved to and fro.

Pressure (squeezing, kneading, rolling, &c.). The pressure and relaxation should be alternate and rhythmical, simulating natural muscular action. During the pressure the veins, capillaries, lymphatic ducts, and lymph-spaces are emptied; and the valves in the vessels preventing the return of the expelled fluids, room is made for a fresh supply.

Percussion (tapping, beating, pounding, and others) causes muscular contraction.

Vibrations act in a similar manner.

Passive movements.—All the normal movements of the joints are fully executed by the operator, the will of the patient being in abeyance. The synovia is increased, if scanty—absorbed, if in excess; deposits around the joints are removed, and nutrition is promoted.

Active or Swedish movements are performed with the combined help of the patient and operator. The will of the patient is concentrated on the muscles under treatment. The patient is directed to cause a muscle to act, and the operator resists the movement, employing slightly less force. When the muscle has fully contracted, the operator employs more force, while the patient, diminishing but not ceasing his resistance, allows the part to be brought back to its original position. This is repeated a suitable number of times, but never so as to cause muscular exhaustion.

Medical gymnastics have for their object the

bringing into action those muscles which are seldom employed, or which, for some special reason, require strengthening.

USES.—The maladies, both medical and surgical, for which these therapeutic agents have been employed with success are very numerous. It must suffice to mention the following: Muscular weakness and wasting; infantile paralysis; Bell's palsy; lead-palsy; neurasthenia; neuralgia; sciatica; peripheral neuritis; anæsthesia; hyperæsthesia; chorea; hysteria; occupation-palsies; some forms of arthritic disease, as rheumatoid arthritis and chronic rheumatism; and the morphine-habit.

JOHN FLETCHER LITTLE.

MASTALGIA

MASTODYNIA

(*μαστός*, the breast; and *ἄλγος*, or *ὀδὴν*, pain).—Pain in the mammary gland. *See* BREAST, Diseases of.

MASTICATION, Disorders of.—Mastication is liable to be disordered from various causes.

1. *Muscular Paralysis*.—Imperfect performance of mastication is frequently the result of cerebral lesions, such as hæmorrhage or tumours. Dependent on the seat and extent of these will be the extent of the paralysis, which may vary from an impaired movement of one cheek, thus permitting the food to collect between it and the gum, to almost an absolute inability to move the lower jaw from side to side, or to close the mouth.

There are several special forms of paralysis in which the movements of mastication are affected, either alone or in common with other muscles of the body.

(*a*) *Labio-glossolaryngeal paralysis* is especially characterised by the impairment of mastication and deglutition, which progresses from a mere escape of saliva, due to paralysis of the orbicularis oris, to complete inability to perform either act. Of the masticatory muscles, it is those of the tongue and lips which are mainly affected. As the disease progresses to its invariably fatal end, the palsy increases in completeness.

(*b*) The loss of power in the muscles of mastication associated with *diphtheria* is far less frequent than paralysis of deglutition, and is usually limited to some weakness of the tongue, and less often of the lips and cheeks.

2. *Muscular Spasm*.—Trismus, or tonic spasm of the muscles of mastication supplied by the motor branch of the fifth nerve, is rarely unilateral. The jaws are usually completely locked, and incapable of separation, thus rendering mastication impossible. The trismus may be a part of a general condition of tetanus, or may be the sole indication of spasm; and in the latter case is usually reflex in origin, being determined by such causes as dental irritation, or facial neuralgia, or, more rarely, by distant wounds or intestinal worms.

Irregular clonic spasms of the muscles of the jaws, such as are frequently seen in chorea and hysteria, and are evidenced by chattering and grinding of the teeth, will offer some difficulty to the proper performance of mastication.

Spasm, whether tonic or clonic, when limited to the facial muscles supplied by the seventh pair, will interfere but slightly with mastication. By preventing the action of the lips and cheeks, the food will not be so easily kept between the teeth, and the saliva will dribble from the unclosed mouth.

3. *Affections of the Temporo-maxillary Articulation*.—Chronic rheumatoid arthritis may lead to such serious disorganisation of the joint as to impair its movements, ankylosis occasionally occurring.

4. *Morbid Conditions of the Mouth*.—Inflammation of the mouth or tongue, and disorders or absence of the teeth, render mastication difficult. Enlargements of the salivary or lymphatic glands, tumours of the thyroid body, epulis, and new-growths of the tongue, as well as abnormal apertures in the palate, floor of the mouth, or cheeks, caused by ulceration or noma, may interfere with mastication.

EFFECTS.—Portions of food imperfectly masticated may produce suffocation, by blocking up the entrance of the glottis, or lodging in the gullet. Imperfectly masticated food is a well-recognised cause of dyspepsia.

TREATMENT.—The treatment of disorders of mastication naturally consists in the removal of their cause, when possible. The reader is referred to the articles in which the several conditions are fully discussed.

W. H. ALLCHIN.

MASTURBATION (*manus*, the hand; and *stupro*, I ravish).—SYNON.: Fr. *Masturbation*; Ger. *Selbstbefleckung*.

DEFINITION.—The excitement of the sexual organs by unnatural means.

ETIOLOGY.—Masturbation is practised under a variety of circumstances. First, in infants and young children, local irritation situated beneath the prepuce in males, or within the vulva in females, leads to manipulation of the parts, and to consequent pleasurable excitement, which is constantly renewed, with an entire unconsciousness of the meaning of the practice.

The second class includes individuals who have reached or are near the age of puberty, and have either accidentally learned, or been taught, this pernicious habit. *Balanitis* is a frequent exciting cause. *Pruritus vulvæ*, due to diabetes mellitus or other causes, may lead to it in the female.

A third class of cases may be mentioned, in which the practice has a central origin, in certain forms of brain-disease, or cerebral deficiency, as is seen in some forms of insanity and in idiocy.

EFFECTS AND SYMPTOMS.—There is no doubt that the practice of masturbation leads to the most serious constitutional effects. These effects are more especially manifested in the nervous system. The mental faculties become more or less affected; and often great despondency, loss of memory, irritability, prostration of strength, headache, and neuralgic pains ensue. The functions of the heart are disturbed. Digestion is disordered. There is general loss of health and strength; and chronic hypochondriasis is set up. In certain cases the urinary organs are affected; and the writer has observed in several instances the constant presence of albumen in the urine, possibly the result of some reflex action on the nerves and vessels of the kidney. The effects on the male genital organs themselves are marked. There is extreme irritability of the neck of the bladder and adjoining parts, accompanied by discharge of mucus and of prostatic secretion, often mistaken for semen. At the same time seminal emissions are prone to occur on the least sexual excitement, either by day or during sleep; and in extreme cases there is impotence.

DIAGNOSIS.—When carefully concealed, the

practice of masturbation is difficult to discover. When, however, the symptoms just described are present, in the absence of any cause to account for them the practitioner may entertain a reasonable suspicion of the existence of this habit, although it may be difficult in many cases to carry his impression beyond the suspicion.

TREATMENT.—In the first class of cases above mentioned—that is, in young children, in whom some local irritation exists—the source of this irritation must be found and removed. Sometimes it may be an elongated prepuce, with irritating matter beneath it; in such cases circumcision may be required. In females cleanliness and simple lotions may suffice; or irritation, caused by the wandering of thread-worms, or the passage of diabetic urine, may require to be treated. In these cases attention to the general health, to the state of the digestion, to the urinary secretion, and to the bowels, should not be neglected. Diabetes mellitus must be treated if present. Extreme watchfulness by the nurse is necessary, and at night it may be even necessary to secure the hands by muffling or tying them behind the back.

In young adults the moral sense must be acted upon. It has been suggested, by way of prevention, that judicious and kind advice may with advantage be given before even a knowledge of the habit is acquired; while too much vigilance cannot be exercised by those who direct and assist in the management of schools.

In the actual treatment of the effects established by masturbation, it is of the highest importance to improve the health, both mentally and bodily. Early rising, healthful exercise, careful diet, and travel if practicable, should be recommended. Remedies directed to the treatment of symptoms connected with the nervous, circulatory, and digestive systems will be required. Of course the habit must be entirely stopped, and all thoughts of a libidinous character must be avoided. The bromides, especially the bromides of potassium and ammonium, are very useful for lessening sexual excitability in both sexes. In certain cases where these remedies, together with iron, and other appropriate drugs, have failed to diminish the frequency of the seminal emissions which are common in males, caustics may be applied to the neck of the bladder. See SPERMATORRHEA.

MATCH-MAKERS.—See OCCUPATION-DISEASES.

MATLOCK, in Derbyshire.—Thermal waters. See MINERAL WATERS.

MAW-WORMS.—A synonym for thread-worms. See ENTOZOA.

McBURNEY'S POINT.—The point midway between the umbilicus and the right anterior superior iliac spine. See APPENDIX VERMIFORMIS, Inflammation of.

MEASLES.—SYNON.: *Morbilli*; Fr. *Rougeole*; Ger. *Masern*.

A specific eruptive fever of an exceedingly infectious nature, in which the appearance of the rash is preceded by well-marked catarrhal symptoms with fever. Very few persons exposed to the infection escape, unless protected by a previous attack; a second attack before puberty is almost unknown.

The *period of incubation* lasts from eight to twelve days: it is not accompanied by any symptoms.

ÆTIOLOGY AND PATHOLOGY.—No specific micro-organism has been satisfactorily identified. When introduced into a country for the first time the disease is apt to possess a quite unwon degree of virulence, as was shown by the fact that during the epidemic in the Fiji Islands in 1875 more than one-fourth of the whole population died from its effects, while in London the usual mortality is about .5 per 1,000; this would seem to show that a considerable degree of protection can be acquired by inheritance. A second attack before puberty is very rare, but in later life subsequent attacks are not infrequently met with, and even old age does not secure exemption for one who throughout his life has shown a special susceptibility. On many occasions, however, when a person is said to have had two attacks of measles a careful investigation proves that one of the attacks was rubella, and it would be safe to assert this if the two attacks occurred in the same year. The rash is not present after death except in malignant and petechial cases, when there is a general lividity of the surface of the body and probably some swelling of the face. Judging from what has been proved in the case of enteric fever it is probable that the rash is due to the presence of the micro-organisms in the skin, and that its subsidence is due to their death. The disease is most infectious when the rash is out, next during the period of invasion, and perhaps during the whole period of incubation. It is probably not infectious during deferescence after the catarrhal symptoms have passed away. A very short exposure is all that is necessary: thus the present writer remembers a child suffering from measles being admitted for only half-an-hour into a ward full of children, with the result that four children who occupied neighbouring beds subsequently developed measles, the rash appearing in all on the same day. The disease is capable of being conveyed by personal contact, by clothes, toys, &c. Outbreaks of measles are most apt to occur either during winter or just when spring passes into summer. See PERIODICITY.

SYMPTOMS.—The *period of invasion* may or may not be ushered in with rigors or convulsions. The occurrence of these depends on the age of the patient and the severity of the attack. There is always some fever; but catarrh is the prominent feature. This is often highly characteristic, and is of great diagnostic value. It commences in the mucous membrane of the nasal cavities, and spreads in all directions, resulting in headache, sneezing, coryza, conjunctivitis, lacrymation, sore throat, hoarseness, pulmonary catarrh, with a harsh hard wheezy or croupy cough, and occasionally epistaxis. On auscultation of the chest, sibilant sounds are heard all over, the tongue is thickly coated, and vomiting or diarrhoea or both are not uncommon. The temperature generally runs up at the onset to 103° or 104° F. or even higher, but is not usually maintained at quite the same level throughout the period of invasion, a slight drop being more commonly observed. This stage lasts from three to four days, and is followed by the *period of eruption*. The rash consists at first of minute, roundish, dull-red, velvety, very slightly raised papules, which disappear under pressure; these quickly multiply and spread, running together to form irregular crescentic blotches with well-

defined scalloped edges. When thus formed the blotches are slightly darker in colour, of the tint of an unripe mulberry; they do not fade so completely or so readily on pressure; and in severe cases a slight degree of ecchymosis into them is common. The skin between these blotches is normal except in very severe cases when on the face the intervening skin may be somewhat œdematous. In the most malignant cases there may be extravasations of blood into the blotches so that the eruption becomes petechial: this form is known as 'black measles'; it is excessively rare and can only be distinguished from hæmorrhagic smallpox with great difficulty. The eruption may in rare instances be altogether absent. The rash generally makes its first appearance on the temples close to the roots of the hair, and behind the ears just below the mastoid bones, and it is these regions which should be closely examined when the rash of measles is expected. It then invades the oral circle and spreads rapidly from the temples to the cheeks, then to the rest of the face, the neck, trunk, and limbs, and is completely out in from twenty-four to forty-eight hours; it remains distinct for about three days, and then declines in the same order. The spots gradually fade, leaving brown or yellowish stains, which in the course of a few days entirely disappear, but may be followed by a branny desquamation best seen on the hands and face, as elsewhere it is masked by the natural perspiration. Some degree of stomatitis is almost invariably present. During the period of invasion even as early as the second day examination of the buccal mucous membrane may reveal numerous small red spots with a more or less well-defined blush of the surrounding parts (Koplik's spots); and sometimes preliminary rashes may be observed on the skin consisting mostly of scattered minute papules which do not run together into blotches but soon disappear, and are apt to lead the inexperienced practitioner, who may have been suspicious of measles, to declare too prematurely that his patient is not suffering from that malady. The disorder is not more severe because the rash is plentiful, or because it appears early; nor on the other hand does delay in its appearance necessarily mean that there are or will be complications. In young children the rash may be ushered in with convulsions; diarrhoea too is apt to occur with the rash. The temperature which had been inclined to fall, or may even have reached the normal during the period of invasion, rises again when the rash appears, probably even to a higher point than it reached at the onset. During the height of the rash it maintains a high level, and may even rise on the fifth or sixth days, a temperature of 105° at this period being not very uncommon, and not necessarily implying the onset of any complication; but should the fever persist after the subsidence of the rash, some complication may be confidently predicted. The frequency of the pulse is generally proportionate to the height of the fever. The catarrhal symptoms gradually increase with the appearance of the rash, and attain their height when the exanthem does, subsiding as this fades; the sibilant sounds heard over the chest are replaced by moist râles which rapidly become less abundant; the cough loses its hard character and the wheezing disappears; while the conjunctivitis and coryza also pass off. The temperature generally reaches the normal about the eighth or ninth day, and the catarrhal symptoms have usually subsided.

completely by the fourteenth. It is generally a mild affection, but all degrees of severity may be met with, and it must always be regarded as serious when it attacks a child who is already the subject of tuberculosis.

COMPLICATIONS.—Young children of neurotic tendency are very likely to have convulsions, but unless these recur frequently they need not cause alarm. Diarrhœa is common and generally unimportant, but may be so severe as to be dangerous, and this is especially likely to be the case when the development of the rash is delayed; sometimes there is a true colitis (*see COLITIS*). Acute laryngitis is occasionally met with. Bronchitis and bronchopneumonia are the most common, and in young children the most dangerous complications; pleural effusions, serous and more often purulent, are by no means uncommon. Among other complications and sequelæ the following may be mentioned: ulceration of the cornea leading to a permanent opacity; otorrhœa; otitis interna, which may or may not lead to facial paralysis; meningitis; abscess of the brain; and thrombosis of the lateral or other sinuses of the brain (*see INSANITY IN SPECIAL DISEASES*). Enlarged and caseous mediastinal or mesenteric glands with subsequent development of more or less generalised tuberculosis are among the various ultimate results, while infantile cerebral or spinal paralysis are also well recognised as occasional sequelæ. Whooping-cough seems peculiarly liable to follow in the train of measles. It is often stated that in children who have suffered from measles or scarlet fever at or before the age of three years, examination of the permanent teeth will reveal either a transverse grooving of the enamel which may be shallow or deep, or one or more spots deficient in enamel arranged transversely; these changes are best seen in the incisors and canines, and are also present in the first molars and occasionally in the bicuspid. The condition is probably due to rickets.

PROGNOSIS.—As already implied, this is generally favourable, but many things must be taken into consideration, and notably the type of the epidemic, for the severity of the attack and the liability to the different complications vary greatly in different epidemics, and every complication of course increases the danger. As a general rule it may be said that the outlook is favourable when the chest-symptoms are moderate in degree; when the exanthem is not ushered in by any great rise of temperature; when the temperature does not show much tendency to rise afterwards, and when there is not much prostration. The prognosis should be more guarded and perhaps unfavourable when the disease occurs in very young subjects, or in delicate children, especially if there is any well-marked tendency to tuberculosis, when the chest-symptoms are severe, when the rash is of a livid colour or becomes petechial; and when convulsions or any other complications supervene. There is additional danger when measles follows upon scarlet fever or diphtheria. *See MORTALITY*.

DIAGNOSIS.—Before the appearance of the rash a positive diagnosis is rarely possible except during an epidemic when the co-existence of fever with the coryza and other catarrhal symptoms would be highly suggestive, and if supported by the discovery of the characteristic eruption on the buccal mucous membrane the suspicion would be converted into a certainty. The rash may have to be differentiated

from those of scarlet fever, variola, and rubella. In regard to *scarlet fever* there should be no confusion; in this the rash is much finer, punctiform, and more evenly diffused, producing uniform redness; and there is an entire absence of the catarrhal symptoms. In *smallpox* at an early stage the difficulty may be very great, and it is sometimes necessary to wait a few hours and examine the patient again before a positive opinion can be safely reached: the points in favour of smallpox would be the history of pain in the back, the shotty feel of the papules, and the fall of temperature with the development of the rash. The eruption in *rubella* closely simulates that of measles; the temperature is, however, as a rule but slightly raised, and prodromal symptoms are slight or altogether wanting, though catarrhal symptoms may develop with the rash, and the tonsils are sometimes much swollen and more or less covered with mucus; the superficial lymphatic glands at the back of the neck, beneath the sterno-mastoid, in the axillæ and in the groins are enlarged and hard; this is a very characteristic sign and an early one, and has been recognised even a week before the eruption.

TREATMENT.—In a case of uncomplicated measles good nursing is alone necessary, the greatest possible care being taken to avoid chill. When there are other children in the family, the attempt should always be made to completely isolate the sick child, and the popular theory that the others had better take the disease and have done with it should receive no practical support from the medical practitioner; but as a matter of fact, inasmuch as the children have been together during the whole period of incubation and often during the best part of the period of invasion as well, the mischief is done before isolation can be attempted, and it is a not uncommon experience, where there are three children in the family, to find the second child develop measles about twelve days after the first, and the third after the expiry of another similar interval. The quarantine-period after a child has been exposed to measles before he is allowed to go back to school or mix with other children is sixteen days, disinfection of the child and his clothes having been carried out immediately after exposure and again at the termination of the quarantine (*see INCUBATION*). A child who has had measles may go home or go back to school in not less than three weeks from the date of the appearance of the rash, provided that all catarrhal symptoms are over and that his clothes have been thoroughly disinfected—the latter by baking or superheated steam after dipping in carbolic acid. The personal disinfection of the patient involves being washed from head to foot in a warm bath with carbolic-acid soap, especial attention being paid to the scalp and hair. *See DISINFECTION*; and *PUBLIC HEALTH*.

JOHN ABERCROMBIE.

MEAT, Poisoning by. — *See FOOD, POISONOUS*.

MECKEL'S DIVERTICULUM.—*See INTES-TINES, Diseases of*; and *INTESTINAL OBSTRUCTION*.

MEDIASTINUM, Diseases of.—The term mediastinum is applied to the central region within the thorax which is bounded by the mesial folds of the pleura. It thus contains the heart, pericardium and great vessels, the vagus, phrenic and cardiac

nerves and plexuses, the thymus gland or its remains, the trachea, œsophagus and thoracic duct, certain lymphatic glands and vessels, and the connective tissue in which these structures are embedded. All diseases affecting these various structures might thus be described as diseases of the mediastinum, but the affections of the different organs are described elsewhere in the special articles dealing with them respectively. In the present article reference will be made only to the following conditions: (1) acute and (2) chronic inflammation of the mediastinal connective tissue, and (3) tumours growing within the thorax, including those arising primarily in the lung. For hernia occurring into the mediastinum, reference may be made to pp. 386 and 682.

1. Acute Suppurative Inflammation. — SYNON.: Abscess of the Mediastinum.

ÆTIOLOGY AND PATHOLOGY.—It is probable that occasionally inflammation of the connective tissue of the mediastinum may run its course without suppuration, but of this condition little is known. In cases of death from pleurisy and pericarditis there may be found at the necropsy some extension of the inflammatory process to the tissues of the mediastinum, and it is probable that, in patients who recover, such an extension may have taken place, the mediastinal inflammation subsiding along with that of the serous cavity. In some cases acute mediastinitis may perhaps pass into a chronic condition, giving rise to the mediastinal fibrosis treated of in the next section.

The two commonest causes of mediastinal abscess are direct traumatism in the form of blows upon the chest or penetrating wounds, and acute inflammation, septic or tubercular, of the lymphatic glands of this region. Inflammation may spread from above, as from a tracheotomy-wound, a retropharyngeal abscess or cellulitis of the neck. Disease of the sternum may result in abscess-formation in the connective tissue behind this bone, and disease of the vertebræ may similarly involve the posterior mediastinum. Erysipelas, pyæmia and acute specific fevers may occasionally give rise to mediastinal abscess; and suppuration in the thymus gland may occur without clearly assigned cause. Perforation of the œsophagus by a foreign body or malignant growth, or rupture of a diverticulum from this canal, may cause suppuration in the posterior mediastinum. Apart from these last cases, the anterior portion of the region is the most common seat of abscess. When pus has once formed it is liable to infect the pericardial or pleural cavities; or it may burst into the œsophagus, trachea, bronchus or lung; or 'point' externally upon the thoracic wall.

CLINICAL PHENOMENA.—The presence of suppuration in the mediastinum may not give rise to any very characteristic symptoms or physical signs. The general phenomena accompanying suppuration in any part of the body will be present, viz. pyrexia of a hectic character, sweating, and chilliness or actual rigors. In some cases there is pain referred to the region behind the sternum or to the back, and there may be some tenderness on percussion over the affected area. If the collection be of any size symptoms of pressure may arise, such as cough, stridor, and dyspnoea; the patient may be unable to lie on one or other side, or continuous orthopnoea may be present. If the anterior mediastinum be the seat of the abscess there may be fullness over

the region of the sternum, and dulness upon percussion over this region. A localised swelling, with œdema and redness, may appear in one of the intercostal spaces, and this may even pulsate owing to the contiguity of the collection of pus to the heart or aorta. In other cases 'pointing' of the abscess may occur at the root of the neck above the manubrium sterni.

DIAGNOSIS AND TREATMENT.—In presence of bone-disease or other cause of inflammation liable to extend to the mediastinum symptoms of pressure within the chest may lead to a suspicion of this condition, otherwise the recognition of mediastinal suppuration is difficult or impossible. The appearance of a fluctuating swelling, due to this cause, upon the chest-wall could not be distinguished with any certainty from the pointing of a localised empyema. The possibility of confusion of a pulsating abscess with an aneurysm should be borne in mind. The ideal treatment of suppuration in the mediastinum, as elsewhere, is evacuation of the pus by surgical measures. When the abscess is situated in the anterior mediastinum these should not present any insuperable difficulties. The sternum might be trephined if necessary to secure an efficient opening. Even in the posterior mediastinum, if the existence of an abscess were certain and the presence of malignant disease could be excluded, it is probable that an attempt should be made to evacuate the pus. If there be a probability only of the existence of suppuration, without definite signs of its occurrence, hot fomentations to the chest may relieve pain, the previous application of leeches also being advantageous as a temporary measure.

2. Chronic Inflammation. —SYNON.: Chronic mediastinitis; Indurative mediastino-pericarditis.

ÆTIOLOGY.—Of the causation of this condition nothing is certainly known. A chronic fibrosis such as is here found must be the result of the presence of an irritant of low intensity acting over a considerable period of time. It is possible that in some cases a rheumatic pericarditis may be the starting-point of the trouble, the inflammation spreading from the serous sac to the surrounding connective tissue. Minor degrees of such 'internal and external pericarditis' are not infrequently met with, without any considerable involvement of the mediastinal areolar tissue. In other and more numerous cases it seems probable that the lymphatic glands of the mediastinum, especially the bronchial glands, are the part first affected, the periadenitis associated with the glandular inflammation extending as a cellulitis to the surrounding regions. Thus cases appear to owe their origin to measles or whooping-cough, which are known to be associated with glandular enlargement. To this cause are probably due the instances in which the mediastinum suffers alone without the pericardium, and it is not impossible that the pericarditis may in other cases be secondary to the inflammation outside the sac. A certain proportion of the cases are probably tubercular in origin. The possibility of syphilis, congenital or acquired, as a causal agent may be suggested. Males are affected far more frequently than females. Thus, of 40 recorded cases 33 were males and only 7 females. It is probable that the affection usually originates in childhood, but that the rate of its progress to a fatal issue is very various, many of the cases dying in childhood or youth, others in early adult life, and a few reaching middle or advanced age.

PATHOLOGY.—The lesion consists in the formation throughout the mediastinum of a dense mass of fibrous tissue, which binds the contents firmly to one another and to the walls of the chest. The mediastinal affection is generally associated with pericarditis (*mediastino-pericarditis*), and often with pleurisy of one side or both. In course of time as the fibrous tissue contracts various morbid effects are produced. The organ most notably affected is the heart. In well-marked cases the two layers of the pericardium are firmly united, and the outer layer is also adherent to the back of the sternum. The heart is thus much hampered in its action, while at the same time the tight coat of fibrous tissue in which it is enclosed prevents it from undergoing the enlargement necessary to compensate for the increased work thrown upon it. Failure of the heart consequently results. The organ is often found *post mortem* to be rather smaller than normal, and the walls may have undergone fatty or fibrous change. The veins traversing the mediastinum are liable to injurious constriction, and thrombosis may take place within them. The liver is much enlarged, partly owing to failure of the heart, partly perhaps to constriction of the inferior vena cava at its entrance into the pericardium. It is also frequently the seat of a process of cirrhosis, the cause of which is not clear, but which may be in part due to the chronic venous congestion, and in part to extension of inflammation from the overlying peritoneum (perihepatitis). Owing to these causes ascites is a marked feature, preceding and being out of proportion to the general cedema which occurs towards the end. The pleuræ may be the seat of considerable effusion, either of an inflammatory or a dropsical character. Collapse of the lung may result. Pulmonary emphysema is not an infrequent feature in these cases, and infarction may also be found *post mortem*. The kidneys may be the seat of interstitial nephritis, or may suffer merely from congestion due to cardiac failure.

In one case which occurred in Charing Cross Hospital it appeared that an acute attack of inflammation had supervened upon the chronic condition. Thus, in addition to the fibrosis of the mediastinum and obliteration of the greater part of the pericardial cavity, there was found a collection of pus in the remaining pocket of the pericardium, recent pleurisy and acute inflammation of the bronchial glands.

CLINICAL PHENOMENA.—The onset of the disease is insidious and the condition cannot be recognised until the process of mediastinal fibrosis is far advanced. Suspicion may first be aroused by the existence in a child or young person, in whom alcoholism is improbable, of great enlargement of the liver and some degree of ascites; or the symptoms may consist of breathlessness and signs of heart-failure without signs of valvular disease. Examination of the chest may reveal an enlarged area of præcordial dulness, extending upwards behind the sternum; but this may be masked by the existence of emphysema. The special signs which may be produced by the pericardial adhesions are displacement outwards of the apex-beat without the presence of murmurs which would account for cardiac dilatation, fixity of the position of the apex-beat when the patient changes his posture, retraction of the chest-wall with systole, and the existence of a pulse which remits with inspiration (pulsus

paradoxus). Venous fulness and pulsation may be seen in the neck. As the case proceeds swelling of the legs occurs, and later on general anasarca, the face and chest being involved. Effusion may occur into the pleural cavities. Dyspnoea and cyanosis become prominent features, and death occurs from exhaustion. The urine towards the end is scanty and albuminous. Life is not likely to be prolonged much beyond eighteen months after the signs of the condition are fully developed.

DIAGNOSIS.—Great enlargement of the liver in a young person along with some ascites and dyspnoea, and apart from obvious causes of cardiac failure or hepatic disease, may cause a suspicion of the existence of mediastinitis; the existence of an enlarged area of præcordial dulness, if present, would be a further indication of this condition, but its absence does not exclude mediastinal fibrosis. The signs peculiar to the condition of external and internal adhesion of the pericardium should also be sought for; and Harris lays stress upon inspiratory filling of the veins in the neck as of importance if present. The progressive nature of the symptoms and the occurrence of general dropsy and pleural effusion will confirm the diagnosis.

TREATMENT.—Nothing can be done to arrest the process of fibrosis in the mediastinum, and treatment must therefore be directed merely to the relief of symptoms. The patient need not be kept in bed, apart from the occurrence of dyspnoea and palpitation, but care must be taken against the occurrence of chill. Any bronchitis present must be treated on ordinary lines, and cardiac failure may be met by digitalis, nux vomica, and ammonia. The ascitic and pleural effusions must be drained as they accumulate, and incisions made in the legs may give considerable temporary relief when general cedema is present.

3. Tumours of the Mediastinum, and of other intrathoracic structures.—**VARIETIES.**—The most common tumours which originate primarily in the mediastinum are lymphosarcomata, starting in the lymphatic glands. These are generally soft rapidly growing tumours, composed of small round cells with a scanty intercellular fibrous stroma. They often reach a large size, and at the time of death may weigh several pounds. In rarer instances the connective tissue is a larger amount, and the growths are firm, tough, and fibrous. Fibrosarcoma (spindle-celled) may also occur, and simple fibroma. Primary carcinoma cannot arise apart from growths originating in the epithelium of the special organs contained within the thorax (œsophagus, thymus gland, bronchi, &c.), but alveolar sarcoma may be found, indistinguishable microscopically from true cancer. Chondromata may grow from the walls of the chest, and mixed tumours containing cartilage may originate in the thymus gland. Malignant tumours of the lung, œsophagus, trachea, thymus, and bones may spread to the mediastinum; and secondary deposits may occur there in cases of malignant growths elsewhere, especially carcinoma of the mamma. Sub-pleural fibromata and fibrosarcomata, as they enlarge, encroach on the area of the mediastinum.

Aneurysm of the aorta or great vessels, hydatid cysts, and enlargements of the lymphatic glands, due to tuberculosis, syphilis, or lymphadenoma, are sometimes classed as tumours of the mediastinum, since they give rise to similar phenomena of pressure; but these conditions are not treated of in the

present article. See AORTA, Aneurysm of; BRONCHIAL GLANDS, Diseases of.

New-growths occurring in the lung.—The commonest primary growth met with in the lung is carcinoma, originating in the epithelium of the bronchi or alveoli. It is generally of the soft variety, known as encephaloid; and secondary growths occur in the bronchial glands and along the lymphatics. The growth may form a mass weighing several pounds. Many of the connective-tissue tumours may be found in the lungs, but these are small, rare, and innocent. Secondary deposits in the lungs are most frequently multiple and bilateral. Sarcomata are the commonest source of such growths, but carcinomata may also give rise to metastatic deposits in these organs, and the uterine tumour known as deciduoma malignum not infrequently does so. Hæmorrhage often occurs into such nodules; or if they are slow in growth cavities may form by breaking down of their substance. Endotheliomata may affect the pleura primarily, giving rise as a rule to a number of well-defined white fibrous thickenings. Growths which originate in the pleura, or involve it by extension or metastasis, are generally accompanied by pleural exudation; this is often hæmorrhagic in character. Secondary growths frequently escape observation when they start at the root of the lung, and gradually invade the organ along the course of the bronchial tubes and vessels.

EFFECTS AND CLINICAL PHENOMENA.—The most important effects produced by tumours growing within the mediastinum are due to the pressure exerted upon the important structures in the neighbourhood; but the extent to which this occurs varies considerably with different kinds of tumour. Thus a firm, well-defined growth, even if quite small, will tend to produce serious pressure-effects, while a soft infiltrating neoplasm may reach a large size before such a result occurs, since it insinuates itself between other structures without compressing them.

The large *veins* within the thorax are often early affected, and thrombosis may occur within them as a result of slowing of the blood-current. Their walls are sometimes invaded by malignant tumours, especially carcinomata. *Edema* results in the parts drained by the affected vein, and is thus frequently met with in one or other upper extremity, in the neck and face, and on the thoracic walls. Enlargement of subcutaneous veins may occur in the course of the establishment of a collateral circulation; and this is most evident when the superior vena cava is seriously compressed, the blood being then compelled to find its way to the heart through the inferior vena cava. The superior epigastric veins and anterior branches of the lumbar veins become enlarged and tortuous; and by pressure upon different parts of these vessels it is possible to ascertain that the current of blood within them is flowing from above downwards.

Serious pressure upon the *arteries* within the thorax is not very frequently met with, but rarely an artery may be perforated by a malignant growth, and occasionally abnormal bending of one of these vessels may interfere with the current of blood within it. Alteration in the size and force of one of the radial pulses may thus occur.

The *heart* may be considerably displaced by a large new-growth, and serious embarrassment of its action may thus be produced. A murmur may

sometimes result from such displacement. The extent to which this organ is displaced is not, however, necessarily proportionate to the size of the tumour. A small growth, situated at the base of the lung, may push the heart noticeably out of its normal position, whereas a large infiltrating tumour may cause little or no alteration in the relation of the heart to the chest-wall. The pericardium may be penetrated by an advancing tumour, and pericardial effusion result; even the walls of the heart may be invaded in rare instances.

The *œsophagus* may be compressed by a tumour suitably situated, and dysphagia may result. This, however, is a very uncommon occurrence; and if difficulty in swallowing is an early and prominent symptom, it is almost certainly due to the presence of primary cancer of the œsophagus. Pressure upon the *thoracic duct* may cause dilatation of the receptaculum chyli, and some escape of chyle from the lacteals into the peritoneal cavity.

Pressure upon the *trachea* or *bronchi* is an important effect of intrathoracic growths, resulting in shortness of breath, and frequently in the occurrence of noisy respiration (stridor). The latter may be either audible at a distance from the patient, or only discoverable upon auscultation of the chest. *Dyspnoea*, due to direct pressure upon the air-passages, is generally continuous; its origin is gradual, and toward the end of the case extreme distress may be produced. The rate of respiration is generally increased. Deficient entry of air into some portion of the lung, or collapse of this area, may result from the pressure, and actual broncho-pneumonia may occur. The trachea may be displaced to one side by tumours growing at the upper part of the thorax. *Dyspnoea* may also result from pressure upon nerves supplying the bronchi or from cardiac disturbance. In such cases it is generally paroxysmal, the attacks resembling those of spasmodic asthma. *Dyspnoea* is often increased by the existence of pleural effusion and by displacement of the heart.

Pain may be produced by pressure upon *sensory nerves*; and this may be referred to the thorax beneath the sternum or to the back, or may radiate round the chest and down the arm. It is generally neuralgic in character, but a constant boring or aching pain may occur in cases where erosion of bone is taking place. This is not frequent, but may be met with in cases of carcinoma. Tenderness of the thoracic wall is not infrequently present. Of *motor nerves*, the recurrent laryngeal is most often paralysed, resulting in loss of movement in one vocal cord, with hoarseness or actual aphonia, and the peculiar ineffectual cough characteristic of laryngeal paralysis. The phrenic nerve may also be interfered with, and immobility of half the diaphragm may result, or in rare cases hiccup due to irritation of the nerve. Pressure upon the sympathetic chain may cause alteration in the size of one pupil; and involvement of the vagus may induce attacks of cardiac disturbance, and also be associated with pneumonic changes in the lungs, considered by some authorities to be of a 'trophic' nature. Very exceptionally a malignant growth may invade the spinal canal and produce symptoms of pressure upon the cord.

Cough is almost invariably present at some stage of the affection, and often becomes progressively more troublesome; but the prominence of this symptom differs in individual instances. In cases of tumours of the lung, and in presence of much

co-existent bronchitis or of pressure upon the air-passages, cough is constant throughout; in the last case it is often stridulous in character. If it be due to irritation of nerve-trunks it is paroxysmal and of a 'brassy' quality: it may be to some extent proportionate to the size of the tumour. There is not usually much *expectoration*, apart from the presence of tuberculosis of the lung, as a complication, or of bronchitis. Copious expectoration is suggestive of a primary growth in the lung. As the case advances *cytosis* is usually a marked symptom, and this, along with the œdema of the neck, face, and upper part of the thorax, may give the patient a very characteristic appearance. A peculiar variable or intermittent *temperature* is sometimes observed, attacks of pyrexia occurring either irregularly or at more or less constant intervals of a few days. Fever may, however, be absent throughout the illness.

The phenomena discoverable upon physical examination of the chest, in addition to those already mentioned, vary with the size and position of the tumour. Alterations may occur in some cases in the shape of the thorax, which may show a local bulging over the growth, the sternum or rib-cartilages being pushed forward, or a depressed area due to collapse of some portion of the lung. The growth may invade the chest-wall and project beneath the skin. In such circumstances pulsation may sometimes be observed if it be very vascular, as in some forms of sarcoma. Dulness on percussion will be found over the tumour when this is directly beneath the thoracic wall, and in the case of tumours growing in close relation with the bronchi, tubular breathing will be audible in the dull area; but if the air-passages be occluded there will be deficient movement with absence of vocal and respiratory sounds over the affected portion of lung. Enlarged glands are frequently discoverable above the clavicles in cases of malignant intrathoracic tumours. The liver and spleen may be displaced downwards into the abdomen by the pressure of very large growths, and the movement of one or other of these organs and of the abdominal walls in respiration may be defective in case of paralysis of the diaphragm.

In the case of *tumours of the lung* itself, there is not often any bulging of the chest-wall: with slowly growing tumours there may even be retraction of the chest, especially if pressure upon the air-passages has led to collapse of part of the lung. Respiratory movement is usually defective over the affected area. The dulness found upon percussion is absolute in degree—of the character often described as 'wooden'—and accompanied by marked increase in resistance. Its distribution is irregular: extension across the middle line of the sternum is a valuable diagnostic sign of the existence of a tumour. The co-existence of pleural effusion may in some cases complicate the diagnosis. Vocal fremitus may be normal or increased; if a large bronchus be compressed it may be entirely absent. Breath-sounds are frequently absent over a tumour of the lung, but bronchial breathing may be found. If degenerative changes be taking place in the growth, or if there be accompanying bronchitis, râles may be audible. Stridor is not so likely to occur as in tumour of the mediastinum; and dyspnoea is seldom so intense as in that condition.

There is generally slight continuous pain, accompanied by loss of flesh, anæmia, and weak-

ness. Cough is generally present, sometimes from an early period; and the sputa may be hæmorrhagic: they are not infrequently of a consistency and appearance which have been compared to 'red-currant jelly.' Profuse hæmoptysis sometimes occurs.

No *age* and neither *sex* is free from liability to mediastinal tumours, but they are more common in males than in females, and primary growths are most frequently met with in early adult life. The duration of the disease varies with the position and rate of growth of the tumour. In cases of 'simple' growths, two years may perhaps be taken as an average duration, but much longer cases are recorded. Malignant growths are usually much more rapidly fatal. Death occurs either by increasing dyspnoea and exhaustion, by hæmorrhage, due to perforation of a vessel or by implication of the heart and pericardium. Secondary growths may occur in other organs, such as the brain, and prove fatal; in one case, seen by the writers, the symptoms first complained of were those of paralysis, due to secondary growths in the brain, the phenomena due to the primary intrathoracic tumour only developing later.

DIAGNOSIS.—It is necessary not only to recognise the existence of a cause of abnormal pressure within the thorax, but also to distinguish true new-growths from aneurysms and glandular swellings, which may produce similar effects. By far the most common cause of intrathoracic pressure is aneurysm of the aorta, and the possibility of its presence should be carefully eliminated before arriving at a diagnosis of mediastinal tumour. Aneurysm is far commoner in males than in females, and occurs chiefly in adult life (35–50 years), and in those who have followed laborious occupations: on the other hand, new-growths occur in much younger subjects, and the proportion of women affected is relatively much larger. The position of an aneurysm is necessarily limited to the course of the great vessels, and the area of dulness produced by it upon the thoracic wall is not often large, whereas new-growths may occur at any part of the thorax, and may cause very extensive impairment of resonance. It is not often that tumours cause marked local bulging of the chest, nor do they frequently pulsate to an extent really comparable with that of aneurysms. The growth of a malignant mediastinal tumour is much more rapid than that of an aneurysm; and the occurrence of secondary deposits and enlarged glands may place the existence of the former beyond doubt. The pressure produced by a tumour is much more frequently exerted upon the venous trunks of the mediastinum than is that of an aneurysm, while, on the other hand, the œsophagus is much less frequently affected. Affection of the right recurrent laryngeal nerve and right sympathetic chain could hardly be produced by an aortic aneurysm; aneurysm of the innominate artery may, however, involve these structures. Dyspnoea is on the whole less frequent in cases of aneurysm than in those of new-growths; and the latter are generally accompanied by anæmia and general constitutional disturbance, not met with in the vascular disease. Irregular pyrexia is in favour of mediastinal tumour, but may be met with in cases of tubercular or lymphadenomatous enlargement of mediastinal glands. It is rare, however, for these to be primarily affected in Hodgkin's disease, the glands in the neck, and often those in the

groin and elsewhere, being enlarged in these patients. Tuberculous glands are not likely to cause symptoms of pressure except in young children (see BRONCHIAL GLANDS, Diseases of). The use of the X-rays is often of considerable service in diagnosis. If the patient's chest be examined with the fluorescent screen, a tumour will produce as dense a shadow as an aneurysm, but the latter can be distinguished by its pulsation and to some extent by its position and shape. It is difficult, however, to distinguish infiltrating growths and scattered nodules from tubercular masses. Fluid in the pleural cavity, and still more a thickened pleura, will obstruct the X-rays, but never to so great an extent as a tumour; the latter also has a more distinct edge than any pleural condition except a localised empyema, which is not likely to give rise to a difficulty of diagnosis. See RÖNTGEN-RAYS.

Tumours originating in the lung and giving rise to no pressure-symptoms have to be distinguished from tuberculosis. In addition to the use of the X-rays just alluded to, there is generally absence of fever, and of tubercle-bacilli from the sputum. The progress of the disease is rapid and continuous, the physical signs extend over a large area, and enlargement of superficial lymphatic glands occurs. Physical signs resembling pleural effusion but with some marked inconsistency and with absence of fluid on exploratory puncture are in favour of the presence of a new-growth. Should actual pleural effusion be present a hæmorrhagic character is in favour of tumour involving the pleura: masses of large swollen endothelial cells may be found in some cases upon microscopical examination of the fluid. The possibility of the co-existence of tubercular disease of the lung with a tumour of the mediastinum, as well as with aneurysm, must be borne in mind, since otherwise the conjunction of symptoms produced may be puzzling.

PROGNOSIS.—The outlook is probably hopeless in all cases in which it is possible to be certain of the existence of a tumour within the thorax. The duration of life will depend upon the nature of the growth and its relation to important intrathoracic structures.

TREATMENT.—Since any growth within the chest is beyond the reach of surgical intervention, all that can be attempted is the relief of distressing symptoms as they arise. Even this object can, unfortunately, be only imperfectly attained. Opium or morphine may be administered without hesitation in view of the necessarily fatal termination of the disease, and much relief may thus be obtained, not only of the pain caused by the growth, but also of the cough, which is frequently both distressing and ineffectual. There is no advantage to be gained by keeping these patients in bed, at least in the earlier stages of the disease: the feelings of the patient are the best guide to follow. Later, as dyspnoea becomes more urgent, rest in bed will become compulsory, and the upright position with a bed-rest or additional pillows is generally found the easiest. Tracheotomy can hardly ever be advisable, however urgent the dyspnoea, since the obstruction is below any point which can be reached; in a very few cases associated with laryngeal spasm it may afford relief. If extreme dysphagia result from pressure upon the œsophagus, the question of the performance of gastrostomy will arise. Authorities differ as to the advisability of removing pleural effusion when it occurs. In many cases its presence

seriously adds to the difficulty in breathing, and there is no valid reason for not removing it by aspiration, an operation which may afford great temporary relief to the sufferer, and may be repeated as often as the fluid reaccumulates. Inhalations of oxygen may be called for if cyanosis becomes a prominent symptom, due to pressure upon the respiratory apparatus. Careful nursing is requisite in protracted cases to avoid the formation of bed-sores.

H. MONTAGUE MURRAY.

W. CECIL BOSANQUET.

MEDITERRANEAN FEVER.—See MALTA FEVER.

MEDITERRANEAN SEA, The.—A moderately dry, warm, and very sunny winter climate. See ALGIERS; CANNES; HYÈRES; MALAGA; MENTONE; NICE; SAN REMO, &c.; and CLIMATE, Treatment of Disease by.

MEDULLA OBLONGATA, Lesions of. SYNON.: Fr. *Maladies de la Moelle Allongée*; Ger. *Krankheiten des verlängerten Marks*.

INTRODUCTION.—The pathology of the medulla oblongata is more than usually complex. Not merely is it liable to injuries, and diseases such as hæmorrhages, softenings—necrobiotic and inflammatory, tumours, &c., having their primary seat there, as in other nerve-centres; but also, and more frequently, the medulla is implicated in diseases of the pons and cerebellum, and affected indirectly by intracranial diseases in general. Being the connecting isthmus between the brain and spinal cord, it is subject to ascending or descending degenerative processes, secondary to lesions in the spinal or cerebral sensory and motor tracts. Further, it is the seat of a chronic form of degeneration, characterised by a very definite group of symptoms, differentiated under the term 'bulbar or labio-glosso-laryngeal paralysis.'

With the indirect affections of the medulla oblongata, in connection with the various forms of intracranial disease, degenerations of the motor or sensory tracts secondary to cerebral or spinal disease, or the pathology and symptomatology of bulbar paralysis, this article does not profess to deal, as these subjects will be found fully discussed under their respective headings. Attention will be directed mainly to the data which serve to establish, so far as this is possible, the regional diagnosis of medullary lesions.

SUMMARY OF PATHOLOGICAL CONDITIONS.—*Traumatic lesions*.—Injuries of the medulla oblongata are not uncommon in consequence of fracture or dislocation of the atlas and axis, as in falls, hanging, twisting of the neck, or as the result of diseased vertebræ. In such cases death is instantaneous, owing to the sudden cessation of the circulation and respiration, from lesion of the centres for these vital functions, which are situated in the medulla.

To commotion or contusion, with punctiform extravasations in the medullary centres, Duret attributed sudden death from blows on the head. Not infrequently lesions of the fourth ventricle, the result of cranial injuries, not proving fatal, give rise to diabetes mellitus or insipidus, along with other symptoms indicative of chronic lesion of the pons or medulla.

Effusions of blood into the fourth ventricle,

whether arising from the medulla itself, the pons, or the cerebellum, or gaining access from the lateral ventricles by the aqueduct of Sylvius, are, as a rule, suddenly fatal from paralysis of the circulation and respiration. Death may occur with or without convulsions.

Tumours.—Tumours implicating the medulla oblongata may have their seat primarily in the medulla; but more commonly the growths are situated at the base of the skull, in the cerebellum or pons, and invade the medulla in their growth. Besides the special localising symptoms of medullary disease, there are general symptoms of intracranial new-growth; of these vomiting is usually pronounced, and optic neuritis rare. Here also, however, some remarkable cases have been put on record, in which, notwithstanding the existence of tumours actually in the substance of the medulla itself, the symptoms during life have presented nothing striking or characteristic.

Hæmorrhage.—Hæmorrhage into the substance of the medulla oblongata, and limited to this, is comparatively rare. More commonly the pons and medulla are affected together. Hæmorrhages here of any extent are very rapidly fatal. In some cases death is instantaneous. In others a few hours may elapse, death occurring in profound coma with stertorous respiration and occasionally convulsions. Whether the hæmorrhage is primarily in the medulla or in the pons cannot be diagnosed with certainty. The other causes of sudden death, such as affections of the heart, must be excluded before hæmorrhage into the medulla can be diagnosed, and this is in many circumstances obviously impossible.

Hæmorrhage into the medulla oblongata is usually fatal, and rarely gives rise to chronic stationary lesions. These are commonly the result of thrombosis or embolism, or, more rarely, acute myelitis.

Thrombosis.—Thrombosis of the vertebral arteries and their branches is the most common origin of softening limited to the medulla oblongata. The onset is frequently sudden, as in hæmorrhage, but the course is more slow. The more chronic nature of the affection is an important diagnostic feature of softening. The symptoms of softening of the medulla thus arising are in many respects like those of progressive bulbar paralysis, but there are also important differences. They are sometimes generalised under the head of 'acute' or 'apoplectic-form' bulbar paralysis, in contra-distinction to the chronic degenerative form of this affection described by Duchenne. See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

LOCALISING PHENOMENA.—The symptoms met with in the affection just named are the most reliable clinical data on which to found a regional diagnosis of lesions of the medulla oblongata. The characteristic symptoms are a conjoint affection of the extremities and one or more of the bulbar cranial nerves, with impairment of speech and deglutition, and cardio-respiratory disturbances. Sometimes all four extremities are paretic or paralysed; sometimes the lower extremities alone; and occasionally the paralysis is of the hemiplegic order. If the paralysis affects only the extremities, without implication of the bulbar nerves, as sometimes occurs, a diagnosis of the medullary seat of the lesion cannot be made with certainty.

Of the cranial nerves the hypoglossal is most commonly involved. The symptoms are impaired

mobility of the tongue, with more or less pronounced dysarthria (see APHASIA). This is not absolutely characteristic of bulbar disease, however, as a similar affection of the hypoglossal may occur in disease of the pons. But a form of crossed paralysis, palsy of the limbs on one side and of the tongue on the opposite side, is probably pathognomonic of coarse disease within the medulla. The tongue and speech are rarely, if ever, so affected as in the classic or progressive bulbar paralysis, nor has the atrophy of the muscles of the tongue, with altered electrical reactions, been noted.

Of more importance as a diagnostic mark is dysphagia, or paralysis of deglutition. This, in the absence of general cerebral symptoms, points to affection of the medulla. Paralysis of the soft palate, on one or both sides, is also a frequent, if not constant, symptom. Occasionally also aphonia occurs, and, taken with the other symptoms, points conclusively to affection of the medulla oblongata.

Irregularity of the heart; acceleration or retardation of the pulse; and sighing and laboured respiration, often amounting to orthopnoea, in the absence of general cerebral symptoms, are also important indications of disease of the medulla oblongata. Among other symptoms have been noted coughing and vomiting, explicable by affection of the respiratory centres. Trismus has been mentioned by Joffroy as a characteristic symptom of acute bulbar paralysis, but Nothnagel, on good grounds, disputes the accuracy of this statement.

A case recorded by Glynn, where a tiny tumour (glioma) was found *post mortem* in the middle line of the medulla at the level of the calamus scriptorius, showed many of the above-mentioned symptoms, the most prominent being intense dyspnoea with paralysis of the diaphragm, vomiting, impaired movement of lips and tongue, dysphagia, loss of reflex action of palate, weakness in both arms and less in legs, and a trace of sugar in the urine.

Albuminuria and glycosuria have also been observed in connection with bulbar lesions, the latter more particularly after injuries affecting the floor of the fourth ventricle; but the occurrence of these symptoms in connection with acute bulbar paralysis requires further investigation, as they cannot as yet be regarded as constant.

An affection simulating disease of the medulla oblongata results from bilateral cortical or sub-cortical disease, giving rise to a condition of double hemiplegia. Such a bilateral lesion causes paralysis of articulation, and also true aphasia if the lesion is cortical, along with a greater or less degree of paralysis of the limbs. The diagnosis must depend on the truly volitional character of the paralysis in such cases, the reflex mechanism of deglutition being unimpaired. There will also be absence of affection of sensibility and of trophic degeneration of the muscles, and absence also of disturbances of the cardiac and respiratory rhythm. Defective comprehension of speech, and obvious aphasia—the movements of articulation not being absolutely paralysed—and also agraphia—the hand not being completely powerless—will differentiate cerebral from bulbar paralysis.

Lesions in the neighbourhood of the pyramidal decussation, though rare, have a characteristic symptomatology. This consists of anæsthesia over the distribution of the fifth cranial nerve on the side of the lesion, with paralysis of the limbs on the

same side, and analgesia of the body and limbs on the opposite side: an association which has been experimentally found to be due to destruction of the tubercle of Rolando.

D. FERRIER.

W. A. TURNER.

MEDULLA OF BONES, Diseases of.—

SYNON.: Fr. *Maladies de la Moelle des Os*; Ger. *Krankheiten des Knochenmarks*.—See BLOOD; Morbid Conditions of; BONE, Diseases of; LEUCOCYTHÆMIA; MOLLITIES OSSIUM; TUMOURS, &c.

MEDULLA SPINALIS, Diseases of.—See

SPINAL CORD, Diseases of.

MEDULLARY CANCER.—A synonym for encephaloid cancer. See CANCER.

MEGRIM.—SYNON.: Migraine; Sick Headache; Nervous Headache; Hemicrania (ἡμί, half; and κράνιον, the head); Fr. *Migraine*; Ger. *Migräne*.

DEFINITION.—Headache of a periodical character; generally ushered in by some premonitory symptoms; more or less unilateral; and frequently associated with nausea and bilious vomiting.

ÆTIOLOGY.—The chief predisposing causes of attacks of megrim are hereditary tendency; anæmia; a general want of tone in the system; and the nervous temperament. Among the exciting causes may be included all those of a depressing or exhausting nature, whether physical or mental, such as prolonged mental work, mental excitement, grief, anxiety, bodily fatigue, late hours, sexual excesses, breathing the impure air of a crowded room, and improper food.

SYMPTOMS.—This complaint seems to have two more or less well-defined stages, the headache being preceded for a variable period by certain disorders of sensation. In some persons the malady stops short here, and is not followed by headache; in others the headache appears to be developed without any premonitory symptoms, until careful inquiry reveals the contrary. The two stages therefore are, first, the stage of disordered sensation; second, the stage of headache, with other symptoms.

The most striking of the disordered sensations is a transient disturbance of vision which sometimes takes place. It commences with a wavy glimmering near the outside corner of the field of vision, and spreads all over the visual area with a zigzag outline, in a straight-lined angular pattern, and with or without lines of colour between the darker lines. Or it may commence by the appearance of a blind spot close to the centre of vision, which soon begins to spread, showing a serrated margin, and presenting a tremor or wavy glimmering in its interior. This condition is often associated with a feeling of chilliness, coldness of the hands and feet, or other symptoms; it may last from five to thirty minutes or longer, and then be succeeded by the stage of headache.

On the other hand, the headache may be, and in many individuals always is, developed without the ocular disturbance, but other sensations are substituted for it. The patient has a feeling of chilliness, and the feet are cold. There is mental depression, with a dread of impending evil; the patient is restless and uneasy; 'cannot quite tell,' as he says, 'what he would be at'; and has what is expressively called 'the fidgets.' This condition may continue half an hour or more, and then the

slight boring piercing pain is felt in the head, with which the aching begins; and the disorder runs its course, as will be presently described. In other cases, this feeling of depression or uneasiness lasts for several hours, the patient goes to bed, and in the early morning wakes with the headache fully developed.

The headache, when preceded by ocular disturbance, shows itself as follows: When the vibratory movement is at its height, a little aching is felt in the head, on the side *opposite* to that on which the glimmering first appeared; it is slight at first, but gradually increases in intensity. Some persons have said that the sensation was as though a point in the temple were being bored with a gimlet, and the gimlet slowly increasing in size. The pain gradually spreads from this point, which may be covered with the finger, and pressure upon which affords relief, first over one side of the head; and then, but not always, it extends to the other. As the headache increases, the ocular disturbance declines; nausea is felt, which increases with the headache; retching and vomiting occur, the latter sometimes, though rarely, giving relief; the head throbs; the slightest movement increases the pain, and any attempt to move from the recumbent posture increases the gastric uneasiness; the mouth feels clammy; the eyeballs ache, and are tender on pressure, one more so than the other; the pupils are rather contracted, and generally unequally so; and the patient lies apparently more dead than alive, his face pale, and the head hot. After a varying number of hours he is somewhat relieved by troubled sleep; he wakes up next morning, free perhaps from headache; but he is listless; his brain is weary; and he feels as if he had undergone a hard mental struggle. There may be now an interval of a few days, weeks, or years, before the disorder again shows itself.

The headache varies much in character, degree, and duration. In some persons the pain is not localised in any particular spot, but seems generally diffused over the head; others have not noticed that there is more pain on one side of the head than the other, or that the aching radiates from one painful spot, until their attention has been directed to the fact, and then they distinctly recognise it; others, again, have neither vomiting nor nausea; and lastly, the duration of the headache may be very short, or not extend over more than two or three hours, or this symptom may be entirely absent. The disorder may even stop short at the vibratory stage, the vision be restored, and no further inconvenience felt.

In a certain proportion of cases during the vibratory stage a tingling is felt in some portion of the body—the part is 'asleep.' Sometimes it is felt in one arm or in the side of the tongue, or on the side of the face, and it is on the same side as that on which the glimmering in the eye begins. Sometimes the hearing, speech, or memory is affected.

The age at which the attacks generally commence is from twelve to twenty-five. Females are more liable to them than males. After a certain period, with advancing age the attacks, as a rule, are less easily developed, and become much less frequent. They cease generally after fifty or sixty, and in women not uncommonly at the change of life.

PATHOLOGY.—Considerable diversity of opinion exists as to the nature of megrim. Formerly it was regarded as being dependent upon gastric or

hepatic derangement—a view, however, which now finds few supporters. Some pathologists hold it to be a form of neuralgia; but though it has a great resemblance to neuralgia, it ‘causes much greater disturbance of the sensorium, it spreads much more generally over the head, and is not infrequently accompanied by nausea and vomiting. After the attack there may be an intermission of weeks or months, and the attack itself runs a more uniform or continuous course’ (Lebert). The view which the writer has advanced is that the affection is to be referred to the sympathetic nervous system. If by fatigue, anxiety, or other depressing cause, the general tone of the body be lowered, and with it the regulating or inhibitory power of the cerebro-spinal over the sympathetic nervous system impaired, then uncontrolled action or excitement of one or more portions of the latter takes place, causing contraction of the blood-vessels under the influence of the affected portions, and so producing the disorders of sensation which precede the headache; this excitement is followed by exhaustion or paralysis of the sympathetic, and is associated (just as would be the case after section of the nerve) with dilatation of the vessels, and with headache. Edward Living, in his classical and exhaustive work on megrim, combats this view, and maintains that the phenomena are those of ‘a nerve-storm traversing more or less of the sensory tract from the optic thalami to the ganglia of the vagus, or else radiating in the same tract from a focus in the neighbourhood of the quadrigeminal bodies.’

TREATMENT.—By careful management very great relief can be afforded to the sufferers from this malady, not only by diminishing the intensity of the attacks, but also by considerably lengthening the intervals between them. We may consider separately the remedial measures to be employed (1) during the intervals between the attacks; (2) during the premonitory stage, or stage of disordered sensation; and (3) during the stage of headache.

1. *During the intervals between the attacks.*—It is to the treatment during this period that the greatest consideration must be given. The cause, if possible, must be discovered, and in a very large majority of cases careful inquiry will reveal the fact that a distinct cause does exist. Overwork, prolonged anxiety, over-fatigue, disappointed hopes or affections, sexual irregularities, defective eyesight, and impoverished nutrition of the body, are among the chief causes; and while these are in operation medicine will prove of little avail. Remove the cause, and then endeavour to brace up the bodily and nervous systems. The chief remedies for this purpose are the vegetable bitters, iron, strychnine, and cod-liver oil. But the success following their use very much depends upon the way in which they are administered. For a day or two after a headache the stomach and bowels may possibly be disordered, and not in a fit state to tolerate iron or cod-liver oil. This condition must be corrected, and for this purpose the simple vegetable bitters, such as gentian with small doses of henbane and some aromatic, may be of service; and if necessary one or two grains of blue pill, with four or five of compound rhubarb pill, may be given at night; but strong purgation must be avoided. Iron may then be given, either in the form of the ammonio-citrate alone, or combined with two or three grains of iodide of potassium; and according to circumstances fifteen or twenty minims of tincture of henbane, or

twenty or thirty minims of aromatic spirit of ammonia, may be added to each dose. Or the iron may be given in the form of the *Mistura Ferri Composita* of the *Pharmacopœia*; the mixture answering better, however, in some cases, without the myrrh. Strychnine is, in the writer's opinion, a very important remedial agent in many forms of this disorder, and may be given with the remedies previously mentioned in the form of *Liquor Strychninæ Hydrochloratis* or *Tinctura Nucis Vomicae*, or may be combined with infusion of quassia or calumba. Where iron is contra-indicated from any cause, or when it is not readily borne, the administration of nux vomica with quassia has seemed to act beneficially. In females with a distinct hysterical temperament nux vomica does not answer so well, and better results will be obtained by giving the vegetable bitters with ten-grain doses of bromide of potassium, and fifteen or twenty of tincture of henbane, twice or three times a day. As a rule, however, the bromide is of more use administered during the headache than in the intervals. Cod-liver oil often acts beneficially, especially when there is much nervous exhaustion. It may be given once a day immediately after breakfast, beginning with a small teaspoonful, and gradually increasing the quantity to a tablespoonful, but not beyond, unless in exceptional cases. If the bowels are constipated, five grains or so of the socotrine-aloes pill may be given at night; or if the constipation be habitual, five grains of the aloes-and-iron pill, given twice a day before meals, will generally induce greater regularity in the action of the bowels.

In some individuals megrim is associated with a strongly marked uric-acid diathesis, as shown by the fact that preceding or succeeding an attack the urine is strongly acid and loaded with lithates. In these cases fifteen or twenty grains of salicylic acid, or salicylate of sodium, twice a day, in conjunction with other remedies, to correct or neutralise the uric-acid formation, will often be of signal service.

Other remedies have been recommended, and are sometimes of service, especially arsenic and quinine.

In persons of feeble bodily power, rest is of the greatest importance, and it is often advisable that such patients should remain in bed at least twelve hours out of the twenty-four, and take their breakfast an hour and a half or two hours before rising in the morning. Whenever the headaches recur frequently, this rule should be enforced. In cases where borne, a tumblerful of new milk, to which two teaspoonfuls of brandy, rum, or whisky have been added, may be taken with advantage before breakfast, directly on waking in the morning.

The diet should be liberal; the food plain and easily digestible; and two or three glasses *per diem* of wine, beer, or porter may generally be taken with benefit, according to the habits of the patient. The more exercise the patient can take in the open air, without fatigue, the better.

2. *During the premonitory stage, or stage of disturbed sensation.*—In the forms attended by disturbance of vision, the longer this lasts the greater will be the headache, and we must endeavour therefore to shorten this stage as much as possible. Directly the glimmering appears the patient should lie down, with the head low; and if the glimmering be on the right or left of the field of vision, he should lie on the *opposite* side. Let him take at once some alcoholic stimulant, a glass of sherry, a tablespoonful of brandy diluted, or a glass of champagne. If alcoholic stimulants be objected to, or

if it be not advisable to recommend them, then a teaspoonful of sal volatile in water may be prescribed instead. If the patient be chilly, or his feet cold, the couch should be drawn near the fire, and a hot bottle applied to the feet. By these means the heart is enabled to drive the blood with greater force to the brain, and the duration of the vibratory movement is thereby materially lessened. After the glimmering has passed off, the patient should lie still for a time, so that it may not return. This injunction will only be necessary when the headache is slight; if it be severe, attended with much nausea or vomiting, the patient will be little disposed, or little able, to leave the recumbent position.

If, instead of the disturbance of vision preceding the headache, there be a feeling of depression or irritability, fidgets, and similar phenomena, the administration of such cerebro-spinal stimulants as henbane, valerian, asafetida, spirit of chloroform, or ether, will often cut short the attack. Fifteen or twenty drops of the tincture of henbane, with the same quantity of spirit of chloroform, will soothe the nervous irritability in the slighter forms, and may be repeated in three or four hours if necessary. If there be great mental depression, then valerian or asafetida should be tried. Half a drachm to a drachm of the ammoniated tincture of valerian, or the same quantity of the fetid spirit of ammonia, may be given. Sometimes Indian hemp is very useful, a quarter to half a grain of the extract in a pill, or five to ten drops of the tincture on a lump of sugar. As a rule, in such cases as these, alcoholic stimulants are not advisable at this stage. A small quantity will cause flushing, heaviness, and slight confusion of thought, without relieving the depression; and though the severe headache may be averted, alcoholic stimulants do not answer so well as the remedies previously mentioned.

3. *During the stage of headache.*—If the headache be slight, and the patient soon able to sit up, there is little to be done. A cup of coffee or tea, cheerful conversation, a walk, drive, or ride, may often help to remove the pain. If, however, the symptoms be severe, then the administration of further remedies is called for. The patient should keep perfectly still and quiet, with the room darkened; for every sound or sight causes pain, and the slightest movement is sufficient to produce gastric uneasiness. Sometimes free evacuation of the contents of the stomach, especially if it contain undigested food, is followed by relief; but, as a rule, it is better to try to relieve and check the vomiting. Iced soda-water, with or without two or three drops of diluted hydrocyanic acid or spirit of chloroform; cold tea; or the effervescing citrate of potassium with diluted hydrocyanic acid, may often afford marked relief. The headache may be lessened by applying cloths dipped in cold water or evaporating lotions to the head. If the extremities be cold, and the headache severe, a warm stimulating foot-bath can be tried, as soon as the nausea will allow the patient to sit up. If the attacks occur in the early part of the day, as soon as the pain has subsided it is generally better for the patient to sit up or move about, or even to take exercise in the open air. During the attack the appetite is diminished, even the idea of taking food provoking disgust. Still, after the nausea has passed away, a cup of soup, or some easily digested food, will often have a good effect in equalising the cerebral circulation, and in relieving the headache.

If the headache be severe, bromide of potassium is a remedy which will often prove of great service. It may be given in doses of fifteen or twenty grains, with fifteen or twenty minims of tincture of henbane, and to these may be added thirty or forty minims of the aromatic spirit of ammonia, in some cases with advantage. If necessary, the dose may be repeated after an interval of two hours or so. In some cases phenacetin or phenazone may be given with benefit in doses of seven to fifteen grains, with thirty minims of sal volatile, or acetanilide in five-grain doses, and repeated in an hour if necessary. In other cases, often of a gouty character, chloride of ammonium in doses of fifteen grains produces marked relief, and may be sometimes advantageously combined with spirit of chloroform and compound tincture of lavender. Guarana powder is a remedy which is used, often with happy results. The sick-headaches which it seems to relieve are those in which distinct premonitory symptoms usher in the attack, and particularly those preceded by disturbance of vision. It may be given in such cases in doses of fifteen grains, with the same quantity of sugar, and repeated in from half an hour to two hours. In those individuals, however, in whom the headache is developed suddenly, where the attacks come on without any or with very indefinite premonitory symptoms, guarana appears to have little effect.

As a rule, the use of purgatives in this stage is decidedly objectionable, but occasionally a saline purgative, at the commencement of an attack is indicated, and is of service.

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MEINBERG, in Lippe-Detmold, in Germany.—Mixed sulphurous saline and chalybeate waters, and mud-baths. See MINERAL WATERS.

MELÆNA (μέλας, black).—SYNON.: *Dysenteria Splenica*; Fr. *Melèna*; Ger. *Schwärze Ruhr*.

This term is used to denote black tar-like evacuations that are passed from the bowel. The colour and appearance are due to *altered* blood, and the expression is not properly applicable to simple hæmorrhage from the alimentary canal, when blood of a normal appearance is voided.

In order that the blood should have undergone the change which produces the characteristic evacuations, it must have been effused high up in the canal, and in some quantity, as well as retained for some time in the bowel. When hæmorrhage takes place in the lower part of the small intestine, or in the colon or rectum, the blood is passed in a scarcely altered state, or at most renders the feces dark, without producing the black viscid motions now referred to.

Blood that is passed into the stomach, or the upper part of the small intestine, is decomposed by the various secretions and intestinal contents with which it comes into contact, and either wholly or in part is finally discharged from the rectum as sulphide of iron. The tar-like consistency is very characteristic and is probably due to the admixture of mucus with the other products of the decomposed blood. Blood derived from the stomach is less likely to be mixed with ordinary fecal matter than when derived from the intestine, as in the former case the cause of the hæmorrhage generally necessitates abstinence from food. The feces may be blackened by iron, bismuth, and other agents, taken

as drugs, but they do not produce the viscid matter like semi-digested blood.

A form of melæna, with or without hæmatemesis, has been occasionally noticed in infants within a day or two after birth. The cause is obscure, but is probably associated in some way with the disturbance of the circulation determined by the ligation of the umbilical cord. Sometimes clean-punched ulcers have been found in the stomach or duodenum in such cases, but oftener there are no indications to be seen *post mortem*. The condition, which is very fatal, is probably allied to other hæmorrhagic states met with in the new-born, such as purpura.

Melæna is the mere expression of a condition brought about by many causes, and these have to be sought for and treated. See HÆMATEMESIS; INTERSTINES, Hæmorrhage from; and STOOLS.

W. H. ALLCHIN.

MELANÆMIA (μέλας, black; and αἷμα, the blood).—See BLOOD, Morbid Conditions of, p. 161.

MELANCHOLIA (μέλας, black; and χολή, bile).
SYNON.: Fr. *Lybénie*; *Mélanholie*; Ger. *Schwermuth*; *Melancholie*.

This name is now usually applied to a form of insanity characterised by great mental depression. The sufferer in this disorder feels his whole existence, mental and bodily, overwhelmed and oppressed by gloom, anxiety, and foreboding. At first it may be only a feeling which takes no definite shape, and there may be no delusions. Sometimes, though rarely, there are none throughout; the morbid feeling constitutes the disorder, which in this form has been called *simple melancholia*. Its access is almost always gradual, and though we may attribute it to grief, overwork, or worry, it often happens that no mental or moral cause can be found, and we are obliged to set it down to inherited predisposition, to some debilitating illness, declining strength, or advancing age. Some are aware that there is no real ground for their sorrow and sadness, and are able to look on it as an illness; others feel that there must be some real cause for the despondency, that something terrible is impending, though they know not what. The majority can argue and converse rationally on subjects unconnected with their feeling of misery.

The bodily health, even at first it appears good, soon participates in the disturbance. Arterial pressure is high, the digestion disordered, the urine loaded with lithates, the skin dry, the bowels constipated, the pulse slow rather than quick, the conjunctiva dull and yellow. The patient will complain of various uneasy feelings in the præcordial or epigastric region, and this, with the state of the excretions, will confirm the notion, so prevalent among many, that the whole mischief is in the liver. Such simple depression may continue for a longer or shorter space of time. It may pass away suddenly or gradually, or the individual will grow worse in one of two ways. The depression becomes greater, and delusions of various kinds present themselves; or it is replaced by the excitement of mania.

Melancholia with delusions is far more common than *simple melancholia*, and is that which most frequently we are called upon to treat. The patient feels utterly changed, which he attributes to various causes, and deduces various results from his condition. He has all manner of diseases—syphilis, leprosy, lice; his stomach is gone, and therefore he

cannot eat. He cannot attend to business, and therefore is ruined. He is so wretched that he must have committed sins unpardonable in this world or the next. The bodily symptoms, like the mental, are aggravated. Sleep is absent or scanty; and there is rapid wasting. The bowels are loaded and resist strong purgatives; the tongue is white and furred; the breath offensive. The patients are for the most part elderly; climacteric insanity is almost always melancholia. Of 338 melancholic patients admitted into St. Luke's Hospital only nine were below the age of twenty.

It cannot be too strongly impressed upon medical men that all melancholic patients, even those whose disorder seems simple and slight, are, especially in the early stage, very apt to commit suicide. We read accounts almost daily in the newspapers of suicides committed by this class of persons; and most lamentable they are, for it is a class which above all others is amenable to treatment.

An asylum is not absolutely requisite for such, if their means allow of proper companions, house, and exercise. They must not be left alone by night or day; must not be left to attendants only; and must have some amusement or diversion. If all this cannot be provided, to an asylum they must go; for if they are resolutely and constantly bent on suicide, it is most difficult to guard against it in an ordinary house.

Whether they are sent to an asylum or not, it is found to be almost invariably necessary to remove them from home. We may think the case a slight one, and hope that amusement and cessation from work, with medical treatment and good living, will remove the depression. Again and again we are disappointed. The sight of home and home scenes, of family and friends, and the contrast between past happiness and present gloom, perpetuate the melancholy and prevent its dispersion. After valuable time is lost, we are compelled to send away the patient to an asylum or quasi-asylum.

PROGNOSIS.—The prognosis in cases of melancholia is favourable, and patients get well in great numbers, even at an advanced age. It is also important to remember that recovery may take place from this form of insanity after considerable periods of time. The writer has recorded cases of melancholia in which recovery took place after five, six, seven, and nine years respectively. In dealing with property it is often necessary to consider the question of probable recovery, and it is well to keep in view the chance of it here, although in perhaps every other form of insanity recovery after such periods would be out of the question.

TREATMENT.—On examination of a melancholic patient, it is generally found that there has been a considerable loss of flesh. This may be due to the mental care and sorrow, but is often caused by an insufficient quantity of food, which has been scanty, either because all appetite has been lost owing to the prevailing wretchedness, or because, from various delusions, there has been an unwillingness to take food. Moreover, there is almost always considerable disorder of the digestive apparatus, the result and not the cause of the depressed nervous condition. The first thing to be done is to correct this disorder; and then to restore the defective nutrition of the brain.

One symptom is obstinate constipation. It may be necessary in the first instance to relieve the

loaded and obstructed bowel by means of turpentine enemata; after which it will be of advantage to give a daily dinner pill of the extracts of aloes and nux vomica, or a daily teaspoonful of castor-oil, following it up if necessary by an enema, but ensuring an action every, or every other, day, and so habituating the bowels to act. Many melancholic patients, especially women, will be found to be persons who have been accustomed to go for long periods without any action of the bowels, or who never had relief without medicine. Food must be given to this class of patients in large quantities. It constantly happens that it is withheld from them under the impression that their malady is essentially dyspepsia, and that the stomach must not be called upon for much exertion. Many, as has been said, refuse it for one reason or other. In either case the melancholia increases, and the patient gets thinner and weaker. Food must be given with no sparing hand—not merely beef-tea and invalid diet, but solid food—bread, meat, and eggs, with a liberal allowance of wine or malt liquor. In some cases artificially digested food may be useful. Some require forcible feeding, and this can hardly be carried out except in an asylum; but many by coaxing or threats will take what is given to them with a spoon, and they must be fed frequently till they will take the meals of their own accord. Under this augmented diet the tongue will become clean, the bowels will act without physic, and the patient's appearance will soon testify to the efficacy of the treatment. *See* FORCIBLE FEEDING.

Sleep, though not entirely absent, will be in defect owing to the high arterial pressure. To lower this, erythrol tetranitrate in half-grain doses has been recommended, and is said to act like a charm, in many cases reducing the pressure and inducing sleep. In some, however, it fails, and hypnotics are necessary. In the same way opium fails in some patients, though in a large number of melancholics it is most valuable, not merely procuring sleep, but alleviating the feeling of wretchedness. It may be given either by the mouth or by subcutaneous injection of morphine. It is of importance that we do not give a preparation which shall cause sickness or constipation; the ordinary preparations of morphine, the acetate and hydrochloride, are apt to do this if given in full doses, and it is better to substitute Dover's powder, Battley's solution, or solid opium if we can be sure that pills will be swallowed. Chloral hydrate will procure sleep here as in other cases, and may be combined with opium to bring about more speedy action of the latter, but has not such a lasting influence on the malady; when its sleep-producing effect has passed away the patient does not feel any benefit from the medicine. Paraldehyde, also, is often useful. The bromides should on no account be given. They will increase the emaciation and depression. When the secretions have been corrected, and digestion is re-established, tonics may be useful, especially the preparations of iron and arsenic.

VARIETIES.—Two varieties may be mentioned.

1. **Acute Melancholia.**—Although the prognosis in simple melancholia, and that which may be called sub-acute, is so favourable, there is an advanced stage which truly merits the name of *acute*, or *acute delirious melancholia*, and generally terminates fatally. The patients are not silent, gloomy, and depressed, but panic-stricken; and in

violent frenzy and terror they try to escape from those about them, tear off their clothes, gouge out their eyes, and injure themselves in every way. They will not lie on a bed unless forced to do so, but will prefer the floor or incessantly pace the room. Food they resist with all their power, thinking that it is poisoned or that they will be punished for taking it. Such patients must be fed by force, and fed early, but it often happens that our feeding here is of no avail, and they sink from the exhaustion of this acute disorder. For it is constantly found in those who are already broken and debilitated in health, and is but the last stage of a series of disorders. The incessant agitation, violence, and sleeplessness produce rapid wasting and sinking; the food administered is not assimilated, and fails to restore the wasted force. This form of melancholia runs a rapid course, but nevertheless tends to recovery in some cases. We may administer the erythrol tetranitrate, opium, or paraldehyde. Cod-liver oil may be added to the food. Warmth and stimulants are demanded; and clothes must be kept on by means of a strong suit which cannot be removed by the patient.

2. **Mélancolie avec Stupeur (Fr.)**—**SYNON.**: Ger. *Schweremuth mit Stumpfsinn*.—A more extreme form of melancholia is thus named, where the patient sits or stands, speechless and motionless, and requires to be fed, washed, and dressed. Though such a one will not speak or do anything for himself, he may be watching every opportunity of committing suicide, and he will even strenuously refuse food with the same motive. The vital powers in these persons are greatly depressed, and they require an exceptional amount of nutritious food and stimulant. This form of melancholia has been confounded by some with that variety of insanity termed 'acute dementia' (*see* DEMENTIA); but the latter occurs only in young people, whereas melancholia as a rule occurs in persons of more advanced years; and the early symptoms are quite different, acute dementia coming on rapidly, and without the depression and gloomy delusions which mark the other complaint. *See* MENTAL STUPOR, p. 766.

G. F. BLANDFORD.

MELANIN.—*See* BLOOD, Morbid Conditions of (p. 161); PIGMENTATION; and URINE, Morbid Conditions of.

MELANOMA (μέλας, black).—Any morbid growth in which the presence of dark pigment is a leading character. *See* TUMOURS.

MELANOPATHIA (μέλας, black; and πάθος, a disease).—An excess of black pigment in the skin, due to abnormal function of the rete mucosum. Melanopathia is rarely general, more frequently partial. In certain instances, as in the 'bronzed skin' of Addison's disease, it is associated with anæmia. *See* PIGMENTARY DISEASES OF THE SKIN.

MELANURIA.—*See* URINE, Morbid Conditions of.

MELASMA (μέλας, black).—A term usually applied to excess of pigment in the skin, from abnormal function of the rete mucosum. *See* PIGMENTARY DISEASES OF THE SKIN.

MELLITURIA (*mellitus*, honeyed; and οὔρον, urine).—A synonym for saccharine urine. *See* DIABETES MELLITUS.

MEMBRANA TYMPANI, Diseases of. —
See EAR, Diseases of.

MEMBRANES OF BRAIN AND CORD, Diseases of. — See MENINGES, CEREBRAL, Diseases of; MENINGES, SPINAL, Diseases of.

MENIÈRE'S DISEASE. — See VERTIGO.

MENINGES, Diseases of. — For purposes of convenience these will be grouped under the heading of (1) *Cerebral Meninges*, and (2) *Spinal Meninges*, although in some instances the cerebral and spinal meninges may be simultaneously affected, as in epidemic cerebro-spinal meningitis and occasionally in a sporadic form of cerebro-spinal meningitis.

The following morbid conditions will be considered under this heading:

I. Cerebral Meninges:

- (1) Simple Idiopathic Meningitis, p. 977.
- (2) Traumatic Meningitis, p. 979.
- (3) Tubercular Meningitis, p. 979.
- (4) Posterior Basic Meningitis, p. 983.
- (5) Pachymeningitis Hæmorrhagica, p. 985.
- (6) Hæmorrhage, p. 986.

II. Spinal Meninges:

- (7) Traumatic and Secondary Meningitis, p. 988.
- (8) Idiopathic and Tubercular Meningitis, p. 988.
- (9) Hæmorrhage, p. 991.

Syphilitic affections of the cerebral meninges are described in the article on BRAIN, Syphilis of; new-growths, in BRAIN, Tumours of; and septic inflammations, in BRAIN and MENINGES, Septic Diseases of. See also INSANITY, Pathology of; and GENERAL PARALYSIS OF THE INSANE.

Tumours and local affections of the spinal meninges are discussed in the sections on Diseases of the Spinal Cord. See also MENINGITIS, Epidemic Cerebro-spinal; and MONSTROSITIES and MALFORMATIONS.

I. Simple Idiopathic Meningitis. — SYNON.: Simple Idiopathic Cerebral Leptomeningitis; *Leptomeningitis Infantum* (in part); Fr. *Méningite Simple*; Ger. *Acute Hirnhautentzündung*.

DEFINITION. — A simple non-tubercular inflammation of the cerebral pia mater, which may be either limited to the convexity, general, or confined to the base of the brain. It is associated with very variable symptoms in different cases; and is probably caused in many different ways.

ÆTIOLOGY AND PATHOLOGY. — Our knowledge of the ætiology and pathology of acute idiopathic cerebral meningitis has of late years become more definite.

It appears that sex exercises an influence in the production of idiopathic meningitis, and that the disease occurs much more frequently in males than in females. In regard to age, it is met with almost as frequently in individuals from ten to twenty as in those below the tenth year. In individuals over twenty the disease is much more rare.

Meningitis is apt to occur during, or as a sequel of, many acute febrile diseases, such as pneumonia, measles, scarlet fever, small-pox, and rheumatic fever. Such associations are, of course, rare, but much less so with pneumonia than with either of the other diseases named. It may also complicate erysipelas of the head and face; or it may occur as an accompaniment of ulcerative endocarditis or pyæmic processes, owing to these conditions leading to minute embolisms of the vessels of the pia mater.

In all these cases it is supposed to have an infective origin, and is associated with the presence of different micro-organisms.

Sometimes it is met with in extremely cachectic subjects, who have not previously been suffering from any acute disease. It has been known to follow prolonged exposure to the sun; to ensue after the occurrence of severe moral perturbations; and likewise to follow a shock or blow, even when this has not been complicated with an external wound, or with a fracture of one of the bones of the skull.

In other cases, also, a meningitis really secondary may appear to be primary and idiopathic, as when (a) it extends from some focus of syphilitic disease of the meninges, or (b) when it occurs as a sequel of some unrecognised chronic inflammation involving the middle ear and portions of the temporal bone.

ANATOMICAL CHARACTERS. — Simple idiopathic inflammation of the cerebral meninges is a condition which varies much in severity in different cases. In its earliest or initial stage, nothing more than a minute and more or less uniform injection of small vessels and capillaries in certain regions of the cortex may be met with. But later on, definite products of inflammation are to be seen; these are for the most part situated beneath the arachnoid, in the meshes of the pia mater. They consist, according to the stage of the morbid process, either of a gelatinous white or yellow lymph-like matter, of actual pus, or of more coherent yellow lymph in the form of membranous layers. In regard to the area involved considerable differences also exist. The inflammation (1) may be limited to the convexity and to the lateral regions of both hemispheres; (2) it may be general, that is, involve the parts above mentioned, and also the base; or (3) it may be limited to the basal regions of the brain. In both the latter cases the ventricles are apt to contain fluid, and the central parts of the brain to be softened, as they are in tubercular meningitis, which also affects the base in a special manner.

Of these varieties as to seat, the first, in which the convexity is involved, is decidedly the most typical, and in this respect simple idiopathic meningitis contrasts in a salient manner with tubercular meningitis, in which the tendency is no less marked to implicate the base of the brain. In the second variety, the inflammation beginning above probably extends to the base by mere continuity, in cases where the condition of the patient, or the intensity of the inflammatory process itself, favours its spread from the original site; or, in certain cases, the inflammation may be from the first general in seat.

In regard to the third variety, doubtless many of such cases have been instances of what we now know as 'posterior basic meningitis.' In these cases the basal inflammation may co-exist with, or be an extension upwards from the spinal meninges of an inflammation beginning there—a case, in fact, of cerebro-spinal meningitis of sporadic type. On the other hand, we may have to do with one of those cases of tubercular meningitis where the general disease manifests itself on the side of the brain first, and in which the patient dies before the local process is at all fully developed. In such a case the inflammation may be really of the tubercular variety, and yet to superficial observation not recognisable as such.

In all these cases the inflammation may be limited to the meninges themselves, or the surface of the

brain may also be manifestly involved in the inflammatory process, so that we then have to do with a *meningo-encephalitis* of varying seat and extent.

SYMPTOMS.—In no disease is the symptomatology more various than it is in acute meningitis—a fact partly due to the varying intensity of the inflammatory process, partly dependent upon the process being localised or more general, and partly according as there is or is not the co-existence of dropsy of the ventricles with inflammation of their walls. Independently of these causes of variation there is the fact that the symptoms of the meningeal inflammation are, in the numerous cases in which it occurs as a secondary affection, apt to be complicated and more or less overshadowed by those of the acute disease to which it owes its origin. Sometimes the disease is almost latent, accompanied only by slight symptoms, merging into stupor and coma a day or two before death. Or the symptoms may be marked and quite tragic in their severity; ushered in either by frightful pains in the head, by well-marked delirium, or by convulsions; subsiding eventually into a condition of stupor or coma; and followed by death within eight or ten days, though this may be delayed till the expiration of three weeks or a month. Recovery, which sometimes occurs, must be regarded as a rare event.

Inasmuch as it is not practicable, within the limits of this article, to give a detailed account of the various groupings of symptoms that may be met with in different cases, we must confine ourselves to an enumeration of the symptoms themselves, most apt to occur (1) in the early stages of the disease, and (2) in its later phases.

(1) Cephalalgia of an intense character, either general or localised in some particular region or regions of the head, may be complained of again and again where the patient is old enough, or, if he be too young, is indicated by cries, by application of the hands to the head, or by other signs. Sometimes, however, this symptom may be almost absent, or it may come on at a later date. Delirium, occasionally furious, at other times more quiet and of a simply loquacious type, is another symptom; or there may be extreme restlessness. Mere insomnia, too, sometimes exists from the commencement; while at other times a semi-comatose condition, gradually deepening into actual coma, may exist from the first, especially in children, or it may succeed a transitory delirious condition. Nausea and vomiting, and also convulsions, either local or general, may be met with in the early stages of the disease, and sometimes as initial symptoms. With them will go general pyrexia and sometimes rigors; also heat of head, rapid pulse, a furred and often thickly coated tongue, constipation, perhaps some intolerance of light and of loud sounds, together with an easily obtainable *tache cérébrale*, and often some amount of hyperæsthesia of the skin.

(2) As later symptoms, we may have localised convulsions or spasms, often of the tonic order, affecting perhaps the head and neck (which, especially when the posterior part of the base is involved, are frequently drawn backwards) or one or both arms; or a condition of trismus may exist. The eyes, too, are sometimes drawn upwards. The pupils may be at first contracted, or, if not, they may be of medium size, unequal and insensitive;

while later on they are most frequently widely dilated and insensitive. The conjunctivæ are often injected. Paralysis of one arm, or sometimes of an arm and a leg, may occur. The condition known as 'Kernig's sign' may also exist, but it is occasional and fitful only in its presence, and the patient may not be in a condition to permit of its being looked for. It consists in the impossibility of completely extending the leg upon the thigh when the patient is in the sitting position. The sensibility of the skin may be either exalted or deadened. The abdomen is often hollow and boat-shaped. The tongue becomes thickly coated, or dry and brown. Difficulty of deglutition is frequently well marked towards the end; and there is incontinence of feces and urine as soon as the stupor becomes marked. Sometimes the pulse is unnaturally slow and infrequent from the first; at other times, and especially towards the end, it is very frequent and irregular. The respiration, too, becomes much disturbed, being often sighing and of very irregular rhythm, tending to become stertorous at last. The temperature is frequently high, but pursues a markedly irregular course. Remissions of the pyrexial condition may take place from time to time. The skin is generally hot and dry, though occasionally there may be copious sweats. Stupor and coma almost invariably occur at the last, if not present at an earlier stage.

PROGNOSIS.—A large number of deaths take place within the first week of acute meningitis; a much smaller number survive till the end of the second week; fewer still reach the end of the third; and only a very few survive to the fourth week. It is difficult to say what the percentage of recoveries may be, but probably less than ten would survive out of a hundred cases of acute idiopathic cerebral meningitis.

DIAGNOSIS.—The diagnosis of idiopathic meningitis involves considerations very similar to those arising in the diagnosis of tubercular meningitis, and need not therefore now be discussed. *See* MENINGES, CEREBRAL, Inflammation of, Tubercular.

The diagnosis of simple from tubercular meningitis must oftentimes be a matter of extreme difficulty. Whether the condition of the blood, as recognised by the aid of the microscope, is the same in simple meningitis as it is in tubercular meningitis the writer is unable to say. Should it not be so, some help might be obtained in this direction. The conditions under which the disease seems to develop may throw some light upon the problem. In regard to special symptoms, the possible range is so great in each variety that it becomes difficult to fix upon any that are positively distinctive of the one or of the other. Delirium is, however, rarely so violent in tubercular as it may be in simple meningitis. Retraction of the head is also not so frequent in the tubercular variety. On the other hand, the temperature much more frequently rises over 101° F. in simple than it does in tubercular meningitis. Finally, it must be borne in mind that the former is an extremely rare disease, the latter unfortunately only too common; and that while in tubercular meningitis the two sexes fall victims with about equal frequency, in the simple variety two out of three are likely to be males.

TREATMENT.—In the early stages of acute simple meningitis aperients may be freely administered. A leech or two might be applied to the

temples, in cases where pain is greatly complained of; or under the same conditions the head may be shaved and an ice-bag or Leiter's tubes may be applied, should such measures not be deemed useless on account of the extreme restlessness of the patient. The writer believes that little or nothing is at present to be expected from drug-treatment towards the cure of this disease, although some alleviation of the more distressing symptoms may at times be brought about by special attention to them. The patient requires to be carefully fed, and assiduously nursed and kept quiet throughout, in the hope that the end may be favourable.

H. CHARLTON BASTIAN.

2. Traumatic Meningitis.—Suppuration of the meninges following injuries to the head is always caused by the introduction of pyogenic micro-organisms, and owing to the intimate relationship of the pia mater and arachnoid to the brain, we always find that they are all three involved in the process. Septic inflammation of the pia mater and arachnoid are considered in the article on BRAIN AND MENINGES, Septic Diseases of. The relation of the *dura mater* to the brain is very different from that of the other two membranes. It is intimately connected with the skull, in all probability acting as regards the cranium like the periosteum of long bones. The thin connective-tissue layer on the outer surface of the cranium termed the pericranium can scarcely be looked upon as a periosteum. It has no osteogenetic function, and its vessels supply but few branches to the bones of the skull. The meningeal arteries are mainly concerned in the blood-supply of the skull, only a very small proportion of the blood passing through them being required for the nourishment of the *dura mater*.

In its behaviour to septic organisms the *dura mater* differs markedly from the other membranes. It acts as a very efficient barrier in preventing septic conditions of the skull-bones from reaching the brain. In cases of compound fractures, whether depressed or not, very free suppuration may take place, and, so long as there is free drainage from the surface of the *dura mater* through the scalp-wound, septic inflammation rarely spreads inwards, but if the *dura mater* is wounded there is at once enormous risk of septic inflammation of the brain and its meninges proper, i.e. leptomeningitis. Although inflammation of the *dura mater* sufficient to cause symptoms, and therefore to be of clinical rather than pathological interest, is nearly always septic in origin, yet there are a few cases in which the inflammatory and reparative processes that may follow injury are of sufficient extent to cause symptoms, e.g. in every simple fracture of the skull there is probably some injury to the *dura mater* at the line of fracture, and some blood is poured out from the small ruptured vessels. The ordinary inflammatory reaction with oedema may cause slight cerebral compression for a few days; again in healing there may be formed an amount of fibrous tissue sufficient to press upon the subjacent brain and, if the motor area be involved, to cause traumatic epilepsy. After injuries in which a large effusion of blood forms between the *dura mater* and the bone blood-cysts may develop, and these together with the thickening of the *dura mater* may cause symptoms of cerebral irritation or compression. The chief affections of the *dura*

mater, however, are septic processes spreading from the bone. In most cases there is a wound of the scalp and there may be fracture of the skull. In the former case the septic osteitis spreads inwards to the *dura mater*. In the latter cases there may be a direct entrance of pyogenic germs to the *dura mater*. This process takes some time to lead to the formation of pus, and the wound of the scalp is very often healed by the time the abscess reveals its presence. From the inflamed bone serous exudation may spread into the scalp and cause a localised oedematous swelling first described by Percival Pott, and termed by him the 'puffy tumour.' In some cases this septic pachymeningitis or sub-cranial abscess may form without any apparent wound of the scalp. No doubt sometimes the septic material enters by small abrasions which are overlooked, and at other times the deeper layers of the tissues of the scalp are crushed, and any organisms lying in the hair-follicles or sebaceous glands may readily gain entrance into the tissue and spread to the bone, bruised by the violence of the injury. In either case the diploic veins become thrombosed. As soon as the organisms reach the bone a septic osteitis commences which results in the formation of an abscess on the outer surface of the *dura mater* and a serous exudation in the adjacent region of the scalp—*puffy tumour*. If the tension existing between the bone and *dura mater* is not relieved by surgical means the septic process will extend to the brain. The resistance of the *dura mater* is proved by the long time that the septic inflammation may exist before spreading to the underlying brain. If the *dura mater* has been wounded at the time of injury it may become adherent to the subjacent membrane and permit the extension of organisms to the brain and the formation of a sub-dural abscess.

The symptoms and treatment of these conditions are the same as those in cerebral abscess and septic leptomeningitis to which the reader is referred. See BRAIN AND MENINGES, Septic Inflammation of.

HENRY PERCY DEAN.

3. Tubercular Meningitis.—SYNON.: Granular Meningitis; Acute Hydrocephalus; *Hydrocephalus Internus*; Brain-Fever (in part); Tubercular Leptomeningitis; Fr. *Fièvre Cérébrale*; *Meningite Granuleuse*; *Meningite Tuberculeuse*; Ger. *Tuberculöse Hirnhautentzündung*.

DEFINITION.—An acute and extremely fatal febrile disease, with a predominance of head-symptoms; terminating in stupor and coma, with or without convulsions; and characterised after death by a 'granular' meningitis affecting the pia mater at the base of the brain, with the frequent accompaniment of dropsy of the lateral ventricles, and softening of their walls. The inflammation of the membranes at the base of the brain is commonly found to be associated with a more or less marked spinal meningitis.

Tubercular meningitis is not an independent affection; it constitutes one important phase of acute tuberculosis. In certain rare cases death takes place from meningitis, before the anatomical marks of the general disease have had time to develop within the chest or abdomen. More frequently, however, the manifestations of the general disease are already well developed in one or other, or in both, of these situations, at the time that they reveal themselves also on the side of the brain. In

the latter, and by far the most common class of cases, the symptoms met with will be in part those of the general affection, and in part (but in a predominant degree) those due to implication of the brain and its membranes. See TUBERCULOSIS.

ÆTIOLOGY.—The ætiology of tubercular meningitis of course resolves itself into the ætiology of the general disease, acute tuberculosis, of which it forms part.

The affection is one which occurs with special frequency in young children, between two and six years old, though it is also met with in infants, in older children, in young adults, and even in persons beyond middle age. In adults it is most apt to manifest itself as an occasional complication in the course of pulmonary phthisis. In children a proclivity to the disease seems often to be inherited, so that two or more in the same family may be carried off by it. But in what proportion of cases any such proclivity exists is unknown.

ANATOMICAL CHARACTERS.—When the calvaria is removed, the dura mater is found to be tightly stretched over the brain; unless, as the writer has seen, the subject be an elderly person, in whom some amount of senile wasting had previously occurred. On stripping back this membrane, the arachnoid presents a dull appearance, and it is slightly sticky when touched. The convolutions of the vertex and lateral regions of the brain are mostly seen to be more or less flattened from pressure, and the sulci are correspondingly indistinct. No lymph may be seen; or at most a small quantity, in the lower parietal regions, along some of the branches of the middle cerebral arteries. When the brain is removed, however, and its under surface is examined, a more or less opaque white or yellowish lymph-like matter may be seen, beneath the arachnoid, in the meshes of the pia mater, extending from the optic commissure backwards over the central portions of the base and onwards over the pons. In certain cases lymph and evidences of recent inflammation are found round the medulla, and even along the whole length of the spinal cord. More or less lymph also extends on each side into the Sylvian fissures. A minute inspection will likewise show that the tip of the temporo-sphenoidal lobe, and the orbital surface of the frontal lobe, are flecked with a number of translucent granulations, as though the parts had been sprinkled with fine sand; and on opening up the Sylvian fissure on each side, similar granulations, with others more opaque and of larger size, may be seen among the lymph in this situation. Translucent granulations also sometimes exist scattered more sparingly over the lateral aspects of the hemispheres, especially along the sides of the vessels.

Examination with the microscope shows that the granulations are composed of overgrowths of tissue-elements immediately surrounding the smaller vessels, and within their perivascular sheaths. Within and among these cellular elements the *Bacillus tuberculosis* is to be met with, though very sparingly. In these situations the tissue-overgrowths may cause a local bulging of the sheath, either all round, or merely on one side of the vessel; and when such growths become opaque from incipient fatty degeneration, they are then more easily visible as minute white specks. A close examination of the prolongations of the pia mater dipping between the convolutions, with the aid of lens or microscope, will often show minute granulations not otherwise

recognisable—and that, too, in many regions of the brain. And in cases of incipient tubercular meningitis, where the amount of lymph about the base is extremely slight, the lens or microscope may show the presence of granulations, not otherwise recognisable, in and around the lower part of the Sylvian fissures—that is, in the regions where they are most prone first to manifest themselves.

The pia mater is generally unduly adherent to the surface of the convolutions, so that it can only be removed in small shreds, and then not without tearing the superficial grey matter. This condition of things is the very opposite of what may be met with in some cases of simple meningitis affecting the vertex, in which the thickened pia mater, with all its prolongations, may sometimes be easily stripped off from the greater portion of a hemisphere in one piece.

The substance of the brain is commonly much more vascular than natural. The lateral ventricles are usually moderately dilated, containing from two to four or six ounces of not very clear serum. The veins on their surface are then engorged, and the fornix and other adjacent parts may be more or less softened, or actually diffuent. Microscopical examination of such softened tissue will reveal the presence of an abundance of fatty débris; and its specific gravity, if estimated, will be found to be diminished—both these characteristics being marks of a pathological softening which has occurred during life, and not of a softening due to mere *post-mortem* maceration. Some have erroneously supposed that such mere maceration has been adequate to produce the softening.

Sometimes the above-described changes are more fully developed in one than in the other hemisphere; and occasionally also in some parts of the brain small nodular growths of a 'tubercular' nature may be met with, varying in size from a small pea to an almond. These growths are most apt to occur in the substance of some of the cerebral convolutions, or near the surface of the cerebellum, or even, as the writer has seen, within the substance of the corpus striatum. In many such cases the small nodular tumours will be found to be in intimate relations with the vessels of the part, and, in fact, to be composed of a mere aggregate of the smaller 'granulations' more or less fused into a single mass.

PATHOLOGY.—The granulations begin to appear first in the meninges of the base under those irritative influences, whatever they may be, which lead to the development of similar grey granulations in other organs of the body. These primary changes excite a common inflammation of the membranes around, and thus entail the production of the lymph, which covers the base of the brain, and extends on either side into the Sylvian fissures. Why the grey granulations should tend to develop first, and specially about the vessels at the base of the brain, cannot at present be explained.

This inflammation of the basal meninges also extends, by direct continuity of tissue, over and around the cerebral peduncles to the velum interpositum, and to the connective tissue at the upper and anterior extremity of the middle lobe of the cerebellum. In one or other situation, and often in both, the tissues are thickened by lymph. The writer has seen the velum interpositum thick and leather-like in consistence, and the *venæ magnæ Galeni* which run through it blocked by thrombosis; and this he

believes to be an additional occasional cause of the central softening and dropsy previously referred to as component parts of the disease. In other cases, where no such thickening or thrombosis is to be detected, there is great swelling of the connective tissue, from development of lymph, opposite the termination of those great veins which return the blood from the surface of the ventricles and from the central parts of the brain—at the point, that is, where the veins of Galen empty themselves into the straight sinus.

In this way the very common association of the central ventricular changes with the basal meningitis may be accounted for, and also the occasional absence of such changes, in instances where the inflammation, apt to be set up through mere continuity of tissue, does not attain sufficient proportions to interfere with the return of blood, either through the veins of Galen, or from them into the straight sinus. Undoubtedly the mere dropsical effusion into the ventricles will also be in part due to the frequent closure of the foramen of Majendie in the fourth ventricle; such closure preventing the draining away of fluid from the lateral ventricles, and thus leading to their dilatation. It is of course possible that the central softening may also be favoured by an independent affection of the small vessels situated in the walls of the ventricles, and a development of granulations around them—though this has not hitherto been recognised. It is, however, well known that thrombosis is extremely apt to occur in those minute vessels in various parts of the brain which are enveloped by granulations—a fact that goes far to account for the extreme gravity of the symptoms in many cases of tubercular meningitis, in which naked-eye changes appear to be slight and altogether disproportionate in amount.

SYMPTOMS.—The symptoms presented in different cases of tubercular meningitis often vary very widely from one another, although among them all there is an underlying bond of similarity. The variation may be easily understood from a consideration of the fact that such symptoms form part of those pertaining to a febrile affection characterised by other local manifestations, of varying importance in different cases; and also from the fact of the differences constantly met with in the relative and absolute development of the different kinds of changes encountered within the cranium itself in this disease—especially in regard to the amount of ventricular effusion and central softening existing in conjunction with the meningeal inflammation, which again itself varies much in intensity and in regard to the area involved in different cases.

It is, therefore, usual and most convenient to enumerate the possible signs and symptoms of this disease as they occur in three stages—artificial and often ill-marked from one another as they are—namely (1) those of the *invasion-stage*; (2) those of the *developed disease*; and (3) those of its *closing phases*.

(1) *Stage of invasion.*—Among the initial symptoms of tubercular meningitis may be mentioned obstinate and recurrent vomiting, often associated with constipation; coming on frequently after a period of previous malaise; and associated with fretfulness, slight wasting, indisposition to play, and disturbed sleep. Soon after, or simultaneously, there may be more or less marked indications of cephalalgia. Young children who cannot speak are fretful and constantly cry; they often also put their

hands to their head. Such children start and cry out in their sleep. The temperature may be as yet scarcely, if at all, elevated; or there may be rigors from time to time, with temporary feverishness, recurring daily about the same hour. The child often cries out when touched, and a more or less general hyperæsthesia of the skin seems to exist.

(2) *Developed disease.*—In the second stage any feverishness that may have existed often abates. There may be less restlessness, so that the child even sleeps more than natural. The pupils are often insensitive to light, and unequal. There is frequently also some slight or perhaps marked strabismus. The pulse is apt to be much less frequent than natural (56–70 per minute perhaps), and decidedly irregular. The hypersensitiveness of skin may have disappeared, but a peculiar vaso-motor irritability exists, so that when the nail of the forefinger is drawn once across the skin of the abdomen or other part a deep red linear mark comes out slowly, and persists a long time. This so-called *tache cérébrale*, while also met with in other affections, is, as Trousseau rightly enough insisted, rarely absent in tubercular meningitis. Frequent plaintive cries may be uttered, though the child is generally more quiet and drowsy; it is apathetic also in regard to food, not asking or crying for it, but still taking it, perhaps well, whenever it is administered. Convulsions may occur during this stage, or weakness of one or more limbs may be noticed, especially where larger tubercular nodules occur in certain regions of the brain-substance. Sometimes, however, the paralysis is of a shifting and transitory nature, varying in degree or even in situation in the course of a few days.

(3) *Closing phases.*—In the closing stages of the disease the drowsiness may gradually deepen into stupor or actual coma; though in conditions short of the latter the child may still more or less frequently utter plaintive cries. The pulse, instead of being less frequent than natural, now becomes preternaturally frequent; while the respiration often assumes a slow, sighing, and markedly irregular type. The face, frequently pale and clammy, flushes at times. The head is hot, and the temperature generally raised, though often not more than to 100° F., and rarely beyond 102°, until quite to the close of the disease. The fontanelle is raised, and there may be unnatural pulsation. The eyes, when examined with the ophthalmoscope, may show evidences of grey granulations in the choroid, and perhaps some amount of optic neuritis. The pupils may be unequal, but are generally dilated and insensitive. Occasionally the writer has seen a rhythmical contraction and dilatation go on, especially on exposing them to light. In this stage, when the patient is sufficiently conscious, it may be found that sight is notably impaired or almost lost.

The patient may take the food which is given up to the last; though at other times there seems to be an actual inability to swallow it, even when it is placed in the mouth, owing to partial paralysis of the muscles of the tongue and pharynx. The abdomen is often boat-shaped and retracted; and an obstinate constipation still continues. Even in this last stage of the disease a temporary and delusive lull may take place; the child may seem to revive a little, but only too soon to lapse again into a state as bad as, or even worse than, before. Frequent and long-continued convulsive seizures are especially apt to occur during this stage of the disease; and

death may take place during or immediately after one of these attacks. At other times the end is brought about more gradually, through progressing failure in the heart's action, combined with disturbance of respiration. In the latter class of cases the temperature may gradually fall, during the last few hours before death takes place, to several degrees below the normal; though in other cases of tubercular meningitis there is a slow and steady rise of temperature up to 105° or even 106° , before the patient expires.

DIAGNOSIS.—In the early stages the diagnosis of tubercular meningitis may present extreme difficulties. We must wait, before expressing a definite opinion in one of these doubtful cases, till the patient has been seen and examined two or three times. The premonitory symptoms and those of the first stage are often far from distinctive. They may, it is true, represent the beginning of tubercular meningitis, but, on the other hand, they may also represent something less serious—for instance, a mere failure of health from various causes, complicated by dentition, by some gastro-intestinal irritation, or perhaps the commencing outbreak of some one or other of the specific fevers. Details as to the child's condition during the last two or three weeks, comprising the order of evolution of the several symptoms, may, however, throw some important light upon the real nature of the case at an early stage of the disease.

A contributory cause of the difficulties besetting the early diagnosis of tubercular meningitis is to be found in the fact that acute tuberculosis is itself extremely difficult to recognise. We cannot, therefore, readily fall back upon a diagnosis of the general condition in order to strengthen our diagnosis of tubercular meningitis. As a matter of fact, it is just the reverse. Of all the local manifestations of this disease, those within the head produce by far the most definite set of symptoms, so that we can always most safely infer the probable existence of acute tuberculosis with grey granulations throughout the body, from the presence of the developed symptoms of tubercular meningitis.

The existence of a particular habit or build of body in cases of acute tuberculosis, to such an extent as to make it possible to use the recognition of it as an aid to diagnosis in a case otherwise obscure, is practically unknown. See **TUBERCULOSIS**; and **PHTHISIS**.

The symptoms of the established disease are therefore alone distinctive, to any really trustworthy extent, of the existence of tubercular meningitis, and through it of the presence of its general underlying condition. We may have our suspicions before, but these can only transform themselves into certainties as the disease actually develops, and as it passes, moreover, into the incurable stage.

At this phase of the disease the alternative conditions to be thought of are in the main either typhoid fever on the one hand, or else some form of intracranial disease other than tubercular meningitis. Here, as in almost all cases of brain-disease, we have to look not to any one or two signs or symptoms which can be regarded as pathognomonic, but rather to the sum total of symptoms, and to the way in which they are grouped. With the possible existence of some or all of the premonitory and initial symptoms already enumerated, if the patient becomes more somnolent; if the pulse falls much below the normal in frequency, and is at the

same time irregular; if, with a condition of fever still existing, the child does not constantly crave for drink; and especially if there is also the combination of obstinate constipation and a retracted abdomen, together with an irregular and sighing form of respiration—we may feel more and more certain that we have not to do with even one of the most anomalous forms of typhoid fever associated with head-symptoms—nor, indeed, with any form of intracranial disease other than tubercular meningitis. An examination of the temperature-chart may considerably aid us in the same direction, and so also may an examination of the blood. See **SERUM-REACTION**; **BLOOD**, Morbid Conditions of (p. 157).

The writer has in many cases found distinctive alterations in the blood drawn from the finger-tip. The white corpuscles (more numerous than natural) within ten to fifteen minutes show signs of great amoeboid activity, by the development of vacuoles within them, and of numerous projections from their outer surface; among the blood-corpuscles groups of protoplasmic particles of various sizes, as well as here and there a small pigment granule or an irregular block of blackish-red pigment, are to be found. The red corpuscles form 'clumps' rather than rouleaux.

Lumbar puncture has of late years been occasionally had recourse to as an aid to diagnosis (see **LUMBAR PUNCTURE**). Tubercle-bacilli may be recognisable in the fluid drawn off, either by simple microscopical examination or by culture-experiments. Widal and others, by centrifuging the slightly turbid fluid thus obtained from cases of tubercular meningitis, have found a considerable number of lymphocytes and large mononucleated leucocytes, which are said to be found only in forms of meningitis, though not peculiar to tubercular meningitis.

For the diagnosis of tubercular from the simple form of meningitis, and from 'posterior basic meningitis' see pp. 978 and 984.

PROGNOSIS.—Death is well-nigh certain within three weeks, or at most a month, from the date of the invasion-symptoms of tubercular meningitis. When the disease has arrived at a stage permitting of pretty certain diagnosis, hope rather than rational expectation may still hold out a chance of recovery. Although instances of this have been said to occur, they are of extreme rarity. If the course of the disease is to be modified by treatment, it must be during those early stages when we are capable of forming only a provisional or tentative diagnosis. In these stages, however, some good observers have hitherto been inclined to think that under judicious treatment the development of the disease may be arrested. This view may quite possibly, and even probably, be an erroneous one. Proof of such a position, or of its opposite, is, from the nature of the case, impossible.

TREATMENT.—From what has just been said, it will be seen that anything like curative treatment is only to be thought of in regard to the early or premonitory stage of the disease, or of conditions of health indistinguishable therefrom. Here the writer thinks he has seen decidedly good results from one to six grains of iodide of potassium, according to the age of the child, administered three times a day, with small doses of cod-liver oil; at the same time attending to the state of the bowels, and giving suitable doses of bromide of potassium at night, till the restless condition with disturbed sleep has passed away.

When the disease definitely declares itself or is further advanced, we may perhaps be able to diminish pain by the application of cold to the head; but we only aggravate the sufferings of the patient by the use of blisters, tartar-emetic ointment, or other irritating applications, such as were often had recourse to by our predecessors. Bromide of potassium may do something to keep convulsions in check, though at other times it seems to be quite powerless, and drugs of this type should never be employed in later stages of tubercular meningitis, unless there is some strong indication for their use. Chloral hydrate, again, is probably a dangerous drug for a patient, the action of whose heart is already so seriously interfered with; though chloroform inhalations may be had recourse to in an extreme case where persistent convulsions cannot otherwise be checked. Beyond this the child needs the most careful nursing, and to be well supported with strong beef-tea and milk, and occasionally with stimulants, by the mouth, so long as it is capable of taking food, but later, if need be, by means of a nasal tube. Attention must also be paid to the bowels, which are often best relieved by means of enemata. In this way, if the patient's case is to prove one of those rare and exceptional instances in which recovery is possible, we at all events do nothing to thwart the course of natural processes which have a chance, however small, of terminating in recovery. Lumbar puncture as a means of cure is useless.

H. CHARLTON BASTIAN.

4. Posterior Basic Meningitis.—**SYNON.**: Chronic Basilar Meningitis.—This disease occurs most commonly in infants, and is characterised chiefly by persistent head-retraction with vomiting. It is an infective form of leptomeningitis, distinct both from tubercular and from secondary suppurative meningitis. It is probably identical with the sporadic cases of so-called epidemic cerebro-spinal meningitis, but there are slight differences, both bacteriological and clinical, which make it perhaps desirable for the present to describe the posterior basic form as a separate condition.

ÆTIOLOGY.—It occurs most often during the first nine months of life; it is very rare after the end of the second year. Girls and boys are equally liable. The onset is usually in winter or spring; more than half the cases begin during the first four months of the year. It has occurred in many large towns and cities, but no evidence of contagion or of epidemic spread has been observed.

BACTERIOLOGY.—The exciting cause of the inflammation in the pia-arachnoid is a micro-organism, the diplococcus of posterior basic meningitis, which is probably identical with the *Diplococcus intracellularis* of epidemic cerebro-spinal meningitis. Until recently this was confused with the pneumococcus, from which it differs in many particulars. The diplococcus has been found in the cerebro-spinal fluid by lumbar puncture during life, and *post mortem* in the exudation at the base of the brain and on the spinal cord. It gradually disappears, however, after a varying time, so that in some cases three or four weeks after the onset of the disease no micro-organisms may be found. It is this tendency to spontaneous disappearance of the diplococcus which probably accounts for the clinical fact that a certain proportion of the cases recover.

SYMPTOMS.—The onset is almost always acute. An infant vomits, and is fretful or drowsy. At the same time, or it may be a few days later, the head is noticed to be drawn back stiffly, and the child cries if any attempt is made to push it forward. Sudden screaming without apparent cause is sometimes an early symptom, and in about one-third of the cases convulsions occur at the onset. Some fulness of the anterior fontanelle may be present at this stage; at a later period the bulging fontanelle may attract even the mother's notice. Head-retraction is the most constant and characteristic feature of the disease. It is almost always an early occurrence, sometimes it is the first symptom noticed. It varies in degree in different cases, and in the same case at different times; it may be so slight as to be only just noticeable, but more often it is well marked, and in some cases is so extreme that the occiput almost touches the buttocks. The opisthotonos may be limited to the cervical spine, or may affect also the dorsal and lumbar portions. With this opisthotonos there is often some rigidity of the limbs; the shoulders also may be strongly retracted. Convulsions may, as already mentioned, occur at the onset, but after this they are not a marked feature of the disease, and in some cases they are absent throughout. The face often has a staring appearance, which is due partly to spasmodic retraction of the upper eyelids, so that the sclerotic is seen above the cornea, and partly in some cases to the fact that the child is blind. Blindness occurs in about one-third of the cases. It comes on gradually, and has been noticed as early as the end of the first week. In cases which have recovered sight has returned completely after several months. The loss of sight does not correspond with any change in the fundus oculi, and the light reflex is retained in such cases, although it is sluggish. It would seem therefore that the blindness is central in origin. Optic neuritis very seldom occurs; nystagmus, usually slight and transient, is sometimes observed; squint is present in about half the cases, but is usually very slight, and may only be noticeable occasionally. Paralysis of any other cranial nerves, or of any of the limbs, is very rare. Grinding of the teeth and champing movements of the lower jaw are common in this condition; it seems likely that they are due to cerebral irritation. Tactile sensation seems to be unaffected. There is no evidence of any general hyperæsthesia, such as is found in epidemic cerebro-spinal meningitis. Deafness was observed by the writer only once in forty-five cases. The knee-jerks are usually brisk, and in the chronic stage of the disease may be definitely exaggerated with ankle-clonus: the superficial reflexes are normal. The temperature is raised at the onset, and in some cases remains high for two or three weeks, and then gradually falls; in others it falls after the first few days, and then remains normal. Less often there is irregular pyrexia throughout the illness. When exhaustion becomes extreme the temperature sometimes becomes subnormal: a fatal ending may be preceded by sudden hyperpyrexia. The pulse is usually regular, but rapid. Respiration, especially in the later stage of the disease, often shows a cyclical irregularity; long pauses alternate with series of rapid respirations, which, however, lack the rhythmical increase and decrease in depth which is characteristic of the Cheyne-Stokes breathing. Vomiting is usually a prominent symptom; it is more persistent than in

tubercular meningitis, and may occur almost daily throughout the illness. Constipation is not so marked a feature as in the tubercular disease; indeed, in the chronic stage there is sometimes troublesome diarrhoea. In most cases the infant gradually wastes; food is taken badly, and with increasing emaciation and exhaustion death occurs.

COMPLICATIONS.—The most serious and the most frequent is hydrocephalus. Within a few weeks of the onset of the meningitis measurements will often show a definite increase in the size of the head. The anterior fontanelle becomes full and tense; and as the intracranial pressure increases there may be some downward rotation of the eyeballs, so that the cornea is unduly covered by the lower eyelid. The hydrocephalus seldom reaches any extreme degree, such as is met with in congenital cases; but it is probably one of the most important factors in the mortality of posterior basic meningitis; for while the inflammation of the meninges tends to subside spontaneously, and in some of the fatal cases has already completely disappeared, leaving only fibrous adhesions, the hydrocephalus which results from the obstruction produced by those adhesions tends to be steadily progressive. A much less common complication is an inflammation about the joints, one or more of which become red and swollen. In some cases, at least, the inflammation is found to be entirely outside the joint—it is, in fact, a peri-arthritis. This condition has been shown to be due to the same diplococcus as is found in the meningeal exudation. As in other exhausting conditions, such accidental complications as bronchitis or bronchopneumonia may supervene, especially towards the end of the illness.

MORBID ANATOMY.—In the acute stage an exudation of yellowish lymph is found in the pia-arachnoid at the base of the brain and on the spinal cord. It is situated chiefly in the posterior fossa about the medulla and the inferior surface of the cerebellum, particularly in the reflection of arachnoid which passes from the medulla to the cerebellum. It extends forward usually to the optic chiasma, and sometimes along the Sylvian fissures; in rare cases there is also a trace of exudation along the sulci up to the vertex. The fluid in the ventricles is often slightly turbid, and there may be a deposit of lymph at the bottom of the cornea, or in the third or fourth ventricles. Even at this stage there is already some adhesion of the medulla to the cerebellum, and the foramina of Luschka and Majendie are more or less blocked by the exudation, so that the ventricles are distended by excess of fluid. On the spinal cord the exudation is most marked in the lumbar region, and more on the posterior surface than the anterior.

In the cases which die at the end of three or four months all exudation may have already disappeared, and only thickening and opacity of the meninges remain, with matting of the medulla to the cerebellum by adhesions, so that extreme distension of the ventricles has resulted. After such subsidence of the meningitis a fresh outbreak of acute inflammation may occur. The *post-mortem* appearances then show a combination of past and recent meningitis. This observation is in accordance with the bacteriological evidence that even where the meningitis has subsided, in rare cases the specific micro-organism is still present in the lateral ventricles at least three months after the onset of the disease.

DIAGNOSIS.—Persistent head-retraction with vomiting in an infant is strong evidence of posterior basic meningitis. The conditions most often confused with it are tubercular and suppurative meningitis.

(1) Posterior basic is distinguished from tubercular meningitis chiefly by the much more marked and persistent retraction of the head, but also by the earlier age at which it occurs—tubercular meningitis is rare under six months of age, and occurs most often in the second year—its more acute onset, the slightness or absence of ocular paralyses, the absence of paralysis of other cranial nerves, the presence of blindness without optic neuritis, and the much slower course. (2) From suppurative meningitis it is distinguished not only by the marked retraction of the head, and much longer duration, but also by the absence of any obvious focus of infection. Suppurative meningitis is usually fatal in a few days, and is almost always secondary either to some pneumococcal lesion, pneumonia or empyema, or to ear-disease or some general pyæmic condition. (3) From epidemic cerebro-spinal meningitis the posterior basic variety has been distinguished by its special incidence on the earliest months of life, the absence of any tendency to become epidemic, the absence usually of the herpes and rashes which are often a marked feature in the epidemic disease, the absence also of the affections of eyes and ears, and lastly the less acute course. No great stress, however, can be laid on such differences, and in the writer's opinion there is little doubt that posterior basic meningitis should be regarded as a sporadic manifestation of the same disease.

Head-retraction does not necessarily indicate meningitis. It may be due to reflex irritation from ear-disease or from teething. Infants also with severe respiratory disease sometimes keep the head thrown back, apparently to assist the extraordinary muscles of respiration. The withdrawal of cerebro-spinal fluid by lumbar puncture has been advocated as a method both of diagnosis and of treatment. No amount of care, however, can render such a method entirely free from risk; and although it may occasionally be advisable, it cannot be recommended as a routine practice.

PROGNOSIS.—This form of meningitis is much less hopeless in its prognosis than tubercular or suppurative meningitis. Recovery occurs in about 10 per cent. of the cases. With recovery, however, there may be mental impairment, which may or may not be associated with some degree of hydrocephalus. A fatal result may occur in the acute stage, three to five weeks after the onset. In nearly half the cases, however, death does not occur until the third or fourth month of the illness.

TREATMENT.—The administration of mercury, either by mouth or by inunction at the back of the neck, or over the abdomen, is worthy of trial. In some of the cases which have recovered this method of treatment has been adopted, and it is possible that it may hasten the absorption of the inflammatory exudation.

Potassium iodide has been used with the same object; but as aggravation of the vomiting has appeared to be due to it, this drug should be given with caution.

The application of ice to the back of the head and neck may have some effect in controlling the inflammation, certainly it seems to check the vomit-

ing in some cases. The use of blisters or other counter-irritants cannot be recommended. They are of very doubtful value, and easily cause troublesome ulceration in the delicate skin of an infant. Pain must be relieved by opiates if necessary. If vomiting is persistent, trial may be made of dilute hydrocyanic acid, in doses of quarter to half a minim for an infant six months old. Feeding is often difficult, and it may be necessary to feed through a nasal or oesophageal tube. Every effort should be made to maintain nutrition, and in the chronic stage, and especially during convalescence, malt-extract or cod-liver oil may be useful. The spontaneous subsidence of the meningitis, and the fact that the later symptoms appear to be due chiefly to mechanical obstruction of the cerebro-spinal passage by adhesions causing hydrocephalus, have raised hopes that it may be possible to establish an artificial drainage of the ventricles by operation, and so to save life. In one case, recorded by Lees, an apparently permanent cure was obtained by the method of subdural drainage; but in other cases treated by this internal drainage, or by the earlier method of external drainage, the results have been disappointing: in many of them a fatal ending, with sudden hyperpyrexia, has followed within a few hours after the operation.

G. F. STILL.

5. Pachymeningitis Hæmorrhagica Interna.—SYNON. : *Hæmatoma duræ matris*; Subdural false membrane; Arachnoid cyst; Fr. *Pachyméningite*; Ger. *Pachymeningitis*.

DEFINITION.—A deposit lying between the dura mater and the pia-arachnoid, and consisting of actual blood or blood-clot, or of the more or less organised remainder of one or more previous hæmorrhages, or possibly of abnormal constituents of the subdural fluid which have transuded from the blood-vessels of the pia-arachnoid or dura mater.

GENERAL ÆTIOLOGY.—This deposit is almost confined to certain varieties of insanity associated with dementia, and to general paralysis of the insane. The frequency with which it occurs is variously stated by different observers. According to Bevan Lewis, it exists in 5·2 per cent. of all cases of insanity, while Wigglesworth found it in 8·47 per cent. of his cases, and Ford Robertson in 25·5 per cent. From the records of the first 1,626 autopsies performed at Claybury, the writer has found a percentage of 9·76 of the males and 5·54 of the females, or 7·75 of the whole of the cases. In the last 300 cases which the writer has himself observed, the percentage has been much higher than this. A subdural deposit rarely occurs in sane individuals, but at times it is found in children, usually as a result of injury, and in advanced age or after certain exhausting diseases such as the specific fevers and the diseases of the blood.

PATHOLOGY AND MORBID ANATOMY.—The occurrence of subdural deposits has till recently been explained on one of two hypotheses. The first, that the deposit is inflammatory in origin, may be a true explanation of certain traumatic cases; the second, that it is due to an effusion of blood into the subdural space, is largely accepted by alienists in this country, and is supported by the fact that injection of blood into the subdural space of animals may result in the formation of a typical false membrane. According to Wigglesworth, subdural hæmorrhage is often compensatory, in association with wasting of

the hemispheres and general or localised congestion of the meninges. More recently G. M. Robertson (1893) has suggested that a sudden lowering of intracranial pressure is an important factor in causing rupture of the dural vessels. Still more recently Ford Robertson has minutely described certain degenerative conditions of the dura mater in the insane which he considers to be important factors in the production of subdural deposits, though he traces these deposits in some cases to hæmorrhage from pial veins. It has been proved by the writer that the morbid appearances inside the skull-cap, which occur in many cases in ordinary insanity, namely, chronic degeneration of the dura mater, excess of intra-cranial fluid and chronic thickening of the pia-arachnoid, &c., are the macroscopic equivalents of, and vary in degree with, the amount of dementia existing, and are independent of the duration of the insanity. These morbid appearances are all especially evident in general paralysis, which is an acute progressive dementia, and in which subdural deposits are especially prone to occur. With reference to the occurrence of these deposits, the most important factor is probably the excess of intra-cranial fluid, or 'hydrops ex vacuo,' which occurs in dementia. This fluid has in general paralysis been shown by Halliburton and Mott to contain choline, and it is certainly abnormal in many cases of ordinary insanity, and is occasionally blood-stained. Externally this fluid interferes with the normal relations between the dura mater and the pia-arachnoid. Especially in the regions where stagnation is most likely to occur, namely, in the anterior and middle fossæ, the former membrane undergoes chronic degeneration, resulting in roughening of the surface, &c. The pia-arachnoid, chiefly over the frontoparietal region, where most of the neuronc degeneration has occurred, makes a hopeless attempt at the formation of replacement- or scar-tissue. Lastly, inside the ventricles, a similar morbid condition to that of the dura, namely, granularity of the ependyma, results, and this also occurs chiefly in the regions most subject to stagnation of the cerebro-spinal fluid, as in the lateral ventricles behind the foramina of Munro, and the lateral sacs and the calamus of the fourth ventricle. This granularity is probably allied to the usual degenerative condition of the ependyma of the central canal of the spinal cord in adults, aggravated by contact with abnormal cerebro-spinal fluid.

The morbid conditions above described are the physiological results of the loss of cerebral substance caused by the neuronc degeneration of dementia reacting on the mechanical conditions existing within the skull-cap. In the dementia of general paralysis the liability to blood-effusions is increased by the epileptiform seizures which are an almost constant symptom of the disease, and which necessarily cause considerable alterations in intra-cranial tension, and the same remark applies also to certain of the insanities associated with epileptiform seizures. Blood having been effused inside the skull-cap, between the dura and the pia-arachnoid, it will, if small in amount and on the convexity, be deposited on the dura of the vertex if this be roughened, or on the basal dura, which is so frequently in a degenerate condition. If the effusion be larger in amount, the parts on which it is deposited will depend largely on gravity, and it is consequently found to a greater or a less extent in the base of the skull above the tentorium. If, finally, the effusion be sudden and

large, or occur in successive small quantities, the blood, as a rule, clots *in situ*. This condition usually exists at the vertex, where, owing to the arrangement of the veins, venous congestion is likely to occur, and to result in rupture of one or more of the pial veins. The result, if the patient recovers from the immediate effusion, is a deposition in the subdural space of thinner or thicker brown films which usually adhere to the dura, but are readily detachable from it; of semi-organised membranes or cakes of blood, which in many cases contain semi-fluid material; or even of calcareous masses. In some cases, on removing the dura, the whole vertex is covered with a thin transparent membrane which may be ballooned out by fluid, and resemble an additional arachnoid. This membrane may even line the whole cavity of the cranium, and extravasations of blood may occur between it and the dura mater, here arising from dural vessels, or between the membrane and the pia-arachnoid, owing to rupture of pial veins. In the case of the thinner transparent films and membranes, it is possible that they arise by deposition, on the degenerated surfaces ready for their reception, of abnormal constituents of the cerebro-spinal fluid, which may be due to neuronie degeneration or to simple transudation from the altered vessels under the abnormal intra-cranial conditions which exist. The occasional cases where no subdural deposit exists, but in which the subdural fluid is blood-stained, and the numerous instances in which old or recent hæmorrhages into the pia-arachnoid exist alone, or in conjunction with the subdural deposit, are probably either conditions prior to the development of a deposit, or may be of a similar nature and have a similar origin. Under certain circumstances similar deposits may occur as a result of accidental hæmorrhages in the sane. The conditions are, however, different, for if the effusion be small, the dura and pia-arachnoid being normal, the blood will mix with the cerebro-spinal fluid and be flushed out by it; or if it exist within the pia-arachnoid it may form hæmatoidin in this position without affecting the dura. If, however, the effusion be large, it will cause symptoms of intra-cranial pressure, owing to the relative absence of cerebro-spinal fluid from the healthy cranium, and will probably either kill the patient or be removed by operation. In dementia, on the other hand, extra-cerebral effusion of blood is prone to occur; the dura is in a degenerate condition which offers a surface for the deposition of the blood; and, lastly, the excess of cerebro-spinal fluid which is present allows space, without increase of intra-cranial pressure, for even enormous effusions of blood. It is hence probable that the *chief causes of subdural deposit* are the degenerative conditions which develop inside the skull-cap during the progress of dementia, together with certain factors which produce a more or less marked condition of venous stasis, namely, epileptiform seizures and thrombosis of important venous channels. This is prone to occur owing to the anatomical arrangement of the veins entering the superior longitudinal sinus.

It is not improbable that slight injuries, &c., may give rise to a subdural deposit in many cases, especially as so many of the subjects are restless, feeble, and liable to fall and bruise themselves. The writer has recently seen a case of general paralysis in which, some weeks before death, the patient, from a fall, developed extremely marked bruising of the

forehead, eyes, and right temple. At the autopsy, a reddish-brown membrane was found in the right middle fossa and in both anterior fossæ of the skull. In another general paralytic, who died of extensive intra-cranial hæmorrhage, affecting the right anterior quadrant and the left posterior quadrant of the encephalon, marked and recent bruising of the right eye and the right temple as far back as the right parietal eminence existed. In a third patient, suffering with epilepsy, who died of erysipelas affecting the right eye, the right temple and the neighbouring parts, a recent subdural deposit of blood existed in the region corresponding to the external lesion.

The thinner membranes usually occur in very old persons, or in association with such exhausting diseases as phthisis and asylum dysentery.

In patients not insane, subdural hæmorrhage may result from injuries during birth, or it may develop in young children from simple strain, as in whooping-cough, &c., or later in life from blows or falls. It has also been described as occurring in the old and feeble, especially when these patients are suffering from exhausting diseases.

SYMPTOMS.—But little can be said concerning the symptomatology of subdural deposit owing to the rarity with which it is diagnosed *ante mortem*. The symptoms occurring in a typical case of the disease are described on the plate facing p. 576. For the symptoms of subdural hæmorrhage in the sane, the reader is referred to the article MENINGES, CEREBRAL, Hæmorrhage into.

DIAGNOSIS.—In cases of general paralysis the seizures and the general mental condition of the patient, as a rule, completely obscure the symptoms due to subdural hæmorrhage. In the fairly numerous cases where rupture of a pial vein follows intense venous engorgement, due to thrombosis of the superior longitudinal sinus, the great anastomotic vein, or other important venous channels, the general symptoms due to the thrombosis would, as a rule, obscure those due to the hæmorrhage. In the less severe cases, and also in those patients who suffer from the epileptiform seizures which frequently occur with degeneration of the cortical arteries, or result from old-standing or slowly progressive lesions of the cortex, the occurrence of subdural deposit will not even be suspected.

PROGNOSIS.—Relatively few patients die directly from subdural hæmorrhage. In the majority of cases, death ensues from the primary disease or from some intercurrent complication.

TREATMENT.—That of ordinary apoplexy. See also the following article.

J. S. BOLTON.

6. Hæmorrhage into Cerebral Meninges.

SYNON.: Fr. *Apoplexie Mèningée*; *Hémorrhagie Mèningée*; Ger. *Hirnhautblutungen*.

DEFINITION.—Effusion of blood in one or other of the following situations: (1) Between the bone and the dura mater; (2) between the dura mater and the arachnoid (into the so-called 'arachnoid sac'); or (3) beneath the arachnoid and into the meshes of the pia mater.

ÆTIOLOGY.—The first of these varieties of meningeal hæmorrhage has an almost exclusively traumatic origin; being a result of falls or blows which occasion the rupture of one of the meningeal arteries lying between the bone and the dura mater. Caries of the bone may in very rare cases lead to

such a hæmorrhage by causing erosion of one of the meningeal arteries.

The other two varieties are not so distinctly separated from one another, since a hæmorrhage occurring in the pia mater, if large, is very apt to break through the arachnoid, and thus lead to effusion of blood into the 'arachnoid sac;' and this whether the primary effusion has been the result of a traumatic injury, or is a sequela of some general or local disease. Effusion into the arachnoid may also occur as a result of rupture of some vessel on the inner surface of the dura mater; this being probably a rare consequence of injury. *See also* PACHYMENINGITIS HÆMORRHAGICA, p. 985.

Effusions of blood are occasionally found beneath the arachnoid which have not originated there, but which have come to the surface, by laceration of brain-substance, from some intracerebral hæmorrhage; or they may have been caused by intraventricular hæmorrhages, finding their way into the fourth ventricle, and thence into the subarachnoid tissue of the pons and cerebellum.

In very young children, whose vessels are presumably healthy, bleeding into the arachnoid may occur from any unusual amount of strain. This occasionally takes place at the time of birth, especially during prolonged labours. Indeed, according to Cruveilhier, arachnoid hæmorrhage is the cause of the death of about one-third of those infants who die almost immediately after birth; and is of extreme frequency in cases in which the labour has been difficult or protracted (Spencer). A little later on in life, a similar accident may occur during paroxysms of whooping-cough, or during other spasmodic respiratory conditions, in which the return of venous blood from the head is impeded. Later still, an arachnoid hæmorrhage not infrequently follows a fall or blow upon the head, or it may result from the rupture of an aneurysm on one of the larger vessels about the base of the brain—especially the basilar or one of the middle cerebrals. Small subarachnoid hæmorrhages, often multiple, are not infrequently produced by the occurrence of thrombosis in the superior longitudinal sinus. They may also occur in persons suffering from scurvy or leucocythæmia. Lastly, they may be met with as one out of the many forms of lesion occurring in patients suffering from general paralysis of the insane.

Meningeal hæmorrhages are decidedly more common in males than in females—in the proportion of about three to one. They do not, however, like cerebral hæmorrhages, occur with progressive frequency as age advances, but are much more uniformly distributed through the different decades of life.

ANATOMICAL CHARACTERS.—When death takes place soon after blood has been effused into the arachnoid as well as in the other situations, it is found in an easily recognisable condition. This is by no means the case, however, after the lapse of many months or even years; then, in the case of small hæmorrhages, we may meet with mere yellowish or rust-coloured stains; while where they have been of larger size, we may find decolorised cyst-like bodies, either free or adherent—or else there may be decolorised membranous masses, adhering mostly to the parietal arachnoid. Where the size of the clot has been large, the surface of the brain is more or less pressed upon, so that some atrophy of its substance follows. *See also* PACHYMENINGITIS HÆMORRHAGICA, p. 985.

SYMPTOMS.—The symptoms attendant upon meningeal hæmorrhage will necessarily vary a great deal in severity, according to the amount and suddenness of the effusion. These symptoms are, moreover, in the great majority of the traumatic cases obscured by those depending upon the mere shock and concussion of the brain, which the original accident or blow occasions.

Where subarachnoid hæmorrhages occur in the course of thrombosis of the longitudinal sinus, no distinctive symptoms are as a rule produced; and those of the primary affection are themselves only too variable and difficult of recognition. Again, where subarachnoid hæmorrhages occur in the course of purpura, leucocythæmia, or allied affections, the amount of blood effused is usually too small to produce definite or recognisable symptoms. At most, the abrupt onset of pain in the head, vertigo, or mental confusion, may give rise to a suspicion that such an event has occurred.

Where a large hæmorrhage takes place beneath and into the arachnoid sac, over one hemisphere, or over both, either as the result of a fall or blow, or from the bursting of an aneurysm on one of the large arteries at the base of the brain, a profound coma is produced which may prove rapidly fatal—that is, in the course of a few minutes or a few hours. Where the amount of blood effused is less, and where it is poured out more gradually at first, there may be premonitory symptoms, in the form of sudden headache, vertigo, mental confusion, vomiting, or convulsions, rapidly followed by unconsciousness. At first there is generally complete relaxation of all the limbs; but later—after some hours or days—the weakness may be distinctly unilateral, that is, of hemiplegic type, though sometimes with very slight implication of the face. There may also be twitches or rigidity of the limbs on one or both sides. On recovery of consciousness there may be no distinct loss of sensibility, only numbness, in the limbs; and the paralysis may after a time grow less up to a certain point, or gradually disappear.

DIAGNOSIS.—In many of the slighter forms of hæmorrhage into the cerebral meninges diagnosis is, for the reasons specified, almost impossible.

In the more severe cases a sudden apoplectic attack is produced, agreeing very closely with that occasioned by some of the most serious forms of intracerebral hæmorrhage. Causal conditions, especially when they have been traumatic, together with the possible youth of the patient, may in some cases help us to diagnose a large arachnoid hæmorrhage from a copious bleeding into the lateral ventricles, or from a sudden hæmorrhage into the middle of the pons Varolii; though it should be borne in mind that in the former of these two conditions the pupils are almost always widely dilated, while in the latter they are as constantly contracted and insensitive; whereas they are likely, so far as the writer's observations have gone, to be in a more intermediate condition in arachnoid hæmorrhage.

PROGNOSIS.—In the case of arachnoid hæmorrhages, whether large or of only moderate volume, should the patient survive the first effects of the effusion (and, it may be, of the injury which caused it), danger to life is no longer to be feared. The only question then is as to the amount of paralysis, mental impairment, or of irritability with cephalalgia, which may remain; or whether or not a

tendency to convulsions may be set up, as a consequence of the original injury and lesion.

TREATMENT.—The treatment of a case of meningeal hæmorrhage does not differ from that appropriate for cerebral hæmorrhage. Perfect rest in the recumbent position, with the head slightly raised, is essential. Cold to the head may be conjoined with hot applications and mustard plasters to the lower extremities. For other indications and details of treatment we must be guided by the varying conditions of the patient. In some cases (especially where the hæmorrhage has been between the dura mater and the bone) the aid of the surgeon has been sought, who, by trephining and giving exit to much of the extravasated blood, has either cured or greatly mitigated the condition of the patient. During convalescence, in the more favourable cases, we must pay great attention to the general health, and above all protect the patient from overwork or excitement of any kind.

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7. Traumatic and Secondary Spinal Meningitis.—**ÆTIOLOGY.**—In the case of the cerebral meninges, inflammation as a result of traumatic injuries is more common than as a phenomenon secondary to disease of the bone or of the scalp. The proportional frequency of these modes of causation is, however, somewhat reversed in the case of the spinal meninges; partly because the head is more liable than the spine to suffer from direct injuries, and partly because disease of the spine and of adjacent parts occurs with considerable frequency in such a manner as to be capable of exciting a secondary inflammation of the spinal meninges. Among the various efficient *traumatic* influences may be mentioned fractures and dislocations of the vertebræ, and stabs or other penetrating wounds implicating the contents of the spinal canal; while among the most frequent morbid conditions, in the course of which there may be a *secondary* development of spinal meningitis, we must cite the following: Caries and tubercular disease of the vertebræ; syphilitic gummata; deep sloughing bed-sores in the sacral region; cancer of the vertebræ; and, more rarely, inflammation of some part of the thoracic or abdominal parietes contiguous to the spinal column, and capable of spreading to the spinal canal from within.

ANATOMICAL CHARACTERS.—In all these cases the signs and products of inflammation may be found in one or other, or in both, of two situations; that is, either implicating the dura mater, principally on its external surface, when we have the condition commonly known as *spinal pachymeningitis*; or affecting the surface of the arachnoid so as to produce a *spinal arachnitis*. Thus the same kind of limitation in the distribution of the inflammation is apt to occur, when it starts under the influence of such causes, as is found to obtain in regard to the traumatic or secondary inflammations of the cerebral meninges. Perhaps there is in the case of inflammation of the spinal membranes, however, a rather more distinct tendency for such inflammations to spread, so as to involve the subjacent pia mater, than is the case in the parallel inflammations of the cerebral meninges.

In *spinal pachymeningitis* the dura mater itself is thickened and more vascular than natural, this being seen more especially on its outer surface; and both it and the surrounding connective tissue are

covered or infiltrated either with yellowish lymph-like matter, or with actual pus. The internal surface of the dura mater may also be more or less covered with inflammatory products. The nerve-roots passing through the membrane are likewise generally affected by the inflammatory process, and they may show signs of compression or even of atrophy. Such inflammation may be either limited to the region of two or three vertebræ, or it may affect more or less the whole length of the spinal membranes.

A more chronic form of spinal pachymeningitis has been observed by Charcot and others, affecting principally the inner layers of the dura mater in the cervical region. In this condition, which is described by the author above named as *pachymeningite cervicale hypertrophique*, there seems to be a considerable hyperplasia of tissue-elements in the inner layers of the dura mater, which is apt to develop into an overgrowth of almost cicatricial hardness, often made up of concentric laminæ. These are frequently adherent to the arachnoid and to the pia mater, which also become more or less thickened. In these latter cases, especially, not only are the spinal nerve-roots greatly damaged, but the spinal cord is itself more or less compressed and softened, so that distinct paralytic symptoms, with muscular rigidities or atrophy, are apt to be produced.

Where *spinal arachnitis* is superadded, or when it exists alone, we find that pus or lymph is situated on the outer surface of the visceral arachnoid, and also to a less extent on that lining the dura mater. The combination of the two conditions is rather more frequent than the existence of arachnitis alone. It is important to remember that all these forms of inflammation are very rarely, if ever, primary and idiopathic (with the exception of the more chronic variety described by Charcot), but that they occur as consequences of injury, or of certain forms of disease adjacent to the spinal canal.

SYMPTOMS, PROGNOSIS, AND TREATMENT.—As the nerve-roots are affected in these forms of inflammation, as well as in the idiopathic meningitis which implicates the pia mater (*spinal leptomeningitis*), and as the symptoms of both sets of affections are in great part dependent upon this, and are therefore in many respects similar (and by no means always capable of being accurately discriminated from one another), it would serve no useful purpose to dwell upon the symptomatology and treatment of spinal pachymeningitis and arachnitis alone. The reader is, therefore, referred to the corresponding sections in the next article.

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8. Idiopathic and Tubercular Spinal Meningitis.—**SYNON.**: Simple and Tubercular Spinal Leptomeningitis.

Simple spinal meningitis of idiopathic origin, and tubercular spinal meningitis, are affections so closely related to one another, both in their clinical and pathological aspects, that no advantage whatsoever would be derived from considering them separately. In each case we have to do with an inflammation involving the spinal pia mater, so that the products of inflammation are situated beneath the arachnoid membrane. In order to distinguish these from other forms of meningitis, such as pachymeningitis and arachnitis, it is desirable that we should use some special term, such as *leptomenin-*

gitis, which is now employed as a distinctive appellation for an inflammation affecting the pia mater, whether cerebral or spinal.

In regard to the extent or area of this kind of inflammation, it must be said that the tubercular variety always involves the presence of a similar inflammation at the base of the brain, though the contrary position is not true—that is to say, the tubercular inflammation may exist at the base of the brain alone, without involving the spinal meninges. Of the non-tubercular forms of spinal leptomeningitis there are two varieties, and of these one form always involves the membranes at the base of the brain and the spinal meninges simultaneously (*see MENINGITIS, EPIDEMIC*, p. 992). The other form may or may not simultaneously involve the membranes at the base of the brain, so that we have in these cases either a 'simple sporadic cerebro-spinal meningitis,' or a 'simple spinal meningitis.'

Whenever the inflammation has a 'cerebro-spinal' distribution, no confusion is involved by retaining the use of the simpler term 'meningitis,' as it is generally understood that forms of inflammation having such a distribution involve the pia mater especially. But in place of the name 'spinal meningitis,' if we mean to imply that the inflammation affects the same tissue, it is best to use the more special and distinctive term 'spinal leptomeningitis.'

From what has been said above, it will be understood that the symptoms resulting from meningitis involving the base of the brain alone, or together with serous effusion and softening of the walls of the ventricles (which, as we have seen, so frequently co-exists with inflammation of the membranes in this situation), have been principally studied in the purely cerebral forms of tubercular meningitis. On the other hand, the symptoms resulting from spinal leptomeningitis are best studied in the simple forms of this disease. It will also be evident that the simple and the tubercular forms of cerebro-spinal meningitis are likely to agree to some extent in their symptomatology with that of the disease known as 'epidemic cerebro-spinal meningitis.' *See* p. 992.

ÆTIOLOGY.—Spinal leptomeningitis is most prone to occur in children and in young persons; and is more frequent in males than in females. Persons who are badly fed, and live under very unfavourable sanitary conditions, are more liable to be attacked than those who are healthy and surrounded by opposite conditions.

For the tubercular form the exciting causes are all such influences or conditions, whatever they may be, as determine the outbreak of acute tuberculosis. The affection of the spinal meninges may be either an extension of the inflammation originally existing at the base of the brain alone, or it may be another independent manifestation of the general disease developing within the spinal canal simultaneously with the cerebral meningitis. *See* MENINGES, CEREBRAL, Inflammation of, Tubercular.

For the simple or non-tubercular form, the exciting causes are various, but the best established of them would seem to be these: Exposure to cold, or cold and wet, in various forms; certain acute diseases, or the period of convalescence therefrom; concussion of the spine, as from falling down stairs, or in other ways; wounds affecting the spinal cord or its membranes, as in stabs of various kinds; or fracture and dislocation of the vertebrae.

The last modes of causation mentioned are simi-

lar to those which obtain for spinal pachymeningitis and arachnitis; for, although these latter conditions may be excited alone under such traumatic influences, they may also in certain cases, and especially arachnitis, be excited in association with a spinal leptomeningitis. Precisely the same kind of thing has also to be said in regard to the occasional action of other causes, such as caries of the vertebrae, deep, sloughing bed-sores in cases of paraplegia, or other instances of inflammatory processes contiguous to the spinal canal. Any of these latter conditions may also set up a leptomeningitis, in association with one of the other forms of meningeal inflammation.

A spinal leptomeningitis may spread so as to implicate the base of the brain; or a cerebral basal leptomeningitis may subsequently implicate the spinal membranes; or, lastly, the inflammation may appear in both regions simultaneously, and thus be from the first cerebro-spinal in seat. The writer has of late seen several cases of the tubercular variety belonging apparently to this latter category; but until the spinal canal has been regularly opened for some time in necropsies of persons dying from this disease, we shall be unable to say what is the exact numerical proportion of such cases as compared with those which are simply cerebral in type. The bulb may be comparatively free from lymph, and yet an inflammation of the spinal meninges may be well-marked. There must, therefore, be a routine opening of the spinal canal for the decision of this question, and not a mere casual inspection of its upper extremity through the foramen magnum.

ANATOMICAL CHARACTERS.—According to the stage of the disease at which death takes place, we may meet with the inflammatory process in one or other of three different stages: (1) that of greatly increased vascularity of the spinal pia mater; (2) one in which, in addition to the increased vascularity, gelatinous serum, lymph, or pus exists in the meshes of the pia mater, often more marked in amount along the posterior columns. This latter is the condition commonly met with; but in rare cases, where patients have survived an acute attack, we may find (3) certain residuary chronic changes in the form of thickenings, opacities, and undue adhesions of the pia and arachnoid to the spinal cord, which perhaps may itself show a more or less marked condition of peripheral sclerosis.

In the tubercular variety we frequently have to do with a mere gelatinous serum, or thin greenish-yellow lymph (similar to that met with at the base of the brain), rather than with actual pus, in the meshes of the pia mater. Careful scrutiny of the vessels in the anterior fissure and in other parts may also show the characteristic 'granulations,' in the form of opalescent, whitish, or yellowish-white specks.

In both forms of the disease the *nerve-roots* are implicated in various ways. They are usually involved in the inflammatory process, and may be much pressed upon by lymph and other hyperplastic products. The nutrition of the cord itself is probably profoundly altered, owing to the existence of an inflammatory process affecting the network of vessels from which its blood-supply is derived; and, moreover, the organic continuity existing between the pia mater and the offshoots of connective tissue which extend into it on all sides, around the blood-vessels that penetrate its substance, makes it easy

for the inflammatory process to invade the substance of the cord itself.

SYMPTOMS.—General listlessness and a sense of chilliness have been noticed as premonitory symptoms in some cases of spinal meningitis. At other times the disease has been observed to commence with a more marked feeling of chilliness, accompanied or quickly followed by some febrile elevation of temperature, together with a full, rapid pulse. Soon there supervenes a deep-seated boring pain in the back, varying in situation according to the degree of intensity of the inflammatory process at different levels. Pains may also extend round the body in girdle fashion, and likewise into the limbs. While the pains in the back are more or less continuous, though greatly aggravated by all attempts at movement, those felt in the limbs and trunk may be only experienced when attempts to move are made. Movement excites the dorsal pain far more than pressure upon the vertebral spines, or light tapping over the same region.

Rigidity of the spine, from muscular spasm, either localised or general, and also rigidity of the limbs, or even of special muscles, may co-exist with the pains in the back and limbs. There is often an exaltation of reflex movements in the early stages of the disease, though this condition is not nearly so well marked as it is in tetanus. 'Kernig's sign' may also be present (p. 820).

At the same time marked hyperæsthesia of the skin exists over considerable regions of the trunk and extremities. The patient cannot bear to be touched, however lightly; and still less can he endure to be moved. He is irritable or plaintive if these proceedings be attempted. Owing to the varying nature and extent of the spasms, and the different degree of pain endured, the position assumed by the patient is very various in different cases.

Difficulty in defecation and in micturition often exists, especially in the early stages of the disease, and this is supposed to be due to a spasmodic condition of the sphincters. The respiration and the heart's action are principally interfered with in cases where the cervical meninges are gravely involved.

The temperature seems to pursue a somewhat irregular course, but concerning this further information is needed. It may be only slightly above the normal; and may not rise much beyond 102° , even in fatal cases, till near the end. Then it may rise considerably in the course of a few hours; while in other cases it may at this same period become depressed below the normal.

In the later stages of the disease some amount of paresis, or actual paralysis, may be noted in one or more limbs; the pains on movement and the cutaneous hyperæsthesia become less, or may indeed be intermixed with tracts in which actual anaesthesia exists. The bladder may at last be paralysed; and respiration may be most gravely interfered with, so that disturbance of this function, as well as of the heart's action, may be the actual cause of death.

These symptoms are, in all probability, as Erb maintains, due in very great part to the inflammatory and other changes by which the anterior and posterior nerve-roots are implicated. Others may be due to extensions of the inflammatory process to the substance of the spinal cord, thus leaving a somewhat uncertain minority of symptoms to be accounted for by the mere implication of the pia mater itself.

The grouping of symptoms is apt to vary much in different cases, according as there is or is not the co-existence of a cerebral meningitis; or, in the absence of this complication, according as the inflammation is more or less localised in different regions of the cord, or general in its distribution. Much will depend also upon the severity of the process, and upon the extent to which the substance of the spinal cord becomes involved in the course of the disease.

DIAGNOSIS.—Fever; pains in the back and limbs, greatly aggravated by movement; together with stiffness of the neck, trunk, or limbs, including 'Kernig's sign'; local muscular spasms; hyperæsthesia of the skin; retention of feces and urine; dyspnoea; with a tendency in the later stages to the supervention of paresis, or actual paralysis of limbs—these are the symptoms, the combination of which to a marked extent becomes almost typical of spinal meningitis.

Its complication with a basal cerebral meningitis is, among other signs, chiefly indicated by the occurrence of vomiting, headache, retraction of head and neck, slight delirium or stupor, paralysis of ocular muscles, difficulty in deglutition, loss of speech, or convulsions. The presence of many of such symptoms may, from their great importance, tend to dwarf or obscure those due to the inflammation of the spinal meninges alone; on the other hand, if they are absent we may feel assured that the inflammation has not also involved the base of the brain.

The fact that a meningitis is spinal in seat, and unaccompanied by cerebral symptoms, is of itself exceedingly good evidence to prove that it is not the tubercular form of the affection.

To settle the question, which membranes of the cord are inflamed in any given case, we must be guided chiefly by what we can learn concerning the causal conditions and the distribution of the inflammation, rather than by any at present known differences in the grouping of symptoms. Thus inflammations of idiopathic origin, or those which are cerebro-spinal in seat, will almost invariably be found to be instances of leptomeningitis; while those set up as a result of caries of the vertebræ, or as a sequence of a sloughing sacral bed sore, are certainly much more prone to take the form of pachymeningitis, or of this in combination with arachnitis.

In reference to the diagnosis of spinal meningitis from other affections, it may be said that a very slight amount of attention to the nature of the pains and attendant conditions will suffice to avoid the mistake of supposing them to be rheumatic in nature. Similarly the absence of trismus in the early stages, and of any extremely well-marked exaltation of reflex excitability, together with the presence of severe pains in the back and limbs, will be negative and positive characters sufficient for distinguishing spinal meningitis from tetanus.

Another disease with which spinal meningitis is liable to be confounded is acute softening of the spinal cord. But the distinction should be easy in the early stages; and the history of the course of the affection will guide us later on, when symptoms of actual paralysis may have become developed. Still, in certain cases, a spinal meningitis may entail a softening of the cord to a marked extent, and then the symptoms of the primary affection will gradually be merged in those of the other which it induces.

A very rare condition, once met with by the pre-

sent writer, is, he thinks, almost impossible to distinguish from spinal meningitis—that is, where a sarcomatous or carcinomatous new-growth springs up rapidly throughout the spinal pia mater in the situation usually occupied by lymph or pus, especially when, as in the instance referred to, the disease seems to be the direct sequence of a fall from a height or over a flight of steps, and death takes place within a period of two or three months.

PROGNOSIS.—The prognosis of spinal meningitis depends a good deal upon the nature of the primary or causal conditions; upon the question whether the disease shows a tendency to extend to the cerebral meninges; upon the severity with which it implicates the cervical region of the cord; and also to some extent upon the age and general state of health of the person attacked.

Spinal meningitis is a disease which proves fatal in the course of a few weeks in a very large percentage of cases. Complete recovery is certainly a rare exception; but late and partial recovery—that is, after the disease has lasted long, and with the remainder of some amount of muscular atrophy or incurable paralysis—is a little more frequent. In such cases the disease after a time lapses into a chronic condition, and the patient very gradually recovers, except, perhaps, for such incurable sequelæ as are above mentioned. But even in these cases tending towards recovery, a relapse is most easily brought about, owing to the recommencement of the disease in an acute form.

Where spinal meningitis supervenes upon a sloughing bed-sore existing in a case of paraplegia, the end is usually not far distant. The gravity of any case of spinal meningitis is also always greatly enhanced when the disease spreads to the cerebral meninges. And, so far as the spinal meninges themselves are concerned, any great intensity of the inflammatory process in the cervical region is always of the gravest import, because of the liability to secondary implication of the cord itself in these regions, either structurally or functionally, and the bringing about from this cause of serious interference with the functions of respiration and circulation. A continuously rising temperature in such a case—to 105° and onwards—is also of fatal import.

TREATMENT.—The severity of spinal meningitis is apt to prompt to the use of active measures of questionable utility; among these may be cited free local blood-letting, the free application of ice to the spine, and active purgation. It is difficult, too, to say on what principle it is thought absolutely necessary to apply cold when we have to do with an inflammation within the spinal canal or within the cranium, while we almost always apply heat externally in the case of an inflamed pleura, an inflamed peritoneum, or even an inflamed area of skin. Probably the application of ice in such cases tends to alleviate pain, so that where this is great its use may bring much relief to present suffering, when hot applications would only aggravate it. But were it not for the fact that in meningeal inflammation (whether spinal or cerebral) increased fulness of vessels around sensitive organs shut in by unyielding walls almost necessarily leads to aggravation of pain, the application of heat would probably be more beneficial than that of cold, so far as the possible resolution of the inflammatory condition itself is concerned.

The patient should certainly be kept in a cool, quiet room, and lying either on his side, or, if pos-

sible, on his face, on a comfortable bed. He should be well supplied with fluid diet of the most nourishing description, together with eggs and a moderate amount of stimulants, according to the indications presented by his symptoms and general condition.

Tincture of iodine may be painted along each side of the spine in the affected region every second or third day. Pain should also be eased by opium or morphine; in fact, an opiate treatment may be resorted to in a large proportion of the cases. When opium and morphine do not agree, or are not admissible, Indian hemp would be worthy of trial as a mere anodyne; or we must fall back upon bromide of potassium and chloral hydrate, though the latter must be used with great caution where the heart's action is slow, irregular, and seriously interfered with. Belladonna and ergot have also been recommended, on somewhat doubtful grounds, as anti-inflammatory remedies in spinal meningitis.

We ought, in fact, to endeavour to combat the most urgent symptoms as much as possible, even if we cannot, by counter-irritants and by the judicious use of drugs, modify the course of the inflammation. Also by suitable feeding and judicious nursing we should endeavour to tide the patient through the disease. And if, happily, the activity of the inflammatory process subsides, the most unremitting attention will still be required to protect the patient against a relapse. Should his condition otherwise admit of it, the absorption of inflammatory products would, in this stage, be likely to be promoted by the use of a small dose of perchloride of mercury (such as one-sixteenth of a grain for an adult), in combination with increasing doses of iodide of potassium. At the same time, every effort must be made to restore the patient's general health, and to combat the emaciation which the disease itself usually involves.

H. CHARLTON BASTIAN.

Hæmorrhage into or upon Spinal Meninges.—**SYNON.**: *Hæmatorrhachis*; Meningeal Apoplexy (Spinal).

Effusions of blood upon, between, or beneath the spinal meninges are altogether rare events, contrasting notably in this respect with the comparative frequency of parallel conditions on the side of the cerebral meninges.

ÆTIOLOGY.—Among the causes of meningeal hæmorrhages, stabs, blows, or falls will hold a first rank. After these causes we should have to cite impediments to the circulation of blood, occasioned by various respiratory or muscular spasms, occurring either in the course of whooping-cough, or during some more than usually violent convulsive attack—epileptic, tetanic, or other. The lifting of heavy weights, or other great voluntary muscular exertions, may likewise at times prove causes of spinal meningeal hæmorrhage. Occasionally, however, it occurs independently of any such, or of other readily assignable causes.

ANATOMICAL CHARACTERS.—Fluid blood or blood-clots may exist in relation with the spinal meninges in three different situations.

The most frequent site of such hæmorrhage is (1) outside the dura mater, between it and the vertebral arches. Here large clots are sometimes found, wholly or more frequently in part surrounding the dura mater in the region in which the hæmorrhage has occurred. Where the effusion is large the cord itself may be distinctly compressed,

but even smaller effusions may produce some amount of compression of nerve-roots. A clot in this situation, as in other sites, will of course become much modified in appearance with age.

Clots and more or less fluid blood may also, but more rarely, be met with (2) inside the dura mater, within the so-called arachnoid sac. This occurs perhaps most frequently as a mere sequel of a similar hæmorrhage taking place in the cerebral meninges, the blood simply gravitating into the spinal canal. Sometimes, however, especially in cases of spinal pachymeningitis, blood is actually effused in this situation—and that where the internal surface of the dura mater is much more vascular than natural. The opening of a thoracic or abdominal aneurysm may also very rarely take place into the spinal canal, and thus produce sudden and grave compression of the spinal cord.

Much smaller extravasations of blood are also met with (3) beneath the arachnoid and within the meshes of the pia mater, over areas perhaps small in extent longitudinally but more or less embracing the cord in one or more regions. The cord or nerve-roots may, however, be decidedly compressed by such hæmorrhages, even when they are small in amount, owing to the space into which the effusion takes place being comparatively shallow.

SYMPTOMS.—The symptoms of these affections are in a large proportion of cases vague and ill-defined. They may be much obscured by the causal conditions. In other cases they will vary in distinctness according to the amount and abruptness of the hæmorrhage.

As a rule the onset of symptoms is sudden. Pain in the region of the spine, in which the hæmorrhage exists, or radiating thence along the nerves emanating from this region, may be the first symptom. More rarely muscular twitchings or spasms may exist, either alone or with pains. These symptoms, dependent upon irritation and compression of sensory and motor nerve-roots, are at other times almost wholly absent. There may then be as abiding symptoms mere numbness or tingling in the parts affected, together with a sense of weight and paresis in the limbs. Actual paralysis is rare; and even when it is present the rectum and bladder mostly escape.

Where pain exists there is often stiffness of the spine; and these in combination greatly interfere with movement. Febrile reaction is usually absent or very slight. The severity of the symptoms may abate after a day or two, leaving only more or less paresis. In the case of large hæmorrhages, however, with extensive compression of the spinal cord, death may be rapid, occurring in the course of some hours or of a day or two.

The symptoms will vary as the effused blood presses upon the cord in the cervical, the dorsal, or the lumbar region. Where the effusion is in the cervical region in a traumatic case, in which there is obvious head-injury with a condition of stupor, it is almost certain not to be diagnosed. The patient is not sensible enough to complain of pain; and the irregular respiration and small disordered pulse, with slight tremor or rigidity of one or both upper extremities, may with more probability be ascribed to multiple head-lesions—as actually happened in a case which recently came under the writer's notice.

DIAGNOSIS.—It may be impossible to diagnose hæmorrhage into the spinal meninges in cases where it occurs as a concomitant of other grave diseases—

such as tetanus, eclampsia, or cerebral hæmorrhage; and also in cases where it merely complicates a traumatic injury of the spinal cord itself. In other cases the presence of certain causal conditions, together with the abrupt commencement of spinal symptoms in such combinations as have been above referred to, is sufficient to enable us to diagnose it from hæmorrhage into the substance of the cord, as well as from meningitis or acute softening (*see SPINAL CORD, Diseases of*). The gradual onset of the symptoms arising from tumours of the spinal cord, or of the spinal meninges, makes it more easy to separate these affections from meningeal hæmorrhages.

PROGNOSIS.—Spinal meningeal hæmorrhages are as a class decidedly less grave than meningeal tumours. They are unlike the latter, moreover, inasmuch as the worst symptoms attendant upon them are produced at once, instead of being only very slowly evolved; so that after a short time, unless the blood effused happen to have produced a certain amount of compression of the spinal cord, the symptoms gradually diminish in severity. Large extra-meningeal hæmorrhages compressing the cervical region of the cord are by far the most serious forms of this affection.

TREATMENT.—In the treatment of spinal meningeal hæmorrhage the patient must of course be kept perfectly quiet and in the recumbent position. Spoon-diet should be administered for a few days; and vascular sedatives, such as aconite, may be given with advantage. Some recommend active purgation and the abstraction of blood from the neighbourhood of the spinal column by cupping or leeches. These measures, however, are of questionable utility, and the former especially might easily do positive harm.

In certain cases, especially in the extra-meningeal form of the disease, a surgical operation for the removal of the compressing blood-clot may be advisable.

H. CHARLTON BASTIAN.

MENINGITIS, Epidemic Cerebro-Spinal.—

SYNON. : Cerebro-spinal Fever; the Black Sickness (popular, Dublin); Spotted Fever; Cerebral Typhus.

DEFINITION.—An acute febrile disease, characterised by sudden invasion with extreme nervous shock, vomiting, excessive pain referred to the back of the neck and spine, spasmodic contraction of muscles, hyperæsthesia, and frequently cutaneous eruptions.

Cerebro-spinal fever occurs both in epidemic and sporadic form. The identity of the sporadic with the epidemic disease has been firmly established by the discovery of the specific micro-organism which is common to both. It seems also well-nigh certain from bacteriological investigation that the disease of infants known as posterior basic meningitis, which is by no means uncommon as a sporadic manifestation, is identical with the sporadic cases of cerebro-spinal fever.

ÆTIOLOGY.—*Age.*—In its epidemic form cerebro-spinal fever is most commonly met with in children beyond the age of infancy, and in young adults; it seldom occurs after the age of thirty-five years and is rare after forty. Sporadic cases of cerebro-spinal fever, considered apart from posterior basic meningitis, are probably most common in the later half of childhood and in early adult life; but if posterior

basic meningitis be included, then sporadic cerebro-spinal fever is probably much commoner in infancy than at any other period of life.

Sex.—Among adults the disease is much more frequent in males than in females; robust males between the ages of fifteen and thirty are its chief victims; but among children it seems to attack both sexes equally.

PREDISPOSING CAUSES.—The epidemic form seems specially to affect young recruits in the army, as was the case in the French epidemics. In Dublin it was specially severe among the recruits of the Royal Irish Constabulary. Probably this special incidence is to some extent related to the conditions under which soldiers live, for the epidemic form of the disease has occurred also in prisons, workhouses, and other public institutions, where crowding and defective sanitation were probable factors.

Cerebro-spinal fever is widely distributed in the temperate zone. It prevails more in cold than in hot weather, and is far more frequent in winter and spring than at any other time of the year.

Excessive fatigue seems to increase the liability to the disease; it has arisen after a hard day's hunting, foot-racing, long walks, dancing, or in children exhausted from outdoor play.

MODE OF SPREAD.—In epidemics it occurs in isolated localities far apart from one another; it does not seem to extend from a district to adjacent districts. The method of communication is uncertain; direct contagion is very rare, if indeed it occurs. The specific micro-organism has been found by several observers in the nasal secretion, and it is possible that dried-up secretion on handkerchiefs or dried-up expectoration may serve in some instances to spread the disease. The occurrence of an epidemic disease with purpura and meningitis in horses, during the prevalence of epidemic cerebro-spinal fever in some places, suggests the possibility of infection from animals.

BACTERIOLOGY.—The specific micro-organism of this disease is the *Diplococcus intracellularis* which was first described by Weichselbaum. As its name implies, it is found chiefly within the cells of the exudate. It stains easily with methylene blue or any of the ordinary stains; it is decolorised by Gram's method. It is aerobic, and grows well on glycerin-agar or blood-serum. In cultures it seldom lives more than two or three days. It has, however, been observed by Jaeger and others that this is not invariably the case: it occasionally lives two or three weeks or even longer. Until recently this micro-organism was confused with the pneumococcus, from which it should be carefully distinguished. As in other forms of meningitis a mixed infection occasionally occurs, thus the pneumococcus, staphylococcus, or streptococcus has been found in some cases with the specific micro-organism (Councilman, Mallory, and Wright). The *Diplococcus intracellularis* has been found not only in the meningeal exudation and cerebro-spinal fluid, but also in the blood during life (Osler), in the pus which occasionally forms in the joints, and in the nasal discharge which is frequent at the onset of the disease. This last discovery suggests that the nose may be the channel of infection in some cases.

SYMPTOMS.—The onset is almost invariably acute. In the most severe cases the patient when apparently in vigorous health is suddenly attacked by faintness, vomiting, and intense pain referred especially to the back of the head and neck; the

extremities become cold; the patient becomes insensible and convulsed; the limbs are rigid. Maniacal delirium may be a marked feature at the onset, and in a later stage the patient may fall into a dull apathetic condition.

In many cases some coryza with nasal discharge is present at the onset, and there is frequently also slight conjunctivitis at this stage.

Pain in the head, back of neck, and along the spine, is generally severe; the head is drawn back so as to be almost at a right angle with the spine; the whole back is sometimes arched as in tetanus; the muscles become rigid, and the skin extremely sensitive; neuralgic pains are also complained of in all parts of the body.

Paralysis of one or more limbs is sometimes present, usually of hemiplegic distribution, and most frequently attacking the arm. Paralysis of cranial nerves is less frequent than in tubercular meningitis; the ocular muscles are most often affected, so that strabismus is present.

The tendon-jerks are very variable, being sometimes increased, sometimes diminished.

Cutaneous eruptions appear in many cases within the first twenty-four hours. Herpes is perhaps the most common, and is sometimes very extensive; it occurs especially on the face, neck, and shoulders. Herpetic eruptions are met with as frequently in mild as in severe cases. A purpuric rash is present in some cases, especially in the more severe, and has been a marked feature in some epidemics. It usually appears first on the legs, and may be confined to the lower extremities. The spots are usually black, raised, about a line in breadth, and feel like a small shot under the skin. Sometimes these raised spots are surrounded by a dark purplish areola; in other cases large purpuric patches many inches in extent form on various parts, and may even coalesce and cover the entire body. Erythematous rashes are seen in some cases, and pemphigoid bullæ sometimes appear in the advanced stages of the disease.

Vomiting may be more or less persistent throughout the illness; the bowels are usually costive. Examination of the optic discs usually reveals nothing abnormal beyond slight congestion; optic neuritis is uncommon: amaurosis without obvious cause, a common symptom in the posterior basic meningitis of infants, would seem to be rare in the cerebro-spinal fever of older children and adults. The pulse commonly shows increased frequency, but only occasionally is irregular. The blood shows some leucocytosis. The temperature in cases where there is much initial prostration may remain low; the respiration then becomes of a sighing character, the pulse rapid with a peculiar jerky beat, giving a sharp upstroke to a sphygmographic tracing, and after a day or two, or it may be only a few hours, the patient dies in a state of collapse. More often the temperature rises and there may be continuous pyrexia, so that the chart resembles that of typhoid fever for two or three weeks; in other cases the temperature is of a remittent or intermittent type.

COMPLICATIONS AND SEQUELÆ.—After the acute stage is past, a persistence of symptoms, especially of vomiting and headache, may be due to the occurrence of hydrocephalus, which may also produce coma and convulsions and eventually prove fatal.

Ocular complications are frequent: the eye may be attacked by a low form of inflammation terminating

in a purulent infiltration of the whole or part of the organ : the cornea is more frequently involved than any other portion ; this sometimes gives way and the whole contents of the globe escape, causing hopeless loss of the eye. The sight is often permanently impaired by iritis or opacity of the cornea. In many cases, however, the inflammation completely subsides. It is remarkable that it is the right eye which is usually attacked, that both eyes are seldom affected, and rarely the left eye alone.

Deafness is not very common, but has been met with in several cases, and is sometimes permanent. Acute inflammation of the larger joints—sometimes an actual arthritis, sometimes a peri-arthritis—is a frequent complication in some epidemics, and has been observed also in the sporadic cases ; this may lead to suppuration in the affected joint.

Hæmorrhages are frequent in the more malignant forms, and are almost always present in cases where the purpuric blotches are of large extent : these hæmorrhages have occurred from the nose, uterus, bowels, kidneys, and ears, in about the foregoing order of frequency. Gangrene is occasionally met with, and the cases in which it occurs are usually fatal, but toes have been lost, and yet the patient has recovered ; the purpuric patches have also sloughed without serious danger to the patient's life.

PROGNOSIS.—The prognosis of cerebro-spinal fever depends much upon the form the affection assumes, and for convenience it may be divided into the following groups : (1) Cases of a very mild form, terminating in recovery ; the duration being usually from one to three weeks. (2) Cases of a very severe form, setting in suddenly, with violent and well-marked symptoms, purpuric spots and blotches, a tendency to hæmorrhages, deep collapse and coma ; usually terminating fatally in from a few hours to three days. (3) Cases of medium severity where all the nervous symptoms set in with less suddenness than in the cases just mentioned, with no purpuric blotches usually, and no hæmorrhages. These usually yield to treatment, and terminate in recovery in from two to six weeks. (4) Cases which set in either in a mild or in a severe form, but in which on the subsidence of the fever the strength does not return, convalescence is retarded or arrested altogether, and the patient falls into a general atrophic condition and usually dies in from three to six months, of marasmus.

By deciding to which of the above groups the case belongs, the prognosis will be to a great extent determined. The chief indication of danger is the early appearance of purpuric and hæmorrhagic conditions ; the supervention of hydrocephalus also makes the prognosis very grave.

The mortality in cerebro-spinal fever is very high, probably about 60 per cent. There are not at present sufficient data to determine whether there is any marked difference in this respect between the sporadic and the epidemic cases. The mortality has varied considerably in different epidemics : in some of those which occurred in America, it is placed as high as 75 per cent. ; among the Irish Constabulary it reached 80 per cent. The mortality is highest at the beginning of epidemics.

DIAGNOSIS.—Cerebro-spinal fever is liable to be confounded with typhus fever on account of the petechial rash, but is distinguished from it by the eruption appearing suddenly without any previous mottling of the skin. The nervous symptoms also distinguish it from typhus, although in a case of

typhus complicated with meningitis the diagnosis may be impossible. It is distinguished from *purpura hæmorrhagica* by the intensity of the fever, and the localised nervous symptoms. The malignant cases are more likely to be mistaken for malignant scarlatina than for any other disease, and must be distinguished therefrom by the rash, sore-throat, and nervous affections ; yet in some cases the two diseases have been indistinguishable from each other, especially where death occurred within twenty-four hours, and both were epidemic at the time. In other cases malignant small-pox has been mistaken for this disease.

Lobar pneumonia with cerebral symptoms, and influenza with headache and pain in the back may simulate cerebro-spinal fever, but in the former the presence of the characteristic signs in the lungs or the marked rapidity of respiration may make the diagnosis clear, while the latter lacks both the rigidity and retraction of the head and also the rashes which characterise the meningeal affection.

A sign which is sometimes of value in the diagnosis of spinal meningitis, and which has been described especially in connection with the diagnosis of cerebro-spinal fever, is the reflex contraction of the flexor muscles at the back of the thigh which occurs when an attempt is made to extend the leg on the thigh, with the thigh at the same time flexed as in the sitting posture. See p. 820.

From other forms of meningitis cerebro-spinal fever is distinguished chiefly by its sudden onset and by the rashes which frequently accompany it. Unlike suppurative meningitis, cerebro-spinal fever shows no obvious focus of infection such as a primary lobar pneumonia, or an empyema, or disease of the ear, and it differs from tubercular meningitis not only in its acute onset and the presence of a rash, but also in the marked opisthotonos, which is usually lacking in the tubercular disease.

The use of lumbar puncture for diagnosis has been advocated recently, and may be advisable in some cases. The naked-eye appearances alone of the fluid withdrawn may be sufficient to determine the presence of meningitis, the nature of which is determined by bacteriological examination of the fluid. See LUMBAR PUNCTURE.

MORBID ANATOMY.—The skull and dura mater are often abnormally vascular ; the cerebral sinuses are much distended with blood. All the membranes of the brain are more or less congested ; the arachnoid especially is vascular and opaque from inflammatory exudation—this opacity varies from slight milkiness to thick and dense deposits.

The characteristic lesion is the whitish- or greenish-yellow fibrino-purulent deposit found at the base of the brain ; in many cases, however, it is not limited to the base but extends up along the sulci to the vertex.

This deposit varies somewhat with the duration of the disease : in cases which have lasted a week or two, it is abundant, yellowish or greenish ; and the origins of the nerves seem to be buried in and compressed by the exudation. The brain-substance itself is more vascular than normal, and there is some inflammatory infiltration of its superficial layers. The ventricles in the more acute cases contain turbid fluid, in the more prolonged they may be much distended by excess of clear cerebro-spinal fluid.

In the spinal cord the lesions are similar to those found in the brain and its membranes.

Purulent infiltration of the eyeball, and effusion into the joints are met with in some cases. The lungs sometimes show extreme congestion or actual broncho-pneumonia; the liver and spleen have been found congested and softened in many cases.

TREATMENT.—The treatment in the early stage must be directed to recovering the patient from the collapse. This is best done by the application of heat, the administration of small quantities of stimulants or stimulating enemata, and the application of sinapisms over the chest and back. In the next stage of the disease attention must be almost altogether directed to allaying the spinal irritation and promoting absorption of the exudation. The extreme irritation will be best diminished by the use of belladonna and bromide of potassium. Ergot has also been found useful for this purpose.

The pain, which is extreme, will yield best to frequent and considerable doses of opium; indeed many physicians have relied altogether on opium as the curative agent. Chloral hydrate has proved beneficial in conjunction with bromides, in allaying restlessness and procuring sleep. For promoting absorption of the exudation, mercury and iodide of potassium have been chiefly relied upon. In the more sthenic cases calomel may be employed with benefit in small and repeated doses. The disease being usually of an asthenic type mercury will seldom be well borne, and iodide of potassium should be preferred.

Leeches applied to the back of the neck, behind the ears, or to the temples produce great relief of the excessive pain in the head and upper part of the spine. The application of ice to the head and spine temporarily allays pain, but there is little evidence of permanent benefit being derived therefrom. In prolonged cases blisters applied along the spine have seemed to do good.

Recently the withdrawal of cerebro-spinal fluid by lumbar puncture has been used as a therapeutic measure; but it seems more than doubtful whether any permanent good is derived from it: probably the most that can be said in its favour is that some temporary improvement, lasting a few hours, has occasionally followed its use. Drainage with laminectomy has also been tried, and recovery after the operation has been recorded. This method of treatment may be 'justifiable in certain severe cases in which the spinal symptoms are very marked' (Osler). Local complications must be treated as they arise. Stimulants are required in considerable quantity in a large number of the cases which present adynamic symptoms.

T. W. GRIMSHAW.

G. F. STILL.

MENINGOCELE.—See SKULL, Diseases and Deformities of.

MENINGO-ENCEPHALITIS.—A name given to a pathological condition in which inflammation of the pia mater implicates the subjacent cortical substance. This condition probably always exists to a certain extent in meningitis, and reveals itself on careful microscopic examination—although the inflammatory changes may not have advanced far enough to produce an easily appreciable amount of softening.

MENINGO-MYELITIS is a term used to indicate a condition in which inflammation of the spinal meninges implicates the surface of the spinal cord. See MENINGO-ENCEPHALITIS.

MENOPAUSE (μηνῆς, the menses; and παῦσις, a cessation).—The natural cessation of the menstrual flow, or 'change of life,' in the female. See CHANGE OF LIFE.

MENORRHAGIA (μηνῆς, the menses; and ῥήγνυμι, I burst forth).—Over-abundant menstruation, whether due to excessive quantity or to undue frequency. See MENSTRUATION, Disorders of.

MENSTRUATION, Disorders of.—SYNON.: Fr. *Troubles de la Menstruation*; Ger. *Störungen des Monatsflusses*; *Störungen der Menstruation*.

For the due performance of menstruation two conditions are essential, namely, sound general health, and normally developed organs of generation. Disorders of the menstrual process may be brought about by very many conditions. These disorders are generally divided into: I. *Amenorrhœa*, where the discharge is absent, or deficient in quantity. II. *Dysmenorrhœa*, where the function is performed with difficulty and pain. III. *Menorrhagia*, where the discharge is profuse.

I. **AMENORRHŒA.**—SYNON.: F. *Aménorrhée*; Ger. *Amenorrhœ*.

ÆTIOLOGY AND SYMPTOMS.—Amenorrhœa is dependent either on *general* states; or on *local* pathological conditions—that is, on lesions of the uterus and ovaries.

(1) All conditions or influences which tend to deteriorate the blood, or which act unfavourably on nutrition, may be causes of amenorrhœa. The most common of these is the demand made on the system in the development of the aptitude for conception, the growth and separation of ova, and the performance of the menstrual function. At this time the breasts develop, the ovaries and uterus enlarge, the pelvis grows, and the whole form becomes altered. Many women who during childhood have enjoyed apparently perfect health, as they approach puberty become gradually or suddenly anæmic or chlorotic, without any assignable cause other than the demand made on nutrition by the process of development through which they at the time pass. Nutrition becomes impaired, tastes perverted, pains of a neuralgic character are felt in various parts of the body, the menstrual discharge does not appear, or it may appear once scantily and then at irregular intervals, or it may disappear for months or even years. All the symptoms of anæmia are present, and the patient is languid and listless, lacks energy, and is in more or less constant suffering. The above course of events may take place even in cases where the surroundings are favourable to healthy development. Hygienic conditions, however, play a most important part in the proper development of the female functions, and when the surroundings are unfavourable, evil is sure to follow. Want of food or improper food, want of fresh air or impure air, want of exercise, foul gases, malaria, are prolific causes of failure or imperfection in the growth and development of the young girl, and are common causes of amenorrhœa. Disease also is by no means an infrequent cause of the condition under consideration, such as phthisis, Bright's disease and diseases of the liver, stomach, and nervous system. Emotion, fright, or grief, change of air and food (as when girls go from the country to London), and cold, may arrest or suspend the monthly discharge.

(2) Amenorrhœa may be due to local conditions.

These are absence or disease of the ovaries, of the uterus, or of both; and imperfect development of one or both organs.

In cases where the ovaries are absent, the change in form, from girl to woman, which normally takes place at puberty, does not occur. The girl grows but does not develop. A masculine appearance supervenes, the breasts remain small, the pelvis narrow, the voice becomes manly and harsh, a beard may grow on the face, sexual passion is absent, and the health remains good.

When the uterus alone is wanting, there may be no indication of the condition in the state of the general health or development, and local examination is necessary in order to detect the circumstance. In these cases the vagina terminates in a *cul de sac*, and the uterus cannot be felt on examination. On introducing a finger into the rectum and a sound into the bladder, it is found that the two organs are in contact, and that there is no uterus between them. There are, however, as a rule, one or two small fibrous masses representing the uterus.

Certain diseases, such as tuberculosis and atrophy, involving the whole substance of the ovaries, and also atrophy of the womb, may cause amenorrhœa.

Amenorrhœa from Retention.—In these cases the sanguineous discharge is separated, but does not appear externally, owing to atresia of the genital canal. The closure may occur at any point between the os uteri and the vaginal orifice. A membrane may close the os tincæ; the hymen may be imperforate; the vagina may be absent, or its walls may be adherent at any part of its course, or along the whole of it. The occlusion may be congenital, or may arise from inflammation during childhood or after severe labour. In these cases the menstrual molimina are periodically present, but the catamenia do not appear. The molimina increase in severity from month to month; the patient has pain in the back and a sense of weight in the pelvis, and becomes pale and sallow; the abdomen after a time begins to enlarge, and continues to increase at monthly intervals. On examination a tumour having the shape of the enlarged uterus may be felt rising from the pelvis. It is smooth, elastic, and dull on percussion. If the condition be not discovered, the distension of the uterus may go on to rupture, or its contents may pass along the Fallopian tubes into the abdomen, causing peritonitis and death.

DIAGNOSIS.—Whenever a patient suffers from amenorrhœa, pregnancy should be thought of. If this state can be excluded, the general condition should be investigated. Anæmia and its causes should be sought for. The chest, heart, and urine should be examined. If there be no general condition to account for the amenorrhœa, the practitioner should see whether the breasts and pelvis are developed, and examine the vulva and vagina for obstruction, if there be any suspicion of such a condition. Finally, it may be necessary to examine the uterus and ovaries.

TREATMENT.—The treatment of the first form of amenorrhœa is the treatment of the general state. If there be want of constitutional vigour, change of air, exercise in the open air, mental occupation, but not severe, and nourishing diet should be advised. The stomach and bowels should be attended to; and gentle aperients and salines given if the tongue be foul; then vegetable tonics, iron, iodides, or

other appropriate remedies. No efforts should be made to act especially upon the uterus, and this is particularly binding when the amenorrhœa is dependent on phthisis, Bright's disease, or similar conditions.

The second form is often incurable. In those cases in which the uterus and ovaries are absent nothing can be done. If the uterus be present, but imperfectly developed, much may be done when the cases are seen early, but nothing if seen late. Attempts have been made to promote the growth and development of the uterus by local treatment, but without success. For this purpose stem-pessaries, galvanic pessaries, and irritants have been used, but with only injurious results. In these cases, whether they be seen early or late, local treatment is of no use, while general treatment, directed to improve the health and favour growth, may prove of the greatest value. Plenty of good plain food, walking-exercise short of fatigue, and freedom from school and hard mental work, are the means which favour the easy and early transition from girlhood to womanhood; while over-work, sedentary habits, deficient or bad food, and bad air produce a rich harvest of physical suffering peculiar to women.

In cases of retention of the menses from atresia of the genital canal, an outlet must be made for the flow. If the hymen be imperforate it should be divided, and the fluid allowed to run out. In cases of absence of the vagina, a canal has in some instances been successfully made. This, however, should only be done in cases in which the uterus and the ovaries are developed. In atresia of the os uteri the offending structure should be divided by the trocar or knife. These operations are accompanied by a considerable amount of danger. Patients not infrequently die after them from peritonitis or shock. It should not be forgotten, however, that it is imperative to remove the menstrual fluid retained, for unless this be accomplished death is inevitable.

II. Dysmenorrhœa.—**SYNON.** : Fr. *Dysménorrhée*; Ger. *Dysmenorrhœ*.

In dysmenorrhœa, menstruation is accompanied by pain—that is, pain referable to the genital organs. This pain is seated in the pelvis, and radiates to the sacrum, groins, and thighs. In some women the menstrual function is performed without pain or discomfort of any kind. As a rule, however, they suffer more or less from dull aching in the pelvis, backache, headache, languor, and lassitude during the catamenial flow. When the dull aching amounts to sharp pain, the function is performed abnormally, and the woman is said to suffer from dysmenorrhœa. This symptom is frequently met with, and in association with many pathological conditions of the pelvis; diseases of the uterus, such as fibroids; of the tubes, such as congestion and inflammation; of the ovaries; and also with general diseases, such as anæmia, gout, and rheumatism.

ÆTIOLOGY AND SYMPTOMS.—Dysmenorrhœa has been referred to five different conditions, upon one or more of which it is supposed to depend. Hence five kinds of dysmenorrhœa have been described: (1) *Mechanical or obstructive*; (2) *Conjunctive or inflammatory*; (3) *Ovarian*; (4) *Membranous*; and (5) *Spasmodic or neuralgic*.

I. Mechanical or obstructive dysmenorrhœa.—This form of dysmenorrhœa was long thought to be the most common; indeed, it has been asserted

that dysmenorrhœa cannot be said to exist without obstruction to the flow of blood from the uterus. Opinions, however, differ greatly with regard to the seat of the obstruction. Robert Barnes believed it to be seated usually at the os tincæ, and to be frequently accompanied by conical cervix; Marion Sims thought its most frequent seat was the os internum uteri; while Graily Hewitt referred the obstruction to flexion of the uterus in the great majority of cases—the obstruction being caused by the narrowing of the uterine canal at the point of flexion. The outer orifice of the uterus may be closed from birth, or in consequence of inflammation occurring subsequently, and thus cause obstruction to the escape of the catamenial fluid. There is no question therefore that this condition is one that may give rise in some way to painful menstruation. Such a narrowing of the os externum as to give rise to dysmenorrhœa is, however, extremely rare. A narrow, or so-called pin-hole, os generally admits the uterine sound, and women, subjects of this condition, frequently menstruate without pain. Indeed, it has been abundantly proved that pin-hole os plays no part in the production of dysmenorrhœa. The same may be said of contraction of the inner orifice. This part is rarely or never met with so small as to prevent the passage of the sound, and just as rarely presents obstruction to the escape of the fluid. Wherever the sound can be passed it may be safely inferred that there can be no obstructive dysmenorrhœa. That flexions of the uterus can interfere with the calibre of the canal and obstruct the menstrual flow is a simple theory, and all observations in the dead body go to show that it is fallacious, for the few flexed uteri which are found in our museums present patent canals. The only instances in which the canal is constricted by flexion are those in which senile atrophy has taken place—that is, when the walls of the organ have become thinned by age; but by this time the menopause has been passed, and dysmenorrhœa become impossible. So-called retroflexion of the uterus may, however, give rise to dysmenorrhœa when the body of the organ sinks into the pouch of Douglas, and it, together with the broad ligaments, becomes constricted by the sacro-uterine ligaments. Just as hernia of the uterus may give rise to congestion and dysmenorrhœa, so such a condition would prevent the return of the blood along the uterine veins, and lead to swelling and congestion of the body of the uterus, and painful menstruation.

2. *Congestive or inflammatory dysmenorrhœa.*—This name has been given to those cases of painful menstruation in which the uterus is enlarged and heavier than natural. It is met with in the married and in the single, but it is probable that it never occurs as a primary affection. Congestion and inflammation are frequent accompaniments of dysmenorrhœa when they are not the cause of it. They are generally the result of labour or abortion, or of dysmenorrhœa itself, of infection or mischievous meddling, more especially the use of instruments, such as the uterine sound. The symptoms are a continuous dull aching pain, chiefly in the sacrum and thighs, and a sense of weight and fulness in the pelvis. This form does not deserve the name of dysmenorrhœa, for the pain is at its worst at other times than during menstruation, and is frequently relieved by the appearance of the menstrual flow.

3. *Ovarian dysmenorrhœa.*—This does not deserve the name of dysmenorrhœa, for it is not due to menstruation—that is, to the discharge of the sanguineous fluid from the uterus, but to the growth and rupture of the Graafian follicles. The Graafian follicles develop gradually, and take a long time to arrive at maturity. It is not a sudden process. Towards the end of their growth, when they are about to rupture, the ovarian pain is experienced. They usually burst some time before the appearance of the menstrual flow, but the rupture may happen during the flow or after its cessation. Pain usually comes on before the flow—a few days or a week—and may cease with the appearance of the menses, or several days before that event; the suffering may, however, come on at any time during the flow or immediately after it has ceased, or at any time during the interval. It is situated usually in the left ovarian region, for the left ovary is more frequently affected than the right, the pain extending down to the thighs and to the sacro-iliac joint of the same side. Not infrequently the corresponding kidney is tender. Pain may occur in the right or left side at alternate periods—or a period may pass without pain. Vomiting and hysterical manifestations are often present. There is superficial and deep tenderness over the painful part. Patients often say that they have a swelling in the side, and, on examination, a diffused fulness is found in the ovarian region, which is tympanitic, and evidently due to local distension of intestine with gas. Examination *per vaginam* and *per rectum* will often detect a small body or swelling, tender and movable at first—later on fixed—on the affected side and a little behind the uterus. Pressure on the swelling calls forth severe pain and a feeling of sickness. At a later period the uterus becomes less movable and drawn to the affected side. This is, doubtless, due to contraction of inflammatory products, and not to distension of the broad ligaments, for it occurs in long-standing cases only. Micturition is frequent and painful. The pathological lesion is inflammation of the uterine appendages—the tubes, the ovaries, and the peritoneum, the Graafian follicles, the stroma, or the surface of the ovaries, one or all may be affected. This condition is again rarely primary. In women who have had children it is often due to parturition and abortion. In the unmarried, it is often the result of long-standing dysmenorrhœa, sometimes of acute specific disease, or of inflammatory mischief set up by causes mentioned in the preceding paragraph, or exposure to cold during menstruation. Gonorrhœa is also a cause.

4. *Membranous dysmenorrhœa.*—In this form a membranous sac, having the shape of the body of the uterus, is expelled with the menses. The sac has three orifices corresponding to the orifices of the Fallopian tubes and the inner orifice of the uterus. It has an internal smooth, punctated, and an external flocculent surface. Occasionally during expulsion the sac is turned inside out. It may be passed with every or every other menstruation, or only occasionally. Instead of being passed in the form of a complete sac, the membrane may be broken up and expelled as shreds of various sizes. Microscopical examination shows that the membrane possesses a structure identical with the lining of the body of the uterus. It contains glands and blood-vessels, and is, in fact, the decidua menstrualis. It has been said that this membrane is always the result of

conception, but ample evidence has been published in refutation of this statement. Other bodies may be expelled from the uterus during menstruation, such as clots of blood, fibrin, masses of mucus forming casts of the uterine canal, casts of the vagina, and products of conception. Several cases of monthly abortion have been recorded. These substances can be distinguished from the decidua menstrualis by microscopical examination only, and easily, except the products of conception. These present the structure of the decidua; but they also present some additional appearances which, if found, are characteristic, such as the sac—partial or complete—formed by the decidua reflexa and the large cells which are met with in the decidua vera and chorionic villi. When cases of this disorder come under notice the uterus is, as a rule, enlarged. This, however, is not always the case. The enlargement is probably a condition secondary to the dysmenorrhœa or to previous gestation. There is commonly tenderness of the pelvic tissues around the uterus, probably of the peritoneum. Ovaritis is frequently present. These conditions are probably secondary. Affections of other mucous membranes may be present, such as bronchial catarrh. Membranes may be passed from the uterus without pain; or pain may be present, but varying in degree from slight discomfort to intense suffering. The severest pains, however, are not due to obstruction caused by the passage of the membranes blocking the os uteri and causing retention of the fluid, but to spasm of the uterus. The passage of the membrane takes place often on the third day of menstruation, but it may occur later. Frequently shreds are passed from the first or second day to the end of the flow. With the expulsion of the membrane there is usually a gush of blood, after which the flow proceeds normally. The catamenial discharge may be normal in amount, considerably increased, or even scanty.

The pathology of this affection is somewhat obscure. Several views have been held with regard to it, some of which deserve no notice. Inflammation, however, is so frequent an accompaniment, that the view that inflammation is its cause is one which deserves attention. Against this view is the fact that about two-thirds or three-fourths of the cases are cases of primary dysmenorrhœa, in which inflammation could not have been present; and it is consequently inferred that the inflammation, which is so often present, is really a secondary development, being the result of the dysmenorrhœa; moreover, on no other surface of the body is inflammation known to give rise to a periodical exudation of this kind, or to change the character of the mucous surface in such a manner as to cause it to be shed in the way the mucous membrane of the uterus is shed in this disease. The cause is probably to be sought in malnutrition of the uterus, which in some cases has existed *ab initio*, while in others it has succeeded to some disease, such as inflammation or imperfect involution of the uterus after pregnancy. It has been met with also in gouty and rheumatic subjects, but it is not known if it holds any relation to these diseases. The pathology of the affection appears to be a failure of the molecular disintegration of the decidua menstrualis which takes place during normal menstruation, and this must be due to the presence in the decidua of some tissue which resists disintegration more than the healthy tissues of that membrane. The only tissue which has been found in the uterine wall which would offer

such resistance to disintegration is fibrous tissue. It is known that this varies somewhat in quantity in the wall of the uterus, according as it is diseased or healthy. In cases of inflammation of the uterus in which imperfect resolution has taken place, excess of fibrous tissue is found in the wall. In cases of sub-involution a similar excess is found, and it would be expected that in cases of imperfect development at puberty a similar excess would be found. It is known that the organs in which membranous dysmenorrhœa is met with are imperfectly developed uteri, uteri that have been inflamed, and uteri that have been pregnant and in which parturition or abortion has been followed by an imperfect return of the organ to its natural state; and it is maintained that the presence of an excess of fibrous tissue in the wall of such uteri is the cause of the shedding of the decidua as a membrane. This is the most probable explanation of the occurrence of membranous dysmenorrhœa.

5. *Spasmodic or neuralgic dysmenorrhœa*.—This form of dysmenorrhœa includes the very great majority of severe cases. For a time spasm or neuralgia were regarded as an asylum for ignorance, and other conditions which formed the basis of the mechanical theory of uterine pathology were put forward in their place. Further research, however, has shown that spasm and neuralgia are by no means conditions of no importance in dysmenorrhœa, that the foundations of the mechanical system were laid in error, and that dysmenorrhœa is but another name for uterine colic. It is well known that with dysmenorrhœa, flexions of the uterus, a narrow external orifice, or a narrow internal orifice may be present, and it is equally well established that these conditions may be removed and yet the dysmenorrhœa remain. The inner and outer orifices may be incised, and the flexion straightened, without relief. The observations of Vedeler and Herman prove that the percentage of virgins who suffer from dysmenorrhœa is almost the same whether the uterus be straight or in a state of flexion. In fact, dysmenorrhœa is just as frequent in the absence of flexion of the uterus as when flexion is present; and every practitioner who has seen a considerable number of cases of dysmenorrhœa has ample proof that a narrow external or internal os is not a common cause of it.

Dysmenorrhœa may be divided into two categories—the primary and the acquired. Primary dysmenorrhœa is present from the commencement of menstruation, or soon afterwards. Acquired dysmenorrhœa comes on at a later period of life, after menstruation has been thoroughly established, often after pregnancy, labour, abortion, and in consequence of chills, inflammatory attacks of the pelvic organs, acute diseases, anæmia, and exhaustion. The primary variety forms the majority of cases, and in these no recognisable disease of the uterus is present. It is generally believed that the uterus undergoes regular contractions during menstruation. These cannot be observed in the healthy organ, but they have been witnessed in cases in which the uterus was enlarged by fibroid tumours. In health these contractions are painless. When they become irregular they are painful, and give rise to dysmenorrhœa. The pain is situated in the pelvis, and is referred to the uterus. It is of varying intensity: it may be slight; or it may be extremely severe—agonising. It radiates to the groins, sacrum, and thighs. It is often said to be all round the pelvis or lower part of the trunk, and is often com-

pared with the pain of labour, or that of abortion. The pain may come on a little before, with, or a little after the appearance of the discharge. Usually it occurs during the first twelve or twenty-four hours, and lasts from four or five hours to twenty-four, or even to the end of the flow. It is paroxysmal. There is often tenderness of the skin of the hypogastrium and groins, vomiting, hiccup, headache, hysteria; and even delirium may be present. The menstrual flow may be scanty or profuse. In the former case it is often followed by an abundant yellow discharge lasting for a few days. There may be leucorrhœa throughout the inter-menstrual interval. Micturition is sometimes frequent and painful. Patients who suffer in this way may enjoy good health during the inter-menstrual interval, but often they suffer from neuralgic pains at other times than at the periods, and they frequently suffer from severe headaches at the time of their period.

TREATMENT.—Dysmenorrhœa is generally obstinate under treatment, and its course is very protracted. In many cases much may be done by attention to the general health, to the state of the stomach, liver, and bowels. During an attack, if severe, rest in bed should be enjoined, and hot baths. As drugs, solution of acetate of ammonium, antipyrine, and phenacetin are useful; sometimes opium and morphine are called for, for the relief of the pain. Alcohol in small doses is useless, and in large quantities intoxicates. During the interval saline aperients, iron, arsenic, bismuth, iodide of potassium, and ergot are of service; guaiacum resin alone or in combination with sulphur is sometimes of use. If there be a gouty or rheumatic tendency, this should be treated. As a rule, in these cases there is nothing to be gained by local treatment of so-called misplacement and conical cervix. Occasionally, however, in cases of severe retroversion or retroflexion, where the fundus of the uterus is grasped in Douglas's pouch, a pessary may be of use. Incision of the external orifice, in so far as the writer knows, is of no use. Relief sometimes follows incision of the internal orifice. Whether this relief is due to the enlargement of the canal caused by the incision, or to the stretching of the neck of the womb, which is generally carried out along with it, either by the introduction of a plug of lint or of a stem-pessary, is still a debated question. The idea of incising the neck of the womb, whether at the external or internal orifice, is based upon an error in pathology, namely, that the pain is due to mechanical obstruction to the flow of the menstrual discharge from the uterus; and, as it is a dangerous, and may be even a fatal proceeding, it should not be undertaken. Dilatation of the cervix, however, is the most efficient means we have for the relief of severe dysmenorrhœa when drugs and other treatment fail. The object of this procedure is not to enlarge the canal for the passage of the menstrual fluid, but to stretch the neck of the womb in order to destroy the tendency to spasm. Indeed, it is done on precisely the same principle as dilatation of the vaginal orifice is done for vaginismus, and of the sphincter ani in cases of spasm of that muscle. The dilatation may be effected by the use of tents made of *Laminaria digitata*, tupelo wood, or sponge; the first two are preferable to the last. They should be allowed to remain in the cervix for from six to ten hours, until the canal has been well dilated. Or, preferably, the dilatation may be carried out by means of bougies;

metallic bougies are the best, graduated according to English measurement. The dilatation may be carried out at one sitting, when the patient is anæsthetised, and a series of bougies from No. 6 or 8 to No. 16 to 18 passed; or at several sittings, when one or two bougies should be passed every second or third day, until the required size has been reached. Hegar's bougies are often used for this purpose, but the English metallic bougies are in every respect preferable. Dilators have been used for this purpose also, such as Priestley's or Ellinger's. Some of these have three blades and some have two. Some are opened by a screw and some by hand pressure. There are many instances in which these instruments have lacerated the cervix severely, and, their use being accompanied by greater risk than that of bougies, they should be avoided. Dilatation cures some cases and relieves others, but in a large number it proves of no use whatever.

III. Menorrhagia and Metrorrhagia.—**SYNON.**: Vulg. Flooding; Fr. *Ménorrhagie*; *Mérorrhagie*; Ger. *Mutterblutfluss*.

Menorrhagia is used to denote profuse menstruation; metrorrhagia, hæmorrhage from the uterus at any other time than the catamenial epoch. The two symptoms are frequently met with. Menorrhagia often exists alone. When metrorrhagia is present during menstrual life, the catamenia are, as a rule, also profuse. These hæmorrhages may be called forth by many lesions. Indeed, they may accompany the majority of the pathological conditions to which the pelvic organs are liable. They may also arise from general states—as scurvy, the hæmorrhagic diathesis, Bright's disease, phthisis, cirrhosis of the liver, and the acute specific diseases. The most common cases are, however, associated with distinct alterations of structure in the pelvic organs, as sub-involution of the uterus, polypus, fibroid tumour, cancer, displacements, retained portions of placenta, moles (fleshy or vesicular), fungous degeneration of the mucous membrane of the uterus, mucous polypi, ulcerations of the cervix, hæmatocele, inversion of the uterus, and congestion of this organ due to obstruction to the circulation through the heart and lungs or liver.

Profuse hæmorrhages of an irregular character occur also in young girls before the advent of regular menstruation. This form of uterine hæmorrhage is not common, but it is sometimes of very serious import, and occasionally has proved fatal. More frequent is the occurrence of irregular bleeding from the uterus at the menopause. The causes of these climacteric hæmorrhages are really not known. They have been said to be due to congestion, but on insufficient evidence.

TREATMENT.—The treatment of hæmorrhage from the uterus resolves itself into the immediate treatment of the attack, and the treatment of the condition leading to it. The treatment of the attack, or the means of arresting the bleeding, consists in great part in securing absolute rest. The patient should remain in bed in the recumbent position, and avoid all exertion—mental and physical. At the same time, internal remedies which tend to check hæmorrhage should be given. Of these, those most commonly used are ergot, hamamelis, hydrastis, gallic acid, the mineral acids, and acetate of lead. Mineral acids, in combination with sulphate of magnesium or sodium, often act well. Should acetate of lead be administered, the patient should be carefully watched, as some persons are

very sensitive to the action of the drug, and manifest symptoms of acute lead-poisoning after the administration of a small quantity. Should these means fail, recourse should be had to plugging the vagina or uterus. The vagina is plugged in the following manner: The patient is placed on her left side, a speculum is introduced, and the vaginal canal is firmly packed with pledgets of iodoform- or salalembroth wool or gauze, tied on a string for convenience of removal. This will arrest the hæmorrhage for a time, but it can only prove a temporary expedient. The plug is liable to become offensive, from decomposition of blood and of the secretions in the vagina, and should consequently be changed in forty-eight to sixty hours, or sooner. A more efficient means of arresting hæmorrhage is plugging the uterus itself. This is done by means of tents of sponge or laminaria, and with a twofold object. The first object is the immediate arrest of the bleeding; but the chief object usually is to dilate the canal of the uterus, so as to permit its exploration by the finger, and the discovery of the cause of the bleeding. This means will not only check the bleeding temporarily, but will in many cases effect a permanent cure. Tents should be rendered aseptic by immersion for some days in a solution of corrosive sublimate in absolute alcohol (1 in 1,000). To facilitate the introduction of a tent, a Sims's speculum should be used, and the cervix of the uterus should be fixed by a sharp hook. In many cases, however, tents will not be necessary. The hæmorrhage will be controlled by the other means enumerated, or the cause of the hæmorrhage will be made out without the use of tents. In all cases, however, in which the hæmorrhage is uncontrollable, or so profuse as to threaten life, or in which the cause of the bleeding is obscure, tents should be had recourse to, both to check the flow and complete the diagnosis. When the cause has been discovered, it should, if possible, be removed. Rapid dilatation of the unimpregnated uterus by means of bougies, so as to allow exploration of the cavity with the finger, is dangerous owing to the laceration of the cervix caused by them. When, however, a tent has been previously used dilatation by bougies is easy and with care safe.

But even after the uterine canal has been dilated, no definite cause may be found for the bleeding. In these cases, styptics, or even caustics, may be applied to the inner surface of the organ. Those chiefly used are a solution of iodine, a solution of perchloride of iron, chromic acid, carbolic acid, and fuming nitric acid. These are best applied through a uterine speculum of platinum or vulcanite, on a probe of similar material. While using these means, it should be borne in mind that internal uterine medication is not free from grave danger.

The rest of the treatment of menorrhagia consists in attention to the general health.

JOHN WILLIAMS.

MENSURATION (*mensura*, a measure).—A synonym for measurement. See PHYSICAL EXAMINATION.

MENTAL DISORDERS.—See INSANITY.

MENTONE, France, on the extreme East of the French Riviera.—A moderately warm, bracing, sheltered, and dry winter climate. Mean temperature in winter 52° F.; rainfall 24 inches. See CLIMATE, Treatment of Disease by.

MERCURY, Diseases arising from.—
SYNON.: Fr. *Hydragryrie*; *Intoxication mercurielle*; Ger. *Quecksilbervergiftung*.

Though considerable discrepancies of opinion have existed as to the poisonous or innocent properties of the metal mercury itself when swallowed, there can be no doubt as to the poisonous character of its soluble and volatile compounds, nor even as to the insidious nature of the vapours of metallic mercury. Metallic mercury has occasionally been administered in enormous quantities without producing any decided physiological effects; while, in other instances, salivation and other specific effects have resulted. These differences are doubtless due to the fact that, in those cases where effects have resulted from the administration, oxidation and solution of a portion of the metal had taken place. See OCCUPATION-DISEASES.

Mercurial poisoning may be either (A) *acute*, or (B) *chronic*; the former resulting from the administration of one or several large doses at short intervals, the latter form of mercurialism arising from the repeated exhibition of small doses of the less active preparations of the metal. There is also a peculiar form of mercurialism which is the effect of the inhalation of the vapours either of the metal or of its volatile compounds, and is characterised by paralysis.

A. Acute mercurial poisoning.—**DESCRIPTION.**—The effects produced by a considerable dose, say 60 grains, of one of the more soluble compounds of mercury, such as corrosive sublimate or the nitrate, are those of a corrosive and irritant poison. The effects are immediate. In the act of swallowing, an intense burning sensation is experienced in the mouth and throat, followed by excruciating pain in the stomach, and extending over the abdomen. The local effects of the poison are frequently visible, as a whitening of the tongue and fauces. There is vomiting, tenesmus, and purging, often of a bloody character. Colic, and great tenderness and swelling of the abdomen, are also symptomatic. Not infrequently there is suppression of the urine. The gustatory sensation is perverted; there is dryness of the mouth; and a brassy or metallic taste is generally experienced after the first local corrosive action of the poison has somewhat abated. The countenance is anxious; the skin is pale, cold, and clammy; and the pulse is small, weak, and rapid. The urine is blood-stained, scanty, or suppressed. Salivation may supervene, accompanied by fetor of the breath. Should recovery not take place, death may occur within a few hours, or may be delayed for one or more days; or the patient may more rarely succumb to some of the ordinary sequelæ of corrosive poisoning. When death supervenes speedily after the administration of the poison, the fatal result is usually due to collapse.

Most of the effects of acute mercurial poisoning may result from the application of a concentrated solution of corrosive sublimate or mercuric nitrate to the unbroken skin.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances seen after acute mercurial poisoning are inflammation and even erosion of the mucous membrane of the stomach, and extravasation of blood beneath this membrane. Ulceration is rare. The intestinal tract also exhibits signs of extensive inflammation, and this has been noticed especially in the large intestine. The rectum is usually much inflamed, and its surface covered with shreds of

bloody mucus. A peculiar slaty appearance of the mucous membrane of the stomach and intestines, where not highly inflamed, has been thought to be characteristic of poisoning by corrosive sublimate.

DIAGNOSIS.—Though the symptoms of poisoning by corrosive sublimate, and other corrosive preparations of mercury, greatly resemble those produced by arsenic, the diagnosis is generally not difficult. The effects following almost immediately on administration, the metallic taste in the mouth, and the greater frequency of bloody stools in mercurial poisoning, serve to differentiate between the poisons. Where doubt exists, an analysis of the secretions may be made; arsenic is most readily detected in the urine, and mercury in the saliva. The existence of salivation and fœtor of the breath—though not always present—may also be valuable aids in completing the diagnosis.

TREATMENT.—In acute poisoning by corrosive sublimate, the best antidote is albumen, or the albuminoids in any soluble form. The white of one or more eggs should be beaten up with water, and swallowed as quickly as possible. Failing an egg, flour made into a thin paste may be administered. Albumen combines directly with corrosive sublimate to form an insoluble compound. On account of the powerful local action of the poison on the stomach, the use of the stomach-pump is not advisable; but if the vomiting be not free, emetics of as simple a character as possible may be administered. The rest of the treatment consists in alleviating pain by means of opiates, and the general treatment applicable for irritant poisons. Thirst must be alleviated by demulcent drinks. For this purpose milk, mixed with once or twice its bulk of lime-water, is excellent; the casein of the milk and the lime both tending to render the mercury insoluble, and so to act as antidotes.

B. Chronic mercurial poisoning.—**SYNON.:** Mercurialism.

DESCRIPTION.—The repeated ingestion of small doses of the more soluble and active preparations of mercury, such as the perchloride and the cyanide, may give rise to chronic symptoms; but these more frequently result from the administration of one or more doses of the more insoluble preparations of the metal, such as calomel or the oxides. When chronic symptoms follow the administration of one dose of a mercurial preparation, this is not altogether due to the peculiar idiosyncrasy of the patient, but is attributable in no small degree to the slowness with which mercury is eliminated from the system. There appears also to be a remarkable difference, not altogether dependent upon their differing solubilities, between *mercuric* or per-salts and *mercurous* or proto-salts, in respect to their toxic properties. Mercuric compounds are greatly more potent than mercurous salts. By far the most common result of the continued administration of mercury compounds is salivation. This consists in a profuse discharge from the salivary glands; swelling and tenderness of the gums; and fœtor of the breath. In children, and more rarely in adults, salivation may pass into sloughing and gangrene of the cheeks; and a fatal result may ensue. Other symptoms are nausea, colicky pains, depression, and those nervous symptoms to which the term 'mercurial palsy' has been applied; but this last group of symptoms, which is most commonly met with after inhalation of the vapours of mercury, must be described more in detail.

Mercurial Paralysis.—Workers in mercury, such as water-gilders, looking-glass makers, and the makers of barometers and thermometers, are apt to suffer from a peculiar form of shaking palsy, known either as 'the trembles,' mercurial tremors or metallic tremors, and, by the French, as *tremblement métallique*. This disease affects those who handle the oxides of the metal, but more frequently those who are exposed to mercurial fumes. Mercury exhibits a small vapour-tension, and consequently is vaporisable at all ordinary temperatures, but the tension of its vapour below 60° F. is very small. The metallic tremors may come on suddenly or gradually, and they may be unaccompanied by salivation. The upper limbs are first affected, and then by degrees the whole muscular system. The patient is affected with tremors when an endeavour is made to exercise the muscles, so that he is unable to guide, for instance, a glass of water steadily to his lips; he cannot put his feet steadily to the ground; and when he tries to walk he breaks into a dancing trot. The muscles of mastication and deglutition are affected in advanced cases. Delirium and mania have occasionally followed the continued inhalation of mercurial fumes. See OCCUPATION-DISEASES.

DIAGNOSIS.—The diagnosis of mercurial tremors is usually not difficult. It must be admitted, however, that in some cases the tremors produced by mercury are in no way distinguishable from those due to the now well-recognised disease known as disseminated, multiple, or insular sclerosis. The former are less readily confounded with ordinary shaking palsy (paralysis agitans) and the convulsive movements of chorea. The history of exposure to mercury will seldom be absent. In paralysis agitans the tremors are finer, and occur when the patient is at rest; and the peculiar forward gait, as if the patient were endeavouring to pass from a walking to a running pace, is characteristic. The metallic tremors come on only when the muscles are exerted, and usually they entirely cease when the patient is lying at rest or is asleep. The same may be said of the tremors of disseminated sclerosis; but here we have the peculiar consensual rotation of the eyes known as nystagmus. In paralysis agitans, when told to raise the affected hand, or to protrude the tongue, the patient performs both actions steadily. In mercurial tremors, and in disseminated sclerosis, the case is different—the tongue when voluntarily protruded is tremulous, and the patient cannot raise his hand, when requested to do so, without shaking. In both mercurial tremors and the tremors of insular sclerosis the muscular agitation ceases for the most part during sleep. In one form of metallic tremors the movements approach in character the convulsive movements of chorea.

TREATMENT.—In chronic mercurial poisoning it is obvious that the patient must at once be removed from the further influence of the metal. Masks worn over the mouth are not of much use. In mercurial tremors cessation from working with the metal and mild tonics of iron usually suffice for the speedy restoration to health; but the tremors occasionally persist throughout life. For salivation and the more formidable gangrene of the mouth, besides cessation of the administration of the metal, and the exhibition of tonics, potassium iodide may be given. Astringent gargles and active local treatment may perhaps be necessary.

THOMAS STEVENSON.

MESENTERIC CYSTS. — See ABDOMINAL CYSTS.

MESENTERIC GLANDS, Diseases of.—

1. Acute Congestion and Inflammation.—These glands are liable to become congested or inflamed in connection with any inflammatory condition affecting the intestinal canal. The situation and number of glands implicated will correspond mainly with the portion of bowel involved. They become enlarged, but the changes are seldom such as to give rise to any evident symptoms, and they subside as the cause of the irritation ceases to operate. When infected with pyogenic cocci, suppuration may supervene and fatal peritonitis follow.

In this connection allusion may be made to the special implication of the glands in typhoid fever and dysentery. The nature of the changes which they exhibit is described in the articles on these diseases, and the glands affected correspond to the portions of intestine involved. They do not always return subsequently to their normal condition, but may remain permanently atrophied or otherwise changed. In exceptional instances the glands in typhoid fever have suppurated or sloughed in their interior, giving rise to symptoms simulating perforation of the intestine, and they have even ruptured into the peritoneum, thus causing fatal peritonitis. See DYSENTERY; and TYPHOID FEVER.

2. Tubercular Disease.—Tubes Mesenterica.—Tuberculosis of the mesenteric glands constitutes a most important disease in children and young persons, and its occurrence has been attributed specially to the consumption of milk obtained from cows with tuberculous udders. It may be associated with tubercular ulceration of the intestines, to which it is then probably secondary, or exist independently of it, especially in children. It is frequently followed by tubercular peritonitis. The disease may be accompanied or preceded by phthisis, although this is comparatively rare in children, and the lung-affection is often secondary in these subjects. In adults, on the other hand, tubercular disease of the glands, when it does occur, is, in the large majority of cases, a complication of phthisis, intestinal ulceration, due to infection by swallowed sputum, being present at the same time.

The changes in the glands are similar to those characteristic of tuberculosis elsewhere in the lymphatic glands, namely, an increase in the number of cells, followed by degeneration and caseation, and ultimately by calcification should the case last sufficiently long; and it is usual in fatal instances to find these conditions associated in different glands. Tubercle-bacilli are present in more or less abundance. Should recovery take place all the involved glands may be converted into inert chalky masses, in which condition they remain for an indefinite time. In other cases the fibrous reticulum may be much increased. A case came under the writer's notice many years ago in which, the patient having died from an independent acute illness, the mesenteric glands were found to be universally calcified, this being associated with scarring of the external glands and other signs of old tubercular disease, from which the patient had quite recovered. The individual glands in mesenteric disease may attain a considerable size, and when they are agglomerated into a mass a distinct tumour is formed.

SYMPTOMS.—The symptoms due to tubercular disease of the mesenteric glands are often combined with, and masked by, those resulting from intestinal ulceration, tubercular peritonitis, pulmonary tuberculosis, or disease of other structures. The digestive organs are usually disordered, and, even if there should not be intestinal ulceration, children who suffer from mesenteric disease are very liable to enteric catarrh. Hence diarrhoea, with unhealthy stools, is a common symptom, which it is often difficult to check or to prevent. In other cases the bowels are constipated. Tuberculous mesenteric glands do not seem to be painful in themselves, but colicky pains in connection with the bowels are of frequent occurrence, and the disease of the glands may have some influence in exciting such pains. The abdomen is almost always distended and prominent, owing to the accumulation of flatus, and it may be markedly tympanitic. Hence, even when the glands are much enlarged it is often impossible to feel them, but they may sometimes be made out by deep pressure with the fingers over the corresponding part of the abdomen. In some instances this region is retracted, and then the glands may be more readily felt. Signs of fluid in the peritoneum, or of chronic tubercular peritonitis, may be present.

The general symptoms in *tabes mesenterica* are usually very prominent, as evidenced by wasting, which may reach extreme emaciation, anæmia, debility, and pyrexia, marked hectic fever ultimately supervening in some cases. Cases in which mesenteric glands are the seat of tubercular disease differ much in their severity; and the symptoms may be so indefinite that it is impossible to make any positive diagnosis of its existence. A large number of cases prove fatal; but it must be remembered that even after severe symptoms recovery may take place, the glands becoming calcareous and inert. When the glandular affection is secondary to pulmonary phthisis, the outlook is very bad.

DIAGNOSIS.—Two facts must be borne in mind. In the first place enlarged mesenteric glands rarely form a mass big enough to be called an 'abdominal tumour;' and in the second place, in the absence of small palpable nodules, and indications of tuberculosis elsewhere, the diagnosis must always remain doubtful. Tuberculous mesenteric glands without any other form of tuberculosis in thorax or abdomen are very rare; whereas wasting with flatulent distension of the abdomen in infants and young children is very common and readily amenable to treatment.

TREATMENT.—This mainly consists in the treatment required for tubercular disease in general, such as the administration of cod-liver oil, preparations of iron, quinine, and other tonics; favourable hygienic conditions and surroundings; change of air, especially to the country or to the seaside; and other appropriate measures. The diet needs particularly careful attention. It should be nutritious and digestible, but has often to be modified so as to render it suitable for the condition of the alimentary canal. Remedies directed to the improvement of the state of this canal, or to the relief of symptoms connected with it, are also often required. Guaiacol may be of service in some cases. The application of Ung. Hydrarg. to the skin of the abdomen is sometimes followed by recovery as in the case of tubercular peritonitis. Dry heat, fomentations, or poultices in connection with the

abdomen may be applied, should occasion call for them. *See* PERITONEUM, Diseases of.

3. **Hypertrophy.**—It will suffice to remark under this head that the lacteal glands are liable to be more or less hypertrophied in cases of lymphadenoma, and in the form of leucocythæmia attended by glandular enlargement. *See* LEUCOCYTHÆMIA; and LYMPHADENOMA.

4. **Atrophy and Degeneration.**—The mesenteric glands atrophy in old age, and they may also become wasted and withered after previous disease, such as typhoid fever.

5. **Morbid Formations.**—*Cancer* is chiefly met with as a secondary formation, the lacteal glands being particularly liable to become affected if the intestine is the seat of malignant disease. A considerable tumour may be formed, firm and nodulated; or the glands may remain separate. Physical examination may reveal the presence of the disease. Further, localised pain, with symptoms due to pressure, should it exist, together with evidence of a cancerous cachexia, and of the implication of other organs, especially of the intestines, constitute the clinical phenomena to be found associated with malignant disease of the lacteal glands.

FREDERICK T. ROBERTS.

MESENTERY, Diseases of.—*See* PERITONEUM, Diseases of.

MESMERISM.—**DEFINITION.**—The name of the process by which, rather more than a century ago, Anthony Mesmer, the deluded (or at all events the deluding) promulgator of the doctrine of 'animal magnetism,' induced the so-called mesmeric trance or sleep. *See* MAGNETISM, ANIMAL.

This mesmeric trance is identical with the condition now known as 'induced somnambulism,' or still more commonly as 'hypnotism' or the 'hypnotic state.'

The induction of the hypnotic state or sleep has hitherto been possible in only a certain, but variable, percentage of the persons with whom trial has been made, though a successful result has been much more frequent with women than with men. According to Richet, however, the operator should not be discouraged by the failure of his first attempts with the same person; as persons may succumb on the fourth or fifth trial, and subsequently prove thoroughly good subjects for experimentation. Persons who have once been hypnotised can in general be again brought with comparative ease into the same condition, and the facility of hypnotising such persons goes on increasing after each operation, owing to the existence of a predisposing mental state. A condition of excited expectancy is indeed a decidedly favouring mental state, though one which is not essential, since, according to Braid, Heidenhain, and others, even male adults who have heard nothing on the subject, and do not know for what purpose they are being experimented with, can often be hypnotised.

In persons who are favourably disposed for passing into the hypnotic state, the condition is easily induced by weak, long-continued, and uniform stimulation of the nerves either of sight, of touch, or of hearing. This state is, on the contrary, almost always easily capable of being abruptly terminated by some strong or suddenly varying stimulation of the same nerves.

Many of the lower animals, such as frogs and

fowls, can be thrown into an extremely similar condition as a result of certain sudden and powerful sensorial impressions. Preyer distinguishes the state into which they are thrown by a different name, namely, 'cataplexy,' because the mode or physiological process by which it is induced seems to be different from that by which hypnotism is caused.

The hypnotic state or sleep is one which varies much in intensity in different persons, and in the same person at different times. The principal phenomena that are exhibited or which can be detected in hypnotised persons are the following: (1) imitative movements; (2) exaltations of special sense; (3) illusions and hallucinations; (4) analgesia, general or unilateral, or even a condition of hemianæsthesia, general and special; (5) increased reflex irritability and tonic spasms of the voluntary muscles; and (6) other miscellaneous phenomena, such as spasm of the accommodative apparatus in the eye, dilatation of the pupils, increased rapidity of respiration and of the pulse, together with profuse perspiration.

According to Charcot, when hypnotism occurs in hysterical subjects more especially, it is divisible into three states, definitely related to one another, though not always occurring in the same order. He wrote: 'These different states which, taken as a whole, include all the symptoms of hypnotism, may be referred to three fundamental types: first, the cataleptic state; second, the lethargic state; and third, the state of artificial somnambulism. Each of these states, including, moreover, a certain number of secondary forms, and leaving room for mixed states, may be displayed suddenly, originally, and separately. They may also, in the course of a single observation, and in one subject, be produced in succession, in varying order, at the will of the observer, by the employment of certain methods. In this latter case the different states mentioned above may be said to represent the phases or periods of a single process.' The hypnotic state, in one or other of its stages, seems to be akin to that met with in some sleeping persons, as well as to the states known as trance, somnambulism, and catalepsy. Its physiological cause was presumed by Heidenhain to be some inhibitory arrest of activity of the ganglion-cells of the cerebral cortex, or, as the writer would rather put it, of certain tracts of these ganglion-cells, varying in their nature or situation in accordance with the different stages of the hypnotic condition that may exist.

The scientific study of the phenomena presented by hypnotised persons is unquestionably of great interest and importance from the point of view of the higher cerebral physiology. But whether the systematic induction of such a state can ever be used as a legitimate or potent means for curing disease, or even for the alleviation of certain distressing symptoms, must be left for the future to decide. The investigations that have been made in recent years are far from being decisive in favour of the method as a remedial agent, especially when taken in conjunction with the actual harm which may result from its induction in some nervous and impressionable persons. The good use to which it was put by Esdaile in India, as a means of inducing insensibility during surgical operations before the general introduction of chloroform, ought, however, never to be forgotten.

H. CHARLTON BASTIAN.

METALLIC.—A peculiar quality of sound, which the name suggests, either elicited by percussion or heard on auscultation, especially in connection with certain adventitious sounds in pulmonary cavities. See PHYSICAL EXAMINATION.

METAMORPHOSIS (μετά, a preposition signifying change : and μορφή, I form).—In a pathological sense this word signifies a form of degeneration, in which one tissue or substance becomes chemically changed into another, as, for example, albuminous structures into fat. See DEGENERATION.

METASTASIS } (μεβίστημι, I change place).
METASTATIC }

These terms are supposed to imply the translation of a disease from one part of the body to another, such as seems to occur occasionally in gout, rheumatism, mumps, and certain affections of the skin and mucous membranes. In modern pathology the term 'metastatic deposits' is sometimes applied to secondary new-growths.

METEORISM (μετεωρίζω, I raise up).—A synonym for tympanites. See TYMPANITES.

METRALGIA (μήτρα, the womb ; and ἄλγος, pain).—Pain in the womb. See UTERUS, Diseases of.

METRITIS (μήτρα, the womb).—Inflammation of the womb. See UTERUS, Diseases of.

MIASM (μαίνω, I pollute).—This term is used vaguely to include all poisonous emanations from the soil.

MICRO-ORGANISMS.—See BACTERIA ; ENTOMOZOA ; and the various diseases with which they are associated.

MICROSPORON (μικρός, small ; and σπόρος, a seed).—*Microsporon furfur* is the fungus of Tinea versicolor, and *Microsporon minutissimum* of erythrasma. See ERYTHRASMA ; and TINEÆ.

MICTURITION, Disorders of.—Under this term will be considered those conditions which interfere with the normal performance of micturition, regarded as a physical act. The following will be treated of as disorders of micturition :

1. *Undue frequency of micturition.*
2. *Diminished size and force of stream.*
3. *Retention of urine, partial and complete.*
4. *Urine passed by an abnormal channel.*
5. *Incontinence and overflow of urine in the adult.*

6. *Incontinence of urine in the child.*

1. *Undue frequency of micturition.*—This condition was formerly known as 'irritability of the bladder.' In all maladies of the bladder, in most that affect the kidney, and in some diseases of the rectum, unnaturally frequent micturition is present. It may vary in degree, and exist alone as a single symptom ; or it may, as is much more usually the case, be accompanied by other symptoms, which aid the diagnosis. Thus it is present in all the inflammatory conditions of the bladder after the administration of certain drugs, whenever calculi, foreign bodies, or tumours exist there, and in parasitic affections of the urinary organs. Also when the bladder is full, and either habitually does not empty itself, or when absolute retention is present, in

either case the want to pass water is frequent and pressing. It is often present in stricture of the urethra, and in inflammations of that passage ; also in chronic pyelitis, in chronic nephritis, in Bright's disease, in tubercular disease of the kidney, in diabetes mellitus, as a result of the increased quantity of urine, or when urine is morbidly acid, or alkaline. It is present likewise during hysterical states, and under emotional excitements in many persons of either sex, and whenever the watery elements of the urine are rapidly and abundantly secreted, arising either from dietetic idiosyncrasy, the administration of diuretics, or changes of climatic condition. The term *urgency* indicates the inability to postpone the act of micturition when the desire has occurred. It is associated with frequency, and is more particularly noticeable in disease affecting the bladder and urethra. *Straining* is another not uncommon symptom which accompanies vesical irritability. It is the uncontrollable and ineffectual endeavour to micturate which calls into play not only the ordinary muscular mechanism of micturition to its full extent, but also many other muscles of the body. It occurs, even when the bladder is free from urine, in inflammatory conditions of the organ and in stone. Straining then usually follows immediately after the act of passing water, and subsides almost directly. But the most pronounced form is seen in cases of retention. The attack or spasm commences with slight desire to micturate, rapidly increasing in severity and intensity until at last almost all the muscles of the body are brought into use in a fruitless endeavour to expel the contents of the bladder. Pain along the urethra, in the lower part of the abdomen, and in the back is often complained of. The struggle may continue for a considerable time, but it gradually decreases in severity and finally subsides altogether, leaving the patient tired and exhausted. Such spasms may come on at frequent intervals, depending to some extent on the degree of distension of the bladder. In acute cases of retention they may occur every quarter of an hour or so, but in chronic they are often absent for a considerable period, and are then only evoked by movement or other stimuli which so frequently produce the desire in health.

2. *Diminished size and force of stream.*—These may occur either with or without organic obstruction of the urethra. The size is always diminished in congenital narrowing of the prepuce or of the external meatus, though the force may be increased ; in organic stricture, in urethral calculus and growth, both are generally diminished, and mostly also in enlarged prostate, though loss of force is most marked. They may occur from inflammation of the urethra and prostate ; and when the power of the bladder to expel its contents is impaired by paralysis, atony, or by other causes, the force is greatly reduced but the size may remain comparatively full. Occasionally the channel is narrowed by irregular actions of the surrounding muscles, and thus 'spasmodic stricture' (an objectionable term) is spoken of as producing a diminution of the stream.

3. *Retention of urine.*—Retention of urine, partial or complete, is not to be confounded with 'suppression,' the latter being of course defective action of the secreting organ, so that no urine is produced, and the bladder remains empty. Retention is the result in almost all cases of mechanical obstruction, such as enlarged prostate from hypertrophy, tumour,

presence of blood-clots in the urethra or bladder, and occasionally from rupture of that organ. One of its most common causes is a narrow stricture of the urethra, especially when inflammation follows the use of instruments, or when associated with alcoholic excess; inflammation alone may also occasion retention, as during an attack of gonorrhoea. Impacted calculus is not infrequently the cause; sometimes also, but most rarely, the spasmodic action referred to above.

TREATMENT.—As the cause is a purely mechanical one in the great majority of instances, the remedy which should be applied without delay is also a mechanical one, namely, a catheter of appropriate size and kind. If a suitable instrument, however, is not within reach, medicinal agents may be of service until it can be obtained. At the head of these no doubt is opium, which allays involuntary straining, and sometimes thus enables the patient to relieve himself by the natural method, at all events to some extent. It should be given in full doses, for the purpose either of relieving the patient's suffering and anxiety, or of acting favourably on the function; and the error in practice which has been most common is to give doses of 10 to 15 minims of laudanum, when 30 to 40 or more were necessary, and might have been highly useful. Of course the form of opiate may be varied, according to the habits of the patient or the views of the attendant. Simple opium is merely mentioned here as the type. Rest in the recumbent position with buttocks and legs raised is to be enjoined. Local bathing, as hot as it can be borne, is also a valuable adjunct; as, for example, a hip-bath, commencing at 100° F., which may with advantage be carried gradually up to 110° in these circumstances. Diuretics, often given, are for the most part injurious; that is to say, when the cause is a mechanical one. As a general principle, also, it is not to be forgotten that active purgation commonly promotes the expulsive action of the bladder.

4. *Urine passing by abnormal passages.*—The urine may escape by abnormal channels, passing from different parts of the urethra to the surface, most commonly by that of the perinæum, constituting urinary fistula. This condition is necessarily named as one of the 'disorders of micturition,' but its treatment belongs exclusively to the surgeon.

5. *Incontinence and overflow of urine in the adult.*—It not infrequently happens that a man between fifty and sixty years of age finds himself gradually compelled to pass urine oftener than has been his custom hitherto, and that, the tendency still increasing, he is compelled to pass it every hour or two, both by day and night; and sometimes even, especially if not relieved by treatment, he may pass it during sleep. During the earlier stages of the affection, it is by no means uncommon that the individual learns or believes that his experience is natural to age, and is either not amenable to treatment, or is not worthy of serious notice. Again, the condition described is often loosely spoken of as 'incontinence' of urine; of which, however, it is not only not an example, but indeed indicates the presence of a condition precisely contrary. The cause of these symptoms is a bladder unable to empty itself, always therefore partially if not completely filled, from which the surplus must be either frequently discharged, or be passed 'incontinently.' The important point, then, is never to lose sight of the fact that frequent micturition, and above all

urine involuntarily passed by elderly men, in nineteen cases out of twenty indicates retention (requiring the catheter), and not incontinence.

True incontinence, which means inability to retain, on the part of the bladder, is a very rare occurrence, and is present only in cases of disease in the nervous system producing paralysis in other parts of the body, as well as the bladder. When the bladder-symptoms alone are present, and no signs of paralysis elsewhere exist, it may be held as almost absolutely certain that the bladder itself is not paralysed. It may be over-distended with fluid from enlarged prostate; or its coats may be thinned and atonic, and so unable to contract on their contents.

TREATMENT.—In these partial retentions of urine, producing its overflow and involuntary discharge, the remedy consists in the more or less frequent use, according to circumstances, of a soft or flexible catheter, under rigid aseptic precautions, and the case is mainly surgical. There are some instances in which restoration of the power of the bladder may be attempted by medicinal agents, such as strychnine and iron, or by electricity, but their effect is little or none, apart from the habitual emptying of the organ by artificial means. In some cases perhaps they may be advantageously associated with the surgical treatment.

6. *Juvenile incontinence.*—In the earliest periods of childhood an undue frequency of passing water is often to be observed among individuals of both sexes, more commonly in boys than in girls. As age advances the infirmity usually lessens, and then disappears; while in exceptional instances it continues, without change, to puberty, and even for some years after that period has arrived. But the peculiarity of the case is that the urine is passed unconsciously during sleep, and this forms the most serious symptom. In spite of all precautions, a quantity of urine is discharged every night during deep sleep, an occurrence of which the child is quite unaware, and which as he advances in age he is wholly unable to control, however strong may be his disposition to do so. On the bladder becoming distended reflex action of the vesical muscular coats takes place, and the contents are discharged. The flow of urine is determined, as it would appear, not by inability on the part of the bladder to retain the ordinary quantity of urine, for the patient does so by day, but by its undue excitability or readiness to contract, so that the water is passed while consciousness is lost, during sleep. In a few instances, certain aberrations from a good standard of health seem to favour the production of these phenomena, especially sources of irritation in the rectum, which produce activity in that muscular apparatus, involving also the kindred muscles of the bladder, which are so closely associated. Thus the presence of ascarides, thread-worms, rectal polypi, fissured anus, or urethral caruncle, may suffice to occasion expulsive action in the bladder. During the period of infancy and early childhood the nervous system is highly impressionable, and the habit in question being accidentally set up, its persistence may result solely from repetition through the force of custom, long after the original cause has disappeared.

Sometimes slight malformations of the male organ favour the occurrence of incontinence; such as a narrow meatus, or a long prepuce which is never retracted and is consequently in an unhealthy state.

Precocious development and extreme activity of the mental faculties, producing disturbed sleep,

seem to favour the occurrence of incontinence. On the other hand, it is sometimes associated with a morbid deficiency of intelligence. In many of the cases an hereditary tendency can be traced.

TREATMENT.—The treatment ordinarily necessary may be to some extent inferred, when examination of the patient has determined the presence or absence of the conditions named. This done, the next indication is to subdue the activity of the expulsive function of the bladder by some agent which possesses that power. The most powerful for this purpose is undoubtedly belladonna. Thus, if administered to an adult whose powers of expelling urine are feeble, such, for example, as are commonly met with in advancing years, complete retention of urine is often produced. Now, as has already been observed, in not a few of the cases of so-called 'juvenile incontinence,' its existence is due solely to persisting habit after the original occasion of it has long ceased; and these are almost certainly and sometimes rapidly cured by administering the drug in question. The object is to induce a partial paralysis of the bladder for a period of some weeks, and, if the case is obstinate, even for two, three, or four months, and by this means not only to destroy the old habit, but to develop a new one, namely, a habit of retention, and the annoyance disappears entirely and for ever. On meeting, therefore, with a case, whether in childhood or youth, the first indication is to correct any manifest deviation from the ordinary standard of general health; and, secondly, to administer belladonna persistently. Small doses, suited to the age of the patient, suffice at first, and may be given every afternoon and evening only—say from three to six minims of the tincture on each occasion during the first week. In the second and third weeks of treatment, the dose may be augmented one-fourth; in the fourth and fifth weeks the original dose is raised a third or half; in the meantime some improvement will almost certainly be manifest. Since the ability to bear belladonna increases rapidly as the system becomes habituated to it, a larger dose may be given during a further term of three successive weeks, by which time the involuntary discharge of urine probably ceases. If not, a gradually increased dose must still be continued. After this the dose may be gradually diminished, and at a rate more rapid than that by which it was augmented. The habit of retention has probably been formed by this time, and when cessation from medicine takes place, no recurrence of the symptoms will be observed. But it must be confessed that a troublesome minority is met with in which the influence of the belladonna has not been permanent. It almost invariably produces some improvement, however, and it is worth while to be careful that the drug has been well prepared. There still remain modes of treatment of a local character, which may prove successful in these cases. These do not include blisters on the sacrum; apparatus to prevent the patient lying on his back, when asleep; arrangements to arouse him during the night once or twice to pass water voluntarily, and such measures—all of which are palliative means, and do little towards a radical cure. The use of a flexible bougie, small of course for children, passed daily, and removed in the course of a minute or so, is sometimes successful, especially when both nocturnal and diurnal incontinence are associated. But if this fails, the injection of a solution of nitrate of silver by means of a sufficiently long tube into

the prostatic portion of the urethra and neck of the bladder has been found in the hands of some surgeons a valuable remedy. The process generally known to surgeons as 'instillation,' by means of a syringe and a flexible tube, is perhaps the best. For young women up to the age of eighteen or twenty in whom this unfortunate infirmity still exists, it is said to be almost always successful. It should be applied immediately after the bladder is emptied, in quantity, say, of a drachm, and of a minimum strength of ten grains to the ounce, up to treble that strength if necessary for subsequent applications. Enough should be employed to produce decided smarting, which shall continue for a day or two. A week or two should be permitted to elapse between each application.

It would not be right to omit the mention of other remedies besides belladonna, which may be used either alone or in combination with it. Such are the tincture of the perchloride of iron, strychnine, and bromide of potassium. The last-mentioned, given at night only, has sometimes a manifestly beneficial effect.

HENRY THOMPSON.
H. T. HERRING.

MIDDLE PARK HOT SPRINGS, in Grand County, Colorado, U.S.A.—Thermal Waters. See MINERAL WATERS.

MIGRAINE.—A synonym for megrim. See MEGRIM.

MILIARIA (*milium*, a millet-seed).—SYNON.: Sudamina; Miliaria crystallina; Fr. *Miliaire*; Ger. *Friesel*.

DEFINITION.—A superficial non-inflammatory vesicular eruption of the skin, generally associated with profuse sweating.

DESCRIPTION.—The proximate causes of miliaria are heat and sweating, and are therefore especially seen in acute rheumatism, acute pneumonia, and typhoid fever. The millet-sized vesicles—either few or very many—are developed between the layers of the stratum corneum, owing to the free flow of the secretion, together with occlusion of the orifice of the ducts; are generally discrete; and are dispersed irregularly over the chest, neck and abdomen, and elsewhere. They are thin-walled, and contain at first pellucid sweat of faintly acid reaction; when the serum becomes milky and opaque, the eruption is called *miliaria alba*. When the retention is followed by inflammation the term *miliaria rubra* is applied to the general redness of the skin of the affected areas, which are crowded with discrete vesicles on inflamed bases. When left to themselves the vesicles subside and dry up into an extremely thin scale. Miliaria, therefore, calls for no special treatment. See SUDORIPAROUS GLANDS, Diseases of.

JOHN HAROLD.

MILIARY ANEURYSMS.—Minute dilatations in connection with the small blood-vessels; especially met with in the brain. See BRAIN, VESSELS OF, Diseases of.

MILIMUM (*milium*, a millet-seed).—SYNON.: *Grutum*; *Strophulus albidus* (Willan).

The name *milium* is applied to small, firm, globular, pearly-white bodies, few or numerous, seen under the cuticle especially of the eyelids, cheeks, temples, orbits, and occasionally on the male and female genitals. In size they vary almost

as much as ordinary pins' heads. They are not accompanied by any sign of inflammation, are usually arranged asymmetrically, and may undergo calcification ('*cutaneous calculi*'). They are unattended by subjective sensations. Infants and young adults are most frequently affected.

Milia consist of the 'distended acini' of sebaceous glands, the ducts of which have been occluded. According to Robinson, of New York, milia originate from 'miscarried embryonic epithelium from a hair follicle or from the rete.'

TREATMENT.—Milia may be opened by a fine needle, and the contents evacuated by curetting; or recourse had to electrolysis.

See PEMPHIGUS; COLLOID MILIUM.

JOHN HAROLD.

MILK, Poisoning by.—See FOOD, POISONOUS.

MILK-FEVER.—SYNON.: Ephemeral Fever; Fr. *Fièvre de Lait*; Ger. *Milchfieber*.

A term applied to a transient rise of temperature which sometimes occurs on the third or fourth day after delivery, on the supposition that it was due to the onset of lactation. It is now generally attributed to constipation, emotion, or septic absorption, though some believe it may occasionally be due to unusual tension in the breasts. Any rise of temperature on or about the third day should be carefully investigated, as it rarely occurs if antiseptics have been carefully used during labour. See PUERPERAL DISEASES.

MILLAR'S ASTHMA.—See THYMUS GLAND, Diseases of.

MIMESIS (μιμῆσαι, I imitate).—A term applied to the phenomena of a disease, which resemble or imitate those of another disease.

MIND, Disorders of.—See IDIOCY; and INSANITY.

MINERAL WATERS.—DEFINITION.—Mineral Water is the name given to those waters which, on account of the different saline or gaseous substances which they hold in solution, or of their elevated temperature, are used in the treatment of disease, either internally or in the various forms of baths.

The science that treats of the effects of mineral waters and baths on a great number of chronic maladies is called *Balneotherapeutics*. In a wider sense this branch of medicine comprises also the use of sea-baths and of common water, but these subjects are treated in separate articles. See HYDROTHERAPEUTICS; and SEA-AIR.

Courses of mineral waters and baths are to be regarded as methods of treatment analogous to courses of other remedies; but they are much more complicated, not only because many of the mineral waters are in themselves compound remedies containing several active substances in combination, but also because in most courses of waters or baths the invalid is influenced in body and mind by several other powerful agents: such as travelling, change of social conditions, occupation, scene, diet, and habits in general, mostly including increased exercise; and by change of climate. Each of these influences has in itself a powerful action, and to their combination we must often ascribe a great part of the curative effects of balneotherapeutic courses; they ought therefore to be carefully considered in

every individual case as part of the plan prescribed.

GENERAL COMPOSITION AND CLASSIFICATION. The principal constituents of mineral waters are: Water, sodium, magnesium, calcium, and iron; combined with hydrochloric, sulphuric, carbonic, and hydrosulphuric acids, the two latter existing also in some waters 'free,' that is, uncombined with bases. Nitrogen and oxygen are likewise present in most mineral waters in various proportions; and in some there are also silica, arsenic, bromine, iodine, lithium, manganese, potassium, alum, and other substances in small quantities.

The substances dissolved in mineral waters are derived from the surface-soil and the rocky strata through which the water deposited from the atmosphere passes. The dissolving power of this water is much increased by the gases which it absorbs, especially carbonic acid and oxygen. The constitution of mineral waters, therefore, varies according to the nature of the strata through which they have passed.

The different mineral waters may be grouped in various ways, as, for instance, according to their chemical constituents, their temperature, their geological origin or geographical distribution, or their physiological or therapeutical actions.

The chemical classification, imperfect though it is, offers the advantage that it directs the attention at once to the most important constituents of the water. Some of the classes, however, are not named according to the substances contained in them in the *largest quantity*, but according to those considered most *potent*; such as the iron, arsenic, and sulphur waters. Another difficulty in the classification is, that some mineral waters contain several active substances in sufficiently large proportions to allow of their being placed in different classes; and, again, that some springs are so deficient in active principles as to render it doubtful where to place them. Of these latter, some appear to owe their virtues to the water alone, and its temperature, aided by the climate with which they are associated. Beginning with the latter as the most simple, we may group the mineral waters in the following principal classes:

I. *Simple Thermal Waters*; II. *Common Salt or Muriated Saline Waters*; III. *Alkaline Waters*; IV. *Sulphated Saline Waters*; V. *Iron or Chalybeate Waters*; VI. *Arsenical Waters*; VII. *Sulphur Waters*; VIII. *Earthy and Calcareous Waters*.

Some of the waters are chiefly used for bathing, others more for drinking, the majority for both purposes. In the consideration of the uses of the different spas, it is important to distinguish between the effects produced by the baths and those caused by the internal use of the waters.

The term 'baths' comprises not only the ordinary tub-bath, but also swimming-baths, or piscines; partial baths for the feet, the hands, and other parts; douches of great variety; vapour-baths; carbonic-acid-baths; and mud-baths. At many places also inhalations of vapour and pulverised spray form part of the treatment.

I. *Simple Thermal Waters*.—The simple thermal waters are characterised by poverty in solid and gaseous substances, and therefore by low specific gravity; by perfect transparency; by great softness; and by elevated temperature—varying in the different spas from about 80° to over 150° F.

Some of them contain nitrogen in larger proportions than the gases of waters usually do, others more oxygen. They are often called *indifferent* waters, on account of the absence of special mineralisation; and also *wild baths* (*Wildbäder*), on account of their being usually situated in wild mountainous regions.

ACTION.—The water of this class of spas when taken internally probably acts only as ordinary, pure, warm water. By the drinking of warm as well as of cold water, the stomach is washed out; the secretion of bile, saliva, pancreatic juice, urine, &c., is increased; the tissue-change is augmented, and the removal of effete matters from the tissues and blood promoted; and by the acceleration of the retrogressive tissue-change, the progressive tissue-change becomes facilitated. As differences between warm water and cold water for drinking purposes, we may mention that the latter acts more as a local excitant on the stomach, while the former is more easily absorbed, and makes less demands on the powers of the constitution, by not causing any expenditure of heat.

The simple thermal waters are much more used for *bathing* than for drinking-courses; and the baths, as such, have probably the effects of ordinary warm baths, varying according to the temperature of the baths, and the time spent in them.

As the *fundamental effects of warm baths*, which effects form part of the action of all kinds of warm baths, simple as well as mineralised, we may regard—

(1) That they soften and cleanse the skin more rapidly than cold baths, and prepare it for perspiration.

(2) That they equalise and diminish the loss of heat, and, according to the temperature of the bath, lessen or prevent it altogether; and that, in the hot bath, heat is even added to the body.

(3) That the flow of blood to the surface and its circulation in the skin are promoted.

(4) That the organic functions and the tissue-change are slightly stimulated, or rather facilitated, without any strong reaction on the part of the organism.

(5) That the nervous system and muscular irritability are calmed.

(6) That the absorption of exudations is promoted.

These effects, as already mentioned, vary considerably with the degree of heat. In the *tepid bath* (from 80° to 95° F.) the central nervous system and the action of the heart are but slightly influenced; in the *warm bath* (from 96° to 102° or 103° F.) the heart's action is quickened, but the respiration is generally only slightly affected; in the *hot bath* (from 103° to 110° F.) the central nervous system becomes much more excited; not only is the heart's action further accelerated, but the respiration becomes rapid, and sometimes irregular; and the hyperæmia of the skin leads to perspiration on removal from the bath. Baths of a temperature above 110° F. are scarcely ever used, and only for a very few minutes.

The effects vary also considerably, according to the duration of the immersion.

USES.—The *drinking-courses* of these waters may assist in the treatment of irritable forms of affections of the throat, stomach, and intestines, often associated with spasmodic cough, cardialgia, constipation from sluggish secretion of bile and intestinal secretions; they do so by diluting these

secretions, by increasing the tissue-change, and by removing used-up material; in this way they are useful in chronic rheumatism and gout.

One of the main uses of the *simple thermal baths* is to allay over-excitability and hyper-sensibility of the nervous system in its various spheres; thus they often act beneficially in cases of neuralgia, hyperæsthesia, painful menstruation, and hysterical tendency. Their reputation in painful wounds and cicatrices is historical. In these cases, as well as in chronic rheumatism in its various forms and sciatica, the hotter are more useful than the tepid baths. In some forms of paralysis and loss of muscular power depending on peripheral changes, such as exudations into nerve-sheaths, good effects are produced; but if they are caused by changes in the centres of the nervous system, not much is to be expected. In gout the internal use of other mineral waters is generally required, but as second courses the simple thermal waters are often useful; in many delicate gouty persons, however, the balneotherapeutic treatment ought to be restricted to courses of tepid baths, aided by climate and diet. Most of these conditions can be also treated with other waters.

The ordinary thermal bath requires in many cases the assistance of douches and massage; and if atheroma of the cerebral vessels be suspected, douches are preferable to the ordinary hot baths, either alone or in combination with massage.

ENUMERATION AND SELECTION.—The choice of a simple thermal spa is to be guided, not by the name of the disease alone, but also by the state of constitution, and many concomitant circumstances. The simple thermal waters deserve, *cæteris paribus*, the preference, when gentle management is required—when it is desirable to make as slight demands as possible on the powers of the constitution. Their action is in this respect greatly assisted by the mountainous climate or large forests in which they

Name	Country	Elevation (approximate). Feet	Temperature of springs. Fahrenheit
Panticosa	Spain (Pyrenees)	5000	77°-92°
Leukerbad (Loèche-les-Bains)	Switzerland	4600	102°-122°
Bormio	Italy	4300	90°-104°
Gastein	Austrian Alps	3300	95°-114° 8'
Pfäfers	Switzerland	2115	100° 4'
Johannisbad	Bohemia	2000	86°
Bagnères de Bigorre	France (Pyrenees)	1850	90°-95°
Ragatz	Switzerland	1570	96°
Badenweiler	Baden	1425	86°-90° 5'
Landeck	Silesia (Prussia)	1400	66°-84° 2'
Wildbad	Württemberg	1323	95°-98° 6'
Plombières	France	1310	66°-156°
Luxeuil	France	1300	65°-165°
Neuhaus	Styria (Austria)	1200	70°-92°
Liebenzell	Württemberg	1113	70°-82°
Warmbrunn	Silesia (Prussia)	1100	96° 8'-104°
Töbelbad	Tyrol	1090	70°-82°
Aix-les-Bains	Savoy, France	1060	86°-120°
Buxton	England	1000 (nearly)	82°
Schlangenbad	Nassau (Prussia)	900	81°-86°
Néris	France	800	114°-125°
Römerbad and Tüffer	Styria (Austria)	700-800	93°-100°
Teplitz	Bohemia	650	95°-120°
Lucca	Italy	500	100°-120°
Dax	France	130	127°-140°
Bath	England	100	100°-120°

are often situated. The selection of a special spa in a given case depends on the nature of the case in the widest sense ; on the degree of elevation which is desirable ; on the means of treatment obtainable and customary at the different spas, including the most important agent—the resident physician ; on the accommodation, the food, manner of living, and social conditions ; on the distance and means of reaching the spa ; on the travelling power of the patient ; and on many other circumstances. Information on these subjects can only be obtained by the study of larger works, and by personal visits. We give above only the names of the principal spas of this class arranged according to their elevation.

Many other slightly mineralised warm waters, whose principal action is to be referred to water and heat, might be mentioned here, while several of the places contained in the list, as Leukerbad, Bormio, Bagnères de Bigorre, and Bath, might find places in other divisions.

The very hot Algerian baths, Hammam-Meskoutin, Biskra, and Hammam-R'irha, the last beautifully situated some sixty miles from Algiers, belong likewise to this class.

The United States of America are rich in simple hot springs, which are partly already in use, partly awaiting development. Sooner or later they will equal and perhaps surpass the most celebrated thermal waters of Europe. We will only mention here a few of them : the ' Hot Springs ' in Virginia ; the ' Hot Springs ' in Arkansas ; the ' Calistoga Hot Springs,' California ; the ' Geysers,' California ; the ' Paso Robles Hot Springs ' in California ; the ' Idaho Hot Springs ' in Colorado ; the ' Warm Springs ' in North Carolina ; the ' Warm Springs ' in Georgia ; ' Lebanon Springs ' in Columbia county, New York ; the ' Warm Springs ' and the ' Healing Springs ' in Bath county, Virginia.

Allied in their action, though more powerful in their demands on the system and in their effects, are the natural hot-vapour baths in the large cave of Monsummano in Upper Italy, and in the smaller excavation in the rocks of Battaglia in the Euganean mountains.

II. Common-Salt or Muriated Saline Waters.—COMPOSITION.—Common salt, or chloride of sodium, is the principal solid constituent of the waters of this class, but this substance is contained also in many other mineral waters, especially in some alkaline, some sulphur, and some sodium-sulphate waters, and has a considerable share in the effects of these waters. Some of the springs in this class contain also appreciable quantities of chlorides of calcium and magnesium, of iron, and of carbonates of sodium, lithium, magnesium, and lime, by which their action is modified.

ACTION.—In order to appreciate the action of the common-salt waters, we must bear in mind that common salt forms part of all the tissues and juices of the body ; that it promotes digestion ; that it is essential to the formation as well as the disintegration of cells and tissues ; that it stimulates not only the retrogressive, but also the progressive tissue-change or nutrition of the body ; and that it is a great agent in the processes of secretion and absorption. Chloride of sodium stimulates the secreting apparatus of the stomach and intestines, and hence the action of the bowels and the circulation of the portal system, and indirectly the general circulation. It quickens the tissue-change ; and

through this, as well as the increased circulation, it promotes absorption of pathological products, without lowering the organism. In larger doses, however, beyond about five drachms *per diem*, irritation of the mucous membrane of the stomach and intestines may be produced. The action of the common-salt waters is modified by their accompanying properties, especially by the carbonic acid contained in them, by their temperature, and by the degree of their concentration.

The *carbonic acid* in this and other classes of waters quiets the sensitive nerves of the stomach ; stimulates the secretion and peristaltic action of the stomach and bowels. In large quantities, however, if not rapidly ejected by eructation, it may, by distending the stomach, interfere with the action of the heart, or may produce, on being absorbed, poisonous effects on the blood and nervous system. The presence of carbonic acid in salt waters increases the effects of chloride of sodium on the stomach and intestines, and by accelerating the passage of the waters from the stomach into the intestinal canal, promotes the action of the bowels.

Elevation of the temperature of the water produces more rapid absorption, and thus diminishes the local and increases the more distant and constitutional effects.

Concentration increases the local stimulation.

As to the action of these waters in the form of *baths*, the chloride of sodium and other chlorides (though any absorption through the skin of these and other salts contained in mineral waters is doubtful, or, at all events, forms only a small part of their therapeutic effects) stimulate the cutaneous ends of the nerves and the capillaries, and promote through this the nutrition and tone of the skin, and indirectly the tissue-change, an action which is heightened by the presence of carbonic acid, as witnessed at the gaseous saline baths of Rehme and Nauheim. The action of the salt and of the carbonic acid on the skin is, however, not to be regarded as merely local, but as transmissible from the nerve-ends to the various nerve-centres, and producing through these reflex effects, especially on the heart and blood-vessels, but also on other organs.

USES.—Salt waters and salt baths are useful in weakness of the skin ; in tendency to rheumatic fever or bronchitis ; in retarded convalescence from acute and chronic illness ; in enlargements of joints from preceding inflammation ; in many forms of anæmia and chlorosis—especially those where iron alone is not borne ; in numerous cases of tropical cachexia ; and in cases of sluggish circulation in the portal system, which leads to innumerable varieties of digestive troubles, to congestion of the liver, and of the pelvic organs in women, and to piles.

ENUMERATION AND SELECTION.—The same classes of cases, as far as the name goes, are treated alike by muriated alkaline and sulphated waters. The individual conditions must guide the practitioner in deciding for either the one or the other kind of waters, and for the special spa, according to the strength of the springs, the additional ingredients, such as lithium and arsenic at Baden-Baden, the amount of carbonic acid, and the climatic and concomitant conditions. Spare and pale persons, we may mention, mostly bear the common-salt waters better than strongly alkaline and sulphated waters. Common-salt waters are to be found in almost all countries ; we only give the most important or best known. In *England*: Droitwich—

perhaps the strongest of all brines, with good arrangements, Nantwich, Ashby-de-la-Zouche (Ivanhoe Baths), Middlewich, Woodhall, Harrogate, and Llandrindod; Leamington and Cheltenham contain likewise much common salt, in addition to sulphate of sodium. In *North America*: St. Catharine's Wells, Michigan Congress Spring, Spring Lake Well, Fruit Port Well, the celebrated Saratoga Springs, and the Ballston Spa. In *Germany*: Kissingen, Homburg, Rehme-Oeynhausien, Nauheim, Kreuznach, Soden, Pyrmont (which contains salt as well as iron springs), Wiesbaden, Hall in Austria, Hall in the Tyrol, Hall in Württemberg, Reichenhall, Ischl, Kreuth, Dürkheim, Kosen, Koenigsdorff-Jastrzemb, Krankenheil, Mondorf, Salzungen, Canstatt, Cronthal, Baden-Baden, Niederbronn (Alsatia), and several others. In *France*: Bourbonne-les-Bains, Lamotte-les-Bains, Bourbon-l'Archambault, Bourbon-Lancy, Balaruc, Salins, and others. In *Italy*: Ischia, Castellamare, Castro-Caro, Monte Cattini, Salsomaggiore, La Porretta. In *Switzerland*: Bex and Rheinfelden. In *Spain*: Las Caldas de Besaya, Las Caldas de Estrae, and Las Caldas de Montbuy.

III. Alkaline Waters.—COMPOSITION.—The alkaline waters contain *carbonate of sodium* as a prominent constituent; they are also more or less rich in *carbonic acid*; and some are distinguished by so large a proportion of *chloride of sodium* as to warrant a subdivision into—(1) *simple alkaline waters*; and (2) *muriated alkaline waters*.

ACTION.—Oxidation and tissue-change seem to be greatly influenced by the presence of soda; various proteid bodies seem to be kept in solution by it; it has a considerable share in the secretion of saliva and bile, and in the digestive processes; and, according to Liebig, it acts as a vehicle for the carbonic acid from the blood to the lungs.

Carbonate of sodium may be considered as an antacid, as a diuretic, as a promoter of tissue-change, and as a solvent. The beneficial effects of alkalis are in general produced only by a systematic use of *small doses*; while *large quantities* cause emaciation by their excessive solvent effect, and diminish the tissue-change by their depressing influence on the heart's action. The action of soda differs in this respect from that of chloride of sodium, which even in considerable doses increases the tissue-change, and does not so easily exercise an emaciating effect.

USES.—The conditions in which alkaline waters are mostly employed are certain forms of dyspepsia, with undue acidity of the stomach; congestive conditions of the liver from sluggish portal circulation; tendency to gall-stones; diabetes; uric-acid diathesis, and its results—gravel and lithiasis; some forms of gout; and especially chronic catarrhal affections of the mucous membranes of the respiratory, digestive, and genital organs.

ENUMERATION AND SELECTION.—Where it is necessary to improve the state of the blood, or to avoid emaciation, the muriated alkaline are preferable to the simple alkaline waters.

1. The principal spas with *simple alkaline waters* are—(a) *Hot*: Vichy, Neuenahr, Mont Dore, Chaudes Aigues, and Neris, the last three being feebly mineralised; (b) *Cold*: Vals, Salzbrunn, Le Boulou, Evian, Bilin, Apollinaris, Gerolstein, Fachingen, Geilnau, Wilhelmsquelle, Taunus, Giesshübel, Soultzmat, and Marcols. In *North America*—the Bladon Springs in Alabama, the Sheldon Springs in Vermont.

2. The chief *muriated alkaline waters* are—(a) Ems, Royat, and La Bourboule, which represent the *hot springs*; (b) Luhatschowitz, Selters, Gleichenberg, Roisdorf, Rosbach, Vic-sur-Cère, and Toennistein, which are *cold*. In *North America* the Congress Springs in California, and the St. Louis Spring in Michigan, belong to this class. The so-called California Seltzer Springs are peculiar in so far as they contain a larger quantity of carbonate of magnesium (10 grains in a pint) than of either carbonate or chloride of sodium. These springs seem therefore to offer advantages in oxaluria, and in some gouty persons.

We ought to remark that several of the waters in this class, especially La Bourboule and Mont Dore, contain *arsenic* in appreciable quantities, and will, therefore, be mentioned again under arsenical waters, while others contain lithium, as Royat.

IV. Sulphated Waters.—COMPOSITION. We include under this term those springs which are characterised by a preponderating amount of the sulphates of sodium or magnesium, or both sulphates together. They may be subdivided into (1) *simple sulphated waters* or *bitter waters*; (2) *alkaline sulphated waters*, which latter contain also carbonate and chloride of sodium; and (3) *muriated sulphated waters*.

ACTION.—The *bitter salts* can scarcely be said to be constituents of the organism; they seem to act by stimulating, and in larger doses irritating, the mucous membrane of the stomach and alimentary canal, causing thin watery secretion, and in large doses diarrhoea. Sulphate of sodium is less irritating than sulphate of magnesium. The peristaltic action of the bowels is likewise increased by them. Their continued employment is apt to cause emaciation. By the presence of carbonate and chloride of sodium, the action of the bitter salts is modified.

USES.—The bitter waters are useful in habits of constipation with sluggish portal circulation, in hæmorrhoidal tendencies, in congestion and enlargement of the liver and spleen, in some forms of dyspepsia, in gall-stones and allied affections, in gouty conditions, lithiasis, and diabetes; and, *ceteris paribus*, have in stout and in so-called phlegoric persons the preference over the muriated saline waters.

ENUMERATION AND SELECTION.—Where prolonged courses are required, the weaker sulphated waters, and especially the alkaline sulphated waters, and the muriated-sulphated waters, are to be preferred; while the stronger bitter waters are more frequently selected for occasional purging doses. (1) The principal *simple sulphated* or *bitter waters* are: Pullna, Saidschutz, Sedlitz, Birmensdorf, Åsculap, Apenta, Ivanda, Hunyadi János and other springs near Ofen, Rubinat, Las Caldas de Cuntio, Aranjuez, Friedrichshall, and Mergentheim, the two latter being also rich in chlorides. (2) The principal *alkaline sulphated waters* are Carlsbad, Marienbad, Tarasp-Schuls, Franzensbad, Elster, and Bertrich. The constitution and the action of the waters of Carlsbad and Bertrich are modified by their thermal nature. *North America* possesses some milder springs of this class: Estrill Springs, Crab Orchard Springs, Bedford Springs, Midland Well. (3) The best known muriated-sulphated waters are Brides-les-Bains, Leamington, Cheltenham, and St. Gervais.

V. Iron or Chalybeate Waters.—COMPOSITION.—Iron is contained in the majority of mineral

waters; but we regard as iron waters only those where the quantity of iron is, in proportion to the other constituents, so far predominant as to give a special therapeutic character to the springs.

ACTION.—The formation of blood-corpuscles, the contractility of the blood-vessels, the oxidation and the production of heat, and the general nutrition of tissues seem to be favoured by the use of iron waters. A small quantity only of iron seems to be absorbed by the stomach; none through the skin. The action of chalybeate baths seems to be due to the influence of the water and carbonic acid only.

USES.—The conditions most benefited by chalybeate waters are the various forms of anæmia, or poverty of blood and particularly of red corpuscles, especially when caused by actual loss of blood, supuration, or previous acute or chronic disease. The liver and digestive organs, however, must be in healthy working order, while in cases of anæmia accompanied by congestion of the liver and spleen, chalybeates alone are rarely useful, but must be preceded or modified in their action by the use of saline waters or other aperients; and this is often the case not only in anæmia of Indian and malarial cachexia, but also in chlorosis. Neuralgia, sterility, impotency, and general debility are often benefited through improvement of the general health. Those iron-waters are most useful which contain the iron in the form of the bicarbonate of the protoxide, kept in solution by free carbonic acid.

ENUMERATION AND SELECTION.—Iron springs are (1) *comparatively pure*, that is, containing only a few grains of other substances in sixteen ounces of water: Schwalbach, Spa, Brückenaue, Schandau, Lieberda, Flinsberg, Freienwalde, Recoaro, Königswarth, Liebenstein, Altwasser, Tunbridge Wells, and one of the springs at Harrogate; (2) *compound iron-springs*, that is, which contain, in addition to iron and carbonic acid, a moderate quantity of other salts, especially the carbonates of sodium, calcium, and magnesium, the sulphates of sodium, magnesium, and calcium, and common salt: Aratapak, Orezza, Pyrmont, Driburg, Rippoldsau, Griesbach, Antogast, Petersthal, Elster, Bocklet, St. Moritz, Reinerz, Godesberg, Cudowa, Imnau, Bussang, Ceresole Reale, and Santa Catarina. In North America: Bailey Springs, Stafford Springs, Green-castle Springs, Estill Springs, Schooley's Mountain Springs, Montvale Springs, Rawley Springs. Somewhat different in their action and less easily assimilated are the springs containing *sulphate of iron*. Representatives of this class are, in America—the Oak Orchard Acid Springs, the Bath Alum Springs, Stribling Springs, Bedford Alum Springs, and Variety Springs. The best known in Europe are: Flitwick in Bedfordshire, Muskau in Silesia, Parad in Hungaria, Alexisbad, and Ratzes. Rich in sulphate of iron in combination with *arsenic* are the waters of Roncegno and of Levico in the Austrian Tyrol, which will be mentioned among arsenical waters; they are very powerful, and can only be taken in small doses.

VI. Arsenical Waters.—Arsenic occurs in sufficiently appreciable quantities in some mineral waters to produce an alternative action in cases of anæmia, and various cachectic conditions, especially malarial cachexia, and in some chronic skin-diseases. We have mentioned already in the preceding group (iron-waters) two of the strongest arsenical waters, viz.: Levico and Roncegno in the Austrian Tyrol. Weaker springs of arsenic in combination with iron

are: Ceresole Reale in Upper Italy, Val Sinestra near Tarasp; Vic-sur-Cère, Sylvanès and Bussang in France; Cudowa and Linda-Pausa in Germany; Grèbernük (Grebernicza) in Bosnia. Combined with alkaline and muriated ingredients we have already mentioned La Bourboule and Mont Dore in France, and we may add Court-Saint-Etienne in Belgium. Arsenic is also contained in some springs of Royat, Saint-Nectaire, Saint-Honoré, Vichy, Vals, Uriage, Lamalou, Bath, Baden-Baden, Plombières, Porretta, &c.

VII. Sulphur-Waters.—**COMPOSITION.**—Among sulphur-waters we class those springs which contain either sulphuret of hydrogen, or the sulphuret of sodium, calcium, potassium, or magnesium in an appreciable and constant proportion. They are partly thermal, partly cold; and some of them, especially Aix-la-Chapelle, Uriage, the Columbia Springs in New York, and the Louisville Artesian Well in Kentucky, U.S.A., contain a considerable proportion of common salt and other solids, which are to be taken into consideration in the appreciation of their effects.

ACTION.—It is difficult to describe the physiological effects of the sulphur-waters, so far as they depend on such minute quantities of sulphur as are contained in them. Sulphur-water baths seem to act in the same manner as simple baths. If the waters are taken internally, some sulphuretted hydrogen is probably absorbed, entering the circulation through the portal vein. The pure sulphur-waters exercise a constipating rather than an aperient effect. The feces become mostly blackened from sulphuret of iron. The protracted use of these waters is apt to lead to a certain degree of anæmia, possibly from the action of the sulphur on the iron of the blood-corpuscles.

USES.—Sulphur-waters are mostly used in combined bathing and drinking courses, as also by inhalation; in cases of metallic poisoning; in congestion of the liver; piles; bronchial, laryngeal, and pharyngeal catarrh; in numerous cutaneous affections, especially the herpetic dyscrasia of the French; in rheumatism and gout; and in constitutional syphilis.

ENUMERATION AND SELECTION.—The best known *thermal sulphur-waters* are: Eaux Bonnes, Eaux Chaudes, Cauterets, Saint Sauveur, Barèges, Bagnères de Luchon, Ax, Escaldes, Le Vernet, Amélie-les-Bains, Uriage, Allevard, Aix-les-Bains, Aix-la-Chapelle, Baden in Austria, Baden in Switzerland, Lavey, and Schinznach in Switzerland; Acqui in Northern Italy; Battaglia and Abano in the Euganean mountains; Panticosa in Spain; Mehadia, and other springs in Hungary; and Helouan or Helwan, near Cairo. *Cold sulphur-springs* are: Eilsen, Nenndorf, Langenbrücken, Weilbach, Meinberg, Reutlingen, Enghien, Challes, Stachelberg, Heustrich, Gurnigel, some Harrogate springs, Llandrindod and Builth in Wales, Moffat and Strathpeffer in Scotland, and Lisdoonvarna in Ireland. There are also several important sulphur-springs in the United States of America.

VIII. Earthy and Calcareous Waters.—**COMPOSITION.**—As *earthy and calcareous waters* we designate those springs in which the earthy substances, especially carbonate and sulphate of calcium and carbonate of magnesium, form the prominent constituents.

ACTION.—In the shape of baths, the earthy waters act almost in the same way as ordinary

water baths. Internally taken, the carbonate of calcium exercises an antacid and a soothing effect on the mucous membrane of the stomach and intestines, and together with the sulphate of calcium is slightly astringent and constipating. If lime is absorbed, it may assist in the formation of bone, and may exercise also a soothing effect on other mucous membranes.

USES.—These waters, according to their composition, are useful in digestive troubles with tendency to acidity, diarrhoea, and undue irritability of the mucous membrane. They are employed also in osteomalacia, rachitis, and tuberculosis; and, further, in some skin-diseases, especially in eczema, and psoriasis, where, however, the long continuation of the warm bath, that is, the soaking of the skin, is of more importance than the nature of the solid constituents contained in the water. Some of these waters possess a great reputation in chronic catarrh of the bladder, and in tendency to gravel and stone; but probably the large quantity of water consumed, as, for instance, at Contrexéville, and the consequent dilution of the urine and the washing out of renal tubules, are here to be regarded as the principal causes of the useful effect. Some of these waters, especially Contrexéville, are also often employed in uric-acid diathesis, and in the glycosuria of fat and of gouty people, and not less so in tendency to biliary concretions. In all these cases the beneficial effect is principally due to the washing out of the tissues or small ducts by the drinking of large quantities of water. The best known earthy or calcareous waters are: Wildungen, Lippspringe, Inselbad, Weissenburg, Contrexéville, Vittel, Bagnères de Bigorre, St. Arnaud, and Cransac; and among the table waters: Couzan, St. Galmier, and the Taunus water. In North America the best known earthy or calcic springs are: Butterworth Springs, Eaton Rapid Wells, and Leslie Well in Michigan; the Gettysburg Spring in Pennsylvania; the Sweet Springs in West Virginia; and the Alleghany Springs in Virginia.

Many of the waters mentioned in other classes might also be mentioned here, such as Bormio, Leuk, Bath, and Lucca, named under the simple thermal waters; and Baden in Austria, Baden in Switzerland, Schinznach, Battaglia, Abano, and others enumerated under the sulphur-waters.

On prescribing Mineral Waters and Baths.—In every case we must first settle the question whether the treatment by mineral waters and baths offers advantages over ordinary treatment. If the question is answered in the affirmative, we have to consider not only the nature of the disease, but quite as much the nature of the individual to whom it occurs; the amount of vital forces in general; the power of reaction; the state of the different organs; and whether they are healthy and vigorous and can assist in relieving the diseased part of the organism, or whether they are feeble or crippled and unable to respond to any unusual demand made on them. Thus we shall be enabled to decide whether stronger therapeutic influences can be employed; whether longer and rougher journeys are permitted, and colder climates and seasons; or whether delicate treatment is essential, comprising the simple thermal baths, summer temperature, mountain climates of moderate elevation, sunny aspects, and easy journeys. The baths and waters are not to be selected according to the chemical constitution of their springs alone, but the means

and appliances in use, and the accustomed methods of treatment at certain places, the qualities of the local physician, the accommodation, the food, the cooking, and the social conditions, the facility of reaching a place, the climate and other elements of 'change,' are each and every one to be taken into consideration. It must be evident already from these remarks that the same morbid affection can occasionally be treated with advantage by different classes of mineral waters and at different spas, and that apparently widely different diseases may be benefited by the same spa; not only because many mineral waters are composed of different active elements, but also because the internal and external administration of the same water may be so much varied as to produce a great variety of effects. In many instances the disease itself cannot be directly attacked, but our efforts must be directed towards improving the general constitution, and through this influencing the diseased portion of the organism.

In most cases the local physician is as important as the nature of the waters, and in some even more so.

We cannot do more here than give some hints regarding the groups of diseased conditions in which mineral waters may be prescribed.

1. *Anæmia.*—In cases of anæmia it is essential to consider whether the condition is caused, first, by direct loss of blood and its component parts; secondly, indirectly by acute or chronic disease, sleeplessness, neuralgia, and inability to take up food; thirdly, by congestion of the pelvic organs, with loss of blood and albuminous juices; or, lastly, by diseases of the lymphatic vessels and glands, or visceral affections resulting from malaria or hot climates or from habitual constipation. The more the first cause preponderates, the more we may expect from the direct use of iron; and we have then to consider whether treatment at home with pharmaceutical preparations, or iron-waters with or without change of climate, with or without baths, are to be preferred. In the indirect forms of anæmia the mildest thermal treatment, with mountain climates of moderate elevation, or the latter alone, with or without suitable medicinal treatment, are often the only beneficial courses in delicate constitutions; while in others somewhat less feeble according to individual conditions, common-salt waters and baths with or without iron, or the gaseous tepid salt-baths of Nauheim and Rehme, or the much stronger influences of sea-air and of sea-baths, are useful. In the third group the common-salt waters, with a certain amount of iron, and occasionally the sulphated saline waters, must generally precede every other attempt at strengthening; for the acceleration of the portal circulation, the regular emptying of the different branches of the portal vein, and the increased tissue-change are essential to the improvement of the nutrition and sanguification; and only after such a preliminary course the purer iron waters and the higher alpine air are likely to become useful. In the treatment of some forms of chronic tuberculosis muriated saline waters with sea-air and sea-baths are of considerable value.

2. *Sluggish Portal Circulation.*—A sluggish condition of the portal system forms a frequent complication, not only of anæmia, but of a great many ailments of the different systems of the body; and is often only a part of a general want of tone in the organic muscular fibre, especially of the right ventricle and of the whole venous system. It is difficult

to find a name for these, by no means rare, constitutional defects, which form the main characteristics of what the old German physicians called 'abdominal plethora.' If we only know what we mean by the terms, we may call these conditions *portal venosity* and *general venosity* according to the extent of the defect. They form the principal complications and in many cases the main cause of the most varied digestive troubles, as acidity, sickness, flatulency, constipation, and intestinal catarrh. They are also at the root of congestion of the hæmorrhoidal vessels and piles, of varicosity of the legs, of congestion of the womb and ovaries and menstrual anomalies, of congestion of the liver and imperfect secretion of bile, and of chronic bronchial catarrh, with dilatation and imperfect contraction of the right ventricle. Gravel and gout are likewise often associated with sluggish portal circulation. In the treatment of these very numerous complaints, widely different though they appear to be, we have therefore always to ask how far they are complicated by portal venosity, and how far diet, regimen, pharmaceutical and balneotherapeutic treatment directed against this venosity may relieve the special case before us. If this portal venosity occur in *lean and delicate* persons, the common-salt waters are often useful, as Kissingen, Homburg, Soden, Bourbonne-les-Bains, Salsomaggiore, the Saratoga Springs, Michigan Congress Spring, Spring Lake Well in North America, &c., which increase the tissue-change without impairing the nutrition, internally and in the form of baths, or the simple thermal baths in sub-alpine situations, assisted by the internal use of salt waters. If the individual be *stout* and inclined to costiveness, the sulphated saline waters with soda and common salt, such as Carlsbad, Marienbad, Franzensbad, Elster, Brides-les-Bains, Luhatschowitz, and Tarasp, are the most effective; while again in others of this class the simple alkaline waters, such as Vichy, the Congress Spring, &c., are preferable. In all these cases, however, the treatment by waters and baths ought to be assisted by regulation of diet and exercise.

3. *Gravel*.—Gravel, especially uric-acid gravel, is usually complicated with portal venosity, and is to be treated accordingly. As a symptomatic treatment, the alkaline mineral waters have a more lasting effect than the administration of alkaline pharmaceutical preparations; but more effective are alkaline waters containing sulphates, and especially the less concentrated and hot springs of Carlsbad. Most useful of all, especially for home treatment, are the waters of Luhatschowitz, with their peculiar combination of carbonates and chlorides. The earthy waters of Contrexéville and Wildbad have a historic reputation, and owe this in part to the large doses which can mostly be prescribed.

4. *Gout*.—Gout is likewise often complicated with and aggravated by a defective portal circulation, and we must always endeavour to facilitate the removal of the products of the retrogressive tissue-change; but gout occurs in the most widely different constitutions. If gout and its allied forms be met with in so-called strong constitutions, with a good primary digestion, ability to sustain a long morning fast, accompanied perhaps by a tendency to stoutness, and an acid urine of tolerably high specific gravity, becoming iridescent with nitric acid, the alkaline sulphated waters of Carlsbad, and sometimes those of Marienbad, Franzensbad, Elster,

Tarasp, and the muriated sulphated waters of Brides-les-Bains, are most useful, though they cannot altogether remove the gouty disposition. If the time be short, and a long rest after the course not permitted, the simple alkaline waters of Vichy may be selected, and in more delicate constitutions the muriated alkaline waters of Royat, Ems, or Baden-Baden. In lean and weak gouty patients, the common-salt waters of Homburg, Kissingen, and Leamington, the arsenical salt waters of La Bourboule, the waters of Wiesbaden, the muriated sulphur-waters of Aix-la-Chapelle, Harrogate, or Llan-drindod, or, again, weak muriated alkaline waters like Baden-Baden, deserve a trial. In many delicate persons the simple thermal waters of Buxton, Teplitz, Schlagenbad, Wildbad, Ragatz, Gastein, and Bath, and the sulphur-waters of Aix-les-Bains and Bagnères de Luchon, offer great advantages; but numerous cases may be regarded as quite intractable by baths and waters, and in these diet and climate and regulation of exercise are the only means of management, assisted by various anti-gouty medicines.

5. *Chronic rheumatism*.—In chronic rheumatism, associated with exudation round the joints, the hot thermal treatment is the most useful, either at the hotter simple thermal spas, as Bath, Teplitz, Acqui, the Euganean baths, the hot springs of North America, or the natural vapour-baths of the cave of Monsummano; at the weaker hot salt waters of Royat, Wiesbaden, and Baden-Baden; or at the thermal sulphur-waters, such as Aix-la-Chapelle, Uriage, Aix-les-Bains, Barèges, Bagnères de Luchon, and Eaux Chaudes. For more delicate cases, especially in those combined with weakness of the muscles of the heart, with or without valvular disease, we have the gaseous thermal salt waters of Rehme and Nauheim. In the muscular varieties, with stiffness, the hotter waters are specially indicated, assisted by douches and shampooing. In many instances, however, the cause of constantly recurring rheumatism is weakness of the skin, and here the tonic forms of the cold water-cure and sea-baths promise more permanent good than hot baths.

We might be expected to give a few hints on diet during mineral-water courses, but no general rules can be laid down. Every individual requires rules for his own case; and rules which may be necessary during the use of muriated saline or sulphated saline waters, are not necessary in other courses—for instance, of simple thermal or of iron waters.

The resident physician ought to guide every invalid, according to his or her individual condition, as well with regard to diet as to the internal or external use of waters, and with regard to exercise and other hygienic and therapeutic aids. The result of a course of waters often depends entirely on this guidance. It is important, therefore, to supply the bath-physician with a statement as to the ailments and the constitution of the invalid.

LENGTH OF TREATMENT.—It is a general belief that three or four weeks is the term for a course of waters or baths; but it is impossible to fix a definite time. As courses of iodide of potassium, of iron, of quinine, or of mercury must be of different duration in different individuals, exactly so we find it with mineral waters; and as two or three courses of a remedy may have to be taken in the same year, so it is often desirable to give two or three courses of Vichy, of Carlsbad, or of other Spa waters, in one year, though not all of them need be taken at the

spring. In many cases preparatory courses are advisable—climatic, medicinal, and balneotherapeutic; and in as large a number secondary courses. Most invalids ought not to return immediately after a course of baths to their usual abodes and accustomed ways of living, but ought to have what the Germans call a 'Nachkur,' at climatic health resorts selected according to the condition of each individual. In many instances, moreover, it is imperatively necessary to abstain from work, and to keep to a simple diet for about a month or more after the course of waters, and this is especially the case with the more powerful waters like Carlsbad and Marienbad. It must be clearly understood that, in order to make a course of waters permanently beneficial, it is necessary to avoid the injurious influences which have led to the morbid condition, not only for one or two months but for the whole life.

SEASON.—As to the period of the year, there is no time when the different waters might not be drunk, if it were necessary. Most spas are open only from May till October, some longer; some only from June till September; but some few localities are partially open also during the winter, especially Aix-la-Chapelle, Aix-les-Bains, Dax, Amélie-les-Bains, Bath, Acqui, Salsomaggiore, Baden-Baden, and Wiesbaden. Many waters can be taken at home, and at any time of the year; but when taken at home the elements of change are wanting, and the strict adherence to regimen and diet is often difficult. During the summer months the demands on the human body are diminished by the external warmth and the greater equability of the meteorological influences; nature is more exhilarating, and invites to outdoor life and exercise, without much risk of chills and their consequences. Delicate persons, therefore, ought to select the summer months for courses at the spas. The later parts of the spring and the autumn, however, offer advantages to the more robust, who at those times find the baths and the hotels less crowded, and who can then receive more attention from the bath-physician. And, besides, those who are unable to bear heat have in the earlier and later parts of the season the benefit of cooler air, which is to the average visitor a real advantage at some of the hotter localities, like Aix-les-Bains, Acqui, Salsomaggiore, Aix-la-Chapelle, Ems, Kreuznach, Wiesbaden, Soden, Baden-Baden, and Ragatz. HERMANN WEBER.

MINER'S LUNG.—See PNEUMOCONIOSES.

MINER'S NYSTAGMUS.—See OCCUPATION-DISEASES; and NYSTAGMUS.

MIRROR-MAKER'S PALSY.—See OCCUPATION-DISEASES.

MIRYACHIT (Russ. *miriatchitje*, to play the fool).—See ECSTASY.

MISCARRIAGE.—SYNON.: Abortion; Fr. *Avortement*; *Fausse Couche*; Ger. *Fehlgeburt*.

DEFINITION.—Miscarriage is the interruption of gestation before the fœtus has become viable.

FREQUENCY.—The relative frequency of miscarriages, of premature labours (between the seventh and ninth months), and of full-time births, cannot be very closely estimated. Early abortions are often unnoticed or forgotten. The statement of Whitehead is very striking, that of sixty-four women

who had lived in wedlock till the menopause, there were only eight who had not at some time had a miscarriage. His statistics show that the period at which abortions most frequently occur is about the third month.

ÆTIOLOGY.—The causes of abortion may be found either: (1) on the part of the ovum or fœtus; or (2) on the part of the mother.

1. *Fœtal.*—The causes of miscarriage on the part of the ovum are: (a) all the diseases of the fœtus itself which compromise its life, such as acute fevers and chronic diseases—chiefly of syphilitic origin; and (b) many of the morbid changes in the fœtal appendages. Of the latter the most noteworthy are, first, diseases of the chorion, the most familiar of which is the hydatidiform degeneration; secondly, abnormal conditions of the umbilical cord, such as excessive torsion with constriction of the vessels, convolutions of it simultaneously round the neck and lower extremities, and the formation of tight knots upon it; and thirdly, abnormal relations and morbid conditions of the placenta. Where the placental area, for example, is of too limited extent, the ovum easily becomes detached from the uterus; where it is too large, extravasations of blood easily take place in the lobules. When the placenta is planted low down in the cavity of the uterus, it is liable to partial detachments; and thus in a great many cases abortion takes place at an early stage in patients who would have been subject to the greater dangers of unavoidable hæmorrhage had the pregnancy gone on towards the usual term. Again, the morbid processes which occur in the placenta, inflammatory, degenerative, or apoplectic, whether due to a syphilitic taint, or to other causes, lead to death of the embryo or fœtus, and thus in many instances to the early casting of the ovum. It is worth while to note that death of the embryo, and morbid changes in its appendages, do not necessarily at once cause abortion. Three or four weeks usually elapse after the death of the fœtus ere its expulsion is effected; the decidual membrane having in the interval undergone retrogressive changes. It is only when such an extravasation of blood takes place as leads to sudden distension of the uterus, or when the membranes burst and such escape of liquor amnii occurs as leads to its sudden collapse, that the organ is stimulated to the immediate evacuation of its contents. Hence, while the ultimate cause of abortion is often enough traceable to the ovum, the immediate occasion is more frequently due to some maternal condition.

2. *Maternal.*—The causes of miscarriage on the part of the mother are either (a) *general*; or (b) *local*. (a) Among the *general* or constitutional conditions that favour the occurrence of abortion we note, first, all the causes that lead to depression of a woman's health. Abortions are frequent, for instance, in times of famine; among women who yield themselves to excesses; in anæmic women; and in those tainted with the syphilitic poison. Often enough, especially in the last class, the cause of the abortion can be traced to some morbid change in the maternal portion of the placenta; but sometimes it seems to be due simply to the impure or impoverished condition of the patient's blood. Secondly, fevers, such as the zymotic fevers, and acute inflammations, more particularly of important viscera, such as pneumonia, occurring in gravid women, very frequently become complicated by abortion. Thirdly, shock may bring on mis-

carriage, whether operating simply through the nervous system, of which we meet occasional examples, or, as is more frequently the case, by producing a more direct physical impression upon the uterus, as in cases where the patient leaps or steps suddenly down from a height, lifts a weight, stretches her arms above her head, or is exposed to any sudden jar or more protracted jolting. Though many cases of abortion are attributed to such a cause, it is always to be borne in mind that in some of these, at least, the supposed cause would not have led to the disaster unless there had already existed a predisposition in some morbid condition of the uterus or its contents.

Among (*b*) the *local* causes we find, first, and most frequently, diseased conditions of the deciduæ. Commonly in these cases the patient had previously been the subject of chronic endometritis; though occasionally cases are met with where there have been no marked symptoms previously, and the degenerative process may affect either the vera or reflexa or serotina, separately or simultaneously. Second in frequency under this head we have the abortions due to displacements of the uterus, these being commonly either descents or retroversions. Thirdly, neoplasms of the uterus, such as cancers or fibroid tumours, sometimes permit the occurrence of conception, but prevent gestation running to its natural term. Fourthly, the presence of tumours in the neighbouring organs, or inflammatory adhesions among them, may prevent the uterus from attaining its full growth, and compel it to early evacuation of its contents.

SYMPTOMS AND DIAGNOSIS.—In dealing with a case of suspected miscarriage, we have to determine first that the patient is pregnant. This we do by a careful inquiry into the patient's history, and a complete physical examination. Supposing that, by the usual investigation into the signs and symptoms of pregnancy, we are satisfied that gestation had begun, we have next to ascertain whether miscarriage is only threatening to come on, has fairly set in, or has already been completed.

The symptom that, in the great run of cases, first attracts attention, which usually goes on till the process is completed, and which continues for some hours or days subsequently, is hæmorrhage. The amount of blood lost varies indefinitely; and so does the manner of its escape. In certain cases the onset of pelvic pains, with the regular intermissions that betray their origin in the muscular contractions of the uterine walls, alarms the patient and attracts her attention before any escape of blood has taken place. These cases are exceptional. Usually the hæmorrhage precedes—and it may be for days or weeks—the expulsive action of the uterus. The cases, however, are rare—unless they be instances of very early abortion—where the process is completed without the accession of appreciably painful contractions. Occasionally there occur discharges of liquor amnii or other watery fluid, or of fragments of the degenerated membranes, or of the disintegrated foetus.

These symptoms call for physical exploration of the uterus. If we find the uterus gravid, with the os undilated and the cervical canal above it unexpanded, the hæmorrhage being slight and the pains controllable, we regard and treat the case as one simply of threatened abortion. But if the pains are persistent, if the os uteri opens to admit the finger, or the canal of the cervix above it is becoming ex-

panded; still more, if the uterine contents are being pressed down within reach of the exploring finger, we have to do with an actual abortion which it is useless to seek to avert. The treatment of actual abortion is often enough called for, even with quiescent uterus and closed canals, when the hæmorrhage is profuse.

In trying to determine whether the miscarriage is completed, we have first to examine the mole or mass that has been expelled. This consists sometimes of the ovum alone; of the ovum and decidua reflexa; or of the ovum with all the uterine deciduæ. Where the uterine contents escape in broken-down fragments, and cannot be satisfactorily pieced together, it becomes necessary to examine the uterus, and even to explore the interior of that organ with the finger; and in these and other cases where the diagnosis is doubtful, it may be requisite sometimes to dilate the cervix with a carbolised sponge or tupelo tent, or with Hegar's dilators, in order to get full access to the uterine cavity.

TREATMENT.—The treatment of miscarriage varies according as we have to do with a case of (1) threatening abortion; or (2) abortion in actual progress; or (3) incomplete abortion.

1. *Treatment of threatening abortion.*—The treatment in a case where abortion is merely threatening is largely expectant. The patient is put to bed, and kept at rest in the recumbent position. All exercise or excitement, physical or psychical, must be forbidden. A light, non-stimulating diet, with fluids for the most part cold, is to be enjoined; and any tendency either to constipation or to diarrhœa is to be combated. Where the hæmorrhage is continuous and the uterus atonic or flaccid, small doses of ergot—twenty drops, every six or eight hours, of the liquid extract—are useful. Diluted sulphuric acid or gallic acid, either alone or in combination with digitalis, may be administered. Where there are occasional pains accompanying the discharge, the best effects are obtained from the administration of opiates, which may be prescribed in the form of the acetate of lead and opium pill. Where the pains constitute the more urgent symptom, and the hæmorrhage is less, it may be well to check the uterine action at once by the use of an anæsthetic followed by opiates, or the administration of a dose of chloral hydrate; and the astringent may then be dispensed with. The opiates in such cases are best administered hypodermically or *per rectum*. In many cases the liquid extract of *Viburnum prunifolium* in doses of ʒss–ʒi has proved useful as a sedative to the uterus in threatened abortion.

2. *Treatment of actual abortion.*—Where the stage of expectancy is clearly over, and the patency of the os internum, the persistence of the pains, or the profusion of the hæmorrhage, calls for active interference, there are two main indications to be fulfilled—namely, to restrain the hæmorrhage; and to ensure the complete evacuation of the uterus.

To restrain the hæmorrhage, we compel the uterus to more energetic contraction, first, by the administration of large repeated doses of ergot. A drachm of the liquid extract may be given every three or four hours; but the effect of the drug can be most speedily and safely ensured by the hypodermic injection of ergotin—according to some such formula as this: ℞ Ergotini ʒij ; $\text{chloral hydratis ʒss}$; $\text{aquæ destillatæ ʒvj}$ —16 drops to be injected into the gluteal muscle (*see HYPODERMIC MEDICATION*). The dialysed solution of ergotin is said

to produce less irritation. Secondly, the genital canal must be plugged. Where we have no other means at command of checking the discharge, a carefully applied vaginal tampon may be trusted; or the vaginal plug may be used where the hæmorrhage is going on but there is still some hope that the abortion may be arrested. Where the indication is more urgent, the introduction of a sponge tent into the cervical canal is very much more satisfactory, and in every way more efficacious. It arrests the hæmorrhage immediately and inevitably; it excites the uterus to more energetic action; and it at the same time expands the cervical canal in all its length. Where the indication is present for still more rapid dilatation of the cervix, this may be effected by means of dilators, such as Hegar's.

The complete evacuation of the uterus may take place by the unaided efforts of its muscular walls. On visiting a patient in the morning, who had a sponge-tent passed into the cervix uteri and a hypodermic injection of ergotin over night, we may find sponge and ovum and all expelled. Where the ovum is still *in utero*, if it be loose and the cervix dilated, compression of the uterus from above the pubes may suffice to make it expel its contents. Usually, however, it becomes necessary to get at the interior of the uterine cavity with a finger or fingers passed through the vaginal canal. In most cases it greatly facilitates the operation to anaesthetise the patient, and in some cases the previous administration of chloroform is absolutely necessary. To render the uterus accessible to the exploring fingers, it must either be pushed down from above or dragged down from below. The patient lying unconscious on her back, the fundus uteri may be depressed by the left hand pushed firmly and steadily down through the pelvic brim. The depression may be effected by an assistant, but never so satisfactorily as by the operator himself. No less than two fingers of the right hand should be used for the internal manipulation; the middle finger being folded in the fornix vaginae, while the index passes through the os to the fundus uteri, and sweeps round the entire ovum, detaching it at any adherent points. Sometimes the middle finger more conveniently enters the uterine cavity; and in most cases of miscarriage in the fourth month, the whole hand, except the thumb, may require to be passed into the vagina, and two or more fingers into the uterine cavity. Even where the vaginal orifice is not at first very wide, if the hand be carefully warmed and soaped, and the interstices of the fingers filled up on their palmar aspect with a quantity of half-melted soap, sufficient dilatation is speedily effected. Occasionally the smaller left hand may be employed for internal manipulation, while the stronger right is engaged in making the external pressure on the fundus uteri. Access to the interior of the uterus may in most cases be gained more easily by dragging the uterus down from below. One or other of the lips of the uterus—usually the anterior—is seized with a vulsellum, double- or triple-pronged, and slightly curved. One of the blades grasps the vaginal aspect of the front lip of the cervix as high up as the roof of the vagina, the other at a corresponding level within the cervical canal. The uterus is capable of being drawn far down without any injury to its ligaments, or any laceration by the bite of the vulsellum. It may be pulled down with the right hand and kept fixed by it, while the fingers of the left pass into the cavity, and explore and evacuate it. Or the vulsellum may

be held in the left hand, or given to an assistant, to keep the uterus depressed, while the more familiar right-hand fingers do the intra-uterine work. The finger or fingers that have detached the ovum commonly succeed in extracting it, aided sometimes by pressure with the other hand from without. If not, there is no objection to laying hold of the loosened body with a pair of long dressing-forceps, or a Lyon's or polypus-forceps, and so withdrawing it; but no such instrument, even though it bear the name of abortion-forceps, ought to be trusted to for the detachment of a retained ovum or fragment of adherent placenta. The separation should always be effected by the direct action of the living finger.

3. *Treatment of incomplete abortion.*—In mismanaged cases the uterus is left imperfectly evacuated, and the patient continues to suffer from menorrhagia and metrorrhagia—it may be for months subsequently. In such circumstances the use of the curette is invaluable. Sometimes a degree of dilatation with tupelo tent or otherwise is required, but usually the cervical canal easily permits of the passage of a curette, with which the surfaces of the uterus can be scraped and the entire cavity of the organ can be cleared out. The curetting should be accompanied or immediately followed by the washing out of the cavity with a stream of hot water, of the temperature of 110° to 120° F., containing 1 in 5,000 of corrosive sublimate, or some such antiseptic, with the view at once of checking further hæmorrhage and disinfecting the uterine cavity. In the cases where the ovum has undergone hydatidiform degeneration, the further progress of the patient should be watched with special care. In a number of women who have been the subjects of such a 'molar pregnancy,' malignant degeneration has taken place in the interior of the uterus, apparently from the retention of some of the chorionic elements. This *deciduoma malignum* may give rise to fatal hæmorrhages or to metastatic processes in the lungs and other organs; and the patient's only chance of cure is obtained by an early hysterectomy. See PLACENTA, Diseases of.

After-treatment.—The uterus having been completely emptied, the patient should be kept at absolute rest in bed, and subjected to the same treatment as an ordinary puerperal female.

ALEXANDER RUSSELL SIMPSON.

MITOSIS (μῖτος, a thread).—SYNON.: Karyokinesis; Indirect Cell-division.—The process by which living animal-cells usually divide and multiply. See CELL.

MITRAL VALVE AND ORIFICE, Diseases of.—See HEART, VALVES AND ORIFICES OF, Diseases of.

MOFFAT, in Scotland.—Sulphur and also chalybeate waters. See MINERAL WATERS.

MOGIGRAPHIA (μόγισ, with difficulty; and γράφω, I write).—A synonym for writer's cramp. See WRITER'S CRAMP.

MOLE.—**MOLAR PREGNANCY.**—DEFINITION.—A degenerated mass formed in the uterus as a result of conception.

There are two varieties of mole: (1) the fleshy, carneous, or blood-mole; and (2) the vesicular or hydatidiform mole.

1. **The Carneous or Fleshy Mole.**—This is sometimes called a *blood-mole* or *apoplectic ovum*

owing to its hæmorrhagic origin. Some authorities think that a *carneous mole* is a later stage of a 'blood-mole,' altered either by organisation or by changes due to decidual endometritis. The carneous mole is due to hæmorrhage into the decidua vera or serotina. The blood may find its way between the decidua vera and reflexa, or between the chorion and amnion, or more rarely may invade the amniotic cavity itself. In any case the involved chorionic villi are separated from the maternal blood-supply, or rendered useless by compression, and if the hæmorrhage be extensive, or be in the neighbourhood of the insertion of the cord, the embryo perishes, and shrinks, and, if the amniotic cavity is encroached upon, may disappear altogether. The resulting mole is a thick-walled cyst, lined by amnion, which is thrown into irregular bosses by the extravasated blood. It is distinguished from extruded fibroids by its general appearance and by the presence of decidual cells and villi, and by the absence of muscle-fibres. From the membrane of membranous dysmenorrhœa, and from the decidua of ectopic gestation, it is distinguished by the presence of villi and by the great thickness of the cyst-wall. A mole of a similar type may form in the Fallopian tube in cases of tubal gestation. See PREGNANCY, Diseases of.

SYMPTOMS AND DIAGNOSIS.—After a few weeks of pregnancy the usual symptoms and physical signs of gestation gradually become less defined. If the uterus be examined bimanually at intervals during this period, it is found to have become suddenly enlarged as a result of the hæmorrhage, and then to have gradually lessened in size, remaining smaller and more incompressible than it would have been in normal pregnancy. The ovum may at once come away, or, as frequently happens, may remain *in utero* (*missed abortion*) till the fifth or sixth month; or even remain, a fact of medico-legal importance, till long after full-term, and cases are reported where menstruation has in the interval been normally re-established. More usually, however, there is a brownish and sometimes offensive discharge, with occasional bright red hæmorrhage, and the diagnosis then has to be made (1) from the hæmorrhage caused by decidual endometritis, when the uterus is usually normal in size and evidences of normal fetal growth are forthcoming, (2) from hydatidiform degeneration of the chorion to be presently considered, and (3) from deciduoma malignum (see PLACENTA, Diseases of; and PREGNANCY, Diseases of), or (4) from a normal pregnancy with such complications as epithelioma, or mucous polypus of the cervix. The differential diagnosis will be discussed under the next variety.

TREATMENT.—The treatment is to empty the uterus. While the mole remains in its cavity, the patient is exposed to the risks both of hæmorrhage and of septic infection. The administration of ergot may be sufficient to stimulate the uterus to expel its contents; but should this not occur, the aseptic use of the sound will often succeed. Should the uterus still remain inert, the cervix must be dilated rapidly, under anæsthesia by preference, and the contents removed by the finger or by ovum-forceps, aided by supra-pubic pressure; and the cavity washed out with some warm antiseptic injection, and lightly packed with iodoform-gauze for twenty-four hours.

2. The Vesicular or Hydatidiform Mole. The term 'hydatid mole' is misleading, as the condition has nothing to do with the *Tania echinococcus*.

ÆTIOLOGY.—The ætiology of these moles is still obscure. They are known to be secondary to fertilisation of the ovum, though a few authorities have endeavoured to prove that it may occur in the uterus of a virgin; it is the cause and not the result of the death of the embryo; it may occur in successive pregnancies in the same woman, and yet may in other cases be found in only one of twins; it may exist in cases where both the mother and child have been dropsical; it occurs with greater relative frequency in woman after thirty years of age; and though there may be no history of syphilis in the woman herself, it is not infrequently found in women whose husbands have had that disease.

MORBID ANATOMY.—The vesicular mole is the result of a myxomatous degeneration of certain of the cells forming the inner lining of the chorionic villi. These cells swell and gradually fill up the cavity of the villus, giving it the appearance of a vesicle, and causing obliteration by pressure of the contained loop of capillaries. This degeneration occurs at intervals along the length of a chorionic villus, giving it a moniliform appearance like a series of vesicles threaded on a fleshy stalk. The disease may be confined to those villi which usually atrophy during the second and third month, or may affect mainly or exclusively those engaged in forming the foetal portion of the placenta. The depth to which the vesicles penetrate into the uterine decidua or even into the muscular tissue depends upon the depth to which the villi had already penetrated. The embryo, in its compressed or obliterated amniotic cavity, is rarely discovered, unless the affected villi cover only a small area.

SYMPTOMS.—After four or five weeks of apparently normal pregnancy the patient notices a little hæmorrhage at irregular intervals, often induced by stooping, lifting, coitus, or straining at stool. The hæmorrhage is, as a rule, slight and watery. Occasionally, if villi are detached from the decidua, hæmorrhage may be profuse, and if vesicles protrude through a dilated cervix, the discharge may become brown and offensive. As a rule the constitutional (reflex) symptoms of pregnancy, such as morning sickness, are more marked and more persistent. The physical signs differ from those of normal pregnancy in that the size of the uterus increases at a greater rate owing to the vesicular growth, and that although intermittent contraction of the uterus and the uterine souffle are observed, the evidences of the presence of a foetus, such as abdominal and vaginal ballotement, foetal movements, and foetal heart-sounds, are absent, except in those rare cases when a second normal foetus is also present.

A vesicular mole may be expelled at any period of gestation, or it may remain and be expelled at an indefinite date after full term.

DIAGNOSIS.—The diagnosis has to be made from normal pregnancy by the hæmorrhage and by the physical signs as already stated. It has also to be distinguished from those cases of pregnancy accompanied by hæmorrhage due to decidual endometritis, or to such complication as mucous polypus or malignant disease of the cervix; as a rule, the fact that in these conditions the uterus is not larger than the size expected for the date serves to make the diagnosis. In case of fleshy mole the uterus is smaller than it would be if a normal pregnancy, and this at once distinguishes the case from that of a vesicular mole, when the uterus is much larger than

normal, reaching the level of the umbilicus at the fourth month. A certain diagnosis can, however, often only be made by discovering vesicles in the discharge, or by dilating the cervix and exploring the uterine cavity. This, for instance, is the only way to differentiate between vesicular mole and deciduoma malignum, and in this connection it must be remembered that cases have been recently reported of new-growths of a similar myxomatous type, apparently growing from tufts of myxomatous villi left in the uterine wall, with secondary growths in the labia majora and lungs.

TREATMENT.—As soon as the hæmorrhage and physical signs have led to a positive or tentative diagnosis, the uterus should be encouraged to expel its contents by drachm-doses of the liquid extract of ergot three times daily, and the patient should use, twice daily, an antiseptic vaginal douche.

If the mole still remain *in utero*, and hæmorrhage persists, the patient should be anæsthetised, the cervix dilated rapidly with graduated bougies, and the uterine contents carefully separated by the exploring finger combined by external manual pressure. This method of 'expressing' the uterine contents while the internal fingers gently separate and extract the vesicles as they come within reach is far the safest plan, for the uterus is so easily lacerated in these cases that the use of the curette is fraught with danger. The uterus should then be irrigated with iodised water and lightly packed with gauze, the patient kept in bed for ten days, and ergot given to encourage involution. Great care should be taken to detach all vesicles, otherwise secondary hæmorrhage or septicæmia are possible complications.

It is important to remember that twin pregnancies may occur in which vesicular degeneration affects the chorion of but one ovum; and that quite possibly the sound ovum may proceed to full development, as is said to have occurred at the birth of the anatomist Beclard.

AMAND ROUTH.

MOLES.—This term is applied to a circumscribed pigmented spot or tumour of the skin, of developmental origin, frequently raised above the surface, roughly papillomatous in structure, and often with an excessive development of hair. Occasionally moles develop in later life, but in such cases the lesion is probably due to some defect during development, which has escaped notice. Subsequent increase in size draws attention to the affected area. Areas of abnormal pigmentation with overgrowth originate from various causes in later life, and to such lesions the word 'mole' is also applied, but they pass, strictly speaking, into a different category. To these congenital defects the term *nævus* is also frequently applied.

Such a mole or *nævus* may consist simply of an excess of melanin deposited in the lowest layers of the stratum mucosum and in the limiting layer of the cutis, and such pigment-spots especially develop at any period of life. But usually the abnormalities of development are more marked. Thus irregularities in the growth of the cutis may produce irregularities of the surface, and lead to the formation of papillomatous excrescences (*nævus verrucosus*, *nævus papillomatosis*). More important are the apparently isolated inclusions of epithelium, which lie deep in the connective tissue. These epithelial inclusions are the areas which probably first show the signs of overgrowth and malignant change, not

infrequently arising in moles. Hairs are usually present on the surface, and may be fine and lanugo-like, or more usually numerous and firm in consistence (*nævus pilosus*). The changes in the connective tissue consist of increase in amount, abnormal development of blood-vessels (*angiomata*), and occasionally excessive formation of fat-cells. The amount of pigmentation is usually great, but it may be only slight, or even in some cases apparently absent (white moles).

The distribution and size of moles vary within wide limits. They may be quite small or occupy large areas of the surface; and occasionally the skin of a limb or of a large segment of the body may develop the *nævus* structure. A linear distribution along the course of a limb, or on the trunk, may sometimes be seen, suggesting the distribution of the cutaneous nerves, but are probably so arranged owing to defects in the development of the body-segments (*nævus unius lateris*).

Small moles give little trouble, but when large and disfiguring, or interfering mechanically by their presence, they should be removed. This should be done thoroughly by excision, and care should be taken to irritate the affected areas as little as possible. In the case of small moles destruction by electrolysis is possible, and the electrolysis of the hairs on a mole is occasionally sufficient to destroy the whole structure. Irritation and attempted destruction by caustic agents are inadvisable, especially in the case of adults. Growths originating in moles are usually melanotic, and often of great malignancy. It cannot be doubted that tampering with moles by means of irritating agents is likely to stimulate the tendency shown by these formations to become malignant.

JAMES GALLOWAY.

MOLLITIES OSSIUM (Lat.).—SYNON.: Osteomalacia; Malacosteon; Fr. *Ramollissement des Os*; Ger. *Knochenerweichung*.

DEFINITION.—A condition characterised by a progressive decalcification of the bones and replacement of the true bone-tissue by a soft very vascular gelatinous material, and leading to great deformity of the skeleton.

ÆTIOLOGY AND PATHOLOGY.—The disease is rare, and much more rare in Great Britain than on the Continent. It chiefly attacks the poor, and is ten times more frequent in women than in men. It is most often met with between the ages of 25 and 35, and pregnancy appears to exert a marked influence in causing the disease. Thus, among women, it is almost limited to multiparæ, and many of the patients have borne children in rapid succession. The symptoms are usually first observed during pregnancy, and the disease may be arrested after parturition and become again active on the recurrence of pregnancy. The removal of the uterine appendages is said to have had a beneficial effect upon the disease. The lime-salts of the affected bones are dissolved by lactic acid, and the disease has therefore been attributed to an excess of this acid in the blood. The occurrence of many cases in certain localities has led to the suggestion that there may be some climatic condition causing the disease, and that it may be endemic. The *vera causa* of this affection is at present unknown. It is not an inflammatory disease of the bones, nor is it a true atrophy such as occurs from disuse, or old age, or in mental and malignant disease, nor can it be described as a neoplasm. It

appears to have no connection with rickets, which is a change in growing bones, whereas mollities ossium attacks the fully-formed skeleton. It is a disease *sui generis*, commencing in the medulla of bone and gradually extending thence to the periphery. Whether it is due to the presence of excess of acid, such as lactic acid, in the medulla, or whether this is one of the effects of a more subtle nutritive change is not determined.

ANATOMICAL CHARACTERS.—The osseous lamellæ become decalcified progressively from the centre towards the periphery of the bone; for a short time after this the lacunæ and the contained corpuscles can still be made out, but gradually all trace of the bone is lost, its place being taken by a very vascular soft gelatinous tissue which has previously replaced the true marrow. Into this soft fibro-cellular tissue hæmorrhages often occur; in extreme cases it may be almost diffluent. The outer shell of bone is the last to be affected, and in advanced stages of the disease this may be perforated in several places. There is no change in the periosteum, nor any formation of new bone beneath it.

As the result of this process the bones become very soft and yield under the influence of gravity and muscular tension, so that the most extreme deformities result. The change first affects the bones of the pelvis and the lower part of the spine, and the disease is often first recognised during parturition. The deformity produced in the pelvis is great and peculiar; the sacral promontory falls forward under the weight of the body, the pubic symphysis is forced forwards like a beak, and each acetabulum is pressed in by the head of the femur. This 'beaked' or 'triphite' pelvis may be the cause of serious difficulty in parturition. Next to the pelvis and spine the ribs are the bones most often affected; numerous fractures and bendings may occur, and the most characteristic change is the formation of a deep vertical groove on each side due to the pressure of the arms; the ribs are often broken at the front and back of this groove. Multiple fractures as well as bendings occur in the long bones of the limbs; in the earlier stages the fractures may unite, but when the disease is advanced union does not occur. Osteomalacia attacks fully-developed bones, and does not begin in, or chiefly affect, the epiphyseal ends of long bones. Excess of lime-salts is present in the urine and fæces, the channels by which the absorbed salts are eliminated.

SYMPTOMS.—The onset of the disease is very insidious; the patients are usually thin, anæmic, and poorly nourished. Pain of a 'rheumatic' character is generally experienced, but as this is referred to the pelvis and lower parts of the spine, and usually accompanies pregnancy, it is often supposed by the patient and her friends to be connected with the enlarging uterus. It therefore happens that as a rule no special attention is paid to these pains, and only when parturition commences and an examination is made are the grave effects of the disease discovered in the narrowed distorted pelvis which gravely interferes with natural delivery. Similar rheumatic pains are felt in other affected bones, and the bones may appear to be slightly enlarged; they are tender to pressure. But here also it is only when the bones are found to be softened so that they yield to pressure in various directions, or can be bent by the surgeon, or undergo numerous spontaneous fractures, that the nature of the disease is recognised. The disease is

chronic; it is very rarely arrested altogether; sometimes the condition seems to improve for a time, more generally the patient slowly becomes more and more crippled, deformed, and helpless, and finally succumbs from some intercurrent disease, especially of the respiratory organs, whose function is gravely interfered with by the softening and fractures of the ribs and sternum. Many of the patients have lost their life during parturition from the severe procedures necessary to effect delivery.

DIAGNOSIS.—The age of the patients affected, the bones first attacked, and the extreme softening that occurs in them, at once distinguish this disease from rickets. Some authorities have described a form of mollities ossium occurring in adolescents, but it is very doubtful whether this differs essentially from late rickets. The extreme softening and bending of the bones, the early and special affection of the pelvis and spine, followed by the wide extent of the changes in the skeleton, and the age and sex of the patients, are the points which serve to distinguish mollities ossium from fatty atrophy and spontaneous fracture from malignant growths or so-called fragilitas ossium.

TREATMENT.—As soon as the disease is discovered, everything possible should be done to improve the general nutritive condition of the patient. She should be removed to a bracing locality—certainly removed far from any district where the disease is endemic—and supplied with a liberal nourishing diet. Careful gentle massage may usefully replace the exercise which has become impossible. If pregnant, abortion should be induced. Future pregnancy must be forbidden. Some good has been reported to follow oophorectomy. If delivery at term has to be effected through the abdominal wall, the uterine appendages should certainly be excised with or without the uterus. The administration of bone-marrow in tablets has been followed by benefit, and appears to be a hopeful method of treatment. A. PEARCE GOULD.

MOLLUSCUM CONTAGIOSUM.—**SYNON. :** Epithelioma contagiosum.—This disease is characterised by the growth of small contagious tumours from the epidermis.

DESCRIPTION.—The tumours vary in size from the smallest visible spot to that of a pea. Very rarely larger masses have been produced by the confluence of smaller ones. They project from the surface of the skin in a distinctly hemispherical form and have a firm elastic consistence. They are often semi-translucent and have a yellowish or 'mother-of-pearl' tint varying to some extent with the texture and pigmentation of the surface which they occupy. Early in the course of their growth they present a depression on the summit which, as the tumour increases in size, is seen to be the orifice of a passage leading to a cavity in the centre of the tumour occupied by epithelial debris, due to degeneration of the epithelial cells composing the tumour. From this orifice the degenerated cells escape or can be expressed as a greyish-white caseous material. Occasionally more than one channel may lead to the softened centre of the tumour. The tumours arise on any part of the skin, but are most frequently observed on parts exposed to contagion. Thus the face, especially in the case of children, is one of the commonest sites. In adults the genital organs and surrounding parts are not infrequently affected, and the breasts in the

case of nursing women may become inoculated. In rare cases the tumours may be scattered over the whole surface of the body. The tumours increase slowly, frequently taking months to attain their full size, and then remain without much alteration for an indefinite period. Slight injuries cause their destruction. They not infrequently suppurate, and may then be a source of much pain and inconvenience; but their tendency is to wither away or to disappear from some accidental injury. Scars do not remain unless suppuration has occurred or they have been removed by violence.

CONTAGION.—That the disease is contagious is shown by the following evidence: (1) Many well-authenticated instances are now recorded of the disease spreading from one individual to another. One child after another in a family becomes affected, and nurses communicate the disease to children under their charge. One of the most interesting examples of conveyance of the disease from one individual to another is given by Neumann. A group of cases consisted of a mother and twins—infants at the breast. The disease showed itself in one of the infants, the mother's breast was then affected, and, finally, the other child showed the disease. (2) Outbreaks of the disease in hospitals and schools have frequently been placed on record. (3) Instances are recorded of physicians and others accidentally inoculating themselves by contact with cases of the disease. Brocq is quoted by Stelwagon as follows: 'I inoculated myself involuntarily with molluscum with my nails, after having pressed out with the nails of the two thumbs the contents of a lesion of molluscum in a patient. Soon afterwards I inadvertently scratched my face. About a month and a half later several lesions of molluscum developed in this region.' (4) Many attempts have been made to inoculate the disease experimentally, and though in the majority of the instances the experiment has failed, enough successful inoculations have been recorded to establish its possibility. The exact nature of the contagion is unknown. The incubation-period seems to vary from four weeks to six months, the usual period being from six weeks to three months.

It is worthy of remark that certain animals, especially birds, e.g. pigeons and domestic fowls, suffer from a disease affecting the head, beak, and claws very similar in appearance to molluscum contagiosum, which has been described as *epithelioma contagiosum* of birds. Cases have been described of persons handling animals so affected who presented lesions resembling those of molluscum contagiosum.

HISTOLOGY.—Recent observations show that the tumours do not arise from the sebaceous glands, as was formerly supposed. The increase in the cells takes place in the rete Malpighii, at one point or at several points in close vicinity. The mass of new tissue pressing downwards tends to flatten the underlying papillae, although some of them may remain and serve to form the fine fibrous partitions which separate the lobules of the growth from each other. The overlying epithelium becomes raised and stretched, and the pressure thus produced serves to retain the little lobules of the tumour in close contact with each other. As the tumour increases in size, one or more depressions or umbilications appear on the surface, due to falling inward of the most projecting point of the growth, and then the mass of newly-formed epithelium below appears to be in continuity with, and invaginated from, the over-

lying epithelium. On examining the interior of the growth the cells of the rete are seen to be large, but may seem normal in other respects, showing their 'prickle-systems' as in the normal skin. The stratum granulosum is present, and usually more developed than usual. Its cells retain a regular arrangement till degeneration of the epithelium occurs, when slight injury readily causes them to become displaced. The keratinisation of the epithelium occurs normally; but it is noted that the cells of what should form the horny layer do not shrivel in the usual way, but remain large and become more and more distended. This distension is due to the appearance of a peculiar colloid or hyaline degeneration of the protoplasm of the cells, which may be noted in the cells of the stratum mucosum, but becomes more obvious as the horny layer is reached. As the cells become distended with the material, they produce certain peculiar microscopic appearances, owing to the distribution of the colloid substance within the cells, which have been recognised for long as the 'molluscum-bodies,'—the cells in the horny layer becoming completely distended, showing almost no trace of their nuclear chromatin, and resembling small cysts. Some observers have affirmed that these bodies are intracellular protozoa. But none of the signs of reproduction have been satisfactorily observed in the molluscum-bodies. They have not been grown externally to the body; and therefore no successful inoculation from a pure culture has been made. The hypothesis that these 'molluscum-cells' show protozoal infection can therefore no longer be sustained.

As the horny cells form they lose their 'prickle systems' and become less adherent to each other. At the same time the hyaline degeneration of their protoplasm renders them soft, so that pressure on the tumours causes the internal degenerated mass to become expressed as the 'curdy' material so easily recognised. At or near the surface of the tumour, intermingled with the molluscum-cells, various bacteria may be recognised. Their presence is accidental, but explains the ease with which suppuration may arise.

TREATMENT.—The injury done to the tumour by expressing its degenerated contents is frequently sufficient to cause it to shrivel and disappear. The introduction of a drop of pure phenol, acid-nitrate of mercury, or of a sufficiently strong solution of iodine, by means of a glass rod or a spill of hard wood, is, however, a more satisfactory method of destroying the growth. Often the most satisfactory method of treatment is to split the small tumours to their bases with a suitable sharp knife, empty the contents by means of a small curette, and then control the small amount of hæmorrhage likely to ensue with a pad of lint and bandage, or with a collodion-gauze dressing. Suppuration is apt to be caused, and should be avoided with care. Occasionally it is wise to excise the tumour entire, so as to produce a 'clean cut,' especially when suppuration must be rigidly avoided, as in the neighbourhood of the orbit. Electrolysis is of considerable service in destroying the tumours when they occur in situations rendering an incision inadvisable. In cases where small tumours are present closely aggregated on the trunk or extremities, it may be advisable to attempt their removal by means of flexile collodion or plaster-muslin, containing from 2 to 10 per cent. of salicylic acid.

JAMES GALLOWAY.

MOLLUSCUM FIBROSUM.—The name is applied loosely to certain forms of fibroma arising from the connective tissue of the skin and subcutaneous tissue. They usually make their appearance in early life, but become larger, and apparently more numerous, later on. They are often of congenital origin, due to defective development of the mesoblastic elements of the skin. In the majority of cases only a few small tumours may be noted; perhaps it may be said that there are few persons who do not develop small examples of molluscum fibrosum at some period.

The tumours are, however, frequently numerous. On palpating the skin small roundish gaps may be felt in the cutis due to the development of the peculiar fibrous tissue of the tumour. The tumours as they develop produce hemispherical projections from the surface, which may attain large dimensions; and these often become pedunculated as their size and weight drag on their attachment. They are in some cases innumerable; cases are not infrequent when, owing to their great numbers and size, they completely disfigure the parts affected; and when the whole body is affected, as is occasionally the case, the sufferer almost loses human semblance. These cases are usually accompanied by other anomalies, such as angiomas, pigmentation, and excess of hair; and not infrequently the sufferers are mentally deficient. Occasionally large areas of the skin are affected by overgrowths of similar character, giving rise to pendulous folds of skin, instead of isolated tumours. To such cases of the disease terms such as *molluscum pendulum*, *dermatolysis*, and *pachydermatocle* have been applied.

After reaching a certain size the tumours usually remain passive for an indefinite period, though occasionally some of them may shrink and disappear. They do not develop the characteristics of malignancy.

The tumours consist of bands of interlacing fibrous tissue bundles of differing consistence, but usually more or less oedematous. In many cases, especially when the tumours are numerous, there can be little doubt that they develop from the connective tissues of the peripheral nerves (von Recklinghausen), and are in reality *neurofibromata*. Such cases are often associated with multiple fibromata of the nerve-trunks.

TREATMENT.—No method of treatment has been suggested to prevent the development of the tumours. When they are in situations likely to be injured, or when they interfere mechanically on account of their size, they may be removed by surgical methods.

JAMES GALLOWAY.

MONOMANIA.—SYNON.: FR. *Monomanie*; Ger. *Wahnsinn*.—This term is falling into disuse by reason of its vagueness, and because it has been employed by various writers to denote different kinds of insanity. Some have used it to denote an insanity which is indicated by some one particular delusion, the mind remaining clear on every other point. Others mean by it an insanity without delusion, an *affective* or *impulsive* insanity, the essence of which is the absence of delusion, and the so-called integrity of the intellectual portion of the mind. Esquirol thought it a disorder of the faculties limited to a few subjects, with excitement, and gay and expansive passion; while, according to others, melancholia without delusion would be an instance of affective monomania. We may take it,

however, that all authors are agreed in using the term 'monomania' to indicate a partial insanity, which enables the patient to converse and act rationally to a considerable degree, and therefore renders his responsibility a matter of question. Such cases form the grounds of forensic contention, whether criminal or civil; but it is better to affix to them some more precise term, and to indicate symptomatologically and pathologically the exact nature of the mental and bodily condition of the alleged lunatic. See *INSANITY*, Delusional, p. 763.

G. F. BLANDFORD.

MONSTROSITIES AND MALFORMATIONS.

—Under the heading of 'Diseases of the Fœtus' (p. 549) the results of the action of morbid agencies upon the unborn infant during the foetal period of antenatal life have been dealt with. When, however, the causes of disease act upon the unborn in the embryonic or formative period of intra-uterine existence there is reason to believe that they produce monstrosities and malformations, and not maladies. Embryonic physiology is embryology, and embryonic pathology is disordered embryology or teratology; when the chief function of the organism is to form organs, the most striking result of disordered function will be malformed organs; normal embryogenesis and organogenesis become under pathological influences teratogenesis and morbid organogenesis. If this theory of the causation of monstrosities and malformations be accepted, much that is inexplicable otherwise is done away with. Of course the period during which malformations can originate is thus narrowed down to the earliest weeks of antenatal life; at the same time all anomalies of formation do not take origin in these few weeks, for some organs and some parts of the body are longer in passing through the embryonic stage than others; and so long as they are in the embryonic or formative stage they are liable under the action of pathological agencies to teratogenesis or malformation. There is thus a difference in degree only between the monstrosities and the malformations, and the usual mode of action of the teratogenic cause is to arrest embryogenesis or organogenesis, leaving as a permanent condition what should normally be only a passing or transitory one. Inasmuch, however, as in the formation of the human embryo various scaffoldings are used, reminiscent of the embryogenesis of organisms lower down in the zoological scale than man, the results of this arresting process become very complicated, as is seen, for instance, in the persistence of various portions of the branchial arches and clefts. One at least of the means by which these arrested developments are produced is defective growth of the amnion, on account of which the membrane either remains in close contact with the developing part exercising pressure upon it, or forms attachments to the embryo which afterwards make traction upon special regions and deform them. In this way a great many of the anomalies which used to be ascribed to foetal diseases can be much more satisfactorily explained; it is not a foetal disease setting up a monstrosity, but a monstrosity arising primarily; in a word, the monstrosities and malformations are the only diseases which can be expected to affect the embryo; they are the diseases of the embryo. The double monsters, however, and monstrosities by excess (showing redundancy of parts) can hardly

be explained by the theory of arrested development due to amniotic pressure, &c. For their origin it is necessary to consider the period of antenatal life preceding the embryonic—the germinal, in other words; there is much evidence to help to prove that the double character of the embryo is determined before the first embryonic rudiments appear in the embryonic area of the blastodermic vesicle. Possibly polyspermia or the entrance into and functional influence of more than one spermatozoon upon the ovum may be one at least of the ways in which double monsters or united twins are determined. When the malformation or monstrosity has been produced *in utero*, the monstrous foetus may be carried through the rest of antenatal life, for as a rule the placenta is not involved to a lethal extent; and it is remarkable to note to what an enormous degree the foetus may be deformed and intra-uterine life still be possible of continuance. It is this fact that has led some to conclude that the monstrosities found on the foetus at birth have been produced during the foetal period of antenatal existence.

CLASSIFICATION.—No scheme of classification yet proposed is altogether satisfactory, for malformations and monstrosities do not lend themselves to arrangement in linear series, but exhibit intermediate types or connecting links for which it is difficult to find a place, or which seem to be equally well placed in two, or even three, different groups. It will be sufficient for practical purposes to make two large divisions, in one of which are placed the malformations and monstrosities characterised by defect of formation or by altered relation of parts (monosomatous terata), while in the other are those with excess of formation as their prominent feature (polysomatous terata). The latter group will be considered first.

Polysomatous Terata or Double (and Triple) Monsters.—Under this heading come the cases of two (or very rarely three) foetuses united to each other with all the possible degrees of fusion, from those with a single body, four limbs, and a head, showing more or less duplicity, to those in which the foetuses are separate save as regards the vessels of the umbilical cords which anastomose with each other. The former are instances of what are commonly called double monsters or united twins, and the latter are known as allantoideo-angiopagous twins.

United Twins.—These may be of two kinds—symmetrical, in which it can be seen that two foetuses of fairly equal size are joined usually by corresponding parts, according to Saint-Hilaire's '*loi d'affinité du soi pour soi*;' and asymmetrical, in which one foetus is much smaller than the other, and is commonly described as parasitic upon it. As examples of the first group may be mentioned the thoracopagous twins, such as the Siamese brothers, in which there are two almost complete bodies united more or less extensively in the thoracic and abdominal region; the dicephalic terata, in which the lower part of the monstrosity is single while the upper part shows more or less complete duplicity (example, Rita-Cristina); and the syncephalic terata, in which the upper part of the bodies is so fused as to appear as single, while there are four lower limbs and sometimes also two vertebral columns in the lower part. Almost all the possible intermediate types have been recorded, and the leading types have thus been proved to

overlap and shade off into one another. As instances of the second group or parasitic monstrosities reference may be made to the cases in which a more or less defective foetus is attached to the thorax, or face, or sacral region of an otherwise normal twin; the name autosite is given to the latter, and parasite to the former.

Placental Parasites or Allantoideo-Angiopagous Twins.—Closely related to the rudimentary twin attached to the umbilicus of the autosite (gastro-parasitus) is the rudiment-twin attached to the umbilical cord or placenta of its normal brother or sister twin. These placental parasites are often the subjects of the most advanced degrees of monstrosity; they may be headless (acephalic), heartless (acardiac), trunkless (acornic), and even formless or rather globular (anidean or myelacephalic). That they continue to live *in utero* is due to their obtaining nourishment from the co-twin, for their vascular system is in communication (through the placental vessels) with its heart and circulation. They do not survive birth. Sometimes the placental parasite may be one of triplets, or, very rarely, of quadruplets.

Monosomatous Terata or Single Monsters.—Far more commonly met with than the double monsters are the cases of deformity or malformation in an infant which shows no sign of duplicity, and gives no indication that it has arisen from two embryos. Various modes of arrangement of anomalies in formation have been proposed; but for convenience we shall here group them under the various regions of the body. At the same time it is a common occurrence to find a foetus deformed in several parts, and in more than one region of the body, as, for example, in the cases of adhesions of the amnion to the foetus, where amputations of the fingers and toes, hare-lip, facial fissure, exomphalos, and spina bifida may all co-exist.

Cranial Malformations.—Perhaps the most common of the marked monstrosities of the cranium is anencephaly (sometimes but incorrectly termed acephaly). In it there is more or less complete absence of the bones of the cranial vault, along with a condition of open spinal canal. On the basis crani, which generally is directed backwards instead of upwards, a mass of fibrous tissue and blood clot is usually found, the brain being absent. The face of the foetus looks upwards, and there is no indication of a cervical groove anteriorly. The pregnancy which results in the birth of such a foetus is often complicated with hydramnios, and ends either prematurely or (less commonly) postmaturely. The malformation is compatible with intra-uterine, but not with extra-uterine existence. Iniencephaly is a state in which the cranium, which is present but defective in the occipital region, is sharply retroflexed upon the spine (retroflexion of the foetus), which is often partly open; in this way the head and trunk of the foetus form together a rounded mass with the limbs as appendages, and the cervical part of the spine may lie almost at the centre of this mass; in very advanced degrees of iniencephaly the gluteal muscles may be attached to the occiput. Microcephaly or unnatural smallness of the head is commonly associated with idiocy. Hydrocephaly, which is more probably to be reckoned as a malformation than as a disease, produces great enlargement of the head, but as the brain-substance is not increased but diminished by the presence of the fluid in the enlarged ventricles,

the individual may likewise be an idiot. When hydrocephaly is marked at birth it may cause some delay in labour; in cases of spina bifida there may be some degree of hydrocephalus without any external indication of it. Various swellings may be met with on the head of the foetus,—defects in the cranial bones allowing the escape of part of the contents account for some of these; according to their contents they may be encephalocoeles, hydran- cephalocoeles, or meningoceles; and according to their position they may be occipital, frontal, or parietal. If sepsis be avoided these swellings can sometimes be successfully removed, but there is always the risk of the subsequent development of hydrocephalus. Imperfect ossification of the parietal bone in the region of the obelion may predispose to the occurrence of subpericranial cephal-hæmatoma.

Facial Malformations.—The number of malformations which may affect the face and the organs of special sense is very great. One of the most striking of these is cyclopia or the fusion (more or less complete) of the two orbits and the two eyeballs into one central cavity and contents. Above this (rarely below it) is a projection or proboscis which indicates the nose; there may be also absence of the lower jaw (hypo-agnathia). Agnathia may occur apart from cyclopia, and may be associated with absence of the mouth or with microstoma; it is a striking fact that agnathia is rare in man but comparatively common in fetal lambs. Macrostoma or a wide buccal opening is often due to or combined with facial fissure (single or double) of the transverse type; evidently the anomaly is due to the persistence of the inter-maxillary fissure of embryonic life. Another type of facial fissure is the ascending or vertical one which passes up from the mouth at the side of the nose towards the inner angle of the orbit, and may be associated with malformations of the eye or eyelids. Hare-lip is a common malformation, and apt to run in families; bilateral fistula of the lower lip is much less common, and may be associated with hare-lip. The nose may show a mesial fissure, and congenital deviations of the septum are not rare; perforation of the septum also may occur as an antenatal anomaly. Anophthalmos is not often met with, but cryptophthalmos is more common; and there are many other anomalies of the eye, such as coloboma of the iris, aniridia, and corectopia. The external ear may be deformed in various ways: there may be a fissure in the lobe or a fistula in the upper part of the helix; and in front of it or attached to it may be found little fibro-chondromata or pre-auricular appendages supposed to be relics of the branchial arches; in agnathia the two ears may be approximated under the face, and give rise to otocephaly. Supernumerary tympanic ossicles have been recorded.

Cervical Malformations.—The commonest malformations in the region of the neck are of the nature of cysts or fistulae connected with permanent rudiments of the branchial arches and thyreo-glossal duct. The cervical auricles are generally of the nature of chondromata. Sometimes cervical cysts attain large and deforming dimensions, hygromata of the neck. There may be an accessory thyroid, or absence of the thyroid, or persistence of the thyreo-glossal duct.

Thoracic Malformations.—The thoracic walls may be defective at various points. When there is

a fissure or defect of the sternum the heart may project outside the thorax (ectopia cordis), and this does not necessarily lead to death. Cervical ribs may be met with, as also a congenital displacement upwards of the scapula. The pectoral muscles may be absent, an anomaly which is not uncommonly combined with absence of the nipple on the same side and with malformations of the hand and fingers, as if some common cause had interfered with the development of the chest and arm. Supernumerary mammary glands may be found in the axilla or below the normal ones (polymastia). The œsophagus may be imperforate or communicate with the trachea. Cardiac malformations, however, constitute the most important of the thoracic anomalies. They may be of the nature of imperfect closure of the interventricular or interauricular septa, of absence of obliteration of the ductus arteriosus, of stenosis or (rarely) obliteration of the pulmonary artery or (less commonly) of the aorta, or of transposition of the great vessels. See HEART, MALFORMATIONS OF.

Abdominal Malformations.—Among the structural anomalies of the abdomen and its contents are the various forms and degrees of defect of the walls, leading to exomphalos when the defect is widespread, and to umbilical hernia when it is small. Exomphalos is often associated with retroflexion of the spine, and the peritoneum may be continuous with the amnion, no true umbilical cord being present. Malposition of various parts of the intestine is not uncommon, and there may be atresia at various points; in the latter case the infant after birth will pass clay-coloured stools instead of the dark green meconium. The rectum may be absent or the anus imperforate; in the latter instance operative means will usually prove sufficient, in the former success is more doubtful. The lower end of the bowel may open into the bladder (atresia ani vesicalis), or into the vagina in the female (atresia ani vaginalis or vulvar anus). The bladder may be open to the front, ectopia vesicæ, and give rise to urinary incontinence, a condition which it has been found very difficult to correct; among the latest suggestions for treatment is transplantation of the trigone of the bladder into the colon. The two kidneys may be fused into one (horse-shoe), and displaced downwards into the pelvic brim; either of the ureters may be double; and there may be persistence of the urachus leading to abscess or cyst in the anterior abdominal wall. Umbilical fistulae may be caused in the same way or may arise from the opening at the navel of an omphalo-mesenteric duct. Supernumerary spleens occur.

Genital Malformations.—So important are the genital malformations that in some text-books a special subdivision is set apart for them under the name of Hermaphrodites, and it is true that many of them give rise to doubts as to the sex of the individual who is thus malformed. This remark applies specially to such malformations of the external genitals as hypospadias (an imperforate condition of the penis, the urethra opening on the under surface of it or in the perineum) or epispadias (urethra opening on the upper surface of the penis) in the male, and to imperforate vagina and vulva in the female, especially when combined with hypertrophy of the clitoris; but these cases are better termed pseudo-hermaphroditism, for the individual is not really anatomically or physiologically

of double sex, but is only of doubtful sex until the internal organs can be examined. Various forms of double uterus and vagina are met with which are due to the persistence of the original condition of duplicity of these organs. The testicles may be undescended or lying in the inguinal canal; in both of these circumstances it is doubtful if they are of any use.

Spinal Malformations.—An open condition of the spinal canal, or spina bifida, is a not uncommon malformation; it generally affects the posterior part of the spine in the lumbar or dorsal region, but it is also met with in the cervical region, and rarely in the anterior or lateral part of the spine. Through the opening the spinal membranes or the cord itself may project, giving rise to a tumour which often ulcerates after birth; but there may be no projection (*spina bifida occulta*), a condition sometimes associated with localised hypertrichosis and club feet. Other spinal malformations are congenital scoliosis and absence of vertebral bodies; and sometimes there are supernumerary coccygeal vertebrae giving rise to a 'tail.'

Malformations of the Limbs.—Many of the minor malformations of the limbs, such as club-foot and hand, webbing of the fingers, &c., are referred to elsewhere. *Symphodia* is a rare monstrosity in which the lower limbs are more or less completely fused into one, constituting the so-called siren foetus; not only are the limbs fused, they are also rotated so that the popliteal spaces face anteriorly; the external genitals are usually quite absent, and the lower end of the bowel grossly malformed. The limbs may show various degrees of imperfect development; they may be completely absent (*amelia*), or shorter than normal (*phocomely*), or defective in any of the segments (*ectromely*). Supernumerary digits are not uncommon (*polydactyly*), the extra digit being most often a little finger or toe; on the other hand, absence of one or more digits is met with (*ectrodactyly*). Any of the long bones of the extremities may be wanting.

Transposition of the Viscera.—*Situs inversus*, or *Heterotaxy*, is an interesting anomaly in which we find on opening the body-cavities that the organs all have the appearance that they would present if seen in a mirror. The stomach lies on the right side and the liver on the left, the spleen is on the right, and so on down even to the smallest differences, such as the height of the kidneys and the arrangement of the vessels. It is sometimes but not invariably associated with left-handedness. A condition, which at first sight resembles it, is due to congenital diaphragmatic hernia; for in it the intrusion of some of the abdominal contents into the thorax causes a displacement of the heart to the right side (*dextrocardia*); but the two conditions are quite different in their origin, although it is interesting to know that they have been met with associated.

Teratogenesis or experimental teratology is a product of recent years; it has been chiefly carried out with the hen's egg in incubation; and it has been found that by disturbing the normal incubation-conditions (by increasing the temperature or by injecting into the albumen poisons, microbes, and toxins) various forms of monstrous development can be produced, although it has not yet been made possible to foretell what type of monstrosity will result in any given case.

J. W. BALLANTYNE.

MONSUMMANA, Cave of, in Upper Italy.—Natural vapour-baths. See MINERAL WATERS.

MONT DORE, in France.—Simple thermal water, containing arsenic and soda. See MINERAL WATERS.

MONTE-CATINI, in Tuscany.—Thermal muriated saline waters.

MONTMIRAIL, in France (Vaucluse).—Sulphated saline, bitter waters.

MONTPELLIER, in the South of France. Variable, fairly warm, winter climate. High winds from N.E. and N.W. See CLIMATE, Treatment of Disease by.

MORAL INSANITY.—See INSANITY.

MORBID (*morbus*, a disease).—This word merely signifies *diseased*, and is used, in its several applications, as a technical or scientific term, in contradistinction to the term *healthy*.

MORBIDITY (*morbus*, a disease).—This term is employed to denote the amount of illness existing in a given community; and, as 'mortality' expresses the death-rate, so 'morbidity' indicates the sick-rate, whether the diseases be fatal or not.

Since health is an extremely ill-defined state, marked out by no absolute boundaries, and since many people suffer from diseases that are concealed intentionally or through ignorance, it becomes a matter of considerable difficulty to express with certainty the amount of illness that may exist at any time. Some information may, however, be obtained from the records of sick clubs and benefit societies, on which statistics may be based of the average time their subscribers are ill during the year, in relation to employment, age, locality, and other circumstances. From the statistics of these friendly societies it has been estimated that nine days in the year per member are lost through sickness.

By an investigation of this subject the rates of mortality come to possess an extended significance, for they thus indicate not merely the proportion between the living and the dead, but between the latter and the two classes of the living, namely, the healthy and the diseased; and, as a branch of State Medicine, they must doubtless come to take a prominent place. As further knowledge provides accurate facts and figures, the subject will have a distinct practical bearing, in estimating the value of men for work, if the average liability to disease and the total amount of illness an individual may expect to suffer be known; while it is reasonable to believe that as the 'aptitudes to disease' are further conditioned, the means for prevention may be extended.

W. H. ALLCHIN.

MORBIFIC (*morbus*, disease; and *facio*, I make).—This word is properly applied to any cause that produces a disease. Such a cause is often spoken of as a *morbific agent*.

MORBILLI (dim. of *morbus*, a disease).—A synonym for measles. See MEASLES.

MORBUS.—This is the Latin word for *disease*. Formerly it was frequently employed, but is not

much in vogue at the present day. It still persists in such terms as *morbus cordis*, disease of the heart; *morbus coxae*, disease of the hip-joint; *morbus cerealis*, ergotism; *morbus Brightii*, Bright's disease; *morbus cæruleus*, blue disease, congenital heart-disease.

MOROCCO, in North Africa.—A warm, healthy winter climate. Tangiers is exposed to cold, damp S.W. winds in autumn and spring, and to E. winds. Living is superior to Malaga. See CLIMATE, Treatment of Disease by.

MORPHINISM.—**SYNON.:** The Morphine-Habit; Fr. *Morphinisme*; Gr. *Morphiumsucht*. See HABITS.

MORPHŒA.—See SCLERODERMIA.

MORTALITY.—**SYNON.:** Rate of Mortality; Death-rate; Fr. *Mortalité*; Ger. *Sterblichkeit*.

DEFINITION.—The proportion of persons dying to those surviving under given circumstances; or, more usually, the proportion borne by the persons who die to the whole number of those subjected to the given circumstances.

Thus we may have to do with the annual mortality of the population of a country, a district, or a city; or of a body of men similarly circumstanced, as of clergymen or of lead-miners; or of bodies of men otherwise alike, but subjected to different conditions of climate, &c., as the British army; or of the population, or any section of the population, at special ages, as of infants in factory towns.

Or we may be concerned with the proportions of deaths to survivors, or to the whole number of entrants, during and after exposure to a special cause or causes of death, operating either speedily or during a protracted period. Hereunder come, for example, the mortality sustained by the population of Rio Janeiro, or New Orleans, during an epidemic of yellow fever; or that suffered by a number of persons in passing through an attack of enteric fever or pneumonia.

ESTIMATION OF MORTALITY.—The annual mortality of a population is reckoned, not on the numbers in existence at the beginning of a year, but on the average number in existence on the several days of the year, or, what is nearly the same thing, on the mean population of the year. The necessity of this becomes evident, when we consider that in our own country the large towns are mostly increasing at a very rapid rate, while many agricultural parishes and unprosperous places actually decline in population. In the towns, therefore, the death-rate, if reckoned on the last census, or even on the number believed or estimated to exist at the beginning of the given year, would come out higher than it ought to be, while in declining parishes it would be somewhat too low. Similarly the annual mortality of bodies of troops is calculated on the mean strength.

Two formulæ are in use for specifying death-rates. In the first the proportion of deaths is taken as unity: thus, the mortality in England and Wales in 1899 would be stated as 1 in 54. In the second, which is more convenient and is now generally employed, the number of lives at risk is taken as 100 or 1,000: thus the mortality of 1899 would come out 18·3. Either formula is convertible into the other by simple division: thus, $1,000 \div 46 = 21\cdot7$: and $1,000 \div 21\cdot7 = 46$.

The death-rates of large civilised countries in which registration is strictly carried out give a pretty fair representation of the viability of the population. So much may be said for England, Wales, and Scotland, and for most of the European states, but not, unfortunately, for Ireland, where the weakness of the registration laws makes the record defective.

MORTALITY OF NATIONS.—The following were the death-rates per 1,000 of most of the principal states of Europe during the period indicated—1884 to 1893 inclusive:—

Norway, 16·9	France, 22·4
Sweden, 16·9	Portugal, 24·0
Ireland, 18·1	Prussia, 24·1
Denmark, 18·9	Russian Poland, 24·4
England and Wales, 19·2	Germany, 24·6
Scotland, 19·2	Württemberg, 25·2
Switzerland, 20·5	Italy, 26·0
Belgium, 20·5	Servia, 27·0
Holland, 20·6	Saxony, 27·3
Finland, 20·8	Bavaria, 27·6
Japan (?), 21·0	Roumania, 30·0
Greece, 21·7	Spain, 31·0
Bulgaria (?), 22·1	Hungary, 32·2
	Russia (Eur.), 34·7

In Russia, Spain, Bavaria, Austria, Hungary, Italy, Prussia, Saxony, Servia, the high figures are largely due to the high mortality under five years of age; while Sweden, Denmark, Switzerland, Ireland, and Greece owe their favourable position largely to their small mortality under that age. And in all of them, except Russia, Austria, Hungary, Spain, and perhaps Greece, the greatest quinquennial mortality after childhood falls from 70 to 75 (in Norway over 75), so that the force of viability does not differ so much as the rate of mortality. In many of the British colonies it is lower than even in Norway. Thus the average mortality during the five years 1882–6 was in

Victoria, 14·8	New South Wales, 15·6
Queensland, 19·7	South Australia, 14·3
West Australia, 18·6	Tasmania, 15·6
New Zealand, 10·8	

and in 1895, which, however, was a very healthy year, the mortality rate was in

New Zealand, 9·91	New South Wales, 11·87
South Australia, 11·20	Victoria, 13·25
Queensland, 11·38	West Australia, 17·32
Tasmania, 11·38	

MORTALITY OF CITIES.—The mortality of cities is in this country almost invariably higher than that of the rural districts. But this rule does not apply to all other countries; the exceptions occur mostly where endemic fevers are prevalent in the country. Thus the mortality-rate in 1887 was in London, 19·6, in Edinburgh, 19·8, and in Dublin, 30·6; and in 28 other large towns in England it varied between 16·9 in Brighton and 28·7 in Manchester; while in 50 towns of the second class the extremes were 12·4 at Maidstone and 13·3 at Burton; 23·8 at Stockport and 24·9 at Wigan; the average of the 28 towns being 20·8 per 1,000, and of the 50 towns 19·0. In the same year the rural districts and small towns of England yielded an average rate of 17·4 only. There are a considerable number of districts, almost all rural, which year after year fall below 17; and 17 was accordingly fixed upon by William Farr as a kind of standard to be aimed at by sanitarians. And there are districts in England, and entire small counties in Scotland, where the corrected rate frequently falls below even 15 or 14.

The following table exhibits the death-rates experienced in 1878, and again in 1887, in a number of foreign and colonial cities :—

	1878	1887		1878	1887
Calcutta	37.7	24.0	Paris	24.0	23.0
Madras	48.8	40.0	Brussels	28.0	21.0
Bombay	41.8	26.0	Amsterdam	24.4	22.0
New York	24.8	26.0	Rotterdam	27.3	21.0
Brooklyn	20.1	23.0	The Hague	26.4	20.0
Philadelphia	18.0	22.0	Copenhagen	22.0	24.0
Montreal	30.9	—	Stockholm	22.4	21.0
Alexandria	45.4	38.0	Christiania	18.5	23.0
Melbourne	—	—	St. Petersburg	47.1	28.0
1873 and 1875	22.8	21.0	Berlin	29.9	22.0
Rome	29.8	29.0	Hamburg	26.9	27.0
Naples	33.1	—	Dresden	24.7	22.0
Turin	31.1	26.0	Munich	34.6	30.0
Venice	28.7	25.0	Breslau	29.9	29.0
Trieste	36.2	29.0	Vienna	29.6	26.0
Geneva	23.6	21.0	Budapest	40.3	32.0

The next table, including several of the same, with some additional cities, shows in several instances, though not in all, the effect of perseverance in sanitary improvement :—

	1882-91	1892-3		1882-91	1892-3
London	20.37	20.79	Vienna	21.53	24.09
Paris	23.80	21.97	Budapest	30.90	27.00
Brussels	24.39	21.63	Rome	31.16	29.02
Berlin	23.09	20.42	Trieste	25.88	21.97
Hamburg	24.78	28.95	Naples	30.08	29.08
Dresden	21.87	23.36	Milan	28.06	25.61
Breslau	29.22	29.42	Venice	30.89	27.51

ANALYSIS OF RESULTS.—These tables awaken, by the enormous differences between the several cities and countries, a curiosity respecting the causes of such differences, which, however, the figures themselves go far towards satisfying. It is at once evident that, whatever may be the case in the open country, cities suffer to a considerable extent in the ratio of their ignorance and neglect of sanitary laws, and of the poverty and squalor or barbarism of their populations. Mark, for example, the contrast between Philadelphia or Geneva, and Alexandria, Budapest, or St. Petersburg! Cities having a steadily warm climate, or a climate of extremes, are more unhealthy than those which enjoy a temperate one. By this consideration, combined with that of their superior civilisation, may be explained the favourable position of the cities of Western as compared with those of Eastern Europe. The short, hot summers are very fatal in the latter region, and even in Southern Germany and at Stockholm; while in Western Europe generally, and especially in Scotland, winter and spring are the deadly seasons. It is noteworthy that in most of the large cities of Italy the short, sharp, and changeable winter is not less deadly than the hot summer and malarious autumn.

In Great Britain the influence of climate *per se* on the annual mortality of the several cities and districts is not very great; and its effects are obscured by those of other agencies. But if we confine our attention to the rural districts, where the disturbing factors are less important, we shall find that the rates of mortality are on the whole slightly more favourable in the north than in the south. Of all the counties in Great Britain Orkney- and-Shetland stands best, with an annual crude mortality, on an average of 10 years, of 15.13, which would be still lower if standardised; and Shetland, the more northern division, stands better than Orkney. Great Britain is, therefore, no exception to the rule that in Europe mortality decreases from south to north. This is in no way inconsistent with the fact that throughout Great Britain winter

is the deadly season, and cold is more fatal than heat, thoracic than abdominal diseases.

INFLUENCE OF SEASONS.—The following were the death-rates of the four seasons in England and Wales, in 1868-77 :—

	Winter	Spring	Summer	Autumn	Year
In the chief towns	25.8	22.5	23.1	24.2	23.7
In the small towns and rural districts	21.7	19.3	17.2	18.5	19.0

In Scotland the seasonal mortality, owing, doubtless, to the less intensity of the summer heat, follows pretty nearly the order of the English small towns and rural districts. Thus, in 1878: winter, 25.2; spring, 23.2; summer, 19.8; autumn, 20.4; year, 22.3. In Ireland (1881-90) the proportions were: winter, 21.7; spring, 19.1; summer, 14.7; autumn, 16.4. Here the influence of cold is strongly marked.

It would seem, however, that in London, in the early part of the seventeenth century, when the death-rate, owing to the closeness and filthiness of the city, was fearfully high, the maximum was attained in summer, the figures standing as follows in 1606-10, during which years the plague was absent. Average mortality per cent.: winter (J. F. M.), 1.4; spring, 1.5; summer, 2.7; autumn, 2.0; total, 7.0.

INFLUENCE OF DENSITY OF POPULATION.—In accordance with a principle already laid down, that in communities sufficiently advanced to furnish mortality statistics the death-rate diminishes with the progress of civilisation, the mortality of London has since the seventeenth century gradually and greatly diminished. At the beginning of the nineteenth century it had sunk to 29, in 1840-49 it was 25.3, in 1870-78, 23, in 1881-90, 20.31, and in 1891-99 only 19.6.

The death-rate is also diminishing in France, Belgium, the Netherlands, Sweden, Germany, and Italy, in all of which countries the population is believed to be advancing in comfort and general well-being; but in those parts of southern and eastern Europe, where comparatively little advance has taken place in these respects, no such diminution can be demonstrated.

Nor, though evident in London and in several other great towns, can a large and permanent diminution of the death-rate be positively affirmed of Great Britain generally. In Scotland, indeed, there was a decided increase from 1855 until 1876, when a decline, which may prove transient only, set in. And in England no improvement could be shown for many years before 1871, after which date there was a long and almost unbroken succession of years of low mortality concurring with a generally low temperature and excessive fall of rain.

The great antagonistic influence in Great Britain may be found in Dr. Farr's principle, 'That mortality increases with density of population.' And 'urbanisation' advances so rapidly in Great Britain, that all the efforts and devices of sanitary and medical science are scarcely able to do more than neutralise its evil effects. The apparent improvement is largely due to the diminution of phthisis and of typhoid fever. Contrary to the general belief, the expectation of life in males aged over 20, and in females over 45, was actually less in the period

1870-80 than in that of 1838-54, and less over 45 in both males and females in 1880-90 than in 1870-1880. But the expectation at birth has been very greatly increased owing to the diminished mortality in childhood and youth.

SOURCES OF FALLACY.—It may be as well to advert to some of the principal sources of fallacy, which hamper us in appreciating national and local death-rates. One of these is the varying number of births. The birth-rate ranges in the Continental States of Europe from about 40 in Germany and Austria, and even more in Russia and Hungary, down to 25 in France; and in Britain from 48 or 50 in some coal and iron districts, down to 22 in the county of Sutherland, and 14·8 in what is called the *City of London*. The late Dr. Letheby maintained that a high birth-rate was a direct cause of a high death-rate, owing to the great mortality among infants. This was an error; the two often concur, but the former is not a cause of the latter, unless where the infants perish in enormous proportion, as in most parts of Russia and in Spain. Thus in seven fertile provinces of Central Russia the birth-rate is 5·27 per cent., and the death-rate 4·09, about half the children perishing in infancy. But the usual result in England of a large and especially of an increasing birth-rate, is to augment in the community the proportion of children beyond infancy, and of young persons, who ordinarily suffer a very low death-rate as compared with old or even middle-aged persons. The favourable rates prevailing among these young persons overpowering the unfavourable ones of the infants, and of the comparatively small number of old people, the apparent death-rate is actually diminished, instead of being increased as Letheby supposed. And this points to the true reason why the death-rate of France is higher than that of England, whereas the expectation of life in the two countries is about the same at most ages, the birth-rate of France being so extremely low (Bertillon). *The lower the average age of the population, the lower the death-rate.*

A considerable amount of emigration or immigration affects the death-rate in proportion to the average age of the migrants. Thus the mortality of most great and growing towns would stand far worse than it does, were it not for the large numbers of young and healthy persons from the country who settle in them. Watering-places and residential towns appear somewhat healthier than they really are, by reason of the numbers of young domestic servants who form a large portion of their population. Various modes have been proposed for exhibiting fairly the true or standardised death-rate of communities, instead of the 'crude' one; or for adjusting the mortalities for differences of age- and sex-constitution of population. It is in our colonies that the effect of migration on the death-rate can best be studied. The unexampled death-rate of New Zealand, already quoted, is the result of two kinds of causes, one set of which we may call real, the other factitious or apparent. The former are the cool, equable climate, and the orderly and comfortable condition of the population; the latter are the constant stream of mostly youthful immigrants, and the very high birth-rate, now beginning to slacken.

INFLUENCE OF AGE AND SEX.—The influence of age and sex on the mortality in England and Wales may be best shown in a tabular form.

Mortality per 1,000 at twelve groups of ages in males and females in the 41 years 1838-78:—

	All Ages	0-	5-	10-	15-	20-	25-
Males	23'3	71	8	4	6	8	9
Females	21'2	62	8	4	7	8	9
		35-	45-	55-	65-	75-	85-
Males	23'3	13	18	32	67	147	311
Females	21'2	12	15	28	59	134	287

The superiority of the women is here well-marked, except during childhood and the years of early married life and much child-bearing. And this difference has been still greater during the later decades, owing chiefly to the decreasing vitality of males after the prime of life; thus in 1871-80 the death-rate of males from 45 to 55 was 20, that of females only 15.

Mortality per 1,000 at eleven groups of ages in males and females in the decade 1881-90:—

	All Ages	0-	5-	10-	15-	20-	25-
Males	20'2	61	5	3	4	5	7
Females	18	52	5	3	4	5	7
		35-	45-	55-	65-	75-	—
Males	20'2	12	19	34	70	162	—
Females	18	10	15	28	60	147	—

INFLUENCE OF RACE.—The influence of race is usually difficult to separate from that of habits of life. In Europe the Jews offer the most notable example. It may be sufficient to quote from Oesterlen Neufville's statistics of Frankfort-on-the-Maine, which show that there the average age of Christians at death was 36·9 years, but that of Jews was 48·7; and from Hoffman, the death-rate of the Jews of Prussia, which was only 21·6 per 1,000, against 29·6 among the Christians.

INFLUENCE OF STATION AND OCCUPATION.—The influence of station and occupation on mortality is very great. The subject has been carefully handled by Dr. Farr in the Supplement to the Registrar-General for England's thirty-fifth *Report*, and again by Dr. W. Ogle, in the Supplement to the forty-fifth, and by Dr. Tatham in that to the fifty-fifth. Briefly, it may be said that of all trades or professions that can be isolated, clergymen, barristers, farmers, agricultural labourers, gardeners, gamekeepers, and grocers seem to stand best in this respect. Booksellers, paper-makers, shipwrights, wheelwrights, and carpenters also suffer but a small mortality; so do lace- and hosiery-makers. Schoolmasters and teachers go on well up to fifty-five. Fishermen stand well. Solicitors, domestic servants, watchmakers, shoemakers, saddlers, bricklayers, and blacksmiths range not far from the average rates; so do bakers (though such is not the current opinion), and most kinds of weavers and of shopkeepers. The workers in iron, as a rule, experience but a low mortality in early life, but a rather high one as they grow older; the same may be said of millers, and, somewhat strangely, and no doubt for very different reasons, of Roman Catholic priests. Artists come out pretty well, musicians very badly.

Tailors begin very ill, and end fairly. Medical men, alas ! perish frequently in early life, and only attain a respectable position after fifty-five; the figures for chemists, also, are rather high. The figures for drapers, formerly bad, have improved of late years, as have those for commercial travellers and clerks. Those for miners, naturally enough, are not very different from those for iron-workers, except the tin and copper miners of Cornwall, who stand badly. Tobacconists, as might be expected, suffer heavily until middle life. Printers, bookbinders, coal-heavers, carriers, hairdressers, hatters, messengers, bargemen, and workers in lead (painters, &c.), glass-manufacturers, dock labourers, porters, railway employés, butchers, cabmen, draymen, and chimney-sweepers, all suffer a very high mortality. And about the worst positions are occupied by dealers in alcohol (brewers, innkeepers, and especially hotel servants), and by costermongers, potters, cutlers, and filemakers. The chief causes of a high death-rate in males are evidently alcohol and the irritation of inhaled dust; and, in a less degree, foul air in early life, and too violent exertion later on.

These facts are of considerable practical interest in relation to questions of life-assurance.

MORTALITY OF DISEASES.—Some acquaintance with the mortality of diseases, and the extent to which it is influenced by age, sex, climate, season, &c., is also of great value for prognosis. Information on this subject will be found under the heads of the several diseases; moreover, the limits of this article are not sufficient to admit of much discussion of the subject.

A few facts respecting the acute infectious diseases will, however, be of interest.

1. *Typhoid Fever.*—The average death-rate of enteric fever was put by Murchison, in accordance with British, French, and German hospital statistics, at 17·4 per cent. There is a good deal of ground for putting the average mortality of children and youths at 11 or 12, but it is probable that only the worst cases occurring in children find their way into hospitals. Over fifty years of age somewhere near one-half usually die (Liebermeister). The mortality from typhoid fever would appear, however, to be decreasing at a more rapid rate than the number of cases of the disease.

Still, the mortality in epidemics occurring among soldiers on campaign is often enormous. In the Nile campaign of 1898 it was 28 per cent. on admissions, in the Soudan in 1884–5 39 per cent. Even in the British army in India, for the last ten years, it has averaged 26·17 per cent., while in soldiers at home it has been as much as 19·16. In the Bloemfontein epidemic it is believed to have been about 21. It would seem that young adults have no advantage over other subjects in this respect. Some epidemics are less deadly than others. In that of Maidstone the percentage of deaths was 7·6. There is a mass of evidence showing that where the cold-bath treatment is carefully applied the mortality is reduced to a singularly constant average of between 7 and 8. See p. 136.

2. *Measles.*—The mortality from epidemics of this disease is often as low as 2 or 3 per cent., but it has been known to rise to 30 per cent. under unfavourable circumstances, as where children, or even adults, are crowded together in a hospital. It seems to be very deadly in Belgium. Among 'virgin' communities (as in well-known epidemics in Iceland,

Faroe, Madagascar, Fiji) the mortality is sometimes frightfully large. It is comparatively small in summer; and decidedly small among the comfortable classes, owing doubtless to the exercise of greater care. It is beyond comparison greatest in the second year of life, and by the tenth has become quite trifling; but adults *may* die of measles.

3. *Scarlatina.*—There is a prodigious difference in the deadliness of different epidemics of this disease, even in the same locality. In Southern Europe it is comparatively a mild disease; in Britain, Sweden, Austria, it is most severe; yet even here eighty successive cases may occur without a death. But a mortality under 10 per cent. may be considered moderate (Thomas, in Ziemssen); it is often much higher. It is at its maximum from the second to the fourth year, but continues very deadly up to ten or twelve; by fifteen it has almost reached a minimum, but, unlike measles, continues to be somewhat formidable throughout life, especially to parturient women. Season and station in life make little difference in its deadliness.

4. *Diphtheria.*—The severity varies extremely in different epidemics. It is much more deadly in Germany and Austria than in Great Britain.

5. *Small-pox.*—Small-pox did and does, in unvaccinated communities, where it has long been at home, destroy somewhere about 10 per cent. of the population; and of persons unprotected by vaccination who are attacked, 40 per cent. often perish. Among 'virgin' communities it is still more deadly. Age makes comparatively little difference in its fatality.

6. *Whooping-Cough.*—The death-rate of this disease is very large in the first year of life, declining afterwards like that of measles, but rather more rapidly, and becoming quite insignificant before the tenth year. Whooping-cough is more fatal in winter than in summer, in towns than in the country, among the poor than among the rich; but these differences, except the first, are not very well-marked. See VITAL STATISTICS.

JOHN BEDDOE.

MORTIFICATION (*mors*, death; and *facio*, I make).—A popular name for gangrene. See GANGRENE.

MORTON'S DISEASE.—A term applied to a form of metatarsal neuralgia in which attacks of severe pain occur at the base, generally of the fourth, but occasionally of the second toe. Ill-fitting boots and prolonged standing are among the accredited causes of the condition. In many cases carefully made boots and other appliances afford relief; in others excision of the head of the metatarsal bone may be necessary.

MORVAN'S DISEASE.—An affection met with especially in a district of Brittany, and named after the physician who first described it. The disease has strong points of resemblance to anæsthetic leprosy. It is a chronic affection, implicating the upper extremities more especially, and characterised by neuralgic pains, cutaneous anæsthesia, and painless and destructive whitlows. Well-marked neuritis and perineuritis have been found in the bodies of those dying of the disease; and in some cases these changes have been associated with syringomyelia. See SPINAL CORD, Special Diseases of: Syringomyelia.

MOUNTAIN SICKNESS.—SYNON. : Fr. *Mal des Montagnes* ; Ger. *Bergkrankheit*.

DEFINITION.—This term has been applied to a group of symptoms comprising lassitude, dyspnoea, headache, and palpitation, which affect some people climbing mountains of considerable height. The lower limit of altitude at which this affection appears cannot be defined, but most sufferers begin to experience discomfort at about 16,000 feet, which usually increases as the climber rises higher. The term 'mountain sickness' is unfortunate, as nausea and vomiting, though they sometimes accompany the attack, do not appear to be essential symptoms, but rather depend upon coincident dyspepsia from indigestible food.

ETIOLOGY AND ANATOMY.—The physical and physiological causes producing this condition are most complex, and it is difficult to assign their relative values. The following have been put forward by various observers : Deficiency of oxygen in rarefied air ; deficiency of carbonic acid, associated with partial paralysis of the vagus ; diminution in the amount of hemoglobin with lessened capacity for absorbing oxygen ; cerebral and spinal anæmia, resembling that occurring in caisson-disease ; asphyxia associated with cerebral congestion. See CAISSON-DISEASE.

Exhaustion from bad food, from want of training, food, and sleep, or from cold or excessive heat, tend to bring on the malady or increase its severity. Stagnation of the air in a mountain hollow has the same effect, whereas the rapidly moving air on a ridge or summit relieves the distress. Prolonged residence at a high altitude (10,000 to 12,000 feet) does not seem to protect the individual when he reaches a greater height. Personal idiosyncrasy is undoubtedly a factor.

SYMPTOMS.—The following are the symptoms, the more common ones being placed first : Lassitude, weakness and dragging of the legs ; increased and irregular respiration-rate, inability to 'hold the breath' for more than a few seconds ; headache, giddiness, palpitation (the pulse often rising to 120 per minute and becoming irregular), lividity of the face and hands ; loss of appetite, nausea, and occasionally vomiting ; mental hebetude, sleepiness, and even unconsciousness.

DIAGNOSIS.—The effects of ptomaine-poisoning (especially from tinned foods), exhaustion from the heat of the sun or from fatigue, are the conditions most likely to be confused with mountain sickness.

PROGNOSIS.—There is little danger to life provided the sufferer can, if necessary, descend to a lower level. Even if unable to do this, the symptoms will often pass off with rest and suitable food, though liable to return if another attempt be made to ascend.

TREATMENT.—This consists in rest, with the head low, maintenance of the body-warmth, the administration of stimulating and easily digestible food or meat extracts, and, if necessary, descent to a lower level. The use of oxygen carried in steel cylinders has been suggested as a means both of relieving mountain sickness and enabling climbers to reach greater heights when so affected, but the question of portability is obviously a serious one.

W. A. WILLS.

MOUTH, Diseases of.—The principal diseases of the mouth may be thus enumerated : (1) Inflammation and its results, including (1a) Thrush ;

(2) Gumboil ; (3) Ranula ; (4) Lingual Dermoids ; (5) Salivary Calculus ; (6) Salivary Fistula ; and (7) New-growths and Epulis. Diseases of the tongue and the teeth are treated of in other articles.

I. Stomatitis.—SYNON. : Fr. *Stomatite* ; Ger. *Mundschleimhautentzündung*.

DEFINITION.—Inflammation of the mouth.

VARIETIES.—Inflammation of the mouth may be *acute* or *chronic*. According to the more striking lesions found, we speak of *catarrhal*, *follicular*, *ulcerative*, and *gangrenous* stomatitis. Again, inflammations of the buccal mucosa may be classed according to their cause, for example, *syphilitic*, *scorbutic*, or *mercurial* stomatitis.

ÆTIOLOGY.—The principal causes, predisposing or exciting, of stomatitis are : Mechanical irritants such as a sharp tooth or a tooth-plate ; the eruption of teeth ; chemical irritants, especially alcohol ; burns and scalds ; hot tobacco-smoke ; mercurialism ; digestive disturbances ; the growth of parasites in the mucosa, such as those of thrush, cancrum oris, erysipelas, diphtheria ; syphilis and scurvy.

SYMPTOMS.—In *acute catarrhal* forms the mouth is hot ; and the mucosa is more or less red, swollen, tender, and unduly sensitive to heat, salt, spices, and the like. All these changes are least marked upon the dense hard palate and gums. The secretion of mucus is increased, and it may form a tough slimy layer upon the surface, more or less turbid from contained epithelial cells and leucocytes. The excessive redness and raw appearance of the surface are largely due to the rapid shedding of the surface-epithelium ; but in less intense cases, white patches of all shapes and sizes indicate increased production of epithelium, the cells remaining adherent for some time. In the less intense forms, especially when of some duration, the mucous glands on the lips, cheeks, and palate swell and form reddish-grey nodules or even small cysts, often surrounded by a ring of injection. This is the form known as *follicular* stomatitis. Sometimes the follicles break down and form small ulcers. But there are certain forms of stomatitis, of which more or less extensive ulceration is characteristic. One of these is especially known as *ulcerative stomatitis*.

Ulcerative stomatitis (putrid sore mouth) is not a further development of the above-described acute catarrh. It occurs chiefly in sickly children living under faulty hygienic conditions. After a slight febrile illness or malaise for two or three days, the child is noticed to eat with some difficulty, to suffer from more or less salivation, and to have a very foul breath. The cheek is perhaps swollen. On one side of the mouth, sometimes on both, one or more grey-based ulcers will be found upon the cheek, gums, and tongue. These may extend in depth till the alveolus is bare, there being no line between ulcerative stomatitis and cancrum oris (see CANCRUM ORIS). If left alone, these ulcers generally heal in seven to fourteen days, but they may become chronic. Ulcers are very prone to form in stomatitis resulting from mercurialism, scurvy, or syphilis.

In *chronic catarrh* of the mouth the chief signs are undue sensitiveness of the mucosa, and the presence of white patches (leucoplakia). Smooth red patches may also occur. Fissures may form in these patches. This chronic epithelial overgrowth derives its chief importance from the fact that it is sometimes the forerunner of epithelioma. The chief causes of chronic catarrh appear to be smoking,

dram-drinking, indulgence in hot spices, and syphilis; but frequently no cause is evident.

TREATMENT.—The avoidance of irritation, and the treatment of any general disease or digestive trouble, which may be regarded as a cause of the stomatitis, are the first points to be attended to. Food should be taken cool, with little or no salt, sugar, or hot spice; alcohol and smoking should be forbidden. The feeding of infants at the breast and, still more, of bottle-fed children, should be inquired into, and all defects remedied. The mouth should be wiped out after each meal with a rag moistened with some mild antiseptic. Borax-lotion or a little powdered borax, allowed to melt slowly in the mouth, is very soothing. In acute cases ice is grateful.

In *ulcerative stomatitis*, chlorate of potassium should be given internally with due care, and used also as a gargle. It is almost a specific. Borax-lotions are useful; and it is well to begin the treatment by wiping the ulcer and thoroughly applying pure phenol, cocaine or chloroform being used. Finely powdered iodoform should be dusted on the sore once daily. The hygiene of the mouth and of the whole body should be cared for. Change of air is most valuable. Nitric acid or ammonia and bark form an excellent tonic; and cod-liver oil and iron are generally useful later.

In *chronic leucoplakia*, treatment in syphilitic cases should include painting the patches with a lotion of bichloride of mercury (gr. ij–v ad 3j). In simple cases they should be painted with bicarbonate of potassium (gr. xx ad 3j), or chromic acid (gr. v–x ad 3j), the latter solution to be used every second day.

1a. Thrush.—**SYNON.**: Fr. *Muguet*; Ger. *Soor*; *Schwämmchen*.

Thrush is a parasitic stomatitis which occurs chiefly in young infants, and usually in such as are depressed in health, congenital syphilis thus ranking as a predisposing cause. It is not, however, rare to find infants in good health attacked. Adults do not, as a rule, suffer from this parasite, except towards the end of exhausting illnesses, especially phthisis; so the appearance of thrush in adults is usually of evil omen. Rarely a non-parasitic stomatitis or sore-throat depresses the mouth-tissues of adults sufficiently to allow the parasite to grow.

The thrush parasite is the *Oidium albicans*. Grawitz's statement that this is identical with the *Mycoderma vini*, or mould of wine, is generally received as correct. It affects mucosæ covered with stratified epithelium, especially that of the mouth and fauces; it is not uncommon in the pharynx and œsophagus, but is rarely found growing lower in the alimentary tract. It is sometimes found in broncho-pneumonic foci, having been inhaled. The parasite grows in the middle layers of stratified epithelial laminae, and consists of numerous mycelial threads mixed up with spores and fine granular debris. The neighbouring epithelial cells are destroyed and ultimately thrown off, leaving what looks like an ulcer; but almost always the deepest layer of epithelium remains covering the papillæ.

SYMPTOMS.—It is said that the first sign of thrush is some general heat and redness of the mouth, together with the secretion of a sticky layer of mucus of acid reaction, containing many cells, among which spores and threads are already recognisable. Then small white roundish patches form, and run together into larger ones. The patches are sur-

rounded by a vascular ring. At first they are pretty firmly adherent, later on easily detachable, leaving 'ulcers.' Sucking may become painful. More or less disturbance of the gastro-intestinal functions is usually present. Mothers suckling children with thrush are very liable to sore nipples.

If the above be taken as the typical symptoms of thrush, we must add that in many cases the parasite excites no inflammatory phenomena whatever, suggesting a parallel with tinea tonsurans, which also may or may not cause inflammation.

Thrush is commonly said by mothers 'to go through' children. This means that, while thrush is present in the mouth, and the child is perhaps suffering from gastro-intestinal symptoms, redness, excoriation, and perhaps ulceration appear at the anus, and spread more or less widely to adjacent parts. Some think that in the child's depressed condition of health any tendency to intertrigo or eczema would show itself, and that acrid evacuations would render the anal region specially liable; but in the writer's opinion the above history is very strongly in favour of congenital syphilis, and he finds that treatment generally supports this view.

TREATMENT.—Care in feeding children regularly at proper intervals, in the selection of milk and preparation of food for hand-fed children, scrupulous cleanliness with regard to bottles, spoons, &c., used in feeding the child, and especially the precaution of wiping out the child's mouth thoroughly after each meal with rag or wool wet with glycerine of borax, make up a highly successful prophylaxis against thrush. When the trouble is present, all these precautions must be carefully observed, and a food suitable to the patient must be found. Gastro-intestinal symptoms must be relieved. The patches of thrush in the mouth and throat should be frequently painted with the Glycerinum Acidi Borici, the B.P. solution of sulphurous acid (1 in 6 of water), salicylic-acid solution (1 in 250), sulphate-of-sodium solution (3j ad 3j), or almost any other non-irritant antiseptic. Good hygienic surroundings, change of air, and supporting treatment are often necessary to bring about the recovery of feeble patients, and are helpful to all.

2. Gumboil.—**SYNON.**: *Parulis*; Fr. *Parulie*; Ger. *Zahnfleischgeschwür*.

DESCRIPTION.—A 'gumboil' is a small abscess pointing upon the gum. The term 'alveolar abscess' includes all gumboils and many other cases; for, while indicating that such abscesses almost invariably start in the alveolus or socket of a tooth, it says nothing as to their pointing, the directions in which they tend being very various.

The cause of an alveolar abscess is almost invariably caries of the tooth in connection with which the abscess arises. The irritant is therefore most probably some septic organism which gains entrance to the pulp-cavity and excites an inflammation of the pulp, which spreads along a fang. Usually the whole tooth dies as a result. An abscess may form at the base of a tooth already dead, though not carious, the irritant probably entering between the socket and the tooth, but not necessarily, for an abscess is rarely found connected with a sound living tooth, and the pulp of a sound, though dead, tooth may be found putrid. An abscess occasionally arises in connection with a partially erupted tooth, especially a wisdom-tooth in the eruption of which there is difficulty; or, again, a cyst round a retained

tooth may suppurate and resemble an ordinary alveolar abscess.

In the ordinary alveolar abscess pus is formed about the apex of the fang within the alveolus, whence the acute early pain. The bone around the apex of the fang is rapidly softened; and the outer plate of the jaw, being decidedly thinner than the inner, yields, and the pus escapes into the tissues outside the jaw. More or less swelling of the face now occurs, and, coincidentally, diminution of pain. The pus then forces its way towards the surface in the direction of least resistance, and very often bursts on the gum, or between this and the cheek. But this is by no means always the result, the point of bursting being sometimes so distant from the seat of irritation that the latter runs some risk of remaining unrecognised. The following, more or less unusual, points are therefore noted. Abscesses from the upper incisors, especially lateral, not uncommonly burrow between the hard palate and its coverings, and open far back; rarely they burst into the nose, causing a discharge from the nostrils; and still more rarely through the lip. The palatine roots of the upper molars may also cause a palatine abscess; while pus starting from the external fangs is sometimes conducted, apparently along the buccinator fascia, to the cheek, where it bursts. Affection of, and suppuration in, the antrum is most likely to result from the first upper molar fangs; but caries of any upper tooth, especially molar or bicuspid, may be the cause of continued suppuration. Abscess from the lower incisors rarely bursts on or below the chin. Abscess from the lower molars and bicusps, more commonly than from any other teeth, bursts upon the face, just above the lower edge of the mandible. From the lower wisdom-tooth an abscess may burst far back upon the fauces, near a molar or bicuspid, or below the jaw. In a few cases the pus of these latter abscesses has burrowed, from insufficient drainage, down the neck, even as low as the clavicle. Rarely an abscess from a lower molar finds its way through the inner plate of the jaw.

SYMPTOMS.—At first the tooth appears long (from periostitis), and is the seat of pain, which is relieved by pressure upon the tooth. Soon it becomes so tender that all pressure is unendurable, and pain increases rapidly, often becoming very severe. After twenty-four to forty-eight hours the pain diminishes or disappears, and the face swells more or less. Finally the abscess, if left to itself, bursts. It may discharge freely through the opening, and sometimes through and around the tooth. Sometimes it undoubtedly heals, and the tooth remains, firmly fixed; or a sinus remains, giving little trouble if it open into the mouth, much disfigurement if on the face; or frequent abscesses may form. Abscess from a non-erupted or carious wisdom-tooth is specially likely to be accompanied by chronic trismus. Grave results, such as death from pyæmia, are very rare.

TREATMENT.—An acute alveolar inflammation may be aborted by disinfecting the tooth-cavity with pure carbolic acid, scarifying the gum, and applying frequent hot fomentations to the mouth. Where the above treatment fails to relieve the early acute pain (toothache), pus not having yet made its escape from the alveolus, extraction of the tooth (discovered by smart tapping with a steel instrument if the patient cannot clearly indicate which is aching) is the best remedy. When the tooth is

valuable, Tomes has suggested that an incision should be made down to the bone opposite the diseased tooth, and the outer plate of the jaw drilled at the level of the apex of the fang—not a very painful operation, he says. When abscess is present and the tooth of little or no value, removal of the tooth often provides sufficient drainage; but if the patient cannot be easily watched, an incision also should be made into the abscess between the cheek and jaw, especially as, with cocaine, it adds little to the pain. This incision should always be made immediately when the abscess is threatening to burst through the skin; also when pus is stripping up the soft parts from the hard palate. The skin over the pus may then be painted with flexile collodion, and elastic pressure maintained with a bandage over cotton-wool. Thinned red skin may thus often be preserved. When bursting through the skin seems inevitable, it should always be anticipated by a short incision, through which a fine tube may be introduced. The result of this will be a much in-drawn scar, but in the course of months the depression diminishes till it may become hardly noticeable. In making incisions between the jaw and cheek, the points to be attended to are to use a small scalpel, cut close and parallel to the jaw, and direct the point a little towards the bone. The presence of pain and swelling are reasons for the immediate performance of extraction, and not for its postponement till these symptoms shall have subsided. In all chronic sinuses of doubtful nature about the mouth, lower part of the face, or neck—even as low as the clavicle—the teeth should be carefully examined and put in order. It will probably be useless to endeavour to save a tooth which has given rise to chronic sinus or to recurrent abscesses. Even after removal of the tooth and gouging of the alveolus, the sinus may remain for months, but ultimately close. An opening upon the face may in these cases close if a probe be passed along the sinus and freely cut upon from within the mouth. To preserve a tooth, the pulp-cavity is to be emptied and rendered aseptic, and then it and its prolongations must be filled. If the process is successful, the tooth heals in as does a clean foreign body; if it fail, fresh abscesses form and extraction becomes necessary. See **TEETH**, Diseases of.

3. **Ranula.**—**SYNON.**: Fr. *Grenouillette*; Ger. *Ranula*; *Fröscheingeschwulst*.

DESCRIPTION.—Ranula is a cystic swelling situated more or less upon one side of the frænum linguæ, rarely upon both sides; affecting both sexes at all ages, but chiefly occurring in adult life. It is not uncommonly referred to an injury after which it has appeared; it increases slowly, rarely becomes larger than a chestnut, is painless, and causes inconvenience only by its size. The swelling in the mouth is smooth, rounded, tense or soft, elastic or fluctuating, and has a bluish pellucid appearance strongly suggestive of its cystic nature. The mucosa is non-adherent and free from folds over the swelling, across which tortuous veins often wind. Only with ranulæ of exceptional size is any swelling perceptible below the jaw.

PATHOLOGY.—This is doubtful, and several modes of origin have been described. Distension of Wharton's duct may certainly occur, but in the majority of cases this duct is clear, and a probe introduced passes over the swelling. Wound of the duct and extravasation of saliva may cause the

appearance of a cystic swelling. Some believe that the sublingual gland and its principal duct (Bartholin's) are the chief seats of ranula, but this is unlikely. Fleischmann's bursa—on each side of the frænum, between the mucosa and the muscles—is probably sometimes the seat of a pathological effusion. Dilatation of a mucous gland may occur beneath the tongue, especially that known as the Blandin-Nühn gland from the names of its describers. This was found adherent to the cyst by von Recklinghausen and Sonnenberg in one instance, and they adduced many good reasons for thinking that this gland was, at least, a very common seat of the cystic distension known as ranula.

TREATMENT.—A thick silk seton may be tied and remain in for three to seven days, according to the degree of irritation excited; this may be serious, and run on to abscess. Other methods of treatment are (a) opening the cyst freely, drying it out, applying pure carbolic acid, and packing with iodoform gauze; (b) cutting a V-shaped flap in the wall, and fixing its apex into the cavity with a stitch; (c) dissecting out the whole cyst, or the superficial part of it, upon which the Blandin-Nühn gland would lie (von Recklinghausen). Woakes reports a cure from the injection of a saturated watery solution of chromic acid.

4. **Lingual Dermoids.**—**DESCRIPTION.**—Lingual dermoids occur in two situations—in the mid-line between the genioglossi, and on either side of the tongue, between the genioglossus and mylohyoid. They are rare, and are due to the inclusion of an epithelial germ connected either with the invagination of the mouth-pit or stomodæum, the closure of the first visceral arch or of the first branchial cleft, or with the foetal thyro-glossal duct (His), which extends from the foramen cæcum of the tongue to the thyroid isthmus, and is regarded as the remains of the hypopharyngeal diverticulum or protrusion from the foregut to form the thyroid isthmus. Cysts between the genioglossi and in the mid-line of the neck, as low as the thyroid isthmus, are probably connected with the thyro-glossal duct, or with the union in the mid-line of the upper branchial arches; cysts on either side of the mid-line with closure of the branchial clefts or with the junction of the stomodæum and foregut. The contents vary, being sometimes the usual cheesy, epithelial mass, with or without pale hairs intermixed; sometimes only a brownish mucoid fluid, in which float small masses of fatty epithelium. The lining of the cyst is generally a delicate layer of stratified cells which vary somewhat in function, as the contents show.

Though really congenital in origin, and usually reaching a size to attract notice in early life, these cysts often remain small for many years, and then, without obvious cause, begin to enlarge. They may not appear until mid-life or even later. They form smooth, round or oval, tense, elastic and fluctuating, or doughy swellings beneath the tongue, in the mid-line or on one side; they are usually firmer to the touch than ranulæ; unlike ranulæ they project chiefly towards the skin and but slightly into the mouth, the mucosa over them being normal unless some dilated veins ramify in it; they never have the translucent appearance of ranulæ, but it is said that some have a yellowish appearance owing to the colour of the sebaceous matter showing through. They cause inconvenience in proportion to their size.

TREATMENT.—This consists of complete removal by dissection, when inconvenience or unsightliness renders treatment desirable. All central and most lateral cysts should be removed by incision below the jaw; the cyst, when bared, is but loosely attached to its surroundings, and it may, if necessary, be emptied. The cavity left should be obliterated by purse-string stitches, and the skin wound closed.

5. **Salivary Calculus.**—**SYNON.** : Fr. *Calcul Salivaire*; Ger. *Speichelstein*.

DESCRIPTION.—Friable concretions, composed chiefly of phosphate of lime, are not very uncommon in the ducts of the parotid, submaxillary, and sublingual glands. They vary in size from a pin's head to a filbert, or even larger, are elongated in form, and not infrequently they form around some small foreign body—a seed or a bit of woody fibre—which has made its way into the duct. They may lie in the substance of the gland, but are usually found in the duct. They are easily felt from the mouth, but are rarely complained of unless they give rise to the following symptoms. Without obvious cause inflammation causes complete or partial obstruction of the duct; the gland swells and becomes hard, but is neither tender nor painful as a rule. In the case of the submaxillary duct, the side of the tongue and floor of the mouth are red, and more or less swollen; an abscess may form in the latter situation. *See* CONCRETIONS.

TREATMENT.—If a concretion can be felt, either with a finger or with a probe passed along the duct, an incision should be made on to it, and the calculus removed with care, lest it break and the fragments excite more inflammation than did the entire mass. Local inflammation requires a poultice outside, and constant fomentation of the mouth with hot solution of boric acid or borax; if an abscess form it should be opened and treated similarly.

6. **Salivary Fistula.**—**SYNON.** : Fr. *Fistule Salivaire*; Ger. *Speichelfistel*.

DESCRIPTION.—Occasionally the duct of the parotid gland (Stensen's duct) is wounded or involved in an ulceration, or an abscess forms in its track and bursts externally. In such cases a salivary fistula is likely to be the result. The secretion from the parotid instead of making its way into the mouth dribbles over the cheek.

TREATMENT.—Whenever the parotid duct is involved in a wound of the cheek, an opening opposite the wound should be made into the mouth; the duct should be most carefully sutured, as also should be the tissues superficial to the duct. When a fistula has formed, all ulceration should have ceased for weeks or months before any operation is undertaken for its closure. Then the first point is to secure free drainage into the mouth for saliva by puncturing the mucosa of the cheek in two places from the depth of the fistula, and passing a stout bit of silver wire through the holes and tying it in the mouth as a ring. The closure of the opening in the skin has been effected by light cauterisations; by paring the edges, dissecting up skin-flaps for some distance and bringing them together; or, best of all, by removing the edges and adjacent skin and twisting in a flap to fill the gap. All movement of the cheek should be avoided.

7. **New-Growths.**—Most of these start from the tongue or jaws. Papillomata and epitheliomata are not uncommon on the inner surface of the cheek, palate, and floor of the mouth; they present their

usual characteristics, and are treated by removal. The swollen mucosa round a sinus, leading to a sequestrum of the hard palate, sometimes presents a striking resemblance to an epithelioma.

Epulis.—SYNON.: Fr. *Epulide*; Ger. *Epulis*.

DESCRIPTION.—‘*Epulis*’ is a somewhat loosely used word applied to tumours projecting upon the gum. Without an adjective indicating the nature of the growth, it really conveys nothing beyond the situation of the mass; but custom has practically limited its application to two common and often clinically indistinguishable pathological varieties, the fibrous and the myeloid.

These tumours spring from the fibrous tissues of the gum, or from the alveolar periosteum and bone—the myeloid grows always, and the fibrous usually, having a connection with bone. Sometimes small growths of the latter nature come away attached to the fang of an extracted tooth, as if they had originated from the fibrous covering of the fang.

Epulides are commoner in connection with the lower than with the upper jaw. They form smooth, rounded, or lobulated masses, usually quite sessile, varying from pinkish-white through red to purple-red in colour, and firm or semi-elastic to the touch. They are covered at first by the epithelium and mucosa of the gum, but ultimately they may ulcerate from pressure against the teeth or other irritation. Most commonly they present between two teeth, displacing one or more; or they may project upon either the superficial or the deep surface of the alveolus—the latter but rarely. Growth is not very rapid, and there are no signs of deep infiltration of the jaw.

TREATMENT.—This consists in free removal of the growth, together with any bone in connection with it. Sometimes this may be effected under cocaine, especially with growths on the superficial surface of the gum; in other cases an anæsthetic is necessary, two or three teeth must be extracted, and the whole thickness of the alveolus cut away with a small saw and bone-forceps. After such treatment, recurrence is unusual; but myeloid growths sometimes show exceptional malignancy, and require specially free handling.

STANLEY BOYD.

MOVABLE KIDNEY.—See KIDNEYS, Diseases of, p. 835.

MOVEMENT, Therapeutical Uses of.—SYNON.: Movement-Cure; Kinesitherapeutics; Fr. *Gymnastique Suédoise*; Ger. *Kinesitherapie*.

DESCRIPTION.—The movements employed are of three classes, namely: (1) *active movements*, executed by the patient himself, or by the patient aided by an assistant; (2) *passive movements*, performed by the assistant on the patient; and (3) *acts of resistance to movements*, whether executed by the assistant against the patient, or by the patient against the assistant.

USES.—The several classes of movements, for which mechanical arrangements are also contrived, when scientifically employed, are used in the treatment of paralysis, curvatures of the spine or limbs, and injuries and diseases of the joints. Movements of the nature of friction or shampooing are also employed in the treatment of certain diseases of internal organs. See FRICTION; MASSAGE; and SHAMPOOING.

MUCOID DEGENERATION.—A form of degeneration, which is associated with the production of a mucus-like substance. See DEGENERATION.

MUCOUS PATCH.—SYNON.: Mucous Tubercle; Condyloma; Fr. *Plaque Muqueuse*; Ger. *Breite Feigwarze*.

When the term ‘condyloma’ is used, ‘syphilitic’ ought always to be prefixed, to avoid confusion with the simple form of growth. See CONDYLOMA.

DEFINITION.—A patch of syphilitic papular eruption upon mucous membrane or moist skin.

ÆTIOLOGY.—Mucous patches belong to what are commonly known as the secondary manifestations of syphilis; they may appear early or late, both in the acquired and in the inherited disease, and are very liable to return time after time.

Experimental inoculation of the discharge of mucous patches has proved that it is capable of producing a hard sore at the point of insertion, followed by general symptoms; and clinical observation shows that these lesions are the most frequent means of spreading syphilis.

DESCRIPTION.—Mucous patches on the skin appear as flattened elevations of a round or oval shape, with a broad base, of a reddish colour, and generally covered by a thin grey pellicle. When in close proximity they coalesce, and form a mass of irregular shape and size, which may be fissured and ulcerated. When situated upon a mucous membrane they are usually less raised, and whitish in colour, especially in the throat, where they have been termed *plaques opalines*. The primary sore may assume the appearance of a mucous patch, especially in women. The growths also sometimes become warty on the surface.

Favourite seats of mucous patches are the genital organs, and the moist skin about the anus. They may also be found at the umbilicus, in the axillæ, auditory meatus, or nose, on the nipples, and between the toes; in stout and dirty persons they may be met with wherever folds of skin meet and perspiration collects. They are very common about the lips, mouth, tongue, and throat; and occasionally are seen on the cervix uteri. Want of cleanliness favours their development, as does irritation from any cause, especially smoking and chewing tobacco.

TREATMENT.—Mucous patches, being highly infectious, should always be got rid of as soon as possible. In the mouth or throat, pure carbolic acid, or a solution of chromic acid (twenty grains to the ounce), may be applied from time to time, and an astringent mouth-wash used several times a day. Mucous patches on external parts usually disappear quickly under strict cleanliness and the application of calomel (one part) and oxide of zinc (three parts), care being taken to keep opposed surfaces apart with lint or wool. If the growths persist, carbolic acid or nitrate of silver or the acid solution of nitrate of mercury should be applied. General treatment must of course be carried out at the same time. See SYPHILIS.

ARTHUR COOPER.

MUCOUS RALE.—An adventitious sound heard on auscultating the chest in certain forms of disease, and due to the passage of air through viscid fluid in the bronchi. See PHYSICAL EXAMINATION.

MUCOUS TUBERCLES.—See MUCOUS PATCH.

MULTILOCULAR (*multus*, many; and *loculus*, a small space).—A term applied to cysts and other

forms of growths, and to pulmonary cavities, when they consist of many small spaces or loculi. See CYSTS.

MULTIPLE NEURITIS.—A synonym for peripheral neuritis. See NEURITIS, MULTIPLE.

MUMPS.—SYNON. : Epidemic Parotitis; Cynanche Parotidea; Fr. *Oreillons*; Ger. *Mumps*.

DEFINITION.—An acute, febrile, infectious disease; attended by swelling of the salivary glands—especially of the parotids; and ending in resolution.

ÆTIOLOGY.—This is an affection more commonly seen in young persons—boys, growing girls, and young men; but it may occur in adults of either sex who are much with those affected with the disease, and who have not had the complaint before. Mumps rarely attacks the same person twice. It may occur as an epidemic in institutions, such as schools and barracks. It is usually conveyed directly from person to person, and is infectious even before the glands are affected, as well as for two or three weeks afterwards. It has an incubation-period of from fourteen days to three weeks.

ANATOMICAL CHARACTERS.—The disease is occasionally limited to one parotid gland, but generally spreads to submaxillary and sublingual on the same side, and not infrequently involves all six salivary glands as well as some of the cervical lymph-glands. It is probable that the inflammation starts from the duct. The surrounding tissues are swollen. Seldom does there appear to be any fibrinous exudation poured out; and still less frequently do the tissues suppurate, although the inflammation may spread to the middle or internal ear. The swelling completely disappears about three days after the fever. On the subsidence of the local lesion a so-called metastasis to the testicle and other glandular and fibrous structures may occur; atrophy of the affected testicles may follow. The sheaths of the nerves and the surfaces of the heart may be affected, especially in those cases in which orchitis has occurred.

SYMPTOMS AND DIAGNOSIS.—Some general symptoms always precede the local manifestations of mumps; they may be so slight as almost to escape notice; or fatigue by day, restlessness at night, slight fever, sore-throat, and vomiting may mark the prodromal stage. After these symptoms have persisted for a few hours or days the fever rises rapidly and the pain and swelling in the parotid or submaxillary glands begin.

One restless night follows, either from pain, or from fever, or both. Sometimes the pain is severe, and the temperature only elevated by one degree; sometimes the fever is more evident. It generally reaches 100° F. or 101°, and frequently rises to 103° or 104°; at this point it is not long maintained, but subsides as the local lesion is established, falling to the normal, or even below it, on the third or fourth day of the disease. The temperature may be low while the swelling is still marked and painful; and in some cases appetite returns before eating is easy. This happens when the patient is kept at rest in bed. Without such precaution, sudden and great elevations of temperature may occur at the end of the first week, either without serious local mischief, or with orchitis, deafness, tinnitus of one ear, and albuminuria, not always transient; rheumatism, and heart-

affections, leaving traces both of pericardial and of endocardial inflammation, may also occur.

From face-ache and enlarged lymphatic glands, the sudden sensation of pain or stiffness in the parotid or submaxillary gland, following on the general symptoms, and absence of any such local trouble as usually affects the lymphatics, together with the history of a possible infection, will generally suffice for the diagnosis of mumps. Further evidence is obtained on examining the spot, where, besides the deeply seated swelling, considerable tumefaction of the parts surrounding the gland exists near the lobe of the ear, filling in the hollow behind the jaw, soon increasing to such an extent as to involve more or less the whole of one side of the face, and passing down on to the neck. If the sublingual glands are affected the tongue is raised. Coincidentally with the appearance of this enlargement, the pyrexia declines in some cases; while in others some days elapse before the subsidence of the fever. Pain is now complained of, and the patient can no longer open his mouth to the usual extent. Yawning excites severe pain; in fact, it can hardly be effected. Mastication and speaking are greatly impaired, and the sufferer prefers to fast, and to remain silent, rather than endure the pain involved in the effort to perform either act. The saliva may be largely increased or much diminished in quantity. Pressure over the swelling is painful; and the sensation afforded by manipulation is that of an elastic tumour, with a slightly softer feeling in the centre. The skin over the swelling may be slightly reddened; often there is no deviation from the normal colour, the skin remaining pale, but glossy, and oedematous. In many cases these symptoms are not nearly so severe, and the disproportion between the amount of distortion of the countenance and the actual suffering is as astonishing to the patient as to the friends. Most frequently the affection is limited to one side of the face; but as the swelling of the one side subsides, the other may become involved, when it runs through the same series of events, with, possibly, an interval of a few days between them. Rarely are the two sides simultaneously affected; but in such a case the uneasiness, pain, and discomfort are of course greatly increased. After the continuance of these symptoms for about six or eight days, they begin to abate, the oedema lessens, the pain is lost, the stiffness and tension disappear, and in a few days the face acquires its usual appearance. A mild uncomplicated case endures about a fortnight. Occasionally there is left, for some time after this, a certain degree of hardness in the neighbourhood of the parotid, which gives no uneasiness, and can rarely be mistaken for tumour.

COMPLICATIONS.—In nearly a third of the cases in adult males orchitis occurs. The epididymis is occasionally involved. In females, ovaritis, vulvitis, or mastitis may be found, but they are rare. Cases are described in which the testicle was affected while the salivary glands escaped. Orchitis may precede, or accompany, but usually follows the swelling of the parotid, at an interval of not more than three weeks. Increase of fever and the usual local signs occur. Rarely is the orchitis bilateral. In the case of the girl the vulva becomes the seat of oedema, and on pressure over the region of the ovary pain is elicited. When the orchitis abates, the parotid may again take on the inflammatory condition. Meningitis is to be feared on sudden subsidence of the inflammation of the parotid, if

no orchitis follow the disappearance of the original affection. If the middle or internal ear has been involved permanent deafness may supervene on the affected side. Delirium, amblyopia, conjunctivitis, albuminuria, or gastro-intestinal disturbances are occasional complications.

PROGNOSIS.—This is almost invariably favourable in mumps, except in the very weakly and in the tuberculous, or in the rare event of meningitis being developed. It may be said to be always a disease of a comparatively trivial nature, producing considerable pain and much discomfort, but not endangering the life of the sufferer. In very exceptional instances the inflammation of the parotid terminates in abscess. The indications of such an untoward result are increased pain in the centre of the swelling, hardness, and dark red appearance of the skin over the spot. In time the abscess discharges outwardly, or into the external auditory meatus. Atrophy of the testis follows orchitis in 70 per cent. of the cases (Laveran).

TREATMENT.—A patient suffering from mumps should be confined to his bed during the febrile stage. But little treatment, beyond rest and care for the week or ten days the disease lasts, is required. The bowels may require relief, but all strong purgatives should be avoided. It may be well to give some simple saline, as bicarbonate of potassium with lemon juice, and diluents during the first few days; ice is always grateful. A dose of chloral hydrate may be required at night (in children a grain for each year of the patient's age) if there be any restlessness.

As to local treatment, not much is required, unless the pain be unusually severe. It will be sufficient in most cases to protect the part from the air by means of cotton-wool and a light handkerchief. Should more active interference be called for, some anodyne may be used, or soothing embrocation, such as the opium liniment, belladonna liniment, or external warmth. If there be the slightest tendency to suppuration, indicated by increase of fever and tenderness over the gland, with redness of the overlying skin, fomentations must be applied, and so soon as distinct fluctuation is discovered the abscess must be opened. The application of leeches is useless in reducing the inflammation, or in staying the formation of the abscess. They may be of service in lessening the pain of metastatic orchitis or ovaritis; but these are well treated by the same gentle means employed in the case of the parotid itself. Tepid sponging is of use during the course of the disease, and a warm bath may be required when metastasis threatens. Sometimes wine or brandy is required.

Considerable anæmia and much debility may persist even when mumps has been mild in its course, especially in the weakly or unhealthy, so that tonics, with iron and cod-liver oil, may have to be continued for some time. C. MUIRHEAD.

MÜNSTER AM STEIN, near Kreuznach, in Rhenish Prussia.—Muriated saline waters. See MINERAL WATERS.

MURMUR.—This term, as used in auscultation, was originally applied to the natural sounds heard over the lungs in respiration; but its employment has since been extended to include a great variety of auscultatory sounds connected with the heart, the blood-vessels, the placenta, &c. See PHYSICAL EXAMINATION.

MUSCÆ VOLITANTES (*musca*, a fly; *volitans*, floating about).—See EYE, AND ITS APPENDAGES, Diseases of.

MUSCLES, Diseases of.—SYNON.: Fr. *Maladies des Muscles*; Ger. *Muskelkrankheiten*.

1. **Acute Inflammation.**—SYNON.: Myositis.

(a) Inflammation of muscle, leading to exudation and suppuration, arises chiefly as a result of injury or rupture of a muscle. Sometimes, however, it appears to arise spontaneously, particularly in the tongue, diaphragm, and psoas muscle; in the last situation forming one variety of psoas abscess. In such cases it is probable that micro-organisms have been carried to the part by the blood or lymph. The symptoms are pain, tenderness and swelling, corresponding to the seat of the inflammation. Exudation of serum and of lymph takes place, and subsequently an abscess may form; occasionally the process goes on to gangrene.

(b) Secondary inflammations and formations of pus are of more frequent occurrence than simple inflammation and abscess. They arise in the course of the various forms of pyæmia and of the acute specific fevers, or may be due to extension of inflammation from neighbouring diseased bone. The presence of such secondary abscesses in muscles is especially characteristic of glanders and farcy, where inflammatory infiltrations of various sizes appear in many of the muscles, especially those of the arm. Disintegration takes place in their centre, and a collection of puriform fluid results.

(c) *Polymyositis* (Dermato-myositis).—This rare affection is characterised by swelling of the muscles in different parts of the body, with oedema of the subcutaneous tissues and an erythematous rash in the affected regions. The disease is probably toxic in nature, some cases having been definitely connected with the eating of shell-fish. The onset is gradual, with fever and feeling of weakness. Later there is acute pain in the muscles, generally those of the extremities first; later the muscles of respiration and deglutition may be involved. Death is frequent in severe cases. Microscopically, the affected muscles show hyaline degeneration with interstitial infiltration of leucocytes. The condition has to be distinguished from trichinosis, in which there are digestive disturbances and more affection of the face. The parasites must be sought for to render diagnosis certain (see p. 465). Treatment must be directed to the relief of pain and maintenance of the patient's strength. The use of hydragogue purgatives may aid in the elimination of the poison.

2. **Chronic Indurating Inflammation and Syphilitic Disease.**—In this form of inflammation there is proliferation of cells in the interstitial tissue, causing the muscle to become hard and painful. The whole muscle may be attacked, or the process may be limited to one or more portions. Infants are often attacked by chronic inflammation of the sterno-mastoid muscle. The whole muscle becomes hard and painful, but rarely suppurates. The disease usually yields to soothing external applications; but if it be of syphilitic origin, the use of internal antisiphilitic remedies may be required. In adults chronic indurative myositis of a syphilitic character may occur in the sterno-mastoid, the various muscles of the leg and arm, the temporal and masseter muscles, the tongue, and other parts. The disease may appear either as a diffuse

inflammation, with the usual signs of pain on movement, tenderness and some swelling—or sometimes a series of beaded swellings; or as a circumscribed inflammation, with an abundant infiltration of nucleated cells. If the inflammation does not soon subside, the cellular exudation becomes organised into contracting fibrous tissue, and the compressed muscular fibres atrophy. In diffuse myositis permanent contraction of the muscle may result from this cause; in circumscribed syphilitic myositis a fibrous tumour in the interior of the muscle may result; sometimes a gummy tumour is formed. Syphilitic tumours thus formed in the muscle bear a great resemblance to malignant tumours. Indeed, it is often found that the only means of distinguishing the two clinically is by the effect of iodide of potassium in causing the disappearance of the former.

3. **Myositis Ossificans.**—Ossification of muscle is a rare result of chronic inflammation or irritation. It is observed to occur in muscles which are subject to pressure, as the deltoid in soldiers, and the adductors of the thighs in riders. In the condition known as Myositis ossificans there takes place a slow, gradually advancing fibrosis of the voluntary muscles, followed in many places by the formation of some bone. The disease may begin in childhood, and its onset is sometimes attributed to injury. The muscles of the back, at first tender, gradually become stiff, so that no flexion is possible: the limbs sometimes become rigid. In course of many years the patient is reduced to a condition resembling a bronze statue, his whole body being rigid and immovable. The cause of the disease is unknown. It appears natural to regard it as a chronic inflammatory process with subsequent conversion of the fibrous tissue into bone. No treatment is known which will influence the course of the malady. The diaphragm, heart, and facial muscles usually escape.

4. **Rheumatic Inflammation.**—The morbid changes in this form of inflammation rarely pass beyond the stage of congestion and serous exudation, though occasionally proliferation of the interstitial tissue may occur, and callosities may be formed. See RHEUMATISM, MUSCULAR.

5. **Hæmorrhage.**—Hæmorrhage takes place in muscle not only from injury, but frequently in the course of typhus and typhoid fevers and pyæmia; also in leucocythæmia.

6. **Rupture.**—Violent contraction of a muscle, without external injury, may lead to partial rupture of its fibres—for example, the gastrocnemius. The violent spasms of tetanus occasionally cause complete rupture of a muscle, particularly of the muscles of the back, the rectus femoris, and the psoas. Rupture of muscles has been known to occur in the delirium of fever; and may be the cause of abscess forming in muscle, as described above.

TREATMENT.—The treatment of ruptured muscle consists mainly in rest; in the support of the muscle by uniform bandaging; and in suitable measures, should abscess form.

7. **Lesions of Sensibility.**—(a) *Myalgia.*—This term is given to a painful condition of the muscles arising in those who are in feeble health. The pain is similar to that which is present in a muscle after long-continued and fatiguing exertion—for example, in the limbs after a long walk, or in the diaphragm and intercostals after violent laughing. In persons who are debilitated, pain may

arise in the muscles after very slight exertion, and this constitutes myalgia. It is often accompanied by cramps at intervals. The pain is most commonly felt at the tendinous insertion of the muscle. The abdominal muscles are frequently the seat of myalgia, such as the costal origin of the external oblique—causing, according to some authorities, that pain in the side which is so common in women—and the pubic insertion of the recti. The muscles of the back, and especially the trapezius, also suffer; the muscles of the limbs much less frequently. When situated in the trunk, myalgia is often mistaken for some congestive or inflammatory condition of the liver, spleen, or other viscus lying beneath. The pains of myalgia are distinguished by their hot and burning character. They are increased by exercise of the affected muscle, and disappear when it is relaxed or artificially supported. However severe the pain may be, the pulse remains unaffected; but it is usually uniformly weak and fast.

The muscles or their fibrous connections are also the seat of pain in the condition known as muscular rheumatism.

TREATMENT.—The muscles should have rest and support by bandaging. Tonic treatment is required. Friction and counter-irritation do little good. Exercise is of no use, unless combined with fresh air and good diet.

(b) *Muscular anesthesia.*—See MUSCULAR SENSE, Disorders of.

8. **Spasm or Cramp.**—This painful affection is too well known to need description. It occurs chiefly in the muscles of the calf, often in persons who are quite healthy. It is most frequent after exertion, and may then be due to an auto-intoxication. Cramps are also a prominent feature in cholera and in certain forms of poisoning, especially by metallic irritants. Treatment in ordinary cases is by forcibly stretching the muscle and maintaining it thus for a few minutes. Kneading the muscle may aid in affording relief.

9. **Atrophy and Degenerations.**—(a) *Simple atrophy.*—Simple atrophy of the substance of muscular fibres arises either from general defective nutrition, during the course of wasting diseases, such as phthisis, in cachectic conditions, or after severe fevers; or as a local condition from disuse of the muscle. The muscles become pale and flabby. The ultimate fibres are reduced in volume, but preserve their anatomical characters, still showing the longitudinal and transverse striation. The atrophy is sometimes so advanced in parts that the muscular substance of the fibre entirely disappears, and nothing is left but the sheath of the sarcolemma, which appears in the form of fibrous bands between the remaining muscular fibres.

As a local condition, atrophy is most frequently seen in muscles in the neighbourhood of a diseased joint, or in a paralysed limb. In these cases the atrophy is usually combined with more or less interstitial deposit of fat between the ultimate fibres, constituting *fatty infiltration* of muscle. Occasionally the amount of fat is so great as to cause an actual increase in bulk of the muscle, so that it appears hypertrophied. The atrophic and other changes arising in paralysed muscles are considered in their appropriate articles. Fatty infiltration of muscles may also arise as a primary condition, when there is an excess of fat in the blood, and atrophy of the muscular substance results from it.

(b) *Fatty degeneration*.—Here the fat is deposited, not between the ultimate fibres, as in fatty infiltration of muscle, but in their interior. Rows of minute granules appear in the longitudinal striæ, and gradually increase until the whole breadth of the fibre is occupied by them, and nothing is left but the sarcolemma. When the degeneration reaches this extent, it is of course irrecoverable. Muscles affected by this change become very soft and friable. This degeneration is met with much oftener in the heart than in voluntary muscles. It is sometimes associated with atrophy of the fibres in the muscles of limbs attacked by certain forms of paralysis. It is met with also in fever and phosphorus-poisoning, granular degeneration being the first stage. *See FATTY DEGENERATION*.

(c) *Granular degeneration*.—Granular degeneration of muscles occurs in fevers and acute diseases. The fibres become swollen and opaque, being filled with fine granules. These clear up on the addition of acetic acid; this test distinguishing granular from fatty degeneration. The muscles which are affected by it are soft and friable and easily rupture. The fibres no doubt ultimately recover their natural appearances; but if the disease be severe and long-continued, granular degeneration advances to fatty degeneration, as is seen in cases of phosphorus-poisoning.

(d) *Waxy degeneration; Zenker's degeneration; Myositis typhosa*.—The affected fibres swell and lose their striation; and become of a homogeneous, translucent aspect. After a time transverse fractures appear in each fibre, dividing it into a series of short cylinders. The nuclei of the sarcolemma also multiply. The change does not attack all the muscular fibres of a part uniformly; for healthy and degenerated fibres are seen side by side. It is observed chiefly in typhoid fever, cholera, and other acute febrile diseases, being often associated with the granular degeneration. It is usually found in the adductor muscles of the thigh, the abdominal and pectoral muscles, and the diaphragm; appearing in patches of one or more square inches, pale and glassy; gradually becoming softened and pulpy. It is now considered to be a condition of *coagulative necrosis* of the muscle. It is frequently associated with hæmorrhage into the substance of the muscle.

(e) *Fibroid degeneration*.—Fibroid degeneration of muscle has already been referred to as a result of myositis. Chronic or repeated inflammation, of a rheumatic or syphilitic character, leads to the formation of fibrous tissue in muscle, and the muscle becomes of a tough, whitish character.

10. *Tumours*.—Muscle is subject to growths of a sarcomatous and cancerous nature, the latter being necessarily secondary to tumours elsewhere. Fatty, cartilaginous, vascular and other tumours are also met with in this tissue, but rarely. Myomata, or tumours consisting of muscular tissue, are also found. *See TUMOURS*.

11. *Parasitic Affections*.—The chief disease of muscles belonging to this group is that due to the presence of *trichine* (*see* ENTOZOA). The *Cysticercus cellulosus* is also sometimes found in muscles. *See also* MUSCULAR SENSE, Disorders of; and MYOPATHY.

ALEXANDER DAVIDSON.
W. CECIL BOSANQUET.

MUSCULAR ATROPHY, PROGRESSIVE.—*See* PROGRESSIVE MUSCULAR ATROPHY.

MUSCULAR DYSTROPHY.—*See* MYOPATHY.

MUSCULAR RHEUMATISM.—A form of rheumatism affecting the muscles. *See* RHEUMATISM, MUSCULAR.

MUSCULAR SENSE, Disorders of.—By the term 'Muscular Sense' is meant the sensory impressions by which we are aware of the state of the muscles during active or passive movements. The degree of muscular sensibility is estimated by the appreciation of the difference between weights held so as to cause but little difference in the stimulation of the cutaneous nerves; and also by the perception of posture, active or especially passive, so produced as to cause an equal stimulation of the cutaneous nerves in the different positions. It is probable that the knowledge is due to impressions from afferent nerves which arise in the connective tissue between the fibres, especially in the muscle-spindles. They seem to be normally stimulated, either by compression, as when the fibres are widened by contraction; or by tension, as when the muscles are elongated. It is only when such stimulation is great that the impressions so affect consciousness as to cause a true sensation, as in forcible extension of a muscle or very strong contraction. When very intense, or when the nerves are hypersensitive, actual pain is felt, as in a strong pinch, or cramp, or extension after cramp. Such pain may be produced through the same nerves, since we are familiar elsewhere with pain as the result of irritation of visceral nerves, whose normal impressions do not directly affect consciousness. The fibres apparently course with the motor fibres in the mixed nerves, but pass to the spinal cord in the posterior roots. The impulses thus generated seem to act first on the spinal cord, then on the cerebellum, and ultimately on the motor cortex of the brain.

HYPERÆSTHESIA.—Increase of the common sensibility of muscles is not unusual, e.g. after cramp; and the muscles become extremely tender in peripheral neuritis such as is caused by alcohol. The tenderness is shown both to pressure and extension. Very little is known of simple increase of the muscular sense except in the form of pain. The sensation of restlessness impelling movement has been attributed to muscular hyperæsthesia, but without sufficient reason.

ANÆSTHESIA.—Diminution of the muscular sensibility is occasionally observed, commonly in consequence of disease of the nerves, sometimes of the nerve-centres, and is usually associated with a diminution of other forms of sensibility. Loss of muscular sense, however, may be present when cutaneous sensibility is unimpaired. The affections with which muscular anæsthesia is commonly observed are locomotor ataxy, some central diseases, and hysteria. In tabes it is probably the cause of the ataxy; it bears no necessary relation to the change in cutaneous sensibility.

SYMPTOMS.—In muscular anæsthesia the patient is unaware of the degree of force exerted by the contracting muscles, and is dependent for his knowledge of the position of his limb, and of its movements, mainly upon cutaneous impressions, if these are preserved. Ignorance of the degree of contraction interferes with muscular co-ordination, by rendering this dependent on cutaneous and ocular perceptions. The condition of the muscular sense is ascertained by observing the accuracy of movement with and without closure of the eyes, by

determining the extent to which the patient is aware of the posture of the limbs in passive movements, and especially by ascertaining the sensitiveness to movement against resistance so applied as to affect the cutaneous nerves as little as possible. The best method for this purpose is to suspend a weight, in a bag or cloth, to the limb, and observe (a) the minimum which can be recognised; and (b) the least increase in a greater weight which can be distinctly perceived. The sensibility of the two limbs may be conveniently compared. In each of these points the muscular sense may present a deviation from the normal, and the change in the two is not always proportioned. The minimum recognisable, and the minimum difference recognisable, vary in different parts. The latter amounts in the case of the arm in health to a difference of $\frac{1}{10}$ th in a weight of three or four pounds. Balls of similar size and appearance, but of different weights, have been employed for the same purpose.

When the sensory muscle-nerves are gravely impaired, as in tabes, the loss of sensibility may be so great that no degree of stimulation will succeed in causing pain. In a case in which inco-ordination was much greater in one leg, forced extension of the calf-muscles in this leg, though unfelt, caused pain in the other leg.

Cases of a mysterious nature are met with in which with normal cutaneous sensation the muscular perceptions are suddenly so much lessened that, as one patient said, 'a poker and a feather seem of equal weight.' Such cases occur in subjects likely to suffer from functional derangements.

TREATMENT.—Muscular anæsthesia usually occurs as part of a wider affection, as in tabes and hysteria, and rarely requires special treatment.

Faradisation of the muscles is the local treatment most useful. It effects powerful stimulation of the afferent muscle-nerves. For the most part the treatment needed is that of the cause. See NERVOUS SYSTEM, Physical Examination of.

W. R. GOWERS.

MUSCULAR TIC.—A synonym for facial spasm. See FACIAL SPASM.

MUSHROOMS, Poisoning by.—SYNON. : Fr. *Empoisonnement par les Champignons*; Ger. *Pilzvergiftung*.

Poisoning by mushrooms is not a very common occurrence. The varied toxic symptoms produced by the ingestion of mushrooms become more easily explicable when we bear in mind that only a few fungi are apparently poisonous under all conditions. They are *Amanita muscaria*, the fly-fungus, which grows not very plentifully in Great Britain, and contains a poisonous alkaloid (*muscarine*); *Russula integra* seu *emetica* (*Agaricus integer* seu *emeticus*), also not very common; *Boletus luridus* (*B. perniciosis*, *B. bovinus*); and *Amanita phalloides* (*A. bulbosa*, *A. venenosa*, *A. viridis*), to which belong the varieties termed *Agaricus citrinus* and *Agaricus virescens*. These owe their toxic properties to *phallin*, a toxalbumen. Other fungi are poisonous only under special conditions, among which may be named *idiosyncrasy*, and the susceptibility of young children to the toxic effects of mushrooms. The delicious edible morel even has been known to produce fatal results. It must not be forgotten that gastro-intestinal catarrh of a severe character may result from the ingestion of a large quantity of ill-cooked indigestible fungus-tissue; that the highly

nitrogenous tissue of fungi is peculiarly prone to rapid decomposition; and that fungi as a class absorb excretory animal matters, perhaps unchanged. These circumstances may serve to explain some of the apparent anomalies connected with mushroom-poisoning. Some kinds of poisonous mushrooms have their active principle either dissipated or destroyed by the prolonged heat employed in thorough cooking.

ANATOMICAL CHARACTERS.—Evidence of gastro-intestinal catarrh, more prominent in the stomach than in the intestines; signs of cardiac paralysis, or of asphyxia; occasionally fatty degeneration of the liver and other viscera; and minute sub-serous extravasations of blood, have all been noted after death from mushroom-poisoning.

SYMPTOMS.—The symptoms of mushroom-poisoning are of a twofold character: gastro-intestinal irritation, and narcosis due to blood-poisoning. After a meal of poisonous mushrooms has been taken, colic sets in, followed by nausea and repeated vomiting; and diarrhoea eventually supervenes. The onset of symptoms does not as a rule manifest itself till after the lapse of some hours, six or eight or more, from partaking of the fungi. But this period is liable to great variation, and may be much shorter. Fragments of the fungi may be recognised in the fæces; and, indeed, were it not for this, and the history of the case, a diagnosis from violent ordinary gastro-intestinal catarrh would often be impossible. In severe and fatal cases the stools of the patient may become rice-watery in character; the patient becomes algid, collapsed, and cyanosed, with muscular contractions; and in children convulsions are not rarely met with. The sufferer eventually becomes somnolent, and falls into a state of sopor; but this is perhaps not due to a true narcosis.

When the *Amanita muscaria* has been taken, cerebral symptoms are more prominent. The patient appears to be in a state of inebriation; and there frequently appears to be a tendency to dash the head against a wall or other solid object. These symptoms are, however, not exclusively met with in muscarine-poisoning, but may be observed when other fungi have been eaten.

DIAGNOSIS.—The history of the case, and the detection of particles of the fungi in the fæces, are usually sufficient; but in the absence of these a diagnosis from natural disease is perhaps impossible. It has been proposed to test for the presence of muscarine, the active alkaloid of the fly-fungus, by applying a drop of the concentrated or unconcentrated urine to the heart of a frog. Muscarine causes the heart of the animal to stop in the state of diastole. It paralyses the cardiac muscle and stimulates the inhibitory ganglia.

PROGNOSIS.—The patient cannot be considered safe for at least three days, unless the more prominent symptoms have been markedly alleviated. Death may occur at any period between six and seventy-two hours. Recovery is frequent.

TREATMENT.—In poisoning by mushrooms emetics should be promptly administered, to evacuate the stomach, and those which are not of a depressing nature should be selected. The stomach-pump and tube are perhaps of little service, seeing how persistently the particles of fungi adhere to the walls of the gastro-intestinal canal. Oleaginous purgatives, as, for example, a tablespoonful of castor oil in olive oil, may be advantageously administered. Atropine appears to be a direct anti-

ote to muscarine; and digitalis appears to be so in less degree. Atropine should therefore be given in small doses in cases of poisoning by *Amanita muscaria*; and failing this some preparation of digitalis. Should atropine be administered, it would be well to avoid the use of opium; but if atropine be not administered, the exhaustive diarrhoea may have to be combated by the use of opiates combined with astringents.

THOMAS STEVENSON.

MUSICIAN'S PALSY. — See OCCUPATION-DISEASES.

MUSKAU, in Silesia, in Germany. — Sulphate-of-iron waters.

MYALGIA (*μῦς*, muscle; and *ἄλγος*, pain).—A name for pain in a muscle. See MUSCLES, Diseases of; and RHEUMATISM, MUSCULAR.

MYASTHENIA GRAVIS (*μῦς*, muscle; *ἀσθένεια*, weakness).—SYNON.: Asthenic Bulbar Paralysis.

DEFINITION.—An affection characterised by motor weakness and an abnormal tendency for fatigue to be induced by voluntary effort, which also temporarily increases the weakness, notably in the muscles that have been in action.

ÆTIOLOGY.—There is no special tendency for the malady to occur in families prone to diseases of the nervous system; in no instance has more than one member of a family been attacked, and the subjects of the affection are not, as a rule, neurotic or hysterical. The sexes are equally affected; the disease usually occurs in early adult life, and women are, as a rule, attacked younger than men. Specific fevers, septic processes, absorption of intestinal products (*copremia*), exhausting diseases, parturition, the onset of menstruation, over-exertion, exposure to cold, shock and emotional disturbances have been supposed to be exciting causes.

MORBID ANATOMY AND PATHOLOGY.—No morbid changes have been found in the majority of the cases examined, and we are not yet in a position to be certain of the significance to be attached to such slight defects as have been observed in a few of them. The condition appears to be due to the action of some toxic agent, possibly the result of disordered metabolism, and related to the ordinary phenomena of fatigue which are induced by the products of decomposition from the muscles that have been in action. Some observers have supposed that the poison is introduced into the system, and it has been suggested that it may be the outcome of microbic action. The toxin probably acts on some part of the peripheral motor neurons, either on the cells in the anterior horns of the spinal cord or cranial nerve-nuclei, the peripheral nerves, or the motor end-plates, though some believe that the muscles are primarily affected.

SYMPTOMS.—The great feature of the malady is the readiness with which fatigue is induced on muscular effort, so that muscles that are voluntarily put into action tire rapidly, though they regain power after a period of rest. The onset of the affection is usually gradual, but in a few cases it has been sudden. The weakness is sometimes first noticed in the parts supplied by some of the cranial nerves, in other cases the limbs are first affected, and in others the defect is more or less general from the outset. Muscles in constant action are especially liable to suffer; hence ptosis, weakness

of the external ocular muscles, and similar weakness of the neck-muscles are all common. The ptosis is usually partial, but is quickly increased when the patient looks upwards for a short time; the frontalis may fail to wrinkle the forehead, or having done so the skin quickly becomes smooth again. The external ocular muscles are often weak, they are readily fatigued, and nystagmoid jerking occurs on lateral turning of the eyes to either side. Diplopia is often present, and a special feature observed is that the two images change their relative positions at different times. The pupils are normal and cannot be fatigued. Weakness of the muscles of mastication is common, and may be so pronounced that chewing is impossible. Difficulty of deglutition is nearly always a symptom, and liquids may regurgitate through the nose owing to weakness of the palate. Both the upper and lower facial muscles are usually weak, so that the eyes can only be feebly closed, and pouting the lips and whistling are impossible. The tongue is imperfectly protruded, it is often tremulous, and soon begins to fall back into the mouth; moreover, the patient may be unable to thrust out either cheek with the tongue. The palate is often weak, and may fail to move at all after the letter 'a' has been vocalised several times in succession. The laryngeal muscles have been affected in a few cases. Speech is much altered, so that although the utterance may be clear when the patients begin, it soon becomes indistinct and nasal, and the voice becomes gradually weaker until they have to stop owing to want of breath. The muscles of the neck may be so weak as to be unable to support the head, and similar weakness is met with in the trunk muscles, so that the patient is unable to rise from the recumbent posture without the aid of the arms, and sitting up soon induces fatigue in the muscles of the back. The muscles of respiration may also be weak and the expansion of the thorax very poor, while even slight exertion induces dyspnoea; but apart from this, serious attacks of breathlessness are liable to come on without obvious cause, when the patient is at rest, and may be associated with feelings of faintness or palpitation of the heart. All of the muscles of the limbs may be involved, but it is especially those of the proximal segments that suffer. The myasthenia may be readily demonstrated by making the patients raise the arms above the head, for in a few seconds they begin to fall in spite of every effort to prevent them. After walking a short distance the lower extremities may become so weak that a rest is imperative, while, in some cases, the limbs give way suddenly and the patient falls.

The muscles may be fatigued by a tetanising faradic current so that they cannot be made to contract again until the electrodes have been removed for one or two minutes; this is known as the 'myasthenic reaction.' All of the muscles are, as a rule, well nourished, though in exceptional cases some of them have wasted. The tendon-jerks are usually active, but in some cases it has been possible to temporarily abolish the knee-jerks by repeated blows on the patellar tendon. The sphincters are intact, and there is no defect of cutaneous sensibility or of the muscular sense. There is a feeling of fatigue in the muscles after they have been in action; they sometimes feel stiff, and they may even be tender. In a few cases pain in the head, neck and back, photophobia and giddiness have

been premonitory symptoms; but, as a rule, sensory disturbances are absent. The symptoms are made worse by muscular exertion, emotional excitement, and exposure to cold, and, in females, menstruation and parturition have a similar influence on the course of the disease.

DIAGNOSIS.—When the manifestations of myasthenia are slight they are liable to be mistaken for the effects of *Neurasthenia*. Many of the symptoms might be due to *hysteria*, but the sensory and spasmodic disturbances that are so common in this affection are absent in myasthenia. In chronic *Bulbar Paralysis* there is no ptosis, the ocular muscles are not affected, the tongue atrophies and its muscles and those of the palate show permanent changes in their electrical excitability. Spasticity and muscular atrophy serve to distinguish cases of *Amyotrophic Lateral Sclerosis*, though there may be some difficulty of diagnosis in the rare cases in which the muscles atrophy in myasthenia gravis. Atrophy of muscles also serves to distinguish cases of *Myopathy*, in addition to which some of the muscles may be enlarged, the knee-jerks are commonly absent, and bulbar symptoms never occur. In *Chronic Progressive Ophthalmoplegia* the pupils are often affected, and optic atrophy is frequent; moreover, the limbs, if affected, are either spastic or ataxic, and the motor weakness is not increased on voluntary effort in the way that it is in myasthenia gravis. It may be difficult to exclude *acute poliomyelitis* when the onset of myasthenia gravis is sudden; but the more usual gradual onset, the variability in the intensity of the symptoms, and the absence of muscular wasting are aids to a correct diagnosis.

PROGNOSIS.—In about a third of the recorded cases the disease has been fatal, on an average in eighteen months, but mild cases no doubt pass unrecognised. The symptoms may persist for many years, remissions occur, and some cases probably recover. Weakness of the respiratory muscles and attacks of breathlessness are unfavourable symptoms, and any intercurrent pulmonary affection is a source of danger.

TREATMENT.—Rest is essential; exertion in any form, emotional excitement, and cold must all be avoided; patients must be warmly clad and they ought to be advised not to take cold baths. When difficulty of mastication and of deglutition are symptoms all solid foods must be finely minced, and it may even become necessary to feed by the stomach-tube, a procedure that is not without risk, as one patient died while the tube was being used. Drugs of all kinds have been singularly ineffective; strychnine is no exception, and iron and arsenic are only useful as tonics. Mercury and iodide of potassium have been employed without notable results, as have thyroid and suprarenal extracts. Massage and electrical treatment have also been unsuccessful; indeed, faradism has seemed to do actual harm.

J. S. RISIEN RUSSELL.

MYCETOMA (μύκης, a mushroom).—**SYN.**: Madura Disease or Madura Foot; Fungus-Disease of India.

DEFINITION.—A diseased condition chiefly of the foot and lower part of the leg, occasionally of the hand, and very rarely of the trunk, characterised by enlargement and distortion of the affected parts, due to thickening of the cutaneous tissues, with caries and subsequent fracture of the osseous struc-

tures. Two varieties of the disease have been described, the white, pale, or ochroid, and the black, dark, or melanoid. These are referred to in detail below.

DISTRIBUTION.—Mycetoma is recognised as a distinctive disease of India. It occurs more frequently in Madras, Bombay, and the westerly and north-westerly parts of India than in Bengal proper. This, however, seems to apply more especially to the dark variety; so far as the foot is concerned, the pale form is apparently the one most commonly met with all over India. A few cases have been met with in Italy and North Africa, and three apparently authentic cases of the white and one of the black variety have been described in America.

ANATOMICAL CHARACTERS AND PATHOLOGY. On laying open a characteristic specimen of the disease, the bones are found to be extremely softened, so that they can readily be divided by means of a common knife. The interior of the hand or foot is occupied by a series of sharply defined cavities, some quite isolated, but the majority communicating with one another and with the exterior by a series of complex channels, or sinuses, containing glairy fluid and solid concretions in various proportions. Both cavities and channels are lined by a dense glistening membrane, composed of white fibrous and elastic tissues. The surrounding tissues are generally in a very fatty condition, and, where the disease is of long standing, are more or less completely blended into an indistinguishable mass. So far a common description is applicable to both forms of the disease; but on proceeding to the consideration of the contents of the cavities, great differences present themselves.

Pale form.—The pale or ochroid form is capable of subdivision into several varieties, according to the nature of its morbid products. In the commonest and most characteristic variety the cavities and channels contain masses of spherical bodies like fish-roe, of a pinkish-yellow or white colour, surrounded by gelatinous glairy matter. In certain cases, however, the roe-like bodies are almost or entirely absent, and the gelatinous matter and liquid oil are generally diffused throughout the tissues. In a third and very rare variety the section looks as though besprinkled with grains of red pepper, from the presence of innumerable minute concretions of a bright red hue.

Dark form.—The appearances presented in the dark form of the disease are strikingly different. Here, in place of the roe-like bodies of the previous form, the cavities and channels contain masses of a dark-brown or black colour. These masses vary greatly in size, some not being larger than the normal fat-lobules surrounding them, others attaining to the size of a small orange. The larger masses greatly exceed any of the roe-like masses of the pale variety in size, and their consistence is also much firmer than that of the latter. They are nodulated on the surface, and closely resemble truffles in appearance. On section, they present a more or less distinctly radiating structure, and the interior is generally somewhat lighter in colour than the tuberculated exterior coating. In some cases they are tightly fitted into the cavities in which they lie, but in others they lie loose, and are surrounded by a certain amount of gelatinous matter. The amount of the latter present is, however, much less than in the pale form.

The masses of morbid material in both forms are primarily situated in spaces normally abounding in fat. Long series of them are frequently found lying among the loculi in the subcutaneous tissue, between healthy fat-lobules; others occupy the interior of the bones; and a third series are developed in the pads of fat lying around muscles and tendons. The muscles and tendons in such cases may frequently be found quite intact, although surrounded by masses of the morbid material. Due to this persistence, fracture and crushing of the softened bones often occur, and it is on this that the distortion of the affected part is in many cases in great measure dependent.

MICROSCOPICAL CHARACTERS.—*Pale form.*—The roe-like particles are composed of a nucleus of granular, waxy consistence, surrounded by a radiating fringe. The bright red particles occurring in certain cases of the ochroid form consist in great part of phosphates and carbonates, and contain a considerable proportion of iron. If sections be made through the diseased tissues, and examined microscopically with a low power, considerable epithelial proliferation will be observed at the surface. In the interior, the vessels are much thickened, and scattered throughout the tissues are yellowish bodies of variable size, and irregular or frequently uniform or crescentic in shape; these are surrounded by a radiating zone, and the whole lies in a mass of round cells, many of which may be pigmented, enclosed within a fibrous capsule. On staining by Gram's method and counterstaining with eosin the periphery of the crescentic bodies, which is stained pink with the eosin, is apparently structureless. Within the crescentic bodies, wedge-shaped or ovoid areas are present within which are masses of fine filaments stained purple by Gram's method, and resembling the mycelial filaments met with in *actinomycosis hominis* and in some cases of *actinomycosis bovis*. The radiating zone surrounding the crescentic bodies shows a more or less distinct radial striation, recalling the rays or clubs of actinomycosis. Many of the rays show a fan-shaped expansion owing to branching, and they may be very large, measuring as much as 40–50 μ in length. As is the rule in *actinomycosis hominis* (but the exception in *actinomycosis bovis*), the rays or clubs in mycetoma do not stain by Gram's method. They seem to be in a degenerate condition, but in a fortunate specimen undoubtedly clubs can be demonstrated by staining with orange-rubin or with the Ehrlich-Biondi triple stain (p. 153), thus proving that the radial zone is composed of rays or clubs analogous to those of actinomycosis. Undoubtedly therefore there are many resemblances between the white variety of mycetoma and actinomycosis, as was first pointed out by Vandyke Carter, the chief differences between them being (a) the extremely chronic character of mycetoma, (b) the absence of visceral infection in mycetoma, (c) the fact that animals do not ever seem to be infected with mycetoma, (d) the very large size and frequency of branching of the clubs of mycetoma as compared with actinomycosis, (e) the inefficiency of treatment with potassium iodide. As a matter of fact a peculiar filamentous organism, the *Streptothrix madura*, has been isolated from the white variety of mycetoma. Morphologically and in its staining reactions it resembles the *actinomyces*, but culturally there are many differences. On agar the *Streptothrix madura* forms whitish, round, raised, umbilicated

growths, which do not become yellow and black like *actinomyces*, but may become reddish, and Shattock has suggested that the 'cayenne-pepper' variety of mycetoma may be simply a stage in which this red pigment has been formed. The *Streptothrix madura* will develop in vegetable infusions in which the *actinomyces* refuses to grow; it does not liquefy gelatin as does the *actinomyces*, it will not grow anaerobically, and it is not pathogenic to any animals. See ACTINOMYCOSIS.

Dark form.—Considerable controversy has taken place as to the relation of the black to the white form of mycetoma, some regarding the former as merely a late stage in the latter, while others have suggested that the two conditions are distinct. Clinical observation bears out the latter view, for in the black variety the black particles are present from the earliest stages of the disease, and transition forms between the white and the black have not been met with, as would be expected had the one been a late form of the other. Kanthack, in sections of the black masses, observed at their periphery club-shaped elements, and he therefore believed that the black variety was a late and degenerate stage of the white. Later observations

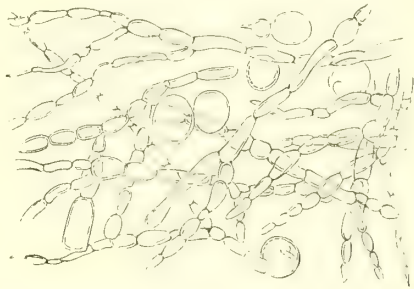


FIG. 1.—Fungoid Filaments and Capsules obtained after the prolonged maceration in caustic potash of the dark masses of Fungus-disease. $\times 500$.

have, however, shown this view to be untenable. By macerating the black masses in liquor potassae or in 'Eau de Javelle' it is possible to distinguish within them septate fungoid filaments in greater or less proportion. These are sometimes difficult to distinguish, but may generally be detected by allowing portions of the material to soak for some days in liquor potassae. The proportion which the filaments bear to the entire mass, when thus separated, is in any case very small, and in some cases extremely so, for on the completion of the soaking only a very small quantity of colourless flocculi, consisting of masses of branched filaments mixed with empty cyst-like cells, is left behind in the fluid (see fig. 1). The latter has assumed a brown colour from the solution of the dark mass. Boyce and Surveyor recognised these elements as belonging to one of the higher fungi, and they suggested that the black masses are the 'sclerotia' of a fungus, and are analogous to ergot, which is the sclerotium of *Claviceps purpurea*. More recently James H. Wright has succeeded in cultivating a fungoid form or hyphomycete from the black particles. The patient, an Italian woman, came under treatment at the end of 1897 at the Massachusetts General Hospital, with a condition which was recognised as being probably Madura foot. The patient had lived in America for several years, and the disease, which was first noticed in July 1897,

affected the base of the second and third toes on the plantar aspect. There was a small sinus which was discharging a dirty greyish fluid containing some black, hard, irregular granules, like grains of gunpowder. Amputation of the toes and metatarsal bones was performed, and on dissection of the removed tissues an ovoid nodule was found, the size of a pigeon's egg, traversed by a reticulum which divided it into small areas in which were embedded the black granules. Treated with sodium hypochlorite these became bleached and softened, and on crushing, under a cover-glass, were found to consist of a mass of fungoid structures as described by Boyce and Surveyor. Typical septate branching hyphæ were seen showing dilatations or varicosities, the periphery of the nodule apparently being made up of closely-set radiating hyphæ, but no undoubted spore-bearing organs were observed. The black granules were sown on various culture-media, and from a large number a pure growth of a hyphomycete was obtained. On potato a dense, widely-spreading, coherent membrane or layer formed, having a velvety surface and pale brown colour, except at the margins, where it was white. After some time small droplets of a dark coffee-coloured fluid appeared, and the potato became stained a dark brown. In bouillon growth appeared in the form of radiating filaments, so that fluffy balls were produced. In potato-infusion the same growth occurred, but in old cultures black granules made their appearance, consisting of closely-packed spherical or polyhedral cells, and being of the nature of sclerotia. On agar-agar the growth consisted of widely-spreading filaments of a greyish colour, with black 'sclerotia' in old cultures. This hyphomycete was composed of long branching hyphæ $3-8\mu$ in diameter, with transverse septa. No spore-bearing organs were detected, and the organism was non-pathogenic to animals. It is not unlikely that there are other varieties of the disease. The writer has described a case which did not seem to belong to either the white or the black variety.

SYMPTOMS.—Dr. Carter writes: 'As a rule the local indications of this disease are the same for all its forms; for commonly it is not possible to discriminate the several varieties by simple inspection or bare clinical history of the case.' The statements made by the patients as to the mode of origin and progress of these complaints are very various; but, taken generally, they seem to imply that the symptoms are analogous to those usually observed in deep-seated disease of the osseous and adjoining tissues. Eventually a more or less hard lump is felt in the sole of the foot or palm of the hand, or in several places. Generally one or more abscess-like formations occur; and several sinuses are ultimately established, the latter, as a rule, presenting a peculiar mammillated appearance—the 'tubercles,' apparently, of earlier writers. Along with these changes, enlargement and distortion of the affected member take place, but unaccompanied by severe pain. The distortion of the hand is very peculiar—it is shortened and thickened, owing to the destruction of the carpus and metacarpus, and the consequent irregular tension of the extensor and flexor tendons. When the foot is affected, it is found to be considerably increased in circumference, the enlargement seldom extending far beyond the ankle; the foot is prone to run in a line with the leg, and may be everted

or inverted. Discharges set in, more or less offensive, according to the nature of the subjacent degeneration, and the limb becomes not only useless, but a burden to its owner. In this manner the sufferer may go on for from one to fifteen or more years, unless relief be sought in a surgical operation. The disease does not seem ever to give rise to metastatic deposits in the internal organs.

COURSE AND DURATION.—Both forms of fungus-disease run a very chronic course, and often without very materially affecting the general health of the patient; in some cases, however, great emaciation accompanies the disease. With regard to the duration of the malady, it may be stated that cases have been recorded as having existed for various periods up to twenty-six or even thirty years.

TREATMENT.—The only treatment which is of any avail is excision of the affected parts, should the disease come under observation at an early period, or amputation. Potassium iodide, which frequently acts as a specific in actinomycosis, seems to be useless in mycetoma. R. T. HEWLETT.

MYCOSES.—During recent years it has been found that many morbid conditions are due to the growth and development of fungous forms in the tissues; such morbid conditions are known as *mycoses*. There is often little to distinguish them clinically from lesions due to the tubercle-bacillus and other well-known organisms, and hence doubtless many cases remain unrecognised. In certain instances the invading organism is an epiphyte, an organism subsisting under favourable conditions upon the morbid products of some other diseased condition. For example, in phthisical cavities and in the external ear, it has long been known that fungi are occasionally met with. Although frequently epiphytic in the ear, a true 'otomycosis' may be set up as the result of the growth of various organisms—generally species of *Aspergillus* (*niger*, *flavescens*, or *fumigatus*) or of *Mucor*—in the external ear. The disease, which is either acute or chronic, so produced results in a condition of suppurative otitis, and may cause perforation of the tympanic membrane, with more or less pain, tinnitus, and deafness. The diagnosis should not be difficult if the ear be carefully examined with a speculum. Strong alcohol, mercuric-chloride solution, calomel, or boric acid will usually effect a cure. *Aspergillus* may also grow upon the cornea. In the lungs, in phthisical cavities or bronchiectases, fungi such as *aspergillus*, *mucor*, and *oidium* may be epiphytic or secondary. The growth of these is probably a precursor of death in such cases, and is not likely to give rise to symptoms.

There are, however, primary mycoses, chiefly of the lungs, occasionally met with, and it has been proved experimentally that the spores of *Aspergillus fumigatus* are pathogenic to the rabbit. Pneumomycosis or pulmonary aspergillosis is especially a trade-disease in Paris among persons who feed pigeons and other birds artificially. The pigeon-feeder takes the grain into his own mouth, and in feeding the birds inhales any spores that may be attached to the seed. The disease has also been rarely met with under other conditions. The species present seems to be always the *Aspergillus fumigatus*. The clinical signs of pneumomycosis are those of chronic bronchitis and emphysema, or of chronic pulmonary tuberculosis; usually at first the signs are those of bronchitis, with recurrent cough

and greenish purulent expectoration, followed later by consolidation with hæmoptysis, pyrexia, and pleurisy. The diagnosis can only be made by the absence of the tubercle-bacillus from, and the presence of the characteristic mycelium and sporangia of the aspergillus in, the sputum. A mycelium alone is not necessarily aspergillar, it might be actinomycotic. In some instances a double infection with the aspergillus and the tubercle-bacillus takes place. The prognosis in pneumono-mycosis is less grave than in pulmonary tuberculosis, and the disease tends to a spontaneous cure. The treatment must be on general principles, together with iodide of potassium and arsenic. The *post-mortem* changes resemble those of tuberculosis, and consist of whitish tubercle-like nodules, smaller and larger cavities, with surrounding consolidation.

Cases of skin-disease characterised by nodular formation, with subsequent ulceration or suppuration and great destruction of tissue, have been described. In the affected tissues large ovoid bodies with a double contour are present, and these have been regarded as protozoa. Other observers have, however, cultivated a mycelial form in this disease, which may, therefore, be a 'mycotic dermatitis' (*blastomycetic dermatitis*). In its clinical characters the disease is not unlike some forms of cutaneous tuberculosis. In the black variety of mycetoma a fungus-form has been isolated. See MYCETOMA.

The ringworms, favus, and thrush are also examples of mycoses. See TINEÆ.

The so-called 'Mycosis tonsillaris,' consisting of small, white, tough, adherent excrescences upon the pharyngeal mucous membrane, seems to be primarily a keratosis, with secondary infection with bacteria and leptothrix. See TONSILS.

R. T. HEWLETT.

MYCOSIS FUNGOIDES (μύκης, a mushroom or fungus).—**SYNON.** : *Granuloma Fungoides* (Auszitz); *Fibroma Fungoides* (Tilbury Fox); *Lèpre Indigène* (Guérard); *Lymphadénie Cutanée* (Gillot); and probably *Ecceema Hypertrophicum et Tuberculatum* (Erasmus Wilson).

DEFINITION.—A chronic, probably infective, disease of the skin, characterised by a more or less prolonged eczematoid condition, followed by the development of multiple fungating tumours; and almost invariably terminating fatally.

ÆTIOLOGY.—Of the ætiology of mycosis fungoides little that is definite is known. It attacks men with much greater frequency than women, and usually in middle adult life—from forty to fifty years of age; but one case is reported to have begun at the age of five (Port). It is certainly not hereditary. It is believed by many observers to be infectious, though up to the present direct evidence of contagion is wanting, and a specific microbe has not yet been isolated.

SYMPTOMS AND COURSE.—The *first* manifestations of mycosis fungoides may resemble an erythema, an urticaria, a lichen, or the earlier stages of an eczema. They usually consist of well-defined macules, or more extensive erythematous patches, of a pinkish or bright red colour which does not completely disappear on pressure. These may remain discrete, or may coalesce so as to cover large areas of skin, and show a marked predilection for development upon the trunk, scalp and face, while the upper extremities are more frequently and more severely affected than the lower. The lesions are

in the majority of cases notably asymmetrical. Erysipelatous outbreaks, with some elevation of temperature, are prone to occur from time to time, and are usually followed by temporary amelioration of all the symptoms. The patches, which are at first on a level with the surrounding skin, soon become elevated, infiltrated, dense to the touch, and covered with some fine desquamation. They are accompanied by a variable, but generally considerable, amount of burning, tingling, or itching, the latter symptom being in exceptional cases excruciating; papules and vesicles may form upon the patches either spontaneously or as the result of scratching or rubbing. It is almost pathognomonic of the disease that all these manifestations appear and disappear spontaneously with remarkable rapidity, leaving previously affected skin apparently healthy, or at most only slightly atrophic, scaly, or pigmented.

Although partial recovery may thus be said to occur, and complete recovery sometimes seems imminent, the disease invariably progresses to its *second stage*, which is characterised by the deeper infiltration of the derma, and the gradually increasing prominence of the patches, to constitute the *plaques lichénoides* of Bazin. They are irregular in outline, sharply demarcated, bossy on the surface, of a deep purplish or brownish-red colour, and may either be covered with thick scales like a psoriasis, or may ooze and scab like an eczema. These lesions present the same peculiarity as to rapid disappearance as those of the first stage. Another phenomenon noted in a few of the recorded cases—which constituted a marked feature in two of the cases observed by the writer—consists in the frequent appearance of deeply seated lumps indistinguishable from boils, most of which disappear without treatment; some, however, rupture and discharge their pus, but never a necrotic 'core' comparable to that of a true boil. They may, or may not, leave disfiguring scars. When the scalp is involved the hair usually falls rapidly. The nails often become yellow, brittle, and deformed.

After a variable period of time the *third stage* of the disease is attained. It is characterised by the development of peculiar, generally multiple tumours upon skin which is either erythematous or infiltrated; occasionally, however, they spring up from apparently healthy skin. The tumours vary greatly in size, those on the hands being often not bigger than a bean and 'let into' the skin, while on the trunk they may be the size of an orange, or even larger. When situated in considerable numbers upon the face they give a leonine expression very similar to that seen in leprosy. The tumours are sessile, sharply defined, firm, generally of a peculiar deep red colour, but sometimes pale pink or yellowish; they are usually lobulated, and have not inaptly been likened to tomatoes; the epidermis over them is intact, but stretched, tense, and glistening. The amount of pain attending them is variable, but usually they are tender to manipulation. Occasionally tumours appear in the mouth or fauces. In some of the reported cases early and conspicuous involvement of lymphatic glands has been noted, as well as leucocythæmia and enlargement of the spleen, but these appear to the writer to be examples of a separate disorder—a form of lymphadenoma—which it is of importance to differentiate from mycosis fungoides. The same may be said of the form described by Vidal as *Mycosis à tumeurs d'emblée*,

in which there is no eczematoid, pre-mycotic stage, but the growths, which are usually localised, constitute the first and only manifestation of the disease. Such cases are possibly examples of a form of true sarcoma of the skin (Perrin, Hallopeau).

When once developed the tumours may remain unaltered for an indefinite period. More frequently, however, a large number of them spontaneously disappear, leaving the skin either hardened, shrunken, pigmented and desquamating, or else apparently healthy. Fresh erythematous and eczematoid patches nevertheless make their appearance, and fresh tumours, either singly or in crops; in one case (Stelwagon) as many as six hundred were finally present. Of these a certain number ulcerate slowly, the epidermis over them being destroyed; and hideous fungating ulcers are thus formed, from which a thin, ichorous, intensely fetid fluid is discharged. At this stage itching and smarting usually cease, and the tumours become painless. But now for the first time—and the point is one of the most curious and characteristic features of the affection—the general health begins to suffer. Although many of the deep ulcerations may cicatrise, a profound marasmus is gradually established, the patient being usually ultimately carried off by diarrhoea, pulmonary complications, or septicæmia.

DURATION.—The duration of mycosis fungoides varies from a few months to twenty years or more (Besnier); on the average it is from five to six years. In one of the cases observed by the writer, apparent complete recovery took place under treatment, and persisted for more than six months, when a relapse occurred, during which the patient, who was very alcoholic, contracted acute pneumonia, of which he died. It is noteworthy that growths in internal organs have only been observed three times (Duhring, Galliard, Pye-Smith), and in each instance the association appears to have been a coincidence, as the internal tumours did not present the characters of the external ones.

MORBID ANATOMY.—The histological changes found in the early or pre-fungoid stage consist almost entirely of a connective tissue-cell proliferation situated in the corium around the blood-vessels, hair-follicles, sebaceous glands, and sweat-glands, accompanied by a variable degree of dilatation of the blood-vessels of the subpapillary and papillary layers. It is this marked cellular increase which gives to the early lesions of mycosis fungoides their peculiar feeling of infiltration. The cells are mainly of two varieties, namely, large, oval, or fusiform connective-tissue cells, many of which exhibit active mitosis, and numerous smaller roundish cells; but besides these there are a few mast-cells, plasma-cells, and imperfect giant-cells, such as are met with in different proportions in the whole group of the infective granulomata. The epidermis presents certain well-defined changes, such as downgrowth of the interpapillary processes into the corium, interepithelial oedema, and imperfect cornification, but none of these changes can be regarded as in themselves characteristic of this disease. In the fungating tumour-stage the cell-proliferation goes on increasing, but the new cells show a marked tendency to break down, and the connective tissue and elastic fibres to disintegrate, so that a histological examination of one of these tumours reveals an ill-defined mass of broken-down cells and fibres scattered throughout with irregular crenated cells which have not yet broken up. The cells of the

epidermis also become destroyed, and the granulomatous mass is found extending often from the depth of the corium to the surface of the epidermis, and covered only by a thin pellicle of the horny layer, or reaching the surface of the skin and forming the floor of an unhealthy-looking ulcer.

PATHOLOGY.—Divergent views are held as to the pathological group to which the tumours in mycosis fungoides belong. By some authorities they are still regarded as *lymphadenomata* and by others as *sarcoma*; but most recent observers, considering not only their microscopical characters, but also their clinical peculiarities, relegate them to the group of *infective granulomata*. Micrococci have been demonstrated in the tumours and in the capillaries surrounding them; but all attempts to reproduce the disease by the inoculation of pure cultures of these in animals have proved unsuccessful.

More recently McVail, Murray, and Atkinson have isolated a 'short white bacillus' which in rabbits produced certain pathological changes which resulted in death. Their observations have not, however, been confirmed. In the breaking-down granulomatous tissue of this disease we have a medium in which many organisms will readily thrive, even pure cultures of the bacillus of Friedländer having been obtained, so that great care is necessary in distinguishing merely adventitious micro-organisms from a specific microbe. In the blood both ordinary leucocytosis and eosinophilia have been recorded (Nékám), but in the recent cases reported by Galloway and MacLeod, neither an increase in the number of leucocytes nor in the proportion of the eosinophiles was detected.

DIAGNOSIS.—In the later stages of mycosis fungoides no difficulty presents itself as regards diagnosis; but in the earliest stage it may be impossible to differentiate the disease from urticaria, erythema multiforme, or even pityriasis rubra. Its persistence and ulterior developments soon, however, settle the point. In the eczematoid phase the localisation of the patches, their asymmetry, the amount of infiltration and sharp delimitation, their chronicity and greater resistance to treatment, the scantiness of discharge, and their spontaneous appearance and disappearance, generally serve to distinguish the condition from any recognised type of eczema. The early stage of mycosis fungoides has been occasionally mistaken for macular leprosy, but in the former condition anæsthesia and leucoderma are absent; in the tumour stage a bacteriological examination would render the diagnosis from tubercular leprosy perfectly simple.

Framboesia may, apparently, resemble it closely, but is a disease of tropical climates only, the tumours are always small, and there is no pre-mycotic eczematoid stage. The main points of difference from sarcoma and various forms of lymphadenoma of the skin have been already briefly alluded to.

PROGNOSIS.—The prognosis of this disease is necessarily gloomy, but its rate of progress varies widely in different cases. It is only when the final ulcerative and cachectic phase is reached that the patient's condition becomes a very painful or utterly hopeless one.

TREATMENT.—One case of complete and permanent recovery is reported (Bazin) after an accidental attack of erysipelas; inoculation with the streptococci of that disease, in a manner similar to that successfully carried out in the treatment of

intractable cases of lupus, carcinoma, and sarcoma (Fehleisen), appears, therefore, to be a rational and legitimate procedure. Injections of Coley's fluid (mixed toxins of *Streptococcus pyogenes* and *Bacillus prodigiosus*) have proved to be temporarily beneficial.

Arsenic internally, in full and steadily increased doses, has yielded in many cases decidedly beneficial results in the early stages of mycosis fungoides—an observation which the writer is in a position to confirm from practical experience. Its administration hypodermically is probably advantageous, but is seldom tolerated. Mercurials and iodides appear to be deleterious rather than beneficial.

Externally, ointments containing pyrogallol (10 to 15 per cent.) seem particularly efficacious in the eczematoid stage. When tumours are present, the injection of strong carbolic acid into their base has been suggested by Radcliffe Crocker. If ulceration of the tumours has set in, the most scrupulous cleanliness and careful nursing are required. The horrible factor is best controlled by dusting with calol (one part to ten of subnitrate of bismuth), and covering with antiseptic gauze and absorbent wool. Washing with camphorated naphthol has been recommended by Brocq, and lotions of creolin or any other antiseptic are probably useful. Surgical ablation of the tumours has been carried out with success, and without local recurrence, in a few cases in which their number was limited.

J. J. PRINGLE.

MYDRIASIS (*μυδρίασις*).—A preternatural dilatation, and sluggishness or immobility of the pupil. It is the opposite of myosis. See PUPIL, Disorders of.

MYELITIS (*μυελίτις*, the marrow).—Inflammation of the spinal cord. A term that has been much abused, and which is still often wrongly applied to many mere degenerative softenings of this organ. See SPINAL CORD, Diseases of.

MYELOID } (*μυελός*, the marrow).—A form of
MYELOMA } carcinoma, characterised by the presence of giant or myeloid cells. See TUMOURS.

MYOCARDITIS (*μῦς*, a muscle; and *καρδία*, the heart).—Inflammation of the walls of the heart. See HEART, Diseases of.

MYOCLONUS MULTIPLEX.—See PARAMYOCLONUS MULTIPLEX.

MYOMALACIA CORDIS.—SYNON.: Softening of the Heart.—Softening of the myocardium is a result of obstruction of the coronary vessels or their branches by arterial sclerosis, syphilitic arteritis, thrombosis or embolism. The affected area, commonly in the neighbourhood of the apex of the left ventricle, is well defined and softened, possesses the characters of an infarct—red or white—in some stage of its development, with associated pericarditis and endocardial thrombosis. The tissue-elements are degenerated, necrosed, and mutually separated. Occasionally incomplete or complete rupture of the heart occurs. If the subject of myomalacia survive these changes, the affected area becomes fibroid as a result of repair. See HEART, Diseases of, p. 641.

J. MITCHELL BRUCE.

MYOPATHY.—SYNON.: Idiopathic muscular atrophy and hypertrophy; Primary progressive myopathy; Progressive muscular dystrophy.

DESCRIPTION.—Under the term 'myopathy' is included a group of cases in which atrophy of muscles occurs, either alone or in association with hypertrophy of other muscles, and in which the defect originates in the muscle itself, and does not depend on any demonstrable morbid condition of the central or peripheral nervous system. Heredity is a prominent factor in the aetiology of these affections, and young people are almost exclusively attacked.

Most of the cases included under the name 'myopathy' fall into one of three classes: pseudo-hypertrophic paralysis; the juvenile, or upper-arm form of idiopathic muscular atrophy; or the facio-scapulo-humeral form of the same affection.

The cases that belong to the first of these classes are broadly distinguished from those included in the other two by the fact that hypertrophy of muscles rather than atrophy dominates the clinical picture, whereas in the cases that fall into the other two classes atrophy of the muscles is the prominent feature. It must, however, be remembered that in pseudo-hypertrophic paralysis certain muscles atrophy, and that in the other two forms of myopathy some muscles may hypertrophy. Indeed, there are cases that belong to the pseudo-hypertrophic form in which, nevertheless, atrophy of muscles dominates the picture, while if there be any hypertrophy it is so slight, and affects so few muscles, that it is a comparatively insignificant feature in the cases. The main justification for including these with the typical cases of pseudo-hypertrophic paralysis is that a particular combination of muscles is found atrophied in them, which is regarded as characteristic of the pseudo-hypertrophic form.

ÆTIOLOGY.—Heredity plays a most important part in the aetiology of these affections; several members of the same family are commonly affected, and the malady can be traced through several generations. This does not always obtain, however, for undoubted examples of myopathy occur in isolated cases where no other member of the family is known to be affected. The pseudo-hypertrophic form is further characterised by the fact that the transmission from parent to offspring is through females in the family, although the males are usually attacked. This tendency for the disease to manifest itself in boys much more often than in girls, which is so characteristic of pseudo-hypertrophic paralysis, is not met with in the other forms of myopathy, for in them the sexes are about equally affected.

The myopathies are essentially diseases of early life, and they thus differ from the muscular atrophies of spinal origin which more commonly attack older people. Pseudo-hypertrophic paralysis usually commences in childhood. The child may never have walked; more commonly it has learnt to do so, but at the age of four or five years it becomes clumsy on its feet; in some cases the onset is delayed until ten or even until puberty. The other forms of myopathy usually commence at the age of puberty rather than earlier, though the facio-scapulo-humeral form of the disease commonly manifests itself in infancy; indeed, Landouzy and Déjerine in their original description of this variety of myopathy considered that the affection usually begins

at this early age. Few cases of myopathy begin later than the twentieth year.

MORBID ANATOMY.—The morbid changes are limited to the muscles; no defect has been found in the central or peripheral nervous system adequate to account for the profound structural alterations that occur in the muscles. The precise appearances which the muscles present vary according to the form of myopathy that is under observation and the stage which the disease has reached before the fatal termination. Muscles which are still enlarged in a case of pseudo-hypertrophic paralysis owe their size chiefly to increase of interstitial fat and fibrous tissue, rather than to any increase in the size of the muscle-fibres, or their multiplication, for most of the muscle-fibres atrophy, lose their transverse striation, and undergo granular disintegration. Erb, however, contends that the muscle-fibres do hypertrophy, that they subdivide, and that their nuclei multiply; though the fibres subsequently atrophy, disappear, and are replaced by proliferation of connective tissue in which fat appears. This view obtains support from the fact that in portions of muscles removed during life some fibres have been found enlarged, while others have been atrophied, a state of things that has also been met with on *post-mortem* examination. It is said to be rare for the muscle-fibres to show evidence of fatty degeneration, longitudinal striation, fissuring or vacuolation, but changes of this kind have been described in some cases.

The proportion between interstitial fat and connective tissue varies in different cases, and whereas the former may be abundant in the muscles that are still enlarged, the latter may alone be obvious in muscles that have never been increased in volume, or in those that have been enlarged and that have subsequently become reduced in size. In no case, however, is there evidence that the connective tissue is the outcome of fibroid degeneration of the muscle. No changes have been found in the muscle-spindles. Changes in the blood-vessels of the muscles have been described, the walls are thickened, and the lumina narrowed, or even blocked. Babes and Fürstner have described slight changes in the intra-muscular nerves and end-plates, while Gombault has found changes in parts of the axis-cylinders of nerves outside of, but close to, the muscles.

PATHOLOGY.—It is probable that the disease is congenitally determined, and it seems certain that there is a hereditary predisposition for the muscles to succumb in the way that they do; but beyond this we possess no positive knowledge as to how the changes are induced in the muscles. Several views have, however, been expressed with regard to the pathology of the affection. One hypothesis is that the changes in the muscles are primarily interstitial, while the muscle-fibres undergo secondary atrophy. Another view is that the disease begins in the muscle-fibres themselves, and that the fibro-fatty proliferation is secondary to this. Although all observers are agreed that the affection of the muscles is independent of any structural change in the nervous system, Erb contends that the juvenile form of idiopathic muscular atrophy is not a pure myopathy, but is due to functional disturbance in the spinal cord. In view of the structural alterations which he has described in the blood-vessels, Babes regards the changes in the muscles as secondary to the affection of the vessels, and consequently

due to defective nutrition. The reason why certain muscles succumb, while others escape, has been supposed by Babinski and Onanoff to be due to the fact that the muscles that become affected are those that are first to be developed, which accordingly tend to undergo degeneration earlier than those which develop later.

SYMPTOMS.—Although we have considered the myopathies collectively up to the present stage of our description of these affections, it is convenient and of advantage to describe each of the forms separately, in so far as their clinical manifestations are concerned. Even in their clinical aspects, however, the different forms present certain features in common, so that it will be instructive to review the symptoms common to them all, after each of the three forms has been described.

1. *Pseudo-hypertrophic Paralysis.*—The child usually comes under observation because it is clumsy in the use of its lower extremities; it is continually falling, and experiences difficulty in regaining the erect posture, or the first defect noticed may be a difficulty in ascending stairs. These disabilities may appear especially remarkable in view of the fact that the muscles of the legs often seem preternaturally well nourished, owing to the tendency which there is for some muscles to become enlarged. The muscles that are increased in size may remain so throughout the course of the disease; but on the other hand, they may subsequently undergo atrophy, and there are other muscles which atrophy without ever showing any hypertrophy.

The muscles that are prone to become enlarged in the lower extremities are those of the calf, the extensors of the knee, notably the vastus externus, and the gluteus maximus; in the upper extremities, the deltoid, supra- and infra-spinati, and triceps are the muscles usually affected in this way; while, of the trunk muscles, the erector spinæ may be enlarged, though in other cases it is atrophied.

Of the muscles that undergo atrophy it is important to recognise that the lower half of the pectoralis major and the latissimus dorsi are almost invariably affected in this way, and that this grouping of the muscular atrophy is of great value in the diagnosis of this form of myopathy. The atrophy may be so complete as to suggest that the muscles are congenitally absent, and yet the upper half of the pectoralis major stands out prominently, in contrast, when this muscle is brought into action. The serratus magnus is affected in some cases, and the biceps commonly wastes. In the lower extremities the muscles that usually atrophy are the flexors of the knee, and the adductors and flexors of the hip.

While certain muscles hypertrophy, and others atrophy, there are others that remain unaltered. This is the rule to which there are but few exceptions in the case of the muscles of the face, though the masseters and tongue are sometimes enlarged. The muscles of the neck also usually escape, as do the trapezius and rhomboids. The intrinsic muscles of the hands are nearly always intact, and the muscles of the forearm usually escape also, though the supinator longus may be hypertrophied or atrophied, and slight enlargement of the extensors of the wrist is sometimes met with.

All the affected muscles, whether atrophied or hypertrophied, are weak; that this is so in the case of the latter class of muscle is well seen in the calf, for though these muscles may be greatly enlarged,

the patient is unable to stand on tip-toe. The most characteristic defects which are due to the muscular weakness are certain deformities, notably of the spine, an alteration in the attitude and gait, a peculiar way in which these patients rise from the recumbent to the erect posture, and later on their inability to do so at all, even by the aid of the dodges which they formerly employed. In the sitting posture there may be no notable deformity of the spine, or there may be a pronounced kyphosis shared by the whole length of the spinal column; but when the patient is in the erect posture a pronounced lordosis commonly appears in the lumbar region, while there is compensatory kyphosis in the cervical and upper thoracic regions of the spine, and the shoulders are thrown unduly backwards, so that a plumb-line dropped from the most prominent part of the convexity of the spine falls well clear of the sacrum.

The patient stands with his feet far apart, so as to rest on a wide base, and in walking he waddles from side to side in order to keep the advancing foot clear of the ground, while the knee is brought forward in advance of the foot when the extensors of the knee are feeble. He experiences difficulty in rising from the sitting posture, chiefly owing to weakness of the extensors of the hip and knee. In attempting to get up from the recumbent position he first rolls over to one side, then draws up his legs to the trunk and twists his body over so as to get on to his hands and knees. He next puts his head down between his arms and gradually extends his knees until he rests on his hands and toes; the hands are then brought nearer and nearer to the feet, which are made the fixed point, and first one and then the other hand is rapidly moved from the ground to his knees, and he then proceeds to climb up himself by placing each hand in turn higher and higher up his thighs, and finally by a sudden movement of his shoulders backwards he attains the erect posture.

With the progressive weakness which occurs in the affected muscles, be they atrophied or hypertrophied, the electrical reactions, which are at first normal, become altered, so that the amount of contraction of the muscles which results when they are stimulated both by faradism and galvanism becomes progressively less, until there are no muscle-fibres left to give a response; but the reaction of degeneration is never present. Fibrillary tremors of the muscles scarcely ever occur, and there is never any spasticity. Contracture of the calf-muscles, which is often an early manifestation, and which leads to talipes equinus, is due to contraction of fibrous tissue, and not to any spasm of the muscles. The knee-jerks are lost or retained according to whether the extensors of the knee are or are not affected, and from Sherrington's experimental observations in the monkey it would appear to be the condition of the vastus internus that is important in this connection. As the extensor muscles become weaker, so the knee-jerk becomes more feeble until ultimately it is abolished. Ankle-clonus is never present. The superficial reflexes persist as long as the muscles on which they depend are capable of responding. There is never any blunting of cutaneous sensibility. The sphincters escape, but may possibly become affected in the final stage of the malady.

2. *Juvenile or upper-arm form of idiopathic muscular atrophy.*—This form was first described by Erb. The disease usually manifests itself about

the time of puberty, the atrophy commencing in the muscles of the upper arm and those of the shoulder-girdle. The muscles that are earliest affected are the biceps, triceps and supinator longus, and these are followed by the pectoralis major, latissimus dorsi, serratus magnus, and trapezius. The trapezius is liable to be atrophied throughout its whole extent, and there is not the tendency for its upper portion to escape in the way that it does in the muscular atrophies of spinal origin. The sternomastoid is also affected in some cases, while later in the course of the disease the muscles of the trunk, the glutei, and the muscles of the thigh, notably the quadriceps-extensor group, suffer. The flexors and adductors of the hip may also be weak, and in some cases the anterior tibial group and the peronei are involved. The deltoid and supra- and infra-spinati usually escape, and the rule that has but few exceptions is that the muscles of the face, those of the forearm, with the exception of the supinator longus, and the intrinsic muscles of the hand also escape. Certain noteworthy deformities of the spine result in consequence of the weakness of the trunk-muscles and glutei. If the erector spinæ is alone affected there is marked lordosis, when the patient stands, so that a plumb-line suspended from the most prominent part of the cervico-dorsal spines drops clear of the sacrum; but when the glutei are also weak the pelvis becomes tilted forward, and the sacrum projects so far back that the plumb-line is no longer able to clear it. In either case the lordosis is replaced by kyphosis when the patient sits down, provided the recti muscles of the abdomen be intact; but the lordosis persists if the recti are weak, as these muscles are then unable to flex the spine by approximating the thorax and pelvis in front.

3. *The facio-scapulo-humeral form of idiopathic muscular atrophy.*—This affection usually commences at the time of puberty, although there is commonly a history that the patient has never been able to close the eyes properly, or whistle. Whether the disease commences in infancy or not, the face is usually the first part affected, and the first muscles to suffer are the orbicularis palpebrarum, and the orbicularis oris. The patient is accordingly unable to close the eyes tightly; the lips are prominent and everted, and cannot be pursed, as in whistling. The levator anguli oris also becomes affected, so that although the angles of the mouth can be retracted, the upper lip cannot be elevated; or the zygomatici may suffer, so that instead of the upper lip being drawn outwards and upwards, when the patient smiles, the lip is drawn upwards only. The forehead commonly looks as smooth as ivory, the expression is characteristically sanctimonious, and when attempts are made to perform facial movements the angles of the mouth are depressed so that the patient looks as if about to cry. Nearly every muscle of the face may in time become affected. A point that is of considerable importance in diagnosis is that the muscles of the eyeballs and tongue escape, despite the fact that the orbicularis palpebrarum and orbicularis oris are affected.

The muscles that next become atrophied are those about the shoulder-girdle and upper arm, and later in the course of the disease the extensors of the wrist and fingers may also be affected. The supra- and infra-spinati escape, as do the flexors of the wrist and fingers, and the intrinsic muscles of the hand. The muscles of the trunk and lower

extremities are attacked in the same way as in the form of myopathy in which the face escapes, and the same deformities of the spine and thorax, and alterations in the character of the gait are met with, so that a separate description of these defects would be superfluous.

Features that the myopathies possess in common. The three forms of myopathy have certain characters in common. They are all progressive. In all of the forms of myopathy, whether there be hypertrophy of any of the muscles or not, some muscles may be unduly hard, in consequence of replacement of the muscle-fibres by fibrous tissue, though this does not obtain in every case. The affected muscles, whether hypertrophied or atrophied, become progressively weak, and their response to both forms of electrical current becomes more and more feeble, until finally there are no muscle-fibres left to respond; but there is never any qualitative change in the mode of response to galvanism such as constitutes the reaction of degeneration. Fibrillary tremors are so rare that most writers on this subject state that they never occur. Although there may be contraction of muscles, consequent on shortening of the fibrous tissue which replaces the muscle-fibres, there is never any spasticity such as commonly accompanies the chronic muscular atrophies of spinal origin. The knee-jerks persist, as long as there are a sufficient number of muscle-fibres intact in the extensors of the knee, and the vastus internus is probably the muscle whose state of nutrition is of the most importance in this connection. The superficial reflexes also persist as long as the muscles on which they depend are intact. There is never any blunting of sensibility; tactile, painful and thermal impressions are all normally perceived. The sphincters remain normal throughout the course of the disease, except, it may be, in the final stages of very advanced cases, when there may be loss of control over both sphincters.

DIAGNOSIS.—The detection of hypertrophy of any muscles makes it certain that the case is myopathic in nature, and that the muscular atrophy is not due to affection of the spinal cord or peripheral nerves. Valuable information is also derived from the combination in which the muscles are atrophied, and, in the absence of hypertrophy of any muscles, diagnosis rests largely on which muscles are atrophied. In the myopathies the muscles of the proximal segments of the upper limbs suffer earliest and in greatest degree, while those of the distal segments usually escape; whereas in the myelopathies muscles of the distal segments of the upper limbs most commonly succumb first, and even when the affection begins in the shoulder, the deltoid is the muscle that atrophies first, whereas this muscle usually escapes atrophy in the myopathies, in spite of the fact that so many other muscles about the shoulder-girdle are affected. Another fact of importance is that the trapezius may become atrophied early, and throughout its whole extent, in the myopathies, whereas the upper portion of this muscle usually escapes to the very end in cases in which the muscular atrophy is due to affection of the spinal cord. The escape of the muscles of the eyes and tongue, despite the fact that the orbicularis palpebrarum and orbicularis oris are affected, is of great value in distinguishing the facial affection of the facio-scapulo-humeral form of myopathy from paralysis due to affection of the nuclei of the third and twelfth nerves; for if the paralysis of the orbicularis palpe-

brarum be due to a lesion of the third nucleus, the ocular nerves are concomitantly affected, and lesions of the hypoglossal nucleus occasion paralysis of the tongue in conjunction with the orbicularis oris. The presence of spasticity associated with exaggerated knee-jerks and ankle-clonus indicates that the muscular atrophy is of myelopathic origin. Extension of the toes when the plantar surface of the foot is stimulated is of similar significance. The absence of these phenomena does not, however, exclude the possibility that the muscular atrophy is due to disease of the spinal cord. When fibrillary tremors are present in the muscles it is highly probable that the disease is myelopathic, though this feature does not absolutely negative a myopathic origin of the muscular atrophy. The age of the patient is a further aid in diagnosis, for the myopathies occur in children and young adults, while the muscular atrophies of spinal origin, as a rule, belong to the later degenerative periods of life. More important still is the hereditary tendency of the disease, as it is most exceptional to have transmission from parent to offspring, or affection of more than one member of a family, in the muscular atrophies of spinal origin.

The peroneal type of muscular atrophy resembles the myopathies in that several members of a family are affected, in addition to which it may be hereditary, and the manifestations begin early in life. The atrophy, however, commences in the peronei and other muscles of the legs below the knees, and the small muscles of the hands become similarly affected. Moreover, there may be pain, cutaneous sensibility is commonly blunted, and vaso-motor changes occur in the skin.

In subacute poliomyelitis, except the most chronic form, paralysis precedes wasting of the muscles, and there is commonly some tendency to recovery, although in other cases the disease is progressive. There is a random distribution of the atrophy instead of the muscles being affected in special combinations, as is the case in the myopathies. Moreover, the affected muscles commonly show the reaction of degeneration on electrical stimulation.

Atrophy of the muscles of the shoulder and upper arm, consequent on a lesion of the fifth and sixth cervical nerve roots, is distinguished by the fact that all the muscles affected are supplied by these roots; and, moreover, all the muscles in their supply are usually affected, instead of some of them escaping, as is the case in the myopathies. In a lesion of the fifth root there is paralysis with atrophy of the supra- and infra-spinati, clavicular portion of the pectoralis major, deltoid, biceps, brachialis anticus, and supinator longus; while damage to the sixth root results in similar affection of the sternal part of the pectoralis major, the latissimus dorsi and triceps. The muscles show the reaction of degeneration on electrical stimulation. A history of pain and the presence of anæsthesia may further help to distinguish cases of this kind from muscular atrophies of myopathic origin.

The exclusion of syringomyelia is based on the fact that sensibility is blunted in that disease; indeed there may be a dissociation of sensibility, so that although painful and thermal impressions are no longer perceived, tactile impressions are nevertheless correctly recognised. Moreover, the patients are subject to trophic disturbances of the skin, spastic phenomena appear in the lower extremities, lateral curvature of the spine usually occurs, nystagmus is

common, and the small muscles of the hands are those that are usually atrophied.

PROGNOSIS varies in the different forms of myopathy. The subjects of pseudo-hypertrophic paralysis die before they reach the age of twenty, commonly as the result of bronchitis or pneumonia; suffocation results as the expiratory muscles are too feeble to expel the secretion from the lungs. Prognosis is, however, favourable in the irregular forms of pseudo-hypertrophic paralysis which attack adults; but, strictly speaking, many of these are cases of idiopathic muscular atrophy in which some muscles are hypertrophied.

The outlook in the other two forms of myopathy is much more favourable than in the ordinary form of pseudo-hypertrophic paralysis, for these affections are compatible with preservation of life for a good many years. Indeed, Erb believes that the malady is sometimes arrested by treatment. Whether as a result of treatment or not, it is certain that some cases do remain stationary for many years. The age at which the malady commences has a distinct influence on prognosis in the facio-scapulo-humeral form, for the cases that begin after puberty are more likely to become arrested than are those that begin in childhood. When there is arrest of the disease, muscles that are atrophied do not recover, but there is no further progress of the atrophy in those that are not already completely destroyed, nor do muscles hitherto unaffected show any tendency to waste.

TREATMENT.—It is important to maintain the patient's general nutrition at as high a standard as possible, in the hope that the nutrition of the affected muscles may also be favourably influenced. With this object in view, a liberal dietary must be provided, and it ought to be supplemented by cod-liver oil, malt, and tonics such as arsenic and iron. The drug that is administered with most hope that benefit may accrue from its use, in this as in other forms of muscular atrophy, is strychnine, which may be taken by the mouth, but which is supposed to be more efficacious when given by subcutaneous injection. Of local measures, in the treatment of these cases, both massage and galvanism are of great importance. By their aid we may hope to influence favourably the nutrition of the muscles, and thus retard, or arrest the progress of the atrophy, so that these measures should be persevered with, no matter how discouraging the result may seem. Passive movements are of great service in preventing contracture, and when shortening of muscles has already occurred tenotomy is indicated, where there is reason to suppose that this measure will enable the patient to use the limbs again. This is especially important in regard to the talipes equinus that is so common in pseudo-hypertrophic paralysis, as the correction of this deformity may enable the patient to resume walking exercise, for it has been observed that these patients usually become rapidly worse when they are no longer able to walk. Although patients suffering from the different forms of myopathy must be encouraged to take exercise, this must not be in sufficient amount to cause fatigue; indeed, any depressing influence, whether physical or mental, is to be avoided. When the patients become bedridden it is important to avoid contracture and fixation of parts in awkward positions, so that the trunk should be propped up to prevent such curvatures of the spine as can thus be avoided; and the limbs ought to be kept, as far as

possible, in suitable positions. As so many of these patients succumb to pulmonary affections, such as bronchitis and pneumonia, great care must be taken to keep them warmly clad, and to avoid exposure such as may occasion a chill.

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MYOPIA (μύωψ; from μύω, I close or blink; and ὤψ, the eye).—That form of *ametropia*, or error of refraction, in which, owing to a high refractive index of the dioptric media, or excessive convexity of the refracting surfaces, or abnormal elongation of the antero-posterior axis of the eyeball, parallel rays of light converge to a focus in front of the retina, and form therefore circles of diffusion upon it. It is the opposite of *hypermetropia* (see *HYPERMETROPIA*), and is sometimes called *brachymetropia* (βραχύς, short; μέτρον, a measure; and ὤψ, the eye), or *hypometropia* (ὑπομέτρος, below the measure). See VISION, Disorders of.

MYOSIS (μύω, I shut).—A preternatural contraction and sluggishness or immobility of the pupil. The opposite of mydriasis. See PUPIL, Disorders of.

MYOSITIS (μῦς, a muscle).—Inflammation of a muscle. See MUSCLES, Diseases of.

MYOTATIC (μῦς, a muscle; and τατικός, extended, tense).—Tense-muscle irritability or action. 'Myotatic irritability' and 'myotatic action' are terms introduced by Gowers, and occasionally employed as designations for the phenomena termed 'tendon-reflex,' or the conditions under which they occur. The term 'myotatic' was proposed as involving no theory of the nature of these phenomena, but merely the unquestionable fact that they can only be elicited when the muscles are in a state of moderate extension, usually passive. See SPINAL CORD, Diseases of.

MYXCEDEMA (μύξα, mucus; and οἶδημα, a swelling).

DEFINITION.—Myxcedema is a disease depending on loss of function of the thyroid gland, characterised by remarkable changes in physiognomy, and associated with many signs of nervous disorder. It appears to be identical with cachexia strumipriva (Kocher) and sporadic cretinism (Curling).

ÆTIOLOGY.—That the disease depends on loss of function of the thyroid gland is shown by the facts that the thyroid gland is constantly found to be atrophied, that a condition similar to myxcedema can be artificially produced both in man and the lower animals by extirpation of the gland by operation, and that the symptoms of the disease can be removed by the administration of thyroid substance from one of the lower animals, and that they return when treatment is suspended. In some cases atrophy of the gland has been preceded by hypertrophy, and sometimes myxcedema has succeeded exophthalmic goitre.

The cause of the atrophy of the thyroid gland is at present unknown. Among common antecedent conditions have been observed profuse hemorrhage, menorrhagia, persistent and severe vomiting, chronic diarrhoea, frequent child-bearing, prolonged lactation, continued mental worry, shock from injury or accident, acute rheumatism and erysipelas.

As in the case of exophthalmic goitre, females are affected more than males (7 to 1). The disease

may occur at any age. In children the malady specially affects growth of body and development of intellect, and in consequence differs somewhat from the adult form, and is described as sporadic cretinism. In adults the disease usually comes on after the age of thirty. Out of 156 cases in which the age at which the disease began was noted, 5 commenced before 20, 24 between 20 and 30, 50 between 30 and 40, 47 between 40 and 50, 24 between 50 and 60, 6 over 60. Thus nearly two-thirds of the cases originated between 30 and 50. As regards the ages at which the disease is met with, it may be observed that up to middle life the disease increases in frequency, after which it gradually diminishes again. Of 231 recorded cases, only 5 were under 25, all females, 34 were between 25 and 35, 60 between 35 and 45, 60 between 45 and 55, 71 between 55 and 70, and 2 over 70. Social position appears to be without influence. Of 109 affected females, 91 were married and 73 had borne children. In a significant number of cases the disease has been found to affect several members of the same family, and there may be direct transmission from father or mother.

ANATOMICAL CHARACTERS.—The body is increased in bulk and there is an excess of subcutaneous fat, and of fat elsewhere throughout the body. A slight degree of anasarca is not uncommon, and passive effusions into the serous cavities have been frequently noted. The thyroid gland is reduced in size, and has been variously described as pale yellowish-white, or buff-coloured, firm, tough and indurated, fibrous and structureless. Atheroma of the larger arteries and interstitial changes in the kidneys are not infrequent. Tuberculosis has been present in a number of the recorded cases. Occasionally the pituitary body has been hypertrophied. The change in the structure of the thyroid gland appears to be of one kind in the very large majority of cases. An early stage of small-celled infiltration of the walls of the vesicles is accompanied or followed by epithelial proliferation in the vesicles themselves. Later on, the gland becomes converted into a delicate fibrous tissue through which are scattered clumps of small round cells, epithelial cells and colloid masses, clearly the remains of the vesicles, until finally the gland-substance is almost entirely replaced by dense fibrous tissue, in which only a few small islets of round cells can be detected. In some parts of the gland the changes may be more advanced than in others. The changes in the skin and tissues which bring about the striking physiognomy of the disease are secondary to the affection of the thyroid. A remarkable overgrowth, consisting in part of hyperplasia, in part of retrograde degeneration of connective tissue, is found in several or all parts of the body. The fibrillar constituent of ordinary connective tissue is everywhere increased, and its elements unnaturally defined, so that the bundles are largely broken up into individual fibres; the corpuscles are enlarged and multiplied; and the interstitial cement is augmented. In the skin there occurs a nucleated fibrous growth in the tissue outside the coiled tubes of the sweat-glands, the lumina of which are obliterated by the swelling and proliferation of the epithelium. A similar change may be observed in the sebaceous glands, which are represented by irregular masses of nuclei. The hair-follicles are surrounded by a cellular growth, external to which nucleated fibrous tissue sometimes develops. Vacuolation of

a considerable proportion of the fat-cell nuclei has been observed. The same sort of interstitial expansion is found in the mucous membranes, in glands of all kinds, in muscles, and in the central ganglia of the nervous system, subjecting the proper structural elements of each tissue to destructive pressure. It is most developed of all, perhaps, in the outer coat of arteries. In the brain, in some cases there appears to be a general increase of neuroglia, and a very considerable growth of the connective tissue around all the vessels. It must be observed that these appearances, while present in the case of persons dying in the full development of the disease, are less marked or absent in cases where the characters of the disease have been altered by treatment or by changes preceding death.

The name myxœdema was given to this disease by Ord, because in his first investigations he found that the skin yielded a greatly increased amount of mucin. Later chemical investigations have shown that increase of mucin is by no means a constant, and seldom a marked, feature. In acute experimental myxœdema in animals, however, a decided increase of mucin has been observed.

SYMPTOMS.—In myxœdema the physiognomy is the first characteristic. The face is swollen in every feature, so as to suggest the existence of renal disease to an inexperienced observer. The distribution and quality of the swelling are however strikingly different from what is observed in common dropsy. The swollen skin is singularly waxy-looking and anæmic. The œdema is resilient, does not pit on pressure or shift by gravitation, and affects dependent and non-dependent features equally. Thus the upper and lower eyelids, and the upper and lower lips are uniformly enlarged; the *alæ nasi* are thickened and broadened; the ridges of expression are blurred and coarsened, or the lines obliterated. There is a characteristic raising and arching of the eyebrows to compensate for the swelling of the lids. The cheeks are overspread with a well-defined dull pink flush in vivid contrast with the pallid skin around.

The conditions observed in the face prevail throughout the body. There is a general increase in bulk and weight, and the abdomen is large and pendulous. The skin is thickened, dry, scaly or branny, and rough to the touch, perspiration being infrequent or absent. The hairs begin to fall out at an early stage, and are ragged and broken. The scalp is scurfy and often bald, the eyebrows thin. The teeth decay, break, or fall out. The hands lose all shapeliness and expression, and become broad and clumsy looking, or 'spade-like.' The feet are similarly affected, and both are cold and often purple or livid. Warty growths and moles are not infrequently present on the body and limbs as well as on the face. There is often a decided fulness above the clavicles. Slight œdema about the feet and ankles is common. The tongue is large and thick.

The nervous system is early affected. An ever increasing hebeticity involves sensation, voluntary movement, and intellect. The face wears a fixed heavy expression; the speech is slow and laboured, though not slurred or slovenly; the voice is monotonous, like that of an automaton, and of a peculiar so-called 'leathery' tone. Sensation is slow, yet finally sure. The movements of the limbs are slow, clumsy, and languid; the maintenance of fixed attitudes requires much effort, and the head, for

example, is apt to drop forward on the chest; sudden falls are not infrequent, in some cases leading to rupture of tendons or fracture of the patella. The walk is waddling, and the movements have been compared to those of the hippopotamus. Frontal or occipital headache, rheumatic pains, numbness, tingling or pricking in the extremities are common subjective sensations. In the operations of the intellect, thought and volition are again slow. Patients complain of being unable to perform the daily actions of life with their natural expedition. Nevertheless what they do is well done, and they are acutely conscious of their slowness. In conversation ideas come deliberately, and are tardily expressed. To write a letter occupies an hour, where it would before have taken ten minutes. Yet the language is correct, and the caligraphy unchanged. There is, in fact, an unwieldy state of mind as of body. Impairment of memory, especially for recent events, occurs early in the disease. The temperament is not infrequently placid or torpid, but irritability or fretfulness occurs from time to time in a large number of cases. Excessive drowsiness during the day, with or without sleeplessness at night, is not uncommon. Delusions or hallucinations are present in many cases. Hearing is very frequently affected, deafness occurring on one or both sides, and there are sometimes noises in the ears. Perversions of taste and smell are not uncommon.

The temperature of the body is almost always subnormal, ranging between 98° and 94° F., or even less. Most patients complain of constant chilliness, without appearing to estimate readily any changes of external temperature. The pulse-rate is either normal or slow. The urine is usually lowered in specific gravity and deficient in urea; but as a rule contains no albumen, sugar, or casts. In the later stages albuminuria usually occurs. The catamenia are very frequently irregular. Sometimes there is amenorrhœa, sometimes menorrhagia, sometimes sexual feelings are in abeyance. In many cases there is a decided tendency to hæmorrhages, which may take place from the gums, throat, nose, or uterus. The extraction of a tooth has been followed by prolonged bleeding from the socket and gums. In the later stages the mind often becomes entirely unheinged. The patient becomes morose, suspicious, and more fretful and irritable; delusions or hallucinations ensue if they have not already occurred. A considerable number of cases drift into asylums, with the usual symptoms of acute or chronic mania, dementia or melancholia. Death comes sometimes by coma, or asphyxia, or with the signs of uræmic poisoning, or by inanition, but in the majority of cases is caused by intercurrent disease.

DIAGNOSIS.—As a rule there is little or no difficulty in recognising the disease when it is well established. The malady was originally confounded with chronic Bright's disease, but there are numerous points of distinction. Scleroderma is sometimes mistaken for myxœdema, but the hide-bound skin and mask-like face are quite unlike the condition produced by the latter disease. Acromegaly, with the enlargement of the extremities, has also distinguishing features. See ACROMEGALY.

PROGNOSIS.—The progress of the untreated disease is slow and far from uniform. There may be many alterations in the symptoms, and the swelling of the skin and the nervous disorder may vary considerably from time to time, so that occasionally

the disease may seem to disappear. But unless the remedy is employed, the ultimate prognosis is unfavourable. The disease has been known to last in untreated cases as long as twenty years, but even then life has been cut short by the disease. Since the introduction of the highly successful mode of treatment by means of thyroid gland the prognosis of the disease has become very different. Even after the disease has been in existence a great many years the treatment is in most cases successful in removing the symptoms. The most unfavourable cases are those in which insanity has supervened.

TREATMENT.—Myxœdema is very amenable to treatment. It was shown by Schiff and others that the cachexia following thyroidectomy in animals could be temporarily prevented or diminished by transplanting a thyroid gland from another animal. As, however, the gland did not take root the benefit did not last long. Gley and Vassale next tried the effects of injections of thyroid juice for the same purpose. The first to try such subcutaneous injections in cases of myxœdema was George Murray, of Newcastle, and the results he obtained quickly showed that a most potent remedy had been found for the disease. The next step was the discovery made independently by the present writer, by E. L. Fox, of Plymouth, and by Howitz, of Copenhagen, that the disease could be equally successfully treated when thyroid-gland substance, or an extract prepared from it, was given by the mouth. The last mode of treatment is that which is followed at the present day, and it has been proved to be thoroughly successful in removing the symptoms of the disease.

In starting thyroid-treatment in a case of myxœdema it must be borne in mind that in this disease the reaction of the remedy is much greater than in health. Small doses should invariably be given at first, such as three to five minims of *Liquor Thyroidei* (B.P.), or three to five grains of *Thyroideum Siccum* (B.P.) once a day. Corresponding doses may be given of any of the various preparations, such as thyroid tablets or capsules, and thyro-colloid, which are prepared by various manufacturers. In many cases larger doses need never be given, but in some cases it may be necessary gradually to increase the dose. It is seldom necessary, however, to give more than the equivalent of ten minims of the *Liquor* twice a day. When the symptoms of the disease have disappeared the dose may be decreased. A small dose once or twice a week may prove sufficient to keep the patient in health. If the remedy is suspended for any length of time the symptoms of the disease will slowly return. It must be explained to the patient that it will be necessary to continue the remedy during the whole of life. Certain unpleasant symptoms are apt to show themselves to a greater or less extent during the cure, but these are most likely to occur when the remedy is being pushed, and as a rule indicate that too large a dose is being employed. Gastro-intestinal disturbance may arise with such symptoms as loss of appetite, fetid breath, burning in stomach, thirst, nausea, vomiting, pains in the bowels, and diarrhœa. Frontal headache—sometimes severe and persistent, pain in the back, rheumatic pains and aching in the limbs, burning, tingling, or smarting in the fingers and toes, itching of the skin—sometimes to an intolerable degree, erythema, urticaria, general œdema, and desquamation of the hands

and feet are not uncommon. The temperature, previously subnormal, may become febrile, and the heart-beats rapid and sometimes irregular; while the patient suffers from palpitation. Great care should be taken to keep the patient at rest in the recumbent position if there are any signs of failing heart, as sometimes death has occurred from cardiac failure during the process of cure. Attacks of faintness or of angina have been observed. Giddiness, sometimes directly after taking food, epileptic

attacks or temporary loss of consciousness, emaciation, prostration, sleeplessness, restlessness, sweating, trembling, and swelling of the parotid and submaxillary glands are among other symptoms which have been occasionally experienced. These untoward symptoms will probably prove very rare when the remedy is given cautiously in small quantities. The dose should be diminished at once should undesirable symptoms be produced.

HECTOR MACKENZIE.

N

NÆVUS.—See TUMOURS.

NAILS, Diseases of.—SYNON.: FR. *Maladies des Ongles*; GER. *Krankheiten der Nägel*.

The ungual diseases may be conveniently grouped into: A. Hypertrophies; B. Atrophies; C. Dys-trophies; D. Morbid conditions accompanying certain cutaneous affections; and E. Parasitic diseases.

A. Hypertrophies.—1. *Simple Hypertrophy* of the nail-substance (onychauxis) is a comparatively rare condition. When it occurs the nail is enlarged, and is both thicker and harder than in the normal state, and this excessive nail-formation is due to an over-activity or an increase in size of the nail-matrix. This condition is found in the clubbed fingers of thoracic disease, and in acromegaly and gigantism. See FINGERS, CLUBBING OF.

2. *Hypertrophy with deformity* of the nail is more common than simple hypertrophy and results in the troublesome conditions known as *ingrowing toe-nail*, *claw-nail*, and *ram's horn-nail* (onychogryphosis), names which sufficiently indicate the more common shapes which the deformed nail assumes. In these affections the nail-substance becomes peculiarly hard, from a closer cohesion of its component cells, its healthy transparency is lost, and it becomes opaque and assumes a dirty brownish colour, and the surface becomes roughened by the presence of longitudinal and wavy transverse ridges.

ETIOLOGY.—These deformities of the nail are most frequently met with in elderly bedridden people, but they may occur at any age. Intermittent external pressure from badly fitting boots is a common cause of these conditions, but of equal importance is the neglect of proper cleanliness of the nails, by permitting irritant substances to accumulate between the nail and the nail-bed, which set up an hyperplasia of the nail-substance. Certain chronic nerve-affections such as myelitis, peripheral neuritis, and tabes dorsalis may be associated with the formation of an imperfect nail-substance, and may even produce an excessive growth and deformity of the nail.

PATHOLOGY.—By pressure or injury a widening of the nail-fold is produced which allows the formation of a thicker nail. At the same time the nail-bed is irritated and a horny mass forms on it below the nail, which acts as a barrier to the forward growth of the nail, and by raising it up, more or less determines the various degrees of deformity. The pressure of the ingrowing or claw-nail on the neighbouring soft parts produces in them a condi-

tion of lessened resistance, and a greater liability to secondary inoculation with septic organisms, which play a most important rôle in the evolution of the various onychopathies.

SYMPTOMS.—Pain—usually intermittent, but in some cases constant—is the most important symptom of ingrowing toe-nails. As a rule it is so severe that walking is rendered impossible. The pain results not only from pressure, but also from exposure of the sensitive nail-bed, which occurs when the nail is much raised up. If secondary inoculation take place in the neighbouring soft parts, suppurative, œdema, gangrene, and even sloughing of the nails may occur (*onychia* and *paronychia*).

TREATMENT.—In the milder forms of onychogryphosis, such as ingrowing nails, it is often sufficient to cut the nail as short as possible, and to insert pledgets of gauze impregnated with some antiseptic lotion between the edge of the nail and the inflamed skin. If the lateral pressure by the nail be severe it is necessary to press down the skin of the lateral nail-folds before the pledget can be introduced. Strips of adhesive diachylon-plaster should then be wound tightly round the phalanx, and a rubber-cot put over the plaster. Much benefit may be obtained by touching any bleeding points or granulations with a silver-nitrate pencil. In twenty-four hours the plaster dressing should be removed, the feet bathed in boric-acid lotion, and a new dressing applied. By this treatment the patient is gradually enabled to wear boots again and to walk about, and the possibilities of complete recovery are considerable. Another method of treatment which is sometimes useful is to moisten the nail with warmed liquor potassæ (40 per cent.), dabbed on with the frayed-end of a piece of cane, and when the nail is softened by this means, to scrape it down with a piece of glass till it is as thin as paper, after which its ingrowing edges may be easily lifted up and cut short with scissors. In the severer forms, namely, the claw- and ram's horn-nails, the above treatment is useless, and complete extirpation should be resorted to. The nail can be removed by cutting-pliers or a fine saw, after which the skin should be stitched over the nail-bed, since in these cases the nail-matrix is as a rule irretrievably damaged, and incapable of re-forming a healthy nail.

B. Atrophies.—Complete or partial atrophy of the nail may be a congenital or an acquired condition. Congenital absence of one or more nails (anonychia) is found in association with other mal-developments of the body. True atrophies of the nail, however, are invariably acquired. An idio-

pathic atrophy of the nails, associated with defluvium capillorum or falling out of the hair, has resulted from severe mental shock (Heller).

For a complete loss of the nail to take place a solution of the continuity between the nail-plate and the nail-matrix is essential, and pathological conditions affecting the nail-bed alone do not, as a rule, produce this solution, as the nail-bed is not in organic connection with the nail-plate, but is only mechanically united to it by the fitting together of the longitudinal ridges on the under surface of the nail-plate and the corresponding grooves on the nail-bed. The more common causes of atrophy of the nail admit of grouping under the following headings:—

ÆTIOLOGY AND PATHOLOGY. — 1. *Trauma.* Severe injuries such as bruises, crushes or blows on the nail, by causing a sudden rupture of the blood-vessels underlying the nail-matrix, and an effusion of blood in the posterior nail-fold, frequently cause a disintegration of the nail-matrix and a shedding of the nail. In these circumstances a new nail usually grows. If the injury is less severe only a temporary arrest in the nail-growth is produced and the nail becomes thin and friable, and presents small ecchymoses in its substance.

2. *Nervous Diseases.*—In cerebral paralysis, tabes dorsalis, syringomyelia, leprosy, division of nerves, and other allied conditions, from the vaso-motor disturbance due to the nerve-lesion, a bleeding may occur about the posterior nail-fold, and atrophic nails may result. In cerebral lesions the great toes are the parts most usually affected. The nails generally grow again if they are shed, unless the shedding results from the division of a large nerve such as the median, when the prognosis is unfavourable.

3. *Blood-Stasis.*—In cyanotic states of the extremities due to cardiac lesions, venous congestions from emphysema, vaso-motor irregularities as in frostbite and Raynaud's disease, atrophic changes in the nails, especially in those of the fingers, are frequently observed. The nails are generally only partially affected and become grey, opaque, lustreless, thin and friable.

4. *Purulent Affections.*—A purulent condition of the soft parts about the nail may cause atrophy and shedding (purulent perionychia). Such an occurrence is familiarly seen in whitlow, but it may occur in tuberculosis, syphilis, and leprosy. As a rule the nail grows again, but in a few rare instances a condition has been set up in which it is no sooner re-formed than it is again undermined and shed (onychitis maligna).

5. *Febrile Atrophy.*—During any acute fever a temporary cessation of growth at the nail-matrix may be noted. In convalescence, however, the nails grow rapidly, but the newly formed nail is peculiarly thin from the narrowing of the posterior nail-fold by the pressure of the turgescient vessels, which are so frequently observed in the extremities before the circulation regains its proper tone.

6. *Scarring Conditions.*—Variola, pustular syphilides, and gummata affecting the nail-matrix or the nail-bed may cause a partial destruction of the matrix, and a loss of the nail, and the subsequent scarring may render the impaired matrix incapable of producing a new one.

7. *Wasting Conditions.*—In chronic tuberculosis, Bright's disease, and diabetes mellitus, a malnutrition of the nail-matrix may take place, and the nails

in consequence may become discoloured, soft, brittle, and tend to crumble.

8. *Cutaneous Affections.*—The nails may be shed in dermatitis exfoliativa, pemphigus, and alopecia areata, and they are frequently partially atrophied in psoriasis, lichen planus, pityriasis rubra pilaris, and ichthyosis.

9. *Intoxications.*—Arsenical, lead- and silver-poisoning may arrest the growth of the nails. Workers in chemical works, who have to put their fingers into acids and alkalis, and bakers or grocers from constantly handling sugar, may suffer from a brittle, opaque state of the nails, similar to that presented by several of the chronic cutaneous affections. In the general intoxications the diseased condition of the nails is caused by an inhibition of the nail-formation at the matrix.

C. Dystrophies.—When the function of the nail-matrix is interfered with, the nails frequently become altered in their texture and colour. They lose their shiny appearance, become dull, brittle, and fissured, and often present whitish opaque specks, whitish striæ, punctiform depressions, or grooves. Together with these degenerations in the nail itself the horny layer of the nail-bed is apt to become thickened in front, causing the nail to be raised up towards its free margin, and sometimes giving rise to a spoon-shaped deformity. At other times the nail-substance formed by the imperfectly acting nail-matrix is peculiarly soft, since the actual cells composing it are swollen and only partially cornified, and the appearance to the naked eye of a bluish translucent wedge usually occupying the lunule-region of the nail is produced. These swollen cells tend to dry and shrivel, air collects between them, and the wedge gradually becomes white, opaque and depressed. When individual papillæ of the matrix are affected whitish specks and punctiform depressions are formed; but when the growth at the matrix is interfered with in its whole extent chalky transverse bands and grooves make their appearance. Similar specks and depressions may also be the result of traumatism from attempts to push back the semilunar fold on the posterior aspect of the nail with a sharp instrument. Dystrophies, such as have been described, are often associated with chronic cutaneous affections, but they occur also in the course of both acute and chronic general illnesses, and lines or furrows may recur with, and serve to mark, each successive attack of an illness.

TREATMENT.—Atrophies and dystrophies of the nails must be treated on purely general principles. Local applications, unless where they are specially indicated by the presence of some purulent affection of the soft parts, are of little value. General treatment, suited to the peculiarities of each case, is of the utmost service. Venous stasis about the extremities should be relieved by improving the general circulation. Many of the apparently hopeless nail-atrophies steadily improve under the influence of tonics, good food and change of climate. In all diseased conditions of the nails careful attention to their culture and absolute cleanliness cannot be too much emphasised. Atrophies due to specific causes must be treated accordingly. The prognosis in the case of atrophies resulting from tuberculosis or leprosy is not so favourable as it is in syphilis.

D. Affections of the nails associated with certain cutaneous diseases.—In most of the chronic dermatoses the nails may become affected at some period or other in the course of the

disease, but it will only be necessary to refer here to psoriasis, eczema, and syphilis, as it is in these diseases that an implication of the nails is most liable to take place.

1. *Psoriasis*.—When the nails are affected in psoriasis the nail-bed is as a rule the chief seat of the disturbance, and the nail becomes implicated secondarily. Small psoriatic spots or papules on the nail-bed cause the latter to become inflamed and swollen, and produce on its surface a heaping-up of partially cornified epidermis, which raises up the nail so that a quill can sometimes be passed under it as far back as the matrix. The nail-plate becomes dry, of a dirty-yellowish colour, and dotted over with opaque specks or punctiform depressions corresponding with underlying papules, or it presents chalky striae or irregular furrows. An implication of the nails is said to occur in about 9 per cent. of cases of psoriasis. This type of diseased nail is occasionally observed in the absence of psoriasis of other parts of the skin, and such cases have been diagnosed as psoriasis of the nails; but as marked variations from the above type are the rule rather than the exception, and as a similar condition of the nails is sometimes present in association with other chronic dermatoses, the diagnosis of psoriasis from the appearance of the nails alone is liable to be fallacious.

2. *Eczema*.—In association with eczema it is the nail itself which is the part most severely affected, for while there is also a heaping up of horny material between the nail and the nail-bed, as in psoriasis, the most pronounced disturbance consists of a roughened and indented condition of the nail and a peculiar worm-eaten appearance of its surface. In moist eczemas there is frequently a sero-purulent perionychia about the nail-fold, and the nail becomes soft, yellow, and opaque. As in psoriasis, the typical appearances of the nails in eczema are so frequently modified that it is doubtful if the description or diagnosis of a specific eczema of the nails is warranted.

3. *Syphilis*.—In both congenital and acquired syphilis the nails may be affected. In congenital syphilis bullae occurring about the nail-bed and matrix may cause a partial destruction or total loss of the nail. In acquired syphilis the nail-bed may be the seat of a primary sore, of papules, pustules, or even gummata, in which conditions the nail itself is always more or less destroyed; but there is no typical manifestation in the nail constantly associated with syphilis.

TREATMENT.—General treatment is of great value in the foregoing diseased states of the nails, and should be carried out on ordinary lines, for since errors in metabolism and want of exercise and fresh air are all potent factors in the production and prolongation of many forms of chronic dermatoses, so are they equally important in keeping up these diseased conditions of the nails. Arsenic has been strongly advocated in nail-affections associated with psoriasis, while those connected with syphilis are amenable to antisyphilitic remedies. Locally it is always an advantage to soften and thin the nails before any application is made. This can be efficiently done by putting the affected digits into rubber-cots containing soft soap for about two hours, and then rubbing the softened nails down with pumice-stone. In eczematous conditions after the nails have been thinned they may be dressed with a paste containing oxide of zinc and salicylic acid, or be painted instead with liquor carbonis

detergens, or some other tar-preparation. Where psoriasis is present elsewhere the following ointment has proved of benefit to the affected nails: Chrysarobin 4 parts, ichthylol 4 parts, salicylic acid 2 parts, and vaseline 100 parts, and this is best applied on pieces of lint covered by a rubber-cot.

E. *Parasitic Diseases of the Nails* (Onychomycosis).—The nail may be attacked by the fungi of ringworm and favus. Occasionally the nail-bed near the free margin of the nail may be the seat of pediculi or scabies, and subungual supuration may result from their presence.

1. *Ringworm of the Nails*.—There is no pathognomonic group of appearances which can be said to represent either ringworm or favus of the nails. As a rule in ringworm the nail is infected first at its free margin. It becomes opaque, and of a dirty yellow or brownish colour. The surface is peculiarly rough, and irregular striae make their appearance on it; the nail becomes friable and is apt to split and even to fall off. The nail-bed is a frequent harbour for the ringworm fungus. If the fungus be confined to the nail-bed the latter becomes thick and spongy, and the nail is raised up, but if the fungus spread back to the matrix an inflammation is set up, which almost invariably ends in the shedding of the nail. When the matrix is not affected the fungus may persist in the nail and the nail-bed for an almost unlimited period. Sometimes in old cases instead of the nail being raised up it becomes unusually thin, indented, and adherent to the nail-bed. This affection of the nails usually occurs with ringworm elsewhere. It is generally met with in the finger-nails, and is more common in adults than in children. Ringworm of the nail, however, is a comparatively rare condition, considering the large number of individuals who must by scratching have the fungus deposited beneath the free margin of the nail. The type of fungus which attacks the nail is usually the large-spored variety, but the small-spored fungus has been found in one or two instances infecting the nails of children.

2. *Favus*.—The favus-parasite when it takes root in the nail-bed produces a superficial catarrh of that region, causing a spongy accumulation beset with fungus to form between the nail-bed and the nail. The nail itself, when attacked by the fungus, becomes yellowish, thickened, rough and opaque. Scutula of favus may form on the nail-bed, and the raised nails present dead-looking areas corresponding to the scutula, and it may become brittle and disintegrate. The differential diagnosis of these conditions is only possible by a microscopical examination, for which purpose scrapings or cuttings of the nail should be softened in warmed 40-per-cent. liquor potassae for fifteen minutes, when the keratin of the nail will be found to have dissolved, and the fungus may be easily demonstrated.

TREATMENT.—When the nail-matrix becomes inflamed and suppurates the nail is shed, and if the matrix be not irretrievably destroyed, a new nail grows. This fact gives the key to the treatment of the parasitic affections of the nails. The most rapid treatment of these conditions is undoubtedly extirpation, and in many cases it is the most satisfactory method. Fourteen days after the excision the patient can use his fingers again, and a new and healthy nail grows in about six months. Where for some reason a surgical operation is inadvisable a similar result may be brought about in the following manner: equal parts of pyrogallic acid and

olive oil smeared on lint are applied twice daily as a dressing to the affected ungual phalanges; in about fourteen days the nail-bed and matrix have begun to inflame and even to suppurate, and the nail may have beneath it a lake of pus and be slightly movable; the application of a moist dressing in a rubber-cot will usually cause a separation of the nail from the matrix in another fortnight. This method of Pellizari, though it gives good results when carefully supervised, when carelessly employed may be followed by serious consequences, for if the pyrogallic acid occasions a too active inflammation at the matrix, the destruction so produced may be so great as to render the formation of a new nail impossible. On this account a slower and less powerful method may be resorted to, which is accompanied by no such risk. Moisten the nail with warmed 40-per-cent. liquor potassæ, rub it thin with pumice-stone, or scrape it with a piece of glass after it has been softened by the alkali, then apply a moist dressing consisting of iodised vaseline (iodine, and iodide of potassium, of each 1 part, water and vaseline, of each 100 parts), spread on lint, and cover the dressing with a rubber-cot. This dressing is renewed twice a week, softening and scraping being repeated if necessary, and a cure is generally obtained in several months. Instead of using the iodised vaseline dressing of Sabouraud, a more rapid result may be obtained by employing a 5-per-cent. chrysarobin ointment, or an ointment containing biniodide of mercury, such as the following: Biniodide of mercury 2 parts, castor oil 12 parts, vaseline 100 parts.

J. M. H. MACLEOD.

NAPLES, in South Italy.—Changeable climate. Mean temperature, winter, 48° F. Cold winds in spring. See CLIMATE, Treatment of Disease by.

NARCOSIS
NARCOTISM } (*ναρκῶς*, I benumb).—A condition of profound insensibility, due to the action of certain drugs introduced from without, or of certain products formed within the body. See CONSCIOUSNESS, Disorders of; NARCOTICS; and URÆMIA.

NARCOTICS (*ναρκῶς*, I benumb). —.SYNON.: Fr. *Narcotiques*; Ger. *Narkotische Mittel*.

DEFINITION.—Remedies which induce loss of consciousness, motion and sensation, or a condition of profound sleep. In large doses they produce complete insensibility and coma.

The phenomena attending narcosis are well illustrated in the progressive effects of opium, the longest known and most widely used member of the class. The cerebral functions are influenced progressively from above downwards. A transient mental excitement is followed by drowsiness and somnolence, ending in deep stupor. The centres for motion and sensation are concurrently depressed, and finally, if the dose is excessive, the important centres of organic life in the medulla—respiration and circulation—are paralysed, and death ensues. A large number of narcotics are useless as remedies for insomnia. Their effect on the various centres is either simultaneous, or the depression of the medulla may be in excess of and precede any marked influence on the consciousness or sensorium (Hydrocyanic Acid, Aconite). In other instances the preliminary stimulation of the intellectual functions, though finally ending in narcosis, may be

disproportionate and delirium ensues (Belladonna, Cannabis Indica). Hence, from a therapeutical point of view, all such aberrant members of the group will be of little use or even dangerous for the production of artificial sleep and the relief of pain; and can only be used in restricted doses for special purposes and under special conditions.

The term 'narcotic' is now often used in a limited sense to include all drugs which produce a temporary suspension of the intellect, of motion and sensation; and hence becomes equivalent to hypnotic or soporific. Many new substances have been introduced of recent years with the object of producing narcosis, and at the same time avoiding as far as possible any excess of preliminary stimulation, or any final depression of the cardiac and respiratory centres. It can hardly be said that we possess at present a narcotic which is at once both absolutely safe and invariably efficient, and great judgment must be exercised in their administration. Hence the indiscriminate use of these drugs by patients themselves, or by unqualified persons, is to be deprecated. Tolerance in most cases is rapidly established, and increasing doses are required to induce sleep, so that finally even large doses may be ineffectual. Many serious cases of accidental poisoning have arisen from their abuse, and the various drug-habits are unfortunately only too well known. See HABITS.

The entire group of drugs having a narcotic or deadening and benumbing effect is naturally divisible into three main classes: (1) general anæsthetics, (2) general nerve sedatives (analgesics and anodynes), and (3) hypnotics or soporifics. A drug which in a full dose would be classed as an anæsthetic or narcotic will in a smaller one play the rôle of a mild hypnotic or nerve sedative.

Hypnotics or Soporifics.—The primary indication in the treatment of sleeplessness must necessarily lie in the removal of the cause. Hence many measures which, strictly speaking, are not narcotic, or even medicinal at all, may indirectly procure tranquillity and comfortable sleep, e.g. warmth, quiet, and the removal of any disturbing influences. Sleep is often promoted by dilating the vessels of other parts of the body than the brain, as, for example, by a warm bath or a full meal. Again, the insomnia of fever may be temporarily relieved by the use of antipyretics, such as quinine, phenacetin, phenazonum, or acetanilidum, but the relief is only temporary, and such drugs must not be employed in a routine fashion. The sleeplessness of exhaustion and feebleness may yield readily to a little nourishment or to a well-timed dose of alcohol. Pain is probably one of the most frequent causes of want of sleep, and if it is removed sleep usually follows. This may be often effected by other means than by internal medication, as, for example, by local surgical procedures, by the application of heat and cold, and by the use of local anodynes. It is generally best, for the reasons already stated, to exhaust all local, non-medicinal, domestic and indirect measures for the relief of sleeplessness before proceeding to the use of hypnotic drugs.

A convenient clinical classification of medicines employed for the artificial production of sleep is into (1) those which are used when pain is the chief cause of the insomnia, and (2) those which are indicated when restlessness, mental excitement, overwork and similar conditions are the predominant factors.

In the first group Opium and Morphine are the most efficient narcotics. The principal drawbacks to opium are (1) the disturbance of digestion and of the secretions and excretions attending its use, and (2) the fact that increasing doses are needed to overcome the toleration which is rapidly developed. As a hypnotic morphine is better given hypodermically than by the mouth, as its narcotic action is more quickly induced, its dose is more accurately regulated, and its effect on secretion is less marked. For the last-named purpose a minute dose of atropine is sometimes combined with the morphine. It will be noted that the official hypodermic injection of morphine was made considerably weaker in the last revision of the 'British Pharmacopœia' (1898), and is now a five-per-cent. solution of the tartrate of morphine.

In the second group of hypnotics, suitable for cases where pain is not the essential cause of the insomnia, many useful drugs are included; and of these, chloral hydrate, the bromides, chloralamide, sulphonal and trional are perhaps the most satisfactory.

As a hypnotic, *Chloral Hydrate* acts more quickly than morphine, even when given subcutaneously. It often succeeds when morphine has failed, and is rarely followed by any unpleasant effects on digestion. It does not, however, relieve pain, and may cause great cardiac and respiratory depression. It is useful in cases of mental excitement and mania, but it must be always borne in mind that a chloral-habit, like a morphine-habit, is easily established. *Butyl-Chloral Hydrate* has a somewhat similar action to chloral hydrate, but is less rapid, less powerful, and less certain. It has, however, the special advantage of relieving trigeminal neuralgia in some instances. The *Bromides* are peculiarly well fitted to soothe the brain when rendered irritable by overwork, but are very uncertain in their action as hypnotics. Bromide of potassium is often usefully combined with chloral hydrate. *Chloral-amide* is a fairly certain and safe hypnotic. It is valuable in the insomnia of cardiac disease and in some cases of nervous excitement. Its effects, however, are often very variable, but it has the advantage of not leading to any 'habit.' *Sulphonal* and its congeners *trional* and *tetronal* do not as a rule derange digestion, nor seriously depress the circulation and respiration, and may therefore be safely ordered in diseases of the heart and lungs, where the more powerful hypnotics such as morphine and chloral hydrate are contra-indicated. Their action is, however, somewhat slow and uncertain, and the use of sulphonal may be followed by prolonged drowsiness, giddiness and eruptions. See SULPHONALISM.

FREDERICK WILLCOCKS.

NASAL CALCULI.—See CONCRETIONS.

NASHA FEVER.—A term applied in India to an inflammation of the mucous membrane of the septum nasi, accompanied by fever, and lasting about a week. It is uncertain whether the condition should be regarded as a definite disease.

NASO-PHARYNX.—See NOSE, Diseases of.

NATAL, in South Africa.—Warm, but healthy climate, with hot, wet summers, and dry, clear winters. High winds from S.E. and N.W. Soil, sandstone and granite. See CLIMATE, Treatment of Disease by.

NAUHEIM, in Germany.—Gaseous thermal salt waters. See MINERAL WATERS.

NAUSEA (*ναῦς*, a ship).—An inclination to vomit. See SEA-SICKNESS; and VOMITING.

NAUSEANTS (*ναῦς*, a ship).—DEFINITION. Agents which produce the feeling of nausea.

ENUMERATION.—The principal nauseants are Warm Water, Tartar Emetic, Ipecacuanha, Tobacco, Squill, and Apomorphine.

ACTION.—These substances produce irritation of the stomach, loss of appetite, general malaise, enfeebled circulation, muscular weakness, and frequently also salivation and sweating.

USES.—Nauseants have been employed to diminish appetite, in the hope of causing absorption of fatty accumulations or of pathological deposits. They are also used in producing relaxation of involuntary muscular fibre, and thus accelerating the passage of calculi through the bile-duct or the ureters; and occasionally they have been employed to relax rigidity of the os uteri in labour. They are sometimes given to excite sweating. See DIAPHORETICS; and EMETICS.

LAUDER BRUNTON.

NEAR-SIGHTEDNESS.—See MYOPIA; and VISION, Disorders of.

NECROBIOSIS (*νεκρός*, a dead body; and *βίος*, life).—Molecular death of a tissue without loss of continuity, especially seen in the various forms of atrophy and degeneration. See ATROPHY; and DEGENERATION.

NECROPSY (*νεκρός*, a dead body; and *ψις*, a view).—SYNON.: Autopsy; Fr. *Nécropsie*; Ger. *Leichenschau*.

DEFINITION.—The inspection and examination of the body after death.

Method.—The external surface of the body should first be carefully examined, note being taken of the presence or absence of wasting, the degree of rigor mortis, changes in colour, partial or general, œdema, marks of injury, and other points which may suggest themselves. In medico-legal cases it is of importance to measure, and accurately record on the spot, the precise position of external wounds or marks of injury.

When the external examination is complete the body is opened. The following general principles should be observed: (1) Examine first the viscera *in situ*, and ascertain whether there is any departure from their natural position and relations. When this has been done, and not before, the individual organs may be removed from their connections and subjected to closer examination. (2) Examine all the viscera and accessible organs, i.e. let the necropsy be thorough and complete. Do not neglect the lymphatic glands, the thyroid and suprarenal bodies, or bone-marrow. Many an erroneous conclusion has been drawn as to the cause of death by a partial examination. It happens not rarely that relatives will only consent to a partial examination of the body. Where this is the case it is still possible to examine all the important thoracic and abdominal viscera through a comparatively small external incision, without in any way hurting the feelings of the relatives, since the restriction may be held to refer to external disfigurement only. (3) In all cases it is important so to conduct the

examination as to give rise to as little disfigurement as possible; attention to the rules given below will render this easy. At the conclusion of the examination the organs should be replaced, all incisions closed by a neat continuous suture, and the surface of the body cleansed. When it is necessary to conduct the necropsy in a private house, care must be taken to avoid needless soiling of linen or furniture. (4) It often happens that a bacteriological examination is required. If the individual conducting the examination is not himself competent to undertake this, he should secure the presence of a bacteriologist at the time the body is opened. Blood, for instance, should always be collected from the unopened heart *in situ*. For ordinary histological purposes portions of suspected organs should be placed as soon as possible in Müller's fluid or some suitable hardening agent.

The order in which the different regions of the body are examined varies with the practice of the individual making the examination. Some prefer to commence with the central nervous system, others leave this till last. In all cases the thorax should be examined before the abdomen, because the pressure-relations of the thorax are much altered by removal of the abdominal viscera. In this article we commence with the central nervous system, but it must be understood that in any given case there may exist reasons for beginning in some special region. Where the spinal cord has to be examined it is usually desirable to complete this before exposing the brain.

Head.—To open the head, make an incision down to the bone, above the vertex from the base of one mastoid process to the other, and reflect the scalp backwards and forwards; then divide the bone all round with the saw, beginning in front a little above the level of the superciliary ridge. The posterior half of this section should make an angle with the anterior half by being brought over the occipital bone, a little behind the apex of the lambdoidal suture. By this means the skull-cap will, when replaced, rest firmly in its position without slipping back, and so causing disfigurement of the forehead. In cases of fracture of the skull the section should be completed with the saw, care being taken not to wound the dura mater. Under other circumstances the inner table may be conveniently divided with a chisel and mallet. The skull-cap must now be forcibly dragged off; if very adherent to the dura mater, a long flexible spatula may be introduced between them, and separation thus effected.

In young subjects before the sutures and fontanelles are united, it is better to remove the dura mater and skull-cap together, by dividing the former with blunt-pointed scissors in a line with the section through the bone, and then cutting through the falx at its anterior and posterior attachments.

The longitudinal sinus may now be opened and examined. The dura mater should next be divided on each side with blunt-pointed scissors, on the level of the section through the bone, and the two lateral flaps turned up; then the falx should be cut near its anterior attachment, and the whole membrane drawn backwards off the hemispheres. The brain must now be removed; a long narrow scalpel being used to cut through the nerves and vessels, while the tentorium is most safely divided with blunt-pointed scissors. The spinal cord should be cut across as low as possible. Any fluid present at the base of the skull should be drawn off with a syringe and measured.

Brain.—The pia mater and surface of the brain should now be examined; for this purpose it is rarely needful to strip the pia mater off the brain, though this can be done when required. The brain should be placed on its base, and, if very soft, supported by a towel wrapped round it. A horizontal incision should be carried through each cerebral hemisphere, on a level with the upper surface of the corpus callosum, from within outwards, not quite reaching the surface, so as to leave the hemispheres still attached to the rest of the brain. These should be turned aside, and numerous vertical incisions made in the upturned surface. Each lateral ventricle should then be opened by a vertical incision through its roof, and any fluid contents withdrawn by a syringe. The fornix should now be divided in front, and with the septum and corpus callosum turned backwards. The velum interpositum and choroid plexus being reflected in a similar manner, numerous longitudinal incisions should be made in the corpora striata and thalami optici, and in the corpora quadrigemina. An incision should now be made through the superior vermiciform process of the cerebellum, so as to lay open the fourth ventricle. The cerebellum may be examined by making parallel incisions on each side through its lobes, not quite detaching the sections. The brain may now be folded together again, and the under surface turned up and examined. Incisions should be made into the under surface of the cerebral lobes, and into the crura and pons; and the medulla divided transversely at different levels. Softened portions should be tested with a stream of water; and parts reserved for microscopical examination at once placed in a hardening solution, such as chromic acid (1 per cent.).

In some cases, however, where it is requisite to determine with the greatest attainable accuracy the precise limits of a cerebral lesion, it is desirable to harden the entire brain before opening it. This is best done by placing it, suitably supported by a bed of tow or cotton-wool to prevent deformation, in a 10-per-cent. solution of formalin. In a few weeks it may be examined by the sections above described, with much more satisfactory result than in the fresh condition.

Base of Skull, Orbit, and Internal Ear.—The base of the skull and its sinuses may now be examined. In cases of fracture, the dura mater should be carefully stripped off, so as to expose the surface of the bone. The contents of the orbit may be examined by removing its roof. The tympanum can be opened by cutting through with a chisel the plate of bone forming its roof. This is situated on the anterior surface of the petrous bone, just in front of the eminence of the superior semicircular canal. To examine the internal ear the petrous bone must be removed. This is best done by two converging incisions made with a saw, and then separating the apex of the wedge from the sphenoid and occipital bones with the chisel.

Spinal Cord.—To examine the spinal cord the body must be turned on its face, with the head hanging over the table, and a block placed under the chest. An incision must be made over the vertebral spines from the top of the sacrum to the occiput, and the vertebral arches laid bare. These are best divided with the rachitome, a double semicircular saw, in the absence of which a short common saw may be used, or a chisel and mallet or bone-forceps. The cord should be removed in its tube of

dura mater, the latter being held by the forceps, and care taken not to bend the cord abruptly. The dura mater should then be slit open with blunt-pointed scissors along its anterior and posterior surfaces, and the cord examined, with as little handling as possible, by means of transverse sections made with a sharp scalpel. For microscopical examination the cord may be placed in spirit for about twenty-four hours; and then, after removal of its membranes, cut into lengths, and transferred to a 1-per-cent. solution of chromic acid. A still better method of hardening the cord for microscopic purposes is to hang it up in a 10-per-cent. solution of formalin. In this it may be kept till it is cut, or it may be transferred after a week or two to Müller's fluid.

A method of opening the spinal canal from the front, preferable in many respects to the above, is practised at Vienna and many places on the Continent. The instruments used are a strong knife-shaped chisel, with a cutting beak, and a mallet. After the removal of the thoracic and abdominal viscera, the beak of the chisel is introduced into the lowest intervertebral foramen, and by successive blows of the mallet the pedicles of the vertebrae are cut through on each side and the canal exposed by removing the bodies. In this way great disfigurement of the body and soiling of the table and linen are avoided, and the spinal ganglia are more easily examined.

Thorax and Abdomen.—The thorax and abdomen should now be examined. It is better to lay the abdominal cavity fully open before removing the sternum. In cutting through the first rib, and disarticulating the clavicle, care should be taken not to wound the innominate vein. By using cutting pliers, which should be directed so as to cut obliquely through the rib into the articulation, all danger is avoided.

If much ascites is present, the belly should be tapped before laying open the peritoneal cavity. So, if either pleura be full of fluid, which will be shown by its pouring out when the cartilages of the ribs are cut through, sufficient should be drawn off with a syringe to prevent any overflow when the sternum is removed.

The lungs should now be drawn out of the chest, adhesions separated, and their posterior surfaces examined. The contents of the mediastinum should next be inspected, and the pericardium opened. If the case be one of thoracic aneurysm, mediastinal tumour, or malformation of the heart or great vessels, the heart and lungs should be removed together. Otherwise, the heart may be first removed and examined.

Heart.—The auricles should be laid freely open with a pair of scissors, by an incision joining the mouths of the great veins and carried to the extremity of the auricular appendage. The competency of the valves may then be tested. All clots must first be removed, the heart held in an upright position, and water poured into the aorta and pulmonary artery successively, the semilunar valves being held back with the handle of a scalpel to allow the ventricle to become filled; on looking into the auricles the competency of the auriculo-ventricular valves may be estimated. To test the semilunar valves an opening must be made into each ventricle; the pulmonary artery and aorta cut sufficiently close to enable the valves to be clearly seen; and then water poured into these two vessels successively,

and the valves looked at from above. The right ventricle may now be opened. The left forefinger should be introduced through the pulmonary artery, and the anterior wall of the ventricle divided with blunt-pointed scissors into the artery, the point of the scissors being guided by the left forefinger to the junction of the valves. The pulmonary artery and aorta should then be separated as much as possible, and the left ventricle opened in a similar manner along its anterior wall, the left forefinger as before guiding the scissors to the point of junction of the semilunar valves. The incision must be carried close to the ventricular septum, and the septum between the aorta and pulmonary artery, but without cutting the latter. The most accurate way of measuring the capacity of the orifices is to pass through them graduated balls fixed on rods, in default of which the fingers may be used. Before concluding the examination of the heart, the condition of the coronary arteries, and of their origins from the aorta, should be investigated. They often share in the atheromatous changes which have occurred in the aorta, and in some cases sudden death seems to be attributable to their blockage.

Lungs.—To remove the lungs, the trachea must be cut across at the root of the neck, and well drawn forwards by inserting the middle finger into the lower end, and the other fingers on each side behind the bifurcation, care being taken not to cut the œsophagus.

To examine the lungs, if the lobes are firmly bound together by adhesions, it is best to carry an incision in a vertical transverse plane from the outer border inwards towards the root. Further incisions may be made parallel to the first, in front and behind it. Cuts made in this direction lie in the plane of the large vessels and air-tubes.

If the lobes are separate, the incisions should be so managed as to give the largest possible sectional area. The lower lobe, as before, should be cut from without inwards, but it is usually more convenient to commence the incision for the upper lobe by inserting the knife in the septum between it and the lower, and carrying the incision midway between its root and its external surface. The vessels and bronchi should be slit up by probe-pointed scissors. The condition of the lymphatic glands at the root of the lung should always be observed and noted.

Larynx and Pharynx.—To remove the larynx and pharynx, the incision in the neck must be carried up to the chin; the floor of the mouth opened from below; the left forefinger introduced, and used to depress the tongue; a long narrow scalpel introduced above the finger, and carried along each side of the ramus of the jaw; the tongue then drawn down under the chin; and the soft palate and pharynx divided transversely. The pharynx and larynx should then be opened along their posterior walls.

The œsophagus, which should have been left undamaged in the removal of the other thoracic viscera, must also be opened and examined. There are many who make a routine practice of removing *en masse* the tongue, fauces, larynx, pharynx, and thoracic viscera, and this proceeding has much to recommend it, as it allows of a more complete examination of the relations of the different parts. After removal of the sternum and study of the thoracic viscera *in situ*, the tongue and fauces are dissected out, and the parts then removed from the vertebral column from above downwards, the œsophagus and aorta being divided at the diaphragm.

The pharynx and œsophagus are then laid open from behind. The larynx, trachea and bronchi are next similarly treated, as is the descending thoracic aorta. The heart and lungs can finally be studied in detail.

Intestines.—In examining the abdomen it is most convenient to begin with the intestines. The large intestine should be divided between two ligatures below the sigmoid flexure, and drawn out, cutting the mesentery close to the bowel. This process should be continued till the duodenum is reached, when it may be again tied and cut. The intestine should be opened along the line of attachment of the mesentery. Before the bowel is opened, it is convenient to wash out its contents. This may be done by tying the upper end to a tap and turning on the water for a few minutes. The mesentery and its lymphatic glands must receive due attention, especially as to the presence of tubercular lesions in the latter. The rectum must finally be examined as far as the anus.

Spleen.—The spleen may next be examined. It should be drawn forwards out of the abdomen, and the gastro-splenic omentum cut through. It should then be placed on its hilum and laid open by a vertical incision.

Stomach.—The stomach should next be removed. A double ligature should be placed round the duodenum about two inches below the pylorus, and another one round the lower end of the œsophagus, and these tubes cut through, so as to remove the stomach without the escape of its contents. If required for chemical analysis, the contents should be emptied into a glass vessel, by removing the œsophageal ligature.

The usual practice is to lay open the stomach along its lesser curvature, from the œsophagus to the duodenum; but in many cases it is better to carry the incision along the greater curvature, for, as ulcers and cancers are more frequently situated near the lesser curvature, this incision is more likely to avoid cutting through them.

Unless required for chemical analysis, the mucous membrane may be washed by a gentle stream of water and then examined.

Pancreas.—After the removal of the stomach the pancreas may be conveniently examined. Before separating it from the duodenum the condition of its duct should be ascertained.

Liver.—In all cases of jaundice the liver and duodenum should be removed together, so as to obtain the bile-duct intact. In removing the liver care should be taken not to injure the right suprarenal capsule, which is in close contact and often adherent. In testing the perviousness of the bile-ducts it is better not to squeeze the gall-bladder, as this will often overcome an obstruction, but to open the duct with scissors, and observe the colour of the lining membrane below an obstruction. This will be found unstained by bile, if the obstruction be complete.

To examine the interior of the liver a number of vertical incisions should be made through the organ extending nearly to the posterior border. The thickness of the capsule, appearance of the surface, and condition of the vessels and ducts on section should be carefully noted. The gall-bladder should also be opened up, and its contents and inner surface observed.

Suprarenal Capsules.—The suprarenal capsules should be removed united with the semilunar ganglia and solar plexus.

Genito-urinary Organs.—In all cases of urinary obstruction the kidneys, ureters, and bladder should be removed in connection. The pelvic organs may be removed *en masse* by carrying a large knife all round the pelvic walls, and drawing the viscera upwards and backwards. As much of the urethra as may be required can be pulled back under the pubic arch. The urethra and bladder should be opened with scissors along their upper wall.

The uterus may be examined by introducing one blade of a pair of probe-pointed scissors through the os; making an incision through the anterior or posterior wall to the fundus; and carrying this on each side to the entrance of the Fallopian tubes, which will be studied along with the ovaries.

The kidney may be bisected by an incision through it from the convex border to the hilum; the capsule should then be stripped off, its thickness and degree of adhesion being noticed; and the state of the surface of the kidney, both external and on section, carefully observed.

The *thyroid gland* and the *thymus* or its remains will have been examined with the thoracic viscera. The *testes* may readily be pulled up from the scrotum and incised. In certain blood-diseases it is of importance to ascertain the condition of the *bone-marrow*. The sternum and the ribs offer special facilities for this, but it is sometimes desirable to examine one of the long bones. A slight prolongation of the abdominal incision will easily enable a section of the upper end of the femur to be made with a saw, or, if preferred a tibia or other long bone may be removed and sawn down. The knees and great-toe joints should be examined for gouty deposits.

There are certain rough chemical reactions which are frequently required in the *post-mortem* room. The chief of these are the iodine-test for amyloid disease, and the iron-reaction in pernicious anemia. Both tests are simple. In the first a section of the suspected organ is made with a clean knife; a weak solution of iodine is then poured over the surface, and, after a minute or two, washed away with a current of water; any amyloid material present stains a dark mahogany-brown colour, in striking contrast to the pale yellow of the other tissues. The iron-reaction is brought out on a similar fresh section of the liver or other organ by pouring over it successively a solution of ferrocyanide of potassium, and then dilute hydrochloric acid. Prussian blue is formed if any notable amount of iron be present in the tissue.

W. CAYLEY.

F. W. ANDREWES.

NECROSIS (*νεκρός*, a dead body).—The absolute death of a circumscribed portion of any tissue: clinically the phrase is usually associated with death of bone. See BONE, Diseases of.

NEGRO-LETHARGY.—SYNON.: The Sleeping Sickness of the Congo; Fr. *Maladie du Sommeil*; *Nélaiane*; *Didane*.

DEFINITION.—An endemic disease of the West Coast of Africa, affecting the central nervous system; characterised by slowly developed and increasing muscular debility, torpor and somnolence; and terminating, after a variable period of months or years, in death.

GEOGRAPHICAL DISTRIBUTION.—This disease is endemic, and liable to epidemic outbursts, in Western Africa between Senegambia in the north,

and S. Paul de Loanda in the south. How far it extends into the interior of the country is not known. It occurs as high up the Congo as Stanley Pool, and is said to be gradually spreading to the upper reaches of this river and its affluents. A case has been reported as high up the Niger as Timbuctoo. It is said to be unknown on the lower part of the Niger (Crosse), at the mouth of the Congo, at Sierra Leone, and at other places in the endemic area. It is just possible that it occurs among the aboriginal Buck Indians in British Guiana, South America. Formerly it was common as an imported disease among the negroes in the West India Islands; since the abolition of the slave-trade it is unknown there. Cases have occurred in England, also in West Coast Negroes. A striking peculiarity about the disease is that it may remain latent for a very long time, and not declare itself until years after the endemic area has been quitted. According to the natives, the liability continues for seven years.

ÆTIOLOGY.—Sleeping sickness has been attributed, on very insufficient grounds, to a variety of causes, none of which, however, on investigation, can be found to account for it. There are some grounds, however, for suspecting that it is in some way connected with the *Filaria perstans*. Certain it is that this parasite has been found in the blood in a large proportion of the cases in which it has been properly searched for. Such a cause would explain the peculiarities of the endemicity of the disease, and also the singular liability to its development years after the victim has left the endemic area. The recent discovery of this filaria among the Buck Indians of British Guiana, and the possibility, as reported by Ozzard, that sleeping sickness occurs among them, seem to indicate a causal relationship.

ANATOMICAL CHARACTERS.—Manifestly sleeping sickness is a brain-disease, and in two cases of which the organs were recently examined under the most favourable conditions, Mott showed that the essential lesion is an extensive meningo-encephalitis, sections of the brain showing extensive and possibly general infiltration of the sheaths of the blood-vessels with leucocytes. No gross lesion was discovered in either case.

SYMPTOMS.—Negro-lethargy attacks both sexes and all ages; it is stated to have a predilection for the young, vigorous, and intelligent of about eighteen or twenty. It commences insidiously with lassitude, muscular and intellectual debility, often moroseness, and an irresistible tendency to fall asleep at unwonted times and even while at work. Dull headache is sometimes complained of, but not always. A tottering and unsteady gait, as if from weakness, is a frequent and early symptom, as is also a peculiar and pathognomonic *facies*: the upper eyelids droop as if weighed down by sleep, the eyes are lustreless and the face puffy, and the expression is sad or taciturn. The memory becomes weak and the senses dull. Little by little, sometimes interrupted by deceptive periods of arrest or improvement, the state of torpor becomes intensified, so that after a time sleep is nearly continuous; or, if not asleep, the patient will lie with closed eyes in an apathetic condition from which he can be roused with difficulty. He may generally be got to reply to questions, but he is unable to sustain a conversation, and speedily relapses into his habitual state of lethargy. At this stage, were he not

roused to take food he would starve to death; even after being roused up, so great is the somnolence that he may fall asleep again in the act of conveying food to his mouth or during mastication. There may be some evening rise of temperature; but for the most part the skin is abnormally cold, the patient evidently feeling chilly and liking to lie asleep in the hot sun. Examination fails to detect any disease of the thoracic or abdominal viscera; the fundus oculi is healthy; and the superficial and deep reflexes are preserved. Although appetite and digestion generally continue unimpaired, towards the end of the disease the body wastes; the sphincters may fail to act; and extensive bed-sores may form. Limited areas of skin may become anæsthetic. Muscular tremor is frequently noted; and as the disease advances, localised muscular spasms or more general convulsions may supervene. Death may occur during one of these convulsions, or it may be brought about by simple inanition or by some intercurrent disease. A certain proportion of the cases exhibit maniacal symptoms at an early stage; these may subside, or recur, or persist for a variable period before the development of the characteristic somnolence. Enlargement of the cervical glands, and of the salivary glands with a degree of salivation, and an itching papular or papulo-vesicular eruption on the chest and limbs are said to be almost invariably observed.

The symptoms described are not all present in every case, and the individual features vary much in different instances, in degree and combination and rate of progress. Progress may be rapid or slow, so that the duration of sleeping sickness is variously stated at from four or five months to as many years. Cases are on record in which recovery seemed to take place, to be followed, however, almost invariably, sooner or later, by relapse and death. It is doubtful, indeed, if permanent recovery ever really does take place. The negro smitten with sleeping sickness considers himself and is looked on by his companions as doomed.

In the districts in which this disease occurs the distribution of the cases appears to be most capricious. A dozen negroes may be sleeping to death in one village, while the neighbouring villages are, and continue to be, entirely exempt or only slightly affected. Similarly, it seems to cling to particular houses and families, and thereby acquires a false appearance of heredity. So terrible are its visitations that whole villages are decimated by it, and entire districts abandoned from the fear of it by their panic-stricken inhabitants.

DIAGNOSIS.—Negro-lethargy has been confounded with beriberi, a disease also endemic on the West Coast; but, if it be borne in mind that the former is a disease of the central, whereas the latter is a disease of the peripheral nervous system, mistake is not likely to be made. See BERIBERI.

TREATMENT.—No treatment has been found to be of any real and lasting service in negro-lethargy, though purging appears to do good temporarily, and arsenic in very large doses has seemed to be followed by improvement, and even arrest. If in the future it should turn out that the *Filaria perstans* is in ætiological relationship to the disease, much may be expected from an intelligent prophylaxis. See FILARIASIS. PATRICK MANSON.

NENNDORF, in Prussia (Hesse).—Cold sulphurous lime-waters.

NEOPLASMS (νέος, new; and πλάσσω, I mould).—A term for new-growths. See TUMOURS.

NEPHRALGIA.—DEFINITION.—The name applied to a variety of renal pain unaccompanied by any ascertained lesion in the kidneys, or by any morbid changes in the urine. Nephralgia is said to be dependent on exhaustion, on exposure to cold, and, according to Grainger Stewart, on the action of malarial, rheumatic, or gouty poisons. The pain is essentially of the same character as that met with in one variety of renal colic. It is felt in the loin and over the anterior surface of the abdomen, but it does not radiate into the groin nor down the thigh. It is usually constant but is liable to exacerbation, especially on exertion; and in this respect also it resembles closely the pain of renal colic, and may be indistinguishable from it if the latter is unaccompanied by the presence of blood or other abnormal constituents in the urine. Most cases of nephralgia occur in neurotic or neurasthenic patients. In many cases where the severity of the nephralgia has led to the diagnosis of renal calculus, nephrotomy and even nephrectomy have been performed, and although no calculus was found, yet a permanent relief of pain followed the operation. It is probable that some cases of so-called nephralgia are really dependent on pre-natural mobility of the kidney causing kinking of the renal vein, and that the distension of the organ so produced has caused the pain. In other cases it is possible that the pain may be dependent on slight anomalies of the ureter causing slight hydro-nephrosis. In both instances the fixation of the organ, produced as a result of the operative interference, may prevent the recurrence of the pain.

DIAGNOSIS.—Nephralgia should not be accepted as a diagnosis until all possible means have been adopted to exclude the presence of calculus, hydro-nephrosis or movable kidney.

PROGNOSIS.—In many cases the discomfort is relatively trivial; in others the severity of the pain is such as to produce a condition of neurasthenia, and this is especially liable to occur if the patient has sought relief by taking morphine or other drugs for the relief of the pain.

TREATMENT.—In mild cases counter-irritation and the support of the organ with a well-fitting pad is all that is required. If the pain produced is very severe an exploratory nephrotomy may not only be justifiable but even advisable.

JOHN ROSE BRADFORD.

NEPHRITIS (νεφρός, the kidney).—A general term for inflammation of the kidney. See BRIGHT'S DISEASE; and KIDNEYS, Diseases of.

NEPHROLITHIASIS (νεφρός; and λίθος, a stone).—The formation of stone in the kidney. See CONCRETIONS; and RENAL CALCULUS.

NEPHROPTOSIS (νεφρός; and πῶσις, falling). Prolapse of the Kidney. See KIDNEY, Malpositions of, p. 835.

NERIS, in France (Allier).—Feebly mineralised, alkaline, saline, thermal waters. See MINERAL WATERS.

NERVES, Diseases and Injuries of.—SYNON.: Fr. *Maladies des Nerfs*; Ger. *Nervenerkrankheiten*.—Nerves, in their origin, course, and distribution, are connected with the several organs and tissues of the body, and are consequently

affected in various ways when such parts are disordered or diseased. But, besides such *secondary* derangements, nerves are subject to many morbid conditions which affect them *primarily*. In the case of certain classes of nerves, connected with special functions, the effects produced by disease are at once so distinct and so important that they require separate consideration. Such, for example, are the glosso-pharyngeal, hypoglossal, olfactory, optic, phrenic, pneumogastric, spinal-accessory, sixth and third cranial nerves, the morbid conditions of which will be found fully discussed under their respective headings. Again, certain forms of congestion or inflammation (whether occurring in the subjects of gout, rheumatism, malaria, plumbism, syphilis, or in other states), when they affect important nerves, cause symptoms of a character so marked, either in their progress or distribution, or by their severity, as to deserve a special designation, and to demand separate description (see INTERCOSTAL NEURALGIA; NEURITIS; SCIATICA; and TIC-DOULOUREUX). In these and in other allied instances the prominent symptoms are referable to functional disturbances of the nerves. In another class of cases similar phenomena originate in interference with the general nutrition, in disease of the nervous centres, or by reflex action; and these phenomena will be found discussed in the articles upon CONVULSIONS, EPILEPSY, and NEURALGIA.

In this place there remain for special consideration the following subjects: (1) the effects of *injuries* of nerves; (2) the most common morbid growths involving nerves, which are generally known as *neuromata*; and (3) the effect of cutting or stretching nerves regarded as a means of *treatment*.

1. Nerves, Injuries of.—Nerves may be divided accidentally either by tearing or cutting, or surgically during an operation, or for the relief of pain or resection of tumours. The nerves most frequently divided accidentally are those of the upper extremity, especially the ulnar, and the median just above the wrist-joint. The injury is very often caused by broken glass.

Sometimes, besides being wholly or partially divided, nerves may be bruised, or have embedded in their substance particles of friable foreign bodies, such as glass or slate. Fractures of the humerus at the upper or lower third are not uncommonly complicated with injury of the musculo-spiral nerve by the sharp edge of one of the fragments; for the nerve passes spirally round and in close contact with the humerus, first on the inner, then on the hinder, and near the elbow at the outer aspect of the bone.

SYMPTOMS.—The symptoms of the division of a nerve are loss of power in the muscles, and of sensation in the skin, supplied by the branches of the injured nerve. The complete or the partial division may be diagnosed by the more or less complete interruption of these functions. It should, however, be remembered that there is often not complete anaesthesia, and that the state of the muscles is more important than is the loss of sensation.

In addition to the paralysis of motion, the *muscles* atrophy with great rapidity, and in many cases shrink so as to cause various deformities. In other cases deformity results from the unbalanced active contraction of the neighbouring healthy muscles. Examined electrically, faradic contractility is found

to be diminished as early as the second day, and completely lost from the third to the sixth. The galvanic contractility remains for from three to twelve weeks, but abnormally strong currents are required to produce contractions in proportion as the muscular tissue disappears. The so-called 'reaction of degeneration' is evident within a week or two of the section, and indicates with certainty that the muscle is for the time separated from its nerve-centre. See ELECTRICITY IN MEDICINE.

The *skin* and its appendages also suffer. At first, on account of the section of the vaso-motor nerves the parts are flushed and hot; but, within a fortnight or three weeks, there is established a spasm of the small arterioles, resulting in diminished blood-supply and coldness of the paralysed parts. In many cases the skin becomes red, shiny, and glossy; and ulcers or whitlows may form painlessly, originating in some cases from injury to an anæsthetic part, in other cases being apparently spontaneous. The *nails* become fibrous and brittle, with ridges and fissures on them, and in some cases are shed. The *hair* breaks off short or else falls out. The *joints* are at first swollen and painful, but later on are liable to become stiff and ankylosed. The *fat* and *subcutaneous tissues* atrophy.

Changes in the divided nerve.—The lower end of the divided nerve undergoes rapid atrophy and degeneration, the myelin being broken up and absorbed and the axis-cylinders quickly destroyed. This degeneration affects simultaneously the whole of the lower end and its branches, but is in all cases succeeded after an interval of some months by imperfect attempts at repair, and the formation of new axis-cylinders from the nuclei of the sheath of Schwann. The proximal end as a whole undergoes no change, except a slow atrophy which ensues after a lapse of years. Its cut extremity, however, soon increases in size, and on it a bulbous swelling forms, which, in a nerve the size of the median, is about half an inch in length by a third of an inch in diameter. This bulb is formed of fibrous tissue and young nerve-fibres. If the cut ends are in apposition, *union* occurs by a growth of nerve-tissue from the nuclei of the sheath; but the process is generally a slow one, and is usually preceded by degeneration of the lower end as already described. If good apposition is not maintained, union either does not occur at all or else it is very imperfect.

TREATMENT.—In the treatment of nerves accidentally divided all foreign bodies are, in the first place, to be carefully removed by means of a thoroughly aseptic sponge or forceps, with as little injury to the nerve-tissue as possible; and the wound, if practicable, is to be treated antiseptically. The limb should afterwards be fixed upon splints in a position which will bring the cut ends of the divided nerve most easily and closely into apposition. Chromicised catgut or kangaroo-tendon sutures should be passed completely through the nerve about a quarter of an inch from the cut surfaces, and tied tightly enough to obtain good apposition, but not so as to crush the nerve-fibres. Two sutures are usually sufficient. If possible, none of the nerve-fibres should be cut away, although a slight trimming off of jagged ends may be advisable. Passive motion of the paralysed muscles should be employed as soon as the wound is united; and afterwards weak galvanism should be applied to the limb, to promote nutrition and stimulate nerve-currents. It is also of much importance to keep the parts suffi-

ciently warm. In most of the cases so treated a good result is ultimately obtained, although it may be delayed as much as one or even two years. Sensation commonly returns before motor power, and voluntary power before electrical excitability.

In cases where a nerve has been for long divided and has not united on account of want of proper treatment by suture, the operation of *secondary suture* should be performed. The limb should be rendered bloodless by an Esmarch's bandage, and the separated ends exposed by a careful dissection. The greater part of the bulb should then be cut cleanly off, and the lower end should be refreshed. Sutures should then be passed as described above. When the ends are too far apart to be brought into apposition a portion of nerve taken from an animal or from a recently amputated limb may be used to connect the separated ends. These operations are sometimes tedious and difficult.

2. Nerves, Tumours of.—SYNON.: Neuro-mata.

The tumours which affect nerve-structure, although no doubt varying in essential character, as they do in other parts of the body, have usually been grouped indiscriminately under the head of Neuro-mata. They may be divided into two classes—(a) *true*; (b) *false*.

A *true* neuroma is one which is composed of nervous tissue, and may contain medullated or non-medullated fibres, and in addition ganglion cells; all such tumours are exceedingly rare.

A *false* neuroma is a tumour situated on a nerve and not itself containing any nerve-elements. Such growths are usually composed of fibrous tissue; but myxomatous, gliomatous, and sarcomatous tumours have also been described. All neuromata are most common on the nerve-trunks of the extremities, and are generally in great measure separable from the nerve-tubules among which they lie. This is especially the case with the fibrous growths. The so-called *traumatic neuromata* have just been described as 'bulbs' on the proximal end of injured nerves.

Numerous cases of multiple neuromata are on record. In most instances they are confined to one particular set of nerves and their branches. In one case they were confined to the posterior tibial and plantar nerves, and in another to the internal cutaneous and interosseous nerves of the arm. See TUMOURS.

SYMPTOMS AND DIAGNOSIS.—The chief symptom is pain, and this is felt not only at the seat of growth, but also in the parts to which the diseased nerve is distributed. The pain is often of an aching character, but is also at times shooting or neuralgic. Hyperæsthesia or anæsthesia of definite cutaneous areas are less common phenomena, and muscular weakness or paralysis is comparatively seldom complained of. On clinical examination a neuroma will be found as a smooth oval or rounded swelling, varying in size, and situated in the course of a nerve-trunk. The tumour is more movable in the transverse diameter of the limb than in its long axis. Handling and pressure cause pain both locally and in the course of the affected nerve.

TREATMENT.—Neuromata on the continuity of a nerve, if painful or situated so as to be easily accessible, and liable to injury, may be dissected out carefully. Sometimes it will be found that the tumour can be extirpated without taking away the entire thickness and destroying the continuity of the nerve, which, when a large one (as for instance

the great sciatic), it is important to preserve. In case this cannot be done, the whole section of the nerve-trunk may be taken away, and the smoothly cut ends brought together with fine chromicised catgut sutures, the limb being placed in a position to relax the nerve and lessen tension to the utmost. If approximation be impossible, union may be obtained by grafting a portion of nerve, taken from an animal or from a recently amputated limb, between the separated ends.

In cases of neuromata in stumps excision is sometimes available and effective. Opening the cicatrix and dissecting out the tumour or tumours may be all that is required, and this operation should in all cases be combined with thorough stretching of the affected trunks. But in other instances the pain and tenderness are so diffused, and the growths so numerous, that re-amputation a few inches higher up gives more complete and satisfactory results. Special care should be taken that the nerve-ends are cut short, so that they may not be included in or compressed by the scar.

3. Nerves, Surgical Division and Stretching of.

(a) *Nerve-section*.—SYNON. : Neurotomy.—Surgical division of nerves has been employed for the cure of painful affections, such as neuralgia, and for obstinate and sustained spasmodic movements. It has been usually performed subcutaneously, and most frequently in the case of the branches of the trifacial nerve, at their exit from the bony foramina, such as the supra-orbital, the infra-orbital, and the mental branches. Efforts have been directed to prevent the union of the cut nerve, by taking away a considerable portion, so as absolutely to prevent contact of the ends; and the operation then must necessarily lose its subcutaneous character. When the nerve spreads out to its distribution in all directions, it is difficult to secure this absolute removal, and a good deal of the adjacent soft parts must be excised to insure its being done thoroughly. The operation has to a great extent been given up since the introduction of nerve-stretching, and should never be performed until the latter method has been given a thorough trial.

(b) *Nerve-stretching*.—Nerve-stretching is effected by cutting down upon the nerve-trunk, detaching it from its connections for the space of a few inches, laying hold of it with the fingers, or passing an aneurysm-needle beneath it, forcibly stretching the whole nerve from its origin to such an extent as to affect powerfully its functions, and then closing up the wound. In some instances a certain amount of loss of sensation or muscular power in parts to which the nerve is distributed is the immediate result; this, however, passes away after a certain interval, and nerve-function becomes more or less completely restored.

APPLICATIONS.—The most useful application of nerve-stretching is its employment for the relief of neuralgia. In many cases of facial neuralgia temporary benefit at least is secured, while in some a permanent cure results. It is specially indicated in the epileptiform variety of facial neuralgia, and in intractable sciatica. The sciatic nerve can also be efficiently stretched without any incision, by what is known as the 'bloodless method.' For this purpose the patient is placed under an anæsthetic, and, the leg being maintained in a position of extension, the thigh is flexed upon the pelvis, and thus all the structures passing from the pelvis to the

posterior aspect of the thigh are put on the stretch. This condition of tension should be maintained for about ten minutes, and the limb should then be thoroughly massaged, the region of the sciatic nerve being especially kneaded and rubbed.

In cases of old nerve-injury or of implications of nerve-trunks in scar-tissue, the operation of nerve-stretching is most useful, but in all such cases the nerve should be exposed at the seat of injury or of thickening, and should be thoroughly freed with the knife before stretching is commenced.

In cases of motor spasm, nerve-stretching has also been employed, but with less success. Thus, it has been used for the treatment of spasmodic wry-neck, and facial tic or *tic convulsif*. In some cases also of tonic spasm and contracture the operation has appeared to be of benefit. For chronic neuritis, and especially for ascending neuritis, with thickening of the nerve-trunk, nerve-stretching is sometimes of much use, and should always be given a trial.

In tabes dorsalis, nerve-stretching was at one time much employed on the Continent, but has now fallen into disuse. It is, however, probable that some patients derived benefit from its application; and although it is not to be expected that the motor inco-ordination will be cured, yet in cases where the lightning-pains are specially severe the operation is possibly worthy of trial. Nerve-stretching has also been employed with benefit for anæsthetic leprosy, and for reflex epilepsy.

ANTHONY A. BOWLBY.

NERVI, in the Eastern Italian Riviera.—Warm, moist, winter climate. See CLIMATE, Treatment of Disease by.

NERVOUS.—A term used variously in reference to persons, to temperaments, or to morbid conditions. A person is said to be nervous, or of a nervous *temperament*, who seems to present a special susceptibility to pain, or who exhibits an undue mobility, as it is termed, of the nervous system—that is to say, when the person starts or shakes on the occasion of abrupt or intense sensorial impressions, or when he exhibits a proneness to convulsions, or manifests an exalted emotional susceptibility. An organisation of this kind characterises children rather than adults, and, among the latter, females more than males.

In reference to *disease*, the term 'nervous' implies 'relating to the nervous system' or 'affecting' it.

NERVOUS SYSTEM, Physical Examination of.—It is all-important that the physical examination should follow a definite routine, so arranged that the functions of every part of the nervous system may come under investigation. The signs of general diseases of the nervous system (Multiple Sclerosis, Tabes Dorsalis, &c.) may be at first almost entirely local, and some unobtrusive physical sign in a distant part of the nervous system may lead up to the recognition of the nature of the disease. If from an insufficient examination such signs be not discovered, an entirely erroneous diagnosis of local disease may be arrived at. Moreover, in no other system in the body may the presence of a single physical sign so entirely alter the conception of the nature of the disease present.

General Aspect of the Patient.—Careful attention must be paid to the general appearance of the patient at a distance of some yards, as in many

diseases the appearance is striking and may reveal important signs which are not so obvious on closer examination. The facial expression and the attitude of paralysis agitans, the facial expression of tetanus, bulbar paralysis and myasthenia gravis; the 'stiff back' of vertebral disease and the attitude of old-standing hemiplegia are among the very characteristic aspects of nervous disease. Among other signs, which are more obvious at a distance, may be especially mentioned partial bilateral ptosis, partial bilateral facial paralysis, and the position of the head in disease of one lateral lobe of the cerebellum (the head is inclined towards and the face is rotated away from the side of the lesion).

Psychic State.—It must be noticed in conversation whether the patient be a good witness or whether any peculiarity of mental state be present, however slight. Loss of reserve, facility, mental instability, exaltation and placidity, and the occurrence of delusions and hallucinations must be looked for, and any alterations of habit noticed.

Convulsions.—Where these occur it is of great importance that the patient be observed at the moment of commencement of the attack, when the facial aspect and colour should be particularly noticed (*see* EPILEPSY). The commencement of the convulsion should be noticed, whether it is local or general, unilateral or bilateral; its mode of spreading when local, and the extent of the body involved; the nature of the movements, whether tonic or clonic, irregular or purposive; the state of consciousness during and after the attack, whether paralysis succeeds the convulsions, whether there is biting of the tongue or incontinence of the sphincters during the attack. Enquiry should be made into the exact nature of any warning that precedes the attack. It should be remembered that just as recurring convulsions, commencing in one part of the body, may be a sign of a local lesion of the cerebrum (motor centres), so may recurring sudden sensations, both in connection with the special senses and general sensation, be signs of disease in the sensory region of the cerebral hemispheres. *See* CONVULSIONS IN CHILDREN.

Speech and Articulation.—Where disorders of articulation are met with, a careful analysis of the pronunciation of consonants will in slighter cases reveal which part of the articulatory mechanism—tongue, lips or palate—is at fault. The most useful test-sounds for palatal defects are final 'b' and 'g' which are pronounced 'm' and 'ng'; the words 'rub' and 'egg' becoming 'rum' and 'eng'; for lingual defects the explosives 'd' and 't,' which then lose their explosive character, and for labial defects initial 'wh' or 'w' as in 'which went.'

Examination of the more complicated kinds of articulatory defect is rendered easy if it be remembered that they consist always of one or more of the following primary faults: (1) slurring, (2) elision of syllables, (3) stumbling over syllables, (4) reduplication of syllables, (5) undue separation of syllables (scanning).

The repetition of such test phrases as 'Mutual Eligibility,' 'West Register Street,' and 'The Royal Irish Horse Artillery' will reveal the nature of the defect present in any given case.

For the investigation of aphasia and allied speech-defects, the following scheme of procedure is most useful. *See also* APHASIA.

General.—(1) Is the person right or left-handed, and, if the latter, does he write with the right

hand? (2) What is the state of education as regards reading and writing? (3) Does he understand the nature and uses of objects, and can he understand or express his wants by pantomime and gesture?

The activity of the auditory word-centre and the glosso-kinæsthetic centre and their connections.—(4) Is he deaf? If so, to what extent, and on one or both sides? (5) Can he recognise ordinary sounds and noises? (6) Can he comprehend speech? If so, does he at once attempt to answer a question? (7) Is spontaneous speech good? If not, to what extent is it impaired? Does he make use of recurring utterances, wrong words or gibberish? (8) Can he repeat words uttered before him?

The activity of the visual word-centre and the cheiro-kinæsthetic centre and their connections.—(9) Is the sight good or bad? Is there hemianopsia or optic neuritis? (10) Does he recognise printed or written words? If not, does he recognise single letters or numerals? (11) Can he write spontaneously? What mistakes occur in writing? Is there paraphasia? Can he read his own writing some time after it is written? (12) Can he copy written words, or from print into writing? Can he write numerals or perform simple mathematical calculations?

The associated activity of the speech-centres.—(13) Can he read aloud? (14) Can he name at sight words, letters, numerals, and common objects? (15) Can he write from dictation?

Special Senses.—1. *Smell.*—Only those substances which are readily oxidisable, such as the volatile oils, affect the terminations of the olfactory nerve. These are perceived as *odours* if they reach the terminations of the olfactory nerve through the anterior nares; as *flavours* if their path is by the posterior nares. Pungent substances, such as ammonia and acetic acid, stimulate the fibres of the fifth nerve, which supply the nasal chambers with common sensation, and such substances may be perceived when smell is entirely lost. For the testing of the sense of smell, such substances as peppermint, asafetida, clove, and camphor are most convenient. Each nostril must be tested separately while the other nostril is closed with the finger, as anosmia is frequently unilateral. *See* OLFACTORY SENSE; and NOSE, Diseases of.

2. *Vision.*—Each eye must be tested separately as regards perception of form, perception of colour, and visual field. The refracting media and fundus must be examined (*see* OPHTHALMOSCOPE; OPHTHALMOSCOPE IN MEDICINE; and EYE, Diseases of). Where vision is greatly reduced perception of light alone can be tested. For perception of form Snellen's test-types should be used, or where only a little vision remains it should be ascertained at what distance the patient can count fingers held up against a strong light. McHardy's self-registering perimeter is most convenient for determining the visual fields. It must be used accurately and a reading taken at every five degrees around the visual field. Reliable information, however, can be obtained without the use of instruments in the following manner. One eye of the patient is closed, either with his hand or with a bandage, and he is directed to look intently at the observer's nose from a distance of two feet. A piece of white paper, half an inch square, held upon a long penholder by being thrust into the slit of the nib, is brought into the patient's field of vision from the periphery in each part of the field. Coloured papers are then

used in place of the white paper, and the fields for the various colours determined.

Loss of perception of colour, hemianopia, quadrantic hemianopia, hemi-achromatopsia, general contraction of the visual fields, crossed amblyopia, helicoid (spiral) fields and central defects (*scotomata*) are to be looked for and may be readily demonstrated by this rough method. The presence of hemianopia may also be detected by means of the pressure-phosphene. If the point of a pencil be pressed firmly against the eyeball, close to the orbital margin, a ring of light is perceived in the opposite part of the visual field; consequently when part of the visual field is lost the phenomenon is absent from the corresponding part of the eyeball. Where detailed examination is impracticable, hemianopia may be detected by standing in front of the patient, who is directed to look straight forwards. The observer then holds up both hands, one on either side and slightly in front of the patient's head, and directs him to touch his hand. The hemianopic patient at once touches the hand he sees. If there be no hemianopia the patient

bringing a watch from a distance towards the ear, the other ear being closed with the finger. Perosseal conduction is investigated by applying a tuning-fork with a disc-shaped foot to the zygoma or mastoid process, the external auditory meatus of the same side being closed with the finger.

Where aerial conduction is much impaired or lost while perosseal conduction remains good, the fault lies in the external auditory meatus (cerumen) or the tympanic ossicles and membranes, and not with the nervous apparatus. Where both aerial and perosseal conductions are impaired or lost (nerve-deafness) there may be primary disease of the nerve, or it may be damaged from disease in its immediate neighbourhood (tumour, meningitis, &c.), in which case the contiguous facial and fifth nerves may be affected. Relative deficiency in the appreciation of tone must be tested with Galton's graduated whistle.

4. *Taste*.—This sense should be tested by means of sugar, common salt, tartaric acid, and quinine applied in the form of powder by means of a moistened camel's-hair brush to the back and front

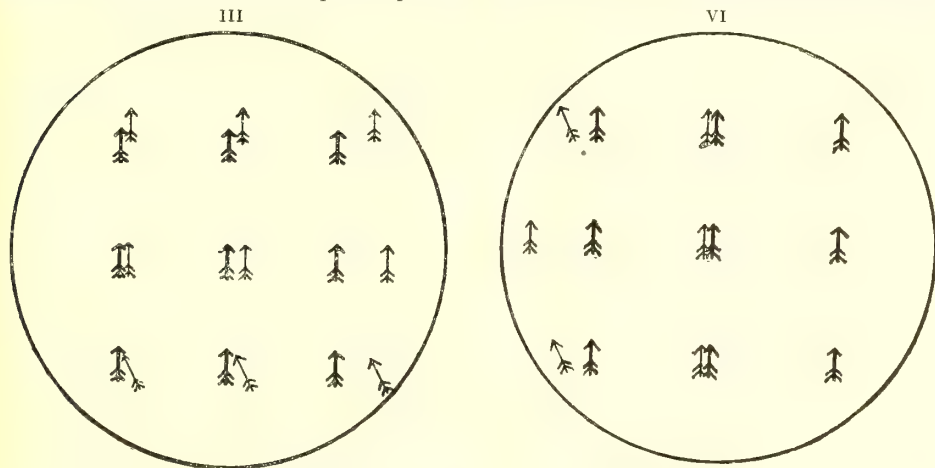


FIG. 1.—Position of images in the binocular field in cases of paralysis of the left third nerve (III), and left sixth nerve (VI) respectively. The thin arrow denotes the false image.

inquires which hand he is required to touch. In a semi-conscious patient the presence of hemianopia may be considered probable if, when a feint is made from the side at either eye (the patient's eyes being open), he constantly winces when the attempt is made from the right and never when it is made from the left, or conversely. Care must be taken, however, in this procedure that the movement of the hand in making the feint causes no draught of air to strike the conjunctiva. When hemianopia is present it must be determined whether this is due to disease of the optic tract or of the occipital lobe by means of Wernicke's reaction. A strong ray of light is thrown on to the blind side of the retina; if the pupil reacts, the reflex fibres of the pupil which separate from the optic fibres at the posterior end of the tract are uninjured, and the disease is presumably not situated in the optic tract but in the occipital lobe. On the other hand, if the pupil does not react it is probable that the situation of the lesion is in the region of the optic tract.

3. *Hearing*.—Perception of sound through the air (aerial conduction) should be tested by gradually

of each half of the tongue, while that organ is at rest in the mouth. Some normal people are entirely unable to taste on the protruded tongue. The sentient areas on the anterior part of the tongue are a small patch on the lateral margin of the tongue on either side near the tip, while the posterior sentient area is far back in the region of the circumvallate papillae, the rest of the tongue being more or less insentient for taste. The velum palati and the glosso-epiglottic folds also contain taste-organs. The nerves for taste leave the brain in the fifth nerve, those for the anterior two-thirds of the tongue, leaving the pons by the second division of the fifth nerve, pass in order by the vidian, great superficial petrosal, facial, chorda tympani and lingual nerves to their distribution, while those for the posterior two-thirds of the tongue and for the palate pass from the fifth into the glosso-pharyngeal trunk; their course has not been demonstrated with certainty.

Other Cranial Nerves.—*Oculo-motor nerves*. The condition of the pupils should be examined as to size and shape, and as to the presence of signs of iritic adhesions which may interfere with the

mobility of the pupil. Both the direct and the consensual light-reflex (the contraction of the pupil of the opposite eye when one eye alone is exposed to light) should be tested, and the light employed should be bright and at some distance from the patient in order to avoid the contraction of the pupil associated with convergence. It should also be noticed whether the pupils dilate when shaded, to an equal extent in both eyes. The contraction of the pupil which occurs when a near object is fixed is associated with the act of convergence and not with the act of accommodation, for when the act of accommodation occurs without convergence there is no associated contraction of the pupil. The condition of the ocular movements should be tested in the following way: the patient is directed to look at an object on a level with his eyes and about a yard in front of his face, and it is noticed whether there is a strabismus present. If so, first one eye alone, and then the other alone, is covered, yet so that the eye can be observed, and it is found that when the normal eye is covered, the erring eye moves in the direction of the weak ocular movement to fix the object. The object is then moved upwards, downwards, and in either lateral direction, until the extremes of possible deviation of the eyes are produced, when weakness of any particular movement will become apparent. Any defective movement involving the corresponding movements of both eyes (as for instance lateral deviation to one side) must be specially noticed; the cause of such defect is always nuclear in situation. Where diplopia is present and the strabismus not very obvious, it is useful to take a chart of the position of the true and false images of an object, such as an arrow drawn on white paper in the binocular field (see fig. 1). By this means the nature of a diplopia becomes at once clear. It is convenient to remember that when from a strabismus the prolongations forward of the optic axes cross (convergent strabismus), the resulting diplopia is homonymous (false image on the same side as erring eye); when the axes diverge, the resulting diplopia is crossed (false image on the same side as sound eye).

Rotatory movements of the eyeball, occurring with certain movements of the eyes, must be looked for where paralysis of either the superior or inferior oblique muscles is suspected. In some ocular palsies characteristic attitudes of the head are assumed in order to avoid diplopia; for instance, in palsy of the inferior oblique the head is often thrown back. The presence of ptosis or of von Graefe's sign must be looked for, and the width of the palpebral aperture examined. The effect of local instillation of cocaine upon the pupil, and width of the palpebral aperture, are important in investigating paralysis of the cervical sympathetic trunk (*vide infra*).

Nystagmus.—It should be noticed whether this sign is present when the eyes are at rest (spontaneous nystagmus) or only on movement of the eyes. The range and nature of the nystagmus should be examined. See NYSTAGMUS.

The Fifth Nerves are to be examined by testing the condition of sensibility of the face and mucous membranes of the mouth and nose (*vide infra*) and by testing the power of the masticatory muscles supplied by that nerve. The observer's fingers are pressed firmly against the temporal muscles or the masseters on both sides, and the patient is directed

to bite 'as if he were cracking a nut.' From the amount of bulging of these muscles during the contraction, deficiency on one or both sides may be proved. Weakness of the mylo-hyoid and anterior belly of the digastric is detected by pressing upwards on to the floor of the mouth with both thumbs, while the patient opens his mouth forcibly; the floor of the mouth hardens only on one side in unilateral paralysis of the fifth nerve. From the unopposed action of the external pterygoid muscle of the sound side, the jaw, when the mouth is open, deviates strongly to the paralysed side when there is unilateral paralysis of the fifth nerve. Hollowing of the temple and flattening of the cheek, from wasting of the muscles of mastication, are readily detected. The affection of taste has been mentioned above. Smell may be ultimately lost on the side of the injured nerve from slow trophic changes occurring in the olfactory mucous membrane. Facial anaesthesia should be looked for where there is corneal ulceration.

Seventh Nerve.—The various regions of the face must be investigated as regards (1) volitional movements, such as closing the eyes against the resistance of a finger placed upon the lid, or showing the teeth; (2) emotional movements, such as those produced by the request for 'a pretty smile'; and (3) associated movements, such as are observed in the lower face when the eyes are forcibly closed. See FACIAL PARALYSIS.

Ninth, tenth and eleventh Nerves.—These may be considered together, as they constitute a physiological group, and are frequently involved together by a lesion situated at the side of the medulla. They are the sensory and motor nerves of the palate, pharynx, oesophagus and larynx. The eleventh nerve also supplies the sterno-mastoid and trapezius in part. The palatal and pharyngeal reflexes depend upon the integrity of these nerves. Sensibility and motion are to be examined in the usual way. See LARYNX, Examination of.

Twelfth Nerve.—The power of the tongue is best tested by the ability to protrude it and to put it into either cheek. Fibrillation and wasting are to be looked for, and when the latter is present the muscles should be examined with electricity, very weak currents being used. In unilateral palsy of the tongue the tip always deviates towards the paralysed side.

Cervical Sympathetic Nerve.—Pathological conditions of this nerve are frequently overlooked, and signs of these should always be sought for (1) in cases of ocular paralysis, (2) where disease of the cervical cord is suspected, and (3) in syringomyelia. A small pupil which does not dilate to cocaine, narrowing of the palpebral aperture, and unilateral flushing and sweating of the face are signs of paralysis of the sympathetic.

Von Graefe's symptom, retraction of the upper eyelid, a large pupil and tachycardia indicate an irritative lesion. Not uncommonly symptoms of paralysis and irritation are alternately present.

Sensation.—The occurrence, nature and exact distribution of subjective sensations, pain, numbness, tingling, &c., and any circumstances causing their exacerbation, must be considered. Such sensations occurring in areas corresponding with the distribution of special nerve-roots or nerve-trunks are of great value as spinal localising symptoms. The girdle-sensation of common occurrence in local disease of the spinal cord, the sense of weight in

the perinæum in tabes, and the 'hide-bound' feeling in the limbs, are examples of subjective sensations of segmental distribution; while the distribution of the pain in trigeminal neuralgia and in sciatica are examples of nerve-trunk sensations.

The accurate objective investigation of sensory disturbances is by far the most laborious and difficult part of the physical examination of the nervous system, for the observer is entirely dependent for his information upon the statements of the patient, and for this reason dulness of attention and the fatigue of a long examination are apt to introduce fallacies. The patient's eyes should be screened from the action of the investigator.

Sensibility to Touch is tested by degrees of pressure with the finger. A more delicate test for examining the slightest changes is to brush the skin gently with a small skein of silk, wool and linen successively. A normal person perceives the difference in the contact of these materials at once. *Sensibility to Pain* is tested with a pin, which should be presented to the skin together with a comparatively large touching surface—for instance, the top of the investigator's finger—lest from the small area of contact the pin is recognised as such where sensation to pain is absent. *Sensibility to Temperature* may be tested by the application of test-tubes containing hot water and iced water respectively. It must be borne in mind that an anæsthetic skin will blister readily on the application of heat. *Sensibility to the Faradic current* is best examined by applying to the skin a pair of wire electrodes such as are in common use in physiological laboratories.

The following course of procedure is absolutely essential if reliable information is to be obtained. The investigation is commenced upon an area where sensibility is normal, and is carried out first in straight lines running in the direction of the length of the body, and secondly, in lines at right angles to these until the whole surface of the body has been tested. Working thus from æsthesia to anæsthesia the points at which the least change is noticed are to be taken as the limit of sensory impairment; and should be marked upon the body with a dermatographic pencil and subsequently filled in upon a chart. The results obtained should be carefully compared with the diagram of segmental areas (see PAIN IN VISCERAL DISEASE). When anæsthesia is partial the power of localising sensory stimuli must be tested. Hyperæsthesia, delayed sensation and allocheiria (p. 48) must be looked for.

Muscular Sense.—Abnormal conditions of this sense may be detected by the examination of (1) the sense of passive position, (2) the sense of active muscular contraction, and (3) the power of maintenance of position.

To test the sense of *passive position*, the patient is blindfolded and the limb is placed by the observer in various positions, which are then indicated by the patient, and in order to avoid inferences that the subject may gain from cutaneous sensation during this process, the limb (a finger, for instance) should be held laterally between the thumb and index-finger of the observer. The sense of active *muscular contraction* is best measured by appreciation of weight. A series of leather-covered balls of equal size and different weights are made for the purpose. Where the muscular sense is impaired

appreciation of differences in weight becomes extremely defective (for other tests see ATAXY, p. 1068). The power of *maintenance of position* is tested in the following manner. The patient is directed to hold his arms straight in front of him, with the fingers spread out. He is then directed to close his eyes, when, if the muscular sense be much impaired, wandering movements of the limbs soon commence, of which the patient is entirely unaware.

The Motor System.—The performance of the complicated voluntary movements to which the various parts of the body are accustomed are first to be observed, for while there may be no discernible paralysis of any muscle in detail, there may be striking paralysis in effect when an attempt is made to perform any complicated act. A patient who, when lying in bed, can make all the movements of the lower extremity powerfully, may show great motor defect in that same limb when attempting to walk, and similarly in writer's cramp the hand may be almost completely paralysed for one kind of action only. In investigating such actions, as, for instance, the patient's gait, scrutiny should be used lest a defect appeal to the observer as a generic idea. As an instance, a patient may be said to stagger when walking; such a description conveys little information, since it may be caused by general asthenia, cerebellar disease, labyrinthine disease, spasticity of muscles or loss of muscular sense; careful analysis, however, will reveal the nature of the defect. The power of each group of muscles should be tested in terms of the performance of some movement against resistance or gravity—a certain movement is possible or not against a certain resistance. The dynamometer should be used, where possible, for by its means the progress or decrease of muscular weakness can be accurately gauged. Localised and general wasting or hypertrophy of muscles should be noticed. Measurements are most correct if the maximum and minimum circumference of a limb be recorded.

The presence of *deformities* due to muscular spasm, paralysis or contracture, the occurrence of *tremor*, *fibrillary twitching*, *rigidities*, *hypotonus*, *muscular tenderness*, and the *excitability* of the muscles to mechanical stimuli (i.e. direct percussion) must be investigated.

Hypotonus.—The signs of this important condition, to which great attention has of late been drawn, are worthy of special notice here. The condition is the opposite of rigidity, it is more than mere flaccidity. The muscles hang loosely from the bones, the joints rattle when shaken, and can be moved 'like loosely sewn-on buttons.' Attitudes like those of an acrobat, which are impossible in normal persons, can be assumed without discomfort: for instance, the thigh can be flexed until the tibia touches the clavicle; the thighs can be spread until they form a straight line with one another at right angles to the trunk; the patient sitting upon the floor can bend forward and touch the ground in front of him with his head when this condition is present in severe degree.

Hypotonus is dependent upon a condition of relaxation of both muscles and ligaments. Its presence implies neither wasting nor paralysis, and sometimes even there may be increase of the deep reflexes. It is always present in some degree in cases of tabes dorsalis. The presence of this sign usually signifies disease of the posterior columns of the cord.

Electrical Reactions.—It is, as a rule, only necessary to examine the electrical excitability of the muscles where wasting or muscular tenderness is present. The all-important point is whether faradic excitability is present, reduced or absent. This is best tested by means of single induction-shocks. Two small electrodes should be applied, without regard to motor points, directly to the surface of the muscle. Single induction-shocks are infinitely less painful than is the interrupted current. For the examination of electrical reactions in children an anæsthetic should always be employed. *See* ELECTRICITY IN MEDICINE.

Individual Muscles.—Space does not permit of the enumeration of the tests of the power of individual muscles, but on account of the importance of weakness of the trunk-muscles, as localising signs of spinal-cord lesions, the methods of their examination are here mentioned.

Erector Spinae.—The patient is seated upon a stool and the head and trunk being flexed to the full, he is directed to raise his head and trunk to the vertical position against the resistance applied with the hand to the back of his head. During this action the observer feels with his hands the hardening and bellying of the erector spinae in its various parts. If the examination is to be carried out upon a patient who must remain in bed, he is placed in the prone position, the pillows being removed, and he is directed to raise his head backwards off the bed without using the upper extremities.

Respiratory Muscles.—Paralysis of the intercostal muscles is recognised by the loss of the expansive movement of the upper eight ribs during inspiration. The movement of elevation is carried out chiefly by the scalene muscles, and in intercostal palsy there is frequently visible action in the auxiliary muscles elevating the chest (sterno-mastoids, infrahyoid muscles, &c.). The eversion of the lower four ribs during inspiration is carried out by the diaphragm, and is frequently present where the intercostal muscles are completely paralysed, and must not be mistaken for intercostal action. Paralysis of the diaphragm is shown by the loss of the inspiratory expansive movement of the abdomen and by the eversion of the lower ribs. The abdomen is sucked in during inspiration, and the normally synchronous expansive movements of the chest and abdomen then alternate.

Abdominal Muscles.—The patient is directed to raise himself in bed from the supine position without using his hands, and the surface of the abdomen is noticed. This act is impossible when any severe degree of weakness of the abdominal muscles is present. When a local lesion of the spinal cord in the region of the supply of the abdominal muscles is present, the appearance of the abdomen during the attempt is characteristic. If there be weakness in the lower parts of the abdominal wall the umbilicus shifts upwards; and if weakness be confined to the upper abdominal wall the umbilicus shifts downwards, and the exact limit of the paralysed muscle may often be determined by the formation of marked retraction-line. (The abdominal musculature is supplied by the lower six dorsal and first lumbar nerve-roots. The umbilical region is supplied by the ninth dorsal root.) Local paralysis of the rectus abdominis and of the oblique muscles is evidenced by vertical and lateral bulging of the abdominal wall respectively during the attempt to sit up.

Ataxy.—Tests further than those given under the heading 'Muscular Sense' are for the upper extremity: (1) the handwriting; (2) picking up a small object, such as a pin, from a flat surface; (3) touching the nose with the index finger of either hand, with the eyes first open and then shut. For the lower extremities, the following tests are convenient: (1) touching the observer's finger, held within easy reach, with the great toe, the eyes being open; (2) placing the heel of one foot upon the knee of the opposite side, the eyes being closed. (These tests, 1 and 2, are of service when the patient is unable to stand.) (3) Standing upright with the feet together, with the eyes open; and (4) with the eyes closed (Romberg's sign); (5) standing upon one foot; (6) walking along a straight line; (7) and, as the most delicate test of all, 'toeing and heeling' a line.

Trophic Disturbances.—Attention must be paid to the occurrence of brittle and 'reeded' nails, thin and glossy skin, induration and desquamation of the skin, localised or general atrophy of the skin, leucoderma, bedsores, perforating ulcer, herpes and local cutaneous hæmorrhages, thickening of bones, arthritic changes (adhesions, grating, Charcot's joints, &c.).

Vaso-motor Disturbances.—The following points should be noticed: the occurrence of local flushing, erythromelalgia (p. 491) or cyanosis, local elevation or depression of surface-temperature, clubbing of the fingers and toes and localised sweating.

Pelvic Reflexes.—Trouble in the performance of the acts of micturition and defæcation should be most carefully inquired into. Slight troubles of this nature are often concealed by patients, especially by women. The exact nature of such defect, whether hesitation, precipitancy, retention, or reflex or paralytic incontinence must be ascertained. The bladder should be examined by palpation and percussion after a natural evacuation in every case where there is dysuria, to ascertain whether there be residual urine—an important predisposing cause for cystitis. The use of the catheter for this purpose, even though the greatest care be used, is apt to set up cystitis.

Where incontinence is complete, the sphincter ani should be examined digitally, to ascertain if there be reflex contraction or not. Reflex contraction of the bulbo-cavernosus muscle may be tested by pinching the skin of the perineum while the finger rests upon the bulb of the urethra.

Defects of sexual power are important: they are said to be constantly associated with defective sensibility of the glans penis.

Reflex Action.—The following superficial reflexes should be examined. The *corneal reflex* is best tested by suddenly blowing upon the patient's face. To obtain the *pharyngeal reflex*, the tongue is depressed and the posterior wall of the pharynx and lower edge of the soft palate are stroked with a pen-holder, and the contraction of the palate and pharynx observed. The *abdominal* and the *cremasteric* and *dartos reflexes* are obtained by stroking the skin lightly over the lower ribs and groin respectively. The *plantar reflex* should be investigated, if possible, when the foot is neither damp nor cold, the knee should be flexed and rolled out, and a light scratching stimulus should be applied from heel to toes, across the sole, and the movement of the toes, whether flexor (normal) or extensor (abnormal, and indicating a lesion of the pyramidal

tract) should be observed, and also whether the tensor fasciæ femoris is the first muscle to contract in the reflex (normal). See BABINSKI'S SIGN.

Tendon-Phenomena.—These phenomena are obtainable in those muscles of the body which are suitably placed for examination. The following should be examined in every case. A heavy wooden stethoscope, with a soft indiarubber-ring fitted round the ear-piece, is the most convenient instrument to use.

1. The *jaw-jerk* is best obtained by allowing the jaw to hang loosely. Percussion is then made downwards, either upon a rigid article, such as a paper-knife, placed upon the lower teeth or upon the observer's finger, which rests upon the chin. Jaw-clonus is produced in the same way, and is evidence of increase of the myotatic irritability of the muscles of mastication.

2. The *forearm-jerk* is produced when the radial border of the forearm is struck with a hammer, the elbow being flexed at a right angle, and the hand supported by the observer so as to ensure complete muscular relaxation.

3. The *triceps-jerk*.—The arm is abducted at the shoulder to the horizontal, and held by the observer immediately above the elbow, the forearm is allowed to hang vertically downwards, the tendon of the triceps is then struck immediately above the olecranon. Care must here be taken lest the muscle of the triceps be struck, and direct muscular contraction (which is an entirely distinct phenomenon) be mistaken for the tendon-jerk.

4. The *adductor-jerk* is obtained when the internal condyle of the femur is struck heavily in an outward direction, the hip and knee being slightly flexed and rolled out. Sometimes a response in the adductor muscles of the opposite thigh is obtained (crossed adductor-jerk).

5. The *knee-jerk*.—The knee should be bared and resting, without muscular exertion, in a position of flexion, the tendon should be struck exactly in the interval between the lower border of the patella and the tuberosity of the tibia. In rare cases of isolated atrophic palsy of the quadriceps, the knee-jerk being absent, percussion of the patellar tendon causes contraction in the ham-strings (flexor knee-jerk).

6. The *depressed patella-jerk* is obtained with the leg extended and the quadriceps flaccid, the upper border of the patella is pressed down forcibly with the index-finger hooked round it, and the finger is then struck in a downward direction with a hammer.

7. The *Achilles-jerk*.—The observer seizes the fore part of the foot with the left hand and raises the whole limb, pressing the foot back so as to make the tendo Achillis taut; the tendon is then smartly and lightly struck with the hammer.

Methods of Reinforcement.—In order to ensure muscular relaxation, to remove the element of expectant attention, and actually to increase the activity of the tendon-phenomena, certain proceedings should always be adopted: (1) In general reinforcement the patient is directed to look away from the observer; he is engaged in conversation; he is directed to squeeze his hands together with all the effort he can muster, or to perform some other act requiring effort, with any one of the limbs that is not being investigated, while the tendon-phenomena are being tested. Where the tendon-phenomena are much diminished, sensory stimulation, such as friction to the skin, the application of faradism and especially a cold shower-bath, tends to

increase the tendon-phenomena, and the effect is most marked about five minutes after such stimulation. (2) Local reinforcement is used for the investigation of the knee-jerk only. The foot is passively pressed back, and the patient is directed to press gently against the resistance with his toes. Severe muscular rigidity and arthritic changes may make the tendon-phenomena impossible to obtain.

Clonus.—Where the tendon-phenomena are exaggerated clonus may be obtained in almost any muscle by suddenly stretching it. Foot-clonus, rectus-clonus, and hand-clonus are most commonly investigated. The least movement of a limb may set up a general clonus (spontaneous clonus, epileptoid spasm).

Spurious Clonus.—Foot-clonus not infrequently occurs in cases of functional disease. Attempts have been made to separate this form of foot-clonus from that occurring in organic diseases under the above term. While this is certainly impossible, yet in some functional patients a form of foot-clonus *sui generis* is met with, and has the following distinctive features: (1) It is often irregular in rhythm; (2) the position of the foot during the occurrence is one of moderate plantar extension (in organic clonus the position is one of dorsiflexion); (3) the clonus is started by a voluntary movement of plantar extension on the part of the patient, and not by sudden passive stretching of the muscle. This useful sign was described by Beevor. In many normal persons clonus of this nature may occur when sitting in certain positions.

The examination of the skull, vertebral column, heart, pulse, temperature, respiration and other parts and phenomena is of great importance in cases of nervous disease. See PHYSICAL EXAMINATION. JAMES COLLIER.

NERVOUS TEMPERAMENT.—See NERVOUS; and TEMPERAMENT.

NETTLE-RASH.—A popular synonym for urticaria. See URTICARIA.

NEUENAUHR, in Germany.—Thermal alkaline waters. See MINERAL WATERS.

NEURALGIA (νεῦρον, nerve; and ἀλγία, I suffer pain).—**SYNON.**: Fr. *Néuralgie*; Ger. *Neuralgie*.—This is a term applied to a disease of the nervous sensory apparatus, marked by paroxysmal pain, which is for the most part unilateral, and in the course of a nerve or its branches. In many cases no evidence of change in the periphery of the nerve is discoverable, and to these the term *neuralgie* is perhaps most properly applied; in others, however, there is reason to think that inflammation of the nerve is at least the starting point of the disorder. The diagnostic points are as yet not sufficiently certain for these cases of neuritis to be absolutely separated from those of simple neuralgia, and they may so far be considered together. Relative constancy in the pain, with paresis and atrophy of muscles supplied by the affected nerve, cutaneous anesthesia, and swelling of the nerve-trunk, point to neuritis. See NEURITIS.

ÆTIOLOGY.—Neuralgia is prone to occur in families marked by neurotic tendencies, not necessarily of neuralgic character, but which display themselves in various phases of psychical disturbance, as insanity, hysteria, hypochondriasis, or in the shape of epilepsy, neurasthenia, and chorea.

Rare in childhood, puberty has a strong predisposing influence. In the middle period of life, though first attacks are not very common, revivals of neuralgia are apt to occur, as a result apparently of the depression occasioned by the wear and tear of life. Premature senility (marked by atheromatous changes in the vessels, arcus senilis, permanent greyness of hair, bagging of the cheeks, pulmonary emphysema) conduces to severe and intractable neuralgias. Malaria is a potent cause. The presence of sewer-gas in a house may occasion persistent neuralgia. Anæmia and malnutrition generally, however brought about, play an important part. So also do sexual excesses, and perhaps likewise a state of celibacy. Pregnancy, over-lactation, and menorrhagia are each predisposing causes. The most frequent exciting causes are cold, especially damp cold; injury to the nerve by violence, or by the encroachment of morbid growths; syphilis; gout; and the presence of lead or mercury in the system. Over-strain of mind or body conduces to attacks. Irritation of peripheric organs may excite neuralgia in nerves nearly or remotely associated. So dental caries may induce supra-orbital neuralgia; indigestion may excite anginal symptoms; uterine disease may excite neuralgia of distant nerve-trunks—as, for example, the occipital; and the presence of intestinal worms may explain the occurrence of neuralgia in parts quite unconnected with the bowels. Neuralgia is a common sequel of enteric fever, relapsing fever, and influenza. Neuralgic pains are prone to occur in the course of diabetes mellitus. They are symmetrical, and are very likely due to multiple neuritis.

ANATOMICAL CHARACTERS.—In simple neuralgia no definite lesions are discoverable—at least, none that are constant enough to deserve the place of necessary accompaniments or factors of the disease. As a result of neuritis or perineuritis the nerve-trunk is sometimes found swollen and hyperæmic; or, in a later stage, it may be atrophied and its fibres degenerated.

SYMPTOMS.—After some little preceding numbness, cutaneous anæsthesia, or other abnormality of sensation, the import of which gets to be well understood by persons liable to neuralgia, the patient is seized with pain, which at first is not severe, and ceases quickly, but returns in a few seconds or minutes, lasting for a short time and then remitting. These darts revive with shorter and shorter intervals, so that in a little time the pain appears to be almost continuous, or interrupted only by waves of increased intensity, and it will last for some seconds or more than a minute together. Then comes a respite, to be followed by recurrence, and these alternations may continue for a few minutes or as many hours. In attacks of long duration, where no treatment is applied, the pain gradually gets less acute, the intermissions longer, and the outbreak passes into a confused feeling of discomfort and bruising about the seat of pain, coupled with a sense of exhaustion and desire for sleep. The character of the pain varies; it is described as darting like a knife or like lightning, crushing, hammering, boring, and sometimes burning. In neuralgia about the head the patient will often be seen to cringe and recede before the plunges of pain, as though he were receiving blows. When the pain is at its worst there is often a radiation of it to other nerves, and especially to those placed symmetrically with the one affected;

but this secondary pain never attains anything like the severity of the original. Not always, but perhaps commonly, certain definite points where pressure is exceedingly painful may be found by palpation. These, the *points douloureux* of Valleix, have a certain amount of diagnostic importance. Rare in first attacks, they are more common in patients who have been subject to recurrences during many years. There is always a nerve-branch under the skin at these points; and more often than not they correspond with the point of emergence of a nerve from a bony groove or opening, or its passage through a muscular aponeurosis. Pallor of the skin followed by intense redness, horripilation, and other evidences of vaso-motor disturbance, are common. In the case of nerves being attacked which preside over glands there is often increased secretion. The tactile sensibility of the skin is almost always diminished after a time in the neighbourhood of the affected nerve, though at first there is some hyperæsthesia. In neuralgia of the fifth nerve spasmodic contraction of facial muscles, of reflex origin, is apt to occur.

LOCAL VARIETIES.—The varieties of neuralgia are divided into two primary groups, namely: I. Superficial; and II. Visceral.

I. Superficial.—These include the following:

(a) *Trigeminal neuralgia.*—See TIC DOULOUREUX.

(b) *Cervico-occipital neuralgia.*—The posterior branches of the first four pairs of spinal nerves may be affected, but it is that of the second, the great occipital, which is most important, from its size, and the frequency with which it is attacked. Shooting pains start from just below the occiput, and run over the back and top of the head, sometimes into the external auditory meatus, and often to the front of the head and face. A point of great tenderness is to be found over the exit of the great occipital nerve. Giddiness, noise in the ears, and some confusion of ideas are often associated, and frequently cause cervico-occipital neuralgia to be mistaken for commencing organic disease of the brain. It may begin by such acute tenderness of the scalp as makes it an agony to brush the hair.

(c) *Cervico-brachial neuralgia.*—The nerves of the brachial plexus and the posterior branches of the four lower cervical nerves are here concerned. The pains affect the neck and shoulders, or shoot down the arm to the hand, in the course of one or more of the nerve-trunks. Painful points may be found in the axilla, over the upper part of the deltoid, at the bend of the elbow, three inches above it externally, in the groove between the inner condyle of the humerus and the olecranon, at the ulnar side of the annular ligament, and where the radial nerve becomes superficial. The ulnar nerve is that most often affected, but the neuralgia usually spreads to other trunks. See p. 170.

(d) *Dorso-intercostal neuralgia.*—See INTERCOSTAL NEURALGIA.

(e) *Lumbo-abdominal neuralgia.*—Here the superficial branches of the lumbar plexus distributed to the abdominal walls are affected. It is less common than intercostal neuralgia, but resembles it generally. The female sex is apt to be most affected. Tender points may be found close to the spine, at the middle of the crest of the ilium, in the hypogastric region, in the groin, and on the scrotum.

(f) *Crural neuralgia.*—This variety is almost always met with as a complication of sciatica, being

rare by itself. Pain occurs in the front of the thigh and knee, and inner surface of the leg and foot. The long saphenous branch of the anterior crural nerve is most commonly affected. This form of neuralgia is not infrequent in hip-joint disease, where it is secondary to irritation of the branches of the obturator nerve supplying the joint.

(g) *Obturator neuralgia* affects the inner side of the thigh.

(h) *Femoro-popliteal neuralgia*.—See SCIATICA.

(i) *Coccygodynia*.—Pain in the neighbourhood of the coccyx, especially apt. to occur in women, is sometimes, but by no means always, due to neuralgia of the coccygeal plexus. See COCCYGODYNIA.

II. **Visceral**.—(a) *Cardiac*.—A certain proportion of the class of cases called angina pectoris depends upon cardiac neuralgia (see ANGINA PECTORIS). There is sudden severe pain at the lower end of the sternum, darting to the back and down the left arm, or it may be diffused over the chest and affect both arms. The heart feels as though it were grasped, the face loses colour, the pulse becomes altered in character, there is cold sweating, and generally the aspect and feeling of approaching death. Such attacks may be confined to two or three repetitions, or there may be a constant tendency to their recurrence under circumstances of fatigue or strong emotion. See PAIN IN VISCERAL DISEASE.

(b) *Uterine and ovarian neuralgia*.—Pain attendant upon menstruation, independent of any mechanical difficulty, is thus named. It may be excited by such sources of peripheral irritation as ascariides, leucorrhœa, renal calculus, prolapsus uteri, tumours, ulceration of the cervix, or impaction of fæces; or the sources may be in some distant part of the body. Ovarian neuralgia may be accompanied by congestion of the ovary.

(c) *The urethra, bladder, rectum, kidney*, and *testis* may each be affected by neuralgia. The last-named may result from self-abuse, or be consequent upon renal concretion. See NEPHRALGIA.

(d) *Gastralgia*.—Abdominal neuralgia is characterised by intensity of colicky pain, occurring in paroxysms, in circumstances differing from those which induce ordinary dyspepsia. There is nearly always a history of neuralgia in some other part of the body. Vomiting sometimes, and constipation invariably, accompanies the attacks. See STOMACH, Neuroses of.

COMPLICATIONS AND SEQUELÆ.—Neuralgia when it attacks mixed nerves may produce muscular powerlessness, which is not merely due to a shrinking from making muscular effort because of the pain attending it, but a temporary paralysis consequent on suspension of motor impulses. Or there may be spasm of muscles. Long-continued neuralgia is attended by more or less atrophy of the muscles supplied by the affected nerves, which may be temporary, or, in cases where frequent recurrences of the attack take place, may be permanent. Certain forms of neuralgia, especially that of the first division of the fifth, intercostal, and sciatic nerves, are liable to be accompanied by a herpetic eruption (see p. 685). Anæsthesia of a portion of the skin will often persist, though the pain itself may be absent. Neuralgia tends to become a chronic disorder, with frequent intervals of more or less long-lasting exemption. It does not often appear as an acute attack of suffering, lasting for weeks or months and never returning. Such a history points

rather to neuritis. Long continuance of severe neuralgia may lead to melancholia, which is sometimes of suicidal character.

DIAGNOSIS.—It is of extreme importance that pain should not be attributed to neuralgia until the existence of an organic lesion capable of causing the sensory disturbance has been excluded by the most careful examination. It may be said perhaps that for pain to be strictly accounted *neuralgic* there should be no obvious cause for it, such as fever, local inflammation, tumour, or injury; it should be intermittent, or at least liable to great exacerbations, and independent of movement or any external agency; it should take the course of one or more nerves; and there should be spots painful to pressure in some of the localities already indicated. Neuralgia is distinguished from myalgia by the latter involving the attachments of a muscle, not occurring in paroxysms, but dependent upon movement; from aneurysm by careful physical examination, which is especially necessary when the pain is about the chest and loins. In chronic rheumatism the pain is diffused, and influenced by movement; and it does not affect the district of a particular nerve. Acute rheumatism is accompanied by elevation of temperature, sweating, and swelling of joints. The thermometer, and the known symptoms and signs of the several diseases, will usually at once exclude pleurisy, pneumonia, and peritonitis. Attacks of neuralgia, especially of the supra-orbital nerve, may be characterised by a remarkable periodicity, recurring daily at the same hour, and lasting about the same time, quite independent of any malarial basis. Syphilitic periostitis is evidenced by the sight and touch, as well as (if it occur early in the disease) by the presence of febrile symptoms. Where pain in the back is supposed to be of neuralgic origin it is important to exclude the presence of hernia. Examination should be made *per vaginam* to exclude flexions or tumours of the uterus, and *per anum* for the presence of abscess about the rectum or of malignant disease. Organic disease of the brain must be excluded by the absence of local palsy, vomiting, intellectual disturbance, and optic neuritis. The pains of Bright's disease must be carefully excluded by search for albumen, signs of arterial thickening, and cardiac hypertrophy. So-called spinal irritation is accompanied by pains, which, however, fail to mark the district of particular nerves, and are vague and shifting. There is hyperæsthesia of the skin over some of the vertebral spines. Locomotor ataxy is characterised by pains of lightning-like rapidity, and neuralgic in character; but they shift, and are often accompanied by a staggering gait, sometimes by diplopia. Absence of the knee-jerk (the quadriceps extensor muscle at the same time responding freely to faradisation and blows), observed along with shifting neuralgic pains, is a strong indication of locomotor ataxy. The pains of syphilis in its second stage may be distinguished by the presence of fever, usually also of a rash, and by the fact that they affect many parts at once.

PROGNOSIS.—Youth, the absence of a strongly marked history of hereditary neurosis, the fact that neuralgia has followed exposures to unusual strain, violence, severe weather, or transient defects of nutrition, and that its attacks are influenced readily by treatment, afford a favourable prognosis. The onset of the disease after middle life, and its concurrence with signs of arterial degeneration, are

unfavourable as regards cure. Neuralgia of itself can scarcely be said to affect the duration of life. On the whole, neuralgia of the fifth nerve is the most persistent.

TREATMENT.—For success in treatment it is necessary that the basis upon which the neuralgia rests should be discovered. In patients suffering from malnutrition the diet should be ample and nutritious, and should include a fair amount of the fatty element, in the form of cod-liver oil, butter, or cream. A little stimulant may sometimes be necessary—enough to promote primary digestion; but no attempt should be made to relieve pain by its direct agency.

Where rheumatism is suspected as a cause of the neuralgia it should be treated by salicylate of sodium in twenty-grain doses three or four times a day. Two or three grains of iodide of potassium with fifteen of bicarbonate of sodium, taken every four hours, will often remove neuralgic pain connected with rheumatism. When malaria is suspected, it is well to follow up this treatment by quinine in doses of from five to ten or fifteen grains twice a day. A mercurial purgative may be usefully combined with a dose of quinine. If there be syphilis, iodide of potassium in ten-grain doses three times a day must be had recourse to; if gout, extract of colchicum may be given in one-grain doses twice daily, coupled with saline purgatives, especially Carlsbad natural salts. The salicylate of sodium is even more useful in gouty than in rheumatic cases. It should be given in doses of fifteen or twenty grains two or three times a day. Even where there is no history of malaria quinine will often be very useful, especially in neuralgia of the first division of the fifth (*see* TIC-DOULOUREUX). The liquor arsenicalis in doses of mij , increased cautiously to mviij or mx , and the tincture of steel, in doses of mxxx , largely diluted with water, may sometimes be used with advantage; and the latter will occasionally succeed even when there are no ordinary signs of chlorosis. As anemia may exist with a well-coloured face, the state of the gums and inner surface of the lower eyelid should be examined for undue pallor. Strychnine, in mij to mv doses of the solution three or four times daily, is especially useful in gastralgia; and belladonna, in $\frac{1}{4}$ gr. doses of the extract or mx doses of the tincture, in neuralgia of the pelvic viscera. Seclusion from irritation of various kinds—movement, cold, noise, dazzling light, worry—should be carefully maintained in cases of trigeminal neuralgia. All sources of peripheral irritation, of which decayed teeth, foreign bodies under the skin, intestinal worms, and imperfectly fitting boots are examples, should be carefully searched for, and where practicable removed. If lead be suspected, the drinking-water should be tested, and if the mineral be found iodide of potassium may be administered. Removal from imperfectly ventilated rooms, or from exposure to noxious gases, is essential. A warm, dry climate, such as Egypt or Algeria, will often cure when all other remedies have failed. For immediate relief in cases of extreme severity which resist other treatment, morphine may be injected hypodermically, either near the seat of pain, or in an indifferent part of the body. It is best used pretty freely diluted, mij of a solution of acetate of morphine, 1 to 30, being commenced with, and repeated, if necessary, when the pain returns. This dose may be gradually increased to one of mxv , but an effort

should be made to do with as little as possible and to avoid narcotic effects. The greatest care must be taken to avoid establishing a morphine-habit (p. 611). The following pill is often useful: R Quinina Sulphatis gr. j , Ferri Tartarati gr. ij , Morphinae Acetatis gr. $\frac{1}{12}$; repeated every hour or two when the onset is expected.

In all forms of neuralgia it is necessary to pay attention to the action of the bowels. In some cases considerable relief may be obtained by the use of small blisters (size of a florin), applied in the neighbourhood of the principal focus of pain, one following another at intervals of two days, not on but near the already blistered surface. The continuous current, derived from a sufficient number of cells of a battery to cause a characteristic feeling of burning, may be so applied that the affected nerve is as completely as possible included in the voltaic circuit. Sponges, or large leather-covered metallic disc-electrodes, moistened with warm salt water, should convey the current, and be kept firmly pressed upon the skin for about ten minutes; or, while one is stationary, the other may be slid along so as to linger in turn upon each focus of pain. To avoid shock the circuit should not be broken by the lifting of an electrode till the battery is 'let down' to zero. If relief be afforded, the application may be repeated many times a day, at intervals of a few minutes or hours. No notice need be taken of the position of the poles (+ and -), the object of the proceeding being simply to alter the electric tension of the tissues which are made to form part of the circuit. The method is often disappointing in its results, but sometimes gives a good deal of palliation.

In rare instances, but especially in ovarian neuralgia, the hypodermic injection of atropine ($\frac{1}{120}$ gr. to $\frac{1}{60}$ gr. of the sulphate) may prove serviceable. Where there is great restlessness and irritability of the nervous system, bromide of potassium in thirty-grain doses two or three times a day should be used. Relief, in slight cases of neuralgia, is obtained by applying to the skin by means of lint such liniments as the following: R Chloroformi zss , Tincturae Opii zss , Linimenti Belladonnae ad zij ; or R Spiritus Ammonia Aromatici, Aetheris, Tincturae Opii, Spiritus Vini Rectificati aa zj . Aconite and veratrine numb the sensory nerves, but they are uncertain remedies and very apt to cause irritation. Camphor-chloral with or without a little menthol may be applied with a camel's hair brush.

Phenazone in doses of five grains every half-hour, or from ten to twenty grains every four hours, is often very useful in neuralgia. An injection of mij of the *Injectio Cocaina Hypodermica* at the seat of pain will scarcely ever fail to produce at least temporary relief. The liability of the use of cocaine to induce a habit must, however, be carefully remembered (*see* HABITS). Phenacetin in doses of ten grains in cachets will frequently be of service. Exalgin may also be tried in doses of from two to four grains. The extract of cannabis indica, in doses of $\frac{1}{4}$ gr. frequently repeated, will sometimes produce great assuagement of suffering.

In unusually severe cases, which have lasted over years, a portion of the nerve may be excised; or, what is better, the nerve, which has been exposed by an incision, may be lifted from its bed and so firmly pulled upon as to be stretched, and then a portion excised (*see* NERVES, Diseases of). Very satisfactory results have sometimes followed this

procedure. Some time after the cure of a neuralgia there may be threatenings of a revival (dull heaviness, with tenderness of the part) following great fatigue or worry, but not immediately amounting to anything. Sleep is the best remedy for this condition, and this, if necessary, may be aided by giving ten grains of chloral hydrate.

For the treatment of coccygodynia see COCCYGO-DYNIA. T. BUZZARD.

NEURASTHENIA (νεῦρον, nerve; ἄ priv. ; and σθένος, strength).

DEFINITION.—A term used to describe a peculiar form of disease, sometimes spoken of as 'nervous exhaustion.' The name has, and with good reason, been objected to on account of its associations, and yet no better one has been proposed. Provisionally, therefore, since some name is essential, this must be used until a better is suggested.

SYMPTOMS.—The symptoms are protean, and vary so much in different cases that no accurate description of them can be given. They are very different, in the majority of cases, from those of 'hysteria' as it is generally understood, although no doubt 'hysterical' symptoms often co-exist. Many of the cases occur in clever, emotional, but not fanciful women, who would give all they possess to be well, and heartily long for good health, if they only knew how to obtain it; and in such cases the disease is as far removed as possible from the condition known as 'hysterical.'

In a large proportion of cases the origin of the illness can be distinctly traced to some cause injuriously affecting the nervous system, such as the loss of a near relative, monetary reverses, disappointments in love, or overstrain—of late so common in the modern system of high-class education in women. In the cases, comparatively rare, but still occurring from time to time in men, a similar origin from the vexations and strains of business affairs is often observed.

The disease is not, as a rule, suddenly established, but is the gradual outcome of deteriorated health. No distinctive or invariable symptom can be mentioned, but eventually there is a continuous inability for any exertion, a constant feeling of weariness and fatigue, until at last all effort is given up, and the patient gradually lapses into a bedridden or sofa-ridden invalid.

The appetite fails, all sorts of vague dyspeptic discomforts—flatulence, constipation, and the like—develop; and general, often excessive, emaciation is a common condition. There is, however, a well-marked, although less common, class of case, in which, along with all the symptoms of general weakness above referred to, there is a deposit of much unwholesome fat in the subcutaneous tissues, giving the patient a bloated, anemic, and very unhealthy appearance. The urine is pale, of low specific gravity, loaded with phosphates, with a diminished amount of urea, and sometimes a slight trace of albumen. Other vague nervous symptoms are present, especially sleeplessness, and various vaso-motor disturbances, such as palpitation and the like. Mental and emotional symptoms are pretty sure to be developed sooner or later, and are generally fostered by the use of drugs, constant fruitless attempts at cure, the habitual resort to chloral hydrate and other sedatives, and above all by the well-meant but often highly injudicious attentions of over-anxious relatives, nurses,

and, it must be added, doctors, which are rarely wanting.

Closely allied to this form of illness, and more properly termed hysterical, are a most important class of cases, in which there is a distinct imitation of real disease. Among these may be mentioned the so-called 'hysterical aepsia' of young girls, associated with most marked emaciation, loathing of food, and a strange unrest, leading to exhaustion of the ill-nourished muscles, or various forms of mimetic disease, such as paresis of the limbs, actual hysterical paralysis, hysterical vomiting, and the like. These, however, it is beyond the province of this article to dwell upon (see HYSTERIA). If the history of such cases be studied, it will be found that they show a lamentable record of fruitless attempts at cure, and in spite of all they have gone steadily down-hill.

TREATMENT.—To Weir Mitchell of Philadelphia undoubtedly belongs the merit of systematising a method of dealing with such cases, based on a common-sense appreciation of their causes, which renders them no longer an *opprobrium medicince*, but makes their recovery as nearly certain as anything medical can be, provided only that the cases are properly selected, and the treatment is intelligently and thoroughly carried out. This 'Weir-Mitchell' treatment is a systematised plan by which the weakened body is placed in thorough condition, by means of continuous rest, enforced feeding, and regular muscular waste produced by massage, which enables food to be taken and assimilated.

The essentials of this method are:—

1. Complete rest, the patient being placed in bed, and kept there during treatment; and it should be a *sine qua non* that this rest should not be in the patient's own house, but in a medical home or in lodgings, the friends and relatives, whose influence is often most injurious, being strictly excluded.

2. Regular muscular exercise to produce tissue-waste, by means of massage of the whole body, at first for ten minutes or a quarter of an hour twice daily, soon increased to an hour or an hour and a half. The influence of this is often misunderstood, and this treatment is frequently erroneously talked of as a 'massage treatment.' It should be borne in mind that massage is nothing more than a remedial agent, used for a specific purpose; that it is not the most important part of the cure; and that, used alone, and without enforced rest and over-feeding, as is unfortunately so often done, it cannot possibly be productive of any real good.

3. Feeding is the *most essential* part of the treatment. At first the patient should be placed on milk alone, about five ounces every three hour. Within a few days this is increased to ten ounces, so that at least two quarts are taken in twenty-four hours. Then, by degrees, solid food is added, so that within a fortnight the patient should be taking three large mixed solid meals daily, in addition to the milk, and often a cup of strong soup, with two teaspoonfuls of beef peptonoids added, twice daily as well. This exaggerated diet is continued for six weeks or two months, when it is gradually lessened, the massage also being discontinued, and the patient allowed to get up. In an average case the patient should gain from fourteen to twenty-three pounds during this time. It is strange to see how, with returning health, all invalid habits are lost, sleep becomes regular without drugs, the bowels cease to require assistance, and the whole appearance,

and apparently even the nature, of the patient is altered.

At the end of the treatment, in most cases, it is advisable that the patient should go for a change, either on a sea voyage or abroad, so as to complete the cure. At any rate, she should not return to her family until her health is re-established. The essential point to remember is that no half-measures should be permitted: if this treatment is not carried out thoroughly and completely, it had much better not be tried at all.

W. S. PLAYFAIR.

NEURITIS (*νεῦρον*, a nerve).—SYNON.: Periphereal Neuritis; Perineuritis; Interstitial Neuritis; Fr. *Névrite*; Ger. *Nerveneitzündung*.

DEFINITION.—Inflammation of a nerve, or of the fibrous sheath of a nerve.

ÆTIOLOGY AND PATHOLOGY.—This process occurs sometimes as an idiopathic change, the origin of which is altogether obscure, as where it implicates some of the intercostal or other spinal nerves, and is then often associated with an eruption of *herpes zoster* in related regions of the skin. At other times, as in some of the cases when it attacks the facial especially, or the sciatic nerve, neuritis seems to be set up as a result of local exposure to cold (see FACIAL PARALYSIS; and SCIATICA). Such forms of neuritis as these are commonly spoken of as 'rheumatic inflammations' of the respective nerves. Sometimes this appellation may be distinctly justified; but whether such changes have necessarily to do either with rheumatism or with a rheumatic predisposition seems, in many other cases, fully open to doubt.

Contusions or traumatic causes of various kinds may also set up inflammation in nerves. At other times a neuritis may be initiated in, and spread along, nerves leading from some wound or sloughing sore. This latter condition of things has been found to exist in some cases of traumatic tetanus (see TETANUS). Or an inflammation already existing may spread from some contiguous structure to adjacent nerves, as when cranial or spinal nerve-roots become involved in the course of a meningitis.

The apparently idiopathic forms of neuritis, as well as those following upon exposure to cold, are specially prone to show themselves when certain predisposing causes have been for some time in operation. Among these some of the best known and most frequently operative are the gouty diathesis, the syphilitic cachexia, and the presence of diabetes; in each of which neuritis in some form or other is of common occurrence. Other more specific predisposing causes of neuritis exist in the presence of leprosy, of epidemic influenza, or of poisoning by lead or arsenic. Many of the toxic causes, in fact, which are most effective in the production of a multiple symmetrical neuritis of parenchymatous type (see NEURITIS, MULTIPLE) are also capable of inducing a localised neuritis of peripheral or interstitial type, such as we are now considering—and this may vary much in intensity and also in its degree of acuteness or chronicity.

ANATOMICAL CHARACTERS.—Strictly speaking, we have mostly to do in this pathological state with inflammation of the sheath of the nerve, or of its interstitial tissues, rather than with changes in the nerve-fibres themselves. It is possible, of course,

that the nerve-fibres in this condition may undergo some distinctive pathological changes; but what is at present known is, that the neurilemma, or connective-tissue sheath of the nerve (including its minute prolongations between and around separate bundles of nerve-fibrils), becomes much more hyperæmic than natural, and that on microscopical examination there is to be found, in addition to the increased vascularity, a multiplication of new tissue-elements and the presence of migrated leucocytes. These changes may cause considerable swelling of the nerve-sheath and of its prolongations, and thus may produce either mere irritation or more or less compression of the nerve-tubules, according to the amount of new elements which accumulate in or are produced within the nerve-sheath. So that degenerative or sub-inflammatory changes in the nerve-fibres may, at least, often be found co-existing as secondary or induced phenomena.

All such changes may be localised to particular regions, or they may extend more or less diffusely along the whole length of a nerve.

SYMPTOMS.—The symptoms of neuritis will necessarily vary much according to the functions with which the affected nerve is concerned. There may be impairment of special or common sensibility, or pain may exist (referred to the peripheral distribution of the nerve), with more or less distinct tenderness along its course or at its point of emergence from some bony canal. In these cases the pain is generally paroxysmal and neuralgic in character, while the skin is hyperæsthetic, and perhaps shows some vaso-motor or trophic changes. All these phenomena are well illustrated in diseases of the trigeminus (see TRIFACIAL NERVE, Diseases of), and therefore need not be dwelt upon further here. Where a strictly motor nerve is implicated, there may be twitchings of the muscles to which it is distributed, followed by more or less distinct paralysis, and subsequently marked wasting of the affected muscles—conditions which are well exemplified in inflammations of the facial nerve (see FACIAL PARALYSIS). In the case of a mixed nerve being involved, both kinds of symptoms present themselves—that is, more or less severe pains and tenderness, with trophic symptoms, and distinct paresis or paralysis, with subsequent atrophy of the muscles to which the nerve is distributed. A detailed consideration of such phenomena would be needless here, as they are set forth in relation to inflammation in the sciatic nerve, under the article SCIATICA; and also because all such symptoms and changes are likewise considered under the article NEURITIS, MULTIPLE.

There is, undoubtedly, an intimate relationship in many cases between different forms of neuritis and neuralgia affecting similar sites; the reader may, therefore, be further referred to the article NEURALGIA for additional information.

TREATMENT.—The treatment of neuritis is both general and local. The general treatment is of especial importance in cases where the condition seems attributable to the influence of syphilis, and then the administration of small doses of perchloride of mercury, in combination with large doses of iodide of potassium, will often produce marvellously beneficial results. Smaller doses of iodide of potassium alone, or with colchicum, are to be given in other cases, in which rheumatism or gout may seem to be one of the factors in exciting the nerve-inflammation. But in these cases, and also in those

which are simple results of exposure to cold, the cure may be often expedited, and the patient also temporarily relieved, by local treatment, such as the application of a few leeches (especially in the early stages), hot fomentations, or small flying blisters.

During the course of the treatment special symptoms may become all-important. Thus, pain may become so agonising as imperatively to demand measures for its relief; and, where paralysis is one of the symptoms, galvanism or massage (or perhaps both) must be employed daily, or two or three times a week, in order to prevent as much as possible the muscles from degenerating while the pathological condition in the nerve is being cured—that is, in cases in which a cure is possible. All these indications, however, will be found more fully considered under the articles SCIATICA; and NEURITIS, MULTIPLE. H. CHARLTON BASTIAN.

NEURITIS, MULTIPLE.—SYNON.: Polyneuritis; Peripheral Neuritis; Fr. *Paralysies Périphériques*; Ger. *Multiplex Neuritis*; *Neuritis Acuta Progressiva*.

DEFINITION.—A nervous affection characterised by various motor, sensory, and trophic symptoms; dependent upon disease occurring simultaneously, or in rapid succession, in the peripheral terminations of nerves in various parts of the body, under the influence of extrinsic or intrinsically derived toxic agents of one or other kind.

ANATOMICAL CHARACTERS.—The morbid changes that occur in the nerves in this disease are met with principally at their finer terminations, or, it may be, even exclusively there. Where the larger branches of the nerves are also involved, the intensity of the process generally diminishes as we recede from the periphery. A second peculiarity is that the changes are most frequently symmetrical on the two sides of the body, whether the nerves affected be in the limbs, where they are by far the most frequently involved, or in other parts. A third peculiarity is that the inflammatory or degenerative changes are found to occur essentially in the nerve-fibres themselves, rather than in their sheaths or interstitial tissues; or, in other words, that the inflammatory changes are in the main parenchymatous, rather than interstitial as they are in simple neuritis. Of course these two kinds of change do not occur singly in either case, but in multiple neuritis it seems plain that the parenchymatous changes are distinctly in excess, and they are believed also to be primary rather than secondary degenerative changes due to strangulation of the nerve-fibres higher up, as some have imagined. Though primary parenchymatous changes may be the rule, we may still admit the occurrence also of secondary degenerative changes in the nerve-fibres.

The naked-eye alterations in the appearance of the nerves may not be great. Their extent depends in part upon the amount of co-existing inflammation in their connective-tissue envelopes, and in part also upon the severity and duration of the inflammatory process in the nerve as a whole. But even where they are not notably swollen and hyperæmic, the nerves may be deficient in their proper lustre, and irregular in their contour, owing to accumulations of fat derived from degenerated myelin; or, in later stages still, they may be wasted and reduced to little more than mere connective-tissue strands. It may often happen, however, that no very appreciable changes are to be detected by

the naked eye, and that, in order to establish the existence of multiple neuritis, the finer branches of the nerves have to be dissected out and submitted to careful microscopical examination.

Where changes exist in the sheath and interstitial tissues of the nerves, they are such as have already been described in the article NEURITIS, consisting in the main of increased vascularity, some amount of exudation of serum, migration of leucocytes, and multiplication of nuclei in the sheath of Schwann and elsewhere. The distinctive changes that occur in multiple neuritis are, however, those which take place in the nerve-fibres themselves. These agree in almost all respects with the changes that occur in the 'secondary degeneration' of a nerve on the distal side of a section or other severe injury. The myelin first becomes cloudy and granular, and then undergoes segmentation—first into large and subsequently into smaller and smaller masses, until at last there is left only a number of fatty-looking globules and particles surrounding the swollen and altered axis-cylinder. As this process advances the sheath of the nerve gives way here and there; surrounding cells swell, and, taking up the fatty particles, become converted into 'granulation-corpuscles,' while ultimately the axis-cylinder may be more or less completely destroyed.

However extreme the changes in the nerves may be, it is now certain that, in periods of from six to twelve months or more, complete regeneration may take place, just as it has been observed to occur in experimental lesions in the lower animals. New axis-cylinders become formed in continuity with the old, and these subsequently become surrounded with sheaths of myelin. Thus it is that the prognosis in a given set of symptoms caused by peripheral neuritis is so much better than it would be if the symptoms had been dependent upon similarly severe lesions occurring in the spinal cord itself.

Atrophic degeneration of muscles, in relation with the affected nerve, progresses *pari passu* with the changes of the latter. The muscle-fibres waste from the occurrence of fatty and granular degeneration, while the nuclei of their sheaths multiply, and hyperplasia of tissue-elements also takes place in the intervening connective tissue.

Occasionally in some of the worst cases, in which death has occurred, it has been established that small changes of different kinds occur also in the spinal cord. Such changes have been found more especially in the cases of multiple neuritis caused by alcohol, lead, arsenic, diphtheria, and chill; but they may well occur also where multiple neuritis follows in the wake of other causes. The changes met with here for the most part consisted of atrophic conditions in the great ganglion-cells of the anterior cornua, together with some amount of over-growth in the surrounding neuroglia.

In addition to these more or less essential changes other morbid conditions might be expected in internal organs, varying widely in their nature in different cases, in accordance with the varying conditions under the influence of which the multiple neuritis has been established; that is, we may meet with visceral changes due to previous alcoholism, to malarial poisoning, or to some antecedent acute specific disease, as the case may be.

ÆTIOLOGY AND PATHOLOGY.—The best-known forms of this disease are undoubtedly due to the action of poisons circulating in the blood upon the peripheral terminations of nerves in different parts

of the body, and especially upon those of the extremities. The evidence of this mode of causation is so strong for so many forms of the affection that it is now pretty legitimately conjectured to be, in one way or another, applicable to all of them—that they are in fact all toxic forms of paralysis, though capable of being arranged into different groups according to the nature, or the mode of production, of the toxic agencies in question. From this point of view the best-attested causes of multiple neuritis may be roughly classified in the following manner:—

A. *Poisons of Extrinsic Origin.*—Alcohol; Arsenic; Bisulphide of Carbon; Lead.

B. *Poisons evolved by Microbes associated with various Infective or Endemic Diseases.*—Diphtheria; Variola; Typhoid Fever; Typhus Fever; Measles; Beriberi; Septicæmia; Malaria; Influenza; Syphilis; Leprosy; Tuberculosis.

C. *Poisons evolved during Metabolic Processes occurring in some of the Organs and Tissues of the Body itself.*—Chill; Excessive Muscular Exercise; Diabetes Mellitus; Gout; Cachectic states; Idiopathic or 'Spontaneous' Cases.

The cases belonging to (A) the first category supply, of course, the clearest evidence that multiple neuritis is due to the presence of poisonous substances circulating with the blood, and thus exerting an irritative influence on the peripheral nerves.

The diseases comprised in (B) the second category, in the course of which, or as sequels of which, multiple neuritis has been found to occur more or less frequently, are all of them associated with the growth and multiplication of micro-organisms either in the blood or in some parts of the body. Under such circumstances it is well known that alkaloidal and other substances of a poisonous nature (toxins) are more or less abundantly produced, as excreted products, during the life-activity of the micro-organisms, which substances, finding their way into the blood, may act upon the peripheral nerves in much the same manner that alcohol or arsenic does. It is well known that such effects do not occur in all cases, but only in a comparatively small proportion of the cases of each of these diseases. We must, therefore, suppose that, in the cases in which multiple neuritis is produced, there has either been some slight variation in the nature of the poisons finding their way into the blood, or else that the ordinary poisons associated with the several diseases are aided, in such individuals, by the co-existence of one or more intrinsic favouring conditions.

In regard to the third category (C) of cases, in two of them at least it is known that the blood is especially apt to be altered in composition, and to contain products which may exercise an irritative influence upon the peripheral nerves in some parts of the body. The excess of uric acid in the blood may operate in this way in gout; while in diabetes the irritative effects upon the nerves are not supposed to be due to the presence of an excess of sugar in the blood (since the frequency of the association of the disease with neuritis bears no regular relation to the amount of sugar contained therein), but rather to the presence in the blood of some acid or ethereal products (derivatives of β -oxybutyric acid), such as are known to occur at times in association with diabetes mellitus. It seems perfectly certain, also, that some of the cases of multiple neuritis are set up after exposure to chill and cold; just as, in other subjects, acute spinal paralysis may be engendered under similar conditions. These are the cases of

so-called 'rheumatic' origin; and it is conjectured that in them some poisonous product is formed within the system, as a result of the chill, which, according to individual proclivity or the nature of the products engendered, excites either multiple neuritis or acute spinal paralysis, or it may be both; just as, under other individual conditions, the malady excited is an acute articular affection, or an acute inflammation of some other internal organ. Lastly, in certain cachectic or anæmic states of the system there is a tendency to the occurrence of neuritis; and perhaps it is principally to this kind of causation or to the occurrence of slight exposure to cold that we may ascribe the so-called idiopathic or 'spontaneous' cases of multiple neuritis—that is, cases for which no very distinct cause of a toxæmic character can be assigned.

If we omit the aetiologically obscure cases last referred to, it will be seen that all the conditions under which multiple neuritis is prone to occur are states of the system in which poisons of one or other kind would be circulating with the blood; and such a mode of origin for the inflammatory condition of the nerves is rather confirmed by the fact of the remarkably symmetrical distribution of the neuritis that is so commonly met with in these cases, and to which reference has already been made.

We must not omit to mention, moreover, the fact of the relation supposed to exist between Raynaud's disease and peripheral neuritis. While referring to the article on that disease (see RAYNAUD'S DISEASE) for further information, it need only be said here that multiple neuritis may or may not show itself as an epiphenomenon rather than as an essential constituent of this remarkable morbid condition.

Then, again, it should be said that 'acute ascending paralysis' (Landry) has been held by some, and especially by the late Dr. Ross, to be one of the idiopathic forms of multiple neuritis. From the clinical point of view there is undoubtedly much to be said in support of this doctrine, but the proof of this position has yet to be established. See SPINAL CORD, Special Diseases of.

Multiple neuritis occurs with all degrees of severity, not only at different times under the operation of the same kind of cause, but also very notably under the influence of the different causes that have been above enumerated. The latter difference is so striking that the several causes above referred to may, from the point of view of the average intensity of the multiple neuritis which they engender, be divided into two classes, as follows:—

Causes of more Intense Forms of Multiple Neuritis. Beriberi; Alcohol; Arsenic; Chill; Diphtheria; Variola; Excessive Muscular Exercise; Typhus Fever; Typhoid Fever; Measles; Influenza.

Causes of Slighter Forms of Multiple Neuritis. Bisulphide of Carbon; Lead; Malaria; Septicæmia; Syphilis; Leprosy; Diabetes Mellitus; Gout; Tuberculosis; Cachectic states.

The cases of multiple neuritis that occur under either of the first class of causes may be, and often are, well-developed typical forms of the malady; those, on the other hand, which occur from one or other of the second class of causes are much more habitually ill-developed and more or less localised forms of the affection, and in some of these cases (especially those associated with leprosy, syphilis, or gout) it is the sheath and interstitial tissues of the nerve, rather than the nerve-fibres themselves, which are most prone to be affected. In these

cases, also, the inflammation is often not limited to the peripheral twigs, but may involve the main trunks and the roots of the nerves. In all respects, indeed, there is in these cases often more the characters of a local neuritis than of the more general affection.

It should be borne in mind, again, that in many of the cases of multiple neuritis there may have been more than one cause in operation; thus, alcohol and anæmic conditions of the system, or syphilis, may co-operate with tuberculosis; chill or lead with alcohol; or local exposure to wet and cold may reinforce syphilis or gout in the production of some of the more localised forms of multiple neuritis. The recent widespread epidemic of peripheral neuritis in certain parts of the north of England, due to the presence of arsenic in one or other of the ingredients used in the manufacture of beer, has, however, shown that one of the most potent combinations for the production of this disease is a mixture of arsenic with alcohol.

In the more intense forms of multiple neuritis lesions may occur in the spinal cord as well as in the peripheral nerves. This is notably the case in some of the 'rheumatic' forms of the disease, and in some of the cases due to lead, diphtheria, or some other of the acute specific diseases.

Neither in the more intense nor in the more localised forms of the disease are we able definitely to bridge the gap which lies between our knowledge of the ætiology and that of the morbid anatomy of this affection—we are unable to say anything definite, that is, as to the exact pathogenesis of the changes in the peripheral terminations of the nerves or in the spinal cord. No light whatever is really thrown upon this obscure subject by the hypothesis that functional changes in spinal trophic centres first occur, which lead secondarily to the observed failure or perversion of nutrition in the peripheral nerves. It would only shift the difficulty farther back, even if this hypothesis explained (which it does not) the localisation of the changes in the nerves to their peripheral terminations. As it is, we can only suppose that the tissues affected, and the particular parts of them, are, by their individual constitution, especially prone to be irritated by the presence in the blood of the various poisons to which we have referred.

Multiple neuritis from one or other of the causes mentioned may occur at almost any age; but the disease is by far the most frequently met with some time between the twentieth and the fiftieth year.

Some separate description or mention must now be made of the different forms of multiple neuritis.

A. Cases due to Common Poisons.—*Multiple Neuritis due to Alcohol.*—This is the commonest of all the forms of multiple neuritis, and it is met with also much more frequently in women than in men. Spirit-drinking seems to be most prone to excite it, and especially when spirit is taken in small or moderate quantities continuously over long periods, and when at the same time there has been little exercise in the open air. While alcoholic neuritis is more common among women, delirium tremens is more frequent among men—differences which may be dependent partly upon sex, but perhaps more notably upon the different habits of the two sexes, especially in regard to extent of open-air exercise.

SYMPTOMS.—The onset of the affection is generally gradual, being often preceded for months or

weeks by gastric symptoms, insomnia, and rapid pulse, together with numbness and tingling, shooting pains, tremors, cramps, and some paresis of the limbs—especially of the lower limbs. Actual loss of power may then come abruptly at any time, at first in the feet and legs, later on in the thighs—though, often before they become involved, the hands and arms also show signs of paralysis, which is most marked in the extensors of the wrists. In many instances the paralysis may stop here; but in the more severe cases it gradually advances so as to involve the nerves supplying the trunk muscles, some of the cranial nerves, and at last perhaps the phrenic nerve.

The 'dropped wrists,' and the 'dropped feet,' as the patient lies in the recumbent position, are very characteristic of multiple neuritis; and the paralysis of the limbs is almost always of the flaccid type. The affected muscles speedily become flabby and much wasted. When in this condition they show a more or less modified electrical 'reaction of degeneration.' They usually cease to respond to faradism; and if they respond at all to galvanism, it is only to very strong currents, and that in a slow, sluggish manner, the reaction to A.C.C. being greater than that to C.C.C. In some exceptional cases, and in the early stages of the affection generally, the electrical reactions may be very little altered.

Various disturbances of sensibility cause the greatest distress to the patient. The limbs are often the seat of excruciating pains, the skin is more or less generally hyperæsthetic, and the muscles also in the affected parts are extremely tender, even to slight pressure. There is often, moreover, marked tenderness along the course of the nerves, so that the patient is apt to cry out when touched or moved even in the gentlest manner. In addition, various paræsthesiæ, such as tingling, numbness, and formication in the limbs, are mostly present. In the more severe cases these latter symptoms may disappear during the height of the malady, when anæsthesia becomes developed, but may be expected to return as recovery advances. With cutaneous anæsthesia of the limbs, often localised to the terminal portions or to the areas of particular nerves, there may also be more or less marked loss of muscular sense. Some of the special senses may also be affected, especially vision, leading to amblyopia and contraction of the field for colour-vision. Occasionally, also, inequality and slight contraction of the pupil has been met with, and very rarely optic neuritis. The cutaneous reflexes are often present, but may be lost where the anæsthesia is marked. The knee-jerks are commonly lost at an early stage of the affection; but occasionally, in exceptional cases, they are found to be retained, and even to be slightly exaggerated for a time.

In many of the slight cases of the disease a certain amount of inco-ordination as well as paralysis can be recognised; and in the cases in which the involvement of the muscular sense is more than usually marked, a slight ataxic condition is produced in both the arms and the legs, but especially in the latter. This, in combination with other symptoms present, such as the pains in the limbs, the loss of the knee-jerks, unsteadiness when the eyes are closed, and a peculiar high-stepping gait, causes the patient's condition to resemble more or less closely that met with in locomotor ataxy. Such cases are now spoken of *pseudo-tabes*; and, although

they may be met with in multiple neuritis from any other cause, they seem to be rather specially frequent in the form of the disease due to alcohol, though perhaps still more so when it is due to arsenic or to a combination of these toxic agents. The points which enable us to distinguish these forms from true tabes will be referred to later under the head of 'Diagnosis.'

Vaso-motor and trophic symptoms occur in some cases about the feet and hands, in the form of oedema, lividity, glossy skin, and more or less profuse sweating. Later on, the limbs are still further altered, especially in old neglected cases, by the occurrence of contractures of the fingers and of the calf-muscles, due to weakness of the extensors. Bed-sores are usually absent; and power over the sphincters of the bladder and rectum is commonly retained.

In the early stages of alcoholic neuritis there is another class of symptoms of great importance commonly present, namely, those of cerebral type. These are sometimes most marked, and of a very varied character. There may be loss of memory, loss of ability to concentrate the attention, together with illusions and hallucinations—especially towards evening and at night, when insomnia is often most obstinate and very exhausting to the patient. In some cases there may be distinct delirium continuing for days; and, even where this is not present, there is often a peculiarly active imagination, together with a sort of waking dream-like state. Thus, patients will give circumstantial details of imaginary events in which they have taken part, or of visits which they have paid within the last hour or so, when they have been quite incapable of even leaving their beds. In the course of a week or so, under proper treatment, such symptoms will gradually subside. The patient will then (or earlier where the more acute symptoms are absent) show distinct evidence of a lowered *morale*. Statements about their habits in regard to alcohol are almost always unreliable, often flagrantly untrue, and made even in the face of the strongest evidence to the contrary. We must look to the relatives or the associates of the patient if we are to obtain correct testimony in regard to this matter.

The course of the disease in alcoholic neuritis is pretty constant, though its duration is extremely variable, according to the degree of severity of the attack. After a slow or more abrupt onset the symptoms go on increasing up to a certain point; then there is a decline of the more urgent symptoms in a gradual fashion; after this the patient may remain in a more or less stationary condition for many months; finally, a period of slow improvement sets in, during which the tenderness of the muscles abates, and their nutrition and power improve, the anaesthesia diminishes, and with it there is a return of numbness and tingling in the hands and feet. The duration of the disease varies from two or three months to from one to two years.

Multiple Neuritis due to Bisulphide of Carbon.—Delpsch showed that the effects of this compound upon the system were somewhat similar to those of alcohol. The symptoms are principally met with in rubber-factory operatives. It appears that intense frontal headaches are common among workers in rubber, even though no further symptoms develop. As the intoxication proceeds, according to F. Peterson, 'giddiness and even actual drunkenness become manifest, the workpeople becoming excitable,

talkative, and hilarious.' Among later chronic manifestations are multiple paresis, due to a multiple neuritis, mental weakening and apathy, amblyopia, tinnitus, fornication, anaesthesia of the feet, muscular cramps, occasionally convulsions, at first increase of sexual appetite, later impotence in men and sterility in women. In the same communication Peterson records three cases of acute mania resulting from inhalation of the fumes from a mixture of bisulphide of carbon and chloride of sulphur. The poisonous effects from the use of these chemicals are now obviated to a considerable extent by maintaining a more thorough ventilation in rubber-factories. See OCCUPATION-DISEASES.

Multiple Neuritis due to Arsenic.—It has been long known, from experiments by Orfila, that in certain cases paraplegic conditions were induced occasionally after the taking of large doses of arsenic. Of late years it has been ascertained that the poisonous effects of arsenic are sometimes very similar to those produced by alcohol. The paralytic effects thus produced were previously ascribed to changes in the spinal cord; but it has now been ascertained that such changes are only occasional, and that the majority of the effects to which we are referring are due rather to peripheral neuritis. These effects occur sometimes, though only very rarely, after the patient has taken for a prolonged period small doses of arsenic, such doses at last giving rise to symptoms of acute poisoning. Then, after a variable interval—some days, or a week or two—the symptoms of peripheral neuritis begin to develop.

In other cases, however, such symptoms may set in a short time after acute poisoning by arsenic, when the drug has been taken in excess either accidentally or with suicidal intent. A typical case of this latter type has been recorded by H. Jolly. The patient had taken, with a view to suicide, a quantity of Schweinfurth Green. There was immediate vomiting, and for two days symptoms of gastritis. She remained very weak, and noticed, when she attempted to rise on the fifth day, some numbness of the feet. Then followed paræsthesiæ simultaneously in feet and hands, with a creeping sensation that was at times very painful. These symptoms continued during the next few weeks, and to them was soon added a more distinct weakness, especially of the lower extremities. After four weeks the patient was unable to walk alone; and when, in the following week, she was admitted into the hospital for nervous diseases, she could only walk when supported on both sides. There was also marked ataxy. The knee-jerks were lost on both sides, and there was great paresis in the legs, with specially impaired movement in the feet and toes. In six weeks the paralysis of the feet and toes was complete; there was atrophy of the muscles of the calves of the legs, and great disturbances of sensibility. The patient was insensible to slight touches, but felt stronger touches well; and there was pronounced hyperalgesia. The tips of the fingers were quite anaesthetic, and there were sharp pains in the hands and in some of the deeper parts of the forearm and arm. In the course of the following week the symptoms became somewhat worse, especially the pains. A feeling of coldness was also complained of in the right upper extremity, and there was profuse sweating in the palms of the hands. From about the twelfth week onwards a decided improvement began to take place in regard to both motor and sensory symptoms.

The superficial reflexes in arsenical polyneuritis are mostly preserved, while the knee-jerks are lost; the legs also are commonly more affected than the arms. There is much variation met with in the symptoms of individual cases, and in many of them distinct impairment or loss of muscular sense and ataxic symptoms are fairly well marked (pseudo-tabes). In almost all respects, in fact, the symptoms of the multiple neuritis due to arsenic are found to accord closely with the alcoholic form of the disease, with the very important exception that the head- or cephalic symptoms, with hallucinations and delirium, are generally absent, while more or less marked pigmentation of the skin is apt to show itself in some persons.

Our knowledge of the symptoms due to chronic poisoning by arsenic has, unhappily, been much extended of late owing to the occurrence of a widespread epidemic of multiple neuritis in certain parts of the north of England caused by the presence of arsenic in different ingredients used in the manufacture of beer. This poisoning has been going on to some extent for several years, but it attained its maximum in the latter part of 1900, when its cause was first pointed out by Ernest Reynolds of Manchester. To him as well as to Judson Bury we are principally indebted for a considerable increase in our knowledge of the symptomatology of the multiple neuritis due to arsenic—or rather to arsenic and alcohol in combination, for there is little room for doubt that the activity of arsenic in the production of this malady has been notably enhanced by the fact of its having been taken in association with alcohol in the form of beer. We are led to this conclusion from the fact that arsenic is taken by many epileptics in association with bromides over long periods and in amounts quite equal to those imbibed by even the most intemperate of the beer-drinkers who have suffered from multiple neuritis in the north, and yet without the production of any such symptoms. It is possible, of course, that bromides may to some extent diminish the poisonous effects of arsenic (though they do not always prevent pigmentation of the skin), but it seems clear that its poisonous influence is heightened by being taken in association with alcohol.

The mixed effects of these two poisons as met with in the recent epidemic have often shown themselves in the following order: (1) digestive symptoms; (2) laryngeal catarrh, bronchitis, and acute skin-affections; (3) disturbances of sensibility; (4) motor paralysis and chronic skin-affections.

The face was often noticed to be puffy, especially about the eyes; the eyes were suffused and watery; and the face often showed some amount of pigmentation. Pigmentation of the skin also in various parts of the body, especially where subject to pressure from clothing, and in special regions, having a patchy or punctiform arrangement, was found to be very common. Erythematous eruptions, with much itching; herpes zoster; and keratosis of the hands and feet; as well as erythromelalgia (p. 491) of the same parts, were likewise frequently met with.

Mental symptoms were commonly slight. Sensory symptoms were rather more marked, and pseudo-tabes more frequent, than with the simple alcoholic form of the disease. Tenderness of the muscles to deep pressure was commonly very marked. In some of the cases there was notable failure of the heart's action, with the usual oedema of feet and legs, and pulse of low tension.

Multiple Neuritis due to Lead.—The effects of the multiple neuritis caused by lead are still more localised than those due to arsenic alone. There is not only the absence of the characteristic group of head-symptoms, but still further a common limitation of the neuritis to the motor nerves, and especially to those of the upper extremity. The ordinary effects of peripheral neuritis due to lead will be found described under LEAD, POISONING BY. The symptoms closely resemble those due to spinal-cord disease, to which, indeed, some of the effects are often partly attributable. For, though peripheral neuritis is the lesion commonly met with, it happens here even more frequently than with other forms of multiple neuritis that lesions in the anterior cornua of the spinal cord are also produced; and it is to these latter lesions that the symptoms are occasionally, in part at least, attributable.

But although it is the rule for the effects of the multiple neuritis due to lead to be thus limited to a part of the field of distribution of the motor nerves of the upper extremities, it is not invariably so. Thus, muscles about the shoulder and arm (especially the deltoid and brachialis anticus) may be affected as well as those of the forearm; and occasionally even some of the leg-muscles, especially the long extensors of the toes and the peronei muscles. Again, in some cases of multiple neuritis due to lead there have been distinct disturbances of sensibility, that is, more or less severe pains, some tenderness of affected muscles, and perhaps also along the affected nerves, together with tingling and numbness in their field of distribution. In certain other cases of multiple neuritis due to lead we may have the association of a more or less marked hemiplegic condition, in which hemi-anæsthesia is well developed. This latter condition is generally of more or less brief duration, and is supposed to be due to some functional defect in the brain, analogous to that occurring in hysteria. In this connection it must not be forgotten, moreover, that intense double optic neuritis and also convulsions may occur in the course of lead-poisoning.

B. Cases due to Microbic Poisons.—*Multiple Neuritis associated with, or sequential to, certain Infective or Endemic Diseases.*—The cases belonging to this category are those forms of multiple neuritis which are associated with, or, as is by far the most common, are sequential to, one or other of the following morbid conditions, namely, diphtheria, variola, typhoid fever, typhus fever, measles, beriberi, septicæmia, malaria, influenza, tuberculosis, syphilis, or leprosy.

Almost all these varieties of the disease were formerly ascribed to morbid conditions, either functional or structural, of the spinal cord. It is true, indeed, that functional diseases as well as certain structural affections of the spinal cord, in the form of indurations (scleroses), softening, hæmorrhages, together with limited inflammations or atrophic conditions of the anterior cornua, are especially apt to occur occasionally as sequels of one or other of these diseases. But since the clinical characteristics of polyneuritis have been more commonly appreciated, it has been recognised that these particular groups of symptoms occur not infrequently as sequels of one or other of the diseases above mentioned—the truth of this diagnosis having, moreover, been often confirmed by necropsies which have demonstrated the presence of

multiple neuritis, mostly alone, but more rarely with some co-existing changes in the spinal cord.

The severity of the disease varies very much indeed in this category of cases. Sometimes it presents itself as an acute progressive affection involving all the limbs, and subsequently the nerves supplying the trunk muscles and those of the face and eyes, together with a well-marked association of sensory symptoms of the usually varied character. At other times, and especially after diphtheria, the motor paralysis is less marked, and symptoms of a more ataxic type present themselves (pseudo-tabes); while, in other cases still, we may have to do with a localised rather than with a more generalised neuritis. The forms of multiple neuritis associated with diphtheria are apt to occur, on different occasions, in each of these types; the severe general cases, however, in which death may occur from paralysis of the diaphragm are rare and exceptional; while localised forms of paralysis limited to the palate, and associated with slight parætic or ataxic conditions, are by far the most common (see DIPHTHERIA). On the other hand, in beriberi we have to do much more frequently with a generalised form of neuritis, in which speedy death occurs not at all rarely (see BERIBERI). In other of these diseases, such as variola, typhus and typhoid fevers, measles, septicæmia, and malaria, polyneuritis supervenes rarely; but in each of these affections it may appear in a well-developed form.

Again, it seems clear that in influenza, tuberculosis, syphilis, and leprosy, in the absence of other co-operating causes, the neuritis that occurs is apt to present itself in a more or less localised form; and, in the case of syphilis and leprosy more especially, the neuritis is principally of the peripheral and interstitial type. These forms of the disease are thus, perhaps, rather more closely allied to ordinary neuritis than to multiple neuritis. Still, it seems probable that both syphilis and tuberculosis may decidedly favour the action of other more potent causes, such as alcohol or chill, in the production of well-marked multiple neuritis.

The only one of these forms of multiple neuritis to which any special reference need be made is that which may be associated with malarial poisoning. Sometimes this, either alone or aided by other causes, such as exposure to cold or excess of alcohol, induces ordinary forms of multiple neuritis of medium severity, affecting the arms and legs, but especially the latter. At other times, however, malaria gives rise to what, since the description given by Romberg, has been known as 'intermittent paraplegia.' This remarkable affection has been found to occur only in patients suffering from malarial poisoning. It is characterised by the sudden onset of paralysis in the lower extremities, which may or may not be accompanied by some anæsthesia and by paralysis of the sphincters. After some hours the paralysis disappears, perhaps accompanied by the appearance of a critical sweat, and reappears in a more or less regular manner, according as the attack assumes a quotidian, tertian, or quartan type. This affection has been found to be very amenable to treatment by quinine. Cases have been recorded by excellent observers, such as Hartwig, Erb, and Westphal, and it is now generally believed that, instead of being due to altered functional conditions of the spinal cord (which was the interpretation formerly given), this remarkable transitory and recurrent form of paralysis is depen-

dent rather upon a peripheral neuritis occasioned by some poison formed during the course of a malarial attack.

C. Cases due to Autogenetic Poisons.—

Multiple Neuritis sequential to Chill.—The disease from this cause not infrequently supervenes in an acute fashion, and may at the onset be associated with distinct febrile symptoms, the temperature rising to some point between 101° and 103° . There may also be pains in the back, limbs, and joints, so as to give rise to the suspicion, perhaps, of a commencing attack of rheumatic fever. To such symptoms may be added headache and anorexia, together with tingling and numbness in the fingers and toes, and more or less tenderness over the principal nerves in the limbs. The legs and arms subsequently show an increasing paralysis, commonly beginning first in the legs. Then paralysis may spread to the trunk-muscles, and to those of the tongue, palate, and larynx (so as to impair deglutition and articulation), even if it does not extend to other cranial or to the phrenic nerves. The knee-jerks are speedily lost, the paralysed muscles soon waste, and their electrical reactions become altered in the usual manner. At the same time various sensory disturbances become developed, such as tenderness of the muscles and hyperæsthesia of the skin (soon to be followed by, or intermixed with, areas of anæsthesia); while glossy skin, increased sweating, and other trophic symptoms may also make their appearance.

These cases subsequently pursue much the same course as severe cases of alcoholic neuritis. Death may occur in the course of a week or ten days from respiratory or cardiac paralysis; or, all fever having subsided, the patient may remain in a tolerably stationary condition for several weeks, before the usual slow improvement begins to set in.

In other instances the multiple neuritis following chill may be less acute and less severe, and the amount of sensory symptoms is very variable. When the latter are very slight, such cases are apt to be mistaken for those of acute spinal paralysis; and when they are more severe (especially where there is much involvement of the muscular sense) the symptoms are more of an ataxic order, so that the case may present itself as a form of pseudo-tabes.

Multiple Neuritis sequential to Excessive Muscular Exercise.—Although typical cases have been recorded, verified by necropsy, seemingly referable to this cause, they are nevertheless of extremely rare occurrence. So far as the form and course of this variety of multiple neuritis are concerned, it seems to agree most closely with that induced by chill, and different cases are liable to individual variation in much the same manner.

Multiple Neuritis associated with Diabetes, Gout, or with Cachectic states.—Each of these varieties of multiple neuritis is liable, though only very rarely, to occur as a more or less generalised affection. As a rule, however, it is much more common for the neuritis when associated with either of these affections, and when other co-operating causes are absent, to occur in a limited form, affecting only some one or two nerves—the inflammation that is established being also peripheral and interstitial rather than parenchymatous in type. Gout has been long known as a common predisposing and even exciting cause of ordinary interstitial neuritis.

Multiple Neuritis of 'Spontaneous' Origin.—Every now and then ordinary attacks of multiple neuritis, sometimes severe and sometimes slight, make their appearance without its being possible to assign them to any of the known causes of the disease, and for which, in fact, no distinct cause can be traced. These are the so-called 'spontaneous' or idiopathic cases of multiple neuritis, which are presumably due to autogenous poisons of some kind.

COMPLICATIONS.—Looking to the varied conditions under which multiple neuritis may arise, it is only natural to expect that the complications should be rather numerous; and this they are found to be. As poisoning by alcohol is the cause of so very large a proportion of the cases of multiple neuritis, we have principally to do with concurrent complications assignable to this cause. Thus the patient may suffer from obstinate symptoms of gastric catarrh, or the liver may be enlarged and hard, or smaller than natural, and other indications of cirrhosis of this organ may exist. It must not be forgotten, moreover, that in the cachectic subjects of chronic malarial poisoning there may be enlargement of spleen as well as of liver. Other complicating effects of alcohol may be due to a chronic or subacute meningitis, the existence of which leads to a great aggravation of the head-symptoms in the form of delirium, or even to a chronic maniacal condition; while in other cases complicating symptoms, due to chronic or subacute spinal meningitis or degenerative conditions of the spinal cord, may be present. Here also—as where gout, or lead, or both combined, have been operative causes of the multiple neuritis—we may find distinct evidences of co-existing renal disease. Pneumonia and phthisis are also frequently present as complications, the latter supervening even in cases where it does not seem to have been present as a cause.

DIAGNOSIS.—It is the great variety in the symptomatology of multiple neuritis, and the fact that it may be associated with actual disease in the spinal cord, which give rise to the principal difficulties in the way of diagnosis. Very many of the cases, however, are so typical in the combination of symptoms presented, as to allow little room for doubt in regard to the proper diagnosis. Thus, more or less paralysis of both feet and both hands, with 'foot-drop' and 'wrist-drop,' or more extensive paralysis of all four limbs of a flaccid type, associated with pains, numbness, and tingling; hyperæsthesia of the skin, marked tenderness of the limb-muscles and perhaps also along the principal nerves, together with absent knee-jerks and the altered electrical reactions which have been described, form such a typical combination that it should be considered to be indicative of multiple neuritis from some cause. And if with this grouping of symptoms there co-exists the combination of cerebral symptoms previously noted, we may feel just as certain that we have to do with a multiple neuritis caused by chronic alcoholic poisoning, even though no distinct evidence of this be at first forthcoming—nay, even though excess in alcohol may be firmly denied by the patient and her friends.

It will be seen that the diagnosis of multiple neuritis from spinal diseases is in the great majority of the cases easy, because pains in the former are mostly prominent symptoms, and because locomotor ataxy is the only form of spinal disease associated with severe pains that could easily be mistaken for multiple neuritis, and that only with

certain exceptional forms of the disease. For there are only four other spinal diseases in which pains in the limbs are apt to constitute prominent symptoms. These are the paralyses associated with cancer of the vertebræ, with scrofulous pachymeningitis, and with cervical hypertrophic pachymeningitis—in each of which (not to mention other characteristic features) there are generally present exaggerated knee-jerks and ankle-clonus—and the somewhat rare affection, syringomyelia, which is almost always characterised by loss of painful and of thermal sensibility in the parts principally affected, while in multiple neuritis these are almost always modes of sensibility that remain unaffected.

There are, however, three varieties of polyneuritis which are especially liable to give trouble in regard to diagnosis. These are, in the first place, acute cases due to chill or to over muscular exercise, in which there happens to be an almost complete absence of pains and other sensory symptoms. Here the condition has to be diagnosed from acute spinal paralysis, or even from acute ascending paralysis. The second set comprises those of the pseudo-tabetic type, in which pains are present, and the diagnosis has to be made from locomotor ataxy. The third class consists of mixed cases, in which multiple neuritis is actually complicated with lesions in the spinal cord or its membranes, or in both. Here there may be at times much room for doubt as to the nature of the affection—that is, whether it is in the main spinal, or in the main peripheral, and to what extent the symptoms of the two conditions can be separated from one another.

Some more definite details must therefore be given in regard to the diagnosis of each of these three forms of the disease.

1. The cases of acute spinal paralysis may agree with some of the more acute cases of polyneuritis in the rapid mode of onset, with or without slight febrile symptoms, in the existence of an atrophic and flaccid form of paralysis in the limbs, together with a more or less complete electrical 'reaction of degeneration' and abolition of knee-jerks. In other forms of polyneuritis, however, the onset is apt to be more gradual and progressive, even in the most acute cases; there is also much more frequently a bilateral symmetry in the parts affected; and even the cases that are most free from sensory symptoms are almost never quite free from such accompaniments, though this is more the rule with cases of acute spinal paralysis. Of course it is only in the early days of these affections that there is room for doubt. After the first few days the progress of the two affections is commonly different. Thus, in acute spinal paralysis the loss of power is at first widespread and simultaneously caused, though after a few days there is a subsidence of the paralysis in some of the parts first attacked. While in multiple neuritis there is a progressive increase in the area of the paralysis during one or more days; and after the paralysis has attained its full development there is no recession of paralytic symptoms till a distinct interval of weeks, or it may be months, has elapsed.

The other disease that may have to be thought of in the early days of one of these acute cases of multiple neuritis is the rare affection known as acute ascending paralysis, concerning which so much doubt exists as to its real nature (*see SPINAL CORD, Special Diseases of: Acute Ascending Paralysis*). This form of disease may be distinguished by the complete absence of sensory symptoms, by

the preservation of the knee-jerks, and by the fact that the paralysis does not progress in the same way that it does in multiple neuritis: in the former it is a more strictly ascending disease, affecting the trunk-muscles after the legs, and then involving the arms; while in the latter the order is almost invariably legs, arms, trunk, and there is also rather less tendency for the bulbar centres to be affected. A little later on, if any doubt should still remain, the absence of muscular atrophy and the preservation of normal electrical reaction would definitely settle the diagnosis in favour of acute ascending paralysis.

2. In reference to the diagnosis of cases of pseudo-tabes from locomotor ataxy, it should be borne in mind that these exceptional forms of multiple neuritis are met with principally after poisoning by alcohol or arsenic, or in the form that is sequential to diphtheria. And then, in reference to the conditions presented, although there may be many characters common to the two affections, there are generally marked differences in the total symptomatology. Thus, in the more rapidly developed pseudo-tabes there are not the characteristic lightning-pains, but more enduring pains, with much more of tenderness in muscles and nerves, together with numbness and tingling in the hands and feet. There is more frequently hyperæsthesia in the limbs, perhaps mixed with patches in which there is analgesia as well as anæsthesia; there may also be some amount of atrophy of muscles, with altered electrical reactions in the direction of the 'reaction of degeneration.' Then, again, there is the absence in multiple neuritis of three signs which are commonly present in locomotor ataxy, viz. the Argyll-Robertson condition of pupil, and temporary bladder-symptoms, together with loss of sexual desire and power. A girdle sensation is also much more likely to be absent in these forms of multiple neuritis, though it has occasionally been met with; and then, again, the lost knee-jerks may return during recovery from multiple neuritis. There still remains the question of the differences in gait characteristic of the two affections. What is known as the *steppage* gait (or the high-stepping gait) has been described as distinctive of multiple neuritis. Its peculiarity is due to the loss of power in the flexors of the ankle, and the consequent dependent attitude of the toes, so that the patient has to lift the foot high, as though he were stepping over a slight obstacle; while in locomotor ataxy, the toes, instead of being dependent, are raised, the heels being generally brought to the ground first, and also in a more irregular and spasmodic manner. Where present the steppage gait is doubtless a sign of value, but it is not always present when it is most wanted in this ataxic class of cases—that is, where paresis of the flexors is, as often happens, present only to a very slight degree.

3. In other cases, where spinal and peripheral lesions co-exist, it may be much more difficult to recognise the real nature of the affection, or, if this can be done, to say which symptoms belong to the one and which to the other set of lesions. In regard to cases where thrombotic softenings, myelitis, or small hæmorrhages co-exist with multiple neuritis (as they may do, more especially, in some few of the cases sequential to the acute specific diseases), it can only be said that such cases are too complicated to be considered here, and that there are

no forms of disease where the aid of an expert is more required in order that a correct diagnosis may be made. It may, however, be added that there are four signs in particular which, when present, may safely be considered to depend in the great majority of cases upon spinal rather than upon peripheral lesions: these are, paralysis of the sphincters, bed-sores, exaggerated knee-jerks with ankle-clonus, and, though less certainly, girdle-sensations.

It should further be borne in mind that there are two affections especially in which the co-existence of peripheral with spinal-cord symptoms is common, viz. acute spinal paralysis and locomotor ataxy. So that where, by reference to the points already laid down, it can be recognised that we have to do with symptoms of double origin, the point to be considered is, whether the central or the peripheral symptoms are primary, and which are merely complicating phenomena. This is a point of considerable importance in regard both to prognosis and to treatment. It is highly important, for instance, to know, in an affection caused by exposure to cold, whether the symptoms are in the main peripheral or whether they are in the main spinal but with some complicating peripheral neuritis. So also it should be remembered that the ataxic cases of multiple neuritis (cases of pseudo-tabes) stand at one end of a series of cases, and at the other stand the somewhat rare cases of locomotor ataxy in which symptoms due to peripheral neuritis are altogether absent; while between these extremes we have almost every kind of transition, furnished either by cases of locomotor ataxy with an increasing number of peripheral lesions, or by cases of pseudo-tabes due to multiple neuritis complicated perhaps by some spinal lesions. The more the patient's symptoms are of peripheral origin, the greater is the relief that is to be expected from treatment.

PROGNOSIS.—Many indications relating to prognosis have already been given, so that little requires now to be added. It may be said, however, that in the great majority of cases life is not imperilled by multiple neuritis. But exceptions to this rule occur in the case of the severer forms of beriberi, and also in the more acute cases occurring in this country, due either to chill, alcohol, or diphtheria, in which the respiratory muscles (including the diaphragm), and the heart, are apt to become paralysed. Otherwise death takes place in multiple neuritis almost solely from the co-existence of one or other of the complications to which reference has previously been made. As to the question of ultimate cure, this, if the cases are not too old and neglected, may in the majority of instances be effected after a prolonged course of treatment. Slight cases may, of course, be cured in the course of a month or two; but in the more severe forms of the disease twelve or eighteen months at least may be required. It is indeed surprising to see the extent to which recovery occurs in this class of cases, even when they are of the worst type, as compared with the amount of improvement that could alone be expected if a similar amount of paralysis with muscular atrophy had been due to spinal rather than to peripheral disease. The signs of commencing improvement to be looked for during the stationary period of the disease are a return of the tingling and numbness in the hands and feet, increased ability to move these parts, together with a gradual improvement in the electrical reactions and

in the firmness and size of the muscles, as well as a progressive diminution of the glossiness of the skin, and sweating in the hands and feet. The return of previously lost knee-jerks may also be looked for.

TREATMENT.—In all cases of multiple neuritis we should, before commencing systematic treatment, have thoroughly decided, as far as it may be possible, what the causal conditions are that have been influential in producing the neuritis, as the first necessity may be to put a stop to some poisoning of the system. It is of the greatest importance to arrive at this knowledge as early as possible, as the longer the poisoning lasts the more protracted and obstinate is the resulting malady. Thus, a rubber-worker should be taken away from his work at once. Steps should be taken to protect the patient from lead or arsenic if either of these have had to do with its development; or, as is most frequently the case, where alcohol is the cause this should at once be cut off completely, whenever the condition of the patient will admit of it. But if too great weakness of the heart be present to permit of this being done, the alcohol should be greatly reduced in quantity at first, and as soon as possible cut off altogether.

In alcoholic cases also, where we have to do with severe pains, mental disturbance, and insomnia, together with gastric symptoms, the greatest care is needed. The latter symptoms may be best checked by keeping the patient upon a strict spoon-diet, pancreatising the milk and beef-tea if necessary, and administering only very small quantities at a time. In the more urgent cases it may be needful at first to have resort to nutrient suppositories. Sleep should be ensured, and the other symptoms relieved as soon as possible by giving morphine, either by subcutaneous injection or by mouth; or, in cases where the symptoms are less urgent, by the administration of full doses of bromide of potassium. At the same time, we must do our best to relieve the local pains and tenderness, by the application of light warm anodyne fomentations, by wrapping the limb in cotton-wool and oil-silk, or occasionally by the use of cold evaporating lotions, if these seem to give more relief to the patient.

In the acute stage of the disease which follows chill, where there are febrile symptoms and perhaps some slight general pains, relief may be derived from salicylate of sodium or salol, given for a few days in full and frequently repeated doses as for rheumatic fever; bromide of potassium may also be given with these drugs night and morning where the restlessness is great and headache is severe. The patient should be kept upon spoon-diet as long as the temperature remains at all elevated; and in all the more severe forms of the disease a water-bed is desirable.

In the less severe cases, to whatsoever cause they may be due, the patients should be kept at rest in bed, partly with a view to warding off any aggravation of the disease, and partly to protect them from cold. In these cases, and also in the more severe forms of the disease, after the acute pains and tenderness have subsided, the diet must be more abundant, though easily digestible and nutritious. Pains may be relieved and sleep favoured by such drugs as acetanilide or phenazone, though where insomnia continues these remedies may be supplemented by bromide of potassium,

chloralamide or trional. At the same time, much may be done by means of tonics to improve the appetite and general health, where, as is so often the case, this has been much impaired by the previous causative conditions. When these have taken the form of malaria, gout, or syphilis, some special treatment may be necessary; otherwise we must trust principally to combinations of iron with small doses of strychnine, aided by extract of malt and cod-liver oil. In cases where there is no marked anæmia, iodide of potassium in six- to eight-grain doses may be given with tincture of nuxvomica instead of the compound of iron, at this stage of the disease, as it may help to allay pains and relieve any accompanying condition of interstitial neuritis. During all this time, in alcoholic cases, alcohol in every form should be entirely forbidden; and to ensure the absolute observance of this order the strictest precautions must be taken. No trust whatever can be placed in the patient in such cases, and injudicious friends (or servants who may be bribed or threatened) should be guarded against. The patient ought to be under the absolute charge of thoroughly reliable nurses, either at home or in some private institution or hospital.

Local treatment must also be assiduously carried on week after week, and month after month, at this stage of the disease. As soon as the tenderness has sufficiently subsided to permit of it, daily massage and gentle passive movements should be had recourse to, and to this very shortly should be added warm or sulphur-baths two or three times a week, and the regular application of galvanism to the atrophied muscles. Even if they do not respond much at first to any currents that can be used without causing pain, the galvanism should be persevered with, and after a time the muscles will begin to respond.

From the first, care should be taken to prevent, as far as possible, the limbs getting into a contracted position. Thus, the knees are apt to be drawn up for the relief of pain in the early stages of the affection, and, if allowed to remain in this position, contractures with rigidity will inevitably result. The 'dropped foot' from weakness of the anterior tibial muscles soon becomes associated with slight contraction of the calf-muscles and shortening of the tendo Achillis. This defective position of the foot may, however, be obviated to a considerable extent if care be taken from an early stage to prevent the foot falling forward by means of some suitable support. This may be most conveniently and effectually done by means of elastic tapes from the anterior part of the foot, fastened on each side of a band encircling the leg just above the knee. Contractions of the wrist and fingers must also be obviated as far as possible. This is best ensured by an early resort to passive movements and massage; and the same means will generally suffice gradually to overcome contractions that may have occurred at the ankles or the knees. Where the former are obstinate, they will in time generally yield when efforts to stand are commenced, and the weight of the body is day by day brought to bear upon the contracted tendo Achillis. It is very rarely indeed that section of tendons becomes necessary.

H. CHARLTON EASTIAN.

NEUROMA (νεῦρον, a nerve).—A tumour connected with a nerve. See NERVES, Diseases of; and TUMOURS.

NEUROSES (νεῦρον, a nerve).—SYNON.: Fr. *Neuroses*; Ger. *Nervenleiden*.

DEFINITION.—Affections of the nervous system occurring without any known material cause, without inflammation or any other constant structural change which can be detected in the nervous centres. In other words, functional affections of the nervous system.

Many of the disorders which may be included here are characterised by symptoms such as neuralgia and convulsions, which also accompany other disorders associated with morbid changes. It is very necessary, therefore, in inquiring into any particular case, not to rest satisfied with the presumption that the disorder is functional until the condition of the nervous centres has been investigated; lest, regarding the symptom as the disease, the central mischief to which it is due may be overlooked. It is highly probable, moreover, that most of what we now regard as functional diseases will, on further investigation, be found to depend upon some corresponding nutritive change in the organ affected—an inference which is being daily verified.

ENUMERATION.—The neuroses may be classified according to the organs or functions involved:—

(a) *Visceral neuroses*, namely, those of the respiratory, circulatory, or digestive organs.

(b) *Localised paralyses*, for instance, palsy of the facial and other peripheral nerves.

(c) *Localised involuntary or reflex movements*, such as spasm of the facial nerve and writer's cramp.

(d) *Disorders of general sensibility*, including the various forms of neuralgia—trigeminal, cervico-occipital, sciatic, crural, &c.

(e) *General neuroses*, namely, chorea, epilepsy, catalepsy, hysteria, and allied affections.

(f) *Disorders of the mental faculties*—hypochondriasis, melancholia, and other forms of mental derangement.

For detailed descriptions of these, see the articles on the various organs concerned.

P. W. LATHAM.

NEUSCHMÉCKS.—See SCHMÉCKS.

NICE, on the French Riviera.—Warm, dry, bracing, winter climate. Temperature 50° F. See CLIMATE, Treatment of Disease by.

NICTITATION (*nictito*, I wink often).—A rapid involuntary winking of the eyelids, usually due to some nervous disturbance. See CHOREA; and FACIAL SPASM.

NIEDERBRONN, in Lower Alsace.—Muried saline waters. See MINERAL WATERS.

NIEDERSELTERS (Selters), in Nassau.—The well-known muried alkaline table-waters. See MINERAL WATERS.

NIGHT-BLINDNESS.—See NYCTALOPIA.

NIGHTMARE.—This is a condition characterised by an abiding sense of discomfort or extreme uneasiness, occurring in the midst of a disturbed sleep, sometimes associated with a feeling of weight at the epigastrium, in conjunction with more or less definitely oppressive dreams. It is principally associated with the taking of a heavy meal or of indigestible food before going to sleep by some persons, especially those of a nervous temperament, whose digestion is weak. A closely allied condition is,

NIPPLE, DISEASES OF

however, apt to be met with as a consequence of brain-exhaustion and chronic disturbance of sleep in those who are overworked, by application either to study, business details, or literary pursuits. Such a condition also has its affinities with certain forms of incipient delirium, occurring either in various febrile diseases or as a result of alcoholic excesses. See SLEEP, Disorders of.

H. CHARLTON BASTIAN.

NIGHT-SIGHT.—See HEMERALOPIA.

NIGHT-TERRORS OF CHILDREN.—The child who is the subject of this ailment may have gone to bed apparently in perfect health and gone to sleep, but will suddenly arouse the household some two hours later with a piercing shriek, and will be found in an agony of terror pointing at some imaginary object and imploring that it be taken away, or crouching in a corner of the bed in the hope of getting away from the dreaded thing. As a rule with judicious soothing the child will before long drop off to sleep again and may sleep perfectly soundly for the rest of the night, and in the morning have no knowledge of the occurrences of the previous evening; the same scene may be enacted on subsequent, but not necessarily successive, nights. If the child suffers from any form of gastro-intestinal disturbance or from 'adenoids,' it is probable that the trouble would be of the nature of nightmare and would be removed by appropriate treatment; but on the other hand there may be no such exciting cause discoverable, while the child is of a highly nervous disposition and there is a family predisposition to neurosis. According to Coult's the true subjects of this ailment all belong to this class, and in them it is not infrequently the forerunner of epilepsy, and has been preceded at an earlier period of life by the occurrence of infantile convulsions. It is equally common in the two sexes, and is most often met with about the commencement of the second dentition, but may occur in children as young as two years. Small doses of bromide of ammonium at bedtime (a grain for each year of the child's age) is the best treatment, though antipyrine has been credited with the power to prevent an attack if given at bedtime. Such children should not be left alone at night or in the dark. If the attack occurred at the time of the second or first dentition, a dental surgeon should be consulted, and in all cases attention should be paid to the diet.

JOHN ABERCROMBIE.

NILE, The.—A very dry winter climate. Mean temperature, winter, 57° F. Unsuitable for cases of active pulmonary disease. See CLIMATE, Treatment of Disease by.

NIPPLE, Diseases of.—SYNON.: Fr. *Maladies du Mamelon*; Ger. *Krankheiten der Brustwarze*.—Some of the more ordinary affections of the nipple will be found described under BREAST, Diseases of; and LACTATION, Disorders of. Here it is proposed to treat of certain graver diseases, which claim a separate consideration.

1. Cancer.—The nipple may be the seat of epithelioma, which commonly commences as a crack or fissure, with an indurated base, often in the areola or at its junction with the nipple. It may be a squamous-celled carcinoma, in which case it differs in no respect from similar disease of the integument of adjoining parts. In some cases it is

a duct-cancer, the more superficial part consisting of columnar cells, and the deeper part of spheroidal epithelium. Hard carcinoma too may attack the nipple, involving its deeper structures, and producing general induration and enlargement, so that the diseased mass projects from the summit of the breast like a knob or large nut. The disease probably originates in the epithelium of the galactophorous ducts, or in that of the sebaceous glands.

2. **Paget's Disease.**—Of greater interest than either of the preceding is an affection frequently associated with malignant disease of the breast, to which Paget first drew attention—an *eczematous* condition of the nipple and areola. It may occur in the form of a dry, scaly, or branny eruption, affecting the entire surface of the areola and nipple, which is darker coloured, a little firmer, and less pliant and elastic than its fellow. Or, with more characteristic signs of inflammation, small vesicles or pustules may form, and, breaking or being rubbed off, may leave behind them tiny scabs or ulcers, or a surface raw and red. Either condition may exist for many months or even years with little alteration, and with scarcely any tendency to spread beyond the margin of the areola. But the second form, causing more irritation than the first, is often subjected to treatment; and being very difficult to cure, is sometimes so severely treated with caustics that destruction ensues, not of the disease, but of the nipple, which appears to have been gradually eaten away by the eczematous affection. Both forms are uncommon, but they are rare before the middle age. A study of their clinical and pathological characters leads to the conclusion that they are due to inflammation. The disease has been noticed in men as well as women.

TREATMENT.—This disease may be treated by protecting the parts with a carefully adjusted, ventilated shield, and by the application of vaseline, or liniment of lead and oil, or similar soothing dressing. But it is very intractable, in some cases apparently incurable. It might seem as if an affection so trivial were not worthy of so much attention; but unfortunately there appears the strongest reason to believe that these conditions of the nipple and areola are not infrequently the precursors of carcinoma of the breast, sometimes by only a few months, more often by a period of years. It is probable, too, that the carcinoma is directly due to the eczematous disease; for it induces changes in the epithelium of the ducts which can be traced deeply into the substance of the breast, whose acini become at length distended with proliferating epithelium. On this account it has been proposed, when all the lesser methods of treatment have been used in vain, to remove the entire breast. Opinions, which are divided on the necessity of this severe measure, are united in its favour when, with the superficial inflammation, there exists an appreciable induration, however slight, within the breast. Care must be taken not to confound these eczematous affections of the nipple and areola with those more widely diffused superficial inflammations of the breast, with which they have little in common, either in the obstinacy with which they resist treatment, or in the deeper disease to which they may give rise.

3. **Sarcoma.**—Sarcoma occasionally occurs in the skin covering the breast. It is either spindle-celled or melanotic: in either case its early removal is advisable.

4. **Benign Formations.**—Among the innocent tumours of the nipple and areola of the breast are (1) *lupus*, which should not be mistaken for the malignant dermatitis known as Paget's disease of the nipple; (2) *molluscum contagiosum*, small pearly white tubercles which may infect the cheeks and eyelids of any child suckled at the breast; (3) painful *subcutaneous fibromata*, which cause intense neuralgia of the breast and local tenderness; (4) *sebaceous cysts*, formed in some of the 15–20 sebaceous glands situated in the areola; (5) *nevi*; and (6) the *pedunculated fibromata* of the nipple. The fibromata often contain glandular elements, they may be warty in appearance, and are so well supplied with blood by arteries which run in the pedicle, that they must not be snipped off without previous ligature.

5. **Syphilis.**—Syphilis affects the nipple and areola of the breast in the primary, secondary and tertiary stages of the disease. Direct infection is usually the result of suckling a syphilitic child while it has mucous patches about its mouth. *Primary* sores, therefore, are met with among wet nurses and in those persons who consider it an act of courtesy to give an occasional feed to a neighbour's child. One or both breasts may be affected by a shallow, circular and painful ulcer with a smooth, glazed surface, and only very slight parchment-like induration at its base. Sometimes there is only a slightly indurated fissure, or the induration may be diffuse without ulceration; occasionally the ulceration is phagedænic. The true nature of the affection is apt to be overlooked since there is no reliable history, and the sores are multiple and very often atypical. In such cases the very slight amount of pain attending the fissure or ulcer and the early enlargement of the axillary glands, which are hard and bullet-like, should raise a suspicion of primary syphilis; while the appearance of secondary symptoms with an examination of the suspected child, where possible, will confirm the diagnosis. The eruptions of *secondary* syphilis and condylomata may occur upon the areola, while the circular and irregular ulcers of *tertiary* syphilis may occur upon the nipple as they do in other parts of the skin. The *treatment* of each of the conditions mentioned above does not differ either locally or constitutionally from that adopted for syphilis.

6. **Tuberculosis.**—Tuberculosis but rarely affects the nipple or its areola in either sex. It needs prompt surgical treatment owing to the tendency shown by the inflammation to spread along the ducts and lymphatics until the breast becomes affected.

HENRY T. BUTLIN.
D'ARCY POWER.

NOCTAMBULATION (*nocte*, in the night; and *ambulo*, I walk).—A term for sleep-walking. See SLEEP, Disorders of.

NOCTURNAL INCONTINENCE.—Involuntary escape of urine during sleep. See MICTURITION, Disorders of.

NODE (*nodus*, a swelling).—A circumscribed swelling on the surface of a bone, connected with the periosteum, and usually due to syphilis. See BONE, Diseases of; and SYPHILIS.

NOMA (*νομή*, a corroding sore; from *νέω*, I devour).—**SYNON.**: Fr. *Nome*; Ger. *Wasserkrebs*. A synonym for cancrum oris. See CANCRUM ORIS.

NOSE, Clinical Examination of.— Before proceeding to examine the nasal passages a preliminary inspection of the external parts should be made. The patency of the passages should be tested by successively closing each passage, and directing the patient to breathe through the other. The nasal passages can be inspected from the front through the anterior nares, or from the back, through the naso-pharynx. The former method is termed 'anterior rhinoscopy,' the latter 'posterior rhinoscopy.' Exploration with the probe, and digital palpation, are often necessary supplements to inspection.

In order to examine the nasal passages from the front, the observer should be seated upright facing the patient. It is always better to employ reflected light, in the same manner as for examination of the larynx (*see* LARYNX, EXAMINATION OF). Having concentrated a bright circle of light on the patient's nose, we can obtain a view of the interior of the vestibule by making the patient tilt the head slightly back, and raising the tip of the nose with the thumb. In order to examine the interior of the nasal fossa, a speculum must be used to dilate the cartilaginous aperture. There are several different forms of nasal speculum, but Duplay's bivalve speculum is perhaps the most generally useful. The speculum should be inserted gently with the thumb and forefinger of the left hand, and the blades separated slowly, the patient's head being tilted slightly backwards while this is being done. The anterior end of the inferior turbinated body will first attract attention, forming a rounded prominence projecting from the outer wall. If the patient's head be now tilted a little forwards, the convex surface and lower border of the inferior turbinated body can be traced backwards for a considerable distance, or even for the whole length, presenting usually an irregular wavy outline. The floor of the nasal passage can often be traced to the posterior extremity; and, if a strong light be directed back along the floor, the posterior wall of the pharynx can sometimes be discerned. If the patient's head be thrown backwards, the middle turbinated body will be seen above and behind the inferior. The anterior border, and the angle between this and the inferior, are the parts chiefly seen. Between the middle turbinated body and the septum is a narrow space, known as the olfactory slit, which can only be imperfectly illuminated. The spaces beneath the inferior and middle turbinated bodies, respectively, can be illuminated to a limited, but variable extent. When the patient's head is thrown well back, the fore part of the roof will be brought into view, but the superior turbinated body cannot be seen.

The actual extent of the parts seen by anterior rhinoscopy varies much in different cases. A deflected septum is a serious obstacle. Vascular turbulence of the inferior turbinated body, especially at the anterior end, often interferes with the view. This swelling can be reduced by the application of a 5-per-cent. solution of the hydrochlorate of cocaine. By its constricting effect on the blood-vessels, cocaine contracts the mucous membrane of the nose to a greater or less extent, wherever it is applied, and is an invaluable aid to a thorough rhinoscopic examination of the nasal passages.

By the method of examination known as posterior rhinoscopy, the posterior nares and naso-pharynx can be inspected. A small mirror, about half or five-eighths of an inch in diameter, is used in the

examination. A small laryngeal mirror serves the purpose, but the mirror should be attached to the stem at more nearly a right angle than is usual for laryngoscopy, and it is convenient to have the handle slightly bent to follow the curve of the tongue. A rhinoscope such as Michel's, however, is much more convenient. In this instrument the mirror is in the same plane as the handle, and can be raised to any angle by pressure on a spring.

The patient sits upright, with the head inclined slightly forwards, and is directed to open the mouth and breathe quietly and naturally. The light is concentrated from a reflector on the palate and pharynx, in the same manner as in laryngoscopy. The tongue must generally be depressed with a spatula. One with the handle at right angles to the blade is most convenient. The rhinoscopic mirror, previously warmed, is introduced rapidly, with its reflecting surface upwards, to one or other side of the uvula, close to, but not touching, the wall of the pharynx. By depressing the handle or by pressing on the spring, according to the form of rhinoscope used, the mirror can be made more vertical, and by various movements of the mirror an image of the different parts can be obtained. Difficulties may occur in the examination. The commonest is the drawing up of the uvula and soft palate against the pharyngeal wall as soon as the examination is begun. The patient should be directed to breathe quietly through the nose, and the effort to do this will cause the velum to hang away from the pharyngeal wall. If this does not succeed, the patient should be made to emit the French nasal sound *en*, or *on*. This will often succeed in giving a view of the parts. Painting the fauces with a 10-per-cent. solution of the hydrochloride of cocaine will often be an assistance. In certain cases, where the depth of the pharynx is small and the soft palate long, a view can only be obtained by drawing the soft palate and uvula forwards by means of some form of palate-hook.

Only a limited portion of the post-nasal region is seen reflected in any one position of the mirror. When the mirror is first raised into a nearly vertical position, the posterior surface of the velum is brought into view. By inclining the mirror to a more horizontal position, the septum appears as a sharp, whitish ridge, which serves as a landmark to the parts in this region. On either side of the septum, the ovoid openings of the nasal fosse, the choanæ, may be brought into view by slightly turning the mirror. Each choana is largely occupied by the posterior ends of the turbinated bodies, the most conspicuous of which is the middle. The inferior is largely hidden by the soft palate, as is also the lower end of the septum. By inclining the mirror to one side, the depression leading to the Eustachian orifice is seen, bounded above and behind by a prominent ridge. By bringing the mirror to a nearly horizontal position, the vault of the naso-pharynx and the region of the pharyngeal tonsil are brought into view.

The probe is often necessary to supplement anterior rhinoscopic examination. By its means the consistence, mobility, and mode of attachment of swellings, hypertrophies, and new-growths can be determined, and the presence of sequestra, foreign bodies and rhinoliths made out. Occasionally a probe, suitably bent, is useful to determine the condition of the parts seen in the posterior rhinoscopic image.

Digital palpation through the anterior nares is rarely practised, but in the naso-pharynx it is an extremely important method of examination, and when, as in children, posterior rhinoscopy is difficult or impossible, digital exploration is the only method open to us. For the purpose of this examination the patient is seated, and the operator stands facing the patient's right side. The left arm is passed round the patient's head, and the left hand rests on the patient's chin. The head is thus steadied. The right forefinger is passed rapidly to the back of the pharynx, inserted behind the soft palate, and then pushed boldly upwards. In this way the posterior ends of the turbinals, the pharyngeal tonsil and other structures can be rapidly explored. A prop may be placed between the teeth, as a preliminary, but it is not really necessary.

JAMES BARRY BALL.

NOSE AND NASO-PHARYNX, Diseases of.

SYNON.: Fr. *Maladies du Nez et du Pharynx Nasal*; Ger. *Krankheiten der Nase und des Nasenrachenraums*.

The diseases of the nasal orifices, nasal fossae, septum nasi, naso-pharynx, and of the accessory cavities of the nose will be considered under the following headings:—

1. Acute Rhinitis and Naso-pharyngitis.
2. Chronic Nasal and Naso-pharyngeal Catarrh:
(a) Simple Chronic Rhinitis; (b) Hypertrophic Rhinitis.
3. Chronic Atrophic Rhinitis (including Ozena).
4. Rhinorrhoea.
5. Post-Nasal Vegetations (Adenoid Growths).
6. Syphilis of the Nose.
7. Tuberculosis of the Nose (Lupus and Tuberculo Disease).
8. Affections of the Nasal Septum.
9. Foreign Bodies in the Nose (including Rhinoliths).
10. Parasites in the Nose.
11. Rhinoscleroma.
12. Nasal Diphtheria.
13. Tumours of the Nose and Naso-pharynx.
14. Affections of the Accessory Sinuses (Maxillary, Frontal, Sphenoidal, and Ethmoidal).
15. Reflex Nasal Neuroses.

EPISTAXIS, HAY-FEVER, and OLFACTORY SENSE are dealt with in separate articles.

1. Acute Catarrhal Rhinitis and Naso-pharyngitis.—SYNON.: Acute Nasal and Naso-pharyngeal Catarrh; Acute Coryza; 'Cold in the head.'

ÆTIOLOGY.—Acute coryza is usually an independent affection, but it may be found in association with some other malady, such as one of the specific eruptive fevers. The disease is most frequent in cold and changeable weather, and is almost certainly of microbic origin. It has an abrupt onset and usually runs a regular course. Sometimes it is found in an epidemic form, especially when many individuals live under the same roof and share the same rooms. Acute rhinitis may also be caused by irritating fumes and certain drugs, especially iodide of potassium; and it is occasionally a prominent feature in influenza. In most cases chill is the accreted cause. See CHILL.

SYMPTOMS.—The patient feels out of sorts, has a sense of chilliness, slight headache, and a feeling at first of irritation and then of dryness and fulness in the nose, with frequent attacks of sneezing.

There is occasionally a rise of temperature (99.5° to 101°) during the first two days of the attack; and the patient complains of feeling tired and heavy in the head. His pulse is quickened; the skin feels hot and dry; and pains in the back and limbs are frequently complained of. Engorgement of the submucous erectile tissue and rapid œdematous swelling of the mucous membrane lead to blocking of the nasal passages. The changes are most marked in the mucosa and submucosa covering the inferior turbinal bones. This sudden obstruction to nasal respiration necessitates breathing through the mouth, which intensifies the dryness of the pharynx and naso-pharynx. At first, notwithstanding the tumefaction and hyperæmia, there is no secretion from the nasal mucosa. Soon, however, a thin serous acid discharge which frequently excoriates the margins of the nostril and the upper lip is poured out, and this, in the course of two days or less, generally becomes muco-purulent. The sense of smell is largely impaired or lost. The catarrhal process may spread up the nasal duct and lacrymal passages and involve the conjunctiva, or may extend into one of the accessory cavities of the nose, most commonly the frontal sinus. Very frequently the naso-pharynx is secondarily involved; in many cases, however, particularly in children in whom post-nasal growths are present, the catarrhal process commences in the naso-pharynx and spreads thence downwards to the nose.

When the *naso-pharynx* is involved the symptoms are those of dryness and heat, described by the patient as being 'behind and above the nose.' Soon a thick viscid muco-purulent secretion is observed, which the patient has to 'hawk' away, as blowing the nose does not seem to clear the naso-pharynx sufficiently. The process is very prone to involve the Eustachian tube and to give rise to deafness, tinnitus aurium, and pain in the ear—these symptoms being caused by obstruction to the lumen of the Eustachian tube and retention of mucous, muco-purulent, or even purulent secretion in the tympanum. Every succeeding attack of acute naso-pharyngeal catarrh is prone to cause further enlargement of any existing post-nasal growths and to run into a chronic form of naso-pharyngeal catarrh which is a serious menace to the ears.

An uncomplicated cold in the head lasts only a few days, generally from four to seven. It must be remembered that an acute rhinitis may be the first symptom of an acute specific fever, particularly of measles or of influenza.

TREATMENT.—Mild cases call for little treatment beyond avoidance of exposure. In others the attack can be cut short by frequently repeated doses (4 grs.) of quinine. If the rise in temperature be above 100°, or if there be any complication, rest in bed is called for. The employment of diaphoretics gives relief to most of the symptoms. A mixture consisting of Liquor. Ammon. Acetat. ʒj, Spirit. Ether. Nitrosi mxx, and Aquæ Camph. ad ʒss, may be given four times daily. At bedtime the feet should be kept for several minutes in a foot-bath containing water as hot as can be borne, and when the patient is in bed a hot drink and ten or twelve grains of Dover's Powder should be given. The irritation and obstructive swelling can be temporarily relieved by spraying with a 1-per-cent. solution of hydrochloride of cocaine; this, however, must be used with caution. The relief (lasting half to three-quarters of an hour) obtained by the

cocaine-spray is most grateful to the patient, and frequently in the case of a busy man very valuable. In infants the obstructive swelling may cause urgent dyspnoea, laryngeal spasm, and inability to suck. These symptoms can generally be temporarily relieved by spraying the nostrils with a few drops of the cocaine-solution.

Acute Purulent Rhinitis presents the symptoms of acute catarrhal rhinitis, but the discharge is purulent and sometimes foetid. When met with in the infant a few days after birth it is probably of gonorrhoeal origin. In older children and adults it is found as a sequel to diphtheria, measles, or scarlet fever, and in general yields readily to a spray containing five grains each of sodium bicarbonate, borax, and common salt to the ounce of water. For extensions of the acute catarrhal process to the frontal and other sinuses, see Affections of the Accessory Cavities of the Nose.

2. Chronic Nasal and Naso-pharyngeal Catarrh.—Under this heading may be described (a) Simple Chronic Rhinitis and Naso-pharyngitis; (b) Chronic Hypertrophic Rhinitis and Naso-pharyngitis; and (c) Chronic Atrophic Rhinitis and Naso-pharyngitis.

The first two conditions cannot be rigidly differentiated, the second type being commonly, if not always, a more advanced stage of the first. They will therefore be in part described together.

(a) *Simple Chronic Rhinitis*.—**SYNON.** : Chronic Catarrhal Rhinitis; Chronic Coryza.—In this condition there is an increased irritability of the nasal mucosa, especially of that covering the middle and inferior turbinated bones. The sufferer catches cold very frequently, and complains of a stuffy feeling in the nose with obstruction, generally temporary and partial, of one or both nasal passages. This condition is in children commonly found in association with post-nasal adenoid vegetations. There is an increase in the amount of the secretion from the mucous membrane, which is at first watery but becomes, as the condition progresses, thick and viscid. The stuffiness in the nose is always worse at night, and the patient wakes in the morning with a dry mouth and pharynx due to mouth-breathing. If in this stage the nose be examined the mucosa is found but slightly altered. There is frequently, however, a mild degree of swelling and congestion, with, in some cases, commencing hypertrophy. There appears to be a loss of tone, of the arterioles, with consequent distension of the erectile tissue covering the two lower turbinated bones. The sense of smell is but little affected. Extension of this chronic catarrhal inflammation frequently involves the naso-pharynx, causing chronic post-nasal catarrh. In this condition the tenacious viscid mucus which is met with in the naso-pharynx is with difficulty removed by hawking. During the day this usually gives little trouble, but when the patient awakes dry-mouthed in the morning he is much worried by the continual efforts required to dislodge the mucus from the naso-pharynx. In America and South Africa, owing chiefly to the inhalation of dust, post-nasal catarrh assumes a far more severe type than in this country, and a much larger proportion of the population are affected.

TREATMENT.—See (b) Chronic Hypertrophic Rhinitis.

(b) *Chronic Hypertrophic Rhinitis*.—**SYNON.** : Chronic Hypertrophic Nasal Catarrh.—This condition is commonly a sequel of simple chronic catarrh,

and is frequently found in association with post-nasal vegetations. The mucosa of the nose and naso-pharynx is thickened, and the enlargement is chiefly found on the two lower turbinated bones, on the lower part of the septum nasi, and around the orifices of the Eustachian tubes. In addition to post-nasal vegetations other causes of chronic hypertrophic rhinitis are abnormalities in the nasal passages which cause obstruction to free nasal respiration or hinder the removal of the secretion, such as spurs or deviations of the septum, polypi and foreign bodies. In many cases, however, no real cause can be assigned. Though the condition may be met with at any period of life, by far the largest number of cases commence in childhood.

ANATOMICAL CHARACTERS.—There is generally redness of the nasal mucosa, which presents thickenings irregularly distributed, generally most marked at one or other end of an inferior turbinated bone or along its whole length. That this condition is a real overgrowth of the mucosa may be shown by the application of a solution of cocaine (5 per cent.) to the affected part. This only slightly reduces the swelling, whereas in cases of dilatation of the erectile tissue, as in simple chronic rhinitis, the application of cocaine causes a very remarkable shrinkage. The hypertrophy frequently affects the anterior segment of the inferior turbinal, and may give rise to a somewhat pedunculated growth of a reddish or purplish colour. This condition is most liable to obstruct nasal respiration when associated with a spur growing from the same side of the septum nasi. Hypertrophy of the posterior ends of the inferior turbinals is recognised by posterior rhinoscopy, or by digital examination. It may be seen as a smooth or, more frequently, as a lobulated ‘mulberry-like’ growth of greyish red or purplish hue projecting into the naso-pharynx, and sometimes all but completely obstructing it. This condition is to be sought for in all cases of impediment to nasal respiration where no post-nasal vegetations, enlarged tonsils or anterior nasal obstruction can be found. Hypertrophy of the middle turbinal is less frequent, and, where present, less marked, but it may touch the septum, and impede respiration through the middle meatus of the nose.

SYMPTOMS.—Obstruction to nasal respiration is constantly present, but is found in varying degrees. It is frequently scarcely noticeable during the daytime, but is troublesome at night when the throat and mouth become dry; and sleep may thus be disturbed. When the Eustachian tubes are involved, deafness, tinnitus, and otitis media may ensue from the resulting obstruction. The voice may become thick or nasal. Sneezing is frequent and sometimes violent, and in certain patients asthmatic seizures and other reflex symptoms—the so-called nasal neuroses—are observed.

TREATMENT.—Treatment will vary according to whether or no there is hypertrophy of the nasal mucous membrane. In strumous children cod-liver oil and tonics are indicated, but in the vast majority of cases the treatment is purely local. (1) When post-nasal vegetations, distinct nasal spurs, or any real impediment to nasal respiration exists, removal of the obstructive body is generally called for. (2) The thorough cleansing of the nasal passages is most essential, and must never be omitted. The nasal douche should never be used, owing to the liability of fluids to enter the tympanic cavity through the Eustachian tube and thus occasion

acute otitis media. A nasal spray should be employed, and the nasal passages should be sprayed twice or thrice daily with an alkaline mildly antiseptic solution, which should be warmed prior to use. An excellent spray-solution is the following: R. Sodii Bicarbonatis, gr. x; Boracis, gr. viij; 'Listerine,' ʒj; Aquæ, ʒj. This softens and washes away the mucus, and thus satisfactorily cleanses and purifies the mucous membrane. (3) In cases of marked hypertrophy of the mucosa operative interference must be undertaken. Where the hypertrophy assumes a polypoid or strawberry-like form it is best removed by a snare, either the cold snare, using piano-wire, or the electric cautery, employing a platinum-wire loop. In cases in which the hyperplasia is more general, the best results are obtained by the employment of the electric cautery. Two or three linear cauterisations may be made from behind forwards over the hypertrophied mucosa, parallel with the lower turbinal margin. Care must be taken not to cauterise the septum lest adhesion between the two raw surfaces occur, when the sloughs have been cast off. A bead of chromic acid is often effectual in destroying the hypertrophied mucosa, but is very inferior in every respect to the electric cautery. A solution of cocaine (5 to 10 per cent.) brushed on the mucous membrane renders cauterisation practically painless. A general anæsthetic is not required, and is inadvisable. After cauterisation the patient need not, as a rule, be confined to bed or even to the house. No after-treatment is required beyond the use of the above-mentioned nasal spray. Very rarely is it necessary or advisable to remove any part of the turbinated bones themselves. Should occasion demand it, the inferior turbinated bone may be removed under nitrous-oxide gas, by means of Carnalt Jones's turbinotome; or portions of the bone may be cut away with one of the forms of punch-forceps. In such cases the bleeding is always free, and sometimes very abundant. It is readily arrested by plugging with double-cyanide or iodoform gauze, which can generally be removed after a few minutes without causing any recurrence of the bleeding. If the electric cautery be thoroughly and skilfully used few cases will call for the removal of any portion of the turbinated bones.

3. Chronic Atrophic Rhinitis.—SYNON.: Chronic Fœtid Rhinitis; Ozæna (ὄζη, a stench); Fr. *Ozène*; Ger. *Stinknase*.

The term 'ozæna' has been applied to diverse ulcerative nasal conditions, in which fœtor is a prominent symptom; thus syphilitic ozæna, cancerous ozæna, &c., are sometimes described. The term 'chronic fœtid rhinitis' would perhaps best differentiate the condition described below. A dry atrophic condition of the nasal mucous membrane is sometimes seen without any accompanying fœtor.

PATHOLOGY.—The essential conditions encountered are atrophy of the nasal mucosa and of the subjacent bones, more particularly the turbinated bones, and a peculiar sickening fœtid odour. The cause of the atrophy of the mucous membrane, erectile tissue and underlying bones is unknown. The nasal secretion when it is first formed is entirely inodorous, and only acquires its disgusting stench some hours afterwards. The odour is due to the action of a specific micro-organism, the *Bacillus fœtidus ozænae* (Hajek), a cultivation of which outside the body reproduces the fœtor characteristic of ozæna. Ozæna commences as a rule in childhood

or youth, the large majority of cases giving a history of the trouble beginning between the tenth and twentieth years of life.

In cases in which atrophic rhinitis is found free from any fœtor it will generally be discovered on inquiry that the symptoms commenced much later in life (e.g. at, or after, the age of forty). Though delicate children seem to be more prone to become the victims of fœtid atrophic rhinitis, it is very frequently encountered in persons who are otherwise in perfect health. It is five times more frequent in the female sex than in the male. It is probably not contagious, and when, as is not infrequently the case, it occurs in two or three members of the same family, hereditary predisposition to the atrophic process may be assigned as the cause.

SYMPTOMS.—On examining the patient the characteristic fœtid odour is at once perceived. It is noteworthy, however, that the patient, though his sense of smell is usually not entirely lost, is quite unconscious of it. The nasal chambers, when examined by the aid of the speculum and rhinoscopic mirror, present a very roomy appearance; the mucosa and subjacent bones, particularly the inferior turbinated, are atrophied to a marked degree. Owing to the shrinkage of the mucous membrane, which has a dry and thin appearance, it is possible to see much farther into the nose than is normally the case, and frequently the openings of some of the accessory sinuses (e.g. the frontal) and even of the Eustachian tubes can be seen. The mucous membrane is in large part covered by crusts, which are very adherent, and result from the drying of the muco-purulent secretion of the mucosa. On removing a crust the subjacent mucous membrane may show light excoriations, but there is no ulceration. The nasopharynx has a similar dry appearance, and may contain crusts and muco-pus; and the atrophic process may affect the mucosa of the pharynx, and even, in rare cases, of the larynx.

DIAGNOSIS.—The diagnosis presents no difficulty: the association of the roominess of the nasal passages, the atrophy of mucous membrane and turbinated bones, the crusts and muco-pus, and the characteristic stench exhaled taken together are unmistakable.

TREATMENT.—Ozæna is still an incurable disease, though simple treatment robs it of its worst symptom. This consists in thoroughly cleansing the nose of the crusts and muco-pus. For this purpose the nasal spray should be employed at least twice daily (night and morning). Strong antiseptics applied to the nasal mucosa are inadvisable, and the best results will generally be obtained by the employment of a mild alkaline solution such as the following: R. Sodii bicarb. gr. viij, Boracis gr. viij, Sodii Chloridi gr. iv, Glycerini m. xxx, Aquæ ad ʒj. The important points are to use the spray-solution warm and liberally, so as to soften and remove the crusts. The vast majority of cases of ozæna require no other treatment. In very neglected cases it may be necessary to remove the crusts with a probe. Some patients derive benefit from the use of paroline used in an atomiser. Gottstein's method of treatment consists in preventing the formation of crusts by stimulating the mucous membrane to pour out an increased amount of watery secretion. With this object in view a plug of cotton-wool is introduced into the nostrils alternately and retained for some hours. The plugs may be soaked in glycerine prior to their introduction. The general condition

will in many cases require attention; thus tonics, good food, and cod-liver oil are useful adjuvants in cases in which anæmia or a scrofulous tendency is met with.

4. **Rhinorrhœa.**—By this name is described a somewhat rare condition, the chief symptom of which is a profuse discharge—generally pale, serous, and watery, but sometimes milky or of the colour of urine—of nervous origin and recurring with a certain regularity. The discharge may be bilateral or may come from one nostril only, and commonly occurs about once a day, lasting from a few minutes to some hours, and ceasing quite suddenly. It may be preceded by some symptoms of irritation in the nose and sneezing, but frequently none such are observed. The fluid comes from the nasal mucosa, which is commonly, in these cases, oedematous and hypertrophied. The treatment consists in cauterising the mucosa covering the middle and inferior turbinated bones, and in giving an astringent nasal spray, e.g. Alum (gr. iv) or Iron-alum (gr. ij) dissolved in an ounce of distilled water. This treatment, as a rule, only mitigates and does not cure this distressing condition, but frequently after months or even years the discharge ceases as suddenly as it commenced, and does not return.

Patients are sometimes seen who have sustained a fracture of the base of the skull in the region of the ethmoid bone, and who suffer, especially in certain positions of the head, from an escape of cerebro-spinal fluid. This condition must not be mistaken for that described above.

5. **Post-nasal Vegetations.**—**SYNON.** : Adenoid Vegetations.—Post-nasal Growths consist of an hypertrophy of 'Luschka's tonsil,' which is a mass of lymphoid tissue situated on the vault and on the posterior wall of the naso-pharynx, and is constantly present in young subjects. The pharyngeal tonsil, when examined in a young child, is found to be bounded on each side by the orifice of the Eustachian tube, and to present on its surface several vertical furrows, which partially subdivide it. This lymphoid structure, which is soft and bulky in the young child, tends to become partly absorbed and in part to undergo a fibrous transformation as adult life is approaching; hence adenoid growths in adults are always firmer and more fibrous than in the young child.

ÆTIOLOGY.—The condition may be observed in infants a few months old, but commonly comes under notice between the third and eighteenth years of life. Heredity certainly plays an important part in its causation, and frequently several members of a family are affected. The growths are frequently found along with hypertrophied tonsils, enlarged cervical glands, and thickening of the nasal mucous membrane, but are not uncommonly observed free from any such association.

SYMPTOMS.—Frequently there are no striking symptoms; and in general the patient is brought for treatment on account of some complication which has arisen, especially ear-ache or otorrhœa.

In infants with bulky adenoid vegetations sucking the nipple may be difficult or impossible. Mouth-breathing is the rule, especially during sleep; and snoring, with semi-suffocative attacks, is frequent. The open mouth, obliteration of the nasolabial fold, short upper lip, and drawing downwards of the inner canthi of the eyes give a vacant, semi-idiotic appearance to the face, which is very characteristic. The voice is modified, and there is

inability to pronounce properly *m*, *n*, *ng*, *th*, *z*, and *t*; *m* is generally, and *n* frequently, mispronounced *b*.

DIAGNOSIS.—There are two methods in general use: (1) Posterior rhinoscopy, which can be employed in less than half of all cases, as it is useless in children under 8 or 10 years of age, and in those who are unmanageable and fractious; and (2) Digital exploration, which gives entirely satisfactory information. The index-finger is passed rapidly into the mouth along the under surface of the soft palate and up into the naso-pharynx. This method of examination is instantaneous, not really painful, and by means of it the presence, size, consistence, and relations of the adenoid mass may be determined. The examining finger must be aseptic, and the examination conducted with the utmost gentleness. On the posterior pharyngeal wall are frequently to be observed in children soft oedematous gelatinous-looking granulations, which increase in size and number as they approach the naso-pharynx. These, when seen, always indicate the presence of post-nasal growths. They are hypertrophied patches of the lymphoid tissue of the part, histologically indistinguishable from 'adenoids' except that they lack their ciliated epithelial covering.

Post-nasal growths present themselves in two very distinct types: (1) In one the growths are soft and oedematous, easily lacerable, highly vascular, and prone to bleed. They hang down as spongy papillary masses, and give to the examining finger a sensation which has been aptly compared to that of touching a bunch of earthworms. (2) Much more frequently the mass presents the character of a distinct tumour. It is firm, fibrous, and smooth, except where it is vertically furrowed, and gives the impression of a tense elastic tumour, springing from the vault and the posterior wall of the pharynx.

PROGNOSIS AND COMPLICATIONS OF ADENOID VEGETATIONS.—Although these growths have a marked tendency to disappear at or shortly after the time of puberty they may yet exercise a most harmful influence. They are always a serious menace to the ears, since obstruction of the Eustachian tubes with consequent deafness and inflammation of the middle ear, purulent or non-purulent, are very prone to result. Catarrhal conditions of the nose, pharynx, larynx, and lungs are at all times liable to be induced by the presence of these growths; and various infectious diseases, chiefly scarlet fever, diphtheria, and measles, are far more dangerous and more likely to be attended by grave complications in children who suffer from post-nasal vegetations. Moreover, these growths undoubtedly produce deformity of the chest, and arrest not only the physical development of the child, but also its mental progress—probably in large part owing to the deafness they occasion. It is beyond question that 99 out of every 100 children from whom adenoid vegetations have been removed improve markedly in every way within a few weeks after the operation.

TREATMENT.—Palliative treatment is of little value. The nasal spray may be used twice or thrice daily to clear away the muco-pus which is commonly present, and thus by reducing any catarrhal inflammation to slightly increase the airway in the naso-pharynx. The only reliable treatment is removal of the growths, and this is essential in all cases in which any aural or respiratory com-

plications (e.g. deafness, pain in the ear, recurring attacks of bronchitis) exist.

An anæsthetic is, in this country, almost always administered. In infants it is perhaps best dispensed with. Chloroform should, in the writer's opinion, never be given, most of the few deaths having occurred in cases in which this anæsthetic was administered. It must be borne in mind that anæsthesia introduces an element of danger into an operation otherwise devoid of risk. For the removal of adenoids alone nitrous-oxide gas with or without admixture with oxygen suffices for a fairly rapid operator, and is the safest of all anæsthetics. If, as frequently happens, hypertrophied tonsils demand removal at the same time, the A.C.E. mixture answers admirably. The best position is with the patient lying on the right side with the head supported by the anæsthetist or assistant beyond the end of the table, and kept about an inch lower than the rest of the body, the face being turned slightly downwards. In this position blood runs freely out of the mouth and nose and cannot enter the larynx. The removal of the growths may be accomplished by means of cutting forceps, of which Löwenberg's forceps, or one of the modifications of this instrument, may be used, but most operators prefer Gottstein's curette. For softish growths this instrument leaves nothing to be desired; for a firm adenoid tumour in an older patient it is often necessary to punch several pieces out of the mass before curetting. Scrupulous care must be taken to ensure all instruments used being sterile. Hæmorrhage is frequently alarming for a few seconds, but rapidly stops spontaneously. Fatal hæmorrhage is almost unknown. Most patients require no after treatment, except perhaps a laxative on the second or third day after the operation. In a small proportion of cases the operation may be followed by some febrile disturbance with swelling of the cervical glands, acute otitis media, or acute tonsillitis, but these accidents can generally be prevented by sterilising the instruments employed. Recurrence to a slight extent is frequent, but, except in very young children under three or four years of age, the surgeon can as a rule promise confidently that no recurrence sufficient to call for a second operation will take place.

The results of the operation are, as above indicated, almost invariably excellent, the deafness and otorrhœa, if present prior to the removal of the growths, rapidly disappear, speech and nasal respiration become normal, and the physical and mental development of the patient improves to a remarkable extent.

6. **Syphilis** may attack the nose in almost any part, and may affect bone, cartilage, skin, or mucous membrane, or any combination of these. The condition may be due to hereditary syphilis or to the acquired form in any of its stages.

Hereditary Syphilis attacks primarily the mucous membrane of the nose. It is met with in infants a few days or weeks old in the form of 'snuffles,' a catarrhal inflammation of the nasal mucosa, the discharge from which soon becomes muco-purulent or purulent. The swelling of the nasal mucosa with formation of crusts obstructs nasal respiration, especially in suckling and during sleep. If not efficiently treated the inflammatory process may spread to the bones and seriously deform the nose. This accident is, however, far more apt to occur in those cases in which the nose is attacked about the

period of puberty. In this delayed hereditary form of nasal syphilis the commencement of the trouble is also a rhinitis with muco-purulent or purulent discharge and much obstruction to nasal respiration. After some days or weeks the discharge (in untreated cases) becomes blood-stained and foul (so-called syphilitic ozæna), and the bones of the nasal chambers become involved in an osteo-periostitis, which leads to necrosis, which may at times be most extensive. The nasal bones, the vomer, inferior turbinals, and palatal processes of the maxillæ are those most frequently affected; and when the two first-mentioned suffer the nose is apt to be characteristically sunken and deformed.

Acquired Syphilis.—*Primary chancres* of skin or mucous membrane are exceedingly rare if we except those situated around the orifice of the Eustachian tube, due to catheterisation with infected instruments. The diagnosis of these chancres of mucous membrane is rarely made with certainty prior to the appearance of secondary symptoms.

In the *secondary* stage mucous patches (*plaques muqueuses*) are met with on the nasal mucous membrane almost always in association with others in the mouth, fauces, tongue, vulva, or anus. They appear as little superficial erosions which may be hidden by overlying scabs; and not infrequently they form a ring around the margin of the nostril and may block the orifice. The process does not extend deeply and never involves the periosteum or bones of the cavities of the nose. The diagnosis is confirmed by the discovery of other syphilitic phenomena.

Tertiary syphilitic lesions of the nose are common and destructive, and often leave behind them serious deformity. They are chiefly observed four years or more after the appearance of the chancre, attack primarily the bones, and, as a rule, involve the mucous membrane subsequently. The bony lesions are in general a combination of necrosis and caries. The bones most frequently attacked are the vomer, the nasal bones, and the upper surface of the palatal processes of the maxillæ. Gummatous destruction of the septal cartilage is likewise very common. The symptoms are at first indefinite. Soon, however, complaint is made of stuffiness in the nose, and of blood-stained fetid purulent discharge, the odour of which is (unlike that in true ozæna) disagreeably manifest to the patient. Frequently small particles of dead bone can be found in the nasal discharge. After cleansing the nose with a spray, an examination reveals the presence of crusts partly covering ulcerated surfaces of mucous membrane, sloughs of greyish or purplish hue, and frequently of bare sequestra of bone, generally much darkened or even black in colour. The above represents a severe and advanced stage of the disease. In an earlier stage, before necrosis has occurred, the gummatous infiltration may be absorbed by appropriate treatment and leave little or no trace of its presence. Should the ethmoid bone be attacked, as rarely happens, the condition is most grave, as septic meningitis is apt to develop. In general after the casting off of several small sequestra, a tendency towards healing, even in untreated cases, is apt to manifest itself; in rare instances, however, particularly in debilitated drunkards, the destructive process may advance with terrible rapidity and involve several of the bones of the face, giving rise to a huge chasm with hideous deformity. Frequently the deformity is increased owing to multiple

gummatous formation and ulceration in the skin covering the nose and cheeks.

TREATMENT.—Primary and secondary lesions in acquired syphilis and snuffles and other early hereditary symptoms require a prolonged mercurial course. In cases in which there is abundant formation of crusts and in which the erosions are spreading, the former may be removed after softening them with an ointment composed of Unguent. hydrarg. nitratis, 15 grains, or of Hydrarg. oxidi flav. 10 grains, to the ounce of vaseline, and the raw surface of the erosion touched with a 2-per-cent. solution of chromic acid in distilled water, or with a solution of perchloride of mercury (1 to 1,000).

In tertiary nasal syphilis, and in late hereditary manifestations, the best results are obtained by the combined administration of mercury and iodide of potassium; the latter drug must in most cases be given in full doses—15 grains to 30 grains thrice daily to adult patients. The other indications are to wash away with a mild antiseptic spray the blood-stained purulent discharge and to remove any loose sequestra of bone. In addition, particularly in late hereditary manifestations, it is frequently wise to administer tonics and cod-liver oil.

7. Tubercular Disease.—Tuberculosis attacks the nose in two very different forms, (a) Lupus, (b) Tubercular disease of mucous membrane. These two conditions, though both caused by the tubercle-bacillus, demand separate description.

(a) *Lupus.*—Lupus affecting the external aspect of the nose is described in the articles LUPUS ERYTHEMATOSUS; and SKIN, Tuberculosis of. Lupus of the nasal mucosa is generally an extension of the disease from the skin, and is rarely a primary condition. It is chiefly met with in young subjects and occurs chiefly in the region of the cartilaginous septum, inferior turbinates, and anterior part of the floor of the nose. It may not infrequently be seen on the upper surface of the soft palate and in the naso-pharynx. The symptoms are at the beginning quite slight, complaint being generally made of stuffiness in the nose and scab-formation. On examination several tiny nodules varying in size from a millet-seed to a hemp-seed are observed; they are generally discrete, superficially ulcerated, and in part covered by crusts. Frequently perforation of the septum is found, the margins of the orifice being surrounded by flabby granulations. The diagnosis is rendered easy by the almost invariable co-existence of lupus of the skin. The progress of the condition is essentially chronic, lasting several years. The bones of the nose are never attacked, necrosis being therefore unknown. This is an important point in the differential diagnosis of lupus and syphilis, though it must be remembered that the two conditions may be found together. The treatment of lupus of the nasal mucosa must be thorough. General treatment (cod-liver oil, iron, tonics, &c.) is necessary, but it is upon local treatment that the chief dependence must be placed. All the diseased tissue must be freely destroyed, preferably by curetting with a small sharp spoon and then applying either a bead of chromic acid or lactic acid (50-per-cent. solution or undiluted). Any small recurrences, which are very apt to form, must be treated in a similar way. The electric cautery is frequently used, but the writer employs it only after a thorough preliminary curetting. The results of this treatment are frequently excellent.

(b) *Tubercular Disease of the Nose* is distinctly rarer than lupus. Usually it is secondary to tuberculosis of the lungs or larynx. Only very rarely is it primary, in which case it generally assumes the form of an irregular, nodular, pedunculated tumour, which is friable and bleeds on the slightest contact, and grows from the septum nasi in a person otherwise in good health. Soon this mass begins to ulcerate, becomes partly hidden by scab-formation, and eats a hole in the septal cartilage. Glandular enlargement in the neck and submaxillary region then follows. Tubercular ulceration, secondary to disease of lungs or larynx, is much more common. It is found generally in the last stages of phthisis, and may attack any part of the nasal mucosa, though most commonly its site is on the septum and floor of the nose. The ulcers may be single or multiple, and vary from the size of a split-pea to that of a florin. Their margins are irregular, excavated, and surrounded by a ring of little tubercles in various stages of formation or of ulceration. Their bases are covered with muco-purulent secretion or sloughs, and present a yellowish-grey or reddish-grey colour. The progress of such ulcers is slow, and perforation of bone or cartilage is common.

DIAGNOSIS.—In the secondary (ulcerative) type no difficulty is encountered owing to the co-existence of phthisis (pulmonary or laryngeal). In tubercular tumour (the primary form) the recognition of the case is frequently extremely difficult. Syphilitic disease, sarcoma, and epithelioma have to be excluded—the first of these by the failure of anti-syphilitic treatment. The diagnosis may in certain cases be made by the microscopical examination of a portion of the growth, or by finding tubercle-bacilli; but where the diagnosis rests between tuberculosis and malignant disease it is wise in general to remove the growth first and ascertain its exact nature afterwards.

TREATMENT.—(1) *Of Tubercular Tumour.*—This consists in encircling the pedicle with the galvanic-caustic snare, and in thoroughly curetting its base. After curetting lactic acid (50-per-cent. solution) must be well rubbed into the raw surface, or iodoform dusted over it.

(2) *Of the Secondary Ulcerative Form.*—Here the treatment can be only palliative, especially when the ulcers are multiple and the phthisis advanced. Mild antiseptic nasal sprays with insufflation of iodoform alone, or mixed with boric acid (1 to 4), may be used. If the ulcer is solitary and the general condition fair, lactic acid may be applied (50 per cent.), after curetting the ulcer. Healing of these secondary tubercular ulcers, however, the writer has never seen.

8. Affections of the Septum Nasi.—The chief affections of the nasal septum are: (1) Deviations and Spurs; (2) Hæmatoma; (3) Abscess; and (4) Perforating Ulcer.

(1) *Deviations and Spurs.*—A perfectly vertical nasal septum is exceptional in the adult though the normal condition in the young child. The cartilaginous portion is commonly the part most deflected, the hinder bony margin of the septum being in general quite vertical. The septum may present a general convexity on one side and a corresponding concavity on the other side; or the deviation may be of the sigmoid type, in which the deflection is towards one side below and anteriorly, and towards the opposite side above and posteriorly.

Spurs of the septum are of two kinds—linear

ridges (crests), and the much rarer conical spines. Both varieties are usually found in or about the line of junction of the vomer with the septal cartilage. They are frequently observed along with deviation of the septum nasi, and consist chiefly of cartilage, though some bony substance is often present in their interior. The symptoms to which they give rise are dependent in large measure upon the degree of obstruction to nasal respiration which they occasion. Slight deviations and spurs produce no symptoms, and call for no treatment. Where, however, they are of large size they tend to produce obstruction, and chronic nasal catarrh with associated affections of the Eustachian tube and middle ear. Sometimes a conical spine presses against the anterior portion of an enlarged inferior turbinal bone and causes ulceration, hæmorrhage, or even suppuration.

TREATMENT.—Cauterisation of the inferior turbinal body with the galvano-cautery, by causing shrinkage of its hypertrophied mucosa, will, in many instances, be found effectual in relieving the symptoms, and should in the majority of cases be given a fair trial prior to resorting to more severe measures. When, however, it is necessary to remove the projecting part of the septum (summit of convexity) or a crest or spine, the mucous membrane covering the prominence is to be incised in a direction from behind forwards, and then the projection sawn off by means of a Bosworth's nasal saw. When the deviation is of the sigmoid type the lower convexity is alone to be attacked, the upper one being as a rule out of reach and not much of an obstacle to nasal respiration.

(2) *Hæmatoma of the Septum.*—Owing to the fact that the mucous membrane covering the nasal septum is not firmly adherent to the cartilage and bones, it is very readily detached from these structures by an effusion of blood. In certain cases a unilateral hæmatoma may be found as the result of some injury associated with sudden twisting of the septal cartilage. More frequently we have to deal with a bilateral hæmatoma, which is always the result of a fracture of the cartilage or one of the bones of the septum. A hæmatoma presents itself as a purplish red swelling on one or both sides of the cartilaginous septum, just inside the nostrils, with a broad base and a distinctly fluctuating elastic feel. In bilateral cases pressure on the one side causes collapse on that side, and an increase in the prominence on the opposite side of the septum. As a rule no treatment is required beyond attending to the fracture (if one be present) of the septum. Should, however, the swelling be of great size, and suppuration threaten, an incision may be made on each side through the mucous membrane; the blood, clot, or blood-stained serum evacuated, and the pockets washed out with an antiseptic solution, e.g. biniodide of mercury, 1 to 2,000. This small operation should be preceded by antiseptic irrigation of the nose, and followed by plugging the nostrils with strips of double-cyanide or iodoform gauze.

(3) *Abscess of the Septum.*—Acute abscess is commonly the result of the suppuration of a hæmatoma. A more serious variety is that of lymphatic origin secondary to a boil in the region of the columella nasi. The former is generally bilateral, the latter unilateral. The symptoms are those of nasal obstruction, and fever with redness and swelling of the nose.

Chronic abscess may be due to necrosis of bone or cartilage the result of traumatism, syphilis, or

tuberculosis; or may be found as a complication of small-pox, typhoid, or other specific fever.

TREATMENT.—An acute abscess requires incision and antiseptic irrigation. A chronic abscess demands in addition curetting and removal of necrotic portions of bone or cartilage. It is apt to leave behind a perforation of the septum.

(4) *Perforating Ulcer of the Septum.*—Apart from perforation of the septum due to syphilis or tuberculosis, there occurs also a slowly progressive circular ulcer situated just within the nostril first described by Jonathan Hutchinson, and known as 'perforating ulcer of the septum.' The process starts in the mucous membrane covering the septal cartilage; a grey sloughy false membrane appears and is cast off, and the denuded cartilage is slowly eroded. The ulcer usually perforates the septum, but rarely it stops short and cicatrises. When the perforation is complete and the margins of the hole have healed, the opening is so wonderfully regular and clean cut that it has been thought to be of congenital origin. Of the true pathology of perforating ulcer little is known. Workers in chromic acid are peculiarly liable to suffer from it. Symptoms may be absent, and the patient may be unaware of the fact that his septum is diseased or perforated. Generally there is a feeling of irritation which leads to scratching and picking the surface of the ulcer. Epistaxis is not infrequent. When the ulcer has perforated, healing of the margins of the perforation generally occurs, but the aperture in the septum does not close.

Treatment consists in spraying the nose with a warm solution of bicarbonate of sodium and borax (of each 3j to water ʒviij) to remove the crusts, and in cauterising the ulcer (after applying a 10-per-cent. solution of cocaine) with the acid nitrate of mercury or the galvano-cautery. Thereafter an ointment consisting of Hydrarg. oxid. flav. or iodoform, ʒss to the ounce of lanoline, may be employed.

9. **Foreign Bodies in the Nose** are very frequent in children, in hysterical women, and in the insane. Among those which are most common may be mentioned peas, beans, glass-beads, pieces of slate-pencil, cherry- and plum-stones; these are almost always introduced by the patient through the anterior nares. A foreign body (e.g. a particle of food) may also be forced into the nose through the posterior nares in the act of vomiting. Sometimes a detached and loose sequestrum plays the part of a foreign body. Foreign bodies introduced by the anterior nares are commonly found situated on the floor of the nose or under cover of the inferior turbinal, while those vomited into the nose are most frequently seen in the middle meatus. The presence of a foreign body generally produces after an interval of time an offensive muco-purulent or purulent discharge commonly tinged with blood; and symptoms of nasal obstruction are manifested. Much depends on the nature of the foreign body: thus while a pea or bean swells very soon by imbibition of fluid, and may even sprout, a glass bead will produce symptoms much more slowly. The discharge is intensely foetid and irritant, causing frequently an eczematous condition of the skin of the upper lip and around the aperture of the nostril. The discharge is necessarily unilateral; and in every case of unilateral nasal discharge occurring in a child, especially if it be foetid, the presence of a foreign body should be suspected.

The treatment consists in spraying the nose with

a 1-per-cent. solution of cocaine, and removing the foreign body. Forceps should not as a rule be used to seize the object, as they are apt to miss their hold and to shoot the foreign body further into the nose. A small hook, or a probe of which the last one-third of an inch is bent to a right angle with the rest of the instrument, is to be insinuated behind the foreign body, which may then be easily withdrawn. Its removal suffices for the cure of the symptoms, but a mild antiseptic nasal spray may be given to cleanse the nose.

Rhinoliths.—Calculus concretions are occasionally found in the nose. They invariably originate round a foreign body or a plug of inspissated mucus. They consist largely of phosphate and carbonate of lime, and give rise to the same symptoms as a foreign body. The resulting purulent rhinitis tends to further deposition of calcareous matter, and thus the rhinolith increases in size. The treatment of rhinoliths is removal. In cases of small nasal stones this is easy enough, but large rhinoliths may require to be crushed *in situ*; or it may be necessary to detach the ala of the nose to facilitate their removal. In other cases they may be pushed back into the pharynx and thus removed. Care should, however, be taken in such cases, especially when an anæsthetic is administered, that the foreign body is not allowed to fall into the larynx. See p. 335.

10. Parasites in the Nose.—In this country they are exceedingly rare, but in hot climates various species of flies, attracted sometimes by the odour of decomposition in cases of *ozæna*, gain entrance to the nose and there lay their eggs. The warmth and moisture ensure that these are hatched, and the maggots swarm all over the nose and accessory cavities, and may destroy the mucosa and even cause necrosis of the bones, and frequently death. Earwigs, leeches, the *Oxyuris vermicularis*, and the *Ascaris lumbricoides* are occasionally found in the nasal passages, and also the larvæ of the common bluebottle. The treatment consists in irrigation of the nose with antiseptic solutions or the introduction of chloroform-vapour into the nose. Both these methods have for their object the destruction of the maggots, but it is obviously a matter of extreme difficulty to ensure that all these are destroyed in the accessory sinuses of the nose.

11. Rhinoscleroma.—This condition, first described by Hebra in 1870, is practically unknown in Great Britain. It is chiefly met with in the eastern districts of Austria, in Southern Russia and in Central America. It is to be grouped with the infective granulomata, and is characterised by the formation of exceedingly hard nodular thickenings in the skin and mucous membrane of the upper respiratory passages. In more than 90 per cent. of all cases the nose is attacked. The nodules are either discrete or diffuse. They are painless, and vary in colour from that of the normal skin to a dusky red hue. Their surface is smooth and shining, and they tend to invade the tissues in their vicinity both superficially and deeply. The condition, which is quite incurable, lasts for years, and other parts besides the nose become infected, chiefly the palate, pharynx, upper lip, maxilla, and larynx. Ulceration is very rarely seen. The nodules on histological examination show chiefly large round cells (much like those of a round-celled sarcoma), and in them is always to be found a short, thick bacillus (*Bacillus* of Frisch) which is undoubtedly

the cause of the disease. Retrogressive changes occur in the nodules, resulting in the formation of cicatricial connective tissue. Thus the infiltrated parts become markedly contracted, and stenoses of nose, pharynx, and larynx are produced. The cartilaginous induration and brawny thickening of nose and upper lip, and the nasal and frequently laryngeal stenosis, along with the entire absence of ulceration, distinguish rhinoscleroma at once from lupus, malignant disease, and syphilis. Prognosis is hopeless, a cure being unknown. Treatment is seldom of any service except for laryngeal stenosis, when tracheotomy will prolong life. Interstitial injections of antiseptic solutions, the rubbing into the affected parts of a 1-per-cent. solution of corrosive sublimate in lanolin (Doutrelepoint), the thermocautery or the sharp spoon have all been advocated.

12. Nasal Diphtheria.—Diphtheria affecting the nose is described in the article on DIPHTHERIA. In this place it will suffice to draw attention to the fact that a sanious diphtheritic discharge from the nasal cavity or a diphtheritic membrane on its mucous surface may exist without any severe constitutional symptoms.

13. Tumours of the Nose.—The commonest tumours of the nasal chambers are myxomata and fibro-myxomata, which almost always tend to assume a polypoid shape. Less frequently there occur angiomata, adenomata, osteomata, enchondromata, and malignant neoplasms, epitheliomata and sarcomata.

Myxomata or *Mucous polypi* are very soft, gelatinous and almost translucent in appearance. Their colour is whitish-grey, with a faint blue or pink tint. They are, when of any size, generally pyriform and pedunculated, more rarely sessile. They vary in size from that of a hemp-seed to that of a plum, and are commonly multiple. Their most frequent site of attachment is the lower border of the middle turbinated bone; less frequently they grow from the superior turbinated bone and superior meatus. Very rarely they spring from the septum and inferior turbinated body, and never from the floor of the nose. They may arise in the interior of the accessory sinuses, and may grow backwards into the naso-pharynx, where they may attain large dimensions. The cause of mucous polypi is most uncertain, though it is probable that affections of the accessory sinuses, around the orifices of which they are most commonly situated, play a part in their production. The tumours consist of gelatinous myxomatous tissue containing a varying amount of fibrous stroma, with few vessels and no nerves. They are commonly covered externally with ciliated epithelium.

SYMPTOMS.—At first these are slight, complaint being made of nasal obstruction, and a feeling of fulness in the head, aggravated in damp weather, as the polypi are very hygroscopic. There is an excessive secretion from the nostrils, which is at first mucous but becomes later muco-purulent or even purulent; epistaxis is not uncommon. Reflex disturbances are prone to occur, the commonest being asthma, hemicrania, neuralgia and cough. When the polypi completely block the nose the latter becomes broadened and total loss of smell with dryness and irritation of the pharynx result.

TREATMENT.—Removal of the polypi is the only efficient treatment, and this is to be followed by cauterisation or curetting of their divided pedicles in order to prevent recurrence. Avulsion with

polypus-forceps is a rude and obsolete method. Either the cold snare or the galvano-caustic loop may be used. In all cases the instrument must be guided by the sense of sight, and a solution of cocaine must be previously employed, preferably in the form of a spray. Cocaine has the triple advantage of deadening pain, causing shrinkage of the neighbouring hypertrophic and hyperæmic mucosa, and lessening the hæmorrhage. The cold wire-snare is the best instrument to use in the case of large polypi. Its employment must be followed by the curettage or the destruction of the base of attachment by the application of the galvano-cautery or of chromic acid. For smaller and very numerous polypi the galvano-caustic snare answers best. It has the advantage that it destroys the pedicles of the polypi, but adhesion between the turbinal body and the septum nasi is apt to follow its use. In bad cases several sittings will be required before every particle of myxomatous tissue can be destroyed. The after-treatment consists in the employment of a mild alkaline nasal spray.

Angiomata of the nose are very rare tumours, of a bluish-red or violet colour, which grow from the septum nasi or upper (olfactory) portion of the nose. They produce nasal obstruction and severe epistaxis. They have a broad base of attachment and an irregularly mammillated appearance, and frequently pulsate. They must not be confounded with mucous polypi. Such a mistake might give rise to profuse hæmorrhage after removal. The treatment is destruction with the galvano-cautery or removal by means of the galvano-caustic snare. If the latter be used it is to be employed at a dull-red heat, and constriction is to be very gradually applied.

Adenomata.—Rarely tumours composed of glandular (mucous gland) tissue are found in the nose. They are of firmer consistence than the mucous polypi, and merge into the surrounding hypertrophic mucosa. The galvano-cautery or galvano-caustic snare must be employed for their destruction or removal.

Chondromata are rare apart from cartilaginous outgrowths or spurs on the inferior and anterior part of the nasal septum (echondromata). These latter are described under affections of the septum nasi. True cartilaginous tumours (enchondromata) may grow from the septum or roof of the nasal passages in children and young adults. They obstruct nasal respiration and cause a muco-purulent blood-stained discharge. If operable at all they demand a formidable operation for their removal, and recurrence of the growth is very frequent.

Osteomata.—Apart from exostoses forming spurs and crests of the septum nasi, which have been already described, tumours composed of bony tissue—sometimes of cancellated but more frequently of ivory compact bone—are met with in the nasal passages and accessory sinuses; they are, however, uncommon. In shape they are generally round or oval, and they sometimes perforate the walls of the bony cavity in which they originate. Until they attain a considerable size they give rise to no symptoms. In their growth, however, by pressure upon neighbouring structures they cause severe neuralgic pains, a feeling of nasal obstruction, ulceration of the mucous membrane that covers them, and thus a hæmorrhagic or purulent discharge which may be foetid. Removal of these tumours is the only effectual treatment. The rarer cancellous forms are

readily removed by means of forceps or a fine saw. The ivory osteomata, particularly when situated in the upper accessory cavities, ethmoidal, frontal or sphenoidal, can, in many cases, only be removed by operations which open or injure the dura mater, and are therefore scarcely justifiable. When growing from the lower two-thirds of the nasal passages or from the antrum of Highmore their removal can be in general safely accomplished.

Papillomata.—True papillomata of the nose are of infrequent occurrence, and must not be confounded with papillary hypertrophy of the mucous membrane covering the inferior turbinal body, which is a far more common condition. Their usual seat of origin is the cartilaginous part of the nasal septum or the floor of the nasal fossa. They are to be snared with the galvano-caustic loop or destroyed by the galvano-cautery.

Malignant Tumours.—Primary malignant tumours of the nasal passages are fortunately rare. The varieties which are met with are (1) epitheliomata and (2) sarcomata. Malignant neoplasms originating in neighbouring parts and involving the nose by extension are much more common.

Epitheliomata are rare as primary tumours, and may be composed of squamous or columnar epithelium. They originate either in the mucous membrane covering the septum or the turbinal bodies, and the course of such growths is at the commencement slow, then after a time it becomes very rapid. The destructive process eats away nose, septum and palate, and gives rise to a horrible chasm in the centre of the face. Rapidly developing cachexia and death supervene.

Sarcomata generally commence in the septum as chondro-sarcomata or fibro-sarcomata. The former variety is sometimes seen in young children, but sarcomata are more frequent in the aged. Malignant tumours of the nose cause frequent epistaxis, foul blood-stained discharge, loss of smell, and rapid wasting. The prognosis is very bad, their removal is always difficult, and in the upper parts of the nasal passages often impossible, owing to infiltration of the cranial bones. Even after apparently free removal recurrence is the rule. In many cases the cautery may be used for their destruction or removal in preference to any severe operation on the bones of the face.

Tumours of the Naso-Pharynx.—(1) *Naso-pharyngeal Fibrous Polypi*.—These tumours are usually composed of almost pure fibrous tissue, sometimes mixed with myxomatous or cellular elements. They are generally covered by an unbroken mucous membrane, and spring by a broad base from the periosteum covering the base of the skull, particularly from that of the basilar process of the occipital bone and the body of the sphenoid. These fibrous polypi of the naso-pharynx are dense, very hard, often lobulated tumours, of great vascularity, and frequently of great size; they are met with most frequently in young male adults. If not removed the mass displaces the bones in the neighbourhood, which may become eroded, and sends large processes into adjacent cavities—e.g. cranium, orbit, and zygomatic fossa. These tumours have a distinct tendency to degenerate into sarcomata.

TREATMENT.—Removal of the tumour is urgently called for, but in large growths the ablation of the mass is a very serious operation, owing to the severe hæmorrhage generally encountered, and the difficulty of gaining access to the pedicle.

Smaller growths may be removed by means of the galvano-caustic snare used very deliberately. Larger growths require either as a preliminary to their removal splitting of the soft palate, which gives generally insufficient access to the growth, or Langenbeck's osteo-plastic resection of both upper jaws.

(2) *Fibro-myxomatous* or purely *Myxomatous Polypi* are occasionally found in the naso-pharynx. These growths have their origin in the nasal passages, and spring from the hinder extremities of the turbinal bodies, or from the septum. They fall backwards into the naso-pharynx, and are therefore sometimes erroneously considered to be of naso-pharyngeal origin. They are readily removed by the galvano-caustic loop.

14. **Affections of the Accessory Nasal Sinuses.**—The maxillary (Antrum of Highmore), ethmoidal, frontal and sphenoidal sinuses are subject to morbid changes, sometimes arising primarily, but far more frequently secondarily, to affections of the nasal fosse.

(a) *Frontal Sinus.*—Injuries to this sinus are frequent as the result of direct violence. Fracture of the outer wall of the sinus is apt to be accompanied by emphysema of the neighbouring soft parts. Compound fractures, which involve the inner wall of the sinus and lay bare or even lacerate the dura mater, are, of course, formidable injuries. Not infrequently they are complicated, and caused by the presence of a foreign body. Operative interference is, of course, demanded to remove the foreign body, and to purify the interior of the sinus. Not very infrequently, particularly in tropical countries, insects gain access to the interior of the nose, and lay their eggs in the frontal sinus; when the young are hatched they are apt to give rise to violent inflammation and suppuration in this cavity.

Inflammation of the frontal sinus is either acute or chronic, and most commonly results from extension of inflammation from the mucous membrane of the nose (in chronic cases this is generally syphilitic or tubercular in origin) from the bone or periosteum. The symptoms of acute inflammation of the frontal sinus are pain—often unilateral and generally radiating upwards and outwards, along the inner two-thirds of the eyebrow—headache, and a feeling of fulness and distension in the frontal bone with swelling and tenderness to the touch. When suppuration has occurred there is dusky discoloration of the skin overlying the cavity.

Suppuration (empyema) of the frontal sinus may be a sequel of acute inflammation, or may result from long-standing distension of the sinus with mucus, when the outlet into the middle meatus of the nose is obstructed. In chronic cases of empyema of the frontal sinus changes are apt to occur in the bony walls of the cavity, the floor of which often bulges into the orbit, displacing the eyeball. In extreme cases the bony wall becomes perforated, and the pus escapes into the orbital or cranial cavities, or into the nose.

TREATMENT.—In the early stages local blood-letting (e.g. leeching) will be beneficial; and when, as is usually the case, the inflammation of the frontal sinus is secondary to some nasal condition, treatment directed to this latter must be at the same time adopted. When pus-formation is demonstrated by the marked increase in the severity of the symptoms evacuation of the purulent contents of the sinus

must be at once undertaken, owing to the risk of cerebral or orbital complications. Evacuation of the pus by operating through the nose by way of the infundibulum is unsatisfactory and dangerous. In all cases it is best to operate from the forehead, and this can be done with a minimum of disfigurement by making the incision in the line of the eyebrow, which has been previously shaved. The sinus is then perforated by a small trephine, and a small drainage-tube passed through the infundibulum into the nose. Through this tube the cavity may be washed out.

Tumours are not infrequently found in the frontal sinus. Those most commonly met with are osteomata, fibromata and myxomata, among innocent tumours, and sarcomata and carcinomata. They call for removal by external incision, and in the case of malignant neoplasm a large amount of the frontal bone may require to be ablated.

(b) *Ethmoidal Sinuses.*—Affections of these sinuses are much less frequent than is the case with the frontal sinus. Still empyema, myxomatous tumours, and caries or necrosis which may give rise to septic meningitis, may be met with.

(c) *Sphenoidal Sinus.*—The affections of this cavity are very similar to those of the ethmoidal sinuses, and the symptoms in the two cases are practically identical. On account of the proximity of the sinus to the great vessels (internal carotid artery and cavernous sinus) caries and necrosis of the bony walls of the cavity are liable to give rise to fatal hemorrhage and septic sinus-thrombosis.

(d) *Maxillary Sinus or Antrum of Highmore.*—The commonest affection of the maxillary sinus is undoubtedly empyema, which may be the result of extension of inflammation from the nasal cavity or from the roots of one of the upper teeth, the fangs of which not uncommonly perforate the bony floor of the antrum.

The symptoms of antral suppuration are tenderness on pressure over the cavity and facial neuralgia; sometimes oedema of the overlying soft tissues; and in chronic cases with occlusion of the orifice of the antrum distension of the cavity, leading it may be to egg-shell cracking when pressure is made over the bone. A sign, that is when present diagnostic, is the sudden discharge of a considerable amount of pus from the nose when the head is held in a certain position, generally with the opposite side of the face resting on a pillow. Examination of the nose shows that the pus proceeds from under cover of the middle turbinated bone. It must be borne in mind, however, that this symptom is absent in the not infrequent cases in which the orifice of the antrum is obstructed. The purulent discharge from the antral cavity is in general fetid. Transillumination of the antrum in a dark room by means of an electric lamp held in the mouth with the lips tightly closed is a valuable guide to the diagnosis of antral empyema, the dark shadow on the affected side being in marked contrast with the translucent brightness on the normal side of the face. This is best seen in the region of the roof of the antrum, i.e. the floor of the orbit. Cystic degeneration of the antrum, neoplasms and marked thickening of the lining of the cavity may, however, cause the same opacity to transillumination.

When inflammation of the mucous lining of the antrum comes under observation before pus-formation has occurred, hot fomentations, local blood-letting and appropriate treatment of any causal

nasal affection will be employed. As soon as pus has formed, particularly in cases in which the antral orifice is blocked, it must be removed. The antrum may be perforated (1) through the socket of a diseased upper molar or bicuspid tooth, (2) through the outer wall of the cavity, or (3) through the inferior meatus of the nose by directing the trocar outwards through the inner (nasal) wall of the antrum. A drainage-tube is placed in the cavity, which is to be irrigated frequently; and if it be found blocked with polypi or granulation-tissue time will be saved by curetting such away. Drainage must be efficiently maintained until all purulent discharge has ceased. In cases of pus-formation in the antrum in which the antral orifice remains patent careful irrigation by means of a nozzle introduced into the antrum through the nose may suffice to cure the affection. The removal of the anterior segment of the middle turbinal bone renders the introduction of the irrigating nozzle much more easy.

Tumours of the antrum are not infrequent: 70 per cent. are malignant, sarcomata and carcinomata being nearly equally common. Osteomata, myxomata, fibromata, and cystic tumours also are not unusual. Innocent tumours may be removed after ablation of the outer bony wall of the cavity; malignant tumours demand excision of the upper jaw and are of very bad prognosis.

15. Reflex Nasal Neuroses.—While there can be no doubt that treatment applied to the interior of the nose does in some cases markedly relieve or even permanently cure certain conditions mostly affecting the respiratory passages, it must be admitted that the exaggerated statements put forward by some rhinologists explain, if they do not entirely justify, the scepticism entertained by the profession regarding the connection between diseased conditions in the nasal passages and other affections.

In the large majority of instances, but by no means in all, it will be found that nervous exhaustion or neurasthenia or hysteria will be found to play an important part in the production of the nasal neurosis, and that either hyperæsthesia of the nasal mucosa or erectile swelling of the turbinated bodies will commonly be present. Space only admits of mention being made of some of the more important morbid conditions which may be relieved or even cured by intra-nasal treatment.

Asthma.—Not infrequently polypi, hypertrophic rhinitis, morbid conditions of the inferior turbinated bodies, and more rarely deformities of the septum nasi, are found in patients suffering from bronchial asthma; and relief—most frequently only partial, but in some instances total and permanent—of the asthmatic affection has followed treatment directed to the removal of the nasal condition.

Paroxysmal Coughing and Sneezing are apt to be induced by hypertrophic and diseased conditions, particularly of the inferior turbinated bodies, and to cease after appropriate treatment, e.g. cauterisation.

Headache, Neuralgia of the Fifth Nerve, Megrin and Spasmodic Closure of the Glottis are in rare cases much benefited by intra-nasal treatment, and may therefore be considered as occasional reflex nasal neuroses; but epilepsy, vertigo, exophthalmic goitre, and many other morbid conditions (many of them of hysterical origin), have no claim to be considered as such. See PAIN IN VISCERAL DISEASE.

In connection with reflex nasal neurosis, mention must here be made of the interesting group of vasomotor coryzas, of which the best known examples are hay-fever (see HAY-FEVER), rose-fever, and rhinorrhœa, which last has already been described in this article.

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NOSTALGIA (*νόστος*, return; and *ἄλγος*, sadness).—**SYNON.**: Fr. *Nostalgie*; Ger. *Heimweh*.—A form of melancholia, sometimes occurring in persons who have left their homes. The symptom from which it derives its name is an intense desire to return home; and this is accompanied by great mental and physical depression, which may end fatally. See MELANCHOLIA.

NUCLEUS.—See CELL.

NUMMULATED SPUTUM (*nummus*, a coin). A form of sputum which, when spreading out on a surface or floating in water, resembles a coin in shape. See SPUTUM, Examination of.

NURSING THE SICK.—Nursing is performed usually by women, under the direction of physicians and surgeons. Good nursing should put the sick under the best possible conditions for Nature to restore health and to cure disease or injury. The physician or surgeon prescribes these conditions—the nurse carries them out. Partly, perhaps mainly, upon nursing must depend whether Nature succeeds or fails in her attempt to cure. Nursing is therefore to help the patient to live. *Training* is to teach the nurse to help the patient to live. Nursing is an art, and an art requiring an organised, practical, and scientific training. For nursing is the skilled servant of medicine, surgery and hygiene.

Nursing may be divided under four heads: (a) *Hospital nursing*. (b) *Private nursing*: that is, nursing one sick or injured person at a time, at home; giving the whole time to that one patient, generally of the richer classes. (c) *District nursing*: that is, nursing the sick or injured poor at home, taking as many cases as can be well attended to by one nurse. District nursing, or nursing the sick poor at home, is a branch of nursing of the highest importance, and requires the highest qualifications, because the district nurse has not, like the hospital nurse, a medical and surgical staff always at her call, and never has hospital appliances to her hand. (d) *Midwifery nursing*, including the nursing of the healthy mother and infant after natural childbirth, the feeding, washing, and clothing of infants, and the teaching the mother the management of her own infant and herself, will not be treated of here; but it may be observed that midwifery and septic or infectious cases should not be attended by the same nurse. No ordinary precautions will secure the lying-in case from danger arising out of this practice.

Nursing proper means, besides giving the medicines and stimulants prescribed, or applying the surgical dressings and other remedies ordered—(1) The provision of fresh air in proper quantity without draughts (ventilation), especially at night, and the maintenance of a proper temperature. (2) Hygienic conditions in the sick-room or ward, which include light, cleanliness of floors and walls, of bed, bedding, and utensils. (3) Personal cleanliness of patient and of nurse, quiet, variety, sympathy, and cheerfulness. (4) The administration and sometimes preparation of diet (food and drink).

(5) The application of remedies. (6) Observation of the patient. We shall now discuss these duties in succession.

1. Ventilation. Warmth and Coolness.—

(a) *Ventilation* is the removal of the air poisoned by the breath and other human emanations, and supplying its place with *fresh* air.

The very first canon of nursing is to keep the air inside as fresh as the air outside, by night as well as by day, without chilling the patient. The best rule of ventilation is still: Poke the fire and open the window. Air should be admitted from the outside. Windows are made to open, doors are made to shut. If the nurse ventilate the patient's room or ward through the door—that is, make the room draw the foul air from the rest of the house or building—she supplies him with foul, not fresh air. But ventilation is impossible without sufficient floor and cubic space, and unless the windows open near the ceiling. All patients need a free supply of air without draughts, especially cases of infectious disease, the danger of spread being reduced in proportion as ventilation is free.

(b) *Warmth or coolness*.—This the physician has to prescribe—the nurse has to see to it. In fever, for instance, the physician will require her to examine the patient's feet and legs, at least every hour, to ascertain whether they are chilled, and to keep the extremities warm, even though his temperature be high, whether in summer or winter.

In bronchitis and in some other cases an even, high, moist temperature may be necessary, and a steaming kettle may be required on the fire night and day.

But ordinarily it is not advisable to keep the sick-room always at the same temperature. A cooler air at night is necessary. But whether cool or warm, the air must be *fresh*. Sick children become fretful in foul air at night. And young as well as old night-nurses require training to see that the physician's orders are obeyed as to keeping the air of the ward fresh by night, and not above or below a certain temperature. If warmth is obtained by means of an open fire, the nurse must replenish it noiselessly, without waking or disturbing the patient. For this purpose lumps of coal wrapped in damp paper may be kept ready.

The head of the sick person should never be higher than the throat of the chimney, which ensures the best air. And the chimney should never be closed with a chimney-board or other contrivance.

2. *Health of Sick-Room, or Ward*.—This might be called 'nursing the room.' The placing the sick-bed in the best position to secure air without draught, light without glare, quiet and cleanliness—and this often necessitates rearrangement of the furniture of the whole room—is one of the essential arts of nursing. In district nursing of the poor, it must be one of the nurse's first duties to put the room in a state so that the patient can recover. The hospital and the hospital-ward must be so built that the patient shall not 'die of hospital.' To get rid of the conditions which have interfered with health is of course the first step in helping Nature to get rid of the effects of those conditions.

(a) *Light*.—Second only to air is light as an essential for growth, health, and recovery from sickness—not only daylight, but sunlight—and indeed *fresh* air must be sun-warmed, sun-penetrated air.

This should be meant to include colour, pleasant and pretty sights for the patient's eyes to rest on—variety of objects, flowers, pictures. People say the effect is on the mind. So it is; but the enlightened physician tells us it is on the body too. The sun is a sculptor as well as a painter. The Greeks were right as to their Apollo.

(b) *Cleanliness*.—Cleanliness and fresh air do not so much give life as they are life itself to the patient. Cleanliness—clean air, clean water, clean surroundings, and a fresh atmosphere everywhere, are the true safeguards against 'infection'—not segregation—or rather segregation by ample floor and cubic space, ample ramparts of fresh atmosphere, not segregation by walls and divisions. You cannot lock-in or lock-out the infectious poison; you cannot wall-out infection. You *can* air it out, diffuse it, and clean it away.

Cleanliness of floors, ceilings, walls, bed, bedding and utensils, and of sinks; also of lockers, if any—but there should be none.

Floors and walls.—Medical men forbid scrubbing in the sick-room. No sick-room floor ought ever to be washed, except by the doctor's orders and at the hour he orders.

The best floor for cleanliness, except in institutions where specially non-absorbent materials can be used, is planed boards, saturated with 'drying' linseed oil, well rubbed in, stained (for appearance sake) not too dark, so as *not* to hide the dirt, and polished with beeswax and turpentine. The floor should be wiped with a damp cloth and dried with a floor-brush, or cleaned by a brush with a cloth tied over it. Anything offensive or infective spilt should be washed off *at once* with soap and water. Hospital-ward floors should be scraped and polished every fortnight by a *frotteur* and dry-rubbed by a man every day. The patients should be provided with slippers. There should be no carpet, of course, in a sick-room, except a piece of washing druggit by the bedside. A dirty carpet literally infects the room.

The only clean wall is one that is absolutely non-absorbent. Of ordinary materials oil-paint and glazed tiles are probably the best. From this you can wash the animal matters. These are what make a room musty. The worst wall is the papered wall. The next worst is the plastered wall. But the plaster can be made safe by frequent lime-washing and occasional scraping. The paper requires frequent renewing. A glazed paper gets rid of a good deal of the danger. But the ordinary bedroom paper is all that it ought not to be.

Furniture—as little as possible in the sick-room—should all be of polished wood, metal, or marble, kept clean by being wiped with a cloth wrung out of hot water.

Air can be soiled just like water. Air is always soiled where walls and carpets are saturated with animal exhalations. Dust consists largely of organic matter. There should be no ledges out of reach capable of holding dust. An Arnott's ventilator in the chimney will keep an ordinary paper longer clean, showing the connection of ventilation and cleanliness. Inattention to these essential matters all but foils the best nurse's best efforts.

How to clean.—Dust is the harbourer and harbingers of disease. Dust in hospitals may contain enormous numbers of living organisms.

The only way to *remove* dust is to wipe everything with a damp cloth. And all furniture ought

to be so made that it may be wiped with a damp cloth without injury to itself, and so polished or glazed that it may be damped without injury to us. Flapping, by way of dusting, is not cleaning. To 'dust,' as usually practised, merely means to distribute dust more equally over a room.

No one atom of dust ever actually leaves the room under the ordinary system of 'dusting.' The greater part of nursing consists in keeping clean. No ventilation can freshen a sick-room where the most scrupulous cleanliness is not observed.

Bed and bedding; linen, &c.—The most dangerous effluvia we know are from the excreta of the sick; these are often placed, at least for a time, where they must throw their effluvia into the underside of the bed, and the space under the bed is seldom aired; indeed, it cannot be, so long as there is a valance or counterpane down to the floor.

An adult in health exhales by the lungs and skin in the twenty-four hours three pints at least of moisture, loaded with poisonous matter. In sickness the quantity is often greatly increased—the quality is always more noxious. This goes chiefly into the bedding, because it cannot go anywhere else; and it stays there, because, except perhaps by a weekly or bi-weekly change of sheets, scarcely any other airing is attempted. A nurse will be careful to fidgetiness about airing the clean sheets from clean 'damp,' the clean nightgown from clean 'damp,' the new mattress from clean 'damp'; but airing the dirty sheets from dirty 'damp,' the dirty night-gown (which she is going to put on the patient after washing him) from dirty 'damp,' never so much as occurs to her. And a mattress is supposed to be aired by somebody else sleeping on it and saturating it with his own 'damp' before the patient comes to exhale into it the patient's 'damp.'

Where a patient has to lie on a mackintosh it is often desirable to place two or three layers of folded blanket under the draw-sheet, in order to secure some ventilation.

The best bed and bedding are: A metal bedstead with rheocline springs, or the woven wire-mattress, no valance and no curtains; one thin hair-mattress, light Witney blankets, no heavy cotton counterpane, which retains perspiration. Beds and bedding are best disinfected with superheated steam.

The patient should, if possible, be able to see out of the window from the bed.

Two beds, one for the day and one for the night, are desirable for the best nursing of the patient. A true nurse always knows how to make a bed, and always makes it herself. Bad bed-making has much to do with bed-sores. She hangs up the whole of the bedding to air for a few hours whenever possible. She makes the changes of linen and bed-linen—sheets and draw-sheets—as often as is necessary, which is a great deal oftener than is usually done. In hospitals, she sees to no patient using his neighbour's towel; and to different towels being used for different purposes. In private houses cases of purulent ophthalmia, ending in blindness, have been known to occur from the use of a soiled towel by another member of the family. She sees to all dirty linen, and especially bandages, being instantly removed and disinfected. In dressing wounds, removing drainage-tubes, catheters, &c., great care should be taken that no drop of discharge or of sputum should fall on the linen or bedding, and if such an accident should occur it should never be allowed to dry, but be at once wiped off with a

piece of cotton-wool wet with perchloride of mercury (1 : 500). Dirty linen should be removed immediately from the sick-room and sent to the laundry at least every day. If we are careful to take away and empty bed-pans directly, surely this is still more important with soiled sheets. In infectious cases linen removed from the bed or the patient should be placed at the bedside with as little disturbance as possible in a wet sheet, and so carried away. When bedding in bulk has to be carried through a ward or through a house before disinfection, it may be rolled in a damp sheet. The nurse who has been in attendance on an infectious case should, on going out, take a prolonged hot bath, wash her hair, and put on a complete change of clothing, which should include fresh boots. In a hospital the laundry should be in a separate building.

Bandages with any discharge on them are always to be burnt at once.

Utensils.—All chamber-utensils and bed-pans should be of white glazed earthenware with well-fitting lids. None should ever be left under the bed, but be brought to the room, and, when used, carried immediately to the closet-sink, emptied, and rinsed there. No zinc pail or pail without a lid should be carried through a ward or sick-room. The pail should be of glazed earthenware or enamelled iron, with a lid. But better no pail at all in a sick-room. Without care for these things, the doctor will tell us, 'it is impossible to nurse.' Excreta have often to be put by for medical inspection; the nurse must see to this being done properly and inoffensively, in a closed vessel—never in the patient's room or ward. As for urine, if it has to be measured and tested, there are glass-measures with covers fit for the purpose. Bed-pans should have in them carbolic-acid lotion (1 in 20). All bed-pans should have lids. Glass urinals, with wide necks, washed with warm water and soda, are the only really clean ones; zinc and white earthenware, with long necks, are never clean. After being used they should be put by the bedside, not under, and taken away and emptied at once. Small white chamber-utensils are useful, and district nurses may find old jam-pots the cleanest thing for urinals. It should be a rule, invariable—rather more important in the private house than elsewhere—that the utensil should be carried directly to the water-closet, emptied there, rinsed there, and not brought back till it is wanted.

There should always be water and a tap in every water-closet for rinsing.

Towels in a hospital should be kept separate for three separate uses, changed for clean ones as often as possible, and marked 'Hands,' 'Bed-pans,' and 'Basins.'

A solution of mercuric chloride (1 : 1,000) and some glycerine should always be by, to wash the hands.

Slop-sinks may be sluiced down with carbolic acid. *Water-closet pans* should be scrubbed with strong nitric acid, if they have been allowed to get at all offensive. Urinals, if allowed to become furred, must be sluiced out with boiling water, and then, if necessary, scraped with a knife all round and inside the grating; so also water-closet slop-sinks. These all should be scrubbed with sand and chlorinated lime at least twice a week. In hospitals the head-nurse ought to mop-out and rinse-down the urinals every morning herself with a little bed-

pan mop, and let boiling water run through; the same with the water-closet pans. The lavatory basins, when used, should be mopped-out and scrubbed with sand every morning. There should be two mops—one new one for lavatory basins, appropriated when a little old to the bed-pans, and the old one replaced with new; the new mop to hang over the lavatory basins, the old one to hang over the slop-sink for bed-pans; an old bottle-brush for the handles of bed-pans, a new bottle-brush, kept in the ward-kitchen, for bottles. Ordinary basins should be washed with tow, which can be burnt after use.

3. Personal Cleanliness, Precautions against Finger-poisoning, &c.—One of the most important points nurses have to be taught on beginning surgical ward-work (and, indeed, surgeons also), is how not to poison their fingers. No good nurse will poison her own fingers any more than her patient's.

The following rules should be strictly observed, noting only that many efficient disinfectants are now in use:—

Anything which has soiled the fingers or wounded the cuticle—a hang-nail, a crack, a pin-puncture—or even the condition known as ‘chapped hands,’ is a possible source of infection to others or to yourself, almost more than an open wound or sore which necessarily exacts attention. Such abrasions must be rendered harmless by thorough washing with water, dusting with iodoform, and protecting with a collodion-film. A rubber finger-stall, which can be disinfected by boiling, should be worn. Immediately *before* beginning any dressing or touching a patient, whether in dressing wounds, rubbing in applications, administering enemata, internal syringing, washing out eyes, ears, nose and mouth, turn up the sleeves, scrub the hands, wrists, and nails thoroughly with hot soda-water and soap, and soak them in some reliable disinfectant such as biniodide or perchloride of mercury, 1 in 1,000; lysol, 1 in 100; carbolic acid, 1 in 20, &c. Carbolic acid of a strength sufficient for effective disinfection causes some skins to crack, and in this way becomes a source of danger. Forceps, scissors, and other instruments, both of metal and glass, should be boiled in water containing 1 per cent. of soda (roughly a teaspoonful to the pint) immediately before use on each case, and after use should, if possible, be taken to pieces, well brushed, with special attention to serrated surfaces and joints, with hot soda-water and soap, and carefully dried before putting together again. Soiled dressings should be removed with dressing-forceps and not with the fingers; the fingers should never touch pus if contact can be avoided: on no account should adhesive plaster or other adhering dressing be scratched up with the nails. The fear of dirt is the beginning of good nursing. With all internal cases, keep the nails short, fill all crevices with soap, and carefully anoint up to their roots the fingers you are about to use, especially the first and second fingers in attending on vaginal cases, with carbolic oil or carbolic vaseline, 1 in 20. Dip the tube or nozzle to be used for any internal application in carbolic oil and, before introducing it, sponge the surrounding parts (such as the urethral orifice in introducing the catheter) with perchloride-of-mercury solution or other disinfectant. It is better to use two basins in washing wounds, so as not to dip the fingers in solution that may become soiled. Metal

and glass catheters must be cleansed and disinfected by running a stream of hot water through them, and then boiling them in soda-solution. Glass nozzles and catheters should be kept after thus cleansing in a covered jar filled with perchloride of mercury, 1 in 1,000. Catheters and nozzles of other material which will not stand boiling must be cleansed first in cold water by pouring a stream through them and briskly agitating them from side to side to clear the eye, and then in some disinfectant, carbolic acid, 1 in 20, or biniodide of mercury, 1 in 1,000, but they must not be *left to soak* in the disinfectant, as the solutions injure varnish and gum. Do not syringe down towards the eye of a catheter, but if at all in the reverse direction. Never fail to take your own carbolic soap, with which you will be provided, in your own soap-tin, into the ward each morning and evening in your pocket. But take it out before beginning ‘dressings,’ as otherwise you put a dirty hand into your pocket. Always dry your cleansed fingers and hands on towels *not* used for any other purpose. Cuffs and sleeves and stuff dresses are possible carriers of contagious matter. Always change the apron and over-sleeves which you have worn about the sick before eating or drinking. Report immediately any scratch or hang-nail or sore you may have to the ward-sister. Never go on duty in the morning without having taken a meal.

4. Food and Drink (Diet).—The physician will tell us that, to give food and stimulants in the way, at the time, of the kind, with the cooking and preparing, that will best enable the poor enfeebled digestion to assimilate it, is one of the great nursing arts. No chemical rules can be given for this as absolute. The patient's stomach is the laboratory, and also the chemist. It is the sole judge of whether the physician's orders are right, and the nurse has to watch and tell him what the patient's stomach says. She must, of course, be trained and cultivated to understand what it says.

The patient's stomach sometimes craves, and assimilates too, what no rules would have prescribed for it. The nurse must ask the physician whether she may gratify these cravings. Sick-cookery should do half the digestion's work; and proper variety is essential. Indeed, how much of the suffering of illness, as well as of its danger, is the fault not of the illness but of the nursing, is well known to the skilful physician and surgeon.

The nurse, of course, has nothing to do with the prescribing of stimulants any more than of medicines. But life often depends—especially in fevers and severe surgical injuries—upon the nurse knowing how to follow the indications of the changes to be looked for in the patient's state given her by the physician, and to change the times of giving the stimulants accordingly.

The nurse must know how to make gruel, arrow-root puddings, egg-flip, drinks, good beef-tea, and other kinds of sick-cookery, so as to please the patients' taste and vary their diet. People say ‘fanciful patients’ must be ‘humoured.’ So they must; but it is in order to excite the proper secretions of saliva and gastric juice necessary for digestion. Nothing should ever be cooked in the ward or in the patient's room.

But though ‘sweet Jack Falstaff’ says ‘A nurse is a cook,’ the whole of the cooking must not be thrown on the nurse, if she is to nurse; and above all, if she is to eat, she must not be expected to

cook for herself. But she will always be required not only to see that the patient's food and drink be as prescribed, but that it be well cooked, and punctually and well served. The physician considers that upon the nurse's power to give weak patients food in the way they like often depends their taking, or at least assimilating, any food at all.

She has also to feed, for example, fever-cases so that they can eat. The mere lifting-up of a patient in bed to give him food may terminate fatally a fever-case. The nourishment or stimulant ordered may have to be put into his mouth perhaps every half-hour—perhaps every few minutes—without rousing the patient. The physician expects the nurse to be able intelligently to make the variations he prescribes in giving these things, especially during the night, according to the state of pulse and other symptoms, which she must know how to observe, in order to follow his conditional directions, upon which hangs the patient's life from hour to hour, often from minute to minute. In convalescence from typhoid fever, one single false indulgence has often induced a fatal termination.

5. Application of Remedies.—The physician or surgeon requires the nurse—

To be able to dress blisters, burns, sores and wounds.

To administer stimulants and medicines as ordered, enemata and injections to men and women, and suppositories.

To manage trusses, appliances in uterine complaints; to pass the catheter—at least for women. The district nurse is often now required to pass the speculum for women, also the catheter for men, because there is no one else to do it.

To use the best methods of friction to the body and extremities; to make and apply fomentations, poultices and minor dressings, wet and dry or greasy; to syringe wounds; to syringe the vagina.

To manage helpless patients—fever, operation and surgical cases—that is to move, to change their linen, to keep them personally clean, warm or cool.

The medical attendant will expect the nurse to maintain an exquisite cleanliness of the patient's whole person and skin, and, in fever—the daughter of dirt—to clean the patient's teeth, gums and tongue, with lemon-juice or white-of-egg beaten to a froth. A nurse is no nurse who cannot wash or sponge a patient's whole body *without exposure or chill to any part*. In typhoid and other fevers, this is often an essential part of the treatment.

To give food and stimulants to helpless patients—fever, operation and surgical cases; to manage the position of such cases; to prevent or to dress bed-sores.

To make the sick-bed, and especially to make the bed with the patient in it; to change the under-sheet without moving the patient, as in fever and operation cases. The 'best way' includes, in this as in all other things, the doing them at the least expense to the patient's vital powers.

To prepare the bed for accidents and various kinds of operations; to undress, handle and put to bed accident cases.

To attend at and prepare for operations: to prepare patients for and manage them after operations and anæsthetics—and all this with the least call upon their small strength.

To be able to do the first thing in case of hæmorrhage, namely, compression by hand or finger, by extemporary tourniquet and plugging.

To bandage all the various parts of the body, arm, leg and chest (in Paris the *infirmiers* of military hospitals are made to practise all this, till not only it is done perfectly, but in a given number of minutes).

To make bandages of various kinds: T-bandages, double-headed, 'binders,' many tailed, ovariectomy, triangular, starched, and plaster-of-Paris, and other stiff bandages.

To make rollers, to line and pad splints, to make leather and gutta-percha splints, fracture- and chaff-pillows, and sand-bags.

The nurse should be able to give subcutaneous injections, to use the galvanic battery, to dry- and wet-cup, and to apply leeches externally and internally.

She is required to be able to apply dry and moist heat; to give inhalations and use the spray-disperser; to apply cold, with the use of syphons and with ice; and to carry out aseptic or antiseptic treatment. Every surgeon and physician has his own 'antiseptic solutions,' his own 'disinfectants,' and every year brings fresh ones. And what is ordered must of course be used by the nurse.

6. Observation of Patients.—The physician and surgeon require every nurse to be able to observe correctly, and to report correctly, on the state or character of secretions, expectoration, pulse, skin, appetite, strength; effect of diet, of stimulants and of medicines; eruptions; the development of swellings or other abnormalities; the characters and starting-points of fits; as to intelligence, with regard to delirium, stupor, &c.; as to breathing, whether quick or slow, regular or irregular, difficult, &c.; as to sleep, whether sound, starting, heavy, &c.; and as to the state of wounds. The physician also requires the nurse to be able to 'take' and to record the temperature—sometimes every quarter of an hour in critical cases—the pulse, the respiration; to measure, and sometimes to test the urine for him. She will be required to make these observations—if possible still more accurately—for child-patients, who cannot tell what is the matter with them; to understand the management of sick children and children's wards, which need a yet more exquisite cleanliness. And children show a much more rapid change of symptoms for life or for death generally than adults. Children are the best air-test, the best test of sanitary conditions.

Other Duties.—She must understand the management of convalescents—a whole department of nursing in itself—and the sooner a convalescent, especially a convalescent child, is removed from hospital to a country 'home' the better.

Housekeeping.—She must understand a certain amount of housekeeping or domestic economy—rather an uncommon talent. She must be able to order the proper quantity of bread, milk, butter, &c., and to prevent waste.

She must be competent to undertake the charge of linen—a most important item of nursing.

Generally.—Nursing is, above all, a progressive calling. Year by year nurses have to learn new and improved methods, as medicine and surgery and hygiene improve. Year by year nurses are called upon to do more and better than they have done. It is felt to be impossible to have a public register of nurses that is not a delusion.

Further, year by year, nursing needs to be more and more of a moral calling.

Night-nursing.—The physician or surgeon requires the night-nurse to be as good as, or even better than, the day-nurse, for the most critical times of fever and severe surgical injury often occur at night, or in the very early morning. But quite the same kind of business capacity is not required in the night-nurse as in the nurse in day charge of wards. Night-nurses, to do their work well, must have at least seven or eight hours in bed where they can sleep undisturbed by day. They must have hot meals prepared for them when they come off duty in the morning, and before they go on duty at night; besides breakfast at 1 or 2 a.m. They must have one and a half or two hours' exercise. In a hospital they should be obliged to show their pass. It is rather more necessary for a night-nurse to be regular in her habits, if she is to be well and efficient, than for a day-nurse. And there appears no reason why nursing by night, if properly managed, should be more trying than by day. But regularity of habits, of meals, of sleep, of exercise, of personal cleanliness, is the *sine quâ non*. Occasional breaks or transfers to day-duty may be necessary; or a night or two in bed every month for a night-superintendent. But a too frequent shifting (e.g. every month or every three months) from day to night duty seems hardly advisable, for it takes some time for a nurse to accustom herself to sleep by day. A night-nurse may deteriorate in two ways, if not carefully supervised: (1) She may become 'bumptious,' and too independent. (2) On the other hand, she may become careless and hard from having too much to do to do it properly. The night-nursing in the medical wards of a large hospital is generally much 'heavier' than in the surgical wards, and the matron ought to know from her own experience when the night-nurse absolutely requires additional help, and to provide it. Instant help should also be within call in an emergency, such as a patient becoming suddenly delirious. And the night-superintendent should always be on the watch for emergencies.

Holidays.—All nurses, especially night-nurses, must have holidays, as well as occasional recreation. A month's regular holiday in the year is not too much. Yet more do matrons and superintendents and all women filling nursing offices of great responsibility require an annual holiday, if they are to maintain vigour of body and mind, and not to wear out prematurely.

What a Nurse is to be.—A really good nurse must needs be of the highest class of character. It need hardly be said that she must be—(1) Chaste, in the sense of the Sermon on the Mount; a good nurse should be the Sermon on the Mount in herself. It should naturally seem impossible to the most unchaste to utter even an immodest jest in her presence. Remember this great and dangerous peculiarity of nursing, and especially of hospital nursing, namely, that it is the only case, queens not excepted, where a woman is really in charge of men. And a really good trained ward 'sister' can keep order in a men's ward better than a military ward-master or sergeant. (2) Sober, in spirit as well as in drink, and temperate in all things. (3) Honest, not accepting the most trifling fee or bribe from patients or friends. (4) Truthful—and to be able to tell the truth demands attention and observation, to observe truly—memory, to remember truly—power of expression, to tell truly

what one has observed truly—as well as intention to speak the truth, the whole truth, and nothing but the truth. (5) Trustworthy, to carry out directions intelligently and perfectly, unseen as well as seen, 'to the Lord' as well as unto men—no mere eye-service. (6) Punctual to a second, and orderly to a hair—having everything ready and in order before she begins her dressings or her work about the patient; nothing forgotten. (7) Quiet, yet quick; quick without hurry; gentle without slowness; discreet without self-importance; no gossip. (8) Cheerful, hopeful, not allowing herself to be discouraged by unfavourable symptoms; not given to depress the patient by anticipations of an unfavourable result. (9) Cleanly to the point of exquisiteness, both for the patient's sake and her own; neat and ready. (10) Thinking of her patient and not of herself; 'tender over his occasions' or wants, cheerful and kindly, patient, ingenious and *feet*. The best definition can be found, as always, in Shakespeare, where he says that to be 'nurse-like' is to be

'So kind, so duteous, diligent,
So tender over his occasions, true,
So feat.'

A patient wants according to his wants, and not according to any nurse's theory of his wants or 'occasions.' 'Tender over his occasions' she must be; but she must have a rule of thought; and this the physician or surgeon has to give her in his directions; which her training must have fitted her to obey intelligently, using discretion. The nurse must have simplicity and a single eye to the patient's good. She must make no demand upon the patient for reciprocation, for acknowledgment or even perception of her services; since the best service a nurse can give *is* that the patient shall scarcely be aware of any—shall perceive her presence only by perceiving that he has *no* wants. The nurse must always be kind and sympathetic, but never emotional. The patient must find a real, not forced or 'put on,' centre of calmness in his nurse. To call upon a patient by emotion for emotion is the most cruel, because useless, demand upon his strength. It is asking him to bear your troubles and your anxiety as well as his own. Suppressed emotion is as bad—it makes the nurse constrained. It is exposing the patient to both frost and fire. Half the battle of nursing is to *relieve your sick from having to think for themselves at all*—least of all for their own nursing.

FLORENCE NIGHTINGALE.

FLORENCE NIGHTINGALE BOYD.

NUTMEG LIVER.—See LIVER, Passive Congestion of.

NYCTALOPIA or NIGHT-BLINDNESS (*νύξ*, night; and *ὤψ*, the eye; the letters 'al' probably represent *alpha privativum*, and express *want of*).—**SYNON.**: Fr. *Nyctalopie*; Ger. *Nyctalopie*; *Nachtblindheit*.—By the term 'nyctalopia' is meant an abnormal degree of impairment of vision at night, or whenever the light is dim. The degree of impairment ranges in different cases from slight embarrassment to practical blindness. During the day or in a good light the vision may be of normal acuteness, but rapidly fails at dusk.

Like hemeralopia, the term 'nyctalopia' has been used in the opposite senses of *night-blindness* and *day-blindness* (see **HEMERALOPIA**). Many modern writers have used it in the latter significa-

tion; but all the ancients, and nearly all the later writers to the end of the seventeenth century, used it in the former. In the nomenclature of the Royal College of Physicians of London the original meaning is adopted.

Though night-blindness is at all times merely a symptom manifested in several diseases, it is convenient to classify it as *symptomatic* when associated with certain appreciable morbid changes in the fundus oculi; and *idiopathic* or *essential* when unaccompanied by such changes.

Of *symptomatic nyctalopia*, by far the most common concomitant is retinitis pigmentosa. It may occur in connection with detachment of the retina, with syphilitic and other forms of retinitis and choroiditis, and with some embryological defects or intra-uterine diseases of the retina or of the choroid. In all the affections of this group the beginning of night-blindness is insidious, and the course is chronic, the physical condition being some morbid alteration in the percipient and pigmentary layers of the retina.

Idiopathic nyctalopia is usually more or less abrupt in its onset, and due to torpor of the retina, showing itself by deficient adaptation of the retina to feeble illumination; and is an exaggeration of the physiological blindness which temporarily takes place in passing from bright into feeble light. In many instances it follows prolonged exposure of the eyes to intense glare of any kind. This is especially apt to occur in persons who have been insufficiently or improperly fed, or who show malnutrition from any cause. Formerly it was often met with as an early symptom of scurvy, and it still occurs epidemically among the poor in those countries where strict religious fasts are observed. It is also observed in some cases of anæmia, albuminuria, liver-disease with or without jaundice, pregnancy, malaria, and chronic alcoholism. In many cases of idiopathic nyctalopia there co-exists a peculiar form of xerosis of the bulbar conjunctiva. On the conjunctiva opposite the palpebral fissures are scaly oily-looking patches, which, by gentle frictions of the lids, can be whipped into a fine white foam abounding in minute bacilli. The exact relation between the xerosis and the nyctalopia has not been determined. The xerosis may only be an expression of impaired nutrition of the eyeball.

The *prognosis* of symptomatic night-blindness is generally unfavourable; while that of the idiopathic form is more favourable—at least in so far as the associated constitutional and local states may be amenable to treatment.

TREATMENT.—This must necessarily be determined by the ætiological and associated pathological conditions. Of the symptomatic form the treatment is that of the accompanying retinal or choroidal affection; and of the idiopathic form the treatment will be that of the associated constitutional and local conditions. The eyes should be protected by tinted glasses, shades, or bandages; faults and errors of diet corrected; cleanliness of the lids ensured; and tonics and other regimen and medicaments suited to the special requirements of the particular case must be employed. JOHN TWEEDY.

NYMPHOMANIA (νύμφη, the nymph, a portion of the female sexual organs; and *μανία*, madness).—A form of mental derangement in women, characterised by an insatiable desire for sexual intercourse.

NYSTAGMUS (νυσταγμός; from νυστάζω, I nod). Rhythmical, involuntary, frequent oscillations of the eyeballs, of small range and more or less continuous. The movements are not commonly horizontal, but may be vertical, rotatory, convergent, or mixed in direction. They are usually binocular, but may be unilateral, and then are said to be generally vertical; they consist usually of a rapid movement in one direction and of a slower return. The rate varies from 60–200 per minute, rarely slower, sometimes faster; the rhythm is regular; the range most commonly from 2–4 mm. (Gowers). The movements cease during sleep, are often increased by observation and by violent exertion, are most obvious during fixation of a somewhat distant object, and are sometimes suppressed on strong convergence; they may be so fine as only to be noticed on ophthalmoscopic examination. The movements of the eyeballs are otherwise unaffected, but the field of fixation is reduced. Nystagmus may be present in all positions of the eyeballs, or only in certain directions of movement, when weakness of affected muscles is often present. Slow movements become quicker when the eyes are moved (Gowers). *Associated movements* sometimes occur; oscillatory movements of the head, generally opposed in direction to the eyes, and partly compensatory; simultaneous movements of the upper eyelid or contractions of the orbicularis palpebrarum; hippus; torticollis spasm; in children clonic contractions of the neck-muscles (Bruns); in one case similar lateral movements of the pharynx and larynx (Gowers).

VARIETIES.—Nystagmus is seen in health in a person looking at a quick succession of moving objects, and also in anyone rapidly rotated on a table spinning on its vertical axis.

Experimentally it is commonly observed, at least temporarily, after electrical irritation of the cerebellum; more frequently in lesions of the cerebellum (Ferrier). Risien Russell considers that the experimental evidence points to lateral nystagmus, with jerks towards the side of lesion, as associated with lesion of a lateral lobe, and irregular or rotatory with affections of the middle lobe of the cerebellum. Nystagmus may be *congenital* or *acquired*.

I. Congenital Nystagmus.—Congenital nystagmus is present during rest and in all positions of the eyeballs; there is no apparent movement of objects, and the patient is only conscious of weakness of vision. It appears soon after birth or in early infancy. It is generally associated with eye-diseases which impair visual acuity, e.g. diseases of the retina and choroid, congenital cataract, corneal nebulae, very rarely anomalies of refraction. It occurs in albinism. Nystagmus may also be present in congenital defects of the brain, and in cerebral disease or injury in early infancy. It very constantly appears in the affection of infancy known as 'Head-Nodding,' in which it is generally bilateral, may precede the head-movements and persist after them, and sooner or later almost always disappears. *See HEAD-NODDING IN CHILDREN.*

II. Acquired Nystagmus.—In acquired nystagmus the patients may be unconscious of its presence, but many complain of 'dancing of objects' before the eyes, this apparent movement being generally in the same direction as the nystagmus. In the following varieties eye-diseases may aggravate but do not cause nystagmus.

1. Occupational.—Miner's nystagmus is the most

common, but it occurs in other occupations involving continued strain on the movements of the eyes. See OCCUPATION-DISEASES.

2. *Symptomatic*.—In disseminated sclerosis it is a common and often an early symptom. The direction varies, but it is most commonly horizontal; it is generally bilateral, and only present when the eyes are moved, but may be constant. In Friedreich's ataxia nystagmus is often present in lateral positions of the eyeballs. It is sometimes found in the chronic stage of acute maladies, such as disseminated myelitis, and in ataxic paraplegia on movement of the eyes (Gowers), and rarely in primary spastic paraplegia, syringomyelia, progressive muscular atrophy, and amaurotic family idiocy. In tumours of the cerebellum it is frequent (24 out of 41 cases, Ferrier), much less so in cerebellar abscess, and absent in cerebellar atrophy. It occurs in cerebral tumours, hæmorrhages, and softening in various situations, according to some authors especially in lesions of the optic thalamus and fourth ventricle; in thrombosis of sinuses, chronic hydrocephalus, pachymeningitis hæmorrhagica, sudden general anæmia of brain, and temporarily after severe concussion. It is rare in tubercular, but more common in posterior basic meningitis. In Cheyne-Stokes' respiration it may accompany the period of activity. It may occur in eclampsia nutans, and rarely as a sequel of epileptic attacks.

3. *Aural*.—Nystagmus may occur in severe attacks of aural vertigo, and in diseases of the ear attended by increase of pressure within it.

4. *Toxic*.—In uræmic convulsions it is occasionally present, and this fact may be connected with its appearance in rabbits after the injection of hypertoxic urine into their veins. It occurs in benzene-poisoning together with immobility of pupils, and in chronic alcoholism as the result of cerebro-spinal sclerosis (Bevan Lewis).

5. *Reflex*.—Nystagmus is said to occur temporarily from some peripheral irritation; carious teeth (Ross); injury to opposite eye.

6. *Voluntary*.—A case is recorded in which rapid lateral nystagmus could be produced voluntarily and maintained for a minute.

DIAGNOSIS. — True nystagmus must be distinguished from slight nystagmoid jerkings, especially in lateral positions of the eyeballs, met with in parietic conditions of the ocular muscles, in debilitated states, and in some hysterical cases; in the last it is often associated with characteristic blinking of the eyelids (Russell). According to the best authorities nystagmus does not occur in hysteria. As the varieties of nystagmus which are found apart from diseases of the nervous system can be excluded by the history and conditions present, nystagmus is valuable in diagnosis chiefly because it points to organic disease of the nervous system, and is sometimes an early symptom of it; in doubtful

cases of nervous disease its presence in different directions of movement of the eyes should be carefully sought for. The association of nystagmus with pallor of the optic discs is important in the early diagnosis of some cases of disseminated sclerosis. As a localising symptom of intracranial disease, it is not available, but the facts given above as regards the cerebellum should be remembered.

MODE OF PRODUCTION.—The causes of nystagmus are thus very numerous, as would be expected in view of the wide connections of the centres for the ocular movements. In all cases the seat of disturbance must be referred to these centres, and not simply to fatigue of the ocular muscles themselves. The power of steady, sustained fixation of the eyes in binocular vision is not present at birth, but during the first few weeks or months of life the neuro-muscular mechanisms of the eyeballs are educated to act in co-ordination with the visual perceptive centres. A reflex action of great delicacy is thus formed, by which in fixation the image of the object falls upon the macula. But this reflex co-ordination of ocular muscles and perceptive centres cannot be established if in early infancy there is defect of vision, especially one which lowers the visual acuity of the yellow-spot region. While the congenital form is thus explained, in the other varieties in which there is no ocular disease the conditions present probably interfere with the action of the oculo-motor centres, either directly or through the tracts which reach them from other parts of the brain. Thus in the nystagmus in rapid rotation of the body, and in certain ear-diseases, the centres are disturbed through their connection with the semicircular canals. It is difficult to explain why under such circumstances the steady contraction of the muscles during fixation in health should be changed into the oscillations of nystagmus. When any movement is carried out the opponents of the muscles concerned also contract, and so the resulting action is rendered steady; if from any cause the centres for the ocular movements did not act exactly synchronously on the two sides, an alternating contraction of the muscles might result. The intermittent character of nystagmus is explained by Gowers on the supposition that the centres have a tendency to rhythmical, intermittent action which is normally kept in check, and when this is removed, intermittent spasm of the ocular muscles results. See NERVOUS SYSTEM, Physical Examination of.

TREATMENT. — Treatment of the occupational form consists in a change of work; resumption of the old employment results sooner or later in a relapse. Tonics, especially strychnine, and sometimes bromides, are useful. In the congenital form an effort may be made to correct any marked strabismus or errors of refraction.

J. MICHELL CLARKE.

O

OBESITY (*obesus*, corpulent; from *ob*, by reason of, and *edo*, I eat).—**SYNON.**: Corpulence; *Poly-sarcia*; Fr. *Obésité*; Ger. *Fettsucht*; *Fettleibigkeit*.

DEFINITION.—This term is applied to a general state of disordered nutrition of the body, characterised by an excessive development of adipose tissue and various disturbances in the bodily functions.

ÆTIOLOGY.—(a) **Predisposing Causes.**—The influence of *heredity* in transmitting the liability to obesity is undoubted, and is a matter of common knowledge. But an examination of the families of fat people appears to show that obesity is indifferently transmitted with rheumatism, gout, oxaluria, diabetes mellitus, and gall-stones; all which perversions of nutrition are in some manner closely correlated. *Sex and Age.*—That excessive corpulence is more common among women than among men is also well known. Several circumstances have been suggested to account for this, such as the menstrual functions of women, pregnancy and suckling—from which fitness often dates, their less muscular activity as compared with men, and their frequently diminished oxidative power, due to poverty of red blood-corpuscles. Age appears to have considerable influence in determining this condition. Under a healthy regimen children get fat from birth, notwithstanding that at the same time the proteid ingesta must be largely employed in the construction of the rapidly growing tissues; and hence, at this period of life, the fat and starchy food-stuffs are the chief sources of the adipose deposit. Children frequently are seen, improperly fed on excess of starchy matter, yet very fat, while their general nutrition is much impaired. At puberty there is frequently a diminution in weight, both relatively to the height and absolutely; but the contrary to this sometimes accompanies the establishment of menstruation, especially if the subject be very chlorotic—that is, with an enfeebled oxygen-carrying blood-power. After the age of forty, particularly in women at the climacteric, the influence of age markedly asserts itself. Even the manifestation of the hereditary tendency may be postponed until that period, and for women to become fat at that time is very common. The perversion of nutrition now under consideration is, in some unknown way, curiously but distinctly associated with the degree of development of the sexual functions, and in an inverse direction. This is very noticeable in eunuchs and animals whose generative organs have been removed, and the part played by the cessation of ovulation has been already mentioned. Even during pregnancy, when ovulation is suspended, it is no uncommon occurrence for the subcutaneous fat to be increased in amount. *Occupations.*—Those compelling a sedentary life undoubtedly favour the development of corpulence, especially if there be hereditary predisposition, together with large eating and alcoholic indulgence. *Race.*—Among certain races obesity appears to prevail, as for instance the Hottentots; and while among some, such as certain castes of Hindoos, the condition has been highly esteemed, among others, as the Greeks and Romans, it was regarded as dis-

graceful. *Climate.*—Although very fat people are met with in all climates, there appears to be a special tendency to their predominance in low-lying, damp countries, while, with certain exceptions, they are less often seen in very hot and in mountainous districts.

(b) **Determining Causes.**—Excess of *food* is the first of these to be mentioned. While no doubt a large excess of food may lead to corpulency, it must be confessed that it very often does not do so, and extremely thin persons are often large eaters. And, on the contrary, many women who become excessively obese have poor appetites. It has been estimated by Bouchard that 40 per cent. of the obese are large eaters, and that 10 per cent. eat less than the normal quantity. Nor does it seem in these different classes of cases that the kind of food makes much difference. Some get fat, eat what they will; others do not, whatever their diet. Nevertheless, starches and sugars are, as a rule, more effective fat-producers than fats. *Drink.*—It is, however, usually the case that very fat people take a large amount of fluid food. How alcohol acts in the production of fat is not very clear. It is asserted that it does so by diminishing oxidation; but this is not the entire explanation, for the extent of obesity is far from being proportionate to the amount taken, and not infrequently an excessive ingestion is not associated with corpulency. There would also seem to be something due to the form in which the alcohol is taken. *Exercise.*—Deficient muscular activity, by diminishing the amount of oxidation of tissue, favours obesity, especially in cases of idleness following on a period of considerable energy, the quantity of food taken remaining the same; and since, as a rule, the stouter the person, the less capable is he of exercise, these two conditions react one upon the other, in favour of fat-production. But lack of exercise, like over-feeding, is not invariably followed by corpulence, nor are the two conditions when combined. Bouchard indeed calculates that only 37 per cent. of fat people take too little exercise, and 28 per cent. even exceed the average man in this respect. *Disease.*—Exceptional cases of corpulence have followed recovery from severe fevers, and extensive bleedings, even when there had been no predisposition; and a similar result has been met with after prolonged administration of mercurials and arsenic, which is perhaps to be explained by the deteriorating influence that these drugs are said to possess on the red blood-corpuscles.

PATHOLOGY.—Assuming that the current views on lipogenesis, or fat-formation, are known to the reader, it is sufficient here to state that from whatever source the fat of the body be derived, whether from the fatty, the starchy, or proteid elements of the food, or from all, as is most probable, the fact of its being stored up as adipose tissue must be regarded chemically as an expression of deficient or delayed oxidation—a process which, if it had been more complete, would have resulted in the conversion of these elements into carbonic acid and water, to which the fat itself is reduced when it is subsequently used up in the economy. It is thus that the corpulence that frequently attends such

morbid states as anæmia, chlorosis, hæmorrhage, some pulmonary and cardiac diseases, and alcoholism, is to be explained; since in all these diseases the oxygenating power of the blood is deficient.

The fat of the body in an average male adult constitutes about one-twentieth, and in the female rather more, of the total weight. It is not for $3\frac{1}{2}$ months after the commencement of development that the adipose tissue is sufficiently differentiated to be distinguishable; it gradually increases in amount, being considerable at birth and up to puberty, when it often diminishes slightly; during maturity it increases, or the reverse, being very variable in amount; and during old age it decreases. During childhood the adipose tissue is more evenly distributed in the subcutaneous tissue than in later life, when fat tends relatively to diminish on the surface in proportion as it becomes deeper-seated.

In the three situations in which the fat is chiefly deposited—namely, the subcutaneous, subserous, and intermuscular connective tissue—there are certain areas which are preferred by it, as there are others which escape. While the abdomen, buttocks, breasts and neck are especially prominent, the wrists, ankles, eyelids, scrotum, and penis are free from fat. Beneath serous membranes it is very unequally distributed. Fat is never seen beneath the peritoneal coat of the stomach or intestines, the parietal pericardium, or the visceral pleura; while the great omentum, which usually weighs about $\frac{2}{3}$ lb., may reach to 7 or 8 lbs., or, it is said, even 30 lbs., and a thickness of half an inch or more of fat may be included between the layers of the mesentery. A considerable amount is developed beneath the visceral pericardium; and under the synovial membranes fat may be deposited to such an extent as to interfere with the movements of the joints. Adipose tissue is never found within the cranial cavity, but may be present in large quantity in the spinal canal.

The ordinary state of the organs found in very corpulent people is, that the lungs are small; the heart and the liver large, and infiltrated with fat, the cavities of the former being dilated, with degeneration of the muscular fibres; and the latter showing a state of partial cirrhosis or being extremely fatty; the gall-bladder containing only a little pale bile or mucus; the stomach large and muscular, but well-developed, and the intestines often dilated; the kidneys small, as also the spleen and lymphatic glands; and the pancreas largely developed. In extreme cases the blood may contain four to five times the normal amount of fat, sufficient to give it an altered appearance to the naked eye.

Like many other conditions of disease, it is impossible to define the exact line at which a morbid obesity may be said to commence. All degrees of corpulence, indicated by such terms as 'stout,' *embonpoint*, &c., occur, to which the notion of disease is wholly inapplicable. Nor can disturbance of function be taken in all cases as the measure of a morbid state, since the impairment of function is not always proportionate to the amount of fat.

In extreme cases one-half, or three-fourths even, of the body-weight may be fat.

SYMPTOMS.—The general appearance of a corpulent person scarcely needs description. The condition may be associated either with a hyperæmic or full-blooded, or with an anæmic state of

body (the hæmoglobin being deficient in amount rather than the corpuscles too few), and it is desirable to recognise this in view of treatment. Owing to the fatty infiltration of the muscular tissues, and the degeneration of the fibres, the muscular energy is diminished, this being especially noticeable in regard to the heart, the action of which is easily disturbed, so that palpitation is a frequent symptom, accompanied by dyspnœa, induced by slight exertion. The affection of the voluntary muscles manifests itself in an indisposition to active exercise. The digestive power is often very well maintained, and this notwithstanding the frequent excess both in the quantity and in the quality of food indulged in. Periodical impairments are, however, frequent; and pyrosis, flatulence, and constipation or diarrhœa, with offensive stools containing much fat, are often troublesome. The cardiac sounds are usually feeble and distant, though the reverse obtains when there is a hypertrophied ventricle. The pulse is very variable, full, or small and weak, according to the plethoric or feeble state of the individual; more or less frequent than normal, irregular or dicrotic, dependent on the degree of fatty change in the heart, or, if of high tension, from fibrosis of the arterioles (*see* HEART, Fatty Accumulation on). The mental activity is uncertain, and many external causes tend to modify it; but the temperament is proverbially 'easy-going,' indolent, and lethargic, especially after meals, although very frequently interrupted by attacks of peevishness and irritability, or by unusual somnolence and quiet. The remarkable condition known as 'narcolepsy,' which is characterised by sudden brief attacks of irresistible sleep, is sometimes observed in the obese, both with and without such complications as albuminuria and diabetes. Examples, however, of considerable intellectual attainments are not unknown among the corpulent. Profuse sweating is induced by slight exertion, and the secretion of the sebaceous glands is abundant and often fetid. The urine is generally acid, often diminished in quantity, and contains an excess of uric acid, urates, and frequently oxalate of calcium. Partly from chafing, and partly from the excessive cutaneous secretions, intertrigo and other eruptions are apt to occur in the folds of the groin, below the mammae, and in similar parts. The vessels share in the general malnutrition of the tissues, and atheroma of the arteries is often found, while the veins become distended and varicose, forming hæmorrhoids and varicocele. Some œdema of the ankles is also usual, and hæmorrhages from nose or lungs are not uncommon. Depending upon these vascular changes are the congested and bloated appearance of the face, and the liability to headaches, vertigo, and giddiness. Disturbances of sight and hearing are frequently noticed in fat people. Irregularities of menstruation, which may be early in appearance, are frequent. The sexual appetite is frequently deficient in both sexes, and sterility is common in women: in man it has been attributed to the deposition of fat along the course of the spermatic vessels, causing impaired nutrition of the testes, as shown by the scanty and inactive spermatozoa found in the semen. Accumulation of fat around the umbilical ring favours the occurrence of hernia in this situation, a not uncommon event in the corpulent. The enormous weight of the abdomen causes a constant backache from painful overstrain of the dorsal muscles.

The condition of obesity, like other general perversions of nutrition, most distinctly presents other characteristics than the mere signs and symptoms above enumerated. 'Corpulence is not only a disease itself, but the harbinger of others,' as Hippocrates pointed out. There are certain tendencies and liabilities which the state engenders; and intercurrent maladies come to possess special features. Periodically, the fat man ails without perhaps any obvious cause, and such ailments must be regarded as the expression of malnutrition of the tissues produced by the excess of fat. Among the more prominent of these affections is a proneness to catarrh of the respiratory and alimentary mucous membranes, and periodical 'colds' and diarrhoeas are frequent. This is in great part due to the fact that the power of self-regulation of temperature, which the body possesses, is diminished by the thick layer of subcutaneous fat, which is a bad conductor of heat, and interferes with compensatory radiation. At the same time the plethoric condition, and the enfeebled circulation due to the weak heart, tend to the same end, namely, a liability to congestion of the ill-supported tissues, such as the mucous membranes, with the results of such congestion in excessive secretion and other derangements of function.

The obese subject is quite as liable to the acute diseases as is the thin man; and these maladies run in him a singularly unfavourable course. The diminished power of heat-radiation increases the pyrexia; and the weak heart favours the establishment of the adynamic state. Such means for lowering the temperature as cold applications have but little effect through the thick fat; and aconite is contra-indicated by the pulse. But since the oxidising process in the corpulent is diminished, the temperature in the febrile state is rarely very high, and at the same time is but ill resisted.

The effective agent in fat-formation, namely, deficient oxidation, also favours the formation of uric acid, and hence the fat are often gouty. Saccharine urine (a condition which, whatever view be taken of its pathology, is manifestly an expression of deficient oxidation)—especially that form which is met with in those advanced in life—very frequently occurs in stout people, not only in an intermittent form, but constantly with other symptoms of true diabetes mellitus. In 32 of 140 cases of diabetes mellitus observed by Seegen, obesity preceded the glycosuria.

A somewhat infrequent form of obesity, occurring chiefly in middle-aged women, has received the name of *adiposis dolorosa* (p. 28). It is characterised by local fatty swellings, sometimes symmetrical and usually in the arms, in addition to a general state of obesity. Severe paroxysmal pain is associated with the swellings, which are often very tender, with a general anæsthesia of the surface elsewhere. Such patients tend to exhibit periods of dementia, or epileptical attacks. Headache, cutaneous pigmentation and a liability to hæmorrhage have been observed. A previous history of alcoholism or of syphilis has been generally obtained. Portions of nerves removed from the painful areas have shown the changes characteristic of neuritis.

PROGRESS AND PROGNOSIS.—The progress of obesity is essentially chronic. Extreme fatness in the very young, however, usually subsides; but the obesity of advanced life never does, unless any exhaustive disease should co-exist, such as cancer

or diabetes; and the latter by no means produces then the emaciation that it causes in young people. Obesity should, on the whole, be regarded as a grave matter, since very fat people rarely reach an advanced age; while a decrease of fat at middle age in a person hitherto stout should be regarded with suspicion.

In obesity, death by syncope may result from an extremely fatty heart; from apoplexy, caused by rupture of an atheromatous vessel in the brain; or from acute pulmonary congestion, with general œdema from cardiac dilatation; while rupture of the heart, angina pectoris, and uræmia are among the dangers to which the obese are liable. Not infrequently some sudden exertion has immediately preceded the fatal end.

TREATMENT.—Recognising that accumulation of fat is a perversion of nutrition, which, if once established, and with a strong hereditary predisposition, cannot be cured, it follows that we should endeavour to prevent as far as possible its increase, by avoidance of those factors which pathology tells us are favourable to its development. The guides as to how far a given plan may be proceeded with are, first of all, the age and general condition of the patient, especially as regards the heart's power; and, secondly, the feelings and capability of the patient, as the treatment is pursued. Each case must be treated according to circumstances, bearing in mind that the objects to be aimed at are to diminish the sources of the fat, and to increase the oxygen-carrying power of the blood and oxidising power of the tissues. Unless the patient actually suffers from the accumulation of fat, it is generally undesirable to pursue any special treatment, and under no circumstances should the weekly loss be allowed to exceed 3 lbs., and the total loss should not go beyond 28 lbs. It must be remembered that some persons far from deriving benefit from any regular plan of treatment distinctly deteriorate in health, and caution should be exercised in the selection of cases.

The diet must be regulated in quantity and quality. Since a healthy diet should consist of certain proportions of nitrogenous, carbohydrate, and fatty principles, and since from all these three substances fat may be formed in the body, the question arises, which can be most advantageously diminished. Experience supports our pathological knowledge in advocating a withdrawal as far as possible of fatty and more especially starchy food, while at the same time a moderate increase in proteid matter is permitted; for with a fair quantity of the other food-stuffs, proteids increase tissue-change. It is on this principle that systems of dietary for the corpulent are founded, the best known of which bears the name of Banting, who in a year (1863) reduced his weight by 44 lbs., and without any recurrence of corpulence when ordinary diet was resumed. There are many other cases recorded. Dr. Cheyne, who weighed 32 stone, reduced himself a third in weight, and lived afterwards in good health to the age of seventy-two. Lean meats, sweetbread, fish—except rich kinds, such as salmon and eels—clear soups, poultry, game, eggs, cheese, green vegetables, toast, gluten-bread, fresh fruit, and pickles are allowable articles of diet. An average diet for an adult would be 12 oz. lean meat, 6 oz. rusks or gluten-bread, 4 oz. green vegetables, 1 oz. butter, and 1½ pint tea. If the normal daily requirements be put at—

proteids 130 grammes, fats 86 grammes, and carbohydrates 380 grammes (these ingredients being water-free); then the Harvey-Banting diet may be taken as—proteids 170 grammes, fats 10 grammes, carbohydrates 80 grammes. It is necessary that the *régime* be steadily adhered to for a considerable period, and recurred to from time to time when the weight shows signs of increasing. Several well-known modifications of this diet have been proposed. Ebstein's plan was based on—proteids 100 grammes, fats 85 grammes, carbohydrates 50 grammes, with 1,750 grammes of fluid, being an amount of proteids much below the normal, while the fats are of the healthy quantity. The deficiency of proteids tends to prevent this diet being pursued for long without loss of strength, and it would be probably more economical if rather less fat and rather more starch were allowed. Oertel's system is characterised by a marked restriction of fluids, the dry proteids being 170 grammes, fats 44, and carbohydrates 114 grammes. The reader will determine for himself how these proportions of alimentary principles may be distributed among various ordinary articles of diet. The exact influence of an excess of water in tissue-metabolism is uncertain, but there is no doubt that a large number of fat people consume an excessive amount of fluid, of which it may not be forgotten that alcohol forms very frequently a considerable proportion, and on such grounds a restricted intake might be regarded as desirable. But while cases of obesity associated with anaemia may be benefited by this treatment, it must be admitted that dehydration is attended with great risk in plethoric persons, especially if the nitrogenous food exceed or even equal the normal quantity, since serious disorders may result from accumulation in the tissues of their products of waste, from lack of fluid to flush them out. Still more is this the case in those extreme methods of dieting which consist of a practically unlimited amount of meat food, with complete abstinence from fats, starches, and sugars; and with such regimen a large allowance of hot water is insisted on. This plan, known as the Salisbury treatment, has been pursued with considerable success. This method consists in a daily diet of about 3 lbs. of lean beef, roasted or boiled, equally divided between the three meals, for a week or ten days, after which chicken or white fish may be given for a similar period, and then fats and later farinaceous foods are very gradually added. The fact is that each plan has its merits and can claim successful results, but judgment is requisite in its application to individual cases. All methods compel a very great decrease from the normal of starches, but on each of the other alimentary principles much diversity of practice exists. It cannot, however, be too strongly insisted on that for any considerable increase in nitrogenous food the kidneys must be in a perfectly healthy state, and this should only be permitted in conjunction with a free exhibition of water and a regular examination of the urine. Alcohol generally should be avoided, but especially spirits and beer, which must be absolutely forbidden, except on emergency; cider or the light dry wines, both white and red, diluted with water, are less objectionable. Tea and coffee are supposed to interfere with tissue-change, and therefore should be taken sparingly; and milk, from the quantity of fat it contains, is to some extent inadmissible, although a skimmed-milk diet has been proposed.

Regular exercise, within the limits of the patient's power, such as riding, walking, rowing, and gymnastics, is of great benefit by directly inducing an increased oxidation of tissue, and improving the quality of the blood, and therefore its oxygen-carrying power. It should form an essential part of every system of treatment, the amount being determined by the state of the patient's heart. When exercise cannot be taken, passive movements and massage should be employed. Cold bathing, if well borne, is of advantage on similar grounds. Free sweating by Turkish baths is a useful adjunct to dietetic treatment, unless the circulation be enfeebled. Breathing compressed air with the object of increasing the tissue-oxidation has been recommended.

It is in carrying out a system rather than in devising one that the difficulty occurs. The regularity and restraint prove irksome to the patient, and are frequently broken. Hence it is that the regimen and spare diet of the various spas, such as Carlsbad, Marienbad, Kissingen, and Ems have great advantages, since at such places and in such surroundings the patient more readily and willingly pursues a given plan.

In the treatment of intercurrent diseases it is essential to remember the enfeebled resisting power of the patient and the frequent necessity for stimulants.

Among the many drugs that have been used for obesity may be mentioned alkalis, iron, and iodine. Some of the good effects of the various 'waters' are ascribed to their alkaline properties, especially the alkaline aperients of the above-mentioned spas. Iron is an essential in those forms of corpulence associated with anaemia, and most satisfactory results follow its administration, as the health improves and the fat diminishes. Young chlorotic subjects benefit by this treatment, which may be advantageously carried out at some chalybeate spring, such as Tunbridge Wells, Harrogate, or Spa. The iodides, such as those of potassium and iron, given in large doses, undoubtedly effect a reduction in the amount of fat, but not always with a corresponding improvement in health. So long as this does not suffer and the patient improves, the drug may be persevered in, but it is frequently very badly borne when taken in quantity. The preparations of *Fucus vesiculosus*, the basis of certain quack remedies, appear to depend for their value on the iodine contained in them. The extract of the thyroid gland has been found useful in some cases, especially those of distinctly neurotic association. W. H. ALLCHIN.

OBLADIS, in the Tyrol.—Earthy saline waters and climatic health-resort.

OBSESSION (*ob*, opposite; *sedere*, to sit).—Formerly used to imply the state of a person supposed to be besieged by an evil spirit antecedent to possession. This term is now employed in psychological medicine to indicate the sudden occurrence in consciousness of an emotion or an idea dominating the mind, over which the individual has no control, and which may end in a sudden impulsive act which cannot be inhibited by the will. *See* INSANITY: *Imperative Ideas*.

OBSCULESCENT (*obsolesco*, I grow out of use).—A term applied to tubercular products which have dried up, shrunken, and become calcified and inert. *See* TUBERCULOSIS.

OBSTETRICAL PARALYSIS. — See BRACHIAL PLEXUS, Lesions of; and CEREBRAL DIPLEGIA.

OCCUPATION-DISEASES. — By the term 'Occupation-disease,' we mean disease directly traceable to the effects of a particular trade in which a person is engaged. As artisans are equally liable to the ordinary ailments of humanity, it is necessary to discriminate carefully so as to eliminate all causes other than those related to the particular industry in question. In the production of occupation-disease there are usually several factors in operation. Even in the causation of such an indisputable malady as lead-poisoning it is well to remember how important is the part played by individual idiosyncrasy, while in the better ventilated state of one factory compared with another, the home-life and surroundings of the workpeople, poverty, heredity, age and sex, are to be found conditions that are contributory to its production, and therefore not to be ignored. See PUBLIC HEALTH.

Arsenic and Colour-Grinding. — In the smelting of ore containing arsenic and in the manufacture of Scheele's green—an arsenite of copper used for colouring toys, artificial flowers, and wall-papers—workmen are sometimes seriously affected. Men who grind and mix arsenical colours often suffer from ulceration of the skin on those parts of the body with which the arsenic comes into contact. There is developed a painful form of eczema (p. 421). Beginning as vesicles the spots soon become pustular, and are extremely irritable. When an individual is exposed for any period to the dust arising from arsenical compounds, he often experiences a dryness and irritation of the lining membrane of the throat and nose, loss of appetite and indigestion, colic, and the passage of mucus in the stools. He loses flesh, and his eyes look congested. The urine may contain albumen, and there may be painful paralysis of the legs owing to peripheral neuritis. When once arsenic is in the system it is only slowly eliminated from it. Hence it is found in the urine long after the symptoms of poisoning have begun to decline. In the form of an alkaline arseniate it is used to dye wool. The girls who noddle and handle the dried wool thus treated inhale the irritating particles that are given off, and have been known to suffer from general ill-health, digestive derangements, and loss of power in their legs. Sempstresses who are in the habit of heedlessly biting off the ends of thread thus dyed, and men engaged in the manufacture of sheep-dipping washes, have also suffered in a similar manner. See pp. 1077-1078.

Cases of industrial arsenical poisoning observed by medical practitioners must be reported to the Home Office. See ARSENIC, Poisoning by.

Compressed Air.—See CAISSON-DISEASE.

Electricity.—Since the introduction of the electric light, and the employment of electricity in an increasing variety of ways, several fatal accidents have occurred. In the act of repairing wires, the cleaning of machinery, and accidental contact with charged parts of dynamos, workmen have received shocks that have proved immediately fatal. Occasionally the injuries received have been of a slighter character, and, for the most part, burns. An individual runs a very serious risk when brought into personal contact with conductors charged to a high pressure, say 1,000 volts; contact with even

240 volts is known to have caused death. As the result of a series of experiments, carried out by Professor Weber of the Polytechnic at Zurich, with the view of deciding what pressure is dangerous on electric railways with overhead trolley-wires, it was ascertained that to touch both poles of an alternating current circuit, where the pressure exceeded 100 volts, was not free from risk; but as there is little chance of passengers or other persons coming into contact with both the leads of the apparatus, the authorities decided to allow a working pressure of 750 volts, the current to be alternating. Apart from electrical pressure there are circumstances that tend to magnify the danger from contact, such, for example, as a workman standing on damp ground or wearing damp boots. If, on the other hand, he is standing on an indiarubber mat, which is a non-conducting material, he might possibly touch a single object, charged to a high pressure, without any harm following. Alternating and direct currents are equally dangerous.

It is not exactly known how electricity kills, but in all probability it is in one of two ways, either (1) by arresting breathing, or (2) by suddenly stopping the heart's action. R. A. Bolam and the writer carried out a series of experiments on anæsthetised animals with the view of throwing some light upon this question. Where high-pressure currents were used the animals were killed immediately. The heart suddenly ceased beating, but respiration was frequently continued for a minute or more afterwards. Although in most of our experiments death was due to sudden arrest of the heart's action, it was not always possible to say upon which organ or nerve-centres the current exerted its harmful influence. Sometimes we were able by artificial respiration to resuscitate an apparently dead animal, and there is no doubt that in the case of all persons who have received an electric shock and in whom life is apparently extinct, artificial respiration is the line of treatment that holds out the greatest promise of recovery. It should be undertaken immediately and persisted in for more than an hour afterwards.

Fatigue-Neuroses.—Over-use of the muscles and strain in certain occupations are apt to be followed by painful spasms or cramps, and occasionally by paralysis of particular muscles. In the production of these there is probably both a muscular and a nervous element in operation. While lengthened but interrupted employment of muscle through increasing the blood-supply improves nutrition and induces muscular hypertrophy, too protracted use, on the other hand, and overstrain, particularly if accompanied by the evil effects of working in a close atmosphere, cause breaking down. Spasm, tremor, or paralysis of muscle may thus be induced. It is customary to speak of either of these as *occupation- or fatigue-neuroses*, and in employing these terms we are not committing ourselves to any expression of opinion as to the pathological nature of the affection. There is an obstinacy or persistency about these neuroses that often defies treatment and makes their recovery tedious; also the muscular atrophy that is induced shows a tendency to get worse: circumstances that raise the question whether the affection in some instances at least should not be considered as outside the pale of a purely functional disorder. The morbid anatomy that underlies the condition is not known. As the industries of the country keep on multiplying and

machinery of a complicated character is introduced, requiring delicate manipulation and constant attention on the part of the worker, there is a corresponding increase in the number of fatigue-neuroses. These neuroses more or less resemble each other in the manner in which the different causes have operated to produce them. In *writer's cramp*, which is elsewhere separately dealt with, the excessive use of the pen is responsible for its production, and not the fact of the pen having been made of steel, as some persons suppose. The hurry and worry of modern life, and the high tension of the nervous system, are factors in its causation that cannot be ignored. In *file-makers*, who cut the tools by hand, paralysis and atrophy of the small muscles of the hand occur; and while plumbism is the prevailing cause in operation, some of the effects may be due to fatigue, especially the loss of power in the left thumb and forefinger which tightly grip the chisel, and the muscles of the right hand which wield a heavy hammer all day when at work. Plumbism and muscular fatigue are therefore both in operation in file-makers' paralysis. See WRITER'S CRAMP.

Clergymen and public singers who use their voice too much may overstrain the delicate muscular mechanisms concerned in vocalisation so that there is a sudden stoppage of the voice in public utterance owing to spasm of the glottis, or the voice becomes tremulous. In other instances there is aphonia so complete as to suggest a possible hysterical origin for the defect. Fatigue of laryngeal muscles is responsible for these attacks, which are in no way dependent upon catarrhal conditions of mucous membrane. When aphonia and globus co-exist the affection is neurotic in all probability. Rest from the use of the voice is a necessary part of the treatment. Bromides and Nux Vomica are useful; electricity may be tried, and change of air and scene recommended.

Typists, persons who work the Morse- or other telegraphic instrument and those who play the piano or the harp, occasionally find that the fingers and hands, owing to the excessive strain imposed upon them, refuse to carry out voluntary movements, and that the muscles become tremulous or are thrown into a state of spasm and do not relax readily. The spasm may be perfectly painless, but, occurring as it does at inconvenient moments, it worries the individual and prevents him accomplishing his task. Sensation is not affected. There may be no actual paralysis of muscle, but there is usually a degree of paresis that prevents co-ordinated movements being readily carried out. Usually the muscles respond to electricity. The battery is therefore well worthy of a trial in these cases along with massage and nerve-tonics. During treatment stimulants should be avoided.

The writer has seen a few instances of atrophy of the muscles of the left shoulder in railway surface-men due to the habit of these men carrying their heavy implements on their shoulders. No doubt exposure to wet and cold also has its influence. The affection sometimes resembles progressive muscular atrophy although it is due to a neuritis. *Bricklayers* from prolonged grasping of the trowel in the right hand sometimes suffer from muscular spasm.

Miners' Nystagmus.—Coal-miners occasionally exhibit a very marked oscillation of the eyeballs in a lateral or rotatory direction when asked to fix

their attention on any particular object, and which obliges them to put their ocular muscles on the stretch. Tremor is then noticeable. This tremor may cause very little inconvenience in some men, but in others it is associated with vertigo. The objects looked at appear to be unsteady. In miners of a nervous temperament nystagmus rather aggravates any neurotic tendencies. The oscillatory movement of the eyeballs is attributed to strain of the muscles of accommodation and convergence caused by the miner working in the flickering light of his Safety lamp. Secondary oscillations of the head are often present. Simeon Snell, of Sheffield, finds that nystagmus only occurs in men who are working in narrow seams of coal, and who in the act of hewing the coal lie on their side with their eyes cast in an upward and oblique direction, thus imposing a lengthened strain upon the levator palpebræ, superior rectus, inferior oblique and external rectus muscles of one eye, with the same in the other, except that the internal rectus is involved instead of the external. In Newcastle-upon-Tyne nystagmus was some years ago very prevalent among coal-miners, but during the last fifteen years it has become much less frequent. Since nystagmus occurs in such diseases as insular sclerosis and Friedreich's ataxia, it is necessary to distinguish between that due to strain of the ocular muscles met with in the coal-miner and those forms arising in the course of cerebro-spinal disease. The history of the case, the age of the patient, and the absence of affections of speech would assist in the diagnosis. In coal-miners' nystagmus there is no evidence of disease of the brain or of the nervous system. It is solely due to fatigue of the external muscles of the eyeball. See NYSTAGMUS.

Gas in Coal-Mines.—When an explosion has occurred in a coal-mine, and has been attended by fatalities, it has generally been believed that the death of the victims has been the result of shock or extensive burns of the body. In a certain percentage these two causes have been in operation, but there is no doubt that many of the colliers meet their death by inhaling the 'after-damp'—a gas that is rich in carbon monoxide. The proof of this is the ruddy colour of the skin of the cadaver, also that on spectroscopic examination of the blood two bands are observed between D and E, in the yellow and the green, which are not, like those of oxyhæmoglobin, affected by adding ammonium sulphide. Apart from explosions the coal-miner is constantly running the risk of inhaling carbon di-oxide, or 'choke-damp.' This is the gas which makes its presence known to the collier by his lamp becoming extinguished, and by a feeling of slight embarrassment of his breathing. Wherever in a coal-pit a miner's lamp cannot burn, it is evident that human life cannot for long be maintained. Education and experience inform him that it is then time to retire. The miner is thus forewarned of the danger of *choke-damp*, but he has no such warning of the presence of carbon monoxide. It is the presence of this *after-damp* in a coal-mine, after an explosion, that makes it dangerous for a rescuing party to descend. Men who are brought into contact with this gas are insensibly overcome by it. They become drowsy and extremely languid, and by degrees pass into a state of unconsciousness. The presence of even such a small quantity as one per cent. of this gas will cause unpleasant symptoms. Ten per cent. has proved fatal. See DYNAMITE-POISONING.

TREATMENT.—After a colliery explosion fresh air should be got as rapidly as possible into the mine, for although colliers may have been rendered unconscious by the carbon monoxide, yet death may not supervene for an hour or more. Cylinders of oxygen should be taken into the mine, the sufferers should be brought to bank as quickly as possible, warm blankets applied, and stimulants very carefully administered. Above all, artificial respiration must be carried on, and a hypodermic injection of strychnine is worthy of a trial.

Glass-makers and Glass-blowers.—The risks incurred by glass-makers are those due to exposure to high temperature, e.g. bronchial and pulmonary affections, and also cataract. Occasionally glass-blowers develop a cystic swelling of the cheek, due to air passing up the duct of the parotid gland; thickened patches of mucous membrane inside the mouth and cheek; and also deafness.

High Temperature and Muscular Strain. Men who are employed at the blast furnaces of iron-works, in addition to running risk from burns and irritating particles of dust settling on their eyes, are, owing to exposure to heat and cold, liable to bronchial and pulmonary affections and to rheumatism. When young the men as a class are physically strong. Their work is hard and their wages are good. They are able to buy good food, therefore they eat well, but later on they take to drinking stimulants rather freely, and, as their work is arduous, the free consumption of alcohol tends to make them break down and to cause them to become prematurely old.

Men who are employed at the *puddling* furnaces are exposed to very severe muscular strain and excessive heat, and consequently, even in the winter months, they work with very little clothing on. As they perspire very freely they imbibe all sorts of liquids to replace the loss. A very large number of puddlers develop heart-disease, particularly commencing at the root of the aorta, owing to the strain put upon the aortic valves. Puddling is generally regarded as one of the most degrading kinds of work in the iron-trade. Since steel is now very largely taking the place of iron, puddling is much on the decline. It is not required in the manufacture of steel, and as a consequence heart-disease in iron-workers is now less frequently met with than formerly. See *HEAT, Effects of*.

Indiarubber.—Makers of indiarubber goods, especially of the finer variety, are liable to illness of rather a serious character. In order that indiarubber may be able to withstand variations of temperature it has to be vulcanised by being passed through bisulphide of carbon. During this process the vapour of the bisulphide is inhaled and it causes intoxication. This may assume a form of drunkenness not unlike that caused by alcohol, in which the individual staggers and even falls; or the symptoms are less acute, and the individual complains of headache and indistinct vision, or is the subject of epileptiform seizures or muscular tremor. Some of the workers lose the power of their legs owing to peripheral neuritis, others suffer from double foot-drop, while others become ataxic. In a few instances the workers when intoxicated have become acutely maniacal. Treatment resolves itself in the minor form of intoxication into removal of the individual from his occupation, and in the severer forms of illness into the treatment of symptoms.

Lead and its Compounds.—Lead is so largely used in the industries that plumbism occurs unexpectedly in people whose occupation sometimes does not suggest immediate contact with the metal or its compounds. Silk thread, for example, is occasionally passed through a solution of acetate of lead to increase its weight. Sempstresses who are in the habit of biting off the ends of the thread are known to have suffered from colic and general ill-health. The sweet taste of the thread ought to forewarn them of the danger. White lace is, on the Continent, sometimes whitened by carbonate of lead.

File-cutters are especially prone to plumbism. The men and women cut the files upon a leaden cushion. A considerable amount of metallic dust is given off which becomes oxidised. Some of this may be inhaled, but as the occupation is a dirty one, and the workpeople as a class are not cleanly and often eat their food with unwashed hands, the probability is that the poison also gains an entrance into the system through the alimentary canal. Colic and muscular paralysis frequently occur. The symptoms of plumbism develop slowly. Health is gradually undermined, albumen appears in the urine, and the individual succumbs to the effects of a chronic interstitial nephritis. In the United States all files are made by machinery, and as a consequence plumbism among file-makers is unknown. In Sheffield, the centre of the industry, efforts have been made to supplant hand-made by machine-made files, but with only qualified success, for the customs of a trade die hard.

Plumbers who handle metallic lead and use the carbonate for jointing purposes; *printers*, especially *compositors* who set the type; and *house-painters* are all liable to lead-poisoning. Medical men are obliged according to the requirements of the Home Office to notify to the Chief Inspector of Factories cases of industrial lead-poisoning occurring in their practice. House-painting, since it is carried out in the open air, scarcely comes under work done in a factory or workshop, although it might possibly come under the latter. Notification of plumbism in house-painters is, however, accepted by the Home Office, and the usual fee of half a crown is given for each case reported. It is only since notification of lead-poisoning has taken place that we have realised how prevalent is plumbism in this trade. During the month of July 1900 there were 12 cases of plumbism in house-painters reported to the Home Office, one of which proved fatal. In Paris, according to Gautier, house-painting is a prolific cause of plumbism. Of 86 fatal cases of lead-poisoning that occurred in the French capital, during 1894-8, 43 were painters, and out of 14,000 painters and varnishers, Gautier found 250 were annually in the hospital. Statistics have only begun to be kept in our own country. During 1899 upwards of 100 cases of plumbism in painters were voluntarily reported to the Home Office, and of these 18 were fatal.

House-painters become poisoned by handling and mixing the colours, possibly too, although less likely, by inhalation of the terebinthinated vapour arising from the paint, but principally by the fume while burning off the old paint or in smoothing a recently painted surface with sand-paper.

Coach-painters similarly suffer, also men who are employed to break up and burn old railway-carriages.

Glass-polishers use what is called in the trade putty-powder, but this contains frequently as much as 60 per cent. of white lead. In the act of polishing glass a considerable amount of spluttering takes place upon the clothes and hands of the workman and the floor of the workshop. On this material becoming dried, dust is given off, which rises into the atmosphere and is inhaled. Colic and paralysis are therefore not unknown among glass-polishers.

In the *enamelling of iron-plates* for advertisement purposes a large amount of lead is used, and during the dusty process known as 'brushing-off' the girls inhale the powder, and frequently suffer in consequence. Some departments of *white-lead making* are extremely dangerous. See LEAD, Poisoning by.

Pottery.—Lead-poisoning in potters is extremely common. The glaze in which earthenware is dipped frequently contains as much as 30 per cent. of white lead. The men who dip the ware, the girls who clean it, the men who place the ware in the kilns and who are known as glaze-placers, the individuals who make the litho-transfers—the coloured papers used for imprinting colours upon china—colour-dusters and majolica-paintresses run the risk of suffering from lead-poisoning. So prevalent, indeed, had plumbism become in the Potteries that Professor Thorpe and the writer were entrusted by the Government with the task of reporting upon this industry, and of suggesting measures for the diminution, and, if possible, suppression of lead-poisoning. Among other things we recommended the total prohibition of raw lead, i.e. the use of white lead, in the glazes, and the employment of 'fritted' compounds. These suggestions, if put into force, would of themselves cause a very marked diminution in the amount of plumbism. The removal of females from the dangerous departments, and repeated medical examination of all workers in the lead processes, are also measures that would make for health. These, with the recommendations previously mentioned, have the unanimous support of the manufacturers. It is otherwise, however, with the suggestion that leadless glazes should for the most part replace those that contain lead. Only by such a substitution is it possible to render certain processes in the manufacture of earthenware healthy to the workpeople.

Mercury.—*Felt-hats*.—In the manufacture of hats a strong solution of nitrate of mercury is used to promote the felting of the fur. The workmen who handle and shape the felt run the risk of becoming salivated, and occasionally they suffer from the constitutional effects of mercury, including tremor. The danger mostly occurs after the fur is dried, for it is then that dust is given off.

Mirror-makers.—Formerly when mercury was employed in the manufacture of mirrors the workmen suffered from hydrargyria, but since the introduction of the new method of employing nitrate of silver and tartaric acid, mercurial poisoning has disappeared.

Cases of industrial mercurial poisoning must be reported to the Home Office.

Nitro- and Di-nitro-benzole and Aniline. Nitro-benzole is used in the preparation of perfumery, the manufacture of aniline, and of certain high explosives for mining purposes. Nitro-benzole when treated with nitric and sulphuric acids at a high temperature becomes di-nitro-benzole. This substance acts as a poison, whether it is absorbed by

the skin or inhaled. The men who work at the trade experience a heavy sleepiness; they complain of throbbing headache and vomiting, and when the symptoms are more severe, there are dyspnoea and cyanosis, dilated pupils and an ataxic gait. The urine becomes reddish-brown, and both from it and the breath of the workman there is given off the odour of bitter almonds. Coma and symptoms of cerebral apoplexy with Cheyne-Stokes' breathing have been observed. The skin becomes dark blue owing to the presence of aniline. The blood exhibits a dark chocolate colour, loses its power of absorbing oxygen, and gives the spectrum of methæmoglobin. Fifteen drops of nitro-benzole when swallowed have caused death. When injected into the veins of an animal it causes death almost as rapidly as prussic acid, and in acute poisoning there is, strange to say, an odour of prussic acid. These two forms of poisoning resemble each other, but when the two substances were administered by the mouth to animals symptoms of hydrocyanic-acid poisoning came on at once, but those due to nitro-benzole were delayed for a brief period.

TREATMENT is to wash out the stomach, apply mustard-poultices to the chest, administer such a stimulant as ammonia, perform artificial respiration, and apply the faradic current.

Aniline is obtained from coal tar and naphtha. It is a narcotic poison, and acts upon the central nervous system, causing insensibility, convulsions, and motor paralysis. The workmen complain of headache and vertigo, and suffer from spasms. Aniline destroys the blood-corpuscles, and forms methæmoglobin. The urine becomes brownish-black, and the skin is often the seat of eruptions (p. 70).

Phosphorus and Lucifer Match-making. Two kinds of phosphorus are used in the manufacture of matches, the red and white. The red or amorphous phosphorus can be handled without risk. It is used in the manufacture of safety matches. White or yellow phosphorus is so inflammable on exposure to the air that it has to be kept in water. It emits fumes which are luminous in the dark. In the act of glowing phosphorus is being oxidised. The fume itself is one of the lower oxides of phosphorus. Most of the white phosphorus used at home and abroad is made at Oldbury, near Birmingham, and although it is a dangerous industry the processes are carried on so much under cover that it is only very rarely men have been known to suffer. In the manufacture of matches the danger is manifold. It is seldom that there is more than 5 per cent. of white phosphorus present in the paste for heading matches. The men who mix the paste in open iron basins, the men who dip the matches, and the girls who 'box' the dried matches inhale the fumes of phosphorus, and as a consequence some of them suffer from necrosis of the jaw-bone. This is apt to be a chronic affection. Attended at its commencement by a good deal of pain owing to the formation of an abscess at the root of a tooth, pain subsides when once the tooth is extracted and a free vent given to the pus. Disease of the bone progresses slowly. It may continue for months or years, and as the purulent discharge escapes into the mouth and is swallowed ill-health and pulmonary disease are induced. When the necrosis affects the upper jaw it is not unusual for the inflammation of the bone if virulent to extend to the base of the skull and set up a purulent meningitis or cerebral abscess. For-

merly in France match-makers suffered from a constitutional form of poisoning known as *phosphorism*, the principal features of which are anæmia, digestive derangements, and albuminuria. This form of the malady has been seldom observed in England, and yet both here and on the Continent, while we are most familiar with its local effects, viz. phosphorus-necrosis or 'phossy-jaw,' a constitutional state of a peculiar kind is developed whereby without any actual traumatism the long bones readily undergo spontaneous fracture.

There is diversity of opinion as to how phosphorus-necrosis is caused. It is generally held that there must be a pre-existing caries of a tooth; and that through the decayed tooth the fumes of phosphorus make their way to the jaw-bone, setting up a periostitis which soon becomes septic. How far necrosis is the result solely of the action of the irritating fumes of phosphorus it is difficult to say. The disease if induced in the first instance by the oxides of phosphorus is certainly later on aggravated by the presence of microbes. Professor Stockman of Glasgow found tubercle-bacilli in the purulent discharge escaping from the necrosing jaw of affected match-makers, and is disposed to regard the lesion as tubercular.

Prevention of phossy-jaw.—The mixing of white phosphorus should be conducted in closed vessels. The dipping of the matches should be done on slabs, on the distal side of which there should be a fan; the boxing of matches should be accomplished by machinery in preference to hand, but if by hand the boxing should be carried on in front of ventilating shafts, up which the fumes can be drawn away from the worker. The hands of the workers should be well washed before eating, and no food should be eaten in the factory. Overalls should be worn, and since turpentine stops the oxidation of phosphorus, terebinth gargles may be used. There should be repeated dental examination, and on the slightest development of pain in the gums and teeth the worker should be removed at once. Weakly persons with an heredity of tubercular disease, and those who are intemperate, ought not to be allowed to work in the dangerous processes of a match-factory. The treatment of phossy-jaw is surgical. In this country Garman obtained good results by expectant treatment, and the use of antiseptic washes. The cases are tedious. There is little deformity left.

All cases of industrial phosphorus-poisoning must be reported to the Chief Inspector of Factories, Home Office.

Pottery and Phthisis.—See PNEUMOCONIOSES.

Zinc- and Copper-workers.—See BRASS-POISONING; and COPPER-POISONING.

ASBESTOS-DISEASE; ANTHRACOSIS; GANISTER-DISEASE; POTTERS' ASTHMA; and COTTON-WORKERS', FLAX-WORKERS', FLOUR-MILLERS', STONE-GRINDERS', STONE-MASONS' and STEEL-GRINDERS' PHTHISIS are dealt with in the article on PNEUMOCONIOSES. THOMAS OLIVER.

ŒDEMA, (*οἰδῶν*, I swell). — SYNON.: Fr. *Œdème*; Ger. *Oedem*.—A dropsical effusion in intercellular tissue, whether subcutaneous, sub-mucous, subserous, or in the interstices of organs. See DROPSY.

ŒDEMA, Angioneurotic or Wandering.— See p. 70.

ŒDEMA NEONATORUM.— See FŒTUS, Diseases of; and SCLEREMA NEONATORUM.

ŒSOPHAGUS, Diseases of.—SYNON.: Fr. *Maladies de l'Œsophage*; Ger. *Krankheiten der Speiseröhre*. In this article will be included:—

1. The Examination of the Œsophagus, p. 1113.
2. Foreign Bodies in the Œsophagus, p. 1113.
3. Rupture, p. 1114.
4. Acute Inflammation, p. 1114.
5. Chronic Inflammation, p. 1114.
6. Simple Ulceration, p. 1114.
7. Pericæsoophageal Abscess, p. 1114.
8. Syphilis and Tuberculosis, p. 1114.
9. Varicose Veins, p. 1114.
10. Cicatricial Stricture, p. 1114.
11. Dilatation, p. 1115.
12. Tumours, Innocent and Malignant, p. 1115.
13. Spasm (*Œsophagismus*), p. 1115.
14. Paralysis, p. 1116.
15. Malformations, Congenital and Acquired, p. 1116.

Pressure-diverticula (*Pharyngocœles*) are described under PHARYNX, Diseases of. See also PAIN IN VISCERAL DISEASE.

1. Examination of the Œsophagus.—

Methods of examining the gullet by means of the œsophagoscope and auscultation have been introduced, but their utility is so questionable that they will not be described. The bougie is the instrument which gives the only reliable information regarding the condition of the tube, and in every case an olivary bougie with a large bulbous extremity and a narrow (preferably whalebone) stem should be employed. This instrument (*cathéter explorateur à boule*) is greatly superior for diagnostic purposes to the cylindrical bougie generally employed, as localisation of the site of a stricture is much more exact than can be the case with a cylindrical instrument. Certain measurements in connection with the œsophagus must be kept in mind. $15\frac{1}{2}$ to 16 inches is the average distance from the incisor teeth to the cardiac opening of the stomach; $9\frac{1}{2}$ to 10 inches of this measurement corresponds with the length of the œsophagus, and 6 inches to the average distance from the incisor teeth to the commencement of the œsophagus. If the examining bougie pass for a distance of 16 inches or more from the incisor teeth the surgeon may conclude that it has traversed the whole length of the œsophagus and has entered the stomach. Prior to passing an œsophageal bougie the pharynx may be sprayed with a 2-per-cent. solution of cocaine. The instrument, warmed and moistened with glycerine, must be passed down the posterior wall of the pharynx. If, as is commonly the case, the passage of the bougie is temporarily arrested at the pharyngo-œsophageal junction, the patient should be requested to try to swallow it. This manœuvre generally overcomes the obstruction.

2. Foreign Bodies.—Foreign bodies in the œsophagus may be of the most varied kinds. They may be swallowed by accident or by design, as in the case of jugglers, as well as by insane patients. Artificial teeth, coins, and fragments of bone are among the commonest objects found. Such bodies are apt to lodge in those parts of the tube in which the lumen of the œsophagus is naturally constricted, viz. (1) its commencement, just behind the larynx; (2) a point just above the junction of the upper and middle thirds; and (3) at the lower end, just above

the diaphragmatic opening. The symptoms are dysphagia, pain at the site of the obstruction and sometimes cough, with regurgitation of part of the food swallowed, and later on discharge of blood and pus without cough or vomiting. Digital exploration of the pharynx may detect a foreign body impacted near the commencement of the gullet, and a bougie will demonstrate the presence of one lower down. The majority of foreign bodies, including coins, bones, and gold tooth-plates, may be strikingly localised by the Röntgen rays. Of the many ingenious instruments designed for the removal of foreign bodies in the œsophagus the safest and best is the horse-hair probang, which may be in most cases passed by the side of the offending body and then opened out. As it is withdrawn it pushes the foreign body in front of it. Should simple measures fail, the gullet may have to be opened (*œsophagotomy*), since, if the foreign body be allowed to remain impacted for several days, ulceration and perforation of the œsophagus will in all probability ensue.

3. **Rupture.**—This very rare and necessarily fatal accident may, it is said, occur in a previously healthy viscus as the result of violent vomiting, though probably some weakness or lesion in the tube has existed in the majority of instances. Intense pain, collapse, and death in a few hours follow the accident. The site of rupture is always in the lower part of the tube. This accident must not be confounded with the far more frequent condition of *post-mortem* digestion of the œsophagus.

4. **Acute Inflammation.**—Acute œsophagitis may occur from various causes. It is of interest in that it may lead to stricture, ulceration, perforation, and pericœsophageal abscess. It is found (1) in cases of scald and burns of the œsophagus, whether caused by boiling fluids, strong acids, or caustic alkalis; (2) in traumatic lesions due to the presence of foreign bodies; (3) in infants, in whom it may occur without apparent cause, or as an extension of an inflammatory process from the pharynx; (4) in the course of any of the specific fevers; and (5) in the last stages of wasting diseases, e.g. phthisis and cancer, especially cancer of the gullet itself.

The chief symptom is pain in swallowing, which may be rendered impossible, combined with tenderness on pressure over the gullet and spitting of tenacious mucus. Frequently, however, particularly in the mild catarrhal cases, the pain is so slight that in the absence of all other symptoms the acute œsophagitis is unsuspected. The catarrhal cases call for no treatment beyond the administration of bland fluid food. In aggravated types of the disease, e.g. in those due to swallowing strong caustics, feeding by the mouth must be discontinued, and nutrient enemata employed. In such severe ulcerative and gangrenous cases with great destruction of mucous membrane, cicatricial strictures of the worst kind frequently result.

5. **Chronic Inflammation.**—Chronic œsophagitis.—This is generally a sequel of the acute affection above described. Chronic inflammation is generally found to a greater or less degree above the site of a stricture, whether cicatricial or malignant, and is of great importance, in that it predisposes to ulceration and perforation of the gullet. In chronic drunkards a peculiar form of chronic œsophagitis is sometimes met with, associated with vomiting, chronic ulceration of the mucous membrane, and, it may be, with fibrous stricture.

6. **Ulceration.**—Not infrequently in wasting diseases small catarrhal ulcers are found in the œsophagus. They have rarely been met with in typhoid fever, syphilis, and tuberculosis. The cicatrization of simple ulcers may result in stricture of the œsophagus. Ulceration with perforation is common in advanced carcinoma.

7. **Pericœsophageal Abscess.**—This term is applied to a suppurative inflammation of the pericœsophageal connective tissue. The inflammation may originate in the œsophagus itself, e.g. ulceration and perforation (due to foreign bodies or carcinoma), or in the neighbouring lymphatic glands; or it may spread from some other focus of inflammation, e.g. retropharyngeal abscess or spinal disease. The œsophagus may be displaced in various directions dependent upon the place of origin of the abscess. The symptoms are pain in the course of the œsophagus, greatly aggravated by swallowing or movement of the head and neck and elevation of temperature. If the suppuration is diffuse death invariably ensues. If it is localised and is situated in the neck, incision will be indicated as soon as the presence of pus is suspected. A localised abscess, situated in the thorax, may burst into the gullet, the pus be vomited or swallowed, and recovery ensue. The diagnosis of pericœsophageal abscess is most difficult, and is generally only made on the *post-mortem* table.

8. **Syphilis and Tuberculosis.**—(a) Syphilis attacks the œsophagus but rarely, and then always in the tertiary stage. Localised gummata or gummatous infiltrations are found in the mucous and submucous coats of the viscus. Ulceration leading to cicatricial contraction is prone to occur. The symptoms are merely those of difficulty and pain in swallowing, and the diagnosis, except in cases in which tertiary syphilis is manifestly active, is most difficult. In the earlier stages, i.e. before cicatricial contraction has supervened, iodide of potassium in full doses will rapidly relieve the symptoms.

(b) Tubercular disease of the œsophagus is very rare. In a few instances multiple ulcers have been found in the œsophagus near its cardiac extremity, surrounded by a zone of submucous induration, and in the majority of cases in conjunction with tubercular ulceration of the intestines or in advanced tuberculosis of the larynx. Their presence might be suspected if in a case of advanced tubercular disease pain in the lower part of the œsophagus became a marked symptom on deglutition.

9. **Varicose Veins.**—In obstruction to the portal circulation, particularly in cirrhosis of the liver, the veins in the lower two inches of the œsophagus may become extremely dilated and varicose. Severe or even fatal hæmorrhage from bursting of such varices is not uncommon. See HÆMORRHAGE.

10. **Cicatricial Stricture.**—Stricture of the œsophagus due to cicatricial narrowing of the lumen of the tube is the result generally of injury caused by burns from hot fluids or caustic substances (acids or alkalis), less frequently of the traumatism caused by the lodgment of a foreign body, and rarely of the healing of syphilitic, tubercular or typhoid ulceration. In addition to cicatricial stricture malignant tumours infiltrating the wall of the tube produce great narrowing of the lumen of the œsophagus, and, in fact, cancerous stricture is far more common than the cicatricial variety. Congenital malformation is a rare cause of innocent stricture. Pressure from without, as in the case of an aortic aneurysm or media-

stinal tumour, may produce symptoms indistinguishable from those of true organic stricture. Cicatricial stricture may be annular or may extend to several inches in length, and while it may be found in any segment of the gullet it is commonly either at the uppermost or lowermost part of the tube. The diminution of the calibre may be slight or may be so extreme that even fluids can scarcely pass through the stricture. The œsophagus above the stricture generally becomes dilated and its wall hypertrophied, and thus a cavity results in which food, partly-altered by the action of the saliva, may remain for a considerable time before it is regurgitated. When of course the stricture is high up, i.e. near the pharynx, it is at once returned. The chief symptom in all cases is difficulty in swallowing. At first this is for solids only, later on fluids are swallowed if at all only with difficulty, and then regurgitation (false vomiting) of food occurs either immediately or after a varying period of time. The narrowing is progressive until at last deglutition becomes impossible, rapid wasting ensues and death from starvation threatens. The diagnosis is seldom a matter of difficulty. In general there is a history of injury or burn. The passage of a bulbous bougie indicates the site and amount of the constriction. Possible sources of error are pressure of tumours from outside the tube and œsophageal pouches. A careful examination of the chest so as to exclude aneurysm and mediastinal tumour should always precede the passage of a bougie.

TREATMENT.—The majority of cases of cicatricial stricture can be treated by gradually progressive dilatation with fairly satisfactory results. In impassable strictures gastrostomy must be performed. It is sometimes found that strictures which were impassable from above downwards admit of the passage of a bougie passed from the incision in the stomach. Permanent intubation of the œsophagus (*Symonds's tube*) has also been employed.

11. Dilatation.—Primary dilatation of the œsophagus is very rare. In the few instances recorded the middle segment was chiefly affected and a spindle-shaped dilatation produced. Hypertrophy of the muscular wall was also present. It has been suggested that these cases were examples of spasm of the cardiac orifice of the stomach which, of course, would disappear after death; but against this explanation is the fact that it is not the lowest part of the tube that undergoes the greatest amount of dilatation. Secondary dilatation above a stricture is a far more common condition. It needs no treatment, except that of the stricture, to which it is due.

12. Tumours.—*Innocent tumours* are rare. Papillomata, mucous cysts, fibromata, and myxomata have been occasionally met with. These tumours tend to assume a polypoid character, and when situated near the upper limit of the œsophagus may be snared or avulsed by forceps.

Malignant Tumours.—Sarcoma is one of the rarities of medicine. Cancer of the œsophagus is unfortunately of common occurrence, and is most frequently met with between the ages of 45 and 60 years. It is four times as frequent in men as in women, and is as often found in the upper as in the lower segment of the gullet. Squamous epithelioma is the commonest variety, but in about 5 per cent. of the cases a spheroidal-celled carcinoma (encephaloid or scirrhus) is met with. The growth usually takes an annular form, and is

1 to 1½ inches in length. The tumour infiltrates all the coats of the gullet and, owing to the irritation caused by the passage of the food, ulceration sets in early. Not infrequently ulceration, by increasing the lumen of the tube, lessens the dysphagia temporarily. The earliest and chief symptom is dysphagia, which is increased by spasm. Regurgitation of food mixed with ropy mucus and perhaps with blood and particles of the growth soon sets in; and cough with foul breath is generally present. The course of the disease is very rapid, death supervening in a large majority of cases in from 6 to 12 months after the commencement of the dysphagia. The patient may die of inanition, of cancerous cachexia, of septic broncho-pneumonia, of gangrene of the lung, of septic pleurisy, or of perforation of the aorta or other large vessels with fatal hæmatemesis. The above complications are largely the result of extension of the ulcerative process to the bronchi, lung, blood-vessels and pleura. Extension to the lymphatic glands in the neck and chest is common, but secondary growths in parts other than the thorax are exceptional. In rare cases the patient may die of œsophageal cancer without having made any complaint of dysphagia. Pain is not usually a prominent symptom, but in some cases it is both extreme and constant.

The diagnosis in the early stages is sometimes difficult, and in the latent cases impossible. The early symptoms are those of stricture. It is therefore necessary to exclude cicatricial stricture and pressure from without (mediastinal growth or aneurysm). The presence of enlarged cervical lymphatic glands suggests cancer. The bougie demonstrates the existence of a stricture, but the instrument must be introduced with extreme care owing to the liability of the diseased wall to perforation.

TREATMENT.—In cancers situated at the upper end of the œsophagus excision is possible and has been performed with success. In inoperable cases (the large majority) permanent 'tubage' or gastrostomy must be undertaken as soon as swallowing becomes impossible. A flexible gum-elastic tube from 4 to 6 inches in length and funnel-shaped above is introduced, with the funnel resting on the stricture by means of a special introducer (*Symonds's tube*). In many instances this tube is not tolerated by the patient, and in impassable strictures it is useless. In such cases the operation of gastrostomy is to be performed, and will be found of the utmost benefit in prolonging life and appeasing the agonising pains of hunger. The common error is to delay this operation until the exhausted state of the patient renders any operative interference inadmissible.

13. Spasm.—*Œsophagismus.*—Spasmodic stricture is met with in hysterical young women and in men of neurotic tendencies. It must be distinguished from the spasm that so frequently co-exists with true strictures, cicatricial and malignant. The spasm generally commences during a meal, a bolus of food sticking in the œsophagus, and is accompanied by coughing, retching, and loud gurgling sounds. After a time the bolus of food is either rejected or passed on into the stomach. A burning pain is commonly felt at the site of the constriction.

Important points in the diagnosis are the co-existence of globus hystericus, the acute character of

the pain, the sudden onset of the spasm, and the varying position of the obstruction, as experienced by the patient and as detected by the sound. The site of the spasm may vary during a single examination. The spasm yields in almost every case to firm equable pressure and always relaxes under anæsthesia. The sound can often be withdrawn without encountering any obstruction on its return journey. The affection is troublesome rather than dangerous, and a high degree of emaciation is but rarely met with. The passage of a full-sized bougie, which may be repeated two or three times a week, is often curative. The neurotic state of the patient requires treatment, and must never be neglected. The drugs most generally useful are the bromides and belladonna, and, in markedly hysterical girls, valerian.

14. **Paralysis.**—Paralysis is but rarely met with. It occurs in diseases of the central nervous system, especially in bulbar paralysis. It may, however, be a result of peripheral neuritis as in diphtheritic paralysis and plumbism. Though sometimes stated to be of hysterical origin, it is very infrequently met with. In hysteria spasmodic stricture is much more likely to occur than paralysis. The prognosis and treatment depend on the cause. Faradic stimulation of the œsophagus has been recommended. In general the employment of the stomach-tube is necessary in order to feed the patient.

15. **Malformations.**—Pressure-diverticula (*Pharyngocœles*) are described in the article on the PHARYNX.

Congenital Malformations.—*Dilatation* has been already described. A *membranous stricture* is very rarely found in the young infant at the junction of the upper fourth with the lower three-fourths of the gullet. Infants have been born with a fistula connecting the œsophagus and the trachea. Death always supervenes in a few days or weeks from the entrance of milk into the respiratory passages and the supervention of septic broncho-pneumonia. An *imperforate œsophagus* is commonly due to the absence of one of the segments of the tube. It is excessively rare. The attempt to take the breast is followed immediately by a suffocative attack owing to the food being unable to enter the stomach, and therefore finding its way into the air-passages. Gastrostomy offers the only chance of saving life; but no successful case has been recorded.

Acquired Malformations.—*Traction-diverticula* result from inflammatory processes near the œsophagus, especially when these end in suppuration, and the subsequent shrinkage of the evacuated abscess-cavity. In these cases a part of the abscess-wall becomes adherent to the wall of the œsophagus, which is thus drawn out as a diverticulum. These traction-diverticula are commonly found about the bifurcation of the trachea, in which case they originate in inflammation of the lymphatic glands, and are seen springing from the anterior wall of the gullet. More rarely they are placed laterally. They tend to form pockets in which food collects and undergoes decomposition and introduce a serious fallacy; bougies introduced into the œsophagus are apt to get caught in a diverticulum. These traction-diverticula are not to be confounded with pressure-diverticula (*Pharyngocœles*). See PHARYNX, Diseases of.

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ŒSTRUS (ὄϊστρος, a gadfly).—SYNON. : Fr. *Œstre*; Ger. *Bremse*.—A genus of dipterous insects, called gadflies, the larvæ of which, vulgarly known as maggots or bots, live parasitically in man and animals. The ordinary human bot, *Œstrus hominis*, is of rare occurrence in England, but is not infrequently met with in warm countries, especially in South America. The larva of the gadfly of the ox, *Œstrus bovis*, also occasionally attacks man.

True maggots and other bot-like larvæ are occasionally encountered in medical practice. Livingstone, when in Africa, was attacked in the leg by a small bot-like larva, which was removed, and is now preserved in the museum of the Royal College of Surgeons of England.

OEYNSHAUSEN, or REHME, in Germany.

Gaseous thermal salt waters, closely resembling those of Nauheim. See MINERAL WATERS.

OÏDIUM ALBICANS.—SYNON. : *Saccharomyces mycoderma*; *Mycoderma vini* (Grawitz).—A vegetable parasite, associated with thrush. See MOUTH, Diseases of (p. 1030).

OINOMANIA (οἶνος, wine; and μανία, madness).—A synonym for dipsomania. See DIPSO-MANIA.

OLD AGE, Signs of.—See SENILITY.

OLFACTORY SENSE, Morbid Conditions of.—The principal morbid conditions that occur in connection with the sense of smell are the following: (1) Hyperæsthesia; (2) Subjective Sensations of Smell; (3) Perversion of the Sense of Smell; and (4) Olfactory Anæsthesia.

1. **Olfactory Hyperæsthesia.**—SYNON. : Hyperosmia.—This condition is seen in the increased nervous sensibility which results from chronic debilitating illness. It occurs also in hysteria, in which remarkable, almost animal, acuteness of the sense is sometimes present, so that not only objects but persons have been discriminated by this means. In insanity the same condition is sometimes met with. A less degree of olfactory hyperæsthesia is occasionally met with in epileptics and persons suffering from myxœdema. It is usually associated with, and has to be distinguished from, an altered appreciation of odours, shown in the abnormal enjoyment of or disgust at the odours which are recognised with natural or preternatural acuteness. The condition is probably of central origin and rarely calls for special treatment.

2. **Subjective Sensations of Smell.**—Subjective sensations of smell occur from central disease, or from irritation of the nerve of smell. In the insane, olfactory hallucinations occur, though less commonly than those of the visual or auditory sense. Schlager met with them in five cases out of six hundred. In epilepsy subjective sensations of smell occur as occasional prodromata of fits, and the disease in these cases probably involves the olfactory centre in the anterior part of the temporal-sphenoidal lobe. It was so in a case of tumour recorded by Sander. Irritation of the nerve from meningeal disease or injury also, in rare cases, causes olfactory hyperæsthesia. Persistent disagreeable smells are occasionally complained of after influenza.

3. **Perversion of the Sense of Smell.** SYNON. : Parosmia.—This is a rare condition which

occasionally results from irritation of the nerve or central organ. In a case recorded by Legg, some time after an injury to the head, all substances 'tasted' of gas or paraffin, and there was marked diminution in the acuteness of the sense of smell.

4. **Olfactory Anæsthesia.** — **SYNON.** : Anosmia.

DEFINITION.—Loss or diminution of the sense of smell.

ÆTIOLOGY.—The causes may be local changes in the organ of smell; disease of the nerve; or disease of the centre.

(a) Among *local* causes may be mentioned the following: (1) Acute and chronic catarrh of the olfactory mucous membrane, the latter causing thickening—a condition sometimes produced by excessive snuff-taking. A large proportion of the cases follow severe and prolonged catarrh, and are due purely to the local effect on the tissues through which the olfactory nerve-endings are stimulated. (2) Dryness of the mucous membrane, as in cases of destruction of the external nose (Notta), or in paralysis of the fifth nerve. (3) Occlusion of the passage by polypus, preventing the access of air to the olfactory region. (4) Impaired access of air consequent on facial paralysis. The loss of the power of dilating and keeping expanded the nostril prevents a due quantity of air being drawn through the nasal passage; and, moreover, the loss of power of compressing the nostril in 'sniffing' prevents the air being directed into the olfactory region. (5) In rare cases loss of pigment in the nose, consequent on general loss of pigment, has appeared the cause of loss of smell.

(b) *Damage to the olfactory nerve* may result from injury or disease. It is not an uncommon result of blows or falls upon the head, and it is probable that in these cases the delicate olfactory nerves are lacerated as they pass through the bone, or may even be torn from the bulb (*see* NOSE, Diseases of). The bulb, or tract, may also suffer in adjacent disease, as tumour, abscess, caries of the bone, and meningeal changes, especially syphilitic. Spontaneous atrophy of the olfactory bulbs occasionally occurs in old age, and has been met with in younger persons, and (rarely) in *tabes dorsalis*. Excessive stimulation of the nerve by a very powerful odour has been followed by anosmia.

(c) In *cerebral disease* the sense of smell is sometimes lost. It may be impaired in so-called functional disease, as in hysteria, and in degenerative disease, as paralytic dementia. It is occasionally lost in organic disease involving the roots of the olfactory nerve. Unilateral anosmia has been met with in cases of aphasia (Hughlings Jackson), an association which is explained by the passage of the external root of the olfactory nerve past the island of Reil to the anterior part of the temporo-sphenoidal lobe. Together with the other special senses, loss of smell has been observed on the side opposite to a lesion near the posterior extremity of the internal capsule in the opposite hemisphere.

It is to be remembered that the olfactory nerves are sometimes congenitally absent.

SYMPTOMS.—The evidence of anosmia is the loss of the perception of odours and flavours—the former term being applied to the sensation when its cause enters by the anterior nares, the latter when it enters by the posterior nares and the sensation is blended with that of taste. It is generally imagined that flavours are tasted, and hence those who are suffer-

ing from anosmia are said to have lost smell and taste. The loss of the olfactory sensation may be partial or complete, according to the extent of involvement of the nerves. In some cases, especially of traumatic origin, the ability to perceive one or two odours may be unimpaired, no others being recognised. It may be lost on both sides, when due to degenerative changes; or on one side only, from local changes or injury or brain-disease. When due to organic brain-disease, it is generally lost on the side of the cerebral lesion. Unilateral loss, in hysterical hemianæsthesia, occurs on the side opposite to the hemisphere which is in a state of partial inhibition; and a similar loss, with that of the other special senses, has been met with in rare cases of organic disease.

DIAGNOSIS.—The diagnosis presents little difficulty. In examination, care must be taken to employ only substances—as aromatic oils, &c.—which affect the olfactory nerve, and not acrid substances, as ammonia and acetic acid, which stimulate also the fifth nerve.

PROGNOSIS.—The prognosis in anosmia is favourable when it is due to a local cause which has existed but a short time, or in which its cause (such as obstruction from polypus) can be removed. Prolonged changes in the mucous membrane of the nose seldom pass away sufficiently to permit restoration of smell; and whenever there is reason to suspect injury or disease of the olfactory nerve or centre, recovery is very improbable. Hence traumatic loss of smell, which has continued for some months, will certainly persist.

TREATMENT.—Anosmia, as a symptom, rarely calls for treatment, which should be directed to its cause. Sometimes local stimulation is of service; and occasionally counter-irritation, by blisters to the neck, has appeared to assist recovery. In hysterical cases faradisation of the nasal mucous membrane has been recommended; but the olfactory nerve itself is not accessible to electrical stimulation. The chief therapeutical measures are those for the treatment of the nasal mucous membrane. *See* NOSE, Diseases of.

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OLIGÆMIA (ὀλίγος, small; and αἷμα, blood).—Deficiency of the total amount of blood in the body. *See* BLOOD, Morbid Conditions of.

OMENTUM, Diseases of.—*See* PERITONEUM, Diseases of.

ONANISM (Onan).—A synonym for masturbation. *See* MASTURBATION.

ONYCHAUXIS (ὄνυξ, a nail; and αὐξάνω, to increase).—Hypertrophy of the substance of the nails. *See* NAILS, Diseases of.

ONYCHIA (ὄνυξ, the nail).—An inflammatory affection of the matrix of the nail. *See* NAILS, Diseases of.

ONYCHOGRYPHOSIS (ὄνυξ, the nail; and γρυπός, curved).—Curvature or deformity of the nails. *See* NAILS, Diseases of.

ONYCHOMYCOSIS (ὄνυξ, the nail; and μύκης, a fungus).—Parasitic disease of the nails. *See* NAILS, Diseases of.

ONYX.—A collection of pus at the bottom of the anterior chamber of the eye. *See* EYE, Diseases of (p. 505).

OPHTHALMIA (ὀφθαλμός, the eye).—**SYNON.** : Fr. *Ophthalmie*; Ger. *Ophthalmia*.—A term restricted by custom to inflammation originating in the superficial structures of the eye, such as the varieties of conjunctivitis, or the phlyctenulæ which sometimes appear upon the cornea and may give rise to shallow ulcers. See EYE, Diseases of.

OPHTHALMITIS (ὀφθαλμίτις, the eye).—**SYNON.** : Panophthalmitis; Fr. *Ophthalmite*; Ger. *Augenentzündung*.—A term which has been used to express inflammation affecting the whole of the structures of the eyeball. See EYE, Diseases of.

OPHTHALMOMALACIA (ὀφθαλμός, eye; μαλακία, softness).—A term recently applied to a diminution of ocular tension appearing spontaneously. The aetiology of the condition is unknown, though there may be a history of traumatism. Neuralgic pains, dilatation of the pupil, and photophobia may co-exist.

OPHTHALMOSCOPE (ὀφθαλμός, the eye; and σκοπέω, I examine).—**SYNON.** : Fr. *Ophthalmoscope*; Ger. *Augenspiegel*.

The ophthalmoscope is an instrument for lighting up the interior of the eye in such a manner as to render the contained structures clearly visible.

DESCRIPTION.—The first ophthalmoscope was invented about 1847, by the late Mr. Charles Babbage, who laid it aside because an ophthalmic surgeon to whom he showed it, and who failed to perceive its probable utility, afforded him no encouragement. In 1851 another form of the instrument was invented by Helmholtz; but, in 1852, Babbage's original form was re-invented by Ruete, and this, with a few unimportant modifications, has ever since held its ground in practice. It consists, essentially, of a slightly concave mirror, with a small central perforation; or, if the mirror be of silvered glass, with the silvering removed from a small circle in the centre. A mirror the size of a shilling is large enough for most practical purposes, and a central aperture of not more than 2 or 3 millimetres in diameter is better than a larger one. The mirror may be attached to a handle of any proportions preferred by the owner, or may be left without one; but it must be accompanied by certain auxiliary convex and concave lenses, the uses of which will be presently explained. The focal length of the mirror is usually about eight inches.

METHOD OF USE.—In order to learn the use of the ophthalmoscope, the beginner will do well to avail himself of a contrivance called Perrin's artificial eye, or of one of the more elaborate forms of it designed by Landolt and by Frost. The instrument essentially consists of a small hollow sphere of metal, to represent the eye, closed in front by a lens, which can be changed at pleasure, and behind by a door for the insertion of pictures of various healthy and diseased conditions of the retina. When an artificial eye is not available, the learner should take the patient into an obscurely lighted room, and should stand or sit facing him, with the two heads upon the same level. A gas- or oil-flame—preferably, from its greater steadiness and superior illumination, that of an argand burner—is then placed upon the same level as the eye which is to be examined, on the same side of the head, and a little behind it, so that no direct light shall fall upon

the cornea. The observer, commencing with his face exactly opposite that of the patient, and about eighteen inches distant from it, places the back of the ophthalmoscopic mirror against his eye, using preferably that which is opposite to the eye to be examined, the right eye for the patient's left, and *vice versa*. The patient is directed to look as if at a distant object, over the shoulder of the observer which is most remote from the eye under inspection, thus looking over the observer's right shoulder when the right eye is being examined. In this position the observed eye is turned a little towards the nose; and the optic nerve-entrance, which is somewhat on the nasal side of the posterior pole, is brought opposite the pupil. Looking through the mirror-aperture, the observer directs the light of the flame, reflected from the polished surface, in such a manner that it falls into the pupil of the observed eye; and this light, returning from the eye, reaches him through the perforation. It exhibits the cavity of the eye illuminated, but, as a rule, shows no objects, but only the pupil as a reddish or yellowish circle. In order to see the contained structures, two methods are employed, the *indirect* and the *direct*; the former of which gives the better general view of the fundus, the latter the greater facilities for studying the condition of single points on the nerve or on the retinal surface. It is therefore necessary to be conversant with both, and to use one or both as circumstances may require.

Indirect Method.—In using the indirect method, the observer takes a biconvex lens, of about two inches focal length, and holds it with his free hand in the track of the returning light, and at about two inches from the eye of the patient. The rays of light, thus rendered convergent, become united into an aerial inverted image of the fundus of the eye, which image, and not the fundus itself, will be the object of vision to the observer. The position of the image is in the focal plane of the lens, on the side next the spectator; and, in order to see it clearly, nothing is necessary but to have the observing eye in the track of the returning rays, and at the right distance from the image; which, it must be remembered, with a two-inch lens, will be four inches or more nearer to the observer than the eye of the patient. The whole art of using the ophthalmoscope for the indirect method may be said to consist in moving the eye to and fro upon the line of sight until the right distance is attained, without moving it laterally so as to get out of the track of the rays, and without losing the illumination. As soon as a vessel, or any other defined object, is seen, the observer knows that his distance is correct, and he then causes the patient to change the direction of his eye until every part of its fundus has come successively into view. The image, it must be remembered, is inverted in every particular; its nasal side representing the temporal side of the retina, and its upper portion the lower portion of the retina. In first attempts to use the ophthalmoscope it is desirable to have the pupil of the observed eye dilated by atropine or duboisine, but, after dexterity has been attained, the dilatation may in most cases be omitted. The details of the retinal image are sometimes more or less obscured by an image or images of the lamp-flame; of which there may be two, one formed by the anterior and one by the posterior surface of the lens. These images are only sources of embarrassment when the lens is held vertically, and may be displaced and put out of

sight by giving it a small degree of obliquity. A bright image of the mirror itself upon the cornea, showing the central perforation as a dark spot, is sometimes troublesome to beginners; and it is said that this image has even been mistaken for that of the optic nerve. The blackness and sharp definition of the perforation should render such a mistake impossible; and the image may readily be displaced by a slight alteration of the angle at which the mirror is held.

In order to magnify the inverted image, and to increase its brightness by bringing the mirror nearer to the eye of the patient, a convex lens may be placed behind the mirror for the observer to look through. Something of this kind is always necessary for observers who have reached the period of life at which spectacles are required for reading; and it is advantageous to all persons. The writer's practice is to use a lens of about seven inches focal length in this manner; and there is thus obtained an image which for many purposes is as good as that afforded by the direct method. With such a lens the eye of the observer can be only seven inches from the image, and, as this will be formed four inches in front of the eye of the patient, it follows that the two faces will be only eleven inches apart. At this comparatively small distance, the illumination of the fundus of the observed eye, which is afforded by a good mirror, is exceedingly satisfactory.

Direct Method.—In the direct method, the observer does not apply any intervening glass between the mirror and the eye of the patient, but comes as close to the latter as possible, and looks, not at an aerial optical image, but at the actual fundus itself, magnified by its own crystalline lens. It is only when the eyes of both observer and patient are of normal refraction, or emmetropic, that this can be done without the aid of a lens, which when required is most conveniently placed behind the mirror. The lens employed for this purpose must be such as to correct the sum of the error of refraction of both the eyes; and must therefore be concave when this error is on the side of myopia, convex when it is on the side of hypermetropia. An observer who is short-sighted will begin his investigation with a concave lens behind his mirror, which corrects his own short sight; and he will add to or diminish the power of this lens to meet any degree of ametropia which the observed eye may present in addition to, or in diminution of, his own. In order to facilitate the required changes, all necessary lenses are now usually mounted upon a revolving disc placed behind the mirror, and so arranged that each one of them can be brought in turn before the aperture. In one of the best of the modern forms of instrument, that of Loring of New York, the mirror itself is made to turn upon pivots in a vertical line independently of the disc of lenses, so that the correcting lens receives no obliquity from the position of the mirror. This contrivance is valuable in some cases, especially when a correcting lens of high power is required, because such a lens, if held obliquely, is liable to produce some distortion of the objects seen through it. In using the revolving disc, a normal-sighted observer commences with no lens behind the aperture; and, if he then obtains clear definition, he knows that the eye into which he is looking is normal-sighted also, or at most is only in a slight degree hypermetropic. If, on the contrary, he does not obtain a clear image, he knows

that the eye into which he is looking, unless the transparency of its media be impaired, is not normal-sighted, but that it is either myopic, or hypermetropic in a somewhat high degree. Keeping the fundus in view, he causes the disc to revolve until a lens comes over the aperture which renders the picture distinct; and he has then only to see the number and kind of the lens in order to know the degree as well as the nature of the defect of refraction. In many cases it is even possible to prescribe spectacles, as the result of such an examination, with a very fair degree of correctness and success. But the chief use of the direct method, especially in the applications of the ophthalmoscope as an instrument of diagnosis in general medicine, is to scrutinise, as already stated, some portion of the fundus of the eye which has been shown, by the indirect method, to require more minute examination than that method will itself permit the observer to accomplish.

OPHTHALMOSCOPIC APPEARANCES.—In order to interpret ophthalmoscopic appearances, and to distinguish physiological variations from pathological changes, it is before all things necessary to bear in mind the anatomy of the structures which are, or may be, rendered visible, and the relations which they bear to each other. The fundus of the eye is composed of several layers, the more anterior of which commonly conceal the posterior; and conceal them in such a manner that, when the former are rendered more transparent by malformation or disease, the latter are brought into view.

1. *Sclerotic.*—Commencing with the posterior layer, it consists of the inner surface of the sclerotic, a smooth and shining white surface, which is ordinarily entirely concealed by the pigmentation of the choroid and of the posterior or epithelial layer of the retina. The sclerotic is naturally visible, as a general white background to a vascular network, in cases of albinism, in which the natural pigment of the eye is congenitally absent, or in some very fair persons, who are not albinos, but whose eyes are very sparingly pigmented. It is rendered visible in patches, as a result of malformation or disease, in cases in which it is exposed by a fissure through the choroid, such as generally accompanies *coloboma iridis*; in cases in which the choroid has suffered atrophy as a result of antecedent hæmorrhage or inflammation; and in the immediate neighbourhood of the optic discs, in the so-called crescents of choroidal atrophy which are so often associated with high degrees of myopia. The whiteness of an exposed sclerotic may be distinguished from that of an opaque white deposit in the choroid or in the retina, by many small physical characters, such as the relation of the borders of the whiteness to the neighbouring tissues and vessels, which will show the one to be the result of the removal, the other of the addition, of material. The most conspicuous white deposits are those associated with albuminuria or diabetes mellitus, with syphilitic retinitis, and with the first stages of retinal glioma. In all these the deposits manifestly cover and conceal vessels, which may be seen to emerge from beneath them; while in complete atrophy of portions of choroid, it is not uncommon to see a few remains of dwindled vessels, and other shreds of choroidal tissue, rendered unusually conspicuous by their white background, and manifestly situated in a plane anterior to it.

2. *Choroid.*—The next layer from behind forwards is the choroid, which is essentially a vascular

network, containing more or less pigment in the intervals between the vessels. In very fair eyes, as already mentioned, the choroid may allow the general whiteness of the sclerotic to shine through; but, in the great majority of cases, it conceals the latter entirely. In like manner, the actual structure of the choroid is itself usually concealed by the pigment in the epithelial layer of the retina; and the choroid generally only plays the part of a red background, varying up to dark chocolate colour in very dark eyes, and exhibiting neither structure nor vessels. When the retinal epithelium is scantily pigmented, as occurs in light eyes, the larger choroidal vessels may be seen through the retina; and they are readily distinguished from those proper to this structure by their different arrangement; the vessels of the retina being arborescent, while those of the choroid are either nearly parallel to one another, or arranged in more or less diamond-shaped reticulations. When both sets are visible together, moreover, the vessels of the retina will be clearly seen to be in a plane anterior to that of the vessels of the choroid, and a variety of minute differences of colour and aspect will suffice to show that the two sets form parts of different circulatory systems.

3. *Retina*.—The retina itself is formed of several layers, the deepest of which contains the perceptive elements, or the rods and cones of the so-called Jacob's membrane. In front of the perceptive elements there are ganglionic and granular layers, subservient to the functions or to the nutrition of the rods and cones; and, in front of these again, a layer of connective tissue, containing and supporting the conducting fibres, which are ultimately massed together in the trunk of the optic nerve, and which convey impressions from the retina to the brain. The fibre-layer and its connective tissue are necessarily thickest in the immediate neighbourhood of the optic nerve, and they thin off towards the peripheral parts of the retina; while all but the perceptive elements are wholly wanting over a small circle or depression at the posterior axis of the eyeball, a little to the outer side of the nerve, and known as the 'yellow spot,' with its *fovea centralis*. The central artery of the retina enters the eye in the trunk of the optic nerve, and the central vein emerges in the same manner, the circulation between the two being almost a closed one, save for a few very small and insignificant anastomoses of the terminal vessels, some at the nerve-entrance itself, others in the ciliary region. The retinal blood-vessels are chiefly lodged in the connective tissue of the fibre layer, and only small twigs dip down into the deeper retinal tissues. The arteries and arterioles divide, and the veinlets and veins unite, in an arborescent fashion; and the two sets of vessels are readily distinguished apart by the larger calibre and deeper colour of those which carry venous blood. At the nerve-entrance, both sets bend at a right angle, or nearly so, in order to pass from the axis of the nerve-trunk into the plane of the retina, or *vice versa*.

Between the rods and cones of Jacob's membrane, and the anterior or capillary layer of the choroid, there is a sheet of pavement-epithelium, the cells of which contain a larger or smaller quantity of pigment. This epithelial layer was at one time regarded as part of the choroid, but more recent histologists refer it to the retina. When full of pigment, it forms an opaque screen, by

which the choroid is concealed from view, and against which the delicate retinal structures, especially near the nerve, may become apparent as a thin, almost pellucid, film, in which blood-vessels ramify. In the eyes of fair people, with only scanty pigmentation, the epithelium neither completely conceals the choroid, nor does it throw up the retina with anything like the same distinctness, so that the retinal blood-vessels are clearly seen, but not the structure which supports them. When the pavement-epithelium has been removed, either by disease or by senile changes, the choroidal tissues become conspicuous.

4. *Optic Nerve*.—The general aspect of the optic nerve varies greatly, within limits defined by differences in the degree of its capillary vascularity, by the effects of contrast arising from the degree of pigmentation of the surrounding parts, and by the mechanical arrangement of the structures of which it is composed. The aperture in the sclerotic, by which the nerve enters the eye, is closed by a cribriform plate of condensed connective tissue, the *lamina cribrosa*; and the fibres normally leave their sheaths on the outer side of this lamina, only the axis-cylinders passing through its perforations. The combined axis-cylinders constitute a mass; the whiteness of which is subdued rather than glistening, and which derives a certain amount of reddish, roseate, or pink colour from the capillary vessels by which it is permeated. The axis-cylinders, like the vessels, bend round as they pass from their original direction into that of the retinal surface; and, in the majority of instances, they leave a central depression in the nerve-disc as they separate, a depression at the bottom of which the glistening whiteness of the *lamina cribrosa* is visible, and which has been called the *porus opticus*. In other instances, this central depression does not exist, but the axis-cylinders are gathered chiefly towards one side of the nerve-entrance, and the lamina is visible laterally instead of centrally. The size of the *porus opticus* is very variable, inasmuch that sometimes, when it constitutes quite a large central depression, it is described as congenital or physiological excavation of the nerve. This congenital excavation is always readily distinguishable from the excavation produced by the pressure consequent upon excess of internal tension; because the former never, and the latter always, extends to the extreme margin of the nerve. In other words, the congenital excavation, however large and remarkable, is always surrounded by a ring of nerve-tissue; while the morbid excavation always extends to the margin of the opening in the sclerotic. The position of the blood-vessels in the nerve-entrance is also another variable factor, since they are sometimes nearly central, and at others are seen to pass into or out of the nerve-tissue close to its margin. In a few cases, moreover, the axis-cylinders at some portion or portions of the circumference carry their sheaths for a short distance into the retina; and the nerve is then surrounded by white glistening processes, with brush-like terminations. Sometimes, again, the margin of the opening in the choroid is richly pigmented, and the nerve is surrounded with a ring, or bordered by a crescent, of chocolate or black colour.

5. *Fundus as a Whole*.—The general appearance of the healthy fundus oculi may be summed up somewhat in the following way: The background seen in the inverted image ranges in colour from an

almost chocolate tint in very dark people or in the dark races, to a closely woven reticulation of vessels carrying red blood, and affording indications of the white sclerotic lying behind them. In light eyes, the retina itself is invisible; but in dark eyes its thickest portion appears as a delicate film, which has been compared to moistened tissue-paper, over the portion of the field which immediately surrounds the optic nerve. Except in very light eyes, the vessels of the choroid are not individually visible, being concealed by the pigmentation of the pavement-epithelium; and, when visible, they are distinguishable by their parallel direction, and by the absence of branches. The vessels of the retina are always clearly visible, and may be traced along their numerous arborescent ramifications to twigs of extreme fineness. The arteries are smaller and brighter than the veins, and often present the appearance of a white line running along the axis of the vessel, almost as if it were a translucent red tube, carrying a white fluid. The veins, larger and darker than the arteries, seldom display the white line. The vessels pass off the optic disc on all sides, but make bold curves which carry them clear of the region of the yellow spot. In the close vicinity of the disc, the vessels are sometimes attended by fine white threads, pursuing the same general course with them, which are apparently coarser portions of the connective tissue by which they are sustained. The optic disc, or termination of the optic nerve itself, the most conspicuous object in the ophthalmoscopic image, stands out boldly against its surroundings, and presents a general colour-effect which depends partly upon the richness of its capillary blood-supply, and partly upon the greater or less degree of pigmentation of the tissues around it. Over part of its surface, generally in or near the centre, but sometimes laterally, it displays the whiter colour of the lamina cribrosa, and the mottling of its perforations for the passage of the nerve-fibres. It is often bordered, either entirely or partially, by a line of dark pigment situated at the margin of the choroidal opening; and it often exhibits also a fine white line at its margin, which is the edge of the opening in the sclerotic, seen through the semi-transparent nerve-tissue. The vessels pass over its margin without deviation or change of plane. The apparent size and shape of the disc depend much upon the refraction of the eye. As seen in the inverted image, it appears comparatively small in a myopic eye, and large in a hypermetropic; while, in cases of astigmatism, it is distorted into the appearance of an oval. In the same way, the refraction modifies the apparent actual, but not the relative calibre of the vessels. In the myopic eye the vessels appear of small diameter, and in the hypermetropic they appear of large diameter; so that no conclusions about their actual size can be drawn until the state of refraction has been taken into account. The fact that the veins are relatively larger or smaller than usual, when compared with the arteries, is, of course, not influenced by refraction, except that, in a hypermetropic eye, such a difference would be more conspicuous than in a myopic, by reason of the more magnified image produced by the optical conditions of the media.

6. *Circulation*.—In a general way, the blood-currents in the vessels of the retina are continuous and uninterrupted; but any hindrance to the entrance of blood may be attended by pulsation, first in the veins and subsequently in the arteries. Such

hindrance may arise from disordered action of the heart, as in cases of insufficiency of the aortic valves; from disease of the coats of the arteries; or from increased resistance on the part of the fluids already occupying the cavity of the eyeball. The venous pulse depends upon an arrest of the outflow through the veins by the pressure of the entering arterial current, which, at the acme of the pulse-wave, has force enough to push back the venous current when there is not room enough for both. Hence, in the venous pulse, the vessels empty themselves in a direction from the centre of the disc towards its periphery, and refill in the opposite direction. The ordinary cause of venous pulse is increased tension or fulness within the eyeball, so that it is among the early symptoms of glaucoma; but it is also to be seen in a small proportion of cases in which no excess of tension is to be discovered either by touch or by symptoms, and in which the eyes appear to be healthy. In the arterial pulse, the resistance to the entrance of blood, or rather the disturbance of the balance between the propulsive and the resisting forces, must be considerable; and the course of events is that the arterial current can only make its way into the eye at the acme of the pulse-wave, during which the arteries fill from the centre of the disc to its periphery, to collapse again as soon as the impulse of the systole diminishes. In such a condition, the impediment to the entrance of arterial blood is sufficient to imperil the nutrition of the nerve-tissue; and the writer has seen at least one case of partial nerve-atrophy, attended by arterial pulsation, for which no other cause than excessive arterial tension could be assigned. Arterial pulse is probably always present in advanced stages of glaucoma; but by the time it is produced, the fundus is usually obscured or rendered invisible by other changes. Apart from glaucoma, its most frequent cause is aortic regurgitation; and in this form the eye does not suffer, except together with other parts of the organism.

7. *Optic Neuritis and Atrophy*.—The morbid appearances seen with the ophthalmoscope, and interesting to the physician, are chiefly those which point to the existence of some diathesis, or to the presence of disease in other organs. Swelling of the intra-ocular extremity of the optic nerve, with obliteration of its margins and obstruction to its vessels, occurs in many forms of intracranial disease, especially in connection with intracranial tumour, and is often followed by atrophy and blindness when life is sufficiently prolonged. The most interesting characteristic of these cases is that, since the swelling affects only the connective-tissue layer, which is absent over the region of the yellow spot, there is commonly no diminution of the acuteness of central vision until the atrophic changes have commenced; by which time, in many instances, the primary swelling has passed away. Hence, for many years, there existed great uncertainty about the cause of the atrophy, and this uncertainty was only removed when physicians began to examine the fundus oculi in all cerebral cases, without regard to the state of sight. Prior to that time, the intra-ocular changes were apt to remain undiscovered in their primary stage, and until commencing impairment of vision produced resort to an ophthalmologist, followed by an ophthalmoscopic examination in due course; and then the atrophy was often attributed to many fanciful causes, among which the smoking of tobacco held a prominent

place. It is not necessary to assume that tobacco is never injurious to the optic nerves, in order to be quite sure that the majority of the instances of atrophy once attributed to its influence were, in reality, due to a totally different cause. The changes associated with intracranial diseases will be found described in a special article. See OPHTHALMOSCOPE IN MEDICINE.

8. '*Albuminuric Retinitis*.'—Very frequently in albuminuria, and occasionally in diabetes mellitus, the fundus of the eye becomes studded over with spots or patches of a glistening white colour, which are probably due to fatty degeneration of the connective tissue of the retina, and which are often associated with scattered hæmorrhages. The blood, in these instances, is usually effused into the fibre-layer, and, following the course of the fibres, becomes spread out into somewhat striated spots, with brush-like terminations. Every case in which either the white patches or the hæmorrhages, or both, are detected by the ophthalmoscope, whether with or without impairment of sight, calls for a careful examination of the urine, and renders it proper to follow mainly the indications of treatment which such an examination may afford. See PLATE IX., fig. 1.

9. *Hæmorrhages*.—Without the white patches, hæmorrhages may occur in the retina under various conditions. Sometimes they are distinctly arterial, in which case they are generally small in absolute amount, and may often be traced to some manifest point of rupture in the vessel from which they have occurred. These hæmorrhages seldom produce extreme impairment of vision, although they are usually discovered on account of some degree of impairment; and their chief importance is derived from the warning they may give of a state of brittleness of the arteries, and of a consequent liability to similar bleedings elsewhere, as in the brain. They call for all the precautions which such a state would suggest, as for the consumption of a diminished quantity of fluid, and for the avoidance of constipation and of all violent bodily efforts.

Hæmorrhages which are distinctly venous occur not infrequently in connection with the disturbances of circulation which are incidental to the cessation of the menstrual function, or to the irregularities by which cessation is preceded. The blood may proceed from comparatively large veins, in which case it often forms a layer immediately beneath the mem-

brana limitans of the retina, causing great temporary impairment of sight, or even total blindness; and yet, in many cases, being quickly absorbed without permanent injury. In other instances it may proceed from smaller and deep-lying veinlets, in which case the effusion will usually be situated in the fibre-layer, and will be moulded, so to speak, by the fibres, into what have been described as 'flame-shaped' hæmorrhages (p. 56). These are generally multiple, and usually cause an impairment of function, which is decided although not total, and is often permanent. The flame-shaped hæmorrhages are said by Hutchinson to occur most frequently in persons of gouty diathesis, and he holds the same doctrine with regard to a less common form, of which some remarkable examples have been observed by himself, and by Eales of Birmingham. In these cases, the subjects were young males, of constipated habit, and in many instances of gouty family history. The bleedings were large in amount, so as to penetrate into the vitreous body and to cause for a time total loss of sight, and were frequently recurrent. To what extent they were due to deficient plasticity of the blood, to abnormal friability of the vessels, to variations in vaso-motor tension, or to the withdrawal of external support from the vessels by diminished tension within the eyeball itself, is at present a matter of conjecture. It is obvious that the treatment of such cases, and of retinal hæmorrhages generally, must resolve itself into that of the constitutional conditions with which they are associated. The only special indications, as regards the eye, will be the enforcement of functional rest, and the maintenance of an elevated position of the head during sleep. In cases connected with the cessation of the menstrual function, the absorption of the effused blood often appears to be promoted by the careful administration of iodide of potassium, which should usually be combined with ammonio-citrate of iron, or with some other suitable tonic, and care should always be taken to maintain a moderately relaxed condition of the bowels. Even apart from the injurious effects likely to be produced by straining, constipation appears to predispose to hæmorrhage.

10. *Embolism of the Central Artery*.—Sudden loss of vision is sometimes occasioned by the plugging of the central retinal artery by an embolus. This is especially to be suspected in cases of known valvular disease of the heart, and the condition is

FIRST PLATE (DIVIDED INTO SIX).

1. *Normal Optic Disc*. The edge is distinct all round. To the outer side of the entrance of the central vessels is a roundish pale area (physiological pit) with a dotted appearance on its floor (lamina cribrosa). Left eye: erect image.

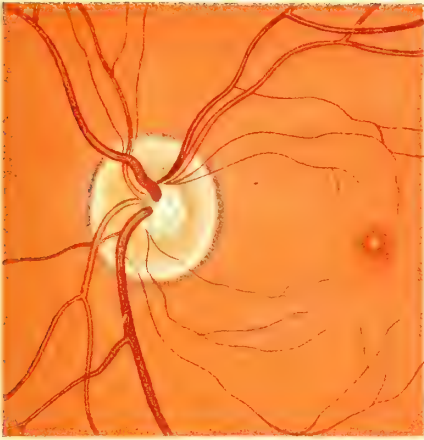
2. *Slight Optic Neuritis*. The edge of the disc is concealed above, and down and in; it is visible, but blurred, elsewhere. The physiological pit is indistinct. Veins somewhat distended. Outside the disc are several retinal hæmorrhages (round and punctate). The drawing is from a case of renal disease, and the degenerated arteries are shown to indent the underlying veins where they cross over them. Left eye: erect image.

3. *Gross Optic Neuritis*. The edge of the disc is quite invisible everywhere. The disc is evidently raised above the level of the adjacent retina, as is indicated by the curve of the vessels. The colour of the nerve is changed, being redder than normal, particularly at its periphery, where numerous red streaks radiate outwards (dilated small vessels lying between the nerve-fibre bundles). The physiological pit is filled in by opaque inflammatory exudation and swollen nerve-fibres, and this opaque material also obscures the arteries in places. The veins are distended and very tortuous. From a case of cerebral tumour. Right eye: erect image.

4. *Optic Neuritis in stage of subsidence, with commencing Atrophy*. The edge of the disc is still concealed except outwards, where it is just beginning to come again into view. In general colour the disc is markedly paler than normal, with some opaque tissue (organised exudation) filling up the physiological pit and obscuring the central vessels at their entrance. The veins are still darker than normal and tortuous. Right eye: erect image.

5. *Optic Atrophy consecutive to Neuritis*. There is still a little blurring of the edges above and below, which will probably disappear later. Elsewhere the edge is distinct, and shows pigmentary change in the adjacent retina. The disc is white almost everywhere, and the lamina cribrosa is concealed. The retinal arteries are becoming reduced in calibre: the veins will also later become smaller. Left eye: inverted image.

6. *Simple Optic Atrophy*. The edge of the disc is sharply defined. In colour, the disc is pale bluish-grey, with distinct white physiological pit. The stippled appearance of the lamina cribrosa is visible over a large area. The central vessels are somewhat reduced in calibre, and the veins show no trace of any increased tortuosity. Left eye: erect image.



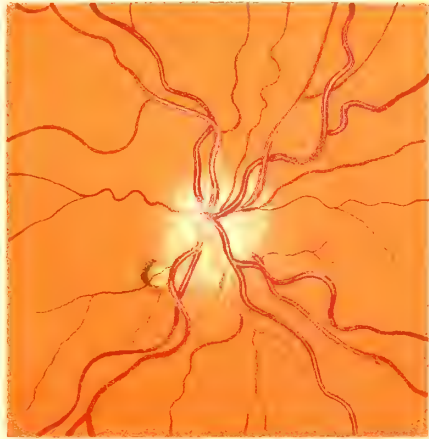
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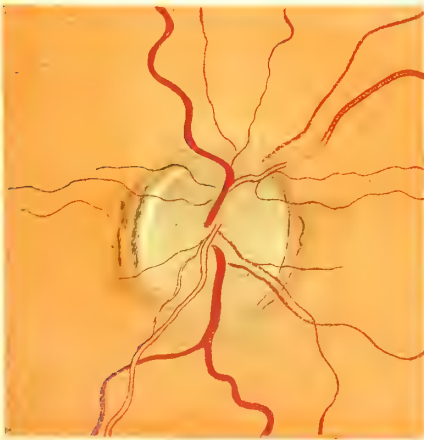
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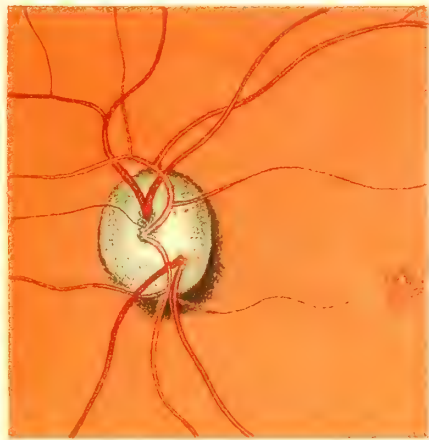
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Commonly bilateral, it is in rare cases unilateral, and is then more common in the eye opposite to the seat of the tumour. It may exist in considerable degree without impairing sight. Perception of colour is affected before acuity of vision. If the tumour be arrested by treatment, as in syphilitic and tubercular growths, the neuritis will subside; it also often lessens when the intracranial pressure (which drives the fluid into the sheath of the nerve) is lessened by trephining the skull, and, still more completely, after removal of the tumour, when this can be effected. Too often, however, before this result is obtained, sight has been damaged beyond recovery. Secondary atrophy of the optic nerves sometimes results from tumours in the neighbourhood of the chiasma, although far less commonly than 'consecutive atrophy.' See also p. 198.

Intracranial aneurysms are rarely accompanied by intra-ocular changes. Now and then, an aneurysm of the internal carotid has caused atrophy by pressure, and even optic neuritis, single or double.

Internal hydrocephalus is usually accompanied by no other ophthalmoscopic changes than slight fullness of the veins. Occasionally simple atrophy occurs, commonly from the pressure of the distended third ventricle on the optic commissure.

Meninges.—Growths in the meninges lead to optic neuritis, just as do tumours in the cerebral substance. The effect of meningitis varies according to its form and seat. Simple meningitis of the convexity is rarely attended by ocular changes. It is very different with basal tubercular meningitis. Occasionally, though rarely, tubercles of the choroid may be seen. In a considerable number of cases there is distinct neuritis; it is well-marked in at least half. Usually too late to be of diagnostic importance, it is now and then sufficiently early to decide the nature of the case. A similar change is common in both syphilitic and traumatic meningitis, but is rare in the epidemic cerebrospinal form, except in cases of unusual duration.

Diseases of the cranial bones.—Caries of the sphenoid bone may cause descending neuritis; caries elsewhere usually only affects the eye by causing meningitis or abscess. Thickening of the cranial bones may be attended by neuritis, but usually the first visible result is atrophy. This is apparently produced by the resulting constriction of the nerve and sheath at the optic foramen. Distension of the sphenoidal sinuses may damage one or both nerves by pressure on them as they pass through the optic canals; in this affection the nasal part of the nerve is first involved. Inflammatory mischief or growths in the orbit frequently cause neuritis or atrophy, the optic nerve-trunk being damaged directly. In these cases the affection is

unilateral, at least for a long time, and is often accompanied by prominence of the eyeball, and tenderness when it is pushed back.

Injuries to the head may affect the eye in various ways: (1) The retina may suffer in consequence of the immediate concussion. (2) Optic neuritis may come on after a few days, commonly as the result of a traumatic meningitis. (3) Direct injury to the optic nerves may cause loss of sight and simple atrophy. (4) Optic neuritis may come on some weeks after the injury, and is usually due to inflammatory processes in the damaged brain.

2. Spinal Cord.—*Acute myelitis and spinal meningitis* are very rarely attended by eye-changes. In one or two cases coincident optic neuritis has been observed. The connection between the two is obscure; probably they are independent effects of a common cause, e.g. a blood-state. *Sclerosis of the posterior columns* (locomotor ataxy) is accompanied by atrophy of the optic nerves in a considerable number of cases, although not perhaps in more than 15 per cent. When it does occur it is frequently an early rather than a late symptom. It is always the simple form of atrophy, often grey, with unnarrowed vessels. Sight usually suffers gravely; the field of vision is much restricted; and perception of colours may be lost. The atrophy is not the result of any extension upwards of the disease in the posterior columns. It may occur when this has scarcely commenced, and even years before the earliest symptoms. It is apparently an associated degeneration. In *lateral sclerosis* changes in the fundus oculi are, as a rule, absent. In *disseminated sclerosis*, optic nerve-atrophy may occur, just as in posterior sclerosis, but less frequently. Damage to sight, without marked ophthalmoscopic changes, occasionally results from the sclerosis invading the optic commissure or nerves. In caries of the spine, changes in the optic disc are practically unknown. In very rare cases of injury to the spine, neuritis and subsequent atrophy have been observed, but these results are so uncommon that their precise significance is doubtful.

3. Functional Diseases.—In *exophthalmic goitre* the only ophthalmoscopic change is increased size of the retinal arteries, which may pulsate visibly. In *chorea*, embolism of the central artery of the retina has been once or twice observed; and optic neuritis, slight in degree, is not unknown. It is usually met with in hypermetropic eyes, but, nevertheless, subsides when the chorea is over. With *neuralgia* of the fifth, optic nerve-atrophy has been observed; the nature of the association is doubtful. In *idiopathic epilepsy* the appearance of the fundus is, as a rule, perfectly normal. Even during an attack it is probable that the only change is distension of the veins in the stage of cyanosis. But

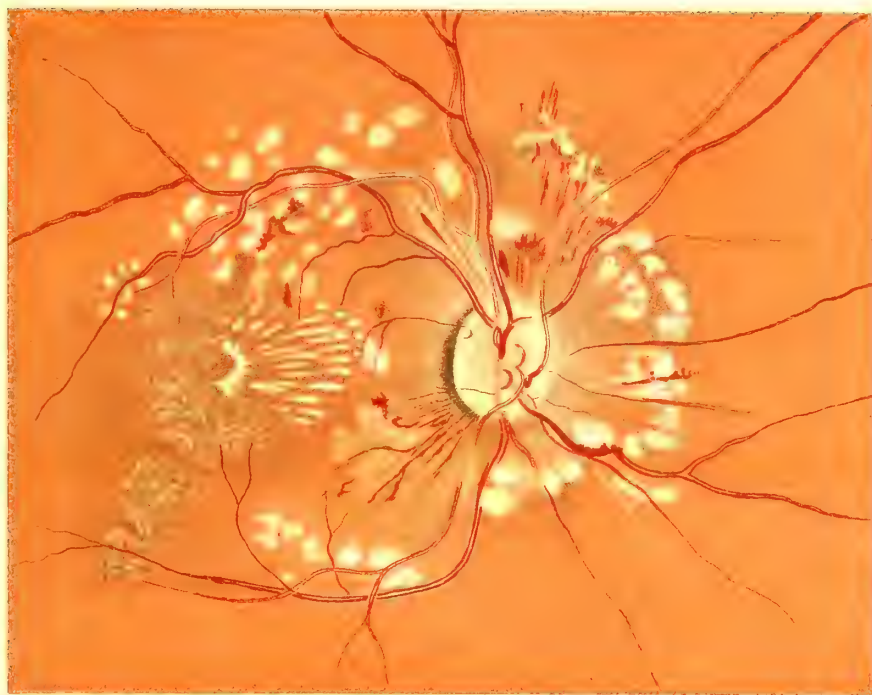
SECOND PLATE

(DIVIDED INTO TWO).

1. Retinitis in Renal Disease. The edges of the disc are hazy, from œdema. There are numerous retinal hæmorrhages, chiefly of striated form near the disc. White fluffy opacities are present in the inner layers of the retina, obscuring the vessels in some places: these were unusually numerous and marked in this particular case. The arteries are rather narrow and show irregularities in calibre, particularly the large branch passing downwards and outwards; they indent the veins where they cross over them. At the macula there is an incomplete stellate figure, formed by white lines and streaks radiating from the fovea centralis: these lines are usually, as here, best marked between the fovea and the disc. Right eye: erect image.

2. Disseminated Choroidal Atrophy in inherited Syphilis. Round or oval patches are seen distributed over the fundus. Each spot or patch has a punched-out appearance, the centre being light in colour and evidently on a deeper level than the adjoining healthy retina. This centre is usually surrounded by a deeply pigmented black border, outside which again there is a pale zone. The retinal vessels pass uninterruptedly over any patch that may chance to underlie them. The disc is paler than normal and has an opaque dead look. In this case the retinal vessels are not decidedly abnormal, but frequently in such cases they are much reduced in size, particularly the arteries. Right eye: erect image.

Our thanks are due to Messrs. Nettleship, Lawford and Spicer for the loan of original drawings from which several of these illustrations have been taken.—R. M. G.



1.



2.



during the status epilepticus, when attacks recur with great severity for several days, a condition of slight neuritis may be produced, subsiding after the attacks are over. In cases of *convulsions* from organic brain-disease, it must be remembered, optic neuritis or its effects are often met with. The frequency with which morbid appearances are to be seen in the eye in *insanity* has been variously stated, and by some writers unquestionably exaggerated. They are most frequent in general paralysis of the insane. Optic nerve-atrophy is the usual change, and is sometimes an early event, just as in locomotor ataxy. In very rare cases slight neuritis has been seen. In mania, melancholia, and dementia it is probable that there are no related morbid appearances in the eye.

II. DISEASES AFFECTING THE URINARY SYSTEM.—1. **Bright's Disease.**—Sight may be impaired in this complaint by uræmic poisoning, or by retinal changes. The latter may occur, even in considerable degree, without any affection of vision. The arteries may occasionally be conspicuously narrow (contracted), and in rare cases may present sclerosis of the outer coat, or minute aneurysms. Aneurysmal dilatations of the capillaries may often be found *post mortem*, in association with other degenerations, and probably sometimes constitute one cause of the occurrence of a very common change in the retina—hæmorrhages. These are usually striated, situated in the nerve-fibre layer; sometimes they are irregular in shape, and situated in the deeper layers. They may detach the retina from the choroid, or burst through into the vitreous. Sometimes they exist alone; more commonly they are conjoined with other changes, to which the term 'albuminuric retinitis' is given. This latter change may occur in all forms of renal disease, but is by far the most common in the granular kidney. It is a late symptom, never appearing until the general system is suffering. The disease of the retina presents certain elements which are variously combined in different cases. (1) Diffuse slight opacity and swelling of the retina, due to the infiltration of its substance by an albuminous coagulable liquid (œdema). (2) White spots and patches of various size and distribution: some large and soft-edged; others minute, and of pearly whiteness. They are due to fatty degeneration of the retinal elements, or to granular degeneration of albuminous exudations. The small white spots often radiate round the fovea centralis. (3) Hæmorrhages. (4) Inflammation of the optic papilla—'neuritis.' (5) The subsidence of the inflammatory changes may be attended by the signs of atrophy of the optic nerve and retina. According to the predominant character, four types of retinal affection may be distinguished: a degenerative, hæmorrhagic, inflammatory, and neuritic form. In the first the white spots predominate, and there are usually extravasations, but there is little diffuse opacity. In the second the hæmorrhages are so abundant as to be the chief feature. In the third there is much diffuse opacity and swelling of the retina. In the fourth the optic neuritis is in excess of the other changes, and the appearance may easily be ascribed to cerebral disease—the more so that it is often conjoined with headache, and other evidence of cerebral disorder, so that the urine should be carefully examined in all such cases. The conspicuous combination of white spots and hæmorrhages usually enables the retinal affection of albuminuria to be

recognised without difficulty. It may be confounded with the degeneration left by a previous wide neuro-retinitis, but in such cases the signs of atrophy will be conspicuous. The course of the affection in Bright's disease is often progressive, but arrest and even recovery may be obtained by the treatment of the renal disease. When extensive, sight is usually impaired, but is rarely completely lost.

2. **Diabetes Mellitus.**—In diabetes mellitus, in rare cases, retinal changes have been observed, very similar to those of the degenerative form of the albuminuric affection, and this when there was no albumen in the urine. Miliary aneurysms have been found *post mortem*. A distinction from the renal form is the frequency with which there are opacities in the vitreous, due probably to slight extravasations of blood.

III. DISEASES OF THE HEART.—The peculiar conditions of the intra-ocular circulation prevent any dynamical changes in the circulation. Venous distension, if considerable, may be visible in the eye, especially in cyanosis. When arterial pulsation is strong, it may be seen in the retinal arteries, as in aortic regurgitation, and also, it is said, in exophthalmic goitre. Embolism of the central artery of the retina may occur, and, like embolism elsewhere, is most common in mitral constriction. In ulcerative endocarditis, accompanied by multiple embolism, retinal hæmorrhages occur, for the most part round, with a pale or white centre. They are almost pathognomonic.

IV. DISEASES OF THE BLOOD.—Acute *anæmia* from hæmorrhage may be followed by loss of sight, slight or considerable, transient or permanent. The accident most commonly follows hæmatemesis, uterine hæmorrhage, or venesection. In some cases no ophthalmoscopic changes have been found; in others there has been neuro-retinitis. The mechanism of the affection is obscure. Simple chronic *anæmia* is accompanied by marked pallor of the veins, sometimes of the choroid and disc, but the latter is always within the physiological variations in tint. Occasionally in chlorosis optic neuritis is met with, developing quickly to a high degree of intensity, such as is met with in cerebral tumour. It disappears rapidly under iron, but too often ignorance of the cause has led to the loss of so much time, while iodide of potassium, &c., have been given, that sight is hopelessly damaged. In *pernicious anæmia* the choroid is notably pale, the arteries small, the veins very broad (atonic) and pale. Hæmorrhages are frequent, especially around the optic disc, and they are often associated with white patches (p. 56). Some extravasations are rounded, with a white or pale centre. Occasionally there is marked neuritis. In *leucocythæmia* the pallor and the width of the veins are very striking. Extravasations are almost invariable at some period. White spots are frequent, some degenerative, others due to aggregations of leucocytes, some are surrounded by a halo of extravasation. There may also be considerable general swelling of the retina, throwing the distended veins into conspicuous antero-posterior curves. In *purpura* and *scurvy* retinal hæmorrhages also occur. In the intense forms of *purpura*, indeed, they are probably constant, and may be large. They have sometimes been seen in the choroid.

In rare cases of *menstrual* disorders, and still rarer instances of *intestinal* disturbance (chronic diarrhœa), optic neuritis has been observed. Suppression

of the menses has been followed by retinal hæmorrhages. The connection between the several events is obscure.

V. CHRONIC GENERAL DISEASES.—In chronic general diseases ophthalmoscopic changes are met with occasionally. In *tuberculosis*, tubercles may form in the choroid, and be recognisable as small, round, yellowish-white spots, free from pigment. They have more frequently been found in this situation after death than during life, perhaps because not looked for with sufficient perseverance, since they may form rapidly. They are sometimes of great diagnostic importance, especially in cases of general tuberculosis, causing obscure and ill-defined symptoms. In *syphilis* ocular changes are, as is well known, common, but they come chiefly under the care of the surgeon. Traces of past iritis, or of choroiditis—areas of choroidal atrophy with irregular accumulation of pigment—frequently afford the physician important evidence of the previous existence of syphilis, acquired or inherited. In the latter the choroidal changes are of especial importance, and may be confined to small round white spots with pigment in the centre, or there may be evidence of more extensive choroiditis or merely of choroiditic atrophy, a yellowish disc, with the edge a little blurred, and very small retinal vessels. *Gout* has been supposed to cause retinal hæmorrhage (Hutchinson), and to it is probably due the widespread ‘hæmorrhagic retinitis’ that is the consequence of thrombosis in the central vein of the retina. Intra-ocular neuritis is seldom due to this cause, except in slight degree, as the result of a more intense inflammation behind the globe. This may occur in the subjects of acquired gout, or in young persons the subjects of the inherited disease, and may be on one side or on both, simultaneously or in succession.

In *lead-poisoning*, besides the amblyopia which may come on without marked ophthalmoscopic changes, atrophy of the disc is occasionally met with, preceded, in some cases, by a stage of congestion—a red disc, with softened edges, without swelling. A considerable degree of neuritis, double, with swelling and hæmorrhages, occurs occasionally, especially in connection with cerebral symptoms, but without any coarse lesion of the brain. In *chronic alcoholism*, optic-nerve atrophy has been described, and also a condition of congestion. The amblyopia which accompanies the atrophy is said by Förster to be characterised by loss of central vision for colour. The same fact is well established with regard to *tobacco amaurosis*, in which similar congestion and atrophy may occur.

VI. ACUTE GENERAL DISEASES.—In acute general diseases, changes in the fundus are for the most part rare. After *typhus*, *typhoid*, and *scarlet fevers*, optic neuritis has been occasionally observed, apart from any renal or cerebral complication. The renal sequelæ of scarlet fever may of course lead to the special retinal changes. *Malarial fevers*, *ague*, &c., are frequently attended by retinal hæmorrhages (Poncet, S. Mackenzie). Sometimes the extravasations have paler centres. Optic neuritis and atrophy have also been observed. *Erysipelas of the face* has been accompanied by loss of sight, and followed by atrophy, probably by the extension of the inflammation to the orbit, and to the trunk of the optic nerve. *Pyæmia* and *septicæmia* have long been known to be occasionally accompanied by metastatic panophthalmitis, and

recent observation has shown that slighter alterations in the fundus oculi frequently accompany the severer forms of these affections. Of these the most important are retinal hæmorrhages, round or irregular, sometimes large, and often with pale centres, as in ulcerative endocarditis. Although it is probable that they are in some cases due to septic embolism, they may occur without endocarditis, and may be due, in some cases, to chemical changes in the blood, produced by the organised virus of the septicæmia. They are almost invariably in puerperal septicæmia (Litten). They are of very grave significance, but not necessarily a fatal omen. In other cases a peculiar form of retinitis has been observed, with white spots about the papilla and macula lutea (Roth).

W. R. GOWERS.

R. MARCUS GUNN.

OPISTHOTONOS (ὀπισθεν, backwards; and *τόνος*, a stretching).—A tetanic spasm, in which the body is arched backwards, so that it rests on the head and heels. See TETANUS.

OPIUM, Poisoning by.—In consequence of the extent to which opium and its preparations, including morphine, are used for the relief of pain, and the readiness with which these drugs are procurable, poisoning by these agents is of frequent occurrence; and there is no doubt that great numbers of infants perish every year in this country through the improper use of quack remedies containing opium.

So far as toxicology is concerned, the effects of opium may be referred exclusively to morphine; since the effects of the other active constituents of the drug are overshadowed by those of its chief alkaloid. See also MORPHINE-HABIT, p. 611.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances after opium-poisoning may be almost *nil*. As a rule the brain is congested, the *puncta cruenta* being especially marked; and the lungs and right side of the heart may exhibit an engorgement, as if from a modified asphyxia; but this condition is by no means invariable.

SYMPTOMS.—The first effect of the administration of a toxic dose of opium—a state of *bien-faisance* or exaltation—commonly observed also after the administration of a medicinal dose, may be either very short or entirely wanting; and this is usually the case when morphine is injected hypodermically. A second stage, in which the symptoms closely resemble those of congestion of the brain, soon sets in. The face is either suffused or cyanosed; the pupils strongly contracted; the skin dry and warm; the breathing slow, deep, and becoming stertorous. The patient is apparently unconscious, but may be aroused by shaking, or shouting in the ear; and when he is aroused, the respirations become more rapid, and the skin may regain its normal colour. The symptoms of this second stage may gradually ameliorate under appropriate treatment; or a third stage—that of prostration—supervenes. The coma is now profound, and it may be impossible to arouse the patient. The pupils are contracted to the size of pin-points; or towards the termination of life may be widely dilated. Respiration is now very slow, shallow, with gradually increasing intervals, during which there are no signs of breathing, and the patient lies in a death-like calm. The face is at once pallid and cyanosed; the skin is bathed in

perspiration, at first warm, and then cold and clammy. The pulse increases in rapidity, with progressively increasing feebleness. The patient may even now recover, signs of life returning very gradually; or death may occur from failure of respiration, the other functions of life becoming also gradually extinguished.

Unusual symptoms in opium-poisoning are trismus and convulsions. In children toxic doses may produce very rapid effects, the second stage of the intoxication being wanting, and severe collapse and complete unconsciousness rapidly supervening.

DIAGNOSIS.—The certain diagnosis of opium-poisoning is often a matter of great difficulty, as the symptoms may differ in no material respect from those exhibited in congestion of the brain, however produced, apoplexy, and uræmia. The case may also be confounded with profound alcoholic intoxication. It may also be difficult or impossible to diagnose from poisoning by chloral hydrate—a matter of less importance, since the treatment of the two cases would be similar. The differential diagnosis of opium-poisoning rests upon the equally and minutely contracted state of the pupils, a condition which is all but universal in the second stage of opium-poisoning; our ability to arouse the patient temporarily, the rousing being followed by more or less complete disappearance of the cyanosis of the countenance, and by increased rapidity of respiration; and the profuse warm or clammy cold perspiration. An examination of the urine, which may have to be drawn off by the catheter, for albumen and morphine by the iodic-acid test should always be made; but it must be borne in mind that uræmia and opium-poisoning may be co-existent.

PROGNOSIS.—This is at all times doubtful. There is great liability to relapse, even when the patient appears to be doing well.

TREATMENT.—First, evacuate the stomach by means of the stomach-pump or tube, or, failing this, by the use of emetics. These, however, act with difficulty in cases of opium-poisoning; and there is a special danger in the use of depressing emetics, as, for example, tartar emetic, on account of the possible retention by the stomach of a fatal dose of the emetic. Warm mustard and water, and carbonate of ammonium are the best emetics to administer. The free administration of a dilute solution of potassium permanganate—1 per 1000—is advisable, morphine being destroyed by this agent. A good plan is to wash out the stomach frequently with the above solution. The patient must be prevented from lapsing into a state of somnolence, by walking him about at frequent intervals; alternate warm and cold applications to the chest; flicking the feet with a damp towel; shouting into the ear; and the application of the faradic current; but in severe cases such a procedure cannot be safely adopted. These means will have the additional advantage of maintaining the flagging respiration, and restoring normal breathing. In the last resort artificial respiration must be freely employed. The absorption of the alkaloids of opium may be delayed by the free administration of solutions containing tannin, so as to render the alkaloids insoluble; and among the best media containing tannin are strong infusions of tea and coffee. The caffeine which these infusions contain itself also exerts a powerful remedial influence in this form of intoxication. Atropine, as a respiratory stimulant, appears also to be

serviceable as a direct antidote to morphine. It is best given by subcutaneous injection, in doses of $\frac{1}{40}$ to $\frac{1}{20}$ grain. Hypodermic injections of strychnine may be employed. Alcoholic stimulants should be freely given.

THOMAS STEVENSON.

OPIMUM-HABIT.—See MORPHINE-HABIT, p. 611.

OPTIC NERVE AND TRACT, Diseases of. The optic nerve may be damaged by various intra-ocular processes; but these, and also its primary atrophy, have been already described (*see EYE, AND ITS APPENDAGES, Diseases of; OPHTHALMOSCOPE; and OPHTHALMOSCOPE IN MEDICINE*). In this article only those affections which are situated behind and independent of the eye will be described.

Passing from the orbit into the intracranial cavity by the optic foramina, into which they closely fit, the optic nerves are connected at the chiasma, where an approximate semi-decussation takes place. From the chiasma each optic tract, containing fibres from the same-named halves of both retinae, passes backwards, between the crus cerebri and the inner edge of the temporo-sphenoidal lobe, to the posterior portion of the optic thalamus, where it becomes connected with this and the external geniculate body. Fibres pass also to the corpora quadrigemina, especially the anterior; and from or by the thalamus to the white substance and convolutions of the occipital lobe.

ÆTIOLOGY.—In the orbit the nerve may be damaged by inflammation, which is sometimes apparently due to gout, and sometimes invades the nerve from the orbit (as in orbital cellulitis from facial erysipelas), or is produced by exposure to cold. Inflammation outside the nerve does not always invade its substance on account of the thickness of the sheath which invests it, but the fibres are damaged by the pressure of the inflammatory products. It may also be compressed by an aneurysm of the ophthalmic artery or by orbital tumours; or may be itself the seat of morbid growths or of hæmorrhage. At the optic foramen the nerve may be compressed by new-growths or by a narrowing of the foramen, such as occurs in thickening of the cranial bones, an occasional consequence of syphilis, acquired and congenital. Within the skull, the nerve in front of the chiasma may be damaged by the extension of inflammation from the meninges. The optic commissure is occasionally compressed by or involved in growths, and may be compressed by great distension of the third ventricle. Interstitial inflammation probably sometimes occurs. The nerves in front of the chiasma, and the chiasma itself, are liable to be damaged by the pressure of aneurysms of adjacent arteries. The optic tracts may be involved in hæmorrhage into, or softening of, the crura cerebri; but the most frequent cause of their damage is a tumour arising at the base of the brain, or in the adjacent part of the temporo-sphenoidal lobe. The central continuation of the optic nerves, constituting the visual path—the optic thalamus, the white substance from it to the occipital lobe, and occipital cortex, may be damaged by tumour, softening, or hæmorrhage, with impairment of sight as the result. The corpora quadrigemina are rarely affected so as to cause visual symptoms, except by growths, which produce this effect by pressure on the adjacent visual path to the cortex.

SYMPTOMS.—Damage to the optic nerve, between the optic commissure and the eye, causes an affection of sight in that eye only. There may be either a concentric, a central, or a sectorial defect in the field, or complete blindness; the direct reflex action of the pupil is impaired. When the nerve is slowly compressed, the loss of sight is followed by slow atrophy of the intra-ocular extremity. When it is invaded by inflammation, this usually descends to the eye, and is visible as intra-ocular neuritis, often slight. Inflammation at the back of the orbit usually also involves the motor nerves, and so may cause paralysis of all the ocular muscles. These recover, however, much more readily than does the optic nerve. When the nerve is compressed by narrowing of the optic foramen, the loss of sight is accompanied by intra-ocular atrophy, sometimes preceded by slight neuritis. This is also present in most cases in which inflammation extends from the meninges to the intracranial part of the optic nerves, or the optic chiasma. Damage to the chiasma usually affects the sight of both eyes. In most cases the decussating fibres suffer chiefly or alone, and consequently there is loss of function of the inner half of each retina, and loss of the outer half of each field of vision—*temporal hemianopia*. Damage to the outer part of the commissure on each side affects the fibres which do not decussate, and so causes loss of function of the outer half of each retina, and so loss of the inner half of each field—*nasal hemianopia*. This is very rare, but has been seen from calcification of the carotid artery on each side (Knapp). Partial nasal hemianopia is also sometimes met with in tabes. In irregular damage to the chiasma the loss of vision may be irregularly distributed in the two eyes.

The optic tract receives fibres from the half of each retina on the same side, and its damage thus causes loss of sight in the opposite half of each field of vision—*lateral or homonymous hemianopia*. The loss is often more extensive in the eye on the side opposite to the lesion than in that on the same side. Since the motor tract, in the adjacent crus cerebri and hemisphere, has decussated at the medulla, if it is also involved in the lesion, there is hemiplegia on the same side as the loss in the field of vision. The patient is unable to see to the side on which he cannot move the limbs. Thus the writer has recorded a case in which a patient had, first, right hemianopia, and afterwards right hemiplegia. Both were due to a small tumour of the inner part of the temporo-sphenoidal lobe, which had first invaded the optic tract and then the crus.

Lateral hemianopia results, occasionally, from disease of the posterior extremity of the optic thalamus, and frequently from a lesion involving the fibres that pass thence, adjacent to the posterior extremity of the internal capsule, and through the white substance of the occipital lobe, to the cortex at the extremity of the hemisphere, and chiefly to the lips of the calcarine fissure, disease of which always entails this symptom. Partial disease of this area causes a partial loss of the half field; the upper quadrant is lost if the lower lip of the fissure is diseased, the lower quadrant of the field if it is the upper lip of the fissure. It is uncertain whether damage to the cortex on the outer surface of the occipital lobe causes hemianopia, or whether, with this lesion, the symptom has been due to the disease penetrating the white substance to the fibres from the cuneus. These fibres, the 'optic radiation of

Gratiolet,' may be reached by extensive disease of the middle part of the cortex, and then hemianopia may be associated with hemiplegia, and even with aphasia. When the cause of hemianopia is disease of the chiasma or tract, the pupil does not contract when light is thrown on the blind half of the retina, because the fibres for the reflex centre are involved. When the disease is in the thalamus or occipital lobe there is no loss of action; this affords a means of distinction, but much care is needed to get clear results.

Double hemianopia involves complete loss of sight, and is the probable cause of such loss, when of sudden onset. Half loss of the colour-fields, with no loss for white light, has been observed, and perhaps depends on the existence of a distinct hemiopic colour-centre.

In hysteria, with loss of the other special senses on one side, there is a peculiar affection of vision, which is said to be sometimes met with as a result of organic disease—'crossed amblyopia.' There is dimness of sight of the eye opposite to an organic lesion, and great contraction of the field, with loss of colour-vision; on the side of the lesion there is a much slighter but similar change. It is stated that in the cases in which the lesion has been ascertained, it has always involved the angular gyrus in the postero-inferior angle of the parietal lobe, in front of a line drawn across the brain from the parieto-occipital fissure. It would seem that here there is a centre related to both half-vision centres, in which they are recombined, but to which the opposite field is chiefly related. A partial lesion in this centre can be compensated by the other hemisphere, but the effect of a partial lesion in the half-vision centre is permanent, as much so as one in the optic path leading to it.

Any one of the diseases to which the brain is subject has the same effect if in a corresponding situation; but, as a rule, softening from arterial obstruction, due to syphilitic disease or atheroma, or a new-growth, is a more frequent cause of damage to the optic path in tract or hemisphere than is cerebral hæmorrhage.

DIAGNOSIS.—The chief points which are our guides in determining the position of post-ocular disease, causing loss of sight, have been already stated. If the affection of sight is confined to one eye, it is probably—and if associated with unilateral optic neuritis it is almost certainly—due to disease of the nerve in front of the chiasma. In this case the reaction of the pupil to light is impaired. On the other hand, if the unioocular affection of sight is associated with hemiplegia, and especially with hemianæsthesia, on the same side, it is probable that the disease is in the hemisphere, and the failure of sight is produced in some manner at present unknown. In this case the pupil acts well to light. Lateral hemianopia indicates disease of the tract, the posterior part of the thalamus, or the white substance between the thalamus and the occipital convolutions, or of these convolutions themselves. In which of these positions it is, must be determined by the indications of the localisation of disease of the brain (BRAIN, Tumours of). Nasal hemianopia or temporal hemianopia indicates disease of the optic chiasma.

PROGNOSIS.—This must be influenced by the position of the lesion, and especially by its nature. When there is simple pressure on the optic nerve, sufficient to abolish sight, the prognosis is very un-

favourable. Damage due to the extension of inflammation often lessens considerably. In disease of the optic commissure or optic tracts the prognosis is also grave, because the morbid processes, from which these parts suffer, rarely recede. Whether it is likely to become stationary must be inferred from its probable nature. On the other hand, in disease of the hemisphere, considerable improvement often takes place, just as it does in other symptoms; but when the hemianopia has become stationary it persists, with little change, to the end of life. Often, however, this symptom is thought to have disappeared when it still persists in a diminished degree.

TREATMENT.—The treatment is essentially that of the disease to which the symptom is due, and need not be further discussed in this place.

W. R. GOWERS.

R. MARCUS GUNN.

OPTIC THALAMUS, Lesions of.—See THALAMUS OPTICUS, Lesions of.

ORBIT, Diseases of.—**SYNON.** : Fr. *Maladies de l'Orbite*; Ger. *Krankheiten der Augenhöhle*.—The diseases of the orbit are not numerous, and are almost exclusively surgical in their character. The bony walls of the cavity are liable to be fractured by direct injury, which generally implicates other portions of the skull; the contained tissues are liable to phlegmonous or suppurative inflammation; and the cavity may be the seat of tumours of various kinds, arising either from the walls or from some portion of the contents.

1. **Hæmorrhage.**—Hæmorrhages into the orbit, excepting as results of injury or from the rupture of aneurysmal tumours, are extremely rare; and the few cases which have been recorded have nearly all occurred in persons of generally hæmorrhagic tendency, as one local manifestation among others of a constitutional malady.

2. **Emphysema.**—Emphysema of the orbit is not unknown, and the writer has seen a young man who, in blowing his nose violently, must have ruptured some of the ethmoidal cells, for he distended his left orbit with air, and, in his own words, blew his eye nearly out of his head. The distension soon subsided, and no permanent injury was done.

3. **Inflammation.**—Inflammation of the tissues within the orbit is not a common affection, but it is liable to occur as a complication of fevers and other debilitating diseases, and especially as a complication of erysipelas of the head and face. It is marked by brawny swelling of the eyelids, with some protrusion of the eyeball and some limitation of its movements, the symptoms appearing too suddenly and increasing too quickly to be attributable to the growth of a tumour. The injection of the conjunctiva is generally less marked than that of the lids, and sight is scarcely or not at all impaired so long as the swelling is only moderate in amount. When the injected conjunctiva of the eyeball becomes oedematous, and more especially when the oedema is limited to one sector of the globe, or is much more pronounced over one sector than elsewhere, it is, in the opinion of the writer, an almost pathognomonic sign of supuration; and the localisation of the oedema will serve as a guide to the position in which pus may be looked for. Other symptoms of supura-

tion, such as rigors, must of course be taken into account.

TREATMENT.—As soon as pus is believed to exist, it should be evacuated, since its retention among the orbital tissues may be productive of serious injury, not only to the eye, but also to the ocular muscles and to the nerves which traverse the orbital cavity. The evacuation is usually best effected by introducing a narrow straight knife through the skin, near the margin of the orbit in the selected position, and by thrusting it carefully onward as far as may be prudent, giving the blade an occasional turn upon its axis, to allow of the escape of pus as soon as it is reached. The direction of the point should be governed by complete knowledge of the anatomy of the parts; and it is better to withdraw the blade too soon than to incur any risk of wounding important structures. When it is withdrawn, if no pus follow, the puncture may be carefully deepened or extended laterally by a probe; but it is not necessary to be very strenuous in such endeavours, because if the wound through the skin and fascia be kept from healing by the introduction of a strip of lint, or of a bit of drainage-tube, the pus will soon find its way into the channel of escape thus provided for it. The cavity of the abscess should be syringed out from time to time, according to the amount of discharge, with some suitable astringent or antiseptic lotion; and care must be taken that a free opening is maintained as long as pus continues to be secreted.

4. **Caries.**—In strumous children, caries of some part of the margin of the orbit is not uncommon; and, after the diseased bone has come away, we frequently see much deformity of the lids produced by adhesions between the skin and the deeper tissues, or by the contraction of cicatrices. Many of such cases require plastic operations; but each one, before any operation is undertaken, must be carefully studied in order to discover the most promising method of procedure. In a lad with inherited syphilis, the writer has seen very extensive necrosis of the orbital margin, subsequent to the partial removal, and partial absorption, of a large gummatous tumour in the cavity.

5. **Tumours.**—Tumours of the orbit may be cysts (hydatid, dermoid, or sebaceous); lipomata; gummata; sarcomata, originating in connective tissue, and presenting the characters of myxoma, or of the sarcomatous growths distinguished respectively by round and by spindle-shaped cells; or they may be gliomata, springing from the connective tissue of the optic nerve. In other instances they may commence as an apparent hypertrophy of the lacrymal gland; or they may be cartilaginous, or osseous. All alike produce protrusion of the eyeball, and limitation of its movements, together with an amount of disturbance of vision, which depends upon the degree of pressure or of stretching to which the optic nerve is subjected, or upon the degree in which the intra-ocular circulation is impeded. Many of the forms are liable to recurrence, and may thus ultimately destroy life.

TREATMENT.—All growing tumours of the orbit require removal, if possible, without sacrifice of the eyeball; but it is usually prudent to leave stationary tumours alone, more especially congenital cysts, which have frequently been found in relation to deficiencies of bone which have opened up a connection with the cranial cavity.

R. BRUDENELL CARTER.

ORCHITIS (*ὄρχις*, a testicle).—Inflammation of the testis. See **TESTES**, Diseases of.

OREZZA, in Corsica.—Iron waters. See **MINERAL WATERS**.

ORGANIC DISEASE.—This expression indicates a disease in which there is a structural change in the part affected, as distinguished from a merely functional disorder, in which no such change has been found.

ORIENTAL SORE.—**SYNON.** : Delhi Boil ; Bagdad Boil ; Aleppo Evil ; Fr. *Bouton d'Alep* ; Ger. *Beule von Alep*.

DEFINITION.—A specific ulcer of the skin, commencing as a papule, subsequently breaking down into an extending and indolent ulcer, tending to run a natural slow course, and ultimately to heal, leaving a permanent depressed cicatrix.

GEOGRAPHICAL DISTRIBUTION.—This affection is endemic in many places, chief among which may be named Asia Minor, Syria, and Mesopotamia, in which latter country, especially in Bagdad, it is probably most common. It is also found in several other countries, and has been variously named from its local prevalence ; thus in Morocco and the Sahara it is known as the Biskra boil or sore (*Bouton de Biskra*), and in India as Delhi Boil, Moulton Sore. It occurs also in Arabia, Persia, Egypt, islands in the Mediterranean, and lately it has been described as common in Bahia, Brazil, from which it would appear that it is not invariably an Eastern affection.

It is more common in cities than in the country, and seems to be influenced in the frequency of its occurrence by sanitary conditions, as Manson mentions its marked decrease in Delhi immediately after certain sanitary improvements in the city. Hirsch states that it is more common at the commencement of the colder season of the year.

ÆTIOLOGY AND PATHOLOGY.—Although up to the present time no micro-organism capable of propagating the sore has been isolated, there is no doubt, from the communicability of the disease by means of inoculation with the discharge, in the case of both man and the lower animals, that it is due to a specific germ, and that no constitutional condition has more than a predisposing influence in its causation. Numerous micro-organisms, for the most part diplococci, have been described in connection with Oriental Sore ; but no one of them has been yet found capable, in pure cultivation, of producing the sore. An attack of the sore seems generally to confer immunity against future attacks, and this has led to the practice among the Jews of Bagdad of inoculation with the virus in parts of the body which are usually hidden from sight, in order to avoid the possibility of an unsightly scar on the face or other exposed part, the chance of escape from an attack of the sore at some time or other being considered very small. There is some doubt as to the absolute certainty of the immunity against subsequent infection, at all events by inoculation. Although called in many places a 'boil,' the Oriental Sore is not a furunculus ; it commences as a papule, which breaks down after a few days into an ulcer, which tends to spread for a variable time and to a variable extent, and then to slowly cicatrise. The essential pathological condition present is, according to all observers, a large de-

velopment of lymphoid or granulation-corpuscles, which infiltrate the derma between the lymphatics, blood-vessels, and sweat-glands. These corpuscles are especially numerous beneath the centre of the lesion, where they replace entirely the proper elements of the tissues. Infection is not improbably conveyed by flies or biting insects, such as fleas, and perhaps mosquitoes. The incubation-period, from infection to appearance of papule, appears from experiment to be about three to five days.

SYMPTOMS AND DESCRIPTION OF SORE.—The first appearance of the sore is in the form of an itching, shotty-feeling papule, which in a day or two develops scales on its surface ; the scales are at first thin and papery, becoming after five or six days thicker and brownish in colour, and forming a crust. After a few more days the crust either falls or is scratched off, and a shallow ulcer with perpendicular jagged edges is left. This tends to spread by further breaking down at its margins, and also in some cases by other papules in its close vicinity forming ulcers which join the original ulcer ; the resulting sore, which is generally oval in shape, thus varies considerably in size, from a diameter of an inch or an inch and a half, which is usual, to one of several inches. The surface of the sore is irregular, the discharge ichorous, and not very profuse, occasionally forming crusts. After a variable time, said to range from two or three to twelve or more months, healing commences, skin commencing to form first in the centre of the ulcer, where the effect of the germ is probably first exhausted, and spreading to the circumference ; ultimately a depressed white or pinkish cicatrix is left, which is permanent. The sores are situated generally on the hands, feet, arms, legs, or face, the last situation being a common one in young children ; there may be only one sore, or two or three, or, in some cases, many ; people of either sex, of all ages, occupations, and social conditions, are liable to attack.

TREATMENT.—Destruction of the original papule, or of the ulcer when small, by means of caustics, or the actual cautery, has been advocated, but it does not appear that any great measure of success has been thus attained. It is probable that were any radical measure likely to be followed by success it would be found in clean excision of the original papule, such excision to include, of course, the whole thickness of the skin and also the subcutaneous fascia ; this operation, however, does not seem to have been tried. All writers are agreed that the best treatment, having regard to the fact that the affection is one that must run its course, is by soothing, palliative measures ; poultices, or hot boric-acid fomentations if the surface of the ulcer be foul, followed by iodoform-ointment or any simple antiseptic dressing. Manson states that, in the experience of Andrew Duncan, a piece of thin sheet-lead strapped over the ulcer is often very successful, a mode of treatment which formerly was a favourite one in certain forms of small irritable ulcer in England. Although the constitutional condition of the patient has probably nothing to do with the causation of the disease, it may have a good deal to do with the rate and regularity of the healing process ; any state, therefore, such as one of anæmia or debility, betokening a deficiency of power of repair, should receive appropriate treatment.

MAX. F. SIMON.

ORIENTATION.—This term is used in medicine to signify the power possessed by a sane person of accurately judging of his relationship to surrounding objects.

OROYA FEVER.—See CARRION'S DISEASE.

ORTHOPNŒA (*ὀρθός*, erect; and *πνέω*, I breathe).—A form of difficult breathing, in which the patient is compelled to assume the sitting or erect posture.

ORTHOTONOS (*ὀρθός*, straight; and *τόνος*, a stretching).—A form of tetanic spasm, in which the body is rigidly extended. See TETANUS.

OSMIDROSIS (*ὀσμή*, odour; and *ἰδρώς*, sweat). A condition of the perspiration in which it yields an unusually strong or fetid odour. See SUDORIPAROUS GLANDS, Disorders of.

OSSEOUS SYSTEM, Diseases of.—See BONE, Diseases of.

OSTEITIS (*ὀστέον*, a bone).—A synonym for inflammation of bone, which may be of various kinds. See BONE, Diseases of.

OSTEITIS DEFORMANS.—See BONE, Diseases of.

OSTEOMALACIA (*ὀστέον*, a bone; and *μαλακός*, soft).—A synonym for mollities ossium. See MOLLITIES OSSIIUM.

OSTEOMYELITIS (*ὀστέον*, a bone; and *μυελός*, the marrow).—A name for inflammation of the médulla of bone. See BONE, Diseases of.

OSTEO-SARCOMA (*ὀστέον*, a bone; and *σάρξ*, flesh).—A sarcoma with a tendency to ossification. See BONE, Diseases of; and TUMOURS.

OTALGIA (*ὄς*, *ὠτός*, the ear; and *ἄλγος*, pain). Pain in the ear: earache. See EAR, Diseases of.

OTITIS (*ὄς*, the ear).—Inflammation of the ear. See EAR, Diseases of.

OTOLITH.—See CONCRETIONS.

OTOMYCOSIS.—A chronic inflammation of the external ear induced by the presence of a fungus. See EAR, Diseases of, p. 434.

OTORRHOEA (*ὄς*, the ear; and *ῥέω*, I flow).—Discharge from the ear, usually purulent. See EAR, Diseases of.

OVARIES, Diseases of.—SYNON. : Fr. *Maladies des Oaires*; Ger. *Krankheiten der Eierstöcke*; *Krankheiten der Ovarien*.—Under the heads MENSTRUATION, Disorders of, and HYSTERIA, much information may be found upon subjects which might be included among the diseases of the ovaries.

Total removal of the ovaries brings about total suppression of menstruation, but this operation is often impracticable or liable to fail for reasons which will be explained under 'Abnormalities.' On the other hand, when, in retro-peritoneal hysterectomy, both ovaries are saved, the patient is spared the discomforts of a sudden premature menopause, such as often, though not invariably, results when both ovaries are removed with the uterus. Yet

observers of experience have found that where the ovaries have been left, their atrophy with consequent menopause always follows within a year or two, it all the tissue of the body of the uterus be removed, and nothing but the cervix left in the stump.

The ovaries swell at about the menstrual period. This phenomenon has been observed when an ovary lies in a hernial sac. In a healthy subject, the ovaries do not seem to be tender during the catamenia, notwithstanding their engorgement. It is best to delay an operation when on the appointed day the period appears prematurely, unless, indeed, the operation be urgent. The vessels of the broad ligament will be found congested and the ovaries swollen, yet their removal under the circumstances seldom causes marked local or general disturbance. Hysterectomy for fibroid tumours is also best delayed if an unexpected period comes on. The more blood there is in the uterus, the greater the risk in that operation, and both uterus and ovaries are certainly engorged during the period. The appearance of true, typical menstruation with distinct molimen, during the first few days after ovariectomy, hysterectomy, and many other operations is usually associated with a distinct rise of temperature, but no other sign of constitutional disturbance. See also PAIN IN VISCERAL DISEASE.

The diseases of the ovaries which will be specially alluded to in this article are as follows: (1) Abnormalities; (2) Displacements; (3) Disturbances of Circulation; (4) Acute Inflammation; (5) Chronic Inflammation; and (6) Tumours, including Cysts.

1. Abnormalities.—Absence of one ovary has been found in subjects where the uterus and the opposite ovary were perfectly well-developed. Total absence of both ovaries and even of the uterus as well has been noted in adults physically and mentally sound. As a rule, however, it is otherwise; and in cases of sexual derangement the genital tract should always be explored. Dysmenorrhœa and dyspareunia have been observed in patients with ill-developed ovaries. A third or accessory ovary has been detected by many authorities, but the term is often applied to a mere lobe projecting from the normal organ and sometimes pedunculated. The 'accessory ovary,' relatively very rare, has been the accredited cause of persistent menstruation after the removal of both normal ovaries or after double ovariectomy. But ovarian tissue is often found in the ovarian ligament, which forms part of the pedicle left behind after these operations. Hence persistent menstruation under such circumstances is not mysterious.

2. Displacements.—Congenital or acquired displacements into the inguinal canal or prolapse into Douglas's pouch have been frequently detected. The 'ovary' sometimes proves to be a testicle. A true ovary has been found in the sac of a femoral, umbilical, obturator and gluteal hernia respectively (see OVARIES, Herniæ of). Non-descent of the ovary into the pelvic cavity is, on the contrary, very rare. It has been noted in about three adults, and causes no inconvenience.

3. Disturbances of Circulation.—Hyperæmia, when not excessive, may be considered as an essential part of normal menstruation. A very little excess may lead to the formation of a large clot in an unbroken Graafian follicle, or extravasation into the stroma of the ovary (*ovarian apoplexy*). In typhoid fever, Asiatic cholera, scurvy, phosphorus-

poisoning, severe burns, and in the acute specific fevers, these hæmorrhages are frequent. The ovarian stroma may be soaked in blood, and the affection is usually bilateral. Undoubtedly it accounts in some of such cases for sterility in patients that recover. In some instances, apoplexy into the follicles is caused by torsion of the ovary and consequent obstruction to its blood-supply. Alleged cases of rupture of the ovary from internal hæmorrhage and consequent development of hæmatoma of the broad ligament, or of an intraperitoneal hæmatocele, have been published. Most probably they were, in reality, instances of rupture of an extra-uterine fetal sac.

4. Acute Inflammation.—The ovary is very subject to acute inflammation, oöphoritis being far more frequent than orchitis notwithstanding the external and more exposed situation of the testicle. The poisons developed by infection during child-birth and abortion, as well as the virus of gonorrhœa, are very apt to cause inflammation of the ovary. Primary acute tuberculosis of the ovary seems unknown, but the ovary is easily infected from the Fallopian tube, a common seat of primary tubercular infection. Acute oöphoritis is not often caused by mere chills, as was once supposed. Abscess or diffuse suppuration of the stroma of the ovary may occur in puerperal septicæmia. Gonorrhœal pus from the tube may infect the ovary through a ruptured follicle.

SYMPTOMS.—This disease is usually accompanied by inflammation of the tube and adjacent peritoneum. There is pain over the pubes, tenderness on pressure over one iliac fossa, and often vesical irritation. On bimanual palpation it is easy to detect the tense, enlarged and extremely tender inflamed ovary. In recent cases the ovary is freely movable; but its mobility is often impaired in a few days by adhesions.

TREATMENT.—Rest and appropriate remedies for inflammation will rapidly cure an uncomplicated case of acute oöphoritis. The convalescent patient must be very careful of her health, especially during subsequent periods. The patient should rest on her back, with the hips raised and thighs flexed and supported by a pillow. At first, fomentations over the iliac fossæ will be needed, followed by linseed-meal and turpentine poultices and hot douching. The douche, in some subjects, gives pain and must be discontinued. The patient should take saline purgatives and bromide of potassium. The condition of the ovary must be ascertained from time to time by gentle bimanual palpation. The inflammation is not cured till the ovary is quite painless, and, indeed, hardly to be detected by the touch. Uncomplicated acute oöphoritis never demands operation. Acute ovarian abscess, denoted by persistence of pain and swelling, with rise of pulse and temperature, is best left alone, and allowed to become chronic before it is opened, as at first the pus is very septic. This condition, however, is hard to distinguish from pyosalpinx, and is almost always complicated. An ovarian abscess, if opened when acute, must be reached from the vagina, not through the abdomen.

5. Chronic Inflammation.—This disease is much more frequent than typical acute oöphoritis, and is often associated with intercurrent attacks of subacute inflammation. It can hardly ever be uncomplicated, it tends to set up inflammation of the adjacent peritoneum which causes adhesions, and may also seal up the abdominal end of the Fallopian tube;

on the other hand, it is a very common result of tubal inflammation. The changes induced in the ovary itself by inflammation vary. Sometimes the follicles are completely or functionally destroyed, and the stroma hypertrophies; the ovary feels enlarged, but not very tender. In another form the follicles remain, but cannot burst, then 'small cystic degeneration' sets in. It is hard to distinguish such an ovary clinically from an incipient cystic tumour; there is little tenderness. In a third form true cirrhotic changes occur. This is the gravest variety; it cannot cause death but always sets up local trouble, especially amenorrhœa, and is not rarely the source of bad neuroses. Tuberculosis of the ovary is always secondary to tuberculosis of the peritoneum or tube, and usually assumes the characters of chronic inflammation. A 'cold abscess' sometimes develops in a tuberculous ovary.

TREATMENT.—The treatment must be the same whether chronic inflammation has succeeded an acute attack or has merely increased in intensity or duration after repeated recurrence. Known causes must be avoided, great attention paid to the general health, and counter-irritation employed. Among the best sedatives are belladonna and the bromides. When an ovary, heavy and enlarged from old inflammation, prolapses, an elastic ring-pessary should be applied for a few weeks. The medical attendant should try all means before thinking of removal of the diseased ovary. When there are widespread complications, especially pyosalpinx, the case is different, although in many instances the patient may be cured by medication.

6. Tumours.—Cystic tumour of the ovary is one of the most frequent diseases to which that organ is subject. For practical purposes of diagnosis and treatment cystic tumours of that ovary may be divided into *simple or unilocular*, and *compound or multilocular*. Under the simple form is included *dropsy of the follicles* or *hydrops ovarii*, which is pathologically not a new-growth, but a result of chronic inflammation of the 'small cystic degeneration' type. Yet a dropsical ovary may attain to a diameter of over three inches. As a rule, however, the common ovarian cyst, especially in its most benign and best-known variety, is unilocular only in the sense that one loculus greatly exceeds the others in capacity. It may hold many pints of fluid, while the others contain under an ounce or a drachm; hence they are of no account to the surgeon, as, when the predominant loculus is emptied during ovariectomy, the rest of the tumour can be extracted with the greatest ease if non-adherent. Even in this sense the more unilocular the tumour the more pathologically innocent it will be; one grave exception must be borne in mind, namely, the true *papillomatous cyst*, semi-malignant in its clinical characters. This kind of cyst is often unilocular, and never consists of more than a few loculi. But while the practically unilocular ovarian cyst usually rises freely into the abdomen, the papillomatous cyst proper burrows more or less into the pelvis. In dermoid cysts one loculus sometimes, but not always, predominates.

The multilocular cyst of the ovary is pathologically an adenoma or glandular tumour. It is the commonest form of cystic tumour of the ovary, and is identical with the practically unilocular type just noted, excepting in two important features. No one loculus much exceeds in capacity that of any other; and from the inner wall of the loculi glandu-

lar growths spring freely. The smaller loculi are stuffed with them, so that hardly any cavity is left. On section the growths appear soft and gelatinous, and much of their glairy secretion hangs on to the knife and exudes on pressure. These glandular masses are not malignant; but malignant changes may take place in a multilocular tumour. Sometimes a multilocular cyst is made up of distinct loculi grouped like a pile of old spherical cannon-balls. In such a case the operator should see that the tumour is carefully examined after removal, as it sometimes contains malignant, and often dermoid, elements. *Dermoid cysts* are more frequent in the ovary than in any other organ; in this form one loculus often exceeds the others in capacity. They contain liquid and solid fat, teeth, bone, hair, skin with sebaceous and sweat-glands, and even more complicated structures. The surgeon chiefly regards their greasy, and sometimes fetid, contents, which must not be allowed to escape into the peritoneal cavity. Old adhesions to intestine are specially dangerous, and involve infection of the tumour, and the hair or bone may find its way into the bladder. Ovarian cysts in infants and little girls are usually dermoid.

The *fluid contents* of ovarian cysts was the subject of much study in the days when tapping for diagnosis was practised. When free from blood and broken-down glandular tissue, the fluid of the common ovarian cyst is glairy and greyish, or faint greenish-grey in colour, and contains paralbumen, while ascitic fluid is pale yellow, not very glairy, and contains much serum-albumen. But old hæmorrhages from the cyst-wall turn ovarian fluid dark, and it often assumes a very characteristic chocolate-brown tint. On the other hand, opaque dead-white masses, consisting of collections of glandular matter, are common in the fluid of multilocular cysts. The fluid of dermoid cysts varies; sometimes it is glairy, and bears all the characters of the fluid of a common multilocular cyst, but even then rarely fails to bear fatty globules; sometimes it is pure fat, solidifying on cooling, but most commonly it is a pulraceous mixture of ovarian fluid, fat, and sebaceous matter. Hairs, rarely absent, further betray its character.

In the pure papillomatous ovarian cyst, relatively malignant, and nearly always difficult to remove, and in the parovarian cyst, perfectly innocent and one of the easiest to extirpate of all pelvic tumours, the fluid is alike watery, of very low specific gravity, and holds chloride of sodium in abundance.

Solid Ovarian Tumours.—*Fibroma* is not exceedingly rare. It has often been seen in girls; in mature women it has been mistaken for a pedunculated uterine fibroid, but unlike fibroid it is apt to cause ascites. Its removal is, as a rule, easy, and it is perfectly innocent. *Myoma* is usually small, clinically it resembles fibroma. *Sarcoma*, often bilateral, may appear as a very large solid tumour. Secondary cysts develop, and hæmorrhages occur in its substance. *Carcinoma* is more common in the ovary than sarcoma. The abundant glandular elements in the common ovarian cyst and the epithelium of the papillomatous cyst furnish materials for cancerous changes. As in sarcoma, this disease is often bilateral. *Free papilloma* of the surface of the ovary, never encysted even at its origin, is clinically very malignant. But if the surgeon can extirpate the affected ovary it does not recur. The opposite ovary is usually involved and should be

removed if it bear the least trace of papilloma. If the papillary growth has spread over the adjacent viscera, it will probably destroy the patient, yet instances are known where it has withered after removal of the affected ovary.

COMPLICATIONS.—*Inflammation, hæmorrhage, and twisting of the pedicle.*—Any ovarian cyst, simple or compound, may be the seat of inflammation either on its surface, when the symptoms do not differ from those of peritonitis, or in the cyst-wall or lining membrane, when (without any peritonitis) there may be pain and considerable fever, sometimes followed by rigors and suppuration. Hæmorrhage into one or more of the cyst-cavities may lead to all the symptoms and effects of internal hæmorrhage. The growth and nutrition of the tumour may become greatly modified, from twisting of the pedicle, which obstructs the circulation of blood in the vessels of the tumour. In some cases twisting of the pedicle may be followed by a complete separation of the tumour from its ordinary supply of blood. In this condition the tumour is nourished by vessels in the omentum, abdominal wall, or some other structure adherent by its peritoneal coat to the tumour. True gangrene of an ovarian tumour from twisting of its pedicle is now known to be rare, for the warmth of the peritoneum maintains its vitality, and adhesions rapidly form and carry nutrition to the growth. Acute and sub-acute torsion of the pedicle cause severe abdominal pain, often accompanied with vomiting. When the tumour has already been diagnosed, these symptoms are easy to interpret; in some cases the pain and sickness have led to the detection of the tumour, while in other instances they have been falsely attributed to intestinal strangulation. Inflammation of the cyst, and still more certainly twisting of the pedicle, leads to the development of adhesions to the pelvic and parietal peritoneum, the omentum, bladder, and intestines. *Rupture of an ovarian cyst* is not rare. It is produced sometimes by external agencies, but often more or less through pathological changes in the tumour itself. The thin-walled parovarian cyst is very liable to burst, and this accident may cure the disease, unless papillomatous growths are present inside the tumour, in which case there will be great danger of diffusion of the growths over the peritoneum. The common ovarian cyst sometimes bursts from external violence; even in such cases the immediate effects are seldom alarming; fatal hæmorrhage and even very severe shock have been reported, but rarely occur. The common ovarian cyst is very apt to give way through degenerative changes in its walls, such as inflammatory softening, atrophy of the tissues from changes in its ill-developed vessels and destruction of the stout fibrous tissue in the walls by the free development of minute cysts. Pints of ovarian fluid may escape rapidly into the peritoneal cavity, yet cause remarkably little pain and disturbance. For a short time there is dulness in the flanks and resonance anteriorly, as the patient lies on her back, while before the accident the reverse condition existed. But the effused fluid is rapidly re-absorbed. The rent in the cyst usually heals, or is stopped up by adhesion to adjacent structures, and the cyst fills up again. Allied to rupture, but much rarer and far more serious, are cases where, after adhesion of the cyst-wall to intestine or bladder, the cavities of the cyst and the adherent organ communicate, and the ovarian fluid escapes by the

anus or urethra. The *diagnosis* of rupture of an ovarian cyst is easy when the clinical history is reliable, and it is certain that an ovarian tumour existed before the accident. But when there is nothing certain, save that the patient believes that the abdomen, previously swollen, has grown smaller in a single day or night, there are numerous sources of fallacy. Phantom tumour, tympanites, temporary dullness in the flanks from loaded intestine, and tubercular peritonitis with encysted collections of peritoneal effusion must be borne in mind.

SYMPTOMS AND DIAGNOSIS.—Spencer Wells used to insist that, clinically, the main questions for consideration in cases of fluctuating abdominal tumours are whether the fluid be contained in a cyst, or whether it be in the peritoneal cavity, either free or limited by visceral adhesions. In solid tumours their seat and nature must be investigated. See ASCITES.

A typical ovarian cyst is not hard to diagnose. It lies centrally on the abdomen, rising from the pubes upwards, sometimes reaching as high as the ensiform cartilage. It is freely movable, dull on percussion, and fluctuates. The wave of fluid cannot be made to pass beyond the line of dullness on percussion. In very multilocular cysts there is often difficulty in detecting fluctuation. In a case of ovarian cyst there is resonance in the flanks unless it happen that there be also free fluid in the peritoneum, or that the colon be loaded. Both these conditions have led to error in diagnosis. The cyst usually reaches below to the pelvic brim, the uterus is found, on passing the sound, to be separate from the tumour, and to lie behind and below it; not rarely its body is forcibly anteverted. Some cysts burrow in the broad ligament; then their lower limits can be felt low down in the pelvis, the uterus is pushed to one side, or, very often, displaced forwards and upwards. The abdominal part of a burrowing ovarian cyst is never freely movable.

Other Abdominal Tumours.—The abdominal growths most liable to be mistaken for ovarian tumours are fibroid or fibro-cystic tumours of the uterus, and cysts or solid tumours of the spleen, liver, pancreas, or kidney. Pregnancy, normal or extra-uterine, may also be mistaken for an ovarian tumour. We must remember that it is not rare for pregnancy to occur in a woman subject to an ovarian or uterine tumour; therefore in examining any woman who has an abdominal tumour the ordinary signs of pregnancy must be borne in mind. The frequency of fecal accumulations, or of tympanitic distension of the intestines, with thick or rigid abdominal walls and a fat omentum, must also be remembered and excluded. The observer may be misled by fatty tumours which form in the omentum or develop from appendices epiploicæ. Large fibroid and fibro-sarcomatous tumours of the abdominal wall may be mistaken for solid ovarian growths. Cysts of the urachus are not always easy to distinguish from ovarian cysts. Any condition which produces a swelling in the abdomen or pelvis must be borne in mind when the ordinary signs of an ovarian cyst or tumour are not sufficiently characteristic to exclude doubt. In nervous subjects, where the abdominal muscles resist and interfere seriously with palpation, free purgation followed by careful examination under an anæsthetic is advisable in order to avoid error. In such a case it often happens that no tumour exists, the abdominal swell-

ing being due to tympanitic distension and loaded bowels.

In advanced malignant disease, when the new growth has extended beyond its original seat, it is often impossible to determine whether the ovary gave origin to the malady. In the unfortunate patient's interests the question of diagnosis has become of no import. In such cases, the disease being evident, it is bad surgery to make an exploratory incision. The mere handling of cancerous structures may set up peritonitis; septic infection which cancerous tissue cannot resist is very probable; and if neither complication follows, the abdominal wound is liable to malignant infection through the suture-tracks, and then the patient suffers all the horrors of external cancer from which, if left alone, she would be spared. If there be much dropsy tapping will relieve. On the other hand, when there is no evidence of hopeless malignancy, exploratory tapping is to be avoided. It involves as much or in many cases more dangers than those which attend careful exploration through an abdominal incision.

PROGNOSIS AND TREATMENT.—The thin-walled parovarian cyst, independent of the ovary, has disappeared after rupture or after a single surgical tapping. Thomas Keith advocated the tapping of parovarian cysts, but they often fill again and occasionally contain papilloma, so that tapping involves great risks and loses valuable time. Operation is easy and satisfactory. All true ovarian tumours destroy life after months or even years of discomfort and distressing symptoms; to this rule there are but few if any exceptions. Early removal renders the operation as easy and as safe as possible; late removal involves great surgical difficulties and subsequent danger, yet it may save an otherwise doomed patient. Error in diagnosis often leads to the detection of a tumour not ovarian, but nevertheless quite as necessary to remove.

Medical treatment is now almost synonymous with preparation for operation. In urgent cases ovariectomy must be done at once, however bad the patient's condition. But as a rule there is time for medication. Thus chronic or acute bronchitis and pneumonia may require treatment where there is a cyst or solid tumour not bulky enough to cause dyspnoea. The urine should always be carefully examined. Albuminuria and diabetes mellitus require appropriate treatment. The bowels in particular must be attended to, and the large intestine freed from scybala. Inflammation of a cyst clearly due to a blow or fall is, perhaps, best treated by a few days' rest and local treatment; but most complications signify that ovariectomy is urgent.

Ovariectomy.—The average risk of ovariectomy in a large number of cases, including the most and the least favourable, has been diminishing for many years past. Through improvements first in antiseptic and then in aseptic surgery, ovariectomy has become one of the most successful of all capital operations. The mortality has fallen to under five per cent. in the hands of experts. In the case of simple or non-adherent multilocular cysts, there should be no mortality; occasionally a case may be lost where the patient is suffering from some other disease aggravated by the bulky tumour. Very large and widely adherent tumours, only removable with extreme difficulty, may nevertheless be safely extirpated if the patient be fairly strong, and many very weak subjects can be pulled through by judi-

cious after-treatment. In certain cases there is always a risk of death, so that out of ten such cases one or more will probably be lost. Under this head suppurating tumours, adherent dermoids, and, above all, malignant ovarian growths are included. A cyst containing pus cannot be removed without distinct risk, nevertheless experience shows that the danger is not very great. There is much greater peril when a dermoid closely adheres to intestine so that its greasy contents have become infected. Bone and masses of hair, fat and sebaceous matter may project into hollow viscera, which are unavoidably damaged when the surgeon sets them free. As a rule, however, this unfavourable condition does not exist, and the dermoid can be removed with ease and safety. A malignant tumour may often be removed with success, and the patient may enjoy months or years of relative comfort. But the danger of removing a malignant tumour is great. This fact is proved by statistics collected at institutions where the pathology of the growth and the after-history of the patient can both be accurately observed and recorded. In Olshausen's practice the mortality *more or less directly* from operation in 100 cases of sarcoma, carcinoma, and malignant or clinically malignant papilloma was 28. Such reports are gloomy but they are also reliable.

The removal of a tumour of one ovary does not appear to affect the number of pregnancies, nor the sex of the children, nor the occurrence of twin pregnancy. Spencer Wells was many years ago in a position to note that the removal of both ovaries, subject to tumour, leads only in exceptional cases to obesity, severe premature menopause, and general neuroses. The relation of double ovariectomy for tumours to removal of both ovaries for the cure of neuroses or the checking of the growth of a uterine myoma is uncertain and deserves close study. We know that double ovariectomy is highly successful, and that the patients rarely have anything to complain about afterwards. Removal of the ovaries for neuroses seems unjustifiable and rarely successful, but though severe and aggravated neuroses may follow the operation, they at least existed before, even if only in a milder form. Many advocates of hysterectomy for uterine fibroids, without removal of the ovaries, declare that when those organs are also removed distressing menopause symptoms are very likely to ensue; but this assertion remains to be proved, as it is not certain that the patient, as a rule, fares worse than after double ovariectomy; and where neurotic symptoms have been noted, it is still less certain that they are due to the loss of the ovaries and not to the removal of the body of the uterus and damage to the innervation of the cervix.

ALBAN DORAN.

OVARIES. Herniæ of the.—In the majority of cases displacements of the ovaries occur downwards into Douglas's space, and in such instances the left ovary is the one usually displaced. The next most frequent forms of ovarian herniæ are those occurring in the inguinal regions, either above Poupart's ligament, or, as is more commonly the case, following the canal of Nuck downwards and forwards and presenting in the labia. In the first named, or directly downward displacement, the ovary, on vaginal examination, may be discovered in the recto-vaginal fossa as a small ovoid, firm, elastic, and highly sensitive tumour, bulging into the post-cervical *cul de sac*.

ÆTIOLOGY.—Although in some instances congenital, ovarian herniæ more usually occur in patients whose abdominal parietes have been relaxed and viscera compressed by repeated gestation, or want of due support after parturition. They may also be induced by causes similar to those of other herniæ, such as the violent muscular efforts of the second stage of labour, lifting a heavy child, or straining at stool. In displacement downwards into Douglas's space, the cause of the protrusion is, however, generally gynæcological rather than obstetric, the result of the *vis à tergo* of abdominal or uterine tumours, or of the tension on the appendages occasioned by displacements of the uterus.

SYMPTOMS AND DIAGNOSIS.—Ovarian displacements, when inguinal, may be mistaken for enlarged glands, or as an enterocele or epiplocele; when labial, for other tumours in that situation; when downwards, for pelvic abscess or hæmatocele, retroversion or retroflexion, or for a fibroid growth from the posterior uterine wall. The sudden occurrence of a small ovoid tumour, possessing the physical characteristics just referred to and located in one or other of these situations, accompanied by constitutional and nervous disturbance, with a peculiar dull sickening pain, aggravated into acute suffering at each menstrual epoch, and a coincident increase of size then manifest, together with the intense tenderness or nausea generally evinced during any examination of the part, are sufficient to enable a correct recognition of the nature of the case to be made by any competent gynæcologist.

TREATMENT.—When the ovarian hernia takes place at either of the abdominal rings, it may in some instances be reduced by taxis. In the majority of cases, however, these displacements are already irreducible when discovered; and even in the cases in which reduction is possible, the retentive pressure of an ordinary truss is too frequently neither endurable nor effectual. In such instances the extruded ovary should, if feasible, be protected from further protrusion or external injury by a well-fitting hollow truss. But before this an attempt should always be made to lessen the local hyperæsthesia of the generally hypertrophied displaced gland by topical sedative applications, and, if necessary, by leeching, &c.; while the constitutional irritation almost always present in such cases should be allayed by suitable constitutional treatment. When, however, these measures prove ineffectual in relieving the persistent, dull, worrying, aching pain so commonly associated with chronic ovarian hernia, and which at each monthly period in these cases becomes accentuated or acute—when, too, the patient's health is endangered by the nervous disturbance and constitutional irritation, we should fall back on extirpation as the only resource available. But this treatment should be regarded as exceptional; nor is the performance of oöphorectomy under these circumstances by any means devoid of risk, or to be undertaken without urgent necessity, and until a fair trial has been first made of other remedial or palliative measures.

With regard to the treatment of prolapse of the ovary into Douglas's space, this caution is especially applicable. In considering the management of this displacement, its causes must be carefully borne in view. Pressure from above downwards of a uterine or ovarian tumour, or the traction of a uterine displacement on the broad ligaments, must be removed or relieved before any successful re-position of the

prolapsed ovary can be made. When the dislocation is due to some accidental circumstance, or to a relaxed state of the parts occasioned by constitutional causes, we may, with greater probability of permanent success, attempt to return the displaced viscus and retain it *in situ*. For this purpose the patient—being first anaesthetised in order to permit of the necessary manipulation of the generally highly sensitive and tumefied ovary—should be placed in the left lateral semi-prone position, when by gentle, steady, conjoint digital pressure through the rectum and vagina, upwards and forwards, we may be able to lift the extruded ovary out of the post-cervical recto-vaginal fossa, and to push it up into its normal position, where it may then be retained by a suitable support. Failing the possibility of such re-position, however, if the local and constitutional effects of the displacement are urgent and otherwise irremediable, the practitioner as a *dernier ressort* must remove the ectopic gland.

THOMAS MORE MADDEN.

OVERLYING. — **SYNON.**: Fr. *Etouffer un Enfant*.—Overlying is an accident which, it is alleged, not infrequently happens to young children, whereby they are killed by suffocation. On an average rather more than four hundred children per annum are registered in London as dying from 'overlying' in bed. The Registrar-General in his report for 1890 states that 1,544 children, mostly in the first year of life, died from 'suffocation in bed'; that this mode of death is on the increase; that it is more frequent in the winter than in the summer; and that the proportion of deaths due to this cause is more than twice as high on Saturday night as on any other night in the week. The only explanation for the pre-eminence of Saturday is the fact that on this day wages are paid and drunkenness is common. The *post-mortem* signs of overlying are those of suffocation. Evidence that a child has really died from this cause is afforded by (1) the *post-mortem* appearances of death from asphyxia; (2) the absence of any other mortal disease; (3) the absence of evidence of any cause of asphyxia other than overlying.

The statement that a child has been overlain should be received with caution. It is reasonable to suppose that a vigorous child would escape from a suffocating position beneath the bed-clothes, or the body of its nurse, by its own efforts; or at least succeed, by its crying and struggling, in waking its nurse. On the other hand, a very weakly child, whose lungs possibly have only partially expanded, might be killed by a very trifling cause, such as the position of its mouth and nose against the body of its nurse, or the accidental temporary obstruction of its air-passages by the bed-clothes. A medical witness, before committing himself to a theory of death from overlying, must consider all the points alluded to above, and must take care not to bring a charge of almost criminal carelessness against a careful nurse, or allow an act of wilful murder to pass under the guise of accidental death.

G. V. POORE.

OXALIC ACID, Poisoning by.—See POISONS.

OXALIC ACID IN URINE.—**SYNON.**: Oxaluria.—There is reason to believe that the oxalic acid which is constantly, or almost constantly, present in human urine is in part derived directly from the food, and is in part a product of metabolism

within the body. That the oxalate is in part derived from food has been clearly demonstrated, and analytical results are here in full accord with the clinical observation that a deposit of crystals of calcium oxalate is wont to follow the ingestion of articles of diet, such as rhubarb, which are specially rich in this substance. The view that some of the oxalate excreted is a product of metabolism is based upon observations, chiefly carried out in Salkowski's laboratory, on its presence in the urine of fasting dogs, and in that of human beings who have for some time taken an oxalate-free diet. It should be mentioned, however, that Gaglio, Bunge, and Dunlop have observed the disappearance of oxalic acid from the urine of persons on diet of this description. Granting that some of the excreted oxalate is endogenous, it is clear, from the observations of Salkowski and others, that it is not a product of the metabolism of carbohydrates or of albuminous substances; whereas a food rich in nuclein, such as thymus, was found by Lommel to cause a conspicuously increased output. Salkowski therefore thinks it highly probable that both oxalic and uric acids are products of nuclein-metabolism, the former being probably formed by the oxidation of the latter. However, Stradomsky, working with Salkowski's new method of estimation, failed to obtain an increased excretion of oxalic acid as the result of a diet rich in nucleo-albumen, and looks upon the gelatin in the tissues and kreatin as the most probable parent-substances of the endogenous oxalic acid. As to the influence of disease upon oxalate excretion little is at present known, and it is clear that any such influence will be chiefly exerted upon that portion of the excreted oxalate which is formed within the body. Diabetes mellitus and jaundice are conditions in which increased excretion has been noted, but Lommel has recently failed to confirm this observation.

The acid sodium phosphate appears to be the agent concerned in holding calcium oxalate in solution in the urine, and it cannot be assumed, without direct proof, that the deposition of crystals of the latter salt necessarily implies its presence in excess. However, Dunlop found that when such deposition takes place there is usually an increased output.

In paroxysmal hæmoglobinuria sediments of calcium oxalate are frequently met with, as also in many cases of so-called 'physiological albuminuria.' Prout and Begbie called attention to a group of symptoms which are often associated with deposits of these crystals, and described a definite 'oxalic acid diathesis.' The symptoms described are such as usually accompany acid dyspepsia, and Dunlop seeks to explain their association with oxaluria by the more free absorption of calcium oxalate from the food, in consequence of the hyperacidity of the gastric contents. Such symptoms are met with apart from oxaluria, and are often wholly wanting in cases in which the urine continuously deposits a sediment of calcium oxalate.

The deposited crystals are usually minute, and are not coloured by the ordinary urinary pigments; they may, however, be tinted by bilirubin or biliverdin when these substances are present. The crystals assume several forms—(a) the common octohedra with short vertical axis, the familiar 'envelope' crystals; (b) short prisms with pyramidal ends; (c) twin octohedra; (d) dumb-bell crystals or biconcave oval discs (see URINARY DEPOSITS: coloured plates). The deposit usually forms a white

cloud resting upon the surface of the normal nodule, and may also form streaks upon the inner surface of the containing vessel. The urine is usually clear and acid in reaction. It may contain a small quantity of albumen, and the microscope may show, in addition to the crystals, a few leucocytes and even red blood-corpuscles. Free indulgence in rhubarb may even give rise to marked hæmaturia, with the passage of large numbers of calcium-oxalate crystals, and some individuals appear to be peculiarly susceptible in this respect.

Oxalate-of-Lime Calculus.—The excretion of calcium oxalate derives its chief clinical importance from its tendency to form calculi in the kidneys or urinary passages. What are the exact conditions which determine the formation of such calculi is not as yet known, but a patient whose urine deposits oxalate-sediments during long periods does not necessarily acquire calculi. Ord and Shattock, who have made a very elaborate study of the structure of these concretions, describe four chief forms: (1) Smooth polished stones, usually met with in the kidneys or renal pelvis, of a pale ashy grey, light yellow or reddish-brown colour. (2) The ordinary 'mulberry' calculi, of a reddish-brown or sepia tint, with rough tuberculated surfaces. (3) Small stones with sharp prominent spines, and (4) small stones with crystalline surfaces beset with large uncoloured crystals of calcium oxalate. Calculi of the two last-mentioned varieties are often quite minute. The dark colour which oxalate-calculi often possess appears to be derived from hæmoglobin. The stones are hard and crystalline in structure, and the nuclei often consist of large transparent crystals of calcium oxalate, or of fan-shaped elements similar to those which form the bulk of the concretion. Speaking of such calculi Mr. Cadge says, 'The great insolubility of oxalate

of lime favours the chances of its deposition in the renal tubules. It has been detected in the kidneys of the fœtus; it is especially liable to occur during childhood; and this liability decreases as age advances. In England calculi composed entirely of oxalate of lime are rare in the adult, but in India they are comparatively frequent. Mulberry calculus in the young causes intense suffering; but in the adult, notwithstanding the formidable tubercles and rough exterior, the symptoms of stone are often mild: not improbably because these projections become entangled in the muscular columns of the bladder, and the calculus is thereby fixed in position.' The symptoms of renal calculus are fully described in another article. See RENAL CALCULUS.

TREATMENT.—Microscopic 'mulberry' calculi, were it possible to detect their existence, could probably be washed away and carried off by diluents and diuretics; but a palpable stone demands surgical treatment only.

That part of the oxalate-excretion which is directly derived from food can, of course, be controlled by suitable dieting, the most obvious indication being the avoidance of vegetables, such as rhubarb and spinach, which are known to be rich in oxalate. The probability of the origin of some of the excreted oxalic acid from uric acid and nuclein suggests the desirability of avoiding food rich in nucleo-albumens. Hyperacid dyspepsia when present should be treated by appropriate means, such as the administration of acids before, or alkalis after meals.

A. E. GARROD.

OXYURIS.—See ENTOZOA.

ÖZ/ENA (ὄζαινα, a fœtid polypus in the nose; from ὄζω, I stink).—See NOSE, Diseases of.

P

PACHYDERMIA LARYNGIS.—See LARYNX, Diseases of, p. 853.

PACHYMENINGITIS (παχύς, thick; and μῆνιγξ, a membrane).—A synonym for inflammation of the dura mater of the brain and of the spinal cord. See MENINGES, CEREBRAL, Diseases of.

PAGET'S DISEASE.—Malignant dermatitis of the nipple (see NIPPLE, Diseases of). The term is also sometimes used as a synonym for osteitis deformans.

PAIN IN VISCERAL DISEASE.—The pain of visceral disease may belong to one of three groups: (1) It may be as local as the pain produced by a bruise or a burn, and be situated directly in the affected part. Such local pain, associated with deep tenderness over the inflamed area, accompanies inflammation of any serous membrane, such as the pleura, peritoneum, or pericardium. (2) It may be referred to the termination of some nerve-trunk implicated in the disease, as, when the elbow is struck, the pain is felt in the little finger. Thus, if a nerve-trunk passes through an inflamed area, the pain will

be felt not only at the inflamed point, but also within the peripheral distribution of the affected nerve. Moreover, the area supplied by the peripheral branches of this nerve may become tender to light touch. In this way the pain of a pleurisy may be situated in the abdomen. (3) Painful impressions may be conducted up the sensory fibres of the sympathetic into the central nervous system, and then referred to the peripheral distribution on the surface of the body of those nerve-fibres that enter the same segment. This is the true visceral reflected pain that forms the main subject of this article.

§ 1. **Method of Examination.**—In visceral disease we have to deal mainly with pain that is either local or reflected. *Local pain*, as its name implies, is characterised by being situated over the focus of disease, and is usually accompanied by tenderness on deep pressure or on percussion.

True *visceral referred pain* is situated over spots of the body that bear no direct relation to the position of the disease. Should the pain be severe, or have lasted for some considerable time, these spots possibly will become superficially tender. Gently pinching the skin and subcutaneous tissue over these spots between the finger and thumb then produces

PLATES X. and XII.

The areas coloured Red or Blue are those affected by the reflected pain and superficial tenderness of visceral disease. Those coloured Black do not stand in direct connection with any organ and can only become tender by spread. They form the upper gap (on the arm) and the lower gap (on the leg).

The following organs stand in connection mainly with the following areas :—

<i>Heart</i>	1. Ventricle	Dorsal 1 (?), Dorsal 2, 3, 4, 5.
	2. Auricle	Dorsal 5, 6, 7, 8 and (?) 9.
<i>Aorta</i>	1. Arch	Cervical 3 and 4, Dorsal 1, 2, 3, 4.
	2. Dorsal Aorta	Dorsal 5, 6, 7, 8, 9.
	3. Abdominal Aorta	Dorsal 10, 11, 12 and Lumbar 1.
<i>Lungs</i>		Cervical 3, 4, Dorsal 3, 4, 5, 6, 7, 8, 9.
<i>Esophagus</i>		Dorsal 5, 6, 7.
<i>Stomach</i>		Dorsal 6, 7, 8, 9, 10.
<i>Intestine</i>	1. Duodenum to Sigmoid Flexure	Dorsal 10, 11, 12.
	2. Rectum	Sacral 2, 3, 4.
<i>Liver and Gall Bladder</i>		Dorsal 7, 8, 9, 10 (R. side).
<i>Kidney and Ureter</i>		Dorsal 10, 11, 12, Lumbar 1, and Occipital Lumbar 2.
<i>Bladder</i>		Sacral 2, 3, 4.
<i>Prostate</i>		Sacral 2, 3, 4, and Occipital Dorsal 10 and (?) 11.
<i>Testicle</i>		Dorsal 10.
<i>Epididymis</i>		Dorsal 11, 12.
<i>Ovary</i>		Dorsal 10.
<i>Uterine Appendages</i>		Dorsal 10, 11, 12, Lumbar 1, and (?) Lumbar 2.
<i>Cervix Uteri and Lower Segment of Uterus</i>		Sacral 2, 3, 4.

	Ventricle	Auricle	Lungs	Stomach	Intestine	Rectum	Liver and Gall-Bladder	Kidney and Ureter	Bladder	Prostate	Testicle	Epididymis	Ovary	Appendages	Cervix and Lower Segment Uterus
Cervical 3			x	?											
Cervical 4			x	x			x								

Here follows the upper gap of areas not affected directly in visceral disease (Cervical 5, 6, 7, 8).

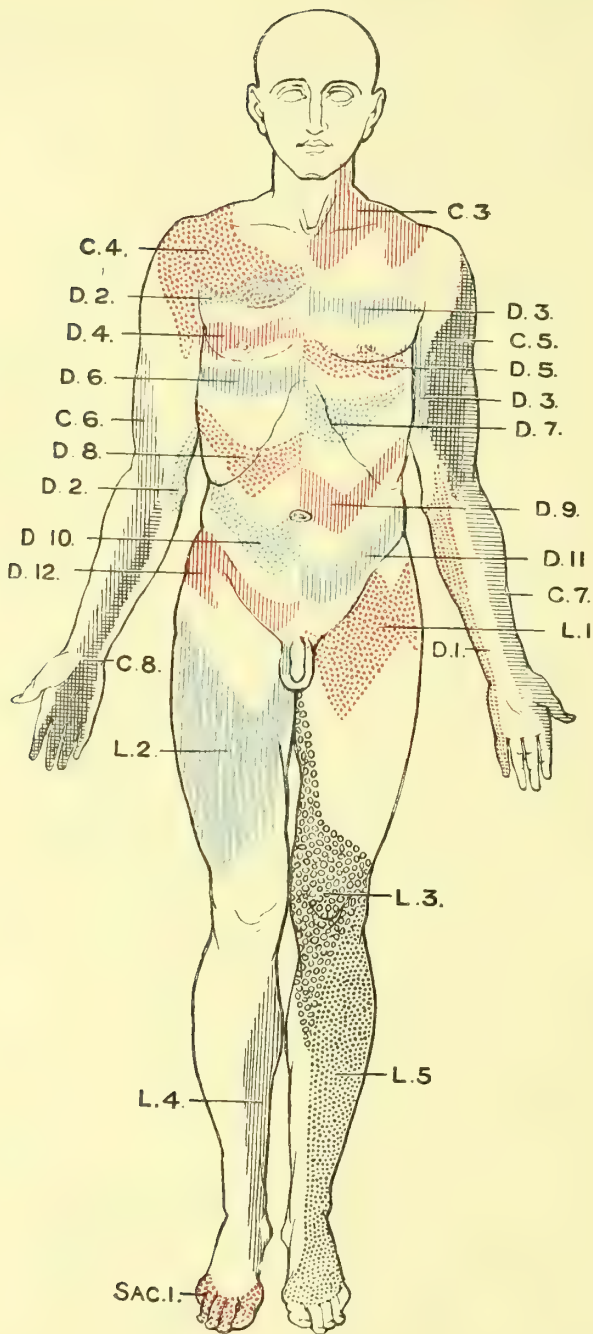
Dorsal 1	?														
Dorsal 2	x		?												
Dorsal 3	x		x												
Dorsal 4	x		x												
Dorsal 5	x	x	x												
Dorsal 6		x	x	x											
Dorsal 7		x	x	x			x								
Dorsal 8		x	x	x			x								
Dorsal 9		?	x	x			x								
Dorsal 10				x	x		x	x		x	x		x	x	
Dorsal 11					x			x		?		x		x	
Dorsal 12					x			x				x		x	
Lumbar 1								x						x	
Lumbar 2								Occ. x						?	

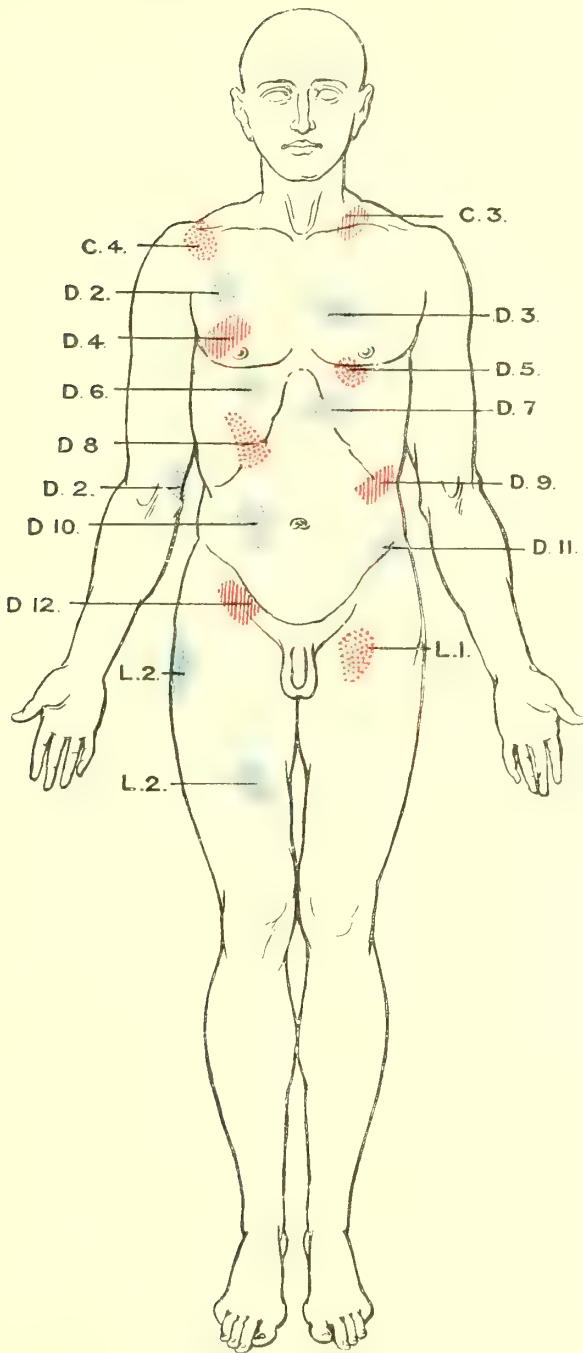
Here follows the lower gap of areas not affected directly in visceral disease (Lumbar 3, 4, and ? 5).

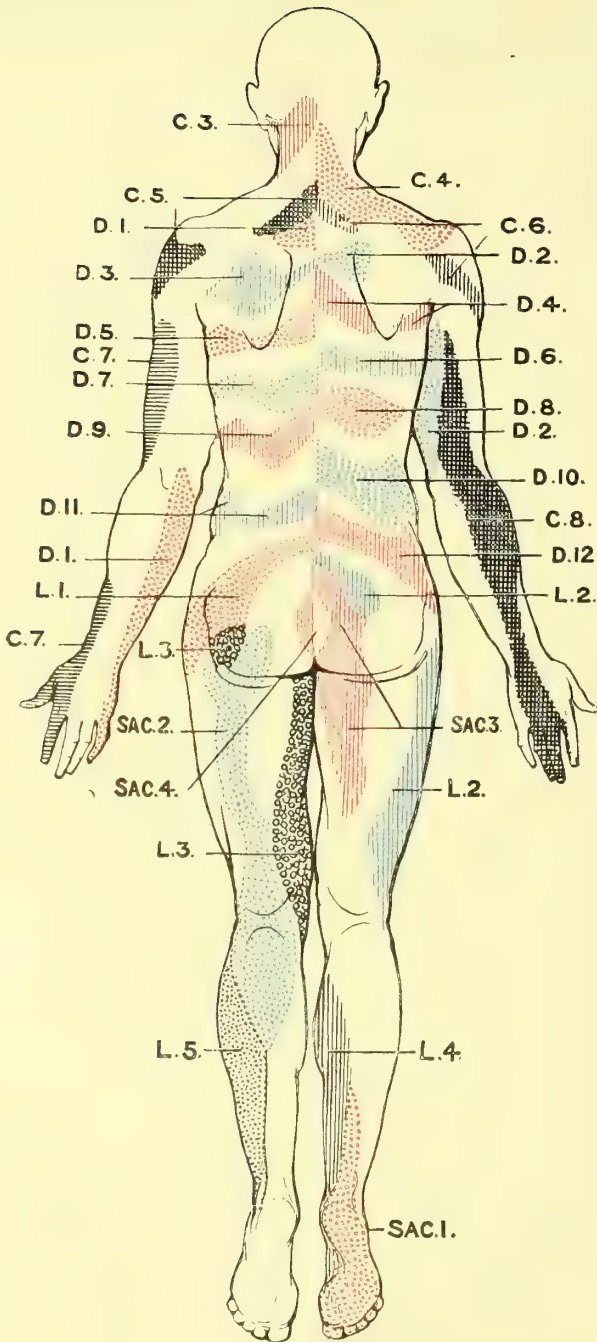
Sacral 1															
Sacral 2						x			x	x					x
Sacral 3						x			x	x					x
Sacral 4						x			x	x					x

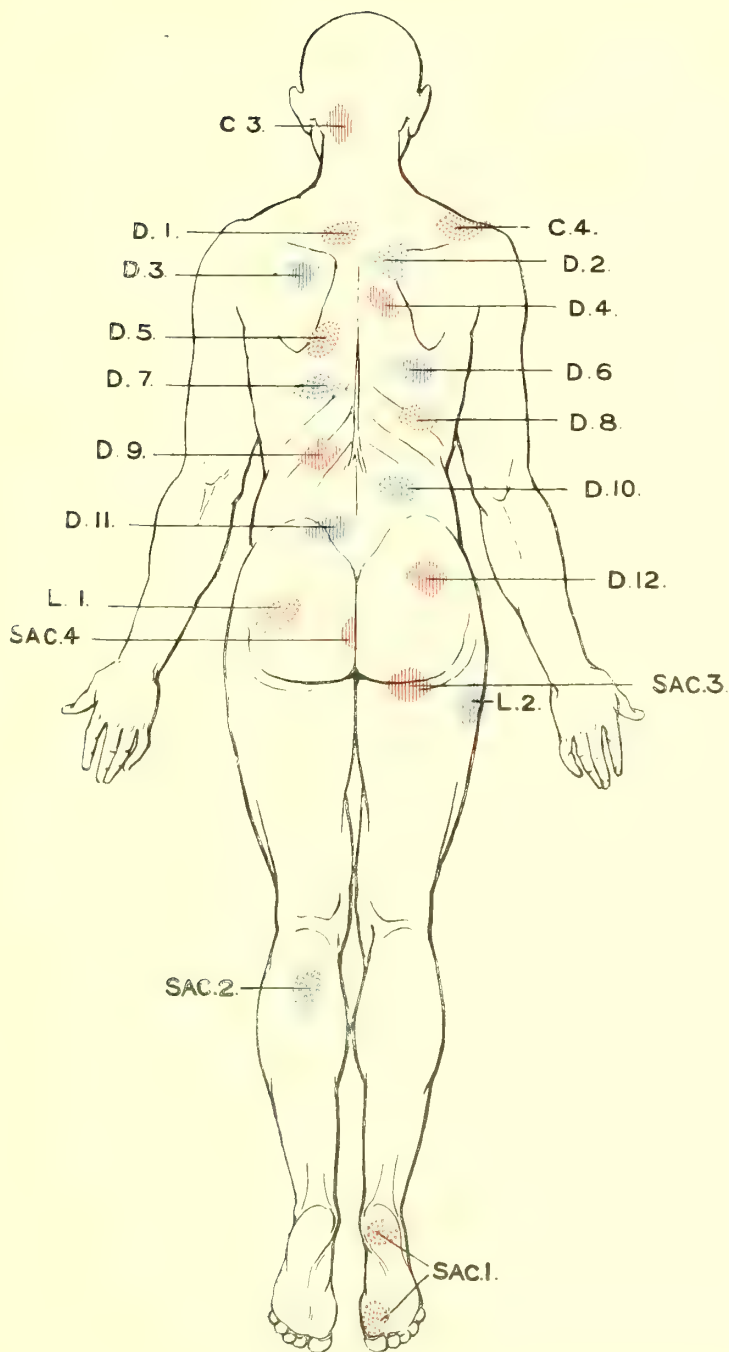
PLATES XI. and XIII.

These plates show the maximum spots of these areas. It is to these spots that the patient refers his pain. Over these spots superficial tenderness first appears, and they remain tender during the subsidence of the pain after the rest of the area to which they belong has ceased to be tender. Thus recognition of these maximum spots is of greater practical importance than that of the full areas which rarely appear alone in visceral disease.









pain, or, at any rate, a sensation different from that produced in normal parts of the skin by this manipulation.

A still better method of marking out the tender spots is to take a pin with a round head of such a size that it is obviously blunt to the normal skin, using the blunt end only in exactly the same way as if the point was being used to test for loss of sensation. In a favourable case the patient does not complain until the limits of the tender areas are reached, when he at once complains that it hurts, and may even cry out that he is being pricked. But in the majority of cases the patient says he knows the blunt end of the pin is being used, but it gives him pain, 'as if a bruise were being touched.' Over these areas the superficial reflexes are markedly increased. But these tender spots represent the maxima of areas that extend round the body more or less in the form of a half girdle, and, if the pain be very intense, or has lasted for a sufficient period, the whole of one or more of these segmental areas may become tender. But for clinical purposes it is a matter of little or no importance whether one of these segments be represented by the complete area of its extent, or by the maximum points.

Thus, in Plates XI. and XIII. the maximum points are given, and on Plates X. and XII. the complete areas determined by the study of the tenderness of referred pain, and the distribution of the eruption of herpes zoster (*see* p. 686).

§ 2. Reason for Cutaneous Tenderness in Visceral Disease.—When impulses pass up sensory sympathetic nerves from an organ which is diseased they set up a disturbance in the segment to which they are conducted. Now any second sensory impulse from another part, e.g. from the surface of the body which passes into this same segment, will be profoundly altered. For it no longer falls into a normal and quiescent segment of the nervous system, but into one whose activity is already disturbed. The resultant stimulus, conducted upwards towards the brain, therefore differs from that which would have passed onwards from that segment under normal circumstances. The second stimulus will appear to be exaggerated, or may perhaps undergo some actual increase in its passage through the excited segment. Thus any otherwise painless stimulation applied to the surface of the body, falling within the area supplied by fibres that enter the disturbed segment, will appear to be painful, and the skin will be said to be tender. The fact that these areas correspond so closely in extent with those occupied by herpes zoster renders it probable that they belong to the same level of the nervous system, and that the cause of the tenderness is to be sought in that portion of the nervous system which, when injured, produces herpes zoster. This has now been shown to be the ganglion of the posterior root. It is therefore probable that impulses passing from an affected internal organ up the white ramus of the sympathetic system produce an alteration in some of the cells of the posterior-root ganglion. These cells form the trophic stations for the sensory fibres from the skin. Thus painful stimuli from an internal organ produce such a disturbance in the ganglion that every stimulus from the peripheral distribution of the fibres entering that ganglion appears to be painful.

§ 3. Pain produced by Disease of Special Viscera.—Brought face to face with pain produced by disease of an internal organ, it is

necessary to determine first of all whether it be local or reflected. Attention to the following points of difference enables us to decide to which class it belongs:—

(1) Reflected pain is not only situated over some point on the front of the chest or abdomen, but also over some point behind, in the back or the loin, away from the affected organ.

(2) Reflected pain is associated with more or less tenderness of the coverings of the chest, abdomen, or back, elicited by picking up the skin and subcutaneous structures between the fingers.

(3) This superficial tenderness frequently passes round the body in a more or less horizontal band from the middle line of the back to the middle line in front (Plates X. and XII.). If less acute it lies over one or more spots, one of which is situated over the anterior, the other over the posterior aspect of the trunk (Plates XI. and XIII.).

(4) Pressure as a rule relieves this superficial tenderness.

(5) The superficial reflexes are increased over an area of superficial tenderness that accompanies visceral reflected pain.

(6) Reflected pain of the visceral type is frequently accompanied by headache and superficial tenderness of the scalp, following rules laid down on p. 1145.

(1) Local pain is confined strictly to the area of the affected organs, and does not pass round the body or through to the back.

(2) Local pain is accompanied by no superficial tenderness. But when the painful area is pressed or percussed pain is produced, the amount of such tenderness depending on the force applied.

(3) This deep tenderness does not extend beyond the limits of the serous membrane inflamed or outside an area over which direct pressure can be brought to bear on the serous membrane or organ affected. It cannot therefore be elicited over the posterior aspect of the trunk unless the inflammation has extended into the tissues of the back.

(4) Pressure increases deep tenderness.

(5) The superficial reflexes are unaltered over the area of deep tenderness that accompanies local pain.

(6) Local pain is not accompanied by that form of headache which is associated with tenderness of the scalp. Headache, if present, is a separate phenomenon, standing in no relation to the local pain and deep tenderness.

The organs of the body will be considered in the following order: Heart, p. 1139; Aorta, p. 1141; Lungs, p. 1141; Oesophagus, p. 1142; Stomach, p. 1142; Intestines, p. 1142; Liver, p. 1142; Kidney and Ureter, p. 1142; Bladder, p. 1143; Prostate, p. 1143; Testis, p. 1143; Epididymis, p. 1143; Ovary, p. 1143; Uterus, p. 1143.

1. Heart.—(a) Local Pain.

(1) *Due to Disease of Pericardium.*—When the pericardium is acutely inflamed the pain produced is in striking contrast to visceral reflected pain. For it is not accompanied by superficial tenderness; but over a greater or less extent of the pericardium the patient complains that percussion or pressure with the stethoscope in the intercostal spaces is painful. This deep tenderness does not transgress the limits

of the pericardium. In many cases this area of deep tenderness corresponds closely to the area over which pericardial friction is audible. Such is the condition not uncommonly found in a recent case of a first attack of pericarditis. Sometimes, however, this condition is complicated by the simultaneous appearance of referred pain and superficial tenderness due to the concomitant endocardial lesion (*vide infra*).

(2) *Due to Disease of Heart*.—Local pain is supposed to be commoner in cardiac diseases than reflected pain. Probably local pain is a distinct feature of the majority of painful cardiac states, but it is usually masked by co-existing reflected pain. But in some cases of cardiac failure when reflected pain is absent local pain is distinct. The patient complains of pain over the apex-beat that does not go through to the back and is increased by exertion. This form of pain is particularly marked in certain cases of mitral stenosis with an irregular beat, and in the form of senile heart that is accompanied by breathlessness and palpitation on exertion. The heart is said to feel as if it were 'bursting,' 'too big for the chest,' &c.

(b) *Reflected Pain*.

(1) *Due to Disease of Aortic Valves*.—True aortic stenosis throws more work on the wall of the left ventricle during systole. The ventricle therefore hypertrophies, compensation takes place, and the patient suffers from no symptoms. Not so aortic regurgitation. For here the ventricle empties itself during systole, but as soon as the wall relaxes at the beginning of the diastole the blood pours back from the aorta and distends its cavity. In true aortic stenosis the ventricle has to perform increased work during the systole, but is at rest during diastole. In pure regurgitation, on the other hand, there is not necessarily any increase in the work required during systole; but just at the moment when the wall is not toned to meet any effort the blood pours back into its cavity from the aorta. Now it is this distension at the moment when the walls of the cavity are at rest that is pernicious, and aortic regurgitation is of all cardiac disorders the

one most liable to lead to reflected pain. When both regurgitation and stenosis are present the condition is still worse. For not only has the systole to overcome a greater intra-ventricular resistance, but the diastole is broken by a rush of blood back from the aorta. The left ventricle hypertrophies to meet this increased resistance in front, but the increase in the force of the ventricular contraction will also increase the force with which the blood rushes back from the aorta at the beginning of the diastole. Thus reflected pain is more likely to be present with aortic regurgitation than stenosis, but most certainly when both lesions are combined. But pressure within the cavity of the ventricle during the diastolic period gives rise to a second evil. The ventricle never quite empties itself, but always remains somewhat distended, an ideal condition for the production of visceral reflected pain. In most cases this is a temporary condition, arising from some additional exertion in a heart working at the top of its power. In young people some excessive effort is necessary to produce this condition, but in older persons, and especially in those cases where the lesion of the aortic valves is caused by atheroma, it may arise after but slight exertion.

This permanent dilatation of the left ventricle cannot exist long or to any great extent without the mitral valve becoming incompetent. Directly this happens the pressure within the ventricle is relieved, for blood can now leave it by two channels. Thus, as soon as the mitral valve yields, the pressure within the left ventricle is relieved by a self-regulating mechanism, and reflected pain ceases. In the same way reflected pain is absent in those cases where mitral regurgitation arises synchronously with the lesion of the aortic valves.

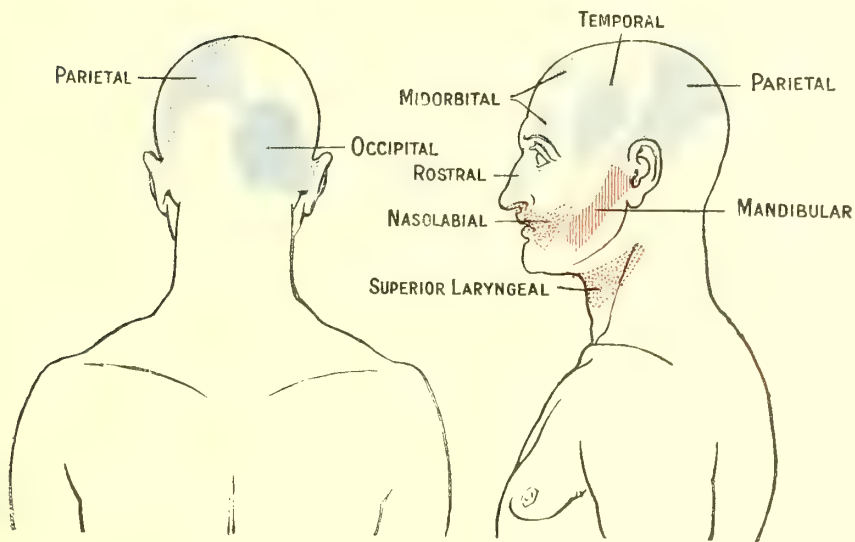
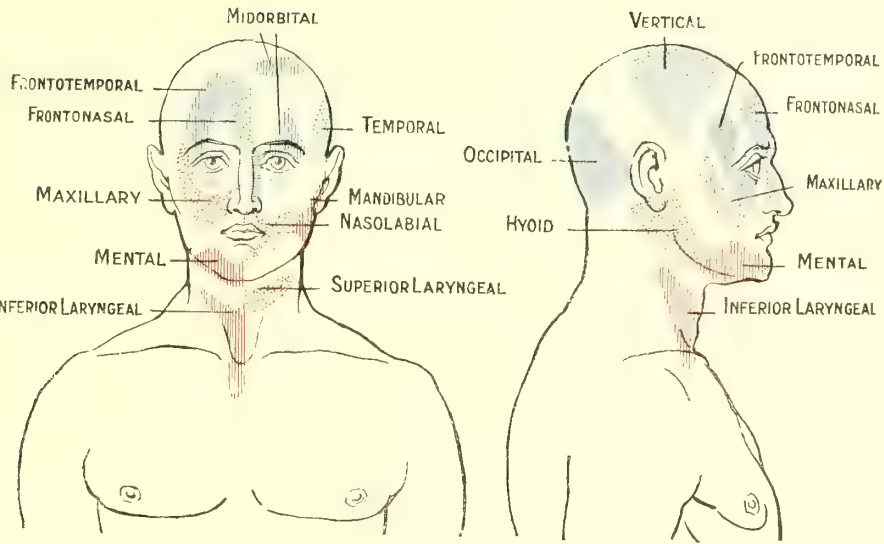
Thus it will be seen that pain produced by aortic disease is due to the left ventricle. It is situated over the upper part of the chest, over the scapula, and down the inner side of the left arm. If severe and of long standing, it may cross to the right side in addition. In some cases it may become paroxysmal (*see below*). It is accompanied by more or less superficial tenderness over the second, third,

PLATE XIV.

The areas coloured Blue stand in relation both to organs of the head (e.g. the eye) and organs of the trunk (e.g. the stomach). Those coloured Red stand in direct connection only with organs of the head—

<i>Eye</i>	1. Cornea and Ant. Chamber	Frontonasal and Midorbital.
	2. Ciliary Region	Mainly Midorbital—also whole forehead if acute.
	3. Iris and Post. Chamber	Mainly Frontotemporal. Temporal and Maxillary.
<i>Teeth</i>	1. Upper Jaw	Incisors. Frontonasal. Canine. ? Nasolabial. 1st Bicuspid. Nasolabial. 2nd Bicuspid Temp. or Maxil. 1st Molar Maxil. 2nd and 3rd Molar Mandibular.
	2. Lower Jaw	Incisors, Canine, 1st Bicuspid. Mental, 2nd Bicuspid? 1st and 2nd Molar, Hyoid. 3rd Molar, Hyoid or Superior Laryngeal.
<i>Ear</i>	Middle ear	Hyoid.
	Internal Ear and Mastoid (with Tension only)	Vertical and Temporal.
<i>Tongue</i>	Mental, Hyoid, Laryngeal and Occipital.	Occipital.
<i>Tonsil</i>	Hyoid.	
<i>Nose</i>	Frontonasal and Midorbital (mainly).	

Area on the Scalp	Area on the Trunk	Organs in relation with these Areas
Frontonasal	Cervical 3	Lungs [Apices], Stomach, Liver (Occipital)
	Cervical 4	
Midorbital	Dorsal 2	Heart [Ventricle], Aortic Arch, Lungs
	Dorsal 3	
	Dorsal 4	
Frontotemporal	Dorsal 5	Lung, Heart
	Dorsal 6	Lungs [lower lobes], Heart [Auricle]
Temporal	Dorsal 7	Lungs [lower lobes], Heart [Auricle], Stomach (Cardiac end), Liver (R. side)
Vertical	Dorsal 8	Stomach, Liver and Gall-Bladder, Lungs
Parietal	Dorsal 9	Stomach (Pyloric end), Liver
Occipital	Dorsal 10	Intestine, Liver, Ovary, Testis, Stomach (Occipital)



fourth, and fifth dorsal segments (*see* Plates X. and XII.).

(2) *Due to Disease of Mitral Valves.*—In cases of mitral incompetence a free path is open into the pulmonary circulation, and the pressure in the ventricle, therefore, always remains low. Cases of pure mitral incompetence are unattended by visceral referred pain. In a similar way in uncomplicated *mitral stenosis* no referred pain is produced. But suppose that, as soon as the auricular systole has expelled the blood through the narrowed mitral orifice, blood is again forced back into the auricle by the ventricular systole, the auricle will be distended at the moment when its walls are relaxing, and pain will result. Thus, where a systolic murmur is heard over the apex, accompanied by the short sharp first sound of stenosis, with or without a diastolic murmur, reflected pain and superficial tenderness are liable to make their appearance. In such cases it is the auricle which bears the brunt of the altered conditions, and the pain and tenderness lie over one or more of the fifth, sixth, seventh, eighth, and possibly ninth, dorsal areas.

(3) *Paroxysmal Pain of Cardiac Origin* (*see also* ANGINA PECTORIS).—Angina Pectoris is not a disease, but a convenient name for a group of symptoms that make their appearance in paroxysms. A typical attack of angina pectoris is made up of one or more of the following groups of symptoms: (1) A sense of faintness or impending death; the face seems to become cold and the jaws stiff, and a dead feeling to spread over the body. (2) A feeling of suffocation accompanied by local pain at the epigastrium or over the heart. (3) Radiating pains mostly over the upper part of the chest and in one or both arms. These pains follow the lines laid down in figs. 1 and 2, are accompanied or followed by superficial tenderness, and are thus of the true visceral reflected type. These symptoms are present to a different degree in individual cases. Thus in those cases where the arteries are degenerated but the heart is free from endocardial murmurs, the first and second symptoms are frequently present alone. Where, however, the degenerate arteries and myocardial signs are accompanied by aortic murmurs the third is usually added in marked form. In young persons with marked aortic regurgitation of rheumatic origin (unaccompanied by mitral regurgitation) attacks of paroxysmal cardiac pain consist almost exclusively of violent radiating pains of the reflected type unaccompanied by the first symptom, and usually by the second to a comparatively slight extent.

Thus we obtain the following rule: If the patient habitually suffers from reflected pain of cardiac origin the attack of angina pectoris will consist of a paroxysmal increase in the intensity and distribution of this pain. If, on the other hand, he is habitually free from visceral reflected pain the attack will more closely resemble sudden heart-failure, and will be accompanied by a feeling of faintness or impending death with more or less local pain at the epigastrium and over the area of the heart.

2. *Aorta.*—An aneurysm of the aorta may produce three forms of pain: (1) Local pain due to pressure on the vertebrae or ribs. This only occurs with aneurysms of considerable size. (2) Pain due to pressure on nerve-trunks which is referred to the peripheral distribution of the sensory fibres of those nerve-trunks, exactly as when the elbow is struck the pain is referred to the little finger. (3) Visceral reflected pain produced according to the general

laws laid down above, the aorta acting as a hollow organ. Visceral referred pain can only be produced so long as the aneurysm is, at any rate in part, contained within a wall composed of the tissues of the aorta. As soon as the aneurysm reaches such a size that its walls are mainly composed of connective tissue, visceral referred pain ceases owing to the destruction of the visceral nerves necessary for its production. Thus visceral referred pain is more commonly present in aneurysms of small size.

The position of the visceral referred pain and its accompanying superficial tenderness vary according to the position of the aneurysm. Thus the nearer the aneurysm lies to the aortic valves the more nearly the pain and tenderness resemble that produced by aortic regurgitation. If the aneurysm is situated at any part of the arch between the heart and the entry of the ductus arteriosus, the superficial tenderness may lie over the first, second, third, or fourth dorsal, or third and fourth cervical areas. Occasionally the areas on the front of the neck seem also to be affected. As soon, however, as the aneurysm invades that part of the aorta on the distal side of the entry of the ductus arteriosus these upper segmental areas are replaced by pain and tenderness over the fifth, sixth, and seventh dorsal areas. The lower the aneurysm on the dorsal aorta the lower the segment mainly affected. Thus aneurysms of the abdominal aorta may produce pain over the ninth, tenth, eleventh, twelfth dorsal, and first lumbar areas. Pain and tenderness over these areas may be extremely misleading. For if it lie over the twelfth dorsal or first lumbar area the patient will complain of pain over the extreme lower part of the lumbar spine, in the groins, and over the front of the thigh. Yet the swelling and pulsation will be felt at or above the umbilicus.

3. *Pain in Lung-Disease.*—(a) *Local Pain.*—Pain of this type occasionally appears in the neighbourhood of tubercular foci or large areas of pneumonic consolidation, but in most cases stands in definite relation to inflammation of the pleura. It is absent when the side is at rest, is intensified by inspiration, particularly by the long inspiration which follows a cough, a laugh, or a sneeze. The pain is said to be stabbing, but does not go through the chest from back to front as does referred visceral pain, and is localised to one area instead of being distributed over a band around the body. This pain is associated with deep tenderness to pressure or percussion in those intercostal spaces within which pleural friction can be heard. If the pleurisy is acute the intercostal trunks may become implicated in the inflammation, and pain appear over their terminal distribution exactly as pain appears in the little finger when the ulnar nerve is injured. Thus an acute pleurisy may cause pain over the abdomen or even the iliac fossa of the same side. This is not a visceral reflected pain, for it is only present over that portion of the nerve-trunk distal to the inflammatory lesion, and is not present over the back. It may occasionally be accompanied by superficial tenderness. But, unlike the superficial tenderness that is associated with true visceral reflected pain, the tenderness that may accompany a pleurisy is only present in front of (on the distal side of) the patch of pleural inflammation.

(b) *Visceral Reflected Pain.*—In order that reflected pain may appear it is necessary that the disease should not render the nerve-end organs in the lungs incapable of stimulation. Now in *acute*

lobar pneumonia the essential process of the disease is a consolidation of one or more lobes of the lung and complete obliteration of the alveoli by exudation. Thus the majority of cases of acute lobar pneumonia are unaccompanied by visceral reflected pain and superficial tenderness owing to the fact that the nerve-terminals are removed from the influence of internal pressure or external traction in consequence of consolidation of large areas of lung-tissue. In the same way if the lung has undergone *fibrotic changes* (as in the so-called fibroid phthisis) it is no longer capable of causing visceral referred pain. *Excavation of the lung*, by the destruction it produces, also renders the affected portion of the lung incapable of producing visceral referred pain, and thus those cases of pulmonary tuberculosis in which excavation forms the principal lesion may be painless. On the other hand, acute bronchitis may be accompanied by visceral reflected pain if the lung has not been altered by repeated previous attacks. In the same way visceral reflected pain accompanies those cases of pulmonary tuberculosis in which the disease advances by repeated bronchitic attacks, each of which leaves the lung more damaged than before. In such cases reflected pain and its accompanying superficial tenderness are frequently a marked feature in the progress of the disease.

The pain and tenderness of pulmonary disease occupy one or more of the following areas: Cervical, 3 and 4; dorsal, 3, 4, 5, 6, 7, 8, and 9. The nearer the disease to the apex of the lung, the more likely are cervical 3 and 4 to be represented in superficial tenderness, the nearer to the base the more certainly will the pain and tenderness lie over the mid-dorsal areas. Thus it is noticeable that disease of the base of the lung can cause pain and superficial tenderness over the three great gastric areas, the seventh, eighth, and ninth dorsal. If pain and tenderness over these areas is a marked feature of lung-disease the patient will suffer from secondary gastric symptoms (*vide* § 5).

4. **Œsophagus.**—As a rule there is not much referred pain in diseases of the œsophagus. But in a case of stricture of the lower part of the œsophagus pain and superficial tenderness may make their appearance over the fifth, sixth, or seventh dorsal areas after passage of a bougie.

5. **Stomach.**—In diseases of the stomach the seventh, eighth, and ninth dorsal areas are frequently mapped out by superficial tenderness to a greater or less extent. Sometimes the sixth dorsal makes its appearance in company with the seventh dorsal, and the tenth may appear with the ninth. Thus the whole supply of the stomach seems to lie between the sixth and tenth dorsal. In cases where pain is present after the ingestion of food, as in gastric ulcer, the higher the area of pain and tenderness, the sooner after food will the pain arise. Thus in those cases where the seventh dorsal segment is mainly affected and the maximum pain and tenderness is situated at and around the ensiform cartilage, the pain will come on within half an hour of taking food. On the other hand, when the ninth or tenth dorsal are mainly affected, the onset of pain may be deferred for an hour and a half. Thus it would seem that the cardiac end of the stomach stands in relation mainly with the sixth and seventh dorsal, the pyloric end mainly with the ninth and tenth dorsal areas.

6. **Intestine.**—(1) *From the Duodenum to the Sigmoid Flexure.*—The pain of intestinal affections

is frequently diffuse, ill-defined, and unaccompanied by tenderness of the skin. Thus lead-colic and many of the colicky pains that accompany an error of diet or summer-diarrhoea do not belong to the type of visceral reflected pains. Where melæna and pain in the abdomen are present, apparently due to ulcer of the duodenum, the pain may be of the reflected type and be accompanied by superficial tenderness over the tenth dorsal area. In the gastro-enteric attacks which sometimes follow chronic constipation or fecal impaction, visceral reflected pain and superficial tenderness may be present from the seventh to the eleventh or twelfth dorsal. In the same way a strangulated hernia may produce pain in the abdomen in the neighbourhood of the umbilicus with superficial tenderness within the tenth dorsal area. Acute diseases of the colon may cause visceral reflected pain and superficial tenderness over the tenth, eleventh, and twelfth dorsal areas. But chronic colitis is usually accompanied by local pain with deep tenderness over the affected part. Thus the intestine from the duodenum to the sigmoid flexure would appear to stand in relation to the tenth, eleventh, and twelfth dorsal segments.

(2) *The Rectum.*—Irritation of the rectum produces not only a local pain of an intense and sickening character, but also, occasionally, reflected visceral pain. This pain is situated over the buttocks and the upper part of the back of the thigh, and when severe is accompanied by superficial tenderness over the third and fourth sacral areas.

7. **Liver and Gall-Bladder.**—Any condition associated with inflammation of the capsule of the liver produces local pain, accompanied by deep tenderness on pressure. Thus, perihepatitis from whatever cause, and secondary malignant growths, are liable to be accompanied by deep tenderness.

Slow enlargements of the liver, and all forms of fibrosis, are unaccompanied by reflected pain. But when, in consequence of cardiac failure, the liver suddenly enlarges, for the first time the patient is liable to complain of pain in the right hypochondrium, right iliac fossa and right loin. This reflected pain may be accompanied by superficial tenderness over the ninth and tenth dorsal areas of the right side. Abscess of the liver, especially when multiple, may cause reflected pain over the eighth, ninth or tenth dorsal, and is also liable to be associated with pain over the right shoulder-joint, probably representing the fourth cervical area. In an attack of biliary colic the pain is of the visceral reflected type, and a paroxysm may be followed by widespread tenderness from the seventh to the tenth dorsal, not only on the right side, but also on the left half of the abdomen. Between the attacks, pain and tenderness are usually situated within the eighth and ninth dorsal of the right side.

Thus the liver seems to be associated with the seventh to the tenth dorsal on the right side, but more particularly with the eighth, ninth, and tenth. The gall-bladder seems to be principally associated with the eighth and ninth dorsal.

8. **Kidney and Ureter.**—Affections of the finer structures of the kidney, such as the various forms of nephritis, cause no reflected pain or tenderness. But all conditions that lead to interference with free outflow from the pelvis of the kidney are liable to be associated with this type of pain. During an attack of renal colic the pain may occupy the greater part of the abdomen and back, but is

usually greatest on the side of the affected kidney, and over the tenth, eleventh, and twelfth dorsal areas. After an attack the tenth, eleventh, and twelfth dorsal may be tender. If a stone passes, the pain may extend to the knee, and all the areas from the tenth dorsal to the second lumbar be tender. If a stone is fixed in the kidney it will cause no pain until the patient is exposed to some unusual jolting. Pain may then appear within the tenth dorsal area. Floating kidney and paroxysmal hydronephrosis may occasionally cause pain and tenderness over the tenth, eleventh and twelfth dorsal areas. Thus the kidney is particularly associated with the tenth dorsal, and also with the eleventh, and probably the twelfth dorsal. The ureter seems occasionally to cause pain as low as the second lumbar.

9. Bladder.—Irritation of the bladder, especially about the neck, is liable to cause pain over the buttocks and back of the thighs. An irritant applied to the mucous membrane, the passage of large clots of blood, and the presence of a calculus will all cause pain over the third and fourth sacral areas. Retention of urine and over-distension of the bladder can lead on rare occasions to radiating pains of the reflected type. These pains lie over the tenth and eleventh dorsal, but are rarely if ever associated with superficial tenderness.

10. Prostate.—Unless the disease is acute no pain is produced; but with acute prostatitis pain may be present down the back of the legs. Tenderness may be present over the second, third and fourth sacral, and occasionally even over the sole of the foot. Such cases are usually diagnosed as sciatica. Sometimes prostatitis seems to be accompanied by pain and tenderness over the tenth dorsal area. It is difficult to say if this is a primary or secondary manifestation, but if the former it is of interest in that it would represent the double origin of the prostate.

11. Testis.—The testicle is so rarely affected alone that it is difficult to be certain of the areas with which it is connected. Where, however, the organ has been crushed, and in a few cases of true orchitis, the patient may refer his pain to the abdomen and loin (dorsal 10).

12. Epididymis.—Acute epididymitis may be associated with reflected as well as local pain. In such cases superficial tenderness may be present over the maximum spots of the eleventh and twelfth dorsal. The tenderness rarely extends over the full areas.

13. Ovary.—‘Ovaritis’ is liable to be associated with widespread pain and tenderness (*vide* § 6). But occasionally, after examination, when the ovary is prolapsed and tender, superficial tenderness may be present over the tenth dorsal area. Moreover, it is not uncommon to find the tenth dorsal area alone the seat of pain and superficial tenderness, when there is reason to believe the ovary is affected. Thus the ovary, like the testis, appears to be mainly associated with the tenth dorsal.

14. The Uterus and its Appendages.—All abnormal states of the female genitalia are liable to lead to widespread reflected pain accompanied by superficial tenderness over the areas from the tenth dorsal to the second lumbar, and the first to the fourth sacral (*vide* § 6). But in cases where the pain is less generalised it is possible to lay down a few general rules. Thus certain cases of disease

of the Fallopian tubes produce pain and tenderness, situated mainly over the eleventh and twelfth dorsal, and first lumbar. Contractions of the uterus are liable to produce pains over areas from the eleventh dorsal to the second lumbar, while forcible dilatation of the os uteri is associated with pains over the sacral areas. The after-pains of labour may extend over both the lower dorso-lumbar and sacral groups, but where the os uteri has previously been extensively lacerated by a previous labour, the sacral pains may be absent. These pains, with their accompanying superficial tenderness of the abdomen, are frequently mistaken for those of peritonitis, from which they differ in that they are present in the loin, and over the sacrum and upper part of the thighs.

15. Eye.—In persons suffering from some error of refraction the headache is said to be ‘over the eyes,’ and when asked to point with two hands to the position of the headache, the patient places the tips of the fingers over the centre of each eyebrow. If we now pick up the skin gently between the finger and thumb, the patient winces as soon as we reach a spot about three-quarters of an inch (2 cm.) from the middle line on either side. If the blunt head of a pin is used he complains that he is tender, or that he is being pricked, as soon as an area over the centre of the eyebrow is reached. By either of these methods the headache produced by *hypermetropia* is found to be associated with superficial tenderness over more or less of the area the writer has called the mid-orbital (*vide* Plate). As a rule, in a pure case of hypermetropia the maximum only of this area is present, but occasionally, especially if the patient is seen immediately after reading or sewing, the whole area, including the patch on the front of the hairy scalp, may be tender. If due to hypermetropia the headache will be found to be worse over that eye in which the error of refraction is greatest. This headache has certain peculiar characteristics. It comes on in the morning as soon as the eyes are opened, is then most intense, and gradually wears off during the day if the patient does not engage in any near work. It is intensified or produced by reading and sewing. It disappears if the eye is put under atropine. Sometimes it is not easy to say whether a headache accompanied by mid-orbital tenderness is due to an error of refraction, which is undoubtedly present, or to some other cause. In such a case, before correcting a slight error of refraction, it is well to make sure of the following points: (1) The headache is definitely made worse by reading and sewing. (2) It comes on in the morning as soon as the eyes are opened. (3) It disappears under atropine applied for a sufficient length of time to paralyse accommodation. (4) There is absolutely no pain or tenderness elsewhere in the body, and, especially, no tenderness over those areas of the body which are definitely associated with the mid-orbital (*vide* 22). If, however, pain and tenderness are present elsewhere in the body, or if the patient is anæmic, it may be quite impossible to say whether the hypermetropia present is really the cause of the headache.

On the other hand, simple *myopia*, however high the error may be, causes no such headache and tenderness. The myope sometimes complains of a ‘tired, aching feeling’ over the forehead. This cannot be definitely localised, and is not accompanied by superficial tenderness of the forehead.

It is never present except after a long and tiring round of looking at objects at a distance (e.g. in a picture-gallery), and is always absent after a night's rest. There is, however, one condition in which simple myopia may produce a mid-orbital headache associated with tenderness—myopia associated with spasm of accommodation. This headache, like that produced by hypermetropia, entirely disappears under atropine. In progressive myopia the conditions are somewhat different. For in this disease destruction is progressing at the back of the eye. Patients suffering from this disease sometimes complain of 'pain on the top of the head,' and some tenderness of the hairy scalp is occasionally found in such cases. Such patients far more commonly complain of a pain 'at the back of the eye,' especially aggravated by movement of the eye. This pain is not associated with superficial tenderness, and appears in many cases to be a local, as opposed to a reflected, pain.

Myopic astigmatism, when uncomplicated, does not produce referred pain or tenderness. On the other hand, *hypermetropic astigmatism* is an even more fruitful source of headache and tenderness over the mid-orbital region than simple hypermetropia. Thus, whereas a simple error of $+1$ or $+2D$. can sometimes be neglected, an astigmatic error of $+1D$. will frequently produce the most definite pain and tenderness.

Anything which tends to produce a permanent abnormal tone of the ciliary muscle will produce referred pain. Thus unsuitable glasses may cause more headache and tenderness than the original error of refraction. This view is supported by the absence of referred pain and tenderness when presbyopia is fully established. A patient may have been hypermetropic, and have suffered from pain over the eyes all his life, without appreciating that he has an error of refraction. When the error manifests itself with the onset of presbyopia, his vision fails for near objects, but his headache improves. Thus headache may be present at forty but not at fifty.

Affections of the *conjunctiva* or of the outer layer of the cornea produce no referred pain. Injury to the surface-epithelium of the cornea produces an intense smarting pain, which is well defined and local. It is exactly equivalent to the well-known local pain produced by injuries to the conjunctiva. If, however, the deeper parts of the *cornea* be affected, as in deep and ragged ulcers of the cornea; or if the tension in the anterior chamber be increased, marked reflected pain may appear. The patient then complains of pain over the forehead; and more or less of the fronto-nasal area may be tender. As a rule, however, the maximum only of this area is present; for if the affection of the cornea is sufficiently grave to produce superficial tenderness over the whole fronto-nasal area, some part of the mid-orbital area will also be affected.

Should the substance of the *iris* be inflamed the pain is usually said to be 'on the temple' or 'in the cheek.' Corresponding to this pain superficial tenderness may be present over the fronto-temporal, maxillary, and temporal areas.

Should the tension in the vitreous chamber rise, as in *glaucoma*, the chief pain shifts further back, and is mainly referred to the temple. Thus in true glaucoma the pain may lie over the fronto-temporal, temporal, and maxillary areas, but the maximum pain is reflected into the temporal area. All

these areas may be more or less tender. The teeth of the upper jaw ache on the side affected. If the pain and tenderness spread forwards to affect the frontal area the teeth of the upper jaw ache up to the middle line; and if the pain and tenderness spread back behind the temporal area, as it not uncommonly does in an acute attack of glaucoma, the teeth of the lower jaw may also ache. Occasionally the teeth affected are tender to percussion, as though periodontitis were present. That this tenderness is reflex, and not due to any actual change in the sockets of the teeth, is shown by the daily variation, during the progress of a particular case, in the position and number of the tender teeth—a variation which seems to be related to the number and position of the areas which are tender on the surface of the head.

It is exceedingly difficult to determine what special areas, if any, are associated with disturbances of the retina and optic nerve. Detachment of the retina, if it occurs suddenly, is sometimes associated with tenderness over the vertical area, and pain 'over the top of the head.'

16. Teeth.—The first stage of caries of a tooth consists of removal of the enamel and excavation of the underlying dentine. During this process pain may or may not be present; but provided the dentine is affected, pain can mostly be elicited by stimulation. Now as long as the pulp cavity is not exposed, the pain remains local. The patient will complain of an aching tooth and will point to the one affected. If, however, the pulp-cavity is exposed, the pain alters in character and distribution. It starts in the affected tooth and shoots into the face, forehead, neck, or ear. Each stab of pain lasts a few moments, only to be followed more or less rapidly by a second twinge. This is the well-known 'neuralgia' caused by affections of the teeth.

On testing the face or neck during this 'neuralgia' some one or more of the areas described will be tender. On the face this tenderness is superficial, but, as pointed out above, parts of the jaw, mouth or tongue may be tender at a distance from the affected tooth, owing to their intimate connection with the distribution of these areas. Thus teeth at a distance from that affected may ache and be tender to the touch.

After caries has reached the pulp-cavity many different changes can occur. The pulp and nerve may become inflamed and reddened, and die rapidly and steadily. But sometimes this death takes place much more slowly. One part of the whole of the pulp in the chamber may die, and with it the nerve in one or more roots, and yet the nerve in one root may remain alive and potent for harm for a long while: or the inflammation and death of the pulp may go on slowly under a cap of sodden dentine. If the pulp becomes fibrous or calcareous, it no longer causes referred pain. But such cases are frequently deceptive; for although the nerve in one root may pull out like a thread of cotton, it is not uncommon to find that another root contains a small vestige of living pulp. Irregular calcification with the formation of pulp-stones frequently leaves some living pulp, and is a frequent cause of referred pain. The condition known as 'polypus of the pulp' is frequently associated with a living nerve. The writer has an impression that where the polypoid formation is large the nerve is likely to be dead; where, on the other hand, the fungating portion is small, the nerve will probably be alive. The point

of the probe will settle this at once. For should the nerve be alive the least touch with the point on the protruding granulation-tissue will cause exquisite pain. These cases show well marked superficial tenderness of the face.

When the pulp and nerve are dead the tooth may still be a source of pain, owing to inflammation of the periodontal membrane or abscess about the tooth. But this pain, in the vast majority of instances, is local and not referred. Sometimes pressure on a tooth in this condition is said to cause 'darting' pain which travels beyond the limits of the tooth. Thus, on pressing a lower bicuspid, which seemed to the patient to be longer than its neighbours, the pain was said to 'run along the jaw.' In the same way pressure on an upper canine produced pain darting up in the line of the socket. Such pain is rarely accompanied by superficial tenderness, and seems to follow a peripheral rather than a central distribution.

The exact innervation of each tooth seems to vary somewhat, but the following table gives the approximate relationship in which the various teeth stand to the areas on fig. 5:—

UPPER JAW.		LOWER JAW.	
Incisors .	Fronto-nasal.	Incisors .	Mental.
Canine .	? Nasolabial.	Canine .	"
1st Bicuspid	Nasolabial.	1st Bicuspid	"
2nd Bicuspid	Temporal or	2nd Bicuspid	?
	Maxillary.	1st Molar	Hyoid.
1st Molar .	Maxillary.	2nd Molar .	"
2nd Molar .	Mandibular.		
3rd Molar .	"	3rd Molar .	{ Hyoid or Superior Laryngeal.

17. Ear.—The pain produced by lesions of the auditory meatus, from its opening to the membrana tympani, is local, although frequently severe. If, however, the membrana tympani or middle ear be affected the pain will be referred to some point at a distance from the source of irritation and may be accompanied by superficial tenderness. When the membrana tympani is affected the middle ear is generally diseased. But in some cases where an accumulation of cerumen in the meatus is decomposing, the membrane is found to be reddened and inflamed. In many cases this redness will subside after removal of the plug, and there is no reason to suppose that the middle ear is affected. Some cases of this kind suffer from pain 'in the ear and under the jaw,' and occasionally superficial tenderness is present over the hyoid area. During suppuration in the middle ear before perforation of the membrana tympani occurs, the pain is intense. In hospital practice patients mostly wait until the membrana tympani is perforated, and the pain has somewhat abated, before finding their way to the ear-department. Thus it is extremely rare to see an adult in this condition. The history given by such patients points to the presence of pain 'at the top of the head' and 'in and behind the ear.' The pain on the top of the head abates when the discharge appears, but the pain 'behind the ear' remains. Occasionally, however, the orifice, once established, becomes blocked. If this should occur the patient begins to suffer from pain on the top of the head, and in such cases the writer has seen superficial tenderness over the vertical and parietal areas of the scalp.

18. Tongue.—It is a well-known fact in surgery that malignant disease of the tongue may cause pain in the ear or over the back of the head, in addition to the local pain in the tongue itself.

If the disease involves the *anterior part* of the tongue, pain is complained of over a spot close to the point where the mental nerve rises through the deeper structures of the jaw to supply the skin. This is the maximum spot of the mental area. This pain in the front of the lower jaw is sometimes associated with superficial tenderness over the mental area. If the *lateral part* only of the tongue is implicated the patient complains of pain 'in the ear,' and behind the ramus of the lower jaw on the side affected. In such cases the hyoid area is not uncommonly found to be tender. If the lesion is situated on the *dorsum* of the tongue behind a line about 3 to 4 cm. from the tip, the pain is 'in the throat' close to the cornu of the hyoid bone. It may also be felt in the back of the head in addition. Occasionally tenderness is present, more commonly in the territory of the superior laryngeal, more rarely in that of the occipital area.

19. Tonsil.—When the tonsils are affected pain appears in the ear, and behind the angle of the jaw (Hyoid area). If the disease is acute and associated with a rise of temperature, wide-spread pain and tenderness may result (*vide* § 6).

20. Nose.—But few nasal affections are painful; and even in those that cause pain, the pain is but rarely acute.

In inflammatory conditions of the olfactory portion of the nose above the middle turbinate bones, pain associated with slight superficial tenderness may appear over the fronto-nasal or mid-orbital areas of the forehead. Inflammation of that part of the nose associated with the lacrymal duct, so common in eye-practice, occasionally leads to mid-orbital headache and tenderness.

21. Larynx.—Affections of the larynx, though producing much discomfort and local pain, do not in the majority of cases produce referred pain and tenderness. The inflammatory conditions, apart from syphilis and tuberculosis, cause great local soreness, but as a rule the soreness is definitely in the larynx.

But where the larynx is in process of destruction by tuberculosis, the pain is not only local, but referred, and superficial tenderness may be present over one or both of the areas called by the writer the superior and inferior laryngeal. When the upper of these areas is tender, the pain is situated a little anteriorly to the cornu of the hyoid bone; when the lower area is affected the pain lies just in front of the anterior border of the sterno-mastoid muscle at about the horizontal level of the cricoid cartilage.

Pain in the Head secondary to disease of organs in the Thorax and Abdomen.

Any organ of the chest and abdomen may, under favourable conditions, cause reflected pain in the head accompanied by tenderness over the areas of the scalp coloured blue (Plate XIV.). The degree to which these pains in the head attract the patient's attention varies greatly. Sometimes he is unaware that the scalp is tender, or he may be aware of the soreness but complain of no pain unless the part be touched. The more acute the pain and tenderness on the body, the more intense will be the pain and soreness of the scalp.

An area on the scalp does not stand in direct primary relation with any organ in the chest or abdomen, but is associated with the segmental areas of the trunk as low as the tenth dorsal. The lower the segment on the trunk that is affected, the more

posterior will be the tender area on the scalp; the higher on the trunk the more certainly will the pain and tenderness be found over the forehead. Thus the third and fourth cervical and the second, third, and fourth dorsal seem to be associated with pain and tenderness over the forehead (fronto-nasal and mid-orbital); the fifth and sixth dorsal with the fronto-temporal; the seventh dorsal with the temporal; the eighth dorsal with the vertical; the ninth dorsal with the parietal; and the tenth dorsal with the occipital areas.

Below the tenth dorsal the segmental areas stand in no relation to those of the scalp. Thus, speaking broadly, it is quite immaterial for the production of temporal headache with tenderness over the temporal area of the scalp, whether the disease be some gastric disturbance, phthisis destroying the base of the lung, or a certain form of mitral disease. The only necessary condition is that one of these diseases should produce pain and tenderness within the seventh dorsal area, when the temporal area will appear tender irrespective of the nature of the diseased organ.

§ 4. Pain produced by Inflammation of Serous Cavities (Pericarditis, Pleurisy, and Peritonitis).

Inflammation of the pericardium, pleura, or peritoneum causes no reflected pain or superficial tenderness of the visceral type. As has been pointed out above (§ 3, 1a and 3a) for the pericardium and pleura, so also with inflammation of the peritoneum local pain makes its appearance accompanied by tenderness on deep pressure or percussion over the affected part. No tenderness is present in the back or at any part where manipulation does not directly affect the inflamed serous cavity. (For the explanation of pain in the abdomen with pleurisy, *vide* 3a, p. 1141.) Thus such tenderness on deep pressure becomes of direct localising value. In the pleura and pericardium the area of deep tenderness corresponds roughly with the area over which friction can be heard. In the peritoneum it corresponds fairly accurately with the area of inflammation found *post mortem* or at an operation for relief of the disease. The absence of superficial tenderness of the visceral type and the presence of well-marked local tenderness are important diagnostic differences between the pain produced by inflammation of the lining of a serous cavity and that caused by disturbance within an organ it contains.

§ 5. Sympathy and Association.

When an organ becomes diseased in such a way that visceral reflected pain is produced, any other organ that receives its sensory supply from the same segment will become tender. Hence during the pain produced by renal calculus the testicle of the same side becomes tender, for both kidney and testicle are supplied mainly from the tenth dorsal segment.

Disease of the lower lobe of the lung tends to produce pain over the seventh, eighth, and ninth dorsal areas. But these are the areas that stand in peculiar relation to the stomach; hence in such cases the stomach tends to become hyperæsthetic, and the patient complains of pain after food, with nausea and, occasionally, vomiting. This is the origin of one form of reflex dyspepsia, occurring in pulmonary tuberculosis, that disappears with the subsidence of the signs of lung-disease. A similar reflex dyspepsia may arise in mitral disease if it is

accompanied by pain and tenderness over the seventh, eighth, and ninth dorsal areas (*vide* § 3, 1b).

Certain organs that are not supplied by the same segment seem to be naturally related to one another. This is the case with the uterus and the breast. Thus within twelve hours after labour it is not uncommon to find a woman suffering from a pain under the heart associated with cutaneous tenderness which comes and goes with the 'after pains.'

§ 6. Conditions which lead to widespread Superficial Tenderness.—So far it has been assumed that a disturbance of each particular organ leads to pain and tenderness over certain areas, and over those areas only. But under the following conditions the pain may spread widely.

If the pain suddenly increases in intensity it is liable to become bilateral. Thus in gall-stone colic more or less tenderness appears over the seventh, eighth, and ninth dorsal areas on the left side, as well as on the right. In the same way the pain and tenderness produced by a calculus in one kidney may spread to the other side at the same spinal level. But in all such cases the pain and tenderness are most marked, appear first and disappear later on the side of the diseased organ.

If the pain assumes a paroxysmal character it may spread widely in all directions, and be followed by extensive superficial tenderness. In renal and gall-stone colic, and in paroxysmal cardiac attacks, the pain of the attack, and superficial tenderness that follows it, may extend beyond the usual renal, hepatic, or cardiac limits. After the attack both pain and tenderness settle down to the usual areas of the particular organ affected.

Then again any condition which decreases the resistance of the nervous system, or relaxes the control of the higher centres, will cause widespread visceral reflected pain.

If a patient suffering from anæmia has a carious tooth, that causes pain and tenderness of the reflected type, the tenderness will be found to spread widely, and may affect the whole or one half of the face and neck, or even spread to the arm of the same side. In such a patient, hypermetropia may cause universal tenderness of the scalp, and a trifling gastric or gastro-intestinal disturbance, pain and tenderness over the greater part of the trunk. Anæmia and chlorosis as such are not necessarily accompanied by headache or gastralgia; but directly a small focus of visceral reflected pain arises in those who are anæmic or chlorotic, the pain and tenderness spread out of all proportion to their primal cause. Moreover, if the cause of the primary focus is removed (e.g. by extracting the diseased tooth) the pain does not cease, at any rate, immediately. For the resistance of the central nervous system being once broken down is with difficulty recovered.

The cachexia caused by wasting diseases may also lead to widespread tenderness, and is probably the cause of the widespread pain and tenderness of the reflected type that accompanies some cases of pulmonary tuberculosis.

A sudden rise of temperature will produce the same effect. Follicular tonsillitis, or any parenchymatous affection of the tonsils, is associated with cutaneous tenderness, extending round the neck from the lower part of the mastoid behind, almost to the middle line of the neck, just above the *pomum Adami*. If one tonsil only is affected one

side of the neck alone is tender over this area. But let the onset be sudden and associated with a rise of temperature to 101° or 102° F. We shall now find the same zone of tenderness, but in addition there will be pain and tenderness over the forehead, in the occipital region, over the maximum of the tenth dorsal area in the back, and at the epigastrium (representing the seventh dorsal), and probably also at several other points in the body and limbs, all of which represent the maxima of their various segmental areas. Thus a rise of temperature has added, to a simple area of tenderness representing the organ primarily affected, a multiplicity of painful and tender spots, representing areas which could in no way be affected by the local manifestation of the disease. In some cases a sudden rise of temperature will produce this widespread tenderness without our being able to recognise any area of primary disturbance. This condition is produced most easily by true epidemic influenza. In this disease the patient notoriously complains of 'pains all over,' and careful examination shows that these pains are associated with tenderness in the skin. But influenza is not the only specific disease which produces these tender areas; they are sometimes present in typhoid fever. Such a condition is, however, uncommon, for the temperature, as a rule, does not run up rapidly enough to produce this widespread tenderness. The more usual condition of a patient suffering from typhoid fever in the first week is a very variable amount of tenderness over some part of the areas occupied by the seventh, eighth, ninth, and tenth dorsal.

In acute rheumatism in the very early stages before the joints begin to swell definitely, similar cutaneous tenderness may appear, though seldom, and always to a less extent than in the diseases before mentioned.

The diminution in resistance that is associated with menstruation is also a potent cause of spreading; for during menstruation trivial causes may lead to widespread pain and tenderness. If the patient has some affection of the cervical canal, such as chronic catarrh or polypi, she may be entirely free from pain until the flow comes on, when she will develop pain and tenderness over one or more of the following areas: tenth, eleventh, twelfth dorsal; first and second lumbar; fourth, third, and second sacral. Most of these areas may be affected, but even at the height of the disturbance the tenderness does not transgress the limits of the areas in connection with the pelvic viscera. Moreover, the areas corresponding to the part which is chronically diseased are always most affected. In other cases of dysmenorrhœa the patient may have suffered from pain and tenderness over a restricted area in the intermenstrual period. As soon as menstruation begins, pain and tenderness spread widely, until at last the whole trunk below the umbilicus, and the whole leg, including even the areas of the gap, may be tender.

Again, as was pointed out above (p. 1143), a normal labour is frequently followed by some pain and cutaneous tenderness. But the pain and tenderness very rarely overstep the areas associated with the pelvic viscera. But after abortion 'spreading' is very common, and although the course of the puerperium may be normal, the patient will frequently complain of pain and tenderness elsewhere than over areas representing the pelvic viscera. It would appear as if the resistance of the nervous system were increased *pari passu* with the progress

of normal pregnancy to bear the strain of normal labour; but that when abortion takes place it is unprepared for the strain put upon it, and generalisation takes place readily.

This breakdown of resistance and spread of pain and tenderness may also be produced by mental causes, or may be due to some inherent defect in the nervous system of the individual. Thus the so-called 'neurotic' individual is noticeable for the ease with which a slight visceral cause produces widespread pain and tenderness, and patients suffering from neurasthenia are particularly liable to widespread visceral pain.

HENRY HEAD.

PAINTER'S COLIC.—SYNON.: *Colica Pictorum*; Lead-Colic; Fr. *Colique des Peintres*; Ger. *Malerkolik*.—A form of intestinal colic, due to plumbism. See COLIC; LEAD, Poisoning by; and OCCUPATION-DISEASES.

PALATE, Paralysis of.—The chief causes of paralysis of the palate are diphtheria (see PARALYSIS, Diphtheritic); degeneration of the nuclei of the medulla oblongata (see LABIO-GLOSSO-LARYNGEAL PARALYSIS); growths in the basis cranii; and compression or inflammation of the nerves of the medulla. The first two causes usually lead to bilateral paralysis. Unilateral paralysis is commonly due to one of the last two causes. It is not caused by disease; the chief nerve-supply to the palate is from the spinal accessory, as clinical observation and experiments alike have proved.

SYMPTOMS.—In *bilateral* paralysis the palate hangs flaccid, and irritation of the mucous membrane excites no reflex movements. It is not raised in breathing or phonation: a convenient test is to make the patient utter the sound 'ah' in a high tone; the central palate should be raised by the levators. Deglutition is interfered with, the soft palate being no longer raised so as to shut off the posterior nares; and liquids are forced up into the nose by the contraction of the pharyngeal muscles. Speech is also affected: the resonance of the nasal chambers gives to it the 'twang' which only the *n* and *ng* sounds should possess. The explosive consonants cannot be well pronounced, because the open passage through the nose prevents the air being sufficiently compressed to give the sudden sound when the passage between the lips is open. Hence *p* and *b* become *f* and *v* respectively.

Unilateral paralysis of the palate causes little interference with deglutition. The chief muscles which raise the palate meet, it will be remembered, in the middle line of the soft palate, and for this reason one muscle is able to effect sufficient elevation of the whole palate to prevent the regurgitation of liquids. The voice may have a slight nasal twang, but the articulation of the labial explosives is not interfered with. When at rest, the paralysed half may or may not be a little lower than the other. The uvula is said to be oblique, inclined towards the opposite side, but this is doubtful; it is often straight in the middle line. A change in form when the azygos contracts may be expected, but is not always to be observed. The chief indication of the paralysis is the unequal elevation, which is recognised during the utterance of the sound 'ah.' It is the middle of the soft palate, just above the uvula, on which the elevators act, and when the muscular contraction is confined to one side, the base of the uvula is drawn a little towards the non-

paralysed side, and a dimple forms above the base of the uvula on that side only. This is the great sign of one-sided palsy. By faradisation a difference in the contractility of the muscles may be recognised, but the difficulties of the test preclude its general utility. Unilateral paralysis of the palate is often associated with that of the vocal cord on the same side, and often with paralysis of the same side of the tongue. This combination is met with especially when there is disease of the nerves at the side of the medulla. The paralysis of the tongue is, of course, due to disease of the roots of the hypoglossal; that of the vocal cord and palate to damage to the higher roots of the spinal accessory nerve.

DIAGNOSIS.—The recognition of bilateral paralysis of the palate depends on its immobility on voluntary effort and reflex stimulation; that of unilateral paralysis on the inequality of movement in the utterance of certain sounds. Difficulty in diagnosis is due to the frequent natural inequality of the arches, and obliquity of the uvula. The latter is so common under normal conditions, that no weight can be attached to it as an indication of paralysis. The opinion that the palate is sometimes paralysed in facial paralysis rests apparently upon the uvula being found to be oblique, and observers have been strangely puzzled by the frequency with which the uvula deviates to, as well as from, the paralysed side, and have formed various ingenious theories to account for the relation; the deviation is, in reality, a 'natural abnormality,' and has no connection with the facial paralysis.

PROGNOSIS AND TREATMENT.—The prognosis and treatment of paralysis of the palate are those of its causes. Locally the muscles may be galvanised by a long electrode, insulated except at its extremity, and furnished with a contact key, so that the circuit is not completed until the instrument is in position. The difficulty of applying electricity for any length of time, and the central nature of its common cause, make its practical value almost imperceptible. Food that is semi-solid is usually swallowed better than liquids; the measures desirable in general management are described under LABIO-GLOSSO-LARYNGEAL PARALYSIS.

W. R. GOWERS.

PALATE AND TONSILS, Diseases of.—*See* TONSILS; and UVULA.

PALERMO, in Sicily.—Moist, warm, equable. No sudden atmospheric changes in winter; the mean temperature being 55° F. *See* CLIMATE, Treatment of Disease by.

PALLIATIVE (*pallium*, a cover).—A term used in connection with the treatment of disease, when it is directed merely to the relief or mitigation of symptoms.

PALLOR (Lat.).—**SYNON.**: Fr. *Pâleur*; Ger. *Blässe*.

This term signifies whiteness or absence of colour. It is due to lack of blood in the affected part, whether due to contraction of the blood-vessels, diminution in the quantity of blood generally, reduction in the number of red-corpuscles, or relative deficiency of hæmoglobin in the individual corpuscles. *See* ANÆMIA; and CHLOROSIS.

PALPATION (*palpo*, I handle gently).—A method of physical examination, in which the hands are employed to appreciate certain conditions perceptible by the sense of touch. *See* PHYSICAL EXAMINATION.

PALPITATION (*palpito*, I beat or throb).—*See* HEART, Functional Disorders of.

PALSY.—A popular synonym for motor paralysis.

PALSY, SHAKING.—A synonym for paralysis agitans. *See* PARALYSIS AGITANS.

PALUDAL
PALUSTRAL } (*palus*, a marsh).—Of or belonging to a marsh. A term generally used in connection with malarial or marsh-fevers, on account of their frequent ætiological association with marshes. *See* MALARIAL DISEASE.

PANCREAS, Diseases of.—**SYNON.**: Fr. *Maladies du Pancréas*; Ger. *Erkrankungen des Pankreas*; *Krankheiten der Bauchspeicheldrüse*.

The diseases of the pancreas will be considered in the following order: 1. Anomalies; 2. General Pathology; 3. Hæmorrhage; 4. Pancreatitis; 5. Pancreatic Cysts; 6. Pancreatic Diabetes; 7. Tumours; 8. Tuberculosis; 9. Syphilis; 10. Pancreatic Concretions, *see* p. 334.

1. **Anomalies** of the pancreas are relatively infrequent and affect the gland itself as well as neighbouring organs, in which accessory pancreatic tissue has been found. In connection with the gland itself may be mentioned—separation of groups of lobules of the head-extremity and their displacement behind the mesenteric artery and vein; still larger islands of pancreatic tissue may be found fully separated and provided with an independent canal emptying into the main pancreatic duct; the tail-extremity may also become completely separated from the body except for the persistence of the duct of Wirsung; the descending portion of the duodenum may be surrounded by a ring of pancreatic tissue. Accessory pancreatic tissue has been found in the stomach, both in the greater and lesser curvature, between the serosa and muscularis and within the submucosa; in the duodenum and jejunum, external to the serosa and beneath the serosa; and in the wall of the ileum. Diverticula of the intestine have been ascribed to these displacements. Variations affecting the ducts also occur. The accessory duct (of Santorini) may be absent; the main duct may have two openings, side by side upon the plica longitudinalis of the duodenum, the bile-duct also having a separate orifice; four orifices are occasionally found, the highest being the accessory duct, the second the common bile-duct, the third the duct of Wirsung, and the fourth a supernumerary duct originating in the head of the pancreas. Again, there may be but one duct opening upon the papilla minor; in this case the ductus choledochus empties alone into the diverticulum of Vater; or with a normal number of ducts, the duct of Wirsung empties into the terminal portion of the bile-duct and the papilla major is wanting. Finally, on account of displacement of the pancreas upward, the pancreatic duct may empty into the stomach.

2. **General Pathology and Symptomatology.**—In an organ in which the physiological functions have been so carefully studied as the pancreas,

disturbance of these might be reasonably expected to lead to constant and characteristic symptoms at once recognisable. The contrary is, however, true. The reasons for this discrepancy are found in the fact that so far, at least, as the digestive juices of the gland are concerned, other organs can easily act vicariously. Thus the proteolytic action can be supplied by the gastric juice; the amylolytic by the salivary secretion; the fat-emulsifying by the bile, and both the latter in part by the intestinal juices; while certain micro-organisms present in the lower intestine are even capable of splitting the fat into glycerine and fatty acids. So far as we know at present, no other organ can supply the internal secretion; but experience has shown that a considerable part of the pancreas may be destroyed without leading to consequences such as might be expected to follow failure or deficiency of that hypothetical material. Moreover, the anatomy of the gland permits grave alterations in structure without producing corresponding symptoms. The presence of two excretory ducts, one of which can take over the entire work in default of the other, is a great safeguard, constituting a favourable anatomical condition as compared with that existing in the liver.

Pancreatic disease is not infrequently associated with morbid conditions in the neighbouring organs, the latter often obscuring the symptoms referable to the pancreas alone. Pathological processes, beginning in the intestine and stomach, may extend to the pancreas, this being especially true of malignant tumours and ulcerations; while inflammations of the bile-duct may involve the pancreatic duct and gland, and these structures may suffer in consequence of the impaction of gall-stones in the diverticulum of Vater. On the other hand, diseases beginning in the pancreas may lead to disturbances in other organs, especially the duodenum and liver, as in tumours of the head of the pancreas, which may erode the intestine, and soon compress and obstruct the common bile-duct. Hæmorrhage and inflammation, moreover, rarely remain limited to the pancreas; neighbouring organs are commonly invaded, and the general peritoneum is not infrequently involved. The symptomatology, therefore, may suggest morbid changes, not in the pancreas, in which the disease has started, but in some secondarily affected organ; or, again, the reverse may be true.

3. Hæmorrhage.—Hæmorrhages of the pancreas are not very infrequent. The punctiform varieties occur in the course of passive congestion in chronic diseases affecting primarily the heart, liver, kidneys, and lungs. Somewhat larger ecchymoses are met with in certain diathetic, infectious, and toxic conditions, as in scurvy, purpura, septicæmia, eclampsia, and phosphorus poisoning. More abundant hæmorrhages are associated chiefly with inflammations and necroses of the gland. The blood may appear in the interstitial tissue or within the acini; more commonly in the former location. The corpuscles may be recognisable as such, or different stages of pigmentary transformation may be found. Experiments upon the pancreas in animals have demonstrated that hæmorrhage is readily excited.

ÆTIOLOGY.—Hæmorrhage of the pancreas is recognised as a cause of sudden death; though the importance of the local lesion is still open to doubt. In some cases even copious bleeding into the pancreas may be of only minor importance, the condition being associated with similar hæmorrhages

in other organs, and resulting chiefly from disturbances of the circulation. Injection of relatively large quantities of blood into the pancreas in the dog sets up a sclerosis only of the organ (Flexner and Pearce).

The cause of the hæmorrhage can be established only in a certain proportion of the cases. The most frequent concomitant pathological condition is vascular disease, such as fatty degeneration, arterio-sclerosis, and lesions secondary to alcoholism and syphilis (Seitz). Fitz regards the anatomical relations of the blood-vessels as favouring hæmorrhage, the abrupt division of the larger branches of the arteries into vessels of small calibre, the height of the aortic pressure, and the variations in blood-content, as determined by the work of the diaphragm and abdominal muscles, all contributing to the production of rupture of their walls. Not infrequently hæmorrhage has been observed in cases in which adipose tissue has been present in the pancreas and the glandular cells have suffered fatty degeneration. Such a hæmorrhage may be the direct cause of death. The hæmorrhage has also been ascribed to fat-necrosis (*see below*). Both in human and experimental necrosis, when fresh, a zone of hæmorrhage, usually not exceeding a line in extent, surrounds the dead area; but inasmuch as such fat-necroses are frequently present in the absence of hæmorrhage, and constantly accompany inflammatory lesions of the pancreas, which themselves are often associated with hæmorrhage, their causal relationship to the bleeding is very doubtful. Hæmorrhage may also take place into cysts, in which case, as a rule, the gland is not implicated (*see ABDOMINAL CYSTS*). Furthermore, a few instances of severe and fatal hæmorrhages from malignant tumours of the pancreas have been recorded. Embolism and thrombosis of the pancreatic arteries are occasionally the causes; traumatism is a more frequent and a not unimportant ætiological factor, while inflammations are among the most common.

MORBID ANATOMY.—The pancreas in this condition displays a picture varying according to the degree and the age of the hæmorrhage. The gland is mottled and presents a predominating dark red or violet colour, due to the presence of blood, chiefly within the interlobular connective tissue. The glandular acini are distinguished as greyish, opaque foci scattered over the surface. The whole organ is swollen; the blood often infiltrates the periglandular tissue, or may collect in the lesser omentum. Upon microscopical examination, blood-corpuscles in great number are found within the interstitial tissue, often pressing the fibres apart, and extending into the glandular acini. The acini may preserve their form, or the cells be disturbed and their arrangement altered; the ducts sometimes contain blood. If the hæmorrhage is older, blood-pigment is found in the interstitial tissue, while the latter may also show an increase in its connective tissue.

When the bleeding is extensive, the entire tissue of the gland is destroyed and the blood invades the retro-peritoneal tissue. In other instances, the peritoneal covering is broken and the blood fills the lesser sac.

SYMPTOMS.—Slight bleeding is not associated with any recognisable symptoms; extensive hæmorrhages may arise during the best of health, and lead quickly to a fatal issue. A host of indefinite

symptoms, such as obstinate constipation, chronic dyspepsia, and colicky pains have been noted as preceding the onset of hæmorrhage. Just prior to a fatal attack severe pain of a colicky character in the upper part of the abdomen is complained of, and endures until the end. Almost from the first it is accompanied by nausea and vomiting, the latter, no matter how frequent and severe, giving no relief. The vomit is usually bile-stained; rarely it consists of a yellowish-brown fluid, or becomes bloody (Hooper). The abdomen is distended; only with extreme rarity does palpation disclose a tumour. In the rapidly progressing cases fever is rarely present; in the stage of collapse, subnormal temperatures are met with. The pulse, as a rule, is weak and quickened; loss of consciousness or delirium is but rarely noted. Not many careful studies of the urine are on record. In the 'lightning cases' it has either shown nothing abnormal or anuria has existed. Albumen has been found, and also sugar; the proportion of the latter having reached 6·1 per cent. in one of Cutler's cases.

TREATMENT.—Therapeutic measures are of little avail. It has been suggested to open the abdomen, expose the pancreas, and relieve the tension, since the fatal result is often due to pressure upon the solar plexus rather than to loss of blood. In the acutely progressive cases the procedure would rarely be resorted to, whereas in the more chronic types, especially where cysts and perhaps abscesses have developed, the surgeon may be able to do a great deal.

4. **Pancreatitis.**—Inflammations of the pancreas are not very infrequent occurrences. Excluding cloudy swelling, or so-called acute parenchymatous pancreatitis, which may occur in all severe infections and intoxications, we can distinguish several well-defined types of inflammation of the pancreas. These may profitably be considered under the headings, acute hæmorrhagic, suppurative, necrotic, gangrenous, and chronic indurative pancreatitis. Moreover, while the several varieties mentioned may have an independent existence, mixed forms may also exist.

(1) *Acute Hæmorrhagic Pancreatitis.*—This form of inflammation has not been sharply separated from pancreatic hæmorrhage. Indeed there is still much difference of opinion as to the occurrence of a well-defined pancreatic disease in which the inflammation is followed by extensive hæmorrhages. The free escape of blood into the gland, if it fails to produce immediate death, is followed by changes of an inflammatory nature in the organ. The majority of writers, however, adhere to the belief in a hæmorrhagic inflammation of the pancreas. Diffuse inflammations of the organ, in which hæmorrhage is either absent or inconspicuous, also occur.

Hæmorrhagic inflammations can also be produced experimentally. Allusion has been made to the readiness with which extravasations occur in the pancreas, and hence even mild operative procedures such as ligaturing and slight crushing of the gland may produce fairly extensive hæmorrhages. The introduction of irritating substances (e.g. artificial gastric juice, bile, caustic alkalis, bacterial cultures) into the pancreas through its ducts brings about a similar change even more strikingly. That the hæmorrhage in the last instance is not primary is shown by the histology of the lesions, for when death has taken place within twelve or fourteen

hours after the operation, great congestion of the vessels will be evident, the number of white cells in the blood will be increased, necroses in the gland will already have formed, and emigration of leucocytes taken place.

A large portion of the cases occur in male adults. Among the 41 instances collected by Körte only four were in women: McPhedran has reported one in a child nine months old.

ETIOLOGY.—The ætiology of the disease is obscure. Some, but not all, of the patients who have succumbed to it have been addicted to alcohol. In several cases cholelithiasis has co-existed (A. Mayo Robson, &c.). In these cases occlusion of the pancreatic duct at its orifice from pressure of a stone lodged in the diverticulum of Vater, by causing a damming-back of the secretions may possibly predispose to bacterial invasion.

The bacteriological study of human cases of this form of pancreatitis has given very inconstant and inconclusive results. By injecting the *B. pyocyaneus* and *B. diphtherie* into the pancreatic duct, the writer obtained intensely hæmorrhagic inflammation of the gland in several instances within forty-eight hours. The absence of demonstrable bacterial invasion in many cases indicates other modes of causation. The writer caused rapidly fatal hæmorrhagic pancreatitis in the dog, associated with peritoneal and pericardial fat-necrosis, by injections of artificial gastric juice into the duct of Wirsung; and Opie has produced a similar condition in the dog by injections of sterile bile into the duct.

MORBID ANATOMY.—The pancreas is found enlarged, and the interstitial and even the glandular tissues are infiltrated with blood. Clots of a considerable size may occur in the interlobular tissue and upon the surface of the organ; or instead of blood, a fibrinous exudate may be found upon the capsule. On section, the gland exhibits, besides a hæmorrhagic mottling, irregular areas of coagulative necrosis. The blood (as in pancreatic hæmorrhage) may pass into the contiguous tissues, infiltrate the mesentery, and escape into the peritoneal cavity. Peritonitis may also be present. Fat-necrosis, appearing as opaque, yellowish-white specks and areas, 3 or 4 mm. in diameter, often surrounded by a zone of hæmorrhage, occurs in the fat of the mesentery, omentum, peri- and inter-pancreatic tissue, retroperitoneum, pericardium and other places. See **FAT-NECROSIS**, p. 526.

In the cases which macroscopically show hæmorrhages sufficient to obscure completely or to a large extent the parenchyma, microscopic examination demonstrates that the blood lies between or within the acini. The connective-tissue septa between the lobules are frequently much expanded and thickened, owing to the presence of the hæmorrhage, the blood-corpuscles not only lying in the tissue-spaces and pressing apart the connective-tissue fibrillæ, but also occupying similar spaces in the adipose tissue and appearing between the individual fat-cells. From these points the hæmorrhage extends into the acini, the amount being somewhat variable. In some instances it follows the fine connective-tissue septa between the acini, while in others it penetrates more diffusely into the substance of the gland, so that the red blood-corpuscles come to lie in intimate contact with the glandular epithelium, which may be displaced from the basement-membrane. The effects on the

arenchyma proper vary with the extent of the infiltration. Where the hæmorrhage is for the most part confined to the connective-tissue septa, the glandular tissue stains sharply and the cells appear quite normal, while, on the other hand, the intra-acinous hæmorrhage is commonly associated with vacuolation, increased granulation, and even necrosis of the cells. The disintegration of the gland may be most marked at some distance from the chief hæmorrhage, and the most extensive leucocytic infiltration, instead of being found where the hæmorrhage has been most profuse, may appear where the disturbance of the normal arrangement of the glandular epithelium is especially pronounced. In the early stages the connective tissue is quiescent, no multiplication of cells apparently taking place. The main blood-vessels remain open. The veins are much dilated, but are free from thrombi; they show, especially near the periphery of the vessel, an increase in the number of leucocytes, which is much less marked in the arteries. The majority of the smaller veins and arteries, as a rule, are open and show changes similar to those observed in the larger vessels—hyperæmia and leucocytosis. Occasionally thrombi (chiefly venous) occur in small vessels; the thrombi are largely fibrinous, but contain a few fragmented nuclei of white blood-cells. Very extensive hæmorrhages may occur in the gland and yet remain limited to the interlobular septa. Where this has happened, the gland-tissue presents a normal appearance, despite the manifest congestion of the blood-vessels in the septa. The products of the hæmorrhage may find their way into the branches of the excretory ducts, the epithelial lining of which appears perfectly normal.

Where coagulative necrosis has occurred, the glandular and connective-tissue cells are devoid of nuclei, granular, and in part disintegrated; the only cells of the foci in which the nuclei are preserved are emigrated leucocytes. The hæmorrhage is outside of these necrotic foci, and frequently outside of the mass of leucocytes, which forms as it were a collar immediately around the necrotic zones. In the most acute instances of this condition no changes of a progressive character are demonstrable in the connective-tissue. The condition of the blood-vessels varies. In the necrotic zones they are, of course, occluded; outside they may still be open and widely distended on account of the active hyperæmia. The zones of the necrotic tissue, even where red blood-corpuscles are absent, show much fibrin. The structure of the acini may still be demonstrable, the cells having an increased refraction, taking the acid stains, and being devoid of nuclei. This loss of nuclei is usually through lysis, but there are exceptions in which large numbers of nuclear fragments exist, particularly in the peripheries of the necrotic zones.

SYMPTOMS.—The onset is likely to be sudden and attended by violent colicky pains, referred chiefly to the upper part of the abdomen. Nausea and vomiting follow, and symptoms of collapse more or less severe, according to the severity of the attack. The abdomen becomes swollen and tense; the bowels are obstinately constipated. At first there may be no fever, but the temperature may rise later, the elevation being often preceded by a chill. Symptoms of collapse supervene, and death occurs usually between the second and the fourth day, or even earlier. Intestinal obstruction, or acute perforative peritonitis, is usually suspected; but cases

have been diagnosed *intra vitam* (Fitz, Thayer, Bloodgood), and even operated upon. With reference to the diagnosis, Fitz says: 'Acute pancreatitis is to be suspected when a previously healthy person, or a sufferer from occasional attacks of indigestion, is suddenly seized with a violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours by a circumscribed epigastric swelling, tympanic or resistant, with slight elevation of temperature. Circumscribed tenderness in the course of the pancreas, and tender spots throughout the abdomen, are valuable diagnostic signs.'

(2) *Acute Suppurative Pancreatitis (pancreatic abscess).*—Two forms of suppurative pancreatitis have been met with: the abscesses may be multiple and small in size, when they are usually distributed throughout the gland; or they are larger, and single or, at most, few in number. Diffuse purulent infiltration is very rare. Multiple suppurative foci are usually due to metastasis; the larger abscesses are more frequently the result of the entrance of pyogenic micro-organisms along the pancreatic ducts.

ÆTIOLOGY.—The ætiology in the majority of cases is doubtful. Dieckhoff has pointed out three possibilities: (1) the infection may be hæmatogenous, pyogenic organisms, chiefly cocci, being brought to the pancreas in the circulating blood; (2) the micro-organisms may extend to the pancreas from a neighbouring viscus, as in ulcer of the stomach; (3) the pyogenic bacteria may enter the ducts from the intestine. Traumatism has been noted as an exciting cause. Of the reported cases, in twenty-four there was a single abscess, and in fourteen there were numerous small abscesses.

Primary abscess arises from infection of the ducts. As predisposing causes may be mentioned, pancreatic calculi, biliary calculi compressing the pancreatic duct, and tumours of the duodenum. Retention of the pancreatic secretion, and perhaps catarrhal inflammations of the duct, favour the entrance and multiplication of the pyogenic micro-organisms. Dieckhoff has traced the bacteria through the larger into the finest ducts, and thence into the acini; they also penetrate the walls of the ducts, and enter the peri-canalicular lymph-spaces and interstitial tissue. Chief among the bacterial forms which have been isolated are the pyogenic cocci, and bacilli of the group of *B. coli communis*.

Suppurative pancreatitis has been brought about experimentally. Doubtless the first to produce it was Claude Bernard, who injected quicksilver into the ducts. The injection of material containing bacteria (perityphlitic pus, Körte), and cultures of pyogenic organisms have usually sufficed to cause abscesses. Other irritants, such as turpentine, may also do so, although a chronic form of inflammation is the rule from the employment of this substance. The writer also obtained occasional suppuration from injections into the duct of acids and alkalis.

MORBID ANATOMY.—In consequence of the suppuration, necrosis of larger or smaller portions of the gland may occur. Indeed, the entire gland has been seen to have undergone necrosis and sequestration, the remains of the pancreas being found bathed in pus and occupying the cavity of the lesser omentum. Putrefactive changes may take place in the suppurating area when gangrene in the adjacent glandular tissue is likely to occur. From

the pancreas as a starting-point, the suppuration may extend to the peripancreatic tissues, or the cavity of the lesser omentum may become infected, so leading to an encapsulated abscess by closure of the foramen of Winslow. The suppuration may also extend to the retroperitoneal tissues and the pus pass towards the right kidney or the spleen, or follow the descending colon down to the pelvis. Through the use of artificial injections, Körte has proved the natural extension to be to the left side. The rupture of pancreatic abscesses may set up a generalised peritonitis. A not infrequent termination is by rupture into the stomach or intestine. Moore has reported an instance in which the abscess burst into the peritoneal cavity, and the pancreaticoduodenal artery. Venous thrombosis, involving the portal, splenic, and superior and inferior mesenteric veins, is not infrequent. Thrombosis of the femoral veins has been noted, and Körte has described parietal thrombosis of the splenic artery. Multiple metastatic abscess may follow the vascular thromboses originating from the pancreatic disease.

SYMPTOMS.—The symptoms of suppurative pancreatitis are not always well defined. The disease may begin suddenly, or may follow disturbances of digestion or biliary colic. Severe pain in the epigastrium and extending over the entire abdomen may be present. Cases without pain have also been described. Sensitiveness upon pressure in the epigastric region is almost constant. Nausea, vomiting, or retching are frequently present. The temperature usually shows an irregular elevation, but chills and delirium are less constant. Constipation is the rule, but in some cases diarrhoea has set in during the first twenty-four hours, or at a later period of the disease. Blood and foetid pus have been noted in the dejections after perforation of an abscess into the gut. The epigastrium is usually distended; in a few instances a tumour has definitely been made out in the upper abdomen. Jaundice is common, and fatty diarrhoea and glycosuria have been met with. After extension of the suppuration to the peripancreatic tissues and contiguous parts of the general peritoneum, the symptoms may belong entirely to these complications.

TREATMENT.—The history of the case, pain, and the presence of a tumour in the region of the pancreas, justify an exploratory incision. Körte advises exploratory puncture, especially when the suppuration has extended backwards towards the retroperitoneal tissues. The needle is introduced in the lumbar region; the withdrawal of cloudy fluid, mixed with fatty fragments, he regards as pathognomonic of pancreatic disease. It may happen that even during the operation the site of the abscess is made clear by the nature of the pus, its richness in fat, and the admixture of fragments of necrotic tissue. In rare instances the diagnosis cannot be made until a pancreatic fistula has developed. The condition, unless relieved by spontaneous rupture into the gut or by operation, is probably fatal. The course of the disease may cover a few days or several months.

(3) *Necrotic and Gangrenous Pancreatitis.*—Necrosis of the pancreas, varying in degree, is an accompaniment of the various forms of acute pancreatitis. It rarely occurs in the chronic types. Larger foci of destruction, the result of occlusion in the blood-vessels, may be the cause of inflammations. While the presence of microscopical and

macroscopical areas of necrosis are the rule in hemorrhagic pancreatitis, in the suppurative form sequestra are frequently contained within the abscesses. Their size is subject to great variation; the middle portion, the tail, or the entire gland may have become separated. Fat-necrosis has been urged as a cause of the glandular necrosis. This is believed to result from extension of the hemorrhage, which commonly attends the fat-necrosis, or from growth in size of the fat-necroses themselves. There is little basis for this view, the evidence at hand pointing rather to the production of fat-necrosis as a consequence of the pancreatic disease. That self-digestion of the pancreas may cause such necroses seems probable from the two instances of the condition reported by Chiari. Pathological conditions in contiguous parts may, by extension to the pancreas, set up necrosis.

There is no special morbid anatomy, histology, or symptomatology for this condition, seeing that it is closely related to hemorrhagic and suppurative pancreatitis. Its affinities with gangrenous pancreatitis are also very close.

Gangrenous pancreatitis may succeed the other forms of the disease described, and represents a stage in which, through the entrance into the already injured organ of putrefactive bacteria, complete tissue-death with subsequent decomposition ensues. The extent of the lesion depends greatly upon the primary exciting cause; there may be complete necrosis of the gland or parts or it, and this may follow either hemorrhage, acute inflammation, or purulent infiltration, while in exceptional instances it may occur after injury, or from extension of disease in neighbouring parts. Perforation of gall-stones, and consequent inflammation and putrefaction of the gland, as well as extension of gastric ulcerations to the pancreas, are established causes. In other cases the aetiology is obscure. Fitz thus describes the gangrenous gland: 'The appearance of the pancreas varied according to the duration of the disease. About the fourth day, the pancreas may be doubled in size, dark red and of somewhat diminished consistency. It may be red, on section, or mottled red and grey. The tip may be shreddy, or the entire gland may be transformed into a dark, slaty-coloured, stinking mass. The adjacent parts may be infiltrated with a discoloured purulent fluid, or the coils of the intestine near the pancreas may be united by recent adhesions together and to the under surface of the diaphragm. . . . On the tenth day the pancreas may be dark brown, dry, firm. It may have a hemorrhagic coating, or lie in a sponge-like mesh-work, infiltrated with dirty, green fluid. . . . Towards the end of the second week, the pancreas may be a soft, black, shreddy, sloughy, gangrenous mass, the central part being encapsulated by the peripheral portion. Between the pancreas and the transverse colon may be a large quantity of chocolate-like fluid, and large bluish-black clots. Or the tail and a part of the body may be soft, grey, discoloured, and infiltrated with a thin, ichorous fluid, while the parapancreatic tissue is partly purulent and partly ichorous. . . . A few days later, the pancreas may be a thin, flabby, friable, greyish band, lying in a cavity behind the stomach, and attached by a few shreds to the omental wall. . . . At the end of three weeks, the dark brown pancreas may lie nearly free in the omental cavity. . . . In the fifth week, the pancreas may lie free as if macerated, in a cavity ex-

tending behind the cæcum and descending colon, on both sides of the spine, as far as the pelvis. . . . At the end of seven weeks, the pancreas may be transformed into a cylindrical, shreddy mass of brownish-black, friable tissue. This may lie in the omental cavity, which is filled with offensive, black fluid, and communicates with the stomach and jejunum.' In two cases reported by Chiari, the necrotic pancreas was discharged *per rectum*, with recovery. In one case the sequestrum showed scarcely any indication of pancreatic structure; but instead there were larger and smaller islands of necrotic tissue, richly infiltrated with polymorphonuclear leucocytes, many of which were fragmented. Micro-organisms were present in immense numbers, among which diplococci, streptococci and large thick bacilli could be distinguished.

Gangrenous pancreatitis has rarely been produced in experiments upon the pancreas of animals, the procedures thus far employed in producing pancreatitis having yielded hemorrhagic and necrotic inflammations. When extensive injury has been inflicted upon the gland, death has resulted in a short time—12 hours to 3 days—presumably before putrefactive organisms have had time to enter the injured organ and increase. In two instances of diffuse hemorrhagic pancreatitis following the injection of artificial gastric juice into the duct of Wirsung, Pearce and the writer have noted invasion of the pancreas by gas-producing bacilli, and the production of extensive necrosis with dark discoloration. Sequestration, on account of the short duration of life, had not taken place.

The symptoms of gangrenous pancreatitis are those of suppurative pancreatitis. The diagnosis would be made upon the same general basis; confirmation would only be rendered possible by a surgical operation, the evacuation of the sequestered organ with the dejecta, or *post-mortem* examination. Peritoneal fat-necrosis is a very common attendant, and may be the 'sign-post' upon the opening of the abdominal cavity by the surgeon. See FAT-NECROSIS.

(4) *Chronic Pancreatitis* is characterised by a new growth of connective tissue with corresponding atrophy of the glandular structures. Two main forms are recognised: (1) a chronic inflammation extending from the ducts and met with in association with chronic catarrhal processes in the stomach and duodenum and in the bile-passages; (2) a chronic pancreatitis of hæmatogenous origin, resulting from toxic material in the blood, produced particularly by alcohol and in vascular disease (Dieckhoff).

An indurative pancreatitis which starts from the ducts (sialangitis pancreatica) is the common form. All conditions which favour the entrance of micro-organisms into the duct (cholelithiasis, carcinoma) predispose to it. Occlusion of the ducts by pancreatic calculi also produces chronic pancreatitis. The new tissue grows about the blood-vessels, around the pancreatic ducts and the nerves, and eventually increases the interstitial bands running between the lobules and separating the acini. The secreting structures undergo fatty degeneration and atrophy, finally disappearing entirely. In closure of the pancreatic duct by stone or chronic inflammation, the pressure of the retained secretion causes atrophy and disappearance of the glandular cells, with a subsequent new growth of connective tissue. The interlobular adipose tissue tends also to become

increased. The sclerosis may involve the whole organ, converting it into a fibrous strand, in which few or no remains of glandular tissue may be found; or it may affect a circumscribed portion (especially the head) of the gland. Riedel has emphasised the occurrence of chronic inflammation of the head of the pancreas in association with gall-stones, and has seen the condition relieved after their removal. Körte has described compression of the common bile-duct by a small nodule of the pancreas involved in chronic pancreatitis. Experimental sclerosis can be produced by a ligaturing of the ducts in animals, and by injections of blood and irritants into the duct of Wirsung (Körte and Flexner).

Indurative pancreatitis may result from endarteritis obliterans, and is then to be regarded as a part of a general process affecting the organs of the body. The degree of connective-tissue growth is variable, and may be only small in amount; or the gland may be reduced to a 'mass of fatty tissue.' The coeliac axis shows advanced arterio-sclerosis with calcification. Syphilis is an undoubted cause of indurative pancreatitis. Its ætiological significance is seen at once in congenital syphilis. Rokitsky and Birch-Hirschfeld dwelt on the syphilitic sclerosis of the pancreas, and pointed out that it occurs both with and without the presence of gummata. In the severer grade of the congenital disease the interstitial portion is increased to such an extent that the secreting tissue may be entirely destroyed. Alcoholism is another cause of connective-tissue proliferation. Sclerosis of the pancreas may be associated with hepatic cirrhosis. A similar association with chronic nephritis has been noted. Hansemann has drawn certain analogies between the small granular kidney and the so-called simple atrophy of the pancreas.

The chief interest in sclerosis and atrophy of the pancreas relate to their association with diabetes mellitus, which will be discussed later in the present article.

5. **Pancreatic Cysts.**—*Retention-Cysts* are the most common (p. 360). The obstruction may be in the main ducts or in the smaller branches. It may result from compression or obliteration of the ducts, or both combined. The most frequent cause is chronic indurative pancreatitis, in which the new growth of connective tissue compresses and obliterates the ducts. A similar condition has, in a few instances, been produced experimentally; once by Miroloix, who injected a mixture of soot and vaseline into the duct of Wirsung, after tying the accessory duct, and again by the writer, who observed cysts develop in the pancreas of the dog, after the injection of dilute acids (hydrochloric, sulphuric). When the animals were killed after periods of from three to four weeks, the pancreas was found contracted and indurated. The cysts contained an opaque glutinous fluid. Tumours pressing upon the main duct may give rise to cystic dilatation. Peripancreatic fibrous induration, enlarged lymphatic glands, and gall-stones within the common duct may be the sources of pressure. Pancreatic calculi are among the important causes. The concretions may be in the main ducts as well as in the smaller ones, and the cystic dilatations may be single and large—as large as a child's head—or smaller and multiple (*varanula pancreatica*). It is uncertain whether catarrhal inflammation can cause closure of the pancreatic duct: cysts from this source are unknown; nor have cysts been described

as a result of the compression of the duct by tumours of the pancreas itself. An unusual cause is the entrance of an *Ascaris lumbricoides* into the duct of Wirsung. That necrosis of the pancreas may lead to the production of cysts is shown by a case reported by Tricomi, in which, besides the presence of concretions, there was necrosis of the gland; moreover, in the writer's experiments the remains of pancreatic tissue were found among the contents of the cysts. Traumatism has been given as a cause of cysts. Blows on the abdomen, or constantly repeated pressure, are the most common forms of injury. In this connection, Osler mentions severe massage. The contents of the cysts are usually bloody, though they may be clear or yellowish. Hemorrhage may have taken place into the retention-cysts, either shortly before operation, or at some more remote period. From the character of the wall and its relation to the ducts it is possible to decide whether the cyst has been preformed or not. Cases of true apoplectic cysts, however, have been reported. When they arise independently of retention-cysts, they must be regarded as curiosities.

Proliferation-Cysts.—In proliferative cysts there is no indication of any relation to retention or simple inflammation (p. 361). They are correctly regarded as representing a neoplastic change, being analogous to the proliferation-cysts of the kidney and ovary. They are usually multiple, and vary in size from a pea to a hen's egg. From the fibrous coats of their walls, outgrowths in the form of excrescences are developed; these, as well as the inner surfaces of the walls, are covered with epithelium. The wall itself also contains acini, which often present lumina. In Menetrier's case there were cysts in the liver also. Echinococcus-cysts of the pancreas appear to be very rare. Cysts are seated more often in the tail than in the head of the pancreas. The quantity of fluid contained within the cysts varies greatly. The average is from one to three litres, and the maximum 20 litres. In rare instances the contents are colourless, pale yellow, or yellowish-green. Usually they are brown in colour, sometimes coffee-coloured, or reddish-brown. Not infrequently they are turbid, muciginous, and more rarely gelatinous (colloid) or purulent. The admixture of blood, whether recent or old, causes profound changes. The fluid may be dark red and contain coagula, or present a chocolate-brown colour. The reaction of the fluid is usually alkaline, sometimes neutral, while in one instance it was acid. The specific gravity varies from 1·007 to 1·028. The amount of albumen fluctuates between 0·56 per cent. and 10 per cent. It is composed chiefly of serum-albumen and globulin. Metacasein and peptone have been found in isolated cases. Sugar has only rarely been found; cholesterol is common; urea, leucine, and tyrosine are unusual, while the pancreatic ferments have not always been demonstrable. In some cases one, in others all the ferments have been discovered.

SYMPTOMS.—The development of pancreatic cysts often proceeds so insidiously, that until they reach a large size no subjective symptoms manifest themselves. Disturbances of digestion, preceding the appearance of a tumour, and extending over a long period, as well as painful colicky attacks, nausea and vomiting, together with progressive enlargement of the abdomen, have commonly been noted. Pancreatic salivation is rare. Emaciation is noted by Osler as a very common symptom. Some of the main symptoms may be referable to

chronic pancreatitis, which is the most frequent cause of the condition, or to disturbances in adjacent organs upon which the cyst exerts pressure. The jaundice may be marked and enduring. Adhesions between the cysts and neighbouring organs may arise, and if the latter chance to be hollow viscera, rupture into them may take place. Hæmatemesis has resulted from rupture of a blood-containing cyst into the stomach, and a very watery stool, with disappearance of the abdominal tumour, has been noted when the cyst has broken through into the intestine.

The swelling caused by the cyst may be regional or produce a general enlargement of the abdomen. Usually it is limited to the epigastrium, and inasmuch as the tail and body are more frequently affected, it is most likely to be marked in the left epigastrium, the mesogastrium, and the left hypochondriac region. Under certain circumstances the abdomen may reach an enormous size. The tumour is smooth; fluctuation is commonly present, but may be absent if the cyst is well filled. On percussion, the note is completely or partially tympanic, depending upon the condition of the overlying hollow viscera. The movements of the diaphragm may cause oscillations of the tumour, and lateral movements may be obtained through the abdominal walls.

TREATMENT.—Cysts of the pancreas come into the category of surgical diseases. Puncture, total removal, partial extirpation with drainage, and incision and drainage have all been practised. In seven cases in which puncture was done, five deaths occurred; in 11 cases of total extirpation, two deaths; in 12 cases of partial extirpation, four deaths; in 12 cases in which incision was done in two operations, there was one death (due to diabetes); and in 58 cases with immediate incision, there were seven deaths (Osler).

6. The relation of diabetes mellitus to disease of the Pancreas.—Extirpation of the pancreas in the dog causes, without exception, diabetes of severe degree. The elimination of sugar by the urine reaches 10 per cent. or more; the animals, notwithstanding their ravenous appetites, lose weight and flesh; they exhibit pronounced thirst, and ultimately become very weak and succumb. Complete extirpation of the gland in cats, rabbits, swine, toads, and frogs, is followed by similar results. In birds, removal of the pancreas gives rise to diabetes, but without causing a constant glycosuria. The blood, on the other hand, shows considerable increase in its sugar-content (hyperglycæmia). Dogs, after extirpation has been practised, may begin to secrete sugar from the kidneys as early as six hours after the operation. The amount gradually increases until on the third day it reaches 8–10 per cent. The maximum recorded is 22 per cent. The highest percentages are maintained until toward the end, but finally, and when the emaciation has become very great, the quantity is diminished. As the sugar falls to the minimum, acetic acid, acetone, and oxybutyric acid appear in the urine. The acme of the secretion of sugar is reached from carbohydrate food; but a pure albuminous diet does not reduce the amount below 5·6 per cent., while fasting for a period of seven days does not suffice to cause its entire disappearance (von Mering and Minkowski). Incomplete removal of the pancreas is not necessarily followed by diabetes. If one-fifth of the gland be left either *in situ* or transplanted beneath the skin, the diabetes may be averted.

Should the amount left behind sink to $\frac{1}{4}$ or $\frac{1}{17}$, or should a larger remnant suffer later in its nutrition and undergo atrophic change, a mild form of diabetes arises which, however, tends, through progressive degenerative changes in the gland, to become more severe, and to terminate fatally. The voided sugar is glucose. Saccharose, maltose, lactose, levulose, and galactose are eliminated chiefly as dextrose. Traces of levulose and galactose appear, however, in the urine. Hyperglycemia goes hand in hand with glycosuria. The former is demonstrable four hours after ablation, and reaches its height in 24 hours (Lépine). Towards the end of the disease, when the glycosuria diminishes, the hyperglycemia also falls.

Diabetes, the result of total extirpation of the pancreas, is fatal. No other organ can act vicariously in regulating the carbohydrate metabolism of the body. Hence the function of the pancreas, the disturbance of which produces diabetes, may be regarded, in Minkowski's sense, as specific. This is not, however, equivalent to saying that the pancreas is the only organ, the disturbance of whose functions is capable of producing diabetes. It is probable that other organs also preside over the carbohydrate metabolism, although perhaps in a less degree, and that the pancreas may act vicariously for them, although they themselves have not the power to be substituted for the pancreas (Naunyn). Reale and Minkowski have shown that the removal of all of the salivary glands in the dog is followed by a transient glycosuria reaching 3 per cent. of sugar.

There are no records of complete removal of the pancreas in human beings. Partial resections of the gland have been followed by glycosuria or diabetes. In the latter cases, the interval between the operation and the appearance of the symptoms was as great as a year and nine months, and the result may have depended upon a progressive degeneration. That the pancreas in human beings shows marked pathological changes in cases of diabetes is shown by numerous observations. Of the causes of the pathological changes in the pancreas which produce diabetes, the chief one is supplied by concretions in the ducts. Of 72 cases 14 were due to pancreatic calculi (Hansemann). The pancreatic changes consist of secondary atrophy (see CHRONIC PANCREATITIS, p. 1153). Next in frequency come the primary atrophies, fibrous indurations, and instances of interstitial lipomatosis. Attention should be paid to another possible explanation of pancreatic atrophy, namely, a cachectic condition which may be the result and not the cause of diabetes (Friedreich, Naunyn). Hansemann has undertaken to distinguish between the two forms of pancreatic atrophy. In the cachectic form, the organ is cylindrical, and sharply demarcated from its surroundings. Microscopically, the stroma and glandular cells are uniformly atrophic. In diabetical atrophy, there is interstitial pancreatitis; on account of adhesions, the gland is with difficulty separated from its surroundings, and presents a brownish coloration (although it is not pigmented), is of smoother texture, and microscopically shows hypertrophy of the stroma and atrophy of the secreting cells and islets of Langerhans (*intra-acinous pancreatitis*). Other forms of interstitial pancreatitis may be associated with diabetes. Hansemann has seen diabetes in an early case of interstitial pancreatitis, and is of the

belief that a particular form of pancreatitis, which he calls granular atrophy, is always associated with diabetes. Hanot has described cases of bronzed diabetes (*diabète bronzé*) in which, besides pancreatic disease, hypertrophic cirrhosis of the liver has been present (p. 889). Opie says, 'clinically, the picture is one of a rapidly fatal diabetes mellitus associated with cirrhosis of the liver, usually the hypertrophic variety. Bronzing of the skin is not constant, but has been present in the majority of cases. At autopsy, has been found a deep pigmentation of the liver and pancreas, associated with cirrhosis and interstitial pancreatitis.' Opie has recently described an instance of pancreatitis in which the lesions of the pancreas were limited to the islets of Langerhans, which showed hyaline degeneration.

Although the evidence is not complete and unmistakable, yet it must be regarded as proving that the pancreas in man, as in animals, is intimately concerned in regulating the carbohydrate metabolism of the organism, and that a failure of the function of the organ in man—analogueous to total extirpation in animals—is followed by symptoms of diabetes. That this failure is independent of the digestive function is proven by the results of ligature and occlusion of the ducts, and that it depends upon an internal secretion supplied by the pancreas to the blood is highly probable. Whether this hypothetical secretion is the product of the cells of the islets of Langerhans is unproven. Should future study prove this view to be true (and the possibility is strengthened by Opie's observation) a closer differentiation of pancreatic disease than exists at present will be rendered necessary, and in this light the discrepancies in the relation of diabetes and pancreatic diseases, now impossible of explanation, may receive their solution.

The search for the cause of pancreatic diabetes has given rise to much ingenious speculation and numerous experiments. Thus far no satisfactory theory has been advanced, nor have the views put forward found support in experiment. Von Mering and Minkowski proposed two alternatives: (1) there takes place after extirpation of the pancreas an abnormal accumulation of injurious substances which it is the function of the gland to destroy; or (2) an important and irremediable function in the regulation of the carbohydrate metabolism is abolished by the removal of the pancreas. The former alternative was abandoned by Minkowski himself in later writings. The latter still has the greatest number of adherents, although it possesses a purely hypothetical basis; its adoption would bring the pancreas into line with other organs, e.g. thyroid gland and adrenals, which secrete internally or exhibit, according to Hansemann, a positive function. The manner of the regulating mechanism of the internal secretion is differently conceived of by different writers. Lépine has concluded, upon an experimental basis, that the normal pancreas contains a glycolytic ferment, which finds its way into the lymph and blood, where it is contained within the white blood-corpuscles. The ferment controls the consumption of sugar by the tissues. Its absence is followed by hyperglycemia and diabetes. The existence of the hypothetical ferment has not been rendered more probable by the results of subsequent experiments, carried out by a number of investigators, and the occurrence of a specific glycolytic power in the blood has, indeed,

been rendered highly doubtful. Chauveau and Kaufmann have proposed the opposite hypothesis. In opposition to the view that diabetes results from diminished sugar-destruction, they have set up the theory that it depends upon increased sugar-production by the liver. In respect to this function, they regard the liver and pancreas as closely related and mutually dependent organs. The pancreas regulates, through the nervous system, the production of sugar by the liver. The inhibitory centre is located in the medulla oblongata, and the centre of irritation is in the upper cervical region of the spinal cord. The pancreas acts upon the centres in opposite ways. The 'nervous' theory, however, was not wholly confirmed by further experiments; and Kaufmann in a later paper admits a partial direct influence of the pancreas upon the functions of the liver. Minkowski is certain that in experimental pancreatic diabetes, the fault is not increased production of sugar, but disturbances of consumption. The brothers Cavazzani would explain diabetes following extirpation of the pancreas as brought about by consequent alterations of the liver. Experiments have shown that irritation of the coeliac plexus causes an increase of sugar in the hepatic blood. A similar irritation may follow removal of the pancreas. Hence an excess of sugar is produced in the liver, the metabolism of that organ is increased, and degenerations result. The theory is scarcely tenable in view of the effects of the transplantation of the pancreas. Thus far, none of the theories advanced suffices to explain all of the phenomena observed. What appears to be beyond doubt is that diabetes results from the failure of a special internal function of the pancreas, and that it is not due to nervous lesions or to the absence of the digestive secretion of the pancreas.

7. Tumours.—*Carcinoma*.—The most important neoplasm of the pancreas is carcinoma, which, while by no means frequent, is probably less rare than statistics would seem to indicate. Primary carcinoma of the pancreas is, however, much less common than the secondary disease. Decision as to the primary seat of the cancer is not always easy, since tumours of similar structure may spring from the duodenum. Even the microscopical examination will not always suffice for making a distinction, and carcinomata springing from the ducts may grow to simulate cylindrical-celled epitheliomata.

The most common seat of the tumours is the head of the organ. The most common form is the scirrhus cancer; only rarely are encephaloid and colloid or cylindrical-celled tumours found. When it is the seat of carcinoma, the pancreas is enlarged and the tumour-nodules vary in size from that of a bird's egg to that of a child's fist; only rarely have larger masses been observed. The portion of the organ not implicated by the tumour may be entirely normal, or show only an increase of connective tissue. By extension of the tumour, the liver, stomach, duodenum, and gall-bladder may become implicated. The omentum, mesentery, large intestine, and other organs suffer less frequently. On account of the predilection for the head of the pancreas, the tumour frequently, even at an early period, compresses the common bile-duct. Compression of the duodenum and stenosis of the pylorus may also result. More rarely compression of the colon, the portal vein, the aorta, the splenic and mesenteric vessels, and inferior vena cava has been noted.

Males are affected nearly twice as frequently as females. The disease has been observed in early life, but its chief incidence has been noted in the four decades, from the thirtieth to the seventieth year inclusive.

SYMPTOMS.—A satisfactory diagnosis is not often possible. The symptoms arise more especially from implication of adjacent organs, and only rarely can be traced to the pancreas itself. Disturbances of digestion appear early; there is more or less loss of appetite, or, on the other hand, abnormal sensations of hunger, pressure, and discomfort following meals, fullness in the epigastrium, heart-burn, eructations, nausea, and even vomiting. As the disease progresses these symptoms become even more marked. Complete anorexia, a positive loathing of food, especially meat, may supervene. Whether or not these symptoms are referable wholly or in part only to implication of the stomach and duodenum has not been decided. There are, however, cases in which the appetite and functional activities of the stomach do not suffer, although, despite the ingestion of a considerable amount of food, there is progressive loss of weight and strength. The disturbances of indigestion are increased if the tumour compresses the pylorus and duodenum, since, as a consequence, dilatation of the stomach and stagnation of the ingesta occur. Perforation of the tumour into the stomach or duodenum is associated with hæmatemesis. Chemical examination of the gastric contents has shown, in a few instances, absence of free hydrochloric acid, although the stomach was not anatomically affected. On the other hand, a case is recorded in which, although the pylorus was compressed, the gastric contents showed 2.6 per cent. of hydrochloric acid.

One of the most notable symptoms is jaundice, which occurs, often early in the disease, not so much in consequence of invasion of the parapancreatic structures as from compression of the common bile-duct in its course through the head of the pancreas. While its onset may be sudden and follow an attack of colic, it usually appears gradually, and shows a steady increase in intensity. On the other hand, jaundice may be entirely wanting.

As indicating interruption of the normal functions of the pancreas, fatty stools are to be noted. The condition has been not infrequently observed. Chemical examination has revealed one-fourth of the entire content of the dejecta to consist of fat. There is disturbance also of the proteid-digestion; the fæces contain undigested muscle, &c., and the total volume of the stools is greatly increased. Instances have been observed in which much of the food passed through the intestine undigested. The exclusion of bile also produces characteristic changes in the stools.

Pain in the epigastrium is usually present; it may be intense and continuous or intermittent. After the extension of the tumour to other viscera, the pain may cease to be localised. Sensitiveness to pressure over the region of the pancreas may become a marked and distressing symptom. After a time, a tumour may be palpated, although the fact that the pancreas lies behind the stomach, and is covered in part by the left lobe of the liver, makes this sign less reliable. Osler estimates that a tumour or resistance is to be made out in 20 to 25

per cent. of all cases. The tumour may be smooth, nodular, or lobulated; it may be sharply circumscribed, and when limited to the head of the pancreas will be found to the right of the vertebral column and in the region of the pylorus. Except in rare cases, it is fixed and immovable. Pulsation, conducted from the aorta, may be observed, and has led to a wrong diagnosis of aneurysm. Confusion with the distended gall-bladder has also occurred, and is a real danger. In some cases two tumours have been made out, one of which was an enlarged and turgid gall-bladder. Pressure of the tumour upon the portal vein has produced ascites: the exudate is clear and serous, and only rarely has chylous fluid been observed (Senti). The enlargement of the spleen and appearance of hæmorrhoids doubtless depend upon compression of the vena porta. Pressure upon the ureter has produced hydronephrosis (Récamier).

General failure of nutrition, increasing epigastric pain and cachexia are present towards the end. In pancreatic carcinoma, however, the cachexia develops relatively early, so that some authors have viewed it as especially characteristic of this form of the disease. The volume of the urine may be increased; albumen is commonly found, sugar much more rarely. Marallié found among fifty reported cases the occurrence of sugar in thirteen, and he expresses the opinion that if looked for earlier it might possibly be found more frequently. Cases have been reported in which it was found at certain stages, but disappeared just before death. On the other hand, cases of complete destruction of the gland by cancer have been noted without the presence of sugar in the urine, a fact attributed by Hansemann to the persistence of the power of internal secretion in the cancer-cells.

The points of greatest diagnostic importance are: intense and permanent jaundice, dilatation of the gall-bladder, rapid emaciation, and the presence of a tumour in the epigastric region. Of less importance are features pointing to the disturbance of the functions of the gland (Osler).

The duration of the disease varies greatly. Some cases end fatally two to three months after the appearance of the first symptoms. Other cases have lasted as long as two years. Most authors take six months as the average duration. The prognosis is always grave. Several patients (six according to Körte) have recovered after surgical operation. Drugs are useless, although certain concomitant symptoms call for the exhibition of appropriate medicaments.

Sarcoma of the pancreas is a rare disease. Of those described, the majority have been secondary. On this account the type of the tumour has varied with that of the original growth.

8. **Tuberculosis.**—Miliary tubercles as a part of the disseminated disease are not uncommonly met with. Larger tuberculous foci occur much more rarely. The chief mode of propagation is hæmatogenous; but infection through the duct would seem to be possible, although no positive instances are on record. Tuberculous lymphatic glands may become united to the pancreas by adhesions, and growth take place into the pancreas. No symptomatology of tuberculosis of the pancreas is known, and no treatment is applicable. The disease has been revealed accidentally by the surgeon, and Sandler has removed a portion of the gland on account of tuberculosis with successful results.

9. **Syphilis** occurs under two forms: a chronic indurative pancreatitis and gummata. Occasionally both are found combined. The chronic indurative form is met with chiefly as an accompaniment of congenital syphilis, less often as a result of the acquired disease (see CHRONIC PANCREATITIS, p. 1153). The gummata in congenital syphilis may be numerous and miliary in size; in the acquired disease they reach a larger size, but are few in number or occur as solitary tumours. In Schlegenhauer's case the gumma was as large as a hazel-nut. The condition has no especial symptomatology; so far as known the clinical manifestations are those of a chronic interstitial pancreatitis. See also PANCREATIC CONCRETIONS, p. 334.

SIMON FLEXNER.

PANDEMIC DISEASES (πᾶν, all; and δῆμος, the people).—Epidemic diseases which affect groups of several countries or the world generally. See EPIDEMIC; and PERIODICITY IN DISEASE.

PANTICOSTA, in the Spanish Pyrenees.—Thermal waters. See MINERAL WATERS.

PAPILLOMA (*papilla*, a nipple, a wart).—A tumour composed of hypertrophied papillæ, either of the skin, or of a mucous or a serous membrane. See TUMOURS.

PAPULE.—SYNON.: Pimple; Fr. *Papule*; Ger. *Papel*.—A small, solid, circumscribed prominence of the skin. A papule may be conical, hemispherical, or flat at the top. The lesion may be the result of inflammatory exudation, of new-growth, or of accumulation of some secretion deep in the skin. See LICHEN PLANUS (p. 871); and DYSIDROSIS.

PARACENTESIS (παρά, at the side; and κεντέω, I prick).—SYNON.: Tapping; Fr. *Paracentèse*; Ger. *Paracentese*.—The term Paracentesis is rapidly becoming obsolete, and replaced by other words such as aspiration, exploration, or drainage. The term, however, is still applied to 'tapping' operations on the abdomen, thorax, and head.

The insertion of a hollow tube, whether in the form of a cannula or of the modern exploring needle, is practised for two distinct objects: (1) For the purpose of diagnosis, i.e. merely to ascertain the presence and nature of fluid in a suspected region; (2) For the purpose of treatment, i.e. with the object of removing the fluid from the cavity. For diagnostic purposes an exploring needle is still constantly employed when fluid is suspected in the pleural cavity, lungs, or pericardium. On the other hand, the employment of this method in the case of the abdomen cannot be too strongly condemned. In this way vascular tumours have been punctured, and the patients have died from the resulting hæmorrhage. Collections of pus or of decomposing urine, as in certain cases of hydronephrosis, have been pierced by the needle, and the fluid has leaked into the peritoneal cavity and caused fatal peritonitis. The writer has seen a patient bleed to death owing to a few punctures that were made into a leucocythæmic liver with a view to localising a suspected hepatic abscess. See ASPIRATOR.

1. **Paracentesis Thoracis.**—The methods employed for diagnostic purposes and for treatment in surgical diseases of the pleuræ, lungs, and

pericardium will be dealt with in the articles PERICARDIUM, Surgery of; and PLEURA, Surgery of.

2. **Paracentesis Abdominis.**—Practically the only condition for which paracentesis is performed is ascites. The best instrument for this operation is Thompson's syphon-trochar, or one of its many modifications. In this instrument the cannula is fixed to the handle and has a lateral opening about its middle to which a long indiarubber tube is attached. The trochar is continued through the handle of the instrument and terminates in a button. The instrument is inserted in the ordinary way, the trochar is then drawn back until its head is past the lateral opening in the cannula, through which the fluid will then flow. The indiarubber tube must be carried into a vessel containing carbolic-acid solution. The result is that a syphon action is established of sufficient force to exhaust the cavity; at the same time the accidental entrance of air is rendered impossible.

In the case of the abdomen it is better not to use any apparatus that exerts any marked degree of suction. The operation is performed in the following way: The abdominal wall should be thoroughly cleansed with soap and water and then with turpentine. The bladder and rectum should be empty, and the maximum area of dullness definitely ascertained by percussion. The distended abdomen is then surrounded by a sterilised many-tailed bandage, and a small opening with a pair of scissors may be made in this just before the instrument is inserted. In ordinary cases it is best to puncture in the middle line, but if there be an enlarged uterus or some abdominal tumour it may be more convenient to perforate in the linea semilunaris or some other spot. If possible, the patient should be placed in a chair which can be tilted backwards if necessary in the event of the patient fainting. If the instrument is sharp a local anæsthetic is not necessary. It is sometimes advisable, in order to allay the apprehensions of the patient, to inject a little sterilised water or β -eucaine. The instrument is pushed firmly and quickly through the abdominal wall, and the trochar is then withdrawn leaving the cannula in place. As the fluid gradually escapes the bandage can be tightened by the nurse or assistant. If the patient shows any signs of collapse it is advisable to remove the cannula, or, at any rate, to stop the flow of fluid, and place the patient in a horizontal position until he recovers sufficiently for the evacuation to be continued. On withdrawing the cannula the wound in the skin may gape a little, in which event one or two stitches should be inserted. The dribbling away of fluid through the puncture-hole is, if anything, beneficial, and an antiseptic dressing removes all fear of septic invasion. *See also* PERITONEUM, Acute Inflammation of; and PERITONEUM, Tuberculosis of.

Paracentesis Capitis.—This operation is sometimes performed in cases of chronic hydrocephalus. When the intra-cranial pressure is very high there is improvement as soon as sufficient fluid has been let out. Within a short time, however, the former tension is generally again reached. Of recent years intermittent withdrawal of fluid by means of a trochar has been tried on a large scale, but with few, if any, instances of permanent benefit. If it is advisable to withdraw some fluid in any given case a small-sized trochar of ordinary shape is sufficient. No aspiration of any kind should be employed. The skin having been properly cleaned

the trochar is inserted through the anterior fontanelle, at some little distance from the middle line, so as to avoid wounding the superior longitudinal sinus; the cerebro-spinal fluid immediately spurts out with considerable force. Very soon, however, the fluid ceases to flow, excepting a few drops with each expiratory movement. The cannula is then withdrawn and the puncture closed with collodion.

HENRY PERCY DEAN.

PARAD, in Hungary.—Sulphate-of-iron waters. *See* MINERAL WATERS.

PARÆSTHESIA (*παρά*, beside, hence amiss, incorrectly; and *αἴσθησις*, sensation).—A term applied to abnormal sensations experienced by a patient, distinct from mere excess or diminution of feeling; for example, tingling, itching, and formication. *See* SENSATION, Disorders of.

PARALDEHYDE-HABIT.—*See* HABITS.

PARALYSIS (*παράλυσις*, weakness, palsy).—By paralysis is meant the inability to move voluntarily a certain muscle or muscles, or to appreciate any of the ordinary sensory stimuli. This implies an interruption or impairment of the continuity which normally exists between the peripheral apparatus and the central nervous mechanism. Strictly speaking such an interruption may either be in the centripetal or sensory structures, leading to impaired or disordered sensibility, or in the centrifugal or motor structures, causing impairment or loss of motor power, or may involve both simultaneously. Sensory impairment, however, may, on account of the erroneous impressions which reach the centre and determine or guide movement, lead to what appears to be motor paralysis. Locomotion, and the accurate use of the upper limbs, may thus be rendered impossible. Such a condition is seen in the ordinary form of locomotor ataxy or tabes dorsalis, and in cerebellar disease. In some cases we have to deal with disorder or impairment of both sensory and motor mechanism, but in the great majority of cases of paralysis the essential lesion is one of the motor apparatus, and to this we shall direct our chief attention.

The motor apparatus consists of nervous and muscular elements. It is true that diseases of bones and joints also lead to interference with motor power, and to that extent may be regarded as causes of paralysis. But such conditions do not necessarily imply any lesion of the nervous or muscular apparatus, and need not be considered here. So that in reference to the causes of paralysis we have to consider only diseased conditions of (1) the nervous system; (2) the muscular apparatus.

The motor nervous system consists of two elements or neurons—the upper motor segment, or upper neuron, comprising the pyramidal cortical cells, and the fibres of the direct and crossed pyramidal tracts, situated respectively in the anterior columns, and the lateral columns of the spinal cord. The lower motor segment or lower neuron comprises the cells of the anterior horns, the motor nerve-fibre, and its termination in the muscle. Between these two neurons it is thought that there is no absolute continuity, but that there is such close contiguity between the branching terminations of the pyramidal fibre and the pro-

cesses of the cells of the anterior horns that the transmission of impulses is rendered quite easy.

Such is the motor nervous mechanism for the limbs, and the parts of the body generally related to the spinal cord. Certain muscles, however, are not supplied from the spinal cord, but from the cranial nerve-nuclei. For these, of course, there is no fibre in the pyramidal tract of the cord, although there is the analogue to this in the fibre which connects the cortical cell with the cell in the nucleus of the cranial nerve related to it, this cell being itself the homologue of that in the anterior horns of the spinal cord, and the fibre from it to the muscle being an ordinary motor nerve-fibre. Thus a muscle in the face is supplied by a fibre from the facial nucleus in the pons, the cells of this nucleus being closely analogous to those of the anterior horns in the spinal cord, and in, at least, close contiguity to the terminations of the fibres which are a direct continuation of the pyramidal cells in that part of the cortex known as the face-area. So that in the parts related to the cranial motor nerves also, we have an upper and lower motor segment or neuron—the upper comprising the cortical cell and its continuation to the vicinity of the nuclear cell, and the lower comprising the nuclear cell and its nerve-fibre to the muscle.

The symptoms accompanying lesions of the two neurons are as a rule quite distinctive. If the lesion is one of the *upper neuron* there is present, loss of power in proportion to the severity of the lesion, and distributed according to its situation. Rigidity or spasticity is also as a rule present, and accompanying this exaggeration of the deep reflexes. If the lesion is such as to affect parts which subserve the lower limbs ankle-clonus is frequently present, its presence or absence depending upon the severity of the lesion. The plantar reflex is also modified, so that the toes, instead of being, as they normally are, flexed, on tickling the sole, are extended (*see BABINSKI'S SIGN*). Along with these symptoms of loss of power, rigidity, and increased deep reflex the *nutrition* of the parts concerned is unimpaired, and the *electrical reactions* are not in any way altered. A common example of such a condition is hemiplegia, the result of a lesion in the cortex or just under it, or, as often happens, in the internal capsule. In such a case the lesion is one of the upper neuron, and the symptoms are those just described—loss of power, spastic rigidity, exaggeration of deep reflexes, and intact nutrition. It is true that in some cases of hemiplegia there is impaired nutrition of the affected limb or limbs, but this is almost, if not quite, invariably associated with some arthritic changes, and really independent of the lesion of the neuron. Sensory impairment may also be present, the result of involvement in the lesion of sensory fibres. Another example is furnished by the ordinary case of so-called lateral sclerosis or spastic paralysis. In this condition the disease is one of the pyramidal tracts in the cord—part of the upper neuron—and in this condition also there is loss of power, spastic rigidity, exaggeration of deep reflexes and unaltered nutrition, and the muscles respond normally to all electrical tests. It must not be forgotten that in many cases of, e.g., spinal caries or other condition giving rise to inflammation of the spinal cord (myelitis) there is, in addition to the ordinary symptoms of disease of the upper neuron, some impaired sensibility, perhaps also some muscular wasting and trophic changes. But

these additional symptoms are the result of disease affecting other structures which are situated close to that part of the upper neuron involved in the diseased condition.

If the *lower motor neuron* is affected there is an entirely different set of symptoms. Besides the loss of power there are wasting, impairment of nutrition, coldness and blueness of extremities or other evidence of trophic disturbance, impairment or loss of deep reflexes, and altered electrical reactions. The alterations in electrical reactions may briefly be described as reduced or suspended reaction to a faradic current and an altered galvanic reaction—altered, perhaps, not only in the quantity of the reaction, but also in its quality (*see ELECTRICITY IN MEDICINE*). Examples of such an affection are found in such a disease as the ordinary infantile spinal paralysis, in which the lesion is one of the cells in the anterior cornua of the spinal cord; in the ordinary form of Bell's paralysis—facial paralysis—in which the lesion is one of the facial nerve as it passes through the temporal bone; in multiple alcoholic neuritis, in which the peripheral nerves of the limbs are the parts principally and often exclusively affected, and in post-diphtheritic paralysis. In all these forms of paralysis we have loss of power, muscular wasting, impaired or lost deep reflexes in the parts affected, trophic changes, and altered electrical reactions; so that it is impossible to mistake them for diseases of the upper segment. It is true that in multiple neuritis there is often a kind of rigidity present, but it is not of the spastic type found in lesions of the upper neuron, but is dependent upon structural alterations and contraction of the muscles themselves, the result of interference with their nutrition.

One disease of the motor nervous system merits particular attention here because it is one in which both the upper and lower neuron are affected—the disease known as amyotrophic lateral sclerosis. As its name implies, this is a disease in which symptoms of lateral sclerosis are associated with those of muscular atrophy; so that the prominent symptoms are loss of power, spastic weakness and muscular wasting. In some cases the spastic weakness is the first sign—affection of the upper motor neuron; in other cases the muscular atrophy is the first sign—affection of the lower motor neuron; in other cases there is evidence that the spastic condition and the muscular atrophy are simultaneous in onset. It seems likely that in this disease we have to deal with a degeneration of the whole motor system, and that this degeneration may show itself in some cases first in the upper neuron, in other cases in the lower, while in still a third class the evidence may point to a simultaneous affection of both neurons. In many instances it is the case that not only are the parts of the motor nervous system subserving the limbs affected, but also the parts connected with the face, tongue, and deglutition affected. In other words, the homologues in the pons and bulb of the anterior-horn cells of the spinal cord may be affected, and so also may the upper neurons related to the same part suffer, viz. the fibres between the cortex and the bulbar and frontal nuclei. In clinical language amyotrophic lateral sclerosis has frequently associated with it the symptoms of labio-glosso-laryngeal or bulbar paralysis.

Morbid conditions also of muscles give rise to motor paralysis. The muscles are so closely related

to the nervous system that they may almost be regarded as the lowest part of the lower motor neuron. So far as the clinical signs are concerned in one large class of muscular diseases causing paralysis—the so-called myopathies—such a conception seems quite reasonable, for in those diseases we have loss of motor power, impaired nutrition—obvious wasting, or apparent hypertrophy, due to an overgrowth of connective tissue and fat with wasting of muscular fibres—loss of deep reflexes and altered electrical reactions. In those diseases no constant change in the nervous system has been discovered, so that we are led to believe either that the changes there are such as the methods used so far have failed to reveal, or that the disease, as seems likely, is essentially one of the muscular tissue itself.

Certain other diseased conditions of muscle must be mentioned, as they cause paralysis. Such is the group of rare diseases of which the so-called Myotonia or Thomsen's disease is the type. In this condition a spasm or cramp of muscles temporarily interferes with their use. As the muscle is used the spasm disappears. The condition known as *Myasthenia* is characterised by great weakness in certain muscles, often in those supplied from the bulb, and has occasionally proved fatal. Careful examination has failed to reveal any change in the nervous system. See MYOPATHY; MYASTHENIA GRAVIS; and special articles on other forms of paralysis. JAMES TAYLOR.

PARALYSIS, ACUTE ASCENDING.—**SYNON.**: Landry's Paralysis. See SPINAL CORD.

PARALYSIS AGITANS.—**SYNON.**: Shaking Palsy; PARKINSON'S DISEASE; Fr. *Paralysie Tremblante*; Ger. *Schüttellähmung*.

DEFINITION.—A disease of advanced life; progressive in its course; and characterised mainly by tremors of the limbs occurring independently of muscular exertion, rigidity of muscles, a tendency in walking to loss of equilibrium, and a peculiar attitude of body, and fixed expression of countenance.

ÆTIOLOGY.—The causes of paralysis agitans are obscure. It is rarely met with prior to forty years of age, but becomes more and more frequent as life advances. It affects both sexes, but men probably more frequently than women. There is little reason to regard it as hereditary. It has been attributed to violent emotion, to excessive bodily fatigue, and to exposure to cold and wet. It has also been referred to wounds or injuries involving nerves. In many cases no cause is assigned or can be discovered.

ANATOMICAL CHARACTERS.—The disease, no doubt, is one of the nervous centres probably of the cortex. But no distinctive lesion has yet been discovered in these parts. Sclerotic and other degenerative changes, evidences of sanguineous exudations in the course of some of the smaller vessels, diseased arteries, and various coarse lesions, have not infrequently been met with in the cord and brain; but the morbid changes hitherto observed have been variable in seat and character, and such only as are frequently present under other circumstances in persons who die in old age.

SYMPTOMS.—Paralysis agitans, with few exceptions, comes on insidiously. The patient is first attacked with occasional tremors in a hand, a

thumb, or a foot. These attacks come on irregularly, without obvious cause, and last for an uncertain period. But gradually they increase in frequency, duration, and severity, and spread from the part first involved, until, at length, probably all the limbs become implicated. In most cases the tremors, commencing in a hand or foot, by slow degrees invade the rest of the limb, and thence spread in hemiplegic fashion to the other limb of the same side. Less commonly the affection spreads in the first instance from one leg to the other; and very rarely do both arms suffer, the legs remaining free, or does the affection involve the limbs diagonally. Associated with the tremors, sometimes preceding them, but much more frequently coming on at a later period of the disease, there may always be observed a peculiar rigidity of the muscles. This is often attended by cramp-like pains, and, like the tremors, is liable at first to more or less obvious and prolonged intermissions. It implicates the muscles of the head and neck and trunk, as well as those of the extremities, and the flexor muscles in greater degree than their opponents. Another remarkable characteristic of the disease, always developed sooner or later, is an inability to maintain equilibrium when walking is attempted.

When paralysis agitans is fully developed, and the several phenomena above enumerated are associated, the collective symptoms produce a very remarkable and characteristic picture. The tremors involve the arms and legs; the head and neck remaining, as a rule, absolutely free. They consist of fine and rapid oscillations, which are more or less constant, but liable to exacerbations; cease during sleep; can occasionally be arrested temporarily by voluntary effort; and often occur with exceptional violence when the patient is otherwise at absolute rest. The movements of the hands are peculiar. The thumbs are usually extended, and the fingers flexed upon them; and collectively they move as though the patient were rolling a pencil or crumbling bread. The oscillations, however, are not limited to the hands, but involve the wrists and other joints of the upper extremities. The tremors of the lower limbs, especially when the patient stands, are necessarily transmitted to the rest of the body. In some cases the entire head, or the lower jaw, presents tremors like those affecting other parts of the body. In rare cases the tremor may be absent, but the other striking features in the clinical picture remain. The rigidity, which affects in a greater or less degree all the muscles, imparts a striking character to the patient's attitude and aspect. It causes the arms to stand out slightly from the trunk; the elbow- and wrist-joints to be slightly flexed; the hands to be tilted towards the ulnar side, and to rest in front of the abdomen at or near the waist; and the fingers to be flexed or distorted at their several joints. It causes the trunk to incline forwards, as the patient stands or walks; the knees to be slightly bent; and the feet to be extended at the ankles; so that he rests upon his toes. But, above all, it causes the head and neck to be thrown forwards, and to be retained rigidly in that position, and the features to be immobile and inexpressive. This peculiar fixity of the head and neck and face, associated as it is with constant tremors in the limbs, constitutes a very striking feature of the disease. The difficulty of maintaining equilibrium, though no doubt increased largely by the presence of muscular tremors and rigidity, is not

wholly due to them, for it may be well developed at a very early stage of the disease. Moreover, it may be long delayed. When thus affected the patient has some difficulty in rising from his seat; and, before he starts off walking, he probably hesitates a little, as though for the purpose of balancing himself. Then, with his body bent forwards, he begins to walk, perhaps with some care, but soon his steps become rapid and short, and he runs forwards in spite of himself, and if not arrested probably falls. Sometimes the tendency of the patient is to run backwards, even though the body incline forwards. Often in these cases, while the patient is being propelled forwards apparently in spite of himself, a sudden pluck at his clothes will reverse or alter the direction of his accelerating movement. These phenomena are not attended by vertigo.

Other symptoms less striking than the above, but of more or less importance, are usually present in shaking palsy. There is generally, even from the first, a great sense of weariness in the affected muscles, especially after exertion or an attack of tremors; but, contrary to what might be supposed, the tremulous and rigid muscles are, as a rule, markedly stronger than their as yet unaffected fellows. The patient, more particularly late in the disease, becomes excessively irritable and fidgety, so that at night especially he finds it difficult or impossible to place himself in a comfortable position; he is apt also to suffer from a painful sense of heat, mainly referred to the epigastrium and back. Speech generally becomes markedly affected, not from loss of language, but from difficulty of enunciation. Words are uttered slowly, and with manifest effort. Associated with this there is often tremulousness of the tongue. But the slowness and difficulty of utterance, which are often associated with slowness and difficulty of deglutition, constitute only one manifestation of the general slowness and difficulty of movement which, for the most part, characterise the disease. Sensation is not impaired; and the patient retains his mental faculties, as well as control over the rectum and bladder, as a rule, but sometimes loss of memory and even dementia occur, as evidence of senile cerebral degeneration.

DIAGNOSIS.—The affections with which paralysis agitans is most likely to be confounded are disseminated sclerosis, and mercurial tremors. But in the former of these the tremors occur only when the muscles are in use, and for the most part involve the head; the limbs early become paralysed; the patient has no tendency to run forwards or backwards; and generally nystagmus is present. In the latter affection there is probably a history of exposure to the fumes of mercury; the tremors involve not only the limbs, but the head and neck, and are symmetrical; and there is an absence of the peculiar gait of paralysis agitans. See MERCURY, Diseases arising from.

PROGRESS AND TERMINATIONS.—Paralysis agitans is of slow and often irregular progress, and usually lasts for many years; indeed it may be many years before it attains its full development. In rare cases it is recovered from in the early stage; but for the most part it is incurable. In its last stage the patient becomes confined to his couch or bed; the muscles waste; the tremors, though generally extreme at the time, occasionally cease; the mental powers fail; bed-sores form; and general prostration ensues. Death is due either to asthenia, or to

some intercurrent disorder, more especially pneumonia.

TREATMENT.—In treating shaking palsy it is of importance to give careful attention to all hygienic measures, and to promote the patient's health, if need be, by tonics. Specific treatment has proved of little or no service. Nervine tonics and sedatives have been largely employed; and those which have found most favour probably are iron, strychnine, and hyoscyamus. The systematic use of baths has occasionally proved of temporary benefit.

J. S. BRISTOWE.

H. H. TOOTH.

PARALYSIS, BULBAR.—See p. 840.

PARALYSIS, DIPHTHERITIC.—See p. 402.

PARALYSIS, ERB'S.—See p. 170.

PARALYSIS, INFANTILE CEREBRAL.—

Cases of paralysis the result of cerebral disease in children may be classified in different ways. Probably the classification most generally useful is a clinical one into cases (1) in which the paralysis affects both sides of the body—cases of cerebral diplegia, and (2) in which only one side of the body is affected—cases of infantile hemiplegia. Such a classification does not of course exclude from the first class those cases in which, although both sides are affected, there is a preponderating affection of one side. The classification according to which cases of cerebral paralysis in children are divided into those in which the paralysis is present at birth, and those in which the paralysis is of subsequent onset, is scarcely a satisfactory one, for although in most cases of diplegia the paralysis is present at the time of birth, there is a certain number in which the affection is of later onset. In the present state of our knowledge it is not possible to classify these cases according to their aetiology.

1. **Cerebral Diplegia.**—See CEREBRAL DIPLEGIA.

2. **Infantile Hemiplegia.**—From this group are to be dissociated cases of hemiplegia in children occurring in the course of an acute illness, such as rheumatism or diphtheria or scarlet fever, in which the condition is one of embolism associated with the endocarditis which is so often present in these diseases. Such cases in no way differ from cases of a similar origin occurring in adults, except in so far as growing structures are interfered with and consequently individual mal-developments ensue. But cases of infantile hemiplegia proper are those which occur in the early years of life—the attacks are ushered in with an acute febrile condition almost invariably associated with unilateral convulsions, the side affected in the convulsions being that subsequently paralysed. The paralysis is of the usual type found in adults—face, arm, and leg being affected, the upper limb most obviously and severely. The subsequent condition is similar to that found at the commencement, so far as paralysis is concerned, only the paralysed limbs are found as a rule to become smaller than those on the other side. This is the result of interference with the development of the affected limbs, and cannot rightly be regarded, as it is sometimes spoken of, as atrophy. At first hemianopsia and sensory impairment are said occasionally to be present. These are never permanent. Mental impairment occurs occasionally, and epileptiform attacks frequently recur, affecting sometimes the paralysed side and sometimes

the non-paralysed side. Occasionally attacks of *petit mal* may be present, and the writer has seen such in a slightly hemiplegic patient, associated with post-epileptic automatism, in which the patient unconsciously carried out apparently purposive and often elaborate movements. Contractures, especially at the ankle, may be present, and there is usually exaggerated knee-jerk, with perhaps also ankle-clonus. The degree of mental impairment varies, but is usually slight.

Involuntary movements are frequently present; the most common is the so-called mobile spasm or athetosis, in which a cycle of slow movements is carried out in the limbs of the affected side. The upper limb is most affected, but movements may also be present in the face and in the leg. Movements of another character may also be present—short, jerky, and only evoked on voluntary movement, in these characteristics closely resembling those of disseminated sclerosis. Sometimes the movements resemble somewhat those of chorea, but the name 'post-hemiplegic chorea' has probably been applied to jerky spasmodic movements occurring on voluntary effort, such as have just been alluded to. Briefly then these cases of infantile hemiplegia differ from the hemiplegia of adults in the mode of onset—usually with an acute febrile condition and a severe convulsion or a series of convulsions—in the absence of any renal or cardiac disease, in the frequent persistence of fits, and in a not uncommon association of involuntary movements of the paralysed limbs.

PATHOLOGY AND PATHOLOGICAL ANATOMY.—Few of such cases can be examined when the condition is still recent, because few die. The subsequent condition then is one of porencephaly or of fibrosis of a part of the cortex, and these conditions are probably the result of one or other of the several morbid conditions alleged by different authorities as the initial lesion. Strumpell regards as most likely an acute inflammatory condition analogous to that occurring in the spinal cord, causing infantile paralysis (acute anterior poliomyelitis); some likelihood is given to such an hypothesis by the facts that a condition like this has actually been demonstrated in adults, and that cases of cerebral paralysis have occurred simultaneously with cases of spinal paralysis in different members of the same family. Other writers have regarded the condition as one of vascular blocking—according to some blocking of superficial veins, according to others of arteries. In some cases embolic blocking has been found, but these have been cases occurring in the course of such diseases as diphtheria and scarlet fever; and, as we have already stated, not therefore to be regarded strictly as cases of the infantile hemiplegia under consideration.

Treatment must concern itself with (a) the condition at the onset of the paralysis, and (b) the resulting paralysis, and perhaps fits. As regards the initial state, a dose of calomel should at once be administered. If convulsions are present it is necessary to administer bromide in a fairly large dose, even to a young child, and in this condition it is best given by the bowel, \mathfrak{zss} or $\mathfrak{3j}$ being administered in about \mathfrak{zviij} of warm water. Further purgation may be necessary if the convulsions persist, and if the child is not feeble and wasted. The subsequent paralysis is best treated by massage and such passive movements as will best prevent contractions and rigidity, and if con-

vulsions persist they must be treated by the systematic use of bromides. Firm discipline and educative influences of a special kind may be needed if there be some mental impairment, as occasionally happens. See IDIOCY; and CHILDREN, Training of.

JAMES TAYLOR.

PARALYSIS, INFANTILE SPINAL.—SYNON.: Acute Atrophic Spinal Paralysis; Acute Anterior Poliomyelitis; Fr. *Paralysie Essentielle de l'Enfance* (Laborde); *Paralysie Atrophique Graisseuse de l'Enfance* (Duchenne); Ger. *Kinderlähmung*.

DEFINITION.—Paralysis chiefly in the limbs and trunk, acute in onset, various in distribution and extent, followed by recovery in some parts and persistence in others, with rapid muscular wasting and the electrical reaction of degeneration; due to inflammation of the anterior grey matter of the spinal cord.

ÆTIOLOGY.—The disease is most common in children during the first five years of life; three-fifths of the cases come on in the first three years, and hence the name by which it is often known; but it also occurs in older children and young adults. In infancy, males and females suffer with equal frequency; subsequently, the affection occurs more often in males. Hereditary influences take but a trifling share in its causation; nevertheless, instances are met with in which two members of the same family suffer. Season has a marked influence; three-quarters of the cases occur during the hottest third of the year. It often follows exposure to cold, as an unaccustomed cold bath in hot weather, or exposure to a draught when heated. The exposure is usually within forty-eight hours of the first symptoms of the malady. It occasionally succeeds a fall at an interval of a few days. Over-exertion probably predisposes to it; in children this cause can often be traced. It seldom follows any different malady; in the cases in which it has been thought to be secondary to some acute illness such as rheumatism, the symptoms of this have really been part of this affection.

PATHOLOGY.—The atrophied muscles are found to have undergone granular or fatty degeneration, with disappearance, to a greater or lesser extent, of the transverse striæ. Many fibres, however, are simply narrower than normal, and occasionally some increase in width is found. Oil-globules and numerous fat-cells are also found between the fibres. Ultimately, in the most wasted muscles, fibrous tracts occupy the place of the muscular tissue, but among them a normal fibre, with its striation preserved, can here and there be seen. The amount of change in the muscles varies according to the amount of wasting. When this is moderate most of the fibres present only slight changes.

The few examinations made in the early stage have revealed a condition of acute inflammation of the anterior cornua of the spinal cord, varying in degree at different parts, but almost limited to the grey matter, extending only a short distance into the adjacent white substance. At an interval of many months or years after the onset, a condition is found such as acute inflammation would leave: there is shrinking of the anterior horns corresponding to the most affected muscles, with increase of the neuroglial tissue in them, and disappearance of many of the motor nerve-cells, most that remain being reduced to small angular bodies, among which a few are seen of normal aspect. The

corresponding fibres of the anterior roots are degenerated, and the degeneration can be followed down the motor-nerves to their fibres in the atrophied muscles. The malady is thus an acute inflammation of the motor structures of the cord with secondary acute degeneration of the fibres proceeding from the motor cells, and secondary degenerative changes in the muscular fibres, which vary in intensity according to the acuteness and inflammatory character of the descending process in the nerves. But the general disturbance, which is often so conspicuous, suggests that the local inflammation is the result of a morbid blood-state, which has a special influence on the grey matter of the cord. The rare occurrence of groups of cases in a quasi-epidemic form has suggested that the toxic agent may be derived from without. Its frequent relation to cold must, however, be remembered. In these features it may be compared to acute pneumonia.

SYMPTOMS.—At the onset of the affection there are commonly symptoms of two classes: (1) the paralysis already mentioned, which is usually preceded by (2) indications of general illness, apparently the result of a morbid blood-state—malaise, pyrexia, vomiting, headache, and rarely general convulsions such as may attend any acute disorder in early life. These vary much in amount and duration; they may be severe, and last for days before the loss of power comes on, or they may be so slight, as to be unnoticed. When severe, there is so much prostration that the process of the onset of the paralysis may not be noticed; it may be mistaken for general weakness due to the acute disease or, as is often thought, severe gastric derangement. It may only be discovered when returning strength reveals local disability. There is frequently pain in the limbs, and sometimes the pain and tenderness in the nerves is so pronounced as to justify the suspicion of a simultaneous disseminated neuritis. The pain in the limbs and nerve-tenderness usually pass away in the course of a few days, but sometimes continue for weeks, so that even passive movements cause much pain. The paralysis, as a rule, is motor only. Sensation is impaired only in extremely rare cases in which inflammation is so intense as temporarily to extend to all the conducting columns of the cord. When sensation is impaired there is always incontinence of urine, and this may be met with when there is only motor paralysis; trophic changes and bed-sores are almost unknown, even in the acute stage of the disease. A slight local elevation of temperature in the most paralysed parts has been noted in the early stage, but subsequently the affected limbs are colder than the others. This is due, at least in part, to the loss of the aid to circulation which, in health, is supplied by muscular action.

Reflex action is necessarily lost in the parts related to the muscles involved. That from the skin is at first abolished wherever there is weakness, but it returns with or soon after recovery of power in the less affected parts. If the paralysis is persistent it remains absent. The muscle-reflex action is lost in the same or even greater degree, so that, for example, no knee-jerk is obtainable if the extensors of the knee are even slightly affected. In rare cases of cervical poliomyelitis the morbid process may spread into the lateral columns, so that, in addition to the wasting and paralysis of the arms, there may be paralysis without wasting in the

legs. In such cases the myotatic irritability in the legs may be, for a time, increased.

When the paralysis of a limb is incomplete, the muscles involved vary in different cases; and as different parts of two or more limbs may be affected, the combinations of palsy which result are extremely varied. Sometimes the muscles affected are those which are functionally associated; more frequently the affection is random. The degree of affection of the individual muscles also varies, the loss of power in some being absolute, in others only partial. In the legs the paralysis is rarely complete; most frequently it is partial, and the muscles below the knee suffer more often than those above the knee. The calf-muscles are affected less frequently than the anterior tibial or peroneal muscles, and hence talipes equinus is a common form of the deformity that ensues from the contraction of the less affected muscles. Sometimes all the muscles shorten, or the calf-muscles may be chiefly affected, giving rise ultimately to talipes equinus.

In the arm any of the muscles may be involved, but all are seldom entirely paralysed. The intrinsic muscles of the hand may suffer, and either the thenar muscles or the interossei may be most damaged. The muscles of the forearm are frequently affected, but the supinators may escape when the extensors are involved. Of all the arm-muscles, the deltoid is that most frequently affected; it may suffer alone or in association with others. The 'upper-arm type' of palsy, of Erb, is sometimes met with, in which the deltoid, supra- and infraspinatus, biceps, and supinators, are all involved; but the irregularity of grouping is shown by the fact that the triceps is often affected with these muscles. The serratus magnus is occasionally affected, and the upper part of the pectoralis major (which is normally associated in function with it) may then suffer, while the lower part escapes. The middle part of the trapezius, and other of the scapular muscles, are occasionally involved. The cervical muscles are rarely affected, but the diaphragm is sometimes paralysed; permanent wasting of the intercostals and trunk-muscles is rare: both these and the dorsal muscles may be weakened at first, and more on one side than on the other. This should be borne in mind, as curvature of the spine may be produced by allowing the patient to sit up while the muscles are still weak. The muscles supplied by cranial nerves are rarely affected; but instances of facial paralysis have been recorded.

COURSE.—The course of the disease has been already indicated. There are: (1) the initial stage of paralysis, lasting for a few hours, a week, or even a month; (2) a stationary period, which lasts from a week to a month; (3) a stage of 'regression,' during which the palsy lessens and passes away, except from certain parts in which wasting occurs; this period usually occupies from one to six months; and (4) a chronic stage, during which the atrophy continues, slight improvement may occur, but contractures and deformities are developed. These are due to distortion of the articulations in consequence of the contraction and permanent shortening of the muscles that are less affected than their opponents; the former become fixed by tissue-changes in their shortened state, and by secondary changes in the ligaments of the articulations. It is especially at the foot that these occur, constituting some form of talipes, especially talipes equinus or equino-varus, less commonly talipes calcaneus. In the case of

children, the growth of the most affected limb is inevitably hindered, and in the case of the leg this may render the effect of the paralysis more obtrusive as time goes on, by its interference with the gait. It is important that the friends should be warned of this, or they will regard it as an increase in the disease.

Wherever muscular tissue remains and some voluntary power returns, this slowly improves, as the muscular tissue that remains develops under the influence of use, and complete recovery, in even the very slightest cases, is extremely rare, and, on the other hand, death from the disease is equally uncommon. If it does occur, it is in the early stage, and it may result from the initial disturbance before the nature of the complaint is recognised. Occasionally death takes place at the end of the first week or ten days, from universal paralysis, or from some profound associated cerebral disturbance. Between the two extremes every variety of degree and extent of palsy is met with, from those in which there is partial affection of all the limbs, often severe in the legs, to those in which there is only a slight affection of certain muscles in one limb.

Relapses and second attacks are almost unknown. Sequelæ are also rare, although in a few cases some other chronic affection of the spinal cord has come on when the subjects of infantile paralysis have reached adult life. Thus, progressive muscular atrophy has been observed to start from a limb paralysed by this affection. Lateral sclerosis has also occurred in adults, the subjects of old infantile paralysis.

DIAGNOSIS.—A difficulty in diagnosis is only likely to occur in the early stage, when the vomiting, which is so common, is apt to lead to the opinion that the affection is only gastric disturbance; and the pyrexia may cause the case to be regarded as a general febrile affection. The mistake can only be prevented by attention to the state of the limbs, and by giving due weight to any sign of defective power. Later, the wasting, the state of the electrical reactions in the muscles, and the loss of reflex action without any sensory change, will usually be sufficient to allow a definite diagnosis to be made. In the earlier condition, the existence of the paralysis is not unlikely to be overlooked, and the immobility to be ascribed to prostration. But total immobility, and still less local immobility, is not produced by prostration. When the pyrexia ceases, while the loss of power persists or increases, the existence of paralysis is always unmistakable. In adults the danger of a mistake in the early stage is less, but the great pain in the limbs is apt to cause an erroneous diagnosis of acute rheumatism.

From chronic diseases of the spinal cord the condition is distinguished by its acute onset. Acute transverse myelitis may be closely simulated if the inflammation of the grey matter is bilateral; the age of the patient usually suggests correctly the nature of the disease. The usual form of transverse myelitis also occurs, as a rule, not in one of the enlargements, but in the dorsal region. Difficulty will, indeed, usually be prevented by the recollection of the fact that transverse myelitis in children is always severe poliomyelitis, spreading to the white columns. The very slow onset of pseudo-hypertrophic paralysis, developing as it does gradually with the child's growth, should sufficiently distinguish it from this disease.

Cases have been mistaken for acute multiple

neuritis, especially in young adults. The error is facilitated by the conspicuous affection of the nerve trunks in severe cases; but the symptoms at the onset, the irregular distribution of the paralyses and the absence of any tendency to preponderant affection of the extremities of the limbs, are distinctive features. Diphtheritic paralysis sometimes offers a difficulty, especially if the preceding sore-throat have been slight or unnoticed, but is distinguished by its gradual onset and special features. The distinction from cerebral palsy is usually easy; in this there is never loss of faradic irritability or of reflex action, nor is there extreme or local muscular wasting. The character of the convulsions also is different; those which take place at the onset of infantile paralysis are general, those which are associated with a cerebral palsy are usually unilateral or commence locally. The mistake is only likely in cases of poliomyelitis in which the arm and leg on the same side are involved.

The only diseases outside the nervous system which may be mistaken for infantile paralysis are those in which movement is associated with pain, such as hip-joint disease, acute necrosis of the femur or the humerus, or the affection known as scorbutic rickets, in which there are symptoms of scurvy associated with those of rickets (*see RICKETS*). A careful examination is usually sufficient to show that pain alone prevents movement. The preservation of the knee-jerk is often of great significance when the legs are moved.

PROGNOSIS.—In the vast majority of cases the disease involves no immediate danger to life. This is probably greatest when there is severe constitutional disturbance, before the characteristic paralytic symptoms have been noticed. There is also some danger from after-effects, especially in consequence of the slight power of resistance to other morbid influences which remains after the severe constitutional disturbance connected with the onset.

If the paralysis has remained stationary, that is, has not increased in extent, for twenty-four hours, the danger of further extension is small. Concerning the prospects of recovery, no answer can be given for a week or ten days, and its nature will depend upon the condition found to be present on electrical examination. The muscles which then have lost faradic irritability will certainly waste, will remain for a long time paralysed, and will probably be to some extent permanently disabled. If, however, there is no loss of faradic irritability at the end of this period, but it is apparent at the end of a fortnight or three weeks, the wasting will be slighter in degree, and some ultimate recovery may be expected even in the most affected part. When there is no loss of faradic irritability, the paralysis will pass away in the course of a few weeks or, at most, a few months. But this condition is unfortunately rare.

In the chronic stage, unless there is some sign of returning power within three weeks, very little recovery will occur. The retention of voltaic irritability in the muscles is so far satisfactory as showing that there has been no destructive degeneration in them, and that there are favourable conditions for the exertion of voluntary power. But it does not lessen the grave significance of the persisting palsy and loss of faradic irritability, indicating nerve-degeneration. If, on the other hand, at the end of two or three months some faradic irritability can be detected, however slight, improvement is pro-

able, and it may be considerable. It is always necessary to remember that in children an apparently increased disability may be due, after a time, to diminished growth of a limb, and may be consistent with progressive improvement.

TREATMENT.—The treatment in the early stage should be that of febrile states generally. The child should be kept at perfect rest, as much as possible on one side, and warmth applied over the affected part of the cord by poultices or fomentations. When there is spinal pain, marked relief is afforded by these means.

Both ergot and belladonna have been credited with the power of arresting the disease, although there is no definite proof of this. Either, however, may be employed apparently without fear of harm.

When the acute stage is over, tonics, especially iron and quinine, are needed. Strychnine also may be given in all cases, but it should not be commenced sooner than three or four weeks after the disease has become stationary. As the malady frequently occurs at an age at which ricketty conditions develop from any interference with the general health, treatment with the view of anticipating such constitutional effects should be adopted in young children. For this purpose iron and cod-liver oil are especially useful.

The use of electricity is an important part of the treatment, and, in order to prevent its ineffective or harmful employment in unsuitable circumstances, the reasons for its employment ought to be clearly understood. There is no evidence that it can or does influence the process of recovery of the damaged elements either in the cord or nerves. The reasons for its use depend upon the fact that the disease entails nerve-degeneration, and that the related muscular fibres undergo changes in nutrition and ultimately perish if no nerve-regeneration occurs. While the influence normally exerted through the nerves is in abeyance, the muscles are destitute of functional stimulation, and it is with the view of supplying the place of this that electricity is to be used. When both cell and fibre have perished, electricity can do no good; but where there has been damage but not destruction, so that the fibres recover and again become capable of conveying nutritional and volitional influences, electricity is of distinct service in preventing a disproportionate failure of muscular nutrition. Only voltaic electricity can stimulate the muscles when the nerve-fibres are degenerated, and this is consequently the form that must be used. The mode of application is determined by the fact that it is only when the current is interrupted that the muscular tissue is stimulated to contract. One terminal is kept still, the other being stroked down the muscles and lifted from the skin at each stroke. Some place the immobile terminal over the spine, but there is no evidence that the spinal cord is reached in this way. The negative pole will be that most generally useful in stroking the muscles, as the normally greater irritability to the negative pole frequently persists. Of course soft terminals, well moistened with salt and water, must be used, the skin also being thoroughly wetted, and a current should be used sufficiently strong to produce visible contraction, provided this does not cause distressing pain; if it does, it is better to be content with the strongest current that can be borne without emotional disturbance. The strength used should be gradually increased, as the child gets more accustomed to the application.

Systematic massage of the affected muscles is also useful. The muscles should be rubbed and gently kneaded daily, upward rubbing being especially useful. Great care should be exercised in keeping the affected limbs as warm as possible; and bronchial catarrh should be guarded against, especially in cases in which the respiratory muscles are involved.

The prevention of deformities of the spine or in connection with contractures causing displacement at joints must be carefully looked to. Mechanical appliances may have to be ultimately adopted, and not infrequently tenotomy is necessary. But such deformities should as far as possible be prevented by systematic movement and attention to posture. The process of slow improvement, once started, may go on for years by the slow growth of the muscle that has recovered under the stimulus of use, and this improvement may be augmented by various contrivances for allowing the muscle to act to the best advantage. It is especially important to compensate diminished growth by a thicker sole to the boot, so as to prevent the tilting of the pelvis and curvature of the spine that would otherwise ensue, and to keep the foot in a natural position.

W. R. GOWERS.

PARALYSIS OF THE INSANE.—See p. 573.

PARALYSIS, PERIODIC.—**SYNON.**: Family Periodic Paralysis; Intermittent Paralysis.

DEFINITION.—An affection in which attacks of temporary flaccid paralysis with loss of electrical excitability of the muscles occur from time to time, without loss of consciousness, and in which the patient recovers completely in the intervals between the attacks.

ÆTIOLOGY.—The malady is most common between the ages of twenty and thirty, but also occurs in older and younger people. Both sexes are attacked; either may transmit the disease, and several members of a family are usually affected, although isolated cases occur. Malaria has been suggested as a possible cause, because the attacks are periodic, because some of the patients have had malaria, and because quinine has done good in some cases; the plasmodium has, however, not been found in the blood of patients suffering from attacks of periodic paralysis. Muscular exertion induces the paralysis in some cases, but in others the attacks are most frequent when the individual is at rest. Certain articles of diet appear liable to induce attacks in some patients.

SYMPTOMS.—The attacks, as a rule, begin at night; but if the patient be awake a feeling of weakness is experienced in the legs, and then in the arms and trunk. The proximal segments of the limbs succumb before the distal; but in the end there is general and complete flaccid paralysis, including the thoracic muscles, so that respiration is carried on by the diaphragm alone. The face muscles, however, as a rule escape, as do those of mastication; but these and other muscles supplied by cranial nerves are occasionally attacked.

The paralysis usually reaches its maximum in three or four hours, after which recovery takes place in about the same length of time, in the reverse order to that of the onset, the legs being the last to recover. In some cases peculiarities of physical development have been observed, such as enlargement of muscles, which, however, were not correspondingly powerful. So, too, permanent weakness

has been noted in certain groups of muscles in some patients.

During the height of an attack the muscles lose their mechanical irritability, and no longer respond either to faradism or galvanism; the tendon-jerks are abolished, and the superficial reflexes may also be lost, though this is not always the case. With return of motor power the muscles regain their electrical excitability, and the tendon-jerks can again be elicited.

The mental faculties are preserved, articulation is not defective, the appetite is often lost, but no difficulty in swallowing is experienced, and there is no respiratory distress, although the rate of the respirations and pulse may be slightly increased. The heart becomes a little enlarged, and a systolic murmur indicative of mitral regurgitation may be heard at its apex; the murmur, however, disappears as the patient recovers. Sensation is unaffected, and the sphincters also escape, though no urine or feces are usually passed during an attack.

The attacks of paralysis last, on an average, from ten to forty-eight hours; they may recur daily or the interval may be a week, a month, or even a year.

MORBID ANATOMY.—None of the cases have come to autopsy, but portions of muscle removed during life have revealed increase in the diameter of the muscle-fibres, vacuolation, and waxy degeneration.

PATHOLOGY.—The affection has been supposed to be due to some form of auto-intoxication by a poison similar to curara; but it is not easy to be certain on what the poison acts, as we know of no poison that can in the same way prevent the muscles from responding to either form of electrical current. Whether the lower neurons are concomitantly affected or not, it seems necessary to assume that the poison acts directly on the muscle-fibres. In one case a toxic substance was isolated from the first feces passed by a patient after an attack of periodic paralysis, and when injected into rabbits and guinea-pigs it caused paralysis in them which disappeared in the course of forty-eight hours. The malady has been regarded by some as related to the muscular dystrophies; but there is little to justify this view. It has also been supposed to be allied to Thomsen's disease (*Myotonia congenita*).

DIAGNOSIS.—Where history alone has to be relied on, cases may be mistaken for epilepsy or post-epileptic paralysis. On the other hand, cases seen for the first time may be confused with 'Landry's paralysis' (*acute ascending paralysis*), unless a history of previous attacks be obtained.

PROGNOSIS.—Although the duration of life does not appear to be altered by the affection, the patients, as a rule, continue liable to attacks all their lives, but in a few there has been a tendency to recovery about the middle period of life.

TREATMENT.—Beyond the fact that quinine appears to have been of some use in a few cases, nothing is known to influence favourably the course of the affection.

J. S. RISIEN RUSSELL.

PARALYSIS, PSEUDO-HYPERTROPHIC MUSCULAR.—See MYOPATHIES.

PARALYSIS, SENSORY.—See SENSATION; Disorders of.

PARALYSIS, TOXIC.—Various kinds of paralysis, due to multiple neuritis in the main, are produced by such poisons as alcohol, arsenic, and lead

on one hand; and by the poisons associated with certain specific diseases on the other, such as diphtheria, tuberculosis, variola, and others of the exanthemata. See NEURITIS, MULTIPLE; ALCOHOLISM; DIPHTHERIA; and LEAD-POISONING.

PARAMENIA (παρά, beside, amiss; and μήν, a month).—A term for irregular menses. See MENSES OR MENSTRUATION, Disorders of.

PARAMYOCLONUS MULTIPLEX (παρά, indicating on one side; μῦς, μύς, muscle; and κλόνος, commotion).

DEFINITION.—An affection, occurring in adults, which is characterised by clonic spasm in parts of the muscles of the trunk and limbs, more or less symmetrical, but varying in degree. It was first well described by Friedreich, who proposed for it this designation. Among the cases which have been since observed, there have been many variations from the type of the original case, and the malady is not well defined.

ÆTIOLOGY.—Little is known of the causes of the affection, beyond the general facts that it occurs chiefly in men, and may begin at any age between puberty and sixty years. It does not appear to be hereditary. The onset has generally been spontaneous without distinct causation; in a few cases it has followed some apparent exciting cause, such as fright, rheumatism, or malarial fever, but such cases have been too rare to suggest more than a general disturbing influence on the nervous system.

SYMPTOMS.—There are sudden spasmodic contractions of the muscles, or more commonly of parts of the muscles, causing irregular movements of the surface, but seldom of the parts to which the muscles are attached. The contractions quickly pass from one part to another. Their frequency has varied from ten to fifty per minute; but they are irregular in time, a series of quick contractions being followed by longer intervals. In some cases tonic spasm has occurred from time to time. On the other hand, very slight, and even fibrillary, contractions have characterised some cases, which have been termed 'myokymia.' They resemble those which accompany progressive muscular atrophy, of which there have generally been indications, and there is reason to regard the condition as only an excessive manifestation of the 'fibrillation' common in the early stages of that disease. In paramyoclonus the contractions are usually in the muscles of the trunk as well as in the limbs; the diaphragm is occasionally involved, causing a peculiar sound, something like hiccough. Voluntary movement lessens the spasm, and occasionally arrests it; it is also diminished by alcohol and by mental excitement. In a few cases the contractions have continued during sleep. They have been so violent in some atypical cases (possibly allied to the usual form) that the energetic spasm has thrown the patient from the chair on which he was sitting. As a rule, there have been no other symptoms in either the nervous or general system beyond hypochondriacal self-attention, which is often conspicuous and troublesome. After lasting for several months, or even for a year, the spasmodic contractions have, in some cases, gradually lessened, but the affection is so chronic that the ultimate result is often unknown. See CHOREA, ELECTRIC.

Nothing is known of the pathology of the affection. Such clonic spasm characterises the 'electric

cal chorea,' which occurs in Italy, and is probably the result of a toxic influence, possibly malarial. But this is a fatal disease, and is thus sharply differentiated from paramyoclonus. There are, however, other choreic affections characterised by clonic spasm, the nature of which is still undefined, and the severer forms of myoclonus seem allied to these. It is supposed that the clonic contractions originate in the spinal cord.

TREATMENT.—It is not clear that any treatment has modified the course of the malady, which has had a definite tendency, in most cases, either to lessen or to persist. But it has been thought that benefit has been afforded by giving bromide of potassium or hyoscine as a sedative, together with quinine, strychnine, and other nerve tonics. Voltaic electricity has been said to do good in a few instances, a current as strong as the patient can bear being passed from the spine to the affected muscles for a quarter of an hour each day. In very severe cases the hypodermic injection of morphine has been useful. The malady is so rare that therapeutical facts can only be very slowly accumulated.

W. R. GOWERS.

PARAMYOTONE (Paramyotonia; *παρά*, one side; *μῦς*, *μυός*, muscle; *τόνος*, stretching).—This name is applied to a condition characterised by muscular spasm. It is thus applicable to Thomsen's disease, a common designation of this condition being *myotonia* (see THOMSEN'S DISEASE). Two varieties of paramyotone have been described—the *congenital* by Eulenborg, and the *ataxic* by Gowers. In the former, several members of the same family were affected, and in some of these the symptoms were present soon after birth. The malady was characterised by transitory tonic spasm, easily excited by cold, and usually dispelled by warmth. The facial muscles were especially liable, and the rigidity was succeeded by weakness. There was no change in the electrical reactions, except that the irritability seemed to be lowered. In the condition of *ataxic* paramyotone, described by Gowers, there was persistent tonic spasm associated with ataxy. There was weakness both of arms and legs, but the inco-ordination was greatest in the arms. There was also impaired sensibility, distinct for touch and pain, slight for temperature, on the hands and feet, especially the palms and soles. No sign of myotatic irritability could be elicited, but the rigidity was such as to account by itself for this absence. There was no change in the electrical irritability of the muscles.

JAMES TAYLOR.

PARAPHIMOSIS (*παρά*, beside; and *φίμω*, I confine).—**SYNON.**: Fr. and Ger. *Paraphimosis*.—A morbid condition of the penis, in which the prepuce, having been drawn or forced back behind the glans, cannot be returned, and thus gives rise to a condition of strangulation of the parts in front of it. See PENIS, Diseases of.

PARAPLEGIA (*παρά*, at the side; and *πλῆσσω*, I strike).—Paralysis of the lower extremities, usually associated with paralysis of the lower part of the trunk, bladder, and rectum. See PARALYSIS; and SPINAL CORD, Diseases of.

PARAPLEGIA, ATAXIC.—The name 'ataxic paraplegia' has been given to a condition in which a lateral and a posterior sclerosis of the spinal cord co-exist. There is undoubtedly a tendency in some

cases for sclerosis to occur simultaneously in these two columns. On the other hand, such a combination with analogous symptoms may present itself in disseminated sclerosis of spinal origin, and the writer has known many cases diagnosed as 'ataxic paraplegia,' which subsequently proved to be unmistakable cases of disseminated sclerosis; it may occur also as an extension of a primary lateral, or even of a primary posterior sclerosis; whilst something very similar presents itself in Friedreich's ataxia (or 'hereditary ataxic paraplegia,' as it is also termed). More recent observations again have shown that in the remarkable affection known as 'combined sclerosis' or 'combined degeneration of the spinal cord' (see p. 325), we also have the combination of widespread morbid changes in the lateral and posterior columns. Bearing in mind, therefore, these various affections and the symptoms to which they give rise, it seems questionable whether any adequate advantage from a clinical point of view can result from the description of a separate morbid condition under the head of 'ataxic paraplegia.' It may be sufficient for us to recognise that, under the various conditions above referred to, we are liable to get a grouping of symptoms indicative of co-existing disease in the lateral and in the posterior columns of the spinal cord. See SPINAL CORD, Special Diseases of.

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PARAPLEGIA, INTERMITTENT.—See NEURITIS, MULTIPLE; and PARALYSIS, PERIODIC.

PARASITES (*παρά*, upon; and *σιτέω*, I feed).—**SYNON.**: Fr. *Parasites*; Ger. *Parasiten*.

DEFINITION.—This term, in its most extended sense, is applied to those organisms which derive their nourishment wholly or in part from other living beings. See BACTERIA; ENTOMOLOGICAL DISEASE; MYCOSES; STREPTOTHRIX; TINÆÆ; and the various diseases due to animal or vegetable parasites.

PARENCHYMATOUS (*παρά*, beside; and *ἐγχέω*, I pour in).—The word *parenchyma* was formerly used to designate the connective tissue of the several viscera; but it is now applied to the protoplasm, or active elements, of a tissue or organ; and morbid processes affecting the characteristic tissue of an organ are hence called *parenchymatous*. See DEGENERATION.

PARENCHYMATOUS HÆMATOCELE.—See HÆMATOCELE, PARENCHYMATOUS.

PARESIS (*παρήμι*, I relax).—A slight or imperfect paralysis of motion. See PARALYSIS.

PARKINSON'S DISEASE.—A synonym for paralysis agitans.

PARONYCHIA (*παρά*, beside; and *ὄνυξ*, the nail). Inflammation in close proximity to a nail. A synonym for whitlow. See NAILS, Diseases of; and WHITLOW.

PAROSMIA.—See OLFACTORY SENSE.

PAROTID GLANDS, Diseases of.—See MUMPS; and SALIVARY GLANDS, Diseases of.

PASO ROBLES HOT SPRINGS, San Luis Obispo County, California, U.S.A.—Muried saline sulphur-springs. See MINERAL WATERS.

PASSIVE.—This epithet is used to express inertia. See **PASSIVE CONGESTION**, p. 301; and **PASSIVE MOVEMENTS**, p. 962.

PASTEUR-TREATMENT.—See p. 698.

PATENT FORAMEN OVALE.—See p. 657.

PATHOGENIC (πάθος, disease; and γεννώ, I give rise to).—Disease-producing. A term applied to any active cause of disease.

PATHOGNOMONIC (πάθος, disease; and γινώσκω, I recognise).—This word is associated with those symptoms and signs which are specially characteristic of a disease, and the presence of which renders its diagnosis certain.

PATHOLOGY (πάθος, disease; and λόγος, a discourse).—Pathology is the science of disease. Since there is no distinct limit between health and disease, the boundaries of this science cannot be definitely fixed. Further, the nature and character of normal or physiological vital processes are at present very imperfectly ascertained; it is therefore impossible to define those departures from the normal standard which constitute the field of pathology. As in the study of normal life it is convenient to deal separately with the structure of the body (anatomy and histology) and with its functions (physiology), so in considering diseased conditions the subject naturally falls under the two heads of Morbid Anatomy and Morbid Physiology. The term 'Pathology' is occasionally confined to this latter branch alone; at other times it is used in a still more restricted sense, as the equivalent of a portion only of this subdivision, namely, the study of the modes of action of morbid agencies. Thus, in many works on medicine, the causation (ætiology) of a disease, its morbid anatomy or underlying structural derangement, and its clinical features or phenomena of disturbed vital activity are dealt with apart from its pathology—this last term implying the mode of action of the exciting cause of the disease and the unseen effects produced by it, which have to be inferred from the phenomena manifested to one observing the sick person from without. The term 'Pathogeny' has been coined to replace this restricted sense of the term 'Pathology.'

PAU, in the Basses Pyrénées, France.—A mild, calm, sedative winter climate. Mean temperature, 42° F. Absence of cold winds; soil, gravel. See **CLIMATE**, Treatment of Disease by.

PECTORILOQUY (pectore, from the chest; and loquor, I speak).—A physical sign, connected with vocal resonance, heard on auscultation in some limited parts of the chest. The sounds of the voice in pectoriloquy are directly conducted to the ear, so that the words spoken by the patient may be distinctly recognised by the observer, as if proceeding from within the chest. See **PHYSICAL EXAMINATION**.

PECTORILOQUY, WHISPERING.—A form of pectoriloquy in which the whispered voice is heard distinctly. See **PHYSICAL EXAMINATION**.

PEDICULUS.—Three species of lice are parasitic on man: (1) *Pediculus capitis*; (2) *Pediculus vestimenti vel corporis*; and (3) *Pediculus pubis*.

1. **Pediculus capitis.**—This species of pediculus infests the head, especially the occiput; and deposits its eggs on the shaft of the hair, usually not far from the root. The ovum is a small oval, semi-transparent body, somewhat cupped at its free extremity, and very firmly attached by a short pedicle to the hair. The young are hatched in about five days. The louse when full-grown is about 2.5 mm. in length, the female being larger than the male. The head, thorax, and abdomen, which is oval, are distinct. The head is furnished with two short antennæ, and large, black, prominent eyes. Springing from the thorax are six well-developed legs, armed with strong claws, with which the animal grasps the hair. On the back of the male is seen a conspicuous, elongated, conical organ, the penis. The posterior end of the female is notched. The animal is of a semi-transparent, dirty-white colour, and is covered with short, scattered hairs.

2. **Pediculus vestimenti.**—This species closely resembles in shape and general appearance the *Pediculus capitis*, but is of larger size. It infests the underclothing, with a preference for that of a woolly kind, and it attacks and irritates the parts of the skin that are covered by clothes. The ova are generally deposited on the wool or fibre of the clothing, but occasionally on the hair of the skin as well, and the young are hatched in about five or six days.

3. **Pediculus pubis.**—This is much smaller and relatively shorter than either of the other species, and the line of separation between abdomen and thorax is less marked. The abdomen is short and rounded, which gives the animal a crab-like shape. Like the other species, it has six legs; of these the four posterior are armed with strong claws for grasping the hair. This louse infests the pubic region, and occasionally the axilla and hairy parts of the body and face, especially the eyelashes. The ova are found firmly attached to the hairs near the roots.

The different species of pediculi do not bite, as they have no jaws; but they pierce the skin or insert their probosces along the ducts opening on the surface, and draw blood by means of a sucking apparatus or *haustellum*, and in this way they derive their sustenance from the human body. Regarded in a pathological aspect, the presence of pediculi is described as a disease under the name of *morbus pedicularis*, or *phthiriasis*. See **PHTHIRIASIS**. ROBERT LIVEING.

PEDILUVIUM.—See **BATHS**.

PELLAGRA (πέλλα, the skin; and ἄγρα, a seizure).—**SYNON.**: *Erythema Pellagrosum*; **Fr.** *Pellagre*; **Ger.** *Pellagra*.—An erythema of the skin, indigenous to hot countries, and common among the peasants in Italy, Spain, and the South of France, which makes its appearance on the parts of the body most exposed to the light, especially the back of the hands, the neck, and the breast. It is accompanied by marked mental symptoms, and is believed to be due to poisonous maize taken as food. See pp. 488 and 554.

PELVIC ABSCESS.—**DEFINITION.**—An abscess situated in the pelvis and generally connected with some uterine or intestinal affection.

ÆTIOLOGY.—Pelvic abscess may result from cellulitis, peritonitis, pyosalpingitis, suppurating

ovaries, hæmatocele, or suppurative disease of the bladder or the intestine: not infrequently many of these are combined, and it is often difficult or impossible to determine its primary origin.

SYMPTOMS AND COURSE.—Pain and increased tenderness, rigors and hectic fever supervening upon the symptoms of inflammation of any of the organs above mentioned, generally point to deep-seated suppuration. As it approaches the surface the skin or vaginal mucous membrane may become oedematous.

An abscess in the pelvis conforms to the same laws as abscesses in other parts, its extension depending upon the relative firmness and tension of the surrounding tissues; it burrows in the direction of least resistance, and may open in any of the following positions: (1) Through the abdominal walls; (2) into the pelvic viscera; (3) through the floor of the pelvis; (4) through the pelvic foramina; (5) into the lumbar region; or (6) into the general peritoneal cavity. No doubt its starting-point has much to do with its subsequent course, which depends chiefly on anatomical conditions. Should an abscess open into the peritoneal cavity, which it rarely does, peritonitis ensues, and the primary disease may be lost to view owing to the gravity of the secondary lesion. Should it open into the bowel, there occurs a discharge of pus and fæces from the bowel, often of a most foetid character, especially if there be entry of gas into the abscess. An opening into the bladder may cause a long-continued cystitis.

TREATMENT.—Pelvic abscess must be treated as deep-seated abscesses in other parts of the body—in the commencement by hot fomentations or poultices, but when matter has formed it should be evacuated as soon as possible. In many cases an abscess can be punctured or incised through the vagina, and the cavity washed out with some antiseptic solution; and the low mortality and freedom from the dangers and risks inseparable from major operations should secure the employment of these methods in every suitable case. But in the majority of cases of pelvic suppuration more radical measures are required, and incision may be made either from the vagina or by the abdominal walls. The uterus and appendages may be removed or not according to the circumstances met with in different cases. The operations have been classified as follows by Sænger:—

1. *Vaginal Operations.*—(a) Anterior colpocœliotomy; (b) posterior colpocœliotomy; (c) anterior and posterior, combined with uni- or bilateral salpingo-oophorectomy; (d) colpohysterectomy; (e) colpo-hystero-salpingo-oophorectomy.
2. *Abdominal Operations.*—(a) Uni- or bilateral cœlio-salpingotomy; (b) cœlio-salpingo-oophorohysterectomy; (c) bilateral cœlio-salpingo-oophorectomy, with supra-vaginal hysterectomy.
3. *Abdomino-vaginal hystero-salpingo-oophorectomy.*
4. *Sacral or parasacral celiotomy.*

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PELVIC CELLULITIS.—**SYNON.** Parametritis.
DEFINITION.—An inflammation of the areolar tissue surrounding the pelvic organs both in the male and female, but much more frequently in the latter, and therefore more especially of the areolar tissue in connection with the uterus and its appendages.

ÆTIOLOGY.—Pelvic cellulitis is never idiopathic,

but always results from the invasion of the tissues by micro-organisms, most frequently streptococci, more rarely staphylococci, colon-bacilli, gonococci, and the bacilli of influenza, and diphtheria. In the great majority of cases these enter by a wound, though the gonococcus may directly penetrate the mucous membrane and invade the cellular tissue (Wertheim).

At least two-thirds of the cases are the result of childbirth. Next to this gynæcological manipulations furnish the largest number of cases; such, for example, as rough and unskilful examination with dirty fingers and uncut nails; explorations of the uterus, especially when sloughing cancer or myomata are encountered; the passage of a sound or dilators, especially sponge and sea-tangle tents without sufficient aseptic precautions; neglected pessaries, especially if dirty, rough, and badly fitting, intra-uterine stems, and vaginal tampons; and operations, either vaginal or intra-peritoneal, where the cellular tissue is exposed and infected.

It has been attributed to excessive venery, masturbation, sponges and other materials employed to prevent conception, and articles introduced for onanistic purposes. Cold is not often a cause of cellulitis, but by depressing vitality it might cause an aggravation of a parametritis previously overlooked. It has also resulted from suppuration or sloughing of myomata, hæmatomata, and hydatid or dermoid cysts; from extension of inflammation from the rectum, bladder or appendix; or from the bones, periosteum, or joints.

PATHOLOGICAL ANATOMY.—Pelvic cellulitis may be a part of a general systemic infection, or it may be localised as a parametric phlegmon. The terms malignant and benign, employed by Schroeder, would suggest that these two forms are essentially distinct; but the difference is only one of degree and not of kind, and depends upon the virulence of the poison and the resisting power of the tissues.

Parametric phlegmon commences with œdema of the cellular tissue followed by a small-celled infiltration. At first it is soft, but as the fluid is absorbed it becomes hard. It usually begins at one side of the cervix, but soon completely surrounds it, and spreads outwards into the broad ligament, separating its layers, and generally causing a bulging of its posterior aspect; though occasionally, when the lesion from which it originates is situated in the anterior part of the cervix, the anterior layer is bulged forwards. It may extend outwards to the pelvic wall, forwards to the obturator foramen, or backwards to the sciatic notch. In some cases it extends into the iliac fossa, and up to the crest of the ilium. It may cause thrombosis of the pelvic veins, and œdema of the lower extremities (*phlegmasia alba dolens*). In other cases it extends over or under Poupart's ligament to the cellular tissue of the thigh. If it extends forwards towards the bladder it may cause a paracystitis, though this is more frequently the result of suppuration or ulceration of the bladder; it may involve the cavum Retzii, and the subperitoneal tissue of the anterior abdominal wall. Sometimes it descends along the vagina (*paracolpitis*); at others it extends backwards, involving the folds of Douglas surrounding the rectum (*paraproctitis*), and extends upwards beneath the peritoneum of the posterior pelvic wall. Should suppuration occur it may form a single abscess or a diffuse purulent infiltration (*éponge purulente*). It most frequently points above

Poupart's ligament. Sometimes it passes backwards, causing extensive burrowing, and sometimes pointing at or above the iliac crest. Sometimes it escapes from the pelvis by the sciatic notch, and opens in the gluteal region. Occasionally it follows the femoral vessels into Scarpa's triangle. In others it opens into the rectum, bladder or vagina.

In chronic or cirrhotic cellulitis bands of cicatricial tissue are found, most frequently at the bases of the broad ligaments, and generally in association with deep cervical lacerations; or cordlike bands radiating outwards, upwards, forwards or backwards in the folds of Douglas (*parametritis posterior*), causing displacement and fixation of the neighbouring organs. Chronic circumscribed cellulitis is generally the result of ulceration of the bladder or rectum.

Chronic cellulitis sometimes leads to complete atrophy of the cellular tissue of the broad ligaments (*parametritis atrophicans*).

SYMPTOMS AND COURSE.—Clinically cellulitis may be divided into: (1) *Acute inflammation*, including (a) malignant form, (b) parametric phlegmon; (2) *Chronic inflammation*, which may be either (a) circumscribed, or (b) diffuse.

The malignant form is a part of acute lymphatic septicæmia (see PUERPERAL FEVER). A parametric phlegmon may in some cases give rise to very slight symptoms, perhaps merely a sense of uneasiness in the lower part of the abdomen, and may easily be overlooked. Usually, however, it commences with a sense of chilliness, seldom a pronounced rigor, followed by fever of a remittent character. The temperature rises rapidly, often reaching 102°–104° F. the first evening, falling a degree or more the following morning, and rising again at night. The pulse corresponds to the temperature, beating from 90–100 per minute with a temperature of 102° F. The patient complains of dull aching pains in the pelvis, thirst and languor. Along with these there may be obstinate constipation and pain on defecation. The urine is scanty, and there is often frequent micturition, but seldom retention of urine. The thigh of the corresponding side is often flexed. In the early stage there being only an effusion of fluid its detection is difficult; but as the phlegmon becomes harder it is easily palpated, and we become aware of a dense mass usually to one or other side of the uterus, but if large entirely surrounding the organ, which is pushed to the opposite side or firmly embedded in it. Favourable cases may recover in a few days, but generally go on for weeks before absorption takes place. Prolonged fever generally points to suppuration, but not invariably, and it may occur after an apyrexial interval attended by shivering high fever and other symptoms of the initial stage.

DIAGNOSIS.—Cellulitis has been mistaken for peritonitis, tubal disease, intra-ligamentous tumours, such as dermoids, sarcomata, hydatid cysts, intra-peritoneal hæmatomata, and malignant infiltration of the cellular tissue.

PROGNOSIS.—The prognosis in cases of ordinary phlegmon is good even when it is extensive, and when suppuration has occurred; in the malignant form, however, it is bad; and there are many cases in which it is doubtful, for example where pyæmia has developed from a secondary infection through the veins, and where an abscess having burst into the rectum its contents become putrid. Chronic

cellulitis is not dangerous to life, but the patient as a rule continues to be an invalid.

TREATMENT.—*Prophylactic* measures include careful asepsis during delivery, and in all gynaecological manipulations; and the immediate suture of cervical lacerations.

Curative.—An ice-bag applied to the hypogastrium is useful, but care must be taken not to freeze the integuments; others prefer hot fomentations or poultices. Abstraction of blood by leeching the groins, or puncturing the cervix, is not so often employed at the present time as formerly. The bowels should be freed by enemata or mild aperients, such as castor oil, calomel, or magnesia. Opium should be used with caution, but alcohol should be freely administered as in all septic diseases. The strength should at the same time be supported by easily assimilable nourishment. To promote absorption in the later stages tonics, such as iron and quinine, are advisable; also iodide of potassium. Moist applications to the abdomen in the form of a wet pack are useful, and copious injections of hot water; glycerine or ichthyol-glycerine plugs are also advisable. Where absorption has been long delayed good results have been reported from massage. Pus should be evacuated as soon as possible after detection.

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PELVIC HÆMORRHAGES.—Hæmorrhages in and around the pelvis may be tabulated as follows:

1. *Hæmorrhages into Viscera*: (a) hæmatocolpos; (b) hæmatometra; (c) hæmatosalpinx; (d) hæmorrhage into the ovary (follicular apoplexy).

2. *Hæmorrhage into Connective Tissue*: (a) vulvar hæmatoma; (b) hæmatoma of the broad ligament.

3. *Hæmorrhage into the Pelvic Peritoneal Cavity (hæmatocele)*: (a) diffuse hæmatocele; (b) encysted hæmatocele.

1. **Intra-visceral Hæmorrhages.**—**ÆTIOLOGY.**—(a) *Hæmatocolpos*.—When the hymen is congenitally imperforate and the ovaries and uterus normal, menstruation occurs at a somewhat postponed puberty, and the resulting hæmorrhage is pent up in the vagina. Each month this accumulation increases and becomes viscid and discoloured owing to the more watery elements becoming reabsorbed and mixed with mucus.

(b) *Hæmatometra*.—In long-standing cases of hæmatocolpos, first the cervix and then the uterus may become gradually distended with altered blood; but, as a rule, hæmatometra is produced when the vagina is blocked at the uterine end by a membranous or solid partition, or when the vagina is more or less completely absent. In such cases it is associated with distension of only the uterine end of the vagina. Hæmatometra alone is usually the result of acquired cicatricial stenosis of the cervix, or of congenital atresia of the external os uteri; and this latter is sometimes observed in a single horn of a double uterus.

(c) *Hæmatosalpinx*.—Hæmorrhage into a Fallopian tube may be secondary to hæmatometra of acquired or congenital formation. It may also be the result either of hæmorrhage into the decidua of a tubal gestation, in which case it leads either to the formation of a mole or to rupture of the tube; or of tubal menstruation with a closed uterine end.

(d) *Hæmorrhage into the Ovary (Ovarian or follicular apoplexy)*.—During the process of matura-

ion of the ovum the vascularity of the Graafian follicle is increased. This may occasionally lead to more hæmorrhage than usually occurs into the follicle either before or after the escape of the ovum, causing a much greater bulging than is usually due to a corpus luteum.

SYMPTOMS AND PHYSICAL SIGNS.—In cases of retained menstruation of congenital origin the girl will, after puberty, complain of primary amenorrhœa associated with periodic pelvic pain. On abdominal examination a hypogastric tumour may be felt, which is either a small uterus perched upon a distended and elongated vagina, or a uterus also distended with blood. On a vulvar examination being made, the hymen, or a membrane immediately adjacent to it, is seen to bulge and to become tense during forced expiration. If the membrane is thin, the dark colour of the retained blood can be observed. Examination *per rectum* will prove this fluid to be in the vagina. Sometimes one half of a double vagina and uterus is thus affected. If the uterus, or the uterus and only the upper end of the vagina are distended, a bulging *cul-de-sac* will be found *per vaginam*, and the distended portion can plainly be felt by rectal examination.

TREATMENT.—The treatment of all such collections of retained menstrual blood has to be carried out with scrupulous attention to antiseptics and avoidance of all haste or force. In cases of uncomplicated hæmatocolpos a crucial incision should be made into the bulging hymen, and the viscid fluid allowed to slowly empty itself for some hours afterwards into antiseptic gauze. The same rule should be adopted in cases of combined hæmatocolpos and hæmatometra. The greatest possible care should be taken to avoid either sudden emptying of the passages by hot or cold douches or by supra-pubic pressure, lest the uterus should be stimulated to contract suddenly and the blood should regurgitate into the peritoneal cavity. This is especially dangerous in cases of hæmatosalpinx. Absolute rest in bed for ten days in all such cases is essential. In cases of uncomplicated hæmatometra the obstruction must be dissected through and the retained fluid allowed to slowly emerge along a thin gauze drain. Subsequently, a tube may have to be worn to keep the part patent. Such uterine and high vaginal obstructions are very difficult to keep permanently open, chiefly because most of the patients with such conditions are not endowed with much mental aptitude for carrying out the requisite measures.

2. Hæmorrhages into Connective Tissue.

(a) *Vulvar Hæmatoma.*—Occasionally a pudendal vein gives way during the second stage of labour or as a result of a blow, and if the skin remains intact a vulvar hæmatoma results. Such an accident occurring during labour is commonest in primiparæ, because in multiparæ the veins are more varicose and superficial, and tend to burst externally. If a large hæmatoma is formed, obstruction to the presenting part may result.

SYMPTOMS.—The usual symptom is severe pain in one labium majus, felt suddenly during a 'pain,' in the later part of the second stage, and not infrequently 'shock' results. On examination, the swelling of the labium is found to be irreducible, dark in colour, bulging over the labium of the other side, tense and fluctuating, but gradually getting less elastic as the blood coagulates, and finally

getting boggy from œdema round the effusions. There is no impulse on coughing. It is distinguished by its history and by its physical signs from a distended Bartholini's gland, labial abscess, hernia, or varicose veins.

TREATMENT.—If there is definite, though slight, obstruction and the head presents, delivery should be accomplished with forceps. If the obstruction is such that forcible delivery would bruise or tear the swollen parts, the tumour, after the vulva has been shaved, must be incised on the cutaneous aspect, along the long axis of the labium, the clot turned out, and pressure applied till the child is born, any bleeding point being tied. If the wound can be made quite clean and free from any adhering clot, a few buried purse-string sutures will approximate the surfaces, and an attempt may be made by outside pressure to promote primary union. As a rule such cavities do not thus heal, and may be packed with gauze, and allowed to granulate up. If the hæmatoma form apart from pregnancy, rest in bed with moderate pressure will ensure absorption in two or three weeks.

(b) *Hæmatoma of the Broad Ligament.*—Hæmatoma of the broad ligament is more common than is generally thought, and one of its causes is the downward rupture of a tubal gestation, a rupture generally about the middle third of the floor of the tube. The blood is collected between the layers of the meso-salpinx—an advantageous position because considerable resistance is offered by the connective tissue of the broad ligament, and by the broad ligament itself, and the hæmorrhage is thereby generally localised. The blood may become absorbed and the pregnancy (tubo-ligamentary) continue. Another cause of the hæmorrhage is the rupture of a vein from parovarian varicocele, leading to an extravasation of blood which may be sufficiently large to produce syncope. Blows, or sexual excesses, or straining at stool or during labour, may be the exciting causes. These hæmatomata are also a common result of faulty ligation of the broad ligament after enucleation of a tumour. They are generally unilateral. Cases are occasionally seen where the hæmorrhage has spread along the back of the uterus, stripping off the peritoneum, and extending to the other side, thus simulating an intra-peritoneal hæmatocœle.

SYMPTOMS.—There is often a history of some strain, though in the case of tubal gestation the rupture may take place spontaneously owing to continued growth and thinning of the fetal coverings. There is definite shock, coming on somewhat gradually, although fairly sudden as compared with anything like the symptoms of an inflammatory attack. Pain soon follows, due to the separation and stripping off of the coverings of the broad ligament. With that follows pressure—pressure on the uterus, and indirectly on the bladder and the rectum. If the hæmorrhage is at all extensive the anæmia is well marked, but rarely is there any pyrexia, which is important in differentiating it from broad-ligament phlegmon, in which a rigor and pyrexia occur with pelvic pain.

Per vaginam there is a distinct tumour, generally rounded and at first not hard, but rapidly becoming so. The uterus is completely dislocated over to the other side, and usually fixed. The fixation of the uterus depends upon the position of the hæmorrhage. If a pregnant tube, for instance, ruptures in its middle third, there is not necessarily

a sufficient amount of hæmorrhage to reach and fix the uterus. Usually there is no exudation in Douglas's pouch, and the fact that there is a lateral tumour, and that Douglas's pouch is empty, with these symptoms of internal hæmorrhage, shows that it is not an intraperitoneal case.

PROGNOSIS.—The prognosis in these hæmatomata is generally excellent. The hæmorrhage is confined by the broad ligament: it soon ceases, and in a few weeks is completely absorbed, leaving only a patch of induration.

TREATMENT.—Recovery soon follows if the patient is kept in bed and the bowels open. If absorption, however, does not occur, it is generally for one of two reasons. Either the diffused blood has become infected from contiguity of bowel, and suppuration has occurred; or, if the hæmatoma is due to the rupture of a tubal gestation, the fœtus has remained in organic union with the placenta by means of its cord and may go on growing as a tubo-ligamentary gestation (*see* PREGNANCY, Diseases of). If suppuration occur, a small incision or puncture should be made into the swelling *per vaginam* just behind and to one side of the cervix, and the wound should be enlarged by sinus-forceps to avoid injuring the ureter or uterine vessels. The degenerated blood can then be washed out by iodised water, and the cavity be treated as an abscess, and drained carefully.

3. Hæmorrhages into the Peritoneal Cavity.—*Pelvic Hæmatocoele.*—The hæmorrhage here is intra-peritoneal.

ÆTIOLOGY.—The most frequent cause is tubal pregnancy, sometimes owing to rupture of the tube, but more commonly to hæmorrhage from the fimbriated end of the tube due to the presence inside it of a partially detached hæmorrhagic mole (missed tubal abortion). Tubal abortion may also occur, but if complete the hæmorrhage usually ceases. If a mole remains in the tube, blood may continue to trickle steadily from the fimbriated end of the tube into the peritoneal cavity. Rupture of an 'interstitial gestation' is also a cause of hæmatocoele, which is usually diffuse and rapidly fatal. Rupture of the uterus during the second stage of labour, or indeed of any abdominal vessel or viscus, may cause pelvic hæmatocoele by gravitation of blood. Hæmatosalpinx (*see* p. 1170), hæmorrhage from a ruptured ovarian cyst after torsion of its pedicle, varicocoele of the broad ligament or separation of vascular peritoneal adhesions are other less likely causes.

COURSE AND PROGRESS.—The extravasated blood may be diffused or encysted. In the *encysted* form, the hæmorrhage may from the first be slight and gradual, or it may be arrested after a short time. This would allow adhesions to be formed by lymph being thrown out round the periphery of the partially coagulated blood, and if no immediate or great strain were put upon these adhesions by a fresh outburst of hæmorrhage, the blood would become 'encysted.' Limiting adhesions are formed by the exudation of lymph and by consolidation of the outer layers of the clotting blood, glueing coils of small intestine to the fundus uteri and the mesosalpinx. Sometimes after the escape of a tubal mole, the blood and mole gravitate behind the mesosalpinx, which is dragged down by the weight of the distended tube and forms a hood over the collection of blood, and constitutes a variety of 'encystment' which, from its lateral position, is

easily mistaken for an intra-ligamentary hæmatoma. If oozing of blood into this space continues, the wall of the pseudo-cyst may give way, or the adhesions which roof over the space may stretch and give more room, or if the upper adhesions are strong Douglas's pouch may be depressed and the blood may burrow deeply between the rectum and the vagina. A hæmatocoele may also become encysted from pre-existing adhesions shutting off the pelvis from the general peritoneal cavity, as a result of antecedent perimetritis. If bleeding ceases, the cyst-wall organises and may become a definite capsule detached from surrounding viscera.

Diffused hæmatocoele is more frequently the result of a tubal rupture than of a tubal abortion or missed abortion or other source, because the loss of blood is so severe, so sudden, and so persistent, that no opportunity for its becoming localised is afforded. The diffuse form is almost invariably fatal if not treated promptly by abdominal section.

SYMPTOMS.—As a result of the visceral injury or rupture, which has led to the hæmorrhage, there is some shock, but the prominent symptoms in these cases are those caused by internal hæmorrhage. These need not be here enumerated, but are, of course, most marked in the cases of a diffuse hæmatocoele. Pain is usually a prominent symptom, and may be very severe. The mental faculties are clear. Metrorrhagia is usually present.

PHYSICAL SIGNS.—In the *diffuse* form there is some abdominal distension, but as a rule no physical evidence of the hæmorrhage is discoverable either by the abdomen or *per vaginam* except a patchy dullness here and there, not confined to the localities usually dull in ascites. The diagnosis has to be made almost entirely by the symptoms of internal hæmorrhage, by the history, which is often extremely vague, and by exclusion of other sources and positions of the blood extravasated.

In the *encysted* form the physical signs are more definite. The blood is collected so as to form at first an elastic mass, which in twenty-four or forty-eight hours gets harder, filling perhaps both posterior quarters of the pelvis and Douglas's pouch, unless limited to one side by adhesions. It is usually roofed over by intestines and omentum and mesosalpinx, and is thus confined primarily to the true pelvis; gradually if hæmorrhage persist or recur it extends upwards and may reach the umbilicus with its upper surface arched like the diaphragm. It exerts pressure upon the uterus and broad ligament in a direction upwards and forwards, upon Douglas's pouch downwards, and upon its intestinal roof upwards, so that the fundus uteri may be elevated and felt above the pubes. If hæmorrhage continue the adhesions may give way, and the hæmatocoele would then become secondarily diffuse, and if it continued and did not again become encysted, it might prove fatal. As a rule, however, the adhesions get denser, the outer layers of the blood become consolidated, and further hæmorrhage is lessened by the resulting pressure, and arrested by thrombosis in the bleeding vessels. Sometimes the temperature rises from the tension present, or fever may accompany the formation of peritoneal adhesions; or more rarely suppuration may ensue by infection from the surrounding intestines.

TREATMENT.—In the *encysted* form of hæmatocoele interference is not called for unless the temperature rises, or the tension greatly increases, or the tumour is definitely enlarging. The writer

has, under such circumstances, operated in all such encysted cases by vaginal incision (posterior colpotomy) followed by gauze-packing, with invariably good results, but cases are recorded where hæmorrhage has recurred, requiring abdominal section to remove the affected tube or seize the bleeding point. In such operations it is rare to find the fœtus or tubal mole among the blood clot, and it is not necessary to try to find and ligate the bleeding point. The hæmorrhage usually ceases at once and does not recur.

In the *diffuse* form abdominal section is absolutely and immediately necessary. If the patient is evidently losing ground, or if there has been a recurrence of hæmorrhage and it is impossible to make out a tumour definitely outlined below in Douglas's pouch and above in the abdomen, it is evident that the blood is not encysted; and in that case it becomes one's duty to operate through the abdomen at once, even if the pulse can only just be felt at the wrist. When the abdomen is opened blood wells out, and fresh hæmorrhage is apt to arise owing to reduction of pressure. The incision should be about three inches long, and no time should be lost in sponging out blood-clot. Feel at once for the fundus uteri, and, presuming the bleeding is tubal, draw the fingers along the two tubes until the lump or irregularity on the one or the other side is felt. Seize this lump and clamp the ligament on each side with Spencer Wells's large forceps, and the hæmorrhage is then entirely under control. The blood-clot, and perhaps the mole or the detached fœtus, can then be removed at leisure, and the patient rallied by a rectal or venous saline injection. Ligatures are then applied on each side of the gestation-sac, which can be removed bodily. This operation, even in apparently moribund cases, has been so often successful that it should be performed as soon as indications are absolute. It is a good plan to have the vein in the arm prepared for a saline injection by another surgeon while the abdominal incision is being made. *See SALINE SOLUTION, Infusion of.* AMAND ROUTH.

PELVIC PERITONITIS.—*SYNON.*: Peri-metritis; Pelveo-peritonitis.

DEFINITION.—An acute or chronic inflammation affecting the pelvic peritoneum.

FREQUENCY.—From the result of 600 autopsies on women Beigle found that 90 per cent. showed evidences of pelvic peritonitis; Bandl estimated its frequency as 66 per cent.

ÆTIOLOGY.—The exciting causes are micro-organisms or their products: especially septic, putrid, gonorrhœal, and tubercular infections. It is almost always secondary to disease in neighbouring organs and tissues. It frequently follows a septic metritis—the result of childbirth, abortion or intra-uterine manipulations—salpingitis and pyosalpinx. In these cases the inflammation may extend to the peritoneum either by continuity through the fimbriated extremity of the tube, by penetrating its wall, or by rupture. It may extend from the ovaries in oophoritis, abscess or tumours, especially dermoids. Pelvic hæmatocele and cellulitis are generally associated with peritonitis; and similarly inflammatory and ulcerative diseases of the bladder or intestine may involve their serous coats; the vermiform appendix especially being a fruitful source of pelvic peritonitis. Primary inflammation, the result of chill during menstruation, has been

described, but its occurrence has not been satisfactorily proved. Peritonitis has followed the injection of irritating fluids, especially a solution of nitrate of silver, into the uterus; probably owing to the escape of some of the fluid through the abdominal ostium of the tube.

PATHOLOGICAL ANATOMY.—The disease commences with injection of the blood-vessels of the peritoneum; the surface loses its lustre and becomes rough and coated with lymph. This is generally but not always followed by a serous exudation which sometimes becomes purulent or putrid. The fluid generally gravitates into Douglas's pouch, where it becomes encapsuled by the agglutination of the intestines above; and as the pouch becomes distended it forms a globular tumour which pushes the uterus forwards against the abdominal wall, the rectum backwards against the sacrum, the posterior fornix of the vagina downwards towards the introitus, and lifts the intestines upwards. In some cases where the pouch of Douglas has been obliterated by previous attacks the fluid may collect in the postero-lateral regions of the pelvis, and in others in the utero-vesical pouch. When the fluid effusion has been absorbed or evacuated, or where no fluid exudation has occurred, as is generally the case where the inflammation is due to a gonorrhœal infection, adhesion of the opposing surfaces takes place. These adhesions are found as broad surfaces, as bands, or merely as thickenings of the peritoneum. They cause fixation of otherwise mobile organs and often cause sterility or chronic invalidism.

SYMPTOMS.—As the inflammation varies immensely both in extent and severity so the symptoms may be almost imperceptible or of a most pronounced and alarming character. When the disease follows childbirth it usually sets in acutely on the second or third day of the puerperium; where it complicates salpingitis or cellulitis it develops more insidiously. It begins with a feeling of chilliness and malaise rapidly increasing to intense pain with marked tenderness. The pain is much more intense where there is a tendency to encapsulation of the effusion, and is therefore relatively a good sign. The bowels are obstinately confined with retention of flatus. There is much tympanites with rigidity of the abdominal muscles. The aspect of the patient is characteristic with sunken eyes, pinched features and anxious expression. The tongue is coated, thirst intense, and anorexia complete; generally there is vomiting and retching, often dysuria or strangury. The temperature rises gradually with slight remissions and in uncomplicated cases seldom exceeds 102° F. The pulse on the contrary rises rapidly to 120–130 or more: it is small and wiry, or in bad cases weak and compressible. The course of the disease varies according to the extent and intensity of the inflammation and the amount of effusion. Resolution is attended by a fall in the temperature, though the pulse usually remains high. Should suppuration occur, however, the fever increases and assumes a hectic character; many of the symptoms of the initial stage return; and the pain and tenderness become more concentrated towards the affected area.

PHYSICAL SIGNS.—At first the chief sign is tenderness when any part related to the peritoneum is disturbed. When effusion has taken place it forms an elastic or fluctuating tumour which

displaces the neighbouring organs in a characteristic manner. Adhesions are diagnosed by touch and by the fixation of the pelvic organs. The uterus for example may be found retroverted and cannot be replaced even when the patient is completely anæsthetised; or if replaced it immediately returns to its abnormal position. Great care must be taken in such manipulations, as injury to neighbouring organs has in some cases led to fatal results; and in no case should the uterine sound be employed. An adherent ovary, whether prolapsed into Douglas's pouch or in its normal position, can be grasped between the fingers with unusual ease and cannot be pushed about as a normal ovary could.

DIAGNOSIS.—Pelvic cellulitis is generally complicated with peritonitis, but the latter occurs very frequently without the former; it is generally possible therefore to distinguish the symptoms of both.

In peritonitis the aspect of the patient, the vomiting, pain and tenderness, are characteristic; the pulse is high and the temperature relatively low. In cellulitis the temperature is higher and the pulse not so rapid; with a temperature of 102° F., for example, a pulse over 120 would point to peritonitis, under 120 to cellulitis. The effusion and absorption of the exudation is more rapid in cellulitis than in peritonitis; in the former the exudation commences to one side of the cervix, surrounds and fixes it; the vaginal mucous membrane is pushed downwards and cannot be moved over the exudation by which it is fixed. In peritonitis the exudation is usually situated much higher in the pelvis. Lateral displacements of the uterus point to cellulitis; ante-position to peritonitis. When the vaginal mucous membrane is pushed down by a peritoneal effusion it is not fixed but can be moved over it. Such an effusion pushes the rectum backwards or to one side, but never surrounds it, and the rectal mucous membrane can be moved over it. In parametritis the rectum is surrounded and narrowed by the exudation, and its mucous membrane firmly attached to it. The relations of the posterior wall of the uterus as felt through the rectum are also important. In cellulitis the tumour never reaches above the line of firm peritoneal attachment, leaving the fundus free; in peritonitis it may reach or go above the fundus.

PROGNOSIS.—The prognosis is generally good, so far as life is concerned, so long as the disease remains localised to the pelvis; but bad as regards complete restoration to health.

TREATMENT.—In the early stage of the inflammation our objects are to favour encapsulation, to relieve pain and to support the patient's strength. With these objects she is kept as quiet as possible in bed; cold is applied to the hypogastrium by frozen compresses frequently renewed, or by Leiter's apparatus; the ice-bag can seldom be borne because of its weight. Opium is given by the mouth or rectum or by hypodermic injection; and fluid nourishment administered. A few leeches applied to the abdomen are often advantageous, but should only be employed in strong and plethoric subjects. Antipyretics are not needed and do harm by disordering the stomach, and purgatives should on no account be resorted to. When the fever has abated the absorption of the exudation should be favoured by warm compresses or poultices.

In the chronic condition efforts are often needed to free the organs from adhesions. Massage (Thure Brandt), breaking down adhesions under an anæsthetic (Schultze), or directly dividing them after celiotomy may be required.

WILLIAM J. SMYLY.

PEMPHIGUS (πέμφιξ, a bladder).—**SYNON.**: Pompholyx; Fr. *Pemphigus*; Ger. *Blasenkrankheit*.—Pemphigus is a disease characterised by the eruption of bullæ on the skin. The term has been employed to indicate almost all the forms of large vesicular or bullous eruptions, but many of these are now differentiated from the group and appear in different categories, especially in the case of dermatitis herpetiformis, and the bullous eruptions due to definite bacterial and nervous influences. There remain certain diseases accompanied by the formation of bullæ, probably of totally distinct origin, but which agree in some of their clinical characters, to which the term 'pemphigus' is specially restricted. Cases of true pemphigus must be reckoned among the rare cutaneous affections.

VARIETIES.—The following are the chief forms still described as pemphigus:—

Pemphigus acutus.—Acute pemphigus occurs less commonly than the chronic variety, but after the exclusion of vesicular and bullous eruptions due to the action of pyogenic and other bacteria, urticaria bullosa and erythema bullosum, there still remain cases which must be regarded as true acute pemphigus. The disease occurs at all ages, but is more common in children than in adults. It commences with symptoms of severe constitutional disturbance—sickness, diarrhoea, and pyrexia. The lesions vary in size from small vesicles to bullæ two or three inches in diameter, and may appear on any part of the skin; the mucous membranes may also be severely affected. The eruption appears in crops, the bullæ, at first full of serum, become turbid rapidly and show a great tendency to rupture. The skin itself becomes covered with scabs composed of the raised epithelium and a certain amount of coagulated serum. The risk of septic infection of the surface under such circumstances is great, and its occurrence has much to do with the causation of the fatal results. Cases are on record in which death has occurred in a few days, although the average duration is from three to six weeks. The prognosis depends greatly upon the amount of epithelium destroyed, as in the case of superficial burns, and is seriously affected by co-existing conditions, the most significant of which is renal disease. The majority of cases are fatal, while others recover as from an acute specific malady. The exact relations of this type of disease are not defined. For purposes of convenience the form of pemphigus arising usually in adults who have to deal with organic material, as in the case of slaughtermen, tanners, and cooks, which is often very fatal, is classified under the term 'acute pemphigus.' In these cases the probability of the inoculation of micro-organisms is very great, and in many of them the pathogenic bacterium known as the diplococcus of Demme has been isolated from the fluid in the vesicles and bullæ, and from the blood.

Pemphigus chronicus (vulgaris).—Chronic pemphigus is the most common variety of the disease and is the type of the affection. The lesions appear as minute vascular points from which rise

small vesicles. The vesicles rapidly increase in size until they reach their maturity, when they appear as bullæ of varying sizes, the majority being from half an inch to an inch in diameter, but large bullæ of three inches in diameter are by no means uncommon. These blisters have a circular or oval outline, but the border may be sinuous owing to the coalescence of two or more neighbouring lesions. The bullæ project abruptly from the surrounding skin, their walls are tense, often forming hemispherical protuberances. The contents at first are clear so that the bullæ are semitranslucent, but gradually they become turbid owing to the presence of leucocytes and the formation of fibrin in the serum. At this time a fine red areola may make its appearance immediately surrounding the bulla. The fluid contents of the bulla become rapidly absorbed, its walls become flaccid, and a thin epidermic scab remains; or if the walls rupture, its contents escape and a scab of coagulated serum and epidermic material results. The loose epidermis or the scab finally falls off, leaving a reddened or excoriated surface, the redness of which gradually gives place to a brownish pigmentation which may last for some time. Any individual lesion may pass through these changes in a few days, but the eruption comes in crops and the attacks are apt to recur, so that the disease deserves the title of 'pemphigus chronicus.'

In the case of children and old persons, the earliest symptoms are those of constitutional disturbances such as moderate pyrexia, shivering, and nausea. Simultaneously numerous small lesions may occur, running their course in a few days, succeeded by a recurrence of constitutional symptoms and of eruption. When the area affected is extensive, much discomfort may arise from the presence of the bullæ. Thus when the lower part of the back is severely affected it is difficult for the patient to assume a comfortable position, and when the excoriations produced by the removal of the epidermis are great, the patient may become greatly prostrated owing to the discomfort, sleeplessness, the effects of septic absorption, &c. A fatal result may ensue.

In the case of healthy adults, on the other hand, it is remarkable how very little disturbance to the general health appears to be caused even when the attack of pemphigus is extensive. A feeling of heat or tingling as the lesions appear, the discomfort and consequent restlessness produced by the mechanical presence of the bullæ and attempts to avoid their rupture along with slight disturbance of appetite and of the general health from confinement may be all the symptoms noted. The mucous membranes are, as a rule, not severely affected. Septic infection of the bullæ, or of the abraded surfaces, gives rise to lymphangitis and lymphadenitis with symptoms of septicæmia. In uncomplicated cases, especially if the kidneys and other organs are healthy, the disease ceases after recurrences which become gradually less and less severe, and complete recovery may occur in a few months.

Pemphigus neonatorum.—This form of disease occurs in newly born children and may be of all degrees of severity. An eruption of small vesicles may be limited to one part of the body, or the eruption may affect the whole surface. The bullæ also may vary in size within wide limits. The course of the disease varies greatly. In slight cases very little disturbance of the general health of the

infant is noted, the degree of pyrexia and the constitutional symptoms as a rule varying directly with the extent of area affected. A few reddened patches remain after the rupture or absorption of the contents of the bullæ, and a slight amount of pigmentation may remain for some time. In cases where the eruption appears over the whole surface of the body, the symptoms as a rule are very severe, and in such cases death may occur in two or three days. The whole surface is red from head to foot owing to denudation of the epidermis, in much the same way as in certain degrees of burning. In some cases, in which secondary infections may play a part, ulceration of the affected areas supervenes, and one of the forms of disease known as *dermatitis gangrenosa infantum* may be produced, leading on to symptoms of septicæmia and pyæmia.

It is noteworthy that in many cases a history can be obtained of some source of septic infection. Thus it is not uncommon to find that there has been slowness of healing or ulceration near the umbilicus, or that a scratch or injury has been noted in some part of the body. Cases are on record in which a child born of a mother suffering from puerperal fever developed pemphigus neonatorum. An instructive example is on record of the simultaneous outbreak of cases of impetigo contagiosa and pemphigus neonatorum among children attended by the same midwife. The clinical as well as the pathological evidence tends to show that in such cases there is direct inoculation of the skin with organisms, such as the *Streptococcus* or *Staphylococcus pyogenes*. Probably many examples of pemphigus occurring in individuals of greater age are of the same character. In such cases bullous eruptions may occur locally about the genitalia, on the trunk, or may be diffused over the surface of the body. In the majority of instances treatment by ordinary antiseptic measures is successful so far as the individual patient is concerned, and attention to disinfection and general hygiene is sufficient to check the epidemics of the disease which sometimes occur.

A form of the disease is mentioned in which the bullæ are flaccid and ill-formed and lead to prolonged desquamation of the type of *exfoliative dermatitis*. The exact nature of such epidemics is not thoroughly ascertained, but the accounts given point to a cause similar to that already suggested in the ordinary variety of pemphigus neonatorum.

Pemphigus contagiosus tropicus.—This is a bullous eruption occurring in very distinct epidemic form in numerous parts of the tropics. The disease may occur on any part of the body, and has many of the characteristics already described in cases of pemphigus neonatorum. It occurs chiefly during the hot weather and is distinctly contagious. The skin is liable to be specially affected where surfaces are in contact. As the bullæ heal, circinate rings of crusted eruption are left for some time, closely resembling the appearances produced in certain cases of impetigo contagiosa (p. 724). Micrococci have been observed in the contents of the bullæ, usually as diplococci or in groups. From the description given of the disease, there can be but little doubt that it is closely allied in ætiology to the form of pemphigus just described as pemphigus neonatorum.

Pemphigus foliaceus.—This disease is one of the forms of dermatitis which becomes universal in distribution, producing general exfoliation. Its

relations to the other varieties of pemphigus are obscure. As a rule the attack commences as an outburst apparently of pemphigus chronicus. After the first onset the typical appearance of the disease is assumed; the bullæ which form are flaccid; their contents lose their translucency and become sero-purulent; their epidermic coverings are detached and expose the underlying excoriated surface, which remains moist and continues to exude a sero-purulent discharge, often of extremely offensive nature. The tendency to the formation of bullæ becomes less and less marked, and the epidermis exfoliates till at length the appearance of general exfoliative dermatitis is closely simulated. The tendency for the altered epidermis to crack and emit sero-purulent fluid and blood remains to a greater or less extent, so that septic absorption from the surface is a constant source of danger. The disease, which may be at first local, tends to become universal, affecting the hands, feet, scalp, and body generally. The nails become distorted, roughened, and may atrophy and disappear, while the hair is either shed or becomes irregular in its growth. The eyelids become inflamed and everted, great discomfort being thereby produced. The mucous membranes of the mouth and throat are affected, the epithelium being thrown off after the appearance of irregular and deformed vesicles. The pyrexia and constitutional disturbance vary greatly. Usually the temperature ranges above the normal with frequent exacerbations of fever. The disease, though almost invariably fatal, is characterised by remissions and exacerbations. During the period of remission the skin may show great improvement and the general condition of the patient improves. Gradually the discomfort and pain, and the effects of septic absorption, undermine and destroy the health of the patient, till at length a condition of severe toxæmia is produced, associated with a low form of delirium, and death occurs from exhaustion. Long before the final result, the difficulties in nursing and feeding the patient become excessive, while the risk of intercurrent disorders of viscera, especially the lungs and kidneys, or of pyæmic accidents is great.

Pemphigus vegetans.—This is one of the rarest of diseases of the skin, but seems to be widely distributed. After a variable period of illness, bullæ, resembling those of pemphigus foliaceus, appear on the hands, feet, axillæ, groins, and the buccal mucous membrane; subsequently other parts of the body become affected. On rupturing, the bullæ do not heal up, but the skin tends to remain raw and even to ulcerate. On the excoriated surfaces granulation-tissue appears in some places producing fungating growths, in others an appearance resembling condylomata. The disease appears to vary greatly in severity, but cases which become widely spread cause most serious debility, and are nearly always fatal within comparatively short periods. In all probability the specific cause of the disease is different from the forms of pemphigus already mentioned.

HISTOLOGY.—The descriptions of the microscopic appearances in the disease vary mainly in the definition of the exact place of origin of the bullæ. According to some observers the collection of fluid makes its appearance at the upper margin of the stratum mucosum, raising above it the stratum corneum, and perhaps a few adherent cells of the stratum mucosum, while others describe the bulla

as commencing in the lowest layers of the rete, raising above it as it collects the whole of the epidermis. No doubt both conditions occur and depend upon the nature and severity of the form of the disease. The serum, at first translucent, gradually contains more and more leucocytes, which pass in from the underlying vessels of the corium, so that the bulla appears turbid and its contents are sero-purulent. A certain amount of fibrin may also be observed in the bulla, frequently containing leucocytes in its meshes; epithelial cells, singly or in groups, may also be present in the vesicle. The changes in the cutis are those of a mild degree of inflammation with congestion of the blood-vessels and widening of the lymph-spaces. The exudation of round cells into the cutis, slight at the commencement, becomes denser as the bulla becomes turbid, the papillæ flattening or swelling owing to the pressure of the fluid from above, and the oedema and infiltration of leucocytes from below. As a rule, the round-cell infiltration becomes absorbed so that very little damage is done to the cutis. Visible cicatrix indeed is rare as the result of uncomplicated pemphigus. In cases of pemphigus foliaceus, and in cases of chronic pemphigus of long duration, changes consisting of condensation of the cutis and atrophy of the epidermis, resembling those found in general exfoliative dermatitis, become evident. The changes in pemphigus vegetans, on the other hand, are completely different in character as the disease affects the mesoblastic structures of the skin rather than those derived from the epiblast.

BACTERIOLOGY.—The bacteriology of the disease has been studied carefully; but the results are not as yet decisive. In some of the cases the contents of the vesicles seem sterile; probably in the majority cocci, resembling the *Staphylococcus epidermidis albus*, may be seen in the contents of the vesicles. In later stages of the disease the white and yellow cocci of suppuration and other organisms are found present. Such results are especially to be expected in the class of cases belonging to the type of pemphigus neonatorum and pemphigus contagiosus tropicus. In certain severe cases of the disease a diplococcus described by Demme has been frequently isolated, and has been suspected of being the principal factor in its causation.

TREATMENT.—Taking pemphigus chronicus as the type of the disease, the treatment during its course should be of a palliative character. Owing to the sensations of heat, pruritus, and irritation characteristic of the first period of this eruption, sedative and astringent lotions containing glycerine of subacetate of lead, calamine, oxide of zinc, lime-water and glycerine are of much service. The use of dusting powders, of starch, oxide of zinc, and siliceous earth, with the addition of boric acid, camphor, menthol, &c., in varying strengths are often of much use on account of the relief to pruritus which they afford. Special care must be taken to protect the bullæ, and to prevent septic inoculation of their contents, or of the denuded surfaces remaining. As a general rule the bullæ should not be opened artificially, but when they occur on situations where accidental mechanical injury is certain, they should be opened with the point of a fine knife, taking precautions against the introduction of pyogenic organisms. The surfaces should be carefully and frequently dressed with antiseptic applications, such as boric-acid ointment, &c. No internal remedy has been definitely shown to have

a specific effect on the treatment of the disease. The drug which has been most widely used is arsenic, and by some this remedy has been vaunted as specific, but it does not occupy this position, and it is doubtful whether it has much beneficial effect. It is certainly capable of producing much harm when given during prolonged periods, and in large doses, as used to be the custom. Benefit may perhaps be expected as the result of administration of internal antiseptic remedies, such as quinine, salicin, &c. With our present knowledge, however, internal treatment consists in taking precautions to sustain the general health by appropriate food and tonics.

In cases of Pemphigus neonatorum, Pemphigus contagiosus, &c., in which the evidence of inoculation of the skin with organisms allied to the ordinary pyogenic organisms is almost complete, the use of stronger antiseptic remedies to the surface is urgently indicated. The rapidity with which bullous eruptions of severe degree of this type are seen to vanish under such treatment and their non-recurrence are strong indications of their etiology. As an external antiseptic the best remedy is mercury. The affected surfaces may be bathed in weak solutions of the perchloride (1-2000 to 1-10000) while the excoriations or bullæ as they collapse are dressed with the diluted ointments of nitrate of mercury or of ammoniated mercury. In the cases where the application of mercury may give rise to danger from absorption of the drug we must content ourselves with the use of less powerful antiseptics. The type of these is boric acid, but many remedies of the organic series, such as carbolic acid, lysol, chinolol, and iodol, have proved very serviceable. In cases of general exfoliation and pemphigus foliaceus, the severity of the disease and its depressing power render it necessary to make still greater exertions to sustain the health of the patient and to protect the epidermis from every form of injury. Careful and repeated dressing of the surface with soothing and antiseptic lotions, ointments and dusting powders must be employed. Good results from the use of warm baths made with gluten, bran, or alkalis, according to the necessities of the case, are frequently obtained. In certain cases the patient finds his only relief from suffering while immersed in such a bath. With due precautions he may remain in the bath for hours at a time, while cases are on record of patients remaining in the bath during periods of weeks, arrangements for retaining a suitable temperature having been made. The risks of an intermittent bath are the chances of chill to which such patients are exceptionally liable, and the injury to the skin owing to the alternations between constant soaking and drying. In cases of pemphigus vegetans the granulomatous character of the lesions demands carefully applied antiseptic treatment, owing not only to the risk of septic contamination, but also to the depression produced by the absorption of septic matter from the surface and the great fetor thence arising.

The care of severe cases of pemphigus, on account of the chronicity of the disease and the serious nature of the symptoms, is a most responsible task, and requires the utmost care and patience on the part of both nurses and medical attendant.

JAMES GALLOWAY.

PENIS, Diseases of.—**SYNON.** : *Maladies de la Vergé* or *du Penis*; *Krankheiten der Ruthe* or *des Penis*.—In this place will be described the

following conditions: (1) congenital abnormalities; (2) phimosis; (3) paraphimosis; (4) adherent prepuce and preputial calculi; (5) inflammation of the penis; (6) gangrene of the penis; (7) herpes preputialis; (8) benign growths; and (9) malignant disease of the penis.

It will be sufficient to mention the occurrence of—double and triple penes (generally no urethra), too large a penis, small genitals, fracture, dislocation, hematoma, and fistula (congenital and inflammatory) of the penis. The remaining diseases of the component parts of the penis will be found in the several articles upon these special subjects, such as diseases of the urethra, chordee, gonorrhœa, gleet, balanoposthitis, syphilis, and priapism.

1. Congenital Abnormalities.—There are two chief abnormalities—(1) hypospadias, and (2) epispadias.

(1) *Hypospadias* is much the more common of the two. It consists in a deficiency in the floor of the urethra. The cause is the failure of union of the two sides of the 'genital groove' which is formed in the mesial line of the genital eminence. It is by the coalescence of these two ridges that the scrotum, floor of the bulbous and penile urethra, the frænum and lower part of glans and prepuce are formed. The membranous and prostatic urethra is formed by the union of the urogenital sinus with the allantois, the urogenital sinus being cut off from the anus by the growth downwards of the transverse fold of the perineum. The extent of the deficiency of the union varies considerably in different cases. (a) There may be a want of development of the frænum only, leaving a very large meatus urinarius and perhaps a large cowl-like prepuce. (b) There may be a complete fissure from the meatus to the scroto-penile junction, or along any part of this distance. (c) There may be a fissure extending the whole length of the urethra from the meatus to the triangular ligament separating the testicles into their respective compartments of scrotum. This is one form of so-called 'hermaphroditism,' the external genitals simulating the female character. Besides these three chief forms there are many curious deformities of the meatus and fossa navicularis which are formed by the depression of the epiblast as a blind pouch, which joins the main urethra before birth. For example, there may be a persistence of this pouch, the real urinary orifice being placed in the position of the frænum, or further back along the inferior surface of the penis; the meatus under such conditions is generally very small. The writer recently saw a case in which there was a double urethral opening at the meatus urinarius. The upper one, an inch long, was blind; the lower one led into a contracted fossa navicularis, and through it urine was passed. Rarer abnormalities are the opening of the urethra into the rectum, or through the perinæum, or through the penile floor at any point, the remainder of the urethra being a solid fibrous cord.

(2) *Epispadias*.—This rarer condition is apparently a deficiency of the dorsum of the penis, in which the floor of the urethra is more or less exposed from the meatus urinarius backwards. The actual condition is really that of hypospadias, the penis being rotated upon itself in its long axis, so that the un-united genital furrow looks upwards, and the corpora cavernosa look downwards; the direction of the rotation, either from right to left or left to right, is often quite obvious at the root of the

penis. There are two chief varieties: (a) partial, in which the deficiency is limited to the penis; (b) complete, in which the deficiency extends backwards and joins with an extroversion of the bladder, the mucous membrane of the bladder being directly continuous through the abdominal wall with the skin-like lining of the genital furrow on the dorsum of the rotated penis. In both hypospadias and epispadias the prepuce is generally large and loose, and often arranged in a fold on either side of the glans and the congenital furrow; there is no properly formed frænum; the floor of the urethra and the surrounding structures are very dense and fibrous, owing to the presence of much scar-tissue in them.

SYMPTOMS.—In slight cases there is only inconvenience in the position and method of passing urine. In the more serious cases the symptoms may be placed under two headings, sexual and urinary. (1) Sexual intercourse is impossible, painful or defective, since the penis is very small or curved in various directions by the bands of scar-tissue; erections are generally painful on this account, and semen is not properly introduced into the vagina owing to the position of the urethral orifice. (2) In bad cases urine is constantly dribbling away, causing soreness, a foul odour, and general disgust. This is the greatest misfortune in complete epispadias. From these causes the general health deteriorates considerably.

TREATMENT.—In slight cases no treatment is necessary, but in the more severe cases the condition of the patient is so terrible that a surgical operation should be performed. The results are not very satisfactory. The operation should be performed after puberty, if it be possible to wait. The wearing of a special apparatus is sometimes useful in alleviating the sufferings of the patient.

2. **Phimosis.**—Phimosis is a constriction of the orifice of the foreskin which limits or prevents its retraction.

There are two varieties, congenital and acquired. (1) *Congenital phimosis* is extremely common. The orifice of the prepuce of the newly born infant is often constricted in varying degree, and may be as small as a pin-point. (2) *Acquired phimosis* comprises three conditions of common occurrence. (a) Inflammatory phimosis, in which swelling of the foreskin (which may have a normal or a slightly constricted orifice) prevents its retraction; (b) that condition in which previous inflammation and ulceration have produced sufficient scar-tissue in the orifice of the foreskin to prevent its retraction; and (c) many cases of warts, cancer, and other diseases in which the implication of the prepuce produces a similar result.

SYMPTOMS.—The presence of phimosis renders cleanliness of the penis an impossibility, as smegma and urine collect underneath the foreskin. The resulting accumulation of filth may produce itching and irritation, and suppurative and ulceration may ensue. From this cause children are continually pulling at the foreskin, which becomes very long. This habit is likely to engender the pernicious habit of masturbation, and is occasionally mistaken for a symptom of stone in the bladder. As a predisposing cause of gonorrhœa and syphilis, phimosis is a most important factor; any discharges which enter the orifice of a phimosis have an excellent situation for developing disease in its most virulent form, since there is deficient drainage, an ample supply of food for the growth

of germs, and a soft delicate mucous surface which is easily damaged. The stream of urine is diminished in size and urination is slowed, and the foreskin is sometimes distended by the outflowing urine. In bad cases there may be retention of urine with dribbling incontinence from the overflow, the patient enduring the greatest suffering and distress. This condition is often accompanied by convulsions in children. Straining in phimosis frequently induces hernia. In a perfect phimosis the glans penis is very small, the frænum very short, and the urethral orifice very small or closed entirely, another meatus existing elsewhere as in hypospadias. Secondary diseases of the bladder and kidney may ensue from back-pressure and infection resulting from phimosis. The irritation of a bad phimosis in children is liable to produce symptoms suggestive of hip-joint disease. Cancer of the penis is very rarely seen in the circumcised.

TREATMENT.—In cases where the constriction is not well marked it is possible by drawing the foreskin back a little more each day to stretch the orifice until it will completely retract. Care should be taken to keep the parts very clean and very dry during these exercises, and to replace the foreskin immediately after retraction, or paraphimosis may ensue. In cases where the constriction is well marked (such being the majority) circumcision should be done at once. In those cases where phimosis is complicated by swelling, discharge, inflammation, ulceration or chances of the glans or foreskin, circumcision should be done immediately and under the strictest antiseptic precautions. If the operation be performed as described below, and if the patient be under the influence of mercury, circumcision may be done for hard chancre, the wound healing well without the development of syphilitic ulceration in the operation-wound.

Circumcision.—If the patient be an infant or a nervous adult a general anæsthetic is necessary, but in an adult patient with no inflammation of the parts, β -eucaine is quite sufficient (see *ANÆSTHESIA, LOCAL*). Fill a hypodermic syringe with boiled solution of β -eucaine containing 2 grs. of the drug, insert the needle in the subcutaneous tissue at the root of the foreskin opposite the root of the frænum, inject a small quantity, then pass the needle in a circular direction around the coronal-preputial junction, injecting a little into the subcutaneous tissues all the way. The foreskin is then pulled on to the needle in the distant semicircle of its extent, so that the needle completes a circle around the root of the foreskin. Wait a short time, then pull the foreskin forwards, clamp its base obliquely from above downwards and forwards, and cut off sufficient skin. Slit up the mucous membrane, remove it with scissors, leaving a short frill at its attachment, the frænal mucous membrane being left long on each side. Now carefully stop every bleeding point by fine catgut ligatures, then sew up by many separate stitches; absolute approximation of the edges should be achieved. The best stitches are fine horse-hair, one end of the knot being left an inch long. If all hemorrhage be stopped and absolute approximation of edges be achieved healing by first intention with no pain is assured. The long black stitches are easily removed on the seventh day. The best dressing for an infant is a narrow strip of butter-cloth covered with eucalyptus-vaseline. For an adult no dressing is needed except a little antiseptic powder. Patients need not stop in bed more than

twenty-four hours. In clean dry cases the penis should be kept in the vertical position. In septic cases ordinary fomentations and dressings should be employed for a day or two.

3. **Paraphimosis.**—When the foreskin has been retracted and cannot be drawn forwards again, the whole of the red portion of the prepuce swells enormously. The glans penis is also swollen, but to a less extent. This characteristic appearance, called paraphimosis, is caused by the retraction of a foreskin with a small orifice either during copulation or otherwise; or it is caused by the retraction of an inflamed prepuce with a normal orifice, in which case the swelling of the foreskin from inflammatory exudation and oedema prevents its replacement. There are two classes of cases. (1) In the first group there is little pain with no inflammation, ulceration, or discharge from the parts. Under these circumstances, as the swelling is only an oedema, the penis should be fixed in the vertical position, and kept clean and dry. The swelling will slowly subside, leaving the penis with all the advantages of a circumcision. (2) Those in which there is a large amount of swelling from oedema and inflammation of the prepuce. In these cases there is acute suffering, and generally a discharge of pus. Gonorrhœa, syphilis or septic ulcers are also frequently present. If the constriction last a day or two, there will be a slough of the constricting ring (the preputial orifice). This slough starts on the dorsum at the bottom of the deepest crease.

TREATMENT.—Reduction should be effected immediately. Wind a piece of narrow elastic webbing around the glans penis from the meatus to the corona, and then on to the swollen red surface of the foreskin, remove the webbing quickly, apply vaseline to the glans penis, and then firmly press the glans through the constriction with the two thumbs. A piece of bandage placed loosely around the penis gives a good circle of counter-pressure. If this method fail, the constricting preputial orifice should be divided on the dorsum with a tenotome. The deepest and tensest constriction should be found: this is generally in the posterior crease. If there be gonorrhœa, chancres or other ulcers present, the best treatment is circumcision (*see* PHIMOSIS). After reduction apply lead and opium or antiseptic fomentations. Elevate the penis to the vertical position, and fix it by bandages around the waist.

4. **Adherent Prepuce.**—In many cases of phimosis there is some adhesion of the foreskin to the glans penis. This condition intensifies the misfortunes of phimosis, and if the adhesion be universal it will greatly retard the growth of the glans. In many cases the adhesions are only small and localised.

In the slighter cases retraction of the foreskin will stretch the adhesions sufficiently, but in the more severe cases circumcision should be performed. In this operation the adherent surfaces must be thoroughly separated by the handle of the scalpel, or if it be necessary by actual dissection.

Preputial Calculi.—In rare cases the secretion of the glandulæ odoriferæ around the coronopreputial junction collects in large quantities, forming calculi under the foreskin. These stone-like masses consist of lime-salts and organic matter. Occasionally they grow to a very large size. The treatment consists in circumcision. *See* p. 335.

5. **Penis, Inflammation of (*Penitis, Cavernitis*).**—A rare condition caused by severe injury or severe venereal disease. Cases have been recorded in the acute specific fevers, gout, and diabetes. Treatment is conducted on general principles.

6. **Penis, Gangrene of.**—A rare condition except as an extension of gangrenous ulcers of venereal origin, or from constriction by ligatures, rings, &c. Cases have been recorded of gangrene of the penis in the acute specific fevers. Death commonly results in the latter cases.

7. **Herpes Preputialis.**—This is a common complaint consisting of an outbreak of a crop of vesicles on the prepuce or skin of the theca. It is an ordinary acute vesicular dermatitis produced by septic discharges or by friction against the clothes, or in copulation. It recurs again and again. It is a very important point to diagnose this simple condition from more serious diseases, as it often causes great mental distress to the patient, who thinks that he has become inoculated with syphilis or other venereal disease.

The parts must be kept clean and dry, and lead-lotion applied. To prevent recurrence bathe the prepuce and glans with spirit-lotion or a solution of alum, in order to harden the epithelium. *See* HERPES PROGENITALIS.

8. **Benign Growths.**—The skin of the penis is liable to any of the ordinary diseases of the skin. The penis itself is very rarely affected by benign growths. The glans penis is sometimes the seat of a naevus, but the common benign growth of this region is the *papilloma* or *venereal wart*. The writer has seen a case of a huge dense fibroma involving the glans and the three corpora of the penis as far back as the pubis. It had been slowly growing for many years and was causing a pressure-stricture of the urethra with chronic retention. It was very pale in colour, hard, dense, and horny to the touch.

Papillomata are very common on any part of the penis or in the urethra, on the abdominal wall, thighs, perineum or anus. They may be associated with urethral discharges, but often they are not so associated, the penis being quite dry. They are infectious, they grow very rapidly and they do not infect glands. They may be small and dry or marvellously exuberant in growth, covering huge areas, red and fungating in appearance, pedunculated or sessile. They are soft growths infiltrating the skin only in their immediate bases. The discharge may be serum, blood, or pus. When they suppurate the stench is abominable. When they are moist the areas grow rapidly and will affect an opposing surface by inoculating it.

TREATMENT.—If the warts be small and dry, keep the parts clean and apply resorcin-powder. Cauterising with pure carbolic acid or ethylate of sodium is also useful. If the warts be large and pedunculated, inject a little β -eucaine into the skin and snip off the wart and its base by sharply curved scissors. Afterwards keep the wounds clean and dry with resorcin or some antiseptic powder. In bad cases the patient must stop in bed and, under a general anæsthetic, the whole must be removed by a superficial dissection of the warts and their bases. In very extensive cases the condition has to be dealt with in sections at different times.

9. **Penis, Malignant disease of.**—There are two varieties of malignant disease of the penis,

epithelioma and sarcoma. The latter is very rare, and occurs in the corpora. *Epithelioma* is a common disease, starting on the prepuce or glans penis as a pimple, wart, fissure, or hard nodule. It is a squamous-celled epithelioma. The apparent cause in many instances is the presence of a phimosis, under which irritant secretions have collected. Epithelioma is very uncommon in the circumcised. It occasionally starts from tertiary syphilitic ulceration of the penis. It is seldom found before the age of forty, the commonest period being from fifty to seventy years of age. There are two varieties of this disease. (1) The excrecent or papillary form is a large cauliflower-shaped mass of papillary growth, red, fungating, bleeding easily, discharging foetid pus. This outgrowth arises from a hard stony mass of infiltration in the glans or prepuce, its edges everted and very hard. (2) The excavating or cavernous form, in which a cavity is formed, has a stonily hard base with infiltration of the surrounding tissues, and a warty floor. It discharges a foul sanious serum or pus. In both varieties of the disease the superficial oblique inguinal glands are soon affected, and as the disease progresses into the corpora penis the iliac and lower aortic glands become epitheliomatous. The progress of the disease is somewhat slow at first, but more rapid when the corpora are involved. In the cavernous variety the disease progresses more rapidly than in the papillary, the amount of infiltration being greater in the former. As the disease advances it destroys every structure, ultimately forming a huge ulcer involving the penis, scrotum, and surrounding skin, the urethra opening as an ulcerated orifice upon it. If there be phimosis the ulcer will be hidden for a time until it ulcerates its way through the prepuce, producing multiple holes. The general symptoms of cachexia, wasting, &c., are well marked.

DIAGNOSIS.—This is not difficult in the majority of cases, but in a man over forty a chancre, gumma or ulcerating wart may be mistaken for cancer. In warts there is no infiltration of the skin or involvement of glands. In chancre and gumma (which may have existed for months) the surrounding tissues are hard and the glands in the groin may be very large and indurated. In the chancre there will be some signs of secondary syphilis in the throat, skin and other glands. In the gumma the diagnosis is often very difficult. Previous history of syphilis, the exhibition of iodide of potassium, and the microscopical examination of a piece of the tissue will confirm the diagnosis. Gummatous ulceration of the prepuce, glans and urethra are more common than is generally supposed. The writer has seen many more cases of gumma than of epithelioma.

TREATMENT.—The whole of the disease should be removed by operation. Either partial or total amputation of the penis and complete removal of the enlarged glands on each side is necessary. If the disease should have involved abdominal glands operation is not justifiable unless there is great suffering from pain, soreness, or the foul odour of the ulcer. In those cases in which the primary disease is very extensive and the secondary glandular involvement has spread to the spermatic cord or to the femoral vessels and anterior crural nerve, no operation will effect any improvement in the patient's condition.

Penis, Elephantiasis of.—See FILARIASIS.

CHARLES GIBBS.

PENTASTOMA DENTICULATUM.—The larval form of a member of the class Arachnida (*P. tenioides*). The adult form is met with in the nasal cavities of dogs, while the larva usually infests the liver of herbivorous animals; rarely it occurs in this organ in man, and still more rarely in the lungs, spleen, intestine, and kidney. Its body is 4 to 5 mm. long, and 1·5 mm. broad; it possesses about nine segments, with spiny margins. The mouth is surrounded by four hooks, with chitinous sheaths. Remnants of the larva, usually hooklets, are occasionally found *post mortem*, embedded in cretaceous nodules the size of peas. This parasite does not give rise to symptoms of disease in man, but may bore into the peritoneal cavity in lower animals and give rise to peritonitis. The mature form has been met with in the nostrils of a human subject. J. BLAND SUTTON.

PEPTONISED FOOD.—This term may be used as the equivalent of the phrase 'artificially digested food.' In natural digestion albuminoid substances are changed into peptones, and starchy matters into dextrin and sugar. These processes are of a purely chemical nature, and they can be imitated outside the body very closely by means of artificially prepared digestive juices. An extract of the stomach, or of the pancreas, in water, has approximately the same powers as the natural secretions of those organs. Hence it is possible for us to subject articles of food beforehand to complete or partial digestion; and to administer such artificially digested food to our patients. In cases where the natural digestive powers are more or less in abeyance, it is an obvious advantage to be able to give food thus modified.

Methods of Preparation.—Peptonised or artificially digested food may be prepared, either by following the gastric method with pepsin and hydrochloric acid, or by following the intestinal method, and using extract of pancreas. The latter method yields by far the better results. The pancreas acts not only upon albuminous substances, but also upon starch. Pepsin, on the other hand, is quite inert in regard to starch. Moreover, the products of artificial digestion with pepsin and acid are much less agreeable to the taste and smell than those produced by pancreatic extract. By the latter method articles of food can be profoundly peptonised with little deterioration of that agreeable savour which makes them inviting to the palate. This article will, therefore, be confined to the pancreatic method, and the modes in which food may be partially digested beforehand, and yet constitute an acceptable nourishment for invalids.

The first necessity is to procure an active extract of the pancreas. Water is the proper solvent of the digestive ferments; but, in order to obtain a stable preparation, some preservative agent must be added to prevent decomposition. A mixture of one part of rectified spirit with three parts of water answers every purpose. The pancreas of the pig yields the most active preparation; but the pancreas of the ox or the sheep may be employed, if that of the pig is not obtainable. The pancreas of the calf also yields an extract which is active on albuminous substances, but it is not active on starchy materials. In procuring a supply of pancreas from the butcher, it is well to remember that the word 'sweetbread,' which is the English vernacular for pancreas, is likewise applied to the thymus gland; and that the

genuine sweetbread of the kitchen is the thymus of the calf. Butchers distinguish the true pancreas as the 'liver-' or 'stomach-bread.' Liquor pancreatis is an official preparation. It is made from the fresh pancreas of the pig, freed from fat and external membrane, and ground up with washed sand or pumice-stone. To each part, four parts of 20 per cent. alcohol are added and the mixture is filtered after macerating for seven days. The dose is 1 to 2 drachms.

Directions for the Preparation of Various Kinds of Peptonised Food.—The articles which are most easily prepared, and are most likely to be serviceable to invalids, are the following:—

Peptonised Milk.—A pint of milk is diluted with a quarter of a pint of water, and heated to a temperature of about 140° F. Should no thermometer be at hand, the diluted milk may be divided into two equal portions, one of which is heated to the boiling-point and added to the cold portion, when the mixture will be of the required temperature. Two teaspoonfuls of the liquor pancreatis and ten grains of bicarbonate of sodium are then added to the warm milk. The mixture is poured into a covered jug, and the jug is placed in a warm situation under a 'cosey,' in order to keep up the heat. At the end of an hour, or an hour and a half, the product is boiled for two or three minutes. It can then be used like ordinary milk.

The object of diluting the milk is to prevent the curdling which would otherwise occur, and greatly delay the peptonising process. The addition of bicarbonate of sodium prevents coagulation during the final boiling, and also hastens the process. The purpose of the final boiling is to put a stop to the ferment-action when this has reached the desired degree, and thereby to prevent certain ulterior changes which would render the product less palatable. The degree to which the peptonising change has advanced is best judged of by the development of a peculiar bitter flavour, which is always associated with the artificial digestion of milk. The point aimed at is to carry the change so far that the bitter flavour is just perceived, but is not unpleasantly pronounced. As it is impossible to obtain pancreatic extract of absolutely constant strength, the directions as to the quantity to be added must be understood with a certain latitude. The extent of the peptonising action can be regulated, either by increasing or diminishing the dose of the liquor pancreatis, or by increasing or diminishing the time during which it is allowed to operate. By skimming the milk beforehand, and restoring the cream after the final boiling, the product is rendered more palatable.

Peptonised food is also conveniently prepared by means of powders (Fairchild's), each of which is sufficient to peptonise a pint of milk. It may be used for partially peptonising milk. One of the powders is mixed with a teacupful of cold water; a pint of cold milk is added and the mixture after stirring is placed for ten minutes in a vessel of water at a temperature as hot as can be borne by the hand (115°).

Peptonised Gruel.—Gruel may be prepared from any of the numerous farinaceous articles in common use—wheaten flour, oatmeal, arrowroot, sago, pearl-barley, pea- or lentil-flour. The gruel should be well boiled, and made thick and strong. It is then poured into a covered jug, and allowed to cool until it becomes lukewarm. Liquor pancrea-

tis is then added, in the proportion of a dessert-spoonful to the pint of gruel, and the jug is kept warm under a 'cosey' as before. At the end of a couple of hours the product is boiled, and strained. The action of pancreatic extract on gruel is twofold—the starch of the meal is converted into dextrin and sugar, and the albuminoid matters are peptonised. The conversion of the starch causes the gruel, however thick it may have been at starting, to become quite thin and watery. The bitter flavour does not appear to be developed in the pancreatic digestion of vegetable proteids, and peptonised gruels are quite devoid of any unpleasant taste. It is difficult to say to what extent the proteids of the meal are peptonised in this process. The product gives an abundant reaction of peptone; but there is a considerable residuum of undissolved material. Most of this, no doubt, consists of insoluble ligneous tissue, but it also contains some unliberated starchy and albuminous matter. Peptonised gruel is not generally, by itself, an acceptable food for invalids, but in conjunction with peptonised milk (peptonised milk-gruel), or as a basis for peptonised soups, jellies, and blanc-manges, it is likely to prove valuable. It is perhaps best reserved for rectal feeding.

Peptonised Milk-gruel.—This is the preparation of which the writer (W. R.) has had the most experience in the treatment of the sick, and with which he has obtained the most satisfactory results. It may be regarded as an artificially digested bread-and-milk, and as forming by itself a complete and highly nutritious food for weak digestions. It is very readily made, and does not require the use of the thermometer. First, a thick gruel is made from any of the farinaceous articles above mentioned. The gruel, while still boiling hot, is added to an equal quantity of cold milk. The mixture will have a temperature of about 125° F. To each pint of this mixture two or three teaspoonfuls of liquor pancreatis, and ten grains of bicarbonate of sodium, are added. It is kept warm in a covered jug under a 'cosey' for an hour or hour and a half, and then boiled for two or three minutes, and strained. If the product has too much bitter flavour, a smaller quantity of the liquor pancreatis must be used in the next operation. Invalids take this compound, as a rule, if not with relish, at least without any objection.

Peptonised Soups, Jellies and Blanc-manges.—The writer (W. R.) has sought to give variety to peptonised dishes by preparing soups, jellies, and blanc-manges containing peptonised aliment. Soups may be prepared in two ways. The first way is to add what cooks call 'stock' to an equal quantity of peptonised gruel or peptonised milk-gruel. A second and better way is to use peptonised gruel, which is quite thin and watery, instead of simple water, for the purpose of extracting the soluble matters of shins of beef and other materials employed in the preparation of soups. Jellies may be prepared by simply adding the due quantity of gelatine or isinglass to hot peptonised gruel, and flavouring the mixture according to taste. Blanc-manges may be made by treating peptonised milk in a similar way, and then adding cream. In preparing all these dishes it is absolutely necessary to complete the operation of peptonising the gruel or the milk, even to the final boiling, before adding the stiffening ingredient; for if pancreatic extract be allowed to act on the gelatine, the gelatine

itself undergoes a process of digestion, and its power of setting on cooling is thereby utterly abolished.

Peptonised Beef-tea.—A pound of finely minced lean beef is mixed with a pint of water, and ten grains of bicarbonate of sodium are added thereto. The mixture is then simmered for an hour and a half in a covered saucepan. The resulting beef-tea is decanted off into a covered jug. The undissolved beef-residue is then beaten up with a spoon into a paste, and added to the beef-tea in the covered jug. When the mixture has cooled down to about 140° F. (or when it is cool enough to be tolerated in the mouth), a table-spoonful of the liquor pancreatis is added, and the whole well stirred together. The covered jug is then kept warm under a 'cosey' for two hours, and agitated occasionally. At the end of this time, the contents of the jug are boiled briskly for two or three minutes and finally strained. The product is then ready for use. Beef-tea prepared in this way is rich in peptone. It contains about 4 per cent. of organic residue, of which more than three-fourths consist of peptone; so that its nutritive value in regard to nitrogenised materials is nearly equivalent to that of milk. When seasoned with salt it is scarcely, if at all, distinguishable in taste from ordinary beef-tea.

Peptonised Enemata.—Pancreatic extract is peculiarly adapted for administration with nutritive enemata. The enema may be prepared in the usual way with a mixture of milk and gruel, or milk, gruel, and beef-tea. A dessert-spoonful of liquor pancreatis is added to it just before administration. In the warm temperature of the bowel the pancreatic ferments find a favourable medium for their action on the nutritive ingredients with which they are mixed; and there is no acid secretion (as in the stomach) to interfere with the progress and completion of the digestive transformation. Experience has satisfied the writers that this method of administering nutriment is a valuable resource when the stomach is obstinately intolerant of food, or when there is obstruction in the higher portions of the digestive tract.

Peptonised beef is made by digesting beef with artificial gastric juice. It is very bitter, and is best administered in suppositories, each of which contains 30 grains of the preparation.

Uses of Peptonised Food.—The employment of food which has been wholly or partially peptonised is indicated when the natural digestive powers are from any cause enfeebled or suspended. The most striking benefits have been observed in cases of gastric catarrh with pain and intolerance of food; in gastric ulcer; in the anorexia and dyspepsia associated with valvular heart-disease; and in the various forms of pyloric and intestinal obstruction. Good results have also been obtained in cases of defective nutrition and intestinal irritation in infants, and in infective diseases with intolerance of food. In using peptonised food it is well to remember that it does not keep well, especially in warm weather. Accordingly it should either be prepared twice a day so that it may be never more than twelve hours old; or, if a quantity sufficient for the twenty-four be prepared at once, the portion which remains over at the end of twelve hours should be re-boiled.

WILLIAM ROBERTS.
SIDNEY MARTIN.

PERICARDIUM, DISEASES OF THE

PEPTONURIA.—A condition in which the urine contains peptones. See ALBUMOSURIA.

PERCUSSION (*percutio*, I strike).—A method of physical examination, performed by striking gently some part of the body, especially the chest or abdomen, for the purpose of producing certain sounds or tactile sensations. It may be performed either by the finger or fingers of one hand striking the surface directly; or indirectly, the fingers of the other hand being interposed; or by means of a special instrument or instruments. See PHYSICAL EXAMINATION.

PERIARTERITIS.—See ARTERIES, Diseases of.

PERICÆCAL ABSCESS.—See APPENDIX VERMIFORMIS, Inflammation of.

PERICARDIUM, Diseases of the.—SYNON.: Fr. *Maladies du Péricarde*; Ger. *Krankheiten des Herzheutels*.

Diseases of the pericardium will be considered in the following order: (1) Congenital defects; (2) Hæmopericardium; (3) Hydropericardium; (4) Melanosis; (5) Milk-spots; (6) Acute Pericarditis; (7) Chronic Pericarditis; (8) Pyopericardium; (9) Pyo-pneumopericardium; (10) Tuberculosis; (11) Tumours.

1. **Congenital defects.**—The parietal layer of the pericardium may be almost entirely absent, being represented only by a fringe of membranous projections around the roots of the great vessels; the epicardium is then in contact with the pleura. The condition is very rare and does not appear to be of clinical importance.

2. **Hæmopericardium** (*Effusion of blood within the pericardial sac*) may occur from rupture of an aneurysm of the ascending arch of the aorta; from wounds or rupture of the heart, the aorta, or a coronary vessel; or from the unexplained hæmorrhage connected with hæmophilia, purpura, and profound conditions of anæmia. Rarely an aneurysm of the descending aorta may rupture into the pericardium from without. The effusion in acute pericarditis is occasionally hæmorrhagic, but this is not usually classed as hæmopericardium. The condition leading to the hæmorrhage is usually fatal, death resulting directly from embarrassment of the heart's action by the effused blood (see HEART, Wounds of; THORACIC ANEURYSM). At the autopsy the pericardium is found much distended, and may appear of a dark bluish colour before it is opened. The blood within the sac is usually partially clotted: the amount effused is the measure of the force of the arterial pressure acting against the resistance offered by the pericardium. In cases due to rupture of the aorta or of an aneurysm, sudden fatal syncope occurs. When life is prolonged for a few hours, the dyspnoea and pallor are suggestive of severe hæmorrhage. The physical signs of the condition would resemble those of a large serous effusion (see below), but death is usually too rapid to allow a diagnosis to be made during life. Treatment is only possible in traumatic cases (see HEART, Wounds of).

Petechiæ, or small extravasations of blood beneath the serous membrane, are frequently found in infective diseases and in chronic wasting conditions.

3. **Hydropericardium** (*Dropsy of the Pericardium*).—These terms are applied to an effusion

of serous fluid into the pericardial sac, which is not due to inflammation. It may occur in cases of general dropsy, cardiac or renal; and is also said to result in rare instances from obstruction to the coronary or pericardial veins, as in cases of thrombosis of the coronary vessels or sinus, of mediastinitis, or of mediastinal tumour. In severe anæmia a large quantity of fluid is occasionally found in the pericardium at the autopsy; indeed it is the rule in such cases to meet with some increase beyond the amount normally present. It is probable that many cases of large effusions classed as hydropericardium are in reality inflammatory in nature; for it appears physically impossible that a merely dropsical effusion can exert a pressure sufficient to stretch the pericardial sac to any great extent. The physical signs presented in cases of dropsy of the pericardium resemble those of inflammatory effusion, but no friction-sound is produced. The heart may be impeded in its action by the surrounding fluid, and the symptoms of failure may ensue or be aggravated if already existing. The condition is, however, not a common one: it does not occur in general dropsy until after the appearance of ascites and hydrothorax; and relief should be sought rather by treatment of the general disease and by draining the other cavities than by any interference with the pericardial effusion.

4. **Melanosis** (*Black pigmentation of the parietal pericardium*) is a condition occasionally met with. Its exact causation is unknown. It is associated with the presence in the pericardial fluid of cells containing particles of pigment.

5. **Milk-Spots** (*maculæ albidæ*) are opaque patches found on the epicardium—occasionally also upon the parietal pericardium—in the hearts of adults and rarely of children. The most common situation is upon the anterior surface of the right ventricle; but these spots may also occur on the left ventricle near the apex and elsewhere. They are generally single, but may be multiple. It is probable that milk-spots are of the nature of callosities, produced by contact between the heart and the chest-wall. They are of no clinical significance; but it is possible that in rare instances they may give rise to friction-sounds and thus lead to a mistaken diagnosis of pericarditis.

6. **Acute Pericarditis** (*Acute Inflammation of the Pericardium*).

ETIOLOGY.—By far the commonest cause of acute pericarditis is (1) rheumatic fever. Thus of 74 cases of pericarditis recently in the wards of Charing Cross Hospital 53 were of a rheumatic nature, while among 350 patients suffering from acute rheumatism pericarditis occurred in 28 (8 per cent.). The second most important cause of this affection is (2) acute and chronic nephritis. Both the tubal and the interstitial form of renal disease are associated with pericarditis, but it is commonly seen in cases of granular kidney, occurring late in the disease, often as a precursor of uræmic symptoms and a harbinger of approaching death. (3) Extension of inflammation from neighbouring structures may involve the pericardium under many conditions. The most common of such causes is probably acute lobar pneumonia: others are myocarditis—the result of septic infection of the heart-wall—empyema, abdominal suppuration, and mediastinitis. Abscesses of the mediastinum, or of parts beneath the diaphragm, may burst directly into the pericardium in rare instances. (4) Traumatic infection

of the pericardial sac may be brought about by penetrating wounds and by fractured ribs, while a non-infective pericarditis may perhaps result from blows, or crushing of the chest-wall. (5) Infective organisms may be carried by the blood to the pericardium, as in septicæmia and pyæmia. (6) Certain general diseases, such as the acute specific fevers (especially scarlatina, variola, enteric fever and erysipelas), may be associated with inflammation of the pericardium: this also occurs in diabetes mellitus and in certain blood-diseases, such as scurvy and purpura. (7) Tuberculosis may attack the pericardium, as it does other serous membranes, and (8) tumours, both carcinoma and sarcoma, affecting the serous membrane may be associated with irritation and effusion around the actual growths.

Age and Sex.—Males are more liable to pericarditis than females. Rheumatic pericarditis attacks especially children and young persons, being comparatively rare in those over 25 years of age. Pericarditis associated with renal disease is commoner in elderly persons.

BACTERIOLOGY.—The organisms most frequently found in connection with this form of inflammation are streptococci, staphylococci, the pneumococcus and the *Bacillus tuberculosis*. It is possible that the streptococcus often recorded as present in rheumatic cases may have been in reality the organism recently described by Poynton and Paine as the specific cause of rheumatism. The pneumococcus is found in cases associated with lobar pneumonia: it may also be found in pericarditis occurring primarily and alone, while the involvement along with the pericardium of some other or of all the serous membranes is not very uncommon, and may be due to the same organism. In cases of wasting disease pericarditis is probably due to invasion by pyogenic organisms, the resistance of the tissues being lowered: this may also occur in tubercular cases, in which streptococci may be found in the pericardial exudation along with a few tubercle-bacilli; the latter apparently only accidentally present. Whether the pericarditis of renal disease is due to organisms thus enabled to establish a footing, or to toxins elaborated within the body (auto-intoxication), is not certain. In three cases reported recently by Chatin the toxicity of the blood-serum was constantly below normal and the pericardial effusion sterile. In cases associated with abdominal suppuration the *Bacillus coli communis* is probably responsible for the inflammation, and in cases associated with gonorrhœa the *Gonococcus*.

PATHOLOGY AND MORBID ANATOMY.—In the pericardium, as in other serous membranes, the inflammatory process may assume various forms, which are generally classed as (1) dry or plastic inflammation; (2) sero-fibrinous effusion; and (3) purulent effusion (*pyopericardium*). In some cases the effused fluid may be hæmorrhagic. The difference in the character of the process is due to varying virulence in the organisms or toxins which produce the condition. The earliest phenomenon in all cases is probably hyperæmia of the serous membrane, produced by dilatation of minute blood-vessels; this is quickly followed by exudation of a smaller or larger quantity of fluid on to the surface. The endothelial cells become swollen and granular, and some of them are cast off. To the naked eye the surface of the pericardium appears dull and opaque, losing its characteristic polish. The opposed surfaces, visceral and parietal, suffer together, but the

inflammation may either be limited to a small area of the sac or may involve the whole of its extent. On looking at the pericardium *post mortem* increased vascularity may be apparent before the sac is opened, a network of small vessels being visible all over the exposed surface between the anterior margins of the lungs; the latter are frequently adherent to the pericardium owing to outward extension of the inflammation. On cutting through the epicardium into the heart-muscle a zone of redness may be seen of appreciable thickness beneath the serous membrane, suggesting involvement of the myocardium; but though this does frequently occur it may often be found that the thin layer of fat lying beneath the epicardium is alone inflamed, thus acting as a protection to the important muscle beneath it. Microscopically, the pericardium is found thickened and infiltrated with round cells, which are probably in part emigrated leucocytes, and in part the offspring of the connective tissue-cells. The endothelial cells disappear at an early period of the disease. The phenomena thus far described are common to all forms of pericarditis. If the process goes no further, the *dry* or *plastic* variety results. The opposing surfaces are roughened and studded with small fibrinous excrescences, or covered with a thin layer of the same material. If the patient survive, more or less organisation of the new material occurs, and adhesion takes place between the two surfaces, or connecting threads or bands of fibrous tissue are produced by the continual movement of the heart. These may rupture and remain adherent only by one extremity. The filaments thus resulting may be so numerous and so closely set together as to give the heart the appearance of being covered with a shaggy coat of hair (*cor hirsutum*). When complete adhesion takes place the condition of chronic pericarditis or adherent pericardium is produced (see p. 1186). In other cases the amount of fluid which escapes from the dilated blood-vessels is much greater, and *serous* or *sero-fibrinous effusion* is the result. The amount varies from one or two ounces up to a pint or more. In the latter case the pericardial sac is distended and takes the form of a gigantic pear, the larger part being downwards; in cases of very large effusion the shape becomes almost globular. The fluid is either clear, yellow, transparent serum, or may be turbid from flocculi of coagulated fibrin, or, in rare cases, hæmorrhagic. It contains a large amount of albumen along with fibrinogen and fibrin-ferment, so that it may coagulate spontaneously. In cases due to the pneumococcus, this organism may be present in enormous numbers in the fluid; in other cases, other varieties of bacteria may be demonstrated by staining. The pericardial lining is generally coated with velvety lymph. In many cases the amount of fluid which exudes is much less, and there is practically a combination of the 'dry' with the 'wet' variety of pericarditis: the two layers of pericardium are adherent throughout a large part of their surfaces by means of thick fibrinous coagulum, while in the pockets remaining there are found collections of fluid, more or less turbid and opaque. In rare cases due to septic infection the effusion is purulent in character (see p. 1187—*Pyopericardium*). If the inflammatory process subsides, the sero-fibrinous fluid is gradually absorbed, but the damage done to the endothelium cannot be repaired. The condition remaining is similar to that found in the simple plastic variety. Adhesion takes place between the

opposed surfaces, and the cavity of the pericardium is more or less completely obliterated.

RESULTS.—The results arising from simple plastic pericarditis are not in themselves considerable, apart from injury to the cardiac muscle, which may accompany the pericarditis, and from the effects of subsequent adhesion (see p. 1186). Some authorities, however, consider that the pericardium normally acts as a support to the heart: according to this view the weakening of the membrane produced by inflammation may be a factor in the causation of the dilatation and failure of the heart which frequently occur. In cases in which there is much effusion the fluid surrounding the heart presses upon it and impedes its action. The enlargement of the sac may also cause pressure upon the œsophagus and resulting dysphagia. Pressure upon the lungs may produce collapse of portions of these organs, especially of the left lung. It has been suggested that the pleural effusion often found along with pericardial may be due in some cases to pressure exerted upon the veins in the mediastinum. The effect of the effusion upon the position of the heart itself has been much discussed. In all probability no constant alteration in position takes place, but the clinical fact of the appearance of the cardiac impulse in a higher situation than usual has not been satisfactorily explained.

CLINICAL FEATURES.—The onset of pericarditis is in the majority of cases insidious. In acute rheumatism it generally appears, if at all, within the first week or ten days of the illness, and more often in earlier than later attacks. The patient may not complain of any additional distress at first, or he may be conscious of a feeling of weight or tightness in the chest. With the exception of the *alae nasi*, which move with respiration, his face is often fixed and his expression anxious. In some cases there is pain in the præcordial region, which may be well marked or even violent; it may be of an aching, burning, or stabbing character, and may be referred to the epigastrium rather than the chest. The respiration is generally quickened, and the pulse full, soft, and increased in frequency. The heart's action may be tumultuous, and palpitation be complained of. The patient's temperature may rise with the onset of pericardial inflammation (101° F. to 103° F.), but in a large number of cases there is no fever. The occurrence of an initial rigor is quite exceptional, and is suggestive of suppurative pericarditis. There may be a short dry cough. In this early stage the physical examination of the patient can alone be relied upon to discover the condition. Tenderness is often found in the præcordial area upon pressure or percussion; in some cases this hyperæsthesia is very pronounced. The hand laid upon the chest may occasionally perceive a sensation as of two rough bodies rubbed together—thrill or friction-fremitus. Auscultation reveals a peculiar characteristic sound or sounds occurring along with, rather than replacing, the normal cardiac sounds. At first it is usually soft and 'whiffing' in character, but it may be harsh and creaking or almost grating. It may occur with both systole and diastole, or may not exactly correspond with either period of the cardiac cycle. A very typical auscultatory phenomenon is the 'triple rhythm' produced by co-existence of the two normal cardiac sounds along with a single adventitious friction-sound; at other times four distinct sounds are heard with each cycle of the heart,

a rhythm which has been compared to the distant puffing of a train leaving a station. Friction-sounds are confined to the præcordial area, and are usually loudest at the base of the heart. Their intensity may be increased by pressure of the stethoscope upon the chest-wall. They have a peculiar superficial quality, as if produced near to the surface of the thorax; and they may alter in intensity with the respiratory rhythm.

In severe cases delirium frequently occurs. It is generally of the noisy, active variety, but may be low and muttering. It is often combined with sleeplessness. The latter may be a troublesome symptom, even when the mind remains clear, as it occasionally does throughout, even in fatal cases. Hiccough is sometimes present, and vomiting may be a distressing and serious feature. The patient generally lies on his back, but may toss about restlessly from side to side. The face is often dusky and the expression anxious.

If the myocardium is seriously involved, symptoms of failure of the heart may appear—a running pulse, urgent dyspnoea, and cyanosis. Death may occur either suddenly by syncope or gradually by exhaustion. When recovery takes place the symptoms gradually diminish in intensity, but relapse may occur in some instances.

When effusion occurs the pain usually disappears; but if the amount of fluid be large the other symptoms may be accentuated. Dyspnoea is generally a prominent feature, and the patient may need to be propped up in bed or even to assume a bent posture with the head held down towards the knees. In extreme cases the cyanosis is marked, the *alæ nasi* work vigorously, and the distress of the sufferer is suggestive of grave disturbance. Physical examination will reveal an increased area of cardiac dullness. This increase is generally first noticed in the upper and left portion of the pericardial area—the dullness extending upwards to the second interspace or even higher—owing to the looseness of the pericardium at the roots of the great vessels, whereby it readily allows the accumulation of additional fluid in this region. As the effusion increases the lower portion of the pericardial sac also stretches and the dullness extends to the right of the sternum and beyond the apex of the heart, so that the whole area is triangular in shape, the base being on the diaphragm. The cardiac impulse may be diffused and present a peculiar wavy character; the apex-beat is often raised, being for a time recognisable in the fourth interspace. In children the wall of the chest may bulge forwards in the præcordial region. In cases of very large effusion an area of diminished resonance on percussion may be found behind at the angle of the left scapula, and also at the bases of the lungs near to the spine. The friction-sound audible at the beginning of the attack frequently disappears when effusion takes place; in other cases, however, it persists at the base of the heart throughout the illness. The normal cardiac sounds have a distant or muffled character; the action of the heart may be irregular, and an alteration of the pulse with the respiratory rhythm has been occasionally observed. The patient may complain of pain of a burning or stabbing character in swallowing, due to pressure of the bolus of food upon the inflamed and tender pericardium; actual difficulty in swallowing may occur, owing to pressure of the distended sac upon the œsophagus.

When the inflammatory process subsides absorption of the effused fluid rapidly takes place. The friction-sound generally returns at this stage, and the character of this sound may be rougher than at first; friction-fremitus is also more likely to occur. If a fatal result be impending the symptoms of cardiac failure are similar to those previously described as found in the 'dry' form of the disease.

The account first given of rheumatic pericarditis applies in the main also to cases which are secondary to pneumonia or pleurisy. In patients suffering from renal disease the onset of pericarditis is still more insidious, and the condition is frequently overlooked. Restlessness may be a marked feature, no pain being complained of, and delirium, drowsiness, or other uræmic symptoms may rapidly ensue. In other cases breathlessness is seen with rapid, feeble pulse, and much distress and anxiety, or even actual pain: this may be referred to the abdomen rather than to the chest. The frequency of the pulse and respiration are usually much increased, and the aspect may suggest the onset of some grave complication, the face seeming pinched, the eyes hollow, and the expression careworn. The temperature may be raised, but here, too, there is frequently no pyrexia to suggest the existence of an inflammatory process.

DIAGNOSIS.—In a few cases pericarditis may be present without giving rise to any distinct signs by which it can be recognised; much more often it is missed through not being looked for. In all cases of rheumatism, in acute pneumonia and other infective conditions, and in renal disease, it is most important to bear in mind the possibility of the occurrence of this complication, and to examine the heart frequently and methodically. In children, even apart from symptoms of rheumatism in the joints, the occurrence of fever and some degree of dyspnoea with a pulse which is more rapid than the general symptoms warrant, should give rise to a suspicion at least of the existence of pericarditis; even infants are not free from liability to the disease, and young adults may suffer from it without very well-marked joint-symptoms. The occurrence of delirium in the course of rheumatic fever should always suggest the onset of pericarditis. The dry form of the disease can only be certainly recognised by means of the characteristic friction-sound. It is possible that certain sounds produced by contact of the heart with the pleura, or by its rhythmical pressure upon a portion of the lung, may simulate this. As a rule, however, these cardio-pulmonary sounds alter much more with the respiratory rhythm than does the *bruit* in true pericarditis; the symptoms of serious disease are also absent in these cases. Endocardial murmurs may in rare cases give rise to doubt, but these are generally distinguished by their typical localisation, their conduction beyond the confines of the pericardium, and the absence of the peculiar superficial quality observed in most pericardial sounds.

Pericardial effusion of a dropsical nature may be indistinguishable from that arising from inflammation: the presence of a friction-sound would establish the existence of the latter, while hydro-pericardium never occurs to any serious extent until ascites and hydrothorax are already present.

A more difficult problem in diagnosis is presented by cases of dilatation of the heart, such as occurs owing to adherent pericardium or in the late stages of mitral disease. Here there may be increase of

præcordial dullness extending over an area of the chest practically identical in shape with that affected in cases of pericardial effusion; the cardiac impulse may be diffuse and wavy, the pulse frequent and soft, and cyanosis and dyspnoea be present owing to the failing action of the heart. In the absence of a friction-sound, or of a knowledge of the earlier physical signs, diagnosis may be impossible; and owing to the existence of such cases the operation of exploring the pericardium with a hollow needle is to be deprecated. Theoretically a localised empyema in the cardiac region might give rise to difficulties in diagnosis; the two conditions may co-exist and the pleural condition be overlooked owing to the predominance of the pericardial phenomena. Hæmorrhage into the pericardium is generally accompanied by almost immediate syncope and is rapidly fatal.

PROGNOSIS.—The immediate outlook varies greatly with the cause of the inflammation. In rheumatic cases recovery is the rule: thus out of 53 cases of this affection occurring in Charing Cross Hospital 10 died (under 20 per cent.). The mortality of this disease is heavy among young children and infants. The occurrence of great dyspnoea with cyanosis and a running or irregular pulse is of grave import: vomiting and the appearance of a low muttering form of delirium are also of bad omen. Cases of pericarditis associated with pneumonia are very prone to terminate fatally: out of 13 cases of this complication 8 succumbed (over 60 per cent.). In cases of renal disease the onset of pericarditis is almost invariably a fatal sign. Occasionally 'rheumatic' pericarditis may complicate Bright's disease; such cases are less frequently fatal.

In patients who recover from the acute attack some degree of adhesion between the layers of pericardium is left. This tends, especially in young persons, to embarrass the action of the heart and to increase the difficulty of establishing due compensation for any co-existing valvular defects. Thus there can be little doubt that in such cases the occurrence of pericarditis tends to shorten life and to render the appearance of heart-failure probable in later adult life (*see below*, Chronic Pericarditis). For purposes of life-assurance the occurrence of an attack of pericarditis would justify the addition of five to ten years to the applicant's actual age.

TREATMENT.—When pericarditis occurs in the course of acute rheumatism little additional treatment is as a rule necessary beyond that adopted for the primary disease. Complete rest is essential, the patient being carefully warned against any sudden exertion, such as sitting up or attempting to get out of bed. Any attitude which is most comfortable may be adopted, a semi-recumbent position maintained by means of pillows being often more restful than a horizontal posture. Milk-diet is to be continued: salicylate of sodium may also be given—indeed the depressing action of the drug upon the heart, when not too pronounced, is actually an advantage, for the excited action of the organ needs to be controlled in order that relative rest may be obtained. Pain may be soothed by means of either hot or cold applications to the præcordia: thus some patients find an ice-bag soothing and quieting, while others resent the application of cold and welcome that of hot fomentations. Blistering of the cardiac area is practised

by some authorities; it has the inconvenience of rendering subsequent auscultation difficult, and is of very doubtful efficacy. More relief may be obtained from the application of leeches: six, ten, or twelve of these may be used according to the age of the patient and the general condition; they should be applied along the left border of the sternum and over rib-cartilages rather than in the intercostal spaces, in order that any excess of hæmorrhage subsequently resulting may be more easily controlled by pressure. If the pain is not relieved by these means, opium or morphine (hypodermically) may be given with care in the early stage of the affection, when there are no signs of failure of the heart. Should such failure occur, stimulants are needed. Alcohol may be given in amounts rising from two to twelve ounces or even more. Ether, ammonia, strychnine and digitalis may all be employed; and inhalations of oxygen appear useful in some cases. If sleeplessness is a prominent and distressing symptom chloralamide is perhaps the safest hypnotic to use; chloral hydrate may also be given with care along with a little aromatic spirit of ammonia to counteract the depressing effect. Morphine may be necessary if insomnia persist, and may be given hypodermically along with small doses of strychnine. In cases in which effusion has occurred the above measures may still be tried. If the effusion be very large the question of its removal may arise. In view of the difficulty, previously alluded to, of distinguishing dilatation of the heart from pericardial effusion, and of the uncertainty that must exist as to the exact position of the heart, it is advisable to adopt the open method of incision (*see PERICARDIUM, Surgery of*) rather than to insert an aspirating-needle. If pleural effusion co-exist with pericardial, the former effusion should be previously removed, since the relief thus obtained to the lymphatics of the thorax may result in rapid absorption of the pericardial fluid. Owing to the danger of relapse the patient should be kept in bed for at least ten days after the symptoms have subsided; salicylates should be reduced very gradually and solid food as gradually permitted. It is possible that a certain degree of absorption of fibrinous or embryonic adhesions may be brought about by the administration of iodide of potassium; 5-grain doses of this drug three times a day might therefore be administered during this period; such absorption may perhaps be aided by light blistering of the præcordial area or counter-irritation with Liquor Iodi Fortis. Iron and quinine may be useful in the convalescent stage, and the greatest possible care must be subsequently taken to guard against exposure to chill, damp, or other condition that may favour recurrence of rheumatism (*see RHEUMATISM*).

7. Chronic Pericarditis (Adherent Pericardium).—This condition is the result of previous acute attacks. It consists in the adherence described above between the visceral and parietal layers of pericardium. The adhesions are formed by the 'organisation' of the effused lymph, that is to say, by the growth of connective tissue-cells into the coagulum, and the union of the opposed surfaces by this means. New blood-vessels form in the fibrous tissue, and this tends to shrink as it grows older, and thus to hamper the action of the heart. Extension of the inflammation to or from neighbouring parts may result in adhesion of the

pericardium externally to the chest-wall, the pleuræ, and the structures within the mediastinum. In some cases, especially those associated with mediastinitis, the fibrous coat surrounding the heart and binding it to surrounding structures is very thick; it may be almost cartilaginous and occasionally becomes infiltrated with calcareous salts—a condition which has given rise to the description of a ‘bony heart.’ When the heart is thus surrounded by a fibrous coat two possible results may occur. If the constricting tissue be very dense, the heart is unable to enlarge and is at the same time forced to work at great disadvantage owing to the resistance offered to systole by the adhesions. In such cases fatal failure of the heart necessarily ensues rapidly. In other cases the fibrous tissue may stretch and allow the heart to enlarge, which it does to a very great extent in response to the continual impediment offered to its action. This condition is frequently seen in rheumatic pericarditis. Failure of the heart in such patients is much less rapid but is none the less certain to occur ultimately. Various clinical signs have been described as indicating the existence of adherent pericardium. Of these the more important are retraction of the chest-wall—especially of the lateral wall of the thorax (Broadbent’s sign)—along with the cardiac systole, diffusion of the cardiac impulse, fixity of the position of the apex-beat in spite of change of posture, persistent enlargement of the area of cardiac dullness and disappearance of the pulse with inspiration (‘pulsus paradoxus’). It is evident that the mere existence of adhesion between the two layers of the pericardium cannot by itself give rise to any of these phenomena, all of which are dependent upon fixation of the heart and pericardium not only to one another but to external parts. Even in the last-mentioned condition no one of these signs is constantly present, and it must be confessed that certain diagnosis of the condition is impossible. In cases of mediastino-pericarditis (mediastinitis) great enlargement of the liver is usually the most prominent feature, pleural effusion is generally present, and general dropsy and anasarca occur (see MEDIASTINUM, Diseases of). Apart from this condition, pericardial adhesion may be suspected in cases of rheumatism presenting chronic cardiac enlargement without signs of valvular disease, or of rheumatic endocarditis in which the enlargement of the heart in all directions is greater than the amount of valvular lesion will account for. In children who have suffered from successive attacks of rheumatism, the pericardium is very frequently found universally adherent at the *post-mortem* examination. Failure of the heart to respond to the usual remedies is frequently due in such cases to the existence of this condition. No therapeutic measures will avail to absorb connective tissue that has once become fibrous. Treatment can only be directed to placing the patient under the most favourable general conditions, avoiding all strain upon the heart and all causes which aggravate existing disease of the valves, and meeting symptoms of cardiac failure, when they arise, by the usual measures. In very rare cases a chronic pericardial effusion may exist and may re-accumulate after tapping.

8. Pyopericardium (*Suppurative Pericarditis*).—The pathology of this condition has already been alluded to (see p. 1183). It may occur as a result of penetrating wounds, by which pyogenic organisms are carried into the pericardium;

from rupture of abscesses or vomicæ from without into the sac, or extension of the lesions of ulcerative endocarditis through the myocardium; and from general septic processes such as pyæmia and septicæmia. It may also be the final stage of a sero-fibrinous pericarditis, just as empyema may succeed to a serous effusion. In rare cases the pus is putrid and stinking, especially if air has gained entrance to the cavity. There are no means of certainly distinguishing purulent effusion from the serous variety, except exploration of the pericardium; although the occurrence of rigors and of hectic temperature establish a strong probability in favour of the former. Exploration should be done if the condition of the patient be grave and the cause of the effusion be such as to render the existence of suppuration probable. See PERICARDIUM, Surgery of.

9. Pyo-pneumopericardium (*Gas in the Pericardium*).—This is a very rare condition, and may be produced by penetrating wounds, by ulceration of carcinoma of the œsophagus into the pericardium, or by rupture into it of a tubercular cavity or of a subphrenic abscess communicating with the stomach or intestine. The presence of gas in the pericardium is almost invariably associated with suppurative, and the pus is frequently putrid and offensive. If the patient be already in an exhausted state there may be no sign to indicate the occurrence of the condition: usually there will be sudden anxiety, dyspnoea, and collapse. The præcordial region appears full; the impulse is feeble or absent; and the churning of gas and fluid produced by the movements of the heart may convey a peculiar sensation to the hand, and be audible as a splashing or rattling noise. A tympanitic percussion-note may be present over the præcordia. Succussion-splash and the bell-sound are said to have been obtained in some cases. The only other condition which may possibly give rise to similar phenomena and lead to an error of diagnosis is the existence of a large pulmonary cavity in the cardiac region near the surface in such a position that the movements of the heart produce splashing of its contents. Prognosis is probably hopeless. The only possible treatment is by incision and drainage of the sac.

10. Tuberculosis of the Pericardium.—In cases of general tuberculosis, miliary tubercles may occur beneath the endothelium of the pericardial sac, but the condition is very uncommon. Chronic tubercular nodules may occur on its walls and give rise to bands of adhesion. These are always secondary to tubercular disease in other organs, especially the lungs. Pericarditis occurring in tuberculous subjects is not always associated with the presence of tubercle-bacilli or with microscopical appearances characteristic of this disease; in such cases it is probably due to infection by other bacteria.

11. Tumours of the Pericardium.—Secondary nodules of carcinoma and sarcoma may occur in the walls of this cavity; they are generally associated with a certain amount of effusion, which is frequently hæmorrhagic. The pericardium may become infiltrated by new-growths arising in the mediastinum. See also p. 1139.

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W. CECIL BOSANQUET.

PERICARDIUM, Surgery of.—Paracentesis of the pericardium for the relief of distension of that cavity must be looked upon chiefly as a palliative

measure, by giving the heart a temporary freedom from the embarrassing fluid surrounding it. At the outset one may state that a distended pericardium cannot be reached with absolute certainty by the exploring needle. Owing to the dislocating effects of old adhesions or of pressure the needle may not enter the pericardium at all, but penetrate the lung, pleural cavity, or even the heart, especially if that organ has become adherent to the pericardium. It is important to remember that the diagnosis between hydropericardium and certain forms of dilated heart may be very difficult (see p. 633 and p. 1185). The writer has known of two cases in which a dilated heart has been punctured on the supposition that there was fluid in the pericardium, and in each case the autopsy revealed a pure hæmopericardium caused by the exploring needle.

At the base of the pericardium, on its anterior aspect, there is a small recess from $\frac{1}{2}$ to $\frac{3}{4}$ inch in depth. In the normal condition this is of course collapsed, but when fluid collects in the pericardium it becomes distended, and then lies opposite the sixth intercostal space on the left side, just at the edge of the sternum. At this spot the pleuræ have diverged from the middle line, and this recess can therefore be reached without opening the left pleural cavity. Unfortunately for practical purposes this little recess of the pericardium is difficult of access owing to the narrowness, or even obliteration, of the sixth intercostal space at this spot by the approximation or junction of the sixth and seventh ribs. Many writers have accordingly recommended a spot above this level on the left side. Here, however, the pleural cavity is very likely to be transfixed, and if pus is present in the pericardium there is considerable probability of the pleura becoming infected.

The operative procedures upon the pericardium are :—

A. Aspiration of the Pericardium (*Paracentesis Pericardii*).—If this operation is performed for diagnostic purposes only, the needle should be a very fine one, such as that of Pravaz. A small vertical incision, $\frac{1}{2}$ inch long, is made at the left border of the sternum at the level of the fifth intercostal space. The needle is then directed backwards exactly at the left border of the sternum so as to avoid the internal mammary artery, which generally lies a little way from the border of the sternum. If fluid is found, the exploring syringe may be withdrawn, and a larger needle attached to an aspirator inserted with a view to removing the fluid. During this procedure the patient should be propped up in bed with pillows, and every available method of stimulating the heart should be ready for immediate use in the event of sudden failure of its action. Apart from the relief afforded by the removal of a mechanical obstruction to cardiac action, the fluid in some cases does not re-form, and so permanent benefit may result.

B. Drainage of Pericardium (*Pericardiotomy*).—A small semicircular flap of skin is made with its base at the left edge of the sternum, extending vertically from the fourth to the sixth space inclusive, and measuring about 5 inches in radius. The fourth, fifth, and sixth costal cartilages are then removed along the whole extent explored; the intercostal muscles and aponeuroses are carefully cut at the edge of the sternum and turned outwards as a flap so that the internal mammary artery is exposed. This vessel is double-ligatured, divided, and drawn

aside with fibres of the triangularis sterni muscle and the reflected fold of the left pleura. The distended pericardium is then carefully incised; the contents evacuated; and a small soft drainage-tube inserted: this should lie at the lowest part of the cavity. The incision into the pericardium should then be sewn up except at the point where the drainage-tube projects.

HENRY PERCY DEAN.

PERIHEPATITIS (*περί*, around; and *ἥπαρ*, the liver).—Inflammation of the capsule of the liver. See LIVER, Inflammation of.

PERINEPHRITIS (*περί*, around; *νεφρός*, the kidney).—SYNON.: Fr. *Périnephrite*; Ger. *Perinephritis*.

DEFINITION.—An acute or chronic inflammation of the fibro-fatty envelope of the kidney, terminating occasionally in resolution, but more frequently in suppuration and the establishment of a chronic purulent discharge.

ÆTIOLOGY.—Perinephritis may develop as a primary affection after exposure to cold, or in association with wounds, bruises, and strains. It may follow surgical operations on the lower urinary passages, testicle, or spermatic cord; and it may also arise in the course of, or as a sequel to, the acute specific fevers, pyæmia and puerperal fever. The disease, however, originates most frequently in a secondary manner in connection with suppurative nephritis or pyelitis, either by direct perforation or by infection through the local veins and lymphatics. It is specially related to calculous pyelo-nephritis. It may also arise by extension from lesions of more distant parts in the abdomen or pelvis, including the cæcum, colon, appendix vermiformis, uterus, gall-bladder, spleen, and spinal column; or in association with thoracic inflammations, such as pneumonia, abscess of the lung and empyema. Perinephritis may occur at any age, but is less common in children than in adults. Among the latter, males show a slight preponderance.

ANATOMICAL CHARACTERS.—The initial congestion of the inflamed cellular tissue is followed by exudation into its meshes, which adds to its bulk and density. The subsequent changes vary in those cases that do not undergo resolution speedily, and a classification into three types has been suggested (Morris), according to the direction taken by the morbid process. *Firstly*, a hypertrophic condition of the fibro-fatty tissue may result, that may deform the kidney by compression or the growth of fibro-fatty processes into its substance; *secondly*, organisation of the exudate into firm white fibroid material, which by its sclerosis and contraction may compress the kidney and wholly obliterate its vessels; and, *thirdly*, suppuration may take place and the phlegmon become converted into a perinephritic abscess. Such abscesses may develop from a central suppuration, but often several foci of pus-formation appear, and the abscess remains for some time loculated or multiple. Hence the difficulty clinically in detecting fluctuation, and the futility in many cases of aspiration as a mode of treatment. The pus may be odourless or fetid from the decomposition of sloughs of cellular tissue floating in it. A fæcal odour may be present, even though no perforative communication with the colon exist. The abscess may attain immense size and occupy the entire loin and iliac fossa, and may ex-

tend by burrowing to the thigh or the thorax. It may be accompanied by œdema of the muscles and integuments around.

SYMPTOMS AND COURSE.—In an acute case the general symptoms are severe, the temperature rising to 101° – 105° F., with recurring rigors and a soft, rapid feeble pulse. The skin is hot and dry at the beginning, but later free perspirations occur and there is much thirst. There is marked constipation, and the urine is high-coloured and concentrated. In a few cases it may be little altered, while in others its characters may be modified by the antecedent renal or vesical disease.

The local symptoms comprise dull aching, less often lancinating, pain in the affected loin, radiating along the branches of the lumbar plexus to the thigh or knee. Slight flexion of the hip and some rigidity of the spine with inclination towards the affected side are common, evidently due to a desire to relax tension. Movement and pressure greatly aggravate the suffering. Later, a firm swelling develops which is dull on percussion. It surrounds the kidney and is inseparable from it, but it is usually possible to isolate it from the liver and spleen. It is not displaced by respiratory movements. The swelling has a solid weighty feel at first, and does not fluctuate till suppuration is well advanced. In simple perinephritis no tumefaction occurs; it is only in the phlegmonous form, that terminates in abscess-formation, that a definite mass can be felt. By its pressure on the veins, more particularly on the right side, œdema of the foot and ankle may be produced. A few cases terminate in resolution, but the majority in suppuration. The abscess so formed steadily enlarges and may discharge spontaneously by the following routes: Through the skin in the loin, groin, or thigh; through the urinary channels by bursting into the renal pelvis, ureter, or bladder; through the colon; or through the diaphragm into the pleura and lung with the establishment of a purulent expectoration. When the pus has been evacuated by any of these channels a natural cure may follow, but frequently a chronic discharging sinus remains, and the patient eventually succumbs to hectic fever or amyloid degeneration of the viscera. A rapidly fatal termination results when the abscess bursts into the peritoneal cavity, or when pyæmia or septicæmia supervene.

DIAGNOSIS.—Perinephritis is distinguished from appendicitis by observing that in the latter the pain and tenderness are anterior and iliac rather than posterior and lumbar, and by the greater disturbance of the stomach and bowels. In spinal caries with psoas abscess the onset is gradual with little or no elevation of temperature, there are bony tenderness and deformity, and the psoas abscess is less painful and has a more perfect fluctuation than the perinephric. In lumbago the pain is sharper and is referred to the muscles of the back: there is no swelling and little fever. Rupture of an aneurysm in the loin is recognised by the sudden development of the swelling, the comparative absence of fever, and by exploratory aspiration in obscure cases. In hydronephrosis there is no fever and little pain, and the fluctuation is very definite. Organic diseases of the kidney, liver, and spleen are distinguished by the situation and relations of the swelling, its consistence and the probable absence of fever. Stercoral colitis is differentiated by the doughy consistence of

the mass, and its rapid disappearance under evacuant treatment.

PROGNOSIS.—The simple form may be expected to end favourably. Phlegmonous perinephritis is a serious malady, that if unrelieved may terminate fatally in from two to four weeks. Rapid recovery in numerous cases follows operation, and in a smaller number a satisfactory result is occasionally obtained, after a tedious illness, by a spontaneous discharge through the skin, bowels, urinary passages or lung—these routes being named in the order of their relative safety. After escape of the pus, should the original cause persist, and prove incapable of removal by radical operations, death ultimately ensues from hectic fever or amyloid degeneration. If the abscess burst into the peritoneum, or pyæmia or septicæmia arise, death is inevitable in a few days.

TREATMENT.—In an acute case a hot bath, rest in bed, and saline aperients, together with the local application of hot sedative stupes and poultices, should be ordered. The advantage of local blood-letting, or even dry cupping, at the outset, is too often overlooked. The diet may consist of nutritious slops with avoidance of stimulants at this stage.

When suppuration is suspected, and even before fluctuation can be made out, a free incision under full antiseptic precautions through the fascia lumborum is indicated. With the finger introduced into the cavity septa are to be gently broken down and sloughs removed. The cavity should be thoroughly irrigated with mild antiseptic solutions and a large drainage tube inserted. This tube should not be discarded too soon owing to the great depth of the space and the tendency of the skin-wound to become valvular. Aspiration, formerly much in vogue for diagnostic and evacuant purposes, is not to be recommended, for the reason, that there may be several foci of suppuration, coagula, sloughs of tissue, calculi or other débris which cannot be satisfactorily dealt with through a needle. When a chronic sinus remains, and refuses to close after astringent injections or caustics have been applied to its walls, the possibility of a radical operation upon the kidney or other organ calculated to remove the original cause may be considered. Otherwise the patient's strength must be maintained by a liberal diet, tonics and stimulants; and the development of hectic fever or amyloid disease combated as long as possible. In those forms of perinephritis tending to sclerosis or hypertrophy of tissue counter-irritation by iodine or blisters, with the internal use of the iodides in tonic mixtures, may be employed.

THOMAS SINCLAIR.

PERINEURITIS (*περί*, around; and *νεῦρον*, a nerve).—Inflammation of the connective-tissue sheath of a nerve, usually more or less associated with neuritis. See NEURITIS.

PERIOD OF INCUBATION.—See INCUBATION.

PERIODIC PARALYSIS.—See PARALYSIS, PERIODIC.

PERIODICITY IN DISEASE.—Laycock, from a general review of the periodical phenomena observed in menstruation, in utero-gestation, in the development of the ova of fishes, and in the metamorphoses of insects, came to the conclusion that

physiological changes occur in animals at intervals of certain numbers of days or weeks. He concluded that there are critical days in health, and endeavoured to show that the changes in some diseases followed a similar rule of periodicity, reverting to the ancient doctrine of 'critical days' in fever of which Hippocrates treated. The existence of cyclical periods in fever is exceedingly doubtful, although, according to Wunderlich's observations, the majority of cases of typhoid fever run a regular course, divided into periods corresponding in time with the division into weeks and half weeks.

The two diseases in the course of which periodicity is most marked are malaria and relapsing fever. In the case of malarial fever it is now recognised that the period at which the paroxysm recurs is determined by the life-history of the parasite causing the disease. The differences between the tertian and quartan intermittents are found to depend upon differences in the parasite. In the regular intermittent fevers the parasite chiefly exists and in great part completes its cycle in the circulation; in a second group, comprising the malarial fevers which occur during the hot season of countries where malaria is most virulent, the parasite chiefly exists and in great part completes its cycle in the internal organs. In this latter group the regularity of recurrence of the paroxysms is wanting (*see* MALARIAL DISEASE). In relapsing fever also the relapses have been shown to be due to the life-history of the spirillum, the organisms increasing in number with the advent and progress of pyrexia and disappearing with its cessation. *See* RELAPSING FEVER.

Seasonal Variations.—The statistical investiga-

tions of Buchan and Mitchell have shown that most diseases have a constant relation to season, many being directly controlled by variations in temperature. Longstaffe, as the result of plotting out the death-rates from the majority of alleged causes, both compared with one another, and with curves representing meteorological elements, was led to classify diseases into four groups—hot diseases, cold diseases, dry diseases, and indifferent diseases. Among the 'hot diseases' he included diarrhoea, want of breast-milk, atrophy and debility, tabes mesenterica, thrush, enteritis and dysentery; among 'cold diseases' he included most affections of the respiratory organs, apoplexy, paralysis, mortification, heart-disease; by 'dry diseases' he meant scarlatina, puerperal fever, erysipelas, acute rheumatism and laryngitis, which he claimed are greatly checked by rainfall, especially frequent rainfall. Winter or summer maxima are thus produced, and with respect to some of these diseases the relation of the mortality to season is not difficult to understand. With others the exact conditions which determine rhythmical prevalence are not understood. The seasonal variation in mortality from some of the more common communicable maladies is well shown by the accompanying diagrams. Scarlet fever, diphtheria, and enteric fever, it will be seen, have their maxima in November, and puerperal fever, erysipelas, and rheumatism resemble them in this respect. Small-pox and whooping-cough have their maxima in the spring, and measles has two maxima. The second maximum of measles so conspicuous in the London curve is less obvious in that of some other English towns.

LONDON.

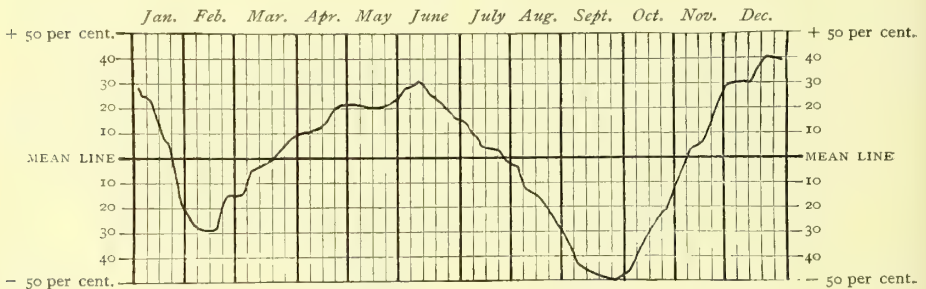


FIG. 1.—MEASLES (60 years, 1841-1900).

The mean line represents an average weekly number of 36 deaths.

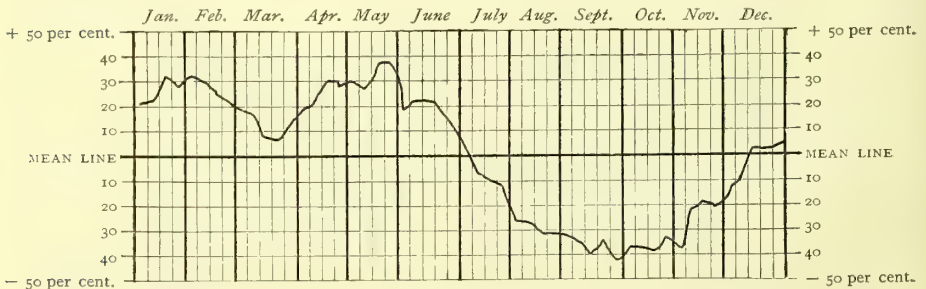


FIG. 2.—SMALL-POX (60 years, 1841-1900).

The mean line represents an average weekly number of 14 deaths.

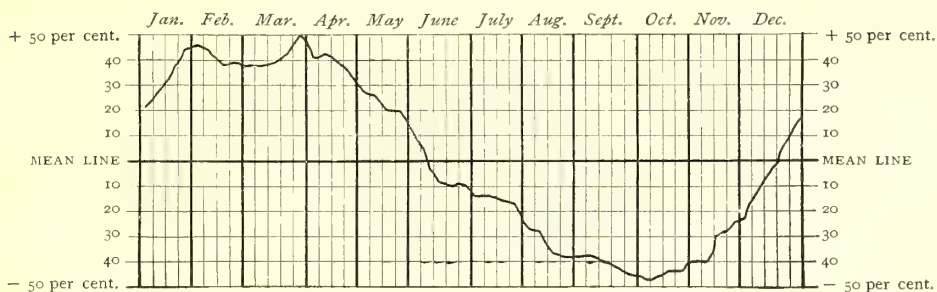


FIG. 3.—WHOOPIING COUGH (60 years, 1841-1900).

The mean line represents an average weekly number of 46 deaths.

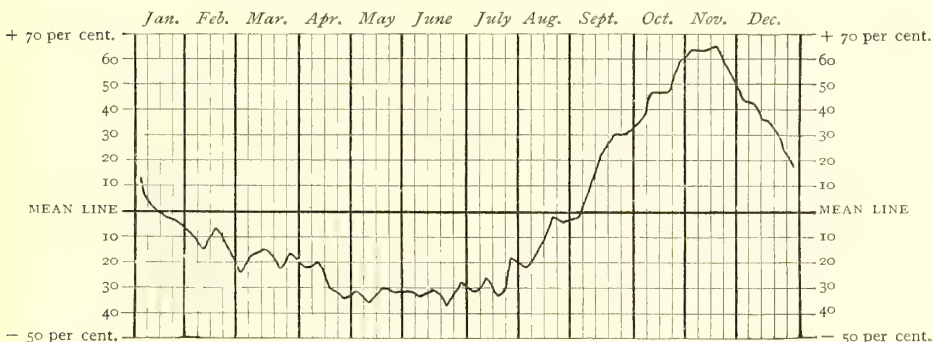


FIG. 4.—ENTERIC FEVER (30 years, 1871-1900).

The mean line represents an average weekly number of 14 deaths.

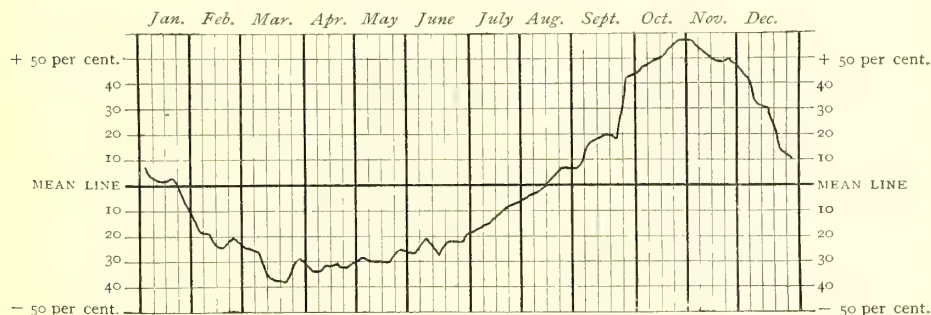


FIG. 5.—SCARLET FEVER (40 years, 1861-1900).

The mean line represents an average weekly number of 37 deaths.

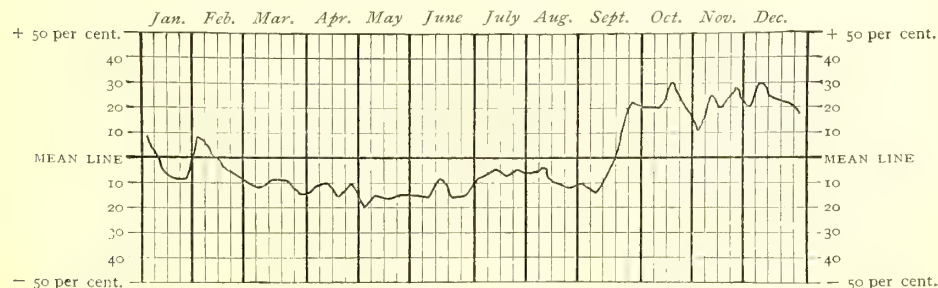


FIG. 6.—DIPHTHERIA (40 years, 1861-1900).

The mean line represents an average weekly number of 20 deaths.

Examination of the notified cases of scarlet fever and diphtheria in London has supplied evidence that these diseases manifest seasonal periodicity in other particulars than that of prevalence; thus both in respect of age-distribution of the notified cases and in respect of fatality there is seasonal variation. It is interesting to observe that the seasonal curves of prevalence do not accord with the curves of seasonal fatality, and that the months of greater prevalence, generally, have the lower fatality. Astley Gresswell, however, as the result of classifying 588 cases of scarlet fever under his care in one of the hospitals of the Metropolitan Asylums Board between September 1887 and February 1888, came to the conclusion that 'as the scarlatina mortality for the whole of London rose, most of the symptoms increased in relative frequency and severity; and that as the prevalence and mortality subsided, so also did the relative frequency and severity of most of the symptoms.' It may be that explanation of the difference between the conclusions suggested by Gresswell's observations and the London notification-figures is due to the fact that hospital cases represent selected cases, the more severe cases being sent to hospital. In the absence of notification of whooping-cough and measles in London, there is no opportunity of determining whether these diseases are subject to similar seasonal variations in fatality, but there is marked seasonal variation in the age-distribution of the deaths, those under one year of age occurring in much greater proportion in the months of May, June, July, August, and September, than in other months of the year, and the maximum occurring in whooping-cough in August and in measles in July.

Variations in periods of years.—Longstaffe (*Studies in Statistics*) has brought out some new and important points in regard to the broader cycles which extend over periods of years. He found that from 1855 to 1880, not only in London, but also in England generally, scarlet-fever mortality rose and fell with considerable regularity at intervals of five or six years, the actual maximum having occurred in 1858, 1863-64, 1869-70, and 1874. This curve was also followed (more or less closely) by a group of diseases which in their seasonal distribution are most like scarlet fever. As regards erysipelas, puerperal fever, and rheumatism of the heart, the parallelism is almost complete, and there are plain indications of affinity in those of laryngitis, diphtheria, croup, and quinsy. During recent years, however, with declining mortality the rhythmic recurrence of scarlet fever has ceased to be perceptible in mortality-returns. Small-pox, which had been epidemic every four or five years, in London at all events, has also in recent years been less rhythmical in its prevalence. Measles has been epidemic at intervals which, as Munro and others have shown, average about two years in most towns, but may be longer or shorter; and whooping-cough at still less regular intervals of two or three years. These three diseases have no similarity to the scarlet-fever group in their seasonal curve. Another group, of which diarrhoea is the most prominent representative in this country, reach their annual maxima shortly after the highest temperature of the year is attained; their true relation being with the temperature of the soil. Diarrhoea often shows a curious tendency to biennial sequence of alternately high and low mortality; but on investigation, this, when it occurs, is usually sufficiently

explained by alternation of hot and cold summers. Whitelegge has shown that, whatever their causes may prove to be, these cycles, like the seasonal changes characteristic of each disease, are too definite to be regarded as due to accident. Nor are they likely to be purely mechanical, for they vary not only with each disease, and to some extent with locality, but also from decade to decade. There is reason to believe that they are connected primarily with variations in the quality of the contagium itself. As a general proposition, it may be stated that the years of greatest mortality are those in which both the prevalence and virulence are greatest. As regards scarlet fever, the case-mortality (that is, the percentage of fatal cases), if traced, year after year, seems to yield a curve which is almost parallel with that of the scarlet-fever death-rate. In the last decade, however, in London, with declining mortality this correspondence has been less exact. Statistics are wanting in respect of the case-mortality in measles, but occasionally, as in Sunderland in 1885 and Hanley in 1889, an exceptional virulence and severity of type are observed; and in these and other instances it has been found that for some ten years previously the usual biennial waves have gradually increased in destructiveness as shown by the records of mortality. Upon similar evidence it may be suspected that small-pox declined in average virulence, as it certainly did in average mortality, from about 1838 to about 1855, increasing again to a maximum in 1871, and thenceforward declining again until 1888 or 1889. Here, however, the question is further complicated by the dominant influence of vaccination, in addition to the social and climatic conditions which are obviously concerned in promoting or retarding epidemic extension.

SHIRLEY F. MURPHY.

PERICESOPHAGEAL ABSCESS.—See (ESOPHAGUS, Diseases of, p. 1114.

PERIOSTEUM, Diseases of.—See BONE, Diseases of.

PERIPHERAL (περί, around; and φέρω, I carry).—Of or belonging to the periphery or circumference, as opposed to the centre. The term is now applied chiefly to morbid conditions connected with nerves and nerve-trunks, as distinguished from the more central parts of the nervous system (see PARALYSIS). Peripheral may also be associated with the vessels, as distinguished from the heart, for example, *peripheral resistance*; and with the outer zone of the lobules of glandular organs, as, for instance, of the liver.

PERIPHERAL NEURITIS.—See NEURITIS, MULTIPLE.

PERIPROCTITIS (περί, around; and πρωκτός, the anus).—DEFINITION.—Inflammation of the connective tissue around the rectum.

ANATOMY.—The fat-bearing connective tissue beneath the peritoneum of the rectum is usually present only in moderate quantity; but posteriorly it becomes continuous, directly or through the meso-rectum, with a considerable layer of this tissue in front of the sacrum. Below the level at which the peritoneum disappears the periproctal connective tissue becomes continuous with that of the pelvis generally, in the male especially with that at the base of the bladder, around the vesiculae and

bladder, and in the female with that in the broad ligament.

The fatty tissue of the ischio-rectal fossæ is separated from the periproctal tissue by the considerable septum formed by the levator ani with the rectovesical fascia upon its pelvic, and the anal fascia upon its perineal aspect. This constitutes an obstacle, but not a bar, to the spread of inflammations from the pelvis to the perinæum.

ÆTIOLOGY.—Primary inflammation of the periproctal (as distinct from the ischio-rectal and circum-anal) tissue is very rare, except as the result of injury with infection. Such injuries have been inflicted by bullets, stabs, falls upon pointed objects, by catheters thrust through the urethra, or fish-bones, injection-tubes and bougies thrust through the rectum, by tears and cuts inflicted in the treatment of simple strictures of the bowel.

In the great majority of cases periproctitis is secondary to some other lesion. Most commonly this is ulceration of the rectum leading to infection and more or less rapid destruction of the rectal wall. Perforating ulcers, simple or malignant, of coils of small gut which adhere to the rectum, of the vagina, uterus, or bladder; sloughing of the vagina from prolonged pressure or intense infection; prostatic abscess and acute vesiculitis; sacral caries with abscess, are occasional causes. Pyæmic abscesses have, rarely, been found in this tissue; more often, though still rarely, an acute suppurative cellulitis has arisen as the result of infection from some wound, e.g. that after ligature of piles or even a fissure of the anus, two instances of which the writer has recorded in the *St. Bartholomew's Hospital Journal*. In all cases of general pelvic peritonitis and in many of pelvic cellulitis, the periproctal tissue is more or less affected, the rectum being commonly 'set' in the exudation, and sometimes perforated by a bursting abscess.

SYMPTOMS.—The symptoms of periproctitis can hardly be separated from those of the condition to which it is secondary. In *acute* cases, sense of weight and pain in the rectum, frequent calls to stool with passage of mucus and perhaps blood, tenesmus, fever—slight or grave—may be expected. Rectal examination should be carefully practised to detect the presence of a primary lesion and evidence of any swelling in the rectal wall or in its vicinity. In *chronic* cases, the bowel is usually contracted; ulceration, stricture, the dimpled orifice of a sinus, its course and source are the main points to look for. The neighbouring parts also must be carefully examined.

TREATMENT.—The exciting cause must be discovered and treated.

F. C. WALLIS.

PERITONEUM, Diseases of.—The morbid conditions of the peritoneum will be considered in the following order:—

Acute General Inflammation, p. 1193.

Acute Localised Inflammation, p. 1200.

Chronic Inflammation, p. 1201.

Hæmorrhage into, p. 1203.

Malformations of, p. 1204.

Morbid Growths of, p. 1204.

Tuberculosis of, p. 1204.

For Dropsy of the Peritoneum, *see* ASCITES.

PERITONEUM, Acute Inflammation of.—

SYNON.: Acute Peritonitis; Fr. *Péritonite aiguë*; Ger. *Acute Bauchfellentzündung*.

ÆTIOLOGY.—It is now admitted on all sides that acute inflammation of the peritoneum is with few exceptions caused by the activity of micro-organisms. At the same time it must be remembered that bacteria can only effect this result under certain conditions. In order thoroughly to appreciate the rôle played by germs in producing peritonitis the philosophical maxim that 'every condition is causal' must be constantly borne in mind.

During the past few years numerous investigations have been undertaken with the view of discovering the organisms met with in peritonitis, and of establishing their exact relationship to the ætiology of the disease. The purely laboratory investigations must of necessity deal with peritonitis produced in animals, and when using facts derived from this source we must try and check the experimental results with the facts gathered from our observations on peritonitis met with in man.

In pre-bacteriological days Wagner had formulated with considerable precision the relations of the peritoneum to different influences. He proved the impunity of contact with air and of certain neutral fluids, and even of some decomposing fluids, provided they were in a quantity which could be easily absorbed by the peritoneum. These observations of Wagner were confirmed by an extensive series of experiments carried out by Grawitz. Grawitz showed that the introduction of saprophytes, even in enormous quantities, when diluted with normal saline fluid, did not harm the healthy peritoneum; also that the contents of the intestines in small quantities, and even in large quantities when sterilised by heat, could be injected into the peritoneal cavity without any harm resulting; finally, he found that well-known pyogenic organisms, such as the staphylococci and streptococci, could be introduced with impunity, provided the following four conditions were present, viz. (1) that the fluid used to dilute the cultures is innocuous to the peritoneum; (2) that the amount of fluid injected does not exceed the power of absorption of the peritoneum; (3) that the peritoneum is uninjured; (4) that no albuminous fluid, which very much favours bacterial growth, is present.

These experiments were repeated by Pawlovsky, who came to conclusions in many instances different from Grawitz. He found that minimal doses of *Staphylococcus pyogenes aureus* led to fatal peritonitis, and that the *Bacillus pyocyaneus* was especially fatal. The explanation of these contradictory results seems to lie in the fact that each experimenter was working with cultures of different virulence. This difference we are seeing constantly in relation to the human peritoneum; e.g. a woman when at work suddenly ruptures an irreducible umbilical hernia; the coils of intestine are bound up in a petticoat or similar garment; she is brought to the hospital and the intestines are washed, returned to the peritoneal cavity, and the patient recovers. On the other hand, a small punctured wound of the abdominal wall without any wound of the gut may cause fatal peritonitis. There is little doubt that in both cases ordinary pyogenic organisms obtain entrance, but they are quite different in their virulence. We must also bear in mind that the defensive powers of the peritoneum may differ markedly in different people.

While studying the effects of micro-organisms under the rigid conditions obtained in a laboratory, where the results are fairly definite and constant,

we are apt to lose sight of subtle differences that may exist in cultures of the same germ, differences that might come into play with great effect when laboratory discipline is once relaxed. From a consideration of the facts derived from operative surgery upon the abdomen and from experimental work we may formulate the conditions under which various micro-organisms will produce peritonitis:—

1. The presence of suitable medium for growth, that is, an albuminous substance such as lymph or blood.

2. The quantity of germs introduced into the peritoneal cavity from without or from the intestines, being in excess of that with which the absorptive power of the peritoneum can deal.

3. Damage to the serous membrane of the peritoneum. This may be of various kinds: (*a*) Necrotic action of the substance upon some part of the peritoneum, which will form a favourable nidus for bacterial growth, e.g. as in perforating gastric ulcer. (*b*) The diminution of absorptive power by the sealing up of lymph-spaces and capillaries. (*c*) An increase in the transuding power, as in the ordinary form of ascites or in cancer of the peritoneum. All these conditions are really causes of the ultimate result; and must be considered as well as the state of activity of the micro-organism. It seems undoubted that a germ which has been producing inflammation in the peritoneum of one person is much more vigorous in its attack if carried immediately to the peritoneum of another. This probably depends upon some property the germ itself has acquired. The best example of this is seen in puerperal septicæmia. The result of inoculation here produces an effect as definite and constant as the effect alluded to above as being obtained in laboratory experiments. It is in such instances as these that the organism stands out as being practically the sole cause of the disease. At any rate the conditions present take a very subordinate part.

Although peritonitis is nearly always associated with germs, we must not forget that certain chemical substances can produce the disease without the assistance of micro-organisms. Experimentally there is ample evidence on this point. Injections of croton oil, turpentine, and silver nitrate into the peritoneal cavity of animals have produced acute inflammation, sometimes of a hæmorrhagic type.

Non-bacterial peritonitis in the human subject is met with in the rupture of ovarian and other abdominal cysts; also in torsion of the pedicle of an ovarian tumour. A few cases have been reported of non-bacterial peritonitis caused by torsion of the spleen. In these cases no organisms can be found, at any rate in the early stages; of course, later on, when necrosis of tissue sets in, the bacterial invasion of the peritoneal cavity proceeds rapidly.

BACTERIOLOGY.—Although there is a great similarity in the pathological appearances and clinical signs of cases of acute peritonitis, yet the kind of organism exerts a very definite influence, and therefore it is important to consider the germs usually associated with peritonitis.

1. *Micrococci*.—(*a*) *Streptococci* are found in a large proportion of cases: according to Tavel and Lanz 75 per cent. (*b*) *Staphylococci* are seldom met with alone. As a rule they are associated with streptococci. (*c*) *Pneumococci* are found alone in about 2 per cent. The peritonitis in these cases tends to terminate favourably after flushing of the

peritoneal cavity. (*d*) *Gonococci*. There is no doubt that pure cases of peritonitis from the gonococcus do occur. The majority of cases in which gonococci are found are cases of mixed infection, streptococci playing a large part.

2. *Bacillus Coli Communis*.—Of the various germs found in the contents of the intestine, the *B. coli communis* is the most conspicuous. It is found in nearly all cases of 'perforative peritonitis,' that is, where the wall of the alimentary canal, whether stomach, intestine or vermiform appendix, is ruptured. Until quite recently the *B. coli communis* was regarded as the principal pathogenic organism in nearly all inflammations of intestinal origin. Tavel and Lanz showed that there were numerous species of this organism, which must be looked upon as a genus; and they threw doubt upon its being the cause of so much trouble. Veillon of Paris has produced good evidence for looking upon the *B. coli communis* as playing only a subordinate part, and has proved that certain anaerobic bacteria, which until now have not been properly isolated, are the true cause of many of the forms of appendicitis, especially those associated with acute gangrene, and those cases in which fetor and the formation of gas are special features. Veillon's work seems thorough and reliable and rids us of the great difficulty of explaining how a usually innocuous germ of the intestine like the *B. coli communis* should, without any apparent cause, suddenly develop such malignant properties. These anaerobic germs have been shown to be very destructive in their action upon tissues, causing intense inflammation resulting in more or less gangrene.

MODES OF ENTRANCE OF BACTERIA. —

1. *Wounds*, including (*a*) penetrating wounds of the abdominal wall, with or without injury to the viscera, and (*b*) injury to the viscera without any wound of the abdominal wall. 2. *Perforation by a foreign body*, including (*a*) those formed in the body, such as gall-stones, and enteroliths, &c., and (*b*) those taken in by the mouth. 3. *Operations on the abdomen*. 4. *Internal strangulations*, including volvulus, intussusception and internal hernie. 5. *Extension from an inflamed organ*, especially the intestine and vermiform appendix. 6. *Extension from a local abscess*, of which the most common forms are suppurative appendicitis and suppurative salpingitis. 7. *Perforation of hollow viscera*, as in gastric or duodenal ulcer, typhoid ulcer, &c.

Of these causes peritonitis, following surgical operations upon the abdomen, is becoming much rarer than formerly owing to improvements in operative technique. By far the most common cause in both sexes is appendicitis.

MODE OF ACTION OF THE BACTERIA.—The peritoneum presents a very large surface, being roughly equal in extent to the surface of the skin. If a normal saline solution be injected into the peritoneal cavity of a living animal, about one-tenth of the body weight is absorbed in an hour, or $2\frac{1}{2}$ times the body weight in a day; thus a man of 12 stone would absorb about 14 pints of saline fluid in one hour or 18 gallons in a day.

Conversely, if a substance be injected into the peritoneal cavity which leads to transudation, such as a concentrated solution of sugar, about $\frac{1}{10}$ of the body weight in fluid is exuded per hour. It is easy to imagine how rapidly the toxins elaborated by some organisms would be absorbed, and to account

for the very rapid onset of symptoms of septic poisoning in cases of peritonitis. In very acute cases the patient rapidly passes into a state resembling shock. It is not known whether this condition is due to the absorption of very rapidly formed toxins, or whether the inflammation of so extensive an area as the peritoneum may not produce, by reflex nervous action, a state similar to that of shock. We know that an acute inflammation of the whole of the skin—and it has already been pointed out that the area of the peritoneum is about equal in extent to that of the skin—has a profound influence upon the patient very much resembling that of shock.

PATHOLOGICAL ANATOMY.—The earliest sign is increased vascularity of the coils of the small intestine; the peritoneum covering them is apparently more readily attacked than that over the large gut. Almost synchronously with this vascularity the intestine begins to dilate, and when this dilatation becomes extreme, we have the condition termed *tympanites*. As the distension increases the coils become pressed together in lines more or less parallel with the long axis of the bowel. The vascular dilatation, which at first is uniform, becomes more marked along the lines of least pressure, which is usually in the triangular space where three coils meet. At this stage the peritoneal fluid is diminished, being absorbed probably by the enormously dilated capillaries and veins, so that the surface of the gut may still be shiny, although it is distinctly dry.

This condition so far is present in all cases, and it depends upon the further progress of the inflammation what the ultimate condition may be. Broadly speaking, the subsequent inflammation produces one of three conditions.

1. *Adhesive or dry peritonitis.*—The peritoneal fluid is absorbed owing to the enormous dilatation of the vessels, and when inflammatory exudation is poured out the fluid part is absorbed, leaving masses of solid exudation matting the coils together. In cases that resolve quickly this lymph soon organises or may become to a large extent removed by wandering cells from the peritoneum. If present in considerable amount, adjacent coils are temporarily united together; but the constant peristalsis gradually stretches the adhesions, which may finally break loose at one end. It is remarkable how quickly these adhesions disappear, a fact which surgeons often have an opportunity of observing on performing a second operation upon the abdomen. It is seldom that more than one adhesion exists; hence the curious phrase 'the solitary band of Gay,' so called after the surgeon who first described these bands and who pointed out how they were as a rule found single. When the exciting agent relaxes its activity slowly the enormous masses of adhesions which result may so distort the relative position of the viscera as to justify the term *peritonitis deformans*. The writer is inclined to believe, however, that these cases are of slow formation from the first and are really examples of chronic peritonitis.

2. *Serous.*—Here we find that there is a definite exudation of serum in addition to the 'lymph.' The amount of this fluid may be quite small or may reach the proportions of a good-sized ascites. Like all inflammatory exudations, its appearance varies in proportion to the intensity of the inflammation from a richly blood-stained to a clear liquid. This condition is more often met with in chronic peritonitis. See p. 1201.

3. *Suppurative.*—There is no sharp line of demarcation between the turbid serum of the former variety and the kind of fluid which is sufficiently opaque to deserve the term 'pus.' It is often difficult to decide whether to call the exudation 'turbid serum' or 'pus.'

PATHOLOGICAL HISTOLOGY.—In the acute stage of peritonitis the inflammation as seen through the microscope differs in no respect from acute inflammation elsewhere. The classical descriptions of early inflammation could be accurately applied to peritonitis. A good many experimental studies have been made of the minute changes in peritonitis. Some observers state that the endothelial cells of the serous membrane play a definite part in the inflammatory processes; that they proliferate in considerable numbers; that they pass into cubical and spindle stages, and into large granulation cells, and that they take the chief part in the formation of new tissue; in other words, fibroblasts are looked upon as descendants of proliferated endothelial cells. On the other hand, other observers are strongly convinced that the new tissue is derived from connective-tissue cells already existing just beneath the pavement-cells, and that the adhesions are formed solely from these sub-epithelial cells, the pavement-cells being destroyed in the process of inflammation.

SYMPTOMS.—When we consider the numerous causes of acute peritonitis it is not surprising that symptoms vary exceedingly in different cases. Sometimes we are confronted with a patient whose appearance suggests typical surgical shock; in another case the local evidences of peritonitis are at once obvious; in yet a third the symptoms generally may be so masked that the most experienced clinician may miss the fact that the central feature of the case is peritonitis. There is no definite line of demarcation between those cases which die within twenty-four hours with symptoms of general saphæmia, and those in which the peritoneal symptoms form so prominent a feature that attention may be withdrawn from the fact that the patient is also suffering from the effects of general septic poisoning. The great majority of cases in which the peritoneum is inflamed present, at first, symptoms strongly suggestive of general peritonitis; yet after a day or two it is quite obvious that the case is one of local inflammation. This class of case is especially met with in cases of appendicitis and salpingitis. Many authorities consider that in these cases the general peritoneum is inflamed at first, but that its protective mechanism has checked the onslaught of the micro-organisms except at the local spot where the headquarters of the germ are situated. In the opinion of the writer the following two cases afford reasons for believing that this is not the only explanation. In each case the patient was a girl eighteen years of age, presenting no previous trouble except a recently developed anæmia; each was taken ill suddenly with marked abdominal pain of the 'doubling up' character, associated with vomiting; in each case there was excessive rigidity and quiescence of the abdominal wall, and in each case pain was complained of chiefly in the epigastrium. One patient had passed on the day preceding and on the morning of the attack a copious stool containing altered blood. The diagnosis of a ruptured gastric ulcer was considered the most likely one. In each patient a median exploratory incision was made above the umbilicus and the whole upper abdomen examined carefully;

there was no sign of inflammation. In one case a second incision was made below the umbilicus, and again no evidence of any inflammation could be found in the abdominal or pelvic organs; there was some slight thickening in the right iliac fossa, and keeping in view the great probability of appendicitis, no attempt was made to pull the cæcum and appendix into view, but the wound was at once closed. On the sixth and eighth days respectively a large abscess in the right iliac fossa was opened. This obviously originated in the appendix. Both patients recovered. The clinical signs of apparent early general peritonitis in such cases as these are, in the opinion of the writer, due to the spread of reflex nervous action, as described in detail later in this article.

There are two classes of cases which may be taken as types of a general peritonitis, viz. that following operations upon the abdomen and that following an appendicitis where the micro-organisms get through the wall of the appendix without necessarily an actual perforation of the wall of the tube taking place. In other words, it may be a bacterial perforation only. The former cases are happily becoming more and more uncommon, and therefore it will be of greater use for us to consider the latter as forming the most common type of general peritonitis. There are the very acute cases before referred to in which the patient appears to be suffering from profound shock and poisoning. In another group of cases the symptoms of peritonitis are slight when compared with the mischief that is found at the operation or autopsy. These cases are termed *latent* or *masked* peritonitis.

A typical case of acute peritonitis.—On a certain day the patient begins to feel unwell; within an hour or two he is cold and may shiver; fairly often there is an actual rigor ushering in the symptoms. The first definite symptom that makes its appearance is nearly always pain which, though very constant in its presence, is very variable in its character and in intensity. This pain, when slight in degree, has the peculiar quality of worrying the patient out of all proportion to its intensity, and confers upon the face a characteristic expression of anxiety. On the other hand, the actual intensity of the pain may be so great that it is quite alarming both to the patient and his friends. At first the pain is referred to the whole of the abdomen, but later on it is generally localised to the region of the umbilicus.

As a rule the abdomen is rigid, and the skin of the whole of or a part of the abdomen is markedly hyperæsthetic. In order to diminish the abdominal tension as much as possible the patient lies on his back with the thighs flexed and the shoulders raised, and later on in order to increase the 'vital capacity' of the thorax the arms may be raised above the head. Owing to this rigidity of the abdominal muscles the respirations become more and more costal in type. The pulse is increased in frequency, is of high tension and small; indeed, the tension is so considerable that this, together with the diminished size, has gained for it the term *wiry*. The temperature soon rises to 103° or 104° ; after the initial rise it keeps at an average of 101° to 102° .

After the first twenty-four hours the hard, somewhat retracted, abdomen becomes distended, and the percussion-note becomes increasingly tympanitic. Distension rapidly increases so that the abdomen

becomes blown out and tense. Vomiting usually sets in about the same time as the pain, and is a prominent feature of the disease. At first the ordinary stomach-contents are ejected; then the fluid is yellow, being distinctly bile-stained; and this is followed by a characteristic green vomit, which is finally succeeded by a brownish-black liquid having an offensive odour distinctly faecal in character. At the onset the bowels may be loose, but as soon as the abdomen distends constipation sets in. The act of micturition is often frequent at first, but later on there may be retention, especially if the origin of the trouble is in the appendix or in the pelvic organs. The urine is small in quantity and high-coloured, usually containing abundance of indican (p. 727).

When all the symptoms are fully developed the aspect of the patient is quite characteristic; so well was it described by Hippocrates that it is often termed '*facies Hippocratica*,' viz. 'a sharp nose, sunken eyeballs and hollow temples, the ears cold and pinched with their lobules turned outwards; the skin of the forehead is rough, puffy and dry; the colour of the whole face being brown, black, livid or leaden in colour'; this description requires modification only in the last statement, where we must remember that the face described belonged probably to the oriental type.

There is usually free fluid causing dulness in the flanks, which may shift on rotating the patient. This typical case of acute peritonitis usually lasts from five to ten days, nearly all the symptoms increasing in severity until the fatal end, which is usually heralded by the skin of the face and hands becoming cold and clammy and tinged with a dark blue colour.

The onset.—As a rule this is quite sudden, but the time relations of the onset are largely determined by the actual cause of the general invasion. If it is due to the perforation of the stomach, intestine or appendix, without any previous inflammation of the peritoneum, the onset comes with alarming suddenness. Within a minute the patient passes from a condition of apparent health to a condition of collapse and shock that has been known to end fatally within an hour.

In other cases where an organ, generally the appendix, has been inflamed for a long time the invasion of the general cavity is comparatively speaking gradual, and it may take twelve to twenty-four hours before it can be said that acute general peritonitis is definitely established. When very acute the onset is associated with a rapid fall in temperature; in other cases there may be a rigor and a temperature of 104° or 105° within an hour or two of the commencement of the attack.

Shock (Peritonism).—The shock produced by an extensive scald of the skin, or by the skin being badly lacerated, is extreme, and bears a very definite proportion to the area of skin involved. In the same way any extensive lesion of the peritoneum produces a similar condition, whether it be due to the irritation of the contents of the stomach, as in perforation of a gastric ulcer, internal strangulation, or the torsion of an ovarian pedicle.

Pain.—This is nearly always the earliest symptom, and may vary from a pain that is accurately described by the term '*agonising*' to a pain slight in intensity but of such a character that the aspect of the patient shows considerable anxiety, as mentioned above. At first the pain is generalised over

the whole abdomen, but later on it is usually referred by the patient to some definite area; very often the region of the umbilicus seems to be the most painful. In cases where inflammation of a definite organ has given rise to the peritonitis, the pain may be referred to the region of the affected organ, especially in the case of the appendix.

Tenderness.—This tenderness may be of two kinds—(1) *Superficial tenderness*, or hyperæsthesia of the skin; (2) *Deep tenderness*, which may reside in the muscles, peritoneum or underlying viscera (see p. 1146). When the superficial tenderness is very extensive it is probably associated with inflamed intestines. The deep tenderness, on the other hand, is due to the direct pressure upon the peritoneum, and is obtainable at any spot where the inflamed peritoneum can be pressed upon. In general peritonitis this deep tenderness, therefore, can be elicited practically over the whole of the abdomen, with the exception of the area posterior to a line drawn from the anterior superior iliac spine to the tip of the last rib, where it is not easy to press directly upon the peritoneum. In the early stages of general peritonitis the superficial tenderness is nearly always extremely well marked. On Head's hypothesis this would mean that the small intestines or some other of the viscera were distinctly affected, and this superficial tenderness would not necessarily be limited to the front of the abdomen, but there might be a band of hyperæsthesia running to the back or downwards towards the groin (see p. 1142). As the peritonitis is becoming established the superficial tenderness becomes much diminished, and may finally disappear, whereas the deep tenderness progressively increases until the general peritonitis is well developed. In nearly all cases of general peritonitis some organ of the abdomen is first affected, and this organ may continue to present its referred superficial tenderness after the general peritonitis has become established. In several cases of general peritonitis arising from appendicitis the writer has found an extensive area of superficial tenderness below the level of the umbilicus co-existing with the deep tenderness, and as the operation advanced general peritonitis has been found. On this point, therefore, we may make two positive assertions, namely, that if the tenderness is only superficial there is no marked affection of the peritoneum; and that in cases of undoubted suppurative peritonitis due to appendicitis there may be superficial tenderness existing at the same time as the deep.

Vomiting.—Next to pain this is the earliest, most constant, most distressing and most persistent symptom. The abdominal muscles take little if any part in the expulsive act, so that the stomach itself does most of the work; hence the act of vomiting appears to occur without much effort. In the early stages the vomiting is principally due to a poisoning of the centre in the medulla oblongata. After paralysis of the small gut has set in it much resembles the vomiting of intestinal obstruction.

Defecation.—During the early hours of the attack there is very often a stool which is generally described as being loose; diarrhoea is not uncommon. A frequent desire to defæcate without any result is sometimes met with, and this may be so intense as to deserve the term 'tenesmus.' As soon as the distension of the small gut sets in the colon becomes inert and constipation is the rule in the fully developed peritonitis.

Distension of the Abdomen (Meteorism).—As the intestinal coils become paralysed and distended, and the abdominal muscles begin to relax, we get the condition of meteorism, which gradually increases until the abdomen is much distended. This gradual distension of the abdomen is undoubtedly diagnostic of acute general peritonitis, and in the opinion of the writer it is a certain indication that no delay should be allowed before opening the abdomen.

Temperature.—This is the most variable symptom of all, so variable indeed that it is safer for the practitioner to place no importance whatever upon this symptom so far as any guidance concerning diagnosis and treatment is concerned. In the sudden perforation the temperature is usually subnormal and often remains so until the fatal termination.

METHOD OF EXAMINATION.—The patient should be placed on his back with the legs extended and the arms by the side of the body. The body is uncovered to such an extent as to show the apex-beat of the heart above and Poupart's ligament below. As the examination proceeds as much of the body should be covered up as is possible. The character and areas of tenderness should be carefully ascertained. For the details of the examination see ABDOMEN, Examination of.

The patient should always be examined *per rectum* or *per vaginam* to ascertain the condition of the pelvic organs.

CLINICAL VARIETIES.—*Peritonitis Fulminans; Ultra-acute peritonitis.*—In these cases, usually caused by the perforation of some part of the alimentary canal, in which no adhesions have formed from previous inflammation, the patient is suddenly seized with most acute pain and, within a few minutes, is suffering from the most profound shock. As the bacterial invasion proceeds symptoms of septic poisoning rapidly develop, and the patient dies within 24 to 48 hours without having rallied sufficiently for operative interference to be seriously entertained.

There is a group of cases following operation upon the abdomen, now happily becoming less and less common, in which the patient never really recovers from the shock of the operation, but dies within 24 to 48 hours with a rather distended abdomen. On *post-mortem* examination there is scarcely any evidence of inflammation of the peritoneum, certainly not enough to justify us in saying that the patient dies of peritonitis. Most surgeons consider they are justified in looking upon this result as death from shock, and consider that the intestinal distension is due to some special reflex nervous mechanism. Some pathologists, on the other hand, have found the peritoneal cavity to be infected with micro-organisms, and they look upon these cases as instances of a very rapid invasion of the peritoneum by virulent bacteria which grow so rapidly that inflammation has no time to develop. A definite series of accurate bacteriological investigations alone can settle this point.

Latent or masked peritonitis.—The patient walks into the hospital and does not feel particularly ill; he usually states that his illness commenced four or five days previously, but that until that particular day he was not ill enough to seek advice. Within two or three days he suddenly becomes collapsed and dies with his peritoneal cavity full of pus. Owing to the absence of urgent symptoms the

surgeon's help is often not summoned until the patient is too collapsed for operative interference to be anything more than a forlorn hope.

Peritonitis of the new-born child.—These cases are due to one of two causes: (1) the septic infection of the umbilical cord, due probably to faulty technique on the part of the accoucheur or midwife; (2) rupture of the distended large intestine, which may be due to the pressure of parturition, but more likely to the giving way of some small ulcer in the colon.

Peritonitis caused by cutaneous erysipelas.—In rare instances erysipelas of the abdominal wall has been followed by fatal peritonitis. This is difficult to explain because the lymphatics of the abdominal wall do not communicate with those of the peritoneum. The symptoms of the peritonitis are often masked and not even suspected during life.

Peritonitis in the course of acute specific fevers.—Peritonitis is most commonly met with in scarlet fever, as we might expect from the tendency of this disease to develop dangerous complications. In typhoid fever it may occur as the direct result of a perforating ulcer or be due to a bacterial penetration of the wall of the alimentary canal without any physical breach of continuity. Peritonitis has also been met with in the course of measles, small-pox, and typhus fever.

Rheumatic peritonitis.—Although doubt has often been thrown upon the existence of this complication of rheumatic fever, it is definitely proved that acute inflammation of the peritoneum does sometimes occur as a complication of acute rheumatic fever. It usually terminates favourably after a few days, although fatal cases have been reported.

Peritonitis in Bright's Disease.—This is a rare complication. The mode of origin of the peritonitis in this disease as in the case of acute rheumatic fever is possibly as follows: A purely chemical peritonitis is set up by toxins. After a time the wall of the intestine is sufficiently injured by this chemical peritonitis to allow the organisms contained in the intestinal contents to work their way through to the peritoneal cavity (p. 221).

Idiopathic or 'cryptogenetic' peritonitis.—The more carefully cases are examined by improved modern methods the rarer does the diagnosis of idiopathic peritonitis become. At present there are two groups of cases of which the origin is absolutely obscure, and hence it is convenient to place them under this heading. In the one group the patients are children, chiefly girls, and in the other the victims are adults. In both these groups a 'chill' is generally quoted as being the cause. The attacks of enteritis with diarrhoea so commonly met with in Europeans living in hot countries, especially in Egypt, seem to be due solely to some reflex vaso-motor disturbance from the sudden chilling of the body in an atmosphere nine times drier than the one they have been accustomed to. The cooling of the body by means of perspiration is therefore rapid, the atmosphere being very hygroscopic, so that a reflex effect is produced causing a dilatation of the vessels of the splanchnic area. If this condition is neglected it is easy to see how some inflamed area of the gut may prove a weak spot and allow the germs to penetrate to the peritoneal cavity.

DIAGNOSIS.—The following conditions may simulate peritonitis:—

1. *Acute intestinal catarrh (Enterocolitis), in*

which diarrhoea and intermittent colic are the chief symptoms.

2. *Intestinal obstruction.*—The differential diagnosis between peritonitis and this condition may be impossible for the first two days and sometimes throughout the disease. See pp. 774–787.

3. *Hysterical peritonitis.*—A condition met with on the Continent more frequently than in this country.

4. *Certain rare conditions*, such as acute hæmorrhagic pancreatitis, or a ruptured tubal pregnancy. See p. 1151, and PREGNANCY, Diseases of.

TREATMENT.—It is an admitted fact that an acute general peritonitis once established and allowed to run its natural course is, except in rare instances, fatal. Moreover, the only method of treatment which gives any practical chance of recovery is operative or surgical. The surgeon, having once satisfied himself that the patient is suffering from acute general peritonitis, has only to decide one point—whether the patient is in a condition to withstand operative interference. To do this he has to consider first of all whether a thorough attempt can be made under a general anæsthetic; in this event, laparotomy, flushing, and drainage of the peritoneal cavity must be promptly carried out. But if he think that the patient cannot stand this ordeal it may be possible to anæsthetise the patient for a short time so that an abdominal section can be rapidly performed and a drainage-tube and gauze introduced. In extreme cases a general anæsthetic may be quite out of the question, and the surgeon must then attempt to do what is necessary with a local anæsthetic. In the experience of the writer, patients who are extremely ill with peritonitis can withstand a rapidly performed abdominal section with local anaesthesia better than might be expected. The details of these operative procedures are as follows:—

1. *Laparotomy, flushing, and drainage.*—The patient being under the influence of ether or chloroform, a free incision is made in the middle line below the umbilicus. There is a considerable diversity of opinion among surgeons concerning the best anæsthetic for abdominal work. From the surgeon's point of view alone it seems to the writer that chloroform is the best both for the rapid and comfortable performance of the operation, and for the absence of the pulmonary disturbances so common after ether. From the anæsthetist's point of view, which is concerned solely with the patient's safety during the actual operation, the consensus of opinion in London is strongly in favour of ether (p. 61). With a competent 'chloroformist' the writer considers, however, that the safety of the patient during the operation and the few succeeding days is greater with chloroform than with ether. A rapid search is made for the seat of origin of the trouble. If the appendix is the offending organ, an incision should be made at a spot over it most convenient for its removal, and, if possible, the appendix is excised. All signs of local suppuration should be washed and sponged away. The part of the abdomen in the region of the appendix, especially the cavity of the pelvis, should be well flushed out with normal saline solution at the temperature of the body. An indiarubber drainage-tube is placed in contact with the spot where the disease originated, whether appendix, Fallopian tubes, or elsewhere, and around this tube some iodoform-gauze should be packed. The median wound should then be

closed, and one or two stitches placed in the lateral incision so as to prevent prolapse of intestine, and at the same time not to interfere with effective drainage. If no local focus can be found, the abdominal wound may be closed after thoroughly flushing the peritoneal cavity.

Some details concerning the *technique of drainage* must be borne in mind. If a tube be inserted, say, six inches from the skin down towards the pelvis, and carefully examined at an autopsy twenty-four hours afterwards, it will be seen that the tube is surrounded by coils of intestine which are sealed together with exudation which completely prevents the tube receiving any appreciable amount of the general peritoneal fluid. The surface of the coils surrounding the tube exudes the turbid serum which fills the test-tube-like cavity. As soon as this liquid is removed by a syringe or by gauze the coils gradually exude another quantity. Unless the tube reaches directly down to a septic focus it seems to be of little use in draining the general peritoneal cavity. By leading down to a septic focus it helps to form a well-protected channel for the exit of any discharge from the suppurating focus. Drainage-tubes, whether made of glass or indiarubber, are quite useless as agents for draining the general cavity of the peritoneum, even when present in numbers; and indeed may do harm by mechanically irritating the coils of intestine. The sole use for a drainage-tube is to obtain a channel through the coils of intestine by which some septic focus can discharge its contents to the exterior with ease. There is one exception to this rule, and that is in the case of the pelvis. Here there is naturally a tendency for fluid to collect in a definite quantity, so that a tube reaching to the bottom of the pelvis must assist in draining that cavity. So important is the draining of the pelvis that some surgeons prefer to drain Douglas's pouch in women by an opening through the vagina. This proceeding, however, necessitates additional shock, which is an important factor in cases of acute peritonitis, and its necessity is doubtful if efficient drainage is secured by a tube and gauze from above.

One of the most disappointing features in the surgical treatment of general peritonitis is that after removing the focus or origin of the trouble and thoroughly flushing the abdomen, without markedly depressing the patient, there is a tendency to distension of the intestine, especially the small gut. The reasons for this distension have been already given, and it is chiefly to the late Greig Smith that we are indebted for pointing out how very important it is to drain the inside of the alimentary tube as well as the peritoneal cavity. If the intestines are much distended one of the most distended coils should be incised, and a T-shaped glass tube inserted. In these desperate cases the first point is to save the patient's life; in the event of recovery the artificial anus can be easily closed by modern methods.

2. *Rapid laparotomy with drainage.*—The patient is quickly anaesthetised, and as soon as the abdominal wall is flaccid a median incision is made and any inflammatory exudation is allowed to escape; if rapid examination reveals the focus of origin a tube and gauze-drain are inserted down to the suppurating spot. If the seat of origin cannot be found a gauze-drain may be left in the peritoneal cavity and the wound partially closed. If the intestine is much dilated the first distended coil should

be opened, and a T-shaped glass tube inserted as described above.

3. *Laparotomy with local anaesthesia.*—If the patient is too bad for the administration of a general anaesthetic, the skin of the abdomen in the middle line should be rendered anaesthetic with *β*-eucaine (see ANÆSTHESIA, LOCAL). As much should be done as the patient can bear under the conditions of local anaesthesia.

In no department of surgery is it more important for the surgeon to form his opinion with decision, to commence operation without delay, and to operate quickly. Nothing is more certain than that every additional minute of exposure in the case of an abdominal section diminishes the patient's chances of recovery.

After-treatment.—(1) If the patient is restless after the operation, a hypodermic injection of morphine (gr. $\frac{1}{4}$) and atropine (gr. $\frac{1}{150}$) should be given. (2) It is so important to keep the alimentary canal at rest that the introduction of food into the stomach should be prohibited. All food should be given by the rectum; perhaps the best is peptonised beef-tea and small quantities of brandy. This is quite sufficient to tide the patient over the crisis of the disease. As a rule, if a patient is alive on the fourth day after operation he will recover. (3) Thirst is best satisfied by injections of warm water *per rectum*. If the patient craves for something by the mouth, a little warm water, or, if much preferred, a little iced-water may be given. In cases of excessive thirst a feeder full of water (5 oz.) may be given. If this is vomited no great harm is done, providing it is not frequently repeated. (4) After the operation no aperient should be given for the first twenty-four hours. As soon as the patient has rallied sufficiently from the operation, and if there is distension of the intestine, an enema of water (a pint) combined with tincture of valerian (a drachm) should be given. If at the end of twenty-four hours the patient is improving, small doses of sulphate of magnesium and sodium may be given, say \mathfrak{zj} of each every three hours, or small doses of calomel (gr. $\frac{1}{2}$) every three hours.

The administration of opium and morphine.—Before the question of operative treatment has been discussed no opium or morphine in any form should be given. The administration of this drug causes an immediate improvement in the symptoms: the pain is diminished, the rigidity of the abdominal muscles may disappear altogether, and the patient's general condition is so much improved that the surgeon may be quite deceived as to the gravity of the case. Many lives have been lost owing to the false appearance of improvement brought about by the administration of this drug (p. 780). The patient's improvement may be maintained until suddenly the dose of poison absorbed from the peritoneal surface reaches its maximum, and the patient suddenly collapses. If the case is considered too bad for operative interference then morphine may be given until the patient is quite relieved of all distressing symptoms. Morphine again may be very useful in stimulating the heart and quieting the nervous system after an operation has been performed. In these cases, however, it should be given with especial care that no inertia of the intestines is produced.

A method of treatment which has gained considerable ground on the Continent, especially in France, is the subcutaneous and intra-venous injection of

saline solution. The principle is to introduce into the vascular system, directly by the veins or indirectly by the subcutaneous lymphatics, a saline solution with a view of washing out the lymphatic vessels and blood-vessels, and so enabling the liver and kidneys to get rid of the toxins. The solution generally used is sodium chloride 5 grms., sodium sulphate 10 grms., distilled water 1 litre. This is injected at the temperature of the body either in the subcutaneous tissue or through the median basilic vein (*see* SALINE SOLUTION, Infusion of). According to French surgeons remarkable improvement and cures have been attained. This treatment seems perfectly rational when used in conjunction with operative interference as recommended by some French surgeons. It is really very similar to the 'infusion' that we often perform upon such patients who are collapsed after the operation. The amount of fluid injected averages about 5 litres in the course of twenty-four hours.

Non-operative treatment.—According to our present knowledge there is no method of attempting to cure a case of acute general peritonitis by medicinal means. In cases too much collapsed for surgical interference the treatment must be directed to the alleviation of all distressing symptoms.

HENRY PERCY DEAN.

PERITONITIS, Acute Localised.—Acute inflammation of a portion of the peritoneum due to some lesion of the organ which it is covering is of very common occurrence. It may occur in the peritoneum covering the liver and spread thence to the peritoneum lining the lower surface of the diaphragm. It not infrequently occurs in the peritoneum covering the stomach in the neighbourhood of a gastric ulcer. Of all the abdominal organs the vermiform appendix is the one most commonly affected. In women there is frequently local peritonitis around the uterus and Fallopian tubes, usually termed 'pelvic peritonitis.' The chief varieties we will consider at greater length.

1. Sub-phrenic Abscess.—Inflammatory conditions may spread from the pleural cavities through the diaphragm to the abdomen. As a rule, however, sub-phrenic peritonitis spreads from the abdominal organs, chiefly the liver and stomach, often developing into an abscess. When the inflammation spreads from the liver and suppuration occurs, the abscess forms beneath the right half of the diaphragm, and is very often diagnosed as an empyema. On the other hand a sub-phrenic abscess caused by lesions of the stomach forms on the left side.

The symptoms and diagnosis of sub-phrenic abscess are considered in the article on STOMACH, Perforation of, and its Results.

TREATMENT.—As soon as pus is suspected in the region of the diaphragm an exploring needle should be used (*see* PARACENTESIS). If the result is positive some indication is obtained as to the situation of the pus and its depth from the surface. If the needle gives a negative result the surgeon must proceed to make a careful dissection down to the diaphragm in the region of its attachment to the ribs. Very often the pleural cavity overlaps the displaced diaphragm to the extent of several inches. In such cases, if the condition of the patient will allow, the lower end of the parietal pleura may be stitched to the chest-wall, and the wound plugged with iodoform-gauze for 24 or 36 hours until adhesion

has formed to a sufficient extent to allow the surgeon to cut straight through the diaphragm. If after removing a piece of rib it is seen that the pleural cavity is normal the surgeon should extend his dissection in a downward direction, until the point of reflection of the pleura is seen. The pleura may then be pushed up, and gauze packed tightly in the upper part of the wound. If immediate evacuation of pus is necessary the diaphragm may be incised, the pus quickly removed, and the edges of the incised diaphragm stitched to the edges of the wound. The risk of infecting the pleural cavity is by no means a light one: it is not due so much to the pus escaping into the pleural cavity at the time of the operation as to the soaking of pus into the surrounding tissues within the few hours succeeding the operation, especially if drainage from the abscess cavity is not quite free or if the upper part of the wound, in the region of the pleura, is not carefully and thoroughly packed with iodoform-gauze. There is one consolation for the surgeon, viz. secondary infection of the pleura is common in sub-phrenic abscess, even before operation, so that if the germs have reached the pleura before the surgeon, nothing the latter can do is likely to prevent the development of an empyema. The following rules may be worth remembering in dealing with the surgery of sub-phrenic abscess: (1) To make the incision over the spot where pus has been found by the needle; (2) if no pus has been obtained by the needle to make a careful exploratory operation through a four-inch incision over the tenth rib in the mid-axillary line; (3) if no great urgency, to pack the exposed pleura or pleural cavity with sterilised gauze and wait 24 to 36 hours before incising the diaphragm; (4) if evacuation of the pus is urgent, to try and expose the lower reflection of pleura, push it upwards from the ribs and pack tightly with gauze, incise the diaphragm and stitch it to the skin, insuring free drainage.

2. Abscess around Appendix.—Owing to the inconstant position of the appendix the situation of the abscess is, as a consequence, variable. Usually it is in the right iliac fossa with the cæcum on its outer side. Sometimes it is wholly within the pelvis. It may be to the left side of the median line, in the iliac region. An important situation is *sub-mesenteric*; the abscess being limited above by the oblique line of the mesenteric attachment, viz. from the right sacro-iliac synchondrosis to the left side of the second lumbar vertebra—below it reaches the hollow of the sacrum, being variably limited by coils of the small gut; to the right side is the cæcum; in front the mesentery itself and numerous coils of gut form the boundary, so that the general peritoneal cavity is exposed after cutting through the abdominal wall.

TREATMENT.—When there is a dull area in the iliac fossa the incision should be made over the spot of maximum intensity. The cutting should be done carefully so that any weak adhesions may be left in order to keep the pus from spreading. In all cases the whole of the pus should be removed at once and the cavity thoroughly washed out. If the appendix can be easily got at, it should be excised; as a rule, however, it forms a part of the abscess-wall and cannot be felt or seen. There is often a faecal concretion present which usually escapes through the gangrenous aperture in the appendix, and it is important to try and get hold of this;

this may often be attained by the gentle use of a blunt Volkmann's spoon. If the whole of the pus is evacuated, a large drainage-tube inserted and surrounded with iodoform-gauze, the cases usually do well without any infection of the general peritoneal cavity. When the pus is chiefly in the pelvis and can be felt *per rectum*, a good plan is to reach the abscess *via* the right ischio-rectal fossa. The skin is incised midway between the margin of the anus and the ischial tuberosity, and the tissues of the fossa are incised until the levator ani is reached. This may be penetrated with a pair of sinus-forceps, and the opening dilated to allow the passage of a large drainage-tube. The writer has found this plan very successful in a case in which the pus was high up and gave no indication of its proximity by any alteration in the tissue of the ischio-rectal fossa. See also APPENDIX VERMIFORMIS, Inflammation of, p. 93.

3. **Pelvic Peritonitis and Abscess.**—This may start in the uterus or Fallopian tubes, usually after parturition. The tubes are most often the infecting focus. These abscesses are often opened *per vaginam*. See p. 1200.

HENRY PERCY DEAN.

PERITONEUM, Chronic Inflammation of.
SYNON. : Chronic Peritonitis.—This affection, like the acute form, may involve the peritoneum more or less generally; or only over a localised and limited area. The conditions included under the term are somewhat indefinite, but not uncommonly they are well marked pathologically, as well as of considerable clinical importance.

ÆTIOLOGY AND PATHOLOGY.—Without entering into details, it must suffice to point out the circumstances under which chronic peritonitis may occur: (1) There is no doubt as to its being, though rarely, a sequel of one or more attacks of acute or subacute peritonitis, either general or local, but especially the latter; and after a circumscribed acute peritonitis chronic changes may gradually spread more or less widely. Moreover, the conditions remaining after an attack of peritonitis are liable to set up further mischief of a chronic nature. (2) Chronic peritonitis may become associated with ascites, but more particularly when repeated paracentesis has been performed for the relief or cure of this condition. In some of these cases probably the ascites is the result of a simple chronic peritonitis. (3) Localised chronic peritonitis is common as the result of continued irritation, associated with some diseased organ, such as a cirrhotic or cancerous liver, cancer or chronic ulcer of the stomach, old hernie, tumours, and various other obvious conditions. There are, however, cases occasionally observed in which the cause is not so evident, and these have been attributed to irritation by accumulation of fæces, or to repeated pressure or other mechanical causes acting from without. Extensive chronic peritonitis also starts occasionally from a local centre, especially perihepatitis. (4) Morbid processes in the peritoneum itself are very liable to set up chronic inflammation. Of these the principal are tuberculosis and cancer; and tubercular and cancerous peritonitis constitute important forms of this disease. (5) In rare instances a chronic inflammatory effusion collects in the peritoneal cavity without any obvious cause. The fluid is generally serous, and cannot be distinguished from that of ascites. This chronic effusion has been noticed

during convalescence from fevers. Distinct chronic peritonitis also sometimes occurs as an independent disease, and has then been referred to chronic Bright's disease, alcoholism, gout, rheumatism, lead-poisoning, cardiac disease, and other causes.

ANATOMICAL CHARACTERS.—Adhesions or fibrous thickenings connected with the serous membrane are almost constantly present in different degrees, and not infrequently they constitute the sole anatomical evidences of chronic peritonitis. They result from the development of the inflammatory products, and the formation of connective or fibrous tissue, with new vessels. The thickening varies much in degree, ranging from what is scarcely perceptible, to the production of a dense fibrous mass an inch or more in thickness, as the writer has seen. It may be evident in the parietal peritoneum; around organs, forming more or less thick and firm capsules; or in the peritoneal folds, especially the omentum and mesentery. Adhesions or agglutinations also form between different parts, thus uniting organs to each other, to the abdominal walls, or to the mesentery or omentum; or sometimes matting the whole together into an inseparable and indistinguishable mass. They present great variety, and by the movements which take place within the abdomen adhesions may be stretched or made looser, or even be got rid of altogether in some instances, when they have formed after an acute attack. On the other hand, in many cases the adhesions and thickenings tend to become gradually stronger and denser, and at the same time to undergo contraction, so that they produce serious effects.

In many cases of chronic peritonitis effusion of some kind is observed. It may be merely a clear straw-coloured serum, or with fibrinous flakes, sero-purulent, or actually purulent. Blood may also be present in it. Occasionally this is the prominent or only anatomical change; and the fluid may range in quantity from a small to an enormous amount. Usually it is associated with the other conditions already described, so that the fluid is not free to move about, and may be actually circumscribed, or even lie in the substance of fibrous masses. Purulent accumulations are likely to make their way in various directions, either outwards or into internal viscera.

When chronic peritonitis is associated with tuberculosis, cancer, or other morbid formations, these will be evident on *post-mortem* examination. Moreover, as the result of degenerative processes, caseous or cretaceous particles or masses may be found. Pigment is also often present in abundance. The peritoneal veins may be extensively dilated and varicose.

It is important to notice the obvious effects liable to be produced upon the abdominal organs and other structures by chronic peritonitis. They are more or less fixed by the adhesions and thickenings, and may be displaced at the same time. Compression or constriction is often produced, especially important in the case of hollow viscera, as well as distortion, twisting or torsion, and incarceration. Some of these effects may occur suddenly or acutely in connection with bands of adhesion, thus giving rise to grave consequences; and fixation of the bowel may also lead to intussusception. The omentum may be greatly distorted, or contracted and gathered up in some abnormal situation; while the mesentery has been found extremely shortened so

as to contract the small intestine to half its length, its serous covering and longitudinal muscular layer being shrivelled, and its mucous lining thrown into transverse folds. It is said, however, that the small intestines are usually not compressed, or even adherent among themselves, though they are contracted in diameter. The deeper tissues of some of the abdominal viscera are likely to be affected by long-continued chronic peritonitis; and atrophy from compression may ensue. The muscular coat of the bowel is generally wasted, but that of the stomach is sometimes much thickened. As one good result of local chronic peritonitis, mention must be made of the fact that it is not uncommonly the means of preventing or modifying the injurious consequences resulting from some forms of perforation of abdominal viscera, by giving rise to previous adhesions and thickenings, and thus obviating the escape of their contents, or limiting their dissemination.

SYMPTOMS.—According to its mode of origin, chronic peritonitis either remains after an acute illness, or after a succession of more or less acute or subacute attacks; or its onset is gradual and chronic from the first, and may be very insidious. Of slight adhesions left after acute peritonitis, or originating from chronic causes, there are often no clinical signs; or there may be uneasiness and discomfort, or even painful sensations at times in some part of the abdomen, especially the iliac region, with a tendency to intestinal disorder, in the way of spasmodic movements and constipation. Even when there are no symptoms whatever, adhesions may at any time cause serious consequences. In well-marked cases of chronic peritonitis the symptoms to be expected are of the following nature: Abnormal subjective sensations are usually experienced in the abdomen, such as tightness, fulness, dragging, or actual pain. The pain, when present, is of a dull character, not severe, and liable to come and go, or to present exacerbations from time to time; it is often localised, and especially if the peritonitis be circumscribed; sometimes there is a feeling of local soreness or heat. The painful sensations tend to be increased by movement, and by shaking the body. They are sometimes aggravated by posture, in some cases by bending forwards, in others by the erect posture; and they may be increased by going up stairs, especially if the abdomen is distended. More or less tenderness on pressure is very common, even when there is no spontaneous pain, but not invariable; it is frequently more evident at certain spots, where it may be considerable. Colicky pains are not unusual in chronic peritonitis, and may occur in severe paroxysms, especially after food, being due to the disturbed action of the bowels, associated with the production and movements of flatus, which may be abundant, even amounting to tympanites. Appetite is often impaired or variable; and dyspeptic symptoms are frequent. Constipation is the rule, and may be very obstinate, even amounting to obstruction under certain conditions. Sometimes diarrhoea is present, or it may supervene at intervals, and occasionally it assumes a dysenteric character. This symptom is most likely to be met with in tubercular peritonitis. In some cases vomiting occurs from time to time. When there is considerable effusion in the peritoneum, the secretion of urine is diminished. Respiration may be mechanically interfered with from the same cause. As the result of pressure by

fibrous thickenings and other conditions upon different structures, jaundice, ascites, oedema of the legs, thrombosis, albuminuria, or neuralgic pains may supervene. When the organs are all matted together, their entire functions must necessarily be more or less interfered with.

General symptoms are usually present in various degrees in cases of chronic peritonitis, but in many instances they depend mainly upon the condition with which this disease is associated, especially tuberculosis and cancer. These symptoms include pyrexia, not high, and having no regular course, but presenting exacerbations, either persistent or occurring at intervals, and in some cases assuming a hectic character; increased frequency of the pulse; a sense of languor or weakness; and more or less general wasting and anæmia, with dryness and harshness of the skin.

It must be noted that in some cases of chronic peritonitis, even where there is considerable effusion, the local and general symptoms are very slight and indefinite, and the patient only suffers from the discomfort due to the accumulation of fluid. On the other hand, the progress is not uncommonly from bad to worse, ending in extreme emaciation and exhaustion, with the formation of bed-sores; or there may be a succession of improvements and relapses; while various phenomena result from the opening of collections of pus in different directions. Thus death may gradually or rapidly terminate a case; or pyæmia may supervene. Even in bad cases, however, comparative recovery may ensue, only the effects of the inflammation remaining, and being more or less troublesome.

PHYSICAL SIGNS.—These may be the only clinical indications of chronic peritonitis. They necessarily differ in detail according to the nature of the abnormal physical conditions present in the abdomen, and are also liable to alter during the progress of a case; but their general characters are sufficiently definite. (1) In general chronic peritonitis enlargement of the abdomen is observed, mainly in proportion to the amount of fluid present; but it depends partly on gas in the intestines, or in certain cases on extensive fibrous thickening. As a rule, it is not very considerable; but the abdomen may attain an enormous size, with stretching of the skin and other accompanying phenomena. While regular in shape on the whole, it may present more or less want of symmetry, especially after a time. On the other hand, in some instances the abdomen becomes locally or generally retracted, and may then exhibit marked irregularities. (2) The sensations on palpation are very variable, but often highly characteristic. It may happen that there is a uniform feeling of fluid, or of more or less firm resistance. More commonly the sensations are not uniform, but differ in different parts of the abdomen, including indistinct fluctuation in localised areas, sometimes very limited and in unusual situations; with firmness or resistance around these areas or in other parts, ill-defined, occasionally nodulated; and even distinct tumours may be felt, more or less irregular. These in some instances are due to morbid growths, such as cancer, but they may also originate in organised inflammatory products. Under certain conditions the abdomen yields a peculiar feeling of being movable as a whole. Abnormal movements of the bowels are sometimes recognised. When there are localised

adhesions between the visceral and parietal peritoneum, if pressure is made at a little distance from the seat of an adhesion, the skin will rise in a fold where this adhesion exists. Possibly general adhesions might be made out by palpation. (3) Percussion occasionally reveals freely movable fluid. As a rule, however, it shows that the fluid is not freely movable, or that it is actually loculated irregularly, this condition being associated with more or less solid material. Hence there is extensive and diffused dullness, which may be noticed mainly in front, and not in dependent parts. Not uncommonly patches of dullness and tympanitic resonance are found contiguous to each other, and irregularly distributed, unaffected by posture. Over the fluid fluctuation may, perhaps, be elicited, but indistinctly; and where there is much solid, the sensation on percussion is that of undue resistance. (4) Friction-fremitus and friction-sound are sometimes present. (5) Changes of posture, as a rule, produce comparatively little or no effect upon the shape of the abdomen, the sensations on palpation, or the percussion-sounds.

When chronic peritonitis is localised, it may be practicable to detect the condition by palpation and percussion. Moreover, when certain organs become fixed by peritoneal adhesions, especially if they are diseased at the same time, this state of things may often be recognised by noticing that the affected organ does not present its normal mobility.

DIAGNOSIS.—In most instances chronic peritonitis, if of any extent, can be recognised without much difficulty, by attending to the history of the case, the symptoms, and the physical signs. It may be very difficult, or even impracticable, to distinguish positively between mere ascites and chronic inflammatory effusion. All the circumstances of the case must be taken into consideration; and in doubtful cases the removal of some of the fluid, by means of a small trocar, will aid the diagnosis. It is important to determine the cause of chronic peritonitis, when present, and especially whether it is simple, tubercular, or malignant. Here, again, the whole case must be considered, not forgetting the age of the patient, the family history, and the presence or absence of tubercle or cancer in other parts. It has been said that a hæmorrhagic character of any fluid removed is significant of tubercular or malignant peritonitis, but this certainly cannot be relied upon.

It is quite impossible to diagnose with certainty obscure cases of localised chronic peritonitis, though the condition might be suspected; and it may become very difficult, even in pronounced cases, to determine the precise changes within the abdomen.

PROGNOSIS.—The prognosis of each case of chronic peritonitis must be considered on its own merits, as regards the cause of the disease; its extent and products; the progress of the morbid changes; the effects produced on the abdominal structures; and the general symptoms. Some cases are of little or no consequence; others are very serious; but even in apparently severe cases great improvement, or even practical recovery, may take place. The dangers to be feared from the opening of purulent collections in various directions must be borne in mind; and also those liable to arise from the presence of bands of adhesion within the abdominal cavity. Tubercular and carcinomatous peritonitis are necessarily very grave forms of the

disease, but the former may certainly be recovered from.

TREATMENT.—With regard to the *local* conditions in chronic peritonitis, it is often desirable to endeavour to promote the removal of morbid products within the abdomen. For this purpose it may be important to keep the patient entirely at rest in bed for a time. The internal administration of iodide of potassium or syrup of iodide of iron may be tried; and in some instances diuretics might be of use. Possibly the judicious administration of some mercurial preparation would be serviceable in appropriate cases. Violent purgation is to be deprecated; but where there is much fluid, advantage might be derived from repeated diaphoresis, induced by means of the hot-air, vapour-, or Turkish bath, or by the use of jaborandi or pilocarpine. Local measures are in some instances of essential service, namely, counter-irritation, especially by the application of iodine; friction with some oil or ointment; the application of mercurial ointment or liniment on flannel; and pressure. The writer has found pressure decidedly valuable in aiding absorption in certain cases, as well as in giving support, the abdomen being covered with cotton-wool, and a suitable bandage applied more or less firmly. A flannel bandage answers best. In cases of large effusion, where absorption cannot be effected, the writer has no hesitation in recommending paracentesis, even repeated when required, having seen signal benefit follow this treatment. A localised purulent accumulation must be treated on ordinary surgical principles. *See also* PERITONEUM, Tuberculosis of.

General treatment is often of essential value in cases of chronic peritonitis. It is directed to the condition upon which the disease depends, such as tuberculosis, or to its effects, but the measures are similar in the main, consisting of good nutritious diet, suitable sanitary conditions, change of air, and the administration of cod-liver oil, quinine, preparations of iron, and other tonics and nutrients. Wine may often be given with advantage.

Symptoms will probably need attention from time to time, such as pain, flatulence, dyspeptic disorders, constipation, diarrhoea, and various other disturbances. The organs in general must be looked to, and their functions promoted. A free flow of urine often follows absorption of fluid, or its removal by operation.

FREDERICK T. ROBERTS.

PERITONEUM, Dropsy of.—*See* ASCITES.

PERITONEUM, Hæmorrhage into.—Blood may escape in quantity into the peritoneal cavity as the result of external injury; or from the rupture or perforation of different structures within the abdomen. An important form of hæmorrhage is that which results from the rupture of an aneurysm. More or less blood may be present in inflammatory or dropsical effusion; or it may originate in the opening of vessels by morbid growths, or the spontaneous rupture of new vessels. Peritoneal hæmorrhage is not uncommon in connection with tubercular and malignant peritonitis. It may also occur from scurvy or purpura. Fatal hæmorrhage has taken place into the peritoneum owing to the rupture of enlarged veins, due to portal obstruction.

SYMPTOMS.—It might possibly happen that peritoneal hæmorrhage could be recognised during life,

if there were some evident cause for this condition ; followed by the physical signs of the presence of the blood in the peritoneal cavity ; and general indications of loss of blood. As a rule, however, the condition cannot be detected clinically. The hæmorrhagic nature of an effusion can only be recognised by withdrawing a portion of it.

TREATMENT.—This merely consists in the local and general treatment for loss of blood, unless any thing can be done surgically.

FREDERICK T. ROBERTS.

PERITONEUM, Malformations of.—It will suffice to mention under this head that the folds of the peritoneum, such as the mesentery, may be abnormal in length or formation ; that unusual bands or openings may be present ; and that prolongations of the peritoneum, which naturally become obliterated or shut out from the general cavity, sometimes do not undergo these changes, as may be illustrated by the occasional patency of the process of the serous membrane which descends with the testis into the scrotum. As the result of these abnormalities, displacements of organs may occur ; or their movements may be restricted or too free ; or constriction of the intestine may take place. These conditions can only be recognised clinically by their effects ; and not uncommonly they cannot be made out in any way. Treatment may sometimes be directed to their cure, as is exemplified in the radical cure of a congenital hernia.

FREDERICK T. ROBERTS.

PERITONEUM, Morbid Growths of.—Malignant growths are comparatively rare in the peritoneum. They are most commonly secondary, originating from extension, or as a distinct secondary formation ; and chiefly following malignant disease of the alimentary canal, especially the stomach, liver, pancreas, retro-peritoneal glands, and female generative organs, particularly the ovary. Malignant growths are very rare under thirty, but have been observed even in childhood. Most cases occur between fifty and sixty. The disease is much more common in females than in males.

Peritoneal cancer is generally of the scirrhus type, but is occasionally encephaloid, melanotic, or colloid, the last being comparatively frequently found here, and it may form an enormous mass. Primary growths, though differing little in structure, are classed as *endotheliomata* (see TUMOURS). Usually chronic, it either takes the form of separate nodules, which are often umbilicated ; or of an infiltration, sometimes of great thickness. Probably this appearance is due to an aggregation of nodules. Each nodule may send out processes, which tend to pucker and drag the neighbouring part of the peritoneum to it as a centre. The distribution of the nodules is very variable. They are usually far more abundant in the flanks and over the diaphragm than elsewhere. The omentum may be drawn up into a solid mass, as in tubercular disease. Generally there are associated signs of chronic peritonitis, with more or less effusion, which is often hæmorrhagic ; extensive hæmorrhages sometimes take place. Abdominal organs are usually found implicated ; or the cancerous process may lead to their destruction or perforation. In some instances there is a large dropsical effusion in the peritoneal cavity.

SYMPTOMS AND DIAGNOSIS.—The clinical phenomena in different cases of peritoneal cancer are

very variable in their exact nature, but their general characters may be readily understood. At first the disease will probably be obscure, and the symptoms indefinite. Abdominal pain is commonly complained of, and is often increased paroxysmally from intestinal disorder ; tenderness here and there is also generally marked. There may be signs of more or less disturbance of abdominal organs, or one or other of these is in most instances also the seat of disease. Cancerous growths in the peritoneum tend to interfere considerably with the alimentary canal, and by their contraction and puckering may cause marked narrowing of the intestinal tract, or even give rise to ileus. They may produce other symptoms by pressure on various structures. Ascites is a very frequent symptom ; and jaundice sometimes occurs. Physical examination will reveal signs of the growth itself ; of chronic peritonitis ; and of ascites. Cancerous nodules may originate friction-sounds. In some instances a hard thickening or mass is evident in the tissues around the umbilicus, which may also look red and inflamed. If fluid is withdrawn from the peritoneal cavity, it is usually more or less mixed with blood ; and marked anaemia or fainting may result from hæmorrhage. The general symptoms and cachexia of cancer become pronounced during the progress of the disease. Its course is occasionally somewhat acute, with pyrexia ; as a rule, it is more or less chronic, with little or no fever, or this only occurs at intervals. The termination is necessarily invariably fatal.

It is necessary to offer a few separate remarks about *colloid* involving the peritoneum. This condition is attributed to a colloid degeneration of a carcinoma. All the abdominal viscera may be enveloped in thick layers, consisting of round gelatinous masses. Many of these are attached only by the most delicate threads, or even seem to be free. It is a very rare disease, but when present in a marked form is, according to the writer's limited experience, easily recognised clinically by the following physical signs : (1) The abdomen is greatly and may be enormously enlarged, but is not quite uniform or symmetrical ; the umbilicus is only stretched, not everted. (2) Palpation generally reveals a feeling of diffused resistance, with rather firm, irregular masses. Fluctuation is either absent or very indistinct. (3) There is extensive dullness, with resistance, over the abdomen, the anterior regions being markedly dull, and there being no indications of accumulation of fluid specially in the flanks. (4) Usually a change of posture produces little or no effect upon the physical signs. (5) A slimy gelatinous fluid may possibly be removed by the exploratory needle or aspirator ; and occasionally a similar fluid is said to be discharged *per rectum*, or from the stomach.

TREATMENT.—Nothing can be done in cases of malignant disease of the peritoneum, except to treat symptoms.

Rare formations.—Among rare formations found in the peritoneum may be mentioned hydatids, associated or not with a similar disease in one or more organs ; serous, dermoid, and colloid cysts ; fibromata ; myxomata ; and remains of blood-clots. The peritoneum may be involved in actinomycosis.

FREDERICK T. ROBERTS.

PERITONEUM, Tuberculosis of.—Tuberculosis of the peritoneum occurs (1) As a part of general

acute tuberculosis; (2) As a localised formation, in connection with tubercular ulcers in the intestines, granulations forming on the corresponding surface of the peritoneum; (3) As a definite local disease, usually assuming a more or less chronic or subacute course, and accompanied by peritonitic changes. The last form is a well-recognised disease of early life, and is then usually a part of the condition termed *tabes mesenterica*, being associated with tubercular ulceration of the intestines, and tuberculosis of the mesenteric glands (*see* p. 1002). Most cases occur under thirty years of age, but the complaint may be met with over fifty. It appears to be much more frequent in males than in females. In the latter it is in the large majority of fatal cases found to be accompanied by tubercular disease of the Fallopian tubes, which is probably set up by extension from the serous surface. In men the epididymis or testis is sometimes affected on one or both sides. The pleure are not uncommonly involved, and occasionally the pericardium. Pulmonary tuberculosis is usually present, to which the peritoneal mischief may be secondary, but this is not always so pronounced as to be diagnosed during life, at any rate without careful examination. Tubercular ulceration of the intestines is frequently associated with the peritoneal lesion.

ANATOMICAL CHARACTERS.—The morbid conditions found on *post-mortem* examination in pronounced cases of tubercular disease of the peritoneum consists of a combination of disseminated tubercles in different stages, with signs of peritonitis. The peritonitis is generally chronic (*see* p. 1201), but may be acute. The tubercles are not uniformly scattered, and are said to be especially numerous on the under surface of the diaphragm and in the flanks, while the surface of the intestines may be comparatively free. They have usually undergone more or less caseation or fibrous change in different parts. As the result of the peritonitis, great thickening and extensive adhesions are usually present, with much contraction. The omentum is in many cases drawn up into a firm flattened mass across the upper part of the abdomen, below the stomach, and often contains much caseous matter, or recent tubercle. The mesentery is also contracted, drawing the intestines together, and distorting them. The bowels may be completely matted together, and perforated by tubercular ulcers at different points. More or less effusion is almost always present, consisting of turbid serum or pus; and it often contains altered blood in variable quantity. Sometimes abundant hæmorrhage takes place into the peritoneum. The effusion is frequently localised by adhesions at different points; or it may be ultimately removed altogether, only adhesions being left.

SYMPTOMS.—The clinical phenomena present considerable variety in different cases, as regards their nature and progress (*see* p. 1201). In some instances tubercular disease of the peritoneum begins acutely, or in a succession of acute attacks, usually circumscribed, with symptoms like those of peritonitis, then subsiding into a chronic condition. Far more commonly the progress is subacute or chronic and insidious, or even latent for a time. In other cases there are marked remissions of the symptoms during their progress, both local and general. The phenomena may be summarised as those of the peritoneal inflammation already described, and the presence of firm lumps in the abdomen, mainly revealed by physical examination; with general sym-

ptoms of tuberculosis, usually in a pronounced degree; and often signs of implication of other important organs and structures in the tubercular mischief. In exceptional instances in the writer's experience the prominent condition has been abundant effusion of fluid in the peritoneal cavity, usually recurring after repeated removal, but in one case ultimately completely cured. The detection of the great omentum, which is felt as a rounded mass, extending more or less obliquely across the upper part of the abdomen, is highly significant in the diagnosis of tubercular disease of the peritoneum. In some cases there is firm thickening, with redness and soreness, around the umbilicus, which is usually due to extension of inflammation from the peritoneum along the obliterated umbilical vessels to the surface. Occasionally the umbilicus has given way, and a discharge of pus has taken place from the peritoneal cavity. Tubercular peritonitis is usually chronic in its progress. It is a serious disease, but is not so fatal as was formerly supposed, especially in young subjects, and when treated in accordance with modern views.

TREATMENT.—The general measures suitable for tubercular disease must be carried out, as well as those already indicated for chronic peritonitis, as above stated. Wilks recommends the administration of iodide of potassium, with or without quinine, and inunction over the abdomen with liniment of mercury, followed by the application of tincture of iodine. The writer has seen remarkable benefit follow the administration of guaiacol carbonate in cases of undoubted tubercular peritonitis, and in one instance a complete cure was effected.

OPERATIVE TREATMENT.—Of late years the principle of opening and draining the peritoneal cavity has been extensively advocated. This treatment is especially recommended in cases in which there is extensive accumulation of fluid either free in the peritoneal cavity or confined to special parts by adhesions. The dry caseating forms with extensive adhesions are less amenable to operative interference, which indeed is itself less practicable. The abdomen is usually opened, with strict antiseptic precautions, in the middle line below the umbilicus. All the fluid is allowed to escape and the cavity carefully dried with 'swabs.' Any diseased omentum that can without difficulty be removed is excised, and, in women, the Fallopian tubes, if tuberculous, should also be removed. When localised collections of fluid are found, the whole peritoneal cavity may be flushed with boric-acid solution or sterilised saline solution. This treatment with the general disturbance of the intestines which it involves is often followed by great benefit. The mortality varies from 3 to 8 per cent., and the cures are said to exceed 60 per cent. These figures, of course, only refer to operations upon carefully selected cases.

FREDERICK T. ROBERTS.

PERITYPHLITIS (περί, around; and τυφλόν, the cæcum).—*See* APPENDIX VERMIFORMIS, Inflammation of.

PERI-UTERINE HÆMATOCELE.—*See* PELVIC HÆMATOCELE.

PERSONAL HEALTH.—Personal hygiene is the science of individual health. As there are public acts and laws which, observed, promote the health of communities, so there are rules of living

and habits of life, by attention to which the health of the individual may be preserved or increased. Personal health admits of being estimated. See LIFE-ASSURANCE.

From birth onwards to old age, health is not uniform; it varies as the body varies, according to wear and tear, and treatment—a sufficiently obvious proposition. At different epochs of life the strain or stress is felt in different parts, falls upon different organs, and issues in proclivity to disorder of their several functions, or in wear or degeneration of the tissues of which they are built. The life of a human being may be divided into the following periods, which will be considered separately in relation to their special physiology, to morbid imminences, and to probable accidents. Rules of guidance will be laid down as to diet, clothing, habits, exercise of body and mind, and all that is conducive to the health of the individual at the different ages.

LIFE-PERIODS.—The following are the periods of life, as they will be here successively considered:—

1. **Intra-uterine life and Gestation.**
2. **Infancy**, the period between birth and the completion of the first dentition.
3. **Childhood**, the period between 2 and 7 years.
4. **Adolescence**, the period between 7 and 14 years.
5. **Puberty**, the period between 14 and 20 years.
6. **Adult age**, the period between 20 and 30 years.
7. **Maturity**, the period between 30 and 45 years.
8. **Turning-time**, the period between 45 and 60 years.
9. **Advanced life**, the period between 60 and 82 years.
10. **Old age**, the period between 82 and 100 years.

1. **The Intra-uterine and Gestation-Period.**—The health, habits, and conduct of the mother during pregnancy modify the future individual considerably. Whatever affects the blood of the mother affects that of her fetus, and *vice versa*.

Alcoholic abuses committed by the mother during pregnancy favour premature delivery, and appear beyond this distinctly prejudicial to the health of the children when these are born alive, the constitutional flaw not showing itself by apparent malnutrition so much as by undue proclivity in them to manifest disorders of the nervous system—chorea and epilepsy in childhood, hysteria and insanity in adult years. Experience shows the hygiene of this period to consist in temperate living. The pregnant woman should avoid excitements of all kinds, take moderate exercise, rise and go to bed early, not alter her habits of life abruptly. She must dress herself appropriately to her state, not so as to interfere with the emergence of the uterus from the pelvis, or so as to limit the movements of the babe *in utero*. See FŒTUS, Diseases of; and MONSTROSITIES.

2. **Infancy.**—The period of infancy may be subdivided into *early* and *late*: *early* comprehending the time from birth to eruption of the first teeth; *late*, that from the commencement to the completion of the first dentition. The leading anatomical features of this age are the large amount

of blood relatively to the solids of the body, the laxity of all the tissues, the disproportionate quantity of component water, and the large relative amount of red blood-corpuscles and of iron, which appears far in excess of that existing in adults.

The circumstance of chief physiological importance is that the greatest growth occurs in the first years of life. Quetelet in his essay, *Sur l'Homme*, shows that the mean average weight of male infants exceeds that of females; boys at birth weighing 3 kilogrammes 20 grammes, and girls 2 kilogrammes 9 grammes. There is no indicator so infallible as the balance to prove whether a baby is or is not being properly nourished. It appears that from birth up to the end of the second day all infants lose weight a little; they do not increase perceptibly till after the end of the first week.

M. Odier states that it is usual to find an infant increase 30 or 40 grammes (461 to 606 grains) *per diem* during the first five months of life, 20 grammes (308 grains) a day from the fifth to the eighth month, and 10 grammes (or 155 grains) daily between the eighth and the twelfth month.

In infantile life all the vital functions go on rapidly. The pulse at birth ranges from 130 to 140 per minute; and to the end of the first year is from 115 to 120. The rate of respiration is from 25 to 30. While the circulation is rapid, the skin, from its softness and vascularity, disperses heat rapidly; the cooling agencies are at a maximum; and the heat-maintaining powers (that is, resistance to depressing influences) are at a minimum. 'The food taken by infants is, in proportion to the weight of the body, from three to six times greater than that taken by adults.'

The perils from without to infant life are mainly derived from cold, those from within result chiefly from improper or defective feeding, and from the over-sensitiveness of the nerve-centres. Young brains and spinal cords are over-alert to impressions received from without. Powers of control become developed as they grow older. The ordinary phrase 'emotional as a young child' expresses a physiological fact. It is not easy to over-feed young infants. If proper food, that is, their own mother's milk, be given them, they get rid of excess quickly enough by vomiting it, and the part not appropriated in growth or maintenance is stored up for future use as fat. Catarrhal diarrhoea and bronchitis, thrush and stomatitis, are due principally to the introduction of micro-organisms in the food or air. Delirium and convulsions attend all febrile disorders. The more closely the evolution of the teeth follows its normal periods the less conscious are infant and mother of their appearance. See DENTITION, Disorders of; and TEETH, Diseases of.

DIET.—Experience proves that no aliment is so appropriate as the milk of the mother, or of a wet nurse aged between twenty-two and thirty-five. Next best to this comes the milk of the ass; and next, again, a mixture of equal parts cow's and ass's milk given by a feeding-bottle. The suckling of her own infant by the mother for nine months is good not only for the child, but for its mother. The uterus passes through its retrograde involution more rapidly, no periodic uterine congestions delay it, and ovulation is deferred. No artificial purgative, oil, gruel, or sugar-water, should be given in lieu of the mother's first colostrum-milk. With respect to the frequency of feeding, and the quantity taken,

the following rules may be laid down. During the first day of life, what with scantiness of the colostrum, mechanical obstacles to suction, and the weakness of the infant's efforts, the child does not extract more than a drachm each time it is placed to the breast. It needs no more, however. During the first week of life it should be nursed ten times in the twenty-four hours, arranging times so that the mother gets six hours' consecutive rest at night. On the second day each suckling should furnish about 5 drachms of milk. On the third day each suckling should furnish about $1\frac{1}{2}$ ounce of milk. On the fourth day each suckling should furnish about 2 ounces of milk. During the first month average-sized infants require and obtain nearly 3 ounces of breast-milk at each nursing, and should be nursed nine times in the twenty-four hours, or receive about 27 ounces of milk a day. During the second month each suckling should furnish $4\frac{1}{2}$ ounces of milk, and the number of feedings may be reduced to seven *per diem*, which allows $31\frac{1}{2}$ ounces each twenty-four hours. At three months old the infant sucks about 5 ounces at a meal, an equivalent of 35 ounces each twenty-four hours; and at four months it extracts as much as $6\frac{1}{2}$ ounces of milk at each meal, which may be again curtailed to six each day, giving $37\frac{1}{2}$ ounces of aliment. This continues to be the quantity of milk and frequency of feeding required of a good nurse up to the end of the ninth month; but the quality of the milk during this period steadily improves, becoming enriched according as the child sucks more vigorously and at longer intervals, a provision fraught with mutual advantage to child and mother.

At the ninth month the child may be gradually weaned, although the age for weaning should be governed by the health of the mother or nurse, the forwardness of dentition, and the infant's own craving for other food. The best time to take for the purpose is the interval or pause after the four lateral incisors are cut, and before the first molars appear.

Dentition, normal order of.—For this subject see DENTITION, Disorders of.

CLEANLINESS AND CARE.—The infant requires washing all over from top of head to sole of foot night and morning every day, and is best, because most quickly, immersed in a tub once daily. Infants who have had convulsions at any period of their lives are, as a rule, better washed all over with a sponge in the lap of their nurse than immersed in a bath, as immersion is apt to frighten them. The water should be the softest procurable; rain-water is best. The temperature of the room during the bath should be between 65° and 70° F.; that of the bath itself, fixed by the thermometer, between 70° and 90° , according to the season of the year. Fixing the temperature of the bath should not be left to the possible indiscretion of a nurse; many a woman's hand will support water at a heat sufficient to parboil a baby.

The nurse should be required not to dawdle over bath or dressing; the former should occupy five minutes, the latter not more than twenty. Little or no soap, or only soft soap, should be employed. The drying should be accomplished with soft dry cloths; and for dusting-powder, to prevent excoriations, fuller's earth cannot be surpassed. Eczema and intertrigo are obviated by due attention to the frequent change of diapers and to cleanliness.

CLOTHING.—No infant ought to be swathed like a mummy; it requires keeping warm, but should not be overweighted with clothes. Its chest must be free to expand, its limbs at liberty to move. From neck to feet it should have woollen garments (with the exception of the napkin) next the skin, since it is impossible to ensure absolute freedom from draughts and rapid changes of temperature. The pernicious custom of heavily weighting an infant's shoulders with clothes, and of leaving the body from the waist to the ankles exposed, cannot be too strongly condemned. The more lightly its head is covered the stronger will be its hair, and the less its susceptibility to catarrh.

GENERAL RULES AND HYGIENIC ADVICE.—Even the youngest infants require sunlight and open air. Due discretion must be employed, however, in sending them out. If the temperature of the external air is about 60° , children may be allowed to go out when they are eight to fifteen days old, after cicatrization of the umbilicus. As soon as they can crawl they should be encouraged to do so, either on a carpet, in a garden, or on a dry sandy pathway protected from wind and open to sunlight. Cold and dark places are specially inimical to them; and when the weather is cold they should be encouraged to amuse themselves on a blanket or soft hearthrug, so as to learn to stretch their limbs and co-ordinate all their muscular movements. They learn first to sit up, then to stand, helped by their arms, against a chair; next to stand without support; and at some period between one year and two years of age should be able to walk about by themselves.

SLEEP.—Infants require sleep by day as well as by night. Very young babies do little else but suck and sleep. As they grow they need and take less and less sleep, and by the time the first dentition is accomplished—three years of age—a child may usually dispense with sleep altogether in the daytime, except a short hour's nap early in the afternoon or between eleven and twelve. The infant should have its own cradle, and the child its own cot, placed close beside the bed of its mother or nurse. In very cold weather, or hard frosts, the cot should be artificially warmed by a hot-water bottle. The sleeping-nursery ought not to be kept warmer than 65° , nor colder than 50° ; while the nearer it is maintained to 55° during the winter months, and 65° during summer, the sounder the child will sleep. The more freely the whole house and nurseries are ventilated, the less prone the infant will be to all infantile disorders.

3. Childhood.—In this period, between the second or third and seventh years of life, the first dentition is accomplished, the second uncommenced. The rate of pulse falls from 115 to 90 per minute, and respiration commensurately. The excretions are all absolutely increased. In the co-ordination of muscular movements and in mental operations great progress is being made. The cerebro-spinal structures, which nearly double in volume between birth and the second year, continue to develop disproportionately to the growth of the trunk and limbs between two and seven. The cellular tissues are loose and vascular still, and the cutaneous and mucous surfaces therefore unduly vulnerable. A notable physiological feature of this age is the readiness of the lymphatic glands to swell upon the slightest irritation, and the general functional activity of all the lymphatic structures. Hence arises a

tendency to eczema and to catarrh of mucous surfaces, diarrhoea, laryngeal and bronchial catarrh, general anasarca, and hydrocephalus; a susceptibility to contagious impressions; and a proclivity to tubercular meningitis, and to functional cerebral disorders like delirium and convulsions. The incontinence of urine, so frequent in early childhood, may be likewise referred to the reflex irritability of the spinal centres characteristic of this age. Undue sexual excitability may occasionally arise and vicious habits be inaugurated. Hence the importance of good nurses and wise supervision cannot be too much insisted on, as also the inculcation of healthy habits and provision of proper amusements and employments.

DIET.—While bread, starch, and flesh-foods are gradually taking the place of cows' milk, they must not be allowed wholly to supplant it. Eight ounces of bread may be reckoned about equivalent in nitrogen-content to one pint of milk, but the former exceeds the latter in carbon. The food must be nutritious and abundant. The error committed is far too often that of under- than of over-feeding. Young children do not require so much variety in their food as adults do, but are greatly benefited by a change in their bread and meal-stuffs, and a dietary not too monotonous. They do not need meat more than once a day, and fish may be substituted for meat, if cream or butter sauce be provided with it, once or twice a week. Milk, bread, porridge, suet-puddings and milky puddings should form the staple of their dietaries; fresh vegetables well cooked, watercress, cooked fruit, and oranges are most useful adjuncts; while the addition of fried bacon, clotted cream, and oil or butter, when the drinking-water is hard, and the tendency of the child is rather towards constipation than otherwise, is now generally understood. It is usually easy and always beneficial to instruct young children to secure an alvine evacuation directly they rise of a morning, and before their bath. Four meals a day are most appropriate—a breakfast at eight, a dinner at twelve, a tea at four, and a supper at half-past six.

CLEANLINESS.—Washing all over once a day, and in the morning, is as necessary as ever; but after first tubbing in hot water between 98° and 100°, the child should stand up and be sponged all over from a basin of cold water, and be briskly dried with a large towel.

SLEEP.—A child should sleep in a cot or bed by itself, but in the same room with its parent or nurse, since it is apt to show any disorder by nocturnal restlessness, delirious talking, or sleep-walking.

Between 2 and 5 most children are the better for twelve hours of sleep out of the twenty-four. At 7 years of age they do not require sleep in the daytime, but should be in bed at seven or half-past seven, and up at six in summer and between six and seven in winter. The best bed for this age is an ordinary iron bedstead, with firm and level wool-and-hair mattresses; not spring beds, which do not adapt themselves so well to light bodies, nor keep them uniformly warm. Cotton sheets, blankets, and counterpane must be used according to season. The day-clothing should be warm, and merino or wool put next the skin.

EXERCISE.—Two things are requisite for healthy growth and development and a happy childhood—a play-room and a garden. Children need a place

like an empty barn, in which they can swing and amuse themselves in wet and wintry, as well as in hot sultry weather, practising those games which are requisite for the schooling alike of their muscles and nerves.

TEACHING.—Teaching such as they need should be conducted on the Kindergarten system; but the main rule for their lives is open air and exercise, the chief objects being to harden their skins, develop their muscles, and teach them self-control, and love and respect for those to whom they render implicit, because well-nigh unconscious, obedience.

4. Adolescence.—The consideration of the period of second dentition, between the ages of 7 and 14, is best prefaced by the order of eruption of the second teeth.

About 7 years the 4 anterior molars (permanent teeth) are cut.

About 8 years the 4 central incisors.

„ 9 „ 4 lateral incisors.

„ 10 „ 4 anterior bicuspids.

„ 11 „ 4 posterior bicuspids.

„ 12 to 12½ „ 4 canines.

„ 12½ to 14 „ 4 posterior molars.

The teeth of the lower jaw usually precede those of the upper. Second dentition is accomplished leisurely, and is therefore usually accompanied by no such grave disorders as mark first dentition; but in nervous children nervous tricks may manifest themselves, as well as marked lack of emotional control. Some are hypersensitive, others contradictory and difficult; and most parents admit that between 7 and 8, if not between 7 and 14, they learn what the characters of their children really are. Physiologically, absorption of the subcutaneous fat goes on rapidly, while the muscles become more developed, the skin gets tougher, its epidermis harder, and it perspires less readily. In our climate the morbid liabilities of this age are to rheumatism, chorea, epilepsy, the exanthemata, and typhoid fever.

Between 7 and 8 the appetite is apt to become capricious; the child physiologically does not require so much fatty food; and, while growing fast and becoming leaner, protests against fat, often while showing a marked longing for fresh fruits, in which nature should be indulged. After 8, however, any marked defect of appetite or loss of weight is suggestive of undue cerebral excitement, attributable to over-study or some infraction of the laws of health.

DIET.—Three good meals a day are sufficient, but four are more advisable. Constipation at this age usually signifies irregular feeding and overloading with pastry or other improper food. Breakfast at eight, dinner at one, tea at five, and supper at eight appears the best distribution. By supper is meant such a meal as growing lads and girls positively need. They require either soup and potatoes, and bread and butter, or some one hot dish of meat or fish; and the drink should be either warm milk or cocoa to about half a pint of fluid: aliment enough is needed to improve the circulation at the extremities and obviate chilblains. Boys and girls may retire to bed within an hour of their supper, which, instead of making them dream, will secure good and refreshing sleep. The greatest dangers at this age arise certainly from defective nutrition and an over-sensitiveness of the skin. Neither wine nor beer should be allowed without medical authorisation.

CLOTHING.—Woollen material should lie next the skin, as it conducts heat less readily than cotton, silk, or linen, and therefore affords the most efficient protection against exposure. A whole chapter could be devoted to the foot alone, and its clothing during its growth and development. The desiderata appear to be length and breadth enough, low heels, impervious soles, old and flexible skins for uppers. Boots for outdoor exercise are advised for children, because their ankles need support; shoes a little later on, because they are cheaper and may be discarded at once when worn out. The same boots should not be worn day after day—they require time to dry properly in damp weather, and the foot at that age profits by change of pressure. During youth the adaptation of clothes to special sports and exercises is far from unimportant to health. For violent muscular exercise, flannel or merino should be worn next the skin, and an easy flannel jacket or over-jersey; both after being used should be hung up to dry and air before being worn again. It is well that youth should be informed how rheumatism is promoted by indolence, neglect of exercise, carelessness in getting overheated, standing about on damp soils, remaining in wet shoes, wearing woollen shirts or underclothing that have been permeated and saturated over and over again with the secretions of the skin.

REST AND EXERCISE.—These are requisite for both body and mind at this age; the duty belongs to parents and schoolmasters to study what is appropriate. We annex, therefore, a table from Friedländer, which shows how the twenty-four hours may be wisely apportioned:—

Age	Hours for			
	Exercise	Work	Leisure	Sleep
7	8	2	4	9 or 10
8	8	2	4	9 or 10
9	8	3	4	9
10	8	4	4	8
11	7	5	4	8
12	6	6	4	8
13	5	7	4	8
14	5	8	4	7
15	4	9	4	7

5. Puberty.—The physiological feature of this period is the more rapid growth of the whole body, and the gradual perfecting in their functions of its several organs. Between 14 and 20 the human organism reaches not only its full size but the completion of its organic endowments. During this period mind and body expand together, but the body develops more rapidly than the mind. Growth proceeds by fits and starts, succeeded by periods of quiescence; seasons affect it, so do supplies of food; boys do not develop so rapidly in autumn and winter as in spring and summer. Girls at this age often fall back, as it were, a little in winter, when they are much more confined indoors, to make a greater push forwards in spring.

It is even difficult for the digestive and assimilative powers to keep pace with the bodily requirements, so that the tendency is for the temperature of the body to fall somewhat, to be ill maintained at the extremities, and for the cold bath to be shunned for lack of adequate reaction in those who are manifestly growing very rapidly.

The heart in some is hardly equal to the task set it, and when it is diseased we perceive both growth and the attainment of puberty retarded. The lungs, again, more often in girls than in boys, do not expand in proportionate ratio with the rest of the body. The body runs up tall, but the thorax remains narrow and flat, and the apices of the lungs approach too closely to each other. The morbid imminences of this age are disorders of the nervous system, chorea, epilepsy, mania, anæmia, rheumatism, and pneumonia. Girls during this period of their lives suffer more illness than boys, probably in consequence of insufficient gymnastic exercises and of over-study in cramped postures. It is the age of all others when good or bad habits of life are formed; the time, too, when the seeds of disease are sown broadcast, to spring up in the after-age of man- and womanhood.

DIET.—Food should be abundant, varied but unstimulating. Three or four moderate meals a day are requisite; if at any period of life fermented liquors are beneficial, now is that time. Light bitter unadulterated table-beer or claret and water should be provided at dinner, but not more than half or three-quarters of a pint of it allowed. If violent exercise has provoked thirst, this may be satisfied with plain water or toast-and-water *ad libitum*. Girls should take cocoa for breakfast, with bread and butter, meat, eggs, bacon, or fish, as much as they like. School dietaries err usually on the side of deficiency. At dinner, as well as substantial meats, fruits, vegetables, and suet and milky puddings are required. Tea should be allowed only once in the twenty-four hours, at six o'clock, and a warm supper be provided at nine o'clock.

CLOTHING.—Nothing need be added to what has been already advised. Without entering into minute particulars, it should be seasonable, rather extra-warm, and offer no uncomfortable restraints. When mothers complain of their daughters' neglected figures, the hygienist retorts, What gymnastic exercises did you require of them? It is the age for exercise of the body as well as of the mind. Boys' spines are straight and girls' backs crooked, because the former use all their muscles and the latter do not. As the body is making its most rapid growth, so the evil of unilateral use of muscles is particularly baneful. Sitting over-long in a slouching attitude will tend to contract the chest, as carrying too heavy weights over the back will spoil the normal spinal curves; so leaning too much on one side, standing too long on one foot, even carrying constantly a pocketful of articles on one side of the dress, will suffice at this age to induce spinal curvature. The daily use of the trapeze, swinging, playing games like *la grâce*, in which both arms are used, badminton, and lawn-tennis, in which arms and legs are employed, and every muscle brought into due action, are quite essential to the proper development of the thorax and the muscles of the trunk. Girls should row and run and ride and swim and skate no less than lads do, in order to become fit mothers for a nation like ours.

The best temperature for a sitting-room is 60°; that for a sleeping-room between 50° and 55°. The hygiene of the bed-room and the bed needs a few words. The temperature of the room should not rise above 65° in summer, or fall below 45° in winter. It must be thoroughly ventilated with a

constant amount of fresh air passing through it during the day. The desiderata for a bed are coolness for the spine, restfulness for the trunk-muscles, and warmth without too great heat or too burdensome a weight of bedclothes. A horse-hair bolster is preferable to a pillow, and a paper-pillow to a feather-pillow; a feather-pillow enwrapping the neck and head heats the upper part of the spinal cord undesirably. Posture in bed is not unimportant. The head should be low, the feet perhaps a trifle raised, certainly not dependent. 'Sleep not on your back, as a dead man,' is a maxim attributed to Confucius; the opposite attitude, on the stomach, is restrictive of the intestinal movements and uncomfortable. It is as well to begin the night lying upon the right side so long as food remains in the stomach, and to turn on first waking upon the left side. The best attitude is probably that crouched one habitually selected. Good advice is to stretch yourself straight whenever you wake, in order to render the circulation of the blood freer. In winter the arms should lie under the clothes, in summer above them.

The cold, or almost cold, bath should be taken by all persons, unless pronounced medically unfit, every morning; with a warm or tepid bath once a week, for cleansing purposes, throughout summer and winter. While the young of both sexes should be encouraged to swim, in seasonable weather, the length of time they stay in the water must be strictly limited according to the temperature.

We abstain purposely from any discussion of the hygiene of mental education. See CHILDREN, Training of.

6. Adult Age.—This is the prime of life, between 20 and 30. Anatomically the body broadens, the chest deepens; for feats of muscular prowess—short, severe labours—it is at its best. The intellectual and cerebro-spinal sexual energies are at their maximum. What the French call the greatest latitude of health and of strength exists at this period; severe strains are supported with apparent ease. In male adults the body gains weight by small amounts for about twenty-eight days, then relapses to its normal average by a sudden crisis, attended by head-heaviness, loss of appetite, and copious discharge of urine or seminal evacuation. It is not a time about which the hygienist has much to say. If the preceding periods of life have been wisely ruled the individual is at his or her best. The morbid imminences directly belonging to this age should be few, and certainly are usually due to direct contravention of the laws of health: to exposure to contagious influences, to irregular living, especially drinking, to excessive strains upon the heart or blood-vessels, to pulmonary inflammations, to contravention of proper sexual relations, to excessive emotional excitement, or to mental worry and loss of sleep.

The guiding rule for this period is succinct enough: '*Sustine et abstine.*' Total abstinence from alcoholic drinks may be recommended. Not only does it favour health, but lessens all the temptations incident to these important years, in which a man carves out his own career. A question not infrequently propounded is, How shall I know when I have eaten as much as is good for me? If individuals are dull or drowsy after a meal they have usually eaten too much; if they can converse, write, or transact business with ease after a meal, they have fed temperately.

Women may be advised to marry not earlier than 21—between 21 and 28—when in our climate they are best fitted to become wives and mothers. Men had better wait till between 28 and 35 before they undertake the responsibilities of being parents. For the generality of men and women we must insist once more on their not giving up out-of-door muscular exercises. An entirely sedentary trade or office-life cannot be a healthy one for either body or mind: the latter appears to suffer most from it—the sense of morality becoming blunted.

SLEEP.—Doubtless different constitutions and individuals differently employed require different amounts of sleep. As nothing dulls the intellect and weakens the recuperative faculties more than too much sleep, except over-feeding and drinking at this age, so few things are more certain than that a man may rise too early for making the best use of his twenty-four hours. John Wesley's advice in this matter is worth recording. He writes that any man can find out how much sleep he really requires to repair his nervous system by rising half an hour earlier every morning until he finds that he no longer lies awake at all on going to rest in bed, or wakes up until it is time for him to get up. Six to eight hours is usually ample for healthy adults, with nine hours every seventh day. The mistake too often made is that of endeavouring to make up for over-hard mental efforts by over-long sleep hours. Mental over-fatigue is to be repaired not by sleep, but by bodily exercise in the open air. Exercise directs the blood-flow from the head towards the muscles, and renews the appetite. As we have pointed this out as the suitable age for marriage, we may mention some things which conduce not slightly to healthful and happy marriages: parity of station, compatibility of temper and tastes, and no marked disproportion either in age or size.

7. Maturity.—The body has now reached its maximum weight and solidarity, and the period is that of maximum endurance. Men reach their full weight at 40; women later, sometimes not till 50. The morbid tendency is towards obesity or dyspepsia, both alike being determined by a too sedentary town-life, and by daily occupation in close, ill-ventilated, and badly lighted chambers. Now are perceived the first attacks of gout; while visceral degenerations and atheroma of arteries may manifest themselves—events all of which may be delayed, if not wholly prevented, by attention to the laws of health. It is desirable that each individual should pay heed to his weight at this age, since this indicates whether or no he is living wisely. When, however, men are engaged in trades or professions, there is no more difficult task than to maintain their weight at this age, the *juste milieu* referred to being a hard matter to secure. The advice given by Celsus (lib. 1, cap. 1) cannot be surpassed in force or brevity: '*Sanus homo qui et bene valet et suæ spontis est nullis obligare se legibus debet; hunc oportet varium habere vitæ genus, modo ruri esse, modo in urbe, sæpiusque in agro, navigare, venari, quiescere interdum; siquidem ignavia corpus hebetat, labor firmat.*' As to diet, clothing, and habits, we need add nothing to what has been already advised for a previous age; but on exercise of body and mind there is much to be written.

A good rule is laid down by Lynch, too, in his *Guide to Health* (p. 290), that the lean should exercise *ad ruborem*, i.e. to glow-point, or until

their bodies and spirits are heated, for that will fatten them; and the fat *ad sudorem*. The more luxuriously a man lives, the more exercise, and the more active exercise, he needs. Want of it, and the costive habit thus superinduced, may, as Kotzebue observes, extinguish the divine flame of genius and seriously impair the intellectual powers. Hypochondriasis and hysteria are the special punishments of ease and affluence and indolence. Obviously a portion of each day should be set apart for those who can take it. In the households of the wealthy a gymnasium is at least as important as a bath-room. The chest should be expanded by clubs and dumb-bells; swinging on the trapeze, and hanging by the arms and legs, may be recommended. Again, before forenoon or midday meal, an hour's ride or walk must be obtained; and a third time in the day an hour and a half's exercise—fencing, or walking, or rowing—should be arranged before bedtime, in the spring and summer seasons. A great point is to vary the exercise by every means at hand: to change the set of muscles called chiefly into play upon different days, as Celsus advised: to swim, ride, fence, sail, row, shoot, fish. Bodily exercise should not be undertaken immediately after a heavy meal; nor should those who have sweated themselves violently sit down at once to a full meal—at least an hour's rest should intervene.

A few words of advice may be given as to what is proper and profitable in mental work, and what is improper and likely to prove detrimental to the cerebral organs. Most brains suffer more from rest than overwork. Exercise is as essential for the healthy nutrition of the cerebral as it is for that of the muscular tissue, and without regular employment daily up to fatigue-point no high quality of intellectual condition is ever attained. Fret, hurry, worry, and the endeavour to accomplish some task in too short a time, is what wearies and wears the thinking organ; but what damages it is always its imperfect nutrition, the insufficient repair of its waste after its active employment. It is a matter of observation that feeble brains, those constructively defective, or those damaged by accidental injury or by disease, most speedily suffer both from overwork and from actual disease. The education of the half-witted and of congenital idiots is a difficult problem as well as a disheartening task; that they admit of some improvement is proved, but directly their daily exercises are discontinued they fall back. Immature brains are doubtless more vulnerable and require more rest, more sleep, and more diversion than fully developed ones. Again, the mere degenerative changes in the blood-vessels and capillaries incident to disease and old age interfere with the nutritive changes requisite for the most perfect intellectual operations. Hence it is easy to point out a true pathological foundation for epileptic insanity and senile dementia, and to perceive why poorly fed children should suffer from over-pressure in schooling.

The cultivation, however, of the human mind, and the determination of what is best for its sound development and maintenance, belong to education, not hygiene. Let it suffice to say that the *mens sana in corpore sano* depends as much upon the judicious training and education of the intellectual centres, as it does upon their minute structures and sound nutrition. The best intellects are built on good foundations; the education of the cerebral

centres begins with the first special-sense impressions made upon them, and, we may hope, does not terminate only with this life. There is still as much to be learned in the training of the brains as there is in that of the muscles. True excellence is only to be reached after much striving, and can be attained only by gradual steps, and slowly.

8. **The Turning Period.**—This period of life, which lies between 45 and 60, is also known as the *grand climacteric* or *middle age*. The skin wrinkles. Up to 60 years of age the skull may continue to increase in size, principally at its anterior part, by enlargement at the frontal sinus; after 60 the skull-cap loses weight, and the brain may waste, but gets tougher and firmer. The heart grows a little larger, and its walls are thicker. The lungs grow denser, a change common to every tissue of the body. The hair grows grey; the features sharpen; the sight alters; and the hearing grows dull. Pressure and wear and tear begin to tell at every part. Upon the blood-vessels their effects are more marked in males than in females, because ordinarily the former labour harder than the latter; further, the death-rate of men is greater than that of women at this age. As the sexual powers decline, which they do by a quick descent between 46 and 63, the intellectual powers increase, so that mentally there is often exhibited a marked increase of vivacity and agreeableness, more noticeable in men than in women. In the latter the cessation of the catamenia is attended usually by some rejuvenescence, attributable to their recovering a little embonpoint. It is an age, however, at which women kick rather, and become restless and uneasy, the change of life being attended in many by a renewal of their juvenile tempers, as between 7 and 14, and occasionally by a revival of their youthful ailments, such as eczema, skin-eruptions, and various neuroses, insomnia, hysteria, and sometimes epilepsy. In character, whatever obstinacy exists reaches its climax.

MORBID IMMINENCES.—The inflammatory disposition is lessened, but there is a tendency to venous plethora of the abdominal viscera, and towards vicarious hæmorrhages. Gout assails its victims with well-characterised attacks. New-growths, simple and malignant, tend to demonstrate themselves, and rheumatoid arthritis to appear. At this time a man first experiences a reluctance to stoop, prefers a carriage to riding on horseback, and perceives each change of the weather affect him.

HYGIENIC RULES.—At the menopause women should be advised to abstain, as a rule, from alcoholic drinks, and to avoid highly spiced and seasoned dishes. They may be recommended to take meat not more than once daily, and to live chiefly on farinaceous food, milk, eggs, vegetables, and fresh fruits. A tablespoonful of lime-juice taken twice daily occasionally for a week or ten days at a time has a salutary, depurating effect upon both stomach and kidneys, and clears the tongue when this is foul in the morning. Riding and walking exercise are highly appropriate, but very violent muscular efforts should be avoided. If the individual be thin, and growing thinner, the clothing should be extra warm. Flannel abdominal belts may be worn advantageously in all seasons, but especially in autumn and winter.

Both sexes should avoid emotional excitement, and the stimulation of waning sexual abilities.

Prolonged exposure to wet and cold is sure to be seriously resented. Hot or Turkish baths, succeeded as they should be by the cold plunge or douches to remove the lassitude otherwise provoked, are very beneficial, and taken once a week may be safely indulged in throughout the year. It becomes increasingly important as the subcutaneous fat gets absorbed, and the skin wrinkles, to keep its pores clean and open and capable of perspiring.

9 and 10. **Advanced Life, and Old Age.**—The period of advanced life—60 to 82, and old age, from 82 upwards, may be advantageously considered together. When a man turns his toes out much in walking and treads upon the whole base of his foot, when he is always stopping and turning round to look back, he is already old. The sagacious 'boots' at an inn can tell a man's age by the state of his shoe-leather.

'*Senectus ipsa morbus insanabilis.*' Some degenerate earlier than others, but the decline of life is characterised in all human kind alike by an indurating condition of every tissue, diametrically opposed to the cellular softness and laxity of infancy. The capillaries thicken, the arteries harden, the nutritive changes proceed more slowly. The muscles waste; the subcutaneous fat lessens; the blood becomes poorer and paler; the skin dry, sallow, and wrinkled; further, it gets less vascular, and the mucous surfaces become relatively more so. The teeth loosen and fall out; the gums recede from them; and the digestive juices fail. The arteries become atheromatous and calcareous, lose their elasticity, and are liable to fibrinous thromboses, or to embolic pluggings; and while they tend to block up at one part, their coats may split and yield to pressure, bulge out, and form aneurysms or dilatations in other directions. Hence happen apoplexies, brain-softenings, and senile gangrenes. The heart up to an uncertain period grows progressively larger and more muscular, to meet the obstacles offered to the circulation; but finally it, too, degenerates, and its walls grow thinner and dilate. The air-cells of the lungs lose their elasticity, and progressively enlarge; then merge into each other; and become emphysematous at the edges of the lobes where least supported. Emphysema implies degeneration of capillaries and diminution of aerating surfaces; and as the pulmonary area becomes thus lessened, the right heart becomes hypertrophied and dilated.

The dryness and lessened secretion of the skin cast harder work upon the kidneys in eliminating water, and increase the disposition to catarrhal fluxes from the nasal passages, the bronchi, and the intestines. Thus, while there is a constant predisposition to irritation of the skin from its dryness, and to eczema from scratching and rubbing it, the other morbid imminences towards bronchial catarrh and diarrhoea very closely follow the direction given them by the season of the year, and greater or less degree of external cold. The bladder grows thicker with age, and its capacity is less; the prostate gland enlarges. Few persons after 60 pass seven hours in bed without requiring to micturate. The pulse feels firmer and fuller; fills quickly after food is taken, but falls in frequency and flags in power in a marked degree after fasting. It is a far less trustworthy indicator of the gravity of any febrile disorder, or of degrees of asthenia, than it was in youth or middle age; and it fails to point to the

practitioner the nearness of death, unless he have large experience of it.

There is a default of reaction manifest in advanced life, so that all acute disease is clinically less easy of recognition, and the beginning of the end is therefore apt to pass unobserved. The thermometer warns the doctor of changes which old people do not notice themselves, but which it may be of considerable importance to notice. A slight elevation of temperature means much in old age, and should be heeded accordingly. The slightest change excites a young child; nothing seems to move the old man. In extreme old age life is little more than vegetative existence; the individual eats and sleeps and dreams. The sleep the aged get by night seldom rests or satisfies them. Memory is one of the first mental faculties to become impaired, but finally every sense and every faculty fail. Up to 75 the strong of both sexes usually retain their digestive powers, and a fair amount of mental and muscular vigour. Later on, indecision, inconsequential reasoning, self-distrust, uncertainty as to facts, delusions of sight and hearing, restlessness, day-dreams, night-wandering, too often prove that the old are no longer what they once were in intellect, and testify to the brain-degenerations belonging to senile dementia.

HYGIENIC RULES.—A prime necessity for old age is warmth: nothing kills the aged so certainly as cold. It is of first hygienic importance after 75 that the individual should be loved and cared for; old people do not, perhaps cannot, take care of themselves.

Those who live longest, and enjoy the fullest measure of activity, are those who do not over-tax their stomachs when their teeth begin to fail them, and who adapt their aliment to their enfeebled powers of mastication, by having their food properly cooked. A moderate amount of wine or some other stimulant often cheers and comforts old people, and is better for them than overloading their stomachs with milk and farinaceous foods.

Great attention should be paid to the functions of the bowels and of the skin. Galen pointed out that old people should not suffer their bowels to remain costive beyond two days; on the third they should take some gentle purge, such as by experience they have found adequate. A hot bath once a week, and a hot foot-bath every night, may be advised. A short nap after breakfast and before dinner is the natural habit of the aged. Further, their clothing should be additionally warm, and their chambers night and day be heated. They should be encouraged to go out in the open air only in seasonable weather; and, when they are equal to it, should take a little walk on a dry gravel path in some warm locality, sheltered from north-easterly winds. All change and cheerful society are good for them. If their purses admit of it, they should follow the swallows to warm winter-quarters. If they must winter in England, let them shut themselves up throughout the season in a well-warmed house.

SUMMARY.—Advice for every age may be thus briefly given: for infancy and childhood—*sustine*; for adult years—*sustine et abstine*; for old age—*sustine* again.

The hygienist, however, seeks not to lengthen out the days of age and decrepitude; his art is not to prolong life beyond its natural term, though this may come subordinately, but to render its period of

activity and utility longer. Some cynic observes that we have pointed out very few habits as worth cultivating, the truth being we believe what we have insisted on—that most bodily habits need resisting. Individual health is attained by self-denial; habits imply self-indulgence. REGINALD SOUTHEY.

H. MONTAGUE MURRAY.

PERSPIRATION, Disorders of.—See SUDORIPAROUS GLANDS, Diseases of.

PERTUSSIS (*per*, signifying excess; and *tussis*, cough).—A synonym for whooping-cough. See WHOOPING COUGH.

PESTIS (Lat.).—A synonym for plague. See PLAGUE.

PETECHIÆ (*petigo*, an attack, eruption; Ital. *Petechie*, fleabites).—SYNON.: *Peticula*; Fr. *Pétichies*; Ger. *Petechien*.

DESCRIPTION.—Petechiæ are small crimson or purple spots in the skin, resembling those that result from the bite of a flea, and due to minute hæmorrhages. See PURPURA.

PETIT MAL (Fr.).—A term applied to attacks of epilepsy which are of short duration and slight intensity. See EPILEPSY.

PETRI-DISH.—A circular glass dish, about 10 c.mm. in diameter, with perpendicular sides, usually 1.5 c.mm. deep, loosely covered by an inverted dish of similar form but of slightly greater diameter. It is used in the plate-cultivation of bacteria.

PETRIFICATION.—See CONCRETIONS.

PFÆFFERS, in Switzerland.—Simple thermal waters. See MINERAL WATERS.

PHAGEDÆNA (*pháγω*, I eat away).—A form of ulceration, which rapidly destroys the surrounding parts. See BUBO; GANGRENE; ULCER AND ULCERATION; and VENEREAL SORE.

PHAGOCYTOSIS (*pháγω*, I eat; and *κύτος*, a corpuscle).

DEFINITION.—A term expressing the property possessed by certain animal cells, called 'phagocytes,' of taking into their substance solid particles, which may either be rejected, be used by the cell for its nutrition, or, when the solid mass is living, may destroy the cell.

Phagocytosis, from this wide definition, is thus a process not limited to diseased conditions of the animal body, but is a part of a general property possessed by cells in animals of every grade, invertebrates and vertebrates. The cells which act as phagocytes are not highly differentiated: they are not, like the muscle-fibre or the nerve-cell, organised to perform special functions, but they possess the characteristics inherent in undifferentiated protoplasm. Two of the chief properties of these cells are the amoeboid movement or power of contractility of the protoplasm, and the power of digestion. They are, moreover, very sensitive to stimuli, both chemical and physical, which diminish or increase their movements, or which attract them to one spot. To a certain extent they show a selective power, that is, they refuse to take in some solid particles; but how far this power is generalised in the different cells which act as phagocytes it is

at present impossible to say. The relation of phagocytosis to disease is in part discussed in the article on IMMUNITY. The anatomical facts which have been discovered by a study of phagocytosis will alone be discussed in the present article. For convenience, it is best to consider the subject under two headings—(1) *physiological*, and (2) *pathological phagocytosis*.

1. *Physiological Phagocytosis*.—Examples of this occur in both the most lowly and the highest animals. The amoeba takes into its substance solid particles which, if suitable for the nutrition of the organism, undergo digestion, and, if unsuitable, are ejected after remaining in the substance of the protoplasm for some time. The ciliated animalculæ (of which Paramœcium may be quoted as a type) also take into their substance particles of the nature just mentioned, which undergo a similar process. This intracellular digestion is also a feature in Hydra, sponges, and other allied animals. In the higher animals there are many examples of this phagocytosis, for example, in the absorption of fat in the small intestines, in which the globules are taken up by the epithelial cells and then by the leucocytes, by which they are conveyed to the lymph-stream; in the absorption of bone during ossification and in old age by the osteoclasts; and in the absorption of the branchiæ and tail of the tadpole during its transformation into a terrestrial animal. These processes are performed by cells, playing the part of scavengers. It is sufficient to mention these facts to bring them into line with what more nearly concerns disease, namely, pathological phagocytosis.

2. *Pathological Phagocytosis*.—This is observed when a living organism is injured mechanically or infested by a parasite, or when an inert foreign body is introduced into its substance. It is a battle between the host and the parasite, in which in some cases the host, in some the parasite, gains the upper hand. Parasitism may not inconveniently be divided into two varieties: in the one, the parasite, by the bulk of its growth, may be fatal to the host by interfering with the essential functions of the organism, or of a vital organ; in the other, the parasite acts on its host not so much by the bulk of its growth as by the poisonous products which it excretes or forms from the tissues of its host. Infective disorders in man and the higher animals, so far as we know them, belong chiefly to the second variety of parasitism; to the first variety belong some of the cases of parasitism in the lower animals; perhaps also in man (e.g. animal parasitism). Whatever truth there may be in this division of parasitism, it must be borne in mind that it is not yet clear that every noxious parasite is capable of secreting products which are poisonous to the host. To illustrate this question of parasitism in the animal kingdom and its relation to phagocytosis, the description given by E. Metchnikoff in his *Leçons sur la Pathologie comparée de l'Inflammation* (1892) will be adhered to.

Commencing with the lowest in the animal grade, the *unicellular organisms*, such as the amoeba, it has been mentioned that the solid living particles (diatoms, bacteria, &c.) ingested by them may serve as food; but some of the living bodies absorbed, instead of being digested, may actually produce a fatal disease. Thus, as Metchnikoff has shown, the amoeba may take into its substance a microsphaera (an organism composed of nucleated round-cells,

multiplying by division) which multiplies to such an extent as to kill the amoeba. This is an example of fatal infection of a unicellular organism by a parasite. Examples of similar parasitism in other lowly plants and animals are well-established. In some instances the living bodies, diatoms and bacteria, are ingested and digested by the organism, in other instances the ingested living body multiplies and destroys its host.

The study of phagocytosis becomes more complicated in *multicellular organisms*. Of the three layers of cells of which the majority of these organisms are composed, namely, the ectoderm, endoderm, and mesoderm, it is the last to which the property of phagocytosis is eventually limited in the process of evolution. This limitation of the phagocytosis to the mesodermic cells is attained only gradually. The sponges, animals composed chiefly of ectoderm and endoderm, with a well-developed mesoderm, protect themselves against harmful bodies by means of their contractile ectodermic cells, but also by the phagocytic power of the mesodermic and endodermic cells. In them the mesodermic cells play a large rôle in the normal digestion of the organism. Some of the Coelenterata possess no mesoderm, and in these (Hydra, for example) the endodermic cells, which have the property of sending out amoeboid processes and of performing the normal digestion of the organism, play the rôle of phagocytes; in other similar animals, the ectodermic cells are also phagocytes. Such animals are without any vascular system. This is also absent in the Medusæ, which, however, possess a mesoderm differing from that of the sponges in the fact that the mesodermic cells play no part in the normal digestion of the animal. These mesodermic cells are, however, the phagocytes. They surround and attack harmful substances introduced into the substance of the animal, and may unite together, forming *plasmodes*, an example of the formation of giant-cells low down in the animal scale. In all animals higher than the medusæ, namely, echinoderms, worms, vertebrates, this formation of plasmodes, or giant-cells, by the union of mesodermic cells is very common, the plasmode in some instances completely surrounding the foreign body or parasite, as in fig. 1.

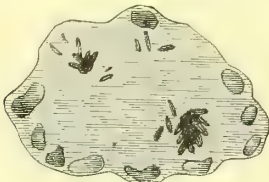


FIG. 1.—A Giant-cell in Actinomyces enclosing the clubs of the fungus (700 diam.). Ruffer.

The Classes Mollusca and Arthropoda possess a vascular system, filled with a liquid containing colourless cells. These cells are mononuclear; their protoplasm is in one variety granular, in another hyaline. They act as the phagocytes of the animal. Numerous experiments have shown that, after a lesion of a part of the animal, these cells accumulate near the injured spot as they do in an 'inflamed' area in warm-blooded animals. A similar accumulation occurs around inert foreign bodies introduced into the animal; and the cells also take up pigment-granules injected into the tissues

(Haeckel, 1862). The best example of infection in such animals has been brought forward by Metchnikoff as occurring in the water-flea, *Daphnia magna*, which in a pond was found infested with a kind of yeast-fungus, *Monospora bicuspidata*. At certain periods an epidemic of this disease will kill off nearly all the daphniæ in the pond. A study of the process shows that the ripe spores of the fungus are eaten by the daphnia, and pass through the intestinal wall into the body cavity. Here the elongated spores become surrounded by the leucocytes, and one of two events may happen. The spores may rapidly increase in number and fill the body-cavity, eventually destroying the life of the daphnia; or, becoming surrounded by leucocytes, they undergo degeneration, so that they are killed or are not fatal to the daphnia.

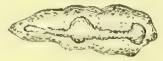


FIG. 2.—A phagocyte of the *Daphnia* containing two undegenerated spores of the *Monospora*. (Metchnikoff, Virchow's *Archiv*, vol. xvi.)

FIG. 3.—A spore of the *Monospora* enclosed by phagocytes, and undergoing degeneration. (Metchnikoff, *ibid.*)

In both cases there is a struggle between the leucocytes and the germinating fungus: in the first, the fungus overcomes the resistance of the leucocytes; in the second, the leucocytes surround and finally digest the spores of the fungus.

In the very young tadpoles of the lower amphibia (urodeles) phenomena similar to those occurring in mollusca have been observed by Metchnikoff and others. Thus, after a slight injury to the non-vascular fin of the tadpole, the amoeboid cells collect at the injured spot, the fixed cells of the tissue taking no part in the process. In older tadpoles, when the blood-vessels are developed in the fin, there is still this accumulation of amoeboid cells at the injured spot; but there are also the phenomena of inflammation such as are observed in the higher animals, namely, acceleration of the blood-stream, &c. As Metchnikoff points out, both the phenomena in the very young and those in the older tadpole must be classed as 'inflammatory,' although in one case there is no vascular change. Similarly, the phenomena following an injury or an infection which have been described as occurring in invertebrate animals possessing a mesoderm must be classed as inflammatory; not as belonging to a completely developed process of inflammation, but as forming one of the stages in the evolution of the inflammatory process in the animal kingdom.

Phagocytosis in the *higher animals* is a part of inflammatory and of infective processes. All primary inflammatory processes are infective, that is, due to a living infective agent. The study of the nature of inflammation and that of the process of infection are in reality to be conducted on the same lines, both being due to the introduction into the healthy organism of a harmful agency, which may either remain localised in its action, or may become generalised.

The cells which act as phagocytes are derived from the middle layer of the embryo (mesoderm), and are some varieties of leucocytes, the endothelial cells of the vessels, and the elements of the splenic pulp. The endothelial cells possess the power of contractility in many animals. In leprosy they not uncommonly contain large masses of the *Bacillus*

lepræ. In rabbits inoculated intravenously with the *Bacillus tuberculosis*, these cells take up the bacillus; a similar phagocytosis occurs with the bacillus of swine-erysipelas. The endothelial cells and the elements of the splenic pulp are also macrophages.

The varieties of leucocytes, including those possessing phagocytic action, are described on p. 156.

Leucocytes can digest suitable particles taken into their substance. This digestive power is a property inherent in all original cells. In some cases of infective disease, the living bacteria have been observed to undergo this digestion in the phagocyte; they lose their protoplasm, leaving the investing membrane, which also finally disappears; and they also lose the property of being stained by some aniline dyes, such as vesuvine. Examples of this are found in recurrent fever in the ape, and in the *Streptococcus erysipelatis* in man (Metchnikoff). Some of the bacteria may be digested and others not, the latter infecting the organism generally. This has been observed in tuberculosis and in swine-erysipelas.

It has been a question whether the bacteria taken in are living or not; but it has been proved by Metchnikoff that they are in some cases not only living, but virulent, since a single phagocyte of a pigeon (an animal refractory to anthrax) containing anthrax-bacilli has been separated, and the bacilli cultivated in a suitable medium, proving on inoculation fatal to mice, &c.

The two varieties of leucocytes which act as phagocytes appear in some instances to be selective in their action. Thus in man it is the neutrophile leucocyte which takes in the *Streptococcus erysipelatis* and the gonococcus; the mononuclear leucocyte remaining inactive. On the other hand, the mononuclear leucocyte is the phagocyte of the *Bacillus lepræ*; the neutrophile variety remaining inactive.

Instead of digesting the bacterium, the phagocyte may prevent it developing. Thus the spores of anthrax injected into fowls and frogs (which are refractory to the disease) commence to germinate, but are taken up by the leucocytes. If, however, the body-temperature of the fowl be lowered by cooling the animal, or that of the frog increased by warming, the activity of the leucocytes is diminished, and the spores then germinate rapidly, the bacilli invading the whole body.

In other cases the leucocytes do not take in the infective agent, as in mice and guinea-pigs infected with anthrax, and in pigeons and rabbits infected with fowl-cholera; and according to Metchnikoff the more virulent the infection, the less phagocytosis occurs.

Why the leucocyte, possessing as it does the property of absorbing solid particles, refuses in one instance to act as a phagocyte, or why in another only one variety of leucocyte acts as a phagocyte, although both are present, is perhaps in part explained by what has been described as *chemiotaxis* (see IMMUNITY). Certain substances have the power of attracting leucocytes, others actually repel them—positive and negative chemiotaxis. Thus most bacteria, living or dead, as well as their chemical products (toxins), papain, and leucine attract leucocytes; while the most virulent bacteria, strong solutions of sodium and potassium salts, alcohol, chloroform, glycerine, bile, quinine, and

jequirity repel them. Some substances act in a neutral manner.

Chronic infective processes.—The best examples of such processes are tuberculosis and leprosy. The formation of miliary tubercle has been ascribed to the proliferation of the fixed cells of the organ affected, that is, the alveolar cells of the lungs, the hepatic cells, &c.; leucocytes playing but little part in the formation. This is the view usually held. From a study of tuberculosis in the early stage, produced by the intravenous injection of the bacillus in rabbits, Metchnikoff has concluded that in the liver the tubercle is formed not by the hepatic cell, but by the phagocytes, the mononuclear leucocytes and the endothelial cells. The agglomeration of phagocytes forms the tubercle, the fusion of some of the cells forming giant-cells; the tubercular nodule is therefore purely mesodermic in origin, and is produced by the phagocytes accumulating to attack the bacilli. The polynuclear leucocytes readily absorb the bacilli, but soon die, being absorbed by the mononuclear leucocytes (the macrophages). The macrophages are more resistant to the bacilli, and may destroy them. In leprosy, also, Metchnikoff considers that the cells containing the bacilli are phagocytes struggling against their invasion; the leprous nodule would thus be ranged with the tubercular nodule as one of purely mesodermic origin.

SIDNEY MARTIN.

PHANTOM TUMOUR.—SYNON. : Hysterical Tympanites; Spurious Pregnancy.

DEFINITION.—A peculiar enlargement of the abdomen, occurring in females belonging to the hysterical class. It is supposed by the patient to be an actual tumour, or to be due to pregnancy, but it can be made at once to disappear by placing her under the influence of chloroform.

DESCRIPTION AND DIAGNOSIS.—The phantom tumour is characterised by a more or less general prominence of the abdomen forwards, varying in degree. The enlargement may attain a considerable size, but is always quite symmetrical. The projection is most marked about the middle of the abdomen, and usually a depression or constriction is observed below the chest and above the pubes. It is rounded, smooth, and quite regular, presenting a uniform soft feeling, quite distinct from that of gaseous distension, fluid accumulation, or a solid mass. The enlargement is peculiarly movable, as a whole, from side to side. There is no sense of true fluctuation. Percussion yields a resonant note, but not usually excessive, and it may be of a muffled character. On examination *per vaginam* nothing abnormal can be detected, such as would be associated with ovarian or uterine enlargements, or with pregnancy. Should there be any doubt whatever as to the nature of the supposed tumour, it may be at once cleared up by placing the patient under the influence of chloroform or other anæsthetic, when the swelling immediately disappears, the abdomen becoming quite flat; but it gradually returns, even before the patient returns to consciousness, on the removal of the anæsthetic. There is no pain or tenderness in connection with the enlargement; nor are any symptoms due to pressure or other causes observed: while the patient usually presents distinct signs of the hysterical condition. There ought, therefore, to be no difficulty in the diagnosis of a phantom tumour. What is the actual cause of the enlargement is

by no means clear, but most probably it is due to a kind of paralysis of the intestines depending upon disordered innervation.

TREATMENT.—In a patient having a phantom tumour, the general and medicinal treatment for hysteria is that principally called for. She should be constantly impressed with the fact that the enlargement is not really a tumour, and is of no consequence. The condition is by no means easy to get rid of, but for this object galvanism may be applied to the abdomen, or, in obstinate cases, the patient may be put repeatedly under chloroform. The use of pressure, by means of an abdominal bandage or elastic apparatus, might be serviceable in some cases. The bowels should be kept freely opened.

FREDERICK T. ROBERTS.

PHARYNX, Affections of.—**SYNON.:** Fr. *Maladies du Pharynx*; Ger. *Krankheiten des Pharynx*.—In this article the affections of the pharynx will be described under the following headings:—

1. Acute Inflammation of the Pharynx, p. 1216.
2. Chronic Inflammation, including Hypertrophic Pharyngitis, and Atrophic Pharyngitis (*Pharyngitis sicca*), p. 1216.
3. Acute Infectious Phlegmon, p. 1217.
4. Erysipelatous Pharyngitis, p. 1217.
5. Gangrene of the Pharynx, p. 1217.
6. Retro-pharyngeal Abscess: (a) acute primary form, (b) chronic secondary tubercular form, p. 1217.
7. Syphilis of the Pharynx, p. 1218.
8. Tuberculosis of the Pharynx, p. 1219.
9. Tumours, Innocent and Malignant, p. 1219.
10. Pharyngocele (pressure-pouch), p. 1219.
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1. Acute Inflammation of the Pharynx.—**SYNON.:** Acute Pharyngitis.—This affection is commonly catarrhal in type, and associated to a greater or less extent with acute inflammation of the tonsils. It may owe its origin (1) to exposure to cold and damp; (2) to a rheumatic or gouty constitution; (3) to the poison of an infectious disease, especially syphilis and scarlet fever; (4) to certain drugs; and (5) to a direct irritant, such as burns and scalds, injury, hot condiments, raw spirits and tobacco-smoke. It is probable that most of the cases classed under (1) and (2) are caused by micro-organisms, the condition appearing at times to be of an infectious nature.

SYMPTOMS.—There is at first a dryness and feeling of heat, stiffness and discomfort in the pharynx, which is increased to actual pain on swallowing. Soon the secretion, which was checked for a time, increases in amount and becomes mucoid and sticky, or muco-purulent in character. The patient has a constant desire to hawk and clear the throat. The soft palate, tonsils and walls of the pharynx are generally congested and swollen, and the uvula appears oedematous, and greatly increased in size. The inflammation has a marked tendency to pass upwards into the naso-pharynx, where it may involve the mucous lining of the Eustachian tubes, or downwards into the larynx, causing hoarseness or temporary loss of voice. Unless there is much tonsillar involvement the constitutional symptoms, elevation of temperature and increase of pulse-rate, are slight.

Frequent attacks of acute inflammation may give rise to the chronic form of pharyngitis, and in adults acute inflammation, particularly in smokers and drinkers of raw spirits, is apt to supervene on a pre-existing chronic pharyngitis. It is to be borne in mind that acute inflammation of the pharynx may, in its early stages, be confounded with other far more serious diseases—e.g. diphtheria, scarlet fever, &c. If the constitutional disturbance be out of proportion to the local inflammation the practitioner should be on his guard lest he mistake for a simple acute pharyngeal catarrh a pharyngitis toxæmic in origin and due to a grave infectious disease.

TREATMENT.—In the uncomplicated cases the inflammation tends naturally to run a short course. A saline aperient, placing the feet in hot water, and a dose of Pulv. Ipecac. Co. (gr. viij to xij) are generally sufficient. A fomentation round the neck affords relief if there be stiffness and pain, and slight enlargement of the cervical lymphatic glands. Steam inhalations (Tinct. Benzoini, ʒj to the pint of hot water) are useful, if there be laryngeal involvement (p. 745). In cases that are distinctly rheumatic in origin salicylate of sodium, and in sthenic gouty inflammations colchicum with citrate of lithium, will be indicated. Guaiacum or krameria lozenges may be given as soon as the secretion from the mucous glands has become free and the acute inflammatory swelling has subsided.

2. Chronic Pharyngitis, including hypertrophic and atrophic pharyngitis (*Pharyngitis sicca*). Chronic inflammation of the pharynx may be the sequel of repeated acute attacks, or may occur in persons who have not suffered from acute pharyngitis, but who are great smokers, or drinkers of spirits, or who habitually overstrain the voice, as in the case of clergymen, auctioneers, costermongers, &c. It is frequently found in association with chronic naso-pharyngeal catarrh. In certain cases the inflammation appears to be of a chronic catarrhal nature, in others there is little to be seen but hypertrophic enlargement of the lymphoid nodules of the pharynx. No useful purpose is served by describing these types as separate conditions, since commonly they are found in association, though one or other may predominate.

The term 'atrophic pharyngitis' or 'pharyngitis sicca' is frequently applied to a chronic condition in which the mucous membrane of the pharynx is thinned, smooth and dry from deficient secretion of mucus; and complaint is made of abnormal dryness of the throat. This condition may be found in association with atrophic rhinitis, but frequently occurs independently. In chronic catarrhal cases the mucous membrane of the pharynx is relaxed; it has a slightly congested appearance, and dilated venules are seen on the posterior pharyngeal wall, which is often partly hidden by sticky plugs of tenacious mucus. The soft palate, tonsils, and uvula frequently present a similar condition. In the hypertrophic or granular form numerous small red gelatinous-looking granules are seen on the posterior wall of the pharynx. These are due to an hypertrophy of the lymphoid tissue of the pharynx which is in general collected around the muciparous glands. Frequently there is also seen a lateral fold of thickened mucous membrane and submucous tissue lying behind the posterior pillar of the fauces and simulating a third faucial pillar (*lateral hypertrophic pharyngitis*). This condition

of hypertrophic pharyngitis in some cases gives rise to no symptoms; in others, particularly in debilitated, neurotic or dyspeptic individuals and in women at the menopause, great complaint is made of loss of voice, heat, a perpetual desire to clear the throat and a certain amount of pain and difficulty in swallowing. In children a condition of enlargement of the lymphoid nodules in the pharynx is often seen and is invariably associated with post-nasal 'adenoid' vegetations.

TREATMENT.—So many cases of this affection are dependent upon a general constitutional condition that attention must always be first directed to the patient's general health. Thus, if gout, rheumatism, dyspepsia, portal engorgement, anæmia, or a general neurotic condition be present, appropriate general treatment must be combined with topical applications to the pharynx. Smoking and over-use of the voice must be prohibited; and rest, bracing air, and tonics enjoined. Topical applications are of limited value. For the removal of tenacious mucus, a solution of bicarbonate of sodium and borax (of each ʒj to eight ounces of warm water) may be employed as a spray, and iodised glycerine (℞ Iodi, gr. vj; Potassii Iodidi, gr. xij; Ol. Menth. pip. ℥viii; Glycerini, ad ʒj), or glycerine of tannic acid may be applied to the pharynx by means of a camel's-hair brush. The most satisfactory results in the treatment of chronic pharyngitis are obtained in the cases in which the granular lymphoid hypertrophy is well marked. The destruction of the granular nodules by means of the galvano-cautery gives marked and often lasting relief. Some cases of chronic pharyngitis defy all treatment, particularly those in which the patient declines to give up the habit of smoking. It is a fact difficult to explain, but undoubted, that destruction with the galvano-cautery of granular lymphoid nodules not infrequently cures the loss of voice from which patients may suffer. Spraying with a 5-per-cent. solution of hydrochloride of cocaine renders the application of the electric cautery quite painless. In atrophic cases (pharyngitis sicca) the local application of glycerine diluted with an equal quantity of rose-water relieves the sensation of dryness in the throat.

3. Phlegmonous Pharyngitis (Acute infectious phlegmon of the pharynx).—This most formidable affection is, fortunately, of rare occurrence. It may be traumatic in origin, but in not a few instances no history of injury can be obtained. It commences as a severe acute pharyngitis; the mucous membrane becomes deeply congested and oedematous, the lymphatic glands enlarge and the neck increases in bulk and becomes infiltrated and brawny. The constitutional symptoms are intense, and there is great elevation of temperature and rapidity of pulse-rate. Deglutition is impossible; and pharyngeal oedema along with laryngeal involvement (oedema glottidis) cause extreme dyspnoea or even death from suffocation. In other cases death results from septicæmia or exhaustion. At the *post-mortem* examination there are found intense diffuse purulent infiltration of the mucous membrane and submucous cellular tissues and the general appearances of septicæmia. In marked examples of this terrible condition treatment is unavailing, and death always results.

4. Erysipelatous Pharyngitis.—By many erysipelas of the pharynx is held to be identical in origin with phlegmonous pharyngitis; but, though

there is much to be said in favour of this view, the clinical differences are so great that it is well for the present to consider them two distinct pathological conditions. Erysipelas may attack the pharynx primarily or may be secondary to facial erysipelas, the latter being of far more frequent occurrence. The symptoms are practically those described as occurring in phlegmonous pharyngitis, but are much less severe. Suppuration is very exceptional in erysipelas. The process may spread to the larynx, causing oedema and intense dyspnoea. There is rapidity of pulse, high fever, and marked constitutional disturbance; albuminuria occurs in almost every case, and not infrequently delirium and coma. The diagnosis is easy in cases secondary to erysipelas of the face, but is extremely difficult in the rare primary examples of the disease. Erysipelas, though frequently fatal, is, as a rule, recovered from. When death results it is in general due to heart-failure, acute oedema of the larynx, or pneumonia.

TREATMENT.—The patient may be given ice to suck, and a cloth wrung out of iced water and frequently changed may be wrapped round the neck. Nourishing fluid food with brandy and digitalis (especially when the pulse is feeble) and quinine must be administered. Tincture of perchloride of iron is, in the writer's opinion, useless. Should symptoms of dyspnoea arise due to acute oedema of the larynx, free scarification of the mucosa may be tried; and if this fail rapidly to relieve the dyspnoea, intubation of the larynx or preferably tracheotomy must be at once undertaken.

5. Gangrene of the Pharynx is rare. It may be a sequel to either of the two last-described affections or may be met with in syphilitic disease of the pharynx. It is a very rare complication of diphtheria, scarlet fever, measles, typhoid fever, and small-pox. In greatly debilitated patients it may supervene on divers forms of ulceration of the pharynx. The gangrene may occur in patches, or a large portion of the pharyngeal wall (diffuse gangrene) may be involved: in the former type recovery commonly takes place after separation of the sloughs; in the latter extreme fetor of the breath, enlargement of the cervical lymphatic glands, profound septicæmic symptoms with high fever and rapid pulse occur, and death from heart-failure or septicæmia results.

TREATMENT.—Inhalations of steam charged with some volatile antiseptic (carbolic acid, thymol or eucalyptus) may be used; or the pharynx may be sprayed with a solution of biniodide of mercury (1 to 3,000) or permanganate of potassium, or be painted with iodised glycerine. Nourishing fluid food must be administered, and stimulants and digitalis will be needed to combat the intense prostration. Treatment is, as a rule, of little value, most cases terminating fatally.

6. Retro-pharyngeal Abscess.—This consists of a collection of pus between the posterior wall of the pharynx and the bodies of the cervical vertebræ. Two types of the affection, differing greatly in their origin, course and prognosis, must be separately described: (1) the acute primary; (2) the chronic, commonly tubercular, type.

(1) Acute primary retro-pharyngeal abscess is the more frequent and more formidable type. It may be met with at any age, but the great majority of cases occur in very young children, between the third and twenty-fourth months of life. In these young patients it always commences as a suppurative

adenitis, affecting definite lymphatic glands, which are placed in front of the upper cervical vertebræ on either side of the middle line of the neck and between the posterior pharyngeal wall in front and the prevertebral aponeurosis behind. These glands are well marked in infants and very young children, but diminish greatly in size, or even disappear entirely, after the seventh year. This suppurative lymphadenitis is the result of infection with micro-organisms, almost always streptococci, which have gained entrance through some small abrasion of the mucous membrane of the pharynx or naso-pharynx, or even of the face (cracks and fissures, or possibly as the result of eczema). Though the vast majority of cases of acute retro-pharyngeal abscess commence as lymphadenitis, the inflammation may commence in the connective tissue as a result of mechanical injury or some ulceration of the pharyngeal mucous membrane. Acute abscesses in the case of adults are of this variety, but such are quite exceptional. Acute retro-pharyngeal abscess tends to occur chiefly in debilitated children, and in those who suffer from syphilis, tuberculosis or acute infective diseases, in which the resistance of the tissues to microbic infection is greatly lowered. In certain cases, however, children apparently perfectly healthy are suddenly attacked. The pus is commonly very foetid. The condition develops rapidly; there is high fever, marked snoring at night, and pain in the throat, greatly increased by swallowing. In certain cases food taken regurgitates through the nose. Dyspnoea becomes urgent; cyanosis appears; there is stridor and often cough. The cervical lymphatic glands are enlarged and the neck is held stiffly, as all movements are attended with much pain. On inspection of the pharynx—provided that there be no difficulty in opening the mouth, as is sometimes the case—the posterior wall of the pharynx is seen deeply congested and bulged forwards; and fluctuation may be made out, almost always in the pharyngeal swelling, and sometimes externally in the neck about the angle of the jaw. In some cases the bulging occurs chiefly in the naso-pharynx, in others in the laryngeal region; in these latter dyspnoea from laryngeal oedema is particularly threatened.

(2) The chronic, commonly tubercular, form is the less frequent type of retro-pharyngeal abscess. It is almost always dependent upon tubercular disease of the bodies of the cervical vertebræ, though it may in rare instances be due to cold abscess arising in connection with the prevertebral glands, unassociated with vertebral caries. The affection is free from the marked constitutional disturbances described in connection with the acute form. Its course is chronic; there is little or no elevation of temperature or pain referred to the pharynx. Commonly symptoms of disease of the bodies of the cervical vertebræ precede the formation of an abscess; and there is generally rigidity and deformity of the neck. In contradistinction to the acute form this affection is found in children or young adults, and is quite unusual in infants under twelve months old. The pharyngeal swelling and fluctuation are, as a rule, easily recognised. The prognosis, owing to the almost constant association with vertebral caries, is unfavourable.

TREATMENT.—It must be borne in mind that if the abscess burst spontaneously death almost inevitably results, owing to the pus gaining entrance to the respiratory passages and causing suffocation

or septic pneumonia; and that even before bursting of the abscess can occur a fatal result may be caused by spasm of the glottis, oedema, or asphyxia, due to the bulk of the abscess obstructing the lower pharynx. Surgical treatment is therefore always demanded. In very urgent cases a vertical incision may be made into the abscess by a knife introduced from the mouth; but this method is objectionable, owing to the risk of pus gaining access to the air-passages, and the probability of septic infection of the abscess-cavity occurring, owing to the open wound communicating directly with the mouth. If this method be employed at all it must be with the child so placed that the pus will tend to run out of the mouth rather than into the larynx, i.e. the mouth must be inclined downward. It is far better to make an external incision along the posterior margin of the sterno-mastoid (Chiene), and after incising vertically the deep cervical fascia and drawing forward the great vessels of the neck, to puncture the abscess and dilate the opening with sinus-forceps. A drainage-tube is then introduced, and in cases secondary to tubercular disease of the vertebral bodies the latter may be attacked directly, any carious or necrotic portions being removed. This operation has the great advantage that it can be aseptically carried out. It must be invariably undertaken in all chronic tubercular cases, and is to be recommended as a rule in the acute type, except perhaps in those instances in which, owing to the abscess being on the point of bursting, or to extreme dyspnoea, the result of oedema of the larynx, the surgeon fears to administer anæsthetic. In such cases incision of the abscess through the mouth is to be performed.

7. Syphilis of the Pharynx.—Syphilis may attack the pharynx in the primary, secondary, or tertiary stage, and may be acquired or of congenital origin.

Primary chancre may occur on any part of the pharynx, but the tonsil is by far its most frequent site. The lesion is seldom characteristic, induration being frequently absent or little marked, and ulceration often very pronounced. Not infrequently a correct diagnosis is only arrived at on the appearance of secondary manifestations of the disease.

Secondary syphilis commonly affects the pharynx, though the parts that suffer most are the tonsils and anterior faucial pillars. An erythema especially involving the above-named parts, and causing shedding of the swollen epithelium, gives place to erosions, and several of these may coalesce to form large irregular or serpiginous patches. These erosions chiefly affect the free margin of the soft palate, the tonsil and pillars of the fauces, and much less frequently are situated on the posterior wall of the pharynx.

Mucous tubercles (*plaques muqueuses*) are common in the same situations. Such ulceration as occurs in secondary syphilis is almost always superficial, and is generally the result of the breaking down of mucous tubercles. The ulcers have a greyish base and well-defined edges, and though they extend superficially they remain shallow. The suffering caused by these lesions seldom exceeds a moderate degree of soreness, thus differing markedly from the intensely painful tubercular ulcerations. For secondary syphilitic ulcers and erosions, in addition to mercurial treatment, the lesions should be touched twice daily with a 2-per-cent. solution of chromic acid.

In *tertiary syphilis*, the pharynx may be the seat of a localised gumma, and the usual situation of this lesion is the posterior wall of the upper half of the pharynx, most commonly in the naso-pharynx, where the swelling is hidden from view by the soft palate. When such a gumma breaks down a deep excavated ulcer (not infrequently involving the vertebral bodies) is seen, with a foul discharge, sloughy base and circular margin, external to which is a ring of intensely congested mucous membrane. Owing to the great loss of substance which these ulcers occasion, much deformity from cicatrization is prone to occur. When the soft palate and the posterior pharyngeal wall are simultaneously involved, adhesion between these parts may result. Stenoses of the pharynx, described below, commonly owe their origin to tertiary syphilis. A puckered radiating cicatrix on the posterior wall of the pharynx is not infrequently found as a result of a healed tertiary ulcer. Gummatous ulcers are wonderfully amenable to treatment with iodide of potassium.

8. Tuberculosis of the Pharynx.—This is a somewhat rare affection and is scarcely ever met with as a primary disease, being almost always secondary to pulmonary or laryngeal tuberculosis. In such cases it is a late symptom and frequently leads to a rapidly fatal termination. It is not uncommon to find a more chronic form of tubercular disease of the pharynx in patients suffering from lupus of the face or nasal passages. The disease presents itself in the pharynx in (1) an acute and (2) a chronic type. The disease commonly commences in the soft palate or pharynx as a thickening of the mucous and submucous tissue in which tubercles develop. Soon these break down into ulcers which are shallow and increase in size superficially by coalescence one with another. Around these ulcers, which have greyish cheesy bases, numerous miliary tubercles are seen covered by gelatinous transparent mucous membrane. These miliary tubercles are characteristic of the disease. Perforation of the soft palate or pharyngeal wall is quite unusual. The affected parts become infiltrated and stiff, and all movement is inefficiently performed. Pain is extreme. Swallowing may cause such agony that the patient refuses food rather than attempt the act of deglutition. There ensue the usual symptoms of advanced tubercular disease—wasting, fever, night-sweats, and marked prostration, end ng in speedy death. In the more chronic form, sometimes described as lupus of the pharynx, there are fewer tubercles, much nodular thickening, and a more chronic callous form of ulceration. In both forms of pharyngeal tuberculosis it is the rule to find the neighbouring lymphatic glands enlarged and caseous.

TREATMENT.—In the most advanced cases palliative treatment alone is possible, e.g. spraying the throat with a 2-per-cent. solution of cocaine hydrochloride or painting the part with a solution (15 per cent.) of menthol in olive-oil or paraffine. In all other cases an attempt at eradicating the disease must be made, for though permanent cure is exceptional, marked improvement often results for a time, at least, from curetting with a sharp spoon the cocaineised pharynx or soft palate, and then applying daily a solution (25 to 75 per cent.) of lactic acid. The general treatment of tubercular disease will be at the same time undertaken.

9. Tumours of the Pharynx, Innocent and Malignant.—The pharynx is not a common seat of tumours. Among innocent growths papillomata alone are not infrequently found. They may be situated on any part of the mucous membrane, most frequently on the margins of the palate, fauces and uvula, and on the posterior wall of the pharynx. Fibromata, lipomata, nevi and cysts are very rarely met with. These tumours give rise to symptoms only when they are of considerable size. Fibromata and lipomata tend to assume a pedunculated appearance. Such tumours as well as papillomata may generally be cut off with scissors, or, if of large size, removed by the galvano-cautery snare. Sessile growths must be enucleated after dividing the mucous membrane underneath which they lie.

Malignant tumours occur more frequently in the pharynx than do innocent growths. Sarcomata are rare, epithelioma being the form of malignant tumour commonly met with. The site of the epithelioma is commonly on the lateral or posterior wall of the pharynx, but primary pharyngeal epithelioma is much more uncommon than an extension of the disease from tongue, tonsil or soft palate on to the walls of the pharynx. In epithelioma pain is a prominent symptom, and the act of deglutition causes so great suffering that the patient refuses food rather than endure the agony attendant on swallowing it. There is a sensation as of a foreign body in the pharynx, intense pain in the ear if the cancer be on the lateral wall of the pharynx, enlargement of the cervical lymphatic glands, and putrid blood-stained mucoid secretion which flows from the mouth. There is frequently difficulty in opening the jaws sufficiently widely to enable inspection of the pharynx to be satisfactorily undertaken. In sarcoma of the pharynx the above symptoms may be present but in a less marked degree, the bulk of the tumour rather than the pain it causes occasioning the chief part of the symptoms.

When ulceration has occurred in malignant disease it sometimes closely simulates the appearance found in tertiary syphilitic disease, and the administration of iodide of potassium may be required to clear up the diagnosis.

TREATMENT of malignant disease of the pharynx consists whenever possible in removing the cancer, taking care to cut widely clear of the disease. The operation is not infrequently successful in sarcoma, but in epithelioma recurrence of the disease is almost invariable. The tumour may be attacked from the mouth, but this gives in all cases insufficient access to the growth. Sub-hyoid or lateral pharyngotomy is a much superior method, and one or other should in all cases be employed (for details concerning these procedures works on operative surgery must be consulted). Epithelioma of the pharynx in the condition in which it commonly comes under the notice of the surgeon, with considerable glandular involvement, is as a rule inoperable, or at any rate operations undertaken for its cure prove futile. In such cases palliative treatment is all that remains to the practitioner. This may be summed up as consisting of spraying with a solution (2 per cent.) of cocaine prior to the taking of food, morphine freely administered to dispel the agonising pain, and feeding *per rectum* when deglutition can no longer be performed.

10. Pharyngocoele is the name given to a peculiar bag-like diverticulum frequently described as an oesophageal pouch (pressure-pouch of the

cæphagus). Almost always, however, these pouches have their origin at the lowest part of the pharynx and should be described in connection with this part. They are rare, but not so rare as is generally held to be the case. Their origin is still very obscure. In a typical example a pouch is found starting from the pharynx just above the commencement of the cæphagus and passing downwards behind the gullet and in front of the bodies of the vertebrae. In general the pouch bulges towards the left side, though the orifice by which it opens into the pharynx is accurately mesial and is commonly slit-like with its long diameter transverse. These pouches are by many held to be due to the pressure exercised by the food in deglutition forcing outwards a hernial protrusion of mucous membrane and submucous tissue. This pressure of the bolus of food undoubtedly tends to enlarge the pouch when once it is formed, but the constant position of the orifice points strongly to the origin being traceable to some congenital malformation; commonly there is no muscular tissue in the wall of the pouch. Pharyngocæles rarely cause symptoms until middle life, when the sac reaches a size sufficiently large to give rise to difficulty in deglutition. The patient may at the beginning of a meal swallow well; soon deglutition becomes difficult and some of the food is returned, usually but slightly changed, but sometimes decomposed. The more the pouch is filled with food the greater becomes the difficulty in deglutition. In certain cases, especially where the pouch is large (i.e. 4 inches long and capable of holding from 2 ounces to a quarter of a pint of fluid), a softish tumour is appreciable in the neck and can be emptied by external pressure. In time the symptoms become so marked that death from starvation threatens. In order to appreciate the information obtainable from the passage of a bougie, it must be remembered that at one time the bougie may pass without difficulty into the stomach (say 16 inches or more) and at the next essay may be caught in the pouch and its further progress arrested. In the slighter cases, in which the pouch is small, pressure on the neck has been recommended during deglutition to prevent food entering the sac. Palliative treatment, however, is of no avail. Extirpation of the sac has been in several cases safely performed with the most gratifying result. It can be carried out by an incision on the left side of the neck along the anterior margin of the sterno-mastoid muscle. A sound introduced into the pouch through the mouth greatly facilitates the procedure.

11. **Stenosis of the Pharynx** is commonly the result of the cicatricial contraction of syphilitic ulcerations, but may owe its origin to burns, scalds, tubercular ulceration or to traumatism. The most frequent form is the result of adhesions, commonly partial, rarely complete, between the soft palate and neighbouring parts, particularly the posterior wall of the pharynx. Stenosis of the laryngeal portion of the pharynx is fortunately much more uncommon. In complete adhesion of the soft palate to the posterior pharyngeal wall nasal respiration is impossible and the mouth becomes dry from mouth-breathing. The senses of smell and taste practically disappear and speech is indistinct. Should the soft palate be dragged downwards and adhere in part to the base of the tongue, the narrowing may become so extreme that only a small rubber tube may be able to pass through the stric-

tured opening. In such instances swallowing becomes a matter of the utmost difficulty and the patient must be fed with fluids or semi-solids only. Examples of stenosis such as the above are easily recognised. In the diagnosis of narrowing of the lower part of the pharynx the laryngoscope will be needed. Stenosis in this situation is most serious, respiration and deglutition being both gravely compromised; and there is great danger of asphyxia due to the impaction of a bolus of food during its prolonged passage through the narrowed tube of the lower pharynx. Complete stenosis in this region is obviously incompatible with life.

TREATMENT.—As a rule examples of partial adhesion of soft palate to the posterior wall of pharynx and to the base of tongue are best left alone. If the former be complete it may be divided, and an india-rubber ball, which can be inserted into the orifice and blown up *in situ*, may be used to prevent re-adhesion. In extreme cases of the latter form the stricture may be ‘nicked’ by cutting upwards into the soft palate, and bougies frequently passed or a tube worn for some time. The tendency to re-contraction is very great. Stenosis of the lower part of the pharynx, when marked, demands operative treatment owing to the risk of asphyxia. A preliminary tracheotomy having been performed, the stricture is incised in a backward and upward direction with the aid of the laryngoscope, and dilatation by bougies must be carried out regularly. In all the above cases treatment is most disheartening owing to the great tendency to re-contraction that is present. It is, however, remarkable how little stenosis due to partial palatal adhesion seems to distress many patients.

12. **Foreign Bodies** in the pharynx are as a rule substances which the patient has attempted to swallow. Their removal is in general easily effected by means of the finger, within reach of which they commonly lie, or by curved forceps. They are frequently fish-bones, or splinters of bones of mammals or birds; not infrequently, however, in gluttonous individuals, a large bolus of food—commonly meat—becomes impacted in the lower pharynx, and may rapidly cause death from asphyxia. Such may demand the instant performance of a laryngotomy to save life, after which the bolus of food may be extracted by the mouth or pushed down into the stomach by means of a bougie. Foreign bodies in the pharynx, e.g. coins, nuts, fruit-stones, &c., which children have previously had in their mouths, may commonly be got rid of by holding the little patients up by the legs and stimulating coughing by tickling the throat with a feather, or by shaking the child so held head downwards.

13. **Neuroses of the Pharynx** may be divided into (a) motor; and (b) sensory.

(a) **Motor neuroses** may be paralytic or spasmodic. *Paralysis* of the soft palate is commonly diphtheritic in origin. Paralysis of the pharyngeal constrictors is more commonly the result of hysteria, or of central origin as in bulbar paralysis, though it may occur as a sequel of diphtheria. Frequently the paralysis is unilateral, though it may affect both sides. Paralysis of the pharyngeal constrictors causes difficulty in deglutition when unilateral, and when bilateral the act of swallowing cannot be performed and the patient must be fed with a tube. The treatment of the paralysis depends upon its cause. In post-diphtheritic paralysis in which the

prognosis is very good, strychnine administered internally is of undoubted benefit, and some good may result from local application of the faradic current.

Spasm of the pharynx is seen as a very formidable symptom in hydrophobia and sometimes in tetanus (see TETANUS), but apart from these terrible maladies it is almost always of functional origin. It is common in hysteria, in which condition difficulty in deglutition due to this cause is not infrequently complained of, and may also be observed in neurotic individuals, particularly in association with tonsillitis and chronic pharyngitis. The treatment must be directed to the cause.

(b) *Sensory neuroses*.—Anæsthesia of the pharynx may be due to hysteria, diphtheria, bulbar paralysis, or general paralysis. It may be complete or partial, unilateral or bilateral, and is commonly found in association with paralysis of the larynx and soft palate. Should the larynx be sufficiently anæsthetic to permit of food entering it the symptoms are urgent, otherwise they are in general but little noticed. Hyperæsthesia and abnormal sensations of heat, foreign body, pricking, smarting, spasm, or obstruction in the pharynx are chiefly noticed in hysterical patients, though they may be met with in gouty patients, in dyspeptic alcoholics and hypochondriacs. The most common form of paresthesia is that in which a neurotic female complains of a lump in the throat (*globus hystericus*).

Neuralgia is uncommon and may be very severe. It is most frequently seen in neurotic women in whom there is some slight abnormality, e.g. chronic pharyngitis.

14. **Pharyngomycosis Leptothricia**.—The *Leptothrix buccalis* is a fungus which may be considered a normal inhabitant of the human mouth, and it is difficult to understand why in certain cases it takes on an abnormal activity of growth and forms milk-white patches on the tonsils, fauces, tongue, posterior third of the soft palate, or posterior wall of pharynx. The scrapings from these patches show, on microscopical examination, the long threads of the fungus with stratified epithelial cells and amorphous granular detritus. The patches of growth are commonly circumscribed, somewhat firmly adherent to the mucous membrane, and raised above its surface. There is no surrounding inflammation. The symptoms caused by this condition are in general trivial; heat, irritation and dryness in the throat are complained of, and sometimes a degree of dysphagia or cough. There is no fever, increase of pulse-rate, pain, or other constitutional symptom, and the absence of these differentiates the condition from diphtheria. When the tonsils alone are affected, the condition might be mistaken for chronic lacunar tonsillitis; a closer examination will, however, show that the adherent milky-white patches are very different from the yellow cheesy foul-smelling pellets that lie in and are easily turned out of the tonsillar crypts. Carious teeth, chronic tonsillar enlargement, dyspepsia and impairment of the general health, all seem to predispose to the immoderate growth of the fungus. The treatment commonly advised consists in removing the patches, preferably by burning with the galvano-cautery, but recurrence is the rule, and this method is very unsatisfactory. It is an undoubted fact that most cases left alone get well without topical treatment. It is therefore wiser to attend to the general health, enjoining sea-air and

tonics, and to pay particular attention to the digestive functions and to the hygiene of the mouth. A visit to the dental surgeon, with extraction of stumps, filling of carious teeth and removal of the tartar, followed by amputation of the tonsils or destruction of their crypts, will in many cases prove effectual in curing a condition which has defied many applications of the galvano-cautery.

HERBERT F. WATERHOUSE.

PHIMOSIS (φίμωσις, I confine).—SYNON.: Fr. and Ger. *Phimosis*.—A morbid condition of the penis, in which the prepuce cannot be drawn back over the corona of the glans. See PENIS, Diseases of.

PHLEBITIS (φλέψ, a vein).—Inflammation of a vein. See PHLEGMASIA DOLENS; and VEINS, Diseases of.

PHLEBOLITH (φλέψ, a vein; and λίθος, a stone). A concretion formed in a vein. See CONCRETIONS; and VEINS, Diseases of.

PHLEBOTOMY (φλέψ, a vein; and τέμνω, I cut).—A synonym for venesection. See BLOOD, Abstraction of.

PHLEGM (φλέγω, I burn; I am inflamed).—A popular name for sputum or expectoration. See EXPECTORATION; and SPUTUM, Examination of.

PHLEGMASIA DOLENS (*Phlegmasia*, inflammation; and *dolens*, painful).—SYNON.: *Phlegmasia Alba Dolens*; Pop. White Leg; Fr. *Phlegmasia Alba Dolens*; Ger. *Phlegmasia Dolens*.

This is a disease having very distinct characters and easily identified. It has, therefore, been long familiarly known both to the profession and the public. Except in lying-in women it is uncommon, few medical men seeing well-marked or characteristic cases of it under any other circumstances; and it is for the most part as a disease of the puerperal state that it has been the subject of study and investigation.

ÆTIOLOGY.—Phlegmasia dolens affects both sexes, and no age is exempt from it. It has been met with in the new-born infant as the result of a septic condition. It may attack any part of the body, but one or other of the lower limbs is the ordinary seat of the disease. Occasionally it seizes one lower limb first and then the other, or may extend from the one to the other. The well-characterised disease, as it affects lying-in women, is an affection of one or other of the lower limbs, very rarely of both. The left leg is far more frequently affected in the puerperal state than the right; and the left leg is supposed to be more frequently affected than the other under whatever circumstances the disease occurs. In lying-in women the comparative frequency of this affection, and of several other morbid conditions on the left side, is believed to depend on the circumstance that the parts on that side of the pelvis are more frequently subjected to pressure and bruising than the parts on the other side. Tears of the cervix, for example, are somewhat more frequent on the left than the right side. This probably arises from the comparative frequency of the right lateral obliquity of the uterus throwing the direction of the uterine power of labour across the mesial line to the left side of the pelvis. The disease affects multipare more than

primiparæ. It is prone to recur in successive confinements.

From the variety of circumstances under which phlegmasia dolens may occur it will be easily apprehended that it may arise in any period of pregnancy or of the puerperal state, but the usual time of its appearance in lying-in women is the second week after delivery. It rarely commences in the first week, less rarely in the third; seldom subsequently in the puerperal state.

The special proneness of lying-in women to this disease probably depends on the changes in the blood, viz. the increase in the amount of water and in the number of the white corpuscles, and the diminution in the amount of the albumen and in the number of red corpuscles, which prevails during pregnancy, and on the natural formation of thromboses in the uterine sinuses at its termination.

Besides the puerperal state, other conditions render a patient liable to phlegmasia dolens. Among these are convalescence from fever—especially typhoid—dysentery, disease of the rectum, malignant disease of the uterus, uterine fibroids, arrest of menses, and malignant and tubercular disease generally. Occurring in connection with any of these conditions the disease may vary greatly in severity, from being scarcely recognisable to its utmost degree of intensity. But its liability to severity is not the same in all circumstances. For example, in connection with malignant diseases of the womb it is often very slight and chronic. The complaint has been frequently observed to affect the leg of the side corresponding with a previously commenced pleurisy. Wallich and Pinard regard this pleurisy as probably due to small pulmonary infarctions.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances referable to phlegmasia dolens vary, especially in the presence or absence of thrombosis of the veins. Phlebitis and thrombosis are, however, generally found, with more or less associated inflammation of connective tissue and of the lymphatics. The intravenous blood-clots vary in extent, sometimes occurring as high as the vena cava inferior. They vary in appearance, being more or less decolourised, more or less softened, or even in parts diffident. They may be adherent to the veins, partially organised or separable from them. They may entirely block the veins, or may allow the passage of blood between them and the wall of the vein or through their substance as by a tunnel. In recent cases the clot adheres to the internal coat of the vein, which is blood-stained. The coats are thickened and inflamed, and the surrounding cellular tissue is also sometimes specially hardened. In cases complicated with pyæmia there may be found suppuration of the clots, and other appearances observed in that condition.

PATHOLOGY.—Various theories, which reflect the pathology of the times at which they appeared, have been held concerning the nature of phlegmasia dolens. The disease was ascribed to a metastasis of lochia by many pathologists, and by others to a metastasis of milk. The discovery of the lymphatics in the last century led to the first attempts of a truly scientific kind to solve the mystery of the nature of this affection, the suggestion being that it arose from their injury and obstruction. The next attempt to account for this disease was based on the important discovery of the thrombosis of the

veins of the affected limb. This was erroneously assumed to be an invariable or essential condition of the disease, which was accordingly now regarded as phlebotic. But the occurrence of the lesions regarded as essential, the phlebitis and thrombosis, without the development of the characteristic appearances of the affected limb; and, on the other hand, the occurrence of the characteristic appearances without the simultaneous presence of the phlebitis and thrombosis, demonstrated the insufficiency of the phlebotic theory. The next theory to be mentioned is a sort of retrogression to humoral pathology. It alleged that a morbid condition of the blood, of undefined nature, is, along with phlebitis and thrombosis, necessary for the production of the disease.

Another theory is that the disease, as it is seen in lying-in women, is essentially a parametritis, probably of septic origin—that is, an affection of the cellular tissue, commencing, indeed, in the close neighbourhood of the womb, but extending to remote parts, and, it may be, prevailing in them, while the original inflammatory affection of the womb and its immediate neighbourhood has diminished, or even disappeared. Parametric inflammation extends in a similar manner occasionally as far as the cellular tissue around the kidney. When it extends to a limb, it is believed to be the cause of the phlegmasia dolens, and to have the phlebitis and lymphangitis with their secondary thromboses as its consequences.

The divergence of the views as to the causation of phlegmasia is probably in part due to the fact that there is more than one variety of the disease. We may distinguish a thrombotic form in which the clotting of the blood in the vein is either a primary condition or due to some mild degree of septic infection, and a cellulitic or parametric form in which the cause of the thrombus is to be found in the inflammatory changes occurring outside the vessel wall. The occurrence of apyrexial cases of endometritis, due to the presence of the streptococcus *in utero*, shows that the absence of rise of temperature does not exclude some degree of septic infection; and the fact that in the great majority of cases of white leg there is some degree of fever tends to show that a mild form of septic infection is the commonest exciting cause. Further evidence in favour of this view is to be found in the fact that Widal and Chantemesse have demonstrated the presence of streptococci in the clot in the affected veins in the uterus and broad ligaments in three cases complicated by phlegmasia alba dolens. It may well be urged that every case of true white leg in which both the veins and the lymphatics are affected, as distinguished from what may be termed a swelled leg where the veins alone are involved, is the result of some degree of septic infection at the time of delivery. That this is so in the parametric variety, associated as it often is with extensive involvement and sloughing of the subcutaneous tissue, there can be no doubt.

In the primary thrombotic form of phlegmasia the occurrence of the thrombus is usually attributed to the increased tendency of the blood in the puerperium to coagulate, acting in conjunction with dilatations of the veins and a feeble circulation. Further bacteriological investigations are required to prove that this form of white leg can occur independently of septic infection.

The great barrier to progress in our knowledge

of the nature of phlegmasia dolens is the rarity of *post-mortem* investigations, and the sometimes doubtful character of the evidence they afford. Very few unexceptionable *post-mortem* investigations have ever been made in this disease. Such an inspection must be made in an early stage, and in a patient dying accidentally from some cause unconnected with the disease of the limb. Now, the disease is not only not fatal in an early stage, but it might be asserted that by itself it is not fatal at all—that death, apparently from it, occurs only in complicated cases—in such as run an extraordinary and rare course.

SYMPTOMS.—As a rule, phlegmasia dolens is preceded by a slight access of feverish phenomena, seldom by a distinct rigor. There is also sometimes an indefinite malaise for a day or two, before the pain in the limb is complained of. Another premonitory symptom is described, but it is certainly not always present—namely, pain and tenderness in the region of the womb, especially affecting that side of it corresponding to the limb about to be swollen.

The first definite announcement of the disease is generally acute pain along the course of the femoral vein, or in the calf, or above the ankle. In these situations the thrombosed vein can frequently be felt, but not invariably, for sometimes the tenderness, sometimes the swelling, prevents its being made out; and sometimes this thrombosis is absent, at least in parts where it can be felt through the skin. Soon the pain and tenderness extend over the whole affected parts, which may be the whole limb, and often a feeling as of aching in the bones is complained of. The pain is sometimes along the internal saphenous vein, which may be traced by the finger till it dips to join the femoral.

Simultaneously with the complaint of pain, or within a day or two after it, swelling appears, which gradually spreads and increases in hardness. This swelling is not like ordinary oedematous or anasarctous swelling in the sensation it communicates to the hand of the physician, or in the history of its commencement and progress. When it commences, and again as it disappears, it may be, comparatively to its perfect state, soft, and it may pit on pressure; but when, a few days after its appearance, it is fully developed, it is very tense, and nearly as hard as a solid india-rubber ball, and does not pit on pressure. The swelling may appear at once all over the limb, but frequently it commences above and spreads downwards. Sometimes the inverse course is followed. It not rarely affects only the lower parts of a limb, very rarely the upper parts only. It does not affect the posterior more than the anterior surface of the limb. It rounds off the figure of the limb, but does not distend the skin or destroy the form so entirely as anasarca. If the skin is pricked the exudation is a coagulable lymph. Vesicles or blisters are at times present on the skin. Occasionally there is an erythematous blush over parts of the limb; but this is not common, and it may be confined to a narrow strip along the course of a subcutaneous vein or lymphatic.

In a characteristic and fully developed case, such as is frequently observed in the puerperal state, the limb presents a remarkable appearance. The swelling affects the labium and hip and the whole limb, only rarely rising higher. The form of the limb is partly retained, but its features are all

rounded and nearly lost in the swelling. Its colour is white, and hence the name occasionally used of 'white leg,' and formerly of 'milk leg,' when it was supposed to be due to a metastasis of milk. But besides being pale, it is glossy; or its surface resembles that of polished marble, and the disease is sometimes called 'marble leg.' In the milder cases the swelling is less, and softer, and may be confined to a part of the limb.

The limb may be kept in an extended attitude, or it may be slightly flexed at the joints. Movement of it causes much suffering, and the power of voluntary motion is almost completely lost while the disease continues.

After the disease has lasted nine days or thereabouts, it generally makes no further progress, but recedes, the pain and swelling diminishing. The rate of this recession varies very much, being probably more or less directly in proportion to the restored permeability of the vessels. In about 68 per cent. of the cases complete resolution occurs. In a favourable case several weeks may elapse before the disease disappears, while in other cases the cure may be further or even indefinitely delayed. The temperature throughout rarely exceeds 102° , and is often less.

COMPLICATIONS.—The disease is sometimes complicated by other affections, or by aggravations of some of its conditions. Among such occurrences are inflammation and suppuration of the intrinsic joints of the pelvis, erysipelas, limited abscesses of periphlebitic origin, diffuse suppuration of cellular tissue, gangrene of any part or of a varying amount of the entire lower portions of the affected limb. These complications or aggravations cause much danger to life, and in this respect their influence varies according to circumstances. But there are other complications or aggravations which are more often fatal. They may be summed up in the terms 'embolism' and 'pyæmia,' and are the consequences, on the one hand, of detachment of a thrombus in the femoral, or in still larger veins, or, on the other hand, of a more slow breaking up of blood-clots into *débris*, more or less puriform, which enters the circulatory current, and induces a general toxæmia, simple or infective, according to the conditions under which the clots soften.

SEQUELÆ.—The most frequent sequela of phlegmasia dolens is persistent aching of the limb. This is liable to be increased by cold and damp weather, and by derangement of the general health, as well as by exercise. Another is a tendency to oedema of the ankles, more or less persistent. Sometimes the limb remains deficient in muscular power. The atrophy of the muscles may lead to the development of club foot. Rarely, the limb is not only powerless but wasted. And in some very uncommon cases it is the subject of a great hypertrophy of the cellular tissue, simultaneous with muscular wasting; and this cellular hypertrophy may be complicated with more or less extensive and intractable ulceration. Such cases probably result from permanent destruction of large vascular areas.

DIAGNOSIS.—The diagnosis of phlegmasia dolens requires no discussion. The disease can scarcely be confounded with any other if its history is taken into consideration: only it is necessary to remember that oedema with phlebitis or accompanying varicose veins may somewhat resemble it.

TREATMENT.—The treatment of phlegmasia dolens should be both constitutional and local. As

the condition often runs a somewhat asthenic course, in the early stage ammonia in effervescence, with quinine, according to the amount of pyrexia present and the general condition of the patient, and in the later stage iron, are generally useful, with as much sedative as may be indicated by the severity of the pain. Local treatment is very important. The limb is to be kept at rest, either in an extended or flexed position, as may prove most comfortable, and supported on a pillow raised at the foot, with the pressure of the bedclothes kept off by a cradle. Sometimes hot belladonna-fomentations are most comfortable to the patient's feelings, but more frequently wrapping the limb in cotton wool sprinkled with equal parts of belladonna and chloroform liniments, with oil-silk outside, gives the greater relief. When the swelling is subsiding gentle bandaging with a light flannel bandage is very serviceable. If the phlegmasia be associated with septicæmia, its general treatment will of course vary with the general treatment of the toxæmia.

After the acute stage of the disease is past, the sequelæ have to be dealt with. Of these the most frequent are aching, swelling, œdema, and muscular weakness of the limb; and for these the most efficient, but by no means invariably successful, remedies are frictions, bandaging, and faradisation. After all active disease has disappeared, and after danger of dislodging thrombi has passed, the patient may resume the use of the leg. No exact statement can be made of the time at which the danger of embolism is passed. It may prove suddenly fatal as late as thirty-seven days after delivery.

The very rare form of the disease accompanied by extensive cellulitic inflammation of the subcutaneous tissues of the thighs requires energetic stimulant treatment, and free incisions may have to be made.

Persistent local hardness and tenderness, probably periphlebitic, may be treated locally by gentle frictions with a mixture of mercurial and belladonna ointments, but in using frictions of all kinds the danger of dislodging a thrombus is not to be overlooked. Internally, small doses of potassium iodide with quinine or iron are useful adjuvants.

J. MATTHEWS DUNCAN.
GEORGE F. BLACKER.

PHLEGMATIC TEMPERAMENT.—See TEMPERAMENT.

PHLEGMON (φλέγω, I burn—as a medical term, glow, am inflamed).—SYNON.: Fr. *Phlegmon*; Ger. *Entzündungsgeschwulst*.—This term may be defined as a circumscribed area of connective tissue in process of acute inflammation. The word is now almost obsolete.

PHLEGMONOUS.—A term applied to extremely acute inflammation of the cellular tissue, spreading widely, and accompanied by great exudation, with brawny hardness, intense redness, heat, and pain. See CELLULITIS.

PHLYCTENULE (φλύκταινα, a blister).—A small vesicle, not exceeding in bulk the diameter of a pea, as in sudamina, miliaria, and herpes. The term is sometimes also used in connection with ophthalmia. See p. 502.

PHOSPHATIC CALCULUS.—DESCRIPTION.

Phosphate-of-lime calculi are sometimes formed in the kidney, but much more frequently phosphatic stones are a secondary deposit on some pre-existing nucleus. They form dense or porous white layers, frequently showing the glistening crystals of the triple phosphate on the surface. Such masses are soluble in acids, insoluble in alkalis or water, friable, and fusible. They develop rapidly, and may reach an enormous size.

TREATMENT.—When of moderate dimensions, phosphatic calculi may be easily removed by lithotripsy; but, as it is often difficult to ensure the removal of every minute particle, and as they are often accompanied by chronic cystitis and deficient expulsive power of the bladder, recurrence is not infrequent, and the ultimate result unfavourable.

WILLIAM CADGE.

PHOSPHATURIA.—A healthy man excretes some 2·3·5 grammes of phosphoric acid in his urine in the course of 24 hours. This is in part derived from food, and is in part a product of metabolism, within the body, of phosphorus-containing substances, such as nuclein and lecithin. In the urine it is in part combined with alkali-metals and in part with calcium and magnesium.

The endogenous phosphate-excretion is subject to marked fluctuations, as also is the relative excretion of phosphates and of nitrogen, but our knowledge of the conditions upon which these depend is not yet sufficient to supply information of conspicuous clinical value. Strictly speaking, the term 'phosphaturia' should only be used to denote an increased excretion of phosphates beyond the normal limits.

However, the name is often incorrectly applied to the passage of urine which is rendered turbid by the precipitation of the earthy phosphates which it contains, the presence of which is thus rendered evident. Such precipitation is merely dependent upon the reaction of the liquid, and affords no evidence of an unduly large output of phosphoric acid. Urine containing such sediments may be alkaline, amphoteric, neutral, or even faintly acid in reaction. The sediment is colourless, amorphous or showing crystalline points, and is readily dissolved by acids. An iridescent film is usually formed upon the surface of the urine.

Amorphous phosphatic sediments consist of the normal phosphates of calcium and magnesium. Calcium Hydrogen Phosphate (CaHPO_4) may also be present in the amorphous deposit, and in amphoteric or faintly acid urines is sometimes deposited in rosettes of wedge-shaped crystals, constituting the so-called stellar-phosphate sediment. Owing to its insolubility in alkaline liquids ammonium-magnesium phosphate (MgNH_4PO_4), usually known as 'triple phosphate,' is apt to be deposited in crystalline form. The crystals are often large enough to be visible to the naked eye, and usually have the form of triangular prisms, with oblique ends—the well-known 'coffin-lid' crystals—but stellate and feathery forms are also met with. The deposition of these crystals is naturally favoured by alkaline fermentation, and the resulting formation of ammonium carbonate, but the requisite ammonia may be derived from the ammonium-salts present in undecomposed urine.

Very rarely crystalline deposits of normal Magnesium Phosphate ($\text{Mg}_3(\text{PO}_4)_2 + 22\text{H}_2\text{O}$) have been observed, consisting of highly refractive rhombic

tables, and in a case of dilated stomach, in which magnesia had been freely administered, Bradshaw recently observed a deposit of long acicular crystals, which he believes to consist of Magnesium-Hydrogen Phosphate ($MgHPO_4$).

CAUSATION.—Deposit of phosphates takes place in many diseases—diseases often of an opposite character, and having no pathological resemblance—for example, in acute cerebral lesions; towards the close of cases of pleurisy, pneumonia, and rheumatic fever; in certain periods of typhoid fever; and in acute mania. But it may be taken as proved that there is no morbid condition, characterised by definite and constantly occurring symptoms, and accompanied by the deposit of phosphates in the urine, which can be entitled to the designation of a ‘diathesis.’ Prout’s description of the phosphatic diathesis is merely that of ammoniacal urine. Golding Bird associated the deposit with symptoms of irritative dyspepsia, hypochondriasis, and temporary exhaustion of the nervous power; symptoms which are not unlike those said to be characteristic of the so-called oxalic-acid diathesis (see p. 1136). Remembering, however, that phosphatic deposit does not necessarily or frequently mean excess, but depends on diminished acidity or on alkalinity of the urine, it will be more profitable to notice this condition.

The urine becomes neutral or alkaline from the presence of either fixed alkali, potash or soda, or of the volatile alkali, ammonia. The continued or frequent presence of alkaline urine from *fixed alkali*, unless due to a vegetarian diet or the free administration of alkalis, denotes disorders, generally characterised by debility, anæmia, and nervous dyspepsia; it may and does occur in the course of many, even acute, diseases; it represents an altered condition of blood and nutrition; but it is not typical of any one malady or diathesis, nor, so far as analytical investigations have yet gone, is there any clear evidence of the truth of the theory that excess or deposit of phosphates and alkaline urine are the result of increased cerebral action or of brain-disease.

Urine rendered alkaline by *carbonate of ammonium* is always accompanied by deposit of both forms of the phosphates. The alkalinity is the result of decomposition of the urea; there is the peculiar ammoniacal odour, reaching sometimes to intense putrid fœtor. This decomposition is due to the presence and action of certain bacteria which cause the urea to split up into carbonate of ammonium and water. These bacteria are probably introduced through the catheter, but the change may occur in those for whom a catheter has never been used, and in them it is supposed that the bacteria may find their way along the mucous membrane of the urethra, or through the medium of the circulation. Ammoniacal urine is always indicative of lowered vitality, either from age or disease, or spinal injury; it points to no altered condition of blood or constitution, but is the result of local disease. The phosphates which are so freely thrown down are the triple phosphate and the amorphous phosphate of calcium; they are readily deposited on any pre-existing nucleus, whether it be a stone, a clot of blood, a roughened ulcerated portion of bladder, or any foreign body; but without this pre-existing nucleus it but rarely, if ever, happens that such concretions form.

TREATMENT.—As there is no real phosphatic diathesis requiring special management, it follows

that the treatment should consist in removing the cause of the alkalinity of the urine from *fixed alkali*. The two most common causes are dyspepsia and nervous and general debility. In those cases of chronic vomiting and irritable dyspepsia in which the urine loses and recovers its acidity several times daily, no special remedies addressed to the state of the urine can be of any avail. The mineral acids have long been relied on for restoring the natural acidity of alkaline urine; it seems certain, however, that they have no very marked influence in this respect, but simply act beneficially by their indirect tonic effect on the system. Phosphoric and benzoic acids may slightly add to the acidity of the urine, and opium and belladonna in certain conditions of nervous irritability are known to have the same effect; but, speaking generally, the mineral, vegetable, and acid tonics are required in almost all cases, and with them the usual adjuncts, namely, good air and exercise; the cold sea-water bath; a well-selected generous diet, largely composed of animal food; and relief from anxiety or over-work.

For alkalinity and phosphatic deposit depending on ammoniacal decomposition, it must be remembered that in this state the urine is almost always secreted acid. The local disease which causes it must, if possible, be remedied: a stone should be removed; an atonic bladder emptied at stated intervals by the catheter, and washed out with antiseptics, if necessary. In certain cases, as in the cystitis of enteric fever, the administration of urotropine has very beneficial effects. The strength is always to be upheld by rest, good diet, and tonics.

WILLIAM CADGE.
A. E. GARROD.

PHOSPHORUS-MAKERS.—See OCCUPATION-DISEASES.

PHOSPHORUS, Poisoning by.—SYNON.: Fr. *Intoxication Phosphorée*; Ger. *Phosphorvergiftung*.

Phosphorus acts as a poison only when in the form of yellow, common, or soluble phosphorus; in the allotropic form of red or insoluble phosphorus it is generally thought to be inert, but this is doubtful. Poisoning by phosphorus may be (1) *acute* or (2) *chronic*.

On the Continent phosphorus, in the form of the tips of lucifer matches, is frequently used for suicidal purposes. Acute poisoning by phosphorus is also not uncommon in this country. In England phosphorus is most commonly taken in the form of ‘rat paste’ or ‘phosphorus paste,’ a vermin-destroyer composed of butter or other fats and phosphorus, coloured with Prussian blue. Chronic phosphorus-poisoning from the inhalation of phosphorus-vapours has long been recognised.

ANATOMICAL CHARACTERS.—These are well-marked, and consist of extreme fatty degeneration of the liver, and also of the gastric mucous membrane, kidneys, and cardiac muscular fibre. The liver is also greatly enlarged and white; and the organ frequently takes fire on the mere application of a spirit-lamp flame. On microscopical examination the organs affected are seen to be infiltrated with granular fatty matter, soluble in ether; the gastric tubuli are also filled with granular fat; and the striated muscular fibre has more or less completely lost its normal appearance, and been converted into a similar granular material.

SYMPTOMS.—1. *Acute Phosphorus-Poisoning.*—When a phosphorus mixture is swallowed a disagreeable taste is perceived, which is occasionally followed by a burning sensation in the throat, gullet, and stomach, and speedy vomiting. But these signs of the local action of the poison may be either absent or altogether inconsiderable. At any rate, as a rule, these and the diarrhoea and colicky pains described by some writers soon pass off, leaving the patient apparently nearly well; though a careful examination may reveal a small, feeble pulse, and when the patient is questioned, the existence of obscure wandering pains may be admitted. In the course of a few days—usually two, three, four, or five—the patient becomes listless, dull, and slightly jaundiced. There is much headache and sleeplessness, together with a general febrile condition, gradually passing into a ‘typhoid’ state; increasing jaundice; scanty, high-coloured, bile-stained urine; and a quick and very feeble pulse. Muttering delirium supervenes; there may be violent vomiting of yellow, biliary mucus; and the patient gradually sinks, and dies after a day or two, or perhaps three or more, of acute disease, and usually within a week of the administration of the poison. Death may occur, however, at any period, from one or two to eight or ten days, after a fatal dose of phosphorus, which may, perhaps, be taken as half a grain for an adult person.

Variations from the above course of symptoms may be noted. In one class of cases the symptoms betoken a predominance of nervous action. Thus there are cramps and pains in the limbs, great prostration and faintness, convulsions, and, finally, coma. In another class, occasionally observed, hæmorrhagic symptoms are prominent, such as bloody vomits and hæmorrhagic diarrhoea. As an early symptom a phosphorescent condition of the vomited matters, and, more rarely, of the urine, may be noted; and in nearly all cases a peculiar garlicky odour of the breath is perceptible. The phosphorescence or luminosity of the rejected matters is of course best seen in the dark. If the phosphorescent condition of the vomit exist, this permits of no mistake in the diagnosis; but if this condition be absent, the garlicky odour of the breath, and an enlarged condition of the liver, greatly aid in the diagnosis.

2. *Chronic Phosphorus-Poisoning.*—Chronic phosphorus-poisoning consists in poisoning by phosphorus-vapours. Workers in common or yellow phosphorus exhibit a singular form of disease from which workers in red or amorphous phosphorus are exempt. This consists in caries of the teeth and necrosis of the lower jaw. See **TEETH**, Diseases of; and p. 1112.

PROGNOSIS.—This is in all cases very unfavourable, and no general rules can be laid down as to the issue.

TREATMENT.—We know but little respecting this matter. Good results appear to be obtained from the administration of an emetic of copper sulphate, followed by a magma of magnesia, and the use of mucilaginous drinks. The best results, however, have followed the administration of oil of turpentine, which some regard as a specific antidote to phosphorus. It may be given in doses of 10 to 20 minims, frequently repeated. Or, dilute solution of hydrogen peroxide or of potassium permanganate may be administered freely.

The chronic form of the disease, which has led

to horrible suffering and deformity, may be prevented by the use of red instead of yellow phosphorus in the making of matches. The use of respirators, and the impregnation of the atmosphere with the vapour of oil of turpentine, are also preventive measures of great service.

THOMAS STEVENSON.

PHOSSY JAW.—See p. 1112.

PHOTOPHOBIA (φῶς, light; and φόβος, fear). Dread or intolerance of light; a symptom, more or less constant, of most forms of inflammation of the eye. In its most pronounced character it occurs in what is called ‘strumous ophthalmia,’ or phlyctenular keratitis. It is, however, present in all forms of inflammation and ulceration of the cornea, in iritis and cyclitis, and more rarely in choroiditis and retinitis. It is also often met with in many diseases of the nervous system, in cerebral irritation, meningitis, encephalitis, &c., and in many pyrexial states. As an ophthalmic symptom, it may occur in eyes perfectly blind, and is probably due to the irritation of the ciliary nerves by light. See **EYE**, AND ITS APPENDAGES, Diseases of.

PHOTOPSÍA (φῶς, light; and ὄψις, vision).—The subjective sensation of flashes of light, or luminous spectra, due to an abnormal state of some part of the special nervous apparatus of the visual sense. It is a modification of the special sensibility, and, like photophobia, may occur in blind eyes. See **VISION**, Disorders of.

PHRENIC NERVE, Diseases of.—**SYNON.** : Fr. *Maladies du Nerf Phrénique*; Ger. *Krankheiten des Nerven Phrenicus*.—The phrenic nerve, arising from the third and fourth cervical roots, is the motor nerve for the diaphragm. Morbid states of the nerve, its roots and centre, are manifested by inaction or over-action of the diaphragm, its paralysis and spasm.

1. **Paralysis.**—**ÆTIOLOGY.**—Paralysis of the phrenic nerve, that is, of the diaphragm, is rarely due to disease of the nerve-trunk. Its common cause is disease at the origin of the phrenic—the anterior grey matter of the spinal cord at the level of the third and fourth cervical nerves. It is often met with as part of acute or chronic spinal muscular atrophy. But the nerve itself has sometimes suffered, with others, in multiple neuritis. In a few cases the paralysis has been apparently due to cold, supposed to have caused a rheumatic neuritis, and rarely to compression in the neck by deep-seated morbid growths.

ANATOMICAL CHARACTERS.—Degeneration of the trunk of the nerve, wasting of the nerve-fibres, and increase of connective tissue have been found in cases of disease of the spinal cord; and in multiple neuritis there are acute degenerative changes in the nerve-fibres. Of the anatomical changes due to other causes nothing is positively known. Degeneration of the muscular fibres of the diaphragm is found as a consequence of disease of the nerve.

SYMPTOMS.—The evidence of paralysis of the phrenic is inaction of the diaphragm. When one nerve only is diseased there is imperfect action on one side, and this may be conspicuous or indistinct. When both nerves are affected, as is commonly the case in central disease, there is an entire absence of the normal protrusion of the abdominal wall during inspiration; there may even be a recession of the upper part of the abdomen, from the movement of

the lower ribs, and a bulging during expiration in the same situation. In ordinary breathing the respiratory action is not quickened by paralysis of the diaphragm, but if any exertion is made the respirations become more frequent, and the extraordinary muscles of respiration are thrown into action. All spasmodic respiratory actions—sneezing, coughing—are performed with less energy. Little inconvenience is experienced unless bronchitis comes on, and then the lessened respiratory power may place the patient in a condition of danger, which is especially great if, as is often the case, the central cause of the palsy has also weakened the intercostal muscles.

The phrenic nerve is accessible to direct stimulation in the root of the neck, and when it is paralysed its irritability is usually lost, and the diaphragm can no longer be made to contract. In rare cases, however, the nerve-trunk retains its excitability.

DIAGNOSIS.—The diagnosis of paralysis of the diaphragm is not always so simple a matter as might be supposed. Its action should be looked for not only in deep breathing, but in ordinary respiration. Many persons if told to 'take a deep breath' do not put the diaphragm into action at all. In forced breathing the chief extra action takes place in the upper part of the chest, to which most of the muscles of extraordinary respiration are attached. It is probable that the centres for normal and extraordinary breathing are functionally not identical, and that the diaphragm is chiefly represented in the former, so that it does not necessarily act in deep breathing. There is a mechanical reason for this. In the extreme action of the intercostal muscles the thorax is widened to such a degree that the diaphragm becomes less curved by the movement outwards and elevation of its points of attachment, so that its contraction does not effect much additional enlargement of the capacity of the thorax. Hence, in many persons, without any paralysis of the diaphragm, if a deep inspiration is taken, the epigastrium does not advance; it may even recede, in consequence of this movement of the lower ribs. This is especially the case in women, in whom breathing is always less diaphragmatic than it is in men. In them, too, conscious attention to the act of breathing is apt to arrest the action of the diaphragm. The tendency of voluntary breathing is to be costal rather than diaphragmatic, no doubt because the centre for extraordinary breathing, which is chiefly voluntary, is brought partially into action. In a woman under the writer's care, paralysis of the diaphragm was suspected, and during two separate and prolonged examinations not the slightest action could be observed. On a third examination, however, more normal conditions were obtained, and the action of the diaphragm was natural. This is the condition which has been termed 'hysterical paralysis of the diaphragm.'

It must not be forgotten that immobility of the diaphragm may result from other causes than paralysis of the phrenic nerve. In diaphragmatic pleurisy and acute peritonitis, its movement is lessened by a reflex inhibitory effect of the pain. In emphysema of the lungs, in which the thorax is greatly widened, the contraction of the diaphragm produces less effect than in health.

On the other hand, when the diaphragm is really paralysed, a doubt may arise as to whether it moves or not. This is due to the circumstance that the movement of the lower ribs may drag forward the

abdominal parietes close to them, and so the protrusion due to descent of the diaphragm may be simulated. This is especially the case when the abdomen is collapsed, so that when the patient is recumbent its level is considerably below that of the ensiform cartilage. This movement may be distinguished from that due to the descent of the diaphragm by a little care; the movement is confined to the part near the thorax, and there is not the general movement of the abdominal viscera and parietes which results from the contraction of the diaphragm.

PROGNOSIS.—The prognosis of paralysis of the diaphragm is favourable in the rare instances which are due to exposure to cold, and in multiple neuritis, if the patient lives. This nerve, however, is seldom paralysed, except in severe cases of polyn neuritis, in which the prognosis is grave; the prospect of recovery is rather less favourable in lead-poisoning. It is also unfavourable when the diaphragm suffers as part of progressive spinal muscular atrophy. When there is acute spinal muscular atrophy (anterior polio-myelitis), the prognosis will depend on the indication afforded by other symptoms of the position of the chief disease, as to whether the region from which the phrenic nerve arises is gravely or slightly damaged. When the paralysis arises from compression, the prognosis depends on the nature and cause of the pressure.

TREATMENT.—The treatment of the paralysis, which is part of spinal amyotrophy, whatever the nature of the morbid process, is that of the central disease. In all cases causal indications must be met. When it is due to cold, sinapisms should be applied over the part of the phrenic nerve which seems, from any attendant pain, to be chiefly affected. If the nerve has not lost its irritability, it may be faradised systematically. The two points to which the rheophores should be applied are (1) in the neck, just above the scaleni, and (2) near the diaphragm. A strong current has to be used.

2. **Spasm.**—Spasm of the diaphragm occurs chiefly in the form of hiccough, and as part of the respiratory spasm in hydrophobia, and does not need further description. See DIAPHRAGM, Diseases of; HICCOUGH; and HYDROPHOBIA.

W. R. GOWERS.

PHTHIRIASIS (*φθελρ*, a louse). — **SYNON.** : Pediculosis; Fr. *Phthiriase*; Ger. *Läuseucht*.

DESCRIPTION.—There are three varieties of phthiriasis, corresponding to the three species of pediculi that infest the human body. See PEDICULUS.

1. **Phthiriasis capitis.**—Phthiriasis affecting the head is met with chiefly in children. The eruption is an artificial pustular eczema, due to the irritation of the insect, the scratching of the sufferer, and the subsequent growth of pyogenic cocci thus introduced. The superficial lymphatic glands at the back of the neck often become enlarged from secondary infection.

2. **Phthiriasis corporis.**—Phthiriasis of the body is confined to the parts covered by the clothes, and is most developed on the back. It is especially met with in the old and feeble. The lesions of the skin consist of small excoriations and scattered papules, the tops of which are seen to be torn and bleeding from the scratching of the sufferer. These bleeding papules give to the eruption its characteristic appearance. In chronic cases the general colour of the skin is darkened from an excessive deposit of pigment.

3. **Phthiriasis pubis.**—This variety of phthiriasis differs little from that of the body, except that it is limited to the regions infested by the crab-louse.

All three varieties of the disease are attended by intolerable itching.

TREATMENT.—In *Phthiriasis Capitis* the parasites may be destroyed by means of an ointment composed of equal parts of mercury oleate (5 per cent.) and ether. The hair must be well and diligently combed to free it from nits. The rubbing in of petroleum is a favourite application for this purpose. In the case of a child the head should be shaved.

Phthiriasis Corporis is easily cured by means of an ointment containing one part of the oil of stavesacre and seven parts of lard; or the white-precipitate ointment may be safely used. The underclothing must be baked in a disinfecting oven. A hot bath, with free use of carbolie soap, facilitates the removal of any ova adherent to the hair.

In *Phthiriasis Pubis* oleate of mercury (5 per cent.) mixed with ether in proportion of 3 to 1, or ammoniated mercury ointment will effect a rapid cure.

ROBERT LIVEING.

PHTHIRIUM INGUINALE (φθείρ, a louse).—One of the synonyms of *Pediculus pubis*, the crab-louse. See *PEDICULUS*; and *PHTHIRIASIS*.

PHTHISIS (φθίσις, I waste).—**SYNON.** : Consumption; Lung-Tuberculosis; Fr. *Phthisie*; Ger. *Lungenschwindsucht*.

DEFINITION.—Phthisis, or consumption, is the term used to designate a disease characterised by progressive wasting of the body; persistent cough, with expectoration of opaque matter and sometimes of blood; loss of colour and strength; shortness of breath; hectic fever, night-sweats, and diarrhoea; these symptoms being associated with certain well-marked pathological changes in the lungs, namely, the formation of consolidations in a granular or diffuse form, associated with the presence and irritating influence of an organism, the *Bacillus tuberculosis*; these undergoing either caseation or disintegration, leaving behind excavations in the lung-tissue, or, becoming indurated and shrinking, causing contraction of the affected organ.

ÆTIOLOGY AND MORBID ANATOMY.—In the article on *TUBERCULOSIS* will be found a full account of the various predisposing conditions of phthisis. See *TUBERCULOSIS* and *PNEUMOCONIOSES*.

PATHOLOGY.—The first effect of the entry of the tubercle-bacillus into the alveoli would be epithelial proliferation, and possibly, from penetration of the capillaries, exudation of fibrin, and leucocytes. If the bacilli enter by the mucous membrane of the bronchi typical tubercles will be formed. If they are very numerous, or the irritation they set up be very considerable, the alveoli may be stuffed with exudation and epithelial proliferations, which would rapidly caseate. If, however, the irritation be less, the process is slower, more new cells are formed, and a powerful attempt of the organ at self-protection is made (see *PHAGOCYTOSIS*). These cells may in turn perish under the attack of the invader, or be converted into fibroid tissue, and thus resist further advance by encapsulation. The interalveolar lymphoid growth may be the result of bacillary irritation of the alveolar wall; but, being devoid of blood-vessels, it soon undergoes caseation through the action of the bacilli. The histological element, the growth of the interlobular tissue, though it may

be due to bacillary irritation, has a distinctly limiting influence on the advance of the organism, and no bacilli have hitherto been found in this tissue. Tubercle-bacilli are found invariably in freshly formed tubercle, but in grey or milky of some date they are often absent, owing to the tubercle having undergone fibrosis. They are present in white and yellow granulations, and in recently formed caseous masses, but not in the other pathological elements, which, though associated with tubercle, are mainly of inflammatory origin.

The process of softening is due partly to overcrowding of the corpuscular products and partly to a chemical process, arising, according to Watson Cheyne, from bacillary action.

When cavities have formed, bacilli swarm on their walls and in their contents, and the more rapidly they are formed the larger the number of bacilli present.

It is obvious that, whatever part the bacillus plays in the causation of phthisis, the condition of the individual attacked, and his constitutional powers of resistance, are quite as important, as on these depend the whole question of vulnerability. It is probable that in most cases of consumption the bacilli reach the lungs through inhalation; but why, out of a number of persons placed under similar conditions, apparently necessitating the inhalation of tubercle-bacilli, in only a few they increase and multiply, is hard to explain, but renders the existence of a predisposing cause necessary. Koch believes the denudation of the bronchial mucous membrane after measles renders the lungs liable to bacillary attack; and that, similarly, denudation of the intestinal mucous membrane by the shedding of its epithelium from any cause offers the chance of infecting this part by the swallowing of sputum laden with bacilli. Moreover, chronic pleurisy or any cause which tends to cripple the movements of the lung, or to prevent the escape of its secretions, will predispose by forming aggregations of epithelial cells, which are the haunt of tubercle-bacilli.

The spread of bacilli in the lung may proceed (1) by continuity, through the alveolar wall, the epithelium having been destroyed; (2) by re-inhalation of bacilli-laden sputum, especially if expectoration be difficult—the secretion, being raised by coughing to a point where two or more bronchi join, is drawn by deep inspiration into a fresh set of alveoli; (3) through the lymphatics, as is often seen in the stellate arrangement of grey tubercle round a cavity or caseous centre; (4) through the arteries and veins, the entry of the bacilli into these having been demonstrated by several observers. This is the channel of infection in acute tuberculosis.

With reference to the order in which the various portions of the lungs are involved in phthisis, the posterior apex (Fowler) is first attacked, and the lesions spread downwards along the anterior aspect of the upper lobes; the posterior upper border of the lower lobe is next infected, and tuberculisation spreads again forwards along its upper edge. The posterior region seems to be attacked altogether earlier than the anterior; excavation is commonest and earliest (Ewart) at the apex, next in the dorso-axillary and mammary regions, and later and less common at the base. Cavities in the dorso-axillary and mammary regions are invariably secondary, and generally the result of re-infection of the lung from secretion from cavities.

CLINICAL FEATURES.—I. Early Lesions.—

Symptoms.—The symptoms of pulmonary phthisis in the first stage may be thus summarised: Cough, becoming more persistent; mucous expectoration; loss of colour and strength; emaciation; night-sweats; sometimes loss of hair; pulse somewhat quickened, though this is not invariable; and a temperature rising above the normal in the afternoon, and sinking below it in the morning. Peter has noted in many cases a rise in temperature on the affected side during this stage; and with regard to the general temperature of the body, though slight pyrexia is often present, tubercle-formation is quite possible without any rise of temperature, or may even be marked by a depression. Pain in the upper parts of the chest is occasionally present (*see PAIN IN VISCERAL DISEASE*); and the number of respirations is generally increased, though this depends on the extent of lung involved and the degree of anæmia present. Some hold that dyspnoea is an early symptom and precedes all others, but the writer has found that as a rule patients do not notice their breath to be short until their lungs are seriously involved. Disturbance of the digestive powers, and considerable irritability of the intestinal mucous membrane, with a red line on the gums, are noticeable in some cases, though chiefly in the acute forms. The tongue becomes white, the bowels torpid, and the urine scanty. The most constant of the above symptoms are the persistent cough, with mucous expectoration, and the progressive emaciation; and in many cases these are the only symptoms discoverable.

Physical signs.—The physical signs, after the first stage, depend to a great extent (1) on the number and aggregation of the miliary tubercles; (2) on the amount of consolidation to which they have given rise; and (3) on the irritation which their formation causes in the lung.

As a rule, tubercle-formation commences at the apex of one lung, and is detected by the presence of certain physical signs in the supra-scapular, supra-clavicular, or sub-clavicular regions, the signs extending downwards at a later date. The signs vary much in particular cases, but consist at the first in an impairment of the ordinary respiratory murmur by a species of crepitation, differing from the pneumonic crepitation chiefly in its more scattered character, in its being audible with both inspiration and expiration, and in its crumpling nature. At first these crepitations may be audible only on deep inspiration immediately after coughing, which should always be practised during auscultation. Many authors, however, maintain that an earlier sign is the 'wavy' breathing or '*respiration saccadée*' of the French. A noteworthy feature is increased loudness of the expiratory murmur. Accompanying these is increased vocal resonance and bronchophony, more distinct conduction of the cardiac sounds, and deficient resonance on percussion in one of the above-mentioned regions. When a certain definite amount of consolidation has taken place some impairment of the mobility of one side of the chest may be noticed: this is to be detected under the clavicle, where, if any adhesion of the pleura exists, there may be some flattening. Another significant sign is the dry friction-sound, audible generally in the supra-scapular and scapular regions, and indicating limited pleurisy. The dulness usually appears first above the scapula, next over the sternal end of the clavicle, and gradually

extends downwards, being limited generally for a considerable period by the lower border of the third rib.

A careful comparison must be made between the two sides of the chest, and often between different portions of the same side, as otherwise the slighter shades of dulness, and the minor differences in the respiration-sounds, which characterise the presence of tubercle in the lung, will escape notice.

When the crepitation and the wheezing—which may be considered as indicative of irritation in the pulmonary tissue, caused by tuberculosis—have subsided, loud bronchophony, prolonged expiration, and certain varieties of tubular sound, show condensation of the lung-tissue around the neighbouring bronchi; and a certain amount of dulness is to be detected.

2. Softening and Excavation.—*Symptoms.*

The symptoms which accompany the softening of tubercular masses and their subsequent excavation are by no means uniform. Many authors associate this stage with marked signs of pyrexia, with copious night-sweats, and increase of cough and emaciation; but this is not always the case, for, according to the writer's experience, the process may go on with even subnormal temperatures, and with gain of weight; but as extension of the disease often accompanies the softening process, some of the above symptoms, which have been assigned to softening, may be due to the fresh infection and pneumonia caused by it. The symptoms which should be most depended upon for the detection of softening are increase of cough and expectoration of a yellow colour, occasionally streaked with blood. If the expectoration be carefully examined, elastic tissue, tubercle-bacilli and other organisms may be found. *See BACTERIA; SPUTUM, Examination of; and TUBERCULOSIS.*

Physical Signs.—The signs which these changes give rise to are often obscure. The percussion-sounds vary: sometimes there is an increase of dulness, possibly due to pneumonia of adjacent lobules; at other times, hyper-resonance, as if air had taken the place of the expectorated masses. In all these cases much depends upon the situation of the lesion. The formation of a cavity deep in the lung, and far from the chest-walls, may take place without being detected (except by the expectation); whereas the formation of a similar cavity on the surface gives rise to unequivocal signs. Auscultation reveals—where formerly bronchophony and fine crepitus existed—crepitation of a very coarse character, commencing with a *click* sound, and after a while developing into a *croak*. When this last note has been reached, loud tubular sounds become audible on coughing, and we soon get the sounds characteristic of a cavity. The great distinguishing features of these moist sounds of softening are their variety, their short duration, and their concentration over one small portion of the lung. In phthisis, crepitation much more commonly signifies tubercle-formation or pneumonia than it does softening of already formed tubercular masses. The formation of a cavity is generally followed by regular morning expectoration, usually opaque, and nummular in form, and in the majority of cases, unless interfered with by treatment, by the usual train of consumptive symptoms, if these have not already appeared. These are—night-sweats, slightly elevated temperature in the afternoon, and rapid loss of flesh, strength, and colour. The drawn look of the face, the hectic spot on the cheek,

with great rapidity, owing probably to the pyrexia and fitfulness of the appetite.

6. Hæmorrhagic Phthisis.—This name is intended to designate, not phthisis arising from the results of hæmoptysis (*phthisis ab hæmoptoe*—Niemeyer), but a form recognised by C. J. B. Williams, Peacock, Hughes Bennett, and the writer, in which large and repeated hæmorrhage is the principal feature, associated with a small amount of detectable disease. It is more common among men than women, in the proportion of five to one; and the period of attack is later than in the ordinary forms, possibly owing to the element of heredity being generally absent. The patient may have had signs of failing health before the hæmoptysis, but often he is apparently in good health, when he is suddenly attacked with profuse hæmoptysis, the blood being florid, the hæmorrhage sometimes lasting many days, and always causing a reduction in flesh and strength. Cough and expectoration follow, yet examination of the chest only indicates slight signs, and sometimes none at all. When present they are to be found in the supra- or inter-scapular regions, or below the clavicle. The patient improves, and often entirely loses his cough before the recurrence of the hæmorrhage, which may not take place for days, weeks, months, or even years. If the attacks recur often, the cough becomes persistent; the expectoration, when not sanguinolent, is mucopurulent; wasting and night-sweats appear; and the physical signs now show unmistakable consolidation, which goes on to softening and excavation. In most cases the disease does not extend beyond consolidation, and large quantities of blood are expectorated without fatal results, the patients recovering in the intervals, and sometimes living to a considerable age. Peacock stated that in most instances some more or less exciting cause of a depressing character is to be detected, in the form of syphilis, cold, dysentery, bodily strain, exertion of voice; but the observer will often fail to find one. The exact pathology of this form of phthisis is uncertain, because few of the patients die in the early stage; but it is probable that the hæmorrhage is produced by the erosion of the pulmonary vessels by bacillary invasion, masses of tubercle-bacilli having been shown to be present in the walls of both arteries and veins; and in hæmorrhagic phthisis it is probable that the larger vessels are thus attacked.

DIAGNOSIS.—Phthisis is distinguished from other chest-affections by the presence of tubercle-bacilli in the sputum and by the evidence of physical signs. The evidences of consolidation separate it from bronchitis; while the tendency of the signs to become localised in the apices of the lungs, their special characters, and the combination of consumptive symptoms, distinguish it generally though not invariably from pneumonia due to other causes.

Of the various forms of phthisis, the most difficult to diagnose from other diseases is acute milary tuberculosis, which at its onset is sometimes mistaken for acute bronchitis, from the fine *râles* and rhonchi accompanying the milary formation. It has also been confounded with enteric fever, from the high pyrexia, the depression of the patient, and the occasional diarrhoea accompanying it; but in both cases the rapidly advancing symptoms, and the steadily progressing physical signs, such as increased and scattered crepitation, if frequent careful

examinations be made, ought to leave us in no doubt as to the nature of the case.

The diagnosis between acute phthisis and croupous pneumonia is not easy at the onset of these complaints, the physical signs not always sufficing for this purpose. In a short time, however, the detection of tubercle-bacilli, and later of lung-tissue in the sputum, and the rapid wasting, make matters quite certain.

The diagnosis of chronic tubercular phthisis from anæmia and chlorosis, sometimes confused with it on account of the amenorrhœa often common to both, is made by the physical signs; by the different kinds of pallor in the two diseases; and, lastly, in chlorosis, by the absence of wasting. The diagnosis of excavation in phthisis from bronchiectasis is by no means easy, as the position of the cavernous sounds is not always sufficient to determine the nature of the lesion. Dilated bronchi are found in the subclavicular and interscapular regions, and where ulceration is proceeding in bronchiectasis lung-tissue may be detected in the sputum; but the presence of tubercle-bacilli at once settles the question in lesions of phthisis, while the convulsive character of the cough, and the fœtid expectoration, abundant, but mixed largely with air, generally enable us to decide in favour of dilated bronchi. The diagnosis from cancer, especially from sarcoma of the lung, depends partly on the symptoms, and partly on the physical signs which, in the case of sarcoma, present larger areas of dulness and generally of a more intense kind, and the remarkable shutting off of all breath- and voice-sound, which cases of lung-cancer present. See MEDIASTINUM, Diseases of.

DURATION AND PROGNOSIS.—Early detection of the disease, and improved treatment, have worked a great revolution in our ideas as to the *duration* of phthisis.

The statistics of C. J. B. Williams and the writer, founded on 1,000 cases among the upper classes, give an average duration in 198 deaths of 7 years 8·72 months; and in 802 living of 8 years 2 months. The fact of these patients having all been one year and upwards under observation necessarily excludes some of the acute cases; but with this limitation these figures, striking though they be, may be taken as a correct average for the duration of the disease among the upper classes under modern treatment, especially as 72 per cent. of the living had recovered sufficiently to pursue their usual avocations, and many among them had already lived upwards of twenty years since their first attack. The duration of the disease is found to be considerably influenced by age; for it is longer in proportion as the age of attack is later, this retarding influence being more conspicuous among males than females. Females are attacked earlier, and the disease in them runs a shorter course by nearly two years than among males.

Of the varieties of phthisis, acute tuberculosis is the most rapid in its course, generally terminating in a few weeks, or occasionally in a few days. Acute phthisis has hardly a less rapid course, though it may occasionally be retarded, the disease becoming chronic, and the patient surviving for many years. Catarrhal phthisis has an average duration somewhat below the average of eight years of ordinary phthisis. Fibroid phthisis, on the other hand, exceeds the ordinary duration by nearly two years. Hæmorrhagic and scrofulous phthisis are both of

long duration. These calculations are based on statistics of patients of the upper classes treated according to the best medical and hygienic treatment known; but if hospital cases are reckoned, the average duration of phthisis generally, and of its various forms, must be held to be much lower than the above estimate.

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without some form of dropsy; (7) marked dyspnoea, especially if not warranted by the condition of the lungs; (8) the existence of considerable excavation in both lungs and the occurrence of pneumothorax.

TREATMENT.—The treatment of phthisis may be considered under four heads: (1) *treatment by inoculation and by anti-tubercular serum*; (2) *medicinal*; (3) *dietetic and hygienic*; and (4) *climatic*.

1. *Inoculation.*—In 1890 Koch introduced the treatment of phthisis by hypodermic injection of *tuberculin*, a glycerine-extract of a pure cultivation of tubercle-bacilli, in which the parasites had been killed; and maintained that early-stage cases could be cured after four to six weeks of this treatment. Subsequent experience from all parts of the world did not confirm this statement, but demonstrated that, while tuberculin had a strong affinity for tuberculous lesions in various organs of the body and gave rise to characteristic constitutional reactions, it caused breaking down of the tubercular masses, and of the lung-tissue in their immediate neighbourhood, fresh eruption of tubercle, and extension of old cavities, the sputum containing an increased number of tubercle-bacilli, and a considerable amount of lung-tissue. Virchow found in the necropsies of patients treated by this method various kinds of pneumonia, including what he termed 'injection catarrhal pneumonia,' and in one case tubercular meningitis, which he attributed to this treatment. By treating it with platinum chloride, Klebs extracted from tuberculin an albumose called tuberculocidin, along with certain alkaloids, and attributed to it the power of arresting tubercle; and these and other modifications of tuberculin have been tried in the treatment of phthisis, without any success. Koch introduced later on two new preparations, viz. *Tuberculin O* and *Tuberculin T R*, prepared by pounding in a mortar dried cultures of the tubercle-bacillus, adding distilled water, and then centrifugalising in a powerful machine. The clear fluid at the top is used for inoculation as *O Tuberculin*, and the residue again treated with water as *T R Tuberculin*. Both preparations are preserved by the addition of 20 per cent. of glycerine before injection. The experience of these preparations has not been altogether favourable. Maragliano, the writer, and others have treated tuberculous patients with anti-tubercular serum, produced by inoculating immune animals, such as horses, goats, and asses, with tuberculin and then withdrawing serum and injecting it into the patients, but the results have never shown any considerable amount of success.

2. *Medicine.*—The medicinal treatment must be directed to four objects: firstly, antipthithical, to raise the standard of nutrition and to fortify the individual against the bacillary invasion; secondly, antiseptic, to promote the destruction and elimination of the *Bacillus tuberculosis*; thirdly, to reduce and allay the local inflammations and congestions which accompany and considerably complicate the tubercular changes; and, fourthly, to relieve the various urgent symptoms. The first object is carried out by tonics, such as iron, quinine, arsenic, the mineral acids, and, above all, cod-liver oil, which has been shown to be the most effective agent of all in improving nutrition and rendering the individual less liable to bacillary attack. Some precautions are, however, necessary to ensure its being tolerated for long periods. The pale oil should be preferred, and ordered in doses of from 5j to 3ss shortly before or after meals. The best

and lips, gastric disturbance and diarrhoea, and occasional delirium, the symptoms closely resembling those of enteric fever, for which the disease is often at first mistaken. Cough and slight expectoration come on; fine crepitation and bronchial rhonchus take the place of the ordinary vesicular sounds; and occasionally some dullness is detected over the posterior regions of the chest. The patient wastes rapidly; the breathing becomes more and more embarrassed; the sputum rusty; the crepitation more general and louder. Later on, the symptoms of collapse appear—the pulse becomes more rapid and feeble, the aspect ghastly or livid, cold perspirations appear; and death occurs within a few weeks from the date of the first onset. Or the symptoms may be more cerebral in character, denoting that the meninges are the seat of miliary tubercle. The patient complains of pain in the head, vomiting, and intolerance of light; begins to mutter and to give wrong answers; and then has marked delirium. The aspect is heavy and confused; hyperæsthesia of skin appears (Empis); and double vision, though squinting is not always noticeable. Granulations can often be detected with the ophthalmoscope in the fundus oculi. Twitchings of the muscles of the extremities and sometimes of the face occur, followed by convulsions, and by paralysis of the sphincters. Dilatation of the pupils and other signs of effusion supervene, and the patient dies comatose. In this variety, as a rule, the temperature remains continuously high (between 100° and 102° F.), but in some instances it may be observed to rise above 100° F. for the last ten days of the patient's life. After death the lungs are found highly congested and pervaded with miliary tubercle, soft in character, but devoid of caseation and containing abundant tubercle-bacilli; the bronchi are full of frothy mucus; and tubercle may be found in the pleura, peritoneum, or cerebral membranes, with effusion into the ventricles. This form is distinguished from capillary bronchitis by the presence of fever; from enteric fever by the different physical signs; from acute phthisis by the great dyspnoea and scanty expectoration; and by the head-symptoms (when present) from all the above.

Acute tuberculosis is the most fatal form of consumption, terminating in a few weeks or even days, and is characterised by gastric disturbance, by the presence of family predisposition (Pollock), and by the absence of hæmoptysis. It may be primary or secondary, but in each case is equally fatal.

2. Acute Phthisis.—The patient, generally young, who may have had a cough previously, is attacked with sharp pain in one side of the chest, quick pulse, high temperature, the skin being quite burning to the hand of the physician, alternating with night-chills and sweats. The general appearance betokens pneumonia, but the crepitation commences at the apices, extending to the whole lungs, and is not so fine and even as in pneumonia. The cough increases; the expectoration becomes opaque and purulent, containing quantities of lung-tissue and swarms of tubercle-bacilli; and the temperature assumes the intermittent type. The physical signs show at first gradual consolidation of both lungs, but later on indicate that excavation has taken place; and this continues, the patient rapidly wasting and dying in a few weeks. Sometimes the cavity opens into the pleura, which in these cases is rarely adherent, and death ensues by pneumothorax. This form is not quite so hopeless as acute

tuberculosis; and in some instances the disease may stop short of utter lung-destruction and become chronic, the patient remaining in a state of crippled respiration and of health for months and even years. Such cases may last for periods extending from three and a half to sixteen years. After death the lungs are found more or less consolidated, with adherent pleurae, the indurations consisting of red hepatisation and caseous infiltration, the latter largely predominating. Excavations abound in all directions, and but little or no miliary tubercle is present. The characteristics of this form are (1) the acuteness of the disorganising processes, excavation quickly succeeding consolidation; (2) the inflammatory nature of the lesions, and the rarity of miliary tubercle; (3) the occurrence of pneumothorax; and (4) the freedom of other organs from tuberculosis. An intermediate form is acute tuberculo-pneumonic phthisis, in which tuberculation predominates, but the tubercular masses soften rapidly, and tubercle forms at the same time in other organs such as the intestines.

3. Catarrhal Phthisis.—The patient has been subject for years, perhaps, to attacks of winter catarrh, which disappear in summer; and at last, owing to a severe season, or from his being in less favourable circumstances than usual, his cough does not cease, as formerly, but remains persistent, and is accompanied by some purulent expectoration, loss of flesh, and night-sweats. The bronchial *râles*, sonorous and liquid, as they disappear from certain parts of the lung, become more prominent and localised in others, especially under the clavicles, and above and between the scapulae. The *râles* become coarser, and the sonorous rhonchus assumes a croaking character. Signs of consolidation soon appear, but are never so prominent as in other forms, owing to the temporary emphysema accompanying bronchitis; the dullness appears in patches over the centres of increased rhonchus; the liquid *râles* diminish, owing to increasing obstruction, and give place to a tubular sound conveyed by the extending consolidation from the larger bronchi, and heard best in situations overlying them, as below the clavicle, and above and within the scapula, in the axillary and middle dorsal regions. The tubular sound has a sharp, whiffing character, and is often unaccompanied by bronchophony, from the consolidation being insufficient, and the bronchial tubes too choked to produce it. If the case goes on unfavourably, the expectoration becomes more abundant, and is found to contain both tubercle-bacilli and shreds of lung-tissue, and excavation is proved to have taken place, with the usual symptoms; the patient assumes all the appearances of advanced cavity-phthisis, and the case from this date can hardly be distinguished clinically from those of a strictly tubercular origin. After death the lungs are found to be more or less consolidated, the indurations taking the direction of certain lobules and generally not affecting entire lobes. The indurations are of a grey or yellowish tint, with numerous yellow masses of caseation intervening, which abound in tubercle-bacilli. Another form of catarrhal phthisis follows on attacks of unresolved pneumonia or of pulmonary affections tending to lung consolidation. Portions of the lung may be found in the first stage of catarrhal consolidation, so well described by Hamilton, with isolated lobules or groups of lobules of a leaden or purple colour, and the adjoining ones may be emphyse-

matous. Wedge-shaped patches of consolidation can be traced on the pleural surface, exuding on section yellow catarrhal fluid similar to that contained in the bronchi. Numerous excavations of irregular form are seen containing bacilli, but in most instances no trace of tubercle is to be found, though it is occasionally present. The bronchi are generally dilated, and full of purulent matter. This form is more common among the young than the old, and arises from whooping-cough, measles, and bronchitis, the pathology being extension of catarrh from the bronchi to the alveoli, implication of the interstitial tissue, large epithelial proliferation, causing pressure and emptying of capillaries, degeneration and caseation of the alveoli and their contents, and consequent excavation, with occasionally lymphatic infection.

4. Fibroid Phthisis.—This term, introduced by the late Andrew Clark, is applied to cases of which fibrosis is the principal feature. While this process accompanies most instances of chronic phthisis, it specially characterises those in which interstitial pneumonia is present, and entirely modifies their history and symptoms. It is generally secondary to attacks of pleurisy and pleuro-pneumonia, or to chronic pneumonia, resulting from long-continued irritation of the lungs, through the inhalation of dust or grit, as prevails among fork- and knife-grinders, colliers, and button-makers. See OCCUPATION-DISEASES.

A patient has an attack of pleurisy with effusion, from which he recovers with absorption of fluid; but percussion shows dulness over the whole side, and somewhat feeble respiration. The patient experiences dragging pains in the side; a dry, hacking cough, sometimes paroxysmal in character, with little expectoration, continues; and the breathing, always short, becomes still more so on exertion. These symptoms increase, and a few months later we find marked immobility of the affected side, dulness throughout, and now considerable shrinking, the circumference of this side measuring one or two inches less than the healthy side. On auscultation we notice the breathing to be very deficient in some parts, and in others bronchial, and sometimes cavernous in character; but generally there is everywhere absence of true vesicular breathing. Careful percussion of the opposite side of the chest shows the line of resonance to extend beyond the usual limit, passing to the edge of the sternum, and often an inch or two farther; demonstrating that the contraction of the affected lung has caused the healthy one to be drawn across. Other organs are likewise displaced. If the left lung be affected, the heart is tilted, not necessarily upwards, as when a cavity is contracting, but outwards. The stomach rises, its note being audible as high as the fourth rib. The heart is not only displaced, but is uncovered by the retreating lung; and the right auricle and ventricle are clearly distinguished by their pulsations. If the right lung is affected, the left may be drawn over, and the area of resonance may extend as far as the inner half of the right clavicle, and a line drawn thence sloping towards the middle of the sternum. The heart is transposed, and its impulse may be traced in the fourth interspace on the right side. The liver rises up to the fifth rib, and shrinking of the chest-walls takes place, as on the other side. The expectoration, if there be any, has rarely been found to contain

tubercle-bacilli. The pulse may be slow; the respiration often rapid, rising to 50 and 60 per minute. The temperature seldom rises above the normal and is sometimes subnormal. When the temperature rises over 100° F. it signifies that something beyond fibrosis is going on. The cough is troublesome and often induces vomiting; and the expectoration becomes more and more difficult, and in time, on account of retention, fetid. Meanwhile the dyspnoea increases, the other lung becoming involved; signs of obstructed circulation appear; dropsy of the extremities takes place, and rapidly increases; the urine becomes albuminous, and the patient dies, either of dyspnoea or of uræmic poisoning, his death contrasting strongly with the ordinary termination of consumptive disease. The patient may, however, die of apnoea, without albuminuria or dropsy. After death we find a lung contracted to the size of a man's fist, with enormously thickened and adherent pleura, and widely dilated bronchi, with interlobular septa much increased in size and encroaching on the lung-structure, which seems to be replaced by a hard fibrous tissue, in parts mottled with grey, deeply pigmented, and resembling cartilage in its resistance to the knife. Embedded in this structure are found caseous and cretaceous masses, or, again, excavations of various sizes; the walls of these and of the dilated bronchi being rigid and inelastic, from the presence of the fibroid material, and thus affording some explanation of the difficult expectoration and consequently troublesome cough. Careful examinations of microscopic sections of this fibroid tissue have, according to Andrew Clark, Watson Cheyne, and Percy Kidd, failed to detect tubercle-bacilli; but in the caseous masses embedded in it Watson Cheyne has found them. Besides these changes, we may find the other lung the seat of tuberculosis, though this is not constant; but commonly the bronchial glands are hardened and deeply pigmented. There is often amyloid disease of the liver, spleen, and kidneys.

5. Scrofulous Phthisis.—This is a variety where disease of the lung is preceded by, or accompanies, tubercular affections of various joints, caries of the sternum, ribs, and vertebrae, lumbar and psoas abscesses, otorrhœa, fistula in ano, or, as is most common, enlarged and caseating glands—cervical, bronchial, axillary, or mesenteric. Cases of secondary phthisis show an early infection of the lymphatic system, and a remarkable correlation appears to be established between the external gland or discharging surface and the condition of the lungs. If the glands are suppurating, or if the fistula is open, or if the carious bone freely discharges, the lung-disease will remain quiescent, and progress may be made towards arrest; but if, on the other hand, any of the above discharges, in the most of which tubercle-bacilli have been detected, should be checked, or cease *without the removal of the diseased tissue*, the lung-disease passes into fresh activity, making considerable advance and extension. This has been used as a strong argument against operating on fistula in ano if the lungs have been affected. The temperature-course in these cases, if active lung-changes are taking place, is remarkably fitful, showing evening exacerbations of 102° to 104° F. and morning depressions of 96° to 97° F.; and night-sweats are usually very profuse. Patients of this type lose and gain flesh

with great rapidity, owing probably to the pyrexia and fitfulness of the appetite.

6. Hæmorrhagic Phthisis.—This name is intended to designate, not phthisis arising from the results of hæmoptysis (*phthisis ab hæmoptoe*—Niemeyer), but a form recognised by C. J. B. Williams, Peacock, Hughes Bennett, and the writer, in which large and repeated hæmorrhage is the principal feature, associated with a small amount of detectable disease. It is more common among men than women, in the proportion of five to one; and the period of attack is later than in the ordinary forms, possibly owing to the element of heredity being generally absent. The patient may have had signs of failing health before the hæmoptysis, but often he is apparently in good health, when he is suddenly attacked with profuse hæmoptysis, the blood being florid, the hæmorrhage sometimes lasting many days, and always causing a reduction in flesh and strength. Cough and expectoration follow, yet examination of the chest only indicates slight signs, and sometimes none at all. When present they are to be found in the supra- or inter-scapular regions, or below the clavicle. The patient improves, and often entirely loses his cough before the recurrence of the hæmorrhage, which may not take place for days, weeks, months, or even years. If the attacks recur often, the cough becomes persistent; the expectoration, when not sanguinolent, is mucopurulent; wasting and night-sweats appear; and the physical signs now show unmistakable consolidation, which goes on to softening and excavation. In most cases the disease does not extend beyond consolidation, and large quantities of blood are expectorated without fatal results, the patients recovering in the intervals, and sometimes living to a considerable age. Peacock stated that in most instances some more or less exciting cause of a depressing character is to be detected, in the form of syphilis, cold, dysentery, bodily strain, exertion of voice; but the observer will often fail to find one. The exact pathology of this form of phthisis is uncertain, because few of the patients die in the early stage; but it is probable that the hæmorrhage is produced by the erosion of the pulmonary vessels by bacillary invasion, masses of tubercle-bacilli having been shown to be present in the walls of both arteries and veins; and in hæmorrhagic phthisis it is probable that the larger vessels are thus attacked.

DIAGNOSIS.—Phthisis is distinguished from other chest-affections by the presence of tubercle-bacilli in the sputum and by the evidence of physical signs. The evidences of consolidation separate it from bronchitis; while the tendency of the signs to become localised in the apices of the lungs, their special characters, and the combination of consumptive symptoms, distinguish it generally though not invariably from pneumonia due to other causes.

Of the various forms of phthisis, the most difficult to diagnose from other diseases is acute miliary tuberculosis, which at its onset is sometimes mistaken for acute bronchitis, from the fine *râles* and rhonchi accompanying the miliary formation. It has also been confounded with enteric fever, from the high pyrexia, the depression of the patient, and the occasional diarrhoea accompanying it; but in both cases the rapidly advancing symptoms, and the steadily progressing physical signs, such as increased and scattered crepitation, if frequent careful

examinations be made, ought to leave us in no doubt as to the nature of the case.

The diagnosis between acute phthisis and croupous pneumonia is not easy at the onset of these complaints, the physical signs not always sufficing for this purpose. In a short time, however, the detection of tubercle-bacilli, and later of lung-tissue in the sputum, and the rapid wasting, make matters quite certain.

The diagnosis of chronic tubercular phthisis from anæmia and chlorosis, sometimes confused with it on account of the amenorrhœa often common to both, is made by the physical signs; by the different kinds of pallor in the two diseases; and, lastly, in chlorosis, by the absence of wasting. The diagnosis of excavation in phthisis from bronchiectasis is by no means easy, as the position of the cavernous sounds is not always sufficient to determine the nature of the lesion. Dilated bronchi are found in the subclavicular and interscapular regions, and where ulceration is proceeding in bronchiectasis lung-tissue may be detected in the sputum; but the presence of tubercle-bacilli at once settles the question in lesions of phthisis, while the convulsive character of the cough, and the fetid expectoration, abundant, but mixed largely with air, generally enable us to decide in favour of dilated bronchi. The diagnosis from cancer, especially from sarcoma of the lung, depends partly on the symptoms, and partly on the physical signs which, in the case of sarcoma, present larger areas of dullness and generally of a more intense kind, and the remarkable shutting off of all breath- and voice-sound, which cases of lung-cancer present. See MEDIASTINUM, Diseases of.

DURATION AND PROGNOSIS.—Early detection of the disease, and improved treatment, have worked a great revolution in our ideas as to the *duration* of phthisis.

The statistics of C. J. B. Williams and the writer, founded on 1,000 cases among the upper classes, give an average duration in 198 deaths of 7 years 8·72 months; and in 802 living of 8 years 2 months. The fact of these patients having all been one year and upwards under observation necessarily excludes some of the acute cases; but with this limitation these figures, striking though they be, may be taken as a correct average for the duration of the disease among the upper classes under modern treatment, especially as 72 per cent. of the living had recovered sufficiently to pursue their usual avocations, and many among them had already lived upwards of twenty years since their first attack. The duration of the disease is found to be considerably influenced by age; for it is longer in proportion as the age of attack is later, this retarding influence being more conspicuous among males than females. Females are attacked earlier, and the disease in them runs a shorter course by nearly two years than among males.

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2. *Medicine.*—The medicinal treatment must be directed to four objects: firstly, antiphthisical, to raise the standard of nutrition and to fortify the individual against the bacillary invasion; secondly, antiseptic, to promote the destruction and elimination of the Bacillus tuberculosis; thirdly, to reduce and allay the local inflammations and congestions which accompany and considerably complicate the tubercular changes; and, fourthly, to relieve the various urgent symptoms. The first object is carried out by tonics, such as iron, quinine, arsenic, the mineral acids, and, above all, cod-liver oil, which has been shown to be the most effective agent of all in improving nutrition and rendering the individual less liable to bacillary attack. Some precautions are, however, necessary to ensure its being tolerated for long periods. The pale oil should be preferred, and ordered in doses of from ʒj to ʒss shortly before or after meals. The best

vehicles for it are the vegetable bitters—such as gentian, calumba, quassia, nux vomica and strychnine, hop, camomile, and cascarrilla—combined with an acid or alkali, according to the state of the gastric mucous membrane, and rendered more palatable by the addition of tincture or infusion of orange-peel or syrup of ginger. Various other vehicles are used, such as milk, salt and water, lemon-juice, orange wine, and sherry; while many patients, especially children, take it best in an emulsion, composed of cod-liver oil, a few drops of liquor potassæ or liquor ammoniæ, with an essential oil, like that of cloves or cinnamon, to cover the taste. In the great majority of cases cod-liver oil is well borne, if exhibited with discretion. Other oils are of use, but few equal the cod-liver oil in efficacy, on account of its great penetrative power, and of its forming with the biliary and pancreatic juices a compound easily absorbed by the lacteals. Malt Extract and similar preparations, though of greatly inferior nutritive power to cod-liver oil, often cause increase of weight, chiefly by assisting the patient to digest more starch, and are often with advantage combined with cod-liver oil, forming a palatable compound; but the stimulating quality of the malt sometimes causes increase of the patient's cough, and does not augment appetite. Of greatly inferior utility to the oil are the preparations of phosphorus and sulphur.

In France the sulphur-springs of Eaux-Bonnes, Cauterets, Bagnères-de-Luchon, and Bagnères-de-Bigorre are largely frequented by consumptives, the ground of this treatment being that the results of Claude Bernard's experiments show that sulphur, when absorbed, is excreted through the respiratory mucous membrane. Peter considers that any benefit that may accrue is owing to the influence of sulphuric acid on the catarrhal conditions. Arsenic appears to exercise a most beneficial effect in those consumptives who can digest it, and often under its influence fever and night-sweats disappear, and weight is gained; and the arsenical waters of Mont Dore, La Bourboule, and Royat are much frequented by consumptives.

The second object of treatment is antiseptic. The admixture of antiseptics with tuberculous sputum and other tuberculous material has rendered the inoculation of guinea-pigs and rabbits with them harmless; such are sulphuretted hydrogen, iodide of mercury, corrosive sublimate, iodine, carbolic (3 per cent.) and boric acid, helenin, creosote, phenyl-acetic and phenyl-propionic acids, and sulpho-carbolate of sodium. Some were actually mixed with tuberculous material, and then injected; others were injected afterwards, separately from the virus. All, when used in sufficient strength, were successful in checking the evolution of the disease in animals, and have been used in consequence in phthisis; but only with very moderate success. Moist heat is, however, the most powerful bactericide; tuberculous sputum can be rendered innocuous by exposure to a boiling temperature. The application of antiseptics has been made—(1) through the air-passages by sprays, inhalations, and 'antiseptic respirators'; (2) by hypodermic injection; (3) by injection *per rectum* of sulphuretted hydrogen and carbonic acid (Bergeon); (4) through the stomach as medicines; and (5) by intra-pulmonary injections through the wall of the thorax into tubercular cavities. It cannot be affirmed that any of these forms of anti-

septic treatment have succeeded in destroying the bacillus or in counteracting the effect of the ptomaines which it is supposed to propagate; and far better results have been obtained by constitutional measures which render the individual less vulnerable to bacillary attack.

The third object of treatment, the reduction of local inflammation, is best accomplished by mild antiphlogistic means, such as salines, with or without antimony; and counter-irritation to the chest-wall by blisters, iodine, or vesicating liniments, mustard, or the milder but still effectual application of linseed-meal poultices. Steady continuance with these will often render sedatives for the cough unnecessary. The *pyrexia* in many cases does not require special treatment, but will subside under the general measures directed towards the reduction of local inflammation. Where this is not the case, and the excessive temperature, rising to 103° to 105° F., torments and wastes the patient, the best method of reducing it is by the open-air treatment to be mentioned presently.

The fourth object, namely, the palliative treatment, includes that of the various urgent symptoms.

The *cough*, when not reduced by the counter-irritation, may be to a certain extent allayed by a combination of sedatives, such as opium and its alkaloids, conium, henbane, diluted hydrocyanic acid, American cherry, with mild expectorants, of which spirits of chloroform, lemon-juice, and squill are examples. Where the cough is frequent and the expectoration difficult, and there is proof of active disease, tubercular or pneumonic, proceeding in the lungs, an effervescing saline, containing carbonate of ammonium, with small doses of opium and antimonial wine, taken two or three times at night, will greatly relieve the symptoms, the rule in the treatment of consumption being to restrict the sedatives, as far as possible, to the night, so as not to interfere with the appetite and digestion. The preparations of tar, in the form of capsule, pill, or solution, are useful in reducing profuse expectoration. The inhalations of iodine, compound tincture of benzoin, carbolic acid, creosote, larch, and turpentine are useful if expectoration is offensive or requires stimulating; or, again, those of chloroform, conium and hop, when the cough is convulsive and dry.

The *pains* in the chest may be alleviated by blistering, painting with tincture of iodine, or stimulating liniments, such as turpentine and ammonia; or else on Frederick Roberts's plan, by securing the immobility of the side by strapping.

Night-sweats, when profuse, may be reduced by oxide of zinc (gr. ij to iv), by gallic or sulphuric acids, by sulphate of iron, by arseniate of iron (gr. $\frac{1}{6}$ to $\frac{1}{4}$), or most effectually of all by the preparations of belladonna, in the form of the extract (gr. $\frac{1}{4}$ to gr. i), or as the solution of sulphate of atropine (m j to ij), or used hypodermically; but these often cause dryness of the mouth and fauces. Dover's powder in 10 gr. doses is useful, but nitrate of pilocarpine (gr. $\frac{1}{30}$), and picrotoxin (gr. $\frac{1}{60}$) in form of a pill, are more effectual.

Diarrhœa, where due to bilious derangement and an acid state of the *primæ viæ*, is best treated by mercurial aperients, combined with carbonate of sodium or lime-water. Where it partakes of the nature of a flux, accompanied by a pale tongue and great debility, it may be checked by astringents, such as hæmatoxylin, catechu, krameria, baël, and

carbonate or subnitrate of bismuth. When ulceration of the intestine is present, it is generally but not always characterised by a red, irritable tongue, pain and tenderness of the abdomen, and persistency of the diarrhoea. Here, as in other forms of ulceration, opium and its alkaloids answer best, and may be given internally with sulphate of copper (gr. $\frac{1}{4}$ to $\frac{1}{2}$) every three or four hours. When the stomach is too irritable to tolerate medicine by the mouth, opium or morphine suppositories are useful, but still better are opiate enemata, which, acting more directly on the ulcers, check the pain and diarrhoea, and often afford considerable relief. The amount of injection to be used at a time must be regulated by the probable extent of ulceration; and as a vehicle, in cases of irritability, linseed tea will be found most serviceable. In very obstinate cases tannic acid (four to five grains) and acetate of lead (three to four grains) may be added to the injection. The opposite state of bowels, namely, constipation, is very common in the early stages of phthisis, and is best corrected by changes in diet, such as the use of brown bread and oatmeal, cooked and fresh fruit, regular exercise, and, if these prove insufficient, a mild aloetic or rhubarb pill, or the use of some mineral water, as Friedrichshall, Pullna, Carlsbad, Æsculap, Rubinat, Hunyadi János, and others.

The *dyspnoea* of advanced cases generally arises from difficulty of expectoration and the greatly curtailed respiratory power, and may be relieved by spirit of ether, carbonate of ammonium, and other diffusible stimulants, or by the inhalation of oxygen or of iodide of ethyl (capsules *m iij* to *v* for inhaling). The pain arising from perforation in pneumothorax is best treated by diffusible stimulants, and strapping the side to limit the movements of respiration; and if much liquid effusion or accumulation of air takes place, it is sometimes advisable to tap the chest; but, as a rule, the state of the patient does not allow of very active measures.

Bed-sores should be prevented by the use of a spring- or water-bed, and the skin of the dependent parts can be fortified by a lotion of spirit and water (1 part in 4). If a bed-sore has formed, it is best to protect it from friction by the use of circular air- or down-cushions, or thick felt-plaster, and the raw surface can be painted with collodion, or be regularly dressed.

3. *Diet*.—The great object being to introduce as large a quantity of nutritious food as can be digested, abundance of meat, plainly cooked, with fresh vegetables, and a fair amount of bread and starchy food, should be given. Fatty material, if it can be digested, should be largely represented in the dietary, and many physicians advise large quantities of cream, butter, and suet. It is found that under the open-air treatment very large quantities of all kinds of food can not only be taken but also assimilated by patients who are credited with weak digestions, and immense gain of weight amounting to 2 st. and 4 st. follows. Milk is the principal item, from 3 to 5 pints a day, with three large meat-meals, with abundant vegetables and bread-stuffs. When cow's milk disagrees, ass's or goat's—the unpleasant flavour of the latter disguised by orange-flower water—may be substituted with advantage. Koumiss and Kéfir, prepared from fermented mare's or cow's milk, are frequently used in Germany and Russia, but

they have not become popular in this country. The digestive powers being, as a rule, weakened, much good may be done by the addition of animal ferments, such as liquor pepticus and liquor pancreatis, to the food, which, becoming peptonised, is much more easily assimilated (*see* PEPTONISED FOOD). In the early stages stimulants are not largely required, as they increase the cough and pulmonary irritation; but when the strength fails, and the powers of digestion are weak, they may be given frequently in the form of brandy or whisky, and advantageously combined with liquid nourishment, such as eggs, soups, various meat-essences and panadas, arrowroot, and jelly. When wine is required, in chronic cases, it will be found that claret, hock, sauterne, and chablis tend to irritate the cough less than the stronger wines. Champagne should be given where the prostration is great, and it often is useful in aiding expectoration. In cases of laryngeal phthisis where the dysphagia is urgent, it is often advisable to feed the patient by nutritive enemata or suppositories.

Hygiene.—The consumptive patient should inhabit a well-ventilated, well-drained house, built on a dry soil, sand or gravel, sheltered from cold winds and well exposed to the south, not hemmed in by trees, the most suitable for the neighbourhood of the house being of the *Conifereæ* order. The bedroom should be lofty, provided with a fire-place for warmth and outlet ventilation; and the cubic space should be 1,500 to 2,000 cubic feet per head. The window should always be open night and day. Chowne's or Tobin's tubes must be employed to supply fresh air, even in stormy weather. Both bedroom and sitting-room should be exposed to the sun's rays, for the vivifying influence of which there is no substitute; and it is well to secure for the patient himself their benefit as long and as often as possible. All consumptives should be placed on the open-air system as early in the disease as possible, and the greater part of the day should be passed, not in the sitting-room, but reclining on a sofa or couch in a balcony, shelter, or 'Liegehalle,' under the full influence of the air, though protected from sun and weather. Patients in the prone position well covered with wraps endure with impunity the most severe cold and benefit greatly by this method of treatment. Pyrexia, often of long continuance, subsides in two to three weeks under this treatment when combined with large feeding. The sputum should be received into a vessel containing a disinfectant, to be frequently emptied, or it may be burnt, as is done at the Hospital for Consumption and Diseases of the Chest, Brompton.

Clothing and Exercise.—The underclothing should be woollen, either flannel or lambswool, or perhaps in summer merino may be allowed, the object being to secure a good non-conductor of changes of the temperature, which will at the same time absorb cutaneous moisture. The rest of the clothes must be adapted to the season, the invalids, male or female, always bearing in mind their greater liability to catarrh than ordinary persons, and using wraps freely, more especially when driving. The wearing of respirators, though undoubtedly a protection against cold, is not always desirable, as, unless the wire meshes are widely separated, they considerably impede free respiration, and form a kind of muzzle. A woollen scarf passed over the mouth and nose, in the fashion of

omnibus-drivers, or for ladies a good Shetland veil or 'fleecy cloud,' answers the same purpose without obstructing respiration.

Exercise must depend on the stage of the disease and the strength of the patient. In the first stage, especially when the disease is limited to one lung, and no fever or hæmorrhage is present, active exercise in the form of walking is advisable. Graduated ascents, as are used at Davos and Nordrach, are excellent when regulated by the medical attendant. Under careful superintendence, certain gymnastic exercises may be of benefit, which, by raising the arms, lift the upper ribs and increase the size of the thoracic cavity, especially in the upper regions, and thus necessitate a larger inspiration of air; and in time this leads to further development, and even to hypertrophy of the healthy lung. Emphysema may be produced in the diseased lung by this means, which is useful in limiting any further advance of infective tubercular disease.

Riding is excellent for a large number of patients, being intermediate between the active and passive varieties of exercise. Where the disease is more extensive and advanced only the passive forms of driving and sailing are possible.

4. *Climate*.—The main point to be held in view is to give the consumptive a climate in which he can live an open-air life and take abundant outdoor exercise, and experience that amount of stimulating influence which, while it improves his appetite and powers of digestion, does not irritate the mucous membrane of the lungs or increase the cough. The selection is generally difficult, and not only depends on the class of cases, but must be sometimes modified by individual peculiarities. *See CLIMATE, Treatment of Disease by.*

The writer's statistics, founded on 251 consumptives, who passed one or more winters in warm climates out of England, assign the most favourable results to sea-voyages, and next to Egypt and other dry climates. The Mediterranean basin, including Riviera, Malaga, Algiers, and the islands of this sea, follows next in point of success; while the moist temperate climates of Pau and Rome give far less good results, and Madeira only slightly surpasses these. The same statistics show the foreign health-stations to be on the whole more successful in prolonging life than the English ones; but we must not forget that the most advanced cases fall to the lot of the latter on account of the difficulty of travelling; and, on the other hand, a great advantage enjoyed by the home stations is the superiority of the food, which may in some degree compensate for the smaller number of days in which exercise can be taken, and the greater vicissitudes of weather. Of the British Channel health-resorts the more easterly, such as Hastings, Ventnor, and Bournemouth, have afforded more favourable results than Torquay and Penzance. It is impossible in a few sentences to lay down rules for climate selection, but a few general outlines may be given of the suitability of different groups of agencies.

The British south-coast stations are beneficial in scrofulous phthisis, and in many cases where the appetite is poor, and tendency to catarrh not the prevailing feature. They are beneficial too in cases of chronic limited cavity. In the catarrhal form of phthisis the Canary Islands, Madeira, and the West India Islands, especially the Blue Hills of Jamaica, are advantageous; the combination of

warmth with saline influence, and the absence of stimulating qualities, seeming to answer best.

Dry stimulating marine climates, such as the Riviera and Malaga, are recommended in phthisis supervening on inflammatory attacks, and in all cases where it is desirable to combine stimulating influence with a moderate degree of warmth, and decided dryness of atmosphere. Where greater warmth with a little more moisture is required, Algiers, and the islands of Corsica, Sicily, and Corfu will suit better. As better examples of warm dry climates may be instanced those of the Pacific—of Sierra Madre, Coronado Beach, Santa Monica, and Los Angeles.

Where the stimulating influence is undesirable, as in patients of excitable temperament, or irritable gastric mucous membrane, the *very dry inland climates*, like those of Egypt or South Africa, are preferred.

Sea-voyages to South America, Australia, and New Zealand, round the Cape, or the shorter one to the Cape itself, are indicated in cases of hæmorrhagic phthisis, of scrofulous phthisis especially with fistula in ano, of phthisis with emphysema, in cases of limited first or third stage, where the strength is unequal to much exercise, and where the patients have suffered from over-work or close confinement in crowded cities. *See SEA-VOYAGES.*

High altitudes.—The considerable mass of testimony in favour of this form of climatic treatment for consumption, in Europe, Africa, and North and South America, has rendered it the most popular climatic treatment of the day. The writer's statistics of 247 consumptives treated in Davos, St. Moritz, Colorado, and the South African highlands show that in unilateral tubercular consolidation more or less extensive improvement occurred in 97½ per cent., and complete arrest of the disease in 66¾ per cent., and that even in bilateral cases the percentage of arrest was 40. The results in cases of excavation were not so favourable, about 54 per cent. improving and 35 per cent. becoming worse. These results are the most favourable, but it must be remembered that the greater part were achieved in incipient and limited cases after residences varying from months to years. These patients are, however, liable to relapse, as occurred in a certain number of these cases.

At present the Andes, the Rocky Mountains, and the Alps, and even the South African highlands, are frequented by consumptives; but the conditions of temperature and altitude manifestly vary greatly, and while the climates of Quito and Santa Fé di Bogota resemble in temperature that of Malaga, the winter extremes of Davos and St. Moritz in the Alps are more nearly akin to those of Canada. The Colorado climate is a very sunny mountain climate, with considerable extremes, but drier than the Swiss. In all these places, however, there exists a distinctly specific influence apart from that of heat and moisture, in the form of diminished barometric pressure, producing rarefaction and diathermancy of the atmosphere, which is shown in the patients residing at high altitudes. The chest becomes expanded, and hypertrophy of the healthy lung-tissue takes place, accompanied by vesicular emphysema around the lesions. Patients with tubercular consolidation of one or both lungs, provided the lesions admit of sufficient lung-surface for proper aëration, the powers of the circulation be sufficiently good to allow of exercise, and there

be no pyrexia, are the proper cases for this form of climate, and in many of such complete arrest of the disease may perhaps be predicted. These high altitudes are also suited for hæmorrhagic phthisis or phthisis of distinctly hereditary origin, provided it be not too advanced; and they are contra-indicated in (1) patients of advanced years, (2) catarrhal and laryngeal phthisis, (3) phthisis with albuminuria, (4) phthisis with double cavities, and wherever the extent of the disease or the condition of the lung places the patient in the category of 'advanced cases.'

C. THEODORE WILLIAMS.

PHYSICAL EDUCATION. — **INTRODUCTION.**—*Physical education* concerns itself with the fullest development of the body, just as education, in the general sense of the term, has for its object the fullest development of the manifold qualities of the mind. A child of normal faculties brought up in the midst of a civilised people, and deprived of systematic teaching of any kind, would, when manhood was reached, exhibit a degree of intellectual development which might surprise those who can conceive no mental worth unless it be the product of the pedagogue. An individual thus left to himself would learn much from observation, experience, and example, but the methods of his mind would be clumsy and imperfect, and he would lack those arts whereby knowledge can be economically and systematically acquired and conveniently employed. He would, in fact, remain imperfectly developed, and would have missed the opportunity of making the best of his faculties.

It is precisely the same with the physical development of the body, if it still be assumed that the individual is born among a civilised people. To simply leave a child to his own devices when he is not engaged in school work, is not to provide him with a sound or even an efficient education of the body. It must be borne in mind that the modern child has departed very far from the primitive savage, and that his environment, the claims upon his energies and the trammels of civilisation, no longer render it possible to leave the perfecting of his body solely to 'nature.' It cannot even be said that the physical development of the higher races of savages, when mythological matter is excepted, leaves nothing to be desired.

Physical education, therefore, to be of the highest service, must be precise and systematic, must be graduated and progressive, and must be adapted to the personality of the individual. It must be conducted, indeed, upon precisely the same lines as is the better formulated education of the mind.

Physical education involves exercise and movement. Save by exercise, there is no means of developing any portion of the organism, even provided that the supply of food and of air be sufficient. Exercise means growth, functional vigour, and the maintenance of a high standard of organic life. Undue rest is followed by feebleness and decay. Absolute rest is found only in death.

It is assumed in the present article that exercise is confined within proper limits. The subject of the abuse of physical exercise is dealt with in another article. See EXERCISE.

1. The effect of Exercise upon the Body.—Exercise increases the size of a muscle, the stoutness of its tendon, and the power it can command. The stronger the muscles, the finer and denser are the aponeuroses with which they

are connected, and the firmer are the fasciæ which hold them in position. Muscles act upon articulations. The duly exercised joint has a good covering of cartilage, powerful ligaments, and well-developed bony parts. Exercise, moreover, influences the size of the bones upon which the muscles act, renders them stronger and denser, and emphasises their anatomical details.

Exercise induces a more vigorous respiration, and under increased breathing-efforts the lung-capacity and the size of the thorax are augmented. It accelerates also the blood-circulation, and the effect of an increased blood-supply upon the size and condition of the tissues concerned is well known.

The secret of the size and proportions of the future man lies buried in the ovum from which the individual is developed. The child of short and stunted parents will probably also be short and stunted, and remain so in spite of an elaborate physical training.

The effects of systematised exercise upon the growth and development of the body have been demonstrated by many observers, and have been expressed in the form of actual measurements by Maclaren and others. In the *Report* of the Anthropometric Committee, the measurements of eighty-nine professional and amateur athletes are given, with the following results: 'Their average stature exceeds that of the general population from which they are drawn by 0·68 inch, while their average weight falls short of that standard by 14·5 lb. The ratio of weight to stature is 2·100 lb. in the athlete, 2·323 lb. in the general population, for each inch of stature.' See also Cecil Hawkins's monograph on the Physical Examination and development of public-school boys.

In noting the effects of physical exercises by means of measurement, the most conspicuous attention is drawn to the increase in height and in the circumference of the chest. In young subjects there is strong evidence in support of the belief that systematised exercise may actually increase height. In cases in which the training has been commenced after the growth of the body has ceased, any increase in stature is to be ascribed to a straightening of the spine and an improved carriage.

In considering the general question of increase in chest-girth, care must be taken not to ascribe this improvement—as some are apt to do—entirely to an increase in the capacity of the thoracic cavity. This is probably in all cases of much less effect than muscular development. In measuring the chest, the tape passes over those very muscles which are prone to the most conspicuous development in those who practise gymnastic exercises, for example, the pectorals and the great muscles passing from the trunk to the upper limb. The effect of proper exercise in augmenting the respiratory capacity to a certain degree is, however, undoubted.

A physical training does something more than merely increase the size of the limbs, and add to the stature. It tends to render all parts of the body symmetrical, and more perfectly proportioned. Of all animals, man is the most subject to variations in proportion and symmetry. Not only do children often grow in a fitful and irregular manner, but they may exhibit unequal developments, one side appearing to be larger than the other. Such deviations, which are distinct from actual deformities, a well-directed system of training will usually correct.

There must needs be a limit to the growth of muscles, and those exercised to too great a degree will, after attaining a certain size, commence to waste.

Moreover, from exercise there result an increase in the contractile force of the muscles, and an improvement in the speedy and complete contraction of their fibres. The muscles of an athlete when in training contract with extraordinary force under the electric current; the muscular sense is developed to its utmost; the perfection of the reflex act is attained; the power of co-ordination possessed by the individual is augmented; and movements at one time complex and difficult are carried out with ease. In this way the nervous system is saved a great expenditure of force.

One conspicuous feature in muscular training is the increase in the possibilities of automatism, and another remarkable element is the economy of force which results from muscular education. He who has been well trained physically, possesses not only a complete, but an intelligent, use of his muscles. His movements are powerful, are under absolute control, are precise, and capable of the finest and most elaborate adjustment. The art of the athlete consists not in employing the greatest amount of power in effecting a movement, but in carrying out that movement with the least possible expenditure of force.

Not only does a systematic training promote the fullest growth of the body, and help the individual to attain to something approaching a perfect symmetry, but it gives to him an easy and graceful carriage, and a bearing which has about it the mark of vigour and completeness. With suitable exercises, the shuffling and shambling gait disappears, the loutish boy ceases to look loutish, and the gawky girl no longer excites comment, rounded shoulders become square, and bending backs are made straight. The athlete, so far as his body and his personal equation are concerned, has reached the full and perfect stature of a man; and the girl whose physical education has been complete, reaches her point of physical perfection as a woman. It must not be forgotten that the beauty of the body depends upon a fully formed skeleton and perfectly developed muscles, and not upon deposits of fat.

The tissues of the ill-developed are flabby and lacking in elasticity and consistence; those of the well-developed are, on the other hand, firm, resisting, and full of evidence of living. The delicate and sensitive complexion of a young woman whose physical training has been efficient is in conspicuous contrast with the dull lustreless integument of the individual who 'never stirs out of the house.' The skin of the recluse is grey, greasy, and unpleasant-looking. Exercise involves more living in the open air, a freer and deeper respiration, and the coursing of a more vigorous flow of blood through the integuments. In the matter of personal comfort, no greater sense of pure pleasure can illumine the human mind than that which results from perfect health; and such health cannot be attained without a full exercise of the manifold energies of the body. He who takes no exercise remains an imperfect creature—he misses at least one-half of the delights which are available to man during a comparatively short existence, and it is not to him that 'joy cometh in the morning.'

2. The effect of Exercise upon the Mind. Moderate, regular, and systematic exercise, by

stimulating the circulation of the body, improves also the circulation of the brain, and is therefore an aid to cerebral movements. By improving the health and physical strength it increases the capability of the individual for mental work, and for the physical strain consequent upon mental concentration. It offers, too, an admirable change of employment, and in this way becomes a valuable means of rest. 'Prescribe fencing, gymnastics with apparatus, and lessons in a riding school,' writes Dr. Lagrange, 'to all those idle persons whose brain languishes for want of work. The effort of will and the work of co-ordination which these exercises demand will give a salutary stimulus to the torpid cerebral cells. But for a child overworked at school, for a person whose nerve-centres are congested owing to persistent mental effort in preparing for an examination, we must prescribe walks or rides, the easily learnt exercise of rowing, and, failing better, the old game of leap-frog and prisoner's base, running games—anything, in fact, rather than difficult exercises and acrobatic gymnastics.'

An excessive and absorbing indulgence in physical exercises is undoubtedly bad, and it may be that in some public schools too great significance is attached to mere athleticism. The all-engrossing pursuit of athletics tends to make the individual too much of an animal, and to afford neither time, opportunity, nor suitable conditions for the development of his brain. Still, on the other hand, in these days of cramming and intense competition, many a man can base his success in life upon physical health before all things; and there are not a few who have attained to eminence among their fellows who have to thank Providence for the tardily recognised blessings of an idle youth.

The systematic and properly arranged pursuit of physical exercise tends to develop certain admirable qualities, and notably those which are so much prized among Englishmen, and which are well designated as 'manly.' These qualities are brought out in those who are enthusiasts in outdoor sports and games. The football-player has done more than merely develop his muscles; and the man who has rowed in his college eight has learnt something beyond the mysteries of the sliding-seat. Such lads and men have learnt in a school where the principles of pluck, courage, endurance, and self-reliance are acquired. They will have learnt to be ready, to be quick of eye and hand, and prompt in judgment. They will have appreciated the value of discipline and of self-control. They will have felt the inspiration of the chivalry of days gone by, and have experienced the influences of good fellowship and loyal comradeship. They will probably have learnt what it is to be patient, to be fair, to be unselfish, and to be true. The following utterances by the head-master of a large public school in England are worthy of note: 'The worst boys intellectually, physically, and morally are the loafers.' 'The boys who work hard and play hard do not ape the vices of men, and are free from the insidious evils that often fasten on unoccupied boyhood.'

3. The Elements of Physical Education.

(a) *The exercises should be adapted to meet the needs of each individual case.*—The object of a proper physical education is to develop health and not mere strength, to bring the body to its highest degree of perfection, and not to convert children and youths into gymnasts and acrobats. Its prin-

principal purpose is to best fit the individual for the duties and work of life, and not to elicit proficiency in mere feats of skill and adroitness.

It must not be forgotten, moreover, that individuals vary greatly in the quality of their physical powers, and in their capacity for muscular exercise. It is just as impossible to form a great mass of children into one gymnastic class as it is to place those children in one school standard under one teacher. Neither age, height, size, nor sex affords sure means of classifying children, so far as the needs of a proper physical education are concerned. Each individual must be considered upon his or her own especial merits, and there is no method of physical training which is universal or all-sufficing, and adapted for all sorts and conditions of human beings. The sending of a child to a gymnasium, or the placing of it under the care of a drill-sergeant, is as crude a procedure as the conducting of a child within the walls of the first school met with, and leaving it there with the impression that it will somehow be educated. Physical education requires as much care as does mental education, and calls for as much subdivision both in the teaching and in the taught. Before planning out a course of instruction, a child's physical condition should be inquired into with as much care as is exercised in examining an adult for life-insurance.

(b) *The exercises should be carefully devised, systematically arranged, and suitably graduated.*—The exercises should be planned upon a definite system, should be suited for the individual, and should aim at the equal employment of all the muscles, and not at the development of a few. The work in an ordinary gymnasium tends to throw strain mainly upon the upper extremities, while most of the outdoor games tend to develop the lower limbs. No great good can be obtained from tedious drilling and purposeless marching, and the time devoted to physical training should never be so absorbing as to allow no leisure for games and other pleasant forms of recreation. It is desirable that the lessons should be as varied and as interesting as possible, and that reasonable opportunity be given for competition, and the encouragement of those who are specially fitted to excel. In every instance violent intermittent exercises should be forbidden, and the performance of feats of strength should never come within the scope of the education scheme.

(c) *The exercises should be carried out under proper guidance, and with suitable and efficient apparatus.*

(d) *The time for the exercises should be carefully selected.*—Violent exercise after a full meal is obviously bad, and a course of physical instruction should not be carried out in the case of children who are tired from a long day's attendance in school, or who are feeble from want of food.

In the matter of schools, it is well that the period for physical training should be interpolated among the hours devoted to ordinary school work. The Rev. Dr. Warre, of Eton, advises that a schoolboy's day should be disposed of as follows: Rest, ten hours; work, seven hours; meals and play, seven hours.

So far as adults are concerned, the taking of violent exercise in the evening, after a long and arduous day's work, is often injurious in its result. There is no better time for such individuals than the early morning.

(e) *Exercises, so far as is possible, should be taken in the open air, or in a large and well-ventilated room, and the subjects of the instruction should be properly clad.*—The atmosphere of many gymnasia and fencing saloons is, especially in the winter-time, close and unwholesome. The garments worn should be light, loose, and always made of wool.

4. The Selection of Exercises according to Individual Needs.

Children.—The physical training of the child should be commenced early, should be made as interesting as possible, and be represented in the main by what may be termed scientific romping.

The exercises should be given, whenever possible, in classes.

The set exercises should not be too formal, and never be too long; and in no instance should they be allowed to take the place of the ordinary outdoor games of children.

Games which involve shouting should be encouraged, and a very prominent position given to running, skipping, games with balls, and jumping.

The most rudimentary of all games, 'touch,' is one of the most excellent. The upper limbs may be encouraged by such amusements as battledore-and-shuttlecock, and the lower by such a game as hop-scotch.

The set exercises should take the form of what are known as Swedish gymnastics, the vocal march, musical drill, and the class exercises with dumb-bell and bar-bell.

Children should avoid exercises of strength; and, in the main, exercises of speed. There is little need for especial gymnastic apparatus. Those best suited for children are the climbing-rope, the inclined ladder, the vaulting-horse, and the parallel bars.

Girls and Women.—The physical condition of a large proportion of the girls and women in this country is quite deplorable, especially among the middle and upper classes. It is apt to be ascribed not to a totally neglected education, but to the belief that growing girls are always awkward, uncouth, and weedy. This belief is not well founded.

The girl is too often encouraged to be dull, to be prim, to be subdued, to suppress the outbursts of pure animal spirits. She is more or less under the curse of that detestable adjective, 'lady-like.' She spends hours in an ill-ventilated schoolroom and upon a piano-stool, and the rest of her time is occupied in eating and sleeping, in preparing lessons, in stooping over needlework, and in taking formal walks with the governess.

A good digestion and vigorous lungs are more useful to a woman in the battle of life than a knowledge of advanced mathematics; and sturdy limbs and strong hands are of more value to the mother of children than even decimal fractions and a familiarity with irregular verbs.

Younger girls may pursue the exercises named in dealing with the education of children. Those who are a little older have an infinite variety of healthy pursuits at their service—running, skipping, outdoor games, riding, skating, swimming, cricket, games with balls, archery, tennis, and certain exercises in the gymnasium. They should practise also such movements as develop the abdominal muscles, and should never neglect rowing.

Fencing is good; a tendency to flat feet and weak ankles may be met by such games as hop-scotch, by step-dancing, and the hornpipe.

Cycling may be practised in moderation. Jumping is probably not advisable for girls who have passed the period of puberty.

For women, such exercises as have just been detailed are open, with the obvious modifications which their age and dispositions suggest.

Lads.—Lads between fourteen and eighteen have almost every form of exercise and physical recreation open to them. They should avoid feats of strength, paper-chases, and exercises of extreme speed, such as sprint-running, which are apt to cause strain of the heart as well as other kinds of injury.

Adults.—Adults between eighteen and twenty-five have the whole of the joys of the athletic world open to them, and if a man keep in training and in practice his period of athletic life may be extended to thirty. The middle-aged and elderly must anticipate a progressive curtailment of their more active pursuits. There remain, however, walking, and all the milder forms of outdoor exercise—riding, rowing, skating, cycling, golfing, and the use of the simpler gymnastic apparatus. After thirty, few men are capable of undertaking exercises of speed without actual risk.

FREDERICK TREVES.

PHYSICAL EXAMINATION.—The object of a physical examination is to ascertain the precise seat, limits, and characters of those evidences of disease which are recognisable by our senses, and which are called physical signs. In the present article a description will be given of the physical examination of—(1) the Patient Generally; (2) the Respiratory System; (3) the Organs of Circulation; (4) the Mediastinum; and (5) the Abdomen. *See also* NERVOUS SYSTEM, Clinical Examination of.

I. General Survey.—Our attention will first of all be naturally attracted to the *physiognomy* of the patient, that is, to his general appearance and build. We note his apparent *weight* and *height*, and, if possible, correct our observation by scale and measure. We observe the *state of nutrition*, firmness or laxness of muscle, corpulence, thinness, emaciation—atrophy of any particular muscle or group of muscles. The *complexion* of the patient is to be remarked, whether clear, sallow, dark, fair, jaundiced, or pigmented: also lividity or pallor of surface and mucous membranes. The *apparent age* as contrasted with actual years of the patient; elasticity of features, condition of hair, presence of arcus senilis. The *symmetry* and *play of features*, the expression whether of vivacity, despondency, suffering, anxiety, paralysis, or hysteria. *See* PHYSIOGNOMY.

While making these preliminary observations, a general outline of the history of the patient and of his present illness will have been elicited.

The *pulse* should next be noted (*see* PULSE). We may, in important cases, extend our inquiries or record our observations by means of the sphygmograph. *See* SPHYGMOGRAPH.

The *respiration* of the patient requires attention as regards rapidity; mechanism, that is, whether abdominal or thoracic in normal proportion; rhythm, regular or irregular, easy or laboured; and freedom or otherwise from pain. The action of the nares, and any recession or otherwise of soft parts during respiration, should be especially observed.

In health and under physiological conditions of age, exercise, emotion, &c., there is a tolerably constant ratio between the respiration and pulse-rate, namely, one respiration to from three to four pulse-beats. In disease this ratio is often much altered. The average respiration-rate in a healthy adult is from 17 to 20 per minute, in the infant about 40 per minute, between one and five years about 26 per minute. In old age the respirations are very slightly accelerated; in children they are quick and often irregular, being momentarily suspended by anything that excites their wonder or close attention.

The *odour of the breath* may attract attention. It may under morbid conditions be fetid, urinous, 'mercurial,' alcoholic, or gangrenous. *See* BREATH, THE.

The condition of the *skin*, whether dry or hot, moist or sweating, and the presence or absence of any eruption, scars, ulcers, or pigmentation, will be duly noted. The presence of *pyrexia* will be exactly ascertained by the use of the clinical thermometer. The condition of the surface may be tested by the hand. The use of the surface-thermometer, in combination with the ordinary clinical instrument, would give us more exact results; but the hand of the skilled observer generally suffices for the purpose. *See* THERMOMETER, CLINICAL.

The condition of the *finger-ends*—clubbing, lividity—must be observed (*see* FINGERS, Clubbing of). Important information as to the previous acute illnesses within the past six months can be obtained by inspecting the nails, a transverse furrow marking the period of defective or arrested nutrition during such illness.

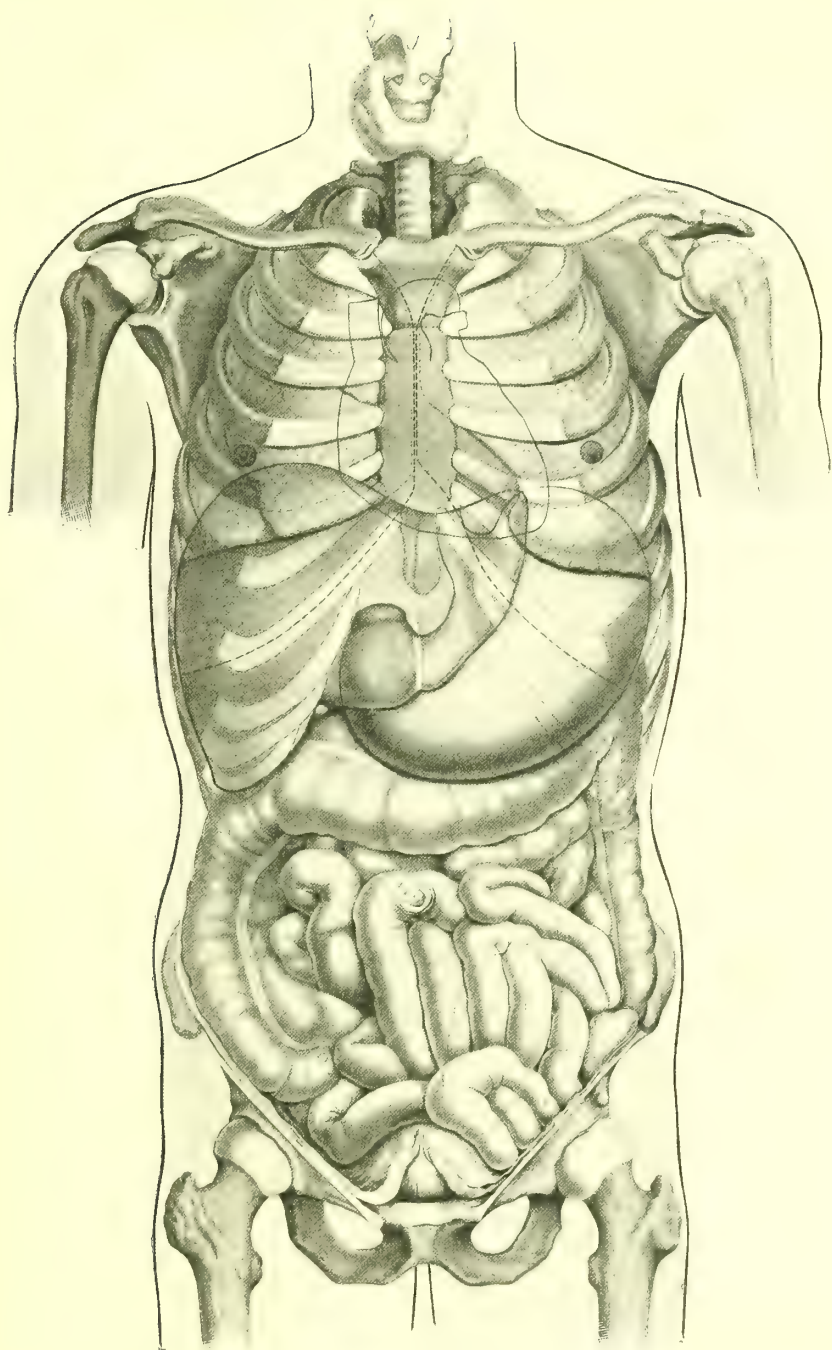
The condition of the *teeth*, *eyes*, especially of the *pupils*, *tongue* and *gums* furnishes us with valuable information. *See* TEETH; EYE; PUPILS; TONGUE; and MOUTH.

The careful superficial inspection of the patient in the manner above sketched will perhaps at once suggest a more minute examination of some one organ or system of organs as the probable seat of disease; and having thus far succeeded in locating the disease, the other organs and functions of the body will of course come under review, but the physician will be more especially inquisitive with regard to organs or functions associated with those

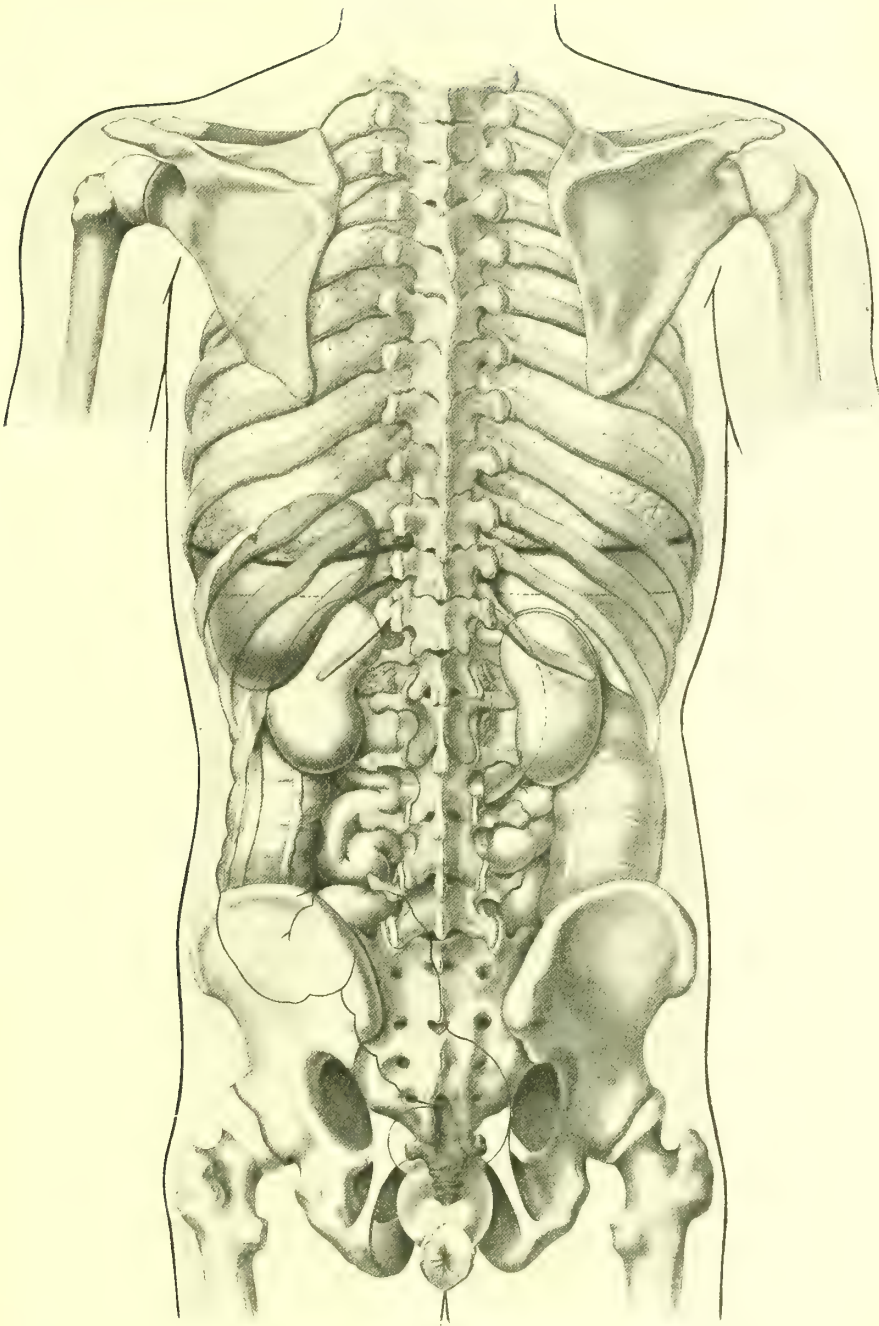
PLATES XV. and XVI.

The anterior line of reflection of the two pleuræ is somewhat variable, for the meeting of the two sacs, though usually placed behind the sternum, may lie quite to the left of the median line. Also the line of reflection of the left pleura may diverge from that of the right as high up as the level of the fifth costal cartilage, or on the other hand the two pleuræ may remain in contact as far down as the ensiform cartilage. The area of pericardium left uncovered by pleura varies in a corresponding manner. The lines indicating the pleural reflections in Plates show the average positions. The inferior line of reflection of the pleura in the mid-lateral line is rather lower on the left (lower border of tenth rib) than on the right side (lower border of ninth rib). Posteriorly the pleura may descend as low as the first lumbar transverse process.

The position occupied by the vermiform process varies considerably, though it will usually be found either as indicated in Plate XVI. or, lying behind the ileum and mesentery, directed upwards and to the left from its origin at the inner and back part of the cæcum.—G. B. MOWER WHITE.



Modified from Luschka by G. Mower White.



Modified from Luschka by G. Mower White

in which disease has been detected. If careful examination fail to reveal any organic lesion sufficient to account for the symptoms present, we may then—but not till then—refer the case to one of those diseases which for a time run their course without manifesting any definite lesion.

2. Nervous System, Physical Examination of.—See p. 1063.

3. Respiratory System, Physical Examination of.—The respiratory system includes the respiratory tract and lungs.

(A) NARES.—The shape of the nose, the narrowing of the anterior nares and the marked naso-labial fold with the mouth partly open would suggest the obstruction of the upper air-passages by enlarged tonsils or adenoids. The condition of the nasal mucous membrane and the character of nasal discharges should also be observed. See NOSE, Examination of; and TONSILS, Diseases of.

(B) LARYNX.—The condition of the larynx and trachea is examined into by listening to the voice, whether husky, altered in tone, or suppressed. Any tenderness or external deformity is ascertained by careful palpation.

By means of the laryngoscope the condition of the epiglottis, larynx, and trachea can be thoroughly explored. See LARYNX, Examination of.

(C) CHEST.—In making an examination of the chest, the physician should follow a methodical routine of inspection, palpation, percussion, auscultation, and occasionally radiography.

(1) Inspection.—The general shape and build of the chest is observed—whether it be the broad, well-formed chest of robust health; or the small, narrow, long chest, with antero-posterior and lateral diameters diminished, costal angle narrow, and ribs oblique and approximated—adapted to small lungs, or sometimes due to chronic obstruction to the entry of air through the upper air-passages. Or the thorax may be unduly expanded, with wide intercostal spaces, straightened ribs, widened costal angle, and deep antero-posterior diameter, to accommodate large lungs. Again, the thorax may be distorted by various kinds of spinal curvature, or as the result of rickets, or from external pressure, as in the depressed lower sternum of shoemakers. Lastly, there may be local flattenings or bulgings. See CHEST, Deformities of.

The movements of the chest are of great importance in diagnosis. We estimate the freedom or otherwise with which air enters the chest during inspiration by the equable expansion of its several parts, or by the immobility or recession of any portion the entry of air into which is retarded or impeded; and this can be accurately done by means of the pneumograph. In cases of general obstruction to entry of air, whether by impediment at the main air-passage or in its entire distribution, there is universal recession of all the soft parts—the supra-clavicular region sinks downwards, the hypo-chondria recede, and the intercostal spaces deepen during the effort to expand the chest against atmospheric pressure. On the other hand, when the difficulty of expansion, whether from intrinsic disease or obstruction of passages, is restricted to one side of the chest or to a portion of one lung, the restrained expansion during inspiration is limited to that portion. Thus from inspection alone we may often form a shrewd guess as to the seat and even the nature of the disease present.

In estimating local alteration of shape, the eye is perhaps more useful than any instrument of measure, and inspection should be made from above as well as from in front and behind. Calipers of various patterns may be used for taking diameters in different directions, and for recording differences of shape on the two sides the *cyrtometer* is very useful. This instrument was originally introduced by Woillez, and consisted of two halves of a jointed whalebone measure, connected by a hinge, which could be adapted accurately to the shape of the chest, and after removal the various curves on the two sides could be traced on paper. The *cyrtometers* now most in use are made of soft metal, two sufficiently long pieces of which are connected by an indiarubber joint or hinge.

Double tape-measures are also used for ascertaining the circumference on the two sides, and by their means the relative expansion during respiration on the two sides can be compared. Various forms of stethometer have been designed for the same purpose. See STETHOMETER.

The vital capacity of the lungs may be very accurately estimated by means of the spirometer. See SPIROMETER.

(2) Palpation.—Palpation is employed in aid of both inspection and percussion.

(a) During preliminary inspection of the chest the position of the heart's apex-beat should be invariably, and as a matter of habit, ascertained, and any deviation from its normal seat, namely, the fifth intercostal space one inch to the sternal side of the left nipple-line, should be noted. The position of a patient during examination should be borne in mind, the effect of posture upon the heart's position varying in different people.

(b) Any local bulging or tumour will naturally be manipulated to ascertain its relation with bone, or soft structure, whether it be solid or soft, oedematous, fluctuating or pulsatile.

(c) In connection with percussion, the trained observer will note differences of resistance, as well as of sound, over diseased areas. Vibration felt along the rib by one hand when its vertebral end is struck by the finger of the other hand has been found to be increased in fluid effusions (Kellock).

(d) Increase or diminution of vocal vibration or fremitus will be noted over any spot of altered resonance, by applying the hand and making the patient utter some resonant words, such as 'ninety-nine.'

Vocal fremitus is increased by consolidation of lung (unless the bronchus be obstructed by growth or otherwise); diminished by much thickening of the pleura, by obstruction to the main bronchus, or by air in the pleura; annulled by fluid in the pleura. In many cases, however, of fluid in the pleura some vibrations are felt, probably communicated from above. The loudness or feebleness of the voice, and also its pitch, must of course be taken into account in estimating fremitus, and corresponding parts on the two sides should always be compared.

Loud, coarse, bronchial rales may cause the chest-walls perceptibly to vibrate, producing *rhonchal fremitus*. Pleuritic friction may likewise be perceptible to the hand applied—*friction-fremitus*. In cases of effusion into the pleural cavity, or in hydatid cysts near the surface, fluctuation may be elicited on palpation.

(3) Percussion.—Percussion is the method of

examination by which we detect the various degrees of resonance of different parts of the chest, depending upon the relative amount of air and solid structure.

It is best to use the fingers for percussing, one finger of the left hand being placed firmly over the point to be percussed, and struck with one or two of the fingers of the right hand, semi-flexed, so that the tips of the fingers fall vertically upon the pleximeter-finger. Percussion should be made from the wrist, not from the elbow; the stroke should, as a rule, be light, and always perfectly even on the two sides; sometimes a heavier stroke may be needed, but, as a rule, far more information is obtained from light than from heavy percussion. In comparing the percussion-note over the two sides of the chest, points exactly corresponding must be taken, and the pleximeter-finger must be placed in a corresponding position; for example, it must not be placed parallel with the ribs on one side and across them on the other.

The sense of touch is very valuable in percussion in estimating *resistance* of the part struck. Dulness, and particularly the hardness and want of resilience over thickened adherent pleura, may thus be readily *felt* by the pleximeter-finger during percussion. This sense of touch should be carefully cultivated, and its deprivation is a great disadvantage in the use of the artificial pleximeters and percussors first introduced by Piorry, although possibly these may be useful for demonstration to a class. The observer should not be content with comparing corresponding points on the two sides of the chest from above downwards, but he should invariably trace any dulness or resonance from either side across the sternum to ascertain the limits of resonance or dulness in this direction. From neglect of this, important information is often missed. The height to which the pulmonary note extends above the clavicle on the two sides should be compared.

Regions of the chest.—For convenience in describing the distribution of signs, both of percussion and auscultation, it is customary to divide the chest into regions. The names employed to distinguish these regions sufficiently define their limits, namely, the supra-clavicular, clavicular, infra- or sub-clavicular, mammary, infra-mammary regions on each side in front; the superior and inferior axillary regions; the supra-spinous, infra-spinous, inter-scapular, and infra-scapular regions on each side posteriorly.

(a) **NORMAL PERCUSSION-SIGNS.**—There is a certain standard degree of resonance over the lungs, only to be duly estimated by experience, which is known as *normal pulmonary resonance*. In certain regions of the chest the pulmonary resonance is naturally lessened or replaced by dulness. Pulmonary resonance should commence $1\frac{1}{2}$ inch above the level of the clavicle. In the clavicular and sub-clavicular regions, on firm percussion, the note should be even on the two sides, as low as the third rib. Below this level, on the *right* side, we still obtain full resonance until we arrive at the fourth space, where in the mammary line the note becomes slightly raised and shortened, becoming dull in the fifth space and downwards to the margin of the cartilages. On very light percussion the pulmonary resonance may be obtained half a space lower; and at least an inch to two inches difference in level may be obtained between the extreme limits of deep expiration and inspiration. In the lateral

(axillary) region the limit of percussion-resonance reaches about an interspace lower. At the sternal margin it is a little higher, from the encroachment of the right side of the heart upon the inferior angle of the lung. Roughly, and for clinical purposes, a line drawn outwards from the base of the xiphoid cartilage may be said to define the upper border of the liver-dulness.

On the *left* side, in the line midway between the sternum and nipple, we already, at the third cartilage, obtain elevation of pitch and shortening of the percussion-note; and at the fourth space dulness, from the underlying heart. Between this (mid-sterno-nipple) line and the sternum, and bounded above by the fourth cartilage and below by the level of the apex-beat, is the normal area of superficial cardiac dulness. In the nipple-line at the corresponding levels¹ some deadening of percussion-note may be obtained, but pulmonary resonance is otherwise clear to the sixth rib; in the lateral axillary region to the seventh. Below the sixth rib in front, and the seventh laterally, gastric resonance is obtained.

Over the *sternum*, percussion is naturally somewhat wooden and resisting, within degrees varying with the condition of the bones. The first piece of the sternum is normally somewhat less resonant than the next two pieces, but it should be, on firm percussion, by no means dull. Below the level of the fourth cartilages the heart and liver cause the note to be dull, although even here a certain degree of resonance is in health communicated from the adjacent right lung.

In the *posterior* regions of the chest the degrees of resonance are almost entirely in accordance with the thickness and character of superjacent tissues. Thus in the scapular and inter-scapular regions increased force of percussion is necessary to elicit pulmonary resonance, while in the lateral and infra-scapular region the percussion-note is full and low-pitched. On the right side this resonance is replaced by dulness below the tenth rib, and deep percussion will elicit a certain impairment of resonance as high as the ninth rib, in the mid-scapular line. On the left side resonance should be good to the extreme base, except that in the posterior axillary line a small and restricted area of dulness may be sometimes made out, corresponding with the position of the spleen.

(b) **MORBID PERCUSSION-SIGNS.**—Modifications in the distribution of percussion-resonance over the chest may be produced either by general or by local causes.

General causes.—Pulmonary vesicular emphysema, by enlarging the lungs and extending their boundaries, causes encroachment of pulmonary resonance over those regions—the præcordial, right infra-mammary, sternal, and right inferior basic—which are normally dull. In congenital smallness of lungs the boundaries of pulmonary resonance are somewhat retracted, so that liver-dulness in front and behind is slightly higher, and heart-dulness more extensive.

Local causes.—One class of these are encroachments of other organs. Enlargement of the heart will cause increased area of præcordial dulness upwards and to

¹ By employing the terms 'lines' and 'levels' to mean the vertical lines and horizontal levels, in connection with definite anatomical points, e.g. mid-scapular, nipple-lines, nipple-, second-, third-, fourth-, &c., rib levels, any portion of chest-surface may be accurately defined.

the left, or upwards and to the right, according as the left or right side of the heart is most affected. Effusion into the pericardium will cause similar dullness, extending upwards towards the manubrium sterni, and to the right beyond the sternum. Retraction of one lung is a frequent cause of extended (uncovered) cardiac dullness. Aneurysmal tumours in connection with the heart or great vessels give rise to dullness, chiefly in the neighbourhood of the sternum above the fourth cartilage, or in one or other inter-scapular region. Enlargement of the liver and spleen will cause them to encroach upon the pulmonary resonance. Effusion into the peritoneum, if extensive, will cause displacement upwards of the abdominal organs and diaphragm, encroaching upon the lower area of pulmonary resonance, and even causing collapse of the lower portion of the lungs, thus giving rise to dullness.

Effusion of fluid into the pleura will give rise to absolute dullness to the level to which the effusion extends upwards. The upper boundary of this dullness, if the lungs be sound, varies slightly with the position of the patient. In order, however, accurately to define the upper margin of dullness from fluid effusion the lightest possible percussion must be employed. In any case of considerable effusion into the pleura the dullness encroaches upon the median line, and towards the opposite side. See PLEURA, Diseases of.

The chief kinds of morbid percussion-signs will now be discussed.

Dullness, hardness, flatness.—These terms are by no means synonymous with regard to percussion-sounds. *Dullness* varies infinitely in degree. Thus over a pleuritic effusion the tonelessness is absolute; and to this degree of completeness of dullness the term *flatness* of percussion-note is sometimes applied. There are but a few other chest-conditions in which such absolute dullness is obtained; for example, extensive pericardial effusions, hydatid tumours, extensive malignant growths invading the lungs and infiltrating the bronchi. In inflammatory consolidation of the lung there is always a certain degree of wooden tone in the percussion-note. In cases of scattered patches, or nodules, of consolidation in the lungs, with air-containing tissue around, the dullness may be only very slight, amounting to a mere shortening of the note with elevation of pitch. In estimating the slight shades of dullness elevation of pitch is the first point to arrest the attention. *Hardness* of percussion, always more or less appreciable with dullness, is associated especially with consolidations of lung overlaid by thickened adherent pleura.

Skodaic resonance.—In all cases of considerable effusion of fluid into the pleura, in which the lung is not completely collapsed, a peculiar high-pitched tympanitic resonance is found at the sterno-clavicular region on the same side. This resonance, called Skodaic resonance, is a very characteristic sign, and has been attributed to relaxation of lung still in contact with the chest-wall. As the effusion advances completely to fill the chest, this resonance becomes replaced by dullness.

When effusion of fluid follows upon pneumothorax, the lung, unless held above by strong adhesions, is already completely collapsed; and above the level of the dullness caused by fluid there is a tympanitic note, caused by free air in the pleura. In this case the level of the fluid in the pleura shifts with every change in the position of the patient.

Whether the effusion be of serum, pus, or blood, the percussion-signs are the same.

Wooden percussion-note is obtained by percussing over thickened pleura with some air-containing tissue beneath. The sense of resistance is marked, the pitch high, and the duration of sound short. This degree of dullness, with increased resistance, is commonly present below the clavicle in cases of phthisis, with thickened pleura, and perhaps small, empty cavities, bounded by hardened lung-tissue.

Amphoric or tubular percussion is the sound elicited by percussing over a superficial empty cavity, connected by adhesions to the chest-wall. The pitch varies with the size of the cavity, but is always somewhat high. The sound can be exactly imitated by percussing the cheek drawn tensely over the teeth, with the mouth slightly open.

Cracked-metal sound, or bruit de pot fêlé, is obtained by sharp percussion over a cavity such as the above. Sudden displacement of air in the cavity will cause the sound, which somewhat resembles that produced by placing the two hands hollowed in apposition, and striking upon the knee. A little secretion in the cavity will facilitate the production of the sound. This physical state may often be appreciated by the touch before the sound can be heard. It is of little clinical value.

Bell-sound is elicited by combined percussion and auscultation, and *when present* is characteristic of a very large thin-walled cavity in immediate contact with the thoracic wall, and is most generally significant of pneumothorax. The stethoscope must be applied over the resonant part of the chest, and at another point within the same area a piece of metal, such as a coin, laid upon the chest, must be smartly struck with a second piece of metal. The auscultator hears a sound of a clear bell-like character within the chest, which is of quite a different quality from that produced by the mere contact of metals. It is essential for the production of this sign that the stethoscope and the struck metal be both within the area of chest-surface corresponding with the air-containing sac of the pleura. If, for instance, either be placed over a point below the level of any fluid effusion present the sound will be lost, to be recovered on altering the position of the patient so as to displace the fluid. By means of this sign, the limits of a pneumothorax may be accurately defined.

Hydatid fremitus is a vibratile sensation, sometimes to be felt on smart percussion over an hydatid effusion. In cases of pyopneumothorax a similar sensation may sometimes be felt, on percussing at the exact level of the surface of the effused liquid.

(4) *Auscultation.*—Auscultation simply means the act of listening; but the art of auscultation implies a great deal more than this, namely, the appreciation of the healthy or morbid conditions which produce the sounds heard on applying the ear to the chest or to other parts. If the ear of the observer be directly applied to the chest or part under observation, auscultation is said to be *immediate*. If some substance or instrument be used as a medium between the ear and the part under observation, *mediate* auscultation is said to be practised. Such an instrument is named a stethoscope. See STETHOSCOPE.

(a) *NORMAL RESPIRATORY SOUNDS.*—If the stethoscope be applied over the trachea of a healthy

person, tubular, blowing, or bronchial respiration is heard—that is, a sound as of air blown to and fro through a tube with moderate velocity; the mechanism of the sound being the entrance and outflow of air-currents through the narrowed glottic aperture of the trachea, producing sonorous vibrations within the tube below. As the stethoscope is passed downwards to the first piece of the sternum, the same sound is still heard, but more distant and muffled. In the upper inter-scapular region, where the great divisions of the bronchi are comparatively superficial, the tracheal sounds may still be indistinctly recognised; but below and to the side of these points these sounds are normally obscured by the vesicular pulmonary sounds, into the production of which they, however, necessarily enter.

The *pulmonary vesicular breathing-sound* is produced by the friction of air entering the air-sacs from the minute bronchioles, and it is supplemented by the condition of what remains of the glottic breath-sound, now infinitely subdivided. During calm breathing, the sound accompanying *inspiration* should be soft and breezy, giving the idea of innumerable similar and associated sounds. In intensity the sound is uniform from commencement to near the end, when it fades without perceptible interval into the expiratory sound. The *expiratory* sound commences at the moment inspiration ceases, being continuous with the inspiratory sound, but it rapidly fades in intensity, ceasing to be audible after the first one-fifth or one-third of the expiratory act. Of the time occupied between the commencement of one inspiration and that of the next, the inspiratory act occupies nearly one-half ($\frac{1}{2}$ ths); the expiratory act occupies the remainder, with the exception of a very brief interval of pause, between the end of expiration and the commencement of the next inspiration. It may be here observed that when the 'expiration' is said to be *prolonged*, it is meant that the expiratory sound is audible through a longer period of the act than natural.

If the respiration be hurried and forced, the inspiratory sound is coarser and louder, and the expiration more audible, these sounds approximating to the *puerile* breathing which is normal to young children.

In health the vesicular breath-sound should be about equally well heard over the front and back of the chest, allowance being made for additional thickness of covering over certain regions.

(b) MORBID RESPIRATORY SOUNDS.—*Puerile, compensatory or supplementary breathing* is characterised by increased loudness of vesicular breath-sound with some prolongation of expiration. Besides being audible over the chest generally in healthy young children, this exaggerated breath-sound may be heard over certain parts of the chest in persons who have some other part disabled or diseased. Thus, with effusion of fluid into one pleura, the respiratory sounds over the opposite lung are exaggerated or puerile. If one apex be diseased, the breath-sound at the other apex is exaggerated. This increased breath-sound to make up for deficient function elsewhere is called *compensatory or supplementary breathing*.

The breath-sound may be *enfeebled* over the whole chest, as in cases of emphysema or thoracic muscular debility. Localised enfeeblement of breath-sound may be due to several causes—(1) local emphysema; (2) adherent and thickened

pleura, as after old pleurisy at the base; (3) blocking of the alveoli by catarrhal products—common in commencing phthisis at one apex; (4) closure of bronchial tubes by plugs of mucus, or from spasm. If the rest of the lungs be free this local enfeeblement is made up for by compensatory breathing on the opposite side, or in other parts of the same lung.

Suppressed breath-sounds signifies removal of lung from the surface by effusion of air or of fluid into the pleura, or occlusion of a main bronchus by compression or morbid growth.

Wavy and jerking respiration are terms characterising a kind of respiration in which the inspiration is either partially or completely interrupted several times. The expiration is rarely thus affected. Waviness of respiration may be due—(1) to an irregular action of the inspiratory muscles, common in nervous people; (2) to cardiac impulse, in which case these interruptions are rhythmic with the heart's pulsation; (3) unequally distributed impairment of the lung-elasticity, for example, in early tubercular deposits. Walshe considered that pleuritic adhesions may have the same effect. It will be seen then that waviness of breath-sound is very commonly independent of any organic change, and requires other signs to render it of any value in diagnosis. Jerking respiration or interrupted breath-sound is more commonly due to organic lesions of the third kind mentioned.

Cogged breath-sound is a somewhat clumsy term applied to a form of interrupted respiration in which the interruptions are very even, three or four to each inspiration. Much importance is attached to the sign by some authors. It appears to be due to obstruction in the smallest bronchioles, either by dryish secretion or small nodules of tubercle, requiring some accumulation of inspiratory force to overcome it. The sounds commonly give place to a bubbling rale.

Harsh respiration with prolongation of expiration implies a want of vesicularity in the sound. While vesicular breath-sound has been compared to the sound produced by the breeze passing through leaf-laden trees, harsh breathing, on the other hand, resembles a similar breeze traversing their naked branch-tops. Some prolongation of the expiratory sound is inseparable from harshness of breath-sound. Harshness of breath-sound by no means implies increased loudness—rather the contrary. Enfeebled respiratory murmur is commonly harsh—always so when due to alveolar obstruction. The meaning of harshness of breath-sound is simply commencing consolidation; it goes with incipient dulness, and is one of the earliest signs of apical disease in consumption. There can be little doubt that its real mechanism depends upon the extinction of the vesicular part of the normal breath-sound, and the better conduction of the glottic sounds, which at peripheral parts of the lung are usually muffled and obscured by the vesicular sounds. The prolongation of the expiration is very characteristic of this early alteration of the respiratory sounds; and it may here be observed, in passing, with regard to morbid breath-sounds, that the expiration is the most important part of the respiratory act to attend to in auscultation.

Divided respiration, usually described as a separate evidence of disease, is really an inseparable factor of harshness of respiration. Instead of the two component sounds, inspiration and expira-

tion, fading imperceptibly into one another, they are more or less distinctly separate, the more so as the more typical bronchial type of breathing is acquired. Deficiency of elasticity is the cause to which the division is usually ascribed; it is, however, a significant feature of glottic breathing.

Bronchial respiration is most typically heard over simple lung-consolidation, as pneumonia at the base or apex. Skoda well describes the sound as acoustically identical with that produced by placing the mouth in the position to pronounce the guttural *ch* (as in *choir* or *christian*), and drawing the breath to and fro. The inspiratory and expiratory sounds are about equal in length, nearly identical in pitch, and distinctly divided from one another. The sound varies in intensity and definition, from the most intense *tubular* or *tracheal* breath-sound to the lower-pitched and more diffuse *blowing respiration* (*diffused bronchial breathing*). Besides hepatisation of lung, this form of respiratory sound may be produced by other condensations of lung, for example, from pressure or by tumours extending from the neighbourhood of a large bronchus to the surface, such as enlarged bronchial glands, mediastinal growths, and aneurysmal tumours. The more diffused blowing sounds are due to less complete consolidation. It is essential that the bronchi be patent, in order that bronchial respiration may be heard; thus, in cases of cancerous growth invading a lung from its root and occluding the bronchi, no respiration is audible. As regards mechanism, however, it can scarcely be maintained that the sound is produced by the passage to and fro of the air in the bronchi of the consolidated lung; for (1) at the period when bronchial breathing is most distinct the lung is immovably fixed by exudation; (2) the play of the chest-wall on the affected side is almost or quite restrained; (3) the air-cells being occupied, there is no reason why air-currents should penetrate the bronchi. Hence it would seem that bronchial respiration is but the glottic breath-sound reverberating through the bronchial tubes, and well conducted to the surface. A remarkable experiment of Bondet and Chauveau strikingly confirms this view. In a horse with hepatisation of the base of one lung and bronchial breath-sound over the part affected, the trachea was incised below the glottis and the wound held widely open; the bronchial breathing immediately disappeared, all respiratory sounds ceasing over this portion of lung, while elsewhere the vesicular breath-sound was unimpaired. A musical reed was now inserted into the wound, and the musical sounds were well conducted over the consolidation, but little audible over the healthy portion of lung.

Cavernous respiration is a breath-sound in which the inspiration and expiration have both a hollow blowing quality. It is to the expiration that the hollow wavering quality characteristic of this breath-sound is especially attached, and, as pointed out by Reginald Thompson, the expiratory sound is lower in pitch than the inspiratory. Cavernous breathing signifies a pulmonary cavity usually phthisical—(1) exceeding in size an unshelled walnut; (2) either empty or at least partially so; and (3) communicating with one or more patent bronchial tubes. Softening of tubercle or caseous pneumonia, pulmonary abscess, or bronchial dilatation of sufficient size, are the most common causes of cavity in the lung. This abnormal sound is formed by—(a) the

passage to and fro of air into a cavity with the respiratory movements; (b) the conduction and modified reinforcement of the glottic respiratory sound within a cavity.

Amphoric breath-sound is a variety of cavernous respiration having the same characters, but on an exaggerated scale: that is, not necessarily exaggerated as regards loudness, but having all the qualities—blowing character and hollowness—intensified. This sound is heard over a large superficial cavity, either in the lung, or in the pleura freely communicating with the lung. Its mechanism is identical with that of cavernous respiration, only that the size of the cavity is large.

(c) **ADVENTITIOUS AUSCULTATORY SIGNS.**—A *r le* or *rhonchus* is a sound produced by impediment to the entry or escape of air within the lungs or bronchial tubes. The impediment may be from narrowing, or secretion within the tubes; from secretion within the alveoli; or from destructive softening or oedema of the lung-tissue. The *r les* that may be audible over the chest are—*sonorous*, *sibilant*, *crepitant*, *sub-crepitant*, *mucous*, *dry crackling*, *moist crackling*, and *cavernous*.

Sonorous and sibilant r les or rhonchi are noises of a snoring or whistling kind, which are produced in the air-passages. They are audible with both inspiration and expiration (or with either), and are for the most part transitory sounds, being temporarily or permanently removed by cough, or in other cases by the relief of the spasm which has occasioned them. They obscure or altogether mask the normal respiratory sounds. Any narrowing of an air-tube will give rise to rhonchus or sibilus according to the degree of narrowing and the size of the tube. Thus, if the larger tubes be affected, and the narrowing not great, the coarser sound is produced. If, on the other hand, the finer tubes be partially occluded, or a larger tube be greatly narrowed, the finer sibilus is caused. Rhonchi are audible throughout the territory of the tubes affected. Thus, if a main bronchus be compressed or narrowed, the sound so occasioned will be heard throughout the lung on that side. Throat-sibilus in croup is conducted all over the chest.

The precise causes of rhonchi are—(1) narrowing of a bronchus from external pressure (uncommon); (2) narrowing from local cicatricial thickening and contraction of the fibrous coat of the tube (uncommon); (3) mucous collections in the tubes giving rise to imperfect plugs which vibrate, causing the musical sounds (very common); and (4) spasmodic contraction of the medium-sized tubes (sibilus in asthma).

Rhonchi signify—(1) bronchial catarrh, or bronchitis, local or general, as the case may be, affecting the larger and medium-sized tubes; (2) tumours pressing upon the trachea or one of the main bronchi; (3) numerous minute bronchial obstructions occasioned by pulmonary miliary tuberculosis; or (4) asthma.

Stridor is a variety of rhonchus, due most generally to pressure of a malignant or aneurysmal tumour upon a main bronchus, and heard chiefly over the corresponding side. It is a coarse, vibrating sound, which, however, the trained ear can readily detect to be of distant origin. Paralysis of the vocal cords will, in some cases, lead to stridor.

Crepitant r le, or *fine, dry crepitation*, is a minute, dry, crackling sound, in which the crackles are infinitely small and even, and occupy chiefly

the latter part of inspiration. The sound has been compared to the crackling of salt upon the fire, or that produced by rubbing a pinch of hair between the fingers close to the ear. Probably the exact mechanism of the sound is the abrupt separation of alveolar surfaces, collapsed by inflammatory or other oedema. But there are difficulties in the way of any present explanation of the sound. There are at least four conditions which will give rise to identically the same sound, as far as the ear can appreciate it—namely (1) incipient pneumonic consolidation (inflamed oedema stage); (2) oedema of the lungs when not excessive, as in certain stages of kidney-disease, in obstructive heart-disease, &c.; (3) mere collapse of lung from disease, crepitant *râle* being often temporarily heard from this cause at the extreme posterior bases, to disappear after a few deep inspirations; and (4) certain cases of oedema of the pleura dependent upon old lung-disease. The fine crepitation of pneumonia is peculiar only in being associated with commencing tubular breath-sound, the consolidation associated with which gives an increased intensity and definition to the crepitant *râle*. When associated with acute febrile symptoms, fine crepitation indicates the congestive stage of acute pneumonia. If seated about the base, the pneumonia is most commonly of the typical croupous or exudative variety. If at the apex, or in patches, the disease may be incipient catarrhal or embolic (pyemic) pneumonia.

Sub-crepitant or muco-crepitant râle is a fine bubbling *râle*, of sharp definition, and well-conducted to the ear, audible principally during inspiration, but in less degree also with expiration. This *râle* is produced in the minute bronchioles and alveoli, by the penetration of air through a thin liquid. A certain amount of lung-condensation is necessary to give sharpness of definition to the sound. Sub-crepitant *râle* is most typically heard in the resolution stage of pneumonia. In the second (secretion) stage of broncho-pneumonia it is also heard. There are many *râle* sounds intermediate between true dry crepitation and the sub-crepitant *râle*, which are fairly described by the general term crepitant *râle*, fine or coarse, according to their size. Many degrees of fineness or coarseness may be distinguished in different parts of the same lung in some pneumonic forms of phthisis, and it will be generally found in any such cases that the *râles* increase in coarseness as we ascend from below upwards.

Dry crackle is the term used to describe a *râle* consisting of three or four distinct small crackles heard during inspiration. The crackles are dry in character, and sharply defined. The inspiratory breath-sound attending this rhonchus is usually feeble and harsh, the expiration harsh and prolonged, but unattended by any *râle*, unless it be some sibilus. Dry crackling most commonly signifies commencing softening of 'tubercular' deposits, and the sound may be most frequently recognised in the sub-clavicular region, where this condition is most often found uncomplicated by conditions depending upon other stages of the disease.

Moist crackle, or humid clicking râle, consists of a few crackles, heard during the latter part of inspiration and the commencement of expiration, sharply defined, sometimes metallic in quality. The crackles vary in size and in the degree of liquidness, as must be the case from the mechanism

by which they are produced. For this *râle* is significant of liquefaction of tubercular or caseous pneumonic nodules in communication with bronchial tubes; and as such adjacent softenings coalesce and increase in size, the crackles become larger, until they develop into the *gurgling* or *cavernous râle*. The moist crackle may be associated with other *râles*, since a softening caseous nodule is often surrounded by congested pulmonary tissue or pneumonia, giving rise to fine crepitant or sub-crepitant sounds. As a rule, the breath-sound is more or less masked by the crackling *râle*.

Cavernous and gurgling râles are but larger and more liquid *râles*, produced in a cavity or cavities of moderate dimensions.

Metallic tinkling râle requires for its development a large empty cavity in which it may be produced—(1) by the bursting of one or more air-bubbles through viscid contents; (2) by the impingement of a drop of secretion against the cavern-wall; or (3) by a bubbling *râle* produced in a bronchus near the cavity, and freely communicating with it. In either case the large empty cavity, necessarily near the surface, resonates and re-echoes the sounds, and gives them their peculiar metallic quality, which has been likened to that produced by a pin dropping into a large empty bottle. Metallic tinkling is by no means solely significant of pleuritic cavity, as was supposed by Laennec; it may be most typically heard over a large dense-walled empty pulmonary cavern.

Metallic echo is sometimes confounded with metallic tinkle, with which it is often associated, and, indeed, of which it may be said to form a part. It is really not a *râle* at all, however, but an echo in a large cavity, produced—(1) by air-vibrations caused by cough; (2) by vibrations on the surface of fluid with a large air-space above; or (3) by vocal vibrations reaching through the cavity after true voice-sound has died away.

Hippocratic succussion-sound is the splashing sound heard in a pleura containing both air and fluid, on shaking the patient somewhat vigorously, while the ear is applied to the chest-surface.

Cough-sounds.—A *cavernous splash-sound* may frequently be heard on listening over a cavity, and causing the patient to cough, the forcible entry of air into the cavity in itself largely contributing to the sound, and setting up gurgling and splashing *râles* by the disturbance of contained fluids.

Cough-sounds require no explanation, but they should be invariably tested in chest-examination. Crepitant sounds are often developed after a cough, which are not to be heard either on ordinary or deep inspiration without it. *Cavities* which are not in free communication with bronchial tubes may yield no characteristic breath-sounds; but the forcible propulsion of air into them at the moment of chest-compression with closed glottis elicits at once a characteristic localised succussion-sound, attended by more or less coarse gurgling *râle*.

Voice-sounds.—In the ordinary healthy spongy condition of lung, the voice-sounds are heard but distantly and imperfectly, save in certain parts of the chest in the neighbourhood of the trachea and its bifurcation—that is, in the upper sternal and the upper interscapular regions, where the sounds are better conducted.

Bronchophony.—At any portion of the chest where there is consolidation of lung, in association with patent air-tubes, the voice-sound is heard

loudly, as though produced near or close, under the stethoscope. Although loudly heard, the sound appears to pass away from under the stethoscope. Any solid medium of conduction between a large bronchus and the stethoscope will give rise to bronchophony, whether by super-position, or by the portion of bronchial tree concerned being embedded in solid lung, as in lobar pneumonia, of which the sound is most typical. If, however, between the conducting medium and the larynx the bronchial channel be occluded, bronchophony is no longer heard, the voice-sounds being enfeebled or annulled.

Pectoriloquy.—If, on the other hand, a cavity be present beneath the spot auscultated, and in free communication with a bronchus, the voice-sound appears to be concentrated at the end of the stethoscope, and to pass *through* the instrument direct to the ear, with exaggerated and even painful distinctness. It is rather the *noise* of the voice that we hear in bronchophony, but in pectoriloquy the sounds are most distinctly *articulated*. This distinction is even better appreciated by listening to a whisper, which under bronchophonic conditions is merely a conducted hissing sound, while in pectoriloquy each syllable penetrates distinctly to the ear. Pectoriloquy may, however, be clearly, although not exactly, imitated by consolidated lung in the neighbourhood of a large bronchus. Hence the diagnosis of a cavity near the root of the lung requires much caution.

Egophony.—Egophony is a form of modified bronchophony in which the voice-sound, conducted through condensed lung, has further to penetrate a thin layer of fluid in which the coarser vibrations are lost, a certain quavering nasal quality being given to the sound that reaches the ear. It is significant of effusion into the pleura. The sound is only to be heard near the upper limits of the effusion, where the layer of fluid is thin.

With regard to the mechanism of these three sounds—bronchophony, pectoriloquy, and egophony—there can be no dispute about their being glottic sounds. In *bronchophony* they are conducted through subdividing tubes of increasing fineness enveloped in solid tissue; hence the sounds, although loudly heard, are not well defined, being largely converted into the coarser vibrations perceptible to palpation as *fremitus*. In *pectoriloquy*, on the other hand, the glottic sounds are conducted through tubes which, after one or two divisions, terminate in a resonating cavity; hence the vocal vibrations are concentrated and conducted with intensity to the ear as through a speaking-tube. Finally, in *egophony* one may suppose the bleating character of the sound to be due to secondary, and to a certain extent disturbing vibrations in the fluid medium through which the sounds are conveyed. In *egophony* one may commonly note a lisp or whisper-sound in addition to the voice-sound, and better conducted than the voice-sound. And it has been affirmed by Bacelli that in cases of *serous* effusion into the pleura the whisper is heard well conducted with distinct articulation—*pectoriloquie aphonique*—through the thickness of the fluid, whereas in *purulent* effusion such whisper is not conducted. This statement will be found to apply, however, only in certain cases. The whisper may sometimes be heard well conducted through purulent fluid.

The voice-sounds are *weakened* or wholly *extin-*

guished by conditions which: (*a*) shut off the main bronchi from the part auscultated, as in malignant growths invading the bronchus at the root of the lung; (*b*) separate the lung-surface from the thoracic wall, as in pleuritic effusions, œdematous thickening of pleura, &c. Here, however, we must make exception in certain cases, in which pectoriloquie aphonique is heard. In (*c*) rarefaction of the lung by emphysema the voice-sound is enfeebled; and in (*d*) pneumothorax it is either much enfeebled or annulled. In cases of pneumothorax, however, a faint *metallic echo* may often be heard with, or rather after, the voice-sound. This echo has probably a precisely analogous mechanism to ægophony, save that the medium of secondary conduction is air instead of fluid, and hence the conduction is less distinct.

Autophony.—On listening over a superficial cavity with condensed lung-tissue around, the voice of the auscultator—for example, when requesting the patient to cough or to speak—will be noticed by himself to be intensified. The term *autophony* is applied to this increased resonance, which is a sign of little clinical value.

Pleural sounds.—The sounds originating in diseased conditions of pleura are commonly included under the general term ‘friction-sounds’—a term, however, very inadequate to describe the varieties.

The *pleuritic rub* or *dry friction* is a wavy or uneven rubbing sound heard close under the ear with both inspiration and expiration, but chiefly with the former, unmoved by cough, and usually attended by pleuritic pain. We may often fail to obtain this sound, through the patient involuntarily restraining the movement of the affected side on account of the pain. A deep inspiration must, therefore, be always called for. In well-marked cases the friction is very loud and leathery, and may be perceptible to the hand applied—*friction-fremitus*.

Pleural creaking is a sound that may be sometimes distinguished over a portion of the chest, when the pleuræ are densely thickened and adherent.

Moist or spongy friction is most difficult to distinguish from fine moist crepitation. It is heard almost entirely at the end of deep inspiration, and closely resembles the crepitation of a moist sponge. The sound is due to the pleura being adherent by moist, recent lymph, as in the early stage of adhesive pleurisy in pleuro-pneumonia.

In cases of œdema of the pleura a fine crepitating inspiratory sound or *pleural crepitus* may be heard, which it is impossible to distinguish from a pulmonary sound. The diagnosis must rest upon the very superficial character of the sound, and its being unchanged by cough; also upon its being associated with deficient breathing without tubular quality, and with lessened vocal fremitus. It is an inspiratory not an expiratory sound, being engendered by the pulling out of the spongy œdematous tissue during inspiration.

(5) *Radiography.*—Examination of the chest by radiography has of late years been increasingly made use of. See RÖNTGEN RAYS.

4. *Circulatory System, Physical Examination of.*—The condition of the heart and circulation may be investigated with great exactness, chiefly by palpation, percussion, and auscultation.

PULSE.—The pulse gives us very important information respecting the state of the circulation.

For a full description of the pulse and its different characters in disease, *see* PULSE, THE; and SPHYGMOGRAPH.

HEART.—(a) **Inspection.**—In health and during quietude the cardiac impulse is barely perceptible. Under excitement, however, a throbbing impulse may be noticed over the præcordia and left epigastrium. In cases of great hypertrophy and dilatation of the heart, especially in children, the præcordial region may be obviously bulged. The impulse of the heart may be observed to be diffused over an increased area, between the nipple-line and sternum, in cases of hypertrophy and dilatation. In cases of dilated hypertrophy of the right ventricle, or in displacement downwards of the heart in emphysema, the impulse is very perceptible at the epigastrium to the left of the ensiform cartilage. A diffused undulating impulse may be observed in some cases of pericardial effusion and in adherent pericardium. The heart is often uncovered, and its impulse revealed on one side or the other by retraction of the lung in contractile or wasting pulmonary diseases.

(b) **Palpation.**—The position of the heart's apex should first be ascertained; and the area, force, and rhythm of the cardiac pulsations, and the presence or absence of thrill or other adventitious palpation-signs, should next be noted.

Normally the heart, enclosed in its own pericardial sac, is situated in the anterior and central part of the thoracic cavity, immediately above the diaphragm. Its position may be roughly defined as within the area bounded above by a line drawn across the sternum at the level of the lower border of the second cartilages; on the left by a vertical line passing just within the left nipple; and on the right by a similar line drawn at one-third of the distance between the border of the sternum and the right nipple-line. A slanting line from the base of the ensiform cartilage to the upper border of the sixth rib in the left nipple-line defines the lower border of the heart. Behind this area the heart lies obliquely, its base directed upwards to the right and backwards, its apex to the left downwards and forwards. The organ, moreover, is so placed that the right auricle and ventricle occupy nearly the whole anterior surface; the left auricle and ventricle the posterior and left surface.

The apex of the heart in the adult impinges in the fifth interspace, one inch within the left nipple-line. The aortic and pulmonary valves correspond with the upper border of the third left cartilage at its junction with the sternum, the aortic being on the right of and a little lower than the pulmonary. A line drawn from the middle of the third left cartilage as it joins the sternum, to the upper border of the fifth right cartilage at the sternal margin, would correspond with the mitral valve superficially and above, the tricuspid more deeply and below.

An *altered* position of the apex-beat may arise from congenital displacement of the organ, for example, from transposition of viscera. It may arise from enlargement of the organ by hypertrophy or dilatation, affecting its right or left cavities; or from displacement of the organ, for instance, *downwards*, by emphysema, aneurysm, or tumour; *aside*, by pleuritic effusion, malignant disease, or contraction of lung; *upwards*, by abdominal distension, disease in the abdomen, or contraction of lung.

In continuance of palpation, the condition of the

arteries and veins at the root of the neck must be observed: whether the arteries unduly pulsate, or the veins on one side or both remain full or pulsate.

(c) **Percussion.**—The præcordial dullness may be enlarged by retraction of the margin of one or both lungs; by effusion of fluid into the pericardium; or by enlargement of the heart itself, either general or restricted to one or more of its divisions. The cardiac dullness may be diminished or obscured by enlargement of the lungs enveloping it, or by gas in the pericardial sac.

(d) **Auscultation.**—By the simultaneous contraction of the ventricles, the closure of the mitral and tricuspid valves, and the impact of the apex of the left ventricle against the ribs, a single sound is produced, the first sound of the heart. The sudden tense closure of the mitral valve is the principal cause of this sound. The first sound is closely followed by the second sound, which is more tapping in quality, and corresponds with the closure of the aortic and pulmonary valves. Then comes the diastolic pause, which may be said to equal in duration that of the two sounds. The first sound is most loudly heard at the apex, the second at the base of the heart.

The sounds of the heart are subject to considerable variations under varied general conditions of health and disease.

(1) In general debility, anæmia, and wasting diseases, the tendency is for the first and second sounds to approximate to each other in character. As the ventricular wall becomes atrophic or ill-nourished the first sound becomes more purely valvular, and at the same time more feeble and tapping, approaching thus in character to the second sound. Sometimes in cases of anæmia the first sound is peculiarly ringing and hollow in character.

(2) In chronic Bright's disease, with thickened vessels and hypertrophied ventricles, the first sound is peculiarly muffled and indistinct, compared with the recognisable force of the beat.

(3) The rhythm of the heart's sounds may be greatly changed: (a) The first or second, or both first and second sounds, may be reduplicated. This may occur as a temporary phenomenon in apparent health, but it is more commonly traceable to increased resistance either in the pulmonary or systemic circulation. (b) Excessive rapidity of action. (c) Irregularity in time and force of beats. (d) Intermittent action. These several phenomena may be significant of disease of the heart itself; or, as is frequently the case, they may be due to functional disturbance through the nervous apparatus, from dyspepsia, or from excessive smoking, tea-drinking, or venery.

A *murmur* or *bruit* is an abnormal sound, invariably of a blowing character, which may more or less replace or obscure the normal heart's sound. Either of the sounds of the heart may be replaced or attended by a murmur; and in auscultation, with regard to prognosis, it is much more important to note whether a murmur wholly or only partially replaces the normal sound—that is, whether the function of the valve be wholly or only partially disabled—than to be guided by mere loudness of *bruit*. The first sound at the apex may be preceded, or, very rarely, succeeded by a murmur. For a description of these murmurs, and of morbid pericardial sounds, *see* HEART, VALVES AND ORIFICES OF, Diseases of; HEART, Functional Disorders of; and PERICARDIUM, Diseases of.

5. Mediastinum, Physical Examination of.—Having examined the thorax with regard specially to the great organs, the lungs and heart, contained within it, the mediastinal region should next be explored, both anteriorly and posteriorly.

NORMAL SIGNS.—The anterior mediastinal region, clinically speaking, corresponds with those portions of the sternum not underlain by lung, namely, the manubrium and the left half of the body, extending from the fourth cartilage downwards.

The lungs normally approximate beneath the upper portion of the second part of the sternum; from that point to the lower border of the fourth cartilages hiding the subjacent parts. In the triangular space behind the *upper* sternum, with its apex at the lower border of the manubrium and its base at the episternal notch, lie the inferior extremity of the trachea, covered by the left innominate vein, the summit of the arch of the aorta, and a prolongation of the pericardial sac, with connective tissue, and a few small lymphatic glands. The summit of the arch of the aorta corresponds with the level of the upper border of the second rib-cartilage.

Imperfect percussion-dulness and modified bronchial respiration, with weakly conducted heart-sounds, are usually present over this region. On deeply depressing the finger behind the sternum in the episternal notch, a slight pulsation, communicated from the aorta—which vessel, however, the fingers cannot reach—is felt. The lower region of mediastinal dulness, that is, below the level of the fourth cartilage, is continuous on the left of the sternum with the heart's dulness, and, indeed, corresponds with the præcordial region.

MORBID SIGNS.—The *upper* mediastinal dulness may be replaced by resonance—(1) from enlargement of the lungs in emphysema; (2) in cases of contraction of the upper part of one lung, enlargement of the opposite lung wholly occupying the lateral region, and effacing the normal mediastinal dulness. The limits of normal mediastinal dulness may be *extended*, from displacement of the anterior margin of the lungs—(1) by dilatation or aneurysm of the aorta; (2) by mediastinal abscess; (3) by simple enlargement of the mediastinal or thymus glands; or (4) by morbid growth—cancer, or lymphoma. (For the diagnosis between these several conditions, see AORTA, Diseases of; and MEDIASTINUM, Diseases of.) It must be borne in mind that considerably increased dulness, and even prominence, may be due to intrinsic disease of the sternal bone, or to thickening from periostitis.

Alterations in the boundaries of the *lower* region of mediastinal dulness are most often due to enlargement of the heart, or dilated pericardium. Aneurysm of the aorta or the heart, or a growth extending forwards, between the heart and the lung, from the posterior mediastinum, are the other causes of increased inferior mediastinal dulness.

Posteriorly there is no inter-pulmonary space apparent save that occupied by the spinal column. But in disease, and especially in tumour, whether aneurysmal or of the nature of morbid growth affecting the root of the lungs (see BRONCHIAL GLANDS, Diseases of), the posterior mediastinal dulness involves the right or left interscapular region, as the case may be.

The bifurcation of the trachea corresponds with the body of the fourth dorsal vertebra. The

descending portion of the arch of the aorta corresponds with the left side of the third dorsal vertebra.

6. Abdomen, Physical Examination of.—The abdomen is that portion of the body included between the diaphragm above and the brim of the true pelvis below; and is usually divided, for convenience of clinical reference, into regions. Two horizontal lines drawn at the level of the ninth ribs, and the highest points of the crests of the ilia respectively, and intersected by vertical lines drawn from the eighth rib on each side down to the middle of Poupart's ligament, divide the abdomen into nine regions, namely, epigastric, umbilical, and hypogastric in the middle; and hypochondriac, lumbar, and iliac on each side, from above downwards.

(a) **Inspection.**—When examining a case of abdominal disease the position naturally assumed by the patient should be noticed: whether it be indifferent; or dorsal with the knees drawn up—a position very characteristic of peritonitis; or lateral, with the thighs flexed and the body bent, as in renal or hepatic colic. Sometimes in cases of colic, especially lead-colic, the patient lies on his belly with the arms compressing the part. Frequent changes of posture are also characteristic of colic rather than of peritoneal inflammation. The general size, shape, tenseness, flaccidity, or retraction of the abdomen will be next observed. Any alteration from perfect symmetry will be noted, with the region of any swelling. The superficial veins of the abdomen may be enlarged, the internal mammary from above meeting the superficial and deep epigastrics, to secure collateral circulation between the superior and inferior cavae, when either is from any cause compressed or occluded.

(b) **Palpation.**—On placing the hand over the abdomen for the purpose of palpation, the rigidity or otherwise of the muscles, especially of the recti, will be noticed; and the observer will be careful to note whether the muscle becomes contracted during manipulation, or was from the first unduly tense. The muscles of the abdominal walls are rigid, as a rule, in all inflammatory conditions of the peritoneum. In local peritonitis, and over special organs or tissues which are painful, the muscles are also tense; thus it is not uncommon to find one rectus notably more rigid than its fellow.

In order properly to examine the abdomen by palpation, it is necessary to place the patient flat on his back, on a slightly inclined plane, with a round pillow placed under the head, so as to flex the chin upon the sternum. The thighs should be similarly flexed upon the pelvis by means of a second incline, on which the legs should rest, or by placing one pillow beneath the thighs and two pillows beneath the legs. In this manner the muscles of the abdomen will be rendered as lax as possible. The patient should further be held in conversation, or told to breathe deeply, but without effort, in order that he may not keep his diaphragm fixed. It is often a good plan, when other efforts fail to prevent the patient from keeping his diaphragm fixed, to make him go on counting 'one,' 'two,' 'three,' up to as high a number as he can possibly reach without drawing breath. In this manner we get the diaphragm thoroughly relaxed; and by keeping the hand on the abdomen,

deep palpation can be effected at any period of the counting most suitable for the purpose. The observer should be comfortably placed at about the same level as his patient. The whole hand, previously warmed, should be evenly applied to the surface, and the fingers then depressed in different directions as the hand is smoothly conveyed to different regions. If the patient be poked about with the ends of the fingers by the physician stooping over him, he is either tickled or hurt, his muscles contract, and proper examination is impossible.

It is sometimes useful to make the patient change his position first to one side, then to the other. This method is particularly to be adopted in examining tumours which are movable, such as floating kidneys, some uterine tumours, and aneurysmal sacs. In the case of tumours lying over the aorta, it may be impossible, without adopting this plan, to be sure whether the pulsation felt over them is communicated or intrinsic.

It is sometimes doubtful whether a tumour is situated within the rectus muscle, or in the abdomen beneath it. By keeping the hand over the tumour and making the patient raise himself half to the sitting posture, so as to cause the recti to start forward in contraction, this point can be cleared up.

The temperature of the surface of the abdomen to the hand applied, and to the surface thermometer, may be distinctly raised above that of the general surface in peritonitis.

It is often difficult to estimate the true degree and nature of pain in the abdomen caused by pressure. In hyperæsthesia of the surface the slightest pressure causes suffering; while deep, even palpation gives little inconvenience. If the surface be pinched up the pain is acute. Pain in the abdominal muscles is less acute, and is intensified by bringing these into action. The pain of peritonitis is superficial in so far as it is commonly associated with hyperæsthesia of surface; but gentle, steady pressure is acutely painful, and deep palpation intolerable. The pain of peritonitis may, with the disease, be general or local. There is often some difficulty in differentiating the pain of localised hyperæsthesia—hysterical pains as they are called—from those of inflammatory origin or from tenderness of organs. By holding the patient persistently in conversation respecting symptoms associated altogether with another part, as minute inquiries about headache, cough, &c., and thus keeping off attention while the hand is steadily compressing the supposed painful parts, all doubts can be removed. In neuralgic and hysterical pains simulating peritonitis, the tenderness extends beyond the confines of the peritoneum.

Abdominal organs may be tender to palpation.

Fluctuation.—Fluctuation is an important sign of the presence of fluid in the abdomen, whether the fluid be free in the peritoneum or enclosed in a sac. It may be obtained by placing one hand lightly on the abdomen, while the fingers of the other hand smartly tap over another part, when a fluid wave will be felt to impinge against the applied hand. In certain tense conditions of the abdomen, a deceptive sense of fluctuation may be obtained from the vibrations of the abdominal walls. To prevent this fallacy the hand of a bystander should be applied edgewise on the abdomen, midway between the two hands of the observer, so as to check superficial vibrations.

Hydatid fremitus.—This is a kind of tense fluctuation appreciable by the pleximeter-finger on sharp percussion over certain cysts, more particularly hydatid cysts. See ENTOZOA, p. 461.

(c) and (d) **Percussion and Auscultation.**—Percussion and auscultation of the abdomen are adopted in accordance with the methods already described. The distribution of dullness and resonance, varying or not with the position of the patient, affords important evidence respecting fluid collections, whether peritoneal or encysted. See ABDOMEN, Diseases of; and ASCITES.

By auscultation friction-sounds may be heard over the seat of peritonitis; vascular, aneurysmal, and placental bruits; or the sounds of the foetal heart may be detected.

PHYSICAL EXAMINATION OF THE LIVER.—In the right mammary line the liver underlies the region from the fifth rib to the costal margin: in the median line from the base of the xiphoid cartilage to an inch and a half below that level. The left extremity of the liver lies just within and behind the apex of the heart. It may, then, be roughly said that a horizontal line drawn from the base of the xiphoid cartilage to the right side of the chest and to the apex of the heart, and a second line slanting from within the apex-beat to the right costal margin in the nipple-line, would mark the site of the liver. It has already been observed how this surface is partially covered above by lung. In health the margin of the liver becomes lost to palpation beneath the cartilages in the right nipple-line. Its upper margin may be defined, as already shown, by deep percussion, its lower margin by very light percussion.

Displacements.—The liver may be lowered in position by certain thoracic conditions, such as emphysema, fluid in the pleura or pericardium, thoracic tumours, or compression by tight-lacing. When the liver is thus lowered, it is somewhat anteverted; and in lax conditions of the abdomen its lower margin may be covered by a coil of intestine, thus requiring somewhat deep palpation in tracing it.

The liver may be raised by contractile thoracic diseases, especially on the right side, so that its margin recedes considerably within the costal margin. In cases of doubt as to whether extension of dullness upwards be liver or lung consolidation, the observer must notice whether the level be shifted by respiratory movements. When the abdomen is distended from any cause, the liver is pushed upwards; and in this case, and also in many instances where the liver is drawn upwards, it becomes also tilted somewhat backwards, so that but little more than the margin presents anteriorly. In this condition there may be but little, if any, liver-dullness discoverable anteriorly, and it may erroneously be concluded that the liver is much diminished in size. In these cases, however, the posterior dullness of the liver is increased in the right lower thoracic region. In cases of enlargement of the liver, therefore, the upper margin must be accurately defined, to see if there be extension upwards, and whether that extension be even or uneven. The lower margin must be traced by palpation; the mobility of the organ with respiration estimated; its hardness, softness, sharpness, evenness, or distortion ascertained; and whether it be free or connected with other parts—for example, the spleen, or an abdominal tumour.

The lower margin of the liver, when the organ is enlarged or depressed, very frequently cannot be defined by percussion, being overlapped by intestines. For instance, in cases of lax abdominal arietes, with moderate fluid effusion in the peritoneum, the intestines float up and press between the margin of the liver and the surface. Greatly diminished or absent liver-dullness may be observed in cases of perforation of the bowel, and in cases of paralytic distension of the bowel in acute enteritis. In other cases the front surface may be indurated, and the margin thus incurvated to certain extent and covered by intestines. The surface must be felt—whether smooth, or rough, or nodulated. The *consistence* must be estimated by palpation—whether hard or soft, or fluctuating at any part. See LIVER, Enlargements of.

The gall-bladder cannot be felt unless it be distended, when it presents as a rounded tumour attached to the margin of the liver in the right nipple-line. See GALL-BLADDER AND GALL-DUCTS, Diseases of.

PHYSICAL EXAMINATION OF THE SPLEEN.—Formally, splenic dullness may be ascertained on light percussion in an area on the left side extending from the ninth to the eleventh ribs, and between the mid-axillary and mid-scapular lines. The shape of the splenic dullness is oval in the slant of the ribs.

In moderate *enlargement* the splenic dullness is increased in all directions; and on placing the hand deeply in the left flank, close under the ribs, the organ may be felt to descend upon it during inspiration. As the organ still enlarges it comes forwards and downwards, raising the apex-beat of the heart, occupying the region in front of the scapular line and below the level of the apex-beat, and projecting downwards beneath the costal margin into the abdomen. As the organ still further enlarges, the anterior margin curves forwards, forming nearly a right angle with the costal margin. It is usually sharply defined, and may extend forwards to the median line, and downwards to the pelvis. The posterior margin of the enlarged spleen is also, in such cases of great enlargement, to be felt thick and rounded immediately in front of the quadratus muscle. An enlarged spleen extending into the abdomen is superficial in its entire area; its anterior and posterior borders are well defined; and it can usually be moved between the two hands forwards and backwards. The notch may be commonly felt. The surface may be quite even or nodulated. Sometimes on auscultation a bruit may be audible over an enlarged spleen. Friction-sound may also sometimes be heard. Certain alterations in the constitution of the blood and in the temperature of the body are intimately associated with diseases of the spleen, and the examination of these conditions forms an important item in their diagnosis.

Diminution in the size of the spleen cannot be accurately estimated, and is of little clinical moment.

PHYSICAL EXAMINATION OF THE KIDNEYS.—The kidneys, when of normal dimensions, cannot, as a rule, be felt, especially in fat people, or when the abdomen is enlarged. They lie one on either side near the spinal column, between the level of the spinous process of the eleventh dorsal and of the second lumbar vertebræ, and in the mid-line between these spinous processes and the outer

margin of the flank. Embedded in fat, they rest on the lumbar muscles. The right kidney is overlain in part by the liver, colon, and intestines; the left by the stomach, colon, and intestines; the left kidney is half an inch higher than the right.

In order to feel for the kidney, the patient should be placed in the position for abdominal examination. The observer, standing on the side opposite to that of the kidney under examination, then places one hand along the mid-flank behind, immediately below the last rib; the other hand should rest upon the corresponding part of the abdomen in front, firmly depressing and manipulating deeply, so as to bring the site of the kidney between the phalangeal portions of the two hands. At the same time the patient should be made to inspire and expire deeply; and it is during the stage of moderately deep expiration that the organ will usually be felt.

Tenderness of the kidney, if present, may thus be estimated with certainty. Undue rigidity of the muscles on one side may be observed.

If the kidney be *uniformly enlarged* it simply extends downwards, and comes more readily under observation. In great enlargement of the kidney, as in cancerous tumour, or of its pelvis, as in pyelitis, the organ forms a tumour occupying the flank, and coming forwards from behind the colon towards the front of the abdomen. Such a tumour is more or less pyramidal or rounded in form, with a distinct band of resonance corresponding with the superior flexure of the colon extending across it. The tumour may be solid or fluctuating, according to its nature. Renal tumours are most common on the left side. The pelvis of the kidney, except when considerably dilated, does not come under palpation. See KIDNEYS, Diseases of.

Movable kidneys.—The mobility of the kidneys varies much, from mere laxness to complete dislocation. See KIDNEYS, Malpositions of.

The examination of the urine forms the most important part of the physical diagnosis of kidney-diseases. See URINE, Morbid Conditions of.

PHYSICAL EXAMINATION OF THE PANCREAS.—In thin subjects with retracted abdomen, the head of the pancreas may be felt as a small, somewhat angular, tumour to the right of the median line, above the level of the umbilicus—in the region, in fact, of the pylorus, with thickening of which it may readily be confounded.

Cancerous enlargement of the pancreas extends forwards, in or near the middle line, above the umbilicus, presenting a tumour difficult to diagnose. See PANCREAS, Diseases of.

PHYSICAL EXAMINATION OF OTHER ABDOMINAL VISCERA.—Diseases of the *stomach* and *intestines* yield their proper signs, requiring no separate description here. Tumours of the *omentum* have to be distinguished, by the method of exclusion, from affections of the solid organs of the abdomen.

The *pelvic organs*—uterus, and ovaries, and bladder, under certain conditions of enlargement—present themselves for diagnosis as abdominal tumours.

When distended, the *bladder* gives rise to a pyramidal area of dullness extending in the median line from the pubes, broadening upwards, towards or even beyond the umbilicus. The tumour is firm but elastic, on palpation tender, and is at once removed by successful catheterisation.

In pregnancy at the fourth month the *uterus* becomes perceptible to deep palpation in the pelvic basin in the pubic region. At and beyond the fifth month a tumour of growing dimensions extends from out of the pelvis towards and beyond the umbilicus. Dulness on percussion extends from the pubes over the whole front of the tumour, while above and in the flanks a resonant intestinal note is obtained. The relations of dulness and resonance are not appreciably changed by position. The tumour is elastic, and in the advanced stages obscurely fluctuating. On deep palpation, an irregular resisting lobulated mass is to be felt; and, on keeping the hand steadily applied, undulating movements, or a distinct shock or jerking movement, may from time to time be felt. On applying the stethoscope with somewhat deep pressure, halfway between umbilicus and pubes and a little on one or other side, the rapid beat of the foetal heart may be distinctly heard, closely resembling the tick of a watch under the pillow. On bringing the stethoscope nearer the inguinal region (usually on the right side), the soft low-pitched placental *souffle*, synchronous with the maternal pulse, is to be heard. Enlargement of the uterus from other causes, especially fibroid and fibro-cystic growths, may lead to abdominal tumours, which are diagnosed by combined vaginal and abdominal examination. See UTERUS, Diseases of.

Ovarian tumours also present in the abdomen, extending upwards from one or other side of the pelvic region, and with a disposition as they extend to become central. They are most commonly cystic, and—especially in the later stages—sometimes present difficulties in diagnosis from pregnancy or peritoneal dropsy. See ABDOMEN, Examination of; ABDOMINAL ANEURYSM; AORTA, Diseases of; OVARIES, Diseases of, and other appropriate headings. R. DOUGLAS POWELL.

PHYSIOGNOMY (*φύσις*, nature; and *γνώμων*, an interpreter).

Physiognomy originally meant the interpretation of the nature of an individual by the light of the indications afforded by his countenance, conformation, and movements; but the term is frequently used for the indications themselves.

PHYSOMETRA (*φύσα*, air; and *μήτρα*, the womb).—A condition in which a collection of gas or air is formed in the uterus. See UTERUS, Diseases of.

PHYTOSIS (*φυτόν*, a plant).—A generic term for plant-formation, applicable to epiphytic, phytiform, or parasitic diseases of the skin, of which there may be enumerated the following species: *Phytosis* or *tinea tonsurans*; *phytosis* or *tinea circinata*; *phytosis favosa* or *favus*; and *phytosis versicolor*. See FAVUS; TINEA TONSURANS; and TINEA VERSICOLOR.

PIA MATER, Diseases of.—See MENINGES, CEREBRAL, Inflammation of.

PIETERMARITZBURG, in Natal.—See SOUTH AFRICA.

PIGEON-BREAST.—A deformity of the chest, in which the ribs are flattened laterally and the sternum thrust forward, so that the chest assumes somewhat the shape of the breast of a pigeon. See CHEST, Deformities of.

PIGMENTARY DISEASES OF THE SKIN.
GENERAL STATEMENTS.—Derangement of the pigmentation of the skin may be in the direction either of excess or of deficiency, and either congenital or acquired. These defects usually occur separately, but may be associated together in the same individual, as in leucoderma and melanoderma.

The special affections of *excessive* pigmentation are Nævus pigmentosus, Lentigo, and Chloasma.

Those of *deficiency* are Albinism and Leucoderma.

PATHOLOGY.—Pigmentation of the skin may be derived either from the colouring-matter of the blood, extravasated or exuded into the tissues, and is then only an exaggeration of a normal process; or from matters introduced into the blood, as bile-pigment producing jaundice; or from without, as arsenic or nitrate of silver; or, again, there may be a local infiltration of the skin, as in tattooing, chrysarobin-inunctions, &c. Except where due to local applications, the pigment is deposited chiefly in the rete mucosum, and almost entirely in the lower layers; it may also be seen in the upper layers of the corium in its passage from the vessels to the rete, to which it is conveyed probably by the branching connective-tissue cells. If these cells atrophy or are absent, the skin of the part is abnormally pale, but the untransferred pigment may be deposited in excess in the surrounding skin, as seen in leucoderma, which is associated with melanoderma. Little is known of the pathological causes of general pigmentation, but disturbed innervation doubtless plays the chief part. Thus the bronzing of Addison's disease is, in all probability, produced by chronic inflammation of the abdominal sympathetic; while the general pigmentation which occasionally follows severe chills, and the disturbed pigment-distribution of leucoderma, which has in many cases followed closely on sun-stroke, must also be regarded as nervous manifestations. Local pigmentation is in the majority of cases the direct consequence of acute or chronic hyperæmia, the blood or its colouring-matter escaping from the vessels and discolouring the tissues. This is especially common on the lower limbs, where the dependent position and frequency of varicose veins favour the exudation; the orange-coloured patches, for instance, seen on the leg being the direct consequence of capillary rupture.

Variations occur in colour, duration, and extent. In colour, pigmentation derived from the colouring-matter of the blood varies from a dull yellow or olive to brown or black. It is of a bright yellow or olive when due to bile, and a slate colour from nitrate of silver. In duration it may be temporary as a sequel of skin-eruption and in jaundice, or permanent as in argyria. In extent, pigmentation may be general as in Addison's disease, hypertrophic cirrhosis of the liver with glycosuria, and acanthosis nigricans (p. 12); in small spots, as in lentigo; or in large patches as in chloasma.

Absence of pigment may also be general as in albinism, or partial as in leucoderma.

ETIOLOGY.—Acquired pigmentation may be idiopathic or symptomatic.

Idiopathic pigmentations comprise those due to local causes, such as irritants, including blisters, sinapisms, friction, pressure, scratching, heat, Röntgen Rays, or exposure to the sun.

Symptomatic pigmentations may be (1) *general*,

as in Addison's disease, Graves's disease, leprosy, syphilis, malaria, cancer, tuberculosis, senile degeneration of the skin, argyria, and arsenical pigmentation. (2) *local*—(a) as a sequela of certain skin-eruptions, such as syphilides, lichen planus, and some forms of urticaria; (b) an accompanying manifestation of certain skin-lesions, such as scleroderma, diffuse or circumscribed, and fibroma; (c) due to parasitic affections, as in tinea cruris, tinea versicolor, erythrasma, and the Pinta disease of Mexico. In these, however, the discoloration is on the skin, or at most only in the superficial layers.

On the legs especially, if varicose veins are present, anything which produces hyperæmia is liable to stain the skin.

1. Nævus Pigmentosus.—**SYNON.**: Pigmentary Mole; *Nævus Pigmentaire*; *Ger. Fleckenmal*.

Moles are congenital structures which may be mere collections of pigment (*nævus spilus* or *congenital lentigo*); or the surface may be uneven and warty (*nævus verrucosus*); or the pigment may be in soft tumours of fat and loose connective tissue (*nævus lipomatodes*); or the tumours may be covered with coarse hair (*nævus pilosus*). They vary infinitely in colour, number, size, and distribution, sometimes occupying a nerve-area. Later in life they sometimes become the seat of melanotic cancer or other malignant neoplasm.

TREATMENT.—Small tumours or hairy moles are best destroyed by electrolysis, both hair and tumour being removed by this method, each hair being separately attacked (*see* DEPILATORIES). Larger tumours where not too large, and when their position renders it advisable, should be removed by the knife or caustics. In elderly people, any active change in a mole should be the signal for instant and complete removal.

2. Lentigo.—**SYNON.**: Freckles; *Ephelides*; *Fr. Lentigo*; *Ger. Sommersprosse*. *See* FRECKLES, p. 560.

Some authors restrict the term 'ephelides' to freckles caused by the sun, reserving the term 'lentigo' for the spots which come in covered parts and are independent of season. Lentigines may be a symptomatic condition, as in xeroderma pigmentosa, and as one manifestation of senile degeneration of the skin. On the other hand, they are occasionally congenital, and are then classed with moles.

3. Chloasma.—Chloasma is a generic term for both patchy and diffuse pigmentation, but is chiefly used for the patchy form.

The patches are well defined, sometimes roundish or oval, but they may be of any shape or size, and any shade from fawn to brown, bronze, or black. When diffuse, the colour is always of deeper hue in some regions than others, namely, the axillæ, nipples, umbilicus, pubes, and genitalia. The causation has been discussed in the general ætiology of pigmentation. The most important variety is *chloasma uterinum* of women who are pregnant, or who suffer from chronic uterine irritation. Its most common positions are the linea alba, nipples, and forehead; but it may come in any part of the face. On the forehead it forms one or more irregular patches, but in other parts it is less defined. It usually fades slowly after parturition, but is occasionally persistent. A similar pigmentation of the face has been observed in abdominal tuberculosis, cirrhosis of the liver, constipation, and cancer of the stomach.

DIAGNOSIS.—Chloasma may be distinguished from discolorations due to vegetable parasites by the latter being on rather than in the skin, except in the surface-layers. If there is any doubt, scrapings of the epidermis, soaked in liquor potassæ and placed under the microscope, will show the fungous elements if the discoloration is due to a parasite.

Accidental or malingering pigmentation can be washed off either with soap and water or with weak chlorinated-lime solution. The deposit in chromidrosis can be readily removed with spirit of chloroform or ether.

PROGNOSIS.—Time generally removes pigmentation due to previous eruptions and that due to pregnancy. The duration and continued activity of the cause are the chief factors in determining the likelihood of the removal of the discoloration.

TREATMENT.—When there are only a few scattered lentigines, superficial electrolysis is very satisfactory, but not when they are numerous and crowded, nor in chloasma. Sometimes discutients are effectual, but in a large proportion of cases their action is only temporary. From a half to 5 grains of corrosive sublimate, but not more than 2 grains to begin with, may be added to a fluid ounce of Almond-emulsion, and this painted on several times a day, or lint wet with the lotion kept on for some hours; and if well borne the stronger solution may be used. Other well-approved applications are acetic acid and sulphur made into a paste, and laid on; citric acid (ʒss to ʒj of water); pure carbolic acid very carefully applied with a brush; or salicylic acid made into a paste with glycerine. These should all be used cautiously over a small surface at a time at first, as occasionally in strongly predisposed persons the pigmentation returns worse than before.

4. Argyria.—Argyria is the term applied to the staining produced by the long-continued administration of nitrate of silver, either internally as a remedy for epilepsy or other diseases, or as a topical application to the throat. The reduced metal is deposited in the rete Malpighii and sweat-glands, and round the hair-roots. It is said that 450 grains is the minimum quantity taken internally which has produced the staining; but it is wise to be well within this limit, as, when once it has commenced to show itself, nothing will stop its further development or remove it afterwards. The parts exposed to light, the face, hands, and visible mucous membranes, are most strongly implicated. The colour is of a bluish-grey, slate, or leaden hue, or it may be almost black. Iodide of potassium in large doses has been recommended, but all treatment is unavailing.

5. Arsenical Pigmentation.—This is due to the deposition of the metal in the skin from prolonged administration of arsenious acid. It is general, but attacks most the parts where diffuse pigmentation is usually most marked. At first it may be recognisable by its sparing the central parts of the hair-follicles, so that on the abdomen there are whitish dots on a yellowish-brown ground, but at a later stage the discoloration is uniform. It may be accompanied by keratosis of the palms, soles, elbows, knees, knuckles, and web of the fingers. When psoriasis has been removed by the administration of arsenic, the site of the psoriasis-patches may be the only parts pigmented. If the drug is stopped, the pigmentation usually fades in the course of time.

6. Albinism.—This disorder of pigmentation is described in a special article. *See* ALBINISM.

7. **Leucodermia.**—SYNON.: *Vitiligo*; *Leucopathia*.

This disease is much more frequent in tropical than in temperate climates. It is a mixed condition of faulty pigment-distribution, but is named from its most conspicuous feature. It is notably symmetrical. Its first stage, though often overlooked, is an increase of pigment in certain regions; in this darker area, a white spot forms and enlarges, driving, so to speak, the pigment before it, so that the borders of the white part are convex, and those of the adjacent over-pigmented part concave. The dark part is sharply defined on the white side, while away from the white area it merges gradually into the normal skin. The number and extent of the white areas vary considerably, but the affection may in course of years affect the whole surface, the white part ever encroaching on the dark; or the excess of pigment may be absorbed. In either case, in some regions, or even over the whole surface, an apparent cure is produced in white races from want of contrast, but the part is really abnormally white, and will no longer tan in the sun.

ÆTIOLOGY.—The affection is certainly more common in dark races, and is generally connected with exposure to the sun, some cases dating from sun-stroke; but it has occurred after severe cold. The neurotic temperament and nerve-depressing influences appear to be favouring conditions. The pathology of the process has been discussed in the general statement.

DIAGNOSIS.—Its striking symmetry, progressive course, and the combination of excess with deficiency of pigment, together with the absence of all symptoms other than the pigmentary anomaly, render the diagnosis of leucodermia easy. The last feature is important, as in India the disease has sometimes been called 'white leprosy.'

PROGNOSIS.—The prospect of cure is bad; spontaneous arrest occurs; but apparent cure by the process previously described is the result to be most desired.

TREATMENT.—This is highly unsatisfactory. General tonic and invigorating treatment offers the best chance of arresting the progress of disease; and, of course, arsenic is recommended by some authors. White areas in a conspicuous position might be slightly stained with walnut-juice to render the contrast less striking.

H. RADCLIFFE CROCKER.

PIGMENTATION.—Abnormal coloration of living tissues, not produced by artificial means. The condition may be due to excess of a pigment which is normally present in the tissue affected, or to the introduction of foreign substances.

Pigment is normally present in the deep layers of the epidermis and in the choroid and retina. The colouring matter is known as 'melanin' and is derived from the blood by the action of the living cells; it does not contain iron (*see* MELANIN). Large amounts of this pigment are found in melanotic tumours. Pigmentation of the skin due to excess of this material is met with after exposure to the sun (freckles; sunburn); in Addison's disease; in irritation of the abdominal sympathetic nerves (pregnancy; tumours); and in the conditions known as melanoderma, acanthosis nigricans, and bronzed diabetes. *See* PIGMENTARY DISEASES OF THE SKIN.

Abnormal forms of pigment may be produced by

destruction of blood-corpuscles, the hæmoglobin breaking down into hæmatoidin, hæmosiderin, and other coloured derivatives. Thus when blood is extravasated into the tissues, the surrounding parts are infiltrated with lymph containing dissolved hæmoglobin, and brown pigment is found deposited in the nearest lymphatic glands (*see* EXTRAVASATION). The series of changes of colour which occurs after a superficial bruise illustrates the resulting pigmentation. Similarly organs become pigmented as a result of chronic congestion or inflammation, owing to the escape of blood-corpuscles which occurs under these circumstances: instances are seen in the 'brown induration' of the lungs which results from the continued mechanical hyperæmia of heart-failure; in the slate-coloured mucosa of the bladder in chronic cystitis, or the 'cut-beard' appearance of the intestine in chronic catarrh; and in the pigmented patches and scars which often remain after chronic lesions of the skin, especially those due to syphilis. The breaking up of blood-corpuscles in the circulation, as in malaria, may lead to deposit of masses of pigment in the liver and spleen.

The so-called pigmentary degeneration of the heart is the result of breaking down of the muscle-substance, brownish granules being found in the fibres chiefly arranged around the nucleus; but it is doubtful whether the dark colour of the heart-muscle produced in this condition can properly be called pigmentation, and is not rather due to concentration of the normally existing pigment in a shrunken organ, as occurs in the liver in cases of starvation.

The staining of the tissues in jaundice is due to absorption of bile-pigment into the general circulation. *See* JAUNDICE.

Various foreign substances may be deposited in living tissues, apart from their artificial introduction, as in tattooing. Thus coal and other forms of dust may be inhaled in respiration and deposited in the lungs, giving rise not only to direct coloration of the tissues, but also to chronic interstitial pneumonia (*see* PNEUMOCONIOSES). Salts of silver taken as remedies over a long period of time produce a bluish discoloration of the skin (argyria) which remains permanently; it is due probably to deposit of reduced silver in the skin. The 'blue line' seen in the gums in cases of lead-poisoning is due to insoluble sulphide of lead formed by the action of sulphuretted hydrogen, given off from decomposing material round the necks of the teeth, upon the lead-salts circulating in the blood. In chronic copper-poisoning a similar line of a greenish tint may be seen.

The growth of the parasitic fungus, *Microsporon furfur*, upon the skin gives rise to brownish patches which may coalesce and cover large areas. The condition is easily distinguished from other causes of pigmentation by microscopical examination of scrapings from the brown areas, in which the fungus is easily recognisable. *See* TINEA VERSICOLOR.

For the discoloration of organs after death, due to the process of decomposition, *see* DEATH, Signs of.

W. CECIL BOSANQUET.

PILES.—A popular name for hæmorrhoids. *See* HÆMORRHOIDS.

PIMPLES.—A popular name for papules. *See* PAPULE.

PINTA.—A term applied to a group of tropical, contagious diseases of the skin, caused by the growth of different fungi. The appearances vary with the species of parasite present.

PISA, in Central Italy.—A rather moist, mild, equable, calm, and relaxing climate. Mean temperature, winter 44° F. East prevailing wind. See CLIMATE, Treatment of Disease by.

PITUITARY BODY.—See ACROMEGALY.

PITYRIASIS (πῑτυρις, bran).—SYNON. : Furfur; Porrigo; Fr. *Pityriasis*; Ger. *Kleingrind*.

Pityriasis is a term indicating the existence of branny exfoliation of the epidermis. In the days of Willan and his followers it was employed to designate a heterogeneous group of skin-diseases exhibiting the one common feature of branny scales. Many members of the group are now recognised to be mild forms of dermatitis. Pityriasis versicolor has become Tinea versicolor. Pityriasis nigra was used by Hebra to designate the pigmentation and scaliness found in chronic pediculosis. Slight degrees of dry eczema, especially seborrhoeic forms, were also called pityriasis, e.g. the pale red patches with small scales often seen in children from using too strong a soap. The form of seborrhoea sicca, called dandriff (p. 362), and the fine silvery scales shed in such abundance by many elderly persons, were both called pityriasis capitis, and it is perhaps advisable to retain the name for this condition. As a generic term it is still used. See PITYRIASIS ROSEA; and PITYRIASIS RUBRA.

H. RADCLIFFE CROCKER.

PITYRIASIS ROSEA.—SYNON. : *Pityriasis Circinata et Maculata*; *Herpes Tonsurans Maculosus* (Hebra).

DEFINITION.—An inflammatory eruption, developing acutely like an exanthem, characterised by the formation of pale red, delicately scaly, roundish or oval patches and circles.

ÆTIOLOGY.—Pityriasis rosea is uncommon, about one in 250 cases of all skin-eruptions. It is more frequent in children, though it may be seen at all ages. It occasionally occurs in more than one member of a family, suggesting a possibility of contagion, but there is no definite proof that the complaint can be so transmitted.

DESCRIPTION.—In a slowly developing case of moderate extent there may be no general symptoms, but in an acute and widespread outbreak, slight febrile and other symptoms of general disturbance may precede the generalisation of the eruption; there may be slight enlargement of the glands behind the sterno-mastoid and under the jaw, and sometimes elsewhere, as well as congestion of the fauces. These symptoms accompany the generalisation of the eruption, but a week or ten days before this there has been a primary patch of the same character as the maculate form about to be described. It is often overlooked by the patient, but may be recognised by its being larger and perhaps more ringed than the more recent ones; after some days others form in the neighbourhood; the abdomen, chest, or side of the neck being the regions usually first affected. From one of these parts the eruption spreads, the old part usually clearing away, leaving slight pigmentation, while fresh patches develop, so that in the course of days or weeks the whole body surface may become in-

volved, and all stages of progression and retrogression may be simultaneously observed in different regions. There are two forms of the eruption, the *maculate* and the *circinate*.

Pityriasis rosea maculata consists of roundish, oval, or irregular pale red patches, from a pin's head to a shilling in size, with well-defined borders, very slightly raised above the surrounding skin, and with the surface more or less covered with very fine delicate scales.

Pityriasis rosea circinata, as its name implies, occurs in circles. These commence as patches, and, as they enlarge, clear in the centre, which is left slightly pigmented, while the border is well defined and slightly scaly. When they meet other patches, gyrate areas are produced. There may be slight itching when the patient is warm. The disease tends to get well spontaneously, lasting from two weeks to two months, occasionally longer.

PATHOLOGY.—The pathology is unknown. The 'Microsporon anomoeon,' described by Vidal, has not been accepted as the cause of the affection, but the commencement in a primary patch which enlarges peripherally, and the generalisation of the eruption after an interval, suggest that the disease is due to an organism which invades the skin from without, and is subsequently absorbed into the circulation.

DIAGNOSIS.—The pale red, slightly scaly, barely elevated patches or circles, covering a wide area, and undergoing spontaneous cure, are the most distinctive features of this disease. It is much less raised, and less scaly, than the early scaly or circinate syphilide; has no scaly crusts, but only delicate branny scales; and is not so red or so raised as psoriasis. It spreads too rapidly and widely for tinea circinata; and there is, of course, no fungus as in trichophyton tonsurans. It is not, like lichen circinatus or seborrhoea corporis, confined to the middle of the back and chest, and is never in papular circles; moreover, while that affection lasts for years if untreated, pityriasis rosea gets well in a few weeks or months.

TREATMENT.—Internal treatment is not required in the majority of cases, but in those which have lasted several months the author has found that salicin in 15-grain doses three times a day produces involution; and there can be no harm in giving it at the commencement in all cases, with a fair chance of shortening the duration. A lotion consisting of liquor carbonis detergens, ʒij to ʒviij of water, is useful in allaying itching, and is perhaps curative. At all events, cases soon get well while using it. If the patient is seen when there are only one or two patches present, these may be painted with tincture of iodine, or vasogen-iodine (10 per cent.) may be rubbed in.

H. RADCLIFFE CROCKER.

PITYRIASIS RUBRA (πῑτυρις, bran; *ruber*, red).—SYNON. : *Dermatitis Exfoliativa*; *Eczema Foliaceum*; Fr. *Herpétides Exfoliatives*; *Erythrodermie Exfoliante*; Ger. *Hautkleie*.

DEFINITION.—An extensive and important natural group of skin-affections, characterised by universal, or almost universal, dermatitis; by marked chronicity; by the severity of the congestion and the intense redness of the parts affected; and by the abundance of desquamation, either in the form of fine branny dust, or of large scales or sheets of epithelium.

The term is not applicable to a somewhat similar

condition, which may be caused by the exanthemata (especially scarlatina, erysipelas, r  theln and measles), or by certain drugs (for example, chloral hydrate, belladonna, quinine, copaiba, and phenazone). See DRUG-ERUPTIONS.

  TIOLOGY.—The causes of pityriasis rubra are very obscure. It occurs more frequently in the male than in the female sex, in the proportion of three to two, or perhaps higher. It is most commonly met with between the ages of forty and sixty years, but has been observed in a child of four months, and in a man of ninety years. It is not hereditary, nor is it contagious; but a condition indistinguishable from pityriasis rubra as regards its objective characters, for a time, prevailed in an epidemic form, especially in the infirmaries and workhouses of the west end of London. Gout, and still more rheumatism, strongly predisposes to its occurrence, either as a primary dermatosis or as an epiphenomenon of other skin-diseases; and the same remark applies to chronic alcoholism and interstitial nephritis in a less degree. Its ‘secondary’ form may supervene upon psoriasis or—in diminishing grade of frequency—upon eczema, lichen ruber, erysipelas, seborrh  a, erythema multiforme, pityriasis rosea, or even artificial dermatitis, for example, from chrysarobin.

The most frequent immediate antecedent of pityriasis rubra is a chill, while the subjects of it are frequently neurotic and intensely susceptible to cold, with other evidences of ill-balanced peripheral circulation. Severe nervous shock and mental strain seem mainly responsible for a certain number of cases.

ANATOMICAL CHARACTERS.—These are in no sense characteristic. They include marked congestion and dilatation of the blood-vessels of the most superficial parts of the skin, and comparatively little of those of the deeper parts; separation of the upper two-thirds from the lower third of the horny layer; thinning of the Malpighian layer over the papill  , with great increase in size and depth of the inter-papillary processes; marked hypertrophy of the papill  , with intense leucocytic infiltration of them, and of the upper layers of the corium. Elacin and collacin are present as in senile skin. In a later stage the whole thickness of the skin is involved, and ultimately a process of sclerosis takes place, with formation of cicatricial tissue, pigmentation, and destruction of sebaceous and sudoriferous glands.

SYMPTOMS.—From the foregoing definition it will be manifest that a large number of maladies present the common characters attributed to pityriasis rubra, the synonym for which (*dermatitis exfoliativa*) is now in habitual use to connote the condition, both in Great Britain and on the Continent.

TYPE I. Secondary pityriasis rubra.—Generalised red and desquamative eruptions secondary to other dermatoses (herp  tides exfoliatives, Bazin).

The diseases upon which this condition may supervene have already been enumerated. In the case of eczema discharge diminishes and small, dry, whitish scales form in increasing numbers. The scaling of psoriasis becomes finer, while the margins of its patches usually become more and more ill-defined, although in one case observed by the writer blebs formed round them. The bull   of pemphigus become flaccid, and resemble the

lesions of pemphigus foliaceus. Gradually from the primarily affected parts the erythrodermia spreads over the whole body surface, which becomes covered with fine branny scales, the feet and soles being the parts most frequently spared. The nails become hypertrophied and claw-like, or may be shed and repeatedly re-formed. The general condition is usually maintained, the appetite and digestive functions being unimpaired; but occasionally marasmus sets in, albumen appears in the urine, severe diarrh  a or pneumonia supervenes, and the case terminates fatally. In the great majority of cases, however, spontaneous recovery ensues in from six to twelve months after the occurrence of an indefinite number of relapses. The primary skin-trouble often reappears, and each recurrence is prone to be followed by a generalised exfoliative dermatitis of gradually increasing severity.

TYPE II. The relapsing, desquamative, scarlatiniform erythema of F  r  l represents the mildest form of primary exfoliative dermatitis. It is little known in this country. It is of more frequent occurrence in children and young adults than the other types. Its onset is well defined, with pyrexia (102  –104  ) and general malaise, sometimes even with sore-throat and a desquamating, scarlatinal-looking tongue. The eruption does not usually make its appearance for two or three days. It consists of an intense, more or less uniform redness, sometimes with small purpuric spots, but without any infiltration; its commonest situation is the legs, from which it spreads over the whole body. After its persistence for three or four days copious fine or coarse desquamation sets in, fever diminishes, and the redness begins to subside. At the end of a fortnight recovery is sometimes complete, but more generally repeated recrudescences prolong the duration of the disease over six weeks or two months. An accurate diagnosis in a first attack can only be a matter of surmise, but the subsequent course of the disease renders its nature indubitable. Relapses presenting the characters of the first attack invariably occur, and generally at regular seasonal intervals, most commonly in spring, when the return is usually attributable to a chill. With each relapse the severity of the disease tends to diminish, and finally perfect recovery generally takes place. It appears that sometimes the ingestion of certain drugs (especially mercurials) or the application of irritants (blisters, &c.) to the skin is responsible for the initial attack, which may be followed by relapses in the manner described. The disease is not infectious, and does not occur in epidemics; it is not accompanied by albuminuria, and loss of hair is very exceptional. A transverse groove on each nail is, however, generally left by each attack, while in the most severe cases the nails may be shed. This brings the disease into close relationship with the next type.

TYPE III. The primary universal exfoliative dermatitis of Erasmus Wilson is a less rare and more severe affection than the foregoing. Its objective characters may, in exceptional instances, be perfectly copied by cases of secondary exfoliative dermatitis, but the differences in clinical history and in their usual course justify their differentiation. As a rule, the subject attacked is a healthy adult male. The onset is acute, with rigors or chills, or may be insidious and accompanied merely

by 'malaise.' The temperature rises to 102° F. or higher; and until the eruption is on the wane it generally remains above the normal, with a marked evening rise. The eruption usually appears in the form of red erythematous patches, with fine desquamation, situated on the trunk, but it rapidly spreads, to become generalised over the whole body-surface. The skin is of an intense bright-red colour, but if exposed to cold often becomes of a dusky blue; in severe cases it is somewhat infiltrated, hard, and stretched. Soon the epidermis begins to exfoliate, and copious branny scales form and fall from the face, head, and neck; while large papery flakes are shed from the trunk, or remain attached by their centres, overlapping like slates at their margins. Two or three pints of such scales may be shed in the twenty-four hours. The skin of the palms and soles is usually detached *en masse*, and the redness there does not show itself until after the first exfoliation. The amount of itching varies within wide limits; generally it is severe, and sometimes it precedes the rash, drawing the patient's attention in the first place to it. Sometimes, however, there is no itching, and the subjective symptoms most commonly complained of are either tenderness or a sense of burning, while a feeling of cold is experienced by the patient as soon as he is exposed. Oozing from the flexures of the knees, elbows, axillæ and thighs sometimes occurs, and is often no doubt attributable, in part at least, to rubbing or scratching of the part. In a few very exceptional cases, bullæ have been observed in these situations, allying the disease with pemphigus foliaceus (p. 1175). The skin over the rest of the body remains intensely harsh, dry, and scaly, although from time to time copious sweating may occur. The scalp is extremely seborrhœic, while pustules, boils, and 'epidermic cones' surrounding lanugo-hairs are of frequent occurrence. The mucous membranes become involved; coryza, conjunctivitis, superficial glossitis, stomatitis, and pharyngitis may be observed; while the occasional occurrence of vomiting and diarrhœa probably indicates implication of the gastric and intestinal mucous membranes. The shedding of nails and loss of hair early in the disease are characteristic of this type; they may occur as early as in the fourth week. In a certain number of cases the general condition remains remarkably good, but in others the appetite is lost, the patient emaciates, and alarming prostration sets in. The appearance of albumen in the urine, or the development of pulmonary troubles or of diarrhœa, may usher in a lethal termination (generally in the third or fourth month); but undoubtedly in the majority of cases ultimate recovery occurs in from six to twelve months, after repeated disappointing recrudescences. As a rule, the redness first diminishes, then the scaling, and the two subside gradually together. Among complications and sequelæ Brocq enumerates carbuncles, abscesses, phlegmons, deafness, iritis, joint-troubles, cardiac complications, partial paralysis, paraplegia, and mental affections.

TYPE IV. *The primary, universal, chronic pityriasis rubra* of Hebra is a rarer and more grave disorder than the preceding. Its onset is always gradual, and unaccompanied by fever; it is evidenced by the appearance of dry, scaly, red patches, generally on the trunk, which gradually extend, to become universal after a period of several months or, it may be, years. The colour is usually dusky,

and the desquamation is always fine. There is little or no infiltration of the skin. Constant chilliness is complained of, but pruritus is seldom a troublesome symptom. Very gradually the skin becomes infiltrated, hard, brawny, and pigmented; then sclerosed, glossy, yellowish, and stretched over prominent bony points, or contracted round the orifices of the orbits and mouth (ectropion, eversion of lips). Slowly, also, but surely and without intermissions, the hair atrophies and falls out; and the nails become thin and crack, or much thickened and friable. Marasmus sets in after years, and the patient gradually sinks as the result of pulmonary tuberculosis, pneumonia, or diarrhœa. In the later stages ulceration and gangrene may hasten the fatal termination.

PATHOLOGY.—Of the intimate pathology of this group of diseases but little is known. Pye-Smith considers the dermatitis as primary; but there seem to be many valid reasons for considering the condition as essentially a vaso-motor and trophic neurosis, more probably of central than of peripheral origin. The co-existence of myelitis has been noted, and inflammatory changes in the peripheral nerves have been observed, but these are certainly not constant either in their occurrence or in their nature when present.

DIAGNOSIS.—This is usually easy; the history of the case, the universality of eruption, the intense redness and dryness of skin without marked infiltration, the absence of discharge, the characters of the desquamation and the moderate amount of itching, combined with the grave general condition, all serve to distinguish pityriasis rubra from extensive psoriasis, eczema, or lichen planus. Pemphigus foliaceus may be very hard to eliminate from the diagnosis, but generally its bullæ may be distinguished with their foul-smelling contents; it also occurs more frequently in women than in men. Epidemic exfoliative dermatitis (Savill) most closely simulates this disease; indeed, there seems to be no criterion for differentiating them beyond the history of the presence of an epidemic of the former, and the discovery and constant presence in the blood of persons attacked by it of a diplococcus of supposed specific nature.

PROGNOSIS.—The prognosis may be gathered from the study of any case in the light of the description of the various types given. Particular stress is to be laid upon the state of the appetite and digestion, and the presence or absence of albuminuria, pulmonary complications, and emaciation.

TREATMENT.—Treatment must be assiduous, and directed both to the local and general condition. The patient must be absolutely confined to bed, even in the mildest cases, and until recovery is complete, as the slightest chill is prone to cause relapse. He ought to lie between blankets, and on a spring mattress or water-bed. The diet must be nutritious but simple, including milk, in as large quantity as possible, eggs, milk-puddings, strong meat-soups or jellies. Alcohol ought not to be prescribed, unless the prostrated condition of the patient demands it; and then sound wine is preferable to spirits, as causing less thirst and gastric disturbance. Cod-liver oil is sometimes of use when emaciation is marked. Iron appears sometimes to be detrimental, but quinine in an effervescing citrate-of-potassium mixture is certainly sometimes useful and grateful to the patient. Diuretics, such as acetate of potassium with digitalis, are

frequently employed and probably beneficial. The promotion of free diaphoresis by jaborandi or the subcutaneous injection of nitrate of pilocarpine has been advocated by Stephen Mackenzie and others. Linseed oil in large quantities, both externally and internally, has been lauded by Sherwell. The bowels must be carefully regulated, preferably with mineral waters and salines, the latter often profitably combined with the liquid extract of cascara sagrada. There is a general consensus of opinion that arsenic is worse than useless in the earlier stages, while few writers accord it anything but faint praise even in the later. The writer has been disappointed with the effects of antimony. He has also noticed aggravation of the eruption after the administration of opium, chloral hydrate, belladonna, and Cannabis Indica, with a view to obtaining sleep.

Oily external applications are certainly the most beneficial. The calamine-liniment recommended by Radcliffe Crocker is an admirable preparation. It is composed thus: Calamine 40 grains; oxide of zinc 2 drachms; olive oil and distilled water, each 1 fluid ounce. The patient may be swathed in rags soaked in this liniment, slightly warmed if necessary. The pharmacopœial linimentum calcis is also useful, as is a lotion consisting of an ounce of glycerole of lead and of glycerine in a pint of water, similarly employed. Pure vaseline is occasionally a valuable substitute.

Prolonged warm baths may be of service in diminishing itching, but are apt to be followed by increase of cutaneous hyperæmia. This risk is materially diminished by the addition of bran (6 lbs.), linseed (1 lb.), gelatine (3 lbs.), or potato starch (1 lb.) to the thirty-gallon bath. The further addition of two or three ounces of borax or bicarbonate of sodium is often advantageous.

Pityriasis Rubra Pilaris (Devergie-Richaud).

SYNON.: *Lichen ruber acuminatus* (Hebra).—

This disease, the ætiology of which is not known, is insidious and non-febrile in its onset, young persons being frequently its subjects. The writer has seen one case in a child two years old. Desquamative, psoriasiform patches on the palms and soles; a seborrhœic, eczematoid condition of the scalp, or a fine branny scalliness of the face are the commonest initial symptoms. Sooner or later the characteristic lesions appear upon the limbs or body, especially where hair is abundant, in the form of small brownish-red, firm, dry, conical papules, seldom larger than a millet-seed, and surrounding atrophied hairs. These 'epidermic cones' give the skin a harsh and grater-like feeling to the touch; they send little plug-like processes into the follicles, from which they can be dislodged by rubbing, leaving fine cribriform pits. As the disease progresses these lesions become confluent, when their characters are lost, and yellowish, infiltrated, desquamating patches are formed which may closely resemble psoriasis, especially about the elbows and knees. A few typical primary lesions are usually present for some time about the periphery of these patches. The surrounding and intervening skin subsequently becomes red and infiltrated, with desquamation either of fine, branny, or coarse mortar-like character, and the condition may become practically universal; the palms and soles, where there are no hair-follicles, are usually conspicuously involved, being hyperæmic and scaly. The primary lesions generally retain their characters on the backs of the first and

second phalanges of the fingers and toes, where they are almost pathognomonic of the disease. Severe seborrhœa capitis is always present, and the hair may fall in quantity; on the other hand, hypertrichosis of the affected parts has been noted in some cases. The nails are usually soft and transversely striated, or may become enormously hypertrophied and claw-like. The face assumes a peculiar powdered appearance, owing to the dry, white scales lying on the reddened skin, and sometimes ectropion results from contraction. Itching is usually slight, if present at all.

PATHOLOGICAL ANATOMY.—The essential lesion is a hyperkeratosis of the epithelium lining the hair-follicle, to the bottom of which it penetrates. This hyperkeratosis extends to the epithelium of the general surface of the skin and, to a slight extent, affects the sweat-ducts. The inflammatory phenomena in the cutis are secondary.

DIAGNOSIS.—This disease must be distinguished from follicular ichthyosis (keratosis), lichen pilaris, psoriasis, and pityriasis rubra, to all of which reference may be made.

PROGNOSIS.—Recovery always takes place, but the duration of the disease is quite indefinite, being frequently marked by remissions and exacerbations without ascertainable cause.

TREATMENT.—The production of diaphoresis by active exercise, Turkish baths, and pilocarpine is probably beneficial. Arsenic is useless, if not actually harmful. The local treatment may be conducted on the lines laid down in Pityriasis rubra and Ichthyosis. J. J. PRINGLE.

PITYRIASIS VERSICOLOR (πίτυρον, bran). See TINEA VERSICOLOR.

PLACENTA, Diseases of.—Comparatively little is known of placental diseases. This is explained by the fact that the normal structure of the organ at different stages of pregnancy has only in recent years been clearly defined; pathological work had been founded upon an inadequate anatomical basis, and many of the earlier investigations of supposed morbid conditions have been invalidated by later knowledge of the normal structure. In this article, accordingly, only such matters will be referred to as are now fully established by recent research.

Changes due to Ripening.—It has been shown by Whitridge, Williams, the present writer, and others, that during the last two or three months of pregnancy certain changes occur in the placental tissues which are preparatory to the process of shedding during labour. Briefly stated, these changes consist in obliterating endarteritis of the foetal arteries leading to atrophy of the chorionic epithelium, to the formation of numerous solid patches in the spongy placental tissue, termed *infarcts*, and to extensive deposition of fibrin from the maternal blood upon the villi in the affected areas. Numerous groups of villi are thus rendered functionless. These changes are in reality senile, and not due to disease at all, but they have frequently been erroneously attributed to inflammation, hæmorrhage, or syphilis.

Changes in Retained Placenta.—When the foetus dies and the ovum is not discharged from the uterus, certain well-defined changes occur in the placenta as a result of the suspension of the circulation through it. These changes consist in extensive formation of blood-clot, in atrophy of villi, and in the

deposition of large quantities of fatty and calcareous material in the altered structures (fatty and calcareous degenerations). These changes, which are in reality *post mortem* (following the death of the foetus), have been frequently cited as the morbid conditions to which the death of the foetus was due, and in this way much confusion has been introduced into the pathology of the placenta.

1. Inflammation (Placenteritis).—So far as is known placenteritis does not occur except as the result of septic or gonorrhoeal infection of the uterine cavity. Retained placental fragments are especially liable to undergo putrefaction, and the whole ovum may become involved in uterine gonorrhoea or sepsis. A dead ovum which has been retained *in utero* for a prolonged period occasionally undergoes putrefaction even if no operative interference of any kind has been practised, infection, in such cases, probably occurring from the bowel. It is possible that the condition known as Morbid Adhesion of the placenta is the result of an inflammatory affection of the decidua serotina, but in any case the foetal portion of the organ is not involved in the process.

2. Hæmorrhage.—The conditions known to obstetricians as Placenta Prævia, and Accidental Hæmorrhage, are instances of hæmorrhage from separation of the placenta from the uterine wall, and in both instances bleeding frequently occurs during the last two months of pregnancy before the onset of labour. In both the placenta is usually healthy, but in the latter morbid conditions are not infrequently present which may cause the premature detachment of the organ. The subject will, however, be found more fully discussed in another place (see PREGNANCY, Diseases of). We are not concerned here with hæmorrhage into the chorion before the formation of the placenta, i.e. during the first three months of pregnancy. This subject is treated under 'Molar Pregnancy' (p. 1016).

Hæmorrhage into the substance of the placenta occurs in two widely different forms: (a) Small interstitial hæmorrhages; (b) Large effusions often termed 'placental apoplexy.'

(a) *Interstitial hæmorrhages* are not uncommon; they occur in the form of black circumscribed areas which stand out in marked contrast to the purplish colour and reticulated structure of the healthy placental tissue. There may be many such small areas scattered through the organ, or an entire cotyledon may be thus affected while the remainder of the placenta is healthy. Under the microscope the hæmorrhagic areas are seen to consist mainly of blood-clot, with a few widely separated villi here and there, which are usually broken or distorted in appearance but otherwise normal. These 'hæmorrhages' appear to be the result of a relatively excessive afflux of blood to the maternal spaces of the affected part, or some hindrance to its return. Speaking strictly, the condition is not hæmorrhage, because the blood is still contained in the inter-villous spaces, and has not escaped from its normal channels. But it differs from mere congestion or engorgement of vessels, inasmuch as the surrounding villi are broken up and their relations altered by the process. It is, therefore, better to regard the process as hæmorrhage. Interstitial hæmorrhages do not appear to affect the functional capacity of the placenta to an extent sufficient to react unfavourably upon it. It is probable that they are often produced during labour by the in-

fluence of the uterine contractions upon the maternal placental circulation.

(b) *Large effusions of blood* are much rarer than the interstitial hæmorrhages just described; they are more rapidly produced, and therefore break up the placental tissues to a greater extent than the interstitial ones. In consequence irregular cavities of considerable size containing blood-clot are formed in the substance of the placenta, limited, in recent cases, by compressed and broken villi, and in older cases by layers of fibrin forming a false capsule. In some of these cases the hæmorrhage is fatal from rupture of a branch of the umbilical vessels within the placental tissue; oftener the blood is maternal, and then the cavity generally communicates with the circular sinus. The effused blood first accumulates in the placenta, and may then burst through the serotina, causing some separation of the placenta from the uterine wall. These hæmorrhages invariably cause the death of the foetus, or lead to almost immediate abortion. They occur in association with maternal albuminuria or chronic nephritis, or with valvular lesions of the heart (maternal); if associated with albuminuria, pathological infarction is often found in the areas unaffected by the hæmorrhage. If abortion does not immediately follow, the changes which usually occur in effused blood are found, viz. clotting with partial organisation or degeneration.

The clinical diagnosis of placental hæmorrhage at the time of its occurrence is not practicable; the amount of blood effused is not large enough to cause constitutional symptoms.

3. Œdema.—Œdema of the placenta is usually associated with œdema of the foetus, and the causes of the two conditions appear to be the same. Instances have been recorded where obstruction to the foetal circulation from intra-uterine closure of the foramen ovale, or thrombosis of the umbilical and hypogastric vessels has been the cause of the œdema; the placenta of acardiac and other monsters is also often œdematous. Anasarca of the mother as from renal or cardiac disease has often been noted, and syphilis is also said to cause it. Virchow and Franck have observed that in general dropsy of the foetus of bovine animals the thoracic duct and mesenteric lymphatics were absent, but this point does not appear to have been ever investigated in the human foetus.

An œdematous placenta is larger, heavier, and paler than normal, and undergoes a great diminution in weight if incised and the fluid allowed to drain away from it. The normal weight of the placenta is about 16 ounces; an œdematous placenta frequently weighs twice that amount; the heaviest on record weighed 56 ounces. On microscopical examination the stroma of the villi and the larger chorionic branches is seen to be the seat of the œdema, and in two specimens which the writer has examined fatty degeneration was also present. When the placenta is œdematous the foetus born with it is usually dead, and often macerated, showing that death occurred some time before labour set in, but, as has been already stated, other morbid conditions of the foetus or the mother are frequently associated with it.

4. Pathological Infarction.—It has been already stated that during the last two months of intra-uterine life certain changes referable to senility occur in the foetal placenta resulting in the formation of small solid bodies termed 'infarcts.'

in the spongy placental substance. In healthy conditions these are neither large enough nor numerous enough to interfere with the functional capacity of the placenta or the development of the foetus. Within the last decade it has been shown that changes similar in their nature, but occurring much earlier in gestation, and affecting a much larger proportion of the placental tissues, are found in connection with a well-defined maternal disorder—albuminuria of pregnancy. This disorder is known to depend either upon chronic nephritis pre-existing pregnancy, or upon a transient form of renal disease which frequently develops during pregnancy and disappears rapidly after labour. A heavy foetal mortality, estimated at about 54 per cent., has long been known to accompany this condition, and there is good reason to believe that the changes which occur in the placenta are the cause of the death of the foetus. In a marked example a considerable proportion—one-third to one-half—of the placental substance is converted into a firm, yellowish, bloodless tissue, which is seen under the microscope to consist of large deposits of fibrin surrounding atrophied chorionic villi, both elements usually showing marked fatty degeneration. Advanced obliterative endarteritis is found in the foetal vessels, where these can be distinguished. Hæmorrhages are frequently found in non-infarcted portions of the placenta. Cases in which labour occurs at the sixth or seventh month frequently show an extreme degree of these changes, and, generally speaking, cases which go nearer to term show less marked changes than those in which labour comes on earlier, although it is rare, in any case, for pregnancy to advance beyond the thirty-sixth week in this condition. In addition to these changes in the placenta, the foetus is smaller and of less weight than the healthy foetus of the corresponding period; it is, in fact, under-developed and possesses little vitality. The foetus suffers, apparently, in direct proportion to the extent of the placental changes, and there is little doubt that these changes exert an unfavourable influence upon its development. Labour is frequently delayed for some time after the death of the foetus, which is then born in a macerated state.

The manner in which the placental changes affect the foetus is sufficiently clear. The infarcted portions of the placenta are functionless because both the foetal and maternal circulation through them have ceased; the villi are atrophied and devascularised, while the maternal inter-villous spaces are obliterated. If a large portion of the placenta is thus affected, the remainder is inadequate to the task of depurating and enriching the foetal blood; the nutrition and development of the foetus therefore suffer. If hæmorrhage should occur into the non-infarcted portions, the foetus will probably perish suddenly; some separation of the placenta from the uterine wall may occur at the same time, leading to accidental hæmorrhage, or there may be no sign until labour sets in. The ætiology of the placental changes is not so clear. Women suffering from albuminuria during pregnancy are generally very anæmic, and there is extensive anasarca: there is some evidence of the existence of a toxæmia in the albuminuria of pregnancy. Impurities may therefore pass through the villi into the foetal circulation, and, by contact with the walls of the foetal vessels, induce the process of obliterative endarteritis, which is probably the initial change.

5. Degenerations.—The tissues which compose the placenta are embryonic in type and prone to degeneration of various kinds.

a. Vesicular Degeneration (Hydatidiform Mole) is a form of myxomatous degeneration of the chorionic stroma which usually occurs at an early period before the differentiation of the placenta takes place; it therefore affects the chorion rather than the placenta, and will be found fully described in another place (p. 1017). In rarer instances it occurs later in gestation and then affects the placental chorion. The disease has then the same general characters as in the hydatidiform mole, but the whole placenta is seldom or never attacked by it; usually one or more cotyledons suffer while the remainder of the organ escapes. The life of the foetus is apparently always destroyed, for no instance is on record where the placenta of a living foetus has been found to be affected by this disease.

b. Fatty and Calcareous Degenerations.—A certain amount of fat is present in the normal human placenta, being located in the superficial layers of the decidua and in the plasmodial layer of the chorionic epithelium (syncytium); it occurs in minute non-confluent droplets, and its total amount is very small. Fatty degeneration is most commonly found as a sequela of pathological infarction in albuminuria, and is then to be referred to malnutrition from extensive obliteration of the foetal vessels. Along with other conditions such as cedema, and in placenta retained *in utero* after the death of the foetus, it is also not uncommon. A deposit of lime-salts—calcareous degeneration—not infrequently occurs in parts which have undergone fatty degeneration. In a fully ripe placenta it is usual to find small thin calcareous plates upon the uterine surface; they are located in the decidua serotina and do not as a rule involve the chorionic villi. Cases have been recorded where the uterine surface has been thus almost covered with a plate of calcareous material, the deposit occurring chiefly in the decidua serotina.

c. Cystic Degeneration.—Cysts occur but rarely in the placenta. They are, however, occasionally observed upon the foetal surface of otherwise healthy placenta in the form of small, multiple collections of clear, yellowish fluid. The amnion can be readily peeled off them, as they are situated in the chorionic membrane; more rarely still, similar cysts are found in the amniotic membrane itself. They are produced by myxomatous degeneration of the connective tissue of the membrane in which they lie, and possess no epithelial lining, their walls being formed of degenerating and compressed connective tissue. The connective tissue of the foetal membranes is embryonic in type and very prone to this form of degeneration. Cysts on the foetal surface of the placenta never exceed the size of a pigeon's egg, and do not interfere in any way with the functional activity of the organ. Infarcts, when incised, are often found to contain cystic cavities with ragged, irregular walls in their interior. These cavities are formed by necrosis and they contain a thick pulpy fluid consisting of *débris*.

Myxoma fibrosum.—This is a peculiar form of myxomatous degeneration with hypertrophy of the chorionic villi. It was first described as a new-growth, but is more accurately classed as a form of degeneration. It is a very rare disease. Usually only a portion of the placenta—perhaps one cotyledon—is affected, and this portion is greatly

increased in size so as to form a protuberance upon the uterine surface; hence its classification as a form of new-growth. On microscopical examination the protuberance is found to consist of enormously enlarged villi, the enlargement being due to myxomatous degeneration of the stroma accompanied by the formation of a certain amount of connective tissue of adult type. The chorionic epithelium is not much altered. The change is thus closely allied to that found in vesicular degeneration of the placenta, the chief difference being the formation of connective tissue in the latter, which greatly alters the gross characters of the affected part.

6. Tubercular disease.—In cases of chronic miliary tuberculosis, and in cases of advanced chronic phthisis, tubercle has been occasionally found in the placenta. The deposit occurs in the substance of the organ and is recognised by its well-known macroscopic and microscopic characters. Very few cases have been recorded, but in a considerable proportion no tubercular changes could be found in the body of the fetus, although the placenta was extensively involved.

7. Decidual Endometritis.—This condition is not strictly a disease of the placenta, for it affects the decidua in all parts, and usually commences at a period preceding the formation of the placenta. It occurs in two distinct forms, the *glandular* and the *interstitial* form. The former corresponds to the common or glandular form of endometritis of the non-gravid uterus; the decidua membrane is thickened and frequently shows small polypoid swellings upon its surface, but no actual inflammatory changes at all; the condition is rather that of glandular proliferation than of inflammation of the tissues. The interstitial variety is rarer; this is a true inflammatory condition characterised by round-cell infiltration of the whole membrane with intense congestion and many interstitial hæmorrhages. It has been shown that this form may be due to the gonococcus, which organism has been demonstrated in the affected membrane. Acute hæmorrhagic endometritis in pregnancy is also said to occur from cholera and the acute exanthemata.

The symptoms associated with decidual endometritis are pain and hæmorrhage during the first three months of pregnancy, and a marked tendency to abortion. If the ovum survives that period, atrophy of the decidua vera and reflexa will occur, and the danger is lessened. Diagnosis can only be established by microscopical examination of the discharged membrane. It is possible that morbid adhesion of the placenta in labour is a sequela of this condition. T. W. EDEN.

PLAGUE (πληγή; *plaga*, a stroke).—**SYNON.** : The Pest; Inguinal, Bubonic, Glandular, Oriental, Indian, Pali, and Levantine Plague; Oriental Typhus; Septic Pestilence; Fr. *La Peste*; Ger. *Die Pest*.

DEFINITION.—An acute infectious disease caused by a specific micro-organism which enters the body by the lymphatic or respiratory systems, the disease taking on the bubonic or pneumonic type according to the mode of infection. The bubonic form is the most common, is distinguished by fever, giddiness, debility, inflammatory swelling of the glands, external or internal, with serous or hæmorrhagic effusion from them into the surrounding tissues, more especially in the region of the groin, arm-pit, or neck, and occasionally accompanied by boils or

carbuncles, petechiæ or pustules. The pneumonic form is characterised usually by no glandular swellings but by acute fever, intense prostration, and slight cough with sputum tinged with blood and containing plague-bacilli. The disease affects the lower animals, especially rodents, and becomes epidemic among rats and mice.

ÆTIOLOGY.—The causal agent of plague is a bacillus discovered by Kitasato and later independently by Yersin. It is a short thick bacillus with rounded ends, staining with all the ordinary dyes, and taking the stain more easily at the poles than in the centre. In one form it is like a coccobacillus, in another like a diplo-bacterium. In a plague-patient it is found in the primary bubo and peri-glandular effusion before suppuration commences, in the blood in the more severe forms and later stages of the disease, and in the sputum in the pneumonic form of plague. In the corpse it is found in the organs and fluids of the body. It grows readily on the ordinary media employed for microbes. On agar the colonies show themselves as small opalescent spots with iridescent borders on reflected light. On gelatine they have the appearance of spots of white wax; they do not liquefy the gelatine. In broth there is after twenty-four hours a deposit at the bottom of the liquid which generally remains clear; after two days a film forms on the surface. In cultures the microbe may appear singly or in chains. Involution-forms of the bacillus are often to be found in the contents of the bubo and in the cultures from them, more especially in old and dry agar-cultures. Alongside of the coccobacilli and diplo-bacteria there are irregular elements resembling yeasts or algae in the form of swellings, gours, and bladders. These polymorphic forms are less easily stained than the typical coccobacilli and may be mistaken for impurities. On cultivating the microbe in broth, to which drops of clarified butter or cocoa-nut oil have been added, a very characteristic growth is obtained. This is called 'Haffkine's stalactite test.' From the drops of oil suspended on the surface of the nutrient medium the bacilli grow down into the depths of the liquid in the form of long threads producing the appearance of stalactites. When disturbed the threads fall in snow-like flakes to the bottom. This stalactite-formation is, under these conditions, peculiar to the growth of the plague-microbe, no other microbe having hitherto been found to possess the same property.

Inoculation of the bacillus into guinea-pigs, rats, and mice produces in them a septicæmia which proves fatal in the course of three to five days. At the site of inoculation there is a sanguineous effusion and the nearest glands are swollen and infiltrated; there is also general congestion of the organs, and hæmorrhages are common in the lungs, heart, liver, and serous cavities. The disease produced is similar to plague, in men the principal lesions being buboes, extravasations, and congestions in the internal organs. In rats and mice the glands and organs contain the bacilli in extraordinary numbers.

An accident in Vienna in October 1898 placed beyond doubt that the bacillus is the causal agent of the disease. Some cultures of the bacillus were brought to Vienna from Bombay by the Austrian Commission. With these cultures experiments were made on animals. Nearly a year after the return of the Commission, Franz Barisch, the

assistant of the Pathological Institution in Vienna, in charge of the experimental animals, fell ill with pneumonic plague. Dr. Mueller and two nurses became infected by Barisch before the disease was stamped out.

The Plague-Bacillus in the Lower Animals.—Usually an epizootic among rats or mice has been observed to precede or accompany an epidemic of plague in man. The rats leave their hiding places and come out into the open in great numbers. They appear very ill, are in a dazed or stupefied condition, their eyes are watery and bleary, their coats are partially deprived of hair, and they hobble about with difficulty, staggering and falling over one another. They lose their timidity for man in their evident desire for fresh air, and they fail in energy even to attempt to escape when approached. The sick and the dead are often heaped together. Examined after death the glands in the groin, axilla, or neck are found to be enlarged, congested, and agglutinated, the internal organs congested and the spleen and liver enlarged and full of bacilli. It is now established that these bacilli are identical with the plague-bacilli in man, and that the epizootic in rats or mice and the epidemic in man are identical in their causation. Though rats and mice are most liable to contract plague under natural conditions, other animals are susceptible, such as monkeys, bandicoots, squirrels, pigs, sheep, cats, dogs, and marmots. In the Transbaikal province of Eastern Siberia and in Eastern Mongolia, a species of marmot called the Tarbagan (*Arctomys bobac*) suffers from a disease which is believed to be plague and which is transmitted from the marmot to human beings.

MODE OF INFECTION AND DISSEMINATION.—Plague can be caused experimentally in a rat or a monkey by the single prick of an infected needle without the production of a visible wound. For this purpose it is sufficient to smear the bacilli over the shaved surface of a guinea-pig or other small animal; to rub them on an intact skin; to squirt them on to the conjunctiva or the Schneiderian membrane, or to feed the animals with cultures. It is believed that by similar means the plague-bacilli gain a lodgment in the lymphatic system of the human body, viz. by the skin, mucous membrane of the nose and mouth, especially of the tonsils, the conjunctiva, respiratory system, and occasionally by the alimentary canal. Two explanations are advanced as to the method of infection from rats to men. One is that the plague-bacilli on the rat infect men through wounds or scratches on the hand or feet. The other, which is propounded by Simonds, is that the fleas infesting rats, and which have been found to contain plague-bacilli, convey the infection to man as well as to healthy rats. The rats are infected from man by means of infected articles belonging to him. In 1896 a man from Bombay, whose wife had died of plague, brought her clothes home to his village, which had hitherto been quite free of plague. In a short time the rats in this man's house began to sicken and die, after which the inmates were attacked with plague and died, the man himself not being attacked till five of his relatives were affected.

CLINICAL FEATURES.—The clinical features depend more or less on the type of plague with which the patient is attacked. The principal types are the bubonic, septic, pneumonic, and ambulant. It not infrequently happens, however, that as the dis-

ease progresses, either the type becomes mixed, as when the pneumonic supervenes in a bubonic case, or there is a transition from one type to another, as from simple bubonic to septicæmic plague.

Premunitory symptoms are frequently absent, or, at all events, little marked. When present they consist of a slight indisposition for one or two days, with loss of appetite, heaviness in the head, headache, nausea and giddiness, weakness and sometimes pains in the limbs. There is nothing characteristic. The usual course is for the illness to set in suddenly, with rigors, high fever, severe headache, great giddiness, and nausea.

There are certain *general symptoms* which are common to all severe forms of plague. These are the peculiar expression of the face, the halting speech, the appearance of the tongue, and the staggering gait. The countenance in the early stages depicts anxiety and distress, in the later stages resignation and apathy, the vacant expression often marking the approaching dissolution. When delirium is present the expression may be one of terror. The eyes are red and congested, the conjunctiva being injected, but there is no photophobia. The patient has the appearance of being under an hypnotic, yet he is awake with eyes wide open. The speech is stuttering, thick, lisping, more or less staccato in character, and indistinct, often like that of a drunken man. When spoken to, the patient often begins a sentence and forgets to finish it. The tongue is early coated with a whitish fur, later it is dry, covered with a yellowish or whitish brown fur, except at the top and edges, which are irritable and red. The gait is staggering, owing to giddiness; there is a great tendency for the patient to fall. In many plague patients there is a desire to wander and there is a difficulty in keeping them in bed. This desire to wander is attended with great danger to the patient owing to the weak condition of the heart, causing a faint which may never be recovered from.

The Bubonic Form.—The ordinary bubonic plague is unlikely to be confused with other diseases. There may or may not be any premonitory symptoms, such as malaise and rigors. The disease usually begins with sudden fever, rising to 103° F. to 104° F., or even to 105° F., with nausea, vomiting, and severe and intense headache, mostly frontal or occipital in its seat. The eyes are suffused and congested, there is great giddiness and the tongue is furred, except at the tip and at the edges. None of these symptoms distinguish it specially from the onset of other acute infections; but, contemporaneously with the fever, or soon after its appearance, intense pain is felt in the groin, the armpit, or the neck, and at the seat of the pain one or more of the glands will be noticed to be swollen and to be particularly painful on pressure. In the course of twelve hours, or even in a shorter time, the swelling rapidly increases in size. This increase is caused by a periglandular effusion, which may be very profuse or only moderate in extent; and the bubo which is thus formed is somewhat doughy to the touch on the surface, and of a hard consistence in the deeper tissue. This bubo, with the other signs, is pathognomonic of plague. It is filled with plague-bacilli. The bubo may appear in the groin, armpit, or neck, and occasionally in the supra-trochlear or popliteal space. The most common site is the groin, and the next is the armpit; but more than one region may be affected, and often groups

of neighbouring glands on the same course of lymphatics become infected. The bubo varies in size, and consists of one or more inflamed lymphatic glands, from which exudes a sero-sanguinolent effusion which mats together the neighbouring glands into a hard mass of glandular tissue and extravasated blood, and infiltrates the tissues around, rendering them firm and oedematous. The oedema may be scanty or profuse. In cases which prove fatal early, the glands may remain hard and painful. In the groin the bubo may reach the size of a man's fist, and may extend into the iliac region, affecting the chain of glands in the abdominal cavity and forming a hard tumour, to be felt through the abdominal wall. In the axilla the bubo often occludes the axillary space, and the infiltration may become so extensive and organised as to form a hard mass which may interfere with the respiratory movements, or become a dangerous slough. In the cervical region the oedema may be so great as to be a serious danger to the patient, and by pressure to produce stridulous respiration, the tonsils and throat generally being inflamed. The position of the patient in bed is always one which tends to relieve as much as possible any tension on the bubo. There is no relation between the volume or size of the bubo and the amount of pain it causes. Consciousness is ordinarily retained, but even apparently with the most perfect consciousness the intellect is slow in answering questions, and the words or sentences are articulated in a hesitating manner, each syllable being pronounced slowly; or the speech is staccato in character, and uttered in an irritable and hurried tone. The temperature is not characteristic. It may reach its maximum in a day or two, or not until the fourth day. It is of a remittent nature, and in favourable cases begins to decline on the sixth and seventh day, and reaches the normal on the tenth day. The symptoms usually attain their height on the fourth day. Restlessness, with a desire to get out of bed and wander, is often a prominent symptom. Dyspnoea and delirium of a quiet or a noisy nature set in, followed in unfavourable cases by coma and gradual or sudden failure of the heart's action. The pulse, which is soft and easily compressible at the outset, becomes intermittent and dicrotic and often difficult to count, and the extremities become cold and clammy. Recovery, when it occurs, usually sets in about the sixth or seventh day, but no prognosis is safe in the early days of the illness. In addition to buboes there may be plague-carbuncles, which sometimes appear over the primary bubo, and secondarily to it or in other parts of the body, either before or after the appearance of the bubo.

The Septic Form.—The septic form of plague is a virulent type in which the lymphatic glands show no special enlargement during life, and consequently the bubo is absent or not readily recognised, but after death the glands are found to be generally affected, being somewhat enlarged and much congested. In this form of plague, the bacilli invade the blood in large numbers at an early stage and are easily detected during the life of the patient. The chief characteristic is its rapidity. The patient is profoundly affected by the amount and strength of the poison received. Usually ushered in with high fever, there is at times no power in the patient for reaction, and the temperature does not reach 100°. The countenance is pale and the expression apathetic. Extreme nervous prostration, muscular

weakness, delirium, picking of the bed-clothes, stupor and coma follow quickly on one another, and the patient dies on the first, second, or third day. In these cases there may be bleeding from the nose, kidneys, and bowels.

The Pneumonic Form.—This variety is very infective, and owes its infectivity to the fact that the sputum frequently contains almost a pure culture of plague-bacilli, which get on to handkerchiefs, clothing, bedding, and other articles of furniture, as well as on to the floor of the patient's room (Childe). The clinical symptoms are not typical of ordinary plague, and the disease is likely to be mistaken for bronchitis, broncho-pneumonia, or pneumonia. Pain, tenderness, and enlargement of the lymphatic glands in the inguinal, femoral, axillary and cervical regions—symptoms which are the most prominent external signs of bubonic plague—are absent unless the pneumonia is a complication of bubonic plague; and beyond cough and fever and a prostration which is exceptionally severe and far exceeding that likely to result from the small amount of lung-mischief discernible, there are few signs to raise suspicion that the disease is plague. The illness commences with a rigor and symptoms of general malaise, intense headache, nausea, vomiting, and pains in the limbs and body, followed by fever varying in range from 102° to 105° F. Cough more or less pronounced with dyspnoea sets in, and a quantity of watery sputum tinged with blood and becoming profuse as the disease advances is coughed up, as a rule, without effort. The sputum has not the glairy, viscid, rusty character of acute pneumonia, though on the clothes it may be readily mistaken for this. The physical signs are moist sounds at the base of the lungs and over the pneumonic patches, the pulse and respiration are rapid, but there is not the same disproportion in their ratio as is observed in acute pneumonia. The symptoms become rapidly worse, the patient becomes delirious, and usually dies on the fourth or fifth day or earlier. This type is the most infectious, and the most fatal. It corresponds with the black death of 1348. The sputum teems with plague-bacilli.

Ambulant or Mild Form.—The ambulant or mild variety of plague is even more likely to escape attention than the pneumonic, because of the slight constitutional disturbance which it may produce, and because it is often taken for some other disease. It is not discernibly infectious, the cases appearing to crop up unconnected with one another. And it is generally mistaken in the early stages of an epidemic for mumps, syphilis, the result of a strain, serofulous glandular affections, and malarial disease. The clinical symptoms are ill defined or well-defined fever, pain, tenderness and enlargement of the lymphatic glands in the groin, axilla, or neck, weakness, a tongue which is coated with a creamy white fur in the centre and is angry and red at the tip and edges. The eyes may be slightly congested, and the speech a little thick. This is the acute form which may only last a week, the bubo resolving rapidly or quickly suppurating. In the more chronic form, which may last two or three months, the bubo or buboes are indolent, and they undergo a slow process of suppurating and sloughing, constituting at times a serious drain on the general health of the patient producing anæmia and debility. These mild or benign forms of plague, often called 'pestis minor,' have been observed in nearly all plague-epidemics. The most recent example of this form

of plague unmixed with other varieties was the glandular sickness, as it was called, in Astrakhan in 1877. In this city an outbreak which affected more than 200 persons occurred without a single death and without being followed by the virulent form. Those affected suffered from fever more or less acute, with swellings and inflammation of the lymphatic glands, which in most cases ended in abscesses. Beyond the inconvenience and discomfort caused by the buboes, sometimes in the neck, sometimes in the armpit, and at other times in the groin, the general symptoms were not such as to prevent the patient from pursuing his ordinary avocations. About the same time there prevailed at Resht in Asia Minor, with which Astrakhan has more or less communication, the same type of glandular sickness along with a severe type of plague. In the next year, in 1878, the glandular sickness of Astrakhan appeared at Vetlianka, a village on the banks of the Volga and some distance from Astrakhan; but in a short time it was succeeded by a most virulent type of plague which assumed the pneumonic form and was of an extremely infectious and fatal character. The mortality at the height of the epidemic, which was limited in its extent, was 100 per cent. Mild cases intermixed with severe have been noticed in many epidemics. Mild cases of plague were observed in Calcutta, Bombay, Alexandria, and Oporto some time before the fatal cases attracted attention. In Calcutta, examination of the blood from the affected gland showed in some cases the characteristic bacillus, but in others none could be detected.

MORBID ANATOMY.—Decomposition of the corpses is not accelerated in plague, unless in the mixed forms, when streptococci are present. On the skin there are often small hæmorrhages, chiefly in the vicinity of the bubo, and on the head, arms, neck and shoulder; these hæmorrhages contain plague-bacilli. Hæmorrhages are often found in the muscles, chiefly in those of the abdomen and of the temporal bones, as well as in the muscles near the primary bubo; they contain polynuclear leucocytes and plague-bacilli. The characteristic appearance in a necropsy of plague is that of engorgement and hæmorrhage, nearly every organ of the body participating more or less. There is also parenchymatous degeneration and change in the heart, liver, spleen, and kidneys. Pathological appearances special to plague occur in the lymphatic glands, lungs, liver, and spleen. There is no other acute infectious disease which shows a similar multiple inflammation of the lymphatic glands. The enlargement of the lymphatic glands may be classed as hæmorrhage. The appearance of the glands depends on the type of the disease and the time at which death takes place. The primary bubo consists of a number of congested glands embedded in a mass of extravasated blood with extensive exudation into the adjoining areolar tissue, the larger blood-vessels being often involved. This infiltration contains polynuclear leucocytes and plague-bacilli. The glands in the bubo are swollen, varying in size from a bean to a walnut. They present a blackish-red appearance, and where cut into are found to be much congested and of a dark purplish colour. In cases in which death takes place later, the swollen and infiltrated glands show signs of commencing necrosis, while in cases in which death does not occur till the eighth or ninth day the necrosis has advanced, the paren-

chyma of the gland contains a yellow or reddish-yellow pus enclosed by the capsule, and the severe changes in the vicinity of the lymphatic gland have usually improved. The inner walls of the adjoining veins near a primary bubo are suffused in large patches, or are permeated with smaller hæmorrhagic points. The vein itself is surrounded by a copious inflammatory exudation, giving the tissue around a yellow gelatinous appearance. From the primary bubo a chain of swollen glands may be seen extending to other parts of the body.

In the septicæmic and pneumonic forms, in which the primary bubo is absent, nearly all the lymphatic glands are slightly enlarged and pink or dark red in colour; some may be engorged. In the pneumonic form there are, in addition to the general engorgement of the lungs, common to all forms of plague, several pneumonic patches, usually scattered but occasionally connected so as to form large areas together with extravasations on the pleura. The patches have the general appearance of red hepatization with an enveloping zone of intense congestion. The spleen is usually enlarged, and in the septicæmic and pneumonic forms of plague contains hæmorrhagic infarcts. Plague-bacilli are to be found in the different varieties of plague, in the lymphatic glands and the serous or sanguinolent exudation around them, in the blood, spleen, lungs, bile, urine, peritoneal fluid, and fluid of the brain. In the pneumonic cases they may be found only in the lungs and the bronchial glands.

DIAGNOSIS.—The difficulty of diagnosis of plague arises from the several types and forms which the disease assumes. In the epidemic in Bombay in 1896, some of the earlier cases with swollen glands and throat-symptoms were mistaken for diphtheria. In Jeddah, where pulmonary symptoms predominated, the earlier cases were taken for influenza; in Asencion for yellow fever and gastro-enteritis; and in other places the ambulant or mild form has been mistaken for non-venereal bubo, syphilis, malaria, parotitis, and even the effects of strain or accident. At the commencement of an epidemic the clinical symptoms are often obscure, and bacteriology alone can elucidate the question. When fever, the peculiar physiognomy, hesitating speech, staggering gait and condition of the tongue are met with, an examination should be made for the presence of buboes or of pulmonary disease, and this should be still further supplemented by an examination of buboes, blood and sputum for bacilli. By puncturing the bubo, even at its earliest stage, a small quantity of the gelatinous contents can be sucked out with a sterilised glass pipette guarded at the mouth-end by sterilised cotton wool. If the contents so obtained are then spread on a glass slide or cover-glass, gently heated as in the ordinary preparation of a microscopical specimen, coloured with carbol-fuchsin or gentian-violet, and then examined by a $\frac{1}{12}$ -inch oil-immersion lens, the field will be seen to be covered with coco-bacilli or diplo-bacteria, large numbers of them being more deeply stained at the ends than in the centre. No other disease with swollen glands presents microbes such as these. Their presence is sufficient to arouse the greatest suspicion at any time, and the material ought at once to be taken to a laboratory where the bacilli can be cultivated and the confirmatory tests applied. When plague is known to prevail in a country the discovery of bacilli by microscopical examination, combined with the clinical features,

is sufficient to make the diagnosis of plague a certainty. Should the case come under observation at a later stage when the bubo is softened, disintegrated and in a purulent condition, no bacilli may be found. Then the serum-test can be applied, the blood of a plague patient giving the specific agglutination-reaction to a culture of plague-bacilli. In pneumonic plague an examination of the sputum for the characteristic bacilli is the chief diagnostic test. In the fulminating or septic type of plague, the bacilli early invade the blood, an examination of which will show the microbe. In these cases several blood-preparations should be made. In all doubtful cases a larger quantity of blood should be taken from a vein. Occasionally the microbe is found in the urine, and in the milk of lying-in women.

In the case of the dead body the *post-mortem* appearances and the detection of the plague-bacillus in the primary buboes and the adjacent inflammatory connective tissues or in the blood, spleen, secondarily affected glands, bone-marrow, bile, urine, pneumonic patches, or dropsical fluid of the lungs, establish the nature of the disease.

TREATMENT.—*Curative.*—Good nursing and stimulants will do much for the patient, but the struggle is between the natural powers of the patient and the weakness or virulence of the microbes. Neutralisation of the toxin and destruction of the plague-bacillus without harm to the patient are the rational objects to be aimed at in treating a patient, and these are not obtained by any ordinary drugs. The only known method of attempting to deal with the problem is serum-therapy, and hitherto this method has varied considerably in its results, ranging from comparative success to complete failure. In twenty-one cases in which Yersin's serum was employed in China, the mortality was only 7 per cent. In Bombay it practically failed; while in Oporto out of 142 cases treated with the serum, only twenty-one of the patients died, which is equal to a mortality of 14.78 per cent. Possibly the differences are due to different methods of preparing the serum. The Chinese serum was obtained from the horse after injecting it with living and virulent cultures, but owing to the dangers attendant on this method, the Bombay serum was prepared by injecting dead cultures. The serum used at Oporto was obtained by injecting horses first of all with dead cultures, and then with living and virulent cultures. Greater success was obtained at Oporto when the serum was used in large doses and when employed intravenously. At the beginning of the illness, or as soon as the patient came under observation, an intravenous injection of 20 cubic centimetres of the serum was given, followed by two subcutaneous injections of 40 cubic centimetres each in the first twenty-four hours, and by subcutaneous injection of from 10 to 20 cubic centimetres or 40 cubic centimetres on the next and subsequent days until the temperature fell to normal and, in some cases, even for two days afterwards. Calmette and Salimbein report that no ill results ensued from these injections, further than an occasional erythema and articular pains, which were no more intense after the intravenous than after the subcutaneous injections.

As regards ordinary treatment the best results are obtained from good nursing, early confinement to bed, the maintenance of the recumbent position to

prevent syncope, careful feeding and general treatment to keep up the patient's strength and to prevent complications. To control the febrile symptoms and check delirium, ice-bags to the head, sponging of the body and the use of hypnotics, which are not depressants, are beneficial. To sustain the action of the heart alcoholic stimulants, ammonia, strychnine and digitalis may be employed. Complications are treated on general principles as they arise. The pain and tenderness of buboes are much relieved by the application of the ice-bag, which has also a good effect on the infiltration. Other applications such as glycerine and belladonna and poultices have been found useful. The bubo is opened when pus forms, but nothing is gained by too early incision. With the most careful treatment, however, the mortality varies according to the virulence and type of the disease and the race attacked. The mortality varies from 50 to 95 per cent. of those attacked. Europeans in recent epidemics have had even a lower mortality than 50.

Preventive measures in an infected house.—The precautions to be taken by the physician to prevent the spread of the disease in the family consist in notifying the case to the local authorities on whom rests the responsibility for carrying out the necessary measures to prevent extension from the house. Until the local authorities can take action, or if the circumstances of the case do not permit of removal to the hospital, the patient should be strictly isolated in the most secluded and best ventilated room in the house. A solution of perchloride of mercury (1:500) is recommended as a disinfectant. Sputum, urine, and excreta should be received in vessels containing perchloride of mercury of this strength, while a solution of half this strength may be used in vessels for soaking soiled clothes, disinfecting cups, spoons, &c., or washing the hands of the medical attendant and nurse after handling the patient. Nothing from the patient should be discharged down a watercloset or drain without being thoroughly mixed, and left in contact, with an abundant quantity of perchloride solution, otherwise rats in the sewers may become infected and carry the disease elsewhere. In the event of death, a sheet soaked in the strong perchloride solution should be wrapped around the body, and carbolic sawdust put into the coffin.

Of preventive measures for the medical attendant, the nurse and the relatives who may have close association with the patient, the most important is inoculation with Haffkine's plague-prophylactic. This prophylactic consists of a culture of plague-bacilli in broth which is kept in a state of growth for six weeks, after which it is submitted to a temperature of 70° C. for one hour in order to kill the bacilli. The mixture is then standardised and 2 to 5 cubic centimetres are employed as a dose. Subcutaneously injected into the arm or the flank, it causes in a few hours a rise of temperature to 102° F., sometimes to 105° F., intense headache and malaise and discomfort which usually lasts about 48 hours. At the site of the inoculation a painful swelling appears which necessitates rest for a day or two and remains evident for at least a week. Observations have shown that the inoculations with Haffkine's prophylactic not only sensibly diminish the chances of being attacked with plague, but also if attacked reduce the chances of death. The incidence of plague among those not inoculated has been shown to be in some cases four, five, and

nine times greater than among the inoculated, and the mortality among the inoculated only two-thirds that of the non-inoculated.

General measures of prevention are of two kinds, international and local. The international were formulated at a meeting of the Powers in Venice in 1897. Previous to that time many of the European nations adopted quarantine-rules framed upon those adopted in Venice in 1485. These consisted in the isolation in lazarettos of all persons from an infected port or country, and the detention and disinfection of goods and merchandise by exposure to the sun, for a period at first of 40 days, and later on for a shorter time. The rules were based mainly on merchandise being the principal carrier of the infection. As commerce increased the inconvenience of this system became extremely burdensome, and it became recognised that in the conveyance of disease infected persons were more dangerous than merchandise. Disinfection by steam and other means had also become more precise and could be effected in a few hours. To prevent importation by infected persons it was only necessary to know the period of incubation of the disease; to deal with the sick coming from an infected locality, to disinfect infected articles and to detain or keep under surveillance the healthy during a period which is considered to be the longest for the incubation of the disease. That period has been fixed at the Venice Conference at ten days. Rules were framed regulating these measures for arrivals both by sea and by land. Every Power also bound itself to notify to the others every outbreak of plague and to issue a bulletin weekly. A place is considered infected until ten days have elapsed after disinfection from the last case. Since the Venice Conference two other measures suggest themselves, based on further information obtained about plague. The first is the use of the protective power of Haffkine's prophylactic, and the second is the destruction of rats at infected ports and on the voyage on ships from infected ports, and in countries and localities in much communication with infected places.

The practical application of Haffkine's prophylactic to the crews of all ships and boats, large and small, coming from infected ports would probably largely reduce the danger of the importation of plague. Similarly, under certain circumstances and precautions the protection afforded to passengers or travellers desirous of crossing the frontiers into a healthy country would prove very valuable.

Local measures in an infected locality are based on the facts that early information is absolutely essential, that plague is due to a microbe, that the sick person is infective, that the period of incubation is usually not more than ten days, that the soiled personal linen of the sick may carry the disease, that rats distribute the infection, and that Haffkine's prophylactic is protective. Notification of sickness to the local authorities, isolation of the sick and the removal of the healthy from the infected house until it has been disinfected, and the destruction of all rats are measures of the first importance. For villages and small towns the removal of all the inhabitants on the infected area into camps is a very valuable measure; but for large cities this is impracticable. In such cases inoculation with Haffkine's prophylactic not only of the inmates of the infected house but also of the

inhabitants of the houses around the house, forming a zone of comparatively immune persons, is the most effective substitute.

By the destruction of rats and mice before plague is imported the chances of the disease developing into an extensive epidemic is very much lessened. If this has not been done, it is an essential part of the sanitary measures against the spread of plague that the destruction of rats and mice should be commenced as soon as plague is recognised. A systematic and regular inspection of houses in the poorer quarters and in the neighbourhood of the docks, warehouses, grain-depots, and other buildings which are likely to contain rats, is necessary. The measures of destruction are to be directed not only to the locality in which the rats are dying but also to the adjacent localities, and the disposition of the rats to migrate is to be borne in mind in order that on the first appearance of this phenomenon the rats coming into a healthy locality may be destroyed. Dead rats should not be removed until boiling water, carbolic acid or corrosive sublimate is thrown plentifully over their bodies to kill infected fleas on them which are liable to convey infection.

W. J. R. SIMPSON.

PLESSOR (πλήσσω, I strike).—A hammer-like instrument used in percussion, for striking the surface of the body, either directly or indirectly. See PHYSICAL EXAMINATION.

PLETHORA (πλήθω, I fill).—Fulness of blood. A condition in which the vessels of the body generally, or of any part, are over-distended with blood. See BLOOD, Morbid Conditions of; and CIRCULATION, Disorders of.

PLEURA, Diseases of.—The serous membrane which lines each cavity of the chest, and is so reflected as to cover the lung, is not infrequently the seat of disease. As in its anatomical and physiological relations, so also in its diseases, it presents analogies to the lining membrane of a joint. Its diseases may be of external or of internal causation. They may be considered under the following headings:—

Pleura, Injuries of, p. 1268.

Pleura, Inflammation of, p. 1268.

Pneumothorax, p. 1282.

Pleura, Dropsy of, p. 1285.

Pleura, Hæmorrhage into, p. 1286.

Chylothorax, p. 1286.

Pleura, Morbid Growths in, p. 1286.

Pleura, Surgery of, p. 1286.

1. Pleura, Injuries of.—These may be caused in several ways: (1) by blows upon the chest—and in this case there is usually at the same time an injury to the lung-tissue, the effects of which may overshadow the pleural lesion and its results; (2) by direct wounds with a knife or blunter instrument, or a bullet; and (3) by fractured ribs. With the surgical aspect of these cases we are not now concerned; the pleural consequences may be gathered from the following paragraphs.

2. Pleura, Inflammation of.—SYNON.: Pleurisy; Fr. *Pleurésie*; Ger. *Pleuritis*.

Pleurisy is defined as an inflammation of the pleura, of whatever nature and extent. Clinically and pathologically, pleurisy is the most common of the serous inflammations, and differs only in its accidents from inflammation of serous membranes elsewhere.

ÆTIOLOGY.—The causes of pleurisy, if local, may be obvious enough; if general, not so obvious. Of local causes the chief are wounds or bruises of the chest-wall; fracture of the ribs; caries of the spine; aneurysm; escape of irritating matter into the pleural cavity, whether from the costal side as in peritonitis or osteitis, or from the pulmonary side as in tuberculosis and in disease of the bronchial glands, or from the side of the abdomen as in gall-stone, hydatid, renal stone, ulcers, subphrenic abscess and the like. Foreign bodies, again, such as bones or coins from the œsophagus or larynx, have been known to find their way into the pleural cavity. Acute pleurisy, the result of local causes, is usually more or less proportioned to these in its severity and duration; pleurisy of general or systemic causation, on the other hand, though less regular in its career than pneumonia, has a certain uniformity. The general or systemic causes of pleurisy are now far better known. Between the ages of twenty and forty at least two-thirds of them are tubercular. That the majority of cases of acute, as well as of the more latent pleuritis (p. 1273), are tubercular is proved (1) by large injections of the serum into guinea-pigs (20 c.c. the first day, and as much again, freshly drawn off, on the second); (2) by cultures on peptone-jelly containing blood; (3) by tuberculin after the fever has fallen, or in apyretic intervals; (4) by Courmont's agglutinin-tests (p. 723), wherein the pleuritic serum of tuberculous cases is said to be more effectual than the blood-serum. In children the pneumococcus is often the cause, and sets up pleurisy without recognisable pneumonia. A chill may be the immediate cause of acute pleurisy; but chill must be associated with other factors. A rheumatic or gouty habit is suspected in some cases, and probably with good reason; in a few the influence of syphilis has been recognised. The depression of over-work or harass, the debility of previous illness, and the poison of malaria, are among the contingencies which enable this microbe or that to get a hold; and of these latter causes the B. tuberculosis is the most common. Tuberculosis confined to the pleura is the least menacing and the most curable of its manifestations; but the patient must be watched for some years afterwards. Acute pleurisy often occurs as a part, or as a complication, of other diseases. Thus in acute pneumonia it is rarely absent, and may run on, especially in children, to empyema; and in this, or in a serous effusion, the pneumococcus may be found, or its toxins only may have entered the cavity. It occurs, as cardiac valvulitis and pericarditis occur, in acute rheumatism, but less frequently. Rheumatic pleurisy is very acute, is apt to be bilateral, does not run to large effusion, and is of no long duration. Acute pleurisy following scarlatina is usually dependent upon a rheumatic or nephritic sequel; if it arise otherwise in this malady the pleurisy more often tends from the outset to empyema, as it does in typhoid fever also. Pleurisy arises sometimes after influenza and after measles, when it is probably due to infection from the lung; and, with or without obvious pneumonia, after enteric fever, when Eberth's bacillus may be found in the effusion. Not a few cases of empyema due to actinomycosis are on record. Pleurisy is also, like pericarditis, no uncommon consequence of disease of the kidneys, or of arterio-sclerosis of Bright's disease; when the effusion may be either dropsical or the product of a pleuritis. In septi-

cæmia and in pyæmia a low pleurisy often arises, as like effusions arise in the joints, and may be equally or more latent; or, again, it may be caused by the rupture of a pyæmic abscess of the lung or of a caseous gland into the cavity. The origin of the pleurisy in puerperal and other septic peritonitis is by way of the lymph-channels between the diaphragm and the pleura; and its supervision in some cases of abscess of the liver may receive a like explanation, though in others the passage is direct, by ulceration. Reversely, septic pleuritis may extend sometimes, though very rarely, from the pleural to the peritoneal cavity. Furthermore, a septic pleurisy may arise directly in a person exposed to insanitary conditions, when it is often preceded by sore-throat; such cases often lead rapidly to sero-purulent effusion, and are of the graver kind. Empyema as a secondary suppuration may appear alone; for instance, in such conditions as otitis media. Actinomycosis must not be overlooked, it is probably a more common cause of pleurisy and empyema than we are quite aware of. Acute pleurisy, when 'idiopathic,' is more often on the left side (three to two), and is rarely bilateral. When due to such infections as acute rheumatism or nephritis, it may be bilateral, though rarely of equal severity on the two sides.

Acute pleurisy is common at all ages; it is recorded often within the first six months of life; in babies it is easily overlooked unless there be abundant effusion, and not rarely even then. In children the symptoms are often very latent, neither cough nor pain being manifest, or pain may be referred to the abdomen; moreover, on account of the great mobility of the parts, there is little obvious displacement of viscera: yet pleurisy is really more common under one year than between the ages of two and five years. At the age of five it is frequent, but it reaches its maximum frequency in middle life (æt. 35-45). The younger the child the more readily the effusion becomes purulent; it is more often pneumococcal than in the adult. In many cases the suppuration extends to the pericardium, but this is more common perhaps in older children. Some pleuritis, of course, may often result from extension of pericarditis, but it is usually unimportant. Cases of simple inflammatory pleurisy have been recorded in persons beyond threescore years of age; but in aged persons it is rare, and presents little reaction or pain. The male sex is more often affected than the female, in the ratio of about seven to five; the difference may be due to the greater exposure of men to changes of weather; pleurisy is often said to vary in prevalence with pneumonia, and to be most frequent in April. With the advance of our knowledge of the subject, however, the number of cases of pleurisy attributed to 'chill' is growing less and less, while the class of tubercular pleuritis, favoured by chill or other depressing conditions, is receiving larger and larger accessions. Under one form or another pleurisy is credited with about 2 per cent. of the deaths in England, and with about 1 per cent. of the deaths of patients in public hospitals.

MORBID ANATOMY. (By Dr. Sidney Martin.)—The anatomical characteristics of inflammation of the pleura do not differ from those of inflammation of the pericardium or peritoneum, with the exception that purulent inflammation is more common than is the case with the pericardium.

In *acute dry pleurisy* the surface of the membrane

is congested, and may show ecchymoses; while on the surface is a false membrane composed of fibrin entangling leucocytes. Some slight effusion no doubt occurs, being the liquid left after the coagulation of the fibrin with the plasma. As a rule, red blood-corpuscles do not exude in any considerable number. Proliferation of the pleural cells occurs, with the formation underneath of granulation-tissue, containing new vessels. The costal pleura, as a rule, is affected sooner and more severely than the visceral. Acute dry pleurisy may resolve without adhesions being formed to any extent. When, however, it occurs between the lobes of the lung, adhesions commonly result. The formation of adhesions—that is, of fibrous tissue binding together two layers of the pleura—does not differ from the formation of fibrous tissue, as the result of any other form of inflammation. The fibrin is absorbed, and the leucocytes for the most part disappear. The fibrous tissue is formed from the fibroblasts of the granulation-tissue, which, with the new vessels, is derived from the permanent tissue beneath. In the process of healing of dry pleurisy some account must be taken of the causes of the inflammation. Thus, in non-tubercular pleurisy, the events are as above described; but when the disease is tubercular a greater thickening of the layers of the pleura occurs, and adhesion almost constantly results.

Pleurisy with effusion is another phase of inflammation of the pleura. All the features of dry pleurisy are present, but, in addition, the cavity contains a greater or less amount of liquid effusion. The amount of this varies considerably. It may be as large as 100 or 150 ounces. The fluid sometimes contains floculi of fibrin, or meshes of fibrin may traverse the fluid as it lies in the pleural cavity. When withdrawn during life the fluid is yellowish or slightly green, alkaline, and with a specific gravity usually between 1016 and 1023. Microscopically, it contains a few leucocytes, some endothelial cells derived from the pleura, and a few red corpuscles. The leucocytes and endothelial cells are frequently swollen. The fluid usually coagulates spontaneously after removal during life, forming a soft gelatinous clot. The coagulation either occurs at once, or may take several hours. It is hastened by the addition of blood. The average composition is as follows: 3·5 to 5 gr. per cent. of albuminous substances, 1·5 to 3 per cent. consisting of serum-globulin, 1 to 3 per cent. of serum-albumen, and 0·02 to 1 per cent. of fibrin. The extractives present are chiefly sugar and cholesterin; but urea, uric acid, leucine, and tyrosine have also been found. *Hæmorrhage* may occur in a pleuritic effusion, due either to the tubercle-bacillus or to one of the other bacteria causing the disease. The fluid removed by the aspirator is distinguished from pure blood by its watery character, and by its specific gravity, which is lower than that of blood. In acute cases the blood is slightly darker in colour, but differs from the blackened bloody effusion observed in some cases of malignant disease of the lung. *Purulent effusion, or empyema*, is a specific result of certain of the micro-organisms causing the pleurisy. The anatomical changes in the pleura are practically the same as previously described, but the fluid which is exuded contains an enormous number of the leucocytes characteristic of pus. These are chiefly the polynuclear neutrophile leucocytes, and are seen in various degrees of degeneration. The character of the purulent effusion varies

in different cases, from a serous effusion containing an excess of leucocytes, whereby it is made turbid, to a thick creamy pus of a slightly greenish tint. The *process of repair* in pleuritic effusion takes place first of all by the absorption of the liquid effusion; subsequently by the changes which have been previously described as occurring in dry pleurisy. Adhesions commonly result, and retraction of the side of the chest, due mainly to the inefficient expansion of the lung. When the pleuritic effusion is tubercular in origin, great thickening of the pleura results during repair, and a greater retraction than in non-tubercular pleurisy. In empyema repair occurs after the pus has been evacuated, though it is possible, from the occurrence of calcareous plates in the pleura, that a small collection of pus, usually tubercular in origin, may be absorbed, and repair take place, without its being evacuated. In repair of empyema after an opening has been made in the chest, great thickening of the pleura results, owing to the abundant granulation-tissue that is formed, and complete repair may not take place for many months or not at all, a sac being left exuding a small quantity of pus from greatly thickened walls.

Putrid effusion and empyema occur by direct infection from the lung (as in cases of gangrene or pneumothorax); when the pleura communicates with the œsophagus (as in cases of cancerous stricture); when the empyema results from a subphrenic abscess; and in some cases of septicæmia, with infarction of the lung.

In cases of large effusion the lung is found compressed, and often bound down by false-membranes extending from the walls of the cavity. In adults the lung is usually found in the space between the vertebræ and scapula; it retracts primarily by its own elasticity, withdrawing itself upwards, inwards, and backwards, and may be compressed to one-quarter or even one-eighth of its normal volume; it is then flattened, leathery, bloodless and airless, and will sink in water. In recent cases as the pressure subsides, the lung may, and generally does, recover much of its former volume; and it is surprising how fully the lung may re-expand in spite of false-membranes, bands, and prolonged compression. Nevertheless, either complete or partial adhesions or bands of connective tissue generally remain indefinitely after acute pleurisy; and for the most part do no direct harm. If the lung fail to re-expand to any extent, the deficiency is made up by the falling inwards, partly of neighbouring soft parts, and partly of the chest-wall. But on the other hand, false-membranes and bands as well as the fettered lung are, or may become, the seat of degenerative processes, and especially of tuberculosis and of fibrosis. During empyema the contact of pus promotes ulcerative and septic changes in the lung, as it may do likewise in the vertebræ, ribs, and other neighbouring parts. Thus the pus, finding for itself a passage in the direction of least resistance, may pierce lung or thorax, and establish a pulmonary or costal fistula.

Subpleural ecchymoses, though often accompanying evidences of inflammation, are not always caused by pleurisy. They occur in deaths of children after broncho-pneumonia and diphtheria; but there is usually a patch of pleuritic inflammation upon and co-extensive with them. As the punctiform ecchymosis of Tardieu, they may also be found on the heart, pericardium, and thymus

glands, and are not by any means peculiar to deaths by pleurisy; but are probably always associated with obstruction to the entrance of air into the lung.

In all cases the position of the heart and other viscera must be observed, and the chambers of the heart, the pulmonary artery, the inferior vena cava, and other vessels examined for clots. In empyema a careful examination of the body for secondary abscesses must be made, not forgetting the brain.

CLINICAL CHARACTERS AND VARIETIES.—For mere convenience pleurisy may be divided into six varieties, as follows: (α) *Dry*; (β) *Acute*; (γ) *Diaphragmatic*; (δ) *Quiet, with large effusion*; (ε) *Tubercular*; (ζ) *Fibroid*. Each of these requires separate consideration. Effusion, when present, may be serous or may be purulent, or a serous effusion may change into a purulent; but since antiseptic precautions in operative procedure have been perfected, this perversion comes more rarely under observation.

(α) **Dry Pleurisy.**—This is so called not because it is attended by no effusion, but because the effusion is so slight as to escape notice. Usually, it results in an adhesion of the opposite surfaces of the membrane. It may not be revealed by any sign or symptom during life. Adhesions, more or less extensive, due to this process, are very often found after death. Dry pleurisy may occur alone, or as a complication of irritative changes in neighbouring tissues, as in the lung or chest-wall. It is common in Bright's disease. It is rarely absent from the surface of an infarct, and may give rise to substantial and recurrent effusion of an aseptic kind. Such is the obscure origin of many a pleurisy in elderly persons. Pain or pyrexia, more or less fugitive, may accompany dry pleurisy; but in many cases they pass unnoticed. Should attention be drawn to the chest, friction may generally be detected at some stage. An obscure pain in the chest or loin, or a frequent teasing dry cough, may at times be traced by the close observer to a patch of dry pleurisy in some part of the chest. A friction-sound due to such a patch may be transient, or may be audible for many weeks. Dry pleurisy, as such, is not revealed by radiography. It is supposed that some of the pains in the chest which accompany phthisis are due to the intercurrent of dry pleurisy; but they are as frequently myalgic (from cough) or neuralgic. On the other hand a friction-sound, as in the upper scapular area, may be the first sign of tubercular infection, and not infrequently of a rapid case. Dry pleurisy, with its resulting adhesions, is rarely directly injurious; indeed, it is rather a safeguard when any destructive process, such as phthisical ulceration, threatens to bore into the pleural cavity. If it fail, and morbid matters escape into the cavity, acute pleurisy and pneumothorax are the probable consequences. Dry pleurisy often ends in but slight thickening, the two pleural surfaces adhering without much increase of substance. In other cases the thickening may be considerable, but this probably would indicate some more persistent irritation, such as we find, for example, in those dense coverings which often invest the apex of a lung in chronic phthisis. Tuberculosis apart, the remoter consequences of dry pleurisy are rarely of importance. In some instances it may limit the chest-movements, or may tie the parts so as to cause transient pains, described as dragging or tightening. Such pains are usually referred to the

sub-axillary or sub-mammary regions, and may be really annoying; more often they do harm by ministering to needless fears. A generally adherent lung is usually retracted, and is more liable to tubercular disease, active or fibrous. It is said that in rare cases hypertrophy of the heart has resulted from embarrassment of its action by pleural bands. Dr. Bowditch told the writer that he had seen this result twice at least.

It is useless to prescribe treatment for a disease which escapes observation, or is but a secondary event in the course of more serious processes. Where dry pleurisy is found, and is doing harm by exciting cough or otherwise, the best practice is to place two or three light blisters in succession over the affected part, and to watch the patient closely, especially his temperature and his urine.

(β) **Acute Pleurisy.**—Acute pleurisy generally appears as an important illness. In the large majority of cases it is unilateral, and perhaps never are both sides attacked simultaneously. Bilateral cases are rheumatic or tubercular. It sets in with fever, pain, embarrassment of the breathing, and cough—sometimes catarrhal, usually reflex. These symptoms bear no certain proportion to each other. The fever has no very characteristic type, but is what is known as a symptomatic pyrexia. Speaking generally, a rigor of onset would indicate pneumonia, but there is often a succession of lesser chills. Nor are there any very definite stages of increment, but rather a daily fluctuation of remittent, more rarely of intermittent type, with evening rise; the elevations not often reaching and rarely exceeding 40° C. (104° F.). Peter stated that the temperature of the affected side is higher by 1°–3° F.; falls with effusion, but rises again if this become purulent. At first the blood-pressure is high, the pulse being small and hard; after the first onset the pressure falls, and the pulse becomes dirotic. A quick pulse is characteristic of pleurisy in most of its forms, even if there be no fever; for it is by this frequency rather than by more powerful contractions of the heart that blood-pressure is kept up fairly well even when large pulmonary areas are restricted, or the auricles compressed. The arterial pressure therefore should be closely watched in cases of copious effusion. In severe cases the difference of pressures in inspiration and expiration may be very perceptible, and occasionally the pulsus paradoxus has been recorded. As the effusion reaches its height, the fever in acute pleurisy gradually recedes, unless the case approaches to the form (δ); and the fever may subside, may fluctuate, or may drift into hectic. The pain is often very characteristic, but at other times is variable, and even delusive. Most commonly it appears as a stitch in the side, about the level of the false ribs, which is intensified by inspiration and cough. The deep breath when partly drawn is cut short, as if with a stab, while the face of the patient is wrung with an expression of sudden distress. Such inspirations are, however, instinctively avoided, and may have to be called for by the physician, so that the face may speak rather of apprehended than of actual suffering; in either case the expression is a telling one to the practised observer. The fixed *alae nasi*, which are dilated but do not oscillate as in some other kinds of dyspnoea, the parted lips, the bright eye of fresh fever, the cheeks flushed but not congested as in pleuro-pneumonia, the preoccupied and apprehensive expression, the posture semi-erect,

slightly bent forward and toward the affected side, the shallow breathing, the fixed chest, the hand on the side, the curt speech, the stifled dry cough, make up a clinical picture often seen, and easy of recognition. It is a curious fact that these symptoms of distress are generally more marked in a robust patient, or one previously healthy, than in the weakly, ailing, or cachectic. The pain, however, may wander from the lateral or antero-lateral aspect of the lower ribs, and appear in the hypochondrium, or even on the opposite side. At other times it may become more diffused, and play upon the brachial plexus, darting from the clavicular and scapular districts to the upper chest, shoulder, or arm. This radiation is more common in the diaphragmatic variety (γ). In some bad cases of empyema the pain is very distressing and prolonged. Severe abiding pain in a case of pneumonia is suggestive of purulent pleurisy. Whatever the treatment, we look for some relief of pain, cough, and conscious dyspnoea on the third or fourth day. The respirations, however, may still range above the normal rate, from the mechanical interference of increasing effusion, or of this increase combined with oedema of the open parts of the embarrassed lung, and perhaps of its overworked fellow. About the end of the week, be it more or less, the pleurisy has run its course, and, in favourable cases, the effusion has attained its maximum; the urinary, gastric, and other glands regain their normal activity; and convalescence, with absorption of the exudation, is to be looked for. If in three weeks signs of considerable effusion remain, the case is passing into a chronic form. Thus far, then, the disease is painful rather than dangerous, death in the first week of ordinary acute pleurisy being practically out of the question. Malignant cases of pleurisy, however, occur, in which the temperature may be 40° or 41° C. (104° , 105.4° F.), the pulse reach 140, the tongue become dry and brown, the prostration be excessive, and the exudation run promptly to pus—pus which may be fetid, although without obvious reason. Except as complications of septic and other diseases, such cases are rare, and even after free evacuation of pus by incision, they are too often fatal. An important instance of recovery from such a case has been reported by Gairdner and Buchanan. By certain signs in the chest we know the height to which the effusion has flowed in the cavity, and we await its ebb. Usually, in a day or two, some fall is noted, and in favourable cases this ebb runs quickly at first, and afterwards more slowly as the products become denser. The false membranes undergo fatty degeneration and slowly vanish, though some remnant is usually to be detected for an indefinite time after the patient is about. If the pleura thickens much, as it frequently does, the signs of it may be carried to the grave. Probable as is the favourable result in strong persons, yet it is not to be too lightly promised even to these. Too often when we are awaiting the ebb we find a new flood, the level of the fluid rises into the upper chest, and the patient, who hitherto has lain on the sound side to avoid pain, turns on the affected side to give full play to the open lung. This flow may recur with or without renewed fever, but is generally attended by a proportionate increase in the rate of the pulse, and diminution of its volume and pressure. These events are more common in streptococcal infection. Coincident with the diminution of blood-pressure

is a diminution of the urine, which, probably, had become more abundant as the fever ceased. That the changes, both of pulse and urine, depend upon the effusion is shown by the rapid recovery of both when fluid is artificially let out from the pleura; the pulse then falls in rate and increases in tone under the finger, and the urine soon becomes more abundant. A little albumen is sometimes present during the time of pulmonary obstruction (see ALBUMOSURIA). Under ordinary circumstances a renewed flow of urine is indicative of pleural absorption; but the case may pass on into the form (δ), or into an empyema. It is hard and often impossible to tell when the full chest contains serum and when pus. Hectic fever may exist with serum, but if with fever be associated tenderness or subcutaneous oedema of the affected side, change of countenance, loss of appetite, wasting of flesh, failure of strength, thrush, diarrhoea, or any of them, and the more if there be any inherent constitutional frailty, a preceding acute specific fever, or a septic infection, we must fear that tuberculosis is present, or the fluid turning to pus. In the later weeks or months of an acute pleurisy which has not ended in resolution, death may threaten, and may not be averted. In some cases, as after scarlatina or pneumonia, and especially in young children, the effusion may be purulent from the beginning, and a fatal result may be feared even in the earlier days of the malady. Under ordinary circumstances, however, in healthy persons who have been carefully treated from the outset, and who have not been exposed to septic influences, we expect to have to deal with an effusion moderate in quantity and stable in quality, which rarely remains at its height more than two or three days; and in three weeks should be largely absorbed. We shall not therefore be too ready to tap in such cases. In other cases again, happily rare, acute pleurisy, with remittent fever, continues for many weeks. Effusion in these cases may not be very rapid, but recurs gradually after the removal of moderate quantities; or it may not seem to need removal. The signs are simply those of acute pleurisy, but resolution does not take place, or is indefinitely deferred. Death may result or the patient may slowly recover. After death may be found evidences only of active simple inflammation, compression of the lung, and sero-fibrinous exudation. The other side, and the rest of the body, may be quite healthy. To these cases the name *relapsing pleurisy* might be given; of those which are not tubercular, the specific nature is unknown. When inflammation falls upon both pleurae, it generally falls also upon the pericardium, and such cases are terribly dangerous. Even if moderate in degree in each part, yet taken together the embarrassments of the patient become very grave, and death may be imminent. It is important to give relief by puncture, pleural at first at any rate, as early as possible. A purulent pericardial effusion may attain great dimensions, and if it be associated with pleurisy, and the case be not seen early, or not followed during its course, diagnosis of it may be impossible. The probability of the association must not be forgotten.

(γ) **Diaphragmatic Pleurisy.**—Diaphragmatic pleurisy is not essentially different from the preceding, but the symptoms are often peculiar. If the inflammation be, as it may be, confined to the diaphragmatic area, then the ordinary physical

signs of pleurisy with effusion are either absent, at any rate at first, or so ill-marked as to puzzle the inexperienced practitioner. The onset may be as acute as in ordinary pleurisy, but the distress is greater and more alarming. The practitioner is surprised and perplexed to find a person, in whom he can discover no important organic defect, in an agony as it were mortal. The presence of pain shooting from the lower ribs of one side suggests pleurisy; but the ribs on both sides play with perhaps more than normal freedom, and no physical signs may be audible, unless it be that the practised ear may detect a want of breath-murmur at the base of one lung, and, after the first day or two, it may be, two finger-breadths of dulness there. Still no friction may be heard, and it seems impossible at first sight to associate signs so slight with clinical phenomena so alarming. For the patient may be as one having a clot in the heart, or a sudden perforation of the pleura, so terrible and so absorbing is the strife for inspirations which never satisfy, so keen the dread of any handling which may interfere with the one permanent need of sitting erect, and of keeping the upper respiratory muscles in full play. These inspirations may range from forty to fifty in the minute, or may even run with the seconds, except only when cut by a hiccup or a heaving of the stomach. To this are added the suffering of pain which shoots through the waist to the back, or darts round the shoulder-blade and collar-bone into the shoulder, and a sense of a fatal grip. There may be tenderness round the insertion of the diaphragm on the affected side, and perhaps in the course of the phrenic nerve over the anterior scalene muscle. The normal character of the heart's action gives confidence to the physician; abdominal breathing, however, is lessened, sometimes on the affected side only, and any pressure upwards upon the diaphragm is resented. All these symptoms finally lead to the conclusion that acute inflammation has partially attacked and so far paralysed or inhibited the diaphragm, without extending on to the pleura above; but the diagnosis is of course the easier if pleurisy be discovered elsewhere in the chest. A patient thus attacked seems to be in no little danger, but recovery may be anticipated if there be no mischief below the diaphragm. Fortunately the malady, thus circumscribed, is far less common than ordinary pleurisy, and indeed may be called rare. As stated above, however, inflammation of the diaphragm may complicate ordinary pleurisy, and produce both the pains in the brachial plexus and the excessive and paroxysmal dyspnoea. An empyema or other effusion lying between the diaphragm and the pleura, as also between the lobes of the lung, may be out of reach of direct diagnosis (unless the needle be used), and may have an obscure commencement. A persistence of fever, especially if hectic in character, would dictate very searching and suspicious examinations of the patient and the repetition of exploring operations so long as any doubt remains. Diaphragmatic pleurisy may arise in inflammatory diseases of the liver or spleen, or of neighbouring parts of the peritoneum. If peritonitis extends, as it not infrequently does, through the diaphragm, it may set up mischief in both pleuræ. But, perhaps because of some special activity in the liver and its capsule and ligament, it comes through more frequently on the right side. In all such cases the base of the chest must be examined re-

peatedly. In cirrhosis of the liver pleuritic effusion is prone to appear, and may be hæmorrhagic, even when the peritoneal fluid is not so. It is because of subdiaphragmatic disease that the death-rate of diaphragmatic pleurisy is so high. See DIAPHRAGM, Diseases of, p. 387.

(δ) **Quiet Pleurisy with Effusion.**—This form of pleurisy is often regarded as the sequel of acute pleurisy; but if we except the cases in which pleurisy, at first sthenic, afterwards follows the asthenic tendencies of the patient, and those in which acute pleurisy has been treated with neglect, we shall find that this form begins not sharply but quietly, and is indeed often unnoticed until the chest is laden with fluid. If the patient suffered pain it was too slight or too indefinite to ensure attention; the fever, unmeasured by the thermometer, escaped observation; the chest, slowly invaded, accommodated itself to circumstances until the fluid had nearly filled the cavity; and the patient may only have sought advice because of dyspnoea on ascending hills or stairs. A quick eye may detect in him an expansion of the *alæ nasi*; or, indeed, may see that the patient is breathing at double, or nearly double, the normal rate; or at the outset a sensitive patient and a vigilant physician may fully perceive the remittent—almost intermittent—fever, the indefinite pain and the encroaching effusion, and may lessen the evil by timely interference. As a rule, where effusion is large the patient lies on the affected side, thereby escaping the pressure of the fluid upon the mediastinum, and enabling the sound lung to have free play. This decubitus is not invariable, and is avoided if the affected side be painful. With pyogenic change in the effusion the patient may turn off the affected side, as this change is sometimes accompanied by a renewal of tenderness to pressure. When the effusion has come on very gradually, the patient is often able to lie on either side indifferently, and indeed to walk about. Occasionally a large pleural effusion may cause some dysphagia. Let the reader then remember that pleurisy running on to large serous effusion is very often quiet in its approaches; and, if in our examination we are content with raising the patient's shirt in front, it may be overlooked until matters become extreme. Even if the effusion be purulent, its accumulation may be equally rapid or equally silent; being silent when it is the further change of a serous effusion, being silent and rapid when it comes as pus almost, or quite, from the outset, as in septic and infectious diseases, and in children. If acute pleurisy drift into chronic pleurisy, which after the lapse of a month without absorption may be said to be the case, the fever, which may have vanished for a while, lights up again fitfully at times, and fresh attacks of inflammation take place in the pleura and in the new membranes. With this there are also renewed outpourings of serum, which sometimes increase so rapidly as to put the patient in imminent danger of death by syncope. The fever in these stages is often hectic in character, so that the presence of hectic alone does not prove the effusion to be purulent. Quiet effusive pleurisy is very uncertain in duration. Should the effusion be not excessive, and remain serous, months may elapse—nay, even years—before it is absorbed; and the absorption may be gradual, or may be deferred for awhile, and then completed more quickly. It is needless to say that even so favourable a result as this cannot do away

with the injury which the chest must suffer from being waterlogged for so long a time. It is well known that a lung thus compressed is very prone to become the seat of tuberculosis. Very frequently, however, the effusion is sero-purulent or purulent, even from the beginning; and if left to itself finds an exit gradually by many minute ulcerated spots through the spongy lung, and so is expectorated gradually; or, by an opening into a bronchial tube, rushes with a sudden and copious discharge into the mouth. The expectoration in the former case, if profuse, is usually inoffensive, and muco-purulent; in the latter, the gush of pus, often stinking, is sometimes so great and so sudden as to swamp the lungs and threaten or even produce suffocation, especially if it occur during sleep. In the latter case we have usually to deal with a subsequent pyopneumothorax, which, if left to itself, may ultimately heal, but is as likely to end in death by slow hectic and marasmus. The issue is scarcely more promising if the pus find its way outwards between the ribs; and this it may do by a direct opening, usually in the fifth space towards the front, or by a sinuous opening: or it may gather between the ribs and skin, forming there a large superficial abscess—'*empyema necessitatis*'—the tension of which varies with respiration and increases with cough, and this of course more or less readily as the communication is more or less direct. These changes in tension aid us in distinguishing such issues of the pleural cavity from local abscesses of the chest-wall. In pleuritic effusions pressure is rarely negative, often highly positive, being high in young elastic chests, with large effusions, low in old persons and with small effusions. The chest in cases of spontaneous opening is imperfectly emptied; the issue is not in a good position, and septic poisoning is hardly prevented; yet a lingering illness is by no means always to be cut short by operation. Or the pus may find its way into the opposite pleura, thus doubling the empyema, and even such patients have recovered; or into the pericardial or peritoneal cavities, though such terrible events are fortunately very rare; or it may burrow between the tissues and appear at distant places, and thus may mimic psoas or other sinuous abscess. A cure of empyema by reabsorption is said to be possible, but, except in the case of small encysted collections, the possibility must be a bare one.

In some cases the fluid may rise in twenty-four hours from the angle of the scapula to the clavicle—an obliteration of breathing-space far more perilous in its rapidity than a more gradual one to which the system slowly adapts itself. Pleurisies with large effusions have no definite course, for, even if serous, absorption is difficult, and so far as it occurs is too often compensated by renewed febrile movements, with renewed effusions.

(e) **Tubercular Pleurisy.**—In considering the relations of pleurisy to tuberculosis we have to deal with four classes of cases: (1) Those in which the pleurisy is tubercular in its origin and development. (2) Those in which one or more attacks of pleurisy, not known to be tubercular, have preceded phthisis. (3) Those in which pleurisies spring up here and there in the course of pulmonary phthisis. (4) Those in which tubercle arises in the exudations of a pleurisy supposed to have been simply inflammatory.

These states may be considered reversely, beginning with the last. Tuberculosis is a far more

frequent cause of pleurisy than 'chill,' or the 'rheumatic' infection; but, apart from tubercle in other parts, rarely destroys life directly, and is not often found alone upon the *post-mortem* table. Tubercle, however, often betrays its presence in the pleura before it manifests itself elsewhere, so that the occurrence of pleurisy should always excite suspicion, and this the more if patches of it spring up here and there without much resulting effusion. As in the peritoneum, tubercular inflammation may cause large effusion, or may be of the more adhesive kind; and many sero-fibrinous or sero-purulent pleurisies are of tubercular causation, direct or indirect; the fact of recovery by no means proves the non-tubercular nature of the case. No difficulty arises in deciding upon the nature of intercurrent pleurisies in pulmonary phthisis, though often these are not tubercular in a strict sense. The difficulty is to detect tubercle in a pleurisy apparently primary and simple, which appears to progress towards recovery, or often, indeed, to reach it. On the other hand the temperature, if it has fallen, may rise again fitfully, and the pulse quicken, yet without much evidence of empyema or of any returning effusion. Then a patch of pleurisy on the other side, or a sign of mischief at the apex of a lung, may betray the character of the relapse, and in the pleura, or the false-membranes, are the caseous or softened residues of the first crop. Most difficult of forecast are the pleurisies of the first class, which, however painful or profuse, end in recovery which seems complete, and is no doubt often permanent. But such convalescents should for years to come, or for life indeed, beware of depressing causes, which might re-awaken the disease. For the temperature may begin to be hectic; a short dry cough occur; signs of phthisis appear in the lung; and the end come in the too familiar way. The tubercle-bacillus is very rarely found in the products of pleural paracentesis, save indirectly by inoculation-experiments; in most cases it is enclosed in the granulations. But for some reason or other tubercular effusions are apt to be malodorous, and when purulent are insidious and tedious. The physician must then regard with anxiety and precaution all pleurisies, however frank in attack, however happy in their resolution, or however free the subjects may seem to be from any taint or character of consumption.

(f) **Fibroid Pleurisy.**—Sometimes as a primary affection, but more often as an ultimate consequence of an acute pleurisy, the membrane slowly thickens, and, allying itself with a like overgrowth of the connective elements of the lung, increases at the expense of the proper pulmonary tissue; and, gradually contracting after its kind, stifles and destroys a great part of this organ. Fibroid pleurisy generally begins at the base, and the visceral layer may increase until it may form a dense leathery covering even one-third of an inch in thickness. Tuberculosis is generally, perhaps always, concerned in the process, but may be difficult to detect, even after death. See LUNG, Cirrhosis of, p. 909.

Hæmorrhagic Exudation.—Pleurisy may be attended by hæmorrhagic exudation—that is, by exudation mixed with more or less blood, the loss of which may be exhausting—even in some cases of empyema. These cases, though sometimes very acute, are usually chronic; the hæmorrhage depends on bleeding of the new vascular tissues, and, moreover, may be aggravated by some other

abnormal state of the patient, such as scurvy, carcinoma, arterio-sclerosis, or tuberculosis. It seems probable that hæmorrhages do not suppurate unless micro-organisms gain access to them, and tend to do well if other conditions are favourable. A small quantity of blood may give to a body of serum a sanguineous hue, but the specific gravity would remain low. After paracentesis a hæmorrhagic may quickly pass into a clear serous effusion.

Pneumothorax.—This is a term applied to that condition in which, owing to perforation into or from some open channel, air finds entrance into a cavity, which is or becomes emphysematous. At the same time pus is evacuated. The lung may in measure re-expand, or in neglected cases may be irrecoverable; the chest-wall falls in more or less, according to the rigidity of the ribs and other conditions in the case. On the entrance of air dullness gives way to clearer and lower notes, except in dependent parts still occupied by effusion; and the pitch will vary with the thickness and density of the false membranes within, and the degree of pulmonary expansion. See LUNG, Perforation of, p. 936; and section 3, Pneumothorax.

PHYSICAL SIGNS.—Throughout the stages of pleurisy inspection may tell us that the movements of the unaffected side are excessive, especially if compared with those of the affected side which hang back, either by the warning of pain indirectly, or directly by effusion which stops the play of the lung. This laggard movement is often to be noted also in the abdomen on the same side, especially in diaphragmatic pleurisy. As the effusion increases the chest may or may not be seen to bulge beyond its true lines; the intercostal spaces are usually, but not always, flattened up to the level of the ribs, and the form of the affected moiety of the chest becomes more cylindrical, as is best shown by the cyrtometer. The xiphoid cartilage is often displaced away from the side affected, to right or left of a string stretched from the manubrial notch to the symphysis pubis. The diaphragm may be so thrust downward and forward as to cause a fulness in the epigastrium, and a slight depression of liver or spleen; with large effusion there may be bulging even of the supraclavicular space; and the outline of the affected side measured on the transverse submammary line will usually measure more than on the healthy side. Half an inch is an important difference, seeing that in a young adult the other and healthy side in overwork expands about half an inch or more beyond the normal, and will fall again as the compressed lung expands after paracentesis. The right side of the chest in health is usually larger, especially in muscular men. In rapid effusions the skin is often obviously stretched. Œdema of the skin on the affected side is occasionally present, and, though not decisive of pus, is less common in serous effusions. It is confined to the affected side, but sometimes extends beyond the chest-wall. A great thickness of it may be attained very rapidly, especially with the more foul sero-purulent exudations. The state of the veins of the neck and of those upon the chest will be noted. Much enlargement of these would suggest intrathoracic tumour with or without fluid; as would inequality of the pupils, or other evidence of solid pressure within. Cyanosis will appear if the yielding right auricle is under much external pressure. Clubbing of the fingers (on both hands) may often be seen in old cases of pleuritic effusion not necessarily phthisical; and the feature

recedes if the patient advances towards recovery (p. 548). The presence or absence of a heart-beat and its position, if visible, must be noted. If fluid be in the left chest, a diffused pulsation in a tumid epigastrium often replaces the proper apex-beat, or the heart-beat may be felt (or heard) towards the right breast; if in the right chest, this beat may be detected towards or upon the left axillary line. The mediastinum is more readily displaced to the right than to the left side. In some cases of limited but complete dullness in the anterior and inferior region of the left chest, when it may be difficult to decide between fluid and pleuro-diaphragmatic adhesions, M. Jaccoud says the distinction may be made in some cases of adhesion by observing traction upon the lower ribs and spaces on forced breathing—the spaces being retracted on inspiration, and the ribs drawn towards the median line. In some rare cases of empyema the whole of the affected side so pulsates as to simulate a large aneurysm, a phenomenon which has not yet received a satisfactory explanation (see Diagnosis, p. 1277). Finally, in large effusions there is often some prominence in the hypocondrium of the same side; the nipple shifts farther from the sternum, and the shoulder-blade is thrust somewhat out and away from the spine. All these displacements and changes of shape are brought about more readily in young subjects; and in women more readily than in men. Yet in children, again, on account of the elasticity of the parts, we find proportionately less visceral displacement. The diaphragm in the female is more readily depressed than in the male, and the right side of it more readily than the left. In chronic cases absorptive and atrophic changes tend to bring about a shrinkage of the affected side; the thoracic and intercostal muscles waste; and the ribs touch and lap over with corresponding flexure of the spine, and great elevation of the heart or liver. Such a deformity, happily rare, may, indeed, if the lung be obliterated, be permanent.

Palpation will help us to estimate differential expansions; to find the heart's beat; to ascertain whether liver or spleen be displaced, and so forth. By the hand we may sometimes detect the creaking of friction, of which, indeed, the patient himself is sometimes aware; and we may verify the imperfect expansion of the side or abdomen, the levelling of the intercostal spaces up to the ribs, and possibly fluctuation in the former. The most distinctive sign to the hand, however, is the loss of the vocal thrill, which is arrested by fluid effusion. This is normally more distinct on the right side and over the lower two-thirds of the chest. Here effusions usually first accumulate, and loss of this thrill is almost pathognomonic of them; for it occurs besides only with those intrathoracic growths which by their size or position close the bronchial tubes, and in certain rare and peculiar cases of pneumonia with blocking of the tubes. Unfortunately, at a time of need the voice may fail in a woman, a child, or a very fat person, to awaken a thrill even in the normal parts. Sometimes the limits of the thrill may give a gauge of the height of the effusion, or may locate an adhesion. Above the limits of the effusion or near the spine the thrill is often more distinct than it is over the corresponding part of the sound side; and it is said to be present at times, or even increased, when, after re-absorption or withdrawal, but a very thin layer of fluid lies between the lung and the chest-wall. In such a case some

dulness would persist. Return of the fremitus is an early sign of recovery.

Percussion reveals a higher or duller note over the whole extent of the fluid; but it cannot tell us the amount of fluid present, as the level of this depends on the state of the lung and of intrathoracic tensions and displacements. Moreover, during absorption, dulness depending upon thick false-membranes cannot be easily distinguished from that due to fluid. Such membranes may diminish vocal fremitus also. When fluid is present in quantity, say in a layer of an inch in thickness, the note struck is dull, as if struck upon the thigh; and the stricken finger receives a peculiar sense of loss of resilience in the chest-wall. Extreme degrees of consolidation, however, may rival fluid in these characters. If the pleura seem full of fluid, but the lung be not much compressed, direct percussion by the finger-tips will give a very dull note and a sense of resistance, while a stronger mediate percussion will bring out a note of somewhat lower pitch. A very small effusion lying below the lung may be detected by comparative percussion on forced inspiration; as on the affected side, fluid will rise in the cleft between the pleural surfaces, which on the normal side will be filled by expanding lung. In an early stage of effusion the level of the dulness may perhaps vary a little with the position of the patient, if its quantity be moderate, and it be unconfined by adhesions. Gravitation, however, helps us less in inflammatory effusions than in hydrothorax. In pleurisy, indeed, the fluid may be sacculated, or even suspended by adhesions above the base-line of the cavity. When free and moderate in amount, the level of the fluid usually sinks a little, in a parabolic curve, from back to front. This curve, formerly attributed to the direction of the shrinking of the lung, is now known to be due to gravity together with the viscous quality of an inflammatory exudation. It is not seen in patients who have not taken to bed, nor in hydrothorax. Inflammatory effusions tend in a few days to 'set' in the position determined at first by gravitation, as the patient was then more upright or more recumbent. In acute pleurisy the fluid does not often rise above the third rib in front; but in quiet effusive pleurisy the whole moiety of the chest may become very dull upwards and across to the opposite parasternal line; and there may be dulness as well as bulging in the supraclavicular space. When the chest contains a good deal of fluid, but is not full, percussion over certain areas may actually give a lower note, or possibly a higher 'cracked pot sound,' but in either case with a tympanitic clang. These tympanitic areas are of three kinds—(1) where a thin layer of fluid lies over expanded lung, as, for instance, at an intermediate zone between clear lung and copious effusion; (2) where distended air-cells compensate cells closed in another part; (3), where, the chest being full of fluid and the lung collapsed, percussion in the neighbourhood of the trachea and large bronchi causes vibration therein. Tympany, when present, is ordinarily under the clavicle (Skodaic), but it may be distinct at the upper level of the effusion behind, say below the spine of the scapula. If the tympany be due to the third cause, it may have something of the cracked-pot quality. In some cases the tympany is decreased on inspiration and increased on expiration. The detection of tympany under the clavicle may mislead the unwary into a belief that the healthy side is morbidly dull; on

the other hand it is an invaluable help to the physician who takes it as a hint to look for mischief below: in rare cases this may be extensive consolidation without fluid. Dulness due to an elevated liver may be distinguished by the removal of its boundaries on inspiration, and by its anterior rather than posterior disposition. In mere hydrothorax the lung generally rises more readily, and the diaphragm and other parts can usually be made to move in respiration. On the left side the tympany of the gastric area becomes obliterated, unless the stomach be retained by adhesions, which has occasionally given rise to doubts.

Auscultation, before any dulness appears, usually reveals the respiration at the part to be defective in quantity, rhythm, or quality; and a friction-sound may be audible. Defective inspiration at the outset is due to checking of that act; afterwards it is due to the false-membranes and effusion which favour retraction of the lung, hinder conduction of sound, and ultimately silence it. A friction-sound, if generated, may be fugitive and escape the observer, or the embarrassed chest-movements may fail to give it distinctness; on the other hand it may be even palpable. If the heart modify its rhythm a pericardial rub may be simulated, but friction will vary with respiration. Usually it appears at the outset, and disappears as effusion separates the surfaces; it may reappear as the fluid is absorbed. Frequently it accompanies one only of the respiratory movements—the inspiration or the expiration. Friction is most frequent in the subaxillary area, and is often compared to the creaking of new leather or of parchment. The 'friction of onset' may last but a few hours, and except in dry pleurisy, or that of some cachexies or of septicæmia, is rarely abiding; returning friction is often coarser than the initial, and may continue for a longer time, even for weeks. It is not uncommon to find a friction-sound abiding so long as the patient can be kept under observation; these permanent rubs often seem to be without much practical importance. Diminished breathing and friction, if effusion gather, are followed by intermediate phenomena due to thin layers of fluid. These are bronchial breathing, bronchophony, and ægophony. Ægophony is nearly always heard near the root of the lung under the scapula; it has the character of a tremulous bleat, and when once heard is not easily forgotten. It may be reinforced by bronchophony, especially if there be solid lung below. Its presence is pathognomonic of fluid;¹ but it is so often absent that it is of little practical value. As fluid increases, these phenomena sound more distant, or give place to silence; and as fluid gathers first at the bottom we often find silence at the base; bronchial resonance, amounting, perhaps, to ægophony, at mid-lung; and either weak or compensatory breathing at the apex. We may also meet with curious inverse changes in the physical signs, notwithstanding an increase of fluid, if at first this be spread over a partially expanded lung, and afterwards accumulate below it as the lung retracts, or as intrathoracic pressure tells on the mediastinum. Thus dulness may actually recede with an increase of fluid, or on the other hand may rise upwards as with a diminution of fluid the re-inflated lung descends. For these and

¹ Some modifications of pectoriloquy have been described by Baccelli and other writers as means of distinguishing between pus and serum in the pleura, but they cannot be relied upon.

other reasons it is very difficult to gauge the ebb of intrathoracic effusions, or accurately to ascertain their behaviour after tapping. The cyrtometer and the tape are perhaps the most useful aids at such stages. If heart or liver had been displaced their return would be significant. The affected side usually retracts more or less as the fluid is removed. In some chronic cases where the lung does not re-expand well, and false membranes contract, the falling in may be considerable; and, as the muscles of the affected side are apt to atrophy, may appear to be even more than it is (*see* PNEUMONIA, CHRONIC). In children, bronchial breathing and bronchophony often, but not always, persist throughout. Where the lung is unbound, gauging is of course the more easy. When the chest is quite full of fluid, there may be no response to ear or hand; but even in such cases a faint but distinct respiratory *souffle* is not infrequently audible almost down to the base; and may deter the inexperienced physician from puncture. Breathing, more or less tubular, and some resonance on percussion are generally to be heard over the root of the lung in the vertebro-scapular space. The gradual formation of a pulmonary fistula may in some cases be revealed some days or hours before evacuation, by the presence of crepitations in the upper part of the affected side. As air re-enters the lung, respiration is at first weak, and accompanied by crepitations which are probably due to degrees of œdema; sometimes this œdema is severe and persistent, and abundant crepitations, with watery expectoration, last for many days. This is not uncommon after paracentesis. Respiration then improves gradually, and reinforces itself as the lung expands and clears. The pneumonia of children being often lobular, the discovery in them of bronchial breathing and bronchophony is suggestive of fluid, though lobar consolidation is of course not uncommon in them. In the other lung there is usually a slight general lowering of the percussion-note and a compensatory increase of breath-sound; if the effusion be large enough to compress the opposite lung, the percussion-note may be much lowered. About the lower half of this overworked lung some crepitations may be heard, especially if the right auricle be compressed. Finally, the stethoscope will aid in ascertaining the position of the heart. Skiagraphy is of use in doubtful cases. Serous effusions give a darker shadow than the normal lung, and purulent effusions are darker still. The position of the heart and diaphragm also may be perceived; so that in subphrenic disease (*see* STOMACH, PERFORATION OF) the X-rays may prove very helpful. The lung also may be perceived lying upward, inward and backward, or otherwise displaced, as in cases of adhesion or of fibrosis.

DIAGNOSIS.—Pleurisy in the earliest stage may be beyond diagnosis; or a pain may be felt, which, setting aside other causes, may be due to pleurisy or to pleurodynia. The pain is often referred to the loin or abdomen, thus leading to suspicion of disorder elsewhere. Prolonged anæmia and leucorrhœa would suggest pleurodynia. A comparison of local temperature in the two sides seems untrustworthy, but the presence of fever would make us strongly suspicious of pleurisy. It must not be supposed that diagnosis in this early stage is unimportant. Few errors are more common than the attribution of pleuritic pains to pleurodynia; the pain disappears, effusion slowly accumulates, and

mischief and peril, perhaps hardly remediable, may be the consequences. The careful observer will listen repeatedly to the chest for a friction-sound, which is conclusive. Fever might be present with pleurodynia, and an immediate diagnosis impossible, unless something characteristic in the stitch and start on deep inspiration betray a pleurisy. Neuralgic and inflammatory diseases of the walls of the chest are not likely to give rise to much misunderstanding. It is said that a pericardial may be mistaken for a pleuritic friction-sound, but the distinction can rarely be difficult; a pleuritic patch overlying the pericardium may chafe with a cardiac rhythm, but the sound would be modified by the respiration. A difficulty is more likely to arise in distinguishing between a pericardial and a circumscribed pleuritic effusion; still, with the inter-current respiratory rhythm, this can hardly be insuperable. In rheumatic fever, scarlatina, and streptococcal diseases pericardial may accompany or ensue upon pleuritic effusion, and when the latter is on the left side, and is abundant, the former may be confused with it, especially as in such cases the pericardial effusion is apt to lie in the back part of the sac, and thus elude discovery. The practical lesson is to remember the likelihood of pericardial effusion, and its frequent latency.

In the later stages of pleurisy, when effusion is abundant, its diagnosis may at times be difficult. Under ordinary circumstances complete and extensive dulness, with loss of sense of elasticity in the chest-wall, of respiratory sound, and of vocal thrill, makes diagnosis easy; or if there be resonance below the clavicle, its high pitch is characteristic of fluid below. But there may be no such resonance, and the voice may fail, or fail to set up thoracic thrill. Moreover, vocal thrill and respiratory murmurs may vanish likewise in intrathoracic tumours. Thus the diagnosis between exudations, pulmonary consolidations, intrathoracic growths, subphrenic mischief, and combination of pleuritic fluid with any of them, is sometimes difficult. If empyema coincides with acute pneumonia and overlies it, a differential diagnosis may be unattainable; but absolute dulness, cessation of vocal fremitus, distance of respiratory phenomena, and a recrudescence of fever after a crisis would suggest exploratory puncture. In the writer's experience inordinate severity of the pain in the side is characteristic of gravity in the nature of the pleurisy. The heart may be dragged over by the retraction of the opposite lung. Leucocytosis, absent in simple serous effusion, would suggest empyema, pneumonia, or tuberculosis. In small effusion the limits of posterior dulness may be raised by a few forcible inspirations, probably due to a re-expansion of collapsed lung; or if there be no effusion they may be reduced as the inflated lower border of the lung reoccupies the cleft behind an elevated diaphragm. Two fingers' breadth of dulness at the base signifies about 15 oz. of effusion. If the voice be ægophonic we decide upon fluid, but if bronchophonic we may distinguish the 'sniffing and metallic' bronchophony of consolidation from the weaker and more diffused bronchial quality in pleuritic effusion. The same may be said of the respiratory sounds. If perchance the dulness and breath-sounds should vary with the position of the patient, fluid is clearly manifest. Circumscribed effusions, such as

encysted empyema, if they do not displace the heart, are at times physically indistinguishable from a like extent of chronic consolidation, or of abscess in the lung. Such effusions, though usually basic, are by no means always so. Retained by adhesions and sometimes multilocular, they may occupy the upper and anterior region, any part of the middle region, or strips or irregular districts in any direction; or, again, they may be interlobar, and in this position perhaps to be recognised by the obliqueness of the strip of dulness. Interlobar effusion is usually purulent, and often is revealed only by breaking upwards, when it behaves as a pulmonary abscess. It may be said, in general terms, that a permanent dull area remaining after an acute pleurisy or pleuro-pneumonia is most probably due to an encysted empyema, but not always. In such cases fever, if it recur after a pneumonic crisis, is a tell-tale symptom, but it may be entirely absent, and, unless clubbed fingers or radiography help us, there may be little to decide the nature of the disease. Obsolescence may result; still such a collection is very likely to work mischief sooner or later. The difficulties of distinguishing the more bulky effusions from pulmonary consolidations are not often great. In the former the intercostal spaces may be bulged, and the moiety of the chest enlarged; but these signs occur in some rare cases of 'caseous pneumonia.' On the other hand, consolidation very rarely reduces the lung to silence, though this may be the case; if so, the bulk of the half-chest would in all probability be lessened, but so, on the other hand, may it be in a chronic effusion. The upper line of dulness in effusion is a very inconstant curve, determined by the posture of the patient during the time of its initiation. The fact is, many chronic cases can be diagnosed only by the needle; and it should be noted that, even with the needle, many punctures may be needed before deciding against fluid. Practically speaking, however, as Wilks has said, 'chronic basal pneumonias,' unless of subphrenic origin, are almost always pleuritic effusions. Pulmonary abscess too is rare, and it may be separated from the visceral pleura by a layer of pervious lung. If it lie near that surface it may be indistinguishable from an empyema; and this is the more embarrassing as adhesion is very often wanting, even in abscesses of some standing, when exploration by the needle is not without danger. It is difficult to do more than to urge the physician before plunging in the needle to consider the importance of a full review of the history of doubtful cases. Between intrathoracic tumours and large pleuritic effusions a difficulty is found only in those cases in which the tumour is confined to one moiety of the chest; this, however, is not very uncommon, especially in aneurysm. If fluid effusion accompany tumour, there may be subtympantic resonance under the clavicle. In favour of fluid alone are the absence of enlarged veins; the equality of hydrostatic displacement of organs, and the absence of signs of local pressure, of retarded arterial wave, of inequality of pupils, of peculiar sputum, and of enlarged glands.

A curious pulsation, of uncertain explanation, is sometimes seen in left-sided empyema, or any effusion, if tense enough, and very rarely in pneumonia alone (Graves). In the case of effusion, indeed, the lung must be indurated and must be tied down to the structures about the heart. This pulsation must not be mistaken for an aneurysmal throb. If

fluctuation be felt in the intercostal spaces, the disease, in part at any rate, is a fluid effusion. Sometimes a gastric, hepatic, biliary, renal, or other subphrenic abscess, making its way by a sinus, occupies some part of the pleural cavity, or excavates an adherent lung. Such an abscess usually extends rapidly, but it may encyst itself, and remain latent or quiescent for months; or it may excite an effusive pleurisy in the remainder of the cavity, so that two effusions co-exist in one pleura. Thus, serum may be withdrawn from one part of a pleural cavity, while a collection of pus remains encysted in another—a collection, even when suspected, perhaps very difficult of discovery. From it of course pulmonary fistula may take its origin, and pus from the same central source, whatever the origin—for instance, hepatic, perihepatic, or perigastric abscess, renal stone, appendicitis, caries of bone, and so forth—may in part issue from the urethra or rectum, and in part issue from the mouth. Pleurisy, even without effusion, at the bases of both cavities is very suggestive of a sub-diaphragmatic origin, and, still worse, of the possible presence of pus in the mediastinum; though tubercular peritonitis is often associated with tubercular pleurisy which may be bilateral. When such abscesses contain air, but not by way of the lung, they may have originated in some perforative disease of stomach or bowel; in these cases, however, there will be little evidence of retracted heart or of increased intrathoracic tension, and the affected area will rarely approach the apex. Dulness from disease below the diaphragm, but encroaching on the thoracic space, may be displaced downwards by a deep inspiration. Radiography is proving itself a valuable aid in exhibiting such displacements, as of mediastinum, heart and diaphragm. Radioscopy in some cases is remarkably efficient; as when on the screen the movements of the diaphragm are rendered visible, and the varying level of fluid, so that in succussion the splashing may be seen. It is stated that in puncture combined with the use of a manometer, when the canula is in a cavity beneath the diaphragm, inspiration is attended with an increase and expiration with a decrease of pressure, being the reverse of that which occurs when the canula lies in the pleura. In peripleuritic abscess tension is of course low, there is no pressure on neighbouring organs, percussion-dulness is less profound, free expansion of the lung may be detected, and axillary glands may be enlarged. It is said (Bartels) that pus from interstitial abscesses is of higher specific gravity (1040) than from large cavities (1028-1030). Pleural effusions arising by direct absorption from puerperal and like forms of peritonitis are usually themselves also septic.

The diagnosis between serous, sero-purulent, and purulent effusions is usually decided by the needle. Even in the case of recent limited basal effusions the physician must suspect empyema. Signs, such as oedema of the skin, said to be indicative of pus, are by no means trustworthy, and the chest-wall may be retracted over pus as over serum. By radiography no doubt the transition of serous into purulent effusion is perceptible, but in ordinary practice this means of diagnosis is rarely available or necessary. Collections of pus or serum in peculiar situations may then be fairly easy to discover, or difficult, or impossible. We shall remember that such collections contained or suspended by adhesions may

occur, and moreover may be multilocular. In the most important of these—the effusion between the lobes of the lung, which is usually purulent—radiography may be very helpful. If the case be followed from the beginning the changes may be noted along the lines of the fissure. Often, however, it is first revealed by perforation into a bronchial tube. If the liability to collection here, or between lung and diaphragm, which is far from uncommon, be present to the mind of the physician, the symptoms and course of the case will usually indicate to him the duty of careful and even repeated exploration in these areas.

A large pulmonary cavity might be taken for encysted empyema with fistulous opening into a bronchus; and here again, as a pulmonary fistula rarely gives rise to tubular breathing, unless the opening be very large or communicate with a secondary cavity, diagnosis by direct signs alone might be impossible. The history of the case and the state of the other lung would be important factors in decision. In another class of cases the distinction between chronic phthisis and pleurisy may be difficult—in those, that is, in which there is some old dulness and retraction of a part of the side, dropped shoulder, atrophied pectoral muscles, weak respiration, indefinite crepitations, and more or less fever. Absence of bacilli and lung-tissue in the expectoration, and the health of the other side, help to exclude all but that fibroid phthisis which is usually a result of chronic tubercular pleurisy. Tubercle-bacilli are rarely to be found in the effusion of such pleurisies and do not survive long in pus; but a positive result may be obtained on the guinea-pig if 20 cc. be injected (*vide* paragraph on *Ætiology*). In the pleurisy of typhoid fever, the effusion, of whatever kind, has a high agglutinative power. In children, enlargement of the spleen, with extension upwards and backwards, has occasionally simulated effusion at the base of the left lung. Hepatic hydatid may rise up to the third rib; it has usually a convex upper border, but it may be distinguishable only by the properties of the fluid drawn off, which is non-albuminous and contains hooklets. The distress and orthopnea of very painful pleurisies—of diaphragmatic pleurisy more especially—may simulate cardiac thrombosis. The state of the pulse alone usually suffices to abate the fears of the physician. A hæmorrhage into the pleura can be distinguished from a serous or purulent effusion only by a careful survey of all the history and symptoms; the direct physical signs help us but little.

PROGNOSIS.—The prognosis of pleurisy, even tubercular, apart from pulmonary tuberculosis or malignant disease, is generally favourable; in tubercular pleuritis it is usually favourable as regards the current illness. In children the prognosis is especially good, although in them the effusion is often purulent (usually pneumococcic). Chronic pulmonary fibrosis is happily rare, and in the particular case the chance of such an event almost vanishes. When the effusion is large it is the less favourable the greater the quantity, the slower the absorption, and the more obstinate the recurrence, especially if the patient makes no way. Coagulation in the serous exudate on standing is favourable, a heavy deposit of leucocytes is less favourable. Irregular or remittent fever and loss of flesh are of grave significance. Signs of hyperæmia and œdema in the working lung must be anxiously watched, especially if an empty radial artery, scanty

urine, and other evidences of high venous pressure be added. When the chest is full, prognosis, apart from operation, is bad. In severe and rapid cases the other lung may become œdematous and congested, bloody and frothy sputa appear, carbonic-acid poisoning become evident in the blue lips and lethargic brain, the pulse slip away, the heart fail, and the extremities grow chill; or, again, death may be sudden, as if due to syncope. Sudden death in pleurisy with effusion has been attributed to torsion of the great vessels, due to the dislocation of the heart; but such torsion does not occur, and the accident may happen in large effusion on either side of the chest, or, indeed, with effusion of moderate degree, and may be due to thrombosis of the pulmonary artery, to pericardial effusion, or to compression of the right auricle. Operation raises the hope of recovery greatly—so much so as to put the chances largely in favour of rapid recovery in ordinary cases. The earlier the relief, the less the damage to the parts and the better the hope of rapid amendment. In bad cases prognosis will be the less favourable the more potent the adverse conditions; and in secondary pleurisies the prognosis may depend upon these other conditions. In old people operation is still useful; the conditions in them unfavourable to operation are still more unfavourable to absorption; but especial care must be taken to draw off the fluid very slowly, and to watch the circulation. In empyema prognosis is graver; unless operation be performed death is probable, either by syncope before the matter escapes, or by exhaustion, chronic septicæmia, or secondary abscesses, during a long period of incomplete drainage of the chest. If operation be submitted to, the prognosis is favourable, save in the worst cases; the earlier the operation is performed the better. Cases of the thick, greenish pus of the pneumococcus, seen more often in young children than in adults, do better than the seropurulent of streptococci. But these characters are not conclusive, and must be checked by cultivation. Sometimes in bad cases, such as those of a septic or gangrenous kind, death is preceded by much dyspnoea, due to cessation of all abdominal breathing. A very small gangrenous patch in lung or pleura will betray itself by its unmistakable odour. Although primary tubercular pleurisy with serous effusion is hopeful as regards immediate prospects, tubercular empyema is far less so; indeed, its nature is often to be inferred mainly from its vicious course. Effusions secondary to pulmonary tuberculosis, when they are serous, as is ordinarily the case, should be left alone as long as possible. Among the deferred dangers are amyloid disease—not a common event, but possible in cases of necrosed rib or other bone or of long and exhausting drain, and phthisis or more general tuberculosis, happily rare, prevented, it may be, by the density of the false-membranes. The presence of albumen in the urine does not preclude recovery nor forbid prompt operation. The precise bearing of age and sex upon prognosis cannot as yet be decided. Experience indicates that it is more hopeful in cases under ten years of age and above twenty years. Bowditch says that full pregnancy is no bar to thoracocentesis.

As regards duration, an ordinary case of inflammatory pleurisy will last from ten days to a month, according to the degree of effusion and the rate of re-absorption. Chronic cases with large effusions

may last any length of time, rarely less than three months. If tapped, the fluid may not return, or may not return after a second tapping; in such a case recovery will be more rapid. Empyema, opened under the most favourable conditions, is often months and sometimes years before final closure; antiseptic operations and dressings have much shortened the mean duration. If left to itself, an empyema usually opens through the lung or externally. In the latter case the issue is most commonly about the fifth interspace anteriorly, but may appear elsewhere. Drainage in such cases is very incomplete, and although some relief is attained, the patient nevertheless drags on, it may be for years, with a permanent fistulous discharge. Operation in such cases is discussed further on. Of double empyema many cases have ended in recovery, especially in children.

It must not be forgotten, however, that simple pleuritis, so often themselves tubercular, may be the forerunners of phthisis, even years afterwards. The occurrence of a pleurisy in a young person is always then an anxious matter. The experienced physician will call to mind many cases in which a pleurisy, to all appearance wholly recovered from at the time, was followed even before many months had passed by definite signs of phthisis. Prognosis, therefore, must concern itself with the ultimate as well as with the immediate issue, and with the appropriate warnings. In the course of pulmonary phthisis a decided attack of pleurisy means or makes mischief, even if quickly got under.

Hæmorrhagic effusions, if kept aseptic, often do well; their gravity depends upon the cause. Of secondary purulent foci which may take rise in empyema, suppurative meningitis and abscess of the brain are the most calamitous, and, unfortunately, not the least common. Nystagmus, or other muscular spasm, may put us on our guard; and the fluid of lumbar puncture may be examined for microbes (see p. 903).

TREATMENT.—(1) *Medicinal.*—Dry pleurisy requires little treatment. In some cases, as in chronic phthisis, it may cause distress, and, if so, may be relieved by spongiopiline and laudanum, or other such soothing measures. In the cases in which a troublesome cough is caused by a patch of chronic dry pleurisy, the cough and pleurisy alike may be relieved by the brief application of blisters. In acute pleurisy, on the other hand, much depends upon treatment at the outset; in few maladies is early attention better rewarded. Our aim in the beginning is to diminish pain, inflammation, fever, and the tendency to excessive exudation. With or without treatment, the pain usually passes off in forty-eight hours, or thereabouts; nevertheless it is very acute while it lasts. In sharp cases, occurring in healthy persons, we may put on six to twelve leeches according to the age, sex, or condition of the patient, and these may bleed freely into a large poultice. This measure, if adopted at the very outset, diminishes the pain, the fever, the exudation, and the duration of the case. When the bleeding has ceased, the chest should be firmly bandaged, and so soon as the state of the leech-bites will allow of it the affected side should be as firmly strapped. This, by giving rest to the part, will favour resolution and resorption, and some patients, though not all, find much comfort from it. Respiration, on the other hand, favours effusion, as does exercise in inflammation of a joint. If called to a

case after the first brunt is over—say after a lapse of forty hours—it is better to omit the leeching, in order that the strapping may be applied at once. It must be applied on the method laid down by F. T. Roberts. Dr. Roberts applies the strapping in all cases from the outset; but the writer's experience is in favour of early leeching in suitable cases. Some physicians recommend that an attempt be made to subdue the local inflammation by the application of ice-bags, but the results of this method do not seem very satisfactory. In addition to local measures, such medicines as the following are required: A combination of compound ipecacuanha powder and antimonial powder (āā gr. iiss) may be given every six or eight hours, for two or three days. When pain is severe, the subcutaneous use of morphine is to be recommended, in doses of one-eighth to one-fourth of a grain. In addition to the above powder, either full doses of solution of acetate of ammonium (ʒij-ʒiv for an adult), or ten grains of salicylate of sodium, every four hours, certainly relieve the symptoms if they do not change the course of the acuter pleuritis. A saline effervescent mixture may be freely used also as a drink, whereby activity of the skin and kidneys is promoted. In the earlier stages free purgation should be avoided, but it is well to call gently upon the alvine excretion by the use of mercurials and salines. All solids must be withdrawn from the dietary, and stimulants, as a rule, forbidden. The saline effervescent or a cream-of-tartar drink is to be continued after the powders are withdrawn, so as to keep up free excretion; for the same purpose, and also to lessen chest-movements, the patient must be kept closely to bed. For some days after the subsidence of the fever the appetite must be held in check, and it is desirable at this stage to lessen the amount of fluid in the dietary. Thus it is to be hoped that, as the patient's general condition improves, the effusion in the chest may likewise fall. If this be not the case blisters seem certainly efficacious; not pushed to full vesication, but repeated frequently. Iodine-paint, in the writer's opinion, is useless. At the same time, or soon after, a pill may be administered twice or thrice daily, containing a grain each of digitalis (fresh leaf) and blue pill. The digitalis, by keeping up the arterial blood-pressure which tends to fail, favours excretion; and mercury is one of the best of diuretics. The use of blisters and mercury must be avoided if the kidneys be not sound, and mercury should be avoided if phthisis be suspected. When a brief and gentle eliminative course of this kind is ended, restorative treatment, with such drugs as iron and quinine, should be substituted. Delicate and anæmic subjects may need iron and bitters, cod-liver oil, and liberal diet from a very early stage, and such cases are not uncommon. But such measures are not to be used in the acuter stages, a precaution too often forgotten; even in phthisis a sharp intercurrent pleurisy must be treated at first by salines, and possibly even a leech or two.

These measures will generally help to reduce not only acute effusions of moderate extent, but also of a more obstinate kind. If, however, the case resist the means prescribed, the effusion will probably increase, and operative interference may be urgent, as is so commonly the case in latent pleurisy. As a general rule, if a primary serous effusion rises much above the angle of the scapula, and abides, in this quantity or more, for two or three weeks in spite of

adequate treatment, it must be drawn off, whether the patient be embarrassed by it or not.¹ An effusion, however small, known to be purulent, must be removed at once. The presence of fever is no obstacle to operation. In cases in which treatment by medicines has not been fairly tried, when the patient is in comparative ease, the effusion is not above the spine of the scapula behind nor above the mamma in front, is not rising, and the neighbouring organs are not much displaced, operation may give place to medicinal treatment for two or three weeks longer. The writer, however, would advise the withdrawal even of a pint of fluid which had lain in the cavity for a month, unless there is pulmonary tuberculosis, as its continued presence promotes retraction of the lung, and tends to destroy the absorbent power of the pleura and of its granulations. Sometimes at a later date, when convalescence should be established, a still smaller quantity of fluid may be detected, and the patient remains poorly and out of condition. An inch or two of fulness persists at the base of the lung, and on puncture such remnant may prove to be pus, which would, of course, be dealt with thoroughly; yet even if serum only be found the withdrawal of it for some obscure reason seems to set the patient free to get well, if there be no other complication. If there be effusion in both pleural cavities, the mounts must be considered, of course, as one quantity. Before speaking at length of operation, however, it is well to say that two other methods remain—the so-called ‘thirst-cure’ and the jaborandi-cure. The first method consists in the withdrawal of fluid from the diet, which should consist of lean cold meat, stale bread, and the like. All fluids are forbidden, except half a pint on the third day, and a pint on the seventh and eighth days. The effusion is said to decrease daily under this method, but it is more painful than tapping, and could not be borne by all patients without injury. The second plan, to which excellent results are imputed, consists in the promotion of profuse sweating, by means of jaborandi. The drug is administered as a liquid extract, ʒj being given every three hours; or one-eighth to one-third of a grain of nitrate of pilocarpine is injected subcutaneously, and repeated as the results may indicate. The writer has not learnt to rely on the former method, and the latter he is indisposed to try. It is well, however, to control the amount of fluid taken. If medicinal and dietetic means fail to remove a moderate effusion, if the fluid be rising fast, or if it occupy the whole or, persistently, a great part of the pleural cavity, the cavity must be tapped. There should be no hesitation in tapping instantly any chest, even in cases of phthisis, which is dull up to the clavicle, or presents but a small tympanic space under the clavicle.

(2) *Operative*.—To ascertain whether the fluid contents of the chest be serous, sero-purulent, or purulent, an exploring syringe may be passed through the wall of the chest, and a specimen of the fluid drawn off. In this way information is obtained as

to the nature of the fluid, its accessibility, and its bacterial contents. Should no fluid be obtained, the puncture can be repeated at another spot more readily, and with less sense of failure, than a greater operation. The site of operation must be chosen with care; happily, there is plenty of margin for error. In encysted empyema with thickened walls we repeat that four or five punctures may be needed before pus be reached, and a negative result must not be accepted without examining under the microscope any drop or fragment within or upon the needle. In cases of multilocular effusion the emptying of one loculus only is of course an incomplete measure. Such cases can be dealt with only by repeated puncture.

The chest, as a rule, is to be tapped in the sub-axillary area, where there is little danger to other organs. Reasons to the contrary may present themselves in the case of adhesions tying the lung or diaphragm to the side or back of the cavity, of displacement of the heart, or of deformity. An oedematous area must be avoided, as access of pus may convert it into a large cellular abscess. The pointing of an empyema forward is no indication for an anterior opening, as this pointing will recede when a lower opening has been made; nor is the distant sound of respiration over the back of the affected side a reason for declining to operate posteriorly. Let any bulging of intercostal spaces be looked for, as at such a spot false-membranes are probably scanty; and let it be ascertained that there is room enough between the ribs for the insertion of a finger into the cavity; if not, a rib or two must be resected. The writer used to prefer the eighth space in a line with the angle of the scapula; but safe as is this position, and good for bottom drainage, elevation of the diaphragm often defeats the latter purpose and interferes with the tube. The anterior axillary line is too forward for safety and for drainage; the posterior line, or perhaps a little behind it, is usually the best place. Except, perhaps, in a very stout person a little difference in the thickness of the chest-wall is unimportant. If it should appear, however, that the fluid is so limited or encysted that it does not gravitate to the bottom of the cavity, or that it has a more anterior seat, a tentative puncture must be made at the dullest spot, regard being had, of course, to the position of neighbouring organs. Some years ago a ‘dry tap’ was a great disappointment to the patient’s friends, and an annoying rebuff for the physician. Now we are better aware that in obscure cases of circumscribed effusion such explorations may fail again and again and yet be finally successful, or in any case be an indispensable means of obtaining information.

After disinfection both of skin and instrument, the needle of the syringe must be thrust quickly through the tissues into the cavity, the operator being careful to enter a little below the mid space, so as to avoid the periosteum of either rib, and the intercostal artery above. There is no objection to freezing the skin beforehand, but if the edge of the canula present no ridge upon the trocar the stab is not very painful. If the fluid drawn be clearly serous, it may be well to wait a day or two to see whether a small draught will set up absorption of the rest. Many such cases are on record. If the case, as is very common, be of a tubercular nature, the reabsorption, thus accelerated, may, as

¹ In the revision of this article these words are left as originally written. The writer is aware that of late recourse has been had earlier and more frequently to paracentesis; but his own experience leads him still to counsel delay so long as the patient is in no distress. Acute serous effusions advance to a moderate limit, and then slowly recede. If drawn off during the period of increase the puncture will not prevent reaccumulation, in which case it is a useless vexation.

is alleged in peritonitis, be useful by the introduction of some antidotal factor into the blood. Certainly many of the cases in which absorption is thus started, either in pleura or peritoneum, do unexpectedly well. As a rule, however, it will be needful to proceed to a further evacuation of the cavity. See PARACENTESIS; ASPIRATOR; and PLEURA, Surgery of.

It may be desirable to inject one-fifth of a grain of morphine beneath the skin after the operation, in order to relieve any irritation either by cough or otherwise, and to secure subsequent rest. In some favourable cases no second tapping is needed, and the heart tends to recover its position on the completion of the operation, moving three inches perhaps in the course of it. In two cases in which the writer noted reduplication of the second cardiac sound before tapping, the sign ceased at once on the emptying of the cavity. In other cases, even of serous effusion, the severity of the pleurisy may have so fettered the lung that the readjustment of the parts is much more gradual, and the space of the effusion is reoccupied but slowly by the unfolding lung, and the yielding of the chest-wall and mediastinum. In this respect there is not much difference between serous and purulent formations, save, of course, that neglected cases are more likely to have become purulent. A rapid return of the physical signs to the normal is a very good omen, and in cases promptly dealt with this is now a common experience. Moreover, if large adhesions take place they seem to stifle tubercle. In cases of slower recovery we get less help from physical signs, the conditions within the chest being in a more stable state of perversion. In pleuropneumonia the lung may not be able to expand after a paracentesis for the pleurisy, so that only some ten ounces or so may be obtainable by resiliency of the ribs. It is rather the rule than the exception for some dulness to remain below the scapula, and this alone is no indication for repeating the operation. In many cases, however, perhaps in the majority, often without any recurrence of fever, the cavity refills, and aspiration may have to be repeated more than once. It is proper, therefore, to warn the patient of this beforehand, lest he be discouraged.

If there be subsequent pain or elevation of temperature, these, under ordinary circumstances, will prove to be transient, but if the rise of temperature continue after the first day or two, the formation of pus is to be feared. This event is not infrequently attended by a re-awakening of pain. As a rule, the pus must be evacuated by free incision as soon as discovered. An anæsthetic must be used that the operation may be performed deliberately. Chloroform in these cases seems to put less strain upon the limited breathing-powers than ether. If for any reason, such as renal disease, general anæsthesia be undesirable, it may be obtained locally. See ANÆSTHESIA, LOCAL.

From the time of the operation the temperature should fall rapidly to the normal, especially if the patient can be in the open air, the value of which cannot be overrated in these cases; if it rise again, the rise will be almost surely due to occlusion of the opening.

In the exudations of phthisical or carcinomatous disease, operation may be justifiable by the temporary relief given to the sufferer; paracentesis must, however, be preferred to incision as long as

possible, as the incision may not heal. If after the removal of a collection of pus, and the establishment of really free drainage, the discharge become more offensive and the fever remain, the disease is probably a progressive tuberculosis, and the forecast unfavourable. It will be remembered, however, that the pus may not all be included in one cavity, or in cavities which communicate; if so, each separate cavity must be opened.

There is no substantial difference of method in operating upon a case in which a pulmonary or other ill-placed fistula has already formed. The performance of the operation in such cases, if the bronchial opening be free and not valvular, and especially if there be much adhesion and length of sinus, is not generally indicated; and its need must be estimated by the behaviour of each particular case, especially by the temperature and the freedom of expectoration. In these cases the open-air treatment may suffice. In rare cases it may be possible to drain a pyopericardium through the pleural opening. In double empyema a day or two should, if possible, be allowed to intervene between the two operations, especially in children; there is some risk of collapse or of pulmonary oedema in doing both operations together, unless the empyema be encysted on one or both sides, when operation adds little to the disturbance.

After each or any removal of fluid from the chest the re-expansion may be assisted by respiratory gymnastics, by graduated exercise, by the inhalation of compressed air, or by residence at high elevations. Massage and faradism may be useful. Finally, the importance of treating the patient from the time of operation in the fresh air, as in phthisis, must be emphasised. The writer has seen very unpromising cases answer surprisingly to this method, even where operation was inadvisable.

3. Pneumothorax.—Fr. *Pneumothorax*; Ger. *Luftbrust*.

Pneumothorax is the state in which the pleural cavity, normally non-existent as a space, contains air or other gas. If the air or gas be present together with pus, blood, or a watery fluid, we give to the resulting states the compound names, *Pyopneumothorax*, *Hæmatopneumothorax*, and *Hydropneumothorax* respectively. The gaseous content in these cases may precede the entry of the fluid or succeed it; or it may be generated in the cavity by specific bacteria.

ÆTIOLOGY.—Pneumothorax is a commoner event than would be supposed, were we to confine our attention to cases recorded under this name. It is often an incident in the course of other diseases, and of none more often than pulmonary tuberculosis. Pneumothorax sometimes, but rarely, seems to appear as a primary event, disappearing again without further complication; usually it results from wounds of the chest, purulent pleuritis, phthisis, ulceration (simple or carcinomatous) of the œsophagus, stomach, bowel, or gall-duets, and so forth, opening into the cavity. If air be admitted mechanically to the pleural cavity, decomposition of other contents may add to the volume of that which was admitted. Even in those few cases in which pneumothorax seems ‘idiopathic’—in which, that is, we find pneumothorax to be the eminent morbid state—we are bound nearly always to assume this state to be secondary, and due to some perforation the cause and place of which may

escape recognition. Yet in some rare cases a strain seems to have ruptured the lung. Three cases in the writer's experience, occurring during violent exertion in vigorous men, recovered permanently in a few days; but, though tubercular perforation rarely or never heals, it is difficult to suppose that in any such cases the lung was perfectly sound. Cases due to wounds of the chest find their place elsewhere.

In empyema pneumothorax is apt to follow perforation of the lung with ejection of the pus upwards; or, more rarely, perforation of the chest-wall by ulceration outwards. In these cases of *pyopneumothorax* we have to deal, of course, with an abundance of both pus and air in the pleura. Pneumothorax, as we have seen, is a common complication of phthisis pulmonalis. When it occurs in the later stages of the disease, it often escapes observation; in the earlier stages its symptoms are less likely to be overlooked. Its occurrence may be determined by such a strain as a fit of coughing. That pneumothorax is not a still more common result of ulcerative processes in the lung is due to the anticipation of a breach of surface by previous adhesive inflammation. In phthisis the perforation is often minute, and the quantity of matter escaping into the cavity small—so small as to be inadequate to produce the physical signs of fluid contents. For obvious reasons the bursting of a phthisical cavity into the pleura is very rare; perforation is due to the ulcerative action of a superficial deposit upon the visceral pleura; such being its nature it rarely heals, but it may be enclosed by consequent adhesions. In other cases the escape is more abundant, or a more abundant effusion comes from the pleura itself, as a consequence of the resulting irritation. We have then to deal with an obvious sero-pneumothorax or pyo-pneumothorax. In cases due to tubercular perforation the effusion is generally serous, and if into a circumscribed cavity the sac may close up by adhesion. Air thus entering the pleural cavity, unless it pass through septic channels, is purified from such elements by its filtration through the lung. The mode of the opening by which air escapes into the pleural cavity is of chief importance; it may be, and often is, *valvular*, so that its entry during inspiration may not be balanced by its exit; in this case air accumulates under pressure. If, as in empyema, the lung be already collapsed, this pressure is the less distressing; if the lung be wholly or in part open, the pressure adds to the degree of the sudden embarrassment due to rapid retraction of the lung, absorption of its air by its blood, and encroachment upon the surrounding parts, including the opposite lung. The valvular action does not usually depend upon the shape of the orifice, but upon the retraction on expiration of the lung around it. This kind of pneumothorax, unless circumscribed, is relatively frequent, and more mischievous, as the tension may reach a very high degree. If the air have free issue, the pressure within is not above that of the atmosphere, and the air is readily reabsorbed. In puncturing the chest-wall with a fine trocar, in cases of serous effusion, the lung is sometimes wounded, and air escapes into the pleura. The accident is an untoward one; air passing through a healthy lung sets up no putrefaction, and is itself quickly absorbed, but a large quantity suddenly admitted may add a good deal to the suffocative distress of the patient.

Perforation into the pleural cavity by ulceration,

simple or cancerous, either in the lung itself or in such neighbouring organs as the œsophagus, the stomach, the bowel or connected ducts, is not very rare; the entry of air, food, or other foreign matters into the pleura in such cases adds greatly to the distresses of the latter days of life.

The last cause which needs notice is the action of ærogenic microbes in the cavity. Of these rare cases the writer has seen very few. In the last of them, a slow pleurisy of the right base, the dullness of fluid rose nearly to the spine of the scapula, with Skoda resonance above. Two or three days later an area of resonance of subtymppanic character formed in the midst of the dull patch and extended during the following two days. On percussion a disc of resonance was surrounded by a dull ring of perhaps a third of the common radius. On operation gas bubbled out and about a quart of turbid serum flowed away, of a peculiar but not gangrenous odour. After resection of two ribs the diaphragm and parietes were examined with the finger. Excepting some strong adhesions the results were negative; the surfaces smooth, no sign of perforation, no fluctuation, no evidence of pulmonary gangrene. But the operation gave no relief, subcutaneous hæmorrhages appeared, and the patient sank with subnormal temperatures and rapidly failing heart. Unfortunately no specimen of the fluid was reserved for bacteriological examination, nor could a necropsy be obtained.

ANATOMICAL CHARACTERS.—A patient rarely dies of simple pneumothorax; or if death be mainly due to this, yet unless it occur within the first few days it is probable that some considerable inflammation will be found also. In the vast majority of cases, of course, the pneumothorax is secondary to some other disease, and any fluid or other matters found with the air in the chest may be due, not to the mere admission of air into the cavity, but to the admission of decomposing elements into it. As concerns air alone, we have only to say that in the cases in which air has reached the pleura by a valvular opening, the affected side of the chest may be visibly distended. In such a chest the pressure of the contained air will be positive, and on puncture may escape with a hissing noise; the out-rush may indeed be strong enough to blow out a candle. This air is usually deoxidised, and rich in carbonic acid; if there be decomposing matters in the cavity, it is likely also to contain sulphuretted hydrogen. Neighbouring parts, especially the heart, will be found more or less dislocated, directly as the degree of positive pressure and inversely as the amount of adhesion. Bilateral pneumothorax, unless the lungs be widely extended by adhesions, is, of course, incompatible with life; if on necropsy it be found double, we may be sure that, on one side at any rate, it came on at the moment of death, and in this cavity no effusion will be found. If the opening eludes observation, it may be discovered by filling the cavity with water and injecting air by the trachea.

SYMPTOMS.—The symptoms of pneumothorax are of course the more distinct the less the symptoms of the primary malady. In those rare cases in which pneumothorax comes on suddenly as the primary disease—that is, in which the mode of entrance of air into the pleural cavity is unknown—we find the chief symptoms to be dyspnoea and a sense of faintness; pain is a less uniform but often very severe symptom, and worst

when the entrance of air is followed by irritation and inflammation due to the fluid or solid matters which accompany the gaseous. Aseptic air alone does not set up much irritation. Fever, in like manner, depends not upon the entrance of air, but of the irritating matters which accompany it. Like the pain, it may be considerable; it may not be present at all; or, again, it may be lost in the fever of the primary malady, or betray itself but as a slight exacerbation of this fever. The dyspnoea, chiefly mechanical, in part reflex, is necessarily attended by increase of pulse-rate; the two events being but different aspects of the same machinery. The degree of these accelerations depends upon the rapidity of attack, the amount of previous accommodation in the chest, and the quality of fluid and solid concurring with the gaseous escape. That in full or extensive pneumothorax the symptoms are in-grossescent, and far more injurious, with the valvular mode of opening we have already pointed out. The escape of air with irritating matters suddenly into the open pleural cavity of a person suffering but little, if at all, from a tubercular ulceration, or of one surprised by an accident in the midst of health, may cause dyspnoea almost suffocative in degree; intense pain, anxiety, lividity, clamminess, chilly breath, faintness, great acceleration of the pulse, and distension of veins in the neck. On the other hand, in pneumothorax occurring towards the end of phthisis, when a pulmonary ulcer breaks through a pleura widely adherent about a lung already half destroyed, the attack of chest-pain which follows a bad fit of coughing may be put down, like the dyspnoea and the pulse-rate, to the fatigues and distress of a restless night. The patient's general condition may not be much altered in such cases. Cough and expectoration in simple pneumothorax are slight; but if pneumothorax be established on the bursting of an empyema into a bronchus, expectoration uniformly purulent will be the most prominent of the symptoms. Emphysema of the skin may also result; on the other hand it may be present without pneumothorax, even in cases of simple fracture of rib, and in cases of interlobular emphysema passing to the neck by way of the mediastinal tissue. In the majority of cases liquid, serous or sero-purulent, accumulates in the cavity; and may often be detected within twelve hours.

The *physical signs* are as follows: the affected side, in valvular cases in comparatively young persons, may be enlarged in girth and of a rounder form. It is, moreover, still in respiration, the half of the chest being fixed (probably in the inspiratory position), or only dragged a little by the efforts of the accessory muscles; the intercostal spaces may fill up; air, like liquid effusion, may press down the diaphragm, thrust the mediastinum aside, and change the place of the heart, liver or spleen. In all cases the lung of the affected side, if free, will tend, of course, to retract, at least to the point of equilibrium of the elasticity. In non-valvular cases the affected side may fall in, so as to be of less girth than the sound side. Vocal fremitus must be absent if the lung be collapsed, or remote from the wall of the chest; if the lung be adherent in part to the chest-wall, vocal fremitus may be so far perceptible, and it may be possible by this sign to map out the extent of the adhesion. Decubitus is usually on the affected side.

Percussion gives us great assistance in the detection of pneumothorax. There is something about

the loud, low-pitched and extensive, if not tympanitic, vibrations of the stricken chest, often extending beyond the normal boundaries of the affected side, which is very characteristic. On the other hand, it is said that if, in valvular pneumothorax, the pleura be tightly distended by air the percussion-note may rise to a high pitch. In these cases a hammer and pleximeter are more effective, though diminished resistance to the finger may be noted. Fluid will dull the percussion considerably or altogether, in districts which will vary with the quantity of it and the position of the patient. In pyopneumothorax, with a bronchial fistula, the sharp line between hyper-resonance and dullness may be changed after a profuse expectoration. In such cases, as in all of free non-valvular opening, percussion is more tympanitic; there is little or no thrusting aside of neighbouring parts. The chest is often retracted. If the opening be large, cracked-pot sound may be obtained. In valvular cases by palpation with percussion dislocation of neighbouring parts and organs may be ascertained. If we confine ourselves to the pneumothorax, auscultation is generally almost negative; in rare cases we may detect by a blowing (amphoric) sound the entrance and exit of air by a free opening. In such cases fluid is always present as well; and a few resonant crepitations may also be heard near the shoulder-blade. The voice-sounds also and the cough may be more or less amphoric. Vesicular breathing is never heard. In pneumothorax there is often present the peculiar epi-phenomenon called *metallic clang*, due to certain inharmonious over-tones, which arise under conditions of which we know little. It never appears at the beginning, and sometimes disappears in the course of a case. After death this metallic echo is always to be obtained. Succussion, known to Hippocrates, is obtained by shaking the patient laterally, when, if free fluid be present, a splash will be heard. The echo of splashing, bubbling, or dripping fluid (*gutta cadens*) in the air-containing pleural cavity, and indeed alien sounds generated inside the patient, such as the heart-beat or cough, may take the metallic clang from the chest-cavity, and may betray pneumothorax or illustrate it. The bell-sound, heard on listening while a bystander taps with a coin another coin laid on the wall of the chest, is well known. Most of these phenomena are best heard by laying the ear on the chest. In the same case, at different times, such sounds may be heard, even at a distance, or may be inaudible—variations which are due to mechanical conditions dependent upon the formation of false membranes, the freedom of the fluid contents, the proportions of the cavity, or the pressure of the contained gases (see PHYSICAL EXAMINATION). In a case of pneumothorax caused in an apparently quite healthy young man during a gymnastic effort, a succession of tinkles was heard, not only by his physician, but also by the bystanders. The sounds, which continued more or less frequently for some little time, seemed to be caused by bubbles of air issuing from the hole in the lung and breaking into the cavity. The patient recovered quickly, and has remained well up to the present time (fifteen years, at least). Finally, in skiagraphy the abnormal clearness of the affected side, the depression of the diaphragm, and the shadow of the retracted lung are manifest, and possibly even waves of succussion.

DIAGNOSIS.—If the presence of adhesions pre-

vent the development of the above symptoms, the case may be more obscure, but by so much the less serious. As, on the one hand, in an enormous moist pulmonary cavity it is conceivable that metallic and succussion-sounds may be heard, so, on the other hand, pyopneumothorax, restricted by adhesions to small dimensions, might simulate a pulmonary cavity. Diagnosis might be impossible in such a case, but, speaking generally, the dullness and retraction of the chest-wall over a cavity would declare the diagnosis. Distension of the stomach with elevation of the diaphragm, or a diaphragmatic hernia, could rarely be mistaken for pneumothorax by anyone who fairly took into consideration all the facts and history of the case. Splashing in the stomach is a much less constant phenomenon, and is obtained by different handling; it varies also in clang. Still, in a particular case of a large stomach suspended by adhesions, some embarrassment arose. A persistence of respiration in the upper part of the chest, signs of mischief below the level of the diaphragm, and perhaps the position of the heart, with the history of the illness, would assist diagnosis. Skiagraphy may help us in such cases of doubt. As empyema, especially in children, may lead to purulent pericarditis, so may pyo-pneumothorax by perforative process have *pyo-pneumo-pericardium* added to it. Emphysema of the lungs may give rise to percussion-sounds of 'horse-hair cushion' quality or as deep as in pneumothorax. Emphysema, however, is practically always bilateral, it is rarely dissociated from signs of open bronchial tubes and vocal vibration is not abolished. In cases of pyo- or sero-pneumothorax there may be a difficulty in determining the quantities of fluid and of gas respectively in the cavity; as much as three quarts of fluid may co-exist with a great deal of resonance above it. Where skiagraphy can be practised, the level of the fluid may thus be distinguished from the abnormally bright air-space above. In emphysema the pulmonary area is abnormally bright on both sides; but in pneumothorax it is still brighter, and is unilateral. The position of the lung also may be perceived. The diaphragm (in cases of high pressure) may be depressed on the affected side. Adhesions may modify all the phenomena infinitely. For the differential diagnosis of subphrenic abscess, often difficult, reference is made to this title. Diaphragmatic hernia (*see* p. 386) presents a curved upper outline, succussion fails, and the history of the case is different.

PROGNOSIS.—This obviously depends so largely upon the causes and circumstances of the pneumothorax that general directions are impossible. The prognosis of chest-wounds, of phthisis, of empyema, contains differences too wide for formulation. The percentage of deaths on the total number of cases is very heavy, perhaps 80 per cent. The tendency of air in the pleura, in the non-valvular cases at any rate, is to absorption. It is asserted that pneumothorax, by sudden oppression of the lung by the indrawn air, may cause rapid or even sudden death.

TREATMENT.—The treatment of pneumothorax must depend greatly upon the nature of the primary malady. Some cases need no treatment at all. To operation in pyopneumothorax from empyema we have referred already. Whether in a case of phthisis pyo-pneumothorax should be dealt with by operation, or not, is a question usually, but not

always, to be decided in the negative. Cases occur in which the pressure or character of the effusion, and the chronicity of the lung-disease, may outweigh the risks; needle-puncture should be tried alone in the first instance. In wounds of the chest-wall, or of the pulmonary pleura, the puncture rarely closes so soon as to imprison the air in a state of higher tension than the atmosphere. In valvular cases the displacement of organs and respiratory distress may call urgently for relief. If so, a fine trocar must be inserted into the chest, and by means of a tube the air and any liquid run off through sterilised water. In such cases with recurrent mischievously high intrapleural pressures, a permanent outward opening with tube and antiseptic dressings may be necessary, in the hope that favourable adhesions may form. Anæsthesia must be local only. The hypodermic use of morphine is not only valuable in soothing the pain and distress of pneumothorax, but also in preventing cough or deep inspirations which, in valvular cases, compel more and more air into the cavity.

4. Pleura, Dropsy of.—SYNON.: Hydrothorax; Fr. *Hydrothorax*; Ger. *Brustwassersucht*.

Hydrothorax is to be classed with dropsies elsewhere, and is often the companion of ascites and anasarca. It may exist alone, but is rarely unilateral. It never exists as a sole malady. We may say generally that it is apt to arise under the following circumstances: when the whole circulation is so impeded that venous pressure is increased, as in heart-disease; when venous arrest is due to some local causes, as, for instance, to the pressure of swellings upon veins, or to venous thrombosis; when the bronchial glands are enlarged; when in renal disease the removal of water from the system is checked and the blood vitiated; or, finally, when the quality of the blood is so deteriorated in the cachexia of cancer or other chronic disease, that its serum oozes from the vessels. In the first and third cases we should expect to find dropsy in both pleural cavities, but such transudations rarely stand at the same height in both; indeed the contents of one of them is often so small in volume that the hydrothorax may seem at first to be unilateral. The right side is generally the fuller, but the difference usually depends upon posture.

DIAGNOSIS.—The diagnosis of hydrothorax and its measure are easy, except in the cases where the effusions are restrained by adhesion. Vesicular breathing may not be absent, as the lung moves more readily than in pleuritic effusion; pressure-signs are rarely observed; the line of dullness does often shift (while in inflammatory effusion it practically does not), and in systemic disease there will be dropsy elsewhere. The pleuritis of arterial and renal disease, often an early event, is unilateral; the effusion contains fibrinous clots, and the pleura is thickened. When hydrothorax co-exists with intrathoracic growths, these signs may not apply, and the needle alone can decide. The fluid is clear, slightly albuminous (2-3 per cent.) and under sp. gr. 1014-17. A higher sp. gr. without fibrin would suggest tubercle or carcinoma. Before assaying the sp. gr. the fluid should stand a while, for coagulation and to let any gases escape from it.

TREATMENT.—Hydrothorax in the majority of cases is not formidable in itself, and (being not uncommonly an event of the last days or hours of life) may be unnoted till the necropsy. Diuretics and hydragogue purgatives act more readily in

hydrothorax than in inflammatory serous effusions; still, if the fluid increase so far as to harass the breathing or to add to the dangers of the disease, it should be drawn away by a fine trocar. The operation may be repeated a great number of times if re-accumulations make it necessary; and sometimes after repeated withdrawals it ceases to recur. In heart-disease or arterial degeneration the transudate is not rarely tinged with blood.

5. Pleura, Hæmorrhage into.—*SYNON. : Hæmothorax.*—Effusions may be bloodstained, as we have said, even in simple pleurisy; but this is more common in such conditions as scurvy, tuberculosis, or cancer. A purely sanguineous effusion is generally the result of wounds of the chest or its viscera; but it may arise from within, as from rupture of a blood-vessel or of an aneurysm, or from a bleeding cancer. The means of examination or treatment of such cases, in so far as these are possible, may be gathered from the preceding sections. Hæmorrhage into the pleura from direct extravasation may be left awhile, on the chance of re-absorption, but if this does not seem on the way, or life be threatened, a tentative puncture may be made, with scrupulous antiseptics. If the issue be ichorous the patient will probably become febrile, and the major operation be needed.

6. Chylothorax.—A very rare event, due to a breach in the thoracic duct, by wound, erosion, or pressure-distension. *Chyliform effusions*, which probably account for nearly all the cases on record, do not contain chyle proper, but are milky from fatty particles and cholesterolin, due to degeneration of cells of pus, epithelium, tuberculosis, or cancer. Such effusions are not very uncommon, and might, of course, have the addition of chyle proper if the duct were perforated. A rapid degeneration of cells, cancerous or tubercular, may give rise to a quantity of fat-droplets so great that a layer of fat may be seen on the top of the serosity withdrawn by tapping. See *FILARIASIS*.

7. Pleura, Morbid Growths in.—Primary cancer of the pleura is infrequent, but the part enjoys no freedom from the invasion of sarcomatous or carcinomatous growths from neighbouring parts, as frequently from mammary cancer, of which it may be the fatal conclusion. It may occur before or after excision. From the cancer in the wall of the chest simple inflammation often extends to the pleura, and produces the ordinary results. In cancer of the lung the pleura, costal or diaphragmatic, is rarely spared, and secondary deposits usually arise, as it were, simultaneously in these parts, and in the pericardium; so that the primary seat may be indistinguishable. Cancer is usually seen in the form of small flattened or rounded elevations, rich in blood-vessels. False membranes in these cases may attain enormous thickness, and friction may be loud and persistent. If septic matters escape into the pleural cavity its effusions, which may be serous, purulent, chyliform (q.v.) or even sero-fibrinous, will soon become putrid. Blood oozes readily from any highly vascular formations—whether cancerous, tubercular, or simply inflammatory; and may be seen in the fluids withdrawn, but a deficiency of fibrin in a hæmorrhagic effusion would suggest cancer.

There is little more to be said of the symptoms and signs of such cases than will be found in former parts of this article; and under *MEDIASTINUM*, Diseases of, p. 967. The diagnosis of cancerous or other such masses from their own effusions, or

from other effusions, may be impossible without the needle, which, if it enter the morbid growth itself, conveys unmistakable information to the finger. A scrap of the growth may be withdrawn. The distribution of areas of dulness in cases of morbid growth often enables us to distinguish them from cases in which the anatomical boundaries of the chest are respected, or the action of gravitation unimpeded. Enlargement of tracheo-bronchial glands may be detected beneath the sternum or in the interscapular space; or again in the supraclavicular fossa, from which latter they might be removed and examined microscopically. Fever is usually but by no means always absent. Still after all in most cases early diagnosis is difficult.

The prognosis will not depend upon the pleuritic changes; and the only remark to be made on the treatment is that paracentesis, in the secondary effusions, is not wholly to be declined. Some patients have obtained from repeated puncture perhaps a prolongation of life, certainly a relief of suffering. If in case of doubt the removal of a substantial effusion give little or no relief, morbid growth may be suspected.

Hydatid of Pleura.—Hydatid is rarely met with in England either in pleura or lung, and in either occurs rather as a secondary than as a primary event. The most common source of thoracic hydatid is the liver, whence it tends to invade rather the right side of the chest, and may rise nearly to the clavicle. In some cases, when the cyst is comparatively small and unbroken, its nature may be suspected from its position and outline.

In other cases, and especially if the cyst has ruptured into the pleural cavity, physical diagnosis may give no conclusive evidence, and we must make what we can of the history and other circumstances of the case. The fluid is non-albuminous, of low sp. gr., and contains hooklets (p. 462). The occurrence of pain suggests pleural relations.

When a hydatid cyst breaks into the pleural cavity, its treatment falls under the ordinary rules of pleuritic effusion, but aspiration alone will not suffice for a cure. T. CLIFFORD ALLBUTT.

PLEURA, Surgery of.—The operations upon the pleura fall naturally into two groups, those undertaken (1) for purposes of diagnosis, and (2) for purposes of treatment.

1. For purposes of Diagnosis.—The best instrument is a large hypodermic syringe that can be rendered absolutely aseptic. The needle is thrust through the middle of an intercostal space at a spot which is well within the limits of the area suspected to cover a collection of fluid. In cases where the patient is very nervous and apprehensive of pain, the skin should be frozen at the spot selected for the insertion of the needle. Inasmuch as the pain is so slight and momentary, it is justifiable to treat the apprehension of the patient by suggestion, and to wipe the area of skin with a substance which the patient is told will prevent the pain. To inject any local anæsthetic would cause almost as much pain as the exploring needle itself. If no fluid escapes into the syringe as the piston is pulled back, the depth of the point of the needle from the surface should be gradually varied. If the result be still negative the syringe should be detached from the needle and the latter cleared by means of the usual stylet; the syringe is then attached again and another attempt made at

the same spot. It is usual to insert the needle in several places, say three or four, until a positive result is obtained. If the needle and syringe have been cleaned out with carbolic-acid lotion and several drops of this fluid have remained in the syringe, this may cause coagulation of any albuminous fluid in the lumen of the needle, and thus plug the passage. Another important drawback to the use of carbolic-acid lotion is that even if fluid is found it may be altered in appearance by mixing with the carbolic-acid lotion; thus, to a somewhat inexperienced eye, a few drops of serous fluid mixed with a drop or two of carbolic lotion, may present the appearance of pus. If it be thought best to cleanse the syringe with carbolic lotion, it is important to finish the cleansing with some boric-acid solution or a little boiled water.

At the present day the treatment of pleuritic effusions depends to a large extent upon the kind of micro-organism producing the inflammation, and therefore it is most important to ascertain what micro-organism, if any, there is in the fluid. If suitable sterilised tubes with nutritive media are to hand, the inoculation should be made at once direct from the syringe. If, however, the fluid has to be sent some distance it should be emptied from the syringe into an empty sterilised test-tube, or the needle itself be sealed with wax and surrounded with aseptic wool or lint, and sent to the bacteriologist without delay. Owing to the fact that tubercle-bacilli are often absent in effusions from a tuberculous pleura, or at any rate owing to the fact that the tubercle-bacillus cannot be demonstrated, inoculation of guinea-pigs may be the only certain means of diagnosis; definite instructions should therefore invariably be sent to the bacteriologist as to the nature and source of the fluid and the exact information required.

2. For purposes of Treatment.—The pathological condition demanding operative interference is the presence of fluid in the pleural cavity which either by its quantity or its toxic action is producing mischief. Broadly speaking, there are two kinds of fluid which are met with: (1) Clear or slightly turbid, as met with in ordinary 'pleurisy with effusion'; and (2) thick or purulent, varying in consistence and appearance according to the nature of the disease.

It is not within the province of this article to discuss the indications for deciding upon any definite line of treatment.

The methods used for evacuating pleural effusions are aspiration and incision with drainage.

(A) *Aspiration of the Thorax, Thoracocentesis.*—An enormous variety of apparatus has been devised for this purpose; most of the instruments aim at emptying the pleural cavity as completely as possible in one operation. Of recent years various methods for permanent aspiration have been devised and used with considerable success, especially on the Continent. In Great Britain simple aspiration, removing as much fluid as possible at one sitting, is the usual practice. The condition of the patient must be carefully watched, and the withdrawal of fluid stopped the moment the patient shows any signs of collapse. The fluid should be removed slowly and, according to some authorities, never more than 50 oz. should be removed at one sitting. The criterion as to the amount that can be removed at one time really depends upon the condition of the patient, which must be most

carefully watched. At the London Hospital Dieulafoy's aspirator is the one in general use. It is unwise to employ an apparatus where any marked degree of forced aspiration is used. Other well-known apparatus are Potain's aspirator and its various modifications, especially that by Debove, in which all stopcocks are done away with. See ASPIRATOR.

Operation of Thoracocentesis.—The spot for aspiration having been selected, the skin is well cleaned and rendered aseptic. If the collection of fluid is considered by the physician to occupy the whole pleural cavity the exact point for aspiration is usually left for the surgeon to decide upon. The consensus of opinion seems to be that the best spot is a point in the mid-axillary line between the fifth and sixth ribs. Next in favour to this point is a spot below the angle of the scapula in the seventh or eighth intercostal space. Any point lower than this should be avoided so as not to wound the diaphragm or the abdominal organs, and to avoid the needle getting blocked by the rising of the abdominal organs as the chest becomes more and more empty of its fluid. The patient should lie on his back in as horizontal a position as possible. At the commencement of the operation the patient's dyspnoea may be so great that he can only breathe in a sitting posture. As the aspiration continues and the dyspnoea is relieved, the patient should gradually be allowed to assume the horizontal position, so as to avoid syncope. When it is decided that sufficient fluid has been removed, or when no more fluid can be obtained, the needle should be rapidly withdrawn and the puncture sealed with a little collodion.

In order to remove all the fluid from the pleural cavity, a procedure which seems to the writer to be absolutely unnecessary, methods have been used by which sterilised air is forced into the pleural cavity so as to completely empty it of effusion. Not only does this method seem unnecessary, but it may be distinctly harmful. To subject an inflamed surface of considerable area to sudden changes of pressure certainly seems unwise.

A method of *permanent aspiration* has been devised by Playfair and by Bulau. The skin is rendered anæsthetic, and a large trochar and cannula is pushed through one of the lower intercostal spaces. After removal of the trochar a soft india-rubber tube is pushed through the cannula into the pleural cavity and then the cannula is withdrawn. This tube is fixed to the skin by a couple of stitches, and is connected with a vessel containing an antiseptic solution. In order to see the exact nature of the fluid a glass junction is inserted. The patient can walk about with the bottle in his pocket. Very favourable reports on this method come from the Continent, but it does not appear to have been employed on any large scale in this country.

At the present time the operation of thoracocentesis is limited to removing serous effusions and the purulent effusions of tubercular disease. Practically in all cases of pyogenic infection the operation of incision and drainage is the one employed.

(B) *Method of Incision and Drainage, Thoracotomy.*—For the removal of purulent collections from the chest this operation has been practised on and off since the days of Hippocrates. There has been a tendency to lay too much stress upon the difficulty of this operation. Practitioners with but little familiarity with surgical procedures

sometimes delay in dealing with an empyema, because they are afraid to push a scalpel between two ribs, and, by postponing the operation, imperil the lives of their patients.

Operation of Thoracotomy. — At the outset we meet with the question as to whether it is advisable or necessary to remove a portion of rib or whether a simple incision through an intercostal space into the cavity is alone sufficient. The general consensus of opinion seems to be that the removal of a portion of rib facilitates drainage and assists in the falling in of the chest-wall, which is one of the essential features in the final closing of the cavity. The operation should be performed under strict antiseptic precautions. Every effort should be made to prevent the entrance of ordinary pyogenic organisms from without. The empyema may have been caused by micro-organisms whose activity is practically exhausted, or which rapidly die when disturbed by drainage, or a mistake may have been made and a tubercular cavity opened.

An incision three inches long is made over the seventh rib, in the posterior axillary line. The periosteum is incised and reflected to the upper and lower borders of the rib and detached carefully with the intercostal muscles. When isolated the rib is divided with bone-forceps and about two inches of it removed. The thickened pleura is then incised parallel with the intercostal incision and the pus evacuated. Any large masses of coagulated exudation presenting at the aperture should be seized with forceps and pulled out. The patient is usually coughing at this stage, and the masses are jerked against the opening with each expiration. Owing to the fact that a sudden stoppage of the heart has followed flushing of the cavity it is wiser not to do this, but having evacuated as much of the pus as possible a drainage-tube should be inserted. Effective means should be taken to prevent the tube slipping into the cavity. One of the best ways is to transfix with a large safety-pin the outer end of the tube. An antiseptic dressing is then applied, and as soon as the discharge soaks through it should be replaced by another. In all cases the tube should be shortened in a few days, and in very favourable cases, where the lung has expanded rapidly and where little or no pus escapes, the tube should be removed and a small gauze drain inserted.

The closing of an empyema-cavity is due to three causes: (1) The expansion of the lungs; (2) the falling in of the chest-wall; and (3) the conversion of the granulation-tissue lining the lung and the parietal pleura into cicatricial tissue. This still further helps in expanding the lung and pulling in the chest-wall. In certain cases these three factors are insufficient to close the cavity, and a very awkward condition is arrived at, especially when the cavity may measure in its three planes respectively, eight or nine inches, four or five inches, and two or three inches. This means a very large superficial area of granulation-tissue, generally septic and discharging many ounces of pus each day. To Estlander is due the credit of first dealing effectively with this condition; recognising that nothing could be done to cause the lung to expand and that the rendering of the cavity aseptic, when possible, is of no avail, there remained naturally the chest-wall to deal with. In order to facilitate the falling in of the chest-wall he excised several inches

of several ribs so that there was an immediate, and afterwards a gradual, approximation of the chest-wall to the lung.

As regards the operative procedure each case must be considered on its merits, and it is here that the experience of an operating surgeon is essential. The operation may be a very extensive one, requiring high surgical skill for its proper performance. The details of such operations do not come within the scope of this work.

HENRY PERCY DEAN.

PLEURODYNIA (πλευρά, the side; and δόνη, pain).—SYNON.: Intercostal Myalgia; Fr. *Pleurodynie*; Ger. *Seitenschmerz*.—A name for muscular rheumatism or cramp affecting the chest-wall. See CHEST-WALLS, Morbid Conditions of; CRAMP; and RHEUMATISM, MUSCULAR.

PLEURO-PNEUMONIA. — This compound word signifies a combination of inflammation of the pleura and of the lung itself. In all cases of acute pneumonia there is a certain degree of pleurisy corresponding to the inflamed lung; but it is of little or no practical significance, there being only some exudation on the pleural surfaces. Pleuro-pneumonia implies that the two morbid conditions are actually associated in various degrees, giving rise to their respective pathological changes, and each thus influencing the symptoms and physical signs. Individual cases, therefore, present many diversities, in accordance with the different ways and degrees in which the two diseases are combined. It may happen that they are associated from the first; or one may supervene during the progress of the other, in this way modifying its course and physical signs, and not uncommonly rendering the diagnosis more or less obscure and difficult. See LUNGS, Inflammation of; and PLEURA, Diseases of. FREDERICK T. ROBERTS.

PLEUROTHOTONOS (πλευρόθεν, laterally; and τόνος, tension).—A form of tetanic spasm, in which the body is bent towards one side. See TETANUS.

PLEXIMETER (πλήσσω, I strike; and μέτρον, a measure).—A flat instrument used in mediate percussion, by being applied to the surface of the body to receive the stroke of the plessor. See PHYSICAL EXAMINATION.

PLOMBIÈRES, in France.—Thermal waters, with some soda, silicate, and arsenic. See MINERAL WATERS.

PLUMBISM. — See LEAD-POISONING; and OCCUPATION-DISEASES.

PNEUMATOCELE (πνεύμων, the lung; and κύλη, a tumour).—Hernia of the lung. See LUNGS, Malpositions of.

PNEUMOCONIOSES (πνεύμων, the lung; and κόνις, dust). — By the term 'pneumoconioses' is meant diseases of the lung due to inhalation of dust. Upon the character of the dust depends the rapidity with which the lung-tissue is destroyed. The dust given off from stone and steel during grinding is hard and angular and is therefore much more harmful than soot or coal-dust, which is soft and less angular. Mineral, metallic, or organic particles when inhaled into the pulmonary alveoli are capable of causing chronic inflammation.

Anthracosis (*ἀνθραξ*, a burning coal).
SYNON. : Coal-miner's Phthisis. — Until coal-mines were ventilated according to the regulations laid down by Act of Parliament, colliers, as a class, suffered much from bronchitis and phthisis, but so effectively is fresh air carried into the furthest recesses of mines and the foul air removed that the coal-miner of to-day enjoys an immunity from phthisis which is denied to the general population. It is the exception now to find coal-miners suffering from consumption. Although the 'black spit' is a thing of the past, the lungs of colliers are found after death to be deeply pigmented without any pronounced pathological changes. The human lung is wonderfully tolerant of coal-dust and the soot from the miner's lamp.

ANATOMICAL CHARACTERS.—The lungs, which may or may not be adherent, are frequently emphysematous and bluish-black in appearance, and through the pleura can be seen and felt small black nodules. The glands at the root of the lung are enlarged and on section black. On cutting the lung there escapes on pressure a black inky fluid which stains the fingers, and the pulmonary tissue is black. The lungs are usually not diminished in size, for carbon-particles when inhaled do not induce fibrotic change like some other forms of dust. On microscopical examination the black particles are seen along the lines of the lymphatics. It is upon the mucous membrane of the bronchi that the particles of coal-dust first fall when inhaled. Here they may be taken up by the epithelial cells and thrown out by the expectoration. Some may penetrate the bronchial mucous membrane, but it can only be a few on account of the thickness of the basement-membrane. What causes the dark pigmentation of the lung is the particles that have been aspirated into the alveoli. There they effect an entrance into the lymphatics by passing between the epithelial cells or through stomata, and they subsequently come to occupy the lymphatics that are distributed around the branches of the pulmonary artery. Most of the black nodules observed on cutting a lung are perivascular accumulations of carbon-particles. In a similar way they also surround the bronchi or appear as long streaks in the pulmonary septa. Thus is explained the generalised pigmentation of the lung. In the bronchial glands to which the lymphatics run the carbon-particles are arrested. In the absorption and conveyance of the pigment, leucocytes probably play an important part.

SYMPTOMS.—It was formerly held that *anthracosis* and *tubercular phthisis* were naturally exclusive of each other. There is nothing to support this contention. In all the pneumoconioses, destructive processes in the lung are induced in the first instance by irritating particles, but in the pulmonary lesions thus induced tubercle-bacilli may subsequently find a suitable nidus. It is only because coal-dust particles are little irritating and the collier works in very pure air that anthracosis is not more frequent. Once the disease is established it runs a slow course. The individual gives a history of recurrent attacks of bronchitis attended by cough and dyspnoea on exertion. His expectoration is black. When upon a lung in this condition there is engrafted tubercular infection there occur the progressive emaciation, failing health and more abundant expectoration observed in ordinary phthisis.

Lithosis (*λίθος*, a stone).—**SYNON.** : Silicosis

(*silex*, flint); Stonemason's, Stonegrinder's and Potter's Phthisis.

The dust given off by stone and by fired clay is composed of silica or silicate of alumina. As the anatomical changes in the lungs in stonemotters and potters are practically the same, one brief pathological description will apply to both. Particles of silica are extremely irritating and destructive to lung-tissue. Their presence in the lung induces a reactive inflammation which is followed by an excessive development of fibro-connective tissue. The lung subsequently contracts, is hard and on section feels gritty, but it may be bulky if the amount of stone-dust contained is large. Hard nodules of dense fibrous tissue are often present, and in these can be seen and felt particles of grit. At places the pleuræ are densely adherent, while at others there can be seen projecting through the pleuræ similar nodules to those just mentioned. The particles of grit observed on section of a fibrotic area do not dissolve on the addition of an acid. Greenhow found on incinerating a portion of cirrhotic lung and boiling the ash with strong hydrochloric acid that none of the ash was dissolved, and that the residue, which was composed of minute angular particles, disappeared on exposure to the fumes of hydrofluoric acid, a circumstance that showed the particles to be silica. Owing to retraction of the fibro-connective tissue bronchiectatic cavities are formed in the lungs. The bronchial glands are much enlarged and extremely hard, and on section exhibit a greyish-black colour. The lungs may show patches of catarrhal pneumonia. Commencing as a non-tubercular disease tubercle-bacilli may ultimately be found both in the expectoration and in the lung. Sometimes the lungs of lead-miners become stonily hard, solid, and heavy: the parenchyma becomes replaced by dense fibro-connective tissue and assumes a bluish-black appearance, but on chemical examination of the lung the writer has never found lead therein, a circumstance which shows that just as in the stonemason's and potter's lung the cirrhosis of the organ is caused by particles of stone (*lead-miner's phthisis*).

It is interesting to note that the granite-workers of Aberdeen do not suffer from pulmonary phthisis in consequence of their occupation. Hamilton attributes this immunity to the igneous origin of the stone, the larger size of the particles, and the fact that they do not reach the pulmonary alveoli, as they become entangled in the upper parts of the respiratory passages. Of all stone-cutting and stone-dressing occupations, one of the most un-healthy is French grindstone-building. 'Buhr' stone is extremely hard. It is chiselled into wedges which are placed together to build up a grindstone. The men who follow this employment die in very large numbers. Peacock estimated the mortality from lung-disease to be 40 per cent., and that the average age at death of 23 labourers out of 41 was 24·1 years. The writer has visited most of the 'buhr' stone-yards in the country and examined the men who work therein, and can bear testimony to the views just expressed. The men are short-lived, they seldom reach the age of 35, most of them dying much earlier. They may follow their occupation for five or ten years without feeling any bad effects, and then without any special explanation they begin to suffer from cough and profuse hæmoptysis. The illness runs a very rapid course. A considerable number of ordinary stonemasons,

too, seek relief on account of pulmonary disease by the age of 35-40.

Potter's Rot or Asthma.—The manufacture of china and earthenware is a prolific cause of pulmonary disease, owing to the clay and flint used in their production. The clay is rich in siliceous material. To it and the sharp angular particles of flint is attributed, when inhaled, the damage done to the lung which causes *potter's rot*. Of all the processes the one most responsible for pulmonary disease is the 'scouring,' or brushing off the dust from the china after it has been fired. A dustier process is scarcely conceivable when done by hand. The work is generally done by women who are ignorant and careless. Owing to the hard and penetrating character of the dust considerable injury is inflicted upon the lung. Its power for harm is only surpassed by the dust given off in the Sheffield dry-grinding trades. The disease it causes develops slowly, at first without any constitutional symptom, for it is a non-tubercular process. There are morning cough, shortness of breath, and mucous expectoration; but, unlike the French millstone-builder, hæmoptysis is usually absent from first to last. So insidious is the malady in its inception that, as the appetite remains good and the worker looks and feels well for a lengthened period he continues to follow his employment, thus exposing himself to further pulmonary implication. For a period he expectorates white frothy material containing a few black streaks due to the presence of dust, but as time goes on the sputa become purulent, the cough more paroxysmal and violent, and the dyspnoea even greater than in ordinary phthisis and out of all proportion to the amount of consolidation of the lung. It is less the apices than the bases of the lungs that are affected. Here and there in the chest patches of alternate dulness and resonance may be detected due to consolidation and emphysema respectively. The disease differs from ordinary tubercular phthisis in its extreme chronicity and in the fact that the apices are much less involved. The patient retains the look of an asthmatic rather than that of a consumptive. The potter thus affected suffers like the victim of chronic bronchitis from dilatation of the right heart with its attendant troubles. Arlidge found, over a series of years, that the mean age at death of male potters aged 20 and upwards was $46\frac{1}{2}$ years, while that of non-potters stood at 54; that potters had a mortality of 12.29 per cent. compared with 7.86 of other workpeople; and as showing how one branch of the industry was more deadly than others, phthisis was the malady in 40 per cent. of china-scourers, and bronchitis in 25 per cent.

Closely allied to potter's rot is the condition which is prevalent among workers in asbestos-factories.

Steel-grinder's Phthisis.—So far as the Sheffield trades are concerned it is the 'dry' grinding that is most provocative of disease. Some of the very fine particles of steel and stone-dust given off during this operation are inhaled by the workman, who, sitting astride his 'horse,' bends closely over the wheel upon which the grinding of scissors and razors is being conducted. Sooner or later, as the result of the inhalation of dusty particles, a considerable amount of tracheal and laryngeal irritation is induced, the voice becomes husky and there is cough, accompanied at first by very little expectoration. Subsequently there are superadded to the

cough dyspnoea on exertion, pain in the chest, muco-purulent expectoration, progressive emaciation, and all the signs and symptoms of pulmonary phthisis with recurrent hæmoptysis, a circumstance which causes steel-grinder's rot to differ symptomatically from the similar lung-affection of the potter. The fibrotic changes in the lungs of steel-grinders have been attributed by some writers to iron-dust, but the amount of iron found in the lung is really small compared with silica. It is more than probable, therefore, that they are due to particles of the grindstone given off during dry-grinding, for this operation is always more harmful than the wet process.

Basic Slag.—When ground, the basic slag obtained from iron-works is used as a manure. The pulverisation of it is a very dusty process. The dust itself is not poisonous, but when inhaled it has a decidedly deleterious influence upon the respiratory organs. Many of the workmen suffer from cough, and have deep husky voices. They become bronchitic and the subjects of emphysema and asthma. They die of pneumonia in a larger proportion than men engaged in other occupations. Once an acute or sub-acute pulmonary disease is superadded to the bronchitis, the illness runs a more rapid course than a similar malady established in men engaged in other dusty trades. This, however, may be partly due to the intemperate habits of the workmen. Time is not usually given for fibrotic changes to occur in the lungs. Within the last few years, owing to improved machinery and the fact that the grinding is conducted in closed mills, there is much less illness among the men than formerly.

Ganister Disease.—Ganister is a siliceous rock found in the under-clays of the lower coal-measures, and when made into bricks is used to line the bottom of Bessemer-steel converters. It is an extremely hard substance. The men who mine and grind the ganister, also those who make it into bricks, owing to the very dusty nature of the operations and the extremely hard and sharp character of the particles, soon become incapable of any lengthened physical exertion. They develop a hacking cough and in four or five years' time die from fibrosis of the lungs. Birmingham found the average annual death rate from lung-disease in ganister-workers to be 22.29 per 1,000, and Robertshaw that the average age at death was 36.6 years. The symptoms at first are those of bronchitis from mechanical irritation, but eventually there is induced fibrosis of both lungs.

In Siderosis (*σίδηρος*, iron) the structure of the lung is altered by iron dust that has been inhaled. The disease occurs in colour-grinders and makers, who use oxides of iron. It was the detection of the red oxide of iron in the lung by Merkel that definitely proved the pathological changes in the lung to be due to varying kinds of particles inhaled. Siderosis runs a much more rapidly fatal course than many forms of pneumoconiosis. It may cause death in two years, the symptoms being those of rapid phthisis with hectic fever. At the autopsy the lungs present a bright red colour, contain a few nodules or small cavities and are otherwise similarly affected as in silicosis generally.

Cotton, Flax, and Flour-dust.—*Cotton.*—Both in opening the compressed bales of raw cotton and during the manufacturing processes known as 'scutching' and 'carding,' that have for their

object the removal of impurities and the conversion of the fibre into fluff ready for spinning, there is a considerable amount of dust given off which when inhaled causes irritation of the naso-larynx and the mucous membrane of the bronchi. The voice becomes husky and there is cough with expectoration. Cotton-fibres may be detected in the sputa on microscopical examination. Fibrosis of the lung is occasionally established and the case becomes one not unlike ordinary phthisis. In some of the cotton-manufacturing districts the mortality among the workpeople is as high as 9 per 1,000 or 1,088 as against 1,000 employed in other occupations.

Apart from the inhalation of dust and fluff, workers in this particular industry are exposed to the possibility of pulmonary disease from adulteration of the cotton. In order to give tenacity to the thread the material is 'sized,' i.e. passed through a mixture of china clay, such as is used in the manufacture of earthenware. This immersion not only aids the weaving of the cloth, but by adding materially to its weight becomes an adulterant. During the process of weaving it becomes absolutely necessary to have the air of the factory at a high temperature and moist. This artificial atmosphere produces in the workpeople languor, loss of appetite, and anæmia. The vital resistance is reduced, and there is a predisposition to catch cold more readily. The factory hand thereby becomes an easier prey to the irritating china-dust that is given off from the sized cotton, and not infrequently falls a victim to phthisis.

Flax.—During the process of 'scutching' there is given off from flax a fine dust that is even more irritating than that from cotton. Purdon found that workers in the linen-mills of Belfast suffered in large proportion from dry throats, cough, and expectoration often tinged with blood, and that many of the cases terminated in true tubercular phthisis to the extent of 6 per 1000.

Flour-dust.—In the milling of wheat by the old methods of horizontal stones a considerable amount of dust is given off, so that men working in the cloudy atmosphere suffer from pulmonary phthisis far in excess of the ordinary male population. The danger of flour-dust is not so much that it is irritating to the bronchial mucous membrane, as that it is apt to form tenacious plugs, requiring strong expulsive efforts of coughing to dislodge them. Pulmonary emphysema is apt to be induced under these circumstances. In addition to the grains of flour there are given off irritating particles of siliceous dust, owing to the wear and tear of the millstones, particles of hair and bran from the grain, especially oats, so that pathological changes are set up in the lungs, that lead to phthisis. Of one hundred millers, Hirt found that ten were the subjects of pulmonary consumption. Since the introduction of the steel-roller method, the grinding of wheat is conducted within closed chambers, so that the air of a flour-mill is no longer made thick with dust, but is, comparatively speaking, clear. The result of the new method of milling is that pulmonary phthisis has practically disappeared from this industry.

PREVENTION.—If an illustration were required to show how occupational phthisis can be prevented allusion need only be made to coal-mining. Formerly consumption and other forms of pulmonary disease were extremely prevalent among coal-miners, but to-day, thanks to the excellent

system of pit-ventilation, they are seldom observed. Since the various forms of pulmonary disease above alluded to are largely due to working in places where the atmosphere is thick, and to the inhalation of particles of dust, it is clear that much of the illness can be prevented by removal of the dust, by improvement of the ventilation of the workshops through the use of fans and down-draughts, and by the substitution of wet for dry processes of production wherever practicable. Repeated floor-sweeping and the sprinkling of the floors with water during meal-hours should be attended to. In dusty operations the workpeople should wear respirators. Persons of a scrofulous habit, or members of a consumptive family, should not continue to work at a dangerously dusty occupation, but retire and seek employment out of doors. Unfortunately, however, in most of the cases the symptoms do not show themselves until the disease is pretty well established.

SYMPTOMS.—The earliest symptoms are cough, due to laryngeal irritation or spasm, and dyspnoea due to emphysema or other destructive processes in the lung. There is at first expectoration of mucus that is often coloured with the dust that has been inhaled, but later on it becomes muco-purulent. In some of the pneumoconioses hæmoptysis is present, in others it is absent. In the terminal stages tubercle-bacilli may be found in the sputa. The physical signs are those of emphysema of the lungs in some, of induration, excavation and retraction in others. As the malady advances emaciation becomes progressive, and the patient often dies from hectic fever. The disease proceeds so very slowly as a rule that the patient continues to follow his employment for years. On the other hand, it may be that life is quickly terminated by recurrent hæmoptysis and its consequences, or by the super-vention of acute pulmonary inflammation.

TREATMENT.—Removal of the patient from his occupation at once suggests itself; and if the illness is only commencing good may follow this procedure. Too often the pulmonary damage already inflicted is too great to be thus arrested. The treatment under these circumstances becomes therefore one of symptoms, and is practically the same as that for tubercular phthisis, viz. expectorants to relieve cough, tonics to improve the appetite and general health, cod-liver oil to help nutrition, and as far as possible life in the open air.

THOMAS OLIVER.

PNEUMOGASTRIC NERVE, Diseases of.—

SYNON. : Fr. *Maladies du Nerf Pneumogastrique* ; Ger. *Krankheiten des Vagus*.—Of all the cranial nerves, the pneumogastric has the most extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach, and even, in part, the intestines and the spleen. In some of the so-called functional diseases of the organs which it supplies its action is conspicuously deranged. The symptoms of its disease are thus very extensive, and it will be well first to describe them generally, and afterwards to consider in detail those which merit separate description.

Some of the functions of the vagus depend upon fibres of the spinal accessory nerve which join it, but it is convenient to consider these in this article.

The pneumogastric nerve, it will be remembered, arises from the side of the medulla, between the glosso-pharyngeal above, and the spinal accessory

below, and to the outer side of the hypoglossal. The fibres of origin come from a tract of grey matter which is continuous below with the nucleus of the spinal accessory, and above lies (in the *calamus scriptorius*) between the hypoglossal and internal auditory nuclei, while to the outer side of its upper extremity, and more deeply seated, is the nucleus of the glosso-pharyngeal. The trunk of the nerve, after receiving fibres from the spinal accessory, and giving off some small branches (of which the most important is one to the external ear), passes down the neck, behind, and in the same sheath with, the carotid artery; enters the thorax on the right side, over the subclavian artery, and, on the left, between the subclavian and the carotid; passes through the thorax beside the oesophagus; and ends in branches to the stomach, spleen, and intestines. The most important branches are the pharyngeal, which, with the glosso-pharyngeal, form the plexus of the same name; the superior laryngeal; the recurrent laryngeal, which passes back, the left around the arch of the aorta, the right around the subclavian artery; branches to the oesophagus; pulmonary branches which, by means of the pulmonary plexus, supply the lung; and branches which form the cardiac plexus for the heart.

ÆTIOLOGY.—The deep position of the pneumogastric and its branches preserves it from some forms of damage, although its extensive course renders it liable to suffer from many causes. The nucleus in the medulla may be damaged by local softening, hæmorrhage, or slow degeneration; but in all these cases other adjacent nuclei suffer (*see LABIO-GLOSSO-LARYNGEAL PARALYSIS*). The nerve, at its origin from the medulla, may be compressed by thickening of the meninges, growths from the meninges or bones, or aneurysm of the vertebral artery. Affections of the nerve due to syphilis are almost always the result of meningeal disease in this situation. Other adjacent nerves commonly suffer at the same time. The trunk of the nerve is sometimes, but rarely, implicated in punctured or gunshot wounds; incised and lacerated wounds damaging it are usually immediately fatal from lesion of the large blood-vessels to which it is contiguous. In surgical operations the trunk and branches of the nerve are occasionally injured. The trunk has been tied in ligature of the carotid, and divided in the removal of deep-seated tumours. In such operations in the lower part of the neck it is often difficult also to avoid injury to the recurrent laryngeal. In excision of an enlarged thyroid both recurrent laryngeal nerves have been many times excised. Sarcomatous and other tumours, and enlarged glands, may compress or involve the nerve in almost any part of its course; and interference with its function especially occurs from such disease in regions limited by rigid structures, as in the upper part of the neck, near the skull, and in the upper part of the thorax. Aneurysms may compress the nerve or its branches; and the recurrent laryngeal nerves suffer from this cause with especial frequency, because they pass round large blood-vessels. The left suffers much more frequently than the right, because the arch of the aorta is more frequently affected by aneurysm than the subclavian. An enlarged thyroid may compress the recurrent laryngeal nerves, and symptoms due to such compression may vary with the varying size of the tumour. The nerve is, in rare cases, the seat of neuromata. Neuritis of the trunk of the nerve, due to cold, is

supposed to be an occasional cause of symptoms, but such cases are extremely rare. It is sometimes involved in very severe and grave cases of multiple neuritis, especially that due to diphtheria and to beriberi.

SYMPTOMS.—It must be remembered that the vagus nerve, besides containing motor fibres for the pharynx and larynx, is the chief afferent nerve for the respiratory centre. It contains accelerating and inhibitory fibres for this centre, but the former predominate, so that experimental division of the nerve in an animal renders the respirations less frequent but deeper, while stimulation of the divided (central) end quickens the respiration, and may even arrest it in tetanic standstill. The inhibitory fibres are contained chiefly in the superior laryngeal nerve, and their stimulation arrests the respiration in muscular relaxation. The vagus is the inhibitory nerve of the heart; slight stimulation increasing the diastolic periods, and stronger stimulation arresting the action of that organ. On division of the nerve the cardiac contractions are accelerated. It has been said to contain trophic fibres for the heart and lungs. The pneumogastric is an afferent nerve for the vasomotor centre, the action of which is lowered by its stimulation, so that the arteries throughout the body are relaxed. It is the motor and sensory nerve for the oesophagus; the sensory nerve for the stomach; and partly also the motor nerve for the stomach and intestines.

Symptoms due to paralysis of the vagus are more frequently met with than those which result from its irritation. Occasionally both are combined. Laryngeal spasm and vomiting are the irritative symptoms most commonly met with, but occasionally cardiac inhibition occurs. Czermak, for instance, was able to arrest his heart for a few beats at will, by pressing a small tumour of the neck against his pneumogastric. Concato had a patient in whom a similar inhibition could be caused by pressure on the right nerve. The increased frequency of pulse which corresponds to paralysis of the nerve has been several times noted, and has occasionally been associated with diminished frequency of respiration, although the laryngeal paralysis, also resulting, has often obscured the effect on the respiratory movements. Roux tied the trunk of the vagus with the left carotid; instantly respiration was arrested, but the pulse was also retarded. The ligature was immediately relaxed, but the patient died in half an hour. Robert also tied the nerve with the carotid; the patient, who was conscious, immediately called out, 'I am suffocated!' and his voice became hoarse. He recovered, but the hoarseness continued for six months. A good example of interference with the functions of the vagus has been recorded by Guttman. A lad, after diphtheria, presented paralysis of the palate and of one sterno-mastoid. His respiration quickly became reduced to 12 per minute, and very laboured, while his pulse rose to 120, and he died in a few hours. In many other cases a similar change in the pulse and respiration has been noted, and even a pulse-rate of 160–200. In the face of these observations, and of experiments on animals, it is not easy to understand a fact, said to have been observed by Billroth, who excised half an inch of one pneumogastric, which was implicated in a tumour, without any resulting symptoms.

The important central relations of the vagus, above alluded to, cause its derangement to form part of many so-called functional disorders of the central nervous system. Its nucleus forms part of, or is connected with, the respiratory centre, which is conspicuously disturbed in hydrophobia and some other diseases. The phenomena of 'Cheyne-Stokes breathing,' or 'respiration of ascending and descending rhythm,' are probably the result of lowered action of the respiratory or pneumogastric centre (*see* RESPIRATION, Disorders of). This symptom is met with in cerebral hæmorrhage, uræmia, meningitis, and in some cardiac diseases. The central connections of the vagus, in the hemispheres, extend to, or are connected with, those parts which are concerned in emotion, and it is probably through the agency of this nerve that the heart's action is affected in excitement and fear. In many epileptic fits the central representations of the nerve are the parts through which the consciousness is first affected, and hence the so-called 'epigastric aura,' which may seem to ascend to the throat.

A similar disturbance is in all probability the cause of the globus hystericus and of the laryngeal spasm, which are conspicuous in some epileptic and hysteroid seizures. The nerve is closely connected with the centre or nerves for equilibration, so that severe vertigo, on whatever dependent, is often followed by vomiting. The pneumogastric nucleus is contiguous to the internal auditory nucleus, and in the vertigo which results from disease of this nerve, or of the semicircular canals (labyrinthine or aural vertigo), vomiting is very common. The nausea and retching of sea-sickness are probably due to the deranged action of the semicircular canals, in consequence of the motion of the endolymph disturbing their nerve-endings, which in turn affect the pneumogastric centre. It is possible that the connection of the vagus with the equilibratory nerves is by means of the cerebellum, disease of which so constantly causes vomiting, although this connection has not yet been traced. Conversely, gastric disturbance of the vagus is often accompanied by vertigo, especially when combined with pre-existing imperfect action of the auditory nerve.

1. Pharyngeal Branches.—The branches of the pneumogastric which enter the pharyngeal plexus supply the constrictors of the pharynx and the soft palate. Some have asserted that all the pharyngeal branches are derived from the spinal accessory. The branches to the soft palate are certainly derived from the spinal accessory. When one vocal cord is paralysed from disease of the roots of the spinal accessory, the levator palati on the same side is, as a rule, also paralysed, and very often there is palsy of that side of the tongue from damage to the adjacent roots of the hypoglossal nerve. *See* PALATE, Paralysis of.

(1) Paralysis.—**ÆTIOLOGY.**—The most common cause of paralysis of the pharynx is disease of the origin of the nerve in the medulla; such disease commonly also involves adjacent nuclei (*see* LABIO-GLOSSO-LARYNGEAL PARALYSIS). Paralysis may, however, result from meningeal disease outside the medulla, from disease of the bones of the base of the skull, but scarcely ever from disease outside the skull. It occasionally forms part of diphtheritic paralysis.

SYMPTOMS.—The chief symptom is difficulty in

swallowing. Food lodges in the pharynx about the epiglottis, and small particles and liquids may enter the larynx. If the paralysis is limited to the superior constrictor, liquids may, it is said, be forced up into the nose by the contraction of the middle constrictor; but it is doubtful whether this occurs unless the palate also is paralysed. The affection of one nerve causes only slight trouble in deglutition, no doubt because of the circular arrangement of the muscular fibres.

DIAGNOSIS.—The only conditions with which paralysis of the pharynx can be confounded are spasm and organic disease. The writer once saw an elderly man with distinct pharyngeal paralysis, who had been sent to an eminent surgeon because the difficulty in swallowing was supposed to indicate cancer of the throat. A careful examination is usually sufficient for the distinction.

(2) Spasm.—Spasm of the pharynx may be recognised by its paroxysmal character, and is almost always part of 'functional' nervous disease. It forms part of the spasm of hydrophobia, and occurs in hysteria, and in some other allied states. Individuals are sometimes met with who are unable to take food except when alone, so great is the amount of pharyngeal spasm which results from the emotion induced by the presence of other people. Other emotional states may have a like effect; in fear and intense grief swallowing may be impossible partly from this cause.

2. Laryngeal Branches.—It will be remembered that, of the two laryngeal nerves, the superior is the sensory nerve for the larynx, and also supplies motor power to the crico-thyroid muscle, which is the tensor of the cords; while the recurrent laryngeal is purely motor, and supplies the other muscles. The motor fibres of both are derived from the spinal accessory. Of the muscles, the most important in regard to paralysis are the chief abductor, the posterior crico-arytænoideus (which draws the postero-external angle of the arytenoid cartilage backwards, and so moves the processus vocalis outwards); the chief adductor, the lateral crico-arytænoideus (which draws the postero-external angle of the arytenoid cartilage outwards, and thus the processus vocalis inwards); and the arytenoideus (which approximates the two arytenoid cartilages). Other muscles, acting at the same time, increase the power of closure. The paralyzes and spasm of the larynx are described in a separate article. *See* LARYNX, Diseases of.

3. Pulmonary Branches.—The muscular fibres of the bronchi are innervated by the nerve, and their paroxysmal contraction in asthma is believed to be produced through its agency. It has been asserted that the plain muscular fibres, said to exist throughout the lung-tissue, are supplied by it (Gerlach), and their contraction has been assumed to explain a peculiar form of emphysema, which has been observed in compression of the pneumogastric (Tuczek); but, since deep breathing of a costo-superior type was observed, it is possible that the effect is the result of the energetic respiration from the disturbance of the centre. The pneumogastric is commonly believed to contain vaso-motor fibres for the vessels of the lungs, but Brown-Séquard and Franck have separately shown that these fibres are contained, not in the vagus, but in the sympathetic. Vascular lesions of the lungs have, however, been observed after section of

the vagus. Michaelson noted rapid congestion and hæmorrhage, and such congestion is sometimes noted after lesions of the pons. In a case of hæmorrhage into the pons, fatal in two hours, the writer found intense congestion with extravasation into the left lung, and hæmorrhages in the left extremity of the stomach.

After section of the vagus, animals die from chronic pneumonia, and hence the vagus has been supposed to be a trophic nerve for the lungs. But the changes have been accounted for by the entrance into the bronchi of food from the pharynx, in consequence of the obstructive paralysis of the œsophagus, and the paralysis of the larynx (Traube, Steiner). All admit that this is one cause of the pulmonary affection, but differ as to its adequacy.

4. Cardiac Branches.—The inhibitory effect of irritation, and the acceleration of the heart's action which results from lessened action of the vagus, have been before alluded to. Increased frequency has been several times observed in cases of local disease of the vagus in the thorax, compression by mediastinal tumours, &c. In a case of phthisis, for instance, in which the pulse was at first occasionally, and afterwards constantly, frequent (130-148), Meixner found the left vagus enclosed in a mass of enlarged glands in the upper opening of the thorax. The vagus is also the afferent nerve from the heart, and although we are normally unconscious of the cardiac action, some of the disordered sensations of disease are apparently produced through its agency. The subject of angina pectoris, and its relation to the vagus, is discussed in a separate article, but it may be here noted that in some anginal attacks the heart's action is, for a time, arrested or retarded, and that in a few cases these symptoms have been found associated with organic disease of the cardiac plexus. Thus in a case in which, during paroxysms of intense anginal anguish, the heart's action was arrested for four or six pulsations, Heine found a tumour involving the cardiac plexus. In a case recorded by Blandin, anginal attacks were associated with a small tumour of the vagus. Further, there are afferent fibres from the heart inhibiting the action of the vaso-motor centre, and these are probably stimulated in some anginal seizures. See *ANGINA PECTORIS*.

After disease or injury of the vagus, the heart has been found in a state of fatty degeneration, and hence it has been thought that the vagus contains trophic fibres for the cardiac substance.

5. Branches to the Alimentary Canal. The branches to the œsophagus are rarely diseased except in cases of affection of the nerve-trunk or of the centre. In very rare cases such disease has caused difficulty in swallowing, simulating stricture. Spasm of the œsophagus is more frequent. The vagus is the sensory, and in part the motor nerve for the stomach. Its fibres are very sensitive to any local irritation, and not rarely the seat of spontaneous neuralgia. Hunger is generally believed to be a pneumogastric sensation, and complete loss of the sensations of hunger and thirst was noted in a case of softening of the root of the vagus from an aneurysm of the vertebral artery (Johnson). Appetite, however, is not always lost in animals when the pneumogastriacs have been divided (Reid). In some cases of disease of the nerve, excessive hunger has been noted. This symptom, for instance, was present, in one case, in conjunction with dyspnœa,

noisy breathing, and vomiting of unaltered food; *post mortem*, both pneumogastriacs were found atrophied (Swan). In another case of insatiable appetite, small neuromata were found on the nerve. It is possible that the polyphagia may be in part the result of the defective digestion of food.

The pneumogastric is also in part the motor nerve of the stomach; after its section the contractions of the organ are lessened, although not altogether arrested. Vomiting is probably produced through its agency, by varied reflex and central irritation. In the latter case (as in meningitis) the vomiting is sometimes extremely rapid. The writer has known paroxysmal vomiting to result from the intermitting pressure of a tumour on the vagus; and Boinet, having exposed the vagus in an operation in the neck, noted that whenever he touched the nerve the patient vomited.

The vagus accelerates the contraction of the intestines, but no intestinal symptoms have been observed to result from its disease.

GENERAL DIAGNOSIS.—The chief symptoms on which the diagnosis of disease of the vagus, in any given case, would rest, are the laryngeal paralysis; retarded respiration; accelerated or retarded heart; and vomiting. The diagnosis of the seat of the disease rests upon the range of the symptoms, and the associated morbid processes. Disease of the trunk of the vagus is much less common than disease of its branches or roots. Paralysis of one vocal cord, for instance, is almost always the result of pressure, either on the recurrent laryngeal nerve, or on the roots of the spinal accessory at the medulla. Bilateral symptoms are usually due to central disease, or else (if slight) are of merely local origin. In most cases of pressure on the trunk and branches of the vagus the cause of the symptoms is distinct, the only exception being deep-seated tumours in the thorax.

PROGNOSIS.—The prognosis is that of the cause of the disease, and is sufficiently discussed in other articles.

TREATMENT.—Little can be said on the general treatment of the diseases of the pneumogastric, since it depends on the different conditions to which the symptoms are due, and which are described elsewhere. Central disease, and causes of pressure on the nerve, are, as a rule, beyond the range of treatment. Whenever there is reason to suspect pressure on the nerve-roots (from the combination of paralysis of the tongue, palate, and one vocal cord), iodide of potassium should be given, since this condition is more frequently due to syphilis than to any other cause. In laryngeal paralysis the local application of electricity is sometimes useful, but more so in the weakness which depends on local causes than in that which is due to nerve-lesions. Injections of strychnine are of especial importance in degenerative diseases, even when its administration by the mouth is without effect. In central paralysis the treatment will depend on the indications given by the mode of onset regarding the nature of the lesion, whether acute softening or degeneration. In all spasmodic affections, sedative inhalations, as chloroform, are useful; and of especial service is the diminution of the afferent impressions from the larynx such as may be produced by the application of cocaine. Bromide of potassium and morphine alone lessen, in effective degree, the irritability of the nerve-centre.

W. R. GOWERS

PNEUMOGRAPH (πνεύμων, the lungs; and γράφω, I write).—An instrument for recording the movements of respiration. See PHYSICAL EXAMINATION.

PNEUMONIA (πνεύμων, the lungs). Inflammation of the substance of the lungs. See LUNGS, Inflammation of.

PNEUMO-PERICARDIUM (πνεύμα, air; and περιкарδιον, the pericardium).—A collection of gas in the pericardium. See PERICARDIUM, Diseases of.

PNEUMOTHORAX (πνεύμα, air; and θώραξ, the chest).—A collection of gas in the cavity of the pleura. See PLEURA, Diseases of; and LUNGS, Perforation of.

POCK.—A popular term for pustule, as though a pocket or pouch in the skin filled with pus. From the plural of pock is derived *pox*; hence, *small-pox*, *chicken-pox*, the *great pox* or *venereal pox*, and so forth.

PODAGRA (πούς, the foot; and ἄγρα, a seizure). A common synonym for gout, as it usually attacks the foot. See GOUT.

PODALGIA (πούς, the foot; and ἄλγος, pain).—A name for pain in the foot, due to any cause, such as gout, rheumatism, &c.

POISONOUS ANIMALS.—See VENOM AND ITS EFFECTS; VENOMOUS ANIMALS.

POISONOUS FOOD.—See FOOD, POISONOUS.

POISONOUS GASES.—See CARBONIC ACID, Poisoning by; CARBONIC OXIDE, Poisoning by; PRUSSIC ACID, Poisoning by; &c.

POISONS.—SYNON.: Fr. *Poisons*; Ger. *Gifte*. DEFINITION.—There is no legal definition of the word *poison*, and the definitions usually proposed are apt to include either too much or too little. Generally a poison may be defined as a substance having an inherent deleterious property, which renders it capable of destroying life by whatever avenue it is taken into the system. Substances which act only mechanically, such as powdered glass, are not poisons. In popular language, a poison is a substance capable of destroying life when taken in small quantities. A poison, then, may be defined as any substance which, when introduced into the system, or applied externally, injures health or destroys life irrespective of mechanical means or direct thermal changes. See FOOD, POISONOUS.

ACTION.—Poisons may exert a twofold action. Their action is either local or remote, or both local and remote. The local action of a poison is usually one of corrosion, inflammation, or an effect on the nerves of sensation or of motion. The remote actions are usually of a specific character, though some writers group the remote effects of poisons under two heads, and speak of the common and specific remote effects of a poison. The local actions of a poison of the corrosive class are usually so well marked, and so easily recognised, that the fact of its administration is obvious. The same may be said, in a lesser degree, of the irritant poisons, especially the mineral irritants; but here the symptoms often so closely simulate those of natural disease, as to render the diagnosis a matter

of great difficulty. An accurate acquaintance with the remote specific effects of the various common poisons is indispensable to the medical practitioner. The class of poison which has been administered or taken will thus be suggested to his mind by the symptoms observed, and not infrequently the specific poison will be suspected. In this way the physician may often be at once able to diagnose, from the symptoms alone, the administration of strychnine, belladonna, or cantharides. Great care must be taken, however, not to draw a rash conclusion from one symptom; as, for instance, from the tetanic spasms which are so marked a feature in strychnine-poisoning.

It is now universally held that absorption is necessary in order that a poison should be able to exert its specific effect.

MODIFYING CIRCUMSTANCES.—The usual action of poisons may be greatly modified—(1) by the largeness of the dose, and the state of aggregation, admixture, or chemical combination of the poisons themselves; (2) by the part or membrane to which they are applied; and (3) by the condition of the patient. Thus, for example, opium may be a medicament or a poison, according to the dose in which it is given; and a dose of opium which may be beneficial to an adult, in certain states of the system, may be fatal to a young child, or to the adult when suffering, for example, from Bright's disease. All barium-salts are poisonous, except the sulphate, which is one of the most insoluble of all mineral substances. The simple cyanides are highly poisonous, and the same may be said of many double cyanides. But the double cyanide of iron and potassium (potassium ferro-cyanide) is almost without action on the system. The part or tissue to which a poison is applied must obviously greatly affect the activity of a poison, owing to the varying rapidity with which absorption takes place through the cutaneous, mucous, serous, and other surfaces of the body. Curare may be swallowed in a considerable dose without producing any appreciable effect, while a small quantity of the same substance introduced into a wound will speedily prove fatal. It has been found that when a poison is slowly absorbed, so that it can be either disposed of in the system or again excreted more rapidly than it is absorbed, no poisonous results ensue; but when absorption occurs so quickly that the poison can neither be excreted nor destroyed in the system as rapidly as it is absorbed, the specific effects of the poison are developed. Curare, for instance, is absorbed by the gastric mucous membrane more slowly than it is excreted through the kidneys. But if the renal arteries be ligatured, the poison accumulates in the blood, and the specific effects of the poison are developed, just as when curare is introduced into a wound.

Idiosyncrasy has much to do with the poisonous or hurtful character of a substance. Thus pork, mutton, certain kinds of fish, notably shell-fish, and fungi (see MUSHROOMS, Poisoning by), have, under certain circumstances, and in certain persons, produced all the symptoms of violent irritant poisoning; while others, who have partaken of the same food at the same time, have enjoyed perfect immunity. More commonly, all who partake are affected, but with varying degrees of severity. Some persons are said, on good authority, to be capable of taking with impunity such violent poisons as corrosive sublimate or opium in enormous doses,

and this independently of habit, which is known to have such a large influence in modifying the effects of some poisons, notably of the narcotics. A tolerance of poisons is sometimes engendered by disease, so that a poison may from this cause fail to produce its accustomed effect. Thus opium is largely tolerated in tetanus, and in mania from drink; and mercurial compounds may in severe febrile affections fail to produce the usual constitutional effects of the metal. On the other hand, kidney-disease, by impeding elimination, may intensify the ordinary effects of a poison; and the like is observed when opiates are given where there is a tendency to cerebral congestion.

EVIDENCE.—In order to raise a valid inference in the mind of a medical attendant that poison has been administered to a patient, certain facts must be brought under his notice; and without the concurrence of at least two or more of these, the actuality of poisoning cannot be maintained. The sources of evidence in cases of suspected poisoning are the *symptoms*; the *post-mortem appearances*; *chemical analysis* of articles of food or drink, or of the body and the excretions; and *experiments upon animals*. The evidence derived from these sources, being compared with the known properties and effects of various poisons in authenticated cases, will enable the physician to form a correct opinion as to the probable administration or not of a poison. The poisons most commonly taken or administered are opium and morphine, carbolic acid, arsenic in various forms, phosphorus, oil of vitriol, strychnine, and oxalic acid.

It is rarely that the *symptoms* exhibited during life do not afford some clue to the cause of illness; and most frequently the symptoms are all that the medical attendant has to guide him to a diagnosis of the nature of the case, during the lifetime of the patient. Sometimes, however, persons are found dead as the result of poison, concerning the manner of whose death nothing whatever can be learned; a suspicion of poisoning arising from the circumstances under which the corpse is found. Here the aid of chemical analysis ought invariably to be invoked; and fortunately in these cases the delay involved in making an analysis is of comparatively little moment. The effects may in the case of many persons be either suddenly or slowly manifested; hence we have *acute* and *chronic* poisoning. Cases of chronic poisoning are usually the result of the repeated administration of small doses of lead, copper, mercury, phosphorus, or arsenic. All of these poisons are treated of in separate articles. The general conditions which should excite a suspicion of poisoning are the sudden onset of serious and increasingly alarming symptoms in a person previously in good health, especially if a prominent symptom be epigastric pain; or where there is complete prostration of the vital powers, a cadaverous expression of the countenance, an abundant perspiration, and speedy death. In all such cases the aid of the chemist is required, either to confirm well-founded, or to rebut ill-founded, suspicions.

CLASSIFICATION.—Various attempts have been made to classify poisons rationally. Perhaps the best classification, for the purposes of the medical practitioner, is that which groups poisons according to the more obvious symptoms which they produce. Our knowledge of the more intimate action of many poisons is still too slight to admit of any

useful classification according to the manner in which they specifically affect the vital organs.

Poisons may in the manner indicated be classified as: (1) **Corrosives**; (2) **Irritants**; and (3) **Neurotics**. It is perhaps at present premature to attempt a systematic division of the last class. The class of neurotics embraces poisons so widely different in their action as opium and strychnine.

1. Corrosive Poisons. — ENUMERATION. —

The action of one of the most typical of these poisons, corrosive sublimate, is fully considered under a special head (*see* MERCURY, Diseases arising from). The most commonly administered corrosives are the mineral acids—sulphuric, nitric, and hydrochloric; oxalic acid; the alkalis—potash, soda, and ammonia; acid, alkaline, and corrosive salts—such as acid potassium sulphate, potassium carbonate, chlorides of zinc, tin, and antimony, and silver nitrate.

SYMPTOMS.—The mineral acids and the alkalis have no specific effect on the system, their action being at first almost purely local. Some of the other corrosives enumerated may have, besides their local effects, a remote and constitutional action. The symptoms of corrosive poisoning are marked and unmistakable, except when the patient is an infant. Immediately after swallowing the corrosive substance, an acid, caustic, or metallic, burning sensation is felt in the mouth, fauces, gullet, and stomach; and this speedily extends over the whole abdominal region. Vomiting is speedy, or may, rarely, be altogether absent. The vomited matters consist at first of the ordinary contents of the stomach, more or less altered by the action of the poison. In the case of *mineral acids* they are intensely acid, and cause copious effervescence when they fall upon limestone or marble. No relief is afforded by the evacuation of the stomach; and later the vomited matters may be more or less mingled with altered blood, which may be dark, or even black. Shreddy mucus, casts of the gullet or stomach formed by the shedding of the mucous membrane, and sometimes even the muscular wall of the œsophagus, are ejected. The abdominal pain is not relieved, but greatly aggravated, by pressure. The whole abdomen becomes distended, owing to the gases evolved by the action of the poison; the diaphragm is pressed upon, and intense dyspnoea may result, owing to pressure upon the thoracic viscera. When a mineral acid has been administered, there is little or no action of the bowels, and the urine may be suppressed; but in poisoning by the *alkalis*, and by the *alkaline carbonates and sulphides*, there may be purging. The mouth, tongue, and fauces exhibit the local effects of the corrosive: a yellow coating in the case of nitric acid; white at first, and as if covered with white paint, from sulphuric acid; and whitish or brown and less thickly coated from hydrochloric acid. Yellow or brown stains may be observed on the skin, extending downwards from the angles of the mouth, and caused by the trickling of acid or other corrosive fluid from the mouth. Meantime the symptoms develop rapidly. The pain, thirst, dyspnoea, and dysphagia increase. The patient, at first excited, with rapid bounding pulse, becomes bathed in cold perspiration, the countenance becomes pinched, the pulse more rapid and thready. Enormous eructations of gas take place, but these afford no relief. The patient may become more or less cyanosed; but this will depend

upon the amount of dyspnoea. The intellect is usually clear to the last. Signs of collapse come on, and the patient may sink within a period varying from six to twenty-four hours. If recovery does not take place, death usually supervenes within a period of twelve to twenty-four hours. Very frequently, and more especially in poisoning by oil of vitriol, the patient survives the first acute symptoms only to perish months after, should not the aid of the surgeon be invoked and gastrostomy be performed, by slow starvation, due to local injury to, and subsequent stricture of, the oesophagus. The use of bougies in these cases, to keep the gullet patent, seldom affords permanent relief.

When *nitric acid*, or *ammonia*, is the poison taken, the vapours of the acid or of the ammonia may gain access to the air-passages and lungs, provoking inflammation, which is commonly fatal. The dyspnoea and chest-symptoms will be greatly aggravated in these cases, and may overshadow the more usual symptoms due to local action on the digestive canal. In poisoning by the *caustic alkalis* (potash- and soda-lyes) diarrhoea, with discharge of blood, is more common than the constipation observed in poisoning by the mineral acids. Entire suppression of urine, or anuria, is the rule in poisoning by corrosive sublimate.

Oxalic acid in concentrated solution is undoubtedly a corrosive and irritant poison. Very commonly, however, it kills by its depressing action upon the heart before symptoms of corrosion have become prominent; or the vomiting, pain, and other more immediate symptoms of corrosive poison are associated with a feeble pulse, clammy skin, nervous symptoms, aphonia, and speedy death, even within ten minutes of the administration of the poison. To quote Christison's language: 'If a person, immediately after swallowing a solution of a crystalline salt, which tasted purely and strongly acid, is attacked with burning in the throat, then with burning in the stomach, vomiting, particularly of bloody matter, imperceptible pulse, and excessive languor, and dies in half an hour, or still more in twenty, fifteen, or ten minutes, I do not know any fallacy which can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly and terminates so soon, and no other crystalline poison has the same effects.' It must be added that binoxalate of potassium, and the soluble oxalates generally, are as poisonous as the acid itself.

ANATOMICAL CHARACTERS.—The distinction between corrosive and irritant poisons is by no means well marked; and indeed corrosive poisons, when diluted, act as irritants. Hence we shall describe the *post-mortem* appearances of corrosive poisoning under the head of Irritant Poisons.

DIAGNOSIS.—The diagnosis of corrosive poisoning rarely admits of difficulty; and in any obscure cases chemical analysis will remove all doubt.

2. Irritant Poisons.—Irritant poisons are of two classes—*metallic irritants*, and *vegetable and animal irritants*, these latter being grouped together. Perhaps none of them, however, act as pure irritants; and the irritant symptoms which they produce are most commonly accompanied by a well-marked effect upon the nervous system also. An irritant is a poison which causes inflammation of the part to which it is applied, usually the alimentary canal. By far the most important of the metallic irritant poisons is arsenic (*see ARSENIC, Poisoning by*).

Other metallic irritants are the salts of antimony, zinc, and other metals. Elaterin, essential oils, and gamboge may be cited as examples of vegetable irritants; and cantharides of animal irritants. Irritant animal and vegetable foods are separately described. *See FOOD, POISONOUS.*

SYMPTOMS.—Irritants differ as a rule from corrosive poisons in the greater slowness with which the symptoms are developed. Usually when an irritant is swallowed, after an interval—greater or less according to the specific character of the poison—a burning pain is felt, and a sense of constriction of the mouth, throat, and gullet, speedily followed by sharp burning pain in the epigastrium; and this is increased by pressure—a mark which serves to distinguish the attack from one of ordinary colic. Nausea, vomiting, and great thirst ensue; speedily followed by pain and sense of distension of the whole abdomen, which is exceedingly tender, and perhaps visibly distended. Most commonly the vomiting is followed by purging, tenesmus, dysenteric stools, and often by dysuria. Should the poison not be speedily removed from the system by vomiting and purging, these continue unrelieved, and increase in severity; and symptoms of inflammatory fever, or it may be of collapse, supervene. The pulse becomes rapid, small, and thready; the countenance is anxious; the skin is bathed in perspiration, now warm, and again cold and clammy. The patient may never rally from the first shock to the nervous system; more rarely, having survived this, he dies in convulsions; or he may perish of inanition after more protracted sufferings. It must be borne in mind that those irritant poisons—such as diluted sulphuric acid—which, when taken in a more concentrated form, act as corrosives, may bring about starvation, necessitating such operative procedure as gastrostomy, by the injury which they inflict upon the oesophagus and stomach. Death after the administration of an irritant poison may, it is obvious, occur at very varying periods after the ingestion of the poison.

DIAGNOSIS.—Irritant poisoning may be mistaken for various forms of natural disease. The diseases with which it is most apt to be confounded are—gastritis; gastric ulcer, with or without perforation; peritonitis; severe colic; sporadic and Asiatic cholera; and rupture of the stomach or intestines. A careful examination of the patient, and the history of the case, will often remove any doubt which may be entertained; but microscopical examination and chemical analysis of the ejecta of the patient will frequently afford the only means of clearing up the case during life. Too frequently irritant poison is not suspected until a *post-mortem* examination is made. In every case where a possibility of irritant poisoning is suggested, the aid of analysis should be invoked. For the diagnostic differences—so far as differences in symptoms are diagnostic—between irritant poisoning and the special diseases above mentioned, the reader is referred to the special articles in this Dictionary.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances in irritant and corrosive poisoning are corrosion of the mouth, fauces, gullet, and stomach, the mucous membrane being shrivelled, altered in consistence and colour, and more or less detached; irritation and inflammation of the stomach and first portion of the small intestines; ulceration; and erosion. In corrosive poisoning the stomach may be perforated, the edges of the aperture being

shreddy; and in the case of sulphuric acid the viscera may be blackened (altered blood) from the action of the acid upon the blood-pigment. The small intestines are implicated to a varying extent, or may altogether escape. The large intestine is often attacked, and this is more especially the case in poisoning by mercurial preparations. Arsenic exerts a specific effect upon the gastric mucous membrane. Remains of irritants may be detected in the intestinal canal, and be recognised by their physical, microscopical, and chemical characters.

3. Neurotic Poisons. — **ENUMERATION.** — Under this head may be ranged a great number of poisons, having this in common, that the symptoms produced by them are more or less prominently associated with the nervous system. The class embraces pure narcotics, such as opium and morphine; chloral hydrate; chloroform; hyoscyamus; digitalis; strychnine; prussic acid; nitro-benzol; phenol (carbolic acid); alcohol; aconite; belladonna, and many others.

SYMPTOMS. — These are necessarily of the most varied character. All that has been already said about the onset of symptoms, their character, and the circumstances under which they have appeared, must be borne in mind in arriving at a diagnosis.

Prussic acid produces its effects in the course of a few minutes; or, it may be, seconds. The course of symptoms is very rapid; and death may be well-nigh instantaneous. The symptoms are convulsions, great disturbance of respiration, with prolonged expiration, dilated pupils, and cyanosis. *See PRUSSIC ACID, Poisoning by.*

Morphine and Opium, after a stage of excitement, produce deep comatose sleep, with slow stertorous breathing; contracted pupils; and clammy perspiring skin; all the other secretions being more or less suppressed. *See OPIUM, Poisoning by.*

Aconite is diagnosed by the peculiar numbness and tingling of the skin which it produces. *See ACONITE, Poisoning by.*

Belladonna, and its alkaloid *Atropine*, widely dilate the pupils, with indistinct vision, and cause intense thirst, mirthful delirium, spectral illusions, flushed face, pyrexia and erythema (p. 421).

Chloroform and *Alcohol* in toxic doses produce profound insensibility; and are, moreover, generally more or less recognisable by circumstances, some of which will be found described under **ALCOHOLISM**.

Nitro-benzol causes symptoms often indistinguishable from those of prussic acid; but in consequence of its insolubility, and the slowness with which the liquid poison is absorbed by the gastro-intestinal mucous membrane, there is often a prolonged interval between the administration of the poison and the onset of alarming symptoms.

Chloral hydrate causes death after a stage of unconsciousness; and there is scarcely any difficulty in ascertaining the nature of the case by the aid of the surroundings of the patient. *See CHLORAL HYDRATE, Poisoning by.*

Carbolic acid or *Phenol* whitens and shrivels the mucous membranes with which it comes in contact, and not only acts as a corrosive, but produces speedy narcosis, and greenish or black urine. The peculiar odour of phenol is always perceptible, though not infrequently overlooked.

DIAGNOSIS. — It is impossible to enter fully into the diagnosis of each individual neurotic poison. The most frequent and important diagnoses have to be made in supposed cases of poisoning by

opium, alcohol, and strychnine respectively (*see p. 338*).

In opium-poisoning the equally contracted pupils; the possibility of rousing the patient by means of external stimuli in all except the later stages—as, for instance, by flicking the feet, the application of the electric current, &c.; and the moist clammy skin, may serve to prevent the case being confounded with one of apoplexy. In alcoholic coma there is great danger of mistaking the nature of the case, in consequence of the frequency with which the alcoholic odour may be met with in cases where alcohol has been taken, either dietetically or medicinally, in moderate or somewhat immoderate doses. The very careful use of the stomach-tube can do no harm, and may not only save the patient if the case be one of alcoholic poisoning, but also serve to clear up the diagnosis. The tetanic spasms of strychnine will have to be differentiated from those of true (traumatic) tetanus. In this there is not usually any insuperable difficulty. Strychnine convulsions are intermittent; do not begin in the lower jaw; are, as a rule, opisthotonic in character, and do not affect the same groups of muscles as are implicated in true tetanus. *See OPIUM, Poisoning by; and STRYCHNINE, Poisoning by.*

TREATMENT. — Only the general principles of the treatment of poisoning can be indicated here. The treatment in poisoning by the most important special poisons is described in separate articles. The question of the use or non-use of the stomach-pump or syphon-tube must be decided by the nature of the poison administered. Where one of the concentrated mineral acids, a caustic alkali, a corrosive salt, oxalic acid in concentrated solution, or carbolic acid, has been swallowed, it is generally held that the stomach-pump should not be used, the danger of perforation of the gullet or stomach being considerable. The soft œsophageal syphon-tube may, however, be nearly always used with safety. In all cases where a non-corrosive poison has been taken, except in the case of prussic acid, where the course of the poisoning is too rapid to permit of the use of the instrument, the application of the tube is advisable and can do no harm; in cases of poisoning by opium and alcohol, the greatest reliance must be placed on evacuation of the stomach by its aid. The corroding acids may be neutralised by the administration of lime-water, or, still better, saccharated lime-water; highly diluted solutions of the caustic alkalis; or, failing these, the continuous use, in frequently repeated doses, of chalk, whiting, or the alkaline carbonates—so as to avoid dangerous distension of the abdomen with carbonic acid gas. On the contrary, the caustic alkalis may be neutralised by the copious imbibition of highly diluted acid liquids. Failing the use of the stomach-tube, or even after the use of this, emetics may be administered, or apomorphine injected. The prompt administration of an emetic is rarely inadmissible. The effects of corrosives and irritants must afterwards be met by appropriate remedies, such as demulcents and oil to sheath the mucous membranes, opiates to relieve pain, &c. The effects of oxalic acid cannot be avoided by the administration of alkalis and alkaline carbonates, for the alkaline oxalates are themselves highly poisonous. Chalk, whiting, and soluble lime-salts precipitate oxalic acid as an insoluble calcium oxalate, and form the best remedies. Soluble sulphates are antidotes for carbolic acid. Oil greatly allays the

intolerable pain attending the local action of this acid. In prussic-acid poisoning artificial respiration, persistently used, is our sheet-anchor, and may be supplemented by galvanism, alternate douches of warm and cold water, and other measures. After the use of the stomach-pump to remove unabsorbed opiates, stimulating liquids containing tannic acid, such as strong black coffee, may be given; and the patient must be kept awake by walking him about, flicking the feet with towels, the application of the faradic current, &c. Atropine in full doses is in some respects antagonistic in its physiological action to opium. Conversely, opiates are regarded as direct antidotes to belladonna. Potassium permanganate destroys morphine. On the same principle of counteracting effects, digitalis and aconite are counter-poisons, and hence antidotes the one to the other. The happiest results have followed the use of full doses of chloral hydrate in strychnine-poisoning; and chloroform may be freely inhaled to allay the tetanic spasms. In alkaloidal poisoning, except where a tetanising poison, such as strychnine or brucine, has been given, the stomach-pump must be employed; and emetics and tannic acid, in the form of tincture of galls, strong black coffee, or strong tea, should also be given, with the object of precipitating the alkaloid as an insoluble tannate. Hypodermic injections of strychnine are invaluable where there is cardiac or respiratory failure.

THOMAS STEVENSON.

POLIO-ENCEPHALITIS.—Inflammation of the cerebral cortex. This is the assumed cause of some cases of infantile hemiplegia, preceded by convulsions and followed by marked rigidity of the paralysed parts. See PARALYSIS, INFANTILE CEREBRAL.

POLYCHROMATOSIS.—A term applied to a condition of the red-corpuscles in which the normal staining-reaction of the hæmoglobin is diminished or lost. See p. 155.

POLYDIPSIA (πολύς, much; and δίψα, thirst).—A synonym for excessive thirst; sometimes used for diabetes. See DIABETES MELLITUS; POLYURIA; and THIRST.

POLYNEURITIS.—See NEURITIS, MULTIPLE.

POLYPHAGIA (πολύς, much; and φάγω, I eat). A synonym for excessive hunger. See APPETITE, Disorders of; and PNEUMOGASTRIC NERVE, Diseases of.

POLYPUS (πολύς, many; and πούς, a foot).—**SYNON.**: Fr. *Polype*; Ger. *Polyp*.

DEFINITION.—This term is generally applied to any simple pedunculated growth, springing from a mucous surface; but it is sometimes extended so as to include malignant pedunculated growths in similar situations.

VARIETIES.—It is clear that no single description will apply to each member of the class. Hence it will be sufficient to enumerate the principal varieties of polypus, a fuller account of most of which will be found in the article TUMOURS, and also in the articles dealing with the diseases of the several organs which they affect.

1. **Polypi of the Nose.**—These are of two varieties—the *mucous* and the *fibrous*; both are classed among the fibromata. Both are covered

with ciliated epithelium. The fibrous variety often involves the structures at the back of the pharynx, forming the so-called *naso-pharyngeal polypus*.

2. **Polypi of the Ear.**—Polypi of the ear resemble polypi of the nose, but present a variety of structure, as some spring from the membrana tympani, others from the interior of the tympanum.

3. **Polypi of the Intestines.**—These polypi are of much more frequent occurrence in the rectum than in any other portion of the intestinal tract. They are composed of tissue resembling that of the mucous membrane of the part, and are described among the adenomata.

4. **Polypi of the Uterus.**—These growths are of three kinds, namely: (a) *cystic*, which are derived from the oviducts of Naboth; (b) *mucous* or *soft*, resembling the polypi of the rectum; (c) *hard* or *fibrous*, consisting of pedunculated fibro-miomata.

5. **Polypi in other situations.**—Less common forms of polypi, consisting of some modification of the mucous membrane from which they are derived, are found in the bladder, the larynx, on the gums, or sometimes in the sinuses communicating with the nose.

TREATMENT.—Though polypi differ somewhat in structure, the treatment of the simple varieties of the class is the same—that is, if removal be considered advisable. Either the pedicle may be grasped and the tumour removed by avulsion; or it may be divided at a stroke by some sharp instrument, or cut through slowly or rapidly by some form of écraseur or ligature.

In removing a malignant polypus the rules to be followed are the same as in the removal of malignant growths elsewhere, see p. 214. See CANCER.

R. J. GODLEE.

POLYSARCIA (πολύς, much; and σάρξ, flesh).—A term for excessive corpulence or obesity. See OBESITY.

POLYURIA (πολύς, much; and οὖρον, urine).—**SYNON.**: Diabetes Insididus; Fr. *Polyurie*; *Diabète Insidide*; Ger. *Zuckerlose Harnruhr*.

DEFINITION.—A malady (or group of maladies) characterised by thirst and a persistently excessive flow of watery urine, which has a low specific gravity and contains neither albumen nor sugar.

Attempts have been made to subdivide this group into smaller sections. One such section is *polydipsia* or *hydruria*, having the characters above specified as those of polyuria; another is *azoturia*, where the solids, especially urea, are in excess of the normal amount; and a third, *anazoturia*, where these are markedly deficient. *Azoturia* has been made to include all cases where urea is unusually abundant, even where the urine is scanty, as in fevers; and will not be discussed in this article. *Anazoturia* very rarely occurs; for, notwithstanding the low specific gravity of the urine in diabetes insipidus, owing to the large amount passed, the quantity of urea may, and often does, exceed that excreted in health. A form of polyuria, often slightly marked, has been described as ‘phosphatic diabetes,’ on account of the excess of phosphates passed. See PHOSPHATURIA.

ETIOLOGY.—Diabetes insipidus is a rare disease. Its incidence is unaffected by conditions of age or sex. It may exist in the new-born infant, and it may be found in the patient of seventy, but on the whole it is a disease of early adult rather than of

advanced life, while it is about twice as frequent in males as in females. Nothing is more marked in connection with the causation of diabetes insipidus than heredity. In a case recorded by Gee, the disease was directly transmitted through four generations. Some members of the family escaped, but some of the children of each of those who escaped were attacked.

Beyond inheritance, nothing very definite can be stated as to the cause and origin of diabetes insipidus. It is often connected with nervous affections or nervous excitement, and sometimes follows upon injuries to the head or disease of the brain. In many cases there is a family history of tuberculosis. Alcoholic excesses, the drinking of large draughts of cold fluids, and sudden exposure to cold, have all been credited with the production of the disease. Beyond these, no valid cause can be assigned; often indeed the disease comes on without even such insufficient reasons as those given above, some of which have doubtless been assigned on the '*post hoc*' principle.

SYMPTOMS.—When diabetes insipidus follows accident or mental emotion, its onset is usually abrupt; in such cases it may disappear with equal suddenness—sometimes as the result of intercurrent febrile disease. During its continuance *unquenchable* thirst (see **THIRST**) and pale watery urine are the two prime symptoms, for there may be little wasting, and the general health may be good. Occasionally there is increased appetite. Usually the bowels are confined and the skin dry. Pruritus and boils, so common in diabetes mellitus, are exceptionally met with in cases of diabetes insipidus, but the skin soon presents an atrophied, dry, withered appearance. Impotence is a well-marked symptom, and amblyopia and troublesome flatulency are not infrequent. The temperature is subnormal, and the arterial tension is not increased. The condition of the patient is tolerable, so long as drink is supplied in plenty, were it not for the disturbed sleep caused by the incessant thirst and the desire to pass water; but any attempt to restrict the quantity of fluid gives rise to intense discomfort. Ultimately this constant strain wears out the patient, and may lead to exhaustion, coma and death, if intercurrent disease do not, in the meantime, supervene.

Of the phenomena of diabetes insipidus the urine alone requires special notice. It is inordinate in its quantity, and of a specific gravity little above that of water. It may remain persistently at 1004 (60° F.); but it may rise to as much as 1008 or 1010. It is transparent, almost like water, of a faint greenish-yellow tint, with little taste or smell, and of faintly acid or neutral reaction. The quantity varies with the amount of water consumed. On the whole, the quantity passed is greater than in diabetes mellitus, and may sometimes be measured by gallons. Urea, though relatively deficient in any specimen of urine examined, has sometimes been found absolutely in excess, sometimes diminished. On the other hand, uric acid seems diminished, but this may depend on the difficulty of estimating it in urine so greatly diluted. Sulphates, chlorides and phosphates, especially the earthy salts of the latter, are usually increased, while the only abnormal constituent, if such it can be called, said to have been occasionally detected is inosite; but the reactions of this substance, which has the same percentage composition as a sugar, but which

belongs to the benzol-series, are very unsatisfactory when occurring in urine. Acetone, diacetic and oxybutyric acids, or other products of abnormal proteid-destruction, are never present.

PATHOLOGY.—As in the case of saccharine diabetes, our insight into the morbid processes concerned in the production of polyuria has been greatly aided by direct experiment. Bernard found that by pricking the floor of the fourth ventricle above the level of the 'sugar puncture' he could produce copious diuresis; and in certain animals injuries to the central lobe of the cerebellum (the vermiform process of human anatomy) are followed by a like result (see **MEDULLA OBLONGATA**, Lesions of). From this part of the nervous system the nervous influence seems propagated to the kidneys, both by the splanchnics and spinal cord, but the exact course of the fibres has not yet been clearly demonstrated. Whether the nerves are merely vaso-motor fibres, section or paralysis of which would produce turgescence of the vessels of the kidneys, or trophic fibres, irritation of which would increase the activity of these organs, is not yet determined; but in all probability paralysis of the vaso-motor fibres is the main factor in the production of the polyuria.

In the definition of diabetes insipidus given above disease of the kidneys was expressly excluded; and, as far as the malady itself is concerned, *post-mortem* examinations reveal nothing abnormal, except increased vascularity of these organs, doubtless due to defective innervation of the renal vaso-motor mechanism. As a consequence of the disease, however, persisting over many years, and giving rise to frequent and severe distension of the bladder, when circumstances may prevent its being emptied with sufficient frequency, thickening of the walls of the bladder, dilatation of the ureters, and sacculation of the kidney have been described; but the accuracy of such observations as the results of simple polyuria is questionable. Undoubtedly the most important lesions which bear on the disease are those which have been found in the brain (p. 192), especially in the neighbourhood of the fourth ventricle. These, besides the injuries already alluded to, comprehend tubercular and other forms of inflammation, tumours of various kinds—gliomatous and syphilitic—together with endarteritis and the results thereof (p. 101).

DIAGNOSIS.—The diagnosis of diabetes insipidus rests on the presence of thirst, and persistent excess of urine, coupled with the absence of grape-sugar and albumen. The condition must be carefully distinguished from mere temporary excess of watery urine. Such an excess may occur where a large quantity of fluid of a diuretic kind has been swallowed, especially when there is little or no cutaneous transpiration. Again, sudden diuresis may occur about the period of early convalescence from fever; after the absorption of serous effusions; after an attack of megrim; or yet again when a hydronephrosis suddenly empties itself. All these are merely temporary and evanescent states. The total absence of grape-sugar distinguishes diabetes insipidus from diabetes mellitus, though the former may pass into the latter. In certain forms of Bright's disease, especially those characterised by contracted kidney, the urine may be excessive and of low specific gravity; but in all of these the presence of albuminuria, urinary casts, hypertrophied left ventricle and increased arterial tension will

allow little room for doubt. Finally, polyuria is not to be confounded with such abnormal discharges of urine as may occur from time to time in hysterical persons. Here the nervous symptoms give a special feature to the malady; nevertheless diabetes insipidus has strongly marked nervous affinities.

PROGNOSIS.—This cannot be called favourable, for, while few actually perish from the uncomplicated disease, still fewer are cured of it, though a good many get well. As a rule it runs a chronic course. The onset of anorexia is suggestive of fatal result.

TREATMENT.—Innumerable energetic therapeutic measures have been resorted to with but slight success. If the disease can be assigned to any definite cause, we must deal with it, rather than with the excessive urination; if not, it must be our endeavour to counterbalance the draining of the tissues and the corresponding waste by a plentiful supply of fluid and a generous nourishing mixed and easily assimilable diet given at definite meal-times. No special injunctions are called for in planning the daily diet, but toxæmia from overloaded bowels must be guarded against. The distressing excessive thirst may be somewhat assuaged by sipping acid drinks, or sucking ice dipped in lemon-juice, but sudden withdrawals of liquid should be avoided. To relieve the kidneys from the unusual stress thrown upon them, diaphoretics have been recommended (*see* p. 385). Great care should be taken that the patient be warmly clothed so as to guard against any risk from cold. Of medicinal remedies, that which was once most lauded is valerian, especially by Trousseau, who gave it in enormous doses. Probably it, like other antispasmodic remedies, would be found of most service in cases allied to hysteria or similar neuroses. The whole range of antispasmodic remedies may in some cases be tried without effect. Opium and its alkaloids, though valuable in diabetes mellitus, are worse than useless in diabetes insipidus, except when specially exhibited for insomnia or nervous symptoms. They diminish the thirst and the urine, but they greatly increase the patient's discomfort. Valerianate of zinc, ergotin, iodide of potassium, belladonna, phenazone, trinitrin, and nitrite of sodium, have been tried with varying success. Tonics, especially strychnine and iron, in combination with arsenic, do good by improving the general health. Tea, coffee, and alcoholic stimulants should be interdicted and salt sparsely partaken of. After every drug has been tried in vain, attention to the constitutional state and resort to bracing inland or seaside air either as a primary remedial agent or as an adjuvant may be followed by almost complete disappearance of the polyuria (p. 310). Suitable doses of bromide of potassium at bedtime may be given to children until restlessness with disturbed sleep has passed away. Finally, the constant electric current (1 to 4 milliampères) has been advocated. This electrical treatment is best carried out by applying the positive pole to the nape of the neck, and the negative pole, properly insulated, to the posterior naso-pharyngeal wall. *See* ELECTRICITY IN MEDICINE.

Symptomatic polyuria calls for the treatment of its primary cause. *See* HYSTERIA.

In the treatment of cases of azoturia, strict attention should be paid to the processes of nutrition and assimilation, and all departures from the standard of health remedied. The chylo-poietic tract should be unloaded by occasional doses of blue

pill or calomel, or by a course of Pil. Hydragr. Subchlor. Co. followed each morning by a mild dose of some aperient water. The patient should have a daily morning bath or a tepid or warm salt-water bath (*see* BATHS, p. 138), followed by systematic friction of the whole body. The general vigour of body should be still further promoted by graduated Swedish exercises. The diet should be simple, and the habits of the patient strictly regulated. An important element in treatment is rest. Complete freedom from mental strain and removal from occupations calculated to foster mental disquietude should be secured. Change of scene and cheerful mental surroundings will therefore be useful adjuncts, and to this end a visit to such bracing air as that of Newquay, Cromer, Margate or Braemar will markedly aid the other therapeutic measures.

JOHN HAROLD.

POMPHOLYX (πομφόλις, a bulla or bladder).—A synonym for pemphigus.

PONS VAROLII, Lesions of.—**SYNON.** : Fr. *Maladies de la Mésocéphale*; Ger. *Krankheiten der Brücke*.

INTRODUCTION.—The pons is liable to a variety of affections, either through morbid processes having their primary seat therein, or by secondary implication from disease originating elsewhere, such as tumours of the cerebellum or base of the skull, aneurysm of the basilar artery, or as an element in chronic degenerative diseases of the central nervous system.

The position of the pons, its close relation to the vital centres of the medulla oblongata, the connection of the sensory and motor paths with the cerebrum and spinal cord on the one hand, and the cerebellum on the other, and the transit through it of many of the cranial nerves, render the symptomatology of pontine affections highly complex.

SUMMARY OF PATHOLOGICAL CONDITIONS.—*Hæmorrhage* into the substance of the pons is by no means uncommon, and may vary from a minute focus up to complete disorganisation and rupture into the fourth ventricle. *Embolism* is not common; but *thrombosis* from *syphilitic* or *atheromatous degeneration* of the basilar artery is frequent, and is the origin of necrotic *softening* of an acute or chronic character.

Hæmorrhage.—Hæmorrhage into the substance of the pons, if of small extent, is not necessarily fatal; but if it be of large amount, death occurs suddenly, or within a very few hours. Sometimes there is a sudden onset of coma, with complete relaxation of the whole muscular system. The pupils are, as a rule, minutely contracted, and the condition resembles profound narcotic poisoning. The temperature may rise to as much as 109° F. or more. Deglutition is difficult or impossible; and death ensues from cardiac and respiratory paralysis, irregularity in the rhythm preceding the fatal issue. At other times, and of great significance from a diagnostic point of view, muscular spasms occur, either general or affecting one side more than the other, with distortion of the face, either from paralysis of one side, or this combined with active spasm of the other.

Tumours may grow in the substance of the pons, but more commonly it is implicated by new-growth originating at the base or in the cerebellum.

Softening.—Acute embolic or thrombotic softening of the pons, with or without loss of consciousness, may lead to death rapidly, with paralytic symptoms; but days may elapse, or even months, after the first onset, with characteristic symptoms indicative of the position of the lesion, and death ensue either from gradual implication of the vital centres, or quite suddenly.

LOCALISING PHENOMENA.—The symptoms most characteristic of lesions of the pons are a combination of paralysis of certain cranial nerves on the one side, and of the limbs on the other. The most common combination is paralysis of the face on the one side, and of the limbs on the opposite, the face being paralysed on the side of the lesion. The facial paralysis in this case resembles peripheral facial paralysis, both in the implication of the orbicularis oculi and degenerative change in the muscles. The limbs may be paralysed as to motion only, or there may be a combination both of sensory and motor paralysis. Sometimes the motor paralysis affects one limb more than the other, and there may be a similar distribution of anæsthesia.

The alternate paralysis of the face on one side, and of the limbs on the opposite, occurs more particularly with lesions situated in the 'crustal' portion of the pons and infra-nuclear in position. If the lesion be higher up, near the *crus cerebri*, the face and limbs may both be paralysed on the side opposite the lesion, and present the characters of ordinary hemiplegia.

Various odd associations of palsies are highly characteristic of pontine disease. Such, for example, are paresis of three limbs, and a bilateral paralysis in which the limbs are affected more on one side than on the other, with or without paralysis of the face on one side.

Next in frequency to affections of the facial nerve, with or without affections of the limbs of the variable character above mentioned, comes paralysis of the abducens or sixth cranial nerve. This gives rise to an internal strabismus, usually of the eye on the same side as the lesion. There may be, therefore, paralysis of the face and abducens on the side of lesion, and of the extremities on the opposite side; but cases have been recorded of paralysis of the abducens on one side, and of the face and limbs on the opposite; and also of paralysis of the face, abducens nerve, and limbs on the same side as the lesion. In some cases there is paralysis of conjugate movement of the eyes towards the side of lesion. This occurs when the nucleus of the sixth, which innervates the external rectus of the same side and the internal of the other, is affected.

The fifth cranial nerve is also not infrequently implicated. The sensory portion seems to suffer more than the motor. But cases have been recorded in which the motor portion of the fifth has been specially affected, leading to paralysis and degeneration of the muscles of mastication.

The paralysis of the sensory division shows itself in more or less marked anæsthesia of the face, which may be general, or limited to the area of distribution of some of the branches. The tongue is not infrequently implicated on the same side, and tactile and gustatory sensibility impaired or abolished over the half of the tongue. The affection of the fifth may occur on the same side as the lesion, with or without affection of the limbs,

but it would appear also that anæsthesia of the face may occur, with implication of the extremities on the side opposite the lesion.

Lesions of a bilateral or multiple character, chiefly in the form of small hæmorrhages or softening, give rise to symptoms of a pseudo-bulbar type; such are weakness of the limbs on both sides, and paresis of articulation and deglutition from interference with the movements of the lips, tongue, and soft palate.

There is thus an extraordinary complexity and variability in the symptoms which may be met with in connection with pontine lesions. Those which have been mentioned are the most common and most significant, especially if they occur in combination. Singly they have less value, and some of them, particularly defects in articulation, are not specially characteristic. But a combination of paralysis of the limbs on one side, either motor alone, or of motility and sensibility, and of the face on the other, is significant of pontine lesion. The addition of paralysis of the abducens adds to the certainty.

The localising phenomena of pontine lesions may be summarised in the following way. The characteristic 'alternate' paralyses are:—

1. Paralysis of the conjugate movement of the eyeballs to one side with palsy of the opposite limbs.
2. Peripheral facial paralysis on one side and paralysis of the limbs on the other.
3. Paralysis of the conjugate movement and of the face on one side and of the limbs on the other.
4. Paralysis of the fifth nerve, motor, sensory or both, on one side, and of the face (cerebral type) and the limbs on the opposite side.
5. Paralysis of one or more cranial nerves on one side, with palsy of motion or sensation or both on the other side.

Many other symptoms might be mentioned which have been noted in connection with lesions of the pons, especially tumours, which ought perhaps to be ascribed to interference with the functions of neighbouring structures. Along with one or more of the previously mentioned symptoms, impairment of deglutition has been observed, due without doubt to pressure on the medulla oblongata. To the same cause should also be ascribed the irregularity and ultimate paralysis of the cardiac and respiratory movements, in connection either with tumours or with hæmorrhagic effusions into the pons itself.

When a tumour presses forward in the direction of the *crura cerebri*, the third cranial nerves may be implicated. Ptosis has been observed in such cases; and external strabismus, from paralysis of the internal rectus, has also occurred, but comparatively rarely.

Vertigo and disorders of equilibrium have been observed, but these may be attributed to an implication of the cerebellum or of its peduncles. Ataxic symptoms have, however, been described as occurring in pontine lesions, without affection either of the cerebellum or of its peduncles. If ataxy is a prominent symptom, disturbances of cutaneous sensibility and of the muscular sense usually co-exist. But the cases which have been recorded are not yet sufficient to establish any very definite propositions in regard to the exact causation or special characteristics of the ataxic disorders in question. In connection with tumours pressing on the pons, hearing

may also be impaired or abolished in one or both ears. Impairment of smell has been observed on one side, when there has been anaesthesia of the face. This is probably due to the impairment of common sensibility in the nostril, intensified in some cases by the defective power of sniffing if the facial nerve is also paralysed.

Albuminuria and glycosuria have occasionally been found in connection with diseases of the pons. It is very doubtful if any causal relationship has been at all satisfactorily established. Very often, when albumen has been found, there is good reason to believe that it has been pre-existing, for lesions of the pons frequently occur in connection with chronic renal disease. Sugar has been found sometimes, and in other cases not. The same has been found in connection with lesions of other nerve-centres.

Diseases which encroach on the intracranial space produce the general symptoms of intracranial tumour, in addition to the special symptoms indicative of their invasion of the pons.

D. FERRIER.

W. A. TURNER.

PORENCEPHALY (πόρος, hole; and ἐγκέφαλος, brain).—A congenital condition in which a number of clefts or depressions extend from the surface of the cerebral cortex into the interior.

POROKERATOSIS.—A thickening and hardening of the epidermis around the orifices of the hair-follicles and sweat-ducts.

PORRETTA (La), in Italy, between Bologna and Pistoja.—Thermal, sulphurous, muriated saline waters. See MINERAL WATERS.

PORTAL OBSTRUCTION.—Strictly speaking, portal obstruction implies that there is some direct impediment to the flow of blood in the portal circulation itself, either affecting the trunk of the vein before it enters the liver, or its branches distributed throughout the substance of this organ. It must be remembered, however, that any condition interfering with the circulation beyond the portal system, whether in the hepatic veins, the upper end of the inferior vena cava, right side of the heart, or lungs, will retard more or less the flow of blood through this system; and also that either of the tributary branches of the portal vein may be affected alone. The portal trunk may be obstructed by direct pressure upon it, as by enlarged glands, a growth projecting from the liver, thickening from perihepatitis, or a neighbouring tumour, or aneurysm; by changes in its walls, leading to constriction or complete closure; or by blocking-up of its channel, as by a thrombus (see PORTAL THROMBOSIS). Cirrhosis of the liver is the most important disease which obstructs the portal circulation within the liver.

EFFECTS.—The effects of portal obstruction will depend on its seat, its degree, and the rapidity with which it is set up. They are merely those which necessarily follow mechanical venous congestion, namely, distension of the small vessels, which may end in changes in their walls and varicosity; escape of serum; a congested or catarrhal condition of mucous surfaces; hæmorrhages; and, in course of time, permanent changes in organs and structures which are thus affected. Their localisa-

tion in this case will correspond with the structures from which the portal vein receives its tributary branches, or with which the latter communicate. Hence any of the following conditions may result in various degrees from portal obstruction: (1) Congestion and catarrh of the mucous membrane lining the lower end of the œsophagus, stomach, and intestines, with consequent disorder of the secretions; dilatation, varicosity, ulceration and rupture of the small vessels; hæmorrhage into other parts of the alimentary canal, especially at the lower end of the œsophagus. (2) Ascites, one of the most frequent and evident phenomena (see ASCITES). (3) Enlargement of the spleen, either from mere accumulation of blood, or in chronic cases with permanent increase and alteration in the splenic structure. (4) Congestion, followed by fibroid changes, in the pancreas. (5) Hæmorrhoids, it is generally believed. (6) After a while, enlargement of the superficial veins of the abdominal wall, owing to their communications with the portal vein; as well as of the veins within the abdomen, which are tributary to it. In rare instances peritoneal hæmorrhage has occurred from the rupture of distended veins. (7) Congestion of the female generative organs in some cases.

Several of the conditions mentioned are obvious on clinical examination during life; others are only evident on *post-mortem* examination, although they assist in originating symptoms, especially in connection with the alimentary canal, such as those of dyspepsia, flatulence, and disordered bowels, diarrhoea being not uncommon. Hæmorrhage into the alimentary tract is usually revealed by the occurrence of hæmatemesis or melæna, but it may prove fatal without any discharge of blood externally. It must necessarily happen that, if the portal circulation is not properly carried on, the functions of the liver are proportionately impaired.

The signs of portal obstruction may set in with great acuteness, or more or less gradually. Those indicative of acute obstruction are the rapid development of abundant ascites, returning speedily after paracentesis; acute enlargement of the spleen; hæmorrhage into the alimentary canal; and speedy dilatation of the superficial abdominal veins. It must be remarked that the most striking phenomena may disappear in chronic cases, after a time, without the removal of the obstruction, probably owing to the establishment of efficient anastomoses, by which the blood is returned to the heart without passing through the liver. These comprise (1) the gastric and œsophageal veins, (2) the mesenteric and inferior hæmorrhoidal, (3) the coronary and phrenic, (4) the veins of Retzius, connecting the intestine and mesentery with the vena cava, (5) the vessels in the suspensory ligament and in any extensive adhesions that may have been formed. See HÆMORRHAGE, p. 620.

DIAGNOSIS.—There ought to be no difficulty in recognising the signs of portal obstruction in marked cases; and it might even be suspected before these signs are well-developed under certain conditions. The cause of the obstruction can only be made out by a consideration of each case in all its features.

TREATMENT.—Rarely can anything be done directly to remove portal obstruction. The portal circulation may often be relieved by acting freely upon the bowels, especially by means of saline and hydragogue purgatives. The special treatment of

the causal conditions upon which portal obstruction depends is described in the articles on those subjects (*see p. 107*). FREDERICK T. ROBERTS.

PORTAL THROMBOSIS.—**SYNON.** : Portal Phlebitis; Pylephlebitis; Fr. *Pyléphlébite*; Ger. *Pylephlebitis*.

Portal Thrombosis may be divided into two kinds: (A) Non-Infective; and (B) Infective.

(A) **Non-Infective Portal Thrombosis.**

CAUSES.—1. *Weakened force of Circulation.*—Thrombosis from this cause—the result of feeble action of the heart, or of marasmus—is a rare occurrence in the portal vein as compared with other veins.

2. *Diseases of the Liver*, causing destruction or constriction of the capillaries of the liver or branches of the portal vein, are the commonest causes. Of this group of causes the most common are:—

(a) *Cirrhosis of the Liver*, the thrombus arising in the smaller branches and then extending to the trunk and main branches; usually firm and hard, partly organised, and adherent to the vessel-wall; the vein itself dilated, and its wall thickened.

(b) *Cancer of the Liver.*—The thrombosis in this case is usually limited to individual branches, which are compressed by the growth. In some cases the thrombus is made up in great part of the new-growth itself. This is specially common with primary cancer of the liver.

(c) *Compression of the Vein* outside the liver by adhesions, the result of chronic peritonitis; or still more frequently by morbid growths of stomach, pancreas, omentum, or retroperitoneal glands. In very rare cases thrombosis of numerous peripheral branches of the portal vein, as the mesenteric veins, has been found giving rise to symptoms very similar to those caused by thrombosis of the trunk of the vein.

SYMPTOMS.—The symptoms of portal thrombosis are those of intense portal obstruction. There is ascites, rapidly developing, and, according to Frerichs, returning rapidly after removal by tapping. The veins of the walls of the belly become rapidly dilated. There may be hæmatemesis or a bloody diarrhoea. The spleen is greatly enlarged. Jaundice may or may not be present.

DIAGNOSIS.—The diagnosis of portal thrombosis is a matter of great difficulty, the symptoms being very like those of cirrhosis, of which, indeed, it is often a mere complication. In general, it is only when the thrombosis is very acute and, affecting the trunk or most of the branches of the vein, and causing very rapid ascites, splenic tumour, dilatation of the superficial abdominal veins, &c., that a diagnosis can be made.

PROGNOSIS AND TREATMENT.—The prognosis is always bad. Instances of recovery are extremely rare; in these cases the vein has been found converted into a fibrous thread, and a collateral circulation established. The treatment must be the same as for cirrhosis in most cases. In the very acute cases, leeches over the liver, cupping, and the administration of saline purgatives should be tried.

(B) **Infective Portal Thrombosis.**—*Pylephlebitis Purulenta.*—Suppurative portal thrombosis is commonly met with in connection with some infective process, in the parts from which the branches of the portal vein arise. Thus infective diseases of the intestines, especially of the vermi-

form appendix (appendicitis) are the most frequent causes. It occurs after dysentery, and more rarely after enteric fever. It is occasionally caused by ulcer and cancer of the stomach, and follows suppurative splenitis. In the newly born, suppuration sometimes extends from the umbilical vein to the liver. The vein is found greatly dilated, and filled with a dirty grey or reddish thrombus, containing organisms. The liver itself shows, on section, the branches of the portal vein filled with a diffuent thrombus and numerous pyæmic abscesses scattered throughout its substance.

SYMPTOMS.—The symptoms closely resemble those of abscess of the liver or of pyæmia. Traube thinks the diagnosis may be made if the liver and spleen be much enlarged, and if there be returning attacks of rigors with raised temperature, while between the attacks the temperature is natural or only slightly raised. Remittent pyrexia, with sweating, vomiting, diarrhoea, and rigors, are the most constant symptoms. The liver, however, is only moderately enlarged in some cases. Pain and tenderness in the right hypochondrium, with an icteric tint, in a case presenting pyæmic symptoms, should suggest this condition. There must be also evidence of some suppuration, which may involve the branches of the portal vein; and pyæmia and endocarditis must be excluded. Often, however, all these signs fail, and suppuration of most of the branches of the portal vein has been found after death when no hepatic symptoms have been present during life. Possibly the occurrence of hepatic symptoms depends upon the acuteness of the process.

PROGNOSIS AND TREATMENT.—The prognosis is always bad; the treatment must be the same as for abscess of the liver or pyæmia.

In view of the frequency with which it is caused by disease of the appendix, any suspicion of pylephlebitis should lead to the careful consideration of the propriety of surgical exploration of the appendix.

WILLIAM HUNTER.

POST-MORTEM EXAMINATION. — *See NECROPSY.*

POST-MORTEM WOUNDS.—**SYNON.** : Dissection-wounds; Fr. *Blessures Anatomiques*; Ger. *Sectionswunden*.—*See* SEPTICÆMIA; WOUNDS; ABSCESS; WHITLOW; CELLULITIS; NAILS, Diseases of; LYMPHATIC VESSELS AND GLANDS.

POST-PHARYNGEAL ABSCESS. — *See* RETRO-PHARYNGEAL ABSCESS.

POSTURE.—In this article it is intended to point out the main practical relations of posture to the ætiology, diagnosis, and treatment of various diseases.

1. **Ætiology of Posture.**—As an immediate cause of disease, posture is chiefly important in connection with occupation. The evil effects of prolonged *standing* are evidenced by the development of varicose veins in the legs, and also by the occurrence of general fatigue and debility, displacements of the uterus, and other conditions, especially in young women, such as those employed in drapers' shops. Those callings which entail constant or frequent *bending forward* of the body are often very injurious, and this may be aggravated by carrying burdens on the back and shoulders. Not uncommonly persons injure themselves by

habitually bending forward when sitting, quite apart from occupation. Another illustration of the influence of posture in causing disease is where individuals have to work in *constrained positions*, such as colliers and miners. The conditions thus induced are chiefly deformities of the chest, and certain diseases of the lungs, heart, and vessels. A peculiar posture in performing certain acts, such as writing, may have some influence in originating affections of the type of writer's cramp. The *recumbent* posture in low febrile and other conditions aids in the causation of hypostatic congestion and its consequences; a similar position promotes the accumulation of morbid products in the smaller bronchi or air-vesicles, in cases of severe acute bronchitis, which may cause further mischief; and if an attack of pleurisy should supervene when a patient is obliged to lie on his back, this will materially modify the way in which the fluid accumulates, for it tends then to collect posteriorly, and may cover the whole area of the chest in this aspect, while there is no sign of any fluid in front.

2. Posture in Diagnosis.—In many cases the posture suggests great debility, helplessness, or prostration, thus affording important indications as to the general condition of a patient. Patients instinctively assume the position which relieves pain, and which enables them to use sound organs to the greatest advantage. An inability to lie down (*orthopnea*) constitutes a prominent feature in certain forms of cardiac and pulmonary disease, in consequence of interference with the respiratory and cardiac functions, so that the patient is obliged to sit or to be propped up in bed, or sometimes even to sit up in a chair, to assume the erect posture, or to bend forward. Again, when anything is pressing upon the main air-tube—such as an aneurysm—causing obstructive dyspnoea, the patient may instinctively lean forward, so as to take off the pressure as much as possible. In cases of unilateral lung-disease or pleurisy, the patient is often unable to lie on one or other side, especially the affected one; while in affections of the heart it is frequently impossible for him to rest on the left side. As regards abdominal diseases, acute peritonitis is often characterised by a very striking posture, the patient lying on his back, with the knees well drawn up and bent, in order to relax the abdominal muscles. In appendicitis the right thigh alone may be similarly flexed. In spasmodic painful attacks connected with the abdomen, it is very common to see the patient bending forwards in a doubled-up position, and pressing upon the abdomen. In nervous diseases posture may be of conspicuous value in diagnosis. Thus, it may reveal paralysis of different parts; in cerebral meningitis the patient often lies in a curled-up position, all the limbs being bent towards the body; in spinal meningitis the head may be involuntarily drawn backwards, in order to try to relax the muscles behind; in tetanus the body is during the spasm fixed in different positions, according to the muscles affected; in cataleptic conditions any posture that is assumed is retained for a considerable or an unlimited time; while in wry-neck the head is turned to one side. Lastly, the position voluntarily assumed by a limb may give important information as to local diseases or injuries likely to influence it in this respect, such as those of the joints. The whole body may be distorted, as well as

the limbs, in connection with diseases of the articulations.

Change of posture is a useful aid to physical examination. It may give valuable help in determining the presence of fluid in cavities, such as the pleura or peritoneum; in distinguishing an internal aneurysm from conditions simulating this lesion; in detecting certain solid formations in the abdominal cavity; and for other purposes. Details on these points are given in appropriate articles in this Dictionary. It is also of importance to study the position of the patient in examining the chest; and to remember that posture materially influences the position of the heart.

3. Posture in Treatment.—Many of the preceding remarks will afford suggestions as to the value of paying attention to posture as a therapeutic measure, and it will at once be evident that if a wrong posture is the cause of any morbid condition, the first principle in treatment should be to rectify it. Besides, it will not uncommonly be found advantageous to watch patients, and to allow them to adopt, or assist them in adopting, such a position as their own sensations dictate to be the most suitable for their condition. In order to illustrate further, however, the benefits to be derived from posture, it may be well to point out some of the diseases in which its value is most strikingly exhibited.

(a) Posture is of great importance when general rest of the body is required, or when there is exhaustion or prostration of the whole system. The recumbent posture is clearly indicated under these circumstances, for it is the most restful of all, and involves little or no expenditure of muscular force. Hence, in acute febrile diseases of all kinds, one of the first indications in treatment is to keep the patient absolutely in bed. This is also desirable where there is excessive fatigue or prostration from any cause. The great importance of rest in bed during and after an attack of influenza may be specially noted.

(b) In the management of affections connected with the respiratory organs, attention to posture is frequently of service. Here its influence as regards rest again comes in, for it may be of much consequence to make as little call as possible upon the respiratory functions. Moreover, symptoms associated with the breathing apparatus are in many cases strikingly influenced by posture, such as pain, dyspnoea, or cough (*see* RESUSCITATION; and STERTOR); and the act of coughing may be materially assisted, and made more effectual as regards expectoration, by the patient assuming a sitting or erect position. The importance of the prone posture, or of bending forwards, must be remembered when there is anything pressing on the main air-tube.

(c) Posture often requires particular consideration in relation to disorders of the cardiac action, or to actual disease of the heart. Thus, in the syncope state the patient should be placed horizontally, or even with the head at a lower level than the body, so that the blood may more readily reach the brain, and in this way life may be sustained. Bending the head downwards between the knees may prevent threatened syncope. In this state, or when the heart is acting with extreme feebleness from any cause, raising the patient into a sitting posture has been known to cause a fatal result, and should be carefully avoided. On the other hand, there are

conditions of the heart in which the patient cannot possibly lie down, and especially where there is much dilatation; under these circumstances it may be of the greatest service to have him constantly sitting up in a properly constructed chair, and the beneficial effects thus produced are sometimes almost marvellous.

(d) In the treatment of aneurysms, whether internal or external, posture is frequently made use of with advantage. In the cure of this lesion in the chest or abdomen, rest is often an important agent, and on this account patients are confined to the recumbent posture for weeks or months, so as to keep the heart as quiet as possible, and also to limit the demand of the system for food, which is only given in a restricted quantity. Aneurysm in the chest is one of the causes which may originate pressure on the air-tube, and on this account attention to posture may be required in connection with it. In the case of aneurysm in the limbs, posture is sometimes made use of to cure them, by causing pressure, as flexion of the knee for the cure of popliteal aneurysm.

(e) The influence of posture with respect to gravitation may often be recognised with advantage in the treatment of certain conditions. This is well exemplified by its effects on dropsical accumulations in the legs and scrotum. Even abundant anasarca may frequently be got rid of completely in a short time by keeping the legs in a horizontal position; and oedema of the scrotum likewise may soon disappear when this part is propped up. The same principle is of essential importance in checking hæmorrhage from a ruptured varicose vein in the leg; and may also be made use of in the treatment of varicose veins. The influence of posture upon dropsy may give useful information as to its cause, and as to the exact conditions upon which it depends.

(f) As miscellaneous illustrations of the employment of posture in treatment may be mentioned the value of the recumbent position in sea-sickness, in attacks of giddiness, megrim, and neuralgic affections about the head; raising the head in comatose conditions; the prone posture in the treatment of certain forms of spinal disease; prolonged decubency or peculiar positions to restore a displaced uterus; and various positions in which limbs are placed on account of local diseases, to relieve pain, to prevent muscular tension, to promote the escape of pus, or for other purposes.

(g) Lastly, it must be remembered that it is not uncommonly requisite to change the position of a patient more or less frequently, if he should be confined to his bed. For instance, this is necessary in low febrile diseases, in order to prevent the occurrence of hypostasis at the bases of the lungs, or the formation of bed-sores on parts subjected to pressure; as well as in many cases of spinal or cerebral disease, and in very emaciated patients. Change of posture is further useful in assisting the escape or expulsion of morbid secretions from the air-passages, when they tend to accumulate there.

FREDERICK T. ROBERTS.

POTTER'S ASTHMA.—See PNEUMOCONIOSES.

POTTERY.—See OCCUPATION-DISEASES.

POUGUES, in Loire, France. — Alkaline chalybeate waters. See MINERAL WATERS.

POULTICE (πόλτος, porridge; *puls*, thick soup).—SYNON. : Cataplasm; Fr. *Cataplasme*; Ger. *Breiumschlag*.—Poultices are soft moist applications, usually applied hot, but occasionally cold. They may be used merely as a means of applying heat and moisture; or they may contain some drug intended to exert a specific effect. They are now largely replaced by fomentations. There are only two poultices in common use, (1) the *simple* poultice, composed of crushed linseed, and (2) the *counter-irritant* poultice, composed generally of crushed linseed and mustard.

1. **Simple Poultice**.—The simple poultice, by its heat, causes a dilatation of the vessels of the part to which it is applied, and thus hastens the progress of inflammation, either towards resolution or suppuration. It softens the cuticle, and relaxes the skin by its moisture, and thus favours swelling, and lessens tension and pain. In internal affections, such as bronchitis, pleurisy, or pericarditis, large poultices are frequently applied to the skin over the inflamed part. They benefit the patient partly by their warmth, and partly by exerting an extremely mild counter-irritant effect (p. 349), consequent upon the redness and congestion of the skin which they produce. They are, however, somewhat troublesome; they soon become cold; and if the patient be restless, their weight causes them to shift, and fragments break off and drop into the bed, and there drying cause considerable discomfort. For application to boils and wounds they are altogether unsuitable. See FOMENTATIONS.

In internal inflammations a poultice may often be advantageously replaced by cotton-wool only, covered with oil-silk and secured by a bandage. If any counter-irritant action is required, a few drops of chloroform or turpentine may be sprinkled on wool or spongio-piline.

Linseed-meal poultices are best prepared with recently crushed linseed (*Linum Contusum*, B.P.), as the meal tends to become rapidly rancid. The following is a useful method of making a linseed-meal poultice: Heat the basin in which the poultice is to be made with boiling water; then empty it and put into it again as much boiling water as may be necessary to make the required poultice; sprinkle the crushed linseed into the water, stirring vigorously, till the proper consistence is attained. By adopting this plan the poultice will be free from lumps. The poultice should then be spread with a broad spatula, previously moistened with water, on a piece of rag or tow. It must be of a uniform thickness, and neither so thick as to be too heavy, nor so thin as to cool and dry too rapidly. A poultice should be changed every two or three hours by day, and every four at night, if the patient is sleeping.

2. **Counter-irritant Poultice**.—*Cataplasma sinapis*, the ordinary mustard poultice, is an invaluable counter-irritant. It is composed of mustard in powder, $2\frac{1}{2}$; crushed linseed, $2\frac{1}{2}$; boiling water, 10. The crushed linseed is to be mixed with the water, and the mustard added, constantly stirring. Its action should extend only to producing redness of the skin, for if kept on too long it will cause vesication, and has even been known to give rise to sloughing. The time a mustard poultice can be kept on varies from ten minutes to half an hour or more, according to the strength of the mustard. The sensations of the patient can usually be relied upon as a guide. An ordinary patient

is not likely to keep it on too long, as the smarting soon becomes unbearable. If the proportion of mustard be reduced to 1 in 4 or 5, the poultice will take an hour or more to produce the desired effect, and, in this form, is a much safer application in the case of young children. Patients who are much in the habit of applying mustard poultices to the same part—as, for instance, the front of the chest—acquire a singular power of resistance to the irritative action of the mustard. The mustard poultice is indicated whenever mild and rapid counter-irritation is desired. It is especially useful in bronchitis, and in muscular rheumatism, as lumbago or pleurodynia. Rigolot's mustard-leaves, and the *charta sinapis* of the *British Pharmacopæia*, are excellent substitutes for the mustard poultice. They are cleaner, more easily applied, and can be more accurately adapted to the spot required. They should always be used in preference when obtainable.

MARCUS BECK.

H. MONTAGUE MURRAY.

PRÆCORDIAL ANXIETY or OPPRESSION.

SYNON. : Fr. *Angoisse* ; Ger. *Præcordialangst*.

DEFINITION.—A sensation of constriction, attended with anxiety, referred to the præcordia ; for the most part persistent, but at times recurrent and depending on dilatation of the heart. See PAIN IN VISCERAL DISEASE, p. 1141 ; ANGINA PECTORIS, p. 66 ; and HEART, Dilatation of, p. 635.

PRÆCORDIAL PAIN.—SYNON. : Heartburn ;

Fr. *Cardialgie* ; Ger. *Magenschmerz*.

DEFINITION.—Pain referred to the region of the heart. Pain in the region of the heart may be due to local causes or may be reflected from the heart or some other viscus associated with the præcordial region of the chest. See PAIN IN VISCERAL DISEASE, p. 1141.

PRÆSYSTOLIC.—A term implying antecedence to the ventricular systole, and used in connection with a cardiac murmur or thrill occurring during this period of the cardiac revolution. See HEART, VALVES AND ORIFICES OF, Diseases of ; and PHYSICAL EXAMINATION.

PREBLAU, in Carinthia, Austria.—Acidulated alkaline waters. See MINERAL WATERS.

PREGNANCY, Disorders and Diseases of.

The most common, and therefore the most important, serious disorder of pregnancy is its premature termination by abortion or miscarriage. This is considered separately under the headings of ABORTION ; PLACENTA, Diseases of the ; and MOLE. In the present article, therefore, abortion will only be considered incidentally in its relation to certain diseased conditions which have an unfavourable influence on the pregnant state and tend to favour its miscarriage.

Under the present heading will be considered :

I. Diseases of pregnancy caused by neuroses, or blood-changes. II. Diseases caused by pressure. III. Diseases caused by misplacement of the pregnancy, or of the uterus containing the pregnancy (under this division *Extra-Uterine Pregnancy* will be considered). IV. Diseases caused by abnormal conditions of the uterus itself (or of the uterine appendages) other than displacement. V. Diseases caused by pelvic deformity or bony outgrowth. VI. Finally, as there are certain medical

and surgical diseases, as well as certain surgical operations, which have been found seriously to affect the normal progress of pregnancy or parturition, these and their influences on the pregnant state will be considered in some detail.

I. Diseases caused by Neuroses or Blood-changes: (1) Vomiting (Hyperemesis) ; (2) Salivation (Ptyalism) ; (3) Unusual appetite ; (4) Dental caries ; (5) Cutaneous disorders ; (6) Chorea gravidarum ; (7) Mental perversion.

1. Vomiting (*Hyperemesis gravidarum*).—The vomiting of early pregnancy is a very common symptom. It is usually (but by no means necessarily) confined to the earlier part of the day, and is generally started by getting out of bed, or by any very marked change of position. It may begin within a week after conception has taken place and persist throughout the whole of pregnancy, but it is during the second, third, and fourth months that the disorder is generally more acute.

In severe cases nearly everything is rejected from the stomach as soon as it is swallowed, and progressive emaciation and weakness may be extreme. When this is the case, it is well to remember that pregnancy may be complicated with graver disease, and that the vomiting may be due, not to one condition only, but to both conditions. In two cases of this kind within the writer's experience, both of which were treated unsuccessfully—one by emptying the uterus, and the other, first by replacement of a gravid retroversion, and afterwards by emptying the uterus—the fatal issue was found to be due to grave complication : in the first case, to malignant disease of an ovary, and in the second to contracting adhesions of the small intestine, due (probably) to old peritonitis in the neighbourhood of a gastric ulcer.

The vomiting of pregnancy is essentially a nervous disorder, but movement of the enlarged uterus, temporary heart-weakness or faintness, and some abnormality of the pregnancy (gravid retroversion, spasmodic closure of the cervix, abnormal placental attachment) may be looked upon as exciting causes, and their consideration as such lead the practitioner to suitable treatment.

TREATMENT.—In minor cases, change of residence, life in the open air, and the administration of a vegetable bitter with dilute mineral acid before food, may be all that is necessary (Acid. Nitro-Hydroch. Dil. m x-xv, Infusi Gentianæ ad zj : t.d.s.). When sudden movement causes nausea, suitable advice should be given regarding this. When the nausea is associated with faintness, a dessertspoonful of brandy may be given once or twice daily, the administration being stopped as soon as the symptom is relieved. When gravid retroversion is found, this, as a rule, should be replaced. When the cervix appears to be specially contracted and sensitive, gentle dilatation may immediately relieve the associated symptom.

In all severe cases, when no intercurrent or associated disease can be made out, the following plan of treatment is recommended. The patient is fed by nutrient enemata, all food by the mouth being temporarily withheld. Twice a day, if necessary, the patient is given an enema containing zj of the mixed bromides (zss Potassium Bromide, zss of Sodium Bromide) and 20 grs. of Chloral Hydrate. This may be administered in zj – zij of beef-tea, and should be entirely retained if possible.

As soon as the bromide and chloral hydrate

begin to affect the system the vomiting lessens, and nourishment may be cautiously given by the mouth. When this is retained by the stomach, the enemata are discontinued and the same drugs given in one-fourth doses, in the form of mixture (R. Chloral Hydrat. grs. iv-v, Potassii Bromidi, Sodii Bromidi aa grs. viiss, Aquam ad ʒj). In cases of less severity this mixture may be employed from the outset.

In all severe cases temporary confinement to bed is a necessity. In some cases this has been apparently continued with advantage throughout the whole of the pregnancy. In other cases dangerous vomiting has been promptly relieved by the induction of abortion. If the method recommended above be thoroughly adopted, these methods of treatment (to which there are grave objections) will rarely, or never, be necessary. *See VOMITING.*

2. *Salivation.*—Salivation is a rare disorder of pregnancy. It is generally associated with nausea and vomiting and sometimes appears to be forced into undue prominence by the refusal of the patient to swallow her saliva. Very similar treatment to that advised for vomiting is most successful. Full doses of the mixed bromides are given persistently, and the patient should be encouraged to dispense with the constant use of the spittoon or handkerchief. In cases where the gums are swollen and the breath offensive, the practitioner should prescribe a mouth-wash of chlorate of potassium (grs. x ad ʒj), and inquire into the possibility of any coincident mercurialism. Atropine, pilocarpine, and galvanisation of the cervical sympathetic have all been tried for the relief of this condition, but on the whole these methods appear to be inferior to the treatment by bromides.

3. *Unusual Appetite.*—The appetite of the pregnant woman may be subject to considerable alteration during the course of pregnancy; a woman who is accustomed to sugar and starchy foods finding that these are distasteful and that meat and highly seasoned dishes are better appreciated and digested, or the reverse of this may be experienced. Within reasonable limits such a change of appetite indicates the special temporary requirements of her organism and may be gratified; in particular, curries and seasoned dishes may often be taken without any consequent nausea, while comparatively tasteless foods are immediately followed by vomiting. Occasionally the appetite becomes absolutely depraved (*Pica*). In this case there is usually some mental disorder or perversion, and control is not only advisable but necessary. *See APPETITE, Disorders of.*

4. *Dental Caries.*—In pregnancy there is almost always a special tendency to dental caries, so much so that 'for every child a tooth' has become a popular proverb. Nevertheless, the drift of all modern thought and investigation is to lay stress on local causes and on these alone as originating dental caries. Decomposition of the starchy foods remaining in contact with the teeth and gums, and excessive growth of micro-organisms within the mouth, are the important factors. In pregnancy (apart from salivation and occasional gingivitis) there appears to be some special predisposition to the decomposition of retained matters, and the toilet which suffices under ordinary conditions may prove insufficient during the time of gestation.

The Eastern habit of washing out the mouth, not only after every meal, but after partaking of any

food, and the careful cleaning of the teeth at night, are the methods by which this tendency may be most successfully fought against.

When caries has already started it needs prompt attention by the dental surgeon, and in weak, strumous and poorly-nourished patients the local treatment may often be advantageously supplemented by the administration of good food, hypophosphites, lime-salts, and cod-liver oil. *See TEETH, Diseases of.*

5. *Cutaneous disorders.*—The chief disorders of the skin encouraged by, or associated with, pregnancy are pigmentation, 'pityriasis versicolor,' herpetiform eruptions (herpes gestationis), and (rarely) a pustular eruption, best described as impetigo. In addition to these, the development of subcutaneous fat demands some notice. This may lead to marked alteration in the shape and expression of the face. The alteration in appearance produced by this and by excessive pigmentation is only temporary; it disappears with the termination of the pregnancy and calls for no treatment.

Parasitic skin-affections need anti-parasitic remedies. Of these, a solution of perchloride of mercury is perhaps that most generally useful. Herpes gestationis may appear at the very beginning of pregnancy, but is more often met with in the later months or immediately preceding delivery. It is attended by much itching and burning, and is usually relieved by local applications of ichthylol (5 to 10 per cent.). *See HYDROA, p. 689.*

Impetigo gravidarum occurs as an eruption closely simulating that of Variola. In one patient who was under the care of the writer, and who suffered from this eruption during each pregnancy (but not at any other time), the resemblance was so striking that her neighbours reported the case to the sanitary authority as one of small-pox. The pustules were numerous, large, and mostly discrete. The chest, back, and face were the parts chiefly affected.

TREATMENT.—In this eruption, as in several other pustular affections (such as furunculosis), the administration of lime-salts (calcium hypophosphite and sulphide) appears to be of decided value. Locally dilute ointments of ammoniated mercury (grs. v ad ʒj) or ichthylol (5 to 10 per cent.) are those most efficacious and grateful. *See IMPETIGO.*

6. *Chorea gravidarum.*—Chorea is sometimes, although very rarely, a special disease of pregnancy in that it first occurs during pregnancy and recurs during subsequent pregnancies. In most cases it must rather be regarded as an intercurrent disease which is aggravated by the pregnant state. As predisposing causes rheumatism and rheumatic surroundings are of chief importance. The dangers of chorea during pregnancy are mental exhaustion, pain, want of rest, with consequent delirium and mania, cerebral hæmorrhage, intra-uterine hæmorrhage, and incomplete abortion.

TREATMENT.—The chief drugs of any value are arsenic and chloral hydrate. When incomplete abortion has occurred and there is any danger of sepsis, the uterus must be emptied; but if this be necessary, the operation should be done so as to cause the least shock to the patient. It is well to remember that the chorea does not necessarily cease with the termination of the pregnancy. Premature delivery unless clearly indicated should be avoided.

7. *Mental Perversion.*—*See INSANITY OF PREGNANCY, p. 758.*

II. Disorders due to Pressure.—(1) Frequency of micturition; (2) Constipation; (3) Hæmorrhoids; (4) Varix; (5) Œdema of the lower extremities; (6) Cramps of the lower extremities; (7) Pruritus vulvæ.

1. *Frequency of micturition* is often complained of during the first two months of pregnancy when the enlarged fundus is lying forward on the bladder, either preventing full distension or, by its pressure, exciting a desire for urination. This always improves as the bladder becomes accustomed to the pressure, and still more so when the uterus rises out of the pelvis into the abdomen and the pressure is removed.

2. *Constipation* is an occasional difficulty during pregnancy, the pressure and stretching caused by the enlarging uterus interfering with the proper action of the rectum. The best treatment is by an aloes-and-belladonna pill given every night: Ext. Aloes Barb. gr. jss; Ext. Belladonnæ gr. $\frac{1}{4}$. A small enema should be administered every other morning if the pill is insufficient. See CONSTIPATION.

3. *Hæmorrhoids* are always made worse by pregnancy, and sometimes during the later months the pain and distress occasioned by the rectal protrusion may be severe. In such a case pregnancy need be no bar to successful operation. In milder cases the daily full evacuation of the bowels, as advised under constipation, and the local application of some preparation of Hamamelis, will afford the most relief to the patient.

4. *Varix* of the superficial veins of the thigh, labium and 'mons' is a pressure-disorder of pregnancy which sometimes causes serious disease. The enlarged veins may form a considerable tumour, which on standing or walking becomes prominent and turgid and involves the patient in the very practical danger of venous rupture, either subcutaneous or open. The treatment of this condition demands a good deal of rest in the recumbent position with the foot of the couch or bedstead permanently raised. All pressure-disorders need this. It is a good rule during all the later months of pregnancy to make the patient lie down on a raised couch or bedstead for two hours in the early afternoon. This divides the day into two short half-days and does much to obviate the evil effects of both weight and pressure. The support of a bandage, which is so useful in varix below the knee, is generally impossible of application to the upper part of the thigh and to the vulva. When the varix is sufficiently localised the dilated veins may be excised. In a very few cases the condition may be so severe and extensive as to call for the induction of premature labour.

5. *Œdema of the lower extremities* is sometimes due to pressure only, but may be due to coincident kidney-disease (see p. 216). If, on examining the urine, no albumen is found, the pelvis and abdomen should be carefully examined to see if there is any accessory or abnormal cause of pressure—such as myoma or co-existing ovarian tumour. If none be found a good prognosis may be given, and the tendency to œdema treated by position (see 4. Varix, above), by aperients, and if there be anæmia, by blood-tonics.

6. *Cramps.*—During the later months of pregnancy cramps of the lower extremities frequently occur at night. These appear to be directly due to pressure on the larger nerve-

trunks in the pelvis, and are met with most commonly while the patient is lying on her back.

Change of position is generally sufficient to relieve this distressing symptom.

7. *Pruritus vulvæ* appears sometimes to be due to the pelvic congestion accompanying pregnancy. When complained of, the case should be investigated for any possible intercurrent cause such as gonorrhœa or glycosuria. A very hot saturated solution of boric acid, freshly made, will usually allay the itching. The parts should then be carefully dried with soft rag and anointed with dilute boric-acid ointment. At night this can be applied on borated gauze and kept *in situ* after the lotion has been used.

When there is marked purulent discharge affecting the urethra as well as the vagina; when this is sudden in its onset and accompanied by much pruritus and dysuria, the possibility of gonorrhœal infection should always be considered. In this case the vagina should be well treated with suppositories of silver nitrate ($\frac{1}{4}$ gr.), and a special disinfection of the genital passages be carried out at the time of the confinement. This will diminish the risk of gonorrhœal ophthalmia. See PRURITUS.

III. Diseases due to Misplacement of the Pregnancy or of the Uterus containing the Pregnancy.—(1) Placenta prævia; (2) Retroversion of the gravid uterus; (3) Cornual pregnancy and interstitial pregnancy; (4) Extra-uterine pregnancy (and its consequences).

1. *Placenta prævia.*—DEFINITION.—A pregnancy in which the placenta is attached over the internal os or lower outlet of the uterus, and therefore in front of the child.

VARIETIES: (1) Placenta prævia centralis or totalis; (2) Placenta prævia lateralis; (3) Placenta prævia marginalis.

FREQUENCY AND CAUSATION.—The frequency of Placenta prævia is about 1 in 1,500 births (Winckel). It is very much more common in multiparæ than in women who have not had a previous pregnancy, and the pregnancy in which it occurs is stated to be often preceded by chronic endometritis.

DANGERS.—Difficult labour and fatal hæmorrhage.

DIAGNOSIS.—This is difficult or impossible in the earlier months so long as the cervix is contracted. It may be suspected (a) when sudden copious bleeding occurs at what would have been a menstrual period, and especially if this is repeated after three or four weeks' interval; (b) when no other site can be discovered for the placenta both by palpation and auscultation; and (c) when there is unusual thickness, vascularity and pulsation in the neighbourhood of the cervix. Very often bleeding does not occur until the last month of pregnancy, sometimes not until labour commences—the bleeding in placenta prævia being due to separation of the placenta (1) from development of the lower uterine segment and (2) from dilatation of the cervix.

When the cervix is dilated the diagnosis becomes easy. Instead of the presenting amniotic sac containing the liquor amnii and fœtus, there is the thick fleshy mass of the placenta intervening. In placenta prævia lateralis and marginalis the edge of this is tangible, and beyond this it may be possible to feel the amniotic sac and fœtus. In placenta prævia totalis, the cervical canal is everywhere covered by placenta.

TREATMENT.—In early and suspected cases, absolute rest in bed and the administration of a Dover's powder will usually be sufficient to check hæmorrhage. Ergot should not be given. If the bleeding continues the vagina may be temporarily plugged with salicylic or sublimate wool, and the uterus may be afterwards emptied of its contents.

In cases at a near term when the cervix is sufficiently dilated to feel the placenta and examine its attachments:—

(a) if the placenta be marginal, the membranes may be ruptured and the head allowed to descend by the side of the placenta, natural labour proceeding (as with normal placental attachment).

(b) if the placenta be central, such separation is made as will conduct the finger (by the shortest route if possible) to the amniotic sac and foetus, and the foot of the child is brought down by 'combined version' with one finger. The examining finger pushes the shoulder aside while the breech is pressed downwards by the hand outside the abdomen. This brings the feet of the foetus in contact with the finger. The membranes are now ruptured, the finger passed around one foot, and by pressure of this and the cervix against the pubes the foot is tightly held until it is conducted through the cervix. The other foot may be afterwards brought down. The body of the child now forms a plug which stops further hæmorrhage, and delivery should be allowed to proceed slowly. The rapid emptying of the uterus is not advisable. The neck of the uterus in cases of placenta prævia is often easily torn, and the hæmorrhage from a deep laceration of the cervix may prove fatal when that from the separated placenta has been practically stopped.

2. *Gravid Retroversion.* — **DEFINITION.** — A uterus at one and the same time displaced backwards and pregnant.

HISTORY. — There is usually some previous history of displacement, and pregnancy must of necessity be a possible condition. In the greater number of cases where pregnancy and backward displacement are combined, the uterus rises as the pregnancy grows and the displacement spontaneously rights itself. In the minority this spontaneous replacement does not occur, and at the end of the second or beginning of the third month serious trouble arises. The pressure of the pregnancy on the neck of the bladder and urethra causes dysuria, and after a short time complete retention of urine. If unrelieved the ureters and kidneys suffer from backward pressure, and fatal sloughing has been known to occur from the increasing pressure within the pelvis.

DIAGNOSIS. — The patient has the ordinary symptoms of pregnancy, nausea and vomiting being sometimes excessive. She probably has a history of amenorrhœa corresponding to the period of gestation at which she has arrived, and the mammary signs may be confirmatory of her condition. Dysuria or retention of urine is probably the prominent symptom.

On examination, a big, elastic, globular mass is found filling the pouch of Douglas behind the cervix and pressing this against the pubes.

On bi-manual examination with the bladder empty, the fundus is found to be wanting anteriorly and the cervix may be felt (from above as well as from below) to pass directly into the posterior tumour. The tumour itself is elastic and resilient

like a gravid uterus of the second month. The difficulty of passing urine is quite of recent date, and with the exception of 'retention,' which may be a sudden phenomenon, there is no history of acute collapse or pain or hæmorrhage (unless abortion be in progress), but the discomfort occasioned by the gravid displacement is one of gradual and steady growth.

The differential diagnosis between this condition and tubal pregnancy with intra-peritoneal hæmatocèle is one of vital importance, and no attempt at replacement of the uterus should be made until the possibility of extra-uterine pregnancy has been rigidly excluded.

Where the pregnancy is outside the uterus and the tumour in Douglas's pouch has been caused by hæmorrhage there is usually a history of rather sudden collapse and pain; pressure-symptoms may be, but are not as a rule particularly prominent; and on careful bi-manual examination (if necessary, under anæsthesia) the whole of the uterus, from cervix to fundus, is found in front, or to one side of the tumour.

TREATMENT. — The bladder is completely emptied. The cervix is seized with volsella, or bullet-forceps, and the patient is placed in the knee-chest position. The cervix is drawn downwards and backwards, and the patient is directed to breathe deeply and regularly. One or two fingers placed in the posterior fornix at the same time assist in pushing the fundus upwards and forwards.

In this way reposition may usually be directly carried out, the womb escaping into the abdomen above the pelvic brim. When this takes place, the posterior tumour vanishes and the cervix becomes directed backwards. A single large ring-pessary inserted while the uterus is in good position will prevent any danger of relapse. The pessary can be removed about the fifth or sixth month.

In cases where attempts at immediate reposition are ineffectual, the patient should be kept in bed with the foot of the bedstead slightly raised, and the urine should be regularly withdrawn by an aseptic catheter. Under these conditions spontaneous reposition usually takes place. It may be assisted by the use of a vaginal 'colpeurynter' (a dilator of indiarubber distended with water).

In a few cases the pregnant uterus may be adherent in retroflexion. If the adhesions are light they will be separated by the growth of the pregnancy or so lengthened and undone as to admit of the manual reposition of the organ. If the adhesions are too dense for this, it is better practice to open the abdomen and separate them carefully from within the peritoneum than to use force or to make repeated ineffectual attempts at replacement from without.

3. *Cornual Pregnancy and Interstitial Pregnancy.* Pregnancy in a rudimentary horn and pregnancy in the substance of the uterine wall (interstitial pregnancy) are very rare. They may be regarded as misplaced pregnancies because they are both outside the proper uterine cavity, and delivery under natural conditions is usually quite impossible. In both conditions rupture is liable to take place, when the investing sheath of tissue proves insufficient for the requirements of the growing pregnancy. This usually happens about the fourth month.

The symptoms are then those of sudden internal hæmorrhage and collapse (see 4. Tubal Pregnancy, p. 1311) combined with the history and

physical signs of a somewhat advanced pregnancy which, in general character, differs but little or not at all from the normal.

As in all true extra-uterine pregnancies, the proper uterine cavity is somewhat enlarged and contains a 'decidua' which generally separates when any disaster happens to the pregnancy. Consequently some bleeding may be taking place from the uterus and vagina at the same time as the more serious hæmorrhage is taking place internally, and this may give rise to the (mistaken) diagnosis that the symptoms are only due to an incomplete abortion.

TREATMENT.—The abdomen must be opened and the pregnancy removed. In a ruptured cornual pregnancy the base or pedicle of the pregnant horn may be ligatured by a chain of three ligatures, and the whole of the pregnant horn removed. In an interstitial pregnancy it is usually necessary to remove the uterus, but if the abdomen be opened before rupture has taken place, the pregnancy may be removed by a uterine incision and the resulting wound closed by suture (*see American Journal of Obstetrics*, xxvii. 694).

4. *Extra-uterine Pregnancy (and its Consequences).*—Nearly all extra-uterine pregnancies begin within the Fallopian tube. A very few cases have been recorded which appear to have begun within the ovary. These are very rare. In *tubal pregnancy* the fructified ovum attaches itself to the inside of the Fallopian tube (instead of the uterus), and grows in this situation. The most common result of this misplaced attachment is recurrent bleeding from the fimbriated end of the tube into the abdomen (with or without rupture) causing intra-peritoneal hæmatocele. The next most common result is rupture with sudden and diffuse intra-peritoneal hæmorrhage. Both of these occur early in the history of the pregnancy. The least common result is the continued growth of the pregnancy outside the uterine cavity. This may take place by invasion of the abdomen from the tube (*tubo-abdominal pregnancy*); by invasion of the sub-peritoneal tissues below the peritoneal reflexion (*tubo-ligamentary or broad-ligament pregnancy*); by invasion of the uterus (*tubo-uterine or interstitial pregnancy*). *See* 3. Interstitial Pregnancy.

Roughly speaking, then, the three conditions, (a) intra-peritoneal hæmatocele, (b) diffuse intra-peritoneal hæmorrhage, and (c) the special tumour caused by an advanced pregnancy outside of the uterus, are the chief phenomena of this disease, each of which separately points to some special accident or stage of extra-uterine pregnancy.

(a) *Intra-peritoneal hæmatocele*, together with the enlarged and distended tube from which the bleeding comes, usually forms a tumour which can be readily felt on bi-manual examination.

It is often retort-shaped, it occupies the pouch of Douglas behind the uterus, it fixes and displaces the uterus generally forwards and to the opposite side, and the tumour is apt to enlarge suddenly owing to fresh hæmorrhage. It is then acutely tender and the abdomen above is distended and very sensitive to touch. The attendant symptoms are those of moderate shock, anæmia, and transient peritonitis. When these signs are found in a woman who is in a position to be pregnant and who has a history of any amenorrhœa (however slight) followed by irregular loss, with or without the history of the passage of a 'decidua' or decidual

shreds from the uterus, there is strong reason to suspect a tubal pregnancy as the cause of the disease. This suspicion is increased if all the symptoms and signs are of comparatively recent date.

TREATMENT.—If the tumour be well marked on examination of the posterior vaginal fornix and evidently extra-uterine—the uterus being well differentiated to one side of the tumour—the case may be treated by posterior vaginal cœliotomy.

The hæmatocele and pregnancy are cleared away after free incision of the pouch of Douglas. If necessary, the tube may be removed by the same route. The incision is afterwards drained by a tampon of iodoform gauze.

(b) *Diffuse intra-peritoneal hæmorrhage.*—This must be recognised by its symptoms. It is peculiarly apt to occur at a very early stage of tubal pregnancy before any recognisable tumour has had time to form, but occasionally takes place at later stages.

The patient, who has been previously quite well but has gone a week or more beyond her usual 'period,' is suddenly seized with abdominal pain. Within a few hours (or less) her condition becomes serious. Her pulse increases in frequency (80, 90, 100, 110, 120, 130, &c.), and this, as a rule, without any rise of temperature; she becomes faint and pale, and the pulse, as it rises in frequency, becomes very much weaker. Occasionally it is fluttering. The abdomen becomes distended and tender, and sometimes fluctuation of the fluid blood within the abdomen may be detected by abdominal palpation and percussion. On vaginal examination it may be possible to feel either the tumour of the pregnant tube or the soft, semi-clotted blood which has collected in Douglas's pouch, and which gives a sensation of slight boggy resistance on vaginal touch.

TREATMENT.—The abdomen must be opened and the hæmorrhage stopped by ligature and removal of the pregnant tube. The abdominal section should be by incision above the pubes, and it is well to thoroughly wash out the blood and blood-clot from the abdomen by copious irrigation with hot water. The pouch of Douglas is drained with a glass drainage-tube.

(c) *The special tumour caused by a more or less advanced extra-uterine pregnancy* differs according to its position. If 'abdominal' or 'tubo-abdominal' the whole of the child may generally be felt lying within the abdomen, uncovered by any containing sac. The uterus is quite distinct from the pelvis, and is but little or not at all enlarged. A lump to one side of or above the uterus (which is neither child nor uterus) marks the substance and situation of the placenta. The fetal heart is very plainly heard if the child be living; the placental 'bruit' may be difficult to find.

If 'ligamentary' or 'tubo-ligamentary' there is generally a fairly thick containing sac—a 'pseudo-uterus'—outside the child. This is sometimes central, but often to one side of the abdomen. The uterus is necessarily attached to one part of the wall of the sac and is therefore somewhat lengthened and enlarged. The sac is not of uniform thickness, and at some point either from the abdominal or vaginal wall 'ballotement' may probably be elicited. The placental 'bruit' is usually more easy to find than the pulsations of the fetal heart, but this is not a certain guide.

Both conditions here described need careful differentiation from a normal pregnancy within a uterus whose walls are exceedingly thin. Under this

condition a foetus may appear to be quite free in the abdominal cavity when it is really within the uterus.

TREATMENT.—If no dangerous symptoms appear, the pregnancy may be watched until the natural term be nearly reached. Then, every preparation having been previously made, the abdomen is opened and the pregnancy removed. If 'abdominal,' it is probably wiser to remove both child and placenta, the latter being usually mainly attached to the remains of the Fallopian tube. If 'ligamentary,' the containing sac of the broad ligament (after incision and removal of the foetus) may often be sewn to the sides of the abdominal incision, and the placenta be allowed to separate slowly and come away in the subsequent discharges.

IV. Diseases caused by Abnormal Conditions of the Uterus itself (or of the Uterine Appendages) other than Displacement.—(1) Pregnancy in one-half of a double uterus; (2) Pregnancy complicated by myoma of the uterus; (3) Pregnancy complicated by ovarian tumour.

1. *Pregnancy in one-half of a double uterus.*—The phenomena occasioned by this abnormality vary according to the extent of the 'doubling.' If the doubling be complete, affecting both uterus and vagina, there are of necessity two vaginæ and two

all stages, especially if we include under this heading any enlargement of the ovary and not only the large cystic mass usually characteristic of ovarian tumour. The ovarian enlargement necessarily forms a swelling outside of, and in addition to, the tumour of the pregnant uterus, and this may easily be found on careful vaginal, abdominal, or bimanual examination.

When complicating pregnancy, an ovarian tumour, however small, is peculiarly dangerous; for at parturition or miscarriage, or even without this, it is specially liable to twisting of its pedicle and then becomes a source of acute peritonitis and sudden danger.

TREATMENT.—If the presence of an ovarian or tubal tumour is made out with certainty, it is advisable to remove it. Pregnancy is no bar to successful operation. If the operation be done carefully, miscarriage very rarely follows. It is the rule for the gestation to continue without disturbance. If on diagnosis immediate operation be refused, the tumour should be watched so that no confusion may occur regarding the cause of any inflammatory symptoms suddenly arising.

V. Diseases caused by Pelvic Deformity or Bony Outgrowth.—The frequency of pelvic deformity varies according to locality, being common in some cities—such as Glasgow and Manchester—and very rare in others (as in Birmingham).

—	Iliac Spines	Iliac Crests	Conj. Ext.	Conj. Diag.	Conj. Vera
1. The simple flattened pelvis . . .	25	28	18	11	9
2. The rachitic flat pelvis . . .	27–29	28	18–13	11–6	9–4
3. The generally contracted pelvis . .	23	26	18	11	9
4. The generally contracted flat pelvis	23	26	16	9	7
The normal pelvis	25–26·3	28–29·3	20	13	11

uteri—the one pregnant and the other not. If examination be made through the vagina leading to the unimpregnated uterus, the tumour of the pregnancy will appear to be extra-uterine. By successive examination of each vagina in turn, the true condition can be ascertained and any danger of mistake avoided.

When the 'doubling' affects the uterus or bodies of the uterus only, and both are pervious to the single vagina, the unimpregnated uterus will form a small tumour to one side of the pregnant uterus, the two uteri being usually set at an angle to each other, the bodies diverging.

If the outlet from the pregnant uterus be free, no special treatment will be necessary; if closed, operation is imperative. See 3. Cornual Pregnancy, p. 1310.

2. *Pregnancy complicated by myoma of the uterus.* The influence of myoma upon pregnancy depends upon its seat or position. If affecting the upper two-thirds of the uterus and free to move, it almost invariably rises with the growth of the pregnancy and does not interfere with normal delivery. If growing from or near the cervix, wholly pelvic and especially if adherent or fixed, the gravest danger may be apprehended from the complication. In these cases it is often necessary to induce an early abortion or to remove the uterus and pregnancy together.

3. *Pregnancy complicated by ovarian tumour.*—This is a complication which needs recognition at

Pelvic deformity is usually (but not always) accompanied by visible general deformity or stunted growth; consequently any small or deformed patient, any one possessing distortion of the lower extremities, any *primipara* with a pendulous abdomen, or any *multipara* with the history of difficult confinements, should be examined by inspection, palpation, and by careful pelvic measurement before attendance during her confinement. The more common forms of contracted pelvis, with the typical measurements of each, are given in the above table, which is copied from the 'Obstetric Manual' of Dührssen. The measurements are in centimetres (10 cmm. = 4 $\frac{1}{2}$ in.).

In addition to these forms of pelvic contraction, extreme narrowing and distortion may occasionally arise from *osteomalacia*, and from local bony outgrowths or tumours. In some of these cases the pelvic canal may be altogether obliterated.

TREATMENT.—The minor degrees of narrowing demand delivery by forceps or version. The major demand either the induction of premature labour or some form of Cæsarian section. In *osteomalacia* it is wise to remove the uterine appendages.

VI. Associated or Intercurrent Diseases or Disorders in their relation to Pregnancy.

(1) Cholera; (2) Small-pox; (3) Scarlet fever; (4) Typhoid fever; (5) Pneumonia; (6) Acute yellow atrophy of the Liver; (7) Diabetes; (8) Malaria; (9) Syphilis; (10) Hereditary Weakness predisposing to Tubercle; (11) Lead-poisoning; (12) Gout;

13) Renal disease and Eclampsia; (14) Appendicitis; (15) Pelvic or Abdominal Adhesions; (16) Vaginal Fixation of the Uterus; (17) Operations on the Cervix; (18) Cancer of the Cervix; (19) Sarcoma; (20) Phthisis; (21) Heart-Disease; (22) Chorea (*see* p. 1308).

All disease caused by the introduction of some poison into the system has a deleterious effect on pregnancy in direct proportion to the dose of poison and force of the (usually) resulting inflammation (hyperpyrexia). Consequently the acute exanthemata (including typhoid-fever, cholera, ague, acute yellow atrophy of the liver, pneumonia, syphilis, and acute mineral or vegetable poisoning) are all exceedingly likely to cause the death of the foetus in any woman who is pregnant.

In *cholera*, the maternal mortality is about 55 per cent. The foetus is usually killed during the stage of intoxication.

In *small-pox* (unprotected) the maternal mortality has been computed at 30 per cent. Abortion occurs in one-half of the cases. When this happens, frequently soon follows the onset of the disease, and the mortality is high. Small-pox and its dangers can, of course, be largely prevented by timely vaccination. During an epidemic, pregnancy is no bar to re-vaccination.

In *scarlatina* both mother and child weather the storm if the attack be a mild one; but some epidemics, under insanitary conditions, have been very fatal. If severe, there appears to be a special danger in this disease of puerperal sepsis following the abortion or premature confinement: 'out of 21 cases, 6 ran a mild course, and in 8 sepsis occurred with 2 deaths' (Meyer).

In *typhoid fever* the maternal mortality has been computed at 30 per cent.: 'out of 91 cases 19 died' (Brieger). Abortion is the rule. 'Out of 22 cases of typhoid, 16 aborted, and the remaining six, who had slight attacks, went on to term' (Playfair).

In *pneumonia* a double poisoning is present; not only have we the direct poison of the disease, but carbonic-acid poisoning as well from interference with normal respiration. The maternal mortality varies from 50 to 100 per cent. More than half the cases abort. 'Pregnancy is interrupted in one-third of all cases before the sixth month, and in two-thirds of all cases from the sixth to the ninth month' (Wallich).

Acute yellow atrophy of the Liver (malignant jaundice) should probably be referred to under this heading, as the cause of it appears to be a blood-poisoning by schizomycetes (Winckel). This disease is peculiarly associated with pregnancy. It is rarely or never found during the first three months of pregnancy, but is more frequent after the fifth month. It usually causes premature expulsion of the foetus (in 42 out of 68 cases, Charpentier), and in many of these cases it has been noticed that the liquor amnii and child have been stained an orange-yellow by the jaundice. The emptying of the uterus may be regarded as simply an index of the strength of the poison. It is not, as a rule, attended by any improvement in the symptoms; on the contrary, the jaundice usually increases afterwards, and the patient dies with septic coma. About half of all patients affected succumb to the disease. The proportion of deaths in pregnant women is probably much higher.

True *diabetes* has a grave effect on pregnancy. Of 22 pregnancies in 15 mothers 4 ended fatally

during the puerperium. In 7 out of 19 cases the child died (after reaching viable age) during the pregnancy, and in 2 more it died shortly after birth' (Matthews Duncan).

In *malaria* the influence of the disease on pregnancy is relative and depends on the severity of the fever. 'Abortion is more frequent in malarial than in non-malarial countries.' We have now very good reason to believe that this danger may be largely or entirely prevented by the careful exclusion of the mosquito—the carrier of the infection. *See* MALARIAL DISEASE.

In cases of chronic poisoning, whether the poison comes from without as in ague, syphilis, and lead-poisoning, or from within as in chronic nephritis, gout, and struma, we not infrequently find a special effect on pregnancy induced which needs separate consideration. This may be characterised as the state of *Habitual Abortion*. The patient becomes repeatedly pregnant and after a varying period of gestation regularly aborts. *See* LEAD, POISONING BY; and OCCUPATION-DISEASES.

Syphilis is well known as a common cause of this, and too often every case of habitual miscarriage is treated with mercury and iodides on the supposition that syphilis is the probable factor, though perhaps concealed. This is bad practice. In nearly every case where syphilis is the cause of habitual abortion, the history or trace of its presence in one or both parents is plainly discoverable. Moreover, if the mother be young, if the general health be well maintained, and if there be no continued exposure to sexual excess or fresh sources of danger, the disease tends of its own accord towards recovery, and the patient, if repeatedly pregnant, tends to go longer without abortion until a still-born or syphilitic child is born at term—no abortion having taken place.

When the evidence of syphilis is plain, then anti-syphilitic treatment should be administered to both parents (the treatment of the wife alone is altogether insufficient). This should be continued for three years and every attention paid throughout the course to nutrition and to hygiene. When no trace or history of syphilis can be found, anti-syphilitic remedies should be carefully withheld. When uncalled for, they may do decided harm.

The hereditary weakness of tubercular families is not recognised as it should be as a cause of habitual abortion. In ordinary family practice it is in many districts quite as common a cause as syphilis, but needs altogether different treatment. When this is the cause there is always some family history of tubercle (phthisis, fistula, bone-disease, lupus, tubercular meningitis, or tubercular peritonitis), and this is probably of nearly as much importance on the side of the husband as on that of the wife. When either one or both parents possess a tubercular family history—showing that there has been some special weakness of one or both families favourable to the growth of the tubercle-bacillus—and when miscarriage following conception is a frequent or habitual occurrence, the consideration of the two conditions as directly associated will usually lead the physician to right and often to successful treatment.

In such parents everything which lowers body weight and general nutrition is fatal; consequently anything which tends to enfeeble the patient, the want of good food, the continued administration of mercury or repeated miscarriage, tends to lessen

the power of successful gestation; and in a series of abortions, the later ones tend to recur at earlier rather than at later periods until, in some cases, the power of conception fails. Exceptions to this rule may occur after prolonged holidays, and after any treatment which improves the general health.

The clue which this affords to treatment is of some importance. Attention to general hygiene and especially to sufficient 'holiday life' in the open air cannot be overrated, but the main essential in treatment is the persistent administration of Cod-liver oil and Syrupus Ferri Phosphatis Compositus to one or both parents until a healthy conception takes place. The same treatment is then continued to the mother throughout the whole period of gestation. By these means in a very considerable number of cases the tendency to abortion is counteracted and a healthy child is born at term.

Lead-poisoning may be a cause of habitual abortion among workers in lead, and lead is sometimes purposely taken in order to produce miscarriage. Several fatal cases have been recorded.

Gout and chronic kidney-disease may be causes of early miscarriage, but the chronic nephritis or special kidney-disease of pregnancy usually affects the organism of the mother much more than that of the infant and is the cause of one of the most dangerous of all the disorders of pregnancy, puerperal convulsions or eclampsia. See PUERPERAL DISEASES; and ECLAMPSIA.

The chief *surgical* affections influencing pregnancy or parturition are appendicitis, pelvic or abdominal adhesions, the operation of anterior colpotomy with vaginal fixation of the uterus, and cancer of the cervix.

Appendicitis is an occasional cause of abortion, sometimes it may cause 'habitual abortion.' Appendicitis gives rise to abortion in two ways. As an acute disease it may cause septic poisoning and pyrexia, destroying the fetus in very much the same way as the acute exanthemata; and, secondly, as a chronic source of trouble through adhesions to the right appendages and right side of the uterus it may interfere with the normal progress of gestation and so induce miscarriage.

Pelvic or abdominal adhesions other than those due to appendicitis may likewise be a cause of habitual abortion, and strong adhesion or fixation of the cervix anteriorly may be a cause of difficult labour. This is an occasional result of some surgical operations on the uterus and of cancer of the cervix.

Anterior vaginal caliotomy with fixation of the uterus.—The opening of the peritoneum between the cervix and bladder and the sewing of the fundus forwards to the sides of the vaginal incision is usually (when performed rightly and with due aseptic care) an operation having no untoward sequelæ. If, however, the union be not rigidly 'sero-serous,' and if the wound become septic and there be any prolonged suppuration, it is quite possible to get such firm union and fixation as to complicate or prevent the proper delivery of a subsequent pregnancy. Under these circumstances it has been necessary on one or two occasions to perform Cæsarian section. For this reason, in the operative treatment of non-adherent retroflexions, the writer prefers and strongly recommends the shortening of the round ligaments without opening the peritoneum.

Other operations on the uterus, such as the repair of a deep laceration of the cervix or the amputation or excision of the cervix, may likewise be followed either by firm fixation or contraction; the resulting cicatricial tissue interfering with the proper discharge of the menstrual function or with the dilatation of the cervix necessary for delivery.

In one case of this kind in which the writer had to remove the uterus, the uterine tissue tore out before the adhesion would give way.

In some of the cases where there is marked obstruction in delivery the vaginal Cæsarian section of Dührssen offers a readier and less dangerous method of delivery than that by the abdominal route. The chief objection to this method consists in the occasional great difficulty of uterine suture through the vagina after delivery.

Cancer of the cervix may produce a very similar condition of rigidity and fixation to that already described as occasionally resulting from inflammation. In a few cases too, cancer of the rectum invading the broad ligament may produce an unyielding bed of tissue in which the uterus cannot properly dilate.

When such conditions are associated with advanced pregnancy Cæsarian section is the usual method of dealing with the difficulty. When found at an early stage of pregnancy the propriety of emptying the uterus may be reasonably considered.

Cancer or sarcoma may affect pregnancy in other ways. One form of sarcoma—deciduoma malignum—only occurs in connection with pregnancy or the post-puerperal period. It grows from the placental site, invades the uterus and is usually rapidly fatal. See PLACENTA, Diseases of the.

Cancer often appears to grow with increased luxuriance during the later stages of pregnancy, and in cases allowed to drift the mortality of the puerperium is very high. Forcible extraction of the child by forceps or turning is often accompanied by rupture of the uterus or bladder, and if the patient survives the hæmorrhage and shock, she may still have to encounter the dangers of peritonitis and sepsis.

Phthisis has, as a rule, less direct effect on pregnancy than the state of constitutional weakness predisposing to consumption. If a woman, already suffering from phthisis, become pregnant, her reproductive power is usually strong, and though abortion may result, it is by no means a necessary consequence.

A very considerable number of women suffering from phthisis immediately improve in health and in general nutrition after conception has taken place, and if the surroundings and diet be good, and if hæmoptysis does not occur, this improvement is maintained throughout the whole course of the pregnancy. The birth may be premature, and the child tubercular, but in most instances the pregnancy advances to term without disturbance, and the child is apparently healthy. But after the pregnancy is over there is almost always a severe relapse. The gain in nutrition is rapidly lost; the lung-disease advances, and the patient very often only lives a few months after the birth of her infant. This may to some extent be guarded against by careful management during the lying-in, but does not interfere with the general rule that the post-puerperal period is very fatal in phthisis.

In *Heart-Disease* it is also the post-puerperal time that is usually more dangerous, but the danger

varies greatly according to the disease and the presence or absence of compensatory changes.

Mitral Stenosis appears to be the most fatal of all cardiac lesions in pregnancy. Out of 54 reported cases no less than 35 were fatal, but this includes some cases of double mitral lesion.

Cases of *aortic disease* come next in importance. The mortality of pregnancy complicated by aortic disease is estimated at 23 per cent. in Norris's System of Obstetrics.

Mitral Regurgitation is of less gravity, especially in earlier years, when the patient is young and healthy. Repeated pregnancies, however, slowly tell upon the power of the heart, and the danger of the patient is markedly increased at every succeeding pregnancy. According to the same authority, the mortality of this complication is stated to be 13 per cent.

In aortic disease the most fatal period is during labour; when this is safely over there is less danger.

In mitral disease the post-puerperal period is quite as dangerous as that of labour. In fatal cases the right side of the heart has been found engorged.

Cardiac disease may induce *secondary pulmonary lesions*, both during pregnancy and in or after parturition. Of these the most important are hæmorrhage into the lung-tissue and hæmoptysis, œdema of the lungs, and congestive pneumonia. Cardiac disease is often the cause of excessive loss of blood at the time of confinement, and of faulty contraction of the uterus after delivery. The latter, by retention of blood-clot which the uterus is unable to expel, favours slow septic sequelæ.

TREATMENT.—'Pregnancy associated with cardiac disease should not be interrupted artificially.' 'The best treatment will therefore be symptomatic, and consist in administering digitalis, strophanthus, nitrate of potassium, diuretics, or diaphoretics, and in regulating the diet' (Winckel). During delivery every effort should be made to calm excitement—moderate anaesthesia—to prevent unnecessary blood-loss, and to secure good uterine contraction. Ergot should be given with caution or altogether withheld if its administration is likely to further embarrass the heart's action. During the lying-in, good nursing is of the utmost value. Psychological excitement or irritation is especially harmful, and trouble should be taken to secure the patient from any possibility of worry or anxiety.

JOHN W. TAYLOR.

PREMONITORY (*præ*, before; and *monéo*, I warn).—This word is associated with symptoms which give an indication or warning of the advent or onset of certain diseases or seizures; for instance, rigors during the invasion of fever, and the various auræ preceding an epileptic fit.

PREPUCE.—See PENIS, Diseases of; and CONCRETIONS.

PRESBYOPIA (*πρέσβυς*, an old man; and *ὤψ*, the eye).—Impairment of the power of accommodation of the eye, the result of progressive senile changes, in consequence of which the nearest point of distinct vision lies at more than nine inches from the eye. Distant vision may be perfect; but the eye, unaided by an appropriate convex lens, cannot see clearly objects less than nine or more inches from the eye. See VISION, Disorders of.

PREVENTION OF DISEASE.—See PERSONAL HEALTH; and PUBLIC HEALTH.

PRIAPISM.—SYNON.: Fr. *Priapisme*; Ger. *Priapismus*; *Ruttenkrampf*.—A continuous erection of the penis, generally unaccompanied by any sexual desire. It may be complete or incomplete. In the former case there is a rigid erection of the penis with much pain and suffering. The condition may last for many days, or even weeks or months. There are many causes of complete priapism, notably injuries to the penis which cause rupture and extravasation of blood. It may also occur in leucæmia and other toxæmias, in diseases of the brain, spinal cord, rectum, bladder, and in tetanus and hydrophobia. In incomplete priapism the penis is merely distended with blood unaccompanied by any muscular contraction, rigidity or pain. This condition is found in injuries to the spinal cord in the cervical and upper dorsal region.

TREATMENT.—In those cases where there is a collection of blood-clot this must be removed by incision. In all cases treatment is very unsatisfactory. The application of cold or heat is useful, and trial may be made of such drugs as bromides, chloral hydrate, hyoscine, and opium.

CHARLES GIBBS.

PRICKLY HEAT.—An acute eruption of minute papules and vesicles which cover the skin more or less extensively, and are attended by burning heat, and a most tormenting prickly itching. The affection occurs for the most part in hot climates, is associated with profuse sweating, and attacks principally those who are unaccustomed to extreme heat; hence it is often experienced by travellers in tropical regions. Pathologically the eruption seems to consist principally of dilatation of the sweat-ducts, caused by the blocking of their orifices. This is due to swelling of the surrounding epidermis. The papillæ are enlarged from inflammatory infiltration. The rash generally subsides in the course of a week.

PROCIDENTIA (*pro*, downwards; and *cado*, I fall).—A falling down of certain organs or structures from their natural position, as of the uterus, rectum, or iris. See PROLAPUS.

PROCTITIS (*πρωκτός*, the anus).—Inflammation of the anus or rectum. See PERIPROCTITIS; and RECTUM, Diseases of.

PRODROMATA (*πρό*, before; and *δρόμος*, a course).—A synonym for premonitory symptoms. See PREMONITORY.

PROGRESSIVE MUSCULAR ATROPHY.—SYNON.: Chronic Spinal Muscular Atrophy; Wasting Palsy; Amyotrophic Lateral Sclerosis; Chronic Poliomyelitis; Fr. *Atrophie Musculaire Graisseuse progressive* (Duchenne); Ger. *Muskelatrophie*; *Muskellähmung*.

DESCRIPTION.—Slow wasting of the muscles, beginning in some particular part, spreading and increasing, until it is wide in extent and extreme in degree. The changes in the muscles depend upon a slow degeneration in the ganglion-cells of the anterior cornua of the spinal cord, accompanied by a degeneration in the motor nerve-fibres arising from the cells. With this degeneration of the cells and fibres there is usually also associated a similar

change in the pyramidal tracts of the cord, sometimes, at least, to be traced up to the motor cortex of the brain, and related to a similar degeneration of their ganglion-cells.

Charcot has divided cases of this disease into two varieties, the so-called 'protopathic' form, in which the affection consists essentially of a primary degeneration of the motor cells of the cord, of the nerve-fibres proceeding from them, and of the muscles; and the 'deuterothropic' in which there is also degeneration in the pyramidal tracts, to which the affection of the spinal cells and fibres was thought to be secondary. To this second class he gave the name 'amyotrophic lateral sclerosis.' But the distinction is not valid. The pyramidal fibres always degenerate with the motor cells and at the same time. The two varieties are essentially the same disease. The difference between them is that, in the one case there is degeneration only of the lower, spinal, motor neuron, in the other there is also decay of the cerebral neuron, which comprises the cortical cell and pyramidal fibre.

ETIOLOGY.—Males are more frequently attacked than females in the proportion of about three to one. The disease is one of adult life, commencing as a rule between the ages of twenty-five and fifty-five. Cases have been recorded in which the affection commenced as early as twelve and as late as seventy, but most juvenile atrophies are primarily muscular. An inherited neuropathic disposition is to be recognised in a small proportion of cases, but direct inheritance of the disease is very rare. All classes of society are affected, and it is doubtful whether workers with the muscles suffer with greater frequency than is accounted for by their greater exposure to certain exciting causes. Of these, one of the most important is mental distress and anxiety, an influence especially potent in later life. Another distinct cause is exposure to wet and cold, which is a cause of so many chronic spinal diseases. Sometimes the exposure has been habitual; sometimes there has been a single severe exposure, immediately succeeded by neuralgic pain, either in the part which subsequently wastes, or in other regions. Excessive use of muscles may cause slight local wasting; it is doubtful, however, whether this influence produces general muscular atrophy. Severe concussion of the cord is a rare cause, and seems usually to act by setting up disseminated myelitis, manifested by muscular wasting combined with other symptoms. In a few cases, however, concussion has been slowly followed by typical progressive muscular atrophy. More rarely a fall, in which a limb has been injured, has been followed by muscular atrophy commencing in that limb. Syphilis is an occasional antecedent, and no other cause may be traceable. In the cases in which atrophy has followed an acute specific disease such as measles, the malady is probably a chronic neuritis. Lead-poisoning may produce chronic muscular atrophy, as well as the common acute paralysis with rapid wasting. The chronic form closely resembles ordinary progressive muscular atrophy in seat and features, differing from the acute extensor palsy. In many cases no cause for the disease can be discovered. It seems then to be a premature failure of vitality in the nerve-elements concerned.

ANATOMICAL CHARACTERS.—The muscles are reduced in size and pale in colour. Parts may be hardly distinguishable from adjacent fat. On the

other hand, the muscle may be dark as a whole, and pale streaks in it may mark the position of local degeneration. There are four well-defined microscopic changes in the fibres: (1) Simple narrowing, with little or no change in the striation. The striae sometimes appear to be farther apart than normal, or the fibrillary segmentation is unusually distinct. (2) Simple fatty degeneration, the transverse striation giving place to granules, mixed ultimately with fatty globules. (3) So-called 'vitreous degeneration,' probably a distinct process from fatty degeneration. The sheath, in such a condition, contains only a clear material enclosing a few fatty globules and a few faint transverse striae. (4) A longitudinal striation develops in the fibre, with which at first the transverse striation co-exists. As the latter becomes more and more faint, the fibre comes to have the appearance of a fasciculus of longitudinal connective-tissue fibres. This change may sometimes be present alone. Fatty globules may accumulate between the fibres, with, in some cases, granules and masses of reddish-brown pigment. The interstitial nuclei are often increased, and sometimes the interstitial tissue. The capillaries may be dilated and distended. Muscular fibres, practically unaltered, may often be seen side by side with others profoundly changed. The sheaths finally become empty and shrink, and may be scarcely distinguishable from the interstitial tissue.

There are many degenerated fibres in the peripheral nerves. These can be traced to the anterior roots, which are diminished in size. The amount of degeneration in them corresponds to the wasting present in the muscles. The posterior roots are unaltered. The spinal cord is often softer than natural at the affected part, and the white substance of the lateral columns is grey and translucent. With the microscope, after due preparation, the anterior cornua are seen to be much changed at the level at which the nerves to the muscles most affected are given off. Most of the large cells have disappeared, or are represented by small angular bodies. Frequently a few large cells can still be seen, but most of these have lost their processes, and are more globular than normal. The interstitial tissue is also increased.

There is distinct degeneration in the anterior root-fibres passing from the cornu through the anterior column. A few fibres may remain, but whole fasciculi appear to be replaced by fibrous tissue. There is also usually distinct degeneration of fibres in the anterior commissure.

In the white columns there is usually considerable, often almost complete, degeneration of the pyramidal tracts, anterior and lateral, and the resulting sclerosis extends, in slight degree, beyond their limits. The degeneration can often be traced up through the decussation of the pyramids, and even through the pons and crus to the internal capsule. By the products of degeneration it has even been traced through the white substance to the cortex. But it has also been found to cease at the crus or at the medulla.

In cases in which bulbar symptoms have been present the motor nuclei in the medulla have shown changes similar to those present in the grey matter of the cord. In other cases the changes in the nuclei have been slight; but in such cases the degeneration of the pyramids has been extreme, and no doubt has involved the fibres connecting those nuclei with the cortex. The sympathetic nerves

and ganglia, when examined, have shown no considerable alteration.

SYMPTOMS.—Wasting and weakness usually come on together; but in parts not readily observed, such as the shoulder, the loss of power is frequently noticed before the wasting has been apparent. In the hand, not infrequently impairment of the power to carry out some fine action, such as writing, draws attention to the wasting.

The disease commences in the upper limb in nine-tenths of the cases, and almost as often in one arm as in the other, and as frequently in the hand as in the shoulder-muscles. From the part first affected it spreads to the other muscles of the limb. In the hand, the thenar muscles and interossei are usually the first to suffer, and of the latter the abductor indicis is usually most conspicuously affected. Occasionally the disease begins in the forearm, especially in the extensor muscles; the ulnar extensors then suffer most. Of the shoulder muscles, the deltoid is generally the first to waste; the supra- and infra-spinati are often affected with it; and the triceps usually suffers less and later than the biceps.

The muscles of the back are in most cases early involved in the wasting. The middle and lower parts of the trapezius suffer first, the rhomboids and erectors of the spine later. The serratus, latissimus, and pectoralis major are subsequently involved, but they may escape wholly or in part. According to the affection of the muscles connected with the capula, the position of the bone changes. The highest part of the trapezius shows sometimes (but not always) a remarkable indisposition to waste, and it was hence termed by Duchenne the *ultimum moriens*. The muscles that extend the head on the spine often suffer in considerable degree, and a difficulty in the carriage and movement of the head is the result. The increased efforts of the weak extensors to balance the head on the spine, under such conditions, often cause a synergic over-action of the frontales, which are habitually associated in action with the extensors. The skin at the back of the neck lies in transverse folds when the head is put back. In striking contrast to the wasting of the neck is the condition of the platysma, which always escapes, and not uncommonly undergoes hypertrophy as if in an attempt at compensation. The muscles of respiration suffer in the majority of cases, and their implication constitutes a grave source of danger to life. The intercostals rarely escape altogether, and the diaphragm is involved in many cases. The muscles of the abdominal wall occasionally waste, but far less frequently than those of the thorax.

Wasting in the legs is much less common than in the arms, and if it occurs is usually slighter in degree; occasionally the disease first manifests itself in the legs, and is more intense there than elsewhere. The face almost always escapes the general atrophy, and its normal appearance may present a striking contrast to the rest of the body. But the lips and tongue often become paralysed as part of the bulbar palsy which so often supervenes on the spinal disease.

As the wasting progresses, hollows and prominences, normally invisible, become manifest, and various contractions and deformities occur as a result of the unequal affection of antagonistic muscles. In the hand the usual deformity is that of the 'claw-hand,' a result of paralysis of interossei,

with unantagonised action of their opponents. Lordosis is common when the muscles of the trunk and thigh are involved.

The electrical irritability of the affected muscles varies in character. The rule is that, in a slowly progressive case, there is diminution in the readiness and degree of response to each current, a diminution which goes on *pari passu* with the wasting, until finally, when the wasting is extreme, it becomes extinct. But the voltaic irritability of the muscular fibres very often persists long after it is impossible to elicit any response to faradism, which acts only on the nerve-fibres. Although the quality of the voltaic irritability is usually normal, the qualitative change of the 'reaction of degeneration' without the quantitative may be present. When the affection is rapid in its course, the muscles may retain considerable voltaic irritability long after that to faradism has become extinct, and a typical reaction of degeneration may be present. Between the extreme forms various intermediate conditions may be met with.

The mechanical irritability of the muscles is considerably increased, and spontaneous flickering—the so-called 'fibrillary twitching'—in the muscles is frequent, but not invariable. It is not uncommonly observed in muscles not yet wasted, but in which atrophy subsequently occurs.

The myotatic irritability in cases in which there is 'atonic atrophy,' the muscles being flaccid and toneless, is lost, and lost early. But where there is rigidity from the first ('tonic atrophy'), even when the wasting is considerable, the myotatic irritability is preserved and sometimes increased, but the wasting does not attain the extreme degree of the 'atonic' form, and faradic irritability is not lost.

Beyond the vague pains already referred to, which sometimes occur in the region in which wasting of the muscles is afterwards perceived, there are no sensory symptoms. 'Numbness' and 'deadness' may be occasionally complained of, but cutaneous sensibility is never impaired, nor do the muscles lose their sensibility.

The functions of the sympathetic are not, as a rule, impaired. Dilatation or contraction of one pupil has been observed, chiefly in connection with atrophy of muscles that are supplied from the lower part of the cervical region. The reflex action of the iris is usually normal, and optic nerve-atrophy never occurs. Nystagmus is rare.

The visceral functions are usually little disturbed. Sexual power, indeed, may be lost, but the sphincters rarely suffer, even when the wasting is extreme. Slight, but inconstant, changes have been found in the urine. Glycosuria has been associated with bulbar symptoms, but only in a few instances.

COURSE AND COMPLICATIONS.—In most cases the malady is steadily progressive, but the rate at which it advances varies much. It may, however, become stationary; unfortunately the period at which this most frequently happens is in the later stages of the disease, when little but life remains. Sometimes progress ceases at an earlier stage, especially when the atrophy is strictly symmetrical. When its course at the commencement of the wasting is rapid, it usually continues rapid until it has attained a wide extent. When it begins slowly, it continues slow throughout, but there is rarely any acute paralysis of some particular group of muscles, which may come on in a few days. The extensors of the wrist and fingers are the muscles most

commonly affected in this way. When there is weakness of the legs without wasting (spastic paralysis), the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. It is rare for the weakness in the legs to occur first.

The most frequent complication of progressive muscular atrophy is bulbar paralysis, the result of a degeneration of the bulbar nuclei similar to that in the spinal cord. In rare cases muscular atrophy in the arms is accompanied by the symptoms of locomotor ataxy in the legs; and general paralysis of the insane has been met with as an exceptional complication. Severe and frequent headaches are occasionally present throughout the course of the disease.

The chief danger to life is from pulmonary disease when the medulla is involved, induced by the entrance of particles of food into the lungs, or by bronchial catarrh, both of which are rendered grave by weakness of the muscles of respiration. Bulbar paralysis also is a frequent cause of death, by its interference with swallowing and nutrition. Less commonly, death results from profound weakness, bedsores, and septicæmia, or from intercurrent maladies which would otherwise be trifling.

PATHOLOGY.—The constancy of the association of changes in the motor cells of the spinal cord, degeneration of the motor root-fibres, and wasting of the muscles, together with the analogous effects of focal lesions of the anterior cornua, leave no doubt of the relation of the muscular wasting to the disease of the cells and of the fibres proceeding from them. The condition of electrical excitability, its slow failure as nerve and muscle degenerate together, is explained by the slowness of the change in the nerves, permitting a similar rate of degeneration in the muscle. It is only when the usual slow process is varied by a more acute change in the cells and fibres, that the muscular tissue is for some time less damaged than the nerve-structures, and so presents paralysis in excess of wasting, and voltaic irritability in excess of faradic. Thus a slow decay of the lower neuron of the motor path is the essential lesion in this disease, to which the conspicuous symptom is merely secondary. But the disease is rarely limited to the lower neuron. The pyramidal tracts, as already stated, are commonly degenerated, and it is probable that the degeneration usually involves the motor cells of the cortex. There may be no symptoms to suggest such a condition. The degeneration of the lower segment causes such atonic atrophy of the muscles as to prevent the usual manifestations of disease of the cerebro-spinal neuron. The latter may be presented as spastic paraplegia when the spinal neuron for the legs is unaffected. The relative degree in which the two neurons are affected varies considerably, and gives rise to corresponding variations in the degree of atrophy with atony, or spasm without atrophy, or with only such as is moderate in degree.

In some cases the muscular wasting is considerable, although there is rigidity and excess of myotatic irritability. In this condition of 'tonic atrophy' it is common to find that many nerve-cells have disappeared or are very small, while others remain normal or slightly changed in aspect. We have then apparently, in addition to the degeneration of the upper neuron and the nutritional changes mentioned, a considerable degeneration of many but not of all the elements of the lower neuron. This cannot be regarded as simply secondary to the

degeneration of the upper segment. It must be the expression of a distinct pathological tendency similar to that which elsewhere causes the atonic atrophy and total wasting, but insufficient to prevent the less affected cells from causing rigidity under the influence of the degeneration of the upper segment. It is doubtful whether the tonic atrophy ever goes on to atonic atrophy. The rigidity which is the effect of degeneration of the upper neuron is not produced if the lower is already so much changed as to abolish myotatic irritability. And yet the pyramidal tracts are constantly found degenerated, although the muscles have been flaccid to the last. In the rare cases in which muscles with atonic atrophy become rigid towards the end of the process, this seems to be the result of changes in the muscles themselves, and is not directly dependent upon the central nervous system.

DIAGNOSIS.—The diagnosis of the developed disease is simple. Doubt is only likely to arise in the early stages and when limitation to the one group of muscles raises the question whether the origin of the wasting is local or central. Such local atrophy is generally the result of an affection of peripheral nerves; and paralysis due to disease of a single nerve, or a plexus, will usually be recognised by its limitation and often by associated sensory symptoms. The distinction of some forms of multiple neuritis is much more difficult. This is especially the case when there has been a subacute onset, such as is common in neuritis, and this difficulty is increased by the fact that neuritis may affect only motor branches, as in lead-palsy. But the subsequent slow wasting of other parts usually renders the character of the spinal disease evident. In cervical pachymeningitis, which may cause extensive muscular atrophy in the arms, the association of sensory symptoms is distinctive, and the same applies to tumours of nerve-roots. Disseminated myelitis may cause widespread muscular atrophy, but symptoms of irregular damage to other than motor parts of the cord are also present. In syringomyelia, atrophy may occur indistinguishable from that in the disease now under consideration, but a careful examination will reveal the peculiar disturbance of sensibility, the loss to pain without loss to touch, characteristic of this condition.

The distinction from idiopathic muscular atrophy, especially when this begins late in life, is sometimes difficult. This is only true of the cases where no pseudo-hypertrophy is present. The characteristic distribution of the wasting, especially its commencement in the legs, its very slow course, and the tendency to affect other members of the same family, will usually suffice to distinguish the idiopathic form. Whenever several cases of muscular atrophy occur in the same family, or atrophy begins during childhood or youth, the probability is great that the affection is idiopathic and not spinal.

PROGNOSIS.—The nature of the disease renders the prognosis in every case grave and uncertain. The chief factors in forming an opinion are the observed progress of the disease, and the age of the patient. The possibility of arrest is greater in middle life than in advanced age. It also seems to be greater in cases in which the wasting is symmetrical than in those in which the two sides are irregularly affected. Spontaneous cessation rarely tends to take place until an advanced stage is reached, but arrest by treatment may take place at any period. Bulbar symptoms increase the gravity of the pro-

gnosis; and weakness of the respiratory muscles, especially combined with an affection of the medulla, is a most grave indication. If the malady ceases to advance, there may be a slight amount of recovery of power, especially if the loss has been rapid and recent. Wasting that has existed for some months will probably remain unchanged. In a typical chronic case there is little hope of any actual recovery of tissue or power, as these depend upon destruction of nerve-elements which seem incapable of restoration, and the definite arrest of such a disease is a far more satisfactory therapeutical result than it can possibly seem to the sufferer.

TREATMENT.—Favourable conditions of life—fresh air, gentle exercise, the absence of mental strain—are essential. Nervine tonics, suited to degenerative processes, should be given, especially quinine and arsenic, alternated with phosphorus. But the only agent which has repeatedly arrested the disease is the hypodermic injection of strychnine, first advocated for this purpose by the writer. His results have been amply confirmed. It is especially in cases which begin between thirty and fifty that the treatment has been successful. In senile cases it has seldom done more than arrest the disease for a year or so. Administration of the drug by the mouth has not the same effect, and failure by the mouth does not lessen the prospect of good from its subcutaneous use. It may be that the drug, administered hypodermically, is brought more rapidly in contact with the nerve-elements, and acts on their nutrition with a relatively greater momentum. It is best to give one injection daily (or two in some cases), at any convenient place. The most convenient salt is the nitrate, and the dose should be $\frac{1}{10}$ of a grain increased to $\frac{3}{10}$ or $\frac{1}{5}$. After four months it is well to intermit the injections for one week in three or four. The treatment needs to be continued for six or twelve months, and, with intermissions, for the same or a longer period. Other nervine tonics, as arsenic, may be given by the mouth at the same time. In a malady so grave it is desirable to neglect nothing that may have a beneficial influence.

Local treatment has not much effect on the wasting. Electricity and massage are of some service, but have no influence on the essential disease. The constant current is that which is in general of most service, but faradism may be used if the muscles respond to a strength of current which is not painful.

No special bath-treatment is of service. Antisyphilitic treatment invariably fails in cases of the true disease, and, if energetic, may accelerate its progress. The writer has twice known the malady to develop after a course of thorough mercurial treatment. It is otherwise, of course, in cases of syphilitic pachymeningitis, which should not, however, be confounded with the disease. The degenerative sequelæ of syphilis are the result of a sequential toxin, and its effects are not lessened by treatment for syphilis. It is important that this should be noted.

W. R. GOWERS.

PROLAPSUS (*pro*, forward; and *labor*, I slip).—This word signifies that an organ or structure has fallen or slipped down, but implies a greater degree of displacement than *procidencia*; so that the organ or structure may protrude through a natural or artificial orifice. The condition is of

most importance in connection with the rectum and the uterus. See *PROCIDENTIA*; *ANUS*, Diseases of; and *UTERUS*, Diseases of.

PROPHYLACTIC } (*πρό*, before; and *φυλά*
PROPHYLAXIS } *άσσω*, I guard).—These terms are used in connection with treatment, and indicate the means employed for the prevention of disease. See *PERSONAL HEALTH*; *PUBLIC HEALTH*; and the articles on the special diseases.

PROSOPALGIA (*πρόσωπον*, the face; and *άλγος*, pain).—Prosopalgia signifies pain about the face. It may depend upon neuralgia of one or more branches of the fifth pair of nerves (see *TIC-DOULOUREUX*). Its paroxysmal character, unilateral position, and anatomical localisation will indicate this form. Another form is of rheumatic origin. In this the pain is more or less constant, diffused about the face or forehead, and does not follow the course of a nerve-branch. Movements, and especially stooping, increase it. Occasionally such pain is of syphilitic origin, and is especially apt to occur in connection with the appearance of the secondary rash.

DIAGNOSIS.—In rheumatic prosopalgia the pain is diffused and increased by pressure. If it depend on syphilitic periostitis there will be tenderness on pressure, and the parts will be swollen and less elastic than normal. There will also very likely be a certain amount of fever; and the pain will be increased at night.

TREATMENT.—Chloride of ammonium in half-drachm doses, dissolved in half a tumbler of water, should be given every four hours. If there be any evidence of syphilitic infection, iodide of potassium should be given, in doses of from ten to twenty grains every four hours.

For the rheumatic form of face-ache five grains of iodide of potassium, with thirty grains of bicarbonate of potassium, may be given every four or six hours, after the administration of an aperient. This may be followed up by sulphate of quinine or iron. Locally, a mixture of equal parts of camphor, chloral hydrate, and menthol, rubbed down together in a mortar, may be applied; or a liniment containing chloroform, belladonna, and opium. Decayed teeth should be properly treated. See *PAIN IN VISCERAL DISEASE*; and *NEURALGIA*.

T. BUZZARD.

PROSTATE, Diseases of.—**SYNON.**: *Fr. Maladies de la Prostate*; *Ger. Krankheiten der Prostate*.

GENERAL RELATIONS.—The points of practical importance in connection with the anatomy of the prostate are as follows: In the examination of the rectum the healthy prostate is felt as a firm substance in the middle line, somewhat divided into two lateral lobes. The whole organ is about $1\frac{1}{2}$ inch in width, with its apex opposite, namely, in the recumbent posture below, the apex of the pubic arch; that is, about $1\frac{1}{2}$ inch from the anus, in a moderately thin subject, but much farther in a very fat one. The whole gland is $1\frac{1}{2}$ inch in length, its posterior limit being usually about three inches from the anus—in other words, about the distance to which the forefinger can reach. From this it may be deduced that, as the *trigonum vesicæ* commences immediately behind it, a fully distended bladder masks more or less completely the natural

outline of the gland. It may also be gathered that the vesiculæ seminales are beyond the ordinary reach of the finger, and that when these are infiltrated by disease, their apices alone, or perhaps only the vasa efferentia, can be detected. The practitioner should by no means neglect the digital examination of the prostate, as it will often yield information of the greatest value; and it may be observed that the best position for the patient, if it be desired to compare the relative size of the lobes of the prostate, is the supine, or the genupectoral; whereas, if it be required to explore the rectum as far up as possible, the patient should be placed on one side with the hips flexed. The deviations from the normal type he may expect to meet with are—uniform or partial enlargement from simple hypertrophy, or from chronic or acute inflammation, in the latter case possibly attended by a sense of yielding or fluctuation, due to abscess; irregular hardness, most marked about the vasa efferentia, depending on a tubercular deposit; the existence of small hard nodular masses, which are calculi in the substance of the gland, and which may sometimes be felt grating against one another; or the irregular enlargement caused by a new-growth. It must be borne in mind that tumours or abscesses originating in neighbouring parts may surround the prostate and completely obscure its outline; thus the writer has met with the case of a large hydatid cyst between the rectum and the bladder that rather closely simulated malignant disease of the prostate, and effectually prevented its actual condition from being determined. It will not be forgotten that a certain degree of tenderness of the prostate does not imply a deviation from health, and that a more or less considerable enlargement in old age is so common as to be practically reckoned by some authors as normal. The effect of this enlargement on micturition will be mentioned farther on. The copious plexus of veins which surrounds the prostate communicates freely with those of the penis and rectum; and it is not unimportant, from a clinical point of view, to remember that these are thus connected not only with the systemic but with the portal circulation. These veins may become the seat of phlebitis and its sequelæ from various causes.

The principal diseases of the prostate may be considered in the following order:—

1. **Prostate, Hypertrophy of.**—The results of Sir Henry Thompson's observations were, that one-third of all men over fifty-five have some enlargement of the prostate; but that a comparatively small number of these suffer any inconvenience from it; and that it usually begins between the ages of fifty-seven and sixty—rarely, if ever, before, though it may more rarely commence later. Very considerable enlargement of the lateral lobes may cause no inconvenience; but if the part which forms the floor of the prostatic urethra, the so-called middle lobe, be even slightly enlarged, or if, as has been shown by McGill, a ring of prostatic tissue be formed at the neck of the bladder, difficulty in micturition is sure to result. It is thus easy to understand how a simple hypertrophy may reach enormous dimensions without giving rise to symptoms, while those which are caused by the enlargement of a prostate, which feels almost normal to the finger introduced into the rectum, may, on the other hand, be very severe indeed.

SYMPTOMS.—The symptoms are briefly these:

The stream of urine becomes dribbling, and there is an obvious difficulty in emptying the bladder; there is frequency of micturition, especially at night and in the early morning; perhaps a little pain before the act, but none afterwards; and no alteration in the character of the urine. If unrelieved, these early symptoms are followed by incontinence, depending upon over-distension of the bladder; and, from the same cause not improbably, cystitis and dilatation of the bladder, dilated ureters, and, perhaps, pyelitis and chronic interstitial nephritis. Patients with chronic hypertrophy of the prostate usually suffer from time to time from attacks of acute congestion, such as are described later on. See PAIN IN VISCERAL DISEASE.

ANATOMICAL CHARACTERS.—The structure of a hypertrophied prostate is but a slight modification of that of the gland itself; but sometimes the glandular element predominates, sometimes the muscular. Distinct encapsuled tumours are often found in hypertrophied prostates, which may be either fibromyomata or adenomata. The latter are occasionally pedunculated.

TREATMENT.—In regard to treatment of hypertrophy of the prostate it is only necessary here to give two words of warning. First, that most of the evils resulting from this condition depend upon the fact that the bladder is never emptied. It is essential, therefore, that the patient's powers in this respect should be ascertained without delay by catheterisation, and if it be discovered that more than an ounce or two of residual urine remains, he should be taught to pass an instrument himself, and directed to do so at least once a day. Secondly, cystitis has often been caused by setting up decomposition of the urine by a catheter not surgically clean. The simple precautions of washing it before and after use in an antiseptic solution (say 1 to 20 carbolic acid, or 1 to 2,000 sublimate), and of applying some really trustworthy antiseptic or aseptic lubricant, will avert with certainty this catastrophe, and prevent the unnecessary loss of many lives. Nothing can be safer or less irritating for the latter purpose than oil recently boiled. The reader must consult surgical works as to the difficulties which an enlarged prostate offers to the introduction of a catheter, and the manner in which they may be overcome. A method of removing the middle lobe or the ring of prostatic tissue, referred to above, has been suggested and practised with success; and cases have been recorded in which practically the whole gland has been enucleated with complete relief. Removal of the testicles has been followed by great diminution in the size of an enlarged prostate, and has been accordingly recommended in very troublesome cases. Division of the vasa deferentia in the groin has also been practised, and apparently sometimes with success, though often no reduction of the size of the prostate has occurred in consequence.

2. **Prostate, Congestion of.**—Congestion is a condition which follows on chronic hypertrophy, and is commonly known as 'an attack of the prostate.'

SYMPTOMS.—An old man, probably but not always suffering from the symptoms already described, is suddenly seized—as the result of some indiscretion in diet, an exposure to cold, or some other apparently trivial cause—with complete retention, accompanied by bloody urine, possibly a raised temperature and quick pulse, and considerable

local uneasiness. If the case do not improve, and especially if the urine be allowed to decompose, the tongue becomes dry and brown, the pulse more rapid and weak, and the patient passes into a low typhoid condition, which is not unlikely to end fatally.

TREATMENT.—The patient must be put to bed and kept in the recumbent posture till the attack has passed off. The treatment is in large measure surgical, consisting in the proper passing of catheters. Scarcely less important are the careful regulation of the bowels and the administration of a diet sufficiently light, and yet not too lowering, together with, in most cases, a certain amount of stimulant, for it must be remembered that the patient is probably weak, and that death from asthenia is much to be dreaded. The writer would again urgently insist on the importance of preventing decomposition of the urine, which is the most fertile source of death in such cases; he can affirm from experience that this end may be attained by the thoughtful employment of antiseptic treatment, even in those cases in which it becomes necessary to keep the bladder empty by tying a catheter into the urethra.

3. Prostate, Chronic Inflammation of.—

SYNON. : Chronic Prostatitis.—This is not an uncommon affection among young and middle-aged men, depending most frequently on a prolonged gonorrhœa, in which the prostatic part of the urethra has been involved.

SYMPTOMS.—The symptoms of this disease resemble rather closely those depending upon stone in the bladder, namely, more or less frequent micturition, with a feeling of heat and weight in the perinæum, and pain, not usually severe, along the penis, extending to the tip; there is also at times a little blood passed at the end of micturition; and all the symptoms are aggravated by exercise. Generally there are frequent nocturnal emissions. The urine is cloudy, and on standing yields a muco-purulent deposit containing small white filiform shreds. A rectal examination shows that the prostate is enlarged, sometimes very slightly, and seldom to any great extent; it is always tender, but the tenderness is not, as a rule, great. The diagnosis can scarcely be made without passing the sound.

TREATMENT.—The treatment consists in rest, the administration of laxative medicines, and the application of blisters or some other form of counter-irritation to the perinæum; alcoholic stimulants are to be avoided; and the urine should be rendered bland by alkalis and diluents, as in cases of urethritis.

4. Acute Inflammation of the Prostate.—

SYNON. : Acute Prostatitis.—Acute prostatitis may arise as the result of a gonorrhœa, or of cystitis; from the irritation produced by calculi or other mechanical causes; perhaps sometimes idiopathically, or from exposure to cold or wet; and from undue sexual excitement, or the too free use of alcohol if gonorrhœa be present. It may occur in men of any age, and is accompanied by symptoms such as those depending on chronic inflammation, but much more intense; the frequency of micturition and pain during the act causing sometimes almost unbearable agony, and the dysuria amounting in some cases to complete retention, while the tenderness of the gland is very great, a condition which makes an action of the bowels very

painful. Such cases may terminate by becoming chronic; they may undergo complete resolution; or suppuration may occur. In any case there will probably be some elevation of temperature, and in the event of the formation of abscess there may be great and sudden rises and falls, accompanied by rigors and sweatings, with a dry, brown tongue, forcibly suggesting pyæmia. Rectal examination reveals a large, hard, and excessively tender prostate. The enlargement may be symmetrical or unilateral. If an abscess have approached the surface, its position will be indicated by a soft boggy sensation. Prostatic abscess may burst into the rectum, bladder, or perinæum.

TREATMENT.—The treatment of acute prostatitis consists in rest, and carefully regulated diet; diluent and alkaline medicines; purgatives; local blood-letting from the perinæum, by leeches or otherwise (some French surgeons have recommended the application of leeches to the interior of the rectum); with hot fomentations, and morphine suppositories. If an abscess forms it may be opened through the rectum, but it is better to incise it through the perinæum, as this plan is most likely to prevent the formation of that most troublesome and almost incurable condition, a recto-vesical fistula.

Abscesses sometimes form *around* the prostate (periprostatic). They are not so likely to involve the danger of the formation of a recto-vesical fistula; and they should be treated by early incision.

5. Prostate, Tuberculosis of.—This, though not a very common affection of the prostate, occurs perhaps more frequently than is generally supposed, and is of great interest, not only on account of its special features, but because it is usually a part only of a more or less general affection of the genito-urinary tract. Thus in cases where the epididymes are hard and swollen and the cords knotty from tubercular deposit, the finger introduced into the rectum will probably detect a hard nodule in one or both of the vasa efferentia. This, if seen *post mortem*, is found to consist of a tubercular or cheesy mass, and if the condition have advanced farther, the prostate itself may have become involved: there may be either separate nodules of tubercular deposit in a more or less advanced state of cheesy or, more rarely, calcareous degeneration; or the whole gland may have become hollowed out into an irregular cavity, filled in part with cheesy material, and discharging pus.

SYMPTOMS.—This disease may begin in childhood, or in adult life. Its symptoms are most obscure. At first there are probably none at all; but as the disease advances, there will arise those of tumour of the prostate, together, perhaps, with those of abscess; that is, there will be occasionally blood, and generally pus, in the urine; frequency and pain in micturition; tenderness and swelling in the rectum, and so forth. Abscess from this cause has been known to burst into the peritoneum.

TREATMENT.—The treatment can only be palliative, and must be directed to the relief of the symptoms as they arise; but at the best it is unsatisfactory. Occasionally it may be possible to open a tubercular abscess through the perinæum, but it is doubtful how far such a procedure is to the advantage of the patient.

6. Prostatic Calculi.—These are small bodies, generally multiple, formed in the glands of the prostate, usually late in life, but occasionally in

comparatively young men. They probably begin as a deposit of animal matter; but later they are made up principally of phosphate, and partly of carbonate, of lime. They may produce no symptoms at all, or they may project into the urethra, and give rise to great irritation at the neck of the bladder, and the symptoms of vesical calculus; such will also be present if, as sometimes happens, they convert the whole gland into a single cavity, in which the calculi lie side by side. In this case they will be felt through the rectum, rubbing against one another; and indeed prostatic calculi, unless they be very small, are, as a rule, to be felt in this situation. See CONCRETIONS, p. 334.

Vesical calculi of considerable size may become encysted in the prostate; and, on the other hand, prostatic calculi may find their way into the bladder. Prostatic calculi may give rise to abscess.

If any treatment be required, it is purely surgical, and must consist in the removal of the stones by forceps, a lithotrite, or a perineal incision.

7. Prostate, Phleboliths of.—The pathologist very often meets with phleboliths in the veins surrounding the prostate, the result no doubt of old phlebitis.

8. Prostate, Tumours of.—The so-called *fibrous tumours* of the prostate are in all probability simply local hypertrophies, and are composed principally of plain muscular tissue. *Cystic disease* is described as a pathological rarity, the gland being occupied by numerous cysts, containing serous or mucous fluid. *Melanosis* of the gland has also been observed. *Cancer* of the prostate occurs not very infrequently, and is usually soft, though it is sometimes hard enough to be worthy of the name of scirrhus. The writer would speak with great caution of malignant tumours of the prostate; such as he has himself examined have been cancers, with a very irregular arrangement of both stroma and epithelial cells.

Tumours of the prostate may be at present considered as beyond the reach of surgical interference, though suggestions for their removal have been gravely made in Germany.

9. Prostate, Atrophy and Absence of.—Atrophy of the prostate is said to occur as the result of pressure, sometimes from an unascertained cause, or from simple senile decay. Congenital absence of the prostate has also in rare cases been observed, but is of little clinical interest.

R. J. GODLEE.

PROSTRATION (*pro*, forward; and *sterno*, I stretch).—In medical science this word is used to express a condition of system in which the bodily energies as a whole, or the more active of them, have so completely succumbed to the effects of injury, disease, or powerful emotional influences, that they cannot be made to respond to ordinary stimuli. See COLLAPSE; DEBILITY; EXHAUSTION; SHOCK; and SYNCOPE.

PROTAGON.—See LECITHIN.

PROXIMATE CAUSES (*proximus*, nearest).—A synonym for the immediate or exciting causes of disease.

PRURIGO (*prurigo*, the itch).

DEFINITION.—Prurigo is a severe and chronic disease of the skin, characterised by intense

pruritus, and the formation of large, pale, scattered papules.

HISTORY AND SYMPTOMS.—Prurigo is comparatively rare, and until lately was overlooked as a distinct disease by English writers, who usually described cases of it as those of congenital eczema. This is explained by the fact that the disease is often masked by a great deal of superadded eczematous eruption, which is much aggravated by the scratching of the patient. 'In every case,' says Hebra, 'the earliest appearance is that of sub-epidermic papules as big as hemp-seeds, and recognised rather by touch than by sight, since they rise but little above the level of the skin, and do not differ from it at all in colour.' The development of these papules is attended by intense itching, and consequently the tops of the more prominent ones are soon scratched off, and a little drop of blood escapes, forming a small dark crust at the summit of the papule; this gives to the disease one of its characteristic appearances. When the affection has lasted for a considerable time, we notice that the skin becomes dark from increased pigmentation, and at the same time thicker and harder than normal, so that it is difficult to pinch it up between the finger and thumb; the parts affected become more or less eczematous, so that the eruption is somewhat masked; and the lymphatic glands in the groin become enlarged.

The regions of the body most commonly affected are the trunk, the buttocks, and the extensor surfaces of the limbs, especially the forearms and the legs below the knees. The scalp, the armpits, the flexor sides of the wrists and elbows, the palms and soles, groins, and hams are generally unaffected, even in severe cases. The lines and furrows of the skin become more plainly marked than normal on the parts attacked; this is especially noticeable on the backs of the wrists and on the forehead, which gives the patient a peculiar expression when the face happens to be a seat of the disease. The skin assumes a rough and brawny texture, which is more easily detected by touch than by sight.

Prurigo, though not strictly congenital, appears first at a very early age. The early form of the eruption is very often like that of lichen urticatus; at other times it closely resembles a papular eczema. As, however, age advances, the distinctive characters of the disease become more marked. The malady is generally worse in the winter than in the summer, and in a severe form is almost incurable.

In addition to the above-described prurigo of the young, we also meet with a spurious form of the malady which first shows itself in old age, and is commonly known as *prurigo senilis*. The eruption in this case is greatly aggravated, if not entirely produced, by the scratching of the patient.

DIAGNOSIS.—The word 'prurigo' is sometimes colloquially misapplied when simple pruritus is meant; thus we often hear the expression 'prurigo senilis' used when no eruption is present. These cases are generally either examples of chronic urticaria or of senile changes in the cutaneous nerves, leading to excessive and persistent itching. For the reasons already indicated, prurigo is more often mistaken for severe congenital eczema than for any other disease. Careful examination, however, of those parts of the body which have not been much scratched, together with the distribution of the eruption, will generally lead to a correct diagnosis. The malady may also be mistaken for scabies or

phthiriasis, but in both these diseases the parasite can be detected, and in scabies the distribution of the eruption and the history of the case are quite different from those of prurigo.

TREATMENT.—As has already been stated, prurigo, in a severe form, is incurable, but rest in bed for a few weeks always has a markedly beneficial effect. The itching may be greatly relieved by warm borax- and soda-baths, followed by the inunction of carbolised oil, or some similar application. Sometimes a very weak sulphur ointment is useful in relieving the itching; also a weak ichthyol ointment. Internal remedies do not appear to be of much value. It is necessary to treat the eczema which is so frequently associated with the disease. See ECZEMA.

ROBERT LIVEING.

PRURITUS (*prurio*, I itch).—A peculiar disturbance of sensation arising in the skin and in certain mucous membranes characterised by itching and giving rise to a desire, which may become uncontrollable, to stimulate the affected surface by scratching.

Pruritus may be produced by certain well-recognised forms of irritation; thus the stimulation produced by a fly moving on the skin or the application of a feather may cause temporary pruritus. In such cases the stimulation of certain sensory nerve-terminals appears plainly to be the cause; and the areas of the body in which pruritus is most easily excited do not necessarily coincide with those in which tactile sensation is most highly developed. In many forms of disease of the skin with local manifestations, pruritus is a prominent symptom. In some of these an efficient source of irritation of the sensory nerves is obviously present. Thus, in the disease especially named the 'itch,' the presence of the *Acarus scabiei*, its products, and the resulting inflammatory exudation furnish an adequate cause. The same may be affirmed of pediculosis. In many other diseased conditions such as urticaria, the lichenoid eruption remaining as the result of urticaria, and lichen planus, the exudation and the material concerned in its production may bring about the required stimulation. In other forms of disease of the skin, morbid changes, widely diffused, may produce a similar result, as in cases of general eczema and pityriasis rubra. It is noteworthy that diseases producing clinically similar lesions may not necessarily cause the same degree of pruritus, while in some cases the symptom of pruritus may be entirely absent. Thus, syphilis, producing an outburst of papular lesions, is usually devoid of pruritus, and the same may be said of certain forms of cutaneous tuberculosis and other granulomata. It may be imagined that the infiltration in such cases destroys the nerve-terminals in the cutis and in the epidermis. Pruritus appears to be an occasional result of the changes characteristic of senile degeneration of the skin. In so-called 'pruritus senilis,' the possibility of pediculosis and certain other conditions must always be carefully considered, but there can be little doubt that the degenerative changes, the rupture and disappearance of the elastic-tissue fibres and concomitant changes in the connective-tissue structures of the skin may produce severe pruritus.

The rubbing and scratching which are the instinctive consequences of pruritus produce in their turn secondary changes in the skin of the nature of

lichenification and eczematization, whereby renewed stimulation of the sensory nerves is produced, the vicious circle is completed, and the pruritus, instead of being temporary, becomes permanent. In addition, it appears to be the case that the habit of pruritus, or at any rate of scratching, may be induced. By the concurrence of these causes certain forms of disease with almost specific features originate. It is probable that at any rate some of the cases of prurigo arise in this way; and also some of the forms of pruritus genitalium, ani, scroti, vulvæ, &c. In this group of diseases the secondary changes are so severe, the cutis becomes so altered in structure as the result of œdema, vascular engorgement and cellular infiltration, the epithelium is so much destroyed or atrophied, that the disease becomes permanent and incurable.

Pruritus of varying degree and often general in distribution, is well known to originate as the consequence of disease of internal viscera. Thus the pruritus associated with disturbances of the liver, causing chœmia and jaundice, is familiar. It is not an uncommon feature in disease of the kidney, and is possibly most severe and trying to the patient in certain of the forms of glycosuria. The last mentioned disease is, unfortunately, specially liable to be accompanied by pruritus genitalium. Certain diseases of the nervous system are also accompanied by pruritus, when mental symptoms are present, especially in conditions of mental depression. The pruritus is occasionally of special type; it may be specially localised, and the condition known as 'formication' (p. 557) may be a feature. Pruritus of various types may occasionally be produced as the consequence of the taking of certain drugs, such as opium. Changes in temperature have a distinct influence on pruritus: occasionally cold has the effect of moderating the sensation, at other times the local application of heat soothes an attack. As a rule pruritus becomes worse when the vessels of the skin are dilated, as soon after going to bed. The changes of temperature in summer and winter have a peculiar effect in certain persons, producing what is known as *pruritus æstivalis*, and *pruritus hiemalis*, forms of the affection with very peculiar features.

TREATMENT.—The treatment consists, in the first instance, of removing all appreciable causes of irritation of the skin. The cure of scabies or pediculosis, if present, must be undertaken, and it should be remarked that many obscure cases of pruritus depend upon unrecognised scabies or pediculosis vestimentorum, pubis, &c. Equally clearly the treatment of such conditions as urticaria, eczema, lichen planus, when present, underlies the cure of pruritus. A disease which has much to do with the causation of the localised varieties of pruritus, such as the pruritus genitalium, is seborrhœa and the dermatitis associated with it. This condition should never be neglected, and frequently antiseborrhœic remedies will be the most effective way of relieving pruritus. As glycosuria is the most common of the general conditions producing pruritus, a careful examination of the urine for sugar should always be undertaken, and if found, treatment on antidiabetic lines is often very successful. Appropriate treatment must also be adopted in cases where the pruritus is associated with hepatic disturbances, dyspepsia, or Bright's disease. Of local remedies in cases of general pruritus, by far the most useful are simple or medicated baths. Warm baths, bran-

baths, and alkaline baths, are all of great service, and in other cases the bath may be medicated by the addition of the liquor picis carbonis, the oleum betule albae, or other antipruritic volatile oils or oleo-resins. A soap containing balsam of peru, camphor, and sulphur (of each 5 per cent.) is often of great service, used with the bath. After the bath the skin should be dried very gently, and the surface must not be allowed to cool rapidly or to feel chilled. The use of a simple dusting powder is agreeable, and the patient should rest as long as is possible. The application of lotions externally is very useful in the treatment of pruritus. Of the antipruritic remedies so used, carbolic acid is one of the best, and may be applied in the strength of 1 in 40 or 1 in 80 with the addition of glycerine (10 per cent. to 50 per cent.); perchloride of mercury (1 : 1,000, or 1 : 2,000), and the liquor picis carbonis are all of service. Such remedies also as menthol, thymol, camphor and chloral hydrate (equal parts rubbed together till liquefied) are of much service, and may be applied with care, or added to starch-powder in various dilutions and dusted on the surface. In cases of *pruritus genitalium*, the application of strong solutions of subacetate of lead, of nitrate of silver (1 per cent. to 4 per cent.) in water, and the compound tincture of benzoin have all been found of service. Ointments containing mercury, sulphur or carbolic acid are frequently of great service applied to the parts in pruritus of the perineum. The local application of water as hot as it can be borne to the scrotum or vulva frequently gives temporary relief. See ECZEMA.

Internal remedies in the treatment of pruritus should be avoided if possible, or used with caution, so as to avoid establishing a habit of taking drugs and the well-known evil results. The drugs found to be of most service are Cannabis Indica, opium and its alkaloids, and cocaine. Good effects have also been obtained by the use of nerve-depressants such as phenazone and acetanilide.

JAMES GALLOWAY.

PRUSSIC ACID, Poisoning by.—SYNON. : Fr. *Empoisonnement par l'Acide Cyanhydrique*; Ger. *Cyanwasserstoffsäurevergiftung*.

Prussic or hydrocyanic acid is one of the best known and most deadly of poisons. In the anhydrous condition it is stated to kill with almost lightning-like rapidity. Prussic acid is met with in commerce only in a diluted state. In this country two strengths of prussic acid are usual, the Pharmacopoeial acid containing 2 per cent., and the so-called Scheele's acid containing about 4 per cent., of anhydrous prussic acid in aqueous solution. The soluble cyanides, more especially cyanide of potassium, largely used by photographers and by electroplaters, are common articles of commerce, and produce the same deadly results as the acid itself. The fatal dose of prussic acid is the equivalent of less than one grain of the anhydrous acid.

ANATOMICAL CHARACTERS.—In persons who have died of prussic-acid poisoning, the eyes are glistening; the extremities are blue; the face is pale or livid; and the lips are cyanosed. The blood throughout the body has frequently the peculiar odour of the acid, and is of a dull hue, with a peculiar bluish cast—a glimmering appearance. The stomach is sometimes reddened, but not more than is common after other asphyxial modes of death.

SYMPTOMS.—In fatal doses the symptoms of prussic-acid poisoning set in very speedily; and in consequence of the readiness with which this poison is absorbed from the alimentary canal, and diffused throughout the circulation, the onset of symptoms is reckoned by seconds rather than by minutes. Occasionally the patient may be able to walk into an adjoining room, to compose himself in bed, or perform like actions; but it is rarely that he will have time to dispose of the cup, glass, or bottle in which the poison was contained, before he is taken seriously ill. The symptoms may be divided into three stages. The *first stage* is very brief, and manifests itself by difficult respiration, slow cardiac action, with a tendency of the heart to stop in diastole, while its beats are irregular. There is disturbed cerebration, and an awe-stricken aspect of countenance. This preliminary stage speedily ushers in the *second or convulsive stage*, the onset of which is occasionally signalled by a piercing shriek, though this is less frequently observed in man than in animals. With widely dilated pupils, the patient is suddenly thrown into violent clonic and tonic convulsions. The respiration is marked by shortness of inspiration, and prolonged efforts at expiration. The countenance becomes cyanotic. Vomiting is commonly observed; and the urine, faeces, and even semen in the male are spasmodically evacuated. The patient now sinks down, probably in a state of unconsciousness, and with complete loss of muscular power. The convulsive stage speedily passes into the *third*, or, as it may be termed, *asphyxial stage*, with slow, gasping, stertorous respiration, extreme collapse, loss of pulse, and more or less complete paralysis of motion. The skin is cold, clammy, and cyanosed. Death may be ushered in with irregular spasms. The onset of symptoms being rarely delayed beyond one or two minutes, death may occur within two or three minutes more. Power of volition is rarely continued in fatal cases for more than two minutes after taking the poison. Fifteen minutes is the longest interval which has been known to elapse between the taking of the poison and the commencement of symptoms; and then the patient recovered. Should the patient survive for thirty minutes, good hopes may be entertained of recovery. The longest period which is known to have elapsed between the taking of the poison and death was one hour and a quarter.

DIAGNOSIS.—This is rarely difficult. The *four-drayant* character of the illness, and the usually speedy death of the patient, coupled with the peculiar odour of the acid, and the finding of a cup or glass containing the remnants of the dose, seldom leave any doubt as to the nature of the case. Nitro-benzol poisoning closely simulates prussic-acid poisoning, however, except that the onset of symptoms is generally much later in nitro-benzol poisoning than when prussic acid has been taken. Nevertheless, when crude bitter-almond oil, impure from the presence of prussic acid, has been swallowed, the close similarity between the odour of the oil and that of nitro-benzol may lead to error. Fortunately, the same treatment may be adopted in both cases.

PROGNOSIS.—This in all cases is very doubtful; and no general rules can be laid down.

TREATMENT.—Prompt inhalation of the fumes of ammonia should, if possible, never be neglected. The successive administration of a solution of the

mixed per- and proto-salts of iron, followed by an alkaline carbonate, so as to convert the acid into an inert ferrocyanide, has been recommended on purely chemical grounds. There is, however, seldom or never time to admit of this elaborate treatment. A more practicable mode is to treat the patient with alternate douches of warm (115° F.) and cold water, so as to stimulate the respiratory functions; artificial respiration may also be employed, together with friction of the limbs. An emetic should be administered. A hypodermic injection of twenty minims of a 5 to 10 per cent. solution of sodium thiosulphate ('hypo'), or a few minims of solution of hydrogen peroxide may be employed. Faradic currents of electricity to the cardiac region should not be neglected. Atropine is not, as has been asserted, a true physiological antidote to prussic acid; but, injected subcutaneously, it may be of use as a respiratory stimulant. In spite of all treatment, the patient usually succumbs.

THOMAS STEVENSON.

PSAMMOMA (ψάμμος, sand).—A meningeal fibroma containing calcareous masses arranged in concentric laminæ. See CONCRETIONS.

PSEUDO- (ψευδής, false).—This is used as a prefix to various names of conditions, and signifies that they simulate certain diseases or conditions for which in their real nature they differ essentially; for example, *pseudo-angina*, *pseudo-asthma*, and *pseudo-cystis*.

PSEUDO-BULBAR PARALYSIS.—See LABIO-GLOSSO-LARYNGEAL PARALYSIS.

PSEUDO-CYESIS (ψευδής, false; and κύσις, pregnancy).—A synonym for spurious pregnancy. See PREGNANCY, Diseases and Disorders of.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.—See MYOPATHY.

PSEUDO-TABES.—A term, signifying false tabes or locomotor ataxy, which was formerly given to certain forms of multiple neuritis occurring more especially after poisoning by alcohol, arsenic, or as a sequel of diphtheria; in which the paralytic symptoms are generally slight, but where the muscular sense and other modes of sensibility are more or less involved, with the result of producing an ataxic condition of the limbs. See NEURITIS, MULTIPLE.

PSILOISIS (ψιλός, bare).—SYNON.: *Psilosis Lingue et Mucosæ Intestini*; Popularly known in the East as 'Sprue,' a word used by the Dutch and Lowland Scotch, which has reference to the symptoms in the mouth.

DEFINITION.—A chronic disease of the digestive tract, occurring, so far as is known, only in certain parts of Asia and the West Indies; unattended by fever; the most marked symptoms being diarrhœa, rawness and soreness of the tongue and throat, with a disposition to superficial ulcerations of the buccal mucous membrane, the presence of dyspeptic symptoms of various kinds, the passage of characteristic frothy, yellowish, or greyish-yellow, pul-taceous stools, progressive debility and emaciation; and, in unfavourable cases, terminating in death from atrophy and exhaustion; the disease in such cases usually lasting over several years.

ÆTIOLOGY.—The disease is climatic. Cases occur in Ceylon, the Straits Settlements, Indo-China, Java, Manila, and apparently the whole coast of China, more particularly the south. It is more rarely met with in the West Indies and Japan. The cause is unknown, but the probability of a bacterial origin is considerable. In one case the writer found that a particular rod-shaped bacterium prevailed in the motions in numbers over the other organisms in proportion as the disease was aggravated; and in cultivation-experiments this organism liquefied the gelatine with great rapidity. In two other cases under the writer's care, Wethered found a similar-shaped bacterium present in almost pure cultivations in enormous quantities.

ANATOMICAL CHARACTERS.—In fatal cases the small intestine is thin, shining, and translucent; generally in a condition recognised as atrophic, and lined by a covering of mucoid material which gives to the gut a peculiar velvety feel when handled, as though there had been some thickening of the coats, which, however, does not occur. In one case of the writer's the stomach, duodenum, upper part of jejunum, and large intestine were found practically healthy. The tongue was found to be denuded of its epithelium in certain circumscribed parts. The whole of the œsophagus was extensively diseased, the lining and glandular epithelium of the tube being entirely destroyed, and the surface formed by the bare submucosa. In the lower part of the jejunum there was slight inflammatory exudation around Lieberkühn's crypts, with a little destruction of the follicles at some parts. In the ileum the mucosa was almost entirely destroyed, its place being taken by a soft structureless substance. The submucosa was thickened, and contained an abundance of fibrous tissue of a solid character. The coats of the blood-vessels were thickened—a condition of sclerosis (Wethered).

SYMPTOMS.—The symptom to which the patient's attention is most directed is the occurrence of diarrhœa, which varies, however, very greatly in different cases. Sometimes it begins insidiously, the one irregular loose motion in the morning scarcely attracting attention, and it might be overlooked entirely if it were not for other symptoms associated with it, such as griping and general uneasiness. In other cases the diarrhœa is more marked from the beginning, the patient having several loose motions during the early morning without any further diarrhœa until the following day. There are cases, on the other hand, of which the writer has observed some well-marked examples, in which the disease begins with a sudden acute diarrhœa which lasts unchecked by remedies, within a short time other characteristic symptoms being observed. The extent of diarrhœa frequently bears no proportion to the other symptoms, being sometimes comparatively slight in otherwise well-marked cases. The motions are characteristic. At the outset they may consist of a copious discharge of a pale straw-coloured yellowish fluid, which is passed especially in the early morning, causing considerable weakness. During the day this diarrhœa ceases, the patient feels gradually stronger as the day advances, and towards the afternoon he usually feels quite well. In this way he may neglect his condition entirely for a period of many months, during which time he is gradually but steadily losing strength and weight. Sooner or later, but sometimes at the beginning, the motion is more

characteristic of the disease, consisting of the discharge of large quantities of a pulraceous mass, frequently of a putty or slate colour, sometimes pale yellow, sometimes brown. During periods of aggravation this large pulraceous discharge may contain an admixture of minute thread-like portions of a whitish substance. The motions have always a tendency to be frothy. When they consist of the abundant putty-coloured pulraceous mass, the frothy appearance is seen, the surface being often found covered with air-bubbles of various sizes, as if a process of active fermentation were going on. Stools of this latter quality are characteristic of the fully-developed stage of the disease. In cases that do badly, the motions, during this stage, may vary considerably from time to time, sometimes being of a watery or a dirty brown colour, and at other times of a light yellow, with intervals of the return of the pulraceous stool, which is found to contain, if carefully examined, portions of whatever food the patient is taking passed quite unchanged. Even when allowance is made for the large quantity of watery discharge in the motions, the quantity passed is out of proportion to the amount of nourishment taken, showing habitual deficiency of absorption in the intestine.

Early in the disease, and simultaneously with the disordered condition of the bowels, the patient's mouth usually becomes tender. At first there may be simply intolerance of hot food (as regards temperature), or of hot condiments, or of alcoholic drinks. Pepper, sherry, &c., burn his mouth, the tongue particularly. At the same time the tip of the tongue is felt to be tender when pressed against the teeth, and this tenderness may lead to a slight defect of articulation. In the early stages the tongue may be observed to be covered with a shining white fur, which being defective at certain parts shows patches of a bright red; the tip particularly, as well as the sides, being of a bright rosy red colour, while the under surface is seen to be redder than usual, and frequently with the redness accentuated at certain spots. As the disease progresses the fur gradually disappears, large papillæ being prominent for a time. While this process is going on, there may develop on any part of the tongue, but more usually on and under the tip and at the sides, characteristic superficial ulcers, which, while they destroy the investing epithelium, do not extend into the subjacent tissue. These painful superficial ulcerations may vary in size from a pin's head to a threepenny piece, or may be even larger, the larger ones being usually found in the mucous membrane of the cheek. The gums may become swollen, tender, and prone to bleed. Sometimes one solitary ulcerated patch may exist on the mucous membrane of the cheek or lip. Eventually the fur completely disappears from the tongue, which becomes apparently entirely denuded of epithelium, no papillæ being visible, and the whole surface presenting a dull red colour, more or less traversed by longitudinal and transverse furrows, the portions between these furrows being smooth. In some cases, in the advanced disease the tongue is very red, dry, glazed, and shining, and may or may not present cracks and crevices. There are rare cases in which the affection of the tongue is almost the only symptom present, there being no marked symptoms of diarrhoea, although the motion when examined will be found to be slightly altered in colour or consistence.

The patient in these cases complains bitterly of the irritability of the mouth and soreness of the tongue, but does not otherwise feel especially distressed. The extreme anæmia which sometimes becomes a prominent symptom produces a peculiar appearance in the tongue, the part towards the tip being of a pale tallow-like colour. At this stage the mucous membrane of the cheeks, lips, and palate (where not the seat of superficial ulceration) is of a dull, very pale yellow colour.

In a case of the writer's, the number of red corpuscles was reduced to 2,000,000 per c.m., the proportion of white corpuscles to red being 1 in 160. There was 68 per cent. of hæmoglobin. After a month of milk-diet the red corpuscles had increased to 3,580,000 per c.m., the proportion of white corpuscles to red being 1 in 190, while the hæmoglobin was 65 per cent.

The rawness is not always confined to the tongue. The passage of hot food or drink may be painfully felt in the whole course of the gullet, the tender condition of this tube sometimes rendering the swallowing of solid morsels extremely painful and difficult, and giving rise to prolonged and painful hawking, coughing, and sometimes vomiting. This condition occasionally develops early, and may be out of all proportion to the other symptoms, the patient sometimes requiring to get up five or six times during the night to rinse his mouth with water in order to relieve the dry hot sensation from which he suffers.

At an early stage of the disease dyspeptic symptoms develop, and sometimes give rise to much distress, the patient being troubled with flatulence, heartburn, and sour eructations. He suffers from a capricious appetite, sometimes characterised by a craving for meat. After the disease has lasted some time and when the will is weakened from the progressive debility, frequently he cannot be restrained from indulging in various kinds of highly indigestible foods, which are found in the stools absolutely unchanged, and which increase his sufferings and weakness by exciting considerable diarrhoea.

The mental condition of the patient is noteworthy. He is irritable, frequently depressed, and towards the later stages of the disease often extremely difficult to manage, refusing to acknowledge the gravity of his condition, and insisting on indulgence in foods which he cannot digest.

COURSE AND TERMINATIONS.—The disease is essentially a chronic one, being unattended by fever. The urine is usually free from albumen. The liver gradually shrinks in size, and eventually it becomes very small; but there is no reason to believe that this organ is primarily at fault. Such *post-mortem* evidence as has been obtained negatives the idea that the frequent paleness of the motions is due to any affection of the liver.

The abdomen, except at the beginning and the end of the disease, is usually tumid, bulging in the epigastrium, tympanitic, giving a soft doughy feeling to the touch; the tumidity being more noticeable as the patient progressively emaciates. The disease, which may last for a period of years, is attended by general atrophy and anæmia, the subcutaneous fat gradually disappearing till the patient reaches an extreme stage of emaciation, which, combined with the projecting eyes, pearly conjunctiva, and extreme pallor, gives rise to a peculiar and characteristic appearance. In the later

stages, and while recovery is still possible, there may be considerable oedema of the legs, with a rough harsh skin. In fatal cases the patient usually sinks with great slowness, death being mainly brought about by his being essentially starved to death, the processes of digestion and absorption having been for a considerable time almost entirely annihilated. Shortly before death blood and mucus may be found in the stools, attended by considerable pain in the rectum.

DIAGNOSIS.—This disease might possibly be mistaken for dysentery by the inexperienced, and is apt to be confounded with various forms of diarrhoea which are common in the tropics. In chronic dysentery, the mucus in the stools, the evidence of local irritation in the colon, inclination to strain, and the previous history of the attack, together with the absence of the characteristic symptoms of psilosis, ought to be sufficient to prevent any confusion between the two diseases. At the beginning of the malady it might be difficult to distinguish psilosis from other forms of diarrhoea, but after a time the condition of the tongue, mouth, and gullet, and the characteristic pulaceous stools which have been described, ought to be sufficient to establish the diagnosis. In simple diarrhoea of the tropics there are not present the peculiar local changes in the epithelium of the tongue and mucous membrane of the mouth, nor are the symptoms in the gullet present which are characteristic of psilosis; and those who are experienced in both affections should have little difficulty in distinguishing the frothy pulaceous masses of the latter disease. The peculiar changes in the mouth and throat are typical and distinctive. In the last stages of psilosis and of prolonged tropical diarrhoea the symptoms of exhaustion and emaciation are much the same; and in chronic exhausting diarrhoea the tongue may become bare and raw, with the formation of numerous aphthous spots in the mouth, which still further tends to obliterate the lines of distinction between the diseases in the fatal stages. In such cases it is necessary to take into account the previous history of the case.

PROGNOSIS.—Psilosis is always a grave disease, its gravity increasing in proportion to the age of the patient. In the earlier stages it is very difficult to get the sufferer to realise the serious nature of his malady. Patients under forty, who are willing to undergo the self-denial of suitable treatment, will probably recover, particularly if they remove to a temperate climate; but relapses are apt to occur for one or two years after recovery is apparently complete, particularly if the patient returns to a country where the disease is endemic. In patients who are middle-aged, and particularly in those who are over fifty, the gravity of the disease is much increased; but even at this age, if the constitution has been naturally strong, permanent recovery may take place even in cases in which the disease has lasted a considerable period. In all cases, even the slightest, it is advisable to point out to the patient and his friends that the seriousness of the case is out of proportion to the mere amount of diarrhoea present.

TREATMENT.—The treatment which has been found on empirical grounds to answer best is in accordance with what has been recently ascertained in connection with the morbid condition of the small intestine. The object is to give the weakened bowel (ileum) as complete rest as possible. The patient should be instructed to keep his bed for

several weeks, after which period the probable improvement of the symptoms usually permits his spending part of the day on the sofa or in an easy chair; but experience has shown that there is great advantage in maintaining the recumbent posture for some time. The abdomen should be covered with a thin layer of cotton wool kept in place by a flannel bandage, and the patient at once put on an exclusive milk-diet. The milk should be sipped slowly, must be pure and fresh, and should be slightly warmed. It is given in half-pint doses at regular intervals from 7 A.M. until 10 P.M. A small person will do very well with four pints daily, which is about the minimum quantity; but the usual quantity to begin with is five or five and a half pints. Should the patient be unable to take half a pint at once, a smaller amount should be given, and the intervals between each shortened, so that he may still take the desired quantity each day. The milk may be boiled, if wished, and some patients prefer it aerated; the aeration being effected in a gasogene or by 'sparklets,' and not by dilution with aerated waters. When the patient is unable to take a sufficient quantity of milk to increase his strength it is useful to have the amount of fluid reduced by evaporation. This is done by warming the milk in a 'Bain Marie' until it is reduced to half its original bulk. It must not be allowed to boil, and requires to be constantly stirred, both during the process of evaporation and until it has cooled. In favourable cases the improvement begins to be evident within a few days; the soreness disappears from the mouth; the motions become gradually more consistent, until eventually they become solid, the colour remaining for some time white and creamy-looking. It has been shown by the writer that this whiteness of the motions, which may continue over long periods during convalescence, is not due to absence of bile. When the motions become solid, the quantity of milk is gradually increased until, in an adult, eight pints or even more are given. After a few weeks there is usually considerable constipation, giving rise to pain and soreness, and frequently necessitating the use of an enema. At this period rhagades are apt to form at the anus, and may require to be touched with nitrate of silver. After the bowels have remained regular for five or six weeks, gradual additions are made to the diet; but here we find much difference in the idiosyncrasies of individual patients. A small quantity of bread is first allowed, beginning with two ounces daily, and if that produces no symptoms the amount is gradually increased to eight or nine ounces per day. If this is well borne the patient may be tried cautiously with boiled white fish—whiting, haddock, or sole. The next change in diet should be a few ounces of chicken or partridge, and after some days this may be alternated with boiled mutton, after which time cauliflower may be tried, and light farinaceous puddings in place of part of the bread. About the same time that fish is given fruit may be tried, bananas or grapes, the skins and seeds being rejected. The writer has had cases in which large quantities of strawberries could be taken with milk, when other additions to the milk disagreed, and in consequence more milk could be taken and rapid improvement of the patient took place. In connection with all added articles of food, it is important to bear in mind that any one of them may in some patients cause a relapse of the symptoms. When this occurs the

patient should be at once put back on milk-diet, and kept on it for four or five days, when the additions to diet may be again begun, this time avoiding the particular article of food that had been found to disagree. The writer has known relapses produced by eggs (which are usually well borne) after recovery had begun. Although fish generally agrees well, he has known a relapse brought on by boiled whiting, and so on with almost every article of food he has tried, with the exception of milk. Unfortunately, there are cases, although very rare, in which milk cannot be borne, and indeed seems to aggravate the symptoms, in which case raw-beef juice may be tried, and to obtain it in sufficient quantity the juice of twelve to twenty pounds of beef daily is required. In order to express the juice it is necessary to use one of the machines specially made for the purpose.

In some cases there is such an intolerance of starchy foods that after the motions have been solid on milk-diet for five or six weeks, an ounce of bread per day will reproduce the diarrhoea. In these cases very finely minced rump-steak is sometimes well borne, taken raw or very lightly cooked. It is best to begin it without any other addition, the diet then consisting of milk and two ounces of the minced beef daily. If this agrees, two ounces may be given twice daily, and after a time it may be tried as a sandwich between very thin slices of bread, and by this time it will generally be found that the bread does not disagree, the patient having become much stronger in the interval.

In cases in which recovery from the disease has been incomplete, and a tendency to frequent relapses is left, a meat-diet on the principle of the 'Salisbury' treatment often answers. The milk-diet is abandoned; and finely minced, very lightly cooked meat is given three times a day, combined with a small quantity of farinaceous food. If this treatment is well borne, as it often is, the tendency to diarrhoea disappears, the patient becomes stronger, and after a time—sometimes a comparatively short time—his diet may be varied without inconvenience.

On account of the tendency to relapse, the patients require to be encouraged and reassured, as in well-managed cases the relapses usually do not last more than four or five days. When the patient has been able to digest solid food, and there is no return of the distinctive symptoms, he should be allowed to go out; but he must for a long time avoid fatigue. A chill will at any time, for months after he has apparently recovered, produce a relapse. Drugs have no effect on the disease, although a slight temporary improvement may sometimes be seen after the exhibition of a small dose of rhubarb. For a severe diarrhoea with pain, which occurs occasionally, particularly in the relapses, a moderate dose of laudanum is useful, but opium has no curative effect on the malady. The patient should not return to the country where the disease occurs if he can avoid doing so, as experience shows that relapses are common in these circumstances. Where the return is unavoidable, if possible a whole year should be allowed to elapse after complete recovery, before leaving again for the East. Patients who remain at home, and who have made a good recovery, usually remain well; and many patients who have been under the writer's observation, and who at one time suffered severely, now enjoy perfect health, and follow their ordinary occupations.

G. THIN.

PSOAS ABSCESS.—SYNON.: Fr. *Abscès du Psoas*; Ger. *Psoasabscess*.

DEFINITION.—An abscess within the sheath of the psoas muscle, almost always of spinal origin.

ÆTIOLOGY.—Caries of the lower dorsal or lumbar spine is the cause of psoas abscess, and these being the regions of the spine most commonly diseased, this is the commonest form of spinal abscess. The caries is almost always tubercular. Syphilis and actinomycosis are rare causes. Rarely, too, an abscess starting from the hip-joint makes its way into the sheath of the psoas, and travels upwards in it, even to a lumbar transverse process, as the writer has seen in a case of pyæmic suppuration in both hip-joints. It is said that a suppurative inflammation may arise within the psoas sheath, unconnected with disease of bone or joint, but supposed to be consequent upon strain or exposure to cold.

ANATOMICAL CHARACTERS.—The disease may begin on the surface of one or two vertebræ; a tubercular periostitis results and generally spreads over several bodies (*anterior caries, caries without curvature*), a form occurring chiefly in adults, specially liable to end in abscess, and formerly regarded as exceptionally fatal. An abscess from such a source lifts up the anterior common ligament, and all structures in front of it, such as vessels, pillars of the diaphragm, and so forth; these become matted together in dense fibrous tissue, and form the anterior wall of the abscess cavity. More commonly the disease appears in the body of one or two vertebræ close to an intervertebral disc, because, as is supposed, it is here, at the junction of bone and cartilage, that the slight injuries which so often precede and predispose to tubercular disease take effect. The result of the settlement of bacilli is the formation round about them of an inflammatory infiltration, which may be small and circumscribed, or, more rarely, diffused throughout the body of the vertebra as a general osteitis. In either case, as a rule, it tends to spread, making its way towards the surface, usually the antero-lateral: a periostitis is now excited. As the infiltration spreads it caseates and dies in its older portions. The infiltration ordinarily eats away the cancelli, and, under the influence of pressure, the part above the eroded area sinks down on to that below it, and a 'Pott's boss,' or projection of the spines, is thus produced. With a less acute infiltration sclerosis of bone results, and a subsequent exacerbation of the inflammation or caseation of the products will lead to the formation of considerable sequestra of dense bone. Smaller sequestra are frequent, and result from caseation of an infiltration which still contains particles of uneroded bone (*caries necrotica*). Having reached the surface and excited a periostitis, the disease may spread to neighbouring vertebræ, skipping over the discs, and destroying them by eating away the bone on each side of them rather than by direct attack; and as body after body is eroded the curvature increases. The disease may be arrested, and often is, under suitable treatment. But in many cases, especially among the poorer classes, and among adults in all classes, the caseous material 'softens,' i.e. becomes mixed up with a quantity of fluid in which the fatty cells and their *débris* float. This mixture constitutes the 'pus' of chronic abscesses; it is thin and watery, quite opaque, pale yellow-white, often contains visible and sometimes large masses of fattily degene-

rated cells, and bony and calcareous particles may be numerous when the abscess springs from bone. This fluid, as it increases, presses onwards in the direction of least resistance. Starting from the lower dorsal spine, it may pass through the diaphragm with the aorta, but much more commonly it extends laterally beneath the pleura so as to reach the highest digitation of the psoas rising from the lower edge of the twelfth dorsal vertebra. With this slip it passes beneath the internal arched ligament of the diaphragm, and thus enters the sheath of the psoas. Pus from lumbar vertebræ may enter the sheath at once, if the disease reaches the surface at a point from which one of the slips of the psoas arises; otherwise the subperiosteal abscess travels on the front of the spine and extends laterally until, perhaps, it raises one of these slips of origin from the bone and thus enters the sheath. The aperture of communication between the psoas sheath and the cavity in front of the diseased bone is often very small; there may be more than one. Having thus entered the sheath of the muscle, the pus by its constant pressure and irritation causes atrophy of the muscle-cells, and inflammation, which leads to thickening of the connective tissue into a capsule for the pus; this capsule is lined with a thick layer of very loosely adherent granulation-tissue, which, no doubt, adds its quota to the fluid and solid contents of the cavity. It is tubercular from the first, and cheesy points in it are numerous. Crossing the interior of the cavity are more or fewer bands, some containing vessels, others nerves of the lumbar plexus; pain or numbness, referred to their distribution, and wasting of special muscles are, therefore, not to be wondered at. Pressing on, the pus may so distend the psoas that it will reach out to the anterior iliac spine; but, usually, fluid as far out as this lies beneath the iliac fascia, with which the psoas fascia is continuous on its outer side. An abscess filling the iliac fossa and not passing beneath Poupart's ligament is called an 'iliac abscess'; it is a stage of a psoas abscess. But from the iliac fossa it is said that pus may pass out over the crest through Petit's triangle, which involves its bursting through the strong iliac fascia. This certainly takes place when the pus makes a way to the surface through the muscles of the abdominal wall just internal to the anterior iliac spine. Having or not having filled the iliac fossa, the pus usually passes down behind Poupart's ligament, lying external to the femoral artery; then, taking the profunda for its guide, it runs inwards behind the main vessels, over the pectineus and adductor brevis, between the adductors longus and magnus, to the inner side of the thigh. Rarely do we see an abscess of such extent nowadays; but formerly they occasionally ran down even to the internal malleolus. It is common for diverticula to pass from the cavity along branches of the profunda, especially along the internal circumflex, which conducts the pus to the face of the quadratus, and the swelling indicating it presents on the buttock beneath the lower fibres of the gluteus maximus. Besides this, there may be no other perceptible swelling in the thigh; it must not be mistaken for an abscess pointing through the great sacro-sciatic notch.

Pus from lumbar vertebræ may pass beneath an arch of origin of the psoas over the side of a vertebra; then, taking the lumbar artery and its posterior division as its guide, the pus runs back

internal to the inter-transverse ligament, and issues beneath the latissimus, having escaped from between the erector and quadratus. This is the 'lumbar abscess' of spinal disease. Rarely, pus from lumbar caries may run down along the great vessels into the pelvis. A pelvic spinal abscess is almost always of sacral origin.

SYMPTOMS.—At first the symptoms are those of spinal disease. If an abscess is forming quickly, and much tension within the muscle-sheath is developed, full extension of the hip causes pain, and persistent flexion is consequently maintained; but in less acute cases there is often no history of pain, lameness, or flexion. There may be pain along the line of one or more branches of the lumbar plexus. Even the ordinary symptoms of spinal disease are not very rarely absent, and a swelling in the groin is the first thing noticed. It often seems to have appeared suddenly; lies external, and perhaps also beneath and internal to the vessels; is smooth, rounded, more or less tense, and more or less fluctuant; is covered by normal skin; is not tender; has a distinct impulse on coughing; and may disappear more or less completely in the recumbent position or on pressure. Fluctuation is obtainable between a fulness above Poupart's ligament and the swelling below it. Though the former may be slight, it is generally considerable, and may actually distend the abdomen up to the ribs. Sooner or later, either above or below Poupart's ligament, the abscess points; the skin then reddens, thins, and finally gives way. This may take place with some acuteness.

DIAGNOSIS.—There is no difficulty when the abscess is typical, and spinal disease marked. But a swelling like an iliac or psoas abscess may be present without obvious spinal disease. The probability is that it is of spinal origin, and searching inquiry must be made for occasional pain in the back or in the stomach; for disinclination for active exercise (especially jumping), and early fatigue; for any tendency to use the arms to take off part of the weight of the body from the legs; and for slowness or difficulty in performing such movements as rising from the stooping or sitting posture. The spine should be carefully examined for any slight prominence; and, if any is found, percussion and heat must be employed here and may strengthen the suspicion excited.

The surgeon should cause the patient—stripped to the hips—to execute before him all the movements of the spine (bending forwards, backwards, and to each side, and rotation towards each side), and should note whether they are completely, sharply, and painlessly performed, and if all the spines seem to separate, or whether certain ones retain their distances, indicating that a length of spine is held fixed. But it is not always possible to establish a certain diagnosis, as cases in the writer's experience show.

Failing to find evidence of spinal disease, it becomes necessary to seek for other possible causes of psoas abscess; the hip-joint must be proved healthy, the renal region must be explored, the urinary history gone into, and the urine examined; the possibility of empyema pointing in the groin must be excluded; in case of iliac abscess, abscess from disease of the ilium or appendix vermiformis, abscesses following on pelvic peritonitis and parametritis, soft rapidly growing tumours and serous and hydatid cysts of the iliac fossa must be thought of.

PROGNOSIS.—Under modern antiseptic treatment the prognosis is relatively good. Its gravity increases, so far as the abscess is concerned, with the size and number of branches of the abscess, and with the age of the patient. The gravity of the case is greatly increased if the abscess cavity becomes septic; children are much more likely than adults to survive this complication.

TREATMENT.—A few cures may be ascribed to simple rest alone or with repeated aspiration. Usually these fail and aspiration often leads to a sinus probably by infection of the skin as the needle is withdrawn.

Here, as elsewhere, in dealing with tubercular disease, the object is to remove as much as possible of the tubercular tissue. This is best effected by careful scraping with a sharp spoon to detach the lining granulation-tissue from the whole surface. Incisions must be so placed—in loin, groin, thigh, and buttock—as to allow the spoon to reach every point of the surface. Either during the scraping, by means of a flushing curette, or afterwards by a suitable tube attached to a douche-can, all débris should be washed out of the cavity with warm sterile, normal saline solution. Then each opening should be closed—fascia to fascia, muscle to muscle, and skin to skin—after disinfection of the edges: the use of a small skin-flap to cover the deep wound is also helpful in preventing infection of the wound and formation of a sinus. It is a good plan to mix with the last pint of saline solution run into the cavity half to one drachm of powdered iodoform, which is thus deposited upon the walls. Often the cavity refills: tension should be warded off by aspiration through a fairly thick part of the wall, and this should be repeated as may be necessary. In successful cases, the fluid withdrawn is honey-like, smells of iodoform, and contains few or no caseous flakes and is formed more and more slowly. In failures, fresh curdy fluid is formed or ordinary pus, if pyococci have gained entry. In the former case scraping may be repeated; in the latter free antiseptic drainage is the proper course. Should a sinus form, it must be carefully guarded from sepsis. The spinal disease and the general health must be attended to. The danger of this treatment is slight: its advantages, when successful, very great.

A psoas or iliac abscess should be thus treated as soon as discovered. If not yet presenting in the groin, it should be opened in the loin through an incision passing outside the erector and through the quadratus on to the psoas.

STANLEY BOYD.

PSORIASIS (ψώρα, scurf).—**SYNON.** : Lepra; Alphas (both obsolete); Fr. *Psoriasis*; Ger. *Schuppenflechte*; *Psoriasis*.

DEFINITION.—A chronic inflammatory disease, occurring chiefly on the extensor aspect of the limbs, and consisting of discoid patches with scaly crusts on a red base.

VARIETIES.—There is really only one kind of psoriasis, but the older writers gave names to the different phases of the disease founded upon (1) the size of the patches, using such qualifying terms as punctata, guttata, nummulata; (2) the extent of the disease—diffusa, universalis; (3) the covering of the patches—rupioides, empyodes; (4) their shape—circinata, gyrata; (5) their duration—in-veterata, &c. Scaly syphilides also are often spoken of as syphilitic psoriasis, but modern derma-

tologists avoid all these artificial and ambiguous varieties, which are of no practical importance and in the case of the syphilides are misnomers.

ÆTIOLOGY.—Psoriasis attacks equally both sexes and all classes. It is rare before three years of age, though cases have occurred in early infancy, while it is very common in childhood and puberty, so that 70 per cent. have their first attack under thirty years of age and only 5 per cent. begin after fifty years, but those previously subject to it may go on having it up to any age. It is hereditary in a large number of cases, but not very strongly, as it is not often that more than one or two in a large family are affected. The general health is often quite good, but depressing influences, whether of mind or body, will often determine an attack in those predisposed to the disease. Gout and other arthritic conditions are predisposing factors in adult life, but only in a small proportion of cases. Spring and winter are the most common seasons for a new outbreak.

ANATOMICAL CHARACTERS.—The true pathology of psoriasis is still unknown, but the theory of a microbe which, perhaps starting in the skin, subsequently gets into the circulation and is distributed symmetrically, is consonant with the clinical facts. Unna's theory that it is part of the seborrhœic process is not accepted. The anatomical changes are those of moderate inflammation in the papillary layer of the corium, producing cell-infiltration and vascular dilatation, chiefly round the hair-follicles and sweat-ducts. The horny layers and those of the rete are enormously and rapidly increased, except over the papillæ, and there is a great tendency to premature cornification and exfoliation. The papillæ are much enlarged by the down-growth of the interpapillary processes. Some pathologists hold that the change in the rete and the premature conversion into horny cells are the primary conditions to which the inflammatory phenomena are secondary.

DESCRIPTION.—No part is absolutely exempt from attack, but the disease generally appears first and in its most typical aspect on the extensor surface of the limbs, especially the elbows and knees, and the parts below; and it is often confined to these regions, the flexor aspect being actually free or only affected in a minor degree. Next in order of frequency are the scalp, trunk, the back more than the front, the face comparatively seldom, but more frequently in women and children than in men, while the palms and soles are rarely attacked. The disease is in the main symmetrical in its distribution.

A well-developed patch, which may be from a half to two or three inches in diameter, is well defined at the border, contrasting sharply with the healthy skin, and consists of a brightly reddened disc more or less concealed by silvery scales adhering into a spongy crust. When this is removed, the under layer being generally firmly attached, bright red, easily bleeding points are brought into view, which are the tops of the enlarged papillæ. The amount of scalliness varies with the acuteness of the process. When slowly formed, the scales are closely adherent to each other and to the plaque, while in acute cases they flake off too quickly to form crusts, and the intensely hyperæmic base is freely exposed. On the back of the hands and the face the scales get washed off. On the scrotum the natural moisture has the same effect, and there is

often fissuring and great irritation. On the palms and soles, patches are seldom seen, but the epidermis is dry, thickened, and cracked, imparting a worm-eaten appearance to the part. In the scalp, the hair is dry, but seldom comes out much, except in acute cases. The nails are, however, often discoloured, pitted, furrowed, and brittle, and separated from their bed, beginning at the distal end at one side. In rare instances the mucous membranes have been slightly involved.

DEVELOPMENT AND COURSE.—Each lesion begins as a papule the size of a pin's head, surmounted almost from the first by a scaly cap; this speedily enlarges to a small disc, which continues to spread peripherally until a patch of two or three inches in diameter may be formed. Larger areas are produced by coalescence of adjoining patches; but, however extensive they may be, there are always some areas of healthy skin. When regression takes place it commences in the centre, and when this is clear a circle is formed, or, if the patches have previously coalesced, a gyrate contour. Occasionally, however, on the trunk gyrate and circinate patches are primarily formed from the disease attacking the hair-follicles only, and following therefore their arrangement; this is the lepra of Willan. As further absorption occurs the circular border is broken up, and ultimately the fragments also disappear, leaving a transitory red stain, or a long-lasting yellowish-brown one when the disease has been treated for some time with arsenic. Itching varies much: it may be absent, or very slight, but as a rule is present in a moderate degree, and is only rarely as intense as in eczema.

COMPLICATIONS AND SEQUELÆ.—Papillary development may occur on the patches, which may be soft and papillomatous or hard and warty. Deep and extensive pigmentation sometimes occurs without arsenic having been given. Scarring, keloid, and epithelioma rarely ensue, the latter developing either on a warty psoriasis, or on the warts left by excessive arsenical treatment.

DIAGNOSIS.—The characteristic features of psoriasis are the position of the patches chiefly on the extensor aspect of the limbs, and especially on the elbows and knees; the borders of the patches being well-defined; the scales being white and adherent into crusts, but without inflammatory exudation. When the crusts are removed, bright red, easily bleeding points are visible. The presence of all these features renders the diagnosis inevitable. Lichen planus, eczema, pityriasis rubra, tinea circinata, and squamous syphilides in some phases, are the diseases most likely to be mistaken for psoriasis.

The patches of lichen planus are roundish, well-defined, and scaly, but they do not choose the special seats of psoriasis; the scales are scanty compared with psoriasis; the colour is violet red instead of bright red. They leave deep pigmentation behind them, and there are almost always some of the characteristic flat papules in the neighbourhood of the patch. Scaly patches of eczema are seldom defined at the borders; the scales are in a single layer or mixed with inflammatory exudation, or there may be a history of discharge; there are no bright red points when the scales are removed; finally eczema is much more common on the flexor than the extensor surface of the limbs, and even on the extensor aspect does not specially choose the elbows and knees.

In pityriasis rubra, confusion could only arise before the disease became truly universal, and when the psoriasis is in an acutely inflammatory condition; then the base is intensely red, and the scales flake off easily and rapidly, so that no scaly crusts are formed. Pityriasis rubra is diffuse, not in patches; the border is less defined; the colour is an intense bright red; the scales are large, thin, and papery, do not conceal the ground-colour, never adhere into crusts nor over their whole surface, and are thrown off almost as rapidly as they are formed. It must be remembered that pityriasis rubra sometimes develops from a pre-existing psoriasis, but the change is usually acute, and there will be a history of previous chronic patches.

In tinea circinata, the small number of non-symmetrical patches coming in any part of the body, the very scanty scales and the at first papular border, ought to lead to microscopic examination of the scales for the fungus, if a positive conclusion cannot be arrived at without it.

Secondary scaly syphilides are rarely acquired before adult age, while psoriasis is a common disease of childhood; on the other hand, psoriasis is rare under three years, and does not therefore clash with congenital syphilides. The scaly syphilide is in small patches, with scanty, dirty-looking scales on a dull red base; does not specially affect the extensor aspect of the limbs; is often associated with other forms of eruption; leaves fawn-coloured pigmentation behind; and is nearly always accompanied by the other symptoms of syphilis. In the circinate scaly syphilide the same distinctions in position, colour, scales, and concomitant symptoms hold good. In the tertiary scaly syphilide the resemblance may be rather close, but position will again assist: the face is often affected; the number of patches is usually small; they are not symmetrically arranged; the edge is more raised, the centre more depressed; ulceration is common; and scarring and pigmentation follow the disappearance of the lesion.

PROGNOSIS.—It is always possible, but often difficult, to remove the lesions of any one attack, but recurrence at some time or other takes place in 90 per cent. of cases, the interval of freedom varying from weeks to years. Sometimes psoriasis disappears spontaneously, but more often continues for years with remissions and exacerbations, which may at any time assume a severe form with widespread distribution.

TREATMENT.—Combined external and internal treatment is the most rapidly efficacious method of removing the eruption of psoriasis, cases of moderate extent requiring from three weeks to three months. The short period assumes that the patient gives himself up to treatment and is in skilled hands. The general health being in a majority of cases undisturbed, specifics find their most fitting opportunity, of which arsenic, thyroid extract and salicin are the most important. The soundest principle, however, is to search carefully for any departure from the highest standard of health, and to endeavour to rectify it, if found, before resorting to the routine treatment with arsenic.

Conditions depressing vitality are the most common: overwork, anxiety, suckling, or any prolonged drain upon the system may be determining factors. Gout and rheumatism take a more subordinate place, but if present require appropriate

treatment; and only when these difficulties have been removed or met should specifics be called in.

Arsenic may be given in either a solid or liquid form; and although practitioners have their fancies for this or that salt, practically arsenious acid for pills, and Fowler's solution for mixtures, meet all requirements. The recently introduced cacodylate of sodium is a dangerous form on account of the large quantity of arsenious acid in it; half a grain three times a day is recommended, but is equal to ninety minims of Fowler's solution. One grain three times a day produced acute poisoning in one case.

The solid form is often the most convenient, as it interferes least with the patient's avocations. The following are useful formulæ: Arsenious acid one grain, liquorice powder \mathfrak{zj} , extract of hop \mathfrak{zj} ; mix thoroughly, and divide into 30 pills. Take one three times a day after meals. The well-known Asiatic pills are stronger, containing nearly $\frac{1}{12}$ of a grain of arsenic in each, and are much used abroad: Arsenious acid 66 grains, powdered black pepper \mathfrak{zix} ; gum arabic and water a sufficiency; mix and divide into 800 pills. Take one three times a day after meals. When the patient is tolerant, and the disease obstinate, the dose may be increased until the limit of his endurance is reached, griping and diarrhoea being obviated by combining opium; of course, the effect on the patient as well as on the disease should be watched. But while the pills will always hold a place on account of their convenience, where practicable, the *Liquor Arsenicalis* is preferable, as it can be freely diluted, and the irritant effect on the stomach is more likely to be avoided. Beginning with 3 to 5 minims, it may be pushed up to 10 or 12 minims or more, three times a day, always after meals.

Improvement is not manifested at once; often the full physiological effects of the drug must be reached before the scales cease to form; then the older ones drop off, the patch begins to clear, first in the centre and then gradually over its whole area. Arsenic is contra-indicated when the eruption is very hyperemic, or is coming out acutely, or when there is acute or chronic irritability of the alimentary canal. It will often make acute cases spread faster, and increase the itching very considerably. Even in suitable cases, however, this increased itching may also be excited at first; but it subsides in a week or two, and the patches begin to clear up.

It must not be supposed that even in apparently suitable cases arsenic is always successful. Unfortunately its failures are only too numerous, and it is a very disappointing drug. Moreover, if continued too long it produces a general pigmentation of the skin and a warty thickening of the palms and soles of a permanent character.

In a limited number of cases the administration of thyroid extract is followed by the removal of the eruption, and in a few cases in a remarkably short time. It should not be given to elderly persons or to those who have weak hearts, nor in cases where the psoriasis is coming out in fresh spots, or when the old patches are spreading. The dose should be a small one to begin with, 5 grains a day. At the end of a week the dose may be increased to 10 grains a day, and at the end of three weeks probably the dose again increased, provided that no symptoms of thyroidism, e.g. headache, giddiness, and rapid pulse, have appeared.

Larger doses are seldom advantageous and may be even dangerous.

Salicin and salicylate of sodium were introduced by the writer for psoriasis, and have the advantage over arsenic or thyroid of being useful even in spreading cases. Salicin is generally preferable to salicylate of sodium. The initial dose of salicin should be 15 grains three times daily, and it may be increased to 20 or even 30 grains. In a few cases it may disturb the digestive function or produce erythema. In most cases the hyperæmia rapidly diminishes, the scales are loosened and fall off, the patch clears in the centre, the scaly ring breaks up, and only small fragments are left which are best removed by local means.

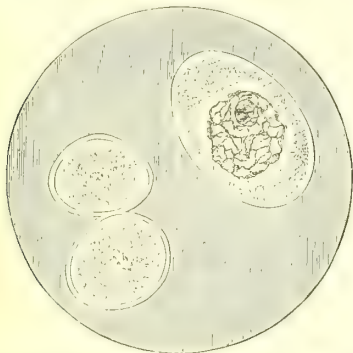
The local treatment consists in removing the scales by alkaline baths or soft soap, and then rubbing in stimulating and antiseptic applications, in the form either of lotions, liniments, or ointments. The local applications may be placed in three divisions, in the grade of their stimulating effect, although that probably is not their sole mode of action, as they are all more or less antiseptic. In the first division may be placed the mercurial preparations, of which the ammonio-chloride, the nitrate, and the yellow oxide are chiefly employed; either may be used as an ointment, in the proportion of \mathfrak{zj} to the \mathfrak{zj} of lard or other base. They should not be used over a very large area, nor continuously, but be rubbed in night and morning. This applies to all stimulating methods of treatment. In the next division come the tarry preparations, naphthol, and thymol. Preparations of tar have long been the classical treatment for psoriasis. They are very numerous, but oil of cade and *oleum rusci pyroligneum* or birch-tar oil are the least objectionable for ointments or liniments, and are used in various strengths from \mathfrak{mxx} to \mathfrak{zjij} to \mathfrak{zj} , or even stronger. For a lotion the alcoholic solution called *liquor carbonis detergens*, from \mathfrak{zss} to \mathfrak{zjij} to \mathfrak{zviij} of water, sponged freely over three times a day, is often very useful. β -Naphthol, resorcin and thymol are generally used as ointments: \mathfrak{zj} to the \mathfrak{zj} is an average strength, the addition of \mathfrak{zj} of prepared chalk facilitating the preparation of the ointment. They have the advantage of being cleanly and free from disagreeable odour. In the third division are the strongest stimulants, such as turpentine, pyrogallic acid, and chrysarobin. Turpentine is used as a liniment from 1 to 4 up to equal parts of it and olive oil. Pyrogallic acid and chrysarobin may be used as ointments, gr. v to \mathfrak{zj} to the \mathfrak{zj} . Pyrogallic acid stains linen, and dangerous absorption may ensue if used over a large surface (p. 615). Chrysarobin also stains both skin, hair, and linen, and may excite an erythema with œdema resembling erysipelas (p. 422); it soon passes off when the drug is stopped. In suitable cases it is the most rapidly efficacious of all local remedies. Moreover, in small quantities such as 1 grain to 5 grains of Ung. Zinci its range of usefulness may be extended even to hyperæmic cases, and remarkably good results obtained with care and judgment. These are only a tithe of the remedies suggested for this obstinate disease, and they may be combined in endless variety and strength. The guide in the selection of a remedy is the amount of hyperæmia present in the patches. The more acute the process, the less stimulation is required; and if very acute, soothing applications, such as

simple olive oil, olive oil combined with calamine, oxide of zinc and lime water, or the glycerin of the subacetate of lead (1 part to 7 of distilled water) may be bandaged on continuously. The patient's circumstances and occupation, as well as the position of the eruption, are also to be taken into account in choosing the remedy and mode of applying it, and considerable experience is requisite to form a correct judgment on all these points.

H. RADCLIFFE CROCKER.

PSOROSPERMOSIS (Greek *ψώρα*, itch).—This term is applied to lesions in the human subject produced by certain forms of Protozoa (*Sporozoa*), allied to the *Coccidium oviforme* which affects the bile-ducts of rabbits. Sporozoa or psorosperms are unicellular organisms which multiply by fission; each of the primary fragments thus produced becomes surrounded by a firm coat and is known as a spore, the whole number of spores being contained in a capsule or cyst. Subsequently each spore may again divide into smaller bodies which ultimately develop into mature individuals. The whole cycle of development probably does not take place in one host.

In man the infection may either be generalised throughout the viscera (rare), or localised in the liver, urinary tract or the skin. In the liver definite tumours are formed which may even be palpable through the abdominal wall. In this situation the parasites give rise to symptoms of pain and indiges-



Three Psorosperms (two shown in section) from a Ureter. (Leitz, obj. $\frac{3}{8}$ in., Zeiss, oc. 2.) (After J. J. Clarke.)

tion, with fever, delirium and ultimately death. In the kidneys and ureters—a comparatively frequent seat of infection—the mucous membrane is found studded with minute cystic nodules, about 2 mm. in diameter, which may even block the ureter and cause hydronephrosis. Hematuria, anæmia and frequent micturition are the prominent features of the condition, and death occurs in a few weeks. In the skin, a spreading dermatitis is produced with nodular or vesicular lesions. When the infection is generalised the condition is comparable to trichinosis. The symptoms comprise pains in the limbs, vomiting, headache, delirium or drowsiness and irregular pyrexia. Death occurs generally in a fortnight or three weeks, the patient passing into a typhoid condition; occasionally the duration of the illness may be rather more prolonged. No treatment is known to influence the course of the infection. Diagnosis can only be made by the discovery of the typical cysts and parasites (see fig.). Psorosperms have been

described in Darier's disease (*Keratosis follicularis*) and in Paget's disease of the nipple; also in molluscum contagiosum and in carcinoma. It is most probable that the bodies taken for protozoa in these conditions are in most cases peculiar products of cellular degeneration. Plimmer and others have cultivated fungi allied to yeasts (*blastomycetes*) from carcinomata, and the same has been done in the disease peculiar to California, known as 'protozoic dermatitis,' which was originally ascribed to psorosperms. It is possible therefore that parasitic fungi have at times been classed as protozoa.

W. CECIL BOSANQUET.

PTOMAÏNES (πτῶμα, a dead body).—SYNON.: Cadaveric Alkaloids; Fr. *Ptomaines*. Ptomaines are alkaloids produced by the decomposition of animal substances. The word *ptomaine* was at first restricted to alkaloids produced by cadaveric decomposition, but it is now also employed to designate alkaloids of animal origin formed during life, as a result of chemical changes induced by some agency or other acting within the organism. The term *leucomaine* has been introduced to particularise the animal alkaloids formed during life from those produced by decomposition of dead animal matter; but it would be preferable for the terms *ptomaines* and *leucomaines* to be abandoned, and to class these bases of animal origin in one category as *animal alkaloids*.

It is now well known that the power of manufacturing alkaloids is not restricted to plants, but is shared by animal organisms.

Creatinine, discovered in urine by Liebig and Pettenkofer, was the first body of animal origin acknowledged to be an alkaloid.

Ptomaines of known composition.—The common ancestor of alkaloids, whether animal or vegetable, is albumen, the complex albumen-molecule being split up, either by bacterial agency or otherwise, into several less complex molecules, among which are the animal alkaloids. The following is a list of the principal ptomaines that have been extracted from putrefying animal matters, and submitted to ultimate analysis:—

Collodine, $C_8H_{11}N$, from putrefying horseflesh and mackerel.

Parvoline, $C_9H_{13}N$, from putrefying horseflesh and mackerel.

Unnamed base, $C_{10}H_{15}N$, from putrefying fibrin of bullock's blood.

Hydro-collodine, $C_8H_{13}N$, from putrefying horseflesh and mackerel.

Putrescine, $C_4H_{12}N_2$, from human corpses.

Neuridine, $C_5H_{11}N_2$, from human corpses, and from putrefying fish and cheese.

Cadaverine, $C_5H_{11}N_2$, from human corpses.

Neurine, $C_5H_{13}NO$, from cadaveric putrefaction.

Choline, $C_5H_{13}NO_2$, from cadaveric putrefaction.

Muscarine, $C_5H_{13}NO_2$, from putrid fish.

Gadinine, $C_7H_{16}NO_2$, from putrid cod-fish.

Tyrotaxonin (diazobenzene butyrate), from decomposing cheese, milk, and cream.

Animal alkaloids are also a necessary product of vital physiological processes, and have been extracted from the secretions of living beings, and from fresh animal tissues. The following is a list of the principal animal alkaloids so obtained:—

Creatinine, $C_4H_7N_3O$, from urine.

Pseudo-xanthine, $C_4H_5N_3O$, from urine and flesh.

Sarkine, $C_3H_4N_2O$, from urine and flesh.

Xanthine, $C_8H_8N_4O_2$, from urine and flesh.

Crusocreatinine, $C_8H_8N_4O$, from fresh meat.

Xanthocreatinine, $C_8H_{10}N_4O$, from fresh meat.

Guanine, $C_5H_7N_5O$, from flesh and guano.

Carnine, $C_7H_8N_4O_3$, from fresh meat.

Betaine, $C_3H_7NO_2$, from urine.

Mytilotoxine, $C_6H_{15}NO_2$, from poisonous mussels.

Ptomaines and Disease.—Animal alkaloids are being incessantly produced within our bodies as a result of the normal physiological processes, and they are eliminated by the bowels, kidneys, liver, skin, and lungs; but if from any cause these eliminating organs fail to perfectly fulfil their excretory functions, then an accumulation of these alkaloids in the system occurs, and a toxic action is exerted by them on the nervous centres. In this way can be explained the headache resulting from constipation, and the more serious nervous symptoms resulting from deficient excretory action of the kidneys in certain diseases of those organs. The removal of these animal alkaloids is, however, not only effected by the excretory organs, but, in addition, a powerful agent for their destruction is at work in the oxygen of the blood, which is continually oxidising and burning them up; and it seems probable that this combustion to a large extent occurs in the liver. If, the excretory organs remaining sound, there is excessive production of animal alkaloids, but inadequate elimination and destruction—a condition which is obtained in all forms of over-exertion, as in a prolonged march—then accumulation of material elaborated in excess and imperfectly eliminated or destroyed occurs, and an auto-infection, a temporary poisoning of the system, results, the poison affecting the nervous centres, and producing the fever of over-exertion, the fever of prostration.

As regards the infectious fevers, it is probable that after the admission of the specific micro-organisms into the body, and provided they find the conditions suitable, they live and multiply, and that, as a result or a residuum of their vital activity, a powerful alkaloidal or other poison is produced, the toxicity of which is the cause of the symptoms of the disease. If so, each infectious fever is the result of a fermentative decomposition of albuminous matter within the body, induced by a special micro-organism, manufacturing its own peculiar poison for each disease. See ANTITOXINS; BACTERIA; IMMUNITY; and TOXINS.

The chief medico-legal interest attaching to the ptomaines arises from their liability to be confounded with some of the vegetable alkaloids, and hence to the possibility of their leading to mistakes in medico-legal practice. There are no chemical reactions by which the ptomaines as a class may be distinguished from the vegetable alkaloids.

For a description of ptomaines in connection with food-poisoning, see FOOD, POISONOUS.

ARTHUR P. LUFF.

PTOSIS (πτῶσις, a fall).—A drooping or falling of the upper eyelid, with inability to raise it, due to paralysis of the third cranial nerve. See THIRD NERVE, Diseases of.

PTYALISM (πτύαλον, saliva).—A synonym for salivation, or excessive flow of saliva. See SALIVARY SECRETION, Disorders of.

PUBLIC HEALTH.¹—Broadly stated, the aim of Public Health, or Preventive Medicine, is to curtail, and, if possible, prevent diseases, to prolong existence, and to render life happier by means of improved physical conditions. Such care as communities took for the protection of their health was in early times taken under the auspices of religion or morality; it was hardly until the century which has just ended that serious attempts were made to understand the conditions making for health and disease among communities. Although many old statutes and provisions of the common law may be shown to have their bearing upon public health, organised legislation in England for the promotion of this object may be said to date from the Public Health Act, 1848. The country had by this time, under the system of registration inaugurated in 1837, and by the aid of Royal Commissions, learned something as to the causes of death in the population. It was apparent that year after year far more lives were being forfeited to home-grown epidemics than had been lost during the foreign epidemic of 1831–32. By 1848, the threat of fresh invasion by Asiatic cholera had become near and loud. The time for sanitary action was auspicious. It had come to be seen that communities, as such, had duties towards their members and towards other communities, in respect of matters affecting health; and with this enlarged sense of public responsibility it was felt that adequate organisations should be provided, in order to instruct, direct, and even coerce ignorant or wilful members of each community. Needless to say, this function of legislation speedily asserted itself more definitely and in fresh directions; until the present system of sanitary observation and administration has grown up.

Considering, first, the area of a country in its broader aspects, and always with more particular reference to England and the public-health arrangements of England, we note the influences exerted by the *condition of open lands, forests, and rivers*. The drainage of land, so as to carry off water readily, and thus to make both ground and air drier, has a great effect on certain aspects of the public health. Ague, once so common in England, has in consequence of the direct or indirect effects of drainage almost disappeared, as also has dysentery, which so often accompanies it.

The movements of the ground-water, which, by its rises and falls, influence the moisture and the amount of air in the soil, and, through these conditions, alter the amount and rapidity of decomposition therein, have been supposed also to influence health, and to be especially connected with the development of typhoid fever, cholera, and, more recently, diphtheria. One view is that a rising subsoil water may force out from the soil pathogenic organisms, and clearly such organisms may be thus washed into shallow wells. A moist ground is also believed, on tolerably strong evidence, to be favourable to the production of destructive lung-diseases; and rheumatism and catarrhal affections would appear to be more common on damp soils. But more accurate observations are necessary on these points. Although the influence

¹ This article treats in the main of the general principles concerned in the prevention and control of infectious diseases. The scheme devised by Dr. Parkes in the original issue is still adhered to, although parts of the article have been rewritten. Sanitary Law, Vital Statistics, and Bacteriology are dealt with in separate articles.—H. T. B.

of the ground-water in cholera is questionable, and it is by no means always active in the production of typhoid fever, it is certain that lowering the level of the ground-water when it is near the surface is often followed by the best results on the general health of the people, and in hot countries malarial diseases have been greatly diminished, even when the lowering of the ground-water has not exceeded a few inches. But in so far as malaria is concerned, recent researches with regard to the part played by certain species of mosquitoes (p. 949) render it extremely probable that drainage has acted by drying up the pools which are now known to form the breeding-places of the mosquitoes in question. A noteworthy relation has also been observed between the autumnal diarrhoea of children in towns and the earth-temperature at a depth of four feet, and recent researches confirm this observation.

The regulation of irrigation operations also has in some countries become an important question.

The control of forests ought to be considered a State matter, as the climate of a country and, therefore, health may be greatly influenced by them. In England such regulation of forests is not a matter of much national importance; it is otherwise in Germany and France, where laws exist which restrain private action; and in Italy, Greece, and Turkey the condition of the forests requires grave consideration as a matter of public health, as well as of climate and rainfall. In India this is also the case, and there are several important sanitary aspects under which the operations of the Forest Department need to be regarded.

The regulation of rivers, such as the embankments, narrowings, deepenings, and removal of obstructions, has generally been concerned with little else than navigation or the prevention of accumulations; but rivers are equally important from a sanitary standpoint, as they may influence the out-flow of the land-water from their drainage areas, and in that way may affect the level of the subsoil water and the dryness of the soil.

In another way the regulation of rivers is a matter of the greatest importance. They supply the drinking-water of the community to a large extent, and freedom from contamination is, therefore, necessary. For years this has been seen to form one of the most difficult questions of public health, and for some years a Royal Commission was engaged in inquiring into the causes and remedies of the pollution of rivers. A Second Commission presented its Report in special relation to supplies of drinking-water for London, and the decision of a Royal Commission which is at the present time considering the best means of sewage purification, and which has now issued an interim report recommending further administrative machinery, is likely to have an important effect upon the purity of our rivers. The chief causes of contamination are the dirty water and sewage coming from towns, and the refuse of trade operations. The former can in some measure be met by irrigation or by filtration through land, though the immense quantity of water to be purified and the price or position of land may cause difficulty. The admixture of trade refuse-water presents, however, the greater difficulty; yet to prohibit the flow into streams would sometimes be to prohibit the trade works. At present there is no settled general standard of purity for either town or trade water before its discharge into streams. Such standards were suggested by the

Rivers Pollution Prevention Commissioners, but they have never been accepted either by Parliament or the public, and it will perhaps be eventually recognised that for actual working the standard must vary with the place and trade, and must depend first on the demands made upon the river for drinking use; and, for the rest, on the purity of the river water into which the dirty effluent is discharged, and the comparative volumes of dirty and clean water thus mixed together—i.e. upon the extent of the dilution of the impure with the pure water. The number and kind of fish living in the river water are also to be taken into account, effluents of the highest obtainable purity only being admitted into trout or salmon rivers, or into those estuaries in which shell-fish such as oysters are cultivated for market. Thus it will be seen that, at present, no great progress has been made in the prevention of the pollution of rivers in England, the interests involved in the continuance of the polluted condition of streams being exceedingly strong, and the enforcement of the law being in the hands of authorities who are often the greatest offenders against its provisions. County Councils, however, who are now possessed of considerable sanitary functions, have been empowered to enforce the provisions of the Rivers Pollution Acts, and on the initiation of these Councils Joint Committees may be and have been formed to control the pollution of a river through its whole course. Much good has already resulted from the action of these bodies, who are to a certain extent free from the trammels of petty and local interests.

Of conditions operative upon the health of the individual and of the community, the one that falls most conspicuously within the province of *Local Sanitary Authorities*, as of the Legislature which created them, is the condition under which people have their dwelling—the state and circumstances of their *habitation*, both in the particular and in the aggregate. So true is this, and so strongly is this consideration felt in practice, that, for the rest, it will be convenient to arrange the subject-matters of the present article with the notion of condition of habitation in the foreground; and to regard each subject as it principally concerns communities inhabiting a larger or smaller place, or as it concerns the particular habitation. Thus, the general subject of public health will, with little exception, be here discussed with reference, first, to collections of houses forming cities, towns, and villages; secondly, to separate houses.

Cities, Towns and Villages.—The health of the inhabitants of English towns, as judged of by the annual rate of mortality, is not so good as that of the people of rural districts. See MORTALITY; and VITAL STATISTICS.

Hygienic Conditions of Cities, Towns, and Villages.—These are conditions referable to:

1. The site and soil.
2. The arrangement and building of houses.
3. The water-supply.
4. The removal of refuse-water and of dry refuse.
5. The removal of excreta.
6. The conservancy of the surface.
7. The supply of food, including the regulation of slaughter-houses, dairies, and bake-houses.
8. The regulation of trades.
9. The arrest and control of infectious diseases.
10. The disposal of the dead.
11. The supervision of nuisances.

1. *The Site and Soil.*—The sites of old cities were fixed by reason of war or commerce, or of vicinity to water-supply; when modern cities arise, it is often in consequence of the development of new industries, such as depend on coal and iron, or such as cotton or woollen works, where the site is determined by convenience of manufactures. In England, even at the present day, new towns and villages still tend to spring up without regulation, and when they attain a certain size, and some sort of municipal government is formed, it is often too late to consider the wholesomeness of the ground or to attend to the arrangement and construction of the existing houses. It were to be desired that the Legislature should secure for towns, during their period of growth and extension, more attention to such matters. For too often in the case of old towns Local Improvement or Health Acts are found to be needed, in order to remedy slowly and laboriously the errors of bygone times.

In respect of the *site* it is necessary to dry the ground if it is at all damp, and to keep it from being contaminated by injurious or offensive matters. It is one of the advantages of sewerage towns that the ground is thereby drained; and brick sewers were formerly laid so as to admit subsoil water and drain the ground as well as to serve as channels for house-waters. For the former object of drying the soil, every town ought to have a system of pervious sewers; and for the second object, it is best to have a system of impervious sewers combined with the deep drainage of the soil. There should be no cesspits or middens, or manure-heaps, in uncemented holes; all refuse of this kind should be removed and never allowed to soak into the ground. The ground ought to be secured against every source of contamination, a fact which, with a fuller knowledge of the behaviour of pathogenic organisms in soil, is becoming of more and more importance. Paving of all streets and courts, so as to prevent surface-impurities from soaking in, and great care in the construction of private drains and public sewers, will keep the soil of a city free from those impurities which, under the influence of heat, water, and air, generate injurious effluvia that may be sucked into houses. It is necessary also to have rules as to 'made ground.' Inequalities in the surface of the ground are often levelled by filling in with refuse of all kinds; house and chemical refuse, and dreggings from rivers, with other rubbish, are sometimes used. Decomposition goes on in such soils, and eventually, if not too foul, they purify themselves, but for this time is required. In the 'cinder refuse' of Liverpool, which is tolerably free from impurities, at least three years are required for the disappearance of the more easily decomposed animal and vegetable matters. In other made soils it may be longer; and when soil is very impure, as in the case of old graveyards, it is uncertain how long it is before it would be safe to build upon it. Sanitary authorities have, under certain conditions, power to prevent new buildings from being erected on ground upon which animal or vegetable matter has been deposited. Every made soil should be well drained, so that air and water may freely pass through it, and the best should have been laid down for three or four years before being built upon. Trial holes should be made in all cases of doubt.

The leakage of coal-gas from pipes is a point to

be guarded against, and the ease of preventing this would be much increased by the use of subways, such as are to be found in certain European cities.

With respect to the means of covering the sides of city streets for foot passengers, good stone paving or the like is essential; for it greatly increases the ease of cleaning the surface. Full powers are given to Sanitary Authorities for this purpose.

The question of the best kind of road for horse and carriage traffic is not quite so easily settled; there are four principal plans: macadamising, granite blocks, wood, and asphalt. As a mere matter of health the last is probably preferable; there is less *débris*, greater ease of cleaning, and less noise. Both macadamised and granite-block roads soon get worn into fine mud, which is made up of finely comminuted stone mixed with droppings from horses, and the like. In wet weather this is washed into the sewers, which it aids in obstructing, and it forms a useless part of the sewage. In dry weather it becomes pulverised, floats in the air and is one of the ingredients of city air, from which it is deposited as dust. Wood and asphalt break up much more slowly and are more easily cleaned both by rain and by washing, but it is necessary that the wood used should be as hard and impervious as possible.

2. *The Arrangement and Building of Houses.*—The arrangement of houses and streets in towns is influenced by many circumstances. A good return for money, facility of locomotion, and beauty, are the chief considerations in new towns. In old cities questions of defence and of materials have especially regulated the size and direction of their streets, and the height and compression of their houses. Many considerations will always influence the formation of streets, but a free passage of air to all parts of a town is a cardinal point, which should receive the utmost attention. The more numerous and the wider the streets are, the less impeded will be the air-flow. It is undesirable that a street should be less in width than one and a half times the height of the houses abutting thereon.

There should be open spaces, arranged to allow ready movement of air through them at the back of the houses, and all 'back-to-back' building should be illegal. The erection of narrow lanes and alleys should be prohibited in all new towns, and the back courts so common in our older towns ought to be gradually removed. Additional open spaces should be provided at intervals. Wide straight streets are useful for ventilation, and are best for the laying of pipes and tramways. Straight lines are by some not considered beautiful, but generally speaking they are certainly most convenient.

Powers are given to Urban Authorities to make by-laws regulating the width of new streets, providing for sewerage, foundation of houses, spaces for air about houses, the drainage of buildings, and many other matters. As regards existing towns, these Authorities are enabled to purchase dwellings in order to improve streets, to set back houses when rebuilt, and in cases where only the front of a house in a street is taken down they may prescribe the line of the new building. Larger powers of demolition and reconstruction, moreover, are given by the Housing of the Working Classes Acts, the facilities for the administration of which have been quite recently extended, and various local statutes. It has been said that due provision

should be made beforehand for the proper construction of the many new towns which must needs spring up in the course of another century. The case seems clear for the community at large to regulate matters so important as these for the general health of the population are proper for Imperial legislation to deal with to a greater degree than has been yet recognised in any Act. In their principles at least, such matters should not be relegated to Local Authorities, for them to accept and to use according to the local caprice of the moment.

An important point to determine is the height of the houses, a matter which is assuming serious dimensions in London and other of our large towns. In England a large proportion of our towns consists of low brick houses. If these are not too crowded they give a good distribution of the inhabitants, and oppose little obstacle to the movement of air and the distribution of light. When the houses are very lofty the air-currents must be much more impeded, and therefore the streets ought to be much wider, and open spaces behind and about them ought to be more carefully secured, a point which has been much neglected in the erection of modern blocks of 'flats.' It is not possible to state with any precision the number of persons who may be located on an acre—this will depend in the main on the construction of the individual houses; but it may be laid down as a general rule that, however small be the size of the houses, the amount of ground not occupied by them in any given acre should be as great as the amount actually taken up by houses, and such a rule should *à fortiori* be maintained in the case of more populous houses. Where the houses of a town are intended, each of them, for the lodgment of several families, and consist (with the name of 'flats' or 'mansions') of a number of separate dwellings piled up on the top of each other, it is of even greater importance that the relation between inhabited and uninhabited area should be properly regulated. Such flats or mansions ought, for the sake of air and light, to be provided with exceptional arrangements for space about them, with, concurrently, abundant ventilation of streets and provision of open spaces. They may serve to illustrate the foregoing contention as to the need for supervision by an Imperial or at least a county authority over the arrangements of streets and houses; for every Local Authority can hardly be expected to be sufficiently alive to the misfortunes that have befallen certain large towns through want of due regulation of houses of this class. The construction of the separate houses should not be altogether a matter of absolute control by a municipality; and certain rules as to ground-plan, foundations, arrangement of closets, and the thickness of party walls are habitually enjoined by the State upon Local Authorities desirous of regulating their new buildings; while sufficient liberty should be afforded to the requirements and tastes of individual owners and architects.

So in all houses, whether urban or rural, there should be means of ventilation for every room; no inhabited room should have a borrowed light, but should have a window opening directly on the external air; every window should open, and especially at the top; every room should be of good height, not less than nine feet in the smallest, and ten and eleven feet in larger rooms; the closets should, when practicable, be arranged in such a

manner that, in addition to ventilation of the closet itself, there should be thorough cross ventilation into the open air between the closet and the rest of the house, and this is best accomplished by having projecting portions of the building to contain the closets; every house should be provided with closets in due proportion to its population; there should be proper water-supply, with easily inspected storage, if house-storage is permitted, and easy and efficient methods of carrying off the dirty house-water: there should be proper arrangements for the collection and temporary storage of dry house refuse; and house drains and pipes should be constructed and ventilated on the principles that will presently be set forth.

All these matters are easy to regulate without interfering unduly with the plans of the architect.

3. *The Water-supply.*—In a town with sewers and water-closets it is generally considered that the supply of water per head daily should not be less than 25 gallons; and if there are trades using large quantities of water, from five to ten gallons additional (reckoned per head of population) are wanted for the town. If there are no water-closets, from 14 to 20 gallons per head daily appears to be the amount usually considered sufficient in large English towns.

The sources of supply are natural lakes, artificial lakes and gathering grounds, rivers, springs, and wells. In towns of any size superficial and shallow wells are always suspicious sources. Local authorities have powers to undertake the public supply of water, and to protect water-supplies; also to close wells, tanks, cisterns, or pumps if the water be polluted. But additional general powers are urgently required to enable a well to be closed in the event of its surroundings being such as to threaten the purity of the water.

The following are the matters of chief importance in towns: (a) The supply should be taken from sources capable of affording a quantity adequate to the present and proximate wants of the town, with such approach to constancy as may be attainable. In quality, the great points are to ensure that the water is clear or is easily or completely freed from sediment by sand-filtration, and is well aerated, pleasant to taste, and without smell; that it contains no injurious animal constituents, and cannot become contaminated with excreta of men or animals, or with foul water from houses; that it contains no injurious amount of vegetable matter (not more than 2 or 3 grains per gallon), and that its mineral constituents are of moderate amount, and consisting of such mineral matters as are not likely to be injurious. With respect to lime especially, much discussion has taken place as to whether soft or temporarily hard (from calcium carbonate) water is best for a town; the soft water is preferred for many trades and is probably best for health, though it has been found impossible to prove this by statistics; it is certain that the inhabitants of numerous towns using a good chalk water have excellent health, and it would seem in fact that the question between water hard from calcium carbonate and soft water is not an important one. The use, however, of hard water leads to enormous waste of soap, is liable to block up cisterns and pipes by a deposit of 'fur,' and is said to be bad for cooking purposes. When water is *permanently* hard from calcium chloride and sulphate it is thought to be objectionable

to health. The great point in choosing water is, in practice, its freedom from any chance of contamination with excreta, or with refuse matter from habitations.

The duties of a medical officer of health should include the supervision of the sources of supply, so as to detect and prevent any possible contamination, and such officer should have legal access at all times to the premises of any Water Company supplying his district with water. Frequent bacteriological, as well as chemical, examinations of the water should be made.

The water when supplied, except in the case of deep-well waters, most commonly needs to be stored and filtered. The reservoirs of our towns contain from one to three months' supply, or less if the supply is very uniform in quantity. The reservoirs require to be placed so as to be clear of trees, and protected from danger of anything being thrown into them. Filters which aim at straining off matter in suspension and oxidising organic substances are usually made of sand about 3 feet in depth resting upon gravel, and the filtration, according to Koch, should be so conducted that (a) the pace should not exceed 3·94 inches per hour, each filter being provided with a regulating contrivance; (b) the water from each filter should be examined bacteriologically once daily; (c) the filtered water should contain no more than 100 micro-organisms per cubic centimetre. Where there is no public supply and domestic filters are used, frequent cleaning is essential. Filters made of unglazed porcelain or of infusorial earth are the most reliable.

After filtration the water is distributed by means of mains usually of iron tarred or varnished inside, for the larger conduits, and then by leaden service-pipes, or, under certain circumstances, tinned-lead pipes, for the smaller. Both iron and lead, and especially the latter, are dissolved by some waters, and the question whether lead is so dissolved has often to be answered; in examining into this matter the water should be taken after it has been in contact with the pipes for some hours—for instance, in the early morning.

Carried down the public pipes, the water is either delivered at intervals to house-cisterns, or, what is far better, is supplied on the constant plan without house-storage. If it be not possible to dispense with house-cisterns, they should be well made of slate, stoneware, or galvanised iron, should be able to be easily inspected and cleaned, and their overflow pipes should always end in the open air, never go into any drain. The greatest care should be taken that the cistern-water shall run no risk of contamination by absorption of foul air or by soakage into the cistern, which should be well covered to prevent dust getting in.

If the 'constant system' is in force, it should be truly constant, for if the water is cut off at intervals and the house-pipes are then emptied, or if leakages take place in the street mains, air must be drawn into them, and this air may be foul; it has even happened that dirty liquids have been sucked into water-pipes, as where a closet service-pipe direct from the house-main has been connected with a choked closet-pan, and in this way excreta have not only passed into these house-pipes, but have even got into the mains. In a similar manner, when sewers are laid in the same trenches as water-mains, liquids escaping from leaky sewers may find their way through bad joints or fissures in water-

pipes. That is most liable to occur during intermissions in the water-service; but the same insuction into water-pipes may occur while the pipes are full, if the water be running through them with great velocity. This fact is not sufficiently recognised, even by water-engineers; yet it has been concerned in the distribution of enteric fever broadcast in a community.

Under a constant system and under an intermittent system alike, small service-cisterns are needed for water-closets and for kitchen-boilers, and precautions have to be taken with these cisterns equally with larger storage-cisterns. In fact, too great care cannot be taken in thoroughly guarding water-pipes and cisterns in every way. Waste-pipes from cisterns must have no connection with the drains, but should discharge freely in the open air.

The sources of contamination of drinking-water are very numerous, and may affect the water at its source, in its flow, in the reservoir, or during distribution. If stored in houses it is especially exposed to risk; and this is the grand argument for constant service, that the water may be delivered immediately after filtration. The plan of cistern-storage, indeed, lessens those risks that are incidental to intermissions; but this plan demands that cisterns be properly made and placed, and be regularly cleaned. For low-rented houses these conditions are very difficult of attainment, and therefore the constant service is peculiarly adapted to the houses of the poor.

In all towns the service should be at high pressure, so that water may be carried to every floor and thus labour be spared, and the freshness of the water be secured. In places where the water is not carried into the houses, but is fetched from pumps or from 'hydrants' in the street, it has to be stored in the houses in buckets, and runs many chances of impurity.

A town requires water for public purposes, such as for public baths, washhouses, flooding and washing streets, flushing sewers, and putting out fires, and for this latter purpose sufficient and constant pressure is essential. Statutory powers are given for carrying out these objects.

4. *The Disposal of Dirty House-water and Dry Refuse.*—After being distributed and used in houses or trades, the water with the impurities it has gathered must be carried out of the town. The inhabitants should have no difficulty in getting rid of their dirty water, or otherwise dirty water will come to be used improperly several times over. Houses ought to have convenient sinks discharging by trapped pipes opening outside the house, not into a drain, but over a grating in connection with a trapped inlet or gully, in the manner provided for in the Model By-laws. From these it must go along pipes or sewers, and be disposed of at the outfall in some way. House-water, besides other impurities, frequently includes some portion of urine which, as we now know, may contain certain pathogenic organisms. It is not fit to be at once discharged into streams, but as its fertilising powers are considerable it is well adapted for irrigation on land. The plan involving the least expense appears to be to filter it by intermittent filtration on a small but sufficient area of properly prepared and drained ground, provided such can be procured.

The dry refuse of houses consists of cinders and ashes, remains of food, dust from sweepings, and

various other used-up articles of house life. In some towns there is little difficulty in disposing of this refuse. After being carted away it is sorted, and every article finds a sale. In other towns, however, the disposal of the house-refuse is a matter of difficulty and expense. A system of destroying the refuse by fire in destructor furnaces has of late years come largely into use in various towns and is making rapid progress. The refuse contains a sufficiency of combustible material in the shape of cinders to ensure its complete destruction in a properly designed furnace, and the waste heat may be utilised for various municipal purposes, e.g. for converting the contents of pail-closets into a dry manure, or for generating steam in boilers for driving electric-lighting machinery. The clinkers, when withdrawn from the furnace, can be ground down in a mortar-mill and converted into mortar, bricks, or concrete. Sanitarily considered, the destruction by heat of dust-bin refuse is far preferable to the old-fashioned sorting method. In some places the dry refuse is placed every day by the inhabitants in front of the houses and is removed by scavengers. In other places there must be storage of refuse on the premises for a varying number of days; if this is requisite, every house should have a properly prepared dust-bin, well paved to prevent soakage, well covered so that its contents may be kept dry and free from the influences of wind, and so placed as to be away from the house, though convenient for the house as well as for the town-savengers. In the building of any house the arrangements for the position of the dust-bin are almost as important as those for the closets. Of late, galvanised iron pails with tight-fitting lids have come largely into use to replace brick dust-bins for the storage of dry house-refuse, and being non-absorbent they are far more cleanly and suitable receptacles than brick structures could ever be. The removal ought to be frequent and regular, but the frequency has to be fixed by special circumstances. As far as possible organic refuse (food scraps &c.) should be burnt in the kitchen fire, and not stored on the premises to await removal by the scavengers.

5. *The Removal of Excreta.*—The excreta of the skin and lungs are got rid of by ventilation and washing, so that this heading refers only to the solid and liquid excrements. These average respectively (for both sexes and all ages) about $2\frac{1}{2}$ ounces avoirdupois of solid excrement and 40 fluid ounces of urine per diem.

The excreta must not soak into the earth, or remain near dwellings. The common privy and the 'midden' of northern towns can only with difficulty be brought to fulfil these conditions. In towns above 10,000 inhabitants it now seems clear that there is no possibility of using the earth-closet system, on account of the expense of preparation and transport of dry earth. Therefore, for towns in this country, two plans alone remain: (1) The dry plan with frequent removal, with perhaps such deodorisation as the ashes of the house may give; the so-called 'pail system' is one form of this dry plan. (2) The 'water system,' the excreta being carried off from the house along drains and sewers, by the aid of water. The air or pneumatic systems of Liernur and Berlier, which are in use in certain parts of the Continent, and by which the excreta, unmixed with water, are sucked through pipes into

a central reservoir by an air-pump, worked by a steam-engine, are perhaps not altogether well suited to English ideas and habits.

It would not be possible to discuss here the relative value and the technical details of the pail- and the water-systems. Both are largely used in England. The pail-system, as the most adaptable (among dry systems) to the conditions of a town, has been used in towns where proper sewers cannot be made or water is deficient, or where land cannot be obtained for irrigation or filtration, owing to the expenses involved. It has the disadvantage of being opposed to one of the first principles of sanitation in that it keeps the excreta for some days near the house, besides being sometimes attended with nuisances in the working. But, nevertheless, it is capable of keeping a town fairly clean when it is properly carried out, and it is an immense advance over the old 'midden' and 'cesspool' systems, which retained the excreta in the filthiest receptacles for long periods in the very midst of the people. It is of the essence of the pail-system that the removal of the excreta should be frequent, that is, if practicable, every day. After removal, the excreta are applied at once to the land, or are made into 'poudrette.' In some towns the house-ashes are thrown on a wire screen, so as to allow the fine ash to fall on the excreta—this is sometimes called the 'ash plan'; in other cases deodorants are used. The 'Goux system' is to place some absorbent material round the interior of the pail to absorb the urine.

The water-system is more elaborate, and probably more expensive, but if properly carried out is more effectual. If a town can make good sewers, and can find water for flushing and proper means for disposal of the sewage, the water-system would certainly appear to be the best for health and general comfort.

There cannot be many towns which could not acquire the quantity of water needed for the proper use of a water-system. By some simple contrivances waste water—the water that has been put to other domestic purposes—can be made use of for washing out the closets. Slop-closets and trough-closets, in which these contrivances are used, are found to work efficiently, and to economise water in places where there is but scant supply.

The drainage of every house that discharges its excrements by the water-system should possess the following arrangements for disconnecting the drain-air from the air of the common sewer, and for dispersing the foul air of its own particular drain: (a) near the junction with the sewer the house-drain should be provided with a 'siphon-trap,' through which all the liquids of the house must pass, and which, therefore, must always be charged with water while the house is inhabited; (b) an opening from the outside air to the house-drain, made on the outside of the siphon-trap, to serve as an inlet to the house-drainage ventilating system; the outlet of this system being (c) a pipe in continuation of the farther end of the drain carried up 'full-bore' above the roof, its end at this spot being left open, save for a wire-grating to prevent leaves or other wind-borne substances from being deposited therein. Means of inspection and of access to the house-drains should be provided at convenient situations.

It is essential that sewers should be well constructed; they should allow no deposit; and they should be thoroughly ventilated. Deposits are prevented by having egg-shaped sewers with a proper

fall, easy means of access for inspection and cleaning, and a regular flow of water, with periodical flushing. The ventilation of sewers is perhaps best effected by having numerous openings—as many, in fact, as can be made—so as to allow constant and free interchange between the sewer-air and the atmosphere. These openings may be by street-gratings or by special shafts of sufficient calibre, according to circumstances. Ventilation through furnace-chimneys is inadvisable, and is of no avail for distant portions of the sewers. For the dead ends of sewers and in narrow streets and courts, shafts of not less than 6 inches diameter, carried up from the crown of the sewer to above the roofs of the houses, should be provided. But in whatever way the ventilation is carried out, the rule must be to have the freest communication between the sewer-air and the general atmosphere. This free ventilation occasions no offence if the sewers are properly made and kept; while, if the air of sewers at the ventilators is found offensive, the ventilation will at least have provided against the more dangerous discharge of the foul air into houses.

Sewers have been objected to on account of the alleged occasional spread of typhoid fever and diarrhoeal affections, and perhaps of cholera and diphtheria, by their agency; but, if properly arranged, and with disconnection between the sewer and houses, there should be no danger, and recent work with regard to the bacteria in sewage and in the air of sewers tends to support this view. It is difficult to see how sewers can be displaced, or any other plan be substituted for them, in a town. The house-water must be carried off, and it is impure even if no excreta are allowed to flow in. Even if the pail- or other system for disposal of excrement be adopted, there must still be town sewers for dirty house-water, and all the precautions above alluded to must be enforced. Sewers, then, whether or not they receive the excreta of a town, are a necessity, and with proper construction and management they should be solely beneficial to the public health. It is to a certain extent a question of engineering detail whether the sewers carrying the house-water should also carry off the rain-water; but there would seem to be considerable sanitary advantages in the 'separate' system by which different channels are provided for house-sewage and rain-water: the chief, perhaps, being that under this system impermeable pipe-sewers of small diameter may be used to convey the sewage, and that the amount being more constant than when a rainfall is admitted, there is less difficulty in dealing with the sewage for purification purposes. Moreover, the sewage is richer in fertilising properties. Wherever towns situated on moist grounds have adopted a system of impermeable sewers for conveying away waste-waters and excreta from houses, means should be taken to dry the subsoil by laying porous drains at a sufficient depth. The rain and surface-waters may be carried off by surface-channels and gutters, where the levels permit, or in other cases by the subsoil drains.

The subject of purification and disposal of sewage which, as has been stated, is now under consideration of a Royal Commission, has received much attention of late owing to the progress of our knowledge as to the action of bacteria in the liquefaction and 'nitrification' of sewage. Briefly stated, sewage may be purified in a larger or smaller degree by mechanical, chemical, and biological agencies, and in practice a combination of such agencies is fre-

quently made use of. Each has its advantage at certain periods and under certain circumstances, and the manner of application of the several agencies is by *Screening*, *Chemical Precipitation*, with *Sedimentation*, *Filtration* (including under this term *Irrigation*), and *Liquefaction* in what is known as the 'Septic Tank.' A few words only can be said in reference to each.

Screening and Deposit are almost essential preliminaries to many methods of treatment in order to separate out from the sewage solid organic and inorganic bodies. Grit-chambers fixed on revolving screens are usually used. What are known as 'Roughing Filters' are sometimes employed.

Chemical Precipitation by aid of chemicals, such as lime, alumina, iron salts, and many other substances, either alone or in combination, seeks, by forming a precipitate, to arrest and carry down, during the process of sedimentation in settling tanks, much of the suspended matter—including, doubtless, a number of the bacteria originally present—together with an uncertain amount of the organic substance in solution. Although the effluent is often clear in appearance, it contains the chlorides and ammonia, and further treatment by filtration or irrigation is necessary to oxidise the organic matter remaining. One of the great drawbacks to precipitation-processes is the difficulty of disposing of the resulting sludge.

Filtration is used either alone or in conjunction with chemical precipitation or the septic tank process. Filters of many substances have been, and are being used, and the object of all, be they of earth, coke, ballast, &c., is, besides separating suspended substances, to oxidise the organic matter. It is necessary to provide, by periods of rest or in other ways, that the filtering material may be duly aerated. In some cases the filter is quite filled with sewage, and allowed to remain thus for a considerable period, the filter being then known as a 'contact' filter; in others the filtration is for the time being continuous. In each case periods of rest for aeration are necessary, and the several filters in use at the present time differ from one another, not only as to the nature and size of the material used, but also in the manner in which aeration is brought about. The principle involved in each case is how best to utilise the bacteria already present, which on the one hand act in the absence of air, and on the other require air for their operations, but it is probable that both aerobic and anaerobic bacteria are partially operative in all instances. When land is used as a filtering agent, it is necessary in most instances to under-drain it, the object of land-filtration being 'the concentration of sewage at short intervals on an area of specially chosen porous ground as small as will absorb and cleanse it, not excluding vegetation, but making the produce of secondary importance.' When, however, the land is used for what is known as *Broad Irrigation*, and where under-draining is not absolutely essential, the object is 'the distribution of sewage over a large surface of ordinary agricultural land, having in view a maximum growth of vegetation (consistent with due purification) for the amount of sewage supplied.'

The 'Septic Tank' or *Liquefaction* process, which was first introduced at Exeter, where it is still in use, consists in allowing the crude sewage to pass slowly (in about 24 hours) through a closed tank from which light and air are excluded. It is

found that during the passage the solids have become liquefied, mainly, it is believed, through the action of the anaërobic organisms; the sewage on leaving the tank being passed through coke-breeze or other filters. The action of the tank may be regarded as roughly similar in principle to that which takes place in the coarse filters of other biological processes.

6. *The Conservancy of the Surface Area.*—The cleansing of the surface-area of towns has long been a function of the public Authority. The sanitary importance of thorough surface-cleansing is obvious; the mud and dirt of towns and refuse of all kinds, wetted by rain and exposed to heat, soon decompose and give out injurious effluvia, especially in narrow courts and lanes where the movement of air is impeded. The necessity of such cleansing is becoming more and more apparent as our knowledge of the relation between polluted soil and disease increases. Public streets of all kinds can be easily kept clean, but want of paving and consequent foulness on private premises require to be sought out. Especially this is of importance where (as in the case of pigsties and stables) neglect of surface-cleansing may give rise to nuisances injurious to health.

7. *The Supply of Food, including the Regulation of Slaughter-houses, Dairies, and Bake-houses.*—A very important duty of a municipality, and one to which more attention is being daily paid, is to supervise the food of the people. While the price and quality must be left to the ordinary operations of commerce, the responsibility of preventing falsifications, and of ensuring that the article shall not be injurious to health, is devolved upon County Councils and on Sanitary Authorities. It is to these latter bodies that the regulation of slaughter-houses and knackers' yards is entrusted. Private *slaughter-houses* may be licensed, and can be visited and subjected to by-laws. They are often constructed out of buildings intended for other purposes, are not fitted with proper appliances, and are generally placed in the densest part of the town. The evils attending them are gradually being removed by the erection of public slaughter-houses, where abundant air, water, good sewers, and means of cleansing obtain, and where facilities for the skilled inspection of beasts and meat, and for the refrigeration of the latter, are, or should be, available. The custom of slaughtering in the country and then sending the meat to cities is increasing, and this again renders private slaughter-houses less necessary.

The transport of cattle and sheep to towns is a matter of very great importance as respects both the goodness of the meat and the comfort of the animals. Space in the trucks, supply of water and food, length of journeys, and other matters, require regulation.

Cow-houses, Dairies, and Milk-shops are now inspected by Sanitary Authorities, in pursuance of the powers of the Dairies, Cow-sheds, and Milk-shops Order of 1885 and of later amending Orders. These orders contain provisions for the registration of cow-keepers, dairymen, and purveyors of milk; for regulating the lighting, ventilation, cleansing, drainage, and water-supply of dairies, cow-sheds, and milk-shops; and prescribe the precautions that must be taken to guard milk against exposure to

infection or contamination. These latter precautions are especially necessary in the light of the knowledge we now possess of the spread of epidemics of enteric fever, scarlet fever, and diphtheria among human communities by the agency of milk; and it is gradually becoming more and more certain that if these disease outbreaks are to be effectually limited, not only must the sanitary arrangements of dairies and cow-sheds be under better and constant supervision, so that the milk may receive no impurities from water, air, or attendants, but that the animals themselves must be periodically inspected; for there is now evidence to hand that diseased conditions of the cows themselves may be the means of imbuing the milk with infective properties. It will be sufficient to mention two diseases alone, namely, tuberculosis and scarlet fever. The experiments recently conducted with tuberculous milk leave little room for doubt that such milk may be instrumental in conveying tuberculosis to human beings, and this notwithstanding the experiments and expression of opinion by Professor Koch to the effect that human tuberculosis is not communicable by bovines. In respect of the latter disease there is evidence pointing to the conclusion that the disease known as the 'Hendon' cow-disease is closely similar in character to human scarlet fever. The growing practice, more particularly on the Continent, of pasteurising milk and cream will do much to reduce the risks of conveying specific disease through the agency of milk, cream, and butter, while the wider application of the tuberculin-test to milch cattle will result in much good in regard to tuberculosis alone. Milk from cows with tuberculous udders may not now be used for human consumption.

Bake-houses are required by law to be kept in a cleanly condition, to be properly ventilated and protected from effluvia, and not to be used as a sleeping-place.

The inspection of the chief *articles of food*, in respect of their wholesomeness, is entrusted to Sanitary Authorities, and has reference to meat, game, poultry, fish, fruit, vegetables, corn, bread, flour, and milk.

The following are the chief sanitary points in each case:—

Meat.—Much doubt exists as to the extent to which the condemnation of meat exposed to sale should be carried. There is no doubt that meat sufficiently decomposed to be discoloured and to have a putrid smell, and meat with abscesses and suppurations, should be condemned, but the difficulty arises with meat apparently sound or not very obviously otherwise, but which is derived from diseased animals.

It is impracticable with our present limited knowledge to determine in all cases by inspection of the dead meat whether disease has been present during life, and the proper safeguard is to be found in an examination by skilled inspectors of the living animal and of the carcase after death, a combination which can only be adequately carried out in public slaughter-houses. Precautions of this nature are the more necessary since, in several instances, meat responsible for outbreaks of poisoning has presented a not unhealthy appearance. It is, too, essential that meat, some of which is thought to have produced illness should be examined as early as possible, rather than after putrefactive organisms have

replaced the pathogenic bacteria. For instance, Durham has recently shown that the *Bacillus enteritidis* of Gärtner, which has been regarded as responsible for numerous outbreaks of poisoning, may be found much more readily if the examination be made early. Meat may produce injurious effects by means of poisonous drugs or foods taken by the animal, by *leucomaines* and *toxins* produced in the living body, from pathogenic bacteria or their products, and by *post-mortem* changes. One or another form of pig's flesh has been responsible for numerous outbreaks of food-poisoning, and what is known as *Botulism* or sausage-poisoning has been common in Germany, where much raw meat is consumed. Cooking may be thought of as likely to destroy certain pathogenic organisms, but those situated in the centre of a roll of meat are likely to escape. Moreover, heat employed may not suffice to render harmless all bacterial products. It seems, however, that the toxin of the *B. botulinus* is rendered inactive by a heat of from 60°–70° C. A specific microbe was found by Klein to be associated with an outbreak of infectious pneumonia at Middlesborough, and the same observer discovered in ham, which was supposed to have given rise to 'Welbeck Disease,' certain bacilli, cultures of which produced the same symptoms when given to guinea-pigs and white mice.

Though opinions differ on this point, and there is obviously need of far more exact knowledge upon the subject, it may perhaps be said that meat derived from animals slaughtered in the early stages of inflammatory diseases and of epidemic pleuro-pneumonia may be used, but that beef from cattle dead of cattle-plague and anthrax (malignant pustule), mutton from sheep with small-pox and splenic apoplexy, and pork from pigs with carbuncular diseases, hog-cholera, hog-typhus, and scarlet fever, should not be used, although it is not easy to give conclusive evidence against all these diseases, as injuring the health of consumers of the meat. Cattle-plague meat, for example, has been largely used without apparent injury. Opinions are much divided as to whether the flesh of braxy sheep, or of cattle dead of foot-and-mouth disease, should be used or not, but it is certainly best to act on the safe side, and the affected parts should not be used. Meat obtained from animals which were suffering from pyæmia, septicæmia or puerperal fever has apparently been responsible for certain outbreaks, and calves and young pigs seem to be prone to septicæmic disorders.

In the case of the parasitic diseases of animals the question is easier. It is of course highly dangerous to use pork infected with *Trichina*. *Cysticerci* in pork, beef, and mutton should also be a valid ground for not permitting the sale, for it is not enough to expect the destruction of the parasite by the cooking that the meat will receive. If it be contended that the prohibition would affect supply, the answer is to be found in the consideration that breeders and salesmen would take greater care in preserving the cattle from parasitic infection; and that this can be done, by supplying pure water and clean food, is shown by the experience of Upper India.

Flukes in the liver do not constitute a valid ground of rejection of the meat, though the liver ought not to be eaten.

On the whole, it may be said that it is certainly wiser to condemn all meat which is derived from

diseased animals, even when the animals are slaughtered, and most certainly when they have died of disease, for there are possible risks in the consumption of such food, and the State is warranted in interfering to prevent the individual being exposed to any such dangers.

The subject of tuberculous meat has within recent years been considered by two Royal Commissions. The evidence adduced, both experimental and other, was held to justify the inference that human tuberculosis can be acquired by the consumption of tuberculous meat; but, having regard to the general custom of cooking meat, and provided due care be taken to exclude obviously tuberculous material, it would seem that actual danger may in the past have been somewhat exaggerated.

The last Royal Commission advised the observation of the following principles in the inspection of tuberculous carcasses of cattle:—

- | | |
|--|---|
| (a) When there is milary tuberculosis of both lungs | The entire carcass, and all the organs, may be seized. |
| (b) When tuberculous lesions are present on the pleura and peritoneum | |
| (c) When tuberculous lesions are present in the muscular system, or in the lymphatic glands embedded in or between the muscles | |
| (d) When tuberculous lesions exist in any part of an emaciated carcass | |
| (a) When the lesions are confined to the lungs and the thoracic lymphatic glands | The carcass, if otherwise healthy, shall not be condemned; but every part of it containing tuberculous lesions shall be seized. |
| (b) When the lesions are confined to the liver | |
| (c) When the lesions are confined to the pharyngeal lymphatic glands | |
| (d) When the lesions are confined to any combination of the foregoing, but are collectively small in extent | |

In regard to pigs, the Commissioners recommend that the presence of tubercular deposit in any degree should involve seizure of the whole carcass and of the organs. Professor Koch does not share these views: he thinks it undesirable to take any precautions against the spread of tubercle by the agency of meat. His views are, however, not generally accepted by pathologists, and a Royal Commission will no doubt have to examine into the question.

Shell-fish.—Certain of the mollusca, such as oysters, mussels and cockles, may be at times instrumental in conveying enteric fever by virtue of their having been gathered near to sewer-outfalls. These mollusca also give rise now and again to acute poisoning, and sometimes to a form of mixed infection, in which attacks of acute gastritis are followed after some days by a typical attack of enteric fever. Proximity to sewage is probably responsible for some of the acute poisoning, as well as for enteric fever, and it is found that when poisonous mussels are placed in water free from contamination they lose their poisonous properties. Some of the crustacea are instrumental in producing severe and even fatal poisoning. The relation of the food of molluscs and crustaceans to the attacks above referred to has not secured adequate attention.

Wheat-flour and bread.—The chief points are to ascertain that there is no ergot, no fungi, nor acari; that alum has not been used; and that other grains or mineral matter are not mixed with it.

Of *Milk*, the chief falsifications consist in addition of water or removal of cream, and the addition of preservatives such as borax, boric acid, formalin, &c., to milk is a subject which is clearly of no small concern to the sanitarian and to the medical

practitioner. The indiscriminate and unknown employment of substances used as therapeutic agents obviously calls for control, and the matter has quite recently been the subject of an inquiry by a Departmental Committee of the Local Government Board. The Committee will, it is expected, recommend that the use of all chemical preservatives in milk should be prohibited. Milk may also be improper for use owing to the presence of blood, lacteal casts, and pus; and unquestionably it will be dangerous if it have been derived from cows affected with the diseases before mentioned. Although there are no very ready means of discerning this dangerous quality, any risk of specific disease may be much diminished by means of pasteurisation.

Other foods are not often concerned in the production of disease, except in so far as they may have undergone decomposition. Accordingly they less often come under the cognisance of health-officers than of the analysts appointed under the Sale of Food and Drugs Acts. It may be defined as the business of these analysts to determine whether or not an article really is what it professes to be; to detect the presence and amount of foreign substances, or of decomposition and putrefaction; and sometimes to show whether or not a given specimen reaches a certain appointed standard of value. The law permits mixtures to be sold in some cases, if the admixture is stated on a label.

The Preparation and Storage of Food.—More attention should be paid to both these matters. Sanitary Authorities should possess extended powers to examine into the methods of food-preparation, such, for instance, as that of preserved and tinned meats, fish, and fruits, the making of sausages, margarine, &c. The manufacture of beers, wines, and temperance-drinks, as also of the substances used in their preparation and preservation, should be under better control, the recent occurrence of arsenical poison in connection with the consumption of certain beers having amply justified the legislature in taking further steps in this direction (p. 557). The position of larders in respect of freedom from drain-effluvia and contaminated dust is important, as also is the storage of foods in hotels, restaurants, &c.

The Investigation of Illness suspected to be caused by Food.—Inquiries of this nature should be approached in a systematic manner, and it will be found useful in dealing with any given household to make a complete list of every article consumed by each inmate, whether such inmate has been attacked or not. By arranging the information in a tabular form it will be possible to see at a glance what, if any, foods come beneath suspicion and what may be dismissed.

8. *The Regulation of Trades.*—Trades are affected by the law under two aspects: 1st, irrespective of the nature of the particular trade, the *place where it is carried on* is regulated under the Mines, Factories, and Workshops Acts, and by the Public Health Act of 1875. Urban Authorities can make by-laws regulating offensive trades, such as blood- and bone-boiling, fell-mongering, soap-, tallow-, and tripe-boiling, &c. The object of these Acts, among other things (such as restriction of labour at certain ages), is to provide that the common conditions of health are not violated. This is a very necessary point, for many workshops are

deficient in light and air, are badly ventilated, or are rendered unhealthy by gas burnt for light. Many small workshops are owned by men of small capital, who would sacrifice the health of workmen by compelling them to work under very unfavourable conditions. Happily the faults are usually easily remedied by a little common sense and simple appliances, and in this respect the Workshops and Factories Acts have done great good. One special fault in many workshops is, however, still common, namely, the burning of gas in large quantities in dark shops, without proper means of carrying off the products; the very great influence of this condition on the lungs was long ago pointed out by the late Dr. Guy.

2nd. The other point in the regulation of trades is to prevent any of the *processes being nuisances* or injurious to the health either of the workpeople or the inhabitants of the surrounding districts. This is an extremely wide subject. Trades may annoy and inconvenience the public, as by offensive effluvia, black smoke, or acid vapour which destroys vegetation, yet may not be distinctly injurious to health. On the other hand, without being notable nuisances in the above sense, they may be hurtful to health, especially those (and they are very numerous) which give rise to dust in the air of any kind. Cotton and woollen *débris*, filings and grindings, particles of size, clay, dry paints, and many other substances, come under this head. There is no doubt that the inhalation of all solid particles, no matter whence derived, is highly injurious to health. Much debate has taken place as to whether certain gases, such as chlorine, iodine, sulphuretted hydrogen, sulphurous acid, or the foetid vapours given off from catgut, gelatine, manure, and other trades, are or are not injurious to the health of the workmen, or persons living near the factories. In many cases the discussion is not closed, and fuller inquiries are necessary; but at present it seems as if these gases and foetid effluvia, in such proportions as they are met with about factories, are not proved to be unhealthy (though their innocuousness cannot be asserted), however disagreeable they may be. On the other hand, some really dangerous gases, such as carbonic oxide, are not offensive to the smell. Phosphorus-fumes escaping into the air have affected the jaw-bones of persons exposed to them; this happens now much less than formerly, though recent cases are on record, owing in large measure to the increased use of red or 'amorphous' phosphorus in the manufacture of lucifer-matches.

The spread of *infection by trade operations*, as of anthrax among wool-sorters, and of small-pox among paper-makers, has recently come to demand recognition.

There is one article, the sale of which gives rise directly and indirectly to a large amount of sickness, and although the matter is one which has not yet been seriously faced by our hygienists, it is clear that the sale of alcohol in all its forms requires better regulation, if the public health is to be regarded. Owing to peculiar social customs, and to the insufficient recognition of the immense amount of harm produced by excess of alcohol, the laws of this country have not only legalised the sale of a dangerous article of diet, but have actually encouraged the sale, until an evil so gigantic has been produced that no one has yet suggested a reasonable remedy. Yet the sale of alcohol is so

distinctly a source of preventable disease and of injury to the State, that it should be considered by those who have charge of the Public Health, and in some way must eventually be restricted (*see* ALCOHOLISM). One source of the error seems to be that alcohol is regarded by the State, not only as a source of revenue, but as the one indispensable article of 'refreshment.' There is, of course, no question that the public must be supplied with houses where they can obtain proper refreshments, such as meat, bread, vegetables, milk, coffee, tea, or other articles of the kind; and 'public-houses' were intended to supply articles of this description as well as the alcoholic liquids which enter into the ordinary diet of most people. Yet, unfortunately, a system has grown up by which our public-houses have become places where little else than alcoholic liquors are sold, and this system is defended on the ground that such liquids constitute 'refreshments.' The amount of temptation which has been put in the way of our working classes by the heedless multiplication, during the last fifty years, of these drinking-shops, when facilities are not afforded for obtaining the drinks which many persons would prefer to alcohol, accounts for much of the drunkenness which so deeply affects our national life, and injures the health of the people. A remedy ought to and must be found for this state of things, or else legislation will continue to present the absurd spectacle of raising up one huge mischief to the public health, while it is all anxiety to rid the community of every other.

9. *The Arrest and Control of Infectious Diseases.*—Although within recent years considerable progress has been made in our knowledge of the natural history of certain infectious diseases, it has to be accepted that the advances both of epidemiology and bacteriology have taught us that the scope of preventive medicine must needs be much extended if we are to trace the pathogenic bacteria to their several haunts. Were all infectious and contagious diseases confined to the human species, and, moreover, had they no existence outside the animal body, the problem of control would be relatively simple.

Unfortunately for administrative methods, we know that in respect, at any rate, of diseases such as tuberculosis, bubonic plague, anthrax, diphtheria, and many others, the specific organisms maintain their existence not only outside the human economy, but, in certain cases, also outside the animal kingdom. So, too, bacteriology, which is assisting administration by its ability to diagnose such diseases as have been associated with specific micro-organisms, has demonstrated that such specific bacilli may exist in apparently healthy persons, and that in those who have suffered from an attack of infectious disease, the specific organisms may exist long after the attack has apparently passed away. If, therefore, preventive medicine is to reap to the full the harvest of its labours, it must take note of all the conditions making for the survival and multiplication of pathogenic organisms.

Up to the present, the scope of preventive medicine has not been so extended as public opinion may presently demand. Efforts have been made by means of notification, isolation, vaccination, disinfection, and other sanitary measures, to control the spread of disease such as typhus fever, enteric

fever, scarlet fever, diphtheria, small-pox, cholera, plague, and certain others. The prevalence of some of these diseases has been much reduced, of others almost total extinction, for the time being, at least, has resulted; others again have not proved very amenable to the methods adopted. There are, however, other preventable diseases which tax our life and health, but which have not, as yet, been brought fully within the field of preventive medicine. Examples of such diseases are tuberculosis, measles, whooping-cough, and diarrhoea. With contagious diseases such as syphilis and gonorrhoea, the sanitarian of to-day would appear to have little concern, although considerations of the health of the race, and of economy, point to the necessity of further control.

A new branch of preventive medicine has, however, made great strides of late, and it may be that in preventive inoculation we may be able to do much during the advance and presence of certain infectious diseases. The duration of protection afforded by one attack of the diseases in question would hardly lead to the hope that such success will accrue as with vaccination against small-pox, but the indications with respect to diseases such as plague, cholera, enteric fever, &c., lead to the hope that attendants and others who have been exposed to infection may reap therefrom some protective or curative benefit.

The general principles on which the control of infectious diseases is based are—(1) The recognition of the places of origin and conditions of formation of the morbid agent, with recognition, also, of the processes to which it gives rise, alike in structures of the human body and in substances outside and independent of the body, with further question as to the nature of these substances, structures, or processes; the more fully these points are known, the more it is to be expected that the formation of the agent can be prevented or the agent be rendered harmless. (2) The recognition of the means of spread of the agent, after its first formation, that is, whether it spreads directly through the air, and, if so, through what distances and under what conditions; or whether it is carried in drinking-water or in food, or is transferred directly from one person to another: in proportion as this recognition has been gained, the carriage of the morbid agent may be stopped. (3) The early removal of the person affected from among the community, so that the risk of spreading in any way may be lessened. The system of compulsory notification of infectious disease which is now in operation throughout the country has rendered it possible for the local authorities of towns to keep infected children out of schools, and by other like means to limit the opportunities of infection; and also to secure the isolation of infectious cases as they arise or are imported into the town, and thus to diminish the opportunities of infection.

In the case of each of these diseases the preventive measures are different, and it is impossible here to go into so large a subject as the specialities of each. The measures include a continual supervision over the conditions of origin, introduction, and spread, as far as they are known.

Two points must, however, be noted more particularly. The first is the isolation of persons ill with any disease which directly or indirectly can spread from one person to another. In the crowded houses of towns some diseases, such as typhus,

scarlet fever, measles, and relapsing fever, may, at times, spread with great rapidity, and the only means that we at present possess for checking them are to remove the sick at the earliest moment from such houses, and to prevent persons ill with infectious diseases from exposing themselves in public places and conveyances. For this purpose Sanitary Authorities have powers to remove persons ill with infectious diseases to a proper hospital in special conveyances; to prevent sick persons frequenting public places or conveyances; and to deal with infected rooms, houses, and clothing. A hospital for infectious disease should therefore be provided in every locality, its advantages being (a) to minimise the risks of infection from invaded households to the remainder of the community; (b) to enable the sick to be separated from the healthy in the household, and the healthy to proceed with their occupation or education as the case may be; and (c) to provide better medical control and nursing than is practicable in the homes of the poor. These hospitals should be substantially built institutions, placed in attractive and easily accessible situations. Temporary structures of wood and iron are unsatisfactory. The hospital buildings should consist of (a) ward-blocks for the reception of the sick; (b) administrative blocks for the housing of the staff and stores, and (c) out-offices, such as laundry, mortuary, and disinfecting rooms. A discharge-block is a useful addition, as also are separate wards for the treatment of doubtful or very severe cases. The proportion of one bed per 1,000 of the population has been suggested as a working basis, and each bed should have at least 12 linear feet of wall-space, 144 square feet of floor-space, and 2,000 cubic feet of air-space. All surfaces should be as impervious as possible, in order to facilitate cleansing and disinfection. Cases of small-pox, owing to the peculiar tendency of this disease to spread to surrounding buildings, should not be treated in the same hospital with cases of other infectious diseases. Small-pox hospitals should be at a distance from aggregations of population. By means of the Public Health Acts and by the Isolation Hospitals Acts, 1893 and 1901, considerable power is conferred upon Local Sanitary Authorities to erect Isolation Hospitals either separately or jointly.

THE PREPARATION OF THE SICK-ROOM IN A CASE OF INFECTIOUS DISEASE.—All unnecessary furniture, carpets, curtains, bed-hangings, &c., should be removed from a room intended for the isolation of a sick person, a room at the top of the house, or one far removed from the other inmates, being selected. Such a room should be light and well ventilated, and a fire for the burning of infected rags, dressings, &c., and to assist ventilation, should, if practicable, be kept burning. A sheet moistened with some disinfectant may be hung over the door by way, mainly, of impressing upon the inmates the importance of isolation. The attendant upon the sick person should keep aloof from the other inmates and be provided with a room on the same floor and adjoining the sick-chamber. The nurse's room should be disinfected in common with the sick-room.

DISINFECTION.—Every Sanitary Authority should provide thoroughly reliable means for the disinfection of infected articles and dwellings, and should not, by the use of obsolete methods, convey a false sense of security more dangerous than recognised infection. For the disinfection of bulky articles,

such as bedding, carpets, and clothing, the use of one or another steam apparatus is essential. Hot air does not possess the penetrative or bactericidal qualities of steam, and is, moreover, liable to scorch and damage articles exposed to it. There are many forms of steam apparatus on the market, some of which use steam under high pressure, some under low pressure, and others with atmospheric pressure alone. There are certain differences with regard to penetrative power and the duration of time necessary to expose the articles, but it may be said that the cheaper low-pressure machine conforms for the most part to the requirements of efficient disinfection. 'Saturated' steam possesses considerable advantages over 'superheated' steam. But the method which has been adopted in certain machines in which hot air is merely moistened by steam is much inferior to those which rely, as do the apparatus referred to above, solely upon steam. Some articles, such as leather, furs, the binding of books, &c., are spoilt by the use of steam; for these, hot air is far less damaging, and many of the steam apparatus provide for the use of hot air as well as steam—indeed, hot air is in many cases essential for the drying of the articles moistened by steam. The disinfecting building should be divided into two parts, and the machine, which should be fitted with a door at each end, should be fixed in the dividing wall in such a manner that the only communication between the two chambers is through the disinfector. Portable steam disinfectors can now be procured. Although it is established that fresh air and sunlight, more particularly its actinic rays, speedily destroy the virus of certain infectious diseases, such as typhus fever, that of others, of which small-pox may be given as an example, appears to be much more resistant.

Free use should always be made of air and sunlight, but as, in the words of Koch, 'a good disinfectant must be rapid in its action as well as certain,' the use of chemical disinfectants is essential in cases where steam or boiling water cannot be brought into requisition, or the infected articles cannot be burnt. By the term *disinfectant* is understood a germicide (p. 412); the terms *antiseptic* and *deodorant* being applied, respectively, to such agencies as inhibit the growth of organisms or destroy effluvia.

The Disinfection of the Sick-room.—All bedding, carpets, clothes, &c., should be removed in canvas-bags moistened on the surface, and in properly appointed vans, to be disinfected by steam, while articles which can be safely boiled should be submitted to that process, and those which are valueless should be burnt. Before disinfecting or boiling all stains of blood or excreta should be removed by soaking the articles in cold water, otherwise the stains are liable to become fixed. The fumigation of rooms may be carried out by such gases as sulphurous acid, chlorine, or nitrous acid, &c. Sulphurous-acid gas may be generated by burning at least 3 lbs. of sulphur for every 1,000 cubic feet of space, by the use of Kingzett's sulphur-candles, or of sulphurous-acid gas liquefied by pressure, and contained in metal cylinders; the receptacle containing the sulphur being supported over a pail of water placed as high up the room as possible to aid the diffusion of the heavy gas. The air of the room should be previously moistened by steam. Bright metal surfaces are likely to be tarnished by the sulphurous gas, and hence they should, where

practicable, be washed over with a 2-per-cent. solution of carbolic acid, and removed from the room. The efficacy of sulphurous-acid fumigation is, however, somewhat open to doubt.

Within recent years formic aldehyde has been increasingly and successfully used as a gaseous disinfectant, and the aldehyde-vapours may be conveniently liberated from 'formalin,' a 40-per-cent. solution of formic aldehyde, by means of some specially devised apparatus such as that of Trillat, or the formaldehyde may be generated in the 'Alformant' lamp by heating tablets of paraformaldehyde together with the simultaneous production of water-vapour. Care must be taken to use sufficient tablets. In the disinfection of walls and other surfaces use may be made of sprayers, by means of which solutions such as perchloride of mercury, or 'formalin,' may be sprayed in finely atomised forms.

In the fumigation of an infected room, whatever be the gas used, all crevices and openings, including the fireplace, should be papered over, as also should the door by which the disinfectant gains exit. The room should be kept closed for some twenty-four hours, and at the termination of that time be thoroughly aerated; indeed, it is this aeration which is one of the advantages of the fumigation-method, and the additional measures now mentioned should never be neglected. The paper should be stripped from the walls, or, if the nature of the disease does not render this necessary, carefully rubbed over with bread 48 hours old, the ceilings lime-washed, and the floor and all suitable surfaces washed with a disinfecting solution; care being taken to select such disinfectants as will do no damage. Disinfecting solutions may be made of many substances, but perhaps the two most efficient are *acidulated* perchloride of mercury (1 in 1,000), and carbolic acid (5 to 10 per cent.). A useful solution recommended by the Local Government Board consists of: Mercuric chloride, 3 ss; hydrochloric acid 3 j; commercial aniline blue, gr. v; water, 3 gallons. But in the use of any such solution regard must be had to its 'working strength,' i.e. if perchloride of mercury 1 in 1,000 be in question, care must be taken not to make it 1 in 5,000 by adding it to the liquid to be disinfected. All disinfectants should be carefully labelled and kept in characteristic receptacles. In the disinfection of excreta, such as those from a case of enteric fever, it is desirable that the vessel receiving such excreta should contain some of the disinfecting solution, that they shall be intimately mixed therewith, and remain thus mixed for some time. Excreta may, if necessary, be burned after being freely mixed with sawdust and saturated with paraffin. In the case of a body dead of infectious disease, it is a useful precaution to envelop the corpse in sheets saturated with very strong solution of perchloride of mercury or carbolic acid.

10. *The Disposal of the Dead.*—Some 500,000 corpses have to be disposed of annually in England and Wales; and as Sir Henry Thompson has tersely expressed it, the problem is 'Given a dead body: to resolve it into carbonic acid, water, and ammonia, and the mineral elements rapidly, safely, and not unpleasantly.' Two points are involved in the disposal of the dead, both in towns and villages.

In this country, where so many families live in single rooms, and where the custom of keeping the

dead five or even six days before burial is usual, it constantly happens that a corpse is kept for days in the room where all the family life is carried on. As decomposition, especially in some diseases, commences early, it cannot be doubted that an unfavourable effect on health must be often produced. To avoid this detention, mortuary chapels ought to be constructed in all towns and villages, and to these all corpses should be removed from the houses of the poor within thirty-six hours after death.

Power has long been given to Local Authorities to provide mortuaries (in London such provision is compulsory) and to remove, when necessary, corpses from rooms where persons live and sleep. Very little has yet been done in this way, and England is in this respect far behind some of the Continental States.

The other point is the disposal of the corpse. The law of England now allows no burial-grounds in large cities, nor burial under churches, consequently cemeteries are provided at convenient distances from towns. These cemeteries ought to have a dry soil, so that the ground-water shall never rise high enough to wet the corpse or to float it up in the vault, as sometimes has happened; they should be as far from houses as practicable, and a limit of 200 feet is contemplated as a permissible minimum. There should be good drainage to permit a free flow of water, and the water should not run into any well or watercourse from which drinking-water is taken. It has, however, to be stated that the risk of the specific pollution of water-supplies by the drainage of cemeteries is not in practice a very serious one, inasmuch as the life of pathogenic organisms in the dead body is not of long duration. Klein has recently shown that the organism largely concerned with the destruction of the body is an anaerobic bacillus, which he terms *Bacillus sporogenes cadaveris*. The site should be well ventilated and well planted, so that the roots of the plants may absorb the decomposing matters. The kind of soil will, of course, depend on the locality; in many cases there is no choice; but if there be a choice a dry porous soil, such as sand or gravel, allowing free permeation by the air and the moisture necessary to allow the micro-organisms associated with the decomposition of organic matter to do their work, should be chosen. Clay-soils, unless very completely under-drained, are, as pointed out by Dr. C. W. Young in a recent report to the London County Council, ill-fitted for burial-grounds, by virtue of the fact that coffins in such a situation are more or less water-logged, and hence the aeration necessary for proper bacterial action is prevented. Decomposition is indefinitely delayed, the body undergoing saponification and passing into what is known as *adipocere* (p. 27). Moreover, clay-soil, more especially in the case of recently made graves, is liable to crack during dry weather, the cracks at times reaching down to the coffin itself, and affording passages to the gases of decomposition.

The practice of burial which at present obtains, although perhaps in a gradually diminishing degree, of preventing the access of earth to the body by means of coffins of a relatively imperishable character, and by burial in brick graves, is to be condemned. Perishable coffins should be used, and brick graves disallowed.

The present law prevents the burial of bodies of

adults within four feet of the surface; but the researches of bacteriology, as also the results of experience, rather indicate that more superficial burial should be legalised, and deep interment forbidden. A study of the flora of the soil at different depths points to the conclusion that bacteria, many of which are necessary to complete the process of decomposition, are not found below the first few feet of soil, and that deep interment leads to much delay in the resolution of the body.

The more superficial the burial of the animal body, the quicker the decomposition, and the bodies of animals merely covered with a layer of soil a foot thick have—save for their bones—entirely disappeared within a year. But extended observations are desirable before more superficial burial could be generally sanctioned in all conditions of soil and in thickly peopled cemeteries.

With the growth of cities there will come a time when it will be impracticable to accord to each corpse the freehold of its grave, and it will be necessary to adopt 'earth to earth' burial in order that the grave may more speedily be ready for another tenant, or to make a more extended use of the practice of cremation. There are now facilities for cremation at Manchester, Glasgow, Liverpool, Hull, and Darlington, while for London there is a crematorium at Woking, where, during 1900, 301 bodies were cremated. In a properly constructed apparatus a body of average dimensions can be converted into 3 lbs. of ashes in about one-and-a-half hours, and this without nuisance of any description. Obviously the process destroys all risks of the spread of infection, and the ashes are disposed of according to the wishes of the friends.

The chief objection to the process—apart from the weight which may be accorded to sentiment—is the risk that deaths from poison, violence, or criminal neglect may at times escape detection, and it may be contended that the opportunities of exhumation which at present exist act as a deterrent to criminal practices. But a better system of death-registration, together with the production of two medical certificates, and a voucher to the effect that the deceased desired cremation, should do much to reduce the risks of undetected crime.

11. The Supervision of Nuisances.—The word 'nuisance' has been adopted into sanitary law, without any fixed idea of the relation of the word to health or disease. 'Nuisances' are defined as being a number of enumerated conditions, some of which have to be 'injurious to health,' while others need not be injurious to health (but have only to be obnoxious), in order to bring them within the provisions of sanitary law. The confusion thus arising has been such that the primary object of sanitary legislation has sometimes been obscured. So much, indeed, has this been the case that, in comparatively recent times, it has been contended that a given condition which habitually does harm to health, but which had not yet, in the particular instance, succeeded in effecting actual disease, is not a 'nuisance injurious to health' within the meaning of a Public Health Act. With the view of facilitating the administration in this direction, the more recent acts have included under the term 'nuisances' not only those injurious to health but also those dangerous to health.

It is the duty of every Sanitary Authority to cause inspection to be made of their district to

discover 'nuisances,' as enumerated in sanitary statutes; and a certain procedure for the abatement of 'nuisances,' and for the prevention of their recurrence, is appointed by sanitary law. For the performance of these functions the Authority is required to appoint one or more Inspectors of Nuisances, to whose office certain powers are attached.

The work of nuisance-inspection in its everyday concern with conditions injurious to health cannot be properly performed without the constant and intimate relation of the Medical Officer of Health with the Inspector; and those districts are unquestionably best served as to sanitary inspection where the Authority has devolved on the Medical Officer the duty of instructing the Inspector and of supervising his work.

Specialities of Rural Sanitation.—While the objects to be gained by sanitary action are of course the same in town and country, the methods of attaining them in rural places must needs depart in some measure from those which are available in towns; and accordingly sanitary law in its administrative aspects recognises rural as distinguished from so-called 'urban' districts. Rural Sanitary Authorities, constituted in 1872, can now exercise considerable powers; and where properly set in action by their Medical Health Officers and Inspectors of Nuisances, a great effect is being gradually produced upon the rural labouring class, whose condition had long been neglected. The Rural Sanitary Authority may provide water for public use, may make public cisterns or baths, may protect water-courses, may construct sewers and dispose of sewage-matter; must take care that no closet or privy is a nuisance; may clean ditches and remove refuse; and may make regulations as to cellar-habitations and common lodging-houses. Much increased power of securing proper water-supply in the particular house within rural districts has been given, and there are few urban powers which cannot be acquired, if wanted, by any locality that can make a claim for consideration as a 'contributory place.' All powers possessed by urban Authorities as to trades, sale of unwholesome food, removal of nuisances, provision of mortuaries and hospitals for infectious diseases, are now also possessed by the rural authorities.

Although County Councils have in certain instances endeavoured to awaken the people from their lethargy, it must be admitted that comparatively little sanitary progress has been made in rural districts; and unquestionably there are some obstacles, inherent in the case, to any rapid progress. The difficulties arise from the houses in the rural districts being, in a great number of cases, old, dilapidated, unsuited for dwellings, and destitute of proper conveniences. When new houses are built, the Sanitary Authority can enforce certain provisions, though it has less control over building operations than is possessed by urban Authorities. In the case of houses already built, however, its power is, from circumstances, even more limited. There is very little money available for improvements; the poor-rates are already often heavy, and guardians hesitate to increase them. The small number of houses in villages also, in comparison with the outlay needful to supply sewers and water, renders the cost per head much greater than in towns. In addition to bad construction and dampness of houses, the most frequent sanitary defects of

rural places are as follows: The water is too often drawn from shallow wells or from small streams liable to pollution, or even from stagnant pools or ditches, and such supply is limited. Often there are no means for carrying away the dirty house-water, and it is thrown on the ground and soaks into the soil close to and under the cottage; the excreta are generally thrown into an ashpit near the house, or pass into a cesspit, from which they gradually soak, in such way as to pollute both ground and water. These difficulties, however, are being gradually overcome in districts which have secured the services of a first-class Officer of Health, who has sought, by means adapted to the special local conditions, to obtain the same sanitary advantages that are got in town districts by the use of more organised sanitary operations. Attempts are now being made to purify and then to guard the wells; to collect rain-water in proper tanks when other sources are not available; or to store the water collected from the surface-soil of some area secure from drainage, manuring, or like impurities. For the disposal of the slop-water, open or partially closed surface drains leading to ditches, or underground drains that shall allow the water to flow into the soil, and other plans, have been proposed. It is, on any plan, important—but especially if shallow wells or the surface soil is to furnish the drinking-water—to carry off to a distance all the slop-water by drains of some kind. For the removal of excreta (as sewers are frequently out of the question) a pail-system, with or without the use of dried earth or charcoal, according to circumstances, has to be used. If the cottages have gardens, then the simplest dry-earth plan, with proper storage and the subsequent digging into the gardens at intervals of not more than three or four weeks, seems to answer well; yet it is very difficult to get peasants to attend even to this simple matter, and at times, especially if they possess allotments, the excrement is stored up until the season for manuring the land arrives. A plan of conjoint action in the procuring, drying, and distributing the earth, and in the removal of the mixed earth and excreta, answers well when care is taken. In other cases a pail-system, with weekly removals, and without the use of earth or other appliance, has been employed, and may answer, as the manure has some value.

These seem at present the directions in which the opinions of Medical Officers of Health are tending where villages and labourers' cottages are concerned, and where larger works cannot be undertaken. The object, of course, is to obtain for the rural community by simple means, and at not too burdensome a rate, the sanitary requirements of pure soil, of pure air, and of pure drinking-water.

Houses.—Although to some extent the inside of a house is beyond the control of the public health authority, the law takes cognisance of the existence of *nuisance* inside, as well as outside, a house; and has special provisions for securing wholesomeness of habitation in the following cases:—

1. *Common lodging-houses* have been regulated since the great Public Health Act of 1848, the authors of which were evidently profoundly impressed with the great evils of overcrowding. The definition of a common lodging-house that is now usually accepted is that given by the law officers of

the Crown in 1853—namely, a house in which persons of the poorer class are received for short periods, and, though strangers to one another, are allowed to inhabit one common room. These houses are registered and inspected; the number of lodgers is fixed; and ventilation, cleanliness, and water-supply are now attended to. A certain cubic space per head in the sleeping-room of these houses is generally fixed by the authority, the minimum amount suggested by the present Model By-laws of the Local Government Board being 300 cubic feet for each adult, every two children under the age of ten years being counted as one lodger. If the rooms are not used exclusively as sleeping apartments, the models give 400 cubic feet for adults, and 200 cubic feet for children.

2. *Cellar-habitations.*—Since 1848 it has been unlawful to use cellar-habitations, unless they are in accord with certain conditions of space, height, window-area, and drainage. By the Public Health Act of 1875 it is made unlawful to use any cellar as a dwelling (that is, a place where any person passes a night) which has been built or rebuilt after the passing of the Act, or which was not lawfully in use when the Act was passed. A cellar is defined by various statutes to be any room of a house, the surface of whose floor is more than three feet below the surface of the footway of the adjoining street; and it has to fulfil a number of conditions before it can be legally inhabited. These conditions are, as regards London, somewhat modified by the Public Health (London) Act, 1891.

With the supervision that has been given to common lodging-houses during recent years, they have become much healthier and more decent habitations. Similarly the number of cellar-dwellings in our towns has much decreased, and the condition of those still used has notably improved.

3. '*Houses let in lodgings*,' or occupied by members of more than one family, are distinguished from common lodging-houses, and would more conveniently be termed *tenemented* houses. Sanitary authorities have various important powers conferred on them in respect of this large class of houses where two or more families live in the same house. But, for these powers to be brought into operation, the consent of the Local Government Board is required.

4. *Overcrowding.*—This condition, so dangerous to the health of the inmates, is to be regarded—no matter in what house it is observed, or whether the inmates be of the same family or not—as a nuisance, and is to be dealt with as such. The question arises, what is overcrowding, and usually the common lodging-house rules are taken, namely, an air-space of 300 cubic feet per head. But there is no legal definition, except in Scotland, where the General Improvement and Police Act of 1862 enacts that children under eight years of age shall have 150 cubic feet, and persons over that age 300. Obviously, the standard of space per person adopted as the minimum in the *bedrooms* of common lodging-houses, where the occupation is by night only, is too small for those who have to occupy the same room both by day and night, as is usually the case, where the question of overcrowding arises in the dwellings of the poor. It would be very desirable to raise the minimum (at all events for persons over ten years) to 400 cubic feet, and this is really little enough.

Causes of Unhealthiness of Houses.—

1. *Dampness*.—Dampness arises from a damp soil, water rising into walls, rain beating through walls or coming from a leaking roof, or blocked water-pipes. Paving, concreting, damp-proof courses, hollow walls, impermeable surfaces, &c., are the remedies. Damp houses are unhealthy, it would appear, by reason of the lowering of warmth conducing to catarrhal and rheumatic affections, and possibly by reason of increased bacterial life due to the constant presence of moisture.

2. *Excessive coldness of air from draughts, or from insufficient warming*.—Although an airy house is the healthiest, there may be excessive or irregular movement of air, so that strong currents are caused, with consequent undue skin-evaporation; whereby the body-temperature may be lower than is good for health, even if persons are well-clothed. The draughtiness is matter of construction, and is obviated by improvement in the methods of ventilation. Then, as to warming. Our present English use of fireplaces is both inefficient and expensive. It might have been expected that, in towns, this plan of warming houses would long ago have been abandoned for some more general use of hot-water or steam pipes. The supply of warmed fresh air is a very simple proceeding when these pipes are used, and thus not only can houses be better warmed, but better ventilated and less draughty. The greater convenience of a furnace serving to heat several houses is not at present appreciated in England; but here and there this consideration has probably contributed to the use of 'flats' in place of separate houses.

3. *Impurity of the air*.—This arises from the following conditions: from the air being drawn from the ground into the house, or passing over impure earth or deposits; or by the air in the house becoming contaminated by effluvia from closets and drain-pipes; from combustion; from respiration and skin-transpiration; from uncleanness of persons, clothes, walls, floors, and furniture.

Each of these conditions has to be examined into and rectified according to the usual principles laid down in works of hygiene. A few remarks may, however, be permitted on some of the headings.

The removal of respiratory impurities can only be accomplished by constantly removing the air of rooms and supplying fresh air. This is *ventilation*; and, if we include in the definition that the supply and removal of air shall be tolerably uniform, ventilation presents a mechanical problem of no little difficulty. The amount of air required for an adult, in order to keep the air relatively pure, has been fixed at 3,000 cubic feet per hour; or if the carbonic acid derived from respiration be taken as a measure of respiratory impurity, it should not exceed .2 per 1,000 volumes of air. Practically, the amount most persons get is not more than 600 to 1,200 cubic feet per hour, if so much, and the air of their rooms smells fusty from organic effluvia. In cold times of the year, the entering air must be warmed, if the change is to be so rapid as is implied in the supply of 3,000 cubic feet of air per hour, equivalent to a change of air in the air-space three, four, or even five times per hour. When warmed to nearly the temperature of the surface of the body (80° to 90° F.) considerable movement of air is borne without difficulty, but if the temperature be much lower a correspondingly slighter movement is felt. Ventilation in this climate is therefore inextricably

mixed up with warming, and thorough ventilation of our rooms is impossible so long as we trust to radiant heat alone for warmth. The problem, therefore, which engineers have to solve in warming and ventilating our rooms is, what is the cheapest and most constant plan of introducing warm air into our houses in cold weather; the conditions of the problem being a supply of 3,000 cubic feet per head per hour, at a rate of movement imperceptible to the feelings of the persons in the room.

The second point is connected with the impurity of the air from drains. The first thing is to be certain that the air of the house-drain is so thoroughly disconnected from the air of the public sewers that no reflux from them is possible; and therefore, that, if there is any drain-air polluting the atmosphere of the house, it is not the air of the common sewer. That point having been settled, it will follow that drain-smell in the house must come either from the ground or from the house-pipes or closets themselves. If from the ground, there is probably (if the ground itself be clean, or if the smell be of new production) a leaky drain-pipe somewhere, and the air is penetrating through the leakage and is drawn into the house. If not from the ground, the smell may be from some pipe in the house; this arises from imperfect junction, especially when metal pipes are joined on to earthenware, or from the pin-hole eating-away of metal pipes. Or a drain-pipe may be choked (generally through 'settling' at a joint occurring in an ill-laid and badly bedded pipe), and decomposition be going on in its retained contents. Or there may be a clogged or imperfect trap, with the water either sucked out of it or becoming thoroughly charged with fetid effluvia. In the latter cases, there is a presumption that the ventilation of the house-drain is not what it should be. Every house should have a plan of its drainage, so as to facilitate the search for a broken pipe.

In order to detect any of these conditions it is necessary that builders should alter their habits in regard to house-plumbing arrangements. At present they try to conceal everything, so that, without pulling a house to pieces, it is impossible to examine if pipes and traps are in order. Instead of this, every pipe above the ground should be kept out of walls; and if cased with wood, the case should be merely bolted, and not nailed. If a drain must be carried under a house, it should have inspection-chambers built upon it at every change of direction, so that the drain, which runs in a straight line from one manhole chamber to the next can be inspected and cleared of obstructions or deposit without breaking into it. The sewage and foul-water arrangements of our houses will never be satisfactory till these matters are attended to, and till the examination of every pipe about the house can be made without difficulty, and clogging or leakage of air or water from pipes can be readily detected.

In closets, the chief points of leakage are in the more horizontal pipes and in the traps. In all cases the soil-pipe should be ventilated by pipes carried to the open air at some points away from windows.

Another matter to be guarded against, whether there be drain-smell or not about a house, is the immediate opening of the cistern overflow-pipe, or of the usual rain-water pipe, into the sewer or house-drain. A common practice, until recently, was to connect them directly with the house-drain,

perhaps with an S-trap at the foot of each pipe, these traps, however, being usually dry. Then sewer-air passes up and enters the cistern, or into rooms which happen to be near the top of the rain-water pipe. All these pipes should open in free air over a grating; and if every householder would insist on the builder attending to these matters, the chances of inflow of sewer-air into houses would be much lessened.

Another, third, point of importance is the way in which the products of gas-combustion are allowed to pass into the air of rooms. Nothing can be worse than the usual arrangement; and, as gas-lights might be made a valuable means of ventilation if tubes were arranged to carry off the burnt gas, the present arrangement of chandeliers is not only hurtful, but involves an ignorant waste of useful force.

4. *Impurity of the water.*—Water delivered to a house may become impure on the premises, usually from uncleaned uncovered cisterns, absorption of air from drains by the surface of the water, and sometimes by more direct leakage from pipes into cisterns. Lead may also be taken up. The remedies for these conditions are obvious. Reference has also been made to extensive epidemics of enteric fever due to the insuction of matter from sewers into public water-pipes, through fissures in those pipes while running full. It is possible that obscure cases of disease in particular houses are sometimes due to an unsuspected pollution by similar means of the water-service of the house.

5. *Impurities from uncleanness of the house.*—Walls and ceilings all absorb impurities which are given out again to the air, and often become highly impregnated with organic matters. The chinks of floors allow matters to collect below them, and then impure air rises into the room.

The custom of re-papering walls without removing the old paper, the decomposition of paste and paper on damp walls, and the use of arsenical pigments, may give rise to impurities of one and another kind in the air of houses. In the houses of the poor which are not regularly whitewashed, the half-crumbling plaster is often highly charged with animal material.

These matters are to be avoided by original good construction and by constant cleanliness. It is a great desideratum to make walls of impermeable material, so that they may be washed without difficulty.

If these various points, which are really questions of purity of air and water, and of temperature and movement of air, are properly dealt with, houses will be healthy. These are conditions which are not difficult to secure if they are clearly understood and if their importance is not underrated. The great point is to have the house-air pure, so as in no way to injure or depress the great function of respiration.

While we look to the Municipality or Local Sanitary Authority to keep the outer air pure, the task of doing the same for the house-air must necessarily fall on the inhabitants of the house.

Duties of the Medical Officer of Health.—It behoves the residents of every district to assist the Sanitary Officers to the best of their ability; and to aid towards such endeavours, this article may usefully conclude by an enumeration of the duties which are imposed upon the Medical Officer of Health for districts in England. They are

extracted from an order of the Local Government Board, dated 1891.

‘The following shall be the duties of the Medical Officer of Health in respect of the district for which he is appointed :—

‘1. He shall inform himself as far as practicable respecting all influences affecting or threatening to affect injuriously the public health within the district.

‘2. He shall inquire into and ascertain by such means as are at his disposal the causes, origin, and distribution of diseases within the district, and ascertain to what extent the same have depended on conditions capable of removal or mitigation.

‘3. He shall by inspection of the district, both systematically at certain periods, and at intervals as occasion may require, keep himself informed of the conditions injurious to health existing therein.

‘4. He shall be prepared to advise the Sanitary Authority on all matters affecting the health of the district, and on all sanitary points involved in the action of the Sanitary Authority; and in cases requiring it, he shall certify, for the guidance of the Sanitary Authority or of the Justices, as to any matter in respect of which the certificate of a Medical Officer of Health or a Medical Practitioner is required as the basis or in aid of sanitary action.

‘5. He shall advise the Sanitary Authority on any question relating to health involved in the framing and subsequent working of such by-laws and regulations as they may have power to make, and as to the adoption by the Sanitary Authority of the Infectious Disease (Prevention) Act, 1890, or of any section or sections of such Act.

‘6. On receiving information of the outbreak of any contagious, infectious, or epidemic disease of a dangerous character within the district, he shall visit without delay the spot where the outbreak has occurred, and inquire into the causes and circumstances of such outbreak; and in case he is not satisfied that all due precautions are being taken, he shall advise the persons competent to act as to the measures which may appear to him to be required to prevent the extension of the disease, and take such measures for the prevention of disease as he is legally authorised to take under any statute in force in the district, or by any resolution of the Sanitary Authority.

‘7. Subject to the instructions of the Sanitary Authority, he shall direct or superintend the work of the Inspector of Nuisances in the way and to the extent that the Sanitary Authority shall approve, and on receiving information from the Inspector of Nuisances that his intervention is required in consequence of the existence of any nuisance injurious to health, or of any overcrowding in a house, he shall, as early as practicable, take such steps as he is legally authorised to take under any statute in force in the district, or by any resolution of the Sanitary Authority, as the circumstances of the case may justify and require.

‘8. In any case in which it may appear to him to be necessary or advisable, or in which he shall be so directed by the Sanitary Authority, he shall himself inspect and examine any animal, carcase, meat, poultry, game, flesh, fish, fruit, vegetables, corn, bread, flour, or milk, and any other article to which the provisions of the Public Health Act, 1875, in this behalf shall apply, exposed for sale, or deposited for the purpose of sale or of preparation for sale, and intended for the food of man,

which is deemed to be diseased, or unsound, or unwholesome, or unfit for the food of man; and if he finds that such animal or article is diseased, or unsound, or unwholesome, or unfit for the food of man, he shall give such directions as may be necessary for causing the same to be dealt with by a Justice according to the provisions of the statutes applicable to the case.

'9. He shall perform all the duties imposed upon him by any by-laws and regulations of the Sanitary Authority, duly confirmed where confirmation is legally required, in respect of any matter affecting the public health, and touching which they are authorised to frame by-laws and regulations.

'10. He shall inquire into any offensive process of trade carried on within the district, and report on the appropriate means for the prevention of any nuisance or injury to health therefrom.

'11. He shall attend at the office of the Sanitary Authority or at some other appointed place, at such stated times as they may direct.

'12. He shall from time to time report in writing to the Sanitary Authority his proceedings, and the measures which may require to be adopted for the improvement or protection of the public health in the district. He shall in like manner report with respect to the sickness and mortality within the district, so far as he has been enabled to ascertain the same.

'13. He shall keep a book or books, to be provided by the Sanitary Authority, in which he shall make an entry of his visits, and notes of his observations and instructions thereon, and also the date and nature of applications made to him, the date and result of the action taken thereon, and of any action taken on previous reports; and shall produce such book or books, whenever required, to the Sanitary Authority.

'14. He shall also make an annual report to the Sanitary Authority, up to the end of December in each year, comprising a summary of the action taken, or which he has advised the Sanitary Authority to take, during the year, for preventing the spread of disease, and an account of the sanitary state of his district generally at the end of the year. The report shall also contain an account of the inquiries which he has made as to conditions injurious to health existing in the district, and of the proceedings in which he has taken part or advised under any statute, so far as such proceedings relate to those conditions; and also an account of the supervision exercised by him, or on his advice, for sanitary purposes over places and houses that the Sanitary Authority have power to regulate, with the nature and results of any proceedings which may have been so required and taken in respect of the same during the year. The report shall also record the action taken by him, or on his advice, during the year, in regard to offensive trades, to dairies, cow-sheds, and milk-shops, and to factories and workshops. The report shall also contain tabular statements (on forms to be supplied by us [Local Government Board], or to the like effect) of the sickness and mortality within the district, classified according to diseases, ages, and localities.

'Provided that, if the Medical Officer of Health shall cease to hold office before the thirty-first day of December in any year, he shall make the like report for so much of the year as shall have expired when he ceases to hold office.

'15. He shall give immediate information to us of any outbreak of dangerous epidemic disease within the district, and shall transmit to us a copy of each annual report and of any special report. He shall make a special report to us of the grounds of any advice which he may give to the Sanitary Authority with a view to their requiring the closure of any school or schools, in pursuance of the Code of Regulations approved by the Education Department, and for the time being in force.

'16. At the same time that he gives information to us of an outbreak of an infectious disease, or transmits to us a copy of his annual report or of any special report, he shall give the like information or transmit a copy of such report to the County Council or County Councils of the county or counties within which his district may be situated.

'17. In matters not specifically provided for in this Order he shall observe and execute any instructions issued by us, and the lawful orders and directions of the Sanitary Authority applicable to his office.

'18. Whenever we shall make regulations for all or any of the purposes specified in section 134 of the Public Health Act, 1875, and shall declare the regulations so made to be in force within any area comprising the whole or any part of the district, he shall observe such regulations, so far as the same relate to or concern his office.'

These duties have been considerably augmented by the provisions of subsequent Acts of Parliament; such, for instance, as the Factory and Workshops Acts.

H. TIMBRELL BULSTRODE.

PUERILE (*puer*, a boy).—This word is associated in medicine with the respiratory murmur when it is exaggerated, possessing the characters heard over the lungs in a healthy child. See PHYSICAL EXAMINATION.

PUERPERAL DISEASES.—The diseases associated with parturition, which fall for consideration in the present article, are: (1) Puerperal Convulsions; (2) Puerperal Fever; (3) Puerperal Peritonitis; and (4) Puerperal Embolism and Thrombosis of the Pulmonary Artery. Certain other pathological conditions of equal importance, occurring during the puerperal state, are more conveniently discussed under their several special names. See PELVIC ABSCESS; PELVIC CELLULITIS; PELVIC PERITONITIS; and PHLEGMASIA DOLENS. Puerperal insanity is described in the article INSANITY, Varieties of.

1. Puerperal Convulsions.—SYNON.: Puerperal Eclampsia; *Eclampsia Gravidarum*, *Parturientium*, *vel Puerperarum*; Fr. *Convulsions des Femmes Enceintes et en Couche*; Ger. *Eklampsie in der Schwangerschaft und im Wochenbett*.

DEFINITION.—A peculiar kind of epileptiform convulsions, characterised by loss of consciousness and sensibility, together with tonic and clonic spasms; occurring in the later months of pregnancy, during labour or after delivery, and directly connected with these states; and causing great danger to the lives of both mother and child.

ÆTIOLOGY.—Two conditions which favour the occurrence of eclampsia are: (1) a first pregnancy, and (2) the presence of twins *in utero*. The pathology of the condition is by no means certain, and it is therefore not surprising to find that numerous theories have been brought forward to explain its

causation. The frequent association of this disorder with albuminuria had till lately given rise to the belief that it is the result of uræmia; but numerous cases have been observed in which albumen was present in the urine in large quantity without convulsions occurring. Cases too have been recorded in which no albumen was found in the urine, but it is probably present in every case, although owing to its rapid appearance and disappearance it may not be found at the particular time the urine is examined.

The toxæmic theory of eclampsia is the one that has most supporters at the present day. It has been shown that in women suffering from eclampsia the toxicity of the blood-serum is increased, and the toxicity of the urine is proportionately diminished, and it has also been shown that in these patients there is a period after the cessation of the fits when the toxicity of the urine is again increased. These facts tend to prove that there is an accumulation of certain toxins in the blood of eclamptic patients. During pregnancy the presence of the fetus calls for increased metabolism on the part of the maternal tissues, and this is of necessity accompanied by an increased production of excrementitious matters. In a perfectly healthy woman the liver and kidneys are capable of dealing with such an excess. If, however, for some reason the functions of the liver are, for the time being, interfered with, it may be assumed that various toxic matters will pass into the circulation unchanged and with their virulence undiminished. The presence of albumen in the urine may be regarded as one result of the action of some such poison upon the renal epithelium, and the accompanying interference with the excretory functions of the kidneys will lead to a further accumulation of these toxic bodies in the blood of the patient. A point will at length be reached at which there is present a sufficient quantity of these poisons in the blood to set up convulsions and coma. Such a view is supported by the fact that the changes in the kidneys are frequently similar to those seen in patients dying of acute infectious diseases, and that Bouchard has isolated from the urine substances capable of producing convulsions and coma when injected into animals. The exact nature of the poisons, the seat of their formation, and whether this is in every case within the body, is uncertain. That the poison is not urea has been shown by Bradford, who has demonstrated that there is no excess of this substance in the blood or in the tissues of patients dying of eclampsia. It has been suggested that it is the product of the growth of an organism, but there is not sufficient evidence to support such a view.

SYMPTOMS.—Puerperal eclampsia is happily not of common occurrence, its estimated frequency being about once in 500 cases. The studies of Goldberg show that the disease appears first in about 21 per cent. of the cases during pregnancy, in 56 per cent. during labour, and in 22 per cent. during the puerperium. Occasionally the outbreak occurs without warning, but usually there are premonitory symptoms. The most frequent of these is oedema of the feet and legs, which may extend to the vulva, abdomen, face, and upper extremities, and should at once suggest an examination of the urine, which will almost invariably be found to contain albumen in more or less quantity, with a diminution in the total quantity of urine and in the amount of urea which should be excreted. The

specific gravity is high, and sometimes casts may be found. Other premonitory symptoms are headache, generally frontal, but occasionally limited to one side; epigastric pain or vertigo; loss of memory; and derangements of vision, such as amblyopia, diplopia, and flashes of light before the eyes.

When the convulsive seizure occurs it cannot be mistaken. The eyes first become fixed, and rapid contraction of the muscles of the face occurs, with rolling of the eyeballs, the pupils being lost under the upper eyelids. The face becomes turned first towards one shoulder, then towards the other. The convulsions rapidly extend to the other parts of the body; after a short period of tonic contraction violent clonic spasms occur. The face becomes livid, the tongue is protruded, and, if care be not taken, it is lacerated by the teeth, colouring the frothy saliva which is emitted at the angles of the mouth. The thumbs become clenched in the palms, and violent jerkings of the arms occur, while the muscles of the face give rise to a variety of contortions. Sometimes involuntary evacuations of the bladder and rectum occur during the fit. There is total loss of consciousness and sensation. After a few minutes the symptoms gradually subside; a longer interval occurs between the clonic muscular contractions; the face loses its lividity; and the breathing becomes more tranquil. When the first fit has passed off, the patient may recover her consciousness; but if another occur with rapidity, and very little time elapse between the paroxysms, complete coma resulting in death may soon supervene. Where there is a considerable time between the attacks, it may be many hours or days before consciousness is restored, and recovery takes place. A remarkable feature of this disorder is that when the patient becomes sensible, and is restored to health, she has invariably no recollection of what occurred, not only during her illness, but for some time preceding the fits.

PROGNOSIS.—This depends upon the severity and frequency of the paroxysms. It is generally considered that about 20 per cent. of the mothers die. The earlier the convulsions appear in labour, the more unfavourable the prognosis. The longer the labour and the more difficult the delivery, the deeper is the coma and the less the prospect of recovery. In about 30 to 40 per cent. of the cases in which the fits begin during labour, they persist after delivery. In cases beginning after the child is born, the mortality is not more than 12 per cent.

MORBID ANATOMY.—Almost any variety of change may be found in the kidneys. In many cases the renal epithelium shows signs of acute degeneration, such as is seen in patients dying from septicæmia. In the liver the most frequent condition is the occurrence of hæmorrhages, with degeneration of the liver-cells, and the formation of minute necrotic areas. Hæmorrhages, no doubt due to the convulsions, are often found in the lungs and the spleen.

TREATMENT.—Recognising that 'prevention is better than cure,' we must first consider what is best to be done when premonitory symptoms arise. The occasional examination of the urine of pregnant women should always be carried out, and is urgently called for whenever oedema of the feet or other parts is noticed. Slight traces of albumen are not infrequent, and are of no importance; but persistent albuminuria to any considerable amount

requires most careful watching and treatment, and the amount of urea passed in the twenty-four hours should be ascertained. A strict milk-diet, and the administration of diuretics and iron, with hydragogue purgatives, should be tried; but, if little or no improvement takes place, premature labour should be induced, since it gives very good results in these cases. When convulsions occur, there are two objects to be aimed at—the lessening of the irritability of the nervous system, and the elimination of the poison from the body. Since the latter has possibly an intestinal origin, free purging should be at once procured by the administration of croton oil. The patient is to be placed in a hot-air- or vapour-bath, and kept in this until she perspires freely. If the power of swallowing is present, some hot brandy and water may be administered, but if she be unconscious, no attempt should be made to give stimulants or food except through a stomach-tube. Venesection to the extent of thirteen to fifteen ounces of blood may be practised if the patient be not very anæmic or feeble (*see* VENESECTION). Normal saline solution (*see* SALINE SOLUTION, Infusion of) should then be introduced into the vein to the amount of several pints. The object of this ‘treatment’ is to get rid of the venesection of a certain amount of the poison, and by the introduction of the saline solution to so dilute the remainder as to render its elimination by the skin and kidneys more probable. The combination of ‘infusion’ with venesection meets many of the objections urged against the employment of the latter alone. The results of this mode of treatment are encouraging. To lessen the irritability of the nervous system, the best drug is morphine. Half a grain may be given hypodermically, and if the fits continue or the patient remains restless, further doses of $\frac{1}{4}$ gr. may be given every half-hour or so. Veit has administered in this way as much as 3 grs. of morphine within four to seven hours.

If the patient be not comatose or under the influence of the morphine, chloroform should be given while any manipulations are being carried out, or until the morphine has had time to produce its effect, but the prolonged administration of chloroform should be avoided, as it is not without danger.

When the patient is completely comatose and no fits are occurring, neither morphine nor chloroform need be given. In such cases reliance must be placed upon baths, venesection and ‘infusion.’ If the patient be so feeble or anæmic as to render the practice of venesection too dangerous, it must be omitted, but even in such a case benefit may be derived from the subcutaneous injection of saline fluid (*see* HYPODERMIC MEDICATION). Pilocarpine should be avoided, and morphine acts more certainly and speedily than chloral hydrate or potassium bromide. These latter drugs, however, given either by the mouth or by the bowel, are used by many practitioners who are not satisfied as to the safety of administering morphine in these cases.

The patient must be guarded against injuring herself during the fits. Labour should be allowed to proceed naturally, only such aid being given as can be rendered without unduly disturbing the patient. *Accouchement forcé* should never be practised. For the convulsions occurring after delivery the administration of morphine is usually sufficient.

2. **Puerperal Fever.**—SYNON.: Child bed Fever; Puerperal Septicæmia; Surgical Septicæmia; Fr. *Fièvre Puerpérale*; Ger. *Puerperal-fieber*; *Kindbettfieber*.

DEFINITION.—A continued fever of a contagious character, occurring in connection with childbirth.

PATHOLOGY AND ETIOLOGY.—Numerous theories and hypotheses have been suggested in regard to the nature and relations of puerperal fever, or puerperal septic infection, but it is now generally accepted that it is the same disease as surgical septicæmia, occurring in a puerperal patient. It must be distinguished from *sapremia* or *septic intoxication*, in which the patient suffers from poisoning by the absorption of the chemical products of putrefaction, the decomposing material being generally retained portions of membranes or placenta. Puerperal infection is due to the entrance into the patient's body of certain organisms and their multiplication in the blood, in the tissues, or in both.

The organism that most commonly causes the disease is the *Streptococcus pyogenes*, but in some of the cases other organisms have been found either alone or in association with the streptococcus. The commonest of these are the *Staphylococcus albus*, *S. aureus* and *S. citreus*, the *Gonococcus*, the *Bacillus coli communis*, the *Pneumococcus* and the *diphtheria-bacillus*. The genital canal of the healthy woman may be considered as containing no pathogenic or disease-producing organisms. It is possible, however, that under certain conditions the non-pathogenic organisms which are present may attain an increased virulence, and by invading the tissues lead to a general infective process. Such an explanation may account for the very uncommon cases of so-called autogenetic septicæmia occurring in patients who have not been exposed to any possibility of local infection. In a few cases the poison is derived from some local source such as the rupture during labour of a suppurating ovarian cyst or of a pyosalpinx. It is quite certain, however, that in the great majority of cases the patient is infected by the fingers of the attendants, by the instruments used, or by contact with infected fomites.

The virus usually enters the body through the placental site, but it may find its way through any of the lesions in the cervix, vagina, or perineum resulting from labour. The actual sources of the contagion are other cases of puerperal fever, infected wounds, cases of erysipelas, the throat in cases of scarlet fever, the *post-mortem* room and the dissecting room. A patient who is suffering from *sapremia* or *septic intoxication* is very likely to acquire general septic infection, and the disease is also predisposed to by extensive injuries to the soft parts, by exhaustion from prolonged labour or hæmorrhage, by grave emotional disturbances, and by exposure either to cold or to poisoning by sewer-gas. Besides her liability to these general predisposing causes, the parturient woman is specially prone to septic infection because of the deep situation of the wounds in the genital canal, and because of the excessive development of the veins and lymphatics in the wall of the pregnant uterus.

ANATOMICAL CHARACTERS.—After its introduction at some raw surface the virus may invade the body generally either through the veins or the lymphatics, giving rise to the so-called venous or

lymphatic forms of septicæmia. In very acute cases death may occur without the development or any local lesion. More commonly, however, acute inflammatory affections of some part of the genital tract are present. If the vagina be involved, its mucous membrane is red, congested and swollen, and ulcers covered with diphtheritic membrane may be present either in the vaginal walls or in the cervix. The walls of the uterus are soft and oedematous, the mucous membrane is often sloughing, and covered with greyish-red debris, numerous cocci are present in the mucous membrane and the muscular tissue. They may invade the peritoneal cavity though the Fallopian tubes, setting up salpingitis and peritonitis, or directly through the lymphatics and veins of the uterine wall. Small miliary abscesses are often met with, due to the presence of pus in the veins and lymphatic spaces. In the very acute cases of infection by the streptococcus the mucous membrane of the uterus is very little altered, and may feel quite smooth to the examining finger. The pelvic cellular tissue frequently becomes affected. The peritoneum is almost certainly involved, and a coagulated exudation or fluid pus is found in the folds around the uterus, in Douglas's pouch and elsewhere, or the peritonitis may be diffuse, and associated with pleurisy and pericarditis and effusions into the joints. If the absorption has taken place through the veins, broken-down thrombi will be found, and from these embolic pyæmic foci and abscesses may occur in the lungs; the spleen is almost always enlarged, and embolic infarcts may be found in it, as well as in the kidneys. Endocarditis and meningitis may arise; and sometimes, though rarely, septic panophthalmitis and destruction of the eyeball.

SYMPTOMS.—In no disease do the symptoms vary more than in puerperal fever, depending upon the violence of the fever, and the localities attacked by the poison. The fever generally originates within four days after delivery, though sometimes later, very rarely after the fifth day. Occasionally there is insomnia and restlessness preceding the rise of temperature, or a feeling of depression, with headache; or often the first symptom is a rigor. In cases of *sapremia* or septic intoxication the temperature may be only slightly elevated to 101° or 102° . In most of the acute cases of septic infection there is a sudden, rapid rise, accompanied by a rigor, the temperature reaching 104° or 105° . It may be maintained at a uniformly high level, the initial rigor not being repeated, or marked oscillation and frequent rigors may occur. These usually accompany the formation of secondary pyæmic foci. The pulse becomes rapid and feeble, 130 or more per minute. It is often a better guide to the patient's condition than the temperature, since the latter in some of the most fatal cases may be hardly above the normal, or even below it. The skin is generally hot and dry, and fugacious scarlatiniform rashes may appear. Vomiting frequently occurs early, the ejecta being like coffee-grounds, and of a peculiar odour. Diarrhoea is often very troublesome, and the evacuations are horribly fetid. The tongue soon becomes coated with a heavy fur, later on becoming dry and rough; and sordes appear on the lips. There is often acute pain in the abdomen, with tenderness and swelling; but peritonitis with effusion may occur without any of these symptoms. Sometimes the tender, sub-involved uterus can be felt in the hypogastrium.

The lochia are generally suppressed, and the secretion of milk arrested, though sometimes the mammae are hard and painful. If present, the lochia are in some cases very offensive and in other cases quite free from odour.

The spleen is frequently enlarged and easily felt. As a rule, the intellect is unimpaired, though low muttering delirium frequently precedes death. The breathing is short and hurried. Pneumonia, pleurisy, or pericarditis occasionally ensues. Jaundice or albuminuria may be present. The joints may swell and suppurate; and abscesses may form in any part of the body.

COURSE AND TERMINATIONS.—The disease, if of the lymphatic variety, begins very early, and generally runs a rapid course, terminating fatally within a week. The pulse becomes more and more rapid and feeble; the breathing more hurried and panting; tympanites sets in; a cold clammy sweat breaks out; finally hiccough, subsultus, and low muttering delirium come on, with frequently incessant vomiting; and the patient sinks from exhaustion. The venous variety generally shows itself much later, is of much longer duration, and is associated with erratic rigors, and an absence of abdominal distension.

DIAGNOSIS.—A marked rise of temperature within the first week after delivery is usually due to septic infection; other possible causes are constipation, mastitis, the onset of some acute illness, such as pneumonia, or emotional disturbance in a neurotic patient.

The diagnosis between septic intoxication and septic infection can usually be made by discovering retained and decomposing portions of placenta or membranes *in utero*, and by the fall of temperature and rapid recovery of the patient which follow their removal.

TREATMENT.—(1) *Prophylactic.*—This is of the greatest importance. How successfully it can be carried out is shown by the results obtained at the present day in Lying-in Hospitals. In these institutions, which, in the pre-antiseptic days, were hot-beds of puerperal fever, and exhibited in many instances a mortality so excessive that they had to be closed, under modern conditions it is the rarest possible thing for a case of septic infection to occur. In view of the fact that the poison is almost always introduced into the patient's body from without, it is necessary to ensure that everything that comes in contact with the patient is *surgically clean*. At the commencement of labour the woman's vulva, thighs, buttocks, and the lower part of the abdomen must be carefully washed with soap and water. Before any internal examination is made the vulva must be sponged over with some antiseptic solution, such as 1 in 1000 perchloride of mercury. The doctor and the nurse on every occasion before touching the patient's genitalia must thoroughly wash their hands with soap and water, and then scrub them in an antiseptic solution, such as 1 in 1000 perchloride of mercury. Sponges should never be used, wool-swabs wrung out of an antiseptic lotion being employed instead. All instruments, which should be constructed entirely of metal or glass, must be sterilised before use by boiling. Glycerine of perchloride of mercury 1 in 1000, or carbolised vaseline, should be used as a lubricant. Failing either of these the finger may be anointed with a piece of soap the outer layer of which has been washed

off. Cold cream, or lard, or dirty vaseline must not be used. Vaginal examination must be made as infrequently as possible, abdominal palpation being practised instead. Since the genital canal of the healthy woman contains, as a rule, no disease-producing organisms, vaginal douches need not be employed either before, during, or after labour, except under special circumstances. If, for instance, there be a profuse purulent discharge, and a suspicion of gonorrhœa, a douche should be given to diminish the risk of ophthalmia neonatorum. Special care must be taken that no portion of placenta or membranes be retained *in utero*, and if this occur it must at once be removed with the finger. Attention should be paid to the patient's general health so that she may enter upon labour under the most favourable conditions. The lying-in room should be roomy and well-ventilated, it must not communicate with a water-closet or a bath-room, and any fixed washing-basins in it must have their pipes plugged, and must not be used. If it be necessary for the practitioner to attend a case of midwifery when he has recently been in contact with a case of septic infection, he should, after the most thorough cleansing of his hands, put on a pair of thin india-rubber gloves which can be sterilised by soaking in a strong antiseptic solution. After delivery the discharge should be absorbed by some form of antiseptic pads, and each time that they are changed the vulva should be cleansed with some efficient antiseptic lotion, the nurse observing the same precautions as during labour.

(2) *General*.—If the temperature rises during the first week after delivery, and no condition other than some septic mischief can be found to account for the fever, the uterus should be explored with the finger, especially if there be any doubt as to whether the whole of the placenta and membranes have come away. If retained portions of placenta or membranes be found, they must be scraped away with the finger, and the uterine cavity washed out with some quarts of a weak antiseptic solution, such as creoline half a drachm to the pint. If an offensive discharge continues, the intra-uterine douche may be repeated; but this should not be done if the uterus is found to be empty and its mucous membrane smooth. Curettage of the recently pregnant uterus should not be practised; a pair of ovum-forceps may be used with care to separate any piece of placenta so adherent that it cannot be removed by the finger. A careful examination must be made of the vulva and vagina, and any ulcers present swabbed over with a 1 in 500 solution of perchloride of mercury. Vaginal douches, if necessary, may be given two or three times a day.

Further treatment must be directed towards keeping up the patient's strength so as to enable her to combat the poison. For this good nursing is essential. It is of the utmost importance that nutritious fluid food, and as much alcohol as may be necessary, be administered in small quantities at frequent intervals. If vomiting prevent food being given by the mouth, resource must be had to nutrient enemata and suppositories. Pain and sleeplessness may be treated with opium or morphine. Abdominal pain is best relieved by belladonna fomentations, or poultices sprinkled with laudanum, and abdominal distension by turpentine enemata, or the introduction of the rectal tube. The most useful drugs for internal administration are quinine, Warburg's tincture, ammonium carbo-

nate, turpentine and strychnine. The latter should be given in large doses. Inhalation of oxygen may be necessary. The daily transfusion of half a pint of 'normal' saline fluid under each breast appears to be of service and does no harm (*see* SALINE SOLUTION, Infusion of). It assists the circulation, and the action of the skin and kidneys, and so no doubt helps to eliminate the poison. If the temperature rise to a dangerous degree, an ice-cap may be applied to the head, or the patient cold-sponged or enveloped in a cold pack. The results obtained from the use of antistreptococcic serum are still doubtful, but in cases of pure streptococcic infection it appears to be beneficial. If it is to produce any result, it must be employed early in the case and persevered with. If localised suppurative peritonitis occur the abscess must be opened where it tends to point, and encysted collections of pus, such as a pyosalpinx or suppurating ovarian cyst, require treatment on ordinary surgical principles. The most abundant supply of fresh air that can be admitted with safety should be secured.

3. *Puerperal Peritonitis*.—This, though a very frequent complication of puerperal fever, at times occurs independently of it, other symptoms than those consequent upon the local inflammatory attack being absent. It may arise from the rupture of a pyosalpinx, or an ovarian cyst, or by the transmission of infection through the Fallopian tubes or the lymphatics of the uterine wall.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances associated with puerperal peritonitis differ from those described in connection with puerperal septicæmia only inasmuch as they are confined to the peritoneal cavity. There will probably be found an abundance of effused serum or sero-pus, and flaky lymph, and intense congestion of the peritoneum; the abdominal viscera will here and there be glued together, and covered with a yellowish exudation which can be stripped off, leaving a raw bleeding surface. In some cases all that is found in the peritoneal cavity is a dirty thin fluid consisting of serum, some blood, and countless micro-organisms. The uterus will probably be found abnormally soft.

SYMPTOMS.—Generally within a week following delivery a well-marked rigor occurs, followed by febrile disturbance. The patient complains of acute pain in the lower part of the abdomen, at first in one particular spot, but soon spreading over a larger area, and often recurring in acute paroxysms. The thighs become flexed on the abdomen to relieve the tension; the belly becomes much swollen, and excessively tender; and there is generally much tympanites, with obstinate constipation. There is an extremely anxious pinched expression of the face, and the pulse is very characteristic, being quick, wiry, and incompressible. Vomiting soon sets in, and is frequent and persistent. If the disease do not give way, the abdomen becomes more swollen and tense, and no pressure upon it can be borne. Everything that is taken is vomited; the pulse becomes more rapid and feeble; the tongue is dry and raspy; the patient complains of intense thirst; the constipation gives way to diarrhœa; the skin becomes clammy, and the extremities cold; and the patient dies, usually within three to seven days from the onset of the symptoms. There may, however, even in the worst cases, be no abdominal tenderness or pain, and no fever.

TREATMENT.—Hot fomentations and counter-

irritants, such as turpentine, and in some cases iced flannels, are often of great service in subduing the local inflammation and relieving pain. In the first stage a copious enema of thin gruel with castor-oil, to obtain a free action of the bowels, should be given. Where there is much tympanites, the addition of turpentine may be of benefit in dispelling the flatus. If vomiting prevent nourishment being taken by the mouth, stimulant and nutritive enemata must be given every six to eight hours. For the relief of excessive pain, or where the issue is certainly fatal, morphine may be administered. Strychnine in doses of $\frac{1}{60}$ or $\frac{1}{40}$ of a grain may be given hypodermically every hour for its effect upon the heart and nervous system. The performance of abdominal section with free flushing out of the peritoneal cavity often offers the only possible chance of recovery.

4. Embolism and Thrombosis of the Pulmonary Artery.—**DEFINITION.**—The occurrence of a blood-clot in the right side of the heart or pulmonary arteries, either formed *in situ* or conveyed there from a distance by the blood-current, often giving rise to sudden death after delivery.

ANATOMICAL CHARACTERS.—The condition of the blood in pregnancy and the puerperal state renders it liable to form a coagulum, and this may occur in distant vessels. It is well known that in the later months of pregnancy the amount of fibrin in the blood is very greatly increased. Together with this, a diminution in the volume of the blood from uterine hæmorrhage produces a state of exhaustion, which causes a great predisposition to thrombosis. If, therefore, such having occurred in distant vessels, a portion of coagulum become detached, and be carried away till it reach the pulmonary arteries, embolism is the result, and this is one of the great causes of sudden death occurring after parturition. Some writers affirm, however, that pulmonary thrombosis may occur independently of embolism; large, firm, decolourised coagula have been found, on *post-mortem* examination, occupying the right side of the heart and the larger branches of the pulmonary arteries, which have evidently formed there, all traces of thrombosis elsewhere being absent. It is not certain that in all these cases a sufficiently careful search has been made for the primary thrombus. From a study of twenty-five cases Playfair concluded that if the accident occurred before the nineteenth day of the puerperium the obstruction is most probably due to a primary *thrombosis*; after the nineteenth day to *embolism*.

SYMPTOMS.—These are common both to embolism and pulmonary thrombosis. In the great majority of cases, the patient is suddenly seized with severe dyspnoea; she starts up and gasps for breath; the face in some cases has been described as pale, in others livid. She feels she is dying, and calls out for air, although it is entering the lungs quite freely; the pulse becomes almost imperceptible; and generally death occurs very rapidly. If the patient be examined during the attack it is probable that a murmur will be heard over the site of the pulmonary artery. In some cases in which the clot is not sufficiently large entirely to obstruct the circulation in the lungs, it appears that absorption may ultimately take place, and complete recovery ensue.

TREATMENT.—In almost every case so rapidly fatal is the seizure that there is no time to think of treatment. When, however, the attack is not so

terribly rapid in its termination, every effort must be made to rally the patient, by the administration of stimulants, such as brandy, ether, or ammonia, if at hand. Ether may be given subcutaneously, and the ammonia in large doses, with a view to dissolving the clot. If the patient survive the attack the most perfect rest must be enjoined, so as to prevent the coagulum from becoming dislodged, and to promote its absorption. *See* PULMONARY VESSELS, Diseases of.

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PUERPERAL INSANITY.—*See* INSANITY, Varieties of.

PUFFY TUMOUR (Pott's).—*See* TRAUMATIC MENINGITIS, p. 979.

PULLNA, in Austria.—Sulphated waters. *See* MINERAL WATERS.

PULMONARY APOPLEXY.—A term for a certain form of hæmorrhage into the lungs.—*See* LUNGS, Hæmorrhage into.

PULMONARY DISEASES.—*See* LUNGS, Diseases of.

PULMONARY HYPERTROPHIC OSTEO-ARTHROPATHY.—*See* p. 19.

PULMONARY MURMUR.—This term may be employed in two senses, namely, as signifying, first, the respiratory sound heard over the lung; or, secondly, a bruit heard in connection with the pulmonary artery and its valves. *See* HEART, Functional Diseases of; HEART, VALVES AND ORIFICES OF, Diseases of; PHYSICAL EXAMINATION; and PULMONARY VESSELS, Diseases of.

PULMONARY VALVES AND ORIFICES, Diseases of.—*See* HEART, VALVES AND ORIFICES OF, Diseases of; and PULMONARY VESSELS, Diseases of.

PULMONARY VESSELS, Diseases of.—The vessels of the pulmonary circulation, more especially the veins, enjoy a considerable immunity from disease. Primary affections of these vessels are of most exceptional occurrence, and the causes leading to their being secondarily involved are not numerous. It is not easy to account for this. The pulmonary arteries much less often present those diseased states which are of frequent occurrence in the arteries of the systemic circulation, and are not even as commonly affected as the systemic veins, with which they somewhat more closely agree in point of structure, and in the kind of blood carried by them. The portal vein, which is comparable to the pulmonary artery in other respects besides its plan of distribution, would appear to be similarly free. For these reasons affections of the pulmonary vessels are rather of pathological interest than clinical importance; in the majority of cases they are not to be recognised during life, or, if so, are beyond the application of any treatment. The trunk of the artery, and especially the orifice in the right ventricle, is singularly liable to present congenital abnormalities, which are treated of in the article HEART, Malformations of.

The diseases of the pulmonary artery will now be discussed in the following order: (1) Inflammation; (2) Degeneration; (3) Ulceration; (4) Dilatation

and Aneurysm; (5) Stenosis; (6) Rupture; and (7) Embolism and Thrombosis.

1. Inflammation.—Acute arteritis affecting the pulmonary artery is of very rare occurrence, and usually co-exists with acute endocarditis. Previous to birth it seems to be more liable to exist than subsequently, and some of the congenital deformities of the pulmonary artery and its valve are to be attributed to it. After birth it is almost invariably associated with such acute blood-diseases as pyæmia, or with those pyrexial states which are apt to assume a septic character, as scarlet fever. Emboli, especially if of a putrid character, which have become lodged in branches of the vessel, are liable to set up inflammation in the contiguous walls. Chronic arteritis, leading to atheroma, though far less frequent than in the aorta, is not of very rare occurrence, and under very much the same conditions, namely, increased strain of the vessels, associated with obstruction to the pulmonary circulation, and an hypertrophied right ventricle. When the vessels have become much dilated, as from extreme mitral stenosis, the walls thus thinned are liable to undergo changes of a chronic inflammatory character. *See* ARTERIES, Diseases of.

The anatomical characters correspond with the usual characters of arteritis and arterio-sclerosis.

This state is only demonstrable after death; during life it is not recognised, unless the valves be affected, by any known signs or symptoms, and a diagnosis of its existence has not hitherto been attempted. Under such circumstances no plan of treatment can be laid down.

2. Degeneration.—*Fatty degeneration.*—Fatty degeneration of the intima, without any previous inflammation, occurs in the pulmonary as in other arteries.

Amyloid disease has been seen in the muscular coat of branches of the pulmonary artery.

3. Ulceration.—As already said, inflammation of the vessel-walls very rarely extends to ulceration of the inner coat, but owing to the extreme frequency of ulcerative destruction of the lung-tissue, the intra-pulmonary branches of the vessels are constantly involved. Phthisis, abscess, or gangrene of the lungs, may each in their progress invade the vessels, the walls of which, though offering considerable resistance to the destructive process, sooner or later yield, and may cause a fatal hæmorrhage, though very frequently this is prevented by thrombosis and obliterative arteritis.

4. Dilatation and Aneurysm.—**ANATOMICAL CHARACTERS.**—Varying degrees of abnormal distension are not unusual, occurring in both sexes and in all ages beyond childhood, and are estimated as forming $\frac{3}{4}$ per cent. of aneurysms of all kinds. The dilatation may affect the trunk uniformly; and an extreme case has been recorded where the circumference of the vessel attained $6\frac{1}{2}$ inches, the normal average being taken at $3\frac{1}{2}$ inches. Or, limited in extent, the bulging forms a sacculated, or, more rarely, a dissecting aneurysm of the trunk or branches, from the size of a walnut to a pea, or even smaller, these latter being frequently multiple. The conditions which lead to these alterations in the normal calibre of the vessel are—(a) Those causing a diminished resistance of their coats to the blood-pressure, especially if this be increased, which is often the case, by obliteration of some vessels, and consequent rise in tension in the remaining ones; or by general obstruction, such as mitral

stenosis or emphysema would cause. (b) Those changes in the lung-structures which diminish the support of the vessels, and so allow of their yielding. The results of arteritis and atheroma will furnish the first condition, and ulceration and destruction of the pulmonary tissue as in local tuberculosis will provide the latter. The trunk of the artery has been frequently found unduly dilated in anæmia. The walls of true aneurysms may be thicker or thinner than those of the healthy vessel, and it is remarkable that their contents are said to be never laminated coagula, even in the largest, but always fresh clots. *See* HÆMORRHAGE, p. 621.

SYMPTOMS.—When the main trunk of the pulmonary artery is the seat of an aneurysmal tumour, there are the usual signs of pulsation and prominence in variable degrees, most marked to the left of the sternum in the second intercostal space; over the same area a systolic bruit of a superficial quality is to be heard, not conducted above the sternum or clavicles; and a systolic thrill is to be felt. There is also accentuation of the second sound, with the signs of hypertrophy of the right ventricle. Should the tumour be of any considerable size, it will give rise to those conditions which commonly follow an obstruction to the pulmonary circulation—namely, lividity, dyspnoea, cough, and general anasarca, with scanty, high-coloured urine; and symptoms of pulmonary embolism may result from detachment of fragments of clot from the sac. In an exceptional case, pallor of the face was noticed. Pain behind the sternum, and headache, also exist. Since the greater part of the artery is included within the pericardium, it is into that sac that rupture will probably occur. Uniform dilatation of the trunk of the artery may be accompanied by sufficient stretching of the cardiac orifice to cause regurgitation into the ventricle.

The small aneurysms of the intra-pulmonic branches give rise to no known symptoms until hæmoptysis indicates their rupture.

DIAGNOSIS.—An aneurysm of the trunk of the pulmonary artery may have to be distinguished from a similar affection of the aorta, or from a post-sternal tumour to which pulsation has been communicated. The tendency of pulmonary aneurysm to extend to the left side, and the non-conduction of the bruit to the vessels at the root of the neck, with the coincident signs of pulmonary obstruction, are grounds upon which to found a distinction, though the distinction from an aortic aneurysm is not always easy.

PROGNOSIS.—This is of necessity grave, whatever the size of the lesion, and many cases of fatal hæmoptysis are due to rupture of a small-sized sac.

TREATMENT.—How far such treatment as galvano-puncture, the administration of iodide of potassium, &c., as pursued in aneurysm of the systemic vessels, is applicable to similar affections of the pulmonary artery, is unknown. For the treatment of the hæmorrhage to which rupture of the smaller aneurysms gives rise, *see* HÆMORRHAGE, p. 621.

5. Stenosis.—A narrowing of the pulmonary artery may take place at the orifice, in the conus arteriosus, or more rarely in the trunk or main branches. In the former situations it is commonly congenital, the result of endocarditis or myocarditis, which, if developed within the first three months of foetal life, is almost invariably accompanied by some compensating lesion, such as intraventricular

communication ; while if the affection of the heart be subsequent to the third month of development, the circulation is carried on through a patent foramen ovale and ductus arteriosus (*see* HEART, Malformations of). It is conceivable that stenosis of the conus arteriosus may be followed by secondary narrowing and closure of the pulmonary artery, and also that defective development of the lungs may cause a narrowed vessel. The condition is very rarely due to any acquired change in the vessel-walls, although a case is recorded of stenosis of the artery from cartilaginous thickening and calcification of its coats. The calibre of the tube may be diminished by the pressure of tumours, such as an aortic aneurysm or adenoid growths, or by the shrinking of cicatricial tissue in the adjacent lungs, or from arteritis following on direct violence.

When stenosis is developed at a very early period of fetal life, the artery remains exceedingly narrow beyond the obstruction. When it occurs late, the vessel may be of normal capacity, and if insufficiency co-exist with the obstruction, it may even be dilated.

SYMPTOMS.—Whatever be the cause of pulmonary stenosis, there will be a deficient supply of blood to the lungs, producing dyspnoea, and the obstruction to the circulation will give rise to all the signs and symptoms of general venous congestion, although to a less degree than in affections of the tricuspid orifice. Hypertrophy of the right ventricle—as evidenced by increased transverse measurement of the area of cardiac dulness, and a forcible impulse felt at the epigastrium, a basic thrill, a systolic bruit, of maximum intensity over the heart's base, and conducted to the left of the sternum, but not audible along the course of the aorta and great vessels, and a marked accentuation of the second sound—is the most important result of this condition. Cyanosis is not a characteristic, and does not occur unless there be extreme venous congestion, or a communication between the two sides of the heart. The association of constriction of the pulmonary artery, both congenital and acquired, with tubercular phthisis, has now been too frequently observed for it to be regarded as a coincidence only, and their relation as cause and effect is generally admitted.

TREATMENT.—This affection is entirely beyond the reach of remedy.

6. Rupture.—Violent effort and great excitement have been followed by rupture of the pulmonary artery, either of the trunk or main branches, when degenerated. Death is often instantaneous, though it may be delayed even some hours. Aneurysms tend to burst sooner or later ; those of the trunk usually opening into the pericardium, while the intra-pulmonary dilations commonly rupture into cavities in the lung. Ulceration, as said, is of very rare occurrence, but a case is recorded of its existence and extension through all the coats of the vessel, with a suddenly fatal termination. Rupture of the pulmonary veins has been recorded.

7. Embolism and Thrombosis.—The pulmonary artery is especially liable to become plugged, both by substances lodged in it from elsewhere, and by coagula originating in the vessel itself. Its relationship to the venous circulation explains this. Portions of broken-down clots developed in the systemic veins, from whatever cause ; the contents of hydatid and other cysts that have burst into the

venous current ; fragments of cancerous and other new-growths, all of which readily travel onwards towards the heart, pass into the pulmonary artery, in the branches of which they become lodged, according to their size. Once located, the plug will increase in size by the deposition on it of successive layers of fibrin, sometimes to such an extent as to obliterate all traces of the original obstructing substance. Occasionally very large thrombi are detached in the systemic veins, and are arrested in the trunk and main branches of the pulmonary artery. The causes of thrombosis of the vessel are various. The rare occurrence of inflammation or degeneration of the artery renders obstruction of the vessel from primary thrombosis very uncommon ; but the development of clots in the smaller branches, in association with pneumonia, phthisis, gangrene, and other destructive lung-diseases, is frequent. In certain septic states, in severe malarial states, in parturient women, and in conditions of extreme anæmia, especially with diminished heart-power, when the blood is prone to clot in the vessels, the pulmonary artery is a favourite locality for this to occur ; though the not unusual occurrence of this condition in women after delivery is more probably embolic in character. Pressure on the pulmonary artery or its main divisions by aortic aneurysm, enlarged glands, or other mediastinal tumours, has been known to cause the formation of a thrombus in the vessel. Thrombi may commence in the right ventricle or, as would appear, sometimes on the semilunar valves, and extend into the trunk and, for variable distances, into the branches of the vessel. Such obstructions are frequently developed during the last hours of life, when the circulation is enfeebled and slow. *See* EMBOLISM ; HEART, Thrombosis of ; and LUNG, Hæmorrhage into.

SYMPTOMS.—The symptoms will, of course, depend upon the extent and completeness of the obliteration of the pulmonary circulation. If only the smaller branches be occluded, the symptoms may be those of pulmonary infarction, and the very moderate dyspnoea or slight hæmoptysis might be equally attributable to the phthisis or other lung-state which had determined the formation of the thrombi.

In another class of cases, when larger branches are blocked, very marked dyspnoea is developed, with such symptoms as are conveniently grouped under the term 'anginal,' such as pain in the præcordia, a sense of great distress and faintness, palpitation, gasping, lividity, and extreme pallor, with cold sweats, but no loss of mental faculties, though often inability to speak, an almost imperceptible rapid or irregular pulse, and jactitation of the limbs. The onset of such a condition may be gradual or sudden ; in the former case it depends on the slow increase in size of a small thrombus ; in the latter on the sudden lodgment in some large branch of the artery of a solid substance that has entered the venous current. In some cases these symptoms are present to an extreme degree, and death follows in a few minutes ; in fact this lesion constitutes one of the causes of sudden death. The appearances are not those of asphyxia, and it is usual to attribute the very rapidly fatal result to syncope or shock, as it would seem to be connected in some way with an arrest of the nerve-governance of the heart. In that class of cases which do not terminate so quickly, it is usual to find that the symptoms abate somewhat, and may be followed at

a variable interval of hours, days or even months by a second or even several attacks, usually ending fatally. The *post-mortem* examination of such cases shows a thrombus of considerable extent, with indications of its having been formed at different times.

Examination of the chest reveals no diagnostic signs. There is very likely to be a basic systolic murmur conducted along the course of the pulmonary artery; but this is not constant, and the heart-sounds are often muffled and indistinct.

DIAGNOSIS.—This is often very uncertain. The conditions in which thrombosis is usually met with, such as in anæmic or parturient women, are those in which breathlessness, cardiac pain, and discomfort, and even a pulmonary hæmic bruit, are of frequent occurrence. The symptoms, when not of extreme rapidity, are very similar to those caused by stenosis of the pulmonary artery, which in itself is difficult to diagnose; and, lastly, the suddenly fatal cases are almost identical in their manifestations with rupture of the heart or of a thoracic aneurysm, or even angina pectoris. The supervention of the above-detailed symptoms in a case of existing phlebitis, in a woman within twelve or fourteen days after child-birth, renders it highly probable that they are due to a clot in the pulmonary artery.

PROGNOSIS.—This is to be looked upon as of the gravest character, if once symptoms arise which indicate the existence of a clot in the pulmonary vessels. The smallest plugs formed in branches which are being invaded by a progressive destructive change in the lungs, are protective in character, and prevent or diminish an hæmoptysis which erosion of the vessels might produce.

TREATMENT.—In the most rapid cases death takes place before anything can be done; but in the less severe cases two points have to be attended to, namely, absolute rest, and free stimulation by brandy, digitalis, ether, and ammonia, for by such means only can any hope be entertained of preventing an extension of the clot. Sinapisms over the cardiac region often afford relief, but opiates for the sleeplessness which is met with in some of the prolonged cases are very badly borne.

W. H. ALLCHIN.

PULSATION (*pulso*, I beat).—Pulsation is a sensation of beating or throbbing, either objectively appreciated by inspection or palpation, or subjectively felt. It originates in the presence of a pulse or rhythmical rise and fall of blood-pressure, whether normal or abnormal, in connection with the part where it is situated. In most instances this is either the heart or some large blood-vessel; but in other instances the pulsation has a different origin, especially when the phenomenon is abnormal. As instances of *normal* pulsation may be mentioned the cardiac impulse; the arterial pulse generally; the pulsation of the umbilical cord; and the beating of the fontanelles. *Abnormal* pulsation may be preferable (1) to dilatation of a blood-vessel, as in aneurysm; (2) to vascular dilatation and cardiac enlargement, as in aortic incompetence; (3) to vascular dilatation and cardiac excitement, as in exophthalmic goitre; (4) to interference with the passage of blood through a vein, or even regurgitation into it, as in the jugular pulse of tricuspid disease; or (5) to the pressure of a tumour upon a large vessel, conveying the normal pulse unnaturally to the surface of the body, as in tumour of the

pancreas or pylorus. Pulsation may also be present (6) in any part when it is the seat of inflammation, the small vessels being dilated; (7) in aneurysm by anastomosis; (8) in malignant disease of bone, which may closely simulate aneurysm; and (9) very rarely in connection with empyema. See PLEURA, Diseases of.

With respect to the characters of this phenomenon, it is of great practical importance to distinguish *true* expansive or eccentric pulsation from pulsation which is *communicated* only. In the former case the seat of pulsation expands rhythmically in all directions; in the latter case it is moved in one direction only, that is, it rises and falls under the influence of the motion conveyed to it.

The various pathological conditions which give rise to pulsation, and their treatment, are fully discussed under appropriate heads.

J. MITCHELL BRUCE.

PULSE, The.—SYNON.: Fr. *Le Poulx*; Ger.

Der Puls.—A pulse can be felt in any accessible artery, but the most convenient is the lower end of the radial artery where it runs over the radius. If a finger be placed gently on the radial vessels it will be raised with each beat of the heart, owing to the varying resistance of the vessels. It has been found by experiments on animals that a pressure, corresponding to the actual blood-pressure in the vessel as measured by a manometer, gives the greatest excursion. When the pressure is insufficient, the tissues outside the artery do not transmit the beat well, and when the pressure is too great the vessel cannot expand efficiently. The pressure on the vessel flattens it, and what is felt is the return of the artery to a circular shape, as the blood-pressure varies. It is not the distension of the artery by the passage of the blood which constitutes the pulse, but chiefly the variations in the impulsive pressures which are suddenly transmitted along the artery with the varying changes in the pressure in the aorta. These variations are more marked with a low arterial tension than with a high. If three fingers be placed over the line of the artery, the first may be employed to produce varying pressures, the second to note the effect on the vessel beyond, and the third to cut off any regurgitant changes of the pressure in the vessel beyond. The size of the vessel of course varies, but the alteration is intensified by the application of pressure to the vessel.

The pressure which is applied by the first finger and which is required to obliterate the pulsation in the vessel beyond should always be noted. This will be found to vary under different conditions. When the vessel is lax, the size of the vessel will be felt to be large, and a moderate pressure will be generally sufficient; when the vessel is contracted, the size will be felt to be smaller, but a greater pressure will be required to compress the vessel completely, so that no pulsation can be felt beyond.

It used to be assumed that the channel under the fingers, with its varying pressures and sizes, consisted only of the artery, and the presence of the veins was ignored. In 1897 it was pointed out by Hill and Barnard that the varying vessels consist of the artery and the adjacent *venæ comites*, and that the distension of the latter markedly altered the character of the pulse. With venous obstruction, the pressure in the vein may equal that in the artery,

and both finger and sphygmograph are quite incapable of determining how much is due to the vein and how much to the artery. Hence in all discussions of the pulse and the size of the artery, as determined by the finger or by various instruments, we must bear in mind that it is the size and the tension of the combined cord consisting of the vein and artery which we are discussing, and that many of the older discussions which dealt solely with the artery were in consequence not infrequently erroneous in their conclusions.

The movement of the vessel, as perceived by the finger, at first appears to be simple, but when registered by a sphygmograph it will be noticed to be complex, consisting of two or more waves, and it will be convenient to study these characters from the tracings, which are discussed under the article SPHYGMOGRAPH.

These characteristics may be best appreciated by placing three fingers over the radial artery and passing them to and fro. The fingers readily note the size of the pulse-wave, and the thickness and rigidity of the vessel wall, but they are not capable of recognising small variations in the size of the vessel under different conditions.

A gradually increasing pressure is applied by the proximal finger. When the tension is low, the pulsation beyond is readily stopped, but when it is high the beat, which was at first not marked, becomes more striking, and the pressure required to finally obliterate the pulsation beyond will be above that which we learn by experience to be the normal. The middle finger notes when the pulsation ceases, pressure also being applied by the distal finger to cut off the reflux current through the palmar arch, which is sometimes well marked. Observation with instruments shows that when the vessel is small and contracted, the finger under-estimates the amount of the pressure. The finger gives only a rough idea of the pressure, as no method by which the pressure is transmitted through a solid medium, such as the finger, can give reliable results when the thickness of the tissues over the vessels, as found in different individuals, varies.

It will be convenient to consider in succession the following characters of the pulse and of the vessel: (1) Vessel-wall; (2) Size of vessel; (3) Tension of pulse; (4) Rate; (5) Rhythm; (6) Characters of pulse; (7) Amplitude of pulsation.

1. Character of the Vessel-walls.—When the tension in the vessel is inhibited by firm pressure above, a finger rolling the artery gently beneath it feels whether it is thickened, rigid or calcareous. It is also noticed whether it is tortuous, and an opinion can be formed as to the amount of atheroma and degeneration there is present. When the vessel is degenerate and inelastic, it also elongates, hence the circulation tends to make the vessel tortuous, and the curvature travels along the vessel, producing a locomotor pulse.

2. The Size of the Vessels.—Although a finger will appreciate whether an artery is constricted or dilated, it will not detect small differences; nor can it record the size for comparison between one observation and another. The most convenient apparatus for this purpose is the arteriometer, designed by Dr. Oliver. This instrument measures the difference in the diameter of the vessels, when they have only the weight of the button resting on them, and when the compression has been sufficiently increased just to obliterate the

pulsation beyond. In using this instrument attention must be paid to several details in order to obtain accuracy, and considerable practice is required before reliable results can be obtained. The muscles of the arm must all be relaxed, the wrist, which, at the time of observation, should be at the same level as the heart, must be over-extended at a constant angle, and the radial side somewhat raised. The arteriometer, held between the finger and thumb, must be placed with the central button accurately on the vessel, care being taken that the two outer legs are well supported on the bone and tendons, and that the instrument is held quite vertically. Increasing pressure is then applied until no pulsation can be detected beyond. In this way the size of the compressible vessels may be measured, and although in health the size of the radial veins is negligible when the wrist is over-extended, this is not the case in asphyxia, when they may cause an increase of as much as .6 mm. Probably this will account for part of the increase in calibre, which is observed in recumbency, in some abnormal conditions when gravity becomes a potent factor. In a vigorous individual the splanchnic vaso-motor tone comes into play, to counteract the effects of gravity, and to prevent the blood from gravitating into the abdominal veins in excess, in the erect posture. Hence this tone is best studied by observing the effects of change of posture, and the calibre should be taken, both in the recumbent and in the sitting or erect posture. In health in the recumbent position, the calibres vary from 1.5 to 1.7 mm., and in the erect from 2.0 to 2.3. In many normal conditions the calibre varies directly with the mean arterial pressure, but this is not universally so.

In healthy, vigorous individuals the calibre is generally found to be at its minimum in the recumbent position. It is at a maximum, during the digestion of a full meal, after muscular effort, and as the result of fatigue, or when an individual is 'out of health.'

A. Conditions under which the radial calibre is diminished in size.—(1) Recumbent position. (2) Congenitally small artery; the mean pressure will then be normal, and the usual changes in calibre with alterations in posture will take place. (3) Exposure to great cold or to a temperature above 100° F. (4) Massage in the form of *pétrissage*. (5) Low arterial pressure. (6) Arterial constriction; in this condition the arterial pressure is raised, and the usual alterations due to changes of posture do not take place.

B. Conditions under which the radial calibre is increased in size.—(1) By exercise, digestion, exposure to a temperature between 97° and 103° F., and by massage in the form of *tapotement* or *effleurage*. (2) Congenitally large vessel; when the pressure and the variations with change of posture are normal. (3) Increased arterial pressure. (4) Dilated atheromatous vessels.

C. Conditions under which the calibre ceases to vary with posture.—(1) Excessive blood-pressure. (2) Marked constriction of the vessels, as with megrim, hysteria, &c. (3) Arterial degeneration, as in syphilis, chronic goutiness, Bright's disease, and arterio-fibrosis.

D. Conditions in which the calibre is less in the erect than in the horizontal posture.—(1) Neurasthenia and exhaustion, from whatever cause the latter may arise.

3. **Tension.**—The tension of the wall varies directly with the blood-pressure, but also inversely as the diameter of the vessel. The pressure can be roughly estimated by the finger, but the tendency is to under-estimate the pressure when the artery is contracted. To detect the slight variations, and to record the amount it is necessary to use some instrument. Although there are several of these, they have not come into daily use in Great Britain, hence they require some practice before reliable results can be obtained with them.

Hill and Barnard have devised two instruments depending upon the principle that if a tampon be applied to a vessel with varying pressure, and be arranged so as to transmit, by means of fluid or air, its pulsations to a recording scale, the greatest pulsation will be obtained when the pressure in the tampon is equal to the mean pressure in the vessel. This has been determined by experiment, and it has been found that the same pressure is recorded in all the superficial vessels whether carotid, radial or femoral, provided (1) that the pad over the vessel is not solid, (2) that the pressure be estimated at that which gives the largest pulsations, and not at that which obliterates the pulsation beyond. In one form an indiarubber bag is strapped round the arm, and the pressure in the bag, which communicates with a manometer, is gradually increased and diminished until the maximum pulsation has been obtained; the corresponding figure gives the arterial pressure. The process is too uncomfortable to be applied to clinical work at all generally. In the other, a small manometer containing fluid is applied over the vessel to be observed, and the pressure which gives the maximum pulsation is the one required.

A similar instrument, the *Hæmodynamometer*, devised by Oliver, fitted with a fluid pad, is the most convenient and gives reliable results.

Some practice is required to determine at which pressure the pulsation has the largest amplitude, and since this is more obvious when the amplitude is small, it is often useful to insert a small piece of wash-leather between the artery and the recorder. The maximal pulsation-pressure, which may be called the *mean pressure*, is about two-thirds of that required to stop the circulation, which may be called the *maximum pressure*.

The same instrument can be used to determine the venous pressure. The pad is applied firmly at the distal part of a superficial vein after it has been emptied by pressure, and as the pressure is relaxed, it is noted at what figure the blood returns into the vein. The effect of gravity on the venous pressure according to the position of the vessel, above or below the heart, can be accurately measured; the pressure in a vein on the foot may be 15 mm. Hg. in the recumbent and 135 mm. in the erect position. The venous pressure varies in proportion more with posture than does the arterial: in the recumbent it may be 10 to 15 mm. and as much as 20 to 30 in the erect; the hand in both cases being kept at the heart-level.

It is immaterial whether the pad rests exactly on the vessel, or whether it be separated by a layer of wash-leather, provided the pulsation is still visible. The pressure, whether arterial or venous, is largely affected by gravity, and 1·9 mm. Hg. must be allowed for every inch that the vessel is above or below the position of the heart. When this allowance is made, it is found that the arterial pressure

over the body is uniform. The mean arterial pressure is in the recumbent position 90 to 100 mm. Hg., and in the erect (the wrist being kept at the level of the ensiform cartilage) 100 to 120. Fatigue or loss of vaso-motor tone may abolish this difference, and in conditions of exhaustion the pressure may be the higher in the recumbent position. Exercise, emotion and digestion all raise the pressure temporarily. The vaso-motor tone may also be measured by noting what effect a weight of 28 lbs. distributed over the abdomen has on the radial pressure in the recumbent position. In a feeble person it may amount to 15 to 30 mm. of Hg., while after exercise or in the vigorous it may be nil. One, if not the most essential change in connection with puberty, is the marked increase in arterial pressure which takes place.

Conditions under which high tension occurs.

The pulse tension will be found to vary considerably in healthy individuals; but every alteration which takes place is of importance to the individual. Increase in the volume of the blood due to ingestion of fluid, and frequent, powerful action of the heart due to exercise or exertion, are only temporary causes of increased blood-pressure. Arterial contraction is the principal cause. This may result from exposure to cold, or may occur in hysteria and megrim. An anginal attack is not infrequently started by chilling of the skin, and the same cause may produce, during swimming, 'fatal cramp'—really an arterial spasm which the heart is unable to overcome. With increasing years, as an hereditary condition, with renal disease, gout, lead-poisoning, pregnancy, and sometimes with anæmia, and also with emphysema and chronic bronchitis, the tension may be markedly raised. In anæmia a systolic apical bruit with a high-tension pulse is more likely to be due to the anæmia than to valvular disease. Craig has drawn attention to the frequency with which high tension is met with in melancholic patients. The daily consumption of large amounts of either meat or alcohol, sedentary habits and constipation, all tend to induce high tension.



FIG. 1.—Trace of Pulse of High Tension.

Characters of a high-tension pulse.—1. An amount of pressure must be applied, considerably above the normal, before the characters of the pulse can be elicited, when the beat will become more forcible. 2. The pressure required to obliterate the pulsation beyond is above the normal. The pulse is generally prolonged, nor can any diastolic wave be felt, until the heart has dilated and failed.

The vessel may be either large or small; when the latter, the pulse may be barely perceptible, until pressure is applied; and in consequence the mistake is often made of considering it to be feeble. There do not appear to be any characteristic mental or physical qualities associated with either high- or low-tension pulses in conditions of health.

Conditions under which low tension occurs.—This may be due to cardiac failure, to dilatation of the peripheral vessels, or to diminution of the fluid in the peripheral circulation, whether due to a

diminution in the total amount of blood or to the dilatation of the abdominal veins, which are capable of holding all the blood normally distributed throughout all the vessels. A great loss of fluid, whether from hæmorrhage, vomiting, or diarrhœa, will induce low tension. External warmth, fatigue, exercise, insufficient food, debility, pyrexia, and many toxic conditions will produce a low-tension pulse. It also occurs congenitally, and in cases of fatty heart.

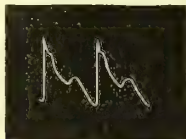


FIG. 2.—Trace of Pulse of Low Tension.

A low-tension pulse is sudden and short, it subsides very quickly, and a very moderate pressure will obliterate it. The dicrotic wave is well marked at the onset of an acute fever, the heart's action is vigorous and the volume of the pulse is much larger than in cases of cardiac failure.

Patients with habitually low tension bear purgatives, especially mercury, badly. The prognosis for a patient with low tension in a disease in which the tension is usually high is unfavourable.

4. **Frequency.**—The frequency of the pulse depends on the rate of the heart's contractions. This rate varies with age, position, sex, stature, and a number of physical and psychical influences. In the newly born infant the heart and pulse beat some 130 to 140 times a minute. The rate gradually falls, and after the sixth year it is usually below 100; and a further decrease of 30 beats a minute gradually occurs before the rate of manhood (70 to 75 a minute) is reached. In old age the pulse-rate often rises again slightly.

In the erect posture the pulse-rate is about ten per minute more in the male, and seven in the female, than when sitting; and when recumbent it is about five less. The pulse-rate of a girl of seven averages about ten beats per minute more than that of a boy of the same age. As regards stature, we may say briefly that the rate is decreased in the tall, being three or four beats less in a man six feet high than in one five feet six. Movement and exertion of all kinds quicken the pulse and emotion or excitement may produce a very high rate in a neurotic person. When examining candidates for life-insurance, as well as when visiting patients, this must be taken into account. A large meal increases the fullness and frequency of the pulse, as does the use of stimulants in health; on the other hand, in acute disease, they often reduce the pulse-rate, which would be good evidence of their beneficial action. The pulse is less frequent in the night and during sleep; it rises in frequency during the early hours of the day, and falls to a minimum about midnight.

Increased Frequency.—In almost all forms of illness the pulse-rate is increased; while in debility and anæmia the rate is also very unstable, being suddenly increased by slight exertion or emotion, the tension generally being below the normal. Pyrexia produces a rapid pulse with dilatation of the peripheral vessels. At the onset of an attack the heart's action is forcible and the vessel lax; hence, not only is the rate increased, but the pulse will be large, sudden, short and dicrotic, the rate

tending to increase with the temperature. As the fever continues the heart's action becomes more feeble, and the pulse smaller, so that its size and rate become of great importance in estimating the condition of the heart. All the conditions for the production of dicrotism are present, and it is in fever therefore that it is most commonly met with. In scarlet fever the pulse is especially rapid, and may run up to 120 or 130 in an adult, and in a child to 150 or 200, without indicating excessive danger. In pneumonia, a pulse of 120 or more in a man at the onset should always give rise to grave anxiety, as it may be the only indication at that time of the seriousness of an attack which may subsequently end fatally. In women or children, on the other hand, such a pulse would, of itself, not give rise to any anxiety, as their circulation is much more unstable. It requires a considerable disturbance to upset the stability of the pulse-rate in a vigorous man, and its occurrence is therefore of greater significance. In phthisis, the presence of a rapid pulse, when the amount of mischief is not great, is of evil omen, as it shows that the patient is very susceptible to the tubercular poison. In all forms of valvular disease of the heart, with the exception of aortic stenosis, the rate is increased. In septicæmia and peritonitis the pulse-rate is of greater importance in prognosis than is the temperature, and is a measure of the effect of the poison on the nervous system. The heart's action is excessively rapid in digitalis-poisoning, and in the later stages of meningitis.

Persistent frequency results from overstraining the heart with exertion, and is not infrequently met with in young soldiers, in those who have over-exerted themselves at athletics, and in middle-aged persons suffering from atheromatous arteries, although the efforts which have set up a dilatation of either one or other cardiac ventricle would have been harmless to younger men.

Rapid action is also a cardinal feature in Graves' disease, and sometimes may be the first symptom. The rate may remain at 120 or much more for weeks at a time in spite of treatment, and is rarely under 100.

Cases of tachycardia or rapid action of the heart as the only abnormal symptom have been recorded by Bristowe and others; the rapidity persists in spite of treatment. These cases generally die within a few years, and often no lesion can be found.

Diminished Frequency.—In comparatively few diseases is the pulse-rate reduced. The most important is that due to high tension in the aorta, with which chronic renal disease and atheromatous arteries are not infrequently associated. It is met with in fatty degeneration of the heart; in aortic stenosis, when the rate may fall to 20 or less; in jaundice; with any lesion that produces intracranial pressure after an epileptic fit; in convalescence from pneumonia or typhoid fever. At the onset of typhoid fever the rate is slow compared with the temperature. See BRADYCARDIA.

5. **Rhythm.**—This depends upon that of the heart; the deviations from regularity may be of two kinds, *Intermittence* and *Irregularity*.

Intermittence is the term applied when there is a beat entirely missing from time to time, while the pulse in between is perfectly regular. The intermission may occur regularly every fourth, fifth, or tenth beat, or it may occur irregularly. Here the auricle contracts on receiving its stimulus, but fails to induce

ventricular contraction, or if it does, the contraction is too feeble to be detectable at the pulse, and intermission results. Hence it generally happens that the next contraction of the left ventricle is more forcible than the previous one. Intermissions may be met with in the young in connection with the excessive use of tobacco; but are more common in later life, when they also occur with dyspepsia, gout, and sometimes from excitement or fatigue, and more rarely from degeneration of the heart-muscle. Effort, such as briskly walking across a room, will increase the intermissions in cases of organic disease, but will cause them to disappear when they are only functional. Intermission disappears usually with an attack of pyrexia.

Irregularity.—In this case the beats are irregular, not only in time but also in force, and the condition is more serious than the preceding. Still, very marked irregularity frequently associated with palpitation may be induced by the use of tobacco or tea, or by flatulence, often producing the greatest distress to the patient; while an organic irregularity may occur without such conscious distress. Irregularity may occur in the healthy on first waking, and also during convalescence from acute diseases. Irregularity is a frequent result of degenerating heart-muscle, and may occur in the last stages of a systole, following various lesions. Most observers consider that irregularity is much more characteristic of mitral stenosis than of regurgitation, probably because the stress in this case falls particularly on the auricle, which more readily fails. Broadbent, however, considers that the reverse is true. When the pulse-rate is irregular it would appear that the failure to contract lies with the auricle.

Sometimes a tracing will show a feeble pulsation when the finger only detects an intermission. The irregularities in these cases are increased by exercise. Inequality in the size of the pulsations often depends on violent expiratory efforts, affecting chiefly the veins. The curve-basis or respiratory line in a sphygmogram falls with deep inspiration as the blood is sucked into the right auricle, while it rises with forced expiration. These variations are most marked with a low-tension pulse, and with high tension are hardly apparent. With forced dyspnoea the curve-basis is undulatory. The pulsus paradoxus is an exaggerated form of this pulse, and is noticeable especially with indurative mediastinopericarditis. The increase in the size of the pulse to be noted with violent expiration is due to the increase in the size of the vein, not in that of the artery, as was formerly supposed.

6. Character of Pulse.—Besides the qualities to which attention has already been drawn, the character of the pulse must be noted. The wave may strike the finger suddenly or lift it deliberately. A sudden or *quick* pulse is produced when the heart empties suddenly into a not over-tense aorta, as in aortic incompetence, with exercise, or at the onset of an acute pyrexial attack. A gradual rise or a *slow* pulse occurs in the opposite conditions, such as rigid vessels, aneurysm, aortic stenosis. The pulse may be felt to last for an appreciable time, forming a *long* pulse, as with high tension; or it may pass rapidly, forming a *short* pulse, as with cardiac failure. The fall may be sudden, as with aortic incompetence; or gradual, as in the early stages of chronic Bright's disease.

A pulse is dicrotic when a second pulsation is felt in the latter part of the beat; this is produced by the momentum of the blood-column as it rebounds on the closed aortic valves, and therefore is best marked when the tension is low. To feel it, only light pressure must be made with the finger.

7. Amplitude of Pulsation.—The amplitude of a pulse is shown in a tracing by the size of the line of ascent and of the tidal wave; these depend on both the amount of blood expelled at each contraction from the heart and also on the state of fulness of the arteries. When the arteries are contracted the pulse is small, hard, and wiry; when the coats are relaxed, it is large and soft. A large pulse is best developed by a vigorously acting heart and a relaxed condition of the arteries, such as obtains at the commencement of an acute specific fever. A flickering pulse is indicative of feeble and unequal ventricular contractions, and the undulatory character noticed in some weak pulses is due to the influence of the respiratory movements on the venous tension. The quality of suddenness, due to the rapid emptying of the left ventricle, gives in a sphygmogram a nearly vertical line of ascent; while the gradual pulse, produced when the ventricle empties slowly, as with aortic stenosis or with a thoracic aneurysm, has an oblique upstroke. The greatest amplitude is met with in cases of aortic incompetence with an enlarged left ventricle, when the pulsation of the vessels in the neck at once catches the eye. The pulse may be impalpable either on account of the feeble condition of the circulation, occlusion of the vessel by a thrombus, an aneurysm, external pressure on the artery, or an abnormal position of the vessel. The pulses in the two radial vessels may be unequal with a thoracic aneurysm; also, one may be retarded and its characteristics but feebly marked. The sudden splashing character of the pulse of aortic regurgitation is greatly intensified when the arm is raised, as the increased pressure in the aorta causes an increased regurgitation, while the same position diminishes the size of the pulse in mitral incompetence.

Gaertner has invented a *tonometer* which is much used in Germany. A finger-tip is rendered anæmic and the middle of the finger is surrounded by an indiarubber bag containing air, which is in connection with a manometer. The pressure is reduced until the blood suddenly rushes back into the capillaries, when the pressure is read off. The instrument is conveniently portable, and an observation does not take more than a couple of minutes. Haig has noted that the time that capillaries take to refill after the skin has been rendered anæmic varies from a normal of about two to about six seconds, when there is contraction of the arterioles. The time can with practice be determined sufficiently accurately with a metronome beating half-seconds.

Capillary Pulsation is most marked in cases of aortic incompetence, and can be brought out by drawing a nail over the skin and thus producing a hyperæmic area which pulsates: or by a microscopic cover-glass pressed on the mucous membrane of the lower lip. It is often present after a hot bath in health, or when the superficial vessels are well dilated from any cause.

Venous Pulsation.—This is often noticeable in the veins of the neck. Normally it is *predicrotic* in rhythm, and the veins do not fill up from below. In tricuspid incompetence, however, with a

vigorously acting right ventricle there will be a *systolic* venous pulsation in the veins of the neck filling up from below, and often also in the liver.

The following arrangement shows in a small compass the principal varieties of pulse met with in practice, apart from the quality of regularity.

A. Varieties of Hard or High-Tension Pulse—*Pulsus durus*.

1. The *hard, frequent, sudden, and small pulse* of peritonitis, enteritis, and pericarditis:—

Pulsus durus, frequens, celer et parvus, fig. 3.



FIG. 3.

2. The *hard, slow, gradual, and large pulse* of contracted kidney:—

Pulsus durus, rarus, tardus et magnus, fig. 4.

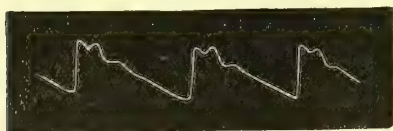


FIG. 4.

3. The *hard, large, often gradual pulse* of cardiac hypertrophy and degeneration of blood-vessels:—

Pulsus durus, magnus et tardus, fig. 5.

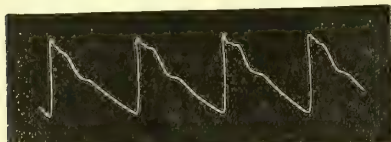


FIG. 5.

4. The *hard, sudden (jerky), large, and vibratory pulse* of aortic insufficiency, with a strong ventricle:—

Pulsus durus, celer, magnus et vibrans, fig. 6.

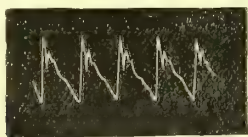


FIG. 6.

B. Varieties of Soft or Low-Tension Pulse—*Pulsus mollis*.

1. The *soft, frequent pulse* of pyrexia; dicrotic, fully dicrotic, and hyperdicrotic pulses:—

Pulsus mollis et frequens, fig. 7.

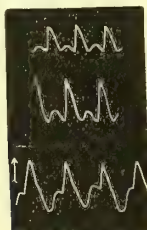


FIG. 7.

2. The *soft, frequent, and large pulse* of rheumatic fever and at the onset of a specific fever:—

Pulsus mollis, frequens et magnus, fig. 8.

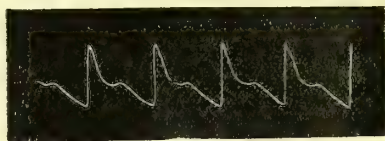


FIG. 8.

3. The *soft, small, frequent, and sudden pulse* of debility:—

Pulsus mollis, frequens, parvus et celer, fig. 9.



FIG. 9.

4. The *soft, frequent, and small (running) pulse* of collapse and cardiac failure:—

Pulsus mollis, frequens et parvus, fig. 10.



FIG. 10.

B. WALTER FOSTER.
G. NEWTON PIIT.

PUNCTURE.—See ACUPUNCTURE and LUMBAR PUNCTURE.

PUPIL.—The pupil is the aperture through which light is transmitted to the back of the eye. It is situated slightly to the nasal side of the middle line and in health is round. It is not of any fixed or definite size even in the individual, and varies with the conditions to be presently attended to. As a rule the pupils in the two eyes are equal. They are occasionally unequal, however, even in a healthy individual, although inequality is regarded by some as an indication of a neurotic tendency. Contraction of the pupil results from the contraction of the *sphincter pupillæ* muscle; dilatation depends upon the action of the *dilatator pupillæ*, but there is probably also some natural resiliency in the iris itself which accounts for some degree of dilatation.

Contraction of the healthy pupil occurs as a result of exposure to light and also during convergence. Contraction to light is a reflex action. The afferent fibres of the reflex arc run in the optic nerve. There is considerable difference of opinion as to their actual path. It is probable that they partially decussate at the optic chiasma, from thence along the optic tract, go to the corpora quadrigemina, and thence to the part of the third-nerve nucleus which subserves contraction of the pupil. The efferent fibres pass down in the third nerve, to the ciliary ganglion by its short root, and thence pass to the pupil by the short ciliary nerves. There is contraction not only in the illuminated eye but also in the other—consensual contraction. This is probably the result of the decussation of the afferent fibres, and there is probably also a direct connection between the two nuclei. The pupil also contracts during convergence. This is, no doubt, the chief

cause at least of the contraction that occurs during accommodation for near vision, and there is probably a common centre for convergence, accommodation and pupil-contraction.

Dilatation of the pupil is probably the result in large measure of stimulation of the cervical sympathetic. This causes inhibition of the sphincter pupillæ. The latest researches have also shown the existence of a *dilatator pupillæ*. There is probably also some inherent dilating power in the iris itself, for if, when there is complete paralysis of the third nerve, atropine be instilled into the eye, further dilatation will take place. In the lower cervical and upper dorsal part of the spinal cord is the cilio-spinal centre to which fibres pass back from the anterior part of the floor of the aqueduct of Sylvius. The fibres emerge in the first two dorsal nerves, pass in the *rami communicantes* to the cervical sympathetic, thence to the cavernous plexus, the ophthalmic division of the fifth nerve, and along the nasal branch to the long ciliary nerves and thence to the iris. Any sensory stimulus, e.g. prick with a pin, pinching of the skin, &c., will also cause dilatation of the pupil. Emotional states, e.g. fear, produce a similar effect. The condition of hippus is one in which there is rapidly alternating dilatation and contraction of the pupil. It indicates an extremely sensitive, perhaps over-sensitive, state of the contracting and dilating mechanism, and is said to be especially well marked in association with epilepsy. It also occurs in nervous individuals, apart from any structural disease of the nervous system.

The size of the pupil varies in accordance with the degree of light to which it is exposed, the accommodative effect of the eyes, and probably also with mental and emotional states. Unusual degrees of contraction (myosis) and dilatation (mydriasis), and departure from normal reaction to light and during convergence and accommodation, occur as the results of disease. *Myosis* may occur as the result of irritation of the nervous mechanism for contraction, or it may result from paralysis of the mechanism on which dilatation depends. The myosis resulting from irritation may be still further increased by exposure to bright light; mydriatics will overcome it. Paralytic myosis is found most frequently as a result of disease in the cervical region involving the sympathetic path for dilatation of the pupil. The contraction of the pupil may be very great, but its reactions remain. Mydriatics will only partially dilate it. It is only from disease of higher structures that the pupil becomes quite inactive to light as well as contracted, but it may still react during convergence—a condition to be presently alluded to as the Argyll-Robertson pupil. Local conditions should also be remembered as favourable causes of myosis, e.g. excessive vascularity of the iris as in iritis, or when a foreign body is present on the cornea.

Mydriasis or dilatation of the pupil may be *irritative*—the result of irritation of the sympathetic, e.g. from disease in the neck or elsewhere in its path, or as a result of blood-states, which probably act as sympathetic irritants. Anæmia or chlorosis may cause mydriasis by diminishing the vascularity of the iris. Paralytic mydriasis is the result of interference with the pupil-contracting centre or with the path leading to that centre. Thus, disease of the third-nerve nucleus may cause it; so may disease of the nerve itself or of its branches, as in the case of intra-ocular growths.

A very important modification of the pupil as the result of disease is that already alluded to as the *Argyll-Robertson pupil*. In this condition there is often myosis, but this is not invariable. The essential condition is that the pupil while still reacting well during convergence does not react on exposure to light. The pupils also are frequently unequal. Sometimes they are irregular in shape, e.g. oval instead of round. The condition is usually present in both eyes. Rarely it is a unioocular phenomenon, and the unaffected pupil reacts normally. It is usually associated with tabes dorsalis; frequently also it is found in general paralysis of the insane and exceptionally in other central and spinal diseases. It may be present as an isolated phenomenon. It is usually significant of the presence of degenerative disease, or at least of a degenerative tendency in the nervous system. Its frequent presence with conditions regarded as syphilitic or parasymphilitic, and its occasional presence as an isolated phenomenon in patients with a syphilitic history, make it probable that this disease has a close causal connection with it.

Another modification of the ordinary condition occurs after diphtheria. It is almost the converse of the Argyll-Robertson pupil, the reaction to light being present while there is paralysis of accommodation and consequent inability to read, or see clearly at a near distance. Rarely this is an isolated phenomenon. More frequently it is associated with other evidence of post-diphtheritic affection, such as weakness of oculo-motor muscles, weakness of limbs, loss of knee-jerk, nasal voice and affection of the palate. The reaction to light may be absent if the efferent limb of the light-reflex arc is interfered with through affection of the third nerve.

Inequality of pupils may be present in healthy people and is without grave significance. It may be present with the Argyll-Robertson phenomenon. It may also be the result of some irritation of the cervical sympathetic, e.g. from glandular enlargement, &c., and may thus be associated with an increase in the palpebral aperture. If the cervical sympathetic, however, is more profoundly affected, a paralytic condition may be established. This would be indicated by a smaller pupil on the affected side and the presence of a slight degree of ptosis. Inequality or irregularity of pupils may also be the result of anterior or posterior synechie, of operations on the iris, or of developmental defects such as coloboma.

JOHN TWEEDY. JAMES TAYLOR.

PURGATIVES.—SYNON.: Fr. *Médicaments Purgatifs*; Ger. *Abführungsmittel*.

DEFINITION.—Substances which cause intestinal evacuations.

ENUMERATION.—Purgatives are divided into several classes, namely, *drastic, simple, saline, hydragogue, cholagogue, and laxative*. Under the *drastic* purgatives may be classed Colocynth, Croton oil, Gamboge, Jalap, Resin of Podophyllum, Scammony, and Elaterin. Among the *simple* purgatives are Aloes, Castor oil, the juice of various species of Rhamnus (e.g. Buckthorn, Cascara sagrada, and Frangula), Rhubarb and Senna. Under the head *saline* we have neutral salts, especially the Sulphates of Magnesium, Potassium, and Sodium; Citrate and Tartrate of Potassium; Bitartrate of Potassium, Tartarated Soda, and Phosphate of Sodium. *Hydragogues* include Bi-

tartrate of Potassium, Elaterin, and Gamboge. *Cholagogues* comprise Aloes; Mercurial preparations such as Calomel, Blue pill, and Grey powder; Resin of Podophyllum, Iridin, Euonymin, and other substances of the same class. The *laxatives* are small doses of simple purgatives, such as Carbonate of Magnesium, Magnesia, Olive oil and Sulphur, as well as such vegetable substances as contain salines and sugar in considerable proportions, namely, Cassia, Figs, Honey, Manna, Prunes, Tamarinds, and Treacle.

ACTION.—The increased intestinal evacuation produced by purgatives is partly due to acceleration of the peristaltic movements of the intestine, so that the intestinal contents are hurried along more quickly, and less time is allowed for their absorption. Many authorities, especially in Germany, have held this to be the only way in which purgatives act; but there is no doubt that many of them also produce increased secretion from the intestinal glands. The different classes of purgatives affect the intestinal movements and intestinal secretion in different degrees. *Laxatives* and *simple purgatives* act chiefly, if not entirely, by increasing the peristaltic action. Some of the *drastic purgatives* act in both ways; while the *hydragogue cathartics*, as well as the *salines*, especially increase the intestinal secretion. In the case of some of the salines, as acid tartrate of potassium, the secretion is greatly increased, while the peristaltic movement is so little affected that the secretion may lie so long in the intestine as to be again reabsorbed, and the drug therefore fails to produce purgation at all. For this reason it is usual to combine such salines with simple purgatives, which will accelerate the peristalsis, as acid tartrate of potassium with ialap, and sulphate of magnesium with senna.

Besides their direct action upon the bowels, purgatives exert an indirect effect upon the circulation, weakening it, and lowering the pressure of blood within the vessels.

Cholagogue purgatives are those which have a special power to remove bile from the body. They may do this either by stimulating the secretion of the liver, or by quickening the expulsion of bile from the gall-bladder and ducts, so that more bile is poured into the intestine at a time when this is in active movement. The bile is therefore hurried down the intestinal tube, and re-absorption is thus prevented. This appears to be the mode of action of such purgatives as euonymin and iridin. Such mercurial preparations as blue pill and calomel appear to act in a somewhat different way. Experiments, contrary to expectation, have shown that they do not increase the secretion of bile, and yet they are among the most efficient cholagogue purgatives which we possess. Their cholagogue action is probably due to their exerting a special stimulating action upon the duodenum, quickening its peristaltic movements, and thus hurrying down the bile, and preventing its re-absorption. Their beneficial action as cholagogues is greatly increased by the subsequent administration of a saline purgative, which will tend to sweep the bile out of the lower part of the small and the large intestine, and prevent re-absorption from these.

USES.—Purgatives are used, firstly, to remove fecal matters from the intestinal tube. They thus not only prevent the accumulation of such matters, but remove the irritation which their presence produces, and which may evidence itself in disturb-

ances of other organs, for example, headache and malaise. These disagreeable symptoms produced by constipation appear to be partly due to the irritation of the intestinal nerves, producing reflex disturbance of the circulation; but it is probable also that they may be caused in part by the toxic action of poisonous gases, liquids, or solids generated in the intestine by imperfect digestion or decomposition of the food. For such purposes as this we may employ, as we find them necessary, laxatives or simple purgatives. The second use of purgatives is to remove from the body an excess of certain secretions such as bile, and substances which may be contained in them, such as metallic or organic poisons which are excreted in the bile or intestinal mucus. The third use is to remove liquid from the body in cases of dropsy, due either to cardiac or to renal disease. For such purposes we use hydragogue cathartics. The fourth use is to lower the temperature in fever, and for this we chiefly use salines. The *modus operandi* here is not yet well understood. The fifth use of purgatives is to lower the blood-pressure, and thus to prevent the rupture of a blood-vessel, and consequent apoplexy; or to prevent further extravasation in a case where the vessel has already burst, as in hæmorrhage from the lungs.

T. LAUDER BRUNTON.

PURPURA.—Fr. *Purpura*; Ger. *Blutfleckenkrankheit*.—**DEFINITION.**—Purpura is characterised by the appearance of spontaneous extravasations of blood into the skin, and sometimes also by hæmorrhage from mucous surfaces. In many instances it is merely a symptom of some recognised disorder, but in other cases, though strictly speaking it is here also a symptom, it is described as 'a disease,' inasmuch as the primary conditions which give rise to it are unknown.

Characters of the cutaneous Hæmorrhage.—Petechiæ, which are the common feature in all forms of purpura, are red or purple hæmorrhagic spots which become brown as they disappear. They are rounded or irregular in shape, varying in size from a mere speck (as in flea-bite) to a spot half an inch in diameter. They do not fade on pressure. They commonly appear in crops, and they do not increase in size except by confluence. As a rule they are not raised, but occasionally the centre is elevated into a papule which is often paler in colour than the rest of the spot. Occasionally they are linear in shape (vibices). Bruises (ecchymoses) and wheals may be associated with petechiæ; less commonly there are large extravasations into the subcutaneous tissue (hæmatomata).

(a) *Purpura as a Symptom.*—From a review of the conditions under which symptomatic purpura (excluding hæmophilia) occurs we must conclude that it may be produced, (1) by the presence in the blood of bacteria or their poisonous products, (2) by the presence in the blood of some poison of non-bacterial origin either manufactured in the body or introduced from without, (3) by some alteration in the formed or other constituents of the blood, (4) by vaso-motor influence. Thus four classes may be recognised as infectious, toxic, cachectic and nervous.

1. *Infectious.*—In typhus the rash is always purpuric, and a petechial eruption may occur in nearly all the specific fevers, especially small-pox, diphtheria, measles, scarlet fever, plague, yellow

fever, and cerebro-spinal fever. It is rare in typhoid fever. It is not uncommon in malignant endocarditis, pyæmia and septicæmia, and it has been known to occur in association with syphilis, gonorrhœa, malaria, pneumonia and erysipelas.

2. *Toxic*.—The use of iodide of potassium can produce in certain individuals an attack which closely resembles purpura simplex. Poisoning by phosphorus may be attended by cutaneous hæmorrhage. Other drugs such as chloral hydrate, quinine, arsenic, mercury, salicylic acid, copaiba, ergot, belladonna, antipyrine and opium are credited with the production of petechiæ in rare cases. Snake-poison may produce large extravasations of blood into the skin. A petechial eruption may follow the injection of diphtheria-antitoxin. The petechiæ which often occur in the course of chronic nephritis, cirrhosis of the liver, and malignant jaundice should probably be included under this head.

3. *Cachectic*.—Petechiæ are often seen in leucocythæmia, pernicious anæmia and lymphadenoma, and occasionally in connection with generalised sarcoma. In scurvy and scurvy-rickets they may be associated with ecchymoses and hæmatomata. They occasionally occur, especially on the lower extremities, shortly before death from any chronic disease such as phthisis or carcinoma, and even in extreme old age.

4. *Nervous*.—A petechial eruption occasionally appears in locomotor ataxy and more rarely in myelitis, alcoholic neuritis and hysteria.

(b) *Purpura as a Disease*.—Five forms of the disease may be recognised clinically, viz. simple, Werlhof's, arthritic, Henoch's, and febrile purpura respectively. This classification is provisional. The relationship of the various forms is uncertain, and it may be that the distinctions between them are artificial.

ÆTIOLOGY.—In all its forms purpura is almost confined to children and young adults. Both sexes are equally liable. Neither the family history nor the previous history nor the surroundings of the patient throw any light on its causation. The patient is often well fed and apparently in perfect health at the onset of the illness. Occasionally some gastro-intestinal disturbance may immediately precede a simple purpura, and in the more severe forms of the disease an initial tonsillitis is sometimes noted.

PATHOLOGY.—Though the origin of the disease is unknown, the close analogy with idiosyncratic (toxic) and with the purpura of diphtheria and malignant endocarditis (infectious) leads strongly to the belief that in all its forms it comes under one or other of these two heads. In these two examples it must be believed that a circulating poison or an altered state of the blood induces in capillaries and small vessels a subtle widespread preparatory change in the direction of impaired vitality of their walls, and that in this condition various local factors, such as gravity, an unsupported state of a vessel, and sudden alterations in blood-pressure produce rupture and extravasation of blood. No constant microscopical change in the vessels is known. Such gross changes as endarteritis or hyaline and amyloid degeneration of the vessel wall which have been described may be disregarded. Capillary emboli have been found in purpura arising in generalised sarcoma. Bacterial emboli have been found in connection with

petechiæ in certain specific fevers, but they are not constant and they are found without the occurrence of purpura. 'Febrile' purpura bears a close resemblance to a specific fever. No specific micro-organism, however, is known. Streptococci, staphylococci, and a capsulated bacillus belonging to the colon-group have been frequently found in the blood and tissues. But these observations having been mostly made *post-mortem* are of little value, and the blood taken from a vein during life is generally sterile.

MORBID ANATOMY.—After death no change is found which can throw light on the cause of the disease. Petechiæ are found as thickly distributed inside the body as on the skin. The serous membranes, stomach and intestine are particularly affected. Larger hæmorrhages may be found in the brain or elsewhere. The mesenteric glands are swollen. The spleen is often enlarged. All organs are pale and often fatty. Iron-holding pigment may sometimes be demonstrated in the hepatic lobules, and fatty transformation of the marrow in the heads of long bones has occasionally been found. After death from Henoch's purpura the kidneys may present the appearances of acute tubal nephritis. Though diapedesis may play a part in the production of petechiæ, it has been shown by Unna that the hæmorrhage mainly occurs by rupture of vessels, the site being commonly in the lower part of the cutis.

SYMPTOMS AND COURSE.—1. *Simple Purpura*. The appearance of petechiæ first attracts attention. Few at first, they appear in successive crops, so that in a few days nearly the whole body, face, trunk, and extremities may become thickly sprinkled with red, purple, or rust-coloured spots. Here and there large patches may be formed by coalescence. Ecchymoses are readily produced and sometimes hæmatomata develop, especially about the buttocks.

As in other forms of purpura there may be evidence of slight pain in some joints, and perhaps a little œdema may be detected on the backs of the hands and feet. There may be some epistaxis and less commonly some oozing of blood from the gums, but there is no other hæmorrhage from mucous surfaces. In many cases there is no indication of illness apart from the petechial eruption and a slight degree of anæmia. There is sometimes slight pyrexia during the first few days and a little constitutional disturbance, but recovery occurs in one or two weeks, and there is no tendency to recurrence. It is important, however, to note that the early stage of the more fatal forms of purpura may be as mild as a simple purpura, and that prognosis should consequently be reserved, especially when the temperature is above 100° F. at the onset.

2. *Werlhof's Purpura*.—The onset and early symptoms of this form are generally identical with those of simple purpura, and it may reasonably be considered as a later or more severe stage. After a few days or a week, during which petechiæ appear with little impairment of the general health, the hæmorrhagic tendency becomes aggravated and often uncontrollable. The gums ooze freely, epistaxis may be severe, blood may appear in urine, stools and vomit, and there may be bleeding from the uterus and auditory meatus. Retinal hæmorrhages are not uncommon. Bruises and hæmatomata may arise. The anæmia is profound and the

red cells may fall below 1,000,000 per c.mm. The tongue is slightly if at all furred, and there is little or no pyrexia. Such cases may recover, but death frequently results from exhaustion or from hæmorrhage into or upon the surface of the brain. In rare cases the disease proves fatal (*purpura fulminans*) by extensive hæmorrhage in a few days.

3. *Arthritic Purpura*.—The early stage is here marked by pains in joints, especially in the knees, ankles, elbows, and wrists. The joints are tender, though there may be very little external evidence of inflammation. The condition resembles a subacute rheumatism, but its connection with rheumatism is very doubtful. After a few days petechiæ appear, more thickly on the lower extremities than elsewhere, and often aggregated round an affected joint. Ecchymoses are not uncommon. Slight œdema of hands and feet may often be seen. Pyrexia is moderate and may be absent. There is often very little feeling of illness, and there is no tendency to hæmorrhage from mucous surfaces.

The attack subsides in two or three weeks and need not give rise to anxiety; but there is a strong tendency to recurrence. Further attacks may occur at short intervals even when the patient is at rest under treatment. An attack is very apt to occur when he first leaves his bed, and many months may elapse before he can walk freely.

Certain cases (*purpura urticans*) may provisionally be included under this head, in which to the articular pains and petechiæ already described there is added the presence of wheals and extensive œdema. The face in whole or in part may be greatly swollen and discoloured with petechiæ, wheals, or ecchymoses. The hands and feet in the neighbourhood of any painful joint may be similarly affected. In these cases the onset is usually sharper than in the simple arthritic form, the pyrexia is more severe, a greater degree of illness is reached, and the urine may contain blood or albumen. Tonsillitis has been known to precede such an attack.

4. *Henock's Purpura*.—During the first few days there is little or nothing to distinguish this variety from arthritic purpura. But the main feature soon appears in the form of severe attacks of abdominal colic with vomiting and constipation. The pain is undoubtedly severe and may induce collapse. The vomit soon comes to contain blood, diarrhoea succeeds the constipation, and the stools are black with blood, while epistaxis and oozing of blood from the gums are not uncommon. Occasionally an acute nephritis is added, the urine becoming scanty and containing blood and albumen. Pyrexia is slight and may be absent. The spleen has been found swollen after death, and its enlargement has been detected during life. There is a marked tendency to recurrence of such attacks, but recovery may be expected. Occasionally death occurs with all the symptoms of uræmia, and in one recorded case perforation of the stomach proved fatal on the fourth day of a second attack.

5. *Febrile Purpura*.—In nearly all instances of this form of purpura it is the appearance of petechiæ, perhaps with epistaxis and oozing from the gums, which brings the patient under observation. Sometimes the patient states that he has been out of health for some weeks previously, pallor and a feeling of weakness being the main complaints, but sometimes the onset is as sudden as in any specific fever. In any case the patient is already extremely

pale. The petechiæ appear in crops all over the body, but they are often much less numerous than in simple purpura. Ecchymoses are common. Tender raised bluish-red patches, an inch or more in diameter, may appear suddenly, and these often become surmounted by a bulla containing blood-stained serum. On the breaking of this an ulcer is left which is apt to deepen and spread by progressive necrosis. Petechiæ are always present in the mouth, and they are apt to give rise to small ulcers. Gangrene of gums or of the inner surface of the mouth is a common event, and sometimes progressive sloughing of tonsil, palate or epiglottis occurs. The breath is fetid and the submaxillary glands enlarged. Blood continually oozes from the gums, which are pale and not swollen. Epistaxis may necessitate plugging of the nostrils. Retinal hæmorrhages are often seen, but bleeding from gastro-intestinal or urinary tracts is uncommon. Enlargement of the spleen has occasionally been noted and the joints are sometimes slightly painful. The blood shows a progressive diminution of red cells, which may fall below 500,000 per c.mm. Leucocytosis is slight or absent. During the first few days pyrexia is not always present; but the temperature rapidly rises, and for the greater part of the illness it reaches a maximum of 103° or 104° with a daily range of 2° or 3° . It is doubtful if recovery ever occurs. Sometimes the patient falls into the typhoid state, sometimes he remains clear-headed to the end, presenting a blanched appearance and complaining of nothing but extreme weakness. Death generally occurs in the first, second, or third week after the patient comes under treatment, its immediate cause being cardiac failure.

TREATMENT.—In simple purpura no special treatment is required beyond rest in bed, with the administration of arsenic or iron. In the arthritic form anti-rheumatic treatment is commonly adopted, though the benefit derived is doubtful. More important is the maintenance of prolonged rest in bed in view of the strong tendency to recurrence. In Henock's purpura the pain generally necessitates the use of opium. When hæmorrhage becomes a danger, as in Werlhof's disease, calcium chloride, ergot and ergotin, sulphuric acid, acetate of lead, or turpentine must be tried, but they frequently fail to produce any effect. In the febrile form also these drugs have little or no value, and neither quinine nor anti-streptococcic serum has any influence on the temperature. In fact, in the severe forms of purpura there is little to be done beyond combating the cardiac weakness by stimulants, strychnine, and the inhalation of oxygen.

HERBERT P. HAWKINS.

PURRING TREMOR or THRILL.—SYNON.: Fr. *Frémissement Catairè*; Ger. *Schnurren*.—A physical sign felt by the hand applied over the heart or vessels in certain conditions, resembling the sensation conveyed by the purring of a cat. See PHYSICAL EXAMINATION.

PURTON, in Wiltshire.—Saline waters, containing iodine. See MINERAL WATERS.

PURULENT INFECTION.—Infection from the absorption of pyococci, introduced from without, or formed within the body. See PYÆMIA.

PUS.—See INFLAMMATION; and ABSCESS.

PUSTULE. — **SYNON.** : Fr. *Pustule* ; Ger. *Pustel*.—A small abscess in the skin. Vesicles originally containing serum are also apt to become pustules if infected by pyogenic bacteria.

PUSTULE, MALIGNANT. — **SYNON.** : Anthrax ; Fr. *Charbon* ; Ger. *Milsbrand*.

NOMENCLATURE.—Under this head will be considered the various effects of poisoning by the *Bacillus anthracis*. Various names are employed to designate the different clinical forms, both in man and animals. These names were derived from prominent features of the disease, or from its supposed origin before the real cause was discovered.

In bovine animals, the great swelling of the spleen led to the names of 'Splenic fever,' 'Splenic apoplexy,' *mal de rate*, *Milsbrand*, &c. From the peculiar characters of the external lesion in man, and less frequently in animals, the terms 'malignant pustule,' 'anthrax,' 'contagious carbuncle,' &c., were derived. The especial liability of those engaged in certain occupations, especially those involving exposure to contagion, led to the names of 'wool-sorter's disease,' 'hair-comber's disease,' &c. In like manner, the disease as affecting animals has received special names.

The *Bacillus anthracis* (*bactériidie du charbon* ; *Milsbrandbacillus*), as seen in the blood of an animal affected with the disease, occurs in the form of comparatively large rods, whose thickness is about $1.2\ \mu$ to $1.5\ \mu$, and whose length is generally about the diameter of a red corpuscle, though both shorter and much longer forms are seen (see *Coloured Plate*, p. 123). It is non-motile. The rods may be present in enormous numbers, sometimes appearing to equal the red corpuscles in number. In the blood and in the spleen the rods occur singly, or two or three may be joined end to end, but long chains are not usually found. In the pleural exudate and in similar fluids, as well as in cultivations outside the body, the organism grows into long filaments, which may be shown to be made up of a large number of individual bacilli. The rods have slightly rounded or nearly square extremities, and their protoplasm has a homogeneous or slightly granular appearance, but spores are not usually found in the bacilli within the living body.

Cultivation, &c.—The anthrax-bacillus can readily be cultivated outside the body on all the ordinary media. It grows at ordinary temperatures, but much better about 35°C ., the limits of growth being generally given at 12° – 45°C . On certain media its growth has a characteristic appearance.

In puncture-cultivations in peptone-gelatine the growth appears along the needle-track as a somewhat whitish line from which fine lateral offshoots spread out horizontally in the medium, these also in their turn giving off lines of growth, so as to produce an appearance which has been compared to bunches of fine bristles. The lateral stems are longest near the surface of the gelatine. Later, liquefaction begins at the surface and spreads downwards, so that the characteristic features are lost, and the growth appears as a white flocculent mass at the junction of the fluid and solid portions. In plate-cultivations minute white colonies appear, which, on reaching the surface of the gelatine, cause a little area of liquefaction around them. If these superficial colonies are examined with a low-power lens, the periphery is found to have an appearance like masses of curly locks of hair, the

centre of the colony being denser. The appearance is due to the growth of the organism in wavy bundles of long parallel filaments, which in the centre of the colony are interwoven and massed together. On potatoes the growth is abundant, and appears as a thick cream-like mass, which remains chiefly localised and has little tendency to spread on the surface. The growth on other media presents nothing characteristic. The bacillus also flourishes well in broth and other liquid media.

Growth takes place freely only in the presence of oxygen, and best on a neutral or slightly alkaline medium. The bacilli can be coloured readily by Gram's method, and by the various simple aniline stains.

Anthrax-bacilli in the spore-free condition have comparatively little resisting power to chemical antiseptics, &c. When dried they are generally found to be dead after a few days, and they soon die in fluids in the presence of putrefactive bacteria. They are very rapidly killed by moist heat at 65°C ., and by a 1-per-cent. solution of carbolic acid ; the action of the gastric juice is also rapidly fatal to them, and they are therefore generally killed in their passage through the stomachs of animals. The spores, on the other hand, show a remarkable tenacity of life. They can be kept in the dry state for an almost indefinite period of time, and still be capable of growth when placed in suitable conditions. Dry heat at 140°C . must be applied for several hours in order to kill the spores with certainty, and they may be placed in a 5-per-cent. solution of carbolic acid for a considerable time without being killed. Unlike the bacilli in the spore-free condition, they can pass through the stomach unaltered, and thus reach the intestine in a virulent condition.

Relations to the disease.—When inoculated into susceptible animals (such as mice, rabbits, guinea-pigs, &c.) even in the minutest quantities, this organism causes a rapidly fatal disease. The bacilli generally soon reach the blood, and rapidly multiply, so that they may be present in enormous numbers ; in the spleen also they are very numerous in some animals, and this organ in most animals shows marked enlargement. Death generally takes place in one or two days. The other *post-mortem* changes are chiefly those of a severe septicæmia—congestion and cloudy swelling of organs, and often small hæmorrhages, which are in many cases associated with plugging of the capillaries with small masses of the bacilli. At the seat of inoculation there may be inflammatory œdema, with extravasation of blood ; or there may be little change. In sections of the tissues the bacilli are found in enormous numbers in the capillaries (in comparatively small numbers in the larger vessels) ; in fact, so numerous are they in the capillaries of the kidneys, liver, intestines, peritoneum, and lungs, that in stained specimens these may appear as if injected with a coloured material. Animals may be infected by inhalation either of the bacilli or their spores, the former generally producing a pneumonic condition, whereas the latter may be rapidly absorbed, and produce a general infection with comparatively little local change. Infection by the spores may also take place by the intestinal tract, and this is probably the common mode in the natural disease in sheep and cattle. The modes of infection in the human subject, and the lesions produced, are described below. Certain animals enjoy a natural

immunity against the action of the anthrax-bacillus, for example, adult dogs, white rats, many birds, and frogs; though the susceptibility may be modified by altered conditions of temperature.

In view of the enormous numbers of bacilli which may be present in the blood, it was formerly thought that their effects were produced partly mechanically, and partly by their using up the oxygen of the blood. And though they may act in both of these ways, it is now known that, in common with other pathogenic bacteria, the anthrax-bacilli produce specific toxins. For further details on the toxins and antitoxins, see ANTITOXINS; IMMUNITY; and TOXINS.

Anthrax is of very wide distribution throughout the world, especially affecting cattle, deer, sheep, goats, and allied animals; less frequently horses. But it can be communicated to nearly all animals by inoculation, and even to some birds and amphibians. Of mammals, herbivora are especially susceptible; while carnivora are with difficulty inoculated, except when young. Rodents are highly susceptible, and hence are commonly used for test-inoculations.

Although apparently endemic in certain regions, there can be little doubt that this is due to the great persistence of the contagium by means of its spores, and that the recurrence of outbreaks in cattle is due to the careless disposal of carcasses of animals which have died of the disease.

In man, the disease is usually traceable directly to inoculation from the fresh carcass, or from parts, such as the skin or hair, which have been kept in a dry state. Hence butchers, shepherds, and stockmen, who flay the carcass or bury it, are most commonly its victims from the more direct mode of inoculation; while wool-packers and sorters, horsehair-cleaners, workers in felt-manufactories, furriers, tanners, and the like, are exposed to the less direct form. In rarer instances no such mode of contagion can be traced, and it is probable that in these the poison has been conveyed by flies, or similar agencies. The flesh of the dead animals can rarely be suspected, unless it is eaten raw or very imperfectly cooked. Even then, as we know from experiment, large quantities of the bacillus or its spores may do no harm. Butter and milk are alleged by Heusinger to be possible carriers of the poison. Nor is it improbable that the use of water which has been contaminated by wool-waste, bone-dust, or other substances, may convey contagion to cattle. But none of these modes is proved to be common in man, and the spread of contagion from one human subject to another is extremely rare.

It will, however, be shown that in man the disease may be acquired either by inoculation or by inhalation of the dust containing the spores of the bacillus, perhaps also by swallowing them.

DESCRIPTION.—The form of the disease depends on the mode of entrance, and may be internal or external.

Anthrax in Cattle.—This may be briefly described, as of importance in relation to man. Whereas in some animals there are marked external manifestations, either pustular or of the nature of diffuse phlegmonous swellings, with glandular enlargement, such conditions are rare in cattle. In them there may be not only no external changes, but even few perceptible symptoms. Only for a few hours before death may there be evident languor, loss of appetite, and then stupor. But sometimes

animals may be found dead in the morning which had been apparently well the night before. If, however, they are carefully observed, it is usually found that there is marked rise of temperature, followed by coldness and lividity before death.

On examining the carcass, the blood is often found fluid, and may be dark in colour. Sometimes there are glandular swellings and inflammatory oedema, especially around the pharynx, œsophagus, and stomach. But these are more frequently absent, and there are only scattered hæmorrhages here and there, especially in the lungs and heart-wall, and sometimes in the submucous tissues.

The most constant and characteristic condition is the enormous swelling of the spleen, which is engorged with blood, and readily breaks down on slight handling. This splenic enlargement, be it remembered, is constant only in cattle, and may be entirely absent in other animals, or very slight and irregular.

Microscopical examination shows enormous numbers of anthrax-bacilli in the blood, especially in the spleen, lungs, and, next in frequency, in the capillaries of the glomeruli of the kidneys, and sometimes in the liver. In these organs many of the capillaries may appear to be completely filled at parts with bacilli.

It is very important to bear in mind that this abundance and this distribution of the bacilli are very inconstant in other animals. Only guinea-pigs show a similar condition with any constancy. In other animals the bacilli may be few and scattered, being found especially in the capillaries of the lungs and kidneys; or they may be mainly limited to the lymphatics, including the glands and lymph-cavities. Hence, while all parts of the body of cattle which have died of the disease are actively poisonous, it may be difficult to convey the disease from other animals except by inoculation of the fluid from the more affected parts.

Moreover, the activity of the bacillus is rapidly destroyed by decomposition, so that no results may be produced by inoculation from the dead animal when decomposition has set in, unless spore-formation has occurred. This is also true of the human subject.

Spore-formation is sometimes alleged not to occur during life. It may, however, do so in the kidney, if the bacilli pass into the urinary tubules, and also in serous exudations in the pleura and elsewhere, either before or very speedily after death.

Destruction of the carcasses by fire or by deep burial is essential to prevent infection from them.

Anthrax in Man.—This is divisible into two primary forms, *external* and *internal*, which differ not only in the character of the disease, but in mortality and treatment. The external variety may be further described as *malignant pustule proper*; and *anthrax-oedema*. The internal form may be subdivided, according to the part specially affected, into a *broncho-pulmonary* form, of which 'wool-sorter's disease' is the type; and a *gastro-intestinal* form. There may also be cases which do not come strictly under either of these heads, but more closely resemble the disease in cattle, and have been called *Anthracæmia*.

The external form, malignant pustule, is caused by direct inoculation of the skin, or of a superficial mucous membrane, for instance, that of the lip or the eye. In some cases there is only present an

intense inflammatory œdema, which spreads like erysipelas, and causes enlargement of the corresponding lymphatic glands. It is like the condition to be immediately described, differing only in the absence of the characteristic pustule.

Of these cases of so-called *anthrax-œdema*, it need only be said that some may not be due to the anthrax-bacillus, but to the bacillus of malignant œdema. But there is no reason to doubt that a similar condition may be produced by the anthrax-bacillus.

Malignant Pustule Proper.—The typical form, malignant pustule, can usually be traced to direct inoculation. It commences as a small red swelling or pimple at the point of inoculation. It may give rise to slight irritation, or be attended by burning or itching, but may be absolutely painless. It occurs commonly on the face, neck, hand, or arm, that is, on the parts most exposed to direct inoculation in handling a carcass. The exact period after inoculation at which it appears is variable, owing doubtless to the presence or absence of a small wound: it may be from a few hours to two or three days, possibly longer. Once formed, it rapidly extends, so that in a few hours a large red swelling may be present. There then usually form upon the surface of the swelling one or more vesicles, which, if not ruptured, may reach a considerable size by confluence. By this time a more marked localised swelling has formed; and if the vesicle or bleb ruptures, or is opened, it discharges a watery fluid, either clear or slightly turbid, and often deeply blood-stained. Beneath the vesicle is a dark-red area, which usually dries, producing a dark-brown or black eschar, seated on a much raised, angry red, indurated base. Further vesicles frequently develop around this eschar; they often form a narrow ring surrounding it. The central necrosed area enlarges, and by this time a widespread livid red area and extensive brawny œdema extend from it, sometimes involving the whole arm or face. The corresponding lymphatic glands usually enlarge, often to a great degree, forming a dense mass, owing to the surrounding inflammatory œdema. The central black eschar may enlarge till it reaches one-third to three-quarters of an inch in width; and if the patient survives it may still be distinct, seated on the raised indurated inflamed base, at the end of ten days or more. These characters will be explained in considering the microscopic changes.

In some cases, this typical lesion is indistinct, the brawny red swelling, with some irregular vesicles, being alone observed. When recovery occurs, the swelling subsides more or less rapidly, the eschar separates, and healing takes place rapidly; but the swelling of the glands may last for some time.

Similar carbuncular swellings, more or less typical in character, have also been observed, though rarely, after an internal or general infection, and not as the result of local inoculation.

Microscopic Anatomy of the Malignant Pustule. The descriptions given by various writers of the changes seen with the microscope are somewhat conflicting. The differences are partly accounted for by the different site or stage of the specimens examined. From an examination of numerous specimens at various stages, the changes appear to be fairly constant in their mode of evolution.

In the early stages there may be no indication of

abrasion of the skin, and it is probable that the virus may gain access by the hair-follicles. There is at first infiltration of the corium, with inflammatory exudation, especially in its papillary portion. The epithelium becomes partially separated, and rises in the form of vesicles, as in an ordinary blister, the deeper layers alone remaining attached. Bacilli are found, though scantily, in the serous exudate.

There then ensues a deeper-seated exudation, penetrating to the deeper layers of the skin. This exudation is accompanied by hæmorrhage, and the tissue-elements swell and undergo rapid necrosis. Both the epidermis and the infiltrated superficial layers of the skin necrose and form a coagulated mass, which constitutes the dark central area of the pustule. Around this the processes of vesication and of subjacent hæmorrhagic infiltration go on spreading, both laterally and in depth.

When fully formed, the central part forms a dense, horny-looking, blood-stained mass; beneath and around it are great infiltration, hæmorrhages, and commencing necrosis. Farther out there is œdema and great vascular engorgement, often with but scanty exudation of leucocytes.

In sections stained for bacilli, these are found almost solely in the vesicles and in the lymphatics around the necrosed area. They occur as scanty clusters, and are often most abundant in the more superficial lymphatics. They can be traced for a certain distance from the area of intense inflammation. Their absence in the necrosed area is doubtless explained by the necrosis in which they share. They have occasionally been seen in large numbers among the epithelial cells and in the superficial layers during the early stages of infection.

In more advanced conditions, leucocytic infiltration becomes abundant around the necrosed area.

Some have supposed that the peculiar characters of the pustule are due to the presence of other micro-organisms, which aid in their destruction and in the necrosis of the tissues. It cannot be said that microscopic examination of pustules in the earlier stages lends much support to this view. They point rather to an intense irritant action of the products of the growth of the bacteria on the tissues and vessels, differing markedly from their effects as seen in the blood-vessels of the cow, in which almost no such action is apparent. When the surface has necrosed, other bacteria do no doubt enter, but they do not usually penetrate deeply into the tissues.

The normal limitation to the lymphatics, and their scanty number, the spread mainly in the superficial lymphatics, and the early and intense protective exudation and necrosis which they excite, are all favourable to recovery; and afford strong grounds for the avoidance of any treatment which will break through these natural barriers and produce risk of contamination of the blood.

SYMPTOMS.—The condition of the patient is very varied. Even in cases which prove fatal, there may be few or no general symptoms at first, and he may even continue at work with a large distinct pustule; or there may be only a slight degree of prostration, with a little fever. In some cases recovery ensues without the development of any general symptoms.

More commonly the development of the pustule and of the lymphatic infection are attended by prostration, fever, and some of the symptoms of

general blood-poisoning. The exact character of the symptoms, which will be more fully described under the head of 'Internal Anthrax,' appears to depend on the system which is more specially involved. In some, gastro-intestinal derangement, with severe vomiting, is prominent. In others, delirium, convulsions, and coma occur. In rarer cases, the course resembles that of the bronchial form to be described later.

The temperature is said by some not to be elevated. This may possibly be true in some cases in which there is no general infection, but it is not confirmed by cases investigated by Spear and the writer, or by the numerous medical men under whose care these patients were, and who had carefully watched other cases. The elevation may be slight at first, and in the more advanced condition the surface-temperature may be subnormal; but the rectal temperature is elevated. It may rise to from 101° to 105° F., and in one fatal case it rose to nearly 107° . This point is of considerable importance in diagnosis.

Recovery may take place without special treatment, even where severe constitutional symptoms have supervened. The mortality appears to be about one in four of cases treated without incision. Convalescence may be rapid and complete, or prolonged debility may result.

In some cases, the diffuse cellulitis which spreads from the seat of inoculation may be followed by suppuration, either diffuse phlegmonous infiltration or localised abscess, especially in the glands. The occurrence of diffuse phlegmonous cellulitis appears to be commoner in a class of cases in which the carbuncular swelling is not fully developed, but forms only a small swelling, which usually vesicates. In these cases, also, blebs may form more widely over the inflamed skin, and extensive desquamation of the cuticle may accompany recovery.

TREATMENT.—When any local lesion forms in a person who is known to be exposed to infection, treatment should be at once commenced. If small, the pimple should be incised with a very sharp knife, and suction immediately applied by means of a cupping-glass or artificial leech; the part should then be thoroughly washed with a strong antiseptic, such as solution of biniodide of mercury (1 per cent.) in excess of iodide of potassium, or corrosive sublimate of the same strength, preferably combined with peroxide of hydrogen to prevent coagulation.

If the condition is evidently anthrax, or there has been known inoculation, the more radical method of complete excision and subsequent cauterisation is probably desirable. Of the various methods which may be used, excision is perhaps the best, if it can be done speedily and with little disturbance of the part; but it must be borne in mind that the great risk to the patient's life is in the entrance of the bacilli into the blood-stream, and that the greatest precautions must be taken to avoid this. During the operation the wound should be kept irrigated with antiseptic lotion, preferably biniodide or perchloride of mercury, 1 in 5,000.

Bleeding from the wound may be encouraged by a loose bandage above, in the case of the arm or hand, the object being to prevent the entrance of bacilli into the veins. Arterial bleeding may of course be controlled in the usual way. Antiseptics should be speedily applied. Of these, strong carbolic acid or fuming nitric acid is often recommended; but the caustic action of these so limits

their sphere that probably sublimate or biniodide is to be recommended, except where a caustic action is desired owing to the difficulty of complete excision, for instance, on the face. For a dressing, biniodide solution 1 in 3,000 to 5,000 in 30 per cent. glycerine in water is especially to be recommended; and if applied on several layers of lint, covered with oiled silk, forms a sort of poultice which aids to draw out the lymph. Some recommend carbolic acid or other antiseptics, but these are comparatively ineffective. Oozing from the wound should be encouraged rather than checked.

The diffuse cellulitis may, if necessary, be treated by superficial linear incisions or scarifications, the parts being immediately washed with biniodide lotion, 1 in 2,000. A large poultice of iodide-of-starch paste, to which a solution of biniodide of mercury in a large excess of potassium iodide has been added, in such proportion as to make the poultice of the strength of 1 in 3,000 of biniodide, should then be applied.

The rationale of this treatment lies in the fact that the bacilli lie mainly in the lymphatics of the superficial layers of the corium. The risk of promoting their entrance into the blood must always be borne in mind. Surgical interference is therefore to be deprecated, unless the disinfectant treatment can also be carried out.

Internally, the treatment must consist in supporting the strength, especially by concentrated animal diet, and perhaps by large doses of quinine. Strophanthus should be given if the heart's action becomes feeble.

Internal Anthrax.—This term is more strictly applicable to those cases in which the starting-point is internal, usually by entrance of the virus into the respiratory or alimentary passages. But the conditions which follow general infection from an external lesion are closely analogous.

Of the forms which internal anthrax assumes, the *bronchial* and *gastro-intestinal* are the most typical. Internal inoculation may, no doubt, occur in the mouth, pharynx, &c., as well as in the stomach or intestine. Some have supposed that there may be an entrance into the blood, such as appears to occur in cattle, causing a general blood-poisoning without local lesion. But the practical evidence in favour of this view is small, and it is very doubtful if it is really true of cattle.

In those exposed to infection by the inhalation of dust charged with anthrax-spores, as wool-sorters, the form of disease is usually bronchial. In all the cases investigated by the writer this was the form observed; and further investigation of wool-sorter's disease by others since that time has fully confirmed the observations then made, both as to the form of disease and the microscopic lesions.

1. Bronchial Form.—**Wool-sorter's Disease.**—In this form inoculation takes place by the inhalation of dust from wool or hair containing the spores of the anthrax-bacillus. The site of inoculation is in the lower part of the trachea and the large bronchi. Local lesions closely resembling those of malignant pustule are here produced in the mucous membrane, and thence the virus spreads by the lymphatics to the bronchial and mediastinal glands. These become greatly swollen, and often the seat of hæmorrhages. Thence infection, with intense inflammatory œdema, spreads to the connective tissue of the mediastinum, and possibly upwards to the neck, to the root of the lungs, and

sometimes to the pericardium. Although the pleura are rarely inflamed, the pleural sacs become filled with serum, and the lungs collapse. Such are the constant lesions. But the virus may spread to other organs, if once it enters the blood, and may then produce hæmorrhages or inflammatory exudations in them, with consequent symptoms. The alimentary canal, the peritoneum, the brain and its membranes, may be especially affected. But death often occurs before they have become involved.

ANATOMICAL CHARACTERS. — Marked lividity is common, often being present even during life. Some swelling may be seen in the lower part of the neck, though rarely. The blood is dark-coloured, and coagulates imperfectly.

On opening the thorax, some emphysema may be present in the anterior mediastinum. But constantly there is seen a diffuse infiltration of the mediastinal tissues, either pale and gelatinous, or deeply blood-stained, or with scattered hæmorrhages. Both pleural cavities contain a large quantity of fluid, if the two layers were not previously adherent; the fluid is usually pale and clear, or very slightly turbid. Rarely it is blood-stained. The quantity in each pleura may be from two to four pints. The serous membrane is usually free from all trace of inflammation, though this occurs in rare cases. The pericardium also often contains an excess of fluid. If inflammation is present, it rarely extends to the epicardium.

The lungs are collapsed; they may show minute scattered hæmorrhages, or small patches of broncho-pneumonia, but this not commonly.

The mediastinal and bronchial glands are greatly swollen, and the hæmorrhage in them may make them look like clots. Extensive infiltration of the whole of the connective tissue around them is present, reaching sometimes up into the neck.

The fluid squeezed from this tissue may contain bacilli, but both here and in the pleural fluid they may be very scanty.

On opening the trachea and bronchi, they are found to contain blood-stained frothy fluid. Towards the lower part of the trachea and in the main bronchi the mucous membrane is irregularly swollen and blood-stained in patches. In addition, hæmorrhages are seen in the mucous membrane, forming raised spots. This condition is usually limited to the main bronchi, but may extend into their primary divisions.

On microscopical examination, the most marked changes correspond to the hæmorrhagic areas. The hæmorrhage lies for the most part immediately beneath the basement-membrane, which may have ruptured, the blood forming a layer beneath the detached epithelium. It may also penetrate deeply into the mucous and submucous coats. All the tissues beneath and around are infiltrated with serum, and their tissue-elements thus separated, but not usually necrotic. The blood-vessels are dilated, but leucocytic emigration is scanty. Dense masses of bacilli are found in the exudation, and escaping to the surface. They also surround and extend in the lymphatics, perivascular and other, and may be traced in them to the deeper lymphatic plexuses. Similar masses of bacilli may also be seen in the superficial layers of the mucosa, where as yet little or no inflammation has occurred. They often lie immediately beneath the basement-membrane. Little or no catarrhal change is seen, the

epithelium simply desquamating; if catarrh is present, it is usually of older date.

In the bronchial glands, the condition is often masked by the extensive hæmorrhage. Bacilli are

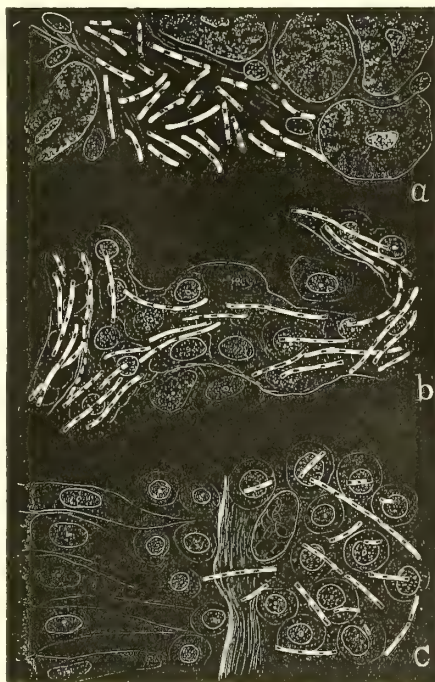


FIG. 1.—Anthrax-Bacilli. *a*. In heart-wall of a cow. *b*. In the pulmonary capillaries of a cow. *c*. In the human bronchial mucous membrane, in a case of 'wool-sorter's disease.' $\times 800$ diam.

found, often scantily, especially in the lymph-sinuses of the cortex. Sometimes they are very abundant, and may then also appear in the capillaries.

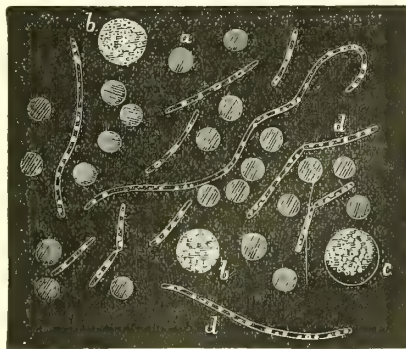


FIG. 2.—Bacilli from the fluid exuded from the lung in a case of internal anthrax. *a*. Red blood-corpuscles. *b* and *c*. Large granular corpuscles from the lung. *d*. Bacilli of various lengths, containing highly refracting granules, or fully formed spores. \times about 700 diam.

In the lungs, scattered patches of hæmorrhage may be present, mainly due to the inhalation of blood from the bronchi. These may also produce areas of lobular collapse and broncho-pneumonia.

But for the most part, nothing but the general collapse and congestive œdema are found in the lungs, and bacilli are often not to be discovered, except here and there in the lymphatics near the root. This common absence of bacilli in the pulmonary capillaries proper is in marked contrast with the condition usual in cattle.

In other organs, the changes are diverse, and may be absent. There is not usually much cloudy swelling or other indication of septicæmia, though they may be present. Minute hæmorrhages are often apparent; or, in the pia mater, and generally in serous membranes, diffuse extravasations. In the stomach and intestines submucous exudation and scattered hæmorrhage may occur.

For the most part, the spleen is but slightly swollen. Examination of the blood and of other organs, such as the kidney, in which bacilli are commonly abundant in many animals, is usually attended with negative results, though in some cases bacilli may be found by careful search. Inoculation with these parts or with the blood may also produce no effect. But if susceptible animals are inoculated with the bronchial fluid, or other parts which contain bacilli, typical anthrax is produced.

The lesions in the trachea and bronchi are essentially similar to the skin-lesion in malignant pustule, allowing for differences in structure.

The multiple nature of the lesion in the bronchi and trachea is no doubt explained by the ready spread of the bacilli in the lymphatics of the mucous membrane, and by secondary inoculation with the bacilli exuded on its surface.

The pleural exudation, which forms so constant a feature in typical cases, usually without any inflammation of the serous membrane, is probably due to the enormous and rapid obstruction of the lymph-channels in the glands and mediastinum. The swelling and obstruction of the bronchi must also aid the collapse of the lungs, by preventing the entrance of air.

A knowledge of these facts is of essential importance in relation to treatment.

SYMPTOMS AND COURSE.—While presenting considerable variety in the additional symptoms and mode of termination, nearly all cases present characteristic features due to the peculiar lesions just described. To these may be added the effects of nerve-irritation, especially of the phrenic nerves, the vagi, and the cardiac and pulmonary plexuses.

Only the more important of the clinical phenomena will be described. The earlier symptoms are usually great lassitude, chilliness, sometimes with rigors, and mental depression, sometimes with insomnia. Headache, dizziness, and nausea are often present. Vomiting may occur early, and be a prominent symptom, possibly due to the nerve-irritation above mentioned. With these or other symptoms there is commonly complained of at an early period a peculiar sense of constriction of the chest, especially at the lower part, and a want of breath. This is sometimes accompanied by marked pain or cramp-like feeling, and is aggravated by exertion. Cramps and tingling sensations in the limbs, palpitation, flushings, and perspirations may occur.

After a period varying from one to five days, during which there may be intervals of improvement, there ensue more severe symptoms, and the patient has to leave work and often to take to bed. These more pronounced symptoms include greatly

increased prostration, accelerated and difficult respiration, usually rise of temperature, and a rapid and feeble pulse. In addition, the sense of painful constriction of the chest is often marked. Cough, which may be slight or almost absent at first, now usually becomes marked; it is dry, hacking, and painful. The sputa are usually scanty, frothy, and blood-stained, but may become abundant and hæmorrhagic. Bacilli may be found in the sputa.

Sometimes death occurs from syncope within twenty-four hours after the patient gives up work, nothing but the dyspnoea and cyanosis being observed.

More commonly the dyspnoea and cyanosis increase, the face becomes pallid or livid, the pulse and respiration are accelerated, and the general symptoms become more marked. Headache is sometimes intense; vomiting may be frequent and urgent, and indications of the involvement of other organs show themselves. Of these, gastro-intestinal irritation is frequent; there may be severe colic; hæmorrhage from the bowels may occur, though infrequently. In fact, the disease may assume the gastro-intestinal form. Or the cerebral or spinal centres may be especially involved; hence delirium and somnolence, more rarely convulsions or tetanic spasms. Death may be preceded by coma, or the patient may be conscious to the last.

In many cases scattered hæmorrhages in the skin or subcutaneous tissue, or diffuse discolourations, are observed during life, but they may be entirely absent. In rare cases typical carbuncles have been observed. Remissions sometimes occur, to be followed by more marked prostration. In nearly all cases, even if the temperature is high, there is great coldness of the extremities, with indications of asphyxia.

The range of temperature is very variable, and it shows also marked oscillations; but observations on the surface-temperature are not reliable, owing to the great chilling of the surface.

Of the physical signs, those of pleural effusion are the most marked, but the effusion sometimes occurs very rapidly, and may not appear till shortly before death. Bronchitic sounds, &c., may of course also be present.

Even when severe symptoms have developed, recovery may take place. In the cases collected by Spear, four out of twenty-three recovered. And in many cases which presented marked initial symptoms, and from their relations to infection were in all probability cases of the disease, the more severe symptoms did not develop. In some, secondary changes in the lungs appear to have caused death at a much later period.

Death usually occurs within from two to six days from the onset of marked symptoms. Eleven of the twenty-three cases mentioned died within three days. Only two died at eight days, and these from septic complications.

When recovery occurs, convalescence is often very prolonged.

TREATMENT.—Prophylactic measures are by far the most important. Of these the careful separation and disinfection by superheated steam of all wool or hair which shows suspicious characters, such as excess of blood-staining, dirt, &c., or which comes from districts where anthrax is common, are of prime importance. The characters and origin of such wool or hair are well known to those engaged in such trades. Next in importance are the

thorough ventilation of the rooms in which the wool is sorted, and the greatest care in washing the hands, changing the clothes, &c. The effect of such precautions, which were first enforced in 1884, has been to reduce the mortality from the disease in the Bradford district to two per annum during the years from 1884 to 1890 inclusive; whereas in the period of ten months at the time of our inquiry in 1879-80 there were nine cases of malignant pustule with two deaths, and twenty-three cases of internal anthrax with nineteen deaths.

Of the treatment to be adopted when the fully pronounced symptoms are present, little can be said. The evacuation of the fluid from the pleuræ is certainly indicated, and the internal administration of strong meat-broths, stimulants, and cardiac tonics, especially straphanthus. But when once the virus has entered the blood, it is probable that little can be done beyond dealing with complications as they arise.

Prophylactic inoculation does not appear to be applicable to man. Moreover, it does not appear that one attack affords protection in man, since internal anthrax has been fatal in some who have suffered previously from malignant pustule. For some points bearing on the subject of protective inoculation, and the toxins and antitoxins of anthrax, see IMMUNITY.

2. Gastro-Intestinal and other Forms.—Of the other forms of internal anthrax it need only be said that they appear to be far rarer, and that their symptoms and treatment correspond with those described above, when they occur as complications of the bronchial form. A few cases are on record in which the disease has followed eating parts of animals which have died from anthrax, as flesh, liver, &c. In these cases lesions were found in the stomach, intestines, &c., corresponding to those described in the bronchi in wool-sorter's disease; the mesenteric glands showing a condition resembling that of the bronchial glands.

The symptoms in such cases are mainly abdominal, and the disease is usually rapidly fatal.

It is questionable whether many of the cases which have been described as gastro-intestinal, or *mycosis intestinalis*, and especially where there is a phlegmonous gastritis, are not due to other bacteria. In none of the cases which the writer has examined have anthrax-bacilli been found; in some only micrococci were present; in others, bacilli which, though bearing a superficial resemblance to anthrax-bacilli, proved to possess entirely different characters. Microscopic examination and inoculation of animals can alone decide in any particular case.

W. S. GREENFIELD.

PUTRID FEVER.—A synonym for typhus fever. See TYPHUS FEVER.

PYÆMIA (πύον, pus; and αἷμα, blood).—SYNON. : Purulent Infection : Fr. *Pyohémie*; Ger. *Pyohämie*; *Pyämie*.

A condition of blood-poisoning which gives rise to fever, accompanied either by severe visceral inflammations and congestions, or by certain local lesions, which are chiefly venous thrombosis, embolic abscesses in the viscera, acute suppurations of the serous membranes and joints, multiple abscesses in the connective tissue, and eruptions upon the skin. The disease is usually, but not always, sequential to a wound or injury.

PYELITIS (πύελος, a vessel).—SYNON. : Fr. *Pyélite*; Ger. *Nierenbeckenentzündung*.—Inflammation of the pelvis of the kidney. See KIDNEYS, Diseases of, p. 834.

PYLEPHLEBITIS (πύλη, gate = *porta*; φλέψ, a vein).—Inflammation of the branches of the portal vein, often associated with thrombosis. See PORTAL THROMBOSIS.

PYLORUS.—See STOMACH, Dilatation of, Inflammation of, Malignant Disease of, and Neuroses of.

PYOGENIC BACTERIA.—While nearly all the pathogenic bacteria possess the power, under certain conditions, of initiating purely pyogenic processes in place of or in addition to their specific lesions (e.g. *Bacillus tuberculosis* and *Pneumococcus*), there are a few organisms which commonly express their pathogenicity by the formation of pus; these are usually grouped together under the title of *Pyogenic Bacteria*, as distinct from those which only occasionally assume a pyogenic rôle.

The most important of the pyogenic bacteria in point of frequency are the *Staphylococcus pyogenes aureus*, the *Staphylococcus pyogenes citreus*, the *Staphylococcus pyogenes albus*, and the *Streptococcus pyogenes longus*; next, and of less constant occurrence, the *Bacillus pyocyaneus*, the *Bacillus* of Friedlander, and the *Bacillus coli communis*; and lastly, and usually in certain well-defined situations, the *Gonococcus*, the *Koch-Weeks bacillus*, and others.

The members of the last of these groups will be found under special headings, or the headings of the specific diseases with which they are causally associated.

Staphylococcus Pyogenes Aureus.—This organism occurs in pus as small zoogloea-masses, each containing from five to twenty spherical bodies, which have remained in close apposition after the completion of the process of fission. These masses of cocci are arranged for the most part between the pus-cells, but occasionally some may be demonstrated within the cells. The individual cocci measure from 0.6 to 0.9 μ in diameter. They stain readily with all the basic aniline dyes, and retain their colour perfectly when treated by Gram's method. The *Staphylococcus aureus* is a non-motile, non-sporing, liquefying, facultative anaërobe, readily cultivated upon nutrient media both at 22° C. and at 37° C.—this latter being the optimum temperature. Its thermal death-point is 58° C. In spite of the fact that spore-formation does not occur, the organism possesses considerable powers of resistance; a solution of perchloride of mercury (1 : 1000) does not effectually destroy the coccus in 24 hours, while a solution, five times as strong (1 : 200), of the same germicide requires from fifteen minutes to one hour to effect its destruction. The action of desiccation and light are also resisted for considerable periods. Cultivations upon artificial media will retain their vitality for years. The chromogenicity of the organism is best brought out in cultivations upon solid media, which have been freely supplied with oxygen and incubated at a temperature of 24° C.

Cultural Characteristics.—In gelatine 'stab-culture' the growth at first shows along the track of the needle as a faint whitish line composed of minute spherical colonies, discrete or confluent,

which rapidly increase in size. By the end of about three days liquefaction of the medium begins to form a broad funnel filled with turbid gelatine. The liquefaction then proceeds rapidly, some of the growth sinking to the apex of the funnel and gradually becoming bright orange-yellow in colour. In gelatine 'shake-cultures' growth occurs uniformly throughout the substance of the medium, liquefaction commencing from the surface. No formation of gas takes place in sugar-gelatine. In bouillon at 37° C. a uniform turbidity of the medium is observed in less than 24 hours, and may be accompanied by slight pellicle-formation. Eventually the greater part of the growth is deposited and assumes a yellowish tint. The indol-reaction can usually be obtained in from 48 to 72 hours. Upon the surface of nutrient agar, a thick, spreading, moist, shining, cream-coloured layer forms, which afterwards becomes orange-coloured. Upon the surface of inspissated blood-serum the growth resembles that upon agar, the medium not being liquefied. Litmus-milk is coagulated by the coccus in the course of its growth, and becomes strongly acid in reaction. Upon sterilised potato, especially if cultivated at the ordinary temperature of the laboratory, the coccus produces a thick whitish layer which soon becomes yellowish in colour and finally bright golden.

The *Staphylococcus aureus* is pathogenic for the rabbit and the mouse (especially the white mouse), the result of inoculation varying, among other factors, with the method employed. Subcutaneous injection is usually followed by abscess-formation, sometimes by septicæmia. Intravenous injections if the dose is fairly large, usually result in death from general septicæmia. By appropriate means peritonitis, meningitis, arthritis or osteomyelitis may be produced.

***Staphylococcus Pyogenes Citreus*.**—This organism differs from the *Staphylococcus pyogenes aureus* only in the production of a lemon-coloured instead of an orange pigment. It is said, too, that the *Staphylococcus citreus* is less virulent than the *aureus*.

***Staphylococcus Pyogenes Albus*.**—This is morphologically identical with the *Staphylococcus aureus*. Culturally it only differs in that its growth is creamy-white. Its virulence, as a rule, is markedly less than that of the *aureus*.

***Streptococcus Pyogenes Longus*.**—**SYNON.**: *Streptococcus Erysipelatosus*. The *Streptococcus longus* occurs in pus and serum, as chains of spherical cocci, but sometimes as diplococci, lying free between the leucocytes, and never within them. The chains vary in length, containing from ten to fifty cocci, and are frequently convoluted; the individual organisms are usually small, averaging 0.5 μ in diameter, though some are occasionally quite twice that size, the latter being probably just about to undergo fission. This streptococcus stains readily with the basic aniline dyes, and, like the pyogenic staphylococci, retains the stain after treatment by Gram's method. The *Streptococcus longus* is a non-motile, non-sporing, non-liquefying facultative anaërobe, which, under laboratory conditions, is best cultivated at the temperature of the body; it will also grow though with difficulty at the ordinary temperature of the room. Its vitality is not great, cultivations upon artificial media dying out in the course of a few weeks; its virulence, too, is quickly lost, and in like manner

its resistance to germicidal agents is but slight—e.g., 2-per-cent. solution of phenol is sufficient to completely destroy the *Streptococcus* within a few minutes. Its thermal death-point is 54° C.

Cultural Characteristics.—Upon the surface of nutrient gelatine a scanty growth of minute, raised, hemispherical, whitish, opaque colonies appears in 48 to 60 hours. The colonies are scattered and discrete, and increase but little in size with age. The medium is not liquefied. Upon the surface of agar the colonies remain discrete, and closely resemble those formed upon gelatine. They are, however, larger, more opaque and greyish, and the base of each colony is frequently surrounded by a delicate halo. The growth upon inspissated blood-serum is similar to that upon agar. Upon the surface of potato, the growth is usually described as 'invisible,' and, when most evident, only appears as a moist-looking area. In bouillon, growth occurs as fine flocculi which settle on the most dependent part of the wall of the tube, or sink to the bottom; the supernatant medium remains clear. The indol-reaction is absent. Luxuriant growth takes place in litmus-milk, accompanied by an acid reaction; clotting may or may not be observed. This *Streptococcus* is pathogenic for the rabbit, guinea-pig and mouse. When injected subcutaneously a local abscess is usually produced, but apparently identical races of streptococci differ in virulence to such a degree that the result of similar inoculations may vary from a transient erythema to death from streptococcic septicæmia.

***Bacillus Pyocyanus*.**—The *Bacillus pyocyanus* was first isolated from blue pus, the colour of which was due to this bacillus. The *Bacillus pyocyanus* occurs as short slender rods with rounded ends, often so short as to resemble cocci; also united in pairs or short chains, and in long threads. Individual elements average 0.3 to 1 μ in diameter by 2 to 3 μ long; are actively motile, and each rod is furnished with a single terminal flagellum. The bacillus stains well with all the ordinary aniline dyes, but is completely decolourised when treated by Gram's method. The *Bacillus pyocyanus* is a non-sporing, liquefying, facultative anaërobe. It grows readily on artificial media at the room-temperature, but its optimum temperature is 37° C. Its thermal death-point is reached at 55° C. The formation of pigment only takes place in the presence of oxygen; at least two pigments are elaborated, the one yellow, *pyoxanthose*, the other blue, *pyocyanin*, and to these pigments the so-called 'blue pus' owes its colour.

Cultural Characteristics.—In gelatine plate-cultures the colonies rapidly develop as small flat plaques, having a dark green central spot surrounded by a delicate radiating lighter zone, the surrounding medium being fluorescent. Liquefaction commences at about three days in the form of a funnel. In gelatine stab-cultures liquefaction at first proceeds in the form of a funnel, the liquid gelatine being turbid and fluorescent, and containing whitish flocculi in suspension. Later the liquefaction extends to the edges of the tube, the flocculi sink and the liquid portion is separated from the solid gelatine by a horizontal line, the deeper parts of the medium away from the air lacking the fluorescence of the upper portion. Shake-cultivations in sugar-gelatine do not give rise to the formation of gas. Upon the surface of agar a moist whitish layer develops, which later becomes greenish in colour,

while the medium itself is fluorescent and appears green by reflected, and orange by transmitted light; subsequently this colour darkens to blue-green or black. Upon the surface of inspissated blood-serum a thick moist layer appears, dirty white at first but afterwards becoming brown, and the medium itself is liquefied. In bouillon, growth occurs as a uniform turbidity accompanied by the formation of a delicate pellicle of a light greenish colour. The indole-reaction can be obtained in about seventy-two hours. Litmus-milk is at first clotted, showing an acid reaction; later the coagulum is dissolved. Upon the surface of potato a light brown or green slimy layer is formed confined to the needle-track, and the substance of the medium is discoloured. The *Bacillus pyocyaneus* is pathogenic for the rabbit and the guinea-pig. Subcutaneous inoculation of a small dose is followed by local abscess; a large dose causes œdema, purulent infiltration of the tissues, and death. Intraperitoneal injections usually cause death in from twenty-four to thirty-six hours from general septicæmia.

Bacillus of Friedländer.—**SYNON.** : *Pneumobacillus*.—This bacillus was first isolated from the lung-tissue in a case of lobar pneumonia. The bacilli, as seen in pus and sputum, are short oval rods, arranged singly, in pairs, or even in short chains of from four to eight individuals; occasionally bacilli are so short as closely to approach the spherical—so closely so, in fact, that the organism was originally described as a coccus. Each element, in combination, is surrounded by a clear refractile space, indicating the presence of a capsule similar to that possessed by the pneumococcus, and capable of demonstration by identical staining-methods. When cultivated upon artificial media the pneumobacillus loses its capsule, while the variations in size and shape of the rods are so marked as to render the polymorphism of the organism one of its most striking characters. The bacillus stains readily with all the aniline dyes, but is completely decolourised when treated by Gram's method. The organism is a non-motile, non-sporing, non-liquefying, facultative anaërobe; easily cultivated upon artificial media, and growing freely at the room-temperature, although its optimum temperature is one more nearly approaching to that of the body. Its thermal death-point is reached at 55° C. It possesses considerable vitality. Cultivations upon artificial media, more than two years old, have yielded successful subcultures.

Cultural Characteristics.—In gelatine streak-cultures the pneumobacillus produces a thick elevated opaque white growth with a smooth shining porcelain-like surface. In gelatine stab-cultures it shows as a typical nail-growth, the head being composed of a hemispherical mass of the size of a split pea, and the shaft, of discrete or confluent colonies down to the extremity of the needle-puncture. A shake-culture in sugar-gelatine demonstrates the abundant production of gas. On the surface of slanted agar a growth is produced resembling that upon gelatine, but somewhat more slimy, and showing a tendency to slip down the inclined surface to the bottom of the tube. Upon inspissated blood-serum a similar condition is produced. The medium is not liquefied. Bouillon exhibits a uniform turbidity in about twenty-four hours, and the presence of indol may be demonstrated in from forty-eight to seventy-two hours. Upon the surface of potato a slimy buff-coloured

growth appears which in the course of a few days becomes a dirty brownish-yellow, while the substance of the medium assumes a greyish tint. Litmus-milk shows clotting in from twenty-four to forty-eight hours, accompanied by the production of a strongly acid reaction. Friedländer's bacillus is pathogenic for ordinary animals, though in varying degrees. When inoculated under the skin of the mouse death ensues in from three to five days from general septicæmia, and the organism can be recovered in pure culture from the blood in the heart. Inoculations into the subcutaneous tissue of the guinea-pig are usually followed by similar results, although recovery occurs fairly often. The rabbit is still more resistant, and recovery is the rule. Cultivations upon artificial media appear to retain their virulence unimpaired for long periods.

Bacillus Coli Communis.—*See p. 121.*

JOHN EYRE.

PYONEPHRITIS.—Inflammation of the kidney, leading to the formation of abscess. *See* KIDNEYS, Diseases of, pp. 821, 823, 834.

PYOPNEUMOTHORAX.—A morbid condition of the pleural cavity, in which it contains both pus and gas. *See* PLEURA, Diseases of.

PYORRHOEA ALVEOLARIS.—*See* TEETH, Diseases of.

PYRENEES.—*See* BIARRITZ; BAGNÈRES-DE-BIGORRE; EAUX-BONNES; EAUX-CHAUDES; LUCHON and PAU; and CLIMATE, Treatment of Disease by.

PYREXIA (πύρ, fire; and ἔχω, I have).—This word is sometimes employed as a synonym for fever; but it is more properly applied to the elevation of the body-heat which is one of the phenomena of fever. *See* FEVER; and TEMPERATURE.

PYRMONT, in Germany.—Iron waters and salt waters. *See* MINERAL WATERS.

PYROMANIA.—A name which has been given to insanity when the patient manifests a propensity to incendiarism. Its claim to be regarded as a special form of insanity has not been established. *See* INSANITY.

PYROSIS (πυρῶς, I burn).—**SYNON.** : Water-brash; Fr. *Pyrosis*; Ger. *Sodbrennen*.

This term was originally applied to the eructation of strongly acid fluid from the stomach accompanied by a burning pain at the epigastrium. By many writers the use of the term has been extended so as to include the expulsion (1) of swallowed saliva collected in the œsophagus above its spasmodically contracted lower end, and (2) of neutral or slightly acid fluid from the stomach some hours after the chyme should have passed into the duodenum. Moreover, the strongly acid fluid to which the term was originally limited is not always of the same character. In some cases the acidity is due to free hydrochloric acid; in others, to butyric and other organic acids. A term of such uncertain meaning should certainly be discarded.

H. MONTAGUE MURRAY.

PYSTJAU, in Hungary.—Thermal sulphur-waters. *See* MINERAL WATERS.

PYURIA (πῦον, pus; and οὔρον, the urine).—A name for a condition of the urine in which it contains pus. *See* URINE, Morbid Conditions of.

Q

QUARANTINE (Ital. *quaranta*, forty). —
SYNON. : Fr. *Quarantaine*; Ger. *Quarantäne*.

DEFINITION.—The enforced isolation of individuals and certain objects coming, either by sea or by land, from a place where dangerous communicable disease is presumably or actually present, with a view of limiting the spread of the malady. The objects liable to quarantine include—on the assumption of their being apt to carry the contagion or infection of the disease—the luggage and personal effects of the individuals isolated, certain articles of merchandise, and ships; and, in land-quarantine, carriages and other vehicles. Sometimes entire communities and districts are subjected to quarantine.

In Great Britain and Ireland quarantine was until 1896 carried out under an Act of Parliament passed in the reign of George IV. (6 George IV. cap. 78). It was practised, and that only to a limited extent, solely with a view of relieving our maritime commerce from disabilities which would else be imposed upon it by other countries, in which quarantine is regarded as an essential part of the public-health administration. The regulation of quarantine was not a function of the department of the government which is concerned with the sanitary administration of the kingdom (the Local Government Board), but of the Privy Council, aided by the Board of Trade, the subject being dealt with as an international commercial question. In what follows an authoritative official memorandum of the late Dr. E. C. Seaton (formerly medical officer of the Local Government Board) on the subject is closely adhered to.

The Quarantine Act provided for land-quarantine and the quarantine of inland waters, as well as for maritime quarantine—internal and external quarantine, so to speak. It does not appear that internal quarantine has ever been enforced in this country. Since the Act was passed maritime quarantine alone has been practised, and this has been applied to three diseases only, all of them infectious diseases of foreign origin, namely, plague, cholera, and yellow fever. Of plague there has been, until quite recently, no question in English ports for fifty years, except a slight alarm in 1879, consequent upon an outbreak in South-eastern Russia in the province of Astrakhan. Against cholera quarantine had not been enforced since 1858, its futility as a precautionary measure in this country having been abundantly manifested. Yellow fever was the sole disease subjected to it in our ports, and this, as already stated, not from the medical necessity, but from the commercial exigency of the case. The only quarantine establishment remaining in England—that at the Motherbank—was maintained in respect of that disease. Infectious diseases habitually current in this country, such as small-pox and scarlet fever, notwithstanding that the phraseology of the Quarantine Act covered any 'infectious disease or distemper,' had always been in practice exempt from quarantine, and dealt with under the general sanitary law of the kingdom. It appears to have been recognised that measures, primarily designed to prevent the introduction into the

country of diseases only coming to us from abroad, and which involved international considerations, would be misapplied if used for the purpose of preventing the importation of diseases, ordinarily existing here, the limitation of which, and not the exclusion, could alone be in question.

In 1896 the Quarantine Act of George IV. was repealed, and the measures which had been applied to cholera were extended to yellow fever and plague. Before discussing these measures we may conveniently refer to the attitude of foreign Powers to the subject of quarantine.

In reference to cholera, foreign countries have, during recent years, shown a readiness to modify the requirements of quarantine, and substitute shorter periods of detention for those formerly insisted on. Conferences between representatives of the Powers have been held at Paris in 1852, Constantinople in 1866, Vienna in 1874, Rome in 1885, Venice in 1892 (the conclusions of which were subsequently modified at Paris), and Dresden in 1893, when Great Britain was represented by Mr. Strachey, the resident Minister at Dresden, Sir Richard Thorne Thorne, K. C. B., Medical Officer of the Local Government Board, and Mr. H. Farnall, C.M.G., of the Foreign Office. A further conference was held in Paris in 1894 for the purpose of devising measures for preventing the spread of cholera by way of the Red Sea and the Persian Gulf. Inasmuch as the conclusions of the conference of 1893 have been accepted by the English Government, it will be convenient to give a brief account of their nature.

I. Notification to Foreign Governments.—A cholera-centre¹ is to be notified to all the Governments signing this agreement. Isolated cases are not necessarily to be notified. Communications concerning progress of disease are to be made at least once a week.

II. Circumstances under which Districts are to be deemed Infected.—A district is to be considered infected when the existence of a cholera-centre has been officially stated; no longer infected when no deaths or fresh cases have occurred within five days, and the measures for necessary disinfection have been taken.

[The word 'district' is applied to any part of a country placed under a well-defined administration.]

III. Limitation to Infected Districts of Preventive Measures.—Measures for limiting the spread of the epidemic are only to apply to products of the infected districts, and not then if the products have left the country five days before the outbreak began.

IV. Merchandise or Dangerous Articles considered from the point of view of (1) Prohibition of Importation and Transit and (2) of Disinfection.

¹ The term actually used is *foyer*, and is thus referred to by the *Lancet* of July 15, 1893, p. 152: 'Practically the use of the term *foyer* means that if there are isolated cases of cholera the French Government will not notify; but if cases are grouped together and form a sort of kernel—a centre, a focus, radiating, scattering around the germs of disease—then the French Government will notify.'

1. The only things to be forbidden are: (a) body-linen, clothes in use, bedding (except when considered as luggage or when moving from one house to another, when they are to be specially dealt with); (b) rags and drills; but the following are not to be prohibited: rags compressed by hydraulic power, which are forwarded as wholesale merchandise and labelled with name of the place of destination, &c.; and clean clippings coming direct from spinning, weaving, making-up, or bleaching establishments; artificial wool, and fresh paper-havings. Goods are not to be detained in quarantine on the frontiers of countries.

2. Disinfection of baggage is to be obligatory in respect of such things as clothes, dirty linen, &c., coming from an infected district, if the local sanitary authority consider them contaminated. Merchandise is to be disinfected so as to damage as little as possible; each State is to arrange the method of disinfection. Letters or printed matter are not to be disinfected.

V. *Measures at Land-Frontiers.*—*Railways, Travellers.*—Infected carriages are not to be retained at frontier towns—if soiled, they are to be detached for disinfection wherever possible. Land-quarantine is no longer to be established. Only persons suffering from cholera and those attacked with a choleraform disease are to be detained. The officials of the railway are to see if all travellers are well—if ill, a medical man is to be called in. Inspection is to be made, if possible, at the Custom-house stations so as to hinder travellers as little as possible. On arrival at destination, travellers coming from infected districts are to be subject to five days' observation dating from the time of their departure from such districts. This, of course, does not apply in the case of Great Britain, which has no land frontier.

Special measures may have to be taken with regard to bohemians, vagabonds, emigrants, and persons travelling in parties.

VI. *Regulation of Frontier-Traffic.*—Frontier-traffic arrangements are to be left to special measures to be taken by the neighbouring (adjoining) countries.

VII. *Waterways.*—The arrangements for regulating traffic are to be left to the States on river banks. Those regulations in force in Germany in 1892 are recommended.

VIII. *Measures to be taken at Ports.*—A ship is to be considered *infected*, which has cholera on board or if it has had fresh cases of cholera during the preceding seven days; to be considered as *suspected* where there have been cases on board though not during the last seven days; to be considered as *healthy*, though coming from an infected port, if having had neither case of cholera nor death from cholera on board before the departure, during the voyage, or since arriving.

Infected ships are to submit to the following:—

(1) The sick are to be disembarked and isolated.
(2) The others are to disembark and remain under observation during a period not exceeding five days. The signature of Great Britain to the Dresden Convention was conditional that for the purposes of England the 'observation' should, both for infected and suspected ships, be in the homes of the persons under observation instead of in any specially provided place.] (3) Dirty linen and clothes, &c., belonging to passengers are to be disinfected, if

deemed contaminated by the sanitary authority, as well as the ship or part of ship infected.

Suspected ships are to submit to—

(1) Medical inspection; (2) disinfection; (3) removal of bilge-water after being disinfected, and substitution of good drinking-water in place of that stored on board.

It is recommended that passengers should be under observation not more than five days from the date of arrival, and that the disembarkation of the crew should be delayed.

Healthy vessels are to be allowed free pratique. It is, however, permissible that the authority of the port may in the case of healthy vessels adopt the same measures as in the case of suspected vessels, except that the period of observation (five days) should date from the time of departure of the vessel instead of the date of arrival.

The authority of the port is to take into consideration, before ordering these measures to be adopted, the presence of a doctor and of a disinfecting-stove on board the vessel. Special precautions are to be taken on vessels crowded with emigrants, and insubstantial vessels. Goods arriving by sea can only be treated similarly to those arriving by land. All ships refusing to submit to the port arrangements are free to put out to sea. They may only unload after the necessary precautions have been taken—(1) isolation; (2) removal of bilge-water; (3) substitution of good water. No passengers are to disembark unless willing to submit to the port regulations. Every country should provide at least one port on each of its coasts with the organisation and apparatus for receiving ships whatever their sanitary state. Coasting vessels are to be subject to special regulations, to be arranged between the countries interested.

Special measures are to be taken with regard to vessels coming from an infected port and going up the Danube. Until the town of Soulina is provided with a good water-supply, all boats going up the river are to be subjected to rigorous hygienic measures.

Overcrowding of passengers is to be strictly forbidden.

In reference to plague, a conference of the Powers was held in Venice in 1897. The principal questions considered were—(a) the measures for preventing the spread of plague out of Europe, including the measures to be taken on board pilgrim-ships; and (b) the measures to be taken in Europe. The principles which governed the decisions of the Dresden Conference in respect of cholera were again accepted, as applicable to plague, some modifications being made in view of the longer incubative period of the latter disease. Thus infected vessels were made to include vessels on which disease had occurred within ten days of arrival.

It is interesting to observe that, while foreign countries are thus agreeing to regulations which approximate in some degree to those which are in force in Great Britain, British colonies, having independent control in respect of this subject, maintain a more rigid system of quarantine, and do not limit its application to diseases which in this country are subject to the quarantine law. Thus Malta, Gibraltar, and Cyprus, as well as the more distant colonies, adhere to the old quarantine system; and, indeed, the West Indian colonies have (in 1893) enacted a quarantine law, to which reference may be made as illustrative of the action of the colonies in this respect.

The measures which have, in England, been substituted for quarantine against cholera, yellow fever, and plague, consist in a 'system of medical inspection,' the details of which are set forth in the Order of the Local Government Board, dated the 9th November, 1896, and which include and extend the provisions of antecedent Orders relating to cholera only. This plan differs from 'quarantine' in the following essential respects:—

(a) It affects only (1) such ships as have been ascertained to be, or as there is reasonable ground to suspect of being, *infected* with cholera or choleraic diarrhoea, yellow fever, or plague; no vessel being deemed infected unless there has been actual occurrence of cholera or of choleraic diarrhoea, or of yellow fever or plague on board in the course of the voyage, or during the stay of the vessel in the port of departure, or in a port in the course of the voyage. (2) Ships not infected with cholera or yellow fever or plague, but having passengers on board who are in a filthy or otherwise unwholesome condition. (3) Ships coming from a place infected with cholera, yellow fever, or plague.

(b) It provides for the detention of the vessel so long as is necessary for the requirements of a medical inspection; for dealing with the sick (if any) in the manner it prescribes; and for carrying out the processes of disinfection.

Any person suffering from cholera, yellow fever, or plague must be removed, if his condition admit of it, to some hospital, or other suitable place for that purpose appointed by the sanitary authority; and no person so removed may leave such hospital or place until the medical officer of health has certified that he is free from the disease. If any person suffering from cholera, yellow fever, or plague cannot be removed, the ship must remain subject to the control of the medical officer of health. If any person is certified by the medical officer of health to be suffering from any illness which he suspects to be cholera, yellow fever, or plague, such person may be removed to some hospital, or other suitable place provided by the sanitary authority, or be detained on board the ship for any period not exceeding two days. If within this time he is found to be so suffering, he will be dealt with as above.

(c) It subjects the healthy on board to detention only for such length of time as admits of their state of health being determined by medical examination, or until they have satisfied the medical officer of health as to their names, places of destination, and addresses at such places. The name and address of any such person is to be forthwith given by the medical officer of health to the clerk to the sanitary authority, who is required to transmit the same to the local authority of the district in which the place of destination of such person is situate. If any such person shall within forty-eight hours arrive at a place of destination or address other than that he has given, he is required upon arrival to notify the same to the medical officer of health of the district.

In the case of ships infected with cholera, yellow fever, or plague, or which have come from a place infected with any of these diseases, the medical officer of health may direct the bilge-water to be pumped out before such ship enters any dock or basin; and, on the sanitary authority providing a proper supply of water for drinking and cooking purposes for persons on board the ship, he may direct all casks or tanks on board the ship containing water for the use of such persons to be emptied;

and the master is required to cause these directions to be carried into effect.

In addition to the above Orders, others have been issued from time to time regulating the admission of rags from foreign countries infected with cholera.

The measures for dealing with the sick under the Orders of the Local Government Board are but an adaptation to a particular exigency of the principles of sanitary administration with regard to infectious diseases which are in force under the general sanitary law of the kingdom.

But though quarantine has no present practical existence in this country, and all other infectious diseases are dealt with either under the general sanitary law of the country, or such modification of it as has been just described with regard to cholera, yellow fever, and plague, the machinery of the repealed Quarantine Act, for obtaining information as to the existence of infectious diseases on board foreign-coming ships, is made available for dealing with all diseases of that kind. The questions, which it is the duty of the Customs to put to the masters of all such vessels, embrace all infectious diseases; and, in the event of any such disease being found to exist on board, or to have existed in the course of the voyage, the Customs officer is required to detain the vessel, and to forward the information with the least practicable delay to the sanitary authority of the port. In regard to cholera, yellow fever, and plague, moreover, both the Customs and the sanitary authority have certain powers of detaining the vessel specified in the Order of the Local Government Board above referred to.

The provisions under Articles 12, 13, and 14 of the Order of the Local Government Board of 1896, as to the mode of dealing with persons who may arrive from abroad infected with cholera, yellow fever, or plague, will be better understood if a succinct statement be made of the ordinary provisions of the law with regard to infectious diseases in England. The authorities which have to administer that law, as now existing under the Public Health Act, 1875, are the urban, rural, and port sanitary authorities of the districts into which the whole kingdom is divided. These authorities are empowered—

(a) To provide hospitals or temporary places for the reception of the sick (section 131);

(b) Where a hospital or place for such purpose is provided, to remove thither by order of any justice, on a certificate signed by a legally qualified medical practitioner, any person who is suffering from any dangerous infectious disorder, and is without proper lodging or accommodation, or lodged in a room occupied by more than one family, *or on board any ship or vessel* (section 124);

(c) To make regulations (to be approved by the Local Government Board) for removing to any hospital, to which the local authority is entitled to remove patients, and for keeping in such hospital so long as may be necessary, any persons brought within their district by any ship or boat who are infected with a dangerous infectious disorder (section 125);

(d) To provide and maintain a carriage or carriages suitable* for the conveyance of persons suffering under any infectious disorder (section 123);

(e) To cleanse and disinfect infected premises, and articles therein; to destroy any bedding, clothing, or other articles which have been exposed to infection from dangerous infectious disorder, giving compensation for the same; and to provide all necessary means for the disinfection of infected things (sections 120, 121, 122);

(f) To take proceedings against (1) any person who, while suffering from any dangerous infectious disorder, wilfully exposes himself, without proper precautions against spreading the said disorder, in any street, public place, shop, inn, or public conveyance, or enters into any public conveyance without previously notifying to the owner, conductor, or driver thereof that he is so suffering; or, (2) any person who, being in charge of any person so suffering, so exposes such sufferer; or (3) any person who gives, lends, sells, transmits, or exposes without previous disinfection, any bedding, clothing, rags, or other things which have been exposed to infection from any such disorder; or (4) any owner or driver of a public conveyance who shall not have immediately provided for the disinfection of such conveyance, after it has to his knowledge conveyed any person suffering from a dangerous infectious disorder; or (5) the owner of any house, in which any person has been suffering from any dangerous infectious disorder, who shall knowingly let it or part of it for hire, without having previously disinfected it, and all articles therein liable to retain infection, to the satisfaction of a legally qualified medical man; or (6) any person who, showing for the purpose of letting for hire any house or part of a house, shall make false statements as to the existence of infectious disease therein, or within six weeks previously (the several acts here enumerated constituting offences liable to penalty under the Public Health Act, ss. 126, 128, 129);

(g) To provide mortuaries, and to obtain the removal thither, by order of a justice, of the body of anyone who has died of any infectious disease, which is retained in a room where persons live or sleep, or of any dead body in such a state as to endanger the health of the inmates of the house or room in which it is retained (sections 141, 142);

(h) To make inspection of their district, with a view to ascertain what nuisances exist calling for abatement under the powers of the Act, and to enforce the provisions of this Act in order to abate the same (section 92): a provision which extends to shipping—any ship or vessel lying in any river, harbour, or other water, within the district of a sanitary authority, being subject to the jurisdiction of that authority, in the same manner as if it were a house within such district;

(i) Finally, to appoint a medical officer of health, inspector of nuisances, or several of those officers, according to the needs of the district, and other requisite officers to aid them in the proper and efficient execution of the Act (sections 189, 190). The duties of the medical officer of health and of the inspector of nuisances, when (as is the case in the greater number of instances) the assent of the Local Government Board has to be given to their appointment, are set forth in Orders of the Board dated March 1880.

Beyond the powers conferred upon Sanitary Authorities by the Public Health Act, 1875, other powers of considerable importance in relation to the subject under discussion are given by the notification of Infectious Diseases Act, 1900, and can be

obtained by Sanitary Authorities adopting the provisions of the Infectious Diseases (Prevention) Act of 1890.

Under the former Act, it is obligatory on the authority to require every medical practitioner attending or called in to visit a patient to give notice to the Medical Officer of Health of the district if the patient be suffering from 'small-pox, cholera, diphtheria, membranous croup, erysipelas, the disease known as scarlatina or scarlet fever, and the fevers known by any of the following names—typhus, typhoid, enteric, relapsing, continued, or puerperal,' as well as any other infectious disease which the authority by special resolution may require. The duty of giving notice to the Sanitary Authority also devolves upon the head of the family to which the patient belongs, and in his default the nearest relative of the patient present in the building or being in attendance on the patient, and in default of any such person, the occupier of the building. The notification of cases of plague has recently been made obligatory by a regulation made by the Local Government Board under powers conferred on that Board by the Public Health Act, for dealing with epidemic disease.

Under the latter Act, powers can be obtained for—

(a) Prohibiting the supply within a district of milk from a dairy believed to be productive of infectious disease.

(b) Prohibiting any person from ceasing to occupy a house, room, or part of a house, which has been occupied by a person suffering from infectious disease, without having such house, room, or part of a house, and all articles therein likely to retain infection, disinfected to the satisfaction of a registered medical practitioner, or without giving notice of the previous existence of such infectious disease to the owner, and prohibiting any such person from giving any false answer to the owner or to any person negotiating for the hire of such house, room, or part of a house, as to the fact of there having within six weeks previously been therein any person suffering from any infectious disease.

(c) Prohibiting any person from retaining without the sanction in writing of the Medical Officer of Health or a registered medical practitioner, elsewhere than in a mortuary or in a room not used at the time as a dwelling-place, sleeping-, or work-room, for more than forty-eight hours, the body of any person who has died of any infectious disease.

(d) Prohibiting any person from removing from any hospital or place of temporary accommodation for the sick, except for the purpose of burial, the body of any person who has died from infectious disease, if the Medical Officer of Health or any other registered medical practitioner has certified he is of opinion that it is desirable, in order to prevent the risk of communicating any infectious disease, or of spreading infection, that such body should not be removed from such hospital or place except for the purpose of being forthwith buried. Under such circumstances the body when removed must be taken directly to a place of burial or to a mortuary.

(e) Empowering a justice of the peace on the application of the Medical Officer of Health to order the removal to a mortuary, and to be buried, the body of any person who has died of infectious disease, which has remained for more than forty-

eight hours without the sanction of the Medical Officer of Health or a registered medical practitioner in a room used at the time as a dwelling-place, sleeping-place, or work-room.

(f) Requiring any person who desires to remove in a public conveyance, other than a hearse, the body of any person who has died from infectious disease, to give notice to the owner or driver, and requiring such owner or driver to disinfect the conveyance.

(g) Authorising a justice of the peace to make an order directing the detention in a hospital, at the cost of the authorities, of any person suffering from infectious disease, who would not on leaving such hospital be provided with lodging or accommodation in which proper precautions could be taken to prevent the spreading of the disorder of such person.

(h) Prohibiting the casting into any ash-pit, ash-tub, or other receptacle for the deposit of refuse matter, any infectious rubbish without previous disinfection.

(i) Empowering the local authority to provide, free of charge, temporary shelter or house-accommodation, with any necessary attendants, for the members of any family in which any infectious disease has appeared, who have been compelled to leave their dwelling for the purpose of enabling such dwelling to be disinfected by such authority.

The general powers above enumerated, if exercised duly and with reasonable diligence, are held sufficient to provide for the exigencies which may arise in our ports from the introduction of infectious diseases by ships, whether the disease be current in this country or be of foreign origin not naturalised here; but in the case of a non-naturalised disease, such as cholera, yellow fever, or plague, certain additional securities are taken by the Order of the Local Government Board previously referred to. The general powers, moreover, which are available against the importation of infectious diseases by shipping, are available also, and have on occasions been used, against their exportation in like way to other places.

SHIRLEY F. MURPHY.

QUARTAN (*quartus*, the fourth).—A form of ague, in which the paroxysm returns after an intermission of two days. See **MALARIAL DISEASE**.

QUEENSTOWN, in South of Ireland.—Mild, not relaxing, winter climate. Southern exposure, with shelter. Mean winter temperature 44.1° F. See **CLIMATE**, Treatment of Disease by.

QUINCKE'S DISEASE.—See **ANGIO-NEUROTIC ŒDEMA**.

QUINISM.—SYNON.: Cinchonism; Fr. *Quinisme*; Ger. *Cinchonismus*.

DEFINITION.—A group of symptoms, chiefly connected with the nervous system, produced by the presence of quinine in the blood.

ANATOMICAL CHARACTERS.—In the rare cases in man in which death has been due to quinism, *post-mortem* examination has revealed only the appearances which are common to every case of gradual suspension of respiration, namely, accumulation of dark blood in the internal veins. The same has been found in experiments on animals.

SYMPTOMS.—Large doses of quinine prove fatal by paralysing, firstly, the brain and respiratory

centre; secondly, the heart. Smaller doses may produce various effects on the different organs.

On the whole, quinine does not disturb *digestion*; on the contrary, of all alkaloids tested on this point (of course in the shape of a readily soluble salt), quinine, when given in small doses, alone promotes digestion. Undoubtedly it may often cause vomiting, but for this there may be three reasons—the excessively bitter taste, the effect of a salt not easily soluble when there is a deficiency of normal hydrochloric acid in the stomach, and the unusual influence of quinine on the brain. The first two reasons can easily be avoided, and also the third, if too large doses are not given at once to susceptible patients. The intolerance of the brain soon ceases. It is advisable to tell the patient that vomiting may perhaps follow on the first dose, but that that must not prevent the second being taken—then only a little nausea will ensue, and after the third dose neither. In whooping-cough the first effect of quinine is even to stop vomiting, and improvement of other symptoms follows.

The local irritation of the stomach often depends on the use of the sulphate, while the hydrochloride is easily borne. The sulphate is soluble in about 800 parts of distilled water, the hydrochloride in about 36 parts. Free hydrochloric acid renders them both equally soluble; but when this acid is deficient in the gastric secretion, as is the case in most fevers, the water alone may sufficiently dissolve the hydrochloride, but not so the sulphate. It remains longer than it should as a foreign body in the stomach, and causes irritation. It is an old mistake to prescribe the sulphate, merely because it happened to be the quinine-salt first introduced. The hydrochloride not only irritates the stomach less, but contains also a greater percentage of the base than the sulphate does (83 to 74). These advantages compensate for a somewhat higher price.

The *toxic* effects of quinine, so often mentioned, are the result of large doses. They manifest themselves as deafness, noises in the ears—humming, or resembling the roar of a distant waterfall, the ringing of bells, or the striking of a clock—sickness, heaviness in the limbs, retching, vomiting, and inclination to sleep. Quinine-intoxication can, of course, become dangerous, should the dose have been too large. It shows itself as paralysis of the nerve-centres, and later as paralysis of the heart. Irritation no longer causes the slightest contraction; one could imagine that the heart was poisoned by digitalis: in animals at any rate this condition has been seen by the writer. The skin is pale and cold, and the temperature of the blood, measured in the rectum, may show an enormous depression.

A man of forty-five, who suffered from constipation, took by mistake three drachms of sulphate of quinine, instead of the same quantity of cream of tartar at a single dose. In an hour, pains came on in the head and stomach, giddiness, and general weakness, followed by unconsciousness. The face was pale; the lips blue and cold, as also the limbs; the pulse was still regular, but slow and almost imperceptible; the respiration languid and shallow; the pupils much dilated; vision and hearing almost gone, even after return of consciousness. The medical attendant, who was called after eight hours, wrapped up the whole body in hot towels, and used frictions of the skin and internal stimulants. Improvement ensued after a few hours, and increased

steadily during the following days ; but even on the fifth day the patient was unable to leave his bed for more than half an hour. The general weakness and that of sight and hearing improved, but did not disappear completely for a long time.

In a French military hospital (1885) a soldier swallowed by mistake a 5-per-cent. solution of sulphate of quinine, instead of a similar solution of sulphate of magnesium, equal to twelve grammes (180 grains) of the former. When he complained of the disagreeable bitter taste and of the humming in the ears, the attendant thought proper to swallow the same quantity. Both were taken very ill after less than half an hour ; both suffered from great general apathy and weakness of the heart. The soldier recovered, because a large portion of the solution of quinine was rejected by spontaneous vomiting ; the attendant died before the lapse of four hours, with the symptoms of paralysis of the nervous centres.

Experiments on rabbits and dogs have proved to the writer that, under the influence of large doses of quinine, the respiration is first paralysed, and that life can be preserved at this stage by artificial respiration. Then the heart becomes paralysed by direct influence of the quinine on it, and death ensues. The physician should therefore ask himself, in every case where he considers large doses of quinine desirable, whether an existing or threatening weakness of the respiration or the circulation might prove an objection to the prescription. Speedy death has been observed in typhoid fever in patients or convalescents after about 20 grains of quinine ; and even less may prove dangerous. In such cases 10 grains is a maximum dose, and stimulants should be administered simultaneously, e.g. camphor dissolved in almond oil, in repeated doses of 2-4 grains.

In connection with the influence of quinine on the heart, it must be mentioned here that its action in *small* doses consists of a slight irritation of the organ, probably of its substance. The vagus of the heart has no connection with the effect ; it requires strong doses of quinine to produce any depressing effects on this nerve.

The disorders of *hearing* caused by quinine generally last only for some hours or a few days ; but severe cases are also reported. A man of thirty-seven took 20 grains of hydrochloride of quinine in one dose for ague. The ague left him, but he almost immediately experienced loud noises in the ears, pain in the left ear, heaviness of the head, fits of giddiness, and intense deafness. The ticking of a watch, and the humming of a tuning-fork, were no longer heard when they were pressed to the skull. Eighty grains of salicylate of sodium, taken within five hours (one must ask for what purpose), aggravated all the symptoms. Aural treatment during several months produced but partial improvement. Hearing was slightly better, but still bad.

Investigations on the hearing of healthy men have yielded some interesting results. The temperature of the outer ear, after a dose of 17 grains of hydrochloride of quinine, sank 0.56° C. on an average in twelve experiments within two or two and a half hours. The external meatus and the tympanum were not hyperæmic—on the contrary, they were pale—when the effect of the quinine was at its height. It does not follow that this must always be the case. Some people may get inflammation of the tympanic cavity ; others, again, in-

flammation of the skin, as will be described presently. Inflammatory extravasations were produced artificially in the *canalis cochleæ spiralis* and other parts of the inner ear in a cat, by dosing it with quinine. The writer knows from his own observation that this animal is certainly very sensitive to quinine. It is only in exceptional instances that quinine produces lasting bad effect on the hearing of human beings.

Disturbances of *vision* after large doses of quinine have often been observed. They are caused by direct paralysis of the optic nerve, not by dimming of the refractive media. A. von Graefe has described two cases, both malarial patients. In one case 360 grains had been taken during several weeks ; in the other case 500 grains. In the first case weakness of sight ensued ; in the second case blindness. Both cases lasted several months. Improvement began of itself, and was apparently aided by artificial bleeding from the temples. Many similar cases have since been reported.

The following case is more recent : A woman of thirty-five had aborted, with symptoms of septic endometritis. Cold baths and quinine—80 grains in the course of thirty hours—were employed to control the fever. An eclamptic fit ensued, and immediately afterwards complete loss of hearing and sight. The urine was free from albumen ; the pupils were much dilated and fixed, the refractive media clear, the retina almost bloodless and perfectly insensible to strong light. Consciousness returned a day after the attack ; hearing within the first few days. The blindness of the peripheral parts of the retina remained permanently ; that of the central parts disappeared slowly within six months. Colour-blindness, which had been total when the sensibility to light returned, persisted partly.

It is evident, however, from the reports of cases, that disturbances of vision in patients treated with quinine may often be caused to a great extent by the illness itself, and are then incorrectly attributed to the remedy.

Transient *affections of the skin* after the ingestion of quinine are especially frequent. They present themselves chiefly as eczema, roseola, erythema, urticaria, and purpura. Here only a few instances need be mentioned. Four cases of purpura hæmorrhagica have been described. The most remarkable point about them was their appearance after only small doses ; for instance, after 2 or 3 grains every six hours. According to another medical report, a lady of forty got œdema of the face and limbs, with violent erythematous eruptions, followed by peeling, after taking quinine. A repetition of the medicine caused the same symptoms.

The *kidneys and bladder* do not remain insensible to the alkaloid. Given in large doses it may cause albuminuria and catarrh of the bladder. The latter has appeared with violent fever after 60 grains in one day. Cases have been communicated where a few grains of quinine caused bloody urine, jaundice, and fever, apparently quite independently of the malaria for which it was ordered. This is less singular than the skin-eruptions, as the greater part of the quinine which leaves the body passes through the kidneys. The points of particular interest to us regarding this secretion may be noticed here. G. Kerner recovered 80, 90, and 96 per cent. of the ingested quinine from the urine. The excretion of the hydrochloride of quinine

began as early as fifteen minutes after its ingestion, was most active in the twelfth hour (30 per cent.), and lasted till the forty-eighth hour, when 1 per cent. still appeared. The sulphate of quinine was first traceable in the urine after forty-five minutes, and showed itself—1 per cent. only—till the sixtieth hour. The greater portion of both quinine-salts passes out in the amorphous modification; a smaller part becomes oxidised.

Abortion and premature birth are often attributed to quinine. It seems to the writer, after carefully reading the literature of the subject, that these results are mostly due to the illnesses for which the quinine has been given. As it is a distinct protoplasmic poison, one must admit the possibility that when given in daily long-continued small doses in pregnancy it acts with specific energy on the tender protoplasm of the fœtus, whose gradual decay would lead to evacuation of the uterus. At all events chronic quinism, even of a mild character, is to be avoided under such circumstances, and regarded with suspicion.

Several cases of so-called contrary quinine-effect, that is, a real *febrile attack*, without apparent inflammation of any organ, have been published from time to time. How they originate is altogether unknown. Even relatively small doses may cause them. Blackwater Fever has been attributed to the action of quinine. See BLACKWATER FEVER, p. 143.

Increase of the *general reflex excitability* has also been described as an unusual effect of small doses of quinine. Epileptic patients are said to react to quinine by increase of their fits in number and severity. This would correspond more with the facts derived from experiments on animals, than the assertion that quinine diminishes greatly the reflex function of the spinal cord. Such a diminution takes place only when one gives large doses, dangerous to life. Then the reflex function of the spinal cord ceases at the same time as all other functions.

It seems to be an accepted fact that quinine is less injurious to children than to adults, of course, taking into consideration that the doses for children are altogether smaller than those for adults.

Quinine has often been employed externally on account of its powerful antiseptic action, without showing injurious effects, when applied in the form of preparations with neutral or weakly alkaline reaction. Repeated frictions of ointment of quinine into the healthy skin cause abrasion and soreness.

TREATMENT.—The treatment of quinine-poisoning will vary with the various possibilities which

have just been described. Should a large dose of quinine be still in the stomach, sickness must be induced by mechanical irritation of the pharynx, or the stomach should be cleared by the pump. Nothing is more unwise than to try to empty the stomach by chemical emetics, such as tartar emetic or ipecacuanha. Firstly, much time is wasted by their application; and secondly—what is still more serious—if they really do act at last, they depress the nervous system and the heart, and diminish the power of resistance to the poison. If any emetic seems to be indicated, only a cautious hypodermic injection of hydrochloride of apomorphine can be permitted. Tannic acid or carbonate of sodium should be introduced into the stomach before or during evacuation, as quinine-salts are precipitated by these bodies in a much less soluble form.

In cases of *acute* quinine-poisoning recourse must be had to artificial respiration, with rhythmical pressure on the heart about thirty times a minute, as it produces strong mechanical irritation of this organ. Further, hot baths (40° C. or 104° F.), with cold affusions in a thin stream over the neck, should be tried. The room and bed in which the patient is lying must be kept as warm as possible. Strong hot coffee or tea is to be administered.

Stimulation of the kidneys is also required, to promote excretion of the alkaloid. Abundance of water containing free carbonic acid, like Seltzer or Apollinaris, mixed with small quantities of wine or other good alcoholic beverage, will answer this indication best.

As the head in such cases is of sufficiently low temperature, in consequence of the general depression of the body-heat, cold compresses to the head are not advisable. This part of the body must be kept low, in order to allow the weak action of the heart to maintain the cerebral circulation as easily as possible.

Most of the other symptoms caused by quinine disappear of themselves as soon as the drug is discontinued.

CARL BINZ.

QUINSY (*cynanche*, sore-throat).—A popular synonym for acute inflammation of the tonsils. See TONSILS, Diseases of.

QUINTAN (*quintus*, the fifth).—A form of ague, in which the paroxysm returns after an intermission of ninety-six hours. See MALARIAL DISEASE.

QUOTIDIAN (*quotidie*, daily).—A form of ague, in which the paroxysm occurs at the same hour every day. See MALARIAL DISEASE.

R

RABBI, in the Austrian Tyrol.—Chalybeate Waters. See MINERAL WATERS.

RABIES.—**DEFINITION.**—An acute disease, especially prevalent among the carnivora and dependent on the presence of a specific poison, chiefly in the nervous system.

ÆTIOLOGY.—The disease never arises spontaneously and is usually transmitted by bites. It is

possible that the virus present in the saliva of affected animals may occasionally infect others owing to the existence of abrasions, without actual biting. Isolated sporadic cases occur in all countries of Europe, and, from time to time, epidemics varying in severity. It is very usual for the malady to remain lurking in certain districts or countries. Thus foci of rabies are common in certain parts of Russia and of France, but rarer in central Europe.

In Great Britain more or less extensive epidemics of rabies have occurred from time to time. Since 1897, owing to the Muzzling Order, the disease has greatly diminished. During 1899 no human being died of hydrophobia in England or Wales, a result which had not been obtained during the last fifty-one years. In the year 1900 eleven cases were reported; this outbreak of rabies was confined to a district in the South-west of Wales. Rabies is, however, more frequent in Ireland. The disease is usually communicated by a rabid animal wandering at large, often over great distances, and biting other animals such as dogs, cats, horses, cattle.

The virus of rabies is especially abundant in the saliva and in the nervous system—both central and peripheral. There is some discrepancy among observers as to whether the virus is present before the symptoms of the malady are well established. According to Nocard and Roux the saliva of the dog may be virulent three days before the onset of symptoms; according to others, e.g. Dowdeswell, the central nervous system is not virulent until the animal shows symptoms of the malady. All parts of the nervous system are virulent at the time of death, but in the earlier stages of the malady both bulb and cerebro-spinal fluid are virulent. The blood, the lymph, the muscles, the liver, the spleen, and such secretions as the urine and the seminal fluid are usually stated not to contain the virus. On the other hand, such glands as the salivary, the lachrymal, and the pancreas contain the virus, and it has also been found occasionally in the milk.

All domestic mammalia are susceptible to rabies, although, from the habits of the animals, the disease is most likely to occur in dogs and cats. Birds are more refractory than mammals, especially to bites, but they can contract the disease through inoculation. Rabies is not rare in the wolf, the fox, and the deer.

Sub-dural inoculation is the most certain method of transmitting the disease experimentally, and next to this, inoculation into the aqueous humour; injection of the virus subcutaneously is a very uncertain method; it is more certain when injected deeply into muscles. Absorption from the gastro-intestinal tract has given different results, and in many cases the feeding of animals on the carcasses of animals dead of rabies has failed to transmit the disease. In other cases this method has succeeded, the explanation being doubtless that in some cases abrasions existed in the mucous membrane of the mouth and throat. The injection of the diluted virus prepared from the bulb into the veins is stated to confer immunity in the inoculated animal in the case of the horse, but in dogs and rabbits this is not the case; no immunity is produced and rabies may ensue. Drying destroys the activity of the virus in fourteen or fifteen days. If the spinal cord, however, be kept moist the virulence will persist for as long as forty days. Heating to a temperature of 48° C. destroys the virus in from five to ten minutes. Sunlight is said to destroy the virus in as short a time as fourteen hours. Putrefaction has an uncertain effect. In many cases the activity of the virus is not destroyed even after as long a period as forty days, but in other cases it may be destroyed earlier. Antiseptics such as perchloride of mercury in the strength of 1 in 1000 destroy the virus in a few minutes, and a 5-per-cent. solution of carbolic acid in approximately one hour.

INCUBATION.—The period of incubation is not only different in different animals but is liable to

somewhat considerable variations in the same animal. It is rather more variable after the transmission of the malady in the usual manner by a bite than when it is communicated by experimental inoculation. In most domestic animals the incubation-period varies from fifteen to sixty days. In dogs, according to Nocard, the disease in more than half the cases declares itself in less than one month after the bite, and in four-fifths of the cases the incubation-period is not more than sixty days.

After the passage of the virus through a considerable number of rabbits the incubation-period is shortened to six or seven days. This exaltation of the activity of the virus is not usually seen with less than 100 successive transmissions. Subsequently to this the activity of the virus becomes constant, the incubation-period being constantly six to seven days. This is spoken of as *le virus fixé* and is used in the Pasteur-method of prophylaxis.

SYMPTOMS.—Two forms of the malady exist, the *furiosus* and the *dumb*. In many animals, e.g. carnivora, one may precede the other, inasmuch as the later stages of the malady beginning as 'furiosus rabies' are characterised by symptoms similar to those seen in the 'dumb' form of the disease. In other animals, especially rabbits, the 'furiosus' form is rare, 'paralytic' or 'dumb' rabies being the usual type. 'Furious' rabies, however, may occur in rabbits as a result of inoculation or spontaneously, and 'dumb' rabies is not very uncommon in the carnivora. In both forms of rabies prodromal or premonitory symptoms are liable to be present. The prodromal period in dogs is characterised principally by an alteration in the habits of the animal. Quiet and friendly dogs become ill-tempered and restless, and are liable to conceal themselves in dark places. During this stage of the malady the animal may still be docile, and does not try to bite, and the appetite is maintained or may be increased. Irritation at the seat of the bite may be present, and very soon hallucinations develop, and the dog tries to avoid imaginary objects and may even snap in the air at them. This prodromal period passes insensibly into the second or fully established stage of the malady, in which the restlessness is greatly increased, and the dog suffers from paroxysms of violent excitement followed by remissions of exhaustion. In the paroxysms of excitement he destroys all objects that he encounters, and will, at this period of the malady, swallow a great variety of foreign bodies, such as sticks, straw, stones, rags, excrement, &c.

During this period the voice becomes altered so that the bark becomes extraordinarily high-pitched and has a peculiar falsetto quality. The peculiar nature of the bark is one of the characteristic features of the disease in its 'furiosus' form, and there is often a tendency for the animal to lie quietly for a time and then to suddenly jump up and utter the peculiar bark. The cause of the peculiar long-drawn bark is in part a paresis of the vocal cords.

When the dog is suffering from the fully established disease it is not uncommon for it to wander long distances, snapping and biting at objects, animals, and persons on its way. It is in this manner that a single rabid dog may communicate the disease to large numbers of other animals and also to man.

The paroxysms of furious excitement tend to become more and more marked for a time, but as

the disease progresses the periods of exhaustion between the paroxysms become longer; and, finally, during the last days of life, paralysis is fully established, and the animal is not only unable to swallow or to stand, but the jaw drops and the mouth gapes, with considerable quantities of stringy saliva hanging from it.

In 'dumb' or 'paralytic' rabies the onset of the malady is marked by somewhat similar sensory symptoms to those seen in the 'furious' form. After a short period, in which anxiety and agitation are the most marked features, the palsy sets in, in the form of paralysis of the muscles of the lower jaw, so that the mouth gapes owing to the dropping of the jaw. Although the dropping of the jaw is the most characteristic form of the palsy, this may assume other types, such as paraplegia, hemiplegia, or even monoplegia. The palsy that begins in the muscles of the jaw rapidly spreads to the muscles of the rest of the body, involving ultimately the muscles of respiration, and so causing death. 'Dumb' rabies is necessarily not so dangerous a malady as the 'furious' form, but although the animal is unable to bite, the disease may be communicated to others by careless handling of the dog; and in this way scratches about the hands may become infected from the saliva hanging round the mouth and teeth.

The average duration of 'furious' rabies in the dog is from four to five days, with a minimum of two and a maximum of ten. In 'dumb' rabies the average duration is from two to three days.

Glycosuria is not uncommonly present both in 'furious' and in 'dumb' rabies, and the urine may be examined for sugar after the death of an animal with suspicious symptoms. Glycosuria may be due to other causes, and its occurrence in rabies is more of interest than of importance.

Rabies runs a very similar course in both cat and dog. The 'furious' form is exceedingly well marked in the cat, and these animals are especially dangerous owing to their tendency to fly at and bite exposed parts of the body, such as the face.

In the horse the 'furious' form is also very well marked, and the animal becomes very savage, but the essential features of the malady are much the same as in the dog, and the average duration of the disease is from three to six days.

In the herbivora the average duration is from four to six days. During the height of the disease cattle will attempt to bite other animals or even man. Rabies in rabbits is usually of the 'dumb' or 'paralytic' variety.

MORBID ANATOMY.—The anatomical changes found in rabies are not well marked, and in many cases no changes at all are found owing to the animal having been killed at an early stage of the malady before the disease has existed a sufficient length of time to produce marked anatomical changes. The mucous membrane of the mouth, fauces, tongue, pharynx, stomach and intestine is usually congested and may present hæmorrhagic erosions or even ulcers. The tongue is often swollen, of a dark red colour, and the mouth generally injected, considerable quantities of ropy saliva hanging about the jaws. The stomach and intestines usually contain no food, but in the former is found a great variety of foreign bodies, such as hair, wood, straw, coals, hay, &c. The blood is often dark-coloured, the spleen congested, and the lungs gorged with blood with minute petechial

hæmorrhages on the surface of the serous membranes. The last, however, are only the results of death from asphyxia, and are of no great value as diagnostic signs of rabies. It is most essential to remember that rabies can never be excluded by *post-mortem* examination because the animal may have been killed, as is often the case, owing to a mere suspicion of the existence of the disease at such an early stage that none of the *post-mortem* phenomena are present.

Babes considers that the malady may be diagnosed with certainty owing to the presence of hyaline thickening and thrombi in the vessels of the central nervous system, especially the medulla. Van Gehuchten has described changes in the ganglia of the vagus, an acute degeneration of the nerve-cells of this ganglion, and he considers that by this means the disease can be diagnosed with certainty.

DIAGNOSIS.—In the 'furious' form of the disease the most characteristic clinical feature is unquestionably the bark. All dogs in which the malady is suspected should be isolated, because it is difficult, if not impossible, by a single examination in the early stages of the disease to determine with certainty the nature of the malady. Its clinical course is so rapid and characteristic that a few days' isolation will generally determine the question with certainty.

The following diseases are liable to be confounded with rabies: Epilepsy and diseases of the external auditory meatus, cysticercus in the brain, distemper, gastritis and enteritis, and in a few instances tetanus. Foreign bodies in the fauces and inflammation of the temporo-maxillary articulation, from rheumatic or other causes, are the diseases most apt to be confounded with 'dumb' rabies.

The *post-mortem* diagnosis is very unreliable for the reasons already mentioned. The most characteristic phenomena are the absence of food in the alimentary canal together with the presence of foreign bodies and hæmorrhages in the stomach. Some writers lay great stress on the presence of sugar in the urine.

EXPERIMENTAL DIAGNOSIS.—The only method of determining with certainty the presence of rabies after death is by the inoculation of a portion of the brain or spinal cord from the suspected animal subdurally in the rabbit. A portion of the central nervous system, preferably the bulb, of the suspected case of rabies should be placed in glycerine for three days; an emulsion of the brain should then be prepared with sterilised salt-solution and a few drops injected subdurally in the rabbit, subdural inoculation being more certain than that into the aqueous humour of the eye. After an incubation-period of from fourteen to nineteen days, but sometimes amounting to as much as six weeks, the paralytic form of the disease manifests itself in the rabbit, and owing to its typical features and characteristic course can be readily diagnosed. In a few instances the experimental method will fail owing to the contamination of the suspected brain with some septicæmic virus, the rabbit dying from the septicæmic infection at a period so early that the rabies cannot be manifested. The experimental method can be used even when the cord and brain are in a state of putrefaction; in a considerable number of cases injection of the decomposed or even putrid brain beneath the dura mater is not only not followed by any immediate serious consequences, but the virus of rabies if present may

still be able to manifest its presence in the usual way. A negative result after the sub-dural injection of decomposed brain-tissue suspected of containing the virus of rabies is of little value, as putrefaction unquestionably may destroy the virus. The great disadvantage of the experimental method of diagnosis is the fact that on an average some three weeks are necessary in order to determine with certainty the presence or absence of rabies.

TREATMENT.—Rabies is such a serious disease in animals, and the danger of the communication of hydrophobia to man is so great, that it is not customary to attempt to treat the malady. Animals undoubtedly suffering from the malady should be killed, and it is usually recommended to burn their carcasses. An animal bitten by another suffering from rabies may be treated on the same lines as the Pasteur treatment of hydrophobia, but the disease is such a terrible one that it is better to immediately kill all animals that have been bitten by an animal suspected of rabies. If an animal suspected of rabies has bitten any human being, it should be isolated and not destroyed until it has been determined whether the animal is rabid or not. When destroyed this opinion should be confirmed by experimental inoculation. If an animal has been in company with one affected with rabies it should be isolated and, according to some writers, the period of isolation should be at least four months. All kennels, dog-collars, &c., which have been used by a rabid animal should be disinfected with a powerful antiseptic such as perchloride of mercury.

Rabies can be checked, and in many cases entirely prevented, by the systematic and thorough muzzling of dogs, and by regulations directed to prevent the importation of animals, especially dogs, from countries where rabies is prevalent. It is to the thorough carrying out of the Muzzling Order that the extinction of the last epidemic in this country is due. In addition to this it would be very advisable that dog-owners should be compelled to have collars with their name and address on their dogs.

JOHN ROSE BRADFORD.

RACHITIS (ράχις, the spine).—A synonym for rickets. See RICKETS.

RAGATZ, in Switzerland.—Simple thermal waters. See MINERAL WATERS.

RAG-SORTER'S DISEASE.—See PUSTULE, MALIGNANT.

RAILWAY INJURIES.—**DESCRIPTION.**—The points of difference between railway injuries and those sustained in other ways, such, for instance, as by a fall from a horse or a carriage, are virtually those of degree. The more serious results are referable, firstly, to the great weight and impulse of the railway train, crushing, perhaps completely, some portion of the body; secondly, in the case of collision, to the sudden arrest of momentum of such ponderous bodies in more or less rapid motion, causing thereby violent vibratory shocks to the travellers; and, thirdly, the occurrence being sudden and unexpected, the muscles are, as it were, taken by surprise, and before contraction can take place the ligaments of the spine are frequently strained or even torn, while in other cases unequal and unbalanced muscular spasm leads to serious lesions. There is no time for preparation; the whole is the work of an instant. In cases of injury

to those who jump or fall from a train in motion, the gravity of the resulting injury depends on the rate of speed of the train at the moment; on the part of the body which first strikes the ground, and the angle at which it is struck; on the weight of the individual; and also on the nature of the ground.

Accidents which happen to persons either getting into or out of trains not in motion, possess no unusual characters. Serious spinal injuries have, however, occurred to those sitting in a train not in motion, when, by a sudden unexpected jerk, as from a train running into it from behind, a violent shock is sustained.

It is, then, the sudden and violent character of the occurrence, the alarm and fright necessarily associated therewith, the general jar or commotion of the nervous system, and possibly the localised physical damage, which constitute the main features of this class of injuries, conditions which do not obtain in cases of less sudden violence.

RESULTS.—1. The *direct* or *immediate* results of railway accidents are of various kinds. Locally, they consist of simple or compound fractures, contusions, and lacerations, caused either by the force of the collision, by the limbs being caught between the seats of the carriage, by the wheels of the carriages where the individual has been run over, or by fragments of splintered wood, iron, or glass. Burns and scalds may add to the sufferings of the injured. These injuries, if not resulting immediately in death, may ultimately prove fatal in various ways, or permanent injury to a greater or less extent may ensue. Such conditions, however, are not peculiar to railway injuries, and need no special description here.

The immediate effects on the cerebro-spinal system are, on the other hand, of special interest and importance. Local injuries, even fractures of the spinal column or head, are frequently met with, while in other instances serious lesions, accompanied by hæmorrhages, occur in the brain or spinal cord or their membranes, without any fracture; but *ligamentous* lesions of the spine are perhaps the most characteristic features of railway injuries. Death has resulted from sheer fright, especially in persons suffering from heart-disease, aneurysm, and the like. The primary depression produced on the nervous and circulatory systems continues and deepens, there is no power to rally, and a fatal result from syncope ensues. General shock or concussion is also commonly met with, where the symptoms are mainly subjective; and in emotional individuals an attack of acute hysteria may be induced, laying the foundation of a chronic condition.

For a description of concussion of the cord, and of the localisation of lesions of the cord, see SPINAL CORD, Diseases of.

2. The *indirect* or *remote* results of railway injuries are numerous and varied in character. *Locally*, it must not be forgotten that an accident may readily light up chronic inflammatory mischief in individuals who are predisposed by some inherited or acquired weakness, or by some particular diathesis. Thus syphilitic, tubercular, gouty, and even cancerous disease may follow and complicate the symptoms arising from the injury. Again, chronic inflammatory conditions of the spinal cord and its membranes may be induced thereby, and the usual train of symptoms accompanying such lesions will be manifested. But, apart from the existence

of such organic mischief, certain indefinite phenomena, subjective and functional in character, are constantly met with, which render the diagnosis and prognosis of railway injuries difficult, and considerably increase the responsibilities of the medical attendants. It is especially, however, when there is no local lesion of importance, such as a fractured limb, that these general 'neurasthenic' conditions are likely to occur; for it can be readily understood that if the force of the accident expend itself upon some injury, say, to one of the extremities, there is less likelihood of a general concussion of the nervous system. The familiar and often-quoted illustration is that of a watch, in which the works are less likely to be damaged by a fall if part of the violence be expended in breaking the glass. This neurasthenic condition is evidently the expression of an exhausted nervous system, and may come on in various ways and at various times after the accident. The mental shock is to be held responsible for its occurrence nearly as much as is the physical. It may arise out of the general collapse or shock into which the patient has been suddenly thrown by the accident, but perhaps more commonly the extent of the mischief is not evident at the time. As a typical illustration of such a case, the following may be taken: A person in a collision receives a sudden, unexpected, and violent shock, the result of being thrown or jolted backwards and forwards. He feels faint and collapsed for a few moments, but recovers sufficiently to be able to assist his fellow sufferers; returns home, and resumes his usual avocations. After an interval of a few hours or days he begins to complain of pain and stiffness in the back and neck, but particularly in the lumbo-dorsal region. He goes to his business, but cannot attend to it, being unable to concentrate his attention or make calculations, any special effort resulting in marked headache. He is unable to read comfortably, as the letters run into one another and become blurred. He goes home and consults his medical man, who probably advises rest in bed, and prescribes bromide of potassium, &c. The temperature in these cases is often sub-normal, and the pulse sometimes rapid, sometimes slow. These symptoms in the majority of cases pass away after a short rest, and terminate in complete recovery. In other cases, fortunately the exceptions, further symptoms develop, such as irritability of the bladder, inability to empty the viscus, occasional dribbling of urine, lightning pains in the spine, spasm of the legs, and others too numerous to mention here. These may depend either upon the condition of neurasthenia, just alluded to; or more rarely upon definite congestive or other pathological changes occurring in the spinal cord. The prognosis of the latter is grave in the extreme.

In the condition of neurasthenia, asthenopia, with difficulty in or real loss of the power of accommodation, may be one of the causes of the impaired vision just mentioned. Physical changes in the retina and optic disc are extremely uncommon. In all probability, they only exist as sequelae of organic lesions of the spinal cord and membranes.

Added to this neurasthenic condition, there is often seen a certain amount of functional disturbance, probably of cerebral (cortical) origin, which may be described as chronic hysteria, under which one includes changes of various kinds in the cutaneous sensibility, spasmodic contractions of limbs, and paralyses; and it is often a most difficult ques-

tion to decide whether these phenomena are functional or organic. Cases such as these are frequently the subject of litigation as regards claims for damages against railway companies.

MEDICO-LEGAL QUESTIONS IN CONNECTION WITH RAILWAY ACCIDENTS.—In cases of claim for compensation for these injuries, it is of the highest importance that the medical men engaged should make themselves thoroughly acquainted with all the circumstances connected with the accident and its results. This applies to the *medical attendant* of the injured person, as well as to the *medical officer* examining on behalf of the company.

Duties of the Medical Attendant.—1. It is desirable to obtain in writing the *patient's statement* as regards: (a) the accident; ascertaining, if possible, the approximate speed of the train when the accident occurred, the position of the injured person in the carriage, and of the carriage in the train; also whether other persons were present or not; (b) his condition from the time of the accident to the time of his examination; and (c) the symptoms complained of at the time of his examination.

2. Investigate the general condition of the patient, especially as to his appetite, and his capacity for sleep or work; ascertain also his previous habits. The possible existence of organic disease, previous to the accident, should not be overlooked, as it has happened that symptoms referable to disease—tabes dorsalis, for example—have been erroneously ascribed to injury. The urine should be carefully examined in every case.

3. Note bruises or any sign of local injury on any part of the body.

4. Where injury to the spine is alleged, the investigation should be conducted as far as possible according to the following systematic plan:—

(i.) Notice the amount of mobility or rigidity of the spine while the patient is undressing.

(ii.) Examine the spine by digital pressure or percussion, and by the application of hot or cold sponges.

(iii.) Investigate any alleged paralytic symptoms by (a) measuring the circumference of the limbs, where needful; the right side, it should be remembered, is usually somewhat fuller than the left, and the existence of any other pre-existing inequality of the limbs should not be lost sight of; (b) testing the electrical excitability of the muscles; (c) noting the existence of spasm or tremor of the muscles of the spine and limbs; and (d) examining the condition of the reflexes, superficial and deep.

5. Investigate the presence of any abnormal cutaneous sensibility. This is entirely a subjective phenomenon, on which reliance cannot always be placed.

6. Ophthalmoscopic examination may be necessary in order to determine the existence or not of local lesion in the fundus oculi, confirmatory or otherwise of cerebral or spinal symptoms; but, as already stated, such pathological changes are exceedingly rare.

During this examination, it should be borne in mind that the simulation of symptoms, such as spinal tenderness or muscular tremor, can frequently be detected by distracting the attention, when pressure on the part previously complained of may be made with impunity, or the muscular tremors will cease. This, however, is not conclusive of inten-

ional imposture, for in hysteria, when the attention is diverted, the same occurs.

As an instance where the truth of a patient's statements may be tested by the astuteness of the medical man, a case may be mentioned in which the plaintiff, who had travelled up some fifty miles to London to be examined, stated, among other symptoms, that his urine continually dribbled from him. The surgeon immediately asked to see his shirt, which had been worn at least six hours, when it was found perfectly dry and devoid of any stain of urine! In another case a man complained of extreme spinal tenderness, even when the part was merely blown upon. A sheet of paper being interposed, without the patient's knowledge, the effect was the same!

It now becomes the duty of the medical attendant to form an opinion on the following points:—

- (a) Has the patient been really injured?
- (b) What is the nature of the injury?
- (c) Is the injury a possible or probable result of the accident as described?
- (d) Are the symptoms consistent with the history and the objective signs?

Duties of the Medical Officer examining on behalf of the Railway Company.—The medical officer of the company should on no account constitute himself the agent of the company for settling the terms of compensation. The examination should be made, if possible, in the presence of the medical attendant of the patient, and not in the presence of the legal advisers of either side; it should be conducted with thoroughness and tact, and without inflicting any unnecessary pain.

A report of the case should be drawn up from notes taken at the time, giving:—

- (1) The patient's account of the accident, and of his subsequent and present symptoms.
- (2) The present condition of the patient, noting particularly any *objective* signs of injury.
- (3) An opinion as to whether the symptoms complained of are likely to be the result of the accident; and as to the probability of recovery, and at what period.

As the plaintiff's solicitors in an action can apply for a copy of this report, it should, of course, be worded with extreme care.

The actual question of pecuniary compensation does not concern either the medical attendant of the patient, or the medical adviser of the company. They merely have respectively to bring forward facts in support of their opinions as to the value of symptoms, and how far they are dependent upon the injury.

Such investigation should take place as soon after the accident as possible, by which means the chances of imposture would be lessened; while by a careful and impartial estimate of the facts of the case as obtained by such a thorough examination as that sketched above, much conflict of medical opinion would be avoided, the medical men being witnesses, not advocates.

At the above examination the question of the future treatment of the plaintiff may be raised by his doctor, but the medical officer of the company should be extremely cautious in giving advice or making suggestions.

Fraudulent Claims.—It will be well to allude to some of the ways in which fraudulent claims are brought against railway companies. These may be conveniently divided into:—

(1) Claims made by persons who, as may be subsequently proved, were not even in the train.

(2) Claims by those who, though present and unhurt, yet simulate the symptoms of injury; and

(3) Claims by those who, having sustained some trifling injury, wilfully and intentionally exaggerate their symptoms in order to obtain a larger amount in compensation.

The medical man should, therefore, be alive to the possibility of wilful deception being practised on him, lest he should be led away by a well-planned history, and thus unwittingly be made a party to such fraudulent transactions.

Unintentional Exaggeration.—The difficulty of assessing the value of subjective symptoms in general is much increased by the fact that there are certain persons who, undoubtedly injured, may, without any fraudulent design, unintentionally exaggerate their symptoms. This is to some extent explained by their thoughts being constantly directed to their sufferings, and the worry necessarily attending a protracted lawsuit, or while an action for damages is pending. The suspense and anxiety, the examinations by the medical men, and the repeated interviews with their solicitors, keep them in a constant state of nervous tension. When, therefore, their claims are settled, it is natural that the relief they experience should frequently be attended by beneficial results, or even complete recovery. See **FEIGNED DISEASES**.

TREATMENT.—The chief injuries received at the time of a railway accident being surgical, the treatment adapted for each particular case will be found in surgical works. Nevertheless there are some general points in the immediate treatment to which any medical man present on such occasions would do well to attend.

In the present day, thanks to the efficient training given on most railways to the guards and other employés in ambulance work, the administration of first aid is more intelligently carried out, and has frequently been the means of saving life.

1. *Hæmorrhage.*—Death from hæmorrhage should be prevented, where possible, by promptly adopting pressure of some kind. If no tourniquet or india-rubber band be available, a handkerchief tied round the limb and twisted tight with a piece of stick, or direct pressure by the finger, will suffice for the time.

2. *Fractures.*—Temporary splints may be improvised out of umbrellas, walking-sticks, cushions, newspapers, and broken pieces of wood, &c., fixed by straps or handkerchiefs, so that the injured may be removed with as little pain as possible, and simple fractures may be prevented from becoming compound. Simple dislocations should be reduced at once, if possible.

3. *Shock, collapse, and fright.*—In the treatment of these conditions great caution is required to maintain the vital powers until reaction sets in. The temperature of the body, the strength and rate of the heart's action, together with the respiration, should be kept up by stimulants and warmth. There is, however, a danger of *over-stimulation*, whereby the flickering powers of nature may be extinguished altogether.

4. *Exposure to wet and cold.*—Every endeavour should of course be made to prevent prolonged exposure, by sheltering the injured as much as possible, and securing their early removal to any neighbouring houses. This question of moving the

injured passengers, many of whom may be suffering from profound shock, is often a difficult one to decide. On the one hand the local means of treatment may be very limited, while, on the other, a long journey may be before the patient, and the means of transport are often inadequate in consequence of the construction of the rolling stock, whereby it is extremely difficult to transfer a recumbent passenger through the narrow doorways. It has been suggested either that every train should have a coach attached to it capable of being used as an ambulance van, or that at the principal stations fully equipped hospital vans should be kept in readiness to be attached to the break-down relief train.

The subsequent treatment of railway injuries is one which requires the exercise of considerable skill and judgment on the part of the medical attendant. Absolute rest is of course a *sine quâ non* where any spinal concussion or local lesion is suspected; but where the symptoms are due to neurasthenia, long confinement in the horizontal position is often productive of harm, whereas change of scene and moderate exercise might prove beneficial.

WILLIAM ROSE.

RÂLES (Fr.) Rattles. — Certain adventitious sounds heard on auscultation, in connection with the respiratory organs, during the act of breathing, in various morbid conditions. See PHYSICAL EXAMINATION; and RHONCHUS.

RANULA (*ranula*, dim. of *rana*, a frog). — A cystic growth in the floor of the mouth. See MOUTH, Diseases of.

RAPE.—SYNON.: Fr. *Viol*; Ger. *Nothzucht*.

DEFINITION.—By the English law rape is defined as ‘the carnal knowledge of a woman forcibly and against her will.’

GENERAL REMARKS.—The crime of rape is punishable by penal servitude for life. By the Criminal Law Amendment Act of 1885 the defilement of a girl under thirteen is a felony punishable by penal servitude for life, and the attempt to have carnal knowledge of a girl under thirteen is a misdemeanour punishable by two years’ hard labour, or by a whipping if the offender be under sixteen. The carnal knowledge of a girl between thirteen and sixteen, or the carnal knowledge of an idiot or imbecile girl, is a misdemeanour punishable by two years’ hard labour.

Of cases of rape recorded by Casper, 73 per cent. were upon the persons of little children under twelve. Of 136 cases put upon record by this author, the ages were as follows:—

From	2½	to 12 years of age,	99 cases
„	12	„ 14	„ 20
„	15	„ 18	„ 8
„	19	„ 25	„ 7
		47	„ 1 case
		68	„ 1

For proof of the crime of rape it is not necessary that the force employed should have been of a violent physical kind. A mere *threat* of violence, or even of moral injury, is ‘force’ in the eyes of the law. The surreptitious administration of chloroform, or a narcotic, for the purpose of having intercourse with a woman against her will, is also force in the eyes of the law. The Criminal Law Amendment Act quoted above has the following clause: ‘Whereas doubts have been entertained whether a

man who induces a married woman to permit him to have connection with her by personating her husband is or is not guilty of rape, it is hereby enacted and declared that every such offender shall be deemed to be guilty of rape.’

The moral character of the woman is theoretically, but seldom practically, beside the question; and, provided force be used, and the woman’s consent be wanting, sexual intercourse even with a prostitute is legally ‘rape.’

The punishment of the crime of rape was provided for in the criminal code of Moses, who ordained that the ravisher of a betrothed damsel should die.

The Roman law punished the crime with death and confiscation of goods, but provided the following saving clause:—

Rapta raptoris, aut mortem, aut indotatas nuptias optet.

Upon this, says Percival, there arose what was thought a doubtful case: ‘*Una nocte quidam duas rapuit; altera mortem optat, altera nuptias.*’

Many accusations of rape are false and trumped up, and are only brought by the woman when she finds that some sexual indiscretion is likely to bring her into trouble, or cannot be concealed by reason of her pregnancy.

This being the case, *stale accusations* should be received with very great caution. The laws of Henry III. provided that the accusation should be made immediately ‘*dum recens fuerit maleficium.*’ By the old Scotch law no delay was allowed in bringing the accusation *ultra unam noctem*, and by the modern Scotch law a delay of three days is alone permitted. By the law of England no limit is placed on the time at which an accusation of rape may be made. An English jury is, however, naturally chary of giving credence to a stale charge of rape. Some few years back a charge of rape was brought against a gentleman of position in one of the home counties by a girl with whom he had had connection some five months previously. There was no evidence that the girl had offered any resistance, and as the accusation was brought only after pregnancy had become evident, and after ineffectual attempts had been made to extort money from the defendant’s relatives, and as the charge was evidently made at the instigation of an uncle who was a superintendent of police, and a cousin who was a lawyer, the case was dismissed. It shows, we think, an imperfection in the English law that it should be possible, under such circumstances, to prefer a charge of so serious a crime.

The law for the substantiation of a charge of rape is satisfied with proof of a minimum amount of ‘carnal knowledge.’ The mere touching of the vulva by the penis is carnal knowledge in the eyes of the law. The complete introduction of the penis into the vagina need not be proved, and still more is proof of emission unnecessary.

THE SIGNS OF RAPE.—From what has gone before, it is evident that there need be no signs whatever. If a girl be overawed by a threat and her vulva be touched by the penis, that is rape; and, if proved, is punishable as such.

On the other hand, the evidence of rape may be very convincing; for example:—

(a) The woman may have been heard to cry for help.

(b) There may be the signs of a struggle at the spot where the rape was alleged to have occurred.

(c) There may be damage to the woman's clothing, and bruises of various parts of her body—signs that she has been subjected to physical force.

(d) The genital organs may be found injured; the vulva bruised and perhaps bleeding; the hymen recently ruptured; and, in cases where the disparity in size between the man and woman is very great, rupture of the perineum and mortal injuries to the vagina.

(e) Seminal spots may be found upon the woman's clothing, which is a certain proof of a previous intimate relation with a male. Blood-spots also afford valuable evidence, but necessarily not so conclusive. Care must be taken not to confound menstrual fluid with blood.

The concurrence of all these signs would amount to certain evidence of forcible connection. It must be borne in mind, however, that violence may be done to the female organs in other ways than by forcible connection, and the medical examiner should be upon his guard against inferring too much from the evidence afforded. He also should be on the look-out for facts which may rebut assertions made by the woman. Thus, signs of a previous pregnancy or the evidence of previous venereal disease (scars in the groin, sores upon the pudenda, or symptoms of constitutional syphilis) may serve to disprove any assertions which might be made as to the woman's virginity or previous chastity. To *prove* whether or no a woman be *virgo intacta* is next to impossible, and we can only state the probabilities for and against. Such a question, however, is quite beside the mark in many cases of rape; but the presence of an unruptured hymen is an unlikely occurrence after forcible connection. An examination of the person of the supposed ravisher may afford some corroborative evidence. Blood or recent seminal spots upon the linen or clothing, and injury to the person or clothing, all afford their quota of evidence of a sexual act combined with violence.

It is a matter of doubt whether the rape of a woman of fair size and strength be possible by an unaided man. If a woman be in the enjoyment of her faculties she is capable of offering an amount of resistance which would be well-nigh insuperable; and if she have offered a decent resistance, the person of the ravisher should bear evidence of it.

Rape, as we have seen, is most often committed on children of tender years. It is well to be on one's guard against error with regard to the rape of little children. It must have come within the experience of most members of the profession, and especially of those engaged in hospital practice, to have brought to them children suffering from a purulent discharge from the vagina, the mother at the same time alleging that some one must have violated the child. It must be borne in mind that purulent discharges from the vagina are not uncommon in ill-fed, dirty, scrofulous children; and that after some of the infantile acute specific, sloughing of the pudenda is a rare, though recognised occurrence. The case of Jane Hampson, *et. 4*, who died of sloughing of the genitals at Manchester in 1791, should stand as an incentive to caution in these matters. The signs were considered as those of defloration, and the coroner's jury returned a verdict of *murder* against the boy who slept with her; but luckily for the male child there occurred many other cases of sloughing of the pudenda in Manchester before he was brought to

trial, and as the doctor who was called to Hampson recognised and acknowledged his error, the boy was discharged. It was at one time a popular belief that connection with a virgin was a sure cure for venereal disease, and this has led, no doubt, to many cases of rape on young children. The presence of venereal disease in one or both of the parties may be of value as evidence. Its presence in the woman and not in the man affords a strong presumption against rape.

The finding of spermatozoa within the vagina is proof positive of connection. But here, again, care must be taken not to mistake for spermatozoa the *Trichomonas vaginalis*—a microscopic organism, not unlike a tadpole in shape, which has been described by Donné as occasionally found in vaginal mucus. It must be remembered, also, that seminal fluid may contain no spermatozoa. Rape is occasionally effected with so much violence that death results. Ogston records the case of one Margaret Paterson, who was raped between Edinburgh and Dalkeith by two carters, who took her into their cart on the pretence of helping her on her journey. They forcibly held her down and repeatedly violated her person, and afterwards took stones from the road, coals, straw, prickly plants, &c., and forced them into the vagina. They then left her in a ditch, and she died in three days of her injuries. *Post mortem* the vagina and rectum were found lacerated and broken down into one passage, and the abdominal viscera in a high state of inflammation. The two carters were convicted and executed. This case does not stand alone, for it is an interesting fact that rape has not infrequently been accompanied by acts of violence which are not only brutal and senseless, but which appear to have no relation to sexual acts or feelings.

It has been doubted whether pregnancy can follow rape, but there seems to be no sufficient grounds for this doubt.

DUTIES OF THE MEDICAL EXAMINER.—When called to a case of supposed rape, the medical examiner must remember to take note of every circumstance—the time that has elapsed since the alleged outrage, the mental state of the woman, her size and physical power as compared with that of the man, and evidences of a struggle in the surroundings of the woman, or on her clothing and person. He should keep his mind open to receive any facts which may throw light on the moral character of the woman. He should accurately take note of the exact condition of the genital organs and linen; should take possession of all stained linen for the purpose of chemical and microscopic examination; and should remove a portion of any discharge which may be found in the vagina for the same purpose. In drawing up a report, he should describe, as accurately and drily as possible, all facts which he may notice; and should be carefully upon his guard against drawing any undue conclusions from those facts.

G. V. POORE.

RASH.—An outbreak of redness of the skin, or efflorescence; called by the Greeks an *exanthema*, or blossoming out. The word conveys the idea of suddenness, while in reference to development it is generally extensive. The best illustrations of the rashes and of the meaning of the term are seen in erythema, roseola, rubeola, scarlatina, and urticaria.

RAYNAUD'S DISEASE. — **SYNON.** : Local Asphyxia and Symmetrical Gangrene of the Extremities ; Fr. *Asphyxie locale* ; *Gangrène Symétrique* ; Ger. *Symmetrische Gangrän*.

DEFINITION.—A disease characterised by disorders of the circulation in the peripheral parts, frequently symmetrical in distribution and depending on paroxysmal diminution or arrest of the flow of blood in the affected parts.

VARIETIES AND DESCRIPTION.—Under the name of Raynaud's disease there are included a number of vaso-motor phenomena which may conveniently be grouped into three main classes ; each of these may constitute the whole disease ; or two or more varieties may co-exist or replace one another. These three varieties are known as (1) Local syncope or regional ischæmia ; (2) Local asphyxia or local cyanosis or acro-asphyxia ; and (3) Local gangrene, symmetrical gangrene or acro-sphacelus.

(1) **Local Syncope.**—This variety chiefly affects the fingers, toes, hands, feet, and more rarely the ears, face or limbs.

An attack of local syncope usually commences with a sensation of numbness, tingling, or slight pain in the affected part. This latter becomes pale and cold, the skin having a waxy look, or being actually shrivelled. The part may become so bloodless that, when pricked or cut into, it does not bleed. The finer movements of the fingers are interfered with—writing, sewing and similar acts becoming difficult or impossible. Tactile sensation is blunted, but impressions of heat and cold are correctly interpreted. Commencing in one finger the condition may spread to other fingers on the same hand, or to the corresponding ones of the opposite hand, or it may remain localised to one finger all the time. The fingers are more frequently affected than the thumbs or the toes.

The attack lasts a time varying from a few minutes to several hours or even days, the part either gradually and insensibly returning to its previous condition, or this restoration being accompanied by a sensation of tingling or of burning pain, and occasionally by sweating of the part. After repeated or prolonged attacks the fingers become tapered at their tips.

An attack of local syncope may occur without any recognised cause, or may follow exposure to cold, such as from washing the hands in cold water ; exercise and mental excitement or emotion may also induce an attack. Attacks may recur from time to time from slight thermal or emotional changes, or may recur regularly in the absence of these in a manner strongly suggestive of some malarial influence.

The frequency of the attacks varies considerably, in some cases intervals of only an hour, in others of a week, being present.

During an attack very little change can be made out in the artery supplying the part, but changes characterised by the breaking up of the red blood-corpuscles have been noted in the blood of the ischæmic part.

The condition of local syncope may be followed by local asphyxia, or even by gangrene of small portions of the skin of the finger-tips.

2. **Local Asphyxia (Local Cyanosis).**—This variety is the one most commonly met with. It may succeed an attack of local syncope, or occur with only a passing pallor ushering it in. It is charac-

terised by the cyanotic appearance of the parts affected. These are most commonly the extremities—fingers, toes, hands, feet, ears, nose, cheeks, and occasionally portions of the trunk. The extent involved in local cyanosis is usually greater than in local syncope, but the distribution is less symmetrical. An attack usually commences with a transient pallor, followed by cyanosis of the part. The degree of cyanosis is inconstant, and gives rise to tints varying from a reddish-purple to a blue-black colour. The colour of the affected part is not uniform, and the same area may present varying shades of colour. Pressure over the cyanotic part is followed by a pallor which is only slowly replaced by the colour of the surrounding parts. The cyanotic portion is cold, swollen, and tender. In one of Raynaud's cases the surface-temperature of the affected hand was 27° F. below the temperature in the axilla. The swelling of the part is usually moderate, and there may be some pitting on pressure, but occasionally it is very marked. Subjective sensations of cold, tingling, pain or even agony are experienced, and pressure is ill-borne. As a rule, cold applications relieve, and hot ones intensify, the pain. Areas of anæsthesia are not uncommon. During the attack the artery supplying the part may be felt to be smaller and harder than the corresponding one on the opposite side, and Barlow has observed moniliform swellings on the veins of the affected part, which altered in position and which he ascribes to varying contractions of the walls of the veins. Sweating of the part is frequently observed during an attack, and the patient may shiver, vomit, and suffer from abdominal pain. The duration of an attack varies from minutes to months. The attack usually passes off slowly, livid spots making their appearance in the cyanotic area, and sometimes reddish-purple areas (*tachetées*) occur in the neighbourhood of the cyanotic region. The return to the normal may be accompanied by tingling sensations. Local cyanosis not infrequently passes on to local gangrene. The onset of an attack may be determined by the same factors which induce local syncope, and attacks are very prone to recur. The blood drawn from the site of local cyanosis is dark, and shows oligocythæmia and a breaking down of red corpuscles and some leucocytosis. Attacks of local cyanosis are sometimes followed, or accompanied, by hæmoglobinuria, sclerodermia, urticaria, fibrous ankylosis of the terminal phalanges, amblyopia, or iridoplegia.

3. **Local or Symmetrical Gangrene.**—This variety never occurs as a primary condition, but may follow either on repeated and prolonged attacks of local syncope, or, much more commonly, of local asphyxia. The parts most commonly affected are the toes, fingers, ears, cheeks, nose, and nates. The distribution of the gangrene is sometimes, but by no means frequently, symmetrical. One or more digits may be attacked. The onset of gangrene is usually signalled by the appearance of small blebs, situated usually on the plantar surface of the toes, or about the nail in the case of the fingers. These blebs give exit to some blood-stained, sterile serum, and leave a little black patch of epidermis, which is gradually shed by a process of ulceration. In more severe cases the true skin and the subcutaneous tissue are involved, and the bones may be laid bare. Occasionally the necrosis extends up the limbs, but as a rule it is confined to the extremities. Although the gangrenous part is neces-

arily insensible, yet the neighbouring parts are often exquisitely painful to the touch during the process of separation. The gangrene is always of the dry variety, i.e. is a process of mummification. Healing takes place slowly, but presents no peculiarities except that the gangrenous areas leave much less loss of tissue than would have been expected. In none of the three varieties of Raynaud's disease is there any marked pyrexia.

ÆTIOLOGY.—*Age.*—Raynaud's disease may occur at any age, but is most common between 20 and 40 years of age. Several cases are recorded in children under one year of age, and cases occurring in patients over 70 years of age are also known.

Sex.—Females are more liable than males. In 180 cases collected by Munro 62·6 per cent. were females, and 37·4 per cent. males; and of the cases admitted into the Middlesex Hospital during ten years, 74 per cent. were females, and 26 per cent. males.

Direct heredity was present in 8 per cent. of Munro's cases.

Relation to other Diseases.—Raynaud's disease has been met with in association with some of the acute specific fevers, such as typhoid fever, also with malaria, syphilis, neurasthenia, insanity, menstrual disorders, scleroderma, carcinoma of the stomach, and paroxysmal hæmoglobinuria. Although in several of Raynaud's cases malaria was present, it is to Mourson and Calmette that we are indebted for the observation of the relationship between the two conditions. Raynaud's disease seems common in some malarious districts, but in a large number of cases there is no history or evidence of malaria, and 8 per cent. is probably a high estimate of the number of cases in which malaria plays any part. Syphilis, either congenital or acquired, plays in relation to Raynaud's disease an interesting part, both from the fact that many cases of paroxysmal hæmoglobinuria occur in the subjects of syphilis, and also from the narrowing of the arteries, which may result from syphilitic arteritis. Of all the diseases with which Raynaud's disease is associated paroxysmal hæmoglobinuria is the most interesting, and the connection is so close that Abercrombie regards this condition as a symptom of Raynaud's disease. Most writers have noted the frequent concurrence of the two conditions, and, in several cases of paroxysmal hæmoglobinuria in children, have observed local syncope and local asphyxia of the hands and feet. The attacks of hæmoglobinuria do not always coincide with the local asphyxia or cyanosis. Paroxysmal hæmoglobinuria occurred in about 6 per cent. of the 180 cases collected by Munro. Scleroderma occurred in 7·2 per cent. of Munro's cases, but it has not been observed in association with hæmoglobinuria.

MORBID ANATOMY AND PATHOLOGY.—No constant change has been found either in the vascular or nervous system. The arteries have in several cases been found healthy; in other cases thickening of all three coats has been observed, and Barlow suggests that this may be the result of repeated spasmodic contractions of the vessel walls. Endarteritis has been observed in both syphilitic and non-syphilitic cases. In both the central and peripheral parts of the nervous system changes are inconstant. In a few cases, peripheral neuritis has been observed in the nerves of the affected part, but it is often difficult to distinguish the neuritis due to the gangrenous processes in a part from those which

may be primary and cause the necrosis. The blood in the affected part has been shown to undergo changes consisting in the breaking up of the red corpuscles and a slight leucocytosis.

The paroxysmal nature of the attacks—the return to normal conditions in the intervals, the absence of any degenerative changes in the vessels in most of the cases in young subjects, and the direct observation of the altered pulse in the part and of the changes in the veins, as well as the evidence of vascular spasm obtained by ophthalmoscopic examination made during an attack of local asphyxia, all point to the morbid condition being one of active spasm of the vessel-walls with diminution or arrest of the flow of blood through the affected vessels. The influence of cold or emotion in inducing an attack points to this condition of vascular spasm as being a reflex act, the vaso-motor centres being in an unduly irritable condition so that vascular spasm is readily induced and maintained. The symmetrical character of many of the attacks lends additional probability to the central nature of the change. Changes once initiated tend to recur with undue readiness, and thus we get from slight causes recurrent attacks of vaso-motor spasm.

The view that the disease is a primary peripheral neuritis is negated by the absence of evidence of such a condition in some of the cases examined.

The actual condition of local syncope is due to contraction of the arterioles and venules; while in local cyanosis there is also contraction of the veins. In local gangrene we have probably complete arrest of the circulation in the part and not the incidence of any special necrosing agents such as those derived from certain micro-organisms, for the serum from the blebs in the gangrenous patches has been shown to be sterile. By some local syncope and asphyxia are regarded as angio-neuroses, while symmetrical gangrene is regarded as an angio-trophoneurosis to which latter category scleroderma is also relegated.

DIAGNOSIS.—The diagnosis of a typical case of Raynaud's disease presents no difficulties, but cases in which the lesions are not symmetrical and in which the paroxysmal character of the attacks is not well marked often present great difficulty. Slight cases of frostbite or of 'dead fingers' differ only in degree from true cases of local syncope. Local cyanosis may be simulated by the cyanosis of *morbus ceruleus*, but here the blueness is seen in the lips and other parts, the nails are blue, the finger-ends are clubbed and painless, and there are the usual auscultatory evidences on examination of the heart. In erythromelalgia the altered colour of the part is induced by the dependent position; the temperature of the part is raised, the affection is usually unilateral and is more common in men than in women. Symmetrical gangrene may be mistaken for gangrene depending on arteritis, cardiac weakness, diabetes or embolism; but in all these cases the lesions are rarely symmetrical, and if so are accompanied by gross changes in the heart or arteries, or occur at a period of life in which Raynaud's disease is rare, or there is evidence in the urine of the presence of sugar. Ergotism may simulate Raynaud's disease, but may be excluded by the investigation of the food and by the epidemic nature of the outbreaks of ergotism. In all doubtful cases the paroxysmal nature of the attacks, the symmetry of the affected parts, the age of the patient, and the absence of evidences of other

vascular disease will enable us to recognise cases of Raynaud's disease. The existence of evidence of loss of substance of the ears, fingers, or toes may render us valuable assistance in satisfying ourselves as to the nature of an attack of local syncope, cyanosis, or gangrene.

PROGNOSIS.—The disease is rarely fatal. Many cases recover entirely; others remain liable to recurrences for years. Death, if it occurs, is usually the result of some complication, such as Bright's disease, pulmonary tuberculosis, carcinoma, exhaustion, diarrhoea, and occasionally of scleroderma or of the extension of the gangrenous process.

It must be remembered that Raynaud's disease, though it may not shorten life, may render the unfortunate sufferer incapable of performing the ordinary duties of life for a considerable period, and may make residence in cold climates during the winter most undesirable.

TREATMENT.—This may be considered under two heads: the treatment during the attacks, and the treatment during the intervals between them.

During an attack treatment by electricity has proved successful. Barlow advises that the part should be immersed in a basin containing warm salt-solution, and that the negative pole of a galvanic circuit should be placed in the basin, while the positive pole is placed on a healthy part of the patient, such as the nape of the neck, or on the limb above the affected part. The current should be as strong as can be comfortably borne, and should cause some contraction of the muscles of the limb. It should be frequently interrupted and reversed, and the patient encouraged to move the affected parts. The galvanisation should be continued till the part is red, and then the part should be gently rubbed and dried. Barlow recommends this treatment both during the attacks and in the intervals between them. Faradism has also proved useful, as has galvanisation of the sympathetic in the neck. Friction and massage of the affected parts, if it can be borne, may cut short an attack, and render fresh attacks less likely to occur. Warmth during an attack often increases the pain. These cases may not infrequently be relieved by cold applications. Laudanum or chloroform sprinkled on moist compresses, or glycerine of belladonna painted on the part, and even oxygen-baths, have all given relief in some cases, but nothing has been so successful as the electrical treatment. Hypodermic injections of morphine, with or without atropine, into a healthy part have been useful in relieving pain. The part must be protected from cold and injury. In slight or early attacks brisk exercise may ward off an attack. Vasodilators, such as amyl nitrite, nitroglycerine and the other nitrites, have proved of little use during the attacks.

In the intervals between the attacks patients should be careful to protect the hands from cold by means of loose woollen gloves and mittens, and the feet by warm socks. Patients should avoid exposure to cold, and should use warm water for washing. Exercise, a generous diet and a moderate use of alcohol are all advisable. In malarious districts quinine in doses of 10–20 grains (in solution) has been successfully employed, as has opium administered regularly in small doses. In cases where syphilis is suspected of being the determining factor, mercury and iodide of potassium have been of use, though in the experience of the writer neither drug

has had any beneficial effect on the paroxysmal hæmoglobinuria or the Raynaud's phenomena. In those who suffer in the cold weather residence during winter months in warm climates may be necessary to prevent recurrences. When gangrene occurs the parts should be simply protected by some aseptic dry material, and any irritant dressing should be carefully avoided. Amputation—rarely necessary—should be performed only when the part is actually dead and the surrounding parts are healthy.

ARTHUR FRANCIS VOELCKER.

RECEPTACULUM CHYLI, Diseases of.—

The only morbid conditions which need be specially noticed in connection with the receptaculum chyli are *dilatation* and *rupture*. It has been found in rare instances enormously dilated, and its walls thickened (*see* FILARIASIS). It has also been known to burst as a result of this dilatation, with the escape of its contents into the peritoneal cavity, fatal peritonitis being thus set up.

FREDERICK T. ROBERTS.

RECEPTION ORDER for Lunatics.—*See* LUNACY, Law of.

RECKLINGHAUSEN'S DISEASE.—A name occasionally employed to denote multiple neurofibromata of the skin.

RECOARO, in the Province of Vicenza, in Italy.—Chalybeate waters. *See* MINERAL WATERS.

CRUDESCENCE (*re-*, again; and *crudescere*, I become fresh).—The increase or exacerbation of a disease or morbid process, after a temporary diminution; for example, of fever or inflammation.

RECTUM, Diseases of. — **SYNON.**: *Fr. Maladies du Rectum*; *Ger. Krankheiten des Mastdarms*.

The diseases of the rectum may be conveniently discussed in the following order: (1) Congenital Imperfections; (2) Fistula in Ano; (3) Cancer; (4) Polypus; (5) Prolapse; (6) Non-Malignant Stricture; and (7) Ulceration. Other diseases connected with the rectum will be found discussed under special headings. *See* ANUS, Diseases of; DEFÆCATION, Disorders of; HÆMORRHOIDS; and STOOLS, Characters of.

1. **Congenital Imperfections.**—Malformations of the rectum may be classed as follows: (1) Imperforate anus, without deficiency of the rectum. (2) Imperforate anus, the rectum being partially or wholly deficient. (3) Anus opening into a *cul-de-sac*, the rectum being partially or wholly deficient. (4) Imperforate anus in the male, the rectum being partially or wholly deficient, the bowel communicating with the urethra or neck of the bladder. (5) Imperforate anus in the female, the rectum being partially deficient, and communicating with the vagina. (6) Imperforate anus, the rectum being partially deficient, and opening externally in an abnormal situation by a narrow outlet. (7) Narrowing of the anus. These imperfections can be remedied either partially or completely by surgical operations.

2. **Fistula in Ano.** — **DESCRIPTION.** — The loose areolar tissue around the lower part of the rectum is occasionally the seat of abscess, which bursts externally near the anus (*see* PERIPROCTITIS),

But instead of the part healing afterwards, like abscesses in other situations, the walls contract and become fistulous, and the patient is annoyed by a discharge from the opening. On introducing a probe it may pass through a small opening in the coats of the rectum into the bowel. The case is then called a *complete fistula*. When there is only one aperture, either mucous or cutaneous, the term *incomplete fistula* is used. The external orifice is usually near the anus, the internal between the two sphincters. The abscess before bursting may have urrowed to some distance, and the external orifice may be situated in the direction of the buttock or perinæum. Fistula in ano arises in several ways. It commonly originates in an abscess in the ischio-rectal fossa, the anatomical condition of the parts not favouring closure after the pus has escaped. An ulcer just within the external sphincter sometimes perforates the bowel, allowing the escape of feculent matter into the areolar tissue, and thus leads to abscess. Ulceration induced by a pointed foreign body, as a fish-bone, may also induce a rectal abscess. Fistula is common in the late stages of rectal cancer. Fistula occurs also in phthisical subjects, owing to tubercular mischief in or around the rectum. This tuberculosis may be primary or secondary, and in the discharge from such fistulæ the *Bacillus tuberculosis* has been found. The inner opening is sometimes found higher up the bowel, and there may be more than one, the sinuses being complicated.

An anal fistula is an annoying complaint. The patient is troubled with a discharge which stains the linen, and with the escape of flatus. Attacks of inflammation and suppuration are common, and the trouble produces often great mental depression, and much constitutional disturbance. Fistula is a disease of middle life, more common in men than in women.

TREATMENT.—The cure of fistula in ano is by a surgical operation.

3. Cancer.—Cancer of the rectum is common; appears with about equal frequency in males and females; and is an affection of middle life or old age. The form of cancer is that known as cylindrical epithelioma or adeno-carcinoma; and the most conspicuous feature is the infiltration of the sub-mucous and muscular coats. The growth may appear as a nodular mass springing from one side of the rectum, and more or less occupying its lumen; or it may assume the aspect of a laminar deposit in the rectal walls; or may take the form of an annular growth. The deposit tends to narrow the bowel, and is disposed to early ulceration. The growth may invade and open the vagina, urethra, or bladder, and is in time attended by glandular enlargements. Malignant disease may attack any part of the bowel, but generally appears in the lower part, within three inches of the anus. It is liable also to affect, though less frequently, the point where the sigmoid flexure terminates.

SYMPTOMS.—The disease generally commences insidiously. Its early symptoms are often similar to those of simple stricture, and the real disease is usually not detected until a considerable change has taken place in the condition of the bowel. The patient is troubled with flatulency; has difficulty in passing his motions; and as the disease progresses, experiences pains about the sacrum, which gradually increase in severity, and dart down the limbs. The stools become relaxed and frequent; contain blood;

and in passing cause a scalding pain. Often also there is a thin offensive serous or sanious discharge. The first symptom complained of may be an obstinate diarrhoea. Loss of retentive power may ensue, from destruction of the sphincter, or of the nerve supplying the muscle. As the disease advances the patient loses flesh, and exhibits the blanched, sallow look, anxious countenance, and emaciated appearance commonly observed in persons suffering from malignant disease. In consequence of communications established with the neighbouring passages, liquid feces may escape from the urethra in the male and vagina in the female; and at length the patient becomes exhausted by this painful and distressing malady. Complete obstruction may occur, and accelerate the fatal termination. There is great variety in the degree of suffering, and of constitutional derangement. The pains are in some instances excruciating, in others very slight. If the growth can be reached by the finger, it will be found to present a hard, nodular, uneven, and ulcerated surface, and to become soon fixed.

TREATMENT.—Little can be effected by remedies in this terrible disease, beyond palliation of the symptoms, and ease from pain. The general health must be supported. The motions must be kept soft by medicines or by injections, and pain must be alleviated by narcotics, such as morphine given in suppositories or by subcutaneous injection. Local applications of cocaine answer well in some cases. The diet must be carefully regulated so that as little *débris* as possible is left in the intestine, and the bowel should be frequently irrigated with some antiseptic injection. In cases of obstruction, as well as in cases of severe suffering, life may be greatly prolonged by inguinal colotomy. Excision of the diseased bowel has also been resorted to, but not with much success.

4. Polypus.—Non-malignant tumours of the rectum are apt to assume a polypoid form and are generally classed under the heading 'polypus.' Polypus of the rectum may be conveniently considered under three headings: (1) *The soft polyp or adenoma*. (2) *The firm polyp or fibroma*. (3) *The villous polyp or papilloma*. They are all innocent growths.

DESCRIPTION.—(1) *The soft polypus* is a true adenoma, with a network of small vessels ramifying in it, and a pedicle which varies in length. The polypus is usually single, but several may exist. In children it usually makes its appearance at the anus after a stool, resembling a small strawberry, being soft in texture, granular on the surface, and of a red colour. It has a narrow pedicle, about the size of a crowquill, attached to the wall of the rectum, as a rule about two inches from the anus. It produces no suffering, but causes a slight bloody discharge, some tenesmus, and a sense of a foreign body in the bowel. It is the commonest form of polyp.

(2) *The hard or fibrous polypus* occurs in adults, is pear-shaped, and has a pedicle more or less long and thick. It seldom bleeds, but occasions a slight mucous discharge, and when the pedicle is long, the growth protrudes at the anus after stool. It is uncommon; and is due to a fibrous growth in the submucous tissue.

(3) *The villous polyp* is a pure papilloma developed from the mucous membrane of the rectum. It is soft in structure, presents the usual appearance

of a papilloma, and may have a pedicle, but is usually sessile. It is rare, is met with in adults, and is innocent. It gives rise to considerable bleeding, and usually to a mucous discharge, and the symptoms of a foreign body in the bowel. There is no doubt but that it may in time become the seat of malignant changes.

Myoma, myxoma, lipoma, cystoma, and nævus are also met with in the rectum.

TREATMENT.—The treatment involves an operation of small magnitude, as in the case of polyp in other situations.

5. Prolapse.—In relaxed states of the sphincter muscle and coats of the bowel, loose folds of mucous membrane are liable to protrude, and require replacement. This protrusion and exposure of thickened mucous membrane, with or without internal hæmorrhoids, has been described as partial prolapse of the rectum. In the true or complete prolapse there is much more than an eversion of the lining membrane of the bowel. The gut is inverted; there is a 'falling-down' and protrusion of the whole of the coats—a change analogous to intus-susception, but differing from it in the circumstance that the involved intestine, instead of being sheathed or invaginated, is uncovered and projects externally. In the majority of instances the prolapse, even when extensive, concerns the mucous membrane only.

ÆTIOLOGY.—Prolapse is observed generally between the ages of two and four, but may occur later in life. In infancy it may be produced by protracted diarrhoea or by worms. The straining efforts to pass water in stone in the bladder also give rise to this affection in young subjects. In adults the descent results chiefly from a weakened condition of the sphincter and levator ani muscles. It is more common in women than in men, arising in the former from the parts being weakened in child-bearing. It may follow also upon dysentery, upon conditions producing straining, and upon rectal polyp. Young subjects generally outgrow this complaint by the period of puberty; and common as is prolapse in early life, it is rare in young adults.

DESCRIPTION.—The length of bowel protruded varies from an inch to six inches or even more. When not of any great length, the protrusion forms a rounded swelling which overlaps the anus, at which part it is contracted into a neck. In its centre there is a circular opening communicating with the intestinal canal. An inversion of greater length forms an elongated pyriform tumour, the free extremity of which is tilted forwards or to one side. The protrusion may present the usual florid appearance of the mucous membrane; or a violet livid colour from congestion, consequent upon contraction of the sphincter. The mucous surface is often thickened and granular, and sometimes ulcerated from friction against the thighs and clothes. Thickening of the coats of the bowel accounts for the difficulty in reducing the parts, and in keeping them reduced afterwards, a trouble so often experienced in the treatment of these cases in children, the bowel being too large to be conveniently lodged in its natural position, and, like a foreign body, exciting the action of expulsion. An atonic or relaxed state of the sphincter muscle is shown by the facility with which one or two fingers can be passed through the anus even in young children.

TREATMENT.—In children irritability of the bowels and diarrhoea must be checked, and disordered secretions corrected by suitable remedies. In slight cases it will be sufficient to direct the nurse by steady compression to press the protrusion back within the sphincter. The relaxed state of the membrane may be treated with astringent injections of alum, or with a solution of tannic acid in glycerine, or with hamamelis. If the bowels slip down when the child moves about, rest must be insisted on. When the exposed surface is ulcerated, it may be painted with a solution of nitrate of silver, 20 grains to the ounce. The patient should be made to relieve the bowels in the recumbent posture. In adults, if no relief follows the removal of the apparent cause, the regulation of the bowels, rest, and the use of astringent applications, the complaint may be remedied by operation.

6. Non-Malignant Stricture.—Non-malignant stricture of the rectum is usually due to the development of cicatricial tissue, and has followed injuries, suppuration, dysentery, syphilis, and tubercular mischief. Cripps has shown that long-continued muscular spasm may play an essential part in the production of stricture.

Simple stricture is met with in adult life, is more common in women than in men, and is usually situated within three inches of the anus. The stricture may be *annular* or ring-like; or the obstruction may assume a *valvular* form; or a large part of the length of the bowel may be involved, producing a *tubular* stricture. The mucous coat above the stricture may be ulcerated. Often ulcerated apertures lead to fistulous passages, extending some distance, and opening externally near the anus or in the buttock, and, in women, in the vagina.

SYMPTOMS.—The earliest symptom is habitual constipation, with difficult defæcation when the motions are solid. As the contraction increases, the constipation becomes more obstinate, and the stools are diminished in calibre, and are often voided in lumps. A brown slimy fluid escapes with the motions, and there is a burning sensation after stool, and flatulent distension of the colon. As the disease makes progress and ulceration ensues, the discharges become purulent and bloody, and the sufferings are much increased. There is sometimes so copious a discharge as to mislead the practitioner, the stricture being overlooked, and the case treated as one of protracted diarrhoea. The appetite may remain good, and the general health may be but little impaired; but in the course of time the derangement of the digestive functions, the irritation kept up by the disease, and the exhausting discharges, bring on symptoms akin to hectic. The appetite fails, the body emaciates, the abdomen becomes more distended, and the stricture directly or indirectly proves the cause of death. This is sometimes hastened by a lodgment of hardened feces or some foreign body just above the stricture, so as to block up the passage, and occasion all the symptoms of intestinal obstruction. In patients with stricture small flattened excrescences are often observed at the margin of the anus. These cutaneous growths resemble collapsed external piles, except that they are redder in colour, and are kept moist by the escape of an irritating discharge from the bowel. Compared with cancer, the progress of the disease is very chronic, the edge of the stricture is hard and ridge-like, ulceration is absent or at least

is not marked, induration is slight, there is less bleeding, and less fixity of the part.

A stricture in the lower part of the rectum can be easily detected by the finger. It must be borne in mind that the bowel is liable to be obstructed by disease of the neighbouring viscera—an enlarged or displaced uterus, fibrous tumours of this organ, an ovarian growth, pelvic hæmatocele, excessively hypertrophied prostate, or hydatid tumour between the bladder and rectum.

TREATMENT.—The main object in treatment is to dilate the contracted parts sufficiently for the free passage of the motions; and this is to be effected by mechanical means—by the frequent passage of bougies, or the occasional use of special dilating instruments. Means must also be adopted to relieve the irritability of the part, and to ensure the regular passage of soft evacuations. Cocaine, belladonna, or morphine suppositories at bedtime, properly selected aperients, careful dieting, and the daily washing out of the bowel, are the remedies required. In cases which resist such measures as are indicated operation may give relief. In old inveterate strictures, wearing out the patient's strength, colotomy may be recommended.

7. Ulceration.—The chronic ulcers met with in the rectum are the following: The *tubercular*, the *syphilitic*, the *dysenteric*, those due to *injury*, *suppuration*, or *fecal impaction*, the *hemorrhoidal*, which has the same pathological basis as the varicose ulcer of the leg, and ulcers associated with chronic diarrhœa, stricture and inflammatory troubles in adjacent parts.

These ulcers have the physical characters of like ulcers met with elsewhere. The tubercular ulcer is very apt to perforate. The chief symptoms are a purulent discharge from the anus; motions loose and mixed, or coated with a slimy fluid and streaked with blood; soreness in defæcation; and occasionally tenesmus. Morning diarrhœa is often one of the earliest symptoms. A burning pain in the rectum is complained of, and often radiating pains about the hips. The characters, position, and extent of the ulceration can be ascertained by examination with the finger and with the speculum. The examination, to be satisfactory, must be conducted when the patient is anæsthetised.

All forms of non-malignant ulceration are more common in women than in men.

TREATMENT.—The treatment depends on the nature and extent of the disease, and upon the constitutional condition of the patient. In severe cases the patient should be kept in the recumbent position, and the diet should be regulated. An exclusively fluid diet may be desirable in some cases. The bowels must be attended to. The local treatment consists of antiseptic or astringent injections or ointments. Weak solutions of nitrate of silver are of value, as are also injections of bismuth mixed with starch. Iodoform or other ointment may be applied by means of an ointment-introducer. Obstinate forms of ulcer may be treated by free scraping or by even more extended operation.

FREDERICK TREVES.

RECURRENT LARYNGEAL NERVE, Diseases of.—See PNEUMOGASTRIC NERVE, Diseases of.

RED GUM.—See STROPHULUS.

REDUPLICATION.—A doubling. A term generally used in reference to the sounds of the heart. See PHYSICAL EXAMINATION.

REDUX (Lat., returned).—A term signifying the return of certain physical signs, after their temporary disappearance in the course of a disease; usually associated with crepitation in pneumonia, and with friction in pleurisy and pericarditis. Redux signs are, as a rule, significant of a favourable tendency in a disease. See PHYSICAL EXAMINATION.

REFLEXES.—See NERVOUS SYSTEM, Clinical Examination of; and SPINAL CORD, Diseases of.

REFRACTION, Disorders of.—See VISION, Disorders of.

REFRIGERANTS (*refrigero*, I cool).—DEFINITION.—Remedial agents which lower the body-heat, either in health or in disease; or which allay thirst, and impart a feeling of coolness.

ENUMERATION.—The chief refrigerants are: The whole class of Febrifuges; Water; Ice; Effervescent drinks; Acidulated drinks; and the juices of Fruits.

ACTION.—As the name implies, anything may be ranked as a refrigerant which lowers the body-temperature, and we may here consider in how far the drugs described under FEBRIFUGES have the property of cooling down the healthy organism. Quinine and alcohol have but a slight and transient lowering effect, and salicylic acid has none at all; and this is readily explained, if we believe that their antipyretic properties in fever depend on their destructive influence over the protoplasm or products of septic ferments.

Refrigerants, however, are popularly held to be those drugs which relieve the thirst of the fever-stricken patient, by moistening his dry lips and cooling his parched tongue. Ice or iced-drinks manifestly fulfil these indications; and acids well diluted, which are often the most grateful of all, act very efficiently by directly stimulating the salivary secretion.

R. FARQUHARSON.

REGIMEN (*rego*, I govern).—This word is not uncommonly used as synonymous with hygienic management. In a more restricted sense it is applied to the regulation of diet, both in health and disease. See DIET; and PERSONAL HEALTH.

REGURGITATION (*re-*, back again; and *gurgilo*, I flood).—This word is technically applied to the reversal of the natural direction in which the current or contents flow through a tube or cavity of the body. Thus the food may regurgitate from the stomach into the œsophagus and mouth; the bile from the duodenum into the stomach; and blood from the aorta or pulmonary artery into the ventricles, from the ventricles into the auricles, or from the heart into the veins, when the respective valves are incompetent. See HEART, VALVES AND ORIFICES OF, Diseases of; and RUMINATION.

REHME (Oeynhausens), in Germany.—Gaseous thermal salt waters. See MINERAL WATERS.

REICHENHALL, in the Bavarian Alps.—Common salt waters. See MINERAL WATERS.

REINERZ, in German Silesia.—Chalybeate waters. See MINERAL WATERS.

RELAPSE (*re-*, back; and *lapsus*, slipping).—The return of a disease, which has apparently ceased, during or immediately after convalescence; or of a particular symptom in the course of a disease. Relapses are well exemplified in typhoid fever and acute rheumatism.

RELAPSING FEVER.—**SYNON.**: Famine-Fever; Fr. *Fièvre à Rechute*; Ger. *Hungerpest*.

DEFINITION.—An acute infectious disease, due to the presence of a minute parasite, the *Spirillum Obermeieri*.

GEOGRAPHICAL DISTRIBUTION.—Northern Europe was the favourite habitat of relapsing fever. It has been met with in America, but not as an epidemic, having been imported from Europe, and not showing a tendency to spread. Epidemics have occurred in India and in Egypt, and were once common in the British Isles. The most extensive epidemics have arisen in Ireland in times of famine, and extended thence to England and Scotland. An epidemic was confined to Scotland in 1843, and another to London in 1868.

ETIOLOGY.—*Predisposing Causes.*—Males suffer more from relapsing fever than females, in the proportion of about 1·5 to 1. The disease is most common between the ages of fifteen and twenty-five. Season seems to have little effect, but it appears to be more prevalent in winter than at other seasons, because the other predisposing causes are more intense at that time of the year. All the causes which predispose to contagious zymotics favour more or less the prevalence of relapsing fever. The most powerful, however, are scarcity of food, overcrowding, and want of cleanliness.

Relapsing fever is contagious, and has always been found to spread in proportion to the facilities for communication. It has been transported from long distances by affected persons; it attacks attendants on the sick, and any persons exposed to its contagion; and it may be communicated by fomites. It seems to act through but a short distance.

A spirillum—*Spirillum* or *Spirochæte Obermeieri*—is found in the blood of patients suffering from relapsing fever. This micro-organism decreases in number as the paroxysms subside, and is absent from the blood during the intermissions. The spirillum was discovered by Obermeier of Berlin in 1872.

Description of the organism.—The spirillum is of considerable length, measuring '1½ to 6 times the diameter of a red blood-corpuscle,' but is exceedingly thin. It shows several regular somewhat abrupt curves, and is of uniform thickness, except at the extremities, where it is pointed. It is possessed of very active motility, having both a wavy and spiral motion, and being also capable of rapid locomotion. At the onset of and during the fever the spirilla may be present in the blood in enormous numbers. The organisms always occur free, being never found within either the red corpuscles or the leucocytes. In dried films of blood they can be stained by any of the simple stains, though they do not take the colour very deeply; but in sections of the tissues their demonstration is a matter of considerable difficulty, and one of the methods for avoiding decolourisation requires to be used, methylene blue being a suitable

dye. They are decolourised by Gram's method. Koch succeeded in staining them in the tissues with Bismarck brown.

In blood outside the body the spirillum has considerable vitality, sometimes showing active movements after several days. It is killed by a temperature of 60° C., but it can be exposed to 0° C. without being killed.

Relations to the disease.—All attempts to cultivate the organism outside the body have hitherto failed. Koch observed that in certain conditions a growth of the organism in length took place, but no actual multiplication. Soon after its discovery, however, Münch, by injecting blood containing the organism, produced the disease in the human subject; and a little later Vandyke Carter and Koch produced the disease in apes by a similar procedure. In such cases there is an incubation-period usually of three to six days before symptoms appear. In all cases, along with the onset of the fever, the spirilla appear in the blood in enormous numbers. As a result of these observations and experiments it may be considered as practically proved, even in the absence of pure cultivations outside the body, that this organism is the cause of the disease.

As already mentioned, the organisms disappear from the blood about the time of the crisis, and are absent during the 'interval.' Metchnikoff, by producing the disease in apes and killing them at various stages of the fever, found that during the period of defervescence the spirilla on disappearing from the blood accumulated in the spleen, and he alleged that they were incorporated within leucocytes in that organ. Within these cells he observed spirilla in various stages of disintegration. This process of 'phagocytosis' never occurs, according to him, in the blood, and after the spirilla have disappeared from the blood they are not present in any other organ than the spleen. Soudakewitch has performed similar experiments, and obtained practically the same results. It is, however, still unexplained why the disappearance of the organisms from the blood should start at a particular time, and should then take place so rapidly. This and other peculiarities suggest a comparison between this disease and malaria, and it has even been supposed that the parasite is a protozoon and is conveyed through the agency of insects.

ANATOMICAL CHARACTERS.—These are not marked, except where complications have caused death. The liver and spleen are both found enlarged in all cases, especially the latter organ. The digestive organs exhibit nothing particular, except in those cases where there has been long deprivation of food, or where dysentery or diarrhoea has accompanied or preceded the disease.

SYMPTOMS.—The period of incubation is uncertain, varying between one and twenty-one days. The invasion of the disease is usually marked by rigors, frequently of a trivial character, amounting only to slight chilliness. This is followed by debility and giddiness; extreme weakness is not so marked as in the early stages of other forms of continued fever. There is headache, followed after a few hours by hot skin; the temperature rises to about 105° F., or sometimes, it is stated, as high as 108°; the pulse rises to from 110 to 130, occasionally even to 140 at an early stage of the disease. The tongue is covered with a moist creamy fur, which in severe cases becomes brown

and dry in the centre, and in the worst forms becomes black all over. There is great thirst, as in all febrile diseases; loss of appetite; some abdominal tenderness, especially in the epigastric region; occasionally nausea, and more rarely vomiting; the bowels are usually confined, but in some cases diarrhoea prevails. In such cases the diarrhoea is of a dysenteric character, and is probably due to the dysenteric tendency which usually prevails in time of famine, when relapsing fever is prevalent. The skin generally presents a jaundiced hue; and careful examination will detect more or less enlargement of the liver and spleen. There is great muscular and articular pain. The pain in the back is frequently of the most intense character. Headache is more complained of than in the other forms of fever. There is sometimes, but not as a rule, delirium towards the end of the first week.

In from five to seven days from the invasion of the disease the symptoms suddenly subside, and the patient quickly becomes convalescent, being for the time apparently well. This convalescence is frequently accompanied or preceded by a critical evacuation from the bowels, kidneys, or uterus, or by profuse diaphoresis. It may be permanent, but more commonly the patient remains well for a few days or a week, and then suddenly relapses, and passes through all the symptoms previously detailed. There may be a second or a third relapse, and even a fourth has been recorded. At no time during the progress of the disease is any specific eruption developed, although on the second or third day a reddish mottled rash has been met with, which, however, is irregular in its appearance, development, and duration, and usually terminates in desquamation. Purpuric spots have been sometimes, and sudamina very frequently, met with.

COMPLICATIONS.—Pulmonary complications are not so common in relapsing fever as in typhus or enteric fever. Bronchitis, pneumonia, and laryngitis may occur, especially bronchitis, but these complications are not severe. Cardiac, arterial, or venous affections are rare, with the exception of hæmorrhages, which must be considered as being connected with the purpuric tendency which usually prevails in times of scarcity. Nervous complications are more rare than in any other form of adynamic fever. Dysentery and diarrhoea in some epidemics have proved to be most serious complications, and are of frequent occurrence whenever relapsing fever prevails. Abscess and other suppurative forms of inflammation are not common. In pregnant females attacked by this fever abortion usually occurs at an early stage of pregnancy; and premature labour, with death of the fœtus, and considerable danger to the mother, in the later stages of pregnancy. Death of the mother has sometimes happened from *post-partum* hæmorrhage.

DIAGNOSIS.—Relapsing fever is most likely to be mistaken for other forms of continued fever, and may be confounded with the eruptive fevers in their earlier stages, especially small-pox. It differs from typhus in having a higher temperature and quicker pulse at the outset; in the absence of the specific eruption, of the extremely heavy aspect of the patient, and of the delirium of typhus; in the presence of extreme pains in the back, vomiting, and jaundiced tinge of the skin; and finally in the sudden cessation of symptoms, and the tendency to relapse.

Relapsing fever differs from enteric fever in the suddenness of its onset, enteric fever having a slow invasion; the want of the marked and extensive daily variations in temperature; the absence of the characteristic abdominal symptoms and eruption; and the absence of the localised iliac tenderness and the peculiar diarrhoea of enteric fever. The tongue also serves to distinguish relapsing from enteric fever; in the latter having a well-marked red tip and edges, in the former a light covering fur. Relapsing fever at its commencement has been confounded with small-pox, on account of the extreme pain in the back and marked vomiting which accompany both these diseases, but the appearance of the specific eruption will soon decide the question.

PROGNOSIS, DURATION, TERMINATIONS, AND MORTALITY.—The prognosis of relapsing fever is usually favourable, the mortality being low, from 1·2 to 2 per cent. in London, up to 4 and 4·5 per cent. in other places; the average rate being about 4 per cent. The chief causes influencing the rate of mortality seem to be the prior state of the patient, and the duration of the disease before medical relief is applied for. Purpuric symptoms, severe dysentery or diarrhoea, serious hæmorrhages, or extensive chest-complications, always indicate a grave prognosis. One attack confers no immunity.

TREATMENT.—The treatment of the disease must be preventive and curative. The chief promoting causes of the disease being famine and contagion, the means for prophylaxis are obvious. The active treatment must chiefly be directed towards the relief of symptoms, and sustaining the strength of the patient. The use of quinine and mineral acids in the earlier stages, and a plentiful supply of light and nourishing food in the later, will be found sufficient. A considerable amount of the success of treatment will depend upon the dieting of the patient. It must be kept in mind that most of these patients have been in a state of starvation. It will be necessary, therefore, to increase the supply of food carefully and gradually. The food at first must be of a most digestible and fluid kind, which may gradually be altered to a diet of a more solid and general character. Milk, light starchy puddings made with milk, thin custards, and finally chicken, chops, and general diet will be found the best course in this disease. Stimulants may be occasionally requisite, but are seldom necessary in any quantity or for a length of time.

T. W. GRIMSHAW.

REMITTENT (*re*-, again; and *mitto*, I send).—A disease is said to be remittent when it is characterised by periodical diminutions of symptoms, followed by exacerbations, as in malarial fever and neuralgia. The period during which the symptoms are in abeyance is called a *remission*. See **MALARIAL DISEASE**.

REMITTENT FEVER.—See **MALARIAL DISEASE**.

RENAL CALCULUS.—**SYNON.**: Nephrolithiasis; Fr. *Calcul Rénal*; Ger. *Nierenstein*.

DEFINITION.—A concretion found in the substance, in one of the calyces, or in the pelvis of the kidney. This concretion is due to the deposition of one or more of the crystalloid substances from the urine.

The size depends upon its position, the shape upon its position and composition. Calculi may be present in one or both kidneys. They may occur at all ages, and are rather more common in the male sex.

ÆTIOLOGY.—In some instances the nucleus of the calculus consists of a blood-clot, of epithelial cells, or of mucus, upon which the crystalline constituents of the urine are deposited. The separation of the solid constituents is favoured by sedentary habits, by gout, and by other conditions which produce either a change in the reaction of the urine or an excess of uric acid, calcium oxalate, or other constituent of calculi. An excess of nitrogenous or saccharine food, or of salts of lime, or an insufficient supply of liquid, will also increase the tendency to deposition. Irritation of the kidney or renal pelvis by inflammation or injury, by affording a nucleus, aggravates any hereditary tendency to the formation of renal calculi. Dietetic errors and metabolic disturbances are regarded as predisposing causes, while pyelitis or other local change is considered the immediate determining cause.

VARIETIES.—Renal calculi present great variations in composition, in number, in size, and in appearance; and these variations to some extent are dependent upon the age at which they become manifest, and upon the duration of their retention at the original site of their formation. They are most frequently found to consist of uric acid, especially when formed during adolescence. In childhood, however, the nucleus commonly consists of ammonium or sodium urate. Calculi composed of calcium oxalate are more common after middle age, and this substance is often regarded as the nucleus, even of uric acid calculi; it is probable, however, that the nucleus is generally some result of local inflammation or irritation. Other lime-salts, as the carbonate, the basic phosphate, and the triple phosphate, are occasionally met with, but they are generally secondary deposits upon nuclei of uric acid or calcium oxalate; they occur when the urine has become alkaline. When the alkalinity is fixed the carbonate and basic phosphate will probably be deposited, while triple phosphate is more frequent when the alkalinity is due to the development of volatile ammonium-compounds. Very rarely calculi may consist of cystin, xanthin, or indigo (p. 333).

When a single renal calculus is found it may be rounded, oval, or slightly nodular if composed of uric acid; if consisting of calcium oxalate the surface is generally more irregular, constituting the 'mulberry' calculus. When several calculi are present, they may have distinct facets, or if small and numerous, they may form irregular granular masses, or they may even be so small as to be designated 'sand.' When long retained in the pelvis of the kidney, the shape may become extremely irregular, processes forming between the pyramids, or projecting towards the ureter. Calculi with secondary deposits may attain great size, and may present alternating strata, phosphates generally constituting the external part. The formation of secondary deposits tends to obscure primary irregularities, and to render the calculus more smooth and round or oval.

PATHOLOGICAL EFFECTS.—The consecutive renal changes depend upon the composition and mobility of the calculus. When small it may become embedded or buried in the renal tissue, and for a length of time may cause no material change be-

yond some local thickening due to chronic interstitial nephritis. Ordinarily, however, it causes some degree of obstruction, which is followed by dilatation of the renal pelvis (hydronephrosis). In either case more active inflammation may be excited, and pyelitis, pyelonephritis, or perinephritis may result, with consecutive atrophic changes. Under these conditions there may be considerable destruction of renal tissue, especially when suppuration is pronounced. Although these changes are primarily limited to one side, the opposite kidney frequently becomes similarly involved, especially when the original calculus is composed of uric acid or urates.

SYMPTOMS.—When the calculi are small they may occasionally be passed without causing any symptoms. Larger concretions, embedded between the pyramids, may also cause no symptoms, but usually there is complaint of unilateral pain referred to the region of the kidney, and subject to exacerbations with moderate movements or with unsuitable diet. The lumbar pain, which is so often met with in gouty patients, is probably due to the presence of renal calculi. These attacks of renal pain are sometimes very acute, and during the attack the pain may radiate along the ureter to the groin and testicle, or it may be referred to the thigh, the calf, or the foot. When, however, there is definite obstruction in the ureter, the result of the passage of a calculus or of a plug of mucus, renal colic is excited, in which the pain is of an agonising character, and frequently accompanied by nausea, vomiting, and hæmaturia. Renal colic is due to the spasmodic contractions of the ureter, and, although it results ordinarily from the displacement of a calculus, it does not invariably indicate that the calculus has actually passed from the pelvis to the ureter; severe spasm may be excited so suddenly that the onward progress of the calculus may be thereby obstructed. The pain is primarily referred to the region of the kidney and the ureter on the affected side, and may cause retraction and tenderness of the testicle. Occasionally it can scarcely be localised; it appears to radiate through the abdominal cavity, and may even be referred to the umbilicus. The duration of renal colic is extremely variable. It may last only for a few hours, being followed by aching or soreness in the course of the ureter, or it may continue, with periods of partial relief and with exacerbations, for several days. There may be sudden relief, especially after a fit of vomiting, when the stone has passed into the bladder, but even this is ordinarily followed by discomfort along the ureter. With the onset of renal colic there is frequent desire to micturate, and there may be tenesmus; the urine is passed in small quantity, and is commonly intimately mixed with blood. Occasionally, however, when there is complete obstruction of one ureter, the urine may remain clear. Cases of suppression of urine are by no means uncommon after repeated attacks of renal colic upon both sides of the body. In these, for five or six days, there may be no symptoms beyond the partial or complete arrest of excretion, and then uræmic symptoms develop, such as hiccough, vomiting, and muscular spasms. The temperature falls, and death may occur between the ninth and twelfth days from failure of respiration and circulation. Convulsions are uncommon with calculous anuria. With repeated attacks of renal colic the urine frequently becomes turbid with pus owing to pyelitis.

DIAGNOSIS.—Renal calculus may be mistaken for recurrent attacks of intestinal colic, for biliary colic, for appendicitis, for lumbago, for tubercular or malignant growth of the kidney, or for hydronephrosis due to some abnormality in the position or length of the ureter. The indications upon which greatest reliance can be placed are the nature of the colic and the frequency of hæmaturia. The association of these two symptoms may render the diagnosis easy, especially when the history shows that small calculi have been passed. The site of fixed pain, and the recurrent exacerbations with sudden jolting movements, favour the diagnosis, even in the absence of hæmaturia. Frequent deposition of oxalates or of uric acid will tend to the same view. The general health suffers very little in renal calculus, as compared with tubercular or malignant growths; when complicated with pyelitis or other suppurative change the deterioration is more marked. Recurrent hæmaturia with comparatively little lumbar pain may be mistaken for chronic nephritis, with sub-acute attacks: the diagnosis is to be based upon the absence of arterial change, of displacement of the apex-beat, and of alteration in the cardiac sounds. With movable kidney and with hydronephrosis from alterations in the ureter, the character of the pain is frequently indistinguishable from renal colic the result of calculus; often the uncertainty is so great that surgical measures are undertaken on the probability of an embedded calculus. In doubtful cases the X rays may afford assistance; but they frequently fail even when subsequent operation has proved the presence of a calculus. *See RÖNTGEN-RAYS.*

PROGNOSIS.—The prognosis is extremely uncertain. Calculi have been found after death when no symptoms have resulted from their presence. The irritation in the kidney may be so slight that, suitably treated, there is little risk. On the other hand the frequent recurrence of pain, when calculi have been passed, and the transference of pain to the opposite side render the prognosis more grave. Persistent slight albuminuria, or the development of pyelitis, will also add to the risk. When the aim has been bilateral the danger of calculus suppression must be borne in mind. The prognosis is most hopeful when, after the passage of a calculus and its removal by surgical measures, there is prolonged freedom from pain and from deposition of oxalates or oxalates.

TREATMENT.—The treatment of renal calculus may be considered under separate headings: (1) Medicinal; (2) Hygienic and dietetic; (3) Surgical. The medicinal treatment must depend upon the symptoms at the time advice is sought. Generally the first indication is the relief of a severe attack of renal colic. The pain may be controlled to some extent by the frequent subcutaneous injection of small doses of morphine. These are preferable to large single dose, which may perhaps increase the severity of vomiting. Simultaneously a hot bath could be employed, or if this is not convenient muscular relaxation may be promoted by a hot pack. To diminish the acid character of the urine, and thus to favour relaxation of spasm, copious draughts of milk, of water, or of dilute alkaline solutions are often recommended; frequently, however, reflex vomiting is so persistent that it is impossible to carry out this treatment. The pain may be so severe that relief may be sought from the administration of an anæsthetic; this rarely gives

much satisfaction although it affords temporary ease. The utmost that can be said in its favour is that it may allow time for the excretion of fluid, which, during the muscular relaxation, may favour the dislodgment of the calculus.

The medicinal treatment during the intervals between attacks of colic is similar to the treatment of gout. An alkaline course is often recommended, and to prevent fresh depositions patients often resort to foreign spas, where alkaline waters are reputed to possess solvent properties. Carlsbad, Vichy, Contrexéville, Ems, and Wildungen may reduce the frequency of attacks, and may prevent the formation of fresh calculi. It must be admitted, however, that in spite of all that has been said in their favour, or in favour of the administration of alkalis, there is no evidence that it is possible to dissolve or to reduce the size of calculi which have already been formed.

Potassium citrate has been given at night in drachm-doses, to reduce the acidity of the morning urine, and many have recommended various compounds of lithium, of potassium, and of calcium with the same object. More recently piperazine, lycetol, lysidine, chinotropine, and urotropine have been largely employed to favour diuresis and to exert solvent action on uric-acid calculi. The last named, urotropine, is frequently of service when the calculi have caused pyelitis.

Similarly hygienic and dietetic measures control the tendency to the formation, but cannot effect a cure. After recovery from an attack of renal colic active open-air exercise should be encouraged to promote metabolism, and the patient should lead a regular temperate life. When the urine is acid, meat should be taken sparingly and the daily amount of water, simple or alkaline, should be increased. Although vegetables are usually recommended, care must be taken to ascertain, if possible, the nature of the calculus which may have been passed, since if it consists of oxalates or phosphates many vegetables and fruits must be excluded from the dietary. Any tendency to obesity should be counteracted, and, partly with this object, ale and stout should not be taken. Most forms of alcohol are prejudicial to patients with renal calculus.

Surgical measures may be necessitated when frequent renal colic and hæmaturia are not followed by any evidence that the calculus has passed along the ureter, or when the renal pain is persistent and exhausting, or when sudden suppression occurs. Surgical interference may also be required when the urine assumes a purulent or a septic character. By comparatively early operation it is often possible to avert the disorganisation of the kidney which so often ensues after prolonged irritation by renal calculi. The nature of the operation must be determined after the exposure of the kidney; unless the calculi are very numerous or the disorganisation is considerable, it is generally advisable not to remove the kidney, since the opposite kidney may already have been destroyed or rendered inefficient by other calculi.

NESTOR TIRARD.

RENAL COLIC.—*SYNON.*: *Nephralgia Calculosa*; *Fr. Colique Néphrétique*; *Ger. Nierenkolik*.—The name commonly applied to the symptoms which arise when a renal calculus, blood-clot, or other substance either passes, or attempts to pass, down the ureter. *See COLIC*; and *RENAL CALCULUS*.

RENAL DISEASES.—See KIDNEYS, Diseases of.

RENNES-LES-BAINS, in France (Aude).—Muriated alkaline and earthy sulphated waters. See MINERAL WATERS.

RESISTANCE.—The sensation recognised by the fingers of the degree to which a part yields or resists when palpation or percussion is being performed. See PHYSICAL EXAMINATION.

RESOLUTION (*resolvo*, I loose).—The return of a diseased part to its natural condition; chiefly applied to the process of inflammation when it subsides without the occurrence of suppuration or other unfavourable termination. See INFLAMMATION.

RESONANCE (*resono*, I sound again, echo).—Resonance signifies the character of the sound yielded on percussion over the greater part of the chest, and, within wide limits, of the abdomen also. The degree of resonance depends principally upon the proportion of air contained in the underlying cavities or organs. *Vocal resonance* is the voice-sound transmitted through the chest to the ear of the auscultator. It is increased or diminished in accordance with the physical conditions present in the chest-cavity.

Hyper-resonance is a term used to signify undue resonance over a given part.

Deficient resonance is commonly called *dulness*. Like *hyper-resonance*, it is often used in a relative sense, in comparing the percussion-note of different parts of the chest or abdomen. See PHYSICAL EXAMINATION.

R. DOUGLAS POWELL.

RESPIRATION, Disorders of.—The numerous causes and conditions which lead to disturbances of respiration can be brought within well-defined groups, and it is desirable in the first instance to study them from such a general point of view. This, however, only gives a superficial insight into the subject, and does not indicate the kind of disorder that is produced by each cause, or how it acts, while many conditions act in more ways than one; and, still further, in any individual case there may be more than one, perhaps several causes at work, all of which ought to be recognised. These causes and conditions may be summarised in the following manner, and it will be seen that several of them act indirectly on the respiratory process:—

1. *Conditions acting directly through the nervous system.*—These include: (a) Centric lesions in connection with the brain, involving the respiratory centre, either directly or indirectly, such as injury, hæmorrhage, or a tumour. (b) Disease or injury of the upper part of the spinal cord, paralysing the nerves supplying the respiratory muscles. (c) Functional nervous disturbance, as from mere nervousness, emotion, hysteria, trance, or chorea. (d) Conditions affecting immediately the nerves concerned in respiration, either irritating or paralysing them, especially the pneumogastric, recurrent laryngeal, or phrenic nerves. These nerves may be themselves diseased, or affected by some neighbouring condition, such as a tumour. (e) Reflex causes, transmitted from the skin, as when cold water is dashed upon its surface; or from internal organs, as the

stomach, intestines, or ovaries. It is important to remember that disturbances connected with the nervous system frequently aggravate disorders of breathing originating from other causes.

2. *Abnormal conditions of the blood.*—In this group are included conditions of the blood as a whole, namely: (a) Deficient quantity of blood, especially from a sudden or rapid loss. (b) Anæmia or hydræmia. (c) Deficient aëration. (d) A poisoned, impure state of the blood associated with narcotism, the anæsthetic state, pyrexia, the typhoid condition, uræmia, pyæmia or septicæmia, diabetes mellitus, and other diseases.

3. *Functional disorders, or organic diseases connected with the heart.*—These are common sources of disturbance of breathing of various kinds, depending upon the intimate relation of the nerves and nerve-centres governing the heart and respiratory organs; upon the effects the cardiac derangements produce as regards the pulmonary and bronchial circulation; or sometimes upon the direct interference by certain affections with the movements of breathing, by exerting pressure upon the lungs, especially the left, upon the left bronchus, or upon the chest-walls, and particularly the diaphragm. This last cause is only noticed in cases of great enlargement of the heart, or of considerable pericardial effusion. The breathing, however, is frequently disturbed in connection with disorders of cardiac action; diseases of valves and orifices, especially the mitral and tricuspid; enlargements of the heart, particularly dilatation; cardiac degenerations; congenital malformations; and clotting of blood in the cavities of the heart.

4. *Abnormal conditions of the air inhaled.*—The physiological effects produced upon the respiratory act by various states of the air inspired are well known. These especially depend upon its composition; its temperature; its degree of moisture or dryness; and its condensation or degree of pressure. From a clinical point of view these effects have to be borne in mind, as they are more liable to be induced in certain diseases, and may also be made available for therapeutic purposes. The presence of certain gases in the inspired air materially affects the breathing, and some are in this way more or less poisonous. Solid particles floating in the atmosphere may also produce disorders of respiration.

5. *Conditions affecting the apparatus concerned in the respiratory movements.*—These refer to the chest-walls and the diaphragm, and they include: (a) Certain painful affections, causing the patient voluntarily or involuntarily to limit or modify the movements, such as the early stage of pleurisy, pleurodynia, or peritonitis. (b) Spasm or paralysis of the muscles, from any cause. (c) Organic changes, as undue softness or rigidity of the thoracic walls, cancerous infiltration, muscular atrophy or fatty degeneration, or acute or chronic inflammation of the diaphragm.

6. *Obstruction involving the main air-passages.* This may be situated in the mouth, throat, nasal cavities, larynx, trachea, or primary bronchial divisions, and is due to a variety of causes, which cannot be discussed here further than to state that the obstruction may depend upon pressure from without; spasm or paralysis of the muscles of the larynx; some internal obstruction, whether from deposits, secretion, abscess (including also retropharyngeal abscess), foreign bodies, or new-

growths; or organic changes in the walls of the tubes, leading to their constriction.

7. *Physical conditions independent of the respiratory apparatus, but interfering with it in various ways.*—These may lie within the chest, as in the case of thoracic aneurysm, or a mediastinal solid tumour or abscess. They act by compressing the lungs or heart, obstructing tubes, affecting nerves, or interfering with the moving apparatus. Or the cause of the disorder may lie in the abdomen, such as excessive flatulence or tympanites, ascites, enlarged organs, ovarian tumours, or a pregnant uterus. They act mainly mechanically, by impairing the movements of the diaphragm. Breathing often becomes worse after food in cases in which it is difficult, in consequence of flatulent distension, especially when digestion is impaired.

8. *Conditions affecting the pleura.*—Any accumulation of air or fluid in one or both pleural sacs will necessarily tend to disturb respiration, as in pneumothorax, pleurisy, hydrothorax, or hæmothorax. It acts mechanically, and the degree of disorder will depend on the amount of the collection, the rapidity with which it takes place, the previous condition of the lungs, and other circumstances. Pleuritic adhesions also tend to embarrass respiration more or less seriously.

9. *Morbid conditions of the lungs.*—These have been left to the last, and it will be readily understood that all diseases of the lungs tend more or less to produce disorders of breathing. At the same time it must not be forgotten that these organs may be affected, even somewhat extensively, under certain conditions, without any obvious respiratory disturbance. Pulmonary diseases act in various ways, of which the most important are by affecting the circulation and the amount of blood in the lungs; by interfering with the entrance or exit of air through the bronchial tubes; by temporarily disabling or permanently destroying more or less of the pulmonary textures; or by influencing the respiratory act through its forces, and especially by the impairment or loss of the elasticity of the lungs required for expiration.

CLASSIFICATION.—The disturbance of breathing may be sudden, acute, or chronic; and its several forms may be included under three main divisions, namely: (1) **Deficient Respiration.** (2) **Dyspnœa or Difficulty of Breathing.** (3) **Peculiar Disorders.**

1. **Deficient Respiration.**—This comprehends the following varieties:—

(a) *Slow breathing.*—The frequency of the respirations may be notably reduced, without any other obvious disorder. Or this may be associated with marked shallowness of the movements, so that in extreme cases breathing seems to have almost entirely ceased, and can scarcely be recognised even by the most delicate tests. These deviations are observed in various conditions or diseases affecting the nervous system, such as hysteria, trance, shock or collapse, narcotic poisoning, and some cases of cerebral disease. They are accompanied by impairment or loss of consciousness, real or assumed, and by other varying symptoms. Sometimes the breathing is slow but deep, and may then be sighing, stertorous, or attended by flapping of the cheeks in expiration; this is noticed in apoplectic conditions. These disorders of breathing do not obviously disturb the patient.

(b) *Restrained breathing.*—By this is meant that the patient makes a voluntary and conscious effort to restrain or modify the act, because it produces or increases some painful or other morbid sensation. It may be obvious at once to the observer, or may only be revealed when the patient is made to take a deep inspiration. The respirations are often increased in frequency, but may be below the normal. The entire movements may be affected, or only those of either the chest or the abdomen, or even only of one side of the chest. The early stage of pleurisy, peritonitis, and angina pectoris afford examples of diseases causing this disorder of breathing.

(c) *Shallow and feeble breathing.*—The most striking feature in some conditions is the extreme feebleness and limitation of the act of respiration. This has already been alluded to, as noted in some cases of slow breathing, but the frequency is often much above the normal, and the class of cases now under consideration differ essentially from those previously mentioned. The disorder indicates gradual cessation of the respiratory functions and pulmonary action, becoming more and more obvious, and gradually terminating in death. Little or no air is changed, and at last the breathing becomes a mere ineffectual gasp. This form of disturbance is observed in persons slowly dying from various causes; in gradual filling of the air-tubes in fatal cases of bronchitis; and in cases of apoplexy or narcotism. It is often accompanied by rattling or gurgling râles, audible to the bystander, due to the presence of fluid in the air-passages, which become by degrees filled up. It may follow certain forms of dyspnœa.

(d) *Ineffectual breathing.*—The derangement thus named can only be recognised by making the patient attempt to draw a full breath. He may then be conscious of an inability to perform this act satisfactorily, or to expand the chest properly. What is more important, however, is that this impairment of the respiratory act is often evident on objective examination, when it is seen that in certain conditions the most powerful efforts to breathe produce little or no result, and the movements are obviously more or less ineffectual, either as a whole, unilaterally, or locally. This may arise from various causes, such as paralysis or spasm of the muscles, rigidity of the chest-walls, distension of the lungs in emphysema, pleuritic and other conditions interfering with their expansion, and certain morbid changes in these organs themselves. Ineffectual breathing is frequently associated with some form of dyspnœa.

2. **Dyspnœa or Difficulty of Breathing.**—Without making too marked a distinction between them, and remembering that they may be variously combined, there are certain forms of disordered breathing, usually characterised as *dyspnœa*, which deserve separate recognition.

(a) *Obstructive dyspnœa; Inspiratory dyspnœa.* This signifies that there is some obvious impediment or difficulty presented to the transmission of air through some part of the air-passages in respiration. The nature and severity of the disorder vary with the seat, cause, and degree of obstruction. Thus it may be that a swollen tongue, enlarged tonsils or other throat-condition, retro-pharyngeal abscess or some obstruction in the nose or naso-pharynx, blocks up the passage more or less completely, and the patient breathes with obvious difficulty; air

passing by the throat causes much noise, especially when the patient is asleep.

The most important form of obstructive dyspnoea, however, is that which is connected with the main air-tube, and it usually attracts immediate attention. It may be associated with the larynx or the trachea, or with both, and in the case of the larynx is liable to exacerbations. The gravity of the phenomena varies with the degree of obstruction, but they are more or less of the following kind. The patient is usually conscious of a difficulty in the passage of the air during respiration, referred to some spot, which may become very distressing; the act of breathing is usually more or less laboured, and this may culminate in a violent effort or struggle to breathe. The frequency of respiration is often below the normal, or at any rate it is but little increased, while the relative length of inspiration and expiration is disturbed. The difficulty may be experienced only during inspiration, or during both divisions of the act of breathing, but is usually most marked in inspiration, though occasionally during expiration. Various noises are produced by the passage of the air through the narrowed part, usually classed as *stridor*, the breathing being termed *stridulous*; and to an experienced and trained ear these become of great importance as indicating the existence and seat of obstruction. Signs of deficient aëration of the blood are very liable to accompany this form of dyspnoea; and in acute or sudden cases, or if the obstruction is very marked, there is danger of actual suffocation which may occur rapidly or even suddenly. Physical examination will indicate that air does not enter properly into the lungs, as evidenced especially by recession of the lower part of the chest during inspiration, particularly marked in children, in whom this form of dyspnoea is likely to lead to most serious consequences.

The obstruction may be situated lower down in the respiratory tract, either in one of the main bronchial divisions, or in the tubes distributed through the lungs, and then the character of the disorder merges in that of ordinary dyspnoea, except that it is likely to be attended by various noises, and that the physical signs of deficient entrance of air into one or both lungs are evident. When there are evident objective signs of deficient entrance of air into the lungs, the condition is termed *inspiratory dyspnoea*. This difficulty, however, may also depend upon weakness of the chest-walls, and of the inspiratory muscles, as in rickets.

(b) *Excessive breathing*.—*Ordinary dyspnoea*.—This is the disorder usually met with in various degrees, and it implies that respiration is carried on in excess. The act may be too frequent, or too powerful, or both, so that more than the ordinary amount of air is changed in a given time. The movements of the chest are more or less free under different circumstances. In severe cases the patient is obviously distressed, and the act of breathing is laboured, and may be noisy. Then the *alæ nasi* are seen to work; the patient cannot speak except in broken sentences, owing to want of breath; and there may be signs of apnoea. This form of dyspnoea is familiarly illustrated by the effects of undue exercise, such as running. Clinically it is associated in different degrees with numerous conditions, such as nervous disorders; fevers and other blood-conditions; many cardiac affections; conditions interfering with the action of the lungs,

such as pleuritic effusion or abdominal accumulations; and various diseases of these organs, impairing their functional activity, especially if acute, such as pneumonia or bronchitis.

(c) *Shortness of breath*.—While associated with other forms of dyspnoea, this disorder frequently exists alone in various degrees, and it may be of much consequence in drawing attention to disease of a serious character. Shortness of breath signifies that the breathing becomes more or less hurried, and the individual becomes conscious of dyspnoea, after making some effort which ordinarily does not cause any such effects, such as walking rather quickly or upstairs, singing, coughing, stripping, or even taking a few deep breaths in physical examination of the chest. When at rest he may feel perfectly comfortable, and breathing is quite natural, but it is easily disturbed in the ways above indicated. This disorder is observed in general debility; very markedly in pronounced anæmia; in many cardiac conditions, especially dilatation and degeneration; in pleuritic effusion frequently; and in many cases of chronic lung-disease, such as phthisis or emphysema.

(d) *Expiratory dyspnoea*.—In the form thus designated the difficulty is experienced during expiration, which becomes prolonged and laboured, in some cases extremely so, the extraordinary muscles of expiration being called fully into play. The relative lengths of inspiration, expiration, and the pauses are thus deranged, and inspiration may become extremely short, even a mere gasp. There is often a sense of discomfort or even distress, and this is liable in certain conditions to be increased by exertion, after taking food, or in certain postures. Expiratory dyspnoea may be a prominent feature in some cases of obstruction of the air-tubes; but is essentially connected with impairment of the expiratory elastic force of the lungs in cases of emphysema, and of the chest-walls when they are rigid, these two conditions often going together. These causes are frequently aided materially by blocking-up of the bronchi, as the result of bronchitis; or by spasmodic contraction of these tubes, in connection with asthma.

(e) *Orthopnoea*.—This is almost always combined with one or more of the other forms of dyspnoea, and the term indicates that the patient can only breathe at all, or at any rate with any degree of comfort, when the body is in a more or less upright posture. In some cases it is sufficient if he is propped up; in others he has to sit bolt upright in bed, or to bend forwards; in others still he is obliged to sit up altogether in some kind of chair, or even to stand, this being the only posture in which breathing can be carried on with any comfort. Cases of extensive cardiac disease, of acute pericardial and pleuritic effusion, of acute pneumonia, of asthma, and of aneurysmal or other thoracic tumours, afford illustrations of the causes of this disorder.

(f) *Paroxysmal dyspnoea*.—This may be of various kinds, but, as its designation implies, it signifies that the dyspnoea comes on mainly or entirely in fits or paroxysms. It is chiefly exemplified by paroxysms of laryngeal dyspnoea; by some cases of cardiac dyspnoea; and, above all, by fits of bronchial asthma. See ASTHMA, SPASMODIC.

3. *Peculiar Disorders*.—It is scarcely practicable to bring these under any definite subdivisions, and it will suffice to notice the very curious and

ten indescribable disorders of breathing observed in certain nervous cases; the interrupted, jerky, gasping, or yawning respiration which may be present in various conditions; and the peculiar disturbance generally known as *Cheyne-Stokes respiration*, but also termed *rhythmic dyspnoea*. This is rare, but may occur in connection with certain cardiac diseases, especially fatty degeneration; Bright's disease; injury to the brain; and cerebral hæmorrhage, or other brain-lesions. It is characterised by the breathing at intervals becoming by degrees more and more rapid and deep up to a certain point; and then subsiding in the same gradual manner, until finally there is a complete cessation of respiration, with a dead silence, the pause lasting a variable time, and then the same series of phenomena being repeated.

EFFECTS.—Many of the disorders of breathing which have now been considered are not attended by any obvious effects, and are practically of little or no consequence. Moreover, it must be noted that patients may become so accustomed even to marked derangements of the function of respiration, that they are not conscious of any injurious results therefrom. Most individuals under such circumstances, however, are conscious of more or less discomfort or other sensations, referable to some part of the respiratory apparatus. These are very unreliable and vague in their meaning; but there are defects which give important information in many cases, and which depend either upon the want of due aëration of the blood, or upon the interference with the general venous circulation which disorders of breathing so frequently induce. These will vary, not only with the nature of the disorder, but also with its degree, and the rapidity with which it is set up. Thus there may be actual suffocation, sudden or rapid, or a condition approaching more or less that of asphyxia or apnoea (*see ASPHYXIA*). Or a chronic state of venous congestion and venosity of the blood may be set up, indicated by a tendency to cyanosis, with enlargement of the visible superficial capillaries; general chilliness and coldness of the extremities; mental apathy or dulness, with headache and other signs of morbid blood-supply to the brain; general languor, laziness, and muscular weakness; dyspeptic disorders; changes in the urine; and other well-known phenomena. In cases where the respiratory functions are chronically affected in children and young persons, in such a way that the blood is never properly aërated, growth and development are markedly impeded. Patients suffering thus may present a peculiarly stunted appearance. The features tend to become permanently thick and coarse; and the ends of the fingers and toes often become clubbed. In certain forms of dyspnoea the fat of the body tends to disappear; while the muscles of respiration not uncommonly become hypertrophied from excessive use.

TREATMENT.—The indications to be fulfilled in treating disorders of respiration, and the measures by which these are to be carried out, must obviously present considerable variety in different cases; and it will only be practicable here to offer a few general hints on the subject. In the first place, no treatment whatever may be called for in some instances; while in other cases nothing can be of any service. The primary indication should always be to attend to the cause of the disorder, and by curing, removing, or alleviating this, the disturbance

may often be got rid of or materially diminished. This may be illustrated by treatment directed to laryngeal obstruction, anæmia, pleuritic pain or effusion, bronchitis, or cardiac derangement. By improving the condition of the blood when anæmia is present, breathing is frequently much improved, even when actual disease exists which disturbs it, such as phthisis or cardiac mischief. Attention to the condition of the air inhaled is in some cases of much importance, as regards its purity, temperature, degree of moisture, pressure, and other points. It must be remembered that some forms of dyspnoea actually require an atmosphere which contains an abnormal proportion of carbonic acid. Great advantage frequently results from giving proper instructions to patients as regards posture, avoidance of exertion, diet, the act of coughing, or even the act of breathing itself. This is especially important in certain forms of paroxysmal dyspnoea; and any cause which is known to produce any such attack should be carefully avoided. Moreover, the patient may sometimes be materially assisted in the act of breathing by mechanical means. Not uncommonly active measures are called for, for the purpose of relieving some more or less urgent form of dyspnoea. For this purpose various means are indicated in different cases, such as venesection, or local removal of blood from the surface of the chest; dry-cupping over this region; the internal administration of antispasmodics, stimulants, pulmonary sedatives, or other appropriate agents; inhalations of different kinds, in the form of gas, vapour, or smoke; subcutaneous injections of morphine or other active drugs; or the application to the chest of sinapisms, hot poultices, fomentations, or turpentine stupes. Treatment directed to the asphyxial condition may be urgently demanded, especially artificial respiration (*see ASPHYXIA*); and operative procedures, such as laryngotomy or tracheotomy, or intubation, may be called for in cases where the main air-tube is obstructed. In chronic cases, where the respiratory functions are imperfectly carried on, the conditions resulting therefrom must be remembered, and as far as possible obviated. Warm clothing is essential under such circumstances; and, if practicable, a residence in a genial and warm climate is often of the greatest practical service.

FREDERICK T. ROBERTS.

RESPIRATORY MURMUR.—The sound heard on auscultation over the lungs in respiration. *See PHYSICAL EXAMINATION.*

REST, Therapeutics of.—In considering rest as a therapeutic agent it is requisite to understand its nature, its varieties, the indications for its use, and the ways of employing it. There are two chief varieties: (1) Rest of the *body and brain generally*; and (2) local rest of a *diseased organ or inflamed part*.

1. *Rest of the body and brain generally.*—*See NEURASTHENIA; PHTHISIS; and PERSONAL HEALTH.*

2. *Local rest.*—This, which may be called *mechanical rest*, is well known to every surgeon to be an agent of supreme value in the treatment of wounds, fractures, displacements, or inflammation of joints; as it is obvious that every movement to which a wounded or inflamed part is subjected must act on the one hand like a repetition of the original injury, and on the other hand like a

continuance of the irritating cause. Thus rest is not only a negative advantage, as saving the patient from renewed injury or irritation, but a positive remedy, as it diminishes the heat of the body, reduces the pulse, and alleviates pain. Rest is of so much value in the treatment of inflammation, that in some instances no means will advance the cure without it, and numerous injuries of the body, external or internal, would do well with perfect local rest and nothing else.

It was on this principle that Pott treated all fractures of the extremities, by placing the limbs in a position of easy flexion, so that the muscles which had been thrown into spasm by the fracture were relaxed.

APPLICATION.—The application of rest in diseased conditions of the different parts of the body is so varied, and the cases in which it should be employed are so numerous, that it would be impossible to enumerate them all. In surgical practice and in the treatment of diseases of the eye, rest in one or all its varieties is the daily routine. In this article we shall only deal with its employment in medical practice, and shall select a few examples out of many to illustrate its benefit in different regions of the body.

A. Diseases of the Respiratory Organs.—

The objects of the treatment by rest may be stated to be (Roberts): (1) To maintain structures which are actually diseased, or in danger of becoming so, in as quiescent a state as possible; in short, to try to produce mechanical rest, as is ordinarily done in the case of a diseased joint. (2) To check or limit the entrance of irritating gases—be they noxious, or simply of a different degree of temperature or humidity from that of the internal part with which the air comes in contact. (3) To quiet the circulation through the organs which are being placed in a condition suitable for repair.

1. *Acute inflammation of the larynx and bronchi.*

The patient is to be placed in an equable and moderately high temperature, and the atmosphere impregnated with moisture; all speaking or using the voice must be forbidden, while the patient's wants may be made known by means of a slate and pencil (Hilton).

In addition to *general rest*, *physiological rest* is obtained by remedies to relieve the congested right heart, and to remove the mucus which is causing the symptoms of asphyxia. Here relief is attainable by restraining on the one hand the outpouring of mucus into the small tubes of the lung, and getting rid of that which is already poured out, by means of alkaline and stimulating expectorants; and by maintaining, on the other hand, the forces of the circulation, and relieving the overloaded right heart, by mercurial and hydragogue cathartics, diuretics, and diaphoretics.

2. *Pleurisy.*—In addition to keeping the patient quiet, restraining breathing, and forbidding conversation, the most effectual way of employing rest to the inflamed surfaces of the serous membrane is by mechanically fixing the side with adhesive plaster, as we should do for an inflamed joint. The forms of pleuritis to which this is most applicable are: Acute general pleurisy, seen early; dry pleurisy of a small area; that accompanying pneumonia, the result of a fractured rib; and in the advanced stages of phthisis pulmonalis with breaking down of lung-tissue, and where fits of coughing and pain are produced by stretching of the bands of organised lymph

which bind the costal and visceral layers together. The plan proposed by F. T. Roberts, and which has answered remarkably well in the hands of the writer, is as follows: Apply two or three layers of plaster, cut in strips of about four inches, thus: the first strip is laid on obliquely in the direction of the ribs, the second across the course of the ribs, the third in the direction of the first, the fourth as the second, and so on until the entire side is covered. A strip is also passed over the shoulder, which is kept down by another fixed round the side across its ends. Each strip should be long enough to extend from the spine to the sternum.

3. *Hæmoptysis.*—Rest is the most important of all measures in the treatment of hæmoptysis; rest of the body, the chest and lungs, and of the mind. The patient must be kept in bed, forbidden to use his voice, told to avoid coughing; and he should be given at once $\frac{1}{4}$ gr. or $\frac{1}{2}$ gr. of morphine hypodermically. All other drugs are quite secondary to this treatment by rest, and under it alone bleeding will cease. See HÆMOPTYSIS, p. 621; and PHTHISIS, p. 1228.

B. Diseases of the Heart and Blood-vessels.—

1. *Pericarditis.*—The mode of applying rest in this disease must necessarily be different from that which obtains in pleurisy, as actual arrest or even limitation, to any degree, of the heart's action—which theoretically and by analogy might be expected to be followed by the best results—would of course be out of the question. Rest must therefore be differently attained, by general rest and quiet, and by physiological medication. The advantages of perfect rest in the horizontal position are evident, as by it the action of the heart is rendered slower, and the attrition of the inflamed surfaces against each other is lessened by some 17,280 beats in the twenty-four hours, and thereby the tendency to effusion diminished, and resolution encouraged. The medicine above all others to produce physiological quiet is opium. When not otherwise contra-indicated, and when carefully watched, it is to be used freely, in grain-doses every second or third hour, as it is remarkably little liable to produce narcotism.

2. *Thoracic aneurysm.*—To Tufnell of Dublin is due the credit of having arranged, according to Valsalva's theory, a routine treatment based on the principle of securing the most perfect rest attainable. Tufnell's method may shortly be stated to be as follows: The patient is to be placed in a bright airy room on a prepared bed or couch, on which he must be contented to remain for eight or ten weeks. He must thus lie in the horizontal position, and not even for a moment assume the erect posture. Accordingly, the bed must be so constructed that the requirements of nature can be attended to without alteration of position. The diet is to be restricted to a minimum of solids and fluids. The patient's mind is to be freed from all anxiety, and pain and sleeplessness relieved by opium. The object of these means is to give rest to the aneurysm (1) by reducing the absolute quantity of blood circulating, without taking any of its ingredients from it by bleeding; (2) by rendering the blood hyperinotic; (3) by diminishing the rate and force of the current through the sac. The horizontal position in a healthy individual makes a difference of at least twelve cardiac beats a minute less than the erect position, and in aneurysm this difference amounts to twenty or even forty beats.

Taking it at the lowest rate of difference, it is evident that in the horizontal position the pulse-wave passes 17,280 times less through the body in the twenty-four hours. The aneurysmal sac is proportionately less often distended, and the threatened breach in the wall of the artery is averted by layers of fibrin being deposited by the more slowly moving and concentrated stream.

C. Diseases of the Abdominal Viscera.—In the therapeutic consideration of disease of these organs the principle of rest is not less plainly indicated than in the other parts of the body we have discussed; and by neglect of so simple and yet so potent an agent all other treatment may significantly fail to relieve or to cure.

1. In *acute inflammation of the stomach and in ulcer*, rest may be secured by strict diet, or in some cases by temporary total deprivation of food, enemas supplying the requisite nourishment. Local rest can best be obtained by the physiological action of opium upon the vermicular movements of the intestines, and by avoiding all irritants or purgatives. Opium by the mouth, or morphine hypodermically, may be required in full doses, so as to arrest all peristalsis; and thus an inflamed or ulcerated surface is placed at rest, and nature is enabled to prevent perforation, and cure the disease. It cannot be too strongly stated that the injudicious employment of purgatives in threatened perforation or peritonitis, is not only unscientific, but the worst possible practice, as it is almost sure to result in the death of the patient.

2. *Inflammation of the kidneys.*—As the skin and bowels may vicariously perform many of the excretory functions of the kidney, the first indication in *acute nephritis* is to relieve and rest that organ, by general rest in bed, local depletion, by calling rigorously upon the skin and intestines, and by withholding all nitrogenous foods and stimulating diuretics.

J. MAGEE FINNY.

RESUSCITATION (*re-*, again; and *suscito*, I arouse).—**DEFINITION.**—The recovery from suspended animation or apparent death. In these conditions, of course, all signs of circulation and respiration have apparently disappeared, but usually the failure of respiration has preceded that of the circulation, hence it is always advisable to attempt resuscitation, however hopeless the case may appear, provided unmistakable signs of death are not present. For the purposes of treatment we may classify as (A) *syncope* those cases where the lips and mucous membrane are found pale and exsanguine; and as (B) *asphyxia* those where cyanosis is marked.

A. Syncope.—Syncope may arise (1) from mental emotion, sudden pain, or shock; (2) from drugs and poisons, including anæsthetics, especially chloroform; (3) from hæmorrhage, or anything which reduces the due supply of blood to the heart; and (4) from fatty degeneration or dilatation of that organ.

TREATMENT.—Place the patient immediately on the floor, turn him on to his right side, with the pelvis and feet raised. Nélaton has urged complete inversion of the body. This is extremely useful, and can be readily performed with infants and children; but in the case of adults the whole of the weight of the intestines and omentum would be thrown on the diaphragm, and by greatly impeding its free action this method might be injurious. The

windows of the room should be opened; the face fanned; cold water dashed on the face and chest; and smelling salts held to the nostrils. If natural breathing has not returned, one or two compressions of the chest will in all probability re-establish it, but if not, Howard's method of artificial respiration should be commenced: *Position of patient.*—Face upwards; a hard roll of clothing beneath thorax, with shoulders slightly declining over it. Head and neck bent back to the utmost. Hands on top of head. Strip clothing from waist and neck. *Position of operator.*—Kneel astride patient's hips; place your hands upon his chest, so that the ball of each thumb and little finger rest upon the inner margin of the free border of the costal cartilages, the tip of each thumb near or upon the xiphoid cartilage, the fingers dipping into the corresponding intercostal spaces. Fix your elbows firmly, making them one with your hips. *Action of operator.*—Pressing upwards and inwards towards the diaphragm, use your knees as a pivot, and throw your weight slowly forwards for two or three seconds until your face almost touches that of your patient, ending with a sharp push which helps to jerk you back to your erect kneeling position. Rest three seconds; then repeat this movement as before, continuing it at the rate of seven to ten times a minute; taking the utmost care, on the occurrence of a natural gasp, to gently aid and deepen it into a longer breath, until natural breathing returns.

This method is said to keep the passage through the larynx free without the aid of an assistant or any contrivance for the purpose, and is recommended for that reason. Artificial respiration must precede the use of the stomach-pump, and be continued until either the pulse or natural respiration returns; but should the stomach-contents be evacuated, then the subject must be turned quickly on his side and the mouth cleared before continuing artificial respiration. Keep up the temperature of the body by hot blankets or hot bottles. Stimulating the heart by galvanism has been recommended, but it is a doubtful remedy. It is not easy to make it produce general and effective contraction, such as would cause the blood to move forward, and, failing to do this, it probably does harm by exhausting the irritability of those parts which it does excite. Ammonia, or nitrite of amyl, may be held to the nostrils. A little brandy and hot water, eau-de-Cologne and water, wine, or other stimulant, as sulphuric ether or sal volatile, is now to be given, with care that none of it enters the trachea. If swallowing is impracticable, inject warm fluids into the rectum. In cases of syncope from loss of blood, infusion of saline solution may be required. See SALINE SOLUTION.

B. Asphyxia.—(a) *Asphyxia neonatorum.*—The mouth and nostrils of the infant should be wiped dry, clearing away all mucus, amniotic fluid or meconium, and the body freely exposed, while the head is allowed to fall back over the hand which supports the nape. One or two compressions of the thorax are then generally sufficient to establish breathing. Failing relief, an effort should be made to arouse the reflexes by plunging the trunk into cold water and water at 100° alternately, a gasping inspiration in nearly all cases occurring during the cold immersion. These immersions may be continued till the child cries vigorously. In the event of a child being born

pale and flaccid, like a corpse, Schultze recommends the cord being immediately ligatured, the mouth cleared, and artificial respiration commenced. Any previous attempt to arouse reflexes he considers as valuable time lost. Sylvester's method, and the 'swinging method' of Schultze, are the best forms of artificial respiration. In mouth-to-mouth insufflation the risk of rupturing the lungs, dilating the stomach, and tubercular or other infection should not be overlooked. Marshall Hall's and Howard's methods may be used after the first inspiration has occurred, or together with mouth-to-mouth insufflation (see ARTIFICIAL RESPIRATION). Experiments made by Dr. Champneys show that Hall's and Howard's methods of artificial respiration are absolutely useless as a means of directly inflating the lungs of still-born children; and also that Sylvester's method, and its modification by Bain and Pacini, introduce more air than any other method.

(b) *Asphyxia from breathing noxious gases.*—The body should be brought into fresh air, and all clothing loosened; artificial respiration at once commenced, while an assistant should devote his attention to keeping the air-way free; and hot blankets and hot-water bottles applied. Venesection is useful if there be much venous engorgement, and inhalation of oxygen when obtainable. Efforts should be persisted in so long as any cardiac action can be detected.

(c) *Asphyxia from mechanical obstruction of the air-passages.*—The cause of obstruction must be removed, if possible by adopting the inverted position of Howard's method. Coins or plum-stones may thus dislodge themselves. In the absence of forceps, a button-hook or the handle of a table-spoon may be useful, especially in the removal of a lump of hard food. Laryngotomy or tracheotomy must be performed the instant the pulse becomes imperceptible at the wrist.

(d) *Asphyxia from poisons or anæsthetics.*—In the asphyxia of advancing coma from narcotics and anæsthetics, the breathing may stop from failure of the centres in the medulla presiding over circulation and respiration. In this case artificial respiration, by simply compressing the chest at intervals of five seconds, may suffice, but very often there is also the mechanical obstruction in the larynx to be considered. If drawing forward the tongue, raising the chin and throwing the head back do not effect a free passage of air, Howard's or some other method of artificial respiration should be commenced (see ARTIFICIAL RESPIRATION). It is well to understand that when the muscles of the larynx are paralysed, the glottis becomes valvular in action, or partially so—that is to say, it permits air to pass outward freely, but only a weak current of air to pass inward. A strong current brings the sides together, and gives rise to complete obstruction. This is chiefly caused by the drawing together of the relaxed arytaeno-epiglottidean folds of mucous membrane; and in order to obviate this kind of obstruction, the folds should be tightened, by throwing back the head and raising the chin as far as possible away from the sternum. This position frequently renders it unnecessary to catch hold of the tongue with artery forceps, the treatment usually recommended.

(e) *Asphyxia from drowning.*—In asphyxia from immersion in water there are two serious complications, namely, first, the presence of water and mud in

the air-passages, and, secondly, the depressing effect of cold. With the view of more effectually removing the water from the air-tubes, Howard gives the following rules: *Position of patient.*—Face downwards. A hard roll of clothing beneath the epigastrium, making that the highest point, the mouth the lowest. Forehead resting on forearm or wrist keeping mouth from ground. *Position and action of operator.*—Place left hand, well-spread, upon the base of the thorax to the left of the spine; the right hand upon the spine, a little below the left. Throw upon them, with a forward motion, all the weight and force the age and sex of the patient will justify, ending this pressure of two or three seconds by a sharp push, which helps you back again into the upright position. Repeat this two or three times, according to the duration of the immersion, and then resort to the method described in the treatment of syncope.

The following rules have been published by the Royal Humane Society. They recommend the Sylvester method, but probably this and the modification by Bain, in which the anterior fold of the axilla on both sides is grasped with the clavicle and pulled upwards, are less useful than the Howard plan, which favours the patency of the air-passages.

DIRECTIONS FOR RESTORING THE APPARENTLY DEAD:—

1. *If from Drowning or other Suffocation, or Narcotic Poisoning.*—Send immediately for medical assistance, blankets, and dry clothing, but proceed to treat the patient *instantly*.

The points to be aimed at are—first and immediately, the restoration of breathing; and secondly, after breathing is restored, the promotion of warmth and circulation.

The efforts to restore life must be persevered in until the arrival of medical assistance, or until the pulse and breathing have ceased for at least an hour.

Treatment to Restore Natural Breathing.

Rule 1.—*To adjust the patient's position.*—Place the patient on his back on a flat surface, inclined a little from the feet upwards; raise and support the head and shoulders on a small firm cushion or folded article of dress placed under the shoulder-blades. Remove all tight clothing about the neck and chest.

Rule 2.—*To maintain a free entrance of air into the windpipe.*—Cleanse the mouth and nostrils; open the mouth; draw forward the patient's tongue and keep it forward; an elastic band over the tongue and under the chin will answer this purpose.

Rule 3. *To imitate the movements of breathing.* Firstly, to induce inspiration, place yourself at the head of the patient, grasp his arms, raise them upwards by the sides of his head, stretch them steadily but gently upwards, for two seconds. By this means fresh air is drawn into the lungs by raising the ribs.

Secondly, to induce expiration, immediately turn down the patient's arms, and press them—or your own hands—gently against the sides of his chest, for two seconds. By this means foul air is expelled from the lungs by depressing the ribs.

Repeat these measures alternately, deliberately, and perseveringly, fifteen times in a minute, until a spontaneous effort to respire is perceived. When a spontaneous effort to respire is perceived cease to

mitate the movements of breathing, and proceed to induce circulation and warmth.

Rule 4.—*To excite inspiration.*—During the employment of the above method excite the nostrils with snuff or smelling-salts, or tickle the throat with a feather. Rub the chest and face briskly, and dash cold and hot water alternately on them. Friction of the limbs and body with dry flannel or cloths should be had recourse to. When there is proof of returning respiration the individual may be placed in a warm bath, the movements of the arms above described being continued until respiration is fully restored. Raise the body in twenty seconds to a sitting position, dash cold water against the chest and face, and pass ammonia under the nose. Should a galvanic apparatus be at hand, apply the sponges to the region of diaphragm and heart.

Treatment after Natural Breathing has been Restored.—*To induce circulation and warmth.*—Wrap the patient in dry blankets, and rub the limbs upwards energetically. Promote the warmth of the body by hot flannels, bottles, or bladders of hot water, heated bricks to the pit of the stomach, the armpits, and the soles of the feet. On the restoration of life, when the power of swallowing has returned, a teaspoonful of warm water, small quantities of wine, warm brandy and water, or coffee should be given. The patient should be kept in bed, and a disposition to sleep encouraged. During reaction large mustard poultices to the chest and below the shoulders will greatly relieve the distressed breathing. In all cases of prolonged immersion in cold water, when the breathing continues, a warm bath should be employed to restore the temperature.

2. **If from Intense Cold.**—Rub the body with snow, ice, or cold water. Restore warmth by slow degrees. It is dangerous to apply heat too early.

3. **If from Intoxication.**—Lay the individual on his side on a bed with his head raised. The patient should be induced to vomit. Stimulants should be avoided.

4. **If from Apoplexy or Sunstroke.**—Cold should be applied to the head, which should be kept raised; clothing removed from neck and chest; and stimulants avoided.

How soon should alcoholic stimulants be given? Certainly not until natural respiration has been induced; and, in cases of narcotic poisoning, not until consciousness has been restored. If, on the return of consciousness, the patient is in pain, or faint, the inhalation of a few drops of ether or smelling ammonia is indicated. In the absence of these a few teaspoonfuls of brandy may be given. Hot tea and coffee should be the first refreshment swallowed, and in general it should not be pressed upon the patient, as vomiting is more exhausting than waiting a few hours for food.

J. T. CLOVER.
CARTER BRAINE.

RETCHING (A.-S. *hræcan*).—An ineffectual effort at vomiting, sometimes accompanied by the expulsion of gas from the stomach. *See* VOMITING.

RETENTION OF URINE.—*See* MICTURITION, Disorders of.

RETINITIS.—Inflammation of the retina. *See* EYE, AND ITS APPENDAGES, Diseases of.

RETRACTED ABDOMEN.—The abdomen as a whole presents under certain circumstances more or less depression of its anterior wall, when it is said to be *retracted*, and this may reach such a degree that the region becomes 'boat-shaped,' and its anterior boundary sometimes seems almost to come into contact with the spinal column behind. The bony prominences of the crest and interior angles of the ilium, the pubes, Poupart's ligament, and the lower margin of the chest often stand out prominently. In some instances the retraction is partial, involving the lower part of the abdomen, while the upper part is distended.

A retracted abdomen frequently renders it more easy to investigate by physical examination the contents of this cavity; and it must be remembered that the condition may be associated with diseases of abdominal organs, which can then be readily detected, or even with abdominal tumours. It may, however, also itself give information of importance in diagnosis. The chief conditions under which a retracted abdomen may be met with, so as to be of clinical importance, are as follows: (1) In certain cases of disease of the brain or its membranes, and especially acute tubercular meningitis. (2) In some forms of intestinal colic, particularly that form associated with lead-poisoning—the so-called *painter's colic*. (3) As a part of marked general emaciation from any cause, but especially that due to starvation, or to chronic diarrhœa from intestinal ulceration and other conditions. (4) In connection with chronic diseases of the œsophagus, stomach, intestine, or pancreas, causing obstruction in some part of the alimentary canal, so that food cannot be taken in, or is prevented from passing along the tube. Here the retraction is also partly due to the general emaciation. (5) As one of the consequences of chronic peritonitis. It will be seen from a consideration of the causes just mentioned, that retraction of the abdomen immediately results either from a spasmodic contraction of the intestines and abdominal muscles; general wasting; absence of food from and contraction of the alimentary canal; or peritoneal adhesions. It may be mentioned that marked temporary retraction of the abdomen is sometimes noticed in connection with the act of breathing, in consequence of disordered action of the diaphragm. *See* DIAPHRAGM, Diseases of.

FREDERICK T. ROBERTS.

RETRACTED CHEST.—*See* DEFORMITIES OF THE CHEST.

RETROCEDENT (*retro*, back; and *cedo*, I go).—A term employed in connection with certain acute diseases, when their prominent external manifestations disappear or, as it were, go back. Retrocession is often associated with the simultaneous occurrence of internal disturbance. The term is often applied to phenomena observed in gout, rheumatism, certain skin-diseases, and the eruptive fevers.

RETROFLEXION (*retro*, back; and *flecto*, I bend).—A form of displacement in which an organ is bent backwards upon itself. *See* UTERUS, Diseases of.

RETRO-PHARYNGEAL ABSCESS.—*See* PHARYNX, Diseases of.

RETROVERSION (*retro*, back; and *verto*, I turn).—A form of displacement in which an organ is turned back but not bent. See UTERUS, Diseases of.

RE-VACCINATION.—The operation of repeated vaccination. See VACCINATION.

RHABDITIS NIELLYI.—An immature parasitic nematode, found in the skin in one or more instances of cutaneous disease, characterised by intense itching and the formation of papules and vesicles.

RHABDONEMA INTESTINALE (*βάβδος*, a rod; *vḗua*, a thread).—A genus of small parasitic nematodes found in the stools in certain cases of diarrhoea (p. 463).

RHEINFELDEN, in Switzerland. — See MINERAL WATERS.

RHEUMATIC FEVER.—A popular synonym for acute rheumatism. See RHEUMATISM, ACUTE.

RHEUMATIC GOUT.—A popular name for several kinds of chronic joint-disease, especially rheumatoid arthritis and chronic rheumatism.

RHEUMATISM, Acute (*ῥευμα*, a fluxion).—**SYNON.** : Rheumatic Fever; Fr. *Rhumatisme Articulaire Aigu*; Ger. *Acuter Gelenkrheumatismus*.

DEFINITION.—An acute disease; caused by certain infective, diathetic or climatic influences; and characterised by fever, sweats, acute shifting inflammatory and other nutritional changes in connection with the joints and related structures, and certain of the viscera, particularly the heart, as well as the serous membranes.

ÆTIOLOGY.—The *efficient* cause of acute rheumatism is unknown. Different micro-organisms have been described in connection with it, but an ætiological relation between any one of them and the disease is not established. Of the *predisposing circumstances* the most important is inheritance, which can be traced in 27 per cent. of all cases ('rheumatic diathesis'). The great majority of first attacks occur in persons under the age of thirty; and the larger proportion of these between the ages of sixteen and twenty-five. At the same time, rheumatism is by no means uncommon either in children or in persons past middle life. Rather more males than females suffer; but the influence of sex is inconsiderable apart from other circumstances. Indeed, when all the varieties of the disease are included, it is more common in girls than in boys, particularly between the ages of ten and fifteen. Occupation and social position are possible predisposing conditions. Laborious outdoor occupations in which persons are exposed to chills, poverty, and the many evils associated with these, are believed to contribute the largest percentage of cases.

The most common *determining circumstance* of acute rheumatism is exposure to cold and wet, or exposure to cold after severe exertion (see PERSONAL HEALTH, p. 1205). In London it is most frequent during the months of October and November. Rheumatism may suddenly make its appearance after a sprain or other injury to a joint, which may also determine the distribution of the disease in the articulations. Similarly, the order of invasion of the several joints is due in some instances to the amount of exercise to which they

have been respectively subjected. An attack of acute rheumatism is occasionally referred to derangement of digestion and of the functions of the liver, especially in subjects who have previously suffered. Indulgence in abundant, rich or indigestible food will certainly determine a relapse in persons convalescing from the disease, and may possibly induce an attack in the predisposed. Bodily exhaustion or depressing mental influences, and acute diseases of other kinds, such as scarlet fever, erysipelas and influenza, may excite rheumatism under similar circumstances. Exhaustion by lactation or by chronic uterine disease, tedious convalescence, the puerperal state and possibly simple despondency may also act in this way in different instances. Certain regions or districts, parts of districts, or even houses, appear to deserve the name of 'rheumatic,' from the number of residents who suffer from the disease, and from the probability that a person, otherwise predisposed to rheumatism, will be more likely to be attacked if he enter such an area.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances in acute articular rheumatism are negative on the whole, not so much in consequence of the absence of morbid changes in the affected parts, as from the slight degree to which these changes have advanced. On opening an affected joint, we find moderate hyperæmia, occasionally with ecchymosis, of the synovial membrane and fibrous tissues connected with the articulation. The synovial surfaces present a somewhat opaque, granular swollen appearance. A considerable amount of inflammatory effusion occupies the articular cavity. This is generally a thin, clear, alkaline, albuminous fluid; occasionally it is turbid, with flakes of fibrin and cell-products; rarely it is purulent. The cartilages connected with the joint probably share in the inflammatory changes, especially if the process be severe; and the associated soft parts, including the tendons and their sheaths, are very frequently hyperæmic and the seat of effusion. In the subcutaneous structures in young subjects there may be found, over the articular ends of the bones and connected with the fasciæ and with tendon-sheaths, small nodules of fibroid tissue of a translucent appearance. These present the microscopical characters of rapidly growing connective tissue.

The non-articular lesions are far more important. Of these the most frequent are inflammation of the cardiac valves, myocardium and pericardium, and congestion or inflammation of the lungs. Inflammation of the pleura is less commonly found; in rarer instances inflammation of the peritoneum, bronchi, larynx, meninges, testes and renal tubules. When pyrexia has been high, the solid viscera, including the heart, present granular degeneration and are prone to rapid decomposition; and in cases of hyperpyrexia the blood is fluid. The blood has frequently been subjected to chemical analysis, but without any positive result of a pathological kind. The reaction of the liquor sanguinis is alkaline, as in health. The fibrin is said to increase in amount to 1 per cent. instead of .25 per cent. (p. 159). The amount of urea is not above the normal (p. 160). Neither excess of uric acid, lactic acid nor any other abnormal principle has been found in the blood during an attack of acute rheumatism. The presence of micrococci, zoogloea-masses and bacilli in the blood and serum has been described, but not substantiated. The leucocytes are increased. The red

puscles rapidly diminish in numbers, as well as their hæmoglobin-value, during the acute stage, and are rapidly restored during convalescence.

SYMPTOMS.—After suffering for a time from aching pains in the limbs and trunk, flying pains and stiffness in the joints, malaise, chilliness and sore-throat, the subject of acute rheumatism is seized with severe pain in one or more of his joints, experiences a chill or slight rigor, and is found to have several degrees of fever. The local and general symptoms quickly develop; and a striking picture is presented by the patient. He lies motionless in bed, flat on his back, with every joint at rest and carefully guarded. The neck, back and legs are straight; the arms folded across the body, or extended along either side; the eyes are closed, and the patient is approached as he approaches the bedside. The face is found bedewed with perspiration; and the rest of the body is profusely covered with sweat, which gives off a strong acrid odour. The countenance is full, heavy, and expressive of a subdued feeling of pain and anxiety of movement; the complexion may be of a dirty sallow colour, or even slightly jaundiced; the cheeks are probably flushed. The affected joints are found to be swollen and red, hot to the touch, remarkably tender, and the seat of pain, which varies much in character and intensity. One joint, or several, or nearly every joint in the body, may be found in the condition just described. Peculiar firm nodules are observed to form in connection with the joints and other osseous and fibrous structures. The patient also complains of a feeling of illness, thirst and anorexia; the tongue is foul and creamy; the throat may be somewhat sore, and the bowels are irregular. The pulse is frequent, soft even to diastole, and rather large. Respiration is somewhat accelerated, and there may be slight cough. The urine is scanty, high-coloured, very acid, and loaded with lithates. The skin is covered with perspiration, congested and warm; and probably presents sudamina or miliaria in places. The patient's mind is perfectly clear, and his attention appears to be chiefly directed to the maintenance of the affected joints in the most easy position possible. Until successful in this endeavour he is distressed and miserable; and even if he have obtained temporary relief and have gone to sleep, he is liable to be suddenly aroused by involuntary spasms of the muscles connected with the affected joints. The pain is so severe when the disease is at its height that sleep cannot be obtained. Meanwhile symptoms and signs of invasion of the heart and pericardium, of the lungs and pleura, or of other of the viscera individually or in various combinations, often make their appearance, as well as erythema, chorea, or rarer rheumatic manifestations and a variety of complications.

For a period, which would appear to be perfectly indefinite, the patient continues in this condition, which varies considerably in intensity from day to day. But while it thus persists, the remarkable and characteristic fact is observed in this disease, that the arthritic phenomena are at once transient and erratic; that is, that the rheumatism passes rapidly from joint to joint, the joints which were affected the one day being nearly well the next, and a fresh series swollen and painful, while the non-articular phenomena may be equally evanescent and changeable. In this manner most of the

joints of the limbs may have been affected in the course of a week, and the number of joints simultaneously affected is very variable. Thereafter the disease may make a further invasion of joints previously involved, and that repeatedly; and the other symptoms and signs vary still.

At last the rheumatism appears to have exhausted itself: no fresh joint is attacked; and the parts last affected lose more or less completely the final traces, both objective and subjective, of the severe process which they have undergone. The patient now assumes a less constrained posture; the other symptoms decline; the perspirations disappear; the countenance becomes more bright; spirits and strength return; the tongue cleans, and the appetite is rapidly restored. If the heart or other of the viscera have escaped, the pulse falls in frequency; the urine is passed in greater quantity, is less acid, and no longer deposits urates; and the temperature falls to the normal. The joints remain for some time, however, stiff, weak, and painful on movement. Relapse is not uncommon at this stage, or a little later. Anæmia and debility are almost constant sequelæ, and in a considerable number of instances the heart is left permanently damaged.

Analysis of Clinical Phenomena.—*Invasion.*—In the great majority of cases the patient gradually 'sickens for' acute rheumatism for several days before the symptoms are fully declared. He feels ill and out of sorts, chilly, indisposed to eat or work; sleeps badly; complains of sore-throat, aching pains in the limbs, and shooting, shifting pains in the joints; and presents a sallow, patchy complexion, and a dull, heavy, yellowish appearance of the eyes. Altogether, the condition of the patient is very much that of the subject of a severe catarrh; the tongue, digestion, bowels, urine and pulse presenting the ordinary characters of moderate fever. On careful examination it is found that the pains are of two kinds. The first kind are by far the more severe, and consist of severe muscular aching in various parts of the limbs and trunk; while the second kind are of the nature of flying pains in the joints or associated parts. The muscular aching appears to be similar to or even identical with the 'break-bone' pains which are familiar in influenza, and in the invasion-stage of some eruptive fevers. They are, therefore, not characteristic. The flying pains, which are, however, not always present—especially in first attacks—are actually situated in the articulations, for instance, the ankles, knees or wrists, and are of the nature of sharp twinges, suddenly leaving one joint to return as quickly in another. Towards the end of the stage of invasion these pains become less 'shifting;' and when, as the patient will say, they have 'settled' in one or more joints, the rheumatism has passed into the second stage, that of the declared disease. Stiffness of the joints may also be present, especially in recurrent attacks.

In the invasion-period the skin does not yet present the perspiratory activity which is so characteristic a symptom of acute rheumatism; but rather a moistness, greasiness or oiliness, with heat and some congestion. The temperature is raised one degree or more. The sore-throat, which consists in pharyngeal catarrh, follicular tonsillitis or even actual acute suppurative inflammation, is remarkably characteristic. The milder forms are soon lost in the more urgent symptoms.

The duration of the stage of invasion of acute

rheumatism varies greatly, the flying pains in the joints 'settling' much more quickly in some cases than in others. In a small proportion of cases the disease is so rapidly developed that the stage of invasion is wanting. The patient on waking in the morning finds one or more joints affected; or he appears to be struck down during the day without the slightest warning; and instances are not unknown in which persons, thus suddenly seized with acute rheumatism, have been removed to hospital for supposed sprain or fracture of the limbs. In rarer instances the feverish symptoms of the invasion-stage may be well marked without any pains whatever.

Declared Disease.—The phenomena connected with the affected joints naturally vary much. The swelling is usually considerable, and is chiefly referable to effusion into the cavity of the articulation, fluctuation being frequently discoverable. It is rare for the periarthritic effusion to be so abundant as to yield pitting on pressure. Neighbouring tendons and tendon-sheaths may be swollen, as well as tender. The amount of intra-articular effusion (as well as the pain) greatly influences the position of the joint, but most joints are maintained in a position a few degrees removed from extension. Careful examination will determine the increase and the disappearance of the swelling, as the joints are attacked and recover respectively.

Pain is the most distressing of all the symptoms in uncomplicated rheumatism. It is always severe, and sometimes almost unbearable; but it varies with the different joints, and with the degree and duration of their involvement. It may be said to increase steadily for several hours; it remains excessive for a time; and it then slowly and steadily declines. Its character is very differently described by different sufferers. When a joint is attacked by rheumatism, the first sensation felt by the patient is one of soreness on movement. As the condition develops, the soreness increases to an ache of a subdued, throbbing character. In the course of a few hours the ache 'works up' into an intense pain, apparently associated with a feeling of cramp, the slightest movement of the articulation being almost unbearable. The severe pain now gradually declines—in some instances from the time the swelling reaches its height. After several hours the only pain that remains is a distressing sensation as if the parts had been severely bruised; and the effusion which accompanied the excessive pain having declined along with it, rest of the joint again becomes all-important, the very slightest movement being sufficient to restore the wearying ache. Finally, the pain completely disappears, and nothing remains beyond a feeling of stiffness and helplessness when the joint is moved.

While the course of the pain of an acute rheumatic attack is usually such as has been described, it is greatly modified by a variety of circumstances, such as the particular joint affected, the age and sex of the patient, the condition of the nervous system, and the presence of certain temperaments. In some instances it is increased at night.

Tenderness is a constant and well-marked symptom of acute rheumatism. Reference has already been made to the effect of movement on the pain in its different stages, especially towards the end; and to the characteristic posture and anxious expression of the patient, who suffers intensely from the slightest shake of the bed, or even a footfall on

the floor. Tenderness finally declines into the feeling of stiffness on movement.

The redness of a rheumatic joint is a simple pink blush of erythema, very rarely purpuric. Its intensity varies much with the superficial or deep situation of the articulation, and it is therefore most marked in connection with the joints of the hands and feet, the knees and the ankles.

Heat of the affected joint is a well-marked objective sign of acute rheumatism. The skin over the articulation feels decidedly warmer to the hand than the surrounding parts; and this observation is confirmed by the thermometer.

The favourite joints involved in acute rheumatism are the larger articulations, especially the knees, ankles, wrists, shoulders and elbows; the hip-joint less frequently than the others. The fingers come next in order of frequency; then the toes; while the remaining articulations are more rarely affected.

Corresponding rather closely with the frequency of attack is the favourite order of invasion; the ankles being most frequently the first to be involved, then the knees, and so on. In other instances it is observed that the disease passes along the joints of the lower limbs, including the hips, to those of the upper limbs; frequently its distribution is symmetrical bilaterally; while in some cases it is unilateral, the homologous joints of the upper and lower limb being simultaneously invaded. The smaller joints suffer, as a rule, towards the termination of the attack.

During the course of acute rheumatism in children and adolescents, more especially in girls, occasionally also in adults, crops of subcutaneous nodules are occasionally developed in connection with the joints, tendon-sheaths and bones. These nodules are said to be fibrous at first, and to contain diplococci (Poynton and Payne). As generally seen they are small fibroid bodies, varying in size from a pea to an almond, and are readily discovered by stretching the integuments over the affected joint, which is best done by gentle flexion. They then stand out as pale elevations on the prominent ridges and prominences of the articulation; firm, scarcely movable, rarely tender, rounded in outline or occasionally acuminate. Several nodules are usually present on a single joint; but as they appear in crops, and have different stages of development, decline and disappearance, their numbers and characters vary from day to day. The favourite joints affected are the elbows, knees, knuckles, wrists and ankles. In connection with the cranium the occipital region frequently presents a great crop of nodules. On the tendon-sheaths of the forearms and hands they are occasionally very distinct, and here they are movable. They have also been found on the vertebræ, clavicle, scapula, sternum, ribs, ilium and long bones. A fact of significance is the usual association of subcutaneous rheumatic nodules, if they are large and numerous, with severe endocarditis and pericarditis.

The muscular pains of the stage of invasion of acute rheumatism disappear in the declared disease, or are lost in the presence of more severe symptoms. They are replaced, however, by pains in the soft parts of the limbs related to the affected joints, especially the muscular insertions and fasciæ; and even the whole limb may ache, with much stiffness and a feeling of utter powerlessness. Painful twitchings are also common, especially during sleep; and when the acute pain has passed off,

marked muscular debility and possibly atrophy remain behind.

Acute rheumatism is attended by well-marked *pyrexia*, but this, like the disease as a whole, is variable in degree, course and duration. The sudden invasion of the several joints, their speedy relief, the alternation of extreme bodily distress with comparative comfort, and especially the variety of pyrexial diseases with which the rheumatism may be complicated, would hardly lead us to expect a typical temperature-curve. Nevertheless, in uncomplicated cases the fever follows a tolerably definite course. *Pyrexia* makes its appearance at invasion; it continues as long as the local symptoms reserve an acute or subacute character; and with them it declines and disappears. The *degree* of the *pyrexia*, in the great majority of cases, is in direct proportion to the severity of the joint-disease. Mild local symptoms—that is, moderate pain, short duration of symptoms in any given joint, and a small number of joints affected—are accompanied by moderate fever, ranging from 99° to 102° F. On the other hand, severe local symptoms—that is, severe pain, the full development of the several signs in the affected parts, and the simultaneous involvement of several joints—are attended by a temperature of 101° to 104° F. In another, but very small, class of cases the temperature, whatever it may have been previously, rises rapidly to an alarming height, so as to be entirely out of proportion to the joint-symptoms, which either continue as before, or even disappear. This condition of *hyperpyrexia* is regarded in the light of a complication, and as such will be presently described.

The *type* of the fever in uncomplicated cases is *emittent*, the thermometer rising 25° , 50° , or 100° F. in the evening. The primary elevation of temperature at the commencement of the disease is somewhat rapid; the decline or defervescence is decidedly more gradual, although it is generally irregular, being almost invariably broken by temporary rises, or interrupted by the supervention of some pyrexial complication. Brief recrudescences of fever, referable to excitement, exertion or neglect of the primary alimentary and eliminative functions, are also common. The occurrence of a true relapse is marked by a return of *pyrexia*, which probably presents the same general characters as before.

Profuse *acid sweats* constitute one of the characteristic phenomena of acute rheumatism. The brow is covered with drops which trickle down the face; and the whole body perspires profusely, and is bathed in an atmosphere of wet steam. Although usually universal, the sweats may sometimes be unequally distributed. It is doubtful whether any relation can be traced between the amount of perspiration and the hour of the day or night, the temperature or the pulse; perhaps it varies directly with the severity of the pain. The sweats continue throughout the whole attack, making their appearance at an early date, and disappearing gradually with the subsidence of the other symptoms. They do not intermit in the striking way of the sweats of the hectic or septic states, except towards the end of a severe protracted attack, when the patient is greatly debilitated; but at certain parts of the day the skin may be found to be perspiring less freely, or even to be perfectly dry. The sweat of acute rheumatism possesses a peculiar sour, acid odour; and this is so powerful, and pervades so thoroughly the neighbourhood of the patient when the blankets

are disturbed, that the diagnosis of the disease can frequently be made from it alone. Like the sweat in health, it is acid in reaction, rarely alkaline from decomposition. No other test can be readily applied to it clinically. The rheumatic patient may complain of the unpleasant, but never of the 'weakening' effect of the perspirations which is observed in hectic fever; on the contrary, he may describe them as bringing great relief to the bodily condition. In less acute cases the skin may present a shiny or greasy appearance, rather than actual perspiration. When the sweats are severe, *sudamina* make their appearance, especially about the trunk; and in some cases the skin is covered with a profuse eruption of *miliaria*.

In connection with the *digestive system*, the tongue is found to be covered with a thick, white, moist fur, which varies closely with the rheumatic condition, and serves as a ready evidence of the same. The thickness of the coating is sometimes very great. Occasionally the tongue is dry; very rarely brown, baked-looking or cracked. The sense of taste is, in a great measure, lost; thirst is urgent and difficult to satisfy; and the reaction of the saliva, or, more correctly, of the fluids of the mouth, is said to become acid. Appetite is lost, until the disease begins to decline, when hunger returns very early and urgently. Sore-throat occurs in some cases during the declared disease, but is much less common than in the stage of invasion. Sickness is rarely present. *Dyspepsia*, attended by flatulence, is common, unless the most digestible food only be given. Irregularity of the bowels is characteristic of acute rheumatism, either constipation or diarrhoea being almost constantly present; and the two conditions frequently alternate. Diarrhoea is perhaps more common in first than in subsequent attacks. The motions are dark and foul. Pains in the belly are by no means rare, and are frequently connected with diarrhoea, but they occur also in constipation; and at times they are accompanied by attacks of distressing flatulence.

The frequency of the *respirations* in simple articular rheumatism is somewhat increased; slight cough is occasionally present; and under these circumstances a few dry rhonchi may be heard over the chest. On the other hand, respiratory affections may be of a serious and even fatal character, and will be considered in their proper place.

Throughout an attack of acute rheumatism the *urine* is scanty, high-coloured and strongly acid; and it deposits a quantity of urates. Temporary albuminuria is not uncommon. Quantitatively examined, the urine is found to contain an actual excess of urea, and a considerable (but probably only a relative) excess of uric acid, sulphates and colouring-matter (*see SPECTROSCOPE IN MEDICINE*); the water is below the normal amount; and the chlorides are diminished, although less so than in pneumonia. Lactic acid has never been found in excess. Any marked departure from these characters of the urine, especially in the appearance of more than a passing trace of albumen, is to be considered as a complication of the rheumatism.

As a rule the *nervous system* is but little disordered in acute rheumatism. Consciousness and clearness of intellect are preserved throughout. Delirium is very uncommon; and when either delirium or stupor supervenes, it will generally be found that, apart from complications or the action

of impure salicylates, the temperature has risen to an excessive height. Very rarely the pyrexia remains moderate in these circumstances, and such cases have been described by the name of 'cerebral rheumatism.' There is generally great distress of mind in acute rheumatism; and, in other than first attacks, the previous experience of its severe and uncertain course and dangers, and the recollection of the pains and other sources of bodily discomfort, greatly affect the patient, and produce an amount of anxiety which is almost characteristic of the disease. Sleep is either impossible, or at best is constantly broken and unrefreshing, when the pain is severe.

The *expression* of the sufferer from this disease calls for brief notice. The debility or prostration, which forms an important element of fever from whatever cause, is present in acute rheumatism, but it is in great measure obscured by the expression referable to pain, and by the effort to preserve an easy position. Towards the end of an attack, when pain is subsiding and movement is comparatively easy, the patient and the practitioner begin to appreciate the degree to which the bodily weight and strength, and the richness of the blood, have been reduced. This loss is always great, and is sometimes extreme, varying, of course, with the severity and duration of the disease.

Cardiac Affections.—These are by far the most frequent and important visceral manifestations of rheumatism, being present in no fewer than 50 per cent. of all cases. The percentage of acute cardiac disease is, however, less than this—almost certainly about one-third, the remaining cases being chronic, or chronic and acute cardiac disease combined. These numbers refer only to structural disease of the heart, namely, simple or ulcerative endocarditis, myocarditis, pericarditis, and the effects of these, singly or in combination. But, besides inflammatory affections, there occur acute dilatation and functional disorders of the heart, characterised chiefly by palpitation, cardiac distress and the presence of various abnormal physical signs. According to some observers, the latter class are of as frequent occurrence as the former. It is probable that the evanescent cardiac murmurs by which they are recognised are produced either by acute dilatation of the ventricle, or by small vegetative growths on the valves which afterwards disappear. When the heart is sound the pulse is regular, 80 to 120, large, sometimes hard, more frequently soft or even dicrotic; but it naturally varies much with the severity and stage of the disease.

The *circumstances* under which cardiac involvement most frequently makes its appearance in acute rheumatism are—first, and specially, early age, rheumatic children rarely escaping disease of the heart, youths seldom, and the liability rapidly diminishing after the thirtieth year; secondly, severity of the rheumatic attack—with which the liability to cardiac affections increases directly—in the adult; thirdly, the female sex—women being more subject to rheumatic disease of the heart than men; and, fourthly, neglect of proper treatment during and after the attack.

The *time of appearance* of cardiac symptoms has been variously stated by different observers. As a matter of fact, they are generally discovered when the patient comes under observation; they certainly begin most frequently in the first week of illness; but they by no means uncommonly make

their appearance in the second week, and may occur at any period.

Inflammation of the heart and pericardium are fully described under their appropriate headings (*see* HEART, Inflammation of; and PERICARDIUM, Diseases of). The influence of the presence of these and other cardiac lesions on the course and prognosis of acute rheumatism is so important as to alter the whole aspect of the case, and to prove the chief cause of anxiety. Affections of the heart are by far the most common cause of death from rheumatism, immediate and remote; and even if they do not prove fatal, they constitute the most distressing of the remote effects of the disease. Rheumatic dilatation and inflammation of the heart in children, like the arthritis in these subjects, are often slight at first, and are therefore liable to be overlooked or disregarded; but they are processes which—partly, no doubt, in consequence of this circumstance—may lead to grave disease.

Respiratory Affections.—Diseases of the respiratory organs have been variously stated to occur in from one in every six to one in every sixteen cases of acute rheumatism; and in the larger proportion of immediately fatal cases they are the direct cause of death—frequently in association with endocarditis and pericarditis. The most common is pleuro-pneumonia; pleurisy alone the next; then pneumonia; severe bronchitis, pulmonary congestion and laryngitis are more rare. They may probably occur at any period of the rheumatic attack; but the gravest forms will necessarily appear towards the termination, for the obvious reason that they so frequently prove fatal. The supervention of acute respiratory diseases is, as a rule, easily recognised by the appearance of their several symptoms and signs.

Affections of Serous Cavities.—Rheumatic pleurisy and pericarditis have been referred to. Rheumatic peritonitis is a very rare but a real disease. Rheumatic 'orchitis,' or inflammation of the tunica vaginalis, is occasionally met with.

Tonsillitis.—This, in different forms and degrees, is a common rheumatic manifestation. It may accompany the arthritis; or precede it by hours, days, or weeks; or be associated with acute endo- or peri-carditis, which proceeds to chronic cardiac disease, while the joints escape.

Chorea.—Chorea bears an intimate relation to acute rheumatism (*see* CHOREA). Occasionally it appears in the course of an attack; and choreic twitchings may thus be the prominent symptoms during the first days of the illness, especially in children.

Cutaneous Affections.—In a small proportion of instances, acute rheumatism, or a condition which practically cannot be distinguished from it, is associated with erythema. Sometimes the arthritic symptoms are the first to appear, sometimes the skin-affection, and the two conditions, or the erythema alone, are further associated with some of the visceral affections already mentioned. Urticaria is less frequently seen in the same connections. A remarkable condition, in which arthritic symptoms are associated with purpura, hemorrhages, vascular thromboses, and possibly ulcerative endocarditis and its complications, is known as *peliosis rheumatica*, or *purpura rheumatica*, but does not appear to be true rheumatism. Sudamina and miliaria have been already noticed. *See* ERYTHEMA; PURPURA; and URTICARIA.

VARIETIES.—The description just given applies to a fully developed attack of acute rheumatism. It is only a minority of cases, however, that are of this nature. Occasionally the symptoms are very mild or the attack is very short, in which event the rheumatism is said to be *subacute*. In another variety or 'type' of the disease, which is common in children, the articular manifestations are insignificant or even 'latent,' but the cardiac, pleuritic and other manifestations are severely marked, and the rheumatic process is peculiarly protracted in the form of relapses and recurrences. These departures from the 'typical' course of acute rheumatism, as it is called, will now be considered.

Subacute Rheumatism.—Under the name of subacute rheumatism are comprised a variety of cases of the disease, which, while of comparatively little severity, exhibit the greatest possible differences in their other clinical characters. Several well-marked groups of these may be distinguished, and demand separate consideration.

The *first* group of subacute cases is one in which the duration of the disease is unusually short—probably from one to three days. The number of joints affected is very small; and the general symptoms appear to be arrested before attaining any considerable severity. In a *second* group of subacute cases, after exceedingly mild invasion-symptoms, a single joint only is attacked, with little pyrexia, while the skin presents a shiny or oily dampness rather than true perspiration. The rheumatism disappears in a few days; or it shortly relapses in the same or in some other joint. This form of subacute rheumatism may be difficult to diagnose from gout. Widely different from the foregoing is another and the most common variety of subacute rheumatism. Such are many of the *recurrent* cases of the disease, and of the instances of first attacks in old subjects. It may be stated broadly that the first attack, or first and second attacks, of rheumatism are more severe than subsequent ones; that the severity diminishes with each recurrence of the disease; and that persons attacked for the first time after middle life suffer less than younger subjects. In all these cases, the phenomena connected with the joints, and the general symptoms, including the pyrexia and the sweats, are mild in degree, although by no means of short duration. In recurrent cases the patients are frequently the subjects of chronic heart-disease, in whom exposure to some of the determining conditions of rheumatism has lighted up fresh endo- and peri-carditis, and therewith moderate fever and subacute rheumatism of the joints. The anxiety of the practitioner will be confined to the condition of the heart; but during the progress of the complaint, joint after joint may become painful, tender, slightly swollen and red.

Acute Rheumatism in Children.—In children and young adolescents acute rheumatism possesses clinical characters so different in many respects from those just described in adults, as to be readily overlooked or mistaken for other diseases, even by the medical attendant. First, the symptoms and signs connected with the joints are relatively mild and may be entirely latent, and the diagnosis is only made on the discovery of acute cardiac disease. The patient is slightly feverish, and may or may not complain of pain and tenderness in the limbs, and of sore throat; moderate swelling of the affected joints passes unheeded;

the pains are either entirely disregarded or referred to 'growing,' to influenza or to 'a cold'; and the sweats are much less profuse or entirely wanting. Secondly, acute rheumatism in these young subjects often commences elsewhere than in the joints—with sore throat, erythema, pleurisy, or pneumonia, or with chorea. In the next place, while the disease in children presents these mild characters, the proportion of cases in which it is accompanied by acute cardiac disease—myo-, endo-, and pericarditis—is comparatively large, a fact which greatly increases the necessity for an early diagnosis. This is accomplished by making it a rule of practice to examine the heart and lungs physically in every case of febrile disease in the child, and that not once only but repeatedly; and at the same time to note with particular attention the colour and expression of the child's face, the posture and the frequency and type of respiration. In addition to these and the discovery of the physical signs of acute inflammation or dilatation of the heart, important evidences of rheumatism in children are the signs of chronic valvular disease—the result of a previous attack, the existence of nodules, the family history, and the effect of salicylates. Lastly, acute rheumatism in children exhibits an exceptional liability to relapse on the slightest departure from the rules of strict treatment and general management in the way of excitement, movement, or premature indulgence in solid food. Relapse takes the same irregular forms as the primary attack: fresh cardiac or pericardial trouble, pleurisy with effusion—particularly on the left side, erythema, tonsillitis, crops of nodules, chorea; and thus cases may be protracted for many months on end, with the further unfortunate disposition to recur in some form or other during the next few years, which in these subjects deserve the name of the 'rheumatic age.'

COMPLICATIONS.—*Hyperpyrexia* is one of the most alarming complications of acute rheumatism, but happily one of the most rare. The condition is fully described in another article (see FEVER; and TEMPERATURE). It may occur at any period of the disease; generally when the symptoms are fully developed; but even during convalescence. The principal indications of the approach of hyperpyrexia, which it is of the last importance to recognise, are flushing of the face; brightness and restlessness of the eyes; an eager, excited expression and behaviour; disappearance of pain and swelling from the joints, and arrest of the perspirations; delirium; and increase of the general symptoms of fever. On the occurrence of any of these symptoms in an otherwise uncomplicated case of acute rheumatism, the temperature should be taken at once, and the observation repeated every half-hour. If the body-heat prove to be over 103° F., and to be still rising, measures must be immediately adopted to prevent the hyperpyrexia which is certainly threatening.

The reputed frequency of *nervous* complications has been greatly reduced since the discovery that the majority of the cases of so-called 'cerebral rheumatism' and 'rheumatic meningitis' are really instances of hyperpyrexia. These cases being excluded, the frequency of cerebral disturbance, in otherwise uncomplicated rheumatism, is not greater than in other pyrexial diseases. Cerebral embolism may occur from endocarditis; meningitis is very rarely observed; insanity has been described in connection with acute rheumatism (see MANIA).

Delirium tremens occasionally supervenes when there is a history of alcoholism. Other causes of delirium in acute rheumatism are pericarditis and the salicylates.

Albuminuria does not occur in more than $\frac{1}{2}$ or 1 per cent. of all cases of acute rheumatism; and the so-called 'rheumatic nephritis' has probably no real existence. The frequency of albuminuria is not greater than can be accounted for by the presence of pyrexia, by renal embolism, by the probable existence of chronic cardiac and renal disease, and by the possible association of scarlatina.

When acute rheumatism attacks a subject of the *gouty* diathesis, its symptoms may be considerably modified. The pain, swelling, and selection of particular joints have all a *gouty* character more or less; and while the disease is more amenable to treatment directed against the *gout*, it has possibly a greater tendency to lapse into a chronic affection of the smaller joints.

Scarlatina may be contracted in the course of acute rheumatism, or during convalescence. The occurrence of rheumatism as a complication or sequela of scarlatina—a much more frequent and important association—is discussed under SCARLET FEVER. The two diseases may mutually predispose to each other, by lowering the general health and increasing the liability to chill; but some authorities hold that the ordinary complications of scarlatina, as well as the arthritis, such as serous inflammations and nephritis, are essentially rheumatic.

COURSE, DURATION, TERMINATIONS AND SEQUELÆ.—The course of acute rheumatism is indefinite. The natural duration under expectant treatment is about three weeks. The average duration of *acute symptoms* under the same treatment has been estimated at nine days; it is rather less under certain other methods of treatment; and it is much prolonged by neglect. The entire duration of an attack is much greater than this, and necessarily less definite, namely, two to six or even ten weeks; and, speaking broadly, it increases with the age of the patient, up to middle age.

The patient being convalescent, a *relapse* of the disease by no means uncommonly occurs, after a few days or weeks. The fresh rheumatism may attain any degree, from a slight swelling, redness and pain of a single joint, to a combination of the various symptoms, as severe as the first, or possibly more so, associated with fresh visceral manifestations. Occasionally two or more relapses may occur.

Convalescence is generally protracted (before the health is perfectly restored); and it is very common to hear persons who have suffered from acute rheumatism give three or four months as the time they were 'ill.' Convalescence is accompanied by desquamation involving the hands and feet, and perhaps the body generally. It is usually marked by obstinate anæmia. In many cases stiffness, pain and weakness continue indefinitely in the joints and neighbouring muscles. Nodules may remain for months.

In children, as described, the disease sometimes assumes an obstinately recurrent type, further dilatation or inflammation of the heart, pleurisy, fresh nodules and possibly chorea making their appearance in various associations, and frequently leading to a fatal result.

The great majority of cases of acute rheumatism

ultimately end in recovery, the proportion of deaths as the immediate result of an attack being only about 4 per cent. On the other hand, a large number of persons suffer from remote effects of the disease, many of which are not only distressing but likely to lead to death. Of the immediately fatal cases, the larger proportion terminate in the first or second weeks, and are associated with, if not actually due to, acute disease of the respiratory organs. The fatal cases which present cardiac disease, especially acute pericarditis, are scarcely less numerous. Altogether it may be said that from a half to three-fourths of all cases of death during acute rheumatism are referable to acute cardiac and pulmonary disease, either separately or combined. It is doubtful whether any patient dies from excessive pain, pyrexia, sweating and consequent exhaustion. Hyperpyrexia is the most common cause of death next to pulmonary and cardiac affections. In a small number of cases, meningitis, acute alcoholism and other complications lead to a fatal termination.

The *remote consequences* of acute rheumatism are, on the whole, more serious than the immediate effects. In a few instances the disease leaves behind it a condition of joints which passes into 'chronic rheumatism' or 'rheumatoid arthritis.' A far more common effect is valvular disease of the heart, which is referable in the majority of instances to acute endocarditis occurring as a complication of rheumatism. It is impossible to estimate the number of cases of disease of the lungs, vessels, brain, kidneys and other organs, which, in their turn, are caused by such heart-disease. It is not probable that the vessels suffer directly from the effects of rheumatism. When, in addition to these effects, we consider the debility, emaciation and anæmia which it induces, the remote effects of pneumonia and pleurisy, of chorea, and of the other less common complications of rheumatism, as well as the liability to a return of the disease and its complications, it is difficult to exaggerate the seriousness of the ultimate results of this malady. See also TABES DORSALIS.

PATHOLOGY.—The pathology of acute rheumatism is still obscure, and in the present article it will be sufficient to enumerate the principal theories that have been advanced respecting its nature. It was suggested that *lactic acid* accumulates in the body, and that the symptoms are directly referable to the action of this poison upon the system (Prout, Todd, Richardson). Other authorities (Canstatt, Seitz) believed that chill of the peripheral parts of the body, especially of the skin and joints, causes disturbance of corresponding parts of the central nervous system, and that this gives rise to pain and vaso-motor (?) or trophic changes of the same peripheral parts, and to fever. A third view was a combination of the two preceding: that chill causes accumulation or retention of lactic acid; that this acts on the central nervous system; and that the disordered nervous centres react upon the joints and other parts (Senator). Fuller and Latham put the relation somewhat differently, maintaining that chill disturbs the nervous system; that this disturbs metabolism generally; and that lactic or uric acid or both are retained, and act as poisons. Bastian (see NEURITIS, MULTIPLE) has suggested that, as the result of chill, some poisonous product is formed within the system, which, according to individual proclivity or the nature of the products engendered,

excites acute articular affection, or multiple neuritis, or acute spinal paralysis. A number of years ago it was suggested by Heter that rheumatism originates with the entrance of micro-organisms into the system; that endocarditis results; and that the joint-symptoms are secondary to this, and embolic, as in pyæmia. Certainly the present trend of opinion is that the disease is due to the presence in the blood of a micro-organism or a variety of micro-organisms (Salisbury, Immermann). These may enter the body by the tonsils or other channel, whereupon their morbid activity may be confined to the lymphatic system, particularly the serous and synovial cavities (Mantle). Once admitted they act specifically on the blood and different viscera, as do the causes of other acute infectious diseases.

Without criticising these theories, we may conclude that, while the true pathology of acute rheumatism is still unsettled, the most promising directions from which we may expect light to be thrown upon it are, first, bacteriology; secondly, the actions of organic poisons, whether introduced from without, or produced within the body as the intermediate products of nutrition; and, thirdly, the intimate relation of the nervous system to the body-heat, to the skin and to nutrition.

The pathological relations of the visceral and serous affections and of the different complications of acute rheumatism are very various. The largest and by far the most important group, comprising cardiac inflammations, pneumonia, pleurisy, peritonitis, erythema nodosum, chorea and meningitis, can only be described as having an intimate *genetic relation* to rheumatism, that is, as being a real part of the disease. This relation is indicated in many ways, such as the frequency of their occurrence during an attack of articular rheumatism; the comparative infrequency of certain of them in any other connection; the manifest analogy that exists between the parts affected in some of them and the joints; the direct increase of their frequency with the intensity of the general rheumatic symptoms, that is, of the cause of the disease; the transient and migratory character which they may present, alternating as they sometimes do with each other and with the arthritis; their occasional occurrence before the joint-symptoms, or even without them, constituting acute rheumatism without arthritis; their appearance in the person of a blood-relation of a rheumatic subject; their amenability to anti-rheumatic treatment; and, lastly, their occurrence in the course of acute rheumatism as a part only of a manifestly general disease.

The complications of acute rheumatism in the more correct sense of the term include certain effects of it, such as albuminuria, hyperpyrexia and mental disorder; scarlatina, dysentery and profuse hæmorrhages, which are perhaps related to it *etiologicaly*; and lastly, bronchitis and delirium tremens, which are to be considered as merely *concomitant* or *intercurrent* conditions.

DIAGNOSIS.—Although acute rheumatism can generally be easily recognised, its diagnosis is sometimes a matter of the greatest difficulty.

In the stage of *invasion*, it is most readily confounded with the acute specific fevers, including influenza and catarrh, in which pyrexia and aching of the limbs are prominent symptoms, and which indeed at times are complicated with rheumatism. If sore-throat be comparatively well-marked, and the development of the joint-affection slow, the

practitioner may be led to diagnose tonsillitis or simple catarrh instead of rheumatism, and to make light of a complaint which is about to develop into a serious disease. In every doubtful case a certain number of facts should be kept clearly in view, namely, the history of the attack; the possible occurrence of previous attacks of rheumatism; the family history; the absence of symptoms characteristic of other diseases, such as eruptions or coryza; the development of pain or tenderness in a definite joint, and of acid sweats; and, most important of all, the discovery of the signs of inflammation of the heart.

In the second or *declared* stage, when one or more joints are involved, an entirely different group of diseases have to be diagnosed from rheumatism, namely, gout, 'rheumatoid arthritis,' gonorrhœal arthritis, pyæmia, glanders, and acute synovitis or arthritis of traumatic or diathetic origin. In doubtful cases the characteristic phenomena of rheumatism must be kept clearly in mind, especially the transient and erratic course of the arthritic symptoms, and the probable presence of cardiac complications.

Acute gout may generally be diagnosed by the sudden invasion at night of a single joint—probably the great toe, in a man of middle or advanced age; by the severity of the pain, which is relieved by the occurrence of a characteristic swelling of the part; by the history of previous attacks of the same description; by the insignificant amount of constitutional disturbance attending the arthritis; and by the strict localisation of tenderness, as compared with swelling, when the acme is passed. The discovery of uric acid in the serum will settle definitely the diagnosis of gout (p. 604).

Pyæmia is usually associated with an injury or pre-existing surgical disease which simplifies the diagnosis. Rigors are the rule, while they are the exception in rheumatism; the fever has a deeply remittent or intermittent character; the sweats are distinctly intermittent; the arthritis is neither transient nor migratory, but may advance to suppuration of the joints; and symptoms of blood-poisoning, splenic enlargement, phlebitis and extensive and multiple visceral disease shortly supervene. Still it is a fact, which cannot be insisted upon too strongly, that cases of pyæmia are frequently mistaken at first for acute rheumatism.

Gonorrhœal arthritis may be recognised by being persistent, whether one or more joints be involved; by the type and degree of the pyrexia, which is not controlled by salicylates; by the absence of cardiac complications as a rule; by the presence of conjunctivitis; and, most certainly of all, by the co-existence of a urethral discharge, containing the gonococcus. See GONORRHOËAL ARTHRITIS.

Chronic rheumatoid arthritis may be distinguished by the characteristic deformities of the joints, but first attacks are readily confounded with acute rheumatism.

For the diagnosis of glanders, see GLANDERS.

Ordinary *synovitis* is rarely multiple. It is persistent and not migratory, and has an appreciable cause, whether traumatic or diathetic. See JOINTS, Diseases of.

The numerous difficulties which beset the diagnosis of subacute rheumatism and of acute rheumatism in children, have already been sufficiently dwelt upon.

PROGNOSIS.—In a disease which runs so uncertain a course, and which may include such a variety

of dangers, the prognosis is necessarily most uncertain. The proportion of fatal cases, and of cardiac affections, and the average duration of an attack, can easily be stated; but in a given case there is at first no positive means of foretelling what course the disease will run in any one of these respects. The prognosis must be specially guarded in persons worn out by mental or physical overwork or anxiety; in young women of full flabby habit, with tendency to anæmia and disturbance of the uterus, stomach and circulation; and in women after delivery—all subjects in whom cardiac inflammation and failure, and pulmonary invasion, are to be apprehended. Ill-declared 'weak' symptoms connected with the joints, indicating that the bodily strength and power of resistance are low, are less favourable than well-pronounced 'honest' pains and a warm sweating skin, which generally point to a favourable termination as regards life. It must, however, be observed that the risk of visceral involvement in some degree increases with the severity of the local symptoms. The probability of cardiac invasion decidedly declines after the first week, but the possibility continues as long as fresh joints are being invaded. Rheumatism of the joints may be expected, on the whole, to run a mild course in children, but it must not be forgotten that the course of it may be protracted by the occurrence of relapses and recurrences; and that the danger of cardiac disease is very great. In old subjects it may be safely assumed that acute rheumatism will end favourably. The supervention of pneumonia or pleuro-pneumonia, especially in association with cardiac disease, or of hyperpyrexia, should cause anxiety, as immediately threatening life. The prognostic significance of nodules is important. Remote danger from acute rheumatism is chiefly to be estimated by the occurrence of heart-disease, and by the form that it takes; and the liability to recurrence with fresh cardiac lesions must never be forgotten.

TREATMENT.—In addition to the ordinary measures proper in every case of an acute febrile disease which will probably prove of some duration, we must especially secure for the rheumatic patient perfect quiet, extreme gentleness of every necessary movement, and the prevention or relief of the discomfort attendant on constant and profuse perspirations. Next to a good strong nurse, and perfect hygienic arrangements of the sick-room, a proper bed is of the utmost importance in the general management of the case. The bed must be firm, stand on a firm floor, be sufficiently narrow, and be placed in such a position as to be readily accessible from either side and allow the attendants to reach any part of the limbs or trunk of the patient without interfering with the position and comfort of the other parts. Further, the bed must be 'made' as a 'rheumatic bed'; that is, a pair of blankets must be placed between the sheets—the one over, the other under the patient, so as to absorb the profuse sweat, and diminish the risk of chill from dampness of the linen. The patient should be furnished with a long flannel bed-gown, made to fasten with tapes down the front and along the arms, so that the chest or any joint may be reached with the least possible disturbance. Arrangements must be made for collecting the urine and stools in bed; and the use of a urinal and a bed-pan, or a properly arranged towel for these purposes, is imperative. Equal care is necessary in the general management

of acute rheumatism in children, however mild the attack may appear to be.

The nursing arrangements being completed, the physician may turn his attention to the consideration of the medicinal and dietetic therapeutics of the case. Two indications have to be fulfilled, namely, first, the control of the morbid process, and secondly, the relief of local symptoms that may be urgent, and the removal of general distress.

Whatever specific remedies may be selected, the medicinal treatment of acute rheumatism should be commenced with a purge consisting of calomel or a mercurial and colocynth pill, followed by a saline.

The use of the *salicylates* and *salicin*, introduced by MacLagan in this country, constitutes the routine drug-treatment of acute rheumatism; and the results obtained from it are certainly more favourable than from any other method. Fifteen to twenty-five grains of sodium salicylate, twenty grains of salicylic acid, or fifteen grains of salicin, are given every one, two, three, or four hours, until the temperature falls to the normal, to establish rapidly the full therapeutical influence of the drug; after which the dose is very gradually reduced, so as to be simply sufficient to maintain the apyrexia and control the arthritis for several (seven to ten) days. Some authorities reduce the dose as soon as symptoms begin to yield. The salicylates are best given in watery solution, variously flavoured; salicylic acid in milk, or combined with solution of ammonium acetate; and salicin in wafers or in solution. Different practitioners prefer the different forms of the active substance; the sodium salicylate is most extensively used. The salicylates reduce the temperature to the normal in the course of twenty-four to seventy-two hours, relieve the pain and other arthritic symptoms, markedly improve the condition of the patient generally, and diminish the risk of cardiac affections. The effect is most marked in first attacks, and in the early stages of an attack.

Unfortunately there are several drawbacks to the use of the salicylates. The first of these is the fact that the rheumatism frequently returns as soon as their exhibition is stopped. On the second or third day after the disease has been checked, the symptoms may be again as severe as at first, and the risk of cardiac involvement again present. Thus it happens that, taking an average of a large number of recorded instances, the total duration of rheumatic symptoms is not less than nineteen days. Still, in a considerable proportion of cases no such relapse occurs, and the patients are completely relieved within forty-eight to sixty hours.

Secondly, while the salicyl-compounds have a considerable influence in preventing visceral affections, by rapidly controlling the specific morbid process, they yet frequently fail to do so; and further, they appear to have little or no effect in subduing such visceral affections as may have already commenced. But if they do not arrest the endocarditis, pericarditis or pleurisy, they will repeatedly dispel the arthritic symptoms that may recur again and again during the course of these internal manifestations of the rheumatism. The best rule to follow, therefore, is to restrain the arthritis and fever by means of sufficient but not excessive doses of salicylates, and to attend to the indications presented by any visceral inflammation that may arise. The salicylates will be resumed or increased, as the case may be, in the event of recrudescence, inas-

much as pyrexial rheumatism, however slight, always depresses the heart.

Thirdly, the salicyl-compounds—particularly if abused—occasionally produce toxic symptoms, such as deafness, tinnitus, delirium, great frequency and weakness of the pulse, flushing of the face, lividity, sickness and collapse; and the termination may be fatal. The depressant action of salicylates on the heart and circulation especially is dreaded by practitioners, who are sometimes too ready to give them up for alkalis and quinine on the appearance of cardiac manifestations. In obstinately recurring cases, also, the protracted use of salicylates is believed to aggravate instead of controlling the rheumatism, by depressing the vitality of the patient. Of the two toxic influences, however, the salicyl-compounds are, in the writer's opinion, far less dangerous than rheumatism; and failure of the heart may be prevented by combining them with digitalis, ammonia, strychnine, ether or alcohol. Further, it has been determined that some of the worst of the toxic effects of salicylates were caused by impurities in the acid prepared artificially from phenol, which are now removed in the process of manufacture. Sodium salicylate can be given by the rectum to children, or to adults when the stomach is intolerant of the drug.

Lastly, it must be confessed that in a very small number of instances the salicylates entirely fail to control the arthritis and fever. The practitioner ought not to conclude that this is the case until he has tried the effect of larger doses. Twenty-five or thirty grains of the sodium-salt will often answer perfectly after twenty grains have been given without obvious effect. Salicin or salol (phenol salicylate) may succeed under the same circumstances.

If the salicylates fail or disagree, recourse must be had to alkaline treatment or to some other method. Certain practitioners combine alkalis with the salicylates from the first; others prescribe alkalis in diminished doses, or equal parts of sodium salicylate and bicarbonate. Possibly anæmia may be more marked, and convalescence more slow, after treatment with salicylates.

Before the introduction of the salicyl-compounds *alkalis* were in general use in the treatment of acute rheumatism. The alkaline method consists in the internal administration of sufficiently large doses of certain alkaline salts, such as the carbonates, citrates, tartrates and acetates, to render the urine quickly alkaline; in maintaining this reaction as long as the rheumatic symptoms continue; and in gradually allowing a neutral or an acid reaction to return by diminishing the dose as the symptoms decline. To obtain the full effect of potassium upon the system, half a drachm of the bicarbonate in an ounce of water should be prescribed at once, either alone or with citric acid in the effervescing form; and the dose is to be repeated every four hours. An equal amount of potassium acetate may be added to each dose, if a still more rapid and powerful action of the alkalis be desired. The urine will probably become alkaline within twenty-four hours; and when once this effect has been obtained, it may very easily be kept up by continuing the alkalis at longer intervals, which may be further increased as the symptoms decline. The effect of the drugs upon the patient must be carefully watched, and the amount and frequency of the dose varied accordingly, or its administration stopped, if necessary. Finally, when the rheumatism is relieved,

quinine may be added to the alkaline mixture; and as convalescence advances, the potassium may be entirely withdrawn. Constipation occurring in the course of treatment may be relieved by combining tartarated soda with the bicarbonate, tartaric acid being used to cause effervescence; or, if more obstinate, with a calomel and colocynth pill. It is claimed for this method that, as the alkalis begin to exhibit their action on the system, the whole aspect of the case becomes more favourable, the general distress being alleviated, the temperature falling, and the local symptoms relieved; that these favourable effects continue to become more and more marked, until the rheumatic condition has disappeared; that the average duration of the attack is greatly shortened, not exceeding 6·75 days in the acute stage and 13·5 days before the disappearance of pain; and that the proportion of visceral affections is reduced as low as 2 per cent. only.

The alkaline influence upon the system may be further increased, in a very agreeable way, by supplying the ordinary effervescing potash or soda water as a drink, either alone, or combined with milk or with fresh lemon-juice. The patient may be encouraged to drink this in quantity, unless there be special indications to the contrary, such as cardiac distress. Should there be diarrhoea, lime-water may be substituted for the potash or soda water. Should alkalis persistently cause purgation, their administration must be discontinued.

A modification of the preceding plan has been highly recommended by Sir Alfred Garrod, and consists in the administration of quinine from the very first in combination with large doses of alkalis; as much as five grains of the alkaloid (thus in the form of a carbonate) being given every four hours.

The most obvious general remedies for the relief of distress are anodynes and antipyretics. So much benefit follows the use of *opium* in some cases, by relieving pain and diminishing nervous irritability, that it has acquired a reputation even as a specific. Although formerly given in large and frequent doses, such as a grain every three to eight hours, either alone or combined with mercury, *opium* is now seldom employed in acute rheumatism, except in the form of a moderate dose of Dover's powder, or of a morphine draught at night, to relieve pain and induce sleep. The effect of the opium must be carefully watched, in the presence of the complications which may possibly arise and contra-indicate its use.

Many other antipyretic remedies besides the salicylates have been recommended, such as phenazone, acetanilide and phenacetin; quinine, tartar emetic, veratrum, digitalis, aconite, mercury and various diaphoretics. The use of quinine has been already referred to. At the present time reliance is chiefly placed upon two powerful remedies of this class, namely, the cold bath and the wet pack.

The cold bath is the most powerful and speedy method of reducing the temperature in acute rheumatism, but is seldom resorted to except in cases of hyperpyrexia. When this condition threatens, the cold bath (p. 136) or the wet pack (p. 699) is to be unhesitatingly employed, in the manner described elsewhere (*see* FEVER; FEBRIFUGES; and TEMPERATURE). If the symptoms be less urgent, cold sponging of the trunk may be sufficient to reduce the temperature.

Hæmatinic remedies may be considered to be indicated by the great anæmia which accompanies and yet more markedly follows an attack of acute rheumatism. Russell Reynoldson recommended iron perchloride in large doses; and favourable results have attended the administration of it in the acute phase. During convalescence iron is invaluable.

Empirical remedies.—Lemon-juice appears to have proved successful in some cases of acute rheumatism in doses of eight ounces or less in twenty-four hours. Other drugs include propylamine and trimethylamine, in four- to eight-minim doses every two hours; potassium nitrate to the amount of an ounce in the twenty-four hours; cynara or artichoke; and the cyanides of potassium and zinc. Colchicum may be given with advantage for rheumatism in a gouty subject, to relieve pain. Guaiacum is useful in sub-acute lingering cases. Potassium bromide has also been found very useful in American practice, probably by relieving pain and restlessness.

Reference must here be made to the observations of Gull and Sutton upon the course of acute rheumatism when treated by simple rest and the exhibition of a *placebo*. The comfort of the patient is secured by ordinary means; and small doses of opium are given to complete this effect when indicated. The results were favourable, but less so than those of several other methods of treatment, nine days being the average duration of acute symptoms. The frequency of cardiac invasion was also low.

For an account of the treatment of the visceral affections and the complications of acute rheumatism, the reader is referred to the respective articles on each of these in other parts of this work (see HEART, Inflammation of; LUNGS, Inflammation of; PERICARDIUM, Diseases of; HYDROTHERAPEUTICS (p. 700); &c.). The plan of treatment which is being pursued for the rheumatism may have to be temporarily suspended or considerably modified on the appearance of any of these grave conditions, but the salicylates ought not to be stopped without good reason, as already described.

The state of the bowels requires the most careful attention. Constipation must be relieved by any of the ordinary means. Diarrhœa may be checked with lime-water or bismuth, or with a judicious dose of castor-oil, according to its cause. The surface of the body should be regularly sponged with a very weak tepid solution of an alkaline carbonate.

The most ready and satisfactory measure for the *relief of local distress* is the application of absorbent cotton-wool to the rheumatic joints. This is to be wrapped in some quantity around the parts, and secured with a moderately firm roller, or a piece of warm flannel with the ends stitched together. The affected articulation is thus at once kept at rest and protected from cold and pressure; while uniform support is obtained. The relief afforded by this simple arrangement is often remarkable. The joints should be carefully sponged with warm water and soap, or warm water slightly alkalisied with sodium carbonate, before this or any other application; and the cotton-wool must be occasionally changed, especially if perspiration be profuse.

When the pain is still more severe, and relief is not to be obtained by simple rest and protection, anodynes may be applied to the rheumatic joints.

Opium in any of its ordinary preparations, belladonna and its allies, and other familiar anodynes, may severally answer best in particular instances. These substances may be applied on the surface of lint, secured and supported by a bandage; or the affected part may be lightly rubbed or smeared with the anodyne preparation in the form of liniment, and then wrapped in cotton-wool or flannel, as already described. Heat is generally grateful to acutely rheumatic joints, but in many cases it is felt to be useless in the acute stage unless it be quite extreme. Thus simple warm fomentations give relief; but the patient may urgently demand their constant renewal, so that they may be almost scalding. There are obvious objections to this treatment. Extreme cold has been recommended by some authorities, notably Professors Esmarch and Hueter, in the form of ice. It is seldom used in this country. A method of treatment once much in favour with some physicians, consisting in the application of blisters to the rheumatic joints, has been abandoned.

Certain lotions, of other than direct anodyne properties, have been recommended as local applications, to produce a specific effect upon the rheumatic joints, and thus indirectly afford relief. The chief of these are alkaline solutions, especially solutions of the carbonates of potassium and sodium, saturating flannel wrapped loosely around the joints. They may be combined with preparations of opium. Their value is somewhat doubtful.

The proper *diet* in acute rheumatism is the same as that in most other kinds of fever. The patient must be fed at short and regular intervals, night and day, with the most digestible forms of liquid nourishment; and may be encouraged to drink milk, or milk and soda-water, occasionally. It must not be forgotten, however, that in all probability the system is already overloaded with the products of imperfect assimilation and transformation; that the digestive system is weak and irritable; and that the heart may be seriously affected by flatulence, and by the addition of much fluid or solid material to the blood. As the acute symptoms decline and appetite returns, fish, milk-puddings, and shortly afterwards chicken, sweetbread, and other 'light' articles of diet, may be cautiously allowed, and will be greatly relished. But, as a rule, the appetite returns before it can be safely indulged without risk of a relapse. Meat must be strictly forbidden until every rheumatic symptom has disappeared; and it is a correct therapeutic rule not to give solid food and salicylates together. Thirst is best relieved in the acute stage, as already stated, with aerated alkaline waters, either alone or in combination with lemon-juice or milk, the quantity given being regulated by the practitioner and accurately recorded.

Alcoholic stimulants, in moderate doses, are indicated when the symptoms are severe and protracted. Besides this routine use of alcohol, a special virtue was claimed for its free administration, as in other fevers, by Todd and his disciples, in preserving the strength and relieving the pain. Brandy must be ordered on general principles in cases attended with extreme depression, or with serious visceral involvement, especially in the event of cardiac failure.

The general management of a case of rheumatism after the decline of the acute symptoms is scarcely less important than at the commencement of the

attack. The patient should be encouraged to keep his bed for several days after the disappearance of the joint-symptoms. This advice becomes imperative when the heart is involved. Rest and comfort of body and mind must be secured at this period, for the purpose of quieting the action of the heart, and allowing cardiac weakness and any endocarditic process that may affect the valves, and that probably outlasts the articular process, to subside quietly. The appetite must still be controlled, and daily evacuation of the bowels carefully attended to. All attempts at completing the cure of acute rheumatism within a certain small number of days must therefore be avoided. Even with these precautions, the first day of sitting up generally proves an anxious time to the practitioner in cardiac cases. Locomotion must be forbidden for several days, and permitted very gradually. When the patient is able to move about and go into the open air, the danger of a relapse during the first weeks must be carefully kept in mind. Sudden and extreme changes of temperature are especially to be avoided; and for this purpose the patient must be warmly clad, and studiously avoid draughts and exposure to cold in other forms. Hydrotherapeutic treatment is often successful in protracted cases, particularly if the articular symptoms are more prominent than the febrile. Careful rubbing or exercises, or in other cases a course of baths and massage at Droitwich, Bath or Buxton, may give excellent results independently of medicine.

Tonics are indicated during convalescence; for example, quinine and iron, separately or combined either with alkalis or acids, and strychnine. Tonics must not be commenced too early.

In the obstinately recurrent type of the disease occasionally met with in *children and adolescents* our therapeutical resources and patience are severely taxed. In the majority of instances the heart is seriously involved, and demands chief consideration; but attempts to treat the cardiac disease are usually vain unless the associated rheumatism, *however slight*, be directly combated. The most rigid and continuous attention to diet, elimination and rest, on the principles already laid down, is an indispensable element of the treatment—or, more correctly, the management—of rheumatism in this phase. As often as pain and pyrexia reappear, salicylates must be had recourse to if they can be borne, and iron and other tonics very cautiously resumed when the temperature has been reduced.

In persons who have suffered from acute rheumatism means ought to be adopted to prevent the return of the disease at a future time. The unfavourable influences to be chiefly avoided are cold and damp localities, exposure to wet, chill after exertion, free eating and drinking, and neglect of the liver and bowels. Woollen garments ought to be worn, summer as well as winter, in this country, of different thickness according to season and weather. These precautions are particularly important in the instance of rheumatic children. When the heart has been damaged in young subjects, so much attention is paid to the cardiac condition, with its attendant anæmia and impairment of nutrition, that the practitioner as well as the parents forget that one of the greatest dangers to which the child is exposed is return of rheumatism and fresh endocarditis.

J. MITCHELL BRUCE.

RHEUMATISM, Chronic.—SYNON.: Fr. *Rhumatisme Articulaire Chronique*; Ger. *Chronischer Rheumatismus*.

DEFINITION.—A disease of the joints, of chronic course; referable to certain obscure influences, possibly of a diathetic, climatic, or infective nature; and characterised by various degrees of inflammatory and degenerative changes in the articular and associated structures.

ÆTIOLOGY.—The causes of chronic rheumatism, as far as they are known, are the same as those of the acute disease. The most powerful predisposing circumstances are inheritance, previous attacks of acute rheumatism, poverty, physical and mental exhaustion, and laborious occupations entailing exposure to chills. For the last reason men are more liable to the disease than women, and certain joints are its favourite seats. Chronic rheumatism is most common in middle life or advanced age, although by no means rare in young adults, and occasionally met with in children. Exacerbations of the symptoms are usually referable to exposure, and are accordingly most frequent and protracted in cold, wet weather. Occasionally they appear to follow influenza.

ANATOMICAL CHARACTERS.—A variety of anatomical changes may be met with in chronic rheumatism, while in the least severe form of the disease no definite lesions of the articular structures can be discovered. In one form recurrent hyperæmia and effusion are found in connection with the synovial structures, and with the articular and peri-articular tissues generally. In the most severe cases the joints are enlarged and deformed, in consequence of anatomical changes which appear to be identical with those of rheumatoid arthritis (*see RHEUMATOID ARTHRITIS*). The heart is often the seat of chronic valvular disease and enlargement.

SYMPTOMS.—The clinical characters of chronic rheumatism vary extremely in different instances. The leading symptoms of the disease are chiefly two, namely, pain and stiffness in connection with the joints and associated structures, recurring indefinitely for any length of time, aggravated by cold, wet weather, and decidedly increased at night. More carefully investigated, the pains are found to have their seat in the joints, in the tissues of the limbs between the joints, or in both of these situations. Any or all of the articulations may be affected, but the site differs considerably in the different classes of the disease to be presently described. The pain is of a severe aching, wearying character, attended by a sense of heaviness and uselessness of the limb; it is relieved by rubbing, and by exposure to a cold atmosphere; and is increased by slight warmth. Free use of the joint, although at first attended by pain, often affords temporary relief; while, on the other hand, undue exercise of the limbs during the day is liable to be followed by severe aching in the night. The affected joints also feel markedly dry and stiff, and creak on movement; but exercise or rubbing may remove these sensations. These symptoms may last indefinitely for years, either recurring at intervals, especially in the winter and spring seasons, or being persistent almost day and night without intermission.

In the mildest degree the pain and stiffness just described are the only articular symptoms present. No anatomical change is apparent in either the joints or associated parts. The subjects of this

form of the disease may be otherwise well, vigorous and long-lived, in spite of the severe pains by which their rest is broken in cold, wet weather. They may or may not have, or have had, acute rheumatism.

In a more severe form of chronic rheumatism the pain is associated with obvious anatomical changes; and the disease takes the form either of a sequel of acute rheumatism or of recurrent subacute rheumatism, making its appearance at intervals for years. The articular phenomena consist of pain, redness, tenderness and swelling, the hands being a favourite seat of the affection. These attacks last indefinitely, shift from joint to joint, and leave behind them a distinct amount of swelling, which may not have completely disappeared before another invasion.

Thus chronic rheumatism of the most marked degree ultimately leads to enlargement and deformity of the affected articulations, the disease gradually invading the joints both large and small, until the whole articular system may be involved. After some years the subacute attacks follow so closely upon each other, and their local effects are so severe, that the patient is never free from distressing pain; the joints become ankylosed, dislocated, and otherwise disorganised; and the associated muscles undergo atrophy. At the same time the general nutrition is gradually impaired; and the sufferer is anæmic, wasted and debilitated. Chronic rheumatism of the most severe degree thus merges into the class of disease known as 'rheumatoid' arthritis, if it be not actually identical with it. See RHEUMATOID ARTHRITIS.

In other subjects the joints enlarge slowly without pain.

COURSE AND TERMINATIONS.—The disease, as its name implies, is essentially chronic, generally lasting throughout the life of the individual whom it attacks, and leading to various conditions of debility and deformity, according to the degree of its intensity. In many instances the patient is rendered unfit for work; and such cases form a considerable proportion of the inmates of Poor-Law infirmaries and other charitable institutions. Death as a direct result of the disease is rare.

COMPLICATIONS.—Myalgia and neuralgia, referable to rheumatism, are frequently associated with the arthritic trouble. Cardiac disease is met with in a considerable number of cases belonging to the more severe degrees of chronic rheumatism, being usually referable to endocarditis, which complicated the original acute attack. Dyspepsia is not infrequently seen in the subjects of the less severe forms. Iritis may be a very troublesome complication.

PATHOLOGY.—This disease is truly rheumatic in its nature, being intimately associated with acute rheumatism. In many of the best marked instances the patient has previously suffered from the acute disease, either immediately before or more remotely; in other instances one or more acute attacks occur in the course of the chronic disease. In another group of cases, a single member of a family will suffer from chronic rheumatism, ending in deformity, while his children, or his brothers and sisters, are attacked by the acute disease. The predisposing and exciting circumstances are also the same in acute and chronic rheumatism. Indeed, the two forms of affection run into each other in every particular, and are inseparably associated.

DIAGNOSIS.—The disease most often confounded with chronic rheumatism is chronic gout, particu-

larly that form of the disease characterised by pains in the limbs only, or that in which the joints—particularly those of the fingers—become enlarged and deformed with the development of articular nodes (*nodus digitorum*) and without tophi. Chronic arthritis may be regarded as gouty when the family history points in that direction; when the patient's history includes the occurrence of megrim, eczema, asthma, fits of depression and insomnia, glycosuria, lithæmia, and a disposition to obesity; and when any cardiac lesion that may be present belongs to the degenerative or senile instead of the inflammatory or juvenile type. It must be observed, however, that the two diseases are occasionally associated; and that then only is the name 'rheumatic gout' (so constantly misapplied) employed properly.

If chronic rheumatism be regarded as a distinct disease from 'chronic rheumatoid arthritis,' it is only in its most severe form that it can be confounded with the latter. A definite history of acute rheumatism; the presence of cardiac disease; and the non-involvement of such articulations as the jaw, the sterno-clavicular joint, and the spine, are considered to be features which render probable the diagnosis of true chronic rheumatism from rheumatoid arthritis.

The pain, swelling, heaviness, weariness and weakness associated with varix of the lower extremities, sometimes resemble closely the symptoms of the milder forms of chronic rheumatism. Physical examination at once removes all doubt.

PROGNOSIS.—The prognosis of chronic rheumatism is favourable as regards life, but very unfavourable as regards recovery. Patients rarely lose the tendency to recurrence of pain throughout the whole of their life. Within a short time of the commencement of the disease it will be easy to discover which of the principal forms it is likely to assume; and the prognosis may be made accordingly.

TREATMENT.—The leading indications for treatment in chronic rheumatism are, first, to control the pathological process; and second, to relieve the distress which characterises it.

The best possible measure for treating the *pathological process and the constitutional condition* with which chronic rheumatism is associated is removal of the patient to a warm and settled climate in some sub-tropical or tropical country. For persons of more moderate means we have recourse to warmth of clothing and housing and relief from muscular exertion, as far as they can be secured; for the very poor these advantages are secured by admission permanently into charitable institutions. The subjects of chronic rheumatism require a nutritious, heat-producing, and at the same time attractive and digestible diet, with a small allowance of alcohol at meal-times in the form either of spirits and water or of a pure red wine. The action of the liver and bowels must be carefully attended to. Iron, cod-liver oil and arsenic are the drugs best suited to improve the health; iodides and sulphur sometimes prove useful. Occasional large doses of salicylate of sodium will control more urgent symptoms. If acute symptoms, particularly pyrexia, supervene, liquid nourishment should be given. Courses of the mineral waters of Bath, Buxton and Strathpeffer in this country; and of many foreign baths, such as Aix-les-Bains, Royat, Aix-la-Chapelle, Wiesbaden, Baden-Baden and Hammam R'Hira, prove inval-

able in many cases to those who can afford to try them. As a rule, massage forms part of the treatment at these places; and passive movement of the joints, slowly but steadily increased in degree day by day, can be practised readily at home with much hope of benefit. Exercise should be taken daily in the open air, unless the weather be unfavourable; and active movement of the other joints besides those of the lower extremities should be maintained in spite of temporary pain. See RHEUMATOID ARTHRITIS.

Directions for *palliative treatment* are always desired by the patient. This chiefly consists in counter-irritation by iodine or cantharides; the application of anodynes, such as preparations of opium, belladonna and chloroform; or friction with various stimulating liniments, containing camphor, soap, turpentine or acetic acid. Regular warm ointmentations night and morning with very warm or even hot water, followed by rubbing and the application of a stimulating liniment under warm rollers, is one of the most efficacious methods of local treatment, the pains being prevented or relieved, and the stiffness removed often to a remarkable degree. The ordinary Turkish bath may also afford temporary relief, if properly employed (p. 137). Altogether, whatever view may be taken of the pathology of the disease, thorough local treatment of the joints and limbs will generally be attended with decided relief.

J. MITCHELL BRUCE.

RHEUMATISM, Muscular.—SYNON.: Fr. *Rhumatisme Musculaire*; Ger. *Muskelrheumatismus*.

DEFINITION.—A disorder of fibro-muscular structures, characterised by local pain and spasm, and a certain degree of fever; and generally associated with the rheumatic diathesis.

ÆTIOLOGY.—Muscular rheumatism is most frequently observed in the subjects of the rheumatic diathesis. It occurs in both sexes, and at all ages; children and adolescents being specially liable to rheumatic torticollis, and older subjects to lumbago and chronic muscular rheumatism of the limbs. The exciting circumstances are chiefly two: first, exposure to cold—above all, exposure of a muscular part to a 'draught' after exertion; and second, sprain or strain of the fibro-muscular structures. A peculiarly obstinate type of the disease has been observed as a sequela of influenza. Muscular pain, tenderness, and spasms are also common in the invasion of acute rheumatism, in chronic articular rheumatism, in chronic gout, and in gonorrhoeal arthritis.

ANATOMICAL CHARACTERS.—Nothing is known respecting the anatomical characters of muscular rheumatism, if, indeed, there be any discoverable change in the muscular or fibrous structures.

SYMPTOMS.—The disorder usually commences with slight febrile disturbance, possibly accompanied by sore-throat. Either simultaneously—possibly, indeed, quite suddenly—or in children after one or two days, pain is experienced in the region of some definite muscle or muscular mass, such as the sterno-mastoid or the muscles of the joints; and this speedily becomes so severe as to constitute the leading symptom of the attack. The pain is present only when the affected muscle is thrown into action, so that it may be perfectly relieved by relaxation or rest of the parts involved.

The slightest movement, however, from the position of relief is instantly attended by excruciating pain, of a peculiar spasmodic character, which persists until relaxation is again secured. The constant effort to avoid pain gives rise to a feeling and appearance of stiffness, causing the patient to assume characteristic attitudes of the head, trunk or limbs. Tenderness on grasping the muscle is occasionally well-marked. In some cases several of the joints may also be affected with pain and stiffness.

The constitutional symptoms of muscular rheumatism are generally those of mild fever. The tongue is furred, the appetite is impaired, the bowels are confined, the pulse is somewhat frequent, large and soft, and there is a feeling of malaise. Cases certainly also occur where the fever both in its type and in its severity resembles that of ordinary acute rheumatism. In other instances these symptoms are extremely slight or altogether wanting, the patient suffering from nothing more than local pain. Occasionally there is catarrhal ophthalmia. Subacute articular rheumatism and sciatica may be present as complications.

Muscular rheumatism usually persists for several days, and gradually declines, but in the more severe cases or in the milder cases it may last for weeks. One form of the disorder is essentially chronic, the patient suffering for years from pain in various fibro-muscular structures, especially those of the shoulder, arm, thigh and leg, during cold wet weather.

VARIETIES.—The following local varieties of muscular rheumatism are recognised by special names:—

1. **Muscular Torticollis.**—SYNON.: Acute Wry-neck; Stiff-neck; *Caput Obstipum*.—Here the sterno-mastoid muscle is chiefly involved, but any or all of the cervical muscles may be painful. This form is most frequently observed in young subjects, and is often markedly recurrent. It is easily recognised by the fixed position of the head; and has to be diagnosed from spasmodic torticollis, sterno-mastoid tumour, sprain, tonsillitis and spinal disease.

2. **Pleurodynia.**—The fibro-muscular structures of the chest-wall are the seat of rheumatism in this variety. Cough is a common exciting condition of the complaint, which is seen chiefly in adults. Pain is complained of in the chest-wall, usually on one side; in some instances it may be excruciating, and of a distressing, spasmodic character. On examination, we find that a particular intercostal space, or the origin of the pectoral or serratus muscle, is the seat of localised tenderness; and that every respiratory act causes lancinating pain in the same situation. The respiratory movements of the affected side are restrained; but the ordinary physical signs of pleural, pulmonary and cardiac diseases are absent, as are also the *points douloureux* that characterise intercostal neuralgia. If the movements of the corresponding ribs be restrained by plaster or bandage, the pain is effectually controlled. The direct constitutional disturbance is generally not great, unless sleep be prevented by pain.

3. **Lumbago.**—The muscular and fibrous structures of the loins are here the seat of pain—most commonly the erector spinæ, less commonly the latissimus dorsi, or other smaller muscles in the same situation, on one or both sides. As the

muscles of the back support the body in the erect position, and participate in the various movements of rising and bending the trunk in all directions, the patient may be compelled to remain at absolute rest in bed. More frequently he is able to move about, although with pain or in a stooping attitude. The amount of febrile disturbance is generally moderate.

Lumbago is easily recognised by the characteristic muscular pain referred to the loins, greatly increased by bending, straightening the back, rising from the sitting posture, or turning in bed; and by tenderness of the muscles on pinching, without the acute defined tenderness on pressure of abscess or neuralgia. At the same time it cannot be insisted upon too strongly, that careful examination of the back, of the abdomen generally, and of the urine will be required to prevent the practitioner from falling into the not uncommon error of treating cases of serious disease as simple lumbago. Renal calculus, lumbar abscess connected with spinal caries, perinephritis, perityphlitis, abdominal aneurysm, disease of the rectum, uterus or bladder, influenza, spinal meningitis, and small-pox, are the principal morbid conditions which must be borne in mind and excluded in every instance, before the diagnosis is settled. Pain in the loins is also a very common accompaniment of affections of the buttock and lower limbs, such as sciatica, rheumatic affections of the hip-joint, and perhaps lameness from any cause. It is also very frequently met with in gonorrhoeal arthritis.

4. **Omodynia.**—**SYNON.**: Dorsodynia; Scapulodynia. — These names are given to rheumatism involving the structures of the upper part of the back and shoulders. It occurs chiefly in persons much exposed to the weather; and has to be diagnosed chiefly from rheumatism of the shoulder-joint, and certain less common forms of neuralgia connected with the upper dorsal nerves and arms, thoracic aneurysm, and diseases of the cord and spine.

5. **Abdominal Rheumatism.** — Muscular rheumatism of the abdominal walls is occasionally observed, either alone or in association with lumbago.

6. **Cephalodynia.** — Muscular rheumatism may affect the scalp, giving rise to a dull, aching kind of headache, on the brow or occiput, aggravated by movement, and occasionally complicated with tenderness of the eyeballs and ophthalmia.

DIAGNOSIS. — Speaking generally, muscular rheumatism has chiefly to be diagnosed from neuralgia, and, as a rule, this can easily be done by the paroxysmal character of the latter, the etiological relations, and especially the physical signs. A very similar disorder that occurs very commonly in gouty subjects must be carefully distinguished (*see* RHEUMATISM, CHRONIC: Diagnosis). The practical diagnosis of the chief local varieties has already been sufficiently indicated.

PROGNOSIS. — The prognosis of muscular rheumatism is highly favourable. Under careful treatment recovery may be anticipated in a few days or weeks. But the disorder is one which is peculiarly liable to recur on exposure to its exciting circumstances.

TREATMENT. — The treatment of muscular rheumatism consists in remedying the constitutional condition; and in relieving the local pain. At the very commencement of the illness, a hot-air or

Turkish bath may answer both these indications, and give immediate relief. The first indication will, however, generally be best fulfilled by free purgation, followed by alkaline salines, such as potassium bicarbonate, citrate or acetate, and solution of ammonium acetate. In more feeble subjects quinine may be given combined with alkalis; or in protracted cases potassium iodide. The diet should be of the simplest character.

The best local treatment consists in absolute rest and warmth of the affected parts, which may be variously secured in different instances by confinement to bed, strapping and plasters. Mustard is one of the most available and successful of remedies. Other counter-irritants or anodynes which give great relief are warm opiate fomentations, or liniments composed of extract of belladonna and glycerine, opium, aconite, chloroform or camphor, or of various combinations of these. Liniments are more efficacious when sprinkled on the surface of a hot fomentation, firmly applied, than when rubbed into the part. If the suffering be great, a hypodermic injection of morphine and atropine should be given. Belladonna plaster gives at once relief and support in mild cases. The continuous galvanic current occasionally dispels the pain and stiffness almost immediately. A prolonged hot or mustard bath, carefully given, relieves some cases of lumbago. In obstinate cases cupping or blistering may be tried. The affected muscles must be kept warm and carefully protected from cold, especially in torticollis. When rheumatism involves the muscles of the limbs, warm anodyne liniments are the best local measures.

Great care should be exercised to prevent the recurrence of muscular rheumatism, by wearing warm woollen clothing; by avoiding exposure to damp and draughts; by attending to the digestion and the bowels; and by abstaining from severe, sudden, and awkward muscular efforts. In chronic or recurrent cases of the disorder, the patient should, if possible, visit some of the English or foreign baths mentioned in the article on RHEUMATOID ARTHRITIS. J. MITCHELL BRUCE.

RHEUMATOID ARTHRITIS.—**SYNON.**: Osteoarthritis; Rheumatic Arthritis; Rheumatic Gout; *Malum Coxæ Senile*; *Fr. Rhumatisme Nouveau*; *Usure des Cartilages Articulaires*; *Ger. Arthritis Deformans*.

DEFINITION. — A disease, the essential nature of which is unknown; mainly characterised by chronic degenerative and inflammatory changes of the articular and associated structures; and leading to deformity.

ÆTIOLOGY. — Persons of all ages may suffer from this disease, including children, but it generally begins between twenty and forty. It is believed to be more frequent in women. Depressing influences of all kinds unquestionably act as predisposing factors. They include acute diseases—such as influenza—menstrual disturbances, chronic uterine disease, frequent pregnancy, puerperal disease, superlactation, the menopause, prolonged physical exertion, privation, unhealthy surroundings and mental distress. The disease is hereditary, appearing in the same form or as acute or chronic rheumatism. In a certain proportion of cases, rheumatoid arthritis follows acute rheumatism immediately, or it appears after an interval of several years, during which time chronic rheumatism of a milder degree

may have been complained of. Gout is very often present in the family history, and not uncommonly tuberculosis also.

The influence of cold and damp as exciting circumstances is very marked. In some instances injury of a joint is the starting-point of the morbid process. Occasionally it follows gonorrhœal arthritis. A variety of micro-organisms have been described as the efficient causes of the disease.

ANATOMICAL CHARACTERS.—Two well-marked forms of rheumatoid arthritis are met with, according as a single joint only, or several—perhaps all—of the joints are affected. The anatomical characters are identical in the two forms.

Examined at an *early* stage of the morbid process, an affected joint is found to be enlarged; the synovial membrane, capsule and ligaments being distended and stretched by a considerable amount of effusion. Bacilli with specific characters have been found in the fluid by some observers. The synovial membrane is hyperæmic, swollen and thickened; its fimbriæ are enlarged and vascular; intra-articular fibro-cartilages, ligaments and tendons are vascular and softened; and the articular cartilages are partially removed, leaving a roughened, vascular, porous-looking surface behind.

In the more *advanced* stage of the process the effusion is considerably less, or may be completely re-absorbed; and the capsule and ligaments are much thickened or even partially calcified. The intra-articular structures, including fibro-cartilages, ligaments, tendons and articular cartilages, may have disappeared in great measure, leaving little or no trace behind. Peculiar bodies, consisting of pendulous masses of fibro-cartilage, are attached to the interior of the synovial membrane; more rarely they are free. The articular cartilages, where their opposed surfaces are in mutual contact, are replaced by an ivory-like layer of bone; while at other parts the surfaces present a pink colouration, with small spots of more intense hyperæmia. The articular surfaces are variously altered in shape and size. Thus articular cavities are widened, and occasionally deepened, by enlargement of the circumference, in the form of 'lips,' or by the production of separate bony masses in the same situation. The heads of bones are enlarged; present similar 'lips' or sharp edges at their widened margins; become flattened at right angles to the axis of pressure; and thus preserve their relations with the corresponding cavities, but can be readily dislocated from them. The shafts of the bones may be considerably altered in shape, increased in size, and changed in density. The associated tendons are frequently dislocated from their course beside the articulations, and atrophied or actually absorbed. The corresponding muscles are similarly atrophied. Bursæ in the neighbourhood of joints may be distended with fluid, and contain fibro-cartilaginous bodies; perioritis may also occur. The anatomical changes in this disease frequently present a remarkably symmetrical distribution. Peripheral neuritis occasionally accompanies the arthritic lesions.

SYMPTOMS.—The symptoms of rheumatoid arthritis in its condition of full development are exceedingly characteristic. The patient complains of pain and stiffness in connection with one or more joints; and on examination these are found to be swollen, more or less deformed, and tender. The history of these changes in the joints proves to be that first one and then others of the articulations were

the seat of acute arthritis, and became painful, tender, hyperæmic and swollen; that the resulting enlargement had not completely disappeared before the acute symptoms recurred; and that, by a repetition of similar acute or sub-acute attacks, the joints have reached their present condition. Thus the disease, while chronic in its course, consists essentially at first of recurrent acute or sub-acute attacks, which increase in frequency while their effects persist, and so finally become fused, as it were, into a continuous whole.

The *local* symptoms and signs vary with the particular joint affected; but in every instance they are chiefly these—pain, tenderness, creaking on movement, impairment of mobility, enlargement and deformity in connection with the joint, and atrophy of the associated muscles. The pain is generally distressing, and may render the patient's life miserable by its continuousness and severity, especially as it increases at night and prevents sleep. It is aggravated by movement, and there is tenderness on forcible disturbance of the articular surfaces rather than on pressure. Creaking or crepitation, audible and palpable, is a highly characteristic feature, which can be elicited and appreciated either by the patient or by the practitioner, and in the case of large joints may be so loud as to be audible at a distance.

The mobility of the affected joints becomes more and more impaired as the disease progresses—at first on account of pain, afterwards in consequence of anatomical changes. Thus the various joints may become fixed by a 'false' (very rarely a 'true') ankylosis, so that the hands cannot be closed; the wrists are immovable; the arms can hardly be moved from the side; the jaws are fixed; the head cannot be rotated; the patient may be unable to sit; and the knees, ankles and toes may be similarly impaired in function.

The variety of deformity is almost endless; and the particular character it assumes depends as much on the joint involved as on the nature of the process itself. Thus the knee, elbow, wrist and knuckles may present considerable intra-articular effusion, especially in the earlier stages; while the shoulder, hip and inter-phalangeal joints exhibit more limited swelling and 'drier' signs. The terminal digital joints become cuboidal or 'nodous'; the middle digital joints assume a spheroidal shape, or are partially dislocated backwards or forwards; and the knuckles are the seat of a peculiar oblique dislocation of the fingers towards the ulnar side. The lower ends of the radius and ulna project backwards, and give a full appearance to the dorsum of the wrist, which may be further increased by extra-articular puffiness, carpal and bursal enlargements, and atrophy of the muscles of the hand and forearm. Altogether, the hands and wrists are the joints most frequently affected. The elbow-joint is swollen; and bursal collections—fluid and solid—develop over the olecranon. The shoulder presents signs of wasting, rather than of enlargement, due to atrophy of the deltoid and other muscles; the head of the humerus at the same time lies unnaturally forwards and upwards; and a corresponding depression is apparent behind. At the hip-joint the disease gives rise to flattening of the buttock, shortening of the limb, and eversion of the foot; enlargement can sometimes be felt in connection with the head of the bone and acetabulum; occasionally the patient may be not only

lame but unable to sit, and accordingly must either stand or lie constantly. The knee, a very common seat of the disease, is enlarged by the presence of considerable effusion in the earlier stage; and when this afterwards becomes absorbed, local bony growths are easily felt, giving increased breadth to the patella, and forming sharp crests at the lateral margins of the articular surface of the condyles. The disease as it affects the ankle and foot does not require special description. At the temporomaxillary articulation rheumatoid arthritis gives rise to obvious enlargement in front of the ears, and possibly to distortion or asymmetry of the chin. Prominent nodular swelling is the principal sign of the disease at the sterno-clavicular articulation. In the spine it chiefly produces rigidity, as well as pain locally and down the arms, and leads to a variety of permanent deformities, with which contractures and distressing spasms of the extremities are sometimes associated.

The *general* condition of the subject of rheumatoid arthritis, when it is advanced, is one of debility and anæmia. The face is pale and expressive of suffering; the complexion is muddy. The skin is peculiarly inactive, and rarely perspires. The patient looks pinched, and complains of a feeling of cold; the extremities are often miserably cold and livid; but the palms of the hands are damp or even soppy. Pyrexia, rising to 101° with considerable remissions or intermissions, may be present in the active phases. Bodily activity is greatly impaired, owing to interference with the movements of the limbs; in many instances the patient is completely crippled and bedridden. Even the voice and the hearing may be impaired, from involvement of the laryngeal and auditory articulations. Pains in the muscles and along the nerve-trunks and in the extremities may accompany or possibly precede those in the joints; and spasms or attacks of 'muscular rheumatism' occur occasionally. Myotatic irritability is frequently increased. The various bodily functions are feeble, and frequently deranged; and although the mind may be active, the condition is rendered wretched in the worst cases by pain, deformity and helplessness. In a considerable proportion of cases (5.75 per cent., Brachet) the patient is the subject of chronic valvular disease of the heart. Kent Spender drew attention to the frequent occurrence in rheumatoid arthritis of other symptoms variously associated with the disease: gastric crises; increased tension and frequency of the pulse, which may rise to 120 or more; excessive pigmentation of the skin, in the form of symmetrical patches, streaks or spots; and yellow bruise-like blotches on different parts of the body.

COURSE AND TERMINATIONS.—The course of rheumatoid arthritis has been already described. Unless it be treated early, the disease progresses essentially towards deformity. Death from rheumatoid arthritis is rare; its other distressing effects have been sufficiently indicated.

PATHOLOGY.—Great diversity of opinion prevails as to the essential nature of this disease. While most authorities acknowledge acute rheumatism as a cause of rheumatoid arthritis, many of them regard it as simply one of the many possible sources of chronic articular irritation. The view most generally held in this country appears to be that it is a disease distinct from rheumatism and gout. Hutchinson maintains that, in a certain

number of instances, there is an element of gout, as well as of rheumatism, in the disease. Ord makes light of the diathetic element, and attaches most importance to a nervous factor in the pathology of this disease, which he regards as a dystrophy of joints, muscles and other associated tissues, traceable to direct or reflex affection of nervous structures, or to general nervous depression. Kent Spender also holds that the affection of the joints is merely one sign of a profound nerve-disorder. Archibald Garrod argues strongly in favour of the dystrophic theory of the disease. The writer's experience is in favour of the strictly rheumatic nature of rheumatoid arthritis, as was maintained by Todd and by Charcot and French authorities generally. In many cases he has found that the morbid process started in an attack of ordinary acute rheumatism, as was originally represented by Adams of Dublin. In numerous instances the family history is distinctly rheumatic. The frequent association of heart-disease is very important evidence in the same direction. Finally, no sharp line can be drawn between acute and sub-acute cases of rheumatism, between sub-acute and chronic cases of rheumatism, or between chronic rheumatism and so-called 'rheumatoid arthritis.' Dyce Duckworth considers it a manifestation of a basic arthritic stock or diathesis—a rheumatic branch of this stock, and therefore a true rheumatism.

DIAGNOSIS.—The diagnosis of rheumatoid arthritis necessarily depends upon the view entertained of its pathology. If considered a distinct disease, it is, as a rule, easily separated from gout by the entire absence of tophi about the joints and in the ears, by the history of the disease, by the absence of manifestations of irregular gout (*see* RHEUMATISM, CHRONIC: Diagnosis), and, in doubtful cases, by the absence of uric acid in the blood. From chronic rheumatism, as ordinarily defined, it is diagnosed by the amount of deformity present. Charcot's disease of joints is known by its nervous associations, and hypertrophic pulmonary osteoarthropathy (p. 19) by precedent affections of the chest. Chronic synovitis of traumatic or constitutional origin may be occasionally mistaken for rheumatoid arthritis; but the presence of the latter disease in several joints, probably symmetrically, should remove all doubt. Rheumatoid arthritis of the hip and shoulder has been confounded with 'dislocation' and 'intracapsular fracture.'

PROGNOSIS.—The prognosis of this disease is favourable as regards life, but unfavourable as regards cure, comfort, or ability to follow active bodily employment. The prognosis is much better in the rich, who can seek relief by change of climate in the earlier stages, than it is among the poor, in whom the disease must in a measure be allowed to pursue its progressive course.

TREATMENT.—The treatment of rheumatoid arthritis must be applied in two directions: first, to arrest, if possible, the morbid process; and secondly, to relieve distress and disability. In a large number of cases the second indication only can be fulfilled, for the disease is frequently too advanced, or the circumstances of the patient are too poor, to afford a prospect of cure.

In the *early stages* of the disease much can be done by energetic treatment, which must be partly constitutional and partly local. If circumstances permit, the patient should be advised to visit, according to the season of the year, the baths either

of Great Britain, of Germany, or of France in summer, or the Algerian springs, the French Riviera, or Italy in winter. Buxton, Bath and Strathpeffer are the best home baths. Aix-les-Bains, Aix-la-Chapelle, Baden-Baden and Wiesbaden may be recommended from May till September. The other places named, especially Hammam-R' Hira, in Algiers, are winter resorts. At all properly appointed bathing establishments massage now forms a part of the treatment. The climate of Egypt proves beneficial in some instances, while a voyage to the tropics or subtropics will suit other patients; and in every case a bright, dry, cheerful locality should be selected for residence if possible. See MINERAL WATERS.

The most valuable internal remedies for rheumatoid arthritis are cod-liver oil, iron, quinine and arsenic. Cod-liver oil should be taken regularly if the digestion permit. Either iron or arsenic, or the two combined, should be taken in full doses for periods of weeks or months, and their effects carefully noted. Sir Alfred Garrod especially recommends the syrup of ferrous iodide. For acute distress, particularly pain, salicylates may sometimes be given with success. In obstinate cases a trial should be given to potassium iodide, and to guaiacum.

The diet should be carefully ordered. While all excess is avoided, as well as indulgence in malt liquors and rich indigestible dishes, a generous supply of mixed animal and vegetable food and wholesome stimulants will be found to be most suitable. The patient should take advantage of every possible opportunity of enjoying daily outdoor exercise in fresh air and sunshine. The clothing must be warm, flannel or other woollen material being worn both summer and winter. Great care must be exercised to avoid cold and damp, in the routine of daily life and in the choice of a residence.

The local treatment is to be considered of hardly less importance than the constitutional. On the first appearance of the disease, counter-irritation should be freely employed around the joints by means of tincture or of strong solution of iodine, which may be used until the skin becomes of a mahogany colour. The joints should then be carefully protected by cotton-wool or flannel, and kept at rest by means of bandages or other means of support, according to the part that is involved. At the end of the sub-acute attacks of the disease, efforts must be made to restore the healthy nutrition of the affected joints by removal of the support, and by local stimulation and graduated exercise. Joints that can be easily reached should be thoroughly fomented night and morning, by wrapping a piece of dry cambric or flannel around them, and sponging water over this, as hot as can be borne. After ten minutes of such treatment the joints should be unwrapped, carefully dried, and then thoroughly rubbed, either with a stimulating liniment, such as the turpentine or acetic turpentine liniment, with a mild mercurial ointment, or with some bland oil, such as cod-liver oil or goose-grease. A light warm covering is then to be applied. The effect of local treatment of this kind, if pursued steadily, is often remarkable, mobility being restored in cases where the joints have been useless for months.

In very advanced cases, especially in old subjects, it is manifestly unreasonable to expect much improvement. Anodyne treatment is then chiefly

called for, and a good deal can be done in this direction by suitable mechanical arrangements and well-chosen local applications, the preparations of opium being, of course, the most successful. Phenazone may relieve pains and spasms of the crippled limbs. The general health will demand support by a well-regulated diet, residence in a warm, dry, moderately bracing climate, and the internal treatment suggested above.

J. MITCHELL BRUCE.

RHINITIS (ῥίς, the nose).—Nasal catarrh. See NOSE, Diseases of.

RHINOLITHS.—See NOSE, Diseases of; and CONCRETIONS.

RHINOPHYMA.—See ACNE ROSACEA.

RHINORRHOEA.—See NOSE, Diseases of.

RHINOSCLEROMA.—See NOSE, Diseases of.

RHINOSCOPY.—See NOSE, Examination of.

RHONCHAL FREMITUS.—A physical sign, appreciated on palpation of the chest, elicited by the act of breathing when certain secretions or other materials are present in the larger air-tubes or in a cavity. See PHYSICAL EXAMINATION.

RHONCHUS (ῥέγκω, I snore).—Rhonchi are sounds heard on auscultation when the air-channels are partially obstructed. The term is restricted by some authors to the so-called *dry* and more or less musical sounds produced in the bronchial tubes, for instance, *sonorous* and *sibilant* rhonchus; the bubbling and crepitating sounds in chest-disease being spoken of as *râles*. By other authorities, again, all such sounds, whether sibilant or crackling, are described either as *rhonchi* or as *râles*, the terms being interchangeable. See PHYSICAL EXAMINATION.

R. DOUGLAS POWELL.

RIBS, Diseases of.—See CHEST-WALLS, Morbid Conditions of; and CHEST, Deformities of.

RICKETS (O. E. *wrikken*, to twist).—SYNON. . *Rachitis*; Fr. *Rhachitisme*; Ger. *Rhachitis*; *Englische Krankheit*.

DEFINITION.—A general disease, affecting the nutrition of the whole body; arresting natural growth and development; perverting and delaying ossification; retarding dentition; causing the bones to become soft, and to yield to pressure, and the muscles and ligaments to waste; and in many cases producing alteration of the brain, liver, spleen, and lymphatic glands.

ÆTIOLOGY.—Rickets is the consequence of slow impairment of nutrition, and the causes which produce it are principally—bad feeding, foul air, damp cold rooms, want of sunlight, want of exercise, and want of cleanliness. Of these, perhaps the first two have the greatest influence in causing the disease; for if the quantity of nutritive material introduced into the system be restricted by an improper selection of food, and if the oxidation of waste matters be hindered by an insufficient supply of fresh air, interference with nutrition is necessarily carried to a high degree. A pure bracing air will by itself do much in counteracting the effects of an improper dietary, for it has been noticed

that injudicious feeding is less hurtful in country places where the air is dry than in large towns. This, however, may be partly explained by the greater vigour of the digestive organs in the former case, enabling the child to derive nourishment from food which, under other conditions, would be in-nutritious. Some children are affected more readily and more severely by these causes than others, for the more the strength of the child is reduced before the actual exciting causes of the disease come into play, the more quickly does the patient fall a victim to their effects. Therefore, all influences which impair the general strength, such as weakness in the parents, or, in the case of the child himself, an attack of acute disease, or even unusually troublesome dentition, must be looked upon as predisposing causes of the disorder. There is no proof that rickets is hereditary. A tubercular family predisposition renders the occurrence of rickets unlikely. Some years ago a distinguished foreign physician attempted, by arguments drawn chiefly from morbid anatomy, to prove rickets to be invariably a consequence of inherited syphilis; but the reasons for rejecting this hypothesis are overwhelming.

ANATOMICAL CHARACTERS.—The bones are affected in three ways: growth is retarded; the spread of ossification into parts still cartilaginous is interfered with; and bone already ossified is softened. The growth of bone is not completely arrested; it rather becomes irregular. There is considerable development of the cartilaginous epiphyses, and also of the fibrous periosteum; but these parts ossify incompletely and slowly; and as the normal increase in size of the medullary cavity continues in the usual way, the bone comes gradually to consist less and less of osseous substance, and more and more of, as yet, unossified matter proliferated at the circumference. It is in this way that the bones become soft, and not from any abnormal absorption of earthy salts from bone already ossified. The process of calcification itself, besides being retarded, is abnormal: it has indeed been described as rather a process of petrification than of true ossification. On account of the softness of the long bones, serious deformities ensue, as will be afterwards described. The flat bones become greatly thickened from proliferation of the periosteum. This is especially noticeable at the edges of the cranial bones; and when ossification is completed, the sutures of the skull can be felt to be prominent. In parts, however, and especially in the occipital bone, the osseous substance becomes thinned in places from absorption under the pressure of the growing brain. This condition, which is called 'craniotabes' (p. 350), can be detected by palpation. Ossification is very slow in the cranial bones, and the fontanelle often remains open long after the end of the second year. See SKULL, Diseases of.

The liver, spleen, lymphatic glands, and kidneys are sometimes enlarged. The increase in size is due, not to the presence of any foreign growth or deposit in these organs, but to irregular hypertrophy of their fibrous and epithelial elements, conjoined with a deficiency in earthy salts—an alteration analogous to the changes in the bones. The brain is enlarged from an increase of the neuroglia, not of the nerve-elements. The voluntary muscles are small, pale, flabby, and soft. Under the microscope their striæ are seen to be indistinct. The urine

contains less urea and uric acid than natural, but more phosphates, especially phosphate of lime.

The exact nature of rickets has yet to be discovered. All evidence is against the hypothesis that the nutritive fault lies in the excessive formation of lactic acid, as suggested by Heitzmann, or that the complaint is produced by deficiency of lime in the food, as was at one time supposed. Mercoli and others attribute the disease to a process of auto-infection by streptococci and staphylococci. According to this theory the organisms are absorbed from the alimentary canal into the blood and are deposited in the various organs, especially in those parts—such as the nervous system and the bones—where development is most active. In the bones they set up a chronic osteomyelitis of a special type.

SYMPTOMS.—In most cases the symptoms proper to rickets are preceded by others which indicate a certain amount of interference with the digestive functions. There is occasional vomiting; the bowels are often relaxed; and the motions are habitually loose, pasty-looking, and offensive.

The beginning of the disease is marked by profuse sweating of the head, face, and neck; this is especially seen if the child fall asleep either at night or in the day. Almost at the same time he begins to throw off the bed-clothes at night. He will do this even in winter, and may be seen lying almost naked in the coldest weather. Characteristic changes soon occur in the bones. The ends of the long bones enlarge; the flat bones become thickened; and all the bones lose their firmness and grow softer. These changes affect the osseous system as a whole, and lead to serious deformities. If the child had been able to walk, he becomes unsteady on his legs, or even loses the power of walking altogether. He sits or lies about; is drowsy in the daytime; and at night moves his head restlessly from side to side, so as in many cases to wear the hair off the occiput. The flesh is soft and flabby; the motions remain loose and offensive; and the child appears to be occasionally troubled with abdominal discomfort, for he may be found asleep in his cot, resting upon his hands and knees, with his head buried in the pillow. When the bone-affection is pronounced the child shows strong dislike to being touched or played with. This wish for quiet is usually ascribed to tenderness, but is more probably occasioned by the knowledge that movement hurries the breathing and raises a demand for air which the chest-wall with its softened ribs is unable to satisfy. If evident tenderness be present, the symptom usually indicates that the case is becoming complicated with scurvy.

A prominent symptom in rickets is the deformity produced by alterations in the shape of the skeleton. The softened ribs yield readily to pressure, and it is to pressure, and not to the force of muscular action, as was at one time supposed, that the distortions are chiefly due. The long bones become bent and twisted. The direction of the bending depends upon the direction in which the force of pressure is applied, and in the lower limbs will therefore manifestly vary according as the child can or cannot walk. Sometimes, however, if the disease begins before the child is able to support himself upon his feet, the lower limbs may escape deformity altogether. In such cases they are usually small and thin, with weak, flabby muscles; but the bones

themselves are straight. Force of gravity is another cause of deformity of bone. Thus, in the humerus there is often a curve where the deltoid is inserted; this is produced in great measure by the weight of the hand and forearm when the limb is raised by the deltoid muscle.

The skull is elongated from before backwards; the anterior fontanelle is wide; the sutures are thickened; the forehead is high, square, and sometimes prominent; and the head generally looks large. By palpation of the occiput the condition named 'craniotabes' can sometimes be detected early in the disease. The face appears unduly small when compared with the head, for the growth of the facial bones is arrested. On account of this pause in the development of the jaw-bones dentition is delayed; but the teeth are not only slow to appear, they are also deficient in dental enamel, so that they are no sooner cut than they begin to decay.

The spine is curved on account of muscular and ligamentous weakness; and if this weakness be great, the natural posterior curve of the spine is so much exaggerated as almost to simulate angular curvature. It disappears, however, at once when the child is lifted up by the shoulders. Sometimes the spine is curved laterally.

The deformity of the chest has the following characters: The softened ribs sink in so as to present a groove passing downwards and outwards on each side of the sternum. The bottom of the groove is formed more by the ribs than the cartilages, so that the enlarged ends of the ribs, looking like a row of beads under the skin, can be seen lying along the inner side of the groove. The sternum is forced forwards by this bending of the ribs, and the antero-posterior diameter of the chest is increased. The deformity is due to the pressure of the external air. In healthy breathing this pressure is overcome by the resistance of the thoracic walls, aided by the force of the inspired air. In the rickety child the resistance offered by the softened ribs is greatly reduced, and they therefore sink in at the parts where they are least supported. On account of the softened state of his ribs, the breathing of a rickety child is quick and laborious.

The pelvis is pressed upon from above by the spine and the abdominal contents, from below by the heads of the thigh-bones; and the direction of these forces varies according to the position of the child. The general shape thus produced is triangular, and the pelvic cavity is often greatly narrowed.

A rickety child is short for his age; for his limbs, besides being bent, are stunted, growth in them being more or less arrested. His joints are large, and loose from relaxation of the ligaments. If the disease be severe, the child gets anæmic and wastes, and the muscles become very flabby and small. His belly is big, even where there is no splenic enlargement, from shallowness of pelvis and flatulent accumulation. Such children give little trouble. They are quiet, and seldom cry if left alone. They are late in walking, late in talking, cut their teeth late, and in nursery phraseology are 'backward children.'

COMPLICATIONS.—One of the chief characteristics of rickets is the *intense sensitiveness to cold* with which it is always accompanied; and it is to hills in different forms that a large proportion of deaths occurring in this disease must be attributed. A catarrh may affect the chest or the belly, and in

either case the complication is a very dangerous one.

A pulmonary catarrh in a young child should never be made light of, on account of its tendency to cause collapse of the lung; and if the child be the subject of rickets, the danger is really imminent on account of the softness of the ribs.

If the chill affect the abdomen, as it is very apt to do, an intestinal catarrh is set up; and unless the diarrhoea be quickly arrested, the strength of the child becomes seriously reduced.

Besides its influence in increasing the susceptibility of the body to cold, rickets also heightens the *nervous excitability* of the child. This effect is not a common result of mere weakness, for in an ordinary case of malnutrition with wasting, the natural sensitiveness of the nervous system to external impressions is impaired. It must be therefore looked upon as a peculiarity of the rickety state. Its effects are seen in the attacks of laryngismus stridulus, tetany, rotatory spasm, and convulsions to which these children are so liable. Few cases of laryngismus occur in children who are not the subjects of rickets. Such patients usually have carpo-pedal contractions, and are liable to be convulsed upon the very slightest provocation. On account of the backwardness of the teeth in this disease, all nervous derangements are commonly attributed to dentition; but in rickets dentition, although delayed, is not necessarily troublesome; in fact, the teeth, when they appear, are often cut with singular ease.

Another important complication is *infantile scurvy*; indeed, in the child scurvy is rarely seen apart from rickets. It is not that the complaint is due directly to the general impairment of nutrition upon which the rickety state depends, but that owing to the impoverishment of the blood the patient has become peculiarly susceptible to a scurvy diet, and if fed largely upon tinned foods rapidly develops the disease.

Infantile scurvy is rarely seen before the sixth month, and is most common between the tenth month and the twelfth. For some days the infant is noticed to be getting pale and spiritless; then suddenly he shows signs of pain when he is washed and dressed. The legs are evidently tender. If able to stand, the child refuses to put his feet to the ground, and cries if required to do so. He shows a great dislike to movement, and as the tenderness increases begins to keep his lower limbs flexed upon his body. He lies quietly in his cot, sometimes moans as if in pain, and screams at the slightest touch. At this time the lower parts of the thighs, the knees, or the legs may be noticed to be swollen, but the colour of the skin is unaltered. The swelling is bilateral and is due to extravasation of blood beneath the periosteum. As a rule it is limited to the parts named, but in exceptional cases may affect the arms, especially about the elbow and wrist or the shoulder. Sometimes there is slight œdema. While these local symptoms continue there may be moderate fever (101°–102° at night), but more often the temperature is normal. The complexion is pallid, with a faint sallow tint; and the child is very feeble and sweats profusely. There are usually petechiæ and bruise-like patches on the skin, and hæmorrhages may occur from the nose, the bowels and the kidney; indeed, hæmaturia is a far from uncommon symptom. If a tooth has been cut, the gums may be spongy and sore, and they

are sometimes excessively swollen. If, however, the complication occur before dentition has begun, the gums are rarely affected. In the worst cases there may be separation of the epiphyses of the affected bones. The child then ceases to flex the limb, and lies with it stretched out as if paralysed.

Under appropriate treatment improvement is rapid. In an ordinary case the more urgent symptoms subside in a few days, and even in bad cases recovery is rarely long delayed. A separated epiphysis under favourable conditions becomes again united with the bone, and the patient quickly regains colour and strength. See SCURVY.

Sometimes rickets is accompanied by *chronic hydrocephalus*; the excess of fluid is, however, small. This complication is often suspected where it does not really exist.

DIAGNOSIS.—When the symptoms of rickets are well marked, the bony distortions themselves are sufficiently characteristic to make the nature of the disease unmistakable. It is, however, of great importance to recognise the early symptoms of the disorder, so that by prompt treatment we may prevent the osseous and other changes taking place. It must be remembered that loss of flesh is a late symptom, and that a rickety child is not necessarily a thin one. If an infant pass the ninth month without any appearance of a tooth; if his wrists enlarge; and if on enquiry we find that he is subject to head-sweats at night, and likes to lie naked in his cot, the diagnosis of rickets may be made without hesitation. Weakness of the legs in a young child is often a source of anxiety to parents, and a medical practitioner is consulted because the child is twelve months old and cannot stand. In these cases the early signs of rickets will almost certainly be discovered. Looseness in the joints is common in those cases of rickets, where the symptoms of the disease manifest themselves at the end of the second year. The relaxation of the ligaments is not as a rule combined with much bone-deformity, although it may be so. Weakness of the legs from rickets is distinguished from essential paralysis by the fact that, although there may be no power of standing, the child is yet able to move his limbs; and that the muscles, although weak, are not powerless.

When scurvy occurs it is important not to misapprehend the nature or gravity of the complication. Early treatment is essential to quick recovery, and delay adds greatly to the child's suffering, if it do not put his life in danger. Marked tenderness in a rickety baby should suggest scurvy at once; and if the child scream loudly, while holding both legs flexed and motionless, our suspicions are amply confirmed. Even if swelling be trifling or absent, gentle manipulation will detect thickening round the shaft of the bone at the seat of tenderness. This is especially likely to be found round the femur for some distance above the knee. In addition, the gums may be spongy, although this symptom is often absent; and there may be petechiæ or bruise-like patches, or even unhealthy sores, on the skin.

PROGNOSIS.—The duration of rickets is dependent upon the duration of the causes which produce it. So long as the unhealthy influences under which the disease originated are in operation, the morbid processes continue; but when a better hygiene is adopted, and failing nutrition is restored, recovery begins.

When recovery takes place, the symptoms gradually become less intense and finally disappear. The

enlargement of the joints greatly diminishes, and even the bony distortions become notably reduced, while the bones themselves become thick and strong. Growth, however, is not rapid, and, if the disease have been severe, the child seldom reaches the average height.

When the disease terminates fatally, it is usually through one of the complications which have been mentioned. Sometimes the child sinks and dies, apparently worn out by the intensity of the general disease; but even in these cases the immediate cause of death is usually asphyxia, through the softened state of the ribs. One cause of the great mortality from bronchitis in children is the frequency with which that complaint attacks rickety subjects, even a mild catarrh being dangerous when the ribs are much softened.

In estimating the prospects of recovery in any particular case, we must pay attention to the amount of chest-distortion; and to the presence or absence of disease of the glandular system. If the ribs be much softened, there is always cause for anxiety; and if in a case of pulmonary catarrh there be great recession of the lower ribs in inspiration, the condition is a serious one. The presence of any complication, except perhaps chronic hydrocephalus, necessarily increases the gravity of the case.

TREATMENT.—As rickets is the direct result of malnutrition produced by the unwholesome conditions in which the child has been living, our first care must be to alter these conditions. We must see that the living-rooms are thoroughly ventilated; that the child is taken out regularly into the open air; that he is warmly dressed; and that in his daily bath the washing-process is conducted rapidly so as to avoid a chill. We must next select for the patient a diet which is at once sufficiently digestible and nutritious. The term 'digestible' as applied to diet is a relative term. Food digestible to one infant is indigestible to another, and food readily digested by a child in his natural state of health becomes indigestible to him when his stomach is temporarily weakened by teething or any febrile attack. It is not, however, sufficient that the diet should be digestible; it must also be nutritious. Children kept too long at the breast frequently become rickety, even although fed at the same time upon other and suitable food; for the watery breast-milk, which forms the principal part of their diet, is sufficient by its bulk to satisfy their desire for food, without supplying the required nourishment to the tissues. Rickety children at the breast should at once be weaned, and, if under twelve months old, should be fed principally upon milk guarded with saccharated solution of lime, in the proportion of fifteen drops to the bottleful. They may take, besides, broths, bread and butter, and occasionally the yolk of an egg lightly boiled or beaten up with milk. Instead of bread and butter, the milk may be thickened for some meals with Chapman's wheat-flour baked in an oven; but farinaceous food should be given with very great caution to these children, on account of their tendency to acid indigestion, which renders a starchy diet particularly likely to disagree. Under twelve months of age the child can seldom bear more than one teaspoonful of a farinaceous powder twice in the day. After the first year, strong beef-gravy, and flower of broccoli stewed, may be added to the diet. At sixteen or eighteen months old, a little mutton may be given, carefully pounded in a warm mortar. A mealy

potato, well boiled and mashed, may be allowed, but the effect of all farinaceous food is to be carefully watched. The presence of flatulent pains is a sure sign that the proper quantity has been exceeded. Often in rickets digestive power is very feeble, so that even with the most carefully regulated diet improvement is halting and progress slow. When this is the case, great advantage may be derived from the use of papain, giving one grain in a teaspoonful of water with a grain of bicarbonate of sodium immediately before each meal.

The diet and general sanitary arrangements having been regulated, the secondary question of drug-giving has to be considered. Before adopting tonic treatment, it is important to improve the condition of the bowels. A dose of castor-oil or of rhubarb and bicarbonate of sodium should be given to clear away undigested food, and afterwards a few grains of bicarbonate of sodium with a drop of tincture of opium in a little aromatic water will soon remove the offensiveness of the motions. Citrate of iron may then be ordered, and the child should begin at once to take cod-liver oil. The dose of the oil should be small at first (m xv-xx), and while it is being taken, the motions should be watched for any reappearance of oil in the stools; if this occur, the dose is too large, and must be diminished. As convalescence advances, other medicines may be given; and iron wine, quinine, and reduced iron are all useful. So long as the previous directions have been attended to, the exact tonic used is of comparatively little moment; but cod-liver oil should never be omitted from the treatment. Lime has been strongly recommended by some authors, but according to the writer's experience is of little value unless combined with iron. It may once more be repeated that in rickets the success of treatment is in direct proportion to the conscientiousness with which the rules relative to diet and general management have been carried out; and the mother should be made to understand that the child's recovery depends upon her own watchfulness and care.

The osseous deformities can be prevented, to a certain extent, by hindering the child from walking while the bones are still soft. The bowing of the legs is often owing to the child's getting upon his feet before the bones have become sufficiently consolidated to bear the weight of the body. In these cases light well-padded splints which project below the feet will be the best safeguard. When the ligaments of the joints are loose and weak, the joints may be much strengthened by a well-fitting elastic support.

After the tenderness of the body has subsided, the child should be well shampooed, especially along the spine, both morning and evening.

With regard to the complications—diarrhoea and pulmonary catarrh must be treated upon ordinary principles. A good flannel bandage very much diminishes the tendency to relaxation of the bowels, and is of further value in retarding the too rapid descent of the diaphragm, and so in diminishing to a certain extent the recession of the chest-walls during inspiration. If convulsions occur, search must be made for any local disturbance which may have set them up. This is often some form of gastrointestinal irritation. It must be remembered that the abnormal sensitiveness of the nervous system in this complaint renders the patient peculiarly responsive to such impressions, and that the beginning of the infectious fevers and the agitation of whoop-

ing-cough are more likely than in a more healthy child to be followed by nervous phenomena. Rotatory cervical spasm is often quickly cured by a few nightly doses of rhubarb and soda, at the same time reducing the quantity of starch and other acid-making matters in the food. The same may be said for the milder cases of tetany. The more severe form of the latter disease yields only to the measures which are effectual in subduing the rickety condition, viz. wise feeding, fresh air and tonics. It is, however, often obstinate, and may only subside as the original disease declines. Chloral hydrate and other nerve sedatives are of small value in reducing the spasms (see CONVULSIONS IN CHILDREN; and HEAD-NODDING OF INFANTS). Laryngismus stridulus is often cured at once by bathing the whole body twice a day with water of the temperature of 70° F. while the child sits in hot water. At the same time it is well to remember that these attacks are usually significant of the presence of adenoid vegetations in the naso-pharynx, and are apt to return unless the vegetations be removed.

The moment scurvy is detected, the child's diet should be revised. He should be given plenty of fresh cow's milk, and this may be thickened once or twice a day with a well-boiled potato. For medicine, half a grain of quinine dissolved in a teaspoonful of fresh lemon-juice can be given three times a day. If the gums are spongy, Cheadle's recommendation of the glycerines of tannic acid and carbolic acid (fifteen drops of each in an ounce of water) is useful, applied with a soft brush. The legs, if tender, should be supported by a soft cushion behind the knees. If the epiphyses have separated light splints must be applied.

EUSTACE SMITH.

RICKETS, FŒTAL.—See ACHONDROPLASIA.

RICKETS, SCURVY.—See RICKETS, p. 1429; and SCURVY, p. 1469.

RIGIDITY (*rigidus*, cold, frozen, stiff). —SYNON.: Fr. *Rigidité*; Ger. *Starrheit*.—This term implies the existence of a more or less fixed condition in parts that ought to be freely movable. It is a state met with principally in the limbs, where it is dependent upon certain unnatural conditions of the joints or of the muscles, either separately or in combination. It may, however, occur in the trunk as a whole, or in the neck, owing to the existence of tetanic or tonic spasms in muscles, due to one or other of various causes. The valves of the heart, and the arteries, when stiffened by fibrosis, are said to be rigid. Again, it is a term commonly applied to a condition of the 'os uteri' during parturition, in certain women, in whom the orifice of the womb does not dilate coördinately with the increase in force of the uterine contractions. The term is also sometimes used in connection with the features. Marked rigidity of a limb at this or that articulation often results from joint-disease. Perhaps more frequently, however, rigidity in a limb is primarily dependent upon altered functional or nutritive conditions of its muscles, which may or may not be associated with actual paralysis implicating the same parts.

Numerous cases exist in which, without the existence of paralysis, or with a comparatively small amount of it, tonic spasms occur in the muscles of a limb, so as to entail rigidity (see

SPASM). This may be met with, for instance, in hysteria, and in the early stages of some spinal diseases, more especially in primary lateral sclerosis, or in the form of paralysis that goes with Pott's disease.

More frequently still, however, rigidity is found in association with distinct paralysis. For many years a distinction has been made between two kinds of rigidity associated with paralysis; the one of which, known as 'early rigidity,' is apt to supervene with or a few days after the onset of a cerebral or spinal paralytic affection; while the other, known as 'late rigidity,' comes on rather in old cases in which mere paralysis with flaccidity of muscles may have been previously present. The former is now believed in many cases to have a tendency to pass into the latter form; and where this becomes well-developed, it is commonly associated with a secondary or with a primary sclerosis of the corresponding lateral column of the spinal cord, even though the initial paralytic lesion may be in some portion of the motor tract of the opposite cerebral hemisphere (see SPINAL CORD, Diseases of). In 'early rigidity' we have to do with mere functional changes in the muscles, and the condition itself of rigidity is not constant; it intermits from time to time; during the day, and commonly disappears during sleep. But in 'late rigidity,' associated with extensive secondary degenerations in the spinal cord, the nutrition of the nerves, as well as of the muscles and their tendons, appears to suffer, and that, for the most part, in an irremediable manner. This more severe condition of rigidity is associated with actual shortening of muscles or tendons, and in this stage but little, if any, difference exists between the degree of rigidity of the limbs by night and by day. See MOTILITY, Disorders of.

H. CHARLTON BASTIAN.

RIGOR.—SYNON. : Shivering Fit; Fr. *Frisson*; Ger. *Schüttelfrost*; *Fieberfrost*.

DEFINITION.—A group of phenomena the essential constituent of which is a series of clonic muscular contractions accompanied by a rise of temperature, and by various subjective sensations, chiefly of cold and heat.

DESCRIPTION.—A rigor generally commences with a sensation of chilliness referred to the back or the extremities, accompanied by a feeling of wretchedness and by headache. The extremities are cold, blue and pinched, and their surface-temperature is lowered. The patient usually sits or lies huddled up, heaping coverings on himself in his endeavour to keep warm. The respirations are shallow and hurried, the pulse small and rapid. Nausea is frequent and actual vomiting may occur. The internal temperature of the body is raised and the surface of the trunk may feel unduly hot. After this condition has lasted a varying period the actual rigor occurs. This consists of a series of involuntary clonic contractions, commencing in the muscles of the lower jaw, and causing chattering of the teeth. The clonic contractions then spread to the muscles of the trunk and limbs, and may cause sufficient movement to shake the bed or chair occupied by the patient. These contractions last for a period varying from a few seconds to two hours, and are succeeded by a general flushing of the skin, accompanied by a feeling of heat and thirst and by copious perspiration. The pulse becomes full and bounding, and

the temperature begins to fall. The headache continues, but consciousness is rarely lost during the attack, though mental processes are interfered with, irritability being often marked at the onset of the attack.

The rise of temperature which accompanies the occurrence of rigors is always rapid, but the actual degree of pyrexia does not appear to influence the severity or duration of the rigors themselves. The rigors do not occur at the maximum temperature, but usually, when the temperature has reached about one-half to two-thirds of its maximum. The rigor itself may be single, or may occur as a series of shivering fits following quickly on one another. Death rarely, if ever, takes place during a rigor.

ÆTIOLOGY AND PATHOLOGY.—In the phenomena above described we recognise the action of the central nervous system. The vaso-motor, respiratory, vomiting and thermotactic as well as the motor and psychical centres are involved. The phenomena are not to be explained by the existence of any special centre concerned with the production and control of rigors, but seem rather to indicate the existence of some toxic agent or agents in the circulation, causing inhibition of the highest—i.e. controlling—function of various cerebral centres. Thus, in the rise of temperature, shivering, vomiting, altered pulse and respiration and altered sensations we see interference with centres we have reason to locate in very different parts of the central nervous system. The similarity rigors bear to one another, their usually short duration, and the fact that paralysis does not follow the muscular movements indicate that we have to deal with a withdrawal or suspension of normal inhibition from, rather than a direct stimulation of, the various centres. Pyrexia itself cannot be assumed to be the direct cause of the rigors, nor can mere change of temperature, for, although we find some of the phenomena, such as the sensation of cold, and the transient shiver as the result of sudden exposure to cold, yet true rigors do not occur when the temperature rises slowly, nor when it falls rapidly.

A consideration of the conditions under which rigors occur enables us to distinguish two main classes of agents causing them: (1) Those in which we must assume the presence of certain poisons in the general circulation—*toxic rigors*; and (2) those in which the rigor appears to depend on the existence of some peripheral mechanical irritant which acts on the sensory nerves, chiefly on those distributed to the mucous membranes; these may be distinguished as *sensory or algæic rigors*.

(1) *Toxic rigors.*—It is probable that several toxic agents are capable of giving rise to rigors, since rigors are common to so many different acute specific diseases. The toxic agents we assume to be the cause of these rigors may be elaborated within the body itself, as in the case of the virus of an acute specific disease. In the case of malaria the occurrence of rigors corresponds to the stage of sporulation of the parasite in the blood (see MALARIAL DISEASE). When rigors occur in the course of diseases attended by suppuration the rigors do not always coincide with the formation of fresh foci of pus. Repeated rigors may occur with a single focus of suppuration. In those cases where transfusion with normal saline solution has been followed by rigors it is probable that some chemical

substances formed within the body are dissolved and carried to the central nervous system by the bloodstream, and in the cases where rigors have followed the introduction of antitoxin into the body a similar sequence of events may likewise be assumed.

(2) *Sensory rigors*.—In some healthy individuals a sudden exposure of the skin to cold, or the mere passage of urine over the mucous membrane of the urethra, is accompanied by a transient shiver representing in miniature the clonic muscular contractions of a rigor.

As the result of more intense stimulation of the nerves of the mucous membrane we produce phenomena approximating a true rigor. Thus, directly after the passage of a catheter in some people a marked shiver may occur, or there may be a definite rigor, with rise of temperature and the other symptoms of a true rigor, occurring a few hours after interference with the urinary passages. A condition analogous to this form of rigor is not infrequently observed soon after the act of parturition, and has no grave significance. Differing from these in the fact that they are attended by severe pain are the rigors due to the presence of urinary or biliary calculi, and occasionally of foreign bodies or fecal masses in the intestine. The rigors which occur under these conditions are probably due either to the reflex action of the nervous system in response to a painful impression, or to toxic agents introduced at the site of mechanical irritation or derived from the tissues which have been injured. If this second view be correct, all causes of rigors may be included in one class—the toxic agents. That this is unlikely is shown by the fact that in the case of rigors following the passage of a catheter or after injuries to the urethra, the rigors may actually occur before the catheter has been withdrawn, or while urine is first passing over the urethral lesion, and before time has been given for the absorption of any toxic material. In many cases of rigors accompanying the passage of calculi, there has been found no evidence of laceration, ulceration, or other injury to the urinary or biliary passages, and the frequency of intestinal ulcerations, and the rarity of rigors traceable to an intestinal lesion, would all seem to negative the view that such rigors were due to a toxic absorption.

Although rigors may be produced as the result of toxic or of sensory stimuli, their occurrence depends very largely on the personal susceptibility of the individual. In children rigors are rare because their place is taken by convulsions; and in epileptics the acute specific diseases are often ushered in by a series of epileptic attacks in place of a rigor.

SIGNIFICANCE OF RIGORS.—Rigors occurring at the onset of one of the *acute specific diseases* have little or no significance. They commonly occur at the onset of acute pneumonia, variola, scarlet fever, and erysipelas; but they may also usher in the onset of other acute *inflammatory processes* as in pleurisy, pericarditis, and peritonitis. There is nothing distinctive in the rigors occurring in these different diseases, though in some, as in acute pneumonia, there is usually one severe rigor, while in others, as in pleurisy, a series of slighter rigors occurs; yet in neither case are they always present, nor can a series of slight rigors serve to negative the diagnosis of acute pneumonia. A rigor occurring in the course of an acute specific disease may have a much graver significance: it may indicate

the formation of some focus of suppuration or the occurrence of some serious complication such as the supervention of pericarditis in the course of pneumonia. In *enteric fever* rigors may be met with either at the onset of the disease or during its course. The initial rigors are rare. Occurring in the course of the disease rigors may indicate the formation of foci of suppuration, or may be the result of intestinal irritation, of which constipation is the most common cause. Osler asserts that rigors may be due in some cases to the too free use of the antipyretics derived from the coal-tar series. Abercrombie has pointed out that rigors in enteric fever, occurring independently of perforation or peritonitis, are commonest in women, and may sometimes be traced to emotional causes.

At the commencement of an attack of *paroxysmal hæmoglobinuria* slight rigors with a rise of temperature may take place. Rigors may accompany the formation of *thrombi* in the veins, or may occur with the formation of an *infarct*, even if this be not infective. In *pyæmia* the rigor generally occurs by day, and there is rarely more than one in the twenty-four hours. Although there may be large numbers of suppurating foci, there is no relationship between them and the number of rigors. In *infective endocarditis* rigors are not uncommon. They do not of necessity indicate that there is a suppurating infarct. Rigors are met with in cases where suppuration occurs in connection with veins, especially in the cerebral, hepatic and uterine veins.

The formation of *abscesses* in the substance of any organ, and the occurrence of *infective inflammation* of a mucous membrane, as in pyelitis, may be accompanied by severe rigors. In cases of *poisoned wounds*, such as may result from infection while making a *post-mortem* examination, rigors may occur without any evidence of the formation of any focus of suppuration.

TREATMENT.—Rigors themselves do not call for any treatment apart from that of the disease causing them. With the object of making the patient as comfortable as possible during the stage of shivering, he should be put to bed and kept as warm as possible, being wrapped in warm blankets, with foot-warmers, and hot drinks containing a diffusible stimulant, such as alcohol, may be given. In this stage, when all the cutaneous blood-vessels are in a condition of spasmodic contraction, such vaso-dilators as nitrite of amyl, nitroglycerin, tetrannitrate of erythrol, and nitrite of sodium have been employed. During the hot stage tepid sponging, or even cold sponging, may prove grateful; and barley-water or soda-water with fresh lemon in it will be found to give welcome relief to the patient's symptoms. In malarial rigors quinine should be given hypodermically. *See* MALARIAL DISEASE.

ARTHUR F. VOELCKER.

RIGOR MORTIS (Lat.: The stiffness of death). **SYNON.:** Fr. *Rigidité Cadavérique*; Ger. *Todtenstarre*.—The stiffening of the muscles after death, due to coagulation of their plasma. *See* DEATH, Signs of.

RINGWORM.—*See* TINEA TRICOPHYTINA.

RIOLO, in Italy, near Bologna.—Chalybeate, sulphurous saline waters. *See* MINERAL WATERS.

RIPPOLDSAU, in the Black Forest, Germany.—Mixed iron waters. See MINERAL WATERS.

RISUS SARDONICUS or **SARDONIUS** (*risus*, a laugh; and *sardonius*, connected with, or caused by, the herb *sardonia* or *sardoa*, that is, belonging to Sardinia).—A peculiar expression of the face, in which the features are distorted by spasm of the muscles, so as to present the appearance of a painful grin or laugh. It is usually observed in tetanus. See TETANUS.

RITTER'S DISEASE.—A synonym' for *Der-matitis exfoliativa neonatorum*.—See FÆTUS, Diseases of, p. 551; and PITYRIASIS RUBRA.

ROBURITE, Poisoning by.—See DYNAMITE.

RODENT ULCER.—SYNON.: Fr. *Cancroïde*; Ger. *Epithelialkrebs*.—Rodent ulcer must be recognised as a kind of tumour; but its exact position among other forms of new-growth is still not absolutely decided. Hence, in this work a special article is devoted to its consideration. Most authors agree in classing it among the epitheliomata—a conclusion which the accumulated evidence of recent years tends to make practically certain.

CLINICAL CHARACTERS.—*Naked-eye appearances*.—A distinguishing feature of most rodent ulcers is the fact that ulceration follows *pari passu* with new-growth, the result being that, as in the case of lupus, instead of the formation of a swelling or tumour, an actual diminution of the size of the part occurs. Another characteristic of the disease is, that while it often makes its appearance at a period of life which might be considered early for an epithelioma, it runs a course of extreme chronicity, and rarely, if ever, affects the lymphatic glands. Many cases last for twenty or thirty years, interfering but little with the general health, and at times in part undergoing a process of feeble cicatrization. In one case observed by the writer the cicatrix was rather extensive, and had stood the test of a considerable period of time.

Rodent ulcer begins as a pimple, usually on the upper part of the face, and most frequently on the side of the nose or about the eye. It is occasionally met with in other parts of the face and of the body. After this has remained quiescent for a long time, perhaps years, ulceration usually occurs, and continues to spread with great slowness, involving in its course every structure that it meets. In rare cases, however, there is no ulceration, the characteristic margin of the growth surrounding a thin uniformly smooth cicatrix. Thus in time huge caverns are excavated in the face; the eye-ball may be destroyed; the nose and upper jaw may disappear; and not infrequently, if the disease reach the forehead, the dura mater is exposed, and the brain is seen pulsating at the bottom of the cavity. The appearance of the ulcer is characteristic: the surface is glistening, and is covered with very imperfect granulations; it has an uneven floor, and is mottled with yellow and red; the margin is very slightly raised, and somewhat indurated, has a purplish-pink colour, and is often considerably undermined. The discharge is thin and purulent. Capillary hæmorrhage not infrequently occurs, but more severe bleeding is rare. A section through the edge shows the narrow margin of new-growth,

in which alone the characteristic structure is to be made out. Death may occur from old age or other causes independent of the disease; from an attack of erysipelas or meningitis, or from marasmus induced by the constant worry and discharge.

MICROSCOPICAL APPEARANCES AND PATHOLOGY.—Many tumours which approach somewhat nearly the condition above described will be found on examination to possess the structure of a lobular epithelioma (see CANCER); but the most typical ones will usually exhibit something like the appearance represented in Plate IV. fig. 3, p. 244. Beneath the epidermis, and embedded in a varying amount of stroma consisting of more or less well-developed fibrous tissue, are large, roundish, and irregular masses of densely packed epithelial cells of small size, the circumferential ones taking an oval shape, while the deeper ones are circular. There is, as a rule, no tendency to the formation of globes; but, in some cases which have run a typical course, imperfect nests have been found. The so-called *prickle-cells* are, as far as the writer has observed, never seen. The cells are smaller than those usually met with in an epithelioma, and suggest the origin of the growth from the sweat-glands, a view which is favoured by the fact that the epithelial masses occasionally assume a more or less distinctly tubular arrangement. Strenuous advocates are found in support of, and in opposition to, this theory of the primary source of rodent ulcer, and the same may be said of other hypotheses, such as that it starts from the hair-follicles or the sebaceous glands; but, in default of stronger evidence than is at present forthcoming, it would be unwise to dogmatise upon the question.

PROGNOSIS.—The prognosis in a case of rodent ulcer may be implied from what has been said of its clinical features, course, and terminations.

TREATMENT.—The obvious treatment is free removal by the knife in the early stages. The method of scraping with a sharp spoon, which is not infrequently adopted, is not so likely to reach the limits of the disease, and does not seem to have sufficient theoretical or practical evidence to support it. C. H. Moore was a strong supporter of the plan of removing even very large ulcers; he was in the habit of proceeding with the knife as far as prudence would allow, and applying chloride-of-zinc paste to any parts it was considered unsafe to cut away. This treatment in his hands and in the hands of other surgeons has been followed by very marked success. R. J. GODLEE.

ROHITSCH, in Styria (Austria).—Acidulated, sulphated waters. See MINERAL WATERS.

ROISDORF, in Germany.—Mixed alkaline table-waters. See MINERAL WATERS.

ROMBERG'S SIGN.—An inability to stand steadily when the feet are close together and the eyes shut. See TABES DORSALIS.

ROME, Central Italy.—Moderately warm, moist, fairly calm winter climate. Mean winter temperature, 45° to 50° F. The air is damper than that of the Riviera. See CLIMATE, Treatment of Disease by.

ROMEGNO, in the Val Sugana, in the Austrian Tyrol.—Arsenical sulphate-of-iron waters. See MINERAL WATERS.

RÖMERBAD, in Styria.—Thermal waters.
see MINERAL WATERS.

RÖNTGEN or X-RAYS.—In December 1895 Professor Röntgen announced in a communication to the Würzburg Physico-Medical Society the discovery of a new form of radiation. The first paragraph of his paper is as follows: 'If we pass a discharge from a large induction-coil through a tiffiori, or a sufficiently exhausted Leonard's or Crookes', or other similar apparatus, and cover the tube with a somewhat closely fitting mantle of thin black cardboard, we observe that a paper screen covered with crystals of barium platino-cyanide lights up brilliantly, and fluoresces equally well, whether the treated side or the other is turned towards the discharge tube; fluorescence is still observable 2 metres away from the tube. It is easy to convince one's self that the cause of the fluorescence is the discharge from the apparatus and nothing else.'

So thoroughly did Röntgen investigate the properties of these new and unknown rays that his original communication contains almost all that is even yet known about them. He called them the X-rays.' In a Crookes' tube after a certain degree of exhaustion, wherever the cathodal rays strike solid matter, these X-rays originate. In the well-known X-rays tubes the cathode or negative terminal is concave, and the cathodal rays coming from it converge to a point or focus. At or near this position is placed the positive terminal (usually of platinum) called the anode. Thus the cathodal stream impinges at its narrowest part on the anode, and in this manner the X-rays originate from a comparatively small surface or point, and so can cast sharp shadows of objects upon a screen or photographic plate. The X-rays have the following properties:

1. They proceed in a straight line—they cannot be refracted or diffracted, and can only be reflected to a small extent, and then as a *scattered* reflection.
2. Ordinary matter is opaque to them in proportion to the atomic weights of its constituents. If equal volumes of all the elements were taken the lightest would allow most of the X-rays to pass through, and so on in the order of the atomic weights. It therefore follows that our own bodies, and all organic matter, are comparatively transparent to X-rays.
3. They have the power of discharging electrified bodies (this is also the case with violet rays).
4. They act on a photographic plate like light; it seems probable that they are due to disturbances in the ether similar to the ordinary waves producing the visible spectrum, only of extremely short wavelength.
5. They cause certain crystals to fluoresce. The best for that purpose are those of platino-cyanide of barium and platino-cyanide of potassium. Tungstate of lime and other substances fluoresce less brilliantly. The usual screens in the market are of cardboard spread over with crystals of the platino-cyanide of barium.

The great practical importance of these rays is their utility in medicine and surgery. So far they seem to serve no useful purpose in any other domain.

The apparatus necessary for the production of X-rays suitable for medical and surgical purposes is

very varied, and here only a very general description can be given.

Induction-coil.—For all-round work a coil giving a ten-inch spark is sufficient.

Current.—From 12 volts (which is given by an accumulator of 6 cells) up to 100 or 200 volts from the mains can be used, depending upon the kind of interrupter employed.

Interrupter.—The electrolytic or Wehnelt is the most effective, but is unfortunately somewhat uncertain, and is apt to destroy the tubes. The mercury-jet break is good, and a rotatory mercury-break, with a blade on an inclined shaft, gives excellent results, and is completely under control, giving the most effective output from the coil according to the speed of rotation with any voltage. A small sliding resistance adjusts the speed of rotation to suit the voltage.

Tube.—There are a good many good tubes in the market. The chief essentials are: (1) that the X-rays should come from a fine point on the anode; and (2) that the tube should possess some arrangement for reducing the vacuum if it gets too high. It is necessary to have several tubes, some with a high vacuum for use in deep structures, such as the hip-joint, and some with a low vacuum for examining the extremities and for therapeutic purposes.

It is advisable to have the whole tube surrounded by some non-conducting material opaque to X-rays, with an opening in it. The opening should be situated opposite the anode at a point where the fluorescent screen has its maximum brilliance when held close up against the glass. The object of this covering is to cut off stray X-rays from the glass of the bulb which fog the photographic plate and prevent a sharp image. The definition is thus much improved. Another advantage is that the operator can keep out of this diverging cone of X-rays, and so avoid the injuries resulting from too long and continuous exposure to the radiation from the tube. The most effectual way of covering a tube is to bury it in red-lead in a wooden box or porcelain or glass vessel and scoop away as much as is necessary at the right place, and with a small cylinder of paper prevent the 'banked-up' powder from falling in. It is more convenient to place it in a box which has been lined by about half an inch thickness of a mixture of red-lead and the white-lead of the shops—which hardens in time and yet is not brittle. The tube can be fastened to the lid by elastic bands, and an opening in it will act as a stop or diaphragm. If desired, smaller 'stops' can be dropped into this one and so a very narrow cone of X-rays obtained giving definition over that area correspondingly sharper and with much less general fog. This method of working the Crookes' tube will be found to be of great practical advantage in dealing with deep structures, and especially with cases of stone in the kidney, bladder, or ureter.

Screen.—The most useful is made by coating a piece of cardboard with crystals of platino-cyanide of barium.

Photographic Plates.—It is important that these should have a *thick* coating of emulsion. This will render short exposures effective, because X-rays pass so readily through the emulsion that, the thicker this is, the more distinct is the result. The support may be glass or celluloid or paper; the material does not seem to affect the 'speed' to any extent.

Röntgen Rays in Surgery.—At first the mere fact that the bones, bullets, or a fragment of needle could be revealed was so astonishing that for a time surgeons were quite content to get a single photograph, and considered it a valuable aid. As the work extended, and as operations based upon such photographs followed, it became evident that something more than a single picture was required. A moment's consideration will show why this is so. The X-ray photograph is not taken under the ordinary conditions with camera and lens: it is simply composed of shadows. Lay a large piece of white paper upon a table in a darkened room, place upon it any object or objects; hold above them a lighted taper or candle at about the distance an X-ray tube would be placed, so that the shadows will fall on the paper; and observe the shadows cast by the light from the taper—what a variety of different shadow-pictures can be produced by moving the light to different positions. The only real difference between such a picture and one produced by the X-rays is that these rays get through many of the objects themselves, and then not only cast a shadow of their outline but even of their very interior structure—e.g. the bones in good skiagrams have not only their outlines shown but also the cancellous structure. The desire to have these skiagrams properly interpreted led to methods of *localisation* being devised. Of these there are many. The writer described a method of precise localisation by means of Röntgen Rays in the *British Medical Journal* (January 1, 1898, and December 3, 1898) which is simple and extremely accurate—so much so that it has been used to locate minute foreign bodies in eyeballs and orbit with most satisfactory results. It is based on geometry. The three co-ordinates of any point can be obtained by measurement only—no calculations being required. It has the further advantage that the two skiagrams are stereoscopic and can be seen in relief, and the position of the bones, bullet, &c., seen in their true relations.

Stereoscopic Skiagraphy.—It appears likely that this method, which is at once simple and effective, will replace all the more intricate and troublesome methods for localisation (except in ophthalmic cases). The tube is first placed in the usual way—as if a single picture only was to be taken. After this is taken, the first plate is replaced by a fresh one, and the tube is displaced six centimetres (the average distance between our eyes when moderately converging) to one side, and another photograph is taken. The resulting negatives on a casual inspection seem very much alike, but when viewed in a Wheatstone stereoscope they combine and produce a single image of extraordinary brilliance; this picture is seen to possess the third dimension, viz. depth, and a most reliable and realistic image results. Of course it is absolutely essential that the observer should himself possess correct binocular vision.

The point of importance in taking these pictures is that when the tube is displaced, an imaginary straight line, connecting the two positions of the tube shall be parallel to one edge of the photographic plates. In order to ensure this there are two plans. (a) If the tube is placed above the patient, then the part to be skiagraphed must rest on a piece of stretched parchment (such as is used in the best drums), and a wire must be stretched along one side near and parallel to the edge of the

parchment, and the bar along which the tube-holder slides must be adjusted to be parallel to this wire. The photographic plate protected in the usual way in black-paper wrapping or envelopes is placed beneath the parchment, so that the plates can be changed without disturbing the position of the patient (a most essential point in order to produce correct stereoscopic pictures). The wire leaves a white line on the negatives, and this of course comes out a black line on the prints. These are then cut to the black line and mounted accordingly. They can then be viewed in a Wheatstone reflecting stereoscope, or for those who have acquired the power they can be viewed by converging the eyes upon the negatives (immediately after development), or upon the prints, and seen as a single image in striking and reliable relief.

The apparatus need not be complicated. The parchment should be stretched over a frame large enough to admit the largest photographic plate that is likely to be used, protected in the usual way in paper envelopes. This simple arrangement can be applied to a couch, or made separately in the form of a stool. It enables the part to rest on the parchment undisturbed, while a fresh plate can be exchanged for the first one. Piano-wires can be stretched across for purposes of localisation.

(b) A still more simple plan is to have a couch like a camp-bed, with canvas or fine strong twill. The tube can then be placed on a holder beneath, or in a box with red-lead, as described above, and can be arranged to slide easily up and down, or across on wooden bars provided for the purpose. In this way all the desired positions for the tube can be obtained. The patient can lie on this couch and not be disturbed, while with a fluorescent screen the surgeon explores the whole body. For photographing, the tube is first placed below the part, and a photographic plate is laid upon the patient; one edge of the plate is placed parallel to the transverse edge of the tube-holder, so that when the tube is displaced to one side (to get stereoscopic skiagrams) it moves in a plane parallel to the edge of the photographic plate. The advantages of a couch like this are numerous. In the first place the patient is not perturbed by the tube hanging over him. The surgeon can by means of the screen decide exactly what part he wishes to photograph, and can place his plate there. He can even watch how the tube is really working by looking through the plate while the photograph is actually being taken. When using large plates there is no risk of breakage or having the plate marked by pressure. Altogether the plan seems the best and simplest of all. Further, a couch of this kind can be used as an operating one, and without any difficulty if required during the operation the surgeon could examine the patient with the fluorescent screen.

Stereoscopic Fluoroscope.—This instrument enables an observer to see the X-ray shadows upon the fluorescent screen *in relief*. In addition to the marked advantage of seeing through the patient in this way, if an object opaque to X-rays, such as a knife, needle, or probe, be employed, it can be used to reach or touch any object visible in the screen-picture. At present the difficulty in the use of this instrument is to be able to get the patient conveniently into the necessary position between the two tubes and the eye-holes in the revolving shutter.

It is not necessary to detail the apparatus fully here; suffice it to say that there are two tubes

placed side by side as close to each other as possible. These by a suitable break and commutator in the secondary circuit of an induction-coil are made to work alternately. Opposite these tubes are two eye-holes for the observer, with a revolving eclipsing shutter, so that each eye only sees the shadow of the one tube, and this taking place alternately, and repeated ten times or more per second, the shadows of an object on the interposed screen, that each of the observer's eyes receives, become a continuous impression, and these are fused by the brain in the usual way, and a 'Stereoscopic shadow' is seen. The value of this instrument will be considerable in the examination of the thorax, where the movements during respiration lessen the definition in photographs. In the examination of fractures, dislocations, or lodged foreign bodies, the Stereoscopic fluoroscope will prove of great practical value, as the shadows of the parts are seen standing out in solid relief, in relatively correct positions. And still more practical is the fact that any object that is seen in this way can be touched by the observer or he use a metal probe, or knife or forceps, since the shadow of the instrument is seen in relief and seen under the same optical conditions as the part of the patient under examination.

We may now sum up the advantages of the Röntgen Rays in surgery, with hints as to procedure in special cases.

1. *Normal.*—It is of interest to have skiagrams of normal parts, as it enables the structure of the body to be seen under new conditions. In young patients an accurate knowledge of the amount of ossification can be acquired.

2. *Fractures and Dislocations.*—Diagnosis can be made painlessly, even when parts are much swollen and painful.

3. *Lodgment of foreign bodies.*—All the metals and glass can be detected.

4. *Calculi.*—In the bladder, ureter or kidney calculi can be detected, provided the patient be not too stout, or the calculus too small. The usual law of the atomic weights, deciding the opacity of a body to X-rays, comes into play here, and given equal volume, the calculus with the greatest atomic weight will be the most opaque, the most easily differentiated from the surrounding tissues, and therefore the most easily detected. Another arrangement that helps towards success in these difficult cases is to regulate the patient's breathing while the photograph is being taken. If possible arrange to turn on the current, and only to expose while the patient holds his breath in the same phase. By this procedure the kidney, which is displaced during the period of breathing, will occupy the same position while the photograph is being taken.

There are automatic arrangements made for this purpose, but these are necessarily complicated. A simple method is to have a long light piece of wood fastened to an elastic band put round the patient's chest; a pin put through the strip of wood is supported from a stand. If a scale is placed near the other end of this indicator, after a few trials the patient will be able to regulate his breathing, so as to bring the point of the indicator always to the same position on the scale, and keep it there by holding his breath for 20 to 40 or more seconds if necessary.

Ophthalmic Surgery.—X-rays have proved of extreme value in this branch of surgery. The writer

has detected and located by their means steel, iron, brass, copper, silver, lead, and glass in the eye and orbit. The method to be followed in precisely localising some of these particles, which are extremely small, is somewhat difficult, but if due pains are taken the result is always correct.

Teeth.—The best plan is to wrap a small piece of a photographic film in black needle-paper, and then enclose it in a piece of gutta-percha tissue, which can be sealed with chloroform. This can then be introduced into the patient's mouth, and held closely up against the part to be skiagraphed. The Crookes' tube is then placed in a suitable position. The patient must have his head supported to avoid movement.

Röntgen Rays in Medicine.—*A. Diagnostic.* Gradual progress is being made, but meantime the aid that the X-rays afford the physician is comparatively limited.

Aneurysm of the aorta in the chest can be seen on the fluorescent screen and photographed. It has a sharply defined border, projects from the general mediastinal shadow, and can be seen to pulsate.

Tumours in the chest are shown, but no clue to their nature is given, they simply cast a darker shadow than lung or pleural fluid. As a rule pleural fluid gives a much less distinct edge than a mediastinal or pulmonary growth.

Abscess in the lung and tubercular lesions are less transparent than the normal lung, except in the cases of large cavities, easily recognisable by other means.

Abscess on the surface of the liver has been detected.

The movements of the diaphragm on the two sides can be studied and contrasted when watched on the screen.

The abdomen is very difficult to examine by means of X-rays. With the exception of gaseous distension of the stomach, renal calculi, and metallic foreign bodies which can be detected, little information can be obtained.

B. *Therapeutic.*—Accidentally it was discovered that sometimes when a patient was long exposed to X-rays, a more or less destructive inflammation of the skin nearest the tube took place, and in severe cases extensive and deep sloughs occurred. At the present moment it is not quite certain if the X-rays are responsible for these injuries, or whether it may be due to some accompanying radiations. The radiations from a Crookes' tube are necessarily complex, and at present these have not been satisfactorily differentiated. It naturally came about that this destructive action was used to try and destroy the germs in certain diseases, just as one ordinarily tries to cure lupus by scraping, or by the action of the cautery, or caustics. The results in lupus have been decidedly encouraging. An X-ray tube with a low vacuum, or, more correctly, a tube which has a very short alternative spark-gap resistance, seems to act better than a 'high' or, as the Germans express it, a 'hard' tube. Repeated exposures have to be given, and the tube need not have too much energy passed through it, so as to heat it. In course of time favourable results ensue. It is not yet established, however, that this method is markedly superior to some of the usual methods of dealing with lupus. It is said that rodent ulcers have been cured by means of X-rays.

Depilatory effect of X-rays.—In several cases successful results have followed exposure to X-rays,

but one must be careful to ascertain the limits of safety, that is, just enough exposure to destroy the hairs, and yet cause no destruction of the skin itself. It seems that as yet there is no exact certainty about this procedure, as in some cases successful depilatory action results, while in others it is disappointing. In most cases the growth of hair is not arrested permanently, but possibly repeated and well-timed application of the radiations from the X-ray tube might prevent growth recurring. The tubes most useful in producing destructive changes are of low vacuum. The exposures have to be long and repeated frequently. *See also* p. 376.

That the X-rays have been of immense value to medical science is unquestionable. It is only a short time since they have been discovered, and we are yet only imperfectly realising their possibilities.

JAMES MACKENZIE DAVIDSON.

ROSACEA.—*See* ACNE ROSACEA.

ROSEOLA.—Fr. *Roséole*; Ger. *Roseola*.—This is a term more used in the past than the present for patchy erythema, in which the tint is less bright than usual, a trivial distinction which scarcely justifies its continued existence. If retained at all, it would be best restricted to the symptomatic eruptions seen in various serious constitutional conditions, as in the early stage of syphilis, in leprosy, and less constantly in variola before the more characteristic rash, and in vaccinia, cholera, diphtheria, malaria, spinal meningitis, and enteric fever.

A so-called idiopathic roseola is sometimes seen in young children, chiefly in the spring and autumn—a disordered alimentary canal, especially during dentition, being the most common ætiological factor. In such cases there may be an elevation of temperature of several degrees, with other febrile symptoms, but without catarrh, and lasting for a few hours only as a rule. The eruption, which is rose red, varies much in shape. There may be patches the size of the end of the finger, faint papules, circles, or gyrate outlines. These come out in any part of the body or nearly all over, fading perhaps at one part and appearing at another, the whole process lasting a few days. *See* FEBRICULA.

TREATMENT.—But little treatment is required for so transitory an affection, which is chiefly interesting from a diagnostic point of view. Any disorder of the alimentary canal should be corrected by a mild mercurial purge; and every likely cause of irritation, either in the gums or elsewhere, should be searched for and removed if possible.

The roseola symptomatic of the grave disorders enumerated requires no special treatment, the serious constitutional condition claiming the whole attention.

H. RADCLIFFE CROCKER.

ROSE-RASH.—A popular name for roseola. *See* ROSEOLA.

RÖTHELN.—The German synonym for rubella; frequently employed by English practitioners. *See* RUBELLA.

ROUND-WORMS.—*See* ENTOZOA.

ROYAT, in France.—Muricated alkaline waters. *See* MINERAL WATERS.

RUBBING-SOUND.—A synonym for friction-sound. *See* PHYSICAL EXAMINATION.

RUBEFACIENTS (*ruber*, red; and *facio*, I make).—A class of counter-irritants which produce simple redness of the skin. *See* COUNTER-IRRITANTS.

RUBELLA.—SYNON.: German Measles; Fr. *Rubéole*; Ger. *Rötheln*.

DEFINITION.—A specific infectious eruptive fever. The rash appears on or after the first day of the illness, beginning on the face in rose-red spots, extending next day to the body and limbs; it subsides with the fever on the third day; and is not preceded or accompanied by catarrh, but is associated with enlargement of the lymphatic glands in the neck.

ÆTIOLOGY.—Propagated by contagion, rubella occurs in epidemics, often of limited extent, commonest in spring and early summer. The period of incubation is mostly over a fortnight, the extremes being from ten to twenty-one days. Hence arises a difficulty in tracing the source of infection, increased by the slight and transient symptoms which permit the illness to be entirely overlooked, the patient consequently mixing freely with others. One attack is not preventive of a recurrence. Very young infants seem less susceptible than older children; a child at the breast has been known to escape when the mother and other children in the family have been attacked; adults not infrequently suffer; sex makes no difference. A recrudescence of the rash from the third to the sixth day is sometimes seen.

SYMPTOMS.—Slight headache or giddiness is felt, with aching of the back or limbs, chilliness, and a little sore-throat, for twelve hours or a day before the rash appears. Very often the rash is first seen with surprise, as the feeling of illness has passed or may have escaped notice. Enlargement of the superficial cervical glands, especially in the posterior triangles of the neck, is an early sign, often developing a week before the rash appears, and most marked in children. There is redness of the fauces and uvula, less mottled than in measles, not so intense as in scarlet fever; the tonsils are a little swollen; there is no ulceration. Sometimes an odour, as in measles, attends the rash. The eyes are suffused, but there is little or no coryza; the lids are somewhat swollen and irritable; the face and the cheeks may be flushed before the appearance of the spots. The rash is first seen behind the ears, around the mouth and about the nostrils; it consists of bright red, raised, rounded spots, with clear skin between them, but they soon coalesce; not grouped as in measles, the spots are more prominent than in scarlet fever, and there is not the finely diffused redness of the neck and chest observed in that disease. Moreover, the rash is already fading from the face and upper part of the body while extending to the limbs, so that it is less intense on the third day. It leaves some itching, or a very fleeting yellowish tinge, but no discoloured mottling of the skin, and desquamation varying in amount with the extent and severity of the eruption. However little illness is felt at the beginning, a rise of temperature may commence with, or just before, the rash. It may reach 102°–103°, or be only 2° F. above the normal. With rest in bed this may fall one degree by the end of the second day, but is evenly maintained as the eruption proceeds, and subsides with it on the third day. Not infrequently, however, there is no rise of temperature at all. In rare cases there may be a relapse during the second week.

The urine is often high-coloured in the early part of the illness; but there is no albuminuria, nor has this ever been known to follow. In some few cases a transient complaint of sore-throat or of fatigue has been made a week before the rash; or epistaxis has occurred. In delicate children bronchitis, broncho-pneumonia or pleuro-pneumonia are sometimes met with as complications, as also are laryngitis, pharyngitis and middle-ear troubles; enlarged glands and tuberculosis have been found as sequelæ in such cases.

DIAGNOSIS.—The rash may very closely resemble that of measles, but it comes out on the first day instead of the fourth; the catarrhal symptoms, if any, are slight; the fever is slight and quite disproportionate to the amount of the rash, and the rash begins to fade within twenty-four hours. The enlargement of the cervical lymphatic glands is characteristic, and is not seen in measles. In scarlet fever the rash is a uniform punctate redness accompanied by definite throat-symptoms, and often preceded by vomiting; the onset is always sudden and often with serious symptoms. By the fourth day there should be the characteristic strawberry tongue, and the gland behind the angle of the jaw is the only one enlarged. The quarantine-period after a child has been exposed to infection before he is allowed to go back to school or mix with other children should be twenty days, provided that disinfection has been carried out at the commencement of the quarantine. *See* INCUBATION.

PATHOLOGY.—No specific bacillus has as yet been identified, but the existence of such an organism cannot be doubted.

PROGNOSIS.—This is invariably good, except in the case of very delicate children or where the disease precedes or follows on one of the other exanthemata.

TREATMENT.—No special treatment is called for; confinement to the house for a week, and perhaps to bed for a day or so, is almost all that is required in rubella. A patient may be regarded as free from infection in not less than ten days from the appearance of the eruption.

WILLIAM SQUIRE.

JOHN ABERCROMBIE.

RUBEOLA.—A synonym for measles. *See* MEASLES.

RUPIA (ῥύπος, dirt).—Rupia is a term applied to thick conical crusts formed in layers over ulcers of the skin. The word is now only used to designate a very characteristic syphilitic.

The lesion consists of a superficial syphilitic infiltration of the cutis, accompanied by the exudation of serum, the elevation of the overlying epithelium, and the formation of a more or less perfect bulla. Its contents, at first clear or blood-stained, rapidly become purulent. The epidermis covering the lesion gives way, the contents escape and dry on the surface, forming a crust. In the meantime ulceration continues to spread peripherally beneath the crust, the ulcerated surface discharges and more crust is formed. By degrees, the stratified limpet-, or oyster-shell-like crust, which is the characteristic feature of the lesion, is formed. Its apex may be in the centre or towards the circumference depending on the direction in which the superficial ulcer spreads, and a well-formed crust may be half-an-inch thick, and as much as two inches in diameter. An areola of inflammatory redness of the skin is seen surrounding the crust, and on removing the crust the punched-out ulcer—deeper towards the centre—is easily perceptible.

An eruption of rupia is usually not profuse, and often a few only of the crusts present the typical appearance. Some of the lesions show crusts of ecchymatous character, while in other situations the imperfect bullæ, the ulcers, and the cicatrices of previous lesions may be noted. An eruption of rupia may continue for some months and varies in severity according to the condition of the patient; it may occur at almost any period of an attack of syphilis, after that of primary inoculation, and is perhaps most usually seen in cases of virulent infection in weakly or cachectic persons. The scars remaining from the lesions are at first pink, gradually assuming the characteristic white colour and pliant structure of the superficial syphilitic cicatrix; it may show a margin of surrounding pigmentation.

Treatment consists in the careful use of anti-syphilitic remedies, taking special care to sustain the general nutrition of the patient. The ulcers should be carefully treated by antiseptic remedies, so as to prevent them spreading rapidly, as sometimes happens in severe cases of syphilitic cachexia.

JAMES GALLOWAY.

RUPTURE (*rumpo*, I break).—**SYNON.** : Fr. *Ruptures*; Ger. *Risse*.—In addition to its ordinary meaning, this word is used in a popular sense as a synonym for hernia, which is spoken of as a *rupture*. *See* HERNIA.

S

SAIDSCHÜTZ, in Bohemia.—Sulphated mineral waters. *See* MINERAL WATERS.

ST. ANTHONY'S FIRE.—A popular synonym for erysipelas. *See* ERYSIPELAS.

ST. CATHERINE'S WELLS, in Ontario, Canada.—Iodo-bromated muriated saline waters. *See* MINERAL WATERS.

ST. GALMIER, in Loire, France.—A simple acidulated table-water. *See* MINERAL WATERS.

ST. GERVAIS, in Savoy.—Saline sulphurous waters, thermal and cold. *See* MINERAL WATERS.

ST. HONORÉ, in Nièvre, France.—Slightly thermal sulphurous and arsenical waters. *See* MINERAL WATERS.

ST. MORITZ, Upper Engadine, Switzerland.—A bracing mountain winter and summer climate. Baths; whey- and milk-cure. Altitude, 5620 feet. See CLIMATE, Treatment of Disease by; MINERAL WATERS; and PHTHISIS.

ST. PAUL'S.—A city in Minnesota, on the banks of the Mississippi, 1200 feet above sea-level. Well protected from winds. Climate cold, clear, dry, and calm; winters rather extreme. Mean annual temperature 44° F.; the mean daily range 19° ; rainfall 30 inches. A health-resort for phthical patients. See CLIMATE, Treatment of Disease by.

ST. SAUVEUR, in the French Pyrenees.—Sulphur-waters, slightly thermal. See MINERAL WATERS.

ST. VITUS'S DANCE.—A popular synonym for chorea. See CHOREA.

SALAAM-CONVULSIONS.—A form of convulsive-tic characterised by violent bowing movements. See ECSTASY.

SALINE SOLUTION, Infusion of.—DEFINITION.—The introduction into the vessels, either directly or indirectly, of a solution of chloride of sodium (6 per cent.) in water.

Wooldridge was the first to demonstrate that only one of the indications formerly regarded as of importance in transfusion was really essential, viz. the increase of the quantity of circulating fluid. In the severest cases of hæmorrhage there is always enough hæmoglobin left to sustain life, if only there is enough fluid in the vessels to enable the heart to ensure its circulation. Arbuthnot Lane, in 1891, successfully infused $3\frac{1}{2}$ pints of saline into the veins of a girl of 13, suffering from extreme collapse due to hæmorrhage after an operation for cleft palate. The method is now frequently employed and is of great value.

Landerer has proposed the addition of 3 to 5 per cent. of sugar, to render the fluid nutrient, to excite osmosis of the tissue-fluids into the vessels, and better to preserve the red corpuscles. Half an ounce of brandy or ten minims of Liq. Strychninæ is sometimes added with apparent advantage.

INDICATIONS.—1. Dangerous loss of blood from injury or disease. 2. Dangerous collapse from injury or disease. 3. Diabetic coma. 4. Acute delirious insanity with typhoid symptoms. In the first case there is not enough fluid in the vessels; in the second the vascular system, especially the great abdominal veins, is dilated, so that, relatively to the enlarged system, there is too little blood. In diabetic coma the dilution of the blood, and therefore of the poison, has not infrequently been followed by temporary return of consciousness, very rarely by recovery. Only a small number of cases of the fourth group have been published, but in some of these subsidence of symptoms followed the infusion.

METHODS.—1. Intravenous. 2. Subcutaneous. 3. Intra-peritoneal. 4. Rectal.

1. Intravenous Infusion.—The fluid is prepared in a jug or beaked measure by diluting a strong sterile solution of salt, or salt and sugar, with boiled water, so that there shall be one drachm of salt to each pint of water; but, in haste, table-salt may be used and hot water from the tap,

diluted, if necessary, with cold tap-water to bring it to a temperature of 98° – 100° . The writer has never seen harm arise from fluid so prepared. Meanwhile a vein is exposed, usually the largest in the region of the elbow; sometimes the internal saphenous near the ankle, or even the external jugular. The selected vein is cleared from its surroundings for three-quarters of an inch. A fine silk loop is passed behind it with forceps or aneurysm-needle and divided—one half being drawn to the proximal and the other to the distal angle of the wound. The latter thread is tied. A short distance above the knot, a bit of the front of the vein is seized with forceps, and a pointed flap—base upwards—is cut with scissors. The flap is raised by the forceps holding its point, and beneath it a boiled glass or metal nozzle is easily passed into the vein towards the heart. The first hitch of a reef-knot is now tied upon the vein and contained nozzle with the proximal thread.

Before being introduced into the vein, the nozzle should be connected by some four feet of boiled rubber-tubing with the sterilised barrel of a 4-ounce glass-syringe (failing this, with a glass or metal funnel). The saline fluid is poured into the barrel of the syringe and, the tube and nozzle being allowed to hang vertically, all air is expelled, and the apparatus is filled with saline-solution; a clip, close to the nozzle, is then allowed to close upon the tube.

The nozzle being in the vein, the syringe is held well up, and the clip removed; as the fluid runs into the vein, more is poured into the syringe, which should never be allowed to become empty. Two to six pints have been introduced; two to four form the usual quantity. As the nozzle is withdrawn, the hitch upon the silk ligature is tightened upon the vein and the reef-knot is completed. The skin-wound is closed by one or two stitches and a dressing of gauze and collodion.

DIFFICULTIES.—1. *Finding the Vein.*—Even in thin people, the superficial veins may hardly be visible. A ligature round the limb above the point at which it is desired to open the vein may be tried, and the limb should be allowed to hang down. If a suitable vein does not show quickly a short incision should be made at a likely place and the vein exposed.

2. *The Introduction of the Fluid.*—Should any difficulty be experienced in introducing the fluid, see that the vein-wall is not tightly pressed against the oblique opening of the nozzle, and that there is nothing compressing the vein between the nozzle and the heart. Hold the barrel of the syringe as high above the nozzle as possible and raise the limb somewhat; or insert the piston of the syringe and inject. Finally try another vein. The writer has failed with all these expedients—a fore-arm vein and the internal saphenous having been opened, the cause of the difficulty was not discovered. In such a case the external jugular should be tried, but ordinarily, a scar in the neck should be avoided.

Effects.—As the infusion is continued the pulse first becomes palpable at the wrist, and then falls in frequency as it increases in volume. The breathing improves, and the extreme pallor disappears. The infusion should be stopped when a satisfactory improvement has been effected, or when it is obvious that a good result cannot be obtained. It is said that hæmatin from destruction of red corpuscles, has sometimes been found in the urine a day or two

fter saline infusion ; and sometimes a rise of temperature has been attributed to it. Saline infusion has been repeated two to six times in cases of hemorrhage, with ultimate success.

2. Subcutaneous Infusion.—This method is easily carried out by inserting an aspirator-needle deeply into the subcutaneous tissue where this is plentiful and loose. The needle is connected by rubber tubing with a douche-can, raised 5 or 6 feet above the puncture. Howard Kelly prefers this method to the intravenous, though his objections to the latter seem to be chiefly theoretical. In bad cases he runs a needle behind each breast, and in twenty minutes he can thus introduce 700 to 1000 c.c. He has never seen any unpleasant effects. See p. 706.

3. Intraperitoneal Infusion.—In bad abdominal cases many surgeons are in the habit of giving 2 or 3 pints of warm saline solution in the peritoneal cavity, whence it is rapidly absorbed.

4. Rectal Infusion.—Half a pint or a pint of saline solution thrown into the rectum is often very useful in cases of moderate shock. It is rapidly absorbed, and half a pint more may be given in one or two hours. For this purpose the writer frequently substitutes saline beef-tea or peptonised milk-gruel for normal saline ; and to these brandy may be added. These large injections are usually retained, unless there is much vomiting. The anus should be supported by a pad of cotton-wool and by the pressure of the hand during straining ; and the foot of the bed should be well raised.

STANLEY BOYD.

SALINS, near Dôle, in France.—See MINERAL WATERS.

SALINS-MONTIER, in Savoy.—Muriated saline waters, with some iron, iodine, and arsenic. See MINERAL WATERS.

SALISBURY TREATMENT.—A name applied to a form of treatment for obesity, consisting in the limitation of the ingesta to animal food and hot water. See OBESITY.

SALIVARY CALCULUS.—See CONCRETIONS ; and MOUTH, Diseases of.

SALIVARY FISTULA.—See MOUTH, Diseases of.

SALIVARY GLANDS, Diseases of.—SYNON. : Fr. *Maladies des Glandes Salivaires* ; Ger. *Krankheiten der Speicheldrüsen*.

The salivary glands are six in number, distributed in three pairs, viz. the parotid, the sub-maxillary, and the sublingual. These glands and their ducts are liable to inflammation. Obstruction of the ducts, salivary calculus, ranula, and salivary fistula are described in the article MOUTH, Diseases of. All the glands are liable to a specific infective disease known as Cynanche parotidea or Mumps (see MUMPS). The parotid gland is also subject to Simple Parotitis and to Acute or Suppurative Parotitis.

Simple Parotitis.—SYNON. : Inflammation of the Salivary Glands ; Sialodochitis fibrinosa ; Fr. *Grenouillette aiguë*.—In all cases the inflammation enters from the mouth, where the causative factors are to be looked for. These may be obstruction of the mouth of the duct by a foreign body, by swelling

of the epithelium, or by some growth on the buccal mucous membrane. Any of the affections of the mouth (see MOUTH, Diseases of) may induce the condition, either by direct spread of inflammation or by the mechanical obstruction set up. In the sublingual gland it is frequently produced by salivary calculus. It is said to result from cold or injury. Simple inflammation may be limited to the ducts, or may extend to the gland.

The SYMPTOMS consist of a certain amount of constitutional reaction together with pain and swelling in the region of the parotid gland. This latter is found to be uniformly enlarged and elastic. In many cases there is no pain or discomfort further than the mechanical inconvenience ; occasionally there may be both pain and tenderness, though never very acute. The condition and appearances are variable, frequently becoming more marked after meals and decreasing at times.

The TREATMENT consists of attention to the causal conditions, and should chiefly be directed to maintaining the patency of the duct.

Acute Parotitis.—SYNON. : Suppurative Parotitis ; Symptomatic Parotitis ; Metastatic Parotitis.—The term Metastatic Parotitis is an unfortunate one as the condition is in no sense a metastasis, but is a complication arising in several different conditions. It is met with in various fevers—scarlet, enteric, rheumatic, and typhus. It also occurs with pyæmia, pneumonia, and phthisis. Occasionally it has been ascribed to gout. An interesting form is that which follows injuries or diseases of the abdominal or pelvic viscera, and is met with in gastric ulcer, peritonitis, dysentery, intestinal obstruction, and after ovariectomy and catheterisation.

Suppurative Parotitis may spread from the mouth along Stensen's duct, when suppuration occurs primarily within the tubules. When it occurs in the course of pyæmia or as a sequela of one of the exanthemata the pus may form in the interstitial tissue. Several glands may be affected simultaneously, but the inflammation is generally limited to the parotid and to one side. The gland becomes enlarged ; the skin over it is shiny, tense, and exceedingly painful. This is chiefly due to the firm fascia covering the gland. This is bound over it so firmly that the suppuration, which is so apt to ensue, cannot readily find its way to the surface, and may discharge into the external auditory meatus or the mouth, or burrow among the muscles of the neck, or even pass upwards and inwards towards the base of the skull. Under these circumstances the constitutional symptoms from septic absorption are generally severe. When it occurs in the course of an acute disease the subjective symptoms may not be so marked and the course of the complication is sometimes less evident. Probably the first indication of the parotitis is a little swelling about the lobe of the ear and some loss of the usual depression between the ramus of the lower jaw and the mastoid process, and its replacement by a more or less hard tumour. Pressure on this will elicit an expression of pain.

The inflammation may end in either resolution or suppuration. The former termination may be looked for if the swelling has formed slowly and during the convalescence from acute disease. When suppuration takes place it is revealed by the irregularly reddened appearance of the swelling, the detection of fluctuation, and the severity of the symptoms. Pyæmic symptoms are apt to supervene

owing to the retention and deep spread of pus, as well as to the fact that large veins and arteries pass through the parotid gland.

PATHOLOGY.—*Post-mortem* examinations show that the duct is always affected and that the lobules of the gland are more or less destroyed before the interstitial tissue is attacked. In this stage the gland appears as if studded with small suppurating islands. At a later period not only does the phlegmonous inflammation reach the interstitial tissue, but spreads to the neighbouring connective tissue enveloping the muscles, by which it is directed downwards even as low as the clavicle, or up to the base of the brain.

The pathogeny of the affection has been much discussed, but still remains uncertain. That it is due to microbic invasion is certain, but nothing has been settled as to the actual organism concerned nor as to how it gains access to the gland. There can be little doubt that its route is along the duct, and that the diminished resistance of the patient is a powerful contributing factor.

DIAGNOSIS.—Some lymphatic glands lie on the outer side of the parotid, and are not infrequently inflamed. This condition can be distinguished from parotitis by observing that these glands are superficial, and that the *socia parotidis* is not enlarged.

PROGNOSIS.—If this disease develops slowly and during convalescence the prognosis is favourable. On the other hand, its occurrence is of the gravest importance if it appears during the acute period of the primary disease. Trousseau remarks that it is an affection from which he has rarely seen enteric or other patients recover.

TREATMENT.—The treatment should be both prophylactic and symptomatic. In all cases of acute disease of an adynamic type particular attention must be given to the care and cleanliness of the teeth and mouth. When there is any suspicion of the parotid gland being affected, attention should be directed to the patency of Stensen's duct, along which, if necessary, a fine probe can be passed, while its orifice is well cleansed with warm alkaline lotions. In the early stages warm fomentations and poultices should be applied, and the pain may to some extent be relieved by the local use of belladonna. The patient's general condition will require increased care, particularly with regard to nourishment, stimulants, and tonics. As soon as pus has formed it should be evacuated. In doing this care must be taken to avoid injury to the facial nerve, and Hilton's method of operating may be advantageously employed.

Tumours of the Parotid Gland.—These may be (a) simple, or (b) malignant. A chronic enlargement may remain for some time after an attack of Mumps or inflammatory Parotitis.

The simple parotid tumours frequently originate in the superficial parts of the gland. They vary much in structure, and among those met with are cysts, fibromata, lipomata, angiofibromata, angio-lymphomata, and tumours of a mixed type. Cartilaginous elements are sometimes met with in these growths, probably due to the persistence of remnants of the embryonic Meckel's cartilage.

Malignant tumours of the parotid occur in the form of either sarcoma or epithelioma. A correct and early diagnosis is of importance, and as the treatment for both classes of tumour can only be operative, a work on surgery must be consulted.

ST. CLAIR THOMSON.

SALIVATION (*Salivatio*; *σάλισμός*).—**SYNON.** : Ptyalism; Fr. *Salivation*; Ger. *Speichelfluss*.—The term is given to an extraordinarily profuse flow of saliva, which is apt to occur under many various conditions. Ptyalism is therefore a symptom and cannot be regarded as a distinct affection, but it obtains a position as an independent disorder in the nomenclature of disease, and therefore requires a separate notice.

ÆTIOLOGY.—The causes of salivation are numerous. Mental conditions will sometimes produce an increased and persistent flow, just as fear will arrest the secretion. Any direct irritation of the mouth is apt to be associated with increased flow of saliva, and hence it is met with in dentition, aphtha, thrush, the various forms of ulceration, cancrum oris, glossitis, &c. It may be set up by reflex irritation in neuralgia and gastric affections, and is sometimes one of the most persistent complications of pregnancy. It is also said to be produced by irritating causes in the liver, spleen, and genital organs. It is met with in hydrophobia and small-pox. The free dribbling of saliva is a striking symptom of bulbar paralysis, but it is not settled whether there is in this disease actual increase of secretion or merely an escape of it through the open lips. This dribbling of saliva—which should be distinguished from salivation—is also met with in double facial paralysis, tonsillitis, mumps, typhus fever, tabes dorsalis, and in the insane. Among the drugs which give rise to ptyalism are iodide of potassium, arsenic, copper, and pilocarpine; but in actual practice all these various ætiological factors are rarely met with in comparison with the frequency with which mercury is found to be the cause. Salivation from mercury is much less frequent than formerly, and is rare in an acute form. There are some subjects with a peculiar idiosyncrasy for this drug, in whom a very small dose—1 gr. of calomel, or even less—will produce decided salivation. But apart from such cases only mild degrees of salivation are met with nowadays.

SYMPTOMS.—The symptoms of salivation are evident enough. The individual complains of the increased secretion in his mouth, interfering possibly with his speaking, swallowing, and even sleeping. When due to mercury the salivation may be preceded by a metallic or 'coppery' taste in the mouth, tenderness of the gums, and a disagreeable odour from the breath. In marked cases this last may be horribly fetid, while the gums become spongy, bleed readily, and recede from the teeth, which are loosened in their sockets. The tongue is large and flabby, the patient is constipated, and emaciation ensues. If the case goes still further there is ulceration of the gums and mouth, and the teeth may fall out. While these symptoms are developing the increase in the secretion of saliva has become so marked that it pours from the patient's mouth night and day. Tired of wiping it away, he may place some receptacle beneath his mouth into which he allows it to dribble during the day; at night he has to protect his pillows by cloths placed below his chin. Instead of the usual 1 to 2 quarts per day, the quantity secreted in twenty-four hours may amount to as much as 5 quarts. The specific gravity generally falls from 1006 to 1002 or 1001. In a well-marked case the condition may persist for two or three weeks.

TREATMENT.—When due to some nervous disorder, the increased secretion of saliva may be

checked by 10-drop doses of tincture of belladonna three times a day, or by the administration of liquor atropinæ $m\frac{1}{4}-\frac{1}{2}$ with liquor strychninæ $mijj-vj$. With careful prophylactic measures salivation from mercury can be avoided in a majority of cases, and need never be severe. Before starting treatment it is well, if possible, to see that the teeth and mouth are rendered as clean and healthy as possible. The dose of mercury should be small at first, and the patient frequently observed until it is seen how he tolerates it. A good precaution is to note his weight frequently and see that it does not decline. Great care should be taken of the mouth and teeth during treatment, and if there is any dental caries it is well to prohibit smoking and spirit-drinking. When any symptoms of salivation appear, the administration of mercury should be discontinued; the mouth should be frequently washed with lotions of chlorate of potassium, alum, myrrh, &c.; and the excretion of the drug from the system may be promoted by giving iodide of potassium, which fortunately generally benefits the condition for which the mercury was being administered. If due to iodide of potassium it is said that the symptoms are relieved by the free administration of bicarbonate of sodium. According to some authorities salivation may be avoided if small doses of chlorate of potassium are administered along with the mercury.

ST. CLAIR THOMSON.

SALSOMAGGIORI, in Italy.—Very rich iodine- and bromine-waters. This resort is at the foot of the Apennines, 520 feet above the sea-level. *See* MINERAL WATERS.

SALTATORY SPASM.—A nervous affection, consisting in alternating contractions of the flexor and extensor muscles of the legs induced by the pressure of the soles of the feet upon the ground, and best marked therefore in the standing posture. Gradual recovery is usual, but the disease is sometimes associated with permanent lesions of the spinal cord.

SALZBRUNN, in German Silesia.—Alkaline waters. *See* MINERAL WATERS.

SALZKAMMERGUT, in Austria.—An inland bracing summer climate. *See* CLIMATE, Treatment of Disease by; and ISCHL.

SALZUNGEN, in Saxe-Meiningen.—Cold muriated saline waters. *See* MINERAL WATERS.

SAN DIEGO.—A health resort in South California, on the Pacific Coast. A dry bracing climate, resembling that of Santa Barbara, free from fog, and equable. Mean temperature 60° F.; the records seldom rise to 80° or sink to the freezing-point. Mean daily range only 15° . Average rainfall 10 inches. Winds principally from the north-west. Well suited for pulmonary complaints. *See* CLIMATE, Treatment of Disease by.

SANDOWN, in the Isle of Wight.—Exposure S.E. Mean temperature 51.4° . An open, bright, bracing health-resort, with good sea-bathing. *See* CLIMATE, Treatment of Disease by; and SEA-AIR and SEA-BATHS.

SAND-WORM.—A term sometimes employed to designate the sand-flea or jigger. *See* CHIGOE.

SANITARY LAW.—The first laws which were passed in England relating to sanitary matters were directed successively against the scourges plague, small-pox, and cholera, and these were placed under control by various statutes which were passed between 1603 and the commencement of the reign of Victoria.

The statute of 1 Jac. I. c. 31 made it a capital offence for any person having an infectious sore upon him uncured to go abroad and converse in company, after being commanded by the proper authority to keep his house. That Act was repealed in the first year of the last reign (1837), but some statutes relating to quarantine, which were passed in the first instance for the purpose of dealing with plague, remain in force to the present day. Small-pox was not the subject of legislation until a generation after the discovery of vaccination, and the first Act dealing with it was passed in 1840 (*see* VACCINATION). Cholera became the subject of legislation in 1832, after the serious visitation with which the country had then been afflicted. By the law that was then passed the Privy Council were empowered to issue such orders as appeared expedient for the purpose of preventing the spread of the epidemic, for securing the proper burial of the dead, and for relieving the necessities of persons suffering from the malady. Apart from epidemic disease, the Legislature was slow to deal with sanitary precautions, and it was left to private persons to vindicate the public right to have nuisances abated by the enforcement of the common law. This procedure was tedious, costly, and uncertain, and hence, in boroughs and populous towns, special local statutory powers were frequently obtained.

Boards of commissioners for the paving, improvement, lighting, and watching of towns were formed for many places under the sanction of Parliament, and these bodies, together with the reformed municipalities created by the Municipal Corporations Act of 1835, formed the nucleus around which developed the local governing bodies as we know them to-day. The first conception of a local authority was that of a body whose existence, except in the case of a corporation created by charter, could only be brought about by the direct sanction of Parliament given by way of a local Act. This procedure, though at first cumbersome, was, under the Ministry of Sir Robert Peel, greatly simplified by the passing of what have been long known as the Model Clauses Acts. The effect of this improvement was to enable comparatively short local Acts to be passed, and to incorporate by mere reference a large number of useful provisions relating to various matters of sanitary and other local administration. Owing, however, to the costliness of obtaining parliamentary sanction to local bills, there was a growing demand for some simpler method of creating a local governing body in a town of sufficient importance to require one; and the sanitary commissioners who had been examining the condition of the country having reported as to the great need for the establishment of a general scheme of local government for towns and populous districts, the attention of Parliament was at length seriously directed to the matter. After two unsuccessful attempts the Public Health Act of 1848 was passed. By this epoch-marking statute a general board of health was established and power was given to it to cause the Act to be applied to any place having a known and defined boundary. These

provisions were taken advantage of freely, and the regulation of such matters as the laying out and sewerage of streets, the construction and drainage of houses, the provision of a water-supply to towns, and the removal of nuisances, were put within the easy reach of towns that had previously been subject to no control whatever. Ten years later this Act was amended and replaced by the Local Government Act, 1858, which Act remained in force till 1875, and under it about eight hundred local boards were formed up to 1872. As a result of the report of the Sanitary Commission of 1869, the scheme of local government was carried still further by the passing of the Public Health Act, 1872, under which the whole country was divided into urban and rural sanitary districts, and the Sanitary Acts applied to the entire country. And when the Public Health Act, 1875, which repealed and consolidated all the previous statutes outside the metropolis that related to sanitary law, was passed, this arrangement was not disturbed, and it now forms the basis of local government as settled by the Local Government Act, 1894. In 1901 there were in existence 1,150 urban authorities and 676 rural authorities, and under the Act mentioned these bodies are continued in force, under the description of urban district councils and rural district councils, for urban districts and rural districts respectively. Of the urban districts, 64 were county boroughs, 244 were non-county boroughs, and the rest were urban districts.

In addition to these bodies, every rural parish having a population of three hundred or upwards has a parish council; and such rural parishes as have a population of one hundred or upwards are enabled to have a parish council if they so resolve, and provision is made to form or dissolve a parish council if the population changes. Some parishes may by order of the county council be grouped under a common parish council. In addition to these bodies, there is in rural districts the parish meeting for every 'rural parish' in such district. This consists of the persons known as the parochial electors—that is, the persons who are registered on the parliamentary and local government register of electors—for the parish. Taking them in order of importance, there are the following local governing bodies: (1) Councils for counties; (2) Councils for municipal boroughs; (3) Councils for urban districts which are not boroughs; and (4) Councils for rural districts. For the purposes of sanitary administration the urban and rural district councils are the most important bodies, and the jurisdiction of the county councils over them is very limited. In some cases, such as the provision of sewerage and water-supply, a parish council has the right to complain to the county council that the rural district council is in default, and the county council can thereupon supersede that body. In the case of urban district councils in default an appeal lies to the Local Government Board.

It is unnecessary here to set forth the mode of election and qualifications of councillors or the details of the constitution of the councils; but it may be stated in general terms that the councils consist of members who are called councillors, and who are elected by the registered electors, with the addition, in the case of municipal corporations and county councils, of aldermen who are elected by the councillors. The constitution of county councils is determined by the Local Government Act, 1888; of

municipal corporations by the Municipal Corporations Act, 1882; and of urban and rural district councils by the Local Government Act, 1894.

It will now be necessary to describe in detail the powers of the local authorities, and the rights and liabilities of individuals in England and Wales, with special reference to sanitary matters only.

POWERS OF LOCAL AUTHORITIES.—A. County Councils.—By the Local Government Act, 1888 (section 17), it is provided that a county council may appoint one or more medical officers of health, and may make arrangements for rendering the services of any such officer regularly available in the district of any district council; and section 19 further provides that if it appears to a county council from the report of the medical officer of health of any district that the Public Health Act, 1875, has not been properly put in force within the district to which the report relates, or that any other matter affecting the public health of the district requires to be remedied, the council may cause a representation to be made to the Local Government Board on the matter. Such a representation would enable that Board to put in force the powers conferred upon them by section 299 of the Public Health Act, 1875. Under that section they may issue an order limiting the time for the local authority to perform its duty; and if such duty is not performed by the time so limited, the order may be enforced by writ of *mandamus*, or the Board may appoint some person to perform such duty.

B. Urban and Rural District Councils.—It is declared by section 7 of the Housing of the Working Classes Act, 1885, to be the duty of every local authority entrusted with the execution of laws relating to public health and local government to put in force from time to time, as occasion may arise, the powers with which they are invested, so as to secure the proper sanitary condition of all premises. These and other powers possessed by urban authorities under the sanitary laws will now be explained. They may be roughly divided into powers (1) which relate to the improvement of the general sanitary condition of the district viewed in its physical aspects; (2) which relate to the maintenance of dwellings and their surroundings, as well as factories and workshops, in a proper sanitary condition. It must be borne in mind that the powers here described are those exercisable by urban authorities, and that the powers of rural authorities, though capable of being extended, as occasion arises, by means of orders issued by the Local Government Board, are under the Public Health Acts limited to certain matters only.

1. *Powers which relate to the improvement of the general sanitary condition of the district.*

Sewerage.—Foremost under this heading comes the provision of sewers, and these the local authority is required to make so far as may be necessary for effectually draining their district. Thus the surface-water and the sewage will be carried off, but it must not be discharged into any stream until it has been purified. Sewage may be disposed of or dealt with by any suitable method, and sewage-works can be provided for the purpose, either in or out of the district of the local authority. One local authority may agree for the communication of their sewers with those of another in an adjoining district.

Water-Supply.—A local authority may provide their district or any part of it with a supply of water proper and sufficient for public and private purposes, and may (1) construct and maintain waterworks, dig wells, and do any other necessary acts; (2) take on lease or hire any waterworks, and (with the sanction of the Local Government Board) purchase any waterworks, or any water or any right to take or convey water, either within or without their district, and any rights, powers, and privileges of any water-company; and (3) contract with any person for a supply of water.

Before commencing to construct waterworks within the limits of supply of any water-company empowered by Act of Parliament or any order confirmed by Parliament to supply water, the local authority must give written notice to every water-company within whose limits of supply the local authority are desirous of supplying water, stating the purposes for which and (as far as may be practicable) the extent to which water is required by the local authority.

It is not lawful for the local authority to construct any waterworks within such limits if and so long as any such company are able and willing to supply water proper and sufficient for all reasonable purposes for which it is required by the local authority; and any difference as to whether the water which any such company are able and willing to lay on is proper and sufficient for the purposes for which it is required, or whether the purposes for which it is required are reasonable, or as to the charges, must be settled by arbitration.

The provisions of the Waterworks Clauses Acts with regard to communication-pipes, the waste or misuse of water, the fouling of water, and the payment and recovery of water-rates, are incorporated with the Public Health Act, 1875, and in this way local authorities are enabled to take advantage of the powers which are generally conferred on water-companies for carrying out the supply of water to any district.

Pollution of Rivers.—Any local authority, with the sanction of the Attorney-General, may, either in their own name or in the name of any other person, with the consent of such person, take proceedings under the Public Health Act for the purpose of protecting any watercourse within their jurisdiction from pollutions arising from sewage either within or without their district. Besides these powers, the Rivers Pollution Prevention Acts require local authorities to enforce a number of penal provisions which are designed to prevent the fouling of streams by solid matters, by sewage, or by manufacturing or mining refuse.

Polluted Wells.—Under the Public Health Act, 1875, it is provided that, on the representation of any person to any local authority that within their district the water in any well, tank, or cistern, public or private, or supplied from any public pump, and used or likely to be used by man for drinking or domestic purposes, or for manufacturing drinks for the use of man, is so polluted as to be injurious to health, such authority may apply to a court of summary jurisdiction for an order to remedy the same; and thereupon such court shall summon the owner or occupier of the premises to which the well, tank, or cistern belongs if it be private, and in the case of a public well, tank, cistern, or pump, any person alleged in the application to be interested in the same, and may either dismiss the

application, or may make an order directing the well, tank, cistern, or pump to be permanently or temporarily closed, or the water to be used for certain purposes only, or such other order as may appear to them to be requisite to prevent injury to the health of persons drinking the water. The court may, if they see fit, cause the water complained of to be analysed at the cost of the local authority applying to them under this section.

Streets.—All streets which are highways repairable at the expense of the inhabitants, and the pavements, stones, and other materials provided for the purposes of the highways by any surveyor of highways, belong to the local authority, and are under their management and control. They must level, pave, flag, channel, alter, and repair the streets as occasion may require, and may raise, lower, or alter the soil, and may place and keep in repair fences and posts for the safety of foot-passengers. They may regulate the line of buildings in streets by reference to existing buildings, but they cannot prescribe a building-line. They may make by-laws as to the width and construction of new streets. The local authority can undertake or contract for the proper cleansing of streets, the removal of house-refuse from premises, and the cleansing of earth-closets, privies, ashpits, and cesspools, and may by public notice require the periodical removal of manure or other refuse-matter from stables or other premises.

Cemeteries.—Under the Public Health (Interments) Act, 1879, a local authority may provide a cemetery, and the provisions of the Cemeteries Clauses Act, 1847, are made applicable. Where the Burial Acts are in force, burial grounds may be provided by the local authorities who exercise the functions of a burial board, or by a specially appointed burial board itself. The control of burial boards was formerly vested in the Home Secretary, but it has now been transferred to the Local Government Board under the Burials Act, 1900.

Pleasure-Grounds.—An important power, the exercise of which must have a very important bearing on the general well-being of our towns, is that which enables a local authority to provide pleasure-grounds for its district. A local authority can provide and maintain public walks and pleasure-grounds and other open spaces by the exercise of the powers conferred by the Public Health Acts and the Open Spaces Acts.

2. *Powers which relate to the maintenance of dwellings and their surroundings, as well as factories and workshops, in a proper sanitary condition.*

Construction of Buildings.—The Public Health Acts contain many important provisions relating to the sanitary condition of dwelling-houses, but the most important are those which authorise the making of by-laws for regulating the erection of new buildings, as defects may be prevented at the outset that could not be remedied at a later stage. This power of making by-laws enables the local authority to require plans of every new building; to regulate the thickness of walls, and the provision of damp-proof courses; to require the site under the floors to be covered with concrete; to regulate the height of rooms; to prescribe definite areas of open space in front and rear so as to secure a free circulation of air; to require that drains shall be properly laid and ventilated; to regulate the mode of construction of water-closets,

privies, ashpits, and cesspools, and their distance from dwellings and sources of water-supply, as well as many other matters. The powers are contained in section 157 of the Act of 1875, as amended by section 23 of the Act of 1890, which latter Act is only in force in the districts which adopt it. These sections are as follows: The Public Health Act, 1875, section 157, provides, as regards buildings, that every urban authority may make by-laws with respect to the following matters—that is to say: With respect to the structure of walls, foundations, roofs, and chimneys of new buildings, for securing stability and the prevention of fires, and for purposes of health; With respect to the sufficiency of the space about buildings, to secure a free circulation of air, and with respect to the ventilation of buildings; With respect to the drainage of buildings, to water-closets, earth-closets, privies, ashpits, and cesspools in connection with buildings, and to the closing of buildings or parts of buildings unfit for human habitation, and to prohibition of their use for such habitation.

And the Public Health Acts (Amendment) Act, 1890, section 23, provides that the above-cited section shall be extended so as to empower every urban authority to make by-laws with respect to the following matters—that is to say: The keeping water-closets supplied with sufficient water for flushing; the structure of floors, hearths, and staircases, and the height of rooms intended to be used for human habitation; and the paving of yards and open spaces in connection with dwelling-houses.

By section 23 of the Act of 1890, it is further provided as follows: 'Every local authority may make by-laws to prevent buildings which have been erected in accordance with by-laws made under the Public Health Acts from being altered in such a way that if at first so constructed they would have contravened the by-laws.' All by-laws made under these enactments require to be confirmed by the Local Government Board, but before such confirmation can be obtained notice of intention to apply for confirmation must be given, in one or more of the local newspapers circulated within the district to which the by-laws relate, one month at least before the making of the application; and for one month at least before the application, a copy of the proposed by-laws must be kept at the office of the sanitary authority, and must be open during office hours thereat for the inspection of the ratepayers of the district to which the by-laws relate, without fee or reward. When such by-laws have been made, it is the duty of the local authority to enforce them against every one with impartiality. The Local Government Board have issued separate model codes of by-laws for urban and rural districts.

Drainage.—It is not lawful in any urban district newly to erect any house or to rebuild any house which has been pulled down to or below the ground floor, or to occupy any house so newly erected or rebuilt, unless and until a covered drain or drains be constructed, of such size and materials, and at such level, and with such fall as, on the report of the surveyor, may appear to the urban authority to be necessary for the effectual drainage of such house; and the drain or drains so to be constructed must empty into a sewer which the urban authority are entitled to use, and which is within one hundred feet of some part of the site of the house to be built or rebuilt; but if no such means

of drainage are within that distance, then into such covered cesspool or other place, not being under any house, as the urban authority direct.

Cleansing.—Where, on the certificate of the medical officer of health or of any two medical practitioners, it appears to any local authority that any house or part thereof is in such a filthy or unwholesome condition that the health of any person is affected or endangered thereby, or that the whitewashing, cleansing, or purifying of any house or part thereof would tend to prevent or check infectious disease, the local authority are required to give notice in writing to the owner or occupier of such house or part thereof to whitewash, cleanse, or purify the same, as the case may require.

Water-Supply.—Where, on the report of the surveyor of a local authority, it appears to such authority that any house within their district is without a proper supply of water, and that such a supply of water can be furnished thereto at a cost not exceeding the water-rate authorised by any local Act in force within the district, or where there is not any local Act so in force, at a cost not exceeding twopence a week, or at such other cost as the Local Government Board may, on the application of the local authority, determine under all the circumstances of the case to be reasonable, the local authority may give notice in writing to the owner, requiring him, within a time therein specified, to obtain such supply, and to do all such works as may be necessary for that purpose. If such notice is not complied with within the time specified, the local authority may, if they think fit, do such works and obtain such supply, and for that purpose may enter into any contract with any water-company supplying water within their district; and water-rates may be made and levied on the premises by the authority or company which furnishes the supply, and may be recovered as if the owner or occupier of the premises had demanded a supply of water and were willing to pay water-rates for the same.

Particular Classes of Dwellings.—In regard to particular classes of dwellings, there are special provisions in the Public Health Acts. Such provisions relate to cellar-dwellings, common lodging-houses, and houses let in lodgings or occupied by members of more than one family. As to the first, it is provided that it shall not be lawful to let or occupy or suffer to be occupied separately as a dwelling any cellar (including for the purposes of the Act in that expression any vault or underground room) built for rebuilt after the passing of the Act (viz. August 11, 1875), or which was not lawfully so let or occupied at the time of the passing of the Act. But cellar-dwellings which were then in actual occupation are allowed to be used subject to certain limitations as to height and situation.

A person may not keep a common lodging-house or receive a lodger therein unless the house is registered in accordance with the provisions of the Act, nor unless his name as the keeper thereof is entered in the register kept under the Act; but when the person so registered dies, his widow or any member of his family may keep the house as a common lodging-house for not more than four weeks after his death without being registered as the keeper thereof.

A house may not be registered as a common lodging-house until it has been inspected and ap-

proved for the purpose by some officer of the local authority; and the local authority may refuse to register as the keeper of a common lodging-house a person who does not produce to the local authority a certificate of character, in such form as the local authority direct, signed by three inhabitant householders of the parish, respectively rated to the relief of the poor of the parish within which the lodging-house is situate for property of the yearly rateable value of six pounds or upwards.

The most effective control that can be exercised over common lodging-houses is that obtained by means of by-laws which every local authority is required to make: (1) for fixing and from time to time varying the number of lodgers who may be received into a common lodging-house, and for the separation of the sexes therein; and (2) for promoting cleanliness and ventilation in such houses; and (3) for the giving of notices and the taking precautions in the case of any infectious disease; and (4) generally for the well ordering of such houses.

As regards houses let in lodgings which are not common lodging-houses, but are commonly known as lodging-houses or tenement-houses, the only special control that can be exercised is by means of by-laws under section 90 of the Public Health Act, 1875. This section is put in force in the district of every local authority by section 8 of the Housing of the Working Classes Act, 1885, and its provisions are very much the same as those of the section relating to by-laws for common lodging-houses.

Conditions to be Implied on Letting Houses for the Working Classes.—It is provided by section 12 of the Housing of the Working Classes Act, 1885, that in any contract for letting for habitation by persons of the working classes a house or part of a house, there shall be implied a condition that the house is at the commencement of the holding in all respects reasonably fit for human habitation. The expression 'letting for habitation by persons of the working classes' means the letting for habitation of a house or part of a house at a rent not exceeding in England the sum named as the limit for the composition of rates by section 3 of the Poor Rate Assessment and Collection Act, 1869.

Nuisances.—Coming now to general nuisances, we find that section 91 of the Public Health Act, 1875, as amended by section 9 of the Housing of the Working Classes Act, 1885, and by the Factory and Workshops Acts, 1878 to 1901, sets out a comprehensive list of nuisances which are capable of being abated, and for the abatement of which the local authority is empowered to serve notices, and on the non-compliance with the terms thereof to apply to a court of summary jurisdiction for an order requiring the abatement of the nuisance, or prohibiting its recurrence, and directing the execution of any works necessary to secure that end. The nuisances which are liable to be dealt with in this way are set out as follows:—

(1) Any premises (including a tent, van, shed, or similar structure used for human habitation) in such a state as to be a nuisance or injurious to health.

(2) Any pool, ditch, gutter, watercourse, privy, urinal, cesspool, drain, or ashpit, so foul or in such a state as to be a nuisance or injurious to health.

(3) Any animal so kept as to be a nuisance or injurious to health.

(4) Any accumulation or deposit which is a nuisance or injurious to health.

(5) Any house or part of a house or a tent, van, shed, or similar structure used for human habitation, so overcrowded as to be dangerous or injurious to the health of the inmates, whether or not members of the same family.

(6) Any factory, workshop, or workplace not kept in a cleanly state, or not ventilated in such a manner as to render harmless as far as practicable any gases, vapours, dust, or other impurities generated in the course of the work carried on therein that are a nuisance or injurious to health, or so overcrowded while work is carried on as to be dangerous or injurious to the health of those employed therein.

(7) Any fireplace or furnace which does not as far as practicable consume the smoke arising from the combustible used therein, and which is used for working engines by steam, or in any mill, factory, dyehouse, brewery, bakehouse, or gaswork, or in any manufacturing or trade process whatsoever; and

Any chimney (not being the chimney of a private dwelling-house) sending forth black smoke in such quantity as to be a nuisance.

Since the Factory and Workshops Act, 1891, the duty of enforcing the sanitary provisions of the law in the case of all factories and workshops is vested in local authorities, and the powers are extended under the Factory and Workshops Act, 1901.

The local authority, or any of their officers, must be admitted into any premises for the purpose of examining as to the existence of any nuisance thereon, or of enforcing the provisions of any Act in force within the district requiring fireplaces and furnaces to consume their own smoke, at any time between the hours of nine in the forenoon and six in the afternoon; or in the case of a nuisance arising in respect of any business, then at any hour when such business is in progress or is usually carried on. In the case of tents, vans, sheds, and similar structures, entry can only be demanded between six o'clock in the morning and the succeeding nine o'clock in the evening.

Where under the Act a nuisance has been ascertained to exist, or an order of abatement or prohibition has been made, the local authority or any of their officers must be admitted from time to time into the premises between the hours aforesaid until the nuisance is abated, or the works ordered to be done are completed, as the case may be.

If admission to premises for any of these purposes is refused, any justice on complaint thereof on oath by any officer of the local authority may, by order under his hand, require the person having custody of the premises to admit the local authority, or their officer, into the premises during the hours aforesaid, and if no person having custody of the premises can be found, the justice shall, on oath made before him of that fact, by order under his hand, authorise the local authority, or any of their officers, to enter such premises during the hours aforesaid.

Any order made by a justice for admission of the local authority, or any of their officers, on premises will continue in force until the nuisance has been abated, or the work for which the entry was necessary has been done.

Where an order of abatement or prohibition has not been complied with, or has been infringed, the

local authority, or any of their officers, are entitled to be admitted from time to time at all reasonable hours, or at all hours during which business is in progress or is usually carried on, into the premises where the nuisance exists, in order to abate the same.

It is the duty of every local authority to cause to be made from time to time an inspection of their district, with a view to ascertain what nuisances exist calling for abatement under the powers of the Public Health Act, and to enforce the provisions of the Act in order to abate the same; also to enforce the provisions of any Act in force within their district requiring fireplaces and furnaces to consume their own smoke.

In addition to the above offences, which are described in the Public Health Act, 1875, as nuisances in every district, whether urban or rural, there is a section (s. 47) which is specially applicable to urban districts. It provides that every person who in an urban district (1) keeps any swine or pigsty in any dwelling-house, or so as to be a nuisance to any person; or (2) suffers any waste or stagnant water to remain in any cellar or place within any dwelling-house for twenty-four hours after written notice to him from the urban authority to remove the same; or (3) allows the contents of any water-closet, privy, or cesspool to overflow or soak therefrom, shall for every such offence be liable to a penalty; and the local authority must abate the nuisance in the manner indicated above.

Every urban authority has power to make by-laws for the prevention of nuisances arising from snow, filth, dust, ashes, and rubbish, and the prevention of the keeping of animals on any premises so as to be injurious to health. Under these powers the deposit and removal of accumulations of filth may be controlled, and the keeping of swine and other animals placed under restrictions. These powers are further extended where the Public Health Acts (Amendment) Act, 1890, is in force, for that enactment confers on local authorities power to make by-laws for prescribing the times for the removal or carriage through the streets of any faecal or offensive or noxious matter or liquid, whether it is in course of removal, or carriage from within or without, or through the district; and requiring that the vessel, receptacle, cart, or carriage used for this purpose is properly constructed and covered so as to prevent the escape of any of the contents, and for compelling the cleansing of any place on which any such matter or liquid has been dropped or spilt in the removal or carriage. The model by-laws of the Local Government Board on this subject may usefully be consulted.

Offensive Trades.—Offensive trades are at times a source of great nuisance, and this was recognised by the framers of the Public Health Act, 1848, the provisions of which Act have been substantially incorporated in the Act of 1875. It is there enacted that any person who establishes any of the offensive trades specified (which are the trades of blood-, bone-, soap-, and tripe-boiler, fell-monger, and tallow-melter), or 'any other noxious or offensive trade, business, or manufacture,' without the consent in writing of the urban authority, shall be liable to penalties. The defect of the earlier Act was that proceedings with respect to offensive trades could only be taken within the limits of a city, town, or populous district; and it was necessary to show that the person complained

of was not using the best practicable means to abate the nuisance. Under the Act of 1875 the proceedings in question may be taken by the authority in any urban district, and the burden of proof that the best practicable means are used to abate the nuisance is thrown upon the defendant. Any urban authority may from time to time make by-laws with respect to any offensive trades established with their consent, in order to prevent or diminish the noxious or injurious effects thereof. It may be well to point out that any 'other' trade besides those specified in the Act to be within the powers of the Act must be *ejusdem generis*. Hence it has been held that brick-burning and the keeping of a fried-fish shop are not 'offensive trades,' but that the business of a rag-and-bone merchant is an offensive trade within the terms of the Act.

Slaughter-houses.—Closely allied to offensive trades are slaughter-houses, and as to these the powers of the Public Health Acts are very extensive. In the first place, every slaughter-house in existence before the Local Government Acts were in force in the place where it is situated has to be registered, and all new ones have to be licensed. Under the Public Health Acts (Amendment) Act, 1890, the license may be made an annual one. The local authority may make by-laws dealing among other things with the maintenance of the slaughter-houses in a cleanly and proper state, and requiring a sufficient supply of water to be provided.

Hospitals.—Of late years great progress has been made in dealing with infectious disease by the provision, under the powers of the Public Health Act, 1875, and the Isolation Hospitals Acts, 1893 and 1901, of hospitals to which patients can be removed. Any local authority may provide for the use of the inhabitants of their district hospitals or temporary places for the reception of the sick, and for that purpose may themselves build such hospitals or places of reception; or contract for the use of any such hospital or part of a hospital or place of reception; or enter into any agreement with any person having the management of any hospital, for the reception of the sick inhabitants of their district, on payment of such annual or other sum as may be agreed on; and two or more local authorities may combine in providing a common hospital.

Where any suitable hospital or place for the reception of the sick is provided within the district of a local authority, or within a convenient distance of such district, any person who is suffering from any dangerous infectious disorder, and is without proper lodging or accommodation, or lodged in a room occupied by more than one family, or is on board any ship or vessel, may, on a certificate signed by a legally qualified medical practitioner, and with the consent of the superintending body of such hospital or place, be removed, by order of any justice, to such hospital or place at the cost of the local authority; and any person so suffering, who is lodged in any common lodging-house, may, with the like consent and on a like certificate, be so removed by order of the local authority; and any person who wilfully disobeys or obstructs the execution of such order will be liable to a penalty not exceeding ten pounds. It is further provided by the Infectious Disease (Prevention) Act, 1890, that any justice of the peace, upon proper cause shown to him, may, where the Act (which is adoptive) is put in operation, make an order directing the detention in hospital, at the cost of the local authority, of any

person suffering from an infectious disease, who is then in a hospital for infectious disease, and would not, on leaving the hospital, be provided with lodging or accommodation in which proper precautions could be taken to prevent his spreading the disorder. The order may be limited to some specific time, but any justice has power to enlarge the time as often as may appear to him to be necessary.

Notification of Infectious Disease.—The Infectious Disease (Notification) Acts, 1889 and 1899, and the Infectious Disease (Prevention) Act, 1890, contain some very important provisions which it is proposed now to describe.

The Infectious Disease (Notification) Act, 1889, was originally adoptive, but by the Infectious Disease Notification Extension Act, 1899, was put in force in every district throughout the kingdom. Under the Act it is provided that where an inmate of any building used for human habitation is suffering from an infectious disease to which the Act applies, then, unless such building is a hospital in which persons suffering from an infectious disease are received,

(a) *The head of the family* to which such inmate (referred to as the patient) belongs, and in his default the nearest relatives of the patient present in the building or being in attendance on the patient, and in default of such relatives every person in charge of or in attendance on the patient, and in default of any such person the occupier of the building, shall, as soon as he becomes aware that the patient is suffering from an infectious disease to which the Act applies, send notice thereof to the medical officer of health of the district.

(b) Every *medical practitioner* attending on or called in to visit the patient shall forthwith, on becoming aware that the patient is suffering from an infectious disease to which the Act applies, send to the medical officer of health for the district a certificate stating the name of the patient, the situation of the building, and the infectious disease from which, in the opinion of such medical practitioner, the patient is suffering.

The certificate must be in the form prescribed by the Local Government Board, and must be given in respect of a case of infectious disease to which the Act applies occurring in any building, not belonging to His Majesty, used for human habitation, unless such building is a hospital in which persons suffering from an infectious disease are received; and also in a case occurring in any ship, vessel, or boat not belonging to His Majesty or to a foreign Government, or in any tent, van, shed, or similar structure used for human habitation and not belonging to His Majesty, in like manner as nearly as may be as if it were a building. The penalty for default in sending the certificate is a fine not exceeding 10s. The forms of certificates are to be supplied gratuitously by the local authority, who will also pay for every certificate sent by a medical practitioner in accordance with the requirements of the Act a fee of 2s. 6d. if the case occurs in the course of his private practice, or a fee of 1s. if the certificate is given in respect of a case occurring in his practice as medical officer of any public body or institution. Where a medical practitioner attending on a patient is himself the medical officer of health of the district, he will be entitled to the fees to which he would be entitled if he were not such medical officer. It seems that if a medical practitioner in the proper exercise of his discretion certi-

fies that a patient is suffering from any disease, his opinion cannot be subjected to review by the local authorities, and the fee to which he is entitled under the Act must be paid.

The infectious diseases to which the Act applies in all cases include the following: small-pox, cholera, diphtheria, membranous croup, erysipelas, the disease known as scarlatina or scarlet fever, and the fevers known by any of the following names: typhus, typhoid, enteric, relapsing, continued, or puerperal. If the authority of any district to which the Act extends desire that the Act shall in their district apply to any infectious disease other than the above, they may from time to time, by a resolution, order that the Act shall in their district apply to such disease.

It is of importance to note that the provisions of the Act apply to every ship, vessel, boat, tent, van, shed, or similar structure used for human habitation, in like manner as nearly as may be as if it were a building, and that a ship, vessel, or boat lying in any river, harbour, or other water not within the district of any local authority within the meaning of the Act, will be deemed for the purposes of the Act to be within the district of such local authority as may be fixed by the Local Government Board, and where no local authority has been fixed, then of the local authority of the district which nearest adjoins the place where such ship, vessel, or boat is lying.

Prevention of Infectious Disease.—The Infectious Disease (Prevention) Act, 1890, is an adoptive Act, and is not in force until the prescribed forms for adopting it have been gone through; but it may be adopted in part only. The diseases to which it applies are the same as in the case of the Act of 1889, and the subjects to which it relates are—

(a) The inspection of dairies, and the prohibition of the supply of milk therefrom in cases where it appears that infectious disease is attributable to, or likely to be caused by, the consumption of milk so supplied;

(b) The cleansing and disinfection of premises, bedding, clothing, &c., for the purpose of preventing or checking infectious disease;

(c) The retention, removal, and burial of the bodies of persons who have died from infectious disease;

(d) The detention in hospital of persons suffering from infectious disease, who would, on leaving the hospital, be without proper lodging; and

(e) The provision of temporary shelter and attendance for members of families who have been compelled to leave their dwellings for the purpose of the dwellings being disinfected.

As during recent years attention has been very prominently directed to milk as a source of infection, it is not surprising to find that this Act confers powers which are considerably in advance of any which the legislature has previously given to local authorities. On this subject the Act provides that in case the medical officer of health of any district in which the section is adopted is in possession of evidence that any person in the district is suffering from infectious disease attributable to milk supplied within the district from any dairy situate within or without the district, or that the consumption of milk from the dairy is likely to cause infectious disease to any person residing in the district, he shall, if authorised in that behalf by an

order of a justice having jurisdiction in the place where the dairy is situate, have power to inspect such dairy, and, if accompanied by a veterinary inspector or some other properly qualified veterinary surgeon, to inspect the animals in the dairy. If, on such inspection, the medical officer of health is of opinion that infectious disease is caused by consumption of the milk supplied from the dairy, he is to make a report to the authority whose officer he is. This report must be accompanied by any report that may be furnished to him by the veterinary inspector or veterinary surgeon above mentioned. The authority may thereupon require the dairyman to appear before them to show cause why an order should not be made requiring him not to supply any milk from the dairy within the district until the order has been withdrawn. If he fails to show such cause, they may make the order.

The Act also contains some important provisions in regard to the use of public conveyances for the conveyance of the bodies of persons dying of infectious disease, the disposal of infectious rubbish, and the cleansing and disinfection of houses and articles therein likely to retain infection. On the last subject the Act provides that where the medical officer of health of any local authority, or any other registered medical practitioner, certifies that the cleansing and disinfecting of any house, or part of it, and of any articles in it likely to retain infection, would tend to prevent or check infectious disease, the clerk to the authority is to give notice in writing to the owner or occupier that the house or part of it, and any articles in it likely to retain infection, will be cleansed and disinfected by the authority at the cost of the owner or occupier, unless he informs the authority, within twenty-four hours from the receipt of the notice, that he will do the cleansing and disinfection, to the satisfaction of the medical officer of health, within a time fixed in the notice. If, within twenty-four hours from the receipt of the notice, the person to whom it is given does not inform the authority that he will so do the cleansing and disinfection, or if, having so informed the authority, he fails to have the work done within the time fixed in the notice, the house or part of the house and articles are to be cleansed and disinfected by the officers of the authority under the superintendence of the medical officer of health, and the expenses incurred may be recovered from the owner or occupier in a summary manner. But where the owner or occupier of the house or part of a house is unable effectually to cleanse and disinfect it, and any article therein likely to retain infection, the officers of the authority, with the consent of the owner or occupier, may undertake the cleansing and disinfection at the cost of the authority.

No person may retain unburied, elsewhere than in a public mortuary, or in a room not used at the time as a dwelling-place, sleeping-place, or work-room, for more than forty-eight hours, the body of any person who has died of an infectious disease, unless he shall have obtained the sanction in writing of the medical officer of health, or of a registered medical practitioner.

In connection with this Act it is of the greatest importance to bear in mind that it is an adoptive Act not only as a whole, but as to sections, so that before any particular enactment is acted on, steps should be taken to ascertain that it has been properly put in force in the particular district.

Dwellings of the Working Classes.—

Some very important alterations of the law were made by the Housing of the Working Classes Act, 1890, the principal of which related to (1) unhealthy areas, and (2) unhealthy dwellings.

As to unhealthy areas, it is provided that where an official representation is made to the local authority by the medical officer of health, that within a certain area in their district either (a) any houses, courts, or alleys are unfit for habitation; or (b) the narrowness, closeness, and bad arrangement or the bad condition of the streets and houses or groups of houses within such area, or the want of light, air, ventilation, or proper conveniences, or any other sanitary defects, or one or more of such causes, are dangerous or injurious to the health of the inhabitants either of the buildings in the said area or of the neighbouring buildings, and that the evils cannot be remedied otherwise than by an improvement-scheme for the re-arrangement and reconstruction of the streets and houses within such area, the local authority, if satisfied of the truth of the representation, shall pass a resolution to the effect that such area is an unhealthy area, and proceed to make a scheme for the improvement of the area. Before the scheme can come into operation it has to be confirmed by provisional order, which requires the sanction of Parliament.

The law relating to the assessment of compensation payable under an improvement scheme in respect of any house or premises situate in an unhealthy area, provides that in such cases evidence shall be receivable by the arbitrator to prove—(1) That the rental of the house or premises was enhanced by reason of the same being used for illegal purposes, or being so overcrowded as to be dangerous or injurious to the health of the inmates; or (2) that they are in such a condition as to be a nuisance within the meaning of the Acts relating to nuisances (which are defined by section 2 as meaning as respects any urban sanitary district in England the Public Health Acts, and as including any local Act which contains any provisions with respect to nuisances), or are in a state of defective sanitation, or are not in reasonably good repair; or (3) that they are unfit, and not reasonably capable of being made fit, for human habitation; and that if the arbitrator is satisfied by such evidence, then the compensation—(a) shall in the first case, so far as it is based on rental, be based on the rental which would have been obtainable if the house or premises were occupied for legal purposes, and only by the number of persons whom they were under all the circumstances of the case fitted to accommodate without such overcrowding as is dangerous or injurious to the health of the inmates; and (b) shall in the second case be the amount estimated as the value of the house and premises if the nuisance had been abated, or if they had been put into a sanitary condition, or into reasonably good repair, after deducting the estimated expense of abating the nuisance or putting them into such condition or repair, as the case may be; and (c) shall in the third case be the value of the land, and of the materials of the buildings thereon.

As to unhealthy dwellings, the Act gives some powers that are far in advance of any previous law. It is declared to be the duty of every local authority to cause to be made from time to time inspection of their district with a view to ascertain whether any dwelling-house therein is in a state so dangerous

or injurious to health as to be unfit for human habitation, and if, on the representation of their medical officer of health or any of their officers, or information given to them, it appears that any dwelling-house is in such state, to forthwith take the proceedings against the owner or occupier for closing it under the Public Health Act, 1875.

Under the Public Health Act it had hitherto been necessary that the proceedings should be taken with a view to the abatement of a nuisance, and that the works requisite to abate the nuisance complained of should be specified. This, however, is no longer the case, as summary proceedings may be taken for the express purpose of causing the dwelling-house to be closed, and appropriate forms are prescribed for this purpose.

The effect of the closing order will be to prohibit the using of the premises for the purpose of human habitation until, in the judgment of the court, they are rendered fit for that purpose. In making it the court may impose a penalty not exceeding 20*l*.

Where a closing order has been made in respect of any dwelling-house, and has not been determined by a subsequent order, the sanitary authority, if of opinion that the house has not been rendered fit for human habitation, and that the necessary steps are not being taken with all diligence to render it so fit, and that the continuance of any building being or being part of the dwelling-house is dangerous or injurious to the health of the public, or of the inhabitants of the neighbouring dwelling-houses, are required to pass a resolution that it is expedient to order the demolition of the building. Unless the building is promptly made fit for human habitation it must be demolished. Some important extensions of the law were made by the Housing of the Working Classes Act, 1900, by which county councils were empowered to acquire land and erect dwellings outside their districts, and thus relieve the congestion in towns.

COUNTY OF LONDON.—The law as set forth above relates to the whole of England and Wales, but does not apply to the county of London, for which there are several special Acts of Parliament. The Local Government Acts, 1888 and 1894, the Metropolitan Local Management Acts, the Metropolitan Building Acts, the Public Health (London) Act, 1891, the London Building Act, 1894, and the London Government Act, 1899, and some other acts, prescribe the law relating to the constitution, powers, and duties of the London County Council and the corporate boroughs into which all the former local vestries and boards are now merged; and also the general provisions as to the laying out of streets, the construction of buildings, and the regulation of all matters of sanitation and public health. It would be impossible to go into these provisions here, but in many respects they are the same as those above described for the provinces.

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SANITATION; SANITARY SCIENCE (*sanus*, sound).—Sanitary science treats of the principles on which health is maintained and disease prevented. The principal agencies which contribute to this result will be found treated of in this work under the following headings: BATHS; DISINFECTION; EXERCISE; FOOD, POISONOUS; HYDROTHERAPEUTICS; IMMUNITY; INFECTION; MORTALITY; PERSONAL HEALTH; PHYSICAL EDUCATION; PUBLIC HEALTH; QUARANTINE; SANITARY LAW;

SEA-AIR and SEA-BATHS; VACCINATION; and VITAL STATISTICS.

SAN REMO, on the Riviera, Italy, near the French Frontier.—A well-sheltered, dry, mild, equable winter climate. Mean winter temperature, 54° F. Early vegetation. *See* CLIMATE, Treatment of Disease by.

SANTA AQUEDA, in Spain.—Cold sulphated and chalybeate waters. *See* MINERAL WATERS.

SANTA BARBARA, in Southern California.—A very mild, equable climate, on the Pacific coast. Temperature—mean annual, 61° F.; winter mean, 54°. Rainfall, 16 inches. Used for phthisis and other pulmonary affections. *See* CLIMATE, Treatment of Disease by.

SANTA FÉ.—The capital of New Mexico, at an altitude of 7,013 feet, in a dry and arid region. Lacks equability of temperature. Mean annual temperature, 48° F.; relative humidity percentage, 45. More suitable for a winter than a summer residence for invalids. It is principally recommended as a winter residence for pulmonary affections on account of its combining a certain degree of warmth with altitude. *See* CLIMATE, Treatment of Disease by.

SAPRÆMIA, SEPTICÆMIA, and PYÆMIA.

The production of pus by the micro-organisms of sepsis never occurs, even in its most localised form, without an accompanying general effect on the blood. This effect is due to the toxins produced by the microbes in the septic area, and absorbed into the circulation. The absorption of toxins of various kinds is in itself the cause of the primary symptoms of all septic disease; but there may occur simultaneously an invasion of the blood by the bacteria which produce the toxins. The most pronounced form of septic disease is that in which the bacteria, starting from some local lesion, have invaded the circulation, and set up at various points secondary areas of inflammation more or less similar to the original focus.

General septic poisoning has accordingly been classified into three main forms.

(a) *Sapræmia*, in which only the toxins of the bacteria are absorbed, the bacteria themselves remaining confined to the primary area of inflammation. In practice, accordingly, when the original wound or abscess is surgically cleansed, the symptoms of intoxication rapidly vanish. A familiar example is seen in one form of puerperal fever, which yields to simple douching of the uterus. In the production of the bacterial poison, a large variety of bacteria may be involved, both pathogenic and putrefactive.

(b) *Septicæmia*.—In this form the bacteria of sepsis, as well as their toxins, find their way into the circulation. To demonstrate this condition clinically, it is necessary to remove portions of the blood, add them to suitable culture-media, and incubate at 37° C. Since, as a rule, few colonies appear, it is desirable to obtain a fairly large sample of blood. The bacteria in this condition, though present in the blood, do not give rise to localised abscesses apart from the original seat of infection. A good example of septicæmia is seen in the blood of rabbits which have had a subcutaneous injection of the exudate of croupous pneumonia. There is

only a small amount of localised inflammation at the site of the injection ; but before death the blood of the animal is swarming with diplococci. There are, however, no secondary foci of suppuration.

(c) *Pyæmia*, the third type of generalised sepsis, occurs when the original focus has given rise to a number of secondary centres of inflammation, situated at various points in the course of the circulation. The secondary foci develop into abscesses.

The classification of the three conditions is a practical one, and is based on the conception that we may regard the diseases as belonging to one type, i.e. essentially toxin-poisoning, and recognise a certain progressive character in the three forms. It must be admitted, however, that our present knowledge does not justify us in adopting this conception without qualification. A case of pyæmia is not necessarily more severe than a case of septicæmia, and we are only partly acquainted with the factors which determine the form which generalised sepsis may take in any particular instance.

SYMPTOMS.—Clinically, *sapræmia*, since it consists solely in the absorption of toxins, distinguishes itself from the other forms by the rapidity of its onset. *Post-mortem* and dissecting-room wounds, though of minute size, often begin to cause severe symptoms within twenty-four hours. There is local pain and swelling. The pulse increases in rate, the temperature rises, and there is a feeling of general weakness and disinclination for mental or bodily exertion. If at this stage the wound is cleansed, especially if the edges are cut away and free drainage encouraged by fomentations, the general and local symptoms rapidly vanish as a rule. In cases which proceed to further stages, rigors occur. There may be profuse perspiration, or a dry, hot skin. The tongue is at first red, and later becomes brown. There is often diarrhœa and vomiting, with slight jaundice. Sometimes there is great tenderness of skin, and even petechiæ may be observed. Emaciation proceeds rapidly. Later the temperature falls, and coma comes on. Death occurs in the second or third day of the disease ; but the patient may remain for some days in the so-called 'typhoid' condition, when death supervenes from exhaustion.

In *septicæmia*, the symptoms being also due to the effect of toxins are not essentially different from those of *sapræmia* ; but the process naturally tends to progress more slowly at first. It generally assumes one of two forms—a mild and an acute. When acute there is, as in cases of *sapræmia*, firstly a stage of violent reaction, followed by that of the 'typhoid' type. The toxins in septicæmia are manufactured both in the local focus and in the blood, and in later stages the disease may become very rapid.

Pyæmia defines itself still later than *sapræmia* or septicæmia. *A priori*, one would expect that the toxin-producing power of the microbe is relatively less, and its power of invading the tissues more developed, as compared with the bacteria of *sapræmia*. The symptoms of pyæmia are observed, generally speaking, within a week after the date of the wound or injury from which it originates. The symptoms of intoxication are the same as in *sapræmia* and septicæmia. The distinctive character of the disease is due to the occurrence of septic thrombosis and embolism in the circulation. This leads to the formation of secondary abscesses and to purulent inflammation of the serous membranes in the large

cavities and in the joints and tendon-sheaths. The brain, lungs, liver, kidneys, spleen, heart, and muscular tissues are the seat of thrombosis, embolism, and abscess-formation, and the symptoms due to such local effects correspond with the particular form of the lesion, e.g. apoplexy, delirium, angina pectoris, dyspnœa, paraplegia, &c. &c. A rash similar to that of scarlet fever is not uncommon, and hæmorrhages in the skin are frequently seen. The patient in fatal cases dies about the tenth day. Mild forms, sometimes called cases of multiple abscess, occur, in which the patient after developing a few metastatic abscesses in the subcutaneous, or some other non-vital area, recovers completely.

The diseases which give rise to pyæmia are chiefly the following : empyema, fetid bronchitis, infective compound fractures, ulcers of the intestine, infected wounds, and, somewhat rarely, infective periostitis.

PATHOLOGY.—The discussion of the structural changes to which septic invasion of the blood gives rise is naturally chiefly concerned with those seen in cases of pyæmia, and historically it is the form of septic disease which has been most closely investigated in this respect. The name was originally introduced by Piörriy in the attempt to explain all forms of general septic infection as due to the intravasation of pus. It was shown subsequently by Virchow and others, however, that it is impossible to group all the phenomena of pyæmia as the effects of a single cause. There are, it was pointed out, at least two series of effects, firstly the mechanical, taking the form of a block in the circulation, and due to thrombosis and embolism, and, secondly, an irritant effect due to the action on the cells in various tissues of some substance which has diffused into the blood from the infected areas. This was called the 'infecting substance,' and is now known to consist of microbes and the chemical poison derived from them.

The sequence of changes is the following. The effect of an inflammatory area on the veins in the neighbourhood is to cause thrombosis. The thrombus may be hard or soft. In any case it contains within itself the microbes and leucocytes which have the power of disintegrating it, e.g. by digesting it, and converting it into a mass of pus and broken-up septic clot (see *ABCESS*, p. 7). This material is in a condition to travel in the blood-vessels, and passes into the still patent part of the vessel to form emboli. The infected emboli settle in a small blood-vessel, and set up abscess-formation. Virchow pointed out, however, that on anatomical grounds this is not the sole explanation. We find, for example, in pyæmia, that large serous cavities are infected, and it is impossible to conceive that the inflammation of the whole surface of a lung could be due to mechanical emboli plugging the vessels. The 'infecting substance' itself causes inflammation, and acts along with the emboli, and in particular gives rise to conditions such as pleurisy and purulent synovitis. See *EMBOLISM*, p. 453.

Certain other changes are to be found within the blood in septic poisoning which fall to be described here. The cells of the blood show evidence of the direct effect of the poison. Commonly we find a marked leucocytosis of the ordinary inflammatory type, the polymorphonuclear neutrophile cells being over 80 per cent. of those present (p. 156). This immigration of leucocytes is due to the chemiotactic effect of the toxins. On the other hand, cases

cur in which there is not only no increase but even a diminution. The explanation of this fact is suggested by certain experiments with the pneumococcus of Fraenkel by Tschistowitch, who found that when he injected rabbits with a coccus of moderate virulence, leucocytosis occurred in the blood; if he used a coccus of exalted virulence, no such effect followed. See p. 157.

An interesting line of investigation is to be found in the study of the aggregation of leucocytes into masses in septic conditions of the blood. It is occasionally mentioned in the older descriptions of pyæmia that the leucocytes were observed in clumps in the blood (Sédillot, Virchow). Recently Metschnikoff has shown that in normal animals a serum can be obtained experimentally with a specific power of bringing about this form of agglutination. From a number of observations the present writer made on cases of pyæmia, he is inclined to regard this phenomenon as one of importance among the changes in pyæmic blood, particularly because a possible mode of explaining the factors which determine the occurrence, in one case of pyæmia, and in another of septicæmia, is suggested by it.

As regards the red corpuscles, it has been observed that the necrobiotic changes described by Ehrlich and Maragliano as occurring in various forms of anæmia occur in pyæmia also. Reference will be made later to Koch's discovery of the aggregation of red corpuscles to form emboli in the pyæmia of rabbits.

Virchow observed in a number of cases that the blood had lost its alkaline reaction. One of the most marked cases had suffered from puerperal pyæmia. The exact value of this observation is still to be decided. The acid present was not a volatile fatty acid.

There has been very little research carried out in the blood as a whole in pyæmia. Roscher in 1894 published the most important contribution to our knowledge of this subject. He showed that with the progress of the disease there is a diminution in the number of red blood-corpuscles per mm., and a proportional decrease in the percentage of hæmoglobin. There is also a diminution in the percentage of solid residue left when the serum is dried. This residue, he finds, cannot diminish below one-half without fatal results ensuing.

The lowering of the percentage of hæmoglobin may be due to mere increase in plasma, or to a true destruction of the red cells. Since, however, (1) hæmoglobinuria somewhat rarely occurs in pyæmia, and (2) the reduction of the residue after drying is marked, it seems probable that we have to deal with both changes in the blood, viz. hydræmia as well as hæmolysis.

Such is the general clinical and pathological picture of the disease which we are able to form on our present-day knowledge. It is unnecessary to go into much detail regarding the affections which appear in the various organs, since it is not an inflammation in a particular site, but the association of groups of inflammatory changes which is the significant fact in the diagnosis of pyæmia. The lung is naturally the most frequent site of abscess. The kidney from the structure of its capillaries is also very liable to abscess-formation. In areas of terminal arteries, as in the spleen and kidney, we find septic infarctions. Where there

are serous surfaces we have an inflammation affecting the whole, or large parts of the membrane. Thus synovitis, pleurisy, pericarditis, are frequent occurrences in the disease. In some cases, especially in children, bronchitis or enteritis is a prominent feature. In the brain, septic meningitis occurs, and also abscesses, which are especially frequent in the occipital lobe. A special form of pyæmia is septic or malignant endocarditis. See p. 652.

The ætiology of the condition was very partially understood until the discovery of the agency of bacteria as the 'infecting substance,' and poison-producer in the disease. The bacteria chiefly concerned in septicæmia and pyæmia are the *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, and *Staphylococcus pyogenes albus*. Many records have been published describing cases due to unusual micro-organisms, e.g. *Micrococcus tetragenus*, *Pneumococcus*, *Bacillus septicus putridus*, and *Bacillus hæmorrhagicus*. It is, however, unnecessary to do more than point out the possibility of such cases, their interest being in the light they throw on cases of inexplicable origin. See ABSCESS; and BACTERIA.

A considerable amount of experimental investigation has been carried out with a view to defining the nature of the toxic bodies which give rise to the phenomena of the disease. It was shown, in the first instance, that cultures of pus-producing microbes, when sterilised by heating, are still, if injected, capable of producing suppuration. The dose necessary for this purpose was, however, found to be very much larger than that of the living culture. It was accordingly concluded (Janowski) that in such an experiment it was necessary to introduce the quantity of toxic substance which is produced in the animal by the living culture before suppuration is brought about. When the effects were more closely investigated it was found that a distinction had to be drawn in regard to the bacteria of sepsis, as in the case of other bacteria, between intra- and extra-cellular poisons. The extra-cellular poisons, it was found, are easily destroyed by boiling; whereas the toxicity of the poisons in the bodies of the microbe is relatively less affected by this temperature. The intra-cellular poisons are by far the more important of the two, and are specially characterised by their chemiotactic power of attracting the leucocytes to the site of inflammation, and thereby producing pus-cells. In Koch's comparative study of infective septic diseases, he discovered that it is possible to reproduce experimentally in animals the conditions corresponding to sapræmia, septicæmia, and pyæmia. Thus, if blood which has putrefied for a few days be injected subcutaneously into a mouse (five drops) it dies in four to eight hours, and on *post-mortem* examination there is found no invasion of bacteria into blood or tissues, nor any sign of inflammation. The animal has died from simple intoxication. If the same fluid be injected in smaller amount, there is no poisoning. The animal remains apparently healthy for twenty-four hours, and then develops symptoms of disease, and dies in forty to sixty hours. There is slight subcutaneous œdema; the spleen may be enlarged; but further signs of disease are wanting on *post-mortem* examination. This animal is infected throughout, and from any organ infective material can be obtained which produces the same condition in other mice

into which it is inoculated. By using another kind of putrid fluid, Koch produced in rabbits a general infection, and from the blood of the animal successful inoculation could be performed. The *post-mortem* changes, local and general, were those of pyæmia, and in particular there were metastatic deposits in the lungs and liver. Koch further observed that the micrococci were deposited in masses on the side of the blood-vessels, and these masses showed a power of causing the adhesion together of red blood-corpuscles on the side of a vessel. The micrococci have thus the power in themselves of giving rise to the formation of thrombi and emboli. This power of aggregating the blood-corpuscles Koch regarded as a characteristic of the micrococcus with which he was experimenting, and to the formation of these masses he attributes the metastatic deposits. He obtained subsequently a true septicæmia in rabbits in which secondary deposits did not occur, and which had not the power of enclosing the red corpuscles in the masses which formed in the blood-vessels.

We see, therefore, that in mice and rabbits the various types of the general septic disease can be reproduced—the difference depending on the specific character of the microbes inoculated. It has also been found that the bacilli of mouse-septicæmia produce no effect on the rabbit, even when inoculated in very large doses. In this again we have evidence of the specific nature of the process. On the other hand, it has been shown in experimenting with the diplococcus of pneumonia that a variety of septic diseases can be produced by selecting for inoculation various types of animals, e.g. rabbits, sheep, dogs, &c. The specific character of the septic disease depends in this series of experiments on the kind of animal employed.

With these facts, pathological and bacteriological, before us, we have to discuss now more particularly the occurrence of general sepsis in the human subject. It must be admitted that in spite of the clinical and experimental investigations which have been shortly referred to in the foregoing paragraphs, we have not before us material to enable us to say decisively why a given case becomes pyæmic, while another is septicæmic. F. Gärtner recently studied two cases of septicæmia, and two of pyæmia, each originating in uterine infection, and each showing the same anatomical lesions of inflammation round a septic focus. In each case he found both streptococci and staphylococci. Gärtner does not propose an answer to the question, but concludes with the demonstration of the very close resemblance of the four cases so far as the origin was concerned. The micrococcus had in each case invaded the blood.

The path of entrance taken by the microbes is clear when a septic wound can be demonstrated. In many cases, however, the mode of invasion is obscure. These cases are sometimes called cryptogenic. Fischl carried out an investigation of great interest on septic infection of infants. He showed that the apparently primary gastro-enteritis and capillary bronchitis are in reality part of a general condition of septicæmia or pyæmia. In a recent *post-mortem* examination, the writer found this condition in the lungs of an adult. The abscesses contained a pure growth of *Staphylococcus pyogenes albus*. Fischl mentions that nine out of fourteen cases he examined showed this coccus in pure cultivation. He regarded the source of infec-

tion as the air or the food. Grawitz has shown that in certain abnormal conditions the general resistance of the tissues to the invasion of microbes is less than that in force when the subject is healthy. Some time ago the present writer performed a *post-mortem* examination on a patient who had shortly before death accidentally fractured a number of ribs. The patient also suffered from acute mania. Round each site of fracture an abscess had developed, although there was no discoverable lesion of the surface by which infection might have gained entrance to the tissues. This case illustrates the effect of disease and injury in lowering resistance to invasion. Gottstein has carried out an experimental investigation on this subject. He injected guinea-pigs with the bacillus of chicken-cholera, a microbe to which they are relatively immune, and at the same time gave them a dose of hydracetic subcutaneously. They died with abscess-formation and inflammatory change in the serous cavities. The point of importance in this experiment according to Gottstein is that the disintegration of red blood-corpuscles by the hydracetic sets free ferments which intoxicate the animal, and thus breaks down the resistance of the tissues. He also experimented on the breaking down of resistance by using pyrogallol, which dissolves the red blood-corpuscles, and came to the conclusion that in cases of sepsis the resistance of the whole organism is broken down by the poison, and this is followed up by an invasion of bacteria.

In considering this question in relation to pyæmia in the human subject we come upon a very difficult pathological problem. In 1886 Stephen Paget showed that a special relationship holds between abscess of the liver and fracture of bone, and that necrosis of bone has a similar relation to abscess of the kidney. Again, pleurisy and pericarditis are more common after injuries to limbs than after injuries to head and trunk. Abscess in the heart is more common in pyæmia involving the lower limbs than in any other. The lungs are the most favourite site of abscess. Among muscles, the pectoral are the most frequently affected, while the calf-muscles are the next in frequency. Another favourite site of inflammation is the synovial sheath of the tendons on the back of the left hand; another, the back of the left eye. The result of Paget's investigations is difficult of interpretation on any theory of local or general resistance. Marmorek has, however, attempted to arrange the observations on pyæmia in a general theory of lines of defence. Organs most liable to abscess-formation in pyæmia are those which are weakest in defence. The anti-bacterial coefficient of any organ is in inverse ratio to the number of bacteria found in it. The anti-bacterial power of a tissue is associated with the number of active living cells found in it. On this theory the first line of defence for the organism is the skin and subcutaneous connective tissue. The second is the lymphatic glands. The third line of defence is the blood. After this, organs are liable to suffer in inverse ratio to their anti-bacterial power. This theory is somewhat difficult to apply to the actual problems of septicæmia and pyæmia in the human subject.

TREATMENT. — As regards treatment of these conditions, little remains to be said. The surgical cleansing of all primary foci, and in some cases of pyæmia of secondary foci also, is the first step which should be taken (*see* CELLULITIS). In the

next place, if the sepsis be due to the streptococcus, the anti-streptococcus serum may be employed. The evidence as to the value of this new therapeutic agent has not yet accumulated in sufficient quantity to be decisive (*see* SERUM-THERAPEUTICS). Medical treatment of the symptoms is to be strenuously carried out. The temperature is to be controlled by antipyretics, and quinine is the drug most recommended for this purpose by the clinical authorities. The maintenance of the strength by suitable diet and stimulants is also of the greatest importance. In some of the cases where treatment has been successful the period of convalescence has been very prolonged. J. LORRAIN SMITH.

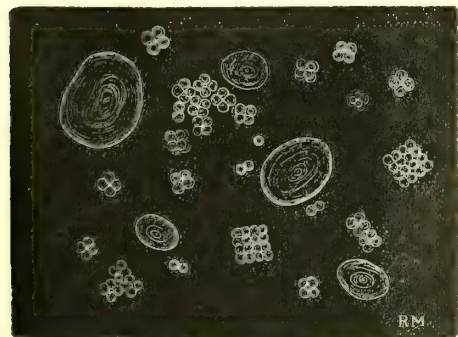
SARATOGA, in Saratoga County, New York, U.S.A.—Alkaline chalybeate and muriated alkaline waters, containing traces of iodine and bromine. The springs of Ballston, a few miles from Saratoga, are of a like character. *See* MINERAL WATERS.

SARCINA (*sarcina*, a pack or bundle).—This is a morphological term applied to various species of micro-organisms belonging to the group of Schizomycetes, which, developing by fission in three planes at right angles to one another, form more or less cubical aggregates of 4, 8, 16, 32, or 64 cocci, having the appearance of corded 'bales' or 'packets.' They are non-motile, do not form spores, and retain the stain after treatment by Gram's method. When cultivated upon artificial media many species are chromogenic, the characteristic arrangement in cubes is lost, and only diplococci and tetrads are to be demonstrated microscopically. *See* BACTERIA.

So far as is known at present, the sarcinæ are not pathogenic, but merely occur in diseased organs as casual saprophytes.

The most important species of sarcina met with in human pathology are the following:—

1. **Sarcina ventriculi**.—This species is of frequent occurrence in the stomach, and in the vomit of gastric dilatation from pyloric obstruction; in some cases of gastric ulcer and carcinoma without dilatation; and in rare cases of gastric catarrh.



Sarcina ventriculi. Showing sarcinæ and starch-granules in vomited matter. $\times 400$ diam.

Their presence, however, is not sufficiently constant to be of much diagnostic value, and is certainly not pathognomonic of a dilated stomach, as has been supposed. Their appearance is usually associated with a state of fermentation of the gastric contents, which appear frothy, and are of an acid reaction and smell; but it would seem that the organism is

not the actual cause of the condition, for cases are recorded of their occurrence in vomit that showed no signs of fermentation; while the introduction of pure cultivations of sarcinæ into the stomach is not followed by fermentative changes in its contents.

Sarcinæ are easily detected under the microscope. A drop of liquor potassæ added to a fragment of vomit on the glass slide, and covered with thin glass, is sufficient to display their characteristic appearances. The constituent cells are about 1.9μ in diameter.

2. **Sarcina pulmonum**.—This species has been described as occurring in certain lung-states associated with suppuration. It is said readily to induce ammoniacal decomposition of the urine when introduced into this fluid; and in one case appeared to contain starch or cellulose in its composition.

Sarcinæ are also stated to have been found in the fæces, in the ventricles of the brain, in hydrocele-fluid, in gangrenous intestines, and in cholera-stools. W. H. ALLCHIN.

SARCOCELE (*σάρξ*, flesh; and *κύλη*, a tumour). A name for any solid enlargement of the testes. *See* TESTES, Diseases of.

SARCOMA (*σάρξ*, flesh).—A form of new-growth. *See* TUMOURS.

SARCOPTES SCABIEI.—A synonym for *Acarus scabiei*. *See* ACARUS.

SATURNISM (*saturnus*, lead).—A synonym for lead-poisoning. *See* LEAD, Poisoning by.

SAWYER'S CRAMP.—*See* OCCUPATION-DISEASES; and WRITER'S CRAMP.

SCABIES (*scabies*, scab, mange).—SYN.: Itch; Fr. *Gale*; Ger. *Krätze*.

DEFINITION.—A simple inflammation of the skin, produced by the irritation of the *Acarus scabiei* and the scratching of the sufferer. The disease is contagious, as the parasite is easily transferred from one person to another. *See* ACARUS.

DESCRIPTION.—The parts of the body most likely to be attacked are the soft skin between the fingers, and on the flexor side of the wrists and elbows; the lower part of the abdomen, buttocks, and penis; and in women the mammæ; in children the feet and legs, in addition to the other parts of the body already mentioned, are very liable to be attacked. The acarus generally attacks both sides of the body symmetrically, and in adults is never met with in the skin of the face or scalp; but any other part of the body may be affected.

The eruption produced by the acari and by scratching, is attended by itching, especially at night, and resembles a scattered eczema; and the extent and severity of the disease will depend chiefly on its duration. The most characteristic feature for the purposes of diagnosis is the scabies-burrow, which resembles roughly an old pin-scratch. Examined closely, it has a dotted and beaded appearance with ragged dirty edges at its entrance, where the roof of the cuniculus has been worn away by rubbing. At the distant end of the burrow may be sometimes seen the parent-acarus, which is easily extracted by inserting the point of a pin along the burrow and touching the acarus, which immediately adheres to the pin, and may thus be removed for the purpose of examination.

DIAGNOSIS.—The following points serve to distinguish scabies: (1) the particular parts attacked as just indicated; (2) the eruption, which consists of scattered and isolated papules and vesicles, and more rarely pustules, with their tops more or less torn by scratching; (3) the history of the case, and especially of contagion; (4) the presence of the scabies-burrow; and (5) the demonstration of the acarus by means of the microscope.

TREATMENT.—The usual plan of treating scabies is by the use of either (1) sulphur-ointment; or (2) sulphur-baths or lotions. The former is the more effectual method, although the latter may be occasionally preferred. In order to cure an ordinary case of scabies, it is simply necessary that the patient, before going to bed, should thoroughly apply and well rub in the sulphur-ointment to every part of the body, excepting the skin of the face and scalp; and in order to keep the ointment in contact with the skin, he should sleep in his under-clothes, such as drawers, jersey, socks and gloves, and in the morning take a warm bath and put on clean clothes. The process should be repeated for three or four nights, after which the ointment should be used every night for a week or ten days to those parts of the body only which are especially attacked by the disease. It is not necessary to boil the under-clothes, ordinary washing and ironing is quite effective.

The ointment should contain a drachm of sulphur to an ounce of benzoated lard, but for young children an ointment of about half this strength is most suitable. A common mistake is to use a strong sulphur-ointment for many weeks, thus producing an irritable state of skin, which is mistaken for a continuation of the scabies.

If a sulphur-bath be preferred, it may be made by dissolving half a pound of sulphurated potash in thirty gallons of water. It is necessary to repeat the bath several times at intervals of a few days.

If it be desired to treat scabies by a lotion, Vlemingx's solution may be used. It is made by boiling five gallons of water with a quarter of a pound of quicklime and half a pound of sulphur until three gallons are left. This lotion is effective, but it is apt to irritate the skin, and is not so generally useful as the sulphur-ointment.

R. LIVEING.

SCALD.—An injury to any part of the body caused by the action of moist heat, either in the form of steam or of a hot fluid. See HEAT, Effects of.

SCARIFICATION (*scarifico*, I make an incision). This is an operation in which small superficial incisions are made, through either the skin or mucous membrane, to allow the escape of blood, as in wet-cupping, or of serous fluid, in relieving dropsical effusions; or to liberate the teeth, as in difficult dentition.

SCARLATINA.—A synonym for scarlet fever. See SCARLET FEVER.

SCARLET FEVER.—**SYNON.**: Scarlatina; Febris scarlatina; Fr. *Scarlatine*; Ger. *Scharlach*.

DEFINITION.—A febrile infectious disease, of which the characteristic symptoms are a punctate erythematous rash and a sore-throat. During convalescence the skin desquamates and certain complications often occur, the most important being inflammation of the ears, kidneys and joints.

GEOGRAPHICAL DISTRIBUTION.—At the present day scarlet fever is confined chiefly to the temperate zones, being more or less endemic in Europe (especially the north-western countries and Russia), in the northern of the United States, and in parts of South America. It is not common in Australia, and is rare in the tropics, even among the European population.

ÆTIOLOGY.—One of the most remarkable facts in connection with the ætiology of scarlet fever is the decrease in its mortality during the past thirty years. Thus the average annual death-rate per million living for England and Wales has fallen from 982 (1861-5) to 241 (1886-90).

Since 1874 there has been an almost unbroken decline in the death-rate; nor has this decrease been confined to England and Wales. It has been observed in Scotland, Ireland, Sweden, Norway, Denmark, Germany, and many large continental cities and towns; there is also evidence of the same decrease in the United States of America. Some writers have suggested that it is in part due to lessened prevalence (morbidity); but that this is far from being an important factor is shown by a study of such notification-statistics as are forthcoming, not only from this country, but also from abroad. J. T. Wilson has shown that the lessened mortality is due chiefly to the lessened fatality (case-mortality). This has been observed not only in hospital cases, but also in all cases in nearly every town where notification has been in force. That there has been of recent years a change of type in the disease will hardly be denied by any one who has had sufficient opportunity for clinical observation. The most severe forms are less frequent than they formerly were. This lessened severity may perhaps be due partly to such causes as improved sanitation and a more general use of hospitals; but there are also at work factors which are unfortunately quite unknown to us, so that we have no guarantee that we have seen the last of virulent scarlet fever. At present the fatality in London is less than 4 per cent.; but as an instance of the height to which it can rise, it may be mentioned that in Stockholm in the year 1870 it was no less than 28.8 per cent.

Age.—Scarlet fever is a disease mostly of children, especially of those from two to ten years of age. The fourth year is the period of greatest susceptibility. Below one and over twenty-five the disease is uncommon. The fatality is highest during the first year, and though falling with each year, yet is high up to the fourth. It then remains low up to the fortieth, after which it again rises slightly.

Sex.—Though at all ages more cases are actually met with among females, yet males are somewhat more susceptible to the disease. The fatality is also higher among males.

Race.—The dark races of mankind are less susceptible than the white.

Seasonal prevalence.—The London notifications show that usually the number of cases begins to rise in June and, with a slight check in August, continues to rise to a maximum in October. It is slightly lower during November and falls somewhat quickly during December and January. The minimum is reached in March or April. The fatality, however, is highest during January, February and March, and lowest from July to November.

Dissemination.—Scarlet fever is highly infectious. The infection is spread chiefly in a direct manner by individuals suffering or convalescent from the

disease; but it is often conveyed by means of infected articles (clothing, books &c.) and by third persons. The virus, when harboured in clothing or other fomites, remains active for long periods of time.

The disease can also be spread by means of a milk-supply. In some outbreaks the origin of the contamination of the milk has not been ascertained; in others it has been found in a case of scarlet fever occurring among persons concerned in the collection or distribution of the milk; while in a third group it has been shown that cows from which the milk was obtained were the subjects of a certain disease of the udders, skin and viscera.

From the evidence collected in the last group of cases, it appears to be certain that cows suffer from a disease capable of giving, through their milk, scarlet fever to human beings. There is reason also to believe that swine suffer from a disorder which must be regarded as scarlet fever.

It is probable that the aggregation of children in schools is not without influence on the prevalence of the disease. At any rate there is every year a fall in the notifications, previously on the rise, during the summer holidays of the School Board in London, the rise being resumed soon after the re-opening of the schools. This fall is most marked at the school ages (3 to 13).

Defective hygiene influences scarlet fever in respect not to its prevalence, but to its type; so that insanitary surroundings aggravate an attack and favour complications. So far no definite effects have been traced to the influence of soil, rainfall or temperature, unless we except its rarity in the tropics.

MORBID ANATOMY.—Beyond the local faucial lesions there is little to be seen *post mortem* by the naked eye. During the eruptive period a microscopic examination of the skin reveals hyperæmia and œdema of the cutis vera, with proliferation of the cells of the Malpighian layer of the epidermis. Besides the cervical, other lymphatic glands, especially the mesenteric, may be enlarged, and occasionally the Peyer's patches are slightly inflamed. In septic cases the spleen may be enlarged. During the acute stage the kidneys present little, if any, changes, at the most a slight infiltration of the interstitial tissue with leucocytes. In cases of nephritis the inflammatory changes affect chiefly the glomeruli and the interstitial tissue of the cortex.

PATHOLOGY.—There can be little doubt that the essential cause of scarlet fever is some kind of micro-organism, which probably gives rise to a general infection, and not to a local one with secondary toxæmia, as in diphtheria. The disease has been produced by the inoculation of healthy individuals with the faucial and buccal secretions of a scarlet-fever case.¹ More than one observer has believed that he has discovered the specific organism. According to Klein it is a particular streptococcus, which he isolated in 1886 from the diseased udders and viscera in an outbreak attributable to the milk of certain cows on a farm at Hendon, and which he has since that date found in the blood, tissues, and skin of cases of human scarlet fever. This organism, which is the same as one subsequently described by Kurth under the name of *Streptococcus conglomeratus*, differs, according to Klein, from the *Streptococcus pyogenes*. When

grown in broth the fluid remains clear, and the organism forms one large or several small conglomerate masses at the bottom of the vessel or test-tube. Gordon has found it in most of several cultivations taken from the tonsils of patients in different stages of scarlet fever, but never in the aural, and rarely in the nasal discharge: a peculiar fact when we remember that there is good reason for believing that the nasal discharge is particularly infectious. If this micro-organism be injected into calves or mice, general infection takes place; and the organism in question can be recovered from the blood and tissues of the affected animals. There is no clear proof, however, that this general infection is the same disease as human scarlet fever; and we have further to remember that it has been established that in the latter disease cocci are present in the fauces and elsewhere, some of which are the cause of the septic and suppurative complications, though not of the disease itself. The *S. conglomeratus* (as well as another coccus to be immediately referred to) may after all be only one of these accidental organisms. More recently, Class has described an organism which he believes to be the cause of scarlet fever. It is a diplococcus, which, when grown on glycerine-agar, to which 5 per cent. of sterilised black garden-earth is added, assumes the form of a large gonococcus. He states that he has isolated this organism from the fauces, blood, and desquamating skin of scarlet-fever cases. If the blood of a patient convalescent from scarlet fever be added to the culture-medium, the organism is prevented from growing. The diplococcus is pathogenic to mice, guinea-pigs, and swine. In white swine it gives rise to an attack of fever with an erythematous rash which is followed by desquamation. The disease thus produced by inoculation is infectious, so that other animals of the same kind contract the disease naturally from those artificially infected.

In connection with these experiments it may be mentioned that Behle has reported an outbreak of a disease in pigs, which accompanied an epidemic of severe scarlet fever among children in the same locality. The chief symptoms were angina, erythema, albuminuria, and desquamation. A healthy pig was inoculated with the blood of a child suffering from severe scarlet fever in this epidemic, and the animal was fatally attacked by a disease which had exactly the same symptoms.

It must be left to future investigators to decide which, if either, of the two organisms above mentioned is the specific cause of scarlet fever.

CLINICAL HISTORY.—The incubation-period is usually of two or three days' duration. It is not infrequently shorter, but rarely longer. Probably it never exceeds five days.

Prodromal period.—The most constant symptoms are vomiting, a rise of temperature, headache, and sore-throat. Occasionally, especially in adults, a sore-throat is complained of for a day or two before the other symptoms appear. In mild cases there may be no vomiting; on the other hand, in a few cases vomiting may be so frequent as to induce within a few hours a serious state of collapse, even though the subsequent course of the attack is by no means severe. A rigor is rare, but the patient may feel chilly. Children are restless and refuse their food. The pulse-rate usually rises considerably (120 to 140 a minute). The tongue is coated with a white fur; sometimes there is diarrhœa.

¹ These inoculations were most unjustifiably performed by Stickler, of Orange, New Jersey, U.S.A., in the hopes of finding a protective virus in the secretions.

After a few hours the initial vomiting ceases, but the other symptoms persist till the appearance of the rash, which usually comes out within twenty-four hours from the onset of the attack. In not a few of the mild cases the rash is observed as early as any of the symptoms; on the other hand, in some of the most severe, its appearance is delayed till the third, fourth, or even fifth day. As a rule the character of the prodromal symptoms corresponds with the severity of the attack.

Three forms of the disease can be distinguished clinically; but it must be remembered that between them are gradations, so that no hard and fast line can be drawn.

1. *Scarlatina Benigna*, or Simple Scarlet Fever.—In this form all the symptoms are of moderate intensity. The rash, though it usually invades the trunk and limbs, may be limited to a portion of the former, or may be absent altogether. It lasts for three or four days, but sometimes for a much shorter time, even for less than a day. While the rash is coming out the temperature is moderately elevated (100° to 102° F.). With the fading of the rash the temperature gradually falls, and reaches the normal at the end of three to seven days. There may be no sore-throat, and the fauces may appear normal. But sore-throat may be present with little if any visible lesion; though there is usually tonsillitis with or without inflammation of the soft palate and pharynx. These structures may be smeared over with loose muco-purulent exudation, and slight superficial ulceration may occur. The cervical glands are moderately enlarged and painful in cases where the tonsils are inflamed. Complications and sequelæ occur less frequently than in the more severe forms of the disease; yet it must not be forgotten that most serious affections, such as nephritis and otitis, do occasionally follow even the mildest cases, especially when neglected. Desquamation may be delayed till the end of three weeks, and may be partial or absent.

2. *Scarlatina Anginosa* (Septic Scarlet Fever).—The prodromal symptoms are usually of some severity; but occasionally a case, at first of the benign form, after a few days becomes septic and presents graver symptoms. The distinguishing feature of this variety is the faucial affection. There is, in the first instance, general and acutely painful inflammation of the fauces, which may be swollen to such a degree as to impede deglutition or respiration. A viscid secretion fills the mouth and naso-pharynx; and pultaceous, sometimes membranous, exudation forms upon the tonsils and palate. The nasal passages are inflamed and blocked; and a discharge, at first thin and watery, later muco-purulent, pours from the nostrils, excoriating the adjacent skin. The cervical glands are much enlarged, of doughy consistency, and very painful. Often, too, the skin and subcutaneous areolar tissue are inflamed, and there is a hard, brawny swelling, extending in the most severe cases from one ear below the chin to the other (bull-neck). The throat is acutely painful. The faucial œdema may resolve without any subsequent lesion; but usually necrosis takes place, and yellow or grey sloughs are seen, the separation of which leads to ulceration of the tonsils, uvula, soft palate, or pharynx. In extreme cases the necrosis is so extensive as to involve the whole of the soft parts of the fauces; in those less severe portions only slough, as the uvula or a tonsil. Occasionally a portion of one or both anterior pillars sloughs, and

oval perforations are left; this local sloughing is probably caused by the pressure of the swollen tonsil against the œdematous anterior pillar. The angle between the base of the uvula and the anterior pillar on each side is a favourite seat of ulceration. The ulceration is usually slow to heal; sometimes, indeed, far from healing, it spreads, and may travel downwards past the root of the tongue to the epiglottis and larynx, in which case symptoms of laryngeal inflammation gradually manifest themselves.

The processes described above are usually in non-fatal cases of two or more weeks' duration. The temperature is at first persistently high, 102° to 104° F., or higher. After a few days or a week or two it becomes irregular; and four or five weeks may elapse, while the faucial ulceration is healing, before it resumes the normal. The pulse-rate is much increased in frequency (140 to 160). At first the pulse is full and bounding, later it is small and feeble. The rash persists for a week or ten days. Most often it is vivid and extensively distributed, but occasionally it is patchy, and it may be altogether wanting. The tongue, at first coated with thick white fur, quickly peels, and assumes the 'strawberry' appearance. Later it becomes dry and dirty. Sordes collect along the gums and lips, and the angles of the mouth are excoriated. Stomatitis with ulceration is not infrequent. In a few cases there is extensive excoriation round the anus.

During the first week or so the patient is restless, sleepless, and delirious. Wasting is rapid and extreme; and vomiting and diarrhoea, especially the latter, are often met with. Desquamation commences about the end of the first week, and is profuse. There is also considerable loss of hair. Complications are common, especially otitis media and suppurative lesions.

Death in septic scarlet fever usually occurs during the second or third week, but it may be delayed for another week or two, the patient dying of exhaustion after the febrile period is over. But in such cases it will be observed that the pulse-rate remains high, and the faucial ulceration shows little, if any, signs of repair. In non-fatal cases recovery is very tedious.

3. *Scarlatina Maligna* (Toxic Scarlet Fever).—The symptoms are severe from the onset: repeated vomiting, a very frequent pulse, a high temperature (104° to 105° F.), and prostration being the most prominent. The rash is usually profuse. The face is of a yellowish hue and waxy appearance. The sore-throat is slight, there being moderate inflammation of the fauces. The cervical glands are little if at all enlarged. The rapidity of the heart's action soon leads to cardiac failure, and there is cyanosis with sighing respiration. Muscular tremor is present. The tongue is covered with dry fur. At first the patient is delirious, but soon becomes comatose; in a few cases, however, the mental condition remains clear to the end. This form of scarlet fever almost invariably proves fatal, death taking place on the fifth or sixth day; occasionally earlier, even on the first or second, before the rash has come out or fully developed.

Hæmorrhagic scarlet fever, in which there are hæmorrhages into the skin and from the mucous membranes, is very rare at the present day.

Other forms of scarlet fever.—Cases are not infrequently met with, especially during epidemics, in which the patient presents no other symptoms

beyond those of a slight febrile attack with sore-throat. Instances have been recorded when such cases have given rise to typical scarlet fever in susceptible persons; hence they may be regarded as *abortive* cases.

Occasionally cases are observed where the chief feature of the attack is a continued fever of four or five weeks' duration, the throat-affection being moderate and the constitutional symptoms not, as a rule, severe. These may be regarded as *prolonged* examples of the benign variety. Occasionally, however, they terminate fatally from exhaustion or some complication.

As for the so-called *surgical* and *puerperal* varieties, the writer sees no reason for believing that they differ from the ordinary forms of the disease. Children the subjects of 'surgical' affections are not more likely to take scarlet fever than those who are suffering from 'medical' ailments; except, perhaps, in the case of burns or scalds. Scarlet fever of the puerperal state is rarely met with, and not necessarily severe.

SYMPTOMS.—The *rash* of scarlet fever consists of two elements, a diffuse erythema and very small distinct spots or puncta. The puncta generally appear earlier than or as soon as the erythema, though occasionally the erythema is visible for some hours before the spots. The papular or punctate element outlasts the erythema, especially in those regions where the skin is naturally coarse, as on the extensor surfaces of the extremities, where the papules often consist of inflamed hair-follicles. The rash is usually most intense about the flexures of joints, where, as also upon the upper part of the chest, it may be petechial. It appears first on the neck and chest, but quickly spreads to the rest of the trunk and upper and lower extremities. It affects also the skin just in front of and behind the ears, reaching the temples; but it spares the scalp, face, palms, and soles. In marked cases it attains its height on the third or fourth day, after which it gradually fades. The colour varies from a faint pink to a bright vermillion. In cases where the circulation or respiration is embarrassed it is of a dusky red or even purplish hue. In some cases of the septic form the rash is morbilliform on the extremities and consists of papules and macules, affecting also the palms and soles; but on the trunk the ordinary character of the rash is retained, and the face is not affected. The face, though always free from rash, is often deeply flushed, with the exception of the area round the nostrils and mouth, which is pale (circum-oral ring of pallor). Occasionally the conjunctivæ are injected.

The rash is most intense in the severe forms of the disease; the skin may then be slightly tumid, especially on the hands, causing the sensation of a tight glove. Miliaria are sometimes seen in large numbers on the wrists and hands, less frequently on the trunk and legs.

The *tongue* is at first coated with white creamy fur, composed chiefly of epithelium in process of being shed. The papillæ are enlarged, and stand out as red points projecting through the white fur. The latter separates along the sides and tip of the organ, and in patches on the dorsum. When all the fur has separated the tongue is left red, raw and moist, with the enlarged papillæ standing out as glistening points. This is the condition known as 'strawberry tongue,' which, though occurring more often in scarlet fever than in any other disease, is

by no means constant in or confined to that affection.

Desquamation is one of the most characteristic features of scarlet fever. It usually commences about the seventh to tenth day; in mild cases, however, it may be delayed to the end of the third week, while in severe it is observed as early as the second or third day. Peeling usually starts about the ears and cheeks; but very soon it is observed on the neck, upper part of the trunk and arms, and lastly upon the rest of the trunk and extremities. Occasionally it commences in the pubic region. The process is usually completed within from six to eight weeks, but occasionally within a shorter period. The palms and soles are the last places to become free from desquamation; indeed, they may go on peeling for three or four weeks after the rest of the skin has become clear. Desquamation is usually universal, even though the rash is not. It is most free in cases where the rash has been intense and the fever acute; but it may occur and even be profuse when there has been little or no rash. On the other hand, a few mild cases of scarlet fever, even with a considerable rash, do not apparently peel at all.

The desquamation assumes three forms, which may often all be observed in the same patient. In the first, the most common and diagnostic variety, the process commences with the appearance of minute white points on the skin, caused by the separating horny layer of the papillæ. These white points are rubbed off and give place to small 'pinhole-like' rings, which, enlarging, coalesce with one another so as to give rise to a general flaky desquamation. This 'pin-hole' form does not occur on the face, where the peeling is almost always of the second variety, which consists of fine branny or powdery scales, such as may follow any erythema. This kind is observed especially in infants and mild cases. Lastly, large irregular areas of epithelium may be shed entire, leaving the skin pink and tender, but very rarely raw. In such cases the skin may separate from the fingers like a glove. Very seldom are the nails cast off.

The *urine* during the febrile period is high-coloured, somewhat diminished in quantity, and deposits urates. It may contain a trace of albumen, which is not necessarily a herald of any subsequent nephritis.

The *skin* is usually dry, and in severe cases frequently hot. During convalescence, after peeling, patients are subject to frequent perspiration.

COMPLICATIONS.—*Otitis media* is the most frequent of all the complications. It is met with most often in the septic form of scarlet fever, arising during the period of the faucial ulceration. But it may occur in mild cases and during convalescence. Children under five are most prone to it. Otitis nearly always arises through the inflammation spreading up the Eustachian tube from the throat. Usually the first sign is a thin watery discharge from the ear, but earache, tenderness of the ear, and pyrexia may precede the discharge for a day or two. After a time the discharge becomes muco-purulent and often offensive. There is nearly always some loss of hearing, which, however, is usually neither complete nor permanent.

In the majority of cases under appropriate treatment, the discharge ceases in a few weeks. Occasionally a mastoid abscess or more serious

complications (inflammation and necrosis of the temporal bone, especially the mastoid process, meningitis, pyæmia, and cerebral abscess) occur early in the course of the otitis. More commonly, however, they are met with after the disease has become chronic. Complete deafness, usually due to implication of the internal ear, with in young children consequent dumbness, is at the present day very rare.

Inflammation of the cervical glands is most common in the septic form of the disease during the acute stage, but it may occur in the milder cases and during convalescence. Suppuration often results. Sometimes, especially during the early period of the attack, the *skin and subcutaneous tissue of the neck* are also involved, and extensive sloughing, especially of the latter, takes place. In a few cases, mild as well as severe, all the superficial glands can be felt to be enlarged, as in rubella.

Nephritis is the most characteristic complication or sequel of scarlet fever. Its incidence varies much in frequency in different years, from 2 to 17 per cent. It is more common in children than in adults, especially between the ages of five and ten. Males are more liable than females. It follows mild nearly as often as severe attacks; and there is no reason to suppose that it is due to 'cold' or to allowing the patient to get up too soon. It most commonly sets in at the end of the third, or the beginning of the fourth, week, but may occur as early as the tenth day, or as late as the sixth or seventh week, when the patient has almost recovered from the attack of scarlet fever. It is rarely seen earlier or later than these periods. The onset is usually sudden. There is headache, nausea or vomiting, a rise of temperature (100° to 105° F.), and occasionally shivering. The urine next voided is found to be smoky in appearance and to contain albumen, blood-discs, and renal casts. The amount of urine is at first much diminished; the pulse-rate is accelerated (but the arterial tension is not usually increased), the tongue is furred, and the bowels are confined. In most cases the acute symptoms pass off in two or three days, but there is irregular pyrexia, which may persist for a week or two, the temperature-curve being very like that of pyæmia. At first there may be slight puffiness about the eyes and ankles, and a little œdema of the loins and scrotum, but pronounced anasarca is uncommon, save in neglected cases. Recovery is gradual, and is not complete for five or six weeks.

In some cases the onset of nephritis is insidious, and its presence is revealed only by albuminuria. Hence the great importance of frequently examining the urine of the convalescent patient. Later more definite signs may appear. These cases are apt to be very tedious and to resist treatment.

In both forms convulsions, coma, and occasionally other cerebral symptoms (squint, retraction of head, &c.) may occur. Acute nephritis sometimes leads to persistent albuminuria, less often to chronic nephritis. Quite half the cases are accompanied by some other complication, such as otitis, adenitis, and secondary sore-throat. Death occurs in about 7 per cent. of the cases, and is due to uræmia, œdema of the lungs, acute cardiac dilatation, or some additional complication, such as pneumonia.

Albuminuria may occur during the febrile period, at the end of which it disappears, not being usually followed by nephritis. Transient albuminuria of

two or three days' duration is not uncommon during convalescence, especially about the time when nephritis is expected. Such cases should always be regarded with suspicion and treated as commencing or slight and transient nephritis, which indeed they may be.

Arthritis may occur under three different pathological conditions. The *first* and most common is the so-called *scarlatinal rheumatism*. This arises about the end of the first week, especially in cases of the septic form of scarlet fever. It is most commonly met with in adolescent patients, particularly females. In many of the patients a history of a previous attack of rheumatic fever can be obtained. The small joints are affected rather than the large, especially the joints of the wrist and hand. Occasionally the inflammation shifts about from one joint to another. Neither the local nor the constitutional symptoms are as a rule severe, pain in the joints and slight pyrexia being often the only signs. The disease is of short duration. Slight relapses may occur. Suppuration is very unusual.

The *second* form of arthritis is rare. It also occurs in the septic variety of scarlet fever. One or two of the large joints become inflamed and suppurate; but there is no evidence of general pyæmia, and under appropriate treatment recovery ensues.

Lastly the joints, usually several, may suppurate in consequence of general pyæmia, either in septic scarlet fever or as the result of such a complication as disease of the ear. The onset of the joint-affection is often very insidious.

Convalescent children are often troubled with *eczema*, especially of the skin about the ears and around the nostrils and lips; a discharge from the ear or nose is sometimes the cause of this. *Rhinitis* may arise during convalescence; in many of the cases the diphtheria-bacillus can be found in the discharge.

Secondary or accidental rashes are not uncommon, especially a papular and blotchy erythema, affecting chiefly the extensor surfaces of the limbs in septic cases. Sometimes such a rash appears to be due to nephritis.

Diphtheria is not infrequently found associated with the acute stage of scarlet fever; but it more often arises during the period of convalescence. It is worth noting in connection with this complication that more than one observer has found the Klebs-Loeffler bacillus in a certain proportion of scarlet-fever cases during the first few days of their illness, even though the fauces have not presented any appearance suggestive of diphtheria.

Of the remaining complications the least infrequent are *tonsillitis*, occurring during convalescence; *ulcerative stomatitis*; and *bronchitis* and *bronchopneumonia*, the two latter especially in very young children. Still more uncommon are *laryngitis*, *lobar pneumonia*, *pleurisy*, *endo- and peri-carditis*, *cardiac dilatation*, *ophthalmia*, *pyæmia*, *meningitis*, *chorea*, *jaundice*, *peritonitis*, and *mania* (p. 770). Both *relapses* and *second attacks* may occur; the former are met with in less than 1 per cent. of hospital cases.

DIAGNOSIS.—Difficulties arise because some cases, usually mild attacks, present little or no rash, or because a rash closely resembling that of scarlet fever is met with in other conditions. In mild cases of the former kind a diagnosis from other throat-affections is often impossible without the occurrence of rheumatism, nephritis or the characteristic de-

desquamation. In severe cases without a rash (usually mistaken for diphtheria) initial vomiting, a very frequent pulse, continuous high temperature, delirium, much œdema and deep ulceration of the fauces, are signs of scarlet fever. Of the latter kind the prodromal rashes of measles, varicella and small-pox, the rashes of measles, rubella, and of septic conditions, rashes following the administration of certain drugs and the use of purgative enemata, and certain idiopathic erythematous rashes, are not infrequently sources of error. The most difficult to differentiate is the scarlatiniform variety of rubella (Clement Duke's 'Fourth disease'), which is exceedingly like scarlet fever of the mild form, especially if there be little or no sore-throat. If the rash affects the face scarlet fever can be excluded. Initial vomiting, a peeling or a 'strawberry' tongue, and circum-oral pallor, are in favour of scarlet fever. The universal though moderate enlargement of the superficial lymphatic glands is in favour though by no means diagnostic of rubella. In some cases, however, it is necessary to suspend the diagnosis, which may be cleared up by the occurrence of other cases or by the onset of some characteristic complication, or by subsequent 'pin-hole' desquamation.

In the other diseases mentioned above a careful attention to the symptoms other than the rash and a consideration of the conditions under which the rash has occurred will usually lead to a correct diagnosis.

The presence of Filatow's or Koplik's spots on the buccal mucous membrane is of great value in the diagnosis of measles (morbilli).

PROGNOSIS.—The younger the patient the more unfavourable the prognosis, the disease being more fatal in children under five than at other ages. The benign form rarely gives rise to any immediate anxiety, but it is to be remembered that most serious complications (e.g. nephritis, or otitis media, with necrosis of the temporal bone), often follow the very mildest attacks. All cases of the septic form should be viewed with apprehension; and nearly every case of scarlatina maligna is fatal.

Of individual symptoms the following are the most grave: repeated vomiting; great frequency of the heart's action maintained for several days (in adults a very frequent pulse is of graver import than in children; but in some cases of septic scarlet fever in children the temperature falls and the local disease of the fauces, though not healing, does not get worse, yet the pulse-rate remains high, and this is an unfavourable sign); coma; sighing respiration, and an alteration of the respiratory rhythm.

In cases of nephritis, scanty urine and coma are very unfavourable events, but most cases of convulsions recover. Those cases of nephritis, which commence abruptly, are usually of shorter duration than those whose onset is insidious. Chronic nephritis does not often ensue.

Patients who are the subjects of tubercular lesions are very liable to have them aggravated by an attack of scarlet fever, and also to develop nephritis and other complications.

TREATMENT.—In the mild forms of the disease it will be sufficient to spray or irrigate the fauces and nasal passages with a solution of boric acid (saturated) or chlorate of potassium (gr. xv ad 3j) or weak chlorine-water, three or four times a day. In cases where the throat-lesions are severe these solutions may be used every two or three hours. If

there is much exudation alkaline solvent applications are useful, such as solution of bicarbonate of sodium.

All the above solutions may be used as gargles, sprays, or irrigations, being applied in the latter case with a ball- or Higginson's-syringe; but in some cases it will be found to be more efficacious to swab out the fauces occasionally with pledgets of cotton-wool lightly wrung out of the solution. In septic cases weak solutions of carbolic acid (1 in 40) or corrosive sublimate (1 in 2,000) may be employed, also by means of swabs. When there is much pain the patient is usually relieved by the application of warm fomentations or poultices to the neck, but in some instances more relief is afforded by sucking ice.

During the febrile stage the diet should consist of milk and water, beaten-up eggs, arrowroot, cornflour, &c.; a little beef-tea, and chicken- or mutton-broth may also be allowed. When the temperature has regained the normal level bread and butter, fish, eggs, custard and milk-puddings may be added, and gradually, after a few days, fish, poultry, and meat. Lemonade, soda-water, tea and coffee, and such fruit as oranges and grapes may be taken at any time. The room should be well ventilated and kept at a temperature of 55° to 60° F. The patient may be allowed to sit up in blankets after the temperature has been normal three or four days, provided no complications have arisen; and after another week he may be fully dressed. If the weather is warm and otherwise favourable he may in another three or four days be permitted to go out of doors for a short time daily. A warm bath every evening, beginning at the time of the patient's first getting up, will facilitate desquamation.

Mild cases need no physic, unless it be for the purpose of securing a daily action of the bowels, for constipation must certainly be avoided. The best treatment for pyrexia and delirium is the tepid wet-pack or sponging with tepid water. Even if the temperature be not very much lowered by these measures, sleep is induced. Of drugs, anti-febrin in small doses repeated at intervals if necessary is the most safe, and has much the same effect. When there is prostration stimulants are required, such as strychnine, alcohol, ether, or ammonia.

For the treatment of complications, the reader is referred to the special articles dealing with these affections. If the patient is treated at home, all precautions should be taken to prevent the spread of infection. The patient should be kept isolated for at least six weeks, and should be released at the end of that time only if no complication exists and peeling (except of the soles) has finished. Otherwise the patient should be kept isolated for a longer period. Patients discharged from fever-hospitals, and also those treated privately, sometimes remain infectious for a longer period than six weeks, even though they appear to have quite recovered their health, as is shown by their giving rise to fresh cases (the so-called 'return cases') when they are allowed to mix with susceptible children. The most infectious condition appears to be the nasal discharge, which often persists for a long period. It is most desirable that this as well as other discharges and morbid conditions should be cured before the patient is allowed to associate with other children. *See* also pp. 769 and 474.

EDWARD W. GOODALL.

SCHANDAU, in Saxony.—Chalybeate waters. See MINERAL WATERS.

SCHINZNACH, in Switzerland.—Sulphur-waters.—See MINERAL WATERS.

SCHLANGENBAD, in Germany.—Simple thermal waters. See MINERAL WATERS.

SCHMECKS (Tatra-Füred, Tatrabad), in Hungary (Alt-Schmecks and Neu-Schmecks, and Unter-Schmecks).—Acidulated alkaline waters, and climatic health-resort. See MINERAL WATERS.

SCHOTT-TREATMENT, The.—During recent years much interest has been centred in the Schott-treatment of disorders of the heart and circulatory system as practised at Nauheim, by the employment of its baths with special exercises. Nauheim, which has long been noted for its famous Springs, lies at an altitude of over 400 feet on the N.E. slope of the Taunus range. Some of its waters are used for drinking, others for baths. Thus the Kur-Brunnen and the Karls-Brunnen Springs are chiefly for internal administration; the Ludwigs-Brunnen and the Schwalheimer-Brunnen are valuable table-waters. Of the waters employed for bathing purposes the Great and Little Sprudel (82°–95° 5' F.) are rich in carbon dioxide and the chlorides of sodium—2 to 3 per cent.—and calcium, with traces of salts of other metals such as iron, arsenic, barium, and lithium.

The varieties of baths employed are as follows: (1) Simple brine-baths, weak and strong; (2) Effervescent brine-bath, rich in CO₂ and salts; (3) Effervescent current-, surf- or wave-bath. The kind, number, strength and temperature of the baths prescribed are controlled, and vary with each case, according to the judgment of the physician after his observation of the symptoms and physical signs presented by the patient. After each bath a sufficient period of repose is enjoined, and the bath is generally intermitted every few days. Many patients go to Nauheim for the baths only, others for the baths and 'resistance' exercises.

The Schott-treatment at Nauheim has been as intemperately decried by some as it has been unwisely recommended by others. A prudent and judicious selection of cases is imperative, inasmuch as important physiological effects on metabolism and on the circulatory system result from its employment. See p. 1470.

The Schott-treatment by gymnastic exercises is employed in conjunction with the Sprudel baths. It consists of a scientifically planned system of voluntary movements on the part of the patient combined with resistance offered by the hand of the physician or skilled attendant. By such measures, carefully graduated, commencing failure of compensation in cardiac valvular disease is often successfully combated; a variety of cardiac symptoms ameliorated; and some cases of the heterogeneous group of functional cardiac disorder much benefited.

Clinical experience shows that some cardiac cases are benefited by rest, others by exercise, some by drugs, some by hydrotherapeutic measures. Hence before prescribing a course of the Schott-treatment at Nauheim, it is necessary to ascertain the condition of the cardiac muscle, the degree of dilatation and the condition of the arterial walls.

The change to bracing air and sunshine, the regulated mode of living, the carefully planned daily

routine, the avoidance of any source of fatigue, the psychical influences counteracting introspection, and—greatest change of all—the change to repose of mind and body, all constitute important factors of the treatment and possibly explain why the Schott-treatment at Nauheim has been more successful than its imitations elsewhere.

JOHN HAROLD.

SCHÜLS.—See TARASP-SCHÜLS.

SCHWALBACH.—See LANGEN-SCHWALBACH; and MINERAL WATERS.

SCIATICA.—SYNON.: Fr. *Néuralgie Sciatique*; Ger. *Hüftweh*.

DEFINITION.—Neuralgia in the district of the sciatic nerve.

Sciatica is signalised by paroxysmal pain in any or all of the following localities: the buttock; the back of the thigh; the knee; the front, back, and outside of the leg; and the whole foot except its inner border. The condition is frequently of peripheral origin, and is then dependent on inflammation of the nerve-trunk. Or it may arise independently, to all appearance, of any local cause in the nerve itself, and is then probably caused by some impairment of nutrition of a nerve-centre. As the diagnosis of interstitial neuritis from neuralgia is as yet not certainly established, the two conditions will be considered together.

ÆTIOLOGY.—*Predisposing causes.*—The predisposing causes of sciatica are the rheumatic and gouty diatheses; anæmia; the toxic influences of malaria, chronic alcoholism, syphilis, and lead; inherited neurotic disposition; fatigue; and a damp and cold climate.

Exciting causes.—These include exposure of the limb to a draught of cold air for some long time, which is a common source of the affection; sitting on a cold or damp seat; over-walking; strains; concussion of the spine; the encroachment of morbid growths; blows upon or wounds of the nerve-trunk; malignant and other tumours of the pelvis; disease of the vertebræ or pelvic bones; the pressure of the gravid uterus; rheumatic or gouty inflammation of the sciatic nerve, or of one or other of its branches; syphilitic periostitis, causing a swelling which presses upon the trunk or some branch of the sciatic nerve; gumma in the sheath of the nerve.

In chronic rheumatoid arthritis of the hip-joint the sciatic nerve may sometimes be felt to have hard, knotty swellings upon it, apparently arising from a chronic perineuritis. This is associated with very obstinate sciatica.

Sciatica affects especially the middle age of life, from forty to fifty years, is rare in youth, and but seldom commences in elderly persons. It is at least four or five times as common in men as in women.

The violent manipulation of an unskilful professional rubber, employed to treat a dull aching in the sciatic region, may bring about acute sciatica.

ANATOMICAL CHARACTERS.—Nothing certain is as yet known of the character of the lesion in cases of neuralgia proper manifested in the district of the sciatic nerves.

The few changes which have been observed *post mortem*, or after removal of a portion of the nerve, in cases of sciatica have been in the direction of

thickening of the sheath of the nerve, with exudation and sometimes small hæmorrhages, the result of inflammation. It has happened, however, that in cases which during life have been supposed to be examples of neuritis, no trace of the inflammatory process has been found in the nerve itself.

SYMPTOMS.—Usually after more or less of vague discomfort in the affected limb, deadness, tingling, stiffness, or some such abnormal sensation, pain occurs in some part of the district already indicated. This is variously spoken of as shooting, darting, screwing, tingling, or burning; and the sufferer will often map out with precision the course of various branches of the nerve as the seat of his distress. Sometimes the leg is described as being numbed, or as if it were going to burst. Occasionally the feeling resembles a very faint shock of a battery. As is characteristic of neuralgia generally, the pain may be apparently spontaneous in origin, while in certain cases it is also liable to be aggravated by movement, but in no case is it *only* excited when muscular contraction takes place. It may either occur in paroxysms, with intervals of complete immunity varying from minutes to hours; or there may be more or less continuous suffering, with frequent and violent exacerbations. Throbbings or pulsations of the pain are often described. It is not, as a rule, accompanied by pyrexia. When the pain is at its height, a powerlessness of the muscles of the limbs is apt to be experienced, and this not simply on account of the distress of moving, but from an actual paresis, dependent possibly upon a depressing influence communicated to the motor centres; or, should the condition be one of neuritis, due to interruption of nervous channels. From a like cause affecting the vaso-motor fibres the limb will feel cold, not only subjectively, but to the touch.

Tender points may be met with in all, but more often in some few only, of the following situations: The posterior inferior spine of the ilium; about the middle of the dorsum of the ilium; about midway between the tuber ischii and trochanter major; the fold of the buttock; head of the fibula; behind each malleolus; in the popliteal space. There is commonly some cutaneous anæsthesia in some part of the limb, and sometimes replacing this there will be found hyperalgesia of certain patches of skin. The tactile sense proper is lowered at these points, while the sense of temperature, especially for heat, is often heightened. At the same place a weak voltaic current is felt as extremely and quite abnormally painful. Cramp of the calf-muscles is common. It is often impossible for the patient to sit, owing to the tenderness of the nerve-trunk near the tuber ischii. The act of putting on a stocking, stooping, or sneezing gives rise to great pain.

There are great differences in the course and character of the affection. In some persons the disease from the first presents the characters of a chronic affection. There is never pain sufficient, for example, to prevent sleep, and it may not arrive at such a degree of severity as to interfere much with locomotion. Indeed the sufferer may experience a certain amount of relief from moving about. But the patient is worn by a more or less constant aching in the district of the sciatic nerve, which becomes especially marked after long sitting, as on a railway journey, and most of all if the seat be somewhat hard. Fatigue of body and mental worry have great effect, in such cases, in intensifying the

symptoms, which, on the other hand, may almost entirely disappear in circumstances favourable to improved health and mental exhilaration. Sciatica of this type is prone to attack the middle-aged, to be permanent, and to acquire additional intensity with advancing years. In contrast with such cases are those which from the first exhibit the characteristics of an acute affection. The pain is so agonising that no posture affords relief, sleep is impossible, the general health greatly suffers, and the patient is placed completely *hors de combat*. The disease may last for weeks or months without material intermission, the patient being confined helplessly to his bed; and when at length the symptoms subside, the period of convalescence may be very prolonged. Or after some weeks of acute suffering, improvement may take place, only to be followed by a relapse, which is even more violent than the original attack.

Although the recumbent position is commonly that which is least irksome to the patient, yet it will happen that after a night passed in sleep attempts to move the affected limb cause it to be more painful than after walking, and there is a distressing stiffness about it.

In some persons the disease appears once, in a very severe form, it may be at a comparatively early period of life, and never recurs. In many, however, it tends to recurrence, and with such persistence that the patient will speak of 'his sciatica' as a companion which is always present, though occasionally for a term out of sight. Or it may alternate with attacks of typical neuralgia in other parts of the body; or be replaced by various functional disorders of the nervous system, accompanying an excess of uric acid in the urine.

The disease (doubtless in its neuritic form) is liable to be accompanied by considerable muscular atrophy of the limb, and a quasi-paralytic condition, which, though capable of improvement, may, exceptionally, leave the patient more or less lame for the rest of his life. Much more often the wasting which has accompanied the acute symptoms gradually ceases as these subside, and during the somewhat prolonged convalescence the limb is restored to its original size, and the lameness is no longer observed. Sciatica often follows, and is sometimes accompanied by lumbago.

DIAGNOSIS.—There are three conditions of the muscular system which are apt to be confounded with sciatica: (1) Myalgia from over-exertion of the flexor muscles at the back of the thigh. Here the pain will be found at the points of insertion of the muscles, and is only felt during their action. (2) Rheumatism in the muscles from exposure to cold. Here, again, the pain is not spontaneous, as in sciatic neuralgia, but is always dependent on muscular action. (3) A low inflammation of the loose bursal tissue which separates the large muscles of the thigh, brought about, it is probable, by the presence of some morbid material conveyed by the lymphatic vessels with which the spaces are in direct communication. Uric acid may apparently cause this affection. Sacro-iliac disease may be distinguished by its always occurring in youth; by the pain being confined to the sacral neighbourhood; and by the limb being lengthened at an early period. From hip-joint disease, especially in the slow insidious form which it sometimes takes, sciatica is not always distinguished with facility. The paroxysmal character of the pain, presence of tender

points, absence of result from forcible movement of the joint, and absence of characteristic position or shortening of the limb, will best serve for diagnosis. The lameness in sciatica may lead to the idea of spinal paraplegia, from which the disease may be distinguished by the perfect integrity of the other limb, as well as by the paroxysmal pains, tender points, and the history showing that the powerlessness was secondary to the pain. From the darting pains in the thigh consequent on renal calculus sciatica may be easily distinguished by careful examination.

The affection may be limited to a single branch, and when this is the terminal portion of the anterior tibial nerve supplying the big toe, there is a *primæ facie* resemblance to gout. It may be distinguished from this, however, by noting the absence of heat, swelling, redness, or pain on moving the joint; and the presence of a small spot, at which alone pressure is extremely painful.

Syphilitic periostitis, with inflammation of the sheath of the sciatic, may be distinguished from malignant disease by the effect of large doses of iodide of potassium. Sciatica may be distinguished from the lightning-like pains of tabes dorsalis by the presence of the patellar tendon-reflex. Should, however, the anterior crural nerve be affected with neuritis coincidently with the great sciatic, the knee-jerk may be temporarily lost. The Achilles-tendon reflex may be lost in cases of sciatic neuritis, and also, according to Babinski, in examples of purely neuralgic sciatica. It is well to remember that sciatica, properly so called, is an unilateral affection. Should it be described as affecting *both* lower extremities coincidently, examination will show that the supposed sciatica is either tabes dorsalis, or neuritis of the cauda equina, of toxic origin, or secondary to the presence of a morbid growth; or it may be due to diabetes mellitus, alcohol, or syphilis.

TREATMENT.—This should be constitutional as well as local. Gout, rheumatism, syphilis, malaria—if inquiry show that any of these conditions possibly lies at the base of the disease—will need the treatment appropriate to each. In the presence of a gouty or rheumatic basis, the most common association, the diet should be spare in meat, with milk—all stimulants being as a rule avoided. Bicarbonate of potassium in effervescence, citrate of lithium, iodide of potassium, and colchicum will be among the drugs most likely to afford relief. If the gouty character be very pronounced, it will be well to give salicylate of sodium in ten- or twenty-grain doses, dissolved in half a tumbler of water, every three or four hours. The bowels should be freely acted upon with mercurial alteratives and Carlsbad salt. If syphilis be suspected, iodide of potassium or of sodium should be given, in doses of from ten to thirty grains three times a day or oftener.

Quinine, in doses of five grains, or arsenic may be employed if there be reason to believe that there is a malarial taint.

The recumbent position is very necessary in all cases of any severity; and, should the distress be very great indeed, it is a good plan to put the patient on a water-mattress. Or the affected limb may be preserved from movement by the application of a long splint, kept in position by a flannel bandage, changed daily. Sometimes the rapidly repeated application of very hot linseed-meal poultices will

give much relief. The application of an indiarubber bottle filled with very hot water rarely fails to soothe. On the other hand, cases of threatened severity have yielded to the application for several hours of bags of ice along the course of the nerve. Occasionally, if the pain and tenderness be very exquisite, and the patient's age and condition do not contra-indicate it, the application of a few leeches to the upper and back part of the thigh is useful. The synthetical compounds, phenazone in ten-grain doses, the same quantity of phenacetin with the addition of three-grain doses of caffeine, may be employed with advantage three or four times in the twenty-four hours. The hypodermic injection of morphine gives, of all remedies, the most speedy relief; but it is a hazardous remedy for the patient, who is very apt to acquire the morphine-habit. It is best to employ a solution of one grain in thirty minims, and to inject at first three minims twice in the twenty-four hours, if necessary. The dose may be increased by one minim at a time if it be found desirable; but it is rarely necessary, except in cases of malignant disease, to exceed eight minims of such a solution. The dose and frequency should be diminished as relief is obtained. The hypodermic injection of hydrochloride of cocaine is sometimes very useful. It should be performed at the seat of pain, and endeavour made to thrust the point of the syringe into the spot of greatest tenderness. The application may be repeated, if necessary, two or three times a day, in doses of gr. $\frac{1}{8}$ to gr. $\frac{1}{4}$. Care must be taken to avoid inducing the morphine- or cocaine-habit. The bowels should be kept thoroughly relieved. Flying blisters, not larger than a five-shilling piece, one following another as it heals, not on the same but on a closely adjoining part of the skin, may usually be applied with much advantage. Even in cases dependent upon malignant disease they will often give relief for a time. Spongiopiline wrung out of boiling water, and sprinkled with the compound mustard liniment, makes a good counter-irritant, and will sometimes take the place of blistering. Massage should only be employed in the absence of acute symptoms.

The continuous galvanic current is occasionally palliative. From twenty to forty cells may be employed; and while one rheophore is applied to the sacro-iliac synchondrosis, the other is dropped into a tub of warm salt water, in which the patient places his foot. The strength should be arrived at gradually, and the application continued for ten minutes at a time. Then, without removing the rheophores, the strength of the current must be gradually reduced to zero. Without this precaution a shock will be given, which is undesirable. The electrical application should not be employed during the very acute stage, but rather in cases of moderate severity, or where the most formidable suffering has subsided. It can be repeated at frequent intervals during the day. The current may be conveyed into a bath if preferred. One rheophore is allowed to fall into the bath, the other is held by the patient above the level of the water, which should be of a temperature of 95° F., and should contain some salt. In some cases, after acute symptoms have subsided, good has appeared to be done by enveloping the limb in a flannel bandage, over the folds of which sulphur is sprinkled. In other cases it is said that oil of turpentine taken internally has cured where other remedies have failed. In obstinate cases the sheath

of the nerve may be punctured in a few places with a sharp needle. See ACUPUNCTURE.

The baths of Buxton, Wiesbaden, Wildbad, Royat, and Gastein have been found useful in treating sciatica; and artificial hot-air or vapour-baths may be beneficial in some instances.

The sciatic nerve has been cut down upon and stretched with advantage in several cases of confirmed sciatica. The bloodless stretching of the nerve is, however, much to be preferred. This can be accomplished by flexing gradually at the hip-joint the extended lower limb of the patient, who lies upon his back, care being taken that the knee-joint is not flexed. See NERVES, Diseases of.

T. BUZZARD.

SCLEREMA NEONATORUM.—This name is applied to a condition of the skin and subcutaneous tissue, occurring congenitally or within a few days of birth, producing the hardening of the cutaneous tissues, which has given rise to a synonym of the disease—viz. the hidebound disease.

It may be present at birth or appear within the first ten days. The skin becomes of a pale white or waxy colour and has the feeling of hard leather. The uniform whiteness disappears in time and the tint becomes livid or even mottled. The subcutaneous tissue, as well as the skin, is also condensed, so that the integuments become bound down to the underlying structures. In this way a firm board-like texture is imparted to the integuments. The disease, as a rule, commences in the lower extremities, spreads upwards over the trunk, especially on the back, then gradually over the rest of the body, the face as a rule being the least affected. In all other parts the skin becomes quite board-like, so that the joints are fixed and the whole figure quite rigid. Even when the face is least affected, the cutaneous structures are so rigid as to prevent sucking or swallowing and the mouth cannot be opened. The nutrition of the infant rapidly fails, the respiration becomes very shallow and infrequent, the child is unable to cry and only produces a feeble moaning, and in a few days the vitality is so diminished that death occurs. Histological examination throws little light upon the nature of the disease. The epidermis is scarcely altered, though it may be thinner than usual. The subcutaneous tissue is firm and condensed, though no round-cell infiltration is present. The vessels of the cutis are small and appear to be contracted. The connective-tissue trabeculae, in the subcutaneous tissue, seem to be more than ordinarily well-marked. The causes of the disease are quite unknown, though it is stated that it is more apt to occur in children born under circumstances of poverty and defective hygiene. The prognosis seems to be inevitably fatal.

Œdema Neonatorum.—This is a form of firm subcutaneous œdema, affecting new-born infants. The disease commences within the first few days of life, generally in feeble children. The lower extremities are as a rule first affected. The disease advances by continuity or as separate patches over the trunk, especially affecting the back and buttocks. The swollen parts show firm œdema and pit as a rule with difficulty; they are red or livid in tint. The whole body may become affected in this way, or only small areas of disease may appear. In the more extensive cases rapid loss of strength is noted, the circulation is enfeebled, breathing becomes shallow, and death occurs from

vital depression or some other malady, such as diarrhoea, bronchial catarrh, &c.

Anatomical examination shows extensive changes in the affected skin. A large amount of serous effusion occurs in the connective tissues, and they seem much firmer than normal. The epithelium appears to undergo only slight change, but may be thinner than is usual. The internal organs have been found affected in various ways, but these alterations apparently have no distinct relationship to the disease. The liver has been found to be enlarged, the lungs catarrhal and congested, and nephritis has been found present.

The nature of the disease is obscure. It occurs in prematurely born children, in those who are feeble, or who are born under bad hygienic conditions. It seems, therefore, to have relationships to many debilitating causes.

The prognosis of the disease is not so hopeless as in sclerema neonatorum. In cases of œdema, if the whole surface is affected, the outlook is exceedingly serious. Many cases of localised œdema of the same type are seen, which gradually recover. As a rule, the course of the disease is longer than in cases of sclerema neonatorum.

TREATMENT.—Efforts should be made to retain the body-temperature by appropriate clothing and external warmth. The child may be wrapped in cotton-wool in a warm room, or placed in an appropriate incubator. As a rule, on account of the face being affected, the child is unable to suck, and must be then fed artificially, by passing a tube into the pharynx. Milk in some form is obviously the only diet permissible. Much advantage has been obtained by the use of gentle massage of the affected parts, especially when the disease is localised.

JAMES GALLOWAY.

SCLERODERMIA (*σκληρός*, hard; and *δέρμα*, the skin).—**SYNON.** : Addison's Keloid; Morphea.

DEFINITION.—A chronic disease of the skin, circumscribed or diffuse, in which an excess of fibrous tissue leads to hardness, change of colour, and by contraction to stiffness and immobility.

ÆTIOLOGY.—Scleroderma is a rare disease of the skin, which occurs at all periods of life, but more frequently in young adults and in middle age. It is more frequent in women than in men. Little is known as to its causation; but rheumatism, especially of the joints, has been known to precede the attacks, and it has sometimes followed exposure to wet and cold. It attacks persons otherwise in good health. The circumscribed form sometimes follows the course of cutaneous nerves, and for this and other reasons it is regarded by some authors as essentially a tropho-neurosis. So far, however, the correctness of this view cannot be considered as having been definitely established. In some cases the circumscribed form has been referred to irritation caused by blows, blisters, pressure, and other causes.

ANATOMICAL CHARACTERS.—The essential structural changes consist in an increased formation of connective tissue, the new elements following especially the lines of the vessels. The number of elastic fibres appears increased in certain parts of the skin. The formation of new fibrous tissue takes place not only in the corium, but in the panniculus adiposus, and extends to the deep fascia and periosteum. In its overgrowth the fat disappears, and the smooth muscular fibres appear to be hypertrophied.

The papillary body is rendered more dense than usual by bands of connective tissue, and there is an increase of pigment both in it and in the rete. Otherwise the epidermis is unaltered. In the early stage the effusion leads to hypertrophy of the cutis, but later the contraction of the fibrous tissue causes atrophy of the integument and narrowing of the blood-vessels. At first the sebaceous and sweat-glands are unchanged, but they are afterwards atrophied. Sometimes there appears to be a dilatation of the sweat-glands in the early stage. In the extreme atrophic stage of diffuse sclerodermia the compression leads to more or less disorganisation of ligamentous tissues.

DESCRIPTION.—Sclerodermia is found in two well-marked forms—*diffuse* and *circumscribed*, which in their typical development differ so much from each other in appearance that they were formerly considered to be distinct diseases, the circumscribed form being known and usually described as *morphea*. Transition-forms are, however, sufficiently common to establish the nosological identity of the two varieties of the affection.

Circumscribed sclerodermia.—A developed patch of circumscribed sclerodermia is usually characterised by a rounded area of white or yellowish-white skin, which in some cases looks like smooth ivory. Around this patch there is a yellowish or brownish pigmentation. The white surface is usually firm to the touch, brawny, and difficult to pinch between the fingers. The sensibility is unimpaired. The earliest stage in the development of such an area is characterised by hyperemia, small dilated blood-vessels shining through the surface. The disease may begin in one or more such isolated patches, which, as they become circumscribed, are surrounded by a well-marked lilac border of minute blood-vessels. Such a patch may remain for months or years without much change, or it may undergo involution, which sometimes takes place quickly, leaving the skin healthy; or, on the other hand, it may become atrophic, the skin becoming thin, parchment-like and cicatricial.

These patches occur in various parts of the body, but most frequently on the neck, face, chest, trunk, arms, and thighs. They are unsymmetrical. There is a sufficient number of cases on record in which the changes in the cutis have followed the track of certain nerves, which would indicate some relation to nerve-distribution. The affected skin does not sweat. There are usually no subjective symptoms, but there may be itching, or tingling, occasionally even pain. Circumscribed sclerodermia sometimes occurs in the form of bands, which are usually depressed below the surface and more or less adherent to underlying parts, and sometimes extend for a considerable length.

Diffuse sclerodermia.—This occurs on different parts of the body, but more usually in its upper half. It may be limited to certain parts, the skin between them remaining normal, or it may be spread over large areas of the integument, such as the back, abdomen, limbs and face. It may develop after a short period of malaise, but as a rule the process is at the beginning not observed by the patient, his attention being first called to it by stiffness of the skin; or pigmentation may be early observed. Stiffness is usually first felt about the neck, chest, shoulders or arms, and spreads with more or less rapidity towards the limbs, the border being usually ill-defined, although to some extent

perceptible to the touch. Doughy infiltration or erythema may precede the induration. As the skin becomes hard and stiff, mobility is affected, the joints becoming fixed and the face immobile. The patient cannot open his mouth widely, and the affected surface of the body has been compared to that of a frozen corpse, without, however, being cold. The skin does not pit, and cannot be pinched up. The patient is usually hidebound. The respiration is impaired, the breasts flattened, the limbs and fingers more or less flexed. The disease may even extend to the mucous membranes. The contraction of the skin leads to the disappearance of wrinkles, old persons looking younger than their years. The surface is generally smooth and shining, but sometimes dry and desquamating, the skin being pale, whitish and waxy. The disease may reach its full extent in a period of weeks or months.

The temperature of the affected skin is on the whole normal, sometimes being slightly above and sometimes below the average. Pressure is felt, and the tactile sensibility is usually unaffected, but sometimes weakened. Secretion of sweat may be diminished. The sebaceous secretion is usually normal. The nutrition of the affected skin being impaired, it is liable to inflammation and ulceration from the ordinary sources of irritation. The limbs, from compression of the continually contracting skin, gradually become smaller. The palms and soles are rarely affected.

The course of the disease varies considerably. In the early stages a spontaneous cure may occur, involution sometimes taking place at one part while new development is showing itself at another. When shrinking and atrophy have proceeded to an extreme extent life becomes threatened. The general health in the earlier stages of the disease does not seem to be notably affected; but as atrophy and shrinking proceed, the patient falls into a condition of marasmus, and death occurs from intercurrent complications of various kinds. Kaposi refers to a case of Strassmann's, which had lasted thirty-one years without the health being materially affected.

DIAGNOSIS.—Sclerodermia is not likely to be confounded with other diseases characterised by hypertrophy and œdema. The tension, rigidity, hardness, and shrinking of the diffuse form are distinctive; in the circumscribed form the pigmented and hyperæmic zone surrounding the waxy patches distinguishes it from leprosy and vitiligo. In anæsthetic leprosy, anæsthesia is characteristic. In atrophic conditions there is a history of a previous hypertrophic stage.

PROGNOSIS.—Recovery may take place so long as the atrophic stage has not set in, improvement showing itself within a period of weeks or years. In the atrophic stage the prognosis is unfavourable, death being due to complications of various kinds.

TREATMENT.—Tonics, such as iron, quinine, vegetable bitters, cod-liver oil, and arsenic, are believed to assist a tendency to recovery when it is present. Baths, vapour-baths, hot packing, change of air, emollients, such as simple ointments, glycerine, or vaseline, and massage, are recommended. The continuous current applied to the affected skin and sympathetic nerve is believed by some authorities to be useful. It is recognised that iodine and mercurials have no value. Warm clothing and general protection against cold are indispensable.

G. THIN.

SCLEROSIS (*σκληρός*, hard).—SYNON.: Fr. *Sclérose*; Ger. *Sclerosis*.—See FIBROSIS; and SPINAL CORD, Diseases of.

SCLEROSTOMA (*σκληρός*, hard; and *στόμα*, a mouth).—A synonym for *Ankylostoma duodenale*. See ENTOZOA.

SCLEROTIC, Diseases of.—See EYE, AND ITS APPENDAGES, Diseases of.

SCORBUTUS.—A synonym for scurvy. See SCURVY.

SCRIVENER'S PALSY.—A synonym for writer's cramp. See WRITER'S CRAMP.

SCROFULA (*scrofa*, a sow).—A term applied to a supposed hereditary tendency to slowly progressive chronic inflammations, and caseation, especially of lymphatic glands. The large majority of such cases are now known to be instances of chronic local tuberculosis, and the term is rapidly falling into disuse. See TUBERCULOSIS.

SCURF.—A popular name for the bran-like exfoliation which forms at the roots of the hair. It is composed of the normal desquamation of the epidermis of the scalp, with the addition of the epithelial exuviae thrown off by the hair-follicles. See PITYRIASIS.

SCURVY.—SYNON.: *Scorbutus*; Fr. *Scorbut*; Ger. *Scharbock*.

DEFINITION.—Scurvy is a disease characterised clinically by intense general debility; sponginess and swelling of the gums; ecchymoses, closely resembling bruises, about the thighs and legs; a brawny hardness about the flexures of the larger joints, and sometimes a contraction of the muscles of the calves; pearly conjunctivæ; and a sallow aspect somewhat akin to mild jaundice.

From a pathological point of view the disease is characterised by effusion of a fibrinous material in the tissues of the gums, between the striæ of the muscles of the thighs, legs, and sometimes (but comparatively seldom) of the arms, and also between the periosteum and the bones of the extremities, and occasionally of the ribs; ecchymoses sometimes found about the thoracic and abdominal aortæ and the alimentary canal; and a generally blanched condition of all the tissues.

ÆTIOLOGY.—At the present time the interest of this disease is largely historical, improved conditions both on sea and land having abolished the extensive and fatal outbreaks which were familiar to physicians of a former age; but experience has shown that when war, famine, or shipwreck reproduce the conditions which gave rise to it, it may be expected to reappear, with all its former virulence. It has also from the earliest times been a chief foe to sailors; and until the beginning of the seventeenth century it constituted a formidable item in the mortality-list of the navy in this as in other countries. Since, however, the prophylactic properties of a vegetable diet have been understood, scurvy has—except under very unusual and always preventable conditions—ceased to assume formidable proportions either ashore or afloat. Cases of the disease are still seen occasionally in hospitals at seaports, usually associated with some other disease, as dysentery, ague, &c.; but in some instances clearly caused by carelessness, either on the part of the

captain in serving out, or on the part of the crew in taking, the lime-juice provided. In recent years it has been recognised that young children brought up on preserved or artificially prepared foods are apt to suffer from scurvy, and this liability seems likely to increase, owing to the number of patented foods which are continually being placed on the market, and used by the well-to-do classes as substitutes for mother's milk.

PATHOLOGY AND ANATOMICAL CHARACTERS.—We are still in ignorance of the precise nature of the alterations of the blood and tissues which precede and accompany the development of the disease. It has, however, been shown that there is a considerable diminution of the red blood-corpuscles, an increase in the amount of fibrin and albumen, and no decrease in the amount of potassium-salts. Ralfe, from observations founded on the effect which the withdrawal, for a considerable period, of all fresh succulent vegetables and fruits had on the urine of healthy persons, and from analyses of urine from patients suffering from the disease, concluded that the 'primary alterations in scurvy depend on a general alteration between the various acids, inorganic as well as organic, and the bases found in the blood, by which (a) the neutral salts, such as the chlorides, are either increased relatively at the expense of the alkaline salts; or (b) these alkaline salts are absolutely decreased. This condition produces diminution of the normal alkalinity of the blood; and he suggests that this diminution produces the same results in scurvy-patients as happens in animals when attempts are made to reduce the alkalinity of the body (either by injecting acids into the blood or feeding with acid salts), namely, dissolution of the blood-corpuscles, ecchymoses and blood-stains on mucous surfaces, and fatty degeneration of the muscles of the heart, the muscles generally, and the secreting cells of the liver and kidney.' This and other chemical explanations are inconsistent with the important fact that the antiscorbutic properties of foods are destroyed by desiccation and overheating, processes which do not affect the proportions of the acids and bases they contain.

The most marked morbid changes of scurvy are the œdematous, spongy, and occasionally ulcerated gums; the bruise-like condition of the legs; and the brawny hardness, confined usually to the gastrocnemii and hamstring muscles. On cutting these across, tough fibrinous effusions are found packed between the muscular striæ, giving the cut surface a streaky appearance. If the anterior surface of the tibia be examined, the same kind of effusion will be often found between the periosteum and the bone. It would appear that the effusion is due, not to the degenerated condition of the vessels, but to a chemical alteration of the blood. True hæmorrhagic effusions are sometimes found beneath the periosteum, or between the epiphyses and diaphyses of the larger joints, especially when scurvy has proved fatal in children.

In severe cases the ribs will sometimes be found detached from the cartilages, and old fractures occasionally become disunited. Beyond a general anæmic condition, and occasional ecchymotic spots about the pleura and pericardium, the contents of the thorax present no special appearances. Lesions due to complications, such as dysentery, malaria and syphilis, will frequently be found. The body is not always badly nourished, and the cheeks are

usually puffy on account of the swollen gums; but local or general dropsy is seldom present.

SYMPTOMS.—The most striking features of scurvy are the spongy condition of the gums and the presence of brawny painful swellings in the hams. The complexion is of a sallow, dull, leaden hue, resembling that of a patient who has been for a long time subject to attacks of some form of remittent or intermittent fever, or that of a person recovering from jaundice; pearly-white conjunctive; puffy and sometimes bloated cheeks; spongy gums, bluish-red in colour, swollen sometimes to such an extent as to hide the teeth both in front and behind, and tending to bleed; teeth more or less loose, some already lost; tongue clean and pale; no special characteristic about trunk and upper limbs (though the latter are now and then slightly ecchymosed); shortness of breath, but no other chest-complication; no abdominal tenderness or anything abnormal as to the functions of the abdominal organs; thighs and legs usually presenting a more or less bruised appearance, particularly just above and below the knees; and brawny indurations of the hams and calves of the legs, often painful and tender. The effusions previously described may be so dense and abundant as to fix the legs in a semi-flexed position. Node-like swellings are also often observed over the tibia, owing to effusions between the periosteum and the bone. The swelling of the gums is specially noticeable round decayed teeth, and old injuries to the limbs seem to be the seat of the characteristic effusions. There are also usually a large number of spots and patches, very much like those of purpura, scattered indifferently about the lower limbs. There is sometimes considerable œdema about the ankles; but, in uncomplicated scurvy, pitting on pressure anywhere is the exception rather than the rule. The bowels are more or less constipated, the appetite is good, and there is no thirst. The breath has a peculiar offensive odour, and this may be aggravated by ulceration or sloughing of the gums, or necrosis of the jaw. General debility varies in degree, but may be excessive, with weak voice, and some tendency to fainting, if the patient is put or kept in a sitting position. He feels more or less general aching, and a sensation of contusion in the legs. The skin is dry and harsh, and desquamates over the legs. Cardiac and pulmonary sounds are normal. The urine is free from albumen, of normal specific gravity, with abundant chlorides. Urea and phosphates are said to be deficient.

In children the disease generally begins between the ages of six and eighteen months. In them the most prominent symptom is extreme tenderness in the legs, so that the patient lies perfectly still, and shrinks at the slightest touch. The gums are affected as in adults if the teeth have appeared or are pushing, and hemorrhages in various parts of the body are not uncommon.

COMPLICATIONS AND SEQUELÆ.—Simple scurvy is now rarely seen inland, except among the extreme poor who may be unable to purchase vegetables, and who live merely on tea and bread. In children, however, it appears to be more common among the middle classes, being due to the extensive use of patent or predigested foods, which are beyond the reach of the poorer classes. Nor is it very often seen afloat. It still, however, complicates to a considerable extent diseases or accidents that occur at sea, and so prolongs convalescence almost indefinitely. A sailor, for example, goes

out from England to Calcutta, and shortly after arrival in that port is attacked with dysentery or intermittent fever, fractures a limb, or becomes syphilitic. He remains in India a very short time, ships in a convalescent and enfeebled condition, lies up before the ship has been at sea many days, and probably does little or no work during the entire passage. The berth that he occupies constantly (and with very little change of clothing) is probably wet, his food scanty and unvaried, and his lime-juice or other antiscorbutic (as he cannot go to fetch it) served out irregularly, or perhaps refused when given. Under such circumstances, scurvy soon begins to 'colour' the original disease. The intestinal canal in cases of dysentery, the spleen in cases of ague, buboes, and chancres in syphilis, are all attacked, so to speak, scorbutically. Wounds, scratches, ulcers, or any other breaches of surface will not heal, and fractures sometimes become disunited; so that, as a consequence, the recovery of the patient after his arrival is deferred (solely on account of the existence of this scorbutic condition) for several weeks or months. In fact, all processes of repair, internally and externally, appear to be arrested, and no advance is made until the scorbutic symptoms have entirely disappeared. Nyctalopia is sometimes associated with scurvy, and it may be considered that night-blindness is induced by scorbutic conditions, inasmuch as this affection has decreased *pari passu* as scurvy has diminished in the British Mercantile Marine, and is now seldom complained of by sailors. In bad cases, hæmorrhage may take place from mucous surfaces. Nausea and vomiting may also occur.

The sequelæ of uncomplicated scurvy are, practically speaking, *nil*, for the patient, when properly treated, makes a rapid and complete recovery, leaving no trace of the disease behind. There appears, however, to be little doubt that one attack renders the patient less able to resist successfully future onsets of the disease, if placed under the same predisposing conditions. Several instances are recorded of old sailors, who have been the subjects of two or three attacks; but these have been generally complicated with some other disorder, delirium tremens being occasionally superadded.

DIAGNOSIS.—The diagnosis of scurvy cannot be difficult if the symptoms described above exist, and a dietetic history is carefully made out. This important item of information being established, the spongy gums, and the bruise-like condition of the lower limbs (this latter condition not being connected with any history of accident or injury), together with great general debility, should be sufficient to determine the nature of the disease. For even in mild cases the condition of the gums is quite unlike that produced by mercury. Moreover, the mercurial fœtor is absent, but an offensive earthy odour exists. Nor has the blue margin seen in the gums in cases of lead-poisoning any resemblance to the scorbutic condition. Scurvy might be occasionally confounded with purpura, as in some cases hæmorrhagic spots only exist about the legs, with no ecchymoses or hardness round the calf and hamstring muscles. But the condition of the mouth, the absence of severe cachexia, and, as Niemeyer remarks, the comparative absence of epistaxis, hæmatemesis, hæmatoma, and bloody evacuations from the bowels, will at once settle the diagnosis.

In children scurvy is not likely to be overlooked

if the possibility of its existence is borne in mind. Some doubt may arise whether the symptoms are not due solely to rickets, which is often present as a complication. It may also be confounded with rheumatism, paralysis, or joint-disease; or if hæmaturia is present with Bright's disease. The peculiar swollen and painful state of the legs, the spongy gums, and still more the effects of treatment, will serve to distinguish the disease.

PROGNOSIS.—Scurvy existing apart from other maladies is not a fatal disease. The patient may be seen in a state of excessive prostration, with feeble pulse, whispering voice, and a tendency to syncope; but a few days' rest, under favourable conditions, and proper treatment, produce a marvellous change, which results in a steady and very satisfactory convalescence. But before a prognosis is given, care should be taken to ascertain that the scurvy does not cover any other disease. Dysentery, syphilis, and the various forms of intermittent fever are undoubtedly its worst complications, and either of these maladies will, even under favourable circumstances, prolong convalescence considerably. The duration of the disease is limited only by the causes that produce it, for as long as the scorbutic diet and other predisposing conditions exist, so long will the disease retain the mastery, and progressively increase in severity.

TREATMENT.—If the patient, when first brought under notice, be so ill as to be unable to walk or stand, great care should be taken that the recumbent position is adopted and maintained. Many severe cases of scurvy have been lost by the neglect of this apparently simple precaution. The patient, in the absence of the nurse, sits up in bed, and has a sudden attack of syncope, from which he never recovers. Having regard to this, let the patient be undressed carefully, and washed (without a bath), any wounds or abrasions being covered with some simple antiseptic dressing. The direct treatment of the disease is almost purely dietetic, starting upon the principle that want of fresh vegetable diet has been the exciting cause of the illness. So the diet should consist largely of mashed potatoes; green vegetables (the *Cruciferae* being perhaps the best); oranges, pears, apples; and, as a convenient antiscorbutic, lime- or lemon-juice at the rate of from three to four ounces daily, mixed with about eight times its bulk of water, sweetened to taste, and used as a drink. Solid animal food should be given at least once a day, and in liberal quantity, as soon as it can be properly masticated. We begin in bad cases with milk, eggs, fish, and minced meat—in fact, any and all varieties of nutritious animal food, in conjunction with the vegetable diet; for the appetite is usually good, and the digestive powers almost unimpaired. If great prostration exist, alcohol, in small and frequent doses, must of course be given; but, as a general rule, very little is required. Mal-liquors are undoubtedly antiscorbutic, and a pint of ale or porter daily may be permitted if no dysenteric complication exist. Milk is also to a certain extent antiscorbutic, and should be given freely. As regards medicinal treatment, little or nothing need be done. All active treatment, general or local, is almost useless. The administration of mercury to scorbutic patients (through errors of diagnosis) did, in former years, an enormous amount of mischief, and, even in the presence of chest-complications, all counter-irritants to the skin must be avoided. Chlorate of potassium, in the form of a mouth-

wash, or given internally, probably assists to cleanse and purify the gums and mouth; and if old ulcers or open sores exist upon any part of the body, lint, wetted with weak lime-juice, is said to promote a healthy surface. But if there be any complication, no processes of elimination or repair will advance satisfactorily until the scorbutic symptoms disappear. If no grave disorder beyond the scurvy exist, recovery is very rapid, and few diseases are so eminently satisfactory to treat. The gum-swellings recede, and the ecchymoses on the legs begin to disappear after two or three days of treatment; and the brawny tenderness of the muscles of the lower limbs diminishes daily, the fibrinous effusions causing it being steadily and quickly absorbed. Dysentery is the most common complication of scurvy, and is usually tedious and troublesome. A fair trial should be given to the treatment above recommended, excluding malt-liquors, and substituting a small allowance of brandy, and as a rule the dysentery and scurvy will disappear together. In children fresh milk that has not been sophisticated in any way should as far as possible be employed, but as a rule is not sufficient by itself. One of the most useful remedies in these cases is potato, which should be well steamed and reduced to a light floury powder and beaten up with boiled milk until it is of the consistence of cream. Vegetable juices may also be given dissolved in broth.

The antiscorbutic treatment proper to combat the advent of this disease is sufficiently indicated in the above remarks, for it will be plainly seen that scurvy is due to the absence of certain necessary ingredients in diet. When these ingredients cannot be given in the usual form, the most convenient substitutes are lime-juice and lemon-juice, and the great mass of evidence, collected during the last fifty years, goes to prove that these substances contain, in natural combination, the best and most convenient prophylactic elements against scurvy. Their use in the Royal Navy has, since the close of the last century, been chiefly instrumental in driving the disease out of the Service; and legislative enactments passed in 1867, whereby a proper and genuine supply of juice was secured to all British sailors, have resulted in the decrease of scurvy in our own Mercantile Marine by more than 80 per cent.

CHARLES H. RALFE.

THOMAS R. BRADSHAW.

SCYBALA (σκύβαλον, dung).—Fæces in the form of hard rounded lumps, whether discharged or retained in the intestine. See *STOOLS*.

SEA-AIR; SEA-BATHS.—The physiological and therapeutical effects of sea-bathing cannot be separated from those of sea-air; for it is impossible to take sea-baths without being under the influence of sea-air; and the stay at the seaside alone, without sea-bathing, produces on many constitutions all the effects which are usually ascribed to sea-bathing. Residence at the seaside, that is, the influence of sea-air, is to be regarded as a special kind of climatic treatment, while the action of the sea-bath is analogous to the stimulating forms of the cold-water treatment.

As the sea-air and the sea-bath owe part of their properties to the constitution of sea-water, it will be well to begin with the latter, then to consider the characters and influences of the sea-air and the

sea-bath, and add some notes on seaside watering-places.

Sea-water.—*Temperature.*—The sea-water is of more equable temperature than the surrounding air. It is, as a rule, warmer than the atmosphere in winter, and cooler in summer; although on chilly days in summer, especially after a series of hot ones, the temperature of the sea-water is often higher than that of the air. The variations of the temperature of the sea-water from night to day, and from one day to another, are much less than those of the air. It would, however, be erroneous to assume, as is sometimes done, that the temperature of the sea-water near the shore is the same at different times of the day. The writer has often measured it at the Riviera, and the south coast of England, and has repeatedly found it on calm and sunny days at one P.M. and two P.M., from 5° to 7° F. higher than on the same days at seven or eight A.M. As to the different seasons, the sea-water reaches its highest temperature in summer much later than the air; and as it loses its heat less rapidly than the latter, it is mostly warmer in autumn and winter than the surrounding air, and gives off warmth to the latter. During the sea-bathing season, namely, from the end of May to the beginning of October, the temperature of the sea-water at the coasts of England, the north of France, Belgium, Holland, and Germany, varies in general from about 56° to 72° F., while in the Bay of Biscay and in the Mediterranean it is considerably higher.

Constituents.—Sea-water holds in solution a large amount of salts, varying somewhat in different localities, and slightly even in the same place at different times. The Mediterranean is richest, with about 2¾ to 3½ per cent.; while the water at the coasts of the British Channel and German Ocean varies from 2¼ to 3¼ per cent. The water of the Baltic, owing to the large number of streams which enter it, is much less salt, containing only about ½ per cent. Five-sixths of all the salts are chlorides of sodium and magnesium, while the remainder consist of the sulphates and carbonates of calcium, magnesium, and potassium.

Sea-air.—The sea-air, and the air at the sea-shore, are considerably influenced by the constant evaporation taking place from the sea, and also by the temperature of the sea-water. Owing to these circumstances, the sea-air contains in general more moisture, relative as well as absolute; and is more equable in temperature, the summer being less hot, and the winter less cold, at the seaside than at inland places in the same latitude; the day also may be regarded as less warm in summer, the night as less cold in winter. A very important fact is the comparative purity of the sea-air from organic admixture and inorganic dust, while the occasional presence of a greater or smaller amount of saline particles cannot be regarded as a disadvantage. The amount of ozone is greater; that of carbonic acid smaller. The variations of the barometer are greater but more regular in their occurrence, and this possibly exercises a beneficial influence on the functions of life. Nor is the fact to be overlooked that the air at the sea-shore is mostly in greater agitation than the inland air; and by this circumstance is probably to be explained the experience of Benecke that the same body of hot water loses its heat more rapidly at the sea-shore than at various elevations in Switzerland, varying from 3,000 to 6,000 feet above sea-level—an experience

from which we may infer that living bodies likewise give off more heat at the sea-shore than in elevated inland regions. The physiological effects of sea-air may be designated as 'powerful stimulation of the change of substance, both retrogressive and formative, expressed in a striking increase of urea, and decrease of uric acid and phosphoric acid in the urine, in the greatly increased requirements of food, and in the rapid and considerable increase of the weight of the body.' A certain power of responding to the increased stimulus of the sea-air is, however, required of the constitution; for the increased tissue-change necessitates an increase in the ingestion of food, and in the processes of excretion of the products of retrogressive tissue-change. If the digestive and assimilative organs be unable to satisfy the former demand, various digestive disturbances arise, the appetite fails, and emaciation is often the consequence. In many of these conditions greater benefit is derived from mountain health-resorts, where the demands made on the constitution are less great, and where less food is required. If the excretory functions be imperfect, as is the case in so-called 'bilious' individuals, and in some undefined gouty tendencies, headaches, giddiness, constipation, or other symptoms usually called 'biliousness,' make their appearance, and sometimes render the removal from the sea-shore necessary, though the use of aperient remedies, reduction in the amount of food and especially of stimulants, and active exercise at some distance from the sea, often suffice to correct this defective elimination and its consequences. In many cases of this kind, however, courses of mineral waters, especially the alkaline, saline, or common-salt springs, ought to precede the stay at the seaside. See MINERAL WATERS.

Sea-baths.—The sea-bath may be regarded as a powerfully stimulating cold-water bath, modified in its action by the saline ingredients; by the admixture of mechanical particles, organic as well as inorganic; by the varying degree of motion through the waves; and by the alternation in the exposure of a part of the body to the waves and to the air. We have already discussed the temperature of the sea-water, and the saline ingredients; but the temperature of the surrounding air, and the degree of motion in the air, also exercise a modifying influence on the effects of the sea-bath. The motion of the water varies constantly, according to the size and force of the waves, and the effect of the bath to a great degree depends on this point. When the waves are in any degree powerful, the upper part of the body is exposed to the coming, the lower to the receding wave, and the cutaneous nerves are not only influenced by the temperature, but also by the force of the water, and by the sand and other substances mixed with it. In a quiet sea these influences are considerably lessened. The alternation of exposure to the water and the air, likewise occasioned by the waves, is peculiar to the sea-bath, and is another source of constantly changing impressions on the cutaneous nerves.

BATHING SEASON.—The season for sea-bathing varies according to the climate of the locality. Thus it extends on the Mediterranean shores from May to October and even November; on the shores of the English Channel and German Ocean from June to September and the beginning of October. The time of the day for sea-bathing must depend on the individual, on the weather, and on the tide. Delicate

persons ought not to bathe with a perfectly empty stomach; but also never after a full meal. The duration of each bath is to be regulated according to the constitution of the bather, the force of the waves, and the temperature of the water. Weakly persons ought not to remain in the water over five minutes, but immersion for one or two minutes is in many such cases all that is useful or permissible; while stronger individuals may remain from five to ten minutes. The bather, we may say in general terms, should leave the water as soon as the reaction manifests itself.

In many cases, the warm sea-water bath may be recommended with advantage, when the cold sea-bath is forbidden. Indeed, courses of bathing in warm sea-water are not sufficiently used in a systematic way, though the medical practitioner possesses in them a gentle, manageable, and efficacious means of treatment during winter as well as during summer. They are in their action analogous to warm common-salt baths (*see* BATHS; and MINERAL WATERS). The tepid swimming-bath of sea-water we may regard as intermediate between the warm bath and the bath in the open sea, and likewise as very useful in appropriate cases. With due care it can be employed also in winter. It offers the advantage of the combination of one of the most perfect modes of muscular exercise, with the exposure of the skin to the influences of the sea-water bath.

The physiological effects of the sea-baths are similar to those of the sea-air. Abstraction of heat and stimulation of the cutaneous nerves lead to increased tissue-change, retrogressive as well as productive. Increased appetite and increased weight of body are usually observed in those who are benefited by sea-baths; while loss of appetite, headache, digestive disturbances, and loss of weight are often observed in those who are unable to bear the shock, or the increased demand on the body, or who remain too long in the bath, or take it too frequently.

CASES NOT SUITED FOR SEA-BATHING.—Persons affected with diseases of the heart, or of the blood-vessels and lungs, with angina pectoris, with epilepsy, with organic diseases of the nervous system, with fainting fits, with enlargement of the liver, or with other organic diseases of the abdominal viscera, ought to avoid bathing in the open sea, which may produce most injurious effects, such as violent palpitation and dyspnoea extending over many months, sleeplessness, total loss of appetite, and great emaciation. Old persons, and persons with feeble circulation, whether from age or otherwise, ought to avoid bathing in the open sea, excepting on warm days, and with a very quiet sea.

CASES TO BE BENEFITED BY SEA-BATHING AND SEA-AIR.—Sea-bathing is useful in many conditions connected with weakness or atony of the skin, such as tendency to profuse perspiration, or to taking cold at every change of temperature, or exposure to wind or draught.

In scrofulous complaints, long-continued residence at the seaside promises more than other climatic agents; but, as we have to deal with constitutional defects, and as our aim must be to alter the constitution, two, three, or even more years are often required. In many cases, judicious courses of sea-bathing, the use of warm sea-water baths, and sponging with sea-water, assist the climatic element of seaside residence. Education at schools situated

at the seaside offers, in scrofulous children, the greatest advantages.

In muscular rheumatism, the moderate use of the sea-bath combined with sea-air is useful. In more recent rheumatic joint-affections the sea-bath is mostly injurious, while the more gentle action of the sea-air, combined with the use of warm sea-water in local and general baths, is frequently beneficial. Persons affected with so-called nervous rheumatism—a term which is applied sometimes to hysterical cases, sometimes to spinal irritation, and also to rheumatism combined with nervous weakness—often derive benefit from the gentle use of the sea-bath, and still more from the sea-air.

In some functional diseases of the nervous system, the sea-bath forms an excellent remedy, if it be adapted to the individual case; for instance, in hysterical paralysis and other forms of hysteria, in the milder forms of diphtheritic paralysis, and in nervous dyspepsia. It must, however, be borne in mind that many persons, with a tendency to neuralgia, nervous asthma, hysterical convulsions, and other forms of hysteria, are unable to stand prolonged residence at the sea, especially at the Riviera. In such cases, mountain climates are generally more advantageous during summer and autumn. In many forms of anæmia, when it does not depend on organic disease of the heart and blood-vessels or other viscera, but on direct loss of blood or its constituents, on confinement, grief, and imperfect food, on slow and imperfect development, sea-air exercises a good effect. Hence the benefit obtained in many cases of amenorrhœa, chlorosis, and allied complaints, if the tendency to constipation is sufficiently combated. Often, however, the demands made by the sea-air on the constitution are too great, and the invalids lose weight; whereas they gain on mountains of moderate elevation.

In chronic pneumonia, in the remains of pleuritic effusion, and in phthisis, the sea-air, by its purity and its more equable temperature, is useful; but as wind is in most cases to be avoided, sheltered localities are essential. Sea-bathing is in this class of cases hazardous. The beneficial effects in whooping cough, when the first stage is over, are well-known. Regarding asthma, nothing can be said with certainty; some cases of nervous asthma are benefited at the seaside, while a large percentage are aggravated; on the whole, the writer's experience is more in favour of elevated regions than of the seaside. Whenever the effect is not yet known, the recommendation of seaside residence or mountain-air must be regarded as a trial; but in the majority of young persons affected with asthma, mountain climates are preferable. The advantage to be obtained in tendency to catarrh we have already mentioned.

In addition to the conditions named, there are many which cannot be designated by the name of any disease; but which are only states of weakness, manifesting themselves in various ways, as inability to sustain mental or bodily efforts, tendency to abortions, to leucorrhœa without any disease, &c. In such states of weakness the stimulating effect of the sea-air, combined with the grand aspect of the sea, are mostly found eminently useful.

SEASIDE WATERING-PLACES.—England is remarkably well provided with seaside places, and the different localities offer considerable variety with regard to climate. The east coast, which may be

designated as drier and more bracing, is especially to be recommended from the middle of June to the middle of October. The principal places on this coast are, beginning with the north, Berwick-on-Tweed, Tynemouth, Redcar, Saltburn-by-the-Sea, Whitby, Scarborough, Filey, Bridlington, Hunstanton, Cromer, Yarmouth, Lowestoft, Aldborough, Felixstowe, Dovercourt, Walton-on-the-Naze, Southend, Margate, Broadstairs, Ramsgate, Deal, and Dover. On the south-eastern and southern coast, which may be regarded as intermediate between the eastern and the south-western coast, we have Folkestone, Sandgate, Hastings with St. Leonards-on-Sea, Eastbourne, Seaford, Brighton, Worthing, Littlehampton, Bognor, the Isle of Wight, Bournemouth, and the Channel Islands. These places differ considerably with respect to the soil on which they lie; the position—close to the sea or on a cliff; the aspect; and the configuration of the locality itself, and the surrounding country. Even different parts of the same place offer different advantages. Thus the lower part of Folkestone, near the lower Sandgate road, is sheltered from the north by the cliff, while the houses on the cliff itself are more or less freely exposed to the winds from all quarters, and therefore preferable during the summer months. Hastings with St. Leonards is remarkably sheltered from the north, north-west, and to some degree from the north-east winds, and is through this, and through the influence of the sea, some degrees warmer during the late autumn and the early winter months—we may say till February—than closely adjacent but less sheltered places. In the Isle of Wight, the Undercliff, with Ventnor and Bonchurch, is sheltered by the hills from north and north-east winds, like Hastings, and more so; and has during winter a more equable and a higher temperature than other parts of the island. The Undercliff is therefore more adapted for climatic treatment during the colder part of the year; while Sandown, Shanklin, Cowes, Ryde, Alum Bay, and Freshwater are more suited for sea-bathing and climatic purposes during the warmer months. Bournemouth is sheltered as well by the configuration of the hills as by the pine-woods, which serve as a protection from violent winds. On the south-western coast, which may be regarded as somewhat moister and more sedative, Swanage, Weymouth, Sidmouth, Budleigh Salterton, Dawlish, Torquay, Teignmouth, Falmouth, Penzance, St. Ives, and Newquay are the principal sea-bathing places. On the North Devon coast, we may name Clovelly, Lynmouth, Ilfracombe, Westward Ho, and Minehead; on the Bristol Channel, Weston-super-Mare, Portishead and Clevedon; on the Welsh coast, Tenby, Aberystwith, Penmaenmawr, Llandudno, Rhyl; and in Lancashire, Westmoreland, and Cumberland, Grange, sheltered by configuration, Southport, Blackpool, Fleetwood, St. Bees, and Silloth. Important by its island climates is the Isle of Man.

Scotland likewise offers abundant localities for sea-bathing, the most frequented of which are Nairn on the east coast, Rothesay in Bute, Ardsrassan near the Firth of Clyde, and the Isle of Arran in the west.

Ireland is even richer, with Bray and Kingstown, near Dublin; Duncannon, Tramore, and Dunmore on the south coast; Rostrevor and Portrush on the north-east; Bundoran in the north-west; Kilkee in the south-west; and Queenstown, a sheltered and warm, but moist, climatic health-

resort in the south; where are also Youghal and Ballycotton.

On the north coast of France, Calais, Boulogne, Berk-sur-Mer, St. Valéry, Tréport, Dieppe, Etretat, Fécamp, Havre, Trouville, Deauville, Villers-sur-Mer, and Dinard are the most favourite resorts; on the south-west, Arcachon and Biarritz; and on the south Marseilles, Hyères, St. Raphael, Cannes, Antibes, and Nice.

The west and south-west coasts of Italy possess many good localities for sea-bathing for those requiring, or at all events bearing, heat, such as Bordighera, Alassio, San Remo, Castellamare, Sorrento, and the islands of Capri and Ischia on the west and south-west coasts, and besides Venice, with the Lido, Ravenna, Ancona, and others on the east coast.

On the coast of Belgium, Holland, and Germany the most important localities are Blankenbergh, Ostend, Scheveningen, Heligoland, Borkum, Nordener, Baltrum, Langeroog, Spikeroog, Wangeroog, Dangast, Cuxhaven, Wyk, and Westerland.

The shores of the Baltic possess many delightful localities, such as Heringsdorf, Putbus on the island of Ruegen, Warnemuende, Travemuende, Doberan; but the sea-baths and the climate are less stimulating than on the shores of the German Ocean.

The coasts of Norway, Sweden, and Denmark offer likewise good opportunities for sea-bathing combined with bracing sea-air from July to September.

HERMANN WEBER.

SEA-SICKNESS.—SYNON. : Fr. *Mal de Mer*; Ger. *Seekrankheit*.

DEFINITION.—A functional disturbance due to the motion of a ship, manifesting itself principally in epigastric discomfort, frontal headache, giddiness, vomiting, and vasomotor changes.

ÆTIOLOGY.—It is well known that many persons suffer from giddiness and even vomiting on swinging, dancing, and other rapid movements; and that in persons in whom vomiting is readily induced, objectionable odours, moving objects, slight derangements of the alimentary tract and mere recollection of previous attacks are any of them liable to cause this condition. It is no matter for surprise that all these incentives to vomiting should be regarded as causes of sea-sickness. That they have a predisposing and subsidiary influence in many cases is undoubted; nor can it be questioned that they may produce vomiting as easily on sea as on land. The smell of the engines will often cause passengers to vomit even though the sea be perfectly calm, and the recollection of previous attacks may give rise to nausea and even to vomiting before the ship has left the harbour. But these are only predisposing causes, and vomiting due to them is not true sea-sickness; in this condition movement alone is the exciting cause. Thus it may be induced during sleep, the patient waking to find himself in the act of vomiting. Much discussion has arisen concerning the conditions of movement under which sea-sickness is most likely to occur, and there seems to be a general consensus of opinion that the essential factor is suddenness of movement or change in the kind of motion, especially the commencement of the descending movement as the ship pitches or rolls—the movement being in all cases different in kind from that to which the patient is ordinarily exposed on land. It has been pointed out that the sudden descent of a lift or the sudden arrest of the

movement of a train may produce in the epigastrium precisely the same sensation as does the sudden downward pitch of a ship, while on the other hand the movement of a swimmer in the roughest sea never produces sea-sickness. Women suffer more severely as a rule than men. Probably no one is absolutely immune. Old people and young children are generally but slightly affected, and persons who vomit easily from causes such as those mentioned above are especially prone to true sea-sickness.

SYMPTOMS.—A slight sensation of chilliness accompanied by pallor is usually the earliest symptom. Almost simultaneously with this the sufferer is conscious of feeling ill at ease and of a desire to remain absolutely undisturbed. Unpleasant sensations as of a tight band over the forehead and of retraction at the epigastrium, especially marked on each descent, are soon experienced. The breathing becomes irregular, owing largely to an unconscious fixing of the muscles at the time of any sudden change in movement. Regular, deep breathing, if persisted in, may postpone the progress of the symptoms for a short time; but sooner or later the feeling of chilliness becomes rapidly worse, giddiness supervenes, and an attack of vomiting and retching soon follows, to be succeeded by a sensation of returning warmth accompanied by dilatation of the cutaneous vessels and slight uniform improvement. The former condition, however, soon recurs, the subsequent attacks of vomiting being progressively worse and the intervals between them shorter.

In many cases there is no headache beyond that already described; but in others, and especially in those liable to headache or in whom the conditions already quoted as predisposing causes readily produce vomiting, headache and giddiness may become most distressing symptoms and may be continuous throughout the whole of the illness. At first the vomit consists of any food which the stomach may contain; later on it becomes greenish yellow, and even bright yellow, with a bitter taste, and consists principally of bile and mucus. During the period of vomiting the heart's action is occasionally irregular. Some people in whom the general distress is very marked vomit but little; hence it is often said that of persons who are sea-sick those who vomit feel better than those who do not. There is frequently a marked tendency to drowsiness, but refreshing sleep is practically unknown during the acute stage. Prolonged retching is occasionally accompanied by hæmorrhage, usually from the pharynx or œsophagus, but sometimes under the conjunctiva and into other tissues as in whooping cough. Diarrhoea is sometimes present, but constipation is more usually met with. The flow of saliva is increased while the urinary secretion is lessened. Appetite is lost, even the sight or smell of food being loathsome. The secretion of milk is frequently arrested in nursing women; in others the menstrual flow is augmented. Sea-sick patients are always worse in the morning. In three to five days a favourable reaction generally takes place, the vomiting and nausea cease spontaneously, appetite returns, and the patient feels well.

In a few instances great exhaustion supervenes rapidly or gradually. The patient feels miserably helpless. He suffers from coldness of the extremities, thirst, headache, and spasmodic pain in the stomach, and complains of numbness of the surface of the body. A semi-comatose condition,

from which the patient is with difficulty roused, is sometimes met with in very severe cases, and requires assiduous treatment. In prolonged cases the reaction may assume a febrile character, with a rapid pulse, flushed face, and hot skin; convalescence is then slow.

An occasional but rare and dangerous form of sea-sickness is swooning without vomiting or any other symptom. The patient thus affected may lie motionless and almost death-like for a variable period. In long voyages attacks due to recurring bad weather generally tend to assume a milder character. There may be giddiness with or without nausea, but no vomiting; and in any case the duration of the attacks will probably be brief.

DURATION.—The ordinary duration of sea-sickness in long voyages is from three to five days, but it may last for weeks. After short voyages, such as crossing the English Channel, most persons recover as soon as they land; others after sleep or food. A few convalesce more slowly, and giddiness may persist for some days.

COMPLICATIONS AND SEQUELÆ.—Fainting and hysterical attacks are the most common complications of sea-sickness in women. Pregnant women occasionally abort. A weak and irritable condition of the stomach, or a state of general debility, may remain for a long time.

PROGNOSIS.—This is almost invariably favourable; yet death, although extremely rare, may occur from syncope or from exhaustion. Patients with heart-disease incur no exceptional risk.

PATHOLOGY.—The equilibrium-apparatus appears to be the essential seat of the disorder. Thus the derangement is common to all those animals in which this apparatus is similar to that of man; it is produced by movements involving considerable deviations from the normal conditions of equilibrium; and it returns on any marked increase of these movements, even when the sufferer has become more or less accustomed to them. The theories which attributed sea-sickness primarily to psychical or vaso-motor changes may be dismissed as inadequate. Rosenbach points out that a degree of tolerance of certain changes in equilibrium is established in each individual, that this varies in different cases, and that any marked excess of the movements to which each individual mechanism is adapted will give rise to the phenomena of sea-sickness. According to his view the semicircular canals play no important part, but 'in the epigastrium lies a mechanism which works as an apparatus regulating movement and balance, and giving information to the brain concerning the position of the body, the direction of the oscillation, and the acceleration of the individual parts.' Our knowledge of the mechanism which controls the maintenance of balance and of its relationship to other parts of the brain is insufficient to render any complete statement possible; but whether we regard the epigastrium as a source of information to the brain or as the seat of the first efferent signs of cerebral disturbance, it seems probable that a satisfactory explanation of the phenomena of sea-sickness must lie in the direction which Rosenbach has indicated.

TREATMENT.—In the treatment of sea-sickness it must be remembered that no single method will be successful in all cases; that in the case of a considerable number of persons treatment is at most palliative; and that the condition tends, after

a few days, to spontaneous cure. It will be convenient to discuss separately the measures advisable in (1) short and (2) long voyages respectively.

(1) *Short voyages.*—In voyages limited to a few hours the treatment is practically confined to prophylaxis, and consists in avoiding the predisposing causes, and in reducing the exciting cause to a minimum. To carry out the first of these principles, the digestive tract should be kept in thorough order for at least two or three days, and it is a good plan to avoid all indigestible articles of diet during that period, and to administer a mild aperient a day before 'crossing.' All fatigue at the time of starting should be avoided. Food should be taken about three hours before going on board. All possible means of imparting courage to the prospective sufferer should be employed, and it is probably because of the influence in this direction of small doses of stimulants that alcohol has obtained a considerable reputation. A flannel band should be worn round the abdomen, and every precaution taken to ensure warmth of all parts of the body. The position on the boat should be as far as possible central, exposed to the breeze but sheltered from hot sun and every source of offensive odour. The horizontal posture is the best, but a deck-chair is often as good if a more central position on the ship can thereby be obtained. Indeed, the question of position is largely determined by the character of the boat and of the weather, for the discomfort resulting from cold and wet and the sight of the sea is sometimes greater than that depending on the lack of fresh air and obnoxious odours usual in a cabin. An exciting book will often be of material assistance by absorbing the attention and preventing the reader from watching the movements of the boat on the horizon. Eating dry biscuits, chewing cloves, or sucking a lemon or any other sour fruit is often of material assistance, and an effort should be made to breathe regularly and deeply. When vomiting has once set in the best procedure as regards food varies in different individuals. In some cases it is found better to abstain until the completion of the voyage; in others, to persist in taking small quantities, even though they be almost immediately rejected. For extreme sufferers disposed to luxury, a chair balanced on the principle of a marine compass is said to be of considerable service.

The medicinal treatment of sea-sickness is very unsatisfactory. Several of those drugs presently to be enumerated will effect in occasional cases a complete cure; but in the majority of instances any given drug is likely to prove useless. Bromides, especially that of sodium given in half-drachm doses once or twice a day for two or three days before embarkation, are much advocated, but actual drowsiness may be produced without any amelioration of the malady. Digitalin (one-sixth of a grain three times a day) taken for two weeks has also its advocates. Chloralamide is occasionally of service, particularly in those suffering from weak cardiac action. Cocaine ($\frac{1}{4}$ gr. every two hours, three doses being taken before going on board) is probably the most reliable of all drugs. Phenacetin, caffeine, nitrites, chloral hydrate, and morphine are occasionally of service. In the administration of alcohol that form should be given which under ordinary circumstances agrees best with the patient. If vomiting of bile has occurred care should be taken

to empty the stomach just before landing, as the retention of bile in the stomach will retard convalescence.

(2) *Long voyages.*—On long voyages the same attention should be paid to the general health before starting, and in rough weather the same general precautions should be adopted. Of drugs, morphine is probably the most satisfactory, but should never be given in cases in which it is known to produce headache and other unpleasant after-effects. The diet should be carefully regulated and food taken frequently and in small quantities. It has been recently urged by Rosenbach and other authorities that the equilibrium-apparatus should be trained by passive movements similar to those of the vessel before going on board. There is reason to suppose that this plan may be effective, but in persons in whom vomiting is readily induced it at best does little more than spread over the few weeks immediately preceding embarkation the discomfort which will be concentrated into the first few days of the voyage; and most persons will prefer to run the risk of the sea-sickness rather than to undergo the repeated giddiness which such exercises will almost certainly involve. The rest of the treatment for syncope, exhaustion, and other complications is on general principles. In very rare cases it may be advisable to land the patient.

H. MONTAGUE MURRAY.

SEA-VOYAGES.—Sea-voyages have from remote antiquity formed a mode of treatment in chronic diseases, especially of the respiratory organs, and have more lately been much recommended in the treatment of consumption and scrofulous affections; but the different influences to which the invalid is exposed on long sea-voyages are but little appreciated in their details by the majority of the public, or by medical men.

The essential advantages which are generally ascribed to sea-voyages are the enjoyment of perfectly pure sea-air, abundance of light, and free exposure to the sea-breezes; absence, or at all events great limitation, of bodily exertion; and the probability of psychical repose. The uninitiated frequently regard these advantages as more or less fixed and, so to say, measurable qualities, and speak of sea-voyages in the same way as of sea-bathing, cold-water treatment, mineral-water cures, or mountain climates. The advantages of sea-voyages are, however, by no means fixed qualities, and they are often mixed up with unfavourable influences, such as bad weather, sea-sickness, improper food, &c. In everyday life it is an acknowledged fact, and not less so in all climatic cures, that the house in which the invalid lives exercises a most powerful influence on his chance of regaining and maintaining his health; and that the house alone often mars the effect of the best-adapted climatic change. In the same way the floating house, the ship, with its arrangements, forms one of the most important elements in the compound agent 'sea-voyage.' The arrangements of ships, however, are notoriously often very imperfect, and the narrow cabin never stands comparison with a good bedroom, the only counterbalance to this drawback often being that the invalid is forced to be the whole day long on deck, that is, in the open air, in order to escape from the confined state of the cabin. By this circumstance alone, however, the majority of the more serious cases ought to be excluded from

sea-voyages in ordinary ships, as they cannot be easily moved from the cabins to the deck, and *vice versa*. The hygienic conditions of the ship, the space allotted to each passenger, the ventilation of the rooms, the arrangement of the decks, must in every case be a matter of careful inquiry; but it would require too much space to enter into the details in this place. There are iron and wooden ships, steamers and sailing vessels. The iron ships have the advantage of being easily kept clean and free from smells, but they are apt to become very hot under the influence of the tropical sun. The sailing vessels can be kept more free from smoke and dust; but they are dependent on wind, and if they meet in the tropics with perfect calms (doldrums), the passengers may have to endure intolerable heat for several days and possibly weeks. The combination, therefore, of sailing power for ordinary conditions, with steam to be used only in case of need, would appear to possess the preference for ships to be used for therapeutic purposes (invalid ships).

A second point of paramount importance for every delicate person is the food, and in this respect again the ship-life on long voyages is less advantageous than the life in well-supplied health-resorts or at home.

Sea-sickness, or rather the degree of liability to sea-sickness, depends on peculiarities of constitution, which are only to be recognised by exposure to the influences of the open sea in different states of agitation. See SEA-SICKNESS.

In the article on SEA-AIR the prominent qualities of sea-climates have been given; but the most cursory consideration of the climatic conditions to be encountered in a long sea-voyage shows that there must be great differences between the physiological and therapeutical influences of sea-climates in latitude 50° and in latitudes 15° and 5°. The air in the tropical regions has a much higher temperature, and a larger amount of absolute moisture; the atmospheric movement is, as a rule, though by no means always, slighter; the barometric pressure is somewhat less in the tropics than in the temperate zones; and the daily and annual variations of atmospheric pressure are greater in the former than in the latter. There is also a difference between the same degrees of latitude on the north and south of the equator, the temperature in the southern hemisphere, for instance, being somewhat lower than in the northern; but these differences are comparatively small. The effects on the constitution of the climatic conditions of sea-life in different latitudes are very complicated. We will here only point to a few facts, namely, that in some delicate constitutions the functions of life are performed more easily under the influence of greater heat; that many delicate persons can eat and digest better, are able to take more exercise, sleep better, and that their mental functions are more active under the same circumstances; but that in the majority of average persons continued great heat produces lassitude, a tendency to diarrhoea and other digestive derangements, and imperfect sleep. Further, that in most individuals the bodily temperature rises above the natural heat (in general about $\frac{1}{2}^{\circ}$ F., and in some exceptional persons as much as 2° and 3° F.); and that pulmonary hæmorrhage occurs more frequently under high than under ordinary degrees of heat. Morbid states accompanied by pyrexia and by a tendency to pulmonary hæmor-

rhage ought therefore not to be exposed to tropical heat.

The climatic conditions to be met with in different voyages through the same regions vary at different seasons, but they vary still more in voyages through different seas, especially according to the longitude and latitude. Faber showed that the equability of sea-climates is by no means so complete as is generally assumed; and that great changes in temperature and atmospheric movements occur not rarely on successive days, and even on the same day.

THERAPEUTICAL USES.—The opinions of different writers on the therapeutical value of sea-voyages in the treatment of disease vary considerably. The majority of physicians entirely, or almost entirely, confine themselves to diseases of the respiratory organs in recommending sea-voyages; but their therapeutical field is no doubt much larger, and the result is probably more generally favourable, in some other complaints.

1. *Phthisis*.—The writer has had the opportunity of witnessing the effects of sea-voyages, of two to seven months' duration, in 72 cases of phthisis, mostly in the first or the beginning of the second stage. Of these 72 cases 38 benefited considerably, 18 remained about stationary, 16 became worse. Of the 16 bad results, 7 occurred in patients who went to Australia and back, with scarcely any rest on land; they seemed to have gained in the first part of the journey, but more than lost the gain in the latter part, apparently from dislike of food, from the monotony of the life, and from exhaustion. Out of 36 cases in the first stage 29 were more or less improved, 4 remained stationary, 3 lost ground. Of 27 cases in the second stage the result was favourable only in 8 cases, indifferent in 11, bad in 8 cases. Of 9 cases in the third stage the result was bad in 5 cases, indifferent in 3, favourable in 1. The writer has also notes of 11 cases of phthisis in the first and the beginning of the second stage, in which long summer voyages (namely, from three to five months) with whalers to the northern seas were tried, the result being favourable in 9 cases, indifferent in 1, and unfavourable in 1, apparently through inability to bear the want of variety in food. Among the 72 cases 12 were females, of whom only 3 were improved, while 5 remained stationary and 4 lost ground. Almost all the female patients expressed a strong opinion that long sea-voyages are not suitable for female patients.

2. *Laryngeal and Bronchial Catarrh and Asthma*.—In simple chronic catarrh of the larynx, sea-voyages, or cruising in yachts from this country to the Mediterranean, to the Azores and Madeira, had very good results in 20 cases out of 24. The effect of a similar plan was also satisfactory in 11 cases of chronic bronchial catarrh. In a tendency to bronchitis from pulmonary emphysema the benefit was likewise evident in 10 cases out of 12; but here the effect was, from the nature of the circumstances, less permanent. Of 8 cases of asthma, 2 cases of a bronchitic kind were benefited; 2 of a nervous character aggravated; and 4 were neither better nor worse. Eight cases of hay-asthma were, while on the high seas, quite free, but those who returned while the complaint was still in season, were immediately attacked.

3. *Scrofula*.—In 15 cases of scrofulous affections (caries of bones, affections of joints, glandular

swellings and ulcerations) one or several long sea-voyages were tried; in 11 of them the effect was quite satisfactory, in 4 less decided.

4. *Vesical Disease.*—In 3 cases of irritable bladder sea-voyages on yachts in warm climates have likewise proved useful.

5. *Cardiac Disease.*—Decidedly injurious was the effect of sea-voyages in 5 cases of dilatation of the heart, combined with chronic bronchitis. In 2 cases of enlargement of the liver, connected with weakness of the heart, the result was likewise unsatisfactory.

6. *Skin-disease.*—Chronic eczema was, in 5 cases out of 6, aggravated by sea-voyages.

7. *Nervous disorders.*—In 4 out of 6 cases of mental irritability, long sea-voyages, especially in yachts, had favourable results; in the fifth the mental condition was aggravated; in the sixth great improvement of the mental state was obtained, but this was accompanied by considerable exhaustion, from inability to take a sufficient amount of food. Of 4 cases of melancholia, 2 were apparently cured, the third remained uninfluenced, and one committed suicide by jumping overboard. In 4 cases of *tabes dorsalis*, in the earlier stage, cruising in comfortable yachts in the Mediterranean, with occasional landing, during the autumn, winter, and spring months, has been very beneficial; in 2 of these the disease has apparently been arrested, by persevering with this course during fifteen and sixteen years.

8. *Dipsomania.*—Finally, the writer has tried long sea-voyages in yachts in 5 cases of dipsomania, stimulants having been entirely excluded from the dietary. In one of these cases the result appears to be permanently good; in the 4 others it was good for the time with regard to the state of the body as well as of the mind, but there were relapses, which in 2 of the cases have led to several repetitions of the trial, each time apparently with more lasting, but as yet no permanent, result. Well-arranged sea-voyages deserve, therefore, at all events, a place in the management of this most terrible affection.

CONCLUSIONS.—From a comparison of these experiences with those of other observers, the writer is inclined to infer that, under favourable circumstances, sea-voyages of not too long duration may be rendered beneficial in the early stages of phthisis occurring in persons with a strong constitution, and without great tendency to sea-sickness. The voyage to Australia and New Zealand and back, after a stay of a few months in these climates (Hobart's Town, in Tasmania, for instance), specially recommends itself. The invalids referred to left in the second half of September, or in October or November, and returned in May or June. In this way the more unfavourable seasons of England are avoided, and instead of the short and sunless days, long and bright ones are obtained. To go to Australia and to return immediately has proved exhausting in several instances.

The voyage to the northern seas requires a peculiar mental disposition, and would, under the present conditions, be resorted to only under exceptional circumstances; but it has been very beneficial in the 9 cases of early phthisis mentioned—all of them possessing a satisfactory fund of strength, combined with love of sea-life and a good digestion.

The combination of yachting in the Mediterranean, and residence at one or several of the health-resorts

of those regions, or with a visit to Upper Egypt, has repeatedly proved successful in cases under the observation of the writer, not only in pulmonary invalids, but also in cases of mental irritability, exhaustion, chronic rheumatism, chronic glycosuria, and gout. This plan, however, is somewhat expensive. The patient ought to be, while in Egypt, under constant medical superintendence.

In hay-asthma, sea-voyages during the season of the complaint are to be recommended; but in other forms of asthma the result is uncertain, and the advice should not be given without consideration of all the circumstances. In most cases of younger persons alpine climates deserve to be preferred to sea-voyages.

In some forms of mental irritability, in overwork, in anxiety about business, and in the earlier stages of locomotor ataxy, sea-voyages, and especially yachting, in the subtropical regions, offer many advantages, particularly during the colder and damper seasons of our climate, as they allow of the combination of the enjoyment of sunlight and pure air with rest of body and mental repose. In slighter forms of mental irritability or overwork, shorter voyages are often sufficient, and even preferable; and the voyages to Madeira, to the West Indies, or to Brazil and the River Plate, may thus be recommended during the colder season.

Dipsomania and other morbid habits may be treated with great advantage by sea-voyages and yachting, provided that stimulants and the other injurious influences which the weak person is unable to resist can thus be entirely removed.

The time is coming when we shall have *therapeutic ships*, specially arranged for different classes of invalids. It would, for instance, not be wise to mix those suffering from dipsomania with sick persons to whom a moderate amount of stimulants is useful.

CIRCUMSTANCES COUNTER-INDICATING SEA-VOYAGES.—The circumstances which render it necessary to avoid sea-voyages are:—

1. Unconquerable sea-sickness.
2. Great temporary or permanent weakness and exhaustion.
3. Permanent delicacy of appetite, with inability to become accustomed to a certain monotony of food, or to a certain coarseness in the preparation of food.
4. Inability to bear the glare of the sea, such as occurs in tendency to glaucoma.
5. Persistent sleeplessness while at sea.
6. Dilatation and weakness of the heart, with or without valvular disease.
7. Enlargement of the liver, especially when caused by dilatation of the right ventricle.
8. Advanced stages of consumption, unless the affection be quite stationary.
9. Morbid conditions with a tendency to pyrexia.
10. A tendency to hæmorrhage.

It is the influence of great heat that ought to be avoided by the two classes of cases last mentioned. A voyage through tropical seas, especially in sailing ships, might prove dangerous in such subjects, from the possibility of being becalmed.

11. A tendency to epilepsy or maniacal fits. This ought specially to contra-indicate sea-voyages to tropical climates.

HERMANN WEBER.

SEAT-WORM.—A synonym for thread-worm. See ENTOZOA.

SEBACEOUS FOLLICLES, Diseases of.—

See ACNE; MILIUM; and COLLOID MILIUM.

SEBORRHOEA (*schum*, fat; and *ῥέω*, I flow).—

An ungrammatical synonym for steatorrhoea. See STEATORRHOEA.

SEDATIVES (*sedo*, to calm).—SYNON.: De-

pressants; Fr. *Sédatifs*; Ger. *Beruhigende Mittel*.

DEFINITION.—Agents which soothe pain and diminish functional activity. The term is often made to include other important therapeutical groups, as hypnotics, anæsthetics, and narcotics, i.e. all drugs which depress the sensory apparatus. See NARCOTICS.

1. *Local Sedatives*.—This class includes all agents which diminish the sensibility of the skin. Among these are cocaine, eucaine, aconite, carbolic acid, ice, ether-spray, atropine, menthol, morphine, veratrine, chloroform, and chloral hydrate. They act by paralysing or depressing the terminals of the cutaneous nerves, and are indicated in superficial neuralgias and in local subcutaneous inflammations, where they may be simply painted on in solution, or injected hypodermically or used by the old endermic method after the cuticle has been removed by blistering with ammonia. Profound cutaneous insensibility may be produced by the ether-spray or cocaine, which enables the surgeon to perform minor operations without pain. Chloride of ethyl is also very valuable as a local anæsthetic. See ANÆSTHESIA, LOCAL.

2. *General Sedatives*.—This class includes all agents which depress the functions of the cerebrum, and in the more accurate nomenclature of the new pharmacology the term 'general sedative' is fast disappearing, the group being now scattered among the hypnotics, such as sulphonal, trional, paraldehyde, chloral hydrate, bromides, &c., which are indicated in simple insomnia, and among the narcotics as opium, morphine, alcohol, Indian hemp, hyoscyamus, &c., which before depressing the sensorium cause some excitement. These agents are used to relieve pain and produce sleep. The drugs which produce total loss of consciousness with abolition of reflex action were formerly grouped under the term 'general sedatives'—these are now always defined as general anæsthetics, and include chloroform, ether, nitrous oxide, methylene, &c.

3. *Cardiac Sedatives* are drugs which diminish the force of the ventricular contractions, and at the same time reduce the pulse-rate. The principal agents employed for this purpose are tartar emetic, aconite and veratrum viride. They act upon the vagus-centre, the cardiac ganglia or the ventricular walls, or upon all these structures, and they are indicated in short sthenic inflammations, as in the fever accompanying tonsillitis, pneumonia, or orchitis, and in the rare conditions in which the heart's action becomes rapid and tumultuous without weakness of the ventricle.

4. *Pulmonary Sedatives* depress, and in large doses paralyse, the respiratory centre. By soothing this centre, any excessive or useless coughing can be prevented. Stramonium, belladonna, datura, and the numerous remedies for asthma act by diminishing the sensibility of the vagus-terminals in the lungs, though the respiratory centre may be stimulated by them. The chief pulmonary sedatives are

opium, morphine, chloral hydrate, codeine, hydrocyanic acid, chloroform, Virginian prune, &c. There are few better instances in the realm of therapeutics of the great danger of using a misleading classification. Pulmonary sedatives are frequently confused with sedative or depressant expectorants, but their actions are widely different. Given, for example, a case of bronchitis with dyspnoea from the difficulty of expelling a viscid secretion in the finer tubes, one full dose of a pulmonary sedative may cause death by depressing the respiratory and cough-centres, while a few doses of a sedative or depressant expectorant like ipecacuanha or antimony, by increasing and liquefying the tough and scanty secretion, may save life.

The pulmonary sedatives are indicated in all irritable conditions of the respiratory and cough-centres in which there is no great bronchial secretion, i.e. in the cough of acute pleuritis, in the early hacking cough of tuberculosis, and in all conditions where the patient is coughing a great deal more than is really necessary for expectoration. They can be combined often to great advantage with depressant expectorants.

5. *Gastric Sedatives* are a most important class of remedies; they act chiefly locally by soothing the inflamed or irritable gastric mucous membrane. The chief are morphine, cocaine, hydrocyanic acid in small doses, creosote, carbolic acid, all the preparations of bismuth, oxalate of cerium, chloroform, atropine, and ice. Gastric pain and vomiting are the chief indications for their use; they should be generally given before food and in frequently repeated doses. Sometimes minute doses of arsenic, and nitrate of silver, are employed as gastric sedatives. Small doses of morphine with prussic acid given in a plain effervescing mixture along with ice and in conjunction with a blister or smart sinapism to the epigastrium will generally control pain, nausea or vomiting of local origin.

6. *Spinal Sedatives* are agents which depress the functions of the cord chiefly by diminishing the activity of the anterior cornua. Though a long list of official agents produce this effect, few are employed in medical practice with this aim. The most reliable are bromides, physostigmine, chloral hydrate, chloroform, ether, camphor, nitrites, alcohol in large doses, salts of silver, copper, zinc and arsenic, gelsmium, antimony, ergot, apomorphine, &c. The chief indication for their use is spasm, whether this be tonic or clonic. The ideal condition calling for the exhibition of this group of therapeutical agents would be seen in a case of strychnine-poisoning, and the writer has found that in this formidable emergency no drug is so reliable and safe as very large doses of alcohol which antagonise the stimulating action of the strychnine upon the anterior cornua of the cord without dangerously depressing the heart at the same time.

7. *Vesical Sedatives* are drugs which on being eliminated by the urinary tract act as sedatives to the irritable mucous membrane of the bladder. They are indicated in cystitis from whatever cause. The most important are boric acid, hyoscyamus, belladonna, buchu, pareira, uva ursi, santal oil, copaiba, benzoic acid, &c.

WILLIAM WHITLA.

SEGMENTAL AREAS. — See PAIN IN VISCERAL DISEASE.

SELTERS, in Germany.—Muriated alkaline table-water. *See* MINERAL WATERS.

SEMIOLGY (σημείον, a symptom; and λόγος, a discourse).—A synonym for symptomatology, or the study of the signs and symptoms of disease.

SENILE INSANITY.—*See* p. 761.

SENILITY (*senex*, an old man).—**SYNON.**: Senile Marasmus; *Fr.* *Vieillesse*; *Ger.* *Greisenalter*.

DEFINITION.—That condition of body which usually supervenes naturally after the seventieth year, but sometimes occurs prematurely.

Senility is separated from the previous period of maturity by the climacteric stage, which in men occurs between the ages of fifty and sixty, and in women about ten years earlier. When of premature occurrence this state is commonly hereditary, or secondary to some exhausting illness, where failure of the trophic influence of the nervous system has been marked. All the signs and symptoms of senility have been seen in individuals under twenty years old.

We do not know why the body, after it has reached a state of maturity and vigour, should gradually decline; why, when once an even balance between tissue-waste and restitution is established, it is not maintained indefinitely. How far the failure is due to some inherent tendency, and how far to external influences, is wholly conjectural. Among the most recent hypotheses is one based upon the theory that the various glands, secreting and ductless—and also perhaps some of the tissues—exert some material influence upon the blood, and through it upon the tissues generally, whereby the normal metabolism of healthy activity is maintained; and, conversely, as this influence—effected, as is supposed, through some ‘internal secretion’—fails, so does the general nutrition deteriorate. According to Brown-Séquard, the weakness and characteristic phenomena of old age are due to ‘a natural series of organic changes and the gradually diminishing action of the spermatic glands and ovaries.’ To refer the senile state to an impairment of the trophic influence exerted by the nervous system, or to a diminution in the water contained in the protoplasmic elements of the tissues, or to any other such cause, however true each and all may be so far as they go, only places the difficulty a step farther back, and does not remove it.

ANATOMICAL CHARACTERS.—Observations, especially those of Humphry, have shown that many of the morbid conditions which are usually regarded as essential features of advanced life are really manifestations of disease occurring at this period, and do not of necessity pertain to senility, which may supervene and progress to death undisturbed by any actual disease—in short, an old age that is healthy, just as any other period of life may be. The term ‘involution’ has been suggested to express the normal senile changes in the tissues and organs, of which a progressive *atrophy*—a simple diminution of material—is the most universal and characteristic. The degree of wasting varies, but there is a general diminution in body-weight and height, except in persons whose climacteric has been marked by an increase of the adipose tissue, when the total loss of weight may be inconsiderable. Among the organs which exhibit simple

atrophy in the highest degree are the brain and spinal cord, the shrinking of the former with increase of the subarachnoid fluid being sometimes very considerable; the generative organs, especially the ovaries, and to a less extent the testes, the uterus, and the mammary glands; the mucous membrane and glands of the digestive tract; the bronchial and vesical mucous membranes; the spleen and lymphatic glands, the latter even to complete disappearance; and the kidneys. A most important change, and one that exerts a very direct influence on nutrition, is an extensive shrinking or even obliteration of the capillaries in almost all the tissues. The skin becomes much diminished in thickness, especially in the papillary layer, the constituent papillæ being very indistinct; and loss of hair and change of colour are well-known features. The senile changes in the skeleton are of peculiar interest. The bones, which in the earliest stages of formation are solid, become, as development proceeds, hollowed internally, by the formation of cancellous tissue, and, in the long bones, of the medullary canals. As the absorption of the central part proceeds the bones nevertheless increase in weight and bulk until maturity is reached by subperiosteal ossification; but in the decline of life, as this process takes place scarcely at all, and the atrophy continues, the bones, while retaining their general size and form, become much thinner, lighter, and weaker. The change takes place especially in the cancellous parts, which are most vascular, and where the agents for absorption are most active—hence the special weakness of the ends of the long bones and their liability to fracture. The alveolar parts of the upper and lower jaw-bones, consisting as they do chiefly of this loose spongy tissue, are characteristically wanting in the aged; and, since these parts carry the teeth, this atrophy of the bone leads to their falling out. The lower jaw is one of the few bones in which an alteration in shape takes place, the ramus and body coming to be more nearly in a straight line, with proportionate widening of the angle. The bone thus resembles the shape it has in the infant, with the difference that it then consists mainly of the alveolar part, while in old age it is the denser basal or sub-alveolar part that alone remains. The altered shape of the mandible has been attributed to the loss of teeth causing a deviation in the line of action of the masticating muscles. Humphry found no confirmation of the commonly accepted statement that the angle between the neck and shaft of the femur becomes smaller with age. The same observer has drawn attention to two exceptional conditions of an opposite character met with in the skull. One of these occurs as nearly symmetrical areas of atrophy of the outer table and diploë, forming well-marked depressions extending obliquely across the parietal bones, which he is inclined to attribute to ‘the pressure of the occipito-frontalis tendon stretched upon, and playing over, the most prominent part of the vertex.’ The other condition he has described as a considerable thickening of the calvaria, especially towards the frontal region, with great density of the bone. The explanation of this very anomalous state, so contrary to what takes place in the cranium and entire skeleton generally, is not obvious, though it may be connected with the diminished pressure of the wasting brain. The age at which these changes in the osseous system set in is very variable, but they

commonly commence earlier in women than in men. Slight atrophy takes place in the cartilages, costal and articular, the latter accounting for the diminution in height of the old person; but calcification is not invariable, and is indeed evidence of disease. Corresponding to the loss of strength in the bones is an increasing weakness in the power of the muscles, which explains, *inter alia*, the fact that fracture by muscular action is no more frequent in the aged than at other periods of life. It is to the weakening of the dorsal muscles that the stoop of age is attributable.

Associated with this atrophy it is usual to find *degenerations*—fatty, calcareous, or pigmentary—all of which are to be regarded as evidences of deficient nutrition, and may be contrasted with the differentiation and growth which characterise the commencement of life. The arcus senilis, the atheroma and calcareous degeneration of the vessels, the calcification of cartilages, the fatty degeneration of muscular and glandular epithelial tissue, the deposition of pigment in some spots, and the deficiency of the same in the hair and skin of the coloured races, are illustrations of true degenerative changes. Inasmuch, however, as these conditions are frequently wanting even in persons dying at an advanced age, they cannot be regarded as essential characteristics of senility; though it may be admitted that the cause, whatever it be, that determines the above described simple atrophic changes might easily be supposed to favour the closely allied states of degeneration. It might, indeed, be reasonably argued that it is the very absence of these and other degenerative changes which permits the attainment of great age, while their occurrence, as is usual, at a somewhat earlier period tends to a rate of mortality below that of these advanced ages.

Another expression of impaired nutrition, not so profound perhaps as degeneration, is a fibroid overgrowth in certain tissues, especially the arterial coats, which are often much thickened therefrom, and the prostate gland, which is commonly enlarged in elderly men from this same morbid change—the so-called hypertrophied prostate. The meninges, and the capsules of the liver and spleen, are sometimes thickened from the same cause; and a similar cirrhotic condition may be set up in the kidney and testis.

The blood contains fewer corpuscles and less solid constituents, is more watery, and is said to coagulate more readily. The total quantity is less.

The semen is very frequently wanting in spermatozoa, and contains in their place granular fatty cells, like colostrum-corpuscles, with a few red blood-corpuscles; but this is not invariably the case, for perfect spermatozoa are occasionally met with at an advanced age.

PHYSIOLOGICAL CHARACTERS.—The results of such structural imperfections appear in deterioration of the purely physical, as well as of the specially vital, properties of the tissues. There is an increased rigidity in some parts, as the tendons and blood-vessels; and a diminished cohesion in others, as the nails and bones, which are brittle and easily broken. Perhaps the most prominent and distinguishing mark of old age is a loss of elasticity; the skin, cartilages, blood-vessels, and lungs show this to a very marked extent, in the wrinkled integuments, dilated vessels, and distended air-cells. It has been noticed that this dilatation particularly affects the thin-walled veins, and more especially

those which do not run with arteries, are more superficial in position, and are concerned less with nutrition than with the proper return of blood—the ‘derivative circulation’ of Sucquet. The advantage of this is apparent, for such an arrangement must be a safety-valve in the case of the brain, to which organ there is a liability to determination of blood, and where the vessels are apt to rupture; hence the frequent turgescence of the nose and ears, and development of the veins of the dlopœ, in the aged. The muscular contractility and nervous irritability are diminished; and atrophy and degeneration of the gland-cells lead to failure in their powers of secretion.

The heart’s beat is weak, and frequently intermittent, from defect in rhythmical nervous stimulation; the cardiac sounds are feeble and often altered; and there is a general tendency to venous congestion. The mean rate of pulse after the age of sixty-five years is 75, gradually diminishing to 70.

The tissues, which differ in chemical composition from those of mature life, must in their metabolism form different products of waste; while the altered blood, circulating in a restricted area with diminished force, must offer to the organs a different pabulum from that which they have hitherto received, supplied as it is by impaired digestive organs. The enfeebled respiration prevents complete oxidation; and the excretory organs, being less capable, but imperfectly withdraw from the body the results of metamorphosis. The quantity of urine is often diminished to fifteen or twenty ounces *per diem* in old men enjoying good health. It contains a total amount of solids less than the normal standard, but the urine itself may be relatively of higher specific gravity, and deeper colour, from its diminished quantity.

As the nutritive functions fail, so do those of the neuro-muscular system. The sense-organs imperfectly receive impressions, which are but dimly communicated to the sensorium, whence feebly emanate the impulses needful to determine movements in muscles, the protoplasmic contractility of which is gradually diminishing. Meanwhile, the higher mental qualities, such as memory, judgment, and reason, dependent as they are upon the most perfect nutrition, gradually fail. The opposite conditions of wakefulness and drowsiness are frequently met with, and seem to be due to brain-wasting, as well as to some change in the cerebral circulation.

As a further manifestation of the lowered vitality, recent careful and continued observations have shown that the average body-temperature is slightly lower than it is in adult life, and the power of resisting cold is diminished.

The power of reproduction, lost by women at the climacteric, before the stage of senility sets in, is occasionally preserved by men to an advanced age.

Thus the old man presents a strong contrast in his vitality to that of the child; for while the life of the latter is so largely dependent upon, and so readily responsive to, external or peripheral impressions, the former lives more and more within himself; the distinctive animal functions gradually failing, as his existence becomes restricted to the performance of those of self-nutrition. The progressive impermeability of the capillaries, and the lessened vitality of the skin, alike tend to withdraw from the surface towards the central organs the manifestations of life.

Diseases of Old Age.—In a certain number of cases, the progressive deterioration in structure and failure in functional capacity mutually adapt themselves, and produce an old age which may be as healthy as maturity or childhood may have been. But in the course of the atrophic changes which normally mark this period, the body is liable to certain influences, both intrinsic and extrinsic, which lead to diseases characteristic of this stage of life, as there are those of infancy and puberty. Death from old age, when the organs have gradually and uniformly failed, is not unknown, but the fatal end is more commonly due to some disease, which has either lasted from an earlier period, or is especially the acquirement of this stage.

The maladies particularly characteristic of old age are marked by certain general features, which they owe to that condition of nutrition in which the tissues are at this period. Thus, as a rule, they present but little activity in their progress, or but slight severity in symptoms, though they are none the less likely to bring about a fatal result, from the ill-resisting power of the whole system. Diseases of an acute character are rare at this time, and such as do occur assume an adynamic form, and are very liable to run a most insidious and even latent course. When once established, an illness tends more perhaps towards maintaining an isolated attitude, without those sympathetic disturbances of many other organs which are so prominent in children. The power of reaction possessed by the aged is but very slight; owing to this, diseases readily lapse into a chronic state, or even present a chronic character from the outset, while comparatively trifling causes may lead to serious results. The observations of Humphry, however, went to show that the reparative power even in persons of advanced age is often greater than is supposed, and he adduces well-authenticated cases of complete recovery from severe accidents and illnesses; in such cases we may reasonably infer that the degenerative changes are wanting or but very slight.

There is probably no single disease which is met with in advanced age only; rather is it the case that many diseases which prevail at certain periods of life are wanting in old age. The atrophy of every tissue and organ entails a general failure in function; and should this failure predominate in any one system, as may readily follow from a marked degeneration of certain special parts, we have some exception to what may be taken as the normal standard of the senile state—in other words, a disease of it. The same difficulties surround the question, why one set of organs should be affected rather than another, as at other ages; but there nevertheless does exist a preference towards affections, of the nervous, circulatory, and respiratory systems. But a very large proportion of old people are entirely free from disease—of 824 persons over eighty years old, 55 per cent. of the women and 35 per cent. of the men were reported to be healthy.

Diseases of the brain.—The cerebral lesions may be a general senile wasting and softening, with complete enfeeblement of nerve-function; or of a more localised character, from rupture or occlusion of some vessel. The liability to venous engorgement is very prone to manifest itself in the brain, and cerebral congestion of varying extent is frequent. The cerebral vessels are especially liable to atheroma, and hence the great frequency of apoplexy in old age. The meninges are free from

morbid change beyond thickening, and offer none of the inflammatory conditions so common in early life. Muscular tremors especially involving the head are frequent, and are probably due to wasting of the cerebral cortex. All degrees of deafness and impaired sight are met with.

Diseases of the heart.—The degeneration of the cardiac substance may lead to a state of asthenia, gradually becoming fatal; dilatation of the orifices may be the more prominent lesion, with all the consequent symptoms of obstructed circulation; or they may be constricted, from atheroma or thickening of the cusps or rings. Cardiac dyspnoea and every form of irregularity in cardiac rhythm occur to variable extents.

Diseases of the lungs.—Pulmonary diseases are important, since they are commonly the immediate causes of death in the aged. A bronchial catarrh, or at least a considerable increase in the bronchial secretion, is a normal state in the very old; and this has been regarded as a compensation for the arrested skin-action. The transition from this to severe bronchitis is both easy and frequent, and is favoured by the liability to lung-congestion, and the enfeebled heart-power. Even more serious is a senile form of lobular pneumonia, which seems to be set up in the congested and oedematous areas, possibly as a further stage of the bronchitis. Such pneumonia is of very frequent occurrence, and often requires carefully looking for, since direct symptoms are wanting. When a generally ill-defined illness is present in an old person, this condition should be suspected.

Diseases of the digestive organs.—The digestive organs, supposing they have escaped the dangers of earlier periods, are not often the seat of disease at this time of life. The perversions of function they present, such as constipation and flatulence, may be conveniently comprised under the term 'atonic dyspepsia,' and are mainly to be attributed to deficient muscular power in the alimentary canal, and to a deficiency in the digestive fluids. Nevertheless, a good appetite and very fair digestion are far from being the exception in old people. An acute form of diarrhoea of a dysenteric character has been described as occurring at this period.

Diseases of the skin.—The skin, which suffers so much diminution in nutrition and thickness, often exhibits as a result marked changes in sensibility, even to the existence of an intolerable pruritus, no cause for which is visible. The scratching which is resorted to for relief sets up a dermatitis which intensifies the discomfort. The unhealthy integument offers a very favourable nidus for pediculi, and phthiriasis is accordingly a common senile affection.

Diseases of the urinary organs.—The most characteristic appearances in the senile kidney are those of a diffuse interstitial nephritis, with progressive atrophy of the tubes, similar in many respects to the 'gouty kidney,' but not, like it, associated with cardiac hypertrophy. The urine is in such cases albuminous, but the general symptoms of Bright's disease met with in middle life are usually wanting. Glycosuria, often intermittent, is of very frequent occurrence in old people; but is rarely attended by the constitutional disturbance of diabetes. Notwithstanding the very imperfect action of the skin and kidneys as excretory organs, diseases directly attributable to non-elimination of waste products are not characteristic of old age.

Irritability of the bladder, or even vesical catarrh, is very common in old men, being largely determined by the enlargement of the middle lobe of the prostate, but the hypertrophy of the organ may exist to a considerable extent without causing any symptoms until the mid-lobe is affected. Both retention and incontinence of urine follow, from the diminished tone of the viscus.

Vascular disease.—The frequency of atheroma of the vessels and thickenings from peri-arteritis has been alluded to. The tendency of the blood to coagulate, added to the opportunities it has for stagnating in the dilated channels and cavities of a weak heart, makes thrombosis and embolism very liable to occur in the senile state. The result of the obstruction is gangrene (*gangræna senilis*), which is readily established in tissues, the capillaries of which have wasted or are obliterated, with corresponding deficiency of nutrition.

Arthritic lesions.—Very few old people escape chronic arthritis in one or other of its many forms, though it may be questioned whether the constant pains complained of are not oftener due to neuritis than actual rheumatism. The fibrous tissues of the joints, fasciæ, and tendons are thickened, and less flexible from lack of moisture, with the result of producing that stiffness of the limbs so characteristic of the aged. The pain is rarely acute, but it is lasting, and aids in bringing about the general deficiency of motor power. True gout is scarcely a disease of old age, though often continued on from an early period of life.

Tuberculosis, Cancer, and Syphilis.—A senile form of tuberculosis occurring in people over sixty years of age, in whom the constitutional signs may have appeared in early life, with almost complete freedom during maturity, is met with. Marked evidences of this affection have been seen for the first time in persons over seventy-five years of age. The general features of this condition are similar to those in childhood, but slower in their course, more complete in the degeneration produced, and far more resistant to treatment. The cancerous and syphilitic cachexiæ very rarely manifest themselves for the first time in old age.

TREATMENT.—Old age itself, as a period of life, is clearly no more to be treated as a state of disease than life at any other stage. But regarded as a marasmus or cachexia, it has been sought to avert it as long as possible, or mitigate its effects; and for the attainment of longevity many means have been proposed, though as yet without any reliable result, the most diverse plans having been equally successful or futile. See PERSONAL HEALTH.

Based upon the hypothesis above mentioned, Brown-Séquard claims that much benefit to old men has followed the daily subcutaneous injection of a milligramme of a watery extract of the testis of an adult guinea-pig filtered through a Pasteur filter. Others have affirmed the same, but further confirmation is wanting.

It is unnecessary in the present article to explain the treatment of the diseases of old age, as appropriate remedies are set forth under the special headings. It is sufficient here to indicate that, whatever may be the malady, and whatever be the treatment pursued, it must not be forgotten that we are dealing with organs in which the structure is deteriorating, and the vitality of which is failing, and that all measures of a depleting or depressing

character must be avoided. The feeding of the aged, therefore, becomes a matter of the greatest importance. Speaking generally, the food should be of the most nutritious character, and given in small bulk and frequently. The old person, whether ill or well, should not be allowed to go for many hours without food, as is too often the case, through the night. A light meal in the small hours of the morning is of the greatest benefit, and goes far to counteract the wakefulness so common at this period of life. In view of the presumably deficient power of the digestive juices, some of the artificial semi-digested foods may be advantageously employed, or the active principles of the digestive secretions administered with the food. The need for alcoholic stimulants is usually imperative, especially for those who have always taken them, but they should only be given with other food. Medicinal tonics are seldom of the same benefit that they are at earlier ages; probably strychnine is of most value. A daily aperient, such as two grains or more of the extract of cascara sagrada, is frequently necessary; and it is generally undesirable to forbid any habit, whether of drugs, food, or smoking, which has been previously long continued. Among the most important hygienic indications is the maintenance of warmth. A fall in temperature both lowers the general vitality, and tends to establish a local disease, and its effects are more easily prevented than cured when once established. Warm clothing is therefore indispensable, and cold feet should be specially guarded against. Equally prejudicial with cold and damp is exposure to high winds, which is frequently responsible for a fatal pulmonary attack.

W. H. ALLCHIN.

SENSATION, Disorders of.—See OLFACTORY SENSE; VISION; OPTIC NERVE; HEARING; TASTE; TOUCH; and PAIN IN VISCERAL DISEASE.

SEPTICÆMIA (σηπτικός, putrid; and αἷμα, blood).—SYNON.: Fr. *Septicémie*; Ger. *Septicæmie*. See SAPRÆMIA.

SEQUELÆ (*sequor*, I follow).—Consequences or sequels. This word is applied to symptoms or morbid conditions which either remain or supervene after a disease has run its usual course; such as renal disease after scarlet fever, paralysis after diphtheria, or cardiac disease after acute rheumatism.

SEROUS FLUID.—See DROPSY, p. 417; and INFLAMMATION, p. 736.

SERPIGINOUS (*serpo*, I creep).—This term is used in connection with certain morbid conditions, such as ulcers or eruptions, when they spread in a creeping manner.

SERUM-REACTIONS.—When an animal, whether belonging to the human or other species, is suffering from, or has recently suffered from, a disease caused by certain of the bacteria which do not produce powerful extra-corporeal toxins, changes take place in the blood of that animal. These changes have been shown within recent years to be extremely complex, and the number of phenomena which result are considerable. To one of these phenomena, seen most especially in the blood, the name *Agglutination* has been given.

If two rabbits be taken, one normal, and the

other after inoculation on two or three occasions with a culture of the *B. typhosus*, upon mixing the sera of these rabbits with an emulsion of a young culture of the *B. typhosus*, and examining the mixture under the microscope, two very different pictures will be seen. The bacilli in the mixture made with the serum of the normal rabbit will be seen to be swimming about freely in the field, whereas those in the mixture made with the serum of the injected rabbit will be all herded together and motionless. This herding together is the Agglutination or Serum-Reaction.

HISTORY.—It was shown by Pfeiffer that when an animal was immunised by frequent injection of sub-lethal doses of an organism such as the *V. cholerae* or *B. typhosus*, the injection into the peritoneal cavity of a living culture of the organism against which the animal has been immunised was followed by disastrous results for the organism. Peritoneal fluid withdrawn at intervals, and examined under the microscope, shows that the bacteria after the lapse of a short time lose their shape, change into small round bodies, and finally disappear. The peritoneal fluid is now bactericidal. This bactericidal power is, however, specific (i.e. the fluid only kills the bacteria against which the animal has been immunised). After Pfeiffer had described this phenomenon, Gruber and Durham, and Bordet showed that the blood-serum of an animal so immunised has a marked effect upon the bacteria when mixed with it *in vitro*; in this case also the action is specific. Both these reactions were originally used for the purpose of determining the specificity of certain bacteria, but Gruber suggested that it was possible that it might also be used in the diagnosis of diseases produced by known bacteria. Grünbaum and Widal both began to work at this subject; both of them found that the surmise of Gruber was correct. Widal published his results first, and the reaction which is produced by the blood of an enteric patient upon typhoid bacilli is now known as 'Widal's reaction.' It has been since shown that the blood of patients suffering from other diseases will agglutinate the bacteria producing those diseases, and serum-reactions are now recognised as valuable aids in diagnosis.

CAUSE OF THE REACTION.—Little is as yet known as to the cause of the reaction, but it appears that after the injection of bacteria and the production of a certain degree of immunity, there are present in the blood of the injected animals certain bodies which are absent from the blood of normal animals. These bodies, which Gruber first called *Glabrificins*, are now known as *Agglutinins*.

According to Ehrlich's '*Seitenbentheorie*,' the immunisation of an animal is accompanied by the formation of a substance which he called 'immune body.' This body is able to fasten itself on to the bacteria which are injected subsequent to immunisation, and by so doing enable the Complement (which is present in the normal serum) to dissolve them and thus render them harmless to the animal. The agglutinins and this 'immune body' are not identical, because the 'immune body' may be formed in the absence of the agglutinins, and the agglutinins may be present and the 'immune body' absent: moreover, when both are present, there is no constant relation between them. It has been shown, however, that as a rule the two bodies are produced and increase together, and sometimes decrease proportionately. For instance, in pro-

portion as the serum of an animal increases in bactericidal power, so does the agglutinating power increase, while the spleen-juice of an enteric patient is both less bactericidal, and contains less agglutinins than the blood-serum. Although, as we shall see, the serum of many normal animals often possesses a slight agglutinating power, the agglutinins are a new product formed on account of the introduction of the bacteria, and have therefore a resemblance to the 'immune body.' The resemblance is still further marked when we remember that the agglutinins become anchored on to the bodies of the bacteria apparently in the same way as the 'immune body.' The reaction may therefore be considered to be one accompanying immunity rather than infection. What we should expect from this statement actually happens in fact; the reaction takes a certain time to develop in the serum after infection, and if there is no attempt on the part of the tissues to produce 'antikörpers' (i.e. immune bodies, agglutinins, &c.), the serum will never show the characteristic agglutination.

Outline of procedure.—As it is known that the serum of many animals, including man, possesses the power of agglutinating certain bacteria when it is undiluted or only slightly diluted, it is necessary to dilute the serum before mixing it with the culture of the bacteria.

In the case of man and the *B. typhosus*, for instance, it is found that about 30 per cent. of all persons possess a serum which will clump the bacteria when equal quantities of serum and a thin emulsion of the bacteria are mixed together. Many observers have found that normal human serum may clump when diluted to a strength of 1 in 10 or 1 in 20, and isolated cases are recorded where the serum in a strength of 1 in 40 has really clumped the bacteria. Great care must therefore be exercised when performing the test for diagnostic purposes to ensure that the blood is sufficiently diluted, in order that the error of the normal blood may be with certainty eliminated.

Collection and Dilution.—The serum may be conveniently collected from either the lobule of the ear or the finger. In either case the skin must be sterilised, the antiseptic removed and the part allowed to dry. The skin is then pricked, and the issuing blood collected in a small pipette. After the blood has coagulated, the serum is removed from the pipette and accurately diluted with sterile water, normal saline solution or broth, to the required degree, and mixed with a suitable quantity of bacterial emulsion. If a mixture containing 1 per cent. of the serum is required, it is customary to make a 2-per-cent. solution of the serum in broth or water, and then mix equal quantities of this and the bacterial emulsion in order to obtain the 1-per-cent. dilution. The undiluted serum should not be diluted with the bacterial emulsion, or the phenomenon of pseudo-clumping may be seen.

Demonstration of the reaction.—The agglutination may be observed either microscopically or macroscopically. In order to examine it microscopically, a loopful of the diluted serum is placed upon a clean cover-slip; after sterilisation, the same loop is filled with bacterial emulsion and a thorough mixture of the two fluids effected upon the cover-slip. This is then inverted upon a hanging-drop slide and examined with the microscope. After the lapse of a short interval—if the serum is going to agglutinate—small groups of bacilli will be found,

and these will grow larger, as time goes on, by the addition of more bacteria, until finally practically every bacillus in the original emulsion is in one of the now large clumps.

The macroscopic agglutination is seen if a small quantity—50 to 100 cubic millimetres—of the diluted serum and of the bacterial emulsion are mixed together in a watch-glass or small test-tube, and sucked into a fine, but not capillary tube, and one end of the tube sealed off. After allowing the tube to stand in a vertical position with the sealed end downwards for a few hours, the agglutination is evidenced by the fact that at the bottom there is a fine white flocculent precipitate, and the supernatant fluid is quite clear.

The reaction in Enteric Fever.—It must always be remembered that the agglutination-test is a quantitative and not merely a qualitative one. As has been previously stated, the serum of 30 per cent. of normal persons will show agglutination when in the strength of 50 per cent., and a considerable number are found to exhibit it in a dilution of 1 in 10; a few may show it in a dilution of 1 in 20, and an occasional person even in a dilution of 1 in 40. The great majority of enteric patients will, during some period of the disease, possess a serum which will agglutinate in a strength of 1 in 50, and not infrequently in much greater dilution (e.g. 1 in 200, 400, or even 800). It is necessary therefore to fix a *standard dilution*. Most clinical bacteriologists use the serum in a strength of about 1 in 20, and in practice this dilution gives an error of about 3 per cent. to 5 per cent.: the error shows itself in both ways; that is, enteric-fever patients may fail to show the reaction, and patients not suffering from enteric fever may give the reaction. A dilution of 1 in 50 will eliminate all the positive reactions from non-enteric patients and will probably eliminate none of the enteric patients who yield a serum which agglutinates in a dilution of 1 in 20.

As the actual agglutination occupies a certain time, and it is certain that the serum of some normal persons will show the phenomenon even when it is diluted, if left for many hours, it is also necessary to have a *standard reaction-time*. If a dilution of 1 in 20 be used, the reaction-time should be half an hour; if 1 in 50, an hour. If the mixture be examined under the microscope at short intervals, it will be found that a considerable proportion of reactions will take place in a few minutes, and the patients yielding these sera are, practically without exception, suffering from enteric fever, even if the dilution is only 1 in 20.

The day of the disease upon which the phenomenon first appears is very variable. Moreover, when the first sign of agglutination appears, it is so feeble that one is not able to use a sufficiently great dilution to differentiate it from normal serum. A reaction in a dilution of 1 in 20 may appear upon the third or fourth day, but this is rare; the usual time is about the eighth, ninth or tenth day; but it may not appear until the eighteenth, nineteenth or twentieth day, and very occasionally it never appears. In a dilution of 1 in 200, the reaction may rarely appear as early as the sixth or seventh day, but in quite a considerable proportion of the cases the serum will not react in this dilution during any period of the disease. There is, moreover, a distinct variation in the strength of the reaction in the same patient from time to time; on one day the serum may agglutinate in a strength of 1 in 400,

and three or four days later the highest dilution which will agglutinate is 1 in 100.

The question now arises 'What is the value of the test in the diagnosis of enteric fever?' The real answer to the question will depend upon whether a positive or negative reaction has been obtained. If a positive reaction has been obtained in a dilution of 1 in 200 within half an hour, the patient, without doubt, is suffering or has recently suffered from enteric fever. If the dilution is 1 in 20 the error may amount to about 1 per cent.

If a negative reaction has been obtained, the value of the observation will depend upon the day of the disease; one negative result early in the disease is of no value. If three or four examinations have been made at regular intervals, the last being upon the twenty-fourth or twenty-fifth day of the disease, and all with negative results, the error will amount to 1 or 2 per cent.; it is undoubtedly true that some patients suffer from a severe attack of enteric fever and die at the end of a fortnight or three weeks, who never give the reaction because the agglutinins are never formed. Although in the case of artificially immunised animals the serum of typhoid-immune animals may agglutinate the *B. enteritidis* of Gärtner, and Gärtner-serum agglutinates typhoid-bacilli in the human subject, this fallacy is practically negligible. The reaction may persist for a considerable time, even for years; care must therefore be taken to ascertain whether the patient has recently suffered from enteric fever. With these reservations the answer to the question may be summed up as follows. In from 97 per cent. to 98 per cent. of the cases, the presence or absence of agglutination will correctly diagnose whether the patient is suffering from, or has recently suffered from, enteric fever.

The reaction in Malta Fever.—What has been said of enteric fever and the *B. typhosus* applies to Malta fever and the *M. melitensis*. The disease is, like enteric fever, a chronic disease. In the case of Malta-fever patients, the serum is found to give a stronger reaction than generally obtains in enteric-fever patients, sometimes a dilution of 1 in 500 or 1 in 1,000 may agglutinate the micrococci. In performing the test the emulsion should be filtered through filter-paper in order to remove the clumps of cocci which are generally found in cultures of this bacterium.

The reaction in Glanders.—In man there are two distinct types of glanders, the acute and chronic. In the acute form the patient generally dies before the agglutinins are formed. In the chronic form, however, it is possible to use the test with advantage. The serum should be diluted to at least 1 in 100.

The reaction in Cholera.—The serum of patients who are recovering or convalescent from this disease usually shows agglutinative power, and the test may be applied to determine whether the patient has had an attack of genuine cholera; but for the diagnosis of the disease during an acute attack, the test is of course without value, since the patient will generally die or be on the road to recovery before the agglutinins are formed.

The reaction in Plague.—The remarks concerning cholera apply with equal force to this disease. In making the examination, the pseudo-clumps found in the cultures must be removed.

The reaction in Dysentery.—Quite recently a bacillus has been described in connection with

dysentery which is agglutinated by the serum of patients suffering from this disease; the reaction will therefore be, most probably, of considerable value as a diagnostic agent for dysentery.

WALTER C. C. PAKES.

SERUM-THERAPEUTICS.—There are two kinds of protective serums used in the treatment of disease, which possess different properties. The one kind is an *antitoxic* serum which neutralises bacterial and allied toxins, and the other is an *anti-bacterial* serum which hinders the multiplication of the bacteria in the tissues. Both kinds of serums are commonly, but incorrectly, designated antitoxins. The principal *antitoxic* serums are the diphtheria-antitoxin, the tetanus-antitoxin and antivenin; while the principal *anti-bacterial* serums are the anti-streptococcic, the anti-pneumococcic, the anti-plague, the anti-cholera, and the anti-typhoid serums.

These serums are specific in the sense that each one is only of use in treating the disease for which it is designed; the diphtheria-antitoxin, for example, is of benefit in the treatment of diphtheria, but has no effect upon a case of tetanus. Protective serums can be used either as prophylactic or as therapeutic agents. They are chiefly used in the latter capacity, as the protection they afford is of short duration, not lasting longer than two or three weeks.

METHOD OF ADMINISTRATION.—The serums are supplied either in the liquid or dried form. They cannot be administered through the alimentary canal, as they are destroyed during the process of absorption. They are accordingly administered by injection into the subcutaneous tissue. The site usually chosen for injection is the subcutaneous tissue of the flank. As a large quantity—about half an ounce—of serum is injected, special antiseptic precautions are necessary. The hands of the operator should be carefully cleansed with soap and water and afterwards with an antiseptic solution, such as carbolic acid (1 in 20) or lysol (2 per cent.). The skin of the patient at the site of injection should be treated in the same way. The syringe and needle should be boiled immediately before use and laid to cool upon a towel wrung out in an antiseptic solution. The cork and neck of the bottle containing the serum should be washed with an antiseptic before opening. The syringe is then carefully filled and the serum slowly injected into the subcutaneous tissue. A piece of antiseptic gauze washed in collodion is laid over the puncture. For each injection a fresh bottle of serum should be used. Many syringes have been designed for the injection of serum. The essential feature is that the piston and connections should be so constructed as not to be injured by boiling. For children it is advisable to connect the needle with the syringe by means of a short indiarubber tube, so as to avoid injury in the case of struggling.

If dried serum is used it must be dissolved in water which has been boiled and allowed to cool; and the solution should be made in a vessel which has been thoroughly cleansed with an antiseptic and subsequently rinsed with boiling water.

ILL-EFFECTS WHICH MAY FOLLOW ADMINISTRATION.—All therapeutic measures are attended by some risk, and the injection of serum comes under this category.

If the serum is contaminated, or if proper pre-

cautions are not taken during the process of injection, septic trouble may arise. Abscess at the site of injection occurred in 1·2 per cent. of the injections at the Metropolitan Asylums Board Hospitals, and one or two cases of death from septicæmia have been recorded.

Apart from these avoidable effects, symptoms sometimes of a serious nature may arise due to the introduction of an animal's serum into the human body, and analogous to the symptoms caused by the injection of the blood-serum of one animal into the tissues of another. In a few instances the administration of serum has led to immediate collapse which has even proved fatal. In several cases severe toxic symptoms, consisting of rigors, pyrexia, vomiting, convulsions, and rapid appearance of a rash have been observed. These serious symptoms are very rare, and only a few cases have been recorded among hundreds of thousands of injections.

Other symptoms not of a serious character more frequently arise. Rashes and articular pains, accompanied by pyrexia, constitute these symptoms. *Rashes* are observed in from 25 to 50 per cent. of the cases. They generally occur about the eighth day after the injection, but they may occur immediately after injection, or as late as the thirty-first day. The rash usually begins on the wrists and ankles, but it may start at the site of injection. It may be partial or may involve the whole of the body. In character the eruption is a blotchy erythema, looking like the rash of measles, or more rarely it is a diffuse punctate erythema similar to that of scarlet fever; it is often complicated with urticaria. With the rash there is malaise and pyrexia; the temperature may reach 104°, but is more frequently between 100° and 101°.

Articular pains are less common than rashes, occurring in about 5 per cent. of the cases. The hips, wrists, and ankles are the parts most frequently affected. There is seldom effusion into the joints, the pain and swelling being situated in the periarticular structures. The affection occurs at about the same time as the rashes, and is frequently accompanied by them and by pyrexia. In a few days the pain and fever subside.

Transient pyrexia, unaccompanied by rash or articular pain, sometimes occurs.

LIMITATIONS OF SERUM-THERAPY.—It has been shown experimentally that a much smaller quantity of serum is required to protect an animal when injected soon after infection than at a later period; and it has been found that after a certain time has elapsed no amount of serum however large is efficacious. Clinical experience bears out these observations: in the later stages of a disease the administration of serum is of very little value; the earlier the treatment is adopted the better the chance of recovery.

I. ANTITOXIC SERUMS. — Diphtheria-Antitoxin.—The value of serum-therapy has been more clearly established in the case of diphtheria than in any other disease. The reasons for this success are as follows. Diphtheria is a local disease in which the symptoms are caused by the slow absorption of toxins elaborated at the site of infection, and the anti-diphtheria serum contains an antitoxin, which, as can be shown experimentally, neutralises these toxins. The disease can be recognised by its local manifestations at an early stage before sufficient toxins have been absorbed to seriously damage the tissues. Detailed instructions concerning the

use and the efficacy of this serum in the treatment of diphtheria are given in the article on DIPHTHERIA, p. 407.

As a prophylactic, where there has been much exposure to infection, it may be considered advisable to administer antitoxin. For this purpose a dose of 200 units appears to protect for three weeks. The writer prefers to make daily bacteriological examinations of the throats of exposed individuals, and to defer using antitoxin until bacilli have been found, when a dose of 5000 units should be given.

Diphtheria-antitoxin does not keep indefinitely, but if placed in a cool dark place it will for practical purposes retain its potency for six months.

Tetanus-Antitoxin.—The toxin produced by the *Bacillus tetani* is a powerful nerve-poison. As can be shown experimentally, the anti-tetanus serum contains an antitoxin which neutralises the toxin. The tetanus-antitoxin is much more powerful than the diphtheria-antitoxin, but it is not so efficacious in human therapeutics. This is due to the fact that the disease cannot be diagnosed until the central nervous system has been severely affected by the toxins. It would appear that the antitoxin which has entered the circulation after subcutaneous injection is not capable of gaining access to the interior of the central nervous system. In accordance with this it has been shown that the intracerebral injection of antitoxin is more efficacious than subcutaneous injection in the treatment of guinea-pigs artificially infected with tetanus. The same method combined with subcutaneous injection has been employed for the treatment of tetanus in man. The dose for subcutaneous administration is 5 grams of dried serum or 10 to 20 c.c. of liquid serum, which should be given at first every 6 or 12 hours, and afterwards at longer intervals, according to the progress of the case.

Intracerebral Injection.—For intracerebral injection the dose is 2 or 3 c.c., the most potent serum being used. The method of administration is as follows: The patient is put under an anæsthetic, and the scalp over the front of the head is shaved and carefully sterilised. An imaginary line is drawn from one auditory meatus to the other, and another line from the base of the nose to the occipital protuberance. From the point where these two lines intersect a line is drawn to the outer angle of the orbit. The centre of this line is situated just in front of the motor convulsions and is the site chosen for injection. A hole is drilled in the skull at this site with a small Archimedean drill of a diameter a little larger than the needle of the syringe. The needle, which is about two inches long with a rounded point, is inserted into the brain as deep as it will go, and the serum is injected very slowly, about ten minutes being taken to inject the two or three cubic centimetres. The same operation is performed on the opposite side. During the operation careful antiseptic precautions must be taken, otherwise there is the risk of the supervention of cerebral abscess.

Efficacy of Treatment.—It is difficult to estimate the value of the treatment of tetanus with antitoxin. In some cases both after subcutaneous and after intracerebral administration the recovery of the patient seems to have been definitely due to the treatment. Subacute cases are more benefited than acute cases. It would appear advisable to combine the intracerebral injection with subcutaneous injection.

The liquid serum will keep in a cool and dark place without appreciable alteration in potency for several months, and the dried serum longer.

Anti-venomous Serum.—The principal constituent of the venom of poisonous snakes is a toxin which is a powerful nerve-poison, and which is identical in all species. In some species, in addition to the nerve-poison, there are other toxic substances which break up the blood-corpuscles and cause local inflammation.

Anti-venomous serum contains an antitoxin, analogous to the diphtheria- and tetanus-antitoxins, which neutralises the nerve-poison contained in the venom. This has been shown experimentally, and it has also been proved that the serum protects an animal when injected subsequent to the introduction of venom into the tissues. A number of cases of recovery, after treatment with serum, of patients bitten by poisonous snakes have been recorded. The dose is 10 to 30 c.c., which may be given subcutaneously, or better by direct injection into a vein. It should be administered as soon as possible after the bite, and the part affected should be treated in the usual way by proximal ligature and the injection of a solution of calcium hypochloride.

II. ANTI-BACTERIAL SERUMS.—**Anti-streptococcic Serum.**—There are many affections produced by the *Streptococcus pyogenes*, such as erysipelas, puerperal fever, and various septic conditions, in all of which the invasion of the tissues by the cocci is the essential factor of the disease. The anti-streptococcic serum does not contain an antitoxin, but it contains anti-bacterial substances which hinder the multiplication of the cocci in the tissues.

The potency of the serum hitherto prepared is not great when tested upon animals, and it has also been found that a serum which will protect against a streptococcus obtained from one source will not always protect against a streptococcus from a different source. The discordant results obtained in the treatment of cases in the human subject may thus be accounted for, especially when one remembers that the different serums in the market vary considerably in potency. Many cases treated successfully have been recorded, and there can be no question of its value in some cases; but we are still unable to say what cases will be benefited by the treatment. The treatment appears to have been most successful in cases of puerperal septicæmia. Sometimes when one brand of serum has been of no value another brand has proved efficacious.

The dose of anti-streptococcic serum is 10 to 30 c.c., which should be given two or three times a day in acute cases, and once a day in chronic cases. It is better to inject the serum near the site of infection if this is practicable. The serum appears to lose its potency if kept for more than a few months.

Anti-pneumococcic Serum.—Acute lobar pneumonia is by no means the only disease caused by the pneumococcus—pleurisy, empyema, meningitis, endocarditis and other affections may be caused by the same micro-organism. The anti-pneumococcic serum resembles the anti-streptococcic in containing anti-bacterial substances, but no antitoxin; and it acts in the same way, namely, by hindering multiplication of the cocci.

The potency of the serum supplied by Pane is such that 1 c.c. will protect a rabbit against 1,000 to 3,000 fatal doses of living cocci when injected simultaneously, but into different parts of the body.

Like the anti-streptococcic serum, a serum which will protect against one variety of pneumococcus will not always protect against another variety.

The value of the treatment of pneumonia with serum is still *sub judice*. We will only say that in some cases the treatment certainly appears to have been successful. The dose is from 10 to 20 c.c., which may be given twice a day until the fever has subsided. The treatment is applicable for any condition caused by the pneumococcus. The serum does not maintain its potency for more than four months.

Anti-typhoid, Anti-cholera, and Anti-plague Serums.—Serums have also been prepared against cholera, typhoid fever, and plague. They all contain anti-bacterial substances and no antitoxin, with perhaps the exception of the plague-serum, which contains a small quantity of antitoxin. We possess very little knowledge of the efficacy of treatment with these serums. They must not be confounded with the vaccines used as prophylactic against typhoid fever, plague, and cholera, and which have been improperly called serums.

J. W. WASHBOURN.

SETON, Use of.—A disused method of counter-irritation, consisting in the insertion of a tape or cord beneath the skin.

SEVENTH NERVE, Diseases of.—*See* FACIAL ATROPHY; FACIAL PARALYSIS; FACIAL SPASM; HEARING, Disorders of; SALIVATION; and TASTE, Disorders of.

SEVILLE, in Spain.—A variable, rather bracing, inland winter climate. *See* CLIMATE, Treatment of Disease by.

SEWAGE-PURIFICATION.—*See* PUBLIC HEALTH, p. 1339.

SEXUAL FUNCTIONS IN THE MALE, Disorders of.—The most important disturbances of the sexual functions in the male are described under the following headings, to which the reader is referred: IMPOTENCE; MASTURBATION; SPERMATORRHOEA; STERILITY IN THE MALE; and TESTES, Diseases of.

SEXUAL ORGANS, Diseases of.—The diseases of the sexual organs in the male and female respectively will be found described under their special headings. *See* PENIS, Diseases of; TESTES, Diseases of; OVARIES, Diseases of; UTERUS, Diseases of; VAGINA, Diseases of.

SHAKING PALSY.—A synonym for paralysis agitans. *See* PARALYSIS AGITANS.

SHAMPOOING.—*See* MASSAGE.

SHANKLIN, in the Isle of Wight.—Exposure E. Prevailing winds westerly. A bright, picturesque, tonic health-resort, with good sea-bathing. *See* CLIMATE, Treatment of Disease by; and SEAIR and SEA-BATHS.

SHINGLES (*cingulum*, a girdle).—A popular name for herpes zoster.—*See* HERPES ZOSTER.

SHIVERING.—*See* RIGOR.

SHOCK.—SYNON.: Fr. *Choc*; Ger. *Shok*; *Wundstupor*; *Wundschreck*.

DEFINITION.—By the term 'Shock' is understood that condition of general depression of vital activity which follows upon a violent stimulation of the peripheral nerves or nerve-endings of the sensory or sympathetic system. It may also be produced by a sudden or severe emotion.

GENERAL DESCRIPTION.—As the result of a severe injury to a part richly supplied with nerves, such as the end of a finger, of laceration of a limb or large joint, or of a blow upon the abdomen, the subject is brought to a condition of general apathy. The surface becomes cold and clammy, the skin covered with perspiration, the aspect vacant yet anxious, the pulse feeble, the respiration weak, sighing, and irregular.

ÆTIOLOGY.—This condition may immediately follow the cause or may arise after an interval of more or less duration, as in the case of severe burns; here a state of excitement may give way suddenly to that of shock, which in such cases is generally followed by death. On the other hand, shock may be transient and may pass off rapidly, the intensity being generally determined by the nature, extent, and situation of the injury which brings it about. The effect is also largely dependent upon the mental and physical condition of the subject; other determining factors being age, sex, and habits. Any injury of even moderate intensity is much more serious in its effects upon the aged than upon younger and more robust subjects, and this in proportion to the evidences of senile degeneration. Children, on the other hand, resist the effects of severe injury, except in the case of severe burns or exposure to cold, far better than adults; while, on the contrary, operations upon the abdominal viscera are extremely hazardous in the very young on account of the supervision of shock even when the operation is in itself of comparatively slight gravity. Women, as a rule, bear severe operations better than men, but the effects of severe injury are more liable to produce disturbance of the nervous system, especially in the neurotic or hysterical.

The condition of the mind at the time of infliction of the injury has marked effect in determining the degree of shock. Thus, soldiers in battle are often unconscious of severe wounds until some time after their occurrence, but the depression that follows may be increased in proportion to the previous excitement. The same is true of emotional shock, where, as in the case of a criminal, the whole nervous system being in a state of the highest tension, a reaction follows on the pronouncement of the verdict, and a state of shock follows. *See* COLLAPSE.

Thus it is seen that shock is produced by injury occurring to parts most richly supplied with sensory nerves or viscera which are largely connected with the sympathetic system. The nature, extent and situation of the injury are important ætiological factors. Thus, crushing of a finger, blows upon the testicles, or injury to any of the abdominal viscera are among the most frequent causes. Burns, by reason of the large area involved and the consequent number of nerve-ends exposed, are most frequently followed by shock (p. 675). Formerly it was noticed that in amputations shock was found to occur when the medulla of a long bone was divided, or when in removal of a testis the

cord was cut through. But the more perfect knowledge of the art of anaesthesia has considerably reduced the frequency and the severity of such occurrences, though it has not abolished them entirely. Thus, it has frequently been observed that in patients only slightly anaesthetised, the division of the spermatic cord is attended by an immediate and sudden failure of the pulse, but that this does not occur when anaesthesia is complete (p. 61). The same is true in such major operations as amputation at the hip or shoulder, though collapse may follow as a result of haemorrhage. Cold or any previous condition which lowers vitality and leaves the nervous system depressed is naturally a predisposing cause. The temperament of the individual has to be taken largely into account, the so-called neurotic being much more prone to its occurrence than the robust and stalwart, while the alcoholic is a type that most frequently exhibits the results of shock in its most marked forms. When the whole nervous system is in a state of tension from expectation the liability to shock is far greater, while previous loss of blood by depleting the nerve-centres renders the patient much more susceptible than he otherwise would be. Subjects of organic disease, especially of the heart or kidneys, are more prone to exhibit the effects of shock, which in such persons is more severe and dangerous. Sharp injuries to tense fibrous structures frequently cause a sudden sense of faintness which is quite out of proportion to the damage done to the tissue. Thus a blow on the eye as from a spent pellet, or a kick upon the ankle or knee, may occasion a transient syncope often followed by a feeling of nausea. Blows upon the epigastrium cause a more lasting condition of the same nature, and the effects are occasionally so serious as to cause death.

PATHOLOGY.—The phenomena of shock are produced by the combined action of (1) the effect on the nerve-centres of massive sensory and sympathetic impulses from the injured part, by which the nerve-centres are exhausted. Though the sensory centres seem to be primarily affected, the closely connected motor areas are also involved. (2) Reflex action through the vagus, by which the heart is inhibited (at least temporarily), so that the blood-pressure rapidly falls. (3) Reflex action through the vaso-motor centre, so that the peripheral vessels are dilated, especially those of the splanchnic area, as a result of which the patient 'bleeds into his own vessels.' This also accounts for a fall of blood-pressure more lasting in effects than the vagus-action upon the heart.

The one striking phenomenon that is revealed, though not invariably, at *post-mortem* examinations of cases which have succumbed to shock, uncomplicated with excessive haemorrhage from the seat of injury, is an enormous distension of the abdominal vessels governed by the splanchnic nerves. Thus the abdominal viscera are engorged with blood destined for distribution over the rest of the system, and, as a consequence, the medulla and spinal cord are reduced to a state of feeble functional power, the first effects of which are shown in the action of the heart and the parts supplied by the vagus. This enfeebled action of the heart, and the scanty supply of blood which passes through its cavities, lead to a great reduction of the general vital activity and produce the symptoms which have been detailed. It has been shown by experiment that by stimulating the central

end of the divided depressor branch of the vagus in the rabbit, or striking the abdomen of the frog, the circulation can be profoundly modified by impulses that reach the vaso-motor centre, and the same result is probably produced in man by severe blows upon the epigastrium. Such procedures, by dilating the arterioles, and so lessening the peripheral resistance to the circulation, cause a fall of arterial blood-pressure more lasting than the vagus-action on the heart. Thus swallowing and all voluntary actions are impaired, though vomiting may herald a return of blood to the medulla. The temperature is lowered two or more degrees, though it may rise before death; the skin is moist, owing to vaso-motor changes, and the secretion of urine is suppressed. As defined by Mansell Moullin, 'shock is an example of reflex paralysis in the strictest and narrowest sense of the term, a reflex inhibition, probably in the majority of cases general, affecting all the functions of the nervous system, and not limited to the heart and vessels only.'

SYMPTOMS.—Whether it be due to injury, to emotion, or to exposure of nerve-ends over a large area or other cause, the occurrence of shock is rapid, though its advent may not be immediate, and may occur insidiously after some interval. It may vary in degree from a transient faintness to prolonged syncope or even death. In the slightest form the patient becomes pale and faint, often falling or clutching at the nearest object to prevent himself from doing so. The surface becomes cold and moist, beads of perspiration break out over the face and body, the hands are clammy, the pupils dilated. At the same time the muscles of the body are relaxed, the eyelids droop, the reflexes are slight. Consciousness is not absolutely lost, and questions are answered in a slow and feeble manner. There are 'all the evidences of general paresis of volition.' The pulse at first is slow, weak, and often dicrotic, with a tendency to become frequent, irregular, and imperceptible. The respirations are feeble, sighing, and shallow. The temperature is always low, often falling as much as two, three, or even more degrees. The expression is vacant. These conditions are more or less marked according to the severity of the cause and the temperament of the individual. The situation of the lesion has important bearing upon the effect produced, a small wound of the intestine producing much more severe consequences than extensive injury to a limb. The same conditions govern the duration of the symptoms. These may last from a few minutes to several hours before reaction commences. This is often heralded by an attack of vomiting due to a hyperemia of the nervous centres which follows the previous abstraction of their blood-supply. After this the pulse gradually becomes rhythmic and fuller, pallor gives place to a flushed colour, and the senses gradually recover, though a feeling of weakness remains, often for a considerable period. On the other hand, the symptoms may increase in severity notwithstanding all treatment, the pulse and respiration becoming more feeble and imperceptible until death occurs.

Travers described shock as occurring in two forms: (1) with great prostration, in which the symptoms are those just described; (2) with exhaustion followed by traumatic delirium. This is sometimes termed *erethitic* or *erethistic* shock, but is not so frequently observed now that the art of anaesthesia is better understood (p. 63). It may, however, occur as a sequel to the former condition, especially

after major operations, or in association with previous hæmorrhage; or when there is great pain, as in crushing injuries of important parts, or after burns or scalds. It is marked by great and increasing restlessness; the consciousness may be but little impaired, but questions are scarcely heeded, and traumatic delirium is a frequent sequel. It is probable that this condition is due to the introduction of a toxic element.

'Railway shock' is a form of traumatic neurosis which may develop immediately or even many weeks after an accident to a train, from which the individual may have experienced no manifest sign of injury (*see* RAILWAY INJURIES). Headaches and a feeling of lassitude are first complained of, and these are followed or accompanied by sleeplessness and nervous irritability. The mental attitude is altered, the patient becomes despondent, and symptoms of melancholia develop. Often there is numbness and tingling in the extremities, and the reflexes are increased as in ordinary neurasthenia. Some cases may have marked hysterical features, or symptoms that suggest organic disease of the brain or spinal cord. Bladder-troubles and signs of sclerosis of the cord may be well marked. The muscular powers of the limbs are enfeebled, the circulation becomes weak, the intellect dull, and the general health deteriorates. These conditions may progress until the patient dies in a condition resembling general paralysis. The anatomical changes are not definite. The brain and spinal cord have sometimes exhibited punctiform hæmorrhages; and arterio-sclerosis of the vessels, with scattered areas of degeneration in the white substance, has been observed, as well as degeneration of the sympathetic ganglia. Although death may not be directly due to this cause, and although a perfect recovery may ensue, yet a deterioration of health is the usual sequel, or some impairment of a special sense, or the mental vigour and temper of the individual may be changed for the worse.

DIAGNOSIS.—This must depend to a great extent upon the nature of the cause and the situation and character of the injury. Where no lesion has occurred and where mental emotion is the only cause of the symptoms there is no question of the nature of the condition. Generally the distinction between shock and collapse is not distinctly marked, the one occurring preliminary to the other (*see* COLLAPSE). In the case of wounds, shock is followed by collapse as the result of loss of blood. In severe injuries attended by excessive hæmorrhage the collapse is mainly due to the loss of blood. Thirst, restlessness, and dyspnoea are then the predominant symptoms, together with the rapid but feeble and easily compressible pulse. The mental condition is little affected except that it evinces great irritability and excitement, which do not quickly subside. In the case of rupture or perforation of the intestine shock precedes collapse, which occurs as the result of peritonitis due to the absorption of toxic elements.

Uncomplicated shock, except in cases of very severe injury, tends towards recovery; collapse, on the other hand, is much more likely to be fatal. Shock differs from syncope in that the prostration is much more prolonged, and consciousness, though blunted, is not abolished. In head-injuries the symptoms of concussion (p. 335) are not necessarily accompanied by those of shock. The senses are almost or completely dulled, but there is not gene-

rally the same blanching of the surface, or the utter prostration which is manifested in shock, nor is there a fall of the temperature to the same extent. The two conditions may, however, co-exist. The state of the pulse will generally indicate the difference, being slow, full, and regular in direct injury to the brain; faint and irregular in cases of shock. Where there is internal hæmorrhage it is often impossible at first to distinguish the conditions, but the advent of syncope and the absence of reaction, together with the nature and situation of the injury, will soon show the predominant features. A faint condition with pallor often precedes vomiting in patients under the influence of an anæsthetic, and is recovered from as soon as vomiting has taken place, when the anæsthesia may be continued.

PROGNOSIS.—This depends mainly upon the nature and extent of the injury, and upon the physical and mental powers of the individual. The longer the condition of shock endures, and the lower the fall of temperature, especially if it sinks below 96° F., the more unfavourable is the outlook, and the prospect becomes graver the longer reaction is postponed.

The question of operation during the continuance of shock must be judged by individual cases. As a general rule it is the practice of surgeons to wait until signs of reaction have appeared; but where it is evident that the injured portion is maintaining the condition, or where bleeding is evidently proceeding, there is no reason to delay. In such cases an anæsthetic should be given, and for this purpose ether is clearly indicated (p. 61), since by its stimulating effects upon the action of the heart it relieves the anæmia of the medullary centres, and as the pulse improves under its influence the effects of injury to the nerve-ends are greatly palliated.

TREATMENT.—A consideration of the causes and conditions obtaining in shock is a sufficient indication as to the lines of treatment to be followed. In slight cases very little is required except to place the patient in a recumbent position and apply some aromatic stimulant, such as ammonia, to the nostrils. In more severe or prolonged conditions the patient must be kept recumbent, with the head at a lower level than the feet. Warmth is the essential to overcome the vaso-motor paralysis, and therefore the temperature of the room must be maintained at between 80°-90° F. Hot-water bottles or hot bricks wrapped in flannel should be applied to the feet and sides of the body. Very hot fomentations placed over the præcordia and on the nape of the neck are most effectual in stimulating the heart's action. Where possible the patient may be placed in a hot bath at a temperature of 104°, gradually rising to 110° F. The pulse and respiration must be carefully watched, and in desperate cases the phrenic nerve may be stimulated by the galvanic battery, the poles being placed along its course in the neck and over the epigastrium. If swallowing is possible small quantities of brandy, or, at a later stage, strong black coffee, should be given; but where this is not feasible subcutaneous injection of ether or of strychnine must be given and repeated at intervals. Where there has been excessive hæmorrhage injections of hot water and brandy into the rectum, or the transfusion of normal saline solution into the veins of the arm, should be resorted to without delay (*see* SALINE SOLUTION, Infusion of). Though not admissible in the torpid

form of shock, in the state of excitement small injections of morphine should be given and an ice-coil applied to the head. The temperature and pulse must be carefully watched for any indication of inflammatory reaction, and the treatment regulated accordingly. The greater the effect produced the greater must be the care in the subsequent treatment of the case. The eyes must be protected from light, and all excitement must be avoided for many days. Belladonna to stimulate the heart's action, or opium to quiet the nervous system, must be sparingly used. *See* also HEAT, Effects of Severe or Extreme, p. 676.

JOHN HAMMOND MORGAN.

SHOEMAKER'S SPASM.—*See* OCCUPATION-DISEASES; and WRITER'S CRAMP.

SHORTNESS OF BREATH.—*See* RESPIRATION, Disorders of.

SHORT-SIGHTEDNESS.—*See* MYOPIA; and VISION, Disorders of.

SIALAGOGUES (*σίαλον*, saliva; and *ἄγω*, I move).—*SYNON.*: *Fr.* *Sialagogues*; *Ger.* *Speicheltreibende Mittel*.

DEFINITION.—Remedies which increase the secretion of saliva.

ENUMERATION.—The principal sialagogues are Dilute Acids, Ether, Ginger, Rhubarb, Horseradish, Iodide of Potassium and other iodides, Jaborandi, Mezeoron, Mercury and its salts, Mustard, Tobacco, Physostigmine, Pyrethrum, and Pebbles.

ACTION.—There are two essential factors in the secretion of saliva: the first is the activity of the secreting cells in the gland; the second is a sufficient supply of nutritive material to them, from which they may form a secretion. This nutritive material, though it may be derived directly from the lymph-spaces around the cells, must be ultimately supplied by the blood circulating through the glands. Usually, therefore, when the gland is in action, the supply of blood is greatly increased, the arteries dilating, and the blood flowing rapidly through them. Some drugs, such as physostigmine, will stimulate the secreting cells, while they contract the blood-vessels; under these circumstances, although the secretion may begin actively, it soon comes to a standstill from want of material. The secreting cells may be excited to activity by substances which stimulate the nervous structures within the gland itself, as, for example, Calabar bean (physostigmine); by stimuli proceeding directly from the encephalon, as seen in salivation occurring from the mere idea of savoury food; and by stimuli applied to the mouth and exciting the gland reflexly. Nausea is almost always accompanied by salivation, and substances which cause nausea almost invariably cause salivation, the irritation of the stomach causing reflex salivary secretion. The stimulus here passes up the afferent nerves to the medulla, and travels down the efferent nerve to the gland.

Sialagogues are divided, according to their mode of action, into two classes—(1) *topical or direct*; and (2) *specific, remote, or indirect* sialagogues. The names *direct* and *indirect* are complete misnomers, just as they are in the case of emetics, and they ought to be discarded, inasmuch as the so-called 'direct' sialagogues are those which do *not* act

directly on the gland, but on the mouth; and the 'indirect' are those which do act upon the gland, affecting either the nervous structure contained within it, or the nerve-centres directly connected with it.

The *topical* sialagogues are dilute acids, ether, ginger, rhubarb, horseradish, mezeoron, mustard, pebbles, pyrethrum, and tobacco. The *remote* sialagogues are iodide of potassium and other iodides, jaborandi, mercury and its salts, physostigmine, and tobacco.

Topical sialagogues excite secretion of saliva reflexly, the afferent nerves being the lingual and buccal branches of the fifth, and the glosso-pharyngeal nerves. The afferent nerves, through which nauseants probably excite the salivary secretion, are the vagi.

Of remote sialagogues, iodide of potassium probably acts upon the gland-structures, but upon which part has not yet been determined. It may, however, also act reflexly, by stimulating the sensory nerves of the mouth, as it is excreted in the saliva, and the taste of it is often persistent. Mercury probably acts partly by affecting the gland-structures, and partly by affecting the mouth. Jaborandi, physostigmine, and tobacco appear to affect the terminal branches of the secretory nerves in the glands.

USES.—Saliva is useful in keeping the mouth moist, and thus facilitating mastication, deglutition, and the movements of the tongue in speaking. By moistening the fauces it also prevents or lessens thirst. A pebble placed under the tongue, or masticated, or an effervescent lozenge, will keep up a slight flow of saliva, and may be useful for these purposes. Where this is insufficient, dilute acids are employed (*see* ACIDS). As the flow of blood to the glands is greatly increased during secretion, sialagogues, and especially pyrethrum, have been used as derivatives, to lessen inflammation, congestion, and pain in other parts of the head, as in toothache, earache, and inflammation of the ear, nose, or scalp. Saliva has also, however, a digestive power upon starch, and increase of the flow may be advantageous in imperfect digestion of this substance. When swallowed, the saliva stimulates the secretion of gastric juice, and increased salivary secretion therefore tends to aid gastric digestion. To attain this object it is best to chew a piece of ginger or of rhubarb.

Dryness of the mouth and tongue, especially on awakening in the morning, often arises from breathing through the mouth in consequence of nasal obstruction. In such cases treatment of nasal congestion and removal of obstruction such as adenoids are more useful than sialagogues.

T. LAUDER BRUNTON.

SIBILANT RÂLE or RHONCHUS: SIBILUS (*sibilus*, whistling).—A variety of dry *râle* or rhonchus, of a whistling or high-pitched musical character, usually produced in the smaller divisions of the bronchi. *See* PHYSICAL EXAMINATION; and RHONCHUS.

SICILY.—A warm, moist, winter climate. Climate of base of Etna more variable than of N. coast. *See* CLIMATE, Treatment of Disease by; and PALERMO.

SICK-HEADACHE.—A popular synonym for *megrin*. *See* MEGRIM.

SICKNESS.—A common synonym for vomiting. See VOMITING.

SICK-ROOM, Disinfection of.—See PUBLIC HEALTH; and DISINFECTION.

SIGHT, Disorders of.—See VISION, Disorders of.

SIGNS OF DISEASE.—See PHYSICAL EXAMINATION.

SILICOSIS.—See PNEUMOCONIOSES.

SINGERS' NODES.—See p. 853.

SINUS and FISTULA. — DEFINITION. — A track leading from a surface into the tissues and ending blindly is called a *sinus*. When the track passes from one surface to another, or from one part of the same surface under a bridge of tissue to another part, it is called a *fistula*.

ÆTIOLOGY.—The chief conditions which give rise to sinuses and fistulae are the following: (1) *Congenital defects*: tracks due to these may be present at birth, or may form in later life; (2) *Wounds*; (3) *Abscesses*, which burst on one or two surfaces, leaving a track which does not close; (4) *Ulceration* through a natural septum, or through a partition formed by the adhesion of separate structures, as of the intestine to the bladder; (5) *Sloughing* of a portion of the wall of a channel or cavity; (6) Infiltration of the wall of a hollow viscus, or of the tissues separating two such viscera, by *new-growth*, with subsequent degeneration and breaking down of the tumour.

The natural tendency of any pathological channel formed in the tissues is to close up and become obliterated. A variety of causes may, however, prevent this, and so lead to the formation of a sinus or fistula. Thus (a) imperfect drainage of a cavity may cause retention of discharge, which may burst out at intervals as the tension rises; (b) Traumatism, gangrene or ulceration may have destroyed so large an amount of tissue that the healing process cannot make good the gap; (c) When union has been long delayed, owing to constant drainage of some secretion or excretion through a track, epidermis may grow in from the surface and form a lining to the tube, thus preventing the possibility of spontaneous obliteration; (d) A foreign body, a sequestrum, or a caseous gland may keep up a discharge for an indefinite period; (e) The wall of the channel may be formed of unhealthy tissue—tuberculous or cancerous—and show no tendency to undergo repair; (f) Continual movement of surrounding muscles may disturb that rest of the part which is necessary for the occurrence of healing.

A common variety of sinus is that seen in connection with chronic disease of bone, due to tubercular or pyococcal infection (acute necrosis). Such a sinus is practically a tubular ulcer. It is lined by granulation-tissue, which is irritated by constant passage of the discharge and by the presence of septic organisms. Examples of fistulae are seen in cases of remnants of branchial clefts in the neck; a 'fistula in ano,' in which a track is found passing beneath the mucous membrane of the anus and opening both externally below and into the gut above; and in cases of vesico-vaginal and recto-vaginal fistulae, caused by injuries inflicted in parturition or by the

involvement of the tissues separating the cavities in masses of malignant growth.

DIAGNOSIS.—When a sinus or fistula opens on to or near the external surface of the body, its nature is readily determined by the eye or by the use of a probe. The opening may be very minute and need careful search. When the orifice is out of sight, the presence of a fistula may be revealed by the escape of abnormal material from a cavity, as of urine from the rectum, or of gas and feces from the urethra.

TREATMENT.—Cure of a sinus or fistula may often be effected by laying it open throughout and plugging the cavity, which then heals from the bottom by granulation. A careful watch must be kept to prevent premature union of the edges. If the condition be chronic, the fibrous wall of the track must be cut quite through on its deep aspect; it is still better to excise the whole track. Excision must also be practised in cases of congenital sinuses and fistulae which are lined by epithelium. Closure of the wound by 'purse-string' sutures sometimes brings about speedy union. Any foreign body, sequestrum or tubercular focus must be removed. Malignant fistulae can rarely be dealt with; excision of the growth and suture of both openings has been performed. In many cases of fistula complicated plastic operations are necessary to effect a cure.

STANLEY BOYD.

SINUSES, CEREBRAL, Diseases of.—See MENINGES, Diseases of.

SINUSES, NASAL, Diseases of.—See NOSE, Diseases of.

SIXTH NERVE, Diseases of.—The sixth nerve, or *abducens oculi*, confers motor power on the external rectus muscle of the eyeball, and its morbid states of excessive or defective function are indicated by corresponding spasm or paralysis of that muscle.

1. **Spasm of the external rectus.**—This condition is very rare, except in myopia, in which an effort at accommodation is needless, and the associated action of convergence is also unnecessary. Hence the muscles causing divergence act in relative excess. Permanent contraction occurs when there is complete paralysis of its antagonist, the internal rectus. Spasm may occur from irritation of the nucleus or fibres of the sixth nerve, as in meningitis of the base. The symptoms are inclination outwards of the affected eye, and consequent divergent strabismus. The treatment is that of the cause on which it depends. See STRABISMUS.

2. **Paralysis of the external rectus.**—**ÆTIOLOGY.**—Among common causes are tumours, and other lesions within the pons, and various forms of syphilitic disease. The latter may cause a specific growth on the nerve, or damage it by inflammation and compression by the new tissue which is so abundantly formed in syphilitic meningitis. The nerve suffers also in other forms of meningitis with great readiness, on account of its long course, which renders it liable to be involved in various regions of the base, and to be affected more frequently than any other cranial nerve. Its course over the convexity of the pons involves a liability to suffer from any cause, such as a tumour, which increases the pressure beneath the tentorium. Hence tumours of the base of every kind are frequent causes of its paralysis. It suffers also from

disease in the anterior part of the base, at the orbital fissure, and within the orbit, when the nerves to other ocular muscles are usually also involved. Isolated neuritis, due to cold, is a rare cause. It may also be paralysed in tabes, with or without other nerves to the ocular muscles.

SYMPTOMS.—Paralysis of the external rectus causes inability to move the affected eye outwards, and hence convergent strabismus, and homonymous diplopia when looking at an object on the affected (e.g. left) side of the middle line, the images becoming more distant as the object is moved to the left; but parallel, and on the same level, so long as it is on the horizontal plane. When looking also up or down, the second image slants, the two being nearer together at the lower end, and the second image the lower of the two when looking up. On looking down, the two images are nearer together at the top than at the bottom, and the second image is on a higher level than the other. There is erroneous projection of the field of vision, so that the hand is moved beyond an object on the side which an attempt is made to touch—the muscular effort being guided by the increased innervation. There may be for a time a tendency to giddiness when the affected muscle is used.

DIAGNOSIS.—Paralysis of the sixth nerve is easily recognised, except when slight in degree. In the latter case it may often be detected by a careful search for the diplopia, or by the secondary deviation of the sound eye in the same direction when that eye is covered and an object fixed by means of the weak muscle. See STRABISMUS.

PROGNOSIS.—The prognosis is most favourable when the paralysis is due to cold or syphilis; least favourable when due to meningitis or tumour. When associated with ataxy recovery is common, but is often followed by a relapse, which frequently does not entirely pass away.

TREATMENT.—When the complaint is of rheumatic origin, the treatment should consist of hot fomentations to the temple; afterwards counter-irritation by blisters; and salicylates, with small doses of mercury and tonics internally. If of syphilitic origin, iodide or mercury should of course be given.

For the palsy associated with tabes strychnine and arsenic may be employed. The use of electricity in any form or mode of application is without appreciable influence. For the palsy which is part of external ophthalmoplegia, the hypodermic injection of strychnine gives the best results in cases that are not of too long duration.

W. R. GOWERS.

SKIN-BOUND DISEASE.—A popular synonym for sclerema neonatorum. See SCLEREMA NEONATORUM.

SKIN-GRAFTING.—Skin-grafting consists in the transplantation of portions of skin of varying size and thickness from a healthy part of the body of the patient himself, or of some other person or animal, on to a recently made raw surface or a granulating sore.

Skin-grafting thus differs from a plastic operation, which involves the shifting of a flap of skin and subcutaneous tissue from one part of the body to another, without completely dividing its vascular connections with contiguous parts.

METHODS.—Several years ago Reverdin introduced the plan, which might more properly be

called epidermis-grafting, of placing minute portions of the superficial layers of the skin, containing, however, the cells of the *rete Malpighii*, on a healthy granulating surface, to which they adhered and served as the starting-points for the spread of epidermis over it. The grafts were shaved off from any healthy part of the skin with a sharp knife, the incision being barely deep enough to draw blood. They were then placed, with the deep surface downwards, on the sore, and not very far from one another, as each graft does not, as a rule, grow readily to a larger size than that of a sixpenny-piece. It is presumed that in all operations of skin-grafting close attention will be paid to keeping both the graft and the sore aseptic, the way of accomplishing which will be given in a later paragraph. Neglect of this precaution exposes the grafts to the irritating influence of putrefaction, which much endangers their vitality.

More recently Thiersch has brought forward a great improvement on this method. It consists in shaving off with a broad razor or special knife a strip of the superficial parts of the skin (as in Reverdin's plan, going just deep enough to draw blood, but no deeper) one inch wide and of any required length, and preferably from a part where the skin can be easily made tense by grasping the opposite side of a limb, such, for example, as the anterior surface of the thigh or the outer side of the arm. This strip is either laid on the raw surface entire, or is cut into pieces half an inch or more long, which are to be placed quite close together over the granulating or raw surface. If the former, the superficial granulations must be previously scraped away, so as to remove any irregularities and leave behind only a material which, though highly vascular, is little prone to contract, because it has already contracted as much as is possible. The grafts must not be placed in position until all bleeding has stopped, and this result may be encouraged by exerting pressure with a sponge over a piece of some smooth material, such as oiled-silk protective, placed upon the wound.

Watson Cheyne has more recently still recommended that the whole thickness of the skin should be used for the grafts. This has the disadvantage that contraction must occur in the part from which the graft is removed, but the advantage that it is reduced to a minimum in the part to which the graft is transplanted. It should certainly be adopted in cases such as excisions of the mamma, where a piece of redundant skin from the axilla may be used to fill a gap between the flaps on the front of the chest. The deep surface of the graft may be prepared by chipping off, with a sharp pair of fine scissors curved on the flat, any pieces of fascia or fat remaining adherent to it.

PRECAUTIONS.—In performing the operation, the part from which the graft is to be taken must be purified by shaving it and washing it thoroughly with a solution of carbolic acid (1 : 40), of corrosive sublimate (1 : 1000), or probably the desired result would be obtained by using simply alcohol mixed with 25 per cent. of water. If any time is to elapse between its removal and its application to its new seat, it may be kept in a sterilised saline solution heated to the temperature of the body. If it is to be placed on a surface which is already septic, this must be prepared by the application for a few days of a moist antiseptic application frequently changed, say boric lint soaked in a 1 to 2000

sublimate solution; and it is often well to begin this treatment by purifying the skin with 1 to 20 carbolic-acid lotion, and the sore with a solution of chloride of zinc, 40 grs. to 1 oz. A piece of oiled-silk protective should be placed on the grafts before the antiseptic dressing, whatever it may be, is applied. Care must be taken to prevent the edges of the grafts from turning over. If the grafts be not placed close together, lines of granulations may form between them, and much interfere with the presentableness and stability of the scar.

Some surgeons have obtained the grafts from the skin of frogs and other animals, but we have not sufficient knowledge yet to speak positively in recommending this plan. The skin of the prepuce has also been used for the purpose. The best and safest source of supply is undoubtedly the skin of the patient himself, for it has happened that both tuberculosis and syphilis have been transmitted when the graft has been taken from another person.

In *plastic operations* a flap of skin and subcutaneous tissue decidedly larger (as three to two) than the surface to be covered is turned up from some contiguous or distant part, but left attached by a sufficiently wide base to ensure its retaining its vitality. The flap is then shifted round and secured by sutures to the raw surface, which it must accurately fit. If the part from which it is to be transplanted be at some distance from that to which it is to be adapted, the part on which the latter is situated must be firmly secured in position until the flap has formed its new connections, when the pedicle is to be divided, and the part to which it has now become adherent is allowed to assume its natural position.

RICKMAN J. GODLEE.

SKIN, Tuberculosis of the.—It is only of late years that the group of cutaneous tubercloses (tuberculodermiae) has been constituted and consolidated from among the affections included under the terms Tubercular Ulcer, Lupus, Scrofulodermia, &c. Virchow taught that the skin was refractory to tuberculation, and possibly the more superficial layers from reasons of anatomical structure or temperature are so; but, however that may be, tuberculosis of the skin is of frequent occurrence, and characterised by a great diversity of features dependent on the virulence and quantity of the bacillus and its products, the quality of the soil, the mode of origin, the anatomical characters of the site involved, and the complications supervening. The skin may be so affected at any age, but children and adolescents are especially prone. Not infrequently the subjects present characteristics which, experience teaches us, mean a vulnerability of the tissues and a susceptibility to tubercular infection. Such features are a feeble peripheral circulation, a quality of skin sometimes portrayed in the 'lymphatic facies,' and a liability to chronic catarrhs of mucous membranes and of the skin with pus-formation. Further, the presence of a special susceptibility may be inferred from the history of tuberculosis in the family or the antecedents of the patient; or tuberculosis may be actually present in some other tissue. Yet often the subjects are apparently quite healthy, and the inoculation may be traceable to the special risks of their surroundings.

The criteria by which the tubercular nature of a skin-lesion is determined are: (1) the clinical features (morphology and infective character) which experience has taught us; (2) the characteristic

pathological changes in the tissues; (3) the presence of the special bacilli, which, however, may be few in number or temporary, and thus difficult to find; (4) the transmission of the disease by the serial inoculation of morbid products into certain animals when proper conditions are fulfilled; (5) the reaction to Koch's tuberculin.

The orderly arrangement of the diverse clinical manifestations which conform to these criteria is not easy, but for clearer exposition it will perhaps be convenient to retain for the present certain distinctive terms, viz.: (1) *verrucose tuberculosis* of Riehl and Paltauf, including the so-called *verruca necrogenica* (S. Wilks), or *post-mortem wart*; (2) the *primary tubercular ulcer*; (3) the *secondary granular tubercular ulcer*, '*tuberculosis propria seu miliaris*' (Kaposi); (4) *Lupus tuberculosus* vel *L. vulgaris*, with its diverse modifications; (5) the *tubercular gumma*; (6) the so-called *Scrofulodermie*; (7) '*Tuberculides*.'

Certain *paratuberculides*, such as pigmentary disturbances, which may occur in association with tuberculosis, will not be further referred to here. *Lupus erythematosus* is also included under cutaneous tuberculosis by some authors.

Lupus vulgaris.—Lupus is a figurative term, which was applied to diverse affections, and, although Willan's definition limited the application of the term, it is only in late years that the tubercular nature of the disease has been definitely established. It is the most common and characteristic reaction of the skin to the direct inoculation of the tubercle-bacillus, and must be considered as the type, but both in the skin and mucous membranes it differs markedly in its symptomatology from the miliary tubercular ulcer of the tongue and muco-cutaneous orifices. It may arise at any period of life, but with especial frequency in childhood and adolescence (75 per cent. before the age of twenty), and nearly twice as often in girls as in boys.

As to the *origin*, the primary patch may be inoculated from some outside source, or may arise by the implication of the skin from pre-existent tuberculosis of deeper structures in the same subject, e.g. from glands, bones, and synovial sheaths. In the latter case it may present the characteristic nodules, or the more diffuse form of infiltration to which the term *Scrofulodermia* is applied. This glandular origin is very frequently seen over the parotid region, or from ear to ear beneath the jaws. Lupus is said to originate commonly by auto-inoculation from the discharges from the lungs, and not uncommonly from a mucous membrane, especially that of the nose. Dubreuilh maintains that there is a tubercular catarrh of the nose without neoplastic tendency, and if so, the analogy with leprosy is interesting. Lastly, there is a rare phenomenon, generally occurring in children after some systemic infection such as measles, characterised by the sudden evolution of more or less numerous disseminated small nodules, sometimes of an acneiform type. All or many may gradually disappear, but in some cases a number persist and assume the recognised characters of lupus. Such eruptions must be studied in connection with the '*tuberculides*' to be discussed later on. Baumgarten thought most cutaneous tubercloses had such a blood-origin.

The *elementary lesion* is a nodule in the derma, superficial or deeper-seated, imbedded or projecting; varying in size from a mere speck to a pin-head, hemp-seed, or pea; of a notably soft consistence, a

reddish-yellow colour and semi-transparent, recalling apple-jelly, especially when the blood is pressed out of the tissues; very indolent, as a rule, and with slow evolution; and unaccompanied by pain or itching. It may persist, or pustulate, or ulcerate, or disappear spontaneously, leaving a cicatrix.

A patch of lupus is formed, and spreads excentrically, by infection of the vicinity of the primary nodule, and the evolution of 'satellite' nodules, which become confluent with the enlarging older lesions. The latter are often replaced by scars, while the excentric spread is maintained around or on one side. Its *infective power* may be little marked, and often after a certain spread, lupus may remain quiescent for years, without infecting other tissues. The attenuation of the virus may also be evidenced by the difficulty in finding bacilli, and by the special conditions required for successful inoculation in animals. Usually, however, lupus pursues a very indolent spreading course, but sometimes takes on quite a rapid extension over large tracts. Infective power, however, may be evidenced, not only by the method of spread in the skin, but by the involvement of the related lymphatic glands, and on a limb of the lymphatic vessels. Much diversity of opinion exists as to the frequency of a systemic infection; for instance, the late J. S. Bristowe made the remarkable statement that 'local skin-tubercle or lupus, instead of producing general tuberculosis, seems to prevent it.' On the other hand, it is common experience that a certain proportion of lupus eventually die of visceral tuberculosis, but we must attach due weight in this connection to the hospitalisation, the confinement, the wretchedness brought about by their disfigurement, and the poverty of many of these sufferers. As to *localisation*, any region may be attacked, but in about 80 per cent. of cases lupus is seated on the face, and especially about the nose and its neighbourhood. It may extend to the forehead, but is of great rarity on the scalp. It is not infrequent about the parotid region, and especially in a band beneath the jaw. The hands are particularly exposed. It is said to be rare on the external genitals, and Dubreuilh says that the *Esthiomène* of the vulva of Huguier is a chronic ulceration of different nature. The mucous membranes of the mouth and nose are frequently involved, usually secondarily to the skin; also the conjunctivæ, the pharynx and larynx; the tongue rarely. In the mucous membranes the redness is accentuated, the surface mammillated and easily bleeding, the infiltration soft. Ulceration is frequent and leads to destruction of the uvula and soft palate, and of the cartilages of the nose with mutilation as in syphilis and leprosy.

The *clinical phases* are infinitely diverse, owing to variations in size, form, aspect, and number of the patches, and to the predominance of certain reactions, such as marked vascularity, epithelial growth with hyperkeratosis (*L. papillosus*, *L. verrucosus*), hypertrophy of collagenous tissue or fibromatosis (*L. hypertrophicus*, *L. scleroticus*), or sero-fibrinous exudation without loss of underlying substance (*L. crustosus*, Unna). Mixed infections cause suppuration and necrosis (*L. vorax*, *L. phagedenicus*).

In size the patches may range from that of a small coin upwards, or the lupus may advance over an extensive tract, e.g. up a limb or from the face over the neck and greater part of the trunk. There may be only one patch present or others may

arise by auto-inoculation, or by lymphatic infection. The number is usually few, except in the exanthematic outbursts mentioned.

There is an old division into non-ulcerating (non-exedens) and ulcerating (exedens) lupus. Non-ulcerating lupus forms red patches, yellowish-brown when the blood is pressed out, generally projecting somewhat, with distinct contours, always distinctly infiltrated and thickened, with a smooth or exfoliating surface. A typical indolent patch of this kind is often seen in the centre of the cheek. Usually the older infiltrations disappear, and atrophic scarring results. The primitive rounded outline of the patch may be lost owing to the extension only in certain directions. On the thigh a circinate form is often preserved, but on the buttocks lupus-patches become hypertrophic. On the hands, and sometimes other parts, the lupus-infiltration is disguised by the warty growth as mentioned under warty tuberculosis.

On the feet lupus-nodules are deeply imbedded beneath the thick corneous layer and present difficulties in diagnosis. The ears may be enormously increased in size and shapeless with myxomatous degeneration. Occasionally soft exuberant red papillæ become very prominent over patches on the face, and exudation takes place *through the thinned epithelium* with crusting.

Ulcerated lupus may be heavily encrusted, and thus the primary infiltration be masked. Granulations may sprout from the surface, and form a picture not to be confounded with papillomatous lupus.

Cedema with lymphatic inflammation may occur in the arm and leg so as to simulate elephantiasis, or in the face.

Mutilations of a distressing character are brought about by the destruction of the cartilages of the ears and nose; by ulceration and suppuration of the hands and feet with loss of digits; and by the more or less contractile character of the scars formed about the eyelids, nose, mouth, and bends of the limbs.

The **Primary Tubercular Ulcer** is produced by the primary inoculation of tuberculosis into punctures, breaches of surface, or other deeper wounds, in various regions of the body. It would be more correct, however, to describe it as a tubercular nodule with a strong tendency to pustulation and ulceration, either from the virulence of the bacilli, the character of the soil, the method of inoculation, or the presence of other organisms. It is questionable whether we are justified in placing these inoculations in a special category. This phase may commence as a little red infiltration, which may pustulate, or ulcerate, and crust; or as a little abscess; or as onychia; or a wound may suppurate and ulcerate. The infective nature may be shown by the intractability to simple healing measures, the excentric spread of the lesion, and the implication of the related lymphatic vessels and glands and production of adenitis, 'cold abscesses,' and 'gummata,' for instance up the arm. Many striking examples of such tubercular inoculations have been recorded. The fingers may be the site of attack, but inoculation has followed the wearing of infected earrings (Unna), a cut on the scalp from falling on the sputum-receiver of a phthisical mother (Dencke), and so on. Various series of cases on record of tuberculosis inoculated by a tuberculous operator in the Jewish rite of

circumcision and again in tattooing are well known. Bacilli are often easily found.

The early recognition of the nature of these lesions is obviously important, that they may be thoroughly destroyed or removed.

Verrucose Tuberculosis (Riehl and Paul-tauf) is a manifestation of cutaneous tuberculosis also due to exogenous inoculation, and is moulded by the influence of site, for it occurs nearly always on the dorsal aspect of the hand or fingers, whence it may extend to the palms. It may be seen on occasion about the forearms and elsewhere. The peculiar features are due to the masking of the granuloma by hypertrophy and down-growth of the epithelium, and sclerotic changes. There is also a marked tendency to the formation of superficial milium abscesses, whence pus oozes between the warty cones, but the lupus-nodules never suppurate (Unna). There may be hardly any signs of congestion, but generally the extending painless warty patch has a violet-red border, and may cicatrise in the central parts. Sometimes the patch is indolent, and may remain stationary for years, while at other times the excentric spread is comparatively rapid, and occasionally the infective nature is further evidenced by the implication of a chain of lymphatic vessels and glands up the limb, and possibly of the viscera. The bacilli present are very variable in number. Bécère says this warty tuberculosis may be secondary to phthisis. Lupus in similar situations tends to assume a similar morphology, and it is difficult to draw a very sharp line of distinction between the two phases.

Verruca Necrogenica (Wilks), **Anatomical Tubercle**, or **Post-mortem Wart**, is a similar persistent thickened keratotic or warty condition of the hands, especially of the finger-points. It is often due to tubercular inoculation, but probably a similar condition is excited by contact with the morbid fluids of diseased bodies. It may begin without breach of surface as a keratotic or warty thickening, or in a wound, or as a pustule.

The Secondary Granular Tubercular Ulcer; Tuberculosis propria seu miliaris (Kaposi).—This is very similar to that met with on the tongue, and is found in the neighbourhood of the lips or anus, where it is secondary to pulmonary or intestinal tuberculosis. Kaposi says that, though rare, it is commoner than generally supposed. In this country it is certainly rare. The ulcer is peculiarly painful, irregular in outline, with ragged undermined borders and a reddish-grey uneven floor, studded with yellow milium granulations (tubercles), which may be seen in the borders and beyond, and which by their degeneration and softening cause the formation and spread of the ulcer. There is little tendency to spontaneous cicatrization. This ulcer is very rare in childhood, but has been recorded in the anal region. Bacilli can be readily detected in the secretion or in scrapings. The attentive and alert observer can hardly confound this ulcer with a syphilide.

Tubercular Gumma.—This term is applied to certain circumscribed tuberculous masses which originate in the hypoderm, and somewhat resemble syphilitic gummata. Two phases are met with. The first is the multiple and bilateral cutaneous cold abscess, the scrofulo-tuberculous gumma of Besnier, so commonly seen in early childhood and frequently associated with other signs of tuberculosis, as dactylitis, glandular trouble, &c. The nodules

are mostly seen about the limbs and commence in the hypoderm as a rounded, indolent, firm infiltration. As they increase from the size of a pea or nut, the overlying skin becomes violet, and then involved more intimately in the process. The softening mass may discharge its contents through a little hole, or sometimes breaks down to form an ulcer. These 'gummata' are of endogenous origin. The second form is seen in the adult. They sometimes assume a chain-formation up a limb from lymphatic infection, but on rare occasions occur in a disseminated phase.

Scrofuloderma we may pass over with an explanation. The term formerly denominated all the supposed phases of the so-called scrofulous diathesis implicating the skin, and so had a very extended use, varying with different authors. In the present day the term is for the most part applied to the so-called gummatous form and to secondary tubercles of the skin arising from disease of deeper structures, as glands, bones, &c. The latter lesions usually present a violaceous diffuse infiltration and they ulcerate.

Tuberculides.—Of late years particular attention has been drawn to certain regional, or more or less generalised, eruptions of multiple lesions, probably of endogenous origin, described under a great variety of names, and ranging from the grouped micro-papular *Lichen scrofulosorum* through some acneiform and nodular eruptions, to the more massive gumma-like *Erythema induratum* (Bazin). These eruptions may occur in various combinations. The representatives of the beginning and end of this series have been described separately elsewhere. The acneiform and nodular tuberculides vary in size from a linseed to a pea or larger, and implicate the derma superficially or more deeply. They often form about a follicle, and hence have been described as folliculites, hydradenites, acnites, folliclis, &c. The lesions are apt to be capped by a little pustular head; they pursue an indolent course and may undergo some necrosis, in any case tending to leave pits. These so-called tuberculides may evolve in one crop, or in several over a course of years. The generalised eruptions have a striking analogy with various acneiform syphilides, and among the regional forms may be specially mentioned an acneiform eruption of the face, simulating *acne necrotica*, and affecting the backs of the hands, which may possibly be confounded with chilblains in subjects with a bad circulation.

There can be no question about the intimate association of these eruptions with tuberculosis, and their occurrence in subjects either infected with the bacillus of Koch or legitimately suspected of so being. However, in some cases the subjects appear healthy. Researches for the bacilli and inoculation-experiments generally prove futile. In some cases a characteristic tubercular structure is disclosed, in other cases not, but the two conditions may be associated. In view of the insufficient proof of the tubercular nature of these eruptions, the hypotheses have been advanced (1) that the lesions may be due to a secondary infection on a tuberculous soil; (2) that they are excited by tubercular toxins; (3) that they originate from emboli of bacilli which are attenuated and come by way of the blood-current and rapidly succumb. This last theory seems to explain most of the facts observed.

PATHOLOGICAL ANATOMY.—Lupus is usually described as composed of a granulation-tissue,

divided into lobes here and there by fibrous bands, and formed of lymphoid cells lodged in the meshes of a fine reticulum. In this tissue are disseminated groups of giant-cells, surrounded by a zone of fusiform epithelioid cells. There is, however, little tendency to caseation, though a molecular necrosis may occur, ending in ulceration. Bacilli may be found, but are often very few and far between, and demand sometimes prolonged search in many sections or in portions crushed between slides. Unna points out that 'areas,' similar to the 'tubercles' of other organs, are not always well characterised in lupus, though recognisable in every case of nodular lupus. He draws a distinction between the circumscribed nodular forms tending to be encapsuled by a firm membrane of compressed healthy tissue without increase of peripheral vascularity, and the *diffuse* forms with more vascular surroundings, and therefore greater tendency to stream radially in all directions in the lymph-spaces and to form crusts by sero-fibrinous exudation. The elementary isolated nodules, according to Unna, often, but not necessarily, arise from the adventitia of a blood-vessel, and consist of a rounded compact collection of large, almost cubical, cells, with unusually large and very granular cell-bodies, and one, rarely two, large vesicular nuclei. These cells are derived mainly from the perithelium of the blood-vessels, but also from simple connective tissue-cells, and he calls them plasma-cells and the whole tissue a plasmoma. The elastic tissue disappears and the collagenous almost completely, leaving a fine reticulum. Secondly, as the nodules increase in size, a remarkable and characteristic degeneration occurs in groups of cells which undergo a 'homogeneous swelling'; and around these groups the cells with well-staining protoplasm and nuclei are compressed and arranged in rings or semilunar form. Thirdly, giant-cells are formed, corresponding to a homogenised group of cells with their ring-formed compressed surroundings. Circumscribed lupus may thus remain for years enclosing reposing bacilli, 'latent, but not dead.' The central homogenised parts have been regarded as the 'tubercle,' and the cellular surrounding as a reactive leucocytic wall, but Unna looks on the whole as a plasmoma with peripheral matrix and central degenerated part.

The diffuse plasmoma has from the commencement a non-distinctive retiform aspect, with thicker strands following the vessels, the follicles and glands, while the finer ones surround the collagenous bundles. Later on more and more collagenous and elastic bundles are liquefied in the centre of the area, and the capillaries are converted into plasmoma-tissue. Diffuse lupus consists mainly of unaltered tubercular plasmoma, which does not always go on through homogeneous swelling to giant-cell formation. Nevertheless, these changes may be slowly brought about. Between these two types all degrees and admixtures may be met with. The lupus-process, especially in the diffuse forms, is almost always accompanied by sero-fibrinous inflammation, which the bacilli are capable of exciting. The papillary layer and the granular layer of the epidermis may disappear and the remaining cells may exfoliate. Secondary hyperplastic processes, however, may occur in the epithelial structures (warty lupus), and in the collagenous tissue (hypertrophic and sclerotic lupus). The *tubercular miliary ulcer* presents the miliary nodules seen in internal organs,

with the typical caseation, breaking down, and formation of ulcers.

DIAGNOSIS.—It is important to appreciate the significance of the characteristic *soft*, yellowish-red, imbedded nodule, though the appearance may be simulated by other nodules covered with tense epithelium. Nodules may often be detected as satellites on the border of a patch, or recurring in scars. Lupus, however, is not always nodular, but often diffuse. Typical cases of *L. erythematosus* and *vulgaris* are easily distinguished, but occasionally a more than usually infiltrated patch of *L. erythematosus* or a congested area of *L. vulgaris* may render the diagnosis exceedingly difficult. Exfoliating dry patches, especially in rare cases where the patches are multiple and bilateral, may simulate psoriasis, but the infiltration will generally attract attention. Both nodules and diffuse areas may be masked by crusting, by ulceration, by the thick corneous coating of the soles, and by verrucose growth. Diffuse infiltrations have to be distinguished from chronic glanders, actinomycosis, sarcoma, leprosy, and especially blastomycotic affections. Tertiary syphilides present the most frequent difficulty, but have a firmer infiltration, pursue a less indolent course, and do not recur in scars. The age of onset is of great assistance. Serpiginous rodent cancer with its firm polished cartilaginous border or nodules should not present any difficulty.

TREATMENT.—Local treatment is always called for, and general measures sometimes. If the patient manifests signs of a feeble constitution, or impaired nutrition, or actual tuberculosis elsewhere, all possible steps should be taken to build up the strength by the administration of cod-liver oil, iron, and other medicaments, and by placing the patient in a position to benefit by good food and air.

Resolvent effects have on rare occasions been claimed for the iodides, but the most striking results are obtained from the judicious hypodermic injection of Koch's tuberculin, especially the one first discovered. Tuberculin is certainly of very great use in certain cases, and it is to be regretted that, because the remedy did not fulfil all expectations, the preparation has been so summarily discarded.

Local treatment.—The choice must depend on the form of the tuberculoderma, the extent of the tissue involved, the locality implicated, and the age, sex, and position of the patient. Combinations of methods are often desirable. It is very rarely that the tubercular infiltration can be removed by simple resolvent or parasiticide measures, such as the application of iodine or perchloride of mercury, and resort until quite recently was made to the destruction of the diseased tissues by such methods as caustics or burning, or their removal by scraping or scarification or excision. Now these methods will be in great measure supplanted by the Finsen-Light method or its modifications and the Röntgen-Ray method.

Of *caustics* in vogue we may mention arsenical paste which is said to be elective, and others such as caustic potash and acid nitrate of mercury which indifferently destroy all tissues. For ulcerated surfaces repeated applications of pure lactic acid are effective; also a ten-per-cent. pyrogallol-acid ointment applied for two or three days and repeated; and a succession of Unna's strong salicylic-acid and creosote plasters are convenient and often useful. Recurrent nodules are easily broken up and bored out by toughened pencils of silver nitrate, zinc

chloride, or by a wooden match moistened with chloride-of-zinc cream, or by the Dental Burr or Paquelin's Cautery. Hollander's method of cauterising by directing a stream of hot air on the lupus is said to be successful. The objection to caustics as to burning is the pain and the difficulty in regulating the amount of destruction. They either do too little and necessitate repeated operations, or too much and cause unnecessary disfigurement. For we must remember that the problem set is to eradicate the lupus with the least possible disfigurement, especially when the face is involved. Consequently even the character of the scar is of great importance.

Surgical treatment has been largely in vogue in late years. Free excision possesses such manifest advantages that it must continue to be largely practised. The wound can often be closed so as to leave only a linear scar, and even when skin-grafting is necessary the degree of disfigurement compares most favourably with that left by many other methods. Large areas can be thus dealt with. The scraping away of the friable diseased tissue by curettes and sharp spoons, even when followed by mild caustic applications as chloride of zinc cream, is on the whole disappointing. It generally necessitates repetitions and the keeping of the case under prolonged observation so that recurrent nodules can be detected and destroyed. Recurrences occur because the scraping only removes the massed friable tissue and not the diseased strands which wander away in the resistant tissues.

Scarification was formerly much used and often gave very beautiful results. The objection is the necessity of repeated, often a great many, weekly operations. A special instrument fitted with closely arranged parallel knives is drawn across the diseased skin to the required depth, and then again at right angles. When the surface has healed, the operation is repeated, and so on. Besnier, who thinks seriously of the danger of systemic infection from bloody operations, has devised a system of *Iguipuncture*. He has arranged a number of small instruments in the form of points and blades, which may be carried to any desired temperature by regulating a battery. Dull heat is employed. He punctures with the electro-caustic needle or scarifiers, or with the galvano-caustic knife, and repeats the operation every eight days or so. Local anaesthesia is not necessary, but the proceeding is painful.

Most of these proceedings are likely to be supplanted by the Light-Method devised by Finsen and by the use of Röntgen Rays. Finsen employs the concentrated actinic rays of light from the sun or generated by electricity by means of an elaborate and very expensive apparatus, which can only be available in certain institutions. The treatment is also a prolonged one, and not well suited to certain regions, as mucous membranes. The results unquestionably are very beautiful. It is very satisfactory to find that modifications of this apparatus are making their appearance at a cost which will enable them to be widely distributed, and of a size manageable in a consulting-room. The careful and expert application of the less expensive Röntgen Rays produces highly satisfactory results in certain cases and is likely to be effective for lupus of mucous membranes.

T. COLCOTT FOX.

SKODAIC RESONANCE.—A peculiar high-pitched resonance, found chiefly at the sterno-clavicular region of the chest, in some cases of pleural effusion and certain other conditions. The importance of this physical sign was first pointed out by Skoda of Vienna. See PHYSICAL EXAMINATION; and PLEURA, Diseases of.

SKOLIOSIS (*σκολιός*, crooked).—A synonym for lateral curvature of the spine. See SPINE, Diseases and Curvatures of.

SKULL, Diseases and Deformities of.—**SYNON.**: Fr. *Maladies du Crâne*; Ger. *Krankheiten des Schädels*.—The principal diseases and deformities of the skull will be discussed in the following order: (1) Changes of shape; (2) Variations in size; (3) Meningocele and Encephalocele; (4) Cephalæmatoma; (5) Inflammation; (6) Rickets; (7) Craniotabes; (8) Syphilis; (9) Tumours; and (10) Other diseases of the skull which form parts of general morbid conditions.

1. **Changes of Shape.**—The shape of the skull not only varies much among the different races of mankind, but in each race variations are to be found, sometimes depending upon, sometimes independent of, disease in the individual. A glance at any extensive collection of crania is sufficient to indicate how much longer some skulls are than others, in proportion to their width; how in some the vertical diameter is proportionately great, in others small; how some have wide cheekbones, some depressed noses, and others projecting jaws.

The skull is seldom perfectly symmetrical; the asymmetry being usually more marked behind than in front. This is shown not only by a coarse examination of the exterior, but by referring to the differences between the sulci and foramina on the two sides, which are so commonly met with. A familiar illustration is afforded by the fact that the nose is rarely, if ever, exactly in the mid-line of the body; but much more striking deviations from perfect symmetry may occur, as, for instance, in a case reported by Pearce Gould, in which one half of the cerebellum was absent, and there was a corresponding deficiency of the cerebellar fossa on the occipital bone. Many savage races produce abnormalities of the shape of the skull, by the application of external pressure during early infancy; and a similar result has been supposed to be consequent on the method of wrapping up the heads of children that is adopted in some parts of France. A marked asymmetry of the skull accompanies 'hemiatrophy of the face' (see FACIAL ATROPHY). There are also recorded cases of enlargement of the bones of the face and skull. A remarkable instance of this disease, or rather of the development of enormous hyperostoses, is recorded by Hutchinson. Here the hyperostoses appeared closely confined to parts which were supplied by branches of the fifth nerve. Some of these hypertrophic cases are, no doubt, examples of exostosis, others of inflammatory enlargement. An uniformly thickened skull, depending presumably, though not certainly, on the latter cause, may be either porous like cancellous bone, or dense and heavy like ivory. There are in the museum of the College of Surgeons examples of both varieties, each of which measures in many parts no less than $\frac{3}{8}$ in. in thickness; in the porous variety the sutures are usually more or less completely ossified. The

writer has seen a case in which, without apparent cause, the growth of one half of the lower jaw appeared to be arrested about the age of puberty, which gave a peculiar inequality to the face. Great deformity of the skull may result from the constrained position in which the head is held by patients suffering from torticollis.

2. Variations in Size.—The size of the skull is also subject to considerable variations in different races. It is somewhat larger, on the average, in men than in women. Among individuals also there are very great differences. Great intellects have sometimes been associated with large crania, but oftener there has been no such relationship, and not infrequently the opposite has been the case. Far greater, however, are the modifications of size which depend upon pathological conditions and defects of development. Some of these are briefly as follows:—

(a) *Microcephalic idiots.*—Among this class of idiots, which must be made to include the cretins, the skull is remarkably deficient in size. Microcephalic skulls may be caused by a too early union of the sutures, in which case the want of development of the brain may be looked upon as a result of this synostosis; or there may be a normal condition of the sutures as regards union, but both the brain and the skull may remain undeveloped. The low forehead and animal face which are characteristic of this condition give a strangely repulsive appearance to the child. The degree of idiocy depends upon the size and structure of the brain, and the development of the convolutions. See CRETINISM; and IDIOCY.

(b) *Anencephalic monsters.*—This class exhibits a more or less complete deficiency in the development of the cranial bones, as well as of the brain. See MONSTROSITIES AND MALFORMATIONS.

(c) *Hydrocephalic infants.*—The skulls of these infants are of a size proportionate to the amount of fluid which is present, and they may thus sometimes reach enormous dimensions. The forehead reaches far over the face, so that the plane of the upper wall of the orbit is continuous with that of the perpendicular part of the frontal bone. The bones become excessively thin, and are often in part replaced by membrane. The sutures gape, and the fontanelles remain open far beyond the normal time of closure, occasionally indeed beyond the period of infancy. See HYDROCEPHALUS, CHRONIC.

3. Meningocele and Encephalocele.—Closely related also to hydrocephalus are the cases of meningocele and encephalocele. It may briefly be stated here that they involve the existence of a deficiency at some point of the skull, through which the membranes of the brain, containing either cerebro-spinal fluid only, or some part of the brain itself, may protrude. The most frequent seat of this disease is the occipital bone, and the next in frequency the nasal part of the frontal bone, but tumours of this nature have been met with in other situations. It is of the highest importance to diagnose these two kinds of tumour from those developed in the bones of the skull or outside them; mistakes in diagnosis have not infrequently led to most disastrous results, as, for example, when a meningocele has simulated a polypus of the nose, and its removal has been undertaken. If patients who have suffered from meningocele or encephalocele recover—a most rare occurrence—a small hole may remain in the bone which presented the

deficiency, or the opening may be completely obliterated.

4. Cephalhæmatoma.—See p. 267.

The term ‘cephalhæmatoma’ might equally well be applied to collections of blood between the dura mater and the skull. Such effusions are probably always traumatic, and result from the rupture of a meningeal artery or vein. If serious results do not immediately follow from pressure on the brain, considerable thickening of the dura mater may be set up, accompanied by the symptoms resulting from pachymeningitis. See MENINGES, CEREBRAL, Inflammation of, Simple Traumatic.

5. Inflammatory Diseases.

(a) *Inflammation of the diploë and its veins.*—In cases of injury to the skull, whether of the nature of fracture or of simple exposure in a scalp-wound, inflammation of the diploic veins is not uncommon, if the wound be allowed to become septic. In a bone so affected, if the outer table be removed, the whole diploë and its veins are found to be filled with pus, or, on applying a trephine, the pus may be seen to exude from the divided veins. The dura mater, under such circumstances, may be inflamed, or pus may collect between it and the bone. Pyæmia, with its characteristic concomitant symptoms, is the frequent, if not the invariable, result. It is not assumed that in this affection the outer and inner tables of the skull escape, but it is only in the diploë that the pathological process is obvious to the naked eye.

The only treatment that has been suggested—trephining—does not offer any hope of curing the disease.

(b) *Chronic osteitis.*—This may affect the bones of the skull without apparent cause, but in the majority of cases it depends upon the syphilitic taint. Sometimes all the bones of the skull become thickened and enormously massive, the surface being much roughened and often worm-eaten. At other times irregular hyperostosis may be the result. Considerable thickenings of some of the cranial bones, the result of an imperfect vascular osseous deposit, are found in some infants affected with congenital syphilis. These are mostly met with about the fontanelles, especially on the frontal and parietal, and sometimes the temporal bones. The irregular hyperostoses are mostly the result of local periostitis; in fact, they are ossified nodes. Chronic osteitis is the cause of the falling-in of the bridge of the nose, or the massive condition of the same part, which gives such a characteristic appearance to a child suffering from congenital syphilis. It is also the cause of some of the exostoses of the external auditory meatus.

The treatment must be directed against the constitutional taint, if any is to be discovered.

(c) *Caries.*—Chronic osteitis can hardly be considered apart from caries, which, again, in the majority of cases, depends upon syphilis, though less commonly on the strumous diathesis. It is usually caused by the penetration of a superficial ulcer into the deeper structures, or by the separation of the periosteum resulting from periostitis. It is frequently associated with more or less chronic osteitis and necrosis. One of the most frequent seats of caries of the skull is the forehead, as a sequel of tertiary syphilitic ulceration (*corona Veneris*). Another common seat is the hard palate, which is often perforated as the disease advances. Caries may occur in the occipito-

atlantal articulation (Pott's disease), followed by a train of symptoms which will be found discussed in other parts of this work. Caries of the temporal bone, either of the petrous or mastoid portions, frequently follows *otitis media*, and is not uncommonly the intermediate stage between this disease and thrombosis of the lateral sinus, meningitis, or cerebral abscess. It usually arises by extension of disease from the middle ear to the mastoid antrum and mastoid cells.

TREATMENT.—Beyond the administration of iodide of potassium and of mercury, the cautious scraping away of the diseased bone, and the adoption of precautions for maintaining cleanliness, little or nothing can be done to relieve syphilitic caries by the surgeon; and with regard to other cases of caries of the skull, whether considered pathologically or clinically, nothing can be added which does not apply to the same disease in other parts of the body. Caries of the occipito-atlantal articulation is best treated in the early stages by the actual cautery.

(d) *Necrosis*.—Necrosis of the skull not infrequently depends upon a traumatic cause, such as a scalp-wound or a burn; but here again the syphilitic form is the most common. It may also depend upon disease of the middle ear. Simple traumatic necrosis leads to the separation of a sequestrum in the usual way; it often consists of a superficial scale of bone. Syphilitic necrosis often depends upon some form of ulceration, or upon periostitis, and may be accompanied by extensive caries and chronic osteitis. It often results from a gumma of the diploë, causing separation of the inner and outer tables. The separation of syphilitic sequestra is generally an exceedingly tedious process; and they are, moreover, often surrounded by little or no reparatory callus, so that after their removal it is no rare occurrence to find the dura mater pulsating over a large area at the bottom of the wound. At the same time, this rule is not invariable; it is common to find great thickening if necrosis of the bones of the orbit occur, which may cause permanent displacement of the eyeball. The writer has seen a large piece of the body of the sphenoid separated as a sequestrum, including the sella turcica, and removed through the nose without any apparent evil result to the patient. Tubercular necrosis sometimes involves the whole thickness of the skull, sequestra of varying size being thrown off and leaving the dura mater exposed; the outer part of the skull is usually more extensively affected than the inner.

Necrosis is not infrequently met with affecting the bones of the face. Thus a part or the whole of the upper or the lower jaw may die, and come away as a sequestrum. Necrosis of the jaws often depends on inflammation set up by carious teeth. It sometimes accompanies actinomycosis. Another cause, happily not now frequently met with, is the poisonous effect of the fumes of phosphorus in persons employed in the manufacture of this substance, and in that of lucifer matches (*see PHOSPHORUS, Poisoning by*). The same remarks apply to the abuse of mercury. But, besides these more special causes, necrosis of the bones of the face may depend upon those more general states which are supposed to stand to necrosis of other bones in the relation of cause and effect; such as fevers and the like. The amount of thickening round a necrosed upper jaw has not infrequently led

to its removal in mistake for a tumour; it is, therefore, of the highest importance to examine all swellings in this region with great care.

TREATMENT.—If the membranes be left exposed, some protection must be provided for the cranial contents; in other respects the treatment of necrosis of the skull must be conducted on general principles. Sequestra in the mastoid process or around the tympanum should be carefully dealt with, on account of the danger of setting up meningitis or injury of the internal carotid or lateral sinus which any surgical interference involves. In dealing with necrosis of one half of the lower jaw it must be remembered that, unless sufficient callus have been thrown out before the removal of the sequestrum, the other half will lose its support and assume an altogether unnatural and almost useless position, being drawn to the opposite side and leaving the patient in a condition in which he can hope for but little relief from surgery.

(e) *Periostitis*.—Periostitis of the skull has been already referred to. It may depend upon syphilis or tuberculosis—most commonly the former; and give rise to what are known as *nodes*. The inflammatory subperiosteal effusion may be fluid or solid (*soft and hard nodes*); and it may undergo true or spurious suppuration or ossification, or may be completely absorbed. The most common but by no means the invariable position for cranial nodes is the frontal bone. As in the case of periostitis elsewhere, nodes are the seat of characteristic nocturnal pain, which is extremely distressing and exhausting to the patient.

TREATMENT.—The treatment in any case is by the administration of iodide of potassium; the effect of which is most marked, however, in syphilitic cases, the pain being usually removed in two or three days. If suppuration occur, incision may be required.

6. *Rickets*.—In a rickety infant the skull looks large; though it may be questioned whether this appearance does not depend on a deficient development of the bones of the face. The frontal and parietal eminences appear too prominent; the fontanelles remain patent much longer than in a healthy infant; and in some cases the anterior fontanelle may be unclosed as late as the fourth or even sixth year. The skulls of rickety children have a peculiarly massive feel; they are sometimes long in proportion to their width, conforming to the shape known as dolichocephalic. The head of an adult who has been the subject of rickets in his childhood has often a very characteristic appearance: an apparently large square skull, with a prominent forehead towering above a diminutive and pinched-up face, giving to the individual a decidedly intellectual aspect.

Craniotabes occurs occasionally in rickety skulls, but, as will be afterwards shown, we do not yet know how far, if at all, it depends upon the constitutional condition.

7. *Craniotabes*.—By this term is meant the occurrence of spots of remarkable thinness in the skull, such that an indentation may be produced by the pressure of the finger. True craniotabes, as opposed to the gelatiniform degeneration of the outer table (Parrot), attacks the inner aspect of the skull. For its production an undue softness of the bone appears to be necessary, together with the occurrence of pressure, either from within or from without. It is rarely found congenitally, and it then

affects the anterior part of the skull. It is common in syphilitic infants under one year of age, and is then usually met with in the posterior parts of the parietal bones. These positions, it will be noticed, are those most subjected to pressure under the two conditions mentioned. Craniotabes has been, by some observers, associated with rickets, but the relation of the one to the other is at present doubtful. It disappears as age advances, and requires no special treatment.

8. Syphilitic Affections.—From the foregoing observations it will be seen that syphilis, congenital or acquired, has much to account for among diseases of the skull. It may cause periostitis, with consequent nodes; chronic osteitis, with consequent hypertrophy, local or general; caries; necrosis; and craniotabes. As a general rule, it may be stated that syphilitic affections of bone are among the later manifestations of this disease. The inflammatory forms are usually accompanied by severe nocturnal pains, and they may be expected in most cases to yield to the administration of iodide of potassium.

9. Tumours.—It is necessary to refer in the briefest possible way to the tumours of the skull. Primary growths may spring from the diploë, or from the inner and outer tables of the cranial bones. Perhaps the most common are exostoses and some of the various kinds of sarcoma, either of which may reach an enormous size. The former may assume various characters (*see* TUMOURS). These, in certain cases, admit of removal by the surgeon. In connection with the bones of the face, tumours of the antrum or of the upper jaw, of various kinds, and tumours of the lower jaw, are not uncommon. In the latter position the various forms of epulis—myeloid, fibrous or malignant—and cystic tumours, are frequently met with. Exostoses often grow from the jaws and the orbit, and in connection with the former the different kinds of odontoma must be mentioned. Secondary tumours of all kinds may affect the skull; thus more than one instance is on record of a pulsating growth, occurring secondarily to a similar growth in the thyroid gland; and scirrhus, following cancer of the breast, is by no means unknown. Of these secondary affections the commonest are those which affect the skull by the direct extension of tumours from within or without; for example, the epitheliomata of the scalp or mouth, or rodent ulcer of the face.

10. Miscellaneous.—Certain characteristic modifications of the shape and size of the skull are met with in such diseases as acromegaly (*see* ACROMEGALY), osteitis deformans (*see* BONE, Diseases of), and leontiasis ossea (*see* BONE, Diseases of). As they have been described in the articles referred to, no further account of them is necessary.

R. J. GODLEE.

SLEEP, Disorders of.—SYNON.: Fr. *Troubles du Sommeil*; Ger. *Störungen des Schlafes*.—A proper amount and kind of sleep is needful in order that the body may be maintained in a state of health; but the actual amount of sleep taken and necessary for persons in health varies, within wide limits, according to age, the soundness of the sleep itself, and individual idiosyncrasy.

Age is a very important modifying factor. Thus an infant may sleep for twenty hours out of the twenty-four, and young children up to the age of ten commonly sleep for fourteen or at least twelve

hours. In children from ten to fifteen years old the duration of sleep usually varies between twelve and ten hours. In persons from fifteen to twenty-five the period should not sink below eight hours; from the latter age on to fifty it may fall to seven hours; and after this age about the same amount of sleep is required by the majority of persons, though some find six hours sufficient, and a few can (without apparent injury) take habitually even as little as five hours' sleep. The instances in which a duration of sleep habitually less than this is needed are altogether rare and exceptional.

Soundness of sleep, too, is subject to much individual variation. In childhood and in early life, sleep is commonly more profound than it is in adults, and much sounder than in old age. But, over and above these variations incident to age, there are individual differences; some persons are naturally 'light' and others 'heavy' sleepers. As a rule, those who can do with a small amount of sleep belong to the latter category. And similarly in regard to amount there are individual differences: some persons, as above indicated, are able to do with a comparatively small amount, while others seem to require to sleep decidedly beyond the average periods above stated.

The disorders of sleep—that is, the variations outside the above limits—belong to three principal categories, in the first of which may be ranged all those cases where sleep is *excessive* in amount; in the second those in which it is *defective* in soundness or in amount; and in the third those in which it is *unnatural* in character.

I. Amount of Sleep Excessive.—This occurs commonly in more or less demented persons or in idiots, whose brain-activity is below the usual level. Such persons, when their natural wants are satisfied, are apt, like many of the lower animals, to sleep away a large portion of their time.

But some individuals of notable intellectual power may occasionally, even in a state of health, after greatly prolonged labours with previous deprivation of rest, continue to sleep soundly for twenty-four or even thirty-six hours.

In many brain-affections, and in some cases of blood-poisoning, a condition of unnatural sleep bordering upon stupor may be present for many days. Obscure cases in which sleep is prolonged for weeks, or even months, are occasionally met with in this country. This rare condition only supervenes in persons of an obviously 'nervous' temperament, and the state itself seems generally to be a kind of trance allied to catalepsy. *See* also NEGRO-LETHARGY; and TRANCE.

II. Amount of Sleep Defective.—Under this head we have to do with two kinds of failure—a defect of quality (*disturbed or restless sleep*); and a defect in quantity (*wakefulness, insomnia, pervigilium*). These two defects often co-exist, though in many cases we may have the former condition existing alone.

(a) *Disturbed or restless sleep.*—This is a very common complaint, apt to occur in persons of all ages, and under the influence of many different causes, some of the most frequent of which are these: Indigestible food, or food of excessive or unaccustomed quantity, taken not long before going to bed; painful conditions of any kind, whether due to mere neuralgia or to serious organic disease of any organ; discomfort induced by undue cold or excessive heat; mental excitement or worry;

chronic alcoholism; the menopause; prolonged overwork (mental); over-fatigue (bodily); febrile conditions; inflammations; gouty states of the system; imperfect action of the liver and various forms of dyspepsia; excessive hæmorrhages; acute and chronic illnesses of various kinds, such as delirium tremens, bronchitis, polyuria, and different forms of heart-disease; the state of convalescence from many acute diseases; the taking of tea or coffee, either too strong or too late at night. Lastly, sleeping in a novel or uneasy condition, or in the midst of unaccustomed noises, may also be mentioned as not infrequent causes. Under any of these various conditions sleep may be fitful and disturbed, the persons often starting or turning about uneasily, dreaming much, and from time to time waking under the influence of dreams of a distressing or oppressive character. In one of the most extreme of the latter conditions, especially when it has been evoked by indigestible food, the state known as *nightmare* is induced. See NIGHTMARE.

TREATMENT.—The treatment of disturbed sleep must of course vary widely according to the nature of the influences under which it has arisen. These may at times be easily corrected, but in other cases, where the disturbed rest is dependent upon pain difficult to annul, or upon some acute or chronic disease, it may be impossible or extremely difficult to ensure sound sleep, notwithstanding the best-directed efforts to correct or neutralise the disturbing causes in operation. It may then be necessary to have recourse to some of the measures recommended under the next heading.

(b) *Insomnia or wakefulness.*—Under this head we may have either complete or partial insomnia. The condition is complete when the person gets no sleep at all for night after night, as in acute mania, delirium tremens, in those suffering from some very severe pain, or in persons under the influence of profound grief or mental anxiety. On the other hand, we may have partial insomnia of different kinds. In the one set of cases the persons who suffer from it may lie awake for long periods (one to several hours) before being able to get to sleep at all, and then sleep may be more or less sound and continuous till morning. In other cases, patients do not experience so much difficulty in getting to sleep, though after they have slept for one, two, or more hours they awake and cannot again fall asleep, or only into sleep of a fitful or restless nature; they may, however, lie awake in a state of mental depression, or even actually tortured by gloomy or horrible forebodings.

Various cases are on record in which absolute insomnia has lasted not only for days but even for weeks, interrupted only by mere snatches of sleep during brief intervals.

In this whole class of cases, however, the sufferers themselves are apt to form exaggerated estimates of the amount of their wakefulness, and to become more or less hypochondriacal upon the subject.

TREATMENT.—In many of these cases the art of the physician is very severely taxed. Whenever it is possible, insomnia should be corrected by a studious attention, to the general health and habits of the patient, and by endeavouring to ensure the presence, as far as possible, of the physiological conditions which favour sleep. Mental repose, bodily comfort, a sufficient degree of warmth, a

certain amount of fatigue, combined with perfect quietude—one or more of these conditions will be found in different cases either important aids or essentials for the production of sleep. To ensure the first of these conditions, it may be needful to prohibit all study for some hours before retiring to rest. An evening walk, so as to induce a certain amount of bodily fatigue, is, where it can be had recourse to, often beneficial; while in other cases a warm bath at bedtime, followed by friction of the skin, may greatly facilitate sleep. Sometimes a mere hot foot-bath, with or without mustard, will prove beneficial. A cup of warm beef-tea, gruel, or some weak stimulant (such as hot whisky and water or a glass of stout) just before going to bed may also have a salutary influence, and the gruel may be repeated, or taken preferentially, during the night. Monotonous sensorial impressions (sounds or gentle frictions); or a monotonous dwelling of the mind upon certain uninteresting imaginary sights or verbal repetitions are, again, sometimes found to act as provocatives of sleep, though in other cases they completely fail.

In debilitated or neurasthenic conditions of the system, a patient's ability to sleep may often be greatly improved by the administration of such nerve tonics as arsenic and strychnine in combination with digitalis or *sumbul*; and some patients suffering from worry or overwork have derived great benefit in the way of improved sleep by taking spirits of turpentine (m 30) in capsules at bedtime.

Where the preceding measures are unavailing, recourse must be had to hypnotics and sedatives, such as bromide of potassium, chloral hydrate, opium in one or other of its forms, morphine, Indian hemp, amorphous hyoscyamine, hyoscyne, &c., in doses appropriate to the age and condition of the patient. In the more urgent cases the doses of such hypnotics may have to be repeated till sleep is procured; but in many of these urgent conditions the sedative influence of packing in the wet sheet must not be forgotten. In all cases of insomnia apart from drug-treatment it is important, till a better habit is established, that the patient should abstain from tea or coffee, or take them only weak and in small quantity.

Several other most useful remedies of this class have been in use during recent years, the most important of which are sulphonal, trional, paraldehyde, chloralamid, chloralose, and chloretone. Of these, sulphonal (gr. 15–30) often acts well, and produces less depressing effects than chloral hydrate. It has, however, of late years been to a considerable extent superseded by trional (given in the same doses), because of its more rapid action and more speedy elimination from the system. Paraldehyde (m 40–120) is a very safe hypnotic to give where such a drug is required in patients suffering from heart- or lung-disease, but the great objection to it is its very nauseous taste and odour. Chloralamid (gr. 20–45), given in a slightly acidulated solution, is free from these objections, and is equally safe, though perhaps less reliable. Chloralose may be given in cachets or tabloids (gr. 6–8), as also may chloretone, though in larger doses (gr. 8–18). The writer has found them both safe and reliable hypnotics, capable, in suitable cases, of producing six or seven hours of sound sleep. For occasional use in time of need they are undoubtedly of great value. Such remedies, however, ought never to

be taken except under the orders and directions of a medical man, who alone would be able to judge as to the most suitable drug, the proper dose, and the mode of its administration. With some of these drugs, a dose on alternate nights is found to be sufficient; and in all cases it is of the greatest importance to obtain the desired result with the minimum quantity that will prove effective. Again, wherever hypnotics are had recourse to, it is of great importance to see that their use is not continued after the need for them has passed. It not infrequently happens, for instance, that much of a patient's insomnia may be due to a bad habit of the system, which yields after it has been broken in upon by the use of hypnotics for a short period. Abrupt discontinuance is generally most inadvisable; there should rather be a gradual diminution of the dose, with or without the knowledge of the patient. See HYPNOTICS; and SULPHONALISM.

III. Sleep Unnatural in Character.—Under this head we have to do with various unnatural conditions, in which the abeyance of the cerebral functions that occurs during sleep is more partial than that which normally exists. In disturbed sleep the physiological condition pertaining to and provocative of sleep is generally less profound than it should be, just as in other cases of unusually deep sleep (akin to stupor) such a condition is generally more profound than natural. In the cases to which we now refer, however, the brain-condition productive of sleep is partial in its area: portions of the brain that are usually involved in the physiological condition peculiar to sleep remain exempt, so that the sleeper exhibits powers which sleep usually annuls. Hence we may have *somniloquy* or *sleep-talking*; *somnambulism* or *sleep-walking*, or what are known as *night-terrors*.

In those who exhibit the first of these phenomena, dream-thoughts are capable of evoking correlative acts of speech, and such persons will sometimes allow a listener to hold a sort of conversation with them, of which in the waking state they recollect nothing. This dream-conversation may be more or less coherent. Dreams themselves, too, vary much in their coherency in different individuals. In some persons, whose sleep is to that extent unnatural, powers are displayed during this condition which even surpass those of the waking state. Mathematical problems have been solved during such sleep; poems and music have been composed and written out, which have altogether surprised the same person when awake. In all these states we have to do with a morbid condition of sleep, partial in its area, in which there is the further peculiarity that certain faculties are in a condition of exalted activity. The alliances here are intimate with the conditions that are now studied under the name of 'hypnotism,' but which were formerly included under the term 'animal magnetism.' See MAGNETISM, ANIMAL.

Many of the above remarks apply to somnambulism also. Here the morbid sleeper possesses an unwonted power of calling his muscles generally into activity in response to his dream-thoughts. Sight in relation to the dream may be good, though unrelated visual impressions are not taken cognisance of. Muscular sense-impressions also are freely acted upon; but the sleep-walker may be quite deaf to all ordinary auditory impressions.

TREATMENT.—These are to be regarded as distinctly morbid conditions, and the persons manifesting them may often be cured by attention to the

general health, and the use of remedies calculated to give tone to, and allay the irritability of, the nervous system. A line of treatment, in fact, not very dissimilar from that to which one would have resort in convulsions or epilepsy will often suffice to cure these minor manifestations of nervous disorder.

Finally, sleep may be disturbed by certain phenomena occurring to the person while he is actually in this condition, which, though scarcely to be spoken of as disorders of sleep, ought at least to be mentioned in this article. One of minor significance is snoring, which at times may be so loud as to awaken the sleeper; but another of far greater significance is the occurrence of convulsive or epileptic attacks, which in some patients occur only during sleep.

H. CHARLTON BASTIAN.

SLEEPING SICKNESS.—See NEGRO-LETHARGY.

SLOUGH (Sax. *slog*, a foul hole or hollow).—A mass of dead material occurring among living tissues. A slough may be in the form of a large mass; or in shreds, as in ulcers and septic wounds. See GANGRENE; ULCER and ULCERATION.

SMALL-POX.—SYNON.: *Variola*; Fr. *La petite vérole*; Ger. *Blattern*.

ÆTIOLOGY.—Except in the case of those protected by vaccination, immunity is rare. One attack does not invariably protect for life. The writer has observed 92 cases of recurrent small-pox out of some 12,000. (See IMMUNITY, p. 722.) Dark-coloured races seem especially liable to the disease.

Seasonal prevalence. See PERIODICITY IN DISEASE, p. 1190, fig. 2.

Age.—No age exempts from attack, apart from protection by recent vaccination. The younger the child the greater the risk of a fatal issue. The fetus is sometimes attacked *in utero*. Some infants resist vaccination and exposure to small-pox, and some are born bearing vesicles or scars, showing that they have suffered from the disease during uterine life.

SEX.—In early life sex appears to cause no difference in susceptibility. After puberty more males than females are attacked.

NATURE OF VIRUS.—The *contagium* of small-pox has not, up to the present, been isolated. Practically the only micro-organisms discovered are such as belong to suppurative processes, but thus far the bacteriological study is very incomplete. The virus of small-pox exists in the secretions and excretions, and is given off freely from the lungs and the skin. It is said to be most active at the period when the clear contents of the pocks begin to turn cloudy, but whether the disease can be communicated before the eruption appears is doubtful; opinion inclines to the affirmative. The disease may be communicated after death. A fine desquamation occurs during convalescence and may be a source of infection.

MORBID ANATOMY.—The skin and those portions of mucous membrane that are in direct contact with the external air, viz. the lining of the mouth, pharynx and trachea, are sometimes found softened, congested, and covered with inflammatory exudation. Such internal parts as may have been inflamed exhibit evidence of such change. The pleural cavity, for instance, may be filled with serum, but as a rule the viscera are found free from

every appearance of special disease. Among very many *post-mortem* examinations made, in no case had the disease extended to the intestinal mucous membrane, but pustules were found in the nose and mouth, on the tongue, tonsils, soft palate and pharynx, also in the trachea and bronchi, the upper part of the œsophagus, and in the lowest part of the rectum, in the vagina and on the vulva; they are never found in the bladder, and only in the urethra near the meatus. The spleen is enlarged and soft, and of a lighter colour than natural. Renal changes are not common. In the hæmorrhagic type there may be found hæmorrhages of various sizes scattered over the serous covering of different viscera, in the connective tissue, into the muscles, and beneath the capsule of the kidneys; also throughout all the mucous membranes, from mouth to rectum, and in the uterus and Fallopian tubes, the ovary and testicles, the pelvis and calices of the kidneys, and the ureters. In the later stages of the disease the corpuscles have a tendency to form irregular clumps instead of *rouleaux*.

SYMPTOMS.—There are five well-marked stages of small-pox: (1) Incubative; (2) Prodromal; (3) Eruptive; (4) Suppurative; (5) Desiccative.

1. *Incubative.*—The incubation-period occasionally varies a little, but is almost invariably twelve days. See INCUBATION, and p. 727.

2. *The Prodromal* or initial stage is ushered in with frontal headache and sickness, which are severe, particularly in young children. Backache, which in the case of older patients awakes suspicion, is absent in children. The position of the pain is said to be sometimes indicative of the type of case: thus, if in the lumbar region, the attack will be very severe; in the dorsal less so; while if it be located entirely in the cervical, a very mild form may be anticipated. The combination of headache, sickness and backache is so significant that if these are alone present, precautionary measures may with advantage be adopted. The temperature rises rapidly and may reach 104° F. or 106° F. by the end of twenty-four hours, the pulse is frequent, and the tongue is creamy or dry. The premature appearance of the menses is also a frequent event, said to be due to a direct action of the variolar poison upon the genital functions. Not infrequently there is coma, preceded by convulsions in children, and in adults delirium. Albuminuria may be present. The chlorides in the urine are diminished, but the other solid constituents remain proportionally undisturbed.

Prodromal rashes.—In this stage peculiar initial rashes are by no means uncommon, and from a prognostic and diagnostic point of view very important. According to McNeill they may be divided into (1) the *Erythematous*; (2) the *Petechial*; and (3) the *Petechio-erythematous rashes*.

(1) *The Erythematous rash*, when fully developed, is of a bright red colour, which disappears momentarily on pressure. In some cases the redness is uniform, like erysipelas; in others it is punctate like scarlet fever; and in others again it occurs in patches and crescents like measles. The sites chosen by this rash when any part of the body is specially affected are the extensor surfaces of the arms and legs. In some cases the redness is smooth and uniform for some inches round the arms above the wrists, and the legs above the ankles, although mottled and scanty elsewhere. In other cases it covers the whole body.

(2) *The Petechial rash* is of a brownish-red colour when discrete. If thickly set it has a blue or purple tinge. The site chosen by this rash is very characteristic. It almost invariably selects the lower part of the abdomen and the groins to about an inch or two below Poupart's ligament. The lower margin runs from a point near the pubes in front of the thighs in an oblique line upwards, outwards and backwards, parallel to Poupart's ligament, to about an inch above the great trochanter, and then upwards to near the middle of the crest of the ilium. In rare cases the rash extends higher in the abdomen, and sometimes covers the flanks up to the axillæ, and the chest to the edge of the pectoral muscles. As a rule it does not reach further back than the edge of the latissimus dorsi. In very exceptional cases is any part of the body affected by this rash while the groin remains free.

(3) *The Petechio-erythematous rash* in the height of its development is redder than the petechial. This redness is dark, with a tinge of purple, and sometimes is almost blue in the groins. The erythematous rash covers a wider area than the petechial, and the colour varies, according as the erythema is more or less mixed with petechiæ. The erythema fades, but the petechiæ are persistent on pressure. The site chosen by this rash is not so well defined as in the case of the petechial. The parts in which the petechiæ are seen in the redness are as a rule similar to those parts affected by the petechial rash. Sometimes, however, there are no petechiæ in the groin, while they are present on the sides of the chest, on the inner surfaces of the upper arms, and in the axillæ. The rash reaches lower down on the thighs and further up on the chest than the petechial rash. It sometimes surrounds the body, leaving the back above the spines of the scapulæ free and the buttocks below the line of the trochanters.

The duration of the initial rashes varies. The erythematous rash remains bright but for a short time; it simply fades to light red and disappears without leaving any pigmentation. The petechial and petechio-erythematous rashes begin to fade slowly one or two days after their first appearance, leaving considerable pigmentation and mottling of the skin, which disappears after periods varying from three to twenty days in different cases.

The prognostic value of the Initial rashes may be summarised as follows:—

(i) If the patient has a purely erythematous rash he is more likely to have a slight attack of small-pox than if he have any other initial rash or none at all.

(ii) If the rash is petechial the case is about twice as likely to be a confluent or semi-confluent attack, but the ultimate result is likely to be almost as favourable as in general cases.

(iii) If the rash is petechio-erythematous the case is almost three times more likely to be severe than in general cases, and the termination is more likely to be unfavourable. The darker the redness, and the more persistent it is on pressure, the more likely is the case to be severe. If a purely petechial rash is on one part of the body and a purely erythematous rash on another, the prognosis is more favourable. In forming an opinion, other circumstances besides the initial rash must be considered and weighed. These rashes appear with great irregularity—some epidemics affording numerous instances, others very few; hence the scanty reference to them in the records of older epidemics.

3. *The Eruptive Stage*.—The peculiar eruption invariably appears after the completion of forty-eight hours of initial fever, and it should be remembered that the statements made by patients as to its accession put it, as a rule, later than it really occurs, the spots being so minute that they often at first escape observation. The forehead and hairy scalp are by far the most common sites for the first appearance of the rash. After these come, in order, the backs of the wrists, the trunk and arms, and finally the legs. With the advent of the rash comes a marked fall of temperature, the normal not infrequently being reached by the time the exanthem is fully out. At the same time the mucous membranes become affected, the eruption appearing in the mouth, on the pharynx, cheeks, lips, tonsils, œsophagus, vulva, vagina, anus, and extreme end of the urethral canal. The papules are at first slightly red, so minute that they may be easily overlooked, but yet conveying a distinct sense of irregularity to the finger as it is passed over the surface, as though millet-seed or small shot were embedded in the skin; if the patient, however, be cold, with contracted facial capillaries, the eruption may not be so easily detected. More slowly, usually some hours later, after the primary papules have appeared on the face, those on the legs develop, and last of all on the feet. The full efflorescence is reached usually in from 48 to 72 hours, and portions of skin upon which mechanical irritation or pressure has existed as from bands of petticoats, garters, collars) will reveal very closely congregated papules, strikingly in contrast with other parts; further, although the eruption may be considerable on the back, breast, arms, and trunk, it is always very sparse on the abdomen. On the third day of the rash, vesicles are formed filled with clear fluid; these enlarge until the fifth day, by which time they have attained the size of a pea, and are semiglobular and umbilicated, in a greater or less number.

4. *The Suppurative Stage*.—About the eighth or ninth day of the eruption, the contents of the vesicles become purulent, the areola seen at first surrounding the pocks is more extended and of a deeper colour, the umbilication is lost and the pustules have a flattened top, appearing of a greyish-yellow colour. These changes are first noticed on the face and follow the order in which the eruption appeared. If the eruption is discrete, the temperature of maturation does not as a rule rise, except for a few hours, and convalescence is not therefore retarded. If there be a plentiful crop of pustules there is generally a considerable degree of swelling of the face which also extends to the scalp, often involving the eyelids in such a manner that the eyes are completely closed. This subsides about the tenth or twelfth day, by which time the hands and feet are seen to have participated. Some pain in the throat with dysphagia is common when the swelling of the face arises, and at the same period an amount of viscid saliva dribbles from the mouth.

5. *The Desiccative Stage* begins about the eleventh or twelfth day, sometimes earlier; the facial pustules first commence to dry up and rapidly proceed to form crusts or scabs, which fall off after a variable time. Some of the pustules are always liable to be prematurely broken by accident or the patient's interference, and these will consequently crust over earlier than they otherwise would have done. So that in fixing the period of desiccation, those pustules only must be regarded of which the natural

progress has not been interfered with. Red spots are always left which are somewhat elevated as long as the skin remains swollen, but after a time either disappear, without leaving a trace, or else change to depressions varying with the amount of damage done to the true skin by ulceration. Those with long-adherent crusts invariably leave deep cicatrices with inverted edges, and a punctated seamed base; such are particularly observable about the *alae nasi*.

VARIETIES.—A slight innovation on the usual nomenclature of the various kinds of the disease is ventured upon, as it is difficult to classify one or two under the usual divisions employed.

They may be described under eight headings: (1) Discrete; (2) Confluent; (3) Semi-confluent; (4) Hæmorrhagic or Malignant; (5) Hæmorrhagic pustular; (6) Modified (Varioloid); (7) Variola Aquosa; (8) Corymbosæ.

1. The *Discrete* form may be taken as the standard for descriptive purposes, as the number of such cases forms by far the larger proportion of those attacked and has already been considered in the foregoing paragraph.

2. The *Confluent* form commences like the discrete, but the initial symptoms are invariably more grave and pronounced; indeed, it may be said that the intensity of the symptoms in the prodromal stage is in direct proportion to the subsequent eruption, and that high temperature and severe disturbances usually (but not invariably) indicate confluent small-pox. The eruption appears earlier and spreads more rapidly than in the milder forms. The papules are always discrete at first, but subsequently coalesce; but however numerous they may be on the face, hands, and feet, they will always, as in the discrete form, be found less scanty on the extremities, and very sparse indeed on the abdomen. The temperature falls but little on the advent of the rash, remaining persistently high from the onset; occasionally, however, there is so marked a reduction of fever that the patient fancies he is progressing satisfactorily. By the eighth or ninth day, the pocks having attained their maximum pustular size, and the bluntness of their summits giving way to a hemispherical shape, there is a recrudescence of temperature, which perhaps reaches 104° or 105° . The pulse is very rapid, there is marked throbbing of the carotids, and great restlessness and insomnia are present. About this time the peculiar characteristic constant greasy odour becomes accentuated, and violent delirium appears—demanding constant nursing. The writer has seen this derangement assume both a grave homicidal and suicidal tendency. Salivation in adults is often profuse, the cervical and inguinal glands enlarge, and great tumefaction of the head and face appears—so pronounced is this general swelling that the patient cannot see, hear, or smell. The presence of the eruption on the mucous membrane renders swallowing, breathing, and micturition almost impossible, and makes up a sum total of misery perhaps unequalled by any other disease. In fatal cases by the tenth or eleventh day a typhoid condition ensues. In other instances the maturation of the pustules goes on for a day or two satisfactorily, then suddenly the pulse falters, the pustules collapse, the extremities become cold, and the patient dies. A fatal termination occurs as a rule on the eleventh day in vaccinated subjects, on the ninth in the unvaccinated. In favourable cases, about the eleventh day the secondary fever abates.

Between the Confluent and the Discrete forms there is necessarily constructed a third class, or :—

3. *The Semi-confluent* cases, in which some of the pustules stand distinct, others coalesce, the confluent patches being more or less numerous. There is in consequence a very appreciable gradation of risk to life, as when the confluent patches are many and extensive the form may be divided by a fine line only from that of the true confluent; indeed, this class furnishes a large mortality—in some epidemics reaching as high as 16 or 18 per cent.

4. *Hæmorrhagic* or *Malignant* form (Synon. : *Variola Nigra*; *Variola Hæmorrhagica*; *Black Small-pox*; *Purpura Variolosa*).—The initial symptoms are very pronounced; there is great pain in the lumbar and præcordial regions, and almost constant vomiting; the rash appears after forty-eight hours of high fever, but it is only on careful inspection that a few scattered papules are discovered in the usual situations, while the intervening skin is of a dark brick-dust colour. About the third or fourth day the greater part of the body is ecchymotic, emitting an almost intolerable odour, in which the breath participates. The face is not only somewhat swollen, but dark purplish in colour, the conjunctivæ are swollen and black, hæmorrhage having taken place beneath them, and the corneæ appear as if sunk in dark-red pits, from which blood often trickles down the cheeks. Hæmorrhage also occurs from the mucous surfaces, from the bowels, stomach, lungs, kidneys, and bladder, and in women from the vagina—occasionally from all combined. If the patient lives until the third or fourth day, a few vesicles may be seen; these are, however, invariably scanty and abortive, and to the inexperienced add little to aid in the diagnosis of the disease. The temperature is very variable, sometimes high, sometimes sub-normal, and the mental faculties are invariably painfully lucid up to the moment of dissolution. Young and strong persons, with dark eyes, hair, and complexion, are more frequently the victims of hæmorrhagic small-pox.

5. *Hæmorrhagic pustular* form.—This form of the disease, from which recovery often takes place, presents certain common features and peculiarities. It is so named from the fact that the contents of the pustules become black-centred, caused by a complete stasis of the blood in a cluster of engorged capillaries. The hæmorrhage occurs during the change from vesicle to pustule, the eruption then aborting and convalescence being soon established. In this mild form there is rarely hæmorrhage from mucous surfaces and the constitutional symptoms are not more marked than in an ordinary case of moderate severity. The blood-containing pocks are also formed on the lower extremities from mechanical causes, as by running about when in a state of delirium, or after much handling by attendants. When with this condition hæmorrhage into the skin, beneath the vesicles or pustules and from the mucous membranes, occurs, the cases prove fatal in the great majority of instances, death generally taking place as soon as the pustular stage is reached or even sooner.

6. *Modified small-pox* is that which occurs subsequent to a more or less successful vaccination, and is seen in the bulk of patients admitted into small-pox hospitals. This was shown by the writer in an analysis of 10,403 cases, of which 6,939 were beyond question vaccinated in some form or the other. The initial symptoms of such

may be severe, but are not usually so; and the influence of the vaccination being still retained, renders the course of the disorder less dangerous and diminishes the intensity of the primary fever. When the eruption appears the difference between variola and varioloid becomes apparent: the rash is sometimes exceedingly scanty; owing, however, to the bad quality of the primary vaccination or to the exhaustion of its protective power, cases are seen in which the eruption is very copious and exceptionally confluent. These cases seldom, however, in early life fail to follow a very different course from that of true variola. On the fifth day the papules become vesicles, which in a day or two dry up, while the almost complete absence of secondary fever is both its constitutional peculiarity and the main source of the patient's safety.

7. *Variola Aquosa* is a very fatal form in which the eruption never becomes pustular. It is characterised by large, watery vesicles, tending to form patches; the skin of the hands and knees looks as if it had been macerated for a long time in water, the epidermis completely separating from the cutis on pressure. The eruption in such cases is scarcely elevated above the surface of the skin of the face and body; it is flattened and without definition of form. The temperature is generally low, there is marked depression of the vital powers from an early stage, the extremities are generally cold and the patient very uneasy and restless.

8. *Corymbosæ* small-pox is a very rare form of the disease, the peculiarity being that the eruption is arranged in clusters like berries. The mortality it causes is quite out of proportion to the amount of rash. Vaccinated persons are more frequently attacked than the unvaccinated, and there is a great tendency to the formation of symmetrical patches. Some capital has been made by anti-vaccinators in reference to this class of case by pointing out that in Marson's patients, as in others, they occurred more frequently in the vaccinated. This was owing only to the small number of this curious form of small-pox being observed by him and others.

DIAGNOSIS.—In a well-marked instance the diagnosis is very easy. In the less severe cases reliance must be placed, not on a single sign or symptom, but on the collection and comparison of all. During the initial stage the difficulty may be great, yet an error at this stage may be followed by disastrous results, not only to the patient but to others, to whom the disease may be inadvertently communicated. A character peculiar to small-pox is the suddenness of the invasion, accompanied by a peculiar and generally persistent state of præcordial trouble, and one of the almost constant precursors is backache; these, combined with frontal headache, high temperature, rapid pulse, and a general feeling of severe illness, are symptoms of marked importance. Still, they are not sufficiently definite to warrant a reliable diagnosis of small-pox until the appearance of the characteristic eruption. Measles is the disease with which variola is most frequently confounded, and the appearance of the skin during the early stage of eruption is certainly very like it, but the maculæ in the former are developed simultaneously on the face, trunk, and extremities, and are accompanied by running at the eyes and nose, and a harsh, dry cough; the exanthem also appears on the *fourth* day, while that of small-pox follows on the *third* day, or after forty-eight hours' illness.

In measles, again, the papules are not so elevated, and therefore are not so distinctly felt as in small-pox. Very important, too, is the degree of fever in variola in the initial stage; although it may at first reach 105° or 106° F. the temperature falls soon after the eruption appears, while in measles it continues the same or even rises.

Varicella often leads to difficulty in diagnosis. See CHICKEN-POX, p. 279.

In the form of syphilitic eruption which looks like small-pox, the rash is scattered symmetrically over the face, limbs, and trunk; it may be discrete or confluent; the papules are hard and shotty at first; they have depressed centres; they form adherent scabs, and they leave scars. The eruption differs from variola in the slowness of its evolution, in its long persistence and its very slow decline. It is only in its early stages that mistakes in diagnosis are likely to occur.

In *Glanders* the initial symptoms somewhat resemble small-pox, and the rash consists of hard papules, which, however, speedily become ulcers (see p. 579). Like *varicella*, this rash appears in successive crops; and there is no defined period (forty-eight hours) of prodromal fever as in variola. Certain *drug-rashes*—*copaiba* and iodide of potassium—are like small-pox, while vaccination with calf-lymph may develop an erythematous rash known as *Roseola vaccinosa* appearing from the 8th–12th day, which has been mistaken for the eruption of variola.

Erysipelas simulates small-pox by causing redness, swelling, and fever, but all the other symptoms are wanting; and other diseases associated with pain in the back have given rise to errors in diagnosis. See LUMBAGO.

PROGNOSIS.—The factor of the highest importance is the presence or absence of one or more typical vaccine-scars. Unmodified small-pox is an exceedingly fatal disease (see MORTALITY, p. 1028). The disease is more fatal at the beginning and during the height of an epidemic than at its close; periods of the year also appear to exercise some influence on the mortality; therefore, in the consideration of the probable result of any given case, several circumstances must be kept in view, as: (1) The character and amount of vaccination. (2) *Age*.—Very few infants under one year recover, and from birth to five years the disorder is very fatal. In old age the chances of recovery are slight. (3) *Pregnancy*.—The mortality in pregnant women is greater than in non-pregnant women, and gestation probably tends to impart the hæmorrhagic character to the disease (see PREGNANCY, Disorders of). (4) *Eruption*.—The quantity and character of the rash must be regarded; if scanty the disease rarely proves fatal. Confluence, on the other hand, is of the utmost gravity. Flattened pustules, lack of acuminations, pasty-white eruption without swelling, and all forms of hæmorrhagic spots render recovery improbable. (5) *Delirium* is a comparatively harmless symptom. In confluent cases patients die from exhaustion and septicæmia; rapidity, dicrotism and feebleness of pulse are critical signs, as also low muttering delirium, restlessness, insomnia, diarrhœa, absence of perspiration, and refusal of nourishment.

COMPLICATIONS.—*Bronchitis*, sometimes associated with *Pneumonia*, is generally present. *Pleurisy*, often purulent, is a tolerably frequent and dangerous complication. *Diphtheria* is an accredited complication. The writer has seen many

laryngeal disorders in small-pox, but never anything resembling characteristic diphtheritic membrane. *Glossitis* arises during the secondary fever; the tongue becomes enormously swollen, causing dyspnoea and dysphagia. Few of those so attacked recover. *Inflammation of the larynx*, leading to ulceration of the mucous membrane and necrosis of the cartilages, is now and then seen; in such the epiglottis and aryteno-epiglottidean folds are oedematous, with the cartilages necrosed and surrounded by pus; this generally occurs during convalescence, and may terminate in either dysphonia or aphonia.

Otorrhœa and its results are not infrequent; but *mastoid abscess* is a very rare complication.

Ulceration of the Cornea is a common complication, and often leads to complete destruction of one or both eyes. It begins at various stages of the disease—the second week is, however, the most common—and runs a very rapid course. Starting as a conjunctivitis with itching and photophobia, it extends in perhaps a few hours only to the covering of the eyeball: the swelling rapidly increases, and at the same time a copious purulent discharge occurs. On examination, a small spot of ulceration can be seen near the margin of the cornea; this spreads with marvellous rapidity, destroying the several layers, and giving rise to hypopyon and panophthalmitis. It is very doubtful if, as stated by some, pocks form on the eyeball. The most favourable result is an interstitial keratitis.

The *abdominal viscera* are rarely affected, but occasionally diarrhœa is very persistent. *Edema of the legs*, due to weakness and exhaustion, is sometimes seen; it may also be due to chronic nephritis, but the latter is a very rare sequela of small-pox. *Permanent psychical disturbance* is the result occasionally of the delirium, and occurs in both mild and severe cases. *Dementia* occasionally results. See INSANITY IN SPECIAL DISEASES, p. 770.

Boils are the most common of all the sequelæ of small-pox. They are very numerous and painful, and protract the convalescence.

Gangrene, usually of penis and scrotum, and *erysipelas* of scalp and face are sometimes met with, and almost invariably after confluent small-pox, at the end of the second week.

Orchitis is an occurrence of considerable frequency, especially in severe cases; it is occasionally met with in lads, but more often in adults, and appears and develops with the eruption. Resolution is the usual termination.

Baldness of a permanent character may result from small-pox, also loss of beard and whiskers.

TREATMENT.—There is no drug capable of exerting an influence over the course of the disease after the manifestation of the symptoms peculiar to small-pox, but at certain stages vaccination can prevent or modify it: for example, if the operation were performed within forty-eight hours after a person had been exposed to, and contracted the disease, his safety would be secured; if delayed for seventy-two hours the attack would be modified; done later, however, it would be absolutely ineffectual, and serve only to bring vaccination into disrepute. The only two drugs that have justified a trial are salol and sulphite of sodium. The ordinary rules for nursing infectious patients must be carried out; the nurses should be protected by vaccination (see NURSING THE SICK; FEVER, p. 535). If the rash come out thickly it will be wise to remove as much of the hair off the head and face

as possible. The diet should be light and nutritious; for ordinary drink fresh-made lemonade is acceptable, and for children cold or iced water. Stimulants with eggs may be required; the diffusible stimulants, as ammonia and ether, may be needed. Constipation should be relieved. Daily tepid sponging of the whole body must be insisted upon, and as it is common for boys and men who have long prepuces to suffer from retention of urine from phimosis, this should be attended to, as also the state of the genital organs of women; indeed the utmost cleanliness cannot be too strongly enforced.

During the stages of suppuration and desiccation prolonged warm baths are appreciated, by which the local inflammation is often much reduced. In France the patient is sometimes completely washed two or three times a day in a solution of perchloride of mercury (1 in 1,000). The use of a dark chamber is said to have modified the eruption; the same results are perhaps obtained by excluding the ultra-violet rays, which have strong chemical action, by means of red window-glass and red curtains. The early convulsions of children are best met with bromide of potassium and henbane, while sleep in adults is obtained either by morphine or the administration of liquor opii sedativus. Chloral hydrate is an untrustworthy narcotic, and by no means devoid of danger. Care should also be taken not to push narcotics too freely, otherwise coma may ensue and a fatal collapse. For the throat-trouble, ice constantly sucked, a gargle of carbolic acid and glycerine, or a solution of chlorate of potassium affords relief. Spraying with glycerine and tannin or alum is sometimes useful. In the violent delirium, so often associated with small-pox, mechanical restraint is often demanded to obviate unexpected results, and although alcohol too often plays an important part in the causation, no doubt it is partly occasioned by the intensity of the fever. Reduction of temperature usually resists the measures employed. Antipyretics avail but little, and are not devoid of danger; even the cold bath is not efficacious, except for very short periods. In the stages of pustulation and scabbing intolerable itching is often complained of, and for its relief vaseline and carbolised oil, or some other greasy material is the most favoured. The result of considerable experience teaches that anti-pruritic remedies are useless, and tend to promote rather than prevent both the pitting and the great irritation that occurs. Lead-lotion, to which has been added liq. carbonis detergens, applied warm, very frequently answers better. In young children nothing avails if this symptom is much in evidence, and it portends an almost certainly fatal result. Gregory's remark that 'the masks and ointments formerly in use for that purpose (the prevention of pits and scars) are in reality more hurtful than beneficial' is as true now as when he made it.

Pain in the feet—sometimes very intense—is best relieved by a hot linseed poultice.

Complications must be treated according to their nature. The most satisfactory procedure as regards the terrible affection of the eyes is the application of the yellow oxide of mercury to the ulcer itself as soon as discovered, supplemented by the frequent and free use of a lotion made of extract of belladonna (gr. x ad ʒj), applied as warm as possible; this keeps the pupil well dilated, diminishes swelling, and reduces the pain, which, however, is as often absent as present.

Small-pox patients convalesce quickly and require few if any so-called tonics; their large appetites are notorious. Warm antiseptic baths should be taken every second or third night during the stage of convalescence, and no patient should be permitted to mix with the healthy until every scab has fallen off and the skin is in a normal condition. Scabs are often found under the nails of both feet and hands, which sometimes are cast off.

The disposal of the dead claims attention, and certain precautions are imperative. Delay in burial should be most strenuously opposed, and cremation by all means carried out if possible. See PUBLIC HEALTH: Disposal of the Dead, p. 1346.

For the disinfection of the room occupied by a small-pox patient, see article DISINFECTION, pp. 412-415; and PUBLIC HEALTH, p. 1345.

W. GAYTON.

SMELL, Disorders of.—See NOSE, Diseases of; and OLFACTORY SENSE.

SNAKE-POISONS.—See VENOM, Effects of; VENOMOUS ANIMALS.

SNEEZING, Paroxysmal.—SYNON.: Fr. *Coryza Spasmodique*; Ger. *Niesekrampf*.

DEFINITION.—An affection characterised by frequent and uncontrollable attacks of sneezing, out of proportion to any nasal secretion.

ÆTIOLOGY.—The causes of excessive sneezing may be broadly classified as *extrinsic* and *intrinsic*. *Extrinsic* causes include especially various vegetable substances in the form of powder, of which tobacco-snuff is the type, and the pollen of certain plants the most frequent example (see HAY FEVER). The *intrinsic* conditions in connection with which the affection occurs vary considerably. In some cases it is associated with whooping-cough or asthma, and it is especially common in young persons. Paroxysmal sneezing, especially in the early morning, is occasionally associated with epilepsy. It has been met with in pregnancy, and even during more than one pregnancy in the same person, ceasing in the intervals, and has been supposed to replace morning-sickness (Barnes). In some persons a bright light or intense colour is sufficient to determine an attack of sneezing, apparently by some abnormal sensory association.

SYMPTOMS.—The morbid sneezing has no special characters. It is distinctly a reflex act, being usually excited by some slight impression on the fifth nerve. A catarrhal condition of the nasal mucous membrane is common. The secretion has been thought to be, in some cases, of a special character, but its nature is unknown.

TREATMENT.—The attack itself may usually be cut short by a strong impression on some branch of the fifth nerve; when this fails, a mustard poultice to the back of the neck, or an emetic, may be employed, if the attack is sufficiently severe. Atomised astringent nasal inhalations, or the vapours of creosote or iodine, are useful, but the most effectual agent is the application to the nasal mucous membrane of a solution of hydrochloride of cocaine by a nasal douche. The general health should be put right, and a course of bromide of potassium is often useful, combined with iron, quinine, or arsenic. See CATARRH; and HAY-FEVER.

W. R. GOWERS.

SNUFFLES.—A popular term for the condition in which a nasal discharge exists in children suffering from congenital syphilis. See *SYPHILIS*.

SODEN, in Taunus, Germany.—Common salt waters. See *MINERAL WATERS*.

SOFTENING.—*SYNON.* : Fr. *Ramollissement*; Ger. *Erweichung*. See *BRAIN*, Softening of; *SPINAL CORD*, Softening of; *FATTY DEGENERATIONS*; *THROMBOSIS*; and *EMBOLISM*.

SOMNAMBULISM.—Sleep-walking. See *SLEEP*, Disorders of.

SOMNILOQUY.—Sleep-talking. See *SLEEP*, Disorders of.

SOMNOLENCE.—An unnatural drowsiness or disposition to sleep. See *SLEEP*, Disorders of.

SONOROUS RHONCHUS.—A variety of dry *râle* or rhonchus, of a low-pitched character, resembling snoring and similar sounds, and produced in the larger air-tubes. See *PHYSICAL EXAMINATION*; and *RHONCHUS*.

SOPOR (Lat.).—An unnatural deep sleep, from which the patient can only be roused with difficulty. See *CONSCIOUSNESS*, Disorders of.

SOPORIFICS (*sopor*, heavy sleep).—*SYNON.* : Fr. *Soporifiques*; *Soporatives*; Ger. *Einschlâfernde Mittel*.—Agents that promote sleep. See *NARCOTICS*; and *SLEEP*, Disorders of.

SORDES (Lat. : filth).—*DEFINITION.*—Crusts which form upon the lips and teeth of persons suffering from extreme exhaustion.

DESCRIPTION.—Sordes occur commonly in what is called the typhoid state, whether this be due to typhoid fever or to the intoxication produced by the poisons of any of the pathogenic organisms which multiply in the body. Sordes appear first as thin, light-yellowish crusts upon the prolabia, generally in close proximity to the teeth; gradually increase in thickness and in area; and, changing their colour to brown, or even black, at length extend to the adjacent surfaces of the teeth. They seldom or never cover those portions of the teeth which are hidden by the lips, but spread over their exposed surfaces; so that, as the patient lies with slightly parted lips, they bridge over the interval in the form of a narrow band upon the middle of the incisors of the upper jaw. When the lips are more widely separated, the sordes do not extend, except in conditions of extreme exhaustion, over the whole of the exposed surfaces of the teeth, but form two ridges, corresponding with the margins of the upper and lower lips.

PATHOLOGY.—Sordes are composed of various schizomycetes, mingled with *débris* of food and epithelium. Micrococci of various forms, and often diplococci, with bacteria and bacilli, occur almost constantly; *Bacillus subtilis* frequently; and one of the writers has found, each in a single instance, *Sarcina ventriculi* and *Spirochaeta plicatilis*.

TREATMENT.—These organisms, which are of constant occurrence on the papillary surface of the healthy tongue, are easily dislodged from the smooth lips and teeth. But in conditions of great prostration, especially when the prostration is associated with delirium, the slight frictions necessary for their

removal are not made. There is often a marked absence of saliva due to its deficient secretion; hence they obtain so firm a hold that they can only be removed by careful and repeated cleansing. Such cleansing may with advantage be performed with a piece of soft rag, or a brush, dipped in a weak solution of Condyl's fluid, or in the glycerine of boric acid. A spoonful of iced water repeated frequently will be a great comfort. So, too, is a small piece of ice allowed to dissolve in the mouth, or lemonade, or weak black tea without milk, slightly acidulated with a slice of lemon.

HENRY T. BUTLIN.
D'ARCY POWER.

SORE-THROAT.—A popular name for various affections of the pharynx, larynx, and tonsils. See *LARYNX*, Diseases of; *PHARYNX*, Diseases of; and *TONSILS*, Diseases of.

SOUFFLE (Fr.).—A soft, blowing sound. The term is applied either to the respiratory murmur heard over the lungs; or to certain murmurs heard in connection with the heart or blood-vessels. See *PHYSICAL EXAMINATION*.

SOULZMATT, in Alsatia.—Alkaline table-waters. See *MINERAL WATERS*.

SOUTH AFRICA.—No description of this country in its present condition (November, 1901) can be of any medical interest.

SPA, in Belgium.—Iron or chalybeate waters. See *MINERAL WATERS*.

SPAIN, Southern.—See *MALAGA*; and *CLIMATE*, Treatment of Disease by.

SPANÆMIA (*σπανός*, rare; and *αἷμα*, blood).—A condition of blood, in which the proportion of its solid constituents is below the normal, the blood thus appearing thin. See *ANÆMIA*; and *BLOOD*, Morbid Conditions of.

SPAS.—See *MINERAL WATERS*.

SPASM.—*SYNON.* : Fr. *Spasme*; Ger. *Krampf*. *DEFINITION.*—A name given to abnormal contraction, occurring either in muscular organs, in single muscles, or in groups of muscles.

I. Spasm of muscular organs.—We may cite as instances those spasms which occur in the pharynx in hydrophobia; the contractions of the œsophagus in œsophagismus and in some cases of hysteria; the painful contractions of the intestine constituting intestinal colic; of the lower end of the rectum in tenesmus; of the bladder or of the urethra in certain cases of inflammation with irritability; of the vagina in vaginismus; of the uterus in rare cases of sudden abortion resulting from shock; possibly of the heart in certain diseases of that organ; of the vessels in various regions of the body, and on various occasions, from overaction of vaso-motor nerves; of the bronchial tubes in certain cases of asthma and hay-fever; of the glottis in laryngismus stridulus, and in pertussis; as well as of the gall-ducts or ureter under conditions of irritation, either direct or reflex. All are due to excessive nervous stimuli, maintaining conditions of muscular contraction which are unusual both in degree and in duration. These spasms are, therefore, tonic in type, and in almost all the cases cited involuntary muscular

fibres are those involved. See COLIC; and the various articles on the special organs involved.

2. *Spasm of single muscles or of groups of muscles.* This is divisible into two main categories, that is, into *tonic spasms*, in which the contractions are continuous, and *clonic spasms*, in which contractions and relaxations occur in quick succession; the former being typified by cramps, and the latter by convulsions.

Under *tonic spasms*, we may have cramps of brief duration, affecting a single muscle, such as the diaphragm in hiccough; or of prolonged duration, as in the sternomastoid in certain cases of wry-neck. The tonic contraction may affect several muscles at the same time, as in lock-jaw, or the painful cramps which occasionally occur in the calves of the legs, or in other parts of the body. Such local spasms occur also in the condition known as tetany, in conjugate deviation of the eyes, and in writer's cramp; likewise in spasmodic spinal paralysis, in hysterical paralysis, and under various conditions of irritative organic disease implicating motor nerves, or motor centres or tracts either in the spinal cord or in the brain. More general tonic spasms occur in cataplexy, in tetanus, and in strychnine-poisoning.

This whole class of tonic spasms is supposed to be due to irritation, mechanical or chemical (nutritive), operating *directly*, either upon motor centres or upon the fibres conveying motor incitations in some part of their course between the brain and the muscles. In other cases, however, tonic spasms are of *reflex* origin, and the cause of irritation operates in or upon sensory surfaces, nerves, or centres.

Clonic spasms are also of various kinds. They may be limited to single muscles, such as the orbicularis palpebrarum; or they may affect particular groups of muscles, such as those of one side of the face, or the muscles of the lower jaw on both sides, or certain of the abdominal muscles, or some of the foot-muscles, as in ankle-clonus. In other cases clonic spasms may be more general, taking the form of unilateral or of bilateral convulsions. The latter also may be irregular or of coördinated type.

Where clonic spasms are much slighter in degree and in range, affecting some muscular fibres and that to a small extent, rather than entire muscles in a more marked manner, we have the production of *tremors*, which may be either fine or coarse, local or general.

Transitional conditions also exist, connecting all these various manifestations more or less closely with one another. See STRYCHNINE, Poisoning by; TETANY; WRITER'S CRAMP.

H. CHARLTON BASTIAN.

SPASMODIC.—SYNON.: Fr. *Spasmodique*; Ger. *Krampfhaft*.—A descriptive epithet applied or applicable to conditions or diseases in which spasms, and mostly those of the tonic class, are met with as prominent or essential constituents; for example, *spasmodic croup*, *spasmodic asthma*, *spasmodic stricture*. See SPASM.

SPASMUS NUTANS.—See HEAD-NODDING OF INFANTS (p. 630).

SPECIFIC.—When applied to a disease, the word 'specific' signifies that such disease is produced by a cause different from that of any other disease, and that it has distinctive characters; for example, syphilis and the eruptive fevers. When

applied to a remedy, it implies that the substance has a distinct and definite effect in the cure of a certain disease, such as mercury in syphilis, or quinine in ague; or that it acts upon a particular organ, as ergot upon the uterus.

SPECTACLES, Uses of.—See STRABISMUS; and VISION, Disorders of.

SPECTROSCOPE IN MEDICINE.—INTRODUCTION.—Defining a prism in optics, as a solid angle bounded by two planes, it may be stated generally, that a spectroscope consists essentially of a prism and a slit. A diffraction-grating may replace the prism, but for medical purposes no advantage is gained by its use.

According to the kind of spectroscope, other accessories are required; thus, there are two kinds of spectroscope used for medical purposes—*chemical spectroscopes* and *micro-spectroscopes*. In the former the slit is set at one end of a tube, known technically as the collimator, at the other end of which, and having the slit in its focus, is a lens which makes the rays admitted through the slit parallel before entering the prism. The prism refracts and disperses (when they can be dispersed) the rays passing through it, and, if they be rays of white light, they emerge from the second face of the prism as a coloured image of the slit, red at one end, violet at the other, passing from red into orange, yellow, green, blue, and violet. This rainbow-band is the continuous spectrum of the light-source. If, on the other hand, a light-source be used containing not compound rays, but those of one colour only—for example, such as are yielded by a colourless flame into which a salt of sodium is introduced—then the rays emerge from the prism as a yellow line, a yellow image of the slit. The prism should be set at the minimum angle of deviation for the sodium-line. (For the meaning of this term, and the method of setting the prism, the reader may consult Glazebrook's *Physical Optics*.) The spectrum is then magnified in a telescope, which is a small astronomical refractor, moving round the table of the spectroscope, and capable of being focussed.

In *direct-vision* spectroscopes, to which the micro-spectroscope belongs, a compound prism is used; by this means the slit and the prism can be placed in the same straight tube.

In the *micro-spectroscope*, which is a combination of a microscopic eye-piece and a direct-vision spectroscope, a lens collects the rays. Passing up in the optic axis of the tube of the microscope, they then fall on the slit and are made parallel by another lens placed between the slit and the compound prism. This compound prism is composed generally of three prisms of crown-glass and two of flint-glass, united to each other by Canada balsam. By such a combination deviation is eliminated, while sufficient dispersion is retained to make the spectrum long enough. A right-angled reflecting prism covers half the slit, so that a second spectrum can be compared with that of the substance beneath the objective of the microscope; a side-stage being adapted to the side of the micro-spectroscope, perforated with a square hole, and provided with spring clips to hold tubes, &c., the light being reflected into the hole by means of a plane mirror suitably mounted. The slit can be widened and narrowed by means of a screw and springs bearing on the

jaws of the slit, which should be made to move away from each other equally, as this prevents displacement of the centre of a band or line when the slit is widened.

It is advisable to work with both these instruments; but if one only is desired, then the micro-spectroscope is to be recommended. Useful observations may be made, however, with a pocket spectroscope, procurable at a trifling cost.

DIFFERENT KINDS OF SPECTRA.—When light from various sources is examined with the spectro-scope, the appearances observed may be classed under three heads: (1) Spectra consisting of bright lines or striæ; (2) Continuous spectra; (3) Absorption-spectra.

(1) *Bright-line spectra* consist of bright lines on a dark background, occurring singly or in groups, placed in the same or different parts of the spectrum. Glowing gases or glowing vapours yield such spectra when their combinations are heated in the Bunsen flame; the heavy metals require to be volatilised at a much higher temperature by means of the electric spark.

Metallic poisons may be detected by their bright-line spectra; and the elements, sodium, potassium, calcium, and others, can be similarly detected in the ashes of calculi, and elsewhere. The late Dr. Bence-Jones, in his study of the 'chemical circulation,' used the spectroscope to determine the rapidity of the distribution of lithium-salts to distant parts of the body, such as the crystalline lens and the hip-joint.

(2) *Continuous spectra* are yielded by incandescent solids and fluids. Gas-light, candle-light, the electric and lime-lights, incandescent platinum, &c., yield a continuous spectrum—the band of colours referred to above, red, orange, yellow, green, blue and violet—one colour passing gradually into the next without break.

(3) *Absorption-spectra* are of two kinds: (a) those consisting of black lines occurring in a continuous spectrum; and (b) those consisting of broad black spaces, or shadings of varying degrees of darkness, in a continuous spectrum. The spectrum of the sun, and of many of the stars, are examples of the former, and the latter are those with which medical spectroscopy has to do. These black lines, bands, or shadings are spaces of darkness in the spectrum due to the absence of certain rays. Those in the solar spectrum, which were discovered by, and named after, Fraunhofer, are caused by the presence of vapour in the sun's atmosphere. Every gas and every vapour when in a comparatively cool state absorbs the same rays that it emits in the incandescent state. If no vapour existed around the sun, then we should have thousands of bright lines belonging to the elements burning in the sun. Some of the solar lines are, however, telluric, i.e. of earthly origin, and are due to absorption in the earth's atmosphere. The most prominent Fraunhofer lines, beginning from the red end of the spectrum, have been called A, a, B, C, D, E, b, F, G, and H.

Solutions of animal and vegetable colouring-matters have the property of arresting certain rays of light passing through them; and corresponding to the rays stopped, or enfeebled, we have spaces of darkness in the spectrum—the *absorption-bands*. Solid pigments may do the same, even when the light analysed (by the prism) is reflected from their

surface. Absorption may, however, be general, that is, one or the other end of the spectrum may be absorbed.

THE STUDY OF ABSORPTION-SPECTRA.—Two pigments may give the same spectrum, and yet not be identical; to prove identity their respective spectra must be changed in the same way by reagents. In studying such spectra the first thing to be done is to map the principal lines in the solar spectrum. By consulting a map of the solar spectrum, and then examining the spectrum of diffused daylight, concentrated, if necessary, by a condenser on the slit, it is easy to identify the most prominent Fraunhofer lines referred to above. Of these A, a, and H are not easy to see. Next they should be mapped on a piece of smooth drawing-paper. Both the chemical spectroscope and the micro-spectroscope should be provided with *photographed scales*; angular measurements for the former are only indispensable for certain measurements, such as the calculation of refractive indices. These scales can in both instruments be illuminated by a mirror at the end of the scale-tube. By means of a millimetre-scale a map is easily made from the readings obtained from the Fraunhofer lines. The scale of this instrument is to be set so that D shall stand at a convenient number; for the chemical spectroscope it is generally set at 50.

In mapping an absorption-spectrum we begin by setting the scale. A flame coloured by a sodium-salt is brought before the slit of the chemical spectroscope, or reflected up by means of the microscope-mirror on to the slit of the micro-spectroscope; then the scale is adjusted until the sodium-line stands at the same number on the scale that the D line stood at. The slit is then illuminated by means of artificial light, say an Argand gas-burner, the light being condensed on the slit by a bull's-eye condenser in the case of the chemical spectroscope. The fluid to be examined is then placed in a vessel, such as a test-tube, held in a suitable holder, before the slit of the chemical spectroscope, or in a small flat-bottomed tube let into a thin slab of wood, for the micro-spectroscope. If the solution be too darkly coloured to transmit the spectrum, either a thinner layer or a more dilute solution must be used. Hermann's hæmatoscope is frequently used for this purpose, as by means of a screw-adjustment any depth of fluid can be examined; it is only applicable to the chemical spectroscope. The slit must not be too wide, and the focus should not be altered after the sodium-line has once been focussed. A black cloth, to exclude extraneous light, should be used to cover the chemical spectroscope. Bands in the red end of the spectrum are best seen by artificial light, in the violet by daylight. For examining portions of organs or tissues a compressorium is very useful, as various degrees of thickness can then be examined.

When publishing results it is necessary to give the measurements of absorption-bands in terms of a universal scale. Now, no two arbitrary scales agree, so that all measurements should be reduced to wave-lengths, denoted by the sign $\lambda\lambda$. Zeiss's micro-spectroscope is provided with a wave-length scale, but the readings of any spectroscope may be reduced to wave-lengths by means of an *interpolation curve*. A sheet of logarithm-paper has the arbitrary numbers of the scale of the spectroscope written out along the top line, each tenth of an inch corresponding to one division of the scale. Along the

side-line, at right angles to this, the wave-lengths are written, each tenth of an inch here being made to correspond to two millionths of a millimetre. It is convenient to begin at the top of this line with 410 millionths of a millimetre, then the next *inch* division has 430 placed opposite to it, and so on down to 730. On referring to Angström's numbers, the wave-lengths of the principal Fraunhofer's lines are found to be as follows in millionths of a millimetre: A = 760, a = 718, B = 686, C = 656, D = 589, E = 526, b = 516, F = 486, G = 430. Their value on the arbitrary scale of our spectroscope has been determined by actual measurement. By means of these data their positions on the paper are denoted by small crosses; through the centres of these crosses a curve is drawn. When now we want to know the value in $\lambda\lambda$ of any number of the scale of the spectroscope, we can find it by running the eye along the perpendicular line joining the top line with the curve, thence to the right-hand side line, where its wave-length is given. In other words, the abscissæ are the arbitrary numbers of the scale, the ordinates the wave-lengths.¹

USES OF THE SPECTROSCOPE IN MEDICINE.—It is not necessary to describe in detail the various pigments and their spectra which have been discovered in the animal body. It will suffice to point out briefly some of the applications of the spectroscope to medicine, especially as a full account of most of these pigments is given in Professor Kendrick's *Text-book of Physiology*, vol. i., and in Professor Halliburton's *Text-book of Chemical Physiology and Pathology*.

In medical jurisprudence the spectroscope is indispensable for the detection of blood-stains on clothing, knife-blades, &c. In poisoning by carbonic oxide from the inhalation of coal-gas, charcoal-fumes, &c., the spectrum of the blood is peculiar, and reduction, by means of sulphide of ammonium, cannot be brought about. Other poisonous gases have been found to enter into peculiar combinations with hæmoglobin, such as nitric oxide, &c.; doubtless research will reveal the existence of many such combinations.

To the physician the instrument is useful in enabling him to detect blood and its decomposition-products, and also bile-pigments and their decomposition-products, in urine and various pathological fluids, under diseased conditions, and from the administration of drugs. In the detection of various pigments, which cannot be recognised by other methods, microscopic or chemical, as well as in enabling us to form an idea as to the extent and nature of the metabolic changes taking place under the influence of disease, the spectroscope promises to be a helpful guide. In fact, pigments have already been found in the urine which can be produced artificially from hæmoglobin and hæmatin, &c., by certain chemical methods, by means of which we have found out how they are produced in the body. These pigments serve as finger-posts to point out the various steps of the abnormal processes by which not only they, but the substances with which they are associated, are originated. The pigment known as uro-hæmatoporphyrin may be given as an example. This can only be produced in the labo-

ratory by the action of very energetic *reducing* agents on hæmoglobin or hæmatin, yet it occurs in many diseased conditions, in which, until recently, it was supposed that increased oxidation took place; but we now know that exactly the opposite occurs, and the spectroscope confirms this. Again, the rapidity of the reduction of oxyhæmoglobin to reduced hæmoglobin may be measured by the spectroscope, as Hénocque has shown. Observing the ungual phalanx of the thumb, at the root of the nail, by means of a direct-vision spectroscope, he finds, if a ligature be quickly applied, that in health reduction takes place in seventy seconds, whereas in anæmic states it may happen in from thirty to forty seconds, being dependent on the amount of hæmoglobin, the amount of hæmoglobin reduced in one second being 0.2 per cent. under normal conditions.

Wertheimer has also made use of the spectroscope in a novel and ingenious manner. He injected sheep-bile into the femoral and mesenteric veins of dogs, and detected the presence of the writer's cholo-hæmatin in the bile of the dog within ten minutes after its injection into the femoral, and five after its injection into a mesenteric vein, showing, as he remarks, that the liver has the power of picking the constituents of bile out of the circulating blood.

Instead of repeating what is already known about the pigments and their spectra, attention is called to the accompanying chart (*coloured*), where the most important spectra are figured. In this, sp. 1 represents the spectrum of oxyhæmoglobin. That of the compounds of carbonic oxide and of nitric oxide, respectively, with hæmoglobin resembles this, except that the bands are nearer the violet. 2 is the spectrum of reduced hæmoglobin, got by adding sulphide of ammonium to a solution of oxyhæmoglobin. 3 is the spectrum of methæmoglobin, a substance intermediate between hæmoglobin and hæmatin, in which the oxygen is more firmly combined than in oxyhæmoglobin. It occurs on the edges of healing wounds, in old blood-stains, in various exudations, &c., and may be produced artificially by treating a solution of blood with a great number of reagents, which are not energetic enough to split the hæmoglobin into hæmatin and a proteid. 4 is the spectrum of alkaline methæmoglobin, *which is very often mistaken for oxyhæmoglobin in alkaline urine*. It may be obtained by adding a little ammonia to a solution of methæmoglobin. 5 is the spectrum of acid hæmatin in ether; and 6, that of alkaline hæmatin in rectified spirit. 7 is that of reduced hæmatin, got by adding a reducing agent, such as ammonium sulphide, to a solution of alkaline hæmatin. Hæmochromogen is another name for this pigment. This is a very important spectrum, as by its means blood can be detected with great certainty when present in minute traces. Sometimes blood may be present in urine, adhering to the sediment; if this be filtered off, digested in rectified spirit containing ammonia, filtered, and the filtrate treated with ammonium sulphide, this spectrum is obtained. Again, in detecting blood-stains on clothing, knife-blades, &c., the bit of cloth, or blade, is treated with the same reagents, with a similar result. 8 is the spectrum of acid hæmatoporphyrin, got by dissolving hæmoglobin in strong sulphuric acid and filtering through asbestos; and 9, that of alkaline hæmatoporphyrin, obtained by dissolving hæmatoporphyrin in alcohol and ammonia. These two spectra, and 14 and 15, should be carefully studied, as a condition known as hæmatopor-

¹ By using the *reciprocals* of the numbers expressing the wave-lengths, the curve becomes nearly a straight line. The reciprocal of a fraction is obtained by making the numerator and the denominator change places. Thus, the reciprocal of $\frac{1}{2}$ is 2, the reciprocal of $\frac{1}{5}$ is 5, and of $\frac{1}{3}$ is 3, i.e. 5.

B C D E b F G

*Solar Spectrum.**Oxy-Haemoglobin.**Reduced
Haemoglobin.**Methaemoglobin.**Alkaline
Methaemoglobin.**Acid Haematin
in Ether.**Alkaline Haematin
in rect. spt.**Reduced
Haematin.**Acid
Haematoporphyrin.**Alkaline
Haematoporphyrin.**Normal Urobilin
in rect. spt. and
 H_2SO_4 .**Do. isolated & treated
with $ZnCl_2$ & NH_4HO .**Pathological Urobilin
in rect. spt. & H_2SO_4 .**Do. isolated & treated
with $ZnCl_2$ & NH_4HO .**Acid Uro-
haematoporphyrin.**Alkaline Uro-
haematoporphyrin.**Gmelin's
Reaction.**Peltenkofer's
Reaction.**Indigo blue and
Indigo Red in
Chloroform.*

Plate representing the Spectra of the most important Pigments.

pyrurina has lately been described, first by the writer, since then by others. In many of these cases it seems to have been caused by the administration of sulphonal or trional, but not in all. The urine is a deep Burgundy-red colour, and decomposes very slowly; a specimen may remain for years unchanged. 10 is the spectrum of normal, and 12 that of 'pathological,'¹ urobilin, in rectified spirit and sulphuric acid; that of stercobilin is almost identical with the latter. 11 and 13 are the spectra of normal and pathological urobilin respectively, treated in a spirit-solution with zinc chloride and ammonia, which develops a green fluorescence. 14 and 15 are the spectra of acid and alkaline urohæmatoporphyrin. This pigment can be obtained by treating hæmatin with energetic reducing agents, aided by heat, such as sodium-amalgam, zinc and sulphuric acid, &c.; and an early stage hæmatoporphyrin is produced, then this pigment.² It occurs as such, as the writer has discovered, in the urine of acute rheumatism, and of many other diseases, including Addison's disease. A. E. Garrod finds that its presence is independent of blood-destruction in rheumatism, and must probably depend upon a failure of those organs, in which it should be metabolised to other pigments, to effect such change. See p. 614.

Bilirubin and biliverdin, the colouring-matters of human bile, give only a *general* absorption of the spectrum; but either of them when treated with nitric acid gives 16, that of Gmelin's reaction, when the spectrum of the violet stage is examined. 17 is the spectrum of Pettenkofer's reaction; and 18, that of indigo-blue and indigo-red from *normal* urine, obtained by heating with hydrochloric acid to which a little nitric acid has been added, then, when cold, agitating with chloroform. This test succeeds when affe's fails.

The above are the most important spectra from a purely medical point of view. There are, of course, other pigments in the human body present under normal conditions, such as myohæmatin and the histohæmatins (discovered by the writer), the porphyrins or fat-pigments, formerly known as steins, and the black pigments included under the name of melanins. The last show no absorption-bands, and probably arise somehow in connection with the lipochromes, although in cases of melæuria associated with melanotic sarcomata a chromogen, or colourless mother-substance, is present which on oxidation changes into melanin. At all events, the origin of a melanin from hæmoglobin seems doubtful.

Many attempts have been made to explain the bronzing of the skin in Addison's disease, but as yet physiologists are not disposed to accept any of the hypotheses offered. The writer finds, however, that the suprarenal bodies of many mammals contain a chromogen, pointing to the indisputable fact that these organs are concerned in the downward metabolism of effete hæmoglobin; this, taken in connection with the presence of urohæmatoporphyrin in the urine, would seem to point out that, if the suprarenal bodies are diseased, then effete blood-colouring matter must be present in excess in the blood, and produce staining of skin and mucous membranes.

The writer thinks it right to add that his reasons

for supposing that there are at least two kinds of urobilin, viz. normal and pathological, and that urohæmatoporphyrin is not identical with ordinary hæmatoporphyrin, are based on the fact that similar substances are obtained by reducing hæmatin *outside* of the body. These pigments correspond with those found in urine, and whether they are mixtures or not, represent various stages of metabolism.

CHARLES A. MAC MUNN.

SPECULUM (Lat.).—SYNON.: Fr. *Speculum*; *Miroir*; Ger. *Speculum*; *Spiegel*.

DEFINITION.—An instrument adapted for exploring the several channels and deeper-seated parts of the human body. The chief of these are the ear, the eye, the nose, the mouth, the throat, the rectum, and the vagina. For each of these there are specially adapted instruments.

DESCRIPTION.—Specula are made of various materials, and in a variety of shapes. The speculum is intended not only to permit and facilitate inspection, but also to dilate the canals and to expose parts, in order that they may be treated surgically, or have medicaments applied to them. For this reason a cylindrical speculum will not always answer the purpose; we have, therefore, bivalve and trivalve specula, and many other forms. On account of the friability of glass, other material has not infrequently to be used, such as white polished metal, celluloid, or wood; the last is objectionable, as it has no reflecting power; but when it becomes necessary to apply the actual cautery through a speculum, a substance must be employed that is a non-conductor of heat and non-friable, such as wood.

VARIETIES.—**Aural specula.**—Some have a trumpet-shaped opening, which facilitates the introduction of light, and greatly increases the illuminating and reflecting power. There are also bivalve aural specula with a screw lever, and others with handles attached so as to separate the blades. See EAR, Diseases of.

Eye specula.—These are known by the name of *eyelid retractors* and *ophthalmoscopes*, both of which are really specula for examining the eye, though not generally classified as such. See OPHTHALMOSCOPE.

Nasal specula.—There are several forms of these, the great purpose they have to serve being that of dilating. One, known as Elsberg's, is three-bladed. See NOSE, Clinical Examination of.

Throat specula.—Specula for examination of the throat are generally called *laryngoscopes*. See LARYNX, Clinical Examination of.

Rectal specula.—These are cylindrical, bivalve, or trivalve. The cylindrical are made on the principle of Fergusson's vaginal speculum, but with an opening so as to expose the wall of the rectum at the part to which it is adapted. The valvular forms are made of white metal.

Vaginal specula.—Of these there are many kinds. Perhaps the most useful is that known as Fergusson's, which is cylindrical and made of glass, with a coating of mercury behind it, so as to give it reflecting power, and backed by vulcanised indiarubber. An improved variety of this is of a tapering form, so as to admit more light (see UTERUS, Diseases of). Sims' duckbill speculum is of great use in retracting the perineum and dilating the vagina, when space is required for operation, as in vesico-vaginal fistula. Then there are bivalve and trivalve metallic specula. Wooden

¹ According to some authors pathological urobilin is a mixture of normal urobilin and hæmatoporphyrin.

² Urohæmatoporphyrin is said by certain observers to be ordinary hæmatoporphyrin.

cylindrical specula are always used when the actual cautery is applied, for reasons already mentioned. For the ordinary purposes glass is the preferable material, as it is unaffected by caustics.

The uses of specula will be found described in connection with the diseases of the several organs to which they have reference.

It is worthy of record that many varieties of specula have been discovered among the ruins of Pompeii, and that recently a quadrivalve vaginal speculum has been unearthed there.

CLEMENT GODSON.

SPEECH, Disorders of.—**SYNON.** : *Troubles du Langage*; Ger. *Störungen der Sprache*.—Defects of speech are very various in their nature, degree, and mode of causation.

ÆTIOLOGY AND PATHOLOGY.—Disorders of speech may depend upon (1) *congenital*, or (2) *acquired* defects of the brain, or of certain of its nerves and sense-organs.

1. *Congenital defects.*—The most frequent and important of these defects is *deafness*, which entails mutism, so that the individuals thus afflicted are known as ‘deaf-mutes.’ It must, however, be borne in mind that this condition of mutism or dumbness may also be brought about by absolute deafness occurring from any cause after birth, but before the child begins to talk; or even after it has learned to talk, up to the fifth or seventh year. In cases of the latter type, the child, when without the accustomed guidance derived through the sense of hearing, soon forgets how to speak and becomes dumb. In addition to this class of cases, there are those of congenital idiocy without deafness, in which the child never learns to talk or articulate in the proper sense of the term (see **IDIOCY**). There are also other cases allied to the last, in which, owing to some intracranial lesion occurring either before, during, or soon after birth, the child’s subsequent mental condition is greatly impaired, as well as its motor power. In these most deplorable cases the child may never be able to speak in any distinct or articulate fashion, it may not be able to walk or even stand, or it may only be able to accomplish these latter acts imperfectly. In some of these children there is evidence of the existence of a hemiplegic condition, with arrest of growth of the paralysed limbs. Such patients are also frequently subject to one-sided fits; but it is not certain whether in such cases the inability to speak is especially prone to occur in those who are congenitally paralysed on the right side. In some of the less severe examples of this latter type which have come under the writer’s observation, speech has been merely deferred—the child has not commenced to speak till the fourth, fifth, or even the sixth year. See **DUMBNESS**; and **CEREBRAL DIPLEGIA**.

Hadden described several curious cases of speech-defect met with in children, to which the term ‘*idioglossia*’ has been applied. These persons have to a certain extent a language of their own; so that when asked to repeat phrases, they make use of sounds of their own instead of those proper to the words that should be employed. The sounds which they substitute are said to be always the same for the same words. Some of these patients at least seem to be capable of writing correctly from dictation. They may also show a fair amount of intelligence generally.

2. *Acquired defects.*—Among acquired defects of speech we have troubles of various degrees and kinds, which may come on at any period between infancy and old age, and which, as regards duration, may be temporary or permanent. The great variation in the extent and nature of these defects is due to the fact that the impeding condition or lesion may act (1) upon parts of the brain concerned with the genesis of thought, and of the will to speak; (2) upon some part of the nervous channels or centres concerned with the actuation of speech; or (3) upon the peripheral nerves and organs concerned with articulation and vocalisation. Thus it happens that acquired defects of speech may, in one set of cases, be associated with the most marked alterations in the intelligence or previous mental condition of the patient, while in others they may be represented by mere defective articulation or vocalisation. In briefly referring to the principal varieties, it will be convenient to pass from the simple to the more complex types.

Proper vocalisation is essential for the production of normal speech; where it alone is defective we have to do with various kinds of aphonia, which may be due to very different causes (see **VOICE, Disorders of**). Again, articulation as a mere motor act may be interfered with or perverted in diverse modes. Where speech-movements are inco-ordinate, we have such common defects as stuttering or stammering (see **STAMMERING**); or else those less marked perversions of speech-movements which are met with in some cases of chorea. Again, where the movements concerned in speech are more simply defective, we have that indistinctness of articulation and blurred utterance which, in various degrees, is so commonly associated with different forms of paralysis due to cerebral disease. To this kind of defect the name ‘*Aphemia*’ is now commonly applied. It presents itself under many various conditions, and with different degrees of completeness. It may show itself in its most extreme form in ‘*labio-glosso-laryngeal paralysis*,’ or in other forms of bulbar disease. This blurred or difficult articulation is also one of the signs met with in general paralysis of the insane, and in disseminated cerebrospinal sclerosis. Again, it occurs in association with hemiplegia caused by different lesions in various parts of the brain, between the bulb below and the cerebral cortex above. As a rule, it is most marked and most persistent in hemiplegia due to disease of the pons Varolii, while in lesions higher up it is apt to be slight and more transitory, especially where such lesions exist on the right side of the brain. It is evident, indeed, that this kind of defect is specially prone to occur where there is damage to the first parts of the outgoing tract leading from the kinæsthetic centres in the left third frontal convolution, or in any lower parts of the same tract, or when there is damage to the actual motor centres for articulation situated in the bulb. Damage to the third left frontal convolution itself gives rise to a form of speech-defect which is commonly known as *motor aphasia* (see **APHASIA**). Related to speech-disorders of this type are the other more complex and extremely varied defects of speech classed under the head of *amnesia*.

Finally, in this relation, reference should be made to certain forms of speechlessness occasionally met with in hysterical females, or in the insane of both sexes, in which there may be a deficiency of will to speak, dependent upon perverted cerebral action,

either without or with a discoverable basis of actual morbid changes. In such cases patients may remain dumb for months or for years; there may be no apparent motive, or the speechless condition may, in the insane, stand in direct or indirect relation to certain delusions.

PROGNOSIS AND TREATMENT.—The treatment of these various defects of speech will, of course, depend upon their nature, causes, and associated conditions. Reference must, therefore, be made to the several special articles in which the different forms of such defects are considered. It may be said here, however, that most of the forms of speech-disorder dependent upon congenital defects are comparatively little amenable to treatment. Those due to brain-lesions occurring later in life are oftener mere temporary defects, diminishing after longer or shorter periods, as the effects of the original lesions become lessened by time and treatment. Some of the marked cases of aphemia, moreover, are capable of being cured or greatly improved by careful daily drilling in the articulation of vowel-sounds and short words, provided this exercise be persistently and methodically carried out.

H. CHARLTON BASTIAN.

SPERMATIC CONCRETIONS.—See CONCRETIONS.

SPERMATORRHOEA (σπέρμα, seed; and ῥέω, I flow).—SYNON.: Fr. *Spermatorrhée*; Ger. *Samenfluss*.

DEFINITION.—A real or apparent discharge of seminal fluid, occurring without voluntary sexual excitement.

Two varieties may be recognised: (1) *True spermatorrhœa* is the discharge of spermatozoa from the urethra, or with the urine, at periods other than during sexual excitement. (2) *False spermatorrhœa*, or *prostatorrhœa*, is the discharge of the seminal-like fluid, destitute of spermatozoa.

ÆTIOLOGY.—Local irritation, whether from masturbation or from some diseased or disordered condition of the genital organs, is the cause of spermatorrhœa in the first instance. General or local morbid conditions, such as balanitis, phimosis, a long prepuce, urethral catarrh, irritability of the prostate, a tender spot in the urethra, varicocele, spasmodic contraction of the levator ani from rectal irritation, worms in the intestinal canal, constipation, and changes in the nerves or nerve-centres supplying the genito-urinary tract, inducing either hyperæsthesia or anæsthesia, may occasion or be the result of masturbation and subsequent spermatorrhœa. Sugar in the urine and involuntary emissions of semen at night are frequently associated in young men. When ætiological factors persist, slight mental or local stimuli are sufficient to perpetuate the discharge.

SYMPTOMS.—The first symptom that alarms the subject of spermatorrhœa is the occurrence of frequent nocturnal emissions, at first with, and afterwards without, erotic sensation. These reduce his strength, render him weak and irritable, and gradually prey upon his mind; and if, as frequently happens, masturbation be practised, they induce a condition of extreme mental depression. When the patient reaches this state, the mere reference to sexual matters, the sight of anything lewd, the act of defæcation, or a chance irritation of the penis during walking, riding, or driving, is often suffi-

cient to cause an abortive or, it may be, a complete emission. The discharge may, in advanced cases, find its way into the bladder and be voided with the urine. The discharge may be the ordinary seminal fluid; or it may be less in quantity, clearer, tenacious, more like synovial fluid in appearance and consistence. In the latter case it seldom contains spermatozoa, but it is usually only the fore-runner of the other more serious state, or it may alternate with it. A tender spot in the lumbar region of the spine is frequently complained of. These conditions combine to render the patient for the time being physically and mentally a wreck, sleepless, listless, nervous, anæmic, and with an old and insipidly anxious look upon his muddy or pimpled face. Intercourse becomes well nigh impracticable, the discharge of semen occurring before the introduction of the organ; or erection may be impossible or imperfect. Men in middle life, owing to weakness of the genital organs, are not infrequently affected by imperfect erections and by false spermatorrhœa.

DIAGNOSIS.—In the diagnosis of the cause of spermatorrhœa, the condition of the external genitals must first be determined. The presence of a tender spot, of hyperæsthesia, anæsthesia, or stricture, in the urethra, can be made out by passing a catheter. Pressure on the perinæum and inspection by aid of the urethroscope may assist in the diagnosis. The urine must be tested for sugar and for albumen. The discharge itself must be found and examined microscopically, the presence of spermatozoa establishing true spermatorrhœa. Glairy fluids, like that of prostatorrhœa, occur in the urethra during the last stages of a gleet, straining at stool, and also in stricture. The history of the case, and catheterism, readily clear up the cause of the discharge.

PROGNOSIS.—In the generality of instances the patient gets quite well, either by ordinary care on his own part, or by medical treatment. In other cases, however, the development of some inherited disease manifests itself simply from the weak condition to which the patient is reduced. In some instances dementia or melancholia is induced, and the patient continues his impure habits even while under watch and ward in a lunatic asylum.

TREATMENT.—Should any local irritation appear sufficient to cause spermatorrhœa, it ought to be treated and removed if possible. A long prepuce should be cut off, balanitis cured, a varicocele treated, or rectal irritation removed. To prevent masturbation many plans have been tried, such as the application of iodine to the penis, or touching the parts with caustics, which, by the pain they cause, prevent the patient meddling with the organ. These measures, or such as these, combined with encouragement from the medical attendant, and resolution on the part of the patient, will help towards a cure. Local tenderness in the urethra may be relieved by counter-irritation to the perinæum, or by applying caustics directly to the tender surface, either in substance or in solution. The solution chiefly used is one of nitrate of silver, varying in strength from five to sixty grains to the ounce, and it is best applied by the silver syringe-catheter, the flexible tube, or through the urethroscope. The patient's digestion and impaired physical and mental condition must be looked after. Stomachic and nervine tonics, such as gentian, strychnine, phosphates, and iron, are the most useful,

and must be given for some time. To allay irritability of the genital organs, the bromides and belladonna may be given, separately or in combination. For hyperæsthesia the extract of belladonna, in half-grain doses morning and evening, is especially useful; it may be given with the tonics recommended above. Should anæsthesia of the urethra and genitals exist, galvanism has been tried and has proved successful. The patient should sleep on a hard bed, and get up the moment he wakes. A separate bedroom should be disallowed. Cold hip-baths morning and evening, rectal injections of cold water, walking exercise, and mixing in company as much as possible, are useful adjuvants to treatment.

JAMES CANTLIE.

SPEZZIA, Bay of, in Central Italy.—A calm, moist, moderately warm, equable winter climate. Mean winter temperature, 50° F. See CLIMATE, Treatment of Disease by.]

SPHACELUS (σφάκελος, gangrene).—The dead mass resulting from the process of gangrene. See GANGRENE.

SPHENOIDAL SINUSES.—See NOSE, Diseases of.

SPHINCTERS, Disorders of.—SYNON.: Fr. *Troubles des Sphincters*; Ger. *Störungen der Schliessmuskeln*. See DEFÆCATION; and MICTURITION.

SPHYGMOGRAPH, The (σφυγμός, the pulse; and γράφω, I write).—An instrument devised to record the character of the pulsations in an artery.

The tracing is called a *pulse-tracing* or *sphygmogram*, and consists of a series of curves, varying in number and form according to the frequency and the characters of the pulse. See PULSE.

The tracing is generally taken from the radial artery. The wrist is over-extended, and fixed steadily, while a small pad is applied accurately over the artery as it passes by the styloid process. The up-and-down movements of the pad are recorded on an enlarged scale by a style on a smoked paper, which is made to travel uniformly by clock-work. There are two forms in use, Marey's, which is superior but is cumbersome, and Dudgeon's, which is handy, but has the disadvantage that the needle is so light that it allows an excessive oscillation with a sudden pulse. The pad is kept applied over the surface of the artery by means of a spring; and the pressure is varied until the tracing with the greatest amplitude is obtained, when the clock-work is started. It is important to choose the right pressure, although, taken in this manner, it does not correspond to the arterial pressure. It is unfortunate that the sphygmograph has fallen out of use, as, within certain limits, it has great value. It enables the variations in the pulse to be recorded from time to time, and it allows the character to be recorded and discussed at leisure. Tracings taken with different varieties of instruments are not comparable, as the impulse-wave, due to the sudden impact of the wave on the pad, varies with the construction of the instrument. It is necessary, therefore, to be familiar with the sort of impulse-wave different pulses produce before discussing a tracing. In a Dudgeon's instrument the impulse-wave, in proportion to the tidal, is higher than with Marey's.

The next difficulty is that the tracing is the result of the combined movements of the artery and the venæ comites, and hence, when there is forced expiration, the height of the tracing is markedly increased, and the amplitude diminished, while with forced inspiration there will be a great fall in the height of the tracing, especially if forced expiration precede this. If these three facts be borne in mind much of the criticism as to the uselessness of the sphygmograph disappears. There is much to be learnt from studying sphygmograms which cannot be apprehended otherwise.

In taking a tracing it is necessary to keep the arm and the instrument still; to have the pad exactly on the centre of the vessel, or the up-stroke will be oblique, the top rounded, and the height deficient; and to choose such pressure as will give the tracing with the maximum amplitude. The papers should be well enamelled, and may be smoked by burning camphor; and when the tracing has been taken, and the necessary details, including the pressure applied, scratched on with a pin, it can be varnished by passing it through gum benzoin or Burgundy pitch (1 in 8) or through tincture of tolu.

A pulse-tracing shows at a glance the rate, rhythm and quality of the pulse, which can be studied at leisure. Irregularities, and especially inequalities, that escape the finger are registered, and indications as to the strength of the cardiac action and condition of the vessel-walls, which are most important for prognosis and treatment, are put on record. Both finger and sphygmograph detect sudden changes; but they both fail to appreciate the minor changes in tension and size, which are constantly going on with the various actions of daily life. These were unrecorded and unsuspected until other means of diagnosis were used (see PULSE). A recorded tracing differs from what can be felt with the finger, in the presence of the impulse-wave. This is produced by the suddenness with which the lever is tossed up. When this lever is light it is carried up higher than the pad by which it is started, and hence the fall in the trace, down again to the tidal wave.

The *tidal wave*, or *predicrotic*, or *first secondary wave*, as it is also called, is due to the impulsive tension in the arteries, following the increased pressure in the aorta and great vessels, from the reception of the ventricular contents. The *dicrotic*, or *great secondary wave*, is an oscillation of the blood-column, mainly, if not wholly, produced by the rebound of the blood from the closed aortic valves under the pressure of the aortic recoil. See DICROTISM.

A pulse-tracing (fig. 1) consists then in a *line of ascent*, *a* to *b*, which ends in the *impulse-wave*, *b*, and its size depends on the form of the instrument and on the suddenness of the pulse; from the impulse-wave the tracing falls slightly, till it is again raised by the *tidal wave*, *c*. After the tidal wave a more marked descent occurs, called the *aortic notch*, *e*, and the line again rises, into the *dicrotic wave*, *d*. The *line of descent*, *b* to *a'*, is thus broken by two waves and two notches. The two waves have already been described; of the two notches one precedes the tidal wave; while the aortic notch, preceding the dicrotic wave, marks the fall of pressure in the arteries antecedent to the closure of the aortic valves. The moment these valves are closed the line of descent rises again. It is the bottom of this notch, marking as

it does the closure of the aortic valves, which points out the termination of the ventricular systole. The remainder of the line of descent corresponds with

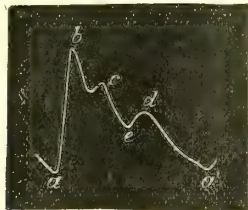


FIG. 1.—Typical Pulse-tracing.—*a* to *b*, line of ascent; *b* to *a'*, line of descent; *b*, impulse-wave; *c*, tidal wave; *d*, dicrotic wave; *e*, aortic wave.

the diastole of the ventricle, and is sometimes broken by smaller waves.

The pulse-tracing is modified in its chief features by the state of arterial fulness or tension. When the tension is high (fig. 2) the line of ascent is less

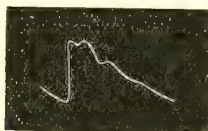


FIG. 2.—Tracing of Pulse of High Tension.

lofty; the tidal wave is large, nearer to, and sometimes blended with the small impulse-wave; the aortic notch is shallow, and high in the line of descent; the dicrotic is not much developed; and the line of descent is gradual. When the tension is low (fig. 3), the line of ascent is lofty; the

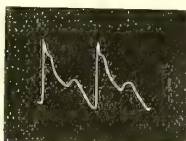


FIG. 3.—Tracing of Pulse of Low Tension.

summit-wave distinct; the tidal wave small; the aortic notch deep and low; the dicrotic highly developed; and the line of descent sudden. These modifications are interfered with if the normal elasticity of the arteries be lost, as in arterial degeneration.

INDICATIONS.—The evidence yielded by the sphygmograph mainly concerns: (1) The mode of the heart's contraction. (2) The condition of the peripheral circulation. (3) The state of the arteries and their coats. (4) Valvular diseases of the heart.

1. The Mode of the Heart's Contraction.—The sphygmographic tracing shows this by the line of ascent. When the heart-muscle acts suddenly and vigorously, the line is vertical and lofty, and terminates in a pointed impulse-wave. Unless the vessels are over-full of blood, there follow well-marked tidal and dicrotic waves. On the other hand, when the heart's contraction is feeble the line of ascent is less vertical and lofty; the impulse-wave is less distinct; the tidal and dicrotic waves are smaller, or the former is blended with the summit wave. The pulse, moreover, unlike the pulse of a vigorous ventricle, is easily

obliterated by pressure. A note of the pressure at which the largest trace is obtained should always be made, as it enables the observer to compare results at different times.

2. The Condition of the Peripheral Circulation.—The easy or difficult passage of the blood through the arterioles, causing a low or high pulse-tension, is roughly estimated by the pressure required to develop or to obliterate the tidal and dicrotic waves. Obstructed peripheral circulation is manifested by increase of the tidal wave, diminution of dicrotic, and lessened height of line of ascent (fig. 4). The heart, apart from febrile or nervous

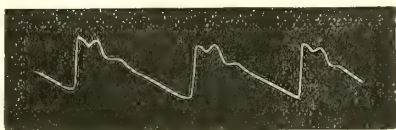


FIG. 4.

excitement, empties less suddenly under these conditions. On the other hand, in easy and quick peripheral circulation, such as occurs in fevers, the sudden heart-contraction causes a more vertical and higher line of ascent, the summit-wave is exaggerated, the tidal wave lessened, and the

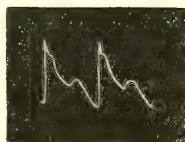


FIG. 5.

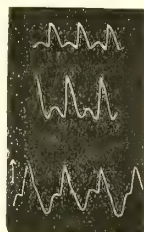


FIG. 6.

dicrotic fully developed (fig. 5). In such conditions the fully dicrotic and hyperdicrotic waves are recorded (fig. 6). The rapid onflow of blood is, moreover, shown by the more sudden fall of the line of descent. It is by the comparative study of the waves, and the pressure required to record them best, that we obtain valuable indications as to heart-strength and pulse-tension in acute diseases, and in the earliest stages of some chronic affections.

3. The State of the Arteries.—There are three chief conditions of the arteries that modify the pulse: (a) The state of the muscular coat; (b) degenerative conditions of the arterial walls; and (c) the presence of aneurysm.

(a) When the muscular coat is contracted the artery imparts to the finger a hard, wiry sensation,

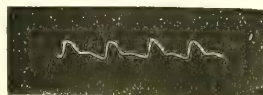


FIG. 7

which shows in the tracing by a short line of ascent, and the blending of the summit and tidal waves in an oblique line of descent, scarcely broken by dicrotism (fig. 7). In the opposite condition of relaxed arterial coats the dicrotic and summit-waves are enlarged and the tidal waves lessened.

These changes can be experimentally illustrated by the application of cold and heat to the surface of the body. The cold and hot stages of ague also show the two states.

(b) When the walls of the vessels have degenerated they become more or less rigid, so that the modifying influence of their elasticity on the blood-pressure is lost. The impulse-wave in the radial artery consequently approaches more to that imparted by the heart's systole. In these conditions the pulse often beats visibly, so that we are prepared for the amplitude of the tracing. The tidal wave is large, nearer to, and often blended with the impulse-wave, while the dicrotic is badly marked. The presence of these peculiarities often

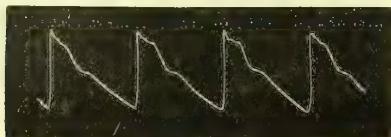


FIG. 8.

leads to the early diagnosis of unsuspected atheroma of the great vessels (fig. 8).

(c) When the sac of an *aneurysm* is seated on a main trunk after its origin from the aorta, it acts as a diverticulum, and so modifies the pulse-form by rendering the line of ascent oblique, diminishing or abolishing the impulse-wave, modifying the dicrotic, and more or less converting the down-tracing into a sloping line. The finger recognises these changes as a slow feeble pulse. Such peculiarities in the left radial artery are produced by an aneurysm of the left subclavian, or in the right radial by innominate aneurysm. When the aneurysm is connected with the thoracic aorta in its ascending portion, there is frequently a dissimilarity between the two radial pulse-traces, which is persistent, one being smaller than the other, more vibratory, or more easily obliterated by increased pressure. The pulse usually more affected is the right, as the aneurysm tends to implicate the innominate artery. In aneurysm of the transverse portion of the arch the left pulse is more commonly diminished in force and amplitude, and is also retarded (figs. 9 and 10). In aneurysms of the

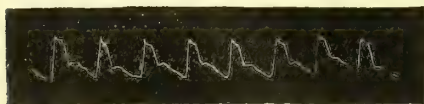


FIG. 9.—Right Radial Tracing.

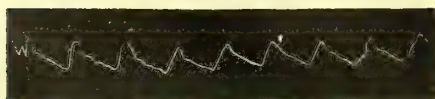


FIG. 10.—Left Radial Tracing.

descending thoracic and abdominal aorta, the dicrotic wave is often much increased in both pulses, while the right radial yields usually the more normally developed trace.

These signs may be more or less simulated by the pressure of tumours on the arterial trunks, or by their partial obstruction by clots. The sphygmographic signs of aneurysm, therefore, require to be confirmed by the use of the ordinary means of

diagnosis. In some cases, however, the pulse-tracings alone suffice to indicate the lesion and its seat.

4. Valvular Diseases of the Heart.—Valvular diseases of the heart generally influence the pulse-tracing. In *aortic regurgitation* this is strikingly seen (fig. 11). The strong, dilated ventricle discharges suddenly an increased charge of blood into a lax aorta, and consequently there is a lofty line of ascent, ending in a pointed impulse-wave. The tidal wave is small in proportion; and the dicrotic, which occurs lower than normal in the line of descent, is generally much diminished, on account of the leakage into the ventricle interfering with the rebound of the blood-column from the closed valves. The dicrotic wave, thus starved, is followed by a rapid fall in the tracing, showing the quick emptying of the artery. It is this contrast between the height of the summit-wave and the rapid fall of the tracing, unbroken by any *sustaining* wave, that gives the pulse its splashing and collapsing character. These features, the small dicrotism and the rapid fall, indicate the amount of regurgitation. In some cases of this valve-lesion the tracing shows a well-marked tidal wave and a fairly developed dicrotic wave; and increased pressure by the spring of the sphygmograph, instead of obliterating the tracing as usual, shows that there is a fair amount of tension. Such features commonly occur in older persons, in whom the valve-defect is small and due to atheroma, and not to rheumatism, and the pulse-form is modified by the addition of the characters of the pulse of degenerated arteries. When such features are observed in rheumatic cases, and the tracing nearly resembles the normal form, they point to perfect compensation and small valve-defect. The characters are much accentuated when the arm is raised.

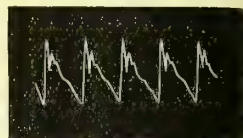


FIG. 11.

In *aortic stenosis* the tracing will indicate the amount of the lesion. When the narrowing is extreme the impulse-wave is lost, the line of ascent becomes oblique and gradual, and the pulse is felt to be slow, i.e. the vessel distends slowly. More commonly a break in the line of ascent marks the position of the impulse-wave, while above it rises the large tidal wave, due to the feeble impulse (fig. 12). In cases in which the obstruction is less in degree, the summit of the tracing may be forked by a sharp division between the impulse- and tidal waves. The dicrotic wave is lessened. A small impulse-wave, followed by an exaggerated tidal wave, arising from a strong and often dilated ventricle, pushing a large blood-wave gradually into the arteries through the narrowed aortic orifice, are the characteristics of this lesion when pure. When it is associated with aortic regurgitation, the large tidal wave is still pronounced.



FIG. 12.

Mitral-valve lesions, which are less immediately connected with the arterial blood-movement, present less decided characteristics. In *mitral regurgitation* the tracing is often of the normal outline, when the compensation is fairly perfect. In some cases, in addition to great rapidity, the pulse is small and flabby, in striking contrast

the vigour of the impulse (fig. 13). The line of descent is sloping, and the tidal and dicrotic waves are only defined. In some cases, where compensation has failed, great irregularity is the chief feature of the tracing, a series of small, ill-developed pulsations being succeeded by large and well-formed pulsations. On analysis, the series of small, ineffectual pulsations correspond to inspiration, and the smaller and more vigorous ones to the respiratory pause. In other conditions similar irregularities are produced by the same influences. Burdon Sanderson, who first referred these irregularities to their cause, says: 'The mechanical effect of inspiration



FIG. 13.

to augment the quantity of blood contained in the pulmonary circulation, and hence to increase the frequency of the contractions of the heart. This increased frequency depends on the distended state of the auricles, in consequence of which the ventricles contract more rapidly during their period of relaxation. In this way the length of the diastolic pause is diminished, and the hurried action of the heart is satisfactorily accounted for; but the question still arises, Why are the rapid beats which occur in inspiration also ineffectual? It is due to the tracing being formed by the combined movements of artery and vein. With inspiration the veins empty and the pulse-trace falls in consequence.

Similar peculiarities are noticeable in tricuspid regurgitation and dilated heart.

In *mitral stenosis* the sphygmographic evidence is very important. The pulse-tracing shows irregularity in the line of descent, which often is greatly prolonged through a missed pulsation—a true intermission in the beat, and sometimes broken by the interpolation of a small, abortive pulsation (fig. 14)—

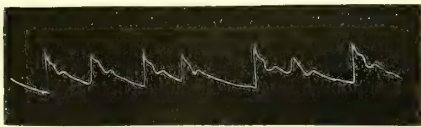


FIG. 14.

In the former, when the auricle has failed to contract, the latter when the ventricle fails, although the auricle has done so. The effect of an excess of digitalis in cases of mitral stenosis is often to produce a *pulsus bigeminus* in which only every second beat reaches the wrist. The abortive pulsations are due to over-distension of the auricle, causing premature auricular contractions, which propagate themselves to the ventricle, and so produce a ventricular contraction on a small charge of blood. They mostly occur during inspiration, from the causes above mentioned. The rhythmical relations between the contractions of the auricle and the ventricle are thus disturbed, and hence the features just described.

These are the special characters of the pulse of mitral stenosis. In some cases, where the stenosis is not great and the compensation perfect, the pulse is regular in time and form, or nearly so; but in these cases characteristic irregularities can often be produced by vigorous exertion.

Finally, in therapeutical investigations the sphygmograph is indispensable as a means of discovering the influence exerted by a drug on the state of the vessels, on the condition of the peripheral circulation, and on the vigour of the heart. The modifications in the form of the pulse-tracing above described enable the investigator to estimate these effects.

B. WALTER FOSTER.

G. NEWTON PITT.

SPINA BIFIDA (*spina*, the spine; and *bifida*, cleft).—SYNON.: Fr. *Hydrorhachis congénitale*; Ger. *Rückenspalte*.

DEFINITION.—A congenital malformation, in which there is a sac-like protrusion of the spinal membranes through a cleft in the neural arches of one or more of the vertebrae.

ETIOLOGY.—The malformation is due to an arrest of development of the spinal cord and vertebral canal, whereby the laminae fail to unite to form the spinous processes.

ANATOMY.—Spina bifida may occur in any part of the vertebral column, but is most frequent in the lumbo-sacral region, where the laminae are latest to unite.

Three chief forms are described: Spinal meningocele, Meningo-myelocele, and Syringo-myelocele.

1. The *spinal meningocele* consists of a protrusion of the dura mater and arachnoid blended together and contains cerebro-spinal fluid. The cord and nerve-roots remain in the spinal canal. This is a rare form.

2. The *meningo-myelocele*, the most common form, consists also of the dura mater and arachnoid, but contains, in addition to cerebro-spinal fluid, the spinal cord and nerve-roots. The cord can be traced as a thin band over the posterior part of the wall of the sac, some of the large nerve-roots given off from the cord running across the interior of the sac to re-enter the spinal canal, and thence passing out of the intervertebral foramina in the normal manner.

3. The *syringo-myelocele* is the most rare form. The central canal of the cord is greatly distended with fluid, the expanded cord being thus spread out as a lining to the sac, with which it is intimately blended. The nerve-roots in this form pass through the walls of the sac to their destination.

The coverings of the sac may be healthy skin, but more commonly normal skin is found only at the sides, the central portion consisting of thin bluish or bluish-white congested membrane. At the lower part of the sac, in the case of a syringo-myelocele, a slight depression is sometimes seen at the spot where the spinal cord terminates. At the bottom of this depression, which is known as the umbilicus, a minute foramen, through which cerebro-spinal fluid may ooze, at times communicates with the central canal of the cord. In some cases of spina bifida there is no protrusion, but the skin, membranes, and cord blended together, and covered by a tuft of hair, form a slight depression between the cleft spinous processes. This is known as *spina bifida occulta*, and is not infrequently associated with paraplegia, contractures, or deformities of the feet.

CLINICAL CHARACTERS.—The common form of spina bifida gives rise to a swelling of globular or ovoid shape, with its long axis vertical, and varies in size from a walnut to the foetal head. It is situated in the middle line of the back, and nearly

always in the lumbo-sacral region. It may be covered with healthy skin, but more commonly the central portion of the tumour consists of a bluish-white translucent membrane. The swelling becomes tense when the child is made to sit up or cries, or when pressure is made on the anterior fontanelle; but can be made less tense by direct pressure on the sac, and when the child is held up by the legs or placed in the prone position. The gap between the laminae can generally be distinctly felt. In the infant the anterior fontanelle protrudes when pressure is made on the tumour.

DIAGNOSIS.—The congenital origin of spina bifida will serve to distinguish it from a new-growth developed subsequently to birth. From other congenital tumours it may be known by its translucency, increase of tension on straining, situation in the middle line, and, when this can be felt, by the gap between the laminae. The diagnosis of the various forms from one another is not always easy. A syringo-myelocoele can hardly be diagnosed from a meningo-myelocoele; but opacity in the middle line at the situation of the spinal cord and nerve-roots, a dimple where the end of the cord is attached to the sac, the existence of a vertical furrow and the presence of paralysis, contracture or deformity of the lower limb, and want of control over the bladder and rectum will help to distinguish these forms from a simple meningocele, which is usually translucent all over, of a smooth contour, and unattended by paralytic symptoms.

COMPLICATIONS.—Spina bifida may be associated with hydrocephalus, meningocele or encephalocoele, club-foot, paralysis or contracture of the lower extremities, paralysis of the bladder and rectum, and imperforate anus. In rare instances the whole spinal canal may remain partially or completely unclosed; or there may be other congenital malformations incompatible with life. See **MONSTROSITIES**.

COURSE AND TERMINATIONS.—The majority of cases of spina bifida terminate fatally, often within a few days or weeks of birth; the child dying from marasmus, or from spinal meningitis and convulsions, frequently preceded by rupture of the sac and the escape of the contained cerebro-spinal fluid. When the fluid only oozes away gradually, spontaneous and complete cure may rarely occur; the tumour contracting to a small nodule, and the aperture in the canal closing more or less completely. When the cutaneous covering is thick and normal, the tumour may gradually decrease in size as age advances, or it may increase in size without material inconvenience up to the adult period of life, attaining the size of a child's head, or even larger dimensions.

PROGNOSIS.—This is unfavourable when the tumour is of large size at birth, and is covered only by a thin membrane, or has a broad base; and this is more especially the case when the spinal cord and large nerve-roots are contained in the sac, and the lower extremities and bladder and rectum are paralysed. When the base of the tumour is narrow, and its cutaneous covering thick and normal, the prognosis is more favourable, especially if the malformation be situated over the sacrum.

TREATMENT.—Whatever the variety of spina bifida, where the sac is covered by a thick pad of skin and shows no tendency to increase, it is advisable simply to protect it with a suitable bandage and shield, since it will probably give rise to no in-

convenience and possibly shrink with age. Where the sac is of large size, merely covered by a thin membrane, and contains in its walls the expanded spinal cord; where the cerebro-spinal fluid is oozing away, or where there is extensive marasmus, or paralysis of the lower limbs, bladder, or rectum, no treatment is of any avail, and the child will probably die in a few days. Suspension of the patient by the legs in an obliquely prone position will stop the leakage, and if the sac is kept aseptic the child may survive for some weeks or even in rare cases recover, the closure of the leak being followed by shrinkage of the sac. The treatment by pressure, tapping, and injections is not satisfactory, though up till recently injection with Morton's iodo-glycerine solution was perhaps the favourite procedure, and was attended with the best results. Now, however, in all suitable cases most surgeons excise the sac since a radical cure is in this way obtained. An elliptical incision is made on either side of the sac through the healthy skin, the two skin-flaps raised, and the sac with its central membranous covering excised, and the neck ligatured or sutured in such a manner that the line of skin-sutures does not correspond to the ligatures or sutures of the neck. In the meningo-myelocoele, after the skin-flaps have been raised, the cord and nerve-trunks are carefully dissected from the sac-wall and replaced in the spinal canal; or, if very adherent to the sac, strips of the sac-wall are left attached and replaced with the cord and nerves. The membranes at the neck of the sac are now drawn together by delicate silk sutures, and the skin-flaps united in the way described above. During the operation the patient should be in the prone position, with the pelvis well raised on pillows so as to avoid as much as possible the draining away of the cerebro-spinal fluid. The strictest antiseptic precautions should of course be taken. Excision, as a rule, should be delayed till the child is a few weeks old.

W. J. WALSHAM.

SPINAL ACCESSORY NERVE, Diseases of.—The upper medullary fibres of the spinal accessory nerve emerge from the surface of the medulla oblongata below the pneumogastric. They arise from a column of nerve-cells adjacent to the nucleus of the hypoglossal, and continuous with the nucleus of the pneumogastric. Their fibres join the latter nerve and innervate the movements of the palate and larynx. Their share in the nerve-supply to the pharynx is undecided, but it is certain that the levator palati is supplied by them. Paralysis of the vocal cord, tongue, and palate is occasionally due to disease at the surface of the medulla in this region, and the fact that stimulation of the roots of this nerve causes movement of the palate has been proved experimentally by Horsley and Beever. The lower, spinal fibres of the nerve emerge from the side of the cord as low as the sixth or seventh cervical nerves, having passed through the substance of the lateral columns, and arising from the anterior cornua, in common with the motor fibres of the upper cervical nerves, which supply muscles associated with those to which this nerve goes. The spinal part ascends through the foramen magnum, and is connected with the bulbar portion for a short distance; the two parts then separate, the latter joining the pneumogastric, the former passing to the neck, and supplying the sternomastoid and the upper part of the trapezius. The double relation

of the nerve is thus correctly expressed in its name. The bulbar fibres alone are 'accessory' to the pneumogastric; the 'spinal' fibres are strictly part of the motor cervical roots.

1. **Paralysis.**—**ÆTIOLOGY.**—The nucleus of origin of the nerve may be diseased owing to slow degeneration of the motor cells, as in progressive muscular atrophy and chronic bulbar paralysis, and also in syringomyelia and central growths that spread from the cervical region to the medulla. The nucleus of origin of the bulbar portion may be damaged by acute processes, especially softening (acute bulbar paralysis); and the grey substance from which the spinal fibres arise may be involved in acute poliomyelitis (infantile spinal paralysis). The roots of the nerve are sometimes damaged by injuries, such as fracture or dislocation of the upper cervical vertebrae; by narrowing of the foramen magnum; by tumours external to the cord; and especially by meningitis, syphilitic or simple, in this region. The spinal part of the nerve, from its long course, is especially liable to suffer. The nerve is rarely injured in fractures of the skull. The causes of paralysis of the vagal portion, after its junction with the pneumogastric, have been considered in the article on diseases of that nerve. The spinal part, in its course to the muscles, may suffer in rare cases from rheumatic inflammation, or from injury; may be compressed by enlarged glands; or implicated in abscesses in its neighbourhood.

SYMPTOMS.—Paralysis of the spinal accessory may be complete, when the disease involves the nerve where both parts are united, but is much more commonly partial, on account of the extensive origin of the spinal portion, and the early separation of the two divisions. The symptoms indicating disease of the accessory part of the nerve, as loss of movement of the vocal cords, are described in the articles on the pneumogastric nerve and on diseases of the larynx. The paralysis of the palate, which is so often associated, is recognised by the defective movement in phonation. See PALATE, Paralysis of.

The loss of function of the spinal portion of the nerve is shown by paralysis of the muscles which it supplies—the sternomastoid and trapezius. Unilateral palsy of these muscles does not affect the normal posture of the head, but when behind the vertical position it cannot be rotated to the opposite side. Paralysis of the trapezius, which may occur alone if the disease of the nerve is behind the sternomastoid, is almost confined to the upper part of the muscle, that proceeding from the occipital bone to the clavicle. The middle part of the muscle receives a sufficient nerve-supply from the cervical nerves to prevent conspicuous paralysis or wasting, although the fibres of the spinal accessory can be traced almost to its lower border. The loss of the upper part alters the contour of the neck, and the shoulder is not raised in deep inspiration. The shoulder can, however, still be elevated voluntarily, this movement being effected by the upper and middle part of the muscle. Abduction of the arm by the deltoid is interfered with, on account of the loss of the support afforded by the upper part of the trapezius; and the supplemental action of other muscles causes a slight rotation of the scapula. If the nerve or its origin is damaged by an acute process, the paralysed muscles undergo wasting, which is usually rapid, and is accompanied by the reactions which characterise nerve-degeneration.

PROGNOSIS AND TREATMENT.—The prognosis and treatment of paralysis of the spinal accessory nerve are those of the morbid process causing the paralysis (see especially PROGRESSIVE MUSCULAR ATROPHY; LABIO-GLOSSO-LARYNGEAL PARALYSIS; and PNEUMOGASTRIC NERVE, Diseases of). In all cases, if the muscles waste and present loss of faradic irritability, voltaic electricity should be applied.

2. **Spasm.**—The muscles supplied by the spinal accessory nerve are frequently the seat of spasm, causing 'torticollis,' or 'wry-neck.' The spasm is due to an affection of the centres, probably in some cases those in the medulla, in others those in the cortex of the brain. This affection is described in a special article. See WRY-NECK.

W. R. GOWERS.

SPINAL CORD, Diseases of.—**SYNON.**: Fr. *Maladies de la Moelle Epinière*; Ger. *Krankheiten des Rückenmarkes*.—Though the spinal cord is commonly regarded as a single organ, yet it is one which is very composite in structure, and still more so in function. It is in part (1) a mere aggregate of connecting fibres between the body generally and the brain—that is, an accumulation of channels of conduction for sensory impressions of all kinds, both superficial and deep, on their way to the brain; and also for outgoing motor incitations from the brain to all the voluntary muscles of the body, as well as to those pertaining to the viscera and their ducts, and to blood-vessels. In part, however, the cord also consists (2) of a serial aggregation of more or less fused ganglia having to do with the execution of voluntary and all sorts of reflex actions; with the functional activity of organs; and with the nutrition of tissues.

Before the diseases of the spinal cord are dealt with seriatim, the following functions will be briefly considered:

Channels of Conduction, p. 1519.

Spinal Reflexes, p. 1520.

General Ætiology and Pathology, p. 1521.

Regional Diagnosis, p. 1526.

Channels of Conduction.—Although it is impossible within the space allotted to this article to enter in detail into the functions of the cord, it may be briefly stated that, with regard to the channels of conduction, both the direct cerebellar and the antero-lateral ascending tract convey impressions to the cerebellum; the former has been supposed to convey impressions of the muscular-sense order, and the latter, if it does not convey impressions of the same kind, may give passage to impressions of common sensibility serving as additional incitations to some of the complex reflex actions regulated by the cerebellum. On the other hand, the columns of Goll are also commonly supposed to transmit muscular-sense impressions, though the ultimate destination of these may be to the cortex in the Rolandic areas of the cerebrum. The evidence derived from syringomyelia makes it highly probable that painful and thermal impressions reach the cerebrum by way of the central grey matter, in accordance with the views of Schiff. As to impressions of touch and pressure nothing definite is known; the paths for these impressions may lie partly through the columns of Burdach, and partly through the grey matter of the posterior horns (Tooth), as the only other ascending tracts seem to be a series of short commissural fibres running

through the lateral columns, and serving to connect the nerve-cells at various levels of the posterior cornua with one another.

In addition to the crossed and the direct pyramidal tracts, there are two other columns seeming to contain outgoing fibres in each half of the spinal cord. The most important is known as the ‘antero-lateral descending cerebellar tract,’ which consists of fibres

is known that the posterior roots of the spinal nerves divide on their entrance into the cord into two main longitudinal branches, ascending and descending. Of these the ascending branch has been hitherto principally referred to, but the descending branch ought also to undergo degeneration. But the only descending degeneration in the posterior columns occurs in a small tract which is known (from its shape) as the ‘comma-tract.’ Mott has found that this degeneration occurs not only in sections of the cord, but also after section of the posterior nerve-roots, and that on each occasion it extends downwards for a limited distance only (half to three-quarters of an inch). It seems, therefore, most probable that this degeneration of the comma-tract represents the degeneration of the fibres belonging to the descending branches of the posterior roots.

Spinal Reflexes.—The reflexes of purely spinal mechanism which are of importance (by their presence, absence, or alteration) as indications of disease of the spinal cord in different longitudinal regions have been divided into (a) the superficial or skin-reflexes, and (b) the deep or so-called ‘tendon-reflexes.’

(a) *Skin-reflexes.*—The most important of these are tabulated below. The designation of the parts of the cord upon which they severally depend is based upon a very useful table published by Gowers.

These skin-reflexes vary much in different indi-

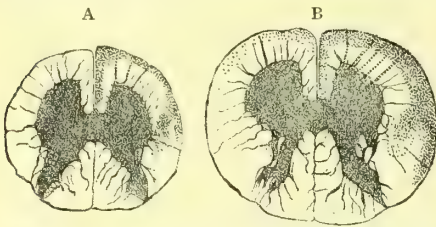


FIG. 1.—Sections showing the ‘Descending Cerebellar Tract’ in the Spinal Cord of the Dog, following upon extirpation of the left half of the cerebellum (Schäfer after Marchi); A, Lumbar Cord; B, Cervical Cord. The degeneration is in the antero-lateral column of the same side as the lesion, except in B, where there is a little degeneration on the opposite side.

that are connected with cells in the cerebellar cortex of the same side, and which undergo degeneration on removal of the corresponding half of the cerebellum (Marchi). These fibres form an extensive

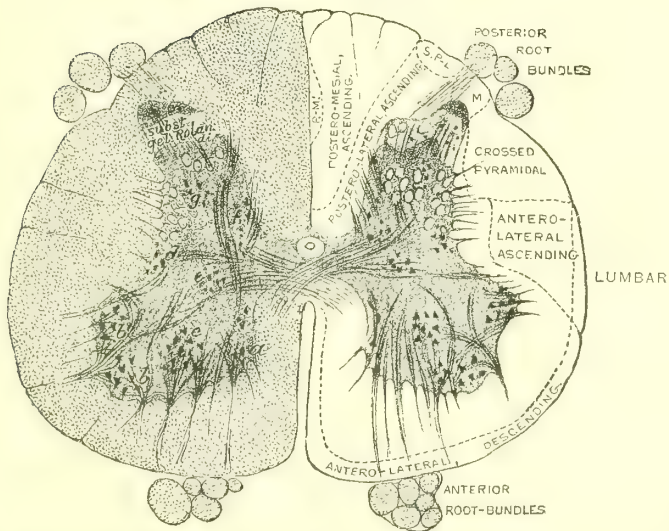
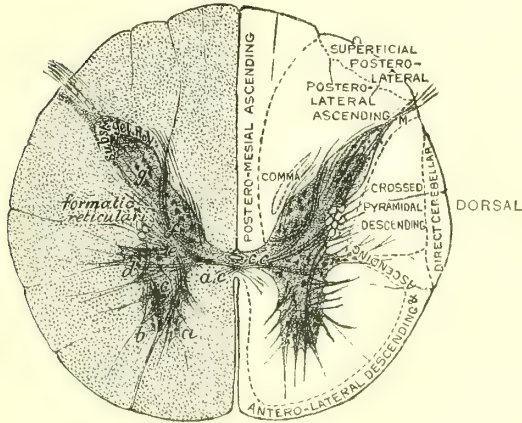
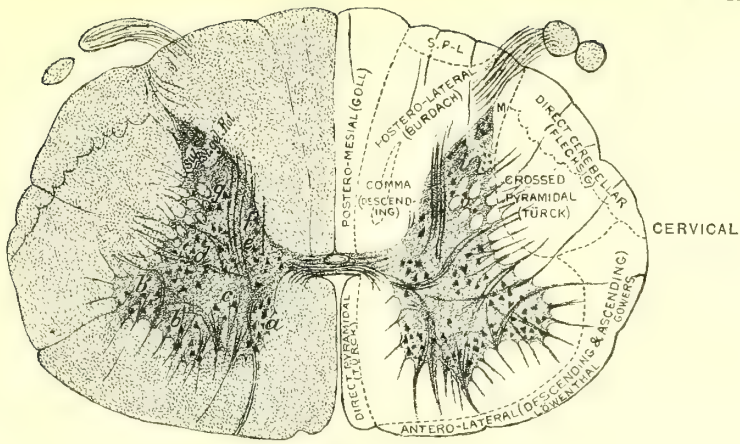
Name of Reflex	Mode of Excitation	Nature of Result	Level of Cord upon which Reflex depends
Plantar reflex	Tickling sole of foot	Movements of toes; of these and foot; or of these and leg	1st, 2nd, and 3rd sacral nerves (lower part of lumbar enlargement)
Gluteal reflex	Irritation of skin of buttock	Contraction of glutæi	4th and 5th lumbar nerves
Cremasteric reflex	Irritation of skin of upper and inner part of thigh	Drawing up of testicle	1st and 2nd lumbar nerves
Abdominal reflex	Irritation of skin of abdomen along edge of ribs, and above Poupart's ligament	Contraction of upper or of lower part of abdominal muscles	8th to 12th dorsal nerves
Epigastric reflex	Strokeing side of chest over 6th and 5th intercostal spaces	A dimpling of corresponding side of epigastric region (contraction of highest fibres of rectus abdominis)	4th to 6th or 7th dorsal nerves
Scapular reflex	Irritation of skin in interscapular region	Contraction of posterior axillary fold (teres), or of several of scapular muscles	6th or 7th cervical to 2nd or 3rd dorsal nerves

circumferential tract in the anterior three-fourths of the antero-lateral column, spreading inwards in front of the crossed pyramidal tract to reach the intermedio-lateral tract of the grey matter. The tract which is thus marked out is most extensive in the upper regions, and gradually lessens in the lower part of the cord, but can be traced almost to its termination. It embraces in the dog the part of the anterior column which in man is occupied by the direct pyramidal tract, and also the whole region of the tract of Gowers, the fibres of these two tracts being intermingled. Some of the fibres of the anterior roots also exhibit degeneration after removal of the cerebellar hemisphere, and are therefore probably directly continued from fibres of this tract. The same holds good for nearly all the efferent cranial nerves (Marchi). These degenerated fibres are numerous on the same side as the lesion, but a few occur in the opposite nerves.

The other tract formerly thought to contain outgoing fibres is now believed, with more probability, to belong really to the afferent system of fibres. It

viduals, as regards the facility with which they may be obtained. They are often more marked in children and in women than in men; though when the latter are of an irritable or nervous temperament, some or all of the skin-reflexes may in them be well marked even in conditions of health. It must be borne in mind, also, that the superficial reflexes are always distinctly exaggerated, when the corresponding regions of the skin are tender in association with related visceral disease (Head).

In cases where extensive transverse lesions exist, situated higher up in the cord than the nerves upon which any of these reflexes depend, such reflexes are commonly supposed to be exaggerated in intensity. This, however, is not the case where we have to do with total transverse lesions. The fact that this or that reflex exists shows not only that the afferent and efferent nerves, but that the path through the spinal cord at the corresponding level, is practically undamaged. It is not, however, necessarily true that absence of any of the reflexes is an indication of disease at the corresponding level



SECTIONS OF SPINAL CORD IN LOWER CERVICAL, MID-DORSAL, AND MID-LUMBAR REGIONS (SCALE 1:1).
ON THE RIGHT SIDE OF EACH SECTION THE CONDUCTING TRACTS ARE INDICATED.
COLUMN OF ANTERIOR HORN — (a) Mesial group of nerve-cells; (b) Lateral or ventro-lateral; (c) dorsal or dorso-lateral; (d) central group; (e) Lateral cell-column; (f) Middle cell-column; (g) Cells of posterior horn.

the spinal cord. It may be so; but it may also be that the disappearance of the reflex is dependent upon disease in some part of the afferent or of the efferent nerves, leaving the cord itself intact. Or it may also happen that the particular reflex is simply not to be obtained in the individual under examination. Or, again, with a complete transverse lesion of the lower cervical or in the upper or middle dorsal regions of the spinal cord in man all reflexes dependent upon lower portions of the cord, excepting perhaps the plantar, will (though this is contrary to usual belief) be found to be abolished. See SPINAL CORD, Special Diseases of: 9. Softening of.

Further, it must be borne in mind that in certain cases of hemiplegia (even where hemianesthesia does not co-exist) these skin-reflexes are often notably diminished or even abolished on the paralyzed side of the body; though the reverse condition of things will probably obtain in regard to the deep or 'tendon-reflexes' next to be considered. It will probably be found, hereafter, that this reversing effect upon the skin-reflexes is associated with the existence of lesions in special parts of the brain, and not with lesions in other localities, though such special sites are at present very imperfectly known.

Recently a modification of the plantar reflex in

reflex is often the earliest to appear in disease of the pyramidal tract. See BABINSKI'S SIGN.

(b) '*Tendon-reflexes*.'—Much discussion has taken place as to whether these are 'reflexes' at all, in the proper sense of the term. Into this question we do not propose to enter. The phenomena themselves, to which alone reference will be made, are chiefly two in number, namely, *ankle-clonus*, and that variously known as the *knee-phenomenon*, *patellar tendon-reflex*, *knee-reflex*, or *knee-jerk*. Corresponding phenomena are met with in the upper extremities in the form of 'wrist-jerk' (produced by a slight blow over the radial side of the wrist), and 'elbow-jerk' (from a blow upon the triceps tendon). These phenomena are increased or diminished under the influence of the same kind of conditions that cause increase or diminution of the 'knee-jerk,' so that no further special reference will be made to them.

There is a distinct difference in regard to the importance of the presence of the 'knee-jerk' and 'ankle-clonus' respectively. The 'knee-jerk' occurs in health, so that it is its absence, as well as its increase, which is of principal significance in certain diseases. 'Ankle-clonus,' on the contrary, is a phenomenon not to be obtained in a state of health, so that its presence was formerly thought by some to be a positive sign of disease of the spinal

Name of Reflex	Mode of Excitation	Nature of Result	Level of Cord upon which Reflex depends
<i>Knee-jerk</i> . . .	By striking patellar tendon with edge of hand or with percussion-hammer, while leg hangs loosely over fellow, or over forearm of operator. Also by striking quadriceps tendon, above patella	A single upward jerk of the leg and foot, slight or distinct	2nd and 3rd lumbar nerves
<i>Ankle-clonus</i> . . .	With knee extended or very slightly flexed, by pressing quickly and firmly against anterior part of sole of foot (so as to stretch calf-muscles) and then keeping up the pressure	A series of clonic contractions at the ankle-joint, continuing as long as the pressure is maintained, and instantly ceasing when it is relaxed If the condition is very highly marked it may spread to the whole limb, or even to that of the opposite side	1st to 3rd sacral nerves (lower part of lumbar enlargement)

association with disease of the pyramidal tract made known in 1896 by Babinski (and now often spoken of as 'Babinski's sign,' or 'Babinski's reflex?') has received much attention. The normal plantar reflex to slight irritation in healthy adults, and in children after learning to walk, is by *flexion* of the toes, whereas Babinski pointed out that in all such persons with lesions of the pyramidal tracts the corresponding response is by *extension* of the toes, especially of the great toe. An extensive investigation as to this sign upon a very large number of individuals by Walton and Paul (*Journ. of Nerv. and Ment. Dis.*, June 1900) has led them to the following, among other, conclusions: (a) in early infancy no constant or characteristic movement of the toes occurs, though extension is rather more frequent than flexion; (b) the Babinski reflex obtains in about 70 per cent. of hemiplegics and epileptics, and in approximately the same proportion of cases with disease involving the pyramidal tract in the spinal cord; (c) it is never present in health, and there is reason for doubting its existence in either functional or organic nervous or other disease not implicating the pyramidal tract; (d) this

cord. But this is a view which requires limitations—and limitations of such a kind as to deprive the manifestation of ankle-clonus of much of its diagnostic significance. It may exist, for instance, after one-sided fits dependent upon disease of the cerebral cortex; and, again, it may exist to a well-marked extent where the antero-lateral columns of the cord are pressed upon at a certain level, even though (as in the condition above referred to) no lateral sclerosis of the cord has been developed.

General Ætiology and Pathology.—The spinal cord itself may be damaged by disease invading it *from without*—that is, taking origin either in the bony canal or in the enveloping membranes; or it may be the seat of *intrinsic* pathological changes. As the former conditions may and do constantly produce functional derangements or actual structural changes of a secondary order in the cord itself, we must take cognisance of them here, so that the various peculiarities as to their occurrence may be made known and considered side by side with those pertaining to the different causes of disease of intrinsic origin,

from which they have to be distinguished at the bedside.

(a) **Extrinsic Causes.**

(1) *Stabs or bullet-wounds* may involve limited regions and parts of the spinal cord.

(2) *Fracture with dislocation of some of the vertebræ* (as results of severe falls or other mechanical violence) exists as an occasional cause of an associated paraplegia, produced by the crushing of, or pressure upon, the spinal cord. This is most apt to occur in the cervical region, though the dorsal and lumbar regions are, to a less extent, liable to similar accidents.

(3) *Tubercular caries of the vertebræ* may exist in any of the regions, and may or may not be associated with *angular curvature* in a corresponding portion of the spine. The paraplegia, or other result of interference with the functions of the cord, in the majority of cases of this disease, is not due so much to its compression by diseased bone, as to the irritation and subsequent compression of the cord by inflammatory products.

(4) *Cancer of the vertebræ* occurs either as a primary or as a secondary affection. Such a new growth may involve the dura mater or not, and as it grows it may at first irritate and subsequently compress the spinal cord itself.

Other diseases of the spine are rare as causes of disease of the spinal cord. Still aneurysmal erosion of the vertebræ with subsequent pressure upon the cord must not be forgotten, and very rarely an aneurysm bursts into the spinal canal. Exostoses and enchondromatous growths from the bones may also quite rarely compress the cord.

(5) *Cancer of the spinal meninges, or new-growths of other kinds* (see 21, SPINAL CORD, Tumours of), may also involve irritation, and subsequently compression, of the anterior or posterior nerve-roots, or of the spinal cord itself in one or other region. *Hydatids*, again, should be remembered as possible causes of spinal disease, especially where their existence has already been detected in the body, in other situations.

(6) *Hæmorrhage into or upon the meninges.*—See MENINGES, SPINAL, Diseases of.

The foregoing groups of causes of disease of the spinal cord give rise to sets of symptoms having a generic resemblance, because in each case *compression* acts upon the cord, or upon the spinal roots and cord, from without, in one or other direction.

(b) **Intrinsic Causes.**

(7) *Hæmorrhage* occurs with extreme rarity into the spinal cord. This is due, in the main, to the firmer texture of the cord, and to the greater abundance of supporting connective tissue around its blood-vessels, as compared with that surrounding the vessels of the brain. When hæmorrhage, of idiopathic origin, does take place into the spinal cord, it almost invariably occurs in the softest portion of the organ, namely, its central core of grey matter—and in this region it may extend for some distance upwards and downwards. As a result of falls or blows, also, hæmorrhage into the substance of the cord is a rare event; still, under these conditions, it occurs occasionally—mostly in association with laceration of the substance of the cord. Of this latter kind of lesion, resulting from a fall from a height of about twenty-five feet, the writer has recorded a remarkable instance, in which, although the cord was severely lacerated, there was

no external wound and no fracture or dislocation of vertebræ.

(8) *Embolism* occurs with great rarity in the spinal cord, and is still more seldom recognised when it does occur. This is due to the fact of the small size of the arteries of the cord, and, as compared with the frequency of embolism in the brain, to the absence among them of any large trunk, like the middle cerebral, coming off more or less directly from one of the great vessels arising from the arch of the aorta. Emboli are known to reach the brain much more rarely by way of the vertebral than by way of the carotid arteries; and the principal arteries of the spinal cord are either direct offsets from the vertebral (anterior spinal), or indirect branches from the same (posterior spinal)—the latter arising from the inferior cerebellar, which are twigs from the termination of the basilar artery. Apart from these vessels, the blood-supply of the cord comes from still smaller twigs, derived from the intercostal and lumbar arteries, which anastomose with and reinforce the anterior and posterior vessels, at intervals, along the whole length of the cord. All the principal vessels, small though they are, seem to anastomose freely with one another. Thus, even if embolism of the spinal arteries should occur, as it probably does very rarely, its effects would be diminished in importance, and obscured clinically as well as *post mortem*, by reason of the very small size of the vessels, and also by the fact of their not being 'end' arteries.

(9) *Thrombosis* would, however, be capable of occurring in diseased spinal arteries, as well as in those of other parts of the body. Subsequent observations may perhaps show that degenerative changes or endarteritis are particularly common in the spinal arteries, so that the occurrence of thrombosis in them would thereby be rendered all the more easy and likely to occur. Once set up, the process of thrombosis might easily spread in this network of small spinal vessels, and yet be very difficult to recognise in them except by the effects that would be produced upon the tissue of the spinal cord—namely, the production of softening. A similar process may also take place in the peculiarly tortuous network of *veins* which surrounds the spinal cord on all sides—perhaps even with more facility than in the veins of other parts—when general and other local conditions favour its occurrence. Ollivier, in fact, called attention to the probably natural slowness of the blood-current through the spinal veins, and to the multiplicity of causes which (owing to their influence upon respiration and cardiac action) tend still further to retard it—such as violent emotions or efforts, and all such diseases as greatly interfere with respiration or with the force and regularity of the heart's action. Ollivier adds that he has often seen in elderly persons fibrinous clots filling the veins of the cord, as well as those which are to be found on its nerve-roots.

Thus one of the common causes of ordinary degenerative softening, as it occurs in the encephalon, would also be operative in the cord.

(10) *White softening* of the spinal cord is, in fact, very common; often implicating its whole transverse area for a variable extent. It differs in no respect in its naked-eye or microscopical appearances from the process as it is met with in the encephalon. It is altogether unreasonable to assume, in accordance with current nomenclature, that this condition

is mostly a result of inflammation, and therefore to be spoken of as a *myelitis*, when the pathologists of our time have declared that the similar process in the cerebrum and cerebellum is mostly of degenerative origin.

(11) *Myelitis*.—The writer is, of course, far from denying that primary inflammation may involve areas of the cord, and entail 'softening' of its substance. He believes, however, that 'acute myelitis' is far more likely to occur as a secondary process, in connection with pressure upon and consequent irritation of some part of the cord, encroached upon by fractures or dislocations of the vertebrae, or otherwise wounded; also as an occasional sequela of tubercular vertebral caries, of the direct pressure made upon the cord by some meningeal tumour, or of hæmorrhage into its substance. The question rather is as to the causation of primary or idiopathic softening—whether this is, in accordance with common phraseology, to be ascribed to inflammation, or whether it is non-inflammatory and of thrombotic origin, as the writer believes. Yet he is also far from believing that *all* the secondary softenings met with in the spinal cord are necessarily of inflammatory origin. Many of these also are probably due to degenerative changes from pressure and thrombosis, rather than to inflammatory causes.

Processes of degenerative 'softening' are mostly brought about quickly, so that they would from a clinical point of view, in the main, correspond with what is commonly spoken of as 'acute myelitis.' As for 'chronic myelitis' (in the commonly understood sense of chronic softening), the writer believes that no such disease should be any longer described. Many 'softenings' are in a certain sense chronic, as, though they may be more or less abrupt in their onset, they tend to last long rather than to kill quickly. Again, other maladies which the older physicians would have ascribed to 'chronic myelitis' or 'chronic softening,' are now known to partake more of the nature of chronic indurations, and to have as their bases various processes of sclerosis.

(12) Processes of *sclerosis* are extremely common in the spinal cord. In nature they are overgrowths of the connective tissue of this organ, altogether similar to those occurring in other organs and tissues, under the name of 'fibroid substitutions' or 'non-inflammatory hyperplasias of connective tissue' (see FIBROSIS). Sclerosis occurs under various forms, and constitutes the basis of several distinct diseases, which are in all cases gradual and more or less slow in their onset, as well as in their progress. It may occur (a) as a *diffuse* general overgrowth (after the manner of a cirrhosis in other organs); (b) in the form of *fasciculi* or bands limited to particular columns of the cord (especially the posterior and the lateral); or (c) in an *insular* manner, so as to form islets of sclerosis, scattered altogether irregularly through the cord at different levels, as in 'disseminated sclerosis.' Sometimes overgrowth of the connective tissue is the primary event, in other cases it is sequential to an atrophy occurring in the nerve-elements themselves.

(13) Tissue-changes allied to these in their results or later stages, though they have a peculiar history and course of their own at the commencement, are the so-called '*secondary degenerations*' which occur in certain regions of the cord, as a result either of

some previous damage or injury to this organ itself, or as a sequel of brain-disease.

These 'secondary degenerations' illustrate facts originally made known by Waller, to the effect that when nerve-fibres are severed from the ganglion-cells from which they are outgrowths, the white substance of Schwann gradually breaks up in the course of seven to fourteen days, and undergoes a process of fatty degeneration, by which it is ultimately resolved into a multitude of fine granules and fat-particles. The white columns of the cord are composed of aggregations of nerve-fibres running parallel with one another, so that when one of these columns is cut across, or when the continuity of its fibres is interrupted by some severe lesion occurring in their midst, a process of 'secondary degeneration' manifests itself simultaneously in all the fibres thus damaged, extending upwards or downwards from the lesion according as the fibres cut across have afferent or efferent functions. Thus the result appears as one or more band-like tracts of degeneration, running upwards or downwards according to the extent and situation of the transverse area of the cord affected.

In order to deal as briefly with this subject as possible, it may be said that experience has hitherto shown that such band-like tracts of 'secondary degeneration' occur especially in each lateral half of the cord, in three situations, namely, (1) in the lateral columns; (2) in the inner portions of the anterior columns; and (3) in the posterior columns. The degenerations in the anterior columns, and in the posterior and median portions of the lateral columns, take place in a direction downwards from the site of section or lesion of the fibres, at whatever level the damage may chance to exist; while those in the posterior columns and in the superficial portions of the lateral columns take place in an upward direction, starting from the section or seat of destructive lesion by which these columns may be invaded.

The fibres that undergo the *descending degeneration* in the lateral columns (constituting the 'crossed pyramidal tract') are generally believed to be those which transmit volitional stimuli to the various voluntary muscles of the body, and which come into relation with motor cells in the anterior cornua at different levels. These different fibres are supposed to enter the lateral columns at the commencement of the spinal cord, passing into them, in fact, as a result of the 'decussation of the pyramids.' Thus, the motor tract continued downwards through the 'internal capsule' from one Rolandic area of the cortex, let us say the *left*, continues along the crus and through the pons on the same side; thence passing into the bulb a considerable proportion of its fibres decussate with their fellows, and thereby reach the right lateral column of the cord, down which they proceed as a compact group in the manner indicated. The remainder of the fibres of the left motor tract (those which do not decussate) pass down also in a compact body (the 'direct pyramidal tract') and occupy most of the inner half of the left anterior column.¹ Thus, if the whole of the left motor tract be seriously damaged or cut across in the internal capsule or at any point above

¹ Though this is the rule, yet it would appear from the observations of Flechsig that developmental anomalies are apt to occur, so that the relative proportion between the decussating and the non-decussating fibres is subject to much variation in different individuals.

the 'decussation of the pyramids,' we should have a small band of degeneration in the anterior column on the same side, and also a larger band of degeneration in the opposite (or right) lateral column (fig. 2, H)—that is, we should have the form of secondary degeneration associated with many cases of hemiplegia. But if there be complete section of or destructive disease involving the antero-lateral columns of one side of the cord itself, then we should have a band of degeneration in the anterior, as well as in the lateral, column of

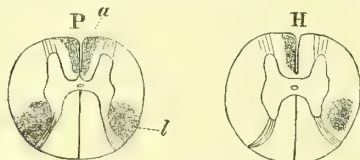


FIG. 2.—P. Showing descending areas of degeneration: *a*, in inner part of anterior columns; *l*, in lateral columns (mid-dorsal region). Case of *paraplegia*, from complete transverse softening in upper dorsal region.

H. Showing descending degenerations in case of *right hemiplegia*, from extensive softening of left 'internal capsule.' (Twice natural size.)

the same half of the cord. Or if either column be cut or damaged singly, then in such column a band of degeneration would be found extending downwards from the seat of lesion. Or if, as so frequently happens, we have to do with a total transverse lesion, represented, for instance, by a focus of softening extending through the whole thickness of the cord in the upper dorsal or in some other region (so that the patient suffers from complete paraplegia), we should then find large areas of secondary degeneration in each lateral column below, as well as smaller areas in the inner part of each anterior column (fig. 2, P). The areas in both situations become less extensive as they descend, and gradually wear themselves out in the lower part of the lumbar swelling. It was stated by Bouchard, and has been commonly repeated by succeeding writers, that the areas in the anterior columns do not appear beyond the mid-dorsal region; but this mode of termination, as the writer pointed out in 1867, is certainly not invariable.

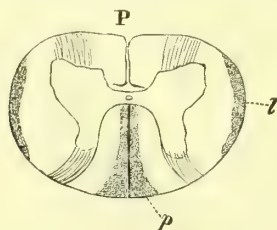


FIG. 3.—P. Showing ascending areas of degeneration: *g*, in columns of Goll; and *l*, along outer border of lateral columns, in middle of cervical swelling (corresponding with direct 'cerebellar tract' and the 'tract of Gowers'). Case of *paraplegia*, from complete transverse softening in upper dorsal region.

In such a case as that last cited, namely, one of paraplegia due to a total transverse lesion in the upper dorsal region, there would be found above the seat of lesion certain *ascending degenerations*—the principal of which would be situated in the posterior columns, though others, smaller and more recently recognised, are to be met with in the outer

part of the lateral columns (fig. 3). The ascending degenerations in the posterior columns are often strictly limited to the so-called 'columns of Goll.' Situated on each side of the posterior median fissure, they together constitute a median wedge-shaped patch, whose apex extends forwards to the commissure, and whose base is at the posterior surface of the cord. This band of degeneration

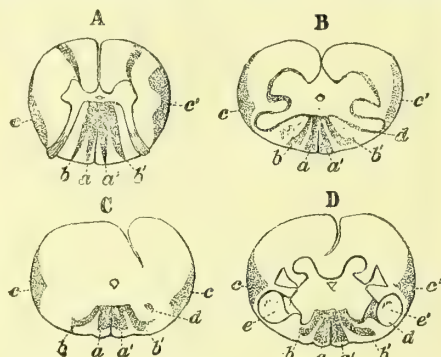


FIG. 4.—Peculiar areas of ascending degeneration, met with in a concussion-lesion of the Spinal Cord.

A, transverse section of the Spinal Cord, near the middle of the cervical region; B, transverse section through the cervical region of the cord, near lower border of medulla; C, section through lower part of medulla oblongata; D, section through medulla about $\frac{1}{4}$ " below the *Calamus scriptorius*.

The areas in the posterior columns (*a*, *a'*, *b*, *b'*) have a very peculiar disposition. The areas in the lateral columns in A are very unsymmetrical; that of the right side, anterior to *c'*, represents the lateral sensory tract.

reaches upwards to the bulb, where its fibres seem to terminate in the nucleus gracilis.

It seems clear, however, that, under certain conditions, the areas of ascending degeneration in the posterior columns may be differently arranged, and not completely limited to the 'columns of Goll,' since in a case with a lesion of some kind in the mid-cervical region (the nature of which is not known, because, unfortunately, this part of the cord was not preserved) the writer long ago found such areas as are represented in fig. 4 in the upper cervical region and in the medulla. The ascending areas of degeneration occupying the superficial portions of the lateral columns were traced by the writer upwards into the restiform bodies. These are areas which we now know to correspond with the subsequently described 'direct cerebellar tract'; while the 'tract of Gowers'—corresponding with the part of the areas anterior to *c'* in fig. 4, A and B—which has still more recently been described, also contains afferent fibres for the cerebellum, though they reach it by a different route. According to Schäfer, they can be traced upwards into the bulb and pons Varolii, while they eventually enter the cerebellum as part of the superior peduncle, passing mainly to the vermis. The fibres of this latter tract are intermingled with those of a part of the descending cerebellar tract (fig. 1), which is the only other long tract of degeneration existing in the spinal cord that has not hitherto been mentioned.

In these areas of degeneration, in addition to the changes already mentioned as occurring in the nerves themselves, other processes take place. There is, for instance, a very distinct but secondary over-

growth of the connective tissue throughout the diseased area, as well as an abundant development of large 'granulation-corpuses,' precisely similar to those met with in ordinary foci of softened nerve-tissue. The granulation-corpuses are closely packed among the meshes of the connective-tissue overgrowth and the atrophied nerve-fibres. In preparations which have been immersed in bichromates or in chromic acid, these corpuses do not become stained to nearly the same extent as the healthy nerve-tissues; hence the areas containing them remain pale, and are consequently to be traced with the greatest ease in spinal cords which have been immersed for a week or two in these fluids, though when they were in the fresh state no such areas may have been detectable, even on the most careful examination, by the naked eye. On the other hand, when sections through such degenerated areas of spinal cord are mounted in the ordinary way in Canada balsam, the granulation-corpuses become invisible, so that Tooth and other recent writers make no mention of the existence of granulation-corpuses in the areas of degeneration—the most obvious change then being the overgrowth of connective tissue, leaving, as Tooth says, 'loculi filled with homogeneous material not staining by Weigert hæmatoxylin.' This 'homogeneous material' in the loculi represents, as the writer believes, granulation-corpuses altered by the preparation of the specimens.

(14) *New-growths* in the substance of the spinal cord itself are not very common, nor, on account of the limitations of space within the spinal canal, do they ever attain a very large size, so long as they take the form of circumscribed growths. For this situation a growth equalling a hazel-nut in bulk would be esteemed large. In regard to the nature of the growth, this is, of course, a matter of purely pathological interest, since the clinical signs and symptoms which a growth in the spinal cord is capable of causing would not vary with its nature, but would be wholly dependent upon its situation, and its rate and manner of increase. *Cancer* occurs within the spinal cord almost solely as a secondary extension from a similar growth pre-existing in the dura mater or in the vertebræ, or possibly in more distant parts. In altogether exceptional cases it may occur primarily in the spinal cord. *Gliomata*, *sarcomata*, and *myxomata* may also occasionally be met with, either in pure or in blended types; and in the case of the former we may have infiltrating growths occupying a wide area in the central and posterior regions of the cord more especially, through a considerable portion of its length, such as occurs in syringo-myelia. *Tubercular* nodules are also apt to occur, either alone or in combination with a tubercular meningitis. *Syphilitic gummata* may likewise be found in the substance of the cord, though their presence in this situation is not so frequent as it is in association with the spinal meninges.

(15) *Atrophy with degeneration of ganglion-cells* is apt to occur as a secondary process with extreme frequency in portions of the grey matter of the cord which happen to be more or less implicated in other contiguous pathological changes. But in two or three distinct diseases the ganglion-cells or the anterior cornua in different parts of the cord are prone to be suddenly overtaken by an ætiologically obscure failure of nutrition, commonly supposed to be of an inflammatory nature, which speedily entails

an atrophy of the particular cells affected. This, for instance, occurs as the anatomical basis of 'infantile paralysis,' and of the similar form of paralysis now known to occur (though more rarely) in adults. In these diseases whole groups of contiguous and functionally related cells are affected simultaneously, and as the atrophy of the ganglion-cells progresses there is generally evidence of a secondary overgrowth of the surrounding neuroglia in the anterior cornua. To assume that this process is inflammatory in type, as the terms 'cornual myelitis' or 'acute anterior polio-myelitis' imply, seems to the writer somewhat questionable. Inflammation does not commonly limit itself to individual tissue-elements, and the slight overgrowth of the contiguous neuroglia may well be a secondary process of simple hyperplasia. This latter process is indeed less evident where, as in 'progressive muscular atrophy,' the initial and mysterious atrophy of individual ganglion-cells occurs more slowly and more sparsely. Cells, here and there, in particular anatomical groups, undergo in this affection the atrophic process, leaving others around them for a time as healthy as ever. Yet, as the disease progresses, the ranks of the healthy cells become gradually thinned in an altogether irregular manner; and this atrophy of nerve-cells, as it occurs, speedily entails, for reasons to be set forth in the next section, a corresponding atrophy of functionally related muscular fibres.

Trophic Relations between different Tissues and different Parts of the Spinal Cord.—Irritation of the posterior cornua, or of the posterior roots of the spinal nerves, may give rise to various pustular or vesicular eruptions in related portions of the skin, often associated with neuralgic pains in these same regions. In other cases, with lesions in some parts of the grey matter, ulceration or actual sloughing of certain related tracts of skin is easily determined—especially under the combined influence of continued external pressure and frequent irritation from urine or fæces, as in some cases of paraplegia. See p. 685.

Degeneration or destruction in any way of the great ganglion-cells of the anterior cornua, or of the anterior roots of the spinal nerves (either within or outside the cord), gives rise, in the course of two or three weeks, to atrophy of the muscle-fibres with which such atrophied cells or nerve-roots are in relation. We thus get an atrophic paralysis, associated with the electrical 'reaction of degeneration.'

Certain diseases affecting the grey matter of the cord (in ways and sites which cannot be precisely defined) are also commonly supposed to be associated with chronic diseases of the joints; it seems, however, to the writer that these articular changes may be due rather to a co-existing peripheral neuritis involving certain nerves proceeding to the joints. Sometimes comparatively unimportant, these joint-changes lead in other instances to great atrophy of the articular ends of the bones, and possibly to dislocation with utter destruction of the joint, as in some cases of locomotor ataxy. Atrophy, with brittleness of bones, may also be met with in the same or in allied cases.

The fact of the existence of these trophic troubles in association with such lesions may be admitted wholly irrespective of the explanation of their pathogenesis. Whether they are due to defective or altered influences transmitted by ordinary motor and

sensory nerves in relation with such tissues, or to altered influences through certain purely hypothetical 'trophic nerves,' lies altogether outside the fact of the mere co-existence of the several trophic troubles with the several lesions—which is the point of more immediate interest for the practitioner of medicine.

Regional Diagnosis.—We already possess a number of valuable clinical data available for throwing light upon the regional side of the problem of diagnosis. It must be borne in mind, however, that the regional diagnosis of diseases of the spinal cord is itself a twofold problem. It involves a consideration: (a) of the *transverse area* involved; and (b) of the *longitudinal situation and extent* of the disease in such areas.

(a) **DIAGNOSIS OF THE TRANSVERSE AREA INVOLVED.**—The facts to be tabulated under this head may be set down in the order of their relation to different component parts or regions of the spinal cord.

(1) *Anterior roots of spinal nerves.*—Irritation of these may give rise to various forms of twitching or to tonic spasms in related muscles. Great pressure upon or destruction of the anterior roots will give rise to local complete or partial paralysis in the related muscles, followed in the course of a week or two by marked atrophy, and the establishment of the electrical 'reaction of degeneration' (see PARALYSIS). There will also be an abolition of reflex excitability of these muscles in response to skin-irritation, or to blows upon or stretchings of their tendons.

(2) *Antero-lateral columns.*—Increasing pressure upon or disease of these columns gives rise to paresis, gradually deepening into motor paralysis of parts deriving their nerve-supply at or below the seat of lesion.

When the disease occurs in the lateral column more especially, there may be twitchings or startings in the muscles below, or well-marked spasms, and possibly painful cramps. There may also be great exaltation of the superficial and deep reflexes, if the manifestation of the latter is not hindered by pre-existing spasms. The plantar reflex shows itself in the form of *extension* of the toes, especially of the great toe—'Babinski's sign' (see p. 121). Motor paralysis exists to some extent, but without any appreciable impairment of sensibility. No marked wasting of muscles, or diminution in electrical reactions, usually occurs.

(3) *Grey matter.*—(a) *Of anterior cornua.*—Disease of these parts causes motor paralysis, with atrophy, loss of faradic excitability and of reflex excitability in related muscles—as in cases of disease of the anterior roots of spinal nerves.

(b) *Of posterior cornua and central parts.*—Damage of these regions of grey matter will, according to its completeness in transverse extent, cause more or less delay or defect in the transmission of painful and thermic impressions, and perhaps interfere also with other modes of sensibility. Some trophic lesions in skin and joints may also be met with.

At different levels in the cord special centres (represented in both anterior and posterior regions of grey matter) in connection with definite functions may be interfered with by morbid conditions implicating the grey matter.

(4) *Posterior columns.*—The results of disease confined to this situation (more especially to the

'root-zones') will be—ataxy or signs of inco-ordination of movements; interference with impressions of touch, pressure, temperature, and of 'muscular sense'; abolition of knee-reflex; and diminution or loss of sexual desire.

(5) *Posterior roots of spinal nerves.*—From irritating lesions there will arise lancinating or other pains in the skin and deeper textures of related portions of the limbs, and possibly trophic skin-lesions. Pressure or destructive lesions will give rise to loss, in various degrees, of different modes of sensibility, superficial and deep; and diminution or abolition of the superficial and deep reflexes in related regions of the body.

It has been shown by Sherrington and others that there is considerable overlapping of the skin-areas supplied by contiguous posterior roots, and this has recently been well exemplified in one of the writer's cases of spinal caries operated upon by V. Horsley. In the course of the operation a single posterior nerve-root in the mid-dorsal region was divided on the right side, while two nerve-roots were divided on the left side. Around this latter half of the body a narrow band of anaesthesia was produced, only one inch wide; while on the right side there was no anaesthesia, this being obviated apparently by the overlapping above referred to. No such overlapping exists, however, in connection with the territories of the nerves concerned with the appreciation of heat and cold, or of painful impressions (Head).

(b) **DIAGNOSIS OF THE LONGITUDINAL SITUATION AND EXTENT OF THE LESION.**—This is a consideration distinctly secondary to the other, since, at whatever longitudinal level the disease may be situated, its clinical characters will always be qualified by the part or parts of the transverse extent of the cord that may be involved. Here we are accustomed to obtain information of a general kind from the fact (1) that *special centres* in connection with different viscera and functions, situated at different longitudinal levels in the cord, may be more or less deranged. But we have to depend in the main (2) upon the signs indicative of the *implication of particular sensory and motor nerves*, whose exact relations with different portions of the spinal cord are known. Such signs may consist of some excess or defect of *sensibility*, of *motility*, or of *reflex activity*. Again, where disease or injury of the vertebrae exists in association with disease of the spinal cord, we may be guided by the known relations of the different vertebral spines to the different nerve-roots and segments of the cord.

Evidence from perverted activity of Spinal Centres.

(1) The lateral columns in the upper cervical region contain the motor paths for the muscles of respiration, so that section or disease of these columns at a lower level may interfere with the movements of respiration on the same side of the chest (thoracic muscles); while if the lesion reaches as high as the fourth and third cervical nerves (the origin of the phrenic) the diaphragm itself also becomes paralysed, and the movements of respiration must therefore almost cease.

(2) Again, the upper cervical region of the cord, though it does not contain actual centres connected with the excitation of the heart's action, is traversed by the augmentor and accelerator nerves, whose point of exit from the cord, as white *rami*

viscerales, is with the second, third, and other upper dorsal anterior roots. These nerves ascend to the stellate ganglion, and thence proceed to the heart.

Thus different lesions in this upper cervical region of the cord may, according to their nature and extent, greatly interfere with the heart's action, as well as with the respiratory movements. The frequency of the pulse may be either notably accelerated or retarded; while the respiratory movements may be slower or much quicker than natural, and also extremely irregular and perverted in rhythm.

(3) The lower cervical and upper dorsal regions of the cord also contain the so-called 'cilio-spinal centre.' The fibres emanating from it pass outwards with the fibres of the anterior roots in the first, second, and third dorsal nerves, but especially in the second, and thence into the cervical sympathetic. Irritation of them causes dilatation of the pupil on the same side, together with widening of the palpebral fissure, while section or other destructive lesion of these parts causes contraction of the pupil.

(4) The vaso-motor nerves for the side of the head and neck, and the pinna of the ear, together with those for the lateral lobe and half the isthmus of the thyroid body, arise in similar regions of the cord, and also leave it similarly. Irritation of them produces contraction of the blood-vessels and a lowering of temperature of the side of the face and head, together with diminution of sensibility, an absence of perspiration, and (should the irritation continue) a tendency to slight atrophy of the corresponding side of the face. Destructive lesions, on the other hand, operating upon these vaso-motor fibres, tend to produce a reverse set of conditions. See SYMPATHETIC SYSTEM, Disorders of.

Sometimes there may be signs of paralysis of oculo-pupillary fibres co-existing with signs of irritation of the vaso-motor fibres, or *vice versâ*.

(5) Gaskell has shown that all the *vaso-motor nerves* of the body leave the spinal cord in the anterior roots of the spinal nerves, from the second dorsal to the second lumbar inclusive, passing thence into the lateral ganglia of the sympathetic, in which these vaso-motor fibres lose their medullary sheaths. He adds: 'This chain might, therefore, most appropriately be called the chain of vaso-motor ganglia instead of its present meaningless title of main sympathetic chain.' Gaskell also thinks, and in this he is supported by Sherrington, that the vaso-motor nerves, as well as certain visceral motor nerves, are connected with ganglion-cells in the lateral regions of the grey matter. (See PLATE XVII., p. 1520.)

Generally it may be said that section of one half of the cord or destruction of it for any extent longitudinally, causes at first paralysis of blood-vessels in the lower parts of the body on the same side—this vaso-motor paralysis carrying with it in the same parts an increase of temperature and an exaltation of sensibility. In a short time, however, the vaso-motor paralysis (and with it the increase of heat and sensibility) passes away, owing to the vaso-motor centres in parts of the spinal cord below, and to the peripheral vaso-motor centres, adapting themselves to act independently of those in higher parts of the cord, and of the supreme regulating centre in the medulla ob-

longata. (As a rule, the higher vaso-motor centres control those lower down, but after temporary paralysis even the peripheral vaso-motor centres seem to resume control over related blood-vessels.)

(6) The movements and secretions of the stomach and intestines generally are certainly influenced by the cord in different regions, so that in various cases, owing to perversions of this normal spinal influence, we may get vomiting, diarrhoea, or obstinate constipation—as direct results, that is, of morbid changes in certain parts of the cord in which intestinal sympathetic fibres have their roots. It seems, however, that through the greater portion of the alimentary canal the circular muscles are innervated by motor fibres distributed with the vagus. Thus peristaltic contractions of the oesophagus, stomach, and intestines can be excited by vagus-stimulation, whether the stimulus be applied to the roots of the nerves as they leave the bulb, or to the main trunk of the vagus in any part of its cervical or thoracic course. On the other hand, the circular fibres of the rectum and of the descending colon are supplied by the lower abdominal splanchnic nerves, which leave the cord with the thoracic and not with the sacral outflow of visceral nerves (Gaskell).

In the grey matter of the lumbar swelling of the cord there are aggregated a number of *centres* having to do with important functions, which may be variously interfered with by disease. These centres are those which regulate—(7) the evacuation of the rectum; (8) the evacuation of the bladder; (9) erection, and ejaculatio seminis; and (10) the contractions of the uterus.

In each case the spinal centre constitutes an independent reflex centre, provided with its afferent and efferent nerves, but in each case also there is more or less of connection between the spinal centre and others in the cerebral hemispheres. There must therefore be double sets of fibres for each centre traversing the whole length of the spinal cord and medulla; partly (commissural) for the transference of afferent impressions from each spinal centre to the brain, and partly (internuncial) for the conduction of efferent impressions from cerebral kinæsthetic to spinal motor centres.

In the case of the uterine centre these cerebral connections are of comparatively slight importance; since, with a complete transverse lesion in the cervical or even in the upper dorsal region, the process of parturition may still be successfully accomplished. So long as the spinal mechanism is complete and perfect, parturition may take place without the need of cerebral co-operation. Our subsequent remarks will, therefore, refer principally to the other three lumbar centres.

Complete transverse lesions occurring in any part of the dorsal or cervical regions will, of course, entirely cut off all the above-mentioned lumbar spinal centres from connection with, and therefore from any voluntary control by, the cerebral hemispheres. But various limited local lesions in particular transverse areas of the cord (though the locality of such areas cannot at present be definitely specified) may produce similar results, so far as the cerebral control of any one or two of the lumbar centres is concerned. According as the severance of these lumbar spinal centres from cerebral correlation and control is complete or partial, one or other of the following results would be produced:—

Name of Centre	Complete Severance from Cerebrum	Incomplete Severance from Cerebrum	
	Afferent and Efferent Fibres	Afferent Commissural Fibres only	Efferent Internuncial Fibres only
<i>Rectal centre</i>	Unconsciousness of need, and inability to prevent evacuation <i>Result.</i> —Constipation, with incontinence of feces after an aperient	Unconsciousness of need, and therefore no attempt to restrain evacuation	Consciousness of need to evacuate, with no ability to restrain the act
<i>Vesical centre</i>	Unconsciousness of need, and inability to prevent micturition <i>Result.</i> —Reflex evacuation in gushes at intervals	Unconsciousness of need, and therefore no attempt to restrain micturition	Consciousness of need, but inability to restrain micturition
<i>Sexual centre</i>	Diminution or absence of sexual desire. Erections and emissions, if they occur, wholly dependent upon the spinal reflex mechanism	With simple destruction of fibres, nearly same results as set down in previous column; but with <i>irritation</i> of afferent fibres there might be great increase of desire (satyriasis or nymphomania)	Feelings of desire, but no erection in response Erection and emissions, if present, purely through spinal reflex But with <i>irritation</i> of efferent fibres there may be persistent erections, mostly without desire

The *rectal* and the *vesical spinal centres* are each composed of two parts, with their separate afferent and efferent nerves—one set in relation with a sphincter muscle, and the other in relation with detrusor or expulsive muscles in functional opposition to the former. The several nerve-fibres, both afferent and efferent, in connection with these centres have been shown by Sherrington to be contained in the first four sacral nerves, and in the case of the bladder also in the third, fourth, and fifth lumbar nerves (though none are contained in the intervening sixth and seventh lumbar). Thus, as Sherrington says: 'If, as one may suppose, the position of the outflow of the efferents fairly indicates the position of the nucleus whence they come, then we may suppose that a long nucleus for the bladder exists in the lumbo-sacral region, which has, however, a gap in its continuity. . . . The outflow from the anterior roots above the gap is into the sympathetic system; from the anterior roots below the gap it is direct by the sacral nerves.' Destruction or irritation of either of these sets of fibres, or of one of the centres, will necessarily interfere to some extent with the working of this particular centre, so that its functions may be interfered with in several different ways. There may be various degrees of irritability of the bladder or rectum, or various degrees of paralysis of these organs.

In cases of paralysis of the bladder, especially when they are due to lesions implicating its spinal centre, the urine is apt soon to become fetid and alkaline, and inflammation (alone or with ulceration) is most prone to be set up in its mucous membrane.

The details as to the modes of disturbance of the *genital function*, where disease implicates its lumbar centre or the afferent and efferent nerves in connection therewith, are less known than where it involves the communicating fibres between this centre and the cerebrum. The *nervi erigentes* have been shown to pass out with the second and third sacral nerves, so that the sexual like the rectal centre is contained in the posterior part of the lumbar swelling. In the female the uterine centre has been shown to be similarly situated.

Evidence from implication of particular Sensory or Motor Nerves.

The more precise indications concerning the

longitudinal implication of the spinal cord are, as already stated, derivable from the level at which alterations in sensibility or in motility (either voluntary or reflex) are to be detected. The more closely the lesion approaches to what is called a 'total transverse lesion,' the more distinctly will signs of this order reveal themselves. It is important, too, to recollect that the fibres of different sensory roots are to some extent dispersed through cutaneous surfaces overlying the muscles supplied by the corresponding motor roots. There are, however, many exceptions to this.

In regard to *sensibility*, the upper limit at which the trunk is affected is often sharply defined by the presence of a feeling of constriction, of pain, or of numbness ('girdle sensation') encircling the body. This sensation is generally supposed to be due to irritation of the roots of the nerves as they traverse the posterior columns (or perhaps outside them) at the upper level of the lesion. This symptom may of course be absent, but in some cases of paraplegia it is well marked, especially where the upper level of the lesion lies anywhere between the third and the twelfth dorsal nerve-roots.

Our knowledge has of late been greatly increased concerning the extent and distribution of the cutaneous areas ('segmental skin-fields') pertaining to each sensory spinal nerve, owing to the labours of Ross, Allen Starr, James Mackenzie, Head, and Sherrington. As the last investigator says: 'Although in a plexus each posterior spinal root gives separate contributions to several nerve-trunks, the cutaneous distribution of the root is composed not of patches which are disjointed, but of patches which are so joined that the distribution of the entire root forms one continuous field. . . . Each segmental skin-field spreads, to a certain extent, across neighbouring segmental skin-fields. . . . The *fore-lap* and the *after-lap* are, throughout the body, very great, and each region of skin appears to be supplied by at least two sensory roots. . . . The shape of a segmental skin-field is, where simplest—e.g. in the trunk and neck—band-like, wrapping transversely round one lateral half of the body; it has fairly parallel edges, but is somewhat broader near its ventral than at its dorsal end. In the limb the segmental skin-fields are *distorted* from the simple band-like type; and they are also seemingly

dislocated from their attachments to the mid-dorsal and mid-ventral lines of the trunk. This peculiar distribution Sherrington explains by supposing that the mid-dorsal as well as the mid-ventral line of the body extends outwards in the position of the limb as a side-branch or secondary axis. 'Upon these dorsal and ventral side lines, as upon secondary

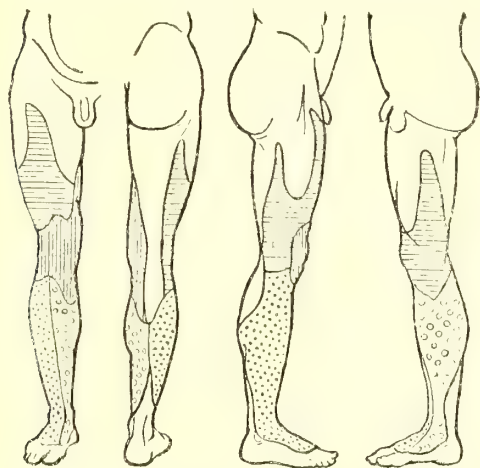


FIG. 5.—To show the presumed distribution of the areas supplied from the 2nd lumbar to the 1st sacral segments. (After Head.)

The area marked in *cross lines* represents L 2.

The area marked in *vertical lines* represents L 3.

The area marked with *circles* represents L 4.

The area marked with *dots* represents L 5.

The area left plain below represents S 1.

dorsal and ventral axes, the cutaneous segments of the limb are ranged, as though upon folded portions of the axial lines of the trunk itself.'

Now, as already stated (p. 1526, (5)), this overlapping of the segmental skin-fields exists, especially for tactile sensations, owing probably to the related tracts of grey matter in the spinal cord each extending into the spinal segments above and below its own, and consequently being represented in the corresponding posterior spinal nerve-roots. The researches of Head, however, seem to show that no such overlapping of segmental skin-fields or extension of related grey matter into adjacent spinal segments exists for painful impressions, or for those of heat and cold. Consequently, to determine the exact boundaries of a cutaneous area of defective sensibility, whether due to a lesion in the cord or of one or more posterior spinal roots, we shall get much more accurate results if we seek to map out the areas in which the patient may be insensitive to painful impressions, or to those of heat and cold, rather than those areas in which he is insensitive to ordinary tactile impressions. These different areas in relation with the several posterior roots have been definitely mapped out by Head. A supplemental figure, showing the probable skin-fields from the second lumbar to the first sacral inclusive, has been reproduced above (fig. 5). For the rest the plates given by Head must be consulted. See p. 1138.

Efforts were also formerly made to define the muscles that were paralysed, in any given case, with a view to determine the upper limit of the lesion in the cord. It was thought that a reference to the

anterior roots by which such muscles are innervated ought to enable us to fix upon the particular segment of the cord from which their nerves proceed, and thus to determine with precision the upper level of the lesion in the *motor regions* of the cord. This is a matter, however, by no means so simple as might be supposed, because the majority of limb- and trunk-muscles receive fibres from more than one motor root, as Preyer and Krause and others showed long ago. The same conclusion has been arrived at by Eckhardt, and later by Sherrington. The former says: 'A great number of muscles in the limb obtain nerve-fibres each of them from several nerve-roots. Most of the thigh-muscles always, some of the leg-muscles frequently, are supplied by three, the latter more often by two, nerve-roots.' According to Sherrington, when a muscle is supplied by three nerve-roots it is noticeable that the middle root of the three usually causes the most powerful contraction. However this may be, it is obvious that the fibres going to each muscle must take their origin 'over a certain considerable longitudinal region of the cord.'

This fact, that muscles receive their nerve-supply from two or three different nerve-roots, was formerly thought to be due to the circumstance that the same muscle is called into play in different co-ordinated movements. And the view of E. Remak, that functionally related or synergic muscles are represented together in the anterior horns of the spinal cord, was supposed to have been confirmed and extended by Ferrier and Yeo by their experimental observations on the functions of the anterior roots. They say that stimulation of the individual roots of the brachial and crural plexuses results, not in mere unrelated contractions of various muscles, but in highly co-ordinated synergic contractions, leading to definite movements. But as the 'muscles thrown into action by each root are innervated in most cases by several nerve-trunks,' the result 'of section of each motor root would therefore be paralysis of the corresponding combination, not necessarily, however, of the individual muscles involved . . . while *weakened*, they might yet act in other combinations in so far as they were supplied by other roots.' The different combined movements which they thought to be dependent upon particular motor roots are cited by the authors in this paper.

This important subject has been re-investigated of late by Sherrington, who comes to an opposite conclusion. He says: 'Contrary to Panizza's doctrine, revived and ably reappointed by Remak and by Ferrier and Yeo, and followed since by Bert, Marcacci, and others, I believe John Müller to have been right in attributing to the arrangement of the motor roots of the limb-plexus an anatomical significance, based on metamerism, rather than a teleological significance dependent upon supposed demands of functional co-ordination.' He considers that in the great majority of cases muscles innervated by the same nerve-root lie adjacent one to another; but 'that the loss of some particular co-ordinated movement results, from the severance of one of the motor roots of the limb-plexus,' he says, 'I have been unable to detect, and seen no evidence to support.' His own investigations lead him to believe that the section of some two, three, or even more anterior roots going to the upper or lower limb-plexus leads to a weakened condition of many movements. He adds: 'The helplessness, at first very apparent,

rapidly diminished up to a certain point. The diminution seemed to be largely due to the over-coming in course of time of an unwillingness to attempt movements with the injured limb.⁷ His experiments with monkeys convinced him that the execution of a co-ordinated movement was still possible when one motor root only of the plexus remained open as a channel from the cord.

All this shows that the upper level of a lesion in the cord can only be fixed approximately by reference to the motor paralysis existing in limbs or in the trunk of the body.

The integrity of those *reflex* actions which can be elicited either in health or in disease depends, of course, upon the integrity of the entire nervous arcs concerned (that is, upon the integrity of incoming fibres, centres, and outgoing fibres). Thus, though the impairment of a reflex may not necessarily be due to central causes, its presence, on the other hand, clearly shows that the grey matter and other regions of the cord which must be traversed by its stimuli are not impassable; while its exaltation will indicate the probable existence of some congestion by which the grey matter in question is rendered more excitable, or else some central change by which it is cut off from cerebral inhibitory influences. In § 4 will be found all the needful indications referable to the presence or absence of reflexes which can be used for the determination of the upper level of a lesion existing in the spinal cord. The lower level of a lesion in this organ is always extremely difficult to determine; and if it is to be made out at all, it must be done by reference to the existence of particular muscular atrophies, rather than from the presence or absence of certain reflexes.

Other data in regard to the longitudinal situation of a lesion in the spinal cord are of a more precise order, and are dependent upon the *relations of the vertebral spines to different anterior nerve-roots*. These data are available in cases where the spinal cord becomes implicated in connection with primary disease or injury of the vertebræ. To obtain such data, it is needful to know the exact relations subsisting between the bodies of the different vertebræ, their spines, and the origins of the different nerve-roots.

Only the highest cervical nerves arise from the cord opposite the place at which they leave the spinal canal. As we descend, the distance between these two points gradually increases, and it attains its maximum when we come to the nerves of the cauda equina. What nerve-origins correspond, therefore, to a given vertebral level can only be decided by careful anatomical investigation.

A further complication is introduced into this question, seeing that the vertebral spines, which we are compelled to deal with as localising guides, have different relations in different parts of the spinal column to the bodies of their respective vertebræ—e.g. they incline downwards in the dorsal region more especially, to a less extent in the cervical, and scarcely at all in the lumbar region.

Some of the principal facts in reference to these two points, namely (1) the relations of spinous processes to bodies of vertebræ, and (2) the relations of vertebral spines to origins of nerve-roots, will be found to be embodied in the next section.

SUMMARY.—For practical purposes it will be well here to group together the various indications

as to longitudinal localisation to which we have referred—classifying them as they are related to one or other of four regions of the spinal cord.

(a) **Cervical Region of the Cord.**—*This corresponds externally with the space between the occiput and the upper border of the seventh cervical spine (eighth cervical nerve).*

The first, second, and third cervical spinous processes are respectively opposite the origins of the third, fourth, and fifth cervical nerves. The phrenic nerve (motor nerve of the diaphragm) arises from the fourth, or from the third and the fourth cervical nerves. Opposite the third cervical spine (level of fifth cervical nerve) the cervical swelling of the cord begins; while it ends opposite the seventh cervical spine (level of the first dorsal nerve).

Disease of this region may involve interference with respiration, and possibly weakness of voice; interference with the heart's action—pulse very frequent, or the reverse; flushing or pallor of the head and neck; continued priapism (with crushing lesions); augmentation of temperature in the body generally (hyperpyrexia); and marked contraction or dilatation of the pupil.

The innervation of the shoulder-, arm-, and hand-muscles is derived from spinal nerves between the sixth cervical and first dorsal inclusive; those supplying the ulnar side of the hand and forearm arising from the lower level, that is, from the upper part of the next region. The upper extremities may, therefore, be more or less paralysed, as well as the trunk and lower extremities.

(b) **Upper Half of the Dorsal Region of the Cord.**—*This corresponds externally with the space between the seventh cervical spine (first dorsal nerve) and the fourth dorsal spine (sixth dorsal nerve).*

The results of disease here are apt to be these: The 'scapular reflex' may be abolished, calling into activity as it does the last two or three cervical and the first two or three dorsal nerves; the intercostal muscles are paralysed at different levels; a 'girdle sensation' is felt at different levels; there may be prominence of certain vertebral spines, and possibly tenderness on pressure or on tapping over them; the 'epigastric reflex' may be abolished, depending as it does upon the spinal cord at the level of the fourth to the sixth or seventh pairs of dorsal nerves; and priapism (with crushing lesions) may occasionally be met with. We may have, therefore, some or all of these signs together with more or less complete paralysis of the lower part of the trunk and of the lower extremities.

(c) **Lower Half of the Dorsal Region of the Cord.**—*This corresponds externally with the space between the upper border of the fifth dorsal spine (seventh dorsal nerve) and the lower border of the tenth dorsal spine (space below twelfth dorsal nerve).*

Disease here may give rise to the following symptoms: The 'abdominal reflex' may be abolished, depending as it does upon the integrity of the cord between the levels of the eighth dorsal and the first lumbar nerves. Paralysis of lower intercostal muscles or of abdominal muscles may possibly occur, in addition to paralysis of the lower extremities. 'Girdle sensation' may be felt at different levels (the umbilicus corresponding with the tenth dorsal nerve, and the 'ensiform area' with the sixth and seventh dorsal nerves). There

may be prominence of certain of the lower dorsal spines, with possible tenderness.

(d) **Lumbar Region of the Cord.**—*This corresponds externally with the space between the lower border of the tenth dorsal spine (just below the twelfth dorsal nerve) and the upper border of the second lumbar vertebra.*

Here the symptoms are: Paralysis, not implicating the abdominal muscles, but limited to more or less of those of the lower extremities. No 'girdle sensations' around the trunk. Three superficial reflexes may be abolished, namely, the 'cremasteric,' which depends upon the integrity of the cord in the upper lumbar region; the 'gluteal' and the 'plantar,' both of which seem to be dependent upon the integrity of the lower part of the lumbar region of the cord. A deep reflex may also be abolished, namely, the so-called 'knee-jerk,' which is dependent upon the upper lumbar region of the cord. 'Ankle-clonus' may be met with when disease affects the upper or mid-lumbar regions of the cord, but not where the lower lumbar region is implicated. Loss of sensibility about the perineum and anus (if not due to disease of nerve-trunks) is indicative of disease of the posterior columns in the lower lumbar region. Absolute paralysis of the bladder and rectum may be present, with tendency to inflammation and ulceration of the former organ. (With lesions higher up in the cord there may also be, according to their extent, some of the alterations in regard to the action of the bladder and rectum which have been set forth in a table on p. 1528.)

Although it is true that the groups of symptoms presented in different diseases of the spinal cord, considered individually and collectively, afford the materials upon which a regional diagnosis must be founded, it is no less true that a part of the symptomatology (namely, that comprised in the mode of origin and the mode of establishment of the disease, together with what may be gathered from the patient's state generally, from his family history, and from his personal history) constitutes the basis upon which a pathological diagnosis has to be arrived at. Again, although the arrival at a regional diagnosis is often spoken of, and may seem to be a process altogether distinct from that involved in the arrival at a pathological diagnosis, yet, as a matter of fact, in the investigation of many individual cases of spinal disease, it will be found that the one problem is not settled first, and the other afterwards, but that both are tentatively considered more or less simultaneously. Thus, certain empirically known pathological conditions may afford at once a ready explanation of a given group or sequence of symptoms, as in 'infantile paralysis,' in 'locomotor ataxy,' or, in a more general sense, in angular curvature of the spine. Here, therefore, the pathological diagnosis goes hand in hand with the regional diagnosis, and in working them out each gathers additional confirmation from the establishment of the other. Sometimes, however, as in the case of traumatic injuries (including stabs, and fractures with dislocations of vertebrae), the pathological diagnosis is at once obvious, and the regional diagnosis alone requires to be settled in detail.

For the above reasons, it has been necessary to tabulate in this article certain pathological data concerning the spinal cord, though it would not be found specially advantageous were we to follow out

this part of the subject further, and attempt here to set down the more general *clinical data and deductions of pathological import*, necessary to be borne in mind for the arrival at a pathological diagnosis, in order to form a series of facts and deductions comparable with those already given in elucidation of the problems of regional diagnosis. These other problems will be dealt with, as far as possible, in the descriptions of the several diseases of the spinal cord.

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SPINAL CORD, Special Diseases of.—These will be described under the following headings.

I. Diseases of the Spinal Cord dependent upon known organic changes:—

- (1) Spinal Cord, Concussion of, p. 1531.
- (2) Spinal Cord, Punctured or Gunshot Wounds of, p. 1532.
- (3) Spinal Cord, Sudden Crushing Lesions of, p. 1534.
- (4) Spinal Cord, Slow Compression of, p. 1535.
- (5) Spinal Cord, Anæmia of, p. 1538.
- (6) Spinal Cord, Hyperæmia of, p. 1538.
- (7) Spinal Cord, Inflammation of, p. 1539.
- (8) Spinal Cord, Hæmorrhage into, p. 1541.
- (9) Spinal Cord, Softening of, p. 1543.
- (10) INFANTILE PARALYSIS, pp. 1161, 1162.
- (11) Acute Spinal Paralysis of Adults, p. 1547.
- (12) Acute Ascending Paralysis, p. 1548.
- (13) Chronic Spinal Paralysis, p. 1549.
- (14) PROGRESSIVE MUSCULAR ATROPHY, p. 1515.
- (15) LOCOMOTOR ATAXY, p. 1660.
- (16) Spastic Spinal Paralysis, p. 1550.
- (17) Amyotrophic Lateral Sclerosis, p. 1552.
- (18) Multiple or Disseminated Sclerosis, p. 1553.
- (19) FRIEDREICH'S ATAXY, p. 560.
- (20) COMBINED DEGENERATION OF THE SPINAL CORD, p. 325.
- (21) Tumours and New Formations of the Meninges and Spinal Cord, p. 1557.
- (22) Syringomyelia, p. 1559, and
- (23) Malformations of the Spinal Cord, p. 1560.

II. Diseases dependent upon unknown or imperfectly known organic changes:—

- (24) TETANUS, p. 1686.
- (25) TETANY, p. 1690.
- (26) TORTICOLLIS. See WRY-NECK.
- (27) WRITER'S CRAMP. See WRITER'S CRAMP.
- (28) Reflex Paraplegia, p. 1561.
- (29) Intermittent Paraplegia, p. 1562.
- (30) Functional Paralysis of Spinal Type, p. 1562.
- (31) Paraplegia dependent on Idea, p. 1563.
- (32) Neurasthenia Spinalis, p. 1564, and
- (33) Toxic Spinal Paralysis, p. 1564.

See also GENERAL PARALYSIS OF THE INSANE; PARALYSIS AGITANS; HYDROPHOBIA; CHOREA; and MENINGES, Diseases of.

I. Spinal Cord, Concussion of.—SYNON.: *Commotio Medullæ Spinalis*; Fr. *Commotion de la Moelle Epinière*; Ger. *Erschütterung des Rückenmarks*.

ÆTIOLOGY.—This condition is met with principally in persons who have fallen from a height, or in those who have been present in a railway collision. In these cases the brain is apt to suffer as well as the spinal cord, and it is not always easy to unravel the respective symptoms due to shock of this or that great segment of the cerebro-spinal system.

ANATOMICAL CHARACTERS.—In many of these cases there are, in all probability, no morbid changes that would be discoverable. In others, however, minute extravasations of blood, or actual ruptures of the nerve-tissue, may occur. Sometimes, as in a case seen by the writer, even notable lesions are produced. An example of the slighter lesions is recorded by Gull, in which small extravasations of blood were found in the anterior and posterior cornua, as well as in the posterior columns of the cord. In neither of these cases was there any external or visible injury; but in each paraplegia was produced immediately after the fall that determined the lesions in the cord. In addition to hæmorrhages into the substance of the spinal cord itself, there is in these cases the possibility of the occurrence of meningeal hæmorrhages, pressing upon the cord or its nerve-roots; and within a day or two after the occurrence of the concussion itself, there is the possibility of some local and subacute inflammation being set up in the membranes of the cord.

SYMPTOMS.—In the great majority of these cases no complete paralysis is induced, even at first. There may at most be paresis of one or more limbs, general prostration, nausea with occasional vomiting, a rapid and possibly irregular or intermittent pulse (especially after the least exertion), with occasional startings and twitchings of the limbs, the sensibility of which may be diminished, exalted, or unaffected. The temperature will probably be at first depressed, as a result of shock, though subsequently a slight febrile elevation may continue for some days. The tongue may be furred, the appetite bad, the bowels constipated; while in regard to micturition there may be either some delay and difficulty, or, on the contrary, an irritability of the bladder, with difficulty in retaining its contents after the desire to micturate is once felt. With this there is often general restlessness, nervousness, and insomnia.

In more severe cases of concussion, even where there is no complication resulting from appreciable lesions, the shock to the system (*see SHOCK*) may be more profound, and there may be paralysis of limbs, lasting perhaps for some days, and then rather suddenly disappearing.

DIAGNOSIS.—The questions to be determined are, whether, looking to the symptoms presented by the patient, there is likely to be any organic lesion or change in the spinal cord or its membranes; or whether we have to do with mere functional perturbations induced by the shock or blow to which the patient has been subjected. In the absence of definite paralysis, or even with its presence for the first few days, the answer to this preliminary question will often be shrouded in doubt. To come to a definite opinion as to the precise nature of the change which a spinal cord, deemed to be damaged in some way after a concussion, has undergone, lapse of time and several examinations of the patient are often required.

In many cases in which compensation for an injury is claimed, a further complication appears. Here it is that the difficulty arises as to how much the symptoms experienced, or said to be experienced, may be due to an excited imagination, and how much to causes independent of the imagination, whether voluntarily or involuntarily aroused. It must be conceded that symptoms of injury are undoubtedly feigned by unscrupulous persons; and it seems also equally clear that, even unknown to

the patient, the excitement consequent upon the accident, the details heard concerning the injuries of others, combined with the inquiries of doctors and of sympathising friends, tend to keep up and to exaggerate symptoms in many nervous patients, over and above those which may have resulted from the shock. Such patients also may make a more speedy recovery subsequent to trial and compensation than they had been making before the trial, and yet they *may* not have been in any sense impostors. It is true that such persons, however, do not recover quite so quickly as those others who for their own unscrupulous ends have been previously exciting their imaginations in a voluntary manner. *See RAILWAY INJURIES.*

PROGNOSIS.—In only the severest cases of concussion or shock is there actual danger to life (*see SHOCK*). Where, however, great prostration is induced, and especially in those who may previously have been suffering from heart-affections, or from a very excitable nervous system, life may be speedily brought to a close; or at most such patients may not survive a severe concussion more than a day or two.

Severe concussions of the cord may also form the starting-points of many and varied deviations from health, which may not begin to show themselves for weeks, or perhaps even months, after the initial shock. Among such sequelæ, which have come under the writer's notice, may be mentioned the following: Loss of flesh with general failure of nutrition, epileptiform fits, progressive muscular atrophy, lateral sclerosis of the spinal cord, a slowly increasing paraplegia (of uncertain pathological basis), and caries of vertebræ followed by angular curvature and paraplegia.

In other and slighter cases, time and rest, with suitable medical treatment, may be expected to lead to perfect recovery, sometimes speedily, but sometimes only after protracted periods of impaired health.

TREATMENT.—In the first instance, symptoms of shock have to be combated by the employment of warmth and stimulants. In subsequent stages, rest in the recumbent position must be enjoined for a time. It is of the first importance to make sure that the patient does take complete rest, and is kept free from excitement during the first few days after any concussion-accident, and that he gets sound sleep at night, under the influence of bromide of potassium, or of this together with chloral hydrate. If the condition of restlessness, with disturbed sleep, can be checked, then a mitigation of other symptoms may be expected to follow. The application of ice to the spinal column may at times be desirable; or pain must be relieved by the subcutaneous injection of small doses of morphia. Later on, tonics, with a simple nutritious diet and plenty of fresh air, together with rest, will be needed for the complete restoration of the patient.

H. CHARLTON BASTIAN.

2. Spinal Cord, Punctured or Gunshot Wounds of.—**SYNON.**: Acute Traumatic Lesions of the Spinal Cord; *Fr. Plaies et Contusions de la Moelle Epinière*; *Ger. Rückenmarkszerreissungen.*

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—Punctured or gunshot wounds of the spinal cord are commonly made with knife, dagger, sword, or bullet.

In each set of cases, the wound in the spinal

cord will be associated with perforation or rupture of some of the membranes, and also with hemorrhage, either between them or into the substance of the cord. The arches of the vertebrae or their articular processes and some of the ligaments connecting them may be more or less damaged, and a wound commonly exists through the contiguous skin and muscles. In the cord itself, there may be either a clean-cut wound through certain of its columns and parts, or a broader crushing lesion. In each case more or less blood may be effused upon and below the cut surfaces of the cord. At later stages, there may be signs of inflammation of the membranes, as well as of local inflammatory softening of the substance of the cord.

SYMPTOMS.—The signs and symptoms consequent upon wounds of this kind are subject to endless variations, in accordance with the different regions of the cord involved, the actual extent of the wound in its substance, and the possible presence of varying amounts of effused blood. These wounds often involve only a portion of the transverse area of the cord. It is indeed in this class of cases more especially that *hemiplegia spinalis* and *hemiparaplegia* are met with. Thus, where a unilateral lesion exists in the mid or upper cervical region, both arm and leg are paralysed, so that the state known as *hemiplegia spinalis* is produced; but where it occurs in the dorsal region, the one leg only is paralysed, and we have what is known as *hemiparaplegia*.

Since the experiments made many years ago by Brown-Séquard on the result of hemisection of the cord in certain animals, it has been commonly supposed that the essential peculiarity in the latter cases is that on the side of lesion there is complete motor paralysis in the limbs or limb below; while on the opposite side, the limbs or limb, and the trunk up to the middle line, are more or less completely anæsthetic—sensitiveness to impressions of touch, pain, temperature, and tickling being alike abolished. The subsequent researches of Mott are opposed to this view in regard to sensory troubles. He maintains that they exist on the same side as the lesion and as the motor paralysis, and, in confirmation, says that where the hemisection is accurately made, no ascending secondary degenerations are seen on the opposite side of the spinal cord.

Other minor peculiarities that have been described are these: On the side of *motor paralysis*, there is also vaso-motor paralysis, which carries with it, as consequences, (a) an elevation of temperature (from $1\frac{1}{2}^{\circ}$ to 2° F.), and (b) a hyperæsthesia for all modes of sensibility (owing in part to hyperæmia in the limb and cord). Surrounding the body, at the level of the upper margin of anæsthesia on the side of sensory defect, there is usually a narrow girdle of hyperæsthesia; while below this level, on the side of the lesion, there is a half-band of hemianæsthesia—whose depth varies with the longitudinal extent of the lesion. The complete zone of hyperæsthesia has been thought to be due to hyperæmia of nerve-roots, and of the grey matter of the cord immediately above the lesion; while the half-zone of anæsthesia is supposed to be dependent upon destruction of the nerve-roots, and of the spinal cord for a certain extent at the seat of lesion.

If bed-sores occur, they are met with on the side of paralysis; while in one or two cases signs of a

joint-affection (in the knee principally) have also occurred on the side of sensory and motor paralysis. There seems no reason for expecting any special muscular atrophy or diminution of faradic irritability on the side of paralysis, except in parts of those muscles whose nerve-supply comes from the portion of the anterior cornua actually destroyed by the lesion. In many cases, especially at first, there is paralysis of the bladder and of the rectum, or there may be incontinence of urine. Later on, these troubles tend to diminish. The superficial and deep reflexes are generally found to be exaggerated on the side of lesion.

In gunshot wounds, whether occasioned by pistol or rifle, splinters of bone may be depressed at times, so as to compress and irritate the cord, and thus the symptoms may be made to approximate more closely to wounds of the next category.

After a few days the symptoms may be complicated by those of spinal meningitis, or extended by the spread of an inflammatory softening of the cord above and below the seat of lesion.

DIAGNOSIS.—The primary cause of the patient's condition is generally only too obvious. It may be clear that we have to do either with a punctured or with a gunshot wound in some region of the spine; but subsequently many, and often very difficult, questions require to be solved. It is of first importance to learn whether the cord itself is really damaged, or whether the symptoms are in the main caused by epi-dural or subarachnoid hemorrhages (see MENINGES, SPINAL, Hemorrhage into). In the former case there will be evidence of complete or partial interruption of conduction in the cord, to or from all parts below the seat of lesion, and not of a mere local implication of nerve-roots. If it seem probable that the cord itself is injured, we have to determine whether it is completely cut across, or only partially damaged—and if the latter, to what extent. These questions must be decided in the main by reference to the signs given on pp. 1526 to 1531.

Should the case be seen for the first time several days after the injury, an exact diagnosis as to the amount of damage to the cord itself is often greatly obscured by the existence then of certain secondary pathological conditions—more especially localised inflammation of the meninges, or secondary inflammatory softening, extending perhaps above or below, or in both directions, from the original wound. A process of softening may also extend transversely through the whole substance of the cord, even where only a unilateral lesion had previously existed.

PROGNOSIS.—This, as a rule, is bad in all cases of traumatic injury of the spinal cord; and the gravity of the case is usually the greater the higher the wound happens to be situated in the cervical region. Wounds of the dorsal or lumbar region of the cord are rather less serious, so far as life is concerned.

The degree of recovery from paralysis of limbs will greatly depend upon the nature and extent of the wound. A clean-cut wound may be filled up by the growth of a kind of cicatricial tissue; but, so far as our knowledge goes at present, it must be said that there is no definite evidence that the nerve-substance of the cord can be reproduced in man. It is remarkable that this should be so, seeing that the power of repair in cut or damaged peripheral nerves has been so clearly established.

TREATMENT.—Absolute rest, with cold applications, and possibly local blood-letting, will be needed in the first instance.

Subsequently, when immediate danger from shock and from the spreading of local inflammation has passed away, the patient must be treated upon the general principles applicable to all cases of paraplegia—which principles will be found set forth in 9. SPINAL CORD, Softening of.

H. CHARLTON BASTIAN.

3. Spinal Cord, Sudden Crushing Lesions of.—Fr. *Compressions Brusques de la Moelle Épinrière*; Ger. *Rückenmarksquetschungen*.

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—

The above form a class of wounds sufficiently distinct to need separate treatment. This kind of damage to the cord may be produced by the sudden giving way of a carious vertebra in any part of the spinal column; more rarely by a heavy blow on the back, which does not fracture the spine; or, in a modified form, by the bursting into the spinal canal of an aortic aneurysm, after its erosion through the vertebrae. But in the majority of cases such wounds of the spinal cord are the results of forms of external violence which cause fracture and dislocation of vertebrae, in some portion of the spinal column between the upper cervical and the upper lumbar region. When this occurs, displacement of vertebrae, even to a slight extent, especially in the dorsal region, in which the spinal canal is narrowest, is sufficient to produce severe pressure upon, or crushing of, the spinal cord. The membranes may not be torn across, and the actual dislocation may at times be only momentary, but the substance of the cord itself may be greatly compressed or reduced to a blood-stained semi-fluid mass of pulp. After some hours there are obvious signs of a commencing inflammatory reaction in the membranes; and above and below the seat of lesion similar changes are apt to be set up in the spinal cord itself, which may go on to the production of a variable amount of inflammatory softening. The patient may die, however, before any of these latter changes have been established.

SYMPTOMS.—These vary much, according to the region of the cord involved. Still, in spite of differences thus dependent upon the seat of injury, there is a certain general similarity in the symptoms produced by all crushing lesions of the spinal cord. They are usually of this nature: There may be more or less marked evidence of shock; complete paralysis, both motor and sensory, of parts below the seat of lesion; in addition to severe pains in the back, girdle-pains surrounding the body at the upper limit of sensory and motor paralysis; increased heat or possibly undue coldness of the body throughout the paralysed parts; complete paralysis of bladder with retention of urine, gradually giving place to incontinence; paralysis of intestine, extremely obstinate at first, but subsequently complicated with involuntary evacuations after the administration of purgatives; and extinction of all reflex actions at first.

In the course of two or three days, if the patient should survive, other general symptoms become well marked, owing to the establishment of a local meningitis, together with some amount of traumatic myelitis. Among these we have general fever, with an increase of the 'girdle sensation,' and of pains in the limbs; twitchings in the limbs or in parti-

cular muscles; and also a general increase in reflex actions, where the crush has been only partial, though where the whole thickness of the cord has been destroyed the deep and most of the superficial reflexes in the paralysed parts continue to be abolished.

The above-mentioned complicating pathological processes may gradually subside, but there will still be danger to life from the supervention of severe cystitis or of extensive bed-sores, together with one or other of the various sequelae to which such conditions are apt to give rise.

The additional symptoms and variations met with, according as the crushing lesion occurs in different regions of the cord, are as follows. They increase in number the higher the lesion occurs in the spinal cord. See SPINAL CORD, Diseases of: § 8.

When it is situated in the *lumbar swelling* we have, in addition to the limitation of the paralysis to the lower extremities, and a more or less complete extinction of related reflex actions, the appearance of rapid atrophy in some of the paralysed muscles, together with the manifestation of the electrical reaction of degeneration. The bladder and rectum are apt to be completely paralysed.

With the lesion in some part of the *dorsal region* we have sensory and motor paralysis of the trunk up to a certain level, with an absence of the rapid atrophy and before-mentioned electrical reaction in the muscles of the lower extremities, though some atrophy and the presence of this reaction may occur in some of the trunk-muscles innervated from the damaged region of the cord. In addition (and notably where the lesion is in higher parts of the dorsal region) there may be some weakness of voice, some interference with the movements of respiration (especially with those of expiration), as well as marked and continuous priapism. The superficial and deep reflexes may be depressed or exalted, according to the complete or partial nature of the damage at the seat of lesion.

With the lesion in the *lower cervical region* the upper extremities are partly paralysed, both as regards sensation and motion; the movements of respiration are much more gravely interfered with (expiration especially), while inspiration is of a purely abdominal type; the voice is notably weak and feeble. Continued erection of the penis is more frequently met with; and in some cases a remarkable hyperpyrexia supervenes, in which the temperature before death may rise to 108°–110° F. Should death not occur in this way, it is very apt to supervene in the course of a few days, by gradual failure of respiration, which grows worse than it was in the early days of the affection, owing to the secondary myelitis which becomes established implicating the cord and nerve-roots at a level higher than the original wound. The pulse is often much interfered with, but variously: it may be less frequent or much more frequent than natural; it may be small, irregular, and frequent; or full, regular, and infrequent in its beats. There may also be signs of paralysis of the vaso-motor nerves supplying the neck and head, perhaps to a more marked extent on one side than on the other.

Where the lesion occurs in the *upper cervical region* of the cord, complete paralysis of the trunk and of all four extremities may be recognised, if death does not occur too suddenly to allow even this to be observed. The sudden death, so apt to

occur in these cases, is due to shock, and to the fact that the diaphragm may be paralysed as well as the other respiratory muscles. Where the lesion does not involve the whole of the roots of the phrenic nerve, and where the shock has not been too abrupt and violent, life (with extremely difficult respiration and almost complete loss of voice) may be prolonged for a few hours.

DIAGNOSIS.—If the existence of fracture and dislocation of vertebræ can be substantiated, the probabilities are always in favour of the presence of a crushing lesion in the spinal cord. Otherwise, after a very severe fall or blow upon the back, doubts may be at first entertained as to whether we have to do with the effects of concussion alone, or with this *plus* some amount of crushing of the cord or of hæmorrhage upon or beneath its membranes. The subsequent course of the symptoms may, however, in a day or two, enable us to resolve these doubts.

PROGNOSIS.—The prognosis in lesions of this kind, as already indicated, is much graver than in the case of mere punctured wounds of the cord—these being oftener slight and partial in their transverse extent. Death may occur immediately, or at any time during the first week—in the main from failure of respiration and of the heart's action. It is only in exceptional cases, and where the lesion is in the dorsal or lumbar region, that life is prolonged for several weeks or months. Such lesions are probably too severe to admit of anything like thorough repair. Paralysis, therefore, of a more or less complete kind, is lasting, and the prognosis will always be distinctly bad wherever the superficial and deep reflexes remain absent after the symptoms of shock have subsided (see SPINAL CORD, Diseases of: p. 1520). But even where life is prolonged for six to twelve months or more, it is, as a rule, ultimately lost, owing to the establishment of sloughing bed-sores and ulcerative cystitis, followed perhaps by septicæmia, extensive meningitis, or other complications.

TREATMENT.—In many of these cases treatment is useless and death inevitable. In those which are of a less urgent nature, the possibility of bringing about some relief by trepanning, with the view of elevating any depressed fragments of the vertebral arches, need not be lost sight of; and, as Bowly has pointed out, this should be borne in mind especially in those cases in which the reflexes in the paralysed parts are not abolished. Except, indeed, for the fact that parts surrounding the cord are damaged, so that rest in one position is often indicated, the treatment of these cases after the first urgent symptoms have abated does not differ from that which is appropriate in other well-marked cases of paraplegia, where there is a tendency to the formation of sloughing bed-sores, and to the establishment of cystitis (see 9. Spinal Cord, Softening of).

H. CHARLTON BASTIAN.

4. **Spinal Cord, Slow Compression of.**—**SYNON.**: Chronic Traumatic Lesion of the Spinal Cord; Fr. *Compression Lente de la Moëlle Epinière*; (in part) *Paraplégie Douleureuse des cancéreux*; Ger. *Langsame Compression des Rückenmarks*.

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—The most frequent causes of the set of symptoms grouped under this head are to be found in diseases

of the vertebræ, and especially simple inflammatory or tubercular caries of the bodies of the vertebræ, leading to *angular curvature*, or 'Pott's disease.' Still, other kinds of disease of the vertebræ may also be productive of slow compression of the spinal cord, and of that form of localised softening of the organ which is so commonly met with in this class of cases, the so-called 'compression-mylitis.' Among these may be mentioned *cancer* of the vertebræ, either primary or secondary; also *exostoses* projecting into the spinal canal, or more irregular thickening of the bones in this situation. In cases of vertebral caries, in addition to the not infrequent presence and pressure of abscesses, a tough, yellow, scrofulous growth often infiltrates the posterior vertebral ligament, and thence spreads to the dura mater, here producing thickening and irregular fungosities which may press injuriously upon the spinal cord—more especially upon its antero-lateral columns. In these cases, though no tubercular disease is found within the dura mater, the spinal cord, as a result of degenerative changes, may, in the worst cases, be distinctly softened opposite, and perhaps for a very short distance above and below, the site of compression. At first such softening is principally apparent in the columns above mentioned; but in cases of longer duration it may involve nearly the whole thickness of the cord, and be followed by the usual ascending and descending 'secondary degenerations' (see p. 1523). The softened matter itself is generally reduced to an almost bloodless fluid or semi-fluid pulp, either of a whitish or dull yellowish-white colour, and there is generally no undue vascularity of the immediately adjacent portions in the cord.

In certain cases of very slowly induced compression no such softening of the cord is produced; there is rather a gradual atrophy or disappearance of the nerve-substance as the pressure increases, together with a sclerosis of what remains. This may occur, for instance, where the cord is pressed upon by some exostosis, or by irregular growth and thickening of the inner surface of the spinal canal, such as occurs occasionally in one or other of the cervical vertebræ.

It has long been known that no constant relation exists between the amount of angular curvature and of paralysis in different cases of vertebral caries. Paralysis may be absent where curvature is most marked. On the other hand, with no curvature and with only a slightly marked projection of one or two vertebral spines, paralysis may yet exist to a well-marked degree. This is due to the fact, that in such cases the cord is only very rarely compressed by the bones, while it is frequently more or less pressed upon by abscesses or by the yellowish growths which protrude from the inflamed or carious vertebræ, or which produce thickening and infiltration of the dura mater at the seat of disease; and changes of this sort may be well marked even where angular curvature is scarcely appreciable.

Again, where angular curvature is present, the posterior surface of the bodies of the vertebræ, corresponding with the angle, is often bent, rough, and eroded, and the cord over it is apt to become softened, though there may be no compressing growths or thickenings of the membranes.

Thus it happens that the paralysis in these cases may be variously produced. And seeing that it is often due to pressure by inflammatory products

rather than to pressure or irritation from the diseased bones themselves, we may the better understand the fact that occasionally a great improvement may set in and become established in regard to the paralysis, although the angular curvature of the spine remains as obvious as it ever has been.

In addition to slow compression of the cord resulting from diseases of the bones or the spine, a somewhat similar condition may be induced by the various kinds of tumours of the meninges, or by hydatid growths implicating these parts (*see* p. 1558). Confined within the narrow limits of the spinal canal, such tumours, even though of small size, may soon come to exercise a very injurious amount of pressure upon the spinal cord.

SYMPTOMS, COURSE, AND TERMINATIONS.—We shall point out some of the distinguishing characteristics of the paralysis which is often associated with vertebral caries, and afterwards refer to the peculiarities met with where meningeal tumours exist.

In *vertebral caries* with commencing pressure upon the spinal cord, the symptoms will be different, according to the part of the column implicated. The affection is frequently ushered in by an abiding pain in the spine and parts adjacent, often supposed to be 'rheumatic' in nature. Such pains commonly disappear when the patient is in the recumbent position, except during the acts of sneezing or coughing. They are commonly induced by particular kinds of movements, which are more or less difficult on this account. There is also some weakness in the lower part of the body and in the lower extremities. The mere 'weakness' may continue for weeks or even months before there is anything like actual paralysis; though at last this may show itself somewhat abruptly. The patient now becomes unable to stand, though still able to move the legs slightly while lying in bed. At this stage sensation is little, if at all, interfered with; but there may already be some increase in the readiness with which the knee-jerk manifests itself, and ankle-clonus may also be easily attainable. Next there may be startings of the limbs, and commencing rigidity of the muscles when passive movements are attempted; followed after a time by a more marked rigidity, which, when present in the calf-muscles, will often prevent the manifestation of ankle-clonus and of the knee-jerk. Later, if pressure increases either upon the spinal cord or upon its posterior nerve-roots, sensibility in its various modes becomes implicated. At this period the exaltation of the reflexes often diminishes. For a time the degree of impairment of sensibility and the freedom with which the knee-jerk and ankle-clonus may be obtained fluctuate. Meanwhile, painful spasmodic contractions of the legs (with flexion of hip- and knee-joints) becomes habitual, persisting through day and night with only rare intermissions.

Although there is some general wasting of the muscles, together with a flabby condition when they are relaxed, they still react almost normally to the faradic current. The skin is often dry and scurfy. The temperature of the limbs is generally slightly lower than normal.

At the first onset there may be for a few days a difficulty in voiding the urine, but this power soon returns, and it often continues long after the limbs have become powerless. The bowels are perhaps somewhat constipated, but there is no incontinence of *fæces*, unless diarrhoea supervenes from any

cause, or unless the reflex activity of the bowel is greatly exalted under the influence of aperient medicines.

The above condition of things may last long without much variation. But after a time there will be a gradual mitigation of the symptoms, or the reverse. In the latter case loss of voluntary control over the bladder and rectum appears; and (especially when the sensibility of the body and limbs becomes impaired) the tendency to the formation of sloughs and gangrenous bed-sores becomes increased. With these conditions other complications, such as cystitis, blood-poisoning, &c., may appear and greatly aggravate the condition of the patient, helping to bring about a more speedy termination.

In the case of *tumours arising from the meninges*, the onset of the affection may also be very gradual at first, though, perhaps, rather suddenly intensified at last. Here, however, the pressure very often comes upon the cord from behind, or it may at the same time implicate one or both lateral regions of the cord. At first, therefore, we commonly get variously impaired sensibility and neuralgic pains, or pains mixed with startings and cramp-like contractions in certain muscles, occurring in those particular regions of the body or limbs which are in relation with the nerve-roots slightly pressed upon and irritated by the new-growth. Great differences exist in different cases in regard to the degree and persistence of the initial pains. Subsequently these nerves and the cord itself may become more severely pressed upon, and then loss of sensibility over the field of distribution of the nerve-roots is met with, together with loss or impairment of sensibility in all or some parts of the body the nerve-supply of which is from the cord below the compressed region. With this a minor amount of motor paralysis also occurs, which, however, subsequently becomes more marked, and ultimately complete. When this takes place we have all the signs and symptoms met with in a case of total transverse softening of the spinal cord at the level implicated (*see* 9. SPINAL CORD, Softening of). This change is, in fact, commonly established by the persistence and increase of pressure due to the new-growth.

These are the broad outlines of the symptoms met with in such cases, which, of course, are subject to innumerable variations in individual cases, in accordance with differences in the region of the cord affected, together with the rate of growth, mode of incidence of pressure, and size of the tumour.

In *cancer of the vertebrae*, also, we have much the same grouping of symptoms; the preliminary pains being here especially severe.

DIAGNOSIS.—In the paralysis associated with *vertebral caries* the diagnosis depends upon the recognition of this causal condition, which, in the early stages, is often a matter of some difficulty. Much will depend upon the existence of pain in particular regions of the spine, or radiating therefrom; of pain which is relieved by the recumbent position, and greatly aggravated by coughing, sneezing, or stooping movements of different kinds, or from jarring of the spine by making sudden pressure upon the patient's shoulders. And yet in the absence of signs of caries, or of a tubercular habit of body or history, or of an exciting cause for caries, in cases where there may be little or no prominence of vertebral spines, and even no pain from firm pressure or the application of a hot sponge, we may be helped in our diagnosis of the existence

of caries by the distinctive characters of the paralysis itself, namely, its implication of motility principally, the exaggeration of the tendon-reflexes, the more or less marked rigidity of the legs, and the continuance of control over the bladder and rectum.

In cases of this type, or where there is only a slight prominence of two to four vertebral spines, it may be difficult, however, to establish a diagnosis between caries and *cancer* of the bodies of the vertebræ. It is true that a rounded prominence of several vertebral spines is met with in cancer more frequently than the angular projection commonly associated with caries; yet this single character will not always aid us: we must look also to the presence or absence of severe pains, to the clinical grouping of symptoms generally, and to the history of the patient, bearing in mind that it may be met with as a sequel of cancer of the breast.

The diagnosis of the other causes of slow compression of the cord to which reference has been made (exostoses or meningeal tumours) is usually a matter of extreme difficulty. We must be guided by probabilities based upon other associated states or conditions that may be recognisable in our patient, and also by the mode of onset of the affection.

PROGNOSIS.—We can only speak in general terms concerning the prognosis of the rather miscellaneous conditions which form the subject of this article. Cancer of the vertebræ or of the dura mater, compressing the cord, is the most serious of them all. The progress of such cases is usually both rapid and very painful, so that the end comes inevitably before many months have elapsed.

In vertebral caries associated with compression of the cord, the prognosis is extremely uncertain, especially where the disease occurs in the cervical region. It is also generally more grave where the pressure upon the cord is due to an abscess rather than to mere thickening of the dura mater. Under suitable treatment many of these cases practically recover more or less fully; in others complete transverse softening of the cord is produced. In the former class of cases the process of caries is arrested, the spoiled vertebræ are strengthened and bridged over by growth of new bony tissue (though, of course, the angular curvature of the spine remains), while recovery from the paralysis may be more or less complete. This latter kind of recovery takes place occasionally even after paralysis, with almost persistent contractions of the lower extremities, has existed for from twelve to eighteen months or even longer.

In cases of exostosis, hydatids, or meningeal tumours compressing the spinal cord, the prognosis will depend upon the part of the cord involved, upon the rate of increase of the symptoms of compression, and upon the extent to which a secondary myelitis or softening is established. The disease in these cases, in spite of stationary periods, or even those of slight improvement, is more or less continuously progressive, though it may last for many months, or, occasionally, even for a year or two. Some of the complications or accidents incident to the paralytic condition ultimately bring the patient's life to a close.

TREATMENT.—Rest in the recumbent or in the prone position is, of course, absolutely essential in cases of vertebral caries or of cancer of the vertebræ. In vertebral caries the mere rest in bed must often be supplemented by extension of the spine—that is by

fixation above, together with weights attached to the feet. In other cases of vertebral caries, a poroplastic or plaster jacket may be needed, in order more effectually to secure absolute immobility of the affected portion of the spinal column. This, however, would have to be reserved for the more chronic cases or stages—for those in which active local treatment was no longer considered to be necessary or desirable, though a prolonged rest in bed may still be needed. Under such conditions the application of the jacket is a means of obtaining more complete rest for the damaged spine. Some surgeons, however, prefer to have recourse to the method of 'elastic extension' above referred to for procuring this fixation of the spinal column.

In cases of paraplegia associated with vertebral caries, the patient's general health requires the utmost attention during the period in which we are endeavouring to check the disease by the influence of rest. Good nutritious food and cod-liver oil will be required, combined with steel wine or the syrup of phosphate of iron. In some cases 10- to 20-grain doses of iodide of potassium (together with iodide of iron or small doses of perchloride of mercury) seem to do good. Where the patient suffers much from painful contractions of the legs (which may greatly disturb sleep), these may be very distinctly lessened by suspending weights to the feet (each being from two to four lbs.) from leather anklets, by a cord which passes over a pulley. Such weights may be used for weeks or months together, where necessary.

In regard to local measures, counter-irritation of some kind is generally had recourse to, either in the form of flying blisters near to and on each side of the portion of the spinal column which is affected, or else by the renewed application of moxas or the actual cautery to these regions. The latter more severe measures are still recommended by some authorities, though the experience of others is against their employment, as being of little or no use, and therefore adding needlessly to the sufferings of the patient. The writer is inclined to think that all the good which moxas or the actual cautery are intended to bring about may generally be as effectually achieved by the application of the strong iodine-solution of the pharmacopœia over the affected region of the spine about every fourth day.

There can be no doubt that treatment of this kind is often successful, though frequently only after prolonged periods. The question, therefore, naturally presents itself, whether shortening the duration of the illness, and in some cases the saving of life, may not be brought about by an early resort to surgical operation, with a view to opening any abscess that may exist and, by laminectomy, diminishing pressure upon the spinal cord. If some signs of recovery do not commence in three or four months—and especially if there are marked sensory defects, showing that the cord is severely pressed upon—it will probably be advisable to have resort to operation. Each case, however, will require anxious consideration in regard to this point.

Again, it must be borne in mind that some cases of compression of the spinal cord by meningeal growths may also be greatly relieved for a time, or even cured, by operative measures.

In the case where a hydatid tumour presses upon the spinal cord, and is also situated in part outside the vertebral spines, tapping might bring much relief. In the majority of the other conditions

comprised within the limits of this article, little can be done to cure the state of things which is the cause of the spinal disease, so that it would only remain for us to treat the paraplegia and its attendant conditions upon the general principles applicable to them. These are fully considered under 9. SPINAL CORD, Softening of. H. CHARLTON BASTIAN.

5. **Spinal Cord, Anæmia of.**—Anæmia is not to be considered as the basis of any ordinary or common disease of the cord: in other words, there is no definite group of symptoms the existence of which is likely to be recognised more than once in a lifetime in any actual patient, which would justify the diagnosis 'anæmia of the cord.'

First, the writer would repudiate the notion that *anæmia* or *chlorosis*, as a mere blood-disease, is capable of producing, on the side of the spinal cord, any set of symptoms which can be marked off from those characterising the condition as a whole. In these diseases the functions of all the organs are impaired by reason of the impoverishment of the blood. The brain and spinal cord, on account of the delicacy of their functions, will, of course, suffer to a notable degree; and when general debility is extreme, a paresis of the lower extremities may be notable beyond that of other parts of the body, because the legs in standing or in walking have to support so great a weight. Where anything more than such paresis exists—that is, where there is actual paraplegia, such symptoms are not to be explained by a mere anæmia of the cord. Other causes are to be looked for. Jaccoud's whole group of *paraplégies dyscrasiques* will probably disappear before a more thorough knowledge of the actual mode of causation of these and many other obscure forms of paraplegia. We now know that in association with some cases of pernicious anæmia there is most marked degeneration of the lateral and posterior columns of the cord of comparatively rapid onset. See COMBINED DEGENERATION OF THE SPINAL CORD.

Secondly, *embolism* and *thrombosis* of spinal arteries will produce temporarily, and in quite limited regions of the cord, a condition of anæmia. Such local anæmia would probably soon be rectified by the establishment of a collateral circulation; and in the event of this not taking place, local 'softening' of the organ would ensue. A paralysis owning such an origin would not, therefore, be spoken of as resulting from 'anæmia of the cord.'

Thirdly, *pressure* upon parts of the cord will occasion anæmia and ultimately softening, but the symptoms in a case of this sort will depend mainly upon the pressure itself interfering with the functions of the nerve-tissue thus affected.

Beyond the conditions above referred to, there is the possibility that definite groups of paralytic symptoms may be occasioned by anæmia induced by mere functional *spasm of the arteries* in certain regions of the cord—spasm, that is, which persists day after day. This has been supposed by Brown-Séquard to be the condition existing in the cases of so-called 'reflex paraplegia' (see No. 28), and also in some forms of functional paralysis of spinal type (see No. 30). If such a condition of persisting arterial spasm be possible, and an actual cause of paralytic symptoms, we may well ask whether it too ought not after a time to lead to actual softening, or at least to some degree of degenerative

change in the spinal cord. It seems possible, however, that where the amount of blood sent to a part of the cord is but small, the degradation of function may be more marked than the degradation of structure.

There will still remain a very few exceptional cases, in which a condition of real anæmia of the spinal cord is brought about in man, just as it has been brought about in some of the lower animals whose abdominal aorta has been tied or compressed. When the blood-supply is thus suddenly cut off from the lumbar region of the cord in animals, their hinder limbs become paralysed almost immediately, and continue paralysed as long as the blood-supply of the cord happens to be arrested. But if, after a mere brief interval, the blood is again allowed to take its natural course, the temporary paralysis disappears completely in a very short time. A condition of this kind seems to have occurred in a patient formerly under the care of Gull, who suddenly became paraplegic, apparently owing to an abrupt arrest of the blood-current through the abdominal aorta, as was indicated by the cessation of the femoral and other pulses in the lower extremities. The man continued paraplegic for months, and only recovered when the collateral circulation became, after a time, pretty fully established. In a very few other cases referred to by Erb, in which paraplegic symptoms were associated with an obstruction of some kind in the abdominal aorta, he thinks that these symptoms, supervening as they did rather less suddenly, may have been in great part due to the deficient blood-supply to the muscles and nerves of the lower extremities, rather than to anæmia of the cord—to a peripheral, that is, rather than to a centric anæmia.

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6. **Spinal Cord, Hyperæmia of.**—This condition again is more frequently talked of than it deserves—looking to the small amount of positive knowledge we possess upon the subject.

Hyperæmia of the cord must be either passive or active, that is, it must be a result of *mechanical congestion* or of *arterial determination*.

Mechanical congestion.—In obstructive heart-disease extreme congestion of the spinal cord may exist for months without producing any distinct symptoms of disease of the spinal cord. A constantly congested spinal cord would doubtless perform its functions in a less vigorous manner than natural, but such effects would be slowly evolved and comparatively obscure. After a long time the effects might become more marked, owing to the overgrowth of connective tissue within the organ. We may indeed have the starting-point of a general sclerosis of the spinal cord under such conditions; but this secondary change, when only slightly marked, may, even in the spinal cord, produce no very definite symptoms.

General mechanical congestion of the cord is probably more frequent and more easily brought about than a congestion involving parts of the organ. Still from various causes there may be undue pressure upon certain veins, which directly or indirectly convey blood away from special regions of the cord and its membranes. Such an event cannot, however, be regarded as a likely cause of a congestion productive of morbid spinal symptoms, if we consider the absence of distinct symptoms resulting from extreme general congestion of the cord; and

also the fact of the very free anastomosis of all the spinal veins.

Active hyperæmia may in its origin be of two kinds—'reflex' or 'inflammatory.'

'Reflex' hyperæmia of the cord and its membranes is possibly a phenomenon of great frequency, manifesting itself locally in certain regions—the seat of the process varying according to the conditions under which it arises. It might be immediately caused by vaso-motor paralysis, implicating certain vessels of the cord and their branches; and would thus involve an increased afflux of blood to the tissues contained in the corresponding vascular territories. We know that such an increased afflux of blood may exist in other tissues for some time without inducing tissue-changes of an appreciable kind (Brown-Séquard). It is fair to suppose, moreover, that any symptoms induced by such increased afflux of blood to certain regions of the cord would be indicative of exalted rather than of depressed function; for example, hyperæsthesia, actual pain and spasms, or increased reflex excitability, rather than their opposites.

In weak and irritable states of the nervous system it is quite possible (though nothing more definite can be said) that such vaso-motor paralysis, and also vaso-motor spasms inducing localised anemias, may manifest themselves in spinal vessels as they do in cutaneous vessels, by familiar flushes or pallors. If occurring in the spinal cord, however, these would mostly be temporary phenomena, and not capable of producing the symptoms of an abiding disease. How frequent such reflex local hyperæmias (whether brief or prolonged) may be in the spinal cord, and in what precise manner they are excited, we do not know. Suppression of the menses or of hæmorrhoidal fluxes, the presence of worms in the intestine, the prolonged incidence of cold and wet, or severe concussions of the spine, any or all may operate in this particular manner—but for proof that, as matter of fact, they do, we may look for evidence in vain.

The subject of 'inflammatory hyperæmia' will be briefly considered under the next heading. In this case, in addition to changes in the vascular system, the effects of the inflammatory process as a whole have to be taken into account. Even in the first stage of inflammation something prior to and beyond the mere 'active' congestion has to be thought of.

From what is said above, it may be seen how shadowy is our present knowledge concerning the existence of any definite sets of symptoms which can be ascribed to non-inflammatory hyperæmia of the cord, either general or local.

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7. Spinal Cord, Inflammation of.—SYNON.: *Myelitis*; *Myelitis Acuta*; Softening of the Spinal Cord (in part); Fr. *Myélite*; *Myélite aiguë*; *Inflammation de la Moelle Epinière*; *Ramollissement de la Moelle Epinière* (in part); Ger. *Myelitis*; *Rückenmarksentzündung*; *Erweichung des Rückenmarks* (in part).

NATURE, ÆTIOLOGY, AND PATHOLOGY.—To speak definitely on this subject, in the present state of knowledge, is extremely difficult. This is due to several causes. In the first place, it is owing to the fact that so much uncertainty exists in the minds of many eminent pathologists and physicians as to what ought rightfully to be included under this

term; and, secondly, because by a very large number of writers the term is understood and used in the vaguest manner, but with a manifest tendency to comprise under it the largest possible number of affections of the spinal cord. Critical discrimination seems to have been, and still to be, in abeyance with many who describe or report cases of disease of the spinal cord. They set down as instances of 'myelitis' not only all cases in which the substance of the spinal cord is softened, but still more all those in which it is indurated—and, no less impartially, those in which it is merely degenerated.

(1) The notion that common 'softenings' of the spinal cord are of inflammatory origin has persisted with little alteration, although for nearly thirty years pathologists have been interpreting altogether differently the mode of production of apparently similar 'softenings' of the cerebrum and cerebellum. Can it be that 'softening' as it occurs in the majority of cases in these latter organs is of non-inflammatory origin; while in the majority of apparently similar cases occurring in the spinal cord, the process is really inflammatory in its nature? It is true that in association with the majority of cases of primary softening in the brain thrombosis or embolism of its arteries may be detected, and that, owing to the peculiarities of its vascular supply, no such occlusions of vessels can often be detected in association with softening of the spinal cord. See p. 1522.

(2) Then, again, perhaps it may be said that without adequate cause the very localised changes occurring in and around the great ganglion-cells of the anterior cornua, in 'acute' and 'chronic spinal paralysis,' have been set down as inflammatory in their nature, and new names have been given to these affections, tending to ratify this view as to their origin. Thus they are spoken of by some as cases of *anterior polio-myelitis*, or more briefly, and, so far, better, as cases of *cornual myelitis*. But localisation of an inflammatory process to great ganglion-cells and their immediate surroundings at present constitutes a rather unintelligible process to many pathologists. And mysterious as these particular changes are, from the point of view of their ætiology, on any hypothesis that has yet been started, it would perhaps be simpler, and more harmonious with the nature of the observed conditions themselves, to regard them as of a degenerative type. If the slower and more isolated changes characteristic of 'progressive muscular atrophy' are to be placed in this category (and in regard to them there is absolutely no evidence either clinical or pathological that can be adduced in favour of an inflammatory origin), then also it becomes easy to believe that, under some at present imperfectly defined toxic conditions, a change of the same kind may set in more rapidly in these, the most specialised of all the anatomical elements met with in the spinal cord, so as to produce the more acute affections above referred to. The slight secondary overgrowth of neuroglia commonly occurring around the degenerated ganglion-cells does not in the least militate against this view as to the pathology of the process; a similar secondary change occurs also in the process next to be referred to, and will be found to be easily explicable without the necessity of having recourse to the ever-ready and fashionable hypothesis of inflammation.

(3) 'Secondary degenerations' of the spinal cord

have indeed, in spite of their name, and of what is known as to their origin, been erroneously regarded by some writers as inflammatory changes. When nerve-fibres are cut across, those portions which are severed from their connection with certain ganglion-cells are no longer able to preserve their nutritive integrity. Simultaneously throughout their whole length fatty degeneration affects their white substance. Myelin breaks up, and becomes disintegrated as it does in non-inflammatory softenings in the brain; and very speedily granulation-corpuscles begin to form abundantly throughout the changing area. But though fatty degeneration thus occurs simultaneously in all the cut fibres of the band, the vascular supply of this tract of tissue has not been altered. Since the blood in the diseased area is not utilised by the nerve-tissues proper, except to a very small extent, a large excess of nutriment is placed at the disposal of the neuroglia, and this undergoes a well-marked hyperplasia. Thus a band of tissue-change is produced in which some of the characteristics of softening are blended with those pertaining to a patch of sclerosis. In brief, we have effects resulting from a primary fatty degeneration of the nerve-fibres, and a secondary hyperplasia of the neuroglia; and from first to last there is not the least reason for believing in the existence of an inflammatory process.

(4) If we turn now to 'sclerosis' of the cord of primary origin, we again meet with processes which are commonly regarded and described as forms of '*chronic myelitis*.' This kind of nomenclature is objectionable as applied to the processes in the spinal cord, just as it is in its application to like processes occurring in other organs, as the liver, the lungs, or the kidneys. Fibroid overgrowth, which forms the basis of so many examples of 'cirrhosis' or 'sclerosis' in different organs and tissues of the body, is a process pathologically intermediate between inflammation on the one hand and degeneration on the other. What were formerly named 'interstitial inflammations' are now the 'non-inflammatory hyperplasias' of some pathologists, and the 'fibroid degenerations' of others. It would seem that the view as to the inflammatory nature of such processes is erroneous, if we look either to what is known concerning their modes of initiation, or to the actual nature of the changes themselves (which agree in many particulars with those of infiltrating new-growths); it would seem, moreover, not less erroneous if we look to the clinical history of the affections themselves in which these sclerosis occur. It conveys, therefore, an altogether erroneous implication to speak of such mere fibroid overgrowths as so many instances of 'chronic myelitis.'

Thus, it will be seen that the writer attributes to inflammation a far more restricted rôle in the production of morbid conditions of the spinal cord than is customary. The various forms of so-called 'chronic myelitis' he would exclude from that category. He would do the same for the set of changes known as 'secondary degenerations'; and also for those which are characterised by more or less acute degenerative processes implicating the great ganglion-cells of the anterior cornua.

Of the processes above referred to in order, there remains, therefore, only the class of 'softenings' of the spinal cord. That many of these are of a simply degenerative type (due to vascular occlusions and disturbances of blood-supply), and that, in the

great majority of cases, these are the instances in which 'softening' appears to occur as a *primary* process, is the view which seems to the writer most in accordance with existing knowledge. On the other hand, it seems clear that in many cases changes, truly inflammatory in their origin and progress, may terminate in the production of states of 'softening' of the cord, which are indistinguishable by the naked eye from the softenings of degenerative type, and which, except in their early stages, can as yet also be very imperfectly discriminated by the microscope.

These latter true *inflammatory softenings* very rarely occur as primary pathological states, and then principally as sequelæ of one or other of the acute infective diseases; they are met with far more commonly as *secondary* changes.

Thus we may get inflammatory softenings (a) spreading around and from wounds or other traumatic lesions of the spinal cord; or (b) starting from some blood-clot or tumour situated in or pressing upon the substance of the cord. It is not by any means clear, however, that all the forms of softening which arise in the latter manner should be regarded as of an inflammatory nature; and much room for doubt also exists as to the real pathogenesis of many cases of so-called 'compression-myelitis.' Again, inflammatory softenings, going on even to suppuration, may be set up in the spinal cord (c) by embolism or thrombosis of septic origin. Still another cause of true inflammatory changes in the spinal cord (*myelitis peripherica*) is to be found (d) in spinal leptomeningitis. See MENINGES, SPINAL, Diseases of: Leptomeningitis.

Suppuration is clearly a process of inflammatory origin, and might therefore be expected to occur occasionally in the midst of 'softenings' which result from inflammation. In the light of what has been said above, the following statement by Erb is of considerable interest. 'Actual suppuration occurs very rarely,' he says, 'in acute myelitis. When abscess of the cord does form, it is generally secondary to a severe traumatic lesion or to suppurative meningitis. In spontaneous myelitis, on the other hand, suppuration is exceedingly rare, and has only been observed in a very few cases.' Thus suppuration is met with just in those forms of softening which are undoubtedly of inflammatory origin; and, on the other hand, it is not met with in the ordinary cases of primary or spontaneous softening, here assumed to be of non-inflammatory nature.

One other condition requires to be referred to here, and that is the so-called *acute central myelitis*. These are cases in which apparently spontaneous 'softening' is met with, implicating in the main the central grey matter, and that often through a considerable extent of the cord. At times, however, the softening extends beyond the grey matter, so as to involve more or less of the surrounding white substance, when it has been termed *myelitis diffusa*. Considerable obscurity still prevails in regard to the ætiology of these affections. In some cases, such a change has been met with as part of an infective process, in which minute vessels in the grey matter of the cord have been found obstructed with micrococci. Occasionally, moreover, in certain at present imperfectly known conditions, minute thromboses may, as Hamilton has shown, occur throughout the spinal cord, and

more especially in its grey matter, and thus lead on to the production of a central softening. In this latter case, the patient was suffering from pyelitis, and it is supposed that there may have been some blood-poisoning. Still it was not ascertained that the multitudes of minute thrombi were either associated with or caused by micrococci in the vessels. It appears probable, however, that if from any cause minute widespread obstructions of small vessels occur in the spinal cord, softening would take place principally in the grey matter, owing to its greater vascularity. We should thus get that particular distribution of this change which is met with principally in cases of so-called 'acute central' or 'diffuse myelitis.'

A careful study of the two cases of this disease recorded by Hayem has by no means sufficed to convince the writer that they ought to be regarded as having had an inflammatory origin. Neither the mode of onset nor the symptoms of the disease lend any distinct support to this view; nor do the results of the elaborate examination, to which the spinal cords were submitted by this accomplished observer, at all satisfy the writer that the pathological conditions met with were inflammatory either at their commencement or in their subsequent progress. See 9. SPINAL CORD, Softening of.

SYMPTOMS, COURSE, AND TERMINATIONS.—From what has been said, it will be seen that true inflammatory conditions of the cord are only rarely of *primary* origin, and that they occur, for the most part, as *secondary* complications in association (a) with wounds or injuries of the cord; (b) with foreign bodies in its substance; (c) with pyæmic processes; or (d) with spinal leptomeningitis, either simple or tubercular.

The supervention of a real myelitis in the course of either of these diseases of the spinal cord would perhaps be associated with an exaggeration of the already existing febrile condition; with an increase in the amount of paralysis, and in the degree of interference with sensibility; possibly also with more pain, restlessness, and spasms.

Myelitis may become associated with more or less of distinct suppuration, and almost certainly goes on to the formation of well-marked foci of softening. These may remain limited in site, but occasionally they have a distinct tendency to spread above and below the original seat of injury or disease. Such deposits would probably undergo subsequent changes, very similar in kind to those which occur in foci of non-inflammatory softening.

DIAGNOSIS.—All that can be said under this head has been referred to above in connection with the symptoms characterising the supervention of myelitis.

PROGNOSIS.—The gravity of any wound or lesion of the spinal cord, or attaching to the presence therein of blood-clot or tumour, is, of course, greatly increased by the supervention of inflammatory changes about their immediate confines. Again, the fact that an inflammation of the spinal meninges is complicated with similar changes in the substance of the spinal cord itself, cannot fail greatly to aggravate a case of simple spinal leptomeningitis. For, even should recovery from the acute affection take place, the actual degree of abiding paralysis, ataxy, or impairment of sensibility would much depend upon the degree in which the substance of the spinal cord had been itself implicated.

TREATMENT.—The amount of power that we possess in controlling an inflammatory condition of the spinal cord is probably not great. Little, if anything, is at present to be done with mere drugs. The patient should, if possible, lie in the prone position, or, failing this, on his side, with absolute rest. The advisability of abstracting blood locally by cupping or leeches should be entertained, and must depend upon the amount of local pain or tenderness. In some cases it seems to be of service. Or we may trust rather to the application of cold externally, in the form of ice-bags, along the spine. At the same time the patient should be kept upon spoon-diet, with a sparing amount of stimulants; and the bowels should be relieved by the aid of copious warm enemata, which may also act usefully as derivatives. The limitations circumscribing our efforts at direct therapeutics must be compensated as far as possible by attention to the state of the general health, and by the most careful and assiduous nursing, in the hope that the morbid process may after a time abate, and that, in the absence of collateral complications, the patient may make at least a partial recovery.

H. CHARLTON BASTIAN.

8. Spinal Cord, Hæmorrhage into. —
SYNON.: *Hæmatomyelia*; *Hæmorrhagia Medullæ Spinalis*; Spinal Apoplexy; Fr. *Hématomyélie*; *Apoplexie de la Moelle Epinière*; *Des hémorrhagies intrarachidiennes*; Ger. *Rückenmarksapoplexie*; *Spinalapoplexie*.

ETIOLOGY AND ANATOMICAL CHARACTERS.—Hæmorrhage into the spinal cord is a comparatively rare event. It occurs under three different conditions, namely: (1) as a result of concussion or violence; (2) as a secondary event, consequent upon some definite pre-existing morbid condition; and (3) as a primary event, or local pathological accident.

We are here specially concerned with hæmorrhages into the spinal cord belonging to the third of these categories, and may in a few words dismiss the other two.

(1) Traumatic hæmorrhage, small in extent, may, as already stated, occur in almost any region or part of the cord as a result of some severe concussion (see 1. SPINAL CORD, Concussion of). Again, it may occur in the grey matter, and even in the white substance to a smaller extent, close to and as an appanage of wounds of the cord. In each of these cases symptoms due to the hæmorrhage itself would probably be obscured by the general set of symptoms resulting from the concussion or injury.

(2) Secondary hæmorrhages are, however, more closely connected, from the point of view of symptomatology, with those forming the special subject of this article. During the growth of certain soft tumours in the cord, a rupture of some of their vessels may take place, so as to cause hæmorrhage either into the growth itself, or else into contiguous regions of the cord. Such an event would be signalled clinically by the sudden exacerbation of the symptoms previously existing. But a combination of greater importance, though one of considerable obscurity, consists in the co-existence of a 'central myelitis' of the grey matter of the cord through more or less of its length, with a central hæmorrhage of nearly similar extent. The existence of any such 'central myelitis' as an independent disease of the cord seems to the writer very doubtful.

It is at least equally probable that the hæmorrhage has been primary, and that the 'myelitis' is of secondary origin around the blood-clot. It need not be denied, of course, that in other cases hæmorrhage does occur occasionally into the midst of a focus of softened tissue in the spinal cord, just as it occurs occasionally under similar conditions in the midst of softened brain-tissue. Again, hæmorrhage may take place into or on the confines of a soft sarcoma or glioma in the substance of the spinal cord.

(3) Primary hæmorrhages differ as regards the amount, the site, and the distribution of the blood effused in different cases. In connection with scorbutic states, and also independently of these, small hæmorrhages may occasionally occur in the substance of the cord, without producing any very distinct symptoms. But, at other times, a comparatively large quantity of blood may be effused into the cord, and then it occurs almost invariably into the central regions of the grey matter, through which it may extend for a variable distance, upwards or downwards, or both. When the quantity is smaller, the blood may be effused into the grey matter of one side only.

Though this kind of hæmorrhage is, in contradistinction to the others, spoken of as primary, yet it is almost invariably preceded by some pathological changes in the vessels of the cord. These constitute the predisposing conditions, and the actual rupture takes place, rarely, when the person is at rest, but more frequently under the influence of some distinct exciting cause—such as muscular exertion of one kind or another.

Primary hæmorrhage, though rare, is most prone to occur in persons between the ages of twenty and forty, and not with increasing frequency as age advances. This constitutes a further notable difference between hæmorrhages of the spinal cord and those into the brain.

SYMPTOMS.—These are necessarily subject to great variations, according to the amount of blood effused, and as the hæmorrhage takes place into the cervical, the dorsal, or the lumbar region respectively. The kind of variation thus induced may be gathered by reference to the article, *SPINAL CORD, Diseases of*, pp. 1526 to 1531.

Here it is of importance to set forth the peculiarities (both as regards mode of onset and nature of the symptoms) which belong to hæmorrhage as compared with other pathological conditions of the cord. First, its tendency is to take place suddenly and without warning; and, secondly, for the blood to be effused into the grey matter for some distance, thus giving rise to a characteristic grouping of symptoms. There may, therefore, be a sudden onset of pain in the back (possibly severe), followed almost immediately by complete motor and sensory paralysis of the legs and trunk up to a certain level, together with complete paralysis of the bladder and rectum. At first there may be an abolition of all reflexes, and possibly a lowering of temperature in the legs; though after a day or two—should the injury be in the dorsal or lower cervical region of the cord—there may for a time be increased heat of legs, owing to vaso-motor paralysis, and a return, with some exaggeration, of various reflexes. Rapid atrophy, with the appearance of the electrical 'reaction of degeneration,' occurs in all muscles that are in immediate functional relations with the portions of the anterior cornua of the cord that may be damaged. Cystitis, together with sloughing bed-

sores and all their consequences, tends to occur early, and that often in spite of all precautions that may be taken.

Where the hæmorrhage invades pretty fully, but is limited to, the grey matter of one-half of the cord, we may have groups of symptoms that take the form of *hemiplegia spinalis* or *hemiparaplegia*. See 2. Punctured or Gunshot Wounds.

DIAGNOSIS.—The absolutely sudden onset of the paralysis, which may be complete in the lower extremities in the course of a few minutes (especially when associated with a sudden painful sensation in the back, or one which radiates into the limbs), as well as the almost complete and sudden loss of sensibility in the paralysed parts, form a group of symptoms which are typically distinctive of hæmorrhage into the grey matter of the cord.

The condition most likely to be confounded with it is a large hæmorrhage outside the *dura mater*, causing compression of the cord. Here the onset would also be sudden, but almost invariably associated with some mechanical injury or shock. The paralysis of motion, too, would generally be much more marked than the interference with sensibility. The subsequent progress of such a case would further tend to separate it from a case of intramedullary hæmorrhage, since (even with a severe meningeal hæmorrhage in the cervical region), if the patient should survive the first effects of the lesion, the symptoms might be expected soon to grow less and less urgent, and recovery may be more or less complete. No such amelioration is, however, to be expected in the case of a well-marked hæmorrhage into the grey matter of the cord, in the cervical region or elsewhere.

On the other side of the brain *embolism* is capable of initiating paralytic symptoms with as much suddenness as a hæmorrhage, but in the spinal cord, for reasons previously stated, this does not occur. See *SPINAL CORD, Diseases of*, p. 1522.

It does, however, happen occasionally that a process of softening—probably caused by *thrombosis*—has its occasioning conditions initiated suddenly. When this occurs, paraplegia sets in almost as abruptly as if it were produced by hæmorrhage; but then it is usually an incomplete paraplegia, and, for a time at least, unaccompanied by loss of sensibility. In the course of a few days, in such a case, sensory paralysis may supervene, and the motor paralysis may become more complete. In the exceptional cases of paraplegia of sudden onset due to this cause, there is generally no initial pain in the back, though there may be pains and burning sensations in the limbs.

PROGNOSIS.—Where the hæmorrhage is at all large, so as to extend through the grey matter for the distance of an inch or more, the prognosis is always grave. Very few of such cases recover. They are, in fact, liable to be aggravated by the establishment of a secondary process of softening in the grey matter, which may slowly extend both above and below the blood-clot as well as around it. Should this softening reach far into the cervical region, or should the hæmorrhage itself implicate this part of the cord, the patient may not survive more than a few days. But if the primary and secondary pathological changes are limited to the lumbar or to the dorsal region of the spinal cord, the fatal event is usually brought about more slowly, after an interval of weeks or perhaps even of months—and then commonly from the occurrence

of sloughing bed-sores, together with cystitis and other frequent accompaniments of a severe paraplegia.

In the case of small hæmorrhages limited to some fractional part of the transverse area of the cord, and of slight longitudinal extent, the prognosis is, of course, much more favourable, and there is no reason why partial recovery, at least, may not occur.

TREATMENT.—In the treatment of a case of spinal hæmorrhage, should the patient be seen immediately after its occurrence, absolute quietude, with rest in the recumbent or prone posture, should be ensured.

Bleeding, either local or general, is useless. Purgatives also are contra-indicated.

Should the pulse be full, and the heart's action excited, decided benefit may be derived from 10-minim doses of tincture of digitalis, in combination with 15 or 20 grains of bromide of potassium, given for the first three doses at intervals of three or four hours, and subsequently every six or eight hours, for two or three days. These drugs will also favour sleep, and exercise a general calmativè influence.

Position and rest are perhaps the means to be principally relied upon to prevent a recurrence or continuance of the hæmorrhage: such measures may be supplemented by warm applications to the feet and calves of the legs; though the patient should in other respects be kept perfectly cool. Ice to the spine may be applied, but is of doubtful utility. Spoon-diet should be strictly enjoined for a few days at least.

The patient's urine will require to be drawn off by catheter; and extreme care ought to be taken to ensure the antiseptic cleanliness of the instrument. After a day or two, if the bowels have not been moved, a laxative or an enema should be administered, since, as in many other forms of paraplegia, there may, at first, be obstinate constipation rather than incontinence of fæces.

Subsequently, the case requires to be treated in all respects like any other very bad case of paraplegia—extra precautions being observed throughout, in order, as far as possible, to guard against the onset of bed-sores and cystitis. Fuller details concerning such treatment will be found under 9. SPINAL CORD, Softening of, since this is by far the most common cause of paraplegia.

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9. Spinal Cord, Softening of.—**SYNON.:** Non-inflammatory, White, or Simple Softening; *Myelomalacia*; *Mollities Medullæ Spinalis*; Acute Myelitis (in part); Compression-Myelitis (in part); *Fr. Ramollissement de la Moelle Epinière*; *Ger. Erweichung des Rückenmarks*.

NATURE OF CHANGE.—The writer has already intimated his opinion that far too large a share is assigned to inflammation in the pathogenesis of diseases of the spinal cord. This mistake is particularly obvious in regard to acute inflammations. It has long been the fashion to speak of almost every focus of 'softening' that occurs in the spinal cord as being the result of an 'acute myelitis'; and we find even Erb putting forward, as characteristics of an inflammatory softening, peculiarities which certainly ought not to be regarded in such a light—and this although he seems otherwise strongly inclined to hold a similar opinion to that above expressed. While admitting that a

true myelitis is not distinguishable macroscopically, in the great majority of cases, from a simple or non-inflammatory softening, Erb adds a statement to the effect that the 'microscopical examination can alone furnish conclusive evidence.' In the opinion of the writer, however, such evidence as that which is cited by Erb is quite inconclusive.

It is evident, indeed, that we are still almost as destitute of microscopical as we are of macroscopical characters, of a trustworthy description, for enabling us to decide whether any given focus of softening has been of inflammatory or of simple non-inflammatory origin. In the early stages, or on the confines of a focus of inflammatory softening, we may expect to find a very distinct infiltration of the tissues with leucocytes, which would, of course, at once give a key to the true nature of the change in question. In the present state of knowledge, however, it would appear that the 'non-inflammatory softenings' of the cord are represented by the great majority of the primary and apparently idiopathic 'softenings' which frequently occur in this organ.

ÆTIOLOGY AND PATHOGENESIS.—Concerning the ætiology of non-inflammatory softening of the spinal cord, it is impossible to speak positively. The disease presents itself as a spontaneous or idiopathic affection, sometimes without apparent cause or definite antecedent conditions of any kind, but at others as a sequel of one or other of various known and common precursors of diseases of the spinal cord.

Thus in certain cases the symptoms set in more or less suddenly after some great bodily fatigue; in others after extreme sexual excesses; or they may occur during the period of convalescence from certain acute fevers, such as variola, typhus, and other exanthemata, or after rheumatic fever. During the first week or two after childbirth there is likewise a liability to such symptoms; and also in the later stages of syphilis. These different conditions may act very variously in contributing to bring about a focus of softening in the spinal cord, and nothing more than conjectures can be advanced in regard to its pathogenesis in the several cases. It may be recognised, however, that most of them tend to entail cardiac weakness, either alone or in association with blood-changes which may favour the occurrence of thrombosis.

Again, the symptoms indicative of a primary softening of the cord may set in after the action of other conditions, regarded by some as exciting rather than as predisposing causes. Of these the following may be enumerated: Prolonged exposure to cold and wet; sudden suppression of the menses or of other accustomed fluxes; violent emotional disturbances; or the existence of some inflammation in one or other of the pelvic organs, such as the uterus or the bladder and urethra (instances of the latter class being some of the cases formerly supposed to be of 'reflex' origin). In regard to these 'exciting causes,' all that is certainly known is, that softening of the cord seems to set in not infrequently in persons who have been subjected to one or other of them; but in what precise mode either of them is related to the subsequent softening, nothing very definite can be said. Something, nevertheless, may be advanced by way of suggestion, with the view more especially of giving direction to the investigations needful for clearing up this subject.

Spinal and cerebral softenings probably own a

similar mode of origin. Of the obstructions of vessels which so largely determine cerebral non-inflammatory softenings, it is those due to thrombosis, rather than to embolism, which intervene in the main for the production of corresponding conditions in the spinal cord. See SPINAL CORD, Diseases of, p. 1522, (8) and (9).

It is well known that the causes of thrombosis are principally three, and that in different cases, now one, now another of them may be most influential; while in other instances two or more of these causes may co-operate. These three causes are—(a) thickenings, irregularities, or degenerations of the inner coats of the vessels; (b) slowness of blood-current; (c) peculiarities in the chemical composition of the blood, rendering it more than usually prone to coagulate, or the occurrence of micro-organisms therein.

The thrombosis may take place in the arteries or in the veins, and the plexiform arrangement of the spinal vessels, together with the slowness of their blood-current, may favour the occurrence, as well as the spread of the process when it has once been initiated. Thus a process of coagulation, beginning, perhaps, in some very small vessel, may gradually extend so as to involve many other contiguous branches, and thereby gradually increase the area of the cord which is deprived of its proper blood-supply. The small size of the vessels, however, makes it extremely difficult to establish the fact of their occlusion, whereas in the large vessels at the base of the brain, thrombosis is easily enough detected. And it is especially worthy of note, in this connection, that the blood-supply of the lower end of the cord (where primary softenings are most common) is peculiar and easily interfered with. In short, the anatomical conditions existing in the cord, both on the arterial and on the venous side of its circulation, are probably of a kind distinctly to favour the occurrence of thrombosis.

ANATOMICAL CHARACTERS.—In regard to their distribution or extent in the cord many varieties of softening exist which have been commonly recognised, though they have been mostly described under corresponding designations as so many varieties of 'myelitis.' Thus, we may have a 'complete transverse softening,' involving the entire thickness of the cord for a variable longitudinal extent, either in the lumbar, the dorsal, or the cervical region. Or the softening may be more limited to certain subdivisions of the cord in one or other of these regions—and then constitute an 'incomplete transverse softening.' Thus it may, in one set of cases, principally affect the anterior columns and grey matter; in another set the posterior columns and more or less of the grey matter. Or the softening may be central, and almost confined to the grey matter through a considerable extent of the cord, as in 'diffuse central softening'; when this change involves the white columns as well as the grey matter for a considerable extent, we have what is called 'diffuse softening' of the cord. When a small focus of softening exists which only involves part of the transverse area of the cord, and that for a very limited extent, we have what is commonly spoken of as a 'circumscribed softening' of the cord; and when many of these small foci are scattered through different parts and regions of the organ, we have what is known as 'disseminated softening.'

An accidental damage during the opening of the

spinal canal must not be confounded with the results of pathological change. In a spinal cord bruised in the manner indicated the nerve-substance may be softened and diffident, and somewhat resemble a patch of real pathological softening. Examination with the microscope, however, would show, among the fragments of myelin from the broken nerve-tubules in the former case, an entire absence of the large granulation-corpuscles, which are, on the contrary, invariably present in a patch of real pathological softening. If there were, after such an examination, still room for doubt, this might be resolved by the fact that the softened nerve-matter in a patch of real softening of the cord has its specific gravity lower by 3-5 degrees than that of other healthy portions of the organ, while in the patch of merely bruised nerve-substance it would not be appreciably lower than normal. The normal specific gravity of the spinal cord varies commonly from 1033-1041 in different individuals—the higher figures being most frequently met with in elderly persons. The modes of estimating the specific gravity have been discussed by the writer in *Journ. of Ment. Science*, vol. xi. 1866.

Where the process of softening has gone on to its final stages—in a case, for instance, of 'complete transverse softening'—the whole substance of the cord in the affected site is reduced to a rather dirty-looking milky fluid, which, when the membranes are cut across, flows out so as to leave a complete gap in the cord-substance for an extent, it may be, of one to three inches. All intermediate stages may be found between such a condition and a slight degree of softening, in which the cord-substance is only a little more pulpy than natural.

SYMPTOMS, COURSE, AND TERMINATIONS.—The symptomatology of this disease presents an extremely wide range, in accordance with the varying extent and sites of the softening in the cord, as existing in different patients.

In 'circumscribed' and 'disseminated' softening, for instance, the symptomatology would be excessively variable in different patients; and, especially in the latter class of cases, it might be extremely difficult to arrive at a diagnosis. The symptoms could, in fact, only be interpreted by the light of the general principles applicable to the regional and pathological diagnosis.

Again, in cases of 'diffuse central softening' the symptoms—except for the fact that they set in gradually rather than abruptly—would bear a close resemblance to those of hæmorrhage into the spinal cord, where the blood is effused into the central grey matter for a certain extent (see 8. SPINAL CORD, Hæmorrhage into). There is some doubt, indeed, whether these latter cases may not occur principally as epiphenomena sequential to a primary central softening.

The symptomatology of 'incomplete transverse softenings' of the cord is for the most part exemplified by the second stages of various forms of so-called 'compression-myelitis'—cases, that is, in which the anterior regions of the cord more especially are, in one set of cases, principally pressed upon either by tumour, or by the inflammatory products associated with vertebral caries ('Pott's disease'); while in another set the posterior columns and posterior grey matter may undergo a similar softening, under the influence of the pressure of a new-growth impinging upon the cord from behind. Cases of this type, however, may easily and do often

merge into 'complete transverse softening' (commonly known as 'complete transverse myelitis'). Both complete and incomplete forms also often occur in the cord quite independently of pressure.

Of these states it will be well, for the sake of brevity, to confine our attention principally to '*complete transverse softening*.'

In a case of *complete transverse softening* involving the mid-dorsal region, the temperature in the axilla usually varies between 98° and 100° F., though with an extension of the pathological process, or towards the close of the disease, it may rise to 101°, 102°, or even higher. Meanwhile the lower extremities themselves are often distinctly cold to the hand—the temperature being in some cases more or less subnormal. It is important to note this, because it might have been supposed that hyperæmia and a slightly elevated temperature would exist, owing to the vaso-motor nerves of the limbs being paralysed.

The motor paralysis of the lower extremities is absolute, and the abdominal muscles are also powerless. The feet, as the patient lies in bed, are extended and often inverted, so that the great toes cross one another. The skin after a time tends to become dry and scurfy. The muscles feel flabby to the hand, but they waste only to a slight extent, and continue week after week to show only a small amount, if any, of diminution in the degree of their irritability to faradic and to galvanic currents.

The sensibility of the limbs is completely abolished both for tactile and painful impressions, as well as for differences of temperature and for tickling. A like abolition of sensibility exists over the trunk up to the level of the 'ensiform area,' while above this level the sensibility is found to be quite natural. Though the upper limit of anaesthesia may be quite sharply defined, yet in these cases of complete transverse softening there is often no distinct 'girdle-sensation.'

The muscles of the lower extremities may show some slight irritability when they are forcibly tapped, and when the soles of the feet are strongly tickled there may be very slight movements of the toes; but beyond this there is often an entire absence of all reflex movements—there is no ankle-clonus, no knee-reflex, and a similar absence of the cremasteric and abdominal reflexes.¹ In the initial stages of the affection, however, and especially when the softening is not completely transverse, all these reflexes may be extremely well-marked for a time, though they tend gradually to diminish.

For the first ten days or a fortnight there is often complete retention of urine, but after this time, when the lumbar region of the cord again becomes capable of manifesting to some extent its centric functions, the initial retention gives place to incontinence of urine. This fluid may be discharged at intervals of two to three hours in small quantities, owing to the

occurrence of reflex contractions of the bladder whenever the organ attains a certain degree of fullness. The passage of a catheter, however, in these cases will often show that the bladder is never completely emptied—two to four ounces remaining after the reflex contractions. Unless special precautions are taken, the urine, in such patients, speedily becomes ammoniacal, and more or less loaded with mucus.

The bowels are usually constipated, and relieved only after the administration of aperients or enemata. At these times there is generally incontinence of feces—the patient having no power of controlling the reflex actions concerned in defecation when they have once been strongly excited. The actual passage of the motion is, moreover, unfelt.

Under the irritative influences emanating from the seat of softening during the period of its establishment, a small bed-sore may begin to form, often amenable to treatment. Later on, sloughs are prone to appear upon the heels, over the malleoli, and in other situations habitually exposed to continuous pressure. But the most frequent site for intractable sloughing bed-sores is over the sacrum. Inflammation of the mucous membrane of the bladder is at last set up; and the inflammation may extend up one or both ureters, so as to implicate the pelvis of the kidney, when minute abscesses may also form in the kidney itself.

Under the influence of these various conditions the patient's appetite and strength gradually fail; emaciation proceeds; and death after a time may come from sheer exhaustion, aided, perhaps, by some intercurrent inflammatory affection of the lungs. Such complications may, however, be occasionally warded off for eighteen months or more. Other modes of death are pointed out in the section on Prognosis.

DIAGNOSIS.—The recognition of this disease at the bedside often presents considerable difficulties. We must be guided partly (*a*) by the patient's history and state; partly (*b*) by the mode of onset of the disease; and partly (*c*) by the symptoms of the fully established affection.

(*a*) The points in regard to previous history which are of principal significance are referred to under the head of *Ætiology*. In regard to (*b*), the mode of onset, this is usually not abrupt and sudden; there is rather a slow increase of paralysis during a week, ten days, or a fortnight. Still, it is a fact that softening of the cord (apparently due to thrombosis) does occasionally cause a sudden incomplete paralysis, though such paralysis increases subsequently in the manner above stated. Such a case must not therefore be confounded with hemorrhage into the cord, merely by reason of its absolutely abrupt onset.

The extent to which the diagnosis turns upon (*c*), the nature of the symptoms of the fully established affection, cannot be very strictly defined except in some cases. When the softening is slight and partial, it gives rise to no distinctive symptoms; but where there are clinical signs of the existence of a complete transverse lesion, the chances are that the lesion itself is, if not a primary, at all events a secondary softening.

In regard to the *regional diagnosis* of softening of the spinal cord, the following points require to be borne in mind:—

The indications as to the transverse area involved, and as to the upper limits of the change in the spinal cord, are wholly derivable from the presence or absence of the various signs and symptoms

¹ In one case in which paraplegia had existed for over three months, in consequence of a complete transverse softening in the upper dorsal region (with the above-mentioned clinical signs), the writer was much struck with the extremely pallid appearance of the grey matter through the whole length of the cord below the seat of softening. The absence of the reflexes may be in part due to such condition of the grey matter, and this itself may be caused by a spasm of its vessels in some way induced by the lesion above. Some amount of spasm may also exist in the vessels of the limbs, whose temperature is often rather sub-normal. See a paper by the writer on 'The Symptomatology of Total Transverse Softening of the Spinal Cord,' in *Med.-Chir. Trans.* vol. lxxiii. 1890.

which have been set forth in the article, SPINAL CORD, Diseases of, pp. 1526 and 1530.

The attempt to ascertain the lower level of the lesion, and consequently its longitudinal extent in the cord, is always difficult, and often cannot be achieved with any success. The indications are all obscure, uncertain, and apt to fail. This is especially the case if we attempt to base an opinion on the fact of the existence or absence of superficial reflexes (*see* § 4, (a)). Thus, complete transverse softening may exist in the upper dorsal region, and extensive secondary degenerations may have been produced, yet for week after week there may be a complete absence of all the reflexes, superficial and deep, dependent upon the cord below the upper dorsal region. This the writer has ascertained by repeated clinical examinations of cases whose nature has been subsequently verified *post mortem*.

PROGNOSIS.—The prognosis in a case of paraplegia must always involve a twofold problem: (1) as to the duration of paralysis, or the probability of recovery; (2) as to the danger to life.

(1) The chance of ultimate recovery from paralysis would vary inversely with the size or extent of the lesion existing after the first ten days or a fortnight—that is, by the time softening has been unmistakably established, and when the chance of such an event being warded off by the establishment of a collateral circulation no longer exists. But where a pretty complete reinstatement of blood-supply does take place, all symptoms of paralysis may gradually disappear in the course of some weeks, or, it may be, months.

(2) Danger to life is brought about in many ways, and a fatal result may be entailed (a) by a gradual extension upwards of the process of softening (especially where it exists in the lower cervical or upper dorsal region), so as to involve paralysis of the diaphragm, or an extreme interference with the heart's action. (b) Inflammation of the bladder, followed by implication of other portions of the urinary tract, may lead on to death after the paralysis has lasted for some months, or perhaps for a year or two. (c) About the same period (sometimes early and sometimes late) extensive bed-sores may form, and the patient may, after a time, die exhausted, or from blood-poisoning. (d) Again, the supervention of an intercurrent pneumonia may lead on to a fatal result; or (e) the end may come from the extension inwards of the process of sloughing, so as to lead to the establishment of a rapidly fatal spinal meningitis. Still in some cases, as above indicated, the patient may remain paralysed for even two years or more before a fatal termination is brought about.

TREATMENT.—Our power to deal with the softened condition itself of the spinal cord is extremely small, whether it may have been caused by thrombosis or by compression. During the early stages probably the less that is done in the way of active interference the better. The principal indications are that the patient should have absolute rest in bed, and for the first few days at least a rather sparing diet; spoon-diet being desirable where distinct elevation of temperature exists. The secretions should be regulated, and the urine, if necessary, drawn off by a thoroughly clean catheter smeared with carbolised oil, and kept in the intervals in a perchloride-of-mercury solution. Sedatives, such as bromide of potassium, either alone or in combination with chloral hydrate, may be needed

at night, for a time, so as to ensure an adequate amount of sleep.

Should the patient's general health be weak or deranged, as is so often the case, every effort must be made to improve it by means of an easily assimilable but generous diet, gradually increased, and by the exhibition of suitable tonics, with or without small doses of cod-liver oil. It is far better to trust to such general means than to the supposed influence of phosphorus, or any other drug. To expect any of them to have a direct influence in restoring softened nerve-tissue is vain; and any good that may be achieved by drugs alone is probably brought about either by their power of regulating some of the principal functions of the body, or by improving its nutritive processes generally.

Still scarcely any morbid condition exists in which more constant care and vigilance are needed than in the *paraplegic state*, in order to correct or ward off its numerous incidental troubles or complications.

One of the first points claiming attention in the early stages of a case of paraplegia is to take such measures as will stave off the occurrence of bed-sores as long as possible. These precautions are especially needful where the paraplegia is complete, and where loss of sensibility exists. The patient should at an early stage of the disease be placed upon a water-bed; and those forms are most suitable in which there is a canal through the centre for the passage downwards of the evacuations. The patient must be kept scrupulously clean and dry; and no folds of the bedclothes must be permitted to press against the skin. If possible, the patient should not be allowed to lie continually upon his back, but occasionally in a prone or lateral position. The skin over the sacrum especially must be carefully watched, and on the least sign of a patch of undue redness there, it should be rubbed once or twice a day with a mixture of equal parts of olive oil and spirits of wine. If it becomes actually abraded, it should be dressed with zinc ointment, smeared over a piece of soft lint.

For the first fortnight or more there may be complete retention of urine, which then requires to be drawn off night and morning by catheter. During this period the greatest care should be taken in regard to the cleanliness of the catheter employed. Carelessness in this respect will tend to bring on cystitis at an early date, with alkalinity of urine, and may thus quite prematurely aggravate the bladder-troubles. As soon as the bladder begins to empty itself again, in a reflex manner, at intervals throughout the day, the use of the catheter may be discontinued as long as the water which comes away continues to be clear and acid. During this period of incontinence it will be necessary to draw off the urine from time to time for the purposes of examination. As before stated, the bladder never completely empties itself. After this state of things has continued for some months, the urine is apt to become alkaline, ammoniacal, and more or less mixed with mucus. At this stage the bladder should again be emptied once or twice daily, and washed out each time with 6 to 8 oz. of quinine-solution (2 grains to the ounce, with enough of diluted sulphuric acid to dissolve it); or with a 2-per-cent. boric-acid solution. This will prove the best means of warding off or of mitigating inflammation of the bladder; and thus perhaps

of preventing its extension to the ureters and kidneys.

In regard to the bowels, purgatives will probably be required from the first, as without their use there will be no evacuation. Sometimes a simple enema will suffice. Scybala tend to accumulate in the large intestine, unless its contractility is aroused occasionally by a large injection, consisting of three pints of warm thin gruel, together with half an ounce of spirit of turpentine and an ounce of castor oil.

Where the disease has reached the chronic stage, and when death is not inevitable, the muscles should be faradised or galvanised three times a week, while massage may be employed on alternate days, with a view to maintaining their nutrition as far as possible.

When in the final stages of paraplegia large and sloughing bed-sores have formed, they will require the most constant care and attention. Poultices may be at first needed till the sloughs have separated, and afterwards the wounds must be variously dressed according to their condition. An ointment composed of ten grains of carbolic acid to one ounce of vaseline may be employed; or more stimulating applications may be needed. Sometimes iodoform or the iodide-of-starch paste forms a suitable dressing.

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10. Infantile Paralysis.—See PARALYSIS, INFANTILE.

11. Acute Spinal Paralysis of Adults.—SYNON.: *Polio-myelitis Anterior Acuta*; Acute Inflammation of the Grey Anterior Horns; Acute Atrophic Spinal Paralysis; Fr. *Paralyse Spinale Atrophique Aiguë*; Ger. *Polio-myelitis Anterior Acuta*; *Acute Spinallähmung bei Erwachsenen*.

This is essentially the same disease as that known as infantile paralysis (see PARALYSIS, INFANTILE), though presenting certain differences from the fact of its occurring in adults. The disease is more difficult to recognise in adults, because in them other affections occur with which it is quite possible that it may be confounded.

ÆTIOLOGY.—The ætiology of this affection in adults is just as obscure as it is in children. Sometimes it manifests itself without any assignable cause; while at other times there is the possibility that exposure to wet and cold, some shock or blow, or some antecedent acute febrile illness may have had to do with its origin. The causative conditions are, in fact, in part similar to those of multiple neuritis (see NEURITIS, MULTIPLE), and the two affections in different proportions sometimes co-exist. There exists, perhaps, in some persons a special vulnerability or instability of the motor ganglion-cells of the grey matter of the spinal cord. To this view Bernheim inclines, in consequence of his having collected nineteen cases in which persons who have suffered from infantile paralysis have in adult age become affected either with acute, sub-acute, or chronic poliomyelitis—sometimes involving the same and sometimes different regions of the spinal cord.

SYMPTOMS.—It will principally be necessary in this case to point out the manner in which the group of signs and symptoms characteristic of the disease in infancy becomes modified when it occurs in the adult.

The first set of differences is due to the minor

irritability of the nervous system in the adult, as compared with that of the young child. The initial febrile symptoms may be so slight as to escape notice; convulsions have never been met with; and preliminary head-symptoms are generally absent or very slight. Still some headache, or mental dullness, may be present; and vomiting occurs not infrequently.

Paralysis then sets in speedily—it may be within a few hours—and is more or less widespread. The muscles are flaccid; reflex actions are abolished or greatly diminished. In the course of a few days, generally, improvement as regards motor power sets in, and very slowly progresses. It may go on continuously to complete recovery in the course of a few months; or, as often happens, such recovery is only partial. In the latter case certain muscles or groups of muscles remain paralysed, and in them a rapid atrophy occurs. When tested electrically, these muscles exhibit the 'reaction of degeneration.' The affected parts are cold, and sometimes more or less cyanotic. There is no impairment of sensibility; and no interference with the functions of the bladder or rectum.

All the characters mentioned in the last paragraph accord with those which present themselves in infantile paralysis, but later on differences again show themselves. One of the characteristic features in the child is arrest of growth in the parts affected, so that the limbs or parts of limbs paralysed remain more or less abortive. This, of course, cannot occur in the adult; and also, owing to the fact that the joints are stronger, the secondary deformities (often so serious in the child) are not met with to the same extent in adults.

DIAGNOSIS.—The mode of onset of the disease; the fact that the paralysis is purely motor, associated with no pain or tenderness, and accompanied by no interference with sensibility; the fact that after the first few days at least the functions of the bladder and rectum are not interfered with; and also that in later stages there is atrophy of muscles, and the existence of the electrical 'reaction of degeneration'—these constitute a group of conditions which, taken as a whole, is thoroughly distinctive.

The disease with which it is most liable to be confounded is that hereafter to be described (see 13. Spinal Paralysis, Chronic Atrophic). The points of distinction will, therefore, be given under it. 'Progressive muscular atrophy,' if we bear in mind its very chronic onset, is much less liable to be confounded with the present disease, as also if we recollect that in it atrophy makes its appearance before paralysis rather than after, and that the electrical reactions are, except in the later stages of the disease, notably different.

The fact of the absence of spasms, the diminution of reflexes, the non-interference with sensibility and with the sphincters, together with the abrupt origin of the disease, suffice to separate the acute spinal paralysis of adults from all other affections of the spinal cord, though not from all cases of multiple neuritis. As to this latter difficulty, the reader may refer to the section on Diagnosis in the article on NEURITIS, MULTIPLE.

PROGNOSIS.—This is not a disease dangerous to life. Complete recovery not infrequently takes place, and that too, as the writer has seen, where the paralysis may have been widespread, affecting all the limbs for a time, and leading to marked atrophy in the muscles of the lower extremities. In

other cases there is left in particular parts a chronic remainder of paralysis with atrophy, just as we find to be the case in children. In so far as any part of the patient's symptoms are due to some co-existing peripheral neuritis, we are generally entitled to make a more hopeful forecast.

TREATMENT.—This disease must be dealt with on precisely the same principles as those which are applicable to the corresponding affection in young children. Repetition is, therefore, here unnecessary. See PARALYSIS, INFANTILE.

H. CHARLTON BASTIAN.

12. Acute Ascending Paralysis.—SYNON. : *Paralysis Ascendens Acuta*; Landry's Paralysis; *Fr. Paralyse Ascendante Aiguë*; *Ger. Paralysis Ascendens Acuta*.

DEFINITION.—A mysterious affection of the spinal cord, first definitely described by Landry in 1859; characterised on its clinical side by the existence of a progressive paralysis, advancing rapidly from below upwards, so as finally to implicate parts dependent for their innervation upon the medulla oblongata; characterised also on its anatomical side by the absence of any appreciable pathological change.

On account of the latter peculiarity, the disease ought not to be described in the present place, but rather to constitute the first of Class II (see p. 1531). But this disease, together with 'acute spinal paralysis' and 'subacute spinal paralysis,' have such an amount of similarity from a clinical point of view, that it seems very desirable for their descriptions to follow one another, so that mutual alliances as well as differences may be the more distinctly appreciated.

ÆTIOLOGY AND PATHOLOGY.—The causes and pathogenesis of this affection are even more obscure than those of the disease last referred to. Exposure to cold, and emotional disturbances (with or without suppression of menstruation in the female), have been observed occasionally as precursors. Occasionally, too, this disease has supervened during convalescence from some previous acute febrile malady, such as small-pox or typhoid fever. Syphilis is thought by a few (but on no sufficient evidence) to have something to do with the pathogenesis of this affection.

Westphal, again, who pointed out that the spleen became enlarged in this affection, inclined to believe in the existence of some toxic influence—a view in support of which there is distinctly more in the way of evidence.

The disease seems principally to occur in persons between the ages of twenty and forty, and to be decidedly more frequent in males than in females. Although the brain and spinal cord of those who have died from this affection have now been frequently examined by skilled observers, the results have hitherto been almost entirely negative, so far as morbid anatomy is concerned. No definite changes have, moreover, been met with in the peripheral nerves with any constancy, though the predisposing influences and the symptoms are such as to have created a strong suspicion that the affection might be due to a multiple neuritis. Further evidence is, however, needed before definite conclusions can be arrived at as to the exact nature of this very puzzling malady.

SYMPTOMS, COURSE, AND TERMINATIONS.—About the prodromata there is nothing distinctive—

they may be absent. When present there may, for a few days, or even for a few weeks, be a slight febrile condition from time to time, with a sense of weariness, and more or less numbness in the limbs, especially in the tips of the fingers and in the feet.

The disease then more definitely declares itself, and follows a very variable course as regards rapidity of evolution; sometimes causing a generalised paralysis which may prove fatal on the second or third day, and at other times lasting as many weeks before death occurs or recovery sets in. The disease commences by a marked weakness of the lower extremities; soon to be followed by actual paralysis, which, as in the 'subacute and chronic spinal paralysis,' shows itself first in the distal portions of the limbs, and gradually approaches the trunk, so that in the course of two or three days, or even much less time, the paralysis of the lower extremities becomes complete.

The trunk-muscles are next, and soon, implicated in a similar manner. The patient can no longer sit up or turn in bed. Respiration becomes more and more affected, and defecation is interfered with, through weakening of the abdominal muscles.

Next, though sometimes after a distinct interval, the upper extremities become implicated; though here again the paralysis first involves the distal portions of the extremities, and thence gradually spreads (after a period in which mere paresis exists), till the whole limbs become completely powerless.

The paralysed limbs, both upper and lower, are lax, and show no trace of contraction. Though the muscles are flaccid, they do not undergo a marked amount of atrophy, as is the case in 'acute spinal paralysis.'

In accordance with this latter peculiarity, there is the further striking characteristic that the *electrical reactions of nerves and muscles continue perfectly normal*. This seems now to be a well-attested fact, and it has been verified by good observers even after complete paralysis (without atrophy) has existed, in the more chronic cases, for several weeks.

Sensibility is scarcely, if at all, affected; nor, as a rule, are pains complained of in the paralysed parts.

The reflexes, both superficial and deep, are generally abolished, though in the more chronic cases they may reappear after a time.

The nutrition of the skin is not impaired, so that there is no tendency to the formation of bed-sores. Coldness and cyanosis of the limbs do not seem to be characteristics of this affection.

The sphincters are usually not at all affected. Constipation is often marked, and defecation may be rendered difficult owing to paralysis of the abdominal muscles.

As a rule, there is no febrile elevation of temperature, though to a slight extent this has been present in some cases.

At the stage above indicated, in nearly one-third of the recorded cases, or it may be even before the arms have become much implicated, the disease becomes arrested, and after a brief interval recovery of power begins to manifest itself—usually in a reverse order, so that power is regained first over the arms, then over the trunk, and subsequently (and perhaps only in the course of several weeks) over the lower extremities.

But in the remaining two-thirds of the cases,

after the arms have become paralysed, the disease still progresses so as to affect the cervical muscles, the diaphragm, and finally the muscles innervated by the motor nerves of the medulla. Thus, in its later phases the disease is characterised by a greatly increasing difficulty in respiration; great weakness of voice; extreme rapidity of pulse; and possibly by inequality of the pupils. Finally, increasing paralysis of the muscles concerned with articulation and deglutition sets in; and, owing to the augmenting difficulties of respiration, death may arrive at any moment by asphyxia. This climax of the disease may be reached in the course even of three or four days; on the other hand, it may not be reached until as many weeks have elapsed. Whenever the disease has advanced so far as seriously to implicate the medulla, recoveries are comparatively rare.

In quite exceptional cases the disease may pursue a reverse order throughout; implicating the nerves of the medulla first, then those of the cervical region of the cord, and so on. The celebrated Cuvier is said to have died from the disease progressing in this very unusual manner.

DIAGNOSIS.—So far as the established disease is concerned, we have in this affection, in 'acute spinal paralysis of adults,' and in subacute forms of 'chronic spinal paralysis,' maladies that present certain well-marked points of similarity. In each we have to do, in typical cases, with simple motor paralysis, with no fever, no tenderness or pains in the spine, no pains in the limbs, or contractions, and with no incontinence of urine or feces, or tendency to the occurrence of bed-sores.

'Acute ascending paralysis' differs from both these affections, however, in the important fact that rapid atrophy does not set in in the paralysed muscles, and that the electrical reactions in no way differ from those met with in healthy nerves and muscles. In the very acute cases, of a few days' duration only, these distinctions would be worthless, as sufficient time would not have elapsed to make it possible to attach any value to such differential indications. In such rapid cases, therefore, the distinctly *progressive* character of the disease is that which will serve to distinguish it from the more severe cases of 'acute spinal paralysis,' in which the paralysis sets in simultaneously throughout the whole of the parts affected (the area of the paralysis subsequently tending to diminish), and often with a pretty distinct initial, though brief, febrile disturbance. Then, again, there is the fact (for consideration further on) that this latter disease has little or no tendency to involve the medulla, and is only very rarely fatal.

It is in the diagnosis of the more slowly evolved forms of 'acute ascending paralysis,' from the similarly progressive cases of 'chronic spinal paralysis,' that the development of rapid atrophy of the muscles, together with the 'reaction of degeneration,' comes to be distinctive of the latter affection. Again, in 'acute ascending paralysis' there is a longer persistence of reflex actions; and a far greater tendency to the manifestation of symptoms showing that the medulla oblongata is involved.

PROGNOSIS.—Nothing can be added concerning prognosis beyond what has been above indicated in speaking of the course and terminations of the disease. It seems the rule that, the more rapid the progress of the disease, and the earlier the medulla is affected, the more is a fatal termination to be

feared. Still, even in the most acute cases, improvement *may* take place.

TREATMENT.—The absence of any known pathological substratum for this disease makes it extremely difficult to lay down any directions for treatment. It would appear that we have to do with a simple alteration of the molecular condition of some parts of the spinal motor nerve-centres, unaccompanied by any known inflammation or irregularity of vascular supply.

Under these circumstances, the patient should be put upon a nutritious but easily assimilable diet, with a fair amount of stimulants; and, further, we may endeavour to induce a change in the nutritive and functional activity of the spinal cord, by having recourse to frictions of the skin or gentle shampooing of the limbs, together with brief daily applications of weak faradic currents to many of the affected muscles.

From drugs, perhaps the best chance of beneficial results may be looked for from combinations of iron and arsenic, or from the cautious use of small doses of strychnine. Iodide of potassium would probably be useless.

H. CHARLTON BASTIAN.

13. Spinal Paralysis, Chronic Atrophic.
SYNON.: Subacute and Chronic Inflammation of the Grey Anterior Horns; *Poliomyelitis Anterior Subacuta et Chronica*; Fr. *Paralysie Générale Spinale Antérieure Subaiguë*; Ger. *Subacute Spinal-lähmung Erwachsener*; *Subacute Spinal-paralyse*.

NATURE, ÆTIOLOGY, AND PATHOLOGY.—This disease was described by Duchenne in 1853, and then again more completely in 1872, as a more or less rapidly advancing motor paralysis, associated with atrophy of the muscles affected, and loss of their faradic excitability.

He believed the disease to be dependent upon a chronic degeneration occurring in the grey anterior horns, and this view is supported by the examinations which have as yet been made of persons who have been the subjects of this affection. The pathological changes in the anterior horns have been associated with atrophy of the anterior nerve-roots.

The causes of the malady are at present almost wholly unknown; but it occurs principally in individuals between the ages of thirty and fifty years. As with other chronic spinal affections, so here, there has often been one or other of the following events occurring some little time before the onset of the disease: Exposure to cold and damp, some shock or concussion, venereal excesses, or great fatigue induced by other causes. But what share the pre-existence of one or other of these conditions may have had in initiating the disease cannot at present be defined.

SYMPTOMS.—In the subacute cases, paralysis may become developed (usually in the lower extremities first) in the course of a few days or weeks; at the same time there may be some very slight initial febrile disturbance, and possibly some shooting pains in the back and limbs.

In the more chronic cases, the latter symptoms may be absent, and the onset of paralysis is very much slower. There may be at first mere paresis, felt most in the ankles and knees; but gradually (often after many months) this deepens into distinct paralysis of certain groups of muscles, or of the entire limbs. The muscles are flabby and progressively waste; at the same time they cease to respond well or even at all to the faradic current, and

become more sensitive to the voltaic. There may also be notable fibrillar twitchings in the muscles undergoing this atrophic process.

Sensibility is unaffected. Skin- and tendon-reflexes are abolished. The temperature of the affected limbs is lowered; and the feet especially are apt to be more or less cold and cyanotic.

Soon the arms become affected in a similar manner, and here the paralysis may first affect either the extensors or the flexors. It may remain more or less limited to certain groups of muscles, or may gradually extend so as to implicate the whole limb. The distal parts are usually, however, more completely involved than the proximal. In the arms the same kind of phenomena occur as in the lower extremities, and there is a similar absence of rigidities or contractures.

There is no tendency to the formation of bed-sores, and the nutrition of the skin seems to be unimpaired.

The rectum, the bladder, and the sexual organs are usually quite unaffected.

After a time, the excessive reaction of the wasted muscles to the galvanic current decidedly diminishes; though in the earlier stages of this affection the electrical 'reaction of degeneration' often exists with all its characteristic details.

DIAGNOSIS.—This malady bears a closer resemblance to the 'acute spinal paralysis' of adults than to any other affection. The two diseases are naturally distinct in their modes of initiation, but as established diseases (that is, in their later phases) they would be very difficult to discriminate from one another in the absence of definite information as to modes of onset—and such information, of a reliable kind, is often not to be obtained. It is the abrupt commencement of the paralysis over a wide area of the body that is met with in, and which is so distinctive of, 'acute spinal paralysis'; while in the subacute forms, and more especially in 'chronic atrophic spinal paralysis,' we have to do with a distinctly progressive spread of the disease from part to part.

In regard to the discrimination of the subacute and chronic forms of spinal paralysis from some other varieties of spinal-cord disease, the reader may refer to what has been said concerning the grounds on which the diagnosis of 'acute spinal paralysis' is to be made. See *Acute Spinal Paralysis of Adults* (p. 1547).

In 'amyotrophic lateral sclerosis' the upper extremities may be paralysed, wasted, and flaccid, as they are in 'chronic spinal paralysis'; but then in the former disease there may be the different combination of paralysis without much wasting, but with more or less rigidity, in the lower extremities.

For the distinguishing characters of 'acute ascending paralysis,' see the account of that affection, in the preceding article (p. 1548).

PROGNOSIS, COURSE, AND TERMINATIONS.—In the subacute cases, after a month or two, improvement may gradually begin to manifest itself; and in exceptional instances this may go on slowly, but steadily, to complete recovery. In other of these cases, however, certain muscles or groups of muscles do not undergo the same improvement as the others; they may continue paralysed, and become more and more atrophied.

In the more chronic cases, recovery is scarcely to be looked for; though, after the symptoms have

developed to a certain extent, it occasionally happens that no further advance is made.* Such patients may remain in much the same condition for years.

In another class of cases the malady proves more continuously progressive, so that, after implicating the upper and lower extremities severely, the morbid process may extend to the upper cervical region of the cord, so as greatly to interfere with respiration; or it may even extend to the medulla, so as to involve the tongue and pharyngeal muscles, and more or less interfere with the functions of articulation and deglutition. In such cases death is liable to occur through asphyxia or slowly progressing exhaustion.

In the majority of cases of this disease, more or less complete recovery occurs, though it may be only after two to four years.

TREATMENT.—Possibly counter-irritation to the spine in the early stages by means of the strong solution of iodine, painted over patches every four or five days, may do good, and should certainly be tried, where the patient comes under treatment at this period. A nutritious and easily digestible diet, tonics (such as a combination of iron, strychnine, and small doses of arsenic), and rest are essential in the early stages, together with a thorough supervision of the general health. Later on, massage and electrical treatment by the voltaic current must be had recourse to, and must be perseveringly continued for long periods, until the muscles again begin to respond to the faradic current. The electrical treatment with massage is what is principally to be relied upon; and except in the subacute cases it may be commenced almost from the first, should the patient happen to come under observation during the early stage of the malady. Sulphur- or brine-baths, two or three times a week, seem also at times to do much good.

H. CHARLTON BASTIAN.

14. Progressive Muscular Atrophy.—See *PROGRESSIVE MUSCULAR ATROPHY*.

15. Locomotor Ataxy.—See *TABES DORSALIS*.

16. Spinal Paralysis, Spastic.—**SYNON.** : *Paralysis Spinalis Spastica*; Primary Sclerosis of the Lateral Columns; Idiopathic or Primary Lateral Sclerosis; Primary Spastic Paraplegia; *Fr. Tabes Dorsal Spasmodique* (Charcot); *Ger. Spastische Spinalparalyse; Primäre Sklerose der Seitenstränge des Rückenmarks; Primäre Lateralsklerose des Rückenmarks.*

ÆTIOLOGY.—This disease is distinctly more common in males than in females. It occurs in the majority of cases in adults from twenty to fifty years of age. Erb and others have also described spasmodic forms of paralysis occurring in children, as a result of double cerebral disease, which have several clinical features in common with this affection ('Little's disease,' see p. 269). The writer met with the ordinary form of the affection once in a child of about ten years of age, but then the lateral sclerosis seemed only to form a prominent part of what was really a 'multiple sclerosis' of cerebro-spinal type.

In some cases the disease appears independently of any appreciable predisposing or exciting causes; but, in other instances, falls or other traumatic influences, or great fatigue after some very prolonged walk, seem to be connected with its origin. On rare occasions exposure to wet and cold has ap-

peared to have had some influence over the genesis of this, as well as over that of so many other forms of spinal disease.

ANATOMICAL CHARACTERS.—In the first undoubted case which was investigated *post mortem*, the following pathological conditions were observed: 'The cord, when examined in the fresh state, showed to the naked eye no abnormality, except softening in the lowest dorsal region. After hardening in bichromate of ammonium, sections of the cord showed already to the naked eye one light-coloured patch in each lateral column'—and this throughout the cervical, the dorsal, and the lumbar regions of the cord. This band of morbid tissue, presenting all the typical characters of a sclerosis, occupied the greater portion of the lateral columns, but without implicating the grey matter or extending quite to the surface of the cord. The anterior and the posterior columns were perfectly healthy. The microscopical characters of primary sclerosis in the spinal cord are briefly described in the article on 'multiple sclerosis.' See Multiple Sclerosis, p. 1553.

The occurrence of the slight softening in this case was an accidental complication, otherwise the lesions actually found agreed very perfectly with Charcot's scientific predictions as to the probable pathological changes peculiar to this affection of the spinal cord. Similar changes have subsequently been encountered in other cases.

SYMPTOMS.—This disease often sets in almost imperceptibly, and the symptoms continue to develop in a very slow and gradual manner.

Patients begin to complain first of mere weakness of the lower extremities, and this continues to increase till a well-marked condition of paresis exists. There is great difficulty in getting upstairs, and the feet begin to drag even when the patient walks on level ground. This paresis may soon be associated with more or less of muscular twitchings, often more marked in the morning, but sometimes more especially at night, and of a painful character. Later, an actual stiffness of the muscles of the legs begins to manifest itself, which becomes apparent principally when passive movements are attempted, or even when the patient seeks himself to move the limbs. At last some amount of rigidity of muscles may be more or less continuously present, so as greatly to interfere with locomotion, or in some cases even to prevent it altogether.

In the early stages of the disease, ankle-clonus can be elicited with the greatest ease, and the knee-jerk is found to be distinctly exaggerated on both sides. When one of these patients is in the sitting posture, commencing pressure on the toes of one foot, as in the act of rising, will often at once initiate the characteristic tremors of ankle-clonus. All such signs, however, will probably diminish as the rigidity becomes more marked. The plantar reflex is of the extensor type.

While the patient is able to walk he often exhibits a typical 'spastic gait.' The legs are generally kept close together, owing to a spasmodic contraction of the abductors of the thighs; the toes are dragged along the ground; and then, when the heel is beginning to be brought down, in some rare cases a spasmodic contraction of the calf-muscles may take place, tending to raise the patient upon his toes and almost throw him forward. In this way a mixed and very irregular kind of walking is necessitated, partly to be accounted for by mere powerlessness, and partly by

the occurrence of strong muscular spasms. In some instances, either owing to variations in the amount of the spasms, or, it may be, to the great weight of the patient, this spastic walk is not well-marked. In all cases, however, it is quite different from the ataxic gait; and when standing with feet close together, no increase of unsteadiness or feeling of vertigo is occasioned on the patient closing his eyes.

Sensibility is little, if at all, affected; still, in some instances, it is apt to be slightly impaired. In one case, not long since under the writer's care, ability to recognise differences of temperature was for a time greatly lessened; and although tactile sensibility was scarcely at all interfered with, the patient had frequently complained of a diminished power of appreciating the exact positions of his legs. Skin-reflexes, other than the plantar, are often normal, but occasionally they may be slightly increased.

The muscles do not very appreciably atrophy, and their electrical reactions continue to be almost normal; while, according to Erb, that of the nerves is slightly but distinctly lowered to both currents. Sexual desires are not affected, but sexual disability may be occasioned to a variable extent—partly owing to weakness or actual paralysis, and partly to mere spasms of muscles. Micturition is often scarcely at all interfered with; there is nothing like incontinence of urine or of feces, though there may be an obstinate amount of constipation.

No vaso-motor or trophic disturbances in the limbs are usually present.

As the disease progresses (it may be very slowly, and in the course of years) the muscles of the trunk become affected, so that weakness and spasms, often of a very painful character, occur in the abdominal and dorsal muscles. After a time the arms also may become implicated, and in the same fashion as the legs, excepting that when permanent contractions of the muscles come on, they mostly fix the arm to the side, while the forearm is pronated and half-flexed, and the fingers and wrist are strongly flexed.

In rare cases the disease is limited to one side of the body, beginning, for instance, first in one leg, and then extending to the arm on the same side, so as to present a kind of hemiplegic distribution. Just as rarely, too, the disease may first affect the two upper extremities, and then extend down the trunk so as ultimately to involve the lower extremities.

During the development of the disease, shivering-fits, affecting the muscles of the jaws as well as almost all the muscles of the body, may occur from time to time, lasting for half an hour or more; and though quite unaccompanied by any changes of temperature, they may, nevertheless, be provoked by cold. Sometimes, however, such attacks occur spontaneously; or they may spread from some accidentally initiated ankle-clonus, or other well-marked spasm.

Persons suffering from this malady often remain in an almost stationary condition for a series of years, at any particular stage of the disease that may happen to have been attained. Ultimately, however, there is a tendency to complete paralysis of the parts affected, with permanent contractions—the legs at this stage being often immovably fixed in a condition of rigid extension, though they are sometimes flexed at hips and

knees. As a rule, pains are not complained of at any stage of the disease, though some patients suffer much from painful cramp-like contractions, occurring either in the lower extremities, or else in some of the abdominal muscles.

COMPLICATIONS.—So long as the morbid process remains limited to the lateral columns, no other symptoms present themselves. Should it, however, invade the grey matter in particular regions of the cord, then characteristic complications are apt to arise, and it may also be said that the gravity of the disease becomes very distinctly increased. The way for a fatal termination may then be paved through the gradual increase, for instance, of bladder-troubles; or through the occurrence of severe bed-sores, and collateral events to which they may give rise.

Another possible extension of the sclerosis is to the posterior columns, so that we may get a variable mixture of the symptoms pertaining to 'spasmodic spinal paralysis,' and to 'locomotor ataxy.' It should be borne in mind, however, that such a complicated clinical grouping sometimes develops in the reverse order.

Usually in patients suffering from this disease, there is no association with cerebral symptoms, nor is there any tendency to the springing up of cerebral complications. Still, in one case under the writer's care a subacute maniacal condition became developed; while in another case diabetes mellitus to a slight but tractable extent has manifested itself. In both instances, however, there happens to have been a marked hereditary predisposition to the occurrence of insanity and of diabetes mellitus respectively.

DIAGNOSIS.—The grouping of symptoms met with in this disease is so characteristic, that there ought in most cases to be no difficulty in recognising it. In no other organic affection of the spinal cord have we the combination of a gradually progressive paralysis beginning in the lower extremities, associated with muscular twitches and rigidities; greatly exalted tendon-reflexes; plantar reflexes of extensor type; no impairment of sensibility and no pains; no wasting of muscles or other trophic changes; and no interference with the functions of the bladder and rectum.

It happens, however, that certain functional diseases of the spinal cord, occurring principally in young women, may present nearly all these characters, and may for a time be excessively difficult of diagnosis from early stages of lateral sclerosis. In other of these functional cases there is an altogether unusual amount of sensory impairment of one or other kind occurring as additional symptoms, and in these the diagnosis is more easy. *See* Functional Paralysis of Spinal Type, p. 1562.

Another difficulty arises in the recognition of the complex forms of the disease, or of combinations of this disease with others, then coming under observation for the first time. This, for instance, is the case where we have to do with a combination of posterior and lateral sclerosis, in which, in order to arrive at a diagnosis of the existing condition, the observer must be able to recognise the respective effects or modifications that may result from the combination of the two diseases. A further difficulty of the same kind arises when the symptoms of the disease are complicated by extension of the sclerosis to the grey anterior horns. Symptoms will then present themselves more or less resembling

those which are described below under the head of Sclerosis, Amyotrophic Lateral.

Again, when 'multiple sclerosis' affects in the main the lateral columns, the real diagnosis can only be arrived at by the recognition of symptoms which could not be produced by a mere affection of the lateral columns. Thus the writer has recently had under his care a little girl, ten years of age, first brought to him on account of head-symptoms, which suggested the possibility of intracranial tumour, but in whom, after a few months, signs of lateral sclerosis have become developed in a very typical manner. She presented the most characteristic spastic gait, being frequently raised quite upon the points of her toes as she walked. There was also great exaggeration of the tendon-reflexes, and no impairment of sensibility. The case seemed clearly one of 'multiple' or 'cerebro-spinal sclerosis.'

PROGNOSIS.—So long as the disease-process remains limited to the lateral columns, as it does in the great majority of cases, 'spasmodic spinal paralysis' carries with it no danger to life. Such patients may, with careful nursing, survive for an indefinite time, even though for years after permanent contractions have become established they may have been absolutely confined to bed.

TREATMENT.—In the treatment of 'spasmodic spinal paralysis,' as in that of locomotor ataxy, we must use such means as are most likely to be of avail in checking the process of sclerosis in the columns of the cord which is the cause of the symptoms. The general health of the patient, and the regulation of his mode of life, must receive our most careful attention. Sound sleep must also be ensured, as far as possible.

Nitrate of silver has been praised by some; but the writer believes that, on the whole, more good is to be obtained from iodide of potassium in eight- or ten-grain doses, either with or without liquor arsenicalis, given for a time at intervals. Small doses of cod-liver oil, either alone or in combination with maltine, also seem to do good. There is no particular indication for electrical treatment in this disease; but stimulation of the skin and subjacent parts, by frictions and massage, may be of service in the early stages, and so also may hot brine- or sulphur-baths. There are mostly no pains to be allayed; but occasionally painful cramp-like contractions of the muscles cause much distress to patients suffering from this disease. These pains are difficult to relieve, though good may be done, in some cases, by the extract of Calabar bean in increasing doses. For the rest, any accidental accompaniments of the malady must be treated upon the general principles applicable to the management of other spinal affections. H. CHARLTON BASTIAN.

17. Sclerosis, Amyotrophic Lateral. — SYNON.: Fr. *Sclérose Latérale Amyotrophique*.

This is a combination of symptoms first described by Charcot as a separate disease. Clinically, there is an admixture of the signs of progressive muscular atrophy with those of lateral sclerosis, while towards the close there is often the addition of a glosso-laryngeal paralysis. The affection is undoubtedly a rare one, and doubt has been felt by some as to how far this combination of symptoms is entitled to an independent place in our nosology.

PATHOLOGY AND ANATOMICAL CHARACTERS.
The peculiarity of this form of lateral sclerosis is

said to lie principally in the fact that it commences in the cervical region, and soon spreads to the contiguous anterior horns of grey matter; thence, after more or less of an interval, it extends in two directions: (a) downwards, so as to involve the dorsal and lumbar lateral columns, and also the contiguous anterior cornua of grey matter; and (b) upwards, so as to implicate the upper cervical region of the cord and the medulla oblongata in a similar fashion.

Thus it will be seen that three peculiarities are asserted concerning this form of lateral sclerosis: (1) that it begins in the cervical region of the cord, and subsequently affects the dorsal and lumbar portions; (2) that it does not remain limited to the lateral columns, but soon spreads to the contiguous anterior cornua, where it leads to destruction of the great motor ganglion-cells; and (3) that it almost invariably extends upwards also, so as to involve the medulla oblongata, and thus gradually brings about the death of the patient.

SYMPTOMS, COURSE, AND TERMINATIONS.—Being marked by the anatomical characters above described, it will be easily understood that patients suffering from this disease present an admixture of such signs and symptoms as may be met with separately in 'lateral sclerosis,' in 'progressive muscular atrophy,' and in 'bulbar paralysis.' We are said to have, in fact, the following grouping and sequence of symptoms:—

(1) Paresis, gradually increasing to actual paralysis of the upper extremities, and soon associated with distinct muscular atrophy and fibrillary twitchings. Any movements that can be executed are weak, and associated with tremors. More or less marked rigidity of muscles, and finally actual contractures occur, in which the arms are fixed close to the sides of the body; the forearms are semi-flexed and pronated, while the hands and fingers are strongly flexed.

(2) After an interval of some months, a similar group of symptoms becomes developed in the lower extremities. Again, we have paresis gradually increasing, with muscular twitchings, exaggerated tendon-reflexes, and an increasing amount of rigidity of the lower limbs, which are usually fixed in the extended position. At a later period in the lower extremities, as compared with the arms, a process of muscular atrophy sets in, with development of the 'reaction of degeneration,' and fibrillary twitchings in the affected muscles.

During the whole of this time, there is little or no interference with sensibility. There is usually no implication of the sphincters, and no tendency to the formation of bed-sores.

(3) In the last stage of the disease, there is evidence of extension of the morbid process upwards to the upper cervical region and the medulla. Signs of bulbar paralysis present themselves in the usual way, by paralysis with atrophy of the tongue and lips, and by progressive weakening of the muscles of the palate, pharynx, and larynx. The phrenic nerve has also generally become involved, and when weakness of the diaphragm is added to weakness or actual paralysis of the other muscles of respiration, this all-important function becomes more and more impaired, and thus a fatal termination may at any time be easily brought about. Increasing difficulty of articulation and deglutition may have existed for some months before death.

In the writer's experience this order of development is by no means always met with. Several

cases have of late been seen in which the initial changes have been most marked in the lower extremities.

Many neurologists are now of opinion that no independent disease such as Charcot has described under this name of 'amyotrophic lateral sclerosis' exists. It is recognised by many to be only a variety of progressive muscular atrophy, and this view is most in accordance with the writer's own experience. It must be admitted, in fact, that more or less of lateral sclerosis almost invariably co-exists in progressive muscular atrophy together with the more distinctive degenerative changes in the ganglion-cells of the anterior cornua; and in some cases it happens that the clinical signs of the former change are obvious, rather than much dwarfed or non-existent as they are in the large proportion of cases of progressive muscular atrophy. The writer is thoroughly in accord with Gowers when he says: 'It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy, that Charcot's distinction is in effect giving a new name to an old disease. Whether there are [clinical] indications of lateral sclerosis or not depends on the circumstance whether the degeneration of the pyramidal fibres is or is not more extensive than the complete degeneration of the nerve-cells that cause atonic atrophy. If the latter is universal, the pyramidal tracts may be totally degenerated, and yet there may be none of the characteristic indications of such degeneration. On the other hand, both arms and legs may be the seat of the spastic paralysis that indicates pyramidal degeneration, and atonic atrophy may be limited to a few muscles of the hands.'

The writer in his practice has often found the atrophy of muscles either preceding or occurring simultaneously with the signs of spastic paralysis, and no regular division into stages such as has been described by Charcot. That bulbar symptoms occur in association with the other signs of progressive muscular atrophy is of course well known; and though their development may be often a late event, it is by no means always so.

The disease being, then, only a variety of progressive muscular atrophy in association with bulbar symptoms, nothing separate need be detailed concerning its **DIAGNOSIS, PROGNOSIS, and TREATMENT**, apart from what has been said concerning this latter disease. See **PROGRESSIVE MUSCULAR ATROPHY**. H. CHARLTON BASTIAN.

18. Multiple Sclerosis of the Spinal Cord.

SYNON.: Disseminated Sclerosis; Insular Sclerosis; Multilocal Sclerosis; Fr. *Sclérose en Plaques Disséminées*; Ger. *Multiple Sklerose des Rückenmarks*.

NATURE and ETIOLOGY.—This is a disease produced by the development of patches of sclerosis of varying size and shape throughout the spinal cord, and most frequently also in different parts of the brain. Clinically the disease is met with under the most diverse forms, according to the different sites and sizes of the patches of sclerosis occurring in different cases. These different forms of the disease are divisible into three partially distinct types, according as the morbid changes and symptoms occur in and are referable to: (1) the spinal cord alone (*spinal type*); (2) the cerebrum alone (*cerebral type*); or (3) the brain and spinal cord

(*cerebro-spinal type*). As the dominant symptoms of the disease are often those of the spinal type, even where there is also an extension of the morbid process to the cerebrum, it will be most convenient to speak here in the main of the 'cerebro-spinal' type. It is, moreover, both more frequent and a more characteristic malady than either of the simpler forms.

In regard to the aetiology of the disease little can be said. It may occur with or without the predisposing influence of a neurotic tendency. It is at least as common in females as it is in males; and though rarely occurring in children under ten years of age, it is perhaps most common between the ages of ten and thirty years. Beyond the age of forty it again becomes very rare.

Among the exciting causes, exposure to wet and cold would seem to take the first rank. After this come traumatic influences of various kinds, mental shocks or troubles, great fatigues from mental or bodily labour, and finally the state of convalescence from several acute diseases, such as typhus, cholera, variola, or other specific fevers. The disease has been said to occur sometimes as a sequel to severe and long-continued hysteria; but in some of such cases at least it would seem to be far more probable that the early and obscure symptoms connected with this affection were those which were regarded as hysterical. 'Hysteria' may be produced or simulated in many ways, but as itself a producer of organic changes its rôle is assuredly open to grave doubts.

ANATOMICAL CHARACTERS.—The patches of sclerosis which constitute the anatomical basis of this disease differ somewhat in their appearance (macroscopic and microscopic) and probably in their nature from the similar overgrowths of the neuroglia that occur in primary lateral sclerosis.

On the cut surface of the spinal cord, medulla, or other portion of brain, the foci of sclerosis mostly reveal themselves as greyish, greyish-red, or semi-gelatinous yellowish patches, differing principally, by reason of slight contrasts in colour, from the dead white of the more healthy columns of the cord, and from the natural appearance of the grey matter. The tissue of the patches may either be level with, project slightly above, or sink slightly beneath, the general cut surface of the cord. The same differences also exist in regard to those patches which involve the external surface of the cord—they may at times, when the new tissue is excessive, rise slightly above the surface; while later on, when shrinking has occurred in the cirrhotic patch, some amount of superficial depression may be met with.

The patches vary much in size; in the spinal cord they range from that of a mere pin's head to that of a large pea, or of a bean; while in the cerebrum or in the cerebellum they may attain still larger dimensions. In the spinal cord the patches occur in all parts of its longitudinal extent, and they may occupy very variable portions of the transverse area of the cord. Some involve principally the lateral, others the anterior or the posterior columns of the cord; or portions of the grey matter, either alone or in conjunction with one or more of these columns, may be implicated for a variable extent, transversely and longitudinally. Patches of different sizes, and varying in their transverse extent, occupy different levels of the cord, and may thus occur in an irregular series throughout the organ.

These spinal foci of sclerosis, again, may be associated with patches of the same kind distributed through the medulla, pons, and cerebral peduncles, in part superficially and in part within their substance. Similar patches may be found in variable number, and quite irregularly distributed, through other parts of the cerebrum, as well as through the cerebellum.

In regard to the *microscopical characters* of these foci of sclerosis, certain differences are met with in different cases, principally dependent upon the age, or stage of formation, of the patches. Without going into minute details, it may be said that there is in all cases a hyperplastic overgrowth of the neuroglia which naturally exists around and between the nerve-elements. The nature of this change becomes quite distinct when properly prepared sections of the cord have been stained with logwood or other dyes. The new tissue takes the staining fluid freely, and when the circumference of a patch (especially some small one) is examined, it becomes obvious that numerous thickened processes of neuroglia connect it with the healthy tissue around. It is by the hypertrophy and gradual fusion of these circumferential prolongations that the morbid growth progressively encroaches upon the previously healthy portions of the cord. As this more intermediate tissue grows, it presses upon and constricts the nerve-fibres and nerve-cells, so as to cause atrophy of the latter and a partial atrophy of the former. For there is reason to believe that the nerve-fibres do not wholly disappear; in these patches of primary sclerosis (as in the case of 'secondary degeneration') it is the white substance of Schwann which disappears, while the axicylinders, or a considerable number of them, persist. In the new tissue itself we find the usual granular or very finely fibrillary matrix, containing minute spherical or ovoidal plastides, also branched cells, and occasionally a few granulation-corpuscles. The latter are met with especially during the earlier stages of a patch of sclerosis; just as corpora amylacea or colloid bodies may be found in older patches. The walls of the capillaries as well as of arteries and veins are generally greatly thickened, and the vessels in a patch of this kind may be both numerous and large; in other cases, however, the number of vessels existing in the patch is by no means so conspicuous. It is well known that the adventitia or outer coat of the vessels in these patches is specially apt to become thickened, and that this sort of overgrowth may extend inwards, so as to cause fibroid degeneration of the middle coat, and even of the intima. It is probable that proliferation also takes place from the inner surface of the intima (an endarteritis), and that occasionally, owing to this cause, a thrombosis may be brought about. Certain it is that the writer has on several occasions found the larger vessels of a patch of spinal sclerosis blocked by an old and firm thrombus.

PATHOGENESIS.—With reference to the starting-point of a patch of sclerosis nothing definite can be said. Not infrequently disseminated sclerosis may be met with in the absence of any cachexia, syphilitic or other; and, moreover, patches of sclerosis may occur in the nervous system only, or to no notable extent in other organs of the body. This, therefore, would indicate the existence of some process of an abnormal kind taking place in the spinal cord or brain, and again not uniformly through them, but in foci situated here and there.

It is no explanation, as some seem content to suppose, merely to say that the abnormal processes are 'chronic inflammations'; since, whether it is or is not advisable to speak of the changes by this name, we should still have to ask what is the cause of such local departures from healthy nutrition. Does the process begin in the connective-tissue elements themselves? or is there some primary change in the small vessels (possibly of the nature of endarteritis) leading to obstructions and a sequential overgrowth of the neuroglia? It would seem pretty certain, at all events, that the change in the nerve-elements proper follows the overgrowth of the neuroglia—as certain, indeed, as that throughout a band of 'secondary degeneration' the order of these changes is exactly reversed. (There fatty degeneration and atrophy of the nerve-fibres are the first events, and these are followed by hyperplasia of the neuroglia.) See SPINAL CORD, Diseases of, p. 1523.

One of the most interesting facts in connection with these patches of primary sclerosis is to be found in the circumstance that they themselves rarely lead to bands of descending 'secondary degeneration' in the anterior or lateral columns, or of ascending degeneration in the posterior or lateral columns. The fact itself has been long observed, and always regarded as rather surprising. The writer believes it to be explicable by the fact previously mentioned, that the bulk of the axis-fibres remain, so that the nerve-fibres below the seat of lesion (or above in the case of the posterior and parts of the lateral columns) are not absolutely cut off from the nerve-cells which exercise a 'trophic' influence over them. Some nerve-tremors may still pass along the damaged fibres in the sclerotic patch,¹ and thus the nerves in the parts beyond do not degenerate, as they would do if the fibres had been absolutely cut across. Some fibres may be completely strangled and then absorbed, and in such a case the continuations of these nerve-fibres would degenerate. In the final stages of a sclerotic patch this kind of sequence is apt to occur; so that towards the end there may be the tendency to the occurrence of some amount of secondary degeneration, even though the degenerated fibres may not constitute a very compact band.

SYMPTOMS.—It can easily be understood, from what has already been said, how much the symptomatology of this disease is liable to vary in different cases, according to the varying situation, extent, and order of evolution of the morbid patches. That it is possible to assign anything like a definite symptomatology for this affection, is due to the fact that there are certain seats of election in which the patches of sclerosis are specially apt to occur. The sites affected with special frequency are the lateral columns of the cord, the medulla, and the pons; and it is with the occurrence of patches of sclerosis in these situations that we have the following set of correlated symptoms pertaining to the 'cerebro-spinal' type of the disease.

A slowly ensuing paresis of the lower extremities begins first in one limb, and then after a time involves the other. During this time the paresis develops into a more and more marked paralysis, though the sensibility of the limbs remains almost

completely unaffected—nothing more than a temporary numbness being complained of in the majority of cases, while 'lightning-pains' and girdle-sensations are altogether absent. After an interval, first one and then another upper extremity may become weak and subsequently more or less paralysed. During these early stages of the disease more or less distinct remissions of symptoms may occur from time to time.

Meanwhile a most typical sign soon shows itself in the paretic or semi-paralysed limbs in the form of a marked trembling or shaking of those muscles or parts of a limb which are called into voluntary action with any intensity (the so-called 'intentional tremor'), although these phenomena immediately subside when the voluntary exertion ceases. The involuntary movements consist either of extremely well-marked tremors, like those met with in some cases of paralysis agitans, or else of movements of greater range, more resembling those of chorea.

Later some paresis and tremors of the trunk-muscles may occur, as well as of those of the neck; and this may be followed by a similar affection of the tongue, lips, and facial muscles—possibly, also, of those of the palate, pharynx, and larynx. When a patient affected in this manner, who has been previously lying perfectly still in bed, is told to endeavour to sit up, shakings and tremors begin in almost all parts of the body, and the scene is strangely changed until all voluntary efforts cease, and the recumbent position is again assumed. The same kind of thing is seen when movements of particular parts of the body are attempted: thus when, in the sitting posture, the patient attempts to hold up one leg, tremors of it immediately begin; ask him to take hold of something or to squeeze a dynamometer, and the upper extremity called into action at once begins to shake; request him to put out his tongue, and immediately irregular protrusions of the organ occur, associated with twitches about the angles of the mouth and even in other parts of the body. The act of walking may cause, in more or less advanced cases, tremors of the legs, arms, trunk, head, and neck—all at the same time.

Movements of slight intensity occasion either no shakings or merely tremors of a very fine kind. The latter are seen in the early stages of the disease when writing is attempted. Almost every letter registers a number of fine tremors, mixed here and there with greater irregularities. In more advanced cases, however, the movements are so disorderly that writing becomes either impossible or wholly illegible.

Just as there is for some time only slight or patchy loss of ordinary sensibility, so we find that patients often remain fully conscious as to the positions and movements of their limbs, and that closure of the eyes causes no increased uncertainty of their movements unless the posterior columns be distinctly affected; and, except under these latter conditions, when in the standing position, they are not rendered more giddy or more unsteady by closure of the eyes. Later on this sign is very commonly met with, just as there is also an impairment of muscular sense in the upper extremities, so that the patient cannot, with eyes closed, readily touch the tip of his nose with either fore-finger. The co-existence of intentional tremor would, of course, exaggerate this disability.

Up to this stage there may be no distinct interference with the functions of the bladder or the

¹ In support of this, there is the fact mentioned by Charcot, that an optic nerve which was affected through its whole thickness by sclerosis was yet capable of performing its functions.

rectum. The tendon-reflexes are, however, generally distinctly exaggerated: ankle-clonus may be obtained with readiness, and the knee-jerk is often more pronounced than usual. The plantar reflex is of extensor type. There is no tendency to the formation of bed-sores (unless sensibility is gravely impaired); no wasting of muscles; nor is any alteration in their electrical excitability met with.

After variable and often long periods, the affected lower extremities, which have become more and more paralysed, may in some cases show signs of commencing bar-like rigidity. The limbs, as the patient lies in bed, are closely drawn together, and in a condition of rigid extension, which is generally increased when any attempts to move them are made. At first this condition of the limbs ensues from time to time, in the form of paroxysms lasting for an hour or two. But, later, the attacks become both more frequent and longer, so that ultimately the condition of rigidity becomes permanent. Contractions of the arms are less common, and when they occur they become fixed at times in a different position from that met with in simple lateral sclerosis (*see* 16. Spastic Spinal Paralysis); that is, like the lower extremities, in a condition of extension, and closely drawn to the sides of the body. At this period ankle-clonus can often be elicited with the greatest ease, and the movements set up in the one leg may extend to the opposite lower extremity, and may indeed lead to more or less of general tremor throughout the body. Exposure to cold, or irritation of the skin in various ways, will also often suffice to initiate this general tremor, which, as Brown-Séquard showed, may commonly be caused to cease instantly by a forcible flexion of one of the great toes. With the cessation of the tremors consequent upon this manœuvre, the limbs may also be left for a time in a supple and flaccid condition.

The manifestation of tremors of the tongue, lips, and face, together with the existence of a distinct jaw-jerk, is of course a sign that the bulb is affected; and when this occurs, simultaneously or very soon after, other evidences of implication of the bulb and of contiguous portions of the cerebrum may be met with. Articulation may become more or less affected, the speech being rendered slow, hesitating, and measured, syllable by syllable; or it may be jerky in character—becoming especially thick and blurred in the later stages of the disease. The power of swallowing is less frequently impaired, but in advanced stages it is apt to be affected.

Nystagmus is almost invariably met with. Diplopia, or actual paralysis of the ocular muscles, is rare. Amblyopia not infrequently exists, perhaps in one eye only, associated with a variable amount of atrophy of the optic discs. Actual blindness is very rare.

Vertigo, sometimes to a marked extent, is no uncommon symptom; and as the cerebrum becomes more and more affected, a condition of well-marked hebétude, or actual dementia, gradually becomes pronounced. This betrays itself externally by a blank, expressionless aspect of the face; the patient becomes childish in manner, his memory fails, and he takes interest only in trifles; he may also laugh constantly without adequate cause, or, on the other hand, is very easily moved to tears.

During this condition of things a subacute maniacal condition may supervene; or the patient may develop 'delusions of grandeur' precisely

similar to those met with in general paralysis of the insane—examples of which the writer has recently seen in two of his own patients. In other cases persons suffering from this disease may lapse into a profoundly melancholic condition. *See* p. 769.

At this stage, too, apoplectiform or epileptiform attacks are particularly apt to occur from time to time. After such attacks, of whichever kind, the limbs on one side of the body and the face are left more or less paralysed; and where the attack has been epileptiform in character, the convulsive twitchings are often limited to this one side of the body. As Charcot has pointed out, these attacks are precisely similar to those which occur in general paralytics, or in cases of old hemiplegia with descending sclerosis. They answer to the so-called 'congestive attacks,' but they do not seem to be associated with any new appreciable lesions of a 'gross' order. Such epileptiform attacks may be brief, or they may last for hours; or, off and on, even for days. In all of them the temperature begins to rise almost at once—without any initial period of depression—and may even reach 104° in a few hours, or in a day or two. The temperature then begins to fall again; or, should it continue to rise to a still higher point, the attack is very apt to terminate fatally.

Every attack of this kind leaves the patient in a manifestly worse condition, both bodily and mentally; and perhaps in one of them at least death may occur.

VARIETIES. — The symptomatology of this disease is likely to be considerably modified in different cases, but principally in two directions, productive of complications of the same kind as those which are also apt to occur in 'spasmodic spinal paralysis.' In each disease there may in some cases be a special affection of the posterior columns, in one or other region of the cord, bringing with it more interference with sensibility, and an admixture of other symptoms pertaining to locomotor ataxy. These are some of the cases which have been described under the separate name of 'ataxic paraplegia' (*see* PARAPLEGIA, ATAXIC). It is, perhaps, principally in these cases that the '*crises gastriques*' (pains, vomiting, and occasionally diarrhoea) are also met with. In other instances there may be an extension of the sclerosis to the grey matter of the anterior cornua in one or other region (as well as to other parts of the grey matter), leading, among other phenomena, to muscular atrophy in related regions of the body. In either of these ways the symptoms of the original disease may be complicated, and, to a certain extent, obscured.

Many other differences also present themselves in special cases, owing to the varying situations in which the morbid patches make their first appearance. In a distinct minority of the cases the disease seems to reveal itself first in the brain rather than in the spinal cord.

TERMINATIONS. — After this disease has pursued a very slow course for years (often five to ten), the miserable sufferers from it may at last be carried off in various ways. Death may take place in one of the apoplectiform or epileptiform attacks, occurring either in patients who are merely slightly demented, or in those who are otherwise actually insane; or, at last, in cases in which there is great interference not only with articulation but also with deglutition, the functions of the heart or of

respiration may also become affected, and this disturbance may lead on to a fatal termination.

In other cases, after the disease has lasted for years, and when the grey matter of the cord has become seriously involved, accidents may supervene similar to those which occur in the final stages of many cases of paraplegia. The bladder may become paralysed, and after a time inflammation and ulceration may be set up, followed by secondary inflammation of the ureters or kidneys. Or bed-sores may form, sloughing may go on extensively, and the patient may at last die exhausted, or from the supravention of blood-poisoning. At other times the patient is cut off by some acute inflammatory disease.

DIAGNOSIS.—In its early stages the diagnosis of this disease may present very considerable difficulties. This is especially the case when the morbid process begins in the cerebrum. Here for a time there may be nothing distinctive, and we have to wait for the further development of the disease before anything like a positive diagnosis is possible. Similarly, where the disease begins only with spinal symptoms, it is often extremely difficult to diagnose it with certainty in its very early stages. The important characters in the more typical forms of the disease are the comparative youthfulness of the patient, the paresis gradually increasing, first in one and then in the other lower extremity, with little or no alteration in sensibility or in the electrical irritability of the nerves or muscles, but with ankle-clonus and exaggerated knee-jerks. Still it must be borne in mind that all these signs may be met with in the spastic forms of Functional Paralysis of Spinal Type (p. 1562); and, especially when they occur in young women, the diagnosis is often beset with the greatest difficulties. Even the most experienced observer may remain for a time in doubt as to whether he has to do with the beginnings of organic disease, or with mere functional derangements of the spinal cord. But when the peculiar tremors and disordered movements on voluntary excitation of the muscles are met with, together with the absence of any such tremors in the condition of rest, and some amount of paresis or of similar symptoms in one or both upper extremities, the diagnosis of the 'spinal' type of this disease can be no longer difficult or doubtful.

By far the most typical cases, however, are those of the 'cerebro-spinal' type, in which, with such symptoms as are above indicated, there are also some others due to disease of the medulla, pons, or adjacent parts, such as tremors about the lips and tongue, altered speech, and nystagmus. In these cases the disease is really quite distinctive; so that the malady ought to be readily recognised when the patient is seen at this stage for the first time. Chorea is the affection with which such a stage of the disease is most apt to be confounded; but the absolute cessation of all tremors and disordered movements in multiple sclerosis when the patient is at rest, and their immediate re-initiation (mainly in the parts moved, but also generally to a less extent in others) on the occurrence of voluntary efforts, is a thoroughly distinctive characteristic.

Paralysis agitans ought to be distinguished from disseminated sclerosis with even more ease. It is scarcely ever met with in persons under the age of thirty-five, while multiple sclerosis does not very often begin in persons beyond such an age. Again, the stooping gait of paralysis agitans is very dis-

tinctive, and the slow movements of patients suffering from this disease are only to a slight extent exaggerated by voluntary exertion of the parts, while sometimes they are actually lessened thereby; and such movements, in the form of fine tremors, do not cease to anything like the same extent under conditions of rest. There is also no shaking of the head and neck in paralysis agitans.

Mercurial poisoning with tremors can be easily distinguished, on inquiry into the history of the patient, and the mode of onset of the disease.

In those more irregular cases of multiple sclerosis, in which there is either an unusual amount of implication of the posterior columns of the cord, or of the grey matter in some region or regions, the diagnosis of the complex nature of the affection must be based upon the general principles applicable to the regional diagnosis of spinal-cord disease.

PROGNOSIS.—Absolute cure of this disease is scarcely to be hoped for. The most that has been done, hitherto, as a result of treatment, has been to help to bring about more or less distinct remissions, and also to delay the progress of the disease. Death often occurs in from five to ten years, in one or other of the modes already indicated, though at times there may be long periods of quiescence, and the duration of life may be much more protracted.

TREATMENT.—Many drugs have been tried, but hitherto with little or no positive result, in the treatment of this affection. Nitrate of silver has seemed to do good in some cases, especially in the early stages. But the writer is much more disposed to trust to iodide of potassium in eight- or ten-grain doses three times a day, with or without moderate doses of liquor arsenicalis, or of perchloride of mercury; combining the use of those drugs with cod-liver oil, alone or with maltine, and a good nourishing diet. From time to time, however, the above medicines should be omitted, and simple tonics taken in their place. In the early stages of the disease, hot brine- or sulphur-baths and massage of the limbs may be of service; and in all cases it is of great importance to see that the patient obtains sound sleep, since in this, as in all other chronic spinal diseases, the patient's downward course is likely to be hastened where an adequate amount of sleep is not obtained.

No distinct indications exist for the treatment of this affection by electricity, and no advantages have as yet been recorded from its use. The complications of the disease, which may occur in its later phases, must be treated in accordance with the general principles applicable to this as well as to other spinal affections. Every effort must be made to preserve the general health of the patient, as this will probably be found to be the surest means of holding in check the progress of the disease.

H. CHARLTON BASTIAN.

19. Friedreich's Ataxy.—See FRIEDREICH'S ATAXY.

20. Combined Degeneration of the Spinal Cord.—See COMBINED DEGENERATION OF THE SPINAL CORD, p. 325.

21. Spinal Cord and Membranes, Tumours of.—SYNON.: Fr. *Tumeurs de la Moelle*; *Tumeurs Rachidiennes*; Ger. *Krankhaften Geschwülste des Rückenmarks*.

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—Tumours originating in the substance of the spinal cord may be regarded as belonging to two classes,

according as they represent (a) mere local accidents in the form of perverted tissue-changes; or (b) such local accidents developing under the influence of a distinct *general state*, such as syphilis or tuberculosis.

(a) Of the purely local overgrowths, the most typical, and perhaps also the most frequently occurring, are *gliomata*. The consideration of these growths comes in natural sequence to that of *sclerosis* affecting different regions of the cord. In such a tumour we have an exuberant overgrowth, as Virchow and other pathologists suppose, starting from the neuroglia of a certain portion of the cord. At first the growth infiltrates and substitutes itself in the place of a certain amount of nerve-tissue; but it soon grows excessive in quantity (spreading in area perhaps at the same time), and thus comes to exercise a more and more marked compression upon the remaining tracts of nerve-tissue composing the cord at the same level, within the narrow and unyielding boundaries of the spinal canal. In extreme cases a spinal cord may, as the writer has seen, become so infiltrated with new-growth throughout the greater part of the cervical and dorsal regions, that it attains almost twice its natural bulk. These gliomata are oftentimes extremely vascular. They are liable to undergo a certain amount of central softening; and into their substance, especially in the softened foci, hæmorrhages are very apt to occur. Softening of nerve-tissue may also, at a certain stage, take place around a circumscribed growth, and thence may extend for a variable distance above and below.

Other tumours of an allied nature, such as *sarcomata* and *myxomata*, also at times develop, either in their pure types or with blended characters, within the spinal cord. They present few intrinsic peculiarities in their manner of affecting the cord. They rarely attain any large size; indeed the limitations of the spinal canal only permit of much increase in one direction. Thus elongated growths are occasionally met with. To a considerable extent, such tumours have an infiltrating mode of growth, though their boundaries are apt to be rather more defined than are those of gliomata.

In regard to the causes of these tumours, almost nothing more definite can be said than that they seem, at times, to find occasion and conditions suitable for their initiation after some blow upon the spine or concussion of the spinal cord.

(b) Of the growths which tend to occur in the spinal cord (as occasionally in other parts of the body) under the influence of some *general disease* or diathetic condition, two are especially to be named. These are *tubercular growths* and *syphilitic gummata*. The former are generally small, varying in size from a mustard-seed to a pea, and only very rarely attaining the dimensions of a hazel-nut. Next to gliomata, they are the new-growths most frequently met with in the substance of the spinal cord. When small, they may occur in association with a cerebro-spinal tubercular meningitis; but at other times they are found, and especially the larger growths, existing independently of any acute inflammation of the meninges. In this latter case, the tumours may be combined with a certain amount of adjacent and secondary softening of the substance of the cord.

Syphilitic gummata, originating in the cord itself, occur only with the greatest rarity. They are more frequently found starting from the meninges, and

then they may press upon or actually grow into the nerve-substance.

Cancer is believed not to occur primarily in the substance of the spinal cord, though it may grow into its substance, or seriously press upon it, when originating either in the meninges or in the vertebræ.

Growths from the spinal meninges, or adventitious products within the spinal canal pressing upon the cord may most advantageously be referred to here. The *new-growths* that occur are similar to those met with in the cord itself, with the addition of others, and more especially *sarcomata* and *myxomata*. The principal adventitious products are such parasites as *cysticerci* and *hydatids*. The former are very rare and comparatively unimportant. Hydatids, however, are of much more importance. They may be found within the dura mater, but they have been met with much more frequently outside this membrane, often forming large tumours contiguous to and encroaching upon the spinal canal.

SYMPTOMS, COURSE, AND TERMINATIONS.—The difficulties of diagnosis are almost always very great in the case of tumours of the spinal cord, because in their early stages, and occasionally for prolonged periods, they are associated with slight and somewhat vague symptoms.

Independently of the variations in different cases, consequent upon the longitudinal situation or level of the tumour in the spinal cord, the symptoms to which they give rise in various parts of the body may be more or less vague anomalies of sensibility in different regions, associated with a certain degree of weakness, often not amounting to actual paralysis.

Growths from the meninges, or from the vertebræ, pressing upon the spinal cord, are not quite so apt to run a latent course for any length of time, since they are rather more prone to involve the anterior or the posterior roots on one or on both sides—at first irritating them, and subsequently causing paralysis from pressure. Thus localised numbness, pains, or anæsthesia, either alone or associated with twitching, cramps, or paralysis, confined to certain parts of the body, are rather more common incidents during the growth of extra- than of intra-medullary tumours. Still the diagnosis between these two classes of tumours may be impossible.

Sclerosis, in its 'insular' form, especially when the patches are few or close together, may also present symptoms almost inseparable from the first stage of some intra-medullary tumour. The important fact is, however, that sclerosis in the cord tends to become more and more generalised, and thus gives rise to a proportionately widening range of symptoms; or else it limits itself to special columns, and thus becomes associated with more special sets of symptoms.

With any of these tumours of the spinal cord, the symptoms are, after a time, liable to undergo a sudden and grave increase, owing to the occurrence of a hæmorrhage into its substance and perhaps into adjacent regions of the spinal cord, or else owing to the commencement of a process of secondary transverse softening. Beyond these possibilities of sudden grave augmentation of symptoms, the course of intra-medullary tumours is also apt to be marked by peculiar exacerbations and remissions from time to time, in association with periods of altered growth or vascularity of the tumour itself.

DIAGNOSIS.—The very gradual onset of the symptoms in cases of tumour of the spinal cord or of its

meninges is a point of great importance in the diagnosis of these conditions. Thus, for instance, we eliminate arachnoid or intra-medullary hæmorrhages, and also the numerous class of cases of softening of the spinal cord, with other affections having a more or less abrupt origin. The diagnosis of tumour of the cord as distinct from its compression by disease of vertebræ (where there is also generally a slow evolution of paralytic symptoms) must be based in part upon the absence of distinct pains and of any evidence of vertebral disease. The diagnosis from meningeal tumours has already been referred to under the head of Symptoms; and so also has the diagnosis from mere sclerosis of the spinal cord, in which the connective-tissue overgrowth is not sufficiently bulky to amount to an actual tumour.

If the arrival at a diagnosis as to the existence of a tumour of the spinal cord is a process beset with difficulties, these by no means cease when, passing from the primary, we have to approach the secondary question as to the *nature* of the growth presumed to exist. But little is possible in this direction. It is true that, with a history of pre-existing syphilis, even without the evidence of other simultaneous manifestations, we should be warranted in assuming it to be even more than possible that an existing growth was syphilitic in nature, and in treating the patient accordingly; and that all the more because this is about the only kind of new-growth as to which we have distinct evidence of its amenability to the influence of remedies. The presumptions in favour of the tubercular nature of a supposed new-growth in the spinal cord would rarely carry with them more than a moderate amount of cogency. Still, occasionally the general habit of the patient, together with the fact of the existence of scrofulous enlargement of glands, or of some forms of phthisis, might give more or less probability to such a conclusion. Beyond this, not much can be done in the way of diagnosing special kinds of tumours. We might be guided in our opinion as to the possible existence of a sarcoma by the presence of one or more of such growths in other parts of the body; or, failing this, we may recollect that primary cancer affecting the spinal cord is almost unknown, and that gliomatous tumours are, next to the tubercular, those which are most frequently met with in the cord itself. The diagnosis of hydatids may be impossible, as the writer has seen in a recent obscure case in which there was no evidence of hydatids elsewhere, and no local swelling in the tissues outside the spinal canal. Operation, on the assumption that the paraplegic condition might be due to Pott's disease, revealed the true nature of the case, and terminated in recovery.

PROGNOSIS.—The prognosis in all the cases of intra-medullary growth is bad. Life, it is true, may last for months or even years, but the tendency is for the primary affection to set up other secondary accidents, in the form either of hæmorrhage or of softening. Thus, paralysis is rendered more complete, and the way is paved for an ultimate fatal termination, through the intervention of cystitis and renal mischief; by way of bed-sores with exhaustion and blood-poisoning; or by extension of softening upwards to the cervical region, and the supervention of respiratory paralysis.

In the cases of extra-medullary growths the prognosis is not so grave. Syphilitic growths from, or

thickenings of, the spinal meninges may yield to treatment; while other forms of meningeal growth or adventitious products may be capable of diagnosis and of relief by operation.

TREATMENT.—In the case of the existence of a syphilitic tumour in the spinal cord or in its meninges, we may attempt (and with some expectation of success) to treat the causal morbid condition with large doses of iodide of potassium (gr. xv–xxx) in combination with perchloride of mercury. But in almost all other cases of intra-medullary growth little can be done in this direction, and we are reduced to the necessity of dealing with the paraplegic state and its attendant conditions as best we can, and also of attending to the general health, with the view of arresting the progress of the disease and keeping its possible complications in check.

See 9. SPINAL CORD, Softening of.

For tumours of the spinal meninges or hydatids within the spinal canal, we must call in the aid of the surgeon.

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22. Syringomyelia.—**SYNON.**: Syringomyelitis; Hydromyelia; *Hydorrhachis Interna*; Central Gliomatosis; Fr. *Myélite Périépendymaire*; Ger. *Syringomyelie*.

This condition has been long known on its pathological side, but it has been only of late years connected with anything like a definite symptomatology. Formerly, therefore, the disease was never diagnosed during life; the morbid condition was only recognised after death as the cause of previously obscure and little-understood symptoms. During recent years, however, it has been otherwise—many diagnoses have been made during life and subsequently verified.

ÆTIOLOGY AND PATHOLOGY.—There are two principal conditions in which cavities are found in the spinal cord; and though in their typical forms they may be quite distinct from one another, in many other cases they exist in combination and are not separable conditions. The simple condition is known as *hydromyelus* or *hydromyelia*—a state in which there is a dilatation of the central canal of the cord, and in which this canal is distended with fluid; this being either a congenital condition, or perhaps a result later in life of the pressure of a tumour on a portion of the central canal, which obliterates it at the seat of pressure, and leads to the dilatation above. The more complex condition is what is known as *syringomyelia*, in which, though there is dilatation of the canal of the cord as before, there is also a gliomatous overgrowth or new-formation around this dilated canal, which extends more or less into different parts of the posterior half of the cord.

It is commonly supposed that there is some congenital defect in the structure of the cord in these latter cases, so that the central canal remains large, and has around it a variable amount of the unaltered embryonic tissue of the organ, closely allied in structure to the neuroglia, some amount of which always exists at the surface of the cord as well as around its central canal. At some variable period in the life of the individual (but mostly between puberty and the twentieth year) growth seems to take place to a more or less marked degree in this embryonic tissue, resulting in the production of a gliomatous new-formation, which infiltrates to a variable extent the grey matter, and also the posterior and postero-lateral columns. The symptoms

of the disease are thus gradually produced by the increase and invasion of the new growth, and they will naturally vary in their rapidity of evolution with the rapidity or otherwise of increase in the new tissue itself. Cavities other than the enlarged central canal are frequently found co-existing in this gliomatous new-formation, which have probably been formed therein by degenerative changes.

The dilatation of the central canal sometimes exists throughout the entire length of the cord, and there may likewise be a dilatation of the *iter* between the third and fourth ventricles of the brain. In other cases—and this is the most frequent condition—the cavity only involves the cervical and the upper dorsal region of the cord, or perhaps a still more limited longitudinal area. The transverse section of the cavity may be oval or circular, while in other cases there are narrow chink-like extensions into one or both posterior cornua. Other independent more or less chink-like cavities are also frequently found, which, as before indicated, are supposed to have resulted from degenerative processes or from hæmorrhages occurring in the new gliomatous tissue.

In brief, the condition seems to be due in the first place to a congenital arrest of development, supplemented most frequently somewhere between the fifteenth and the thirtieth year, without assignable cause, by an overgrowth of gliomatous tissue of varying extent and rapidity around the dilated central canal and into contiguous regions of the cord; the symptoms being due in the main to this latter process, to pressure occasioned by the fluid or by the new tissue itself, as well as to the subsequent changes taking place in the infiltrated regions of the cord.

SYMPTOMS.—The symptoms of the disease often begin between the fifteenth and the twentieth years (rarely later than the thirtieth), and they may continue for ten to twenty years before a fatal termination occurs.

Patients complain of variable pains in the cervico-scapular region, and some amount of muscular atrophy soon shows itself about the arms or shoulders. Occasionally there may be atrophy also of some of the muscles of the leg.

The arms, or arms and legs, also become more or less weak; the deep reflexes are exaggerated; and some amount of rigidity may likewise show itself in the lower extremities.

With these symptoms there are very characteristic modifications of sensibility. Tactile sensibility and the muscular sense may be little if at all impaired, but painful and thermic impressions (which are conducted to the brain through the central regions of the cord) are lost in one or both arms and in contiguous parts of the trunk. The distribution of the areas affected by this loss of sensibility varies much in different cases; sometimes they are unilateral and sometimes bilateral. In one case under the writer's care the whole of the trunk and the two upper extremities were involved, except for a broad band about six inches wide which encircled the body just above the level of the umbilicus. Less frequently, and mostly in the later stages of the disease, there may be complete anæsthesia of the skin and mucous membranes over the whole or part of one side of the body. The special senses are generally unaffected, and power over the sphincters is also unimpaired. Some of the ocular muscles may be weakened or paralysed where the dilatation of the central canal

extends upwards so as to affect the *iter*, and is so great as to lead to pressure upon some of the contiguous nuclei for the ocular nerves. Under similar conditions nystagmus may also not infrequently be present.

Trophic troubles, other than the muscular atrophy, are not at all uncommon in the course of this disease. In the first place it may be said that such patients frequently burn themselves about the upper extremities, owing to the loss of painful and thermic sensibility. Other changes, however, occur spontaneously, such as alterations in the joints of the tabetic type, or ulcerations of the skin. In these cases it may be suspected that affections of the peripheral nerves are associated with the changes in the spinal cord—a combination which has been proved to exist in a remarkable variety of syringomyelia first recognised in Brittany, and known as 'Morvan's disease,' which is characterised by neuralgic pains, cutaneous anæsthesia, and painless but destructive whitlows. See p. 1028.

DIAGNOSIS.—The diagnosis of syringomyelia is now fairly easy in the majority of cases. It is the combination of loss of painful and thermic sensibility, without loss of tactile sensibility, in association with muscular atrophy in the upper extremities more especially, which is characteristic of the disease.

Formerly this affection was sometimes confounded with anomalous cases of disseminated sclerosis, though such a mistake is much less likely to occur now. It bears most resemblance to certain cases of 'hypertrophic cervical pachymeningitis' (see MENINGES, SPINAL, Inflammation of, p. 977), though these latter cases may usually be distinguished by the greater amount of pain in the nape of the neck and in the upper extremities, together with a certain amount of spasm, and the loss of tactile as well as other modes of sensibility, instead of the dissociated anæsthesia so characteristic of syringomyelia.

PROGNOSIS.—The prognosis is hopeless as regards cure or control of the disease, but the affection is a very chronic one, and therefore not specially dangerous to life till a rather long series of years has elapsed.

TREATMENT.—Treatment can only be directed to the maintenance of the general health, and to the alleviation of pains where they are distressing by means of acetanilide or of morphine. Massage and electrical treatment may also be of some use during the early stages of the disease.

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23. Spinal Cord, Malformations of.—Various conditions are comprised under this head which are of little or no interest to the practitioner. The spinal cord may be absent, imperfectly developed, or double. Again, cases occur in which the spinal cord is either unduly long or unduly short, or in which it may present some trifling lack of symmetry. One of the most interesting of these latter conditions is due to the fact discovered by Flechsig of the possible non-uniform distribution of the pyramidal tracts upon the two sides of the cord, so that the amount of decussation of the motor fibres, not only in different individuals, but also in the two halves of the same cord, may be quite unequal. In the latter case a slight asymmetrical development of the antero-lateral columns on the two sides would be met with.

Congenital Dilatation of the central canal of the Spinal Cord (*Hydrorrhachis interna*, or *Hydromyelus*) has already been referred to as constituting one side of the disease described as 'Syringomyelia'; in which, however, other cavities are frequently found resulting from the breaking-down of the gliomatous new-growth.

Congenital dilatation of the central canal in its most developed form is apt to be met with also in some cases of *spina bifida*; while in others the canal in the lumbar region is open posteriorly, and the halves of the posterior columns are more or less widely separated; there is, in fact, a congenital arrest of development in the spinal cord very similar to that which exists in the spinal canal. See SPINA BIFIDA.
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II. Diseases of the Spinal Cord dependent upon unknown, or very imperfectly known, organic changes.

24. Tetanus.—See TETANUS.

25. Tetany.—See TETANY.

6. Torticollis.—See WRY-NECK.

27. Writer's Cramp, &c.—See WRITER'S CRAMP.

28. Reflex Paraplegia.—SYNON.: Urinary Paraplegia (in part); Fr. *Paraplégie Reflexe*; Ger. *Reflexlahmung*.

GENERAL REMARKS.—Some practitioners believe that paralyzes of various kinds are brought about purely by reflex influences. They would include under this category some of the cases of paralyzes of separate muscles, such as the ocular; some cases of paralysis of one or both arms; or some of the cases of paralysis of one or both lower extremities. It is the last class of cases with which we are now specially concerned, though most of what is to be said in the present article may, *mutatis mutandis*, be considered applicable to the whole class of so-called 'reflex paralyzes.'

Those who believe in the frequent existence of this form of paralysis are considerably less numerous than they were about forty years ago, when the notion of its frequency and importance was warmly espoused by Brown-Séquard, at a time when the morbid anatomy of the spinal cord was very imperfectly known. The number of competent observers was then smaller, and the difficulty in detecting morbid changes in this organ was also much greater than it is at the present time, when we are accustomed to employ more elaborate methods for its preservation and for its examination. Yet one of the strongest of the arguments brought forward in favour of the existence of 'reflex paraplegia' was the absence of discovered lesions in the spinal cord in a class of cases reported upon by Stanley, in which paraplegia was associated with various morbid conditions of the urinary organs—cases, in fact, of the so-called 'urinary paraplegia.' And one of the main supports for the opinion of those who still believe in the existence of a class of reflex paraplegias would even now lie in the absence, in certain cases of paraplegia terminating fatally, of any actually discovered lesion.

ÆTIOLOGY AND PATHOGENESIS.—The interpretation of the paralyzes of this class put forward by Brown-Séquard was as follows: That an irritation, operating upon certain sensory nerves, produced impressions which, after impinging upon the pro-

perly related grey matter in the spinal cord, are thence in part reflected along vaso-motor nerves regulating the calibre of certain blood-vessels which supply either (a) the portion of the spinal cord in relation with the paralysed parts, or else (b) the great nerves or the muscles themselves of the paralysed parts. In either case this reflection of impressions resulting from irritation of sensory nerves, upon such special groups of vaso-motor nerves, is supposed to lead to a persistent spasm of the vessels which they innervate, so as to cause a continuous anæmic condition, either of certain vascular territories in the spinal cord itself, or else of the related nerve-trunks and muscles. In either case, too, the nutrition of the parts involved in this anæmia is supposed to suffer—so that their functions can no longer be carried on, or only in a very imperfect manner—and thus a more or less complete paralysis results, which is capable, however, of being mitigated from time to time, of actually intermitting, or indeed of being abruptly cured, according as temporary diminutions or a complete disappearance of the original exciting cause may lead to a diminution or to an actual cessation of the supposed profound anæmia produced by the postulated spasms of vessels. These are the theories upon which the doctrines of 'reflex paraplegia' are based.

Among the sources from which the initial irritation is supposed to proceed, almost all parts of the body, internal as well as external, are included. Thus irritative impressions, it is thought, may emanate from almost any part of the urinary tract—from the urethra to the kidney; in other cases similar impressions may emanate from some portion of the female genital organs; in others from the intestinal canal, owing to the presence of worms or some such persistent causes of irritation; in others from some portion of the thoracic organs; or, as it seems to be held, from irritated sensory nerves in almost any part of the body, whether situated near the surface or deep among the tissues.

The assemblage of symptoms supposed to characterise these forms of reflex paralysis presents nothing like a distinctive mode of grouping. And of the several components of the group put forward by Brown-Séquard, as pertaining to one of the most typical varieties, namely, 'urinary paraplegia,' none can now have any pretensions to be regarded as distinctive, excepting the alleged tendency of the paralysis to vary in degree with variations in the malady on which it is supposed to depend, together with its tendency to spontaneous or easy cure coincidentally with or soon after the cessation of the urinary troubles, whatever they may have been. In harmony with this latter character also are the alleged facts that speedy cures have been brought about of cases of paraplegia, especially in children, after the expulsion from the alimentary canal of tapeworms or round-worms; or of cures of the same disease in adult females after the cessation of some uterine inflammation; or of cures of a paralysis of ocular muscles after the removal of some carious tooth which had previously been exercising an irritative influence upon branches of the dental nerve.

It would be useless to attempt to deny the existence of such cases; they are theoretically possible. On the other hand, the writer is compelled to believe, after a very extensive experience, that, if they exist, they can only occur as extremely rare events.

Although it is theoretically possible that an irritation of a sensory nerve may be reflected on to vaso-motor nerves, so as to lead to arterial spasms in certain territories of the spinal cord, or in certain groups of muscles, it is difficult here, as it is when postulated in explanation of other functional diseases of the spinal cord, to imagine that such a condition of spasm could be maintained for weeks or even months. Nor, if it could occur for these prolonged periods, and to such an extent as to annul some of the most important functions of the spinal cord during this time, is it at all clear that the nutrition of the cord in the affected regions would not be seriously interfered with by such prolonged anæmia; and, if so, the assumed speedy resumption of healthy functions *pari passu* with the diminution or disappearance of the vascular spasms would constitute another difficulty, since such speedy recovery would be scarcely compatible with the theory upon which the explanation of the disease is based.

Again, it is almost certain that many of the cases formerly supposed to belong to this category of 'reflex paralysis' had no right to figure therein. Cases of diphtheritic paralysis have been proved to belong to a different category; and there is good reason to believe that in other instances the morbid conditions really existing as causes of the paralysis have simply been overlooked, either because the appreciable changes were only slightly advanced at the time of the patient's death (owing to the brief duration of the illness); or because of the want of a thorough examination of the cord, conducted with all needful aids, care, and expenditure of time; or, finally, because the disease may have been in the peripheral nerves rather than in the spinal cord itself.

It seems clear, therefore, that the opinions of those who believe in the existence of 'reflex paralysis,' and of 'reflex paraplegia' in particular, stand much in need of further support and definition. Well-observed and well-recorded instances of the disease are urgently wanted, if 'reflex paraplegia' is to retain its claim to a place in our nosology.

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29. Intermittent Paraplegia.—SYNON.: Intermittent Spinal Paralysis; Fr. *Paralysie Spinale Intermittente*; Ger. *Intermittirende Paralysis Spinalis*.—Very few cases of paraplegia of this type have been recorded, and it must also be a condition of extreme rarity.

The earliest recorded example was made known by Romberg, and as this, both in its nature and its course, seems to have been a typical instance, it may be cited here. 'A woman, sixty-four years of age, after being quite well the day before, was suddenly attacked with paralysis of the lower extremities and of the sphincters. Sensibility was unchanged, consciousness clear, the temperature cool, pulse 80, small and empty, no pain in the spinal cord. The next day there was an astonishing change in the condition. The patient could walk again and void urine voluntarily, and only complained of weakness in the legs. The following morning there was paraplegia again, which had set in at the same hour as it had done two days before. A third paroxysm was awaited, which also set in at the appointed time, although without paralysis of the sphincters. Quinine effected a rapid cure.'

Additional cases have since been recorded by Erb, Hartwig, and other observers; and the view now

entertained concerning them is that they are due to multiple neuritis or to a multiple peripheral neuritis, rather than to any disease of the spinal cord itself. See NEURITIS, MULTIPLE.

Any future cases should be observed and recorded with the greatest care. Meanwhile it must be remembered that those already observed seem to have proved extremely amenable to the influences of quinine and of arsenic.

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30. Functional Paralysis of Spinal Type. SYNON.: Hysterical Paraplegia.

It is generally admitted that paralysees of the 'hysterical' type more frequently belong to the paraplegic than to the hemiplegic variety. Even good observers, however, are rather too prone to look upon the terms 'functional' and 'hysterical' as interchangeable. This is apt to create confusion. Surely there may be functional paralysees which have no right, merely as such, to the appellation 'hysterical.' Certain it is that in a considerable proportion of cases, which we seem justified in regarding as functional, there may be, apart from the paralysis, no symptoms or mental peculiarities which could be described as 'hysterical.'

This whole subject bristles with difficulties, for, as the writer has endeavoured to point out, the different forms of functional paralysis must be dependent upon faulty nutrition and faulty functioning (apart from gross organic disease) of this or that territory of the brain, and probably also of this or that region of the spinal cord. This being so, functional paralysees should closely accord in their characters with the forms of paralysis caused by actual gross lesions in different regions of the brain and spinal cord; though the two classes may differ in severity and in curability—one or both.

'Some appear to think (or their language seems to imply) that all cases of paralysis not due to a structural cause must be of a hysterical order; and, as they believe hysteria to be due to a perverted activity of the brain, it perhaps never occurs to them to consider whether some of the cases of functional paralysis coming before them may not have their origin in a depressed or perverted activity in some portion of the spinal cord. For one or other of these reasons, it happens that up to the present time almost nothing has been said upon this subject; so that no rules of any kind are laid down in our text-books to help us in distinguishing spinal from cerebral cases of functional paralysis.'

SYMPTOMS.—It seems clear, however, that if there are in reality cases of functional paralysis of *spinal type*, they at least could have no sort of right to be spoken of as 'hysterical,' seeing that hysteria is now generally admitted to be a functional disease of the brain. The writer believes that such cases are to be met with not infrequently, and for the present he is disposed to range them tentatively in two classes, viz.:—

A. *Cases of the Spastic Type, due to functional perversion or defect in some part of the pyramidal system of fibres in the spinal cord.*

B. *Cases of the Flaccid Type, due to functional defect in the anterior cornua in certain segments of the spinal cord.*

In both sets of cases there is mostly a complete absence of ordinary hysterical symptoms. Instances of Class A approximate somewhat in their symptoms to those of 'spastic spinal paralysis'; that is,

there is paresis or actual paralysis, with more or less of rigidity and exaggerated reflexes in the lower extremities. Defects of sensibility may or may not be present in the paralysed parts and over the trunk to a certain level, though the distribution of anæsthesia is often unequal on the two sides of the body.

In Class B we have to do with cases of paralysis of flaccid type unassociated, it may be, with any diminution of muscular sense. But in other of these cases, where the functional defect is more diffused over the grey matter rather than limited to the anterior cornual regions, there may be a more or less marked impairment of common modes of sensibility, and possibly also some amount of diminution of muscular sense owing to interference with its afferent channels. It may, perhaps, be assumed that the functional defect in the anterior cornua of the cord is adequate to bring about a more or less definite paralysis, even though the defect may not be sufficiently severe to entail any special muscular atrophy.

DIAGNOSIS.—The characteristics of hysteria and of hysterical paralysis have been set forth elsewhere (see HYSTERIA); it must suffice, therefore, to say here that true hysterical paralysis (while it may be hemiplegic or paraplegic) is very frequently associated with a hemianæsthesia of cerebral type (in which the special senses are more or less involved), while there may be the association of hysterical convulsions, and more or less special mental peculiarities. In the cases of functional paralysis of spinal type, however, with which we are now concerned, there is an absence of these associated symptoms; anæsthesia is not always present, and when it is it does not affect the head and face, with the special senses. Future investigation will probably lead to the recognition of other more definite differences between these two classes of functional paralysis.

In the diagnosis of these cases, reliance is always to be placed principally upon the absence of distinct evidence of organic disease such as would be furnished by muscular atrophy together with the electrical reaction of degeneration, by incontinence of urine or of feces, and by the occurrence of bed-sores. The diagnosis has, in fact, to be made by way of exclusion; we must be satisfied that the symptoms in the case before us are not explicable by supposing the existence of any known organic disease of the spinal cord.

It may be said, moreover, that the cases of organic disease which are most likely to be confounded with functional paralyses, whether of cerebral or of spinal type, are spastic spinal paralysis, disseminated sclerosis, subacute or chronic spinal paralysis, Friedreich's disease, or the paralysis associated with Pott's disease. In many cases, however, such mistakes are made as much from the want of a sufficiently thorough examination of the patient, as from defective knowledge of the respective characteristics of the organic diseases just named.

PROGNOSIS.—The prognosis is of course always very much more hopeful in functional cases than it would be in anything like corresponding cases due to organic disease. The ultimate establishment of a cure may be considered the rule in cases of functional paralysis, of this as well as of other types, provided the cases come under systematic and efficient treatment; though the duration of the

disease must be considered to be altogether uncertain. Sudden cures are not to be expected in this class of cases as frequently as they are in the functional paralyses of cerebral type. For the most part they will require many weeks or months of continuous treatment before the patient slowly recovers.

TREATMENT.—This must be conducted upon very much the same principles that are applicable to the treatment of functional paralysis of cerebral type, so that details need not be here recapitulated. See HYSTERIA.

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31. Paraplegia dependent on Idea.—

NATURE AND ÆTIOLOGY.—This is a form of paralysis, of purely 'functional' type, occasionally occurring in neurotic impressionable persons, and yet not dependent upon any ordinary hysterical condition. Attention was first called to such cases by Russell Reynolds, who cited among others a typical instance in which a young lady, while attending to a paraplegic father, amid the additional anxieties consequent upon straitened circumstances and the fatigues incident to teaching in order to obtain the bare necessities of life, became at last, under the influence of long-continued strain, together with an abiding fear (inspired by actual physical weakness) that she herself was becoming paralysed, reduced *de facto* to this condition, as the final outcome of a slowly increasing weakness.

PATHOLOGY.—Such a condition may occur quite independently of hysteria, and be just as free from anything like conscious simulation or desire to exaggerate. We cannot say positively that the state is induced by what is called 'inhibition,' or by definite vascular spasms such as are supposed to form one of the pathological bases of the class of so-called 'reflex' paralyses, and yet both these modifying influences over the functional activity of the spinal cord *may* be in part operative when imagination, continuously excited in some one direction, has a tendency to pervert the functional activity of this portion of the nervous system.

The same conditions that exist as more lasting states in these cases probably exist temporarily, under the influence of suggestion, in hypnotised persons. See MAGNETISM, ANIMAL.

SYMPTOMS.—There is a paralysis of motion in the lower extremities, more or less complete, often partial, and generally without implication of sensibility. There is unabated control over the bladder and rectum.

Reynolds pointed out that, while such patients may be wholly incapable, when bidden, of lifting a foot from the bed, they often find themselves able to turn or sit up without any assistance. And in slighter cases, though they may be unable to stand for a moment, such patients may yet be able to move the legs in any direction while in the recumbent position.

DIAGNOSIS.—The character of the paralysis, and its limitation in range, is thought to be of importance. But still more important is the establishment of the fact of the pre-existence of long-continued fears or fancies (in a person of delicate or neurotic temperament), of such a nature as to be in accordance with the patient's now-present condition, combined with the absence of all signs positively indicative of any structural defect in the spinal cord.

When such a condition exists (as it may) as a mere complication of an actually existing structural disease, the diagnosis becomes either impossible or extremely difficult. It is, in fact, only possible after prolonged observation and experience as to the course of the symptoms.

PROGNOSIS.—The prognosis is extremely good if the nature of the malady be divined, and a right course of treatment adopted. Under such circumstances, an almost complete cure may easily be brought about in a week or ten days; but, failing this recognition, the morbid condition may, it is said, under ordinary treatment, persist for an almost unlimited period.

TREATMENT.—The practitioner must inspire the patient with confidence that the malady is curable, and surround her (or him) with cheerful, hope-inspiring attendants and influences. At the same time, with the view of supporting her confidence (if for no other reason), the muscles of the apparently paralysed limbs should be faradised daily, or recourse should be had to frictions or massage combined with passive movements. He must make the patient attempt to stand or walk, with the necessary support; administer opiates, or bromide of potassium with chloral hydrate, to procure sleep, if necessary; and carefully seek to restore the patient's general health and nutrition. In this class of cases, especially, it would seem probable that the influence of 'suggestion,' if hypnotism could be induced, might be capable of producing an almost immediate cure. **H. CHARLTON BASTIAN.**

32. *Neurasthenia Spinalis.* — **SYNON.** : Functional Nervous Weakness of the Spinal Cord.

NATURE AND ÆTIOLOGY.—Under this name, descriptions have been given of a combination of symptoms not infrequently met with in males as well as females, but more especially in the latter. They are supposed to represent a condition of extreme nervous debility, coming on obscurely, or at all events not as a sequel of some previous severe illness or shock. Still the symptoms met with often approximate closely to those pertaining to a state of convalescence from some serious febrile illness; and are not at all unlike some of those which may follow concussion of the spinal cord.

Such symptoms, when occurring independently, are most prone to show themselves in those who are naturally of a neurotic temperament. They may be excited by over-fatigue of various kinds, especially when this has been coupled with disturbed sleep for some time. Prolonged exercise or mental overwork may have been the particular exciting cause of fatigue; though perhaps much more frequently this is to be found in sexual excesses (of a natural or unnatural order), either extending in the form of habitual indulgence over a considerable period, or as more isolated but marked excesses. At other times, symptoms of neurasthenia spinalis set in after influenza, or without obvious provocatives of any kind.

PATHOLOGY.—Concerning the actual cause of spinal neurasthenia little or nothing can be said. Sometimes there may be the co-existence of distinct cerebral symptoms of an analogous type; though on other occasions the symptoms are more purely spinal. This malady is perhaps capable of being induced by mere altered molecular states and actions of the tissue-elements of the spinal cord. A kind of

persistent condition of fatigue exists. Although some may imagine the existence in these cases of a more than usually anæmic condition of the spinal cord, of this, as a fact, there is no evidence. To speculate upon other modes in which such a set of symptoms might be brought about would, in the present state of our knowledge, be of little service. There is, however, the possibility that this morbid condition may be due in the main to a functional disease of the cerebellum—especially if the views of Rolando and others, as to the functions of this great organ, should prove correct even in part.

SYMPTOMS.—A feeling of utter weakness and prostration, induced by even the smallest amount of muscular exertion, is the central symptom, though this is usually associated with coldness and more or less numbness of the extremities. The knee-jerks are commonly much exaggerated, and when the tendons are struck there is generally more or less starting in other parts of the body. Pains, too, may be felt in the muscles of the limbs and in some parts of the back, though there is commonly no tenderness over any part of the spine. These symptoms may be unusually distinct after any activity of the genital function, and they may then be associated with extreme wakefulness, or sometimes with protracted inability to sleep. Occasionally, and especially when this latter symptom is not present, the patients may present a florid and fairly healthy appearance, strangely at variance with the extreme debility complained of.

DIALOGUS.—The points of greatest importance are the existence of extreme weakness, with no evidence of anything like actual paralysis, or indeed of any symptoms which would indicate an actual structural disease of the spinal cord. This being so, and diabetes being also eliminated, we may oftentimes (and especially where the existence of one or other of the above-mentioned exciting causes has been established) pretty confidently conclude that we have to deal with what is here named '*neurasthenia spinalis*.'

PROGNOSIS.—A relief of this condition is ultimately to be looked for under the influence of rest and suitable treatment; but in regard to the rapidity with which any such amelioration of the patient's symptoms is to be brought about, great differences exist in different cases. Weeks, months, or even years may be required before a natural amount of vigour is restored.

TREATMENT.—Rest, especially in the direction of previous excesses, is the first and indispensable requisite. Every effort should be used to obtain regular and sound sleep. The action of these potent restoratives should be supplemented by a generous and easily assimilable diet, together with a moderate amount of stimulants. Hypophosphites of the alkalis with iron and small doses of strychnine (which may be conveniently given in the form of a syrup) often prove decidedly beneficial. An abundance of fresh air is desirable, and especially that of elevated and bracing situations. Daily frictions and massage, aided by stimulating saline baths, may also prove to be of much use; while in the more severe cases a complete course of Weir-Mitchell treatment is indicated (p. 1073).

H. CHARLTON BASTIAN.

33. *Toxic Spinal Paralysis.*—Under this name it will be right to refer to a class of cases of paraplegia produced by poisons of various kinds. It

constitutes a somewhat heterogeneous group, concerning which our knowledge is still very defective.

Of the toxic agents taken into the body, and capable of entailing a paraplegia, some are minerals such as arsenic and lead; others are of vegetable origin, such as aconitine, conine, veratrine, prussic acid, ergot, and alcohol; while others again are of animal origin. In the majority of cases, their action as 'causes' is not sufficiently potent to lead to paralysis as anything like an invariable effect. They need the concurrence of other favouring circumstances, probably in the main intrinsic; but under the combination of conditions thus resulting a paraplegia may be induced. It is only in this attenuated sense that the above-mentioned poisons are to be regarded as 'causes' of paraplegia. They ought perhaps, from this point of view, to be considered as predisposing rather than as exciting, and in no case as proximate, causes of paraplegia.

Moreover, investigations that have been made during the last few years have shown that these various poisons act more powerfully upon the peripheral nerves than upon the spinal cord itself. So that cases of paralysis due to lead, arsenic, or alcohol, may be in the main occasioned by changes in the peripheral nerves (*see* NEURITIS, MULTIPLE), although some changes in the spinal cord may also co-exist, which vary in their nature in different cases. They mostly take the form of atrophy of the ganglion-cells in the anterior cornua, with some amount of surrounding sclerosis, though occasionally minute foci of softening may be met with.

Besides the more specific effects of lead in producing the above-mentioned changes in the peripheral nerves and spinal cord, it commonly induces a condition of lowered vitality which favours the development of degenerative conditions in the spinal cord. In such cases it can generally only be considered as one among other determining conditions, tending to bring about some form of paralysis. In this way it has seemed to the writer to be occasionally one of the concurrent causes in the production of primary lateral sclerosis and of progressive muscular atrophy.

These considerations will help to account for the fitful and irregular manner in which arsenic, lead, or alcohol (and probably to a similar extent other toxic substances) give rise to *paraplegic* symptoms in those who have taken them to excess. Thus, according to Tanquerel des Planches, out of 200 cases of lead-poisoning, in only fifteen did the paralysis implicate the lower extremities; and in only one of these did it occur as a distinct paraplegia. This case might, therefore, have been a coincidence rather than a definite result of the taking of lead. Again, in regard to arsenic, it is true that in certain cases Orfila observed paraplegic conditions in dogs which had taken large quantities of this drug; but such symptoms dependent upon actual disease of the spinal cord would seem to be met with only occasionally as a result of acute arsenical poisoning in man, and with equal rarity in those who habitually consume large quantities of this substance. On the other hand, the recent epidemic of multiple peripheral neuritis in the North of England, caused by the consumption of beer contaminated with arsenic, has abundantly demonstrated its capacity for the production of paralysis by causing neuritis—especially when taken in association with alcohol. *See* pp. 1077, 1078, and 1109.

The notion was advanced by Moxon that a certain class of poisons, which own the common property of being 'depressants of the circulation,' have a tendency to paralyse the hind-legs rather than the fore-legs of animals. In this group are included aconitine, conine, and possibly also veratrine, chloral hydrate, and prussic acid. He was of opinion that these drugs act by causing further impediments 'to the exceedingly and peculiarly difficult blood-supply of the caudal end of the spinal cord.' It should be borne in mind that extreme feebleness of blood-current is of itself a common cause predisposing to the occurrence of thrombosis both in arteries and in veins, and that such a condition may intervene in some of these cases of poisoning, and lead to the development of paraplegia. This would enable us to account for the otherwise inexplicable fact of the maintenance of the paralysis long after other effects of the poison have passed away.

PROGNOSIS AND TREATMENT.—The prognosis in this class of cases is generally good. Often, indeed, the mere cessation from the consumption of the poison, aided by favourable conditions for the maintenance of the general health, suffices to restore the patient to health. In the more severe cases, however, the treatment has to be prolonged, and we must avail ourselves of all the local measures indicated in the article NEURITIS, MULTIPLE, in order to bring about a very gradual restoration of the patient's strength.

H. CHARLTON BASTIAN.

SPINE, Disease and Curvatures of.—

SYNON.: Fr. *Maladies et Courbures du Rachis*; Ger. *Krankheiten und Krümmungen des Rückgrates*.

The structures composing the vertebral column are subject to disease such as is met with in other parts of the body where similar structures exist. In this article will be described tubercular caries, by far the most common affection of the spine, syphilitic disease, acute osteo-mylitis, tumours of the bones, and lateral anterior and posterior curvature. Angular curvature, which is merely a symptom of tuberculous, or more rarely of syphilitic or malignant destruction, of the bodies of the vertebrae, is described under TUBERCULAR CARIES.

Caries of the Spine.—SYNON.: Pott's Disease; Fr. *Mal vertébral de Pott*; Ger. *Die Pottische Krankheit*.

DEFINITION.—A destructive disease of one or more of the bodies of the vertebrae and their intervertebral cartilages, frequently leading to angular deformity of the spine in consequence of the bodies of the vertebrae above and below the seat of the disease falling together.

ÆTIOLOGY.—Spinal caries is nearly always of tubercular origin. It is occasionally due to acute osteomyelitis or the breaking down of a syphilitic gumma. The disease may occur at any age, but is most common in childhood. It may be confined to one vertebra or may spread to the neighbouring vertebrae, or several vertebrae may be simultaneously affected. The onset can frequently be traced to a slight strain or other injury of the vertebral column. The tubercle-bacillus, as in tuberculosis of other bones, commonly affects the actively growing and vascular bone beneath the epiphysal discs or, in the case of adults, the deeper or osteogenetic layers of the periosteum covering the anterior portion of the

vertebral bodies. In rare instances the periosteum of the posterior part of the bodies is affected. As the result of the bacillary invasion, granulation-tissue is formed and the bodies of the affected vertebrae and the neighbouring intervertebral discs are gradually destroyed in the way described under tuberculosis of bone (p. 168). At this stage the disease may cease and the granulation-tissue undergo ossification, causing ankylosis of adjacent vertebrae without any angular deformity. More commonly, however, the granulation-tissue, having destroyed the osseous trabeculae, caseates and breaks down, forming a tubercular abscess. If the process has been very acute a large portion of the cancellous tissue may die *en masse*, forming sequestra (*caries necrotica*). In any of these cases angular deformity may be the result, since, partly by its own weight and partly by the dragging of the abdominal muscles, the upper portion of the spine thus undermined falls forward at the seat of the disease, and forms an angle with the lower portion. In consequence of the patient's efforts to hold himself upright the normal lumbar and cervical curves, when the disease occurs in the dorsal region—the common situation—will be greatly increased, the angular projection being thus thrown backwards and the well-known 'hump-back' produced. Should an abscess have formed, its contents may undergo changes similar to those occurring in other regions (p. 9). Thus it may dry up leaving a caseous mass, or it may extend down the spine on one or both sides of the anterior common ligament, or more rarely make its way into the spinal canal. In the cervical region the abscess usually points at the back of the pharynx or at the side of the neck; in the lower dorsal and lumbar regions it either makes its way posteriorly between the last rib and crest of the ilium as a lumbar abscess, or it follows the course of the psoas muscle, forming an iliac or psoas abscess (see PHARYNX; and PSOAS ABSCESS). Occasionally and especially in the upper dorsal region it may take a circuitous course, for example, tracking along a rib towards the sternum or between the abdominal muscles, when it may open at the umbilicus, or may enter the pelvis, pass through the great sciatic notch, and point in the gluteal region, or may track down by the side of the rectum, forming an ischio-rectal abscess. When the disease begins or extends posteriorly, a tuberculous collection may form beneath the posterior common ligament; the inflammation may then involve the dura mater, setting up a pachymeningitis; in either instance a temporary or permanent paralysis may ensue from pressure on the cord. The anterior motor column being nearer to the seat of the disease, paralysis of motion is much more common than that of sensation. In rare instances the cord itself may become involved in the inflammation and undergo softening leading to permanent paraplegia.

SYMPTOMS.—In the earlier stages of spinal caries the symptoms are often obscure and an absolute diagnosis may be impossible. By the time an angular projection has formed, the disease is obvious, but to wait for this is to nullify the advantage of an early diagnosis, namely, the prevention, by judicious treatment, of any deformity. Pain on movement and pain on pressure over the spines of the vertebrae, although not conclusive signs, are of material value when taken in conjunction with rigidity of certain portions of the spine due to muscular contraction calculated to prevent movement

and consequently pain at the diseased spot. The presence of *pain* may be detected by percussing or pressing on the head, on the shoulders, on the head of the rib or over the diseased spot, or it may be elicited by certain movements of the body. It may be felt at the diseased spot or may be referred along the course of the spinal nerves to various regions according to the situation of the disease. Pain on motion may be elicited by asking the patient to stoop, hyper-extend the spine, rotate and bend the body suddenly from side to side, or to rise on tiptoe and suddenly come down on the heels. If a patient can jump without pain from a chair or low stool he is almost certainly free from caries of the spine. For the detection of *rigidity* the patient may be put through certain movements, or the apices of the spinous processes may be dotted with an aniline pencil while he is in the prone position, and a note taken whether these marks are approximated when he stands upright. Rigidity is an early sign in the cervical region, moderately early in the dorsal region, and late in the lumbar.

In the *upper cervical region* a constrained and fixed position of the head always exists in the early stages of the disease, and the patient, finding a difficulty in keeping the head erect, acquires a habit of supporting the chin upon the hands with the elbows resting on a table or chair. The neck is held rigidly. The patient, if asked to look round, turns his whole body rather than his neck. When the occipito atlantal joint is affected, the rigidity may be detected by the inability to nod the head; when the atlanto-axial, to turn the head from side to side; and when in the next succeeding vertebrae, to hyper-extend the neck. The pain is increased by bending the neck forwards and rolling the head from side to side, but is relieved by supporting the chin with the hand. The pain radiates along the course of the sub-occipital, great occipital, and great auricular and descending branches of the cervical nerves, giving rise to occipital headache and pain in the lower part of the neck and upper part of the chest.

In the *lower cervical and upper dorsal region* there are no very distinctive symptoms, but pain or tingling may be felt in the shoulders, arms, and even fingers, being reflected along the nerves given off from the brachial plexus. In children there is sometimes a troublesome cough, which has been attributed to reflex irritation of the recurrent laryngeal nerve. Some rigidity of the muscles in the posterior triangle may be detected.

In the *mid-dorsal region* the absence of symptoms in the early stages is most marked owing to the comparative immobility of this portion of the spine and the absence of any muscular attachments of the bodies of the vertebrae. Local pain and pain on percussion are sometimes present. The pain may be felt along the course of the intercostal nerves or over the sternum. The patient moves about slowly and cautiously, and generally sits with his arms extended and his hands resting on a chair to relieve the dorsal vertebrae of the superincumbent weight and to assist breathing; or walks about supporting the spine by placing his hands on various portions of furniture. If a child, he soon gets tired of play and lies about on the floor.

In the *lower dorsal and upper lumbar region* there may be pain in performing the various movements in which the psoas muscle is brought into play, such as stooping in drawing on stockings or

lacing boots, lifting even a light weight from the ground, going up and down stairs, rising suddenly from the horizontal to the sitting or standing position, and in turning suddenly from the back on to the abdomen while lying down. If asked to pick up anything from the floor the child does not bend his back; he places his hand upon his knee to support the spine and reaches the ground by bending his legs while holding his back straight. If asked to turn round he rotates the whole body, not the back. The pain may be transmitted along the lower intercostal nerves to the umbilicus, and the child complain of stomach-ache (pain on both sides of the abdomen is most characteristic of spinal caries), or it may be transmitted along the course of some of the nerves coming off from the lumbar enlargement to the front of the thighs or even to the knee or foot by the long saphenous nerve.

DIAGNOSIS.—In the early stages caries must be diagnosed from rheumatism, neuralgia, and lumbago. The history of former attacks of a like nature, and a careful consideration of the signs above given, will usually serve to distinguish them. Hysteria may simulate caries, but the absence of rigidity, the complaint of pain at different spots at different times, the effect of pressure in producing squirming movements instead of rigidity, the presence of other signs of hysteria, the evident absence of suffering on watching the face at the time that much pain is said to be felt, are the points to be attended to. Careful percussion and auscultation of the chest and abdomen will exclude aneurysm. From a gumma or malignant disease leading to breaking down of the vertebral bodies, tubercular caries cannot at first be diagnosed, as all three give rise to the same symptoms, but the presence of other signs of syphilis, congenital or acquired, will point to a gummatous origin. The age of the patient, the history or presence of a primary carcinoma or sarcoma in other regions will raise a suspicion of malignant disease; while rapidly increasing pain and paralysis, in spite of rest, wasting, and later, perhaps, evident swelling at the seat of the disease will soon clear away the difficulty. *See also* PSOAS ABSCESS.

In the later stages, when an angular projection of one or more of the spinous processes has taken place, the diagnosis as a rule is evident. It should not be forgotten, however, that the disease may run its course, even to the production of paralysis or the formation of an abscess without any angular projection. Conversely the normal prominence of the spinous processes of the seventh cervical, first dorsal, eighth and ninth dorsal, and lower dorsal and upper lumbar vertebræ may be exaggerated. The projection, when occurring in the lower cervical and upper dorsal vertebræ, may be due to a natural conformation and family peculiarity, as we see in some short-necked and round-shouldered persons; it is sometimes accompanied by local pain on pressure or percussion, the pain extending along the shoulders and down the arms, thus simulating to some extent cervical caries. A projection of the lower dorsal and upper lumbar vertebræ is not uncommon in girls, and when combined with back-ache, muscular debility, and neuro-mimesis may be mistaken for spinal caries, especially when through the rubbing of the clothes a callosity of the skin or an inflamed and tender bursa has formed over the prominent spine. In such cases, however, the tenderness and swelling will be quite superficial and

the spine mobile. In rickets, again, the kyphotic curve may be distinguished from the angular projection of caries by the projection being more uniform and involving several vertebræ instead of being sharp and angular, and localised to one vertebra, as is usually the case in caries. The kyphotic curve of rickets, moreover, disappears more or less completely when the child is held up by its arms or extended across the nurse's knees in the prone position; and he does not resist or cry on making these manipulations. In caries, on the other hand, the projection does not disappear, and the lumbar spine if affected will become rigid on pulling the legs, the manipulation evidently giving pain. In the rare cases in which the spinal caries depends upon a gumma in the vertebræ there will be other evidence of syphilis, congenital or acquired, which will set the diagnosis at rest.

COURSE, DURATION, AND TERMINATIONS.—Like other tuberculososes, tubercular caries of the spine usually runs an insidious course. Within a period of from six to nine months the angular curvature is generally produced. If the case proceeds favourably without external abscess or paralysis, sound bony ankylosis takes place in about three years. When abscess or paralysis occurs the period of recumbency has frequently to be prolonged to five or more years. Paralysis under complete recumbency usually passes off in about two years. Should death occur, it may be due to septic absorption, and hectic or lardaceous disease from a neglected spinal abscess; to paralysis due to pachymeningitis and softening of the cord; or to dissemination of the tubercle to the lungs, brain, or peritoneum.

If the case is neglected, especially when the disease is in the cervical or upper dorsal region, some weakness of the lower limbs is noticed with increase of the knee-jerk and possibly ankle-clonus; while, later, complete loss of motion may ensue. Sensation, however, is rarely affected, nor is the bladder or rectum usually involved.

PROGNOSIS.—When the disease has been diagnosed early, and the patient can be placed absolutely recumbent under the best hygienic conditions, perfect recovery may be hoped for without any angular deformity, paralysis, or abscess. When angular deformity has already occurred a good recovery may still be obtained under judicious treatment, but the angular projection will be permanent unless the risky process of forcible or mechanical straightening is undertaken. The same with regard to paralysis—this will usually completely disappear after a long period of absolute rest. For the prognosis when abscess has occurred *see* PSOAS ABSCESS.

TREATMENT.—The *local treatment* in the earlier stages is directed towards keeping the diseased vertebræ at rest, and in removing the superincumbent weight of the spine from the diseased spot. These objects are best attained by absolute recumbency combined in some cases with fixation of the spine by some form of mechanical contrivance, or in other cases by extension and counter-extension of the spine. The patient may be in the supine or in the prone position, either on a firm mattress or on an ordinary bedstead with a low pillow, or on a special spinal couch. The supine position is, as a rule, the more comfortable. He should on no account be allowed to raise himself. In the case of a child it may be necessary to apply a strap across the

chest and around the arms. For the fixation of the spine during the period of recumbency the writer has largely used, especially in children, what is known as a double Thomas's splint. The splint is placed next the skin so as not to require removal while the child is washed and dressed. It not only fixes the spine and takes off the weight of the head and upper limbs, but also fixes the lower limbs, and thus prevents the psoas muscles from dragging on the lumbar vertebrae. In this apparatus the patient can be carried about, and as the acute stage passes off, the upper end can be gradually lifted up so that the patient can lie in a slanting position and see what is going on around him.

When the disease is in the upper cervical region during the period of recumbency, which is here absolutely essential in the acute stages, no pillow whatever should be employed; the head should be immovably fixed by sandbags, and should never be raised for feeding or any other purpose, to avoid the risk of the spinal ligaments giving way, and of the upper vertebrae slipping forward on the lower and so compressing the spinal cord and possibly causing paralysis or even sudden death. In place of sand-bags the head may be fixed to the double Thomas's splint by suitably adjusted straps, or a cervical collar may be applied. In some cases of disease of the cervical and upper dorsal vertebrae extension of the head may be employed in conjunction with absolute recumbency, the extension being applied by means of a weight and pulley fixed to the end of the bedstead or spinal couch. Absolute recumbency when properly carried out offers the best prospect of preventing serious angular deformity and paralysis or abscess-formation. Roughly speaking, this position should be maintained for six to twelve months. When the acute symptoms have subsided, a spinal support or poroplastic felt jacket or plaster-of-Paris case should be applied till the vertebrae have become consolidated, the patient in the meantime being allowed to take a gradually increasing amount of exercise, though at first being kept recumbent (with the apparatus on) the greater part of the day. Very numerous varieties of spinal supports have been invented. Of these the best, perhaps, consists of a pelvic plate which fits over the sacrum and neighbouring parts of the iliac crests, and two upright steels moulded to the shape of the back on each side of the spine, and ending above in a scapula-plate, from which straps are carried under the axilla and over the shoulders back again to the plate. This form of apparatus possesses the advantage that the chest is left free, and the movements of respiration are not confined. It is somewhat heavy, however, and a little difficult to keep properly adjusted. The poroplastic felt jacket is made of porous felt impregnated with certain gums. The jacket is made to measure, and when about to be applied is put in a steam oven to soften the gums. While hot it is moulded to the figure. On cooling, it retains its shape. During its application the patient is gently suspended by a pulley-apparatus fixed by suitable straps to the head and under the axilla, the heels barely touching the floor.

A plaster-of-Paris case is applied either with the patient suspended or in the recumbent position in a hammock-apparatus. A skin-fitting vest is first put on and marked with a pencil at the line of the axilla to indicate the upper limit of the jacket. Plaster-of-Paris bandages are next wound round

and round the trunk till a sufficient thickness is obtained, perforated strips of tin being inserted vertically between the layers of the bandage to further strengthen it. When finished the case should reach from just below the pencil line to the crest of the ilium, but should not reach to the great trochanter and pubes. A folded silk handkerchief is placed over the abdomen during the application, and removed afterwards so as to ensure a space for abdominal respiration. When the plaster case is dry it may be sawn down the front, carefully stretched open and removed, and the fronts edged with leather and perforated with eyelet holes, so that it can be worn laced up and taken off from time to time for purposes of cleanliness.

During the last few years forcible straightening of the spine when angular curvature has occurred has been reintroduced by Calot, and has been extensively practised. Extension is made on the arms and legs, and direct pressure at the same time applied to the prominent vertebrae; a plaster of Paris or poroplastic jacket is then applied. The effect of this treatment is, of course, to tear asunder the approximated vertebrae, leaving a gap which can at the best be only very slowly filled up. The method is not unattended by risk, abscess, paralysis and even death having followed its use. There is always the further chance of a relapse from the vertebrae again falling together as soon as the patient gets about. In place of forcible extension, Taylor of Baltimore recommends mechanical extension of the spine by means of an instrument which he calls a kyphotone, and claims that in this way in suitable cases the hump can be made to disappear without the risks attending forcible extension.

Where paralysis has occurred in consequence of compression of the cord by inflammatory effusions or tuberculous collections, laminectomy may in some instances be successful in removing the source of pressure, but as a rule the paralysis passes off if the patient is kept perfectly rigid and recumbent for a sufficiently long period. The indications for laminectomy may be said roughly to be persistent paralysis in spite of recumbency and fixation of the spine, impairment of sensation as well as of motion, contractures or rigidity of the muscles of the lower limb, implication of the bladder or rectum, intractable cystitis, secondary chest-troubles, and severe pain. The operation should not be undertaken when there is evidence of general tuberculosis or extensive spinal meningitis.

It may sometimes be possible to evacuate tuberculous collections or remove a sequestrum by working towards the front of the vertebrae through a suitable incision. Thus in the neck the disease may be reached by an incision made along the line of the sterno-mastoid; in the dorsal region the affected vertebrae may be exposed by resecting the necks of several ribs and working backwards behind the pleura; in the lumbar region the incision is made vertically over the transverse processes, and the dissection carried down between the lumbar muscles towards the bodies of the vertebrae.

Acute osteo-myelitis of the spine is an acute infective inflammation attacking any portion of the vertebrae.

For a general account of this affection see *OSTEO-MYELITIS*, p. 166. All that need be said here is that suppuration always takes place. The pus may make its way forwards into the pharynx (retro-

pharyngeal abscess), thorax or abdomen (mediastinal and psoas abscess), backwards into the cervical, dorsal or lumbar region, or into the vertebral canal, giving rise to inflammation of the membranes and cord. The general symptoms are those of osteomyelitis elsewhere. The local symptoms are pain, tenderness, and later abscess-formation about the affected vertebrae. The sudden onset and acute course distinguish it from tubercular caries. The treatment consists in recumbency and fomentations to relieve pain. It is apt to terminate in septicæmia and pyæmia. Hence, as soon as there is local evidence of pus-formation free incisions should be made.

Osteo-arthritis.—**SYNON.**: Rheumatoid arthritis.

PATHOLOGY.—Osteo-arthritis may be met with in the spine after middle life, and, as in other structures, ultimately leads to absorption of the intervertebral cartilages, the production of osteophytes upon the vertebral bodies, ossification of the ligaments, and destruction of the interarticular joints, thus leading to complete bony ankylosis of the spine.

SYMPTOMS.—The patient has a stooping gait owing to the backward curvature, and kyphosis of the spine, which is rigid and immovable; and complains of pain in the back, which is worse at night and in damp weather and moist climates. Should the articulations of the ribs with the vertebrae become affected, the movements of the chest are impeded, the breathing becomes abdominal, and pulmonary symptoms supervene. The treatment is that for osteo-arthritis in other situations.

W. J. WALSHAM.

SPINE, Curvatures of.—Four forms of curvature of the spine are described: (1) lateral curvature or scoliosis; (2) posterior curvature or kyphosis; (3) anterior curvature or lordosis; and (4) angular curvature or spondylitis, which is a sequel of tuberculosis, or more rarely of syphilis or malignant disease.

In the normal spine the thoracic or dorsal curve, which has its convexity backwards to increase the capacity of the chest, depends upon the shape of the bodies of the vertebrae, while the lumbar and cervical curves, which are convex forwards to compensate for the backward thoracic curve, depend upon the shape of the intervertebral cartilages. Lateral curvature is due primarily to one-sided compression of the intervertebral cartilages; kyphotic and lordotic curves primarily to compression of the posterior or anterior part respectively of the intervertebral cartilages; and angular curvature to various destruction of the bodies of the vertebrae whereby the vertebrae above fall forwards on to the vertebrae below and the projecting spinous processes produce the angular projection.

1. Lateral Curvature of the Spine.
SYNON.: Scoliosis; Fr. *Scoliose*; Ger. *Seitliche Rückgratsverkrümmung*.

DEFINITION.—A deformity in which the bodies of the vertebrae deviate laterally, and at the same time undergo rotation on their vertical axes.

ÆTIOLOGY.—The causes of lateral curvature are local and constitutional; these are generally combined. The local cause of lateral curvature may be said to be any mechanical condition that induces a long-continued unequal compression of the intervertebral cartilages and bodies of the

vertebrae. In the majority of cases these mechanical conditions are associated with sedentary habits, rickets, muscular debility, relaxation of the ligaments, and a softened state of the bones, the spine being thus rendered less resistant to injurious mechanical pressure. Indeed the constitutional conditions may be the chief factors, and it may be difficult to trace the deformity to any special mechanical cause. When there is no evidence of any muscular weakness, the curvature is generally confined to the lumbar region and dependent upon obliquity of the pelvis; in these cases it rarely assumes the grave proportions met with where debilitating influences are important factors. Lateral curvature is more common in girls than in boys, and frequently commences during a period of rapid growth or of convalescence from some illness. It can frequently be traced through two or three generations, and may occur in several members of the same family. Very rarely it is congenital and dependent on malformation of the bodies of the vertebrae.

The mechanical causes may be classed as follows: (1) Permanent or habitual obliquity of the pelvis due, for example, to inequality of the length of the legs, knock-knee, flat-foot, congenital dislocation of the hip, standing on one leg, or sitting cross-legged; (2) A one-sided position of the body such as was assumed in the old-fashioned method of writing, or when sitting for long periods at low flat-topped desks and on stools without backs; or such as is entailed by employments necessitating the carrying of heavy weights in one hand, or the use of one arm, as in book-folding, feather-curling, ironing, violin-playing, and carrying heavy children; (3) Contraction of the sternomastoid in congenital wry-neck; (4) Unilateral paralysis of the spinal muscles due to poliomyelitis.

ANATOMICAL CHARACTERS.—As the result of the long-continued unequal compression the cartilages, and later to a less degree the bones, become wedge-shaped and the spine consequently assumes a lateral curve. While, however, a curve is thus being produced, say, in the dorsal region with its convexity to the right, a secondary compensating curve is also being produced in the lumbar region, and to a less extent in the cervical, with the convexity to the left, in order to maintain the equilibrium of the body. Coincidentally with these changes the vertebrae undergo rotation on their vertical axes, so that while the anterior surface of the body is directed towards the convexity of the curve the spinous processes are directed towards the concavity, the transverse process on the convex side being carried backwards, and the transverse process on the concave side forwards. Hence in addition to the lateral deviation forming the so-called primary and secondary or compensating curves there is in the later stages a general rotation of the vertebrae upon themselves, giving the spine a spirally-twisted appearance. In severe cases this rotation may extend to a quarter of a circle in the centre of the curve but diminishes from this point to the two extremities. The amount of rotation of the bodies of the vertebrae does not always accord with the amount of lateral deviation of the spinous processes; indeed there is always much greater internal rotation of the bodies than is apparent from the deviation of the spinous processes externally. In the dorsal region the ribs necessarily follow the transverse processes;

hence in a dorsal curvature with the convexity to the right the ribs on the convex side are carried backwards, thus forming at first a slight projection and later a distinct hump, while the ribs on the concave side are carried forwards, producing a prominence of the corresponding breast. As the result the sternum becomes very oblique, and its lower extremity projects.

SYMPTOMS.—Back-ache, feeling of weakness in the lower part of the spine, malaise, anæmia, inability to hold the spine erect, or a stooping gait, may occasionally draw attention to the condition, but in many cases patients suffer no inconvenience whatever, and attention is first called to the spine by the dressmaker or mother who notices that the shoulder or hip is growing out. In the early stages there may be no lateral deviation of the apices of the spinous processes, although considerable rotation of the vertebræ has occurred; while conversely in quickly-growing girls with muscular debility and ligamentous relaxation there may be marked deviation without rotation, and the slight curvature that exists can be made to disappear completely on suspension or the prone position. These, however, may be regarded as cases of weak spine or incipient curvature rather than as cases of true lateral curvature. To detect true lateral curvature, evidence of rotation should be sought. The back should be fully exposed as low as the iliac crest, and in the case of children all the clothes should be removed. The patient without shoes, with the heels together, the knees straight and the hands raised above the head as if about to dive, should bend the body forwards till the fingers approach or touch the toes. If rotation has occurred it may then be detected by a slight backward projection of the ribs in the dorsal region or prominence of the erector spinæ in the lumbar region; whereas in cases of merely weak spine the lateral deviation of the apices of the spinous processes will have disappeared, and the back be symmetrical. Some want of symmetry due to very slight rotation may also be detected by placing the patient in a sitting posture and then looking down the back from above.

In severer cases the signs are unmistakable. In the more common forms a well-marked curve with its convexity to the right is seen in the dorsal region, and a slighter and shorter curve with its convexity to the left in the lumbar region. The right shoulder is generally elevated, and the angle of the right scapula is prominent owing to the backward rotation of the ribs which follow their vertebræ on the convexity of the curve. There is also apparent prominence of the right iliac crest owing to the muscles being carried away from it by the forward rotation of the lumbar transverse process; while the lumbar muscles on the left side form a prominent ridge over the backwardly rotated lumbar transverse processes on the convexity of the lumbar curve. In other cases the compensating curves may be so slight that there is apparently a long single curve only with its convexity either to the right or to the left; while in other cases again, instead of the dorsal curve having its convexity to the right and the lumbar its convexity to the left, the opposite condition may exist.

DIAGNOSIS.—Lateral curvature may have to be diagnosed from hysteria and from the lateral deviation which sometimes occurs in spinal caries. In hysteria, although the curve may appear severe, if

the patient is made to assume the diving posture and bend the body forwards, as the fingers approach the feet it will gradually disappear. An hysterical curve will also disappear during sleep and anæsthesia. In the lateral deviation that may occur in spinal caries there is generally some angular projection as well, and rigidity of the spine at the seat of disease. Pain may also be present in the course of the nerves leaving the intervertebral foramina of the affected vertebræ; or may be elicited on applying the tests for pain and rigidity described under spinal caries (p. 1566).

PROGNOSIS.—In the early stages where there is no rotation, a complete cure may be obtained, but when rotation has once occurred, although the general appearance of the figure may be improved by strengthening the muscles that hold the spine erect, the pain relieved, and the curve prevented from getting worse, the removal of the rotation, and consequent complete cure of the curve, is impossible. In those rare cases depending upon spinal paralysis or severe contraction of the chest following empyema, little or nothing can be done to improve the deformity.

TREATMENT.—This may be discussed under the heads of (1) Removal of the cause; (2) Posture; (3) Exercises and massage; (4) Rest; (5) Reduction of rigidity; and (6) Mechanical supports.

1. *Removal of the cause.*—In the majority of cases this will consist in the correction of faulty habits in sitting and standing, and in improving the general health. In other cases, where the curvature depends upon some structural defect, it is necessary that this should, if possible, be removed or corrected. Again, when the curvature depends on wry-neck, the sterno-mastoid should be divided; when upon rickets, constitutional remedies for this disease should be employed. The avoidance of all faulty positions in sitting and writing is most important. Occupations necessitating stooping should be given up. The patient should be forbidden to stand on one leg or sit cross-legged. While in class he should stand at attention. The one-sided position with the spine twisted and the arm on the table assumed in the old sloping method of writing should be given up, and vertical or back-hand writing taught instead, the patient sitting upright and square the while. He should not be permitted to sit at a flat-topped desk on a stool or bench without a back. The height of the desk should be capable of being regulated and the slope adjusted to the proper angle according as the patient is writing (15° to 20°) or reading (40°). The stool should have a high slightly sloping back, so moulded as to correspond to the normal outline of the body. It should be provided with a foot-rest fixed at a proper angle for the feet. A similar stool should be used at meals and during music-lessons. Older patients should use a properly shaped reclining chair or couch with a movable back, and while reading should not be allowed to hold the book with one hand, but have it supported at a proper angle on a book-rest. To prevent stooping while reading short-sight should be corrected by glasses, and all articles of dress should be quite loose across the chest so as to allow of the shoulders being freely thrown back. The effects of the one-sided position of women during horse-exercise should be neutralised by riding alternately on the off and near side, while young girls should ride boy-fashion. Long violin-practice should be forbidden; and such games as tennis should be

played with the right and left hand alternately. Whenever necessary the general health should be improved by abundant outdoor exercise, games of all kinds—tennis, hockey, cricket, skipping, sculling (not rowing), swimming, and horse-exercise, with the reservations stated above. A cold or tepid bath should be taken every morning. Exercise, however, should always stop short of fatigue; and the patient, if weakly or growing rapidly, should, after taking exercise, lie absolutely flat on the back for half an hour to an hour. Early hours and country-life or residence by the sea are, of course, desirable. Tonics, and in some cases cod-liver oil and arsenic, are generally indicated, while a prolonged course of some mild astringent preparation of iron is frequently most beneficial.

2. *Posture*.—The constant assumption of a good posture is imperative. At first the patient should be placed in the posture in which the curve is obliterated, or is least noticeable. It is generally necessary to so arrange two mirrors that the patient may see her own back. At first the correct posture can only be maintained for a few minutes, but as the muscular tone is gradually improved it will become habitual.

3. *Exercises and massage*.—Exercises for strengthening the muscular system in general and the spinal muscles in particular should be employed. For the first purpose, drilling, dancing, and the ordinary gymnastic exercises are all of service, as are also the exercises performed with the Sandow's and Whiteley's apparatus. The patient may beneficially make use of the latter for five to ten minutes daily after the course of special exercises has been completed. The special exercises should be carried out by a properly trained nurse under the occasional supervision of the surgeon. Put broadly, the exercises employed are modifications more or less of the so-called Swedish and Danish systems. The objects aimed at are not only the general improvement of the spinal muscles, but the special strengthening of such of the spinal and other muscles as tend to hold the spine in the corrected position (*see* MASSAGE). The resistance exerted by the exerciser is better than that of any weight or elastic tension, since it is more sympathetic and can be better regulated to the strength of the patient, and more readily be brought to bear on the muscles it is desired to improve. Moreover, the properly trained exerciser, sees that the right movements are executed, whereas the patient, if left to the use of mere weights and cords or in the hands of an unskilful exerciser, may bring into play such movements as increase rather than improve the curvature. Special attention during the course should be given to those exercises which tend to increase the respiratory capacity of the chest. The exercises should be employed once, or, in some cases, twice daily, and be continued from three to six months. Each sitting should occupy about three-quarters of an hour. After the exercises general massage of the muscles of the back is frequently very beneficial. There are now a number of efficient nurses, who have been well trained in properly carrying out these exercises, but the occasional supervision of the surgeon is desirable. Early cases can be completely cured if the treatment is thoroughly carried out, and especially if the patient can be made to take an intelligent interest in her own cure. Even when there is considerable rotation patients will still be improved both in their bodily health and general appearance,

while the asymmetry of the back may be easily concealed by the dressmaker.

4. *Rest*.—The prolonged rest formerly prescribed for lateral curvature is objectionable from two points of view. Firstly it produces wasting and atrophy of the muscles of the back; and, secondly, it is apt to engender in the patient a condition of chronic invalidism. A judicious amount of rest is however advisable, not only in lateral curvature but for all growing girls; since, when the spinal muscles are fatigued, positions are assumed in which the weight is supported by the passive structures, the ligaments and fibro-cartilages, and the forces which produce the lateral curvature come into play. After exercise, the fatigue of the morning and afternoon lessons, &c., it is desirable that the patient, if a girl, should have from half an hour to one hour, never longer, absolute rest in the recumbent position, and this rest may be repeated once or twice according to the length of the rest-interval and the general stamina of the patient.

5. *Removal of rigidity*.—In some advanced cases of lateral curvature the spine is rigidly held in the deformed position, which cannot be improved by any amount of manipulation by the surgeon. Until this rigidity has been to some extent overcome, it is useless to recommend the patient to hold himself in a posture he is unable to assume or be manipulated into, and it is folly to endeavour to exercise muscles whose points of attachments are immovably fixed. To overcome this rigidity the writer has found the so-called weight treatment of considerable service. The patient is placed near the wall, with the body in the horizontal position parallel to the wall, the hands or elbows according to his height resting on a chair. A broad bandage fixed to a staple on the wall is passed across the patient's back, and to the free end of the bandage a weight is attached. The staple is fixed at such a height that the bandage while passing across the back does not touch the apices of the spinous processes, nor the ribs on the concave side of the curve, but embraces the projection caused by the backwardly rotated ribs on the convex side. When sufficient weight is applied the ribs are pressed on and so carried forwards. A large water-can answers admirably, as the weight can be regulated by increasing or lessening the quantity of water. In some cases a counter-weight may be applied in a similar way to the lumbar curvature at the same time.

The weight treatment should be used cautiously, the weight being at first applied for a few minutes only, and then for longer periods up to ten minutes or a quarter of an hour according to the strength of the patient. Massage and passive manipulations will also be of assistance. As soon as the rigidity has been overcome the treatment by posture and exercises may be begun.

6. *Mechanical supports*.—These should never be employed in early and slight cases, nor as a general rule in advanced cases. They have practically no influence in correcting rotation; they are cumbersome and heavy, and tend to induce wasting and atrophy of the muscles. In those cases in which the curvature depends on spinal paralysis a support is, as a rule, essential; it is moreover almost a necessity for the poorer class of patients met with in the out-patient room. To tell a girl entirely dependent on her own exertions in some such trade as flower-making, book-binding, and the like to hold herself in a good position and to take a long

course of exercises is useless and cruel. For such patients a support must, however unwillingly, be prescribed: It will probably prevent the curvature getting worse, it will relieve the pains in the back, and it will enable her to follow her employment in comparative comfort. But she should be given to understand that she cannot expect in this way to be cured. She should be urged to attend once or twice a week at a hospital for a systematic course of exercises, and to snatch a few minutes daily for the practice of them.

Spinal braces are objectionable if worn all day. When, however, they are worn for an hour or so at a time as a reminder to the patient, who as soon as she feels them become tight knows that she is lapsing from the erect posture, they are of considerable service; but it should be clearly understood that she is not to trust to the brace for holding herself upright, but to regard it as a monitor of each relapse to her former stoop.

Kyphosis (SYNON.: Posterior Curvature) is a general curving of the spine, with its convexity backwards, and is an exaggeration of the normal backward dorsal curve. In infants it may be the result of weakness or general debility, or of long nursing in the upright position. In slightly older children it is generally due to the yielding of the softened bones as a result of rickets. In adolescents it is usually caused by muscular debility and relaxation of the ligaments, slouching habits in sitting or standing. In girls it is a frequent precursor of the more serious lateral curvature of the spine. In adult life it may be traced to occupations necessitating constant stooping, and is familiar in the round shoulders of the farm-labourer. In the old it is frequently the result of osteo-arthritis. A history of an hereditary tendency to round shoulders can frequently be obtained.

TREATMENT.—In all cases where possible the cause should be removed. In infants recumbency is indicated; in adolescents the avoidance of stooping habits and constant assumption of correct postures, and gymnastic exercises, should be insisted upon. In adults in whom the kyphotic curve is confirmed, and osseous changes have occurred, and in the subjects of osteo-arthritis, little or nothing can be done.

Lordosis (SYNON.: Anterior Curvature or Saddle-back) is a curvature of the spine with its convexity forwards, and is an exaggeration of the normal forward lumbar curve. It should be regarded as a symptom rather than as a disease, in that the curvature is formed to compensate for the loss of equilibrium in the spinal column when from any cause its antero-posterior curves have been disturbed. Thus it is met with in rickets, hip-disease, coxa vara, congenital or unreduced dislocation of the hip or ankylosis of the hip—conditions in which, owing to the femur being fixed in the position of partial flexion at the hip, the pelvis is tilted forwards when the affected leg is brought down parallel with its fellow. Lordosis is also met with as a compensating curve in the angular deformity of Pott's disease and in kyphosis, also in enlargement of the abdomen as from pregnancy, ovarian dropsy, &c., to restore the erect position. It also occurs in pseudo-hypertrophic paralysis, to compensate for the tendency of the spine to fall forward, owing to the weakness of the spinal muscles.

Angular Curvature.—See CARIES, p. 1565.
W. J. WALSHAM.

SPIRRILLUM (dim. of *spira*, a twist, a curl).—*See* BACTERIA.

SPIROCHÆTE.—*See* BACTERIA.

SPIROMETER (*spiro*, I breathe; and *μέτρον*, a measure).—SYNON.: Fr. *Spiromètre*; Ger. *Spirometer*.

DEFINITION.—An instrument for measuring the vital capacity of the chest.

DESCRIPTION.—The object of the several instruments that have been designed for this purpose is to measure the total amount of air expelled from the chest by the deepest expiration following upon the deepest inspiration.

The instrument designed by Hutchinson consisted of a mouth-piece and tube communicating with a gasometer of registered and graduated capacity, into which the patient breathed. Various modifications of Hutchinson's instrument have been devised.

A convenient and accurate spirometer, which works on the principle of the anemometer, was introduced by Lowne. The advantage of this instrument is its portability.

RESULTS.—According to Hutchinson, the vital capacity varies according to *height, weight, age, and disease*.

1. *Height.*—There is an increase of 8 cub. in. in vital capacity for every inch in height between 5 ft. and 6 ft. Thus the vital capacity of a healthy person at 5 ft. to 5 ft. 1 in. being 174 cub. in., at 5 ft. 4 in. it would be $174 + 32 = 206$ cub. in.; at 5 ft. 8 in., 238; &c.

2. *Weight.*—Excess in body-weight is associated with diminished capacity, in the proportion of about 1 cub. in. per lb. excess.

3. *Age.*—From thirty to sixty years the vital capacity decreases nearly $1\frac{1}{2}$ cub. in. per year.

4. *Disease.*—The spirometer furnishes a very accurate standard of health or of the extent of disease, as regards the chest, the vital capacity in lung-disease diminishing from 10 to 70 per cent.

R. DOUGLAS POWELL.

SPITTING OF BLOOD.—*See* p. 620.

SPLEEN, Diseases of.—SYNON.: Fr. *Maladies de la Rate*; Ger. *Krankheiten der Milz*.

GENERAL REMARKS.—Although certain affections have been, and still are, described under the heading of diseases of the spleen, it is doubtful if, with the exception of the results of traumatism, and perhaps one form of enlargement, there is one single disease affecting the spleen which is not secondary to, or at least coincident with, disease elsewhere in the body, such disease being almost invariably of a kind in which the blood is deeply affected. This fact has become more and more recognised of late years, so that diseases of the spleen described as such have grown less and less in number, the various disordered conditions of the organ being for the most part classed under *malaria, anaemia, leucocythæmia*, and other diseases in which it is variously affected. The diseases in which it is common to find the spleen enlarged are numerous, and in nearly all these the splenic affection is distinctly secondary, the only possible exception being 'splenic anaemia,' in which it seems that it may perhaps be primary; in 'splenic (or spleno-medullary) leucocythæmia' it would appear that the splenic affection is probably secondary to the marrow-

changes. Uncertainty as to the relation of these splenic affections to the anæmias must prevail until more is known of the physiology of the spleen as concerned with the formation and destruction of blood-corpuscles, and the condition of the blood generally. At present the one point apparently settled is that the spleen is intimately concerned with hæmolysis, or destruction of red corpuscles. Numerous cases of removal of the spleen, both when more or less diseased and when apparently healthy, have been now recorded, and it seems that the loss of this organ can be borne without other than occasional temporary impairment of health. Removal of diseased or enlarged spleens has been followed by no symptoms, and a similar result has followed in some cases the removal of spleens for traumatic rupture; but in two cases reported by Pitts and Ballance, of removal of healthy spleens for rupture, the operation was followed by extreme anæmia, a daily rise of temperature of 1° to 3° F., increased frequency of pulse, enlargement of external lymphatics, diminution of red corpuscles in blood, with increase in leucocytes and hæmoglobin, all of which symptoms gradually passed off. According to Rolleston, when the spleen becomes diseased some sort of compensatory action as regards its functions is apparently set up, possibly in the bone-marrow, and the more complete this compensation the less is the spleen missed; hence the absence of symptoms after removal of enlarged spleens, their presence after removal of healthy spleens for rupture, and their absence in many instances of removal of spleens for rupture, when, as is often the case, this accident has occurred to a spleen the subject of old-standing disease.

The spleen, from the nature of its structure, is capable of accommodating at times a very large amount of blood, and is very susceptible to hyperæmia. It varies considerably in size in health; thus its bulk increases for some hours after taking food, while digestion is going on, after which it lessens again. A peculiarity of the spleen that may be mentioned here is that in many cases of infectious disease dependent on micro-organisms, such as anthrax, enteric fever, and plague, these organisms are present in large numbers in the blood of the spleen, though they be found with difficulty, or perhaps at times not at all, in the general systemic blood. In cases of malarial infection, also, the splenic blood contains the parasite in large numbers, and also quantities of pigment and *débris* of broken-down parasites. As regards these points, the question has been raised as to whether the spleen may not exercise some protective influence over the blood.

VARIETIES.—The diseased conditions of the spleen will be noticed in the following order: (1) acute inflammation; (2) the various forms of enlargement of the organ, both such as may be primary and those that are certainly secondary to, or dependent on, other diseases; (3) other splenic affections, malformations, and anomalies; (4) rupture.

1. Acute Inflammation.—**SYNON.** : *Splenitis*. Acute hyperæmia of the spleen is common in certain forms of fever, but it is extremely doubtful if this condition ever proceeds to a general inflammation of the spleen; it is doubtful, also, if any general inflammation of the organ is ever caused traumatically, inflammation from violence being more likely to occur in isolated spots in consequence of laceration

of the structure of the gland and extravasation of blood, i.e. of slight degrees of rupture. Acute inflammation is not by any means common, but, when it occurs, it is in the great majority of cases the result of embolism, such embolism in a branch of the splenic artery resulting in an 'infarct' (see **EMBOLISM**). Splenic infarcts are usually of the red variety, though they may ultimately become pale as they shrink and disappear. In their later stages they are surrounded with a dense whitish layer of cicatricial tissue. Septic infarction leads to abscess-formation, and if suppurating infarcts be numerous and near one another, the resulting foci may coalesce, and a large abscess be formed. Splenic emboli are apt to occur in valvular disease of the heart, in chorea, in infective endocarditis, in pyæmia and septicæmia, in typhoid and other fevers, and in any case in which thrombi may form in the blood; and it is obvious that, according to their origin, they may be innocent or infective. Inflammation of either kind may be accompanied by hyperæmia of the organ generally, which may thus be enlarged; or the peritoneal covering of the gland may be affected, leading to adhesions to neighbouring parts.

Inflammation of the spleen may go on to suppuration without any marked local symptoms, and the evidences of such inflammation are far more frequently seen on the *post-mortem* table than diagnosed during life. Local symptoms consist mainly of pain in the region of the spleen, with some tenderness, especially if local peritonitis is present, and perhaps some enlargement of the organ. Suppuration may be indicated by shivering and other constitutional signs of that process; it does not cause any local symptoms unless the abscess is large and near the surface, and the spleen adherent to the abdominal wall, in which case there may be swelling, tenderness, pain, and even local œdema and fluctuation.

2. Enlargements.—The enlargements of this organ will be described according to the several diseases in which they occur.

(a) *In splenic anæmia.*—In this affection, sometimes called *primary splenomegaly*, it is probable that the splenic disease is primary; the anæmia is progressive and is characterised by considerable diminution in the number of the red-corpuscles (which, however, rarely fall below 50 per cent. of their normal quantity), by great diminution of hæmoglobin, and by a lower colour-index ($\cdot 5$) than any other form of anæmia; there is no increase in the number of leucocytes. The spleen is greatly enlarged, though not to the extent often met with in splenic leucocythæmia; it is tender; and the patient is subject to attacks of pain in the region of the gland. The spleen may weigh from two to eight pounds, and contains enormous numbers of large nucleated cells enclosing blood-corpuscles; there is general fibrosis of the organ, the capsule is firm, and it and the trabeculæ are thickened; the pulp appears to be diminished in quantity, and the Malpighian bodies are small and atrophied; there may be peritoneal adhesions. There is often some irregularity of temperature, but no enlargement of lymphatic glands. Hemorrhage is infrequent and the stomach is the commonest source. The disease is generally fatal in three years.

(b) *In splenic or spleno-medullary leucocythæmia.* In this affection the spleen is generally greatly enlarged. See p. 866.

(c) *In malaria.*—In acute malarial disease the

spleen is enlarged from hyperæmia ; in cases which end fatally the substance is found to be diffult, dark, and full of pigmented elements and *débris* of parasites. In chronic malarial disease, or malarial cachexia, the spleen is often very much enlarged ; the capsule and trabeculæ are thickened, the consequence probably of constant hyperæmia, but the pulp is at the same time increased, and hence the organ is soft and friable. Spleens thus enlarged, which are very common among Asiatic races, and probably in malarious countries generally, are very liable to rupture under slight violence. See MALARIAL DISEASE.

(d) *In amyloid disease.*—In this disease the spleen only shares with other organs in the effects of a general affection (see AMYLOID DISEASE). A spleen affected with amyloid disease is uniformly enlarged, hard, smooth on the surface, and painless. It is inclined to be globular in shape. No ascites or other evidence of pressure on the portal vein or bile-ducts occurs unless the disease is complicated by some other condition.

(e) *Under other conditions.*—The spleen is temporarily enlarged from hyperæmia during the course of certain fevers, especially of enteric fever and plague ; in such cases the enlargement subsides with recovery from the fever. It is also nearly always enlarged, though not to any very great extent, in lymphadenoma, and occasionally in idiopathic or pernicious anæmia. In the former disease the Malpighian bodies are enlarged and yellowish, exhibiting the proliferation of lymphoid cells characteristic of the disease ; in idiopathic anæmia iron is found deposited in the spleen, and it is possible that the organ plays an accessory part in the destruction of red blood-corpuscles in this disease. Enlargement of the spleen also takes place in consequence of general congestion of the portal system, notably in cirrhosis of the liver, and to a less degree in valvular disease of the heart ; it also may be due to malignant disease which is usually secondary.

The only subjective symptoms associated with enlargements of the spleen are occasional pain in the side with some tenderness ; and, if the spleen be very large, a feeling of weight in the left side of the abdomen, and inconvenience from increased size in that region. For the physical signs of enlargement of the spleen see PHYSICAL EXAMINATION, and accompanying plates.

3. *Miscellaneous affections.*—*Tuberculosis* may affect the spleen—so far as is known, always secondarily to other organs—and the appearances presented may be either of the ‘miliary’ or ‘caseous’ type. The spleen is said to be often enlarged in *syphilis*, but it is rare to find gummata in this gland. New-growths and hydatid cysts have been described as occasionally found in the spleen, but both these conditions are excessively rare, and malignant disease is probably always secondary. The chief anomalies of the spleen are those known as ‘wandering spleen’ and ‘supernumerary spleens’ ; the latter condition is simply an anatomical variation, and possesses no pathological interest, the former may occasionally cause trouble in much the same way as movable kidney, and may sometimes justify surgical measures for its relief.

4. *Rupture.*—In England rupture of the spleen only happens from injuries similar to those which cause rupture of other internal organs, but in Asiatic (and probably in all malarious) countries, where the possession of an enlarged spleen with a friable pulp

is common among natives, this accident is of common occurrence, and has led to trials for manslaughter in cases in which no serious injury was intended. The possibility of this accident having occurred should always be borne in mind in cases of sudden death following any slight tussle. Death generally follows rapidly on the injury, but this is not always the case : in two cases which came under the observation of the writer an injury was followed by intense pain and collapse, from which temporary recovery took place, to be followed by sudden collapse and death about three days after. In both these cases *post-mortem* examination showed a torn spleen enclosed in a torn distended capsule, the whole appearance reminding one of a placenta in a bag of membranes ; the first violence had ruptured the spleen, but not the capsule, into which hæmorrhage had taken place, distending it greatly ; subsequently the capsule, possibly from some sudden movement of the patient, had given way, and the mass of fluid and clotted blood had been suddenly discharged into the peritoneal cavity, causing collapse and death. The symptoms of rupture of the spleen are pain, collapse, and signs of internal hæmorrhage.

TREATMENT.—The treatment of splenic diseases resolves itself for the most part into the treatment of the accompanying conditions. In acute inflammation pain may be relieved by hot applications, or if great active hyperæmia be suspected, by leeches. In the rare event of it being possible to localise suppuration in the spleen, and to determine adhesion of the organ to the abdominal wall, it would be right to evacuate the pus, but indiscriminate exploratory puncture is to be deprecated, from fear of hæmorrhage. In pure splenic anæmia no satisfactory treatment has been found ; it has been stated that removal of the spleen has done good in this affection, and, if so, this would seem to indicate that the splenic condition is primary. In the splenic affection consequent on malarial poisoning the enlargement is often very obstinate ; it is best treated by measures which tend to improve the general health and combat the malarial cachexia. Iron and quinine, or more especially arsenic, will be found of service, with an occasional dose of Carlsbad salts ; locally, tincture or solution of iodine may be employed, or a piece of lint about four inches square spread with the ointment of red iodide of mercury may be applied and kept on for four or six hours ; this last remedy is often very useful, but it requires a little care, as if left on too long it is apt to blister the skin ; in any case the irritant effect is more lasting than that of a mustard plaster. Flannel cholera-belts should be worn in cases of malarial spleen. For rupture of the spleen the treatment is laparotomy and removal, as speedily as possible, of the ruptured gland ; several successful cases of this operation have now been reported. See LEUCOCYTHÆMIA.

MAX. F. SIMON.

SPLENIC ANÆMIA.—See pp. 1573 and 865.

SPLENIC FEVER.—See PUSTULE, MALIGNANT.

SPORADIC (σπειρώ, I scatter).—This term is used in connection with diseases which occur occasionally, and in an isolated manner, among individuals ; as distinguished from those which prevail endemically or epidemically.

SPORADIC CHOLERA.—See CHOLERAIC DIARRHŒA.

SPRAYS, Therapeutical Uses of.—See INHALATIONS.

SPURUE.—See PSILLOSIS.

SPUTUM.—SYNON.: Fr. *Crachats*; Ger. *Auswurf*; *Sputum*.—This article deals with the diagnostic indications afforded by a microscopical examination of the sputum, and with the requisite methods of preparation. The pathological constituents of the sputum to which definite diagnostic importance can be attributed are comparatively few in number. These will be considered under the following heads: (1) Micro-organisms; (2) Elastic tissue; (3) Curschmann's spirals.

No special description will be devoted to the rare instances in which hooklets derived from hydatid cysts in the lung, or portions of laryngeal or pulmonary new-growths, may be discovered in the expectoration, inasmuch as these structures require no special preparation for their identification.

THE COLLECTION OF SPECIMENS OF SPUTUM FOR EXAMINATION.—The early morning expectoration, representing the gradual accumulation formed during the hours of sleep, is the most likely to contain a mixture of the secretions of the respiratory tract, and is therefore the most suitable for examination; moreover, the sputum expelled at this time is less likely to contain particles of food. It is often important that the specimen should be as free as possible from contamination with the saprophytic micro-organisms of the buccal cavity; consequently the patient should be directed to rinse the mouth out thoroughly, using sterile water (e.g. water that has been boiled and allowed to cool), three or four times in succession before expectorating. The sputum is then coughed up from the lungs, and expectorated into a clean wide-mouthed glass bottle or other suitable vessel, which must not contain any disinfectant solution whatever.

1. Micro-Organisms.—*Tubercle-bacillus*.—As a preliminary to microscopical examination the sputum should be poured out into a flat glass dish, and search made for yellowish, i.e. purulent, streaks and spots (a process which is facilitated by placing a piece of black paper under the dish); where the sputum is more or less opaque throughout, the thickest and most curdy parts are to be selected. A small selected portion of the sputum is now transferred to a clean cover-slip by the aid of a stout platinum rod, or a piece of copper wire having one end beaten out to form a small spatula, spread out in a thin film, and allowed to dry thoroughly in the air. In the case of thin watery sputum, a second or even a third film may be spread over the first. The cover-glass, film upwards, is now passed rapidly three or four times through the flame of a spirit-lamp or Bunsen's burner; this coagulates the albuminous matter and fixes the film firmly to the glass. The film is now ready for staining, a process which is most conveniently effected by the Ziehl-Neelsen method. This method depends on the fact that the tubercle-bacillus, probably by reason of the wax that enters so largely into its composition, is extremely difficult to stain with aniline dyes as ordinarily used, but having once taken up the stain, is equally reluctant to part with it to decolourising reagents. This peculiarity the tubercle-bacillus shares with several other organisms, such as the bacillus of leprosy, the smegma-bacillus and the Timothy-grass bacillus.

The following reagents are needed:

(A) Carbol-fuchsin. This is prepared by dissolving 1 gramme of basic fuchsin in 15 cc. of absolute alcohol, and making up to 100 cc. with a 5-per-cent. solution of carbolic acid.

(B) Alcohol—preferably rectified spirit, although methylated spirit will answer the purpose.

(C) A 25-per-cent. solution of sulphuric acid.

(D) Methylene blue—a 1-per-cent. watery solution.

Method of staining.—(1) Warm some of the carbol-fuchsin (A) in a watch-glass over the naked flame or on a sand-bath, until steam commences to rise; float the cover-slip preparation, film downwards, on the surface of the fluid, and keep gently steaming for 5–10 minutes.

(2) Remove the cover-slip from the stain and wash in water.

(3) Wash with alcohol (B) until no more colour is discharged.

(4) Place in sulphuric acid (C) for one or two seconds, then immediately wash in water. If the film is 'red,' it must be again immersed in the acid, then washed in water. The film should now be of a light pink colour.

(5) Counterstain with methylene blue (D) for 10 to 20 seconds.

(6) Wash in water, blot off the excess of water with blotting-paper, complete the drying by holding the cover-slip between thumb and forefinger over the flame, and mount in a solution of Canada balsam in xylol.

For the recognition of the tubercle-bacilli, a magnifying power of 250 to 300 is sufficient in most cases, if the illumination be brilliant. Abbé's sub-stage condenser greatly facilitates the detection of the bacilli; and in all doubtful cases, where the microbes are scanty, the combination of Abbé's condenser with a $\frac{1}{2}$ -inch oil-immersion lens is indispensable. With the method of staining above described, the bacilli appear as delicate crimson rods, in length from one-quarter to one-half the



FIG. 1.—Tubercle-Bacilli in Sputum. $\times 550$.
The bacilli are seen as dark rods, lying amid threads of mucus and a few pus-corpuscles.

diameter of a human red blood-corpuscle. The rods may present a uniform bright-red appearance, the staining may be unequal, or the microbes may present a beaded appearance resembling a string of red granules. Very little clinical importance can be attributed to these morphological differences, although the moniliform appearance is indicative of young actively multiplying tubercle-bacilli. Various other bacilli and micrococci contained in the sputum, as well as the nuclei of cells and filaments of mucin, take the blue stain, contrasting sharply with the red tubercle-bacilli.

SIGNIFICANCE.—The discovery of tubercle-bacilli in the sputum is a decisive proof of the existence of tubercular disease of some part of the respiratory system. In cases of tuberculosis of the air-passages, bacilli are discharged from the surface of the ulceration; but the number derived from this source is insignificant as compared with the quantities that come from cavities in the lungs. If we exclude the extremely rare cases of primary tubercular ulceration of the main air-passages, it may be affirmed that a large number of bacilli in the sputum is a certain sign of pulmonary excavation. The size of the cavity or cavities is immaterial, but free communication between a vomica and a bronchus is a necessary condition.

The number of the bacilli is no certain index of the severity or extent of the disease, and may be said to be largely a question of discharge. In some acute pneumonic forms of tuberculosis the microbes may for a time be very scanty, whereas in cases of the most chronic and limited disease the sputum may teem with tubercle-bacilli. It has been asserted that where tubercular ulceration of the larynx exists the number of bacilli is always large. This statement is incorrect, as numerous observations have convinced the writer. Complete and permanent disappearance of the bacilli from the sputum is a most favourable sign; but, on the other hand, persistent expectoration of these micro-organisms is not incompatible with a favourable and chronic course of the disease.

Actinomycosis.—In the rare instances in which this disease attacks the respiratory system, the characteristic ray-shaped organisms may be discharged in the expectoration. These appear as minute yellowish granules visible to the naked eye in the purulent discharge. When a drop of the pus is examined without any staining, the rosettes of clubs can readily be recognised with a moderately high power. If dried cover-glass preparations be stained by Gram's method, combined with rubine or eosine as a counter-stain, the mycelial threads and clubs may be demonstrated. The presence of the ray-fungus is pathognomonic of actinomycosis. See ACTINOMYCOSIS.

Pneumonia.—Among the various micro-organisms that may be found in the sputum of croupous pneumonia, contaminated as it usually is in its passage through the mouth, the *Diplococcus pneumoniae* of Fränkel is the most important numerically in a large percentage of the cases. Occasionally its place is taken by the short pneumobacillus of Friedländer, or by the convoluted chains of the *Streptococcus pyogenes longus*. The *Diplococcus pneumoniae* and the pneumobacillus are characterised by the possession of distinct capsules, which may be demonstrated microscopically by the use of MacConkey's capsule-stain. This is prepared by dissolving 0.5 gramme of Dahlia and 1.5 gramme of Methyl Green (oo crystals) in 100 cc. distilled water, adding 10 cc. of a saturated alcoholic solution of basic fuchsin, and making up to 200 cc. by the addition of distilled water. In specimens of pneumonic sputum stained by this mixture, the capsules appear as delicate magenta-coloured halos surrounding the more darkly stained bacteria. These organisms may be stained by any of the anilin dyes, and of the three above-mentioned forms, the pneumobacillus is alone decolourised by Gram's method. See also p. 132.

As similar microbes are sometimes met with in

other diseases, such as bronchitis, and occasionally in healthy saliva, no definite diagnostic significance can be attributed to them when occurring without any other indications of disease.

Influenza.—The sputum should be carefully examined for small semi-solid yellowish-green masses: in these the specific bacillus of influenza can usually be demonstrated by staining film-preparations for long periods with very weak solutions of fuchsin; 1 cc. of a saturated alcoholic solution of basic fuchsin in 10 cc. of distilled water makes a suitable strength, and the preparation should be subjected to its action for a period of two or three hours. The influenza-bacillus then appears as a minute bacillus, in length about equal to one fifth the diameter of a red blood-disc, often staining most deeply at the two extremities, and so resembling a diplococcus; usually situated within or in the neighbourhood of pus-cells. The bacillus is decolourised by Gram's method.

The presence of the *Bacillus influenzae* in the sputum may be looked upon as pathognomonic of the disease, and as it makes its appearance within about twelve hours of the onset of an attack, the examination of the sputum is often of great assistance in diagnosing between influenza and pneumonia.

2. Elastic Tissue.—In destructive pulmonary disease of all kinds, portions of the elastic framework of the lungs are from time to time expectorated, and can be recognised with the microscope. In some cases the elastic tissue may be detected without any special preparation. The sputum is poured out into a flat glass dish, and some of the most opaque parts are selected and transferred to a glass slide; a cover-glass is then pressed lightly on the sputum, and the specimen can be at once examined.

But for general use Fenwick's solvent method is much more satisfactory. Equal quantities of the sputum and a solution of caustic soda (twenty grains to the ounce) are boiled for a few minutes until the mixture becomes liquefied. It is important not to continue the boiling too long, as the elastic fibres themselves ultimately become much altered in ap-



FIG. 2.—Elastic tissue of the lung in Sputum. $\times 550$.

pearance. The fluid is now set aside for two to twelve hours in a tall conical glass, to allow the elastic tissue to sink to the bottom; or, better still, placed in the centrifugal machine, in a conical test-tube, and run at a fairly high rate of speed for some minutes. A drop of the sediment is then withdrawn with a pipette, and examined with the microscope in the usual way. A magnifying power of 90 to 100 diameters is sufficient in most cases; but when the fibres are isolated or much broken up, a higher power may be required. In the latter case difficulties are apt to arise from the fact that the sputum

commonly contains adventitious fibres closely resembling elastic tissue. In some cases the curled-up ends and branching fibres may serve to distinguish the elastic tissue of the lung; but unless some trace of the characteristic alveolar arrangement be recognisable, it is unwise to hazard a positive diagnosis.

SIGNIFICANCE.—The discovery of elastic tissue in the expectoration may be regarded as definite evidence of destructive disease of some portion of the respiratory tract. With rare exceptions the elastic fibres come from the lung, though at times they may be derived from the larynx or bronchi, in which case no alveolar grouping will be found. Tuberculosis, by far the commonest ulcerative disease of the lung, may generally be suspected when elastic tissue is found in the sputum; but in non-tubercular destructive affections the same discovery may be made. Hence elastic fibres do not possess the pathognomonic significance of the tubercle-bacilli. In acutely progressing excavation large groups of alveoli are often expectorated; whereas in cases of chronic disease the fibres are usually more isolated, and are not uncommonly encrusted with lime-salts.

In the sputum of pulmonary gangrene the elastic fibres are generally scanty or ill-defined, and sometimes cannot be recognised at all. It has been suggested that this is due to the presence of a ferment in the gangrenous secretion, which dissolves the lung-tissue.

3. **Curschmann's Spirals.**—In the sputum expelled towards the close of an asthmatic attack small gelatinous sago-like lumps of mucus are generally present. If these masses be gently pressed out between a cover-glass and a glass slide certain spiral structures may commonly be recognised. The spirals vary much in size, often being visible to the naked eye, whereas at other times they can only be discovered with the help of the microscope. These structures consist of gelatinous threads of a mucoid substance, coiled in the form of a corkscrew. In many cases a central bright wavy or twisted thread occupies the hollow of the coil. Von Jaksch believes the central fibre to be chemically allied to fibrin, the spiral coil being composed of mucin.

SIGNIFICANCE.—These spirals are to be regarded as casts of the bronchioles. Curschmann considers that they are the result of an exudative bronchiolitis, and are causally related to the paroxysms of asthma. The spirals are not pathognomonic of asthma, as they are occasionally found in the sputum of bronchitis and croupous pneumonia. But, as von Jaksch suggests, their presence in a case of asthma of doubtful nature would stamp the bronchial nature of the attack. Charcot-Leyden crystals (p. 272) are often found adhering to the spirals, and sometimes seem to develop after the sputum has been allowed to stand for some hours. Von Jaksch believes that the crystals are products of chemical decomposition of mucin.

PERCY KIDD.

SQUINTING.—See STRABISMUS.

STAGNATION OF BLOOD.—Local arrest of the circulation. See CIRCULATION, Disorders of; and INFLAMMATION.

STAMMERING.—**DEFINITION.**—The word 'stammering' has been used by some writers in its broadest sense so as to include several different

forms of defective articulation, such as inability (congenital or acquired) to pronounce certain letters or combinations of letters, the tendency to hesitate or stumble in utterance, or to transpose letters or syllables, &c. It is better, however, to restrict its use to imply a spasmodic affection of the organs concerned in speech, in virtue of which the enunciation of words becomes suddenly checked, and a painful pause ensues, usually marked either by a prolongation, or a repetition in rapid sequence, of the particular literal sound at which the check arises. By several writers the word 'stuttering' is used as synonymous, but it is better to confine the use of the term 'stuttering,' as will be pointed out later, to one clinical variety of stammering which it is well to employ as the general descriptive title.

ÆTIOLOGY.—Stammering is to some extent hereditary, although in many cases no such connection can be traced. It is very frequently imitative, e.g. children may learn it from a companion—a point of considerable importance as regards its preventive treatment. The defect rarely shows itself before the age of four or five years. Usually it comes on from this time up to the period of puberty, but it may originate at any age. It is a curious fact that men stammer in much larger proportion than women.

DESCRIPTION.—The varieties of stammering are so numerous that anything like a complete consideration of them here would be impossible. It should be noted, however, that there are two great groups of stammerers: (1) those whose peculiarity consists in an attempt to overcome their difficulty in articulation by a series of fitfully and rapidly recurring repetitions of the letter at which they have come to grief, a condition to which the term 'stuttering' is best restricted; and (2) those who 'silently stick' at the difficult letter, which is not audibly produced for some seconds, although the patient may be making violent and even visible spasmodic attempts to enunciate it. Both classes of patients thus 'stammer'; the first 'stutters,' the second 'silently sticks.' As one writer has put it, the stutterer in trying to say such a word as 'top' enunciates a series of small 't's' in rapid succession, thus 't-t-t-top,' while a patient of the other class silently builds up a great big T! The clinical picture of each type is so familiar that little further description is called for, varying degrees of severity being met with. In his attempts to overcome difficult letters the patient may make such spasmodic efforts at ejaculation that he becomes quite flushed or even purple in the face, while still oftener an 'overflow' of muscular energy takes place to other groups of muscles, leading to *involuntary* twitchings of the lips, eyebrows, or eyelids, &c., or even violent jerking of the head and neck in severe cases. This condition should be clearly distinguished from another in which the patient finds his difficulty lessened if he *voluntarily* throws some other muscles into action—e.g. by stamping with one or both feet, tapping the fingers, clenching the hand, &c.—the outlet of muscular energy appearing thus to lessen the obstruction to his articulation. Sometimes it will be found that patients have been taught this as a method of overcoming their difficulty; in other cases the stammerer has learnt it by experience.

In by far the greater proportion of cases the consonants, and especially the 'explosive' consonants, are the chief stumbling-blocks of stammerers, only a very small proportion finding a difficulty with

vowels. Consideration of this part of the subject will come in more fittingly, however, when the treatment is taken up.

PATHOLOGY.—Stammering being essentially a functional disorder, a definite pathology is scarcely to be expected. Attempts have been made at times to associate its occurrence with various conditions present in the glottis, tongue, &c.; but there is little doubt that the condition is inherently due to a *faulty working* of a complex mechanism rather than any *defect of structure*. It should be borne in mind that in the enunciation of words a threefold mechanism is at work—(a) a current of air must be regularly supplied by the lungs; (b) phonation is effected by the laryngeal mechanism; while (c) articulation is obtained by the coaptation and separation of the lips, teeth, tongue, palate, &c. A form of stammering has been described in which the primary fault is said to lie in the respiratory mechanism, but this is so rare that for all practical purposes stammering may be considered as arising essentially from a *want of the proper harmony between the functions of phonation and articulation*. To illustrate this no better example could be got than that used in his work on ‘The Disorders of Speech,’ by Professor Wyllie, who points out that in violin-playing, in order to ensure the production of the proper musical sounds, the two mechanisms of the ‘bow-hand’ *producing* the sound and the ‘string-hand’ *modifying* the same must work in complete harmony. No overaction on the part of the ‘string-hand’ can ever make up for a failure on the part of the ‘bow-hand’ to perform its function. This overaction on the part of one of the co-ordinated mechanisms concerned in the enunciation of words is the essential error of the stammerer.

TREATMENT.—This—the most important portion of the subject—is unfortunately too often dealt with inadequately, or in merely vague general terms. And yet the necessity for treatment, and the satisfactory results that may be obtained, are such that an endeavour should always be made to remedy the defect. It is not sufficient to give merely general instructions to the patient as to speaking slowly and from a full chest, &c. He must be taught how to surmount his individual difficulties. The first essential is that the stammerer be made clearly to understand the double nature of the process in speaking, the equal need for *phonation* as well as *articulation*; and Wyllie’s example of the violinist’s combined working of two mechanisms harmoniously can scarcely fail to be understood by even young patients. It should then be explained to the pupil that his difficulty arises essentially from a failure to allot the *due proportion* to phonation and articulation respectively in his enunciation of words; and a reference to the fact (which he has probably discovered for himself) that stammering seldom, if ever, occurs in *singing*, will impress this more firmly on his memory. The teacher should then demonstrate to him that certain letters of the alphabet have no ‘voice’ in themselves, and that any attempt to insert ‘voice’ into such will only lead to an aggravation of the patient’s trouble. Similarly, if he tries to pronounce any of those letters which *normally* contain voice without allowing phonation to have its proper share along with articulation in their enunciation, stammering will again result. It is therefore with a view to making such points perfectly clear to pupils that Wyllie has drawn up what he terms a ‘Physiological Alphabet,’ which is here

reproduced by his kind sanction from his work on ‘The Disorders of Speech.’

A Physiological Alphabet (Wyllie).

I. VOWELS.

Y—IEAOU—W.

These should be pronounced in the Latin manner, as—
ēē, eh, ah, oh, oo; Y and W, though consonants, are given here for reasons in the text.

II. CONSONANTS.

	Voiceless Oral	Voiced Oral	Voiced Nasal Resonants
Labials (1st stop-position)	P (W)	B W	M
Labio-Dentals	F	V	
Linguo-Dentals	Th ¹ S	Th ² Z	
Anterior Linguo-Palatals (2nd stop-position)	Sh T (L)	Zh D L R	N
Posterior Linguo-Palatals (3rd stop-position)	K H or Ch	G Y (R)	Ng

The voiceless W and the voiceless L have been given in brackets, the former being now almost confined to Scotland, the latter being peculiar to Wales. The uvular (burring) R is also in brackets.

In it this all-important distinction between ‘voiceless’ and ‘voiced’ letters is emphasised, the vertical columns showing a complete list of the consonants grouped according to definite principles. The vowels, of course, are essentially ‘voiced.’ The letters W and Y are placed by Wyllie along with the vowels as being more closely allied to them than to consonants for reasons fully given in his work referred to. It is sufficient here to explain that since an *initial* W is really phonetically equivalent (as Wyllie says) ‘to a tight oo,’ such words as ‘will’ ‘wet’ should really be pronounced as if they began with a vowel, e.g. ‘ōoill,’ ‘ōoet.’ Similarly, an *initial* Y (as in ‘you’) is phonetically equivalent to ēē, and must be treated as a ‘voiced’ letter by the stammerer, who will be surprised at the ease with which such words as ‘Will you’ can be produced, if he pronounces them as if they were spelt ‘ōoill ēeou’; for the *initial* Y, pronounced *voicelessly* as ‘yeh,’ is an extremely common difficulty with stammerers. [The horizontal divisions of the table are not of the same importance from the stammerer’s point of view, but are given here as the table is reproduced from Wyllie’s work.]

The essential principle, then, of the equal importance of phonation with articulation in the enunciation of words having been made perfectly clear to the pupil, all that is required is that the teacher should go seriatim through the letters of the ordinary alphabet, and explain how and why they come to be arranged as shown in Wyllie’s table. It is not necessary here to do more than indicate a few of the points there shown. Thus, it is quite evident that if P (which is merely a ‘voiceless’

labial with an *explosive* character *silently* prepared for its enunciation) has 'voice' added to it, the letter B is produced; and similarly D and G ('hard') are the 'voiced' equivalents of T and K (or C 'hard'). The distinction between the *voiceless* 'th' of 'thin' and the *voiced* 'th' of 'thee' must also be made clear to the patient.

When the pupil has thoroughly grasped the inherent difference between the two groups of consonants, he should be taught that the essential principle that he has to remember in working out his own cure is this: that all letters possessing 'voice' should have their full vocal element given them, or even more than their normal quantity of 'voice'; while those letters which are themselves 'voiceless' are to be touched off as lightly as possible, the attention of the pupil being directed rather to the 'voiced' letter that precedes or follows the 'voiceless' consonant. Thus in saying, e.g., '*Big Peter made no sound,*' the stammerer is to emphasise the letters italicised—the lips are merely to be apposed for the formation of the 'P,' but no *sound* is attempted to be produced, the '-eter' merely acquires the *explosive* character of the voiceless 'P.' The fact that the nasal resonants (m and n) are both 'voiced' consonants should be clearly demonstrated, for the attempt to pronounce *voicelessly* all words commencing with these consonants is one of the commonest errors of stammerers.

Before summing up the various points in treatment, it may be well to refer to a few of the specially common difficulties of stammerers, and indicate briefly how best to surmount these. Words commencing with 'wh' often give rise to very great trouble owing to the patient trying to pronounce these *voicelessly*. If he will bear in mind the fact that 'w' is really a vowel (i.e. voiced), and further grasp the fact, which is clearly brought out by Wyllie, that phonetically the 'h' precedes the 'w' (so that the physiological spelling becomes 'hw'), he will discover that the enunciation of such words as 'when,' 'whether,' becomes quite easy, provided they are produced as if they were spelt (phonetically) 'hōoen,' 'hōoether.'

A similar difficulty often occurs with words commencing with 'sw,' which the stammerer usually attempts to utter *voicelessly*. Here, again, the 'voiced' oo of the 'w' must be given its full value, so that words such as 'swim' become phonetically 'sōoim.'

Lastly, the pupil should be made to understand clearly that all words in which the initial letter is J or (soft) G must really be pronounced by him as if they began with a (fully-voiced) D, e.g., 'Jew' is phonetically equivalent to 'Dzhōo.' J and (soft) G are thus really compound letter-sounds, the real *initial* of which is D.

In conclusion, then, the treatment aimed at is as follows. The pupil must be made to understand thoroughly the essential difference between phonation and articulation in speaking, and that it is worse than useless to attempt to make up for deficiency in vocalisation by overaction in articulation. He must practise speaking in a full ringing voice, and in troublesome cases it is sometimes useful to practise 'intoning' his lessons, so as to emphasise the value of the musical element in speech. He should then be taught the 'Physiological Alphabet,' and in connection therewith the following illustrative sentences (reproduced by Prof. Wyllie's kind sanction) will be found most useful, since they bring

out clearly the essential physiological distinctions between the groups of consonants:—

A. *Initials that contain voice.* [In pronouncing these the voice must be thrown boldly into the initial.]

1. 'Even ancient elves are awed over oozing.'
2. 'We visit the Zulus like ramblers yearly.'
3. 'My nephew.'
4. 'Best gold dust.'

B. *Initials that do not contain voice.* [These must be touched off lightly and the voice be promptly brought out in the succeeding vowel or voiced consonant.]

1. 'Far shores seem thinly hazy.'
2. 'Two poor comrades.'

The pupil should be enjoined to practise reading aloud daily for at least ten minutes, commencing always with the above set of sentences; and he should be encouraged further to draw up for himself (and practise the repetition of) other illustrative sentences, not only those containing initial consonants of *similar* character, but also those of *dissimilar* nature, so that he may readily acquire the habit of distinguishing between the *voiceless* and *voiced* consonants. Once he has mastered the principles upon which the treatment depends, all that is necessary is for him to notice in reading or speaking any letters that occasion him particular difficulty, so that he may learn from his teacher (if he has been unable to discover for himself from the 'alphabet') how such letters are to be enunciated. Lastly, he should learn to take inspirations at short intervals so that he may always have ample breath to enable him to give the full vocal element to all those letters requiring it, and he should make it a constant practice to speak or read (as Wyllie says) 'with voice, making music with the voice, and listening to it as he speaks.'

H. GRAHAM LANGWILL.

STANDARDISATION—Standardisation is the name given to the process by which certain official preparations are made to contain a definite and uniform amount of the alkaloid or active principle of the vegetable drug from which they are extracted.

To obtain the requisite strength, stronger percolates, containing a known quantity of active principle, are diluted with alcohol; or, again, to obtain extracts of proper strength and consistence, stronger and weaker extracts may be mixed, and stronger extracts may be diluted with distilled water or with milk-sugar as may be necessary. The Aqua Laurocerasi, mentioned below, has its strength adjusted either by the addition of hydrocyanic acid or by dilution of the distillate with distilled water. The details of the procedures required in each individual instance are given fully in the British Pharmacopœia under each preparation.

The following are the standardised preparations according to the last revision of the British Pharmacopœia in 1898. It will be noted that 1 per cent. equals 1 grain in 110 minims, or according to the metric system 1 gramme in 100 cubic centimetres:—

Extractum Belladonnæ Alcoholicum, containing 1 per cent. of the alkaloids of Belladonna-root.

Extractum Belladonnæ Liquidum, containing 0.75 per cent. of the alkaloids of Belladonna-root.

Tinctura Belladonnæ, containing not less than 0.048 per cent. nor more than 0.052 per cent. of the alkaloids of Belladonna-root.

Emplastrum Belladonnæ, containing 0·5 per cent. of the alkaloids of Belladonna-root.

Linimentum Belladonnæ, containing 0·375 per cent. of the alkaloids of Belladonna-root.

Unguentum Belladonnæ, containing 0·6 per cent. of the alkaloids of Belladonna-root.

Extractum Cinchonæ Liquidum, containing 5 per cent. of the alkaloids of Red Cinchona Bark.

Tinctura Cinchonæ, containing 1 per cent. of alkaloids of Red Cinchona Bark.

Tinctura Cinchonæ Composita, containing 0·5 per cent. of alkaloids.

Acetum Ipecacuanhæ, containing 0·1 per cent. of alkaloids.

Extractum Ipecacuanhæ Liquidum, containing from 2·0 to 2·25 per cent. of alkaloids.

Vinum Ipecacuanhæ, containing 0·1 per cent. of alkaloids.

Tinctura Jalapæ, containing 1·5 per cent. of Jalap Resin.

Aqua Laurocerasi, containing 0·1 per cent. of Hydrocyanic Acid.

Extractum Nucis Vomiceæ, containing 5 per cent. of Strychnine.

Extractum Nucis Vomiceæ Liquidum, containing 1·5 per cent. of Strychnine.

Tinctura Nucis Vomiceæ, containing 0·25 per cent. of Strychnine.

Extractum Opii, containing 20 per cent. of Morphine.

Extractum Opii Liquidum, containing 0·75 per cent. of Morphine.

Tinctura Opii, containing 0·75 per cent. of Morphine.

FREDERICK WILLCOCKS.

STAPHYLOMA (σταφυλή, a bunch of grapes).
 SYNON.: Fr. *Staphylôme*; Ger. *Staphylom*.—This word was applied by old writers, in the jargon which was once supposed to be scientific, to any limited protrusion of the tunics of the eyeball. It was first used to denote the protrusion which occurs in the circum-corneal sclerotic zone, as a consequence of localised inflammation of this region. The tissue affected by the inflammation in such a case becomes softened, yields to the intra-ocular tension, and projects; but being restrained by bands of lymph, or by thicker portions of its own structure, from projecting uniformly, the prominence becomes more or less sacculated; and the most prominent portions, being thinner than the rest, and permitting the dark pigment of the interior of the eye to show through, present an appearance which may be compared to that of a miniature bunch of purple grapes—a real or fancied resemblance from which the term ‘staphyloma’ was derived. This form has more recently been termed *staphyloma of the sclerotic*, to distinguish it from *staphyloma of the cornea*, which is the protrusion left when the corneal tissue has been destroyed by ulceration, either wholly or in part, and the resulting cicatrix, formed of iris-tissue coated over by lymph, yields to the pressure of the fluids within the eye and becomes prominent. Corneal staphyloma is described as either partial or complete, according to the amount of cornea which is replaced by cicatrix.

Staphyloma posticum is a phrase applied to that protrusion of a circumscribed portion of the sclerotic, in the immediate vicinity of the optic nerve, which occurs in some cases of myopia; and which, by increasing the elongation of the eyeball, increases also

the degree of the short sight. It would be highly desirable to abandon the term ‘staphyloma,’ in favour of ‘protrusion,’ with such appended words as might serve to indicate the place and nature of the change. See EYE, AND ITS APPENDAGES, Diseases of.

R. BRUDENELL CARTER.

STARVATION (Sax. *steorfan*, to perish).—This term is generally applied either to deprivation of food, or to the series of phenomena to which such want gives rise. The word is often used synonymously with *fasting*, which, however, may be more accurately applied to voluntary starvation. See FASTING.

STASIS (ἵσταμι, I stop).—Local arrest of the circulation. See INFLAMMATION.

STATISTICS, MEDICAL.—See MORBIDITY; MORTALITY; PERIODICITY IN DISEASE; PUBLIC HEALTH; and VITAL STATISTICS.

STEARRHŒA.—SYNON.: Seborrhœa; Steatorrhœa; Fr. *Stéarrhée*; *Séborrhée*; Ger. *Talgdrüsenausschwitzung*.

Stearrhœa is an affection of the skin which is met with in an idiopathic form, but more often as an attendant on other skin-diseases, such as folliculitis, acne, gutta rosea, and lupus.

DESCRIPTION.—Two varieties of this disease are described: (1) *Stearrhœa sicca*; and (2) *Stearrhœa oleosa*.

1. *Stearrhœa sicca*.—This form is usually associated with folliculitis, and constitutes common pityriasis capitis, or dandruff of the scalp. A similar affection is often met with as a ringed eruption on the back between the shoulder-blades, and on a corresponding part of the chest. This eruption is a variety of follicular inflammation involving the sebaceous glands as well as the follicles, in which stearrhœa is a constant feature; it is very often associated with stearrhœa capitis, so that it is impossible to dissociate the two diseases.

Stearrhœa sicca is usually met with on the scalp and eyebrows, and in adults is characterised by the formation of dirty-white or yellowish scales. The outermost of these become dry and fall off as a scurf. At the same time the hairs are shed, and those which replace them are imperfectly developed, so that partial baldness is caused. In uncomplicated stearrhœa there is no inflammation of the skin, and little itching; but it generally happens that the affection is associated with folliculitis, and then itching becomes a troublesome feature. This condition of the scalp is commonly known as *pityriasis capitis* or *alopecia furfuracea*, in which the skin becomes covered with great quantities of fine, pearly-white, glistening scales, which are constantly shed and fall upon the shoulders, giving the appearance of the hair having been powdered. There is, in fact, an excessive desquamation of cuticle; and when the hairs participate in the changes, as they often do, the vertex becomes bald. The degree of itching depends on the amount of folliculitis present. Stearrhœa capitis may be mistaken for dry eczema or psoriasis; but these diseases are rarely limited to the hairy part of the scalp, and the changes in the skin are much more marked.

2. *Stearrhœa oleosa*.—This variety of the disease is usually confined to the face, and is a very common complication of acne rosacea. It consists of an excessive secretion of sebum, which is changed

in character into an oily fluid. This oil is poured out on the surface of the skin, and gives it a shiny appearance. When dabbed with a piece of blotting-paper, the fluid is absorbed and its greasy nature at once becomes evident. From the ready adhesion of dust, the skin assumes a dirty look which is very characteristic. This affection is especially prevalent in spirit-drinkers.

TREATMENT.—The treatment of stearrhœa is chiefly local, and consists, in the first instance, in removing any crusts which may have formed from the accumulation of sebum. These should first be softened by a thorough application of oil, and then well washed with hot water and soap. The subsequent treatment consists in the daily use of the white precipitate, or nitrate, or red oxide of mercury ointment, well diluted with vaseline. At the same time the skin should occasionally be washed with a lotion containing soft soap or a little borax and ether. Before any local treatment is adopted, it should be carefully explained to patients that the process of rubbing off the crusts is always attended by the removal of a large number of loose hairs, otherwise they are apt to think that the loss of hair is caused by the treatment rather than by the disease.

ROBERT LIVEING.

STEATOMA (στέαρ, fat; and the termination *oma*, adopted to indicate a tumour).—**SYNON.**: Fr. *Stéatome*; Ger. *Steatom*.—A sebaceous cyst. See CYSTS.

STEATOZOON (στέαρ, fat or sebum; and ζῶον, an animal).—A synonym for the *Acarus folliculorum*. See ACARUS.

STEEL-GRINDER'S PHTHISIS.—See PNEUMOCONIOSES.

STELLWAG'S SIGN.—A widening of the palpebral fissure occurring in many cases of exophthalmic goitre, bearing no constant relation to the exophthalmos, and due partly to retraction of the upper eyelid.

STENOSIS (στενῶω, I constrict).—A constriction, narrowing, or stricture of an opening or a tube; for instance, *mitral* or *aortic stenosis*, in the heart; and *stenosis of the œsophagus*.

STERCORACEOUS (stercus, dung).—Fæcal; a term generally applied to vomited matter when it presents the characters of feces. See VOMIT: Examination of Vomited Matters.

STERILITY IN THE FEMALE.—**SYNON.**: Barrenness; Fr. *Stérilité*; Ger. *Unfruchtbarkeit*.

DEFINITION.—Want of the power of reproduction in the female.

FREQUENCY.—In the general community the proportion of childless marriages seems to be about 1 in 8 or 8·5; among members of the peerage 1 in 6·11. Whether Kehler be correct in estimating that the husband is in fault in at least one-fourth of the cases of *sterilitas matrimonii* remains to be proved. Doubtless he is nearer the truth than those who attribute the sterility in nine cases out of ten to some fault in the wife, because while the comparatively rare cases of male impotence are readily enough recognised, and also the rarer cases of *aspermatis*m, the cases of *azoöspermatis*m, where

an azoic semen is ejaculated, are for the most part altogether overlooked. The possibility that the cause of the childlessness may be found in the male must, therefore, always be borne in mind. See IMPOTENCY; and STERILITY IN THE MALE.

ÆTIOLOGY.—For generation the essential product in the female is the ovum; and in her reproductive apparatus we find (i.) *oviparous organs* for its production; (ii.) *oviducts* for its transmission; (iii.) an *ovigerent organ* or nest in which the ovum is hatched; and (iv.) *copulative organs* for the reception of the semen, the spermatozoa of which constitute the essential contribution of the male. In a married woman in whom the generative function is in abeyance, the sterility may be either primitive or acquired. In the former case we have to do with a female who has never borne a child; in the latter the woman may have borne one or more children, but has for some years ceased to conceive. In either case we search for the fault in one or more of these four planes of her sexual apparatus.

I. Faults in the Ovaries.—The ova are developed in the ovaries, and the conditions which interfere with ovulation—that is, the regular ripening of an ovisac, and the discharge of an ovum—diminish or destroy the possibility of conception. Such conditions are found in:—

(1) *Absence or imperfect development.*—Cases of absence or defective development of the ovaries are rarely met with, except in women in whom the rest of the sexual apparatus is also anomalous.

(2) *Displacements.*—One or both ovaries may be found displaced. Instead of lying at the level of the pelvic brim, they have fallen into the pouch of Douglas. In this position, though the ripening and dehiscence of the ovisacs may be duly taking place, the discharged ova are not received into the free extremity of the Fallopian tube. The displaced ovary, moreover, is extremely likely to be the seat of some degree of inflammation.

(3) *Inflammation.*—Oöphoritis, acute or chronic, lessens the conceptive power in various ways. It may lead, first, to destruction of the follicles, so that no ova are produced; secondly, to condensation of the stroma, so that the regular ripening of the ovisacs is impeded; thirdly, to deposits on the surface, which prevent the dehiscence of the ovisacs; or, fourthly, to adhesions of the ovary in situations which hinder the entrance of the discharged ova into the oviducts.

(4) *Degenerations.*—The neoplastic degenerations to which the ovaries are most liable are the cystic; and all the varieties of cystomata, as well as the fibromata, the sarcomata, and the carcinomata, are commonly attended by sterility. Where both ovaries are affected the sterility is absolute, from the complete loss of function in the organs; and even where only one is affected, the disturbance in the relations of the pelvic organs, caused by the growing mass, is likely to prevent impregnation. See OVARIES, Diseases of.

II. Faults in the Oviducts.—The Fallopian tubes or oviducts serve not only for the reception of the discharged ova, and their transmission downwards to the uterus; they may serve also for the upward transit of the spermatozoa. Lawson Tait maintained that the entrance of spermatozoa into the tubes is pathological; but most authorities are of opinion that in them the male and female elements come into union.

(1) *Absence*.—Defective development of the Fallopian tubes is usually associated with other abnormalities of the sexual apparatus, especially with rudimentary conditions of the uterus.

(2) *Inflammation*.—Inflammatory changes may be found affecting either the external serous covering, or the internal mucous lining. In the former case sterility results from adhesions, which lead to displacements of the free extremities, so that they are not in a position to receive the ova discharged on the bursting of an ovisac; or from bands which constrict the tubes, and so occlude their canal. In the latter, changes in the secretion may prejudice the vitality of the spermatozoa or ova; or the thickenings, polypoidal or other, may obstruct the canal; or complete atresia may be produced, and their permeability be thus entirely lost. See FALLOPIAN TUBES, Diseases of.

(3) *Degenerations*.—The tubes are rarely enough the seat of neoplasms; but when such do develop in their walls, occlusion of their canal and consequent loss of function may ensue.

III. *Faults of the Uterus*.—In the process of reproduction, the uterus serves as the receptacle or nest, in which the fertilised ovum is carried during the period of incubation. In its proliferating mucous membrane the chorionic villi take root; through its expanded blood-vessels the foetal blood is brought into relation with the maternal; its walls grow in correspondence with the increase in size of the ovum; and its largely developed muscular fibres are the main agents in the expulsion of the ovum when it is finally hatched. It plays such an important part in the female economy that the name of it is often used as synonymous with the sexual apparatus; and some of its morbid conditions are among the commonest causes of sterility.

(1) *Defective development*.—First, the uterus may be absent altogether, or represented merely by a fibrous nodule. Secondly, it may be small, having undergone arrest at some stage of its growth, and remaining infantile, juvenile, or adolescent. Thirdly, it may be *bicornuous*—retaining the trace of its original duplicity by the presence of a septum running through the body alone, or running through both body and cervix, perhaps through the vaginal canal as well. Fourthly, it may be *unicornuous*—only one of the halves of the organ having been developed, while the other tube may be obliterated, or attached as a rudimentary by-horn to the better-developed tube. Fifthly, a more frequent malformation is found in a *conical* form of the cervix, which is not infrequently complicated with, sixthly, *narrowness of the os*. This last condition may exist by itself, forming a well-recognised cause of sterility, and furnishing some of the cases in which a most satisfactory cure can be accomplished.

(2) *Displacements*.—First, *descent* of the uterus is found as the predominant morbid condition in some cases of acquired sterility, but this is more frequently associated with the deviations anteriorly or posteriorly. Of these, secondly, the *antrorsions*, flexion and version, are very frequent among women who have never conceived at all; thirdly, the *retroversions*, flexion and version, are more common in women who have given birth to one or more children, and have subsequently remained sterile. The flexions, in particular, form a very clearly recognisable and often remediable cause of sterility.

(3) *Changes in size*.—The retrogressive changes which occur in the uterus after labour sometimes go

on morbidly, and in one group of cases leave the organ in a condition of (1) *super-involution*. The uterus may be reduced to a little tube which only admits the sound for half an inch. Even when the degree of super-involution is less, and the uterus still measures two and a quarter inches in length, it is apt to cause amenorrhoea and sterility. In another group of cases the uterus remains hypertrophied in a condition of (2) *sub-involution*, which is inimical to conception; and when conception does take place in such a uterus, abortion is liable to occur.

(4) *Inflammation*.—Among the commonest causes of sterility must be ranked the inflammatory changes to which the uterus is so liable, whether the process have affected mainly the external, middle, or internal coat; and in many of the cases where some other condition tending to sterility is present, inflammatory changes come in to increase the difficulty, and to cloud the prospects of recovery. First, *perimetritis* is usually only an element of a more general pelvic peritonitis, which often leaves behind it fixations and displacements of the uterus, preventing conception or promoting early abortion. Secondly, *mesometritis*, leading to thickening of the walls of the organ, produces an expansion of its cavity and disturbance of its function. It is rarely possible to dissociate this from, thirdly, *endometritis*, which is attended also by dilatation of the cavity, but which is further mischievous from the deleterious influence of its abnormal secretions on the life and progress of the spermatozoa, and from the difficulty with which a fertilised ovum gets healthily engrafted on its surface. Moreover, in certain cases of long standing, some of the uterine orifices may become more or less occluded, a result which is more especially apt to ensue in the external orifice when caustics have been applied to the cervical canal.

(5) *Degenerations*.—First, *myomata*, or fibroid tumours, are found in a considerable proportion of barren women. Whether sub-peritoneal, intramural, or sub-mucous, they interfere in many ways with conception, and give a proclivity to miscarriages or dangerous labours when conception has occurred. Secondly, *sarcomata* have usually their seat in the uterine cavity, and seem to be an absolute bar to impregnation. Thirdly, *carcinomata* have been sometimes met with in the pregnant uterus; but these are commonly seated in the cervix, and it is usually only in an early stage of the mischief that conception can occur. See UTERUS, Diseases of.

IV. *Faults in the External Organs*.—In various ways the organs which serve for the reception of the spermatic fluid may be so affected that their copulative function is disturbed or destroyed, and the patient remains sterile.

(1) *Malformations*.—Occlusions of the labia are rare; but the vaginal canal may be impervious, firstly, from abnormal conditions of the *hymen*; secondly, from *atresia* in some part of its course; or, thirdly, from complete *absence*. It will even be found that in certain cases where the rest of the generative apparatus seems to be well-developed, a preternatural *shortness* of the canal exists in some sterile women, from whom the semen escapes immediately after it is thrown into the cavity.

(2) *Injuries*.—The injurious influences of a bad labour on the reproductive power of a woman may be found, first, in an undue *patency* of the canal, usually from extensive rupture of the perinæum; secondly, more frequently from *atresia*, partial or

complete; or, thirdly, from *fistulous* formations, leading to communication with the neighbouring cavities.

(3) *Inflammation*.—In its acute stages, inflammation of the pudenda and vagina produces, first, *dyspareunia*; in its more chronic forms it may be productive of, secondly, *unhealthy discharges*, which endanger the vitality of the spermatozoa; or it may lead, thirdly, to *occlusions* of the labia, or of the vaginal orifice or canal. Partly of inflammatory origin is the condition, fourthly, of *vaginismus*, which is not an uncommon cause of impossibility of connection.

(4) *Degenerations*.—The various neoplasms occur with rarity in the vaginal canal; but in the pudenda—sometimes from their bulk, sometimes from their sensitiveness—they interfere with connection, as in cases of elephantiasis labiorum or of urethral carcinoma. *See* VAGINA, Diseases of.

DIAGNOSIS AND PROGNOSIS.—Investigation into a case of sterility may require us to satisfy ourselves as to the fertilising powers of the male, and the due fulfilment of the marital function. Occasionally some concurrent disturbance in the functions of the sexual apparatus of the female, or of the neighbouring organs, may enable us to make a close guess at the cause of her barrenness; but we can only arrive at a true conclusion by a careful physical examination, having in view such causes as we have given. Some of the conditions, as, for example, the more pronounced malformations, or imperfect development, make us regard a woman as hopelessly sterile; others, such as uterine flexions and stenosis, some vaginal occlusions, and injuries and tenderness, we may undertake to treat with good hope of fruitful result.

TREATMENT.—In commencing the treatment of any case, we must bear in mind that morbid conditions may be present in more than one of the planes of the sexual system, and that we must begin with the removal of the obstacle that lies nearest the surface. Urethral caruncles and other sensitive structures in the vulva must be cut off or cauterised. Contractions of the vaginal orifice or canal must be stretched; and where there is complete atresia an aperture must be formed and kept patulous. Stenosis of the uterine orifices may be overcome by temporary dilatation by means of graduated bougies introduced under chloroform, or with a tupelo-tent, which the writer has more than once seen followed by impregnation. Where such dilatation fails, the os may be dilated more permanently, by tearing it with an instrument like a pair of long dressing-forceps, the blades of which are forced apart after it has been passed into the cervix; or by dividing the cervix at both sides, or in one or other lip, with a hysterotomy; or the orifice and canal may be dilated by means of graduated bougies. The deviations of the uterus must be rectified: versions, after replacement, being usually retained by some modification of Hodge's pessary, flexions demanding in addition the use of an intra-uterine stem. The stem-pessary of zinc and copper, introduced into the interior, is the best means of stimulating to its full function the imperfectly developed uterus, and the uterus which has withered from super-involution. Morbid conditions in the interior of the uterus require direct applications to its cavity. And as in a large proportion of the cases some inflammatory mischief complicates the other morbid condition, it is often helpful to the cure to make the patient use hot

douches and baths, and the internal remedies which tend to remove the effects of inflammatory action. It is to the beneficial influence which the waters of Ems, Aix, Kissingen, and other spas exert on chronic metritis that their reputation for curing sterility is mainly due. In cases where the natural method of getting spermatozoa brought into relation with the ova has failed, success is said to have followed the introduction of seminal fluid by means of a fine syringe and tube into the cavity of the uterus—a line of treatment legitimate, it may be, but only to be adopted in quite exceptional circumstances.

ALEXANDER RUSSELL SIMPSON.

STERILITY IN THE MALE.—SYNON.: Fr. *Stérité chez l'homme*; Ger. *Unfruchtbarkeit des Mannes*.

DESCRIPTION.—By sterility is meant an inability to procreate. This condition must not be confused with impotence, which is an inability to perform sexual intercourse. A perfectly impotent man is necessarily sterile, but a sterile man may be able to perform the sexual act. The condition may result from widely differing causes.

1. *Abnormalities of the Genitals*.—(a) In those cases in which both testicles are undescended or are otherwise misplaced, these organs remain small, undergoing no increase in size at the time of puberty; moreover, they do not form spermatozoa. The writer has examined a number of testicles removed by operation in this class of case, and has found them wholly destitute of spermatozoa. In these cases the patient is sterile but not generally impotent, and the fluid emitted in coition is destitute of spermatozoa (*azoospermism*). Cryptorchies are sometimes reputed to have procreated children; but it is remarkable that as yet no case has been found in which a retained testicle has been fully proved to be capable of secreting a fertilising fluid.

TREATMENT.—All cases of undescended or misplaced testicle should be operated upon as soon as possible. If the testicles are brought well down into the ill-developed scrotum before puberty they enlarge at that period, and may attain the ordinary size. As a rule, although they do not enlarge to the full extent, they grow into useful organs, sterility thus being avoided. An additional reason for operation is that the majority of these cases also suffer from herniæ.

(b) In epispadias or hypospadias, in hermaphrodites, and in many other cases of malformation of the penis, the semen is not properly introduced into the vagina, and sterility results. *See* PENIS, Diseases of.

2. *Obstruction to the emission of Semen*. In double acute epididymitis the inflammatory exudation occludes the canal of each epididymis. This condition may be only temporary, but if a fibrous nodule remain in the globus minor on each side, the ducts will be occluded, and the sterility be permanent. The treatment of epididymitis should be continued until the inflammatory exudation is quite absorbed and no induration left. Other causes of obstruction and sterility are tubercular disease and gumma of the epididymis, disease of the vas deferens, or pressure upon these ducts from without, and the removal of the epididymes or portions of the vasa deferentia. Three other common causes of sterility are stricture of the urethra, enlarged prostate and severe phimosis. A severe stricture of the urethra obstructs the flow of

semen during erection, the semen passing back into the bladder. An enlarged prostate and any disease of or operation upon the prostate may occlude the ejaculatory ducts. In phimosis of a severe type the semen may collect in the prepuce. In perineal fistulæ the semen may escape through these canals.

3. **Diseases of the Testicles.**—By interference with the functions these may prevent the formation of spermatozoa. In double orchitis after mumps the testicles sometimes atrophy and become sterile.

4. **Aspermatism.**—Even though there be no obstruction or lack of secretion the ejaculation of semen does not always follow coitus, even in persons who are subject to nocturnal emissions. This appears to arise from defective sensibility of the glans penis. In one case of non-ejaculation the nerves proceeding to the glans apparently had been destroyed by ulceration and cicatrization; in some cases the surface of the glans is converted into a mass of scar-tissue which contains no nerve-fibres. Loss of the glans penis by operation or from ulceration may thus cause sterility, although the writer has seen a case in which efficient and fertile intercourse was possible after amputation of the penis, leaving only a small stump.

5. **AzoöspERMATISM.**—A few cases have been recorded of healthy, well-formed male adults, apparently capable of normal coition, who yet proved sterile. Repeated microscopical examination of the semen has demonstrated the absence of spermatozoa. No explanation of this condition has as yet been discovered.

TREATMENT.—If any of those conditions which are amenable to surgical assistance be present they should be treated according to general principles. Phimosis, retained testicle, stricture of the urethra, hydrocele, hernia, &c., should be at once subjected to an operation for their radical cure.

A question as to the advisability of marriage may arise. Proved sterility is a legal disqualification; and even if this point be not raised, it will accord with the experience of most practitioners, that for such men to marry is usually unwise.

CHARLES GIBBS.

STERNUTATORIES (*sternuo*, I sneeze).—**SYNON.**: Errhines; Fr. *Sternutatoires*; Ger. *Niesmittel*.

Drugs which cause sneezing, and produce an increased secretion from the mucous membrane of the nose. The principal sternutatories are Tobacco-snuff, Veratrum album, Euphorbium, and Ipecacuanha.

STERTOR (*sterto*, I snore).—**SYNON.**: Fr. *Sterteur*; *Ronflement*; Ger. *Schnarchen*; *Röcheln*.

DEFINITION.—A term commonly applied to sounds in the throat resembling snoring, which occur in the apoplectic and like conditions. The writer considers the name should be extended to other sounds formed in any part of the respiratory passages or mouth by the movements of the air, under similar circumstances.

VARIETIES.—Several varieties of stertor may be recognised, as follows:—

1. **Nasal.**—Nasal stertor arises from approximation of the alæ nasi towards the septum by the ingoing air, as in the act of sniffing.

2. **Buccal.**—This form of stertor is due to vibrations of the lips, and puffings and flappings of the cheeks, during inspiration or expiration.

3. **Palatal.**—This arises from vibrations of the soft palate, whether the breath passes through the mouth or the nose.

4. **Pharyngeal.**—Pharyngeal stertor is caused by the lolling back of the base of the tongue into close contact with the posterior wall of the pharynx.

5. **Laryngeal.**—This variety is referable to vibrations of the chordæ vocales.

6. **Mucous.**—Mucous stertor is a term which may be given to the bubbling of air through mucus in the trachea or larger air-tubes.

ÆTIOLOGY.—One or more of the varieties of stertor, in varying degrees of intensity, may occur in any of the following morbid conditions, namely: Suffocation; apoplexy; epilepsy; convulsions in children; the death-agony; fractures of the skull, and concussion of the brain; bronchitis—particularly in old subjects, sudden œdema of the lungs, and large hæmorrhages into the lungs; great exhaustion; chloroform-poisoning, drunkenness, and opium-poisoning; drowning, and all conditions in which mucus or other fluid is present in the lungs; and all forms of sopor, whether natural or the result of accident or disease.

DESCRIPTION.—The general phenomena of stertor are those of suffocation.

A patient may be found lying in a state of complete unconsciousness, with a congested, turgid, and expressionless face; usually dilated and fixed pupils; insensitive conjunctivæ; a hot and perspiring skin; throbbing arteries; a full and bounding pulse; and, lastly, noisy breathing, the direct result of mechanical interference with the passage of air into or out of the lungs, whether arising from contractions of the orifices, and vibrations of the soft parts of the nose, lips, cheeks, palate, pharynx, or larynx, or from mucus or fluids in the trachea and bronchial tubes. When the obstruction to the breathing is only slight, but long-continued, the face may be of a dusky pallor, and there is an entire absence of turgidity and congestion.

PATHOLOGY.—The noise in stertor depends on the vibration of certain parts just enumerated. The primary disease causes partial paralysis or spasm of the muscles contained in these parts, and this factor, together with the position of the patient, causes the paralysed or rigid structures to occupy positions interfering with the passage of air into and from the lungs. Thus is produced a double result. In the first place, the air passing over the affected parts throws them into vibration, and thus gives rise to the characteristic sounds, and in the second the obstruction to respiration produces the characteristic symptoms of asphyxia, which may be sometimes so great as to be fatal in itself. The congested and turgid face, the noisy breathing, the râles in the chest, the throbbing arteries, and the full and bounding pulse, are signs of suffocation. Directly the impediments to respiration are removed, all is quiet in apoplexy, and the practitioner is enabled to judge of the real state of the case—which side is paralysed, whether the nerves are losing or recovering their power, and what evidences exist as to greater or less interference with the functions of organic life.

TREATMENT.—In stertor, as in strangulation, we must proceed at once to remove the impediment to free respiration.

Nasal stertor.—This may be relieved by pressing upwards the tip of the nose, or by keeping the nares open by the handle of a common salt-spoon.

Laryngeal stertor.—In apoplexy this never appears dangerous enough to warrant tracheotomy, which alone would remove it.

Buccal, pharyngeal, palatal, and mucous stertor.

—These varieties of stertor are readily treated by placing the patient comfortably on one side, and affording support by well-arranged pillows. In this position the buccal and palatal stertor, if any remain, will be too feeble an impediment to require further attention. The tongue drops to the side of the pharynx and leaves plenty of room for the in-going air. The mucus or fluid, too, whether resulting from these or other forms of stertor, drains away into the lowermost lung, thus preventing the formation of large foam-vesicles in the trachea (the 'death-rattles'), which are always dangerous respiratory impediments. Care should be taken to keep the neck rather straight, as, if the chin be brought too near the sternum, the thyroid cartilage presses upwards and backwards, and pushes the base of the tongue towards the back of the pharynx. In the management of mucous stertor it must be observed that, after a time, varying from one day to three or four, the lower lung becomes filled with mucus, though the patient is still breathing quite placidly. If at this stage the patient be turned over on the other side, the mucus begins travelling across the trachea into the opposite lung; is caught on its passage by the in-going air; and is whipped into foam, which at once blocks up the larger air-tubes of the only lung that can work, and so instant distress and danger result. If the life of the patient be not at once destroyed, still the additional shock reduces very much the chances of ultimate recovery. Under these circumstances change of position should always be tentative, and time for some return of nerve-vigour should be allowed before it is attempted. This warning applies with equal force to all cases where mucus or fluid obstructs the air-passages, as in drowning, hæmoptysis, and bronchitis. In drowning, it may be remarked that the water on entering the lungs becomes quickly insapissated with mucus, forming a milky foam, which can only be slowly evacuated by the application of Dr. Marshall Hall's or other process of artificial respiration. See ARTIFICIAL RESPIRATION; and RESUSCITATION.

ROBERT L. BOWLES.

STETHOGRAPH (στήθος, the chest; and γράφω, I write).—An instrument for recording the movements of the chest.

STETHOMETER (στήθος, the chest; and μέτρον, a measure).—SYNON.: Fr. *Stéthomètre*; Ger. *Stethometer*.—An instrument for measuring the mobility of the chest, and of its several parts, during respiration.

STETHOSCOPE (στήθος, the chest; and σκοπέω, I examine).—SYNON.: Fr. *Stéthoscope*; Ger. *Stethoscop*.

DEFINITION.—An instrument employed as a medium for the conduction of sound, between the ear and the chest or other parts, in auscultation.

DESCRIPTION.—Stethoscopes are of various patterns. They are often made of a thin, cylindrical piece of wood, perforated throughout its length, which is of about 6 inches to 8 inches; expanded at one end to a somewhat trumpet-like extremity, for convenient application to the chest; and at the other end provided with a nearly flat, broad surface,

to which the ear can be comfortably applied. Some practitioners prefer a solid stethoscope—that is, one in which there is no central canal. Such was Laennec's original instrument. Others prefer the stethoscope to be made of metal; others, again, of vulcanite. An instrument made of cedar-wood, with a perforation of about $\frac{1}{8}$ inch in diameter, a chest-piece about $1\frac{1}{2}$ inch in diameter, and a slightly concave ear-piece $2\frac{1}{2}$ inches to $2\frac{3}{4}$ inches in diameter, is perhaps the best adapted for auscultation.

Most auscultators at the present day use the *binaural stethoscope*, which consists of a short hollow chest-piece, of an elongated conical shape, from which two flexible tubes extend, terminating in metal tubes tipped with ivory, to fill the meatus of the ear on each side. This instrument has some advantages. It can be more readily applied to different parts of the chest without the observer being obliged to adopt constrained postures; and by occupying both ears while the chest-piece is applied, it excludes extraneous sounds, and considerably intensifies the chest-sounds. One disadvantage is, that sounds produced in the mouth and throat of the patient, which would be recognised by the disengaged ear of an observer using the ordinary stethoscope, are apt to be mistaken for modified pulmonary sounds. Again, with the aid of the rigid stethoscope impulses of various kinds, cardiac or aneurysmal, not recognisable on ordinary palpation, are very appreciable by the ear, which might escape attention with the binaural instrument.

The *differential stethoscope*, which is a binaural stethoscope having the tube connected with each ear attached to a separate chest-piece, is useful in some cases of heart-disease, and for simultaneously comparing the two sides of the chest—provided the two ears of the observer are of equal auscultatory power. Finally, some practitioners prefer a single flexible tube, with an ear-piece fitting into the meatus, and a chest-piece. It has been attempted to apply the telephone, and even the microphone, to stethoscopy, but as yet without success.

R. DOUGLAS POWELL.

STHENIC (σθένος, strength).—This term is applied, first, to individuals when they are vigorous and strong; and, secondly, to inflammatory diseases, when they assume an active character, such as *sthenic pneumonia*, as distinguished from *asthenic*.

STIFF-NECK.—A popular name for muscular torticollis. See RHEUMATISM, MUSCULAR.

STIMULANTS (*stimulo*, I excite).—SYNON.: Excitants.

DEFINITION.—Any agent which increases the function of a tissue or of an organ.

The term is fast disappearing from therapeutics since pharmacology is steadily demonstrating the way in which remedies exert their action upon the body.

Used in the general sense as above defined, it includes nearly all the remedial agents in the pharmacopœia if we exclude the class Sedatives or Depressants. A very objectionable employment of the term is made in circumscribing its use as a synonym for alcohol and its preparations.

Nevertheless there are some therapeutic groups where the term is still admissible in the present state of our knowledge, and these may be briefly enumerated.

1. *Local Stimulants*, or more correctly *External Stimulants*, include the long list of agents which are employed as counter-irritants, rubefacients, vesicants, pustulants, irritants, and even caustics. These the reader will find discussed under the heading Counter-irritants.

2. *General Stimulants*.—This is a most misleading term, and if employed at all should be used as a synonym for *Cerebral Stimulants*. These are drugs which increase the activity of the brain either by their stimulation of the cerebral cells or by their power of increasing the vascular supply. The excitation of the mental functions may be so great as to cause marked exhilaration or even delirium, and since the most typical members of this group of drugs are the narcotics, when the action of the drugs is pushed, sleep or depression of the mental faculties to the extent of coma occurs.

The agents generally employed as cerebral stimulants are alcohol, tea, coffee, coca (*Erythroxylum coca*), opium, absinth, and Indian hemp. Belladonna, hyoscyamus, camphor, and ether are also powerful excitants of the cerebrum. Of course the most important of all these is alcohol, and its use in disease is one of the most serious problems that can be placed before the physician, but it must be clearly recognised that alcohol as a remedial agent in fever should not be prescribed as a cerebral or general stimulant, but as a food and as a cardiac stimulant. See ALCOHOL.

3. *Cardiac Stimulants*.—This group is of great therapeutic importance, and comprises those drugs which notably augment the force of the ventricular contractions, and at the same time markedly increase the frequency of the pulse. In theory and in practice they must be carefully distinguished from cardiac *tonics* which in a slower and more lasting manner also increase the power of the ventricle, but at the same time diminish the number of its contractions. The action of cardiac stimulants is utilised in emergencies as in threatening failure of the circulation from shock, injury, mental emotions, or from the effects of the depressing poisons elaborated during fever. These drugs act upon the heart through its nerve-supply, directly, and also by reflex action on the sensory filaments of the vagus distributed to the stomach. Ammonia acts in this manner, while at the same time it powerfully stimulates the vaso-motor centre in the medulla. The chief cardiac stimulants are alcohol in all its forms, free ammonia and its preparations, heat, camphor, turpentine, ether, nearly all the aromatic or essential oils, chloroform in small doses, and strychnine.

The indication for the use of alcohol in fevers should be sought for mainly in the condition of the heart and circulation as evidenced by the force of the ventricular contractions, the frequency of the pulse, and the heat of the extremities. At the same time its value as a food (the greater part of a medicinal dose is burned up as fuel in the system) and its power of retarding metabolism must not be lost sight of. In desperate emergencies ammonia must be relied upon, applied to the nostrils or injected directly into the veins or swallowed in combination with alcohol.

4. *Vascular Stimulants*.—Though the previous group is often spoken of as 'vascular stimulants,' this term should be strictly confined to those drugs which stimulate the retarded circulation by their action in causing dilatation of the blood-vessels, as

amyl nitrite, nitroglycerin, ethyl nitrite, sweet spirit of nitre, sodium nitrite, and to some extent alcohol—especially if administered hot—and some of the sudorifics.

5. *Spinal Stimulants* are agents which notably increase the functional activity of the spinal cord. They act mainly by stimulating the large ganglion-cells in the anterior cornua. The most powerful are strychnine, brucine, thebaine, and ammonia. A host of drugs in certain definite doses also affect the cornua similarly, but are never employed for this purpose: among these are ergot, nicotine, ether, opium, gelseminine, and even chloroform. The indications for the exhibition of spinal stimulants are seldom clearly marked save in the case of paralysis, hemiplegia, or polio-myelitis anterior acuta after all inflammatory action has subsided.

6. *Gastric Stimulants* include those drugs which act in the opposite way to gastric sedatives, but the term is misleading as it includes all the gastric tonics, the local emetics, and nearly all the substances which if applied to the skin would cause irritation (*External Stimulants*), all aromatics, and alcohol in small undiluted doses, and the various spices, sauces, liqueurs and small dishes which enter into the Russian Zarkuiska or into the Branwinsbrod of the Swedes.

7. *Intestinal Stimulants* also include the long list of so-called carminatives and the various cathartics and laxatives which the reader will find described under PURGATIVES.

8. *Hepatic Stimulants* are drugs which increase the functional activity of the liver so far as the manufacture of bile is concerned, and after their administration the amount of this fluid is always augmented. They are sometimes called cholagogues, but must be distinguished from indirect cholagogues or so-called cholagogue purgatives which simply lead to the expulsion of the bile from the body by irritating the upper part of the small intestine, which causes the hepatic secretion to be expelled before it has had time for its absorption, as in the case of calomel and podophyllum. Many of the hepatic stimulants are also purgatives, but their direct cholagogue or hepatic excitant action is destroyed or suspended when they are administered in purgative doses. They are indicated in all sluggish conditions of the liver where there is reason to believe that the secretion of bile is deficient or where the physician wishes to raise the pressure of the bile in the ducts, as in cases of mild obstruction like catarrhal jaundice. The following are the more important hepatic stimulants: dilute nitric and nitro-hydrochloric acids, ipecacuanha, sodium salicylate, sulphate, benzoate, phosphate and tartrate, aloes, rhubarb, podophyllum-resin, iridin, euonymin, leptandrin, colocynth, colchicin, baptisin, corrosive sublimate, jalap, scammony, and some arsenious compounds. It is very doubtful if calomel has any direct cholagogue action. By the administration of small doses of any of the above hepatic stimulants, followed by one large dose of a cholagogue purgative like calomel, podophyllum-resin, Glauber's or Rochelle salt, very large amounts of bile may be removed from the system.

9. *Renal Stimulants*.—This group should include all the diuretics or drugs which increase the amount of the urinary secretion, but for practical purposes it is best to use it as a synonym for the stimulating diuretics. These are drugs which act

locally or directly upon the kidneys or renal cells without markedly raising the general blood-pressure. The most important members of the group are: Broom, buchu, copaiba, turpentine, caffeine, juniper, gin, calomel, urea, potassium-salts, uva ursi, nitrites, &c. Their use is limited for the most part to the treatment of diseased conditions of the bladder and urethra, or as agents for the relief of dropsy or anasarca where the kidneys are sound. In exerting their diuretic effects the urine is so altered in its chemical composition that most of these agents act as vesical sedatives and allay irritability of the bladder and urethra, or counteract the dangerous evils of the cystitis which accompanies obstructed flow, as in the senile enlargement of the prostate gland.

WILLIAM WHITLA.

STING: STINGING PLANTS AND ANIMALS.—SYNON.: Fr. *Aiguillon*; Ger. *Stachel*.

DEFINITION.—A sting is an abnormal sensation, partly painful, and partly itching in character, usually caused by the introduction beneath the skin of some poison of animal or vegetable origin. Either increment of the sensation may predominate; the stung surface may be simply painful and tender, or the itching may be intense, and lead to considerable scratching for its relief.

In the widest acceptance of the word, the effect produced by the application to the skin of such substances as mustard, cantharides, strong carbolic acid, and the like, may be denominated as stinging; but here it is proposed to consider only the wounds inflicted by stinging plants and animals. The subject of venomous animals is separately discussed. See **VENOMOUS ANIMALS**.

ETIOLOGY.—The severity of the sensation and of the local constitutional effects of stinging depends, not only on the quality and quantity of the irritant, but also on individual susceptibility; in some persons the effect may be extremely mild and transient, in others severe symptoms may ensue. In respect to the peculiar susceptibility of the person attacked, the greatest difference is observable among individuals, and even in the same person at different times. In many of the recorded fatal cases, the victim had been previously stung and had suffered severely. There seems, however, reason to believe that the system becomes more resistant to the effects after repeated stings, as is seen to be the case among bee-keepers, and those continually exposed to mosquitoes. Some variation in the virulence of the poison, whether of animal or plant, occurs with the season of the year.

Stinging Plants.—These are almost entirely limited to the order *Urticaceæ*, of which the following species are the most important: *U. urens*, *U. dioica* (British), *U. crenulata*, *U. stimulans* (Indian), *U. ferax* (New Zealand), *U. gigas* (New South Wales), which forms lofty trees, and *U. urentissima* (Java). A few species of the order Malpighiaceæ also possess stinging properties.

In the nettles the urticating organs consist of unicellular hairs tapering towards the free end, each of which terminates in a bent knob, and, swelling out at the attached extremity, is received into a cup-shaped depression of a cellular pedicle. The acrid fluid, the nature of which has not been determined, but is supposed to be an acid similar to malic or acetic acid, is regarded as being secreted by the pedicle and stored in the stinging hair, from which

it escapes into the integument when the brittle knobbed tip is broken off by contact. The hairs in the Malpighiads are peltate and not tapering.

Stinging Animals.—Urticating organs, known as *trichocysts*, *cnide*, or *thread-cells*, similar in function only to the stinging cells of nettles, are found in many animals, such as the Infusoria, some Annelida, and several Nudibranchiate mollusca. They are, however, best developed and most characteristic in the Cœlenterata, of which the jelly-fishes or sea-nettles are the best known. These organs consist of cells, containing an acrid fluid, and prolonged into a long filament which presents numerous modifications of barbs and serrations. The filament is usually spirally coiled within the cell, from which it is everted on contact, conveying the fluid into the surface that it penetrates. Great variety exists in the form, size, and disposition of these organs; in many of the Actinozoa or sea-anemones they are arranged in rope-like clusters, enclosed in fine tubes, within the body-cavity.

The power of stinging is possessed very generally by members of the articulate sub-kingdom, such as spiders and scorpions among the Arachnida; bees, wasps, mosquitoes, gnats, and ants among the Insecta. The bite of the flea or bug produces itching rather than a sting. In all cases of true stinging an irritant fluid, thought to be of the nature of formic acid, is introduced beneath the skin by some penetrating organ, which may be connected with the mouth or with the terminal segment of the abdomen, and is in some animals—as bees, but less frequently wasps—torn out and left in the wound when the sting is inflicted.

EFFECTS.—The introduction of the poison of a stinging vegetable or animal is followed, either immediately or within a very short time, by erythema of the affected part, the surface being red, swollen, and exhibiting all degrees of pain and tenderness. If a mucous membrane, as of the mouth, be the seat of the wound, the swelling is intense, the tongue cannot be protruded, and swallowing becomes difficult or even impossible. Nor are the results limited to the locality of the sting, the erythema often spreading to a considerable extent, from the hands or face, which are obviously the most frequent starting-point, to the arms, neck, and trunk, sometimes presenting the appearance of a scarlatiniform or urticarial eruption. Associated with the local manifestations, general symptoms, often of a most severe and even fatal character, have been known to occur. Well-authenticated cases of death from the stings of bees, wasps, scorpions, and even some species of tropical nettles, have been placed on record. In the majority of such cases the poison has brought on a state of syncope; severe prostration, pallor, and pulselessness being the most general symptoms; and death has been known to occur within a quarter of an hour, or from that to a few hours. Other cases, especially of scorpion-stings, are characterised by excitement and some delirium; and tetanic spasms even sufficient to cause death have been not infrequently known to follow the stings of bees, wasps, and spiders (see **TARANTISM**). When the case does not prove fatal, recovery is generally rapid and the patient is quite well in a day or two; but this is not always so, and the effects of some nettles, for example, *Urtica urentissima*, the Devil's Leaf of Java, are said to last for years.

TREATMENT.—(a) *Local.*—Innumerable applications have been suggested as specifics in cases of

stinging. Their efficacy, without doubt, depends mainly upon their being applied to the seat of the sting quickly after infliction. The *modus operandi* of many is quite empirical, but others would seem to decompose the irritating material, and so prevent or arrest its effects. Among those most generally resorted to are alkalis, such as liquor ammoniæ or strong solutions of bicarbonate of sodium or potassium; and it is probable that many popular remedies, such as soap, the 'blue-bag,' &c., depend for their effect upon the alkali they contain. Concentrated solutions or even the pure crystals of alum, and tartaric or other vegetable acids, often afford distinct relief if rubbed in at the site of the sting. The bruised leaves of plants such as the dock, or the juice of a raw onion, applied similarly, will act in the same way. For the more serious stings of tropical plants and animals, more potent remedies are necessary, such as the application of lint soaked in chloroform and laid over the wound; poultices of ipecacuanha, or preferably an extract of the same drug made by mixing equal parts of powdered ipecacuanha, rectified spirit, and ether; or rubbing the part with hydrate of chloral—pure, or liquefied by mixture with camphor, in the proportion of three parts of the former to one of the latter. Menthol-camphor will also relieve the pain and irritation; carbolic oil (1 in 20), or even subcutaneous injections of carbolic acid, have been used. The aromatic oils of pennyroyal, lavender, cloves, or cinnamon smeared over the hands and face, are most effective in preventing the attack of mosquitoes, gnats, &c., and often give relief when applied to the stung part.

In the case of bee-stings it is important to search for and remove any parts of the penetrating organ which may have been left in the wound.

(b) *General*.—When symptoms of prostration or collapse are produced, stimulation with brandy, ammonia, or ether is an absolute necessity, and one or other of these should be administered without stint, as recovery undoubtedly depends on counteracting the cardiac depression.

W. H. ALLCHIN.

STITCH.—A sharp catching pain in the side, generally associated with pleurisy, pleurodynia, or intercostal neuralgia. See PLEURA, Diseases of.

STOKES-ADAMS DISEASE. — See ANGINA PECTORIS; and BRADYCARDIA.

STOMACH, Diseases of.—The diseases of the stomach will be considered in the following order:

- Clinical Examination of, p. 1588.
- Atrophy and Degeneration of Glands of, p. 1592.
- Cirrrosis of, p. 1593.
- Concretions in, p. 1594.
- Congenital Pyloric Hypertrophy of, p. 1594.
- Dilatation of, p. 1595.
- Functional Disorders of, p. 1597.
- Inflammation of, p. 1602.
- Malposition of, p. 1607.
- Morbid Growths of, p. 1608.
- Neuroses of, p. 1612.
- Perforation of, and its Results, p. 1619.
- Ulceration of, p. 1621.

The following articles on cognate subjects appear in alphabetical order in other parts of the Dictionary: ABDOMEN; ALIMENT; ANTACIDS; APPETITE; CARMINATIVES; DIET; DIGES-

TION; EMETICS; EPIGASTRIC PAIN; FLATULENCE; FOOD, POISONOUS; FORCIBLE FEEDING; HÆMORRHAGE; LAVAGE; NAUSEANTS; PAIN IN VISCERAL DISEASE; PEPTONISED FOOD; PERSONAL HEALTH; PHYSICAL EXAMINATION; STIMULANTS; VOMITING.

STOMACH, Clinical Examination of the. The subject will be considered under the headings of (1) *Physical Examination*; and (2) *Examination of the Functions*.

1. **Physical Examination**.—The shape and relations of the stomach are shown on p. 1242. Its average capacity is from 35 oz. to 40 oz.; a capacity of three pints is to be considered as pathological.

Physical examination of the abdomen gives no sign when the stomach is normal and undistended. After a large meal, more especially with much liquid, the organ forms, in some cases, a distinct mass in the epigastric region, the borders of which, however, cannot be defined. The peristaltic movements of the healthy organ are never visible, nor can they be rendered visible by manipulation. Succussion of the normal stomach produces no splashing. As digestion proceeds and finishes, the organ empties itself, and the prominence of the epigastrium disappears.

In disease, a physical examination must be directed to determining the size, both before meals and after meals, as well as alterations in position of the organ.

The body of the enlarged stomach sometimes forms a globular tumour in the front of the abdomen. It may be situated in the epigastrium, or in the umbilical region, as in cases of gastroptosis. Palpation of the tumour reveals an elastic mass, of which the upper edge cannot be defined; although in some cases the greater curvature may be felt as a transverse line of resistance below the umbilicus. Visible peristalsis may be observed in this tumour, either spontaneous, or elicited by friction with the palm of the hand. The wave of contraction passes downwards from the left hypochondrium towards the umbilicus, and in the course of its progress, which is slow, the tumour may appear divided into two or more globular masses. Visible peristalsis, in the position and of the character above stated, indicates dilatation of the stomach secondary to stenosis of the pylorus or duodenum. It is only evident when the stomach is distended with gas or liquid, or both.

A succussion-splash may be brought out by palpation, either below the left hypochondrium, on a level with the umbilicus or to the left of the umbilicus. The succussion-splash may be absent in cases of dilated stomach, when the organ is empty and flabby. When present, it shows the presence of dilatation of the organ, but it is no guide to the degree of dilatation, since it may be obtained in cases of myasthenia, with only slight dilatation, as well as in cases of great dilatation, due to pyloric obstruction. In cases of myasthenia (atony), the occurrence of this physical sign in relation to the meals is of importance. It is usually absent before meals, and is observed best from two to three hours after a meal. In advanced cases of dilatation of the stomach it is often constantly present, but the stomach may be so distended that no splash can be obtained. The sign is elicited by placing the left hand to the right of the umbilicus, and the other to the left, slightly below. A sudden jerk of the right

hand will cause the splashing, which may be elicited by the patient himself if he suddenly contracts the diaphragm.

Pulsation in the epigastrium may be present in dilatation of the stomach, and, if the organ is full of liquid, a systolic epigastric thrill may be felt. The sign is, however, of no great importance.

Percussion by itself is of no value in the diagnosis of stomach-conditions. A tympanitic note is obtained over the enlarged stomach distended with gas, but neither the size of the organ nor the existence of dilatation can be diagnosed by this means, inasmuch as the transverse colon, when distended with gas, may occupy the epigastrium, pushing the stomach upwards. In normal conditions, a tympanitic stomach-note is not obtained in the axillary region. When the stomach is dilated and distended with gas, a tympanitic note is obtained in the left axillary region, backwards as far as the posterior axillary line, and upwards as far as the fourth rib, and over this area a bell-sound may frequently be heard by auscultatory percussion. The presence of a tympanitic note in the axillary region is usually only a temporary condition. A permanent stomach-note may, in some cases, be present in this region, as when the fundus of the stomach is adherent to the diaphragm, and is dragged upwards by chronic disease of the left lung and pleura, as in some cases of pulmonary tuberculosis. The presence of gas in the stomach is a great aid to the diagnosis of its size, and, for the purposes of diagnosis, it may be necessary to distend the stomach artificially. This may be done, either by pumping in air through a stomach-tube, or more readily by making the patient drink fifteen to thirty grains of citric acid dissolved in half a tumbler of water, and, immediately afterwards, the same amount of bicarbonate of sodium dissolved in a similar amount of water. The carbonic acid evolved distends the organ and facilitates examination.

Auscultation is of no value by itself in the recognition of enlargement of the stomach. The splashing of fluid may be heard, and if the patient be given some water (2 or 3 oz.) to drink, the liquid may be heard to fall into a dilated stomach distended with gas, with a characteristic sound. This is not a sign, however, of much value.

More is learnt by a combination of auscultation and percussion, that is, by placing the end of the stethoscope over the stomach-area, below the left hypochondrium, and either percussing or tapping with two coins in radial lines below the stethoscope. If the stomach contains gas, the area over which the percussion is heard, or the bell-sound, gives a very fair idea of the size of the organ. By this means, too, in many cases, as when the sound is obtained from the axillary region, distension of the colon may be distinguished from dilatation of the stomach.

Examination for Tumours of the Stomach.—The commonest tumour of the stomach is situated at the pylorus, and, in not a few cases, this can be recognised through the abdominal wall. The tumour may be fixed behind the liver, and only appear below the edge, or it may be felt as an indefinite thickening to the right, and above the umbilicus. In cases where the pyloric tumour is freely movable, it may be from time to time detected, even if the patient is lying on the back, but a useful method of examination, in such cases, is to palpate the abdo-

men with the patient lying on the left side, with the knees drawn up. The movable tumour then tends to fall downwards and to the left, and can readily be felt. A stomach distended with liquid or gas may push the tumour up behind the liver, so that emptying the organ may be of value in the examination. The first segment of the right rectus muscle is frequently a bar to a recognition of tumours in the upper part of the abdomen. Sometimes patience and a little manipulation will overcome this difficulty, but, in other cases, it is necessary to give the patient an anæsthetic in order to make a complete examination. Tumours of the cardiac end are sometimes felt below the left hypochondrium, and the contracted stomach, in diffuse carcinoma, may be felt as a pyriform, or oval tumour, across the epigastrium.

Internal Examination of the Stomach.—Distension of the stomach artificially with gas has already been described as an aid to the diagnosis of the size of the stomach by external examination. The amount of liquid which the stomach can contain is also a valuable aid in the diagnosis. This is, in some cases, simply gauged by the large amounts of liquid matter (over three pints) which the patient will bring up at one time; but, in other cases, it has to be determined by putting liquid into the stomach and regaining it. This is done through a soft stomach-tube, with a syphon-arrangement. Water, which has been boiled, is slowly added through the funnel, to the amount of three or four pints, the amount being gauged by the sensations of the patient, any attempt at vomiting or sensation of great abdominal distension being an indication to stop the filling. The liquid must now be syphoned off, and it may be found that more is obtained than was put into the stomach, making the total capacity of the stomach over three pints. All the liquid cannot be regained by syphoning, but most of it can be made to return by placing the patient on the left side, and expressing the contents of the stomach by manipulation of the abdomen from right to left. In some cases of hour-glass stomach where physical signs show a dilated organ, much less liquid may be obtained by syphonage than was put in. This occurrence suggests the diagnosis of hour-glass contraction.

Electric illumination of the stomach by means of the electrophane is not of great value in the diagnosis of the size of the stomach, as this can usually be detected by other means. It does, however, show the size of the organ, and may prove of value in the diagnosis of unusual conditions, such as hour-glass contraction.

2. Examination of the Functions.—The functions of the stomach are divided into (1) the movements; (2) the absorption of its contents; and (3) the chemical processes occurring in its interior.

In some cases it is necessary to determine the capabilities of the stomach by means of a test-meal. It is not of much value obtaining the gastric juice by artificial means, such as the injection of ice-cold water into the stomach, inasmuch as what is required to be known is the activity of the stomach during the digestion of a meal. Ewald's test-breakfast consists of 35 grms. of bread and one-third of a litre of water; it is of value in determining whether the gastric juice is secreted in response to the stimulus of food, but it is not of much value in determining the prolonged activity of the functions of the organ. A better meal is one consisting of about 5 oz. of beefsteak, 2 to 3 oz. of bread, and

half a tumblerful of water at the end of the meal. The patient is allowed to rest after the meal, and, in four to five hours, a portion of the stomach-contents is removed; this shows disintegration of the muscle-fibres, and the presence of hydrochloric acid and peptones. In six or seven hours the digestion ought to be completed. Such a test is of importance in certain cases of prolonged indigestion of food, where there is great diminution of function of the stomach (see p. 1600). It may also be of service in early cases of carcinoma of the stomach, for the determination of the amount of hydrochloric and organic acids present, as well as in neuroses of the organ.

(1) *Test for the Mechanical Power of the Stomach.* The test-meal serves as a guide to the mechanical power of the stomach, but other methods have been devised, none of which are of much value. (a) *Salol-test.* Salol is decomposed by the pancreatic juice into salicylic acid and carbolic acid. The salicylic acid is excreted in the urine, and can be recognised by the reddish-violet colour produced by the addition of a solution of ferric chloride. To test the mechanical power of the stomach fifteen grains of salol are given in a gelatine capsule, just after the mid-day meal. In healthy individuals, the salicylic-acid reaction appears in the urine in thirty to ninety minutes. Delay in the reaction has been ascribed to diminution of the mechanical power of the stomach, or to stenosis, owing to the drug not being expelled through the pylorus. It is evident that, for the test to be of any value, it is necessary to suppose that the pancreas, the intestinal mucous membrane, and the kidney are performing their functions in a normal manner. (b) *Oil-test.* It has also been attempted to test the mechanical power by means of 100 grammes of oil, placed in the stomach through the tube. In two hours the stomach is washed out, and the residue of oil estimated. Seventy to eighty per cent. of the oil ought to be discharged into the duodenum in two hours under normal conditions. This is not a method, however, which is applicable clinically.

(2) *Testing the Absorptive Power of the Stomach.* This has been attempted by means of iodide of potassium, three grains of which are given in a gelatine capsule, three ounces of water being drunk afterwards. The iodide can be detected in the saliva in about ten minutes, by adding a little fuming nitric acid, which sets free the iodine, which may be dissolved in chloroform, or turned blue by the addition of starch. The delay in absorption was found to be great in dilatation and carcinoma of the stomach, but less in chronic catarrh and in ulcer. The fallacy of the method, however, is that it is only a test for the absorption of iodide of potassium, which undergoes but little change during its passage through the mucous membrane. It is not a test of the absorption of proteids or sugar by the stomach, as these substances undergo a transformation while passing through the mucous membrane; their method of absorption being totally different from that of iodide of potassium.

It may be affirmed that not much advantage clinically is obtained by these methods of testing either the mechanical power or the absorptive capacity of the stomach. Diminution of absorption is seen in the retention of liquid in the organ; diminution of mechanical power is shown by dilatation. The best method of determining alteration in these functions is by means of a test-meal.

(3) *Examination of the Stomach-Contents and of Vomited Matters.*—The examination of these liquids frequently gives very valuable information regarding the process of digestion in the diseased stomach. The presence of undigested food long after a meal has been taken shows deficiency in function of the organ, and may indicate some degree of obstruction in the pylorus or duodenum. A diminished quantity of hydrochloric acid is an indication of diminution of function, and the determination of this diminution is important in gastric catarrh, in atrophy of the glands, and in cancer. An increase in acidity, due to hydrochloric acid, is a sign of irritation of the stomach, and occurs in functional disorder, in some cases of ulcer, in some cases of non-malignant obstruction of the pylorus, and in obstruction of the duodenum. The presence of mucus indicates irritation or catarrh; the presence of pus indicates an abscess in the walls of the stomach, or one perforating the coats from outside. The presence of blood indicates either a local condition, such as gastric catarrh, ulcer, or carcinoma, or cirrhosis of the liver, or a general condition, such as occurs in the profound anæmias, and in some specific febrile disorders. The presence of a large amount of organic acids (lactic, butyric, acetic), as well as that of micro-organisms, shows bacterial decomposition of the stomach-contents, the most frequent causes of which are prolonged gastric catarrh and obstruction of the pylorus.

The stomach-contents must be examined for: (a) the presence of undigested food; (b) the presence of micro-organisms; (c) the presence of mucus, pus, and blood, and fragments of new-growth; (d) the total degree of acidity; (e) the character of the free acids present (hydrochloric, lactic, butyric, acetic); (f) the presence of the products of digestion (albumoses and peptone); (g) the presence of pepsin and the curdling ferment. The methods which will be described are those easy of performance, and which can be done as ordinary clinical tests.

(a) *Undigested food* must be examined microscopically to determine the amount of digestion of muscle-fibre and of starch, and by dilution to determine the presence of any large masses of undigested matter. This is carried out by diluting the stomach-contents with a large amount of water in a beaker; after stirring the mixture and allowing it to settle, the supernatant liquid is poured off, and the sediment again washed, until only the largest particles are left. In this way it may be found that there are large masses of vegetable-fibre in the stomach-contents long after food has been taken, and even many days after the eating of fruit or vegetables. In such a case, their presence is suggestive of pyloric stenosis. Small fragments of new-growth may also be detected in this way.

(b) *Microscopical examination for bacteria* is, as a rule, sufficient from a clinical standpoint. Cover-glass preparations, carefully dried and stained with methylene-blue, may show the presence of bacilli and cocci, or torulæ and sarcinæ. The presence of any demonstrable number of bacteria or sarcinæ in the recently evacuated stomach-contents is a sign of bacterial fermentation in the stomach. This occurs in cases of gastric catarrh, in some of the rare cases of atrophy of the glands, but, more particularly, in pyloric and other obstruction. Bacteria are not seen in microscopical preparations of the stomach-

contents in cases of irritation of the organ due to irregularities in diet, or in cases of ulcer. In some cases where there is no bacterial fermentation, yeast-cells may be seen, especially when bread has recently been eaten, but the cells are obviously not budding; that is, they are in the resting stage.

(c) The *mucus* present in the vomit may be derived either from the bronchial tubes and pharynx, or from the stomach itself. The difference rests in the fact that mucus from the bronchial tubes and lungs is pigmented, while that secreted by the stomach is translucent and unpigmented. Only in cases of prolonged catarrhal inflammation of the stomach is pigmented mucus seen. The mucus which comes from the stomach may be tinged with blood, showing a diffuse lemon colour or streaks of red or dark blood. Mucus is found in the vomit and stomach-contents in cases of hyperchlorhydria and catarrh, and in some cases of pyloric cancer, but a large amount of mucus is found only in catarrh; most of it is not vomited, but is passed in the motions in long strings, as the stomach-mucus is not digested in any part of the alimentary tract.

Pus is to be recognised microscopically. It is frequently present in the vomited matters as mucopus, from the pharynx, bronchial tubes, or lungs. It comes from the stomach only in the rare cases of abscesses, local or diffuse, of the stomach-wall, and is, in these cases, not mixed with mucus, as is the pus from the lungs. Cover-glass preparations are best stained with methylene blue.

Blood in the vomit may come from the lungs or the stomach, and is discussed elsewhere (see p. 620). It may, however, be noted that the guaiacum- and hæmin-tests (p. 622) are quite unreliable, and that the best test for the presence of blood, especially in a case of coffee-ground vomiting, which is the only condition difficult to diagnose, is the Prussian-blue test for the presence of iron. This is easily performed by adding to some of the black sediment in a porcelain capsule a small quantity of chloride of potassium and a few drops of strong hydrochloric acid. The mixture is heated over the flame, and becomes greenish-yellow; the addition of a few drops of solution of potassium ferrocyanide (5 per cent.) causes the development of Prussian blue, if blood is present. The patient must, of course, not be taking any preparation of iron. Other dark colouring matters in vomit (such as wine or tea) are, unlike blood, soluble in alcohol.

(d) *Determination of the total degree of acidity.*—The total degree of acidity of the stomach-contents is due to acid salts, to acids in combination with the proteids, and to free acids. The free acidity may be due to hydrochloric acid alone, or to hydrochloric acid in addition to one or more organic acids (lactic, acetic, butyric), or it may be due to organic acids alone. The acid in combination with proteids is chiefly hydrochloric acid, but organic acids, to some extent, also combine with proteids. The determination of the total acidity is done as follows: A 1-per-cent. solution of phenol-phthalein is used as an indicator; it is slightly yellow, and is turned red by alkalis. The stomach-contents are shaken up in a bottle until completely broken up: 20 c.c. are taken, and 8 drops of the solution of phenol-phthalein are added. Water is then added up to 300 c.c. 150 c.c. of this mixture is placed in each of two flasks, and

to one flask decinormal solution of sodium hydrate ($\frac{N}{10}$ NaHO) is added, until a pinkish tinge appears; the acid liquid is now neutralised, and a control estimation may be made with the other flask. The decinormal solution of sodium hydrate contains four grammes of sodium hydrate to the litre. This exactly neutralises 3.65 grms. of hydrochloric acid. The acidity may be expressed in terms of hydrochloric acid. Thus, if 5 c.c. of the decinormal solution neutralises 10 c.c. of the stomach-contents, 50 c.c. would neutralise 100 c.c. of the stomach-contents. This would be equal to an acidity of 0.1825 gramme per cent. of hydrochloric acid. The total acidity is, however, not always expressed in terms of hydrochloric acid, but sometimes as a number of cubic centimetres of the sodium-hydrate solution required to neutralise 100 c.c. of the stomach-contents.

(e) *Determination of Acidity due to Hydrochloric Acid and to Organic Acids.*

Hydrochloric Acid.—Free hydrochloric acid exists in the stomach-contents in combination with proteids (albumoses, peptone), and uncombined. Hydrochloric acid also exists as chlorides. The first two forms indicate the hydrochloric acid secreted by the stomach in the gastric juice.

Colour-Tests for Free Hydrochloric Acid.—The colour-tests which have been devised show the presence only of free hydrochloric acid, namely, that which is not combined with proteids. The two chief solutions are as follows: *Gunsberg's solution*, consisting of phloroglucin 2 grms., vanillin 1 grm., absolute alcohol 30 c.c.; *Boas's solution*, consisting of 5 grms. of resorcin, with 3 grms. of cane-sugar, in 100 c.c. of weak spirit. Both these solutions are used in the same way, and give the same result. A drop is mixed with a drop of the stomach-contents, placed in a porcelain dish, and evaporated to dryness in a water-bath. If free hydrochloric acid be present, a rose-red colour is developed, due to the formation of red crystals. As little as 0.05 per cent. of hydrochloric acid may be detected in this way. These and other colour-reactions for the presence of free hydrochloric acid are, however, of no great clinical value. As soon as the hydrochloric acid is secreted, it combines with the proteids present in the food, and the colour-tests indicate only the excess of acid which is not so combined. The absence of reaction, therefore, does not indicate that the stomach secretes no hydrochloric acid; indeed, a large quantity may be secreted, and yet its presence will not be indicated by the colour-reactions. Tested in this way, some samples of stomach-contents will give the reaction, and some will not, and, if reliance is placed on the colour-reaction alone, false deductions regarding the functions of the stomach will be made. The only serviceable plan is to estimate the total amount of free hydrochloric acid, that is, both the acid which is free, and that in combination with proteids.

Method for estimating total amount of Free Hydrochloric Acid.—Several methods have been devised; the one given here is Lüttke's. The total quantity of chlorine is first estimated, and then the quantity of fixed chlorides, after the free hydrochloric acid has been driven off at a red heat. Subtracting the second result from the first gives the amount of chlorine not in combination.

The solutions used are: (1) Decinormal solution of silver nitrate, containing 16.997 grms. of pure silver nitrate, dissolved in 100 c.c. of 25-per-cent.

nitric acid; 50 c.c. of the liquor ferri persulphatis are added, and the mixture diluted with water to a litre. (2) Decinormal solution of ammonium sulphocyanide, containing 7.6 grms. of the salt to the litre. This solution, added to the solution of silver nitrate, gives in excess a reddish colour. It must be standardised against the silver solution, so that on mixing 10 c.c. of each solution a reddish colouration just appears. The analysis is carried out as follows: To 10 c.c. of the shaken gastric contents 20 c.c. of the silver solution are added. After standing for a few minutes, the mixture is diluted to 100 c.c. and filtered through a dry filter. The precipitate on the filter contains the chlorine in combination with silver, while the filtrate contains the excess of silver solution used. This excess is estimated by taking 50 c.c. of the filtrate, and adding the sulphocyanide solution until a red colour appears. The number of cubic centimetres multiplied by two equals the number of cubic centimetres of silver solution used in excess. Thus, if 5 c.c. of the sulphocyanide solution were used, the excess of silver solution = 5×2 , and the amount of silver solution used = $20 - (5 \times 2) = 10$ c.c.

The estimation of the fixed chlorides is as follows: 10 c.c. of the gastric contents are carefully evaporated to dryness in a platinum capsule; the residue is ignited until it no longer burns with a flame, and is then ground up with 100 c.c. of boiling distilled water to extract the chlorides. The liquid is filtered, and to the filtrate 10 c.c. of the silver solution is added. The excess of silver solution used must be estimated as in the first process. The amount of hydrochloric acid is determined as follows: The number of cubic centimetres of silver solution used in the second estimation is subtracted from the number obtained in the first; multiplying the difference by 0.0365 gives the weight of hydrochloric acid present in 100 c.c. of the gastric contents. Thus, if the first estimation gives 10 c.c. and the second 5 c.c., $10 - 5 = 5 \times 0.0365 = 0.1825$ gm. per cent. HCl. The whole estimation takes about an hour to perform.

Determination of the Organic Acids present.—These are usually lactic, butyric, or acetic acid, chiefly the two former. Acetic and butyric acids are volatile, while lactic acid is fixed. The total amount of organic acid present may be roughly estimated by subtracting the acidity due to free hydrochloric acid from the total acidity. The nature of the acids present is determined in the following manner: 50 or 100 c.c. of the stomach contents are shaken up with an equal volume of ether, and after standing for a time, the ether is poured off. A second and third extraction with ether is made, and the ethereal liquids allowed to evaporate in an open dish. If the residue is acid to test-paper, it may be mixed with a little warm water, and the following reactions performed.

Test for Lactic Acid.—The test-solution is made by adding one or two drops of liquor ferri perchloridi to 50 c.c. of water. The mixture is almost colourless, but, on the addition of lactic acid, becomes yellow. Another test-solution consists of one or two drops of liquor ferri perchloridi added to 30 c.c. of 1 in 60 carbolic-acid solution. The blue liquid which results is changed to a clear yellow, or greenish-yellow, by a trace of lactic acid.

Test for Butyric Acid.—This has a pungent smell like rancid butter. To the liquid to be tested add a small quantity of alcohol and two drops of strong

sulphuric acid. On heating, there is a characteristic smell of butyric ether.

Test for Acetic Acid.—Perchloride of iron gives a blood-red colour, and the nauseous pungent smell of cacodyl is developed when potash and a little arsenious acid are added to the liquid containing it, and the mixture evaporated in a test-tube.

It is important to estimate the total amount of free hydrochloric acid in the stomach-contents in cases of hyperacidity due to excessive secretion of hydrochloric acid, and in cases where there is a diminution in the secretion, more particularly in cases of gastric catarrh and in cancer of the stomach. The presence of a large amount of organic acids in the stomach-contents, or in the vomit, shows the occurrence of bacterial fermentation in the stomach. A small amount of organic acids (chiefly lactic and butyric) is derived from the food.

(f) *Determination of the presence of digestive products.*—The presence of albumoses and peptone in a liquid coming from the stomach shows that the organ is capable of secreting active gastric juice. A necessary precaution must be observed in ascertaining whether peptonised food is being taken or not. Albumoses and peptone are usually to be found in the vomited matters, unless vomiting occurs soon after food has been taken. In cases of obstruction of the lower end of the oesophagus, the regurgitated liquid, which is sometimes in large quantity, does not contain these substances, since the food has never entered the stomach, and the examination for albumoses may thus be of great importance from the point of view of diagnosis. They are tested for as follows: If the stomach-contents are very acid, they must be neutralised by sodium hydrate, and filtered. To the filtrate a trace of Fehling's solution may be added, and an excess of liquor potassae. If albumoses are present a pink colour develops (biuret reaction). See p. 35.

(g) The determination of the presence of pepsin and of the curdling ferment in the stomach-contents is not of great clinical importance.

SIDNEY MARTIN.

STOMACH, Atrophy and Degeneration of the Glands of.—Degeneration of the glands of the stomach may be secondary to inflammation, or it may be non-inflammatory. The inflammatory conditions which lead to degeneration are the ordinary forms of gastric catarrh or gastritis, as well as toxic gastritis (due to irritant poison) and bacterial gastritis. With the non-inflammatory causes a primary atrophy has been described. Fatty degeneration of the glands also occurs in some cases of cancer of the stomach; more rarely in ulcer, and it may occur in tuberculosis. The glands may also be fatty in poisoning by phosphorus and arsenic, and in some of the profound anæmias, more particularly pernicious anæmia. Amyloid degeneration of the mucous membrane of the stomach may also occur.

PATHOLOGICAL ANATOMY.—Degeneration of the glands, seen as the result of inflammation, consists, in some cases, almost solely of fatty degeneration of the secretory cells. In other cases, however, there is an increase of interstitial fibrous tissue, and the glands atrophy partly from pressure. Cysts may be formed, but these are, as a rule, not numerous: cystic formation is usually associated with some increase of interstitial tissue. The gross injury which is done to the mucous mem-

brane by irritant poisons leads to its destruction over large areas, and to an increase of interstitial tissue. In this case there is, as a rule, no universal degeneration of the glands. In some cases, especially over the pyloric portion of the stomach, the mucous membrane is in a polypoid condition (*état mamellonné*). One part of the mucous membrane may be quite normal, and adjoining it is an area smooth, thin, and atrophied, while at another part there are projections, or polypi, varying in diameter from one or two lines to a quarter or half an inch. The chief change in the mucous membrane, besides destruction of the glands, is an increase of fibrous tissue; small cysts may also be formed.

Primary atrophy of the stomach is not a common disease, and some of the cases may probably have been mistaken for inflammatory degeneration. The walls of the stomach are greatly thinned in some cases, so as to be semi-transparent, the normal appearance being a whitish-yellow opaque mucous membrane folded into rugæ. The organ is dilated, and more or less empty, and there is no *post-mortem* digestion of the mucous membrane, there being but little gastric juice secreted. Microscopically all the coats of the stomach are seen to be affected; the glands are in an advanced stage of degeneration; in many parts absent, in other parts shown only by fatty granules.

The so-called primary atrophy, therefore, is associated with fatty degeneration of the secretory cells. This is the same with the fatty degeneration which occurs in cases of cancer anywhere in the body, in cases of tuberculosis, of pernicious anæmia, and of poisoning by phosphorus and by arsenic. In these cases there is no apparent increase of interstitial fibrous tissue.

Amyloid degeneration occurs chiefly in long-standing cases of pulmonary tuberculosis and of syphilis, and in cases of prolonged suppuration. It affects the vessels of the sub-mucous and mucous coats, and the muscular fibres and the connective tissue between the glands. The glands themselves degenerate; some of the cells being hyaline, others fatty, while the nuclei have for the most part disappeared.

CONDITION OF DIGESTION.—In all cases of degeneration of glands of the stomach there is a diminished secretion of gastric juice, both of hydrochloric acid and of pepsin, and a diminished motor activity. Not only, therefore, are the chemical processes of digestion very inefficiently performed, but the stomach is unable to empty itself. Dilatation of the organ results. The absorptive processes are also deficient.

SYMPTOMS.—The symptoms referable to the stomach in degeneration of the mucous membrane vary according to whether the degeneration is due to a chronic inflammatory process, or whether it is non-inflammatory. In the former case symptoms of irritability of the organ may be present, as shown by pain and vomiting. In the latter case the symptoms of irritation are absent, and the chief sign is an inability to take food, except in a liquid or predigested form. The symptoms due to the inflammatory degeneration are discussed elsewhere. With the exception of primary atrophy other degenerations are associated with some profound disease, or some form of poisoning, as by phosphorus or arsenic, and the general disease, such as cancer, tuberculosis, or pernicious anæmia,

to a great extent masks the changes in the stomach. Primary atrophy of the stomach occurs chiefly in patients beyond middle age. Thus, of thirteen cases recorded by various observers, six were over sixty years of age, four were between the ages of thirty and fifty, and three between the ages of nineteen and thirty. In such cases there is inability to take solid food, and there is great wasting and anæmia. It cannot be considered that, in most of the cases, the mere atrophy of the mucous membrane was the cause of death. An animal or man can exist without a stomach, and in primary atrophy of the stomach there is also probably an associated degeneration of the other digestive glands, or some general disease, such as granular contracted kidney.

DIAGNOSIS AND TREATMENT.—In many cases of so-called primary atrophy a diagnosis is not made during life. The chief disease which may be mistaken for it is Addison's disease, when there is no pigmentation. The absence of fever, the great prostration, the wasting, and the feeble pulse, are points of similarity. Vomiting, however, occurs in Addison's disease, and cases without pigmentation are rare. The treatment is by means of predigested food, and by rectal injections; in these ways the patient may be enabled for a time to take a sufficiency of nourishment.

SIDNEY MARTIN.

STOMACH, Cirrhosis of the.—**SYNON.:** Hypertrophy of the Stomach-Walls; Chronic Interstitial Gastritis; *Fibrosis Ventriculi*; Sclerotic Gastritis.

Cirrhosis of the tissues forming the stomach-wall is generally one of the later results of protracted chronic gastritis. Occasionally it would appear to occur apart from any antecedent gastritic affection. The more localised hypertrophy of the muscular coat in the pyloric region of the stomach, which is associated with stenosis and simple enlargement of the pylorus, is of a somewhat similar nature, though of different origin.

When *cirrhosis ventriculi* follows chronic gastritis, in addition to hyperplasia of the connective and muscular tissues, some atrophy of the mucous membrane is often present, a legacy from the earlier lesion. Otherwise the mucous membrane may be normal.

ÆTIOLOGY.—Cirrhosis of the stomach is by no means a common disease. According to some authorities it shows a preference for men, to others for women; the weight of evidence available inclines towards its greater frequency in males. It is a disease of the middle-aged. Brinton states the average age of its occurrence as thirty-four, and that of cancer as fifty.

Its mode of causation is unknown. Alcohol, misused, has been blamed, but the disease has attacked rigid teetotallers; traumata or irritation from gastric ulcerations have been accused of causing it. Perhaps the most probable hypothesis, provided that chronic gastritis has not previously existed, is that the excessive development of fibrous and muscular tissue forms but one among many possible manifestations of a constitutional predisposition towards fibroid change; similar to the lesions which occur without evident cause in the liver, kidney, spleen, and other organs. Many of the most severe and prolonged examples of chronic gastritis fail to develop any marked degree of hyper-

trophic cirrhosis, however pronounced the atrophy produced may be; others, without adequate cause being shown to account for the difference, proceed to hypertrophic cirrhosis under apparently identical circumstances. Usually of long duration, up to fifteen years in some instances, it may run a rapid course and prove fatal in a few months. The dividing line, however, between the later stages of all types of chronic gastritis and gastric cirrhosis is often largely hypothetical, unless the term 'cirrhosis' be only applied to those cases wherein undoubted hypertrophic and cirrhotic changes, affecting rather the submucous and muscular layers than the mucosa, exist, and to all appearance exist primarily.

MORBID ANATOMY.—As in other organs, cirrhosis of the tissues of the gastric walls implies hyperplastic increase of the connective tissue, to the detriment of the muscular and glandular elements. But unlike the common accompaniment of chronic gastritis this increase invades all the coats of the stomach-wall, except the mucosa; the outer coverings are sclerosed, the inner only affected secondarily. In time the submucosa, muscular and peritoneal layers become so gravely involved that almost all power of movement is lost, followed consequently by deterioration of digestive function, degeneration of the glands, contraction of the organ, and finally invasion of the mucosa.

SYMPTOMS.—Primary cirrhosis of the stomach presents few pathognomonic symptoms. The condition is rarely diagnosed *ante mortem*. The symptoms common to chronic gastritis are also common to it with but slight variation. Chief among these may be placed epigastric pain and tenderness; emesis; diminished gastric function, accompanying, and due to, hypochlorhydria; and in the more advanced cases hypopepsia. The capacity of the organ is small, nor can it be dilated by any of the methods employed for such purpose. Appetite fails gradually. Slight hæmatemesis sometimes occurs. The symptoms are generally closely associated with the taking of food.

The physical signs present are seldom sufficiently distinct to afford definite evidence of the true nature of the disease before an advanced stage has been reached. Then, on palpating the abdomen, a tender firm mass can be detected either lying obliquely from the left hypochondrium down to the middle line, or transversely in the lower epigastrium.

DIAGNOSIS.—Cancer of the stomach *en cuirasse*, with extensive infiltration of the walls, presents so similar a clinical picture that perhaps the only appreciable point of difference between it and cirrhosis ventriculi obtains in the duration of the disease. If the tumour persists for more than three years, it is probably of simple type. Peritoneal tumours may simulate cirrhosis of the stomach, but physical examination of the gastric contents and motility suffice to distinguish them.

Foreign bodies in the stomach in large accumulation offer some faint resemblance to this condition; usually met with in the insane and neurotics, local tests readily serve to prove the true nature of the lesion.

PROGNOSIS.—A complete cure can never be looked for where cirrhosis of the stomach-walls has become established; the earlier attention is drawn to the condition, and the sooner the patient regulates his diet and habits as advised, the greater the chance will be of checking further progress of the disease, and of assuring a fairly comfortable future for him

for some years. But in some cases rapid progress of the symptoms from the date of their first recognition tells only too clearly that no favourable result can be hoped for.

TREATMENT.—As the stomach is small, and has lost much of its motile power, food must be taken in small quantities, and be in liquid form. Milk and meat-extracts or soups are best suited for administration. The state of the bowels must be carefully attended to; no stimulants allowed. Opium is often required to soothe the distress. Otherwise treatment is symptomatic.

ALEXANDER LOCKHART GILLESPIE.

STOMACH, Concretions in.—Concretions in the stomach are composed of various indigestible substances that have been swallowed, such as hair, paper, cotton, cocoa-nut fibre, &c. They chiefly occur in idiots and lunatics. In some of the cases recorded a tumour has been observed during the life of the patient, which in one case was mistaken for a floating kidney. Concretions of this kind may give rise to perforation, but more generally they set up inflammation of the mucous membrane, followed by peritonitis. See CONCRETIONS.

STOMACH, Congenital Pyloric Hypertrophy of.—This condition, the origin of which is a matter of doubt, has only been recognised within recent years. It is met with in new-born infants, and usually consists in so great a measure of hypertrophy of the coats which combine with the pyloric sphincter to form the pylorus that the passage of the gastric contents into the duodenum is almost or wholly arrested.

The victims of this abnormality generally exhibit no sign of ill-health during the first few days of life, save that the milk ingested is rather more apt to be returned in part than normally. After an interval, of variable duration, has elapsed since birth, a few days or, it may be, a few weeks, everything swallowed is rejected either at once or from half an hour to an hour afterwards; no therapeutic measures have the least effect on the vomiting; the infant gradually grows weaker and more emaciated, and finally succumbs to inanition. *Post mortem* the only constant lesions found consist of some degree of dilatation of the œsophagus, marked dilatation of the stomach, and pyloric stenosis from hypertrophy of the muscular layer in the pyloric region, including the portion which goes to form the sphincter, and of a larger or smaller extent of adjacent stomach-wall. The cardiac end of the stomach is rarely affected.

The hypertrophy of the pyloric muscular layer is often so great, as in two of John Thomson's reported cases, that after death compression of the stomach fails to force any of its contents into the duodenum, although they readily enter the dilated œsophagus. The mucous membrane of the dilated stomach seldom shows signs of more than a variable degree of congestion—no ulcers nor hypertrophy. The morbid thickening of the pyloric and adjacent gastric coats was entirely due to pure muscular hypertrophy in Thomson's and Fitz's cases, with decrease of connective tissue; the mucous and serous membranes were unaffected. In other recorded instances of this condition the submucosa has been found to be also involved, or, again, all the coats implicated. Maier has described two varieties of congenital pyloric stenosis, a funnel- and a ring-shaped, the first-named owing its shape and name

to a more extensive infiltration of the stomach-wall than the second.

DIAGNOSIS.—A diagnosis of congenital pyloric stenosis can only be arrived at by a process of exclusion combined with some degree of conjecture. The pyloric hypertrophy is seldom if ever palpable. But whenever obstinate and continuous vomiting, resisting all medicinal or dietetic treatment, persists in infants but lately born, with progressive emaciation and enfeeblement, and with scanty stools, pyloric obstruction should be suspected.

PROGNOSIS.—The prognosis is hopeless as regards ultimate recovery, unless the hypertrophied pyloric section be removed by surgical interference. A successful result after such an operation will depend greatly upon the extent to which the muscular hypertrophy has invaded the stomach-wall.

TREATMENT can only be palliative, unless it be feasible to employ surgical measures.

ALEXANDER LOCKHART GILLESPIE.

STOMACH, Dilatation of.—Grave defects in nutrition result when the stomach is dilated, owing chiefly to the diminished digestion and assimilation of the food which is taken into it. There is delay of food in the organ, extending, it may be, over months; moreover, the food may undergo bacterial fermentation, whereby it is broken up into substances not used in the nutrition of the body.

The causes of dilatation may be either organic disease or a primary weakness of the muscular coat; in other words, it may be either obstructive or non-obstructive.

1. The commonest form of obstruction occurs at the pylorus, in the form of a stricture, malignant or non-malignant. The effects of obstruction in the duodenum are similar to those of obstruction of the pylorus. Adhesions of the stomach to a neighbouring tumour may hamper its movements; but a more common external cause of dilatation is the presence of adhesions round the pylorus, or between the stomach and liver or gall-bladder, and transverse colon and spleen, or between the left pleura, diaphragm, and stomach. Such adhesions may be caused by previous tubercular peritonitis or pleurisy, or by inflammation of the gall-bladder or perihepatitis. Local adhesions are frequently the result of chronic ulcer of the stomach. Adhesions round the pylorus may lead to dilatation without obvious obstruction, predisposing to functional weakness.

2. The causes of the muscular weakness of the stomach-wall, which may end in dilatation, are chiefly two in number. The first is the excessive work thrown on the stomach by the repetition of large meals for a number of years, the dilatation showing itself chiefly towards middle age. The second and accessory cause of this form of dilatation is excessive muscular exercise. Primary weakness of the muscular wall, known as atony or myasthenia, occurs in cases of gastric insufficiency, and is associated with anæmia, and with neuroses of the stomach. It may also follow any acute febrile disease, such as pneumonia, typhoid fever, rheumatic fever, scarlet fever, and measles. With chronic gastric catarrh, dilatation is a common symptom, and is not associated with any organic change in the muscular tissue. The dilatation following weakness of the muscular wall is of varying degree. In the majority of cases it is but slight, but, beyond middle age, the stomach may be very greatly enlarged. The distending force in dilatation is mainly the

accumulation of gas in the organ, more particularly when there is bacterial fermentation of the food.

The causes of dilatation of the stomach may be classified as follows:—

(a) *Obstructive dilatation occurs in:* (1) Stenosis of the pylorus, caused by cancer, or fibroid contraction as from chronic ulcer; or congenital disease.

(2) Pressure on the duodenum by a new-growth, or stricture of the duodenum, following the healing of a duodenal ulcer.

(3) Contraction of the pylorus by adhesions and chronic peritonitis, caused by ulcer of the stomach, tubercular peritonitis or inflammation of the gall-bladder.

(4) Contraction of the cardiac end of the stomach by adhesions, resulting from a severe left-sided pleurisy, usually tubercular and associated with disease of the lung.

(5) Hour-glass contraction of the stomach, with which may be associated pyloric stenosis, or scarring of the stomach after ulcer.

(b) *Non-obstructive dilatation occurs:* (1) As a sequel to gastric irritation.

(2) In gastric insufficiency.

(3) As a result of subacute or chronic catarrh of the stomach.

PATHOLOGICAL CONDITIONS PRESENT.—There is a change in the functions of the stomach in all cases of dilatation, but of different kinds. The amount of hydrochloric acid varies according to the cause of the dilatation. Thus, there are cases of dilatation associated with hypersecretion of hydrochloric acid (*hyperchlorhydria*), and other cases associated with a diminished secretion (*hypochlorhydria*). Hyperchlorhydria and dilatation are associated in cases of gastric irritation, but they are also observed in cases of non-malignant stricture of the pylorus, in hour-glass contraction of the stomach, and in some cases where the dilatation is caused by pressure on the duodenum, even by a malignant tumour, e.g. of the pancreas. In such cases there is no bacterial fermentation of the food. Diminution of the secretion of hydrochloric acid occurs with dilatation in cases of gastric insufficiency; in gastric catarrh; and in prolonged cases of ulcer, with great scarring and deformity of the organ. It is, however, seen most markedly in cancer of the pylorus.

In hyperchlorhydria the percentage of free hydrochloric acid found is, on the average, about 0.3; in hypochlorhydria none may be found, or only a small percentage (0.05). Bacterial fermentation is apt to occur where the secretion of hydrochloric acid is deficient.

Bacterial Fermentation of Food.—There are numbers of bacteria taken into the stomach with food, but their development is hindered by the presence of hydrochloric acid in the gastric juice. The acid does not actually kill the majority of the bacteria taken with the food. The forms of bacterial fermentation occurring in the stomach are the acid fermentations and the alcoholic; both of these affecting carbohydrates. Putrefaction, in which the proteids are decomposed, is of rare occurrence in the organ.

1. *Acid Fermentation* is of three kinds: lactic-acid, butyric-acid, and acetic-acid fermentation. The *lactic-acid fermentation* is produced by the *Bacillus acidilactici*, which is the cause of sour milk, and is found in beet, cheese, and in sour grapes. It is seen as short, thick cells, usually

united in pairs. It forms spores when grown in milk. It is aerobic, and decomposes milk-sugar, cane-sugar, dextrin, and mannite, forming a large quantity of lactic acid, together with carbon dioxide. Starch is first converted into sugar. *Butyric-acid fermentation* is due to the *Bacillus butyricus*, which is found in decaying vegetable infusions, old cheese, and in milk kept a long time. It consists of actively motile, long, slender rods, and acts upon starch, dextrin, and cane-sugar, with the production of a large quantity of butyric acid, as well as carbon dioxide and hydrogen. It also transforms lactic acid into butyric acid, with the formation of the same gases. The *acetic acid* which is found in some cases of bacterial fermentation in the stomach is probably the product of fermentation by yeast. *Sarcina ventriculi* is an acid-producing micro-organism, but its exact rôle in acid-fermentation is not known (see SARCINA). *Alcoholic fermentation*, due to the yeast-fungus (*Saccharomyces ellipsoideus*), rarely occurs in the stomach-contents. The commonest forms of bacterial fermentation are the lactic- and butyric-acid fermentations. The presence of putrefactive gases in the stomach, as well as other products of putrefaction, is almost invariably due to regurgitation from the intestine. The total amount of organic acidity of the stomach-contents, due to bacterial fermentation, may be very high (0.4 to 0.6 per cent.), the acids present being chiefly lactic or butyric, in rarer cases acetic acid. The amount of lactic acid found varies between 0.15 and 0.63 per cent. The gases eructated in cases of dilated stomach consist chiefly of carbonic acid, hydrogen, and nitrogen. These are derived from the acid fermentation and from swallowed air. Marsh gas and sulphuretted hydrogen may be present, so that the gas may be inflammable. See FLATULENCE.

SYMPTOMS.—1. Obstructive Dilatation.—The cases which occur under this heading may be divided into two groups: those due to malignant disease of the pylorus, and those resulting from the non-malignant forms of stenosis, such as that due to a chronic ulcer, to congenital stenosis, or to adhesions round the stomach. In all obstructive cases great dilatation and marked bacterial fermentation may occur: this statement, however, applies more particularly to the malignant cases, for in these there is a diminution in the amount of hydrochloric acid, whereas, in the non-malignant cases, the secretion of acid is not only undiminished, but, in many cases, actually increased in quantity. The stomach-symptoms which are due to the dilatation have, as a rule, no direct reference to the ingestion of food, although there are exceptions to this. They come on in four, five, or six hours after a meal, or only towards the end of the day, or at intervals of two or three days, and are shown chiefly in epigastric distress and pain, followed by vomiting and gaseous eructations. The pain is diffused over all the region of the stomach, and is accompanied by a hot, burning sensation immediately before the act of vomiting. The ejection of the contents of the stomach gives relief. The quantity brought up may be as much as six pints, and, in cases of bacterial fermentation, is grumous in appearance, yellowish and frothy. It may contain bile, but bile is only constantly found when there is duodenal obstruction beyond the opening of the common bile-duct. Hæmatemesis may be present (see p. 620). The condition of the undigested food varies considerably,

and washing the vomited matters in the manner previously described (see p. 1590) may show the presence of masses of vegetable fibre, which have been eaten some days before, and have been unable to pass the pylorus. Flatulence, without vomiting, is frequently a severe symptom. The presence of a greatly dilated stomach affects the circulation and respiration. Dyspnoea and increased rapidity or irregularity of the pulse may be present. In cases where the dilatation occurs more or less acutely, the embarrassment of the respiration and of the cardiac action may end in death. The appetite is diminished, or lost in cases—more particularly those of cancer—where hydrochloric acid is diminished; but, in cases of dilatation with hyperchlorhydria, the appetite may be retained, or there may be hunger. Thirst is usually present, and sometimes dry-mouth (*xerostomia*). Dilatation of the stomach affects the general nutrition, so that wasting and even emaciation follow. The loss of weight is to be ascribed to the fact that the food is not only not properly digested, but is not absorbed. This is the chief explanation of the wasting, even in cases of carcinoma, as in these cases, when the obstruction is relieved, a great gain of weight takes place. The bowels are usually constipated, but bacterial fermentation may spread along the intestinal tract, resulting in the passing of loose and offensive motions. The urine is usually scanty, and contains an excess of phosphates and of ethereal hydrogen-sulphates.

2. Non-obstructive Dilatation.—This occurs in cases of chronic gastric irritation, of gastric insufficiency or atrophy of the stomach, and of gastric catarrh. In the first group of cases the dilatation is usually associated with hyperacidity; in the last two groups, with a diminished quantity of hydrochloric acid, which is most marked in gastric catarrh and atrophy.

An appreciable degree of dilatation may be present without there being any vomiting, or any of the severe symptoms associated with obstructive dilatation, so that the dilatation is only discovered on a physical examination of the patient. Such a condition of things is constantly found in cases of chronic gastric irritation, and the symptoms are those which are described under that heading (see FUNCTIONAL DISORDERS, p. 1597). If the dilatation is excessive, especially in cases of gastric catarrh, and of prolonged gastric irritation, vomiting and flatulence may be severe symptoms. The dilatation of catarrh or atrophy may be associated with bacterial fermentation, but this is not observed in cases of gastric irritation.

PHYSICAL SIGNS.—See STOMACH, Examination of.

DIAGNOSIS.—Two points have to be considered, namely, the diagnosis of the presence of a dilated stomach, and the diagnosis of the causes of dilatation.

The presence of a dilated stomach is indicated by the physical signs which have already been described, as well as by the vomiting of large quantities of liquid. A chemical and physical examination of the vomited matters is of great importance in the diagnosis. The discovery of large masses of vegetable fibre may indicate obstruction of the pylorus. The repeated presence of bile in the vomit is against the presence of pyloric stenosis. The occurrence of blood in the vomit in large quantity as a rule indicates ulcer or cancer. As has been previously stated, the hydrochloric acid may be either increased or

diminished in dilatation, and it is of great importance, in such cases, to make a quantitative estimation of the acid. It is never increased, but gradually diminishes, in cases of dilatation due to cancer of the pylorus. A similar diminution also occurs in the dilatation of gastric catarrh and of atrophy. The presence of organic acids in large quantity indicates bacterial fermentation, and this most commonly occurs in cases of malignant obstruction of the pylorus. An excess of hydrochloric acid, without an excess of organic acids, may occur in cases of obstructive dilatation, due to any cause other than malignant stricture of the pylorus. The presence of an excess of hydrochloric acid does not exclude a diagnosis of pyloric stenosis, though it is against the presence of cancer of the stomach. Obstructive dilatation is indicated by visible peristalsis of the stomach, but this may occur in some cases of subacute gastric catarrh. Its repeated occurrence, however, is in favour of stenosis.

In deciding as to the pathological cause of dilatation of the stomach, the foregoing facts have to be taken into account, as well as the history of the case. In obstructive dilatation, the prolonged duration of the case, the presence of hyperchlorhydria, and the history of hæmatemesis some years previously, is in favour of non-malignant obstruction, while the definite origin of the illness during a period of a few months, its progressive character, its association with hypochlorhydria and with bacterial fermentation, are in favour of malignant stenosis.

PROGNOSIS.—The prognosis depends on the cause. Relief may be given in cases of cancer, but no cure is possible. In the non-malignant forms of obstructive dilatation relief, more or less permanent, is given by gastro-enterostomy, or other operation. Cases of dilatation, due to gastric catarrh, gastric insufficiency, or irritation may get well with appropriate treatment. Those associated with atrophy are, as a rule, fatal.

TREATMENT.—*Obstructive Dilatation.*—This usually leads to bacterial fermentation, and relief may be given by systematic lavage of the stomach, a soft tube being used, and two, three, or more pints of liquid being employed, such as a solution of boric acid (4 drachms to the pint), of permanganate of potassium (light pink colour), of sodium bicarbonate (3 to 6 drachms to the pint), or of common salt ($1\frac{1}{2}$ drachm to the pint). The washing out is to be done daily, either morning or evening, and preferably by the syphon-arrangement (see **LAVAGE**). In the intervals of washing out, anti-fermentative remedies may be given, such as hyposulphite of sodium (10 to 15 grains), glycerine of carbolic acid (5 to 15 minims), and creosote in one-minim doses. Great relief may be given by the lavage in cases of obstructive dilatation, but greater relief is frequently given by an operation. Thus, in cases of malignant pyloric stenosis, gastro-enterostomy may give the patient many months of comfort, instead of dire distress. It is not possible to operate in all cases, owing partly to the infiltration of the stomach-walls by the growth, or to the advanced age and degenerate arteries of the patient. In cases of non-malignant stenosis, gastro-enterostomy is the proper procedure, and has been very successful in some cases. Where there are adhesions round the stomach, the freeing of these by operation is a legitimate procedure, and sometimes leads to success in the cure of the condition; but the results are not

always favourable, and new adhesions may be formed.

Non-obstructive dilatation is to be treated on other lines. Cases associated with gastric irritation or insufficiency, or with neuroses, are to be treated generally on the lines laid down in discussing functional disorders. In some cases it is advisable, when there is great epigastric distress, to wash out the stomach each day for a week or ten days. Massage of the abdomen, and especially of the stomach, with rest, is of great advantage in the treatment of these cases. The diet must be frequently varied, according to the idiosyncrasies of the case. If there is a large amount of vomiting and epigastric pain, rectal feeding is to be adopted for a time, especially in cases of catarrh, of ulcer, and of neurosis. If there is bacterial fermentation, carbohydrates must be withheld. These may be given if there is hyperchlorhydria. The diet is to consist solely of fresh and digestible food; large quantities of liquid being forbidden. Minced mutton, minced chicken, or meat-balls are usually allowable, with thin dry toast. In obstructive dilatation, when the patient recovers from the operation of gastro-enterostomy, the functions of the stomach also recover, and, in some cases, the ordinary diet may be resumed. In other cases, the greatest care has to be exercised in the diet. The medicinal treatment is to be on the lines already laid down in functional disorder; alkalis to relieve the acidity, and antispasmodics to relieve the flatulence, are the chief remedies to be given.

SIDNEY MARTIN.

STOMACH, Functional Disorders of.—By functional disorder is meant the condition of indigestion of food, which results from a change in the functions of the stomach, or in its innervation, apart from organic disorder of the organ. The changes which occur may not be of a permanent character.

The cases of functional disorder may be divided into three classes. In all food is the direct exciting cause of the symptoms of the disorder. In the first class of cases, *gastric irritation*, food plays a very important part in the ætiology of the disorder. In the second class, *gastric insufficiency*, there is a primary functional defect in the stomach, resulting from some general condition, such as anæmia, gout, acute infective disease, or old age. In the third class, *nervous dyspepsia* or *neurosis of the stomach*, the symptoms are mainly those referable to the nervous system. All these classes of cases run into each other, and may, at one time or another, exhibit very similar symptoms. A distinction can, however, be made in the majority of cases, not only from the history of the disorder, but from the symptoms present, and from the result of treatment.

Gastric Irritation.—The symptoms in gastric irritation are, in the first instance, referable to the stomach, and are the result of irritation of the organ, all the functions of the stomach being disordered. It includes by far the largest number of cases of functional disorder, and nervous dyspepsia is really only a part of gastric irritation, with a prominence of nervous symptoms. The disorder is usually primary, but it may be associated with disease elsewhere. It may last a considerable time—even years—and shows, during this time, periods of quiescence, during which digestion is fairly well performed, and periods of exacerbation, in which the symptoms are well marked, and may be almost intolerable. In some cases it leads to gastric catarrh.

ÆTIOLOGY.—It may occur at any age; in the infant fed by the wet-nurse or with the bottle, in the child, and in adult life. Sex has no influence; the more direct cause in women being excess of tea-drinking, and in men excess of food and alcohol. Those of a nervous temperament are especially prone to the disorder, inasmuch as they are more affected by periods of worry, hard work, or excitement. Heredity plays some part, as when there is the inheritance of gout, or a nervous disposition. The chief factor in the ætiology is, however, the food, and the mode of life. Insufficiency and irregularity of food, even with much exercise, lead to the disorder, but, more frequently, there is a history of an insufficiency of exercise, with an excess of food, alcohol, or tea. It is thus met with in all classes.

With regard to the food there are several factors to be considered: the arrangement of the meals, imperfect mastication, composition of the food, chemical reaction of the food, and the effect of food-accessories. A large meal ought to be followed by a period of bodily rest, and should not be followed by another large meal within five hours. Meals eaten hurriedly, or hard work directly after a meal, may lead to the disorder, as well as large meals taken just before going to bed. Imperfect mastication is a very important factor. It may be due to toughness and bad cooking of the food, to rapid eating, to pain caused by decayed teeth, or to the absence of teeth. The last two points are frequently overlooked. Frequently a deficiency of the molars in the upper or lower jaws prevents mastication, and so leads to irritation of the stomach. Excess of food in time throws too much work on the organ, which thus has no period of rest. Eventually it leads to hypersecretion and deficiency of movement. The food must contain a certain amount of proteids, fats, carbohydrates, salts, and water, in the daily diet. An excess of meat in the diet of the well-to-do, and an excess of vegetable food in that of the poorer classes, are frequent causes of gastric irritation. Both lead to hyperacidity of the stomach-contents, not only by stimulating an excessive secretion of hydrochloric acid, but also from the fact that they contain organic acids and salts; in vegetable food there is also an excess of cellulose, which acts as an irritant. Thus, fine wheaten flour contains 0.29 per cent. of cellulose by weight, whole-meal flour 1.9 per cent., barley and rice-flour about 0.5 per cent., potatoes 0.69 per cent., spinach and cauliflower about 1 per cent., and fine oatmeal 1.86 per cent. Food must not be taken too acid, and cooking is important as regards the digestibility of food. Bad cooking makes the food tough, or does not sufficiently prepare the starch for digestion. Food-accessories, such as alcoholic drinks, tea and coffee, and rich sauces, are useful in digestion, but, in excess, they act as irritants. Alcoholic drinks, especially beer, port, sherry, hock, burgundy, and claret, delay the chemical processes of digestion, and lead to an increased organic acidity of the stomach-contents. Taken in moderation with a meal, alcohol causes a slowing of the process of digestion, but, owing to the fact that it stimulates the secretion of gastric juice, it does not produce an imperfect digestion—indeed it helps the digestion of a large meal. The effect of alcohol, however, varies in different individuals. Taken in excess, or between meals, it leads directly to hypersecretion, and many cases of hyperchlorhydria are

due directly to the taking of alcohol. The excess of secretion which it causes persists even between meals.

Gastric irritation commonly exists by itself, but it is not infrequently associated with tuberculosis, chronic malaria, and convalescence from typhoid fever, scarlet fever, measles, rheumatic fever and influenza. It may be observed in ulcer of the stomach.

PATHOLOGICAL CONDITIONS.—The pathological conditions present in gastric irritation may be divided into (1) the condition of acidity of the stomach-contents; (2) the condition of the movements; and (3) the condition of digestion. During digestion there is a secretion of very acid gastric juice, which may continue after the food has left the stomach. In some instances a large excess of acid is secreted in an hour or an hour and a half after the meal, and this continues during the process of digestion. In severer cases, however, the secretion continues after the mass of food has been expelled into the duodenum, so that, during fasting, liquids removed from the stomach are acid. The degree of acidity varies, and may be 0.3 per cent. or higher in an hour after a meal. This is the acidity due to free hydrochloric acid. In normal conditions, the acidity of the stomach-contents after a meal only gradually increases in the first hour, in which time it is about 0.1 per cent.; it increases later to about 0.2 per cent., as a rule not much higher. The continued irritation of the mucous membrane by an excess of food causes an increased secretion of mucus, which may occasionally be vomited, or, more rarely, be observed in the motions. The excessive secretion of mucus is not a feature of gastric irritation, as it is in gastric catarrh.

The conditions of the movements of the organ are of two different kinds. In some instances the hyperacidity is associated with spasmodic contraction of the stomach, which is only relieved by the expulsion of the contents of the organ in the natural way or by vomiting. In some cases there is spasm of the cardia and pylorus; a spasm leads to great distress, and may result in rapid expulsion of the contents into the duodenum. In an important and very numerous class of cases the hyperacidity is associated with a weakness of the muscular coats (myasthenia, atony). Just before the food is expelled into the duodenum, the organ tends to become flaccid, and it is unable completely to empty itself. Prolongation of the myasthenic condition leads to dilatation of the organ, which may be very great. See DILATATION, p. 1595.

The congestion which occurs in gastric irritation is, like the hyperacidity, an exaggeration of the normal condition of the organ during digestion. It renders the stomach more sensitive. Observed during life, as in one case of gastro-enterostomy for hour-glass stomach, the mucous membrane was of a dusky red colour, and slightly covered with mucus.

In cases of gastric irritation there is no bacterial fermentation of the stomach-contents. The presence of an excess of hydrochloric acid prevents the growth of the bacteria. Thus the vomit will keep for a long time without undergoing decomposition.

SYMPTOMS.—The mode of onset is usually insidious. The symptoms are at first to be directly ascribed to repeated slight indiscretions in diet, or irregularity in mode of life. The disorder is characterised by periods of only slight digestive distress, intervening between acute or subacute

attacks. *Acute gastric irritation* occurs usually as the result of very large meals or of irritating food, but most frequently it is a food-debauch, and referred to as a *bilious attack*. In such cases the symptoms are not developed for, perhaps, two or more hours after the meal. A sense of fullness and discomfort is experienced in the epigastrium, accompanied by nausea, and relief is only obtained by vomiting or by washing out the stomach. An examination of the vomited matters shows hyperacidity, due chiefly to hydrochloric acid, but also to organic acids, which have been taken in with the food. Subsequently the appetite is lost for a day or two, and loose motions may be passed. Acute dilatation of the organ may result from a single large meal. A physical examination may show the prominence of the epigastrium, due to the stomach. There is, however, little or no tenderness, but manipulation may cause eructation or vomiting. The tongue is thickly coated, and lassitude, pallor, and frequently headache follow.

Chronic gastric irritation, with subacute attacks, is a more common disorder. The symptoms, which preserve their individual features in each particular case, are both general and referable to the stomach. The general symptoms are referable to the nervous system, such as headache, palpitation, drowsiness, mental depression, sleeplessness, vertigo, and hiccup. The symptoms referable to the stomach always appear in relation to the food, coming on either immediately or one or two hours after a meal, and are most marked after the principal meal of the day, and in the evening or night. A sense of weight, fullness, and oppression in the epigastric region may last until the next meal, which relieves it. So with the pain in the chest and between the shoulders. The time of onset of the pain is dependent on the degree of hyperacidity, so that, when it appears directly after a meal, it is usually in prolonged cases, where the stomach-contents are always acid. The continuance of the pain is due to the delay of food in the organ, or to the continuance of secretion of hyperacid gastric juice after the food has been expelled. In prolonged cases the pain tends to become continuous, more especially in the front of the chest, between the shoulders, and in the lower part of the left axilla and the left hypochondrium.

Flatulence is a frequent symptom, and is not due to bacterial fermentation of the food, but to one or other of the following conditions: (1) In the middle-aged and old it may be due to accumulation of small quantities of gas, chiefly carbonic acid, which are developed from time to time in the stomach and small intestine, and are retained there. (2) It is sometimes due to swallowed air or to swallowed saliva, which is secreted in excess, and the carbonates of which are decomposed by the acids in the contents of the stomach. (3) It may be due to the regurgitation of gas from the small intestine, or of pancreatic juice, the carbonates of which are decomposed by the hydrochloric acid. This occurs in *myasthenia* of the stomach with a flaccid pylorus. In some cases it has been supposed that large quantities of gas may be discharged from the blood into the stomach and intestines, and then eructated.

The occurrence of vomiting varies in different cases of gastric irritation. When present, it is directly due to the irritating food in the stomach, to the excessive secretion of hydrochloric acid, espe-

cially in anæmic patients, or in those with excitable nervous systems. In chronic gastric irritation the attacks of vomiting are few and far between, and are due usually to some particular indiscretion of diet, such as the eating of heavy suppers. In the subacute attacks of gastric irritation, vomiting is a rule, although it may be absent. It may occur after every meal, either directly or in one or two hours. The vomiting consists of partially digested food and a little mucus. It is hyperacid, due to hydrochloric acid. This hyperacidity may not be present if the vomiting occurs directly after food, and, in the prolonged cases, the stomach-contents may be, from time to time, deficient in hydrochloric acid, although this deficiency is never so marked as in cases of carcinoma or of gastric catarrh. At first the appetite is normal; later on it is increased, and may even be voracious; it may become capricious, or may suffer marked diminution. The sinking feeling, or the sensation of want of food, which so many of these patients experience, is most commonly due to the presence of hyperacid gastric contents. It is relieved by food, the proteids of which, combining with the free hydrochloric acid, relieve the symptoms. The tongue is frequently coated, especially in the morning on waking, and there is a nasty taste in the mouth, which is clammy from mucus. Eructation of small quantities of acid fluid (pyrosis) frequently occurs, and is sometimes the cause of the coated tongue. Recurrent and severe attacks of hyperchlorhydria are frequently ushered in by salivation, or rather by gushes of saliva into the mouth, and the secretion of saliva may amount to pints during an attack; it is swallowed and then vomited. Salivation may occur before a meal, but is usually observed after meals, at the time of the greatest acidity of the stomach-contents. More rarely, and in cases of long duration, there is a greatly deficient secretion of saliva, so that the mouth is always dry (*xerostomia*). Constipation is the rule in gastric irritation, the bowels being opened once in one or two days, or a week, or only with medicine. Frequently, however, diarrhoea and constipation alternate, one or two days of looseness of the bowels being followed by constipation. Lienteric diarrhoea may also occur, coming on directly after a meal. This condition appears to be associated with irritability of the muscular coat, whereby the food is rapidly discharged into the small intestine. The urine shows a tendency to alkalinity, an excessive excretion of phosphates, and a diminution in quantity. Albuminuria is rarely present, but albumoses may be found.

Patients subject to this disorder do not waste, owing, no doubt, to the fact that the increased secretion of gastric juice does not lead to a diminution in the amount of food assimilated, but, in prolonged cases, mal-assimilation of the food does occur, owing to *myasthenia* of the stomach, so that such patients are pale, and have flabby muscles, which are irritable to tapping. Wasting is usually associated with some dilatation of the stomach and with prolonged diarrhoea, but the loss of flesh is not progressive, and there are periods during which the patient may regain even a stone of the weight lost. Cases of gastric irritation may continue for many years, with periods of remission and of exacerbation. By a proper treatment, complete recovery is the rule; through neglect permanent injury may be done. Catarrh of the stomach may result, with a profound diminution in the functions of the organ,

or permanent and great dilatation may occur, with sometimes bacterial fermentation.

Gastric Insufficiency.—This is a condition in which the functions of the stomach are diminished. It may result from prolonged gastric irritation, and occurs in catarrh and cancer of the organ, but the term is here applied more particularly to those cases in which disorder of the organ follows, or is associated with, certain diseases. The symptoms are frequently brought out by the presence of food in the organ, but irritation is not the direct cause of the primary condition of the stomach.

ÆTIOLOGY.—The general depression of function which occurs in those who lead a sedentary life, with much mental work, affects the stomach, leading to deficient activity, especially towards the middle period of life in both men and women. In old age a similar diminution occurs, leading to the taking of a smaller amount of food. In women at the menopause, and in prolonged lactation, insufficiency is frequently observed. In young adults, however, some recognisable morbid condition of the body is present. A state of fever diminishes the activity of the digestive organs, and the return of function is usually slow. Deficient motor activity, and a deficient secretion of gastric juice are both observed, for example, with typhoid fever, scarlet fever, rheumatic fever, measles, and influenza, and are frequently associated with pulmonary tuberculosis. In other cases gastric insufficiency is produced by continued hæmorrhage, whether rectal or uterine, by prolonged suppuration, syphilis, or anæmia.

PATHOLOGICAL CONDITIONS.—In response to the stimulation of food, there is deficient secretion of gastric juice, and a diminished motor activity, both functions failing before the meal is digested. This leads to great delay of food in the organ, and to subsequent dilatation. Bacterial fermentation is, however, not very common. The stomach-contents, when removed or vomited, show imperfect digestion of the food, and a deficiency of hydrochloric acid (*hypochlorhydria*), or even an absence of the acid two hours after a test-meal (*achlorhydria*, *achylia gastrica*).

SYMPTOMS.—The symptoms come on directly after a meal, and they may last the whole day, as well as during the night. There is epigastric fullness, and a sense of depression in the chest, which may be associated with dyspnoea. Flatulence is a constant symptom, and is usually associated with signs of dilatation of the organ; fluid eructations do not usually occur, and vomiting is not a symptom. Reflex symptoms are extremely common, and are due partly to the excitability of the nervous system, and partly to the condition of anæmia present. Pain and tenderness in the lower part of the left axilla are observed, associated with flatulence. Headache is common, as well as drowsiness after meals, sleeplessness, lassitude, disinclination to exertion, mental inaptitude and apathy, and a melancholy view of life. The face is often pale and anxious-looking, and the complexion muddy; the appetite is always diminished, and the tongue is broad, pale, flabby, and tooth-indented, but it is not usually coated. The foul taste in the mouth, so common in gastric irritation, is not so frequently observed in insufficiency. Constipation is the rule; the pulse is feeble, not very frequent, but regular; wasting is very common, and the muscles are flabby. Gastric insufficiency may be a permanent condition in old age, or in those who have inherited a weak digestion.

When it occurs in febrile disorders, it is highly amenable to treatment, but, if neglected, it leads to great dilatation of the organ.

Nervous Dyspepsia.—*Gastric Irritation, with Prominence of Nervous Symptoms.* See STOMACH, Neuroses of.

DIAGNOSIS.—The diagnosis of functional disease has to be made from ulcer, catarrh, and cancer of the stomach.

1. *Diagnosis of functional disease from ulcer.*—There are not a few cases of ulcer which present the features of gastric irritation, and only declare themselves by an attack of hæmatemesis. In the diagnosis the points to be looked to are the character of the pain and vomiting, and the course of the disease. Both may occur in young women. In ulcer the pain and tenderness are localised in the epigastrium, are dependent on the ingestion of food, and are relieved by vomiting. Vomiting is frequent, especially on the continued taking of an unsuitable diet. A history of hæmatemesis would make the diagnosis clear. Gastric irritation, on the other hand, presents quite different features. During its course, there may be times at which nausea and vomiting occur after food over a period of several weeks, but the vomiting is not a serious or frequent symptom, and is readily controlled by treatment. It frequently ceases, owing to the abstinence by the patient from food. The pain is not commonly epigastric, nor is it associated with localised tenderness. If epigastric pain is present, the tenderness is usually diffused, and is not so persistent as in ulcer.

2. *Diagnosis of functional disease from gastric catarrh.*—Chronic gastric irritation and chronic catarrh bear a superficial resemblance in the symptoms they produce. The epigastric pain, which is only a transient symptom in gastric irritation, is frequent and often severe in catarrh. It is diffuse and associated with one or more areas of tenderness. Vomiting is associated with epigastric pain, and, in chronic catarrh, an examination of the vomited matter is the chief aid in the diagnosis. It usually, but not always, contains an appreciable quantity of glairy mucus, but the chief sign it shows is the greatly diminished quantity of free hydrochloric acid, with but slight digestion of the food. A small amount of blood may be seen, which is never observed in the vomit of gastric irritation. There is great tendency to dilatation of the stomach in catarrh. Bacterial fermentation may occur. A test-meal may be of great service in the diagnosis of the condition. See p. 1589.

Gastric insufficiency is distinguished from catarrh by the absence of continuous gastric pain and vomiting, and by the history, which shows some coincident or precedent disease, accounting for the diminution of the functions of the stomach.

3. *Diagnosis of functional disease from cancer.*—The suspicion of cancer of an important organ arises when, in a patient of middle age or beyond, there is progressive wasting, weakness and anæmia, without fever, and when there is no sign of wasting disease elsewhere, such as tuberculosis, chronic renal disease, and diabetes. Such symptoms during a definite period are observed in cancer of the stomach, and are associated with symptoms and physical signs referable to the organ. The symptoms which more particularly separate cancer of the stomach from functional disease are, in the early stages, the progressive loss of appetite and of weight, the presence of pain in the epigastrium, and

the occurrence of vomiting, with flatulence. The pain may at first be referable to food and may be relieved by diet; later it has no particular relation to the food. An examination of the vomited matter shows a diminution of the amount of free hydrochloric acid, an excess of organic acids, especially lactic acid, and the presence of bacteria and sarcinæ. When first seen the patient may present physical signs of dilatation of the stomach only (q.v.), or dilatation with an epigastric tumour. If dilatation alone be found, the diagnosis from functional disease is to be made mainly from the history, as in cancer there is a definite period during which the symptoms have lasted—a period, say of six or eight months—while in functional disease with great dilatation the history is that of years, and the wasting is not a feature as in cancer, although it may occur in cases of long standing. Treatment may also aid the diagnosis, as a great gain of weight, sometimes over fourteen pounds, may follow successful treatment of functional dilatation.

TREATMENT.—The treatment is both medicinal and dietetic, aided by massage and washing out the stomach. The conditions to be treated in functional disorders are hyperchlorhydria and hypochlorhydria, a diminished motor activity of the organ or occasionally spasm, and a varying degree of hyper-excitability of the nerves, associated with congestion. Restoration of the functions of the organ is a matter of time and treatment, but it does occur, except in cases of pronounced nervous dyspepsia. Powerful remedies and treatment do more harm than good. In the dietetic treatment, the objects are to give the patient, during the day, only such food as the stomach can digest with the least discomfort and to remove irritant articles from the diet. Food is to be reduced in quantity, and only freshly cooked digestible food is allowed. Articles of diet containing a large quantity of organic acids or salts, such as beef, beef-tea, many vegetable foods and fruits, are to be disallowed, as well as those containing an excessive amount of cellulose and of fat. Most important in the treatment is the removal, in the case of irritation of the stomach, of food-accessories, such as alcoholic drinks, strong tea, and coffee. Meals are to be regularly taken and not later than four hours before going to bed, and attention to the teeth is of the highest importance.

1. *Gastric irritation.*—In one class of cases there is great discomfort after food, with referred pains in the chest, irregular vomiting of very acid fluid, and flatulence. There is hypersecretion of hydrochloric acid and hyperæsthesia of the organ. The administration of acids, and in most cases of bitters, increases the discomfort. Alkalis are given as antacids, but are not given in sufficient doses to completely neutralise the acid contents of the stomach. They are best given with some sedative, such as bromide of potassium (5 to 10 grains), iodide of potassium (2 grains), cocaine hydrochloride ($\frac{1}{10}$ to $\frac{1}{5}$ of a grain), dilute hydrocyanic acid (3 minims), liquor morphinæ hydrochloridi (5 to 10 minims), or glycerine of carbolic acid (5 to 10 minims). It is usually best to begin with an alkaline mixture, containing 15 grains of bicarbonate of sodium, 3 minims of dilute hydrocyanic acid, and 5 minims of spirit of chloroform, to which may be added, in some cases, 10 grains of carbonate of bismuth. This mixture is to be taken half an hour after breakfast and after the midday meal, and on going to bed. It may also be taken once in the night,

if there is discomfort and insomnia. The administration of pepsin and other digestive ferments is of no avail. As regards diet, all alcoholic drinks are to be disallowed, as well as green vegetables and fruit. The patient is to live chiefly on a diet of milk, white bread, milk puddings, fresh fish, mutton and chicken. If the symptoms are severe, lying down is of great importance, and a further reduction in the solid articles of diet, which is then to be restricted to milk, prepared foods, eggs and bread.

If the condition has lasted a long time, especially in those cases in which there is wasting and flabbiness of the muscles, a period of four to six weeks' rest, with abstention from work, is necessary, and this is to be combined with careful dietetic and medicinal treatment, and with massage.

During recovery from gastric irritation, the first symptom to disappear is the discomfort after food, and the last is the constipation, which may last for some months after the gastric symptoms have abated.

2. *Gastric insufficiency.*—Although, in this condition, the treatment of the digestion has to be carried out carefully by means of the diet, yet a tonic treatment is essential, in order to improve the general condition. Such tonic treatment may be, in mild cases, limited to the prescription of fresh air, with a mixture containing a small dose of sulphate of iron and sulphate of quinine, or of dilute nitro-hydrochloric acid with nux vomica. Small quantities of alcohol may have to be allowed, and the diet is to consist solely of digestible articles. In pronounced cases, occurring after infective disease, general massage is of great service, especially if there is well-marked muscular weakness of the organ.

3. *Nervous dyspepsia.*—The treatment is one of extreme difficulty, and cases present an infinite variety of aspect; the effect of treatment is but slightly noticeable at first. Great idiosyncrasy regarding diet is observed, and, as a rule, it may be said that an exclusive milk-diet is not very serviceable. Thus it is found that in some cases with persistent vomiting the condition is not relieved by a diet of peptonised milk, nor even by rectal feeding; but is benefited by the prescription of readily digestible solid food, such as pounded fish, minced chicken, or mutton in small quantities at frequent intervals. Some patients, with severe epigastric pain or repeated vomiting, have to be treated, in the early stages, by rectal feeding; in others, daily washing out of the organ appears to be of service, but this is a practice which is frequently adopted by such patients, and continued for years, leading to a persistence of the symptoms. No special rules can be laid down for the medicinal treatment of such patients. They show intolerance of some drugs, as they do for articles of diet. A stomachic sedative is, however, usually required: such as cocaine hydrochloride in pill ($\frac{1}{10}$ to $\frac{1}{5}$ of a grain) or solution of hydrochloride of morphine (5 to 10 minims), with bicarbonate of sodium (10 grains). There may be intolerance either of acids or alkalis, and sometimes a tonic mixture of quinine and iron does most good. When the symptoms are pronounced and have lasted for a long time, and when there is well-marked insomnia and wasting, the only treatment available is complete rest in bed for four to six weeks, with massage and careful dieting. Even if improvement occurs, relapses are extremely frequent.

SIDNEY MARTIN.

STOMACH, Inflammation of the.—SYNON.: Gastritis; Gastric Catarrh.—The various forms of gastritis may be arranged as in the following scheme:—

A. Acute—

1. { Simple.
Toxic.

2. Phlegmonous { General.
Localised.

B. Subacute.

C. Chronic—

1. Simple.

2. Toxæmic.

3. Cirrhotic { General.
Localised.

D. Tubercular.

E. Syphilitic—

1. Gummatous.

2. Ulcerative.

A. Acute Inflammation.—1. *Acute Gastritis* (simple and toxic). SYNON.: *Gastritis glandularis acuta*; Acute Gastric Catarrh; Fr. *Gastrite aiguë*; Ger. *Gastritis akute*.

DEFINITION.—An acute inflammation of the coats of the stomach, especially of the mucous membrane, resulting in disturbances of function and alterations in form.

ÆTIOLOGY.—Acute gastritis may arise from direct local irritation of the tissues involved, or may be secondary to lesions of other parts of the body, general or localised.

Primary causes may be classified as: (1) Dietetic, (2) Toxic, (a) Organised, (b) Chemical.

Secondary causes are: (1) Acute Infectious Diseases, and (2) Constitutional Conditions (Gout, Rheumatism, Diabetes).

In addition to these causes as given above, it should be remembered that various chronic diseases, particularly those of the heart, liver, and kidneys, tend to render attacks of acute gastritis of readier occurrence.

Acute gastric catarrh, caused by *errors in diet*, is common at all ages and in all ranks of life. Among children it is frequently accompanied by a similar inflammation of the intestinal mucous membrane, and known as *gastro-enteritis*; hence it is often described as being most common among middle-aged and elderly persons, the cases in the young falling under another category. Women are said to be more subject to it than men, but this is doubtful. Too hot, too rich, or too much food ingested at one time; iced drinks; food swallowed in imperfectly chewed lumps, or in large quantity too quickly, are sufficient in themselves to set up irritation and lead to inflammation of the gastric mucous membrane.

But apart from such errors in diet *toxic and irritant substances* introduced into the stomach along with the food are frequently the cause of acute attacks. The commonest of these is undoubtedly alcohol, while the products of decomposition of articles of diet not infrequently are at fault. Several poisonous chemical substances act in the same way—notably arsenic, phosphorus, and mercuric chloride. It is still a moot point whether a true infective gastritis occurs or not. Oser and Lebert have suggested such a possibility, but no evidence has been brought forward as to the presence of any bacteria in the stomach to which it can be due. On the other hand the acute forms of gastritis which so often accompany such infectious diseases

as scarlet fever, measles, and other exanthems, certainly represent local irritation of the gastric mucous membrane caused by the presence of the *materies morbi*. This form is often known by the name of *Erythematous Gastritis*.

Constitutional disorders may either induce attacks of acute gastritis (for example, an attack of acute gout may select the walls of the stomach as its locus in place of a joint), or render the individual suffering from them far more liable to be attacked. Local errors, also, of function or motility, leading to the presence of hyperacid contents, whether from hydrochloric acid or the organic acids of fermentation and prolonged stay of food in the stomach are occasionally provocative agencies.

MORBID ANATOMY.—The naked-eye appearances shown by the mucous surface of the stomach in a state of acute catarrh rarely come before the pathologist; the disease is seldom fatal in itself, while the signs presented after death are very unlike those during life. Our chief source of information is still contained in Beaumont's observations upon Alexis St. Martin. The stomach is usually empty, all food introduced having been expelled; small in size, and contracted, but with a soft mucous membrane, more or less studded with superficial hæmorrhages, which are perhaps most numerous in the neighbourhood of the pylorus; irregularly congested and red in patches, and covered by a tough layer of thick and tenacious mucus, here and there, it may be, showing a pink tinge. Beaumont described small vesicles, surrounded by zones of deep congestion, especially in the pyloric region.

Microscopically, the epithelial cells are distended and granular, the granules picking up colouring agents badly, if a section be examined after the inflammatory process has proceeded beyond the superficial stage. The cells in the pyloric region are those which suffer most.

SYMPTOMS.—Sudden in onset, ushered in by feelings of intense nausea and gastric discomfort, acute gastric catarrh is early marked by severe vomiting and retching. The nerve-endings in the gastric wall and the neurons of the local gastric mechanism are so powerfully irritated that abnormal vaso-motor changes are reflexly induced throughout the body; the skin is pale, cold, frequently in goose-skin, and beaded with drops of sweat; patches of *herpes labialis* may rapidly form; the brain is also affected, functionally and mentally; the body-temperature may temporarily rise, one or more of the senses and sensibility in general may become deranged, vertigo occur, and exertion be impossible. The pain in the gastric region is severe, often burning, and frequently associated with a feeling of great weight in the stomach. Tenderness may be present on pressure.

Gastric contents vomited during an attack of acute catarrh vary less with the nature of the food taken beforehand than in almost any other condition where emesis occurs. They are almost always thick, porridgy, of evil odour, rich in mucus, fermented and sour. The sourness is due to organic acids, not to hydrochloric acid, which indeed is very seldom present in the free state, and scantily in proteid combination. The porridgy consistence of the vomited material apparently bears no relation to the fluidity of the ingesta recently swallowed; the condition may arise by a rapid passage of the supernatant fluid portion of the contents through the pylorus or from admixture with mucus.

When the acts of emesis are very powerful, and especially if they persist after the stomach is empty, blood may be brought up mingled with the food or with mucus only.

Beginning suddenly, running an acute course, the catarrh almost as quickly loses all its characteristic features. In favourable instances return to healthy conditions follows; in others the symptoms acquire a subacute character; and in others again pass into those of chronic gastritis.

DIAGNOSIS.—The diagnosis of acute gastric catarrh itself offers, as a rule, no difficulty; a correct appreciation, however, of how it has been caused and what it betokens is a much more important and occasionally obscure question. When a sequel to a debauch—in solids, fluids, or both—no trouble arises; if due to irritant poisoning, the rules for detection of such are laid down in text-books of Forensic Medicine. It is when intercurrent attacks complicate other forms of digestive disease, chronic constitutional maladies or acute infectious disorders, that accurate ascription to the true cause may be of so much value. The rapidity of onset, the nature of the vomited matter, the general vasomotor and sensory disturbance, the local gastric pain, serve to point out acute gastric catarrh. Careful investigation of the anamnesis, and intelligent appraisement of all the conditions which may possibly have induced an attack, should serve to indicate the basal lesion at fault.

PROGNOSIS.—As already mentioned, acute gastric catarrh is often completely recovered from after a short space of time, if reasonably treated. But it may take on a subacute character, and last in this form for a longer or shorter period, although cure is still possible. Should repeated acute attacks be experienced, the chronic forms commonly supervene.

TREATMENT.—The less drugs employed in the treatment of acute attacks of gastric inflammation, and the greater the sense shown by the physician, the greater the chance of speedy recovery. Acute gastritis usually arises from direct irritation of the gastric surface, and is best relieved by removal of the irritant, associated with as complete a condition of gastric quiescence as possible. As a general rule the exhibition of drugs proves but palliative, and may even exaggerate the condition, with the single exception perhaps of simple emetics. Nature strives to cure by expulsion of the irritant material by antiperistaltic action, or, if this is not possible (as not infrequently happens owing to personal idiosyncrasy and inability to vomit), by evacuation *per anum* in the form of diarrhoeal stools. In the first place, if nature has failed to induce her remedial measure of emesis, her well-meant endeavours should be aided; while, if these prove ineffectual, and the space of time which has elapsed since the primary inception of the morbid process would suggest that the stomach should have emptied the larger portion of its contents through the pylorus into the duodenum, the administration of intestinal lubricants, to hasten the passage of the deleterious stuff through the bowel, and its external riddance, is indicated.

One may be able to coax victims of acute gastritis, naturally incapable of unaided emesis, to vomit by giving them tepid or warm water in bulk to drink, or mustard in watery solution, preferably warm; or if the general reflex depression be so acute and urgent that instant evacuation of the

stomach-contents is demanded, by employment of zinc or copper sulphate by the mouth, or of apomorphine hypodermically. Such measures are to be succeeded by stomachic rest. Deprivation of all food for some time, if it be possible, acts well; if food must be given, it should only consist of milk, or milk and water, taken in small amounts at any one time; and in the course of some hours, according to the progress of the patient, added to by the inclusion of the blandest forms of proteid and farinaceous foods. If the source of the acute attack lie in the misuse of alcohol, the vomiting be severe, and the subsequent depression of mind or body intense, the need may become imperative for a temporary stimulation of the vital functions, lest they should stand still. The adoption of actively detergent methods in such cases requires always to be combined with the most careful and constant supervision, attention to the condition of the patient, and readiness to counteract any signs of collapse by the prompt exhibition of stimulants—‘a hair of the dog that bit him,’ preparations of ammonium, strong solutions of such readily absorbable proteid nourishment and extractive stimulants as are afforded by Bovril, Liebig’s extract and similar substances, or by various predigested proteid products exemplified by somatose, tropon and the like. If the symptoms of collapse are very urgent, the more diffusible stimulants are indicated; if less so, and the stomach is tolerant of them, the beneficial effects of liquid nutriment, although less rapidly produced, generally prove more lasting and curative in character. In all cases of collapse the external application of heat by means of hot-water bottles, blankets, &c., is always indicated; and every measure calculated to restore peripheral circulation, to diminish engorgement of the abdominal veins and remove the consequent over-accumulation of stagnating blood in them.

2. *Phlegmonous Inflammation of the Stomach (Gastritis Phlegmonosa Suppurativa vel Purulenta; Abscess of the Stomach).*

DEFINITION.—Circumscribed or diffuse suppurative inflammation situated in the submucous and muscular layers of the stomach.

ÆTIOLOGY.—Phlegmonous gastritis is a comparatively rare disease, either arising primarily, or developing secondarily during the course of pyæmia, fevers, or severe exanthemata. Males appear to be more liable to it than females, in the proportion of 5 to 1 according to Brinton, 4 to 1 according to Glaze. The active cause consists in the presence of pyogenic bacteria in the tissues of the stomach-wall.

MOREID ANATOMY.—Two forms can be distinguished. First, where the area of suppuration is circumscribed (*gastritis phlegmonosa circumscripta*), and only one focus, or at most two or three foci, are present; second, where there is a diffuse purulent infiltration of the submucosa and muscular layer. In both forms, the seat of inflammation is at first in the submucosa and muscularis; during the later stages the mucous membrane or the serous covering may become implicated, leading possibly to rupture into the gastric cavity or the peritoneal sac. The region most frequently involved is that in the neighbourhood of the pylorus. When circumscribed, the point of entrance of the bacterial agent is commonly to be found in a local solution of continuity in the mucous covering of the stomach; a broken-down cicatrix, the inheritance of an old gastric ulcer;

a gastric erosion ; or traumata of the mucosa. When diffuse, the condition is due to the presence in the general blood-stream of pyogenic organisms, as in pyæmia and puerperal septicæmia, rarely of the *Bacillus coli communis*, or of the bacterial agents actually proved or hypothetically supposed to be the source of the acute exanthemata, admixed as they usually are with some of the ordinary pyogenic forms, which induce the miliary points of suppuration. When the abdomen is opened after death in a fatal case of diffuse phlegmonous gastritis, the outer surface of the stomach is found to be dotted over with a large number of raised swellings of variable size, while on opening the viscus, similar and even more numerous elevations can be seen. The individual abscesses, which generally account for these projections, vary in size from a pea to a hazel-nut or walnut.

As the disease progresses, infiltration of the mucous membrane, by extension between the glandular tubules, and fatty degeneration of the muscular fibres themselves, often result.

The stomach-wall, enormously thickened and engorged with pus, may resemble a sponge, being penetrated by many communicating apertures, so that on compression pus wells out here and there over its surface. Localised sloughing of the superficial tissues may lead to the formation of ulcers at different points.

SYMPTOMS.—In the large majority of cases phlegmonous gastritis runs an acute course. The onset is sudden ; the symptoms rapidly become severe in character. From the beginning the patient is conscious of being seriously ill ; his temperature quickly runs up to, or above, 104° F. ; he is restless and alarmed and, it may be, delirious ; and death closes the scene after a few days, or at most after the lapse of a fortnight. Violent acts of vomiting usually, but not invariably, accompany it, and jaundice has been recorded in cases belonging to the idiopathic primary group (Brinton), due to an extension of the disease to the duodenum, or to the unrecognised presence of pyæmia giving rise to a pyæmic icterus. The material vomited during the earlier stages consists mainly of undigested food, mucus, and bile ; towards the close it may contain pus in varying quantity. Severe epigastric pain, an intense sensation of burning in the stomach, great thirst, parched tongue, and complete loss of appetite, are among the common symptoms met with.

DIAGNOSIS.—This is difficult during life. Even the appearance of pus in the vomit affords no absolute proof of the nature of the lesion, because the pus may have come from perigastric or extra-gastric suppuration, which has found its way into the stomach cavity. The symptoms simulate so closely those of acute localised peritonitis, of hepatic abscess (in the left lobe), and of splenic abscess, that no positive diagnosis can possibly be justified. A conjecture as to the actual location of the lesion may happen to be correct, but its correctness can only be a matter of chance.

TREATMENT.—Little can be done for this condition.

B. Subacute Gastritis. — **SYNON.** : Dyspepsia ; Gastric Catarrh.

DEFINITION. — Inflammation of the gastric mucous membrane, less severe than in acute gastritis, of moderate duration and variable course.

ÆTIOLOGY.—The customary division of gastritis into acute and chronic groups, with more or less sharply defined characteristics, seems to err in an insufficient regard for those common examples of gastric catarrh which assuredly are not acute, in the true sense of that qualification, nor yet can be correctly described as chronic. Many cases of simple dyspepsia of organic origin, entirely innocent of abnormal nervous influences, belong to this group ; the local inflammatory changes being of moderate intensity, having as yet induced but slight organic alterations of a lasting character. More frequent in females than in males, subacute gastritis principally occurs between the ages of fifteen and thirty in females, from twenty to sixty in males. Among women, domestic servants and factory-hands, the former class particularly, appear to be liable to attack ; outdoor labourers and miners among males. Anæmia and chlorosis are often associated with it in females ; out of 568 female cases collected from the Edinburgh Royal Infirmary records, 23·4 per cent. suffered from concomitant anæmia.

Subacute gastritis may proceed from any of the causes already detailed for acute gastritis, acting with less intensity and of more gradual occurrence ; or may follow for a time an acute attack. As a general rule the condition, if disregarded, if treated improperly or not at all, forms but an antecedent to the onset of chronic gastritis.

SYMPTOMS.—Yielding very similar symptoms to those met in acute catarrh, but diminished in intensity, the victim of subacute gastritis also suffers from epigastric pain and vomiting, flatulence and nausea, with distaste for food, chiefly because of the discomfort which follows its ingestion ; but he has little or none of the complete feeling of prostration caused by acute attacks ; no rise of temperature ; and retains greater command over the act, time, and place of vomiting. Mucus is not so conspicuous a constituent of the material expelled, but frequently is in larger quantities in the feces. The symptoms and signs afforded by the disturbance of gastric digestion, altered appetite and palate, vary directly with the underlying cause of the catarrh, and depend largely upon the extent of the gastric surface affected, as well as the depth ; upon the state of the nervous system, and how much it has had to do with its origin ; upon the condition of other associated digestive organs ; and lastly upon the habits and circumstances of the individual. The great majority of the commoner cases of dyspepsia are neither acute nor chronic, but suffer more or less distinctly from subacute gastric catarrh. Purely functional types of dyspepsia, it is true, have been described by many authorities as existing without any accompanying catarrh, and as capable of enduring for a length of time without inducing the same. It is probable, however, that very few cases of functional indigestion are to be met with in which organic lesions are completely absent.

C. Chronic Gastritis.—*Gastritis Catarrhalis Chronica* ; *Gastritis Glandularis Chronica*.

DEFINITION.—A chronic inflammatory affection of the various cellular elements in the gastric mucosa, leading to increase of connective tissue, and loss of function.

ÆTIOLOGY.—Chronic gastric catarrh almost always follows after precedent attacks of acute or subacute gastritis ; especially if the attacks have recurred more or less frequently, and have been neglected or maltreated. It is generally said to be

more common in males than in females, but the records of the Edinburgh Royal Infirmary for 1892-9 yield 79 cases of chronic gastric catarrh among males, 77 among females, out of 205 and 289 cases of gastric catarrh of all varieties respectively. It is met with chiefly in adults under 55; for women the decennium from 20 to 30 seems to be the most dangerous, for men from 30 to 50 years of age. Although distinct from chronic gastric irritation, where the primary sources of error lie in functional faults, chronic catarrh may be safely said never to be very far distant, in fact and in act, after gastric irritation in any form has been established. Prolonged disturbance of function or motility, and persistence in dietetic indiscretions, tend to initiate chronic inflammation of the gastric glands. Continued and excessive indulgence in protoplasmic stimulants and irritants, such as tea, too hot foods, alcohol, powerful condiments and spices; constant avoidance of any one of the necessary food-elements, or more or less rigorous confinement to articles of diet of a special and insufficient kind, form some of the commoner personal causes of chronic catarrh. Anæmic conditions; faulty metabolic processes; constitutional maladies—tubercular, syphilitic, gouty, rheumatic, glycosuric; attacks of acute disease, especially during the period of convalescence, constitute the general predisposing causes. Thus the ætiology of chronic gastric catarrh is seen to be akin to that of acute or subacute catarrh. The type of catarrh excited by any one or other of these pathological conditions varies directly with the variety and energy displayed by the agents severally, and with their duration. Repetition of irritation, however, soon leads, as in all else, to diminution in response, and progressively to more lasting organic changes in the tissues.

MORBID ANATOMY.—That chronic gastric catarrh may be separated into several more or less distinctly defined forms is suggested by the conditions presented by the mucosa and submucosa in different cases. Thus chronic catarrhal, chronic proliferating glandular and chronic atrophic glandular forms may be recognised, differing greatly from one another in their anatomical lesions during the earlier stages of disease; approximating more and more to a common type as time advances; and gradually passing through the earlier stages, in which the glandular elements are chiefly affected, into later stages marked by increase of connective-tissue cells at the expense of the cells of the glands. Fibrosis of the stomach, separately described, may be one of their sequels.

The appearances presented by the mucous membrane during the earlier stages of the lesions may be described as follows:

1. In chronic catarrhal gastritis the columnar epithelium of the glands proliferates and undergoes degeneration, goblet-cells being found in increased numbers, while the peptic cells are diminished: the acid-forming cells are unchanged. The connective-tissue is infiltrated with embryonic cells. The walls of the stomach are thickened and the mucosa is swollen, the surface being of an irregularly mottled, red colour, and covered with thick mucus. The gastric secretion contains more mucus than usual, along with less than the normal amount of acid and pepsin: digestive power is therefore weak. Motility remains normal.

2. In the chronic proliferating glandular form (*G. chronica glandularis hypersthenica*) the columnar

epithelium may disappear over large areas. Goblet-cells are lessened in number, peptic and acid-forming cells increased; there is slight increase of the connective tissue of the stomach-wall. The lining membrane is covered with mucus, and the mucosa is thick and swollen, denuded in places of its epithelium. The gastric juice contains excess of acid and pepsin, with varying amounts of mucus. Digestive power and motility vary in different cases, the former being often hindered by fermentative processes.

3. In the atrophic form (*G. glandularis atrophicans*) the columnar epithelium degenerates and goblet-cells are found in increased numbers; the peptic and acid-forming cells disappear. The connective tissue is increased, especially that lying between the glands. The stomach-wall is thin, and the inner surface smooth and lacking in folds. The gastric juice contains diminished amounts of acid and pepsin; mucus may occur in increased quantity. Digestive power is weak, but motility remains practically unaffected.

SYMPTOMS.—The symptoms of chronic gastric catarrh are so protean and so indefinite in character, and so often accompany other pathological conditions, that a full description of them would require too great a space. In its simplest form, and in the earlier stages, discomfort, feelings of gastric fullness or weight after food may be the only complaint of the patient. These symptoms may be combined with those resulting from gastric fermentation—flatulence, acidity, heartburn, variable appetite and capricious taste. As the ingestion of food, especially of solids, is followed by sensations of unrest, a distaste for it in any form gradually arises; less and less nourishment is taken; more and more does the consequent weakening of the organism aggravate the gastric condition.

Intestinal indigestion is a very common complication. The diminished acidity of the gastric contents permits bacterial forms to reach the bowel in large numbers, owing to inefficient antiseptic properties, and incomplete digestion of the food in the stomach causes increased intestinal fermentation and irritation. Loose motions, two or more each morning, of no great bulk, fetid, fluid or pulaceous, are frequently experienced, alternating with constipation of temporary duration and marked by excessive flatus passed *per anum*.

The intestinal processes, themselves due to the abnormal gastric condition, in turn exert a malign influence upon the cause of their being, aggravating the morbid state of the stomach, contributing to the upset of the body generally; and by so doing react upon themselves again, intensifying the causes of their misdoings, and establishing a true vicious chain, difficult to break.

Whether a chronic gastritis is catarrhal, hypersthenic or atrophic matters little clinically. The symptoms of each variety differ from one another, no doubt, to a certain extent, but the phenomena met with in the majority of cases of chronic gastric catarrh, whatever be their anatomical pathology, depend so much more upon individual idiosyncrasies, environment, habit of life, previous diet, and actual characters of the gastric contents, that such are of little value with regard to treatment and advisory rules. This one may exhibit pain after food, intense nausea before it; this pain before, nausea after. Another complains of a constant bitter taste in the mouth, of a sour taste, or

a sickly sweetish nauseating flavour; again, flatulence may be the *bête noire* of one, heartburn of another; inability to indulge in alcohol, however dilute, or the necessity for some quantity of the same before digestion can be carried on. One subject will eschew meat, a second vegetables, a third farinaceous foods. What one can tolerate another cannot.

Under these circumstances no hard and fast rule can be laid down as to the symptoms characteristic of chronic gastritis. Lesions apparently identical produce symptoms most diverse. All the abnormalities which can be associated with gastric disorders may be met with: heartburn; nausea; emesis; epigastric pain; sensation of fulness and weight; eructations of gas, tasteless and odourless, or evil-flavoured and nasty; retching; loss of appetite; capricious appetite; hunger appeased by a mouthful; sleeplessness, restlessness, hypochondriasis, and even melancholia; intestinal derangements; symptoms common to gastric ulcer and cancer; local tenderness and sensitiveness; dilatation and displacement—all are at times exhibited by patients suffering from chronic catarrh of the stomach of whatever type. Bulimia and excessive thirst, the latter especially when the gastric contents are regularly rejected or removed, are also met with.

When the condition is simply catarrhal the symptoms are chiefly subjective; no abnormal resistance, splashing, or sensitiveness may be evident. When hypersthenic, they only occur as a rule during the gastric digestion of food. Proteids are borne better than carbohydrates; heartburn and hyperacidity are common. Atrophic types are especially characterised by intestinal disorders, diarrhoea, or constipation; severe pain during digestion and marked inanition. Decrease in the normal proportion of the red corpuscles in the blood, more pronounced than in that of the hæmoglobin, is also a common accompaniment. The white corpuscles are fewer and more variable in type than usual.

DIAGNOSIS.—Chronic gastritis has to be differentiated from chronic functional disease of the stomach, from dilatation, displacements, chronic ulcer and cancer. It is frequently a matter of the greatest difficulty to come to a conclusion as to the nature of the disease. The points in favour of chronic gastritis are almost entirely negative; the absence of dilatation, or displacement, of hæmatemesis, past or present, of localised sensitive spots, of tumour, of dorsal painful areas, of neurotic antecedent and reflex causes aid in determining the nature of the disease. The chemical composition of the gastric contents aids still more. If hyperchlorhydria be present cancer is excluded, catarrhal and atrophic gastritis are negative; hypersthenic gastritis, ulcer and neurotic dyspepsia remaining. If hyperacidity from organic acids be found, ulcer is out of the question, catarrhal or atrophic gastritis or cancer probable; dilatation or displacement only if clinically supported.

Nervous dyspepsia is often chronic, and often simulates gastritis. Here, however, the subjective symptoms overshadow the objective, and usually some primary source can be discovered, mental, intestinal, or generative, while the corporeal effects are much less pronounced in dyspepsias which are primarily of purely nervous origin than in those arising from chronic gastritic lesions; less weight is lost, less outward appearance of illness presented;

more depression is shown, and greater tendency to magnify symptoms.

PROGNOSIS.—In all cases of chronic gastritis the prognosis should be guarded. Owing to the organic changes which originate it, little hope of any complete restoration of normal conditions can be entertained. But as the intestines lie behind the stomach as a powerful reserve force, if proper precautions be taken and persisted in, if the diet be regulated *secundum artem*, and the habits be irreproachable, the maimed remainder of the active agents in gastric digestion may suffice, aided by the bowel, to encompass all that is necessary for the maintenance of bodily health. But although the defect is thus compensated for, the subject represents a damaged life, and will need to avoid carefully all excesses and eccentricities of diet for the future.

TREATMENT.—Every case of gastritis presents individual points of difference. No hard and fast rule for treatment can therefore be laid down, though 'cut and dried' systems which take no heed of the sex, habits, general traits and special symptoms of individual patients may succeed in some instances, more by good luck than good guidance. For scientific treatment each case needs careful study and the exercise of sound judgment. Thus it is not only useless but actually harmful to order a milk-diet for one who is unable to digest it or dislikes it, unless it be prescribed in some special tentative form—for example, administered in small quantities at frequent intervals, or largely diluted with some bland watery fluid or water itself. Equally harmful is it to advise the ingestion of vegetables or bread in cases of hyperchlorhydria or of bacterial fermentation. All the facts require to be studied and the lines of treatment adjusted to them.

The most important rules of treatment may be thus formulated: (1) Never tax a chemically deranged stomach; (2) Never give such a case any rigid diet without first ascertaining the nature of the gastric secretion, and the kind of food best tolerated by the patient; (3) Give no large meals, but 'little and often'; (4) Alcohol is permissible, if helpful, but not if it acts as a disturbing agent; (5) Exercise, short, sharp and vigorous, with a rest afterwards, is beneficial, as opposed to official constitutional saunters; (6) Fresh air is most necessary; (7) The less medicine the better; (8) If the patient be anæmic, iron tonics should not be given until the general condition has been improved by the above measures; (9) If the gastric juice be deficient in hydrochloric acid, this may be given tentatively—best with strychnine, after meals, in a dilute form; (10) If, however, this acid be present in excess, it may be administered in small doses before meals; (11) Alkalis should not be given in hyperchlorhydria, except for temporary relief; (12) A careful search should be made for any possible complicating morbid condition, accessory or even primary; when found, this must be carefully treated; (13) All irritating articles of diet must be prohibited for a period—tea, coffee (unless it be 'milk and coffee,' not 'coffee and milk'), uncooked fruit, raw vegetables, excess of sugar—unless they are well tolerated in small amounts and much desired by the patient; (14) Any disorder of the intestine co-existing must be treated rather by antiseptics and antifermentatives than by purgative drugs. By careful attention to such rules as the

foregoing, cases which have not reached the most advanced stage, termed *Fibrosis ventriculi*, may be enabled to live in comparative comfort, although actual cure of the morbid condition is perhaps impossible. See p. 1593.

D. Gastritis Tuberculosa.—Tubercular disease of the gastric mucous membrane is not very common. Out of 1,420 cases of dyspepsia and gastritis treated in the Edinburgh Royal Infirmary from 1892 to 1899, tubercular concomitants were only recorded in 18; while among 760 patients admitted for gastric ulcer, only one was directly registered as tubercular; pulmonary phthisis was noted in only 6 instances. The small numbers shown by these returns do not imply that dyspeptic symptoms and gastric catarrh are not frequently met with as secondary adjuncts of tubercular disease; only that they rarely constitute so important a part of the symptom-complex as to merit registration. They are usually entirely subsidiary, and due rather to general than to local conditions. The indigestion common to pulmonary tuberculosis usually conforms to this rule. It generally arises from the constitutional lesion alone, or combined with actual tuberculosis of some portion of the alimentary tract above or below the stomach; seldom from local organic changes of a tubercular character, although simple catarrhal changes may often be present, resulting from the general dyscrasia. Van Valzah in his recent work is only able to refer to twenty-three authentic records of tubercular ulcer of the stomach. An important point in this connection is the frequent precedence of chronic gastritis and gastrectasis of tubercular disease of the lungs. The stomach-lesion, leading to inefficient metabolism, and thus to lessened vitality, facilitates tubercular invasion, not necessarily of the stomach itself—while this organ often suffers vicariously on development of tubercle elsewhere. The therapeutic measures often adopted in tubercular diseases are well fitted to aggravate any earlier gastric troubles, or even to originate such through the overtaxing of an organ already weakened by the tubercular toxins.

E. Gastritis Syphilitica.—Syphilis does not often invade the coats of the stomach, or, at least, syphilitic changes have seldom been met with in the tissues of the organ *post mortem*. Indeed the existence of such lesions is denied by many. Several cases, however, have been recorded in which multiple flat gummatous areas, a syphilitic ulcer, or a cicatrix of a previous ulcer (only occasionally more than one) were found in the stomach. The gummatous infiltration first begins in the submucous coat, spreads into the mucosa, now and then involving the whole mucous surface of the stomach by the coalescence of the separate growths. Einhorn, however, has lately insisted on three forms of syphilitic gastritis: an ulcerative, a gummatous, and a form productive of pyloric constriction. It has to be borne in mind that in the great majority of the recorded cases purporting to relate to gastric syphilis the accuracy of the diagnosis is open to grave doubt. On the other hand a very intractable chronic form of dyspepsia is met with during the third stage of syphilis, which yields more readily to iodides than to any routine method. The unfortunate heirs to hereditary syphilis are also said to develop similar lesions on occasion. Diagnosis of syphilitic gastritis during life is difficult. Loss of appetite,

gastric pains (especially after food), eructations, obstinate vomiting and diarrhoea, and tenderness over the bowel, are, as Herz says, scarcely definite enough to judge by. Even if a history of syphilis be clearly proved, little is gained, for similar symptoms accompany the dyspeptic disorders common in anæmia, and in several of the chronic systemic diseases. No opinion therefore can be safely offered as to the syphilitic causation of any case of dyspepsia, until the effects of anti-syphilitic treatment have been ascertained. It is an important rule, therefore, in the management of obstinate dyspeptic cases which refuse to yield to the usual methods, to make trial of anti-syphilitic remedies.

ALEXANDER LOCKHART GILLESPIE.

STOMACH, Malposition of.—Malposition of the stomach may be considered under the two headings: Malposition of the healthy viscus, and Malposition of the diseased viscus.

A. Malposition of the Healthy Stomach. The healthy stomach may take part in the displacement of all the abdominal viscera which occurs in general enteroptosis (p. 458). It may be dragged from its normal site by the great omentum contracting adhesions to the pelvic viscera or becoming involved in a hernial sac. It may itself form part of the contents of a hernia, more especially of the umbilical variety. As the result of congenital or acquired defects of the diaphragm, the stomach may be situated wholly or in part within the thoracic cavity. It may be pushed from its normal situation in any direction by abdominal, pelvic, or thoracic tumours. The displacement of the stomach forwards and downwards by collections of fluid occupying the position of the pancreas and the lesser sac of the omentum is one of the most characteristic signs of this interesting disease. See PANCREAS, p. 1154.

SYMPTOMS.—In many of the above conditions the symptoms of the primary disease overshadow any that may be due to the displacement of the stomach. In others, notwithstanding the malposition, the gastric functions are normally performed. There remains a small proportion of cases in which, the cause being unimportant, the displacement of the stomach gives rise to definite symptoms. These symptoms are either those of atonic dyspepsia or of pyloric obstruction; the former being induced by defective blood-supply or impaired mobility of the organ, and the latter by mechanical interference with the exit of the gastric contents.

TREATMENT.—The treatment of dyspepsia due to malposition of the stomach must be based on the same principles as guide us in dealing with dyspepsia generally. The diet must be carefully regulated as to quality and quantity. Muscular activity must be stimulated by nuxvomica, gentian, and other tonics; and the secretions regulated by acids or alkalis, presented before or after meals according to individual requirements. Should the existence of some adhesion fixing the stomach in an abnormal position be diagnosed with any degree of certainty, and the symptoms be unrelieved by simpler measures, an exploratory laparotomy may be indicated. Surgical interference may be more confidently undertaken if the symptoms are those of persistent pyloric obstruction. For in these circumstances, should the surgeon fail to find this suspected cause of the obstruction, he may be able to demonstrate its true nature and overcome it by some operative measure.

B. Malposition of the Diseased Stomach.

A stomach which is already the seat of disease is more likely to be displaced than the healthy organ. The increased weight of the viscus and its contents in a permanently dilated stomach may cause it to occupy a lower position than normal. In such cases the outline of the organ may sometimes be distinctly seen, the greater curvature reaching almost to the pubes and the lesser crossing the abdomen above the umbilicus. In cancer or chronic ulcer of the stomach when the peritoneum becomes involved adhesions are formed to neighbouring viscera, and thus the stomach may be gradually dragged from its normal position.

SYMPTOMS.—It is generally difficult to distinguish between the symptoms of the original disease and those which are the result of the malposition. When to the symptoms of simple dilatation are subsequently added those of obstructive dilatation, the latter may reasonably be attributed to kinking of the pyloric extremity from sagging down of the stomach. In ulcer or growth symptoms of obstruction are more likely to be due to organic stenosis of the pylorus.

TREATMENT.—The therapeutic measures are those indicated for the treatment of the original disease.

LAURISTON E. SHAW.

STOMACH, Morbid Growths of.—**PATHOLOGY.**—New-growths of the stomach may be either simple or malignant.

Simple Growths are rare, and when present seldom give rise to symptoms or exert any influence upon the duration of life. They vary in structure and appearance according to the coat of the organ in which they arise. Local over-growth of the mucous membrane results in filiform or cauliflower-like processes which are termed *papillomata* or *simple villous tumours*. When the submucous tissue is implicated, smooth sessile or pedunculated excrescences—*polypi*—are produced. Polypi may be single or multiple, and are sometimes met with in the neighbourhood of a gastric ulcer. If the muscular coat is the seat of a simple tumour, localised firm nodules are formed, producing slightly elevated flat projections beneath the mucous membranes. They are called *myomata* or *fibro-myomata* according to the proportions of unstriated muscle and fibrous tissue composing them. *Lipomata* have been described arising from the subperitoneal connective tissue. In association with Hodgkin's disease aggregations of uniform small round-cells may be found in the submucous tissue of the stomach, giving rise to tumours of various size projecting into the cavity of the organ and covered by normal mucous membrane. This condition, termed *lymphadenoma*, occupies, in a pathological sense, an intermediate position between simple and malignant growths. Clinically these masses resemble the simple growths in the rarity with which they give rise to symptoms of gastric disorder.

Malignant New-growths.—While primary growth of the stomach is common, deposits secondary to growths elsewhere are rare. A primary growth in one part of the organ may, however, be associated with secondary nodules in neighbouring portions of the mucous membrane. Of primary growths there is a great preponderance of carcinoma as compared with sarcoma.

Carcinoma.—Carcinomata of the stomach may be of the scirrhous and medullary types, and contain

spheroidal or cylindrical cells. The proportion of fibrous stroma to the cellular elements appears to be an accidental condition, for in certain examples of the disease different portions of the same growth may exhibit considerable variation in this respect. The character of the cells lining the alveoli appears to be an essential feature of the growth, as evidenced by the fact that the same histological features may be found not only in all parts of a very diffuse growth, but also in its secondary deposits in distant parts of the body. Colloid degeneration is of common occurrence in all forms of carcinoma of the stomach. Of forty-four unselected cases of carcinoma of the stomach, Dr. Perry and the present writer found that thirty-two belonged to the spheroidal-celled and twelve to the cylindrical-celled group. At the same time it was noted that, on the average, the stroma was more abundant in spheroidal than in cylindrical-celled growths, and that as a consequence large soft medullary tumours generally belonged to the latter, and hard contracting growths to the former category. Growths which caused universal infiltration of the organ were usually found to be of the spheroidal-celled type. Examples of each type of growth were found in all parts of the stomach, except at the cardiac orifice. The few examples of growth limited to this orifice were all found to have their alveoli filled with spheroidal cells. The age or sex of the patient seemed to have no influence in determining the variety of cancer affecting the stomach.

Carcinoma of the stomach has its origin in the deeper layers of the mucous membrane, and gradually extends thence into all the coats of the organ. The greatest development of new tissue takes place in the submucosa. Columns of cells dip down between the muscular fibres, and ultimately destroy them. The remaining fibres undergo a considerable hypertrophy, so that the muscular coat may often be recognised by the naked eye to be more than twice its usual thickness. The implication of the superficial layer of the mucous membrane leads to its destruction and to the production of an ulcer. Sooner or later the serous coat is affected, and nodules of growth are visible from the exterior of the organ, usually following the course of the lymph-vessels. According as the development of the new-growth takes place in a perpendicular or in a horizontal plane carcinoma presents itself as a prominent fungoid mass projecting into the cavity, or as a diffuse plaque-like infiltration of the wall of the organ. In many cases these two conditions are combined. As already stated, the mucous membrane generally becomes ulcerated, but in the early stages it may merely present an appearance of undue rugosity, or it may be smoothly stretched over sub-jacent masses. Very rarely it becomes villous. Small nodules of growth, called satellites, are sometimes seen around the main mass.

In slowly growing carcinomata, especially of the spheroidal-celled variety, the portion of the organ implicated may undergo considerable shrinking, and as a result the whole stomach may be contracted or its lumen narrowed at some particular point, especially the pylorus. Cancer of the stomach may be found limited to the pylorus, to the lesser curvature, to the cardiac orifice, or more rarely to the fundus of the organ. Before death takes place the disease is generally pretty widely diffused, and in certain cases the entire organ is involved. The most frequent site of carcinoma is the pylorus.

In a series of cases examined by the writer the pylorus was implicated in 70 per cent. Whether the disease starts at the pylorus or ultimately reaches this orifice from some other site, it is exceedingly rare for carcinoma to extend beyond the stomach into the duodenum. Such an extension through the pylorus is, however, common in sarcoma. On the other hand, gastric carcinoma involving the cardiac orifice usually implicates the lower end of the œsophagus. Some authors have suggested that such growths are primary in the œsophagus, and extend thence into the stomach. The fact, however, that they are usually spheroidal-celled growths, while cancer of the œsophagus is a squamous-celled epithelioma, makes this suggestion improbable. Whenever either orifice of the stomach is involved in the growth, obstruction is likely to occur, usually as the result of stenosis from contraction of the fibrous stroma of the neoplasm, rarely by prominent masses projecting into the lumen. Obstruction of the cardia produces a simple atrophy of the stomach such as is seen in stenosis of the œsophagus higher up; obstruction of the pylorus by malignant growth is one of the most important causes of hypertrophy and dilatation of the stomach. *See p. 1595.*

When the entire stomach is infiltrated by scirrhus carcinoma there may be produced a remarkable diminution in the size of the viscus associated with a moderate thickening of its coats. A specimen is preserved in the museum of Guy's Hospital which measures only 7 inches along its greater curvature and 2 inches transversely at its widest part, the wall being not more than $\frac{1}{8}$ of an inch in thickness. In its earlier stages this special form of carcinomatous affection probably exhibits a greater thickening of the wall and a more moderate shrinking of the organ. To this general contraction and thickening of the viscus the name, 'india-rubber-bottle stomach' has been given, and much obscurity has existed as to its origin. It was at one time thought to be due to a special form of chronic gastritis. The difficulty in establishing its essentially neoplastic origin is due to the almost complete disappearance of the epithelial contents of the alveoli from the fibrous stroma of the growth. When limited to the pylorus such a condition was called 'simple scirrhus' of the pylorus; and when affecting the whole organ, 'chronic indurative gastritis' or 'cirrhosis of the stomach.' The recognition of typical carcinomatous elements in the neighbouring lymphatic glands revealed the true nature of the condition.

Death sometimes ensues while the malignant growth is strictly limited to the viscus. In many cases, however, secondary affections follow. The malignant deposit may ulcerate through all the coats of the organ, and lay open the peritoneal cavity, or it may become adherent to and invade adjacent viscera. Sloughing ulcerous excavations may be produced in the liver; the duodenum or colon may be opened and their cavities become continuous with that of the stomach. The abdominal wall may be involved in the growth, and very rarely an external fistula result. The spleen, diaphragm, pleura or pericardium may also be directly invaded. A large artery in the neighbourhood of the stomach may be laid open, and fatal hæmatemesis produced. The growth may also be carried by lymphatics or blood-vessels into other parts: the glands, liver and peritoneum being the

organs most frequently affected by secondary deposits in this way. The abdominal glands are those usually invaded, but it is important to remember that carcinomatous enlargement of cervical or inguinal glands has sometimes been the first symptom to attract attention in cases of gastric growth. Secondary deposits are found in between 70 and 80 per cent. of the cases.

The association between cancer of the stomach and simple ulcer demands attention. Many cases are recorded in which patients who have for many years suffered from symptoms of simple ulcer have been found upon *post-mortem* examination to be affected with carcinoma of the stomach. In some such cases a healed or open ulcer, having the appearance of a simple ulcer, is seen in the midst of the cancerous infiltration. The assumption that the ulcer has proved a suitable soil for the development of malignant disease seems justified. On the other hand, in some examples of cancer of the stomach in which the symptoms have been of short duration, a punched-out smooth-based ulcer may be found on or in the neighbourhood of a malignant deposit, and it is perhaps not unreasonable to imagine that the solvent action of the gastric juice, which is mainly responsible for producing simple ulceration, may influence the course of ulceration in cancer. It must be remembered also that the edges of a chronic simple ulcer may be thickened by inflammatory exudation and surrounded by a warty condition of the mucous membrane—appearances which may be mistaken for cancer, unless a microscopical examination is made. No case should therefore be regarded as an example of carcinoma engrafted on simple ulcer, unless there is a conclusive history of the pre-existence of an ulcer and histological evidence of carcinomatous deposit.

Sarcoma.—As already stated, sarcoma is a much rarer disease than carcinoma. Its rarity is perhaps not so great as existing statistics would seem to indicate. In the absence of systematic histological examination, some cases have doubtless been included as examples of the commoner disease. The writer has submitted to microscopical examination six specimens of sarcoma of the stomach, and in all the growth was found to be of the small-round-celled type, sometimes called lympho-sarcoma.

From an investigation of these specimens and of other recorded cases the following appear to be the chief particulars in which the morbid anatomy of sarcoma of the stomach differs from that of carcinoma. There is less tendency to ulceration of the mucous membrane. When the mucous membrane is destroyed it is usually in connection with extensive sloughing of the sarcomatous tumour. Earlier and more complete destruction of the muscular tissue takes place, and as a consequence the hypertrophy of this coat, so common in carcinoma, is not observed. There appears to be little if any tendency to contraction of the growth, and the pylorus is therefore not so commonly obstructed as in carcinoma. The pylorus is, however, frequently involved in the sarcomatous infiltration, and when this is the case the deposit may extend into the duodenum, an event which is very rare in the other form of malignant disease. The sarcomatous deposit may form localised nodular masses, or may exist as a general infiltration of the whole organ. The amount of new tissue formed is usually greater in sarcoma than in carcinoma, and when the whole

organ is involved tumours of great size may be produced.

ÆTIOLOGY.—In describing the ætiology and symptoms of malignant disease of the stomach the term 'cancer' will be used to include both forms of growth, certain special points of distinction being indicated as occasion may arise. Cancer of the stomach is essentially a disease of the *latter half of life*. Welch found in analysing over 2,000 cases that 75 per cent. occurred between the ages of 40 and 70, the percentage for the three decades being as follows: from 40 to 50, 25 per cent.; from 50 to 60, 30 per cent.; from 60 to 70, 20 per cent. Dr. Perry and the present writer, in investigating the histology of a small group of cases, found the average age at death in cases of true carcinoma to be 52, the oldest being 81 and the youngest 32. As to the relative frequency in the two *sexes*, considerable variety has been noticed in different series. A preponderance of males over females is however constant, some series indicating the ratio as 5 to 1, and others as 5 to 4. The exciting cause of cancer is still unknown, and beyond the fact already mentioned of the possible relationship of carcinoma to simple ulcer of the stomach, there is no evidence of the existence of any constant predisposing causes of the invasion of the stomach by malignant disease. *Hereditary predisposition* is not more often present than in cases of cancer of other organs. Chronic dyspepsia, gastritis, and traumatism, when noted as preceding cancer of the stomach, are probably merely accidental associations.

SYMPTOMS.—The symptoms of a typical case are as follows: A man between fifty and sixty years of age, having previously enjoyed good health, begins to lose strength and weight and to become pale. His appetite is capricious; easily digested food causes pain or a feeling of discomfort, and it is now and then rejected a few hours after it has been swallowed. At first the vomit has merely the appearance of undigested food, but later it is dark in colour and mixed with a black sooty fluid resembling coffee-grounds. If the abdomen is now carefully examined some tenderness and resistance may be detected in the epigastrium or below the left costal arch, or a more or less definite mass may be felt varying somewhat in position from day to day. Careful diet will at this period almost certainly relieve the more urgent symptoms; but ultimately these return with increased severity, pain after food of any description becomes intense, and vomiting and hæmatemesis are almost of daily occurrence. Side by side with these symptoms of gastric disturbance the general health rapidly deteriorates; the emaciation and anæmia are extreme, and muscular power is altogether lost. The circulation fails, attacks of syncope are common, and the dependent parts of the body become œdematous. Towards the end the more distressing symptoms, such as pain and vomiting, may be in abeyance; but the progress of the disease is constant, and death takes place in from six months to a year from the onset of symptoms. Before referring more in detail to these symptoms it is perhaps well to draw attention to the fact that cancer of the stomach may certainly reach an advanced stage without any symptoms whatever being noticed. Extensive malignant growth of the stomach is not very uncommonly found in the bodies of patients who have been killed or died of some acute disease, and who have regarded themselves as in good health until the

moment of the accident or the onset of the sudden illness. This class of case must be distinguished from the so-called 'latent cancer of the stomach,' a term which has been applied to a still commoner group, in which the patient dies from the effect of cancer of the stomach, but presents such indefinite symptoms that the essential disease is overlooked.

The symptoms of cancer of the stomach may be considered in detail under four headings.

1. Symptoms due to involvement of the Stomach.—These are pain, vomiting, and hæmatemesis.

Pain is a frequent symptom (80 or 90 per cent.), and may have any of the features indicative of gastric disorder. It may be constant and uninfluenced in any way by the ingestion of food, in which case it is probably the result of the compression of nerves by the growth or by its secondary deposits. When related to the swallowing of food, the pain may occur immediately after a meal, or may be delayed till one or more hours after food is taken. On the other hand, pain which comes on some three or four hours after one meal may be distinctly alleviated by the next. The pain may be generally diffused over the gastric region, may be confined to the epigastrium, or felt only in the back. It is sometimes associated with localised areas of hyperæsthesia (*see* p. 1142). The pain most characteristic of cancer of the stomach is a constant gnawing sensation in the epigastrium and back, greatly aggravated shortly after food is swallowed, and considerably relieved if vomiting takes place. With our present knowledge it is not usually possible to attach any special pathological significance to different varieties of pain. It is reasonable to suppose that a hyperæsthetic condition of the mucous membrane, flatulent distension of the organ, vigorous peristaltic action, and the dragging upon external adhesions may all play a part in causing pain in individual cases.

Vomiting is not so common a symptom as pain. Only 10 or 20 per cent. of cases run their whole course without vomiting, but many patients who have persistent pain vomit only occasionally. The most constant cause of vomiting in cancer of the stomach is obstruction of the pylorus associated with gastric dilatation. In these cases the vomiting takes place at fairly long intervals (36 to 48 hours); the matter ejected is large in volume, may present evidence of having undergone fermentation, and sometimes contains undigested particles of food eaten many days before.

In cases where there is general contraction of the stomach vomiting is sometimes entirely absent. When present it usually occurs at short intervals, and the amount ejected is of course small. In many cases no mechanical explanation for the vomiting can be found. This symptom may result from the failure of the diseased organ to digest the food, or it may be a more or less voluntary act in order to relieve pain. It is rarely induced by an over-distension of the viscus by blood, a common cause in simple gastric ulcer. Of more importance than the frequency and character of the act of vomiting are the features of the material vomited, or of the contents of the stomach withdrawn by a soft tube after a test-meal has been given (p. 1589). Very occasionally the vomit has a horribly offensive odour. In such cases necrosis of large masses of growth or a fistulous communication between the stomach and lower bowel has been found at the autopsy. The absence

of free hydrochloric acid from the contents of the stomach has frequently been observed in cases of gastric cancer. A notable exception to this rule is said to exist in cases of cancer following upon simple ulcer. Further investigation on this point is desirable, but it may be stated generally that the existence of free hydrochloric acid in normal amount is strong evidence against the presence of malignant disease of the stomach. On the other hand, the presence of lactic acid in the gastric contents favours a diagnosis of cancer. Careful microscopical examination may sometimes reveal the presence of portions of malignant growth.

Hæmatemesis.—The number of cases in which bleeding takes place into the stomach from the ulcerated surface of the growth cannot be accurately determined. Hæmorrhage is rarely so profuse as to induce vomiting, and unless vomiting takes place concurrently from some other cause, the existence of bleeding may be overlooked. Altered blood is found in the motions, with or without hæmatemesis. Osler observed the vomiting of blood in 24 per cent. of his cases. The amount vomited may be large or small, but is usually small. The blood is generally dark in colour, as if it had been long retained in the stomach. When in large quantities it gives the characteristic 'coffee-ground' appearance to the vomit. Profuse hæmatemesis is but rarely the direct cause of death; repeated small losses accelerate the patient's end. It is unusual for the pathologist to be able to recognise an open artery as the source of the bleeding. See HÆMATEMESIS, p. 620.

2. Physical Signs of the Involvement of the Stomach.—Careful inspection and palpation of the abdomen will usually enable the physician to detect some departure from the normal condition. The abdomen should be entirely uncovered in a good light, and no pains spared to obtain complete relaxation of the abdominal muscles. An area of hyperæsthesia, a point of tenderness on deep palpation, or a sense of resistance may be the only indication of the existence of growth. In some cases clear evidence of hypertrophy and dilatation of the stomach may be found as indicated by distension of the upper part of the abdomen, succussion, and visible peristalsis (p. 1596). In others a tumour may be seen or felt, in or near the gastric region (p. 1588). The normal relations of the parts may be altered, and in the absence of symptoms of gastric disorder, it may be difficult to determine the viscus in which the tumour is situated. Many cases of gastric neoplasm have been mistaken for growth in the spleen, liver, colon, pancreas or lymphatic glands. Careful investigation as to the relation of the tumour to the gastric resonance may enable a diagnosis to be made, and with the object of determining this relation it may be desirable cautiously to distend the stomach with gas. A gastric tumour may vary its position in accordance with the amount of distension of the viscus. It commonly descends on inspiration, and may be moved by the physician's hand, or by the patient turning from side to side. It is obvious that the tumour will be most easily felt if it occupies the pylorus or the greater curvature. If the lesser curvature is alone involved, or if the whole organ is contracted, it is likely to escape detection. A small 'india-rubber-bottle stomach' has, however, sometimes been recognised as a hard mass projecting beyond the left costal arch. Whether the gastric growth forms a palpable tumour or not, it is some-

times possible to detect masses formed by secondary deposits in neighbouring organs.

3. General Symptoms.—These symptoms, usually included in the term 'cancerous cachexia,' are wasting, anæmia, pyrexia, and œdema.

Wasting and loss of strength are prominent symptoms in nearly all cases. They are partly due to the nature and partly to the situation of the disease. In most cases the wasting is easily recognised to be out of proportion to the loss of digestive power, and is often indeed observed before any symptoms of gastric disturbance are in evidence. Anæmia is equally common, and is an early symptom. Great attention has lately been paid to examination of the blood; but with our present knowledge little assistance in diagnosis is thereby given, except to differentiate between pernicious anæmia and gastric cancer. The anæmia of cancer is rarely so extreme as in the idiopathic variety, and is invariably associated with a deficiency of hæmoglobin relatively greater than the diminution in the number of red blood-corpuscles. Osler found more or less fever in half his cases, and failed to establish its relationship either to ulceration of the growth or to the existence of secondary deposits. It is probably a direct effect of the neoplasm. Œdema of the lower extremities is fairly common, and general anasarca rare. They are both attributable to the anæmic state and to the enfeebled circulation. The possibility of secondary deposits causing pressure upon veins must not be disregarded. The same conditions of the circulation which favour œdema are probably also responsible for producing thrombosis of the veins.

4. Secondary Symptoms.—These are the result of an extension of the growth to other parts, or of the development of secondary deposits. A moderate degree of ascites may be present, either as the result of cancerous deposits in the peritoneum or of obstruction to the portal vein. Jaundice, from growth in the liver, may be the earliest manifestation of disease, or it may come on a few days before the patient's death. General peritonitis from perforation of the stomach is very rare. If perforation takes place a localised peritoneal abscess is the usual result. Metastases in the supraclavicular or inguinal lymphatic glands, or in the region of the umbilicus, may sometimes be the first clue to the malignant nature of the gastric trouble from which a patient is suffering.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of cancer of the stomach depends largely upon the recognition of symptoms belonging to two or more of the four groups enumerated above. It is not uncommon in the earlier stages, or even throughout the disease, for cases to present symptoms strictly confined to one of the above groups, or the symptoms in one group may so predominate as to cause others to be overlooked. The conditions for which cancer of the stomach may be mistaken will of course depend upon the group to which the predominant symptoms belong. When pain and vomiting are the main symptoms, dyspepsia and chronic gastritis must be considered; while if these symptoms are complicated by hæmatemesis, gastric ulcer or cirrhosis of the liver may be mistaken for cancer. Hypertrophy and dilatation of the stomach may be caused by cicatrisation of a simple ulcer, as well as by cancerous stenosis of the pylorus. A mass of growth in the stomach producing a definite tumour unaccompanied by symptoms of gastric

disturbance may be mistaken for simple or neoplastic enlargement of neighbouring viscera. The greatest difficulty in diagnosis occurs when the general symptoms of cancerous cachexia are present without symptoms or physical signs of local involvement of the stomach, or when the latter are in abeyance while the secondary effects of the growth are evident. Such cases have received the name of 'latent cancer' of the stomach. During life they may be regarded as examples of pernicious anæmia, general tuberculosis or Bright's disease, or if their cancerous nature is recognised the situation of the growth may be undetermined, or its secondary deposits in the liver or peritoneum alone detected.

TREATMENT.—There is no evidence that cancer of the stomach, when once established, can be 'cured' except as a result of its complete removal by the surgeon. Even when so removed, the tendency to recurrence is very great. The earlier the operation is performed the less the tendency to recurrence. Herein lies the great difficulty in surgical treatment. In the early stages the diagnosis is often impossible; in the later stages, when diagnosis becomes certain, successful removal is improbable. The want of success in the later stages is due to two causes. In the first place the patient's cachectic condition renders him an unsuitable subject for so serious an operation, and in the second place metastases in distant parts are likely to have already occurred. Some authors have confidently stated that no case in which the diagnosis can be made is suitable for surgical treatment. But constant progress in methods of early diagnosis and in surgical technique alters our point of view. If the physician bears constantly in mind that every case of gastric trouble in a patient of middle age may be one of cancer of the stomach, and carefully employs all the means of diagnosis at his disposal, he will be able to place many suitable cases in the hands of the surgeon.

Hitherto we have considered surgical treatment in early cases with the object of complete removal of the growth. The surgeon may also be called in later to alleviate symptoms in cases that are obviously too far advanced for radical cure. When there is obstruction at either orifice life may be prolonged by appropriate surgical measures. With growth involving the cardiac orifice gastrostomy, if performed before the patient has reached an extreme condition of starvation, may prove of great benefit. Nourishment can then be well maintained, and the removal of irritation to the growth by the passage of food may alleviate pain and arrest the progress of the neoplasm. On the other hand, pyloric obstruction should be met by making an anastomosis between the stomach and a loop of small intestine. Not only may life be considerably prolonged by such an operation, but a most striking improvement may ensue in the patient's condition. Pain and vomiting may be entirely relieved, and the patient's ability to take food greatly increased.

Apart from surgical measures considerable alleviation to suffering may be brought about by diet, by washing out the stomach, and by drugs. So great may be the effect of medical treatment that it is necessary to bear in mind that a rapid relief of symptoms must not cause a diagnosis of cancer to be lightly given up.

Diet.—The dietetic treatment of cancer of the stomach does not materially differ from that to be employed in cases of digestive failure from other

causes. As a general principle it is well to allow the patient to take the most substantial diet that experience shows him to be able to digest without serious pain. The time may come when nothing but predigested milk will be tolerated by the stomach, or when feeding must be entirely effected by nutrient enemata. This date will probably be only hastened if the diet is too much restricted in the earlier stages of the disease. Bearing in mind this principle, and having regard to the fact that we cannot hope to restore the patient's normal digestive powers, the diet must be chosen according to the directions laid down in pp. 1597, 1601, 1603, and 1606. It is often wise to consult the patient's palate, even though his choice of diet may appear to run counter to physiological laws.

Lavage.—The operation should at first be undertaken daily, and subsequently two or three times a week. The most suitable time is about half an hour before one of the principal meals, either in the morning or the evening. Washing is especially indicated in cases of pyloric obstruction with retention of food; but excellent results are often obtained in cases where no organic obstruction exists. Products of decomposition or portions of necrotic growth are likely to be present in all cases of cancer, and it is their complete removal which is responsible for the relief of pain and vomiting which so often follows this proceeding. Tepid boiled water is the best medium to use. Various antiseptic solutions have been recommended, but if such are employed it must be borne in mind that very occasionally the fluid injected cannot be easily removed. The antiseptic solution should therefore be dilute and innocuous. See pp. 1597 and 854.

Medicinal.—Various drugs may be given with the object of increasing the digestive power, arresting decomposition, or allaying pain. The absence of free hydrochloric acid from the gastric secretions in cancer of the stomach would seem to indicate that this drug might be given with advantage, and in the early stages an acid mixture containing nux vomica is often usefully prescribed after meals. As a general rule, however, alkaline mixtures prove more effectual in relieving pain. Opium or morphine should not be withheld if the pain is severe, and is unrelieved by washing out the stomach. Morphine may be conveniently combined with bismuth suspended in mucilage, while a pill containing opium, nux vomica, and creosote often proves beneficial where pain is associated with rapid fermentation of the gastric contents.

LAURISTON E. SHAW.

STOMACH, Neuroses of the.—Gastric neuroses may be classified as: I. Motor; II. Sensory; III. Secretory. Under each one of these we may distinguish (a) states of excitation, and (b) states of depression.

Irritative Motor Neuroses.—1. *Cardiospasm.*—Cardiospasm is a secondary disease in the great majority of cases, due either to hyperæsthesia, to very strong irritation of the mucous membrane of the cardia, or to abnormal expansion of the stomach by air and gases. It may occur in ulcer and carcinoma of the lower end of the œsophagus and of the cardia.

The prominent symptoms depend upon the inability to pass solids, liquids or gases, in either direction from the cardia: the strongest efforts at vomiting bring up nothing. There are two forms

of this neurosis, the acute and chronic. The acute cardiospasm appears suddenly, spasmodically, and is of short duration. It is recognised only when a tube is, for instance, introduced into the stomach, or when food or drink is taken by the patient. The chronic form is a very stubborn and grave disease, and may even become the cause of death through inanition. The patients have an unmistakable feeling that deglutition is impeded if they happen to swallow during an acute cramp. If in spite of this food is still swallowed, the lower part of the œsophagus immediately above the stomach becomes distended.

The recognition of cardiospasm is made by auscultating for the deglutition-sounds with a stethoscope while the patient is directed to swallow some water; absence of the second deglutition-sound means an obstruction at the lower end of the œsophagus or at the cardia. Passage of the stomach-tube gives information concerning the permeability of the œsophagus.

Treatment consists in the avoidance of causes increasing neurasthenia; correction of aerophagia; introduction of firm thick tubes, which are allowed to remain in position for twenty minutes; and application of cocaine hydrochloride and camphor-menthol to the lower end of the œsophagus and cardia. The galvanic current is of value in the treatment, the anode being placed in the cardia by an intragastric electrode, the cathode over the cervical spine. The bromides may give relief if continued two to three weeks.

2. *Pyloric spasm*.—This may be due to gastric hyperæsthesia, to chemical irritation by excess of hydrochloric acid or by abnormal formation of organic acids, to dilatation of the stomach by gases, and to toxic and irritative substances formed by intragastric fermentation. Secondary spasm may accompany ulcer, or ulcerating carcinoma of the pylorus.

It may be assumed to exist when atony of the stomach occurs without any assignable cause. Persistent pyloric spasm may lead to pronounced gastric dilatation. If associated with intense pain and hyperacidity, gastric ulcer should be suspected.

By way of treatment the pylorus may be intubated by the author's method of duodenal intubation (Hemmeter's *Diseases of the Stomach*, 2nd edition, p. 53). The bromides, 30 grs., combined with $\frac{1}{2}$ gr. ext. belladonnæ, or 5 grs. chloral hydrate, are very useful in this condition. The galvanic current, and spraying with cocaine and menthol, may also be employed.

3. *Gastrospasm*.—Gastrospasm signifies a spasmodic contraction of the entire gastric musculature. The whole stomach may be contracted, so as to resemble a cannon-ball. It is fortunately a very rare condition. It closely resembles gastric hyperperistalsis.

4. *Gastric hyperperistalsis*.—‘Peristaltic unrest’ is the name Kussmaul gives to a state of the stomach characterised by the appearance of extraordinarily rapid contractions, appearing especially after meals, but continuing also during the day and sometimes through the night with an entirely empty stomach. The patients complain of disagreeable sensations of heaving to and fro, of unrest, and contractions in the region of the stomach. When there is ectasis or dislocation of the stomach present simultaneously these abnormally strong contractions may be seen and felt externally. Sometimes, also, peristaltic

unrest of the small intestine co-exists with that of the stomach.

Increased activity of the stomach may be brought about—(1) As a reflex process, through hyperæsthesia of the sensory nerves; (2) by a strong stimulation of the mucous membrane of the stomach by excess of hydrochloric acid, by organic acids, and by distension from abnormal fermentation; (3) as the result of an advanced stenosis of the pylorus or duodenum; and (4) owing to an increased irritability of the motor nerves.

Sexual excesses, repeated intense emotions, an unsuitable mode of living, general nervousness, as well as anæmia, increase the disposition to this disease.

The patients frequently complain of a lack of appetite, belching, nausea, and vomiting. Loss of flesh may occur, so that a suspicion of malignant disease may arise. If the small intestine is also the seat of active peristaltic unrest, intestinal gases and liquids sometimes regurgitate into the stomach; the eructations are then very foul-smelling, and even fæulent.

Treatment consists in avoidance of sexual excesses, mental shocks, and over-exertion. If the peristaltic unrest is a partial or resultant effect of a decided neurasthenia or anæmia, change of air and hydropathic procedures are useful. Anæmia must also be corrected. Sedatives, like the bromides, opium, and belladonna, are said to exert a more controlling effect on the intestinal than on gastric hyperperistalsis.

In severe cases in which the peristaltic unrest continues through the night, rest in bed and a milk diet are recommended, and cold bandages or packings of the stomach should be tried, and if these do not relieve, then warm cataplasms. Any immoderate distension of the stomach is to be strictly avoided. The present writer has had satisfactory results with exclusive rectal feeding in two cases of peristaltic unrest.

Of medicines, sodium, ammonium, or strontium bromide, in doses of 45 to 75 grs. three times in twelve hours, or extract of belladonna ($\frac{1}{4}$ to $\frac{1}{2}$ of a grain), are to be recommended. In a persistent case of peristaltic unrest in a gouty patient salicylate of sodium (20 grs.) along with bismuth subnitrate (16 grs.) three times daily appeared useful.

5. *Nervous eructation*.—The belching up of tasteless or offensive gases is a frequent symptom in most gastric diseases, and occurs at times in every normal person. The pathological eructation which occurs in neurasthenia consists of the explosive evacuation of tasteless gas in large quantities. The attacks are usually paroxysmal, and the gas that is expelled is air which has been swallowed. Nervous individuals, particularly hysterical patients, may belch up air during the entire day, and often during the night.

An exaggerated form of this disease is known as *pneumatosis*. Considerable dyspnoea may occur in these cases resembling the ‘asthma dyspepticum’ of Hensch. In many cases persistent constipation will be found to be an ætiological factor.

In order to cure this disorder the patient and his attendant must be instructed that the eructation is largely a habit, and that by close observation the patient can be interrupted in the act of swallowing air. Penzoldt cured a patient of this kind by making him keep his mouth open for a half-hour,

as it is impossible to swallow air when the mouth is open. The explosive eructations of hysterical patients are best treated by methods directed towards the psychical condition of the case. Quincke has seen cures by introducing a thick, soft stomach-tube, which is permitted to rest for a while in the œsophagus. Cases that depend upon aspiration by alternate expansion and contraction of the stomach are benefited by the intragastric application of the galvanic current. The neurasthenic foundation of the disease should receive careful attention, e.g. by a course of surf-bathing. Cold sponging and massage are very useful aids in treatment. Among the drugs that have been recommended are small and frequently repeated doses of arsenic, belladonna, or atropine. Hypodermic injections of morphine may be required in severe cases.

6. *Nervous, habitual, or reflex vomiting.*—There are three forms of nervous vomiting: (1) The cerebral or spinal vomiting, which is caused by direct or indirect stimulation of the vomiting centre in the medulla oblongata from foci of irritation in the brain and spinal marrow. (2) Nervous vomiting, occurring as a symptom of hysteria or neurasthenia. (3) The reflex vomiting, in a more restricted sense, brought about by reflex irritations from various other organs in the body.

(1) *Cerebral vomiting.*—See VOMITING.

Spinal vomiting (gastric crises).—See TABES DORSALIS.

Periodical vomiting (Leyden).—This is a combination of symptoms in which the prominent feature is vomiting that returns at regular intervals. The attacks begin without any marked prodromal symptoms, in the midst of apparently good health. Gastralgia may introduce the attack or follow it. The appetite is lost, pulse small and frequent, tongue coated and dry. The patients may have intense headache or even slight delirium. It appears occasionally as a primary, idiopathic neurosis of the vagi; it has also been known to occur with hydronephrosis, floating kidney, diseases of the uterus and ovaries, intestinal entozoa, and nicotine-poisoning.

(2) *Nervous vomiting in the course of neurasthenia and hysteria* (M. Rosenthal).—These patients frequently complain of severe pain in the gastric region, pointing to a hyperæsthesia of the sensory nerves. The following points are characteristic of vomiting of nervous origin (Stiller): (1) the facility of the emesis; (2) the independence of the quality and quantity of the ingesta; (3) the capriciousness with which very bizarre articles of diet are frequently retained to the exclusion of others; (4) sometimes the elective vomiting of certain substances which seemingly are separated from the mixed chyme; (5) the carelessness with which the patient endures the habitual sickness; (6) the tolerance of the body to the effect of inanition caused by the habitual vomiting; (7) the extraordinary influence of the slightest causes that act on mood or temperament; (8) the frequent occurrence of emesis when no food has been taken and the stomach is apparently empty; (9) the presence of other nervous symptoms alternating or contemporaneous with the vomiting; to these Boas adds (10) the absence of important secretory or motor disturbances. Nausea and retching are absent in the vomiting of hysterical patients.

Juvenile vomiting is an expression of a dyspepsia developed in school children as the result of

mental over-exertion. The symptoms are dyspeptic complaints, gastralgia, vomiting, great pallor, dilatation of pupils, slowing of the pulse, and constipation.

(3) *Reflex vomiting in a more restricted sense.*—This may occur as a result of abdominal disease, not only grave disorders such as peritonitis and the various forms of colic, but also of slight derangements of the female sexual organs. Normal menstruation and pregnancy are occasionally accompanied by emesis (see PREGNANCY, Disorders of).

Whenever possible the treatment must be directed to the underlying causal diseases. Gastralgic pains and hyperæsthesia can be relieved by a hot cataplasm on the stomach, but the application of the galvanic current—the anode on or in the stomach, the cathode alternately on the sternum and spinal column—will be more efficacious. Internal gastric douching with warm water, and spraying the inside of the stomach with menthol and cocaine, are also generally followed by cessation of the pain. With regard to diet, the idiosyncrasy of the patient should be carefully studied. ‘No diet can be suggested that shall be universally applicable to all cases.’ The ingestion of liquids must, as a rule, be very much limited, and thirst relieved by enemata. If every meal is vomited, it is best to give nourishment in very small quantities—iced-milk, champagne, cold tea or coffee, or egg-albumen with brandy, or clam-bouillon in teaspoonful doses. Superacidity must receive appropriate treatment. When there is abnormal hyperæsthesia of the stomach, it is well to feed the patient by rectal enemata for about a week or ten days. The most effective sedative is morphine, particularly the hypodermic injection of $\frac{1}{4}$ of a grain, together with $\frac{1}{150}$ of a grain of atropine sulphate; or suppositories containing extr. belladonnæ, gr. $\frac{1}{4}$, with codeine phosphate, gr. j, may be tried. When the vomiting persists, even during the night, the bromides, together with chloral hydrate, are of approved efficacy. Hysterical vomiting is best controlled by withholding all food by mouth, and giving nutrient enemata.

Depressive Motor Neuroses.—(1) *Insufficiency or incontinence of the cardia.*—Incontinence of the cardia, due to paresis or paralysis of the motor nerves of the ring-muscle, is a comparatively rare malady, though somewhat more frequent than that of the pylorus. It appears either as an independent disease or as a resultant of other neuroses. Energetic peristalsis of the stomach, with the additional influence of the abdominal pressure, will in this condition raise portions of the gastric contents into the œsophagus, and even into the mouth. From this result regurgitation and rumination.

Regurgitation.—At a longer or shorter period after meals large quantities of the liquid and solid contents of the stomach are, at first involuntarily, brought up again into the mouth, and are then ejected. The patient generally learns how to facilitate the ascension of the ingesta by means of rather severe contractions of the musculature of the abdomen. Long-continued regurgitation may develop into rumination.

Since regurgitation is promoted by hasty eating and quick swallowing of insufficiently chewed foods, especially when the latter are difficult of digestion, a diet should be prescribed that is easily digestible, and the patients should be directed to eat slowly

and chew thoroughly. They should combat regurgitation to the utmost of their power. In stubborn cases the swallowing of small pieces of ice is recommended (Alt), which may reflexly induce the musculature of the cardia to contract. In addition massage of the epigastrium, internal and external galvanism and faradism, as well as internal administration of strychnine nitrate ($\frac{1}{30}$ gr.) are indicated. The galvanic current should be applied in the same manner as in cardiospasm.

Rumination or Merycism.—Patients afflicted with this neurosis return the ingesta from the stomach through the oesophagus back into the mouth, to reswallow them, as a rule, after they have been chewed again. This is not only done without exertion or nausea, but apparently with a certain enjoyment. The regurgitation of ingesta from the stomach into the mouth is usually at first voluntary, but later on involuntary. The ascent of the food causes a pleasurable sensation, and the patients assist the act by means of the pressure of their abdominal muscles. The condition of the secretory function is variable. A neuropathic constitution is a frequent factor in the development of rumination. Heredity seems to have some effect in the matter, but the element of imitation and suggestion cannot well be eliminated here.

Medicinal treatment in this disease is of little value. The state of the secretions should be carefully determined, and subacidity or superacidity corrected by appropriate means. The physician should, however, in all cases insist on slow eating and careful chewing; the food should be easily digestible and largely composed of gruels and diet of a soft consistency. The patient should always take his meals in the presence of persons for whom he has respect and who understand how to combat the morbid habit. The success of the treatment will depend upon the will-power of the patient himself, who must resist the temptation to ruminate with all the self-control at his command. He should be guarded against using the contraction of the abdominal muscles to assist the act. A trial might be made with the intragastric use of the faradic and galvanic currents. Hydropathic methods are sometimes useful. In one case observed by the present writer, in which every meal was persistently ruminated, rectal alimentation for twelve days entirely cured the patient. In another case the patient was cured by the administration of ten grains of quinine after each meal; probably the quinine rendered the food so disgustingly bitter that the patient suppressed the regurgitation.

(2) *Insufficiency or incontinence of the pylorus.*—This may be caused by organic diseases of the stomach and intestines, by carcinoma and ulcer, or by a stenosis of the duodenum leading to advanced dilatation of the initial part of the same. Ebstein has described rare cases of insufficiency, which appear, in the absence of anatomical changes, genuine neuroses, due to paralysis of the motor nerves of the pyloric sphincter; also instances in which it was a concomitant of myelitis due to compression, of hysteria and of gout. It may, perhaps, occur also as an idiopathic malady. If the muscular insufficiency is confined to the pylorus, then the foods and liquids either remain a very much shorter time than usual in the stomach, or enter the intestines immediately after their ingestion; thus the whole, or almost the whole, burden of digestion falls upon the intestine. Since, however, the in-

testine, in normal conditions, may entirely make up for lack of digestion in the stomach, disturbances of nutrition generally fail to appear, especially when an easily digestible diet is prescribed.

The symptoms of pyloric insufficiency are, in brief, the following: If frequent vomiting and belching existed, these suddenly cease after the setting in of the insufficiency. If rather large particles of food get into the intestine, the increased peristalsis may cause diarrhoea. This may also be brought about by very cold or very hot drinks or foods. If a quantity of air was swallowed with the foods, or if drinks rich in carbonic acid have been imbibed, a very acute tympanites of the intestine may develop from the escape of air or carbonic-acid gas.

Insufficiency of the pylorus should be suspected if one cannot succeed in distending the stomach by the artificial production of carbon dioxide in the organ by tartaric acid and sod. bicarb. Ewald, Boas, and other authors contend that this evidence is not conclusive; but any possible source of error may be removed by letting the patient eat a test-breakfast before the distension of the stomach, since then the closure of the normal pylorus becomes so firm that the carbonic-acid gas set free cannot at once escape into the intestine. If the air passes quickly into the intestine, tympanites of jejunum and ileum soon becomes evident. For a proof of the purely nervous origin of insufficiency it is necessary to exclude the above-mentioned diseases of the stomach and intestine, and such organic diseases of the stomach as chronic gastritis, which probably bring about a serous infiltration of the annular muscle, and may lead to a temporary insufficiency (Eichhorst, Boas).

Insufficiency of the pylorus may be recognised by the author's method of intubating the duodenum (*Archiv f. Verdauungskrankheiten*, Bd. II. S. 85), and by the spiral revolving sound of Kuhn and of Türk.

Only dietetic treatment is necessary, if there are no symptoms of intestinal irritation. In order to relieve the intestine of its additional task, easily digestible, well-prepared foods, which are to be carefully masticated, and are not to be taken too hot or too cold, should be prescribed. If, on the other hand, complaints such as diarrhoea appear, we must attempt, in addition to the treatment of the causal disease, to get rid of the insufficiency by means of massage, internal and external galvanism, and faradism applied directly to the pylorus, douches, and eventually also by giving strychnine sulphate, gr. $\frac{1}{30}$, three times a day. Flatulence and constipation call for massage and electricity, to be applied also to the intestine, and dilute hydrochloric acid should be administered in doses of 30 drops largely diluted if test-meal analysis shows absence of this acid.

(3) *Atony of the stomach* (Myasthenia Gastrica; Insufficiency of the Stomach). See STOMACH, Dilatation of.

Irritative Sensory Neuroses.—(1) *Hyperæsthesia.*—Hyperæsthesia of the stomach is probably a neurosis of the vagus, and a mild form of gastralgia. Clinically, the two forms of gastric sensibility—viz. gastralgia and hyperæsthesia—are differentiated by the following facts: The unpleasant sensations of pressure, fulness, and pain in the epigastrium, with eructations, nausea, and vomiting, occur in hyperæsthesia only after the

ingestion of food ; that is, there must be a digestive stimulation of the mucosa. The distress occurs only after meals, very rarely with an empty stomach. In gastralgia the pains and other distress occur with equal intensity in the full as well as in the empty stomach ; digestive irritation is not necessary to cause gastralgia. Hyperæsthesia lasts several days, weeks, or even months, with uniform or gradually increasing intensity, and during this time dyspeptic symptoms occur daily after each meal ; in gastralgia, however, the pains last only during the attacks, generally for a few hours.

The primary idiopathic form occurs very frequently as an accompaniment to chlorosis and anæmia, particularly with women and young girls. Also after repeated overloading of the stomach with indigestible food ; long-continued use of very salt or acid or spiced foods ; and the ingestion of very hot or very cold drinks after long fasting, and in debilitated states following sexual and alcoholic excesses, and chloroform-narcosis.

Patients with this neurosis frequently feel the pulsations of the abdominal aorta, and complain of beating and pulsating in the stomach. Then, again, they have a feeling of heat or cold, or a gnawing, burning sensation, and an impression of restlessness through the entire stomach. The ingestion of food, no matter of what consistency, causes a sensation of discomfort, fullness, nausea, and even vomiting. These sensations may increase to a typical gastralgia.

The treatment will be directed in the first place to the correction of the underlying fundamental disease. Wherever this is not possible, or wherever an idiopathic form of hyperæsthesia is present, all irritants which can exert detrimental influence upon the stomach must be excluded. All bodily and mental exertion must be avoided. In severe cases the Weir-Mitchell rest-cure, together with Leube's ulcer-cure, has, in our experience, been very efficacious. Hot, moist applications to the stomach are very soothing. Galvanism is a capital method of treating this affection.

Gastric idiosyncrasies are those peculiar forms of hyperæsthesia in which neuropathic and sometimes perfectly healthy persons have morbid sensations only after ingesting certain foods. These sensations consist of headache, light fever, cutaneous erythema, and urticaria. The author has observed persons who developed urticaria after eating crabs, potatoes, cheese or strawberries. One patient regularly develops an acute acne whenever she eats cheese, another develops fever whenever he partakes of crabs. Pick strongly recommends the use of creosote in these idiosyncrasies. Ichthyol internally is also efficacious.

(2) *Gastralgia* (Cardialgia ; Gastrodynia).—Gastralgia, or neuralgia of the stomach, occurs in periodical and spasmodic attacks of severe gastric pain, alternating with intervals of freedom from pain.

Gastralgia is frequently a result of motor or secretory neuroses—of gastrosplasm, pylorospasm, and cardiosplasm, superacidity, and supersecretion. The root of the vagus nerve may be irritated by functional and anatomical diseases of the medulla and adjacent portions of the central nervous system. We have observed a number of cases of malarial gastralgia in which the attacks occurred at regular intervals, and could be distinctly associated with the evolution of the characteristic malarial parasite in the blood.

A very careful examination is necessary before we decide that there is no real organic trouble at the foundation of the gastralgia. Occasionally we may find that gastralgias occur with small median herniæ of the linea alba. Leyden has described gastralgia with subacute myelitis, and Oser with myelitis due to compression.

In case of doubt it is advisable to treat the disease as if it were an ulcer. In true gastralgia the fundamental cause must, if possible, be discovered and removed. When we can find no organic cause, the most effective agent in our experience has been the galvanic current, using very strong currents—not less than twenty-five milliamperes. Oser claims to have observed cessation of the pains after application of the faradic current. When the pains are not too intense the internal administration of phosphate of codeine (half of a grain every three hours), chloral hydrate (fifteen grains every four hours), Dover's powder, tincture or extract of hyoscyamus, extract of belladonna, and camphorated tincture of opium are available remedies.

Gastralgokenosis.—Under this name Boas describes a painful emptiness of the stomach which occurs one to two hours after meals, and may be so severe as to embarrass the respiration of the patient. The paroxysms last only from one quarter to one half an hour and are not connected with bulimia. These attacks are said to be relieved by the ingestion of bread.

(3) *Anomalies of the sensations of hunger and appetite. Bulimia or Hyperorexia*.—Morbid increase of the sensation of hunger may occur as an independent neurosis, as a result of abnormal irritability of the centre controlling the sensation of hunger, or as a symptom of organic diseases (diabetes, Graves's disease, gastric ulcer, tapeworm). The affection expresses itself by violent sensations of hunger coming on suddenly, even shortly after the completion of a full meal, and if the desire for food is not immediately gratified, the patients exhibit signs of weakness, headache, pallor of the face, palpitation of the heart, roaring noises in the ears, and gastric diseases. In the intervals between the attacks hunger and appetite are normal, but there are cases in which bulimia may alternate with anorexia.

Depressive Sensory Neuroses.—(1) *Acoria* (ἀ, priv. ; κόρος, satiety).—Absence of the normal feeling of satiation, even after very abundant meals, without increase of hunger or appetite. The disease is generally secondary to neurasthenia, hysteria, or certain psychoses, and occurs in sexual neurasthenics. In these patients no impression whatever from the stomach informs them that they have eaten enough.

The treatment of bulimia when it is a secondary disease must have regard to removal of the primary cause, such as intestinal parasites, genito-urinary diseases, hyperacidity or ulcer, and any existing neurasthenia, hysteria or psychosis. The bromides are very valuable remedies to reduce the irritability of the hunger-centre. Arsenic, in the form of Fowler's solution, is highly recommended by Boas. Rosenthal recommends subcutaneous injections of extract of opium, and has seen good results in bulimia from cocaine hydrochloride. Morphine is a remedy that has been followed by good results in this affection. Electricity is sometimes useful. These patients should be watched during their eating and the meal interrupted when a normal

quantity has been ingested; thorough mastication and insalivation should be insisted upon. Strychnine and massage of the stomach suggest themselves as rational means of treatment.

(2) *Nervous anorexia*.—By anorexia is meant the entire absence of appetite and loss of the sensation of hunger. The superlative degree of this sensation is disgust and repugnance toward all food. There are probably no pathological conditions, either of the stomach or of any other organ of the body, in which anorexia is not occasionally met with. Very frequently chronic gastritis, incipient tuberculosis and carcinoma, begin with this symptom before any other signs or symptoms are manifest.

The primary object of treatment must be to improve the general nervous condition, to correct any existing fundamental disease, to act upon the psychological sphere by persuasion, suggestion, and firm but kind argument; and finally, to combat the anorexia itself directly. In anæmia mild preparations of iron are almost indispensable. The Weir-Mitchell rest-cure, together with the use of baths, massage and electricity, has, in many cases in our experience, produced happy results. When there is an absolute repugnance for food, or when the patient is insane, artificial compulsory alimentation should not be postponed too long.

Irritative Neuroses of Secretion.—(1) *Hyperchylia* (Hyper- or Superacidity, Hyperchlorhydria). See STOMACH, Functional Disorders of.

(2) *Periodical atypical flow of gastric juice* (Gastroxynsis (Rossbach), Gastroxie (Lépine), Gastro-succorrhœa Periodica (Reichmann)).—Gastroxynsis, or periodical flow of gastric juice, is an atypical secretion of the peptic glands, since it does not occur after a normal digestive stimulation, but rather when the stomach is empty. The attacks are associated with intense gastric distress, severe spasmodic pain, and vomiting of considerable quantities of very acid gastric juice. This peculiar neurosis is found almost exclusively among the educated classes, and particularly among those individuals who are subjected to unremitting mental exertion. It may also follow nicotine-poisoning and dietetic errors. The attacks occur, as a rule, in the middle of the night, or in the early hours of the morning. The patient has a very pale appearance, and the extremities are frequently cold. A few hours after the first vomiting of gastric juice the attack may be repeated, and again an equally large quantity of gastric secretion, containing no food-particles whatever, may be vomited. The patients are usually in good health, and severe thirst, loss of appetite, cephalalgia and great prostration, are characteristic symptoms. Periodical vomiting, when associated with hyperacidity, must be carefully distinguished from gastroxynsis.

Treatment includes, in the first place, the avoidance of stimulants and narcotics, alcohol and tobacco, as well as strong coffee. It is most essential that the patients should avoid mental overwork. Physical exercise should be indulged in moderately but systematically. The bicycle is an excellent remedy for periodical flow of gastric juice; and also horse-exercise, swimming, rowing, fencing, gymnastic exercises, and outdoor games. During the attack itself, the effects of the excess of acid should be counterbalanced by copious draughts of suspensions of calcined magnesina, ammonio-magnesium phosphate, or bicarbonate of sodium. When the vomiting has

occurred at short intervals, one should not hesitate to pass the stomach-tube and wash out the stomach.

Chronic continuous flow of gastric juice (Chronic Hyper- or Supersecretion (Riegel), Gastro-succorrhœa Chronica (Reichmann)).—The diagnosis of this hypothetical disease depends upon the presence of gastric juice containing hydrochloric acid and ferments in the fasting stomach.

The present writer inclines to the opinion that chronic hypersecretion is in the majority of cases not a spontaneous idiopathic neurosis, but a secondary symptomatic phenomenon. The most frequent causes are ulcer, pylorospasm and mechanical insufficiency.

Depressive Neuroses of Secretion.—

(1) *Subacidity* (Hypochlorhydria or Hypochylia). See STOMACH, Functional Disorders of.

(2) *Achylia gastrica* (absence of the secretion of gastric juice; nervous inactivity, atrophy of the stomach; anadenia ventriculi; phthisis ventriculi; achlorhydria).—The term *achylia gastrica* was first proposed by Einhorn to designate a class of diseases in which no gastric juice is secreted.

The affection is found to exist in two varieties—first, the primary idiopathic, possibly inherited, achylia; secondly, the acquired or secondary achylia. The primary idiopathic variety of achylia is characterised by the fact that absence of secretion is evident before any marked anatomical changes have occurred in the mucosa which could explain the loss of function. It is, therefore, as a rule, not regarded as a result of an actual disease, but as an individual peculiarity, possibly an inherited functional debility. There are undoubtedly persons in whom gastric secretion may be absent for years, or permanently wanting, yet who, apparently, may enjoy robust health. The majority of these individuals, however, have suffered from frequent dyspeptic complaints, which are partly of a purely nervous character.

The results of the examination of the gastric contents, in simple, uncomplicated achylia, are quite characteristic. The fasting stomach, examined in the morning before any food is taken, is empty. One hour after the Ewald test-breakfast the contents of the stomach have the same appearance as they have in the mouth before they are swallowed. Contents drawn in this manner are generally slightly acid, equal to the acidity found in the test-meal before it is eaten. The gastric contents, when filtered and mixed with hydrochloric acid sufficient to produce the reaction with Congo-red paper, cannot digest discs of egg-albumen. Milk taken by achylic patients may be drawn out twenty to thirty minutes afterwards uncoagulated. Persons with achylia may for many years have no subjective or objective disturbance of any kind; but sooner or later dyspeptic complaints arise, accompanied by eructation, fulness, and pressure after eating, gradually leading to attacks of severe gastralgia.

Achylia gastrica is due to two conditions (Martius): (1) a primary secretory debility of the stomach, constituting simple achylia gastrica; it has been supposed that bacterial infection is an ætiological factor in bringing about this state of the mucosa; (2) atrophy of the gastric mucosa (anadenia), with secondary achylia gastrica. The primary achylia gastrica is either congenital or developed on the basis of a very early predisposition. It is associated with inherited debility of

the nervous system, and prevails among so-called neuropathic patients.

It is logical to prescribe dilute hydrochloric acid for all these cases whenever it agrees well. When the appetite is absolutely lost, it may be restored by washing out the stomach with bitter tonics, such as gentian and quassia. It is expedient not to be too exacting with diet-orders. All foods should be well chewed, or preferably finely divided during the process of cooking. The more food that is ingested and well tolerated, the better for the patient in these cases.

A remedy little known, but a very valuable adjuvant in treatment for lack of digestive ferment, is the juice of fresh pineapple. This has decided proteolytic power, and, besides, is a pleasant, easily procured remedy. The ferment is active only in the fresh fruit, and is destroyed in the preserved pineapple.

Nervous dyspepsia (Leube).—(*Neurasthenia gastrica*, Ewald).—There are two main forms of gastric indigestion that may be classified as nervous dyspepsia: (a) that in which the nervous channels are sympathetically involved owing to anatomical changes in the stomach; and (b) that co-existing with an apparently normal anatomical state of the organ. Whatever the underlying basis or etiology of the disease, the ultimate symptoms can be ascribed to a functional sensory neurosis and over-excitability of the gastric nerves, which may become so acute that they react in a pathological manner upon the influence of normal digestive stimulation. *Neurasthenia gastrica* has been observed after intense emotional excitement, exhausting mental work, alcoholic and sexual excesses, and abuse of tobacco; also associated with pulmonary phthisis, nephritis, and malaria.

Jürgens has discovered total degeneration of the plexus of Meissner and Auerbach in forty-one cases of nervous dyspepsia. In one of the cases in which the sensory disturbances were predominant, and the intestinal functions involved also, this author found a distinct degeneration of the muscularis of the stomach and intestine.

The clinical picture is extremely variable. It is characteristic of nervous dyspepsia that the gastric distress is directly dependent upon the ingestion of food—that it occurs, as a rule, only after meals, and not on an empty stomach. Furthermore, the quality and quantity of the food and dietetic errors exert little influence upon the dyspepsia, but the sensations of the patient are very much under the influence of the emotional state. The suffering is most severe when the *neurasthenia gastrica* is accompanied by hyperacidity. In this case it increases during the second period of digestion, as the acidity becomes greater. Such types are relieved by the administration of alkalis, which, however, are useless with achylia. The epigastric region is not very sensitive, nor are there any characteristic pain-points discoverable.

There may be normal gastric secretion, hyperacidity, or inactivity. When inactivity exists, the ferments can still be demonstrated in the gastric contents. This important fact will serve to distinguish this type of nervous dyspepsia from typical achylia gastrica. The peristalsis of the stomach is in most cases undisturbed, but there are cases in which temporary motor insufficiency occurs.

There are pain and pressure in the head, giddiness, tinnitus aurium, flashes before the eyes, rapid pulse, exhaustion, cool extremities, attacks of faint-

ing, palpitations of the heart, dyspnoea; obstinate constipation is often met with; also borborygmi and flatulence.

The course of the disease is chronic, and it may, in severe cases, by continued and progressive loss of strength and emaciation, prove fatal. When the cause, such as sexual excesses, abuse of alcohol and tobacco, bodily and mental over-exertion, can be removed, the resulting nervous dyspepsia may be permanently cured.

Heterochylia (*érepes*, other; and *χυλός*, secretion).—This term is suggested by the writer to denote a rapidly alternating state of secretion, occurring chiefly in nervous dyspepsia.

Nervous dyspepsia with inactivity may be confounded with chronic gastritis or carcinoma, and nervous dyspepsia with hyperacidity may be confounded with ulcer, while other forms again may bear a striking resemblance to atony or myasthenia. For the separation of chronic gastritis from nervous dyspepsia, the following facts are of importance: Chronic gastritis is accompanied more frequently by vomiting; the stomach contains large quantities of mucus and a few streaks of blood; we may also have stagnation of the contents. The course of chronic gastritis is more uniform and typical, and the dyspeptic symptoms are directly influenced by the quality and quantity of the ingesta. In carcinoma the distress is present at all times, even on an empty stomach, vomiting is frequent, and the ferments are absent when the HCl-secretion is lost. In nervous dyspepsia the ferments are still present, though hydrochloric acid may be absent. The differentiation of nervous dyspepsia from ulcer becomes difficult only in those cases in which there has been no hæmatemesis. The constant dependence of gastric pain upon the food, and the sharply circumscribed painful points in the epigastric region and in the back, are unmistakable criteria.

In treating these cases the fundamental causes of disease should be sought out, and, if possible, removed. In those forms of nervous dyspepsia which depend upon an undue excitation of the nervous system, due to sexual excesses, abuse of alcohol and nicotine, or excessive mental or bodily exertion, improvement cannot be hoped for unless these states are remedied.

The physician must gain the absolute confidence of his patient. For this purpose he should show a sincere interest in the suffering of his patient, even if, after a repeated and thorough examination, he should become convinced that his complaints are exaggerated.

A course of mild gymnastic training—riding, rowing, fencing, or the bicycle, moderately used—is a better means of promoting appetite and regular evacuations than drugs. A sojourn in the mountains or at the sea-side is a great help. Massage improves the nutrition of the muscles and nerves, and favours a vigorous circulation, metabolism, and regular evacuations. Cold sponge-baths, also, taken in the morning immediately after rising, have a bracing effect. The favourable effects of hydrotherapeutic methods do not become manifest until they have been applied for two or three weeks. They are then followed by improvement in the appetite and sleep. When the insomnia is persistent a warm salt bath, at the temperature of the body, often acts similarly to an hypnotic.

Irrigations and douches of the gastric mucosa are used to reduce the hyperæsthesia of the gastric

nerves, and for this purpose carbonated waters are preferable to still waters. Galvanism of the abdomen and the spinal region and general faradisation are also applicable in these cases.

The physician must see that the articles of food possess considerable variety and are well cooked and appetising. Experience is the best guide.

Large enemata of pure olive-oil (300 c.c. at a time), combined with abdominal massage, are sometimes curative in the nervous constipation, particularly the membranous colitis present in these patients. Perhaps the most effective treatment, on the whole, is that designated as the Weir-Mitchell rest-cure, a combination of hydrotherapeutic, electrical, and dietetic treatment, with gymnastics, rest, massage, and as much sleep as possible.

The drugs that have been employed in neurasthenia gastrica are the tonics, sedatives, and hypnotics. In complete acidity the basic orexine (5 grains three times a day) has been much lauded by Penzoldt, but the writer has not been able to convince himself that this drug can restore the secretion of HCl. The fluid extract of condurango (one teaspoonful three times a day) and the bitter tonics, calumba, gentian, quassia, in doses of one drachm three times a day, are sometimes of value. The remedy we have most faith in is the sulphate of strychnine ($\frac{1}{10}$ of a grain three times a day continued for one month at least). When malaria is associated with the nervous dyspepsia quinine is the best remedy.

JOHN C. HEMMETER.

STOMACH, Perforation of, and its Results.—Perforation of the stomach is but rarely the result of mechanical injury by swallowed bodies; it is usually the result of disease—sometimes of the action of corrosive poison, but more commonly of ulcer or carcinoma of the organ. It occurs in from 13 to 18 per cent. of all cases of ulcer, and is a comparatively rare event in cancer. Perforation in ulcer of the stomach is most frequent in young females, and is more likely to occur if the ulcer is situated towards the anterior surface, where it is not in contact with any solid organ. Eighty per cent. of ulcers on the anterior surface are said to perforate. The results of perforation in ulcer depend on adhesions of the base of the ulcer to a solid organ, such as the pancreas, spleen, or liver, or to the abdominal wall. If the base is completely adherent to the solid organ, and perforation occurs, abscess may be formed in the liver, pancreas, or spleen. In other cases there is abscess-formation in the tough adhesions uniting the ulcer to the transverse colon or to the anterior abdominal wall. In yet other cases perforation may have one of two results: the contents of the stomach may be extruded into the general peritoneal cavity, or they may be shut into the upper peritoneal cavity, sometimes by adhesions, though not in all cases. General peritonitis may be the result of the first, and a localised abscess (subphrenic) the result of the second event.

The clinical aspects of perforation are best considered under three headings: (1) Perforation into an adhesion; (2) Perforation into the general peritoneal cavity, succeeded by general peritonitis; (3) Perforation into the upper peritoneal cavity, succeeded by formation of a subphrenic abscess.

Rupture of an ulcer usually occurs without warning; taking place after a meal, not uncom-

monly of indigestible food, or after some sudden exertion, perhaps on a full stomach. It may occur in a fit of sneezing or coughing, or in vomiting, and has been known to follow the administration of strong purgatives. It sometimes occurs in the early morning, on getting out of bed, but it more frequently happens when the patient is walking about in the day. The earliest symptom is that of acute pain, referred to the upper part of the abdomen, with the sensation of faintness; sometimes there is nausea and vomiting. Acute pain is sometimes absent, and the onset of perforation is shown only by an exacerbation of the pain previously experienced after food. There may be slight hæmatemesis. The pulse is extremely rapid, and there may be dyspnoea, and the features soon become pinched, drawn, and haggard.

1. **Perforation into an adhesion** may be accompanied by the symptoms which have just been described, and by no others, except, perhaps, a slight rise of temperature in the next few days. There are no physical signs of the extrusion of the stomach-contents into the peritoneal cavity, and, after a period of treatment, the patient gets well without surgical interference.

2. **Perforation into the general peritoneal cavity.**—The escaped stomach-contents may either be retained in the upper part of the peritoneal cavity—that is, between the diaphragm and the upper abdominal organs (liver, stomach, spleen), or they may pass to the lower part of the cavity. The contents consist of gas and liquid, usually acid. In perforations of ulcers on the anterior surface, when there are no adhesions, the stomach-contents enter the lower part of the peritoneal cavity; but, when there are adhesions, or when the perforation is on the posterior surface, the stomach-contents may be retained in the upper part of the abdomen. The clinical features of cases when there is extrusion of a large quantity of stomach-contents into the peritoneal cavity are very characteristic. The patient lies in bed, on the back, sometimes with the knees drawn up; the features are pinched or haggard, and expressive of pain; the pulse, some hours after the perforation, is small, very rapid, and compressible. An examination of the abdomen shows uniform and sometimes great distension, with the absence of abdominal respiration and a great increase of tension in all parts. Percussion gives a tympanitic note over the whole of the front of the abdomen, including the hepatic area, and, not infrequently, in the flanks, a dull note, indicative of fluid. A rectal examination may show a tense swelling in Douglas's pouch.

3. **Perforation into the upper peritoneal cavity.**—In this event, the physical signs are discovered chiefly in that region, and on one or other side. There is an absence of abdominal respiration, and the tenseness of the abdomen is chiefly marked in the upper part, in the epigastric and hypochondriac regions. Tenderness may be acute over this region. Percussion reveals the disappearance of the liver-dullness above the costal margin. Here there may be a tympanitic note, which may extend upwards over the sternum, as high as the fourth rib. The heart's apex-beat may be displaced, directly outwards or upwards. In the later stage of the illness general peritonitis supervenes, if the perforation is into the general peritoneal cavity, or a subphrenic abscess if the perforation is into the upper peritoneal cavity. In the former

case the fever which ensues is not, as a rule, high—ranging from 100° to 102° . The severe general symptoms continue, and vomiting and retching may be frequent, as well as a distressing hiccup. Great tenderness may develop, especially over the lower two-thirds of the abdomen.

Subphrenic Abscess.—The subphrenic abscess is below the diaphragm, and is shut in by adhesions between the stomach and the surrounding organs. It may occur from perforation of an ulcer in any part of the duodenum and stomach, except the lower part of the anterior wall. If the ulcer is near the pylorus the abscess is formed on the right side; if it is on the posterior wall, or on the anterior wall, near the cardiac end, the abscess is formed on the left side. The falciform ligament of the liver forms the boundary of the abscess. In twenty-nine cases of subphrenic abscess, a perforating ulcer had formed in the small curve, near the cardiac end in twelve cases, on the posterior wall in ten cases, and on the anterior surface in seven cases.

Subphrenic abscess is not always due to perforating ulcer of the stomach or duodenum. About 40 per cent. are due to this cause; about 4 per cent. to perforation in cancer of the stomach or oesophagus, while the other cases are due to appendicitis, gall-stones, perinephritic abscess, splenic abscess, injury, and hydatid of the liver. Some of these conditions produce an abscess in the subperitoneal cellular tissue, as in the extension upwards of abscesses from the appendix or kidney. An intraperitoneal abscess, however, results from rupture of a gastric or duodenal ulcer, or of an hepatic or splenic abscess, or an abscess of the gall-bladder. It is the intraperitoneal abscess which will be discussed here.

MORBID ANATOMY.—In right subphrenic abscess the left wall is formed by the falciform ligament of the liver. On the right it is bounded by the thorax, below by the liver, and above by the diaphragm. In left subphrenic abscess the boundaries are as follows: above the diaphragm, below the left lobe of the liver and the anterior surface of the stomach; in front the abdominal wall, united by adhesions to the anterior surface of the stomach; on the right side the falciform ligament, and on the left adhesions between the cardiac end of the stomach, the spleen, and the diaphragm. Although originally right- or left-sided, a subphrenic abscess may infect the opposite side, and thus occupy all the space beneath the diaphragm. In this case, the falciform ligament is sometimes perforated, and sometimes intact.

The formation of a large abscess in this neighbourhood compresses the lung above it, displaces the heart either away from it or directly upwards, and if on the right side pushes down the liver, even as far as the umbilicus. The contents of the abscess vary somewhat. In all cases pus and gas are present; in some cases they are sweet, in other cases foul-smelling, owing to putrefactive decomposition. The gas in the abscess is derived partly from the gas present in the stomach at the time of perforation, and partly from putrefactive decomposition occurring subsequently in the extruded stomach-contents. Remains of food may be found in a recent abscess.

The perforation in the stomach shows various appearances. It is usually patent, but it is sometimes closed, a pucker being left in the ulcer showing where it had perforated.

EFFECTS.—An abscess in this situation, foul as it often is, will excite inflammation in the neighbourhood, and therefore the usual accompaniment of subphrenic abscess is an affection of the pleura and lung above it. Thus pleurisy, with or without effusion, or empyema with sweet or fetid pus, may be present on the same side as the abscess; the lung may be pneumonic, or an abscess may be developed in it, and extensive destruction of the lung may occur in consequence, or gangrene may result. Affections of the pleura and lung may occur without a direct connection between the abscess and pleura; in some cases, however, the pleura and abscess communicate through an opening in the diaphragm. Of 45 cases collected by Maydl, the pleura was normal in 11, adherent in 10, and contained a serous fluid in 9, and pus in 15 cases.

SYMPTOMS.—The development of a subphrenic abscess is preceded by the symptoms which have been described as occurring in perforation of the stomach or duodenum and in the peritonitis resulting; that is, there is sudden pain followed by fever, tenderness of the abdomen, and the other signs of peritonitis. But there are soon signs of localisation of the inflammation to the upper part of the abdomen; and in the fully developed subphrenic abscess the symptoms resemble those of pneumothorax; there being fever of varying intensity, usually from about 100° to 102° F., with dyspnoea, but, as a rule, no cough or expectoration. The general appearance of the patient indicates in many cases the presence of a severe illness, and there is bodily weakness, with an expression of pain and a drawn countenance. The chief signs of the disease are elicited by physical examination.

PHYSICAL EXAMINATION.—*Inspection.*—The heart's apex is slightly displaced horizontally away from the disease; in right subphrenic abscess towards the left, and in left subphrenic abscess either upwards or towards the right. There is but slight bulging of the side, the intercostal spaces being practically normal. The respiratory movements vary somewhat; in some cases there is cessation of the abdominal movements, in others these are normal; but there is deficient movement as regards expansion and elevation on the same side of the thorax as the abscess.

Palpation.—Palpation confirms inspection in the above points, and in some cases a thrill may be elicited over the abscess by a sudden jerking movement given to the abdominal wall. Tenderness may be completely absent. Examination of the abdomen by palpation also reveals the displacement of the liver, the lower edge of which in right subphrenic abscess may be discovered nearly on a level with the umbilicus. In left-sided abscess the spleen is not usually felt, being pushed against the thoracic wall.

Percussion.—Percussion gives very varying signs. Over the lower part of the thorax on the side of the abscess, there is a tympanitic note over an area varying in extent according to the amount of gas contained in the cavity. The upper limit of this area is rounded and sharply marked off from the resonance obtained over the lung. Below, on the right side, the liver-dulness over the lower thoracic wall may be completely absent, a tympanitic note only being elicited; and when present this obliteration of the thoracic liver-dulness is an important sign of subphrenic abscess. Posteriorly in the thorax there may be dulness when the patient is in the recum-

bent position; and this dulness shifts its position with alteration of the posture of the patient. On the left side, similar variations in resonance may be discovered, and in front there is not infrequently tympanitic resonance in the upper epigastric region, and this may be continuous with an area of similar resonance over the cardiac area.

Auscultation.—Commencing from above, vesicular breathing is heard from below the clavicle down to the upper margin of the tympanitic resonance, diminishing in intensity below owing to the collapse of the lung. Over the area of the tympanitic resonance normal breath-sounds are quite absent and may be replaced by amphoric breathing. Posteriorly the breath-sound is absent over the area of dulness. Adventitious sounds may be elicited, viz. metallic tinkle by tapping the abdomen near the abscess, or the bell-sound by the usual means.

Variations in these physical signs are observed when, in addition to the subphrenic abscess, the pleura or lung above is affected. There may be above the abscess signs of fluid in the pleura, viz. dulness, with absence of breath-sounds; or there may be tubular breathing and increased vocal resonance with crepitations, showing consolidation of the lung. In some cases the signs of pneumonia may obscure those due to subphrenic abscess.

On aspirating the abscess both gas and fluid are obtained, the latter having the character previously described. If a mercurial manometer be connected with the abscess-cavity it will be found that there is an increase of pressure during inspiration, and a decrease during expiration (Pfuhl). This is directly opposite to what occurs when a manometer is introduced into the pleural cavity, and is a distinguishing point between an abscess above and one below the diaphragm. The test, however, does not always succeed.

DIAGNOSIS.—1. *As to the Presence of Subphrenic Abscess.*—The chief point in the diagnosis to be settled is whether the cavity is above or below the diaphragm—whether it is a pyo-pneumothorax or a subphrenic abscess. The physical signs are practically the same in pyo-pneumothorax and in subphrenic abscess; that is, they are the signs of a cavity containing fluid and gas. But the subphrenic abscess differs from pneumothorax in producing less displacement of the heart and less bulging of the side affected. These two physical signs, however, vary so much in pneumothorax that no great stress can be laid on them as a distinction between the two diseases. Of more importance are the physical signs of tuberculosis which are present in pneumothorax, when this occurs in either the early or the late stages of the disease, viz. consolidation or excavation of one or other apex. The history of the case is of great importance. The absence of cough and expectoration and the mode of onset of the disease demonstrate its occurrence in the upper part of the abdomen and not in the chest. From other chest-conditions subphrenic abscess is readily distinguished by the physical signs which have been dealt with. It must be borne in mind, however, that diseases of the lung and pleura may complicate subphrenic abscess; and physical signs may be obtained posteriorly, and above the upper line of tympanitic resonance, indicating an affection of the lung or pleura—pleurisy with effusion, empyema, pneumonia, or pneumonic abscess.

2. *Diagnosis as to the Cause of the Subphrenic Abscess.*—The diagnosis as to whether perforating

ulcer of the stomach or of the duodenum is the cause of the abscess is not usually possible; it rests on the previous history of ulcer as regards pain directly after food, vomiting, hæmatemesis, melæna, &c., and on the occurrence of the symptoms of perforation as previously described. The history of ulcer may be absent, but the symptoms of perforation always precede the development of a subphrenic abscess due to ulcer.

The cases due to carcinoma may be diagnosed from the presence of symptoms peculiar to cancer of the œsophagus or stomach. It is in the later stages of carcinoma that perforation occurs, and it is not a common event.

Abscesses occurring on the right side of the upper part of the abdomen may be in the liver or may be subphrenic and due to typhlitis and perinephritis (when it is extra-peritoneal), or to rupture of a gastric ulcer (when it is intra-peritoneal). On the left side, the abscess may be due to a gastric ulcer or to disease of the colon or kidney. Great difficulty is sometimes experienced in the diagnosis between a collection of pus in the liver and one between the liver and the diaphragm. In the latter case the abscess may contain gas; in the former there is either the history of dysentery preceding the illness, or there may be the negative history of a benign tumour (hydatid). The exact nature of some of these cases is, however, unrecognised before an operation. With typhlitic abscess the diagnosis rests on the previous history of disease localised in the right iliac fossa and the presence of actual signs of disease in that situation, viz. induration or abscess. With renal abscess, the diagnosis may be made clear by a history of the symptoms of calculus, and by the presence of pus in the urine.

COURSE.—Subphrenic abscess is a serious condition, which ends fatally either by septic poisoning or more commonly by producing putrid empyema and pneumonic abscess. In some cases the abscess discharges itself through the lung, and although this must be considered the most favourable natural mode of cure of the disease, yet it is one which entails a prolonged illness to the patient, and which sets up destructive changes in the lung. In other cases the abscess bursts into the general peritoneal cavity, causing death by general peritonitis. The rapidity with which subphrenic abscess kills varies greatly; it is to some extent dependent on the affections of the lung which complicate it. Of 178 cases collected by Maydl, 98 died without an operation, 6 healed without operation, and of the 74 operated upon 35 died and 39 recovered. It may, therefore, be looked upon as practically a fatal disease unless relieved by operation.

SIDNEY MARTIN.

STOMACH, Ulceration of.—INTRODUCTION.

The term 'ulcer of the stomach' is sometimes reserved for that particular kind of ulceration known under the names of 'perforating,' 'simple,' 'round,' or 'peptic' ulcer. Ulcers are also met with in the course of various disorders of the stomach as well as of certain of the general infective diseases. Acute ulcers prone to rapid healing occur in some forms of acute gastric catarrh, and chronic ulcers varying in size, depth, and duration are seen in certain forms of chronic gastric catarrh. Superficial erosions and ulcers are not uncommon in atrophied conditions of the mucosa. Ulcers may also follow injury, the ingestion of corrosive poisons,

and venous congestion due to the portal obstruction of certain hepatic and cardiac diseases. They are even rarer in general infective diseases, such as yellow fever, typhoid fever, diphtheria, pyæmia, anthrax, small-pox, and tuberculosis, and when present are generally multiple. Their mode of origin is probably similar in all these cases, viz. by a hæmorrhagic infiltration of a part of the mucosa, to which the stomach is very liable, impeding and arresting the circulation therein, and thus allowing of its digestion and erosion through the action of the gastric juice. Their first stage is probably identical with superficial erosions (hæmorrhagic erosions) which involve only a slight thickness of mucosa and generally heal rapidly. In some cases, however, where the nutrition becomes more extensively impaired, the ulceration spreads and may even lead to perforation. Ulceration also occurs in cancer of the stomach (see p. 1600). The signs and symptoms to which these different forms of ulcers give rise may closely simulate those of the simple or perforating form, while in other cases they are subsidiary to or even masked by those appertaining to the disease of which they are but a part.

Simple Gastric Ulcer.—**DEFINITION.**—A disease of the stomach characterised by the presence of an ulcer of varying depth, beginning in the mucous coat and attended by pain, vomiting, and hæmatemesis.

ÆTIOLOGY.—The exact causation of gastric ulcer is still an unsolved problem, notwithstanding the many ingenious theories which have been advanced. There is a strong weight of evidence in favour of its being produced by many very different conditions. Thus any agent, whether nutritional, toxic, chemical, mechanical, or bacterial, of sufficient power to interfere seriously with the vitality of a part of the stomach-wall, may cause its ulceration. Indeed, more than one such agent may sometimes be at work. The chief facts and theories supposed to influence the causation of the ulcer may be arranged as follows:

1. *Frequency.*—*Post-mortem* records, counting together both open ulcers and scars, give a considerably higher proportion of cases than do clinical records, viz. 4 to 5 per cent., as against .75 to 1 per cent.; and inasmuch as the former, though open to fallacies, are less liable to error than the latter, it may be tentatively suggested that about 4 per cent. of all individuals are the subjects of gastric ulcer at some time of their life. For many reasons it is only possible to reach the actual frequency approximately. The 5 per cent. generally accepted by authorities is probably rather high.

2. *Sex.*—It is more frequent in women than men. There is some discrepancy between the *post-mortem* and clinical results. So far as *post-mortem* records go, the proportion of three females to two males would seem to be fairly correct. On the other hand, clinical records give a much higher proportion of women, two or three or even more females to one male. The discrepancy is probably due to the fact that the disease is more frequently fatal in men than in women. The acute form which is so prone to heal occurs almost exclusively in young women.

3. *Age.*—In determining the frequency of ulcer at any age, attention must be paid to the time at which the disease apparently began, hence clinical records are in this respect likely to be more correct than *post-mortem* results. There is now a general

consensus of opinion that it is most frequent, in the case of women, from the age of twenty to thirty, and in men from that of thirty to fifty. It is rare before puberty.

4. *Occupation.*—This cannot be held to have much influence in the production of the ulcer, though cooks, housemaids, metal-turners, tailors, and all workmen whose work entails mechanical pressure upon the epigastrium, are held by some authorities to be more prone to the disease. It is also more common among the poor, probably because of their lower state of general nutrition.

5. *Associated diseases.*—The simple ulcer of the stomach has been met with in a number of diseases, such as chlorosis, amenorrhœa, syphilis, tuberculosis, malaria, chronic cardiac and renal disease, and a causal relationship between them has been suggested. The evidence in its favour is slight, except in the case of chlorosis, in which ulcer of the stomach is of very frequent occurrence. This is particularly true of the acute variety seen in young women. Chlorosis is, however, only rarely accompanied by gastric ulcer, and, moreover, when the two occur together it is generally in the later and not in the earlier stages of the disease, so that the ulcer may be merely related to the long-standing poverty of the blood which is common to all anæmias. A causal relationship between them can neither be denied nor proved at the present time.

PATHOGENESIS.—It is when we seek for the actual exciting causes that we find the greatest diversity of opinion. Practically every local condition which might possibly cause ulceration has been brought forward in explanation, e.g. injury, inflammation, impeded nutrition through one or other change in the arteries, a congestion of the veins, an affection of the nerves, and an alteration in the composition and conditions of the gastric secretions. Since the simple ulcer is met with only in the stomach and adjacent parts of the œsophagus and duodenum it is reasonable to suppose that the corroding action of the gastric juice has much to do with its production. So long as the stomach-wall preserves a certain standard of vitality, it resists this action, but if it falls below this standard in any area, destruction of that area is likely to follow from the digestive and corroding action of the gastric juice upon it. The histological appearances of the edges and floor of the ulcer support this view, as they exhibit in all cases where the process is still advancing, purely necrotic phenomena with an entire absence of all signs of inflammation. The conditions which may bring about such a lowered state of vitality in a limited area of the gastric wall naturally then require attention. They are as follows:—

(a) *Embolism.*—The arteries of the stomach show a free collateral circulation, but it has been thought that if a branch be occluded by an embolus, the part of the stomach-wall supplied by that branch will necrose and, becoming eroded, form an ulcer. The appearances presented by the ulcer are in such complete harmony with the theory of its production in this manner, that it passed unchallenged for a long time. It was only when it was realised that such an embolism had never been demonstrated, that in the majority of cases of gastric ulcer the ordinary sources of emboli did not exist, and that emboli artificially introduced into the blood-stream rarely entered the coronary arteries, that doubt was thrown upon the explanation.

(b) *Thrombosis*.—An acute thrombosis of a branch of the coronary artery is theoretically an equally satisfactory explanation, but practical experience shows that it rarely, if ever, occurs. The gastric arteries are usually healthy in stomachs which are the seat of gastric ulcer, and thrombosis does not occur in healthy arteries. It is true that in certain cases the gastric arteries are diseased, and various forms of endarteritis, amyloid disease, and other degenerations have been found; but while these may help to account for the formation of individual ulcers, they cannot be held to throw light upon its production generally.

(c) *Spasm of the arteries*.—A local spasm might lower nutrition in a definite area of the stomach-wall, and thus produce ulceration, but the existence of such a spasm is purely hypothetical and its production difficult of explanation.

(d) *Venous engorgement*.—This is not a common condition in gastric ulcer, whereas it is common in other morbid states, e.g. hepatic cirrhosis and cardiac disease, in which gastric ulceration is uncommon. It may, however, at times assist in its production by causing a hemorrhagic infiltration of a local area. It has also been suggested that spasm of the muscular coat may lead to a similar condition by compression of the veins.

(e) *Nervous influences*.—The nerves of the stomach influence its blood-vessels, its muscular contractions, the secretion of its juices, and its general state of nutrition. The result of stimulation of the gastric mucosa in health is a relaxation of vascular tone, and therefore dilatation; but it is conceivable that morbid stimuli might produce the opposite result, viz. an increase of tone, amounting to spasm and so to ulceration. It is also conceivable that they might bring about irregular contractions of the muscular coats of the stomach-wall, and hence ulceration through compression of the veins. The loss of the normal trophic influence by a part of the stomach-wall has also been suggested, but all these theories entirely lack evidence in their support. The alterations in the gastric secretions brought about by nervous or other influences still remain to be considered. The pepsin is unaltered, but the hydrochloric acid is usually increased in amount. This increased secretion of hydrochloric acid is held by many to be the chief causal factor in the production of gastric ulcer, but if so, it is difficult to explain why it should act over such a strictly limited area. Some explain this by suggesting that the abnormal nervous impulses act only on a circular group of glands. More serious objections still consist in the facts that hyperacidity is not a constant feature in the disease, being generally absent in the acute variety and in about one-third of the chronic cases, and that hyperacidity occurs as a functional disease of the stomach in which ulceration is rare.

(f) *Inflammation*.—Ordinary inflammations of the stomach are only accompanied by slight ulcers which quickly heal. Severe inflammations may cause necrosis or hemorrhagic infiltrations and subsequent ulceration of the mucosa, but ulcers produced in this way show inflammatory conditions in their edges and floor, and affect the fundus and greater curvature rather than the pyloric region and lesser curvature. Inflammation of the solitary glands has been advocated by Fenwick as the origin of some forms of gastric ulcer, and this view is probably correct, as such ulcers by extension may easily assume the form of acute perforating ulcers.

(g) *Bacterial necrosis*.—Bacteria have occasionally been found in the margins of an ulcer and in partially detached sloughs, and it is further known that the ingestion of large quantities of fluid may for a time inhibit the bactericidal properties of the gastric juice, so that active bacteria may come to settle in the stomach-wall. It is no argument against a bacterial causation that no specific micro-organism has so far been described, for any of the ordinary inflammatory bacteria of sufficient virulence to cause necrosis elsewhere might act in the same way in the stomach. Indeed we know that certain bacteria, e.g. those of typhoid fever and tuberculosis, can cause the same necrosis in the stomach-wall which they produce in other parts of the body. In these cases, however, the general disease is of great importance, whereas in gastric ulcer the patients are generally otherwise healthy. Moreover, bacteria have been found only in the necrotic tissue of the edges and floor of the ulcer, never in the healthy tissue around, or in the margins of a fully formed ulcer, and hence it is at least as probable that they are subsequent to, as that they are prior to the necrosis. Numerous bacteria enter the stomach with the food, and will naturally settle in a necrosed area.

(h) *Mechanical, Chemical, and Thermal injuries*.—Mechanical injuries of the stomach usually heal rapidly without ulceration, but they may result in hemorrhagic infiltration and ulceration. Such ulcers are of acute formation, and usually heal rapidly. Chemical injuries such as follow upon the ingestion of corrosive fluids may result in necrosis or intense inflammation followed by ulceration. Such ulcers may come to be indistinguishable from the ordinary gastric ulcer. Thermal injuries from the ingestion of hot food or liquid have been shown experimentally in dogs to produce hemorrhagic infiltration and ulceration, but there is no clinical evidence to show that they do so in man.

MORBID ANATOMY.—The number, situation, size, shape, and general appearances of the ulcer have to be carefully described, and a distinction drawn between ulcers of acute and chronic formation.

1. *Number*.—In the chronic variety a single ulcer is met with about nine times more frequently than a greater number; but in the acute variety multiple ulcers are at least as common as a single ulcer. When multiple, two are more frequent than three, three than four, and so on. In exceptional cases large numbers are present.

2. *Situation*.—The posterior surface near the lesser curvature in the pyloric region is the site in about three-fourths of all cases. The chronic form occurs here fully three times oftener than elsewhere, but the acute form is as frequent in the middle or cardiac regions as it is in the pyloric. Occasionally the ulcer involves the pyloric, or much more rarely the cardiac orifice, either in the primary ulceration or the subsequent cicatrisation, causing obstruction to the entrance or exit of food. When the ulcers are multiple, they mostly affect the posterior surface, though when acute one or more are usually seen also on the anterior surface. Puckerings and cicatrices representing former ulcers are seen much more frequently than the open ulcer, especially in the middle and cardiac regions; three to one is the generally accepted proportion, but it should not be looked upon as more than an approximate estimate. When present along with open ulcers, they are generally situated near them. They are rarely seen on the anterior surface, as such ulcers are prone to perforate

and not to heal. When the preceding ulcer has been small, they are very difficult to detect; when large, there is a smooth central depression devoid of mucosa, much smaller than was the original ulcer, for healing has caused considerable contraction and drawing together of the mucous margins.

3. *Size*.—The acute are usually smaller than the chronic. The former are often likened in size to a threepenny-piece, and the latter to a sixpenny- or shilling-piece. This rough and ready method has tended to exaggerate their size. A threepenny-piece is over half an inch in diameter, whereas the acute ulcer often does not exceed a quarter of an inch, and the chronic form generally measures half to one inch, sometimes less and often more. Exceptional cases with diameters of several inches have been seen.

4. *Shape*.—The smaller ones, particularly the acute, are usually circular or oval, while the larger ones, almost always chronic, are often oval, triangular, quadrilateral, scalloped or irregular in shape. These last usually arise from the confluence of several adjacent ulcers. Horseshoe or girdle-shaped ulcers generally occur in the middle or lower third of the stomach, and by their large size and extensive contraction cause hour-glass and other deformities of the organ.

5. *Edges*.—The edges of the acute ulcer are soft, smooth, and clean-cut as if punched out, while those of the chronic variety are firm, thickened and fibrous in appearance, or firm, smooth, and depressed. The depth generally reaches some part of the muscular coats, but often penetrates further to the serous coat. In the acute variety the diameter usually gets markedly less as each successive coat is penetrated, so that the ulcer presents a typical terraced appearance, as if a smaller punch had been used for each coat. In the chronic variety, the terraced appearance is not seen, but a generally smooth, firm, and sloping surface from the mucous margin to the floor. Thus both varieties have a typical funnel shape. Occasionally the chronic form shows great thickening in the walls and floor.

6. *Floor*.—In the acute variety the floor and edges may show a little hæmorrhage or some necrotic tissue, but usually they are formed by one of the but little altered coats of the stomach—submucous, muscular, or serous. The serous coat may show on its outer surface some congestion and fibrous exudation; more pronounced if it be actually penetrated. If the perforation be small a clear watery mucous fluid, which fills and distends the stomach, usually wells out of the aperture; if large, the swollen and congested mucous coat may also pour through it. In the chronic variety the floor is usually smooth and firm, if formed by any of the coats of the stomach, the serous coat often showing considerable fibrous thickening. Perforation may produce a general peritonitis, adhesion of the affected part of the stomach to some neighbouring organ, or a local abscess (see pp. 1619 to 1621).

7. *The condition of the stomach-wall in the neighbourhood of the ulcer*.—The state of the surrounding mucous membrane is of the greatest interest. Judging from *post-mortem* appearances it is generally quite unaltered right up to the very margins of the ulcer, but certain observations made during life assert that this is really not a true picture, inasmuch as it has lost all traces of the inflammatory changes of greater or less severity which, always present during life, rapidly disappear after death. If there be such a gastritis of any severity and extent,

whether concomitant with or subsequent to the ulcer, and the congestion and swelling have been described as great, it would help to explain the symptoms, and, moreover, be of much practical importance in treatment. The general experience of surgeons, however, who are rapidly becoming better informed of the appearances of the stomach in various morbid states, does not appear to support the existence of much, if any, gastritis in most cases, and hence we are at present inclined to believe that the *post-mortem* picture is a fairly faithful one.

8. *Microscopical appearances of the ulcer*.—These can only be very shortly referred to here. Suffice it to say that in the acute and advancing chronic forms there are signs only of necrosis, none of inflammation. When healing has begun the inflammatory reaction in the floor and edges becomes well marked, but this is a result not a part of the ulcerative process. The edges become adherent to the floor, and the new tissue is always fibrous, little regeneration of glandular or muscular tissue occurring in most cases. There is no doubt that some regeneration of the glandular part of the gastric mucosa does occur in certain circumstances; and it is probable that it does so at any rate in the small and superficial gastric ulcers which may thus heal without a recognisable scar.

SYMPTOMS.—1. There may be practically no symptoms, as shown by the fact that an unsuspected gastric ulcer or cicatrix is sometimes discovered at a *post-mortem*, or by rapidly occurring death from its perforation in persons previously in the best of health. This is spoken of clinically as the 'latent type.' 2. There may be only various symptoms of dyspepsia, e.g. discomfort and fullness after food, distension and flatulence, acidity, &c. The appetite is generally good, but thirst is often great. Constipation is the rule. These indications may last for months or years without the appearance of any of the characteristic symptoms. This is the clinical 'chronic dyspeptic type,' which includes also cases attended by pain and occasional vomiting. 3. The characteristic symptoms are pain, vomiting, and hæmatemesis; these require detailed consideration.

Pain.—This is the most constant of the symptoms, although it may be entirely absent. In its typical and severe forms it is more characteristic of the chronic than of the acute variety. The situation, intensity, time of onset, and duration of the pain are of importance. (1) *Situation*.—The pain is usually distinctly localised to the epigastric region below the ensiform cartilage, though in the more chronic cases it may be lower and more diffused. It is also, especially in the latter cases, often experienced at the back, in the middle line between the sixth and eighth dorsal vertebrae, or lower and to the left. It is sometimes described as passing through from the front to the back. It also sometimes radiates to the sides of the body (see p. 1142), and up to the shoulders, and even down the arms. Even when it is strictly localised or influenced by the position of the patient it is not a safe guide to the exact site of the ulcer. The passage of a weak electric current from the epigastrium or left hypochondrium (negative pole) to the lower dorsal region (positive pole) soon after a meal is more trustworthy but often fails. The negative electrode is moved about on the skin of the epigastric and left hypochondriac regions and gives rise to a slight tingling sensation only, until it comes to be placed over the site of the ulcer, when severe pain

is felt. (2) *Intensity*.—In the acute and in the early stages of the chronic variety, the pain is usually not very intense, but as the chronic form progresses the pain usually becomes more severe, and is described as heavy, boring, burning, or stabbing; sometimes it is spasmodic or colicky. It is often so severe that no drug except morphine gives relief. It is frequently influenced favourably by the position of the patient, being less when lying down, and sometimes, though rarely, also relieved by firm pressure, which is, however, not a safe procedure. It is usually increased by palpation, slight pressure, muscular effort, mental exertion, and by the menstrual periods. (3) *Time of onset*.—The pain usually commences shortly after meals—ten to twenty minutes—but it is sometimes delayed much longer, even hours. It is occasionally only experienced at night as a gnawing or burning sensation, which is relieved by taking a small quantity of food. Palpation or slight pressure, even that of the wearing apparel, may bring it on. (4) *Duration*.—The pain is rarely continuous, particularly in the acute variety. It usually lasts until the food leaves the stomach by the pylorus or is vomited, when it generally disappears more or less quickly. It thus often lasts several hours. It may occur at intervals for days, weeks, or months, or be entirely absent for like periods. It gradually disappears with the cicatrization of the ulcer. When the pain is the most marked and prominent symptom it constitutes clinically the 'chronic gastralgic type.' There is often, in addition to the pain, a hyperæsthesia of the skin in the epigastrium, and also sometimes under the left breast (p. 1142).

Vomiting.—This symptom is rarer in the acute than in the chronic variety. It is frequently preceded by nausea, discomfort, or waterbrash in both varieties, and it usually relieves the pain in the latter much more completely than in the former. It may occur early or late in the course of the disease. It may follow the ingestion of food immediately, or be delayed for an hour or longer. In the former case it consists of partly digested food, in the latter it is more copious, liquid, and frothy like barm. The filtrate shows the presence of free hydrochloric acid (p. 1591). It is increased in amount in about two-thirds of the chronic cases, while in the remainder it is normal in amount except for a few cases, particularly those of marked anæmia, in which it is diminished. In the acute variety there is usually no hyperacidity. The filtrate may also show the presence of lactic acid (p. 1592). The amount of pepsin is generally unaltered, though occasionally increased, and in a few old cases diminished. When vomiting is the prominent feature it constitutes clinically the 'vomiting' or 'catarrhal type.'

Hæmatemesis.—This occurs in roughly one-third to one-half of all cases. It may take place early or late in the course of the disease, and with or without previous pain or vomiting, or any other symptom. (1) *Source*.—When small in amount the blood may come from capillaries and small vascular twigs in the edges and floor of the ulcer. When considerable in amount it always comes from erosion of one of the coronary arteries or other medium-sized or larger vessel. (2) *Amount*.—In the acute variety it is usually sudden and profuse—8 to 10 oz. or more in a few minutes—and tends to cease rapidly. In chronic cases the amount is liable to greater variation and the attacks have a greater tendency to recur.

Usually there are several recurrences at intervals of a few days, and then complete cessation for some time. The attacks are apt to come on during digestion or exercise, and may be preceded by a feeling of nausea or faintness. (3) *Appearances*.—It is generally bright red, clotted, and alkaline, especially in the acute variety. When allowed to remain longer in the stomach it is variously altered by the gastric juice, and appears dark red, or coloured like prune-juice or coffee-grounds. (4) *Melæna*.—The motions are black like tar. This occurs with or without hæmatemesis, particularly when the ulcer is near the pylorus, and is present only in those cases in which a considerable quantity of blood passes on into the intestine. It has always to be carefully inquired for, as much blood may be unsuspectingly lost in this way. The usual indications of considerable losses of blood, e.g. giddiness, faintness, weakness, clammy skin, low temperature, and soft, quick, small pulse, are observed. Two clinical types, the 'acute hæmorrhagic' and the 'chronic hæmorrhagic,' are often spoken of, in which the hæmatemesis is the prominent feature.

The other symptoms refer to the alimentary and urinary systems, and are chiefly covered by the dyspeptic symptoms already mentioned. The appetite is good, but if there be pain the patient is afraid to eat. Thirst and constipation are common. The urine may contain albumoses, acetone, or diacetic acid. The chlorides are often diminished, and the phosphates increased. The general nutrition may be good or much impaired (the cachectic type). There may be marked anæmia, amenorrhœa, headache, and sleeplessness.

Perforation and its results are considered elsewhere (p. 1619).

DIAGNOSIS.—In many cases a diagnosis is easy, in some it is difficult, in a few it is impossible. Even after a careful analysis of all the symptoms, the case may have to be kept under observation for some time, before a diagnosis of ulcer can be ventured upon. None of the characteristic symptoms of ulcer can be absolutely relied upon, for epigastric pain is met with in several other diseases, e.g. gastric cancer, chronic gastric catarrh, nervous dyspepsia, and gall-stones; vomiting in the same diseases and in the gastric crises of locomotor ataxia; while hæmatemesis occurs in a large number of both gastric and other diseases, e.g. gastric cancer, gastritis, varicose veins of stomach or œsophagus, passive congestion of the portal system from hepatic cirrhosis, pressure of a tumour, chronic cardiac or pulmonary disease, or enlarged spleen; toxic diseases such as yellow fever, acute yellow atrophy, purpura, &c.; constitutional states such as hæmophilia, &c.; injuries to the stomach-wall; or the blood may come from some position higher up than the stomach, e.g. nose, pharynx, lung, œsophagus, &c., and flow into the stomach and be afterwards vomited. Further, the diagnosis between ulcer and some forms of anæmia, e.g. chlorosis and pernicious anæmia, is often difficult, and it is to be remembered that they may co-exist.

PROGNOSIS.—The prognosis is very favourable, for probably over 90 per cent. of all cases recover, while in the acute variety alone the percentage is still higher. It may be taken as a fact that hospital cases make a more uniform recovery than those treated outside, and on the average the hospital cases are the more serious examples of the disease. A certain proportion of the cases which leave the

hospital cured or greatly improved suffer relapses afterwards, which may readily yield to appropriate treatment on re-admission to hospital. It is not unreasonable to think that, if the proper treatment were always rigorously carried out, very few if any would relapse. It is very rare indeed for a hospital case not to improve steadily under proper treatment, provided the patient survives the first few days after admission, and it does not seem too much to say that all cases, with very few exceptions, may recover if proper treatment be applied early enough. If all symptoms have disappeared for months and subsequently break out again, the case is practically a new one and not a true relapse, even though the ulcer occurs at its previous site. If the ulcer be large, the cicatrization will be correspondingly extensive, and in the pyloric region especially this may cause trouble from pyloric obstruction long afterwards. This is due, however, to the anatomical peculiarities of the parts, and so far as the ulcer is concerned ought not to be considered as an imperfect cure. Recent advances in abdominal surgery have made it possible to cope with this condition with most gratifying success. There remain then only those cases in which the ulcer passes on rapidly to perforation or gives rise to severe and uncontrollable hæmorrhage, and those cases which die from exhaustion or some rare complication. It is extremely difficult to arrive at an accurate estimate of the mortality. Of course, if over 90 per cent. recover, then less than 10 per cent. die, and it appears for many reasons probable that this is not far from the truth. Most of the statistics on this point are too old to be reliable, for it is obvious that the advances in recognition and treatment, which have been made within recent years and will we hope continue to be made in the future, must tend to decrease the mortality from all these sources. It is the general experience that death from perforation is much more common than from hæmorrhage, so that if 10 per cent. be the true total mortality, at least 6 per cent. must be put to the credit of perforation, and not more than 3 per cent. to hæmorrhage. Even 3 per cent. is probably too large, for it is the universal experience that hæmorrhage is very rarely suddenly fatal, and under the present improved methods of treatment it is likely to be almost always successfully controlled in the further progress of the case.

TREATMENT.—Medical.—Medical treatment is all that is necessary in the great majority of cases. It aims at giving the stomach as much rest as possible, and may be considered under the heads of rest, diet, and drugs. It is impossible to lay down general rules to suit all cases, and modifications must be made to suit individual peculiarities. Patience is the keynote of success. The slightest return of untoward symptoms requires immediate attention, and a rigorous continuance of appropriate treatment. The treatment should last on the average at least three months—in the very chronic cases much longer, a year or more—and should in all cases be continued for many weeks after all symptoms have disappeared.

Rest is best obtained in bed, and at least the first fortnight should be spent entirely there. It is advisable for most cases to keep their beds still longer, three weeks or more, and sometimes during the greater part of the treatment. The recurrence of active symptoms should always be followed by a period of rest in bed. This procedure ought to be

rigorously enforced whenever it is possible, and relaxed only in exceptional cases.

Diet.—Milk is the ideal diet, and it should be carefully tried in all cases. It is a favourite plan with many to give 5 oz. every hour for the first three days, then 10 oz. every two hours for the next week or so, and then gradually increase it. To find out what suits each case is far better than to follow any rule. Proceed cautiously at first, and aim at reaching doses of 10 to 15 oz. four times daily at four-hourly intervals as soon as possible, remembering carefully that any distension or irritation of the stomach has to be avoided. Afterwards increase the quantity to between one and two quarts in the twenty-four hours. Each dose should be moderately warmed and sipped slowly, especially at first. Occasionally it suits better when iced. These milk-meals should be continued for a month, and as patients get tired of them they may be varied, after the first day or two, by alternating them once or twice a day with weak beef-tea, Leube's, Liebig's or Valentine's meat-juice, Carnigen, a good beef-powder, or somatose, either by themselves or added to the milk. It is advisable also to add a little lime-water or a pinch of bicarbonate of sodium to the milk to prevent the formation of large masses of casein and favour digestion. Barley-water, rice-water, oatmeal or oat-flour water added to the milk varies the flavour and increases its digestibility. An egg beaten up in milk may be tried at the end of the first week, and given once or twice daily thereafter, or a little Benger's food or ground rice or arrowroot may be used as an alternative to thicken the milk. Skimmed milk or butter-milk is often well borne when ordinary milk is not. This is also true of sterilised milk. When none of these is tolerated, peptonised milk should be given, and when all of them fail nutrient enemata must be resorted to. Avoid tea, coffee, and alcohol in all its forms.

Nutrient Enemata.—Before giving a nutrient enema the bowel should be washed out with a quart of warm water, to which a teaspoonful of common salt has been added. This should be done twice in the twenty-four hours, and the nutrient enema given one hour after the injected water has been voided. The temperature of the nutrient enemata should be 100° F., and they should be administered every four to six hours. They should be about 8 to 10 oz. in bulk, but if such quantities are not well borne, 2 to 3 oz. may be tried. They should consist chiefly of peptonised milk or beef-tea, to which the yolk of one or two eggs and 5 grains of bicarbonate of sodium or two teaspoonfuls of wine, whisky, or rum, if there is much weakness, or a few drops of laudanum, if much irritation, may be added. Many prefer to give an enema (2 to 3 oz.) of beef-tea and raw egg with a little brandy. It is rarely necessary to continue exclusive rectal feeding longer than two to three days, except in persistent vomiting and after hæmorrhage, when it may be necessary to do so for a week or even a fortnight. It is to be remembered, however, that it is a very imperfect method of nutrition, and a little peptonised milk (1 or 2 oz. frequently) ought to be given by the mouth as soon as possible, and then cautiously increased while the nutrient enemata are gradually dropped.

Solid Food.—After about a month, if pain and vomiting have long disappeared, begin to give solid food cautiously—a little bread and milk, arrowroot,

tapioca or sago, or ground rice may be given with the milk; and after three or four days more a scrambled or softly steamed egg may be tried. After a week, a little grated boiled fish or chicken, or raw-meat pulp or scraped raw beef (the latter are often well borne much earlier in the treatment, even when a milk-diet disagrees). After another week, raw eggs or soft-boiled or poached eggs, sweetbreads, tripe, brains, stale bread, thin bread and butter, may be given. Then lamb-chops, mutton-chops, underdone steaks with thin thoroughly toasted or stale bread may be tried. Then fish, chicken, and white meat with milk and rice-puddings may be allowed. The patient may now return to his work and gradually resume ordinary diet, but all indigestible foods, such as curries, shell-fish, vegetables (except rice), pastry, &c., must be avoided. Any form of food, however simple, which the patient finds to be followed by discomfort, must be rigorously excluded from his dietary. He must avoid all muscular fatigue and mental worry, and scrupulously continue his treatment for months after he is apparently cured.

Drugs.—No drugs exercise any influence upon the ulcer other than to protect it somewhat from the irritative action of the gastric juice. Drugs are chiefly given to relieve pain, vomiting, hæmatemesis, and constipation, and to correct the anæmia if present. (1) *Pain*.—This usually subsides after a few days' rest in bed and milk-diet, but certain drugs may be of much use, e.g. a dilute warm solution of bicarbonate of sodium frequently sipped; a little prepared chalk, spirit of peppermint, and carbonate of magnesium added to it increase its flavour and efficacy; or 30 to 60 grs. of the sub-nitrate or carbonate of bismuth, with 15 grs. of bicarbonate of sodium thrice daily, half an hour before each meal, to which may be added a little dilute hydrocyanic acid (3–5 m), or to m of tincture of nux vomica, or $\frac{1}{10}$ gr. of morphine or $\frac{1}{4}$ gr. of hydrochloride of cocaine, or a combination of them. Small doses of morphine frequently repeated and given by the mouth often do much good. Very severe cases require morphine-injections. Linseed-meal poultices and hot fomentations or even blisters over the epigastrium are often of great value in relieving pain, and should be used as a routine procedure for the first three weeks whenever pain is present. When the patient cannot lie up, nitrate of silver is of value in relieving pain. It should be given in $\frac{1}{4}$ gr. doses in pill or preferably in solution thrice daily, and gradually increased to $\frac{1}{2}$ to 1 gr. Discontinue it for a week during every month to avoid argyria. (2) *Vomiting*.—Rest in bed, rectal feeding, and bismuth and cocaine, &c., as above. (3) *Hæmatemesis*.—The patient should lie on his back with his head low, and should not move, even slightly. He should be covered with a tight blanket and have ice applied to the epigastrium. No drug is to be given by the mouth, but hypodermic injections of morphine and ergotina may be useful. Some have found oil of turpentine (30 m to 60 m) given in capsules or as an emulsion, with white of egg, very valuable in severe bleeding. When collapsed a pint or so of saline solution (6 parts NaCl to 1,000 parts water) should be slowly injected subcutaneously. Rectal feeding should be exclusively employed for a time. The patient should be confined to bed for a month after the bleeding has ceased. (4) *Constipation*.—This is often very troublesome, and requires to be relieved by purga-

tives for the first month at least. Enemata of soap and water, of castor oil or glycerine, are to be used. Laxatives may be given afterwards by the mouth, and castor oil is by far the best. Carlsbad salts (NaCl 1 oz., NaHCO₃ 2 oz., Na₂SO₄ 4 oz.) in 1- to 2-teaspoonful doses in a large tumblerful of warm water every morning before breakfast, or some natural aperient water, such as Friedrichshall, &c., may be substituted, or aloin or a little cascara sagrada given at night. (5) *Anæmia*.—Iron, in its scale-preparations, or reduced iron, or the saccharated carbonate, or some of its more modern preparations must be given, but not too soon, not until healing is complete, as it is liable to retard recovery.

Surgical.—This is required in perforation, hæmatemesis, obstruction, and complications such as subphrenic abscess. In perforation, laparotomy is performed and the perforation closed by suture. If this be not done a fatal result is almost inevitable (95 per cent.). The outlook is better the shorter the interval which occurs between the perforation and the laparotomy. In profuse and recurring hæmorrhage the ulcer may be cauterised and a gastro-enterostomy performed. Several successful cases have been recorded. For obstruction and gastric dilatation, divulsion of the pylorus, pyloroplasty, or gastro-enterostomy may be resorted to.

R. F. C. LEITH.

STOMACHICS (στόμαχος, the stomach).—

SYNON.: Fr. *Stomachiques*; Ger. *Magenmittel*.

DEFINITION.—Substances which increase the functional activity of the stomach.

ENUMERATION.—The most important stomachics are alcohol, acids, alkalis, aromatics, arsenic, bitters, pepsin, and strychnine or nux vomica.

ACTION.—Some stomachics, such as alcohol and dilute alkalis, increase the secretion of gastric juice; possibly also bitters, and small doses of arsenic. Dilute acids, given after meals, and pepsin supply the essentials of gastric juice when secretion is deficient. It is not improbable that the peristaltic movements of the stomach are increased by strychnine and nux vomica. We want experiments on the action of drugs which increase absorption. It is also probable that some of the good results of bitters are due to their preventing abnormal processes of fermentation in the stomach.

T. LAUDER BRUNTON.

STOMATITIS (στόμα, the mouth).—Inflammation of the mouth. See MOUTH, Diseases of.

STONE.—A popular name for calculus. See CALCULI; and CONCRETIONS.

STONE - GRINDER'S PHTHISIS. — See PNEUMOCONIOSES.

STOOLS.—SYNON.: Fæces; Motions; Fr. *Excréments*; *les Celles*; Ger. *Stuhlgänge*; *Stühle*.

INTRODUCTION.—An examination of the fæces frequently affords valuable evidence of the condition of the organs engaged in the process of digestion, and furnishes important data on which to found a diagnosis and suggest a rational treatment. Not only may structural changes in the alimentary tract be discovered, but also the completeness of action of the various digestive juices be recognised. As with the examination of the renal secretion, a previous knowledge of the healthy characters of the fæces is an essential: this being granted, the

investigation may be pursued on the same lines in both cases, as regards the general, microscopical, and chemical characters. Since, however, the characters of the *fecës* are much more directly dependent on the ingesta, their examination cannot be so valuable an index of tissue-change as is that of the urine.

I. Physical Examination.—**1. QUANTITY.**—This is extremely variable. Taking the normal average for an adult to be about 5 ounces daily, it may vary from $2\frac{1}{2}$ to $10\frac{1}{2}$ ounces. The quantity would seem to bear no relation to the size or weight of the individual, but is rather influenced by the quantity and kind of food taken, and by the activity of the secretions of the alimentary canal. With an average diet, it is estimated that the *fecës* form one-seventh to one-eighth of the weight of the ingesta. As a rule, the amount is increased by a vegetable diet. In children it would seem the total daily amount is relatively slightly greater, while in old age there is an absolute diminution. When in disease the quantity is increased, it is chiefly that of the fluid portion, while a diminution affects both the solid and fluid parts. The writer has noticed a class of cases of intestinal derangement characterised, among other symptoms, by the daily passage—especially in the earlier hours of the day—of several very large pultaceous stools, amounting to a total quantity several times in excess of the normal, and this without a large ingestion of food; the lower part of the ileum, the cæcum, and the ascending colon appear to be in a condition of chronic catarrh in these cases. Tea is said to diminish the quantity of the *fecës* (Chambers). See CONSTIPATION; and DIARRHOEA.

2. CONSISTENCE AND APPEARANCE.—Departures from the normal cylindrical shape are frequent, and depend very much on the existence of constipation or diarrhoea. In infants the evacuations should be unformed and of a pappy consistency. The contents of the bowels pass from a semi-fluid condition in the ileum to the firmer state in the colon, mainly from an absorption of fluid constituents; should there be any delay in the passage the motions are liable to become hard and nodular (*scybalæ*), and this condition may occasionally be extreme, the *fecës* having all the appearance of sheep's dung, and being passed with considerable pain. Certain drugs, such as vegetable astringents, and many preparations of iron, bismuth, or lead, tend to make the *fecës* hard and firm. On the other hand, an increased peristalsis is associated with motions of all degrees of fluidity. This is very marked in the various forms of irritation to which the intestinal mucous membrane is liable, from the simple effects of a saline purge to the extreme conditions of ulceration, as in typhoid fever or dysentery. The rate of passage of the intestinal contents, together with the amount of intestinal secretion, are the conditions determining the various degrees of consistency of the stools designated by the terms '*scybalous*,' '*formed*,' '*semi-formed*,' '*pultaceous*,' '*loose*,' and '*fluid*.'

- The existence of hæmorrhoids, rectal growths, or an enlarged prostate, may be recognised by groovings and marks on the excrement. It is very common in cases of stricture of the colon to find the motions flattened and ribbon-like, or as cylinders much contracted in diameter. Such appearances, however, are by no means constant, for it has been clearly shown that a considerable stricture, even as

low down as the junction of the sigmoid flexure and rectum, may co-exist with motions of normal appearance, the *fecæ*l matter becoming remoulded below the seat of constriction. But the invariable occurrence of such contracted motions is strongly in favour of stricture. Occasionally, as in *psilosis intestini*, the motions are passed in a fermenting condition, presenting a frothy brown or yeast-like appearance, and containing *sarcinæ*, similar to certain vomits. See *PSILOSIS*.

3. COLOUR.—This is dependent on stercobilin, which is regarded as identical with hydrobilirubin (*urobilin*), a derivative of bilirubin ($C_{16}H_{18}N_2O_3$), the chief bile-pigment, and is subject to considerable variation even within the limits of health. The usual brown colour becomes much darker if the *fecës* be long retained, or with an exclusive meat-diet; pale yellow with milk-food, as seen in infants; while it tends towards a greenish tint when vegetables form the bulk of the food.

Unaltered bile-pigment is never found in healthy stools. The conversion of bilirubin into stercobilin, regarded by MacMunn as a process of intermediate oxidation, normally commences in the first part of the colon; but if from any other cause—such as the use of aperients or ulceration or other affection of the bowels—the intestinal contents be hurried along and a diarrhoea established, then bile-pigments, as such, may be found in the motions, causing the bright yellow colour of the well-known '*bilious stool*,' and often leading to much smarting of the anus when voided. In dysentery, the presence of bile in the motions is frequent, especially staining the mucus which is so abundant in the motions of that disease; globules of mucus from the small intestine, similarly coloured, may be seen in the stools of typhoid fever. Other constituents of the *fecës*, such as the shed intestinal epithelium, may also be stained with unaltered bile under the same circumstances.

It is noteworthy that the meconium contains a considerable amount of the normal bile-pigments, bilirubin and biliverdin, but no stercobilin.

The principal alterations in the colour of the stools are—

(a) *Pale and colourless stools.*—All degrees of deficiency of colour may be met with, producing the so-called '*clayey*' or '*putty-like*' stools. This condition is seen in its most marked degree when the bile is absolutely excluded from the intestine, as by an impacted gall-stone; the motions being unformed, lumpy, '*porridge-like*,' or *scybalous*, and almost or quite colourless, frequently, however, accompanied by a small quantity of distinctly bile-coloured fluid, which is secreted by the mucous membrane of the bowel from the bile-laden blood. The causes of pale stools when there is no such obstruction to the bile-flow are varied and not always apparent. In children the explanation is sometimes to be found in the presence in the motions of a large proportion of undigested fat (derived from the milk-food); in various anæmic states the fundamental deficiency of blood-pigment appears to be the cause, and this may account for the frequent occurrence of this condition in rickets. But there remain many cases in which such explanations do not apply, and for which the cause can only be definitely ascertained when we know more certainly than we do at present what becomes of the bile as regards its reabsorption and decomposition in the intestine. The occurrence of colourless

or clay-coloured stools in cases where there is no perversion of the biliary secretion or of its discharge into the duodenum, but where disease of the pancreas has been proved to exist, has suggested 'that the formation of the colouring-matter of the fæces depends on the mutual reaction of the bile and pancreatic fluid, under the influences met with in the intestinal tract;' though what the nature of this interaction is remains unexplained (*see PANCREAS, Diseases of*). A remarkable case which was under the writer's care would tend to show that the production of clay-coloured stools, in cases where the bile reaches the intestine and there is no jaundice, is due to some interference with those changes in the bile which normally take place in the first part of the colon. The patient died from the remote effects of an accident in which he had been severely squeezed between the buffers of a train; for several months previous to death he suffered from constant diarrhoea, the motions being invariably copious and quite colourless. *Post mortem*, the cæcum was found to be represented by a quantity of inflammatory fibrous tissue involving the adjacent parietes, excavated in which were a series of irregular spaces and channels outside the peritoneal cavity, by which continuity was maintained between the ileum and ascending colon; down to the end of the uninjured ileum the bowel-contents were of normal condition and colour (the patient taking almost an ordinary diet) and contained bile, but in the colon they were colourless, as voided, while in the segment of mesentery corresponding to the cæcum and commencement of the colon, the lymphatic vessels were outlined by black pigment, presumably due to absorption of the biliary colouring-matter from the damaged portion of the intestinal canal. Further support for this view is to be found in the character of the motions met with in psilosis of the intestine, they being large, formed, white, and acid in reaction. *See PSILOSIS*.

(β) *Black stools*.—This condition is well marked when blood has escaped into the stomach and passes through the bowels. The hæmoglobin is decomposed by the gastric juice, and its constituent iron is converted into the black sulphide by the sulphuretted hydrogen present in the intestines. Such stools may look like tar; or if they have been long retained they become hard and almost coke-like in appearance (*see MELÆNA*). Iron, bismuth, or charcoal taken internally will similarly colour the fæces; but if they are taken in only small quantities the motions are slaty rather than black.

(γ) *Green stools*.—These are of frequent occurrence in children, and seem to be due to the presence of biliverdin ($C_{16}H_{18}N_2O_6$), an oxidation product of the biliary colouring-matter, bilirubin, which under normal circumstances is reduced; and it not infrequently results from a dose of calomel, which prevents the reduction taking place.

(δ) Certain articles of diet and drugs are liable to colour the fæces more or less distinctly. Thus spinach, coffee, claret, and logwood impart to the excrement their characteristic tints; rhubarb, senna, and santalin cause a bright-yellow colour; and much cocoa may make the stools grey.

4. *ODOUR*.—The characteristic odour of the fæces is chiefly due to certain substances, especially skatol, and to a less degree indol, developed during the pancreatic digestion of proteids, and partly to special secretions from the glands of the colon. Impaired intestinal digestion, particularly, it would

appear, if there be any abnormal bacillary action, is apt to cause fœtid stools, but this effect is very variable. Absence of bile from the alimentary canal, by interfering with the pancreatic digestion, and also by the want of its own special antiseptic power, is often, but not always, accompanied by very ill-smelling motions. In certain diseases of the colon, as dysentery, the evacuations are of a most foul and acrid character, owing to putrid changes in the sloughs thrown off from the ulcerated bowel. In children, the alvine discharges are frequently offensive from errors in diet, and emit a peculiar sour odour.

5. *REACTION*.—The reaction of the fæces in health may be slightly acid, or slightly alkaline. This variability is due to the nature of the food, and still more to the extent and character of the changes in the colon. The stools in some diseases, as typhoid fever, are distinctly alkaline, and in others, as infantile diarrhoea, markedly acid; the colourless stools in obstruction of the bile-duct are also very acid from a large excess of free fatty acids.

6. *FOREIGN BODIES*.—By inspection of the evacuations we may discover—

(a) Substances *accidentally* swallowed, such as coins, pins, &c.

(b) *Indigestible food-residua*, such as fish-bones, the stones, skins, and peel of fruits, fig-seeds, the ligneous grit in pears, &c. Small seeds and particles of vegetable tissue, if long retained in the canal, may become encrusted with lime-salts, and, if abundant, form what has been called intestinal sand (*sable intestinal*). *See CONCRETIONS*.

(c) *Undigested food*, that is, portions of food that have escaped digestion. In certain severe disturbances of the digestive functions some of the food may be passed scarcely, if at all, altered, and may be easily recognised. This condition, known as 'lenty,' is especially prone to occur in the intestinal catarrh of young children during dentition. Curdy lumps derived from milk, and consisting of undigested masses of coagulated casein entangling fat, are often met with in infantile diarrhoea. 'On a moderate diet unaltered proteid is never found' (Halliburton).

(d) *Fat*.—This special element of the food may occur in the motions in such quantity and in such forms as to require separate mention. Anything more than a trace in the fæces is to be regarded as abnormal, and as the expression of a failure in its digestion or absorption. This may be due to an excessive ingestion, as may occur in a milk-diet, or where undue quantities of olive or cod-liver oil are given for medicinal purposes; or to a deficiency of the digestive fluids, especially the pancreatic juice. The presence of fat in the stools has long been known as a diagnostic sign of pancreatic disease (*see PANCREAS, Diseases of*), and is of not infrequent occurrence in diabetes. In those morbid states characterised by extensive destruction of the epithelium of the small intestine, fat may appear in the motions from not being absorbed, although the quantity taken may be but moderate, and the conditions for its digestion unimpaired.

The form in which fatty matter appears in the stools is very variable, and much depends upon the kind of fat taken. Masses of almost pure fat may be found; or lumps of greasy-looking substances, consisting mainly of crystals of the fatty acids, or compounds of these with earthy bases, forming soaps, may be met with. Occasionally these

substances may be voided in cylindrical form, which has been compared to macaroni.

(e) *Mucus*.—The amount of this substance in normal fæces is quite inappreciable, but in catarrhal and other inflammatory states of the lower end of the ileum and colon it is formed in considerable quantity. Its disposition in respect to the fecal matter is, to a great extent, diagnostic of its site of production. Thus in catarrh of the ileum, with the very bulky stools above-mentioned, it is intimately diffused through the fæces in microscopic particles; in the motions of typhoid fever, bile-stained globules of mucus may be seen with the naked eye. It may be taken as a general rule that the more distinctly separate the mucus is from the excrement, the lower down in the bowel does it come from. When it occurs in masses adherent to or apart from the fecal matter, it comes from the sigmoid flexure or rectum; while from the upper part of the colon the mucus appears as fair-sized globules mixed with the stool. In dysenteric inflammation and ulceration of the colon the mucus frequently forms discrete particles, which have been compared to grains of boiled sago or to frog-spawn, and may be mixed with pus and blood.

(f) *Blood*.—Blood voided *per anum* presents very different appearances according to its source. When coming from the stomach or upper part of the intestine it is much altered, being black and tarry (see MELÆNA). Even when recognisable as blood, there is a considerable range of variety in colour, as well as degree of admixture with the fecal matter. On the one hand, it may be passed in a perfectly pure state, quite free from foreign matter, as would be the case when the bleeding took place from an eroded vessel low down in the bowel, or even as high up as the ileum, provided the quantity were considerable, and it were discharged immediately after its escape. Practically, the purer and brighter the blood, the lower down is its source. Or it may occur as a brownish-red material thoroughly mixed with the fæces, and then comes from the commencement of the colon or small intestine; or streaks or smears of blood scarcely altered in appearance, with or without mucus, and attached to the surface of the motion, suggest a lesion in the lower part of the colon and rectum. The blood, from internal piles or extreme congestion of the venules of the rectum, has a characteristic purplish tint, and is frequently passed quite free from feculent matter. See also p. 789.

(g) *Pus*.—This is not a very frequent constituent of the stools, the amount in ordinary catarrhal states of the bowel being too small, as a rule, to be recognised without the microscope. In dysentery, however, the quantity is greater, and pus then becomes a prominent feature in the motions. When large quantities are passed, the source is usually an abscess which has burst into the canal.

(h) *Entozoa*, such as segments of tænia, the various round-worms, or the contents of hydatid cysts that may have burst into the canal, may be present in the fæces.

(i) *Miscellaneous*.—Under this head may be included gall-stones, intestinal concretions, mucous or membranous casts of the intestines (pp. 254, 795), portions of bowel sloughed off from intussusception (p. 783), or of new-growths &c.

II. Microscopical Examination.—See pp. 800, 121.

III. Chemical Examination.—It is seldom if ever that such examination furnishes evidence of primary importance, as does analysis of the urine.

The food is of course the main source of the constituents of the fæces, a few only of these components being derived from the bile and other digestive secretions. The shed epithelium and detritus from the intestinal mucous membrane form, however, no inconsiderable share of the bulk of the fæces.

For the chemical processes—often elaborate—required for their detection, reference must be made to standard works on physiological and pathological chemistry.

1. *Phenol* (C_6H_5OH), *indol* (C_8H_7N), and *skatol* (C_9H_9N) are normally formed in the intestines during the pancreatic digestion of proteids. A considerable proportion of these substances is absorbed, and, undergoing various chemical changes, is eliminated in the urine; but a considerable amount of each also leaves the body in the fæces, where they may be separated and detected by appropriate reagents. To skatol the odour of the fæces is mainly due, and it is said by Brieger to be absent from the evacuations of typhoid fever.

Leucine and *tyrosine*, which are also derived from the proteid elements of food by tryptic digestion, are not found in the fæces in health, though of frequent occurrence in the stools of various conditions of diarrhœa, especially cholera. See LEUCINE; and TYROSINE.

2. Two bodies—*excretin* and *stercorin*—have been described as characteristic of the fæces, but their existence is doubtful. They are non-nitrogenous, crystallisable, non-saponifiable bodies, differing in their crystalline form, and their solubility in alcohol and ether. They appear to be closely related to cholesterol, with which in many points they agree. It is said that when the bile which normally contains cholesterol is prevented reaching the intestines, neither of these bodies occurs in the fæces; and that they are also absent, being replaced by cholesterol, in the meconium, and in starving and hibernating animals, when there is no obstruction to the bile-flow. Flint considers about ten grains *per diem* to be the average amount of stercorin, and the excretin of Marcet to be about a fifth of that. *Cholesterol* itself, except in very small quantities, cannot be regarded as a normal constituent of the fæces; but the precise significance of its occurrence is uncertain. Its chief source is the bile, and only a small quantity comes from the food. See CHOLESTERIN.

3. *FATTY ACIDS, FATS, AND SOAPS*.—Various members of the fatty-acid series, such as acetic, butyric, caproic, valerianic acids, and the higher terms, oleic, palmitic, and stearic, only occur free in minute proportions, in ordinary circumstances, but the latter are readily increased to considerable amounts when the alkaline secretions of the liver and the pancreas are prevented from entering the intestines. The presence of neutral fats in the excrement may be taken to indicate that there has been excessive ingestion or a diminished digestion, since under ordinary circumstances they would be absorbed. When occurring in considerable amount in the stools they produce the so-called 'fat-diarrhœa,' described in various dyspeptic states. The soluble sodium- and potassium-soaps ordinarily formed by the ingested fats with the bile and pancreatic juices should be in great part taken up by the lacteals; but fats, meeting with any calcium- or

magnesium-salts that may be present in the alimentary canal, form with them insoluble hard soaps, which are passed in the fæces.

4. **SALTS.**—These are but in small amount, and are chiefly the earthy and triple phosphates, with small quantities of iron and silica; there is in health a marked absence of chlorides. When the stools are very alkaline the triple phosphates may be very abundant, as in typhoid fever; and in cholera discharges the whole amount of salts is largely increased, there being nearly an ounce in every hundred fluid ounces of evacuation, a large proportion of which consists of chlorides. A similar excess of these salts occurs in the stools of other diarrhoeal states, with a corresponding decrease in the urine.

5. **PIGMENT.**—The characters of the fæcal pigments have been already referred to.

6. **MUCIN, ALBUMEN, &c.**—A very appreciable amount of *muçin* may be extracted from the fæces by well mixing with lime-water, and adding acetic acid to the filtrate.

Albumen, as such, can scarcely be looked upon as a normal ingredient of the evacuations; but the fluid part of cholera-stools, which resembles dilute blood-serum in composition, contains a very appreciable quantity. In typhoid and other diarrhoeal stools albumen can be detected.

Peptones are not found in healthy fæces, but occur in considerable quantities in various diseases, of which the most frequent are typhoid fever, dysentery, tubercular ulceration of the intestines, and cirrhosis and carcinoma of the liver.

Ferments similar in action to ptyalin and pepsin are described as existing in the fæces, but how they may be modified in disease is quite unknown.

Certain of the groups of putrefactive bodies termed *ptomaines*, such as cadaverine and putrescine, have been recognised in the fæces. See p. 1333.

In uræmia and also in intestinal catarrh *urea* has been detected in the stools.

In certain diseases, especially cholera, dysentery, typhoid fever, intestinal catarrh, both acute and chronic, and to a less degree hepatic and pancreatic affections, the stools are more or less characteristic, though the diagnosis rarely depends on their appearances alone. They are described under their respective headings.

W. H. ALLCHIN.

STRABISMUS.—SYNON.: Squint; Fr. *Strabisme*; Ger. *Strabismus*; *Schielen*.

DEFINITION.—A condition in which the two eyes are not directed to the same point in space.

DESCRIPTION.—Squint is commonly either (1) *convergent*, or (2) *divergent*; but (3) it may be complex, where there is a deviation either *upwards* or *downwards*. When one eye appears to be normally directed, and the other to deviate, it is convenient to distinguish the former as the *working*, and the latter as the *squinting* eye.

The extent or degree of strabismus, or, as it is more usually called, the *magnitude* of a squint, is expressed in terms of millimetres. In convergent or divergent squint it is customary to measure the distance between an imaginary vertical line bisecting the palpebral fissure, and another imaginary vertical line bisecting the pupil of the deviating eye. In an upward or downward squint, the distance between the horizontal diameter of the pupil, and an imaginary horizontal line bisecting the palpebral fissure, would give the measurement required.

1. **Convergent Squint.**—This is seen under two principal forms: (a) that which depends upon *paralysis* or *paresis* of one of the external recti muscles, permitting the antagonist, the internal rectus, to exert a preponderating influence upon the position of the eye; and that which depends upon *excessive development* of both interni, in consequence of an error of refraction, whether (b) *hypermetropia*, or (c) *myopia*.

(a) *Paralytic Convergent Strabismus.*—This form of strabismus is met with in all degrees, from the slightest weakening of the affected external rectus to complete paralysis.

ÆTIOLOGY.—Paralytic convergent strabismus is primarily a nerve-affection, in which, however, the muscle concerned will after a time be liable to undergo degenerative changes. The strabismus usually commences somewhat suddenly, in persons of adult age, and often rapidly increases in degree; the paralysis, which at first was only partial, becoming complete. In the great majority of cases it is associated with syphilis; but it is also met with as a result of impaired nutrition or degenerative change in the nervous centres, consequent upon anxiety or over-work. In some of the syphilitic cases, it appears to be due to central mischief, such as gumma or arterial occlusion; in others to pressure upon the trunk of the sixth nerve by periosteal thickening or other morbid growth.

DIAGNOSIS.—In pronounced cases, the diagnosis is easy; and depends upon the fact that, even when the working eye is closed or covered, the squinting eye cannot be directed outwards by voluntary effort. If the paralysis, although considerable in degree, be not complete, the eye cannot be directed outwards as far as usual; if the paralysis be complete, the pupil cannot be carried external to the middle line of the palpebral fissure. When the affected muscle is only slightly weakened, the nature of the condition may not be at once apparent from the limitation of movement; and the degree of deviation may be so slight that it is not easy immediately to pronounce which eye is affected. This doubt may be removed, and the existence of paresis made manifest, by the following tests. The surgeon should stand in front of the seated patient, and should hold up before him, in the middle line and at a convenient reading distance, some small object, telling him to look at it steadily. By his own hand, or by a piece of ground-glass, the surgeon then cuts off the view of the object first from one of the patient's eyes and then from the other, watching their movements as he does so. When the object is concealed from the squinting eye, the other one, being already rightly directed, will remain stationary to continue the act of seeing, and the squinting eye will also remain stationary; but when the object is concealed from the working eye, the other, or squinting eye, being wrongly directed, and not receiving the image of the object upon its yellow spot, will make a slight outward movement in order to take up correct fixation. At the same moment, the working eye, behind the obstruction, will execute an inward movement of somewhat greater amplitude than the outward movement of its fellow. Let it be supposed that there is slight weakening of the right externus, producing slight inversion of the right eye. When the object is screened from the right eye, the left still sees it clearly and sharply, and both eyes remain at rest. When the object is screened from the left eye, the right receives the image upon a

point of its retina internal to its yellow spot, and sees it only indistinctly. The right eye, therefore, makes an excursion outwards, sufficient in amount to bring the image of the object upon its yellow spot, and to enable it to see better; but the motor impulse by which the necessary movement of the external rectus is called forth is conveyed at the same time to the internal rectus of the left eye, as a result of the habitual association of the two eyes and of their muscles in the act of looking towards the right; and the sound muscle, under a given motor impulse, contracts more vigorously than the weakened one. The result is that the excursion inwards of the working eye is larger than the excursion outwards of the squinting one; and in this way the fact of paresis of the right externus is rendered manifest.

This form of strabismus is at first attended by distressing double vision, which often produces giddiness, but which diminishes in time, as the patient learns to neglect or mentally to suppress the image of the squinting eye. The smaller the deviation, the more distressing will be the double vision; because, the nearer to the yellow spot the image of the squinting eye is, the more definite will it be, and the less readily will it be distinguished from that of the other. In cases of very slight deviation, the equality of the two images renders it difficult to tell the true from the false, and leads the patient into frequent error with regard to the position of the object looked at.

TREATMENT.—The treatment of paralytic strabismus is primarily that of the syphilis or of the nerve-exhaustion upon which the paralysis depends; but it is also necessary to endeavour to minimise the inconveniences of the double vision while it continues, and to provide against permanent degeneration of the paralysed muscle from disuse. The former indication may be fulfilled by covering the squinting eye with a patch, or with an opaque glass in a spectacle-frame; and, as the double vision is only troublesome when the eyes are directed to the side of the affected muscle, it is often sufficient to render opaque, by grinding or otherwise, the outer half of the glass which covers the affected eye. The nutrition of the muscle may be preserved, when the paralysis is incomplete, by systematic voluntary endeavours to call it into action; these endeavours being made three or four times a day for a few minutes at a time. For this purpose, the working eye should be closed or covered, and the squinting eye should be directed as much as possible towards the outer side. When the paralysis is complete, so that the eye cannot be carried beyond the middle line of the palpebral fissure, it may be necessary to exercise the affected muscle by localised electric currents, after the manner of Duchenne. In cases where there is no response to induced currents, those of a cell-battery will sometimes be found effectual. The exercise by electricity should be repeated at short intervals, such as every two or three days, until the nerve-function is beginning to be restored, so that the muscle can again be exercised by the will.

In cases of paralytic strabismus of old standing, it is sometimes necessary to have recourse to tenotomy of the contracted internus, before the eye can be restored to its correct position. It may be laid down as a general principle that nearly every case can be cured, by combined tenotomy and volitional or electrical exercises, as long as the paralysed muscle

will respond, in however small a degree, either to the will or to one form or other of electric current; but that where the eye does not move outwards in obedience to the will, and where neither induced nor battery-currents produce contraction of the paralysed muscle, no good is to be expected from either operative or medicinal treatment.

(b) *Convergent Strabismus due to Excessive Development of both Interni.*

ÆTIOLOGY.—As a result of errors of refraction, about 90 per cent. of this class of cases are due to flat-eye or *hypermetropia*. The flat formation of the eye requires, for acute vision of near objects, a strenuous accommodation-effort; and this, by the intimate association which exists between the nerve-centres governing the accommodation-muscles and those governing the interni, produces a corresponding effort of convergence. As soon as a child who is born with flat eyes begins to take careful notice of near things, his accommodation-muscles and his internal recti are both called into frequent and energetic exercise; and the consequence is that the interni become excessively developed in relation to their antagonists, the externi, so that the normal or resting position of the eyes, instead of being one of parallelism, becomes one of convergence. The result of this is that the child would receive double images, of equal intensity, of all objects situated either nearer to him, or farther from him, than the point at which the convergent optic axes would meet if prolonged. Let us suppose that this point is one foot distant from the eyes; and that the child wishes to look at an object which is eighteen inches distant. He cannot do this with both eyes, because the externi are unable to overcome their more powerful antagonists. If, however, he combines the right externus with the left internus, as in the act of looking to the right with both eyes, he becomes able to fix the object correctly with his right eye; and if he combines the left externus with the right internus, as in the action of looking to the left with both eyes, he becomes able to fix the object correctly with his left eye. But as, in either case, both eyes start from a position not of parallelism, but of convergence, the effort which carries the right eye from its convergent state to the middle of its palpebral fissure will carry the left from its convergent state to one of much greater convergence, and *vice versa* with the left eye; so that, while one eye is directed to the object of vision, the other is rolled far inwards. In this way, the image is received upon the yellow spot of the working eye, and upon so peripheral a portion of the retina of the squinting eye that it is easily neglected by the consciousness, and ceases to be a source of confusion or embarrassment.

DIAGNOSIS.—The state of things in an ordinary case of squint beginning in childhood is the following. In a state of rest, as when the attention is not directed to any object, or during sleep, or under an anæsthetic, the eyes are equally convergent; but as soon as any object is looked at, one eye fixes this object and the other rolls inwards. If the degree of flatness is alike in both eyes, and if the muscles in both are of equal power, sometimes one will be the squinting eye and sometimes the other; and in most cases this condition obtains for a time. The squint is then said to be 'alternating.' Generally speaking, however, it is for some reason easier to work with one eye than with the other; either because it is flat in a less degree, or because its

accommodation-muscle or its external rectus is stronger than the corresponding muscle of its fellow ; and then this eye is used in preference to, and gradually supersedes, its fellow ; becoming always the working, while that is always the squinting eye. The squint is then said to be 'permanent.'

TREATMENT.—It would appear at first sight, from the *rationale* of the affection, that the squint which depends upon flat-eye could always be prevented, or even cured, by the habitual use of convex spectacles ; but, as a matter of fact, the balance of power between the externi and the interni becomes deranged at so early a period of life that spectacles could not be applied until too late. In every pronounced case of strabismus it is necessary to perform an operation ; and the only question to be considered is that of the nature of this operation, and of the time most favourable for its performance.

In determining this question, the points chiefly to be taken into account are the state of vision, the balance of muscular power, and the age of the patient. When a squint becomes permanent, the vision of the habitually squinting eye frequently becomes impaired, apparently as a result of the continued mental suppression of the image which it receives, and in a person who is suffered to grow up to adult age squinting, this impairment of vision often falls little short of blindness, and admits of no remedy. No change is discoverable, generally speaking, by the ophthalmoscope ; but the power of responding to impressions upon the retina seems to be lost. On the other hand, as long as the squint is alternating, and each eye is used by turns, the sight does not usually suffer.

The immediate effect of tenotomy of one or both interni is to release the eyes from their position of enforced convergence, and to diminish the power of the interni to rotate them inwards. The divided muscles soon acquire new attachments farther back upon the eyeball than their former ones, so that their power is permanently diminished, and this diminution may even be in excess, so as to leave an undesirable preponderance of the externi, and a corresponding tendency to eversion. The surgeon, even by the best planned operation, cannot absolutely determine the future position of the eyes. That determination has to be effected by the muscles themselves under the guidance of vision ; and a perfect result after a squint-operation, by which is meant the restoration of parallelism when at rest, without impairment of the power of volitional convergence, can only be obtained by an instinctive rearrangement of the muscular forces concerned, a rearrangement mainly brought about by efforts to avoid double vision, which is often the immediate result of an operation. While, therefore, it is always possible to remove by tenotomy a coarse and manifest malposition, it is only possible to command a perfect result when the recti muscles are well-developed, when the acuteness of vision is equal or nearly so in the two eyes, and when the power of attention to visual impressions is sufficiently active to render double images distressing. The muscular development and the power of attention are both deficient in early childhood ; and hence, so long as vision does not suffer, it is better to defer operating for squint until about eight years of age. As long as the squint is alternating, there is no fear that the vision will suffer, and it is then safe and desirable

to wait ; but as soon as the squint becomes permanent, it is necessary to test the vision of the squinting eye from time to time, and to provide for this eye being exercised every day, by keeping the other closed or covered for short periods. If, in spite of such exercise, the vision of the squinting eye is found to be undergoing progressive deterioration, an operation should be performed without delay, at however early an age ; and the parents should be warned that it may perhaps be necessary, for the attainment of perfect harmony of movement between the two eyes, to operate again at some future time. Of late years many surgeons have preferred increasing the power of the externi by bringing their attachments nearer to the cornea, to the older operation of tenotomy ; but so much of the effect will depend upon the skill with which either operation is performed that it is impossible to lay down any general statement of their relative advantages.

It will sometimes happen that a child is first brought for advice at an earlier age than eight, in whom the squint has already become permanent in one eye, and in whom the vision of that eye has already begun to suffer. In such cases it is best to devote a few weeks to endeavours to improve the vision of the squinting eye by compulsory exercise ; and, if these endeavours should be in any degree successful, to continue them as long as improvement under their employment is perceptible. If no improvement should be produced, an operation should be performed without further loss of time.

(c) *The Convergent Squint of Short-sighted People.*—This is not a very common affection, and depends upon the fact that, spectacles to afford distant vision not having been worn, the externi, which produce the approximate parallelism of the optic axes required for distant vision, have not been exercised ; while the interni have been constantly exercised in producing convergence for the vision of near objects. The former muscles, therefore, have been suffered to fall into a condition of feebleness from disuse, while the latter have undergone abnormal development. In such cases the eyes are usually equally convergent, such a position giving single vision of near objects ; while double vision of distant ones is not irksome, on account of the indistinctness with which they are seen.

TREATMENT.—When the convergent squint of a short-sighted person is of small magnitude, it may sometimes be cured by wearing glasses which correct the short-sight for distance, and call upon the external recti to take up their proper function. More frequently, however, they fail to respond ; double vision is produced ; and tenotomy or advancement, followed by the use of spectacles, is required. Such cases usually turn out perfectly well.

2. **Divergent Squint.**—This is nearly always a consequence of defective vision of the squinting eye, which wanders outwards for want of guidance from visual impressions. It may also follow from unskilfully performed or excessive operations for the cure of convergent squint.

TREATMENT.—The operation for divergent squint is not a mere tenotomy, but requires the shortening of the internal rectus of the squinting eye, or its advancement to a point of attachment nearer to the corneal margin ; and the results of such an operation are less under command than those of tenotomy. The muscle may not attach itself firmly in the new position, or the connecting medium may stretch

after a short time. The operation may be undertaken more hopefully, the better the vision of the divergent eye; and it is often very successful. It is nevertheless most prudent, in every instance, to prepare the patient for the possibility of failure, or of only partial success. It is in no case likely that the defect will be increased by failure of the operation; and, as the chief motive for its performance is usually the improvement of appearance, it may be undertaken with propriety in almost every case.

3. **Complex Squint.**—The forms of strabismus in which the deviation is either upwards or downwards are not sufficiently numerous to be brought under general rule. They depend either upon spasm of the muscle producing the deviation, or upon paralysis or paresis of its antagonist; and every case must be investigated and treated upon its merits, by tenotomy or electrification, or both combined, according to the particular circumstances. Various irregular forms of strabismus are also seen, in the course of certain acute and chronic diseases of the nervous system, which entail loss or impairment of muscular co-ordination, such as meningitis and tabes dorsalis; but such forms are usually easily to be distinguished as symptoms of the general disorder, requiring no treatment or consideration apart from it. In chronic diseases, such as tabes dorsalis, it may be conducive to comfort to exclude the squinting eye from vision, for the purpose of obviating the inconveniences incidental to double images.

R. BRUDENELL CARTER.

STRANGULATION (*strangulo*, I choke).—

In pathology this term is employed to express either the process or the condition of constriction of a tube or pedicle, when it is so complete that the passage of the contents, or the circulation of the blood, is prevented. See HERNIA; INTESTINAL OBSTRUCTION; and OVARIES, Diseases of.

STRANGULATION as a Mode of Death (*strangulo*, I choke).—SYNON.: Fr. *Strangulation*; Ger. *Erwürgung*.

DEFINITION.—The act and the effect of constriction of the neck and air-passages by means of a ligature or of manual pressure (throttling). Death results essentially from asphyxia.

ÆTIOLOGY.—Strangulation is chiefly homicidal, but it may be suicidal or accidental. Accidental strangulation may occur in a variety of ways, as in the case of a child by tightening of a cravat round the neck, from the end catching in the wheel of a perambulator; in the case of a drunken woman by fixation and tightening of her bonnet-strings; and in the case of a cripple by a rope attached to a weight accidentally becoming tightened in front of the neck.

It was at one time doubted whether suicide could be effected by strangulation, owing to the fact that the hands relax when insensibility comes on, rendering it impossible to keep up sufficient tension on the ends of the ligature. But when the ligature is wound more than once round the neck, or some method is adopted by which the ligature can be tightened like a tourniquet, as by the insertion of a piece of stick which catches behind the ear or elsewhere, it is quite possible; and numerous instances are on record of suicide so effected. In most cases, however, the presumption is in favour of homicide, and in all cases of strangulation by manual pressure this may be looked upon as certain.

SIGNS.—In addition to the general indications of asphyxia (see ASPHYXIA) there are special signs of strangulation which vary with the degree of force employed, and the amount of resistance offered by the victim.

To strangle an individual of normal strength, and in full possession of all his faculties, is barely possible, without causing evident signs of violence on various parts of the body. The existence of injuries of this kind is valuable evidence of the mode of death. Very often cranial injuries are found, from the individual having been first knocked down by a blow on the head. Ecchymoses, abrasions, and other signs of mechanical violence are generally to be found in various parts of the body. If the strangulation has been effected by manual pressure, the front or sides of the neck exhibit bruised marks, corresponding to the thumb and fingers, with, perhaps, curved excoriations corresponding to the nails. The relative size of the marks produced by the thumb and fingers, and the direction of the nail marks, indicate the way in which the pressure has been exerted, and whether with the right or left hand.

When a ligature has been employed a mark is left on the neck, which varies with the nature of the ligature and the way it has been disposed. Usually it is a transverse, shallow furrow; single, double, or multiple, according to the number of folds; and continuous, or interrupted in places; differing from that of hanging, which is generally single and oblique, and higher up in the neck. The bottom of the groove is generally pale, and not parchmented as in hanging, owing to the pressure not being kept up so long as to lead to desiccation. Ecchymoses in the course of the groove are met with more frequently than in hanging, owing to the great violence frequently exerted.

Very commonly punctate ecchymoses are visible on the conjunctivæ, face, neck, and chest. They are considered by Tardieu to be more frequent in strangulation than in asphyxia from other causes, or than in cases of over-straining, which likewise may lead to them. In the subcutaneous cellular tissue, and in the fasciæ of the muscles above and below the hyoid bone, extravasations are frequently found, as well as on the external surface of the thyroid cartilage and trachea. The lungs vary as regards their vascularity; but on the surface it is common to find pseudo-membranous patches, which are due to the rupture of some of the superficial air-cells and collection of air-bubbles under the pleura. In the substance of the lungs congested patches, or apoplectic extravasations, are often found, varying in size, according to Tardieu's description, from half a franc up to a five-franc piece—extravasations, therefore, much larger than those usually found in suffocation.

TREATMENT.—The treatment of asphyxia from strangulation is that of asphyxia in general. See ARTIFICIAL RESPIRATION; ASPHYXIA; and RESUSCITATION.

D. FERRIER.

STRANGURY (*σπράγξ*, a drop; and *οὔρον*, urine).—SYNON.: *Stillicidium Urinæ*; Fr. *Strangurie*; Ger. *Harnstrenge*.

DEFINITION.—A spasmodic condition characterised by a frequent and urgent desire to pass urine, which is voided in drops or in very small quantities, with a sense of painful spasm in the perinæum and anus, and often unaccompanied by feeling of relief.

ÆTIOLOGY.—Strangury occurs in acute nephritis, in tubercular disease of the kidney, in renal colic, and in cases of intense congestion of the kidneys, however induced. It is also a diagnostic symptom of poisoning by cantharides, either from the internal use of the drug, or the external application of cantharides-plaster or of blistering fluid (see **CANTHARIDES**, Poisoning by). It is also met with after the administration of large doses of turpentine and its allies. Many morbid states of the kidneys, ureters, bladder, prostate, and urethra own strangury as a symptom. Especially is it a symptom of acute inflammations of the prostate (abscess in particular); acute inflammations of the bladder; or when the trigone is involved by morbid conditions, such as vesical calculus, growths, and tubercular disease. When acute gonorrhoea involves the *deep* urethra, this symptom is likewise met with. It must therefore be remembered that strangury may be produced by any condition which, directly or indirectly, leads to inflammatory affections of the trigone, or deep urethra; and it is in this way that the affection accompanies stricture of the urethra, enlarged prostate, foreign bodies in the bladder introduced from without, or fecal fistula.

Apart from inflammatory causes, morbid conditions of the urine, such as are met with in the oxalic-acid and uric-acid diatheses, may occasion strangury. Gouty prostatitis is therefore another factor in its causation.

TREATMENT.—The treatment of strangury is either preventive or curative. If it occurs during the administration or other use of drugs, such as turpentine or cantharides, it is imperative to discontinue their employment.

If we consider strangury as but a symptom of some other morbid condition, we must endeavour to remove the condition on which it depends. To relieve the more urgent and distressing local symptoms, we may have recourse to hot fomentations to the perineum, warm sitz-baths, suppositories of morphine and belladonna, or the cautious use of hypodermic injection of morphine.

JOHN HAROLD.

STRATHPEFFER, in Ross-shire.—Sulphur-waters. See **MINERAL WATERS**.

STREPTOTHRIX - INFECTIONS.—The *Streptotrichæ* are a group of micro-organisms, the exact position of which has given rise to some controversy. They seem to be somewhere intermediate between the fission-fungi or true bacteria, and the moulds, having probably a nearer affinity to the latter, among which they are placed by some writers. The genus itself has had various appellations, e.g. *streptothrix*, *cladotrich*, *actinomyces*, *oospora*. They are characterised by forming a network or mycelium of tangled filaments; these undergo true, though not dichotomous, branching. Spore-formation occurs, and in the tissues the more or less spherical nodules of the growth are often encased at their periphery within a palisade of radially disposed club-shaped structures, which are the swollen terminations of the mycelial filaments, and which in some instances seem to divide transversely and to form spores.

The best-known member of the group is the *actinomyces* or 'ray-fungus' (see **ACTINOMYCOSIS**), of which there seem to be several varieties or species. Another well-known condition due to a *streptothrix*

is the white variety of mycetoma or madura disease. See **MYCETOMA**.

The streptothrix-infections in their clinical characters generally resemble tuberculosis, from which they can be distinguished only by a microscopical or bacteriological examination of the tissues or discharges; hence it is not unlikely that they are of more frequent occurrence than has hitherto been supposed. Sooner or later chronic suppuration usually occurs. Flexner has described a case in which the clinical signs and symptoms were those of pulmonary tuberculosis.

Rarely, certain bacillary infections seem to form nodules with radiating filaments and clubs at the periphery. R. T. HEWLETT.

STRICTURE (*stringo*, I bind).—A contraction of a tube, duct, or orifice, for instance, of any part of the alimentary canal, or of the urethra. See **URETHRA**, Diseases of.

STRIDOR: STRIDULOUS (*strideo*, I creak). Stridor is the name given to a peculiar noisy form of breathing, produced in the larynx, trachea, or main bronchus; varying greatly in its character—being either harsh, musical, or crowing; and due to various forms of obstruction. The term *stridulous* is applicable to the respiration, the cough, or the voice, when they possess the characters of stridor. See **CROUP**; **LARYNX**; **PNEUMOGASTRIC NERVE**; **TRACHEA**; and **VOICE**.

STROKE.—A popular synonym for an attack of apoplexy or sudden paralysis. It is also used in the compound words, *sun-stroke*, *heat-stroke*, and *wind-stroke*, to indicate severally the sudden effects of these agents.

STRONGYLUS (*στρογγύλος*, cylindrical).—**SYNON.**: Fr. *Strongle*; Ger. *Pallisadenwurm*.—A genus comprising many species of nematoid worms. See **ENTOZOA**.

STROPHULUS.—The term is applied to a form of sweat-eruption occurring in infants and in children. It consists of small vesicles and papules, often appearing on the face, but also on other parts of the body. Thus it may occur on the side of the face in contact with the nurse, or elsewhere if the clothing is too warm or is allowed to remain wet. Occasionally it makes its appearance in febrile conditions, and if the digestion is disturbed, as during dentition, when the eruption is more universal, and its relation to miliaria becomes more evident. The eruption is the 'red gum-rash' of nurses. Probably in all cases the eruption is caused by retention of sweat, and the formation of superficial retention-cysts.

TREATMENT.—The eruption is usually trivial, but care should be taken to prevent irritation or infection of the eruption, by which eczematous conditions may readily be excited. The food and digestion of the infant should be attended to, and the site of the eruption kept dry and clean. The application of a dusting-powder containing oxide of zinc, talc, and, if necessary, a small quantity of boric acid, is useful, or a lotion of limewater, glycerine and oxide of zinc if the parts are reddened.

JAMES GALLOWAY.

STRUMA: STRUMOUS (*struma*, a scrofulous swelling).—Synonyms for scrofula and scrofulous. See **SCROFULA**.

STRYCHNINE, Poisoning by. — **SYNON. :** Fr. *Empoisonnement par la Strychnine*; Ger. *Strychninvergiftung*.—The seeds of *Strychnos nux vomica*, commonly known as *nux vomica*, as well as several other plants, owe their powerful toxic (excitomotor) properties to an alkaloid, *strychnine*; and in a minor degree to another alkaloid, *brucine*, which is said to produce the same physiological effects as strychnine. Strychnine is a white crystalline substance, very sparingly soluble in aqueous liquids, to which, however, it communicates an intolerably bitter taste. It is more freely soluble in acid and alcoholic liquids. When mixed with flour and sugar, and coloured by admixture with either soot or Prussian blue, strychnine forms the basis of several well-known forms of 'vermin-killer.' In spite of its repulsively bitter taste, strychnine has been administered with homicidal intent in such liquids as infusions of tea and cocoa, and in other media.

ANATOMICAL CHARACTERS.—The anatomical characters after death by strychnine-poisoning are very ill-marked, and at most consist of some congestion of the vessels of the spinal cord; and even this may be wanting.

SYMPTOMS.—Except when taken in the form of pill, strychnine and all substances containing it produce an immediate and intensely bitter taste, which is also at the same time of a quasi-metallic character, and is very persistent. Since the fatal dose—half a grain of the alkaloid—is small, and the poison is readily soluble in the acid gastric fluid, its physiological effects are, as a rule, not long delayed. They may be unmistakable after the lapse of two minutes; but commonly they are not well-marked till five, ten, or even twenty minutes after administration. They begin with a stage of restlessness, excitement, and a vague sense of impending peril. The special senses, too, are often preternaturally sharpened. A feeling of choking or impending suffocation ensues; then there is a trembling of the whole body; jerkings of the head; and, often in a moment, the whole body becomes stiff and rigid, assuming a bow-like form (*opisthotonos*), that is, bent backwards and resting perhaps on the head and heels only. The muscles even of the chest and abdomen are tense and fixed, so that respiration is impeded, giving rise to more or less cyanosis. The feet are either incurvated or excurvated. The angles of the mouth are drawn down, so as to give rise to the well-known *risus sardonius* of tetanus. Attempts to administer medicine by either cup or spoon have been known to result in the patients biting the cup or spoon in two, in consequence of a violent spasmodic closing of the jaws. During the paroxysm, and indeed throughout the intoxication, the cerebral faculties are unimpaired, and the convulsions are purely of spinal origin. The pupils are dilated. In a few minutes, and often in half a minute, the muscular tension relaxes, and there is a complete remission of the spasms. The patient lies exhausted, and bathed in perspiration; the rapid pulse of excitement falls in frequency; respiration becomes more normal; and the dusky lividity of the countenance passes off. This remission is, however, of no long duration. A gentle touch, a footstep, even a breath of air impinging on the patient, results in a new crisis; and often with a wild, despairing cry, a renewed convulsion, similar to but more intense than the preceding one, is ushered in. The patient rarely dies during the first or second paroxysm, but the alternation of convulsions and quiet is repeated

again and again till death ensues, usually in half an hour or an hour; or in non-fatal cases the fits become less and less frequent, less intense, and eventually cease. Death takes place commonly during a paroxysm, from asphyxia; but it may also occur in the intervals between the paroxysms, from exhaustion.

DIAGNOSIS.—The characteristics of strychnine-convulsions are so well marked, as already described, that there is little likelihood of the nature of the case being overlooked; and the only disease with which strychnine-poisoning can readily be confounded is tetanus—traumatic, idiopathic, or hysterical. In the hysterical form of the disease, as described by some writers, the well-marked hysterical symptoms, the closed or half-closed eyes, the desire to be fanned, and the incomplete remissions of spasm, serve for diagnosis. Except in the history, there is nothing to distinguish between the traumatic and idiopathic forms of the disease, so that what is here stated with regard to the diagnosis between strychnine-tetanus and traumatic tetanus, applies also to the idiopathic form. In traumatic tetanus the muscular symptoms begin with pain and stiffness of the neck and jaws, gradually passing into spasms; and the jaw is one of the earliest parts affected. The strychnine-tetanus, on the other hand, develops rapidly, and begins in the back of the neck or the extremities, or a general convulsion at once seizes the whole body. Moreover, the jaw is usually last affected, and its muscles relax first. The strychnine-relaxation is complete, or rarely almost complete; while in traumatic tetanus there is permanent muscular rigidity, and no complete remission of spasm. Fatal strychnine-tetanus is an affair of minutes, or at most of half a dozen hours; while traumatic tetanus never kills within twelve hours, and generally extends over a few days. In strychnine-poisoning the most trivial movement or touch will set up a convulsion; while during the spasms firm grasping of the hands, and hard rubbing of the rigid muscles, will often afford grateful relief. This distinction is not marked in traumatic tetanus. An analysis of the urine by Stass's method, which often affords certain indications of strychnine, and may be roughly made in a few minutes, will, in doubtful cases, at once remove all uncertainty as to the nature of the disease.

PROGNOSIS.—The prognosis is at all times doubtful. The patient's life cannot be considered safe till the convulsions clearly exhibit marked decrease, both in frequency and intensity.

TREATMENT.—Should the convulsions have already set in, the use of the stomach-pump is out of the question. An emetic of warm water with mustard, or ammonium carbonate, should be given without a moment's delay. The free use of chloroform not only alleviates the pain and allays the spasms, but allows time for the elimination of the poison from the system, and the administration of remedies. The patient should be touched as little as possible, and absolute quiet observed in the sick-room. Excellent results have ensued from the administration of large doses of potassium bromide; even half an ounce in one dose has been given. The gastric irritation produced by such large doses of the bromide as are necessary militates, however, against its use. Chloral-hydrate in full doses, and the anæsthetic administration of chloroform-vapour, are the best remedies. The introduction into the stomach of a diluted solution of potassium permanganate has

been recommended. Nitrite of amyl has been advised by some. Strychnine-poisoning more often ends fatally either from the lateness with which remedies are applied, or their non-application, than from their inefficient character.

THOMAS STEVENSON.

STUPE (*stupa*, tow).—A synonym for a fomentation. See FOMENTATION.

STUPOR (Lat.).—SYNON.: Fr. *Stupeur*; Ger. *Stupor*; *Stumpfsinn*.—A partial loss of consciousness. See CONSCIOUSNESS, Disorders of.

STUTTERING.—See STAMMERING.

STY (Sax. *steigan*, to rise up).—SYNON.: *Hordolum*; Fr. *Compère-Loriot*; *Orgelet*; Ger. *Gerstenkorn*.—This term is applied to a boil on the margin of the eyelid. A sty does not differ in any essential respect from a boil in any other situation, but it is usually of small size, and commences in the follicle of an eyelash. See BOILS.

STYPTICS (στυψω, I contract, make firm).

DEFINITION.—Styptics are substances which arrest superficial bleeding by their local astringent action. They include many of the drugs which are known as internal astringents or hæmostatics.

ENUMERATION.—The chief styptics are: Cold; Pressure and other mechanical measures; the Actual Cautery; Perchloride of Iron; Alum; Tannic Acid and Hamamelis; Acetate of Lead; Nitrate of Silver; Extract of supra-renal bodies; and Collodion.

ACTIONS AND USES.—Styptics act in various ways, and assist the natural modes of arrest, such as retraction and contraction of vessels and coagulation of the blood. Some arrest hæmorrhage by aiding the rapid formation of a coagulum, others by contracting the smaller vessels, and a third class by condensing and coagulating the albuminous tissues around the vessels. Some styptic substances act in more than one direction.

All mechanical measures which promote the coagulation of the blood will obviously tend to arrest bleeding by plugging the wounded vessels. Cotton-wool and similar materials are useful for this purpose, and a pledget of cotton-wool soaked in the liquid extract of Hamamelis (Hazeline) is a well-known remedy for bleeding piles. Collodium, also, acts mechanically by exerting pressure over the surface and thus preventing the blood from issuing. Alum, acetate of lead, and perchloride of iron cause coagulation of the blood. The actual cautery at a dull heat seals up small bleeding points. Pressure to the surface, ice or cold sponges, cause the vessels to contract, and thus prevent the blood-flow in superficial hæmorrhage. Tannic acid and substances containing it act more directly on the tissues around the vessels, and also assist in the formation of blood-clot.

The use of styptics is usually limited to the general oozing which may follow the application of leeches or the infliction of small wounds by accident or for surgical purposes. In some cases of *post-partum* hæmorrhage the injection of hot water into the vagina and uterus has a powerful action in arresting hæmorrhage, probably due to reflex contraction of the vessels and of the uterus itself (see PUERPERAL DISEASES). As adjuvants to the action of styptics all measures which lessen the local blood-pressure, such as complete quiescence, cold

food, elevation of a wounded limb, and the like, are of the first importance; and internal medication by ergot and other drugs which contract the blood-vessels is also indicated. If the oozing is severe and continuous, recourse must be finally had to surgical interference by local ligation of vessels when practicable.

FREDERICK WILLCOCKS.

SUBCUTANEOUS INJECTION.—See HYPODERMIC MEDICATION.

SUBCUTANEOUS NODULES.—See RHEUMATISM, Acute, p. 1412.

SUBPHRENIC ABSCESS.—See p. 1620.

SUBSULTUS TENDINUM (Lat.).—A twitching movement of the tendons, caused by sudden momentary contractions of the muscles to which they belong. This is especially apt to show itself in the tendons about the wrist in the later stages of many low fevers. It manifests itself principally in states of great prostration, and is often associated with delirium or other signs of cerebral irritation. See TYPHUS FEVER.

SUCCUSSION (*succussio*, a shaking).—A method of physical examination, which consists in suddenly shaking the trunk of the patient, so that certain sensations or sounds may be elicited, which are indicative of the presence of gas and fluid in a hollow space, such as the pleural cavity. See PHYSICAL EXAMINATION; and p. 1588.

SUDAMINA.—See MILIARIA; and SUDORIPAROUS GLANDS, Diseases of.

SUDORIFICS (*sudor*, sweat; and *facio*, I make).—A synonym for diaphoretics. See DIAPHORETICS.

SUDORIPAROUS GLANDS, Diseases of.—

The sweat-glands are liable to diseases of structure and of function, which are often difficult to elucidate on account of the close relation of the sweat-coils and ducts with the epithelium of the integument and sebaceous glands.

DISEASES OF STRUCTURE.—As a result of the position of the sweat-coils which lie deep in the cutis, and the length and tortuous course of their ducts through the cutis and epidermis, the sweat-glands are very liable to cystic dilatations. These dilatations may be due simply to excessive action of the sweat-coil, or perhaps more usually to some obstruction in the course of the duct. The obstruction may be of temporary character only, or due to chronic forms of dermatitis; in the latter case the cystic changes become permanent and often of peculiar character.

It is rare to find the coil itself suffering from cystic change, but the duct readily becomes distended in its course both through the cutis and through the epithelium.

Hidrocystoma is one of the most characteristic of the diseases caused by sweat-cysts. It occurs most commonly on the face, appearing as a multiple eruption of small thick-walled hemispherical vesicles, without obvious signs of inflammation, which last for some time and disappear without breaking. They are larger and the eruption more extensive in hot weather or during sweating due to hard work. The disease has been specially noted in the case of

washerwomen, cooks, and others liable to hyperidrosis. The cyst in this case appears to occur in the subepithelial portion of the duct.

The terms *Miliaria*, *Miliaria crystallina*, *Crystallina*, *Sudamina*, have been applied to small collections of clear fluid occurring in the upper layers of the epithelium and in relation to the course of the sweat-duct through the epithelium. They occur in cases of profuse sweating, as in fevers, and are characterised by their perfectly clear contents and non-inflammatory characters to the naked eye.

Miliaria rubra, *Miliaria papulosa* are names given to the lesions occurring in the eruption known as *prickly heat*. The evidence is not perfect, but it seems most probable that the lesion consists of a dilatation of the sweat-duct in the lower horny layers and below this level. Inflammation of the epidermis and the immediately underlying cutis occurs, and the profuse, intensely irritable eruption occurring even in this country in hot weather, but much more commonly in the tropics, results. The eruption affects large areas of the body, and as the result of scratching is apt to lead on to various secondary eruptions of eczematous or presenting purulent characters.

Miliaria vesiculosa, *Miliaria alba* (see STROPHULUS) are names occasionally applied to sweat-cyst eruptions with less violent symptoms. When the cyst occurs in the sweat-pore and involves the horny layer mainly or altogether, its contents may become firm and epithelial cells collect in it, and small concretions may be at length produced (see MILIUM). It is very rare to find actual *suppuration* in a sweat-gland, but certain small abscesses in such positions as the axilla and perinæum are said to arise in this way. A rare condition of multiple suppurative necrosis of sweat-glands has been described (*Spiradenitis vel Hidroadenitis disseminata suppurativa*, Dubreuilh, Unna, and others).

Adenoma of the sweat-glands (*Spiradenoma*; *Syringadenoma*) is described, but the cases recorded are few and the histological descriptions not very convincing. *Carcinoma* commencing in the sweat-glands has been more definitely described, and there can be little doubt that some of the unusual forms of superficial carcinoma are actually sweat-gland cancers. They may appear as single growths, but in some cases a multiple eruption of small malignant tumours with ulceration has been traced to the coil-glands. At present the diagnosis of this type of malignant disease cannot be made with certainty apart from careful histological study.

DISEASES OF FUNCTION.—Hyperidrosis.—Excessive sweating occurs as a concomitant of pyrexia, and associated with conditions of severe debility and collapse. It is also found to occur in cases of nervous and emotional disturbance. In such cases it is usually general, and as a rule requires no special treatment. Precautions should be taken to cleanse the surface of the skin by sponging or baths, so as to prevent decomposition of the sweat especially in the axillæ, groins and flexures, and the development of troublesome dermatitis. Occasionally cases are seen of localised sweating, such as on one side of the face or one side of the body. These cases are often due to injury or disease of the nervous system, but may be excited reflexly by the stimulation of sensory nerves. Thus in some cases mastication—for example, eating anything

sour, such as an apple—invariably produces profuse sweating on one side of the face.

More trouble and suffering is experienced by patients who suffer from hyperidrosis in ordinary health. The whole body may be affected, but usually certain portions sweat most profusely, especially the scalp, face, axilla, groin and perinæum, and the hands and feet. The sweating may produce a moist surface or be so profuse as to roll in drops and streams from the affected parts. In the case of a young lady, whenever she concentrates attention on her hands, e.g. by taking up a needle to sew, the hands become moist, and in a few seconds the sweat pours off them so as to render work impossible. In many patients the condition is chronic, so that the usually affected parts are in a constant state of unpleasant wetness. In such cases the epithelium of the palms and soles becomes sodden, thickened, and corrugated, the skin on the feet becomes tender, easily chafed, and blistered, and in all situations may become reddened and inflamed, thus producing eruptions resembling intertrigo and eczema. The liability of such a surface to become infected with bacteria is great, and consequently pustular eruptions in the axilla and about the genitals arise; but most troublesome is the condition of *Bromidrosis*, produced by the growth of bacteria, which emit foul odours. The ordinary white staphylococcus of the skin is capable of producing the unpleasant odour of butyric acid, and is the organism mainly concerned in the production of bromidrosis, but in all probability other organisms may also produce this effect. In the case of persons careless about personal cleanliness this condition is common, but even in others the greatest care as to cleanliness is necessary to prevent the pungent odour arising to such an extent as to render their presence in a room at once noticeable and unpleasant.

TREATMENT.—In cases of hyperidrosis care should be taken to secure as good a state of health as possible whenever debility is ascertained. In many cases a condition of feeble circulation through the extremities, with cold and congested hands and feet, is noticeable. For such patients suitable clothing, exercise, massage, and bathing are of importance, and the administration of remedies acting on the vascular system, such as digitalis, strophanthus or strychnine, is of great service. In a few cases the carefully regulated administration of ergot has done much good. The internal administration of sulphur is said to produce very good results. Where the nervous system is at fault, especially in cases of hemi-hyperidrosis, specific remedies have proved of little service; in functional nervous disorders, especially of hysterical character, general treatment is often very valuable in checking and curing hyperidrosis. Belladonna and atropine, though frequently administered, are very disappointing in their results, and only cause much inconvenience and even danger.

In localised hyperidrosis the utmost precautions as to cleanliness must be observed, so as to prevent bromidrosis, or to cure this most offensive condition if it has occurred. The affected parts, especially the feet, should be carefully washed with warm water and soap once, twice, or even more frequently daily, and thoroughly dried. They should then be disinfected as thoroughly as possible—for this purpose solutions of boric acid are very effectual—and then dusted over with the fine powder of boric acid either pure or diluted as may be necessary with

oxide of zinc and talc-powder. Other antiseptics or astringents used for this purpose and found to be of service are carbolic acid, salicylic acid, solutions of formalin, trichloroacetic acid, alcohol, quinine, chromic acid, alum, and tannin. When much walking has to be done the feet are very liable to suffer injury readily, to chafe, and become badly affected. In addition to cleanliness, a lubricant for the skin is then necessary. Benzoated lard containing from 2 or 3 per cent. of salicylic acid rubbed into the stockings or feet regularly is one of the best precautionary measures in such circumstances.

Anidrosis.—Absence or diminution of sweat occurs in various degrees. It may be symptomatic as in fevers and diabetes, due to congenital defects such as ichthyosis even in slight degree, or the result of skin-diseases such as scleroderma, leprosy, &c. Persons in good health frequently sweat very slightly, even on exertion.

TREATMENT.—In some cases warm baths followed by shampooing are of service; but, as a rule, general 'tonic' treatment is indicated, with special reference to any defect of the health which may be discovered.

Chromidrosis.—Cases of coloured sweating are rare, and due to several causes. Such cases should be investigated with care so that imposture is not overlooked.

The word is perhaps especially used in connection with a peculiar and unexplained group of cases in which a pigmented material varying in colour from sepia to a tint of blue is deposited from the sweat on the forehead, cheeks, and sides of the nose, more rarely on other parts of the body. It occurs sometimes in large quantity, and may appear as a scaly or powdery deposit on the surface.

The condition occurs nearly always in females, and is usually associated with marked neurotic phenomena, but its exact aetiology is unknown.

Red sweat occasionally occurs in the axillæ and groin and elsewhere; blue tints are also noted; both are due to the presence of bacterial secretions. Green sweat occurs in copper-workers and others who have absorbed the metal in food. See p. 209.

Hæmidrosis or bloody sweat is very rare, and occurring among hysterical women should be investigated with scepticism to ascertain the exact source of the blood, and to avoid overlooking an artificial eruption. It has apparently occurred in infants, and perhaps in hæmophilic persons and others a certain amount of blood-pigment may occur in the sweat.

Phosphorescent sweat has been noted.

Uridrosis.—Urea is said to be occasionally excreted by the skin in more than the usual minute traces, and to be actually deposited on the surface as fine powder or scales. It has recently been ascertained that the sweat contains excretory matter, toxic on injection into the lower animals, and increased on muscular exertion.

See also **DYSIDROSIS**.

JAMES GALLOWAY.

SUFFOCATION (*suffoco*, I stifle).—SYNON. : Fr. *Suffocation*; Ger. *Erstickung*.

DEFINITION.—The term 'suffocation' is sometimes employed synonymously with asphyxia. In the strict medico-legal sense it signifies asphyxia induced by obstruction of the respiration otherwise than by direct pressure on the neck (hanging, strangulation), or submersion (drowning).

ÆTIOLOGY.—Death by suffocation is usually the result either of accident or of homicide, rarely of suicide.

Suicide by suffocation is indeed not unknown. Cases of suicide by immurement in a box or trunk, or by thrusting a pad or other obstruction down the throat, have been reported; and it has been averred that slaves, both in ancient and modern times, have committed suicide by rolling the tongue back into the pharynx.

Accidental suffocation is very common in diseases causing occlusion of the air-passages; it may be due to the impaction of pieces of food or other obstacles in the pharynx; to the entry of foreign bodies into the larynx, as a seed, coin, or food, in cases of bulbar or general paralysis, or matters vomited during a state of insensibility; to mechanical pressure on the chest and abdomen, as in crowds, or in falls of earth or heavy bodies; to various diseases preventing the expansion of the lungs; to diseases of the lungs themselves; or to obstruction of the pulmonary circulation. Suffocation of new-born children by smothering under bed-clothes, non-removal of maternal envelopes, or overlying, may happen from carelessness as well as from intent. See **OVERLYING**.

Homicidal suffocation is resorted to chiefly in infants, or in the case of persons feeble and infirm, or rendered powerless or insensible by intoxication or narcotics. Closure of the mouth and nostrils by the hands, or obstruction of the mouth and nostrils by a pillow, mattress, or the like, perhaps combined with pressure on the chest, is the method usually adopted. Formerly suffocation by mechanical pressure on the chest was a judicial punishment—the *peine forte et dure*.

SYMPTOMS AND SIGNS.—The mode of death, and the general *post-mortem* indications, are those of asphyxia (see **ASPHYXIA**). The special indications of suffocation, and the way in which it has been brought about, may be evident from the place where the body is found, and its surroundings; or foreign bodies, or disease obstructing the air-passages or respiratory mechanism, may be clearly evident on *post-mortem* dissection; or there may be marks of violence and indications of pressure on the chest, flattening of the nose, &c., pointing to homicidal violence. The absence of marks of constriction of the neck excludes strangulation and hanging.

But in the absence of all such indications as have been enumerated above—and they may all be absent, especially in cases of infanticide—the question is whether any trustworthy conclusion can be formed as to asphyxia by suffocation.

The condition of the lungs is of especial importance in this relation. The lungs may be congested, or pale, or congested only posteriorly; but the surface is often uneven, owing to an emphysematous condition of some of the superficial air-cells; and in particular the lung looks as if it had been sprinkled with minute drops of a dark purple fluid. These spots, not much larger than a pin's head, are known as 'Tardieu's spots,' and are due to minute capillary extravasations under the pleura. They are not, however, confined to the surface of the lungs, but are found also in considerable numbers on the thymus gland, the base of the great vessels, under the parietal pleura, and also under the pericranium. Tardieu, who first called special attention to these spots, considered them absolutely diagnostic of

death by suffocation, as distinguished from other modes of asphyxia. But numerous other observations have shown that this cannot be accepted as correct, inasmuch as similar extravasations have been found in cases of hanging, strangulation, drowning, and deaths from cerebral injuries. It seems, however, fairly well established that they occur most frequently, and in largest number, in suffocation, especially in infants. Their formation depends on excessive vascular tension during the asphyxiating process. Similar spots have been found in the lungs of still-born foetuses, conditioned by obstruction of the placental circulation; and in the lungs of new-born children, perishing from other causes, extravasations of a like nature have been observed. It would, therefore, be unsafe to rely absolutely on Tardieu's spots as indications of suffocation, though, in the absence of other causes of death, and in presence of these spots in large numbers and in clusters, the opinion of death by suffocation would be fairly justified.

TREATMENT.—The treatment of impending suffocation is that of asphyxia. *See* ARTIFICIAL RESPIRATION; ASPHYXIA; and RESUSCITATION. D. FERRIER.

SUFFOCATIVE BREAST-PANG.—A synonym for angina pectoris. *See* ANGINA PECTORIS.

SUFFUSION (*suffundo*, I pour down).—The process or the result of the unnatural pouring out of a fluid into the tissues; closely analogous to effusion and extravasation. *See* EXTRAVASATION.

SUICIDAL INSANITY.—*See* INSANITY; and MELANCHOLIA.

SULPHONALISM.—**DEFINITION.**—A group of symptoms said to be occasioned by the prolonged administration of sulphonal.

Sulphonal is one of the favourite hypnotics in institutions for the insane. Being tasteless and odourless, fairly soluble in such vehicles as hot soups, not productive of digestive derangements, and obtainable without medical prescription, it has become very generally known to the laity; and thus a 'sulphonal-habit' has been met with which demands recognition and treatment.

Similar habits have been noted in the case of antipyrine and trional.

SYMPTOMS.—The prominent symptoms noted in the recorded cases of poisoning by sulphonal are vomiting, abdominal discomfort, gastro-intestinal disturbance, obstinate constipation, cephalalgia, tinnitus aurium, drowsiness, inco-ordination, diminution of reflex excitability, puffiness of the eyelids, ptosis, and more or less mental, physical, and cardiac weakness. The urine, which decomposes slowly, becomes of a port-wine or Burgundy-red colour, possibly with coincident evidences of renal inflammation. This peculiar colour of the renal excretion, which is said to be due to the presence of non-ferrous hæmatin or hæmatoporphyrin (*see* SPECTROSCOPE IN MEDICINE; and HÆMATOPORPHYRINURIA), resulting from extensive disorganisation of hæmoglobin, is thus of clinical significance, serving as a means of recognising the abuse of the drug, and to be regarded as an index for its immediate temporary discontinuance. Of rarer occurrence is a papular, measly, erythematous, or scarlatiniform eruption. Possibly many of the phenomena noted in the recorded cases were symptoms of the disease

from which the patient was suffering, and ought not to have been accredited to the drug. *See* HABITS; and DRUG-ERUPTIONS.

Antipyrine may give rise to various affections of the skin (p. 421); trional produces but few after-effects, though its continued use leads to mental and moral deterioration.

TREATMENT.—The treatment of sulphonalism consists in suspending the administration of the sulphonal until all unfavourable symptoms have disappeared; and by the use of diuretics, promoting the elimination of the drug, at least in cases where it has been taken in large doses. If it should have to be taken regularly for a lengthened period, the use of it should be occasionally intermitted for a few days, another suitable hypnotic being substituted temporarily.

JOHN HAROLD.

SULPHUR-WATERS.—*See* MINERAL WATERS.

SUNBURN.—**DEFINITION.**—Under this designation are included the superficial local effects of exposure to the sun's rays.

Such effects vary greatly with the susceptibility of the individual, the surrounding circumstances and conditions, and the duration or repetition of the exposure. They may be comparatively slight and transient, as is commonly the case; or they may be very severe, and involve considerable and prolonged suffering, and sometimes serious constitutional disturbance.

ÆTIOLOGY.—Delicate, thin-skinned, fair-complexioned subjects are most liable to suffer from sunburn; the thick-skinned and swarthy much less so. The rays reflected from snow or ice (especially through rarefied atmosphere), as experienced by Alpine climbers, or from water-surface (sea or river), are most hurtful. Exposure to the rays from powerful electric arc-lights may be followed by effects more or less similar to those resulting from exposure to the solar rays (Tyndall, Hewetson).

There is evidence to suggest, if not to prove, that sunburn is due not to the heat-rays alone, but rather to other influences, especially to those of the violet and ultra-violet rays. A beam of sunlight on striking the skin is, to a great extent, filtered of its red and calorific rays by the epithelial layer, so leaving the violet and ultra-violet rays free to begin their work on the blood and the vaso-motor nerves lying in their path. Active and inflammatory changes of very varying intensity follow, and, not infrequently, local necrosis as well—effects almost identical with those produced by Röntgen Rays, with which, physiologically, these rays of short vibration, especially the reflected rays, may in some way be associated.

SIGNS AND SYMPTOMS.—First, in the slight, familiar cases, there is redness of the skin from vascular congestion (*erythema solare*), accompanied by tingling and sense of heat. This is followed by desquamation and subsequent pigmentation, either uniformly diffused ('tanning') or especially localised in spots, giving rise to freckles (*ephelis*). All these subside more or less speedily, and are the only points to be noted. Pigmentation may occur without previous noticeable erythema, as is evidenced by the 'browning' of patients who have wintered at Davos or other Alpine resorts, or who have been otherwise exposed to snow-reflected sun-rays.

Secondly, in the more severe cases, the erythema is followed by vesication, and more or less deeply

extending inflammation of the skin. An erysipematoid condition may ensue, with œdema and considerable swelling of the part, severe pain, and great tenderness. This may be accompanied by constitutional disturbance.

Thirdly, in the worst cases, happily very rare, sloughing and ulceration to greater or less extent occurs. See HEAT, Effects of Severe and Extreme; and SUNSTROKE.

TREATMENT.—In the slighter cases of sunburn little or no treatment is required. Simple powdering with starch, oxide of zinc, bismuth or boric acid, or dabbing with elder-flower or rose-water, with a little eau-de-Cologne or solution of acetate of ammonium, may be pleasant. The more severe cases must be treated on general principles. Lead-lotion with morphine or cocaine will relieve the heat and pain. The solution of subacetate of lead with glycerine and elder-flower or rose-water is also a good application.

It is, however, almost more important to prevent than to cure. The preventive treatment obviously consists in protecting exposed parts from the deleterious effects of the sun's rays. When sunshades, veils, and masks are inconvenient—especially in cases of Alpine climbers and others—the writer recommends that the face, arms, and other exposed parts should be painted over with the 'grease paints' used by actors for 'making up.' Brown is the best colour, but flesh colour will serve. Pigments mixed with glycerine of starch may be used. For sunshades, veils, and similar protections, brown or some shade of red or yellow is the best colour. The 'tanning' of the skin, which naturally takes place, is the best protective against suffering from subsequent exposure.

In those who have been year by year subject to 'sunburn,' the skin becomes dry and wrinkled, from the effect upon the sudoriparous and sebaceous follicles, as well as browned by pigmentation. Such result may be to some extent prevented by the ancient practice of oiling the skin.

For the freckling and browning of the skin after sunburn, lotions of perchloride of mercury or boric acid, with emulsion of almonds, and glycerine, or juice of cucumber, are extensively used with more or less beneficial effect.

ROBERT L. BOWLES.

SUNSTROKE.—**SYNON.** : Insolation ; Heat-stroke ; Fr. *Coup de Soleil* ; Ger. *Sonnenstich*.

DEFINITION.—Certain pathological conditions resulting from exposure to solar or artificial heat.

Three well-marked varieties of sunstroke are recognised, namely: (1) Exhaustion and failure of the heart's action in *syncope*; (2) A condition like shock, in which the nerve-centres, and especially the *respiratory*, are affected, causing rapid failure of the respiration and circulation; and (3) Intense *pyrexia*, due to vaso-motor paralysis, and to the nerve-centres being over-stimulated and then exhausted by the action of heat on the body generally.

ÆTIOLOGY AND PATHOLOGY.—These morbid conditions being due to heat alone, are not peculiar to any country or climate, and are liable to occur wherever persons are exposed under any circumstances to great heat, whether solar or artificial. Soldiers marching or fighting, when oppressed by weight of clothing and accoutrements, are apt to suffer either from simple heat-exhaustion, or from that form of insolation which results from direct action

of the sun on the head and neck. This is common enough in India during the hot season, in other tropical countries, and in America; and is not unknown in Europe or even in England during the heat-of summer. Workmen, artificers, stokers, and other persons employed in heated rooms, hospitals, barracks, tents, and even ships, especially in hot climates, are liable to suffer from heat-exhaustion, which may pass into the dangerous condition of fever or insolation.

But the most frequent cases are those which occur in houses, barracks, tents, ships, by night or in the day, away from the direct solar rays. A form of disease sometimes described as 'ardent fever' in India is this condition supervening on the ordinary phenomena of ephemeral fever. It seems pretty well understood that heat alone is the effective cause of the so-called sunstroke.

A dry air, such as that of North India, with hot winds, is much better tolerated at a high temperature, than the damp atmosphere of Bengal at a much lower one; for the dry hot air favours evaporation from the surface, and thus keeps the body cool, while in the damp, heavy atmosphere the natural cooling function is almost in abeyance. Defective ventilation, stillness of air, crowded habitations, and close valleys and ravines are conditions in which heat-shock and heat-fever are prone to occur. Vigorous, healthy persons of moderately spare frame, possessing sound viscera, and leading temperate and regular lives, can tolerate a great amount of heat, in an otherwise pure atmosphere, and are much less liable to suffer from it than those whose environment is unfavourable and power of resistance impaired. Acclimatisation has also considerable influence in conferring toleration. New arrivals are more prone to suffer than those who have become accustomed to the climate. It is well known that the native can bear an amount of sun on his shorn head, neck, and half-naked body with indifference, if not pleasure, that would very soon prostrate a European. But to a temperature of the air rising above a certain standard, all succumb; and the natives of India suffer like others, and die in numbers every year from *loomarna* or 'hot-wind stroke.'

The exact amount and duration of toleration of a high temperature depend to a great extent, therefore, on the vigour of constitution and the present state of health. The natural refrigerating powers of the body, when in health, are such as to enable men to support very high temperatures, much above that of the normal state of the body. Disordered health, dissipation, over-fatigue, anything in fact that depresses nerve-power, reduces the normal physiological capacity, and consequently renders a man more liable to succumb. Persons who habitually indulge in alcohol to excess are peculiarly vulnerable by heat, and fevers by disturbing the thermotactic arrangements of the body are apt to predispose to the injurious effects of high temperatures.

ANATOMICAL CHARACTERS.—In cases where death has taken place suddenly, as from *shock*, there is no very remarkable *post-mortem* appearance. The heart may be found firmly contracted, but not always so—it may be flaccid. The lungs and the brain and its membranes may be somewhat congested, but not invariably. As in cases of shock, the venous trunks, specially those of the abdomen and the right side of the heart, may be too full of blood, and the pulmonary vessels overloaded; but

the lungs in some cases are blanched from absence of blood, owing to contraction of the pulmonary arterioles. The blood itself is dark and grumous, and is found effused in patches of ecchymosis, indeed rendering the body more or less livid; the coagulability of the blood is also impaired, and it is wanting in oxygen.

In death from ordinary cases of *thermic fever*, the lungs and respiratory organs are often deeply congested; the heart is firmly contracted from coagulation of myosin; the whole venous system is engorged; and the body even before death is marked by petechial patches, or extensive ecchymoses of a livid appearance. The blood is generally more fluid and grumous than natural; its coagulability is impaired; and it is acid in reaction. The red corpuscles, though generally presenting no abnormal change, are somewhat crenated, and have less tendency to form rouleaux than in health; and the quantity of oxygen is much diminished. The body for some time after death retains a high temperature; when first opened, the viscera feel pungently hot, and the incisions drip dark blood. *Rigor mortis* comes on very rapidly, from early coagulation of myosin.

The brain and membranes may be found congested, and in some cases there may be evidence of meningitis. Serous effusions into the ventricles, or hæmorrhage into the brain-substance, may have occurred, and are not improbable in the congested condition sometimes existing in the head; but the cause of death is asphyxia, not apoplexy, and the most important changes are found in connection with the thoracic viscera.

SYMPTOMS.—(1) Syncopal form.—**SYNON.** : Heat-exhaustion.—Simple exhaustion and syncope may occur under great fatigue or over-exertion, or depression from any cause, during exposure to a high temperature. There is depression of nerve-force, and prostration of muscular power; the skin is pale, cold, and moist; and the pulse is quick and feeble. Death may occur rapidly in the state of collapse from failure of the heart. Complete recovery is frequent.

(2) Asphyxial form.—**SYNON.** : Sunstroke proper.—Asphyxia and apnoea may come on very rapidly, after certain premonitory symptoms of depression and weakness, though occasionally without prodromata, during exposure, especially of the head and spine, to the direct rays of a powerful sun, when the atmosphere is much heated, and the nervous energy has been depressed by over-fatigue, dissipation, or illness. The brain and nerve-centres, especially the respiratory, are overwhelmed by the sudden elevation of temperature, and respiration and circulation fail, the failure of the latter being probably due to the inhibitory influence of the vagus. When death takes place, as it does sometimes very suddenly, during great excitement or exertion, it has been attributed to rapid *ante-mortem* coagulation of the cardiac myosin, which takes place at a temperature of 113° F. or a little below that level in cases of over-fatigue. This, however, though it may occur occasionally, is generally a *post-mortem* change, the heart's action being brought to a close by the heat; in the same manner as it has been shown by Claude Bernard and Lauder Brunton that the effect of high temperature on animals is first to accelerate and finally to stop the heart, and especially the ventricles, in a state of contraction. Recovery is frequently com-

plete, but sometimes tedious, and in many cases imperfect, ending in serious impairment of health or intellect, indicative of structural changes caused in the nerve-centres. The symptoms of this form of insolation, the real *coup de soleil*, are those of sudden and violent injury to the nerve-centres—unconsciousness, cold skin, feeble pulse, and all the symptoms of depression; death resulting from rapid failure of the respiration and circulation. If not fatal, reaction may result in a variety of conditions indicative of the injury done to the cerebro-spinal system.

(3) Hyperpyrexial form.—**SYNON.** : Heat-fever.—An intense state of fever, the result of the influence of heat on the nerve-centres, and through them on the vaso-motor nerves, and of the heating of the body generally, by the direct action of either artificial or solar heat, may occur, quite independently of the immediate operation of the sun's rays. It comes on as frequently at night, or in the shade, as in the day or in the sunshine, especially in persons who are exhausted by fatigue, overcrowding, depression from any cause, such as dissipation, want of rest, present or recent illness, and notably when the atmosphere is impure from overcrowding or want of cubic space.

The temperature of the body rises to 108° , 110° F., or higher. The brain, medulla, and cord, the nerve-centres generally, and especially the respiratory, suffer from over-stimulation, followed by exhaustion. Respiration and circulation fail; there is dyspnoea, with hurried, gasping breathing; great restlessness; thirst; fever; frequent micturition; and a pungent burning heat of skin, which is sometimes dry, sometimes moist. The pulse varies; in some it is full and laboured, in others quick and jerking. The face, head, and neck are congested to lividity, and the carotid pulsations are visible. The pupils, contracted at first, may dilate widely before death. Delirium with convulsions, frequently epileptiform in character, coma, relaxation of the sphincters, and suppression of urine, come on, and are frequently the precursors of death. Partial recovery not infrequently occurs, to be followed by relapse and death; or secondary consequences result in meningitis or cerebral changes, which may destroy life or intellect at a later period, or permanently compromise the whole health or that of some important function.

The premonitory symptoms of this form of insolation often manifest themselves for some hours, and it may be days, before they culminate in the dangerous condition just described. These premonitory symptoms are general malaise; disordered alvine or other secretions; profuse and frequent micturition; restlessness; sleeplessness, and apprehension of impending evil; hurried and shallow breathing; præcordial anxiety; giddiness and headache; occasionally nausea or vomiting; thirst and anorexia; and feverishness, which soon amounts to a pungent heat of skin with high temperature. These symptoms vary considerably, but they point to a profoundly disturbed state of the cerebro-spinal nerve-centres, and to pathological changes in the organs or structures whose functions have been so gravely disturbed. Recovery is often incomplete; or is followed by permanent impairment of health, and generally by intolerance of heat and exposure to the sun.

TERMINATIONS.—The mortality from sunstroke is about 45 to 50 per cent.; but of those who re-

cover many are permanently injured, and remain invalids for the remainder of life, which is often shortened by the changes induced. There may be some weakness, due to obscure structural change in the cerebrum, or to a chronic form of meningitis which affects the sufferer in various degrees of intensity; or epilepsy, impairment of memory, great nervous irritability, headache, insanity, partial paraplegia, partial or complete blindness, and extreme intolerance of heat—especially of the sun's heat—rendering the person utterly incapable of serving or living in a hot climate, or of enduring exposure to the sun. Or the case may gradually end in complete fatuity, insanity, or meningitis, which accounts for the intense cephalic pain; or, in a lesser degree, in disordered innervation and derangement of the functions generally, thus seriously compromising the general health. See INSANITY IN SPECIAL DISEASES.

Prophylaxis.—The protection of the nervous centres from the direct rays of the sun by a suitable helmet and spinal pad is a matter of prime importance in tropical countries. Light, easy clothing is also necessary. The avoidance of all the conditions which have been indicated as predisposing or auxiliary causes, especially of alcohol and over-fatigue, is also of great consequence. In houses, barracks, and hospitals plenty of space and the freest possible ventilation should be adopted. Ravines and hollows ought to be avoided. Soldiers should be marched in the early morning by short stages, in open order, and frequent halts for rest and refreshment made.

TREATMENT.—(1) In cases of *simple exhaustion* mild treatment is all that is needed. Removal to a cooler locality, the cold douche (but not too much prolonged), or the administration of stimulants may be beneficial. Tight or oppressive clothing should be removed, and the patient treated as in syncope from other causes. See RESUSCITATION.

Rest, and freedom from exposure to over-exertion, fatigue, or great heat, should afterwards be enjoined.

(2) In that form of sunstroke where the person is *struck down* suddenly by a hot sun, the patient should be removed into the shade. Here a douche of cold water must be allowed to fall in a stream on the head and body, from a pump (or as in India from the mussack, or other similar contrivance), the object being twofold—to reduce the temperature of the over-heated centres, and to rouse them into action. During the assault on the White House picket in the second Burmese war, numbers of men were struck down by the direct action of the sun during the month of April. They were laid out perfectly unconscious, in their red coats and stocks (worn in 1852), but were revived by the cold douche freely applied by the mussack over the head and body. In some cases flagellation with a broom was added; and all recovered with the exception of two cases, both of which had been bled on the spot where they fell. Mustard plasters and purgative enemata may also be useful.

If recovery be imperfect, and followed by any indication of injury to the nerve-centres, or by the supervention of meningitis, other treatment may be necessary, according to the indications. Much exposure to the sun should be carefully guarded against; and unless recovery be complete and rapid, the sufferer should be removed to a cooler climate, the most perfect rest and tranquillity of mind and

body enjoined, and the greatest care observed with regard to extreme moderation in the use of stimulants.

(3) In the cases of *thermic fever*, heat being the essential cause of the disease, the object is to reduce the temperature of the body as quickly as possible, and before tissue-changes have resulted. As the hyperpyrexia is due not only to the direct operation of heat, but to fever set up, remedies such as may influence this disturbed condition have been suggested. The results have appeared in some cases to justify the theory, and the hypodermic injection of quinine has been considered to produce good results.

Bleeding has now happily been almost abandoned. The congested livid surface, coma, and stertor, which formerly suggested it, are not now so treated. There are cases in which it may still be practised with advantage; but they are the exception and not the rule. In cases where venesection has appeared at first to give relief and mitigate the symptoms, the improvement has been often transient, and followed by relapse into a more dangerous condition, which has terminated fatally. At the same time no absolute rule can be laid down in this disease with reference to the abstraction of blood; and it is quite possible that greater immediate danger to life may exist in an over-distended right heart than in the loss of an amount of blood which might have tided the patient over that state of peril. Each case must in this respect be treated on its merits. The treatment generally consists in the judicious use of cold, either by affusion or by the application of ice to the surface; the reduction of the temperature being watched with a thermometer in the axilla, mouth, or rectum.

Care should be taken not to continue the cold application too long, as danger arises from depressing the temperature below the normal standard. The bowels should be relieved; and quinine may be given internally or in the form of hypodermic injection.

In the epileptiform convulsions that occur so frequently, the inhalation of chloroform or of ether may be of benefit, but their administration must be carefully watched. The earliest and most severe symptoms having subsided, the febrile condition that follows is treated on ordinary principles—salines and aperients being given, but not to the extent of depressing the patient. The diet must be carefully regulated, and of the blandest and most nourishing nature.

As improvement progresses, other symptoms may supervene, pointing to intracranial mischief. Where they are indicative of meningitis, iodide of potassium and counter-irritants may be used with advantage. Removal to a cooler climate is essential. As a general rule, it is desirable that the sufferer should not, for a long period at least, return to a hot or tropical climate; and he should be guarded against all undue exposure to heat, work, or mental anxiety of any kind.

The sequelæ of sunstroke are frequently from such causes most distressing, rendering the patient a source of suffering to himself and of anxiety to his friends.

The less severe symptoms—those, probably, indicative of the slighter forms of meningitis, or of lesions of the brain or nervous system—occasionally pass away after protracted residence in a cool climate; but they not infrequently also cause much

suffering, and shorten life. As they point to permanently disturbed, if not structurally injured, cerebro-spinal centres, the treatment required is as varied as the symptoms presented.

JOSEPH FAYRER.

KENNETH MACLEOD.

SUPPOSITORY (*suppono*, I place below).—

SYNON.: Fr. *Suppositoire*; Ger. *Stuhlzäpfchen*.—A suppository is a solid mass, which is introduced through the anus into the rectum for certain therapeutic purposes. The material of which it is made should be capable either of being dissolved, or of melting at the temperature to which it is exposed in the rectum.

Suppositories may be divided into *simple, medicated, and nutrient*. Simple suppositories may be exemplified by pieces of soap or tallow-candle, which are in popular use as aperients. The British Pharmacopœia now recognises seven suppositories, one of which may be regarded as simple, the remainder being medicated. The latter are made up with oil of theobroma, in one instance white beeswax also being used. Each suppository is cast into a mould of a conical or other suitable shape, so as to facilitate its introduction into the bowel. It may be well to give a list of these official preparations, with the proportions of their active ingredients, as follows:—

S. Glycerini=70 per cent. by weight of Glycerine with gelatin and water. *S. Belladonnæ*=Alcoholic Extract gr. 1½. *S. Plumbi & Opio*=Acetate of Lead gr. 3, Opium gr. 1. *S. Acidi Carbolic*=Carbolic Acid gr. 1. *S. Iodoformi*=Iodoform gr. 3. *S. Acidi Tannici*=Tannic Acid gr. 3. *S. Morphina*=Hydrochloride of Morphine gr. 4.

Besides these official suppositories, others are often prepared and used of different strengths from those mentioned, or containing other ingredients; and the practitioner may employ many drugs in this way with advantage, according to his own judgment.

Nutrient suppositories usually consist of lean meat that has been finely minced and peptonised.

APPLICATION.—A suppository must be introduced well into the rectum beyond the sphincter ani. At first this should be done by the practitioner, or by a competent nurse; but subsequently most patients learn to use suppositories for themselves without any difficulty. The suppository should be oiled, and passed in gradually and gently with a screwing movement and without any undue force. It may be necessary to keep the finger applied for a moment over the anal aperture, until the tendency to expulsive action on the part of the rectum has subsided.

USES.—A suppository may be used for the following purposes: (1) As a mere aperient, by exciting the expulsive action of the bowel through local irritation, which also has a reflex effect upon the intestine above. (2) On the other hand, to subdue excessive action of the bowel, and thus check diarrhoea. (3) To bring medicinal agents into contact with the rectum in a suitable form, in order to affect some local disease. Astringents and antiseptics are thus used. (4) To influence adjacent organs, the active ingredients of the suppository being absorbed. For instance, a morphine suppository will often produce a marked effect upon the

bladder and generative organs. (5) To produce the general effects of a drug upon the system, particularly when it cannot be given by the mouth. This of course occurs only after its absorption through the mucous membrane, and may be exemplified by the effects of morphine or mercury. (6) To nourish patients when rectal feeding is called for.

FREDERICK T. ROBERTS.

SUPPRESSION OF URINE.—See URINE, Suppression of.

SUPPURATION.—The formation of pus. See ABSCESS; and INFLAMMATION.

SUPRARENAL GLANDS, Diseases of.—

In the article on Addison's disease (p. 23) the well-known association between its characteristic symptoms and morbid changes in the suprarenal glands was described. Addison's disease is much the most important morbid entity connected with these glands, and is essentially a chronic disease; occasionally acute symptoms rapidly develop and prove fatal to a patient known to be the subject of the disease. In some instances similar acute toxic symptoms, collapse, vomiting, and convulsions suddenly occur, and lead to the death of a person previously in fair health and not suspected of any disease, and after death lesions such as hæmorrhage, suppuration, or tubercle are found in the suprarenal glands. These are cases of toxæmia due to acute suprarenal insufficiency, and are extremely difficult to recognise during life.

Morbid changes of various kinds are frequently found after death in the suprarenal glands, but as a rule without any corresponding clinical manifestations. This latency may be due to the lesion being localised, and thus leaving a sufficient amount of suprarenal substance in a state of functional activity, or possibly to compensatory hyperplasia of accessory adrenal bodies. Extreme want of development of the suprarenal glands is sometimes associated with anencephaly, while almost complete atrophy has been found in some instances of Addison's disease. The glands may undergo various forms of degeneration; amyloid change, chiefly in the cortex, is not very rare; cloudy swelling and fatty change are commonly seen in toxic and infective conditions. Gummata and syphilitic fibrosis are very rare, but tuberculosis without any associated signs of Addison's disease is fairly common. Small suppurative foci may occur in pyæmia or infective endocarditis, but primary abscess-formation is almost a pathological curiosity.

Hæmorrhage into the substance of the medulla of the suprarenal glands occurs under a number of conditions, such as traumatism more especially in infants during difficult delivery, in chronic venous engorgement (for example, in asphyxia) and in some septic and toxic conditions, such as purpura. Small hæmorrhages, as a result of embolism in pyæmia, may occur in the cortex, while considerable extravasation may take place into the substance of primary suprarenal tumours.

H. D. ROLLESTON.

SUPRARENAL GLANDS, Tumours of.—

Tumours of the suprarenal glands may be innocent or malignant.

Innocent Tumours.—Adenomata formed of encapsuled masses of suprarenal cells are not uncommon; they usually arise from the cortex, are

multiple and small in size, in a condition of fatty change, and are only discovered after death. Occasionally large adenomata occur; they may be bilateral and tend to degenerate and soften down in the centre into a hæmorrhagic pseudocyst; in rare instances they are palpable during life. Exceptionally other simple growths, fibroma, lymphangioma, angioma, &c., have been met with. Cysts are very infrequent, and when met with are generally due to secondary changes of a degenerative nature in an adenoma. Isolated cases of hydatid and dermoid cysts have been reported. Innocent tumours of the suprarenal glands are of little or no clinical interest.

Malignant Tumours may be primary or secondary. Secondary malignant tumours may be either carcinomatous or sarcomatous. As a rule no special symptoms can be correlated with their presence, but occasionally some of the phenomena of Addison's disease occur. Primary malignant disease is rare. It may be either sarcoma or carcinoma, and in undoubted instances of malignant disease, as proved by the presence of secondary growths, difficulty may arise, from the anomalous microscopic appearances, in settling the precise nature of the neoplasm. It is met with rather more commonly in males, and most often in early middle-age. The growth forms a large tumour with a special tendency to caseate and soften; a hæmorrhagic cavity in the centre may thus result. During life it usually resembles a tumour of the kidney; in fact a correct diagnosis is always difficult and often impossible. The clinical features are not those typical of Addison's disease, but in some instances of exceptional nature overgrowth of hair and pigmentation have been met with. Weakness and debility are marked features, and the disease runs a rapid course. It may imitate not only renal growths but tumours of the liver or pancreas, and retroperitoneal sarcomata. The prognosis is that of malignant disease elsewhere, but is worse from the difficulty of complete removal before metastatic infection has taken place. Removal is of course the only treatment; it has been successful in a few instances.

The accessory suprarenal glands may be the site of new-growth. When accessory suprarenal glands become embedded in the substance of the kidney, a not uncommon event, they are spoken of as suprarenal 'rests.' Primary malignant disease may start in these 'rests' and give rise to malignant disease in, though not strictly speaking of, the kidney. Though of great pathological interest, these cases of malignant disease of suprarenal 'rests' in the kidney are clinically indistinguishable from primary malignant growths of the kidney (p. 836).

H. D. ROLLESTON.

SURGICAL KIDNEY. — See KIDNEY, Consecutive Inflammation of, p. 823.

SUSCEPTIBILITY. — See IMMUNITY.

SWEAT-GLANDS, Diseases of. — See SUDORIPAROUS GLANDS, Diseases of.

SWEDISH EXERCISES. — See MASSAGE.

SYCOSIS. — SYNON. : *Acne Mentagra*; Fr. *Sycose*; Ger. *Barbfinne*.

DEFINITION. — A form of folliculitis confined to the hairy parts of the face.

SYMPTOMS. — Sycosis is a somewhat rare disease. It usually begins on the chin or upper lip,

where it is apt to occupy the central part; it is, however, by no means limited to these regions. A similar form of follicular inflammation may attack the whiskers, eyebrows, and eyelashes. In an early stage the eruption consists of acneiform papules or nodules, which sooner or later develop into pustules with a hair passing through the centre of each. As the number of these increases, the skin assumes a swollen and thickened appearance. In acute cases the infiltration and thickening are considerable, in more chronic forms but slight. In most instances the hairs are not very easily extracted, unless the free supuration extends deep down into the follicle. In cases of long standing the inflammation leads to a complete destruction of the sac, and the formation of scars and permanently bald spots. In all the pustular forms staphylococci are present.

DIAGNOSIS. — In the diagnosis of this disease the following points should be especially remembered: (1) It is chiefly met with in adult males. (2) It generally attacks in the first instance the upper lip or chin, but occasionally the region of the whisker. (3) It is strictly confined to the hairy parts. (4) The papules, nodules, or pustules each have a hair running through them. (5) It is usually a very chronic affection, spreading slowly and lasting for months or years. (6) The inflammation is attended by pain and burning sensations, but by little or no itching. (7) Ultimately, the disease, if not cured, leads to permanently bald patches of scar-like tissue; this, however, is prevented by steady epilation.

Sycosis may be easily confounded with impetigo of the chin, especially when the latter is confined to the hairy parts. Impetigo has, however, a much more rapid development than sycosis, and the discharge and crusts are much more abundant. Moreover, the disease is not usually limited to the hairy parts, as is always the case with sycosis. Another disease for which sycosis may be mistaken is tinea tonsurans of the beard. The chief points of distinction are that tinea generally begins with a circinate patch, and spreads much more rapidly than does sycosis. Subsequently, when supuration is free, the differential diagnosis is more difficult. The hairs, however, in tinea barbæ present some of the characters of tinea tonsurans of the scalp, and their microscopical examination will readily determine the presence of the fungus.

TREATMENT. — There is only one way of curing chronic sycosis with any certainty, and that is by steady epilation. The best plan is first to remove all crusts with oil and fomentations. This softens the skin and renders epilation less painful. The hair of the part affected should be cut rather short with a pair of scissors, and then, wherever a yellow point is seen, the hair passing through its centre should be pulled out with a pair of depilatory forceps. These hairs usually come out with their sheaths attached. When this has been done over a limited area, dilute citrine ointment should be applied. At first, epilation should be confined to extracting those hairs only which pass through pustules; afterwards, however, the parts affected should be completely denuded of hairs by the extraction of a small number every day. This process is attended by considerable pain, and the patience and perseverance of the patient are severely taxed. The young hairs which appear

some time after epilation should be also removed, and the process continued until the skin is healthy; after each removal mild citrine ointment may be applied. Perseverance in this plan of treatment invariably cures the disease; whereas, if left to itself, it leads in the end to the total destruction of the hair and the formation of permanent cicatrices. The process of cure, however, is extremely tedious. ROBERT LIVEING.

SYMPATHETIC (σύν, with; and πάθος, suffering).—This term implies that a part or organ suffers 'in sympathy' with some other part or organ which is diseased. Most disorders which are popularly supposed to arise in this way admit of an obvious pathological explanation. Thus, a morbid process may extend directly along blood-vessels, lymphatics, or other tissues; a morbid agent may be conveyed by the blood or lymph from one part to another; or a secondary lesion may be produced by direct nervous influence. The 'sympathetic' disturbance may be indicated by pain or other subjective sensations; by functional derangements, as of secretions or actions; or by positive organic lesions. The occurrence of such phenomena in corresponding parts on both sides of the body, when a disease has commenced on one side, is sometimes very curious, especially as regards organic lesions. As illustrative of the associations in which the word 'sympathetic' is employed may be mentioned *sympathetic pain*, *sympathetic headache*, *sympathetic ophthalmitis*, *sympathetic vomiting*, *sympathetic bubo*.

FREDERICK T. ROBERTS.

SYMPATHETIC SYSTEM, Disorders of.—

SYNON.: Fr. *Maladies du Nerf Sympathique*; Ger. *Krankheiten der Nervus Sympathicus*.

1. Diseases of the Sympathetic System in connection with the Cerebro-spinal System.

(a) *The Spinal Cord*.—Lesions of the cervical region of the spinal cord may be associated with extreme contraction or extreme dilatation of the pupil on one or both sides; with increased heat and redness, or the reverse, of the head and neck; with perverted respiration; with perverted action of the heart; and possibly with an exalted febrile heat of the whole body (see SPINAL CORD, Diseases of, pp. 1526, 1527). Though we regard these phenomena as signs of disease in this particular portion of the spinal cord, it is none the less true that such phenomena are due to altered activities in those root-portions of the sympathetic system of nerves which take origin in, or traverse, this region of the cord. This is shown by the fact that similar sets of symptoms are produced by injuries, tumours, or other morbid processes implicating the cervical sympathetic itself.

It will be well to cite here the phenomena commonly associated with *irritation* or *paralysis* of the cervical sympathetic nerve, on account of their importance as diagnostic indications.

The signs dependent upon *irritation* of the cervical sympathetic in its *oculo-pupillary* fibres are—dilatation of the corresponding pupil with sluggish action, widening of the palpebral fissure, prominence of the eyeball, feeling of tension in the eye (as in glaucoma), and a scanty secretion of tears and mucus; while in its *vaso-motor* fibres they are

—lowering of temperature of the side of the face and head, diminution of sensibility, an absence of perspiration, with (if the irritation continue) a tendency to slight atrophy of the side of the face. The signs of *paralysis* of the cervical sympathetic in its two sets of fibres are the direct opposites of those just cited, so that it is not necessary to enumerate them. Of these signs, those dependent upon irritation or paralysis of the oculo-pupillary fibres are usually much more constant and durable than those which depend upon irritation or paralysis of the vaso-motor fibres. These latter signs are, for reasons at present unknown, often transitory and fitful. Sometimes there may be signs of paralysis of oculo-pupillary fibres co-existing with signs of irritation of the vaso-motor fibres, or *vice versa*. It has been definitely determined that injury in the lower cervical region of the cord, and as far down as the level of the *second dorsal* nerve, may give rise to the oculo-pupillary signs of one or other kind; and, on the other hand, that damage to the cord in these same parts, or as low down as the *fourth dorsal* nerve, may give rise to the above-mentioned vaso-motor signs.

When the *dorsal* and *lumbar* regions of the spinal cord are the seats of disease, other groups of phenomena will doubtless, after a time, be more fully recognised as results of irritation or paralysis of those roots of the sympathetic system which have their origin in or which traverse these particular regions of the spinal cord. It is therefore important to bear in mind the place of origin and the distribution of the different internal branches from the lateral sympathetic cords, which proceed from these regions to the different glandular organs or hollow viscera. Diarrhœa, sickness, obstinate constipation, sexual defects, and bladder-troubles, are among the symptoms which have such an origin, as well as undue heat or unnatural coldness of the lower extremities. See also PAIN IN VISCERAL DISEASE, p. 1137.

(b) *The Brain*.—In different portions of the brain some of the signs and symptoms of disease are also referable to direct or indirect interference with the functions of the sympathetic system of nerves; but they constitute (apart from vaso-motor derangements, which are very common and often well-marked) far less distinctive aggregates, owing to the fact that the sympathetic system of nerves has a much less extensive relation with the brain than with the spinal cord. In this direction, however, and in connection especially with diseases of the medulla oblongata, we have to bear in mind the occasional occurrence of diabetes, polyuria, or albuminuria; also of some cardiac and respiratory derangements.

2. *Diseases of the Sympathetic System proper*.—Where disease exists in the ganglia of the sympathetic system itself, or where it involves them, we get groups of symptoms more clearly referable to disordered activity of this system of nerves alone.

These will differ in particular cases, according to the nature of the morbid change, that is, according as it is destructive or merely irritative; and according to the number or particular combinations of ganglia and fibres affected. The ganglia and related plexuses may either be affected by *intrinsic* morbid processes, or may be variously involved from without by morbid processes having their origin in adjacent tissues.

(a) *Intrinsic changes*.—The principal intrinsic morbid processes which have been hitherto recognised *post mortem* in some one or other of the sympathetic ganglia are: Pigmentary degeneration; irritable overgrowth of their connective tissues, with or without secondary atrophy (the ganglia in such cases being either smaller or larger than natural); a highly congested and varicose state of their blood-vessels; effusion of blood into their substance; new-growths starting from their substance; and fatty degeneration, with more or less marked atrophy.

(b) *Extrinsic disease*.—Different parts of the sympathetic system may become involved in new-growths or in abscesses; or they may be simply pressed upon by aneurysmal or other tumours occurring in contiguous regions of the body.

Besides the pathological conditions already enumerated, it should be borne in mind that in altered blood-states, whether cachectic or of febrile origin, we commonly have, and especially in the latter class of cases, a greatly perverted activity of the sympathetic system throughout the body—as evidenced by the altered vascular conditions, increased tissue-metamorphosis and body-heat, together with the perverted activity of most of the glands in the body. *See* FEVER.

The principal disorders apart from those due to structural diseases of the cord and of the brain, in which derangements of the sympathetic system of nerves exist, or are believed to exist, and in which such derangements have either wholly or in part a causal relationship to the principal signs and symptoms of the respective disorders, are as follows: Epilepsy; convulsions; megrim (hemicrania); ophthalmic goitre; unilateral hyperidrosis; progressive facial hemiatrophy; angina pectoris; asthma; diabetes mellitus; Addison's disease; astralgia; enteralgia (colic); neuralgia cœliaca; neuralgia spermatica; and uterine neuralgia. Among the affections more doubtfully or partially related to disorders of the sympathetic, we may mention glaucoma; neuro-retinitis; progressive muscular atrophy; pseudo-hypertrophic paralysis; locomotor ataxy; diphtheritic paralysis; and so-called 'reflex paralysis.' In the special articles on most of the first group of affections, the reader will find references to the dependence of such conditions upon disorders in one or other department of the sympathetic system.

H. CHARLTON BASTIAN.

SYNCOPE (συνκοπή, a faint).—*SYNON.*: Fainting; *Fr.* *Syncope*; *Ger.* *Ohnmacht*.

DEFINITION.—A state of suspended animation, due to sudden failure of the action of the heart.

ÆTIOLOGY.—Syncope may be due to any condition which interferes with the action of the heart, whether acting (a) *intrinsically*; (b) through the *nervous system*; (c) through the *blood*; (d) through *gravity*; (e) through *more than one* of these channels.

(a) Syncope due to *intrinsic* cardiac conditions is chiefly seen in structural diseases of the heart, especially fatty degeneration. Among other instances of this class of causes may be mentioned compression of the heart by morbid growths, products or processes, or by tight articles of dress; excessive heat, whether natural or artificial, as in sunstroke and the hot bath; lightning; and certain drugs and poisons, including alcohol, chloroform, tobacco and ptomaines.

(b) The most common *nervous* causes of fainting are of an emotional kind, such as fear, grief or joy, in nervous or hysterical individuals. Sudden injury of the central nervous system, as in concussion of the brain, has partly the same effect. In a larger number of instances the nervous causes of syncope act reflexly, and are to be found in conditions of the stomach or intestines (corrosive and irritant poisoning, heavy meals, indigestion, worms, scybala); in the liver, kidneys, or uterus (injuries, calculi, displacements); or in the limbs or body generally (painful injuries or operations of any kind). Spasm of the arteries, due to reflex irritation of the vaso-motor nerves (cold and certain poisons), also may lead to syncope.

(c) Of the causes of syncope connected with the *blood* the most frequent is hæmorrhage. Chronic anæmia, as seen in idiopathic and pernicious cases, or accompanying chronic constitutional diseases, is a common cause of serious fainting.

(d) Syncope produced by disturbances of the force of *gravity* in the circulation is familiar in the faintness which may occur when patients first assume the erect posture in convalescence.

(e) In a large number of instances, the causes of syncope are *complex*. Thus in fainting from hunger and exhaustion the heart is depressed directly, as well as through the nervous system, and through the blood; and in severe injuries, such as railway accidents, there may be a combination of depressing causes, including fear and grief, hæmorrhage, painful lesions, cerebral concussion and shock. Fainting in a hot impure atmosphere appears to be due partly to the direct effect of heat upon the circulation; partly to the interference with respiration, and indirectly with the heart, produced by carbonic acid and other excrementitious products.

In the subject of any of the predisposing causes of syncope already mentioned, the occurrence of fainting may be determined by a very slight exciting circumstance. It is thus that in serious cardiac disease, in hysterical subjects, in delicate overgrown adolescents, and in persons suffering from anæmia, the smallest excitement or exertion, unpleasant sights or smells, or exposure to an impure and heated atmosphere, may cause faintness, and in some instances even fatal syncope.

ANATOMICAL CHARACTERS.—In death by syncope the organs generally are found to be anæmic; and this condition is particularly marked if hæmorrhage have occurred. The state of the heart varies with the cause of its failure, the ventricles being either dilated and full of blood, or empty, as in cases of fatal hæmorrhage, and possibly contracted.

SYMPTOMS.—A syncopal attack presents three stages, namely: (1) *a period preceding loss of consciousness*; (2) *a condition characterised by insensibility*; (3) *a period of recovery* from the fainting state.

(1) A person about to faint is observed to turn suddenly pale; he staggers, or leans against the nearest support; the eyes roll upwards, while the eyelids tremble or close; and consciousness and general sensibility are impaired. The pulse fails, generally becoming weak, small, and frequent; in other instances it is infrequent, irregular, or intermittent. The respiration is irregular and feeble. Vomiting may possibly occur.

At the same time the patient has a number of subjective sensations. The most urgent of these are a sense of 'sinking' in the epigastrium, a feeling

of increasing debility, 'giddiness' in the head, and a tendency to fall. Vision becomes indistinct; the hearing is usually impaired or 'distant'—rarely more acute; and tinnitus may be present. Mentally there is a rapid fading of sensory impressions and of consciousness; while in cases of fainting from loss of blood there may be restlessness, agitation and delirium.

(2) The first stage is now complete. Thereupon the muscles are relaxed; the patient falls; and consciousness is completely lost. The surface is pallid, and possibly cold and clammy; the eyes are closed, and the pupils dilated; the pulse and the cardiac impulse and sounds are nearly or quite imperceptible; respiration is indistinguishable, or occurs as occasional weak sighs; and the vital functions generally appear to have ceased. In syncope due to severe hæmorrhage general convulsions may occur.

(3) The third stage, that of recovery from syncope, is marked by signs of gradually returning consciousness, increase of the pulse at the wrist, and restoration of the functions generally. Usually the first evidences of improvement are slight movements of the hands and features, and deep sighing. The pulse now becomes more distinct; the cardiac impulse and sounds are found to be stronger; the senses of sight and hearing can be excited; colour returns to the face and lips, and warmth to the extremities; and intelligence is gradually restored. Very shortly the patient may be able to resume the sitting posture; and the seizure is at an end.

DURATION AND TERMINATIONS.—The duration of the several stages of syncope varies greatly, from a few seconds even to hours. In many instances the attack does not pass beyond the first stage; in rarer cases insensibility may last for an almost indefinite time. The most common termination is in recovery; but syncope is one of the ordinary modes of death, especially in hæmorrhage and structural disease of the heart. In nervous subjects partial recovery may be quickly followed by return of the fainting state, the patient being said to 'pass out of one faint into another.' Where referable to structural disease or to hysteria, syncope may recur at intervals for many years.

PATHOLOGY.—Syncope consists essentially in sudden failure of the action of the heart, originating in any of the causes already mentioned, and leading to a condition of acute general anæmia. Whether from some affection of the heart itself, from sudden interference with the nervous impulses which regulate its action, from failure in the supply of blood within its cavities and in its substance, or from a combination of such causes, the systolic contraction suddenly becomes short and feeble. If there has been no hæmorrhage nor vaso-motor paralysis, the cardiac cavities are distended with blood, and further embarrassed; if profuse hæmorrhage has occurred, the heart is deprived of blood, and thus of the natural stimulus to contraction, and the same effect will follow failure of compensation for the hydrostatic effect of gravity by vaso-motor activity. In either case fatal cardiac paralysis may be the result, unless the contractile power of the heart be speedily restored.

The acute general anæmia that results specially affects the central nervous system. In the erect posture the circulation fails first within the cerebrum, producing rapid disturbance and then loss of

consciousness, and depressing the centres that regulate the heart, vessels, respiration and stomach. The general muscular paralysis which occurs at the same time is also partly of central origin. Similarly, the convulsions which may ensue in cases of hæmorrhage are probably referable to sudden circulatory disturbance within the basal ganglia and cord. The senses are further obscured by anæmia of their special organs; the heart is more depressed by failure of the coronary circulation; the paralysis of the muscles is increased by want of blood within them; and the temperature falls from failure of the circulation generally.

In non-fatal cases recovery naturally occurs by restoration of the cerebral circulation in the recumbent position, and consequent stimulation of the cardiac centre. Other circumstances favour the recovery of the general circulation, such as the relaxation of the arteries, and the partial restoration of the respiratory and other functions, which quickly re-act upon the heart.

DIAGNOSIS.—Syncope has to be diagnosed from other conditions or events in which loss of consciousness is a prominent symptom; and chiefly from epilepsy, apoplexy from any cause, vertigo, concussion of the brain, shock, and poisoning of many kinds, including suffocation by certain gases and drunkenness. From such of these conditions as commence in the brain, and from poisoning (unless the poisons act as cardiac depressants) syncope is distinguished by the characters of the pulse. Certain cases of hysterical faints, which are strictly cerebral, not cardiac, in origin, are also readily diagnosed by the pulse, which may be of good volume and force. The diagnosis of shock, which usually produces a degree of syncope, is described in the article on that subject. *See SHOCK.*

PROGNOSIS.—The prognosis of syncope depends upon its cause, and upon the practicability of immediate treatment. If due to structural disease of the organs of circulation, or to serious injury, acute poisoning, excessive heat, or profuse hæmorrhage, the case may be serious, and prove fatal unless treatment be instantly applied. If, on the other hand, the cause of the faintness lie in an excitable nervous system, momentarily depressed by some passing emotional disturbance, by impurity of the atmosphere, or by digestive derangement, the attack may be pronounced free from danger, although liable to recur.

TREATMENT.—In the treatment of syncope two indications are equally urgent, namely, removal of the cause of faintness, and restoration of the action of the heart. If the patient should not have fallen, he must be immediately laid flat on his back; the atmosphere should be rendered as pure as possible, by throwing open the windows and doors, or by removing the patient to the open air, and preventing people from crowding round him; at the same time the dress should be loosened about the neck, chest, and abdomen. If hæmorrhage have occurred, means must be taken to stop it. Cardiac stimulants, direct or indirect, must then be employed. The most available and powerful of these is alcohol, in the form of brandy or other spirit; and this may be given either pure or in water, and in an amount which will vary with the individual case, as estimated by the immediate result. *Sal volatile*, ether and *eau-de-Cologne* are equally valuable cardiac stimulants, if available. Should the patient be

unable to swallow, these substances, as well as warm liquids, must be given at once as enemata; or ether may be injected under the skin. Ammonium carbonate ('smelling salts') and other strong smelling compounds, including perfumes, fanning, cold douches, and refrigerant applications of eau-de-Cologne or other spirit to the temples and hands, are other ready means of exciting the heart reflexly through the nervous centres. If these measures fail after a fair trial, the condition of the patient is very serious. The systematic employment of efficient means of resuscitation must then be had recourse to, including friction of the limbs and trunk, inversion, galvanisation of the region of the heart, and even transfusion. See RESUSCITATION (A); and ANÆSTHETICS.

In cases ending favourably the patient must be careful not to assume the erect position too hastily, or to undergo much exertion, until some rest has been obtained, or some stimulant or nourishment administered.

The occurrence of syncope is sometimes the first indication of the existence of serious structural disease of the heart or other organ; and it should suggest a careful examination of the patient, and the adoption of measures likely to prevent the return of such a dangerous event, that is, the avoidance of the principal causes already mentioned, as far as they are avoidable.

J. MITCHELL BRUCE.

SYNOVIAL MEMBRANES, Diseases of.—
See JOINTS, Diseases of.

SYPHILIS (etymology uncertain. Perhaps from 'Syphilus,' the title of a Latin poem by Fracastorius, published in 1530; or from *σῦς*, a swine; and *φιλέω*, I love).—SYNON.: Vulg., Pox; Fr. *Vérole*; Ger. *Lustseuche*.

DEFINITION.—An infective general disease; communicable by contact of a specific virus with a breach of surface, or by hereditary transmission. Syphilis is characterised by a period of incubation; and, in the acquired form, by certain changes at the seat of inoculation, and in the proximate lymphatic glands. These are followed by an eruption on the skin and mucous membrane, and sometimes by lesions of the deeper tissues and viscera.

ÆTIOLOGY.—The various phenomena of syphilis are caused by the absorption of an infective virus into the blood, and its diffusion throughout the body. It is now generally agreed that the 'soft chancre' is a local affection, quite distinct from syphilis. From this point of view the present account is written. The local suppurating sore or soft chancre is described elsewhere. See VENEREAL SORE.

One attack of syphilis usually affords protection against a second throughout the lifetime of the individual, but in rare instances, as in other infectious diseases, the same person suffers more than once.

Besides being capable of contaminating others by inoculation, the subject of acquired syphilis is also liable, during a variable period, to transmit the disease to the offspring; but whether the inherited form of syphilis be further transmissible to the next generation remains doubtful.

PATHOLOGY AND ANATOMICAL CHARACTERS.—The essential nature of the syphilitic virus is unknown. Various micro-organisms have been reported from time to time as found exclusively in the

blood or tissues of syphilitic persons; but evidence is still wanting to settle the question, one difficulty in the way being that syphilis seems not to be communicable to the lower animals. Syphilis resembles the exanthemata in having a period of incubation, in the development of a rash, and in the protection commonly afforded by one attack against subsequent infection; but it differs from them in its long duration and liability to relapse, and in not being communicable through the atmosphere.

When the syphilitic poison has been absorbed, it multiplies until the whole body becomes pervaded by it. How soon this absorption takes place is not known. Excision of the initial lesion has been extensively practised with the object of preventing further development of the disease; but the evidence at present available, from observation as well as from experiment, indicates that by the time the initial lesion appears the virus has already passed far beyond its boundaries.

The changes produced by syphilis have been well described by Gowers as depending partly on a process of inflammation, and partly on a process of tissue-formation. These two are generally combined in varying degree according to the lesion and according to the stage of the disease. The inflammatory element is most marked in the early eruptions of the skin and mucous membrane, and is also probably the chief factor in causing certain early functional disorders of the internal organs. The process of new growth is most distinct in what is called the *syphiloma* or *gumma*. The change begins by the production of a small-celled growth, which at first resembles granulation-tissue—hence named by Virchow *granuloma*—but which soon shows a marked tendency to vascular occlusion and consequent degeneration.

The new-growth may develop in a diffused infiltrating form, or in the circumscribed masses known as *gummata*. Any of the structures of the body may be attacked, most commonly, perhaps, the skin and subcutaneous connective tissue; but bone, muscle, glands, and the viscera are all liable to suffer, the change probably beginning in the blood-vessels of the affected part; and although the morbid growth is essentially the same wherever it is developed, it presents differences in appearance, as well as in behaviour, according to the tissue or organ affected, and the period at which it occurs.

A *gumma*, which is the most characteristic product of syphilis, in its typical form appears as a yellowish, tough, somewhat elastic, and sharply defined mass, which is often caseous in the middle. *Gummata* vary greatly, from a mere granule to the size of a duck's egg, or even larger still. They may be single or multiple, and are frequently associated with the diffused form of growth, which after a time becomes converted into a tough fibrous tissue; this finally contracts, and thus puckers, deforms, and often seriously affects the functions of the organ in which it grows.

Besides the specific effects of syphilis there are certain degenerative diseases of the nervous system, notably *tabes dorsalis* and *general paralysis*, which are thought to be chiefly caused by syphilis, as there is evidence of that disease in over 70 per cent. of such cases.

A syphilitic origin has also been claimed by certain authors for a variety of maladies connected with malnutrition—rickets, for example—when they occur in the children or even grandchildren of syphilitic

parents. There is considerable difference of opinion concerning these so-called 'para-syphilitic' affections, but all are agreed that *amyloid disease* may be caused by syphilis, especially, but not exclusively, in cases where long-standing suppuration has been present. See AMYLOID DISEASE.

INFECTION.—Before considering the different ways in which syphilis may be propagated, it is necessary to mention the vehicles of the virus. These are—

1. The discharge of the initial lesion.
2. The secretions of all the secondary eruptive lesions, especially of those known as mucous patches or tubercles.
3. The blood, during the earlier stages of the disease.

The secretions of the later or tertiary affections have not been proved to be inoculable, nor have the physiological secretions of a syphilitic person—for example, the saliva, sweat, tears, milk—unless mixed with the secretions of syphilitic lesions or with the blood, even though the disease be in an early stage. The semen, however, though apparently not inoculable in the ordinary way, seems to be capable of infecting the ovum during a period which probably varies considerably in different cases. Lastly, the secretions of other diseases from which a syphilitic person may be suffering are not always infectious. This at least is the case with regard to vaccinia; for healthy children have often been vaccinated from syphilitic subjects without contracting syphilis.

Modes of Communication.—The sources of infection being so numerous, it is easy to understand that the modes of communication must be so also. They may be described under three heads: (1) *direct communication*; (2) *mediate communication*; and (3) *hereditary transmission*.

1. *Direct communication.*—In the great majority of cases syphilis is imparted during sexual intercourse—first, because the genital organs are the most frequent seat of the infectious lesions; secondly, because the delicate epithelium of these organs is especially liable to abrasion during coitus. Hence syphilis is usually described as a venereal disease, but it should always be remembered that it is not necessarily so. Wherever the poison comes in contact with a broken surface, it may thence be absorbed, and general infection follow. Instances of syphilis being conveyed quite independently of sexual relations are unfortunately far from rare. Perhaps the most frequent form of extra-genital infection is that of a nurse by a syphilitic child, or of a child by its nurse. The disease may also be spread by kissing, syphilitic lesions being very common about the lips and in the mouth. Again, medical men and midwives not infrequently contract syphilis by attending diseased women in labour; and surgeons by examining or operating upon syphilitic persons.

2. *Mediate communication.*—When syphilis is communicated indirectly, the medium may be of almost endless variety. Articles which are used in common by different persons, such as spoons, drinking-vessels, and pipes, are perhaps the commonest media; implements used in various trades—shaving and glass-blowing, for example—have also acted in the same way. Again, through the performance of tattooing, cupping, catheterisation of the Eustachian tube, and other operations, chiefly by ignorant persons and quacks, the disease has been communicated; and it has also happened during vaccination; but now that calf-lymph only is used there should be

no danger whatever. The mode of communication in which the foetus is believed to be the medium between husband and wife is noticed under the next heading.

3. *Hereditary transmission.*—Our knowledge concerning the transmission of syphilis from parent to child is still imperfect in many respects, and the questions involved are much too wide for discussion in this article. Consequently, no attempt will be made to do more than state briefly the points which are generally accepted, and the usual course of events when syphilitic persons become parents.

In the first place, it must be remembered that a child born of syphilitic parents does not always show signs of the disease. When the infection of both parents is recent, however, the offspring almost invariably suffers, and this is also the case if the mother alone be diseased. In such circumstances abortion or premature birth is common. When the father alone is syphilitic, and especially if his disease be not of recent date, both mother and child may escape.

Syphilitic infection of the offspring may be either germinal, from one or both of the parents, or in the case of the mother it may be post-conceptual, that is, through the placental circulation.

When a previously healthy woman bears a syphilitic child to a syphilitic father, and shows no sign of infection herself, she is supposed to have gained immunity from her foetus *in utero*. The most weighty evidence in support of the view that she is really immune is that known as Colles's Law, namely, that the mother of a syphilitic child, although she may suckle it, does not acquire syphilis from it after its birth.

But if in similar circumstances the mother suffers afterwards from some of the later signs of syphilis without having previously shown any of the earlier signs of acquired syphilis, she is supposed to have been infected by her foetus *in utero* (syphilis by conception, or *choc en retour*).

Again, if, as more rarely happens, a syphilitic woman bears a child which shows no sign of infection, the child is supposed to have gained immunity through the placental circulation. But neither in the case of the mother nor of the child is it known how long the immunity lasts.

On the supposition that syphilis is due to a microbe, these phenomena have been explained as follows: Under normal conditions, as is well known, there is no direct communication between the blood-vessels of the placenta and those of the foetus, and if those conditions remain unaltered in spite of the syphilitic infection of mother or foetus, then whichever of the two is healthy will by diffusion of the toxins gain immunity from the other. On the other hand, if the continuity of the walls of the blood-vessels be broken—which is probably most frequently the case in syphilis—then the micro-organisms themselves will pass, and mother or child, as the case may be, will be infected by the other.

If the mother acquire syphilis during the earlier months of pregnancy the child commonly suffers. If she be infected after the seventh month it is said that the child may escape, but further and more exact evidence on this point is required.

The duration of the transmissive power varies in different cases, according to the influence of treatment and other circumstances; but the liability to transmit syphilis to the offspring seems to last longer than the ability to infect by inoculation. In the

absence of treatment the child or children born nearest to the date of parental infection usually suffer more severely than those born later, which gradually suffer less and less severely as time goes on; but this is not always the case. A healthy child may be born under the influence of mercury; but a subsequent one may be tainted, if treatment have been discontinued, and the disease have resumed activity. There are also periods of quiescence in syphilis independent of treatment, during which apparently healthy children are sometimes born.

SYMPTOMS.—The symptoms of syphilis are usually divided into three groups—*primary, secondary, and tertiary*. And although such division is really artificial, it is useful to retain these terms for the purpose of description; but it must be borne in mind that in the great majority of cases tertiary symptoms do not occur at all; that they sometimes appear quite early in the disease; and that the signs proper to all three periods may be present at the same time.

1. Primary Syphilis.—A patient is said to be suffering from primary syphilis as long as the initial manifestation, and the accompanying glandular enlargement, remain the sole signs of the disease.

When the syphilitic poison, unminged with any irritating matter, has been inoculated, the abrasion quickly heals, and no further change is observed for three or four weeks. This interval is called the period of *incubation*. Its average length, judging from cases of experimental inoculation, is about twenty-four days; but it may perhaps be as short as ten, or as long as forty-six days, or even longer still. After this a small red spot appears at the site of inoculation, which is called the *initial or primary lesion*. Later, this may assume one of several forms, and is then often termed a hard or infecting chancre, and sometimes, incorrectly, a Hunterian chancre. The initial lesion presents certain differences in appearance, according to its position, and the presence or absence of irritation. Its chief characteristic is the presence of induration at its base; and the aspect of the sore is much affected by the degree to which this hardness is developed. Sometimes the lesion is observed as a hard desquamating papule; sometimes as a prominent ulcer, having a hard well-defined base and thickened adherent margins; but most commonly as an erosion or shallow ulcer, with an amount of induration which varies much in different cases, and which is liable to be overlooked unless care be taken. Sometimes the induration develops in a thin layer, like a piece of parchment, or even paper; hence the term ‘*parchment sore*’ often applied to this form of the initial lesion. In rare instances, especially in women, induration appears to be absent. The secretion is thin, scanty, non-purulent; and though not readily inoculable on the bearer, it has nevertheless been successfully inoculated in certain cases during the primary stage. The syphilitic primary lesion is usually *single*, contrasting strongly in this respect with the local venereal sore, in which multiplicity is the rule. It is also indolent, often painless, tends to disappear spontaneously, and does not necessarily leave a scar.

The *seat* of the initial lesion is most frequently the genital organs, but, as has already been stated, absorption of the poison may occur in any situation where a breach of surface exists. Consequently the initial lesion may be found on any part of the body, as about the lips or mouth in children, on the breast

in nurses, or on the hand or finger in the case of doctors and midwives.

If, as frequently happens in practice, irritating matter of any kind have been inoculated as well as the syphilitic poison, the course of events will vary according to the nature of the irritant. For example, if the pus of the local chancre have also been absorbed, the incubation-period of syphilis will be occupied by the course of the suppurating sore, which may or may not have healed at the time the change peculiar to syphilis occurs. If the sore be still present, induration will develop, and the lesion will for a time assume the characters of both kinds of sore; but if it have healed, the cicatrix will harden and eventually assume more or less closely the appearance of one or other of the forms of initial lesion which have just been mentioned.

The initial lesion in rare instances may disappear in a few days, but its duration usually varies from two or three weeks to several months, according to its size and the influence of treatment. It sometimes breaks out again after cicatrization; and induration may reappear, even repeatedly, without fresh infection. If the sore, from irritation of any kind, be made to suppurate, the secretion may become freely inoculable on the bearer. When the chancrous and syphilitic poisons have both been inoculated, the resulting lesion has been termed by Rollet a ‘*mixed chancre*.’

Glandular Enlargement.—On whatever part of the body the initial lesion may be situated, the nearest lymphatic gland or glands become perceptibly enlarged in from seven to fourteen days after its appearance. Where the glands themselves are multiple, the whole group is usually more or less affected; but the gland most directly connected with the point of inoculation enlarges first, and often attains a greater size than the others. In the absence of inflammation of the surrounding parts, each gland can be felt as a separate distinct indolent swelling, usually about the size of a marble, the cellular tissue and skin remaining free. At this time the enlargement commonly remains limited to the nearest group, but later it is sometimes general. When the genital region is the seat of the initial lesion the glands of both groins are in most cases enlarged, though often to an unequal extent. Suppuration of the glands probably never occurs unless there be some source of irritation in addition to the syphilitic poison.

Besides the glands, the *lymphatic vessels* leading to them—those of the penis, for example—can sometimes be felt as hard cords, freely movable beneath the skin. These cords have also been found to contain thickened blood-vessels.

2. Secondary Syphilis.—After the development of the initial manifestation, with its accompanying adenopathy, another interval occurs before further signs appear. This is sometimes called the *second incubation*, to distinguish it from that which intervenes between infection and the appearance of the initial lesion. This second period of quiescence, counting from the appearance of the initial lesion to the appearance of the general eruption, is usually about six weeks. Thus, as a general rule, it may be stated that, in the absence of specific treatment, the rash appears from sixty to seventy days after infection; from forty to fifty after the initial lesion; and from thirty to forty after enlargement of the nearest lymphatic glands.

During the earlier part of this period the patient

commonly does not feel ill; but towards its close, and shortly before the appearance of the eruption, some patients, especially women, if they be not already under the influence of mercury, develop certain symptoms of constitutional derangement which have received the name of *prodromata*. Thus the patient may become pale and anæmic, and may suffer from shortness of breath and lassitude. In addition, headache, loss of appetite, pains in the limbs and back, rise of temperature, and other symptoms known under the name of 'syphilitic fever,' may be present. During this period also, as was first pointed out by Ricord and Grassi, the proportion of red blood-corpuscles is diminished. The prodromata are usually mild in degree, but occasionally they are severe. Thus, headache may be agonising, and in rare instances the amount of constitutional disturbance is so great that the onset of one of the acute fevers may be suspected.

Cutaneous System.—At the end of this second interval of apparent quiescence, then, and having been preceded or not by some of the symptoms just mentioned, the first eruption appears. This, in the great majority of cases, consists of irregularly scattered red spots (*Erythematous Syphilide*, *Syphilitic Roseola*) varying in size from a hemp-seed to a shilling, not raised above the surface, fading on pressure at first and often throughout. The rash is unattended by itching or any other subjective sign, so that it is often overlooked by the patient. The spots vary in colour, being often at first of a paler or deeper red according to the complexion of the bearer; but if they last long they usually become dull red or brownish, and then may not fade on pressure. Finally they disappear completely, and occasionally slight desquamation follows. The spots usually appear first about the flanks and abdomen. The face and hands generally escape. The extent and duration of the eruption vary much in different persons. It may be limited to a few faint spots on the trunk, or the whole body may be covered with the rash, in which case it often resembles that of measles. The erythematous syphilide may appear suddenly and disappear quickly; or it may last for several weeks, and indeed occasionally for months, if untreated. It sometimes recurs, but in that case is usually, though not invariably, limited to a few blotches or rings on the trunk or limbs.

As the macular eruption fades, papules not uncommonly appear, or some of the spots may be papular from the first, so that a maculo-papular eruption is often seen in early syphilis. The papules are bright red at first, but later often assume a colour resembling that of raw ham, and later still a 'coppery' hue. The papules are very liable to relapse, and to become scaly. When they fade, a brown stain is often left, which gradually disappears completely. At this time also crusts are frequently present on the scalp among the hair, which itself becomes dry and withered, and at a later period often falls out, either generally or in patches. Syphilitic alopecia is in most cases limited to a temporary general thinning of the hair of the head, but all the hairy regions of the body may suffer. The nails also are liable to be attacked in several ways. See NAILS, Diseases of.

Though the macular and papular syphilides are by far the most frequent forms of early rash, vesicular, pustular, rupial, and ulcerating eruptions sometimes appear during the secondary stage, the last two being most common in patients of bad constitution.

The earlier eruptions are usually superficial, and widely spread. Except perhaps in the case of the earliest rash, the papule forms the basis of the spots. Irritation and itching are usually absent. The eruption has certain favourite seats, namely, the anterior and lateral surface of the trunk, the flexor surface of the limbs, the border of the hairy scalp, and the neighbourhood of the various orifices of the body. Several forms of eruption are often present at the same time; most commonly maculæ and papules, but sometimes papules and pustules. They all tend to disappear spontaneously, and, except the pustular form, without leaving any scar.

Pigmentary changes in the skin are common in syphilis. Temporary excess of pigment may follow any syphilide, and this again may be followed by its loss, so that sometimes yellow or brown stains are seen, and sometimes white-spots. A much rarer change, which differs from those just mentioned in that it does not follow another lesion on the same site, is that known as the *pigmentary syphilide*. It consists of irregular brownish patches situated chiefly about the sides of the neck, and observed mostly in women. Opinions are divided as to whether this is an essentially syphilitic affection or not. It lasts a long time, and is but little affected by antisymphilitic remedies.

Mucous Membrane.—Besides the affections of the skin, lesions of the mucous membrane, particularly that of the mouth and throat, are nearly always present during the secondary stage of syphilis. Erythema, excoriations, or shallow ulcers are very common about the fauces and tonsils, as well as on the buccal surface and tongue, during the early portion of this stage; and the nasal and laryngeal mucous membrane is also liable to be affected. Equally common, though usually somewhat later, are mucous patches or tubercles (see MUCOUS PATCH). They may occur on any moist surface, but are most frequently seen about the mouth, genital organs, and anus. The initial lesion also sometimes assumes the appearance of a mucous patch or tubercle, especially in women.

These affections of the skin and mucous surfaces are often accompanied by more or less deterioration of the general health, with pallor, lassitude, and loss of weight. Aching in the muscles, bones, or joints (*osteocopic pains*), and sometimes periosteal swellings or synovial effusions, also occur, as well as more or less general enlargement of remote lymphatic glands, particularly those of the neck and beneath the jaw; but the axillary, epitrochlear, and indeed all the glands within reach, may be enlarged. Iritis is also liable to come on about this time, and, later, choroiditis. Sometimes, but more rarely, affections of the auditory apparatus are present. See EYE; and EAR.

Enlargement of the *epididymis*, chiefly its head, is another, though rare, phenomenon in secondary syphilis. Temporary albuminuria and jaundice also sometimes occur during this stage.

The number, extent, and severity of the secondary manifestations vary greatly in different persons. In some cases erythema on the skin and slight redness and excoriation of the throat are the only signs that are discovered. In other instances the erythematous rash is succeeded by papular and scaly syphilides, and by obstinately recurring lesions of the mouth, throat, genital organs, or other parts of the body.

After the lapse of a period which varies usually

from six to eighteen months, the secondary stage comes to an end, and in most cases the disease troubles the patient no longer. But supposing this not to be the case, there may be an interval of months or years during which no symptoms appear. Sometimes, however, symptoms continue to develop from time to time, which partake of the characters of both the secondary and tertiary stages, and which gradually merge into the latter without any strict line of demarcation between the two. Examples of these intermediate signs are thickened scaly patches on the skin, which often take a circular or serpiginous form, and sometimes ulcerate; obstinate eruptions of the soles and palms; ulcers and induration of the tongue; periosteal swellings; orchitis; and affections of the choroid and retina.

3. Tertiary Syphilis.—While the secondary stage of syphilis is mainly characterised by the occurrence of superficial lesions, more or less widely spread over the surface, and tending to spontaneous disappearance; the main features of tertiary affections, on the other hand, are that they usually attack only a limited area, that they have a tendency to extend and to cause destruction of tissue with consequent contraction and scarring, and that they do not tend to spontaneous recovery.

The lesions classed as tertiary are due to invasion of some part of the body by the granulomatous or gummy growth which has been already described. The tertiary period, also, is sometimes, but by no means always, attended by severe cachexia, with a peculiar pallor of the skin.

Whether the syphilitic growth assumes a diffused or a circumscribed form, unless it be checked by treatment, degeneration is often rapid, leading to obstinate ulceration when the superficial structures are attacked, and to the production of tough contractile fibrous tissue when the viscera are the seat of the new-formation. Of the internal organs those belonging to the nervous system are probably most frequently affected.

When the gummy growth attacks the skin or mucous membrane in a diffused form, it produces hard, flat plaques of varying extent. The skin after a time becomes purplish-red and adherent, and finally breaks down at one or more points; an ulcer is left, which sometimes creeps over the surface (serpiginous ulceration), healing in the middle and extending at the margins, until a considerable amount of tissue is destroyed. When this diffused infiltration occurs in the pharynx, air-passages, or rectum, the disease is very obstinate, unless actively treated at an early stage, and the subsequent contraction may produce incurable and fatal stricture in any of those situations.

When gummata develop in the skin itself, the result is often called a *nodular syphilide*. Gummata in the cellular tissue, when discovered early, appear as small hard nodules, movable beneath the skin; but after a time, unless treated, they enlarge, soften, and become adherent to the discoloured integument, which finally gives way, exposing a mass of yellowish-white material, which is gradually cast off in the form of shreddy matter. The cavity then heals by granulation, leaving a depressed scar.

Local irritation or injury seems often to determine both the appearance and the site of some of the manifestations of syphilis; for example, the effects of smoking and broken teeth in the case of the throat and tongue, and a blow or squeeze in the case of the bones and testes.

The syphilitic affections of the other tissues, and of the various organs of the body, will be found described along with the other diseases of the several parts under their proper headings. See BRAIN, Syphilis of; BONE, Diseases of; LIVER, Syphilitic Disease of; TESTES, Diseases of, &c.

Malignant Syphilis.—It has already been mentioned that the division of syphilis into a secondary and a tertiary stage is to a great extent artificial; indeed, it is sometimes impossible to say under which title certain lesions ought to be classed. There are again cases, fortunately not common, to which the term 'malignant' or 'galloping syphilis' has been applied, in which the disease from the first pursues a rapid, destructive, and sometimes uncontrollable course. Skin-eruptions, which partake of the secondary stage in their wide-spread character, and of the tertiary in their tendency to ulcerate—*rupia*, for example—appear early, even while the initial lesion is still present. Deep ulcers form also in the mucous membrane, and the gummy growth may develop in the internal organs, and cause death; or the patient may die worn out by exhaustion, or from some intercurrent inflammation.

Inherited Syphilis.—Syphilis is a very frequent cause of *abortion*, especially if the parental infection be of recent date, or treatment have been neglected. If the child be carried the full term, it may be born dead, with or without signs of syphilis; most commonly, however, if the child be born alive, it does not show any signs of the disease at birth.

In inherited syphilis the initial manifestation is presumably absent, and after birth the disease appears to begin at the secondary stage. So far as is at present known there is no difference in the clinical signs of infantile syphilis, whether the infection be derived from one or both parents, or whether it be germinal or placentar. The symptoms of the inherited are in their main features similar to those of the acquired disease, allowance being made for the difference between the actively growing tissues of the child and the fully developed organs of the adult. There are, however, some points that deserve notice. The first is the frequency with which the nasal mucous membrane is attacked, the rhinitis giving rise to one of the earliest and most characteristic symptoms, namely, snuffling. If the inflammation extend to the deeper structures, flattening or other deformity of the nose may follow. Another peculiarity is a tendency, much more marked in inherited than in acquired syphilis, to early mingling of secondary and tertiary symptoms. Lesions of the bones and viscera, for instance, may occur, not only with the earlier skin-eruptions, but even during intra-uterine life. The bones most liable to be attacked are those of the cranial vault, and the long bones of the extremities. *Craniotabes*, especially of the occipital region, seems to be frequently, though not always, due to syphilis. See BONE, Diseases of; and SKULL, Diseases of.

Supposing the child to have been born alive, it usually shows no evidence of disease for a period ranging between two and six weeks, sometimes longer, after birth. It then, as a rule, gradually loses its healthy appearance, begins to snuffle, becomes fretful, and wastes more or less rapidly. The child's skin assumes a dull dirty colour, and, though loose and wrinkled, is very brittle and easily breaks round the mouth and nose into chaps and

fissures, the scars of which often form a characteristic sign in after-life, if the child survive.

The *erythematous syphilide* in inherited syphilis shows a marked difference from that of acquired syphilis. Instead of numerous small spots spread widely over the surface, it usually consists of larger dusky patches, which generally appear first about the buttocks and genital region, and often extend down the lower extremities. The soles and palms also are frequently affected, and sometimes the eruption spreads over the whole body. Desquamation very often follows. Papular or pustular rashes, resembling those seen in the adult, may also appear; and mucous patches are nearly always present, sooner or later, about the orifices of the body. Affections of the hair, the nails, the testes, and the viscera, similar to those of acquired syphilis, are also found in young children. A bullous eruption is seen only in severe cases; it appears very early, and is frequently fatal. Though it has been asserted recently that infection from a child who has inherited syphilis is very rare, still in practice it will be wise to look upon the earlier lesions as equally infectious with those of the acquired form of the disease.

Though a large majority of those who suffer in infancy show no signs afterwards, yet later on, usually from about the fifth to the fifteenth year of life, inherited syphilis may manifest itself by a number of symptoms which were formerly ascribed to 'scrofula.' The growth is checked, the individual being stunted, weakly, ill-nourished, and of low vitality. The forehead is prominent, and the bridge of the nose sunken. The skin is pallid, and perhaps ulcerated, or scarred by previous ulceration. The permanent teeth, especially the central upper incisors, as was first pointed out by Hutchinson, may be dwarfed, narrowed towards the cutting edge, and notched at the centre of the free border (see *TEETH*). The cornea is liable to a form of diffuse inflammation (interstitial keratitis), leading to more or less opacity and impairment of vision; the iris and the deeper structures are also liable to be invaded; and deafness is sometimes present. These affections of the eye and ear are, according to Hutchinson, much more common in girls than in boys (see *EYE*; and *EAR*). The bones of the palate and nose may be diseased; and nodes may appear in various situations; as well as a form of chronic synovitis, most frequently affecting the knee-joints. The fingers and toes also, and, more rarely, the metacarpal and metatarsal bones, may become enlarged. This form of dactylitis is most common in early life; but it has been observed after puberty, as well as rarely in connection with acquired syphilis. The soft palate and pharynx may be the seat of ulceration, which sometimes becomes phagedænic. The deeper tissues and the viscera are also liable to be attacked by processes similar to those which occur in the acquired disease, giving rise to a variety of symptoms, and sometimes leading to a fatal issue. Some authors believe that early life may be passed without symptoms, and that these later affections may constitute the first signs of inherited taint.

COURSE, DURATION, AND TERMINATIONS.—The course of syphilis varies very much, according to the individual, and according to the treatment adopted. In the majority of cases of acquired syphilis the disease runs its course within a year and a half or two years; but in certain rare

cases it appears to end with the first eruption; sometimes, perhaps, even earlier than this. When tertiary symptoms ensue, the commonest period for their appearance is probably from three to five years after infection; but they may be delayed for ten or twenty years, or even longer. On the other hand, as has been already stated, the secondary may run on into the tertiary stage without any appreciable interval. When tertiary symptoms have once appeared, the duration of the disease is uncertain, depending greatly on the constitution and habits of the patient, and on the effects of treatment. In some cases the patient continues to suffer throughout his life.

Besides what, for want of knowledge, we call idiosyncrasy, the course and duration of syphilis are also influenced by many other circumstances—for example, climate, age, pregnancy, and the hygienic surroundings of the patient.

The greater or less severity of the disease, or the stage at which it may have arrived in the person from whom it is contracted, does not appear to have any appreciable influence on the course of acquired syphilis.

The usual termination of syphilis is in recovery, and this in many cases probably without treatment, if the patient suffer only from the more superficial forms of disease. In syphilitic disease of the viscera, unless it be recognised and treated at an early stage, the termination is frequently fatal.

In *inherited syphilis*, if the child be born with signs of syphilis it usually dies; but when it remains healthy for some weeks after birth, the disease mostly ends in recovery within a year, provided proper treatment be carried out. In later childhood, however, or in adolescence, even up to the thirtieth year according to Hutchinson, further symptoms may develop; in which case the duration and termination will depend greatly upon their due recognition and appropriate treatment. When the disease is neglected, death is a frequent termination, especially among the ill-fed children of the poor.

COMPLICATIONS.—*Phagedæna* is an occasional complication of the initial lesion, but more frequently of the later ulcerating syphilides. The *local chancre* is another complication, giving rise to what has been termed the 'mixed chancre.' *Erysipelas* sometimes attacks syphilitic patients, and it is said to act beneficially in causing the disappearance of certain obstinate eruptions. This influence, however, is only seen in some chronic cases when the patient is otherwise in fair health. If erysipelas attack a cachectic person, with rapidly spreading lesions, the complication is a serious one, and may prove fatal. In *tuberculous* patients syphilis is often obstinate and severe. The skin-affections in such persons are prone to ulcerate, and syphilis seems in some cases to rouse the tuberculosis into activity. The *gouty* diathesis also influences the course of syphilis. The skin-eruptions often assume the scaly form, and sometimes resist treatment obstinately. *Bright's disease* is a serious complication. Patients whose kidneys are diseased are liable to suffer severely both as regards the superficial tissues and the internal organs. *Ague*, especially if recent and severe, is also a serious complication. *Alcoholism* is detrimental in two ways: first, by its injurious influence on the system generally; secondly, preventing the proper action of specific remedies.

DIAGNOSIS.—In the diagnosis of syphilis much will depend on the stage at which the disease has arrived when the patient comes under observation. Before the incubation-period has come to an end the diagnosis will of course be impossible; but when the initial lesion with its accompanying glandular enlargement has appeared, there is usually no difficulty, unless some local complication be present, or unless the diagnostic signs have been obscured by giving mercury too soon. The initial lesion is distinguished by its indurated base, the indolent superficial character of the ulceration when that is present, the thin serous discharge, and the indolent painless enlargement of the nearest lymphatic glands. Other points in connection with the diagnosis of primary syphilis are considered elsewhere. See VENEREAL SORE.

The diagnosis of the early syphilides does not, as a rule, present much difficulty, if the general characters already mentioned be attended to. In some cases, however, when pyrexia and general constitutional disturbance precede or accompany the outbreak of the eruption, the symptoms may be mistaken for those of some other disease. The erythematous syphilide has been mistaken for measles or scarlatina, and a pustular syphilide for small-pox. The various forms of drug-eruption, especially that due to copaiba, as well as other rashes due to toxæmia of various kinds, should also be borne in mind. In doubtful cases, careful attention to the temperature, and the condition of the tongue, throat, and air-passages, together with the history and the presence or absence of other signs of syphilis, will usually decide the question in a few days. The colour of a syphilide or scar, no matter how coppery it may be, is by itself of little value from a diagnostic point of view.

It is, however, at a later stage—perhaps many years after infection, and long after the disappearance of outward signs of the disease—that the diagnosis presents most difficulty to the physician. For example, a patient comes with obscure symptoms, pointing to some lesion of the nervous system, heart, liver, or other viscus. In such cases the skin and mucous membrane, particularly of the genital organs, mouth and throat, should be carefully inspected, and the bones and testes examined for irregularity or swelling. The eyes may afford important aid, by the detection of iritic adhesions or changes in the deeper structures. The presence of local paralyses, especially of the ocular muscles, is a valuable diagnostic sign. The absence of signs or characters distinctive of other diseases—cancer or tuberculosis, for example—is often also of value in doubtful cases.

When no conclusive information can be gained from any of these sources, the history becomes of the greatest importance. A venereal sore with lumps in the groins, and followed by a rash on the skin, sore throat or tongue, loss of hair, pains in the bones worse at night, and bad eyes, are some of the points that should be inquired into, and in women the occurrence of abortion or of still-birth. Symptoms of fever, either continued, remittent or intermittent, may occur both early and late in syphilis, and with or without obvious signs of the disease. In obscure cases of fever, therefore, syphilis should be thought of as a possible cause.

In investigating the history of a supposed syphilitic person, it should be borne in mind that syphilis is not necessarily a venereal disease, and that conse-

quently the initial lesion may have been far away from the genital organs, and thus mistaken or overlooked; also that the early manifestations, particularly in women, may be so slight and so evanescent as to escape observation. Again, some may have forgotten that they have suffered from earlier signs; and others who do remember obstinately conceal the fact. However this may be, the practitioner frequently fails to elicit any clear history of earlier manifestations in those who suffer from visceral syphilis. Sometimes also, for various reasons, the history is not available. If neither the symptoms present at the time, nor inspection of the patient's body, nor the method of exclusion, nor the history of the case, separately or combined, suffice to render the diagnosis clear, specific remedies should be administered, and their effect awaited before an opinion is given. But it is well to be cautious in drawing conclusions from such evidence. The failure of specifics, in an affection of the nervous system for instance, does not prove that the case is not syphilitic, for they may have not been given early enough to check the disease. Again, improvement may occur under mercury or iodides in cases where there is no syphilis.

In the diagnosis of *inherited syphilis* at an early age, snuffling and coryza, when persistent and well-marked, are of much importance. Radiating cracks around the mouth and nostrils are also valuable signs. Besides the eruption on the skin, especially that of the buttocks and soles of the feet, mucous patches should be looked for about the mouth, anus, and genital organs. A bullous eruption of the palms and soles is nearly always syphilitic, but it is rare. Enlargement of the spleen is a valuable corroborative sign. The bones of the skull should always be examined for nodes and cranio-tabes, and those of the limbs for nodes and epiphysal enlargement. Nodes on the skull occur most frequently on the frontal and parietal bones, especially near the anterior fontanelle (Parrot's nodes). As a distinction from rickets it has been pointed out that while the frontal and parietal eminences themselves are affected by rickets they are not affected by syphilis. Pallor and wasting are common; indeed wasting alone, without other discoverable cause, points towards syphilis; but it must be remembered also that a syphilitic child who suffers but slightly may retain a healthy appearance throughout.

In later childhood and adolescence the most valuable diagnostic signs are the low stature and puny development; the dull, pasty complexion; radiating scars about the mouth; the prominent forehead; the broad and sunken bridge of the nose; signs of present or past mischief in the cornea, iris, or choroid; dwarfed and notched median upper incisors; deafness of high degree, symmetrical, and without otorrhœa; nodes on the bones; unhealthy ulceration, often closely resembling lupus, or its scars, especially of the face or throat. Here, again, if no conclusive signs be present, the history becomes most important, both of the patient himself and of his immediate relations, and here also the diagnosis has occasionally to be postponed until antisyphilitic remedies have been administered. Lastly, it is too often forgotten that syphilis in children is not necessarily inherited, but that it may have been acquired by suckling or in some other way. In the diagnosis between acquired and inherited syphilis in children, the points to be attended to are the presence or absence of a primary

lesion, and of glandular enlargement, and the characters of any eruption that may appear, as well as the other signs already mentioned.

PROGNOSIS.—This, in the great majority of cases, is favourable, if the patient be otherwise in good health, of temperate habits, and especially if his disease be properly treated from an early period. The effect of other constitutional diseases has already been indicated among the *complications* of syphilis. Probably nothing tends more to prolong and aggravate the course of syphilis than habits of drinking. In persons given to alcohol, therefore, the prognosis should always be guarded.

An interesting question, and one on which further information is needed, is whether any trustworthy data as regards prognosis can be gained from the character of the early manifestations. Neither the length of incubation, nor the amount of induration or ulceration of the initial lesion, has been shown to afford any trustworthy evidence as regards prognosis. Early *general* glandular enlargement is often an unfavourable sign. Persons thus affected become anæmic, and consequently more liable to grave affections. As regards the early syphilides, it may be mentioned that the ordinary well-marked macular and papular eruptions appear to be less common precursors of late visceral affections than early rashes that are ill-marked and scanty. Obstinate, frequently recurring lesions of the skin and mucous membrane also seem to be rarely associated with visceral disease. In connection with this may be mentioned the frequent failure of the physician to elicit any history of early symptoms in those who suffer at a later period from grave visceral affections, especially from syphilis of the nervous system.

In the cases of malignant or galloping syphilis, in which ulcerating and rapidly spreading lesions attack the skin and mucous membranes, and are associated with a tendency to phagedæna and great prostration at an early period after infection, the prognosis should be guarded: the patient may die worn out by the pain and profuse discharge of the superficial lesions, or of some acute affection. In syphilitic disease of any of the internal organs, the prognosis will depend greatly on its early recognition and treatment.

One of the most important points to be considered in prognosis is the way in which the patient has been treated. For, although we do not yet know any certain sign—save re-infection—which proves that syphilis has come to an end, it may with much confidence be stated that if mercurial treatment have been begun at an early period, and continued a sufficient length of time, and if the patient be constitutionally healthy and of temperate habits, the chances are very greatly in favour of complete subsidence of the disease within two years.

When syphilis causes death it is usually through the so-called tertiary lesions, especially perhaps syphiloma of the blood-vessels and notably those of the brain and spinal cord. The proportion of cases in which tertiary affections occur is probably not more than 10 per cent., and in a large majority of these the vital organs escape. Much progress has been made of late years towards the better and earlier diagnosis of visceral syphilis, and it seems probable that the prognosis will become more and more favourable as time goes on.

Much of what has been said as to the prognosis

of acquired syphilis applies also to the inherited form of the disease. A child in whom symptoms do not appear until several weeks after birth, who is well cared for, properly fed, and who receives proper medical treatment, will probably recover; one who is ill-fed and neglected will most probably die. Indeed, among the children of the poor, syphilis is one of the most fatal diseases of infantile life.

TREATMENT.—I. **Preventive Treatment.**—At the present time there are unfortunately no State regulations to check the spreading of venereal diseases in this country.

Among individuals the strict daily observance of cleanliness, too often neglected by both sexes, constitutes the best protection against disease, and by preserving the epithelial surface in a sound and healthy condition would of itself, if generally practised, do much towards reducing the prevalence of syphilis as well as of other venereal diseases.

There is no trustworthy evidence that syphilis can be cut short by removal or destruction of the initial lesion. Indeed, cauterisation within twelve hours after exposure has failed to prevent infection.

Every person who contracts syphilis should be warned of the danger of communicating it to others. Sexual intercourse should be prohibited while the disease remains active. This direction should never be omitted, as many patients are unaware that they are liable to communicate the disease after the primary sore has healed.

The infectious nature of the lesions of the mouth and throat should also be pointed out, and the consequent risk of infection by kissing, or by the use of drinking-vessels, pipes, towels, or other articles in common with other persons.

The question of *marriage* in relation to syphilis is one of great importance, and it is the duty of the practitioner to prevent the contamination of a healthy person, or the procreation of syphilitic children, whenever it is in his power to do so. No person who shows signs of active syphilis should marry, however long a time may have elapsed since infection; for although communication of the disease is probably rare after two years have gone by, it may take place after ten years, or even longer in neglected cases. It has already been stated that syphilis usually ceases to be active within two years; but this is not always so. After the last symptoms have disappeared, there should be an interval of at least a year before marriage takes place; but the period between infection and marriage should never be less than three years. If the treatment during the early stages have not been systematic and prolonged, it will often be advisable to subject the candidate for marriage to a course of mercury for about three months, and after this to keep him under observation for a year before the marriage takes place.

If a syphilitic man have a relapse after marriage with a healthy woman, he ought at once to desist from intercourse—indeed, from contact of every kind—and undergo thorough treatment. The wife also should be watched, that treatment may be begun as early as possible, if she have contracted the disease. A woman who has borne a syphilitic child should as a rule be treated with mercury whether she show signs herself or not.

The spread of syphilis from children to nurses, and *vice versa*, must also be guarded against, and the practitioner should never allow a syphilitic child

to be entrusted to a healthy wet-nurse, nor a syphilitic nurse to suckle a healthy child.

2. Curative Treatment.—The treatment of syphilis may be divided into (a) *general*; (b) *specific*; and (c) *local treatment*.

(a) *General treatment.*—Syphilis is essentially a debilitating disease; hence it is important that the general health of the patient should be supported by nourishing diet, good air, warm clothing, extreme moderation as regards alcoholic liquors, cleanliness, cheerful society, and moderate exercise in the open air. In most cases the patient may follow his usual employment, unless it involve prolonged exposure to damp and cold. The skin should be made to act freely, by the frequent use of soap and water, and an occasional vapour- or Turkish bath if thought desirable. Some woollen material should be worn next the skin, and chills avoided as much as possible. Smoking in moderation may be allowed as long as the mouth and throat remain free; but when syphilitic lesions are present, it should always be discontinued, and if they show a tendency to recur smoking should be abandoned altogether. Regular action of the bowels is also important.

(b) *Specific treatment.*—Besides attention to the general health of the patient on ordinary principles, the administration of certain drugs which are known to have special influence over syphilis should never be omitted, however mild the earlier manifestations of the disease may be. The subcutaneous injection of serum both from inoculated animals and from syphilitic persons has been practised to some extent during recent years; but nothing can be definitely stated at present as to the value of this mode of treatment.

The special drugs now almost exclusively used in the treatment of syphilis are *mercury* and the various salts of *iodine*. Probably the former only can be looked on as a permanent remedy. The iodides are of great value in some of the earlier as well as in most of the later manifestations of syphilis; but the effects are not always lasting, and therefore they cannot be trusted to alone.

Much of the prejudice against the use of *mercury* has arisen from the fact that in former times its administration was carried to a poisonous extent. It is now known that if the drug be administered in suitable doses, it acts as a tonic, syphilitic patients rapidly improving in health and gaining weight under its use.

As soon as a diagnosis of syphilis has been made mercury should be given. It is sometimes advised that the rash should be waited for 'to clinch the diagnosis'; but in most cases the diagnosis can and ought to be made before that, and it must be remembered also that the rash may be mistaken or overlooked as well as the earlier signs. But the strongest argument in favour of early treatment is to be found in the fact that grave and sometimes fatal lesions may occur within a few months after infection. For all the lesions both of the primary and of the secondary stage mercury is appropriate, and in the later stages it should be used in conjunction with iodides, as well as in cases where the iodides fail to relieve. Mercury may be given to almost all persons when its use is indicated, but its effect must be carefully watched if the patient be in feeble health or the subject of disease of the kidneys. Mercury is sometimes said to be contra-indicated in tuberculous patients, but in the writer's experience

they bear the drug as well as others if care be taken in its administration.

Before a mercurial course is begun the mouth should, as far as possible, be put in a healthy condition. Unsound teeth should be stopped or removed, and tartar got rid of. If the gums are spongy, an astringent mouth-wash should be used frequently. The *urine* should always be examined before treatment is begun, and at intervals afterwards. At present it is by no means clear how much of the renal irregularity indicated by albuminuria in syphilis is due to the disease and how much to other causes, including possibly the drugs employed in its treatment. The *weight* of a syphilitic patient under treatment should also be noted from time to time. The patient should also be warned that fruit and green vegetables may cause diarrhoea during a course of mercury. Milk usually agrees well, and may be taken freely.

When mercury is taken in syphilis, the patient usually improves rapidly. If a rash be present it soon begins to fade, and ulcerated surfaces begin to heal. All the necessary results are in most cases attained without any, or with only the slightest, visible effect on the gums.

Every case of acquired syphilis in an adult should be treated by mercury for at least a year. During this period it will be necessary to omit the drug from time to time, and perhaps to vary its form and mode of administration; but what should be the length of each course, and what the length of the intervals, are questions that must be decided according to the symptoms that appear, and according to the effects of mercury in each case. After the first year no fixed rules can be laid down. As long as there are signs of syphilis treatment must of course be carried out; but the particular drug to be chosen, its dose, and the length of time it should be continued, ought to depend on what has been learned beforehand about the patient and his malady, as well as on the treatment he has already undergone.

Mercury may be introduced into the system in various ways—by the stomach, rectum, vagina, skin, or subcutaneous tissues; but its action is essentially the same by whatever channel it is administered.

Mercury is most conveniently given by the mouth, and in ordinary cases the results are probably quite as good as under any other mode of administration. In early syphilis *blue pill* usually fulfils all the requirements of the case; and the writer has found this more generally useful and less likely to disagree than grey powder. If no urgent symptoms be present, a grain of one or other of these preparations may be given, with a little extract of gentian, three or four times a day, immediately after meals. Quinine or reduced iron may be added, if thought desirable, but opium is usually unnecessary. In such doses mercury rarely causes either stomatitis or purging, but the possibility of both should always be mentioned to the patient. If, however, a rapid effect be desired—in *iritis*, for example—2 or 3 grains of blue pill, with $\frac{1}{4}$ or $\frac{1}{3}$ grain of opium, may be given three or four times a day, the patient being kept indoors, until the requisite effect is produced, after which the drug should be given less frequently, or be reduced in quantity. If salivation be inadvertently set up, the mercury should be discontinued for a time, a purge administered, and the mouth washed out frequently with a gargle of alum or chlorate of potassium. See MERCURY, Diseases arising from; and SALIVATION.

The green iodide of mercury is an active salt, which is largely used on the Continent, especially in France, but it is more liable to decomposition and to cause irritation than the forms just mentioned. It may be given in doses of $\frac{1}{4}$ to 1 grain, with a little opium, twice or thrice a day.

The perchloride of mercury is mostly given in the later and more chronic forms of syphilis, but in the early stages it is sometimes better borne than almost any other preparation. It may be prescribed in doses of $\frac{1}{16}$ to $\frac{1}{8}$ grain, either in a mixture with iodide of potassium or with perchloride of iron, or in a pill, according to circumstances. The red iodide is also useful, especially in cases of relapsing scaly syphilides.

When other preparations purge or otherwise disagree, the *tannate of mercury* in 1 or 1½ grain doses sometimes answers well.

A mixture of sarsaparilla and aromatics with antimony or mercury, known as *Zittmann's decoction*, is sometimes useful in tertiary syphilis.

Inunction is very effective, but it is troublesome to the patient; hence he may neglect to carry it out properly. Provided the skin be in a suitable condition, it may be employed whenever mercury is indicated, especially when for any reason it is deemed inadvisable to give the drug by the mouth. See INUNCTION.

The mercurial vapour-bath is not a convenient routine method of treatment, but is beneficial in certain cases, especially those of widespread rash. See FUMIGATION.

Subcutaneous or intra-muscular injection is probably the quickest way of obtaining the effects of mercury, but it is more or less painful, and not altogether free from risk; hence it is only to be recommended when other means fail, or in very urgent cases, or perhaps in the case of the Army and Navy if it be found that the men cannot be trusted to carry out their treatment in the ordinary way. As a rule this method should not be employed on a patient who has not previously taken mercury, lest he chance to be peculiarly susceptible to the drug, and so in danger of being poisoned. Mercurial injections are usually best made deeply into the muscles of the buttock or back. Nearly every known preparation of mercury, soluble and insoluble, has in turn been employed, but there is no general agreement as to which is the best. A solution containing 1 part of the red iodide with sufficient iodide of sodium to dissolve it in 64 parts of water answers well. The dose is from 4 to 8 minims, and it may be repeated daily or less often according to the case and the effects produced.

The *intravenous* injection of mercury has been advocated by a very few surgeons. The writer has no experience of it.

The salts of *iodine* are principally used in the treatment of tertiary syphilis; but it is a mistake to look upon them as adapted only for that stage. In the secondary period, and in cases where the lesions partake of the character of both stages, the iodides may often be advantageously combined with mercury. Speaking generally, the writer believes that the iodides are indicated when there is evidence of a marked tendency to new-growth at any time after the primary stage. In cases where the symptoms have been controlled by iodine, mercury should, as a rule, be given to complete the cure.

Iodide of potassium is the salt most frequently used; but the iodides of *sodium* and of *ammonium*

are also employed. The action of all is similar, but the sodium and ammonium salts appear to be less depressing. They may be given separately or combined. The dose of the iodides varies from 2 or 3 to 100 grains or even more. In ordinary circumstances it is best to begin with 3 or 4 grains, with a little ammonia, three times a day, and to increase the dose if necessary. In urgent cases, such as those of naso-pharyngeal disease, in which deformity is threatened, as well as in some cases of visceral disease, particularly in syphilitic affections of the nervous system, from 10 to 20 grains may be given at first, and the dose rapidly increased until some effect is produced. In such cases, also, it will often be prudent to carry out inunction, or the subcutaneous injection of mercury, at the same time. The iodides should be given well diluted; and any of the bitter infusions, or the citrate of iron and ammonia, may be added when their use is indicated. Milk is an excellent vehicle, especially for children. Iodides may also be effectively administered *per rectum*.

Donovan's solution (Liquor arsenii et hydrargyri iodidi) acts well in many of the later affections of the skin and mucous membranes. The dose is from 5 to 30 minims.

Iodoform is occasionally given internally in the later stages of syphilis; but it frequently causes gastric and intestinal irritation. The dose is about a grain, in the form of a pill.

The *bromides* of potassium, sodium, and ammonium are serviceable in certain cases of syphilitic affection of the nervous system. They may be given alone or with iodides, according to circumstances.

Besides the foregoing remedies, many others may be required in the treatment of syphilis. Iron and nux vomica or strychnine are drugs that are frequently of the greatest value, either in conjunction with specifics or with quinine. Cod-liver oil also is valuable in many cases. Sarsaparilla is sometimes useful during or after a prolonged course of specifics. Mineral acids and vegetable bitters are often of service during the intervals of specific treatment. Opium is sometimes necessary to prevent the purgative action of mercury, especially when large doses are taken, as well as to relieve pain in some of the affections produced by syphilis.

Certain bathing-resorts which possess *sulphur-springs*—Aix-la-Chapelle, for example—have become noted for the cure of syphilis; and there can be no doubt that great benefit is often derived from the course of treatment pursued at such places. The good results, however, are due rather to a combination of specific remedies with strict attention to diet and general hygiene, than to any special virtues of the waters. As far as the writer's observation goes cure is not more certain, and relapse is not less liable to occur after treatment at Aix than elsewhere.

When syphilis is complicated with tuberculosis, gout, rheumatism, or other disease, the appropriate remedies should be given with those proper for syphilis, or temporarily substituted for them according to circumstances.

A certain number of persons are either unusually susceptible or unusually insusceptible to mercury or iodides in ordinary doses. Nearly all such cases, however, can be managed by making the dose small enough or large enough as the case may be, by combining tonics or sedatives with the specific, or by altering the mode of administration, together

with strict attention to diet and the manner of living generally.

(c) *Local treatment.*—The uncomplicated initial lesion of syphilis usually requires only cleanliness, and the application of a little calomel-powder or black wash. If the sore suppurate, it should be cleaned, dried, and dressed with finely-powdered iodoform. Phagedæna must be treated by immersion, caustics, or the actual cautery, while the general health receives attention (*see* VENEREAL SORE). If the lymphatic glands become tender or inflamed, warm fomentations should be applied, and the patient kept lying down. If an abscess form, it must be treated according to the directions given in the article on BUBO.

The early eruptions on the skin rarely require local treatment; oleate-of-mercury ointment may be used for conspicuous spots. Erosions or fissures may be dressed with an ointment of calomel and vaseline, or with iodoform. In the scaly affections of the palms and soles, an ointment of ammoniated mercury and oxide of zinc may be well rubbed in at bedtime, gloves being worn during the night. Mercurial plaster also is a useful application. Mucous patches should be cleansed and dried several times daily, powdered with calomel (1 part) and oxide of zinc (3 parts), and covered with dry lint. If they are large and prominent, pure carbolic acid or acid nitrate of mercury may be applied from time to time. Cracks and ulcers about the nails should be dressed with ointment of red oxide of mercury.

Ulcers or fissures, or mucous patches of the tongue, mouth, and throat should be touched with carbolic acid, and a chlorate of potassium and borax gargle or spray used frequently, especially after eating. Other valuable applications are a mouth-wash of perchloride of mercury, $\frac{1}{8}$ to $\frac{1}{4}$ grain to the ounce, or a stronger solution applied with a mop; and a solution of chromic acid, from 10 to 20 grains to the ounce of water. For the later ulcers internal treatment is of the first importance.

Ulcers about the lips and nostrils should be kept moist with ointment of red oxide of mercury and vaseline. When necrosis of the nasal or palatal bones has occurred, a lotion of permanganate of potassium or chlorinated soda should be used frequently with a syringe or as a coarse spray, and the fragments removed as soon as they become loose. The early affections of the larynx usually disappear without local treatment. In the later affections general treatment is most important, but tracheotomy may sometimes be required.

Ulcers about the anus require careful cleansing and the application of calomel- or iodoform-ointment. For ulceration within the rectum iodoform suppositories and antiseptic injections should be used. In stricture of the rectum careful dilatation by means of bougies may be tried. In extreme cases proctotomy, or even colotomy, may have to be performed.

As regards the bones, the pain produced by early nodes is relieved by painting with a solution of iodine. As a rule, nodes should not be incised. If necrosis take place, the dead bone should be removed as soon as it becomes loose.

In iritis, besides the prompt administration of mercury, a solution of sulphate of atropine (1 grain to 2 drachms of distilled water) should be dropped

into the eye every two hours till the pupil is well dilated. Afterwards a weaker solution may be used less frequently to keep up the effect. In interstitial keratitis also atropine should be used. Iridectomy is occasionally necessary. In choroiditis and retinitis leeching is sometimes useful when there is much pain, but constitutional treatment is most important.

In syphilitic orchitis a suspensory bandage should be worn, but other local treatment is usually unnecessary, unless a gumma break down and fungous protrusion occur, in which case support should be given by strapping. Syphilides of the female genital organs should be dressed with iodoform, and the application of strong carbolic acid is also often beneficial. If the cervix uteri be affected, vaginal injections will be needed as well.

Treatment of Inherited Syphilis.—In the case of a syphilitic child, whether the disease has been acquired or inherited, mercurial treatment should always be adopted as soon as symptoms appear. Grey powder may be given in one-grain doses twice a day; or a strip of flannel smeared with diluted mercurial ointment may be worn round the waist. The ointment should be renewed every night, and the skin cleansed every third day. Some preparations of iron and cod-liver oil are also often useful.

Iodide of potassium is most valuable in the later forms of the disease. The rules for its employment, alone or with mercury, are the same as in acquired syphilis, but the dose must of course be adapted to the age of the patient.

Opinions differ as to the length of time the treatment of a syphilitic child should be continued. Probably three months at least is usually advisable, and if symptoms then persist, it will be wise to continue treatment for several weeks after they have disappeared.

The *local treatment* of the syphilitic affections of children is similar to that recommended for adults. When the nostrils are obstructed by inspissated mucus, they must be carefully cleansed with a borax-and-glycerine lotion by means of a camel's-hair brush or a syringe, and sores dressed with dilute ointment of red oxide of mercury.

The *general management and diet* of syphilitic children are most important. Whenever the mother can suckle her child, she should do so. Though a very few exceptions to Colles's Law have been reported, this rule, in the writer's opinion, need not be altered; but if it be impracticable, a wet-nurse who has already suffered from the disease is the best substitute. If neither be available, ass's, goat's, or cow's milk must be given. Extreme cleanliness and fresh air are essential.

ARTHUR COOPER.

SYRINGOMYELIA (σὺριγγῆ, a pipe or tube; and μῦελος, the marrow).—A name under which Ollivier grouped numerous cases in which cavities of different kinds were met with within the substance of the spinal cord. *See* SPINAL CORD, p. 1559.

SYSTOLIC.—Of or belonging to the systole or contraction of the heart, and usually associated with the cardiac impulse or sounds, or with murmurs. *See* PHYSICAL EXAMINATION.

T

TABES (*tabes*, a consumption).—A term formerly employed to denote consumption or wasting of the body.

TABES DORSALIS.—SYNON.: Locomotor Ataxy; Fr. *Ataxie Locomotrice*; Ger. *Graue Degeneration der Hinterstränge des Rückenmarks*.

DEFINITION.—A disease of the spinal cord alone, or of the spinal cord and peripheral nerves; manifested, when considerable, by inco-ordination of movement, peculiar pains, defective sensibility, and loss of muscle-reflex action (myotatic irritability; 'tendon-reflexes').

ÆTIOLOGY.—Males suffer more frequently than females, the proportion being about ten to one. A neurotic heredity can occasionally be traced; seldom direct inheritance. Among individual causes one greatly exceeds the rest, namely, the influence of syphilis. When every allowance is made for accidental coincidence, it is found that in at least one-half the cases the coincidence must depend upon causal relationship, and it is probable that this is true of a much larger proportion. Many authorities, indeed, believe that is the almost constant antecedent. The interval between the primary manifestations of syphilis and the first symptoms of tabes varies from one to twenty years. It is rarely less than three years, although cases are met with in which the first symptoms occur during the active stage of syphilis. But syphilis is not the only cause, and in a few cases (less than 10 per cent. of the whole) it can be excluded with confidence. The other causes are traumatism, exposure to cold and wet, and over-exertion. Alcoholic and sexual excess are doubtful factors in its production; it has been said to occur as a sequel to certain acute diseases, such as acute rheumatism, typhoid fever, and diphtheria, and as a manifestation of a primary tendency to degeneration. Symptoms of tabes also occasionally succeed other diseases of the spinal cord, such as myelitis, such sequence being especially common in syphilitic subjects. This form has been called 'secondary tabes.'

ANATOMICAL CHARACTERS.—In most cases the spinal cord presents changes visible to the naked eye. These consist in a grey translucent appearance of the posterior columns. The whole of these columns may be thus changed or only parts of them, the extent of the changes being usually proportional to the severity and duration of the disease. With the microscope this change, or 'sclerosis' as it is called, is found to consist in an overgrowth of the interstitial elements, at the expense of the nervous elements in the areas affected. In the most common conditions the sclerosis occupies the whole of the posterior columns in the lumbar region, and becomes gradually restricted to the postero-median columns higher up, having thus the character of an ascending degeneration. In rare cases of severe character and long duration the posterior columns are occupied by connective tissue throughout the whole vertical extent of the cord. In slight cases the sclerosis may be limited in the lumbar region to the posterior root-zone, affecting the median column higher up. Sclerosis may also affect other areas of the cord. The fine fibres of the posterior

root, Lissauer's tract, are generally affected. The direct cerebellar and ascending antero-lateral tracts may also show degeneration. In rare cases complicated with paralysis even the pyramidal tracts are affected. Often in old cases there is a slight general increase of the connective tissue throughout the cord.

Changes in the grey matter are difficult to discover in the slighter cases. Some atrophy of the anterior cornual cells may be detected in cases complicated with true muscular atrophy. The column of Clarke may show distinct degeneration, and this is said to be associated with sclerosis of the direct cerebellar tract. The grey commissure also may be atrophied, and the nerve-cells and fibres in the posterior horn probably undergo changes which are difficult to detect.

The posterior nerve-roots are also atrophied, and the degree of atrophy depends upon the severity of the affection. The histological changes extend as far as the ganglion. The peripheral nerves are altered, the change consisting in a wasting of fibres, which seems to commence in the white substance and to extend to the axis-cylinders. There may be an increase of interstitial tissue, but the change is essentially one in the nerve-elements themselves. This degeneration is greatest in the terminal filaments, and usually lessens upwards, but may extend up to the posterior ganglion. The sensory fibres are alone affected. The changes in these have been chiefly found in the nerves supplying the skin, but Déjérine has demonstrated the presence of similar conditions in the sensory nerves of muscle.

SYMPTOMS.—The most common and characteristic symptom of tabes is the inco-ordination in walking which has given to the disease its common name. This varies in degree in different cases, and may be entirely absent. Before the condition is sufficiently marked to cause the characteristic disorder in movement, it may render standing difficult, especially if the eyes are closed ('Romberg's symptom'); and a still slighter degree may be evidenced only by the startings of tendons in the uncovered feet in the effort to maintain the equilibrium. As the defect progresses, it gradually impairs the patient's ability to walk; and finally locomotion is rendered impossible without assistance. As a rule, the affection of the lower limbs is earlier and much more severe, but the arms may be similarly affected, and, rarely, the ataxy in the arms is the earliest obtrusive symptom. There is often a difficulty in fine movements, such as buttoning the coat; the grasp is not sustained; and there are slow unintentional movements in the fingers, when these are kept in one posture with the eyes closed.

Among sensory symptoms the most obtrusive are pains, especially the severe and distressing 'lightning pains.' These are present in the majority of cases, and are characterised by their momentary, acute, stabbing character. The darts of pain recur in the same place for hours, and may be felt in another part the next time. Such attacks may alternate with periods of entire freedom, varying in duration. Their onset often seems to be determined by the changes in the weather. In some cases they are absent. There are also frequently

present dull aching pains or other acute pains, very various in character and sensory paræsthesiæ, such as numbness and tingling. The sharp pains are often superficial, and then render the skin extremely tender at the place. Another sensory symptom often complained of is a sense of constriction in some part of the trunk, usually round the waist—the so-called ‘girdle-sensation.’ A similar sensation may be present in the leg or thigh or knee, as if a band were tied tightly round it.

Sensory disturbance may also be manifested by impairment of cutaneous sensibility. This is most frequent in the legs and feet. Sensibility for pain and for temperature usually suffers first and most. The temperature-sense seldom escapes when sensibility to pain is much impaired. There is still more frequently delay in the transmission of painful sensations, which may amount to one or two seconds, seldom more. Sometimes there is a rhythmical recurrence of the sensation. Localisation may be disordered, so that a prick on one leg is referred to the other, or a prick at one spot may be felt in several (*allochiria*). Tactile sensation is often perfect when that to pain is almost lost. In rare cases it is impaired more than that to pain, and now and then affected alone. The same changes are met with on the trunk and arms, even sometimes on the head. Sensory loss may also be present in the muscles, so that their contraction on electrical stimulation may be unfelt, and the muscular sense may be so impaired that passive extension is unfelt and the position of the limbs is not known unless they are seen.

Reflex action is also impaired. Cutaneous reflex action is usually impaired in proportion to the loss of cutaneous sensibility. In some early cases there may be some cutaneous hyperæsthesia, and the reflex action from the skin may then be in excess. More important is the condition of the muscle-reflex action shown by the state of the knee-jerk, the loss of which, as Westphal first pointed out, is one of the earliest and most constant symptoms of tabes. It is very rare for the knee-jerk to be obtained in any case of true tabes; its loss may precede years the onset of ataxy, although cases are sometimes met with in which it is lost only on one side and diminished on the other.

Many ocular symptoms occur in connection with tabes. Of these, the most important is the loss of the reaction of the pupil to light, while it still continues to act with accommodation—the so-called Argyll-Robertson phenomenon.² This condition is present in about five-sixths of the cases of tabes. Often also the normal dilatation that occurs on painful stimulation of the skin of the neck cannot be produced. The pupils may be very small, but not infrequently they are of normal size. Paralysis of different ocular muscles may also be present, with accompanying diplopia. The muscles supplied by the third nerve are most frequently affected, especially the levator palpebræ, causing ptosis. This paralysis may be transient—lasting a few days or weeks—or permanent. The former is most common in the early stage, the latter in the late stage of the disease. There may be a condition identical with ‘progressive ophthalmoplegia.’ Other cranial nerves are rarely affected, with the exception of those of the larynx. Abductor palsy of the vocal cords is an occasional and grave symptom.

Among the more serious and troublesome effects of tabes the various so-called ‘crises’ must be

enumerated. These are named after the various viscera affected. Thus, the most common are the gastric crises, which are manifested by severe sickness and nausea, often associated with epigastric pain. Severe attacks of laryngeal cough and spasm are not uncommon (‘laryngeal crises’). These are often associated with bilateral weakness of the abductors of the vocal cords. There are also vesical, bronchial, and rectal ‘crises,’ manifested by pain and other symptoms referable to the various viscera indicated.

The action of the sphincters is usually impaired. Retention of urine may be followed by incontinence. The latter is not infrequently the result of over-distension, and is often associated with cystitis. Constipation is common; in the later stages incontinence of fæces may be present. The sexual functions are usually altered. In the early stage there is sometimes increased sexual activity, associated with excess of the cutaneous reflex action, but in most cases there is impaired or abolished sexual power, often as an early symptom, but sometimes deferred to a late stage.

Vaso-motor and trophic disturbances are important. Among them may be enumerated local sweatings, disappearance of hair from the skin, ecchymoses, and altered growth of hair in connection with attacks of pain. The so-called perforating ulcer of the foot is almost confined to tabes, and is usually excited by cutting a corn too deeply. Changes in the teeth and nails also occur, leading to partial decay or total loss. Changes in the joints and bones are especially important, and were carefully investigated by Charcot. The bones may become brittle, and so render the occurrence of spontaneous fractures easy; but the most important condition is the so-called ‘Charcot’s joint,’ consisting in an irregular enlargement of the ends of the bones, often with atrophy of the articular surfaces. The irregular outgrowth of bone may be extensive, and the fibrous tissue is often accompanied by œdema outside it. The larger joints are most commonly affected, especially the elbows, knees, ankles, and tarsal joints. It is often excited by a slight sprain, which causes an excessive inflammation, followed by the chronic alterations.

COURSE AND TERMINATIONS.—The course of the disease is extremely variable. There are usually three stages—the first one, in which the gait is not yet affected (*pre-ataxic*); the second, in which there is distinct inco-ordination (*ataxic*); and a third, in which the ability to walk alone is lost (*paralytic*). The first stage varies in duration from a few weeks or months to as long as twenty-five years; and if the patients are subjected to careful treatment there is no progressive tendency shown in probably as large a proportion as one-half the cases. Some patients in the second or third stage may even much improve, so that a patient at one time unable to walk becomes able to get about with a fair amount of ease. When optic-nerve atrophy is present, this and the loss of the knee-jerk may remain for many years the only evidence of the disease. There seems less tendency in such cases for the spinal symptoms to increase.

The dangers of locomotor ataxy consist not so much in the rapid spread of degenerative processes in the nervous system as in the occurrence of renal disease secondary to retention of urine, which is apt to develop insidiously. Laryngeal palsy also

constitutes an element of danger; and suddenly occurring trophic disturbances have been known to prove fatal. General paralysis of the insane is a not unusual complication, or rather cerebral development of the same degeneration; and it runs the usual course as a rule. Valvular heart-disease is an occasional complication of the disease, and is probably the result of a syphilitic process. This also may lead to a fatal termination.

PATHOLOGY.—In discussing the pathology of this disease it must be recognised that in tabes the main incidence of the marked change is upon the afferent nervous system, and that it is a primary degeneration of the nervous elements. Its incidence is on the neurons, which have their nutritional centre in the cells of the posterior spinal ganglia, and the primary incidence of the disease seems to be on the afferent nerves from the muscles, and the fibres which continue their path, from the posterior ganglia, in the posterior median columns of the cord. The symmetry of the lesions suggests a blood-state as the cause, and the very frequent antecedence of syphilis leaves little doubt of its dependence on a post-syphilitic toxin, acting especially on these neurons, diminishing their vital endurance, and entailing their subsequent degeneration. It has also an action on the fibres of the optic nerve, and, in extreme cases, on many other structures in the nervous system. But its primary influence on the afferent muscle-neurons explains the early loss of the knee-jerk and the dominant ataxy. Similar changes and symptoms have been proved to result from other toxic influences, especially from alcohol. This may cause loss of the knee-jerk and ataxy from its action on the peripheral nerves, in what has been termed 'alcoholic pseudo-tabes.' The long interval which elapses between the primary disease and its nervous sequel is only in proportion to the chronicity of the primary specific disease; and the general conditions of its occurrence suggest that the action of the toxin is to induce the premature decay of the nerve-elements specially affected. This explanation enables us to understand the inutility of antisyphilitic treatment in tabes.

The variability in the amount of ataxy and the affection of cutaneous sensibility sometimes transcends the usual limits of the disease. After syphilis characteristic tabetic pains may occur, with loss of the light-reflex but without ataxy or even loss of the knee-jerk. In such cases there is often slight impairment of sensation in the skin, especially retardation to pain, and such cases may be designated 'cutaneous neuro-tabes.'

DIAGNOSIS.—Tabes dorsalis may easily be confounded with alcoholic neuritis. In the latter, however, there is the tenderness along the nerve-trunks, and the sphincters and pupils are unaffected. The history of alcoholic excess will also help in the diagnosis. This last, and improvement when the cause is withdrawn, are the chief distinctions in 'alcoholic pseudo-tabes.' The so-called hereditary ataxy 'offers some points of resemblance to tabes, but is distinguished by the earlier age at which it occurs, and the absence of the usual cause and of attacks of pain.' Diphtheritic paralysis may also bear some resemblance, but the history of the onset will usually make it distinct. Other forms of unsteadiness, such as ataxic paraplegia, are distinguished by the presence of the knee-jerk. Injury affecting the posterior columns,

or a tumour pressing on them, may simulate tabes but have distinctive symptoms. A tumour in the cerebellum, by giving rise to unsteadiness, may raise a diagnostic doubt, but its own special symptoms are never absent.

PROGNOSIS.—This must be based upon the considerations mentioned in the description of the course of the disease. Some cases are obstinately progressive, but arrest occurs, under treatment, in at least half the cases. It may be obtained in any stage, but is more frequent in the first, or 'pre-ataxic,' stage, before inco-ordination has developed. The prognosis is usually fair as regards duration of life, for the malady does not itself kill; and the most serious dangers to life may be to a large extent avoided. Optic-nerve atrophy is usually progressive, but not invariably so. The prognosis in any case must largely depend upon the ability and willingness of the patient to carry out necessary instructions and treatment.

TREATMENT.—Much can be done by treatment in tabes, at least to relieve, apparently even to modify considerably the course of the disease. The influence of rest and hygienic surroundings is very great. The avoidance of mental worry and fatigue is of the first importance. Exercise should be gentle, and should always stop short of fatigue. The avoidance of sexual excess is of the greatest importance, since it may lead to a most disastrous increase in the symptoms. Care should be taken to avoid dyspepsia and constipation.

Much certainly may be done by drugs. The almost invariable inefficacy of antisyphilitic remedies finds an explanation in the pathology already enunciated. It is seldom that anything but harm results from the energetic treatment for syphilis so commonly adopted. Arsenic is one agent generally useful. It may be combined with minute doses of mercury, such as seem tonic to those who have had syphilis, and may be alternated with phosphorus, quinine, and strychnine. Others that have been recommended are nitrate of silver, and ergot. Physostigma in some cases, belladonna in others, seems to have a slight beneficial influence, and in such a disease no agent even slightly useful can be despised. The writer has found chloride of aluminium very useful in lessening the tendency to attacks of pain. Counter-irritation to the spine is beneficial in cases in which symptoms come on acutely. The best way to apply it is by means of the mild actual cautery. Electricity has little influence on the disease. The application of faradism to the muscles to stimulate the afferent nerves might be expected to diminish the ataxy, and sometimes, but not often, does seem to exert a slight influence. The use of voltaic electricity, in any method, has no beneficial effect. Gymnastic exercises for the feet in the recumbent posture have been recommended, but they have not much influence on the steadiness of locomotion. Regulated steps in footmarks is more effective. Nerve-stretching and suspension, although once fashionable, have passed out of use as valueless. The attacks of pain and the crises are usually temporarily relieved, and often arrested, by antipyrine (15 gr.) or acetanilide (10 gr.). Hypodermic injections of the hydrochloride of cocaine are useful for superficial pains with tenderness of the skin. These may also be relieved by the application of chloroform sprinkled on lint covered with oiled-silk. The most severe attacks may, however, yield only to the hypodermic injection of morphine. Gastric

crises need treatment for any exciting cause, such as an attack of dyspepsia or constipation. Simple food must be administered, and the agents which relieve the attacks of pain may arrest the crises. In some cases morphine is necessary, by hypodermic injection. Laryngeal crises are usually relieved by nitroglycerine or nitrite of amyl. The condition of the bladder must be attended to most carefully. No hesitation should be felt in using the catheter as often or as early as is necessary, if the bladder is imperfectly emptied; and it should be washed out with a weak solution of boric acid if there is turbidity of the urine. The general hygienic conditions should be the best possible. Over-exertion and exposure to cold and wet should be carefully avoided. The course of the disease depends very much on attention to small details.

W. R. GOWERS.

TABES MESENTERICA (*tabes*, a consumption; and *mesenterica*, mesenteric).—An affection attended by marked wasting, and caused by abdominal tuberculosis, especially involving the mesenteric glands. *See* MESENTERIC GLANDS, Diseases of.

TACHE CÉRÉBRALE.—A term applied by Trousseau to a streak of hyperæmia, produced by drawing the nail over the skin (usually of the abdomen) in certain cases of cerebral meningitis. *See* MENINGES, CEREBRAL, Diseases of, p. 981.

TACHYCARDIA (*ταχύς*, rapid; and *καρδία*, the heart).—A form of functional disturbance of the heart, characterised by greatly increased frequency of action. *See* HEART, Functional Disorders of.

TÆNIA.—SYNON.: Tape-worm. *See* ENTOZOA.

TANGIERS, in North Africa.—*See* MOROCCO.

TAPE-WORMS.—*See* ENTOZOA.

TAPPING.—A popular name for paracentesis. *See* PARACENTESIS.

TARANTISM (*tarantula*, a ground-spider).—An epidemic dancing-mania, prevalent in Italy in the sixteenth and seventeenth centuries, originating in fear of the bite of the tarantula, as a remedy for which the dance was adopted. *See* ECSTASY.

TARASP-SCHULS, in Unterengadin, Switzerland.—Alkaline sulphated waters. *See* MINERAL WATERS.

TARTAR.—*See* TEETH; and CONCRETIONS.

TARTAR EMETIC, Poisoning by.—SYNON.: Fr. *Empoisonnement par l'Antimoine*; Ger. *Antimoniumvergiftung*.—Poisoning by tartar emetic, a soluble double tartrate of antimony and potassium, is not very common. The emetic properties of the salt generally ensure its speedy ejection from the stomach. Poisoning by the salt may be either (1) *acute*, from the ingestion of a large dose; or (2) *chronic*, the patient succumbing under the exhaustion consequent upon its prolonged administration.

1. **Acute poisoning**.—Shortly after taking a large dose of tartar emetic, the patient is seized with intense nausea and faintness, accompanied by depression of the force of the pulse, and increased perspiration. Violent vomiting and retching follow, with a burning pain and sense of constriction in the

mouth, throat, and gullet. Vomiting affords no relief to the nausea and pain, and is repeated, the vomited matters becoming bilious, and ultimately perhaps blood-tinged. A metallic taste is felt in the mouth; the abdomen becomes painful and tender; and profuse diarrhoea sets in, the fæces often containing a considerable quantity of blood. The urine is at first increased in quantity; but later it may be scanty, blood-tinged, or suppressed. The circulation is throughout depressed; and the skin cold, clammy, and bathed in profuse perspiration. The muscular system is relaxed; but cramps of the extremities may torture the patient. In rare cases a pustular rash appears, like that produced by the external medicinal application of the drug. In some cases neither vomiting nor purging has occurred, the symptoms being simply those of intense prostration, with embarrassed respiration. In fatal cases death occurs within a few hours.

2. **Chronic poisoning**.—The administration of repeated small doses of tartar emetic causes nausea, vomiting, purging, exhaustion, and debility, which not infrequently prove fatal. These symptoms are accompanied by depressed irregular circulation, profuse perspiration, and disturbances of respiration.

ANATOMICAL CHARACTERS.—These are somewhat variable, but on the whole are those of a metallic irritant poison. In most cases there is inflammation of the stomach and intestinal tract generally, not so patchy or marked by such bright redness as in arsenical poisoning. Not infrequently the stomach and small intestines escape, and the inflammation may be confined to the large bowel; even ulceration of the intestines may occur, accompanied by hæmorrhagic extravasations. Hypostatic congestion of the lungs is often prominent.

DIAGNOSIS.—Poisoning by tartar emetic may be diagnosed from that due to other irritants, especially arsenic, by the greater and earlier depression, the profuse perspiration, greater irregularity of pulse, and the more irregular respiration. An analysis of the ejecta or of the urine is always advisable, and often indispensable to complete the diagnosis. In chronic cases an analysis of the urine is the only satisfactory mode of determining the nature of the illness, where tartar emetic is not known to have been administered as a medicine.

PROGNOSIS.—This must always be grave, so long as the ejecta contain considerable quantities of the poison. In acute cases the patient cannot be considered out of danger till not only the vomiting has ceased, but an obvious return of strength has set in for some time.

Fatal dose.—Fifteen grains may prove fatal to an adult. Much larger doses may, however, as a rule, be taken with impunity. The danger is much increased if the poison be taken in conjunction with some substance, such as opium, which deadens the susceptibilities of the stomach.

TREATMENT.—The stomach-tube may be used, notwithstanding profuse vomiting. The elimination of the poison may be hastened by free administration of diluents, and the stomach protected by mucilaginous drinks, or the stomach may be washed out by means of the siphon-tube. The most effective antidote is tannic acid, which forms an insoluble tannate of antimony. For this purpose tincture of cinchona bark, decoction of oak-bark, or strong infusions of tea or coffee may be administered; or the stomach may be washed out with similar fluids, or with a solution of half a drachm of tannic acid.

Not till after this has been done, or the stomach well and repeatedly cleansed by free vomiting, should opium be administered. The after-treatment will depend upon the symptoms. The treatment of chronic cases consists in cessation of the administration of the poison; in the exhibition of ammonia, stimulants, and tonics; and in careful support of the strength. Nutrient and opiate enemata are of the greatest service.

THOMAS STEVENSON.

TASMANIA.—A warm, equable, subtropical climate. Mean temperature of Hobart Town, the capital, 54° F. Prevalent winds, N.E. and S.W. See AUSTRALASIA; and CLIMATE, Treatment of Disease by.

TASTE, Disorders of.—SYNON.: Fr. *Troubles du Goût*; Ger. *Störungen des Geschmacks*.

INTRODUCTION.—Disorders of taste have to be carefully distinguished from those of smell, on which all perception of flavour depends. The chief sensations of taste are those called sweet, sour, salt, bitter, and metallic. Flavours are sensations of smell due to an agent which reaches the olfactory mucous membrane through the posterior nares; their cause usually at the same time acts upon the nerves of taste, so that the flavour blends with the gustatory sensation, and seems like a part of taste proper. But if the sense of smell is lost, or even if the passage of air into the nasal cavities by the posterior nares is prevented, no flavour is perceived and taste alone remains. It is also necessary to distinguish disorders of taste from altered appreciation of the sensation—increased or diminished enjoyment of, or disgust at, sensations which are themselves normal.

The sensation is subserved by structures in the mucous membrane on the back of the tongue, the palate and the palatine arches, and to a slighter extent by those on the tip and edges of the fore-part of the tongue. On the last sweet and sour, on the former salt and bitter, are best perceived; but, except for individual peculiarities, all the sensations can be perceived in each region. The sense is related especially to certain ‘papillæ’ on the back of the tongue, but elsewhere to less differentiated structures of the mucous membrane. Substances must be in solution to excite the sensation, but the solution may be effected by the moisture on the tongue. A knowledge of these facts is important for testing the sense of taste.

In the front of the tongue the sensation is certainly subserved by fibres from the lingual branch of the fifth, belonging to this nerve, but reaching its root by a circuitous path—the chorda tympani, facial, and Vidian nerves, sphenopalatine ganglion, and thence to the fifth by the petrosal nerve. The nerve-fibres connected with the papillæ at the back of the tongue are those of the terminal branches of the glosso-pharyngeal, but disease of this nerve within the skull does not impair taste, and destruction of the root of the fifth nerve abolishes it, not only on the back of the tongue, but also on the palate. Apparent exceptions to this rule are probably due to imperfect destruction of the root, or to the disease of its fibres being within the pons. Hence it seems that the taste-fibres ascending from the back of the tongue leave the glosso-pharyngeal, just as those from the front of the tongue leave the lingual, and reach the root of the fifth by an equally

circuitous path, probably (from other pathological facts) chiefly by the tympanic nerve of Jacobson.

Within the pons, the fibres for taste seem to take a course separate from the proper sensory root-fibres, and lie at first near the motor fibres or nucleus, since the writer has met with complete loss of taste and paralysis of the muscles of mastication on one side, without anæsthesia, from disease within the pons. The path ascends to the opposite hemisphere of the brain, but the position of its cortical centre is not known.

1. Increased Sensitiveness of the Nerves of Taste.—SYNON.: Gustatory Hyperæsthesia; *Hypergeusia*.—This condition is manifested by detection of a substance too minute in quantity to be perceived by normal taste, or by an abnormally intense impression when a given quantity of a substance is tasted. In excitable states of the nervous system, as in general malnutrition, substances in small quantities in the blood are tasted with great readiness; the bitterness of morphine injected beneath the skin may be at once noticed (Wernich), and, for a long time after a bitter substance has been taken, whatever is tasted may seem bitter. Gustatory hyperæsthesia is often met with in hysterical persons, and sometimes in the insane. It is not usually a symptom of sufficient prominence to demand special treatment.

2. Perverted Sense of Taste.—SYNON.: Gustatory Paræsthesia; *Parageusia*.—This is not uncommon in neurotic states. Substances excite a different taste from that to which they naturally give rise. A bitter flavour, for instance, is detected in a simple saline. This condition is commonly conjoined with altered appreciation of the taste which is recognised, so that substances commonly considered pleasant are disliked, and those are enjoyed which commonly excite disgust. It is seen in a slight degree in some toxic conditions, but more frequently in psychical disturbances. In hysteria it leads to strange tendencies in diet. It is also met with, as a rare symptom, in some degenerative diseases, such as locomotor ataxy.

3. Subjective Sensations of Taste.—These may occur from disorders of the central nervous system, in hysteria, insanity, and occasionally as the aura of epileptic attacks. The sensation is usually of an unpleasant description, and is probably produced in the region to which the taste-fibres are conveyed by the branches of the glosso-pharyngeal nerve. It is remarkable (as evidence of the association of the cerebral centres) that sensations of flavour and of taste are combined in the epileptic aura, but a subjective sense of smell is always distinguished from these.

Subjective sensations of taste occur also, very rarely, from irritation of the gustatory nerves. They have been produced experimentally by galvanising the chorda tympani, when this has been exposed by disease of the internal ear; and have also occurred in some cases of disease of the petrosal part of the facial nerve. These subjective sensations have to be distinguished from abnormal sensations due to substances in the blood, or to secretions of the mouth. The treatment of the symptom is that of its cause.

4. Loss of the Sense of Taste.—SYNON.: Gustatory Anæsthesia; *Ageusia*.

ÆTIOLOGY.—Diminished sense of taste may be produced by: (a) Thickening or other changes in the mucous membrane of the mouth, rendering the

nerve-endings less accessible to sapid solutions. This is, however, a rare cause. (b) Local applications lessening the irritability of the nerve-endings. Hot or cold applications may temporarily destroy the sense of taste. Cool substances cannot be tasted so well as those which are warm. (c) Hysterical and other functional nervous disturbances. It then forms part of 'hysterical hemianæsthesia,' the unilateral loss of the special senses, accompanied by hæsthesia of the limbs and half of the trunk on the corresponding side. (d) A similar loss, in the same association, has been met with in rare cases of organic disease of the opposite hemisphere. (e) Disease of the nerves which conduct the sensation to the sensory root of the fifth nerve, especially of the tympanic plexus, in consequence of caries of the temporal bone. It is also often produced, in the front of the tongue, by disease of the chorda tympani, when a neuritis of the facial nerve passes up its canal.

SYMPTOMS.—Loss of taste involves the perception of bitter, sweet, sour, saline, and metallic qualities. These may be lost in varying degrees, sometimes entirely; occasionally some are more impaired than others. The loss, from disease of the nerves, may be on the whole or part of one side, according to the relation of the several nerves to the function in different parts of the gustatory region, as already explained. Each part of the tongue possesses the power of recognising every quality, but not in the same degree; bitterness and sweetness are appreciated chiefly by the glosso-pharyngeal at the back, acidity and saltiness by the lingual in the fore-part of the tongue, chiefly at the tip and edges. The onset of the defect may be sudden, as in hysteria; or gradual, as in most forms of nerve-lesion. It is usually unilateral; in rare cases both sides are involved. It is associated with other symptoms of loss of function of the affected nerves.

DIAGNOSIS.—Loss of taste has always to be carefully distinguished from loss of smell. All flavours are recognised by the olfactory nerve, and it is commonly assumed that when these cease to be perceived taste is lost. The power of tasting must be ascertained by powders or colourless solutions which shall convey no information. Citric acid, quinine, sugar, and salt, in powder or solution, answer well. The tongue must be held out, and the substance or solution placed on the part of the tongue it is desired to test, and after each observation the mouth must be rinsed with water. It must be remembered also that the anterior part of the tongue is almost destitute of the sense of taste, except on the edges and tip. If the loss is unilateral, the powder may be rubbed on the side of the protruded tongue, near the tip, with the finger, and the patient should indicate, by nodding or shaking the head, whether it is tasted, before the tongue is withdrawn into the mouth. The moisture on the mucous membrane is sufficient to dissolve the substance and enable it to act upon the nervous structures.

PROGNOSIS.—This depends on the cause, and the extent to which the morbid condition can be influenced by treatment. It is good in hysteria, less favourable in nerve-affections. The loss due to affection of the facial nerve is frequently recovered from, but may persist even when the function of the latter is recovered. In intracranial disease of nerves the prognosis is, as a rule, unfavourable.

TREATMENT.—The treatment in nerve-disease is that of the cause of the symptom. Stimulation of

the nerves in the tongue may possibly aid the recovery of function, and for this faradisation may be employed. Hysterical loss of taste, part of hemianæsthesia, is best treated by neglect. Where the loss depends on an affection of the mucous membrane of the tongue, local measures alone are effective, in so far as the treatment can be influenced.

W. R. GOWERS.

TEETH, Diseases of.—**SYNON.** : Fr. *Maladies des Dents*; Ger. *Krankheiten der Zähne*.

The teeth fulfil two principal functions: they serve for articulation and for the mastication of food. The loss of the teeth leads to many forms of dyspepsia, and it must be remembered that the loss of even some of the teeth may, through throwing their opponent teeth out of work, render inefficient a set of teeth which at first glance might appear to be adequate for mastication. The restoration of the missing teeth will often bring about the cure of dyspeptic troubles which diet and drugs have failed to influence. Elaborate cooking, while rendering the food of such consistence that it may even without much mastication be accessible to the gastric juice, has the effect of rendering its sojourn in the mouth too short for the action of the salivary ferments to be complete, and has the further indirect effect of giving the teeth too little work for their well-being. Teeth which are not subjected to the normal amount of attrition are more prone to decay, so that it has even been contended that a principal factor in the causation of dental caries is the removal of all hard and fibrous elements from the food. Moreover, disease of the sockets of the teeth is more apt to occur when they are in comparative disuse, while the self-cleansing action of the mastication of hard foods diminishes the pabulum left about the interstices of the teeth, and hence has a beneficial effect in diminishing the numbers of micro-organisms which swarm in a dirty mouth. This is exemplified by the clean and healthy condition of the teeth in wild animals and in the lower races of mankind, notwithstanding that the latter do not use a tooth-brush. But among civilised people, consuming much softened food, the use of the tooth-brush, and indeed of some form of antiseptic wash or tooth-powder, is indispensable in order to keep the mouth in clean condition. No degree of care attainable in everyday life will render the mouth sterile, but the number of micro-organisms in the mouth can be immensely reduced by proper care.

A perfect set of teeth should be typical in form, in which case the points of contact of contiguous teeth are rounded and small in area, and the colour a yellowish-white; they should not look very transparent, and pearly: transparent, bluish-white teeth appear to be specially liable to early and rapid decay. It was formerly supposed that such teeth were deficient in calcium-salts, but, so far as the dentine at all events is concerned, this has been shown not to be the case. The enamel, owing to the difficulty of isolating it in sufficient quantity, has not been investigated with the same completeness, and it is possible that differences of chemical constitution may exist in this, the first line of defence. All that can safely be said is that there is no known difference in the chemical constitution of good and bad teeth, though they can be recognised by the eye.

Defects in the form of teeth are very common and predispose to caries; they may be of the nature

of pits almost or completely going through the enamel, in which case they afford a lodgment for particles of food. Other defects take the form of lines marking a check of growth at some period, somewhat like the similar lines which are seen upon the nails. If the history of the child be inquired into, it will generally be found that they correspond with the stage of development of the tooth appropriate to the age at which some acute illness occurred. According to some authorities they are invariably due to rickets. Stomatitic affections, especially such as have been due to the administration of mercury, leave their mark upon the teeth in the form of an irregular, craggy, or honeycombed condition of the enamel; and the poison of inherited syphilis produces a peculiar form of tooth, to be presently more exactly described, but which for the present may be designated as a stunting of the portions first formed. Teeth, from their exposed position, are subject to accident and to irregularities of position, which are not uncommonly due to a disproportion between the size of the teeth and of the jaws, it being a fact that the jaws are more readily affected and more subject to variation than the teeth, as is well exemplified in short-muzzled breeds of dogs, whose teeth are almost always crowded.

1. Absorption of Permanent Teeth.—A process similar to that by which the milk-teeth are shed may be set up on the roots of permanent teeth and lead to their loss. A tooth which has been knocked out and replanted is very apt to be ultimately shed in this way, and teeth, the roots of which have long been the subject of slight irritation, are apt to be more or less absorbed, though this rarely happens with otherwise sound teeth. The pressure of an erupting wisdom-tooth will cause absorption of the root of the tooth in front of it, and an erupting canine may cause damage to a lateral incisor in the same way. The only symptoms are the loosening of the teeth and some tenderness on vertical pressure, due to the sharpness and roughness of the root; nothing can be done in the way of treatment.

2. Accidents to the Teeth.—A tooth which has been knocked out will usually, if cleansed and replaced in its socket, become again fixed. Under these circumstances the cementum appears to remain alive, though the pulp is killed. Hence a tooth knocked out should be well washed in boiled water or in a weak antiseptic solution and replaced; temporary support may be afforded to it by tying it to its neighbours or by moulding some gutta-percha round it.

3. Bacteriology of the Mouth.—Warmth, moisture, and the presence of abundant pabulum in the form of the saliva and buccal mucus, as well as of introduced foods, render the mouth an excellent culture-chamber for bacteria, while its exposed position and the frequent introduction of uncooked material supply it with a great variety of forms of micrococci and bacilli. The nooks and crannies in the interstices of the teeth furnish places where they can flourish with little disturbance, while the constant removal of their products by the swallowing of the saliva prevents any interference with their free development. Hence it might have been expected, as is the case, that a great variety of forms, some of which go on to the formation of zoogloea-masses and others to the formation of thread-forms and masses of spores, have been de-

tected in the mouth. Some difficulty attends their investigation, as a good many of them have not been successfully cultivated, and are hence not known in pure culture; the identification of these therefore rests on the unsatisfactory basis of morphological character only, and their biological characters are unknown. Hence it is possible that some of the filamentous forms, such as *leptothrix*, may be stages in the growth of other described forms. Some half-dozen forms occur in almost every mouth, and recently Goadby has succeeded in cultivating some which others had previously failed to grow. These all grow best on an alkaline medium containing gelatine; those which liquefy gelatine do not always liquefy the decalcified dentine-matrix, but those which liquefy blood-serum generally do so; these organisms are mostly anaërobic and work in an alkaline medium only. This being the case it is pretty clear that the initial stages of dental caries, when decalcification is the prominent feature, and its subsequent stages, when the whole tissue breaks down, are either not accomplished by the same organisms, or at least are not affected by the organism working under similar conditions; hence it is not surprising that no specific organism of dental caries has been discovered, and that it is generally believed that many different organisms may be able to cause it. Besides the organisms more or less peculiar to the mouth, over a hundred different forms have been found in it, some of which are pathogenic and some not. It would be out of place to enter into any detailed account of the organisms of the mouth here, but it may be mentioned that although no mouth is free from organisms, yet they are enormously more abundant in unhealthy and ill-kept mouths, in which sundry vibrios of 'comma' and spiral form are always present. It is certain that dental caries is dependent upon the existence of micro-organisms, and it is in the highest degree probable that *pyorrhœa alveolaris* is due to bacterial infection; hence the two commonest diseases of the teeth owe their origin to infections of this kind. Apart from the bacteria directly concerned in the causation of dental disease, the principal pyogenic bacteria are often met with, even where no suppurative affection appears to exist, such as the various kinds of *Streptococci* and *Staphylococci*; and, with less frequency, species of *Sarcina* have been found in the surface-layers of carious dentine. Comma-bacilli, like but not identical with Koch's cholera-bacillus, are often seen, and pneumococci are also common. Hence there is much to favour the idea that the mouth is often the source of otherwise inexplicable infections, and it is therefore of great importance that the mouth and teeth should be kept in such condition as to minimise the risk of their forming a focus of infection (*see ANEMIA, PERNICIOUS*, p. 55). Suppurations in connection with the teeth are exceedingly common; carious dentine appears to form a pabulum for many bacterial forms, and there is a growing belief that infection from the mouth is the cause and the means of keeping up certain gastro-intestinal troubles of an intractable character, while it is more than suspected that many other infections, the origin of which has been obscure, have come from the mouth. Actinomycosis of the lung has been distinctly traced to infection from the mouth, and septic pneumonia is a common sequela of surgical operations on the jaws and tongue. And as pathogenic bacteria are frequently in the mouth, quite independently of any operation

wound, it is reasonable to suppose that they occasionally find their way to the stomach or the lung. Thus, symptoms attributed to carcinoma of the stomach have been known to disappear with the cure of pyorrhoea alveolaris, a condition in which small quantities of pus loaded with organisms are constantly being swallowed; and Godlee records cases of this disease in which mistaken diagnoses of serious lung-disease have been made. He also records a case of acute glossitis, apparently due to infection from this disease. Beside these cases in which the connection is well proved, it is by some believed that malignant endocarditis, the recurring attacks of putridity in bronchiectasis, and other infections difficult to explain, may turn out to have their origin in the mouth.

4. Dental Caries.—This disease has no relation to caries of a bone, and so is in this respect misnamed. It is exceedingly common, the milk-teeth rarely escaping entirely, while there are few adults in whom it is not found in some degree. It invariably starts from the outside, generally on the enamel-covered portion of the tooth, and progresses inwards with increasing rapidity and spreads wider when once the enamel has been penetrated. Occasionally on exposed positions, where the softened portions are rapidly worn away, a spontaneous cure occurs, and a hard brown polished surface is left. This is termed *arrested decay*, and in it the dentinal tubes appear to be blocked up, perhaps by calcification of their contents. A similar result may sometimes be attained in suitable positions by cutting away the affected parts and thoroughly polishing the surface. Various histological changes occur, into which it is out of place to enter here; but it may be mentioned that, not only is the disintegrating material loaded with micro-organisms, but micrococci and short bacilli penetrate the dentinal tubes to a considerable distance beyond the area which shows any naked-eye changes. The first step in the process is decalcification, the lime-salts being dissolved by acids; these acids are furnished by fermentations leading to the production of lactic and other organic acids, and for their formation there are required fermentable material and the bacteria capable of setting up these fermentations. Subsequently the decalcified organic matrix, the collagen, is softened and broken down by a species of peptonising action; but whether prior to its liquefaction it undergoes conversion into gelatine is not known, though this would appear probable. No specific bacteria of caries have been discovered; it is probable that many different forms are capable of producing it. From this it will readily be understood that experiments in the production of artificial caries in a flask fail completely if the fluid be sterilised; but if it be not sterilised the process can with proper precaution be very completely imitated. And although the mouth cannot be kept sterile, the nearer it approaches to this state, and the less food remains lodged in it, the less will the teeth be attacked by caries. A curious feature of dental caries is that it is very rife in young and adolescent persons, but that during middle life it is comparatively easily controlled; in aged persons, however, it not infrequently again becomes rife.

ÆTIOLOGY.—The *predisposing causes* are local and general. *Local.*—The most important of these are defects of structure, such as deep pits, irregular or crowded disposition of the teeth leading to

lodgment of food and want of cleanliness; abnormal condition of secretions of the mouth, such as scanty secretion of saliva, met with in various states of ill-health, and very notably in fevers; and unsuitable diet—for instance, prolonged sucking of sweets leads to a gummy deposit on the teeth, probably due to mannite, a product of the fermentation of cane-sugar. Dyspeptic conditions probably influence the teeth locally by vitiating the secretions of the mouth; with a furred tongue one often sees the saliva stringy, that is to say, the mucus is in excess of the watery secretion, and this has a very damaging effect by interfering with the cleanliness of the mouth.

General.—Though the precise connection is not at present understood, certain general constitutional conditions are associated with abundant decay of the teeth. Thus during pregnancy the teeth often suffer more than at other times, and it is observed that markedly anæmic women rarely escape, though here the question may arise whether the state of the teeth and the abundant micro-organisms which are being continually swallowed may not be the cause of the anæmia (*see p. 55*). At all events, putting the mouth in order by the extraction of very bad teeth and the filling of others often has the most beneficial result upon the anæmia, even where the teeth had been previously fairly adequate for mastication. The teeth of patients placed upon a milk-diet on account of acute dyspepsia also suffer markedly, though here again there is room for doubt whether the disuse of the teeth and the supply of very suitable pabulum for the growth of bacteria may not be the real cause rather than the dyspepsia. It is also very doubtful whether such disorders as gout, rheumatism, diabetes mellitus, and tuberculosis, &c., so influence the teeth that ill-effects can be attributed to them, at all events otherwise than by the interference with digestion, and so with the oral secretions. It is also open to doubt how far bad teeth are inherited; there is a strong general impression that they are, but on the other hand, the environment is likely to be similar in the same family, and the upsetting of the old idea that teeth liable to early decay are, so far as the dentine is concerned, less highly calcified renders it more difficult to see how inheritance affects them. But it must be remembered that our knowledge is very incomplete on this point; thus it is possible that the enamel may be different in good and bad teeth; and that the lime-salts, while in the aggregate of the same amount, may be in different combinations. Of the organic matrix, also, practically nothing is known save that it is collagen. It is an over-statement of the case to say that there is no difference in chemical composition between good and bad teeth, though it is true that none has been demonstrated. *The exciting causes* have already been alluded to. They are comparative disuse leading to a loss of natural cleansing, or the presence of acid-forming bacteria and their appropriate pabulum and the lodgment of particles of food.

Acid medicines are incapable *per se* of initiating caries, but they may do much mischief by superficial action on the enamel, whereby it is roughened and bacterial plaques the better enabled to adhere, while the decalcifying process is thereby commenced.

Caries invariably commences from the outside; when it has penetrated the enamel it spreads

rapidly along the direction of the dentinal tubes, but it is also able to spread laterally, and always does so as it approaches the pulp. Some attempt at defence is frequently made, especially when the progress is not very rapid, by new deposits of secondary dentine on the threatened side; but this is rarely of much avail unless the caries is arrested by other means. Rapid caries leaves the softened dentine almost white; whereas when its progress is slow the dentine takes on a brownish discolouration, which is believed to be due to pigment-forming bacteria rather than to any form of decomposition of the organic matrix. Carious dentine has a peculiar odour, which has been compared to that of gangrene of the lung, or to the scent of the little neuropterous insect, *Chrysopa*. If it be not checked the pulp becomes infected, exposed, and inflamed, often ultimately dying in consequence.

TREATMENT.—Prophylactic treatment has already been indicated, and will be further dealt with under the head 'Hygiene of the Mouth' (see p. 1669). Remedial treatment consists in the removal of the carious material and the filling of the cavity with some suitable material, except in the few cases where complete removal and polishing of the residual surface is feasible. As caries is dependent on external causes, a small amount of carious dentine may sometimes be left in the floor of the cavity, if it be well soaked with an antiseptic and thoroughly sealed in, rather than run a risk of exposing the pulp in its removal.

5. Enamel, Pitted. — **SYNON.**: Mercurial Teeth (Hutchinson); Stomatitic Teeth; Honey-combed Teeth.

It is of great importance to distinguish between syphilitic teeth and other malformed teeth which have no similar signification. Much unnecessary distress has been occasioned by confounding teeth having rocky and pitted enamel with those which were truly syphilitic in their origin; and many persons supposed themselves to inherit syphilis, who merely possessed teeth bearing marks upon them which registered a temporary illness or a condition of depressed nutrition in childhood when the affected teeth were forming. Teeth with rocky and pitted enamel vary indefinitely as to the extent of their defective formation, from a slight horizontal grooving in the enamel, to a condition in which the whole surface is rugged, and studded with pits like a thimble. In extreme cases the enamel may be almost entirely wanting; but there is *no narrowing of the apices of the crowns of the teeth, and no crescentic notch in the superior incisors*, as in syphilis. The defects of the enamel are nearly always horizontal in their disposition; and even the pits have such an arrangement in series. This condition is most frequently seen in the permanent teeth, though sometimes in the temporary. It is most manifest in the first molars, the incisors, and the canines. Rarely it affects the bicusps, near the apices of the cusps; and still more rarely the whole bicuspid crown suffers. But it will be observed that the malformation is symmetrical in the corresponding teeth, and that in the different teeth it has occurred at a point in its development which each tooth had attained at one particular date; and probably the mildest and severest cases are essentially the same in their pathological meaning, the difference being only one of degree. It has been supposed by Hutchinson that this condition of teeth is the result of the

constitutional influence of mercury given in childhood; but it is extremely rare for mercury to affect the mouth in children, and it occurs in persons not known to have been treated with mercurial teething-powders. The microscopical structure of teeth, thus degenerated, shows that the condition is essentially one of imperfect calcification of the enamel; and any influence or disease of childhood, suspending for a time or depressing the nutrition, may quite possibly be an efficient cause.

6. Erosion.—Erosion is the name given to the process by which saucer-shaped cavities or grooves are formed on the labial aspects of the necks of the teeth. They are generally placed transversely; and though at times attacking all teeth, are most frequently seen on front ones. The surface of erosion is hard, polished, and often not at all discoloured until the pulp is approached, wherein it differs from caries; and the teeth attacked frequently become so weakened as to break off. Erosion seems to be due to a chemical solvent, the source of which is probably the gum-margins.

Some doubt remains as to its ætiology; it may be a softening due to the same agencies as the commencement of caries, attrition constantly removing the softened portions; and it has been suggested that it may be due to some agent which primarily attacks the organic portion, leaving the lime-salts free to be rubbed away.

TREATMENT.—The cavities due to erosion should be filled if deep enough; otherwise the sensitiveness generally present should be treated by nitrate of silver in the form of the solid pencil, which, however, discolours the dentine deeply, or by chloride of zinc.

7. Eruption of Wisdom-Teeth, Difficult.—Insufficiency of room in the jaw for the advent of the wisdom-teeth is sometimes attended by very painful and even serious symptoms, especially in the lower jaw. The wisdom-tooth remains impacted at the base of the ascending ramus of the jaw, growing and pressing against the second molar. This gives rise to inflammation and pain, wandering down the neck and arm, the latter being often weakened in muscular power. In acute and neglected cases, abscess forms at the angle of the jaw, and burrows about the cheek. One remarkable symptom is trismus, which is very usual in these cases, and is characteristic; the masseter muscle becoming contracted and firmly set, so that the jaws can hardly be opened a quarter of an inch. This locking of the jaw sometimes occasions very unnecessary alarm.

Reflex nervous symptoms are sometimes occasioned by the resisted eruption of the lower wisdom-teeth, and cured by removal of the cause of irritation. Upper wisdom-teeth occasionally give trouble by growing into and causing ulceration of the cheek.

TREATMENT.—The treatment depends much on the degree of obstruction. Excision of the flap of ulcerated gum which overlies the wisdom-tooth is occasionally sufficient to relieve the symptoms; otherwise removal of the wisdom-tooth is the proper cure. Where there is trismus, it is necessary to force open the jaws with a gag under an anæsthetic, and then extract the tooth if possible. If it cannot be reached, the second molar should be extracted. The relief to pressure thus afforded will often give complete relief, though if there be much suppuration in association with an obstructed wisdom-tooth, the

removal of that tooth is usually advisable. The trismus vanishes on extraction of the tooth.

8. Extraction of Teeth.—In these days it might hardly appear necessary to say that forceps should be sterilised either by boiling or by dipping their jaws into strong carbolic acid, yet many who would not think of using any other surgically dirty instrument are careless about tooth-forceps, notwithstanding that several fatal cases of septicaemia have been traced almost certainly to dirty forceps. If a little concentrated carbolic acid remains upon the jaws of the instrument it does no harm. It is a matter of everyday observation that the more the bone of a socket is damaged in an extraction, the more do subsequent inflammatory conditions prevail, so that this should be avoided as far as possible; the interior of the socket is filled up with and protected by clot, but any lacerated portions of the edge of bone denuded of their gum-covering rarely heal kindly.

9. Hæmorrhage after Tooth-Extraction.

When a tooth is extracted the nutrient artery of the pulp is torn across and usually soon ceases to bleed. When troublesome hæmorrhage ensues it is commonly from this vessel and not from those of the socket that it proceeds. Primary hæmorrhage is that which commonly occurs, and if there be no great damage done to the bony walls of the socket, is rarely difficult to arrest. Very hot or very cold water generally stops the bleeding at once, or failing this, temporary arrest of the bleeding by pressure of the finger over the open mouth of the socket often succeeds by giving time for a clot to form. Failing this, a tent of matico-leaf rolled up rough side outwards and placed in the socket of each root assists in the formation of a firm clot, but it may sometimes be necessary to pack each socket firmly with graduated compresses of wool dipped in styptic colodion or some other astringent, or the tooth itself may be used as a plug. The objection to using any form of plug is that the natural process of healing is greatly retarded. If the sockets be much damaged there may be a difficulty in making an efficient plug. The routine use of applications such as perchloride of iron is much to be deprecated; if there be any strong hæmorrhagic tendency a much larger bleeding surface is thus created. If these measures fail, a firm compress should be placed over the plugged sockets and pressure made upon it by means of the opposing teeth, which may be maintained by the help of a four-tailed bandage. The patient, even if faint, should take no alcohol nor any hot drink; a very hot foot-bath is of service, and in some cases an emetic, by its depressant effect, arrests obstinate bleeding. Hæmostatics may be given internally, but, as a rule, are inefficient. If it become necessary to extract a tooth for a patient in whom hæmorrhagic tendencies are suspected or known to exist, it is safer to plug the socket at once without waiting; in one such case the writer had no trouble with the tooth though the patient nearly lost his life from epistaxis, which came on immediately after recovery from nitrous-oxide anaesthesia. A few fatal cases have occurred in which the difficulty has been greatly increased by caustic applications; the actual cautery is of little use, as it is impracticable to reach the bleeding point at the bottom of the socket. *See p. 617.*

10. Hygiene of the Mouth.—The established fact that dental caries is dependent upon micro-organisms, and the growing belief that other infec-

tions may be due to the micro-organisms of the mouth, render it important that intelligent care should be given to keeping the mouth in a condition as pure as possible. The use of foods which require considerable mastication tends in this direction, and in so far as elaborate cooking defeats this end, the tooth-brush must take its place. A tooth-powder should contain some soap, or a soap may contain a polishing powder. In addition there should be some antiseptic which is efficient, and at the same time is not injurious to the teeth, nor to health if a little be swallowed. These requirements are fairly well fulfilled by benzoic acid and saccharin, but many other substances might be employed, such as thymol and the various essential oils. Where the form of teeth is such that particles of food lodge easily, they should be removed either by drawing silk between the teeth or by the use of a wooden or quill toothpick. Nothing conduces so much to the growth of organisms and the occurrence of caries as the lodgment of food, and antiseptic washes fail to reach the matter packed in between teeth. And in mouths where there is any suppurating an antiseptic mouth-wash should be freely used several times a day. Mercuric chloride is too unpleasant and too poisonous for everyday use in the mouth, but experiment has shown that by its use the nearest approach to a sterile mouth has been obtained. Hence for special purposes, such as the preparation of the mouth for any operation, it should be employed in the strength of 1:2500. It need hardly be said that no untreated carious teeth should be allowed to remain, nor should any suppurating stumps. *See also p. 209.*

Too little attention is paid to the condition of the mouth during acute disease. At such times a diet requiring no use of the teeth and vitiated secretions call for more than the usual amount of care. Nurses should be instructed, in every case in which it is practicable, to brush the patient's teeth with a soft tooth-brush, and to use a mouth-wash for the purpose. An agreeable one consists of Thymol, gr. iij; Benzoic Acid, ʒss; Oil of Wintergreen, ʒss; Sp. Vini, ʒiij, enough to be dropped into a wine-glass of water to make it quite cloudy. Condy's Toilet Fluid much diluted is also often very refreshing when the patient's mouth is dry and an unpleasant taste is present.

11. Anomalies in Number, Shape, and Position of Teeth.—These are for the most part of interest to the dentist only. Total deficiency of teeth sometimes occurs, the subjects commonly having other abnormalities, such as hair all over the face and body, or sometimes partial deficiency of hair, or imperfect development of nasal bones, or of the external ear. Individual teeth are sometimes absent, the lateral incisor being the one most frequently suppressed, and this peculiarity sometimes occurs in so many members as to become almost a 'family mark.' Occasionally also a temporary tooth is retained and it becomes a question whether it should be extracted or not. The existence or non-existence of its permanent successor can often be determined by a skiagram. Excess in number is also met with, the added teeth being termed supernumerary teeth; they are usually abnormal in form and furnished with imperfect roots.

Wisdom-teeth seldom conform exactly to the form typical of the human molar, at least in the higher races of mankind, and together with their irregularity of form there is great uncertainty in the

date of their eruption. Hence it is argued that the wisdom-tooth is gradually disappearing in the higher races of mankind.

Anomalies in position are very common and are more often due to overcrowding than to any other cause, the growth of the jaws being inadequate to provide space for the teeth to occupy a regular arch. Apart from the disfigurement the crowding tends to the lodgment of food and so to the decay of the teeth, so that it should never be neglected. In children with 'adenoids' the habitually open mouth seems to lead to a transversely contracted arch so that the range of the teeth is more or less V-shaped, and thumb-sucking through continued pressure leads to a deformation of the jaw in which undue prominence of the front teeth is the most conspicuous effect. See p. 716.

12. Loosening of Teeth.—See 16. Periosteum, Diseases of Dental.

13. Necrosis of the Jaw and Teeth after the Eruptive Fevers.—Necrosis of the jaw and teeth is among the secondary maladies which are apt to occur after small-pox, scarlet fever, and measles in children, and the cases are all singularly alike. They usually occur between the third and eighth years, and the severity of the previous attack of fever is immaterial. The local symptoms, which usually appear from three to six weeks after the primary disease, consist in a separation of the gum around one or more of the temporary teeth; and this continues until the bare jaw is exposed to a depth which corresponds not only to the temporary teeth, but to the bony capsules of their immature successors. Transverse ulceration then usually follows; and the temporary teeth, their alveoli, the immature permanent teeth, and their bony capsules are shed. Frequently this occurs on both sides of the mouth symmetrically. There is no swelling or formation of supplemental bone. In the lower jaw, the base of the bone is rarely, if ever, involved; and the consequent disfigurement is singularly slight. These exfoliations occur much more frequently after scarlet fever than after measles; and they are rare after small-pox. Limited necrosis occurs sometimes in consequence of alveolar abscess, and sometimes thin flakes of the alveolus overlying the front teeth necrose without apparent cause. More severe necroses also occur when arsenical dressings have escaped from the tooth-cavity.

TREATMENT.—The treatment of these cases should consist in as little local interference as possible. Little need be done, beyond attention to cleanliness and deodorisation, and the removal of the sequestra when quite loose. The general condition of the patient will require careful treatment.

14. Phosphorus-Necrosis.—Workers in match-works are subject to necrosis of the jaws, sometimes of great severity. The accepted idea has been that they are only in danger of this when there are carious teeth, it being supposed that the poison obtains direct access to the bone through the tooth-cavity. There can be no doubt that it is important that the teeth in lucifer-match workers, notably where ordinary phosphorus is employed, should be carefully attended to, but it is open to doubt whether the common explanation of the causation of the lesion is correct. There is not the smallest reason to suppose that the existence of a carious cavity, before the pulp has become involved, can be a means of access; it is only when the pulp has become involved and is dying or dead that it can

be important. And in the majority of cases which have been sufficiently well recorded, the mischief has only happened when teeth were dead and suppurating. As there are cases of phosphorus-necrosis occurring long after the patients have ceased to work in match-factories, and as there is some evidence to show that in persons long exposed to the fumes of phosphorus (it being probably the lower oxides which are the most mischievous) other bones in no way exposed get affected, it seems probable that the frequent incidence upon the jaw-bones is rather to be explained by the supposition that the poison induces a general condition of pre-disposition to periostitis and necrosis, while the jaw-bones being through the medium of septic teeth infinitely more frequently affected with periostitis than any other bones naturally become the usual site of this form of necrosis. The treatment is to be conducted on general principles, premature interference being especially harmful. See p. 1113.

15. Nervous disorders caused by the Teeth.—The most frequent and the most familiar of these is pain of neuralgic type referred to spots at a distance from the teeth, and unaccompanied by localised pain in these organs. Thus pain may be felt in the eye, in the region of the ear, in the supra-orbital region, or even in the neck and upper arm; and this pain may come and go at definite times in the day, being temporarily cured by a meal, being induced by extra fatigue, and in fact behaving precisely like what we term a neuralgia. Hence a pain that is relieved by quinine and not felt in any tooth may nevertheless be due to disease of the teeth. The condition which is usually found in such cases is a very local but intense inflammation of the pulp of one of the teeth; when the inflammation of the pulp is more widespread local pain is seldom absent (see p. 1671). Pain of a neuralgic type may also, but less commonly, be produced by other causes such as exostosis of the roots of teeth, while inflammatory affections of the roots of teeth generally cause a distinctly local pain. Paroxysmal neuralgia of excessive severity, the 'tic' of older writers, is rarely, if ever, due to diseases of the teeth.

Besides pain other reflex disturbances may sometimes be traced to teeth. Lachrymation on the affected side, blepharospasm, and even amaurosis have been observed, and such severe nutritional disturbance as glaucoma has been attributed to the same cause. Muscular spasm in the form of trismus has been alluded to in connection with the eruption of wisdom-teeth, and the writer has seen strong and very painful spasm of the muscles which open the jaw induced by closure of the mouth making contact with an exceedingly tender spot; the spasms continued for some time afterwards.

Nerves may become involved by direct extension of inflammation from the teeth; thus the facial nerve has been partially paralysed by inflammation extending from an upper wisdom-tooth; the inferior dental nerve has been similarly involved in its bony canal, and the nerves passing through the upper jaw have been affected: these cases are rare.

16. Periosteum, Alveolar Dental, Diseases of (Periostitis).—SYNON.: Periodontitis; Pericementitis.—Mechanical injury, such as blows upon the teeth, and in rare cases general conditions probably of rheumatic character, may induce periostitis, in this latter case affecting many or all of the teeth simultaneously. Mercury also will cause it if pushed too far, and it is sometimes observed in

tertiary syphilis independently of the administration of mercury.

In the vast majority of cases, however, dental periostitis is septic and is due to infection through the apical foramen by which the vessels and nerves enter the tooth-pulp. It is associated with a dead or nearly dead tooth-pulp, which is always crammed with organisms of a highly virulent character. The periosteum has some little power of resistance, so that when infected it by no means always runs on to suppuration, if early treatment is undertaken. If the septic material is not afforded free exit and antagonised by antiseptics, the inflammation spreads widely through the tooth-socket, while suppuration occurs at the apex of the root which becomes detached and lies in a small abscess; if the tooth is extracted at this stage the abscess-sac comes away with the root. If the root-canal is not sufficiently patent to give exit to the pus, the overlying bone becomes perforated and the pus finds an outlet between the alveolus and the lip or the cheek, or sometimes makes its way to the outer surface and points on the face.

In the earlier stages the tooth is elongated and tender, though gentle continued pressure often affords relief, and the margin of the gum is marked by a red line. In the later stages the pain is severe, especially at night and in the recumbent position, and does not abate until the pus has found an exit from the bone. Then external swelling appears, and with its appearance the pain usually moderates, unless the case is of exceptional severity. A chronic gumboil which provides free exit for any pus that may form is not necessarily painful nor is the tooth always tender. In a few rare instances the infection has been so intense that diffuse osteitis is set up with consequent necrosis; and even fatal cases occur. These fatal complications take the form of septicæmia, pyæmia, erysipelatous inflammations of the face and neck, or extension of inflammation through the foramina at the base of the skull to the meninges.

TREATMENT.—Cases of simple periostitis, whether due to injury or to constitutional conditions, do not run on to suppuration. Rest to the affected teeth, slight counter-irritation such as the application to the gums of equal parts of Tincture of Iodine and Fleming's Tincture of Aconite, warm lotions and the like, are generally sufficient to give relief. When constitutional conditions exist they should be appropriately treated. Iodide of Potassium in a few full doses is often of great value in general dental periostitis, although it itself is capable of actually causing a similar condition when its use is prolonged. In septic periostitis the first thing to be done is thoroughly to open up and disinfect the offending tooth, taking especial care that the root-canals are cleansed and pervious to their very ends; and if there is reason to suppose that any pus has already been formed the tooth should be closed but very lightly, so that there may be drainage. For this disinfection peroxide of hydrogen is one of the best agents, or perchloride of mercury may be used. So soon as it is found that the inflammation has subsided and the interior of the tooth is clean and aseptic, the root-canals and the cavity may be filled. The tooth is then harmless, and although the pulp is gone and the dentine dead, the cementum remains alive and has healthy periosteum attached to it. Such a tooth must not be filled (unless there be a fistula on the gum) till

all suppuration has ceased, and the success of the operation depends on the completeness with which the roots can be disinfected and filled; with teeth with large and straight roots the percentage of success is very high. If inflammation has extended far beyond the immediate surroundings of the tooth, and does not yield to treatment promptly, it may be advisable to extract it.

A chronic abscess-sac on the root of a tooth sometimes becomes apparently cystic and contains cholesterolin. Although the cementum of a 'dead' tooth, i.e. a tooth of which the pulp is dead, usually remains alive, it also may die, and the tooth is then described as 'necrosed'; if not extracted it will speedily be expelled by suppuration. This result has been known to follow an arsenical dressing if too large a dose be used, or if it be left too long in the tooth after the destruction of the nerve has been accomplished. The periosteum may become detached from a portion only of the cementum, the rest remaining alive and healthy; such teeth may be long retained.

17. Exostosis of the Cementum.—The normal cementum on a human tooth is quite thin, but it is subject to subsequent deposits which may thicken it. This may occur on sound teeth as well as upon those which are diseased, and may take the form of nodular excrescences, or may be generally distributed. The bony socket is absorbed to make room for it, and it may lead to the fusion of the several roots which were originally distinct or may even unite the roots of two contiguous teeth. A peculiarity of the process is that it is generally intermittent, periods of absorption alternating with those of deposition, the latter preponderating. In slight degree it gives rise to no inconvenience; but when considerable, the tooth may become tender to percussion, and may give rise to pain not very distinctly located, and even to considerable neuralgia. Sometimes the enlargement can be felt through the gums, but it is often impossible to diagnose it with certainty except by the help of a skiagraph (which, however, is by no means always conclusive), until one or more teeth have been extracted and seen. A good many of the teeth are usually affected, often successively, and nothing can be done in the way of remedy save extraction. But a dead tooth which has repeatedly been the subject of slight degrees of inflammation is usually the site of more or less exostosis, and this would be no indication that others are likely to be affected.

18. Tooth-pulp, Inflammation of.—When the pulp is actually exposed, or sufficiently nearly so to be infected by organisms, inflammation speedily ensues. This may be limited to a small area, in which case pain may be absent, slight or severe, intermittent or neuralgic in type; or it may involve the whole pulp, in which case pain is always severe, throbbing, aggravated by changes of temperature, or by anything which increases the blood-pressure, such as dependent position of the head. When cold water held in the mouth gives relief, and the pain returns as soon as the tooth warms up again, severe inflammation which will soon result in the death of the pulp *en masse* may be diagnosed. The tooth-pulp is singularly devoid of recuperative power: when once it has ached severely there is no chance of its ultimate recovery to health, and the sooner it is killed the better for the patient's comfort. The death of the pulp is generally sudden; a violently aching pulp ceases to ache

almost abruptly, and this cessation is coincident with thrombosis of its nutrient vessels. A period of freedom from pain then ensues during which decomposition of the pulp takes place, toxic products and organisms escape from the apical foramen, and periosteal inflammation and ultimately gumboil follow. These sequelæ may not take place if the opening into the pulp-cavity is sufficiently free to allow of the escape of gas and other liquid products by that route.

Acute general inflammation of the pulp may also be caused by changes in temperature conducted through metallic fillings which are too close to it without any actual exposure having ever existed.

TREATMENT.—Pure carbolic acid or creosote applied on a pledget of wool to the exposed spot will often give temporary relief, if the inflammation be not of the most violent character, but the nerve should as soon as possible be destroyed with arsenious acid, of which about a sixteenth of a grain is sufficient; if the nerve be left to die of itself there is much more risk of septic infection of the periosteum, but the application of arsenic requires skill and care. It should be very exactly applied to the point of exposure, as if it gets to the pulp imperfectly it causes unnecessary pain by further irritating it without speedily killing it. If any of it escapes on to the gum it causes sloughing and even exfoliation of portions of the edge of the socket. It should not be left in more than two days, when the dead pulp can usually be removed.

When the inflammation is localised it sometimes happens that the congested pulp swells through the opening and, new-growth taking place in it, a polypus of the pulp, which may fill up the whole of the carious cavity, is formed. This appears not to contain nervous tissue in any quantity, as it is usually not tender to the touch, unless pressure be indirectly made thereby on the pulp within the walls of the pulp-chamber. Such a pulp may still be destroyed with arsenic, but the ultimate results are not very satisfactory; no pain is attendant on this condition. A special bacillus, *B. gangrenæ pulpæ*, has been described as infesting dead pulps; the reaction is always alkaline, so that softening of the walls of the pulp-cavity in the root does not usually occur.

19. Tooth-pulp, Degenerations of the.—In old age and in teeth which are loosening the pulp loses to a considerable extent its nervous and cellular elements, which are replaced by fibrous tissue. When subject to slight but prolonged irritation, such as is produced, for instance, by gradual wearing down of the tooth until the pulp is approached, it may partially or wholly calcify, so that it does not become exposed on further wear. Irregular calcifications in the form of hard nodules also occur in its substance even in sound teeth; these are often unsuspected as they cause no inconvenience, but occasionally, perhaps, because they have involved nerve-filaments, they cause neuralgic pains and hyperæsthesia of the tooth. They never, however, cause ordinary toothache of any severity.

20. Pyorrhœa Alveolaris.—This disease is of much importance on account of its frequency, and because in it the patient is swallowing in the twenty-four hours larger quantities of pus than in any other dental disorder. The first indication seen is that the margin of the gum, which ought to adhere closely to the necks of the teeth, is in places detached from them and is thickened. Even at

this stage, though more markedly later, if firm pressure be made with the finger, a trace of pus or blood-stained fluid can be squeezed out of the intervening space. As it progresses, the detachment proceeds to greater depth, so that an instrument or a piece of silk can be passed down for some distance between the tooth and its socket, the pouch ultimately often extending the whole length of the root, and leading to the complete detachment and loss of the tooth. Several teeth are affected, and it may extend round the whole mouth. From these pouches there is a continual secretion of pus, which is swallowed during the day, but at night sometimes runs out and stains the pillow. This pus is full of organisms, but no specific organism of the disease has been discovered. As it progresses, the edges of the sockets are absorbed, the absorption being deepest next to the tooth, so that the edge is cupped and the appearance readily recognisable in a macerated skull. It thus progresses from the surface inwards; but it is not certain whether it originates in the gum, in the edge of the alveolar periosteum, or in the edge of the bone itself. It was formerly supposed to be set up by the irritation of deposits of tartar; but there are many reasons for rejecting this idea: the tartar, when present at all, is secondary, being deposited in the vacant pouches. There is neither tenderness nor pain in its earlier stages, so that it is often overlooked. It is rare before middle life; but although it has much in common with the processes by which teeth are lost in advanced age, it appears to be distinct from these, especially in the abundance of the secretion. It appears to spread from tooth to tooth, but it is not clear that this is by virtue of an infection, as it is apt to be more or less symmetrical on the two sides of the mouth. Its ætiology remains obscure, and it is not certain that it is connected with any recognised constitutional condition, though a somewhat similar disease is found in stall-fed animals and in some wild animals kept in confinement, notably the lemurs. Perfectly sound teeth are attacked at least as frequently as those which are or have been carious.

TREATMENT.—This is unsatisfactory; for although great temporary improvement can easily be attained, the disease almost always recurs. The margins of the gum should be painted with cocaine, all tartar carefully removed, and the pouches syringed, any especially deep ones being slit up. Into the larger pouches a strong antiseptic is passed by means of filaments of wool, or into the smaller pouches by means of a thin slip of wood. Powdered sulphate of copper, with or without carbolic acid, gives good results, and is not harmful to the teeth. Mercuric chloride (1 : 500) is sometimes used, and this treatment is repeated every three or four days till improvement is seen in the diminution or absence of secretion. The patient can be instructed to carry it out for himself in accessible positions, and in addition the teeth should be brushed with a soft brush and an antiseptic mouth-wash several times a day. Shampooing the gums with the finger-tip tends to empty the pouches, and at the same time to remove the congestion which is always present.

21. Syphilitic Teeth.—Hutchinson first pointed out that children who inherit syphilis are liable to characteristic deformity of certain teeth, and that this is not infrequently associated with specific interstitial inflammation of the cornea. Syphilitic

teeth are small, narrow, more or less pointed, and usually of a dirty-grey colour. Both the temporary and permanent sets may be affected; but it is the front teeth of the latter, and often the first permanent molars, that exhibit the characteristic and most marked deformity. The lower incisors are peggy and pointed; those of the upper jaw are narrowed, instead of expanded towards the cutting edge; and the central incisors frequently have a crescentic notch. This is due to the tooth having been subjected to a stunting influence at the period when its calcification commenced. Later on this is more or less surmounted, so that the parts first formed are the smallest. The other irregularities of shape in the teeth may arise, or be closely imitated, where there is no specific taint, but the crescentic notch in the contracted cutting edge of the superior permanent central incisor is believed to be diagnostic of inherited syphilis. Hutchinson considers that these malformations of teeth are occasioned by specific stomatitis. But perverted form and nutrition need not be inflammatory in origin; and it may be doubted whether such action arises in these cases. The teeth are dermal organs, and upon the skin syphilis inflicts some of its chief injuries.

22. Tartar.—The teeth at or near the margins of the gums are liable to become coated with a more or less hard deposit of lime-salts derived from the saliva. This consists mainly of calcium phosphate, with a varying proportion of calcium carbonate and a certain proportion of organic matter; a large variety of bacteria thrive in it. It is of importance that it should be removed; for it is uncleanly, is a nidus for bacteria, and causes an unhealthy condition of the subjacent gum. Its accumulation may lead to a progressive recession of the gum, with waste of the alveolus and the loss of the tooth. It is more than doubtful, however, whether it can actually set up the condition described as *Pyorrhœa alveolaris*. When it is present in that disease it is probably secondary to it. A form of tartar is found deep in the alveolus where pouches exist which is darker in colour and harder; all sorts of hypotheses, quite unsupported by any reliable evidence, have been propounded as to its real nature, one being that it is a gouty deposit containing uric acid or urates. See p. 334.

23. Toothache, Diagnosis of.—It may be convenient to enumerate the ordinary causes of toothache and their diagnosis, although this involves some recapitulation.

Pain tolerably constant: (1) *Teeth tender to pressure.*—Dental periostitis, septic or general; advanced stage of *Pyorrhœa alveolaris*; pressure exercised by un-erupted tooth on its neighbours.

(2) *Teeth not tender to pressure.*—Inflamed pulp, locally or generally; irregular calcification of pulp (rarely).

Pain intermittent.—Localised inflammation of pulp; exostosis; mild periostitis; calcifications in pulp.

Great sensitiveness to change of temperature.—Exposed pulp; inflamed pulp, the latter more aggravated by heat than by cold, which often relieves it.

Moderate sensitiveness to change of temperature. Pulp nearly exposed; slight inflammation of pulp; periostitis; exostosis. See also PAIN IN VISCERAL DISEASE, p. 1144.

The examination of the teeth should be made systematically. They should first be inspected with

a dental or laryngeal mirror, and cavities searched for with a fine instrument. Then the teeth should be tapped in succession, to find if any be tender, and finally they should be individually tested with heat and cold. The former can best be applied by touching them with a heated instrument, the latter by a pledget of wool dipped repeatedly in cold water. Finally the patient should be asked to take a mouthful of warm water, followed by one of cold water. See also RÖNTGEN RAYS, p. 1437.

With regard to teeth already filled, it must not be forgotten that a tooth-pulp may be dying under a filling which has in no way failed.

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TEETH, Grinding of.—Automatic movements of the mouth are common to many diseases, especially in young subjects. The movements may be vertical, as if the patient were chewing, or lateral, the teeth being ground together, so as to give rise to a hard unpleasant grating sound. This symptom is not necessarily dependent upon disease, as it may occur in excitable children during sleep, and is often an indication that too much excitement or fatigue has been permitted. Grinding of the teeth may be observed at all ages, and in many different complaints. It is commonly present in cases of cerebral disease and of intestinal disturbance, being often, and probably erroneously, attributed to the presence of worms.

Sometimes children will grind their teeth voluntarily, and apparently with full consciousness of what they are doing. Such cases are, however, rare.

The treatment of teeth-grinding consists in the correction or removal, if possible, of the condition upon which it depends.

TEETHING, Disorders of.—See DENTITION.

TEICHOPSIA (τεῖχος, a wall; ὄψις, vision).—A visual sensation occurring in megrim, and consisting of a dark zigzag line suggestive of the ground-plan of fortifications (p. 972).

TELANGEIECTASIS (τῆλε, far; ἀγγεῖον, a blood-vessel; and ἐκτασις, a dilatation).—Dilatation of capillaries. See TUMOURS.

TELEGRAPHIST'S CRAMP.—See OCCUPATIONAL-DISEASES.

TELESE, in Italy (Benevento).—Sulphur-waters. See MINERAL WATERS.

TEMPERAMENT.—SYNON.: Fr. *Tempérament*; Ger. *Körperanlage*.—In its earliest significance this term was used to denote a relation between the physical characteristics of each individual and his mental attitude to external stimuli of various kinds. Subsequently its significance was narrowed until at the present time it is used in a loose way to indicate the mental characteristics alone.

It was early recognised that no two individuals are exactly alike, either in conformation of body or in attributes of mind. An endeavour was therefore made to group men in definite classes according to their salient characteristics. This endeavour took shape in the recognition by Galen of temperaments which depended upon variations in the amounts of 'fire' and 'water' that entered into their

composition, these being the two of the four 'elements' that were supposed to take part in the process. Galen, therefore, described nine different kinds of temperament. First there came the *ideal* or *balanced* temperament, which was neither hot nor cold, moist nor dry, and in which all qualities of body and mind were therefore present, but no single one present either in excess or deficiency. Then followed four temperaments of simple excess or deficiency, forming the *hot*, *cold*, *moist*, and *dry* types. Lastly came four types in which the two elements were mixed, with the formation of the *hot and dry*, *hot and moist*, *cold and dry*, and *cold and moist* temperaments. Of this classification we still see remnants in the expressions 'hot-tempered,' 'cold-blooded,' and probably also in that of 'dry humour.'

As time went on, the doctrine of 'spirits' arose and exerted its influence upon the doctrine of 'temperaments,' as witness the expressions 'high-spirited,' 'mean-spirited,' 'volatile,' &c.

With the re-entry of the humoral theory into physic an alteration took place in the nomenclature of temperaments, while at the same time an effort was made to correlate the mental characteristics with physical peculiarities determined in some degree by anatomical factors. Here were introduced many of the uncertainties of anatomy. These uncertainties were least in the case of the heart, the connection of which with the blood was about the most certain point of ancient knowledge in anatomy. Of the three types of temperament, therefore, that were recognised at this period, viz. the *sanguine*, the *melancholic*, and the *choleric*, the *sanguine* was regarded as being bound up with a special development of the heart, but the seats of dominion over the other temperaments were more uncertain. Thus there is the greatest difficulty in arriving at an opinion as to the organ that was supposed to be concerned with the choleric temperament. Etymologically the liver with its formation of bile would seem to have been so regarded, but the bile was supposed to be concerned especially with the production of melancholy, and one can hardly credit physicians at this early date with a conception of the multiple functions of the liver, especially when so important a mass of tissue as the spleen remained unappropriated to a temperament. The confusion is still further increased by the fact that by some of the old writers the spleen is definitely stated to be associated with the production of the choleric temperament. The word 'testy' indicates that at a later date the head was substituted for both liver and spleen in this connection. Use of the expression 'lily-livered' by Shakespeare shows that at even his comparatively late date the influence of the liver upon the production of terror was regarded as certain.

Still later the theory of temperaments underwent further compression and modification, with the result that only three varieties—the *sanguine*, the *nervous*, and the *lymphatic*—were recognised. It is unnecessary to discuss the features of these temperaments in detail, but it may be said that the compression took place chiefly in the physical direction, so that although certain broad features were held to characterise the three temperaments so far as the anatomical conformation of their possessors was concerned, the principal force of the distinctions lay in the different classes of character that they described.

With the foregoing brief sketch the theory of temperaments has been brought up to the present day. But it must be confessed that our greatest difficulty has now only begun. For the question is treated both by physicians and by psychologists, and except, to some degree, in the matter of terminology, no two authors agree; while the divergences between the physicians and the psychologists as classes of writers are remarkable. Speaking generally, however, it may be said that modern writers recognise four different kinds of temperament—the *sanguine*, the *choleric*, the *melancholic*, and the *phlegmatic*. These expressions at the present day have entirely lost any significance that they formerly had with reference to the anatomical characteristics of the individual, so that the term 'temperament' has come to possess an entirely psychological significance.

The greatest difficulty with which we have to contend in reference to consideration of the subject of temperaments is that we are face to face with a number of terms that have obtained a firm root in the language. It therefore becomes a question not merely whether we ought or ought not to break loose from all old traditions and build up a fresh doctrine of temperament upon the basis of scientific experiment; but the question also arises whether we can do so, even if we consider that such an iconoclastic course is advisable. The very fact that it has long been customary to speak of four kinds of temperament is even regarded by one writer (Wundt) as a reason why such a division should be fundamental.

The present position in which the matter finds itself is due to the fact that the subject has not been approached from the point of view of experiment. Modern medicine is so strongly bound up with experimental methods that all forms of knowledge that are not founded upon experiment are regarded with disfavour, if not with suspicion. But though such a course is intelligible it is not justifiable. For there is no doubt that persons differ considerably in their mental and physical reactions to external stimuli, so that among men we meet with the pessimist or melancholic person and the optimist or sanguine, to mention two of the most striking contrasts. It must be conceded that the reactions of individuals to external stimuli are largely influenced by education and surroundings. But since we see that the temperaments of parents are reproduced in their children, no less than are their physical characteristics, we shall probably not err if we hold that temperament may be inherited, however much its main characteristics may be subsequently intensified or modified by other factors. Further, even if we put on one side the general impression that the mental characteristics of a child are liable to resemble those of the parent to which it bears the greatest physical resemblance, we are still forced to recognise the fact that the mental processes must result from chemical and physical processes taking place in cells. That is to say, there must be a physical foundation for the mental aspect of temperament. Without presuming, therefore, to assert what must be the way in which temperaments are to be divided in the future, it may be pointed out that inquiry will have to take place in two directions—the physical and the psychological. Physical research will have to be partly ethnological and partly individual, and the same is true of the psychological side of the question.

Upon the physical side a certain, though very small, amount of work has been done already. We know that the specific gravity of the blood is lower in persons with light, than it is in persons with dark hair, eyes, and complexions (Lloyd Jones). Moreover, we know that the specific gravity of the blood-plasma which is normal for one class of individuals is not normal for another class; that some persons in perfect health breathe more slowly than others who are quite comparable with them; and that rates of heart-beat vary in healthy adult individuals of the same sex under conditions that are identical so far as we can determine. And a few other facts of a like nature are known.

Upon the psychological side it is only within the last few years that the subject has been approached from the experimental side at all. Such work as has been done we owe to Kraepelin and his pupils, and they have certainly added a number of points of the greatest importance from the individual side. From the ethnological point of view little or nothing has been done upon this aspect of temperament. Nevertheless the predominating minor character of Anglo-Saxon and Slavonic music is a well-established example of the kind of subject that might be investigated with advantage.

A hopeful branch in which investigation might be carried on is medicine generally. We are already acquainted with certain points which have a bearing upon the subject. Thus the whole class of conditions that are summed up under the name of 'diatheses' are important in this respect. It is well established that rheumatism and gout, to mention only two examples, are diseases that show a great tendency to affect persons of one family. It is not impossible that a series of investigations into a number of points in connection with the physiological characters of persons possessing these diatheses would show that they have certain common peculiarities.

Using the term 'temperament' more from its psychological aspect, we have evidence, too, that it may become altered in the course of disease. This is markedly the case in the different varieties of insanity, in the altered mental attitude that characterises hysteria, in the hopefulness of persons afflicted with thoracic disease as contrasted with the melancholy of those suffering from abdominal disease, in the mental changes that accompany pregnancy and the onset of the climacteric in women, and so on. In the case of all of these, such investigation as has been done, has been done on the psychological side alone. And yet if we allow that the mental processes are but the manifestations of chemical and physical changes occurring in nerve-cells, it is clear that the physical side must be considered also. The matter is, it must be confessed, a very difficult one to approach. For it is quite possible that the processes going on in the nerve-cells are themselves modified by the processes going on in other parts of the body, perhaps, even, by the chemical substances formed during the functional activity of these non-mental parts.

From a practical point of view the subject of temperaments is one of considerable importance. We already know that in the case of hysteria the only treatment that is of any real value is that which is directed to the mental side, when once the physical conditions of the patient have been placed on a satisfactory basis. And in the case of the wounded in warfare it is said that those who have belonged

to the victorious army make better recoveries, *ceteris paribus*, than those who have belonged to the defeated army. The facts upon which this statement is made are difficult to arrive at, but it is not improbable that the statement itself contains a large element of truth. To what extent questions of treatment would be modified by full investigation of the subject of temperament it is impossible to say. But that it is one worthy of research there can be no doubt. It has sufficient in common with the subjects of immunity and predisposition to make us hope that its investigation would be accompanied by recognition of facts of the greatest practical importance. Just as no one could have predicted that researches directed to the explanation of immunity would have given us the treatment of certain diseases by specific sera, so no one can at the present time prophesy as to what benefits investigation into the subject of temperament shall, or shall not, confer upon mankind.

W. S. LAZARUS-BARLOW.

TEMPERATURE.—SYNON.: FR. *Température*; Ger. *Körperwärme*; *Eigenwärme*.

TEMPERATURE IN HEALTH.—The heat of the blood is at every moment of life the result of different forces balancing each other, namely, the heat-producing and the heat-destroying processes and influences. So, also, the temperature of a single part of the body results from the heat of the blood circulating, and heat-production going on, within it, and its exposure to cooling influences, and is chiefly dependent upon its blood-supply. The heat of the body is, therefore, most variable on the surface, and is lowest in its uncovered parts, especially in the most projecting ones; almost uniform, on the contrary, in the interior, where only slight differences, amounting to a few tenths of a degree, exist. In the lower animals Claude Bernard found the temperature highest in the hepatic veins and the right auricle. The heat of an organ increases when its functional activity is heightened, as, for instance, when the brain, a muscle, or a gland is stimulated to increased function.

It is important for practical purposes to consider chiefly the temperature in the interior, or the blood-heat. The blood-heat is measured, as nearly as possible, by means of the clinical thermometer. See THERMOMETER, CLINICAL.

In a healthy man the temperature of the body, as measured in the axilla, is about 98·6° F. (37·0° C.). Inside the mouth, underneath the tongue, it is almost the same; whereas in the vagina or rectum it is 0·3° to 0·6° higher. Under special circumstances—for instance, when a considerable cooling of the surface takes place, or when the skin freely perspires—the difference may be somewhat greater, and there may be a difference of 20° between the temperature of exposed parts of the skin and the interior; on the other hand, all parts may be pretty equally warm in the morning in bed, or in a warm room, or when the circulation has been influenced by slight exercise, or by a good meal and a moderate amount of alcohol. Of considerable influence upon the temperature of the surface of the body is the amount of fat in the subcutaneous tissue. In plump children and in very obese adults the surface may be considerably colder than the interior, and in the latter case there generally is yet another and even more effective cause for this difference, namely, weakness of the heart's action.

The temperature of the body is not the same all through the day. Numerous careful observations, of which those of Jürgensen, Liebermeister, and V. W. Ogle may be specially mentioned, have shown that the temperature of a healthy person, even when kept entirely at rest in bed, will fluctuate from about $1^{\circ}8'$ to $2^{\circ}3'$ F. in the course of the twenty-four hours; the mercury standing lowest between 2 A.M. and 6 A.M., and then gradually rising until it reaches the highest point between 5 P.M. and 8 P.M. This rise is mostly not continuous, but becomes somewhat slower, or even interrupted by a slight decrease, in the middle of the day, the afternoon hours showing a more rapid elevation.

This daily fluctuation of the body-heat is a fact of fundamental importance, for it not only takes place in health, but also when, in disease, the whole range of the temperature is either depressed or abnormally elevated. The causes of this daily fluctuation of temperature are not yet fully made out; but this much is certain, that rest and movement, as well as the taking of food, have some share in producing it. Another cause which must not be overlooked, and which was pointed out by Liebermeister, is the force of habit and inheritance.

Race and sex have no influence, to speak of, upon the range of the temperature. Age, on the contrary, has by different observers been found to influence the range as well as the daily fluctuation. In the infant, immediately after its birth, the temperature is slightly higher than later on, its temperature in the rectum having even been found higher than that of the vagina of its mother. A higher range is maintained in the first weeks of life, and there has also been found wanting in infants that steady course of the daily fluctuation which is observed in adults. In children, even somewhat more advanced in age, the temperature is still easily influenced by external causes, and the range of the daily fluctuation is greater. In old age, again, the range is a little greater than in adult life, and here, also, a greater mobility of the temperature under various external circumstances is observed. The menstrual process, as shown by Goodman, Mrs. Jacobi, and Reinl, causes a slight elevation of temperature, amounting, however, to only a few tenths of a degree in the pre-menstrual period, the temperature going gradually down again during the flow and after it is over. Pregnancy has no appreciable influence in healthy women. Parturition slightly increases the temperature, evidently by the increased muscular action, an increase which is compensated by a corresponding fall after the birth of the child. If no complications occur, the temperature in the puerperal state generally does not deviate from the normal.

Such, in fact, is the constancy of the body-heat in health, that the general conditions of life, occupation, &c., hardly show any influence upon it, and whenever, by muscular exertion, or by the effect of external heat or cold—as, for instance, by baths of various temperatures—a more considerable deviation from the normal range has for a time been caused, there is a strong tendency in the system to compensate for the increase or the loss of heat by a corresponding fall or rise afterwards. This faculty is somewhat altered in disease; and even in those states which are on the borderland of disease, as well as under the influence of fatigue or exhaustion, we frequently find a less perfect action of the regulation of the local or general temperature of the body.

LOCAL CHANGES OF TEMPERATURE.—Local changes of temperature are brought about by external thermic influences acting locally, or by disease. Thus the normal symmetry of the two sides of the body, as regards their temperature, may be disturbed. Local stoppage of the blood-supply or local death lowers the temperature of the part; inflammation, in its first stage, raises it. Considerable local changes of temperature may arise simply from vaso-motor disturbances. Thus, in a paralysed limb the temperature may either be lower or higher than in the corresponding limb of the other side; in hemiplegia the temperature of the paralysed side is frequently found 0.5° – 1.5° higher than on the normal side. Neuralgia is sometimes accompanied by dilatation of the blood-vessels, and a considerable rise of the local temperature; and, as a purely vaso-motor disturbance, local heat and redness of the skin, due to a passing dilatation of blood-vessels, sometimes occur in hysteria.

CHANGES OF THE GENERAL TEMPERATURE.—Of much greater practical importance than local disturbances of the body-heat are changes of the general temperature, such as occur in many diseases, whether of the nature of *depression* or of *elevation*.

1. Depression.—Depression of the general temperature is observed as a consequence of considerable loss of blood; in starvation from any cause; and in the wasting of some chronic diseases, such as cancer of various organs; or in diseases of the brain and spinal cord. In brain-disease, with the symptoms of melancholia, extreme coldness of the surface and lowering of the general temperature occasionally occur. In a case of hæmorrhage into the medulla oblongata, Lemcke observed a temperature as low as 73.4° F. (23° C.) in the rectum twenty-seven hours before death; and extreme loss of heat, though hardly to such a degree, by vaso-motor paralysis and dilatation of the blood-vessels, may be caused by severe injuries to the upper part of the spine. A considerable fall of temperature may take place in a very rapid manner in the collapse sometimes occurring in the course of typhoid fever, but especially in that of acute peritonitis, and of poisoning by various substances. In the collapse of cholera the lowering of the temperature of the axilla may be considerable, a temperature as low as 89.6° F. (32° C.) in the axilla, and even less under the tongue, having been observed; at the same time the temperature of the interior of the body is generally very high, reaching 104° F. (40° C.), and sometimes much more. In peritonitis a low general temperature may be present for days, even if the peritonitis supervene in the course of typhoid fever. With the collapse caused by alcoholic intoxication great depression of the general temperature occurs when the patient is exposed to cold and wet; and in a case of carbolic-acid poisoning, which came under the observation of the writer, the vaginal temperature fell as low as 93.92° F. (34.4° C.). A temperature of 71.6° F. (22° C.) has been observed in sclerema neonatorum.

In chronic diseases of the respiratory organs, not of an inflammatory or tubercular nature, as well as in chronic heart- or kidney-disease, the temperature is generally found somewhat below normal; and the same is the case in chronic nephritis, more especially in those cases which are accompanied by general dropsy. In cases of the latter kind we sometimes see even a febrile temperature, caused, for instance,

by tubercular disease of the lungs, become considerably abated, if not entirely reversed, when chronic kidney-disease supervenes; as also when intestinal ulceration becomes more prominent, or leads to peritonitis. From some observations of the writer of this article, it would seem as if intracranial disease, such as softening or tumour, arising during the course of a febrile disorder, e.g. pulmonary phthisis, may not only disturb the regularity of the febrile temperature-changes, but, by a kind of inhibitory action, entirely suppress the febrile elevation of temperature.

2. Elevation.—Elevation of the general temperature, as part of the febrile process, is the most prominent symptom in most diseases of an infectious origin, with or without inflammation. An increased production of heat, no less than a disturbance of the regulation of the temperature, is the effect of the presence in the blood of some foreign substance, acting injuriously on the nervous system, and causing altered chemical processes. Modern research gives the most prominent part in these actions to micro-organisms, which by chemical substances contained in their own bodies, or by chemical products of their own life-changes, or by changes which they cause in the cells of the body, are assumed to be the cause of the febrile process, and thus of the increased temperature.

Some such substances, belonging chemically to the albuminoids and acting as ferments, have recently been extracted from pure cultures of the yeast-fungus by Roussy, and of various bacteria by Koch (tuberculin), Buchner, and others; and their action on the temperature of the body has been studied by Krehl and Matthis. *See FEVER.*

The proper balance of the heat-forming and heat-destroying processes may also be disturbed by other influences acting upon, and by primary diseases of, the nervous system. When the body is subjected to external cold after it has been fatigued by exercise and already cooled by perspiration—if, for instance, a cold bath were taken under such circumstances—a rigor, with rapid rising of the temperature, may follow; but, no local disease becoming developed, the temperature quickly goes down again amid profuse perspiration, and the whole attack may be over. Or a disturbance of the heat-regulating functions of the nervous system may be caused by the irritation of some nerve-filaments, as by a gall-stone passing the biliary duct, or a stone passing through the ureter or the urethra, and a febrile attack will follow. The rigor, leading to a high fever of an evanescent character, which may follow the introduction of a catheter (urethral fever), sometimes belongs to the same group of cases. But generally, in cases of this kind, the nervous system is already in an abnormal state through the previous disease, which, besides, may be of an infectious nature and thereby give rise to pyrexia. The functions of the nervous system may further be deranged by injury; and a rise of temperature has not only been observed in injuries to the brain, but, in a most excessive degree, sometimes after injury of the cervical part of the spinal cord, when temperatures of 110° to 111° F. (43.3° to 44° C.) have been observed (B. Brodie, H. Weber, Teale, and others). In tetanus a very high temperature may occur, rising still higher a little after death; as much as 112.55° F. (44.75° C.) was reached in a case observed by Wunderlich. An alteration of the heat-regulating functions of the nervous system may be brought about by a con-

siderable external heat acting upon the body, especially when combined with moisture of the air and fatigue. In such cases of sunstroke or heat-stroke, it is quite common to see the temperature of the body rise to 108° F. (42.2° C.) and more; and it would seem probable that a febrile elevation of temperature, if going on unchecked for a considerable time, by causing exhaustion of the nervous system, may lead to hyperpyrexia. Thus it is not very uncommon to see the temperature rise excessively in infectious diseases, especially scarlatina, towards the close of life (*proagonic hyperpyrexia*); and the temperature may, in such cases, even rise a little more immediately after death. This is due to the loss of heat being greatly reduced after the stoppage of the circulation, the heat-production going on in the interior for a time; and the gradual failure of the circulation probably also takes a great share among the causes of a proagonic hyperpyrexia.

Hyperpyrexia sometimes comes on during convalescence from acute rheumatism, even after the fever has entirely subsided, and when the patient is apparently convalescent. An excessive rise has occurred and caused death in severe cases of hysteria; and in hysterical patients hyperpyrexia has occasionally been observed without any of the symptoms which in other cases usually accompany so grave a phenomenon. Cases of this kind are extremely suspicious, and in some of them it was discovered how this hyperpyrexia was simulated. Thus a patient had driven up the mercury by rubbing the bulb of the thermometer between the folds of her night-dress; while in other cases the high elevation of the mercury had been brought about by means of a hot poultice, or by the patient having lowered the top of the instrument, so that the column of mercury began moving by its own weight. This, however, is not possible with a thermometer of the thin bore which English thermometers now generally have. A very high temperature, to which the pulse and respiration and the other symptoms do not correspond, must always arouse a suspicion that the rise of the mercury has been artificially produced, and the verification will be easy if the physician carefully watch the mercury as it rises, or take the temperature in the rectum or vagina.

The very important part which the nervous system plays in regulating the blood-heat is also seen in the great liability of the temperature easily to deviate from the normal range during convalescence from acute disease, when the weakness of the nervous system, brought on by the previous illness, will show itself in this no less than in other alterations of function. This can frequently be observed in convalescence, not only from the eruptive fevers, but also from pneumonia and other acute febrile diseases, when trifling external influences may cause a considerable rise of the temperature, which, however, is generally of short duration only, but which, in the case of specific fevers, may cause apprehension lest a relapse or a complication be coming on. In a somewhat different manner, and more lasting, a slight sub-febrile elevation of temperature may be observed in the convalescence from acute rheumatism, in which it may persist for weeks without any joint- or heart-symptoms being present.

SIGNIFICANCE OF ABNORMAL TEMPERATURE FOR DIAGNOSIS AND PROGNOSIS.—The maintenance of the heat of the body within a certain range being so insured, any deviation of the general

temperature from the normal standard, however slight in degree—unless of a very transient nature, or brought on by evident external causes—is to be taken as a sign of disease. Such deviation may be of a variable degree, along with symptoms which, in part, are the consequence of the abnormal temperature, such as an abnormal rate of pulse and respiration, and in part due to the nervous system. The whole range of deviation within which life can be maintained is comprised between 90°F . (32°C .) and 110°F . (43.4°C .). A temperature approaching either end of this range indicates a condition of extreme danger, which is already great with a temperature of 95°F . (35°C .), or beyond 106.5°F . (41.5°C .). With reference to the general condition of a patient who presents an abnormal temperature, a few distinctions may be conveniently tabulated.

(1) *Temperature below the normal*:—

(a) Temperature of collapse, below 97°F . (36.2°C .).

(b) Subnormal temperature, 97 – 98°F . (36.2 – 36.7°C .).

(2) *Normal temperature*: 98.0 – 99.5°F . (36.7 – 37.5°C .).

(3) *Temperature above the normal*:—

(a) Sub-febrile temperature, 99.5 – 100.5°F . (37.5 – 38.05°C .).

(b) Febrile temperature of moderate degree, 100.5 – 102°F . (38.05 – 38.88°C .), morning; 102.2 – 103°F . (39 – 39.44°C .), evening.

(c) Febrile temperature of high degree, 102.5°F . (39.2°C .) and more in the morning; 105 – 106°F . (40.6 – 41.1°C .) in the evening.

(d) Hyperpyrexia, 105.8 – 107.5°F . (41 – 42°C .) and more. Extremely dangerous.

Single Observations.—Near the extreme points of this scale a single observation of the temperature of a patient may at once decide the prognosis. Thus a temperature below 93°F . (33.88°C .) or above 108°F . (42.22°C .) is almost always fatal, although cases have recovered by active treatment, in which the latter point has been exceeded by several degrees. No less valuable may single observations be for diagnosis, chiefly in a general way, in showing that there is disease when, perhaps, no other symptom points to it, but also for the diagnosis of a special disease in some instances. Where there are other symptoms of disease, the discovery of an abnormally high or a febrile temperature may at once give quite a different aspect to a case, as, for instance, when a patient who has been suffering for some time from a troublesome cough without expectoration, but in whom the most careful examination of the chest could not detect any lung-disease, is found to have pyrexia. The suspicion that there is commencing phthisis may thereby be at once confirmed, or aroused for the first time. Or, again, in a case where the patient simply complains of dyspepsia and lassitude, the thermometer may give a degree of heat which would not have been expected either from the looks of the patient or from the temperature of his hands or chest, and the attention may thereby at once be directed to the possibility of the case being one of typhoid or some other specific fever. One reservation must be made with regard to single observations in febrile patients who have not been kept at rest for some time before, for example, in patients who have walked to the physician's house, or who had

to undergo a journey to the hospital. In such cases the temperature may become somewhat altered by the fatigue; and it is quite common to find the first temperature in a patient, immediately after his admission into the hospital, considerably higher than after a few hours' rest, or, if he have been exposed to cold, much lower than what would otherwise correspond to his condition.

Systematic Series of Observations.—But of much greater value than isolated observations of temperature is the regular and continued watching of the course which the temperature takes in a disease. Many diseases present a deviation from the normal temperature, showing a typical course as regards the duration, as well as the daily fluctuations, of the abnormal temperature. The course of its temperature being part of the natural history of a disease, the study of this is of great importance for diagnosis. See FEVER.

TYPES OF PYREXIA.—First, it is the *mode of rising* of the temperature which varies, and by which some diseases may be distinguished. In some diseases a contraction of the peripheral arteries takes place at the onset, which, by diminishing the peripheral circulation and the giving-off of heat, leads to a rapid rise of the internal temperature, and is accompanied by a sensation of cold. In pneumonia, therefore, and other diseases commencing with a rigor, the temperature rises rapidly and continuously to a height of 104°F . (40°C .) or more; whereas diseases with a more gradual beginning show simply a slow elevation of the normal range, both morning and evening temperatures becoming gradually higher, the usual daily fluctuation being maintained. Thus, in the first few days of typhoid fever the temperature rises every day about 2°F .; but the temperature going down again in the morning by about 1°F ., the maximum of about 105°F . is only attained on the fifth or sixth day.

At the *height* of a disease the temperature may fluctuate round an average temperature of about 103°F . (39.5°C .) or more, while it shows the same daily course as in health, that is, is lowest in the morning and highest in the evening. The range of this daily fluctuation may, however, differ considerably in different diseases; and according to the extent of the daily fluctuation three types may be distinguished. When the daily fluctuation of an elevated temperature shows only the normal difference, or even a smaller difference, between the morning and evening temperatures, we speak of *continuous*, or, more correctly, *sub-continuous* pyrexia; when the difference is greater than the normal, the remission having a tendency to a low temperature, and the exacerbation, on the contrary, to a considerable rise, the pyrexia is called *remittent*; and, thirdly, when the remissions reach the normal, or recede even below it, we have the *intermittent* type of pyrexia.

A *continuous* elevation of temperature is observed soon after the commencement of a disease, and during its height.

Considerable *remissions*, or even *intermissions*, of the febrile temperature are chiefly observed in the decline of some acute diseases, and in chronic inflammatory diseases, especially of a tubercular nature, or in chronic syphilitic affections, in pernicious anæmia and leucæmia, as well as in some cases of lymphadenoma (Hodgkin's disease), the remissions generally becoming more marked as the exhaustion of the patient increases.

The *intermittent* type of pyrexia is most typically shown in malarial diseases, in which the elevation of temperature may follow a quotidian, tertian, or quartan type. The same also sometimes occurs in chronic tubercular disease of the lungs and in pyæmia. Pyrexia of a remittent type may present a peculiarity which is worthy of note, as being of some diagnostic value. Whereas in the great majority of cases the daily fluctuation follows the rule of health, the exacerbation taking place in the evening, we sometimes meet with cases where this order is reversed, the rise taking place in the morning, and the remission occurring in the evening. This 'inverse type,' as Traube has called it, of the daily fluctuation of a febrile temperature has been observed in some rare instances in typhoid fever; more frequently in cases of chronic lung-disease. In doubtful cases of inflammation of the lungs it has some significance as to the disease being tubercular.

Slight deviations in the maximum daily rise of a febrile temperature occur sometimes in such a way, that the height is reached in the middle of the day, or that the exacerbation takes place in the night, or that two or more considerable elevations, instead of one only, take place in the twenty-four hours. Such occurrences, which have been observed in typhoid fever and in phthisis, can, of course, only be found out by the observations of the temperature being repeated with sufficient frequency. A more frequent application of the thermometer will also be necessary in some cases of ague, where the attacks are not well marked, or occur in the night, in order correctly to judge of the case.

The *decline* of the elevation of temperature, at the termination of a disease, may be gradual, the daily fluctuation, however, taking place as usual; or it may be rapid, by a continuous sinking of the temperature to, or somewhat below, the normal, in the course of from twelve to thirty-six hours, or even in six to eight hours, as in relapsing fever. The latter mode of termination of a fever is called *crisis*, whereas the former is designated *lysis*. A crisis may sometimes be accompanied by symptoms of collapse, and, in some rare instances, by acute delirium, which, however, generally passes off within a day or two, and is not of bad omen provided the general condition of the patient remain good. Symptoms of this kind, as well as a more considerable elevation of the temperature just previously to its fall, or a great irregularity in the course of the temperature preceding it, may be called *perturbatio critica*. It would appear that diseases caused by the action in the system of some foreign substance—as, for instance, some infective agent, its action being of a limited duration—have a tendency to a critical defervescence. Diseases, on the contrary, in which an organ has become materially altered, as by an injury, or by some infection either primarily or, in the course of a chronic disease, by a longer duration of the morbid action, or by some supervening secondary infection, show a slow decline of the pyrexia, with a tendency to a remittent type. The repair of the damaged structures taking some time, the decline of the pyrexia is slow, and the defervescence by lysis. Examples of the former mode of defervescence are furnished by acute pneumonia, erysipelas, typhus, relapsing fever, and measles when not complicated by more serious inflammation; the latter type is shown by typhoid fever, in which the specific process produces deep

ulcerations in the glandular structures of the intestine, which persist for some time after the specific process has terminated. The ancient physicians believed that a crisis took place on certain days in particular, as, for instance, the seventh day of an illness; but more extended experience, gained by means of the thermometer, has shown that, although a change or a termination of a disease may take place at a certain definite period, the latter is not bound to one particular day. See *CRISIS*.

Any *irregularity of the course* of the temperature in a disease in which, as a rule, it runs a very regular and definite course, is indicative of some disturbance or complication, and its early detection is therefore important for diagnosis, no less than for prognosis and treatment.

On the *approach of death* the temperature in many cases gradually sinks; but instances are not of rare occurrence in which, on the contrary, especially in diseases with high fever, a continuous rise takes place towards the fatal termination, reaching sometimes hyperpyrexial degrees.

In *convalescence* the temperature is more easily influenced by external causes, as well as by internal changes, and the approach of a relapse or complication being at once indicated by a rise of temperature, the continuance of regular thermometrical observations in the first period of convalescence is of very great importance; the more so, as convalescents are sometimes not sensible to changes, which at first only show themselves in an alteration of the temperature.

TREATMENT.—Abnormal states of temperature ought not, as a rule, to be considered as objects of treatment by themselves, all the concomitant symptoms, in fact the whole state of the patient, having to be taken into consideration, in order properly to treat a case of febrile disease. But there are exceptional cases in which the state of the temperature at once urgently requires a symptomatic treatment. Such are, for instance, cases of *hyperpyrexia* in sunstroke or heat-stroke, in which the most energetic means ought at once to be applied to reduce the temperature. As the experience of Levick and other American physicians has shown, life may in such cases sometimes be saved by perseveringly rubbing the surface of the body with large pieces of ice, using at the same time stimulants by the rectum or subcutaneously. A rapid abstraction of heat by rubbing with ice, or cold bathing with affusions, may also be the only means of saving a patient in whom, in the course of acute rheumatism, hyperpyrexia has set in; and a case published by Wilson Fox, in which the temperature reached 110° F. in the rectum, showed that external cooling may be successful, when even very large doses of quinine (120 grains had been given in six hours) had been administered without effect. The same plan must be followed in hyperpyrexia occurring in the course of other diseases. Complications, such as pneumonia, or even pericarditis, do not contra-indicate this treatment, the success of which is, however, dependent upon the possibility of rousing the nervous system, and upon the circulation remaining sufficiently active.

An *abnormally low temperature* requires the external application of heat, which will be materially assisted by warm stimulating drinks or injections, using eventually subcutaneous injections of ether or of tincture of musk, or intravenous or subcutaneous injections of a 0·6 per cent. solution of

chloride of sodium in water, to stimulate the action of the heart, and to increase the blood-pressure. See SHOCK.

Apart from such exceptional cases, the treatment of the abnormal states of the temperature must be subordinated to the general treatment of the case. In many cases the abnormal temperature being dependent upon some local cause, the removal of the latter will make the abnormal temperature also disappear, or at least reduce it—an experience with which surgeons are quite familiar.

Rise of temperature being, however, the most appreciable and a most important symptom of *pyrexia*, which, by weakening the heart's action, by lowering blood-pressure, and in other ways, may of itself lead to serious consequences, it would appear advisable in many cases of protracted febrile disease, besides the general or special treatment which the case requires, to treat the febrile temperature symptomatically. An overwhelming experience has actually shown that the course of the specific fevers, such as typhus, typhoid and scarlatina, although it cannot be cut short, can yet be materially influenced, by measures the primary object of which originally was to keep the febrile temperature artificially down, by means of cold baths or wet packing, or by antipyretic medicines. In rehabilitating the cold-water treatment of fevers, which already in the hands of J. Currie had been so successful, E. Brand, Jürgensen, Liebermeister, and their followers started from the idea that the increased blood-heat ought to be combated; and Liebermeister, than whom few physicians have more practical experience in these matters, laid down as the principle to be followed in the antipyretic treatment of fevers, to increase the remissions that normally take place in the daily fluctuation of temperature, and to prolong them as much as possible. Comparative experiments having shown that external cooling by baths, as well as antipyretic medicines, are of greater effect on the temperature at those times of the day and night when it spontaneously has a tendency to decline, these periods were declared the most favourable for the employment of antipyretic measures, necessitating their least frequent repetition. Others have not laid so much weight on the normal daily fluctuations of febrile heat, but have used the bath whenever the temperature rose to a certain point, say 102.2° F. (39.0° C.) or 103° F. (39.5° C.), obtaining in this way equally favourable results, though, perhaps, with more inconvenience to the patients. More extensive experience has, however, shown that lowering the febrile temperature in itself—which at present, by using one or the other of the antipyretic medicines which modern chemistry supplies in such variety and profusion, can be accomplished to a very high degree—does not produce the same good effect on the whole condition of the patient, or so favourably influence mortality as cold bathing or other methods of external cooling. It therefore appears very probable that the beneficial action of the cooling treatment by these external means is due not so much to the reduction of the temperature, as to their powerful influence on the circulation and on the tone of the nervous system. So there may, perhaps, after all, be no reason to feel disappointed at the very transient effect which even a very cold bath has in a case of high fever, and which, as far as mere reduction of temperature is concerned, makes its action appear much inferior to that of antipyretic

medicines. For, troublesome as it may be for the patient, as well as for his attendants, to repeat the external cooling over and over again in the course of a day, it may yet be this very repetition of the bath, with all its effects, which is of essential importance.

The theory from which antipyretic treatment originally started—that pyrexial elevation of temperature is an unmixed evil—is becoming more and more modified as the conviction is gaining ground that a process like the febrile reaction of the system following upon various external influences, which was, no doubt, gradually acquired through evolution, must be of some advantage to its possessor, if it be only this—that by the increased chemical action which goes on at a higher temperature, abnormal and noxious products of tissue-change and of the life-process of infectious germs will be more rapidly destroyed and cast out of the body. But it would certainly be a step in the wrong direction if such considerations were to lead to giving up all antipyretic measures. On the contrary, experience teaches that it is very important not to wait in a case with continuous high temperature until symptoms of failure of the heart's action—a weak pulse, cold extremities, cyanosis and congestion of the lungs, and muttering delirium—show themselves, but to try to prevent these symptoms by at once resorting to external cooling. Patients treated early on this principle will be found much less frequently to pass into that state, to sleep more soundly, and to retain their appetite, bedsores and other serious complications being of much rarer occurrence; and it has been established that the mortality in specific fevers has by the cold-water treatment been very considerably diminished, and that convalescence also is quicker than in cases treated on a purely expectant plan. If consistently carried through, the good effects of such treatment mostly show themselves in a few days, not only in the whole condition of the patient, but also in the effect of successive baths on the temperature becoming greater and more lasting, so that the number of baths, as required by a certain height of temperature, becomes less every day.

As regards the methods of external cooling, by far the most effective means are cold baths from 60° to 70° F. (15° to 20° C.), of about ten minutes' duration. More agreeable to the patient are baths of about 95° F. (35° C.), gradually cooled down by the addition of cold water to 70° F. (20° C.), or less, as recommended by von Ziemssen, but their duration must be longer to have the same effect as the former. Riess tried prolonged immersions in warm water of 90° F. (32° C.), but, although acting very powerfully on the temperature, the general effect of such permanent baths is inferior to that of repeated short cold baths. Cold wet-packing is less effectual, but may replace baths in special cases, and when they cannot be given. The same holds good as regards cold sponging. See HYDROTHERAPEUTICS; and COLD, Therapeutics of.

Antipyretic medicines may be used sparingly in order to prolong the effect of a bath on the temperature in cases of very high and persistent fever, but principally with a view to allay some of the troublesome symptoms accompanying pyrexia, especially the headache, the feeling of heat, and uneasiness. For a long time sulphate of quinine had been the only reliable antipyretic drug, but now we possess a great number of chemical preparations, which are

much more powerful in reducing a febrile temperature and subduing some of the other symptoms of fever than anything before known. But it ought never to be lost sight of that these preparations, foremost among which stands phenazone, although in individual cases they may be of considerable service, are substances whose effects on the tissues of the body, and especially on the nervous system, are as yet but very imperfectly known, and that a routine treatment of elevated temperature with their aid is much to be deprecated, and cannot compare in innocuity and results with even a routine treatment with cold water, which, it need hardly be said, must also be carefully adapted to the requirements of the case and the individuality of the patient.

C. G. H. BÄUMLER.

TENDERNESS.—*See* PAIN IN VISCERAL DISEASE.

TENDON-REFLEX.—*See* NERVOUS SYSTEM, Examination of; and SPINAL CORD, Diseases of.

TENESMUS (τέλινω, I stretch).—**SYNON.** : Fr. *Ténésie*; Ger. *Stuhlzwang*.—Tenesmus is a comprehensive term applied to certain morbid sensations referred to the anus and its vicinity. There is a feeling of fulness and weight, an uncontrollable desire to go to stool, and straining during the act of defecation, little or nothing being passed, and that often of the nature of slimy mucus or blood, while little or no sense of relief is experienced afterwards. Tenesmus is a common symptom in cases of dysentery. It may also be associated with local diseases about the lower part of the rectum or anus, such as piles, fistula, or malignant disease. Other morbid sensations are often present at the same time.

TREATMENT.—Any local cause of tenesmus must be removed or cured, if practicable. The sensations are best relieved by local applications of heat or cold, wet pads, or the use of small enemata containing laudanum, or of suppositories of morphine, belladonna, or cocaine.

FREDERICK T. ROBERTS.

TEPLITZ-SCHOENAU, in Bohemia.—Simple thermal waters. *See* MINERAL WATERS.

TERMINI IMERESI, in Sicily (*Thermæ Himienses* of the Romans).—Thermal waters. *See* MINERAL WATERS.

TERTIAN (*tertius*, the third).—A term applied to a form of intermittent fever in which the paroxysms return on the third day, or at an interval of about forty-eight hours. *See* MALARIAL DISEASE.

TERTIARY (*tertius*, the third).—This word is usually associated with the advanced forms of syphilitic disease. *See* SYPHILIS.

TESTAMENTARY CAPACITY.—*See* CIVIL INCAPACITY.

TESTES, Diseases of.—The diseases of the testes will be discussed in the following order:—

1. Abnormalities, p. 1681.
2. Atrophy, p. 1682.
3. Chondroma, p. 1682.
4. Cystic Disease, p. 1683.
5. Fibroma, p. 1683.

6. Hernia Testis, p. 1683.
7. Hypertrophy, p. 1683.
8. Inflammation, p. 1683.
9. Injuries, p. 1685.
10. Carcinoma and Sarcoma, p. 1685.
11. Neuralgia, p. 1686.
12. Teratoma, p. 1686.

See also HEMATOCELE; HYDROCELE; and PAIN IN VISCERAL DISEASE.

1. **Abnormalities.**—(a) *Absence.*—There may be complete absence of the testicles. The subjects of this imperfection, if they attain the age of puberty, present the ordinary characteristics of eunuchs. As the complete gland is formed from two distinct parts, the failure or arrest of development may be limited to either part, separately from the other. Thus cases are described where a well-developed vesicula seminalis and vas deferens have been found, without any trace of a testicle; and others, where a testicle existed with complete or partial absence of the vas deferens.

(b) *Excess.*—Supernumerary testicles have been described, and men not infrequently believe themselves to be so gifted. The mistake has arisen from the presence of encysted hydroceles, or of fatty or fibrous tumours of the cord, or of an old epiplocele. There is no perfectly authenticated case recorded of the presence of more than two testicles.

(c) *Malposition.*—The testicles are developed in the abdomen, and at birth, or shortly after, are lodged in the scrotum. A pouch of peritoneum, the processus vaginalis, precedes the testis into the scrotum. This change of position is frequently described as ‘descent of the testicle,’ an obvious misuse of words, if regard be paid to the usual position of the foetus in the uterus.

The testicle may be retained in the *abdomen*, or in the *inguinal canal*; or may pass through the inguinal canal into the *perineum*, and be lodged between the bulb of the urethra and the anterior part of the tuber ischii; or over the external pillar of the ring into the subcutaneous tissue of the *upper part of the thigh*; or through the crural canal to the *upper and inner part of the thigh*; or, if it has passed into the scrotum, may be *rotated*, so that the epididymis is in front and the testicle behind. More rarely it has the long axis transverse instead of oblique; or it may be completely inverted, so that the globus major is below, the globus minor above.

Retention in the abdomen or inguinal canal may be the result of non-attachment of the gubernaculum to the testis, of adhesions from intra-uterine inflammation, or of disproportion between the gland and the orifices through which it has to pass, or of some constricting band. The passing through the crural canal to the thigh, or through the inguinal canal to the perineum or thigh, is explained by the wide and extensive attachments of the lower end of the gubernaculum. Malposition in the scrotum may be caused by some abnormality in the development of the cord.

The consequences will vary with the position. If the testicle is retained in the *abdomen*, the corresponding half of the scrotum remains undeveloped, and the gland is always much smaller than normal. Sometimes there is an arrest of development, or it undergoes fatty or fibrous degeneration, or, if otherwise normal, does not secrete a fertilising fluid. This seems fairly established by numerous observations, both in men and the lower animals, where the testicle has been abnormally retained in

the abdomen. One case, however, has been recorded by Hutchinson, where the observer, to whom the retained testicle was submitted for microscopical examination, stated that he found numerous spermatozoa.

When the testicle lies at the internal inguinal ring, the epididymis is frequently found partly in the badly developed scrotum, into which also extends the processus vaginalis. As the communication between this and the peritoneal cavity is usually maintained under such conditions, in case of peritonitis with effusion there may be distension of this process, so as closely to simulate a hernia, and render an exploratory examination necessary. In most instances testicles which are retained in the inguinal canal do not produce spermatozoa.

Retention of the testicle in the *inguinal canal* is more liable to produce complications than retention in the abdomen. It is often accompanied by inguinal hernia; the organ is more exposed to injury; and when enlarged at puberty, or by inflammation, may cause severe pain from constriction by the surrounding parts. The retained testis may also undergo axial rotation causing torsion of its pedicle. This usually follows sudden descent into the scrotum. The writer has known the descent occur at the ages of six months, ten years, and sixty-eight years. The sudden change in position is usually followed by a sharp attack of orchitis. But, owing to the length of the mesorchium, the spermatic cord may become twisted, and obstruct the return of the venous blood. The whole testicle and epididymis then become infiltrated with blood, and subsequently inflamed. The tumour in the inguinal canal and scrotum closely simulates a strangulated hernia, and the accompanying pain and vomiting heighten the resemblance. The bowels, however, continue to act together with the passage of flatus and fæces. The treatment of torsion of the cord is by open incision so as to allow of the twist being undone, or the testicle removed if obviously gangrenous.

Inflammation of a testicle retained in the inguinal canal has been mistaken for strangulated bubonocoele, or for a bubo. Careful examination of the scrotum should, therefore, be made in doubtful cases.

Retraction can usually be distinguished from retention of the testicle by the state of development of the corresponding half of the scrotum.

The *perineal* or *femoral* position of the gland is not of necessity attended by any bad results. A testicle, however, so situated is usually smaller than normal and is more exposed to injury. This is especially the case in the perineal position. In addition it is important to remember before undertaking operative proceedings that the processus vaginalis accompanies the testis into the perineum and usually communicates with the abdomen.

TREATMENT.—If retention of the gland in the inguinal canal be attended by any inconvenience, operative interference may succeed in placing it in the scrotum. But if this should fail from shortness of the cord, and in spite of separation of the cord and inversion of the testis, then extirpation might be necessary. It is undesirable to thrust the gland back into the abdomen. When in infants retention of the testicle is complicated by an inguinal hernia, the use of a truss is not to be recommended, as it will prevent the possible descent of the testicle, and the hernia not infrequently spontaneously sub-

sides. But if a truss with a horseshoe-pad can be adjusted above the testicle, the hernia is prevented and the descent of the testicle assisted. If the retention of the gland be permanent and cause inconvenience, it is better to operate at once. If the gland has passed through the crural canal, little can be done to remedy the malposition; but when it has passed through the inguinal canal into the perineum or the thigh, an attempt may be made to place it in the normal position. In an adult such a proceeding might be difficult owing to the non-development of the scrotum.

John Wood successfully transplanted in an infant a testicle from the perineum to the scrotum subcutaneously. When a band of tissue in the perineum, probably the gubernaculum, had been divided with a tenotomy knife, the gland could be pushed up to the inguinal canal, and from thence into the scrotum, where it was retained by a hairlip pin passed above it as in acupressure. This method is now replaced by the open operation. As sometimes the unaided efforts of nature draw the gland from the perineum up to the inguinal ring, where it is comparatively safe from injury, and more favourably situated for any attempt at removal to the scrotum, it is advisable always in infants to allow time for such a possible modification. Should an operation be deemed advisable, an open incision should be practised, and each step guided by vision.

Of malpositions in the scrotum, that in which the epididymis is in front, and the testicle proper behind, is alone of any practical importance. In any operation for hydrocele or hæmatocele of the tunica vaginalis, the position of the testicle ought first to be ascertained.

(d) *Arrest of development.*—This sometimes occurs after the testicles have passed into the scrotum, so that these glands remain permanently in their infantile condition. No general cause has been discovered for this abnormality.

2. **Atrophy.**—Wasting of the testicle may result from inflammation, from lesions of the spinal cord caused by injury or disease or secondary to injuries of the head. It may also be produced by early and excessive venereal excitement; or by deficient blood-supply, due to aneurysm or other causes. It is frequently found associated with varicocele, and is also caused by the pressure of the fluid of a long-standing hydrocele. It follows division of the spermatic arteries and veins, or of the vas deferens.

3. **Chondroma.**—Cartilage, either cellular or hyaline, but sometimes of the fibrous variety, is found in association with many new-growths in the testicle. Pure chondroma is comparatively rare; and the cartilage is usually associated with sarcoma, which is composed of spindle-cells. It has been seen in association with carcinoma.

DESCRIPTION.—This form of tumour of the testis occurs as disseminated nodules, connected by fibrous tissue; or as elongated masses with branching processes. By compression of the seminal tubules, it leads to dilatation of other parts of the tubules; and by invagination of the walls of such dilatations the growth often seems to be in the interior of a tubule. Careful examination, however, will always prove it to be of extra-tubular origin. It also invades the lymphatics, and through them has a great tendency to infect other parts of the system. It is often associated with myxoma; and sometimes, though

rarely, it develops into bone. It can only be diagnosed with any certainty when the tumour has attained a large size, and is then characterised by the hardness and slow growth of the mass.

TREATMENT.—Castration is the only treatment; and in consequence of the tendency of this disease to invade other organs, it is usual to recommend an operation without unnecessary delay, in all cases of large sarcocele which do not give any indication of yielding to treatment.

4. **Cystic Disease.**—Cysts are frequently found in the testicle, either separately or associated with other growths.

DESCRIPTION.—In true cystic disease or simple cystoma, the whole or part of the body of the testicle is replaced by a closely aggregated mass of cysts, of very variable size. Some are so minute as only to be visible on microscopical examination, while others may attain a diameter of from one to three centimetres. When only part of the gland is so affected, healthy glandular substance is found at the periphery, enveloping the cystic growth. The cysts have no thick fibrous wall, and are lined with shallow cylindrical epithelium, which is sometimes ciliated. They are filled with either clear watery, or sometimes very viscid, fluid; or with atheromatous matter, resembling the contents of a sebaceous cyst. These latter are usually dermoid cysts and owe their origin to developmental causes. Dermoid cysts containing teeth and hair are also met with. Very frequently nodules of cartilage are found interposed between the cysts. The disease usually occurs in adults, but one case has been recorded where the enlargement was first observed at the age of three months.

There is another form of cystic disease, where the cysts are separated by a considerable quantity of gelatinous connective tissue, and often contain polypoid ingrowths, which sometimes completely fill their cavities. This form is regarded as cystic adenoma of the gland, and may exhibit a varying degree of malignancy. A true malignant cystic sarcoma of the testis is also met with.

Cysts of the epididymis are described in the article on HYDROCELE. The origin of many of the cysts of the hilum of the testis and epididymis from the remains of the Wolffian body needs to be further elucidated.

SYMPTOMS.—Cystic disease of the testicle is usually attended by very little pain. The tumour is of an oval form, either with a smooth surface, or with irregular elevations; and does not attain a very considerable size, being generally from five to ten centimetres in diameter. There is an indistinct sense of fluctuation, unequal at different parts.

DIAGNOSIS.—Cystic disease of the testis has sometimes been mistaken for hydrocele or hæmatocele. The form of the tumour, its relatively greater weight, the absence of transparency, and the impossibility of detecting the body of the testicle at any part, distinguish it from hydrocele. The distinction from hæmatocele is in some cases more difficult. An exploratory incision will clear up the diagnosis.

TREATMENT.—Castration is the only remedy for this condition.

5. **Fibroma.**—Fibrous tissue is found in abnormal quantity in atrophy of the testis, in chronic orchitis, and associated with new-growths. By fibroma of the testicle, however, is meant a new-formation of fibrous tissue to a considerable extent,

without any other important change. In structure it resembles fibrous tumours of the uterus. It so rarely occurs, however, as to be practically devoid of clinical importance.

6. **Hernia Testis.**—This morbid condition will be referred to under the heading of tubercular orchitis (p. 1684). It also ensues in rare cases upon the softening of gummata. A third variety, fungus hæmatodes, is sometimes described. It is caused by the fungation of a malignant growth, and is now seldom seen. A fourth may be caused by traumatism, such as sloughing of the scrotum after injuries or operations for the radical cure of hernia, of varicocele, or of hydrocele—all accidents which are becoming increasingly rare.

DESCRIPTION.—It consists of a fungous protrusion from the scrotum, of a red or yellowish-red colour, and varies from one to five centimetres in diameter. There are two varieties, which may be distinguished as *superficial* and *deep*. The superficial form springs from the visceral layer of the tunica vaginalis, and is very comparable to the fungous granulations occasionally met with in cases of suppuration, or in wounds of the synovial sheaths of tendons. In this the tunica albuginea is intact, but probably altered in structure. In the deep form the tunica albuginea has been perforated, and the protruded mass consists largely of seminal tubules. It cannot be regarded as an evidence of any special disease of the gland, as it may occur, but by no means necessarily, after any form of orchitis in which there has been suppuration.

TREATMENT.—The treatment depends upon the cause. Tubercular and malignant disease of the testis will usually demand castration. Syphilitic orchitis is more amenable to treatment with mercury and iodide of potassium. Locally the protrusion may, in suitable cases, be treated with caustics, red oxide of mercury, or nitrate of silver, or with the galvanic cautery. Some anaesthesia with cocaine should be induced before any of these applications. Hernia of the testis has become excessively rare, and many of the palliative measures which used to be attempted are now forsaken.

7. **Hypertrophy.**—When only one testicle has been retained in the abdomen, the other sometimes attains an unusually large size. Such cases of, as it were, compensatory development are, however, the exception and not the rule.

8. **Inflammation.**—A. *Acute.*—When acute inflammation attacks the body of the gland solely or chiefly, it is called *orchitis*; when the epididymis, *epididymitis*. For the comparatively rare cases in which the vas deferens, or this duct along with the other structures of the spermatic cord, is affected, without the testicle being implicated, the barbarous hybrids *deferentitis* and *funiculitis* have been coined.

ÆTIOLOGY.—Acute inflammation may be caused by direct violence, or by the extension of inflammatory processes from the mucous membrane of the urethra. It may also occur, though rarely, as a sequela in typhoid fever or small-pox, or in pyæmia. Orchitis is not infrequently a concomitant of parotitis or mumps.

Of these varieties, the most frequent is gonorrhœal epididymitis. This was at one time regarded as an instance of 'sympathetic inflammation.' Careful examination will, however, always prove that the vas deferens is also affected, though sometimes in so slight a degree that its participation in

the inflammation might easily escape notice. This form is, therefore, due to direct extension of the inflammation from the urethral mucous membrane. Orchitis associated with mumps used to be attributed to such vague abstractions as 'sympathy' or 'metastasis.' Kocher, however, considers it to be the result of urethritis. According to this experienced observer, the disease commences as stomatitis, by which the parotid, or sometimes the sub-maxillary and neighbouring lymphatic glands become infected. The morbid material is carried by the blood to the kidneys, and in its course through the urinary passages sets up cystitis or urethritis, and thus the orchitis results. This explanation is not satisfactory, since in some epidemics of mumps orchitis has occurred prior to or without any affection of the parotid gland. We may, however, safely infer that an infective agency is concerned in its causation. Orchitis and epididymitis sometimes occur in rheumatic or gouty subjects. Occasionally cases are met with in which no exciting cause can be discovered. It sometimes occurs in very young children, and, in the absence of any constitutional taint or other discoverable cause, probably results from some accidental injury in these cases.

SYMPTOMS.—The symptoms of orchitis are local pain and swelling, with, in cases of orchitis sometimes, and in cases of gonorrhoeal epididymitis frequently, redness and tension of the corresponding part of the scrotum. Severe lumbar pain is in some cases felt, especially by labouring men, who apply for relief on account of some supposed sprain or injury, being ignorant of, or attaching no importance to, the affection of the testicle. This is probably due to inflammation of the lumbar lymphatic glands, with which the lymphatics of the testicle freely communicate; but it may possibly be an example of 'referred sensation.'

PROGNOSIS.—The prognosis is good. The inflammation usually subsides speedily, and leaves the gland in a healthy condition. Atrophy sometimes results after inflammation associated with mumps, or caused by severe contusion. Chronic induration of the epididymis may persist; but after some months it usually disappears. Stricture of the epididymis, or of the vas deferens, is very rare. Suppuration does not occur except in pyæmia, or after the eruptive fevers, or in tubercular orchitis.

TREATMENT.—Rest in the recumbent position, with the scrotum supported by a crutch-pad, and the application of ice locally, are in ordinary cases sufficient. Where rest is impossible, well-adjusted strapping of the part affords considerable relief, and promotes absorption of the products of inflammation so rapidly as not infrequently to necessitate the re-application of the strapping within twenty-four hours. Attention to diet, and avoidance of all violent exercise, will be requisite. The acute pain of the early stages is relieved by tartarated antimony and free purging. In more protracted cases mercury, taken in small doses internally, or applied locally by inunction or on strapping, will be found of benefit. Small and repeated doses of quinine and of iodide of potassium are a valuable adjunct. The practice, of late years recommended by some eminent surgeons, of puncture or incision of the ordinarily inflamed gland, is seldom necessary. If suppuration, however, occur, a free incision should be made as early as possible.

B. Chronic Inflammation.—Chronic orchitis may sometimes be the result of an acute attack, but is much more frequently induced by *syphilis*, *tuberculosis* or *gout*.

(a) *Syphilitic Orchitis.*—This variety may occur in young children who are the subjects of inherited syphilis, in the form of hard nodules in the testicle. In adults it belongs to the gummatous stage of the disease, and is very often symmetrical. It is usually painless, the patient being frequently ignorant of its existence. The gland is enlarged, very hard, insensitive to pressure, and often nodular in form. A gummatous testis has not infrequently been mistaken for malignant disease and excised. The diagnosis may be exceedingly difficult, but treatment with mercury and iodide of potassium usually sets the doubt at rest.

During the period of general eruption syphilitic orchitis sometimes commences in an acute form. The testicle is enlarged, very tender on pressure, with dull aching pain limited to the gland or radiating to the groin and back. There is usually effusion in the tunica vaginalis, varying in quantity in different cases, and the scrotum is congested and cedematous. Generally only one testicle is affected, but after some weeks the other gland may also present similar symptoms. The condition resembles gonorrhoeal epididymitis, but may be distinguished from it by the absence of any existing or precedent urethral discharge, by the body of the gland being solely or chiefly affected, and by the existence of concomitant syphilitic symptoms. It rapidly improves under mercurial treatment, but if left untreated generally passes into the ordinary chronic form.

TREATMENT.—This form of orchitis is usually very amenable to treatment, but has a tendency to recur. Iodide of potassium combined with mercury in small doses internally, when it can be tolerated, and strapping locally, will in most cases produce rapid disappearance of the disease, for a time at any rate. In obstinate cases intramuscular injections of mercury are of great value. Atrophy may sometimes result; and in some cases suppuration, with the formation of troublesome sinuses, may occur. In some cases the gland has, after very prolonged and unsuccessful treatment, to be removed.

(b) *Tubercular Orchitis.*—Tuberculosis nearly always begins in the epididymis, and may spread thence along the vas deferens to the vesicula seminalis. It ultimately attacks the rete and body of the testes. It is, however, unusual for tuberculosis to affect solely the genito-urinary organs, so that other foci may be found in the lungs, cervical lymphatic glands, or elsewhere. In children tubercular orchitis is not uncommon, and may begin in the body of the testis. It usually runs a rapid course, and is associated with tuberculosis elsewhere.

Tuberculosis runs the same course in the testicle as it does in other organs. After the deposition of tubercle-bacilli an inflammatory process takes place, and the inflammatory products tend to undergo fatty degeneration and caseation. The disease may continue at this stage for a long period, or the caseous nodule may become enveloped in a fibrous capsule, or calcification may in very rare instances occur. As a rule, however, the tuberculous mass softens and an abscess is the result. The pus may escape by extension of the inflammatory and degenerative processes through the back of the

epididymis to the scrotum. In the final stage the skin of the scrotum becomes inflamed and ulcerated, and the abscess empties itself, leaving, perhaps, a tubercular sinus. Or the inflammatory process may spread from the tunica vaginalis vera to the tunica vaginalis reflexa, causing an adhesion of those layers of serous membrane, and allowing the tubercular inflammation to reach the scrotum, with subsequent ulceration and escape of the pus. Not infrequently the escape of the pus is followed by a protrusion of granulation-tissue and testicular substance, constituting one of the commonest of the varieties of hernia of the testis.

CLINICAL PHENOMENA.—Tubercular disease of the testes is usually insidious in its onset. Pain is seldom a marked feature, and, perhaps by accident, an indolent, nodular swelling is felt in the epididymis or body of the testis. This nodule is hard to the touch, and can be handled without causing an excess of pain. After a while 'testicular sensation' is altered or lost—a point on which some lay great stress. In some cases tubercular disease of the testis runs a more acute and rapid course and quickly results in the formation of a tumour of considerable size, and with a decided tendency to soften, ulcerate, fungate, and become septic. In this class the general condition of the patient may be serious, owing to tuberculosis of the lungs, hectic fever, and the pain and discomfort of the scrotal tumour. Under such circumstances as these the removal of the testicle affords great relief, and is borne exceedingly well.

In most instances the vas deferens becomes inflamed and thickened, and tubercular nodules may form along its course. The vesiculæ seminales, bladder, prostate, ureters, and kidneys may become infected by extension along the vas deferens. A rectal examination is, therefore, always necessary, and an endoscopic examination of the urethra and bladder may be required.

DIAGNOSIS.—The diagnosis of tubercular orchitis is not, as a rule, difficult, but may present many difficulties. Gonorrhoeal epididymitis sometimes leaves indurations which have a close resemblance to those caused by tuberculosis. Furthermore, gonorrhoeal epididymitis may actually be followed by tuberculosis. Cysts of the epididymis, gummata, and malignant growths have also been mistaken for tubercle. In cases of doubt a test-dose of Koch's tuberculin may be administered.

Tubercular orchitis is sometimes a complication of general military tuberculosis. This variety does not require surgical intervention.

The tunica vaginalis may be full of inflammatory effusion, so as to obscure the tubercular nodules in the testicle. For purposes of diagnosis the fluid should be removed with a fine trochar and cannula.

TREATMENT.—When the tubercular nodule is small and localised, it may be explored under an anæsthetic, and thoroughly evacuated with a sharp spoon. The wound is rubbed with iodoform, and closed after the insertion of a temporary drain. If necessary this operation may be repeated. When the testicle contains several tubercular nodules, the operation which has just been described is unlikely to remove all the disease, or, if it did so, leave an organ of any size or utility. Under such circumstances as these castration is required. If the cord is healthy it may be divided at the external abdominal ring, but should doubt exist as to its healthiness, the inguinal canal should be opened so that as

much as possible of it can be excised. More extensive operations have been performed for the excision of the vas deferens and vesicula seminalis, but have not found favour in the eyes of British surgeons.

When an operation is contra-indicated the progress of the disease may be delayed by careful attention to the general health, cod-liver oil, malt-extract, carbonate of guaiacol, and abundance of fresh, dry air.

(c) *Gouty Orchitis.*—Chronic orchitis from gout can be diagnosed by the history of the patient, and yields readily to the ordinary treatment for gout, but is very apt to recur.

9. *Injuries.*—The testes are greatly protected from accidental violence by their mobility, and the laxity of the surrounding structures. Immediate death has resulted from severe contusions of the testicle, probably from reflex inhibition of the action of the heart. Contusions and wounds require appropriate surgical treatment.

10. *Carcinoma and Sarcoma.*—These are classed together, because, though histologically of very different origin, the distinction between them in any individual case is often impossible, except by microscopical examination of the tumour after removal. Even then the distinction is sometimes impossible, if we may judge from the description of recorded cases of mixed sarcoma and carcinoma.

Carcinoma originates in the epithelial structures of the gland, and is almost invariably of the encephaloid variety. Scirrhus occurs sometimes, but many specimens in museums classified under that heading are probably examples of fibrous sarcoma. Encephaloid cancer usually commences in the body of the testis, by the formation of one or more nodules. Sometimes the epididymis is first attacked. Very rarely is there general infiltration of the gland. In an early stage of the disease the testis is hard, from tension of the tunica albuginea, but when this has been destroyed in the progress of the growth, the mass is soft, and there is often distinct fluctuation. This may be unequal at different parts, from the presence of cysts. The growth is usually painless, but in some cases there is acute pain, either locally or in the lumbar region. The chief aids to diagnosis at this period are the rapidity of the growth, the enlargement of the blood-vessels of the cord, and the age of the patient. For while encephaloid cancer has been met with in young children and old people, still the vast majority of recorded cases have occurred between twenty and forty years of age. The writer has met with cases of malignant adenoma of the testis which closely resembled the Wolffian body in structure.

If the tumour attain a very large size, the scrotum may slough, and a bleeding fungus protrude. The disease has a great tendency to invade other parts of the system, and especially at an early period the lumbar lymphatic glands. This may lead to oedema of the lower extremities, from pressure on the abdominal veins. The inguinal glands generally escape infection, except in some cases where the scrotum has been involved in the disease. Secondary growths have been found in the mesenteric glands, liver, spleen, and lungs.

Sarcoma originates in the connective tissue of the testicle, and sometimes develops in both glands simultaneously. On microscopical examination two varieties can be distinguished, the round- and the spindle-celled. The latter grows more slowly, and

both are often associated with cystic and cartilaginous formations. The symptoms are very similar to those of cancer.

Sarcoma sometimes commences in the tunica vaginalis, and is then usually accompanied by extravasation of blood into the sac. Two such cases were met with in which the shape of the tumour, the complete absence of pain, the history of gradual enlargement, and the very distinct fluctuation were suggestive of hæmatocele. Exploratory examination, however, proved them to be cases of sarcoma. Castration was performed, and the testicles were found to be only slightly affected by the disease. Both cases died within a short period after the operations, from secondary affection of other organs, accompanied by similar hæmorrhages.

Melanoma of the testis was formerly regarded as a form of cancer, but is now considered to be sarcomatous. It is extremely rare, and in the few recorded cases of it similar growths were found in many other organs of the body.

DIAGNOSIS.—As a general rule it may be stated that sarcoma occurs most frequently under ten and over forty years of age; and that the epididymis is more frequently the primary seat of the disease, and, when secondarily involved, is attacked at an earlier period than in cancer. The distinction in any individual case must, however, be very uncertain, and is of little importance.

PROGNOSIS AND TREATMENT.—The prognosis is very unfavourable in both, as recurrence of the growth in other organs after the removal of the tumour is the rule to which there are but few exceptions. But hitherto an early exploration of doubtful tumour has hardly been practised by surgeons. The present writer (C. B. L.) has had much more favourable results by early intervention. It is now, with modern antiseptic precautions, much safer to explore doubtful tumours of the scrotum. Castration is the only possible treatment for both diseases.

II. Neuralgia and Irritability.—The testicle is sometimes the seat of very acute persistent or periodically recurring neuralgia. This must be distinguished from hyperæsthesia or irritability of the gland, which is occasionally associated with varicocele, or may be the result of self-abuse, excessive venery, or even of unsatisfied sexual excitement. Neuralgia may be due to some local cause, to varicocele, to a descending hernia, or to induration of some part of the glandular apparatus from precedent inflammation. It may be a transferred sensation, as in renal colic. Sometimes it occurs in cases of enlargement of the prostate, and more especially when prostatic calculi are present. The presence of a urethral stricture ought to be suspected and sought for. Occasionally no cause can be discovered, and we have to assume that it is due to some affection of the central nervous system. A family history of epilepsy or of lunacy would also have to be taken into consideration.

TREATMENT.—When of local origin, the treatment of neuralgia of the testis must be directed to the removal of the cause; and if all other methods fail, and the pain be severe enough to warrant it, castration may be required. When due to affections of other parts of the body, the treatment must be regulated accordingly.

Hyperæsthesia of the gland usually yields in time to tonics and attention to ordinary hygienic conditions.

12. Teratoma.—The testis, like the ovary, may be the seat of cysts containing hair, skin, bones, &c. The cysts are sometimes within, sometimes upon the gland. The more complex cases may be best explained as resulting from the inclusion of a second fertilised germ; while the simpler cases may be due possibly to the accidental inclusion of part of one of the proto-vertebræ within the rudimentary testicle. The history of a congenital tumour will suffice to direct attention to any such case. They are very rarely met with, and castration is the only suitable treatment. In this class we may include some of the cystic tumours originating near the hilum of the testis, which, in addition to the cysts, contain cartilage and muscle of both the smooth and striped varieties. The cysts in these cases originate, it is thought, in Wolffian remains.

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TETANUS.—SYNON. : Lock-jaw ; Fr. *Tétanos* ; Gr. *Starrkrampf*.

DEFINITION.—Tetanus is an acute infectious disease due to a specific micro-organism, and is characterised by painful tonic spasms, which at first affect certain groups of muscles, but tend to become generalised, and are accompanied by exacerbations of a paroxysmal character.

ÆTIOLOGY.—Though in many cases of tetanus no wound or focus of infection has been discovered, yet the modern conception of the nature of the disease compels us to the belief that in all alike the infective material must have gained admission through some breach of surface. The term 'idiopathic tetanus' is now used merely to indicate that the primary focus has not been found. In the majority of cases a wound or a recent cicatrix is present or a definite history of a wound can be obtained.

The primary lesion in tetanus may be of the most varied character, but the commonest forms are punctured, contused, or lacerated wounds, and the most frequent sites for these are the extremities. As the causal agent is a constant inhabitant of certain soils it is easily understood how these are the parts most commonly infected. Dirty wounds, especially those which have been contaminated with soil, and which have been neglected or which are difficult to cleanse, are the most fruitful source of the disease. Rarer cases have been described as following tooth-extraction, hypodermic injection, ligature of piles, frost-bite, and fractures. It has also occurred as a complication of typhoid fever (Rose); and such cases help us to understand how easily the focus of infection in 'idiopathic' tetanus may have been overlooked. Tetanus is commoner in hot than in temperate climates, and in the dark-skinned than in the Caucasian races.

Epidemics of tetanus have been described. Indeed their occurrence suggested the infective nature of the disease long before its actual demonstration. In certain wars tetanus has been a frequent complication of wounds, and there appears to be no doubt that excessive exertion, great fatigue, and exposure to cold are important predisposing causes. The fact that the wounded frequently lie on the ground for some time, and that their wounds are apt to be contaminated by dust and soil, are important factors to be noted in this connection. In emphasising a chill as a predisposing cause of tetanus Macnamara says 'it is a matter of common observation in Bengal that after sudden changes of

temperature cases of tetanus appear among surgical patients.' The introduction of *antisepsis* has almost abolished the disease as a result of surgical operations. In the Transvaal war (1899-1901) it appears to have been almost unknown. It is no doubt in most part due to the same cause that there has been great diminution in the number of puerperal cases, and of cases of the tetanus met with in new-born children (*tetanus neonatorum*) which occurred in epidemic form in certain West Indian islands and in St. Kilda. Of the domestic animals the horse is most susceptible to tetanus.

Isolation of the Bacillus.—The bacillus of tetanus was first successfully isolated and grown in pure culture by Kitasato in 1889. To isolate the bacillus, pus from the wound in a case of tetanus is inoculated on the surface of sloping agar-agar or solidified blood-serum. The tetanus-bacillus grows along with the other organisms present. After forty-eight hours' growth in the incubator the culture is subjected to a temperature of 80° C. in a water-bath for an hour. As a rule only the spores of the tetanus-bacillus survive this treatment. Plate-cultures on gelatine or agar-agar inoculated from the heated material and grown in an atmosphere of hydrogen give a growth of the tetanus-bacillus.

Morphology of the Bacillus.—The rods vary in size from 2-5 μ in length and from .5-.8 μ in breadth. The round or ovoid spores may reach a diameter of 2 μ . They are most frequently situated on the end of a rod, giving rise to a characteristic pin or drumstick form, which, however, is not strictly limited to the tetanus-bacillus. Filaments which may form spores, and have been described as sometimes branching, are often present. The bacillus is motile and possesses numerous cilia, which may be situated at the ends or round the rod. The bacillus is readily stained by any of the ordinary aniline dyes, and also by Gram's method.

Habit of Growth.—When newly isolated from the animal body the bacillus grows only in the absence of free oxygen, but after long cultivation on artificial media it tends to become less strictly anaërobic. With this change loss of virulence usually occurs. On the other hand, when grown under anaërobic conditions the organism retains its virulence well for years.

Races of the tetanus bacillus which grow best under aerobic conditions have been described by several observers, but these are exceptional. Aërobic bacilli growing in association with the tetanus bacillus may favour its growth by absorbing oxygen. It grows best at a temperature of 36°-38° C., the spores appearing at the end of thirty hours. Below 14° C. its growth ceases; between 20° and 25° C. it is very slow, and spores do not appear for seven or eight days. The spores show great resistance to heat, to drying, and to antiseptics. A moist heat of 80° C. for an hour does not kill them, and kept in a dry condition they retain their vitality for years. Water or steam at 100° C. kills them in five to fifteen minutes; steam at 115° C. in five minutes. They resist the action of carbolic acid (5 per cent.) for ten hours, and a solution of corrosive sublimate (1:1000) for several days.

Cultural Characters.—Only the most striking features can be described here. Grown on gelatine-plates in an atmosphere of hydrogen the colonies appear as minute grey specks with spike-like projections from their margins. Under a low magni-

fication these projections are seen in the form of columns of fine wavy threads running out from and forming a halo round the central mass of the colony. After a time the gelatine is liquefied. Deep puncture-cultures in agar show no growth at the surface of the medium, but in the depth along the needle-track a line of growth takes place, and from this radiate delicate processes, the whole resembling the outline of a fir-tree.

When sugar is present in the medium gas is evolved. Certain substances, such as glucose and sulphindigotate of sodium, when added to the media aid the growth by acting as reducing substances.

Distribution.—Not only is the tetanus-bacillus found in the wounds of men and the lower animals suffering from tetanus, but it has a wide distribution in nature. It is common in earth, especially the soil of cultivated land, and in the dust of cities and dwellings. It is constantly present in the dejecta of the herbivora, especially of the horse, and is not infrequent in the fæces of the healthy human subject.

Relation of the Bacillus to the Disease.—The tetanus-bacillus when introduced under favourable conditions into a wound multiplies in its depth, and here elaborates its toxin or poison, which, becoming diffused, attacks the central nervous system, for which it has a special affinity, and thus gives rise to the spasms which characterise the disease. The bacillus remains localised at the focus of infection; it is not distributed throughout the body in the blood or organs. Nicolaïer, arguing from this fact, formulated the correct hypothesis of its method of action even before the demonstration of a toxin. The fact that the tetanus-spore is so widely distributed in nature, and that, notwithstanding, tetanus is a rare complication of wounds, requires explanation. Vaillard and Rouget have thrown light on this by demonstrating experimentally that the spores of tetanus deprived by heat of all toxin do not develop in healthy tissues, and that certain conditions are necessary for their development. If there are introduced with the spores wood-splinters, other microbes, or substances such as lactic acid, or if the spores are surrounded by a collodion sac, then they grow and give rise to tetanus. The favouring influence of these substances is supposed to be due to an interference with the action of the phagocytes. In the case of wounds in the human subject the organisms carried in with the tetanus-bacillus probably play the most important part in aiding its growth.

Tetanus-toxin.—The fact that the tetanus-bacillus produces a toxin in artificial media was first demonstrated in 1890 by Knud Faber. He found on filtering a broth-culture of the bacillus through a porcelain filter that the filtrate, which was free from all formed elements, could even in minute doses produce all the symptoms of tetanus in experimental animals. These results were confirmed by Tizzoni and Cattani, who concluded that the toxin was of the nature of a soluble ferment or enzyme. Brieger, by using chemicals such as alcohol and ammonium sulphate, precipitated from cultures a substance which gave rise to tetanus, and which he regarded as a toxalbumin. It is probable that these precipitates only carry down albuminous substances in which the real toxin is entangled. Buchner showed that the precipitate obtained by saturating with ammonium sulphate, when kept in the dry state, was more stable than unprecipitated toxin. This

fact has been used by Knorr and Behring to obtain a stable test for the standardising of antitoxins. Such toxins may be so powerful that 0·000,000,04 grammes is sufficient to kill a mouse.

Kitasato stated that five minutes at 65° C. destroys the toxin, but Vaillard and Rouget have shown that an hour at 80° is necessary to remove the tetanising properties completely. The liquid toxin rapidly loses strength when exposed to sunlight and air, and even when kept in the dark *in vacuo* undergoes changes similar to those occurring in diphtheria-toxin, non-lethal toxoids being formed. In two months it may be reduced to one-fifth of its original strength. Tetanus-toxin has thus many points of resemblance to the soluble ferments: it is difficult to dialyse, is soluble in water, is precipitated by alcohol and tends to adhere to precipitates, is modified or destroyed by the action of air, sunlight, and comparatively low temperatures, and requires an incubation-period for its action on animals.

The injection of toxin produces in many animals the symptoms of tetanus as they occur spontaneously, or from the injection of cultures of the bacillus. Different species of animals have different degrees of susceptibility, which have been accurately worked out by Behring. The horse, probably next to man, is the most susceptible. Of experimental animals, the guinea-pig, mouse, and rabbit are most frequently used. The frog is more susceptible when kept at the temperature of warm-blooded animals than at lower temperatures. The tortoise is completely insusceptible and retains the toxin for several months unchanged, so that its blood is capable of producing tetanus in mice (Metchnikoff). Only one case of injection of tetanus-toxin into man has been recorded. M. Nicolas accidentally pricked his thenar eminence with the needle of a Pravaz syringe which was only moist with tetanus-toxin. After an incubation-period of four days a severe attack of tetanus supervened. The muscular spasms began in the hand but became generalised. Recovery took place after forty-one days. If one can judge from this single case man must be reckoned among the most susceptible of animals.

Between the injection of tetanic toxin into an animal and the development of the symptoms, there is always an incubation-period, which varies in length with the species of the animal and with the dose of toxin. In the mouse, rabbit, and guinea-pig it varies from 8 to 36 hours, in the fowl from 4 to 9 days, in the horse from 5 to 7 days. Even if one injects into a guinea-pig a dose of toxin many thousand times the minimal lethal dose, there is always an incubation-period of several hours. In such cases the disease, when it appears, runs a very rapid course, and it may be stated that as a rule the shorter the incubation the more severe and rapidly fatal is the disease. Tetanus-toxin, in requiring a latent period, differs markedly from such poisons as strychnine.

In the guinea-pig, rabbit, and mouse, after subcutaneous injection of toxin, spasms of the muscles first occur near the site of inoculation. With small doses of toxin the condition may remain localised and recovery may take place. With larger doses the muscular spasms become generalised and death usually occurs. Intravenous injection causes generalised spasms.

Roux and Borrel, by injecting tetanus-toxin into the cerebral cortex of animals, have succeeded in producing a distinctive form of tetanus, 'cerebral

tetanus,' which is chiefly characterised by psychical phenomena. The animal appears to labour under hallucinations, has periods of excitement, during which it runs wildly about, and epileptiform crises, alternating with periods of repose. Animals actively or passively immunised are not protected against this cerebral tetanus, even though their blood-serum is strongly antitoxic. Ehrlich has shown that, besides the tetanising substance in tetanus-toxin, another body, tetano-lysin, is present, which has the property of destroying or laking the red blood-corpuscles, and that an antilysin which neutralises this hæmolytic action is present in tetanus-antitoxin.

The affinity long suspected by clinicians to exist between the tetanus-poison and the nervous centres has been demonstrated experimentally by Wassermann and Takaki in a tangible and striking manner. If the brain or spinal cord of a healthy animal is emulsified and added to tetanus-toxin, the mixture is found to be no longer toxic when injected into a susceptible animal. 1 c.c. of brain-emulsion completely neutralises ten minimal lethal doses for the mouse. The protective action may be exercised even when the brain-emulsion is injected before or at different points from the toxin. They regard the experiment as confirmatory of Ehrlich's theory of immunity and attribute the neutralisation to the presence of preformed antitoxin in the nerve-cells. Roux, Metchnikoff, and others interpret the observation in a different manner. According to them the toxin is unchanged, but adheres to the nerve-cell like a colouring matter, and the phagocytes can deal more readily with the poison when thus fixed. The toxin reaches the nervous centres by two paths. One of these is along the course of the nerves, and hence probably the local muscular spasms in experimental animals, and the affection of the facial nerve in cephalic tetanus. Marie has proved experimentally that resection of part of a nerve prevents the onset of tetanus, if the toxin is injected into the paralysed muscle. The other path is by the blood-stream.

MORBID ANATOMY.—Hæmorrhages into the anterior cornua of the spinal cord and chromatolysis of the nerve-cells, as stained by Nissl's method, have been described by Marinesco as the essential changes in the disease. Goldscheider, Flatau, and others have found these same changes in strychnine-poisoning, and have failed to find them in tetanus in certain species, such as the dog. They therefore maintain that they cannot be held to be characteristic of tetanus only.

EXPERIMENTAL BASIS OF THE ANTITOXIC TREATMENT.—Behring and Kitasato were the first to show that susceptible animals could be rendered immune to tetanus-infection and that the blood-serum of such immunised animals possesses protective and curative properties when injected into other animals. Their first experiments were conducted on rabbits, and they succeeded in immunising a rabbit so that it could resist twenty times the lethal dose of toxin or of culture. They found that 0·2 c.c. of the serum of this immune animal injected into a mouse conferred immunity on it, and that tetanus-toxin, when mixed with this serum, was deprived of its toxic action. They also stated that mice which had already developed symptoms of tetanus could be cured by injections of the serum.

These experiments first showed the possibility of conferring passive immunity and were the basis of

serum-treatment. With the view of obtaining serum in sufficient quantities for therapeutic purposes, the horse was selected. Though naturally extremely susceptible to tetanus it was found possible to obtain a very high degree of immunity. Various methods of immunising have been employed. Small and progressively increasing doses of filtered toxin may be injected, or toxin modified by heating or by the addition of iodine terchloride, or Lugol's solution. After several months of treatment the horse may attain such a high degree of resistance that many thousand lethal doses of toxin or culture are tolerated. The serum from such a highly immune animal attains wonderful *preventive powers*. 1 c.c. of a serum of moderate strength can protect 100,000,000 grammes weight of mice against a lethal dose, and much stronger sera than this have been obtained. Behring and Kitasato's statements as to the *curative action* of the serum in experimental animals have been much criticised, and the evidence goes to show that after the appearance of symptoms a very small proportion of animals can be saved by subcutaneous injection of even the most powerful serum.

In the treatment of experimental tetanus Roux and Borrel obtained very favourable results by injecting the antitoxin into the cerebral substance. Out of 45 guinea-pigs, already tetanic, treated in this way, 35 recovered; out of 17 by subcutaneous injections of much larger doses of antitoxin only 2 recovered; the 17 controls which received no serum all died. A few drops of antitoxin injected into the brain had much greater curative power than large quantities given subcutaneously or intravenously.

Tetanus in Man.—**INCUBATION.**—In tetanus the incubation-period varies from days to months. About one-third of the cases occur in the first week and nearly one-half in the second week after the injury (Rose).

SYMPTOMS.—The symptoms begin in man as a rule with the occurrence of tonic spasm of the muscles of expression. The eyebrows tend to be elevated and the angles of the mouth pulled outwards and downwards, thus giving rise to the risus sardonius, which may however be absent in well-marked cases of tetanus. Even before this the patient may complain of stiffness of the neck or jaws. The spasm of the muscles of mastication increases till the jaws may be completely locked. Deglutition is impeded by spasm of the muscles of the pharynx. The stiffness of the muscles of the neck may extend to those of the trunk, and in severe cases the muscles of the lower extremities are also involved, the knee- and ankle-joints being extended. The tonic spasm of the muscles involved is never quite relaxed, but in addition paroxysmal exacerbations occur during which the contractions are so violent that rupture of muscles may take place. These paroxysms are accompanied by agonising pain. There is great hyper-excitability to stimuli so that even a draught may excite a paroxysm.

The muscular spasm may be so great that the body and limbs may be perfectly rigid (*orthotonos*), or arched like a bow, the patient resting on his occiput and heels (*opisthotonos*). More rarely the body is bent to one side (*pleurothotonos*), or forwards (*emprosthotonos*). In the severest type the respiratory muscles are affected, the thorax being compressed as in a vice (Watson), and spasm of the glottis may give rise to asphyxia. Hunger, thirst,

and inability to speak are very painful features of the worst cases. The body is usually bathed in sweat.

The temperature may be normal or little elevated even in severe cases; on the other hand some cases have pyrexia throughout. After death the temperature rises and may reach 110° or even higher. The intellect generally remains clear to the last. The immediate cause of death is asphyxia, heart-failure, or exhaustion.

Head-tetanus or tetanus facialis.—This form is due to wounds on the head usually in the region of the facial nerve, and the symptoms of tetanus are associated with paralysis of that nerve on the injured side. The prognosis is better than in the ordinary form.

Tetanus or trismus neonatorum.—The focus of infection is at the umbilical cord. It is rare in this country, though not uncommon in the tropics. Difficulty in sucking is the most prominent symptom.

Puerperal tetanus most frequently occurs after abortion, abnormal labour, and the use of instruments. It differs from other forms only in the site of infection.

PROGNOSIS.—Most writers estimate the mortality as over 80 per cent. Rose from the statistics of 716 traumatic cases finds a mortality of 88 per cent. He also found from an examination of 73 cases that those with an incubation-period within one week had a mortality of 91 per cent., within two weeks of 81.3 per cent., within the three and four weeks of 52.9 per cent., and of very late cases 50 per cent.

The length of incubation is perhaps the most important indication in prognosis. If the spasm rapidly affects one group of muscles after another the prognosis is bad; if it affects only the jaws and neck the prognosis is more favourable.

Macnamara from a large experience relies largely on temperature: 'if it rises beyond 101° there is impending danger, and if it reaches 103° the case is one to cause the greatest anxiety.'

TREATMENT.—**Local.**—It is essential when possible to put an end to the further elaboration of toxin, and this may often be effected by removal of foreign bodies such as wood-splinters by excision, or thermo-cauterisation and subsequent antiseptic treatment of the wound.

Amputation is sometimes advisable, especially of fingers or toes, where these are seriously injured. For the local treatment of the wound, solutions of iodine have been recommended by Sahli in view of Behring's observations regarding the power this substance has to neutralise the poison. Tizzoni and Cattani have shown that nitrate of silver in 1-per-cent. solution is one of the most powerful of antiseptics against the tetanus-organism.

General.—The patient must be guarded against all disturbing influences, which tend to cause paroxysms, such as draughts, noises, and too minute clinical examinations. Where the feeding is difficult from locking of the jaws the food must be administered by the nose or rectum. With the view of alleviating the severity of the spasms the pharmacopœia has been ransacked, but the most experienced observers are doubtful whether the course of the disease is in any way influenced by the use of drugs. Opium, chloral hydrate, and bromide of potassium are most frequently employed, but curare, calabar bean, Indian hemp, belladonna and many

others have been used. The administration of chloroform is indicated where the paroxysms are very violent and exhausting. Baccelli claims to have obtained excellent results by subcutaneous injection of carbolic acid in 2-per-cent. solution. About 5 grains of carbolic acid are given per day divided into several doses to which $\frac{1}{2}$ grain of morphine may be added.

The antitoxin-treatment of tetanus has hardly fulfilled the high expectations that were originally formed of it. As a prophylactic it has been proved, especially in veterinary practice where it has been used under test-conditions, to be almost infallible. Thus Nocard has collected the results obtained during twenty-two months by sixty-three veterinary surgeons who used preventive injections before operations such as castration, &c., in 2,300 animals, or after wounds in 400 animals. The cases both operative and traumatic they selected from those in which from their experience they knew that tetanus was liable to occur. Not one of these 2,700 animals died of tetanus, whereas the same observers recorded during the same period 259 cases of tetanus in untreated animals.

In the treatment of declared cases, however, both in man and in the lower animals, the bulk of opinion can hardly be said to be so favourable. The reason for the failure in many cases is due to the fact that the disease can be recognised only when the toxin has already seriously injured the nerve-cells. Though the antitoxin fails to remedy this injury, there is no doubt that it neutralises the toxin which is being elaborated and is entering the blood.

It ought to be administered in large doses on the very first symptoms appearing; and to give it, as has often been done, in small doses or as a last resort is to court failure. Neglect of these two requirements has been the cause of much of the discredit into which the treatment has fallen. One hundred c.cm. of the serum should be at once injected, and this repeated on the second and third days, and even if the case appears to be progressing favourably another injection ought to be given ten to fourteen days after the first. Even allowing that the results have fallen short of the original anticipation, the statistics tend to show that in cases treated with antitoxin the mortality is lower than in cases treated by the older methods. Hewlett in 1895 out of 50 cases found a mortality of 32 per cent. More recently Koehler dealing with statistics of 96 cases treated by antitoxin found the death-rate to be only 34.4 per cent.; of the cases with an incubation under ten days the mortality was 43.8 per cent.; when the incubation was longer than that the mortality was only 18.5 per cent. The favourable results obtained by Roux and Borrel from intracerebral injection of antitoxin in animals in which tetanic symptoms had already appeared has led to the treatment by this method of a number of cases of tetanus in the human subject. A small hole is drilled with an Archimedean drill through the skull just in front of the motor areas, and the needle of the syringe, which is 2 inches in length and blunt-pointed, is inserted through the trephine-hole and pushed its whole length into the brain-substance. $2\frac{1}{2}$ c.cm. are injected very slowly into each side. Under strict asepsis the operation is well tolerated.

The writer has before him the results in 27 cases treated by this method: 7 cases recovered, 20 died;

a mortality of 74.7 per cent. The results, therefore, have not been encouraging.

SUMMARY.—The most hopeful line of treatment consists in (1) the subcutaneous injection of large doses of antitoxin as described; (2) excision and antiseptic treatment of the focus of infection; (3) the use of chloral hydrate in large doses, or chloroform to restrain the violence of the paroxysms. See SERUM-THERAPEUTICS.

GEORGE DEAN.

TETANY.—SYNON.: *Tetanilla*; Idiopathic Muscular Spasm; Fr. *Tétanos Intermittent*; Ger. *Tetanie*.

Tetany is a neurosis described originally by Dance in 1831, and more or less fully since under many names, especially in France. It is probably much more common in that country than in England, where it is very rarely met with. It is mostly a comparatively trivial and temporary malady.

ÆTIOLOGY.—Tetany is associated with no recognised organic changes in any part of the nervous system; and much uncertainty prevails in regard to its causation. Apart from rickets it occurs mostly between the ages of fifteen and thirty years, though it may show itself in older people, as well as in children and in infants. It occurs in either sex, but is more common among females. Persons of a neurotic temperament, or those whose constitutions have been disturbed or weakened from many causes, are specially liable. Rickets, teething, the establishment of menstruation, chronic diarrhoea, lactation, and the state of convalescence from many acute diseases, are all conditions which predispose to this affection; while exposure to cold and emotional disturbance seem to act as the most common exciting causes.

SYMPTOMS.—The morbid manifestations of tetany consist, in the main, of tonic spasms, frequently recurring for brief periods in one or other part of the body, painful in character, and unaccompanied by loss of consciousness. The attacks in different individuals vary widely, the spasms being sometimes quite local, sometimes involving many different regions of the body.

In the *slighter kinds* of attack, a numbness and tingling is felt in the fingers and toes, which speedily become fixed in tonic spasm. As the spasms strengthen, they may extend to higher parts of the limb and become painful. The fingers are drawn together and slightly flexed, the thumb is bent into the palm, and the wrist slightly flexed. The toes also are drawn together and towards the sole, the big toe being drawn under them. The dorsum of the foot is arched, and the heel pulled up, while the leg and thigh are more or less rigidly extended. One or more of the limbs may be affected in this way; or if all are implicated, it may be simultaneously or successively. This condition of things lasts for a few minutes, or even for an hour or two, accompanied often by severe pain along the nerve-trunks, and by some diminution of sensibility in the parts affected. When the attack is about to terminate formication sets in, as at the commencement of the spasm. After variable intervals the attacks are renewed, it may be in an hour or two, or only after several days. Such paroxysms may be frequent during several months; and, according to Trousseau, so long as a tendency to recurrence of the spasms exists, they may always be excited anew by simply tapping or 'compressing the affected parts, either in the course of their principal nerve-trunks, or over their

blood-vessels, so as to impede the venous or arterial circulation' (*facial irritability*). On the other hand, the application of cold to the parts affected frequently arrests the spasms for a time.

In the *more severe forms* of tetany, the attacks may begin in the manner above indicated in the upper extremities, next in the lower extremities, and then, while diminishing in the parts first affected, the spasms may extend more or less generally to the muscles of the trunk. The contractions are invariably more or less painful. The spasms may even spread to the facial muscles, so that the jaws may be firmly clenched, and speech greatly embarrassed. If the muscles of the larynx are involved, as well as those of the chest and abdomen, extreme dyspnoea may be induced. Still there is no loss of consciousness. These attacks may be of brief duration; or they may be extreme in degree, long-continued, and frequently repeated. In these latter severe cases there is slight elevation of temperature, with greatly quickened pulse, and a furred tongue. After some weeks or months the paroxysms usually become less severe and less frequent, and finally cease altogether.

DIAGNOSIS.—The diagnosis must be based upon the progressive character of the attacks; upon the fact that they begin in the upper and lower extremities, and after a time completely intermit; upon the absence of all loss of consciousness during the attack; and upon the fact of the possibility of re-inducing the paroxysms by pressure upon the nerves or vessels of the parts affected. These characters will suffice to distinguish the affection from tetanus, epilepsy, and hysteria.

PROGNOSIS.—The prognosis is usually favourable, the complaint gradually subsiding after a few months. Still, in very exceptional cases, the patient may die asphyxiated during one of the extremely severe attacks.

TREATMENT.—The treatment of tetany should in the main be directed to the improvement of the patient's general health, and the diminution of all debilitating conditions or causes of irritation. At the same time, we must endeavour to lessen the general mobility of the nervous system, by seeing that the patient obtains regular and sound sleep, as well as by the administration of one or other of the bromides in suitable doses, in combination with valerian, musk, conium, or other antispasmodic remedies.

H. CHARLTON BASTIAN.

THALAMUS OPTICUS, Lesions of. —
SYNON. : Fr. *Maladies des Couches Optiques*; Ger. *Krankheiten der Sehhügel*.

INTRODUCTION.—Diseases of the optic thalamus vary in their symptomatology, according as the lesion is strictly limited to the ganglion itself, or implicates also neighbouring structures.

In the former case it is apparently well established by numerous recorded cases that lesions, such as apoplectic cysts or areas of softening, may exist without producing any discoverable symptoms, in the domain of either motility or sensibility, general or special. This is more particularly the case when the lesions occupy the convexity or ventricular aspect of the optic thalamus.

But more frequently diseases affecting the optic thalamus implicate also, directly or indirectly, the corpus striatum, internal capsule, crus cerebri, or corpora quadrigemina. Owing to the community

of vascular supply between the corpus striatum and optic thalamus through the optico-striate arteries of Duret, embolism or rupture of these vessels leads to conjoint destruction, more or less extensive, of both ganglia, as well as rupture of, or pressure on, the fibres of the internal capsule. A hæmorrhage or embolism in this region produces hemiplegia of the opposite side of the body. But that the hemiplegia is not necessarily due to the lesion of the optic thalamus is clear from the fact that such lesions may exist without any motor paralysis whatever. It is, therefore, more logical to attribute motor paralysis, when it does occur in connection with lesions of the optic thalamus, to implication, direct or indirect, of the motor fibres of the internal capsule.

LOCALISING PHENOMENA.—It is a question whether, apart from considerations as to causation, there are any symptoms specially characteristic of hæmorrhages in the region of the optic thalamus.

It has been stated, and there is considerable clinical evidence to show, that lesions of the optic thalamus are characterised by paralysis of the emotional movements of the face, as in laughing and crying; while the voluntary movements, as in showing the teeth, are unaffected, or only to a slight extent. The evidence on this point is somewhat conflicting, so that the relation of the optic thalamus to the emotional movements has not yet been satisfactorily established.

The occurrence of anæsthesia on the paralysed side is more constant and more enduring when the lesion invades the optic thalamus and its neighbourhood than when it is confined to the ganglia of the corpus striatum. This is owing to the fact that the posterior fibres of the internal capsule are directly injured, and not merely pressed on, as in the latter case. The anæsthesia may involve the special senses as well as common sensibility, but more frequently the tactile sensibility only is distinctly impaired. The reflex cutaneous excitability is also greatly diminished, as has been shown by Crichton Browne. The paralysed limbs are frequently also affected with unsteadiness, tremors, or chorea-like spasms, intensified on volitional efforts. This affection, termed *post-hemiplegic chorea*, is generally, if not invariably, associated with a greater or less degree of impairment of sensibility in the affected limbs. It is doubtful how much, if anything, can be assigned to the lesion of the optic thalamus itself in the causation of these symptoms. But for regional diagnostic purposes they may be regarded as significant of lesion of the optic thalamus and its immediate neighbourhood. When the lesion involves only the posterior fibres of the internal capsule, lying external to the optic thalamus, the result is hemianæsthesia, general and special, of the opposite side of the body. The power of movement may not be apparently affected. If it be, the leg is, in general, relatively more affected than the arm. But, though the motility is retained, the muscular sense is lost, so that the patient is unaware of the state of contraction of the muscles or the position of the limb, and requires the aid of vision in guiding its movements.

Cases have been recorded which render it in the highest degree probable that lesions of the posterior aspect of the optic thalamus, and region of the corpora geniculata, cause hemianopsia towards the side opposite the lesion, from paralysis of both retinæ on the corresponding side. In an experimental destruction of the pulvinar thalami in a monkey,

there was found temporary total blindness of the opposite eye and hemianopsia in that on the same side. This observation taken in association with the fact that there is an obvious anatomical relation between the fibres of the optic tract and thalamus on the one hand, and of the occipital lobe and the pulvinar on the other, would point to a close connection between the optic thalamus and the sense of sight. The facts of secondary degenerations also show that a large number of ascending bulbar and pontine tracts, including probably those of common sensation, enter the optic thalamus before being transmitted to the cerebral cortex. Hence the symptoms which have been found to follow destruction of the optic thalamus by disease may be due to direct interference with the functions of this ganglion. On the other hand, it is clearly difficult to exclude some implication, direct or indirect, of the adjacent internal capsule. Therefore hemianopsia alone, without other symptoms, cannot be taken as absolutely diagnostic of lesion of the posterior aspect of the optic thalamus. Conjoined with affection of the other forms of sensibility, however, it points to lesion in this region.

Tumours of the optic thalamus, in addition to the general symptoms of intracranial growths (though sometimes these even seem to have been wanting), produce either no special symptoms, or such a variety as to render the regional diagnosis very uncertain or altogether impossible. The symptoms may be those indicative of lesion of the internal capsule, both its motor and sensory strands; or they may be such as have been observed in connection with lesions of the corpora quadrigemina.

A direct relation between lesion of the optic thalamus and the production of pyrexia has been maintained by some observers (Hale White, Ott.). Many experimental facts and numerous recorded cases, however, show that there is no constant relation between changes in temperature and lesions of the optic thalamus.

D. FERRIER.

W. A. TURNER.

THANET, Isle of, in Kent.—A dry, bright, bracing, marine climate. Principal resorts: Margate, Ramsgate, and Broadstairs. See CLIMATE, Treatment of Disease by.

THERMOMETER, Clinical (θερμῆν, heat; and μέτρον, a measure).—SYNON.: Fr. *Thermomètre*; Ger. *Thermometer*.

DEFINITION.—An instrument for measuring different degrees of heat or cold of the human body.

DESCRIPTION.—Clinical thermometers used in Great Britain and in the United States are graduated with *Fahrenheit's* scale, whereas on the Continent of Europe the *Centigrade* or *Celsius* scale is now everywhere used for medical and scientific purposes, the *Réaumur* scale falling more and more out of use. The difference between these three scales is this, that in the Centigrade and Réaumur scales the melting-point of ice is marked zero, and the boiling-point of water (or rather the heat of the steam of water boiling at an atmospheric pressure equal to 29·92 inches of mercury) marked 100° and 80° respectively; while Fahrenheit marked the former 32° and the latter 212°. Therefore 180 degrees of the Fahrenheit scale are equal to 100 degrees Centigrade and 80 degrees

Réaumur; and the relation of the three scales to each other is as follows:—

F.	C.	R.
9	:	5
	:	4

One degree of F. = $\frac{5}{9}$ C. or $\frac{4}{9}$ R.; one degree C. = $\frac{9}{5}$ F.

In converting degrees of the Fahrenheit scale into Centigrade degrees, it must, however, be borne in mind that zero of the C. scale corresponds to 32 of the F. scale; 32 must therefore be deducted in converting a certain degree of the F. scale into the corresponding degree of the C. scale, and 32 must be added when C. degrees are to be expressed by the corresponding degrees of F. The formulæ for these conversions are, therefore:—

$$x \text{ deg. F.} = (x - 32) \times \frac{5}{9} \text{ deg. C.}$$

$$x \text{ deg. C.} = (x \times \frac{9}{5}) + 32 \text{ deg. F.}$$

For instance:—

$$99\cdot5 \text{ F.} = (99\cdot5 - 32) \times \frac{5}{9} = 67\cdot5 \times \frac{5}{9} = 37\cdot5 \text{ C.}$$

$$39 \text{ C.} = (39 \times \frac{9}{5}) + 32 = 70\cdot2 + 32 = 102\cdot2 \text{ F.}$$

It will be convenient, for quick reference, to give the corresponding degrees of the Fahrenheit and Centigrade scales within the range with which human physiology and pathology are concerned, side by side:—

F.	C.	F.	C.
95·0	35·0	104·0	40·0
96·0	35·55	104·9	40·5
96·8	36·0	105·0	40·55
97·0	36·11	105·8	41·0
98·0	36·66	106·0	41·11
98·6	37·0	106·7	41·5
99·0	37·22	107·0	41·66
99·5	37·5	107·6	42·0
100·0	37·77	108·0	42·22
100·4	38·0	108·5	42·5
101·0	38·33	109·0	42·77
101·3	38·5	109·4	43·0
102·0	38·88	110·0	43·33
102·2	39·0	111·2	44·0
103·0	39·44	112·1	44·5
103·1	39·5	113·0	45·0

In thermometers for clinical use the degrees on the scale ought to be divided into fifths. Thermometers ought to be carefully compared from time to time with a standard instrument, as they are liable, after a certain time, to give abnormally high indications owing to the bulb gradually contracting a little. In England thermometers may be sent for comparison with a standard instrument to the Kew Observatory; in Germany to the Imperial Physico-Technical Institute, Sect. II., at Charlottenburg, near Berlin.

Of great convenience for clinical use has been the introduction of self-registering mercurial maximum thermometers. Casella was the first maker who constructed a registering clinical thermometer, by introducing a small quantity of air into the tube, and thereby separating a small part of the mercurial column from the rest. Instruments are now made in which the index—that is, the small separated part of the mercurial column—is prevented from falling back into the bulb, or in which an index is only formed each time the mercury rises out of the bulb. In using an instrument of this kind, the index, it need hardly be said, must be carefully shaken down below 95° or 90° before the thermometer is applied to the patient.

Another principle has been followed in the construction of very sensitive instruments for special researches on temperature, namely, that of the thermo-electric apparatus. The electric current that is produced in a circuit composed of two different metals when their point of contact assumes a different temperature from that of the other ends, or again, as first observed by Svanberg, the changes that a galvanic current shows when the resistance of a part of the circuit is altered by a change of temperature acting on it, can be measured by a galvanometer enclosed in the circuit. Gavaret, Heidenhain, and other physiologists have used the thermo-electric pile in physiological investigations in animals. J. S. Lombard, Hankel, Kunkel, and others, have applied it to observations in man. More recently a convenient form of thermo-electric apparatus for clinical purposes has been devised by Redard. The apparatus, constructed on the last-mentioned principle by O. W. Siemens, for measuring deep-sea temperatures, might also easily be adapted for clinical purposes.

A self-registering apparatus for continuous observations, on the principle of an air-thermometer, has been constructed by Marey; and an instrument automatically registering the changes of temperature on the surface of the body during a certain time was devised by W. D. Bowkett. A very neat little metal thermometer in the shape of a small watch, showing the movements of a 'Bourdon tube,' which are caused by variations of temperature, by the hand moving on the watch-face, has been constructed by Immisch. This can be placed in the axilla or between any folds of the skin, on the surface of the body or inside the mouth, and will correctly indicate the temperature. By a special arrangement the hand can be fixed before removing the instrument, which then acts as a maximum thermometer. But, although convenient for isolated observations and under special circumstances, it is less cleanly, and cannot for general clinical purposes compare with the ordinary glass thermometer, which, it need hardly be said, it surpasses in solidity.

For measuring surface-temperatures, mercurial thermometers of special shape, namely, a long cylindrical bulb coiled up in one plane at a right angle to the stem, have also been constructed. A thermo-electrical apparatus, or Bowkett's or Immisch's instruments, are, however, more sensitive and more convenient for that purpose.

Applications of the Thermometer. — The object we generally have in view with clinical thermometry being to examine as nearly as possible the temperature in the interior of the body, or the blood-heat, which is less variable than that of the surface (*see* TEMPERATURE), the localities most suitable for applying the thermometer would be the natural cavities, or the openings by which a thermometer might be introduced to a certain depth into the interior of the body. In the rectum, vagina, or bladder, the temperature is not subject to the ordinary changes acting from without, and the time required for taking an observation with the thermometer in any of these localities would be only such as is necessary for raising the temperature of the mercury to that of the surrounding mucous membrane. This time might be materially shortened by previously heating the thermometer to a degree a little below or above that to be expected in the body. With this preliminary measure an observa-

tion of the temperature in the rectum or vagina will not take more than half a minute.

The case is very different if we take the temperature in a cavity of the body which is not always closed, such as the mouth; or in the axilla, which can be formed into a closed cavity only by placing the arm closely against the chest. Here, quite independently of the sensitiveness of the thermometer, the time required for an observation is much longer, because the temperature of the mucous membrane of the mouth, or of the skin of the axilla, begins itself slowly to rise after the closing of these cavities, until it is raised to that of the deeper tissues which are not exposed to the loss of heat from without. Whereas nine to eleven minutes on an average are required for an observation of the temperature in the mouth, ten to twenty-four may be necessary for the mercury to become stationary in the axilla. The time varies also according to the state of the general circulation. It will be found much longer in persons with a weak circulation, for instance in a case of heart-disease, than in the case of a vigorous patient with a good circulation and with febrile heat. It is evident that, as was first pointed out by Liebermeister, the time for an observation in the mouth or axilla can be materially shortened, not so much by previously heating the thermometer, as by, previously to the introduction of the latter, keeping the mouth or axilla closed for ten to fifteen minutes. These cavities will then have assumed a steady temperature, and the time required for the observation will only be that necessary for raising the temperature of the mercury and the glass to the temperature of the surrounding parts. It is, therefore, a good plan, if the patient had been lying on one side, to turn him over to the other, or to make him lie on one side for a time before the thermometer is introduced, and then to place the thermometer in that axilla which had been closed by the position of the patient. If the skin of the axilla be very wet with perspiration, it ought to be wiped dry before applying the thermometer.

For practical purposes the rule generally recommended in observations being taken in the axilla, to leave the thermometer until the mercury has remained stationary for five minutes—a rule which naturally applies to self-registering no less than to ordinary thermometers—secures sufficient accuracy; and this rule should be given to nurses and attendants to whom the observations are left. Especially in obscure cases, in which much depends upon the discovery of even a trifling elevation of the temperature above the normal standard, which may be of great importance for diagnosis, this precaution ought never to be omitted; and for observations requiring scientific accuracy, as, for instance, when the effect of some drug on the temperature of the body is being studied, the observations ought to be made by the physician himself.

For various reasons the axilla is the locality most suitable, and therefore generally used, for thermometrical observations. In very young or restless children, however, as well as in very emaciated adults, axillary observations would become untrustworthy. In such cases, or where patients are in an insensible state, or under special circumstances—for instance, when a great divergence exists between the axillary and the internal temperature, or when doubts arise as to the

correctness of an axillary observation—the rectum, or the vagina, may be used for applying the thermometer, and with a self-registering thermometer this can be done without unnecessarily uncovering the patient. In using the rectum, great care must be taken not to let a small instrument slip into it, and in restless children to prevent the instrument from being broken. This is best prevented by placing the patient on his side, and while the thermometer is kept *in situ* with one hand, letting the other one rest on the hip of the patient, in order to be able at once to arrest any turning movement which he might happen to make. The thermometer ought to be introduced about two inches deep into the rectum; and may, before being taken out, be gently pushed forward a little more, in order to bring the bulb in contact with a fresh part of the mucous membrane, which has not been cooled by the thermometer. When large masses of feces fill the rectum, the thermometer passing into them may indicate a somewhat lower temperature than when in contact with the mucous membrane.

Other places of application, such as the inguinal fold, or the fold of skin between the thumb and the second metacarpus, may be used for special, but are quite unsuitable for general, clinical purposes. The clinical thermometer ought always to be carefully washed after being used.

Thermometrical Records.—It is extremely useful to register on a chart the thermometrical observations in a case of disease, and to connect the marks by lines; the curves which are thus formed being quite typical in many diseases. On the same chart may be entered, also by marks and lines, or otherwise, the numbers of the pulse and respirations, as well as remarks concerning other symptoms, or the treatment. For observations made with the thermometer, see TEMPERATURE.

The use of the thermometer for estimating the temperature of rooms and especially of wards, is fully described in other appropriate articles. See NURSING THE SICK; and PERSONAL HEALTH.

C. G. H. BÄUMLER.

THIRD NERVE, Diseases of.—INTRODUCTION.—The third nerve is purely motor in function. It supplies the levator palpebræ superioris; the superior, inferior, and internal recti; the inferior oblique; and the ciliary muscle with the sphincter of the iris. It arises from the surface of the crus cerebri by a series of fasciculi, proceeding from a prolonged nucleus of grey matter which lies beneath the hinder part of the floor of the third ventricle and the aqueduct of Sylvius, and is continuous behind with the nucleus for the fourth nerve. From the experiments of Hensen and Voelcker, and some limited facts observed in man, there seems to be a serial representation of the several functions of the nerves in this tract of grey matter. The anterior portion innervates the ciliary muscle subserving accommodation; next is the centre for the sphincter of the iris, through which contraction of the pupil is produced. Behind these centres for the internal muscles are, successively, those for the levator palpebræ and the rectus superior; while that for the internal rectus is probably by the side of the latter; farther back are centres for the inferior rectus and inferior oblique, while behind the former is the centre for the superior oblique (fourth nerve), associated in function with the inferior

rectus. It is probable that the series of separate roots of the third nerve corresponds to this series of centres and of functions. We do not yet know whether the associated actions in convergence, and those of the lateral movements of the two eyeballs, are arranged in this nucleus or in adjacent centres. The fibres for the internal rectus seem to pass up to the nucleus of the third nerve from that of the sixth—at least those fibres for the internal rectus that are concerned in the conjoined lateral movement.

SYMPTOMS.—Morbid states of the third nerve are revealed by (1) Spasm, or (2) Paralysis in the muscles supplied by it, that is, those of the eyeball and the upper eyelid; in the iris; and in the ciliary muscle.

1. **Spasm.**—The functions of the fibres of the third nerve vary much, so as to render it equivalent to several nerves. Spasm is never met with at the same time in all the muscles supplied by it. Isolated muscles may be involved, especially the internal rectus, in conditions of irritation of the trunk and nucleus of the nerve, as in meningitis, and hysteria; also in hypermetropia, and in paralysis of the antagonist-muscle. When extreme, the eyeball is turned inwards, and cannot be moved out. Clonic spasm of the muscles occurs in ‘nystagmus,’ which may be present only during voluntary effort, or may be spontaneous. The elevator of the upper eyelid is occasionally spasmodically contracted, so that the eye cannot be shut, but remains widely or partly open (*lagophthalmos*). Some degree of this elevation is common in exophthalmic goitre, and may occur alone, as a partial manifestation of a similar state. Slight contraction of this muscle occurs also in cases of long-continued paralysis of the orbicularis palpebrarum. Spasm of the levator may also be due, in rare cases, to peripheral irritation in the region of the fifth nerve; it has been observed for several hours after the extraction of a tooth.

Contraction of the sphincter of the iris produces diminution of the pupil (*myosis*), sometimes to very small dimensions. It may result from irritation of the trunk of the third nerve; or from stimulation, central or reflex, of the nucleus; or it may be secondary to paralysis of the dilator fibres supplied by the sympathetic. It may result from excessive (associated) efforts at accommodation. It is, however, most frequently met with in locomotor ataxy, and is associated with loss of reflex action to light. The condition is described more fully in the next section. Spasm of the ciliary muscle may result from the other causes of irritation of the nerve-trunk, or from excessive efforts at accommodation in hypermetropia. Its effect is to produce a fixed accommodation for near objects.

TREATMENT.—The treatment of the central causes of overaction of the third nerve commonly resolves itself into that of the primary condition. Rest is important, and attempts to converge and accommodate should be, as far as possible, avoided. If no exciting cause can be discovered, the over-action should be reduced by sedatives, of which bromide of potassium is the safest. Sometimes belladonna may be added with advantage, but its effect should be watched. It is needless to say that distinct errors of refraction should be corrected by glasses. When a peripheral source of reflex excitation can be ascertained its influence may be lessened by injections of morphine or cocaine. Functional over-action is often the expression of weakness, and needs nervine tonics in addition to

the other measures. Counter-irritation, in the region of the related sensory nerve, is frequently of great service in this as in most other forms of functional spasm. Persistent convergence in hysteria can generally be removed by a small blister to each temple. Atropine will overcome spasm of the sphincter pupillæ or of the ciliary muscle. The cold douche to the eyeball is useful in spasmodic lagophthalmos, but many cases of this affection are extremely obstinate. The treatment of the retarded descent of the lid occurring in exophthalmic goitre, and now and then met with as an isolated symptom (or combined only with a quick pulse), is that of this disease as a whole.

2. Paralysis.—**ÆTIOLOGY.**—The commonest cause of paralysis of the third nerve is some affection of its trunk as it passes through the membranes at the base of the brain, through the orbital fissure, or along the orbit; due either to rheumatic inflammation of the nerve-sheath, to syphilitic inflammation of the nerve or membranes, or to injuries of various kinds. Less frequent causes are acute lesions—hæmorrhage, softening, inflammation—in the nucleus of the nerve, or at the inner part of the crus cerebri through which the fibres pass and from which they emerge; compression of this part by an aneurysm or a growth in the interpeduncular space; basal meningitis; and aneurysm of the termination of the internal carotid. A syphilitic growth, on the nerve or near it, may compress the fibres. Paralysis is also met with as a result of diphtheria and in association with disease of the spinal cord, especially locomotor ataxy. It occurs also, in rare cases, as part of multiple neuritis, after diphtheria and other acute specific diseases, and from some toxic influences. Sudden palsy of a branch, occurring during effort or cough, is probably due to an extravasation into the sheath of the nerve.

Two peculiar causal varieties deserve mention. In *relapsing palsy* the nerve on one side becomes paralysed subacutely, the whole, or more commonly some, of its branches suffering; it recovers imperfectly; and then the other nerve becomes similarly affected. Afterwards the palsy of the first returns, and some permanent affection of both nerves remains. This form is met with in the subjects of old syphilis. *Periodical or recurring palsy* is a peculiar variety, perhaps allied to megrim, in which transient palsy of the third nerve occurs at intervals during many years, sometimes even from childhood up to adult life; each attack being completely recovered from.

Occasionally all the muscles supplied by the third nerve become paralysed, together with the other orbital muscles—‘*ophthalmoplegia externa*’—from degeneration of the nuclei of these nerves; and hence it is also termed ‘*chronic nuclear palsy*.’ This is due to alterations in the grey matter similar to those which, in the spinal cord, give rise to progressive muscular atrophy. It is met with in association with tabes, and also alone; and in both cases is most common in the subjects of syphilis, being met with in both the acquired and inherited forms.

SYMPTOMS.—Paralysis may affect some or all of the fibres of the third nerve. When complete, the upper eyelid is dropped and cannot be raised, and the eye can be moved only outwards, or also a little downwards; after a short time it constantly deviates outwards. The pupil is in a mid-state between contraction and dilatation, and cannot be made to con-

tract by light; power of accommodation in the eye is lost, owing to paralysis of the ciliary muscle.

Each muscle supplied by the nerve may be paralysed separately, by an affection of its special branch of the nerve after it leaves the main trunk.

When the levator palpebræ superioris is affected, *ptosis*, or drooping of the eyelid, alone results. It is seldom an isolated symptom of disease of the third nerve, since the branch to the levator is seldom alone affected; but it occurs, with palsy of the associated superior rectus, from limited disease of the nucleus, and may then be bilateral. It is met with in partial disease of the nerve, whatever be its cause. It is also congenital. A form of ‘*morning ptosis*’ sometimes occurs, owing to a difficulty in raising the lids on first waking. In hysteria, ptosis may be simulated by slight contraction of the orbicularis, a mechanism which becomes distinct if contraction of the occipito-frontalis is induced by making the patient look up.

Paralysis of one of the three straight muscles supplied by the third nerve is indicated by symptoms which occur chiefly during movements in which the paralysed muscle is needed; they are the series of symptoms that result from paralysis of any ocular muscle, and which are elsewhere described in detail—limitation of movement, primary deviation, strabismus, secondary deviation of the unaffected eye, diplopia, erroneous projection of the field of vision, and often vertigo as the result of the last. See p. 1065.

In paralysis of the sphincter iridis, the elasticity of the structure maintains the pupil at middle size, and it can be further dilated by atropine, but all power of contraction beyond that degree is lost. When the ciliary muscle is paralysed, the power of accommodation is lost: the far point of vision remains the same, but the near point is rendered much more distant.

The remarkable loss of reflex action of the iris which occurs in association with locomotor ataxy is often accompanied by myosis. Then, the pupil does not dilate on stimulation of the skin (Erb). When the pupil is not small, this reflex dilatation is usually preserved. In loss of the light-reflex the contraction on accommodation and convergence is often preserved (Argyll-Robertson). Sometimes this is also lost, the ciliary muscle being completely paralysed—the ‘*ophthalmoplegia interna*’ of Hutchinson. Power of accommodation may be lost although the pupil still contracts on an effort at accommodation. These symptoms also occur in cases of old syphilis, apart from spinal disease. They probably then depend on limited degeneration in the nuclei of the third nerve, the result of the more limited action of a post-syphilitic toxin.

DIAGNOSIS.—Paralysis of the third nerve is generally obvious; only the slighter palsy of separate branches supplying the ocular muscles is sometimes not easy to recognise; and for this purpose, a careful examination of the double images is often necessary. The diagnosis of the cause is less easy. Rheumatic paralysis succeeds exposure to cold, and is often attended by much pain, but it is rare; in syphilis other cranial nerves are often affected independently; in meningeal and spinal disease there are the respective distinctive symptoms; in organic disease of the crus there is hemiplegia of the opposite side, coincident in onset with the affection of the third nerve; in interpeduncular disease the affection of the third nerve may precede the hemiplegia, but

both third nerves commonly suffer. After diphtheria the ciliary muscle is usually alone affected. In the pseudo-ptosis of hysteria, the contraction of the orbicularis can be rendered evident by making the patient try to look up; the contraction of the levator has to be counteracted by conspicuous contraction of the orbicularis.

PROGNOSIS.—When due to cold or to recent syphilitic mischief, or occurring after diphtheria, the prognosis of paralysis of the third nerve is good if proper treatment can be secured. In cases of organic cerebral disease it is subordinated to that of its cause. In association with spinal disease the ultimate prognosis is unfavourable, for, although the early attacks are usually recovered from, the affection often recurs, and each recurrence leaves some residue. As a general rule, however, the prognosis must be guided by the nature of the morbid process on which the palsy depends, the extent to which this can be influenced by treatment, and the duration of the changes in the nerve-elements on which the symptoms directly depend.

TREATMENT.—In rheumatic paralysis of the third nerve from cold, hot fomentations in the early stages and afterwards counter-irritation by blisters to the temple, with an alkaline diuretic or salicylates, should be employed. In all forms of acute or subacute inflammation it is wise to give small repeated doses of mercury. Subsequently tonics are the most useful. When the palsy is of syphilitic origin, iodide of potassium usually suffices to effect a cure. If associated with spinal mischief, strychnine, iron, and arsenic are occasionally of some service. In intracranial disease—tumour, aneurysm, or meningitis—the treatment is that of its cause. After diphtheria tonics are alone necessary; it is indeed a merely subordinate symptom in most cases. In paralysis of the sphincter pupillæ and ciliary muscle, occasional instillation of a small quantity of solution of eserine is said to do good, by stimulating locally the paralysed fibres, and also to be beneficial in affections of other branches of the nerve. In the paralysis of the ocular muscles electricity has been recommended, but is practically useless, whether an attempt is made to apply it directly or whether applied indirectly as to the temple or forehead. The only 'satisfactory' results of electrical treatment have been in syphilitic cases in which iodide of potassium was also given.

W. R. GOWERS.

THIRST.—**SYNON.** : Fr. *Soif*; Ger. *Durst*.—Thirst is a sensation indicating a necessity on the part of the system for an increased supply of water, as appetite shows there is a need for the introduction of food. Although the sensation is referred to the back of the throat, it is not a purely local feeling, as is proved by the fact, well known to physiologists, that it cannot be allayed by the swallowing of water, unless the fluid reach the stomach and be absorbed. It is always present in febrile disorders, an increased supply of liquid being required both to reduce the heat, by promoting the evaporation of moisture from the skin and lungs, and also to wash away the products of the increased tissue-changes that accompany these complaints. In like manner it is always present when much fluid has been abstracted from the system; thus, it shows itself after all surgical operations attended by hæmorrhage. It is a prominent symptom in cholera and

diarrhoea, in which diseases large quantities of serum are rapidly removed from the gastro-intestinal circulation, and equally so in diabetes mellitus, where fluid is largely excreted along with sugar by the urinary organs. A craving for cold and acid drinks presents itself in acute gastritis, the intensity of the thirst being perhaps due to the incessant vomiting, which prevents fluids remaining long enough in the stomach to be absorbed. In chronic gastritis thirst is usually present, and is chiefly complained of towards evening. It forms a useful diagnostic sign between this disease and mere atonic dyspepsia.

TREATMENT.—Thirst is relieved by the agents usually recognised as refrigerants, such as water, barley-water, toast and water, and similar drinks; sucking small pieces of ice; effervescing drinks; freely diluted acid drinks, especially those made with vegetable acids or phosphoric acid, alone or combined with a little aromatic bitter; the juices of fruits, or these made into drinks. Care has often to be exercised in the employment of these apparently harmless agents, and their consumption has to be checked, otherwise patients will take them to excess, and may thus do themselves considerable injury.

SAMUEL FENWICK.

THOMSEN'S DISEASE.—This is also known as Myotonia Congenita, an unsuitable name because it is not always congenital. The name of *transient myotonia* has been suggested, but it is unlikely to displace the more familiar name of Thomsen's disease—a name depending upon the fact that the most complete description of it, and one of the earliest, has been given by a physician of that name, who is also the subject of the disease. Its most striking symptom is a rigidity of muscles which is set up on first attempting to use them. With repeated efforts the spasm is gradually overcome, and when the muscles originally at fault are once brought under voluntary control, their normal activity can be maintained without difficulty.

The muscles most generally affected are those of the lower limbs. Sometimes, however, others suffer, e.g. those of the hands or jaws, and even the acts of mastication, swallowing, respiration, micturition, and defæcation have been interfered with by spasm occurring in the muscles concerned in those acts.

The disease usually manifests itself in childhood. It may do so in early infancy, while, on the other hand, its onset may be delayed until puberty or even later. As a rule it is a hereditary condition, and affects more than one member of the same family. Occasionally, however, isolated instances have been met with, without any indication of family affection. When it is slight in degree it may cause only slight inconvenience; when it is severe the disability occasioned may constitute a very serious drawback to an active life. Its cause is probably to be sought for in some inherited peculiarity in the muscular condition. The muscular fibres are found to be larger than usual and not so distinctly striated.

The response of the muscles to an interrupted current is peculiar, the muscles when stimulated remaining unduly long in a condition of contraction—the stimulus, as it were, setting up a condition of spasm. A special name has been applied to this—the myotonic reaction. A galvanic current applied to the muscles is said to set up a series of

wave-like contractions, passing from the cathode to the anode.

No treatment seems to be efficacious in overcoming the tendency to spasm, but Thomsen is of opinion that a life of activity tends to ameliorate the condition.

JAMES TAYLOR.

THORACIC ANEURYSM.—Under this head are included aneurysms of (A) the Intra-Thoracic Aorta; and (B) the Arteria Innominata. Aneurysms of the Pulmonary Artery are discussed on pp. 262 and 1357, and Aneurysms of the Heart and Cardiac Valves on pp. 632 and 663.

A. Aneurysm of the Intra-Thoracic Aorta.—This may be most conveniently discussed in its clinical aspects under two heads, namely (1) *aneurysm of the arch*, and (2) *aneurysm of the descending thoracic aorta*; while the former may be subdivided into aneurysm of (a) the *ascending*, (b) the *transverse*, and (c) the *descending* portion.

Relative frequency.—Of 76 cases analysed by Hayden, the seat of aneurysm, single or multiple, stated in the order of relative frequency, was as follows: *Single*: ascending portion of arch, 30; transverse portion, 17; descending thoracic aorta, 10; ascending and transverse portions of arch, 9; transverse and descending portions, 2; entire arch, 2; descending portion, 1; thoraco-abdominal aorta, 1. *Multiple*: ascending portion of arch and descending thoracic aorta, 2; ascending portion of arch and abdominal aorta, 2.

1. Aneurysm of the Arch.—The different parts of the arch of the aorta must be considered separately.

(a) *Ascending portion.*—**ANATOMICAL CHARACTERS.**—Aneurysms arising from one of the sinuses of Valsalva rarely attain a size larger than that of a billiard-ball. They are sacular and not infrequently pedunculated, communicating with the aorta by a small orifice. They further exhibit a remarkable tendency to *descend* in the progress of growth, involving in their course the heart or the root of the pulmonary artery. By their position they are sheltered from direct influx from the ventricle, while they are exposed to the maximum force of reflux from the aorta. When, however, the orifice is partially or entirely above the level of the valves, the main pressure sustained by the sac is that of efflux from the ventricle; hence the direction of growth is upwards. Aneurysm of the portion of the vessel immediately above the level of the valves is especially prone to advance towards the right side, forming a tumour visibly projecting, or detectable by palpation and percussion, in the vicinity of the right nipple. It may be fusiform or sacular, true or false; it usually attains a large size; and, when fusiform, not infrequently extends over a great portion or even the whole of the arch. The direction of growth may, however, be backwards or to either side; the aneurysm in its progress implicating the œsophagus, the pulmonary artery or one of its branches, the superior vena cava, or either auricle; it is in such cases usually sacular, and of comparatively small size. Aneurysms of the extrapericardial portion of the ascending aorta usually tend forwards and upwards in the line of main blood-pressure, projecting at the right margin of the sternum above the fourth costal cartilage, and occasionally likewise into the root of the neck, involving the arteria innominata. They may, however, grow backwards and to the right, implicating

the right bronchus or lung, or the superior cava; directly backwards, pressing upon the œsophagus or the bifurcation of the trachea; or, projecting mainly towards the left side, they may involve the left branch of the pulmonary artery, and the left bronchus or lung.

SYMPTOMS AND SIGNS.—Aneurysm of the sinuses is rarely attended by very definite symptoms; indeed, only when it presents at the anterior wall of the chest.

Owing to its position within the pericardium, and its close proximity to the heart, the symptoms produced by aneurysm in this situation may be readily confounded with structural or valvular disease of the heart itself. The acoustic signs are, for the purpose of diagnosis, no less indefinite; because, from the position of the aneurysm close to the orifice of the aorta, a murmur produced by it, whether of influx or of efflux, may be easily mistaken for one of the same rhythm caused by obstruction at the aortic orifice or inadequacy of the valves. The difficulty of diagnosis is further increased by the usual co-existence of atheroma with dilatation of the first portion of the aorta, relative incompetency of the valves, and eccentric hypertrophy of the left ventricle. The ordinary symptoms are those of palpitation and derangement of the rhythm of the heart, from affection of the cardiac plexus. But the diseases just mentioned may, in the absence of aneurysm, give rise to similar phenomena. The existence of venous stasis and congestion of the upper half of the body, viewed in conjunction with tumultuous and irregular action of the heart, and in the absence of discoverable cause of venous obstruction at a higher point in the chest, would, however, warrant the *presumptive* diagnosis of aneurysm at the root of the aorta, implicating the right auricle or the termination of the superior cava; and if with these symptoms were associated systolic murmur at the base, not transmitted in the course of the aorta, or a double murmur, a positive diagnosis to the above effect might be made. Were the diastolic murmur preceded by a distinct second sound, valvular inadequacy from dilatation of the aorta, without valvular disease, would be thereby indicated, and the diagnosis of aneurysm *pro tanto* sustained. Symptoms of obstruction of both cavæ, namely, general venous congestion and engorgement of the liver, would in the foregoing connection justify the special diagnosis of pressure upon the sinus of the right auricle. The symptoms of pressure upon the other chambers of the heart are those only of deranged rhythm and circulation, such as may be due to various causes inherent in the heart. Systolic murmur in the pulmonary artery may result from the pressure of an aneurysm on the root of that vessel. Communication of an aneurysm with one of the chambers of the heart is usually effected by an aperture not more than two to three lines in diameter. It is the result of progressive absorption, and the symptoms are scarcely to be distinguished from those of antecedent pressure. The physical signs are more characteristic; they consist in a loud murmur, systolic or diastolic, of a 'booming' or 'splashing' character, accompanied by thrill, traceable from the root of the aorta in the direction of abnormal influx, and not transmitted in any of the ordinary lines of valvular murmur. If two murmurs exist, they are fused or converted into a continuous rumble. Sudden transfer of the seat of greatest

intensity of such a murmur, from the aortic area to some other point of the præcordia, would be conclusive, not only as to the irruption of an aneurysm into one of the chambers of the heart, but likewise as to the date of its occurrence. The sac of an aneurysm in the situation of the aortic sinuses does not attain any considerable size, as it usually bursts into the pericardium, thus causing sudden death.

Aneurysm of the ascending aorta, external to the pericardium, is occasionally latent, but ordinarily it is characterised by very definite physical signs. A large fusiform aneurysm of this portion of the vessel, or one engaging the entire arch, equally expanded, not in contact with the anterior thoracic wall or pressing inconveniently upon any of the adjacent organs, may be virtually latent; exhibiting no symptoms of aneurysm except vague neuralgic pains darting over the chest, shoulders, arms, and back, and no sign but exaggerated double sound. Pointing externally, or in persistent contact with the chest-wall, an aneurysm may be readily identified by the circumstance that it presents a second centre of pulsation and intensified sounds. The ordinary pulsation is systolic, expansile, and diffused (though not always equally) over the entire surface; but a second and minor impulse of diastolic rhythm may likewise exist (*diastolic shock*). The former is in many cases accompanied by tactile thrill. The acoustic signs consist either in two sharply accentuated sounds, nearly alike in character, and corresponding in rhythm to those of the heart; or in a single or double murmur of blowing or 'booming' quality. There is likewise absolute dulness, with suppression of respiratory sounds and of vocal fremitus, to the extent of the tumour.

Pressure upon the superior cava is characterised by venous congestion, limited to the upper half of the body: while actual communication with that vessel is evinced by cyanosis to the same extent; extreme engorgement with pulsation of the jugular veins; a buzzing systolic murmur; and intense thrill, at the seat of communication and transmitted into the veins of the neck. According to Mahomed, in cases of arterio-venous aneurysm *inspiration* alters the characters of a sphygmogram, by diminishing the volume of blood in the artery. Pressure upon the main bronchus is indicated by diminished or suppressed respiration, with normal percussion-sound, in the corresponding lung; and occasionally by 'whiffing' or 'jerking' inspiratory sound. Diminished respiration throughout either lung, with inequality as between its upper and lower portions, would indicate pressure, but unequal in degree, upon the primary bronchus and its superior secondary branch; while partial or complete suppression confined to the upper lobe would show that the superior lobular branch was alone implicated. Pneumonia from occlusion of the pulmonary vessels, or from pressure upon the pneumogastric or pulmonary plexuses, is a frequent result of the presence of an aneurysm. The consolidation of lung-substance so produced is due to implication of the laryngeal and pharyngeal nerves. Bronchitis may likewise arise from mechanical irritation; and where present may, in greater or less degree, mask the physical signs of aneurysm. The sudden irruption of an aneurysm into one of the bronchi is indicated by copious discharge of florid blood from the mouth and nostrils, and is instantly fatal by syncope or asphyxia. An opening established into the pulmo-

nary substance is followed by 'leakage' of blood, or repeated but limited hæmoptysis. Pressure upon the pulmonary artery is necessarily attended by engorgement of the right chambers of the heart, and general venous congestion; and the establishment of an opening into that vessel, by sudden and urgent dyspnoea without spasm or stridor, extreme congestion of the lung, and hæmoptysis. Death is rapid in such cases; but should an opportunity for physical exploration be afforded, a 'buzzing' systolic hum might be detected in the second and third left intercostal spaces, close to the sternum. Pressure upon the œsophagus is less common than in the case of aneurysms arising from other parts of the aorta. It is indicated by dysphagia, referred by the patient to a corresponding point of the chest. Dysphagia due to the pressure of an aneurysm is remittent, and varies in some degree with posture—traits by which it is distinguished from that produced by cancer. Dysphagia from volvulus of the œsophagus may, however, exhibit similar variations.

'Clubbing' of the finger-ends has been observed in aneurysm of the aorta where there has been implication of the subclavian artery and its surroundings. The same effect has been noted in cases of aneurysm of the axillary artery. See p. 548.

(b) *Transverse portion.*—ANATOMICAL CHARACTERS.—Aneurysms of this portion of the aorta are usually fusiform: they involve mainly its anterior and superior wall, pushing forward the upper end of the sternum, projecting into the neck, compressing the left innominate vein, and modifying, in many cases, the circulation in the primary arteries and their branches. They likewise frequently press backwards upon the œsophagus and trachea, the pneumogastric or sympathetic of either side, or the left recurrent nerve. Owing to the backward course of the left extremity of the arch, aneurysms arising from this portion of the vessel rarely appear in front. They project above the left clavicle, involving the innominate vein, the pneumogastric, sympathetic, or recurrent nerve of the left side, and occasionally all three; or posteriorly in the left scapular region.

SYMPTOMS AND SIGNS.—Pressure upon the left innominate vein is accompanied by visible engorgement of the thyroid, left jugular, subclavian, brachial, and superficial thoracic veins and their tributaries, with œdema of the left arm. The circulation in the carotid or subclavian artery of one side is often diminished or suppressed by the lateral pressure of an aneurysm, or by clot-formation in the sac. Pressure upon the trachea is indicated by clanging or metallic cough, and stridor 'from below,' that is, loudest at the upper part of the sternum, and distinctly audible over the lower cervical and upper dorsal vertebrae. The symptoms of aneurysmal pressure upon the sympathetic, pneumogastric, and recurrent nerves are most frequently exhibited on the left side only. Those due to implication of the sympathetic or its cilio-spinal roots are manifested in the pupil of the affected side. They consist in dilatation or contraction of the pupil according to the degree of pressure; the former from irritation, and the latter, which is the more usual phenomenon, from paresis of the nerve. Laryngeal stridor, huskiness or loss of voice, and harsh metallic cough, in the absence of local disease of the larynx, are eminently diagnostic of pressure upon either recurrent nerve. All these symptoms are paroxysmal in character, a fact recognised as regards aneurysmal

disease since the time of Morgagni. By means of the laryngoscope the vocal cord on the side of disease, and in rare cases the cords on both sides, are seen to be fixed during breathing and vocalisation, from unilateral or bilateral paralysis of the abductor muscles of the larynx. In bilateral paralysis the cough, as was pointed out by Wyllie, assumes the character of a loud long grunt or wheeze, like the cough of a cow, hence the name given to it of *bovine cough*. The cough in such cases is deficient in the explosive element, which indicates the first act of a normal cough—the forcible closure of the vocal cords. The cow has no false vocal cords or ventricles of Morgagni, and its cough represents a long loud grunt or wheeze without the initiatory explosion. The condition of the glottis in man when both adductors are paralysed resembles the condition of the larynx in the cow; hence the peculiarity of the cough described becomes a valuable sign of aneurysm. George Johnson held that unilateral paralysis is distinguished by slight huskiness of voice, with stridor on full inspiration; and bilateral paralysis, by permanent dyspnoea and stridor. Paroxysmal dyspnoea or fatal asphyxia may result from collapse of the arytenoid cartilages in such cases. Pressure upon either pneumogastric is especially characterised by paroxysms of remittent spasm of the glottis, which may be suddenly fatal; but, where the recurrent nerve is not likewise implicated, persistent stridulous breathing, aphonia, and metallic cough are not exhibited. Wherever the laryngeal mucous membrane is found to be normal in appearance, symptoms simulating those of laryngeal disease are due to intra-thoracic pressure, the tumour pressing upon one or other of the laryngeal nerves, usually the left. The great majority of such tumours are found to be aneurysmal in their nature. A marked difference in the volume of the pulse at the wrists is frequently noted in aneurysm involving the innominate or subclavian arteries.

The physical signs are identical with those already described in connection with aneurysm of the ascending portion of the vessel. *Tracheal tugging* is met with in deep-seated aneurysms. The following directions in reference to it are given by Oliver: 'Place the patient in the erect position and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurysm exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand.'

(c) *Descending portion.*—ANATOMICAL CHARACTERS.—Aneurysm of the left curvature and descending portion of the arch involves the left recurrent nerve in nearly every instance. In the progress of growth it passes into the root of the neck; backwards towards the left scapula; or backwards and outwards into the substance of the lung.

SYMPTOMS AND SIGNS.—These include symptoms of pressure upon the recurrent or pneumogastric nerve, as well as upon the subclavian or internal jugular vein; a pulsating tumour in the left interscapular space, which may attain very large proportions; and signs of congestion and consolidation of the upper and back part of the left lung. In the last case the aneurysm, being involved in the pulmonary structure, may afford no specific evidence of its existence.

The physical signs differ in no respect from those which characterise aneurysm of the other portions of the arch.

2. *Descending Thoracic Aorta.*—ANATOMICAL CHARACTERS.—Aneurysms of the upper portion of this division of the vessel rarely attain a large size. They may pass upwards and to the right side, implicating and perforating the trachea or oesophagus; or directly to the right, stretching the oesophagus or thoracic duct, and ultimately opening into one of them or into the right pleura. The aneurysm may advance to the left, and ultimately prove fatal by rupture into the left pleural cavity; it may erode the vertebræ and ribs, opening into the spinal canal, causing paraplegia; it may point in the left infrascapular region; or it may advance towards the anterior wall of the chest, displacing the heart, and involving itself in the substance of the left lung. Aneurysm of the lower part of the vessel usually extends into the abdomen, constituting the thoraco-abdominal form of the disease. It may displace the heart forwards, and the liver downwards; it may likewise extend backwards, eroding the vertebræ, and pointing in the lower dorsal or the lumbar region on the left side. Finally, an aneurysm in this situation may prove fatal by simultaneous hæmorrhage into the left pleural cavity and left retro-peritoneal space, or into the vertebral canal.

SYMPTOMS AND SIGNS.—To what has been already stated on this subject it is only necessary to add, that progressive absorption of the vertebræ is indicated by fixed and boring pain referred to a particular point of the vertebral column, which is tender to pressure, or not infrequently radiating or 'nipping' pains extend round the chest from implication of the intercostal nerves. Forward displacement of the heart by an aneurysm would be characterised by violent impulse, simulating that of cardiac hypertrophy (the distinction would rest upon the presence of the special symptoms and signs of aneurysm); a remarkable derangement of cardiac impulse, constituting the 'double jog' of Hope; with absence of the positive signs of hypertrophy. Pressure upon the thoracic duct is very rare; it would be indicated by the symptoms of mal-assimilation, wasting, and inanition—symptoms which are foreign to aneurysm under its ordinary forms.

The physical signs of aneurysm of the descending thoracic aorta are ordinarily limited to a sharp sound, single or double, audible over the dorsal vertebræ and somewhat to the left; and, more rarely, perceptible impulse. The existence of murmur is exceptional; when present, murmur is all but invariably single and post-systolic, and is inaudible in the erect posture.

The application of the X Rays has been utilised in the diagnosis of internal aneurysms. See RÖNTGEN RAYS.

DURATION AND TERMINATIONS.—The duration of aneurysm of the intra-thoracic aorta may vary from a few days to several years. Death is most frequently caused by rupture of the sac into various parts; or by gradual exhaustion from insomnia and inanition. Of 71 cases of aneurysm in this situation tabulated by Hayden, 26 were fatal by rupture of the sac; namely, into the pericardium 10—all being aneurysms of the ascending aorta; into the left lung or pleura 5—4 being of the transverse, and one of the descending thoracic aorta; into the trachea 4—

3 of the transverse, and one of the ascending aorta ; into the right lung or pleura 3—2 of the ascending, and one of the descending thoracic aorta ; into the left bronchus or œsophagus 3—2 of the descending thoracic, and one of the transverse aorta (in one of these an opening existed both into the left bronchus and the œsophagus) ; externally one—the aneurysm having arisen from the transverse aorta. Death may also result from asphyxia, from intercurrent inflammation of the lungs or pleura, or from coma.

B. Aneurysm of the Arteria Innominata. Aneurysm involving the innominate artery may be mistaken for aneurysm of the aorta, at or near the first curve of the arch.

SYMPTOMS AND SIGNS.—Those which are most distinctive of innominate aneurysm are the early appearance of pulsating tumour above the right clavicle, accompanied by arterial obstruction on the right side ; displacement of the trachea and larynx to the left ; and pulsation with accentuated sound, localised at the right sternoclavicular joint and immediately above it. Diminished circulation in the right carotid and subclavian arteries at an early period of the disease, and the reduction or arrest of pulsation in the sac by digital pressure upon these vessels, afford the most constant and least equivocal evidence of innominate aneurysm. The early occurrence of neuralgic pains in the right side of the neck, the right shoulder, and ear, followed by œdema and partial paralysis of the right arm, is likewise suggestive of innominate, as distinguished from aortic aneurysm. The symptoms of nerve-pressure on the right side, as exhibited in the larynx and pupil, are usually well-pronounced in this disease. The physical signs are in no respect different from those of aneurysm of the arch. Concurrent implication of the aorta is ordinarily determined with the greatest difficulty, and occasionally a positive diagnosis in this respect cannot be made. If pulsation and sound of maximal intensity exist at the level of the second costal cartilage, or an inch and a half below the sternal end of the clavicle, while the pulse-tracing of the right radial artery exhibits imperfect aneurysmal characters, the aorta may be considered as involved in the disease.

DIAGNOSIS.—Aneurysm of the ascending portion of the arch of the aorta is in most cases easily recognisable, owing to the formation of a distinct tumour upon the chest-wall, in the region of the second and third right costal cartilages, which is dull upon percussion—the dullness merging into the cardiac area—and presents distinct pulsation synchronous with the heart-beat. A pulsating sarcoma in this region—a very rare affection—may give rise to difficulty, but the impulse lacks the heaving character generally found with aneurysms, and the boundaries of the tumour rarely correspond so distinctly with the line of the vessel. The presence of a diastolic shock, or on the other hand of an aortic regurgitant murmur, would indicate a vascular lesion.

Aneurysm of the transverse portion of the aorta presents very much greater difficulties, since the presence of a pulsating tumour is here the exception, and diagnosis must be made upon the existence of symptoms of pressure within the thorax. Mediastinal tumours may give rise to very similar effects. The principal points of difference are that in aneurysms the arterial and nervous structures are more frequently affected, so that irregularity of the radial pulses on the two sides of the body and laryngeal paralysis are more likely to be present :

irritation or paralysis of the sympathetic, as shown by affection of the pupil, is also more common with aneurysms, as is the existence of dysphagia, which last is uncommon with new-growths other than those originating in the œsophagus itself. The presence of well-marked tracheal tugging is commoner in aneurysm. The age and past occupation of the patient are of some assistance in arriving at a probable conclusion, aneurysm being much more common in males than in females, and originating generally in middle life and in those who have followed laborious occupations. Soldiers are specially frequent subjects of this condition. A history of syphilis is a point in favour of aneurysm (see *MEDIASTINUM, Diseases of*, p. 969).

Aneurysm of the descending part of the aorta is a rare affection, and the difficulty of diagnosis may at times be insuperable. The existence of a bruit, heard best at the back of the chest, along with pain radiating round the body or referred to the abdomen, may at times betray the presence of the condition : in other cases fatal rupture into the pleural cavity or other part may be the first and last clear manifestation of the disease. Aneurysm of the descending aorta which has eroded the vertebræ may simulate spinal caries so closely as to render distinction impossible ; and *vice versa* an abscess due to spinal caries and pressing backwards may form a pulsating tumour and be mistaken for aneurysm. The resemblance of a large aneurysm of the descending aorta to pulsating empyema must not be overlooked. The use of the X-rays gives prospect of being of the greatest use in the diagnosis of all intrathoracic aneurysms, since the shadow of a pulsating mass may be distinctly visible upon the fluorescent screen.

The signs present in cases of innominate aneurysm have already been mentioned.

PROGNOSIS.—In all cases of aneurysm within the thorax the outlook is most grave. The probable duration of life depends greatly upon the ability of the patient to lead a quiet life, and upon his willingness to undergo protracted treatment. Even in favourable cases in which apparent cure has been effected, there is often a tendency to fresh aneurysmal dilatation of neighbouring parts of the diseased vessel. When there are signs of implication of the skin over an aneurysmal tumour on the chest-wall, the patient's condition becomes alarming. Actual leakage of blood may occur and threaten a fatal issue by external hemorrhage ; but even in these circumstances the fatal rupture of the sac is usually internal, and in rare instances death may not take place for several weeks after hemorrhage first occurs. The occurrence of asphyxial attacks is a serious symptom, and points to a speedily fatal issue, but in individual attacks the patient may rally from an apparently moribund condition under energetic treatment (venesection, &c.). Hemorrhage occurring into the larger air-passages is generally the precursor of death within at most one or two days, but the presence of streaks of blood in the sputum is not infrequent in earlier stages, owing to compression of the lung and the strain of coughing.

TREATMENT.—The treatment of aneurysm will be found described in the article *ABDOMINAL ANEURYSM*. The *curative* treatment of aortic or innominate aneurysm should be directed to the single object of effecting consolidation of the contents of the sac. With this object in view, three methods have been pursued, either separately or conjointly, namely, the *postural* and *dietetic* ; the

medicinal; and the *operative*. As an assistance to the first plan of treatment of thoracic aneurysm, an occasional blood-letting by venesection, to the amount of eight to ten ounces, for the purpose of reducing arterial tension or venous engorgement, may be demanded. With a view to causing or promoting deposition of fibrin in the sac, several agents have been used, namely, acetate of lead, in doses of four to eight grains; iodide of potassium, ten to thirty grains; and aconite, five minims of the tincture thrice daily. Of these drugs by far the most satisfactory results have been obtained by the use of iodide of potassium given in doses ranging from ten to thirty grains three times a day, the administration of the drug being continued, having regard to the tolerance of the patient, for months. The explanation of the therapeutic value of iodide of potassium according to Balfour is to be found in the fact that under its use the lowering of the cardiac and vascular tension arrests or greatly modifies the dilatation of the sac at each stroke of the heart, and thus brings into operation again the natural elasticity of the arterial coats which contract the sac temporarily until ultimately this contraction is strengthened and made permanent by the hypertrophy of these coats—mainly of the *adventitia*. Ergotin has been used hypodermically by Langenbeck. The latest method of the treatment of aneurysm by medicinal means is that advocated by Lancereaux and Paulesco. It consists in the injection into the muscles of the buttock of 2-per-cent. sterilised solution of white gelatine (p. 707). Three ounces of the fluid are injected by means of a special apparatus devised for the purpose, the injection being made in rather less than fifteen minutes. The injection is repeated every six or eight days until consolidation of the sac has taken place. The results of the treatment are still *sub judice*. In some of the cases treated death followed symptoms resembling those of tetanus, the cause of which, having regard to the antiseptic precautions taken, is obscure. Each of the agents mentioned has been credited with success in the treatment of aneurysm, but, as spontaneous cure has been occasionally witnessed under favourable circumstances as to diet and rest, where no medicines had been given, a more than promotive influence, by retarding the circulation and reducing vascular pressure, can scarcely be assigned to the medicine used, where rest and restricted diet have been observed. Galvano-puncture of the sac has been practised with success. Macewen advocates the production of white thrombi in organic connection with the wall of the aneurysm by the introduction of sterilised needles, by which the inner surface of the sac is gently scratched. Deligation of the common carotid artery, or of that vessel and the subclavian, may be followed by the most favourable result, in cases where pressure upon these vessels has been found to control pulsation in the sac.

A few leeches applied from time to time in the vicinity of the sac, or a hypodermic injection of morphine or hydrochloride of cocaine, will relieve the pain and allay the inflammation caused by ex-centric pressure.

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THORACIC DUCT, Diseases of.—**SYNON.** : *Fr. Maladies du Canal Thoracique*; *Ger. Krankheiten des Ductus Thoracicus*.

The diseases of the thoracic duct resemble in kind those of the absorbent vessels generally (*see*

LYMPHATIC VESSELS, Diseases of); and it will suffice to indicate here the following practical points : (1) The passage of fluid along the duct and its escape into the subclavian vein may be impeded by any condition which interferes seriously with the venous circulation, and distends the veins considerably, such as certain cardiac diseases. (2) Local obstruction of the thoracic duct may arise at any point, from direct pressure upon it, especially by an aortic aneurysm, and it may become thus permanently occluded; or from intrinsic tubercular disease, which is of special importance, inasmuch as it may involve the duct extensively, break down, and lead to acute general tuberculosis by infection. (3) As a consequence, dilatation of the portion of the tube behind the seat of obstruction will probably supervene in various degrees, and it may become considerably enlarged and thickened. The portion beyond tends to become contracted and atrophied. (4) Perforation of the thoracic duct occurs in exceptional instances, owing to the destructive effects of an aneurysm or other morbid condition, or as the result of injury.

It is, as a rule, quite impossible to determine during life that the thoracic duct is diseased. This might be suspected if, along with some known cause which might lead to obstruction of the tube, the patient became extremely emaciated, anæmic, and weak, without other obvious reasons to account for these symptoms. No treatment directed immediately to the thoracic duct can be practicable under any circumstances.

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THORACIC TUMOUR.—A tumour within the chest. *See* **BRONCHIAL GLANDS, Diseases of**; **MEDIASTINUM, Diseases of**; and **THORACIC ANEURYSM**.

THORACOCENTESIS (θώραξ, the chest; and κεντέω, I prick).—A synonym for paracentesis thoracis, or tapping of the chest. *See* **PARACENTESIS**; **PLEURA, Diseases of**; and **PLEURA, Surgery of**.

THORAX.—*See* **CHEST**.

THREAD-WORM.—*See* **ENTOZOA**.

THROAT, Diseases of.—**SYNON.** : *Fr. Maladies de la Gorge*; *Ger. Rachenkrankheiten*.

The throat is a comprehensive term, its diseases including those of the pharynx, tonsils, palate, and uvula, and in popular language even those of the larynx and trachea. The affections of these different structures are described in their appropriate articles.

THROMBOSIS (θρόμβος, a clot).—**SYNON.** : *Fr. Thrombose*; *Ger. Thrombose*.

DEFINITION.—The formation of a solid clot or 'thrombus' in the heart or vessels during life. The earliest stage in the formation of at least some thrombi has been shown to consist in the agglutination of blood-platelets, or sometimes of leucocytes. Hence the term 'thrombosis' has a slightly wider significance than the formation of a fibrous coagulum, though for practical purposes the deposition of fibrin is its most essential feature. Clotting may occur anywhere in the cardio-vascular system, and rarely even in lymphatics. In many cases it serves a useful end. The coagulability of

the blood is a property of vast importance in the arrest of hæmorrhage: the deposit of clot upon the walls of an aneurysm tends to check its extension and may promote its cure. But more often thrombosis is harmful in its effects.

PATHOLOGY.—The following may be accepted as a working hypothesis as to the nature of blood-coagulation. Fibrin is derived from a globulin called 'fibrinogen,' present in the plasma. The immediate agent of its conversion is the 'fibrin-ferment' (thrombin), and this appears to be a combination of a nucleo-proteid substance with lime. The nucleo-proteid is derived from the breaking up of leucocytes and blood-platelets; the platelets themselves are probably derived from the disintegration of red corpuscles. Thus all the elements of the blood may have a share in the process. The one factor lacking to coagulation in normal circulating blood is the special nucleo-proteid which goes to form the fibrin-ferment.

This factor can be experimentally added in animals, thereby causing intra-vascular clotting. Wooldridge showed that emulsions or extracts of lymphatic gland or thymus rapidly injected in sufficient quantity into the vessels produce this effect. Preformed fibrin-ferment gives rise to a similar result, as in Köhler's experiment, in which serum, expressed from the blood-clot of a rabbit, and re-injected into the veins, causes death in a few minutes from intravascular coagulation. The injection of such chemical bodies as break up the corpuscles and liberate the requisite nucleo-proteid may produce the same effects: bile-salts, ether, and large doses of snake-venom have been found to do so. These experiments give rise to thrombosis in the presence of a perfectly intact vascular endothelium: there can be no doubt that the phenomena depend simply on the presence of free fibrin-ferment in the circulation. The clinical occurrence of such forms of thrombosis in man is very doubtful, though some writers have advocated the view that thrombi occurring in cachexias and in certain infections have this origin. The intra-vascular clotting that may follow transfusion of defibrinated blood is, however, evidently parallel to that above described. The presence of lesions of the vascular or cardiac endothelium is a factor of much importance in the production of thrombosis. An intact endothelium is commonly spoken of as exercising a restraining influence upon coagulation (Brücke); it would be a more natural way of putting the matter to say that the roughened surface caused by endothelial lesions, or presented by foreign bodies, affords opportunities for the adhesion and disintegration of the blood-corpuscles and the consequent liberation of fibrin-ferment. It is even possible that the ferment may be liberated from necrotic endothelial cells.

Since the discovery of the part played by the blood-platelets in coagulation, endeavours have been made to trace a connection between the absolute number of platelets present in the blood and the tendency to thrombosis. Although the method of estimating their number leaves something to be desired, some sort of connection seems traceable. In chlorosis the platelets are much increased in number and thrombosis is common; in pernicious anæmia their number is reduced, and thrombosis is extremely rare. In hæmorrhagic purpura there is extreme diminution in the platelets, and the condition is associated with marked indisposition to

coagulation. In post-hæmorrhagic anæmia the disposition to coagulation is increased, and here the platelets are increased too. This apparent relation between increase of platelets and tendency to thrombosis may, however, be an indirect rather than a direct one; both phenomena may depend upon an increased vulnerability of the red corpuscles.

There is yet another factor which plays an important part in the production of thrombosis, namely, retardation of the blood-stream—local or general. Local slowing may depend upon the production of eddies by obstruction, narrowing or dilatation of the blood-channels. General slowing depends upon feebleness of the heart's action in debility and cachexia arising from any cause. The efficacy of this condition in assisting thrombosis depends in part on the fact that fibrin is more readily deposited from stagnating blood, in part on the fact that slowing of the circulation interferes with the nutrition of the endothelium lining the vessels, and so promotes degenerative changes in the intima. It may be also that in conditions of marasmus chemical changes are present in the blood which predispose to coagulation, or that the vulnerability of the corpuscles is increased so that they more readily yield up the nucleo-proteid necessary for the formation of the fibrin-ferment. Also, with slowing of the stream, comes a broadening of the axial current of corpuscles and platelets, and a greater disposition of these to come into contact with the vascular walls. The thrombosis occurring in conditions of debility and marasmus is commonly spoken of as '*marantic*' thrombosis.

ÆTIOLOGY.—The general conditions leading to coagulation of the blood during life have been indicated above. It remains to enumerate the chief clinical conditions under which thrombosis is apt to occur.

1. Traumatic lesions in continuity of the vessel-wall.—Thrombosis is the natural method by which such lesions are remedied. When a surgeon ligatures a vessel in its continuity, he deliberately aims at the rupture of the inner coats in order that a thrombus may form which shall, when organised, permanently seal the artery.

2. Foreign bodies occasionally lodge in the blood-vessels and cause clotting of the blood around them.

3. Whenever a vessel is obstructed, even partially, by an embolus or a thrombus, such masses act as foreign bodies and induce further deposit of clot. In this way an embolus may form the nucleus for an extensive thrombosis, and by such means a thrombus is itself 'produced' or 'protracted.' In these cases the effect of stagnation or retardation of the current is added to that of the abnormal surface.

4. Any chronic lesion, inflammatory or degenerative, of the heart or vessel-wall, is apt to set up a local thrombosis in virtue of the surface-roughening which it implies. Thus it comes to pass that chronic endarteritis and atheroma are fertile causes of thrombosis, especially in the smaller vessels where the current is less rapid. Thrombosis of the cerebral arteries is usually secondary to such chronic changes in the intima, as, for example, in syphilis; and this is the way in which syphilis, tuberculosis, and gout act in producing thrombosis. The usual absence of thrombosis in the aorta and great vessels, even when highly atheromatous, is probably due to the great velocity of the blood-

stream in such situations, which must tend to prevent the adhesion of corpuscular elements, and to sweep away any commencing thrombus. When, however, any dilatation or *cul-de-sac* is present in addition, as in aneurysm, the conditions for the deposit of clot are very favourable. In endocarditis the diseased surface of the endocardium is commonly the seat of some thrombotic deposit; vegetations are usually capped by some recent fibrin, and in this way, by gradual organisation of the deposit, they increase in size.

5. Acute inflammatory changes in the walls of the heart and vessels may equally be attended by thrombosis, whether the affection be primary or due to extension from adjacent foci of inflammation. Phlebitis is thus usually accompanied by thrombosis, nor is it always easy to say which is the primary condition. In the majority of such acute inflammatory changes the process is septic in its nature, and thus bacterial invasion comes to be one of the most serious causes of thrombosis. Infective phlebitis is unfortunately a common affection. It is seen in the lateral sinus as a complication of middle-ear disease; and, starting from the uterine veins, it is one of the recognised dangers of childbirth. In fact, in all septic conditions it constitutes a serious risk. Bacterial invasion of the arteries is relatively rare, but in the heart it is by no means uncommon, and septic, malignant, or ulcerative endocarditis is usually associated with pronounced thrombotic deposit, with its attendant liability to embolism. It is possible that many of the cases of thrombosis occurring in the course of infective processes, such as the specific fevers, tuberculosis, &c., are in reality of bacterial origin.

6. Marasmus and debility of all kinds are liable to lead to thrombosis, for the reasons which have been already given. It is probable that slowing of the circulation cannot alone cause clotting, since blood will long remain fluid even when absolute stasis has been induced by the careful double ligaturing of a vein, provided always that the endothelium has not been damaged. Some additional cause is to be sought in most cases of marantic thrombosis, and it is likely that an infective element is often present; where this is not the case, malnutrition of the vessel-wall, with possible chemical changes in the blood, must be invoked as contributing causes. The clinical conditions under which stagnation-thrombi are formed comprise anæmias, especially chlorosis, and cachexias of all kinds, such as occur in phthisis, or after protracted fevers, e.g. typhoid. Thrombosis of the cerebral sinuses is occasionally seen in infancy as a result of extreme marasmus.

Structure.—There is experimental evidence that some thrombi consist, at least at their earliest commencement, not of coagulated fibrin, but of a dense agglutination of preformed morphological elements. Eberth and Schimmelbusch witnessed the agglutination of blood-platelets in the mesenteric vessels of warm-blooded animals. Others have demonstrated the occurrence of thrombi consisting of leucocytes alone, while Wlassow maintains the first origin of the white thrombus to lie in the sudden destruction and coagulation of the red corpuscles. Purely fibrinous thrombi are also described, especially in the capillaries in inflamed areas, e.g. in croupous pneumonia. They are often hyaline in appearance, and are then known as hyaline thrombi.

But in ordinary thrombosis the mass consists of a mixture of different elements. Weigert's method of staining fibrin is especially useful in demonstrating the structure of a thrombus. Whatever its primitive origin, it soon comes to consist of fibrin deposited from the blood, and entangling in its meshes leucocytes and red corpuscles in varying proportions. Speaking generally, the clot formed from blood at rest contains chiefly red corpuscles, and is known as a '*red thrombus*,' while that deposited from blood in motion contains a preponderance of leucocytes, and is called a '*white thrombus*.' The clot formed in a popliteal aneurysm after ligature of the femoral artery is a red thrombus; that deposited on the walls of an aortic aneurysm is a laminated white thrombus. A newly formed red thrombus is soft and not laminated; it shows a loose fibrin-network, entangling a multitude of red corpuscles, with a few leucocytes, perhaps proportionately more than in normal blood. The white thrombus is firmer and denser in its texture, and commonly presents a laminated structure. The fibrin is present in fibrillary form, or in broader and more irregular trabeculae. Leucocytes are the chief morphological elements present, though a varying proportion of red corpuscles may be seen; besides these recognisable elements there is present among the meshes of the fibrin-network much granular material believed to be derived from broken-down blood-platelets. A very common form is the '*mixed thrombus*' containing layers or areas of both the forms of clot just described. A white thrombus may become fissured, especially when laminated, and so fluid blood may penetrate and coagulate as red thrombus. Or the clot may originally form in alternate layers, according to the varying conditions under which it is deposited. A *primitive* thrombus is one occupying its original seat of deposition; when it extends along a vessel it is called a *produced* or *protracted* thrombus. A clot adhering to the vessel-wall but not completely filling the lumen is known as a *parietal* thrombus; if it absolutely obstructs the blood-channel the term *obliterating* thrombus is applied to it.

Growth and Extension.—When once thrombosis has commenced it tends to spread because the thrombus itself acts as a foreign body upon which new material is deposited from the blood in contact with it. Thus arises the propagated thrombus. In this manner the parietal may become an occluding thrombus. A clot may spread in this way along a vessel until, where it joins a larger trunk, the velocity of the current is sufficiently great to check its further growth, so that it projects by a free rounded extremity into the lumen of the obstructed channel.

Later changes.—Thrombi undergo a variety of secondary changes which are sometimes of great practical importance. They may be described under the following headings:—

1. *Hyaline transformation.*—The cellular elements break down, and such hæmoglobin as may be present is dissolved out. A granular mass thus arises which later becomes translucent and hyaline, shrinking in the process so that the lumen of the vessel may be partially reopened; nevertheless the thrombus commonly remains adherent at some points.

2. *Organisation.*—The thrombus itself is an inert mass: its organisation takes place from without, at its points of adhesion to the vessel-wall. The endothelium of the latter proliferates, and extends over

the clot; beneath the endothelial layer connective tissue is developed. Some hold that it takes its origin from the endothelial cells themselves, others that it is derived from the migration of wandering cells. It ultimately permeates the entire mass. Meanwhile the thrombus becomes vascularised; the new vessels arise in part from processes dipping down from its endothelial covering, in part by budding from the vasa vasorum of the vessel-wall; the two sets ultimately come into communication, and thus a complete circulation is established throughout the thrombus. The newly formed connective tissue undergoes contraction, and the whole mass may finally appear as a mere thickening of the vessel-wall, or as a fibrous strand crossing its lumen. The thrombotic obstruction may thus practically disappear in favourable cases; at other times a collateral circulation having been established permanent obliteration of the vascular channel may produce no harmful result.

3. *Calcification* is an occasional occurrence in a thrombus. Lime-salts may be deposited in it as in any other relatively dead tissue: thus arise *phleboliths*.

4. *Simple softening*.—In certain cases organisation does not take place, and softening and breaking down of the mass occur in its centre. This sometimes happens in venous clots, and commonly in large cardiac thrombi. It is probably due to the digestive action of ferments liberated from the disintegrating leucocytes, and seems undoubtedly to occur in truly aseptic cases.

5. *Septic softening*.—In clots which are due to bacterial action, or which have been secondarily infected, softening and liquefaction readily occur, as, for example, in lateral-sinus thrombosis secondary to middle-ear disease. To the naked eye the process may resemble simple softening; the clot breaks down into a creamy, puriform material which readily gives rise to septic embolism. Even simple softening may lead to a crumbling consistency and bland embolism.

Localisation of thrombosis.—There is no part of the cardio-vascular system in which clotting may not occur. Nevertheless, there are certain situations in which it is commoner than in others. It appears to commence more often in vessels of medium size than in the very large or the very small. When it arises from general conditions the veins are far more frequently affected than the arteries; among the reasons for this those which seem of most weight are the greater slowness of the blood-stream in veins, and the larger percentage of carbon dioxide in the blood. Where thrombosis arises from local conditions the proportions are more equal; the arteries are the more subject to chronic disease and embolism; the veins are more liable to be attacked by septic inflammation.

Arterial thrombosis is usually due either to embolism or to some form of chronic arteritis; in the latter case aneurysmal dilatation is an especially favourable condition. The lower extremities are more commonly affected than the upper, and the prevalence of chronic arteritis in the cerebral and coronary arteries renders these also common sites of thrombosis. In the veins, again, clotting occurs with especial frequency in the lower extremity; marantic thrombi are very commonly found in this situation. There are special causes which lead to a special incidence of the process on other veins; thus the slowness of the current in the cerebral

sinuses predisposes to the condition, while the proximity of the lateral sinus to the middle ear renders it liable to septic infection in otitis media. A similar septic condition after parturition is liable to spread from the uterine to the iliac veins, whence arises phlegmasia alba dolens. In the heart thrombosis is common. Endocarditis, especially in its malignant form, is frequently associated with thrombotic deposit upon the vegetations, and in addition to this, in cardiac feebleness and dilatation, it is common to find *ante-mortem* clotting in certain situations, notably in the auricular appendices and at the apices of the ventricles. As a pathological curiosity may be mentioned the free 'ball thrombi,' which in rare instances have been found in the left auricle in cases of mitral stenosis, and which probably result from the accretion of fibrin round a detached portion of clot.

True thrombosis in the heart and vessels must be carefully distinguished from *post-mortem* clotting. Coagulation of the blood after death leads to the formation of clots which are usually voluminous in proportion to the distension of the cavities at the moment of death, and are best marked in the right chambers of the heart. If the clotting has occurred quickly they are of uniform dark red colour; but if sufficient time has elapsed for the precipitation of the corpuscles they are yellow and gelatinous, except at their most dependent parts. They are soft and somewhat tenacious, never crumbling or breaking down centrally. They are never truly stratified, and their surfaces are not ragged or honeycombed. They are never truly adherent, though they may be closely entangled among the columnæ carneæ; when peeled off from the heart-wall the smooth intact endocardium is found beneath them. Attention to these points will prevent any errors in distinguishing between the two conditions. The term 'agony clot' is sometimes erroneously employed to designate these *post-mortem* clots. The fact that they are so frequently decolourised by subsidence of the corpuscles is sufficient to prove that they have been formed after death and not during the death-agony.

Capillary thrombosis is for the most part limited to areas of necrosis or of intense inflammatory change passing on to tissue-death. Hyaline capillary thrombi are described in various infective conditions. Thrombosis of the lymphatics is a condition of minor importance, but it has been described in connection with septic conditions in the uterine lymphatics. The lymph is only feebly coagulable, and lymphatic obstruction is rarely due to primary thrombosis.

Results of thrombosis.—In the left heart and in the larger systemic arteries the detachment of thrombi leads to embolism of the systemic vessels. In the veins and in the right heart such detachment leads to pulmonary embolism. But for the most part, the results and the symptoms of thrombosis are those of vascular obstruction, and so far as these concern the arterial system they have been described in the article on Embolism. Venous thrombosis leads to its own special train of symptoms (see article on VEINS, Diseases of). Anastomosis between veins is commonly more abundant than in the case of arteries, so that a collateral circulation is readily established when the obstruction is limited in extent and does not affect a main trunk. But often a main trunk, such as the femoral vein, is affected, and the degree of obstruction produced leads to marked oedema, i.e. the increase of pressure

in the capillaries behind the obstruction leads to a greater transudation of lymph than can be dealt with by the lymphatics. Even here the oedema is not due solely to the venous obstruction, for the femoral vein may be tied in man or animals without resultant dropsy. The accessory factor which probably contributes to the production of the oedema is an increased permeability of the capillary wall, which may depend partly on nutritional changes in the endothelium from impeded circulation, partly on the cachectic or other conditions which have led to the thrombosis. Thrombosis of the portal vein seems always to lead to ascites, and sometimes in this and in other veins (e.g. the renal) hæmorrhagic infarction may result. Cases have been reported in which moist gangrene of the extremities has apparently resulted from thrombosis of the main venous trunk, but this an extremely rare sequel of venous thrombosis, common as it is when the artery is the obstructed trunk. When a septic element is added to the mechanical obstruction of a vein, as is too commonly the case in uterine thrombosis after parturition, the dangers of septicæmia overshadow other considerations.

The SYMPTOMS of thrombosis are those of vascular obstruction, and naturally vary with the situation of the affected vessel and the character and degree of obstruction. In the heart and sometimes in the peripheral circulation embolism may be an important symptom. If the affected vessel be accessible to the touch, a firm and often tender cord may be felt.

The TREATMENT of thrombosis depends upon its cause and seat (see articles on HEART, Thrombosis of; PORTAL THROMBOSIS; VEINS, Diseases of, &c.). The general principles of treatment are directed (1) to the removal, where possible, of the primary cause. Anæmia, gout, and similar constitutional conditions are to be met by appropriate remedies; (2) to the obviating of the dangers and results which may arise. Perfect rest will, in venous thrombosis, limit the liability to pulmonary embolism, and assist in the development of a collateral circulation. In arterial thrombosis it will check the tendency to necrosis and gangrene.

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THRUSH.—A popular name for parasitic stomatitis. See MOUTH, Diseases of.

THYMUS GLAND, Diseases of.—SYNON.: *Fr. Maladies du Thymus*; *Ger. Krankheiten der Thymusdrüse.*

The thymus has but a short period of full activity. At birth it weighs from 100 to 200 grains, and it reaches its full development by the end of the second year. It then remains in much the same condition till about the eighth year, after which it gradually diminishes in size, being converted into fat, till at puberty only a trace remains. Extracts of the thymus when injected either beneath the skin or directly into a vein have not been found to possess any special physiological action.

Persistence and Hypertrophy.—In some adults the thymus is found to persist as large as in the infant, while in others it is even larger and may weigh as much as 300 or even 400 grains. Cases have been reported of children dying suddenly from suffocation, with no other cause assigned for their death than pressure on the trachea of an enlarged thymus gland. Considering its position between the sternum and the windpipe, and the small power

of resistance possessed by the rings of the trachea during infancy, an enlarged thymus may be an occasional cause of asphyxia. Enlargement of the thymus may also occur as part of a condition known as lymphatism, in which death from cardiac failure may take place from some trivial cause such as a hypodermic injection. An enlarged thymus is frequently found in some diseases such as exophthalmic goitre, Addison's disease, acromegaly and lymphatic leucæmia.

Inflammation.—Acute inflammation of the thymus is rare and difficult to recognise. It may be followed by suppuration and the formation of an abscess. In one such instance the abscess burst into the trachea.

Tuberculosis.—Primary tuberculosis is very rare, but in general tuberculosis the thymus may be infected just as any other organ.

Syphilis.—Small gummata are sometimes found, or there may be a general syphilitic fibrosis.

Sarcoma.—Sarcoma of the thymus occurs more frequently than carcinoma. The sarcoma may be either round-celled or spindle-celled or of a mixed type. In some so large an amount of fibrous tissue is present as to warrant the name of fibro-sarcoma.

Carcinoma.—Carcinoma of the thymus may vary in type. The cells are generally rather large, and vacuolation has been found in them by H. D. Rolleston. In some cases 'epithelial pearls' occur which show that the growth is derived from the original diverticulum of the fore-gut from which the concentric corpuscles of Hassall are developed. Some malignant growths of the thymus show such different types of structure in the same tumour as to suggest that they are really combinations of carcinoma with sarcoma.

SYMPTOMS OF THYMUS TUMOURS.—The symptoms of a tumour of the thymus-gland are those of a growth in the anterior mediastinum. The tumour may extend upwards so as to be felt projecting above the upper end of the sternum, or it may grow downwards over the anterior surface of the pericardium. The superior vena cava and the innominate veins may be compressed; then the head and neck are cyanosed, the veins of the upper part of the body are distended and the subcutaneous tissues become oedematous. In addition there may be pleural effusion.

PUGIN THORNTON.
GEORGE MURRAY.

THYROID GLAND, Diseases of.—SYNON.: *Fr. Maladies de la Glande Thyroïde*; *Ger. Krankheiten der Schilddrüse.*

The thyroid gland produces, in the form of the colloid material which fills its alveoli, an internal secretion which enters the blood and plays an important part in the normal metabolism of the tissues. Diseases of the gland are accompanied, therefore, by general or local symptoms, according as the supply of secretion is or is not influenced by the morbid change in the gland. Thus associated with fibrosis and atrophy of the gland we have myxoedema and cretinism (see CRETINISM; and MYXŒDEMA). In exophthalmic goitre there is a special form of hypertrophy of the gland, while other forms of enlargement are included under the name of goitre (see EXOPHTHALMIC GOITRE; and GOITRE). As these conditions are described separately we shall here consider the remaining less common local diseases of the gland, which do not, as a rule, appreciably affect its functional activity.

1. **Acute Thyroiditis.**—Acute thyroiditis may be either primary, secondary, or traumatic in origin. The cause of the primary form is not known. Secondary thyroiditis has occurred as a complication or sequel of acute rheumatism, influenza, diphtheria, measles, and other acute infective diseases. In some of these the inflammation has been shown to be due to the bacteria of the primary disease. Traumatic thyroiditis has occurred as the result of a wound or burn in the thyroidal region.

SYMPTOMS.—The onset is generally acute and may be accompanied by a rigor, the temperature rising rapidly to 103° F. Pain and a sensation of fullness are commonly felt in the thyroidal region. One lobe or the whole gland then becomes tense and swollen. In severe cases there may even be dyspnoea and dysphagia from compression of the trachea and oesophagus. The attack may terminate by resolution, suppuration, or gangrene.

TREATMENT.—The patient should be kept in bed and fed on a light or liquid diet. Locally the application of ice or of leeches will give relief; if these fail, belladonna-fomentations may be employed instead. At the commencement 5 or 10 grains of calomel should be given at night, followed by a saline aperient in the morning. During the acute stage 10 or 15 grains of sodium salicylate should be given every three or four hours. If an abscess forms it should be emptied at once by aspiration or by incision and drainage.

2. **Hydatid Cysts.**—These have in a few cases produced an enlargement of the thyroid gland. A hydatid cyst closely resembles a cystic adenoma, but a microscopical examination of the fluid withdrawn from the cyst may, by the discovery of hooklets, enable a correct diagnosis to be made.

3. **Malignant Disease.**—(a) *Carcinoma.*—Primary cancer of the thyroid gland is a rare disease, which develops less frequently in a healthy gland than in one which is already goitrous. It occurs most frequently between 30 and 50 years of age. It may develop from the epithelium which lines the alveoli of the gland. It has, however, been shown by Horsley that some of the carcinomatous growths probably originate in the parathyroid gland, which they closely resemble in structure.

In carcinoma there is rapid enlargement of the thyroid gland, followed in an early stage by duskeness of the skin over the most prominent part of the swelling. Such signs are all the more important if the patient has passed middle-life. The enlarged gland feels hard and irregular, and is less movable than a non-malignant goitre. There may be radiating pain over the front of the neck. In some cases there is complete paralysis of one or even both vocal cords, owing to impaction of the recurrent laryngeal nerve in the growth which may involve the large blood-vessels in the neck as well. Dyspnoea and dysphagia are both common symptoms, especially in the later stages, the trachea and oesophagus being first compressed and then invaded by the growth. In advanced cases the skin may be destroyed, when superficial ulceration occurs with hæmorrhage. The lymphatic glands are not infected until the growth extends through the capsule of the gland. The total duration of life after the recognition of the disease rarely exceeds a year.

TREATMENT.—The only treatment which is of any avail is *total* excision at the earliest possible

date. It is most important, as Kocher has shown, that this should always be done even when the growth appears to be confined to one lobe. When the disease is too far advanced for this radical treatment, tracheotomy may be performed in some cases for the relief of dyspnoea.

(b) *Sarcoma.*—Sarcoma occurs much less frequently than carcinoma. It may develop in either a healthy thyroid gland or in one which is already goitrous. It grows rapidly, and may reach the size of an orange or even a coconut. The tumour is uniform and soft in consistence, and may contain cysts. The growth soon extends through the capsule and may involve the vessel and nerves in the neck, and the trachea and oesophagus as well. It is liable to involve the skin and cause ulceration with free hæmorrhage. Secondary growths may occur in the lymphatic glands in the neck and in the internal organs. The total duration of the disease is rarely more than a year. The growth may be either a round- or spindle-celled sarcoma. The entire gland should be removed as soon as the nature of the enlargement is determined.

(c) *Recurrent Thyroid Tumours.*—In this form of malignant disease the primary growth resembles a simple adenoma in structure. Secondary growths develop in the bones of the skull, long bones and vertebrae, and also in the lungs and lymphatic glands, the microscopical structure of which resembles that of the normal thyroid gland. They are, however, very malignant, as they rapidly extend and lead to a fatal termination. *See* TUMOURS.

4. **Tuberculosis, Syphilis, and Actinomycosis** of the thyroid gland are rare, but do not essentially differ from the same diseases in other organs except when the two latter, in very rare cases, so far interfere with the functions of the gland as to lead to the development of myxœdema.

Therapeutic Action and Uses of Thyroid Gland.—The thyroid gland and its preparations are now largely used as therapeutic agents. The two official preparations, *Liquor Thyroidei* and *Thyroideum Siccum*, are in general use, six minims of the former being equivalent to one grain of the latter. They are chiefly employed in the treatment of myxœdema and cretinism, for which purpose the *Liquor Thyroidei* was originally devised and introduced by one of the writers (G. M.). The usual dose is from five to fifteen minims of the *Liquor Thyroidei* or from one to three grains of the dry thyroid each day, under the influence of which the symptoms of myxœdema and cretinism rapidly disappear (*see* CRETINISM; and MYXŒDEMA). The same preparations are also useful in the treatment of simple goitre in young adults (*see* GOITRE), and of some diseases of the skin such as psoriasis and ichthyosis.

PUGIN THORNTON.

GEORGE MURRAY.

TIC-DOULOUREUX (Fr.).—**SYNON.** : Facial Neuralgia; Prosopalgia.

DEFINITION.—Neuralgia of the trigeminal, trifacial, or fifth nerve.

One alone, more often two, but rarely all three divisions of the fifth nerve of one side may be coincidentally the seat of neuralgia. It is less common for the third division to suffer than the first and second. Tic has often been preceded or followed by neuralgia in other districts, especially in that of the occipital nerve.

ÆTIOLOGY.—Trigeminal neuralgia is rare in young children, but may occasionally occur in them, associated, perhaps, with delayed or irregular eruption of the permanent teeth. But sex and occupation do not appear to influence its occurrence. In women utero-gestation, the exhaustion from hæmorrhage at parturition, menorrhagia, or over-suckling, as well as the sexual changes in middle-life, are especially prone to reproduce facial neuralgia, which has appeared in a slight form earlier in life. Exposure to cold and hardship apparently conduce to its production. It is, however, in the period of degeneration that the worst and most intractable instances occur.

The term 'brow ague' is still frequently applied to a neuralgia of the first division of the trigeminal, which cannot always be referred to a malarial origin. Formerly, when malarial fevers were rife in this country, such an affection was, doubtless, of common occurrence; but at the present day, owing to drainage and cultivation, it is rare for this cause to be in operation. Cases do, however, occur, and these may usually be recognised by regular periodicity in the attacks of pain, a semi-algid condition of the system, and the rapid and effectual influence of quinine. But a remarkable periodicity is often to be found in the recurrence of attacks, unconnected with ague. Cold wind, especially with a moist atmosphere, has an undoubted influence in starting neuralgia of the fifth nerve, the unprotected condition of the face explaining probably its peculiar liability to be so attacked. There appears reason to think, however, that when damp with cold excites an attack of neuralgia, there must be at the same time a peculiar condition of the system, or neuralgia of the fifth would be much more common in this climate than it is. Such a condition is probably of a rheumatic or gouty nature, and the cold seems to start a subacute inflammation in the sheath of the nerve. As regards other general conditions predisposing to the affection, they are those common to neuralgia. See NEURALGIA.

Injury of the nerve; foreign bodies, irritating either this or some other nerve; morbid growths of bone, especially such as cause contraction of bony canals traversed by branches of the nerve; and syphilitic periostitis, may act as exciting causes of tic-douloureux. Or the immediate cause may be in the floor of the cranium, in the form of tumours or disease of bone or of membranes, aneurysm, or abscess.

SYMPTOMS.—Some obscure feeling of discomfort may precede the outburst of actual pain, or this may occur suddenly and without warning in some part of the district supplied by the fifth nerve. There will be one or more foci from which the pain will seem to emanate in swift flashes, a dull aching remaining in the intervals, of a very wearying character. Generally short-lived at first, the paroxysms of darting, burning, boring pain gradually increase in severity and duration. The patient sometimes cringes under the violence of the agony. When fairly pronounced, there is a great tendency to the excitement of a paroxysm by the influence of such slight irritants as a current of air, a sudden noise, or the muscular movements concerned in speaking, laughing, chewing, blowing the nose, or coughing. The attack of tic may vary to any extent in degree and duration, from a short-lived paroxysm which never returns, to a disease of the most obstinate character, embittering, with more or less constantly

repeated attacks, the whole of a long life. In such cases it has a tendency to remit during the course of severe intercurrent diseases.

When the *ophthalmic division* of the nerve is affected, it sometimes happens that the first notice of the attack is an exceeding soreness of some spot on the scalp, recognised only on brushing the hair, and this is followed some hours afterwards by pains in the branch of nerve distributed to this point. The pains are most marked, and tenderness on pressure can generally be noted, in one or more of the following places: the supra-orbital notch, at a point a little above the parietal eminence, in the upper eyelid, at the junction of the nasal bone with its cartilage, within the eyeball, or at the inner angle of the orbit. There is often lachrymation, with redness of the conjunctiva, and sometimes intolerance of light. There is sometimes so much tenderness that the patient cannot wear a hat, or even wash his forehead. Or he may be unable to blow his nose. If one or two hairs be drawn over this hyperæsthetic surface, it will be found that the tactile discrimination is diminished, as compared with the corresponding region of the opposite side. The pain is sometimes described as shooting into the upper eyelid, or going between the eyeball and the cavity of the orbit, extending thence over the brow, as though the forehead were being slit open. Pressure upon the parietal eminence will send a sort of heavy dull shock into the eye. To neuralgia of this division, in consequence of the pain being limited to one-half of the anterior aspect of the head, the term *hemicrania* has been applied, whence the *migraine* of the French, and the vernacular 'megrim.' The neuralgic affection constitutes, however, only a portion of the complex group of phenomena to which the term 'migraine' is properly applicable, and which may include, besides, subjective sensations of dazzling lights or colours, often of a zigzag shape, transient hemiopia, vertigo, unilateral numbness and tingling of extremities, impairment of speech, nausea, and vomiting. See MEGRIM.

When the *superior maxillary division* is in fault, violent pain is experienced in the cheek-bone or jaw, or in both, points of tenderness being found at the site of emergence of the infra-orbital nerve, over the malar bone, or on the gum of the upper jaw. The pain may be so definitely localised in one or more teeth that these may be thought to be the cause of the suffering. In these circumstances extraction of teeth is often performed quite uselessly. The teeth are found to be healthy. The attacks of pain are sometimes accompanied by profuse watery secretion from the nasal and buccal mucous membranes. There may be swelling and acute sensitiveness of the lip and nostril, the slightest contact with which causes pain to shoot widely in various directions, sometimes appearing to affect distant parts of the body. Acute pain may also occur in the parotid gland, accompanied by a great flow of saliva.

When the *third division* is attacked, points of intensity may be found on the temple, a little in front of the ear, at the place of exit of the inferior dental nerve, at the side of the tip of the tongue, or more rarely in the lower lip. The writer has met with cases in which neuralgia of this division, attacking both sides of the lower jaw and the under-surface of both borders of the tongue, occurred in plunges of agony which caused the patient to utter a kind of shrieking groan, and the head was jerked

convulsively. Such attacks may be brought on by chewing, swallowing, and even by speaking. In other cases the pain may be entirely localised in one side of the tongue-tip, but here at times it is so intense that the patient rolls on the floor in agony. To cases of this kind, marked by lightning-like seizures of exquisite character, and accompanied by spasmodic movements of the facial muscles, the term *epileptiform neuralgia* is sometimes applied. Hereditary tendency to insanity sometimes accompanies this form. The lingual branch of the fifth is happily not so often affected as the other portions, for there is probably no form of neuralgia involving more exquisite suffering. When the auriculo-temporal division is affected, pain is situated in the outer auditory meatus and temple. Some cases of so-called *erache* are doubtless examples of neuralgia affecting this nerve.

There is a variety of trigeminal neuralgia which is known as *clavus hystericus*, and which occurs chiefly, but by no means solely, in females, and affects the period of bodily development. It is characterised by intense pain, limited to one or two small points (the parietal most often, or supra-orbital), and resembling the driving of a nail into the skull. It occurs most often in the anæmic, and has sometimes been mistaken, with unfortunate results, for some inflammatory affection demanding depletory measures.

In severe tic-douloureux the facial muscles, those of the tongue, and even sometimes those rotating the head, may be spasmodically contracted by reflex action (*see FIFTH NERVE, Diseases of*). With supra-orbital neuralgia there may be blepharospasm or strabismus. The writer once saw a patient liable to neuralgia of the supra-orbital division for a period of twenty years, in whom, one year before, the attack began to be attended by complete ptosis and external strabismus, from paralysis of the third nerve. A certain amount of weakness continued between the attacks, but it was as an immediate sequel of the pain that the loss of power was most strongly marked. Two days after the cessation of the pain the eyelid could be raised fairly well and the eyeball moved, though never quite freely. Some degree of vaso-motor paralysis was shown by the reddening and heightened temperature of the face and swelling of the veins, which occurred unilaterally during the paroxysms of pain, as well as by the soreness of skin, indicating probably a temporary congestion which remained behind. To the same cause must be referred the extreme redness of the conjunctiva and mucous membrane of the nostrils, with increased lachrymation and flow of nasal and buccal secretion, often observed in this form of neuralgia. The hair is liable to be changed in colour about the seat of pain. There may be a permanent blanching, as of the greater part of an eyebrow or a small tuft upon the head, or the change may be a fluctuating one, the colour returning during the intermissions of the disease. Individual hairs in the district of the affected nerve may be hypertrophied, or the converse may happen; and during the persistence of attacks the hair may become brittle and fall out, to return, however, when the neuralgia subsides. Anstie described a state of thickening, the result of subacute inflammation, in the periosteum of bone and in fibrous fasciæ in the neighbourhood of the painful points of neuralgic nerves. Pressure on these swellings may not merely excite pains in the

affected branches, but send a powerful reflex influence through the cord to distant organs, causing vomiting, or affecting the action of the heart. The skin is apt to grow coarse; and patches of pigment to occur in the painful situations. In neuralgia of the second and third divisions the corresponding half of the tongue is sometimes seen to be covered with fur from overgrowth of epithelium, and this even when the process of mastication has taken place equally on each side; or there may be salivation severe enough to cause a suspicion of mercurial action, but which may be distinguished by its being unilateral. Acute inflammation of the skin, in the form of herpes, sometimes attends neuralgia of the fifth, especially its first division; and the eyeball itself may become in similar circumstances the seat of serious inflammatory action in one or other of its tissues. There may be a profuse and extensive eruption of herpes, leaving cicatrices which suggest confluent small-pox. In such a case neuralgic pains may cease during the eruption, to recur with great violence while this is scabbing. Acute glaucoma is attended by symptoms which appear to refer it to trophic changes consequent on neuralgia of the ophthalmic division of the fifth. There appears reason to think that recurrent iritis may sometimes be related to neuralgia or some kindred affection of the same division. Common sensation is frequently blunted during and after paroxysms of tic; but occasionally there will be so much and such persistent hyperæsthesia of the skin, that the patient cannot bear the face to be washed. In epileptiform tic the sufferer will often be observed to rub violently with a handkerchief the part of the face affected; but, on the other hand, there are cases in which not even the touch of a light bonnet can be borne, so exquisitely sensitive is a portion of the scalp. A touch with the finger upon this locality will sometimes cause the patient to fall to the ground.

DIAGNOSIS.—The paroxysmal character of the pains, coupled with the tenderness on pressing various points, sufficiently indicates tic-douloureux. The only condition with which it is at all easily confounded is the painful anaesthesia which is apt to be an early symptom of the encroachment of a tumour upon, or some other destructive lesion of the trunk of the nerve within the cranium (*see FIFTH NERVE, Diseases of*). The presence of the pain will distinguish the spasmodic contractions of the facial muscles secondary to neuralgia from mimetic spasm proper. There is no doubt that neuralgia about the forehead has been often mistaken for some inflammatory intracranial mischief, and this is peculiarly liable to happen where either ptosis or strabismus forms part of the symptoms. Careful examination, bearing in mind the points of diagnosis described, ought to obviate error. Still more important is it to avoid the error of ascribing to this form of neuralgia the pain occasioned by the growth of intracranial tumour (*see BRAIN, Tumours and New-Growths of*). It must be remembered that pus in the antrum may occasion neuralgic symptoms; that a fruitful source of trigeminal neuralgia is caries of the teeth; and that careful examination should be made for any disease of the nares.

DURATION AND PROGNOSIS.—Tic-douloureux occurring in youth and apparently as an accident of exposure, or as a result of faulty teeth, may never recur. It is perhaps more common, however, for repetitions of the attack to take place, alternating, it

may be, with neuralgia in other quarters. Tic is not infrequently liable to recur, especially under circumstances of depression, through a whole lifetime, but it may never have the character of extreme severity. In certain few cases, however, it is not only obstinate, but of terrible violence, the patient being incapacitated through many years by the constantly recurring affection. As a rule the disease, however severe the agony entailed by it, does not seem of itself to shorten the duration of the life which it fills with suffering.

TREATMENT.—As in neuralgia generally, the treatment of tic is partly constitutional, and partly local and palliative of suffering (*see* NEURALGIA). The first care in a case should be to have the state of the teeth accurately investigated, and faulty teeth removed or otherwise treated. It may happen that a tooth betraying no outward signs of decay is carious internally, and may thus easily escape recognition. The state of the ear and the nares demands equally careful attention.

As a local means of palliation blisters are often of great service. A small one should be applied over the branches of the cervico-occipital nerve at the nape of the neck, and repeated if necessary. The constant voltaic current is sometimes useful. A sufficient number of cells (from four to eight or ten) should be employed to give such a current as causes the negative pole to impart a very distinct burning sensation. The circuit should never be abruptly opened by the removal of the rheophore; a gradual heightening and lowering of the strength, respectively, should take place, both on applying and when leaving off, to avoid a shock. The rheophores should be fitted with sponges well moistened with salt and water, or with carbons plunged for a minute or two in hot water. The application may be continued for five minutes at a time, and be repeated at very short intervals indeed, it may be for some hours. The medicinal treatment of tic embraces the employment of numerous drugs among which the synthetical compounds have acquired an important place not only on account of their efficacy in relieving pain, but also because they are to a considerable extent free from the objections attending the use of opium and its derivatives. In some cases of tic a combination of 20 or 30 grains of chloride of ammonium with 5 drops of liquid extract of opium, taken two or three times a day, is singularly efficacious. Butyl-chloral hydrate—2 to 4 grains in a pill taken every two hours for a few times—may be sometimes employed with advantage. So, also, the tincture of Gelsemium sempervirens, given in doses of 5 to 15 minims every two hours, till relief is obtained. This remedy requires close observation. Phenazone, in doses from 5 grains every half-hour, or from 10 to 20 grains every four or five hours for a few times, is sometimes particularly useful. So also phenacetin in doses of from 10 to 15 grains with 3 or 4 grains of caffeine will often give great palliation. Salicylate of sodium 10 grains, with 5 grains of iodide of sodium and 5-10 of phenazone 3 times a day, is often very useful. The hydrate of chloral in a dose of 20 grains will occasionally be of great service, if the pain is not very severe, in procuring sleep from which the patient wakes relieved. If, however, the pain be severe, chloral hydrate is useless. It is important not to confound the epithelial overgrowth of the tongue, which so commonly occurs in tic, with the ordinary furring from digestive difficulties

which is supposed to indicate a necessity for purgatives. It will be observed that it only affects a lateral half. The hypodermic injection of morphine should be avoided except in cases of extremely intractable character in which the employment of other drugs has proved useless.

It is in cases of this class that the aid of the surgeon may be invoked with advantage. Stretching and resection of the affected branch of nerve will often procure immunity from suffering for periods varying from a few months to a year or two. Where all three branches of the nerve are involved it is sometimes found necessary to remove the Gasserian ganglion, an operation which in recent years has found considerable favour.

T. BUZZARD.

TIN, Poisoning by.—**SYNON.**: Fr. *Em-poisonnement par l'Étain*; Ger. *Zinnvergiftung*.

The importance of tin as a toxic agent has only comparatively recently been recognised; for although attention was drawn nearly a century ago to the possible danger attending the use of tinned vessels for culinary purposes, the danger was supposed to be due simply to the contamination of the metal with arsenic. Subsequently alarm was excited in consequence of the employment of alloys of tin for the storage of preserved articles of food, such as meat, vegetables, and fruits; but here again the danger has been assigned to the lead with which the tin is alloyed, a lead-tin alloy being used for the construction of the capsules employed. Attention has also been directed to the tin with which tinned foods and fruits are almost invariably contaminated, as itself presenting a source of danger to the consumer. It is known that the soluble salts of tin, met with in commerce as *Dyer's salt*, *pink salt*, &c., are poisonous; and experiments on animals show that even the insoluble hydrated oxides of tin are fatally poisonous, the oxides being no doubt dissolved in the alimentary fluids, tin having been detected after death in such cases in the liver and other solid viscera. Certain kinds of sugar are prepared by a process which introduces tin to the extent of from one-fourth to one-third of a grain per lb. of sugar into the manufactured article; but it is not known that these sugars are deleterious.

ANATOMICAL CHARACTERS.—The *post-mortem* appearances in fatal poisoning by tin are those of the mineral irritants. The cases observed have been too rare to be spoken of otherwise than generally. *See* POISONS.

SYMPTOMS.—Of these but little is known. Concentrated solutions of stannous chloride and of stannic chloride—the lower and higher chlorides respectively of the metal—are known to act as irritant poisons. Neurotic symptoms are also manifested, so that tin-salts do not appear to act simply as irritants. In large doses the chlorides produce fatal results. Probably the toxic results are heightened by the free hydrochloric acid invariably present in the commercial solutions of stannous and stannic chlorides.

TREATMENT.—The treatment to be employed is that for mineral acids. *See* POISONS.

THOMAS STEVENSON.

TINEA (*tinea*, a moth-worm).—**SYNON.**: Ring-worm.

DEFINITION.—Contagious affections of the epidermic structures of man and some animals, caused

by the growth of various species of fungi, the *Microspora* and the *Trichophyta*. In addition to the various groups of bacteria (cocci, bacilli) recognised as pathogenic vegetable parasites of the human integument and its appendages, the hair and nails, there are certain cryptogamic organisms which contribute to make up the group of dermatophytes. The botanical place of some of these cryptogams is not definitely established, but we may mention in order the parasite causative of *Botryomycosis*; the *Streptothrix* or *Oospora actinomyces*, associated with the various forms of *Actinomycosis* and *Madura Foot*; the *Microsporon furfur* (Eichstedt) causing *Pityriasis (Tinea) versicolor*; the *Microsporon minutissimum* (Burkhardt) causing *Erythrasma*; the parasite associated with the hair-affection called *Piedra*; the *Achorions* of *Favus (Tinea favosa)*; and lastly the fungi, varieties of *Microsporon* and *Trichophyton*, causing respectively the *Tinea tonsurans* with little spores (Gruby-Sabouraud) and *Tinea trichophytina*, both of which are popularly known as *Ringworms*.

The term *Tinea* (Fr. *teigne*) was formerly in vogue to denominate a great variety of affections of the scalp, but is now used as a generic term to denote the various disorders excited by the *Achorion*, the *Microsporon Audouini*, and the *Trichophyton-fungi*. *Pityriasis versicolor* is frequently included under this generic term, and *Erythrasma* should be so likewise.

GENERAL REMARKS ON THE RINGWORMS.—A few years after Bassi and Audouin had discovered the *Muscardine* disease of the silkworm, and so prompted Schönlein's investigations, by which he recognised the *Achorion-fungus*, Malmsten found a chain-fungus in ringworm, and Gruby differentiated the three types of infection which we recognise to-day. The importance of Gruby's researches from one cause and another was not appreciated, and until recent years all the ringworms were believed to be caused by one kind of fungus (called *Trichophyton tonsurans*). Differences in the size of the spores and construction of the mycelium were attributed to differences of soil, &c. Chiefly owing to the brilliant researches of Sabouraud the whole subject has been elucidated and placed on a firm basis. We now distinguish, by the objective features of the ringworm-lesions and by the microscopical examination of the scales and hairs, by the special features of cultures on artificial media, and by microscopical examination of these cultures, one group of ringworms associated with the growth of different species of *Microsporon*, and another due to species of *Trichophyton*.

As to the source of these fungi, some appear to be exclusively human parasites. Others are inoculated directly or indirectly from animals, as the horse, cattle, dog, cat, birds, hedgehog, &c.; all these are liable to set up considerable inflammation. We may find a particular species constantly associated with some species of animal, perhaps because the animal in its life is frequently exposed to this special contagion, and passes it to its fellows. These animal ringworms are, however, often intercommunicable. There is also considerable evidence to support the view that these fungi pursue a saprophytic existence, and evidence has been adduced in favour of the theory that they are but larval forms of superior fungi.

The ringworms are *highly contagious*, directly or indirectly. The exclusively human forms (*Micro-*

sporon Audouini and the common school *Trichophyton megalosporon endothrix*), which comprise the bulk of the cases met with in London, are inoculated from person to person (chiefly children) by simple contact, or through the medium of infected caps, brushes, towels, pillow-cases, clothes, or hairdressers' appliances. Hence, institutions in which children are collected are fertile centres of contagion. Particular forms of ringworm may occur in single cases, or be endemic or epidemic.

Their *geographical distribution* is interesting, for the common microsporon of this country and France gets less frequent in Spain and Belgium, and disappears in Italy and Eastern Europe. A trichophyton giving a peculiar red-violet culture is rare in London and Paris, but forms the prevalent type in Parma, and so on.

Little is known of the *qualities of soil* favourable to the growth of the fungi in the human skin. The fungi differ. Some like children, some adults. The microsporon and common endothrix trichophyton especially attack children, and those with finer fair hair are perhaps preferred to those with coarser dark hair. There is no evidence, however, that any particular constitutional state predisposes to attack or favours the growth. In warm countries many forms of ringworm flourish under conditions of heat and moisture. Generally speaking, it may be said that these fungi occur mostly in the poorer classes or communities, where they are favoured by personal habits, and greater facilities for inoculation and persistence.

SYMPTOMS.—The ringworm-fungi give rise to characteristic lesions in the epidermic structures invaded, and when one fungus only was recognised it was customary to discuss the symptoms regionally. It will be seen, however, that all fungi do not affect all sites.

Ringworm of glabrous regions (commonly called *Tinea circinata*, from the character of the lesions) may be caused by all these fungi, and the latter grow excentrically and regularly, and set up an inflammatory reaction, which varies widely with different fungi. In the majority of cases, enlarging red perfectly rounded macules or slightly infiltrated areas are produced, either scaly or bearing superficial vesicles or pustules, with or without special implication of the follicles. The rate of extension varies, but is usually rapid, and the areas have a marked tendency to clear up in the centres, owing either to the lessened activity of the fungus or the tolerance of the tissues, so that the spreading margin (scaly, vesicular, or pustular) stands out prominently, and rings are formed, whence the name *ringworm*. Rarely concentric rings are formed. The follicles and lanugo-hairs may be involved. The *Microsporon Audouini* causes small and ephemeral macules, but some trichophyta form lesions of great extent, and may be persistent. The variations in aspect are mostly due to the qualities of the parasite and its products, and are to a great extent constant for each species of fungus. There may only be a single lesion, or by auto-inoculation many may arise and become confluent to form gyrate patterns. They usually occupy exposed regions, but may be more or less generalised. One or two lesions call for special remark. Some fungi, especially some trichophyta of animal origin, whether they are inoculated on the uncovered parts of the glabrous skin or in hairy regions, such as the scalp and beard, set up an *agminate pustular folliculitis*, which is

known as the *Kerion of Celsus*. The lesion commences by a firm cedematous lesion, simulating a furuncle, but less painful, situated around a pilosebaceous follicle, within which suppuration occurs. Pretty rapidly surrounding follicles become similarly involved, and a perfectly round, red, granuloma-like projecting swelling is evolved, which may attain 7-8 centimetres in diameter. The follicles exude pus freely, and a crust forms. The diseased hairs are detached from the papillæ and fall out, thus effecting a spontaneous cure of the lesions.

Again, there is a special phase affecting the inguinal regions primarily (*T. inguinalis*), and from the secondary complications brought about by scratching, &c., it has been included with erythrasma and some forms of superficial dermatitis under the generic term *Eczema marginatum*. This ringworm is apparently due to a special trichophyton-fungus, which does not infect the hairs, and it is notable that almost all the adult members of a household may be attacked at the same time. The ringworm begins as a small red itching spot, spreads downwards on the thigh by a marginal border, scaly or vesicular, and may also advance over the genitals, perineum, buttocks, and pubes. A similar state of things may be found in the axilla.

It will be convenient to refer here to the subject of *tropical ringworm*. The trichophyta are exceedingly common and flourish actively in damp warm atmospheres. In India there is a marked incidence in the rainy season, and the eruptions die down after a time, to reappear again in the following year. No doubt a number of different fungi are concerned, and possibly each country has its special flora; but the subject has not yet been worked out by modern methods of investigation. Though hairy regions may be affected, ringworm of glabrous skin seems to be far more common, and has monopolised attention. It occurs nearly always in adult life, and may attack any region; but in India and elsewhere it commences very frequently and has its headquarters in the inguinal region of the male, like the *T. inguinalis* of Europe, but in the hotter climates it is apt to spread far more widely and rapidly, to cover wide areas with confluent-margined patches from the knee upwards over the body. Sweating and scratching cause a secondary eczematoid dermatitis, and the intense irritation may be most distressing and sleep-destroying. Chronic patches may get much thickened and pigmented, and on the hands and feet may be intractable and present great difficulties in diagnosis. See p. 1717.

Ringworm of the Scalp, or *T. capitis*, usually has special objective features, because the circular or circinate rounded areas with excentric spread due to the growth of the fungi in the surface-epithelium, and comparable to *Tinea circinata* of glabrous skin, are ephemeral, and give place to the destruction of the hair which dominates the picture. We shall have occasion to describe in detail this hair-infection, which in this country is due in almost all cases to one of two kinds of fungus, the *Microsporon Audouini* or the common *endothrix trichophyton*. They affect children particularly, and very rarely the adult, but other forms of trichophyton may infect the adult scalp, and Surgeon-major Garden wrote in 1876 that ringworm of the scalp was very common in the native army in India. Fungi of

animal origin may set up vesiculo-pustular (cat) or pustular infiltrated patches (cattle), and the *kerion*, which we have already described, is occasionally met with. Sabouraud thinks that kerion is invariably due to ectothrix fungi of animal origin, but English observers hold it may be associated with the microsporon, and the writer finds the usual kerion of children's scalps in London is due to an endothrix fungus.

Ringworm of the Beard (*T. barbae*, *T. sycosis*) is fairly common in England, and, as in the scalp, may commence by circinate lesions, though the hair-infection or the folliculitis produced are the striking features. As in the scalp, also, most varied appearances are brought about by different species of fungi. The most common picture is produced by multiple areas of agminated or conglomerate pustular folliculitis analogous to that described as kerion, and due to a horse-fungus. From the horse also comes a fungus which sets up a less degree of inflammation, but vesiculo-pustular and encrusted, sometimes simulating eczema or sycosis. Highly inflammatory lesions may be set up again by cattle-ringworm. In striking contrast there are phases in which the inflammatory symptoms are practically absent, so that we only see very short stumps studding the beard-area and the follicle-mouths surrounded by a little redness, or only scalliness. A bird-ringworm, with rose-coloured culture, has been isolated from these lesions, and another with a violet culture. The eyelids may be involved in rare cases.

Ringworm of the Nails (*T. unguium* or *Onychomycosis*) is invariably due, according to Sabouraud, to the various species of animal trichophyta. It is rare in this country and in Paris. The clinical characters are similar in all cases, viz. thickening, discolouration and friability of the nail. Exceptionally a single finger-nail is affected, more often several, irregularly and on both hands, rarely all the nails. The toe-nails may also suffer. See p. 1054.

MYCOLOGY.—*Cultures* of the fungi can be obtained on suitable media from the epidermic lesions, and the same type of malady be reproduced by inoculation of the cultures. In culture each family has its formal and proper biological characters, and by culture each fixed botanical species can be differentiated, for 'a given fungus upon a given medium of culture takes a form personal, specific and invariable' (Sabouraud). The technique for the isolation and pure cultivation of these fungi, the composition of the media, and the methods of inoculation in animals, require detailed study, and the reader is referred to special works for information. As to *mycology*, the ringworms belong to the *Mucedines* or *moulds*, and hence are filamentous fungi, reproducing themselves by external spores. Each filament is composed of cylindrical cells placed end to end, and the cells are composed of a cellulose envelope containing protoplasm. The filaments may be composed of long cells, giving the appearance of a tube partitioned at comparatively long intervals (*plain filaments*), or of short cells, and then oval, round or square, forming a chain-like or necklace-like filament (*sporulated filaments*). A filament may break up into its constituent cells, which are then called *spores*, but they are *internal* or *mycelial spores*, and are not a part of true fructificative organs. The filaments of both sorts are commonly called *mycelia*, and the whole is the vegetative part of the fungus, which may be reproduced by any of its segments. Under favourable

conditions of culture the filamentous meshwork or *thallus* sends down root-like filaments (the mycelium proper of mycologists) into the substance on which the fungus grows, and emits to the free air aerial filaments. The latter give off special branches (*hyphæ*) bearing true *external spores* or *conidia*, which constitute the true organs of fructification, and on which mycologists largely depend for purposes of classification. In the imperfectly aerated submerged portion of a culture, numerous metamorphoses of the filaments are found, some of which are regarded by some observers as various phases of reproductive organs, and of corresponding importance.

The study of these various changes in sections of the cultures is not satisfactory. The Continental method of examining the mycology of these ring-worm-fungi has been for the most part the 'hanging drop,' but by this method the aerial hyphæ tend to be imperfectly developed. Sabouraud by this method concluded that the microspora belong to an entirely different family from that of the trichophyta. Thus, in the latter, he describes the fructification as taking a bunch-of-grapes formation, while that of the microspora is on a different plan. Colcott Fox and Blaxall, by making *Klatsch* or impression-specimens of the aerial filaments from plate-cultures, demonstrated that both the microspora and trichophyta were closely related by these organs of fructification. In each group delicate hyphæ form, supporting external spores or conidia, and though these spore-bearing organs differ slightly in detail, the plan is similar in all.

For immediate *microscopical examination* a suspected *hair* should be carefully extracted, if possible entire. Should the root-sheath come away with the hair, the relations of the fungus are very completely shown. The hair, from which grease can be extracted by ether, should be immersed in a drop of Liquor Potassæ, B.P., on a glass slide, and care must be taken not to press down the cover-slip unduly or the relations of the fungus to the hair are disturbed. The clearing of the specimen should be watched. A low power will display the general aspect, and a magnifying power of about 800 diameters is desirable for a study of details. If it is desired to preserve the specimen, the potash can be washed out with a 15-per-cent. solution of spirit and water before staining. Sabouraud advocates potash-solutions up to 40-per-cent. strength. *Scales* can be examined in a similar manner, after dissociation with needles. *Pus* can be investigated immediately, after being spread out between two glasses. *Nails* require preliminary powdering with a sterilised nail-file. The fungus is conveniently *stained* on a slide by the following method. Stain in gentian-violet aniline-water and fix in Gram's iodine-solution; decolourise in aniline-oil, watching the process; dissolve out the aniline-oil with xylol and mount in Canada balsam in xylol. The microsporon-fungus stains well in a few minutes, and the epithelial tissue needs, therefore, less decolourisation; but the endothrix trichophyton requires a prolonged soaking, sometimes one hour, and a corresponding decolourisation of the hair, which may be advanced by mixing a drop of nitric acid with a few drops of aniline-oil, and afterwards soaking the acid out with pure aniline-oil.

A few words may be added on *the method of examining the scalp*. The patient should be placed in a good light, and with the blade of a pair of

epilating forceps the hair should be turned over in a direction against the lie of the hair. The elastic healthy hair flies back to its place, while the diseased stumps do not. The operator should use a magnifying glass, preferably a watchmaker's glass, as it leaves both hands free. In this way, with a little practice, the eye detects any signs of disorder in the scalp, such as scales, and the presence of any hair which departs from the normal in length, shape, or colour. Scales should be lifted, as they often hide very short or incurved stumps.

DIAGNOSIS.—From a clinical point of view, a lengthy section might be devoted to the diagnosis. In the first place, all round or circinate, scaly or congestive, or vesiculo-pustular eruptions must be considered, and these would include pityriasis alba, eczema seborrhoicum, erythema multiforme, psoriasis, lupus, syphilides, leprides, circinate impetigo contagiosa, &c. Further, we have to consider the more marked inflammatory phases, and alopecia, with its atrophied 'note of exclamation' stumps. In untreated cases the task is comparatively easy, but the difficulty arises when complications such as eczema mask the typical features, or when the areas of disease are broken up and defaced by treatment, or, lastly, towards the termination of a scalp-case, when only a few isolated stumps crop up. It is essential for a correct diagnosis that the observer should be thoroughly skilled in the detection of the fungus and the characteristically diseased *hairs*, for without this no amount of detailed description will avail.

Tinea due to Microspora (*Gruby-Sabouraud*).—This ringworm contributes something like 90 per cent. of the cases of ringworm of the scalp in London, and most cases are due to the fungus described by Gruby as *Microsporon Audouini*. A microsporon, however, has been cultivated from the horse, and another from the dog, which may be contracted by human beings. So also a ringworm derived from the cat (Fox and Blaxall) is not infrequent in the child, and is probably a microsporon. The so-called *Microsporon minutissimum* of Erythrasma and the *Microsporon furfur* of Pityriasis versicolor do not belong to this family.

The *Microsporon*-family is easily distinguished by the objective characters of the eruption and of the diseased hairs, by the morphological characters of the parasite in the lesions, and by the cultural peculiarities of the fungi.

The *Microsporon Audouini* is apparently exclusively human, and attacks the scalp-hair with avidity (*T. tonsurans*), the glabrous skin only slightly (*T. circinata*), *but never the beard or nails*. It is rarely contracted before three, and more rarely still after fourteen, years of age. Puberty seems to determine its extinction, but within these limits its duration is apt to be prolonged over years, during which period it remains highly contagious.

The characteristic of this ringworm is the predilection for the scalp and the formation there of considerable, more or less rounded, scaly areas, over which almost every hair becomes invaded by the fungus and thus rendered fragile, lustreless, decolourised, and broken off about 5-7 millimetres above the skin. Hence the qualifying term *tonsurans* applied to this and other ringworms in which the scalp-hairs are so diseased. These stumps may be straight, bent, or twisted by entanglement in the scales, and lie about in different directions. One can extract between the fingers a bunch of diseased

stumps which break off in the follicle, leaving a portion of the diseased hair. A further distinctive feature is a grey sheath on the stump extending upwards for about 3 millimetres from the mouth of the follicle. This adherent sheath is due to a mosaic of spores, and must not be confounded with an occasional sheath of epithelial scales. When the hair is cut in a typical case the pattern of one or more large parent-patches with numerous satellites in all stages of evolution is notable. Sometimes when the case comes under observation there may be only a single patch, or at other times by dissemination and confluence of patches the greater part of the scalp may be involved. The degree of inflammation set up is very slight, and the areas are only occasionally reddened and ringed. In the scalp the marked inflammatory effects, however, known as *kerion*, and lesser degrees, may on rare occasions be associated with this family of fungi, possibly due to members of animal origin. Lastly, the objective features may be masked on the one hand by the effects of scratching, weeping eczema, seborrhoea, and pus-inoculation from pediculosis, and, on the other hand, stumps may be worn down by rubbing, scales removed by treatment, and towards the end of a case only a few hairs or perhaps only a single diseased hair, and that a comparatively long one, may remain. It is, therefore, obvious that the medical man must be thoroughly versed in the detection of the diseased hairs.

On the *glabrous skin* reddened scaly rounded macules, sometimes ringed, are not infrequently seen about the face, neck, and shoulders at the outset, and in the course of neglected cases, but tend to die away spontaneously. M. Audouini does not care about the glabrous skin, but the species isolated from the cat by Fox and Blaxall cause striking multiple circinate lesions of the skin. Adults in intimate contact with the children may also contract a lesion, but the scalp is very rarely affected in them.

The *microscopical appearances* of the diseased hairs are also highly characteristic. On the surface of the bulb will be found columns, two or three deep, of spores extending upwards towards the shaft, and at the lower part of the latter they join a dense mosaic of spores, each 2-3 μ diameter and polyhedral from mutual pressure. This mosaic adheres to, and encases, the hair, and, as we have pointed out, extends beyond the intrafollicular portion of the shaft some 3-5 millimetres. Beyond that again to the free end a few collections of spores will be found dotted over the shaft. Careful focusing will convince the observer that these spores are outside the hair. Beneath this sheath, however, and in the hair, a certain amount of mycelium is found. It ends, except in some dead hairs, just above the bulb in clubbed feelers forming a well-marked fringe. It is more or less obscured higher up by the mosaic of spores, but characteristic little pieces with irregular branching are seen again towards the free end. The mycelial threads are generally plain, but occasionally more or less sporulated. It should be stated that Sabouraud describes the mycelium in the hair as giving off lateral branches, and these others, until very fine ones penetrate the cuticle of the hair and end by giving rise to a series of spores attached to one side of the branch (an organ of fructification). The writer controverts this view.

Tinea due to *Megalospora* (*Trichophyta*, *Trichophytosis*; Fr. *Trichophytie*).—The *trichophyton*-fungi form a family of which many different fixed species are already known. Some are rare, and as investigations proceed in other countries no doubt a considerable list will be formed. We already know that some are specially prevalent in particular countries or places. These trichophyta are arranged in two great groups. In one (*Endothrix*) the fungus attacking a hair becomes localised exclusively within such hair. In the other the fungus either remains outside the hair in its intrafollicular portion (*Ectothrix*), perhaps invading the epithelium of the follicle, or also enters the hair and is found both within and without (*Endo-ectothrix*). The writer doubts the existence of a pure ectothrix type. Some fungi, however, as in the case of the *Microsporon furfur*, do not appear to attack the hair at all, e.g. the one causing *T. inguinalis*. Only certain varieties attack the nails. When the hairs are infected they are rendered fragile and break off to form stumps, as in the microsporon-infection.

The mycelium of the trichophyta can be easily distinguished from that of the microspora, in that it is for the most part sporulated and forms characteristic beaded filaments, and almost exclusively so in the hair and nail. Then again, the diameter, and consequently the size, of the 'spores' is invariably larger (4-5 μ to 8-9 μ), and the qualification *megalosporon* applies with few exceptions. In some endo-ectothrix species a marked variation in the size of the spores is noticed.

The trichophytic lesions appear under two aspects—one practically without inflammatory reaction, so that in the regions of the scalp and beard the hair-lesions are the prominent feature; the other characterised habitually by more or less marked inflammation, which is focussed about the follicles and makes the hair-infection a secondary feature. An endothrix trichophyton, which with the *Microsporon Audouini* causes the bulk of the ringworm in children, is, like the latter, exclusively human. The rest are probably of animal origin, and among them are those causing the marked inflammatory reaction and pustular folliculitis.

***Endothrix Trichophytosis*.**—Special attention must be directed to the common *large-spored Endothrix trichophyton* of childhood, giving yellow crateriform cultures on Sabouraud's proof-medium. This fungus, which appears to be exclusively human, attacks the scalp and glabrous skin of exposed regions, but never the beard and nails (Sabouraud). It is almost as common in the elementary schools of Paris as the *Microsporon Audouini*, but among hospital out-patients in western London it forms only about 2 or 3 per cent. of the scalp-cases. In certain institutions, however, it is very common. It attacks children especially, but may survive puberty to perhaps twenty years of age. The glabrous skin of the adult may be occasionally involved, but not the scalp (Sabouraud).

The clinical picture is characteristic. More frequently than microsporon, this fungus causes multiple lesions of the *glabrous skin* (face, neck, and hands), and they are apt to be larger, but still small and ephemeral, more often circinate, and longer-lived. They begin as lenticular, prominent, rosy patches, punctate owing to more active congestion about the glandular orifices. They extend to form circular areas with raised red borders, finely scaly or faintly vesicular, while the centres fade with

some scaling. Let it be noted that the occurrence of such lesions where children are congregated always calls for a careful examination of the heads.

In the *scalp* the initial symptom is a rosy circinate lesion of rapid extension and short existence, so that it usually escapes observation. The fungus enters the follicles and then the hairs, but, contrary to the microsporon, it selects some hairs only. If an infected scalp be carefully and systematically examined, a number of little scaly areas will be found disseminated over the surface, each bearing a single stump or groups of two to ten darkened swollen stumps interspersed among healthy hairs. These stumps are so fragile that they tend to break off close to, or within, the mouth of the follicle ('black-dot ringworm'), and the desquamation present often hides the growing stump and causes it to become incurved or twisted in a spiral. Nevertheless, straight stumps, quite as long as those seen in microsporon, may be met with, and also light-coloured ones in fair-haired children. There are, therefore, no large *initial* patches, over which almost every hair is broken off, as in microsporon-infection. However, the small areas tend to enlarge and gradually to infect more and more hairs, so that in long-standing or severe cases the greater part of the scalp may be covered with large confluent areas.

Microscopical examination of scales discloses mycelial threads dividing dichotomously, some plain and others sporulated, the latter composed of equal-sized, rounded, or oblong segments, flattened at the junction with the next cell. *Hairs* in the early stages of infection show sporulated mycelium winding over the intrafollicular portion of the shaft and entering the hair beneath the cuticle. In the fully-infected hair all this outside fungus disappears, as may be ascertained with certainty when the root-sheath remains attached, and the shaft is found packed with monomorphic rectilinear sporulated filaments (4-6 μ broad). They terminate in a fringe above the bulb. The cuticle remains intact for a long time, and the chains do not break up readily. Thus the size of the cells, the chain-formation, and the exclusive location in the hair, except in the early stages, and the absence of a mosaic sheath clearly differentiate the picture from that of microsporon.

Sabouraud says that *other endothrix trichophyta* are met with exceptionally, and are only differentiated by culture, though there may be certain clinical differences, such as the formation of an initial large patch. In London there is a well-marked form of *kerion* of the child's scalp due to an endothrix trichophyton of uncertain origin, and the same or another may set up a vesiculo-pustular ringworm of glabrous regions.

Ectothrix and Endo-ectothrix Trichophyta.—These fungi are of animal origin, and those more commonly met with in this country set up marked inflammatory reaction about the follicles, culminating in the agminated pustular folliculitis which we have described as *kerion*. Thus there is an important group giving characteristic white cultures on Sabouraud's proof-medium. One isolated from the cat sets up vesiculo-pustular circinate lesions in man. A horse-specimen is not at all infrequent, especially in the beard, and causes raised inflammatory patches of agminated suppurating perifolliculitis. A third from the calf is a frequent cause of angry pustular ringworm of the hands, forearms, face, and neck in some country districts. Belonging to a second

category is another horse-fungus, setting up a lesser degree of inflammation, which may simulate crusted eczema. To a third group belongs a fungus contracted from birds, which sets up a very slight degree of dry inflammation. And, fourthly, a fungus of unknown origin simply affects the hair without causing any notable reaction. Lastly, we may mention a ringworm almost indistinguishable from the common endothrix, except that a large parent-patch is formed on the scalp, surrounded by satellites, each studded with swollen black stumps broken off in the follicles. Sabouraud indeed formerly described this as an endothrix fungus, and called the ringworm *peladoid*, from a certain simulation of Alopecia areata.

Microscopical examination discloses the sporulated filaments characteristic of the trichophyta. The constant presence of the fungus outside the hair in the follicle or in its wall will distinguish it from the endothrix group, and confusion can only arise with an endothrix-infection in its early stage before the fungus has died away from the follicle. The size of the fungus-elements and the almost exclusive presence of sporulated threads will facilitate the distinction from microsporon. Error can only arise from the dense massing of the filaments around the hair, so that the chain-formation is obscured. This sheath is not in mosaic. In rare cases, too, the fungus is comparatively small, and not strikingly larger than microsporon. The fungus has an intra-follicular location, and the sheath around the hair does not extend well beyond the follicle.

TREATMENT.—There is no method of dieting or internal medication known which will so alter the soil as perceptibly to retard or prevent the growth of the fungi, or directly kill the parasite. Obviously, however, it will be proper to correct any departure from the standard of health presented by the patient.

The spread of these diseases would be greatly curbed if all children were systematically inspected before admission to schools or other institutions, and at stated intervals afterwards. So also hair-dressers and barbers should take special precautions to prevent contamination.

Every child found to be suffering from ringworm should be isolated, as far as possible, from other children, and separate toilet-requisites should be provided and kept disinfected. The source of contagion should also be traced if possible.

The principles only which should govern the *local treatment* can be discussed here, as it would be impossible to give in detail all the remedies and methods so strongly recommended from time to time. We know that the fungi can be destroyed by many agents, for instance a culture is readily killed by formalin-vapour. The difficulty in effecting our purpose is almost entirely the problem of making the parasiticide penetrate to the fungus.

Ringworm of the glabrous skin can as a rule be readily exterminated, because the fungus grows in the superficial layers, and we can remove these by causing exfoliation, so that our remedies easily reach the parasite. Thus repeated painting with tincture of iodine is very successful, and any one of the numerous parasitocides can be applied in the form of lotions, liniments, pigments or ointments, care being taken to regulate the strength so that unnecessary pain or inflammatory reaction is avoided. Invertebrate areas of ringworm, as in chronic T. inguinalis, sometimes require a combination of remedies which

are resolvent and parasiticide, and cause desquamation. Goa powder in ointment, or moistened with citric acid or lemon-juice, is a favourite application for tropical ringworm.

Ringworm of the scalp is the phase which causes most trouble. The following general directions may be given: (1) Where the disease is limited to a single patch or two or three areas the hair should be kept cut well round these places. If, however, more patches exist—and careful search should be made—it is advisable to keep the hair over the whole scalp cut or clipped very short, leaving perhaps a bordering fringe for the sake of appearance, or even to shave, with antiseptic precautions, at intervals. The latter proceeding removes all disease above ground, so to speak, but the diseased areas must be previously noted or marked. Epilation of the zone of healthy hairs around the patches seems unnecessary. (2) The head should be thoroughly washed at the outset with soft soap, or some medicated soap, and all scales and crusts removed. Subsequent washings are useful occasionally to cleanse out plugs from the follicles. (3) Fresh auto-inoculations should be prevented by rubbing over the whole scalp daily some parasiticide application, such as weak formalin-ointment or a glycerine of carbolic acid. (4) A light head-covering should be worn night and morning, and lined with waxed paper to prevent soiling of the cap, pillows, &c. (5) The diseased areas can then be specially attacked. Various principles have been tried, such as the exclusion of oxygen, the application of parasiticides, and the removal of diseased hairs (*a*) by causing them to atrophy, (*b*) by withdrawing them (*epilation*), (*c*) by causing their fall by setting up perifolliculitis.

With regard to *atrophy of the hairs*, this happily occurs now and again accidentally, so to speak, but cannot be produced at will. It has most commonly followed a treatment by a thorough daily washing with soap and water and the mopping in of Cayley's boracic-acid lotion ($\frac{1}{2}$ Acid. Borici, $\frac{3}{4}$ vel q.s.; Etheris Methylati, $\frac{3}{4}$ x; Olei Rosmarini, $\frac{1}{2}$ j; Spiritum Methylatum, ad $\frac{3}{4}$ xl, eight times a day (Aldersmith); or the evil-smelling parasiticide gas-water sulphide of ammonium (in an excess of ammonia) dabbed on three or four times daily.

There is an endless number of parasiticide applications to select from, such as the perchloride, biniodide, oleate, nitrate, and oxides of mercury; the acetate, sulphate, and oleate of copper; silver nitrate; sulphur, sulphurous acid, hyposulphite of sodium, and iodide of sulphur; iodine-preparations; carbolic, salicylic, and acetic acids; chrysarobin, resorcin, β -naphthol, creosote, thymol and menthol, tar-preparations, formalin, &c. These can be used in various combinations and alternations, in the form of lotions, liniments, pigments, ointments, pastes, or plasters.

The selection of a parasiticide and its mode of application must depend on the age and sensitiveness of the child, the degree and character of inflammatory reaction produced, and the extent of diseased area. Thus equal parts of Ung. Sulphuris and Ung. Acid. Salicylici can be rubbed in freely for prolonged periods over extensive areas. Ung. Chrysarobin, however, will require care, and will often need weakening. A favourite ointment is one composed of an ounce of phenol, of olive oil, and of white wax respectively, two ounces of citrine ointment, and half an ounce of sublimed sulphur.

A pigment much used consists of equal parts of tincture and strong solution of iodine applied after the area has been cleansed and soaked with turpentine. When the coating gets too thick it can be removed with vaseline. A very important principle has now to be considered. We must carefully distinguish between the purely parasiticide and the inflammatory effects produced by applications. If we consider that the diseased hairs fit closely into the follicles, are infiltrated with fungus, and embedded to a considerable depth, we need not be surprised to find that there is an almost insuperable barrier to the penetration of the parasiticide into the whole extent of the hair. Consequently experience teaches us that, in contrast to what we see with ringworm of glabrous regions, it is almost impossible to cure ringworm of the scalp with purely parasiticide remedies; at any rate the treatment is very prolonged. Attempts have been made to surmount this difficulty by using media which are supposed to penetrate, such as glycerine, turpentine, ether, chloroform, acetone, benzol, &c., but avail us little. On the other hand the majority of parasiticides are irritants, and it is the constant irritation set up about the follicles which cures ringworm by causing the fall of the diseased hairs. Setting up mere desquamation or superficial serous catarrh is useless, and the latter to be avoided entirely. The ideal effect is to excite a deep-seated folliculitis. Unfortunately, scalps react so differently that we cannot easily command the effect we aim at. Thus with a saturated solution of Hepar Sulphuris in spirit very extensive cases may sometimes be rapidly cured by setting up folliculitis, but the remedy mostly fails. When we have to deal with a single patch—and extensive areas may be treated piecemeal—it is often convenient to set up considerable inflammation by parasiticide pigments, such as Coster's paste, composed of Iodine, $\frac{3}{4}$ j in $\frac{3}{4}$ vj of Rectified Oil of Tar, or Biniodide-of-Mercury pigment (Hydrarg. iodi rubri, gr. iv ad gr. vij, Sodii Iodid. $\frac{7}{8}$ ss, Sp. Chlorof. $\frac{3}{4}$ j ad $\frac{3}{4}$ iv, Aquam ad $\frac{3}{4}$ j). Or one of the following ointments may be used, viz. Ung. Cupri Oleatis ($\frac{3}{4}$ –iv to $\frac{3}{4}$ of base) or Ung. Hydrarg. Oleatis (Hydrarg. Oleatis, $\frac{3}{4}$ ss ad $\frac{3}{4}$ ss; Lanolin. Anhyd. $\frac{3}{4}$ ss ad $\frac{3}{4}$ j; Acidum Oleicum ad $\frac{3}{4}$ j) (Aldersmith). In this connection we will discuss the important principle of *epilation*. If the entire hair with its bulb, and preferably with its root-sheath, can be drawn out with forceps we have at hand a radical method of curing a patch. Every follicle so emptied is cured. Unfortunately we can, as a rule, only achieve our object when the hair-papilla is invaded by inflammatory exudation, and the attachment of the hair is weakened or completely severed. If the hairs break, epilation is a waste of time, but in the course of any case of scalp-ringworm it is well to try from time to time if the hairs can be drawn. When we excite deep-seated inflammation by the applications just mentioned, we greatly expedite the cure if the dried crust is carefully removed and the hairs epilated before renewing the application.

This brings us to a further development of this method. It is well known that ringworm-patches characterised by pustular folliculitis and the agminate lesions of it, known as kerion, heal spontaneously. The diseased hairs fall, and with the casting out of the fungus the source of inflammation is gone. Aldersmith, many years ago, elaborated a method

of exciting pustular folliculitis and kerion by the painless use of croton oil; and the writer has long been a keen upholder of the value of this method. Isolated patches, not bigger than half-a-crown, are painted with croton oil, and the area is continuously poulticed with linseed meal, which should be fixed on the part by elastic bands, and not allowed to shift. From time to time further applications of croton oil may be made, until a raised boggy area is formed, and then all the diseased hairs fall out or can be epilated entire. It is essential to prevent any accumulation of pus, and this should be bathed away as fast as it forms. The experienced hand never produces scars. In many cases the writer finds that the rubbing in of an ointment containing one drachm of croton oil to the ounce of parasiticide ointment does equally well, and is less risky in inexperienced hands. Fingernail-sized areas can be painted with the croton oil. Where single stumps are disseminated over the scalp, these can be removed by dipping a darning-needle in croton oil and needling the follicle.

In conclusion, it may be said that experience teaches that the expeditious cure of ringworm demands detailed attention on the part of the practitioner or some intelligent attendant whom he has instructed. Merely writing prescriptions and giving general directions end most unsatisfactorily to all concerned.

A cure can only be pronounced after new hair has grown, and repeated examinations have failed to disclose any isolated diseased hairs. The latter, though breaking easily, may sometimes escape recognition, as they tend to grow long and may cling to a healthy hair. Cases prematurely certified as cured are a frequent source of unpleasantness to all concerned.

Ringworm of the beard demands different treatment in different cases. Kerion, as elsewhere, soon subsides if boric-acid fomentations are applied and the surrounding skin is protected from auto-inoculations. The less inflamed lesions may require a strong ointment. The rarer cases without any inflammation are very obstinate, and the hairs need individual treatment by needling.

Ringworm of the nails is extremely tenacious, and may persist for many years. Dubreuilh says that the aspect of the nail is not a true guide to the extent of infection, for the fungus usually completely invades the nail. Treatment, to be efficacious, must attack the fungus by penetrating down to the matrix and well behind the subungual fold. Penetration of the parasiticides through the nail is practically impossible. The nails may be removed under chloroform, and a parasiticide applied, but even then it is essential to kill thoroughly any trace of fungus left under the subungual fold. C. Pellizzari and Dubreuilh advocate the plan of setting up a suppurative inflammation of the nail-bed, and so causing the fall of the nail. For this purpose an application composed of equal parts of pyrogallol acid and olive oil is made twice daily all round the nail. An indiarubber finger-stall increases the reaction. The operator must direct this treatment daily himself. Short of such strong measures there is nothing for it but to soften the nail by constant soaking in as strong a potash-solution as can be borne, and gradually file or scrape or cut away all the horny portion of the nail. When the quick is reached parasiticides can be applied. Radcliffe Crocker recommends the treatment advocated by

Harrison, of Bristol, for ringworm. Between the scrapings he applies a solution of Liquor Potassæ and Aqua Distillata $\bar{a}\bar{a}$ \bar{z} ss., Potass. Iodid. \bar{z} ss., oiled silk for fifteen minutes; this is immediately followed by a solution of Hydrarg. Perchlor. gr. iv, Sp. Vini Rectif. Aq. Dest. $\bar{a}\bar{a}$ \bar{z} ss. similarly applied for twenty-four hours. T. COLCOTT FOX.

TINEA FAVOSA.—SYNON.: *Favus* (Lat., the cells of honeycomb); *Dermato-mycosis achorina*; Fr. *Teigne favéuse*; Ger. *Erbgrind*.

DEFINITION.—A contagious affection of the skin of man and some animals caused by the growth of a fungus, the *Achorion Schönleinii* ($\acute{\alpha}\text{-}\chi\omega\rho\iota\omicron\nu$, from the difficulty in detecting the double-contoured cellulose wall).

SYMPTOMS.—Favus may attack the epidermic structures of hairy regions, glabrous parts, and nails, but the scalp is the most important, as it is the most frequent site affected, and on account of the intractability and destructive effects of the disease in this region. On the scalp the fungus may first extend in the epithelium, and occasion a circinate inflammatory eruption of mild intensity, but usually it rapidly finds its way into the hair-follicle and enters the epidermic wall and the hair. It develops with special luxuriance around the infundibulum, and soon occasions a little whitish elevation. In some weeks the latter enlarges to form a pathognomonic, sulphur-yellow, intra-epidermic, superficial, dry, umbilicated or cupuliform disc, which has been compared to a lupine-seed (the *godet favique*, *scutulum*, *favus*); lying in a red, moist, cup-like bed, fashioned by the pressure of the growth; and having a hair in the centre. Such a disc may remain isolated and on occasion attain a diameter of half an inch or more. The disease is, however, extensive, and other discs tend to form, but may remain disseminated, or become confluent into honeycomb-like masses. As these lesions enlarge and age, the overlying epithelium ruptures, their consistence becomes more friable, the yellow colour fades, the characteristic aspect is lost, and a mortar-like crust results. As they crumble and fall succeeding formations are less perfect, the growth of hair becomes less vigorous, and atrophic baldness results from pressure. The picture, however, is often marred and complicated by the effects of scratching and pustulation, perhaps from pediculi, and the production of ulceration and scars. It is remarkable that in the most extensive cases a fringe of hair is left around the head. A peculiar mousy smell is also noticeable, which must not be confounded with the stale scent of old dried-up pus.

From the follicle the fungus invades the *hair*, which gets lustreless and decolourised from the introduction of air, but does not become so disintegrated and broken off as in ringworm. When extracted, the hair usually comes away with its root-sheath. Such diseased hair may be found apart from *godets*.

It is now recognised that the formation of *favi* is not invariable. Three atypical forms of scalp-favus are especially distinguished (Dubreuilh, Sabouraud) in which *godets* and obviously infected hairs are absent. In an *impetiginiform* variety yellow impetigo-like crusts lead to a cicatrix and form around the latter. In a *pityriasis* variety irregular, defined, scaly patches of rapid extension are the feature. In a third *alopecic* variety, very difficult to diagnose, a quiet chronic follicular irritation causes the fall of the hairs and gives rise to a delicate cicatrix.

Glabrous regions may be attacked primarily, but generally follow the scalp-infection, and extensive areas may be involved. Here, too, favi form, surrounded by inflammatory halos, or are preceded by a circinate ringworm-like eruption (erythematous or vesico-pustular). One or several *nails* of the hands, or even of the feet, may be involved without favus elsewhere. The favi may form beneath, and gradually erode the nail, but usually there is a dystrophy indistinguishable from that of ringworm.

Kundrat has recorded a case in which the *mucous membrane* of the oesophagus and stomach was involved.

When the infected hair is soaked in Liq. Potassæ (7–20 per cent.) the fungus is found *microscopically* under two mycelial forms, and it is remarkable that in a given case all the hairs are infected by one type to the exclusion of the other (Sabouraud). In the one case the hair is nearly filled with parallel, non-flexuous tubes, from 2 to 3μ wide and partitioned at lengths of 12–15 μ , running in the direction of the long axis of the hair, dividing dichotomously, and staining with difficulty. In more frequent cases the mycelial tubes are comparatively scanty, very multi-form in each hair, and consist of flexuous filaments of all sizes, sporulated (mycelial endospores in chain-formation) or not, dividing by tri- and tetra-comy (*tarses faviques*), and as regards their protoplasm easily stained. The polymorphism of the filaments and of the segments when dissociated is a distinguishing feature of the fungus in all situations.

The *godet* consists of mycelial threads, like one another, packed side by side, with some granular débris, and is best displayed in a stained section, or the elements may be pressed out in 40-per-cent. potash-solution. The fungus ramifies in the epidermic wall of the follicle, and on a level with the infundibulum multiplies in the epidermis with great luxuriance to form a mass like the head of a daisy. The threads become more and more sporulated as they approach the surface.

For the complicated subject of the *culture* and *mycology* of the Achorian-fungus the reader is referred to Sabouraud ('La Pratique Dermatologique,' t. 1, 1900), and to Sabrazès ('Sur le Favus de l'Homme, de la Poule, et du Chien,' 1893). The conclusion reached by the former is that at present we are not in a position accurately to define the favus-fungus either clinically, culturally, or mycologically.

ÆTIOLOGY.—Favus may be met with at all ages, and a case has been known to last throughout life to eighty-four years of age. It rarely commences after twenty, and nearly always arises in children and in those living amid sordid surroundings. Cases in human beings are seen more frequently in the great centres of Scotland and Ireland than in England, where it is rare. In New York foreign immigrants contribute the greater proportion of cases.

Affected mice and fowls, however, are not rare in England. Favus is more common in France and especially in Holland, in Italy, among the male Jewish population of Russia, Poland, Galicia, and the Levant, and in the Mohammedans of Turkey, Asia Minor, Syria, Persia, Egypt, Algiers, and Morocco; also in parts of India, China, and Brazil.

Contagion occurs usually from child to child, and is exceptional from animals, though many cases are on record, and its possibility has been proved by

experiment with the fungus from the mouse and the dog (Sabrazès). The difficult study of the plurality of favus-fungi has given conflicting results. Some observers contend for the plurality in man, though corresponding clinical types are not identical. Sabrazès, after laborious work, has, however, isolated only one fungus in 'spontaneous human favus,' and this can reproduce *godets* in series in woman and in the mouse (an animal peculiarly susceptible to favus from all sources), and superficial ones in the rabbit and fowl. It determines a mycotic pseudo-tuberculosis when injected into the peritoneum of a guinea-pig. He finds also that a favus in the dog (*Oospora canina*, Cost-Sabrazès) is inoculable on the human being, and gives rise to a favic pseudo-tuberculosis when injected into the auricular veins of a rabbit. There is also a favus common in fowls. Their saprophytic existence is hypothetical, but the fungus can remain in a state of latent life for long periods. Sabouraud, without denying the plurality, has only found one species in over eighty cultivated cases. He is not quite convinced of the distinct favus of the dog and fowl.

DIAGNOSIS.—In the scalp in the absence of the pathognomonic favi the diagnosis may be difficult and only assured after repeated microscopical examination. Mortar-like crusts, lustreless decolourised hairs, a mousy odour, and irregular alopecic areas should always attract attention. Favus, however, may appear under such manifold guises and with so many complications that the possibility of this infection should be kept in mind in all chronic scaly, impetiginous, crusted, or scarring conditions of the scalp. The fungus itself, when found, is generally sufficiently characteristic for identification, but sometimes, as in the nails, cultivation can alone decide. See p. 1054.

TREATMENT.—Favus of the scalp and nails is a persistent and rebellious affection, and the problem presented is similar to that of ringworm of the same regions. Luckily the diseased hair is so little disintegrated that it can be extracted entire with its vitreous sheath, and the most experienced practitioners hold that epilation is the only effective means of cure. This operation must be systematically carried out. For the rest all crusts must be removed by boracic-starch poultices, or boracic acid fomentations, or soaking in carbolic oil, or curetting. All eczematous inflammation, excoriations, and ulcerations should be healed, but a degree of more deeply-seated inflammation or folliculitis may be encouraged as causing shedding of the hair. In addition to the epilation, the scalp should be treated with antiseptics and parasitocides as in ringworm. The treatment of the nails is also similar to that for ringworm of these parts. See pp. 1716 and 1054.

T. COLCOTT FOX.

TINEA IMBRICATA. — SYNON.: Solomon Island or Tokelau Ringworm; *Herpes Desquamans* (Turner).

DEFINITION.—An epiphytic skin-disease, produced by a fungus resembling the trichophyton, and characterised by concentric rings of imbricated scaly desquamation.

GEOGRAPHICAL DISTRIBUTION.—As far as is known, this disease is at present confined to the islands and countries included in the area within 140° W. and 95° E. longitude, and the north and south tropics; but, as it spreads with great rapidity

when introduced into districts with a favourable climate, it is not improbable that, as communication extends, it will in time become endemic in all tropical countries possessing a damp atmosphere and a temperature ranging between 70° and 90° F. In certain places within the endemic area it is exceedingly prevalent; in the Florida Islands, for example, where one-half, and the Treasury Islands, where four-fifths, of the inhabitants are affected (Guppy). Throughout the Malay country it is common, and cases have frequently been noted on the Chinese seaboard as far north as Foochow. It attacks foreigners as well as natives.

SYMPTOMS.—Usually the disease occupies a wide area of skin. In old-standing cases the entire integument, with the exception, perhaps, of the palms of the hands and the soles of the feet, the axillæ, crutch, and part of the hairy scalp, is covered with the characteristic thin, tissue-paper-like scales arranged in concentric parallel lines. These scales vary in size, according to their situation and the amount of friction they have been subjected to, from one-sixteenth to half an inch in breadth, by a quarter of an inch to one inch or more in length. They are firmly attached to and continuous with the healthy epidermis at the periphery or advancing edge of the circle they belong to, and free and ragged at the other edge. At and just beyond the line of their attachment, the skin is darkened from aggregation of the fungus, which, in its advance, is raising and throwing off the epidermis. The parallel lines of scales may be from an eighth to half an inch apart.

The pattern, so to speak, of the disease is readily understood from an inoculation-experiment. By rubbing scales from a case into abraded healthy skin the fungus is easily implanted. In about ten days thereafter it has so multiplied that it forms a brown layer underneath the epidermis. As this layer extends, the epidermis over its centre breaks down, and a scaly ring is formed. Gradually the ring so formed enlarges, and a second brown spot forms in the centre. This, in its turn, extends, its centre also breaking down. Then a third ring forms in the same way, and a fourth, and so on in never-ending series—like the waves made by a stone thrown into a pond.

Where two or more systems of rings impinge, a gyrated or scalloped pattern is produced.

Itching is sometimes complained of, but there is rarely any sign of inflammation of the skin, as in common ringworm.

The fungus is readily demonstrated by placing a scale moistened with liquor potassæ under the microscope. It closely resembles the trichophyton, but may be distinguished from it by its prodigious profusion, its position just under the epidermis, and, perhaps, by the presence in the interior of the conidia and mycelium of many brown particles. Unlike trichophyton, it has no special predilection for the hair or hair-follicles; in fact, it seems to thrive best on the non-hairy parts of the body.

Tribondeau describes the fungus as not a trichophyton, but as allied to aspergillus, and calls it lepidophyton; he states, however, that its full development is rarely seen in the body. He made unsuccessful attempts, using many media, to cultivate it outside the body.

DIAGNOSIS.—*Tinea imbricata* is distinguishable from ordinary ringworm by the size of the scales,

by the numerous, closely approximated, concentric parallel rings, by the absence of inflammatory redness, and by the extreme profusion of the fungus. From psoriasis and ichthyosis it is diagnosed by the presence of the fungus, the concentric rings, and by the attachment of the scales being at one edge only.

TREATMENT.—Painting the affected parts—taking a limb or part of the body at a time—with iodine-liniment is at once effective. All clothes must be boiled or destroyed, and relapses at once treated. In the labour-ships of the South Pacific, inunction of sulphur-ointment is the treatment adopted, and with success. Dr. Tribondeau finds treatment by chrysophanic acid to answer best.

PATRICK MANSON.

TINEA VERSICOLOR.—**SYNON.** : *Pityriasis versicolor*; *Chromophytosis*.

DEFINITION.—A chronic affection of the epidermis due to the presence of a fungus, the *Microsporon furfur* (Eichstedt) in the cuticle, characterised by a slight dissociation of the superficial scales and the formation of excentrically spreading macules, generally of a peculiar fawn colour.

DESCRIPTION.—*Tinea versicolor* mostly affects the covered parts of the body, and notably the trunk, whence it may extend to the groins, and rarely to the lower and upper extremities, and very rarely to the face. The colour of the lesions is most characteristic. It differs with the degree of pigmentation of the skin. In the fair skin it is a light fawn, in more pigmented Europeans it may be darker, and rare cases are on record in which the colour is a deep dirty brown. In coloured races the patches are grey. The presence of the fungus excites but little reaction, only some dissociation of scales, which becomes apparent on scraping a diseased area. Sometimes, however, especially if the body be unduly heated, the patches may appear more or less reddened. The eruption begins as punctate lesions about the follicles, and macules are formed by excentric spread. These coalesce and form extensive sheets, and a symmetrical distribution is effected. The macules are very rarely discoid or ringed. The hairs and nails are never affected. Itching is not very pronounced as a rule.

ÆTIOLOGY.—*Tinea versicolor* is due to the presence of a characteristic fungus, which loves warmth and moisture, and some other conditions of soil which are not yet well understood.

It is of more frequent occurrence in warm countries, and in Great Britain appears most often in those who clothe themselves warmly in flannel or wool. Hence it is particularly frequent in delicate people, such as phthisics. Its growth is fostered also in those who wear warm clothing by day and night, sweat freely, and do not wash their body thoroughly. The affection is very rare in childhood. It is only slightly contagious, but may affect husband and wife occasionally, or several members of a family.

Description of Fungus.—The fungus is very characteristic, and easily recognised under the microscope by soaking a scale in liquor potassæ. It consists of irregular clusters of mycelial spores (20–50) like fish-roe, each spore being 5–7 μ diameter, double-contoured, with a highly refractile nucleus, and often showing signs of budding. Proceeding from these clusters are short curved mycelial threads partitioned into long segments. The fungus

presents great difficulty in cultivation, and its mycology awaits study.

DIAGNOSIS.—Experience teaches that *Tinea versicolor* is most commonly confounded with the macular syphilide, though the colours are usually so distinct. The other trouble is the discoid and circinate eczema seborrhoicum (*Lichen annulatus* of Wilson) of the chest and back, which leaves dirty stains. *Pityriasis rosea* can hardly be confounded. Pigmentations such as 'liver spots' might also be mistaken. The microscope will readily decide in any doubtful case.

TREATMENT.—Parasitocides readily penetrate the superficial layers of the cuticle and kill the fungus, and desquamation removes the fungus mechanically. Repeated scrubbings with soap and water are therefore useful as a preliminary and accessory treatment. Besnier recommends the thorough application of the following ointment: Resorcin, Acid. Salicylici, āā gr. xv–xlv; Sulph. Precip. ʒss; Lanolin, Vaseline et Adipis, āā ʒvj. A more cleanly remedy is, however, often used, such as vinegar, followed by a lotion of hyposulphite of sodium (saturated solution), or that recommended by McCall Anderson (*Hydrarg. Bichloridi*, gr. xx, *Saponis Viridis*, ʒij, *Spiritus Rectificati*, ʒij; *Ol. Lavandulæ*, mxx). The vests should be boiled or disinfected with formalin. It is very important to keep up the treatment for some time, so as thoroughly to eradicate the fungus, or the eruption spreads again.

T. COLCOTT FOX.

TINKLING, METALLIC.—A sound of a peculiar quality, which the name sufficiently defines, occasionally heard on auscultation in connection with cavities in the lungs, or when air and fluid are present in the pleura. See PHYSICAL EXAMINATION.

TINNITUS (Lat.).—SYNON.: Fr. *Bourdonnement d'Oreilles*; Ger. *Summen*; *Klingen*.—Tinnitus, a term which is commonly used when speaking of noises in the ears, is a frequent symptom in many diseases of the external, middle, and inner ear. It is usually present in all those conditions where there is undue pressure on the labyrinth; for instance, when there is pressure on the tympanic membrane from cerumen, imperfect entrance of air into the tympanum, due to obstruction of the Eustachian tubes, or effusion within the tympanic cavity. It accompanies most inflammatory diseases of the external or middle ear; follows injuries to the tympanic membrane, and blows on the head or ear; may occur in aneurysm at the base of the skull; and is a prominent symptom in all nervous affections of the auditory apparatus, as well as in many states of disordered hearing where the ear is healthy. So infinite are the degrees and variations in this symptom, that there is probably no known sound to which it has not been compared by patients. When tinnitus is due to some curable local cause, the symptom rapidly disappears with its removal. When, however, it accompanies the deafness in nervous affections, it is often the more troublesome symptom of the two, and the less amenable to treatment. Strychnine is the most useful tonic in ear-affections, and quinine the least suitable, as this drug exercises a distinctly injurious effect, if taken in large doses. See EAR, Diseases of; HEARING, Disorders of; and VERTIGO.

W. B. DALBY.

TISANE (*ptisana*, peeled barley).—SYNON.: *Ptisan*.—A generic term applied, especially in France, to weak decoctions or infusions of barley and other farinaceous substances, whether used for domestic or medicinal purposes.

TITUBATION (*titubo*, I stagger).—A term for staggering or stumbling gait. See CEREBELLUM, Diseases of; and VERTIGO.

TOBACCO, Poisoning by.—SYNON.: Fr. *Empoisonnement par le Tabac*; Ger. *Tabakvergiftung*.—The minor effects of tobacco-poisoning—nausea, depression, vomiting, vertigo—are well known to the incipient smoker. Fatal poisoning by tobacco rarely occurs, except through its ignorant administration by mouth or rectum for the purpose of drugging. Cases of nicotine-poisoning are rare, though this, the volatile active alkaloid of tobacco, is now readily procurable for the purpose of fumigating plants.

ANATOMICAL CHARACTERS.—After death from tobacco-poisoning the organs and tissues have a tobacco-like odour, and the odour of nicotine becomes more pronounced on treating them with liquor potassæ. Turgescence of the brain has been described; but, beyond the odour, there is nothing diagnostic in the appearances.

SYMPTOMS.—When a strong decoction of tobacco or snuff is administered, either by mouth or rectum, very speedily—usually in about five minutes—the patient is seized with vertigo, acute abdominal pain, nausea, and vomiting. The skin is pallid and bathed in perspiration. Stupor supervenes, with partial or general convulsions, and stertorous respiration; and death may result in fifteen or twenty minutes, preceded by dilatation and insensibility of the pupils. When the alkaloid, nicotine, is swallowed, insensibility supervenes almost immediately; the pupils are widely dilated; respiration is speedily suspended; and the patient dies in three or four minutes.

The toxic effect on vision of the continued use of tobacco is described on p. 513, and that on the heart on p. 643.

DIAGNOSIS.—The odour, coupled with the above-described symptoms, would leave no doubt as to the nature of the case. Usually there is a history of administration.

PROGNOSIS.—The prognosis is in all cases of acute poisoning unfavourable. *Fatal dose.*—Thirty grains of tobacco are said to have proved fatal. A drop or two of nicotine would doubtless prove fatal.

TREATMENT.—The treatment of poisoning by tobacco consists in the exhibition of emetics, followed by tannic acid freely in any form, to render the alkaloid insoluble. Strong tea, coffee, and stimulants should also be administered. Iodine, dissolved with iodide of potassium, has been recommended; but is probably of little efficacy, and is undoubtedly irritating to the stomach. Twenty to thirty minims of tincture of nuxvomica may be given, and repeated at intervals; or, better, a hypodermic injection of $\frac{1}{32}$ grain of the nitrate or other soluble salt of strychnine may be administered.

THOMAS STEVENSON.

TOKELAU RINGWORM.—See TINEA IMBRI-CATA.

TONE, Want of.—This expression, although commonly employed in a somewhat loose and unscientific manner, is sufficiently understood in the main. To appreciate its meaning, it is first necessary to inquire what tone is. In the widest sense of the term, a person may be described as being *in tune* when his organs individually discharge their functions in a perfect manner, and act harmoniously as a whole, just as a violin is said to be *in tune* when melody can be educed by striking its individual strings. More correctly, tone is applied to a condition of the muscular system, as signifying that state of tension in which voluntary muscular efforts can be produced and continued with a healthy and pleasurable feeling; and also to muscular organs, to indicate a certain degree or power of contraction of their walls.

ÆTIOLOGY AND PATHOLOGY.—Various factors combine to produce and maintain muscular tone. First, this state is dependent on a proper supply of *nervous energy*; secondly, *nourishment* and *oxygen* must be furnished in abundance; thirdly, the *products of waste* must be thoroughly removed from the system; and fourthly, the *work* required of the muscular tissue must not be excessive, that is, a certain amount of rest must be afforded to it.

Want of muscular tone results, then, from failure of any of these conditions.

1. *Nervous failure.*—Whatever view may be taken of the nature of nervous and muscular force, modern research has shown that they are intimately connected with each other. If the nerve-supply be cut off, muscles waste; and *vice versâ*, if the muscles remain unemployed, their nerve-centres suffer. The effect of nervous disease or disorder in producing loss of muscular tone is illustrated by such neuroses as hysteria and epilepsy, by some forms of mental disease, and by the large but indefinite class of cases known as 'nervous debility,' of which mental strain, anxiety, sexual excess, masturbation or alcoholism are frequently the exciting causes.

2. *Failure of nutrition.*—Deficiency of the blood-supply immediately lowers muscular activity, in consequence of interference both with the nutrient and the oxygenating processes. It is on this account that want of tone is found in anæmia, in convalescence from acute diseases, and to a certain extent in chronic dyspepsia. Want of oxygen and impurity of the atmosphere lead as distinctly to lassitude; and, if long continued, to lowering of muscular energy and loss of tone, as among the inhabitants of large towns, and in persons employed in close, ill-ventilated rooms.

3. *Retention of waste products.*—When muscles are called into action certain compounds are formed within them, which must be eliminated by being passed back into the circulation and excreted. The lungs, skin, kidneys, liver, and bowels must, therefore, discharge their functions properly to keep the muscular system in tone. We have here the explanation of a very common class of cases of want of tone. Many persons, either from choice or from necessity, habitually take an amount of active bodily exercise insufficient for the removal of the waste products from the muscular and other systems. In the former case this is the result of indulgence in abundant rich food, combined with lazy habits, confinement to warm 'relaxing' rooms, and the avoidance of 'bracing' exercise. In the latter case the metabolic inactivity is refer-

able to enforced confinement in sedentary employments, often of an exhausting kind, carried on perhaps in an impure atmosphere, or throwing a continuous strain upon one set of muscles, such as those involved in standing or sitting. Both these classes of cases also are met with chiefly in large towns, and they constitute a considerable proportion of the persons who 'require tonics.'

4. *Muscular exhaustion.*—Excessive muscular exercise leads to loss of tone: first, by interfering with nutrition, which is most active during rest; secondly, by wear and tear; and thirdly, in the case of hollow muscular organs, such as the intestines (the muscular walls of which have to resist internal pressure), by gradual exhaustion of muscular irritability from continuous excitement, or possibly even by over-stretching and dislocation of the fibres. The first two forms of muscular atony are well illustrated by certain instances of cardiac exhaustion; the third form is met with not only in the alimentary canal, but in the bladder, in the blood-vessels, and indeed in all muscular tubes and ducts, when over-distended by solid, fluid, or gaseous contents.

5. *Combined causes.*—In many cases two or more of the causes mentioned under the preceding head are combined. Thus in a large class of cases of debility with which practitioners in large towns are familiar—and which especially includes young female subjects engaged in business—overwork, impure air, insufficient light, badly cooked or otherwise improper food, the constant strain of the muscles of the legs and back without sufficient movement, and frequently many other circumstances injurious to the nervous system, are all combined. Again, the subjects of chronic nervous affections, such as epilepsy and hysteria, are too frequently overfed, nursed in warm rooms, and spared every form of healthy exertion, with the result of producing a flabby, atonic state of system.

SYMPTOMS.—Want of tone in the muscular system generally is characterised by a number of symptoms, which are all more or less ill-defined and difficult to describe, being chiefly of a negative and subjective kind. The chief of these are a peculiar feeling of want of muscular vigour; weakness, heaviness, and even aching of the limbs; languor, inability and unwillingness to undertake or to continue any kind of physical or mental exertion, and a desire to remain passive and undisturbed. This reacts upon the mind, causing depression of spirits, melancholy, and other subjective symptoms.

The symptoms of want of tone or atony of muscular organs vary greatly with the part affected. Thus atony of the stomach is characterised by a familiar form of dyspepsia, which is called 'atonic'; atony of the bowels is chiefly attended by constipation and flatulence; atony of the bladder is associated with retention of urine. Ulcers are said to 'want tone' when the healing process flags.

TREATMENT.—It will be gathered from the preceding remarks that want of tone, whether general or local, is a condition which calls for very different kinds of treatment, according to its cause. One of the principal reasons of the want of success which frequently attends attempts to restore tone to the system is failure on the part of the practitioner to appreciate this truth, and to discover and remove the cause or causes of the morbid state. Tonic drugs suggest themselves only too readily as the proper means to be employed; and so great is the number of remedies which go by this name, and so

complex are the combinations in which they are now presented by the pharmacist, that recourse is often had to them before an accurate estimate has been made of the direction in which the system or the affected organ is really at fault, and hence injury, not benefit, results. These remarks apply both to loss of tone generally, and to atony of special organs. Thus it happens that the best tonic measure in one case may be rest, in another case exercise ; in a third case food and stimulants may be urgently called for ; in a fourth case depletory measures are essential at the commencement of treatment. Time is an equally important factor in the process of restoration to tone. This is especially true in the instances where rest is necessary ; but even in the very opposite class of cases, where exercise is demanded, this regimen may need to be carried on for a long time, being commenced with caution and slowly increased.

The details of tonic treatment are indicated in a special article (*see* TONICS). Here it need only be added that when the nervous elements are distinctly deficient in activity, strychnine, cinchona, phosphorus, and cod-liver oil appear to be specially indicated, while galvanism and massage may be useful. Massage is a most potent agent in increasing bodily tone. But it requires careful regulation, as in some patients it proves too exhausting—it may be from too quick destruction of the muscular tissues, and insufficient elimination of the waste products (*see* MASSAGE). On the other hand, Weir Mitchell's treatment, where massage is combined with forced feeding and seclusion, is of great value where failure of tone arises chiefly from a badly nourished nervous system. Alcoholic stimulants and iron are best adapted to cases in which nourishment and oxygenation have fallen below par. When the activity of the organs is diminished from accumulation of waste products, we must have recourse to moderate cholagogue purgation, to such diuretics as digitalis, to diaphoretics, and especially to change of occupation, and exercise of such a kind in an open healthy atmosphere as shall bring all the voluntary muscles into action, and stimulate if possible every bodily function. In the converse class of cases, where exhaustion is the result of over-exertion, we have, after removing the cause, to exhibit antispasmodic or even sedative drugs, such as belladonna and opium, which are especially useful in the first stage of atony of the stomach and alimentary canal, as well as in atony of the bladder.

Finally, no class of remedies is so likely to prove efficacious in restoring tone as that of mineral springs and baths. Where these are chosen with due discrimination, every one of the indications for treatment may be fulfilled. In *nervous failure*, whatever may have been the exciting cause, the change of air and scene, the regulated life and the freedom from daily cares, all tend to restore a proper working of the nervous system and thereby a due tone of the whole system. The same remarks apply to *failure of nutrition*, where early hours and exercise, which form a part of the regimen at every Spa, are so conducive to an improved tone of the muscles, voluntary and involuntary, of the body. But it is in cases of *retention of waste products* that natural mineral springs and baths play so important a part. Doubtless the principal agent is water, and the different saline or other ingredients are merely subsidiary helps. Yet even the mere flushing of the system is not to be despised ; and in no other fashion can it be so thoroughly—if not always very

pleasantly—secured. The fourth cause—*muscular exhaustion*—is one also which can be very successfully combated at a watering-place. One of the most necessary injunctions which require to be laid down by the bath-physician is attention to graduation of the amount of exercise. The improved sleep, too, following on better digestion, inhalation of pure air, and increased elimination, tends greatly to give tone to the system by ensuring sufficient intervals of rest. Enough has been said to show that mineral waters, bathing, and the modern system of massage available at all Spas, must do much to counteract the causes of 'want of tone.'

WILLIAM BRUCE.

TONGUE, The.—*SYNON.* : Fr. *La Langue* ; Ger. *Die Zunge*.—Apart from its own particular diseases, which are described in a separate article (*see* TONGUE, Diseases of), the tongue, as is well known, gives important clinical indications regarding various morbid conditions affecting the general system, as well as many local diseases.

In general terms it may be stated that we examine the tongue, for clinical purposes, with reference, first, to its *subjective sensations* ; secondly, to its *movements* ; and, thirdly, to the *objective characters* which it presents.

1. The *subjective sensations* chiefly include ordinary tactile sensation, and the sense of taste. In most cases we rely for information on these points upon the statements of patients ; but under certain circumstances common sensibility may be tested experimentally by some suitable instrument ; and taste, by applying different articles to the tongue with a brush, or in other ways. *See* p. 1065 ; and TASTE, Disorders of.

2. The *movements* of the tongue are studied by watching them directly ; and by noticing any abnormal affection of speech or deglutition arising from defect or disorder of these movements. A peculiar thickness of speech, or an inability to swallow properly, may result from this cause ; and in extreme cases articulation and deglutition may become impossible. To examine the tongue directly as a motor organ, it should first be looked at while in the mouth, both at rest and when the patient moves it from side to side, or in other directions ; then it should be directed to put it out, and to perform similar movements when the tongue is protruded. This mode of investigation gives important information in certain cases. *See* p. 1066.

3. The *objective examination* of the tongue in itself is of far more frequent application than the methods just considered, being indeed called for and commonly practised in every case, although it may not necessarily afford any positive information. The examination is usually carried out by inspection ; but it may also be requisite, and very instructive, to feel the tongue with the finger. In looking at the organ, an endeavour should be made to inspect its entire upper surface, and for this purpose the patient should be directed to open the mouth, and protrude the tongue as far as possible, a good light being also needed for observing it. In some instances, as in infants and rebellious children, as the result of congenital malformations or wearing false teeth, in many low febrile cases, and in certain nervous diseases, the tongue must be examined while in the mouth, as the patient either cannot or will not put it out properly ; for this purpose it is sometimes necessary to open the mouth

somewhat forcibly, but with due care, and it may be desirable to employ some artificial light. For more minute information in some cases it is requisite to scrape the surface of the organ, and to examine microscopically what is thus removed. The points to be noticed in the objective examination of the tongue are: (a) Its size and shape as a whole, as well as the characters of its point and margins. (b) Its condition as to firmness or flabbiness. (c) The colour of the mucous membrane. (d) Whether the surface is normal, smooth and glazed, being more or less denuded of its usual epithelium, or presenting a dry membranous covering, furrowed, fissured, or otherwise altered. (e) The condition and appearance of the papillæ, especially the fungiform papillæ. (f) Whether the tongue is moist, sticky, or dry. (g) The absence or presence of any accumulation on the dorsum of the organ, commonly known as 'fur'; and, if present, its arrangement, thickness, colour, and other general characters, as well as in some cases its microscopical appearances. It may be remarked that in relation to this examination of the tongue it is often advantageous to notice the condition of the mouth generally, but especially of the gums and teeth, and of the lips. Further, the patient may afford information with regard to the objective conditions of the tongue, not coming immediately under the notice of the practitioner, such as whether it is much furred on waking in the morning, or if it is inclined to dryness.

It is desirable to offer a few remarks respecting abnormal covering or *fur* on the tongue. This varies much in its extent, distribution, thickness, and characters. As regards its general distribution, it may cover the whole surface of the organ; or only its posterior or anterior part, one lateral half, or even a limited patch when due to local causes. The covering may be a mere film, or of considerable thickness. Its chief colours are white, whitish-yellow, yellow, yellowish-brown, brown, brownish-black, and black. It is either moist and easily separated, sticky and viscid, or dry, being then often cracked and peeling off in fragments. It usually clears gradually from the tip and sides towards the centre of the tongue, sometimes in strips; sometimes it breaks away abruptly in scales. Under certain circumstances fur appears and disappears with great rapidity. See p. 1709.

In a very instructive classification of tongues, Dickinson employs the following terms: (1) *Stippled* or *dotted*, where the papillæ are separately capped with a minute white patch. (2) *Coated*, where the spots coalesce and become confluent, so that the covering is continuous, the intervals between the papillæ being also more or less filled up. (3) *Plastered*, where the coat is thick, uniform, and conspicuous, and often looks as if laid on with a trowel. (4) *Furred* or *shaggy*, in which there is great elongation of the papillæ, which remain separate from each other, at least at their extremities, so as to give a shaggy look, or one suggestive of coarse hair or fur. (5) *Encrusted*, in which the papillæ are concealed by an incrustation, or thick felted coat, usually dry and brown, by which the surface is overlaid.

As regards the nature of the changes in the varieties of abnormal tongues just indicated, microscopical examination shows that in most of them the papillæ are elongated. The white coating is found to consist mainly of accumulated epithelium, which,

according to Dickinson, is the result chiefly of overgrowth. In the superficial layers of many accumulations on the surface of the tongue, especially the dark encrusted form, there are also present the remains of food, amorphous material, fat-globules, blood or altered blood and pigments, different micro-organisms, especially bacilli and micrococci, and sometimes the *Oidium albicans*. In the deeper parts of the epithelial structure, more especially in the Malpighian layer, there is often much profusion of nucleation or cell-growth, and the corium is in many cases distinctly hyper-nucleated and often hyperæmic. More rarely leucocytes are extruded within the papillæ and elsewhere (Dickinson).

With reference to the immediate causation of these abnormal conditions of the tongue, Dickinson concludes that the formation of more or less coating, with lengthening of the papillæ, depends partly on disuse of the organ, with want of rubbing and washing, and consequent deficient removal of the epithelium; but mainly on increased overgrowth, associated probably in some cases with a morbid poison in the system, but chiefly with pyrexia, the coat increasing in degree in proportion to the temperature, up to a certain point. Dryness of the tongue he attributes principally to deficient secretion of saliva; but it may also be due partly to habitual openness of the mouth during sleep, to pyrexia, or to general dehydration of the body, from deprivation of water or excessive discharge, as in diarrhoea and diabetes. The condition to which this observer specially applies the term 'furring,' as well as incrustation, he considers as essentially connected with want of saliva. Incrustation is a secondary process, in which various matters accumulate on the surface of the tongue, including parasites. The brown colour he refers chiefly to dryness, as many animal substances dry brown; partly to staining by food and medicines.

The condition of tongue in which its surface is abnormally smooth, bare, and denuded, also calls for brief notice. This may be general or partial, and usually follows the removal of a coating or incrustation; but it may be a primary change. It presents various degrees, the organ being at the same time more or less red, and generally dry. A glazed, shining, or polished appearance is common. In extreme cases this variety of tongue becomes intensely red and raw-looking, being compared to raw beef; and it may be excoriated or cracked. In describing the minute changes, Dickinson states that the epithelium and the papillæ are removed down to the level of the Malpighian layer, but this usually remains, and becomes covered with fresh horny epithelium, forming a thin membrane. Should the denuding process extend further, the Malpighian layer itself is removed, exposing the corium; and this structure is sometimes encroached upon, even almost down to the muscular fibres. Vascular injection, hypernucleation, and the extrusion of leucocytes often take place; and in some cases there is a general inflammatory infiltration of the superficial parts. A completely bare tongue is comparatively rare, and such a degree of change is generally limited in extent. The variety of tongue now under consideration is that on which aphthous growths are most liable to occur. It implies failing nutrition. The appearances are due partly to want of epithelial covering; but the exposure resulting from loss of this covering also

causes irritation and dryness. Dickinson attributes the changes partly to want of saliva.

CLINICAL INDICATIONS.—In the following remarks it is intended to call attention to some of the principal conditions in which the tongue affords useful information, and to indicate the main characters associated therewith. Dickinson affirms that 'the tongue is an index of constitutional states, seldom of individual diseases'; 'it seldom points to solitary organs or isolated disorders, but is rather a gauge of the effects of the disease upon the system than an indication as to the locality of it.' Allowance must always be made for individual peculiarities in the shape or size, and in the appearances presented by this organ; for the effects of certain habits, such as excessive smoking, or chewing tobacco and other substances; for changes in colour due to taking iron or other medicines; and for the results of any local irritation. Moreover, most people, but especially those who sleep with the mouth open, have a more or less furred tongue in the morning, which is of no special importance, or it may tend to dryness; while in some individuals its surface presents constantly a thick coating, without evident disturbance of any organ, appetite being excellent, and the digestive functions performed in a most satisfactory manner. On the other hand, a perfectly clean and healthy-looking tongue may be associated with severe dyspeptic symptoms, or even with serious organic disease of the alimentary canal or its related organs. Many persons have the surface of the tongue more or less furrowed as the normal state.

1. Nervous Diseases.—In this class of diseases the tongue often affords information of much value, and it is here that its sensations and movements are mainly disordered. In many cases of cerebral lesion, one half of the tongue is paralysed in its muscles, so that the organ is unsymmetrical in the shape of its two sides; and deviates when in the mouth to the healthy side, but when protruded towards the paralysed side. In exceptional cases the entire organ is affected, so that it cannot be protruded or even moved. The way in which a patient attempts to put out the tongue, when asked to do so, may be made use of to indicate the state of consciousness in various conditions affecting the brain. In many cases of cerebral disease the tongue shows a marked and speedy tendency to become thickly furred, and very foul. This is well seen in cases of apoplexy due to hæmorrhage in connection with the brain.

The tongue is specially affected in certain peculiar nervous diseases, particularly labio-glossolaryngeal paralysis, many cases of diphtheritic paralysis, general paralysis of the insane, and extreme cases of wasting palsy. Beginning with slight indications of loss of power, as tremulousness, thickness of speech, and difficulty in swallowing, the affection is liable to end in complete paralysis of the organ, which may also involve its sensibility. These results depend on disease involving the roots of the nerves supplying the tongue. The organ may also be thus affected in various degrees, owing to some morbid condition implicating its nerves in their course or at their origin. When completely paralysed, the tongue in time comes to present the appearance of a sodden mass lying in the mouth. In cases of severe neuralgia of one side of the face, the tongue occasionally presents peculiar appearances, such as unilateral furring, thickening of the

mucous membrane, or enlargement of the papillæ; coating of one side of the tongue has also been noticed in connection with painful teeth (Hilton). The tremulous and foul tongue of acute or chronic alcoholism may be mentioned under this head. Dickinson has often noticed the tongue to be white and sodden-looking after an exacerbation of nervousness, but attributes this to a want of saliva. Signs of the organ having been bitten may be useful in the diagnosis of obscure cases of epilepsy. Jerking and irregular movements of the tongue are very striking in many cases of chorea. During attacks of megrim its surface usually becomes much furred. *See also* PAIN IN VISCERAL DISEASE.

2. General Conditions and Diseases.—The tongue is usually markedly altered in the febrile state, whether associated with specific fevers, or with inflammatory or other diseases. It becomes covered with more or less fur, often of considerable thickness, and usually either white or yellowish-white. In particular fevers the organ commonly presents peculiar characters. Thus, in many cases of typhoid fever it is small and irritable, with enlarged papillæ, and a thin whitish or yellowish fur; it may become red, smooth, and glazed or shining. In this disease it may also be peculiarly tremulous, which has been regarded as a bad sign, indicating deep ulceration of the intestine. In scarlatina the papillæ tend to become peculiarly prominent as well as injected, projecting through the fur, the tongue presenting the so-called 'strawberry' appearance; this condition may, however, be met with in other diseases. In diphtheria the tongue may exhibit a diphtheritic deposit upon its surface. There is generally a very thick creamy coating in acute rheumatism, as well as before and during attacks of gout, and in the latter disease especially it often becomes yellowish or brownish. In acute pneumonia also the tongue is often thickly plastered, but presents much variety. In the 'typhoid state,' whatever this condition may be associated with, the tongue tends to be dry, and usually covered with a brown or even blackish crust, looking as if it were baked. It is often very red and irritable in itself, and occasionally the typhoid tongue has little or nothing on its surface, but is very dry, deeply red, like raw beef, and fissured. The organ may be in such a condition that it can scarcely be moved at all. At the same time sordes are present on the teeth and gums. The red, raw, and dry tongue is generally associated with exhaustion, especially when this is due to exhausting discharges, particularly of pus; and it is linked with the constitutional state described as hectic. It not uncommonly occurs, however, in various conditions producing prostration and depression, apart from discharges, and these are often connected with the abdominal organs. In malarial fevers the margins of the tongue are said to present sometimes a faint bluish tinge.

It may be remarked here that the tongue not uncommonly affords evidence of constitutional syphilis, owing to the past effects of this disease upon the organ. It may also indicate the general tone of the system, being often large and flabby when this is below par.

3. Conditions affecting the Blood and Circulation.—The tongue often exhibits abnormal characters, due to the state of the blood or circulation. Thus, in anemia it is more or less pale; generally large, flat, broad, and flabby; and

frequently indented at the sides by the teeth. In the plethoric condition it is also large, but tends to be of a deeper colour than normal, and may present a venous tint. The anæmic tongue is frequently quite clean, though this will depend much on its associations; but the tongue met with in plethora is generally furred. A temporarily congested condition of the digestive organs and liver is supposed to be accompanied by a corresponding plethoric state of the tongue. Any cause of general obstruction to the venous circulation, or of interference with the due aëration of the blood, whether seated in the heart or lungs, is likely to give rise to enlargement of the tongue, and to make it assume a congested, or even a cyanotic appearance in marked cases, such as those of congenital malformation of the heart. It occasionally happens that some local obstruction, affecting the large veins in the thorax, as from the pressure of an aneurysm, produces a similar condition of the tongue. In his classification, Dickinson applies the term *cyanotic* to a particular variety of tongue, which is injected, hypernucleated, with excess of deep epithelium.

4. Affections of the Alimentary Canal and its Related Organs.—The tongue is commonly regarded as of peculiar importance in relation to morbid conditions of the digestive apparatus and liver, but Dickinson's observations do not support this idea, and he attributes the changes presented by the organ rather to the pyrexia or other constitutional disturbance associated with these conditions. As already stated, there is no definite relation between the state of the tongue and the performance of the digestive functions, or the presence or absence of even serious disease, but at the same time there are certain general indications which may be worthy of note.

(a) The tongue is peculiarly liable to be altered in local affections of the mouth and throat. In catarrh of these parts it is more or less coated. In tonsillitis there is usually a very thick covering; and it may be most marked on the side of the inflamed tonsil. Even a local irritation, such as that caused by decayed teeth, may originate a fur, and it is under such circumstances that it is liable to be localised. Associated stomatitis may explain the state of the tongue in some cases of gastric catarrh or inflammation.

(b) In any acute disorder of the alimentary canal the tongue tends speedily to become coated, usually either white or yellowish-white, but it may be more or less brown. This is seen in so-called acute dyspepsia, gastric catarrh, and hepatic disorders. The fur often clears away very speedily when the cause subsides. In severe acute gastritis the organ frequently presents a strikingly red and irritable appearance, especially at the tip and edges, with enlarged papillæ, and a tendency to dryness. In acute intestinal obstruction the tongue becomes stippled at an early period, or coated and dry.

(c) In chronic forms of dyspepsia and gastric catarrh, the tongue often presents abnormal appearances. In the atonic variety it is usually large, flat, soft, and flabby; frequently marked by the teeth; and more or less furred, though it may be quite clean. In the irritative form it tends to be small, elongated, and pointed; contracted and firm; red and irritable; with enlarged papillæ; and generally only having a thin white fur, through which these papillæ project, but it may appear unusually clean and raw-looking. The organ may

present characters more or less similar to those just described, in those cases where the food passes rapidly out of the stomach into the duodenum in an undigested state. In the more grave diseases of the stomach, namely, cancer and ulceration, the tongue has no special characters, and indeed is often very healthy-looking. Dickinson has not been able to discover any state of tongue especially connected with dyspepsia or ulcer of the stomach.

(d) The relation of the tongue to affections of the intestines is indefinite. The bowels are often much deranged in their functions, or diseased, without any abnormal appearances being exhibited by the organ. It may be said, however, that constipation, especially if habitual, tends to make the tongue large and furred, particularly if associated with portal congestion and deficiency of bile. Cases of prolonged constipation have not uncommonly been met with, however, in which it has remained perfectly clean and normal. Chronic accumulation of feces in the rectum seems to give rise to thick coating, which Dickinson attributes to general disturbance, and probably pyrexia connected with morbid absorption. Diarrhœa tends speedily to affect the tongue in various ways. There is scarcely any condition in which the tongue becomes more rapidly dry, coated, furred, and encrusted, than in severe diarrhœa. In dysentery, especially with hepatic abscess, it often becomes shrunken, red, smooth and polished, or fissured.

(e) As regards the hepatic apparatus, portal obstruction is believed to cause an enlarged and congested state of the tongue. It tends to become more or less coated in biliary disorders, especially when acute; and is often very foul in cases of obstructive jaundice, when it may also be coloured yellow.

5. Special Diseases.—In diabetes mellitus the tongue is often peculiarly irritable, red, clean, cracked, and dry. In acute peritonitis it is usually remarkably small and contracted, also red and irritable, with but little fur, and tending to dryness. In advanced cases of phthisis, especially with a high temperature, it frequently becomes red and raw, and exhibits enlarged papillæ; the occurrence of thrush upon its surface may also be a sign of approaching dissolution in this disease. These illustrations will suffice to point out the special information which the tongue may afford as regards particular diseases. FREDERICK T. ROBERTS.

TONGUE, Diseases of.—SYNON.: Fr. *Maladies de la Langue*; Ger. *Krankheiten der Zunge*.

The principal morbid conditions affecting the tongue may be thus enumerated in alphabetical order: (1) Chronic Abscess; (2) Adhesions; (3) Atrophy; (4) Cancer; (5) Cysts (mucous); (6) Hypertrophy; (7) Inflammation and Acute Abscess; (8) Leucoma, Leukoplakia; (9) Parasitic Affections; (10) Syphilis; (11) Tongue-tie; (12) Tubercular Ulcer; (13) Tumours; and (14) Ulceration. Aphthæ and thrush are discussed under their respective headings (see APHTHÆ; and MOUTH, Diseases of). The simpler disturbances of the surface of the tongue, of which the practitioner avails himself as an aid to diagnosis, are separately discussed. See TONGUE, The.

1. Chronic Abscess.—This may occur at any age, and its cause is generally obscure. It is most common in front of the circumvallate papillæ, and forms a tense, elastic, more or less prominent swelling, presenting beneath the dorsal mucosa. Its

outline is generally quite clear. The mucous membrane over it is free and normal in appearance; the swelling is neither tender nor painful. However prominent, it is not translucent. Growth is very slow, and the history of the swelling may extend over some years. The disease has to be diagnosed chiefly from mucous cyst.

The treatment consists in the application of cocaine, incision, and scraping out the abscess with a sharp spoon.

2. **Adhesions.**—Occasionally the tongue is attached more or less extensively at its sides and under-surface to the corresponding surfaces of the mouth. These adhesions may be congenital, but more frequently they are the result of ulceration or sloughing.

TREATMENT.—In congenital cases the membranous bands may be divided with scissors or scalpel. Sometimes a well-formed tongue exists, bound down laterally to the jaws: these cases do well. When the tongue is not really formed, treatment is of no avail. Adhesions consequent upon ulceration may be dealt with similarly, but the result is much less hopeful, unless a mucous flap, from a part which will not lie in contact with another raw surface, can be twisted into the wound made. The writer is not acquainted with any case in which the transplantation into the mouth of a piece of mucous membrane from another animal has succeeded.

3. **Atrophy.**—Atrophy of the tongue may be either unilateral or bilateral. It may result from either acute softenings or chronic sclerosis of the nuclei of the twelfth nerve in the medulla, and in both of these cases the palsy is usually bilateral, the two nuclei being so close together as to be almost necessarily involved by even a small lesion. Injuries to the nerve-tract between the centre at the lower end of the ascending frontal convolution and the nucleus cause paralysis, with but little and late wasting. Injuries below the nuclei, on the other hand, lead to rapid wasting of the tongue on the side of the injury.

TREATMENT.—Treatment must be directed to the cause of the complaint. See HYPOGLOSSAL NERVE, Diseases of.

4. **Cancer.**—**ÆTIOLOGY.**—Cancer of the tongue is most frequent between the ages of forty and forty-five; it may occur at any age after twenty-five, but is rare under thirty-five. It is more than twice as common in men as in women. Irritation from a tooth or hot smoke sometimes seems to be the exciting cause; again, the growth may supervene upon leukoplakia, glazed red tongue, a wart, or a chronic gummatous ulcer. Cancer of the tongue is much more prevalent in some districts than in others.

DESCRIPTION.—Squamous epithelioma is the only form met with in the tongue. Its usual seat is the edge in the middle third, but it may begin at any spot. It starts as an ulcer, a fissure, or a wart—sometimes the patient says, ‘as a lump in the tongue’; but growing as it does from the epithelium, this ‘lump’ is always intimately connected with the mucosa, and the epithelium is never normal over the whole mass, but is either destroyed by ulceration or is papillary. The earliest characteristic point is induration of the base of the lesion, from invasion of the mucosa by epithelial cells and round-celled infiltration about them; the edges of an ulcer or fissure become similarly thickened. The

epithelium on the surface usually undergoes degeneration and is cast off, thus leading to the formation of an ‘ulcer,’ at the base and edges of which the tissues are being progressively invaded by growing epithelial columns. This invasion is sometimes more marked along the surface, at other times it takes place chiefly towards the substance of the tongue, and may involve almost the whole organ. It is sometimes slow, again rapid, and is almost always so much more rapid than the ‘ulceration,’ mentioned above, that induration round the ulcer is well marked. Rarely papillary masses project from the surface of an epithelioma, destruction being reduced to a minimum; unfortunately, because often delaying a correct diagnosis, it is in these cases that infiltration of the base also is apt to be least marked, the products of epithelial multiplication escaping towards the surface instead of being pressed into the substance of the part. As the ‘ulceration’ and infiltration extend into the substance of the tongue, to the floor of the mouth, the jaw, the fauces or soft palate, the movements of the tongue become more limited, till ultimately it may be quite bound down. Sooner or later the submaxillary lymphatic glands, or those at the base of the carotid triangle, swell and become painful. They may be few or many, may remain of small or moderate size, or may grow rapidly and discharge or fungate through the skin, and this with but a small growth in the mouth. Extension to distant organs by the blood-path is rare. Pain may be slight or even absent, but, as a rule, it is severe, radiating over the whole distribution of the fifth nerve and often to the region of the small occipital. The tongue is tender and sensitive; when fixed, it fails to push food between the teeth for mastication. Ptyalism is constant, and may be very marked. The saliva is ropy, and is expelled with difficulty. The ulcer discharges into the mouth, while sloughs form upon its surface and putrefy; hence mouth and breath become offensive. Perhaps in relation with the increased secretion of ropy saliva the submaxillary salivary gland is large, firm, and easily felt. In consequence of inability to take sufficient food, of chronic putrid poisoning, of pain and anxiety and other influences connected with the nature of the growth, the patient loses health and strength steadily. Death may result from simple exhaustion; not uncommonly septic bronchitis and broncho-pneumonia hasten the end. Dangerous or fatal hæmorrhage from the epithelioma is rare. Cancer of the tongue is one of the most painful and most rapidly fatal forms of malignant disease. According to Clarke, its average duration is fifty-seven weeks. But the statistics of surgeons practising carefully the modern operation of removal of the growth, together with the nearest lymphatic glands, show a better result than this—including a fair number of cases of prolonged immunity. Cancer of the frænum and its neighbourhood should be mentioned, as it is credited with exceptional malignancy. This may be the consequence of imperfect treatment, due to a surgeon's reluctance to draw healthy front teeth (to get a good view), to resect the symphysis, and to remove the submental and submaxillary lymphatics on both sides. Cancer behind the circumvallate papillæ is, fortunately, rare. It may not be visible without a laryngoscope or palpable unless both the fore- and index-fingers are fully introduced through the mouth.

DIAGNOSIS.—The difficulties in recognising a cancer of the tongue vary according as it presents itself under the form of an ulcer, a wart, or a tumour beneath the mucosa. In all cases the diagnosis should be effected before the local disease is extensive, or glands are enlarged, or it will probably be too late for successful treatment. A cancerous *ulcer* must be distinguished from a simple ulcer, a hard chancre, a broken-down gumma, and a tubercular ulcer. A simple ulcer generally forms quickly, and bears an obvious relation to its cause—some injury, a sharp tooth, or a projecting part of a tooth-plate. It is less defined in outline than cancer, and induration is absent or slight; the edges are not thick, rounded, raised, or everted; the base is not warty and irregular. A scraping of its surface after cleansing shows but few epithelial cells, and they are of normal appearance. After removal of the irritant, and under suitable treatment, a simple ulcer should heal. Hard chancre affects the apex; is exceedingly rare; of rapid formation up to a certain size; very hard, defined, with smooth edges, and but slightly indurated base. The submaxillary glands very soon enlarge, and general symptoms follow. A gummatous ulcer is usually a deep ragged cleft on the dorsum, parallel to the raphé, with thin, irregular margins, undermined and gaping as the tongue is protruded, not at all indurated; in the cavity yellowish sloughs may still adhere; there are sometimes other masses in the tongue-substance, and the surface frequently presents evidence of previous ulceration or of superficial glossitis; a history and other signs of syphilis are perhaps obtainable; a scraping of the side of the ulcer after cleansing shows but few epithelial cells; appropriate treatment is successful. Both the simple and the gummatous ulcer may become cancerous; so, if progressive induration leads to the suspicion of cancer, either the ulcer should be at once freely excised, or a good piece should be cut out under cocaine and examined microscopically. A tubercular ulcer in a male over thirty-five may be indistinguishable from cancer. If the ulcer is secondary, the presence of tubercle elsewhere should make us careful. Primary tubercular ulcers of the tongue are rare. They are less indurated, but may, if clean, present pale pinkish granulations not unlike those of a clean epithelioma. Sometimes in deeper specimens muscular fibres may be recognised, laid bare by the progressing ulceration. The effect of medicinal treatment is usually *nil*. It is stated that these ulcers yield a scraping like that of a simple or syphilitic sore. Fortunately, the diagnosis is of less importance here than in the previous cases, for the primary tubercular ulcer, or a secondary ulcer with but slight lesions elsewhere, should be excised.

A *warty* epithelioma resembles a simple wart. The diagnostic point is induration at the base of the epithelioma. No wise man would keep even an undoubted wart on his tongue.

An epithelioma in form of a submucous *tumour* has to be distinguished chiefly from an unbroken gumma. A normal or smooth red epithelial surface over the mass, absence of definite outline, of marked induration, and of enlarged glands, the presence of syphilitic lesions on the tongue or elsewhere, and a history of syphilis, excite suspicion as to the nature of the growth. Full doses of iodide of potassium confirm it.

So much stress has been laid upon the importance

of induration in the diagnosis of cancer, that it is right to add that rapidly ulcerating epitheliomata rarely do occur in which there is but little induration.

In no case should the nature of a small ulcer of the tongue in a patient of thirty-five or over be allowed long to remain doubtful. If it does not heal after removal of any local irritant, upon the application of local antiseptics, especially borax and iodoform, and upon the administration of iodide of potassium, a small ulcer should be excised—the operation is so trifling. If there is any doubt about the nature of a larger mass, a portion should be excised for examination.

TREATMENT.—All epitheliomata, in which it appears possible to remove the whole of the disease, local and glandular, by an operation which the patient seems likely to survive, should be removed without delay by knife or scissors. In all cases, even the earliest, the lymphatic glands supplied from the seat of disease, and, as far as possible, also the tissue between them and the primary lesion, should be removed. The mortality after operation upon cases of all degrees of severity is about 12 per cent., rising from *nil* in slight, early cases to 25 per cent. in extensive cases. When there is a fair prospect of preventing local recurrence, it is still often right to remove that part which lies in the mouth, to relieve pain and the suffering which a cleanly patient undergoes on account of the foul state of the part. Excision of half an inch of the lingual nerve has been performed to relieve pain, and ligation of a lingual artery to check hæmorrhage. The treatment of incurable cases consists in the administration of anodynes in doses *sufficient to give relief*; in the use of antiseptic washes and powders for the mouth; and in careful feeding.

5. **Cysts.**—In addition to the rare lingual dermoids described under MOUTH, Diseases of: 4, and the parasitic cysts (*Cysticercus cellulosa*, extremely rare; and *echinococcus*, rare), cysts from dilatation of a mucous gland are sometimes met with, and at any age. They are most common where the mucous glands are largest and most numerous—behind the circumvallate papillæ, but they may occur elsewhere in the tongue. They form tense, elastic, circumscribed swellings, sometimes pushing their way into the tongue, at other times projecting sufficiently from the surface to render it evident that they are translucent. They cause no pain, and the mucosa over them is normal or simply stretched.

DIAGNOSIS.—When a cyst is prominent, the diagnosis presents no difficulty except from chronic abscess, which generally occurs in the anterior part of the tongue, and is never translucent. A puncture will decide. When it lies deep and far back, it may be difficult to be sure even that the mass is fluid; the situation, elasticity, and smoothness of surface of the swelling will always suggest a mucous cyst, and here, again, a puncture will prove its nature.

TREATMENT.—This consists in application of cocaine to the surface, and excision of part of the wall. The interior should be touched over with pure carbolic acid.

6. **Hypertrophy.**—**SYNON.** : *Macroglossia*.—Hypertrophy of the tongue is generally congenital; sometimes it follows an attack of inflammation. The enlargement seems to be due to blocking of the lymph-paths; the lips are sometimes similarly

affected (*macrocheilia*). In the congenital cases it is often complicated with imbecility or idiocy. The enlargement of the tongue may not be evident at birth. At first the enlarged organ has a normal appearance; but usually it becomes too large for the mouth, and part is constantly or frequently prolapsed. Gradually, from exposure to the air and constant irritation, the mucosa becomes dry, thick, and callous, or covered with a slimy secretion; the papillæ enlarge greatly. After a time, the pressure of the teeth acts as a line of constriction, and the protruding portion swells from mechanical congestion. The surface of the tongue becomes bluish or brown; the mucosa roughened and cracked; there is a tendency to ulceration and hæmorrhage; the muscles become palsied, and are unable to retract the organ. As time goes on, the lower jaw is pressed down by the superincumbent weight, and occasionally this goes so far as to produce dislocation. The teeth project forwards, the lips are everted, and there is a constant flow of saliva from the mouth. Altogether, the patient's appearance is most unsightly, and his condition very distressing; for with such a tongue mastication and deglutition are difficult, and speech is thick and indistinct.

TREATMENT.—The redundant part of the tongue must be removed by a V-incision, and the wound sutured.

7. Acute Inflammation.—**SYNON.**: *Glossitis*.—Sometimes the glossitis is *superficial*, only the mucosa being affected: again it is deep or *interstitial*.

ÆTIOLOGY.—Fifty years ago by far the most common cause of acute interstitial glossitis was the excessive use of mercury, happily now seldom seen. Sometimes it is due to septic wounds, to wasp-stings, or to taking putrid, corrosive, or acrid substances into the mouth; it may arise in the course of the acute specific diseases; usually no cause is discoverable.

SYMPTOMS.—Simple superficial glossitis needs no description: it occurs chiefly in young children. Sometimes vesicles form in patches—a kind of herpes. The earliest symptoms of mercurial glossitis, which may be taken as typical, are a foul metallic taste, peculiarly offensive breath, and a red line along the gums, at their junction with the teeth. The gums are tender, spongy, and apt to bleed. As the case advances, the gums, the tongue, and the inside of the lips and cheeks become much swollen. The tongue is sometimes so large as to protrude constantly from the mouth. At its edges it becomes deeply marked by the teeth, and it and the cheeks and gums often ulcerate. The flow of saliva is incessant; the salivary glands are swollen and painful; the teeth ache and become loose, but seldom drop out. The pulse and respiration are hurried. There is great thirst, but the patient has difficulty in swallowing; and he is wholly unable to speak. As a rule, this state of things subsides under proper treatment, but occasionally pus forms in the substance of the tongue, or it may become ulcerated or even gangrenous.

In the acute spontaneous form, the swelling and hardening of the tongue are greater than in the mercurial, and all the symptoms are more severe; the mercurial fetor is absent. There is a greater tendency to suppuration, localised or diffuse, and to sloughing of more or less of the organ. The

fever is often marked. Sometimes the swelling is chiefly at the base of the tongue, though the anterior less swollen part is pushed out of the mouth. General distress and difficulty in breathing are great. Abscess is probable in these cases.

TREATMENT.—In acute inflammation of the substance of the tongue, the first point is to discover any abscess which may be present, by special swelling, tenderness, and localised softening. The abscess is usually far back and hard to find. If found, immediate relief follows incision. If none be found, a sharp purge should be given; and either ice may be freely sucked or the mouth be frequently washed out with hot water—whichever is found to give most relief. Cold or heat may be applied to the submaxillary region. Two long cuts through the mucosa of the dorsum on each side of the raphe may give relief.

8. Leucoma.—**SYNON.**: *Leukoplakia Lingualis et Buccalis*; *Ichthyosis Lingue*; *Hairy Tongue*; *Smooth Red Tongue*; *Tylosis*.

DEFINITION.—These names have been applied to the results of a chronic superficial inflammation (probably) of the mucosa, resulting now in a white patch or patches; now in hypertrophy of the papillæ, giving the tongue a warty or hairy appearance; again in a swollen, smooth red surface.

ÆTIOLOGY.—Nothing is certainly known on this point. Mechanical irritation, hot spices and food, ardent spirits, smoking, syphilis, gout, and various forms of dyspepsia are all regarded as causes of this condition. It seems to run in families. It is rare among women, and among men under twenty. It does not begin after sixty. It occurs on the lingual mucosa chiefly; to a much less extent on the buccal. It is said not to occur behind the circumvallate papillæ.

DESCRIPTION.—The commonest form is that known as *leukoplakia*. Blue or yellowish-white patches (leucomata) form, oval or irregular in outline, smooth on the surface, and showing some degree of resistance when pinched between the fingers. There may be but one such patch. Usually there are no symptoms; sometimes the patches are irritable, casting their epithelial scales, and prone to form fissures and ulcers. With irritability there is more or less *ptyalism*. A much rarer form is that known as *ichthyosis*, in which the papillæ are hypertrophied and covered with horny epithelium. When the filiform papillæ have been specially hypertrophied, the name *hairy tongue* has been given. Lastly, the tongue in some cases, from rapid shedding of epithelium, becomes smooth, red, and slightly swollen, very sensitive and irritable, and liable to fissures or ulcers.

PROGNOSIS.—Once the disease is established, treatment avails but little as a rule. The danger of the trouble is that it tends in all forms to pass on into epithelioma; induration at the base, ulceration, or warty outgrowth being the signs to be watched for.

TREATMENT.—Every source of irritation must be removed, and any constitutional defect treated, in the hope that good may come. Borax-and-glycerine lotion, solution of bicarbonate of sodium (gr. xx ad ʒj), or chromic acid (gr. i-ij ad ʒj), may be used as a mouth-wash after meals. Honey of borax, bichloride of mercury (gr. ij-iv ad ʒj), chromic acid (gr. v-x ad ʒj), or salicylic acid (a saturated watery solution) may be applied directly over the dried part. On no account should irritant or caustic substances be used.

A single obstinate patch may be excised under cocaine. A developing epithelioma will require prompt excision.

9. **Parasitic Affections.**—The most important parasite connected with the tongue is the *Oidium albicans*, which is present in thrush (see MOUTH, Diseases of). *Hydatid cysts* are occasionally met with; and so is the *Cysticercus cellulose*. Among the nematodes the Guinea worm (*Dracunculus* or *Filaria medinensis*) and the *Trichina spiralis* have been found. The dracunculus may give rise to an abscess, which will require to be opened; and hydatid cysts may have to be incised and removed.

10. **Syphilitic Affections.**—These form five-ninths of all the lesions of the tongue which come under our notice. Primary sores are occasionally seen in this situation, but their occurrence is so rare that we need only mention them. For the sake of clearness, it is well to arrange these syphilitic affections into four classes: (A) *Superficial Ulcerations*; (B) *Mucous Tubercles*; (C) *Gummata and Deep Infiltrations*; and (D) *Chronic Morbid States of the Mucous Membrane*.

A. **Superficial Ulcerations.**—Slight superficial ulcerations of the tongue are very common in what is called the secondary stage of syphilis. They are usually situated on the sides, tip, and under-surface of the free portion of the organ; and are often associated with similar ulcerations upon the inside of the cheeks, the lips, and the angles of the mouth. They begin in small inflamed spots, and spread into linear cracks and fissures (rhagades). These are exquisitely sensitive; and, as it is very difficult to keep the tongue at rest, are a source of constant suffering. When these ulcerations heal, whitish scars and cicatrices are left, which are very persistent, and resemble leukoplakial patches.

B. **Mucous Tubercles.**—Mucous tubercles, when they occur on the tongue, are generally met with about the sides and under-surface of the organ; on the fold of mucosa that is reflected to the floor of the mouth; or on one side of the raphé far back. They occur in the early years of the disease.

C. **Gummata and Deep Infiltrations.**—Gummata are common in the tongue, varying in size, when they come under notice, from a pea to a marble. Not uncommonly they are multiple, or the principal mass may be set in a diffuse infiltration of the tongue. As a gumma enlarges and softens, the mucosa over it becomes red and smooth, and ultimately it gives way. A ragged opening is thus formed, through which fluid escapes, and adherent slough protrudes. The edges are irregular, thin and flexible; the walls are not indurated; the opening gapes in certain positions of the tongue. The cavity may heal, or may remain for an indefinite time if no treatment is adopted, and some induration of the walls may supervene. It is in these chronic cases that downgrowth of epithelium (epithelioma) sometimes takes place. So long as a gumma is unbroken, there is strong hope that treatment will cause its absorption. After this, no trace of it may be left upon the surface; but when a gumma has burst, a fissure-like scar results from its healing, and there is more or less induration about it. Sometimes, though rarely, a tongue becomes generally fibroid from the development of gumma-tissue throughout (*chronic interstitial glossitis*). A tongue may thus become deeply and irregularly scarred, enlarged or diminished in size,

or much deformed, by alternating contraction and swelling; while the mucosa presents, perhaps, patches of normal appearance, white or yellow scars with thickened epidermis, red areas where irritation is still active, and bluish surfaces indicating mechanical congestion from scar-contraction.

D. **Chronic Syphilitic Disease.**—Various morbid conditions of the mucosa of the tongue are often seen in association with the later stages of syphilis. Sometimes circumscribed patches of the epithelium become dead-white, and drop off, leaving a red raw surface beneath. The epithelium is speedily restored, but another patch becomes affected in a similar manner, and so the disease continues—one patch healing and another desquamating. This is the proper *psoriasis lingue*. Again, there is a much more extensive disease, to which the name *chronic superficial glossitis* has been given. At the commencement some portions of the membrane present their natural appearance, while others are of a deep red colour and raw-looking. These patches are often oval or oblong. Their surface is smooth and glossy. They are either entirely denuded of epithelium, or this is reduced to an extremely thin layer, and the papillæ are obliterated by distension. These patches are slightly elevated and hard to the touch, in consequence of interstitial thickening. The tongue is swollen. At its edges it takes the impression of the teeth, and the lines thus produced are prone to ulcerate. Sometimes the whole organ has a bluish, congested hue. The mucous secretion all over the affected part is viscid and glairy, giving the organ a peculiar, smooth, glazed appearance; and sometimes the patient's breath is so fetid that he is offensive to himself and to all about him. He complains of thirst; his mouth is parched, especially at night; and when he wakes in the morning his tongue feels dry and chipped. The disease is, in fact, a chronic superficial glossitis. If the more active mischief is checked, the swelling subsides, but the mucous membrane never resumes its healthy character. The patches that have been affected remain smooth and shining. The papillary structure has been impaired, and what is left is in fact cicatricial tissue, a tissue which is sensitive to the contact of hot, acid, or pungent substances, and apt to inflame easily. At a later date, portions of this cicatricial membrane become more completely fibrous, presenting a whitish appearance, and being callous to the touch.

TREATMENT.—In the treatment of all of these affections it is important to look to the general health of the patient; to see that he smokes little or not at all—any smoke reaching the mouth should be cool; to forbid alcohol, or allow it only in dilute forms; and to order a simple, nourishing diet, from which hot spices and condiments should be excluded.

Mucous tubercles and small ulcers of the secondary stage require a mercurial course. Drying the lesions and painting them once or twice daily with a solution of the bichloride of mercury (grs. ij—viij ad ʒj) is helpful.

Gummata and gummatus infiltrations disappear most quickly under iodide of potassium. With this, if the gummata are appearing early, or if the patient has never been satisfactorily treated with mercury, or, again, if the iodide does not act quickly, a little mercury should be given. Broken gummata should be treated with a borax-wash

used frequently, and dusted twice daily, after food and subsequent washing of the mouth, with a powder composed of grey powder, iodoform, and starch in equal parts.

The treatment of chronic superficial glossitis is far from satisfactory. The first object should be to improve the digestion, and to regulate the general health. When there is much superficial soreness, a mouth-wash of borax and glycerine, bismuth and glycerine, or chlorate of potassium, should be ordered. The patches may be painted with a non-irritating mercurial lotion. Irritants should never be used, in view of the danger that these cases may develop epithelioma. The treatment too often consists in combating soreness and irritation, and in watching for induration, indicating the down-growth of epithelium. Free operation should follow at once upon the discovery of this.

11. Tongue-tie.—The tongue is said to be 'tied' when the frænum is either too short or comes farther forward than it should, and thus restrains the movements of the tip, which often seems slightly bifid. The infant cannot put out its tongue, or use it in sucking; and, if allowed to remain, the defect interferes with speech.

TREATMENT.—Operative interference is required, as follows: Place the child with its head on the nurse's knees, towards the surgeon; raise the tip of the tongue and stretch the frænum with the left forefinger, and snip through the thin part of the frænum with scissors, which will, naturally, be pointed downwards, parallel to the ranine veins. A little pressure down and back into the wound with the left forefinger will still further free the tongue. Mothers often suppose that their children are tongue-tied when, in truth, they are only backward in speaking; so the surgeon should form his own opinion before operating.

12. Tubercular Ulceration.—This may be either primary or secondary; the former is very rare. The latter is secondary to laryngeal or pulmonary tuberculosis, suggesting the possibility of auto-inoculation.

DESCRIPTION.—Tubercular ulcers of the tongue are much more common in men than in women. They occur at all ages, are not uncommonly multiple, and rarely reach a large size (one inch square) or a great depth (quarter of an inch). They are most frequent upon the tip and edge adjacent, then upon the dorsum, and then underneath the free part. They begin as small nodules in the mucosa, which ulcerate, exposing a greyish or yellowish slough. Cheesy stuff may be seen on the surface. If the extension is slow, the edge becomes somewhat raised, red and rounded, the floor is formed of pale granulations, and the surrounding parts are little or not at all cedematous. There may be enough induration to raise a suspicion of cancer. When the spread is rapid, the edge is thin and ragged, often undermined; the base may be sloughy, or the structure of the eroded tissue may be clear; there is no induration. The symptoms are soreness, sensitiveness, and more or less ptialism; when advanced, the ulcers may be very painful. The lymphatic glands may enlarge, but this is by no means constant. Microscopically, they present all the characteristics of a tubercular ulcer.

DIAGNOSIS.—Many tubercular ulcers have been excised as malignant—a mistake of little consequence. Gummatus ulcers are distinguished chiefly

by a history of syphilis, situation on the dorsum, depth and thin ragged edges, absence of tubercle-bacilli in the discharge, and the effects of treatment.

PROGNOSIS.—A small tubercular ulcer may heal, but only to break out again. After suffering much from the ulcer, a patient usually dies from disease of the larynx or lungs.

TREATMENT.—If the ulcer is primary, or if the primary disease of lung or larynx is early, the ulcer should be excised under cocaine, and the sides of the wedge-cut sewn up. If there is advanced disease of lung or larynx, and the ulcer is not painful, iodoform and starch in equal parts may be applied after food. If there is much suffering, excision may be done, or phenol freely applied.

13. Tumours.—The mucous membrane of the tongue is occasionally the seat of simple warts; and fatty, fibrous, cartilaginous, bony, and nævoid growths have more or less rarely been removed from this situation. They are sometimes deep in the tongue-substance, or more or less polypoid. The lymphoid tissue beneath the mucosa in front of the epiglottis may hypertrophy and form a small protuberance on one or both sides of the mid-line, giving rise to little or no trouble. A few instances have been recorded (all in women) of simple tumours having the structure of the thyroid, growing in the base of the tongue and probably connected with the thyroglossal duct. Sarcoma is very rare; cancer is the tumour of the tongue.

TREATMENT.—Small warts may be dried and touched with 30-per-cent. solution of chromic acid till they shrivel and fall off; or they may be snipped off, together with the mucosa whence they spring. Larger masses require excision. The other tumours mentioned, if troublesome, all require excision. Eucaïne anaesthesia is often sufficient (*see* p. 59). Removal of a part of thyroid growths has been followed first by increase, then by wasting of the remainder.

14. Ulceration.—Ulcers of the tongue are best classified according to their causes, so far as we know them. We thus get the following groups: *simple, tubercular, syphilitic, and cancerous.* Under the heading 'simple' we include *traumatic, dyspeptic, and aphthous* ulcers, and the sores on the tongue which form in non-specific leukoplakia, or in ulcerative and mercurial stomatitis. Only the traumatic and dyspeptic ulcers remain undescribed; for the other varieties reference may be made to the special sections.

Traumatic ulcers may be due to injuries of all kinds, but the injury caused by teeth often leads to an ulcer difficult to recognise. A sharp tooth or tooth-plate, though constantly present, does not irritate equally at all times, but more (it is supposed) in dyspeptic or depressed states. The ulcer at first has the characteristics of a small spreading ulcer—sloughy or bright red base, sharp-cut low margins, more or less cedematous surroundings. It is irritable and painful. After weeks or months, the base becomes paler, the margins thicker and rounder, and more or less indefinite induration of the base appears. Naturally, the seat of these ulcers is almost invariably the edge or tip. In their diagnosis, the relation to an irritant, the absence of a history of syphilis or tuberculosis, the absence of cancerous induration, and the effect of removal of the cause and subsequent treatment, are the chief points.

Dyspeptic ulcers are apt to occur in ill-fed children, and also in adults who habitually eat and drink

too freely. Such ulcers are generally situated upon the sides or upper surface near the tip; but not infrequently they are on the frenum. They are encircled by an inflamed margin; shallow; their bases being flat and covered with a greyish slough. They are very sensitive to the touch, and painful when the organ is moved. Sometimes there is offensive discharge, with a good deal of swelling of the sublingual and submaxillary glands.

TREATMENT.—Defects of digestion must be attended to; a cholagogue purge is often a good beginning. The mouth should be kept scrupulously clean, and all sources of mechanical irritation removed. Honey of borax should be used frequently; and the ulcers dried and painted with chromic-acid solution (grs. v-x to ʒj) every two or three days.

STANLEY BOYD.

TONIC (τόνος, tension). — A distinctive term used in reference to the nature of spasms, which are usually divided into two classes, namely, *tonic spasms* and *clonic spasms*; the former being those in which the muscles concerned remain in a state of continuous rather than in one of intermittent contraction or clonic spasm. See SPASM.

TONICS (τόνος, tension, tone). — **SYNON.**: Fr. *Toniques*; Ger. *Tonische Mittel*.

DEFINITION. — Therapeutic agents which increase the strength of the body or its parts.

ENUMERATION. — Among the most typical medicinal tonics, which impart a feeling of strength, are Iron, Nux Vomica, Quinine, and Vegetable Bitters. As the strength of the body generally depends on the proper action of its various parts, tonics have been subdivided into those which have an especial action on the blood-circulation, digestion, and nervous system.

1. **Blood-Tonics.**—Cod-liver oil and other fats, and iron and its salts, are the most important of this group of tonic remedies. Perhaps also phosphate and hypophosphite of calcium, salts of potassium and sodium, arsenic, and phosphorus should be included. Light, fresh air, good food, bathing, and exercise are valuable adjuncts.

2. **Cardio-vascular Tonics.**—The principal vascular tonics are Nux Vomica and Strychnine, Digitalis, Strophanthus, Erythrophloeum, Squill, and Caffeine. The local application of warmth and cold, friction, and massage increase the effect of these medicines.

3. **Gastric Tonics.**—Small doses of Sulphuric, Nitric, Hydrochloric, and Phosphoric Acids, small doses of Arsenic, Alum, Bismuth, Copper, Silver or Zinc, Aloes, Bitter Beer, Chamomile, Cinchona, Cusparia, Cascarella, Calumba, Hops, Gentian, Orange, and Lemon-Peel, Quassia, Rhubarb, Strychnine, and Vegetable Bitters, generally all impart vigour to the gastric function. Valuable adjuncts are pepsin and hydrochloric acid.

4. **Intestinal Tonics.**—These are chiefly Nux Vomica, Belladonna, Rhubarb, the Mineral Acids and Metallic Salts just mentioned, and astringents.

5. **Nervine Tonics.**—Nux Vomica and Strychnine, Cinchona and its alkaloids, Coca, Phosphorus, Arsenic and its compounds, salts of Iron, Zinc, Copper and Silver, are all included under this head. The tonics which act especially on other parts of the system increase also the power of the nervous system, and act indirectly as nervine tonics.

ACTION.—The derivation of the word 'tonics' indicates the nature of their action. When a person feels limp and weak, and unfit for exertion, tonics restore the energy and strength, and render him again fit for work. The exact mode in which tonics act is not yet perfectly ascertained, but in all probability they increase the functions of the different parts of the body by aiding tissue-change, either by increased nutrition, increased tissue-metabolism, more rapid removal of waste, or possibly by all three taken together. See TONE, Want of.

USES.—Tonics are employed in conditions of debility, either of the body generally or of its different parts, the selection of each depending upon the part of the body affected.

In cases where the malnutrition of the body appears to be dependent on the want of the proper constituents of the blood, as in anæmia, struma, or general debility, without any affection of a particular organ, *blood-tonics*, including iron, cod-liver oil, phosphates, and hypophosphites are employed; and these are also useful where impoverishment of the blood is due to a definite constitutional disease, such as phthisis, or Bright's disease. In pernicious anæmia phosphorus or arsenic may be used. Where enfeeblement of the stomach appears to be present, as shown by loss of appetite and such signs of imperfect digestion as flatulence, weight and pain after eating, *gastric tonics* are used. Should its muscular coat be feeble or inactive, as shown by tendency to dilatation, and splashing of the contents on movement, strychnine is especially indicated, and galvanism or systematic kneading may be also employed. Where the stomach is too debilitated to respond sufficiently to this form of treatment, as after long-continued gastric catarrh, or in old age, its work must be partly done for it, and then such substances as hydrochloric acid and pepsin are useful. When the muscular movements of the intestine are sluggish, as indicated by constipation, and by a tendency to the distension of the bowel with gas, nux vomica and belladonna may be given; and when its mucous membrane appears to be relaxed and flabby, and secreting too profusely, the mineral acids, astringents, and metallic salts may be of much service. When the pulse is soft and feeble, and there is a tendency to vascular dilatation, either general or local, as shown by local congestion and cedema of dependent parts, or by drowsiness in the upright position and sleeplessness in the recumbent posture, *vascular tonics* are serviceable. *Nervine tonics* are used where the nervous functions are imperfectly performed, as shown by dulness, loss of memory, incapacity for work, languor, or tendency to spasm, as in chorea, and also in paralysis. As the functions of this system depend very greatly upon the quality of the blood with which the nervous system is supplied, and on the rapidity of the circulation, the other tonics frequently require to be given in addition to nervine tonics.

In administering tonics care should always be taken to ascertain that the case is suitable, for in very many cases of apparent debility the imperfect functional activity of the body or of its parts does not depend upon insufficient nutrition, but upon imperfect removal of the products of waste. The proper treatment in these cases is not to give tonics, but to remove the waste products by cholagogues, purgatives, and diuretics.

T. LAUDER BRUNTON.

TONSILS, Diseases of.—SYNON. : Fr. *Maladies des Amygdales* ; Ger. *Krankheiten der Mandeln*.

Of recent years increased attention has been paid to the tonsils as the starting-point of general infections. Just as large foreign bodies, such as fish-bones, &c., may become impacted in the tonsil, so pathogenic bacteria, contained in food or drink or taken in with inspired air, may find a lodgment, multiply and excite a local disease, which may be the commencement of a general infection. This is especially likely to happen if the tonsils be unhealthy, e.g. the seat of chronic hyperplasia. Thus the seat of inoculation of such diseases as scarlet fever, rheumatic fever and diphtheria is not improbably the tonsil, and the occurrence of pericarditis and infective endocarditis, immediately following attacks of tonsillitis, has led to the suspicion that the tonsil may afford the means of entry for the infecting virus.

Examination of chronically enlarged tonsils has led to the detection of tubercle-bacilli, and from this site the deep cervical glands may be infected and further progress may lead to infection of the bronchial glands and lungs.

Among the less common diseases of the tonsil are the following :—

1. *Neoplasms*.—Lymphomata, fibromata, papillomata, angiomata, cystic tumours and calculi (carbonate and phosphate of lime). Carcinoma is generally primary. Sarcomata are usually of the small round-celled variety.

2. *Tuberculosis*.—Lupus of the tonsil is generally associated with similar disease of the palate and fauces. Tubercular ulcers, lenticular in shape, with freely granulating sloughy bases, are of somewhat rare occurrence.

3. *Syphilis*.—The tonsil may rarely be the seat of the primary chancre, commonly of mucous patches, or of a gumma.

4. *Actinomycosis*. See p. 22.

5. *Diphtheria*. See p. 398.

6. *Herpes facialis*. See p. 685.

7. *Concretions*. See p. 334.

The acute and chronic inflammatory processes affecting the tonsils are of peculiar interest to the student and practitioner. For purposes of classification they may be divided into two groups, superficial and deep. In the former will be included acute and chronic lacunar (follicular) tonsillitis, membranous tonsillitis, and ulcerative tonsillitis ; in the latter acute parenchymatous tonsillitis and peritonsillitis. Hypertrophy of the tonsils will be described under a separate head.

Acute lacunar tonsillitis.—This and the allied conditions of membranous and ulcerative tonsillitis have to be sharply distinguished from diphtheria, and we would strongly urge the advisability of routine bacteriological examination in all such cases. By this method, and by this method only, can the diagnosis be certainly established.

Our knowledge of the ætiology of superficial tonsillitis has undergone considerable modification of recent years. While it is generally admitted that cold, rheumatism, gout and general debility may be predisposing causes, it is believed that the direct exciting causes of these attacks are micro-organisms, and cultures may reveal the presence of streptococci, staphylococci, *Micrococcus tetragonus*, *pneumococcus*, *Hoffman's bacillus*, *B. coli*, or the fusiform bacillus, either alone or with various associates. An explanation is thus afforded of the undoubtedly

infectious character of tonsillitis, and its causation by drainage-defects, septic surroundings, dust, &c.

SYMPTOMS.—Acute lacunar tonsillitis is marked at its commencement by high temperature (102°–104° F.), pains in the limbs, headache, and the usual accompaniments of fever. The patient complains of the throat being sore, and there is difficulty in swallowing. The tongue is coated and bowels constipated. A puffiness may be present at the angles of the jaw, and the neck is stiff and painful on movement. Inspection shows the tonsils enlarged and red, with the lacunæ plugged with yellowish-white exudation, consisting of dead epithelium, leucocytes, bacteria, &c. The palate and uvula share in the inflammatory process. This condition may persist from three to seven days, leading to a condition of extreme prostration. The enlargement of the tonsils subsides gradually, but occasionally a deep-seated parenchymatous or peritonsillar abscess may form (quinsy).

PATHOLOGY.—An inflammation limited to the crypts and covering mucous membrane.

TREATMENT.—A brisk saline purgative should always be given at once. The patient should be kept in bed so long as the temperature is above normal. A mixture of quinine, tinct. ferri perchlor. and potass. chlorat. used as a gargle and swallowed is very useful. Salicylate of sodium is also a valuable drug, given every three or four hours. *Locally* a gargle or preferably a spray (as gargling is so very painful) of solution of permanganate of potassium, formalin (1 in 250), hydrarg. perchlor. (1 in 4,000), resorcin (10 per cent.) to be used frequently, will remove all septic plugs from the lacunæ in three or four days in the majority of cases. As much non-irritating nutritious food as the patient can take is advisable.

Chronic lacunar tonsillitis.—In this condition the tonsils may or may not be enlarged or inflamed, but the crypts are seen to be distended with caseous material which may have a very offensive odour. There is constant faucial irritability, and often some pain in swallowing, but no febrile disturbance except when, as not infrequently happens, acute attacks of tonsillitis supervene.

TREATMENT.—Occasionally relief may follow the scraping of the crypts and subsequent dressing of the tonsil. Or we may plunge the galvano-cautery point into them. Frequently the best plan is to cut out or punch out the diseased gland.

Membranous tonsillitis.—This condition is seen in the acute exanthemata, such as scarlet fever. It may, however, arise quite independently. The membrane is of a yellowish-white colour, soft, contains much mucus, and is readily detached from the underlying surface which is not ulcerated. It may spread to the palate and larynx. The condition can be differentiated from diphtheria by repeated bacteriological examinations. The organisms usually found in simple membranous tonsillitis are staphylococci, streptococci, the fusiform bacillus, and a few other bacillary forms. The urine may contain a trace of albumen.

Ulcerative tonsillitis.—Two varieties of this condition are met with : (1) small superficial ulcers following coagulative necrosis (drain-throat) ; (2) larger single or double ulcers about the size of a threepenny-bit with sloughy base and punched-out edges.

Treatment is essentially the same as that described under acute lacunar tonsillitis.

Acute parenchymatous tonsillitis and peritonsillitis (Quinsy).—This condition is peculiarly common in rheumatic subjects. One or both tonsils may be attacked either simultaneously or in succession.

PATHOLOGY.—An inflammation attacking the lymph-nodes and peritonsillar tissue, frequently terminating in suppuration. Bacteriological examination of the pus shows the presence of micro-organisms, most frequently streptococci.

SYMPTOMS.—From the commencement of the attack fever is high, and when pus forms there may be a rigor. There is headache, pain in the neck and throat, radiating to the ear, due to cellulitis and periadenitis, difficulty in speech and swallowing, and, as the case progresses, in breathing. Much stringy mucus forms and flows from the mouth, the tongue is foul, the bowels constipated. The swelling at the angle of the jaw may lead to difficulty in opening the mouth. There may be delirium, and prostration is always marked.

On inspection, the tonsil and soft palate on one or both sides are seen to be red, swollen, hard, and tender, and more or less coated with mucus. The tonsils may meet in the middle line. As the swelling progresses it tends to bulge towards the upper and front part of the anterior pillar, and if pus is present the abscess may show signs of pointing here. The glands at the angle of the jaw enlarge.

The inflammation may undergo resolution, or pus may form and escape spontaneously. The condition may last for from seven to ten days, but in a great many cases we can either secure resolution before this, or open the abscess early, and so relieve the patient of this intense suffering.

TREATMENT.—The general condition is relieved by a purge, and by the administration of salicylate of sodium in 20-gr. doses every 4 hours. The mouth and throat should be frequently syringed or gargled with an alkaline or antiseptic solution. The local condition may be checked by the use of guaiacum-lozenges, sucking ice, or by painting the tonsils with a 10-per-cent. solution of eucaine, or with a 50-per-cent. solution of guaiacol in oil of almonds.

During convalescence tonics are indicated in the form of iron or bark.

If suppuration has occurred and there is sign of pointing, the abscess should be incised by means of a bistoury and sinus-forceps to reach the pus, which is often very deeply situated. The incision can generally be made with greatest advantage through the soft palate, or upper and front part of the anterior pillar, in a direction from without inwards. A hot antiseptic gargle should then be used for a day or two.

Chronic enlargement of the tonsil.—This condition is most common in strumous, tubercular and rachitic subjects, but it is often seen in perfectly healthy children. Bad hygiene and repeated attacks of follicular or parenchymatous tonsillitis may be causes, and not infrequently the tonsils enlarge subsequent to an attack of one of the exanthemata. Hypertrophy of the tonsils is commonly associate with nasal obstruction and adenoid vegetations in the naso-pharynx, and no examination of the throat can be regarded as complete which does not include careful investigation of the condition of these cavities.

PATHOLOGY.—When the tonsils are soft and spongy the condition is one of increase of the lymphoid tissue of the glands. As a rule in adults

where the tonsils are firmer there is also an increase of the fibrous connective tissue.

SYMPTOMS.—On inspection, one or both tonsils are seen to be enlarged, sometimes completely blocking the fauces. The mouths of the crypts may be filled with caseous exudation, or the tonsil may be traversed by fibrous-looking bands giving rise to a 'honeycomb' appearance. Occasionally the anterior pillar of the fauces is adherent to the tonsil, which, instead of projecting forwards, shows a broad flattened surface extending back towards the posterior wall of the pharynx. The deep cervical glands may enlarge. Beyond giving rise to a slight choking sensation or difficulty in swallowing and alteration of voice, enlarged tonsils do not directly trouble the patient. Gradually, however, from obstruction of the naso-pharynx, mouth-breathing is adopted, the voice loses its clearness, becomes muffled or nasal in character, and there is difficulty in pronouncing palatal consonants. As a result of the respiratory obstruction the lungs are imperfectly distended, and the chest may assume the narrow, pigeon-breast type. Deafness and cough are common accompaniments, because adenoid vegetations are so often associated. Children with enlarged tonsils are often lethargic, ill-developed, and badly nourished. This results not only from the imperfect oxygenation of the blood, but also from constant absorption of bacteria and their products from the distended lacunæ. The repeated attacks of acute tonsillitis to which such persons are subject must, too, contribute to the general malaise. It seems certain that enlarged tonsils may excite attacks of asthma, bronchial catarrh, and in children be responsible for disturbed rest, night-terrors, &c., but all these symptoms are greatly exaggerated where adenoids coincidently exist.

TREATMENT.—In some cases the hypertrophy disappears at the age of puberty, and no treatment is called for. On the other hand, where there is definite respiratory obstruction, and the health and development of the patient are obviously suffering, treatment is imperative. No local treatment is of any avail in diminishing the enlargement, and the usual remedies have only the effect of distressing and frightening the patient. Enlarged tonsils in children should be excised with the tonsillotome or with a tonsil-punch; and in adults, as a rule, the glands may be removed in the same manner, though some surgeons use the galvanocautic snare, or an écraseur, or cauterise with the electric point at one or more sittings. The last procedure is tedious, and our experience of anything approaching serious hæmorrhage is so very slight that the tonsil-guillotine may be looked on as a safe instrument, even in persons many years older than adult age.

J. BARCLAY BARON.

TOOTHACHE.—SYNON.: Fr. *Odontalgie*; *Mal de Dents*; Ger. *Zahnweh*.—Pain in connection with the teeth. See **TEETH**, Diseases of.

TOOTH-RASH.—See **DENTITION**, Disorders of.

TOPHUS (*tophus*, sand).—A term associated with the concretions, consisting chiefly of sodium biurate, which are met with in gout, in connection with the joints and other structures (p. 332). It is also sometimes applied to gravel in the urine, and to the collection of tartar on the teeth. See **GOUT**.

TORMINA (Lat. griping).—This word is applied to severe griping or colicky pains in the abdomen, due to flatus and other causes. See COLIC; DYSENTERY; and INTESTINES, Diseases of.

TORPOR (Lat. numbness).—**SYNON.**: Fr. *Torpeur*; Ger. *Torpidität*.—A condition of inactivity, bodily and mental, which may be met with in certain cerebral diseases or febrile states, more especially in aged persons. The cerebral condition associated with torpor is an unnatural state of consciousness, closely allied to that known as stupor. See CONSCIOUSNESS, Disorders of.

TORQUAY, in South Devonshire.—A mild, rather relaxing, and sedative marine climate. Sheltered from W., N., and E. winds. Mean winter temperature 44° F. See CLIMATE, Treatment of Disease by.

TORSION (*torquco*, I twist).—This word signifies a twisting, and is used in medical literature in the following associations:—

1. In relation to certain organs or growths, it indicates a form of displacement in which the mass is twisted on itself, a condition especially noticed in connection with the intestines. It gives rise to more or less narrowing of canals, and may close them completely, so as to cause absolute obstruction. Torsion also interferes with the local circulation, thus leading to congestion, inflammation, or ultimately even to gangrene of organs or tumours. See INTESTINAL OBSTRUCTION; and OVARIES, Diseases of.

2. As a method of treatment, torsion is employed in checking arterial hæmorrhage, the end of the bleeding artery being seized by the aid of suitable forceps and twisted. It is chiefly used in bleeding from small arteries, but may prove efficient even when arteries of some size are the source of the hæmorrhage. FREDERICK T. ROBERTS.

TORTICOLLIS (*tortum*, twisted; and *collum*, the neck).—A synonym for wry-neck. See WRY-NECK.

TORULA.—**DESCRIPTION**.—Torula is a form of microscopic fungus, belonging to the group *Saccharomycetes*. It consists of round or ovoid cells, of an average diameter of about $\frac{1}{3000}$ inch, without nuclei, but composed of masses of vacuolated protoplasm, confined within a definite cell-wall. Occasionally they are free, but they are frequently associated into branching chains.

Vinous, acetous, and other fermentations are due to the presence of organisms, of which the 'yeast-plant,' *Mycoderma* or *Torula cerevisiæ*, is the best known. Certain varieties of torula are of constant occurrence in the alimentary canal, and would seem to be normally associated with intestinal digestion. In those cases of vomiting where the ejected matters ferment, torulæ are always to be found, together with sarcinæ. These bodies are also of frequent occurrence in the urine of diabetes mellitus, if left standing; but they have likewise been found in non-saccharine urine. The pathological significance of torula, if any, is not known.

W. H. ALLCHIN.

TOUCH, Disorders of.—**SYNON.**: Fr. *Troubles du Tact*; Ger. *Störungen des Tactsinnes*.—The sense of touch may be considered as a compound of four distinct senses, namely, those

of contact, pain, temperature, and muscular activity; and it is not necessary that all these should be affected simultaneously or in an equal degree. Sometimes but one is the seat of disorder, and occasionally only one escapes. The lesion producing tactile disorder may be in any part of the sensory apparatus—in the peripheral end-organ in the skin, which receives impressions, in the trunk of the nerve which conveys them, or in the central ganglion, the reaction in which is represented in consciousness as feeling. For the most part disorders of touch must consist either in a defective or in an unnaturally heightened reaction to impressions—conditions which are termed respectively *anæsthesia* (or *hypesthesia*) and *hyperæsthesia*. But there are besides certain abnormalities of sensation which cannot be referred to either of these categories, as, for example, when a touch causes a sensation of burning, or the electric current is felt as something cold, and in these circumstances the term *par-æsthesia* is used.

1. **Increased Sensibility.**—**DESCRIPTION.**—

It is doubtful whether the sense of touch proper, the power of tactile discrimination, is ever morbidly increased, except possibly in certain cases of hysteria and mental disorder. The term *hyper-æsthesia* would be properly applied to such a condition instead of, as it is more commonly used, to excess of sensibility to painful impressions, which is better called *hyperalgesia*. In cutaneous *hyperalgesia* even a light touch upon the skin produces more or less exquisite pain. The patient often cannot even wash the skin, which is described as feeling raw or sore to the touch (see PAIN IN VISCERAL DISEASE). The symptom frequently occurs in connection with neuralgia (especially of the trigeminal nerve), and in hysteria, as well as in the various forms of local inflammation. It may precede by some days the characteristic pains of neuralgia; is often associated with excess of sensibility to heat and cold; and usually with diminution of sensibility of the tactile sense proper. It is seen in its severest form in connection with gunshot injuries of nerves.

There may be heightened sensibility to temperature, either as regards heat or cold singly, or in respect to both at the same time. This symptom is observed in connection with both peripheral and central disease—as an accompaniment of neuritis, as well as of degenerative changes in the cord or cerebral ganglia.

Heightening of the sense of contact is rarely observed, and is of but little practical importance. Perhaps the condition known as 'fidgets' is best explained as depending upon a heightened sense of muscular activity.

TREATMENT.—So far as is practicable, the lesion which is the cause of hyperalgesia, whether peripheral or central, must be discovered and become the subject of treatment. But the symptom itself may be mitigated by appropriate means. Such are the local application of moist heat by fomentation or poultice; of cold by means of ice; or of anodynes, such as veratrine-ointment somewhat diluted, or atropine-ointment; or, as a last resource only, to be avoided if in any way possible, the hypodermic injection of morphine (gr. $\frac{1}{10}$ to gr. $\frac{1}{8}$). Spongipiline may be sprinkled with a liniment composed of chloroform one part and belladonna liniment three parts; or equal parts of ether, sal volatile, laudanum, and eau de Cologne may be

applied. A piece of lint soaked in chloroform may be laid upon the painful portion of skin and covered with oiled silk, or the part may be rubbed with camphor-chloral and vaseline, equal parts; or painted with amyl-colloid. The application of one pole of the continuous current to the hyperalgesic spots, while the other is placed on an indifferent part, will often be of service, the power of bearing a gradually increased strength showing the improvement produced. Hysterical hyperalgesia can sometimes be successfully treated by the application of a strong induced current, by means of the wire brush, the patient, if necessary, being placed under the influence of ether.

2. Defective Sensibility.—DESCRIPTION.—Cutaneous anaesthesia may result from local abstraction of heat, as from exposure to a very low temperature. In such a case anaemia is produced, from spastic contraction of blood-vessels, followed by hyperaemia from their secondary relaxation. In the anæmic stage, while the other tactile sensations are lowered, that of temperature is heightened. Deficient sensibility may be caused by irritating applications, such as soda used by laundresses, and various chemicals employed in the arts. In such cases there is numbness in the hands and forearms, with a sensation of 'going to sleep' in the fingers. It may occur in connection with *herpes zoster*, the skin between the groups of vesicles being often partially anaesthetic. In *lepra anaesthetica*, in which there are enlargements of the cutaneous nerves, the senses of temperature and pain are often abolished, and severe burns may take place without being recognised. Anaesthesia may be produced by pressure upon sensory or mixed nerves by syphilitic and other growths in adjacent tissues. Narcotics, as chloroform and ether, may quell the sense of pain, that of contact being, to a certain extent, retained. Wounds and lacerations of the sensory or mixed nerves, followed by inflammatory processes, may, by irritation, cause pain to precede the anaesthesia arising from the interruption of conductivity in the nerve-fibres. Simple mechanical pressure upon a nerve, if long continued, will often, especially if its nutrition be impaired by constitutional causes, excite a low inflammatory condition. In traumatic cases, as also in *lepra anaesthetica* and in cases of new-growths pressing on the nerve, motor and nutritive disturbances are apt to accompany the anaesthesia, the nerve-trunks conveying not only sensory, but also motor, vaso-motor, and trophic fibres. Severe trophic disorder is usually associated with the anaesthesia occasioned by lesion of the fifth nerve; and to a less extent with that accompanying trigeminal neuralgia. *See FIFTH NERVE, Diseases of.*

Preceding attacks of neuralgia, the skin of the part about to be affected is often found to be anaesthetic, and during attacks of sciatica and cervico-brachial neuralgia there is often much diminution of tactile sensibility, in the foot and lower part of the leg, and in the fingers respectively; while the skin around the eye may be greatly deficient in tactile sensibility during severe supra-orbital neuralgia. It is important to discriminate anaesthesia of the skin caused by diseases of the nervous centres from that which is of peripheral origin.

Cutaneous anaesthesia is occasionally an important symptom of an approaching cerebral hæmorrhage. A sudden and increasing numbness is experienced in one half of the face, or in the limbs on one side

of the body, which may be followed shortly by hemiplegia and coma. An apoplectic seizure usually causes unilateral cutaneous anaesthesia, which is at first widely diffused, owing probably to the disturbance of circulation in, and consequent disarrangement of, the nervous molecules, and extends at first far beyond the site of the effusion. A few hours or days usually suffice for the clearing up of this anaesthesia, leaving, however, a subjective feeling of numbness, which may endure for a longer or shorter period.

The extent of anaesthesia bears no necessary relation to the amount of motor paralysis. It usually affects the paralysed side of the body, but in certain cases of hæmorrhage into or other lesion of the medulla oblongata and pons Varolii, it may occupy the opposite side. Complete hemi-anaesthesia of central origin may persist long after the paralysis of motion has disappeared, and in such a case a lesion is likely to be found involving the internal capsule. Occasionally, too, hemi-anaesthesia may from the first be unaccompanied by motor paralysis. Much more frequently, however, cutaneous anaesthesia (except for the first few hours) is of comparatively slight and transitory character, even in cases where there has been extensive disorganisation of the brain from hæmorrhage or softening, and where the resulting paralysis of the muscles is complete and permanent. Recovery is gradual, and proceeds downwards, the fingers sometimes retaining slight anaesthesia long after the rest of the arm has entirely recovered.

Cerebral tumours may give rise to cutaneous anaesthesia by pressure upon the Gasserian ganglion, or upon the trunk or branches of the fifth nerve as they traverse the floor of the skull. Like the motor paresis or paralysis which may be occasioned at the same time, the loss of sensibility is usually, but not always, gradual, tending to increase rather than to diminish as time goes on. It is not usually a prominent symptom in cerebral abscess.

Lesion of the spinal cord or of its membranes may give rise to cutaneous anaesthesia, which is frequently, in the lower extremities, extensive and complete; but it may be absent when—as, for example, in very advanced sclerosis of the antero-lateral columns—there is complete paraplegia. There is a form of progressive disease of the central and posterior grey matter of the spinal cord, giving rise to excavation, in which cutaneous sensibility is apt to be modified in a remarkable manner. The sense of touch, the sense of pressure, as well as the electro-muscular sensibility and the muscular sense, are preserved; but the appreciation of temperature and the sensation of pain (one or both) are lost in many situations. The condition is accompanied by paralysis and various forms of nutritional disturbances (*see SPINAL CORD, Diseases of: 22. Syringomyelia, p. 1559*). A varying amount of cutaneous anaesthesia, especially affecting the soles of the feet, is apt to occur in tabes. Anaesthesia of spinal origin is usually bilateral; but it is believed to affect that lower extremity alone which is opposite to the one paralysed in its motility, when the causative lesion is limited to one lateral half of the cord. Intercurrent complications from disturbances of circulation, the temperature of the limb, the extension or subsidence of inflammation, and the effusion of inflammatory products about the posterior roots, as well as the spread of sclerotic changes, may cause the extent and completeness of

cutaneous anæsthesia to vary considerably in cases dependent upon spinal-cord disease. Where the lesion lies tolerably high up, tickling the soles of the feet, although quite unfelt by the patient, is able to excite the motor nerves, and produce reflex muscular contractions, which the loss of muscular sense prevents him from recognising. It is extremely important to remember that cutaneous anæsthesia of spinal origin is liable to be associated with bed-sores. In certain cases, probably where trophic and vaso-motor nerves have been included in the lesion, this liability is excessive, and may defy all precautions.

Anæsthesia is of very frequent occurrence as a result of peripheral neuritis, both in its localised and multiple form. See NEURITIS, MULTIPLE.

Loss of the *sense of muscular activity* may occur in an isolated form, the other modes of tactile sensibility being unaffected; or it may be associated with impairment of some or all of them. The symptom is especially notable in progressive locomotor ataxy (see TABES DORSALIS). It may also occur in connection with paresis resulting from coarse disease of the occipital lobe of the cerebrum. Loss of muscular sense may accompany hemiplegia, attended by strongly marked and prolonged anæsthesia, from disease of the internal capsule. It occurs sometimes in hysteria. There is a form of anæsthesia occasionally met with in hysteria which it is important to recognise, so as not to confound it with a somewhat similar condition resulting from organic disease of the internal capsule. In this the patient may lose the power of perceiving impressions of contact, temperature, and pain throughout the whole of one lateral half of the body, sharply divided from the sound side by a line passing downwards from the vertex to the os pubis. Accompanying this *hemianæsthesia*, as it is called, there is often amblyopia and colour-blindness of the corresponding eye, loss of taste and smell, together with tenderness on deep pressure over the region of the ovary on the same side. In some cases, too, the skin is unnaturally pale and cold, and pricks with a pin are said to be not followed by bleeding, which readily takes place in corresponding circumstances on the opposite side of the body.

There is a curious form of disturbance of tactile sensation in which, the patient being blindfolded, a point of contact is referred not to its true locality, but either to some part of the opposite limb, or to a distant part of the same member (*allochiria*).

DIAGNOSIS.—As regards both hyperæsthesia and anæsthesia the most important consideration, after establishing the existence of either, is as to whether the cause be central or peripheral. It is impossible to do more than indicate the general principles upon which this inquiry is to be conducted. The patient's history, the condition of viscera and circulation, the existence or not of accompanying paralysis or of modifications of the organs of special sense, will lend important aid. As a rule, the anæsthesia of central origin is much more widely diffused, though less complete, than that dependent upon lesion of nerve-trunks, when it is also often accompanied by localised atrophy of muscles or other trophic disturbance. It is very rarely that hemianæsthesia of central origin is so complete as the hysterical, and it is not accompanied, like the latter, by tenderness on deep pressure over the ovarian region. There is no doubt that hyperæsthesia has often been mistaken for localised inflammation, and

treated accordingly. The absence of febrile movement, and the fact that it is mainly upon light surface-touching that the exquisite tenderness occurs, which fails to be felt when deeper pressure is made, coupled with the history, and a study of the concomitant condition, ought to suffice to prevent all mistakes.

TREATMENT.—Anæsthesia is a symptom of a lesion either in the central nervous system or in a peripheral nerve, and its treatment is bound up with that of the disorder which gives rise to it. But there are many cases in which, apparently as a result of disease, the sensory nerves fail to convey impressions for a considerable time after the lesion which interfered with their function has been healed. In such circumstances very much good can often be done by electrical treatment. The skin, carefully dried, should be brushed over for a few minutes every day with the wire brush, connected with an induction-machine; or the well-wetted rheophore connected with the negative pole of a continuous-current battery may be slid about over the affected surface, well moistened with hot water, the positive pole, also well wetted, being placed on an indifferent spot.

Static electricity is a valuable means of combating anæsthesia of this kind. The patient, seated on an insulating chair, is connected by a conducting chain with a frictional electrical machine, and sparks are drawn from the affected surface.

3. *Paræsthesia*. — DESCRIPTION. — The varieties in disorder of the different kinds of tactile sensibility—touch, pain, temperature, muscular activity—are very numerous. Pinches or pricks with a needle may be felt as touch only, while a very light touch with the finger is appreciated as heat, cold, or pain. Strong faradic currents (intolerable to the healthy) may be felt as cold. Heat may be felt as cold, but, being kept still longer applied, may be recognised as heat or warmth. A limb plunged into hot or cold water may get the feeling, not of heat or cold, but of pain. To such modifications, as well as to feelings of burning or cold, tingling, creeping of ants, or actual numbness, the term *paræsthesia* is often applied. A seamstress may be able to pick up and thread her needle, evincing thereby considerable delicacy of sense of contact, and yet be scarcely able to feel a prick of a needle in the finger-tip. Or the sense of contact may be in abeyance, as well as that of pain and temperature, and the movements may be then guided by the sense of muscular activity, aided by sight.

T. BUZZARD.

TOXÆMIA (τοξικόν, a poison; and αἷμα, blood).—This word literally signifies poisoning of the blood. It is not employed with any very strict or definite meaning, but most commonly implies blood-poisoning due to some pathological condition within the body itself, in contradistinction to that which results from the introduction of the ordinary poisons from without. As illustrations of toxæmic states may be mentioned pyæmia and septicæmia; uræmia; and certain forms of jaundice.

TOXINS.—The word 'toxin' was introduced by Brierger for certain poisonous bodies produced in connection with the vital processes of bacteria and chemically related to the alkaloids. It was afterwards used for all bacterial poisons, alkaloidal and non-alkaloidal. With the development of the

idea that these non-alkaloidal poisons are proteid in character, the term 'toxalbumin' was used as the collective name for bodies of this class, and included also certain vegetable poisons like ricin, crotin, and abrin, along with certain animal poisons like snake-venom. Still, the word 'toxin' is used loosely for poisons of varying origin and of different chemical constitution.

The origin of bacterial toxins may be considered from two points of view, which are fundamentally different. They are either produced as secretory products by bacteria, or they are contained within the bodies of bacteria and are integral parts of the protoplasm. In the first class the poisons exist and manifest their effects apart from the bacteria which produced them. In the poisons in the second group no toxic effect is produced when the bodies of the bacteria are removed by filtration. As these two types of poisons undoubtedly differ chemically, it is well to separate them as suggested by Buchner, who has proposed to call the endobacterial poisons 'proteins,' reserving the name 'toxins' for the soluble poisons in the first group. From a practical point of view the important bacterial toxins are those of diphtheria, tetanus, and botulismus, while closely allied to them are poisonous vegetable proteids like ricin, abrin, crotin, and certain poisonous proteids of animal origin like cobra-venom. Perhaps one should also include the complex hæmotoxins which are found in certain normal sera, the most important poison of this group being represented by ichthyotoxin, the poison present in the serum of the eel.

As these toxins are unknown as chemically pure products, one usually understands by the term a solution of the poison in some more or less indifferent fluid. For the bacterial toxins this fluid is usually some medium like bouillon or serum, from which the bacteria have been removed by filtration. From the researches of Brieger and Boer it does not appear probable that bacterial toxins are proteid in character, whereas, as far as is known, the vegetable poisons ricin, crotin, and snake-venom are proteids; at any rate, if all proteid is removed, toxic effects can no longer be obtained. In general, the differentiation of these toxins can be made only biologically, and of these biological characters the most important is the ability to produce antitoxin (*see* ANTITOXIN).

With the exception of snake-venom and ichthyotoxin, which act immediately, a definite period elapses between the introduction of the poison and the manifestation of its effects, this incubation-period depending on the nature of the poison to a large extent. In most cases the incubation-period is 6-24 hours; in the case of certain modifications of diphtheria-toxin (toxoid) it may be much longer (2-3 weeks).

The toxins are, as above mentioned, able to produce antibodies, a property which is not possessed by any of the poisons which have an ascertained chemical constitution (morphine, strychnine). This points to the view that the mode of action of the two types of poison on the organism must be different. Apparently the union of poisons like alkaloids with the tissues is slight, as is shown by the fact that the poison can readily be extracted by chemical reagents (Ehrlich, Mayer, Spiro), whereas the true toxins cannot be removed after union has taken place, and are thus probably firmly anchored to the tissues. This fixation takes place very rapidly, probably in

virtue of a specific haptophoric atom-group in the toxin-molecule, and the protoplasm is thus brought under the influence of the poison, the clinical and pathological effects of which are due to a toxophoric atom-group. This theoretical conception of the constitution of a toxin is also borne out experimentally in the case of the frog inoculated with tetanus-toxin. As long as the animal is kept at a low temperature tetanic symptoms do not break out, whereas they manifest themselves in a few hours if the animal's temperature be raised, the explanation of this apparently being that the haptophoric atom-group of the toxin-molecule acts at the low temperature, whereas the toxophoric group acts only when the temperature is raised. The separation of the toxin-molecule into a haptophoric and toxophoric group permits of the explanation of many of the phenomena observed in connection with toxins. Apparently the same haptophoric group may be present, although the toxophore varies. This is the case, e.g., with diphtheria-toxin (the poison of diphtheria, which produces marked lethal effects and local necrosis), and diphtheria-toxon, a poison which is also produced in the diphtheria-cultures, and which is not acutely fatal, but after a long incubation-period gives rise to paralysis. Both the toxin and the toxon possess the same haptophoric group, as immunisation with toxin produces immunity against toxon, and immunisation with toxon protects against toxin (Madsen and Dreyer). For these non-lethal modifications of toxins, Ehrlich has proposed the name 'toxoids,' and they appear to be important bodies which still require much investigation.

For some considerable time it has also been believed that the toxins are related to the ferments, and there is a good deal which supports this view, especially (1) the specific action of the toxins in extraordinarily small doses; (2) the attachment of toxins to precipitates; (3) the instability of toxins in the presence of injurious agents like heat, light, chemicals; (4) the fact that certain ferments, notably rennin, are capable of producing antitoxin, still further bears out the analogy, both toxin and ferment possessing a haptophoric group, and a toxophoric group in the case of toxins, and a zymophoric group in the case of ferments.

The conditions under which bacterial toxins are produced are complicated. There appear to exist toxigenic races of bacteria, from which the toxins are secreted. In some cases toxic bacteria are aerobic (*B. diphtheriæ*), in others anaerobic (*B. tetani*, *B. botulinus*). In most cases the cultures are grown under conditions of aërobiosis or anaërobiosis for 7-10 days, and then filtered through a Chamberland bougie, the filtrate ('toxin') being conserved in the absence of air and light.

The susceptibility of an animal tissue for a toxin depends apparently on the presence or absence of atom-groups in the protoplasm, which have an affinity for the haptophoric group of the toxin-molecule. These protoplasmic receiving-groups ('Receptors' of Ehrlich) vary in quantity and in quality, and the natural immunity to certain poisons probably depends on the absence of suitable receptors, and this absence or relatively slight development of receptors may occur in different animals or vary individually in animals of the same species. For example, the botulismus-toxin, which affects rabbits and monkeys in minute doses, is inert, even in large doses, in dogs. Further, crotin may be

toxic or non-toxic for rabbits. The suitable receptors may be widely distributed in different tissues, or they may be specialised in individual tissues, e.g. in the case of the central nervous system of the mouse and the guinea-pig in regard to tetanus-toxin. In this way one can recognise why nervous symptoms predominate in these animals after the injection of tetanus-toxin. The large local lesions produced by the injection of diphtheria-toxin or abrin would go to show that the receptors for the lymphophores of these toxins are not limited to special organs, and as a consequence that antitoxins against these toxins may be formed in the most varied sites; and this seems to have been proved by the experiments of Roemer as regards the formation of anti-abrin, the antitoxins being vital products of the cells and corresponding to Ehrlich's 'side-chains' or 'receptors' (see ANTITOXIN). The deeper study of toxins in the last few years has shown that they are much more complicated than was originally supposed, and it is not at all impossible that one and the same micro-organism may secrete different toxins, and may also be capable of producing different antitoxins (e.g. anti-tetanolyisin and anti-tetanospasmin). See TETANUS.

For the great majority of bacteria which affect man, and in all of which poisonous effects are manifested, the poisons belong to Buchner's 'proteins,' i.e. they do not exist apart from the bacteria producing them, and are not, so far as is known, secretory products. In most cases young cultures yield filtrates, which are not at all or only slightly toxic. With old cultures a certain amount of poisonous effect may be observed, and this is usually referred to the diffusion of certain quantities of poison out of the bodies of the bacteria which have died. For example, the filtrates of young cultures of Bacterium influenzae are only slightly toxic, whereas living or dead cultures produce signs of profound intoxication, and in man the symptoms of influenza must largely be toxic, when it is known that in the majority of cases the bacillus remains local in the respiratory system.

In some cases such as anthrax, gonorrhoea, and cholera, various poisonous bodies of complicated constitution have been obtained by precipitation of the cultures, but it has justly been doubted whether such artificially prepared products pre-exist in the cultures. In the majority of cases the introduction into the animal of dead or living cultures containing proteins does not lead to the formation of antitoxins, and the serum of such animals is capable of protecting other animals only to a very slight extent. For cholera and typhoid R. Pfeiffer has shown that such immune serums do contain antibodies, but these in themselves are unable to destroy the *Vibrio cholerae* and *Bacillus typhosus*. For this purpose another body, ferment-like in character, must be present, so that the destruction of such poisonous bacteria depends on the collaboration of antibodies ('immune body') and a ferment-like substance, known as the 'alexine' or 'complement.' For the complex hæmotoxins, which destroy the red blood-corpuscles and produce laking of the blood, Bordet, Ehrlich, and Morgenroth have shown that two similar substances are necessary—viz. a specific antibody produced by the immunisation, and a complement, which is normally present in the serum in relatively small amount. The immune body may be present in large amount, and yet fail to protect a healthy animal if there is not sufficient

complement. What apparently happens is, that the immune body is fixed to the cell (bacterium or blood-corpuscle) for which, as a result of the immunisation, it has a specific affinity; it also fixes the complement which is, in reality, the active agent of destruction. The complement itself does not become fixed to the cell directly, but only mediately through the immune body. The complement is thermo-unstable and rapidly breaks up when kept any time outside the body, and this explains why such antibacterial serums manifest extraordinarily slight bacteriolytic effects *in vitro*, unless they are perfectly fresh; and even when perfectly fresh their destructive power is limited, as the complement is used up and *in vitro* cannot be reproduced. In the living body such antibacterial serums may, however, be used as prophylactics. The most recent researches would also go to show that these cell-poisons are very numerous, and likewise that there exist many complements in one and the same animal. The exact chemistry of such poisons is at present, however, quite unknown.

WILLIAM BULLOCH.

TOXOID.—See TOXINS.

TRACHEA, Diseases of.—SYNON.: Fr. *Maladies de la Trachée*; Ger. *Krankheiten der Luftröhre*.—The trachea is but little prone to disease, except in association with affections of the larynx, bronchi, and neighbouring parts. The diagnosis and treatment of these several diseases are greatly facilitated by the laryngoscope. With this instrument a skilful manipulator can in many cases examine the trachea in its whole length; and an accurate diagnosis being thus attained, remedies may be applied, and instruments may be introduced for the removal or destruction of growths, or for other purposes, either through the larynx, or by an artificial opening made in the trachea. The principal morbid affections of the trachea will be discussed in the following order: (1) Malformations; (2) Inflammation; (3) Ulceration and Perforation; (4) Syphilis; (5) Tuberculosis; (6) Tumours; (7) Stenosis; and (8) Foreign Bodies.

1. **Malformations.**—Defects in the development of the trachea occur as rare causes of the death of newly born infants. The tube may be short and imperforate; or communication may exist with the œsophagus. These conditions are necessarily fatal. A fistulous opening through the skin occasionally occurs, giving rise to no serious symptoms. *Tracheocele*, a hernia of the mucous lining of the trachea, is a rare malformation, easily recognised, which may arise from a congenital defect, but is more frequently acquired.

2. **Inflammation.**—Tracheitis—simple, specific, or diphtheritic—may result from the extension of inflammation, either from the larynx above, or from the bronchial tubes below; it is rare except in this connection. Some degree of congestion is a usual condition of ordinary catarrh; and tracheitis is a frequent cause of irritable cough. See BRONCHI, Diseases of; DIPHTHERIA; and LARYNX, Diseases of.

3. **Ulceration and Perforation.**—Ulceration and perforation of the walls of the trachea may result from carcinoma of the œsophagus; from the pressure of an aneurysm, which ends by bursting into the air-passages, where it meets with least resistance; or from an abscess which has taken a

similar course. In the last case suppuration usually occurs in connection with a caseous bronchial gland. Occasionally, as witnessed by more than one writer, a caseous gland may become dislodged and impacted in the trachea, causing sudden death by suffocation.

4. **Syphilis.**—Syphilis, in its secondary and tertiary stages, may affect the trachea. In the tertiary stage it gives rise to a localised form of infiltration or syphiloma; and to ulcers, which contract in healing, and cause a formidable condition of stricture, to be presently considered. Tracheal syphilis, being in its advanced form so grave a matter, calls for active constitutional treatment before this irremediable stage is reached.

5. **Tuberculosis.**—Tubercle affecting the trachea occurs in connection with pulmonary phthisis, and is rarely found without advanced tubercular disease of the larynx. Two main forms may be distinguished, namely—multiple shallow ulcers, which often coalesce to form large sinuous tracts of ulceration, mostly affecting the cartilaginous portion; and deep infiltration and ulceration, confined to the posterior or membranous part of the tube. When the disease has proceeded to ulceration, it may cause the rare complication of general emphysema, the air being forced into the cellular tissue by cough and other expiratory efforts, made when the larynx is closed. In the treatment of this affection, palliative measures are alone available. See LARYNX, Diseases of.

6. **Tumours.**—(a) *Cancer.*—Primary carcinoma of the trachea is very rare, but the organ is frequently affected by the extension of the disease from neighbouring parts. The growth first causes the symptoms of stenosis, and then, as ulceration proceeds, it gives rise to expectoration and cough; whereas, as Gerhardt has pointed out, in syphilitic disease cough and expectoration precede the onset of dyspnoea, which develops very gradually. The diagnosis may be very difficult in the primary form, and can only be made with the aid of the laryngoscope. In the case of extension from neighbouring parts the diagnosis is comparatively easy. The only treatment available for prolonging life is tracheotomy, if the seat of the disease is high enough to admit of it. (b) *Non-malignant growths.*—Polypi are very rare. The symptoms are those of obstructed breathing, modified by the size and seat of the growth. A certain diagnosis can be attained only by tracheoscopy. Without treatment, a polypus is very likely to cause death by suffocation, its rate of growth depending on its pathological nature. Small growths situated high up may be treated by the galvano-cautery or by other applications through the larynx; larger tumours can only be removed through a free opening made into the trachea. A tumour may be so situated that tracheotomy, without extirpation, may ensure the safety of the patient.

7. **Stenosis.**—The calibre of the trachea may be lessened (a) by *stricture*, or by *tumours growing within it*; or (b) by *pressure from without*.

(a) *Stenosis from true stricture, or internal tumour.*—Tracheal stricture is almost always the result of syphilis; it may be annular and limited, but usually involves the tube for some length towards its lower end, and frequently extends into the bronchi. Tracheal narrowing is indicated by inspiratory stridor and dyspnoea, unaccompanied by the up-and-down movement of the larynx, and the affection of the voice characteristic of laryngeal

dyspnoea, and also in uncomplicated cases without the stethoscopic signs of pressure on, or plugging of, a bronchial tube. Under these circumstances, and in the absence of any tumour in the neck or thorax pressing on the trachea, the stenosis must depend on a stricture, or on a tumour within the tube. A syphilitic history would lead us to suspect the former; and a tracheoscopic examination may make the diagnosis certain.

Cicatrical stricture at times develops in consequence of the long-continued irritation of a tracheotomy tube.

In rare instances stenosis of the trachea may result from a chronic form of tuberculosis resembling lupus, as well as from leprosy, glanders, and rhinoscleroma.

Symptoms of stenosis are occasionally evoked by spasm of the trachealis muscle.

PROGNOSIS.—Stricture being usually cicatrical, the prognosis is most unfavourable.

TREATMENT.—Treatment other than operative is seldom available. If the stricture be high up, tracheotomy must be performed below it; or, an opening being made above the contraction, a long flexible tube may be introduced and passed through it. Stricture of the trachea is less amenable than laryngeal stenosis to treatment by mechanical dilatation with hollow bougies, but this method must be borne in mind for exceptional cases.

(b) *Stenosis from compression.*—The source of stenosis caused by pressure from without is usually obvious, as in the common case of enlargement of the thyroid gland, thoracic tumours being diagnosed by their physical signs and concomitant symptoms. The paroxysmal dyspnoea frequently caused by these tumours is not laryngeal in most cases, but depends, as shown by Bristowe, on the accumulation of secretion below the seat of obstruction. It is not relieved by tracheotomy, except where the upper part of the trachea is alone compressed. In cases of stenosis from pressure of a goitre, division of the isthmus or excision of part of the thyroid gland is sometimes very successful, and should always be tried, if possible, before tracheotomy is resorted to.

8. **Foreign Bodies.**—A foreign body entering the air-passages from the pharynx may lodge in the larynx, either becoming impacted or lying loose; but, unless prevented by its form or bulk, it usually falls or is drawn through the open glottis into the trachea. Here it may lodge; but it more frequently passes on into one of the bronchial divisions—most frequently into the right bronchus, the orifice of which is slightly larger than that of the left, and occupies more of the floor of the trachea.

SYMPTOMS.—Occlusion of the larynx by a foreign body, which from its bulk obstructs the passage, may cause instant death; and the same may be said of the trachea, as when a person vomiting, in a state of unconsciousness from intoxication, or from the action of an anæsthetic, draws in a quantity of food sufficient to choke up the air-passages. If the body be smaller, it causes dyspnoea, with severe exacerbations from spasm. A very small body, such as a sharp piece of bone or a pin, may be impacted in a position in which it causes only pain and dysphagia without dyspnoea. Speaking generally, it may be said that when the substance has passed into the trachea, the symptoms to which it gives rise depend on its bulk and weight. Rarely, it lies in the air-passage, giving rise to no symptoms; more frequently, varying its position with

the rush of air in coughing, &c., it gives rise to paroxysmal dyspnoea, light bodies being forced up to the glottis and exciting spasm. If the substance pass into the bronchus, it may become impacted there, and will give rise to characteristic physical signs, usually exciting a chronic circumscribed inflammation, with symptoms akin to pulmonary phthisis.

TREATMENT.—From the larynx a foreign body may be removed by the finger or a suitable forceps, its presence having been determined from the symptoms, aided by digital or laryngoscopic examination. Although different forms of long forceps are made for passing through the larynx into the trachea, it is seldom practicable to remove *per vias naturales* a foreign body which has once passed through the rima. A free opening must be made in the trachea, and its edges held well asunder, to give a chance of the body being expelled by cough. If this fail, a forceps must be introduced through the opening, and the body, if possible, extracted. Inversion of the patient, so as to allow a heavy substance to fall back through the glottis into the pharynx, is sometimes successful, but not as a rule without previous tracheotomy.

T. J. WALKER.
PERCY KIDD.

TRANCE (*transitus*), a going beyond—of the soul from the body).—**SYNON.**: Lethargy; Fr. *Léthargie*; *Maladie du Sommeil*; Ger. *Schlafsucht*.

DEFINITION.—A sleep-like state, which comes on spontaneously, apart from any gross lesion of the brain or toxic cause, and from which the sleeper cannot be roused.

The term 'trance,' in its derivative meaning, aptly expresses the apparent reduction to a vegetative life, but the popular use of the word refers rather to the separate activity of the mind than to the inactivity of the body. Hence many writers prefer the term 'lethargy,' which also, though etymologically exact, is currently employed in a modified sense. The condition is sometimes included under the generic term 'catalepsy,' according to its etymological meaning, 'a seizing'; but this term is usually restricted to those forms which present a peculiar rigidity. See **CALEPSY**, p. 255; **MENTAL STUPOR**, p. 766.

The common form of trance (which is not frequent) is one of many symptoms of a general state of the nervous system, seldom dangerous to life. In the 'African lethargy' the tendency to sleep is the chief manifestation of a disease, the gravity of which is extreme.

1. Common Trance.—**ÆTIOLOGY.**—The influence of heredity in relation to trance is to be traced only in the existence of a 'neuropathic disposition.' The state occurs chiefly in the female sex, between the ages of twelve and thirty; very rarely in young men or children. The subjects are seldom in perfect health; they usually present various manifestations of hysteria, and are often anæmic. The condition has been immediately due in some cases to exhausting diseases, as typhoid fever and influenza, to excessive brain-work, to insolation, or to mechanical obstruction to the supply of blood to the head. The immediate cause is either the cumulative effect of such persistent influence, exhaustion, or some emotional disturbance. It may succeed an hysterical convulsion. Rarely

no exciting cause may be discoverable. In still more rare instances the state has been voluntarily induced, as in the well-known case of Colonel Townsend, who could throw himself into a condition of apparent death, lasting several hours. Such voluntary induction is occasionally seen in the East. Lastly, minor degrees of trance may, without difficulty, be artificially produced in most hysterical persons, and less readily in many others, by the methods described in the article on **HYPNOTISM**. The state now designated 'hypnotism' is really induced trance, and trance has been accurately termed 'spontaneous hypnotism.'

Its various causes seem thus to involve impaired or deranged nutrition, or the action of a toxic agent (as after acute specific diseases), or an influence that disturbs the function of an ill-nourished brain.

SYMPTOMS.—The onset of the state of trance is usually sudden. For instance, in a case which came under the writer's notice, a girl went into a room by herself, and was found, shortly afterwards, in a state of trance-sleep, which lasted for thirty-eight hours. In another case (Madden) a young lady went into a room to change her dress, and was presently found on the bed in a state of trance which lasted for a fortnight. As already stated, it may succeed an hysteroid convulsion, and in some other cases the onset has been attended by an aura, resembling the globus hystericus. In the cases which succeeded typhoid fever (Madden) the delirium of the fever passed gradually into comatose sleep, which continued for several weeks. The condition is analogous to the speechlessness which may succeed typhoid. At the end of the acute stage of influenza spontaneous trance has suddenly come on, lasting hours or days.

During the state of trance, the countenance is usually extremely pale. The limbs are relaxed; although brief initial rigidity, and sometimes occasional recurring cataleptic rigidity, or transient convulsive spasms, tonic or clonic, have been noted. In a few instances distinct hysteroid fits have occurred from time to time during the course of the trance. The eyelids are usually closed, and may resist and quiver on attempts being made to open them. The eyeballs are directed upwards in most cases; they often deviate from the middle line, and sometimes diverge slightly. The pupils are usually moderately dilated; rarely they are somewhat contracted. The state of reflex action varies according to the depth of the trance. That from the limbs is sometimes excessive, so that cutaneous stimulation produces tonic rigidity. Much more frequently reflex action is lost; snuff blown into the nostrils causes no sneezing, ovarian compression has no effect, and pressure on 'hysterogenic' points, which may have existed before, no longer causes the usual convulsive symptoms. Reflex action from the conjunctiva, and even from the cornea, is commonly absent. The pupil may contract to light, but in lessened degree, and sometimes no distinct action can be observed.

The mental functions seem, in most cases, to be in complete abeyance. No manifestation of consciousness can be observed, or elicited by the most powerful cutaneous stimulation, and on recovery no recollection of the state is preserved. But in some cases volition only is lost, and the patient is aware of all that passes, although unable to give the slightest evidence of consciousness. The senses

may be even preternaturally acute, as in the analogous phase of induced hypnotism; or there may be spontaneous mental action, irrelevant to external impressions, and analogous to, probably identical with, the state of ordinary dreaming; it is manifested by exclamations, and even by movements. Rarely the 'obedient automatism' seen in induced hypnotism may be present; hallucinations occur, and actions are performed, according to suggestions made to the patient. The usual condition, however, is that of an entire absence of all evidence of mental activity.

The pallor of the face is the result of a profound depression of the vascular system. The pulse may be less or more frequent than normal, but it is invariably weaker, and it may be imperceptible. The cardiac impulse may disappear, although the heart-sounds are still to be heard, sometimes much weakened. Very rarely they have been inaudible. The breathing may be tranquil, slightly quickened, or slowed, or it may be so feeble and deliberate that no movement of the thoracic walls can be observed, no respiratory murmur can be heard in the lungs, and a mirror held over the mouth is undimmed by moisture. Temperature, when observed, has been normal in the central parts, lowered at the periphery. The secretions go on; the urine may be retained in the bladder, or passed into the bed. The catamenia are usually absent, but menstruation has been known to occur without modifying the course of the trance-sleep. In the cases in which the depression of the vital functions reaches an extreme degree, the patient appears dead to casual and sometimes to careful observation. This condition has been termed 'death-trance' (*Scheintod*). Persons have certainly been buried in this state, and during the recent epidemic of influenza an Italian narrowly escaped interment during the consequent trance.

DURATION AND COURSE.—The duration of trance has varied from a few hours or days to several weeks, months, or even a year. When of short duration, the trance-sleep may be unbroken; but when it lasts for more than a few days, there are usually remissions of a greater or less degree, in which, for instance, the patient will half-wake, take food in an automatic manner, and then relapse into stupor. A long trance-sleep may be more profound at first than later. Recovery may be sudden or gradual. Occasionally it is attended by some vaso-motor disturbance; in a well-authenticated case of death-trance the intense mental excitement produced by the preparations for fastening the coffin-lid occasioned a sweat to break out over the body. After the trance is over, nervous prostration remains for a time. In some cases repeated attacks occur, at intervals of days, months, or years. Most cases end favourably. The depression of the vital functions enables life to continue with a very small amount of nourishment, but occasionally death occurs in persons previously weakened.

PATHOLOGY.—The very few *post-mortem* examinations which have been made after death in trance throw no light on its nature. The theoretical pathology of the subject is involved in the obscurity which envelops all the psychical processes in health and disease, the nature of volition, and ordinary sleep. The lowered action of the brain in sleep, and its lessened blood-supply, have suggested the existence of cerebral anæmia, which the meagre results of anatomical investigation have been sup-

posed to confirm. It is certain that the condition is sometimes associated with defective cerebral nutrition; but that much more than cerebral anæmia is needed to explain the state of trance is evident from the facts that, on the one hand, it may occur when there is no preceding sign of defective blood-supply to the brain, while, on the other hand, the occurrence of cerebral anæmia without trance-sleep is a matter of daily observation. The phenomena of hypnotism also afford little support to the theory of the dependence of trance-sleep on cerebral anæmia; together with the symptoms of catalepsy, they suggest that the primary element is a peculiar state of inaction ('inhibition,' 'increased resistance') of some of the nerve-structures concerned in associating the highest cerebral functions with those of the lower cortical centres of motion and sensation.

DIAGNOSIS.—The diagnosis of trance rests on the impossibility of rousing the sleeper, combined with the absence of any evidence of a local cerebral lesion or a toxic cause. Other diagnostic symptoms are the pallor and vascular depression, the occurrence of convulsive phenomena of hysteroid type, and the history of other manifestations of hysteria. These symptoms sufficiently distinguish trance-sleep from apoplexy, for which, at the onset, it is sometimes mistaken. The distinction from catalepsy rests on the absence of the *flexibilitas cerea*, but catalepsy is merely a variety of trance. The peculiar tendency to brief sleep termed *narcolepsy* is distinguished from trance by the shortness of the periods of unconsciousness. Thus a man had from youth fallen asleep for a few minutes under various influences, and always did so when a probe was passed down a nasal fistula. It is to be remarked, however, that the term *narcolepsy* has been also applied in America to cases of true epilepsy, in which the attacks of *petit mal* are characterised by sudden somnolence.

In cases of 'death-trance,' in which no sign of vitality can be recognised, the presence of life may be ascertained (1) by the absence of any sign of decomposition; (2) by the normal appearance of the fundus oculi as seen with the ophthalmoscope; (3) by the persistence of the excitability of the muscles by electricity. This excitability disappears in three hours after actual death. In a case observed by Rosenthal, thirty hours after supposed death the muscles were still excitable, and in forty-four hours the patient awoke. See DEATH, Signs of.

Deepening sleepiness, not amounting, however, to coma, and scarcely to be called stupor, has attended some cases of subacute inflammation of the region of the oculo-motor nuclei described by Wernicke—a curious fact considering the manner in which relaxation of the levator is associated with ordinary sleep. Lastly, a benign form of 'summer somnolence' observed among the Swiss has been termed 'Gerlier's disease.'

PROGNOSIS.—In cases of hysterical lethargy the prognosis is fairly good. The attack usually passes off. In very rare cases death has occurred. The slighter the degree of the trance, the shorter is likely to be its duration. The prognosis is grave only when lethargy has been preceded by a state of great physical depression, and is the most serious when the condition succeeds an acute disease.

TREATMENT.—The treatment has to be directed to two ends: the maintenance of life, and the arrest

of the trance. Advantage must be taken of any intervals of semi-consciousness to give nourishment in a concentrated form. If swallowing is continuously impossible, food must be given by the nasal tube, or by enemata. Warmth should be applied to the extremities, and care taken to prevent bed-sores. In severe cases, every attempt at arrest is often fruitless. Errhines, as snuff, have usually no influence, and it is only in slight cases that this, or stimulation of the skin, as by sinapisms, is effective. The most powerful cutaneous excitant is strong faradisation. In a case under the writer's notice, which had lasted for thirty-six hours, strong faradisation to the arm quickly roused the patient. In another case, which lasted for several months, this treatment had, for a long time, no influence; afterwards the patient could be partially roused for a short time by faradisation, and by repeating the application at the same hour every day a tendency to periodical waking was established, the remissions became longer and more complete, and the attack was ultimately brought to an end. Nerve stimulants, such as ether and valerian, may be given by the bowel, or sulphuric ether may be injected subcutaneously. Alcohol must be given with caution and in small quantities; enemata of strong coffee are often more useful. A remedy which, from its effect on the vascular system, would certainly deserve trial in trance, is the inhalation of nitrite of amyl or the administration of nitroglycerin; of the latter $\frac{1}{100}$ to $\frac{1}{50}$ grain may be given in alcoholic solution three times a day. Transfusion of blood has been proposed, and would be justified in very grave cases following exhausting disease. The recurrence of attacks must be prevented by the improvement of health, physical and moral.

2. **African Lethargy.**—This form of trance is described separately. See NEGRO-LETHARGY.

W. R. GOWERS.

TRANSFUSION OF BLOOD (*trans*, across; *fundo*, I pour).—**DEFINITION.**—The pouring of blood from one animal into another.

This method of restoring patients believed to be dying was in vogue for nearly 300 years, but has now fallen into disuse. Often when an emergency calling for transfusion arose, no donor could be obtained and no apparatus was at hand. Most commonly the donor was bled into a clean bowl, the blood was at once taken up with an ordinary 2 or 3 oz. syringe, air was expelled and the blood was injected into a vein of the recipient—the injection being repeated till 10 or 12 oz. had been given. Galabin, relying upon the blood-pressure in the veins of the donor, improved upon the suggestion of Libavius by connecting the two cannulae with a short rubber tube. Aveling added a 2-drachm rubber bulb in the centre of the tube; the whole apparatus acted like a Higginson's syringe, fingers being used to compress the tube first on the recipient's, then (during compression of the bulb) on the donor's side of the bulb. This was, probably, the best of the many instruments invented for *direct* transfusion. Air was expelled from all tubes by filling them with a solution of salt or phosphate of sodium, which was usually allowed to escape before the entering blood.

In spite of all speed the blood sometimes coagulated during the transfusion, which could not be completed; sometimes it was supposed that clots were injected and that embolism occurred; some-

times air was thought to have entered the veins and right heart, but this danger was probably exaggerated; finally, in some cases hæmoglobinuria or hæmatinuria, fever, rigors, symptoms of septicæmia and even death occurred. In more than one instance the donor died, doubtless from some general septic infection, for evidence of infection of the wound was not uncommon.

To avoid the dangers connected with coagulation phosphate of sodium (Braxton Hicks) or ammonia (Richardson) was added to the blood; or it was decalcified by oxalate of ammonium (Wright); or the blood was defibrinated and strained through fine muslin before injection, Panum having shown that the absence of fibrin made no difference to the success of the transfusion. In spite of this a preference for entire blood continued to be felt.

Gradually it was shown that there were unavoidable dangers connected with transfusion. Landois, C. Ludwig, and others gave reasons for believing that all red corpuscles injected were destroyed within a few days, the hæmoglobin being dissolved in the serum—whence the hæmoglobinuria noted. Sachsendlahl found that a solution of hæmoglobin induces rapid destruction of leucocytes and consequent accumulation of ferment in the blood with symptoms of ferment fever and possibly thrombosis in great vessels. On the other hand it became increasingly clear that in the treatment of hæmorrhage, which was the main indication for transfusion, the essential point was to refill the vessels with some innocuous fluid—such as normal saline. Consequently, it has come to pass that, at all events in the treatment of hæmorrhage, transfusion of blood has been replaced by the infusion of normal saline solution. See SALINE SOLUTION, Infusion of.

STANLEY BOYD.

TRANSFUSION OF MILK.—This operation, or, as it is more correctly termed, *Infusion or Intravenous Injection of Milk*, was recommended in America by Thomas and others, as a substitute for transfusion of blood. Saline solutions have more recently replaced milk.

TRANSFUSION OF SALINE FLUIDS.—See SALINE SOLUTION, Infusion of.

TRAUMATIC (τραῦμα, a wound). — That which is associated or connected with a wound or injury.

TREMOR (Lat. trembling).—The most delicate form of clonic spasm, consisting of successive movements of very small amplitude. Tremors are seen principally in the hands, the head, the tongue, or the facial muscles, as a result of disease or of old age. They are commonly spoken of as 'coarse' or 'fine,' according to the amount of movement which they involve. See ALCOHOLISM; DISSEMINATED SCLEROSIS, p. 1553; GENERAL PARALYSIS OF THE INSANE; MERCURY, Poisoning by; PARALYSIS AGITANS; and SENILITY.

TRICHIASIS (τριχίς, the hair).—A morbid condition in which the eyelashes are inverted towards the eye. See EYE, AND ITS APPENDAGES, Diseases of, p. 518.

TRICHINA (τρίχινος, made of hair). — See ENTOZOA.

TRICHINOSIS or **TRICHINIASIS**. — *See* ENTOZOA.

TRICHOCEPHALUS (τριξ, a hair; and κεφαλή, a head). — *See* ENTOZOA.

TRICHOCLASIS (τριξ, a hair; and κλάσις, fracture). — *See* HAIR, Diseases of.

TRICHOMONAS VAGINALIS (τριξ, a hair; μονάς, a monad; and vaginalis, connected with the vagina). — A ciliated infusorial animalcule, discovered by Donne in the vaginal mucus, and somewhat resembling a spermatozoon. *See* RAPE.

TRICHOPHYTON (τριξ, a hair; and φυτόν, a plant). — A species of parasitic fungus, which attacks man and some animals. *See* TINEA, p. 1713.

TRICHORRHEXIS NODOSA (τριξ, a hair; ῥήξις, breaking; nodosus, knotty). — *See* HAIR, Diseases of.

TRICUSPID VALVES and **ORIFICE**, Diseases of. — *See* HEART, VALVES AND ORIFICES OF, Diseases of.

TRIFACIAL NERVE, Diseases of. — *See* FIFTH NERVE, Diseases of.

TRIONAL-HABIT. — *See* SULPHONALISM.

TRISMUS (τριζω, I gnash). — Lockjaw, or tetanic closure of the jaws; a prominent symptom in tetanus. *See* TETANUS.

TRISMUS NEONATORUM (Lat.). — A form of tetanus occurring in newly-born children. *See* TETANUS.

TROPHIC LESIONS. — **DESCRIPTION**. — This name is given to various departures from healthy nutrition, which are caused (a) by the cutting off, from certain tissues or parts, of some customary nervous influence, as in the production of 'secondary degenerations' in the nervous system (*see* SPINAL CORD, Diseases of, § 6), or in the production of rapid muscular atrophy, consequent upon the severance of or severe damage to motor nerves or their related ganglion-cells in the anterior cornua of the cord; and also to lesions or morbid changes which are caused (b) by some irritative or perverted influences passing *outwards* along sensory nerves to certain tissues, so as to weaken or otherwise disturb their nutrition. In this latter way, the nutrition of the skin and its appendages may be variously affected, leading to eruptions of different kinds, to atrophy, to ulceration, or undue proneness to inflammation, as well as to altered pigmentation of the skin or blanching of the hair. Or the nutrition of the joints may be affected, as in some forms of hemiplegia, and in locomotor ataxy more especially. In these various cases there may be disease, secondary or primary, of the grey matter of the spinal cord, or some irritative lesions of the sensory nerve-roots or trunks.

PATHOLOGY. — Much dispute has taken place during recent years as to the modes in which nutritive changes are brought about. Some have endeavoured to establish the existence of special 'trophic nerves,' and have taught that the various trophic lesions referred to above are to be explained

by a cutting off or a perversion of the influences usually operating upon the tissues through such nerves. Others believe that these nutritive changes can be accounted for by altered states of excitation of the vaso-motor nerves, leading to spasm or dilatation of the vessels supplying the parts affected, and, as consequences, to the nutritive changes themselves. Much evidence, however, could be cited against both these modes of explanation; and it seems, on the whole, more probable that trophic lesions are due either (a) to the cutting off of certain accustomed influences (*viâ* motor channels), or (b) to the action upon the tissues of perverted or unnatural influences (travelling in a peripheral direction *viâ* sensory channels).

On the subject of these trophic lesions *see also* SPINAL CORD, Diseases of; NEURITIS, MULTIPLE; and FACIAL ATROPHY.

H. CHARLTON BASTIAN.

TROPICAL DISEASES. — Diseases incident to hot climates. *See* AINNUM; BERIBERI; BLACK-WATER FEVER; CHOLERA, ASIATIC; CLIMATE; DENGUE; DISTOMA RINGERI; DYSENTERY; ECSTASY; ENTOZOA; EPIDEMIC DROPSY; FILARIASIS; FRAMBESIA; KALA-AZAR; LIVER, Diseases of; MALARIAL DISEASE; MYCETOMA; NEGRO-LETHARGY; ORIENTAL SORE; PLAGUE; PSILLOSIS; SUNSTROKE; TINEA IMBRICATA; and YELLOW FEVER.

TUBERCULAR. — This term was formerly applied to eruptions consisting of small prominences of the skin. This use of an expression suggesting a relationship to tuberculosis is open to serious objection, and should therefore be discontinued. 'Tubercular' should signify 'of the nature of tubercle,' e.g. *tubercular meningitis*; 'tuberculous,' 'full of tubercle,' e.g. *a tuberculous lung*.

TUBERCULAR MENINGITIS. — A form of meningitis dependent on the presence of tubercle-bacilli. *See* pp. 979 and 988.

TUBERCULOSIS (from *tuberculum*, a little lump). — **SYNON.**: Fr. *Tuberculose*; Ger. *Die Tuberkulose*.

Tuberculosis is an infectious disease caused by the invasion of the tubercle-bacillus. It is characterised by the formation of 'little lumps' or 'tubercles,' which show a tendency to unite and to undergo caseous, fibroid, or other degenerative changes. The organ most frequently attacked is the lung, but the pleura, the peritoneum, the meninges, the intestinal tract, the generative organs, and the bones are all common seats of the disease. Indeed, no tissue or organ in the body enjoys complete immunity from attack.

Frequency of the Disease. — Tuberculosis of the lungs has been known from the earliest times. To this disease Hippocrates (460–375 B.C.) first applied the term 'Phthisis,' while a classical description of its clinical manifestations may be found in the writings of Aretæus (first century A.D.). That the disease should thus have been well known to the ancient physicians is not surprising when we consider its extraordinary frequency. It is stated that one-seventh of mankind die of tuberculosis, and though this statement is not literally correct when applied to our own country, it is undoubtedly approximately true as regards the world in general.

For England and Wales the following are the exact figures during the decennial period (1881-1890), the last for which complete data are available:—

Total deaths from all causes	5,244,771
Deaths from phthisis	473,968
Deaths from 'other tubercular and scrofulous diseases'	115,422
Total from all tubercular diseases	589,390

From these figures it results that during these ten years approximately 1 out of every 11 deaths in our own country was caused by phthisis, and 1 out of every 9 by tubercular diseases in general.

Age and Sex.—No age is exempt from the ravages of the disease. In young children the glands, bones, and alimentary canal are chiefly attacked, while in the adult pulmonary tuberculosis is far more frequent. The age at which the greatest mortality from the latter is seen differs somewhat in different countries, but in England the maximum incidence occurs between the ages of 35 and 45. Between 20 and 60, however, the disease is very common.

Geographical Distribution.—Tuberculosis flourishes in every country and in every clime. It is, however, less frequent wherever the population is scattered and leads an open-air life; more frequent where overcrowding in large towns is common. Probably damp and variable climates are more favourable to the disease than bright and dry ones, but it would seem that aggregation together of the population is a factor of greater importance in determining the incidence of the disease than the exact nature of the climate. That *high altitude* may possess some antagonistic influence is not to be denied, but the extent of the rôle played by it has probably been exaggerated.

The Tubercle-Bacillus.—The experiments of Villemin (1865) proved conclusively that the old ideas as to the ætiology of tuberculosis were erroneous. Villemin showed that human tuberculous material injected into the rabbit was capable of setting up in this animal a generalised form of the disease, and further that the tuberculosis thus produced could be communicated in series from rabbit to rabbit by repeating the process of inoculation. These experiments were confirmed and amplified by other observers, and their result was to prove definitely that tuberculosis, so far from being a constitutional disease, the result of a special 'Diathesis' or 'Dyscrasia,' as until then had been believed, was in reality an infectious disease, produced by a definite and specific virus. The exact nature of the virus was, however, long debated, and it was not until March 1882, when Koch announced his discovery of the bacillus, that the question was finally set at rest.

Staining Properties.—The tubercle-bacillus differs from the majority of pathogenic micro-organisms in not staining by ordinary simple methods. Koch himself succeeded in colouring it by using a concentrated alcoholic solution of methylene blue made *alkaline* with caustic potash. The method, however, was tedious, and a better was speedily introduced by Ehrlich, which, with various modifications, has since held the field. Ehrlich's method depends upon the fact that though the tubercle-bacillus takes up stain with considerable difficulty, yet, having once imbibed it, it parts with it

only with reluctance. Hence, to stain the organism, strong dyes are necessary, while to decolourise the surrounding tissues and other bacteria strong acids are permissible. To express this unusual resistance on the part of the bacillus to the decolourising action of strong acids, the term 'acid-fast' (Ger. *sauer-fest*) has lately been applied to it. The method of staining as generally performed at the present day is as follows: The tissue to be examined is dried upon a cover-glass, and then stained in Ziehl's carbol-fuchsin (concentrated alcoholic solution of fuchsin, 11 c.c.; 5-per-cent. aqueous solution of carbolic acid, 100 c.c.). The staining solution must be warmed until steaming occurs, and should be allowed to act for 5 minutes. The specimen is next washed in water, then placed for a few seconds (5-10) in 25-per-cent. hydrochloric acid. When no further colour is discharged it should be transferred to 70-per-cent. alcohol. After a few seconds, if no further decolourisation takes place, it should again be washed in water, counterstained in Loeffler's methylene-blue (10-20 seconds), washed once more in water, dried between blotting-paper, and finally mounted in Canada balsam. Stained in this way, the tubercle-bacillus acquires a bright pink colour, and is sharply differentiated from the surrounding blue tissues. See also pp. 132 and 1575.

It should be at once stated, however, that this method of staining is not specific. As Koch himself showed, the bacillus of leprosy stains in an identical manner, and quite recently numerous other bacilli having similar staining properties have been discovered (see p. 1575).

Morphology of the Bacillus.—The following are the morphological appearances presented by the bacillus. As a rule the micro-organisms, whether occurring in the sputum, the tissues, or derived from cultures, take the form of minute rods 1.5-3.5 μ in length (about a quarter to half the diameter of a red blood-corpuscle), and .3 μ in breadth. They may be straight, but very commonly they are somewhat curved. When stained they either take the dye uniformly, or, as frequently happens, may present a beaded appearance, portions of the bacilli taking the colour well, while the intervening parts remain unstained. Koch himself regarded these unstained portions as spores, but it seems on the whole more probable that they are really minute empty spaces within the bacillary sheath, produced by the aggregation into little masses of the cell-protoplasm.

The above description does not, however, by any means exhaust the morphological appearances of the bacillus. Recent researches by Metchnikoff, Coppen Jones, and others have shown that sometimes in the secretions, and always in old agar-cultures, certain of the bacilli become much elongated and filamentous, and show also *true branching*. Further, in not a few cases, the terminal ends of these long forms, or of their branches, present definite *club-formations*. These appearances suggested to Metchnikoff that 'the bacillus, as ordinarily met with, is not the end-stage, but only a stage in the developmental cycle of a filamentous fungus.' This view has lately received much support from the work of Babes and Levaditi. These observers injected tubercle-bacilli into the subdural space of rabbits, and found that after thirty days the foci presented appearances very closely resembling those of actinomycosis, a central mass of bacilli and

filaments being observed, surrounded by a radiating zone of clubs. Similar results have also been obtained by Friedrich and by Schultze, and it would seem therefore that the bacillus should be regarded not as a simple bacillus, but as really belonging to the large *Streptothrix*-group, among which the *Actinomyces* or *Ray-Fungus* takes its place. It should be added, however, that though this view seems on the whole the most probable one, yet certain critics regard these less common morphological variations of the bacillus not as representing its highest developmental form, but indicating, on the contrary, degenerative changes.

The Chemistry of the Bacillus.—Concerning the chemical composition of the tubercle-bacillus but little knowledge has hitherto been available. It has generally been assumed, following the researches of Unna and Klebs, that the bacilli contained a considerable percentage of fat, and to the presence of this body was attributed their peculiar method of staining, but nothing really definite was known. A very important communication has recently (July 1901) been made to the Pathological Society of London on this subject by Bulloch and MacLeod. These observers show that a certain amount of *neutral fat* is present in the bacilli, as had been believed, but that the acid-fast property of the organisms (their inability, that is to say, to be decolourised even by strong acids, unless the action of the latter be unduly prolonged) is not due to this substance, since even after its complete extraction with boiling methylated spirit the bacilli retain their appearance and staining properties unaltered. These properties, however, are completely lost if now the micro-organisms are further extracted with boiling chloroform or benzene. If this be done, a substance possessing all the characters of a *wax* is obtained. This body itself is extremely acid-fast, while the residue of the bacilli, now an amorphous mass, is found to have lost this characteristic, and to stain easily with methylene-blue. The conclusion, therefore, that the peculiar staining properties of the bacillus are due to the presence in it of the wax seems unimpeachable.

Besides the neutral fat and wax thus discovered, certain *fatty acids*, *proteids*, and *salts* (chiefly phosphates) were also found. After their complete elimination a residue still remained behind, apparently allied in nature to *chitin*. In addition to the above a *lipochrome* was also isolated, which in strong alcoholic solution presented a golden yellow colour. Dissolved in chloroform, the liquid showed a distinct band at β , a little to the right of the E band of the spectrum, while the violet end was also absorbed. To this pigment is probably to be attributed the colour acquired by tubercular cultures.

The exact proportions in which these various constituents are each of them present within the micro-organism cannot as yet be stated. It may be noted, however, that according to Bulloch and MacLeod the fat, the fatty acids, the wax, and the lipochrome account altogether for 43.7 per cent. of the total weight of the bacillus.

For further details concerning the methods followed by the observers in this interesting and valuable research, reference must be made to their original paper.

Cultivation of the Bacillus.—Koch himself was successful in first cultivating the bacillus which he had discovered. For this purpose, finding that nutrient gelatine and broth were in this case un-

suitable, he introduced as a culture medium *blood-serum*, solidified at a temperature of 65° C. He showed that if material containing tubercle-bacilli were rubbed over the surface of this medium, and the tubes kept at a temperature of 37°, after 8-10 days colonies visible to the naked eye make their appearance. These have the aspect of minute dry scales, which later coalescing form a thin brownish crinkled growth. The serum is never liquefied, and the growth never penetrates below the surface. Under the microscope the colonies, especially those near the edges of the growth, present a very characteristic appearance. They consist of innumerable winding lines, often having the shape of the letter S, and by their interwindings giving rise to beautiful serpentine figures.

Koch had found that ordinary broth was but little adapted to the growth of the tubercle-bacillus, and the same is also true of nutrient agar. Nocard and Roux, however, discovered that both became admirable media for subcultures, if 6 per cent. of *glycerine* be added to them. The growth so obtained on the glycerine-agar is dry and crinkled, and resembles closely that occurring on blood-serum. On the broth a similar yellowish crinkled growth occurs on the surface, and from time to time sinks to the bottom. The liquid itself remains always clear. Another valuable medium, and one hardly sufficiently known, is *glycerinated potato*. The growth on this medium is more luxuriant than usual, and for obtaining cultures of the bacillus direct from tuberculous material, it may be recommended in preference to blood-serum. In milk also the bacillus grows well (Klein).

It will be seen therefore that the micro-organism may be readily cultivated at a temperature of 37° C. on glycerinated potato-agar or broth, in milk, and also, though not so luxuriantly, on serum. Media containing gelatine are not suitable for its cultivation, since under ordinary circumstances the bacillus does not grow at a temperature below 29° C. It must be added, nevertheless, that Sander claims to have obtained growth in potato-broth even at a temperature of 22° C. The fact is of interest, but should hardly be taken as indicating the likelihood in any but the rarest circumstances of a saprophytic growth of the organism outside the human body. Only very occasionally could all the circumstances necessary combine to be present in nature, and even should this occur we must remember what a potent aid to the destruction of the bacillus we always possess in the germicidal action of direct sunlight.

Resisting powers of the Bacillus.—The tubercle-bacillus shows towards certain destructive agencies unusual power of resistance. This resistance is not indeed so great as that possessed by spore-bearing organisms, and is probably to be explained by the peculiar chemical composition of the bacillus, and the large amount of wax which it contains. *Putrefaction*, for example, has but little effect upon it. Thus sputum has been found to retain its virulence, though allowed to putrefy for more than a month in the air, while portions of tuberculous material, buried and dug up again after an interval of 167 days, have still been able to produce the disease when inoculated into suitable animals. *Desiccation*, if prolonged for six months, leads to the destruction of the organism (Cornet), but sputum, dried at the ordinary temperature of the air for shorter periods, still remains virulent. *Extremely low temperature* seems to produce no result. Thus Swithinbank

found that direct contact with liquid air during 42 days (temperature $-186^{\circ}\text{C}.$) produced absolutely no effect whatever, subcultures, perfectly typical in every way, growing from the bacilli so exposed. Repeated freezing and thawing, and prolonged immersion in water, are similarly without result. No more efficacious is the *gastric juice*, the bacilli still remaining alive after an exposure of six hours to this secretion. *Heat*, on the other hand, is more effective. The bacilli are destroyed in milk by raising the liquid to the boiling-point (Bang), while similar results are also obtained by longer exposure at lower temperatures ($70^{\circ}\text{C}.$). Dry heat, however, is not nearly so certain in its results. Of the agents which in nature make for the destruction of the bacillus, the action of *direct sunlight* is by far the most important. As Koch showed many years ago, the bacilli can resist diffused daylight for some days, but are rapidly killed (sometimes within a few minutes) by the direct action of the sun's rays.

Concerning *antiseptics* it may be stated that carbolic acid (1 in 20) directly applied to the bacilli destroys them within one minute. It has been further shown that many substances can inhibit in culture the growth of the organism, and among these cyanide of gold is one of the most effective. No antiseptic, however, has as yet been found capable of arresting tuberculosis when injected into animals already suffering from the disease.

Experimental Tuberculosis.—The final proof that the bacillus discovered by Koch was the actual cause of the disease was effected by means of inoculation-experiments. Koch showed conclusively that the micro-organism, whether isolated from tubercular lesions of the lung or joints, or from scrofulous glands, or from lupus, was capable, even after numerous subcultures, of producing tuberculosis in susceptible animals, and that in the lesions so produced the bacillus could again be demonstrated and re-obtained in pure culture. These original observations have met with universal acceptance, thus proving indubitably the causal connection of the micro-organism with the disease.

Of the animals which may be used for such experiments, or for demonstrating the presence or absence of bacilli in suspected material, the one most usually chosen is the *guinea-pig*, for although spontaneous tuberculosis occurs but rarely in this animal, to inoculation it is extremely sensitive. If the tuberculous material be injected *subcutaneously into the groin*, the site generally chosen, the course of events noted is usually as follows. After about a week a local swelling appears at the seat of inoculation. Later the surface over this breaks down and gives entrance to a small cavity filled with caseous material. About this time also the inguinal glands become swollen and palpable. The animal then commences to emaciate, and finally dies at a period varying from six weeks to three or even six months from the date of inoculation, the exact time depending upon the dose of the virus injected, and in part upon individual differences in susceptibility. After death enlargement and caseation of the inguinal and lumbar glands will be found, tuberculosis of the liver and spleen, and finally, if the animal survive long enough, tubercular infiltration of the bronchial and posterior mediastinal glands, and of the lungs. In the guinea-pig it may be noticed the kidney is never attacked, though in the cat disease of this organ is frequently seen.

Such then are the classical appearances presented

in a guinea-pig inoculated subcutaneously in the groin, appearances which may be seen at any time in almost any laboratory, the method being now constantly employed for diagnostic purposes in connection with suspected sputum, and more especially suspected urine.

Other methods of inoculation, however, and indeed other animals, may be used instead of the preceding. Thus in the rabbit *intravenous injection* of the bacilli has been often practised, while inoculation into the *anterior chamber of the eye*, a favourite proceeding with the earlier observers (Cohnheim and Salomonsen) since the resulting tuberculosis could be easily observed, may in some cases be of value. Again, the virus may be injected directly into the *peritoneal cavity*, a method which, in the guinea-pig, offers most trustworthy results.

The Toxins of the Tubercle-Bacillus.—It was shown by Koch that the subcutaneous injection of *dead* tubercle-bacilli gave rise in the guinea-pig merely to a local abscess. In 1891, however, Prudden and Hopendyl made the unexpected discovery that if intravenous injection be resorted to (in the rabbit), after a definite period tuberculosis develops in the lungs, the tubercles thus formed resembling most closely those produced by the living organisms. They were found to contain giant-cells, epithelioid cells and bacilli, but caseation, though sometimes present, was not nearly so marked a feature as usual. In addition they manifested no tendency to spread, since no multiplication of the bacilli contained within them could take place. These observations have been confirmed by Strauss and Gamaleia, and from them a very important consequence follows. We are led, namely, to admit that *the tubercular poison is chiefly intracellular*, that it remains in great part at least within the body of the bacillus, and even after death but very slowly diffuses therefrom.

Concerning the exact chemical nature of the toxin, or toxins, but little can as yet be said. Koch's original '*Tuberculin*,' which consists of a glycerine-broth culture of the micro-organism evaporated at a temperature of $100^{\circ}\text{C}.$ down to one-tenth of its original bulk, and then filtered through porcelain, obviously (from its action upon the animal body) contains certain poisonous substances derived from the bacillus. This liquid was analysed by Hunter, and found to contain various albumoses, as well as alkaloids, salts, and extractives. Of these the albumoses were shown by Kühne to be each of them capable of producing in tuberculous guinea-pigs the typical tuberculin-reaction, and it at first seemed as though they themselves constituted the true tubercular poison. But further observations showed that if the bacillus be grown in a proteid-free medium, the fluid after cultivation still possesses the properties of tuberculin, though it contains no albumoses, nor any proteid matter at all. It results, therefore, that these bodies are not themselves the toxin, but that the latter merely attaches itself to them. But beyond this negative fact our knowledge of the subject does not at present go.

Spontaneous Tuberculosis in Animals.—

Tuberculosis, as we have seen, may be communicated experimentally and with great ease to numerous animals. In addition to this the disease occurs spontaneously in many species, and is especially frequent among those which are brought much in contact with man himself. Thus it is very often

met with both in *cows* (Bovine Tuberculosis; *Perlsucht*; *Pommelière*), and also in *pigs*. It is not uncommon in the *cat* and *dog*, and it occurs, though but rarely, in the *horse*, the *sheep*, the *rabbit*, and the *guinea-pig*. Among *monkeys* in captivity, again, it is often seen. Lastly it is a very well known disease in *birds*, and attacks fowls with especial frequency. The following figures giving the exact incidence of the disease as observed at the slaughter-houses of Copenhagen during the four years 1890-1893 may be quoted from the Report of the Royal Commission on Tuberculosis, 1895: Of 132,294 oxen and cows examined, 17.7 per cent. were found to be tuberculous; of 8,292 swine, 15.3 per cent.; of 185,765 calves, 2 per cent.; and of 337,014 sheep, only .0003 per cent. The frequency of tuberculosis in pigs is here probably somewhat exaggerated, but the figures indicate sufficiently how common the disease is in this class of animal, and how very common it is also in cows.

For our present purpose it will not be necessary to describe in detail the lesions which characterise the disease in the various classes of animals. It may be said, however, that in *cows* the lungs and pleura are most often attacked, and on the latter the tuberculous masses often attain a large size, and are frequently penedunculated. They show a special tendency to calcify. The *udder* is rarely affected primarily, but in some 2.3 per cent. of cases it is invaded during the course of the disease.

In *pigs* the stress of the disease falls upon the alimentary canal, ulceration of the intestine, with secondary invasion of the other abdominal organs, being very common. The tubercular products, as a rule, undergo early caseation. Not uncommonly, however, in the pig, the virus enters through the tonsils, and the submaxillary and cervical glands become swollen and tuberculous. To this form of the disease the name 'scrofula of pigs' is given, and it throws a striking light upon the pathology of the similar affection occurring in the child.

In the *horse*, when the disease occurs, the alimentary canal, the abdominal glands, the peritoneum, liver, and spleen, are chiefly affected, the glands sometimes being converted into enormous tumours (Strauss). In some cases, however, the lungs are primarily diseased. In the *monkey*, lastly, in which the lungs are for the most part attacked, the disease is characterised by the rapid breaking down of the caseous patches into areas of semipurulent material.

Relation of the Human Tubercle-Bacillus to that of Bovine and Avian Tuberculosis, and to other Acid-fast Organisms.

1. *Bovine Tuberculosis*.—The frequent occurrence of tuberculosis in other animals besides man renders the question as to the identity of the micro-organisms found one of great importance. It has hitherto been accepted that, in spite of certain slight morphological differences and slight variations in virulence, the human tubercle-bacillus was identical with that found in other mammalia, and hence that the disease could be conveyed from animals, and especially cattle, to man, and that the reverse could occur. At the recent British Congress on Tuberculosis (July, 1901) this view was combated by Dr. Koch, who showed that, though the human bacillus can produce tuberculosis in rabbits and guinea-pigs, just like the bacillus from bovine sources, yet it differs from the latter in being in-

capable of generating the disease in the higher mammals, such as cattle, pigs, and sheep. He concluded, therefore, that in spite of their extraordinary superficial similarity the bacilli are really different, and that the bovine variety is not, as a rule, a source of danger to man. This view, it must be stated, has the support of Virchow, but does not commend itself to the majority of scientific observers. Further observation and experiment can, however, alone decide the question. Meanwhile, it will be well still to regard the bacilli from the two sources as really identical (and therefore each of them dangerous to man), though *temporarily* somewhat altered as the result of their different environment. (For further consideration of this important question, see paragraphs on 'Channels of Infection,' pp. 1751, 1752.)

2. *Avian Tuberculosis*.—As regards the bacillus of *Avian Tuberculosis* the question is more simple. The latter is identical in appearance and in staining properties with the bacillus derived from human sources, but differs obviously in its cultures, and also in the effect of inoculation. The hen, for example, is extremely sensitive to the avian stock, but proves refractory to the human variety. In the dog, on the other hand, the reverse is the case. There can be no doubt, therefore, as to the essential differences now existing between the two varieties of bacilli, and although these differences may be greatly diminished experimentally, as Nocard has most ingeniously shown, the conditions requisite are never likely to occur during life, and consequently the possibility of the human subject acquiring tuberculosis through the avian disease may be neglected.

3. *Other Acid-fast Organisms*.—The relation of the true tubercle-bacillus to the other acid-fast organisms, which have recently been discovered, forms a new and very difficult problem. These organisms, of which the best known are the *Timothy bacillus* and the *Grass-bacillus*, were first discovered by Moëller on certain grasses (notably the Timothy Grass), having a wide distribution in nature, and extensively used as fodder. Micro-organisms belonging to the same group have since been found in the dung of horses, goats, pigs, and especially mules (*Mistbacillus*), frequently in butter, sometimes in milk, and sometimes also in the urine, fæces, and nasal secretion of healthy people.

Morphologically and in staining properties these all exactly resemble the tubercle-bacillus, and on inoculation into guinea-pigs certain of them, notably the Timothy and Grass-bacillus, produce changes which both macro- and microscopically are quite indistinguishable from true tuberculosis. On cultivation, however, differences at once become apparent, the tubercle-like organisms growing more rapidly than the true tubercle-bacilli, and showing also other points of difference. It is evident, therefore, that these micro-organisms are not tubercle-bacilli, though they are very closely allied to them.

What the exact relationship may be we cannot as yet definitely say, but it should be noticed that they bear exactly the same relation to the tubercle-bacillus that the members of the Coli-family do to the typhoid-bacillus; and just as we have reason to believe that the typhoid-bacillus is but the most highly specialised member of this great group, all the members of which are derived from a common ancestor, so it is possible that the same obtains as

regards the tubercle-bacillus and its allies. On this view the various members of the group would form separate and distinct species, and no conversion of the Timothy bacillus into the tubercle-bacillus need be expected. Such a view, however, does not exclude the possibility of these tubercle-like organisms possessing some pathogenic power against the human subject, but to what degree such power, should it be shown to exist, may extend, we have as yet no knowledge. In the single instance in which so far a member of the group (the Timothy bacillus) has been inoculated into man, a local papule of inflammatory nature alone resulted (Lubarsch). But too much stress must not be laid upon a single experiment.

MORBID ANATOMY.—The pathological changes produced in man by the action of the tubercle-bacillus present in certain organs, and in the lungs especially, appearances which to the naked eye often differ markedly from one another. Thus miliary tuberculosis, chronic phthisis, and fibroid tuberculosis of the lungs are at first sight sufficiently distinct. A more careful examination, however, shows that their fundamental constitution is the same, and that their distinctive appearances are due to the different ages of the lesions and the varying changes which the latter have undergone.

The Miliary Tubercle.—The fundamental lesion resulting from the presence in the body of the bacillus is the so-called 'Miliary Tubercle.' In its most characteristic form this consists of a central giant-cell, surrounded by a varying number of epithelioid cells, these again being enclosed by a zone of ordinary leucocytes. The whole is non-vascular. Such tubercles are invisible to the naked eye, but are easily recognised with the microscope. They may be found, often in large numbers, in the liver in cases of general miliary tuberculosis.

As a rule, however, the tubercles do not remain isolated. Owing to the multiplication of the bacilli further tubercles develop around the one first formed, and the miliary tubercle, such as we know it in the *post-mortem* room, results. In appearance this presents the form of a small round body varying in size from .1 mm. up to 2 or 3 mm. in diameter. When first formed it is greyish in colour, and translucent, but as retrogressive changes set in, it becomes yellow and opaque. Under the microscope each individual tubercle comprised within it will be found to show with more or less distinctness the appearances already referred to—a central giant-cell, a surrounding zone of epithelioid cells, and most externally a layer of leucocytes. Of these the most characteristic, though not the most important, is the *Giant-Cell*. This body, which may attain a diameter of .3 mm., is irregular in shape and presents at its periphery numerous processes. These penetrate among the surrounding epithelioid cells and thus become lost to view. The substance of the cell is composed of slightly granular protoplasm, and is found to contain very numerous nuclei, sometimes even as many as thirty or more being present. These are sometimes scattered irregularly through the cell, but are more often arranged in a ring round the margin or grouped together near one extremity. The *Epithelioid Cells*, which form the second layer, are much smaller than the giant-cell, which they surround. They are somewhat irregular in outline and contain a granular protoplasm, within which one or more nuclei are embedded. The *Leucocytes* lastly, which form the peripheral zone,

present nothing characteristic. They are smaller than the epithelioid cells and seem to be nothing more than ordinary leucocytes, or inflammatory cells, attracted by chemiotaxis to the seat of the disease. If sections be prepared and suitably stained, tubercle-bacilli will be found scattered among the cells of the tubercle, or perhaps lying within the giant-cells, but in the human subject their occurrence within the latter is somewhat rare. As a rule also, the micro-organisms seen are not so numerous as might be expected. The tissue surrounding the tubercle is commonly hyperæmic, but the new formation itself is non-vascular.

Such then are the characteristic appearances of what may be spoken of as a typical tubercle. It must be clearly understood, however, that *giant-cell-formation, usually regarded as so characteristic of the disease, does not by any means always occur*. The essential feature of the tubercle, as will be shown, is the production of epithelioid cells, and if caseation takes place very rapidly, as in the so-called 'Caseous Pneumonia,' giant-cells will be but rarely found. On the other hand, if the tubercle-bacilli in question be not very virulent, or if the organism be somewhat resistant, then giant-cell-formation becomes a more marked feature, while on the contrary the invasion of leucocytes to form the circumferential zone is correspondingly less obvious, or may be even completely absent.

Histogenesis of Tubercle.—We may now consider more exactly the manner in which the tubercle comes to be developed. This question has long been debated between the rival schools of Germany and France, Baumgarten, on the one hand, holding that epithelioid cells and giant-cells originate from the *fixed cells* of the tissues (endo- and epithelial cells, and connective tissue cells); Metchnikoff, on the other hand, maintaining that they are derived from the *wandering cells* or leucocytes. The question at last seems finally settled in favour of the former. Briefly, the evidence which Baumgarten has brought forward is as follows: He produced tuberculosis in the iris (as well as in other organs) in rabbits, and examined with great care the changes thus induced. Nothing could be noticed for about a week, but at the end of this time he found that the bacilli injected were in very close apposition to, or even within, certain of the connective-tissue and epithelial cells of the tissue. The cells so affected were noticed also to be swelling up and to be assuming the appearance of epithelioid cells. Karyokinetic changes were also observed in the nuclei, and later cell-division followed. *The primary change therefore resulting from the action of the bacillus was not an influx of leucocytes, but the formation of epithelioid cells from the fixed cells of the tissue.* This fundamental connection of the latter with the epithelioid cells, it may be added here, is supported by the observation of Strauss that in experimental tuberculosis of the liver in rabbits the epithelioid cells often have a highly granular protoplasm, and contain also glycogen, thus testifying strongly to their origin from the hepatic cells. The secondary changes which next followed in Baumgarten's experiments were found to vary somewhat according to the intensity of the virus, and the resisting powers of the tissue. If the virus was very powerful then the invasion of leucocytes took place early, and the tubercle sometimes came to consist almost entirely of these cells. Caseation in such cases rapidly followed, and giant-

cells not uncommonly never made their appearance. If, however, the process was less acute, then giant-cell-formation occurred, and the leucocytic invasion was less marked, or even remained totally absent. *In no case were karyokinetic changes or cell-division observed in the wandering cells, and their relation to the tubercle would therefore seem to be definitely a secondary one.* Concerning the mode of origin of the giant-cells there may be still some difference of opinion. Some observers hold that they are produced by the union of several epithelioid cells, while Baumgarten believes that they result from the enlargement of individual epithelioid cells, and by the division of their nuclei, without the latter process being followed by cell-division. He was, however, unable to demonstrate karyokinesis in the nuclei of the giant-cells, so that, probable as his view is, it cannot be regarded as absolutely proved.

These results, obtained in the iris, were confirmed by similar observations in other organs, and there can be no doubt as to their correctness. *Consequently the true tubercle-cells, both epithelioid and giant-cells, must be in future regarded as originating in the fixed cells of the tissues, under the stimulus afforded by the presence of the bacillus. The share taken by the leucocytes, on the other hand, must be looked upon as quite subsidiary. It is limited to the formation of an inflammatory external zone, and even this under certain circumstances may be entirely absent.*

Retgressive Changes.—The tubercle having originated in the manner thus described does not retain its perfect form for any length of time. Certain degenerative changes soon set in, which we must now proceed to consider. These may be divided into two great classes, according as the degeneration assumes the caseous or the fibroid form. Of the two the former is by far the most common, and it may be added also the most important, seeing that in many cases its occurrence and the further changes which it undergoes are fraught with serious danger to the organism.

1. *Caseation.*—This term dates from the commencement of the last century (Vetter, 1803), and indicates, as the name implies, that the degenerated tubercular matter assumes to the naked eye an appearance somewhat similar to that of cheese. The tubercular deposit, transparent when first formed, is seen after a few weeks, or it may be even earlier, to show in its centre, when caseation is commencing, minute opaque spots. Gradually these spread until at last the whole patch assumes an opaque aspect, and becomes yellowish-white in colour. Under the microscope these changes are seen to be due to the death and destruction of the tubercle-cells. The protoplasm of these latter becomes homogeneous or slightly granular, while the outline of the cell first grows indistinct, and later gradually fades away. A similar process also affects the nuclei. As a result, therefore, the caseated mass assumes, when examined microscopically, a faintly granular appearance, in which no definite cell-elements can be made out. The whole stains feebly with ordinary dyes. Within the patch tubercle-bacilli may sometimes be demonstrated by appropriate staining, but unless the process has been very acute, they will usually be few in number. In very many cases, however, experiment upon animals proves the caseated material to be still virulent, though the microscope may have failed to reveal the specific micro-organism.

Such then is caseation which occurs with so extraordinary a frequency in tubercular formations. It would be an error, nevertheless, to believe that it is peculiar to this disease. On the contrary, it occurs in syphilitic gummata, and may also be seen in inflammatory exudations produced by the action of other micro-organisms ('pseudo-tuberculosis'). Concerning the cause which leads to the transformation it may be said that for many years it has been attributed to the non-vascularity of the tubercular nodule. Such an explanation is insufficient, for caseation may occur in most minute tubercles, even the central cells of which could be easily nourished by lymph which had permeated from the periphery. Without doubt, therefore, the change is due to a direct poison emanating from the bacillus, which coagulates and destroys the cells, which its primary irritative action has produced.

The tubercular material having undergone caseation may occasionally remain in this condition for some considerable time. If this occurs the mass nearly always becomes surrounded by a definite capsule of fibrous tissue, which acts as a safeguard to the organism by preventing the further spread of the bacilli. Such a persistence, however, of the unaltered caseous material is by no means common. Far more frequently softening occurs. In this case the cheesy material breaks up into a molecular débris, consisting for the most part of fine fatty and albuminoid granules, which lie bathed in serous fluid. The amount of the latter varies greatly. In some cases only very small quantities may be present, and the softened tubercular mass may be really semi-solid and possess a putty-like consistency, while in other instances, as for example in the case of a psoas abscess, much larger quantities may be present, and the softened cheesy matter thus acquires the characters of thick, curdy 'tubercular pus.' All variations between these two extremes are to be met with.

Another well-known change which may follow upon caseation is *calcification*. When this occurs calcium phosphate and carbonate, but chiefly the former, become deposited within the degenerated material, and by their gradual augmentation eventually convert it into a mortary or stony mass. This change constitutes one of the most perfect methods of natural healing, for whereas the caseous matter remains infective and therefore dangerous to the individual, the calcified mass is harmless, owing to the bacilli contained within it having undergone destruction. Calcification only occurs in those forms of the disease in which the malady runs a chronic course. It is consequently never seen in the guinea-pig, is met with fairly frequently in man, and is often observed in the cow. How early in the course of the disease the change may occur it is impossible to say with any certainty. In the pig, however, it has been observed by Sidney Martin 106 days after infection.

Lastly, it is permissible to believe that sometimes *absorption* of cheesy material may at least in part occur. In the case, for example, of tuberculous glands in the neck, so common in children, it is very probable that in some cases the tubercular matter is thus eliminated.

2. *Fibrosis.*—As already mentioned, fibrosis is the other great change which tubercular products may undergo. In its most perfect form, in which the whole of the tubercular nodule is directly converted into fibrous tissue, it is infinitely rarer than

the caseous degeneration. Not uncommonly, however, it may accompany the latter, as when in large racemose tubercles little areas of caseation are found embedded, as it were, in a matrix of grey fibroid material. In this way the degeneration tends to check the further spread of the disease. Indeed, such formation of fibrous tissue is the ordinary method of spontaneous cure occurring in tubercular disease, while to assist the process in every way is the constant aim of the physician.

MORBID APPEARANCES OF PULMONARY TUBERCULOSIS.—When any organ in the body is attacked by tuberculosis, the resulting changes are fundamentally those that have been just described. Certain variations, however, will be noticed according to the organ or tissue affected.

As pointed out in their recent work by Fowles and Godlee, four sufficiently distinct pathological (and also clinical) varieties of pulmonary tuberculosis may be described, according as the disease assumes the miliary type, or as caseation, fibrosis, or a combination of the two latter with cavity-formation, constitute the chief feature. Of these several varieties a more detailed description now follows, and it will be seen that into one or other group will fall the numerous clinical types of pulmonary tuberculosis which have been from time to time described.

1. *Acute Miliary Tuberculosis of the Lungs.*—In this variety the lungs are found thickly studded throughout with very numerous miliary tubercles. Owing to the fact that death in this variety takes place very speedily, generally within three weeks from the onset of the disease, the tubercles are for the most part still transparent, or only just commencing to caseate. Under the microscope they show the typical appearance of miliary tubercles already described. The pulmonary tissue intervening between the tubercles is in general greatly engorged, but is otherwise healthy. Sometimes, however, there may be tubercular disease of older date at one or other apex. Discrete tubercles similar to those in the lung are not uncommonly found also upon the visceral surface of the pleura, and in many cases are seen as well in other organs of the body.

This variety of the pulmonary disease is always ascribed to the softening of a caseous focus, very commonly situated in a bronchial gland, and to the entrance therefrom of the infective material into the blood-stream. In the majority of cases this is no doubt the true pathology of the process. Where, however, the lungs alone are affected, as is not uncommonly the case, it is possible, the writer believes, that we may be really dealing with an aspiration-process, and not the penetration of bacilli into the circulating blood.

2. *Acute Caseous Tuberculosis of the Lungs.*—**SYNON.** : (among others) '*Acute Phthisis*'; '*Scrofulous Pneumonia*'; and '*Acute Tuberculo-pneumonic Phthisis*.'

This variety of the disease is chiefly characterised by the absence of fibrosis, by the large areas of the lung which become affected by tubercular consolidation, and especially by the rapidity with which the latter undergoes caseation. Two types may be distinguished—the lobar and the lobular.

(a) *The Lobar Type.*—This variety, perhaps most generally termed '*acute pneumonic phthisis*,' is extremely uncommon. In its typical form it attacks a previously healthy lung, but in some cases

'*caseous pneumonia*,' as the condition is often pathologically named, will be found as the terminal event in a case of ordinary chronic phthisis. The disease runs a rapid course, and the fatal issue is usually not delayed beyond two or three months, and may even take place earlier. After death the lungs present a very typical appearance. The affected lobe (in a primary case, as a rule, the upper lobe), or it may be the whole lung, is found to be in a condition of consolidation. This consolidation is not, however, like that of pneumonia, but presents a yellowish-white opaque granular appearance, due to the fact that the inflammatory products have already undergone caseation, while the capillaries also have been to a great extent obliterated. Should the patient have died early these will be all the changes noted. If, on the other hand, the case has been less acute, then some softening may have taken place, and one or more ragged, irregular cavities will be found.

Under the microscope the alveoli of the consolidated lung will be found in the early stages to be crowded with leucocytes and cells of an epithelioid type, embedded in a fine fibrin-network. When, however, caseation is well established, then no cell-elements remain visible, but the section presents merely a granular homogeneous appearance. By appropriate staining innumerable tubercle-bacilli may often be demonstrated. Owing to the acuteness and severity of the process giant-cells are but rarely seen.

This variety of the disease is apparently produced by the inhalation into one lobe, or it may be the whole of one lung, of numerous and very virulent bacilli, in a patient whose resistance is not great. As a result the tubercular process set up by each original bacillus spreads with great rapidity. Consolidation follows, and caseation rapidly ensues.

(b) *The Lobular or Broncho-pneumonic Type.*—This type, though much less rare than the preceding one, is still, in adults, at least, far from common. At the Brompton Hospital it constitutes, according to Kingston Fowler's statistics, 2·4 per cent. of all cases of pulmonary tuberculosis. In children, however, following after measles or whooping-cough, it is much more frequent.

In essentials it resembles the lobar form. The disease, that is to say, runs the same rapid course, and is characterised by the same early caseation of the tubercular products. The chief difference between the varieties lies in the fact, that whereas in the former entire lobes are affected, in this case the affected areas are smaller, so that the disease assumes the broncho-pneumonic type. Yellowish caseated areas, some as large as a shilling, are thus seen scattered through the lungs, while the intervening tissue remains comparatively healthy. Early softening also is rather more prone to occur, so that ragged irregular cavities are frequently met with. Not uncommonly also the necrotic process spreads to the pleura, and a pneumothorax results. It is to this variety of the disease that the names '*Galloping Consumption*,' or '*Phthisis Florida*,' have not infrequently been applied.

3. *Fibroid Tuberculosis of the Lungs.*—This in its perfect form is an extremely rare disease, and must not be confounded with '*Fibroid Phthisis*,' which is of comparatively common occurrence. Fibroid tuberculosis indicates a disease of the lungs in which tubercles become deposited at their apices, just as in an ordinary case of phthisis. These,

however, do not undergo caseation, but from the first take on the fibroid change, and, as a result, become converted into firm deeply pigmented bodies. These sometimes remain discrete, but sometimes become aggregated into little racemose masses, to which the name 'Carswell's Grapes' has been applied. Occasionally, also, larger areas of fibrosis may be produced. The lung-tissue in the neighbourhood of the tubercles undergoes, as a rule, secondary emphysema; hence it is often difficult or impossible to obtain during life definite physical signs of the disease. It will be seen from what has been said that this variety of pulmonary tuberculosis differs much from 'Fibroid Phthisis,' since in the latter, though fibrosis is always present, cavity-formation and falling in of the chest-wall are both also well-marked features of the disease.

4. *Fibro-caseous Tuberculosis, or ordinary Chronic Pulmonary Phthisis.*—The morbid lesions of this, the commonest variety of pulmonary tuberculosis, differ somewhat according to the chronicity or relative acuteness of the process, but in all cases we find caseation, followed by softening and cavity-formation, and in all cases, also, more or less fibrosis. The morbid appearances, in fact, show a combination of the lesions met with in the two preceding varieties of the disease, with the additional fact that cavity-formation always constitutes a marked feature of the process.

The disease commences in nearly every case near the apex of the upper lobe, where the primary tubercles are deposited. These are at first miliary in character, but as they enlarge they not uncommonly unite, and little 'racemose' masses are formed. These again join others, and finally largish areas of tubercular disease are produced. But meanwhile degenerative changes have set in. Caseation first occurs, followed later by softening. Finally, a communication with a bronchus is established, the liquefied caseous matter, containing numerous tubercle-bacilli, is coughed up in the sputum, and a cavity results. Owing, however, to the somewhat chronic nature of the process, some fibrosis is also seen. Areas of firm greyish fibroid material form around the cavities, and in the partly caseating racemose patches some fibrosis is often also to be made out, especially if Van Giessen's method of staining be employed. In this way the disease may be finally arrested at this early stage, and if examined some years later, but little will be seen beyond a puckered fibrous spot, containing a contracted cavity, with, perhaps, in addition, a small caseous or calcareous mass embedded in a firm capsule of fibrous tissue. Not uncommonly, also, the surrounding lung is emphysematous.

More frequently, however, the disease does not become arrested, but continues to spread, partly by means of the lymphatics, and partly through aspiration of the infected sputum into bronchi hitherto unattacked. In this way the disease invades first the apex of the corresponding lower lobe, next the apex of the opposite lung, and finally the opposite lower lobe. The pathological changes continue to be the same. Whenever the patient's strength ebbs, then we have fresh formation of racemose tubercles, the junction of these, caseation and formation of cavities; when his resistance increases, renewed formation of fibroid material, and an attempt to arrest the disease. Finally, however, unless complete arrest occurs, the patient succumbs to his disease. The lungs are then found to present nume-

rous cavities, of old or recent formation, while the remainder of the lung-tissue is strewn with small, greyish-yellow caseating racemose tubercles, or it may be patches of larger size. The lung-tissue intervening between these is often natural or simply engorged, but sometimes it may show a terminal miliary tuberculosis, or possibly be in a condition of pneumonia, either of the simple broncho-pneumonic variety, or, much more rarely, of the caseous type. In some cases the fibrosis, which to some extent is always present, becomes very marked, and as a result the excavated lung becomes contracted, the chest is drawn over, the side falls in, and we have the clinical picture of '*Fibroid Phthisis*,' as drawn by Sir Andrew Clark.

The cavities which form so conspicuous a feature of this type of tuberculosis are described elsewhere. See CAVITY.

CHANGES IN OTHER ORGANS FOLLOWING PULMONARY TUBERCULOSIS. — In chronic pulmonary phthisis the tubercular lesions are very rarely limited to the lungs. Thus the *pleura*, as a rule, becomes invaded and inflamed, and its layers adherent, while later it may become greatly thickened. Occasionally, however, adhesions fail, and then, should the pleura necrose, a pneumothorax results.

Again, the *trachea* and *bronchi* are very often affected. In all cases they are injected, and it is not uncommon also to find their mucous membrane studded with recent miliary tubercles, or shallow ulcers resulting from the caseation and softening of the latter. In rare cases the tubercular ulceration may be more extensive, and the cartilaginous rings of the trachea may be exposed. In 47 per cent. of cases the *larynx* also shows at the time of death definite signs of tubercular disease.

Very common, too, is some change in the *bronchial glands*. These are nearly always swollen and enlarged, and may show in their substance scattered miliary tubercles. Sometimes, also, small patches of caseation or small calcareous masses may be found in them. It is very rare, however, in chronic pulmonary tuberculosis, in spite of statements to the contrary, to find the bronchial glands in a general state of caseation.

In a very large proportion of cases (77·4 per cent., according to the records of the Brompton Hospital) *tubercular ulceration of the intestine* may be observed, produced by the careless swallowing of the infected sputum. The ulceration is most frequently situated in the ileum and cæcum, but the disease may extend as high as the duodenum; and in some cases spread downwards even as far as the rectum and anus. The appendix is very commonly attacked, but the stomach is only most rarely affected. The ulcers are for the most part circular, but, unless they originate in a Peyer's patch, show some tendency to spread transversely. Their appearance is sufficiently characteristic. The heaped up thickened edge and nodular floor suggest at once their tubercular nature, while the presence of numerous miliary tubercles on their peritoneal surface serves as a rule to confirm the diagnosis.

As a result of this ulceration of the alimentary canal, but sometimes even apart from it, we very commonly find tubercular disease of the *mesenteric glands*. These are seen to be enlarged, and on section are found to be in a condition of caseation. In rarer cases also irregular nodules of calcareous degeneration may be observed in them. *Peritonitis* again is another sequel which sometimes results

from the intestinal disease. In general it is of tubercular nature, and spreads from the peritoneal covering of the ulcers, which as already pointed out are the first portions of the serous membrane to be affected. As a result the intestines become adherent and matted together, while the omentum becomes rolled up and filled with tubercular deposits. Ascites is uncommon. Much more rarely the peritonitis is of the septic variety, and arises from perforation of one of the ulcers, or is due to the passage of micro-organisms through the greatly thinned floor without definite rupture having taken place.

Tubercular Meningitis is another but much less common complication of chronic pulmonary tuberculosis. According to Dr. Kingston Fowler's statistics only 3·3 per cent. of cases are terminated in this way. In even a smaller proportion of cases (2·7 per cent.) do we find tubercular disease of the male and female *generative organs*, while only very rarely indeed are tubercular *endo- or pericarditis* to be observed. The *spleen* is very often found softened, and occasionally shows tubercular deposits. The *kidneys* are commonly fatty, and it is not rare to find in them a few scattered miliary tubercles, the result of the entrance into the blood-stream of a very few micro-organisms. Occasionally also more advanced tubercular disease may be present. The liver is frequently in a condition of fatty infiltration, but beyond this is generally unaffected.

One other complication often met with in cases of phthisis of some considerable duration remains to be mentioned, namely, *amyloid degeneration*. This change occurs in about 11 per cent. of all cases of the disease. The exact proportion in which the various organs are affected is stated by Kingston Fowler, from the Brompton Hospital Records, to be as follows: spleen 11·1 per cent.; liver and kidneys each 6·1 per cent.; intestines 5·3 per cent.; and stomach 1·8 per cent.

CHANNELS OF INFECTION.—Now that we have studied the chief peculiarities of the tubercle-bacillus, and of the lesions produced by it, we are in a position to consider what are the channels by means of which it gains access to the human body, and thus originates the disease. Three methods of infection exist: (1) Inoculation; (2) Inhalation; (3) Ingestion; and of these the second is by far the most important.

1. Inoculation. — Only very rarely can tuberculosis in man be attributed to this cause. Laennec was himself, however, infected in this manner, and similar cases are recorded from time to time. At the Brompton Hospital the two following cases have quite recently occurred. The first was a nurse who wounded her left wrist with a spittoon containing virulent sputum. A local tuberculosis followed. This was excised four months later by Mr. Boyd, when complete recovery ensued. The second case occurred in the person of the museum-attendant (H. M. aged 62), who, while staining the sputa, ran a pen covered with virulent expectoration into the flexor tendon-sheath of the middle finger of the right hand. Tubercular teno-synovitis resulted, but the trouble was arrested, and the patient cured by the timely scraping out of the disease within two months of the primary infection. These cases, though very exceptional, prove that this method of infection does exist. They show also the happy effect of early operation, especially when we remember that unless so treated the disease spreads

from its primary seat to the glands and lymphatics, and then to the lungs and other organs of the body.

2. Inhalation. — We now pass to the great means whereby tuberculosis is spread, namely, the inhalation of dried sputum.

In considering this question we must first remember that the sputum, though by far the most important, is not the only means whereby the tubercle-bacilli may obtain exit from the human body. The breath and sweat of the consumptive patient never indeed contain the specific micro-organisms, and are consequently innocuous, but when the genito-urinary organs or the alimentary canal are diseased, the urine and faeces contain appreciable numbers of bacilli, and thus become possible sources of contagion. In view, however, of the high standard of modern sanitation the inhalation of dried particles of these excretions can only most rarely occur, and infection through these channels may therefore for practical purposes be neglected. Far otherwise is the case with the *sputum*. In the first place we must bear in mind the enormous numbers of bacilli which a consumptive patient may expectorate for weeks and months together. This has been computed by Nuttall as sometimes reaching the enormous total of four thousand millions a day or even higher. In the sputum, therefore, we have obviously a grave source of danger, even though a certain number of the micro-organisms may be really dead, as has been suggested. It can be, however, only rarely that inhalation of the liquid viscid sputum occurs, though occasionally in speaking or coughing minute portions may be scattered abroad and thus inhaled. The serious risk does not undoubtedly arise until the sputum has been allowed to dry, whether on handkerchiefs, on the floor, or elsewhere. Such desiccation, even when accompanied by some putrefaction, has been shown to have but little effect upon the bacillus, which retains its virulence under these conditions for many months, while its ability to pass into the air in the form of dust is at once rendered easy. Remembering the extreme frequency of primary tuberculosis of the lungs, and bearing in mind that a similar disease in animals has often been produced by causing them to inhale for a short time only dry and pulverised sputum, there can be no reasonable doubt that the disease in man originates, in the vast majority of cases at least, in a similar way.

It may be asked, however, if these facts are so, how it is, not that one-eleventh of our race die of phthisis, but that we do not all perish from it. Probably the chief explanation is to be found in the destructive action of sunlight, owing to which *tubercle-bacilli are not ubiquitous*, as might very reasonably be imagined after what has been said. This has been very clearly demonstrated by Cornet, who, using the animal test, examined for the presence of tubercle-bacilli 147 samples of dust from various sources. His observations proved that while virulent bacilli could be found in the dust from wards and rooms inhabited by phthisical patients, they could not be found in similar situations which had not been exposed to contamination. More important still bacilli were never discovered in dust taken from the streets, though 12 different specimens were examined.

Another very important fact to remember in this connection is that *those who actually harbour the*

bacilli do not by any means always acquire the disease. Thus virulent bacilli have been found by Strauss in the nasal cavities of people in perfect health, and who have shown no symptom of the disease even after this discovery. In truth, as with so many other infectious diseases, we must not lose sight of the fact that *the bacillus is but one factor in the causation of the disease*, and that to produce the malady other influences, often of a debilitating nature, are also required. In the case of pulmonary tuberculosis these *predisposing causes* may be briefly mentioned. Any cause, for example, which produces catarrh of the bronchi, such as measles, whooping cough, dusty occupations, dampness of the house and soil, will necessarily favour the onset of the disease. The effect of the bronchitis, however produced, will be to enfeeble, or even to annihilate for a time, the action of the ciliary epithelium in the respiratory tract. As a consequence, the bacillus, should it now gain admission, would not be expelled at once, but would acquire time and opportunity for development. Again alcoholism, insufficient food, prolonged lactation, and mental worry, all exercise a debilitating influence upon the patient, and by weakening the local and general protective forces of the body predispose towards the disease. In a similar way, too, act diabetes and influenza, though in many cases the local action of the latter upon the respiratory tract is also of importance. Lastly, heredity is without doubt a factor which cannot be neglected. *See below.*

Such then are the chief causes which favour the onset of the disease, and the important part which they play in giving rise to it should never be overlooked.

3. *Ingestion.*—That tuberculosis may be conveyed from animals to man has been accepted for many years by the scientific world. The opinion is based upon considerations already stated; and regulations have accordingly been made in all civilised countries to control in some measure at least the sale of meat and milk derived from infected animals.

These opinions have been, however, again placed upon their trial by the pronouncement of Dr. Koch at the recent British Congress of Tuberculosis (July 23, 1901). Dr. Koch, as already stated, has observed that though tuberculosis can be communicated to certain small animals (rabbits, guinea-pigs, &c.) by tubercle-bacilli derived from human sources, yet in the case of the higher mammals, such as cattle, pigs, asses, sheep, and goats, this is not possible, though the latter readily succumb if bacilli owning a bovine origin are used. He concludes therefore that 'human tuberculosis differs from bovine and cannot be transmitted to cattle.' Relying also upon certain German statistics which apparently show the rarity in children of primary intestinal tuberculosis, he accepts also the converse of this proposition, namely, that 'man is hardly if at all susceptible to the bovine disease, and that consequently infection of human beings through tuberculous meat or milk can only most rarely occur, and need not therefore be guarded against.'

In the present article it is not possible to criticise fully these important dicta. The following facts, however, may be noted. In the first place primary intestinal tuberculosis in children is not at all uncommon in England and Scotland, whatever may be the case in Germany. Thus recent statistics

dealing with tuberculosis in children (Still and Shennan) show that out of 547 cases in nearly 30 per cent. primary infection would appear to have taken place through the alimentary canal. Secondly, several cases are on record in which veterinary surgeons and others have accidentally inoculated themselves with bovine virus, and yet the disease has run its course in them, sometimes to a fatal termination, just as though the bacilli had been derived from the human subject. From these facts it is clear that the bovine organism is capable in some cases at least of producing tuberculosis in man. Hence it is possible that the primary tuberculosis of the alimentary canal so common in children in this country may really owe its origin to a bovine source. At the same time it may be pointed out that from their habit of crawling upon floors, dirtying their fingers, and immediately sucking them, young children in infected houses have ample opportunity of swallowing human tubercle-bacilli, and that their intestinal disease may conceivably originate in this way.

On the whole the provisional conclusion at which we must arrive is that though the danger of infection through food has no doubt been exaggerated, yet to some extent, for the present at least, it must be held to exist. Consequently, until it be definitely proved that precautions are unnecessary, we must continue to protect ourselves as heretofore.

Of the two articles of diet, namely, *meat* and *milk*, which must thus engage our attention, the latter is by far the more important. This arises from the fact that tubercular disease of the cow's udder, which is chiefly responsible for the presence of the bacilli in the milk, is by no means uncommon (according to MacFadyean probably 2 per cent. of all cows in this country are so affected), that the clinical symptoms of the disease are often most obscure during life, and lastly, that the milk itself may be for long quite unaffected to the naked eye, even though it be really swarming with tubercle-bacilli. The dangers therefore to be associated with cow's milk are subtle as well as real. They may, however, be entirely avoided by boiling the milk and thus destroying the bacilli, a precaution which under no circumstances should ever be omitted in any household.

With regard to *meat* the danger is less marked, even though perhaps 30 per cent. of all cows in this country be tuberculous (MacFadyean). The muscles, which constitute the 'meat' of the butcher, are but rarely affected with tubercular disease, and superficial contamination from other parts, acquired during the processes of 'flaying' and 'dressing,' is for practical purposes obviated in nearly all cases by the subsequent cooking.

Influence of Heredity.—We may now pass to consider the great question of heredity, which comes into play whatever be the exact means whereby the bacillus gains access to the organism. It is matter of common knowledge that tuberculosis is far more frequent in certain families than in others, and that consequently among those attacked by it a family history of the disease may very commonly be obtained. Quain, for example, found that among the patients attending the Brompton Hospital 25 per cent. gave a history of the disease in one or both parents (*First Report of the Brompton Hospital*). More important, however, are the observations of Theodore Williams, who, dealing entirely with the

upper classes, in whom, owing to their habits, the chances of infection would be somewhat less, was able to trace among 1,000 cases a history of the disease in one or both parents in 12 per cent. and in some near relative in 48 per cent. of the cases.

How are these facts to be explained? Before the discovery of the bacillus, and the acceptance of the infectious nature of the disease, they presented no difficulty; the tendency to the mysterious malady was hereditary, and that explained all. We now know, however, that many of the cases thought to be hereditary must have been in reality produced by infection, and that, in part at least, the reason why tuberculosis runs in families is that, when once introduced, the chance in favour of other members becoming infected is thereby greatly increased. At the present moment, indeed, among some there is a tendency to believe that this increased exposure to infection is sufficient to account for all the facts, and that tuberculosis is not hereditary at all. This, however, can hardly be maintained, for though it is true that many so-called 'hereditary' cases are really the result of 'family infection,' yet instances are not wanting in which the latter danger has been obviated, and in which, nevertheless, first one and then another member of the family has fallen a victim to the disease. Again, it is admitted that other infectious diseases, such as typhoid fever, are prone to attack certain families rather than others; hence there is no improbability in believing that the same may be true also of tuberculosis.

Accepting, then, that heredity is a factor of some importance in the ætiology of the disease, we must consider how it may be explained. Is it due, as Koch first suggested, to the inheritance of certain peculiarities in the tissues favourable to the growth of the bacillus; or is it due, as Baumgarten has so forcibly argued, to the direct infection of the ovum through the placenta, or more rarely by means of the spermatozoa, that is to say, to the direct inheritance of the germ itself? In favour of the latter view, which presupposes, be it noted, a latent period often of many years before the microbe takes on active growth—a sufficiently startling assumption—but little evidence can be brought. On the other hand, the extreme rarity of congenital tuberculosis, and the fact that tubercle-bacilli are in nearly all cases completely absent from the tissues of the fetus, originating from a tuberculous mother, militate most strongly against it.

Until, therefore, more definite proof is forthcoming we must believe that *the hereditary factor in the disease, which cannot be gainsaid, though it doubtless does not exist to the extent once imagined, is the result of a special idiosyncrasy of the tissues, whereby in certain families they become more than usually favourable to the development of the tubercle-bacillus.*

DIAGNOSIS.—The differential diagnosis of the various forms of tubercular disease need not be considered in this article. One or two points of a more general character may, however, be referred to. They may be considered under the following headings:—

1. *Pseudo-Tuberculosis.*—It has been shown by recent research that *the power of forming tubercles, quite indistinguishable to the naked eye from those produced by the bacillus of Koch, is by no means limited to the latter micro-organism or its allies.* On the contrary, it is now recognised that even in man typical tubercles may be also produced

by mechanical particles (seeds, fragments of oyster-shell, &c.), by various forms of fungi (*Aspergillus fumigatus*, &c.), and by various micro-organisms totally distinct from the tubercle-bacillus, and among which the 'Bacillus of Pfeiffer' may be especially mentioned. To these various granulomata, for the most part infective in nature, the inclusive name 'pseudo-tuberculosis' has by some been given. That such forms of disease are to be met with it is important to remember, for, apart from the fact that they are probably more common than is generally supposed, it follows from their existence that the mere development of the naked-eye appearances of tuberculosis in an animal after an inoculation made for diagnostic purposes is not *conclusive* evidence that the bacillus of Koch was present in the suspected matter. To be certain, the caseous material found must also be stained for tubercle-bacilli, and, if necessary, cultivations made from it, so that the presence of the specific micro-organism may be thereby conclusively demonstrated. Such a method of examination should therefore be made a routine procedure in all cases.

A further question now presents itself, namely:

2. *What diagnostic value should be placed upon the detection in tissues or in excretions of bacilli which stain like the tubercle-bacillus?*—Until quite recently the matter presented no difficulty. All such organisms were held to be without doubt true tubercle-bacilli, since in this country at least the possibility of leprosy needed only rarely to be considered, while the Smegma-bacillus could be differentiated by certain morphological differences, as well as by its decolouration by alcohol, subsequently to its treatment with acid, a routine method of procedure which should never be omitted in staining for the tubercle-bacillus. See 'Staining Properties,' p. 1743.

The discovery, however, already alluded to (see p. 1743) of acid-fast and tubercle-producing bacilli in nature (Timothy bacillus and Grass-bacillus), and of allied micro-organisms, nineteen in all, in urine, fæces, and nasal secretion of healthy people, in the dung of various animals (*Mistbacillus*), in butter, in milk, and finally in a case of non-tubercular gangrene of the lung, has made it evident that we can no longer rely implicitly upon staining properties for the diagnosis of the tubercle-bacillus. According to Moeller, however, to whom we owe most of our knowledge on this subject, the margin of error thus introduced into clinical work is really a very small one. Consequently, if acid-fast bacilli are found in the sputum, we must, for the present at least, continue to regard them as true tubercle-bacilli, though to obtain absolute proof of their identity resort must be had to *cultures*, the slow growth of Koch's bacillus differentiating it from all allied organisms.

3. *Tuberculin, value of, as a diagnostic agent.*—It was shown by Koch that if a minute dose ('001 grm.) of the original tuberculin (see pp. 1745 and 1754) be injected under the skin of a healthy man no result followed. If, however, the patient were the subject of tubercular disease, then, after about four hours, the temperature began to rise, reaching 102°–104° F., or even higher; the patient felt ill, suffered from cough and vomiting, and might even have a rigor. These symptoms lasted about twelve hours, and then gradually passed away.

Similar differences were also noticed in cattle, and

the method has consequently come into general use among veterinary surgeons for the early diagnosis in these animals of tubercular disease. So accurate indeed is the test that the errors in diagnosis connected with it are only estimated at about 5 per cent. of the total cases injected.

Is the test of equal value in the case of man? This has been doubted by some, who have stated that in cases of cancer and syphilis a similar reaction occurs, but it would seem that if the test be performed with care, the greatest reliance may be placed upon it. To quote a signal instance of its value, we may note the observations of Dr. France among the insane patients at the Claybury Asylum. France injected 75 patients. Of these 20 showed no clinical symptoms of the disease and gave no reaction. Of the 55 suspected cases 45 reacted. Of these 45, 34 subsequently died, and upon 29 of them *post-mortem* examinations were performed. Each of these 29 showed active tuberculosis. Of the 11 remaining patients, still alive at the time of writing, 6 now showed clinically very obvious signs of tubercular disease.

There can be little doubt, therefore, that the drug is a potent aid to diagnosis in difficult cases, and as such its employment may be encouraged. At the present time, indeed, it is in routine use in Osler's wards in the Johns Hopkins Hospital, Baltimore.

4. *The Agglutination-Test.*—In a very numerous series of papers it has recently been contended by Arloing and Courmont of Lyons that the serum of tuberculous patients will agglutinate the tubercle-bacillus, exactly in the same way as the corresponding typhoid serum agglutinates the typhoid bacillus, although as a rule in a far lower degree. In approximately 75 per cent. of cases of pulmonary tuberculosis, and in nearly the same percentage of cases of tubercular disease of bones and joints, they found that the serum of the patient would agglutinate their homogeneous culture of the tubercle-bacillus in a dilution of 1 in 10 or higher. Such a reaction they regard as specific, and almost certainly indicative of the presence in the patient of tubercular disease.

These results have, however, not been confirmed by Beck and Rabinowitsch, and a similar experience has befallen the writer and Mr. Armit ('Transactions of British Congress of Tuberculosis,' 1901). Thus, out of 31 cases of pulmonary tuberculosis examined by them at the Brompton Hospital 3 only were found to clump in a dilution of 1 in 10, or only about 10 per cent. of the cases, a figure which agrees well with the 12.5 per cent. obtained by Beck and Rabinowitsch. Further, it has been shown by the latter observers that, contrary to the statements of Arloing and Courmont, an exactly similar agglutination may occur in cattle, which have been proved by autopsy to be non-tuberculous.

There can be little doubt, therefore, that *the reaction occurs quite inconstantly in tuberculosis, and, further, that it is not specific. Consequently, as at present carried out, the test can be of no value as a means of diagnosis.*

PROPHYLAXIS.—The preventive measures necessary to check the spread of the disease have been sufficiently indicated in the paragraphs dealing with the methods of infection (see p. 1751). The following are the most important precautions necessary, and they may be thus summarised.

Concerning the *sputum*, the chief source of infection, the public must be educated to appreciate the

dangers arising from it. Spitting except into spittoons (containing where possible some 1 in 20 carbolic acid) must be strictly forbidden, and patients must learn that expectorating into a handkerchief is only less serious than indiscriminate spitting. The sputum should be collected at least once in every 24 hours, and preferably be placed upon the fire and burnt. Failing this, it must be covered with carbolic acid and cast down the drain. It should not be buried, as is sometimes done. The spittoon should be afterwards washed out with carbolic acid and then with boiling water. A phthisical patient must occupy a room by himself, and after his death or removal the room must be thoroughly disinfected, re-papered, and re-white-washed. If precautions such as these be taken, the direct risk of infection becomes but very slight.

As regards food, it is satisfactory to think that with care any possible risks may be entirely avoided. *Milk*, wherein lies the chief danger, should never on any account be drunk without being previously boiled, while if this be done even virulent milk is rendered innocuous since the bacilli are thereby destroyed. It may nevertheless be hoped that in the future the use of tuberculin may be greatly extended, so that a pure milk-supply may be thus obtained. Concerning *meat* the possible risk is less serious. Animals extensively diseased are now confiscated by the authorities, and for the reasons already given the meat of animals only slightly infected is rarely dangerous. Thorough cooking, however, as an additional safeguard should always be advised.

Lastly, to guard against the possibility of *inoculation*, those much associated with consumptives, or who are in the habit of performing autopsies on tuberculous subjects, should be careful to cover with collodion all cracks and abrasions on the hands and exposed parts. If this be done, the danger from this form of infection becomes extremely small.

TREATMENT.—The remedial measures generally adopted in cases of tuberculosis will be found described in other portions of this work. The specific treatment of the disease need alone be here briefly referred to. It may be discussed under the two following headings:—

1. *The tuberculin-treatment.*—Into the history of this treatment it is unnecessary to enter. It may suffice to state that, according to Koch, a cure may be effected in very many cases of *early* pulmonary disease by the continued use of the drug. To effect this it is essential that the cases be early ones, and that the tuberculin (either the original tuberculin of 1890, for further details of which see p. 1745, or the modified 'Tuberculin O,' suggested in 1897) be injected in minute doses, and only after the patient's temperature has remained normal for at least 24 hours previously. In later and rapidly advancing cases its use is contra-indicated or even harmful. It can be seen, therefore, that since the cases in which alone it is indicated are precisely those in which other forms of treatment offer the largest measure of success its use must always remain limited. Partly on this account, and partly also because, according to Virchow, the drug is sometimes not free from danger, a dissemination of the bacilli and a spread of the disease following its use, its employment as a therapeutic agent is now rare. Even in lupus, indeed, in which the results were at first so encouraging, further experience has shown that the benefit is only temporary, and that a permanent

ure, if it ever occurs, is most exceptional. The therapeutic use, therefore, of tuberculin, except, perhaps, as an adjunct to the open-air treatment in certain very early cases of pulmonary tuberculosis, can hardly be recommended.

2. *Serum-treatment*.—The striking value of the anti-diphtheritic serum led almost at once to attempts to manufacture a serum which should be equally effective in the case of tuberculosis. Very numerous experiments in this direction have been made, some using the natural serum of various animals, themselves more or less immune to the disease, others again employing the serum only after subjecting the animals to repeated injections of living and dead tubercular cultures or of their toxins. In some cases the results obtained have been encouraging, notably those of Maraghiano, but as yet no practical results have followed from them. No good purpose would accordingly be served by describing in this article the various experiments in greater detail.

So far, therefore, it must be confessed that the treatment of tuberculosis along specific lines has made but little progress. In the near future, indeed, it may be hoped that great developments in this direction may be attained, but until this time arrives we must continue to trust as heretofore to other remedies and in other measures. Meanwhile we must do all in our power to check the spread of the disease by careful attention to the preventive measures, which have been already described. P. HORTON-SMITH.

TUBULAR.—A term employed to denote (1) a peculiar quality of sound, indicated by the name, either elicited by percussion, or heard on auscultation, in certain conditions (see PHYSICAL EXAMINATION); or (2) that which pertains to the tubules of an organ, e.g. tubular nephritis.

TUMOURS.—SYNON.: FR. *Tumeurs*; Ger. *Teschwulste*.

DEFINITION.—In the broadest sense of the word, a tumour signifies a *swelling*, and must therefore include conditions so far apart as a phantom-tumour, hypertrophied muscle, an abscess, a hernia, or a cancer; but in its more restricted sense its application is confined to a *swelling caused by some form of new-growth*.

CLASSIFICATION.—The separation of new-growths into benign and malignant, though very useful as an approximate clinical distinction, is not admissible in a scientific discussion. Nor again is one that is founded upon the seat or shape of the tumour sufficiently accurate for the purpose. This arrangement would involve such antiquated terms as parenchymatous or superficial, nodules, infiltrations, fungus-growths, &c. The true classification must depend upon the actual structure, that is, the microscopical characters of the growth. Such a classification is the following:—

A. **Tumours composed of a normal tissue of the adult human body, or of such a tissue very slightly modified.**—(1) Fibroma; hard and soft, including Cheloid; (2) Lipoma; (3) Chondroma; (4) Osteoma; (5) Papilloma (warts and corns); (6) Adenoma; (7) Lymphadenoma; (8) True Myoma, including Myo-fibroma; (9) True Neuroma; (10) Angioma; and (11) Lymphangioma.

B. **Tumours consisting of some modification of embryonic connective tissue**, that is, the *Sarcomata*.—(1) Round-celled sarcoma, including Glioma; (2) Oval-celled sarcoma; (3) Spindle-celled sarcoma, large and small; (4) Alveolar sarcoma; (5) Mixed sarcoma; (6) Myeloid sarcoma; (7) Myxoma; (8) Osteo-sarcoma; (9) Chondro-sarcoma; (10) Melanotic sarcoma; and (11) Psammoma.

C. **Tumours consisting of a modification of epidermic, epithelial, and secreting-gland structures.**—These forms of new-growth are described in special articles. See CANCER; and RODENT ULCER.

D. **Tumours consisting of an inflammatory growth.**—(1) *Simple*.—Granulation-tumours, Osteophytes, &c. (2) *Specific*.—Depending on the presence of syphilis, tuberculosis, leprosy, glanders, actinomycosis, and conditions due to other parasitic organisms.

The inflammatory tumours included under this class do not come strictly within the scope of the present discussion; its various sub-divisions must be sought under the description of the diseases which give rise to them, in the several articles bearing their respective names.

E. **Cysts.**—This division is also dealt with in a separate article. See CYSTS.

A. **Tumours composed of a normal tissue of the adult human body.**—In this class are included representatives of each of the primary tissues of the adult body. The members of it, therefore, differ widely in structure and appearance; but they are distinguished from those of the second and third classes by one important feature, namely, that, though often multiple, they show little or no tendency to return after complete removal—that is, they are essentially *benignant*. To this may be added another less characteristic distinction, namely, that they have but little tendency to ulcerate; and that, as a result, if they interfere with life at all, it is by pressure on important organs, or in such an accidental way as by the bleeding which may result from a uterine fibroid, rather than by the production of direct constitutional disturbance.

1. **Fibromata.**—**DEFINITION.**—Tumours consisting simply of fibrous tissue or some modification of it.

VARIETIES, CLINICAL CHARACTERS, AND MICROSCOPICAL APPEARANCES.—Fibromata may be divided into *soft* and *hard fibromata*.

(a) *Soft fibrous tumours.*—The soft fibromata are simple masses of connective tissue, occurring in the submucous or subcutaneous structures, and generally, but not always, more or less pedunculated. In many cases there are overgrown papillae on the surface; and overgrown and distorted glands of the skin or mucous membrane are often entangled among the meshes of the tumour. The *subcutaneous* variety occurs in all parts of the body, but is perhaps most common in the labia majora and the lower limbs; and to it the name of *molluscum fibrosum* has been applied. These tumours often contain a considerable amount of fat, and thus approach the lipomata. They often appear oedematous; and may undergo calcareous or other forms of degeneration. The *submucous* variety includes the simple polypi of the nose. In these the fibrous tissue is somewhat modified; the ordinary connective-tissue cells, oval, oat-shaped, or branched, being embedded in a more or less copious gelatinous

(? mucous) matrix. Such tumours are nearly related, on the one hand, to the myxomata, and are covered by a mucous membrane corresponding to the region in which they occur—ciliated, for example, in the nose (Plate XIX.), and columnar in the intestine. On the other hand, they often contain in their interior the characteristic glands of the part they affect, and thus approximate to the adenomata. To the naked eye they have a gelatinous appearance.

(b) *Hard fibrous tumours*.—These tumours are made up of pure fibrous tissue, but it is very difficult to draw the line between them and some forms of sarcoma. They are firm, usually encapsuled, and often pedunculated. To the naked eye a section is white or pinkish, and presents an appearance as if its component parts were arranged concentrically round a number of points. This appearance is more marked on microscopical examination, which, while it shows this concentric arrangement in bundles that have been cut across, exhibits others which have been divided longitudinally (see Plate XIX.). Hard fibromata occur in many situations: in the subcutaneous tissue, including among others the cheloid tumours, the fibrous tumours of the pinna, which are sometimes caused by the piercing of the ears for earrings, or may result from the hæmatomata not infrequently met with in idiots, and some, at least, of that peculiar class of tumours called 'the painful subcutaneous tubercle'; in sub-mucous tissues, including many of the naso-pharyngeal polypi; in connection with the periosteum, including the fibrous epulis, which probably often starts as a myeloid, the so-called fibrous tumours of bone, and, according to some authorities, though this is doubtful, some kinds of subungual exostosis; in nerves, including the common neuromata, and bulbous nerves in a stump; and in the intermuscular planes. They are liable to various forms of degeneration; they often calcify; and those in connection with the periosteum may undergo ossification. Some fibrous tumours cause serious danger to life from the position they occupy; it will be enough to cite the cases of naso-pharyngeal polypi, and polypi of the uterus.

TREATMENT.—Fibromata can only be treated, if interference of any kind be necessary, by complete removal, the nature of the operation depending upon the position of the growth. If completely removed, except in the case of cheloid tumours, they have no tendency to recurrence.

2. **Lipoma.**—**DEFINITION.**—A tumour composed of normal adipose tissue.

VARIETIES AND CLINICAL CHARACTERS.—Occasionally more or less extensive local hypertrophies of the subcutaneous fatty layer occur. To these the name 'diffuse lipoma' is applied. They occur usually in those who are addicted to alcohol, and especially those who take freely of malt liquors. The circumscribed lipoma is a well defined tumour, made up of a larger or smaller number of overgrown fat-lobules. These are sometimes of enormous size, so that only two or three are found in a tumour of considerable dimensions. The skin presents a very characteristic dimpling, when moved to and fro over such a subcutaneous fatty tumour. The superficial parts of the mass may generally be easily separated from the surrounding structures during an operation for its removal; but the deeper parts, often consisting of smaller lobules, and generally containing a vessel of some magnitude,

require more careful enucleation. Fatty tumours are met with in all parts of the body in which adipose tissue is normally developed. The writer once met with a fatty tumour inside the spinal column. They may occur congenitally, but are more common in middle and advanced life. They are often multiple, and are apparently in some cases developed as the result of pressure; and sometimes they are remarkably symmetrical. They involve no danger to life, but are often very painful, as the result of pressure upon cutaneous nerves. Microscopically, the structure is that of ordinary adipose tissue.

TREATMENT.—Though fatty tumours are said to shift their position, and sometimes to diminish in size spontaneously, they are not to be dispersed by internal remedies or external applications. If necessary, they must be removed by the knife, an operation which, as already mentioned, is usually easy in the case of the circumscribed variety. The removal of diffuse lipomata is difficult, and hæmorrhage is free. It is said that if they result from beer-drinking they will diminish if the habit be discontinued. It is to be observed that recurrence may take place unless they be completely removed.

3. **Chondromata.**—**DEFINITION.**—Tumours made up altogether, or in great measure, of cartilage.

VARIETIES, COURSE, AND CLINICAL CHARACTERS.—Cartilage-tumours may be divided into those which grow in connection with a bone; and those which are developed in the soft parts.

Cartilaginous tumours growing in connection with bone.—These may be again subdivided into those which grow from the surface of the bone—*echondromata*; and those which grow from the interior—*enchondromata*. The latter are the simplest form of cartilage-tumours; they commence usually during the period of adolescence, and affect by preference the fingers and toes, but are occasionally found elsewhere; they are almost always multiple, but never show a malignant tendency; they may reach a very considerable size; and they are sometimes coated with a thin layer of bony tissue. The *echondromata* are developed, as a rule, during a later period of life; are found in connection with any of the bones of the body; and often attain an enormous size, as, for example, in the pelvis. Some of these tumours, to which Virchow has given the name of *osteoid chondroma*, such as are occasionally found forming elongated swellings in the shaft of a long bone, present a high degree of malignancy, recurring as such in distant parts of the body.

Cartilaginous tumours of the soft parts.—These tumours occur principally in connection with certain glands, and especially in the neighbourhood of the parotid, and in the testicle. It has been suggested that they may originate from some remains of foetal structures. Rarer situations for such tumours are the submaxillary gland, the breast, the ovary, the lachrymal gland, the kidney, and, it is said, the lung. These tumours are comparatively seldom pure, but are usually mixed with myxomatous, adenoid, or sarcomatous structure; the degree of such admixture determining in great part the benignity or malignancy of the growth.

An account of the so-called *ossifying chondromata* is given under heading 4.

NAKED-EYE APPEARANCES.—Cartilaginous tumours vary very much in density; the hardest

tain fibrous tissue, and are, in fact, fibro-cartilaginous growths; the softest are very soft, and are very closely related to the myxomata; indeed, it may be held that many myxomata are merely varieties of chondroma. Some chondromata soften, either in many parts or in the centre, giving rise to one or more cysts in the interior; the bursting of such may lead to a permanent sinus. Other forms of degeneration are not uncommon, and especially calcification. True ossification is not rare.

MICROSCOPICAL CHARACTERS.—Microscopically the structure often differs widely in different specimens of chondroma, and in different parts of the same tumour; the matrix may be hyaline or fibrous; and the cells round, irregular, stellate, or much-branched (Plate XIX.). It will easily be understood that with a soft hyaline matrix and much-branched cells, the appearance of myxoma is very easily simulated.

TREATMENT.—Bearing in mind the great variety of these tumours, it will be seen that it is impossible to sum up the treatment of them in a few words. The simple enchondromata of the fingers should only be removed to cure deformity or similar inconvenience. As a rule, other forms should be removed as early as possible; but many chondromata spring from regions which are altogether beyond the reach of the surgeon's knife.

4. Osteomata. — **DEFINITION.** — Tumours composed of bone.

VARIETIES.—If the inflammatory exostoses be excluded, such as those which are found round a joint affected with chronic rheumatoid arthritis, or those which depend upon the ossification of a node, or of inflammatory products in the external auditory meatus, we may divide this class of tumours as follows:—

(a) Osteomata developed as such on the exterior of a bone, including the ivory exostosis—*periosteal exostoses*.

(b) Osteomata developed as such in the interior of a bone—*enostoses*.

(c) *Pedunculated exostoses*.

(d) Osseous tumours of the *soft parts*.

(e) Osseous tumours produced by the *ossification of other kinds of new-growths*.

(f) And lastly (though not strictly under the same category) the *odontomata*.

(a) *Periosteal exostoses.*—These are irregular bony tumours, appearing usually in adult life; they are directly continuous with the bone from which they spring; and are composed sometimes of cancellous structure, with a thin coating of compact tissue, but much more frequently of denser material. They most often affect the bones of the face, where they produce horrible and distressing deformities; but are also found on the skull, in the meatus of the ear, or, more rarely, on the long bones. From the importance of the neighbouring structures it is, in most cases, impossible to remove them, but where it is possible and advisable, as for example in the ear, they should be removed with the gouge and hammer, or by means of a drill. Some of these tumours are of extreme density, and have hence been called *ivory exostoses*; these, when they affect the upper jaw, their commonest seat, must be distinguished from the *odontomata*, to be presently described.

(b) *Enostoses.*—Enostoses need only be mentioned in order to point out their extreme rarity; but it may be remarked that many of the last-

described series of tumours probably spring from the diploë of the cranial bones, and should thus, perhaps, more properly be included under this heading. Certain bony enlargements, the pathology of which is not understood, such as *Leontiasis Ossea*, must be mentioned here. These may result in great disfiguration, and from pressure on nerves cause great pain. Extensive surgical operations are sometimes undertaken for their removal.

(c) *Pedunculated exostoses.*—The pedunculated exostosis, or ossifying chondroma, is a subperiosteal chondroma, with a tendency to ossification, developed in young people, near the junction of an epiphysis with a diaphysis. They are common in the subjects of rickets.

Pedunculated exostoses approach the spherical shape, but are sometimes irregular or flattish, and often tuberculated. The peduncle varies in its relative size, the growth being often nearly sessile. On section the tumour shows a layer of periosteum superficially; beneath this is a layer of cartilage, sometimes thick, sometimes almost imperceptible. The deeper part of the cartilaginous layer is calcified, and looks like imperfectly formed bone. The centre of the tumour consists of true bone, with Haversian systems complete, and is directly continuous with the tissue of the bone itself. The cancelli are sometimes dilated so as to form actual cysts containing clear fluid. In adult life the layer of cartilage may disappear; the tumours always cease to grow.

The microscopical appearance answers exactly to the structure which is apparent to the naked eye (Plate XIX.).

These tumours occur most often near the ends of the long bones, but may be found elsewhere; on the scapula, for example. They are often multiple and symmetrical, and if multiple are frequently hereditary. In certain situations, as, for instance, on the inner side of the knees, they may cause much inconvenience, from the pain produced by pressure as in riding, or from the catching and sudden slipping over the tumour of tendons or other fibrous structures in the neighbourhood. For this reason, or from their size, they may require removal. This may be done freely if antiseptic precautions be adopted; otherwise the opening of the cancellous structure of the bone, and the danger of wounding the contiguous joint, may perhaps involve greater risk than the amount of inconvenience entailed by the tumour would justify. They have sometimes been either purposely or accidentally separated from their attachment by a blow, without inflicting a wound on the soft parts at all. Unless the whole cartilage-layer be removed, recurrence will probably happen, as growth of the tumour takes place by increase of this layer only, the process of calcification and subsequent ossification being secondary, and, so to speak, accidental.

(d) *Osseous tumours of the soft parts.*—These osteomata are also uncommon. They include such conditions as the following: tumours springing from the periosteum, but not actually united to the bone; ossifying chondromata not connected with the bone; detached exostoses; ossification taking place in muscles or tendons, such as that which is occasionally met with in the adductor longus; and perhaps some other varieties of greater rarity.

(e) *Osseous tumours produced by the ossification of other kinds of new-growths.*—These are only secondarily, and often only partially, worthy of the name

of osteomata; they are the result of a process of ossification taking place in tumours of a different nature originally, such as fibromata or sarcomata. It must be remembered that, while calcification of new-growths is a common form of degeneration, the occurrence of true ossification is very rare.

(f) *Odontomata*.—Odontomata are tumours composed of one or more of the constituents of the teeth, and they may be classified accordingly: 1. Epithelial odontomes, arising from the enamel-organ and forming encapsuled, multilocular cystic tumours, usually in the lower jaw. 2. Follicular odontomes or dentigerous cysts (*see* article CYSTS). 3. Fibrous odontomes, uncommon tumours developed from the tooth-sac. 4. Cæmentomata, including exostoses from the fangs, which are common, usually small, and often extend from one tooth to another; and also a rarer form of tumour, consisting of larger masses of cæmentum in which teeth are embedded. 5. Compound follicular odontomes, resulting from irregular ossification of the capsule and containing numerous small fragments of cæmentum, dentine, or small ill-shaped teeth, embedded in fibrous tissue. 6. Radicular odontomes composed of cæmentum and dentine, and growing from the root. 7. Composite odontomes, which are masses of cæmentum, dentine, and enamel indiscriminately arranged and forming tumours in the substance of the jaws, usually the lower, or occupying the antrum of Highmore. Many of these odontomata are of great rarity. This arrangement is borrowed from Mr. J. Bland-Sutton. They are only found in connection with the jaws. *See* TEETH, Diseases of.

5. *Papillomata*.—DEFINITION.—Papillary and villous over-growths, whether occurring on mucous membrane, skin, or serous membrane, which do not present malignant characters.

VARIETIES AND SYMPTOMS.—Mucous papillomata are found on the lips, tongue, and soft palate, and in the larynx; in the intestines, especially at the lower part; round the anus (condylomata); in the bladder (the simple villous tumour); on the conjunctiva; and at the orifice of the female meatus urinarius. Epidermic papillomata include warts and corns, and may occur on any part of the skin; those about the external genitals may reach an enormous size, and are frequently the result of gonorrhœa. The serous and synovial papillomata are more rare; under this class must be named the Pacchionian bodies, and the enlarged synovial fringes which are often the commencements of loose bodies in joints. Many papillomata are of syphilitic origin; others may be caused by local irritation, as, for instance, the cadaveric wart, in which the papillary growth is accompanied by inflammation and suppuration beneath the skin, due in most cases, if not in all, to the presence of the tubercle-bacillus.

NAKED-EYE APPEARANCES.—The naked-eye appearances vary very much with the locality in which the growths are developed; but they present this character in common, that they are obviously composed of the papillæ or villi of the part from which they grow, though these may be of much more than the natural size. Those springing from mucous and serous membranes are soft, and usually moist on the surface; while epidermic papillomata are dry and hard.

MICROSCOPICAL CHARACTERS.—Microscopically papillomata are made up of connective tissue containing numerous vessels, covered by the characteristic epithelium or epidermis of the part (Plate IV.

p. 244). The arrangement of the connective tissue and epithelial elements is a close imitation of that of the normal tissues from which they grow. The microscopical structure of the benign polypus of the intestine (fig. 4, Plate IV, p. 244) is hardly to be distinguished from that of many malignant growths in this situation. *See* CANCER.

TREATMENT.—If affecting the larynx or bladder, papillomata may give rise to distressing and dangerous symptoms, and may necessitate severe operations for their extirpation. Those which occur on accessible parts are generally removed without any risk, except such as arises from their great vascularity; and show, as a rule, but little tendency to recur after complete removal. The common wart, however, often gives great trouble. Various caustics may be used, such as the acid nitrate of mercury, fuming nitric acid, caustic potash, glacial acetic acid, or nitrate of silver; salicylic acid is also often extremely useful; but while these remedies are sometimes efficacious, the warts will often recur with the greatest possible obstinacy. At the same time it must be remembered that they frequently show a most capricious tendency to spontaneous cure; the surgeon will sometimes be mortified at finding that while his energies have been devoted with but partial success to the cure of one or two out of an extensive crop of these growths, the remainder have, in the meantime, spontaneously disappeared. The cadaveric or dissecting-room wart is best treated by excision, for it must be regarded as a local tubercular lesion which may lead to general infection. The best local treatment for condylomata and gonorrhœal warts is the application of some desiccating powder, such as dried alum. It is well to remember that warts which remain a long time uncured are apt to become epitheliomatous; and not only so, but that any chronic irritation, whether from the application of insufficient methods of cure or other causes, is likely to lead to the same result.

6. *Adenomata*.—DEFINITION.—An ill-defined group of tumours, a typical member of which is essentially non-malignant, and is made up of tissue exactly resembling that of the gland from which it springs; but the departures from the ordinary type are so many and so varied, and, at times, so indefinite, that it becomes impossible to draw a clear line between the adenomata and the carcinomata, or indeed between the adenomata and the sarcomata.

(a) *Adenoma of the Sweat-glands*.—This class is said to include some non-ulcerating cutaneous tumours (the tubular epitheliomata of some authors); and some ulcerating ones—the cancroids or rodent ulcers have been erroneously placed under this heading. *See* RODENT ULCER.

(b) *Adenoma of the Sebaceous glands*.—This is a rare tumour, growing on different parts of the skin. Though not strictly a tumour, that local hypertrophy of the skin of the nose, to which the popular name of 'grog-blossom' has been given, must be mentioned here. This forms a nodular purplish tumour, growing from the end of the nose, and often reaching an enormous size. When cut into, it bleeds freely, and exudes from innumerable cavities in its interior an inspissated sebaceous secretion. *Microscopically* it is seen that the connective tissue and the vessels are hypertrophied, as well as the sebaceous glands. The *treatment* consists in removal by the knife; and cicatrization generally

occurs with wonderful rapidity. Recurrence is not uncommon.

(c) *Adenoma of the Mucous Glands.*—Under this heading might be included some of the simple polypi of the nose (see FIBROMATA). The best representative, however, is the simple polypus of the rectum. This is a sessile or pedunculated roundish tumour, occurring mostly in children and young subjects. It bleeds freely from the surface, and is often the cause of painful and somewhat troublesome symptoms, among which prolapsus ani is the most common. *Microscopically* it consists of tissue closely resembling that of the mucous membrane of the rectum, but the hypertrophied follicles are often imbedded in a tissue very similar to that which forms the basis of the mucous polypus of the nose (Plate IV. p. 244). The treatment is by removal either with the knife or ligature, or some form of snare or écraseur. Recurrence does not, as a rule, take place.

As is stated in the article CANCER, and as is shown on Plate IV., it is often almost impossible to distinguish, by their microscopical appearances, the simple adenomata of the large intestine from the malignant growths affecting the same structures. The same observation applies to many tumours of the jaws, such as that in Plate IV., which, though classed among the adenomata, often exhibit a high degree of malignancy.

(d) *Adenoma of the Breast.*—The most typical adenoma of the breast is a rounded tumour of moderate size, occurring often at the margin of the gland and frequently near the axillary border, completely encapsuled, and consisting of tissue which hardly differs from ordinary mammary structure (Plate IV.). It occurs usually in young women, often during the child-bearing period. It shows no tendency to recur after removal. A large number of adenomata of the breast do not, however, agree with this description, either as regards position or structure. In structure departure may take place from the normal type in two directions: by an excessive or abnormal development either (1) of the epithelial or (2) of the connective-tissue elements. In the former case the tumour, in proportion to its abnormality, approaches the cancers; in the latter it assumes more and more closely the characters of the sarcoma. Thus we find some of the softer adenomata, as shown in Plate IV., exhibiting a tendency to recur after removal, in the same way as a carcinoma, and with an almost equal degree of malignancy; while others, presenting characters like those of Plate IV., may follow the same course as the sarcoma, both as regards the manner of involving surrounding tissues and the way in which they recur in the viscera. These latter are called *adeno-sarcomata*, and often attain an enormous size. If the stroma of one of these tumours be in large amount, and fibrous or myxomatous, the names of *fibro-adenoma* and *myxo-adenoma* may be applied. Adenomata often contain cysts, and these cysts not infrequently intracystic growths. They must be treated by removal of the growth.

The reader must be content with the foregoing approximate description of this important class of tumours, the varieties in the nomenclature of which are equal to the number of the authors who have written on the subject. The writer believes that such unnecessary confusion has been caused by his multiplication of names, for a detailed account of which special works must be consulted, as their

discussion would lead far beyond the limits of the present article.

(e) *Other forms of Adenoma.*—Among other rarer forms of adenoma, bearing a more or less close relationship to the glands from which they spring, may be enumerated the following: Adenomata of the testicle or ovary, of the salivary and lachrymal glands, and of the liver, and some tumours of the thyroid. Many of the symmetrical enlargements of the thyroid, however, are simply hypertrophies of the gland itself, and should therefore be classed with simple hypertrophy of the breast and ordinary enlarged prostate, rather than with the tumours now under discussion. It should be noted that many of the less typical adenomata, especially those of the salivary glands and of the testicle, frequently are found in combination with other heteroplastic growths such as chondroma, myxoma, or some form of sarcoma.

7. *Lymphadenoma.*—Under certain circumstances, for an account of which the reader is referred to the article LYMPHADENOMA, the lymphatic glands throughout the body become enlarged, forming tumours, often of enormous size.

8. *Myoma.*—DEFINITION.—A tumour composed of muscular tissue.

With certain very rare exceptions, the only form of muscle-tissue occurring as an integral part of a tumour is the *unstripped* variety; and almost the only position in which this is found is in the so-called 'fibroma' (myoma) of the uterus. This growth consists of a mixture of fibrous tissue with plain muscular fibres in varying amount, usually exhibiting the concentric arrangement of its elements, which was described as characteristic of fibromata generally. Fibromata of the uterus may form pedunculated tumours on the external or internal surface of the uterus, or they may not extend beyond the uterine wall. They give rise to a variety of special symptoms, and require special methods of treatment, which it is beyond the scope of this article to discuss. See UTERUS, Diseases of.

Striped muscular fibre has been found in a few cases of congenital tumour of the kidneys; and the writer has also seen it in a fatty tumour growing inside the spinal canal.

9. *Neuromata.*—DEFINITION. — Tumours composed essentially of any form of nerve-tissue.

The majority of neuromata are really fibromata, that is, fibrous tumours developed among and around the fibres of the nerve from which they spring. The idiopathic forms are sometimes single, but generally multiple, of small size, very hard, and affecting usually the branches of a particular cutaneous nerve. The amount of pain and tenderness caused by these growths varies very much, but is sometimes excessive. A clinical feature of some use in diagnosis is the fact that they usually move readily in the lateral, but very imperfectly in the vertical direction. What may be called traumatic neuromata are fibromata containing tortuous outgrowths of the nerve-fibres developed at the end of a divided nerve in a stump. These often cause excessive pain and peculiar reflex phenomena; while they show a remarkable tendency to recur after removal. Neuromata on the cranial nerves are rare; they may cause serious and characteristic symptoms. Still rarer are the true neuromata of the brain and spinal column, and those occasionally met with in connection with the nerves or ganglia of the sympathetic.

It must not be forgotten that less simple tumours not infrequently affect nerves, such as the various forms of sarcoma, or myxoma. No special description of these growths in this situation is, however, required.

10. **Angioma.**—SYNON.: Telangiectasis.

DEFINITION.—A tumour composed of blood-vessels.

Angiomata divide themselves naturally into those in which the *capillary* element predominates; and those which are chiefly made up of *cavernous* tissue.

(a) *Capillary angiomata.*—DESCRIPTION.—These are the *nævi*, which, while they sometimes form tumours of some magnitude, often, as in the case of so-called ‘claret-cheek,’ involve no increase in the size of the affected part. Nævi are nearly always congenital, hence the term ‘mother’s mark.’ They may be subdivided into *cutaneous* and *subcutaneous* nævi. The former are of a more or less bright red colour, and affect only the cutaneous structures or mucous membrane; the latter, as seen through the skin, in cases where this remains unaffected, have a purplish tint, and may involve any of the deeper structures of the body. They are usually as circumscribed as fatty tumours, and very commonly involve the skin. Nævi often grow with extreme rapidity, and though they involve no danger to life (except in such rare conditions as when serious or fatal hæmorrhage occurs from a nævus of the pelvis of the kidney or of the rectum), they may cause serious inconvenience and great disfigurement. Sometimes, however, they exhibit a tendency to spontaneous disappearance, and often they remain permanently stationary. They are liable to various forms of degeneration, notably the cystic, and the ulcerative or suppurative.

Microscopically a nævus is composed of large capillaries, among which are seen arterial and venous trunks of larger size. Between the vessels is found connective tissue or fat, and sometimes the special constituents of the skin, such as sweat-glands or sebaceous glands. The nævus-element enters rather largely into the composition of some other tumours, and notably of congenital moles (*benign melanoses*).

TREATMENT.—Inflammation of a nævus generally leads to spontaneous cure; nature thus suggesting one of the best methods of treatment at the disposal of the surgeon, namely, the injection of the tumour with some suitable irritant, such as carbolic acid. In adopting this line of treatment, it must be remembered that a danger exists of the irritating fluid entering a larger vessel, and by passing to the heart and setting up coagulation there causing instant death; this may be guarded against by the application of a temporary ligature. Other recognised and useful plans of treatment are the following: electrolysis, puncture with the actual cautery, and complete excision of the mass. The last in available situations affords the most speedy cure, and often leaves the least conspicuous scar. Resort is now seldom had to the ligature or treatment by pressure or the application of caustics.

(b) *Cavernous angiomata.*—DESCRIPTION.—Those angiomata which are made up of larger vessels, and which are hence called the cavernous angiomata, from their resemblance in structure to erectile tissue, consist of cavernous spaces, communicating by smaller or larger vessels, and separated by trabeculae of greater or less thickness and substance. These are the *pulsating nævi*, and

perhaps some of the so-called *aneurysms by anastomosis*. They are of a more dusky colour than simple nævi, and often present a distinct thrill or bruit, which is perceptible both to the patient and to the surgeon. They are sometimes encapsuled, sometimes diffused; and in the latter case show an almost malignant tendency to involve neighbouring structures. The pulsation is often a most distressing symptom to the patient, if the tumour be found occupying such positions as the pinna of the ear or the fat of the orbit, both of which situations are not at all uncommon for the occurrence of the disease. *Microscopically* a cavernous angioma presents fibrous trabeculae, lined with the characteristic vascular endothelium, and in parts perhaps separated by layers of areolar or any other tissue which the tumour may happen to involve.

TREATMENT.—The treatment must be pursued on the same lines as that for the simple forms of nævus. It should, however, be undertaken with caution; for injection is a much more dangerous proceeding on account of the large size of the vessels, and excision is apt to be accompanied with serious hæmorrhage, because of the enlargement of the vessels in the neighbourhood.

11. **Lymphangioma.**—DEFINITION.—A rare kind of tumour, which may briefly be described as a cavernous angioma made up of lymphatic vessels.

To this class belong some curious and uncommon flat pinkish elevations of the skin, characterised by the presence of minute vessels containing clear fluid, and particularly prone to erysipelatous inflammation. It also in all probability includes the *cystic hygromata* occasionally met with congenitally. See CYSTIC LYMPHANGIOMA.

B. **Sarcomata.**—DEFINITION.—It is to be regretted that the term ‘sarcoma’ has not been allowed to slip out of pathological terminology. From the days of Galen almost to our own time it has served, in the hands of different authors, to designate different classes of tumours, sometimes of the simplest, sometimes of the most malignant character. Virchow, however, has given a meaning to the word which is now generally recognised by pathologists. He includes under *sarcomata* those new-growths which, while they do not actually consist of any of the tissues of the adult body, are evidently built on the connective-tissue type, and consist of a modification of the connective tissue of the embryo. They are thus very closely related to some of the simple tumours, and indeed often include portions of some normal tissue, such as bone, cartilage, or fibrous tissue—a fact which necessitates the employment of complicated and confusing names, including *osteo-sarcoma*, *chondro-sarcoma*, *fibro-sarcoma*, and such-like.

Certain forms of sarcoma, especially of the rounded variety, are believed to originate in the endothelial lining of lymphatics or capillary blood-vessels. To these tumours the name *endothelioma* has been applied. The subject will be referred to later on.

MICROSCOPICAL CHARACTERS.—Histologically, then, a sarcoma is made up simply of cells of the connective-tissue type, which may assume very various shapes and sizes in different tumours, and which are surrounded by a varying amount of inter-cellular substance.

CLINICAL CHARACTERS.—Theoretically sarcomata should always be developed in one of the connective-tissue structures, and practically they are

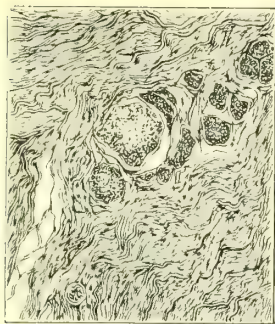


FIG. 1.—Fibroma (Neuroma).



FIG. 2.—Polypus of Nose.



FIG. 3.—Myxoma.

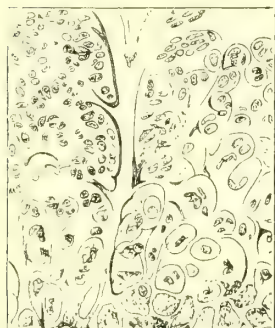


FIG. 4.—Ossifying Chondroma.

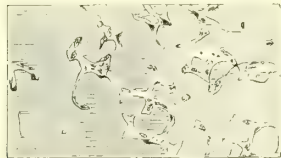


FIG. 5.—Enchondroma (of Jaw).

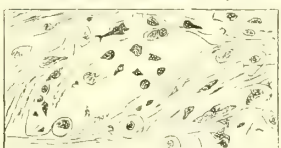


FIG. 6.—Enchondroma (of Orbit).

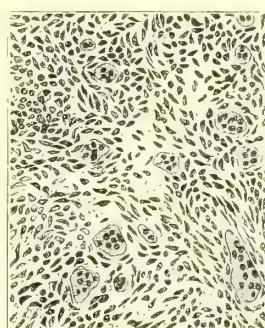


FIG. 7.—Myeloid of Jaw.

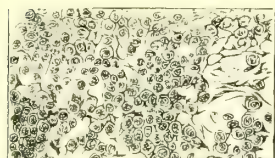


FIG. 8.—Large Round-celled Sarcoma.

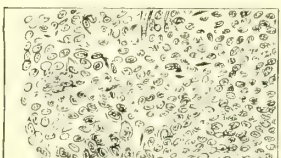


FIG. 10.—Oval-celled Sarcoma.



FIG. 9.—Small Round-celled Sarcoma.

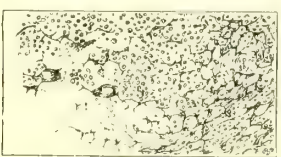


FIG. 11.—Lymphoma.

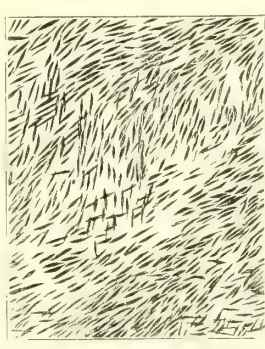


FIG. 12.—Small Spindle-celled Sarcoma.

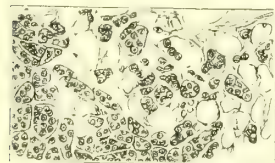


FIG. 13.—Alveolar Sarcoma.

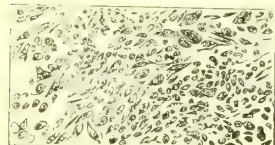


FIG. 14.—Mixed Sarcoma.



FIG. 15.—Melanotic Sarcoma.



FIG. 16.—Large Spindle-celled Sarcoma.

not very often seen to originate in a secreting gland. But a glandular origin is not by any means very uncommon, as indeed might have been expected when it is remembered that every gland contains a considerable amount of connective tissue. Sarcomata present all degrees of malignancy; but, as a rough rule, it may be stated that the higher the degree of development of the tumour, the less likely is recurrence to take place after removal. In connection with this point it is interesting to note that each recurrence of a sarcoma often shows a more rudimentary structure, and at the same time a greater degree of malignancy.

Dissemination does not follow the same rule that has been observed in the case of cancer. The lymphatic glands often escape altogether, or are but slightly affected; while fresh tumours spring up in abundance in distant parts of the body.

Sarcomata increase in size in the same way as cancers, but in the less malignant forms are often surrounded by a more or less distinct capsule.

VARIETIES.—1. *Round-celled Sarcomata*.—These are the most rudimentary, and, as a rule, the most malignant of this class. They are usually soft—sometimes very soft—seldom encapsuled, and generally of a whitish colour. Formerly they were classed among the medullary cancers. They are made up of round cells, as the name implies, and have a tendency to group themselves into two classes, in one of which the cells are *small* and uniform in size (fig. 9), closely resembling those of granulation-tissue; and in the other large, sometimes very large, and often somewhat irregular (fig. 8). The intercellular substance may be copious and homogeneous, in which case the tumour approaches the myxomata; or smaller in amount and fibrous, when, if the cells are small, it may be difficult to separate such a sarcoma from the lymphomata. To some of these the term *lymphosarcoma* has been applied. Such tumours occur in the tonsil, or may start in the lymphatic glands.

Under this head must be placed the *Gliomata*—soft medullary tumours met with in the brain or in connection with nerves, frequently with the retina, and occurring commonly in children. They show microscopically a delicate stroma. See p. 196.

2. *Oval-celled Sarcomata*.—This variety of sarcoma represents the next stage in advance from the simplest towards a more complex structure. They differ, in fact, but slightly from the round-celled growth, either in clinical or in microscopical characters (fig. 10), but may be looked upon as intermediate between these and the spindle-celled sarcomata.

3. *Spindle-celled Sarcomata*.—This class must be subdivided into the *small spindle-celled*, and the *large spindle-celled* varieties.

The *small spindle-celled sarcomata* are firm, whitish, well-defined tumours, which approach the fibromata, sometimes very closely, and which after complete removal show a comparatively slight tendency to recur. These were the *recurrent fibroids* of older writers. *Microscopically* (fig. 12) they consist of broad interlacing bands of elongated cells, with but little intercellular material—a structure which does not differ widely from imperfectly formed fibrous tissue.

The *large spindle-celled sarcomata* (fig. 16) are softer growths, frequently supplied with a very imperfect capsule, generally pinkish on section, or stained in parts dark red from extravasated blood,

and often showing cysts. They may occur in any fibrous structure, but are rather common in connection with the periosteum. The malignancy of these growths is much greater than that of the small-celled class. *Microscopically* they consist of very large nucleated cells, with long tapering tails, and but little intercellular substance. These are the *fibro-plastic* tumours.

4. *Alveolar Sarcomata*.—Examined *microscopically* this rare form of tumour (fig. 13) bears a superficial resemblance to a cancer. There is, that is to say, a coarse stroma forming alveolar spaces, each of which contains a variable number of large, round, nucleated cells; each space is, however, again subdivided by a very delicate secondary intercellular stroma. This is only demonstrable on pencilling out the cells, which, unlike a similar process applied to a cancer, is a work of considerable difficulty. Alveolar sarcomata affect most commonly subcutaneous tissues primarily; they are apt to recur in other parts of the body, and ultimately in internal organs, but often run a very chronic course.

5. *Mixed Sarcomata*.—Tumours are often met with which present a mixture of the different structures just described. Such are conveniently called mixed sarcomata; and the term may with advantage be made to include those growths which contain, besides sarcoma-tissue, bone, cartilage, gland-tissue, &c. The latter growths will necessitate the employment of such names as osteosarcoma, chondro-sarcoma, adeno-sarcoma, &c.

6. *Myeloid*.—**SYNON.**: Giant-celled Sarcomata. — These tumours are best classed among the sarcomata. *Microscopically* (fig. 7) they present the following elements, one or other of which may greatly predominate: fibro-plastic or spindle-cells, oval or round cells, and very large nucleated cells, the so-called 'myeloid' or 'giant' cells. These last are irregular or rounded masses of granular protoplasm, in which occur numerous clear oval nuclei, each containing a well-marked nucleolus. The amount of intercellular substance is small. In a fresh scraping the clear oval nuclei are set free by the breaking up of the giant-cells. To the naked eye a section of a myeloid tumour is pink or yellowish, but almost always mottled with darker spots, the result of extravasations of blood. Very frequently, tumours of this class present points of ossification or calcification, and not infrequently cysts. They are very soft, and usually yield a thick juice on scraping. They generally originate in, or, more rarely, close to, the end of one of the long bones; probably most often in the medullary cavity or the cancellous structure. Another frequent seat is the alveolar border of the jaws, where they form the myeloid epulis. They occur most often in young people, and if the bone involved be removed completely or in great part, have no tendency to recur; but, on the other hand, they may exhibit a high degree of malignancy, recurring not only in other bones, but in internal organs. It is said that myeloid epulides may in time become completely fibrous.

7. *Myxoma*.—It is not easy to say whether myxomata should be classed among the sarcomata or among the simple tumours. On the one hand, the tissue of which a myxoma is composed finds no representative in the adult, unless that of the vitreous body be taken as such; but, on the other hand, they approach very closely some of the soft

fibromata, and are nearly related to fatty tumours. At the same time a large number of myxomata appear to be actual chondromata. It must be added that some modern pathologists assert that many myxomata are really endotheliomata.

Microscopically (fig. 3) a myxoma presents elongated or roundish much-branched cells, the prolongations of which intercommunicate freely. These cells are embedded in a copious, homogeneous, transparent matrix. The structure thus resembles that of foetal fat; but, in myxomata developed from cartilage, each cell is contained in a space similar to that enclosing a cartilage-cell.

Clinically these tumours are very soft and elastic, strictly encapsuled, gelatinous, semi-transparent on section, and exuding a peculiar mucous juice. Myxomata are not malignant; and as a rule, if completely removed, do not recur locally. They may be found in many parts of the body, but perhaps most frequently in the subcutaneous tissue, or in connection with some gland, particularly the parotid. Here, however, they are often mixed with adenoid and cartilaginous material. Not infrequently myxoma-tissue is combined with that of an undoubted sarcoma, which necessitates the term *myxo-sarcoma*. These tumours may occasionally be mistaken for colloid cancers. Histologically some of the soft enchondromata resemble them very closely.

8 and 9. *Osteo-Sarcoma*; and *Chondro-Sarcoma*.—These forms of sarcoma have just been referred to under the head of *Mixed Sarcomata*.

10. *Melanotic Sarcomata*.—These tumours are, in the experience of the writer, usually of the mixed round- and spindle-celled variety. Some, but not all, of the cells contain a brown pigment, but in very varying amount, and pigment is also found outside the cells (fig. 15). This gives the tumour a brown or blackish appearance. These tumours, though often completely encapsuled, show a high degree of malignancy; but are often succeeded by a mixture of white and black tumours, or sometimes by white tumours alone. They are not uncommon in connection with the choroid of the eye, and the papillæ of the skin (malignant mole), but have often been met with primarily in other parts of the body. The secondary growths are often found disseminated through every tissue of the body, forming tumours in such situations as the intestine, of peculiar and characteristic appearance. Secondary deposits in lymphatic glands are more common than is the case in other forms of sarcoma. It has been observed that in some cases of melanosis black pigment, accompanied by other morbid appearances, has been found in the blood, and also in the urine.

11. *Psammodoma*.—It is only necessary to mention this very rare tumour, which is found only in connection with the membranes of the brain. It is composed of flattened cells, and is characterised by the peculiarity of containing brain-sand in its interior. It seldom or never gives rise to symptoms. See p. 332.

Endothelioma.—It is difficult at the present time to write definitely on this class of tumours, which are supposed to arise from the lining membrane of the blood-vessels and lymphatics; for, while some observers describe them as rare, others consider that all the simple tumours of the parotid region and many cancers of the breast belong to this cate-

gory. Under the circumstances it seems impossible to attempt to describe them. The reader must be satisfied to know that this subject is under discussion, and to consult the most recent literature about it.

Teratomata.—Mention must be made, in conclusion, of a form of growth, which does not come within the classification here selected. This is a congenital tumour not infrequently met with in the region of the sacrum (*congenital sacral tumour*), but occasionally seen elsewhere; often reaching a size almost equal to that of the infant itself. In structure these tumours consist of various imperfectly developed foetal or mature elements of the body, mixed together in apparently great confusion. Various theories as to their causation have been propounded; such, for example, as that they consist of an imperfect attached foetus, or that they originate from Luschka's gland; but we have not sufficient data at present to express an opinion on this point. Attempts at their cure by removal are attended with great danger (see p. 1518).

R. J. GODLEE.

TUNBRIDGE WELLS, in Kent.—Iron waters. See MINERAL WATERS.

TUNNEL-WORM.—A synonym for the Ankylostoma duodenale. See ENTOZOA.

TURGESCE (*turgesco*, I swell).—A term applied to a swollen condition of a part, generally associated with fulness of the blood-vessels, as in the mucous membrane of the conjunctiva, the fauces, or the rectum.

TUSSIVE (*tussis*, a cough).—This word is applied to certain physical signs which are elicited by the act of coughing, such as *tussive fremitus* and *tussive resonance*. See PHYSICAL EXAMINATION.

TYLOSIS (τύλος, callus).—SYNON. : Callosity.—This name originally signified any hardening of the skin, the result of pressure, or any other cause. It has, however, become customary to apply the term to certain fairly well-defined conditions. In its clinical significance it may be regarded as a thickening of the horny layer of the skin, accompanied by appreciable inflammatory reaction. In this category is included the condition known as *tylosis palma et planta*. This disease, which appears to be in many cases congenital, consists in the thickening of the epithelium of the palms and soles, so that a firm parchment-like or horny layer is produced. This is usually of a yellowish or brownish tint, and semi-translucent in appearance. On account of the stiffening produced, and the movements of the hand, cracks are liable to occur, and this may be the cause of much pain; the surface also may lose its yellowish, horny appearance, and become greyish or black as the result of the discolouration of epithelium, and the adhesion of dirt and foreign matter. The condition is usually limited to the palmar and plantar surfaces.

Several of the cases reported have occurred in family groups, and may be regarded as congenital. In other cases the disease appears in adult life, and is often associated with hyperidrosis. Arsenical keratosis in bad cases may simulate this affection, but its papular appearance in the early stage, and the increase of pigmentation, which is frequent, as

well as other symptoms of arsenical poisoning, serve to distinguish the one condition from the other.

In addition, one or two rare affections of the palms and soles produce thickening of the epithelium to a very marked extent. These are, however, associated with a greater or less degree of erythema, which may be especially distinguished at the margins of the horny areas. In this category come the Erythema keratodes of Brooke, and the Keratoderma erythematosa symmetrica, described by Besnier.

The term 'tylosis' is loosely applied to the thickening of the palms and soles secondary to conditions such as eczema, psoriasis, and lichen planus. The inflammatory reaction in these conditions is usually recognisable, and the darkened, cracked, and crusty surface serves to distinguish them from the congenital diseases, to which the name is perhaps more appropriately limited.

In many of these pathological states the peculiar thickening which is given rise to may be the result of imperfect development of the epithelium as the result of abnormal conditions of the epitrachium. This condition would especially account for congenital cases of the disease unassociated with inflammation.

TREATMENT.—Even in congenital tylosis, and more especially in the irregular varieties of other types, much can be done by softening the epithelium by means of baths, or even by alkaline applications of soft soap or borax, and the subsequent application of salicylic acid. This drug may be applied in the form of solutions, as ointments, or plasters. The strength of these ranges from 5 to 50 per cent. The horny epithelium is thus removed from time to time, and in those cases which cannot be cured the skin is rendered more supple, while in certain cases actual cure can be brought about.

JAMES GALLOWAY.

TYMPANITES (τύμπανον, a drum).—SYNON.: Fr. *Tympanite*; Ger. *Windsucht*.—This word signifies the distension of the abdomen that results from excessive accumulation of gas within its cavity. As a rule, the gas collects in the interior of the alimentary canal, especially the intestines; but in exceptional cases it occupies the peritoneal cavity.

ÆTIOLOGY.—Tympanites is chiefly met with under the following circumstances: 1. In connection with certain diseases which, from their local effects, tend to paralyse the intestines, especially acute peritonitis, typhoid fever, and dysentery. 2. In cases of intestinal obstruction from any cause, but particularly when this condition is acute, and situated low down. 3. In certain low febrile diseases, accompanied by the 'typhoid state,' and tending towards a fatal issue, such as typhus fever, small-pox, erysipelas, and pneumonia. 4. As a result of perforation of the stomach or intestine. 5. In certain cases of chronic disease of the spinal cord. 6. In connection with hysteria sometimes. In all these conditions, except where the gas escapes into the peritoneal cavity, the immediate cause of the tympanites is a more or less paralysed state of the walls of the intestines; but there is often, at the same time, an excessive formation of gas in their interior, which distends them.

SYMPTOMS.—The symptoms of tympanites are due to the mechanical effects of the gaseous accumulation. The patient is usually conscious of the distension of the abdomen, and the sensation may

amount to extreme discomfort or actual suffering and great distress, there being a feeling in some instances as if the abdomen must burst if the condition is not relieved. The mental state of the patient may, however, be such that he is unconscious of, or indifferent to, any unusual sensations. Breathing is often interfered with in various degrees, and the act may be very hurried, with a feeling of urgent dyspnoea. The heart is also liable to be affected, and its action more or less disturbed. The secretion of urine may be interfered with, even almost to actual suppression.

PHYSICAL SIGNS.—These are usually very characteristic. 1. The abdomen is uniformly enlarged, often to an extreme degree; being of a rounded shape; equal and symmetrical in every part, unless there happen to be a portion of bowel specially distended, and without any tendency to undue prominence in dependent parts. The skin is stretched more or less, but there is no protrusion of the umbilicus. 2. The sensations on palpation are those of perfect smoothness and regularity, with great tension or a drum-like feel. 3. Percussion gives a general tympanitic sound over the abdomen, and also brings out the drum-like sensation. If, however, the distension is extreme, the sound becomes more or less muffled and toneless. Generally the dullness of the solid organs in the abdomen is partially or entirely obscured, or is displaced upwards. 4. Change of posture produces no alteration in the physical signs. 5. There may be indications of displacement of the thoracic organs. It must be mentioned that tympanites may be associated with some fluid in the peritoneal cavity, or with other conditions, and the physical signs will be modified accordingly.

TREATMENT.—In the first instance, any obvious and immediate cause of tympanites must be removed, if practicable, such as intestinal obstruction, or an accumulation of fæces. If the symptom calls for direct treatment, relief may be afforded in some cases by applying heat or cold, sinapisms, or turpentine stupes over the abdomen; and administering internally such remedies as brandy, aromatic spirit of ammonia, the various ethers, the volatile oils, camphor, musk, valerian, sumpul, galbanum, asa-fetida, or other gum-resins. Should these fail, enemata containing asafetida or turpentine may not uncommonly be used with advantage. The passage of a long tube, such as an oesophagus-tube, through the anus into the bowel, reaching as high up as possible, is often very serviceable. In extreme cases it has been recommended to open the abdomen and make a longitudinal incision in the bowel, thus establishing an artificial anus for the time being. When gas accumulates in the peritoneal cavity, it can only be got rid of by surgical interference, should such interference be otherwise indicated.

FREDERICK T. ROBERTS.

TYMPANITIC (*tympanum*, a drum).—A peculiar drum-like quality of sound elicited by percussion (see PHYSICAL EXAMINATION). The term is also applied to the abdomen, when it is distended with gas. See TYMPANITES.

TYMPANUM, Diseases of.—See EAR, Diseases of.

TYPE-WRITER'S CRAMP.—See WRITER'S CRAMP; and OCCUPATION-DISEASES.

TYPHLITIS (τυφλόν, the cæcum).—Inflammation of the cæcum. See APPENDIX VERMIFORMIS, Inflammation of; and INTESTINES, Diseases of.

TYPHOID FEVER (τύφος, stupor).—SYNON.: Enteric Fever; Pythogenic Fever; Gastric Fever; Infantile Remittent Fever; Fr. *Fièvre Typhoïde*; *Fièvre Gastrique*; *Dothiëntérie*; Ger. *Typhus Abdominalis*.

DEFINITION.—A continued fever of long duration; usually attended by diarrhoea; and characterised by peculiar intestinal lesions, an eruption of small rose spots, and enlargement of the spleen.

ÆTIOLOGY.—Typhoid fever is now known to be due to a specific *typhoid-bacillus* which is reproduced in the system during the fever. Its chief outlet is in the intestinal discharges. There is no evidence that it is conveyed by the breath or perspiration; the bacilli, however, have been abundantly found in the urine. While the contagium is present in the fæces and urine, it has not apparently always at the moment of their passage its full virulence, but requires for its complete development a certain period of time, and this is forwarded by some conditions, retarded or prevented by others. Exposure to sunlight and open air, as in flowing rivers, is inimical to the development of the typhoid-microbe, and probably indeed destroys it. On the other hand, warmth, stagnation, seclusion from open air and light, accumulation and concentration of the infected discharges intensify the poison, and it would seem that a small amount of typhoid-evacuations may give rise to a large development of the contagium in excretory matters with which they become mingled, and even in milk to which they may obtain access. This multiplication of the bacilli outside the body explains the autumnal prevalence of typhoid fever observed in large towns, and the association pointed out by Murchison between a hot and dry summer and a high fever-rate in London. The drains not being flushed by abundant rainfall, sewage accumulates and stagnates in them, and typhoid stools never being wanting, the specific fermentation goes on rapidly under the influence of the high temperature, and produces the poison in quantity and intensity. See p. 1191.

The modes in which typhoid fever is disseminated are various. It is not commonly transmitted directly from person to person. Medical men, clergymen, and others visiting those who are suffering are not attacked, nurses very rarely, when proper precautions are observed. If, however, bedclothes or carpets soiled by the evacuations are not removed and disinfected, and still more where gross neglect of cleanliness and decency is permitted, attendants will contract the disease.

Flies may carry the bacilli from urine or fæces or perhaps from the mouth of the patient to food or drink in the sick-room, and thus be the means of communicating the disease from person to person.

The most common vehicle of the poison is drinking-water, which may be contaminated in various ways, mostly through sewage. The water-supply of a town may be thus poisoned at its source, as in the case of a river into which drains empty themselves, or a reservoir or well accidentally contaminated; or the pipes of distribution, when the supply is intermittent, may while empty become charged

with sewer-gases, or may even receive sewage; or excessive rainfall may, in villages and small towns, wash the contents of cesspools into wells. This same rainfall, by flushing the sewers of well-drained towns, and washing away the specific poison, has often a contrary effect in towns and country. In some places the subsoil water, permeating a bed of gravel, is at the same time the well-water of a village and a reservoir of its sewage. An imported case of fever will under these conditions poison almost the entire community. With these examples of wholesale dissemination of typhoid fever must be mentioned the so-called 'milk epidemics' already alluded to. A case of fever occurs at a farm, or among the employés of a dairy; from defective sanitary arrangements the water used at the farm or dairy becomes contaminated by the excreta; this is added to the milk as an adulteration, or, as is usually said, is used in washing out the cans, and in this way the poison obtains access to the milk, where apparently it must increase very rapidly. Hundreds of cases have been traced to a single dairy.

The poison may lurk in various articles of food and drink. The cheap ices sold on barrows in the streets have been shown to be frequently contaminated with typhoid bacilli, and even the cream given with strawberries at a fashionable dinner-party has been known to convey the poison. Oysters and other molluscs, again, have been proved by conclusive evidence to have been the means of disseminating typhoid fever, and an investigation instituted by the Public Health Department of the Local Government Board showed that certain oyster-beds were open to contamination by typhoid excreta. The bacilli, moreover, have been found in oysters.

More commonly, perhaps, the occurrence of typhoid fever is traceable to the absence of proper sanitary arrangements in individual houses. Not to speak of cesspools and leaking drain-pipes allowing the basement to be sodden with sewage, sinks or water-closets may be imperfectly trapped, and sewer-gas diffuses through the apartments or is drawn into the living-rooms by fires, or is forced into the house by pressure in the main drains, or the waste-pipe of the cistern is in direct communication with the drains, and sewer-gases conveyed by it are confined in the space under the cistern-lid, and absorbed by the water used for drinking purposes, which conveys the poison. In connection with these modes of dissemination it should be borne in mind that well-made and close-fitting doors and windows may aid in compelling foul air to enter from the drains or subsoil, and that houses in elevated situations, and thus apparently well placed for drainage, are in greater danger from pressure of gases in the main drains. It is said, however, that the bacilli do not leave the sewage and the moist walls of drain-pipes, and sewer-gas has on examination always been found free from bacilli. This would seem to invalidate the explanation just given, but the fact remains that in some way or other defective drains and leaking soil-pipes give rise to enteric fever.

It is again possible that emanations from a newly opened drain, or cesspool, or foul privy, may communicate the disease by atmospheric contagion—though this is comparatively rare—it being understood always that typhoid excreta form part of the contents. But a drain open to the air throughout

its course, however offensive, is not so likely to give rise to the disease as closed and unventilated sewers; and sewage-farms, if at all well managed, are quite harmless.

It should be added that while, in this country and in Europe generally, all the evidence tends increasingly to confirm the dependence of typhoid fever on dissemination by drinking-water as its chief mode of propagation, observations in India show that the part played by water here is in India played by dust, which conveys the poison in the dry state to articles of food and drink. Contaminated dust has apparently been one of the agencies in the spread of enteric fever in the recent campaigns in South Africa.

It is unnecessary here to discuss Pettenkofer's hypothesis that the varying prevalence of typhoid fever is connected with the varying level of the subsoil water, which as it rises displaces gases which have become saturated with poison from the soil into the atmosphere. It certainly does not apply to the facts as observed in this country.

One word must be said with regard to individual susceptibility to the disease, and with regard to predisposing causes acting on the individual. It is a matter of almost daily observation that some persons never contract typhoid fever, however much they may be exposed to the poison, while others take it readily; and it is almost equally obvious that certain families are extremely susceptible, and liable to have the disease in a severe form. It is always a reason to apprehend a formidable attack if a parent have died of the fever. Typhoid fever may occur at any age, but it is very rare in advanced life. It is probably more common in infancy than is generally supposed, as it is easily overlooked or confounded with common infantile ailments. The period of life at which the disease is most common is during adolescence and the first decade of adult age. Among the predisposing causes are mental depression or shock, over-work, and debility, however induced. A person habitually imbibing the poison may not be attacked until his power of resistance is impaired by some depressing influence. In this way a chill may appear to bring on the disease. It is natural to suppose that unfavourable hygienic conditions would generate a predisposition, but doubt is thrown on this by the fact that typhoid fever does not by any means predominantly affect the poor. The influence of habitual consumption of impure water, again, is not very clear. In some instances it has appeared to make an epidemic severe, but, on the other hand, it would almost seem that the inhabitants of some towns, the water of which is constantly contaminated, acquire an immunity from the disease.

Influenza undoubtedly acts as a predisposing cause of enteric fever, and apparently in a more direct way than simply as a debilitating influence. Beginning with the characteristic symptoms of influenza, the sudden onset, the severe general pains, the rapid rise of temperature, and the prostration, there may be a gradual development of symptoms belonging to typhoid fever without any marked fall of the temperature. In other cases there is a distinct remission of the initial influenza temperature to be followed by the gradual rise commonly seen in enteric fever. In others again there is a brief period of convalescence before the typhoid fever sets in. Influenza opens the door to enteric fever so frequently that there would seem to

be some such relation between these two diseases as between measles and pertussis.

BACTERIOLOGY (by DR. EYRE).—The *Bacillus typhosus* or *Bacillus typhi abdominalis* was first detected by Eberth in 1880, in sections of spleen, Peyer's patches and lymphatic glands of cases of typhoid fever; but was isolated, cultivated and described by Gaffky. During life the organism may be isolated from the faeces (from the sixth day onwards) and urine (from about the twenty-first day) of patients suffering from enterica; but although one observer claims to have obtained it from the blood of the 'rose spots,' the bacillus is not present in the blood of the general circulation. *Post mortem*, the typhoid-bacillus can be demonstrated constantly in the spleen, mesenteric glands and Peyer's patches; and frequently in the liver, heart, muscle and lungs.

Morphology.—The typhoid-bacillus is a short rod with rounded ends, 2μ to 4μ in length, by 0.4μ to 0.8μ in breadth. In artificial cultures, the bacillus is pleomorphic, and long thread forms of 10μ to 20μ

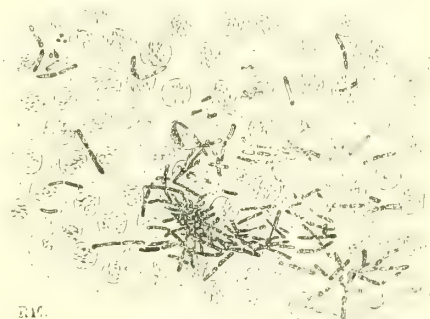


FIG. 1.—Typhoid-Bacilli in section of a swollen Peyer's patch. Showing the usual appearance in stained sections. $\times 950$.

are common. The bacillus is extremely motile (although in old cultivations this character may be almost lost), and possesses from 12 to 20 flagella, arranged at the poles and also laterally. Spore-formation does not take place, but central or ter-

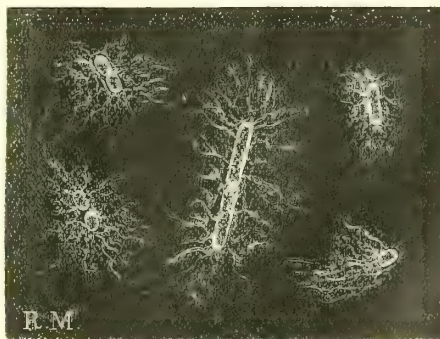


FIG. 2.—Typhoid-Bacilli.—Stained to show cilia. $\times 2,000$ diam. From a specimen prepared in the Berlin Health Laboratory.

minal vacuoles may be noted in the bacilli derived from certain media, e.g. potato. The bacillus stains with the ordinary anilin dyes, usually evenly throughout but occasionally showing polar or irregular distribution of the colour; it is decolourised when treated by Gram's method.

Biological.—The typhoid-bacillus multiplies rapidly under either aerobic or anaerobic conditions, and is readily cultivated upon artificial nutrient media, even those containing a fair amount of free acid. The temperature-limits within which growth takes place are 10° C. and 46° C.; the most luxuriant growth occurs between 35° C. and 42° C.; the thermal death-point is reached at 58° C. The vitality of the organism is maintained for many months upon artificial media. It is killed by exposure to direct sunlight for from four to eight hours, but resists desiccation and freezing for considerable periods. The typhoid-bacillus remains alive in sterilised water for about one month, but dies in about fourteen days in aerated water; and in about the same time in ordinary unsterilised water. It has been recovered after eighteen days in dry garden-earth, and after forty-two days in moist garden-earth, but disappears after seven days in artificially dried soil. In sterilised sewage it also disappears within a few days. The admixture of highly diluted serum from an animal immunised against the *Bacillus typhosus*, or a patient suffering from typhoid fever, with a suspension of the bacilli in some indifferent fluid, causes the organism to become aggregated into masses or clumps, and affords a valuable means of differentiating the typhoid-bacillus from the colon-bacillus. See SERUM-REACTIONS.

Cultural Characters.—On gelatine plate-cultivations incubated at 20° C. the superficial colonies appear at 48 to 72 hours as flat, thin, bluish, translucent plaques, 1 to 3 mm. in diameter, with irregular crenated edges. They increase somewhat in size during the next few days, the central portion of each colony becomes slightly elevated, the surface granular and covered with lines and grooves, resembling the veining on a vine-leaf, the margin sloped and irregular in outline. The bluish translucent tint is retained. The deep colonies are not characteristic, being simply small spherical points, with sharp regular contour, finely granular surface, and yellowish in colour. The gelatine is not liquefied.

A gelatine streak-cultivation shows as a delicate bluish translucent film, with a granular surface and irregular sinuous margins, spreading a little at the bottom of the tube, where the medium is most abundant, but closely restricted to the needle-track at the upper parts. In old cultivations the growth may become more opaque, and iridescent; and deposits of phosphate-crystals make their appearance in the gelatine around and below the growth.

In gelatine stab-culture, small lenticular colonies, which sometimes coalesce, appear along the needle-track, while a semi-transparent filmy disc with scalloped edges develops on the surface.

Shake-cultivations in sugar-gelatine show abundant growth throughout the medium, but no gas-formation occurs.

Bouillon-cultivations incubated at 37° C. show a uniform turbidity within 24 hours, and when shaken present a peculiar shot-silk appearance. After a time some of the growth is precipitated to the bottom of the tube, but the supernatant fluid never becomes quite clear. The nitroso-indol reaction cannot be obtained from cultivations of the typhoid-bacillus.

In agar and glycerine-agar streak-cultivations the growth is rapid and luxuriant, and forms a raised, moist, shining, greyish-white layer, which, how-

ever, does not extend to any great distance from the needle-track.

On inspissated blood-serum, the growth resembles that upon agar; the medium is not liquefied.

In litmus-milk free growth takes place resulting in the production of a faint though distinct acid reaction, but not accompanied by any clotting of the medium.

Upon potatoes of acid reaction, the growth of the typhoid-bacillus is apparent only as a moist patch, but if the reaction of the medium chances to be alkaline, a moist yellowish or buff-coloured layer is formed.

PATHOGENESIS.—The *Bacillus typhosus* is pathogenic for the ordinary laboratory animals, though extreme variations in virulence are observed in 'strains' obtained from different sources, and in nearly every case a series of passages through the serous cavities of a number of animals, assisted perhaps at first by the simultaneous injection of 'killed' cultures of the colon-bacillus or some other indol-producing organism, is essential for the production of a fixed virus. The typhoid-bacillus is pathogenic for the guinea-pig, the mouse, and the rabbit, in the order named, death from general septicæmia resulting after subcutaneous, intrapleural, intraperitoneal or intravenous inoculation of a virulent culture, in from twelve hours to two or three days, according to the dose employed. A very small dose of a highly virulent culture or the inoculation with an attenuated virus results in purulent infiltration of the infected tissues, which either spreads extensively and leads to general infection and death, or is limited in area, producing an eschar, after which recovery ensues. In fatal cases, identical lesions are produced by all forms of inoculation, and consist in sero-fibrinous or sero-purulent exudation into the serous cavities, varying in amount from a few drops to 8 or 10 c.c., and teeming with typhoid-bacilli; hæmorrhagic enlargement of the spleen, which may be almost black in colour; injection of the intestinal vessels, with increased mucous secretion; and tumefaction of Peyer's patches; and hæmorrhagic infiltration of the adrenals and, to a less extent, of the kidneys. The bacilli can be demonstrated in these organs, though in progressively smaller numbers, in the order named. The heart-blood also contains the bacilli, but in very small numbers. Similar anatomical lesions are noted when death follows the subcutaneous injection of virulent toxins obtained from bouillon-cultivations of the *Bacillus typhosus*.

ANATOMICAL CHARACTERS AND PATHOLOGY.—Special interest attaches to the structural lesions which take place in typhoid fever, as they are closely associated with the symptoms, and are accountable for many of the complications which occur. The relation of the intestinal lesions to the febrile process has been differently interpreted. They have been considered, on the one hand, as the focus in which the morbid germs multiply, and from which the blood is infected; and, on the other, the primary change has been supposed to take place in the blood, the lesions being secondary to this, like the pustules in small-pox. In fatal cases local congestions and inflammations are met with in the lungs and other organs, but the special and characteristic lesions are those taking place in the intestines and mesenteric glands. The intestinal mucous membrane of the ileum generally presents the appearances of acute catarrh; but the

chief seat of the morbid changes is Peyer's patches, and the changes consist in a gradual infiltration of the glands here crowded together, followed by ulceration. The process is divisible into three stages—of *infiltration*, *ulceration*, and *separation* or *resolution*, each of which may be said, speaking roughly, to occupy a week. In the first, that of infiltration, the glands of Peyer's patches are swollen and distended by a leucocytic exudation. The entire patch is thickened and raised above the level of the surrounding mucous membrane; has a reddish, or fawn, or grey colour, according to the intensity and stage of the inflammation, and an irregular surface; is firm to the touch; and opaque when the intestine is held up to the light, often showing through the peritoneal covering.

The patches are attacked successively from below upwards; and as they are largest and most numerous at the lower end of the ileum near the ileo-cæcal valve, it is here that the lesions are most extensive and most advanced.

In about a week the follicles begin to ulcerate, or, as it is sometimes said, burst. This marks the beginning of the second stage. As it progresses, the minute ulcerations extend and coalesce, the patch having first a worm-eaten appearance, and later becoming one large ulcer, which may be superficial or deep; in very severe cases the patches slough and fall off as a whole, a deep line of demarcation forming round them. At this period they are stained with bile, or when gangrenous are almost black.

During the third week in mild cases a sort of resolution may occur, the infiltrated material being broken down and absorbed; and this change probably takes place in patches high up in the ileum when in those lower down there is ulcerative destruction. For the most part this stage is occupied by the separation by ulceration or sloughing of the affected patches, and an ulcer is left of corresponding size and shape. Of course, as the patches run longitudinally, along the aspect of the bowel away from the mesenteric attachment, the ulcers also have their long diameter in the same direction. The superficial layer only of the mucous membrane may be ulcerated, or its entire thickness may be destroyed; and where there has been necrosis of a patch as a whole, the muscular coat may be implicated, and even in some cases the peritoneal covering. When the muscular fibres are laid bare, and especially when they are partially destroyed, the base of the ulcer will have a shreddy appearance. Large vessels may be opened, giving rise to hæmorrhage; or perforation of the intestine may take place from necrosis of the serous coat, near the centre of the ulcer, and a perforation formed in this way will usually be large.

At the end of the third week the separation of the diseased patches will be completed, and the ulcerations left begin to granulate. There is for some time a liability to hæmorrhage from erosion of vessels, and perforation may still occur, the apertures now, however, being as a rule minute. Unless perforation has been preceded by adhesion to some neighbouring coil of intestine, which may possibly be the case at this period, the escape of gas and extravasation of liquid fecal matter will set up general peritonitis, which is almost always fatal. The time required for complete healing of the ulcers varies.

Besides the large ulcers formed in the patches of

Peyer, it is not uncommon to find small circular ulcerations scattered over the mucous membrane, and at times minute disseminated ulcers constitute the predominant lesion, Peyer's patches being absent, or, if present, little affected. The large intestine is usually healthy or nearly so, the ileo-cæcal valve forming a sharp demarcation between healthy and diseased mucous membrane, but in some cases there are numerous small ulcerations in the cæcum and colon.

The changes in the mesenteric glands are secondary to those in the intestinal mucous membrane. The glands are enlarged, firm, pink or fawn-coloured, and present on section a corporcular infiltration like that affecting the agminated glands. Later, they become paler and softer, and may gradually return to a normal condition, or may undergo caseation. Ulcerations, superficial or deep, are not uncommonly present in the larynx.

The spleen is almost always much enlarged, dark in colour, and soft.

Granular degeneration of the gland-cells of the liver and kidneys, of the muscular fibres of the heart, and of the voluntary muscles generally, is a constant morbid change. It is due to the prolonged high temperature and the associated toxæmia, and is proportionate to the severity and duration of the fever; when it reaches an advanced stage, it may be the cause of fatal syncope from failure of the heart.

SYMPTOMS.—The period of *incubation* of typhoid fever is not definitely known. There is great difficulty in fixing it, as the date of exposure to the poison can rarely be exactly ascertained, and the onset of the attack is usually insidious. It is certain, however, that the incubation-period is long, probably in most cases from fourteen to twenty-one days. Cases are met with from time to time which suggest that the poison may reside in the system for an indefinite period until the resistance is lowered by a chill, or an attack of influenza, or by great fatigue. On the other hand, instances are on record in which the disease appears to have come on immediately after exposure to powerful emanations from sewers which have burst, or have been opened on account of obstruction, but they are quite exceptional. The incubation is not attended by any marked symptoms; sometimes the appetite falls off, the tongue becomes furred, and there is headache or depression; but, as a rule, the patient is not debarred from his avocation, and there may be no complaint of any kind.

Invasion.—The invasion is almost always insidious. Occasionally severe headache sets in suddenly, with depression, muscular weakness, general pains, and chilliness almost amounting to rigor; but, as a rule, the patient at first feels simply out of sorts, loses appetite, and is indisposed for his usual work or for exertion of any kind. Epistaxis is not uncommon. The headache increases; the prostration becomes greater; sensations of cold down the back, alternating with heat and slight flushing, come on at intervals. At this point medical advice is usually sought, when the patient, giving the above history, will be found to present the ordinary indications of the febrile state—the pulse will be increased in frequency, the temperature raised, and the tongue will have a whitish or yellow coat, thick or thin in different cases. The bowels may be confined or relaxed; the urine high-coloured and diminished in quantity, or so far apparently normal.

In endeavouring at this early period to decide whether the case is one of enteric fever, the first thing to be done is to exclude local inflammation as a cause of the pyrexia. Among the positive indications the appearance of the patient is often a guide; he may look more heavy and oppressed than is accounted for by the temperature or the duration of the illness, and may be more prostrate. The tongue, as a rule, is not thickly coated, and the fur does not extend quite to the margins or tip, which even now may be unduly red. The abdomen may be tumid, and there may be some tenderness over the right iliac fossa, but the absence of these symptoms does not exclude enteric fever. The recurrence two or three times of slight epistaxis during the first few days of a febrile attack would increase the presumption of the disease being typhoid fever. The temperature, however, if it is watched from the first, affords the most conclusive early evidence of the disease; it rises with remarkable regularity from day to day, and is from one to two degrees higher in the evening than in the morning; the appearance of the temperature-chart recording morning and evening observations is highly characteristic, and may, indeed, almost be called diagnostic. The opportunity of watching this gradual rise, however, is often wanting; but if, on the third or fourth day of an illness, without obvious local cause we find a temperature of 103° or 104° F., and especially if the evening rise and morning fall are marked, the probabilities are that the case is one of enteric fever.

As has been stated, the fever is of long duration, lasting from twenty-one to thirty days on an average, and probably the clearest idea of the course and progress of the disease will be conveyed by a brief description of the condition of the patient week by week, during a fairly severe attack.

At the end of the first week the temperature will have reached the level, whatever that may be, which will be maintained, in the absence of complications, throughout the dominant stage of the disease— 103° , 104° , or 105° F. in the evening, 1° or $1\frac{1}{2}^{\circ}$ F. lower in the morning; in mild cases it may not be more than 102° F. when highest. The fever is now well-established, and all its characteristic features will be more or less pronounced. The initial headache will in most cases have disappeared; the patient lies on the back or on either side; the face is flushed, often presenting a bright patch on the cheeks, rarely dusky; the expression is good, though usually suggestive of the disease; the eyes are bright and observant; the skin is more or less hot, usually dry, but often moist at some period of the day. The pulse is frequent, 80, 100, or 120—short, large, and very often dicrotic. A short cough is common; and a scattered sibilus, heard on auscultating the lungs, is so frequent as to constitute a feature of the disease. The tongue will be moist, with a white or yellowish fur thinning off towards the edges and tip; the margins are red, and a red triangle invades the dorsum at the tip. The abdomen will be more or less tumid; and on pressure tenderness may be elicited in the right iliac fossa, and gurgling of liquid and gas may be felt in the intestine. It is to be understood that the gurgling is an incident attendant on diarrhoea, and is absent in cases where there is constipation. The spleen may sometimes be felt below the ribs, and when not palpable in the left hypochondrium may give an

enlarged area of dullness on percussion, but it will not yet have reached its full size. The characteristic spots may occasionally be found, but though they have been seen as early as the fourth or fifth day, they do not usually come out till after a week or more of fever. The bowels will mostly act loosely, and sometimes frequently, so that there is diarrhoea. The urine will be high-coloured and generally turbid; it not infrequently shows a trace of albumin and may contain an appreciable amount. It will not respond to Ehrlich's test. The motions will have a powerful and offensive peculiar odour; they will be copious, liquid, rather pale, yellowish or drab in colour, with flocculi. A familiar descriptive comparison is to 'pea-soup.' The blood should be periodically examined for the Widal reaction. See SERUM-REACTIONS.

A week later—that is, on the fourteenth or fifteenth day—the disease will have told obviously upon the patient. He will lie mostly on his back, and little on his side. The face, still flushed, and presenting often the red patches on the cheeks, will begin to look worn and thin; the eyes, still bright, will be less observant. The aspect and expression of the countenance are so characteristic as to be at once recognised by an experienced observer. The hand may be unsteady when held out, and there may be slight twitchings of the lips. There may be restlessness or even sleeplessness, and very often delirium. It has been stated that delirium mostly sets in about the twelfth day, but the time varies greatly. At first the patient is confused on waking from sleep, does not quite know where he is, and appears to have been dreaming, but soon collects himself; later he fails to shake off the confusion of thought, and he may talk incoherently,—may ramble, in fact, as it is said. At this period the delirium does not go beyond slight rambling, and it is rarely violent at any stage. In severe cases there may be retention of urine.

The pulse will be more frequent, still less full, weaker; dicrotism is rarely well-marked. The lungs will not have undergone much change; the sibilant sounds may be more numerous.

The tongue will be more characteristic; the fur will be represented by a thin yellow or brownish streak down the centre, while the tip and edges will be red and angry. There will be a tendency to dryness, and generally the entire tongue looks shrunken and pointed. See p. 1723.

The abdomen will be larger and more tense, and the tenderness and gurgling in the right iliac fossa more marked; the spleen will be larger. Now the eruption may be expected to present itself, if at all; but it must be remembered that it is absent in a considerable proportion of cases—a proportion estimated by some observers at 30 per cent. The eruption consists of small pink spots, about the size of a pin's head, slightly raised and pointed, well-defined, and disappearing on pressure, or when the skin is stretched, to reappear when the pressure is withdrawn. They are usually few in number, and are distributed irregularly over the abdomen and chest; but sometimes they are very profuse, the number having no relation whatever to the severity or character of the attack. The spots do not all come out at once, but in successive crops till the end of the fever; the duration of individual spots, as observed by surrounding them with a ring of ink and dating them, being about four days. It has been recommended that the back should be searched

for spots, but conclusions from eruption found here only would be untrustworthy, as the back is seldom free from spots, and there is nothing very peculiar in the spots of typhoid fever to distinguish them from others.

If the case is characterised by diarrhoea, it is usually at this period that it begins to be most troublesome; the stools have the appearances already described.

The temperature will be maintained at about the same height as at the end of the first week, but not uncommonly there is about this time a deceptive remission of fever, and it is perhaps worthy of remark that the active ulceration is now coming to an end. The fever which persists after this period has indeed been attributed to septic absorption from the intestinal ulcers—with doubtful justice, however.

It is from the latter part of the second week onward that complications, both local, such as hæmorrhage or perforation from ulceration and separation of sloughs, and general, such as pneumonia, are to be apprehended.

The end of the third week finds the sufferer at his worst. If he is now able to turn in bed and to lie on either side, and if other symptoms correspond with this indication of power, the case is favourable. In a severe attack he will lie on his back, and probably tend to slip down in bed. The face may be either pale or dusky; its look will be that of prostration. The patient is mostly very deaf, often dull of apprehension, so that he is with difficulty made to put out his tongue, and when it is protruded he may keep it in this position when told to withdraw it; he may be half unconscious, or in the condition of coma-vigil, a stuporous sleep with the eyes half open; or he may be restless, with muttering delirium, picking at imaginary objects in the air, or at the bedclothes. Sometimes the slightest pressure on any part of the body appears to give pain, or the patient may start in great alarm when spoken to. The body will be emaciated; the skin thin, pale, dry, and harsh; the muscles wasted; the tendons starting up, from sudden irregular muscular contractions (*subsultus tendinum*) when the limbs are at rest, while attempted movements are attended by jactitation and tremors; the lips tremble when the patient speaks, and the tongue twitches when protruded. A tap with the end of the finger on the pectoral muscle will cause a small swelling to rise on the spot—the so-called ‘myoidema,’ due to contraction of the degenerated fibres; it lasts for twenty or thirty seconds. The tongue is shrivelled, brown or even black, destitute of true epithelium, or dry and shiny, or coated with sticky slime or with black sordes. Sometimes it cannot be protruded at all. The roof of the mouth will be similarly coated; and there will be sordes on the teeth, and perhaps on the lips.

The pulse will be small, soft, extremely weak, often very frequent—130 or upwards per minute in bad cases; the beats run into each other, and are not distinct, giving an undulatory sphygmographic trace. The heart is extremely weak: the impulse absent, or a mere tap; the first sound short and feeble, or altogether extinguished. The lungs exhibit evidence of hypostatic congestion, impairment of resonance, and imperfect entry of air all over the posterior aspect of one or both sides of the chest.

The abdomen will usually be distended; spots may be present and continue to come out. There may or may not be diarrhoea. The stools may be

passed unconsciously; and there may be retention, or incontinence, of urine. Bedsores often form rapidly, unless the nursing is both careful and skilful.

A tendency to recovery is usually indicated by improvement in the temperature, pulse, tongue, and abdomen. The temperature gradually falls, but at first this is shown chiefly by the increasing morning remissions, giving a greater difference between the morning and evening temperatures, and showing on the chart greater zigzags; the evening temperature then begins to fall day by day. The pulse becomes less frequent, and more full and distinct; the tongue gradually cleans, beginning at the margins; the abdomen subsides, the diarrhoea ceases; and strength returns little by little. In fatal cases the mode of death, when not due to some complication, may be by asthenia, or more frequently by coma and asthenia combined; hyperpyrexia is not uncommon as a phenomenon of the moribund condition; and, as the heart fails, hypostatic congestion of the lungs is usually very marked.

Relapses.—Relapse is very common in enteric fever. It usually occurs about ten days after the subsidence of the temperature in the primary attack, and is sometimes attributed to premature administration of solid food, but more commonly has no such cause. A true relapse is not merely a recurrence of pyrexia, but a return of all the phenomena of the fever. Fresh Peyer's patches are attacked, and there is frequently a fresh outbreak of the spots at about the same period after the initial symptoms, or often earlier. The relapse, however, is rarely as long as the original fever; very commonly, indeed, the third stage, that of so-called infective or pyæmic fever, is wanting; and when this is so, it is probable that there is no ulceration of the intestinal glands. Fortunately, also, it is not so often fatal as might be expected, seeing that the patient is reduced by the first attack. A second or a third relapse may take place; the writer has even indeed seen a fourth, the patient recovering and surviving five distinct attacks of fever, each of which was severe. No satisfactory explanation of the relapse of typhoid fever has been given. It has been said that there is a re-infection of the subject by poison lodged in the mesenteric glands; but it is not easy to understand why he should be susceptible to the influence of the poison from this source, when for the most part the susceptibility to the disease is exhausted by an attack.

Special Symptoms and Complications.—These are so numerous and varied that it has been thought better to describe them separately, rather than to interrupt the account of the fever.

Hæmorrhage.—This occurs in about 6 or 8 per cent. of the cases. It may come on as early as the tenth day, but more commonly it is between the fourteenth and twenty-fourth days, and in the later rather than the earlier part of this period; bleeding may be provoked by imprudence in diet, or by exertion later still. The hæmorrhage is due to the erosion of some vessel during the ulceration of Peyer's patches, without protective plugging by fibrin, or to vessels being laid open by the detachment of a slough. The quantity of blood lost, and the rate at which it is poured out, vary greatly. Sometimes the fact of hæmorrhage having taken place is only known by the stools being black, sticky, and offensive. At other times the blood is

discharged in large clots, or it may be extravasated so rapidly that it is liquid and red, not having had time to become blackened and coagulated by the intestinal contents or secretions. When the hæmorrhage is considerable, the patient is rendered pale and pulseless. The temperature always falls, and not infrequently the loss of blood can be recognised before it appears in the stools by this fall of temperature, and by the pulse and general appearance of the patient. Confirmatory evidence may be obtained by careful examination of the cæcum and ascending colon: there will be diminished resonance and the colon will feel heavy when lifted from the loin. It is said that severe and persistent headache early in the attack is often followed by hæmorrhage in a later stage.

This complication is always a source of anxiety, and often exhausts the strength of the sufferer; but it is not by any means necessarily fatal. In many cases, indeed, the occurrence of considerable hæmorrhage marks the setting in of improvement; the temperature which falls on account of the loss of blood does not rise again; the patient becomes clearer and less heavy and oppressed; the tongue cleans; and all unfavourable symptoms gradually subside.

Perforation.—This, like hæmorrhage, may occur early, or at a late period; when early it is due to the entire thickness of the intestinal wall being implicated in the necrosis of the Peyer's patch, and the perforation may then be large. Later, at the end of the third week or afterwards, the perforation results from ulceration, and is often very minute. Unless adhesive inflammation at once glues the affected point to a neighbouring coil of intestine or to the abdominal wall, there is extravasation of intestinal gases and liquid into the peritoneal cavity, producing general and usually fatal peritonitis.

It might be expected that this occurrence would give rise to severe pain, but very often there is neither pain nor tenderness. The abdomen, however, rapidly becomes distended, often to an extreme degree; its walls are absolutely motionless in respiration; and on the application of the hand and making pressure, there is not only tension but resistance of a peculiar kind, which is highly characteristic. With the local symptoms there are evidences of shock: great anxiety of countenance, which may be flushed and beaded with perspiration, or pallid and livid; extreme frequency of pulse, often 140, 160, or 200, which is small and weak; and very great frequency of respiration. The respiratory distress is so marked that, in some instances which the writer has been called to see, the perforation had been overlooked, and the condition attributed to some lung-complication.

Peritonitis.—Peritonitis, without antecedent perforation, is an occasional complication of typhoid fever. It differs from the peritonitis caused by perforation in the more gradual onset, and in the less urgent character both of local signs and general symptoms—of the latter especially.

Tympanites.—Great distension of the abdomen by flatus is not uncommon in the course of typhoid fever, coming on independently of peritonitis or perforation. Most frequently it gradually appears late in the disease, together with other symptoms of prostration of the nervous system, but it may set in abruptly within the first week, and it is then prognostic of an early fatal issue. Simple tympanites is distinguished from peritonitic distension by the

difference in the feel to the hand; but chiefly by the fact that the respiratory movements of the abdomen are not suppressed, though, of course, impeded. Absence of pain is so common in the peritonitis of typhoid fever, that it is no criterion by which to distinguish inflammation from tympanitic distension. See p. 1763.

Diarrhœa.—As has been stated, the bowels are usually loose in typhoid fever. Not infrequently this looseness becomes excessive, and is thus a cause of exhaustion and a source of danger. Unless there is blood in them, the stools are more liquid, frequent, and copious, but not otherwise different from the usual typhoid motions. They may contain curds of undigested milk.

Albuminuria.—Albuminuria is not uncommon as a complication of typhoid fever, and it may be accompanied by convulsions, though it is by no means so dangerous as in typhus. The albuminuria may be the effect of the poison or of the fever-processes on the blood, or it may indicate nephritis. In the former case, which is much more common, the urine does not differ in appearance from this secretion as usually seen at the stage of the disease at which it is present, and the albumen is only detected by examination; in the latter, the urine will be scanty and dark-coloured, as in desquamative nephritis when it is not a complication of typhoid fever.

Pneumonia.—Inflammation of the lung may come on early or late in the course of typhoid fever. When early, it is usually a lobar croupous pneumonia, not differing greatly from ordinary pneumonia; in the later stages it is often a combination of lobular catarrh with hypostatic consolidation. The symptoms are not marked. The temperature rises or is more sustained; there is increased frequency of the pulse and respiration, and perhaps obvious respiratory distress; the face may be flushed or anxious. As a rule, there is little or no cough or expectoration. Examination, when practicable, reveals the ordinary physical signs.

Pulmonary Gangrene.—Gangrene of the lung may occur from obstruction of branches of the pulmonary artery by fibrinous coagula carried from the heart.

Pleurisy.—Pleurisy is occasionally met with as a complication of typhoid fever. It does not give rise to much pain, and may easily be overlooked, the condition of the patient precluding careful examination.

Venous Thrombosis.—Thrombosis may be either a complication or a sequel of typhoid fever. It arises from the languid state of the circulation, and from the condition of the blood, which is liable to coagulate. The femoral vein and its branches are the vessels most commonly obstructed, the thrombosis usually beginning in the small deep-seated branches and extending to the larger trunks, so that there may be deep-seated pain and tenderness in the calf or in the gluteal region for a day or two before the large vein is affected. Thrombosis gives rise to pain and swelling of the limb, with some oedema, and the vein can usually be felt as a solid cord; there is a new access of pyrexia, especially if convalescence has already set in.

Embolism.—Embolism by plugs of fibrin deposited in the heart, and subsequently launched into the arteries, may occur in any part of the body. Hemiplegia may happen from this cause in the

course of typhoid fever; or occasionally, but very rarely, gangrene of part of a limb.

Parotitis.—Parotid abscess is less common in typhoid than in typhus fever, and when present is usually due to secondary infection of the gland by the ordinary pyogenic cocci, although occasionally the organism responsible for the pus-formation is undoubtedly the typhoid-bacillus; but it may occur in very severe cases, and is then an additional source of danger.

Baldness, neuritis, epididymitis, cystitis, perioritis, and perichondritis may occasionally accompany or follow typhoid fever. These conditions are described elsewhere.

Sequelæ.—Enteric fever always leaves the patient weak, and the debility lasts long. This is due not merely to the waste of tissue by the protracted fever, but to the fact that the intestinal and mesenteric lesions interfere with absorption of nutrient material. In some cases the patient never recovers strength, but gradually becomes emaciated, and dies from asthenia or from some intercurrent attack. Occasionally the ulcerations do not heal, and they may prove fatal after the lapse of a considerable time. Phthisis, again, may be started by an attack of typhoid fever, usually, but not invariably, in an individual predisposed to the disease. Insanity is another occasional sequel; the most common form of mental derangement is melancholia, but there may be acute mania. It may come on almost immediately, the patient never appearing quite to recover his faculties after the stupor of a severe attack; or it may develop itself at an early or late stage of convalescence, or not till a still later period.

VARIETIES.—Typhoid fever runs a very varied course, and different epidemics may be characterised by special features as well as by differences in the degree of severity. The state of health or general condition of the individual or of the community may have an important influence in respect of this, but just as the virulence of the bacillus may be intensified or moderated in the laboratory, so it may be modified by climatic and other influences. The typhoid fever of this country, of the continent of India, and of Africa, may thus come to present important clinical diversities. To complete the account of typhoid fever, some of the principal deviations from the ordinary course and type of the disease must be enumerated; and in the first place it must be understood that, both in individual cases and in entire epidemics, typhoid fever may run its course without rise of temperature, the characteristic lesions being found after death. Again, there is sometimes so little general depression of strength that the sufferer may walk about, and carry on his usual avocations up to a late period of the attack. The term *typhus ambulans*, or *ambulatorius*, has been applied to such cases. The writer has known several instances in which patients have *walked* into the London Fever Hospital with perforation.

Infantile Remittent Fever.—Infantile remittent fever is so called from the remissions often observed in typhoid fever affecting young children. No special description is necessary, the disease pursuing much the same course in children as in adults, only with greater fluctuations. Spots are less frequently seen.

Typhoid Fever with Constipation.—This really constitutes a distinct variety, the constipation persisting throughout, and not merely lasting for a

few days and then giving place to diarrhœa. It is a difficult form of the disease to deal with, and is often followed by relapse.

Bilious Fever (*Fièvre bilieuse*).—This is recognised as a special form of typhoid fever by French observers, and deservedly so. The characteristic feature is frequent, sometimes almost incessant, vomiting of liquid containing bile, both after food and when no food has been taken. Severe headache often persists throughout the attack. The temperature does not, as a rule, rise high, and it fluctuates much; the face is pale and anxious; the pulse is frequent and very weak; the tongue is usually coated, and may be white or yellow. It is a very dangerous form of the disease, and may end fatally in ten or fourteen days by simple asthenia, without delirium or comatose symptoms.

Typhoid Fever with Meningitis.—True meningitis sometimes, though rarely, occurs at an early period of typhoid fever, and is attended by pain in the head, excitement, which may be maniacal in character, or violent delirium.

Abortive Typhoid.—This is sometimes described as ‘fourteen-day fever.’ After well-marked symptoms of the attack, the temperature is not maintained, but gradually subsides. Such cases are mostly set down as common continued fever, or febricula. See *FEBRICULA*.

DIAGNOSIS.—A well-marked case of enteric fever at the height of the disease is easily recognised by the aspect of the patient, and by the symptoms already described: the red tip and edges of the tongue, the tumid abdomen, the enlarged spleen, the rose spots, and the character of the stools. In the early stage, however, it is often necessary to suspend the judgment for a day or two, and there may be difficulty in forming a definite opinion for a much longer period. The diseases which have most frequently been taken for typhoid fever are tubercular meningitis, acute pulmonary tuberculosis, and gastro-intestinal catarrh; typhlitis, catarrhal pneumonia and other acute affections of the lungs, glands, pyæmia, and ulcerative endocarditis, have also from time to time been confounded with it. On the other hand, typhoid fever may possibly be taken for one of these affections, or may more easily be masked by some complication, such as pneumonia or peritonitis. What is most liable to happen is, that it may be entirely overlooked.

The insidious onset of typhoid fever is very characteristic; and, as has been said before, the thermometric chart of the first four days, showing a rise day by day with the morning remissions, might of itself suggest the diagnosis. But occasionally the invasion is abrupt, or there is some pulmonary complication at the outset, which raises the initial temperature, so that it would be unsafe to rely too implicitly on the temperature. The first thing to be done in establishing a diagnosis of enteric fever is to exclude local inflammation as a cause of the febrile condition, which will be effected by physical examination and other means. It must not be forgotten during this investigation that in pneumonia the consolidation may not occur for three or four days. When there is doubt, epistaxis will be evidence in favour of, herpes labialis against, such a diagnosis, though herpes may break out when there has been a rigor. The presumption of typhoid fever arrived at by excluding local

inflammation and other acute diseases will soon be strengthened by the appearance of corroborative indications which we need not again specify, or be overthrown by their continued absence.

As regards the particular diseases enumerated as those with which it is specially liable to be confounded—in tubercular meningitis the temperature is not usually so high as in typhoid fever, the pulse is at first not very frequent, and is often hesitating, while it almost always presents the condition of tension in the early stage when the confusion is liable to occur, thus contrasting with the soft short pulse of fever. There are usually, but not constantly, headache and vomiting early in the attack, the bowels are mostly confined, and the abdominal wall is retracted. Squint, inequality of the pupils, or double optic neuritis, would be unmistakable indications of meningitis. In acute pulmonary tuberculosis there is more cough; and although at first there may be only scattered sibilant sounds, such as are heard in typhoid fever, these soon become more abundant, and other physical signs of the infiltration of the lungs are added, such as impairment of resonance, and imperfect entry of air. The temperature also is usually more sustained. Gastro-intestinal catarrh in children is sometimes attended by so much febrile disturbance as to give rise to a suspicion, or even a diagnosis, of typhoid fever, especially as the abdomen is tumid, and there may be diarrhoea; overfeeding with milk may keep up the appearance of fever for some time. There are, however, intermissions and variations which, when carefully noted, are found to be inconsistent with continued fever; the tongue is more thickly and coarsely coated; and the stools, though they may be pale from undigested milk, have not the typhoid character. In typhlitis there is more local pain and tenderness, and a lower temperature; and the symptoms set in more abruptly, vomiting being common. It is unnecessary to enter upon the distinction between typhoid fever and the other diseases mentioned; for the most part a few days will clear up any uncertainty or confusion.

As a matter of fact, errors rarely arise, when it is deliberately considered whether a given case is or is not one of typhoid fever. The danger is that the idea of fever may not be entertained at all, its symptoms being attributed to some slight local ailment. Or there is pneumonia or peritonitis as a complication, when the patient first comes under observation, which is not very uncommon among the poor or in hospital practice. The possibility, therefore, that typhoid fever may underlie an acute local affection should always be borne in mind, and when the desired crisis does not come on in pneumonia, or if in peritonitis the general symptoms are not altogether those of inflammation of the peritoneum, indications of enteric fever should be carefully looked for. When, again, a patient complaining only of some functional derangement, or of weakness and loss of appetite, has a look of illness and prostration which is disproportionate to the assigned cause, enteric fever should be suspected.

There is often great difficulty in distinguishing *infantile* remittent fever from tubercular meningitis on the one hand, and from gastric catarrh on the other. In gastric catarrh the tongue is more loaded; the temperature is less sustained and more irregular; constipation is more common than diarrhoea; and the evacuations, whether solid or liquid, have not

the specific characters, but consist more or undigested food. In meningitis there will usually be purposeless vomiting, but not always; the temperature is not so high; the pulse is often slow or irregular, and the respiration shallow and sighing; the abdomen is not distended, and may be retracted; the bowels are confined. A squint would at once confirm suspicions of meningitis. Ehrlich's urinary test¹ and the Widal reaction in the blood will often aid in the formation of a definite diagnosis, and these examinations should never be omitted, although the responses are not absolutely conclusive. Clumping of the bacilli may take place as the result of an antecedent attack, and the writer has seen a characteristic Ehrlich reaction in a case of septicæmia with abscesses.

PROGNOSIS.—It is never safe to speak confidently of the recovery of a case of typhoid fever, in view of the complications which may arise; but the prognosis is favourable or unfavourable, according to the antecedent condition of the patient, and to the severity of the attack, as estimated chiefly by the temperature. The mortality varies from 15 to 25 per cent., but there may be epidemics in which it may be throughout lower or higher than the average here stated.

Children seldom die of typhoid fever, and in the young the attack is less likely to be severe, and there is a better chance of recovery even when it is. In debilitated subjects, and especially in persons addicted to alcohol, typhoid fever is always attended by danger; even in a mild attack there may be failure of the heart, and pulmonary complications or thrombosis, which may prove fatal. Pregnancy also renders an attack dangerous (*see* p. 1313). Apart from causes of anxiety in the general condition of the patient, and exception being made of cases in which the fever assumes the bilious type, attended by frequent vomiting of bile, headache, and prostration, and of the rare instances of meningitis as an early complication, the prognosis turns mainly on the temperature. If this is not more than 102·5° F. in the evening at the end of the first week, there is very little danger; on the other hand, if the evening temperature reaches 105° F., nearly half the cases prove fatal. When the temperature ranges high, it is of great importance to ascertain whether it is so for many consecutive hours, or only for a short period; a heat of 104° F. sustained for a great part of the day is a more serious matter than a brief rise to 105° F. On this account, and also because the maximum may be attained at different periods of the day, it is desirable that the temperature should

¹ Ehrlich's reaction or the diazo-reaction depends upon the temporary presence (between the 5th and 22nd days), in the urine of typhoid patients, of a chromogen, which when treated with diazo-benzene-sulphonic acid and ammonia gives a characteristic colour-reaction.

In applying this test, the sulphonie-acid reagent is prepared by mixing the two following stock solutions:

1. Hydrochloric acid 5 c.cm., diluted with distilled water to 100 c.cm., and saturated with sulphanilic acid.
2. Sodium nitrite, 0·5 per cent. aqueous solution, in the proportion of 40 parts of the first to one part of the second.

Two or three cubic centimetres of the suspected urine are added to an equal quantity of the sulphonie-acid reagent, in a narrow test-tube. Ammonia is then carefully run in so as to form a colourless zone above the mixture of urine and acid: this results in the formation of a more or less deeply coloured ring at the junction of the two liquids. If now the contents of the tube are poured into a porcelain basin containing some water, the production of a salmon-red colour will indicate a positive reaction; a yellow or orange colour only being obtained when the result is negative.

be taken in serious cases every two or three hours, or even more frequently. In typhoid fever, as in other acute diseases, a contrast between the surface-temperature and the temperature of the blood, as revealed by the thermometer, is of unfavourable significance. If with high temperature there are indications of failure either in the nervous system or in the heart, the prognosis becomes serious; a tendency to stupor or retention of urine early in the disease is a bad sign; and acute tympanites at this period, indicating, as it does, paralysis of the muscular walls of the intestine, and presumably of the sympathetic nervous system, almost invariably points to a speedily fatal termination; the later gradual distension of the abdomen ranks with the unfavourable signs, but is often met with in cases which recover.

Unusual frequency of the pulse, marked diastole early, and extreme weakness of the beat and compressibility of the vessel later, intimate danger, as does also an increasing frequency day by day towards the end of the third week. When the beats run into each other, and the pulse is a mere flutter, the danger is immediate and extreme. The sounds of the heart should be noted throughout the disease; the first sound, as heard at the apex, tends to become, first, short and sharp, then weak, and at last may altogether cease to be audible; a good or bad first sound is of good or bad augury respectively. A systolic apex-murmur is not uncommon, and occasionally a distinct presystolic murmur is heard; these murmurs have no great significance, and usually disappear as the patient recovers.

TREATMENT.—The principles on which the treatment of typhoid fever should be conducted are generally accepted and well understood. Success depends greatly on their intelligent application to individual cases, and on careful attention to details at every stage of the disease.

The patient should, if possible, be placed in a large, airy, and well-ventilated room, the windows and door of which should be more or less continually open, according to the season and weather. The bed should not be too wide, and it should be approachable on both sides, so that the patient can be easily reached from either side; it should be firm, but comfortable. A feather- or flock-bed is very objectionable, on account of the hollow into which the patient sinks in it. When practicable, it is a great comfort to have two beds—one for day, the other for night. The covering should be light, but sufficient to protect the patient from changes in the external temperature; eider-down quilting, or any material impervious to the insensible perspiration, should be forbidden; the under-surface of such coverings will often be found quite damp, and exhaling an offensive odour. Conscientious, skilful, and efficient nursing is of the utmost consequence; and that nothing may be overlooked, a record should be kept of the condition of the patient, of the food, stimulants, and medicines administered, and of the evacuations passed.

The patient should be sponged night and morning with tepid water, to which a little vinegar, or eau-de-Cologne, or permanganate of potassium may be added. The temperature of the water may vary, the feelings of the patient being consulted, and the effects in producing sleep and quiet noted. The hands and face may be washed or bathed frequently, and the general sponging may be repeated

at any time when it seems to be required owing to heat or restlessness.

It is useful to habituate the patient to the use of the bed-pan from the first. It is true that very often the chair or utensil is used throughout; but in severe cases, or on the occurrence of hæmorrhage, the sitting posture is dangerous, and indeed impossible, and it may be most distressing to have to pass the excretions into the bed-pan for the first time under circumstances of extreme prostration; this may, indeed, destroy the patient's chance of recovery. A disinfectant solution should be placed in the pan or other vessel into which the stools are received, and more should be added and left in contact with them before they are thrown away. The urine should also be carefully disinfected in a similar manner. *See* DISINFECTION.

Perhaps the most important element in the treatment of typhoid fever is the regulation of the diet. This should be exclusively liquid, and the staple constituents will be milk, and beef-tea or broths of one kind or another. It should be borne in mind that these liquids are food, and not mere drinks, and they should be given with strict regularity. Two or three pints of milk, and a pint or pint and a-half of beef-tea or the equivalent of broth of some kind will be about the quantity required for twenty-four hours' consumption in the first instance; and it should be so divided that milk and beef-tea are given alternately about every three hours, judgment being exercised in waking up the patient if he is sleeping when food is due, or allowing him to sleep beyond the hour. The great tendency now, on the part of the public, is to overfeed cases of fever. When the patient asks for drink, milk is offered, and if it is iced or diluted with soda- or seltzer-water it is sufficiently grateful in quenching thirst to be accepted in quantity altogether beyond the digestive powers; it then coagulates in masses, escapes solution by the gastric and pancreatic juices, and passes down the intestine in heavy curds, which irritate the ulcerated surfaces, besides producing other disturbances. It is in this way that milk has been discredited as an article of diet. The writer's experience is in accord with that of Parkes, who looked upon milk as the typical diet for enteric fever. In later stages of the disease it may be necessary to give nourishment more frequently, but the attention of the medical attendant will in most cases be required rather to moderate the amount of food given than to urge its administration; it may, however, be found necessary to take precautions against neglect in this respect during the night. In emergencies, concentrated meat-extracts may have to be given in teaspoonfuls every few minutes. The patient should, of course, be allowed to drink freely of cold water, toast-water, or any simple drink. The key to the regulation of the diet—it may almost be said to the management of the patient—is to be found in the careful inspection of the stools; the medical attendant ought to see every evacuation, or, at any rate, one motion every day, supplementing his own information by the report of a trustworthy nurse. When curds appear in the dejections, either too much milk is taken, or too much at a time, or its digestion is interfered with. If the passage of undigested milk is not remedied, there will certainly be flatulence, discomfort, and restlessness, elevation of the temperature, and in most cases diarrhoea. When, without error in the administration of the

milk, curds appear in the stools, dilution with soda- or seltzer-water, or the addition of lime-water or bicarbonate of sodium, may prevent premature and unduly firm coagulation; or the admixture of arrowroot, barley-water, or gelatine may cause the curds to be subdivided, and thus ensure their digestion. A more efficacious method, however, is to peptonise the milk (*see* PEPTONISED FOOD). In some cases beef-tea excites diarrhoea, and, given in excess, it will almost always have this effect. Chicken-broth should be substituted when beef-tea disagrees, and it is often more grateful to the patient.

As the fever subsides, eggs beaten up, or lightly boiled or poached, may be added to the dietary. In all cases the return to ordinary diet must be made with great caution, bearing in mind the fact that there may be intestinal ulcerations unhealed, and it should be a rule that no really solid food be given till the temperature has been normal for a week; even at the end of this time it is not uncommon for the temperature to rise one or two degrees, after a moderate amount of fish or meat with bread has been taken. These rules have been pronounced to be antiquated and the precautions unnecessary, and it is said that food of a semi-solid character may be given throughout the fever, and that the returning appetite may be at once gratified. The advantage claimed for this departure from the cautious administration of nourishment so long recommended is that the period of convalescence is shortened. It has yet to be seen how far any such advantage is real, and whether it may not be too dearly purchased.

It is an unfortunate aggravation of the sufferings in typhoid fever that fruit, which is so grateful when the mouth is dry and parched, cannot be given freely, on account of its liability to excite diarrhoea; but a few grapes may be permitted, care being taken that the skins and seeds are not swallowed, and the effects being carefully watched.

The question of stimulants is an important one. Here, again, the prepossession in the public mind is in the direction of almost indiscriminate administration of alcohol, and the importunities of friends may have to be resisted. The amount of brandy or wine given must therefore be carefully checked and controlled. In a large proportion of cases no alcohol is necessary from first to last; it is scarcely ever required in the early stages of the disease, except perhaps in drunkards; and at no period should it be given as a matter of routine, or merely because the case is one of fever, but only to meet certain definite indications. These are mainly evidences of weakness of the heart, frequent, weak, and fluttering pulse, and weakness or absence of the first sound of the heart. When, as is usually the case, the tongue is also dry, and the teeth and lips are covered and the mouth lined with sordes, the indications for the use of stimulants are unmistakable. The effects should be watched; when alcohol does good the pulse becomes less frequent, and of better strength and volume, and the temperature is usually lowered; an important indication also is that the odour of spirit is not detected in the breath. When very high temperature and other unfavourable prognostic symptoms set in very early, stimulants may be given without waiting for the conditions above-mentioned. Alcohol is again often required as an adjunct to the treatment of fever by the cold bath. The safest form of stimulant is

brandy or whisky; the quantity needed will vary greatly in different cases; in most two or three ounces in the twenty-four hours will be sufficient, in some ten or twelve ounces may be required. It is scarcely ever necessary or useful to go beyond this amount, and the administration of alcohol should always be checked when it can be smelt in the breath. It should be given in divided doses in, or immediately after, the milk or beef-tea.

It will be convenient to say here a word on the use of opium. Its employment in certain complications—tympanites, peritonitis with or without perforation, hæmorrhage, and excessive diarrhoea—will be described later; the question now is whether it is well to give it for the relief of sleeplessness and restlessness. In the writer's opinion, when the restlessness is not so far allayed by cold or tepid sponging as to permit of sleep, it is of great advantage to the patient to give ten or fifteen minims of laudanum at night, or its equivalent in some other form. The writer has not found it to interfere with the digestion.

In a large proportion of cases no medicine need be given from first to last, but effervescent salines are usually grateful; the mineral acids, at one time very largely employed, often seem in small doses to do good; and one or two grains of quinine may frequently be given with advantage. It should be understood that medicines are of less importance than food, and that they are not to be allowed to interfere with its regular administration. A gentle aperient, calomel or grey powder followed by a mild saline draught, at the outset, is often useful, but it may be blamed for subsequent diarrhoea. In Germany and America the treatment is often begun by administering six or eight grains of calomel in two doses, and at an early period of the disease this is more likely to do good than harm, though the dose is unnecessarily large.

Daily washing out of the large intestine by copious enemata of some weak antiseptic solution—permanganate of potassium, boric acid, carbolic acid, or sulphocarbonate of sodium—apparently exercises a favourable influence on the course of the disease. The temperature of the enema may be varied so as to have some effect on the temperature of the body.

When treatment, other than the careful watching, nursing, and feeding which are sufficient in mild cases, is required, this should not be a mere matter of routine; and it may conduce to precision in meeting the various indications presented from time to time if an attempt is made to classify and distinguish them.

We can recognise, then, in the course of typhoid fever, effects attributable (1) to the action of the poison; (2) to the intestinal lesions; (3) to protracted high temperature.

1. The entire fever-process, with all its concomitants, is of course due to the action of the specific microbe, but certain effects appear to have a more direct relation with the poison than others. The immediate agents in the production of nearly all, whether direct or indirect, is a ptomaine secreted or formed out of disintegrated proteids by the typhoid-bacillus; and an important question bearing on treatment is whether the toxic material is formed entirely in the intestinal canal or also in the blood and tissues. It is apparently on the first of these two suppositions that the employment of mercury, iodine, carbolic acid, sulpho-carbolates,

alol, salicylates, and other intestinal disinfectants suggested by the theory of the bacterial origin of typhoid fever, has been based. Good results have undoubtedly been obtained, but not such as to invalidate the long-established conclusion that it is not within the power of medicinal agencies to cut short an attack of typhoid fever, or even effectually to modify its course. Undoubtedly ptomaines are formed in the small intestine, and this not only by the typhoid-bacillus, but by other bacteria which find a congenial soil in the albuminoid exudations poured out by the irritated mucous membrane. It has seemed to the writer that this is the explanation of the dark and peculiarly offensive stools sometimes met with in the early stage of typhoid fever accompanied by a high temperature, and of the extraordinary oppression of the nervous system and depression of the circulation occasionally seen without high temperature, or distension or tenderness of the abdomen, or diarrhœa, or other indication of a severe attack.

These are, therefore, the cases in which intestinal antiseptics are indicated; and, according to the writer's experience, mercurial preparations are the most trustworthy. When the motions are specially offensive and accompanied by much gas, the abdomen being tumid and the temperature high, from a half to one fluid drachm of the solution of perchloride of mercury, with perhaps one or two grains of quinine, may be given every four hours for two or three days with excellent effects, or calomel in small doses of one-sixth to one-third may be given every three or four hours. When the nervous system is evidently overwhelmed by poison, while the abdominal symptoms are slight, the local lesions may be disregarded, and two or three grains of calomel may be administered once or on two successive days. The entire aspect of a case threatening to prove fatal may sometimes be changed by thus clearing out and disinfecting the intestinal canal. It will be remembered that two or three grains of grey powder were formerly given three times a day throughout the early part of the attack.

The formation of ptomaines in the blood and tissues is little, if at all, influenced by remedies. The chief and most characteristic effect is the rise of temperature; and this, when not aggravated by adventitious causes, is not controlled by antiseptics of any kind.

2. Severity of the intestinal lesions, as indicated by marked tenderness in the right iliac region, tumidity and tension of the abdomen, and diarrhœa, may call for opiate fomentations, and, when the motions are unduly frequent, for starch-and-opium enemata. Careful examination of all the stools is specially necessary in case of irritation by undigested milk, and for the early detection of sloughs and blood-clots which may give warning of hæmorrhage.

3. The great source of danger in typhoid fever is the prolonged high temperature; and it is to this, together with the fever-poison or process, that are due the prostration of the nervous system and the weakness of the heart which are the most frequent causes of death. To keep down, therefore, the febrile heat of the body is to diminish very greatly the danger attending this disease. It was natural and inevitable that the remarkable class of drugs which have been found to have the power of lowering the temperature—acetanilide, phenazone, phenacetin, and the like—should be eagerly employed for

this purpose in typhoid fever; but while an immediate effect can be obtained, it is only fugitive, and a continued administration is usually disastrous. The most trustworthy means of controlling the temperature is cold bathing. The cold-water treatment of fever was, as is well known, originated by James Currie, and practised with marked success by him and many other distinguished and trustworthy physicians. Notwithstanding results obviously good, it fell into disuse, till revived by Brand of Stettin. Brand's method is to place the patient in water at a temperature of 65° or 70° F. whenever the temperature of the body, as taken in the rectum, reaches 102·2°, and to keep him there for ten, fifteen, or twenty-five minutes, until the heat is reduced 2°, or at any rate until he has been shivering for some minutes. Liebermeister takes 103° as the temperature which requires the bath, and this he does not make quite so cold, that is, 75° F., while he recommends ten minutes only as the period of immersion. The bath has to be repeated three, six, or eight times a day—as often indeed as the heat of the body rises to the point named; and it must be persevered with for two or three weeks or more, as may be required. A little brandy is given before or during the bath; and when the patient is taken out of the water he is placed in bed, dried, covered up, but not too warmly, and kept at rest (*see* HYDROTHERAPEUTICS). In order that the full benefit may be obtained from this treatment, it must be begun early, in which case the temperature need never be allowed to reach an injurious height; and it is claimed that the intestinal lesions are also held in check. When thus carried out, it must be taken as established that the mortality of enteric fever is very greatly reduced by the bathing. Jürgensen's statistics show a reduction from 15·4 per cent. to 3·1; Liebermeister's from 27·3 to 8·2 (this observer, however, employing quinine largely); others show a still larger fall; and it may be safely estimated that in France and Germany the deaths have been diminished by at least one-half. Cayley has shown that like results are to be expected in this country, and the writer's own experience is corroborative of his conclusions. If, however, the remarkable success obtained by Barr of Liverpool by the continuous bath—the patient being kept in water at a regulated temperature throughout the attack—is confirmed by general experience, this method ought to supersede all other treatment. Against the advantages of cold bathing are to be set off the difficulty of carrying it out, and the labour it involves; the prejudices of the public, and the dislike and dread of some patients; the facts that some cases may be protracted (which, by the way, might otherwise end fatally); and that relapse is certainly more frequent. These are good reasons for trying whether the same end may not be attained by other means; but unless we are successful in this, the duty of the medical attendant is to insist on the uncompromising employment of the cold bath. The graduated bath, in which the initial temperature of the water is 90° F., or, as the writer has found to answer the purpose equally well, 80°, is often more acceptable to patients, the water being cooled down rapidly to 70° or 65°. The good effects of bathing are not due simply to the abstraction of heat, a favourable influence is exerted on the nervous system; and if the fever be high, and the nervous prostration great—conditions in which the shock of sudden cold is of great service—the

cold water added to bring down the temperature may be poured over the patient's head. The cold wet pack and Thornton's ice-cap have been tried as alternatives to the bath, but without effects adequate to the requirements of the case. A valuable expedient is to apply over the entire abdomen wet lint or thin flannel, upon which small pieces of ice are distributed. A cage will be necessary over the body, and the water from the melting ice must not be allowed to saturate the bed. It has been hoped to prolong the effects of the bath, and so render frequent repetition unnecessary, by the administration of large doses of quinine or salicylic acid. Quinine, to keep down the temperature for any considerable time, must be given in large doses—thirty or forty grains; and even this amount will, as a rule, produce no decided impression unless the way has been cleared by a bath. It is to be given, then, shortly after a bath in two or three equal portions within the space of about half an hour, and a little laudanum may be given with, or just before the quinine, to prevent vomiting. Very frequently the temperature remains depressed for twenty-four hours afterwards, sometimes even for a longer period. The patient often suffers from severe symptoms of cinchonism, but in view of the advantage obtained this need not be seriously regarded. Sometimes, however, violent and protracted vomiting is set up, which is an absolute bar to the further employment of quinine, except hypodermically. The neutral sulphate is the most convenient form for this purpose, and it is sufficiently soluble to be given in adequate doses, that is, of five or six grains. The writer's own experience tends to the conclusion that the frequent bathing has advantages over the combination of quinine with the bath, both as regards the safety and the comfort of the patient; though he has also seen cases in which the bath alone seemed inadequate, while with the aid of quinine the fever was kept down. He has twice seen severe tetanus produced by the bath and quinine, both cases, however, recovering. Salicylic acid and the salicylates have appeared to him to have a dangerously depressing effect. Of digitalis, employed in large doses as an antipyretic, he has had no experience.

Without going so far as to say that the cold or graduated bath should be employed in all cases, the writer is of opinion that many lives would be saved were cold-bathing at once put in practice, whenever a temperature of 103.5° or 104° F. in the first few days shows that the attack is of more than average severity; and it is of the greatest importance that this should be done early, so that the pyrexia may never get the upper hand, and that the intestinal lesions may, if such a thing is possible, be modified. And no patient should be deprived of the chance which is afforded by the bath when, at any stage of the disease, life is threatened by hyperpyrexia or by consequences of high temperature, such as violent excitement, sleeplessness, restlessness, or nervous prostration. In such emergencies it may be necessary to plunge the patient into ice-cold water. The only complications which render the bath inadmissible are hæmorrhage, peritonitis, and the advanced cardiac weakness and degeneration sometimes found late in the disease. Albuminuria or pulmonary affections do not constitute a bar to cold bathing. The writer has known albumen to disappear from the urine at once, and pulmonary congestion to clear up after a single bath.

Treatment of Complications.—In severe and protracted cases a state of extreme prostration ensues which must be combated by special therapeutic measures. A feature of this condition is great frequency and weakness of the pulse, with loss of tension, so that the beats run into each other. Here digitalis may be of very great service, not only by diminishing the frequency and increasing the strength of the heart's action, but by giving tone to the vessels. Probably, however, the most valuable resource is the hypodermic injection of strychnine, which may undoubtedly save life. One-sixtieth to one-thirtieth of a grain may be given every three or four hours as long as necessary, the effect being carefully watched. Digitalin or ether, or both, may be combined with strychnine. Strychnine hypodermically is again the best remedy in the sudden intercurrent attacks of prostration which sometimes occur in the course of fever. The most careful watch should be kept for complications at all stages of the fever, but especially after the end of the second week. A rise of temperature must always be taken to have some definite significance requiring explanation. With increased frequency of respiration, it may be the sole indication of the accession of pneumonia or pleurisy. A fall of temperature may give warning of hæmorrhage. Retention of urine, though uncommon in typhoid as compared with typhus fever, should always be borne in mind; and unusual restlessness should at once suggest an examination of the hypogastrium. The passage of a catheter has often put an end to sleeplessness and excitement. Bed-sores ought never to occur, and it will conduce to their prevention for the medical attendant to inspect the sacral region and other parts where they are liable to be produced.

Hæmorrhage.—In the treatment of hæmorrhage the first thing to be done is to arrest the peristaltic action of the bowels by a full dose of opium; and as an immediate effect is required, one of the liquid preparations should be given—the tincture or liquid extract, and the dose may be from twenty minims to a drachm. The bleeding vessel has to be closed by a clot, and peristaltic movements will tend to disturb this, while it must be remembered that the blood poured out is a continued provocative of movements on the part of the intestine. Direct astringents which depend on local action are useless; before they could travel the whole length of the small intestine, and reach the bleeding point, they would be neutralised by combining with the intestinal contents. Physiological hæmostatics may be of more service. Ergotine hypodermically, or alternate doses every hour or every two hours of the liquid extract of ergot, ʒss to ʒi, and of turpentine m℥x-xv, have seemed to the writer to have most effect. Acetate of lead and opium or morphine, sulphuric acid and decoction of logwood, tannic acid, and all the known styptics have been recommended. An important adjunct—probably more effectual than any internal remedy except opium—is an ice-bag over the region of the cæcum: this at the same time quiets peristalsis and contracts the vessels.

Stimulants must be given if the patient is in danger of dying from syncope, but it must be borne in mind that fainting gives time for the vessels to close; and until the bleeding has stopped, this condition must not be too diligently averted. We must, in fact, sail as close to the wind as is consistent with safety. The same considerations apply

to the administration of food: very little, if any, should be given by the mouth for twenty-four hours, nourishment being supplied by small nutrient enemata.

Perforation.—The only medicinal treatment likely to be of service is the administration of large doses (ʒj or more) of laudanum or the liquid extract of opium or an equivalent of morphine, hypodermically or by the mouth. This has saved life in a few cases, but in the immense majority of cases a fatal termination speedily ensues. Laparotomy may also be proposed. Many cases are now on record in which it has been successful.

Peritonitis.—Here, again, opium in repeated moderate doses is the most useful remedy; with poultices applied over the abdomen.

Tympanites.—Once more the remedy is opium, which should be given in pill three or four times a day when the distension is late and gradual; in very large doses of some liquid preparation when it is sudden, and especially if early in the disease. Charcoal has been recommended, but in the writer's experience it has rarely been other than hurtful.

Diarrhœa.—As long as the stools do not exceed three a day, or while they do not appear to distress or exhaust the patient, nothing special need be done to check the diarrhœa. It must always be borne in mind that beef-tea or other strong flesh-juices may excite diarrhœa: undigested curds of milk may have the same effect. As soon as undue frequency in the action of the bowels is observed, any possible cause in the diet should be eliminated; this failing, the best remedy is an opiate enema, twenty or thirty drops of laudanum in two ounces of thin starch. In most cases two or three enemata will arrest the diarrhœa; should this not happen, astringents must be given by the mouth—acetate of lead and opium or morphine, sulphuric acid, laudanum, logwood, or tannic acid.

Constipation.—This, whether intercurrent when there has been diarrhœa, or present throughout the fever, is a perplexing symptom. It is best relieved by enemata given every other day, but sometimes these are insufficient: a teaspoonful of castor oil may then be given, care being taken that any accumulation in the rectum which might give rise to difficulty of defæcation is previously removed by enema.

Bilious Vomiting.—When this is present from an early period of the attack, attended as it usually is by great prostration, it has seemed to the writer that life is endangered by the formation and absorption of some poisonous ptomaine, and that the intestinal lesions are then of secondary consequence. Acting on this idea, calomel has been given in doses of two or three grains, in order to carry off the poison and disinfect the intestinal tract, with excellent results.

Albuminuria.—Nephritis must be treated by poultices and dry cupping over the kidneys. Albuminuria due to alteration of the blood requires no special treatment, and it is not a bar to cold bathing, but on the contrary may be among the symptoms calling for it. The disappearance of albumen from the urine, which is sometimes observed when the temperature has been reduced by bathing, seems to show that the blood-change is due to the pyrexia, and not to the poison. If the typhoid-bacilli are found to persist in the urine during convalescence, as occasionally happens, some urinary disinfectant, such as urotropine, should be admin-

istered, and the disinfection of the urine carefully attended to.

Pneumonia.—When pneumonia sets in, stimulants are generally required, and the patient will derive benefit from quinine, which may be given in the form of the ammoniated tincture. Turpentine in small doses, or the stimulant balsams, will often be found useful. Turpentine stupes are generally better than poultices.

Thrombosis.—Elevation of the leg on soft cushions, warmth, and gentle support by means of a flannel bandage, will be the treatment required.

W. H. BROADBENT.

TYPHOID STATE (τύφος, stupor).—SYNON.: Fr. *Etat Typhoïde*; *Etat Adynamique*; Ger. *Typhose Erscheinungen*.

DEFINITION.—A condition which may arise in the course of any febrile disorder, when the approach of death is gradual, and the rise of temperature either excessive or long-continued. The typhoid state was so named because of its similarity to the condition usually seen in severe cases of typhus fever. It is most frequent in enteric fever, yellow fever, and pernicious malarial fevers; and in such cases the tendency is often evident from the very beginning of the attack.

DESCRIPTION.—The symptoms of the typhoid state comprise extreme prostration, low muttering delirium, passing into stupor, with little or no true sleep (*coma-vigil*); the 'chaff-collecting' movements of the hands (*carphology*); derangement of the senses, and hallucinations of sight and hearing. The urine and fæces are passed unconsciously, or there may be retention of urine. The sensibility of the skin is greatly impaired; flies creep unnoticed even over the eyelids. General muscular weakness is shown, the patient lying on his back, sunk down in the bed. The lips and gums are covered with sordes, and the tongue is dry and black. *Subsultus tendinum* is present. The pulse is frequently small, weak, and even running in character, sometimes irregular, and easily affected by slight causes—for instance, failing distinctly during inspiration. The heart's impulse is greatly weakened; the first sound almost or quite lost at the apex. The skin is dry, if, as is often the case, the temperature is high, 104–5° F. or upwards; or it is bathed in clammy sweats, which rarely prevail at one time over the entire surface. There is lividity of, or even ecchymoses on, the under-aspect of the trunk and limbs; and in the exanthematous fevers the eruption becomes petechial. The breathing is hurried and shallow, and towards the close frequently assumes the type known as Cheyne-Stokes respiration.

PATHOLOGY.—It is unwise to attempt to attribute all these different symptoms to one single cause. The disorder of the nervous centres which are principally affected is probably due to the presence of toxins in the blood, and to the enfeebled circulation.

PROGNOSIS.—The prognosis of the typhoid state is always grave, but in fevers which run a definite course recovery may take place, even when the condition is fully developed, if it be so only at or towards the end of the febrile period.

TREATMENT.—The treatment consists in the free use of stimulants. Of these brandy is the best, and may be given in doses of half an ounce every half-hour or hour; but the quantity must be determined not by measure, but by the effects upon the patient.

If no improvement follows its exhibition by the mouth, frequently repeated rectal injections of brandy with egg or strong beef-tea ought to be tried. The subcutaneous injection of five to fifteen minims of ether is often so successful in increasing the vigour of the heart's action, that it well deserves a trial here, too, if other means prove ineffectual. If the temperature is excessive, the cold or tepid bath, or the cold pack, should be used; but immediately before or during the application of external cold, half an ounce of brandy at least should always be given. The salutary effects of cold are maintained and increased by full doses of quinine, such as five or ten grains of the sulphate every hour, until twenty or thirty grains have been taken. Hypodermic injections of strychnine (gr. $\frac{30}{100}$) and the inhalation of oxygen are occasionally of service. External stimulating applications are not without value, for example, vesication by blistering fluid, or by strong solution of ammonia to the shaven scalp, when nervous symptoms predominate, or a flying blister to the præcordial region when the heart's action is seriously impaired. Throughout the typhoid state the patient's strength must be husbanded as carefully as possible; and the great object of the nursing should be to save him from the necessity of any mental or muscular effort whatever.

JAMES ANDREW.

H. MONTAGUE MURRAY.

TYPHUS FEVER (τῦφος, stupor).—SYNON. : Malignant Fever; Petechial Fever; Gaol-Fever; *Typhus Exanthematicus*; Fr. *Typhus*; Ger. *Flecktyphus*; *Kriegspest*; *Lazarethfieber*.

DEFINITION.—Perhaps no better definition has been given in modern times than that which Hilderbrand formerly assigned to typhus: 'Morbus acutus, febrilis, contagiosus, exanthemate proprio ac eminenti systematis nervosi passione stipatus.'

Within the past decade or two typhus has remarkably decreased in prevalence, and there is good reason to suppose that the time is not far distant when it will have entirely disappeared, its poison being readily neutralised by fresh air and possibly by exposure to sunlight.

ÆTIOLOGY.—All experience goes to prove that food, bad in quality and deficient in quantity, is the most potent predisposing cause of typhus. All the great epidemics of fever in Ireland and elsewhere have followed times of famine, owing to bad seasons and failure of the crops. It has been assumed on high authority that typhus is a hunger-fever, but against this view a protest must be entered. That want and misery, and consequent mental depression, overcrowding, and dirt, render the person susceptible and a ready recipient of the poison must be admitted. But that there is a something in the air—'a contagium vivum,' a typhus-miasma, which, taken into the system, produces fever—must also be admitted. Up to the present, the most ardent and able investigators have failed to discover the presence of a fever-producing parasite in typhus, such as has been found in some infective diseases.

BACTERIOLOGY.—Hlava (1891), Lewaschew (1892), Calmette (1893), Dubief and Bruhl (1893), are among the most recent writers on the bacteriology of typhus. Nothing definite, however, has as yet resulted from their researches or those of other investigators. In 1898 E. J. McWeeney, of Dublin, made an unsuccessful attempt to isolate micro-organisms from the blood of two typical

cases of spotted typhus. In both cases the blood proved to be absolutely sterile, tubes of glycerine-agar and faintly alkaline peptone-broth failing to show any signs of growth when put away at 37° C. to incubate. In both cases also the blood-serum, tested by the Gruber-Widal method, gave a negative reaction, so excluding a diagnosis of enteric fever. McWeeney, as a result of his experiments, concludes that our present methods of investigation are not capable of solving the question as to the nature of the *materies morbi* in typhus.

The poison, whatever it may be, is present in certain localities, causing endemic disease, and under favourable conditions assuming the epidemic form.

ORIGIN OF TYPHUS.—The doctrine of the *de novo* or spontaneous generation of typhus is opposed to all analogy, so far as the specific fevers are concerned. Indeed it is beset with greater difficulties than those which it is designed to explain. The theory would become untenable, were the microbic origin of the disease once proved. It is unnecessary, if we assume that in cases of obscure origin we have to deal with a sudden increase of virulence in the infecting agent on the one hand and an acutely developed susceptibility by the patient on the other hand. It must, however, be admitted that now and again a case of typhus occurs in which it is impossible to trace the infection. There is no disease, with the exception of smallpox and scarlatina, which is admittedly more infectious than typhus fever.

In Ireland the question of infectiousness was many years since set at rest by Marsh. Cheyne's reports of the Hardwicke Hospital are also replete with facts pointing to the same conclusions. The greater number are not conscious when they are poisoned by the typhus miasm, but the exceptions are not rare. Marsh perceived, on suddenly turning down the clothes of a patient, a highly disagreeable odour; he was oppressed, and at once overwhelmed *uno ictu*, being conscious at the time of having absorbed the poison.

PRODROMAL OR LATENT PERIOD: PERIOD OF INCUBATION.—The time intervening between exposure to infection and the advent of the series of phenomena constituting fever is generally from three to seven days; but it may be shorter or more protracted. There may be no incubation—*typhus siderans*; the overt symptoms appearing simultaneously with the reception of the poison. The incubation-period is not generally a time of health: sleep is disturbed, there is often a malaise, an undefined sense of illness, a depression, and often a fear of some impending calamity. The patient may struggle against it, and follow his ordinary occupations, until it becomes impossible to further resist.

SYMPTOMS.—The symptom which ushers in the disease is generally a rigor, a sensation as if cold water were trickling down the back. There may, however, be no distinct rigor, merely a *frisson*, a chilliness; and even this may not be noticed. There may be one distinct severe rigor, or a number of slight shiverings. After the rigor a rapid rise of temperature takes place; and now, if not before, the patient is in most cases compelled to take to bed. The symptoms then are headache, giddiness, heaviness, languor, dulness, pain in back and limbs—especially the thighs—sleeplessness or disturbed sleep, thirst, anorexia; perhaps nausea or even

vomiting; the tongue may be loaded, white or yellowish.

The early symptoms are at times so slight that the affected person may be able to walk about; and in three, four, or seven days defervescence takes place, and restoration to health follows. This is termed 'febricula'—*Febris ambulatoria* of the Germans. In by far the majority, however, the symptoms become aggravated day by day; sometimes catarrhal symptoms supervene—often they are the earliest manifestations. Headache is complained of, with intense flashes of light before the eyes, or intolerance of light; the skin, at first red, assumes a dusky hue; the tongue becomes brownish and dry. On the fourth or fifth day of the fever the rash (the exanthem to which the disease *typhus exanthematicus* owes its name) appears. This rash is generally first observed on the trunk, then spreads to the extremities; it rarely appears first on the backs of the hands and face. The spots vary in size from a slight speck to three or four lines; sparse at first, they soon extend; isolated or grouped together, they have been compared to pieces of marquetry; they may be of an intense red, but generally they are pale; little papules are seen in some few cases. The typhus-exanthem bears a close resemblance at its commencement to the rash of measles; hence it is often termed 'measly eruption.' The spots disappear on pressure. In the space of two or three days the eruption is complete; the colour gradually assumes a more or less dark hue, livid or mulberry, no longer disappearing on pressure. The usual duration of the rash is from a few days to fourteen or fifteen days. Its colour is often deeper on the back than elsewhere; and when a doubt exists as to its having appeared, it should be looked for there.

The rash does not seem to have any influence on the fever, and it may continue after the defervescence; but the more copious it is, and the deeper its colour, the more severe is the disease. This is the opinion which is held by most observers. The change of the eruption from a measly rash to true petechiæ or cutaneous hæmorrhages is the rule. The skin in the course of the disease may be the seat of various epiphenomena—erysipelas, furuncles, sudamina, œdema, abscesses or gangrene of the parts most exposed to pressure; and sometimes even the nose and the genitals are affected. In some epidemics icterus occurs, and its appearance has in some cases been remarkably sudden. The febrile phenomena are not mitigated with the development of the rash.

It sometimes happens that towards the end of the first week there is an amelioration of the symptoms; but this is but a lull in the storm in the greater number of cases. The temperature, which had fallen, rises again, and often exceeds the former record. The pulse attains a frequency of 100 in the morning, and 112 to 120 in the evening, and in the severest cases far exceeds this. In the early stages it is fairly full, but it may become weak, compressible, and fluttering; or, which is perhaps a more evil omen than great frequency, slowness is observed, owing to the diminished power of the ventricle to make every contraction felt at the wrist. The patient lies on his back in a state of great prostration; there is a tendency to slip downwards in the bed; the headache of the early days has been succeeded by delirium, the character of which varies much. At first, chiefly at night or on awaking from

disturbed sleep, he is confused and unable to answer questions correctly; at the end of the first or beginning of the second week the delirium becomes more pronounced—noisy, restless, or apathetic—and often the strangest phantasies arise. A fixed idea may take possession of the mind; there may be muttering delirium, coma vigil, sopor, sudden furious mania alternating with sopor; often there is difficulty of restraining the patient in bed; hence the necessity of closest watching, especially at night. The tongue is dry, contracted, and black, covered with fuliginous deposit—'parrot tongue'—as are also the gums. Retention occurs from paralysis of the detrusor vesicæ, or involuntary discharge of urine; and the bladder should be examined frequently, as the discharge is sometimes merely an overflow; for days it may be necessary to employ the catheter. The urine is high-coloured, scanty, and often albuminous. Such are the symptoms of typhus in its severe form.

Towards the end of the second week, in, fortunately, the great majority of cases, a favourable change takes place. There is no disease in which the change may be so sudden from a state of extreme danger. The patient, who had been violently delirious, or lying in a torpid state of low muttering delirium with subsultus tendinum, falls into a tranquil and profound sleep which may last for many hours—so long that it is necessary to awaken him for nourishment; and on awakening the temperature may be normal or below it, the tongue moist, the pulse—which had been rapid, thready, and tremulous—firm and moderate in frequency, and intelligence to a great extent restored, with returning desire for food and drink. This change—crisis by sleep—is the most favourable event. The skin, which was burning hot—*calor mordax*—may become moist, bedewed with a gentle sweat; and this seems in some instances critical, altogether different from the copious sweat which, occurring at an earlier stage, is almost always indicative of danger. A change, too, occurs in the urine which has been considered critical: it has been high-coloured and albuminous, or even bloody, and now the secretion is abundant, of normal colour, or even paler than natural, with a copious deposit of the lithates and phosphates. To this change in the urine the term 'crisis by urine' has been given.

COMPLICATIONS.—Many are the dangers and difficulties from complications which threaten the patient in the course of typhus, and prolong the disease; one of the most common is implication of the respiratory organs. In some epidemics so frequent is bronchitis, that the disease has received the name of 'broncho-typhus.' From the onset there may be a bronchial affection unmarked by the ordinary symptoms, even in advanced stages—no expectoration, perhaps not much acceleration of breathing; it may involve the whole extent of the bronchi (bronchiolitis), even producing collapse; its presence being discoverable only by auscultation. It is noteworthy that secondary affections in typhus are more or less latent. Broncho-pneumonia, hypostatic congestion, diffuse pneumonia, are among the most formidable complications in fever, and often indicate a lethal termination. Multiple abscesses, or gangrene in various situations, not confined to the parts exposed to pressure, may occur. Convulsions may usher in a fatal termination. Sudden death has occurred when all seemed to progress favourably.

DIAGNOSIS.—There are cases of typhus the course of which is normal, and which present no difficulties to the experienced physician in arriving at the diagnosis, and hence it is said the disease is easy of diagnosis; but he will meet with others, though few in number, in which it is occasionally impossible.

The fevers with which it is most likely to be confounded are enteric and relapsing fever, both of which are found to exist in company with typhus in many epidemics of the latter. Cases of enteric fever may be wanting in the characteristic changes in the intestinal canal and the glands; and, again, typhus may exhibit changes resembling those of enteric fever, and so the two may, as it were, shade into each other. The knowledge of the exposure to either disease will assist very much, for typhus never gives rise to enteric, nor enteric to typhus. Important aid also in the diagnosis may be obtained by observation of the temperature: marked elevation of temperature is noted in typhus from the onset, and the rapidity of the rise and the shortness of its duration exclude the idea of enteric. The course of typhus resembles that of relapsing fever in the first days. In both of these diseases the temperature is high; but it is higher in relapsing fever, and on the fifth or seventh day defervescence takes place, and the patient is for the time fever-free, to be attacked later by a new access of fever. Other febrile diseases seldom maintain so high a temperature as typhus for so long a period. The return of the temperature to the normal from fever-height in typhus is often very rapid, the defervescence, as before mentioned, taking place in one night.

Comparing typhus with enteric fever, the defervescence is extremely rare at so early a period in the latter, or at so late a date in an acute exanthem or in relapsing fever. The rapid fall of the temperature in typhus, as compared with the gradually decreasing fever in enteric, is also a distinguishing feature. The profuse petechial rash contrasts strongly with the sparse rosy spots of enteric fever, which usually come out in successive crops, and are slightly elevated. The absence of epistaxis and intestinal lesion, also of hæmorrhage and of marked splenic enlargement; as a rule, absence of the diarrhoea characteristic of enteric fever, and of ileo-cæcal tenderness and gurgling; and the comparative rarity of meteorism,—all are to be taken into consideration. In both, the respiratory and circulatory organs are liable to be implicated, and so are the kidneys.

It has been affirmed that no single symptom proclaims the presence of typhus; but the order in which the symptoms are presented, the grouping of symptoms—what German writers call ‘symptom-complex’—and the disturbance of the nervous system, powerfully aid in arriving at the diagnosis.

PROGNOSIS.—In forming a prognosis of typhus fever, the character of the epidemic, and the rate of mortality, may first be taken into account. Some epidemics are peculiarly mild and benign, others are malignant in the extreme. The period of the epidemic is also important, as the earliest cases are generally most fatal. Then the condition of the patient, if previously in broken-down health, ill fed, ill clothed, ill housed—such fall ready victims to the pestilence; perhaps nothing renders the prognosis more unfavourable than habits of intemperance. In the upper ranks of life, occupations

involving anxiety and mental stress predispose to bad attacks, in which the nervous symptoms are prominent at an early stage; such cases have received the popular name of ‘brain-fever.’ Sex-mortality is also to be noted, being greater in adult men than in women. The symptoms indicative of danger are the early existence of delirium alternating with stupor, the peculiar affection denominated ‘coma vigil,’ subultus tendinum, and other low nervous phenomena. See COMA VIGIL.

Very high or very low temperatures, and extreme rapidity of pulse, always proclaim the severity of the disease and consequent danger. It has been observed, however, that in no disease is high temperature so well borne as in typhus. The character of the pulse is one of the most reliable indications of danger. To rapidity may be added irregularity, feebleness, compressibility. The opposite state however (slowness) is also of evil augury; for the first sound of the heart may be absent, and the impulse diminished in force or absent—conditions of the heart indicating the need of wine and other stimulants, as first pointed out by Stokes. Great tympanic distension is another indication of danger; and the presence of rash of the deepest hue.

Other conditions which add to the danger in typhus fever are weakness or paralysis of various parts; hypostatic congestion, which may end in gangrene of the lung; strabismus; and contraction of the pupil; spasm, convulsions, stiffness of neck, utter unconsciousness, automatic movements; and, above all, advanced age.

The prognosis in uncomplicated typhus is favourable; in early life the mortality is moderate. It is not so great in women, except when they are pregnant. Danger increases after passing the meridian of life; a quarter and even half of the cases between fifty and sixty years of age succumb. In some epidemics the enormous mortality of 40 or 50 per cent. has been reached, contrasting remarkably with the low rate of others—only 5, 6, or 7 per cent.

ANATOMICAL CHARACTERS.—It is admitted by all who have had the most extensive opportunities of investigating the disease, that there are no anatomical changes which can be considered pathognomonic of typhus.

If death takes place early the body is little emaciated; the petechiæ which are caused by blood-extravasations are visible, and, as in life, most marked on the back. Almost all the organs of the body are the seat of change in structure, congestions and hyaline, granular, or fatty degeneration. The mucous membrane of the intestinal tract is infiltrated, and shows traces of catarrh, and in exceptional cases even superficial ulceration, but there is nothing constant. The brain and membranes in the early stages are hyperæmic; if death occurs late in the disease they may be anæmic, with serous effusions. The heart-walls are softened, and the seat of granular and fatty change; there is fluid blood in the ventricles. The lungs are the seat of hypostatic congestion, the blood being almost black. The veins and arteries have been found to be the seats of thromboses and emboli.

TREATMENT.—Impressed with the communicability of typhus, it is the duty of the physician to insist on the isolation of the sick from the healthy. On the first access of fever the patient should be ordered to bed. Every exertion, mental or bodily,

should be forbidden. The transport of those sick of typhus in war has been known to intensify the symptoms; so it is, too, in civil life. Therefore removal from the locality where first attacked should be avoided, except under the stress of absolute necessity.

In the management of typhus-cases, an abundant supply of air should be afforded. From the chamber all unnecessary furniture should be removed; the covering of the bed should be light. The temperature of the apartment should not be under 59° F. or over 65° F. During the height of the fever, when the temperature is high, exposure to cold does not act injuriously; but it is harmful when once defervescence has taken place. The patient should not be permitted to change from the prone position or any purpose—an order often difficult to enforce during the early period of the illness. The strictest injunction should be issued against noise, or even whispering, in the room. Light should be modified, but not excluded. The patient will sleep better if the room is not darkened during the day. No one should be admitted to the room except the necessary attendants.

The disease in uncomplicated cases, when of moderate severity, runs a certain course, tending towards a favourable termination, and then the treatment is absolutely expectant. The idea of arresting typhus fever is now altogether abandoned. The regulation of the diet is of paramount importance. Thirst is constantly felt, and the craving should be satisfied, and the fancies of the sick, when possible, be indulged. Cold water or some mineral water will generally be found to be most acceptable; when the tongue is dry, iced water or ice may be permitted. If the patient's sensibilities are so blunted that he cannot give expression to his wants and wishes, care must be taken that at regular intervals drink and nourishment be given; no more suitable drink than milk and water or soda-water can be given, thus supplying nourishment at the same time that the thirst is assuaged. Broths of different kinds are freely allowed, and, when there is great debility and manifest failure of strength, more concentrated nourishment, as essence of meat, or eggs. The administration of wine and stronger alcohols is one of the points in the treatment of fever demanding the most careful consideration on the part of the physician. In most cases, except in those stamped by malignancy from the commencement, it may be postponed to the end of the first or beginning or even the middle of the second week, and alcohol may then be cautiously, tentatively given, marking its effects. The selection of the stimulant should depend on the age and previous habits of the patient: the young or previously temperate require wine in only small quantities, and diluted; the aged or intemperate a liberal allowance of strong wine or even brandy. Without reference to the day of the fever, if there be prostration, a tendency to sinking of the vital powers, stimulants must be freely given; and thus the threatened paralysis of the heart may be ward off. Medicinal agents, such as ammonia and ether, are often advisable. See STIMULANTS.

The cerebral symptoms—headache and delirium—are frequently relieved by the application of ice or, what is sometimes more effectual, cold or tepid affusion. Care should be taken, when the ice-bladder is employed, that it should not remain too long on the head, as it seems to have a depressing

effect. When insomnia is present, and the temperature is high, tepid affusion often has the effect of inducing sleep; so has the abstraction of a small quantity of blood from the temples (two or three leeches on each temple), sleep often coming on while the blood is flowing. In conditions of a comatose nature blisters should be employed—strips of blisters on the vertex, and not to the nucha, the part usually selected, but which should be avoided. If insomnia continues, the use of the bromides may be resorted to, and often with advantage; but when they fail, as they may, then opium or morphine must be administered. Hall has found sulphonal a remedy of much value, but it often has a depressing effect.

The application of water to the head has been spoken of, but in all cases it is also essential for the whole body. The most scrupulous cleanliness is to be observed, and sponging with cool or tepid water may be used when the antipyretic treatment by cold baths has not been fully carried out. This treatment is now being adopted in typhus also, in which, as in other cognate diseases, the danger of high temperature is universally admitted. At the same time our object must be to control temperature, not recklessly to reduce it too much or too fast. The nature of the fever-process is now far better understood than it was even a few years ago, and we have learned that 'fever,' or elevation of bodily temperature above the standard of health, or 'normal,' serves a useful purpose, provided that it is properly controlled.

Quinine has been looked on by some as a specific. Liebermeister recommended it in colossal doses, and said he treated no serious case of typhus without it. It is used as an adjunct to the cold bath in the antipyretic treatment of fever; it may be the sole antipyretic when the cold bath is contraindicated. Mosler, who had the experience of two epidemics of typhus, attributes the low mortality of the second to the fact of the antipyretic treatment having been by him more energetically carried out in that epidemic than in the first.

The best means of removing constipation is by warm- or cold-water enemata, which never cause over-action, which even mild aperients by the mouth often do. One of the most dangerous complications in fever is meteorism, and the best means of combating it is by the internal and external use of turpentine. Another is bed-sore, which in low forms of fever, despite all efforts to guard against it, will sometimes appear. The back should be examined daily, and the surface, which has become red, should be bathed with brandy or camphorated spirit; the position of the patient should be changed, and air- or water-cushions placed under the back, or a water-bed employed. In all cases it is most desirable to have two beds; a change can be effected without difficulty or danger, and the comfort and advantage of separate day- and night-beds are incalculable. It is most difficult at times to limit the ravages of bed-sores; the nates have in some instances literally sloughed away, but even in such a case recovery has taken place. For the treatment of GANGRENE, see p. 571.

The danger of hypostasis has been alluded to, and this may be averted by altering the position of the patient, and also by the free administration of wine or brandy to stimulate the heart's action.

In pneumonia occurring as a complication,

quinine is a most useful remedy; and in cardiac failure it seems to have almost a specific influence on the muscular tissue of the heart, for it has been often found that under its influence the weak, irregular, and fluttering heart has become regular.

The physician must be always on the watch for complications, for their approach is generally insidious, and wanting in the symptoms which in ordinary cases appertain to them; they may be truly termed latent. The restoration to health after ordinary typhus is rapid; but typhus protracted beyond its ordinary duration leaves the patient's bodily strength at a very low ebb, and sometimes the brain does not speedily recover from the shock it has received. Mania may be a sequel, but the form of cerebral disturbance is more frequently characterised by mental weakness, with inability to fix the attention and follow any train of thought. The prognosis is generally favourable, for usually in time the mind recovers its wonted vigour.

JOHN BANKS.
J. W. MOORE.

TYROSINE (τυρός, cheese).—SYNON.: Fr.

Tyrosine.—Tyrosine, or para-oxyphenyl- α -amido-propionic acid, is, like leucine, with which it is generally associated, a decomposition-product resulting from the prolonged action of the pancreatic secretion on the complex proteid molecules. Its chemical composition is represented by the formula $(C_6H_4(OH)CH_2CH(NH_2)COOH)$. Tyrosine is never a constituent of *fresh* tissues. It is normally a constituent of the intestinal contents, being one of the products of the action of trypsin on the hemipeptone resulting from proteolysis by the gastric and pancreatic ferments. See ALKAPTONURIA.

CHARACTERS.—Tyrosine-crystals are soluble in acids and alkalis, but very sparingly soluble in cold water, much more soluble in hot water, and practically may be considered insoluble in ether and strong alcohol. It cannot be sublimed without decomposition.

Microscopically, tyrosine-crystals are usually seen in the form of very fine needles, either

separate, or occurring in feathery or 'wheatsheaf' bundles or rosettes (*see* p. 1798).

Chemically, tyrosine may be separated from the urine, by the method described under LEUCINE, but the residue obtained, insoluble in boiling alcohol, is treated with boiling water, and allowed to crystallise. The most reliable and delicate reactions for tyrosine are the following:—

Piria's Reaction.—Warm on a water-bath for about five minutes a watch-glass containing tyrosine moistened with concentrated sulphuric acid; a pink colour is produced. Dilute with water, warm, neutralise with calcium carbonate; filter while hot. Add to the filtrate a few drops, carefully avoiding an excess, of a weak solution of the perchloride of iron. A violet colouration is indicative of the presence of tyrosine. Leucine interferes with Piria's reaction.

Hoffmann's Reaction.—If to a heated solution of tyrosine a little Millon's reagent be added, the fluid assumes a rose-red or crimson colour; and if tyrosine be present in quantity, a precipitate of a similar colour will be thrown down.

Scherer's Reaction.—Apply a mixture of strong nitric acid and water to a solution of tyrosine, evaporate to dryness, a yellow residue is obtained; moisten and warm with liquor sodæ—a yellow turning to a deep reddish-yellow colour is produced.

PATHOLOGICAL SIGNIFICANCE.—Tyrosine is never present in healthy livers; it, however, is probably normally present in very small amount in the pancreas and the secretion of this gland, and in the spleen. It is not considered a normal constituent of the urine (Ulrich regards leucine and tyrosine as normally present in minute quantity), but its presence in this excretion along with leucine is of value diagnostically in certain morbid states of the liver, especially acute yellow atrophy, in which the secreting cells are quickly disintegrated. It has also been detected in the urine in cases of acute poisoning by phosphorus. See LEUCINE; LIVER, Atrophy of, Acute Yellow; XANTHOMA; and URINARY DEPOSITS, Plate XX. JOHN HAROLD.

U

ULCER AND ULCERATION (*ulcus*, a sore).

SYNON.: Fr. *Ulçère*; Ger. *Geschwür*.

DEFINITION.—An ulcer is a solution of continuity of an epithelial or endothelial surface, resulting from the destructive changes associated with inflammation. *Surgically* the term 'ulcer' signifies an open sore.

ÆTIOLOGY.—The inflammatory process to which ulceration is due results in almost all instances from the action of micro-organisms. In a few cases continued mechanical irritation may cause an ulcer, but even in such instances extension of the process is due to the invasion of the wound by bacteria. The poisons formed in the growth of these cause the phenomena of inflammation, with death and disintegration of the tissues. In the majority of ulcers the organisms at work are the common pyogenic cocci (*see* PYOGENIC BACTERIA). Ulcers may also be due to the tubercle-bacillus, the

B. typhosus, the organism of dysentery, and others. In syphilitic (tertiary) ulceration, a breach of surface results from the breaking down of a gummatous deposit, and secondary invasion by pyogenic organisms takes place: this secondary invasion may occur in other 'specific' ulcers, such as the tubercular, or in those arising from necrosis of new-growths.

'Predisposing' causes of ulceration are seen in any conditions which diminish the resistance of the tissues to bacterial attack. Thus the defective nutrition of the skin due to varicose veins causes slight injury (scratching, blows) to result in the formation of an ulcer. In diabetes mellitus small abrasions or wounds may give rise to ulcers, which show little tendency to heal, the factors at work in this disease being not only the presence of sugar in the tissues, but also special changes in the vessels and nerves. Owing to their poor vitality the cells

of tumours rapidly undergo fatty degeneration and necrosis, which result in the early appearance of ulceration in superficial malignant growths. Ulcerative endocarditis is generally secondary to old rheumatic lesions, which become the seat of fresh bacterial invasion.

It is not yet certain whether the nervous system exerts a direct trophic influence upon the tissues, but the evidence appears to favour this view. Perforating ulcer of the foot is met with in cases of tabes dorsalis, and less commonly along with other nervous lesions. If it be not due to direct nervous action it must be supposed that the resistance of the tissues is weakened by disturbance of their vascular supply, and that organisms are thus enabled to gain a footing as a result of insignificant injuries. The same alternative explanations may be offered in the case of bed-sores; or the combined causes may be at work in both instances.

A breach of surface by which bacteria may enter and set up the process of ulceration may arise from external causes, such as mechanical violence, chemical irritation, heat and cold, continued pressure, &c. So-called constitutional causes are in reality only 'predisposing' conditions, in the sense given above.

PATHOLOGY AND MORBID ANATOMY.—The process of ulceration consists in a gradually extending molecular death of inflamed tissues, and is exactly the same as that by which an abscess-cavity is formed and extends (*see* ABSCESS). The wall of an ulcer is in structure the same as that of an abscess, and varies according as the destructive process is still spreading or has been arrested. If the ulcer is spreading the surface will be formed of a layer of leucocytes, and these cells also infiltrate the underlying tissues; the elements of the latter are granular, and in process of destruction, and the blood-vessels dilated for some distance around the lesion. Sloughs of tissue killed *en masse* may here and there remain adherent to the surface of the ulcer. If arrest of the ulceration have taken place, the ulcer is practically a healing wound, and its floor is covered with 'granulations,' the deeper layers showing all stages in the gradual conversion of granulation-tissue into fibrous cicatrix (*see* WOUNDS, Healing of). The naked-eye appearances of the different varieties of ulcers are given below, the essential parts for study in any ulcer being—the surface or floor; the edges and surrounding parts; the base or underlying tissues; and the discharge secreted.

The discharge from an ulcer consists of serous exudation from the vessels, with a varying amount of leucocytes, which die and constitute pus. Occasionally blood is present either from intensity of inflammation or from rupture of capillary loops in the granulations. If the discharge is retained beneath a crust or dressing, it may accumulate and putrefy.

CLASSIFICATION AND VARIETIES.—Ulcers may be classified according to their most obvious causes, predisposing and exciting; thus there are tubercular, typhoid, syphilitic, paralytic or perforating, varicose, malignant and other kinds. For clinical purposes the following varieties may be distinguished:

1. *Healing or healthy ulcer.*—This is practically a granulating wound. The surface is covered by granulations, which are small and bleed when smartly touched. There is a thin, blue epidermic pellicle, consisting of the epidermis advancing from

the edges over the surface. The edges are on a level with the surface, of a pink tint, and free from induration. The surroundings are free from induration, and normal in appearance. The discharge is small or moderate in quantity, and, unless free from pyogenic bacteria, consists of so-called 'laudable pus.' The pain is inconsiderable.

2. *Weak ulcer.*—The cause of one form of weak ulcer is the prolonged use of emollient applications. The characters are: flabby, watery, gelatinous granulations, rising above the level of the surroundings as so-called 'proud flesh.' The edges are overlapped by the granulations. The surroundings may be fairly normal. The discharge is thin and watery. There is little or no pain.

Another cause of 'weak' ulcers is where, owing to some local or constitutional cause affecting the circulation, there is a defective blood-supply to the ulcerating surface. The granulations under these circumstances become smooth and yellowish, the edges look pale and flat, the secretion is thin and small in quantity, and a dry crust or scab is apt to form. In yet a third variety the ulcer becomes oedematous, a condition frequently associated with Bright's disease, and any local or constitutional trouble in which the circulation in the larger vessels is defective.

3. *Indolent, callous, or 'chronic' ulcer.*—This sub-variety occurs when, from chronic irritation in a part devoid of sufficient blood-supply, the fibrous induration is excessive, and the blood-vessels reach the surface of the ulcer in too minute quantities to build up healthy granulations.

The surface is sunk below the level of its surroundings, is destitute of granulations, and looks glazed. The edges are raised, hard, and irregular. The surroundings are indurated and raised, and the veins in them frequently become varicose. The discharge is thin, serous, and small in quantity. The pain at times is very great, especially at night.

4. *Irritable ulcers.*—These are met with on the legs of neurotic and anæmic women, and in women suffering from menstrual disturbances. The surface is uneven, often covered partially with a grey slough. The edges are thin and irregular. The surroundings are red and glazed, but not raised or thickened. The discharge is a thin sanious pus. The pain is excessive, of an aching or neuralgic kind.

5. *Inflamed ulcers.*—Any form of ulcer may become inflamed, owing to fresh bacterial invasion. This sub-variety may arise during the course of an indolent ulcer, from derangement of the patient's health or local irritation.

The surface is covered with a greenish-grey exudation. The edges are swollen, everted, red, and angry. The surroundings are red, swollen, and hot. The discharge is ichorous, offensive, and often bloody, causing irritation wherever it touches. The pain is of a throbbing kind.

6. *Phagedænic and sloughing ulcers.*—When an inflamed ulcer commences to spread, the edges liquefy and rapidly break down. The process may take place with extraordinary rapidity, as in chancre, when it is called phagedæna; or as in hospital gangrene, when it spreads chiefly by sloughing. The patient in either case is usually in a cachectic state; the ulcerated part is dusky red, angry-looking, hot, and painful. The surface is covered by a grey or black slough, and the edges are sharply cut and undermined. *See* BUBO; CANCRUM ORIS; GANGRENE; and VENEREAL SORE.

7. *Varicose ulcers*.—These are small at first, and gradually enlarge with œdema of the edges, soft prominent granulations, and eczematous surroundings. They seldom bleed except at the moment of the bursting of a periphlebitic ulcer, from which they sometimes originate. The chronic ulcer, induced by varicose veins, develops a widespread induration, which, by interfering with the venous circulation, tends to increase the tendency to varicosity.

8. *Hæmorrhagic ulcers*.—Hæmorrhagic ulcers occur in persons suffering from amenorrhœa, scurvy, chronic jaundice, or hæmophilia. They possess many of the characters of irritable ulcers, but in addition have a special tendency to bleed. The blood is of a capillary-venous character, and flows fairly freely at times.

9. *Atonic (paralytic) ulcers*.—In the feet of paralytic patients ulcers may develop with extraordinary rapidity. They are painless, frequently multiple, and present uneven, often scooped-out, surfaces covered by granulations of irregular size. *Perforating ulcer of the foot* is an affection of this nature. The ball of the great toe or the under surface of any of the metatarsal bones may become the seat of this ailment. There is first seen a callosity on the sole of the foot; pus develops beneath this, and when the abscess bursts a long sinus will be found leading down to bone. The bone may in turn become involved and undergo absorption. The peculiarity of this form of ulcer is that it is funnel-shaped, the granulations are protuberant, the discharge profuse and foul, and the surfaces of the sinus become lined by an epithelial coating. The ulcer may heal under rest and antiseptic treatment, but generally reappears when the patient once more walks about.

Syphilitic, lupoid, rodent, scorbutic, endothelial, mucous, tubercular, and cancerous ulcerations, as well as those occurring in *mycetoma, leprosy, cancerum oris*, &c., are described under the diseases of which they form part.

TREATMENT.—To treat an ulcer on a rational basis it is necessary to ascertain as exactly as possible the cause of the ailment, both as regards its genesis and its continuance. When a constitutional factor plays a part in the ætiology, suitable treatment for the condition should be adopted. When, on the other hand, the cause is of local origin merely, attention must be devoted to allaying or removing the source of trouble. Some ulcers from their very nature may be incurable, such as those of a malignant or leprosy nature; others, such as diabetic, syphilitic, or scorbutic ulcers, heal only when suitable drugs or diet are exhibited; while a third group, including all those ulcers caused more essentially by local disturbances, such as varicose ulcers, pressure-ulcers (bed-sores), senile ulcers, inflamed, weak, septic, and phagedænic ulcers, require local treatment for the most part.

The removal of the constitutional causes is dealt with under the several diseases with which the ulcer may be associated, so that only the local treatment need be commented upon here.

Rest.—Of all the aids to cure this is perhaps the most important. When the lower extremity is the seat of the trouble a splint or stiffened bandage may be applied at times with advantage; the upper limb may require to be supported by a splint or sling. The joints immediately above and below the section of the limb on which the ulcer exists should be kept at rest.

Position.—Elevation of the limb while the patient is recumbent is a primary consideration, especially in ulcers of the lower extremity.

Massage is an excellent accessory when extensive induration of the tissues around an ulcer is marked.

Removal of sepsis.—To render the parts around a chronic ulcer aseptic proceed as follows: wash the parts around the wound with soap and water, and shave off the hairs if necessary. Dry the part and apply turpentine to get rid of the sebaceous materials in the skin. The skin is again washed with soap and water, using a large nail-brush for the purpose. Carbolic-acid lotion (1–20), corrosive-sublimate solution (1–5000), or other reliable antiseptic lotion is applied to the skin thoroughly by a nail-brush. The process should be repeated several times on successive days, to ensure complete surgical cleanliness.

To render the surface of the ulcer itself aseptic, it is to be converted into a healthy wound at once by scraping it with a sharp spoon while the patient is anæsthetised, and then thoroughly mopping the part over with pure carbolic acid or some other corrosive. The surface of the wound and the surroundings are then enveloped in antiseptic dressings, of which, perhaps, cyanide-gauze dipped in corrosive-sublimate solution (1–2000) is the best.

When, for any reason, this radical method of treatment is inexpedient, the ulcer, after the surroundings have been dealt with as recommended above, may be dressed with lint soaked in carbolic oil (1–5), the lint being firmly pressed into the surface of the sore at all points. When the oil is used, daily dressing will be required, but when the wound has been scraped and covered by gauze, the dressing may be left in place for two, three, or more days.

To promote healing after the ulcer has been rendered aseptic, and when it appears fairly healthy, proceed as follows: Cut a piece of protective oiled silk to the size of the ulcer; wash it clean in carbolic lotion (5 per cent.), and afterwards in a saturated solution of boric acid; apply the oiled silk thus prepared directly upon the wound. Cover the 'protective' by layers of boric-acid lint, wetted in a saturated solution of boric acid and cut somewhat larger than the protective. Maintain the whole in position by a firmly applied bandage, commencing at the foot (or hand) and carried upwards to a considerable distance above the dressing.

In place of the dressing, protective ointments or dusting powders may be employed. An excellent ointment is one consisting of the unguentum acidi borici, B.P., 1 part, and vaseline 3 parts. The ointment is best spread on butter-cloth, not on lint. The part is then covered over with a dry antiseptic lint or gauze, and bandaged. Iodoform, boric acid, or a mixture of the two, are efficient dusting-powders.

When ulcers are *inflamed* it is necessary to allay the pain and inflammation by rest and by boric-acid fomentations. A boric fomentation is made by soaking boric-acid lint in boric-acid lotion or warm (boiled) water, laying it on the ulcer and for some distance beyond, then covering the whole with a piece of mackintosh and bandaging. This dressing is changed two, three, or more times daily until inflammatory signs and symptoms disappear, when the ulcer may be treated in the ordinary way by protective, &c.

Should boric fomentations fail to allay pain it may be necessary to *incise the margins and surface* of the ulcer by a few strokes of a scalpel carried parallel to each other and to the long axis of the sore. After incising, fomentations may be applied until inflammatory signs have gone, when the ulcer to be treated as the granulations indicate.

An *irritable* ulcer may require to have the surface destroyed by blistering with liquor epispasticus, or by rubbing nitrate of silver, nitric acid, or other corrosive, upon it, before a healthy action can be induced. As the irritable ulcer is a frequent concomitant of menstrual abnormality, this condition has to be tended to as an essential element in treatment.

When *weak* or *callous* ulcers become quite *chronic* it is necessary to stimulate activity in their condition by every available means. Rest and elevation of the limb, with attention to the general health, may alone serve to attain an alteration in the nutrition of the part. The ulcerated surface may be stimulated by the application of a fly-blister to the surface, or still better to the thickened parts around the edges of the ulcer.

Pressure, when equably applied, proves at times a reliable method of stimulating a callous ulcer. Pressure by *strapping* is applied as follows : Cut strips of plaster one-third longer than the circumference of the part of the limb to be covered. For the leg the strips should be one inch in breadth. Apply the centre of the strips on the opposite side of the limb, commencing two or three inches below the ulcer. Each succeeding strip should overlap its predecessor by two-thirds of its breadth. The ends are crossed over the ulcerated surface and pulled fairly tight. The strapping is continued for two or three inches above the ulcer, and holes cut in it if the discharge is very free.

Martin's elastic bandage, perforated, with or without a suitable dressing on the ulcer, may be applied to the whole limb, commencing, in the case of the leg, at the foot, and carrying the bandage to just below or above the knee. The bandage ought to be removed at night, cleaned and dried, and applied on the following morning before the patient gets out of bed.

Unna's bandage consists of a double-headed roller of porous material applied to the limb, commencing at the ulcer ; over the bandage a mixture, consisting of gelatine 10 parts, glycerine 40 parts, a little oxide of zinc, and 40 parts water, is melted and rubbed on the bandage ; over the mixture, and before it has set, a second covering bandage is applied. In this way the parts are kept at rest and pressure kept up. To remove the bandage, place the limb in warm water, when the hardened mixture is melted and the bandage can be unwound. The application may be renewed every second or third day, or less frequently, as occasion requires. The ulcers and parts around must be first thoroughly disinfected and then dusted with iodoform before applying the bandage.

Varicose ulcers should be treated by rest, pressure, stimulation, or blistering, before any attempt at removing the varicose veins by operation is made.

Phagedenic ulcers demand the thorough scraping away of the foul surface by a sharp spoon and the chopping over of the exposed tissues with a corrosive such as nitric acid or caustic potash ; or the tissues may be touched lightly by the actual cautery. Carbolic oil (1 to 5) is the best dressing after the ulcer is scraped and cauterised. The ulcers occur-

ring in *paralytic* patients are treated for the most part after the manner of bed-sores.

Perforating ulcer of the foot requires to be completely eradicated ; the fistulous opening, the sinus leading down to the bone, and the carious surface of the bone itself must be cut or scraped away and the cavity packed with gauze.

The treatment of *syphilitic, lupoid, rodent, scorbutic, endothelial, and mucous* ulcerations is described in the articles devoted to these special subjects.

Bed-sore.—SYNON. : Fr. *Décubitus* ; Ger. *Decubitus* ; *Wundliegen*.

DEFINITION.—A form of ulcer caused by continued pressure, consequent on maintenance of the recumbent position.

ETIOLOGY.—Bed-sores are dependent either on a low condition of the nutrition of the tissues of the patient, on bad nursing, or on a combination of the two. In patients suffering from fractured spine, especially if the spinal cord be torn ; in those paralysed from other causes ; in cases of fractures of the lower extremity ; in angular curvature of the spine ; in patients suffering from hip-joint diseases ; in the acute specific fevers ; and in the aged—in fact, in any disease or condition necessitating long confinement to bed and rest in one position—bed-sores may be developed. When, in addition to the illness, the nursing is badly conducted, as shown by urine, pus, blood, or any discharge whatever being allowed to remain on the bedding on which the patient lies, a strong determining cause is set up, which will in all probability originate a bed-sore. Other strongly predisposing causes are the use of a feather-bed, and the presence of a blanket between the mattress and the under-sheet, into which the perspiration from the patient's body soaks, causing the blanket to act like a poultice. Hence it will be seen that paralysis and an enfeebled state of the circulation, combined with pressure and inattention to strict rules of cleanliness, are the main elements in the production of a bed-sore.

The various sites on which bed-sores form are arranged here in the order of frequency with which they are met—the sacrum, the heel, the buttocks, over the trochanters, between the shoulders, on the middle of the back from the shoulders to the sacrum, on the malleoli, on the elbows, and on the calf of the leg. On the heel, the usual cause is the pressure of a splint ; on the elbow, bed-sores frequently supervene in such cases as hip-joint disease, owing to the tendency patients have to support themselves on one or both elbows.

PATHOLOGY.—A bed-sore is only one of the many forms of the evil results of pressure. The passage of blood through a part where pressure is great becomes mechanically difficult. Exudation from the blood-vessels takes place, causing the cuticle to be first raised, and then to peel off ; finally a moist catarrhal surface results. Complete stagnation of the blood in the blood-vessels may occur, and as a consequence part of the skin may become practically dead. In the surrounding parts the blood-vessels become engorged ; and, the presence of the slough acting as a foreign body, the irritation causes inflammation, and an exudation of inflammatory products occurs between the living and dead parts. This takes place all around and beneath the slough, but is first apparent as a furrow in the skin. This furrow gets gradually deeper and deeper, and, the process of separation extending

beneath the slough, it becomes detached and finally thrown off. An ulcerating surface now results, the further history of which will vary with the patient's health, and according as the illness, which renders confinement to bed necessary, is of a curable or an incurable nature.

SYMPTOMS.—The premonitory symptoms of bed-sore may be either subjective or objective. Subjectively the patient complains that the bed feels hard, that there is a crease in the sheet, that there are crumbs of bread or salt in the bed; along with these generally imaginary troubles, a pricking, numbing sensation is felt at the point of pressure; but, on examination of the part complained of, no change may be apparent. Or, again, the complaints may be *nil*, as in the paralysed, and yet the effects of pressure may be far advanced. The objective symptoms may here be the first indications of the effects of pressure. These are—alteration in the colour of the skin, a roughening of the cuticle, and a variable amount of pain on pressure with the finger. Any of these is sufficient indication that preventive measures must be immediately undertaken, otherwise a bed-sore will develop. As the symptoms advance, the discolouration becomes deeper, passing from red to livid red, from purple to black. The pain, except in the paralysed, becomes for a time severe, and then finally disappears, as all nervous connections are severed. The circulation through the part being completely stayed, a dark slough is formed, and a line of demarcation between the living and dead tissue is set up. A foul discharge runs from the part, the tissues around become red and congested, the edges are undermined, and a feeble attempt is made to throw off the central slough. Should the slough be thrown off, the muscles, fasciæ, and even the bone of the damaged part may be exposed. The general symptoms associated with bed-sores are chiefly those of the disease in the course of which this complication has supervened. In some instances a form of pyrexia may, however, be induced by the discharging ulcer itself—a condition which constitutes one form of 'bed-fever.'

PROGRESS AND PROGNOSIS.—Should the patient recover from the illness for which confinement to bed had become necessary, the bed-sore will in all probability heal. Bed-sores, however, can scarcely be healed in those patients whose maladies do not subside, or in whom recovery does not take place. At times pyæmia supervenes; or the exhaustion, consequent on a long-continued and profuse discharge from the sore, proves too great a drain on the patient; or ulceration into the spinal canal may rarely occur and set up meningitis. The prognosis in regard to bed-sores developing in any individual case will depend on the disease, the age of the patient, and the care taken in nursing. The patients in whom a bad prognosis might be given, in regard to the appearance of a bed-sore, would be the old and the partially paralysed, especially when bad nursing is superadded.

TREATMENT.—It is necessary to consider this subject under the heads of (1) the *preventive measures*; (2) the treatment *when abrasions have taken place*; and (3) the treatment or cure of the *sore when formed*.

1. The *preventive measures* have, in paralysed patients and in patients suffering from chronic illness or incontinence of urine, to be commenced at the beginning of the illness, and signs of changes in

the skin are not to be waited for. The part must be thoroughly cleaned, the circulation stimulated, the skin hardened, and pressure removed from the parts where it is greatest. The best means by which to gain these ends are as follows: First, the part is to be washed with soap and water, and then thoroughly dried. Secondly, a piece of cotton wool dipped in spirits of wine, eau-de-Cologne, or brandy, must be applied to the part with gentle rubbing, until it has become thoroughly dry; and this process must be repeated three or four times at each dressing. Thirdly, to keep the skin supple, a very small quantity of oxide-of-zinc ointment or simple ointment must be rubbed into the part, until all greasy feel has disappeared. Fourthly, a draw-sheet must be placed beneath the patient, and boric-acid powder sprinkled on the part on which the patient is to lie; the sheet must be changed the moment it becomes wet, whether from urine, blood, pus, or sweat. Pressure must be removed by frequent changes of position, and by suitable pads, air-cushions, water-pillows, or water-beds. The part where the edges of the cushion press is to be treated by the same preventive measures, and to be dressed two or three times a day. Lastly, it is necessary to avoid a feather-bed, and a blanket beneath the under-sheet. In many cases bed-sores are caused by careless nursing, but in others the best nursing possible cannot prevent badly nourished tissues from falling into decay.

2. When *abrasion* or roughening of the cuticle occurs, the same precautions in regard to pressure, moisture, and the use of the draw-sheet have to be observed; and in addition some specific applications are used. Sometimes the part is greased over with zinc-ointment or vaseline; this is useful in certain cases, preventing urine or other irritating fluid from touching the tender parts. At other times collodion or flexible collodion may be used. The best fluid application is rectified spirit, or camphorated spirit, with one-third water; it is necessary to add water, owing to the pain caused by pure spirit. The surface may also be mopped over with a solution of two grains of perchloride of mercury in one ounce of spirit, or with a lotion consisting of five grains of nitrate of silver to an ounce of water.

3. The treatment of the *bed-sore itself* consists in keeping the part sweet; in aiding the removal of the slough; in applying some one of the many stimulant and antiseptic lotions in use; and in preventing the neighbouring tissues from breaking down. To clean the parts, and hasten the removal of the slough, apply a compress of wet salicylic or boric-acid wool. When the discharge is foul, the part should be washed with perchloride-of-mercury lotion (1-2000) between each application of the compress.

An excellent dressing is the Balsam of Peru, both before and after the slough has separated.

When the slough is large, wet applications should not be applied, as the moisture only favours putrefaction. To prevent this, dry the sloughing surface with absorbent wool; dust it over with iodoform, or boric-acid powder; place a piece of oiled silk or 'protective' exactly over the slough; cover the whole with dry cyanide gauze or boric wool. All dressings should be fixed by strips of plaster, and not by a bandage, owing to the heat and moisture engendered by use of the latter.

The neighbouring tissues must be treated with the preventive measures already mentioned.

When the slough has separated, an attempt is to be made to heal the part by improving the patient's general health, provided the existing malady permits. The usual precautions have to be taken with the surrounding parts; and the ulcer treated by one of the methods recommended under the head of Chronic Ulcers.

JAMES CANTLIE.

UNCONSCIOUSNESS.—See CONSCIOUSNESS, Disorders of.

UNDERCLIFF, on the South Coast of the Isle of Wight.—Extends from Bonchurch to Niton. A mild, tonic climate. See VENTNOR; and CLIMATE, Treatment of Disease by.

UNILATERAL HYPERIDROSIS.—Excessive perspiration on one half of the body occurs more or less habitually, or only after exercise. It may be limited to one side of the face and head, or the neck and arm may be included; or it may implicate the whole of one half of the body. It has sometimes, and especially when limited to the face and neck, seemed to be one of the symptoms due to paralysis of the cervical sympathetic nerve. In other cases no such relation has been ascertained to exist. It has been met with principally in association with various nervous diseases, such as hemicrania, diabetes mellitus, locomotor ataxy, dementia paralytica, and Graves's disease.

TREATMENT.—In the absence of definite information as to the pathology of this affection, it is not possible to indicate any rational principles or treatment. Empirically some of the remedies used for checking perspiration, such as zinc, belladonna, and quinine, may be tried, in combination with nerve tonics, such as arsenic and strychnine. See SUDORIPAROUS GLANDS, Disorders of.

H. CHARLTON BASTIAN.

URÆMIA.—SYNON.: Fr. *Urémie*; Ger. *Urämie*. **DEFINITION.**—This term is applied to a group of symptoms, acute or chronic, which may arise when the secretion of urine is gravely interfered with in the course of acute or chronic Bright's disease, and in other maladies.

ÆTIOLOGY AND PATHOLOGY.—Uræmia occurs when there is interference with the secretion or discharge of urine; hence it is met with in all the forms of Bright's disease, in cystic, tubercular, and cancerous disease of the kidney, in suppurative nephritis, and in suppression of urine (anuria), whether of obstructive or of non-obstructive origin; in hysterical suppression, however, uræmic symptoms are generally absent. Although uræmia depends upon the accumulation within the body of poisonous products that should be eliminated by the kidneys, the immediate cause is not easy to determine. Without apparent alteration in general health, uræmic symptoms may suddenly develop. Sometimes, however, an increase in the toxic co-efficient may be attributed to conditions which provoke additional engorgement of the renal vessels, such as indulgence in alcohol or exposure to cold; the albuminuria of pregnancy may be associated with uræmic symptoms, though some women who suffer from Bright's disease may pass through pregnancy without any uræmic manifestations.

Various explanations of the uræmic process have been suggested, and it is generally admitted that it depends upon some interference with the eliminative

work of the kidney, by which some urinary constituent is retained within the system, and either exerts its influence directly or after undergoing some chemical change. The suggested explanations may be divided into (1) *Mechanical*; (2) *Chemical*.

1. *Mechanical.*—Owen Rees noted wateriness of the brain in many cases of uræmia, while Traube laid stress upon anæmia of the brain induced by œdema, which in turn he attributed to marked hydræmia and increased arterial pressure; he thought coma would ensue when the whole cerebrum was thus affected, while convulsions would occur when the middle lobes were mainly involved. Traube's theory is favoured by (1) the frequency of cardiac hypertrophy and of hydræmia in uræmia; (2) the production of coma and spasms in dogs by the injection of water into the carotid after ligation of the ureters and of one of the jugular veins. Although supported by numerous experiments, Traube's theory is not wholly satisfactory, since in clinical work cardiac hypertrophy and hydræmia are not always accompaniments of uræmia, while *post-mortem* examination frequently fails to demonstrate œdema of the brain, and even when present œdema may be a result of the convulsions, and not the cause. Still, in a modified form Traube's explanation has found several adherents. Leichtenstern and Rühle suggest modifications which link together the mechanical and the chemical theories; the former holds that in scarlet fever inflammatory œdematous conditions of the brain and its meninges co-operate with the action of some result of infection, while Rühle believes the attacks to depend upon the admixture with the dropsical fluid of a toxic substance formed in the blood.

2. *Chemical.*—One of the earliest theories of uræmia referred the process to the non-elimination of urea, its circulation in the blood, and its deposition in various parts of the body. This theory is favoured by the fact that when the excretion has been experimentally arrested by ligation of the renal arteries or of the ureters, or by extirpation of the kidney, many of the ordinary symptoms, such as drowsiness, convulsions, vomiting, and diarrhoea, are produced. The acceptance of this theory on the other hand is difficult, since frequently there may be no symptoms of uræmia, though large quantities of urea may be detected in the blood; moreover, urea administered by the mouth provokes diuresis instead of producing toxic effects. Frerichs thought uræmic symptoms might result from the transformation of urea into ammonium carbonate under the influence of a ferment. Treitz considered that this change occurred in the alimentary canal rather than in the blood. Others have thought that urochrome might be decomposed in the circulation, or have attributed toxic action to creatin, creatinin, or other extractives. Feltz and Ritter maintain that uræmia is due to potassium-salts, while Bouchard suggests that, in addition, certain organic metabolic products of the nature of ptomaines may be developed; it must be admitted, however, that these various products have not been isolated, and that their existence is purely hypothetical. The influence of the failure of the internal secretion of the kidneys is as yet unknown. At present it is impossible to accept any single theory of explanation; probably numerous causes contribute to the result, and their individual importance is not always the same, otherwise it

would be impossible to account for the different types of uræmia. In short, although the term 'uræmia' is convenient, it is possible that it includes several allied conditions whose causation is not identical.

SYMPTOMS.—The symptoms which result from auto-intoxication through deficient renal action are extremely diverse; many are to be attributed to changes in the nervous system; these may be manifested through the sensory system, the motor system, or through modifications of the other cerebral functions. A gastro-intestinal type has also been recognised, though in these cases the symptoms are probably due to eliminatory efforts rather than to nervous influence. Changes in respiratory functions may be due to both causes, although nerve-influence usually predominates.

It is convenient to divide uræmic symptoms into two classes, *acute* and *chronic*.

1. *Acute uræmia.*—This includes all varieties in which the symptoms are suddenly developed. The onset may be marked by convulsive seizures or by coma: these occur without warning, or they may be preceded for a few hours or days by premonitory symptoms, such as severe headache, apathy, drowsiness, and vertigo; nausea or vomiting may occur; more rarely there may be severe dyspnoea, pains in the extremities, spasmodic contraction of the muscles of the face or of one of the limbs. Charcot also mentions tremors of the extremities among premonitory symptoms.

Uræmic convulsions are of an epileptiform type, and range from slight passing contractions to violent spasms affecting the whole of the body. During the convulsions the temperature is usually subnormal; in exceptional cases, however, the fall of temperature for a short time is preceded by a sudden elevation to 105° F. or higher. The clonic spasms are of short duration, and, as they gradually cease, the patient passes into a drowsy or comatose condition; from this he may awake as out of profound sleep. In unfavourable cases fresh convulsions may occur, and in the intervals between the attacks, the coma become more profound. Death sometimes occurs at the height of the paroxysm or during the coma.

The convulsions may be unilateral, and they may be followed by tonic spasm of tetanic type. Frequently during the convulsion and the subsequent coma there is noisy, hissing dyspnoea, which is not attributable to laryngeal or pulmonary obstruction.

In another type of acute uræmia, after similar premonitory symptoms or without warning, coma is rapidly developed without any preceding convulsions. The coma may rapidly deepen, and death ensue within a few hours; or the patient may rally and all urgency may pass away; ordinarily, however, further uræmic symptoms may be expected. Occasionally, without convulsions or coma, delirium of a restless or active nature may supervene.

Any type, but especially the convulsive, may be followed by transient hemiplegia, hemianæsthesia, and aphasia; but, even when the face is affected, ptosis is exceptional. Bilateral amaurosis is frequent; this may be partial or complete; it is not accompanied by any obvious retinal changes, and sight is ordinarily recovered in from 24 to 36 hours. Complete or partial deafness and tinnitus have rarely been noted. After acute uræmia there is occasionally marked lethargy, a long pause preceding the answer to a question.

2. *Chronic uræmia.*—In this the cerebral symptoms are less striking, and disturbances of respiration and digestion are more frequent, though they may be due to cerebral toxic influences.

The cerebral symptoms consist of frontal or occipital headache, usually dull and persistent, but sometimes acute and paroxysmal. The headache is often associated with lethargy and slow, indistinct articulation, sometimes with dimness of sight and ringing in the ears. Frequently there is severe insomnia, though there may be drowsiness during the day. Giddiness, languor, and inertia increase, and may eventually lead to coma, delirium, or convulsive seizures like those of acute uræmia. Definite psychical disturbances, with hallucinations and insomnia, may mark exacerbations of the primary disease. Neuralgic pains and pruritus are also to be mentioned.

Uræmic dyspnoea is frequent, especially when the primary disease results from alcoholism; sometimes the attacks occur at night only, like those of spasmodic asthma. In advanced cases similar difficulty of breathing may be due to gradual circulatory failure, with pulmonary engorgement or effusion into the pleural cavity. Persistent hic-cough and Cheyne-Stokes respiration are not uncommon shortly before death.

Disturbances of the functions of the alimentary canal are extremely common. These may range from chronic dyspeptic troubles to severe vomiting and diarrhoea. Sometimes these symptoms are associated with definite lesions, but frequently they appear to result from central irritation. Vomiting and diarrhoea are often terminal symptoms of chronic Bright's disease.

Erythematous, papular, and vesicular rashes, often associated with desquamation, are sometimes associated with uræmia, while epistaxis, intestinal and cutaneous hæmorrhages occasionally occur.

DIAGNOSIS.—No great difficulty is experienced when the previous history is known or when there is definite evidence of Bright's disease in alterations of the urine, in the character of the heart and the pulse, or in the presence of general dropsy. Outward displacement of the apex-beat, accentuation of the cardiac sounds, high tension of the pulse, and albuminous urine afford indications of great diagnostic value.

In acute uræmia the nature of the convulsions and coma should be considered; the former may be mistaken for hysterical or epileptic seizures, while the latter resembles alcoholism, opium-poisoning, diabetic coma, and cerebral hæmorrhage into the pons. Hemiplegic and paralytic conditions are more frequent with cerebral hæmorrhage, which may be associated with chronic forms of Bright's disease; they may sometimes be distinguished by their greater duration. An ammoniacal odour of the breath may aid the diagnosis of uræmia, but for this purpose the temperature is of very little value; it may occasionally be high with the onset of acute uræmia, and is generally normal or subnormal at the onset of apoplexy. Uræmic coma in its early phases is rarely so deep as that of cerebral hæmorrhage; the patient may be momentarily roused to answer questions; and the hissing dyspnoea of the former differs greatly from the stertorous breathing of the latter. In opium-poisoning the pupils are contracted; in uræmia their condition may vary, and they usually respond to stimulation.

The diagnosis of chronic uræmia must frequently be based upon the results of treatment, as well as upon consideration of the state of the urine and the consecutive changes in the circulatory system. Dyspeptic symptoms and vomiting may be ascribed to uræmia when they persist in spite of treatment, when there are definite indications of chronic renal trouble, and when there are no physical signs pointing to malignant disease; the examination of the vomit and the detection of urea, or the recognition of alterations in the proportion of free acid, will afford material help.

PROGNOSIS.—Uræmic symptoms necessitate a grave prognosis. With acute uræmia death may occur in rare cases during the first attack, but the danger is greater when attacks are repeated at short intervals and the coma deepens. In acute nephritis the prognosis is more favourable when consciousness is rapidly regained and when the urine shows signs of improved eliminative work.

Some of the symptoms of chronic uræmia, such as rigidity, headache, and insomnia, are of prognostic significance as indications of progressive stages of a chronic disease, but their gravity is greatly increased when they are accompanied by a reduction in the urine, and by the development of lethargy and coma. Uræmic dyspnoea, persistent vomiting and diarrhoea, add largely to the immediate danger, while prolonged stupor, coma, or convulsions in chronic nephritis are of fatal import. Puerperal cases, although involving great immediate danger, are more fatal when they precede delivery; the prognosis is influenced by the number of seizures, their duration, and the degree of coma between the attacks.

TREATMENT.—In acute uræmia the first indication is to provide for the elimination of nitrogenous waste pending the re-establishment of the work of the kidneys. This may be effected by diaphoretics and purgatives. Warm baths, warm packs, or vapour-baths may be of great service in acute uræmia associated with dropsy. Dry cupping, leeching, or poulticing over the loins may give relief. In acute cases the removal of eight or ten ounces of blood by venesection and the injection of saline solution have been beneficial. Hydragogue purgatives favour elimination; compound jalap powder or jalap and scammony may be given when consciousness is retained, but when coma is profound croton oil may be administered. To promote diaphoresis the subcutaneous injection of nitrate of pilocarpine has been advocated; it is now very rarely employed, as it causes much distress and may seriously embarrass respiration. When there are no indications of active engorgement of the renal vessels diuretics are frequently of service, especially in chronic uræmia. Digitalis is useful as a diuretic and also for its influence upon the heart in chronic nephritis. When convulsions are severe chloroform may be employed, or bromides may be administered; the subcutaneous injection of morphine has also been advocated, but, although of service in some cases, it is generally considered to increase the risks of further convulsions; the rectal injection of chloral hydrate is preferable, unless contraindicated by the condition of the heart. Milk should be given freely, and in acute uræmia it should constitute the sole diet. Inhalations of oxygen often relieve spasmodic dyspnoea, while nitroglycerin, amyl nitrite, and other nitrites may control the severe headache, though sometimes

antipyrine and phenacetin are preferred. The diarrhoea of chronic uræmia should not be checked unless sufficiently profuse to be exhausting; efforts should, however, be made to control vomiting by iced drinks, by sucking fragments of ice, or by the use of bismuth and hydrocyanic acid. When convulsions occur during pregnancy, labour should be induced.

NESTOR TIRARD.

URETERS, Diseases of.—**SYNON.** : **FR.** *Maladies des Uretères*; **GER.** *Krankheiten der Harnleiter*.—Morbid conditions of the ureters are so generally parts of, or associated with, diseases of the bladder or kidney, and so naturally come to be described in the articles treating of these several diseases, that a separate account or distinct classification of them is perhaps scarcely called for; and a brief description of the most important will be sufficient for all practical purposes.

1. **Congenital Malformations.**—A double ureter is the most frequent malformation, the division sometimes reaching as far as the bladder. In very rare cases a triple ureter has been found. Congenital malformations leading to more or less complete obstruction of the ureter are found most often near the pelvis of the kidney. If the obstruction is complete, it gives rise to hydronephrosis (*see* **HYDRONEPHROSIS**). If incomplete, a slighter degree of the same condition results, the pelvis and calyces becoming dilated, and the kidney-substance atrophied and indurated. The chief congenital obstructions are—(1) Total obliteration of the ureter as it leaves the pelvis. (2) Valvular opening from the pelvis into the ureter. This becomes gradually more complete as the dilatation of the pelvis increases. (3) A spiral arrangement of the ureter. (4) An abnormal renal artery pressing on the ureter as it leaves the pelvis. The incomplete obstructions frequently give way at intervals, when there will be a copious flow of urine, usually of low specific gravity and pale colour.

2. **Acquired Obstruction.**—*Complete* obstructions which are not congenital may arise : First, from impaction of a renal calculus; this may take place at any point, the most frequent being at the brim of the pelvis or the vesical orifice. Secondly, from external pressure, as from uterine cancer, pelvic tumours, fibrous bands, enlarged glands, or fecal accumulations, in which case the condition would at first be incomplete. *Incomplete* obstruction arises most frequently from impediments to the discharge of urine, existing in the prostate or urethra. In the former case, the bladder being in a state of constant over-distension, the valved opening of the ureter is closed with unnatural force. In the latter the hypertrophy of the bladder increases the length of the portion of ureter lying in the bladder-walls, and the bundles of muscular fibres more or less constrict the orifice. Any cause giving rise to hypertrophy of the bladder will therefore tend to obstruct the orifice of the ureter, as is seen in chronic cystitis, stone in the bladder, and other conditions. The opening is still further narrowed by swelling of the mucous membrane, the result of subacute or chronic inflammation. Occasionally the orifice may be partially obliterated by a villous growth in the bladder. *Stricture* of the ureter is found in rare cases, apparently the result of previous inflammation and ulceration. Slight obstruction may occur during pregnancy, from the pressure of the gravid uterus.

3. **Dilatation.**—The part of the ureter above any obstruction is always found more or less dilated, according to the degree and the duration of the impediment. If the condition be acute and complete, little or no hypertrophy of the coats of the ureter will be found; but if chronic and incomplete, its walls will be thickened, both by fibroid change and by hypertrophy of the muscular coat. The dilated ureter is always tortuous, being increased in length as well as in diameter, and may even resemble a portion of small gut. The mucous membrane is always opaque and somewhat thickened, occasionally red and injected, and frequently pigmented from previous attacks of inflammation. A largely dilated ureter may sometimes be felt through the abdominal wall. There will probably be some degree of pain or uneasiness in the course of the canal or in the loin, and a tendency to sickness.

4. **Inflammation.**—Simple inflammation of the ureter is frequently found, as an extension either from the bladder or from the pelvis of the kidney. Septic inflammation accompanies septic pyelitis, due to extension of decomposition from the bladder to the pelvis of the kidney (see p. 823). Tubercular inflammation almost invariably accompanies a similar condition of the bladder or kidney.

5. **Malignant Disease.**—Cancer may extend from neighbouring organs into the ureter, but there is no reason to believe it ever primarily takes origin there.

Neither the diagnosis nor the treatment of an affection of the ureter can be separated from that of the disease to which it is secondary.

MARCUS BECK.

H. MONTAGUE MURRAY.

URETHRA, Clinical Examination of. —

Examination of the male urethra can be effected: (1) by the use of bougies, both metallic and elastic; (2) by the electric air-inflation urethroscope.

The former method is principally adapted for the detection of strictures, both traumatic and inflammatory in their origin, the latter to the diagnosis and treatment of the cause of stricture, which in the majority of cases is chronic gonorrhœal inflammation.

In the detection of strictures by bougies it is well always to commence with the passage of a large instrument. The author advises the use of an acorn-shaped head on a slender shaft, which goes by the name of Otis's exploratory bougie, the metal being preferable to the gum-elastic. By these instruments much greater accuracy in location of stricture may be obtained than by the use of the ordinary bougie, whether of steel or gum-elastic—which instruments are better reserved for treatment. When the exploratory bougie approaches the face of the stricture its passage is arrested. A smaller instrument is chosen again and again until the size that can be passed is reached. In many cases this instrument will pass into the bladder. In a certain proportion it will be arrested by a tighter stricture at a greater distance from the meatus. Smaller instruments again are taken, and the manœuvre repeated. But possibly the smallest 'explorer' (or exploratory bougie) will not pass, when we shall have to fall back on some form of filiform bougie with which to gauge the calibre of the stricture. One of the objects in taking a large instrument to commence with is that the presence of strictures of large calibre, through which a smaller

bougie would slip readily, may be detected. In connection with the use of both rigid and pliable bougies, attention must be drawn to the fact that in sensitive urethræ a spasm of the compressor muscle is frequently induced. So tightly does this muscle close the canal, that in many text-books will be found a description of a condition known as 'spasmodic stricture.' A diagnosis of stricture has often been made quite erroneously from this cause. The injection of a solution of cocaine in such cases by an urethral syringe will be followed by the easy passage of the instrument through what appeared at first sight an impassable stricture.

Considerable assistance is afforded in the examination of the urethra by digital palpation. Thickened and hardened portions of the penile urethra can be detected, and by a finger in the rectum a certain amount of knowledge can be gained as to the condition of the membranous and prostatic portions of the canal. In the case of a chronic gleet, where it is desirable to locate the portion of the canal most affected the following simple manœuvre is often of service:—

The patient is desired to hold his urine as long as possible, preferably to come to the surgeon after a night's rest without passing water. The anterior urethra is then washed out with boiled water into a 12-ounce conical specimen-glass. (It must be premised that for clinical purposes the male urethra may be divided into two portions—the anterior stretching from the external meatus to the compressor urethræ or 'cut-off' muscle, the posterior from this muscle to the internal meatus.) For this purpose a 6-ounce syringe of glass, vulcanite, or metal, with a conical and detachable nozzle, may be used; a shield attached to the syringe to prevent loss of the liquid as it spurts from the meatus is advisable. An ordinary brass or large glass dressing-syringe will serve all practical purposes, or an irrigator-tank may be used, fitted with a suitable nozzle and stop-cock. With the latter apparatus care must be taken not to hang the tank at too great a height, or the compressor will be forced and our object defeated. The anterior urethra having been washed out, the patient is directed to pass urine into a second glass, and, according as the first or second specimen contains the threads and particles of inspissated mucus, we know roughly the portion of the canal most affected.

Examination by the Urethroscope.—There are several forms of urethroscope in use, but perhaps

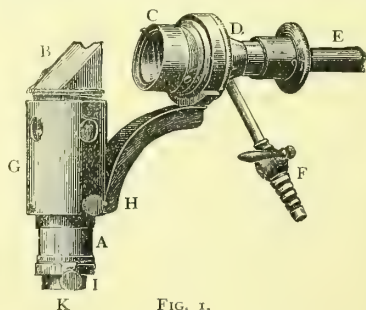


FIG. 1.

the most convenient and serviceable is that known as the Schall urethroscope (fig. 1). In this instrument the light is furnished by an incandescent lamp fixed in a metal holder (A). The lamp casts its light

upwards into a hood (B), which contains an arrangement of prisms by which the beam of light is rendered horizontal and projected towards (C) a glass window closing the short barrel (D), on which fits the cannula E. The tube (F) fitted with a stop-cock is connected to the rubber tube of an air-inflation bag. The light-carrier (A) moves easily in the larger chamber (G), and on its upward or downward movement the focussing of the light depends. When the light is focussed the carrier is fixed by the screw (H). The current is carried to the lamp by an ordinary insulated wire, terminating in a plug which fits into the carrier at (K), the current being made or broken by the turning of the screw (I). The urethra should first be anaesthetised by the injection of a 2-5-per-cent. solution of cocaine, or if preferred β -eucaine may be used. If the anterior urethra only is to be examined, an ordinary urethral syringe is sufficient to inject the solution, the nozzle being inserted just within the meatus. If, however, it is desirable to examine the deep urethra with the light, it is necessary to pass a catheter, or, better, a Guyon's tube (fig. 2), through the compressor, and to inject behind that muscle. The cannula should then be passed, no force being used, and if, as is often the case, the meatus is too small to take a full-sized tube, thorough dilatation or a cut with a

as we proceed. The normal urethra varies considerably in appearance, according as it is distended with air or in a flaccid condition. If the latter, the mucous membrane is of much the same colour as the healthy palpebral conjunctiva, and is folded in a radiating manner at the end of the tube. But when distended it is a more or less cylindrical tube that we see stretching away to the point where the urethra curves, the walls glistening, whitish, or pale pink in colour, and where much distension is employed the circular muscular fibres stand out in the form of white rings, and might be mistaken for stricture. Some of the openings of Littre's glands may be seen as shallow depressions, and on the floor, just in front of the compressor, are the openings of Cowper's ducts, generally invisible. The compressor shuts off the deep urethra from view, occasionally opening a little to allow the passage of air if the pressure is increased.

In the examination of the membrano-prostatic urethra a cannula with a slight elbow-bend like a *coudé* catheter, and open on the floor, or a straight tube of rather smaller size than those described, may be used. In this case air-distension is not available, as the air would simply pass into the bladder. In the centre of the urethral floor is seen the verumontanum with the sinus pocularis, on either side of

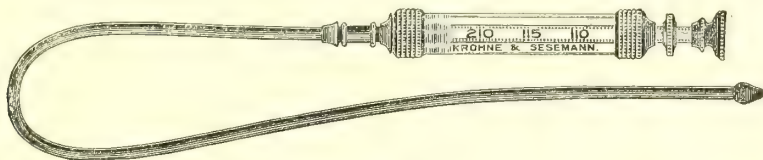


FIG. 2.



FIG. 3.

hystoury will give room. The largest tube that will pass without force is the best to use, for the larger the tube the better is the illumination. No. 24 or No. 26 (French gauge) cannulae, open at the end (figs. 3 and 4), are useful sizes, and the adult urethra will usually take the larger size easily, except sometimes at the meatus. The urethroscope is then fitted on to the end of the cannula and the light

which is the opening of the vas deferens; here the mucous membrane is of a much deeper colour than in the anterior urethra.

F. SWINFORD EDWARDS.

URETHRA, Diseases of.—SYNON. : Fr. *Maladies de l'Urèthre*; Ger. *Krankheiten der Harnröhre*.—The affections of this canal comprise urethritis with its various consequences, such as gleet, warts, periurethral abscess, inflamed follicles, and stricture; syphilis and soft chancre may also affect the urethra. For descriptions of most of these affections the reader is referred to the articles GONORRHOEA; GLEET; SYPHILIS; and VENEREAL SORE. Some forms of (1) Urethritis, (2) Neuralgia, (3) Congestion and Spasm, (4) Organic Stricture, and (5) Foreign Bodies, remain for consideration here.

1. Urethritis.—The origin of urethritis is not exclusively due to inoculation with gonorrhoeal pus, a fact of great medico-legal importance. Inflammation of the urethra, usually running a less severe course than the gonorrhoeal form, may be set up by leucorrhoeal, menstrual, and other discharges from the vagina, or result from the use of an injection which a patient may have used in the hope of warding off venereal disease, which he dreads having contracted. Traumatic irritation of the

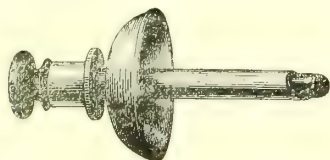


FIG. 4.

turned on. The excess of cocaine-solution must be removed with cotton-wool on a fine sponge-holder. The window (c in fig. 1) is then closed and air admitted by turning the tap (at F, fig. 1).

The examination of the anterior urethra should first be undertaken. The cannula should be introduced only just within the meatus, and the air then turned on. By this means the urethra is inflated as far as the compressor, and the tube can be easily passed down, the state of the walls being examined

urethra may be excited by the passage of instruments or a calculus, or by the introduction of foreign bodies by the patient; in such cases soreness and scalding attend the first subsequent micturition, and swelling and discharge follow within twenty-four hours.

Asparagus, beer, arsenic, and other substances, have the power of producing urethritis in some persons; highly acid urine and oxaluria may also occasion slight urethral irritation. A very mild form of urethritis is an occasional concomitant of early syphilis; it is, however, always very limited in extent, and subsides spontaneously. In gouty urethritis the inflammation may be as severe as in well-marked gonorrhœa, and attended by the complications met with in that disease, and affecting the testes, bladder, eye, or joints. The leading distinctions of gouty urethritis are the milkiness of the discharge, and the absence or small amount of swelling of the fore part of the urethra; while scalding in the perineum and irritability of the bladder are severe and prominent symptoms.

Tuberculosis sometimes affects the urethral passage. It is always a very indolent affection, being due to the slow degeneration and ulceration of tubercular deposits in the deeper portions of the canal, and usually associated with similar disease in the prostate or bladder.

In such cases the irritability and inflammation of the bladder are always more urgent than the urethritis, while the evidence of tubercular disease in other parts of the body is usually sufficient for diagnosis. The treatment of urethritis arising from any of these causes necessarily varies in detail with such cause. The local treatment is similar to that for gonorrhœa, and is described in the article dealing with that subject.

2. Neuralgia. Pain in the Urethra.—The cause of pain in the urethra may be found in some diseased condition of the tube; or it may be independent of any local morbid action. Gouty irritation is a frequent cause, and a nervous condition produced by prolonged debauchery of various kinds is not uncommon. The gouty state is relieved by the treatment of the diathesis; the nervous irritation by suitable general treatment, to which may be added local applications which have the effect of dulling the sensibility of the urethra, such as the passage of sounds, and the use of astringent or (in obstinate cases) of caustic injections to the deeper portions of the urethra. In some cases instrumentation of the urethra excites severe pain; this is best avoided by first injecting about thirty minims of a 10-per-cent. solution of cocaine. Oxaluria and a highly acid state of the urine may sometimes produce considerable smarting at the time of micturition; the exhibition of alkalis and suitable dietary will usually suffice to relieve this, which is often a source of considerable annoyance to the patient.

3. Congestion and Spasm of the urethra are sometimes incorrectly described as congestive or spasmodic stricture; incorrectly, for although complete retention may arise from these conditions, which are due to transient causes, the effects are temporary only, and involve no permanent lesion.

Congestion and swelling of the urethral mucous membrane, with proportionate narrowing of the passage, accompany all forms of urethritis, and may also result from any cause of mechanical irritation, from exposure to cold, sexual or alcoholic excess, riding, bicycling, and the like. Simple congestion

rarely calls for much attention, provided there be no other abnormal condition present, but in cases of organic stricture or of prostatic enlargement the advent of congestion may lead to complete retention. Under such circumstances a soft indiarubber catheter should be passed, if possible; but if this cannot be accomplished, the patient should be placed in a hot bath and a morphine or belladonna-and-camphor suppository should be prescribed. The bowels must be acted on by saline aperients and complete rest enjoined. The congestion usually rapidly subsides, and the patient must then be warned of the circumstances likely to lead to its reappearance.

Spasm of the muscular fibres surrounding the urethra, and of the deep perineal muscles, may accompany or be independent of congestion of the mucous membrane. It is induced by any cause of mechanical irritation, and occurs in cases of stricture of any part of the canal, especially if a rude attempt be made to pass an instrument. Spasm also sometimes results from irritation about or operations on the rectum. A highly acid state of the urine may induce spasm of a painful nature. The treatment is practically that indicated for congestion; if spasm is sufficient to arrest the onward passage of an instrument, it usually may be overcome by steady gentle pressure for a few moments, but the instrument must never be forcibly pushed onwards; chloroform is occasionally, though rarely, necessary.

4. Organic Stricture.—The average distensile capacity of the urethra reaches a diameter of ten, or a circumference of thirty, millimetres. This measurement varies in different portions, being widest behind the triangular ligament (the anterior layer of the deep perinæal fascia), slightly less in the bulbous portion, and least of all at the meatus. The meatus varies very much in size, between a mere pinhole and a diameter of fourteen or fifteen millimetres, the most common size being a circumference of twenty-five millimetres. In different persons the general distensile capacity of the urethra ranges between twenty and forty millimetres in circumference.

Strictures are abrupt abnormal contractions of the urethra at any given point of its course.

In the great majority of instances organic stricture is the result of long-continued inflammation following gonorrhœa, leading to exudation into the sub-mucous tissue. This exudate subsequently organises into a low form of fibrous cicatricial tissue, the contraction of which leads to the permanent narrowing. A similar result may also follow on injury of the urethra (*traumatic stricture*), and is then more severe, the new tissue being very dense. The cicatrix left by the healing of a urethral chancre may likewise cause stricture. Congenital narrowing of the meatus is common, and congenital strictures are sometimes met with within the second inch from the meatus.

Organic strictures may be situated at any part of the urethra except the prostatic, being most common in the first inch from the meatus.

The tightest and toughest strictures are generally found near the junction of the bulbous and membranous portions of the urethra; that is, about five inches from the meatus. The membranous portion is only affected when there is a large amount of cicatricial tissue spreading backwards from the bulb. The new fibrous tissue is developed in various

orms; thus it may be present as a membranous fold stretching across (*bridle-stricture*), or occasionally encircling (*annular stricture*) the canal. There may be only one bridle or several.

Superficial scars of healed ulcers and warts may give rise to patches of tough tissue. Indurated areas and fibroid nodules in the submucous tissue, standing forwards on the surface of the canal, cause irregular projections as well as unyielding indurated patches. These twist the course of the canal and impede the passage of bougies. In other cases, instead of being in limited patches, the indurated tissue may extend evenly along the canal for some distance, making it smooth and gristly, and diminishing its calibre.

The indurated new tissue may form only a thin layer of fibres beneath the mucous membrane, or may extend more deeply, infiltrating the whole thickness of the corpus spongiosum, or even extending beyond it into the tissues of the perinæum, rendering them dense and unyielding. The degree of narrowing of the passage depends in great measure upon the age of the stricture, being greater as time goes on; it may be temporarily increased by muscular spasm or inflammatory congestion. The canal never becomes obliterated, as the term 'impermeable stricture' would seem to imply. Strictures are always permeable to urine, but may not be so to the passage of an instrument. Strictures vary in behaviour: some yield easily to dilatation and slowly recontract; others are rigid, and can be dilated only to a small extent (*indurated strictures*). Lastly, some dilate quickly, but as quickly shrink back to their previous size (*resilient strictures*). These differences are in the main dependent on the age of the new tissue: when quite young the scar is soft and yielding, although after dilatation it may quickly recontract. In old strictures the fibrous tissue is dense, hard, and indurated.

SECONDARY EFFECTS OF ORGANIC STRICTURE. The urethra in front of a stricture may be quite healthy, but if the condition has existed for some time the mucous membrane between the meatus and narrowest contraction is often puckered and indurated, or is excoriated or ulcerated. Sometimes just in front of the stricture artificial openings are found; these are the mouths of false passages which have been made by inexpert instrumentation. False passages may, after passing outside the urethra in front of the stricture, run for some distance, ending as *cul-de-sacs*; or they may reopen into the urethra behind the stricture; less often they extend up for some distance between the bladder and rectum, or open into the latter. At the seat of stricture the mucous membrane is often roughened, puckered, and of a dull white opaque colour; behind it the urethra is dilated, pouched, pigmented, and even ulcerated; the mucous membrane may be coated with muco-pus, sometimes mixed with phosphates. The pouches, formed by distension of the follicles, may be the starting-point of perinæal abscess, which may burst into the urethra or on to the surface of the perinæum; or, lastly, may burrow upwards between the bladder and rectum and round the prostate. If a perinæal abscess bursts into the urethra and on to the surface of the perinæum, fistulous tracts are formed, which ultimately become very dense and unyielding. Such fistulae are often multiple, and may open in various situations. The bladder, ureters, and kidneys will, in neglected cases, show evidence of secondary mischief. The

changes induced are partly dependent upon the obstruction to the free flow of urine, and partly inflammatory as the result of decomposition of the retained urine. Thus the bladder-walls hypertrophy in order to overcome the obstruction; but if the compensation is inefficient, dilatation and sacculation will ensue. The ureters dilate, and the walls hypertrophy; the pelvis dilates, and the pyramids of the kidney-substance become flattened, crescentic, and subsequently wasted; the remaining renal substance is stretched and sclerosed, and the condition known as hydronephrosis results. If the urine should decompose, acute cystitis, becoming chronic, will follow, and the micro-organisms will find their way to the ureter and pelvis of the kidney, exciting pyelitis, and in severe cases acute nephritis with numerous points of suppuration—the so-called 'surgical kidney' (see p. 823). These results only occur in cases which have not been treated at all, or only imperfectly.

SYMPTOMS AND DIAGNOSIS OF ORGANIC STRICTURE.—The symptoms of organic stricture of the urethra are more evident in the case of an old and narrow stricture. Few symptoms attract observation before the stricture is well advanced. The most common and the earliest is a scanty muco-purulent discharge. This sign is, indeed, very often the indication of the chronic inflammation of limited areas of the passage which ends in stricture. When the stricture is developed the symptoms are more definite, namely, slight dribbling of urine after micturition has apparently ceased; small size of the stream, which may be forked and twisted; and gradually increasing difficulty, with straining and pain, in micturition, which is more frequent than normal. Any or all of these symptoms may be absent, even when the canal has lost as much as two-thirds of its distensibility. Usually to these troubles are added occasional attacks of complete retention of urine from temporary spasm or congestion. There may also be the signs caused by the various secondary complications.

Examination of the urethra by means of a steel sound or gum-elastic bougie will reveal the number and situation of the strictures present. The distensible capacity of the various parts of the passage can be determined by the urethrometer.

TREATMENT.—The treatment of organic stricture consists, first, in removing from the habits and diet of the patient all causes of functional disorder.

Temperance in alcoholic drinks, in stimulating or highly seasoned foods, in sexual indulgence, and in some forms of exercise, such as horse- and bicycle-riding, must be enforced. Due attention to the condition of the skin and bowels, and sufficient bodily exercise, are requisite. Although attention to these matters will not in any way remedy the existing condition of a stricture, yet they will prevent congestion and spasm, and so obviate the temporary and often dangerous aggravation of the sufferings caused by these conditions.

Locally the passage must be restored to, and maintained at, such calibre as, on the one hand, will enable the bladder to empty itself completely by each act of micturition; and, on the other, will allow a sufficient margin to prevent stoppage of the flow of urine during occasions of temporary swelling or spasm. Such an expansibility would seem for most persons to be about equal in circumference to that of No. 10 English; though in practice it is advisable to dilate somewhat further (No. 12 English)

than this, to allow sufficient margin for neglect on the part of the patient. In all organic strictures there is a tendency to contract again; hence, when sufficient expansibility has been established, it must be maintained by the frequent passage of a bougie. In most cases the patient should be taught to do this for himself.

Various methods, none of which is applicable to all cases, are employed for dilating the strictured part of the passage. Some methods, such as the use of caustics, or the cautery and forcible dilatation, have fallen into desuetude; those in modern use are (a) gradual interrupted dilatation; (b) gradual continuous dilatation; (c) internal urethrotomy; (d) external urethrotomy.

(a) *Gradual interrupted dilatation* is attained by passing metal sounds or flexible bougies (preferably the latter) through the stricture. At the first sitting that size which will just pass through the stricture should be used; subsequently larger sizes must be used, until No. 12 or 13 English can be passed. It is probable that in most, if not all, cases the stricture is partly stretched and partly torn in this method of treatment. The instruments may usually be passed at intervals of two or three days.

(b) *Gradual continuous dilatation*.—In this method a flexible catheter is passed through the stricture and tied in. As the stricture yields the instrument must be removed, and a larger one introduced from time to time, lest, being loose, the catheter escape from the urethra. A week or ten days usually suffice to dilate the stricture to the requisite dimensions. The nature of the process appears similar to that of a seton in ordinary sinuses or cellular tissue; the fibrous tissue becomes loose and succulent, and its fibres permeated by leucocytes; superficial ulceration with destruction of the cicatricial tissue also occurs in some cases.

(c) *Internal urethrotomy* is accomplished by means of a concealed knife conducted to the stricture by running in a split sound which passes through it into the bladder. As the knife is pushed on it is made to project and divide any resisting tissue met with. A full-sized catheter is then passed, and the bladder emptied. At the end of ten days instrumentation should be commenced, in order that the passage may be kept properly dilated.

(d) *External urethrotomy*.—When attacked from without, the situation of the stricture is indicated by a staff passed down to or through it. In the former case the cicatrix is divided from before backwards, and in the latter from behind forwards. Cutting from before backwards is the more common operation, since if the staff can be passed through the stricture internal urethrotomy can usually be done. If the urethra is laid open in front of the stricture the orifice must be sought for—too frequently a long and tedious process—and the contraction then slit up along a fine director which has been passed into the bladder.

Various instruments have been devised and are employed for carrying out the methods shortly described here, some more suited than others to particular cases.

Comparison of the different methods.—Gradual interrupted dilatation is applicable in a larger number of cases than is any of the other methods described. It is attended by least evil consequences, and is most effectual in strictures of recent formation situate near the bulb. Objections to this method are the rapidity with which recontraction takes place

if the passage of bougies be neglected by the patient, and the impossibility in some cases of dilating the stricture beyond a certain calibre without exciting shivering, pain, pyrexia, or even local inflammation and abscess. Epididymitis is sometimes set up by the passage of instruments. Indurated strictures are sometimes so tough and dense that they cannot be sufficiently dilated.

Gradual continuous dilatation is easy and rapid, but is liable to be followed by speedy recontraction. It is useful in cases in which it is necessary to obtain a rapid dilatation of a very narrow stricture, in consequence of advanced vesical or renal disease. In these the bladder must be given rest without delay, yet the condition of the kidneys is most unfavourable to any operation. If there is great difficulty in passing an instrument into the bladder, it is advisable, when it is passed, to tie it in. The objections to continuous dilatation are that it confines the patient to bed, and in some cases excites local irritation and constitutional fever sufficiently severe to require its abandonment.

Internal urethrotomy is applicable with best results in young patients, in whom renal changes are absent or only slight. It is also preferable for those patients who prove intolerant of interrupted or continuous dilatation, since serious disturbance rarely follows incisions made in strictures between the meatus and the bulb. The locality of stricture has much to do with deciding upon the adoption of internal division. The shallow constrictions, so-called 'bridle-strictures,' and the elastic rapidly yielding and as rapidly contracting resilient strictures, are always situate in the penile portion of the urethra, and are specially amenable to incision, while they are very little affected by the passage of bougies.

The great thickness and toughness of certain strictures of the bulb render their division the only mode of securing even moderate duration of their dilatation. The recontraction of a stricture after it has been divided would seem to follow less speedily than after it has been treated by gradual dilatation.

External urethrotomy is needed in comparatively few cases. It is advisable when the perinæum is hardened and beset with fistulae; or in those still more rare cases in which no instrument can be introduced into the bladder. Under such circumstances it is requisite to attack the stricture from the surface.

5. Foreign Bodies impacted in the Urethra.—Foreign bodies of various kinds may be introduced into the urethra, and may lodge in the canal, impeding the free flow of urine, or even arresting it, and eventually exciting inflammation and abscess. Calculi passing from the bladder may lodge behind a stricture, or close to the meatus. Portions of a bougie or catheter may be broken off in attempts to pass a stricture. *See also* p. 332.

The symptoms pointing to the presence of a foreign body are usually pronounced. There is pain, with difficulty in passing water, and perhaps complete retention; while the foreign body can usually be felt in the canal, and can be seen through the endoscopic tube.

TREATMENT.—The foreign body must be removed by means of appropriate forceps introduced through the largest-sized endoscopic tube which can be passed. If this is found to be impracticable, the urethra must be opened from without.

CHARLES STONHAM.

URIAGE, in Isère, France.—Thermal muriated sulphur-waters. See MINERAL WATERS.

URIC-ACID DIATHESIS.—SYNON.: Lithæmia, lithiasis, Lithuria; Fr. *Diathèse Urrique*.

The most definite manifestations of what has been commonly called the uric-acid diathesis are seen in gout and uric-acid gravel. It is necessary, however, to discuss under the same head the more vague conditions of lithæmia, so-called; using the term with the somewhat special meaning first attached to it by Murchison, who ascribed certain symptoms, one of which is a more or less permanent appearance of uratic deposits in the urine, to a functional derangement of the liver, with excessive production of uric acid. To this condition Murchison gave the name 'lithæmia,' and the name, with the same connotation, has of late come into fresh prominence. It is further necessary to bear in mind in dealing with this subject, that a view has also recently gained attention in which uric acid is held to play a much more comprehensive part, and to be the chief factor in a large number of apparently very diverse disturbances.

But, since the deposition which occurs in gout and gravel is, almost certainly, not due to any increased production of uric acid in metabolism, but to quite other factors, any term meant to associate these two sufficiently dissimilar ailments would better be some other than that which heads this article. Again, closer examination seems to show that the condition called lithæmia by Murchison is scarcely capable of even loose definition; and, even if we are able to recognise the existence of a symptom-group consistent with his description, there is no proof that the condition it betokens is due to the special influence of uric acid. Lastly, those who ascribe to this substance the extensive influence in disease referred to above, expressly disavow any belief in a special diathesis; the troubles being assumed to arise from errors of diet and faulty elimination. It will be seen, therefore, that there is probably no justification for the retention of the term 'uric-acid diathesis,' except those of custom and convenience.

ÆTIOLOGY, &c.—Although uric acid is closely related to urea chemically, and takes the place of the latter substance in the urine of birds and reptiles, it is probable that in the mammal its metabolic history is quite distinct from that of urea. Recent physiological study has shown that of the total quantity excreted, only a part arises from the essential metabolism of the tissues. Another part takes origin directly from precursors in food-stuffs, and the amount of this portion depends to a great extent upon the quality as well as upon the quantity of the diet. The two moieties of the excretion—that which is independent of the intake and that which arises directly from it—are conveniently, if not very appropriately, termed the endogenous and exogenous portions respectively.

While the daily output of urea rises and falls with the amount of nitrogenous material absorbed, and, under normal circumstances, very nearly proportional to the amount of proteid ingested, the output of uric acid, on the other hand, varies only with the amount of certain quite special constituents in the diet. We may choose a dietary, rich in proteid, which scarcely affects the uric-acid excretion at all; in other words, upon such a dietary the exogenous production is absent. The essential

character of such a diet is freedom from organised tissue-elements. It must contain no muscle, especially no glands (sweetbread, liver, kidney, &c.), and none of the preparations which come under the head of soups and meat-extracts. With any of these the exogenous production of uric acid rises in proportion to the amount taken. A satisfactory explanation of these facts is ready to hand. As essential constituents of cells, and especially of their nuclei, are certain phosphorus-containing proteids—the nucleo-proteids. These are found, when hydrolysed by laboratory processes, to yield among their decomposition-products certain bases, the xanthin-, alloxur-, or purin-bases, which are closely allied to uric acid. Other proteids yield none of these, and no products so immediately related to uric acid. This distinction would seem to hold in the processes of metabolism, and it thus happens that only a dietary which contains nucleo-proteid, that is, one which comprises cellular elements—animal or vegetable—increases (in virtue at least of its proteid content) the exogenous uric acid. Proteids such as the myosin of muscle, the caseinogen of milk, the albumen of eggs, or the gluten of wheat, give rise to no increase when ingested. It is true that muscle, no less than the glands or other organs more or less commonly consumed as food, contains, in addition to its nucleo-proteids, certain products of its own previous metabolism ('extractives'), some of which may act also as precursors of uric acid; in the case, indeed, of an ordinary meat-meal these contribute a large proportion of the excretory increase which follows its consumption.

A dietary leading to scarcely any exogenous production may consist of such materials as milk, cheese, eggs, white bread, rice, potatoes, and most green vegetables, other than those of the leguminous variety.

For that moiety of uric-acid production in the body which is independent of diet—the endogenous product—it is, of course, more difficult to trace precursors. But the facts justify the supposition that it arises in the main from the metabolism of the nucleo-proteids of the tissues, and does not spring, in company with urea, from the breakdown of proteids in general. There is evidence that at least some fraction arises by synthetic processes occurring in the liver or, as is less likely, in the kidney.

The total excretion varies considerably in different individuals, but is never large. The limits may vary from 5 grains or somewhat less to 15 grains or a little more in 24 hours. From what has been said it will be understood that the amount excreted will be affected by the character of the food; but it may vary within the above limits in different individuals when all are taking an ordinary mixed diet. A person excreting, say, 12 grains upon an ordinary dietary may excrete as little as 6 or 7 grains upon a diet of milk and eggs; the latter figure indicating in this case what is practically his 'endogenous' production.

It is quite certain that the sufferer from lithiasis is not necessarily one whose uric-acid excretion is higher than the average.

We have but little experimental evidence bearing upon the amount of uric acid in the blood and tissues under normal conditions. Most recent observers have failed to find any at all in the blood. But it may be impossible to demonstrate it even in the blood of birds (Luff), and this fact has been used as an argument—not wholly conclusive—of its

formation in the kidney. In mammals, the small amount excreted makes it impossible to draw any such conclusion; 10 or 12 grains of an excretive could of course be brought to the kidneys in the course of 24 hours without detectable quantities being present at any moment in a sample of the blood. Conclusions drawn from older observations pointing to the presence of proportionately large amounts in the spleen are erroneous, in so far as such uric acid is probably formed in the organ *post mortem* from certain precursors. While we are without strict proof of the matter, there is much justification for looking to the liver as the chief seat of uric-acid formation.

As regards the quantity of the substance in the blood when pathological conditions exist, the classical observation of Garrod, showing the increase in gout, remains alone in the abundant confirmation which it has received. There is, however, no doubt that a large increase is found in certain types of leucæmia, here arising from increased production, and not merely from the retention—due to aberrations of solution and elimination—which is the feature of gout. The researches of v. Jaksch point to an increase in the blood in pneumonia, in renal disease—especially with granular kidney—and in cases of advanced anæmia. In none of these conditions can we assert that uric acid exerts *per se* a toxic influence in the proper sense, apart from mechanical effects due to its deposition. There is no toxic symptom common to these various disorders in which an excess of uric acid is present in the body. Certain combinations of the symptoms, which Murchison held to indicate a condition of lithæmia, are common enough in the dyspeptic, though it may be doubted if those he enumerated are often seen in such definite combination as to warrant their being grouped under a name suggesting that they have a common toxic factor in their production, and together constitute some sort of pathological and clinical entity. In any case, the name seems an undesirable one for such conditions as he describes, for there is no evidence whatever that such patients have any excess of uric acid in their blood. The symptoms enumerated constitute, it is true, a picture very similar to that seen in latent or irregular gout, just as they are also very similar to those of the chronic dyspeptic; but Murchison himself urged that 'they are also very common in persons who neither inherit articular gout nor ever have it themselves.'

That a tendency to deposition in the urine of pigmented urates goes commonly with certain dyspeptic symptoms is undoubtedly true, though it is probably due (as in colds and fevers) to the general character of the urine rather than to an increase in the day's excretion of urates, for the precipitation of these is never a proof of an increased proportion. It is also true that many people take habitually an excess of food of a kind which keeps their 'exogenous' uric acid high; and if they also happen to consume chiefly alcoholic beverages, with a proportionately small quantity of efficient diluents, they pass an acid concentrated urine naturally prone to deposit its urates. But the ills which follow this course of life are not to be attributed to any special toxic effects on the part of uric acid, nor can such individuals be said to suffer from a dyscrasia in any legitimate sense.

It is essential, at least at the present time, when 'the uric-acid diathesis' and drugs to combat it are

so insistently kept before the eyes of the medical profession and of the public, to bear in mind that, save in gout and gravel, where its physical properties and those of its salts play so indubitable a part, the pathological influence of uric acid is wholly hypothetical. It may be taken for granted that the circulation and toxic effects of the products of deranged and abnormal metabolism are common factors in disease; but to ascribe so much to one particular metabolite, without good evidence, and to employ a name which embodies an assumption, is but to delay more discriminating study in an important field of chemical pathology. Uric acid is probably not a poison in the proper sense at all, and but for its insolubility it might have been as little heard of in pathology as that other normal metabolite, the excretion of which is in comparable amount—creatinine.

Quite distinct, of course, from the present tendency to popularise the uric-acid 'diathesis' is the endeavour, already referred to, to extend the field of influence of uric acid into the causation of such disorders as epilepsy, asthma, bronchitis, Raynaud's disease, diabetes, and many others (Haig). This is not the place to discuss these views in detail, but the following remarks may be permitted.

Quite essential to the theory involved is a proof that in this or that condition there is at one time or another an abnormal retention of uric acid in the circulation and tissues. The evidence which is relied upon in proof of such retention is, broadly stated, as follows: Uric acid and urea are first assumed to be formed in the body in a definite relation one to the other, which varies but little from the ratio of 1 to 33. It follows that if, on a given day, the ratio of uric acid excreted to the urea excreted fall below this value, retention must have occurred; if it rise above this figure, urates previously stored in the body must have been brought to excretion. A period showing a low ratio means, therefore, a period of accumulation.

But if recent physiological teaching is correct, and uric acid has no common origin with urea, it is, to say the least, unlikely that the two substances appear in metabolism in a continuously balanced ratio. The following considerations with regard to the point are based entirely upon experiments, many times repeated. If upon a fixed diet of any sort a healthy individual is found to excrete, say, one gramme of uric acid for each thirty grammes of urea, it only requires that about a pint and a half of milk should be added to his intake to lower the ratio at once to 1 : 40; for milk, as already stated, scarcely affects the uric-acid output, while the quantity mentioned will produce ten grammes of urea. It is clear, therefore, that an alteration in the ratio may be no proof at all of retained urates. One other line of evidence for retention has been employed in the exposition of the theory. Certain granules, produced in the blood by admixture with ammonium chloride, are assumed to consist of urates, and these, when counted under the microscope, are said to be increased in conditions when, in the view under discussion, retention might be expected. It is sufficient to say that such granules may form in blood which, when examined in quantity, is found to yield no detectable amount of uric acid (Luff).

If the evidence for retention be rejected, close examination of the remaining arguments will show that they have lost their force, so far at least as

they aim at the ascription of symptoms to uric acid itself.

At the present time, then, we have no really satisfactory evidence to show that uric acid is a factor in disease, save in conditions where it or its salts are obviously deposited; that is to say, in gout and lithiasis. With the former of these this article is only indirectly concerned; there remain for consideration certain facts related to the ætiology and pathology of uric-acid gravel.

Whatever view may be held as to any deeper relationship between gout and gravel, there is no doubt that there are fundamental differences in the chemical side of their pathology; the final factors involved in producing the actual deposition of urates on the one side of the kidneys, and of uric acid on the other, are essentially diverse. As Sir William Roberts insisted, the conclusion is clear 'that gravel should be regarded as a primary vice of the urinary function; that it requires to be studied by and for itself; and that the urine is the proper and natural field for the investigation of its ætiology and therapeutics.'

Normal urine usually contains much more uric acid than it can hold in simple solution, and most specimens eventually deposit nearly the whole of this excretive in a crystalline form. But this never occurs within the urinary passages, and, as a rule, even after the urine is voided the deposition is long delayed. The urine of sufferers from gravel may, on the other hand, sparkle with crystals when it is passed, and in any case rapidly yields a crystalline sediment.

The factors concerned in the physiological maintenance of solution, and, on the other hand, in bringing about the normal slow separation, are complex. Roberts put our knowledge of certain of these factors upon a definite basis, and showed how variations in them may render urine unable to hold its uric acid even while warm and within the passages.

The percentage of uric acid is of influence. Although, as already urged, a tendency to gravel or stone does not depend upon increased production of uric acid, but may exist when the habitual output is below the normal and be quite absent when the excretion is abnormally high, it is yet clear that, other things being equal, and the tendency to precipitation present, there will be greater danger and, it may be, worse results when the urine contains excess than when the proportion is low. The existence of a proportionately large amount of uric acid in the excretion of children probably accounts for the relative frequency with which they suffer from lithiasis.

A second and prepotent factor in determining deposition is the degree of urinary acidity. An alkaline urine cannot, of course, deposit free uric acid, and there is always an increased tendency to separation with increase of acidity.

Yet another character in the urine which promotes separation is a deficiency in inorganic salts. The immunity from stone and gravel displayed by sailors has been attributed to the large quantity of salt which they take with their food (Plowright).

Lastly, there is a character of less importance, a deficiency, namely, in pigments. To this Roberts attributed the free sedimentation which often occurs in the pale urine of contracting kidneys.

These factors are undoubtedly important, and the first three and most influential can, fortunately,

be controlled with great benefit in the treatment of gravel. At the same time, it must be admitted that other urinary variations of a character at present unknown make themselves felt. Urine may deposit its uric acid very rapidly when analysis shows it to contain no excess of the excretive, and none of the aberrations just enumerated (Smith-Jerome).

Moreover, there seems to be no doubt that heredity plays a part in the ætiology of gravel, and what is transmitted can only be partly and superficially expressed in urinary characters such as have been discussed, which are, after all (with the possible exception of the acidity), very variable even in the habitual sufferer from gravel. With regard to the acidity, it appears to be the case that some people—and among them some prone to lithiasis—habitually pass a hyper-acid urine under conditions which offer no obvious cause for the phenomenon. It is conceivable that some anomaly in metabolism may lead to the habitual excretion of an acid product or products not yet recognised. This is, of course, entirely hypothetical; but it is perhaps useful to remember, in this and other connections, that we have, in cases of cystinuria (p. 359) and alkaptonuria (p. 48), such metabolic anomalies—'chemical malformations' as they have been called—which have been recognised because the abnormal products give such obvious indications of their presence. It is not impossible that there may be other anomalies of this kind not yet discovered.

Nature of Deposits.—The appearance of uric acid as it separates from the urine is too familiar to need detailed description. The crystals are, almost without exception, pigmented, and in the commonest case have the colour and, after sedimentation, present the appearance of cayenne pepper. Sometimes they are yellow or brown. Under the microscope their shape is found to be very various, departing always from the developed rhombic forms of the pure substance (by the suppressing of angles or faces, and by cohesion) and forming rectangular prisms, or falling into 'whetstone,' 'barrel,' or 'dumb-bell' forms. Sometimes they are aggregated, and occasionally the aggregates are of considerable size. Their departures from the typical forms are due to the effect of associated impurities, an albuminous or mucoid substance, and the colouring matters (Ord). The shape assumed largely depends upon the nature of the associated pigment (A. E. Garrod). Their pigmentation, insolubility in acids, and solubility in alkalis, are sufficient to characterise them.

In the kidney, concretions may be seen *post mortem* as glistening yellow-brown sand, as somewhat larger tuberculated masses, or, of course, as still larger calculi, ranging even to several ounces in weight. Stones which occur in the kidney-substance are always composed of nearly pure uric acid and are dense and hard; those which have been long in the pelvis may contain urates or phosphates, with uric acid as a basis; these are more friable. We are still without information as to why some patients with lithiasis pass gravel for years without calculus-formation, while others, less fortunate, tend to build up stones. The albuminous material which is always associated with a calculus undoubtedly affects the mode of aggregation; but there is nothing to explain these individual differences.

SYMPTOMS.—The symptoms enumerated by Murchison as indicating a condition of lithæmia comprise, in addition to the pathognomonic urinary deposits, an extensive group, from which we can select some or others which will fit any case of disturbed metabolism—flatulent eructations, constipation, disturbed appetite, furred tongue, excessive accumulation of mucus in the fauces, circulatory disturbances, headache, vertigo, and dimness of sight. To all these, and others, some more recent authorities (Da Costa and others), in drawing the picture of 'lithæmia,' have added and have laid stress upon inflammatory conditions of the external coats of the eye.

The condition of lithiasis, apart from calculus, can always be recognised by a sufficiently frequent examination of the urine. The separation of crystals in the freshly voided urine may not occur at all hours of the day, but is nearly always seen in the early morning urine; and in well-marked cases, not under treatment, is seldom absent at any time.

In some people this objective sign in the urine may exist for years without associated symptoms, but as a rule there are sooner or later some indications of renal irritation, and the condition should not be left without treatment.

In other cases symptoms may be more urgent. In children there are frequently colicky abdominal pains, usually occurring suddenly and located at the umbilicus or iliac regions. In severe cases there may be some nausea and vomiting, and perhaps slight hæmaturia. Strangury may be present in boys. In adults there may be a dull continuous pain in the loins, often attributed to lumbago, but disappearing with any treatment which keeps the urine free from deposit. The pain may radiate and be mistaken for other conditions. The disorder is often very chronic, and Levison has followed cases for twenty to thirty years without seeing true renal colic or other severe symptoms supervene. When gravel is associated with or gives place to calculus, there are the familiar signs of that affection; but as none of these are special to uric-acid stones, they need not be enumerated here.

TREATMENT.—In conditions of lithiasis it is clearly of importance to keep the production of uric acid as low as possible, and to this end the diet should be regulated. As was stated above, taking a dietary of milk, eggs, white bread and green vegetables results in an almost complete disappearance of that moiety of uric acid which arises from the food and largely reduces the excretion, a fact of real importance in treatment. But such a diet should not be prescribed empirically nor without due observation of its effects. Many patients are apt to take too little proteid when employing it, and such deficiency, if long continued, may lead to results more serious than can be attributed to uric-acid gravel. White meats can usually be added with safety, but even with this addition so unstimulating a diet does not suit all cases. The extractives of meat undoubtedly increase uric-acid production, but there is evidence that they may subserve physiological functions; for instance, in stimulating the secretion of gastric juice (Pawlow). But the proteid in the diet, while it should be sufficient, must never be in excess, and the flesh-food should not be in disproportion to the vegetable food taken; this, not because there is any essential difference between animal and vegetable proteids in their effect upon uric-acid production, but because

flesh-food increases and vegetable food diminishes the alkalinity of the urine. Luff looks upon the mineral constituents of vegetables as having a special power of keeping uric acid in solution, a power more marked than their chemical nature as revealed by analysis can be made to explain.

It has been satisfactorily proved by quantitative study that quinic acid, a constituent of many fruits, definitely diminishes the production of uric acid in the body, probably by interfering with its synthesis (Weiss). As a result of these experiments certain compounds of quinic acid have been introduced as drugs. These are 'Urosine' (quinic acid of lithium), given in doses of about ten grains, and 'Sidonal' (quinic acid of piperazine), the dose of which is about the same. These are of too recent introduction for any knowledge of their benefit to be available; but it is certain that the administration of quinic acid, especially when combined with a dietary introducing no uric-acid precursors, leads to a remarkably low excretion of urates.

With the end of preventing the separation of the acid from the urine, abundant water-drinking should be recommended, and the free use of table-salt is a desirable procedure. During any acute stage of the disorder the urine should be kept fully alkaline by the free administration of alkalis. Valuable, but perhaps not more valuable than the above means, is the use of various alkaline or saline mineral waters, the choice of which must depend upon the particular case under treatment. If drunk at their source the action of such waters is assisted by accessory hygienic factors.

There are various organic substances which form highly soluble compounds with uric acid. Used as drugs it would seem that they do not reach the urine in sufficient concentration to exert much influence as solvents. The benefit which is said to follow their use can, therefore, be only empirically explained. Prominent among these are piperazine and piperidine, the latter base being given as a tartrate in 10-grain doses (Tunncliffe and Rosenheim).

F. GOWLAND HOPKINS.

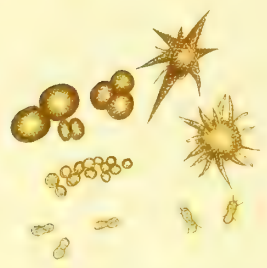
URINARY CALCULUS.—A calculus formed in any part of the urinary apparatus. See CONCRETIONS; and RENAL CALCULUS.

URINARY DEPOSITS.—Under healthy conditions the normal urine does not deposit a sediment, unless that flocculent suspension floating at various levels in the clear fluid and composed of thin mucus entangling a few epithelial cells be called a deposit. But even trivial deviations from the normal habit of body are prone to be indicated by the presence of a sediment in the urine, while in grave general disturbances and severe local derangements the composition of the urine is often materially altered, and may then contain substances which reveal themselves either immediately after it is passed, or after it has been allowed to stand for a while. With these sediments naturally deposited, i.e. without the intervention of reagents, it is proposed to deal here.

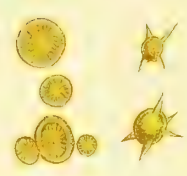
Method of examination.—Some 5 or 6 ounces of the urine to be examined are placed in a conical or cylindrical glass vessel, and after standing for some hours the supernatant fluid is decanted off. A sample of the sediment may be more easily and quickly obtained by the use of the centrifuge, and this instrument may become a necessity should the



Uric Acid



Acid Ammonium Urate



Acid Sodium Urate



Calcium Oxalate



Calcium Phosphate



Ammonio-magnesium



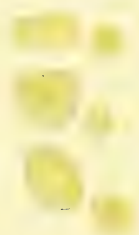
Phosphate



Cystine



Tyrosine



Leucine



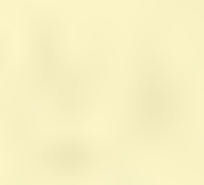
Phenyl-glucosazone



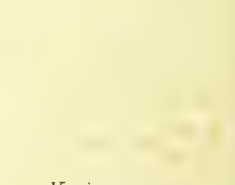
Cholesterine



Hippuric Acid



Pus cells before and after acetic acid



Various appearances of red blood cells



Vaginal

Urethral

*Vesical and renal-pelvis
Epithelium*

Tubal

Hyaline

waxy

granular

fatty

leucocyte and epithelial Casts

Cotton fibres

Linen fibres

Silk fibres

Wool fibres

Human hair

Varieties of Starch

Spermatozoa

*Yeast-cells and
mould-spores*

Mould forming mycelium

solid particles be scanty or of light specific weight. It is often advisable to add some preservative, the best of which are formalin and chloroform; but if the centrifuge be used this addition is unnecessary, unless the sediment be required for future demonstration.

After noting the naked-eye characters of the deposit, a drop or two should be pipetted on to a slide and submitted to microscopical examination. The examination of the sediment may be facilitated by the use of staining reagents, such as methylene-blue, fuchsin, methyl violet, iodo-potassic iodide, smic acid, Sudan iij, mucicarmine, muchæmatin, &c. These staining reagents may be run under the cover-glass or previously added to the sediment.

For microscopical examination low and medium powers are usually amply sufficient, though occasionally it may be needful to employ an immersion lens. When examining unstained samples, it is advisable not to use too much light, as some objects, such as hyaline casts and cylindroids, may be easily escape observation owing to their great transparency. As a rule, renal tube-casts are easily recognised, but cylindroids, aggregations of small crystals and granular debris, epithelial scales and minute fragments of wood, are from their somewhat cylindrical shape sometimes mistaken for renal casts. Some of the difficulties are removed by the use of staining reagents, and these should always be employed if doubts arise as to the exact nature and origin of cells.

The substances to be dealt with may be divided into three classes—unorganised, organised, and adenitious, and though most may be designated sediments or deposits, to some the terms 'suspensions' or 'scums' seem more appropriate.

Unorganised Deposits.—*Uric acid* (Lithic acid) is found in the free state, in urine which has, or has had, an acid reaction, as yellow or brownish-yellow crystals. Though the type-form is a rhombic prism, the shape of the crystals is extremely variable. From their microscopic appearance they have been compared to lozenges, whetstones, flat plates, cubes, and long spikes. Often the crystals are massed together in irregular agglomerations and in stellate or rosette-like clusters. Clusters of these crystals are frequently met with in cases of uric-acid gravel, and then resemble red sand or Cayenne pepper at the bottom of the vessel. In size also they exhibit considerable differences; the Cayenne deposit being composed of quite large grains, while on other occasions so small are they that a high power is required to make out their characters. Uric-acid crystals are recognised by their yellow colour and their shape, and the diagnosis may be confirmed by their micro-chemical reactions. They are soluble in caustic alkalis, and respond to the murexide test.

Urates are met with both in an amorphous and in a crystalline form, and are not infrequently associated with uric acid.

Amorphous urates consist of a mixture of sodium, potassium, and ammonium urates, the sodium urate usually predominating. They are deposited from urine with an acid reaction, and make their appearance as soon as the fluid cools. The sediment is coloured by urinary pigment (uroerythrin), and its hue varies from a pale fawn colour to pink or red, and very occasionally purple. The ruddy deposit is known as lateritious or brick-dust urates. Any doubts as to the nature of the deposit may be re-

solved by heating the fluid, when the turbidity disappears, or by applying the murexide test.

Urates also occur in a crystalline form as striated yellow spherules with and without projecting colourless spicules or spines, and also as small dumb-bells. Those in which the spicules are large and a distinctive feature (hedgehog) are found in urine with an acid reaction, and also *post mortem* in the bladder and kidneys of children. They are composed of *urate of sodium*. Dark yellow, almost opaque spherules, often showing a few small spicules, and consisting of *urate of ammonium*, are formed in urine which is alkaline from ammoniacal fermentation. Sometimes these crystals are quite small, and when paired assume a dumb-bell shape. *Urate of calcium* is occasionally met with in the urine, both as an amorphous white deposit and as acicular crystals, much resembling the urate of sodium in gouty deposits.

Xanthin, which very rarely occurs as a deposit, forms colourless rhombic plates. In composition it is very closely allied to uric acid, but may be distinguished therefrom by dissolving on heating, by the addition of ammonia, by its insolubility in acetic acid, and by not giving the murexide test.

Hippuric acid has occasionally been observed in the urine after eating certain fruits (cranberries, bilberries) and vegetables, and after the administration of benzoic acid, occurring as longish white rhomboid prisms; it is soluble in ammonia and insoluble in hydrochloric acid.

Oxalates.—Crystals of oxalate of lime are found in acid urine, especially after standing. Occasionally they may be found in quite fresh urine. They may be detected by the naked eye as white specks on the mucus-film, on contaminations, or on the sides of the urine-glass. Their presence is, however, usually discovered on microscopical examination, when they are seen as colourless or white octahedra, the so-called 'envelope crystals,' of variable size. Sometimes oxalate of lime assumes a dumb-bell or hour-glass shape, but these are usually accompanied by the common 'envelope' form. Oxalates are insoluble in acetic acid, but they dissolve in strong mineral acids without effervescence.

Carbonate of lime and sulphate of lime occasionally occur in human urine, the former as small spherules or as dumb-bells, the latter as colourless needles, or thin, elongated plates. The reaction of the urine in the first instance is alkaline, in the latter acid. Carbonates dissolve with effervescence on the addition of acids. Sulphates are insoluble in ammonia, and in acetic and sulphuric acids.

Phosphates occur in urine both in an amorphous and in a crystalline form. *Amorphous phosphates* form a white deposit in alkaline or neutral urine. They are a mixture of calcium and magnesium phosphates. The deposit is easily dissolved by acetic acid, and is undiminished or even increased by heat.

Crystalline Phosphates.—1. *Phosphate of lime*.—Neutral or stellar phosphate is found as a sediment and also as a scum on the urine. The single crystal is an elongated prism pointed at one end. The crystals may be discrete or arranged like stars or rosettes, the pointed end being directed towards the centre. They are easily soluble in acetic acid.

2. *Triple phosphate*.—Ammonium-magnesium phosphate occurs in alkaline urine, especially after ammoniacal fermentation. The most common form of the crystal is a triangular prism with bevelled

ends (coffin-lid); there are, however, numerous deviations from this type. Triple phosphate also crystallises in feathery flakes, occurring singly, in pairs, or crossed.

3. *Magnesium phosphate*.—This has been met with, but only very rarely, in strongly alkaline urine. The crystals are depicted as highly refractive elongated prisms with blunt-pointed ends.

Cystin.—*Cystic oxide* occurs usually in faintly acid urine which readily undergoes ammoniacal fermentation with production of sulphuretted hydrogen, due to the large amount of sulphur (about 26 per cent.) the substance contains. The crystals are colourless, hexagonal plates, and these are frequently imbricated or superimposed. They are insoluble in water, alcohol, and acetic acid, but readily dissolve in ammonia. See CHOLESTERIN, p. 295; CYSTINURIA, p. 359; LEUCINE, p. 864; TYROSINE, p. 1782.

Bilirubin and *hematoidin* are deposited in the urine, chiefly as fine needle-like crystals, singly or in tufts, also as amorphous granules and occasionally as rhombic plates. In colour they vary from yellowish-red to dark red. They have been found in inflammatory and malignant disease of the urinary apparatus, severe jaundice (icterus catarrhalis, gravis, neonatorum), cancer of the liver, and phosphorus-poisoning. They are soluble in acids and caustic alkalis and many other micro-chemical reagents, and with nitric acid give Gmelin's reaction.

Indigo has been found in urine undergoing ammoniacal fermentation; it occurs as rough, irregular masses of a dark blue colour, and as acicular crystals, scattered or in clusters. In the nascent condition it has the property of staining substances and structures such as uric-acid crystals, urates, cells, fungi, and also the urine, blue. Hence its presence is easily recognised. As blue indigo it is not a common deposit, though its immediate antecedent, indican, is frequently present in urine.

Fat occurs in urine in chyluria, after much fatty or oily food, in chronic Bright's disease either free or in casts and cells, in diabetes, in phosphorus-poisoning, and also as an accidental, and occasionally as an intentional, contamination. The highly refractive globules of varying size are easily recognised by their optical appearance, or by staining with osmic acid or Sudan iij, or by their ready solubility in ether.

Organised Deposits.—*Mucus* exists only in small quantity in normal urine and then appears as a cloudy deposit or as a suspension. It is without definite organisation, and is only recognisable by its delicate and irregular striation and by the foreign particles (dust, debris, cells, crystals) it entangles. Under certain morbid conditions it becomes inspissated and increased in quantity, as in cystitis, where it is ropy and copious. It may be seen as long longitudinally-striated ribands (cylindroids), having often much resemblance to hyaline casts, in the urine of nephritis, &c., and even in normal urine. Long bands of mucus are found in the urine of urethritis (urethral threads), and these may entangle epithelial cells, pus-corpuscles, and microbes, e.g. gonococci. Mucus is precipitated by acetic acid, and may be stained with mucicarmine or with mucæmætin.

Epithelial cells.—In normal urine a few epithelial cells, mostly squamous, may be found on microscopical examination. In diseased conditions of the urinary passages and of the kidneys, cells of various

kinds are not infrequent, and, though it might be anticipated that their shape would give evidence of the locality of origin and so aid in diagnosis, this is often far from being the case. In the case of an inflammatory or a cancerous process, the cells from the part affected are abnormal *ab initio* and liable to changes arising from degeneration, contact with morbid secretions, pressure, or osmosis. Taking these pathological conditions into account, as well as the absence of any distinctive criteria in the cells of transitional epithelium derived from different portions of the urinary tract, it is obvious that caution is required in coming to a conclusion as to the exact source of origin of cells found in the urine. Still, certain cells may be definitely recognised, such as squamous epithelium from the vagina, or from the anterior extremity of the urethra and prepuce; and columnar cells from the male urethra, the uterus, and the prostate; but while those from the vagina are common, the others are comparatively rare. Transitional epithelial cells may proceed from other parts of the urinary tract, but they present few localising features, though in all probability a multinuclear, polyhedral cell comes from the bladder. Bladder cells are not infrequently large and squamous. Renal epithelium is cubical or polyhedral, with comparatively large and well-defined nuclei. It is well to bear in mind that leucocytes, owing to imbibition, swell up to quite a large size; and if there be the slightest doubt about the nucleus it is advisable to stain the sediment, lest, with a too great regard for tradition, they be assigned to a renal origin and a desquamative nephritis.

Tumour-cells and tissue-fragments.—Cells from malignant or inflammatory growths affecting the bladder, ureters or kidneys, or adjacent structures, e.g. uterus and rectum, may be discovered in the urine, and it is sometimes possible to verify a diagnosis of cancer or other neoplasm by detecting keratinised epithelial cells, pigmented cells, cells with inclusions or cancer-bodies, portions of new growth, and fragments of organised tissue. It is rare, however, to make a diagnosis of cancer or other tumour from a microscopical examination of urinary sediment without some previous clinical indication.

Spermatozoa may often be observed in the urine. The presence of a few of them is devoid of special significance. Sometimes, however, glairy or whitish flakes are seen in urine, and these on examination are found to consist of mucus containing spermatozoa. Spermatozoa are easily recognised by their characteristic shape, which is fairly uniform, though occasionally a few individuals are abnormal both as to shape and size. Their average length is about 50 μ . They can be stained by anilin dyes, but this is scarcely necessary, as they are easily seen with high and even medium powers. It is not advisable to swear to the identity of single specimens if incomplete, e.g. if the tail have broken off short by the neck. They have no resemblance to *Trichomonas vaginalis* either in shape or size.

Blood (Erythrocytes; Red Corpuscles).—When there is admixture of blood with the urine, the colour of the fluid and the character of the sediment vary with the source. If from the kidney, the supernatant fluid is reddish or smoky, and the deposit is mostly of a dirty brown hue, the sediment containing casts, crystals, cells, granular debris, &c., as well as blood-corpuscles. If from the bladder

or ureter the urine is generally red, and the deposit may contain, among other things, actual blood-clot. Owing to the different density and reaction of different urines, the red corpuscles may or may not lose their typical bi-concave appearance, and either swell up so as to resemble circular colourless discs, or shrink and become crenated. They last longer in acid than in alkaline urine, in which they are quickly destroyed. In hæmoglobinuria the urine is of a dark brown hue, and deposits a more or less copious brownish sediment. This sediment consists chiefly of the blood-colouring matter in the condition of yellow granules, but may also contain casts, mostly granular. The microscopical diagnosis may be confirmed by the 'guaiacum and ozonic æther' test, by the brown-coloured precipitate formed on boiling with liquor potassæ, by the hæmin-crystal test, and by the spectroscope.

Leucocytes (White Corpuscles; Pus).—Leucocytes of fairly normal appearance are occasionally met with in normal urine. In the deposit of nephritis they occur both free and in casts, and sometimes are quite numerous. In suppurative inflammation of the urinary tract below the kidney a yellowish-white or drab sediment may be deposited, which, if the reaction be acid, is loose and flocculent, and if alkaline, as in some cases of cystitis, viscid and stringy. Pus in bulk, when treated with caustic alkali, becomes a slimy, tenacious, ropy mass. On microscopical examination the leucocyte is a granular spheroidal cell, the nucleus of which is of irregular shape and may be imperceptible. Like the erythrocyte, its size and appearance are variable, and for similar reasons (end- and exosmosis and reaction). By treating a sample with acetic acid the granules clear up, and the nuclei become more evident. The nucleus is single and irregular in shape, or multiple (1-4) and fragmented. Iodopottassic iodide stains pus-corpuscles dark brown (glycogen-reaction). In cases where it may be important to distinguish the nucleus of the leucocyte from that of the epithelial cell, staining with hæmatoxylin or with anilin pigments is advisable.

Bacteria (Bacteriuria).—Normal urine is not only clear but sterile. Under disease-conditions, however, it may not only contain bacteria, but be cloudy or turbid from their presence. In some cases the bacteria are found chiefly in the sediment, but in others they are disseminated as a suspension throughout the fluid. The conditions in which they occur are both local and general, and the organisms most frequently met with are *Gonococcus*, *Bacillus tuberculosus*, *Pyogenic cocci*, *B. coli communis*, and *B. typhosus*. The microbes of glanders, actinomycosis, and relapsing fever have also been found in urine. The foregoing list does not include organisms casually introduced from without, e.g. through the intermediary of a catheter, &c., for these are rather accidental contaminations than disease-germs proper.

For the morphological characters and special methods of detecting bacteria, see BACTERIA.

Entozoa.—In cases of *hydatid* of the kidney, vesicles, hooklets, and shreds of laminated membrane of echinococcus may be found in the urine.

The eggs and ciliated embryo of the *Distoma hæmatobium* are found in the bloody urine of cases of the endemic hæmaturia of North and South Africa. The ova, which are about 0.12 mm. long and 0.4 mm. broad, or the ruptured shell-cases, are easily recognised by their oval shape and the characteristic terminal or latero-terminal spine.

The contents appear granular, or resemble a collection of spherules. The ripe ova are easily hatched on a warm stage, so that the free-swimming embryo can be observed.

In cases of chyluria, the embryos of the *Filaria sanguinis hominis* are usually present in the milky urine. Manson recommends that the coagulum should be broken up with a glass rod, and after an hour or two the sediment examined after the manner of looking for casts (see p. 1798).

Adventitious Matter.—Normal urine is quite sterile, and if passed aseptically will keep indefinitely and remain perfectly clear, though crystals of uric acid may be deposited. Under ordinary conditions, however, the acidity of the urine may increase at first (acid fermentation), but soon, according to the temperature and its own constitution, it becomes neutral or amphoteric, and finally alkaline (alkaline fermentation). The exact cause—indeed, the existence—of the acid fermentation is disputed, but that of the ammoniacal is due to the action of microbes, *Micrococcus ureæ* and other bacteria, which secrete a ferment, urase, by which the urea is converted into ammonium carbonate. Besides these, other adventitious microbes belonging to hyphomycetes, blastomycetes, and schizomycetes may settle in the urine and develop more or less rapidly according as its composition and reaction and the temperature are suitable. Certain urines are specially prone to decompose, such as those containing blood or pus, the urine of cystitis and of diabetes, and that containing cystin.

While the fermentation is in process, and after it has subsided, the sediment of a sample of normal or of abnormal urine will mostly be found to contain organic and inorganic débris, crystals, casts, various cells in different stages of degeneration, and fungi (moulds, yeasts, bacteria). The nature of the sediment will materially depend on the substances originally entering into the constitution and composition of the urine.

In addition to the above-mentioned active and living bodies and their sedimentary effects, the presence of certain inert particles may be remarked, such as wool, cotton, linen, and silk fibres, hairs, feathers, wood, and dust. These are of constant occurrence as accidental contaminations, and they must be recognised by their special and peculiar characters. Among other extraneous substances met with in the urine may be mentioned oil-globules, starch-grains, fæces, sputum, and menstrual blood. See PLATE.

R. G. HEBB.

URINARY FEVER.—SYNON. : Urethral Fever; Catheter-Fever.

DEFINITION AND VARIETIES.—The above terms have been applied by different writers to various conditions which may arise after mechanical irritation of the urethra. The resulting symptoms vary, according to circumstances, in their nature, mode of onset, severity, and prognosis. The subject of urinary fever has been much discussed, and various views as to its nature have been advanced; and this is doubtless partly due to the fact that the ill-effects consequent on instrumentation of the urethra have not been sufficiently differentiated. These injurious consequences may be conveniently grouped as follows:—

1. Shock, slight or severe in nature, appearing immediately after the passage of the instrument, transient in duration, and ending in recovery.

2. An acute febrile attack, occurring some time after instrumentation (usually following the first act of micturition), transient in nature, but of some severity while it lasts, and ending in recovery. *Acute transient urethral fever; urinary sepsis.*

3. Acute recurrent attacks, similar in nature to that just mentioned. *Acute recurrent urethral fever; recurrent urinary sepsis.*

4. Acute septic infection, with definite pyæmic lesions and secondary centres of inflammation. This condition is precisely similar in all respects to septic infection arising in a wound of any other part of the body.

5. An insidious attack, without any well-defined symptoms, but marked by gradual loss of health and strength, often with an ultimately fatal termination. *Urinary cachexia.*

ÆTIOLOGY.—Any form of mechanical irritation of the urethra may lead to one of the conditions just mentioned. There is no doubt that unfavourable symptoms are most likely to occur if the disease rendering instrumentation necessary is situated in the membrano-prostatic portion of the urethra. Urinary fever, using the term in its broad signification, may occur alike in those who have been subjected to instrumentation before, and in those on whom a catheter has been passed for the first time. In the case of patients accustomed to catheterism, an attack of urinary fever may be determined by the use of an instrument of larger size than customary, because such an one is likely to cause some slight lesion of the mucous membrane unless used with care and by a skilled hand. In many cases of the more severe forms of urinary fever there is evidence of disease of the kidneys, but in others these organs are apparently healthy, or the disease is so slight, as shown by *post-mortem* examination, that it seems inadequate *per se* to account for death.

PATHOLOGY.—Setting aside cases of obvious septic infection, and those in which the patient is suffering from pronounced renal mischief, considerable difference of opinion exists as to the nature of urinary fever. Two separate theories have been advanced, and each of them has its advocates and opponents. In the opinion of the writer, both these theories are essential for the rational explanation of the conditions above-mentioned, each playing its own part, as will be shown.

1. *Neurotic theory.*—It is contended by the supporters of this view that urinary fever is directly traceable to reflex congestion and inflammation of the kidneys, resulting from the irritation of the sympathetic and cerebro-spinal nerves of the urethra. That the nervous excitability of the urethra is very great is undoubted, and is fully proved by numerous observations. It has already been stated that irritation of the membrano-prostatic urethra is more likely to be followed by untoward results than is that of the penile portion, and it is worthy of note that it is precisely in this part that the nervous supply is most abundant. That irritation of the nerves of the urethra may produce reflex effects upon the kidneys excites no surprise when it is remembered that the sympathetic nerves of the urethra are derived from the abdominal sympathetic, and are therefore in direct anatomical continuity with the nerves of the renal plexus. Irritation of the urethral nerves might thus induce extreme congestion of the kidneys, associated with suppression of urine, followed by hæmaturia.

If such a condition were excited in kidneys previously healthy it would be transient in nature, and would, under ordinary circumstances, speedily terminate in recovery; on the other hand, if the kidneys were already the seat of some inflammatory mischief, it is by no means improbable that the fresh irritation would seriously interfere with the functions of organs already damaged. See p. 823.

It is perfectly well known that an involuntary shudder often accompanies the end of the act of micturition in healthy men. The state of shock which occasionally follows the introduction of a catheter is no doubt largely explained by this physiological phenomenon. It shows that in some men a nervous system exists so sensitive as to be seriously impressed by even a normal act; much more so, then, by so rude a measure as the passage of a catheter. Irritation of the sympathetic nerves of the urethra might reflexly induce engorgement of the abdominal vessels, and thus induce the temporary faintness and shock not infrequently met with as the result of catheterism.

2. *Septic theory.*—Although some of the conditions arising from mechanical irritation of the urethra are, and others may be, due to nervous influences, yet in the majority of cases of acute febrile attacks there is little doubt that the ill-effects are dependent upon sepsis. Acute septic intoxication and septic infection may occur through a wound in the urethral mucous membrane or in the bladder, as they may do in the case of a wound of any other part. But while this is fully recognised and conceded, it is asserted by some that, irrespective of the undoubtedly septic and infected cases, so-called urinary fever (excluding shock) is dependent upon the absorption of toxic matters from the urine as it passes over the wound made in the urethra by instrumentation. It is highly probable that this view is correct in the majority of cases. Traumatism is a constant accompaniment of acute urinary fever, a few drops of blood following the withdrawal of the instrument. In support of this septic theory it must be borne in mind that the temperature remains normal until after the first act of micturition succeeding instrumentation: the urine, charged with septic material, is thus brought into contact with the wounded surface; the septic matter is absorbed, and the febrile symptoms follow. If the urine be in a healthy condition and antiseptic precautions be taken, urinary sepsis is much less likely to occur. Further, if a perineal opening be first made, and thus free drainage be afforded, urethral operations for stricture are not followed by urinary fever. And again, ill-consequences of this nature rarely follow internal urethrotomy, although they may have done so in the same case when an attempt has been made to treat the case by dilatation, thus pointing to the conclusion that free drainage is an important preventive measure.

In most cases of stricture and of enlargement of the prostate with residual urine there is an ample supply of septic material, which, under suitable circumstances, would be readily absorbed. In the recurrent form of urinary fever it is almost certain that each attack of fever occurs in response to the absorption of a fresh dose of poison, for it is difficult to suppose (unless we are prepared to admit that the mere passage of the urine, without absorption, can produce a febrile attack) that the nervous system is responsible for repeated attacks at in-

intervals of time during which no fresh mechanical irritant has been applied.

Some cases, perhaps, are of a complex nature, due in part to reflex congestion with inflammation of the kidneys, and in part to some form of septic absorption.

SYMPTOMS, PROGNOSIS, AND TREATMENT OF THE VARIOUS FORMS.—(1) *Urethral shock.*—In certain persons, especially those of a neurotic temperament, the passage of an instrument, even when used with the greatest dexterity and gentleness, produces symptoms of more or less severe shock. In the slightest, and fortunately by far the most frequent, cases, the patient, although not experiencing pain, becomes pale, breaks into a profuse cold sweat, feels dizzy, and not infrequently faints. Rarely the state of insensibility is accompanied by convulsive movements of the whole body. These symptoms are more likely to occur when an instrument is passed for the first time, or when the surgeon uses one of a larger size than that to which the patient has been accustomed. Recovery ensues in a few moments, and the patient experiences no further ill-effects. Fresh air, the recumbent position, and a dose of sal volatile is the only treatment necessary at the time; but the patient should be directed to keep quiet for the remainder of the day.

In very rare cases this condition of shock is very much more serious, and may terminate fatally within twenty-four hours. In such there is total suppression of urine; and *post mortem* the kidneys are found to be intensely congested, although they are often otherwise healthy; it is probable that the fatal termination is due to cessation of the excretion of urea. Treatment is of little avail. Free purging by croton oil or elaterin, hot stupes to the loins, warm diluent drinks, the hypodermic injection of pilocarpine, and the hot vapour-bath offer the best chance of success.

(2) *Acute transient urinary fever.* *Acute urinary sepsis.*—The symptoms characterising this condition come on soon after the first act of micturition following instrumentation. In most cases it will have been noticed that there was perhaps some slight resistance to the onward passage of the instrument—resistance, it may be, so slight that moderate pressure easily overcame it; on withdrawing the instrument a drop or two of blood was observed to follow it, indicating the infliction of a wound.

No symptoms occur immediately, but after micturition the patient is seized with shivering or perhaps a severe rigor, and experiences pain in the loins and back, often severe in nature. The temperature rises, and may reach 105°; the pulse is accelerated; headache and nausea are fairly constant; and vomiting and diarrhoea are occasional accompaniments of the condition. The urine is diminished in quantity and may be bloody. The cold stage is quickly succeeded by profuse sweating and a feeling of great heat. The temperature falls, the pains diminish and ultimately disappear, the urine loses its bloody tint (if such was present) and is secreted in larger quantity, and the patient quickly recovers and experiences no ill after-effects.

Such an attack usually lasts from twenty-four to thirty-six hours. No doubt it may attack alike those whose kidneys are known to be diseased and those in whom these organs are apparently healthy. In some cases the patient may remain febrile for a longer period than above stated—a matter for

anxiety, as it may indicate the supervention of acute nephritis. Subsequent instrumental attempts may induce other attacks, but by no means necessarily do so. That this form of urinary fever is of septic origin there can be no doubt, and for its prevention the surgeon should endeavour to avoid injury to the urethra, and should adopt antiseptic precautions. Those who believe in the neurotic origin of this form of fever advise the administration of quinine and opium for some days before any instrumentation is resorted to, but this treatment is of no practical value.

The treatment of the patient during the attack is conducted on ordinary principles. During the cold stage the patient should be wrapped in blankets, and hot bottles should be applied; hot drinks may be freely taken, but alcohol is to be avoided. The sweating stage hardly calls for treatment; it is in itself a sign of recovery. The bowels should be opened by croton oil placed on the back of the tongue, and hot fomentations may be applied to the loins if there be much pain. Complete rest for some days is advisable, and all irritation of the urethra must be avoided.

(3) *Recurrent urinary sepsis.*—In some cases recurrent febrile attacks similar to that just described may occur at intervals of two or three days, quite independently of any fresh mechanical irritation. During the interval between successive attacks the patient is apparently quite well. Such cases appear to be analogous to chronic septic intoxication as met with in wounds elsewhere. The treatment is conducted on the same lines as above indicated; the bowels should be kept open freely, and diuretics with diaphoretics are useful. In malarious subjects quinine may prove beneficial.

(4) *Acute septic infection with or without suppuration* may occur after instrumentation with wound of the urethra as in wounds elsewhere. Secondary centres of inflammation in the viscera and distant parts, suppuration of the joints, with repeated rigors, high temperature, and the usual symptoms attendant on infective processes, serve to distinguish the case from one of acute urinary sepsis.

(5) *Persistent urinary fever.*—In some instances, especially in cases of enlarged prostate with residual urine necessitating the daily employment of the catheter, the patient falls into a general state of ill-health, insidious in its onset and progress, and not infrequently ending in death.

In such cases it will be found that there is old-standing disease of the kidneys. The irritation of the urethra appears to light up fresh mischief—mischief in many cases added to by the concomitant absorption of septic material. In cases of enlarged prostate with residual urine, there is unfortunately in many cases an ample source of septicity in the decomposing urine present in the bladder.

The symptoms may date from the first passing of the catheter. There is very rarely any decided rigor, and the symptoms, unless distinctly septicaemic in nature, may be at first indefinite. The patient complains of general malaise, loss of appetite, and perhaps a feeling of chilliness. Fever may be entirely absent, and is never very marked. The urine is not diminished in quantity as a rule, nor is there any noticeable alteration in the excretion of urea. Perhaps the symptoms of this condition are best explained by the statement often made by the patient himself—‘He is not the same man that he

was before using the catheter,' and yet he is unable to state definitely from what he suffers.

In the later stages of a fatal case the condition of languor so commonly met with becomes more marked. The patient becomes drowsy and apathetic, and may wander a little at night. Sometimes there may be temporary improvement, only to be followed by a relapse. Death may occur in from six weeks to as many months. Fortunately many cases after some few weeks gradually recover.

In all cases in which the surgeon deems it necessary to resort to the habitual use of the catheter, it is most important that every care should be taken to avoid urinary fever. The patient should be confined to his room, which should be kept at an equable temperature; the bowels should be regulated, and the action of the skin and kidneys promoted; the diet should be light, nutritious, and non-stimulating. If the amount of residual urine is considerable, it should on no account be all drawn off at once, but the bladder must be gradually emptied in the course of a few days. Attention to these fundamental points will do much to ward off serious effects. Should they, however, in spite of all precautions, manifest themselves, the patient must be kept quiet in an equable temperature, and instrumentation must be carried out carefully and as seldom as is consistent with the exigencies of the case. The action of the bowels, skin, and kidneys must be regulated. If rest be broken, opium or some other form of narcotic must be given; but the use of opium requires prudence and supervision in view of the renal imperfection. Stimulants are frequently necessary. C. STONHAM.

URINE, Incontinence of.—The involuntary discharge of urine, the patient being either unable to retain it or unaware of its escape. See MIC-TURITION, Disorders of; and SPINAL CORD, Diseases of.

URINE, Morbid Conditions of.—As a result of morbid processes the normal constituents of the urine may be either increased or diminished in amount, or abnormal substances may be present.

Quantity.—The quantity of urine secreted may be either increased or diminished; if increased, the increase may be permanent or temporary. The conditions affecting the quantity of urine secreted are: (1) the amount of fluid ingested; (2) the quantity of fluid lost by other channels, more particularly the skin, the lungs, and the bowel; and (3) the quantity of fluid actually present in the tissues. The last factor does not vary greatly in conditions of health, but in renal diseases and other diseases accompanied by dropsy, great variations in the amount of the fluid in the tissues are met with, influencing the quantity of urine secreted. The proximate cause of the quantity of urine secreted is the rate of flow of the blood through the glomerular vessels; all conditions tending to diminish this are accompanied by a scanty flow of urine. There is further experimental evidence to show that great diminution in the amount of renal tissue (as, for instance, the removal of large portions of one kidney and the whole of the other) causes a great and permanent increase in the quantity of urine excreted.

A temporary increase in the quantity of urine excreted is seen in a number of conditions, some of nervous origin, as, for instance, the large excretion

which may occur not only in nervousness and fear, but also in a number of functional diseases of the nervous system, such as after epileptic seizures or asthma. In such diseases as diabetes insipidus and diabetes mellitus and in a large number of organic diseases of the kidney, more especially in granular kidney, in the contracted white kidney, in the amyloid kidney, and in cystic degeneration, the quantity of urine excreted is permanently increased. The latter are diseases diminishing the quantity of available kidney-substance.

In other renal diseases, as, for instance, ordinary chronic Bright's disease, the flow of urine is often diminished; but in these cases it is possible that the real cause of this is not always an incapacity on the part of the kidney to excrete, but rather the simultaneous presence of dropsy. Subsidence of dropsy is invariably accompanied by an increase in the excretion of urinary water without any other prominent change occurring in its composition. The quantity of urine is diminished in all conditions which impair the efficiency of the circulation through the organ, as in passive congestion and active congestion, and also in a certain number of destructive diseases, more especially acute and certain forms of chronic Bright's disease.

The diminution in the quantity may be so great as to amount to suppression, and two varieties of suppression are usually recognised, the obstructive and the non-obstructive. In *obstructive suppression* there is some mechanical impediment to the exit of urine along the ureter, produced by the impaction of a calculus or the pressure of a tumour. In some cases mere inflammatory thickening of the mucous membrane of the ureter is sufficient to cause obstruction. In true obstructive suppression little or no urine is pent up behind the obstruction, the secretion of the kidney having undergone complete arrest, and the term 'suppression' should be limited to this. In other cases the obstruction may lead to the distension of the ureter and pelvis, and so produce a hydro- or pyo-nephrosis.

Suppression may occur without the presence of any obstruction, and at least two forms of *non-obstructive suppression* may be recognised: (1) One, that in which where the circulation through the organ is arrested, either as a result of embolism or thrombosis of the renal vessels, large or small, or else owing to the presence of extreme congestion brought about by inflammation, set up by cantharides, scarlet fever, and other poisons, &c. (2) Non-obstructive suppression may also be seen without any serious lesion of the renal blood-vessels, and then it occurs apparently as a result of reflex influences, originating in some other part of the body. Such complete suppression may result from the passage of a catheter or from an exploratory nephrotomy, or even from the presence of acute disease in other parts, e.g. cystitis, peritonitis, &c. In most of these instances the kidney-tissues are not thoroughly healthy, but cases have been described where complete and fatal non-obstructive suppression has occurred as a result of injury or disease in some other part of the body, and yet the kidneys have not presented any signs of gross disease. See URINE, SUPPRESSION OF.

Colour.—The normal colour of the urine is dependent mainly on the presence of urochrome; small quantities of urobilin, uro-erythrin, and hæmatoporphyrin are also present. The urine also contains a number of chromogens, especially those

giving rise to indigo and skatol-pigments, in the form of indoxyl- and skatoxyl-sulphates of potassium. Indoxyl is also present in combination with glycuronic acid, and the normal urine may yield as much as twenty milligrammes of indigo. Several of these pigments and chromogens of normal urine have probably an intestinal origin, and this is certainly so in the case of indican. The source of the urochrome is by no means certain, and it has been thought to owe its origin to the action of microbes on bile-pigments in the alimentary canal; but several cases of biliary fistula with complete obstruction of the bile duct have been described where, notwithstanding the discharge of the whole of the bile to the surface of the body, the urine has maintained its normal yellow colour. The colour of the urine is necessarily influenced by the quantity secreted, and thus in all diseases where there is a copious flow of urine the colour is usually pale. This is notably the case in diabetes and in renal diseases. In many renal diseases the colour, however, is pale, notwithstanding that the increase in the amount of the secretion is not necessarily very great. This is especially seen in some forms of chronic Bright's disease and in the granular kidney. Dark-coloured urines are secreted in conditions where the quantity of urine is diminished as a result of profuse perspiration, and also in a number of diseased conditions where the quantity is diminished, but in disease the dark colour is often dependent on the presence of abnormal pigments.

Urobilin normally occurs only in traces in the urine, but in a number of diseases it may be excreted in sufficient amount to cause the urine to become a dark mahogany colour. It was at one time supposed that this urobilin differed slightly from normal urobilin. This view is not now held; the two bodies are considered identical, the anomalous colour of the urine being dependent on the excretion of the urobilin in such large amounts. The presence of large quantities of urobilin in the urine causes the urine to assume a colour somewhat resembling that seen when bile is present. The two conditions can be distinguished by spectroscopical examination and by the absence of the reaction of bile. Urobilinuria is seen after free internal hæmorrhage, in pernicious anæmia, in cirrhosis and other diseases of the liver, in some febrile diseases, and in porphyria.

Hæmatoporphyrin.—This body is present in traces in normal urine, but occasionally it is excreted in sufficient amount to cause the urine to be of a deep red colour. Its presence in large amounts is especially associated with the administration of sulphonal, but only in exceptional instances. Smaller quantities of the same pigment are excreted in Addison's disease, in various destructive diseases of the liver and also in rheumatic fever. See HÆMATOPORPHYRINURIA, p. 615.

Hæmaturia.—In hæmaturia blood-corpuscles are present in the urine. See HÆMATURIA, p. 621.

Hæmoglobinuria.—In this condition the blood-pigment only is excreted. See HÆMOGLOBINURIA.

Choluria.—See JAUNDICE.

Melanuria.—Melanin is a sulphur-containing pigment and, in rare instances, is excreted in the urine; in such conditions the urine is often of a *café au lait* colour. On the addition of an oxidising agent, such as nitric acid, the urine becomes black owing to the conversion of the chromogen into melanin. Melanuria is seen as a rare complication

in several wasting diseases, but more especially in patients suffering from melanotic sarcoma. It is, however, not an invariable accompaniment of this condition, and a high degree of melanuria may exist without the presence of melanotic sarcoma. The only instance of melanuria seen by the writer was one of melanotic sarcoma of the liver secondary to a melanotic sarcoma of the eyeball. The addition of ferric chloride in cases of melanuria yields a greyish-brown or black precipitate soluble in excess of ferric chloride. Bromine-water gives a yellow precipitate which gradually turns black.

Pyrocatechin.—Pyrocatechin is said to occur in minute traces in the normal urine, but it is present in considerable amount, together with hydroquinone, in cases of carboluria. Both these substances exist in the urine as aromatic sulphates. The urine when passed is of a normal colour but darkens on exposure to air, and if large quantities are present may become quite black.

Alkaptonuria.—See p. 48.

Chyluria.—See pp. 300 and 543.

A number of drugs impart more or less characteristic colours to the urine. Santonin causes acid urine to become yellow or greenish in colour; the addition of a caustic alkali causes the urine to become a cherry-red or purple, the colour disappearing on the addition of an acid. The administration of rhubarb or senna may cause the urine to become red, on the addition of an alkali, from the presence of chrysophanic acid.

Reaction.—The acidity of normal urine is dependent on the presence of acid phosphates and, according to some authorities, to traces of free organic acids, such as lactic and hippuric (see ACIDITY, p. 14). The degree of acidity is not constant, being most marked in the morning urine and least marked after meals, when it may become not only neutral but even alkaline. The mixed urine of the twenty-four hours, however, is acid, and it is unusual even for the urine voided after a meal to be alkaline, although that secreted by the kidney may be; in other words admixture takes place in the bladder with previously secreted acid urine. The acidity of the urine is diminished by a vegetable diet and by the administration of alkalis or their compounds with vegetable acids, such as the citrates. The acidity is increased by a meat-diet, and is still more marked in cases of diabetes when diabetic coma is present. In this latter condition, however, the acidity is dependent on the presence of abnormal acids, especially β -oxybutyric acid (see DIABETES, p. 831). The acidity is liable to be increased if the quantity of urine is diminished, as in febrile conditions. In diseases of the stomach, especially in dilatation of the stomach, the acidity of the urine may be diminished, and it may even be alkaline.

Alkalinity of the urine may be due to the presence of either fixed or volatile alkali (see ALKALINITY, p. 44). The latter is a serious condition, dependent on the decomposition of the urine in some part of the urinary tract as the result of microbial infection. The infection usually reaches the urine owing to the introduction of a dirty catheter, but it is possible for the infecting agent to reach the urine in other ways, as, for instance, by the rupture of an abscess into some part of the urinary tract. It is also possible that the infecting agent may reach the bladder by passing up the urethra, or may reach the urine by excretion

through the kidney, as there are a great number of conditions in which micro-organisms may be excreted by the kidney.

Alkalinity of the urine owing to the presence of fixed alkali is not usually a condition of great moment. It may lead, however, to the excretion of a milky urine owing to the precipitation of phosphates.

Specific Gravity.—The specific gravity in health varies between 1015 and 1025. It is rare for it to be below this in health, although exceptional cases have been recorded with the lower limit of 1010, and others where it has been as high as 1038. Observations on the specific gravity of the urine are of little importance unless made with a specimen of the whole twenty-four hours' urine, as the specific gravity of individual samples of urine is influenced by a great number of conditions, e.g. drinking, sweating, nervousness, &c. A low specific gravity, if constant, is often a sign of disease, such as diabetes insipidus, and of a number of conditions in which the quantity of kidney-tissue is greatly diminished, as for instance cystic disease, hydronephrosis, granular kidney, amyloid disease, and some varieties of chronic Bright's disease. In a number of functional diseases, such as epilepsy, asthma and hysteria, large quantities of dilute urine may be secreted from time to time, and more especially after the subsidence of paroxysmal seizures. The specific gravity is raised by the presence of large quantities of urea and of salts in the urine, and especially by the presence of sugar. It is usual to suspect glycosuria where the specific gravity is over 1035. The real characteristic of glycosuria is rather that a pale, dilute-looking urine has a high specific gravity. Sugar may, however, be present with a specific gravity as low as 1010. The presence of albumin increases the specific gravity slightly, but in renal disease the specific gravity is usually low owing to the excretion of a urine poor in saline ingredients, and often deficient in urea and other extractives.

Urea.—From 20 to 40 grammes of urea are excreted daily in health, the average being approximately 30 grammes, corresponding to '5 grammes of urea per kilogram of body weight. The quantity excreted is greatly influenced by diet and also by the quantity of water drunk. Copious water-drinking increases notably, at any rate temporarily, the quantity of urea excreted. If very large quantities of nitrogenous foods are consumed, the whole of the nitrogen ingested is not excreted as urea owing to the production of diarrhoea whereby the excessive ingesta are got rid of. Quantitative observations on the amount of urea excreted in disease are of comparatively little value unless accurate determinations of the nitrogen in the ingesta and in the feces are made simultaneously. In diabetes mellitus large quantities of urea are excreted owing partly to the increased appetite, and also owing to the proteid tissue-waste that occurs in this disease. In diabetes insipidus the quantity is also slightly increased. In many diseases there may be no absolute increase, but only a relative increase; in other words urea-excretion may remain approximately at the normal amount notwithstanding a great diminution in the ingesta; this is notably the case in many febrile diseases.

In those diseases of the liver in which the liver-pancreas is diminished in amount, the excretion of urea is diminished, e.g. hepatic cirrhosis, primary and secondary atrophy, phosphorus-poisoning. In

acute yellow atrophy urea may almost disappear from the urine, the quantity excreted being so small. In all wasting diseases, such as carcinoma, and especially in carcinoma of the stomach, the amount of urea excreted may be very small.

In renal diseases great variations exist; in many chronic destructive diseases of the kidney, quantities of urea equal to the normal or but little below them may be excreted notwithstanding the great destruction of renal tissue, e.g. hydronephrosis, cystic kidneys, granular contracted kidney, and some forms of chronic Bright's disease. The percentage excreted, however, is nearly always below the normal, but there is not such a great diminution in the total amount owing to the increased excretion of urine seen in many of these diseases. In acute nephritis and in some forms of chronic nephritis the quantity excreted may be small. In some instances a sudden diminution in the quantity of urea excreted in chronic renal disease may point to the imminence of uræmia, but this complication may occur at a time when the patient is passing quantities of urea, little, if at all, below the normal. In many cases of renal disease the diminution in urea excreted is more apparent than real, and is dependent not so much on any incapacity on the part of the kidney to excrete, as upon the associated vomiting, diarrhoea, and albuminuria all diminishing the amount of proteid available for conversion into urea. The persistent excretion of a low percentage of urea is very suggestive of the existence of one of the more chronic and insidious forms of renal disease.

The name *azoturia* has been given to a condition in which the excretion of urea is excessive in proportion to the weight of the body. This may occur in persons who are apparently perfectly healthy, or it may be associated with gastric and intestinal derangement, together with various nervous symptoms.

Uric Acid.—Uric acid is present in the normal urine in quantities varying from half to one gramme, creatinin being more abundant but being of less importance owing to its free solubility. Uric acid derives most of its clinical importance from its insolubility, being soluble to the extent of 1 in 15,000 of cold water and at the temperature of the body of 1 in 7,000 to 8,000 parts of water. The normal urine may contain as much as two grammes of uric acid dissolved in the 1,500 to 2,000 c.c. of urine. Different authorities have explained the increased solubility of uric acid in the urine in different ways. According to some the uric acid is present in the urine in the form of a soluble quadriurate. According to others the uric acid exists in the urine in the form of an acid urate of sodium. The main reason for thinking that the uric acid in the urine was in the form of a quadriurate rested on the fact that, if the urine was allowed to stand without undergoing decomposition, a considerable proportion of the uric acid was deposited in the form of free uric acid. Further, the analyses of several urinary deposits yielded, according to Bence Jones, Scherer, and Roberts, results showing that the uric acid was present in larger amount than that necessary to form a biurate with all the bases present. Moreover the action of distilled water on these so-called quadriurates led to the formation of free uric acid and biurate of sodium in sensibly equal proportions, and this has been looked upon by some as a characteristic reaction of the quadriurates. The alterna-

tive view is to regard what have been looked upon as quadriurates as mixtures of uric acid and acid urate of sodium, and further that the deposition of free uric acid from urine kept from decomposition is dependent on an interaction between the acid urate of sodium and the acid phosphate of sodium. The liberation of free uric acid from an uratic deposit by the addition of distilled water may be dependent on the existence of uric acid in an amorphous form and its subsequent crystallisation as a result of the action of the water. Recent analyses of natural uratic sediments have shown that a certain quantity of phosphoric acid is present, and hence for these reasons the existence of quadriurates cannot be looked upon as certain. The deposition of uric acid from the urine is influenced mainly by the acidity of the urine, the quantity of pigment, and of salts present. The uric acid excreted in the urine has a double origin, part being derived from alloxur-bases in the food and part from the metabolism of the tissues. Milk, eggs, rice, white bread, potatoes, salad and cauliflower contain practically no alloxur-bases, and with a diet constructed from these substances the uric-acid secretion will be greatly diminished. It is not possible, inasmuch as part of the uric acid arises from the metabolism of the tissues, entirely to eliminate this substance from the urine.

The uric-acid excretion is diminished in many chronic diseases, in chlorosis and during the paroxysms of gout. It is increased in pernicious anæmia, in splenic leucocythæmia, in certain febrile diseases, in ague, and in some forms of diabetes mellitus. Uric acid normally is excreted in the form of an urate, and the normal urine does not deposit free uric acid when it cools. Further the quantity of urates is usually such that they are easily soluble in the urine when cool, but if the quantity of urate be diminished, or that of the urates much increased, it may readily come about that the urates are deposited in the well-known pink amorphous uratic sediment when the fluid cools. This deposition is more especially liable to take place in febrile diseases and in a large number of affections of the stomach, liver and digestive organs. Urates are also deposited freely in the dense urines secreted in cases of diseases of the heart and lungs. In some instances in addition to the amorphous urates of sodium, calcium, potassium and ammonium, crystalline urates of sodium and ammonium are excreted in the form of 'dumb-bell' and 'hedgehog' crystals or of rosettes, which may cause considerable irritation of the urinary passages, especially in children.

Oxalates.—Approximately 20 milligrammes of oxalic acid are excreted in the urine daily in the form of calcium oxalate, which is kept in solution owing to the acidity produced by the acid phosphate of sodium. It is probable that the oxalates do not arise from metabolism of the tissues of the body, but entirely from the oxalic acid contained in various articles of diet. The deposition of oxalate of lime in the urine may depend not so much on the quantity present as on other factors—e.g. the acidity of the urine may be such that it cannot hold it in solution. The mere deposition of oxalates in apparent abundance must not be taken as indicating a so-called oxaluria or increased excretion; this can only be determined by quantitative observations. Some cases of oxaluria, where the quantity excreted is large, are dependent on fermenta-

tion of the gastric contents associated more especially with a diminution in the secretion of the hydrochloric acid of the gastric juice.

The form of crystallisation of oxalate of lime is greatly influenced by the presence of colloid material, so that the presence of pus, blood, mucus or albumin in the urine may determine the formation of large crystals of oxalate of lime and so produce the nucleus of a calculus. See OXALIC ACID IN URINE, p. 1136.

Phosphates.—The phosphates present in the urine are the phosphates of potassium, sodium, calcium, and magnesium, the acidity of the urine being dependent on the acid phosphate of sodium. Approximately two-thirds of the phosphoric acid is in combination with the bases potassium and sodium in the form of soluble phosphates, the remaining third being in combination with calcium and magnesium forming the so-called earthy phosphates. From two to six grammes of phosphoric acid are excreted daily in the urine. Sodium and potassium phosphates are freely soluble in the urine, whether the salts formed be acid, neutral, or basic. The earthy phosphates are only soluble in acid urine and become deposited in neutral and alkaline urines, and to a small extent in faintly acid urines. Urines alkaline from the presence of ammonia contain ammonio-magnesium phosphate, which is readily deposited in the form of so-called triple-phosphate crystals. Occasionally small quantities of ammonio-magnesium phosphate may be deposited from urines still faintly acid. The quantity of phosphates excreted is largely dependent on the amount ingested in the food, but the quantity is to a certain extent influenced by the metabolic processes in the body, and in the later stages of febrile diseases it undergoes an increase. According to some authorities the phosphatic excretion is increased in certain forms of neurasthenia, and a condition of so-called phosphatic diabetes has been described where large quantities of urine are passed containing an excess of phosphates but no sugar. The quantity of phosphates can only be determined by quantitative determination, and the deposition of the earthy phosphates from the urine is no indication of the presence of an excessive excretion of phosphoric acid. The earthy phosphates tend to be deposited in all conditions in which the acidity of the urine is diminished, and hence it is common for the urine secreted after meals to be cloudy or even very turbid from the deposition of amorphous phosphates. Occasionally stellar phosphate or di-calcium phosphate occurs in the urine in some quantity. This has been described in diabetes and other maladies, especially wasting diseases where there is grave disturbance of nutrition. The deposition of triple phosphate from urines alkaline from decomposition is of serious import if it occurs in the bladder or urinary passages. The deposition of earthy phosphates due to a diminution in the acidity of the urine is usually readily ameliorated by measures directed to improving the digestion or by the administration of acids. Faintly acid or neutral or alkaline urines when heated often show a cloud owing to the precipitation of earthy phosphates, and if precautions are not taken to acidify the urine prior to the boiling this phosphatic cloud may be mistaken for the presence of albumin. It is probable that the precipitation of phosphates is dependent on the boiling of the urine causing a decomposition so that two molecules of di-calcic phosphate and one molecule of mono-calcic

phosphate lead to the formation of one molecule of triple and one molecule of di-calcic phosphate, and the tri-calcic phosphate being insoluble is precipitated.

Sulphates.—Sulphur is excreted in the urine in three forms. A small quantity is in combination with amides, forming bodies of the taurin-group. Cystin, a substance which is sometimes excreted in large quantity, is a body of similar constitution, taurin being amido-ethyl-sulphonic acid and cystin amido-sulpho-lactic acid (*see* CYSTINURIA, p. 359). The great bulk of the sulphur is excreted in the form of sulphates combined with the bases of sodium, potassium, and magnesium. A portion, however, of the sulphuric acid is in combination not only with one of these bases but also with aromatic bodies such as phenol, indol, skatol, cresol, forming the so-called aromatic sulphates. The sulphuric acid present both in the inorganic and aromatic sulphates is derived in part from the food and in part from the metabolism of the tissue-proteids. The aromatic sulphates are formed by the decomposition not only of proteids of the tissues, but also of the proteid matter present in the intestine. Normally, twelve to twenty times as much sulphuric acid is excreted in the form of inorganic sulphates as is excreted in the form of aromatic sulphates. The excretion of aromatic sulphates is greatly increased in all conditions of intestinal putrefaction and retention of intestinal contents, e.g. intestinal obstruction, intestinal ulceration, and peritonitis. They are also increased in such conditions as pulmonary gangrene and in pyæmia and deep-seated collections of pus. The increase is not only relative but also absolute. The ingestion of aromatic radicles, like phenol and other anilin-bodies, is followed by their excretion in the form of aromatic sulphates, and consequently there is a great diminution in the amount of ordinary sulphates in the urine in carbolic-acid poisoning.

Chlorides.—From ten to fifteen grammes of sodium chloride are excreted daily, but the quantity depends very largely on the amount in the food. In many febrile diseases the quantity of chlorides excreted is diminished during the height of the pyrexia, and this is more especially the case in pneumonia where, during the height of the disease, the chlorides may almost disappear from the urine. This diminished excretion is not, however, restricted to pneumonia, but may occur to a less extent in the other diseases where the body-temperature is high, as, for instance, tonsillitis.

Proteids.—The proteids usually met with in the urine in renal diseases are serum-albumen and serum-globulin and various forms of albumoses. The albuminuria of renal disease is probably always dependent on morbid changes in the epithelial structures of the kidney, both tubular and glomerular, and it is probable that the albuminuria is dependent on the action of various toxic substances damaging this epithelium, and so allowing the proteids of the blood to pass through. The origin of the albumosuria is more obscure (p. 35), inasmuch as the blood normally does not contain these bodies in quantity, if at all. The amount of proteid lost in the urine *per diem* is very variable, but may amount to as much as forty or more grammes. The greatest loss is seen in cases where the quantity of urine is considerably increased at a time when it contains a moderate percentage of albumen. Acute Bright's disease may lead to the excretion of a urine

loaded with albumin, but owing to the diminution in quantity of urine the total amount lost may not be as great as in cases of chronic renal disease where the percentage amount is less, but owing to the greatly increased quantity of urine excreted the total amount is greater. Amyloid disease and the contracted form of Bright's disease are the two conditions in which the greatest loss of albumin occurs. In the granular kidney and the cystic kidney the amounts are usually small, and in acute Bright's disease and in chronic Bright's disease, though the percentage amount is high, the total amount is moderate. *See* ALBUMINURIA; and ALBUMOSURIA.

Casts—hyaline, granular, epithelial, or consisting of blood or pus—are generally moulds or casts formed by the exudation of proteid matter into the tubules of the kidney. In other instances the epithelial elements of the tubules are incorporated in the casts forming the well-known epithelial casts, the cells of which may show signs of fatty degeneration. *See* URINARY DEPOSITS; and CASTS.

Ehrlich's Reaction is described on p. 1772.

JOHN ROSE BRADFORD.

URINE, Retention of.—A morbid condition in which there is difficulty or inability to expel the urine from the bladder. *See* MICTURITION.

URINE, Suppression of.—SYNON.—Fr. *Suppression de l'Urine*; *Anurie*; *Ischurie*; Ger. *Harnverhaltung*.—This term signifies an arrested or greatly diminished secretion of urine, which may result from some obstruction to the outflow, or from some interference with renal excretion, acting primarily or reflexly upon the kidney.

1. Obstructive Suppression.—When one kidney has already been rendered useless by disorganisation following tuberculosis, or as a result of renal calculus or other cause of hydronephrosis, the impaction of a calculus in the ureter of the opposite kidney may produce obstructive suppression. When both kidneys are physiologically active, the excretion may be arrested by any pressure simultaneously affecting both ureters, as with uterine cancer, which may either exert direct pressure or may occlude the ureters by dragging on the walls of the bladder, and thus narrowing the orifices of the ureters. Under these conditions a small quantity of urine may be passed, characterised by low specific gravity, by the small proportion of urea and of other normal constituents, and occasionally by the presence of a trace of albumen. Apart from the suppression of urine, the symptoms differ with the cause. When due to calculus, suppression is usually preceded by one or more attacks of renal colic and hæmaturia; suppression may remain the chief symptom for seven or eight days, and then death may occur suddenly, or for two or three days before death there may be muscular twitchings and weakness, contraction of pupils, drowsiness, and fall of temperature. Convulsions, dropsy, and ammoniacal or urinous odour of the breath are commonly absent.

2. Non-obstructive Suppression.—This may occur in the course of acute nephritis, with sub-acute nephritis in tubal or interstitial forms of Bright's disease, or with severe congestive conditions which may result from toxic agencies, from cholera or from other diseases. It may also ensue after irritation or injury by a catheter, or after

severe abdominal injuries or operations, especially when followed by acute peritonitis. Extensive burns are sometimes associated with suppression. In many of these conditions suppression must be regarded as a symptom produced reflexly through the nervous system, but in diphtheria and in scarlet fever it may result from irritation of the renal epithelium by toxins, comparable to the irritation of mineral acids, turpentine, and cantharides. The symptoms of non-obstructive suppression usually follow a rapid course; acute uræmic symptoms, severe vomiting, headache, convulsions, and coma cause death within two or three days.

When suppression occurs as a symptom of hysteria, the clinical aspect is far less grave; the arrest of the excretion of urine is as complete as in the other varieties, but it is accompanied by persistent vomiting of fluid which contains an appreciable quantity of urea. This condition ordinarily terminates with polyuria. These attacks may be repeated, and they may alternate with other hysterical manifestations. Hysterical suppression does not entail risk of uræmia.

TREATMENT.—This necessarily depends upon the cause of the suppression. The *obstructive* forms are occasionally amenable to surgical measures, which should be undertaken early, before the onset of muscular twitchings. In the *non-obstructive* varieties, especially when due to congestive or inflammatory affections of the kidneys, hot baths and other sudorific treatment are usually beneficial, and hydragogue purgatives may be employed. It is important to avoid the use of all diuretic remedies. *Hysterical* suppression is best treated with antispasmodics; no attempt should be made to check the vomiting.

NESTOR TIRARD.

UROSTEALITH.—See CONCRETIONS, p. 329.

URTICARIA.—SYNON.: *Cnidosis*; Nettlerash; Fr. *Urticaire*; Ger. *Nesselausschlag*.

DEFINITION.—Urticaria is an eruption accompanied by sensations of stinging, itching, and burning, like those produced by the sting of a nettle, and is characterised by the formation of wheals.

SYMPTOMS.—Urticaria is a very common disease, and is remarkable for its very variable and fugitive character, and for the great variety of circumstances under which it is developed. In all cases, however, the presence of wheals or some equivalent eruption is pathognomonic of the disease. The wheal consists of a circumscribed swelling of the skin, attended by active congestion of the vascular layer and an exudation of serum into the immediate neighbourhood of the vessels. This exudation does not usually extend into the epidermic structures. The degree of swelling is very variable, and depends on the amount of exudation. The occasional, almost sudden, disappearance of these curious formations is explained by the close proximity of the serous exudation to the absorbent vessels; for when the fluid finds its way into the epidermis, the process of resorption is much slower, and traces of the inflammation are often left behind for several days. Usually the central part of a fully developed wheal is pale, compared with the circumference. This is said to be due to the increased pressure of the exudation at that point, which is often so great as to empty the capillaries of the skin; it may, however, be caused by a

spasmodic contraction of the muscular coats of the vessels. At all events, vaso-motor disturbance is always present in the formation of a wheal.

The size, form, and general appearance of the wheals vary greatly. Sometimes they are no larger than a split pea, while at other times they may occupy a considerable surface, and cause much swelling of the skin. In typical examples they are round or oval; not infrequently they take the form of streaks or irregularly shaped patches. Sometimes the only eruption consists of a diffuse erythematous bright red blush. But whatever be its form, it is very evanescent, and liable to appear and disappear almost suddenly, leaving little or no trace behind. In all cases the subjective phenomena are nearly the same, and consist in excessive itching, tingling, stinging, and burning sensations. Sometimes the itching preponderates, at other times, perhaps, the stinging and burning sensations are the most marked, for, like the eruption itself, they are liable to constant change. These subjective sensations are by no means always proportional to the amount of visible eruption, and often remain long after this has disappeared. The rash occasionally appears with a sudden outburst all over the body, while at other times it is developed more slowly, and appears successively on different parts. It is roughly symmetrical, though the wheals have no definite arrangement. Sometimes the mucous membrane of the mouth, tongue, and larynx is involved. The sufferer invariably rubs and scratches the skin. This greatly aggravates the symptoms, and brings out fresh wheals wherever the finger-nails are applied, and, in severe cases, small excoriations and little spots of coagulated blood may be seen scattered about the skin as the result of scratching. An ordinary attack of urticaria may last from a few hours to several days, and in chronic forms of the disease one attack follows so closely on another as to give the impression that they are continuous; this, however, is not the case, as there are always remissions and exacerbations, and generally short intervals of complete freedom.

ÆTIOLOGY.—Urticaria is often only a symptomatic rash, and differs also from most other eruptions in the fact that it is a frequent attendant upon other diseases of the skin. The circumstances under which it is commonly met with may be divided conveniently into five groups: (1) It is extremely apt to complicate other irritable affections of the skin, such as scabies, phthiriasis, prurigo, and eczema. This is especially the case in children. The production of nettlerash under these circumstances is due to reflex action set up by scratching. (2) The bites and stings of poisonous insects, and the hairs of stinging plants, such as the common nettle, will produce in some people pretty severe local attacks of urticaria; a bee-sting will sometimes produce general urticaria, with much swelling of the face and limbs. (3) Nettlerash produced by irritation of some part of the mucous tract is not uncommon, and belongs also to the group of reflex nervous actions. We often meet with examples of this kind dependent on uterine irritation from pregnancy and other causes; also in children who suffer from worms. (4) Certain kinds of food are apt to produce nettlerash; among these may be specially mentioned shell-fish, mushrooms, acid wines, and many kinds of fruit; but in these cases much depends on the idiosyncrasy of the individual.

To this category belong the red rashes which are occasionally produced by certain drugs. Among the best known of these are copaiba, capsicum, turpentine, cubebs, and quinine (*see* DRUG-ERUPTIONS). (5) The most common form of urticaria is met with in people in whom it is impossible to trace a definite exciting cause, or in whom the exciting causes are trivial and various. In these cases the disease is distinctly and wholly of nervous origin, and occurs in people whose nervous system has been overtaxed, being often associated with such nervous symptoms as neuralgia. This form of the disease is always recurrent. It may come on with great regularity at a certain time of the day, and even replace neuralgia. Urticaria is occasionally rheumatic.

VARIETIES OF URTICARIA.—In addition to the ordinary form there are three principal varieties of urticaria: (1) Acute febrile urticaria; (2) Urticaria papulosa; (3) Urticaria pigmentosa.

1. *Acute febrile urticaria* is an idiopathic affection but rarely met with. It is ushered in with febrile symptoms, especially headache and sickness; the pulse and temperature may both be high, and the tongue furred. The characteristic feature is the suddenness of the attack, and the general outburst of red rash, which may cover the whole of the trunk, face, and limbs, and produce much swelling of the skin. The mucous membrane is also liable to be affected. This is especially the case about the fauces and throat, which become suddenly swollen, so as even to threaten suffocation. The disease may be distinguished from scarlet fever by the suddenness of the attack, the swelling of the skin, and the subjective sensations.

2. *Urticaria papulosa* is a disease which is met with chiefly in children, and is commonly known as lichen urticatus; it is very obstinate, usually lasting for several years, but with intervals of subsidence. The itching and irritation are very great, especially at night; this is always aggravated by the scratching of the patient. The eruption consists of wheals mixed with permanent pruriginous papules, which are probably the result of urticarial exudation into the cuticular structures. It is often mistaken for scabies, but does not especially affect those parts of the body which are liable to the attacks of the latter disease.

3. *Urticaria pigmentosa* is a peculiar and rare form of persistent urticaria, associated with buff-coloured pigmentation of the skin. The eruption appears as persistent nodules, and red measly-looking patches, mixed with yellowish pigment-spots, resembling pityriasis versicolor. The peculiarity of the colour, and the persistence of these spots, constitute the chief characteristics of this rare variety of urticaria. Usually, after the disease has lasted for some time, the neurotic symptoms subside, leaving simply raised patches of yellowish skin, either with or without nodules; but the more active symptoms may be reproduced by rubbing and scratching.

TREATMENT.—As has already been stated, urticaria, unlike most other skin-diseases, is a symptomatic rash rather than a definite disease. That it is very difficult to deal with is proved by the vast number of different remedies recommended by writers on this subject. The first point in any given case is to ascertain whether there is any definite local cause of irritation giving rise to the nettlerash; and with this view the skin should be

carefully examined for bug-bites and pediculi, which are very common exciting causes among the poorer classes. It must not be forgotten that the dye or dressing of the underclothes is sometimes a local irritant to the skin. If we fail to find any external cause of irritation, our attention should next be directed to the alimentary canal. Worms in children are not an infrequent cause of urticaria, and injudicious feeding may be also mentioned as occasionally giving rise to it; therefore, in treating urticaria of this kind, a careful and well-regulated diet is in all cases indicated. With regard to medicines, those are most efficacious that promote the process of digestion; one of the most generally useful is a mixture containing carbonate of magnesium and bicarbonate of sodium, with some aromatic or bitter infusion, taken before each meal.

Subacute urticaria of the ordinary kind produced by poisoning from shell-fish or fungi or some other poisonous food is best relieved by a simple emetic and a quickly acting saline purge, so as to remove the offending matter from the stomach and intestinal canal.

A large proportion of cases of chronic urticaria are distinctly of neurotic origin, and are often associated with other disturbances of the nervous system. Of all remedies for this class of cases, the most generally useful is complete rest both of mind and body. Of medicines, quinine and arsenic may be mentioned as often beneficial, especially when the attacks are of a periodic character. Complete change of air sometimes succeeds when all other remedies fail. The malady is, however, always very obstinate.

ROBERT LIVEING.

USSAT, in Ariège, France.—Thermal saline and sulphurous waters. *See* MINERAL WATERS.

UTERUS, Diseases of.—**SYNON.**: Fr. *Maladies de l'Utérus*; Ger. *Krankheiten der Gebärmutter*.

The diseases of the uterus will be considered in the following sections:—

Methods of Physical Examination, p. 1810.

General Ætiology and Pathology, p. 1813.

Inflammatory and Congestive Diseases, p. 1813.

Displacements, p. 1816.

Carcinoma, p. 1818.

Other Tumours, p. 1820.

Methods of Physical Examination.—

1. *Digital Examination.*—Of all methods of examining the uterine organs, the most important is by the finger alone, nor is the necessary *tactus eruditus* by any means easy to acquire. In this country the lateral position is generally adopted, and, except under special circumstances, is preferable as involving less exposure than the dorsal. The patient should lie on her left side, with her hips as near the edge of the bed as possible. The semi-prone position is the most convenient, the patient lying more or less on her face, her left arm being placed behind her back, her knees being flexed, the upper one more so than the lower. A good position much facilitates a complete examination of the pelvic cavity, and attention to these details is never superfluous. The index finger of the right hand is now carefully introduced, at first in the axis of the vaginal outlet, and then in that of the pelvic brim. The unimpregnated uterus is suspended, as it were, at the top of the vaginal canal, into which the cervix projects. The latter is the part of the uterus

which the finger first reaches. As the normal direction of the uterus corresponds with the axis of the upper part of the pelvis, or, roughly speaking, with a line extending from the umbilicus to the coccyx, the cervix, in a healthy state, projects into the vagina, and points backwards towards the sacrum. Its shape varies in women who have had children, and in the unmarried or nulliparous. In the latter it is conical or nipple-shaped, and the opening of the os uteri is felt at the apex of the cone, as a circular aperture about the size of a pea. The anterior and posterior boundaries of the os uteri are known as the lips of the cervix, and they are very liable to alterations in size, becoming congested or enlarged under various morbid states, one often to a greater extent than the other. In women who have borne children the shape of the cervix is altered, and it becomes shorter and less regularly conical. The os is also changed from a circular opening into a transverse fissure, which is often more or less nodular and irregular at its edges, from lacerations of its tissues during labour; and is sometimes sufficiently open to admit the tip of the finger. When healthy, the mucous membrane covering the cervix is smooth and velvety to the touch; and through the speculum it is seen to be of a uniform rose-pink colour. Under various morbid conditions it becomes rough, granular, stripped of its epithelium, and covered with hypertrophied papillæ, and these alterations are of much importance from a diagnostic point of view. Having ascertained the conditions of the cervix, paying particular attention to its size, shape, density, and sensibility, and to the shape of the os uteri, we may next proceed to examine the body of the uterus, passing the finger for this purpose past the cervix into the vaginal *cul-de-sac* behind, in front, and on either side of the uterus. In this way we feel whether the uterus is of normal size, or hypertrophied, as it often is; whether it is painful on pressure, or not; whether the uterus is freely movable by the finger, as it ought to be; or whether it is fixed and immovable in any part of its contour, as is often the case from inflammatory adhesion in its vicinity. Then again, in the same examination, we ascertain if any swelling exists in any part of the vaginal *cul-de-sac*, in front, behind, or at either side; and if so we try to determine its form, density, mobility, sensibility, and whether it is attached to the uterus, or is independent of it—all points of importance in arriving at an accurate diagnosis.

2. *Palpation*.—In this part of the examination we may often gain much assistance by combining *abdominal palpation* with vaginal examination. This method of bi-manual examination is always of great utility, and is sometimes indispensable for accurate diagnosis, and it is not so generally practised as it ought to be. It may be used to some extent while the patient is still lying on her side, the left hand being passed over her right hip. But to practise it thoroughly we must make the patient turn over on to her back, and then by pressing down the abdominal parietes with the left hand, acting in concert with the examining finger, we may thoroughly explore the pelvic cavity, and ascertain much more completely the form and relations of any tumour within it, than by vaginal examination alone. In some cases valuable information can be obtained by a rectal examination, especially when there is a swelling or tumour in Douglas's pouch, or attached to the posterior part

of the uterus, which may often be more accurately examined in this way than *per vaginam*. Simple abdominal palpation is often necessary in investigating the nature of any tumour supposed to be uterine. This is best carried out by laying the patient on her back, with her knees elevated, so as to relax the abdominal parietes. Percussion may often be advantageously combined with palpation. By using one or two fingers of the left hand as a pleximeter, and percussing with the right, we get a dull or tympanitic sound. If the latter is marked where there is much abdominal distension, we know that it indicates bowel distended with gas, and that there is probably no tumour. If there be dulness we can limit its area, and thus verify the results of palpation by mapping out any abdominal swelling met with. By these means also we discover the existence of fluid, either free in the abdomen, as in ascites, or contained in an ovarian cyst, the presence of fluctuation being often very readily determined.

3. *Use of the Speculum*.—We now proceed to consider the means at our disposal for examining the lower segment of the uterus with the eye; and from the re-invention of the speculum by Recamier, in the early part of the present century, we may date the commencement of the accurate study of uterine diseases. Numerous varieties of specula have been used. One often employed is Cusco's bivalve speculum (fig. 1). This has the advan-

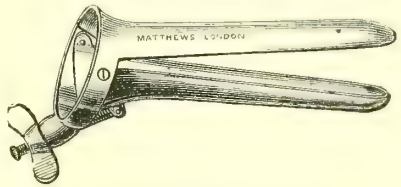


FIG. 1.—Cusco's bivalve speculum.

tage of being easy to introduce, and of being adapted for either a capacious or a narrow vagina. It exposes the cervix well. The objections to it are its expense, and the fact that the metal is apt to be affected by various applications made to the cervix. Personally the writer prefers the tubular (Fergusson's) speculum (fig. 2), made of glass and

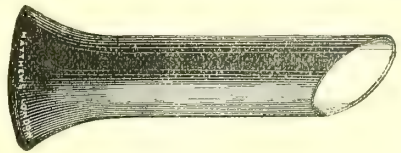


FIG. 2.—Fergusson's speculum.

covered with caoutchouc; and that which is bevelled at the end is the easiest to introduce, and the best for exposing the cervix. For cleanliness and brilliancy of illumination nothing can equal it. It is unaffected by any remedy used for local application, and has the advantage, which is wanting in all other specula, of embracing and steadying the cervix. In certain cases requiring local medication this is of great value. The objection to this form of speculum is its fragility, and the necessity of having instruments of various sizes, adapted to vaginæ of different dimensions. Practitioners, therefore, at a distance from surgical-instrument makers, will do well to provide themselves with the

more expensive instrument. Another form of speculum much used of late years, and in some cases superior to all others, is the 'duck-bill' speculum (fig. 3). This acts on the principle of drawing



FIG. 3.—The duck-bill speculum.

aside the perinæum and posterior vaginal wall, so as to allow air to distend the canal, and thus expose the cervix. For certain operations it is invaluable, and is most valuable even in ordinary examinations if the practitioner be skilled in its use. Considerable practice is required, however, to employ it satisfactorily, and it has the further drawback of necessitating the assistance of a second person.

The passing of a speculum, without pain, is an operation requiring some little practice to perform skilfully. In using a tubular glass speculum we must first choose one corresponding to the size of the vagina. This is a point of some importance; for the attempt to pass a large speculum into a small vagina causes much suffering; and if, on the other hand, too small an instrument be used, the cervix is not properly exposed. The patient should lie in the ordinary lateral position. The speculum should then be taken in the hollow of the right hand, its bevelled extremity resting on the under-surface of the index finger, the point of which should project a little over its edge, so as to guide it through the ostium vaginae. The point of the finger having been inserted into the vagina, the mouth of the speculum, the centre of which is grasped gently by the other fingers of the right hand, is held by the left hand, and the instrument, guided along the under-surface of the right index finger, is gently insinuated into the vagina, pressing back the perinæum as it enters. When it is fully introduced, it is turned gently round until the cervix is well exposed.

By the speculum we can speedily recognise any changes in the cervix and os uteri; we can see if the mucous membrane covering the former be pale or congested, smooth or abraded, or perhaps covered with granulations which bleed on being touched. The character and amount of discharge should be noted, and it may be wiped away with cotton wool held in the speculum-forceps. If the discharge come in great measure from the interior of the cervix and body of the womb, it is glairy, transparent, and very tenacious, and it may often be seen oozing out of the os uteri like white of egg. This is a certain sign of some morbid state of the mucous lining of the uterus. If the discharge come from the surface of the cervix, it is yellow and purulent in appearance. We can also recognise any abnormal growth that exists about the cervix, as, for instance, small gelatinous polypi which have evaded the sense of touch.

4. *Examination with the Sound.*—In certain cases in which more accurate information regarding the state of the uterus is required, other accessory means of examination are used. One of these is

the uterine sound (fig. 4), by means of which we can explore the interior of the uterus, measure its length, ascertain its direction, &c. The instrument is a thin rod of flexible metal, which can be bent into any desired shape, as it is sometimes necessary to adapt it to the altered curve of the uterine cavity. Its length is divided into inches by means of small notches in the metal, and at two and a half inches from its point is a small knob, indicating the normal length of the healthy uterus. In introducing the sound, the index finger of the right hand is passed into the vagina until its tip rests on the opening of the os uteri, which can be felt as a dimple or depression at the extremity of the cervix. The handle of the sound being lightly held in the left hand, its point, previously rendered aseptic and oiled, is guided along the palmar surface of the index finger of the right hand, until it enters the os uteri. It is a good plan to pass the sound through the vagina with its concavity looking towards the perinæum, and, after it has entered the os, to turn it gently round, so that its further progress may be in the ordinary direction of the uterine cavity. It is now gently pushed on, no force whatever being used, until its point is arrested by the fundus uteri. The tip of the right index finger is now placed at the os, and withdrawn in contact with the sound, so that the exact length to which it has entered may be ascertained. Considerable practice is required to pass the sound easily and without pain. Gentleness is necessary above all, and the sound should be coaxed to pass, being withdrawn if any resistance is met with, and never pushed on by force. Sometimes the sound will not pass in the ordinary direction, and then an endeavour must be made to adapt it to the curve of the uterine cavity, by bending it, or by passing it with its concavity backwards, as in the cases of retroflexion. The kind of information to be derived from the use of the sound will be best appreciated when treating of the separate diseases of the uterus.



FIG. 4.

5. *Dilatation of the Cervix.*—Another mode of examination, sometimes of much use, is the dilatation of the cervix by sponge or laminaria tents, or by Hegar's dilators, so as to admit of the introduction of a finger, and complete exploration of the uterine cavity. This is of immense service in cases of profuse menorrhagia, when the existence of an intra-uterine polypus, or portion of retained placenta, is suspected. The tent is a mass of compressed sponge, or a cylinder of *Laminaria digitata*, sufficiently small to enter the cervix, where it swells by the imbibition of moisture, and in doing so expands the surrounding tissues. These contrivances are properly now rarely used, on account of the difficulty of keeping them aseptic. Hegar's dilators are a series of graduated uterine bougies, made of vulcanite or metal, one of which is inserted into the uterus for about two minutes, beginning with No. 1, and gradually progressing to No. 23 or 24. By this means comparatively rapid and very effective

dilatation can be produced. Careful antiseptic precautions are essential, the dilators having been thoroughly boiled, and the operation must be performed under an anæsthetic. In the hands of an expert this is the best method at our disposal. Dilatation of the cervix is an operation that should not be undertaken without due consideration, as it is occasionally followed by considerable irritation.

General Ætiology and Pathology.—Much as is the attention which has been paid to uterine disease of late years, the opinions of the profession are as yet far from being decided on many elementary facts connected with it. It is beyond doubt that in this class of disease there are a series of symptoms common to all cases alike, such as pain in the lower part of the abdomen and back, inability to walk, leucorrhœal discharge, and disordered menstruation. If, however, modern writers on gynecology are consulted, it will be at once seen how various and irreconcilable are the explanations given of these symptoms. Thus some have taught that in inflammation and congestion, either of the cervix alone, or of the body or lining membrane of the uterus, we have the key to uterine pathology, and that all other changes detected in the uterus, such as displacements or flexions, are merely secondary results of the primary affection. On the other hand, many influential gynecologists have referred all uterine disease to mechanical causes: these considered displacements to be the primary cause of nearly every morbid state of the uterus, inflammation and congestion being merely secondary results; and naturally limited their treatment to an endeavour to replace and support the uterus in its normal position. The want of sufficient pathological study of morbid states of the uterus accounts for these varying opinions, which are much to be regretted; for, while one set of theorists apply themselves to an endeavour to relieve the inflammatory symptoms which they find, and which undoubtedly generally exist, by leeches, rest, and suitable local applications, they are apt to undervalue and neglect the mechanical means by which the displaced organ may be supported and steadied, on which their opponents too exclusively rely, but the real value of which it is impossible to call in question. Hence it follows that these partial and one-sided views lead to neglect of really important measures in one direction or the other, and to an amount of uncertainty in the mind of the profession which materially impedes a due recognition of the true importance of uterine disease. The fact, no doubt, is that neither of these opposing views is entirely right, but that there is a large measure of truth in both. Of the importance of inflammatory conditions no one, who impartially studies the clinical history of cases coming under his care, can entertain any reasonable doubt. The large, tender, and congested uterus, with its thickened and hypertrophied walls, the inflamed and granular mucous membrane covering the cervix, and pouring out abundance of morbid secretion, are conditions too obvious to be overlooked, and which are very frequently indeed associated with displacements, resulting from alterations in portions of the hypertrophied and overweighted organ. In some of these cases treatment directed to the original inflammatory condition may, of itself, suffice to effect a cure; in others this fails, unless attention be at the same time paid to the secondary displacements. On the other hand, it is equally impossible to ignore the

occasional remarkable influence of a simple displacement in producing disease. Who, for example, that has witnessed the long chain of distressing symptoms following a traumatic displacement, such as retroflexion from a fall, and the instantaneous relief sometimes following the introduction of a suitable pessary, can doubt this? In fact, all these conditions act and re-act on each other, and too excessive attention to one set of symptoms, based on theoretical dogmas, is as fallacious in uterine as in all other forms of disease.

The causes of uterine disease are very numerous. Among the most common may be mentioned errors in the mode of life consequent on the habits of modern society, such as tight-lacing, want of proper exercise, heated rooms, imprudence during menstruation, and the like. By far the most prolific source of uterine disease is to be found in the changes in the uterus consequent on parturition. Many accidental circumstances are apt to check and arrest the involution of the hypertrophied muscle-fibres which normally occurs after delivery. Hence the uterus remains large, congested, heavy, tender, and in the condition known as sub-involution, its cavity, as measured by the uterine sound, being elongated. In such cases the symptoms of uterine disease creep on insidiously after abortion or child-bearing, and, in a large proportion of cases, it will be possible to trace its origin to this source.

i. Inflammatory and Congestive Diseases.—Under this heading we may consider together those morbid states of the uterus which are variously described under such terms as *acute and chronic metritis*, *chronic parenchymatous metritis*, *areolar hyperplasia*, *acute and chronic endometritis*, *cervical endometritis*, *chronic uterine catarrh*, *granular degeneration of the cervix*, *ulceration of the cervix*, *congestion of the uterus*, and others.

This course involves the disadvantage of describing together diseases which, while they are very generally associated, and have much that is common in their symptomatology and treatment, may often, on the other hand, occur separately, and require important modifications in their management, according to the particular parts of the uterus affected. It is impossible, however, in so short an article, to discuss their individual peculiarities, as would naturally be done in a systematic treatise.

Acute inflammatory affections of the uterus, whether of its body or of its lining membrane, are of comparatively rare occurrence when unconnected with the puerperal state; and if a contrary opinion is expressed in many of our gynecological works, it is probably because various other inflammatory diseases, especially localised inflammations of the peritoneum and cellular tissue near the uterus, have been confounded with inflammations of the uterus itself. No practical harm will result, therefore, if we limit ourselves to the consideration of the more *chronic* conditions which are of such common occurrence, and produce such important consequences. One of the most common is undoubtedly congestion of the uterus, associated with enlargement of its vessels, and very often leading secondarily to more important and lasting disease, such as inflammation of its lining membrane, and the condition described as *areolar hyperplasia* or *chronic parenchymatous metritis*.

ÆTIOLOGY AND ANATOMICAL CHARACTERS.—The causes of such congestions are very numerous,

and indeed they occur normally in connection with every menstrual period, and may readily be perpetuated. By far the most important, however, is some interference with the proper involution of the uterus after delivery or abortion, to which a large proportion of such disease may be traced. If such congestion continue to be repeated, whatever be its cause, it very often leads to inflammation of the mucous membrane lining the cervix or body of the uterus; and then the diseases known as *corporeal* and *cervical endometritis* are established, which are of much importance. In these the largely developed glandular structures of the mucous membrane are the parts chiefly involved. On microscopical examination they are found altered in character, dilated at their mouths, and pouring out abundantly the transparent glairy mucus which is so characteristic of these affections. The villi of the cervix, both those within the canal and on its exterior, become altered, stripped of their epithelium, and eventually hypertrophied. These enlarged and abraded papillæ on the surface of the cervix, when seen through the speculum, form the characteristic red, strawberry-like abrasions round the os, which, under the name of ulceration, have formed so fruitful a subject of controversy in uterine disease. The detection of this condition—which is in no sense of the word an ulceration, since the epithelium is the only structure destroyed—is of much importance from a diagnostic point of view, but chiefly as leading to a knowledge of the more deep-seated changes which have produced it as a secondary result, which are themselves beyond the sphere of observation, but which are truly at the root of the evil. Hence the granular and abraded state of the cervix must be looked upon as a mere indication of disease elsewhere, not as being in itself a primary disease. Moreover, it is to be noted that many apparent abrasions of the cervix are really due to laceration of its tissue, and eversion of its altered lining membrane, a condition which can only be satisfactorily made out when the duck-bill speculum is used.

In more advanced stages of these inflammations of the mucous membrane deeper-seated alterations occur. The glands become obliterated or atrophied, and sometimes undergo cystic degeneration; and the whole mucous membrane may become adherent, stripped of its epithelium, covered with granulations, or finally converted into a layer of connective tissue covered with polymorphous cells (Klebs). In no long time, moreover, other morbid states of the uterus are developed. The organ becomes enlarged and tender to touch, and very often there is more or less forward or backward displacement. The cervix especially is apt to be hypertrophied, the os patulous, much leucorrhœal discharge is present, and all the distressing chain of symptoms accompanying confirmed uterine disease is established. Pathologically, this enlarged and tender state of the body and cervix of the uterus is, by most recent writers, believed to depend on excessive growth of the connective tissue, associated with vascular hyperæmia and hyperæsthesia of the nerves. It should be remembered, however, that it is identical with the condition commonly described as *chronic metritis*, the essentially inflammatory origin of which has long been an axiom in gynecology.

SYMPTOMS.—The symptoms accompanying these morbid states of the uterus are, in a great measure, those which are common to a large number of

uterine complaints. Pain in the lower part of the abdomen and back, increased by exercise of any kind; pain in defecation or micturition, and, in married women, on sexual intercourse; profuse glairy, tenacious, or purulent discharge, in old-standing cases very abundant; disordered menstruation, either scanty or irregular, or more often profuse, and frequently very painful; and eventually, if the true character of the disease be not recognised, a long and distressing catalogue of general symptoms, such as dyspepsia, hysteria, sickness or vomiting, headache, and others too protean in their character to be described, are among those which are most commonly observed.

The conditions met with on physical examination vary with the duration and extent of the disease, and the tissues of the uterus chiefly implicated. In the simpler cases the uterus is merely somewhat heavy and enlarged, and tender to the touch. When there is cervical or corporeal endometritis to any extent, the cervix is somewhat puffy and enlarged, and the external os patulous, so that the sound passes easily; and, in the same way, a dilated state of the cervical canal and internal os is recognised. Very generally also the surface of the cervix is rough, granular, and greatly abraded, bleeding on being touched, while strings of the characteristic gelatinous discharge are seen to exude from the os, and the cervix may be extensively fissured. Lastly, on bi-manual examination, in the more chronic and confirmed cases, the whole uterus will be found to be distinctly enlarged, probably somewhat elongated when measured by the sound, and very commonly the subject of some of the forms of displacement to be presently described.

PROGNOSIS.—The prognosis of these diseases must, of course, depend on their extent and duration. In their earlier stages they are readily susceptible of improvement and cure. In old-standing cases, which have lasted for years, and produced all the local and general results above described, the treatment is surrounded with difficulties, and the prospect is far from encouraging. It is of such states that Scanzoni speaks when he says, 'We do not remember a single case in which we have cured an abundant uterine leucorrhœa of several years' standing'—a dictum which was doubtless true with reference to the methods of treatment generally employed, but which fortunately cannot be endorsed by those who have employed more radical means of cure applied directly to the seat of the disease.

TREATMENT.—The treatment resolves itself into *general* and *local*. With regard to the former, the indications are to do all in our power to improve the nutrition and general health by ordinary means of treatment, such as attention to diet, fresh air, and the administration of appropriate remedies, of which such drugs as quinine, small doses of arsenic or strychnine, various ferruginous preparations, and bromide of potassium, either alone or in combination with other remedies, especially when there is much nervous irritability, are among the most generally useful. In old-standing cases resort to some of the Continental watering-places is occasionally of much service. These points, however, all depend on general principles, and cannot be further dwelt upon. Among the local measures, one of the first and most important to attend to is *rest*. If moving about produce pain, repose in the recumbent attitude ought certainly to be enforced,

and in recent and acute cases it should be absolute. In chronic cases continuous rest leads to the evils of deterioration of the general health, and the risk of acquiring habits of chronic invalidism. This must, then, be decided by the exigencies of the particular case, and the judgment of the practitioner. Generally, some daily gentle exercise, short of fatigue, should be advised, such as walking a little distance, driving in an easy carriage or bath-chair, or sitting in the open air. We may safely assume that exercise which does not produce or increase pain is doing good.

In cases of simple hyperæmia, especially when not of old standing, and when the uterus is tender to the touch, the local abstraction of blood is often of marked benefit. This may be effected either by applying from two to four leeches to the cervix through a cylindrical glass speculum, or, still better, by puncturing the cervix with a scarificator made for the purpose. Another very effectual means of relieving congestion and tenderness of the uterus is the use of pledgets of cotton wool, to which a string is attached, thoroughly soaked in glycerine, or in equal parts of simple glycerine and glycerine of ichthyol. If one of these is introduced into the vagina at night, and removed by means of the string in the morning, it will be found to produce an abundant watery discharge which saturates the linen of the patient. Great relief is thus given, and there is hardly any form of congestive uterine disease which is not benefited by this treatment, which most women can apply for themselves. The glycerine pledgets may be used every night, and they do not interfere with other modes of treatment. Continuous irrigations of hot water at 110° , night and morning, are also most serviceable, but to be of use not less than from one to two gallons must be used, with a suitable cistern-syringe. When properly applied these give immense relief. When the uterus is enlarged as well as tender, much benefit may be derived from the application of a pledget of iodised cotton to the cervix once a week. This should be passed through the speculum, and retained in position by a large pledget soaked in glycerine. It rarely causes pain; if it do, it should be at once removed; and it often remarkably reduces the size of the subinvolved and hypertrophied womb.

When there is evidence of endometritis, cervical or corporeal, other treatment is required. Now the desideratum is the application of alternative remedies to the diseased mucous membrane, not with the view of destroying it, but of so modifying its circulation and nutrition as to set up healthy action. The want of success so common in treating these cases may be traced to the fact that remedies have not been applied directly to the interior of the cervix or uterus, but that practitioners have contented themselves with treating the abraded or granular condition of the cervix, thus leaving the real seat of the disease untouched. Of late years much advance has been made, and we need not now talk of these chronic inflammatory affections of the lining membrane of the uterus in the same hopeless strain as before. One of the earliest modes of intra-uterine medication was the injection of fluids into the uterine cavity, such as tincture of iodine, or solutions of nitrate of silver. It was soon found that such injections, when the cervix had not been previously dilated, were apt to be followed by very alarming and dangerous symptoms; and it is now

generally admitted that they are inadmissible, unless the cervix has been previously dilated. This in itself is a procedure not to be lightly undertaken; and to repeat it frequently for a length of time—as would be essential in the treatment of these chronic cases—would be altogether out of the question. Some other method of attaining the desired object is therefore necessary, and this we obtain in perfection in the local application of the desired alternatives on suitable probes covered with a thin layer of cotton wool. By this means we can reach the mucous membrane at any part of the uterine cavity, and apply our remedies to it, without the necessity of any preliminary dilatation. The probes used by the writer are made of flexible metal (fig. 5), attached to a wooden handle, and of such a size that, when tightly wrapped round with a thin film of wool, they are not thicker than an ordinary uterine sound. They are covered by teasing out a small portion of wool, which is flattened between the palms of the hands. The probe is then dipped in water, to cause the wool to adhere, and by twisting round the handle, the wool being held between the forefinger and thumb of the left hand, the wool is smoothly and firmly wrapped round it. A little practice enables us to effect this with great neatness. The cervix being generally abnormally patulous, there is no difficulty in passing the probe through the os, previously exposed by the speculum, so as to reach the entire uterine cavity. The writer is in the habit of using a mixture of equal parts of crystallised carbolic acid and glycerine, as a local alternative, than which, he believes, there is no better application. Others, however, employ tincture of iodine, iodised phenol, or strong solutions of

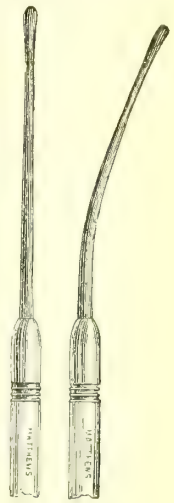


FIG. 5.—Uterine Probes.

nitrate of silver; or they even, as recommended by Courty of Montpellier, pass a solid piece of nitrate of silver into the uterine cavity, leaving it there to melt and flow over the mucous membrane. The writer first swabs out the uterine cavity with one or two probes covered with dry wool, so as to remove the glutinous discharge as much as possible, and then passes in another probe, covered with the carbolic solution, so as to paint over the lining membrane of the cervix and body of the uterus, the external abrasions on the cervix being subsequently swabbed over with the same solution. This rarely gives rise to any pain or discomfort, and may be combined with the other plans of treatment already mentioned. Intra-uterine medication is most useful in the week immediately succeeding menstruation, when the superficial layer of the mucous membrane has been shed. If used too near the advent of the next period it may prove too irritating, and may bring on menstruation prematurely. As a rule, two applications, at intervals of a few days, in the early part of the inter-menstrual interval are amply sufficient, and it may be necessary to continue the treatment for many weeks or months. The writer has found the application of the negative pole of a galvanic current, through a

carefully insulated sound, a still more efficient and much more rapidly effectual method of intra-uterine medication. Some three or four applications of about 100 milliamperes will often effectually cure an intra-uterine leucorrhœa of long persistence. Unfortunately this is a method of treatment that is not generally applicable, since it requires an expensive plant, and very considerable practical experience, in order that it may be safely and efficiently used. Should laceration of the cervix and ectropion exist, Emmett's operation of trachelorrhaphy may be indicated. In very severe and obstinate cases of this kind Recamier used to scrape the uterine mucous membrane with a curette, so as to remove the granulations, especially when there was much metrorrhagia, and where there was reason to suspect the existence of a granular condition of the intra-uterine mucous membrane. This operation, 'curettement' or 'curetting,' has been much used of late years in such cases, as well as in chronic metrorrhagia. With strict antiseptic precautions it is quite safe and often effective. For its exact technique the reader must be referred to treatises on operative gynecology. The Dublin physicians recommend the application of fuming nitric acid, the cervix having been first dilated. This is a very strong measure, which we would not willingly adopt; but it is only fair to say that in the cases in which the writer has used it he has found it exceedingly useful. Valuable as intra-uterine medication certainly is in suitable cases, it should never be rashly or indiscriminately employed, and the writer would strongly insist that, before resorting to it, we should satisfy ourselves that the uterus is likely to bear it with impunity. Whenever, therefore, there is much tenderness of the womb on being touched, even when the case is otherwise suitable, the writer deems it advisable first to remove the congested condition by rest, the local abstraction of blood, the use of glycerine pledgets, hot-water irrigations, and other appropriate means; and, above all, the slightest evidence of any concomitant mischief or irritation, recent or of old standing, in the neighbourhood of the uterus, as shown by tenderness on pressure in the region of the broad ligaments, or fixity of the uterus, should be an absolute contra-indication.

2. **Displacements.**—Under this head we have to discuss a variety of diseases which have furnished a fruitful theme for controversy among gynecologists. Practically, physicians have divided themselves into two great schools with regard to these affections. One of these schools teaches that deviations of the uterus, in whatever direction they occur, whether forwards, backwards, or to either side (for descent of the uterus is of a different character, and must be separately discussed), form of themselves serious maladies accompanied by definite symptoms; and, as a logical deduction, that their treatment should primarily consist in the endeavour to replace the dislocated uterus in its normal position, and to maintain its position by mechanical contrivances known as *pessaries*. The other, and much larger, school holds that versions or flexions are not *per se* the cause of the undoubted symptoms which are met with in the cases in which they are found to be present; that flexions may, and often do, exist, without giving rise to any symptoms at all; that in all cases the symptoms may be traced to the uterine engorgements and congestions which accompany the deviations, and are their primary cause; and

that, therefore, it is unnecessary to pay attention to the displacement, which may be left to itself, while associated conditions are remedied by appropriate treatment. As is generally the case in all such controversies, it is probable that neither side is entirely in the right, and that the truth is to be found between the two. It is certain that in some cases, and under certain peculiarities of constitution, flexions produce symptoms which cannot be explained by the accompanying condition of the uterine structures, which can only be relieved by mechanically supporting the dislocated womb, and frequently are so relieved in the most remarkable manner. It is unquestionable, however, that marked flexions often exist without producing any appreciable symptoms; and as these are only found out accidentally, when, from some other cause, an examination is made, it is not improbable that such cases are much more common than is generally supposed. Upon the whole, the writer is inclined to think that displacements are rather the result than the cause of the associated morbid state of the uterus, and that one of the chief elements of treatment is to get rid of the congested, hypertrophied, or sub-involted state of the organ which produces them. But, even if this be admitted, it by no means follows that proper mechanical support is not needed. Far from this being the case, the writer believes it to be of the greatest possible value in supporting and steadying the overweighted and misplaced organ, thereby facilitating the removal of its morbid states, as well as relieving its most urgent symptoms.

Properly speaking, displacements of the uterus should be divided into two classes, *versions* and *flexions*. In the former the body of the uterus retains its normal shape, but not its normal direction, the entire organ being displaced either forwards (*ante-version*), backwards (*retro-version*), or to one side (*latero-version*). In the latter the shape of the uterus is altered, and its body is more or less acutely bent over the cervix in the form of a retort, producing the analogous conditions of *ante-flexion*, *retro-flexion*, or *latero-flexion*. But these states are very closely related to each other. Very often they are combined, they arise from similar causes, and they produce similar results. For the sake of brevity, therefore, they will be discussed together.

ÆTIOLOGY AND PATHOLOGY.—In order to understand how uterine displacements are produced, it is necessary to remember the means by which the uterus is maintained in its natural position. In the healthy state the uterus is situated high in the pelvic cavity, its fundus projecting somewhat above the plane of the pelvic brim, to the axis of which—that is, to a line drawn from the umbilicus to the coccyx—its own axis corresponds. It is maintained in this position partly by the muscular column of the vagina below, on which it is, as it were, poised; partly by the folds of the peritoneum forming Douglas's pouch behind, especially that portion of them called the utero-sacral ligaments; and partly by fibrous portions of pelvic fascia in front, attached to the pubes and passing by the side of the bladder. These are fixed to the uterus above the points of junction of the cervix and body, which is, therefore, the part of the organ least liable to change of position, and that at which flexions almost invariably take place. The fundus and body are much more mobile, but their movements are somewhat controlled by the round ligaments in front

and the broad ligaments at the side. The shape of the uterus is further maintained by the well-marked inherent rigidity of its tissue, and when this is altered by disease, as by congestion, sub-involution, and the like, displacements are much more apt to occur. The axis of the uterus is naturally apt to alter its position under various conditions. Thus it falls less or more forwards, according as the bladder is distended or otherwise. The uterus is also so placed that it ascends or descends with more or less freedom, and, as it does so, its axis corresponds with the axis of the pelvis at the particular level of that part. This fact goes far to explain why similar causes should at one period produce a forward, and at another a backward, displacement. The causes of displacements are chiefly any conditions which weaken the supports, or the resisting power, of the uterus. They are, therefore, most frequently found in association with the results of parturition—sub-involution, congestion, hyperplasia, and endometritis—which all diminish the inherent tonicity of the uterine walls, as well as weaken its supports, and prevent its regaining its natural shape when accidentally altered. The displacement itself may be caused or favoured by a variety of conditions, such as blows, falls, tight clothing, fibroid tumours developed in the uterine walls, and many other analogous states. When a flexion has been produced, the venous capillaries at the point of flexion are more or less obstructed, and the return of blood through them hindered, while the arteries continue to supply blood. The fundus, therefore, becomes congested, and subsequent structural alterations are developed. This point was insisted on by Graily Hewitt, and there can be no doubt of its accuracy. This explains the fact that, even when flexions are secondary, it is impossible to treat them satisfactorily by general means alone, and that, in course of time, a flexion, originally secondary in its causation, may require to be the chief object of treatment.

SYMPTOMS.—The symptoms of flexion of the uterus are in no way special. They are very much those which have already been described as accompanying the inflammatory varieties of uterine disease, and there are none which would enable the practitioner to foretell its existence with certainty. Among those most commonly observed are pain, weight, and bearing down, often decidedly increased by exercise of any kind, not infrequently rendering locomotion an impossibility; pain in one or other ovarian region; pain, and sometimes difficulty, in micturition; and various disorders of the menstrual function, more especially dysmenorrhœa and menorrhagia. After these have lasted some time the secondary derangements of the general health, so common in uterine disease, become established, and they lead to very serious consequences.

DIAGNOSIS.—The diagnosis of displacements is not difficult on vaginal examination. Supposing we have to do with a flexion, the cervix is found in its normal position; but either in front, behind, or, more rarely, on one or other side, is a rounded swelling about the size of half an orange, which can be pushed away by the examining finger. This might be confounded with various other conditions, such as small uterine fibroids, inflammatory exudation, hæmatocele, and small ovarian tumours. The diagnosis can, however, be readily cleared up by the sound, which will only pass in the direction in which the uterus is flexed; and, when it is turned

gently and cautiously round, the flexed fundus is lifted with it, and can no longer be felt in its former position. In ante- or retro-version, in addition to feeling the body of the uterus in its abnormal situation, we also find that the cervix is lifted out of its usual central position, and points forwards or backwards respectively.

TREATMENT.—In arranging a plan of treatment for uterine displacements, the concomitant conditions should be carefully attended to, and endometritis, congestion, and other complications relieved, if they exist. Unless this be done, the treatment may entirely fail, or may be very unnecessarily prolonged. Here we must limit ourselves to a very brief description of the best plans of mechanical treatment, strongly insisting, however, that they should not be too exclusively relied on. In backward displacements we have a very satisfactory means of supporting the misplaced organ in the well-known Hodge's pessary, numerous modifications of which exist. The important point to bear in mind is to select an instrument not too bulky, nor too long, for the individual case; the best material being either wire covered with soft india-rubber, vulcanite, or aluminium. The treatment often fails from want of proper selection; since a pessary, to be thoroughly useful, should be fitted as accurately as a shoe to a foot. Before introducing it, the sound should be gently passed, and the uterus replaced and held in its proper direction for a few minutes; and this manœuvre should be repeated from time to time, until the uterus does not re-assume its abnormal position. Even then, however, the pessary should be worn continuously for several months, until we feel quite sure that the displacement is permanently relieved. In many cases relief is instantaneous and remarkable; in others the fundus is too tender to bear the pressure of the pessary at all. When this is the case it should be removed, and an endeavour should be made to prepare the uterus for the use of this support by the local abstraction of blood, hot irrigations, the application of glycerine pledgets, or sedative pessaries. In introducing the Hodge's pessary, care should be taken to guide its upper part into its proper position behind the cervix, so as to press up the fundus; and, as the case improves, a larger instrument should be introduced, so as to follow up the re-treating fundus. Reposition of the uterus must, of course, never be attempted if there is reason to think that the fundus is bound down by adhesions, or if the uterus is very tender. In suitable cases, however, it materially facilitates the cure.

Anterior displacements must be treated on the same principles. Unfortunately, we do not possess anything like an equally good means of mechanical support, and a thoroughly efficient ante-flexion-pessary is still a desideratum. After trying a good many, the writer has come to the conclusion that the best is Graily Hewitt's vulcanite cradle-pessary, of a size suitable to the case. It is, however, difficult to introduce and remove. Nor can it always be borne. A well-fitting abdominal belt—and the best is that known as the American belt—is often of great assistance, by removing the weight of the superincumbent intestines. When all other means of restoring a flexed uterus fail, an intra-uterine stem-pessary may possibly succeed. Great caution, however, is necessary, and it should never be used unless the patient is under constant supervision, so that it may be

removed on the slightest appearance of undue irritation. Various operative measures have lately been adopted for the radical cure of uterine flexions. During the treatment of all flexions, rest is of paramount importance—not absolute, but as much as possible; and exercise should be very sparingly permitted, and only in a tentative manner. In this (and the same remark applies to every other form of uterine disease), care should be taken that local examinations should be as infrequent as the necessities of the case will allow. In all such cases there is a distinct danger that ill-judged and unnecessarily frequent local treatment may end in producing neurotic complications which may prove to be worse than the disease they profess to cure.

Prolapsus.—The only other displacement of the uterus requiring mention is descent or *prolapsus*, for the corresponding condition of undue elevation is of little practical importance. Descent of the uterus is far from rare, and it sometimes causes much discomfort. In it the uterus descends from its normal position to a varying extent, so that the prolapsus may be only partial, or it may be complete. When the entire uterus lies outside the vulva, along with the everted vagina, the mass is known as *procidencia uteri*.

ÆTIOLOGY AND PATHOLOGY.—Descent of the uterus depends upon a variety of causes which lead to a weakening of the uterine supports, either from above or below. These supports are chiefly, above, the various uterine ligaments, with the cellular tissue of the pelvis; below, the muscular column of the vagina. When from any cause, such as imperfect recovery after childbirth, senile absorption of adipose tissue, or rupture of the perinæum tending to prolapse of the vagina, they are no longer able to support the uterus efficiently, a comparatively slight cause may suffice to allow the womb to be pushed or drawn out of its place, as, for example, straining, lifting heavy weights, undue weight of the organ itself, and many other causes. As soon as the organ is prolapsed, to whatever degree this may occur, various morbid alterations are pretty sure to follow. The uterus becomes irritated, congested, and hypertrophied; and the everted mucous membrane of the vagina, which then covers it, becomes greatly altered, and assumes almost the appearance of skin. The most characteristic change, however, is generally elongation of the cervix, through traction from below, especially in cases in which prolapse follows previous descent of the vaginal walls. This elongation is due to yielding of the elastic tissue of the cervix, especially that portion above the roof of the vagina, which becomes greatly elongated, so that the sound introduced into the procident organ may pass for six or seven inches. When the uterus is replaced, the normal elasticity again comes into play, and the cervix rapidly contracts. This is the condition described by Huguier as *hypertrophic elongation of the cervix*, and it is rarely absent.

SYMPTOMS AND DIAGNOSIS.—The symptoms of prolapse of the womb are mainly due to the mechanical discomfort attending it, such as weight, difficulty in progression, interference with micturition, and much general inconvenience. The diagnosis is a matter of no difficulty. In the greater degrees the procident organ, covered with the hardened and altered vaginal mucous membrane, is at once apparent. The only condition with which it is at all likely to be confounded is old-standing inversion of the

uterus, and from this mistake the presence of the os uteri at the apex of the tumour will at once guard us. In the slighter degrees the cervix will be felt low in the vagina, or even at its orifice.

TREATMENT.—The treatment comprises two principal indications: First, rest, and consequent reduction of the size and weight of the procident organ; thereby greatly facilitating the second indication, namely, reposition, and maintenance of the uterus in its normal position. The effect of mere rest in diminishing the size of a prolapsed uterus is often very remarkable. A week or ten days in bed will often, of itself, reduce the cervical elongation to a considerable amount. Reposition is generally easily effected, and the chief difficulty is in support. In devising mechanical contrivances for keeping the uterus in position, the chief thing to bear in mind is that we should strengthen the natural uterine supports, so that they may regain their lost power of keeping the organ in its place. Hence the old ball-pessaries, which greatly distend the vagina, are absolutely inadmissible. In the slighter degrees an ordinary Hodge's pessary may answer every purpose, an endeavour being, at the same time, made to give better support from below. This is generally best done by a perineal pad, and also sometimes by using an astringent to the vagina, so as to make it a more efficient column of support, such as alum or oak-bark injections. In the greater degrees the best kind of pessary is that known as the 'vaginal stem,' which is in the shape of the cup of the ordinary toy known as the cup-and-ball, on the extremity of which the cervix rests, the other end being attached to a perineal band. If this does not answer, the pessary known as Zwancke's may succeed. This has the advantage of supporting the uterus without unduly distending the vagina. These measures are merely palliative, and a more radical cure may be hoped for by various operative procedures, into the details of which it is impossible to enter. It may suffice to say that they consist of: First, the removal of a small portion of the elongated cervix, either by the galvano-caustic wire or by the écraseur, in the hope of stimulating the remainder to contract, a procedure only occasionally required, but sometimes of undoubted utility. Secondly, the making of a new perinæum, in cases in which the prolapse seems secondary to undue descent of the vagina—a very valuable resource. Thirdly, narrowing of the vagina itself, for which a variety of operations are practised, which, like removal of a portion of the cervix, are rarely required.

3. **Carcinoma.**—Carcinomata are of common occurrence in the generative organs of females, and indeed they are more often met with in these than in any other part of the body.

ÆTIOLOGY.—Malignant disease of the uterus is most common about the middle period of life, the largest number of cases occurring between forty and fifty years of age. Occasionally we meet with it in old women who have long ceased to menstruate, or with rapidly advancing cases in younger women under thirty years of age. Hereditary predisposition apparently plays some part in its production, but not so much as was formerly believed. Frequent child-bearing has a decided influence in favouring its development, since the proportion of cases is larger in *multiparæ*. To this may be added any causes of constitutional debility, since malignant disease unquestionably occurs more often in

weak and unhealthy, than in strong and robust women.

ANATOMICAL CHARACTERS.—All the recognised forms of cancer occur in the uterus, but some of them are more common than others. Scirrhus is that which is least frequently observed, and medullary carcinoma most often. Klebs, however, has pointed out that in the earliest stages of carcinomatous disease the fibrous element preponderates in the affected part; while, as the malady progresses, cell-growth rapidly advances, so that in old-standing cases, and at *post-mortem* examinations, the medullary carcinoma is that which is found, although, at first, the scirrhus form may probably have existed. Another common variety is the epithelial cancer, which chiefly affects the mucous membrane of the cervix. In the large majority of cases the cervix is the part first affected, although, in exceptional instances, the disease may originate in the body of the uterus, the cervix being at first unimplicated.

At the commencement of the more ordinary variety the cervix is hard, somewhat nodular, and hypertrophied. It is comparatively rare for the disease to be seen at so early a stage, and, as the uterus is then quite movable, it is impossible to distinguish it from enlargement of the cervix due to congestive forms of disease. As the disease progresses, more and more tissue is involved; and the roof of the vagina becomes implicated, so that the uterus is fixed in a mass of new-growth. Before long a destructive process commences; portions of the growth slough and come away; hæmorrhage takes place from the opening of vessels; and in advanced stages of the disease, the cervix may be entirely destroyed, and even the bladder or rectum opened, so as to form a common opening with the vagina. The epithelial variety of cancer commences on the mucous surface of the cervix by the growth of its villi into a papillary tumour, which, at first, it may be impossible to distinguish from similar growths of a benign character. It rapidly spreads, however, soon forming a fungating soft mass, not unlike the flower of a cauliflower in appearance; hence its popular name 'the cauliflower excrescence.' As in medullary carcinoma, destructive changes soon occur; ulceration progresses; and, as the disease advances, the neighbouring structures are implicated. When the disease involves the more deeply seated tissues, *post-mortem* examination shows that the malignant growth there assumes more of the character of medullary cancer.

SYMPTOMS.—The earliest sign that arouses suspicion as to the existence of carcinoma is generally the occurrence of hæmorrhage, at first merely an excessive menstrual flow, subsequently loss of blood, sometimes very great in amount, at irregular intervals. Sometimes it is brought on by trivial causes, and a not infrequent complaint is that it always occurs after sexual intercourse. Excessive hæmorrhages, which are sometimes very alarming in amount, do not, as a rule, occur until advanced stages of the disease, when destruction of tissue is taking place. Another marked symptom is profuse discharge, often having a peculiar and pathognomonic fœtor, from the admixture of minute portions of sloughing tissue. In medullary carcinoma the discharge is not usually abundant, but it is sometimes very ichorous in character, producing excoriations, intense pruritus, or other disagreeable symptoms, in consequence of its irritating property.

In epithelioma it is often very abundant, watery in character, and tinged with blood. Pain is sometimes excessive. The writer has often, however, seen cases terminate fatally without any pain at all. General constitutional disturbance soon results; the peculiar cachexia of malignant disease is developed; and the patient becomes sallow, emaciated, and extremely debilitated.

DIAGNOSIS.—In advanced cases vaginal examination at once clears up the nature of the case. The cervix and roof of the vagina are infiltrated with the characteristic hard growth, and the uterus is quite fixed. If ulceration is advanced, the ragged broken edges of the cervix are apparent, bleeding on being touched, and breaking down under the finger. In a case of this kind mistake is hardly possible. In the earlier stages, before fixation and ulceration have advanced, error is easy, and it is far from uncommon to find non-malignant alterations of the cervix which have been mistaken for cancer, and even the reverse. Nothing but time will clear up such doubtful cases, and care should be taken not to give a positive diagnosis, unless the character of the case is marked and undoubted. The most difficult cases to diagnose are those in which the body of the uterus is alone affected. Here hæmorrhage, foetid discharge, and recurrent attacks of spasmodic pain (probably caused by the efforts of the uterus to expel discharge collected in its cavity) may arouse suspicion; but nothing except dilatation of the cervix, and thorough exploration of the uterine cavity, can clear up the nature of the case. On account of the friability of the tissues, this must always be done with great caution. In epithelial cancer the soft, fungating, bleeding, and easily broken-down mass can hardly be mistaken for anything else.

PROGNOSIS.—The prognosis, is of course, most unfavourable. There are, no doubt, a few authentic cases on record in which the patients have recovered after amputation of the cervix, but these are of the utmost rarity. In the vast majority death takes place, in a time varying from a few months to one or two years. Probably the disease advances most rapidly in younger patients, but few last longer than two years. Death takes place either from exhaustion, hæmorrhage, septicæmia, or some other complication. The writer has seen it on one or two occasions result from uræmia, the consequence of occlusion of the ureters through extension of the cancerous growth.

TREATMENT.—In the large majority of cases seen for the first time at an advanced stage, treatment must, of necessity, be palliative only. Here there are two chief indications—the maintenance of the general health; and the relief of pain, fœtor, or other distressing local complications. For the former we must rely mainly on a suitable diet, and the administration of some tonic which may be found to agree with the patient. The preparations of arsenic, as a rule, answer better than either iron or quinine. For the relief of pain the use of opiates, either in the form of morphine suppositories or administered subcutaneously, must be our sheet-anchor, and, when the pain is severe, it may be necessary to exhibit them in large doses. Fœtor may be best arrested by the local use of antiseptics. The plan which the writer has found to be most successful for the purpose is to introduce at night into the vagina a pledget of cotton wool, soaked in the glycerine of tannic acid, to which a small

quantity of the glycerine of carbolic acid has been added, in the proportion of about one ounce of the latter to eight ounces of the former. This effectually destroys foetor, while the astringent property of the tannic acid serves to check unduly rapid cell-growth, and prevent hæmorrhage. In the morning the vagina may be syringed out with a weak solution of creoline. Iodoform pessaries are also extremely useful for this purpose. When hæmorrhage is excessive, local hæmostatics, such as the diluted solution of perchloride of iron, must be used, and, if necessary, the vagina plugged. Surgical treatment may be adopted in the hope either of entirely removing the disease, or of lessening the hæmorrhage and discharges, and so retarding its progress. Either indication is most easily fulfilled in epithelial cancer. When this is limited to the cervix, and there is a fair margin of healthy tissue between the diseased portion of the cervix and the body of the uterus, supra-vaginal amputation or amputation by the galvano-caustic wire or wire écraseur should be resorted to, the actual cautery being subsequently freely applied to the stump, to destroy, as much as possible, any infiltrated cancer-cells. Even when this fails to prevent the recurrence of the disease, it will certainly prolong the life of the patient, and increase her comfort. In other cases destruction of the exuberant growth, both in epithelial and medullary cancer, may be advantageously effected by local caustic applications, such as strong solutions of chloride of zinc, nitric acid, or bromine. The same object has been sought by excising the diseased tissue, or by scraping it away as much as possible by peculiar scoop-shaped curettes made for the purpose, after which one of the above-mentioned caustics, or the actual cautery, may be applied. It is in the epitheliomatous form of malignant disease that this procedure is most valuable, and a carefully performed operation may prolong life for months or years.

Vaginal extirpation of the uterus has been largely practised, and improved technique has now reduced the mortality of the operation to very small limits. It has certainly not been followed by a satisfactory proportion of good results, as in most cases the disease has recurred within a comparatively short period. If, however, the disease is detected early, when the uterus is absolutely mobile, and there is good ground for hoping that there is no spread of the disease to the neighbouring glands and tissues, then the operation is certainly indicated; for even if the disease eventually recurs, the patient will have been saved much suffering from the sloughing of the uterine tissues, and she will at least have had the chance of complete cure. Some more thorough operative procedure is desirable, which shall do for uterine cancer what surgery has lately done for mammary cancer.

4. Other Tumours.—(1) *Fibroid Tumour.* By far the most common variety of uterine tumours—so common that, according to some authorities, it is found in 60 per cent. of women who die after middle age—is the so-called fibroid tumour (fibro-myoma).

ÆTIOLOGY.—Fibroid tumours are most common after twenty years of age, and in certain races—the African especially, they occur with great frequency. Beyond this nothing is known of the causes which produce or favour their growth.

ANATOMICAL CHARACTERS.—Fibromata are limited hypertrophies of the substance of the uterus,

existing in the form of more or less globular tumours, contained loosely in a capsule of fibro-cellular tissue. Structurally they are homologous with the tissue of the uterus itself, consisting of connective tissue, mixed with unstriped muscular fibres. They are found of every size, varying from dimensions scarcely larger than a millet-seed, up to enormous masses weighing as much as fifty pounds. Most generally they occupy the fundus and body of the womb, and they are conveniently divided into three classes, according to the position they occupy, namely, the *sub-peritoneal*, occupying chiefly the outer surface of the uterus; the *intra-mural*, chiefly developed in the substance of the walls; and the *sub-mucous*, which project into its cavity; and these may be either completely sessile, with a broad base of attachment, or they may have become more or less pedunculated, and then approach in character to the fibroid polyp.

When once formed, the future progress of fibroids of the womb varies much. Generally they are of very slow growth, and although malignant degeneration of their structure has been observed in rare instances, they most commonly exist for the rest of the patient's life, without giving rise to any troublesome effects, beyond those resulting from mechanical pressure, provided they are not accompanied by hæmorrhage. They are, however, subject to certain occasional alterations, such as inflammation and even sloughing, when mechanically injured, fatty and calcareous degeneration, and even to complete absorption. The possibility of their entire spontaneous disappearance has been strongly questioned. The writer has published several instances coming under his own observation, and the fact is now pretty generally admitted, the explanation probably being that on account of similarity of texture to the tissue of the uterus, they are subject to a process of involution like that which the uterus undergoes after delivery.

SYMPTOMS.—The symptoms of fibroids of the womb depend to a great extent on their position. The sub-peritoneal and interstitial varieties, when not very large, are often unaccompanied by any symptoms whatever, and those that exist are chiefly the result of mechanical pressure, such as weight, difficulty in walking, irritability of the bladder, constipation, and the like. In the sub-mucous variety, the prominent symptom is hæmorrhage, which sometimes occurs to a very alarming extent, and may even put the patient's life in danger. The source of the hæmorrhage is probably, in the majority of cases, minute capillary vessels in the mucous membrane covering the tumour, which keeps up irritation and congestion in its neighbourhood. It is similar in its origin, therefore, to the discharge in menstruation, and is greatest in amount at the menstrual period. The more severe hæmorrhages are probably caused by openings in various vessels developed in the periphery of the tumour, where the vessels are increased in size, just as they are in pregnancy.

The physical signs vary with the size and situation of the tumours. If large enough to be perceptible on abdominal palpation, they have generally a more or less rounded or lobular outline, and a hard, firm feel, without any fluctuation, which serves to distinguish them from other varieties of abdominal growth. Small tumours, however, growing from the sides of the uterus, may be easily confounded with other conditions, especially flexions

of the uterus, and deposits or exudations in its vicinity, such as hæmatocele, or pelvic cellulitis or peritonitis. The mobility of the uterus, which in the latter conditions is generally impaired, and the use of the uterine sound, which shows, in tumours, that the cavity of the uterus is considerably elongated, ought to enable us to avoid such mistakes.

TREATMENT.—The treatment resolves itself into *medical and surgical*. The former may be said to be powerless, all the so-called absorbents—iodides, bromides, preparations of calcium, Kreutznach and other waters, being admitted to be of no reliable value whatever. The subcutaneous injection of ergotin, in doses of half a grain to a grain daily, is said by Hildebrandt to be of great value, and is favourably spoken of by Atthill and other authors. It is, however, not reliable, and has often to be discontinued on account of the irritation produced by the injections. Of late years the treatment of such tumours by the electrical current, chiefly advocated by Apostoli, has been much discussed. It is claimed that electro-negative puncture has a decided effect in promoting absorption. The writer believes this to be frequently the case, but the procedure is not without risk, and it seems to him to be only very rarely permissible. Surgical methods of treatment are chiefly called for when the hæmorrhages are excessive and exhausting.

(a) The hæmostatic effect of the electro-positive current applied to the uterine cavity by an insulated platinum or carbon sound the writer believes to be of great value. He has now seen numerous cases in which the most severe hæmorrhages have been controlled by this means. It apparently acts by hardening the mucous covering of the tumour. The great objection to this treatment is the necessity for a special and costly plant, its length, and its uncertainty, for it unquestionably often fails. Under favourable circumstances, however, it is well worthy of trial.

(b) Injection of *styptics* into the uterine cavity, such as tincture of iodine, or diluted tincture of the perchloride of iron, is valuable when the hæmorrhage is excessive, but is a plan which should not be tried unless the cervix has been previously dilated.

(c) *Incision of the cervix* is decidedly beneficial when the hæmorrhage is frequent and severe, and is supposed to act by allowing the uterine fibres to contract upon the tumour.

(d) *Incision of the capsule of the tumour* itself is useful in certain sessile tumours projecting into the uterine cavity, certainly diminishing the hæmorrhage, and facilitating subsequent enucleation.

(e) *Removal of as much as possible of the tumour*, with the *écraseur* or galvano-caustic wire, may be undertaken, when it is so situated as to be within reach.

(f) *Enucleation* is very valuable when the tumour is projecting into the uterine cavity, and is already partially separated from its attachments. Under such circumstances it may be shelled out from its capsule *en masse*. The operation, however, is difficult and severe, and must be reserved for very urgent cases.

(g) *Hysterectomy*, with the removal of the mass of the tumour, and even of the entire uterus, has now been brought to such perfection, that it may be resorted to with much more confidence than formerly. The tendency in modern practice is,

perhaps, to adopt this expedient too often, and too early, in cases in which there was no urgent call for so serious a procedure.

(h) The artificial production of the menopause by *removal of the uterine appendages* is an operation which has been highly recommended, and is chiefly valuable when the tumour is of small size.

(2) *Polypus*.—Polypus of the uterus may be considered in connection with this subject, since the large majority of polypi are merely *fibroid* tumours, to a great extent enucleated from their capsules, and attached to the uterus by a narrow pedicle. There are, however, two other varieties described, the *glandular*, and the *cellular* polypi.

ANATOMICAL CHARACTERS.—The *glandular* polypus is a localised hypertrophy of connective tissue, rarely larger than an egg, and generally attached to the cervix; the *cellular* is a hypertrophy of some of the glandular structures of the cervix, either of the Nabothian follicles or the utricular glands, and it is generally of small size. *Fibroid* polypi, like fibroid tumours, may be of any dimensions, but they are commonly met with about the size of a small pear.

SYMPTOMS AND DIAGNOSIS.—Polypi are only important because of the hæmorrhage which so frequently accompanies them. When extruded from the cervix, or growing from it, they offer no difficulty in diagnosis. Nor is there any condition apt to be mistaken for them, except a chronic inversion of the womb, which can be readily differentiated by tracing the pedicle through the os, and finding that the sound enters the uterus to its usual depth. Intra-uterine polypi are much more difficult to discover, and here the only plan is complete dilatation of the cervix, and thorough exploration of the uterine cavity. This procedure is essential in all cases of severe hæmorrhage resisting ordinary hæmostatic treatment.

TREATMENT.—When once a polypus has been detected, the only treatment is its removal. In cases in which the polypus is chiefly in the vagina, this is easy enough, the wire of an *écraseur* being passed round the pedicle, which is separated in this way from its attachment. It is not essential that the wire should touch the base of the pedicle, since the part left, after the bulk of the polypus is removed, always shrivels up and disappears. In intra-uterine polypi it is sometimes difficult to pass the wire round the pedicle, but by thoroughly anæsthetising the patient, and using a single wire, it can generally be managed. No other method of removing polypi is so good. Excision may give rise to hæmorrhage, and the old method of ligature is dangerous from the risk of inducing septicæmia.

(3) *Fibro-cystic and Sarcomatous Tumours*.—The only other uterine growths requiring mention are the fibro-cystic tumours (cysto-fibroma, cysto-sarcoma), and the sarcomatous tumours.

The *fibro-cystic* tumour is specially important on account of its great resemblance to cystic tumours of the ovary, and the extreme difficulty of differential diagnosis, which is so great that probably there is no ovariologist of any experience who has not mistaken the one for the other. The fibro-cystic tumour is a fibroid tumour of the uterus, generally of the sub-peritoneal variety, which has grown to a large size from the development of cysts in its substance. This is most usually effected by degeneration of its tissue, which becomes

liquefied and transformed into an albuminous fluid. Thus we have a tumour partially solid, partially fluctuating—although rarely so distinctly so as an ovarian tumour—from which fluid can be drawn by an exploratory puncture, and which may attain dimensions, and produce constitutional effects, not less marked than those of ovarian cystic disease. The differential diagnosis may well baffle even the most expert gynaecologist. The sound may possibly enable us to ascertain the uterine character of the growth, as it enters for a considerable length, and on moving it the connection of the tumour with the uterus may be demonstrated. Atthill has laid stress on the character of the fluid removed on puncture as diagnostic, since, unlike ovarian fluid, it coagulates spontaneously on standing, and on microscopical examination elongated fibre-cells, similar to those of the uterus itself, are found in it. The prognosis of these growths is unfavourable, since they produce all the evil effects of ovarian tumours, and the risk attending their removal by operation, which is the only available method of treatment, is much greater than that of ovariotomy.

Sarcomatous tumours of the uterus have been but little studied. They are in many respects like fibroid growths, but have a marked tendency to fungate and ulcerate, and to return after removal. Hence, they hold a place midway between the benign fibroid and the malignant cancerous growths, and they generally spring from the uterine tissue, like the sub-mucous fibroids, but without any distinct capsule. The symptoms are chiefly those of fibroid in that situation, namely, hæmorrhage; profuse watery discharges, and sometimes pain, probably the result of uterine contractions. The treatment must consist either in complete hysterectomy, or, under certain circumstances, in the removal of as much as is possible of the tumour by the *écraseur* or galvano-caustic wire; and, if the nature of the tumour be ascertained, its attachment should be thoroughly cauterised with strong nitric acid.

Deciduoma malignum is the name given to a peculiar and very severe form of disease occurring shortly after child-birth or abortion, which has attracted much attention of late years. Pathologically it is of great interest because of its connection with recent pregnancy; clinically it is not different from a very rapidly progressing form of intra-uterine sarcoma (see p. 1314). Its diagnosis requires the dilatation of the uterine cavity, and thorough exploration under an anæsthetic, and this is called for when we meet with profuse hæmorrhage and offensive discharges shortly after labour or abortion. (See p. 1018). The only remedy is immediate vaginal hysterectomy, any merely palliative measures, such as curetting and the like, being worse than useless.

W. S. PLAYFAIR.

UVULA, Diseases of.—SYNON.: Fr. *Maladies de la Lette*; Ger. *Krankheiten des Zäpfchens*.—The uvula consists of a fold of mucous membrane enclosing muscles, aponeuroses, vessels, nerves, and glands. It may be bifid, either partially or completely, in cleft palate; it may be absent or rudimentary; and it may be attached irregularly.

From its intimate relation with the soft palate and other parts of the throat, naso-pharynx, and nose, it is frequently involved in pathological conditions affecting these structures. Indeed, it can scarcely be said to have any disease restricted to itself, but an extremely acute inflammatory oedema has been noted in gout, without coincidental implication of surrounding parts.

It may be affected by (1) Erosion in syphilis (mucous patches) and tuberculosis, including lupus; (2) Congestion and rash in acute specific fevers, e.g. scarlatina, small-pox, chicken-pox, &c.; (3) Oedema in acute pharyngitis, and hydræmia; (4) Membranous exudation in diphtheria, septic tonsillitis, gangrene; (5) Paralysis following diphtheria, or part of a bulbar paralysis; (6) New-growths—papilloma, cancer. See also p. 685.

Enlargement.—The condition of the uvula which most frequently calls for attention is simple hypertrophy or elongation combined with paresis, so that it is very imperfectly retracted when the effort is made.

ÆTIOLOGY.—This condition may be due to (1) nasal obstruction, whether it arise from hypertrophy of turbinates, spurs, deviation of septum, or overgrowth of the naso-pharyngeal tonsil (adenoids); (2) simple congestion of the pharynx from any cause, usually associated with paresis of the palate, so that the uvula not only is elongated, but is permitted to hang too low in the pharynx.

SYMPTOMS.—(1) Constant sensation of the presence of a foreign body in the throat, leading to hawking and coughing, and sometimes to inclination to retching—all worse on lying down; (2) alteration of voice—loss of power, clearness, and range; (3) spasm of glottis: the last is not common, but we have seen it occur where the uvula is greatly elongated.

TREATMENT.—Removal of the cause will cure a slight case of elongated uvula, but we must make the nose, naso-pharynx, and pharynx healthy to do this. Locally, astringent and antiseptic gargles and sprays will help. Where the hypertrophy is really considerable the only proper treatment is removal of a piece of the uvula by means of forceps and scissors after cocaineisation. Care should be taken to remove it obliquely so that the front is longer than the back, as causing less soreness in swallowing; and to leave a piece one-third of an inch in length; in fact we should only remove the redundant mucous membrane, leaving the original muscle intact.

J. BARCLAY BARON.

V

VACCINATION (*vacca*, a cow).—SYNON. : Fr. *Vaccination* ; Ger. *Kuhpockenimpfung*.

DEFINITION.—Inoculation with the material of vaccinia or the cow-pox for the purpose of protecting the person so inoculated or vaccinated from an attack, and especially from a severe or fatal attack, of small-pox. The material is now derived almost exclusively from the results of calf-inoculation and after-admixture with glycerine, in a manner to be presently described ; it is officially called *Glycerinated calf-lymph*, and is here spoken of as *Vaccine*.

The cow-pox, which is a natural, though not a common, disease of the cow and horse, never occurs spontaneously in the human subject. Nor is it communicated to him by effluvia, or in any other way than by the direct inoculation of its own specific virus. Such inoculation, before the time of Jenner, was merely accidental, and occurred with comparative rarity. There was a popular tradition that persons who had thus been accidentally vaccinated enjoyed immunity subsequently from small-pox. It was left for Jenner to demonstrate the facts upon which this tradition was founded, and to explain the deeply significant truth which accounted for their existence.

Relationship of Variola and Vaccinia.—

The title of Jenner's work published in 1798, viz. 'An Inquiry into the Causes and Effects of the Variolæ Vaccinæ,' carried the suggestion of identity of the two diseases, cow-pox and small-pox. It was, indeed, one of his fundamental doctrines that the cow-pox was the product of the same virus as that which produced the small-pox in man. The malignancy and peculiarly infectious character of human variola were not, in his view, any essential part of the effects producible by the variolous poison on the human constitution, but were properties at first accidentally superadded, and, when once acquired, capable of propagation. The author of this article as it first appeared in this Dictionary, Edward Cator Seaton (the father of the present writer), the chief authority of the time, and one who made the organisation of public vaccination in this country his life-work, upheld Jenner's views. The relation of cow-pox to small-pox has, however, until of late been the subject of doubt and conjecture. But now Monckton Copeman sums up the results obtained by numerous observers who, in various parts of the world, during the past century have endeavoured by experimental methods to solve the problem of the true relationship of the two diseases, and he declares without hesitation that : 'As the outcome of this work it may now be definitely stated that small-pox lymph, more especially if obtained from the primary vesicle of a case of the inoculated form of the disease, by passing through the system of the calf, can be so altered in character as to become deprived of its power of causing a generalised eruption, while inducing at the site of inoculation a vesicle indistinguishable from a typical vaccine vesicle ; and, more important still, that when transferred again to man, it has by such treatment completely lost its former infectious character. Such being the case, it may fairly be asserted that cow-pox—or rather that artificially inoculated form

of the disease which we term vaccinia—is nothing more nor less than variola modified by transmission through the bovine animal. An outbreak of small-pox, indeed, may be turned to account for raising, by appropriate experimental methods, a fresh stock of vaccine lymph.'

Vaccine.—Before proceeding to give an account of the phenomena of vaccination, it may be well to describe shortly the process of preparing vaccine, which, by the courtesy of Dr. Blaxall, the present writer has been able to observe for himself both at the Government Calf Vaccination Station at Lamb's Conduit Street, and in the Government laboratories on the Chelsea Embankment.

The material of vaccinia or cow-pox from which the lymph is derived came from various stocks (whose pedigree is recorded, but whose remote origin could not be demonstrated at the present day). It is grown on calves whose bellies are shaven for the purpose of inoculation with the virus, which is done by numerous long incisions. The resulting pocks or vesicles are carefully scooped out on the fifth day with a sterilised Volkmann's spoon, and transferred with aseptic precautions to bottles, and taken to the Government laboratories. The pulpy material is then finely ground up by machine, and subsequently triturated with four times its weight of a 50-per-cent. solution of chemically pure glycerine in distilled water. When the process of attrition and admixture is complete, the compound is a viscid fluid of a creamy consistence and appearance. This emulsion is run into 'stock-tubes,' i.e. miniature test-tubes, each containing 3 to 8 c.c. These, when filled, plugged, and sealed with aseptic precautions, are placed in earthenware jars and put in a dark cool cupboard or ice-chest, the temperature of which is kept at 15° C. the first week and at 10° C. afterwards. The glycerine possesses the peculiar property, which Monckton Copeman was the first to demonstrate in this country, viz. that organisms which might give a septic character to the stuff taken from the calves' bodies are destroyed without impairing the efficiency of the material, *quâ* vaccine, for practical use. After storage for about a month the process of bacteriological purification is found to be complete. How this has been effected is thus explained in simple language by Monckton Copeman :—

'There is much evidence to prove that the results following on vaccination are due to a specific contagium, and, moreover, that the particular micro-organism concerned is capable of existing, during one period of its life-cycle, in a resting or spore form, in which condition it is more resistant to the germicidal effects of glycerine than is the case with non-sporing microbes.'

The retention of the specific quality of the stuff is thus accounted for, while its purification is explained as follows :—

'By thoroughly incorporating the lymph or vesicle pulp with a sterilised 50 per cent. solution in water of chemically pure glycerine, and afterwards storing the mixture for some weeks prior to use, protected from light and air, all the ordinary saprophytes found associated with lymph are

eventually destroyed. This result is proved by the fact that no growth arises in any of the ordinary culture-media inoculated with such glycerinated lymph. This statement applies equally to the bacillus of tubercle and to the streptococcus of erysipelas, should these microbes have been originally present in, or have been added experimentally to, the lymph.'

As soon as the vaccine is proved to be bacteriologically pure by the tests applied week by week, capillary glass tubes of suitable size are charged with it and sent out in large numbers every day, ready for use by public vaccinators.

Performance of Vaccination.—Vaccine of clinical activity and bacteriological purity being thus produced, it becomes necessary for the purpose of efficient vaccination that it should be used directly or within two or three days, the operation being skilfully and thoroughly performed, with every possible attention to cleanliness, and with every practicable aseptic precaution. Non-recognition of the full importance of these details in the past may have contributed in some measure to the unpopularity of vaccination. But, in saying this, it is right to add that much more discredit has been brought on its prophylactic value by its very imperfect performance; that is to say, by private practitioners (not public vaccinators) being content with aiming at only producing one small vesicle. It is only too true that a very considerable part of the vaccination to-day is of this most unsatisfactory kind. On this and other points connected with the protective effects of vaccination, as to which there has been so much controversy, it is well to reproduce, in their own words, the conclusions arrived at by the Royal Commission in their Final Report in 1896, after their very prolonged investigation.

Paragraph 377 of the report (pp. 98-9), reads as follows:—

'We have not disregarded the arguments adduced for the purpose of showing that a belief in vaccination is unsupported by a just view of the facts. We have endeavoured to give full weight to them. Having done so, it has appeared to us impossible to resist the conclusion that vaccination has a protective effect in relation to small-pox. We think :

'1. That it diminishes the liability to be attacked by the disease.

'2. That it modifies the character of the disease, and renders it (a) less fatal and (b) of a milder or less severe type.

'3. That the protection it affords against attacks of the disease is greatest during the years immediately succeeding the operation of vaccination. It is impossible to fix with precision the length of this period of highest protection. Though not in all cases the same, if a period is to be fixed, it might, we think, fairly be said to cover in general a period of nine or ten years.

'4. That after the lapse of the period of highest protective potency, the efficacy of vaccination to protect against attack rapidly diminishes, but that it is still considerable in the next quinquennium, and possibly never altogether ceases.

'5. That its power to modify the character of the disease is also greatest in the period in which its power to protect from attack is greatest, but that its power thus to modify the disease does not diminish as rapidly as its protective influence against attacks, and its efficacy during the later periods of life to modify the disease is still very considerable.

'6. That re-vaccination restores the protection which lapse of time has diminished, but the evidence shows that this protection again diminishes, and that, to ensure the highest degree of protection which vaccination can give, the operation should be at intervals repeated.

'7. That the beneficial effects of vaccination are most experienced by those in whose case it has been most thorough. We think it may fairly be concluded, that where the vaccine matter is inserted in three or four places it is more effectual than when introduced into one or two places only; and that if the vaccination marks are of an area of half a square inch, they indicate a better state of protection than if their area be at all considerably below this.'

The object of the vaccinator should be to ensure the insertion of vaccine-matter in four separate places, so as to produce scars of the requisite size. The methods generally recommended are scarification by 'cross-hatching' or tattooing. The arm should be first washed with soap and warm water, and then carefully dried with a soft towel, gentle friction being employed. Vaccine is put on the surface of the arm, the skin being kept on the stretch by the fingers of the left hand, the operator scarifying through the vaccine, and being careful not to make his insertions too close together. An ordinary bleeding-lancet, the point of which has been slightly blunted, or a spear-pointed surgical needle, are the most suitable instruments. The lancet must, of course, be sterilised immediately before use. The advantage of the needle is that a new one can be employed on each occasion. Complicated instruments are inadvisable on account of the difficulty in keeping them clean.

In dealing with this part of the subject—viz. the performance of the operation—it remains to add a few words as to the age and state of health of the child at the time of vaccination.

The statutory period, after the expiration of which a parent having an unvaccinated child falls into default, has been extended from three to six months.

It is under ordinary circumstances a preliminary condition of the performance of vaccination that the child to be vaccinated should be healthy, and a careful examination to ascertain this is the first duty of the vaccinator. The child should not only be free from any acute febrile disease, but also from diarrhoea and from cutaneous diseases, especially those of the vesicular type. The states of constitution associated with herpes and eczema singularly interfere with the proper course of vaccination, and seem to be the most frequent causes of those spurious results of vaccination just described. They may both—especially intertrigo—without care, be overlooked; hence examination of the scalp, and of the folds of skin behind the ears, in the neck, and in the groins, is indispensable. Vaccination should also be postponed if erysipelas be prevalent in the neighbourhood in which the child is living, or if it has been recently exposed to the infection of measles or scarlatina. There is, however, a state of things under which these conditions must be disregarded—namely, when there may be *immediate* exposure to the infection of small-pox, as when an unvaccinated child is in a house in which the infection exists, or has come into direct contact with an infected person. Under such circumstances, it cannot be too strongly impressed that no age is too early for vaccination, and no state of health, except the presence of acute disease of a serious character, can be held to contra-

indicate it. Life then may depend on the promptitude with which the vaccination is done.

The Phenomena of Vaccination.—These are practically the same with the new vaccine as under the former system of arm-to-arm vaccination. Histologically there is no difference in the phenomena.

Course of Primary Vaccination.—When vaccine is inserted into the skin by puncture, or is applied to a small abraded surface of the skin of an unprotected person, no particular effect is noticeable till about the end of the second day, or early on the third day. By this time, if the vaccination be about to succeed, a slight papular elevation becomes perceptible. This by the fifth or sixth day has become a distinct vesicle of a bluish-white colour, with raised edge and central cup-like depression. By the eighth day (the day-week from that on which the lymph was inserted) it has attained its perfect growth; it is then plump, round, more decidedly pearl-coloured, and distended with clear lymph; its margin is firm and central depression very marked. On this day, or sometimes even by the end of the seventh day, a ring of inflammation, called the areola, begins to form about its base; and the vesicle and areola together continue to spread for the next two days. The areola is circular, and when fully developed has a diameter of from one to three inches, being then often attended by considerable hardness and swelling of the subjacent connective tissue. After the tenth day the areola begins to fade; and in two or three days more it has usually disappeared, along with any hardness or swelling which may have existed. With the decline of the areola the vesicle begins to dry in the centre; the lymph remaining in it becomes opaque and gradually concretes; and by the fourteenth or fifteenth day a hard brown scab is formed, which gradually contracts, dries, and blackens, and from the twentieth to the twenty-fifth day, but usually about the twenty-first day, falls off. There is then left a cicatrix, which is circular, somewhat depressed, foveated, sometimes radiating, and, with rare exceptions, permanent in after-life.

If the lymph has been inserted by two, three, or more punctures set near together about one spot, or by abrasion over a sufficient surface, two or more vesicles may arise at the spot; and in the course of their growth either form a large vesicle of a compound character, with but one central depression, or a crop of vesicles, generally coalescing, but each retaining its own central depression. These compound vesicles and crops are round, oval, or of irregular outline, according to the manner in which the cutis has been penetrated or exposed; and the shape of the resulting cicatrices varies accordingly.

Vaccination which has gone through the course above described is held to be protective against small-pox.

The constitutional symptoms attending these local phenomena are a rise of temperature, sometimes detectable by thermometer as early as the fourth day; more marked, but still often very slight, from the fifth to the seventh day; more obvious feverishness, with restlessness, and frequently derangement of the stomach and bowels, from the eighth to the tenth day—that is, during the stage of areola, subsiding as that subsides. The general symptoms are in most cases quite moderate, and often exceedingly slight. Occasionally, when the areola is at its height, swelling of the axillary glands may be

intense; and occasionally also at that period, in young children of full habit, especially in hot weather, an eruption of roseola (*Vaccinal roseola*) may occur, chiefly on the extremities; or a papular eruption (*Vaccinal lichen*), or a vesicular one—the vesicles, however, differing from vaccine-vesicles in being entirely free from central depression. The duration of any of these forms of eruption, when they do occur, is very transitory, usually not extending beyond a week, and very seldom indeed beyond the falling of the scab.

Deviations.—The above is a description of the regular course of vaccination. There may be some deviations in point of time, the development of the vesicle being somewhat retarded on the one hand or accelerated on the other. But if the phenomena are in all other respects regular, then variations in time do not, as far as known, affect the protective power of vaccination. It is, however, quite otherwise where the irregularity is in the course and character of the vesicle. Papules or vesicles which do not bear the character of the true vaccine-vesicle must be regarded with great suspicion, as not being worthy of reliance for protection against small-pox.

Complications.—The occurrence of complications in the course of primary vaccination should be very much diminished by the introduction of the new system. It is perhaps too early yet to judge of the effects of the substitution of glycerinated vaccine in place of the ‘arm-to-arm’ lymph, and other changes recently introduced into our national system of public vaccination. The present writer has officially seen thousands of vaccinations under the old system, but cannot as yet claim sufficient experience of the new system to make a fair comparison. But the accounts generally given by able and experienced observers and vaccinators are most favourable. We are, indeed, led to hope that the rare but serious risks of vaccination, such as erysipelas and other infective diseases, are things of the past, and that even those troublesome (though to a certain extent necessary) complications, perhaps rather lightly referred to as ‘sore arms,’ are likely to be very sensibly diminished under the new system.

Re-vaccination.—In some persons the regular phenomena of vaccination can only be produced once in the lifetime. But this is not always the case, and vesicles may be produced by a second vaccination not distinguishable in their appearance from primary vesicles, though usually having a smaller and more transitory areola, and leaving only a small cicatrix. Much the most frequently the result of that process is the production of a spurious papule or acuminate vesicle, with hard, irregular areola, reaching its height by the fifth or sixth day, and having by the eighth day an imperfect scab, which soon falls. There is often much itching and more serious local irritation; and the constitutional symptoms are out of all proportion more frequent after re-vaccination than after primary vaccination.

The question whether re-vaccination should be systematically carried out among the population generally, as in the case of primary vaccination, is one on which opinions of responsible officials are divided at the present day. The conclusions of the Royal Commission have already been quoted. The great value of vaccination, and the immense boon it has been to the community, were recognised by all the Commissioners except two. In legislating upon

the subject Parliament has shown a desire to promote the adoption of vaccination and to diminish as far as practicable the number of the unvaccinated infant and child-population, which if allowed to assume very large proportions would constitute a serious menace to the public health. For it must be clearly understood that no medical organisation, however complete, and no system of isolation, sanitation, and general preparedness for dealing with outbreaks, could be safely trusted to in the absence of systematic primary vaccination. Every medical officer of health, however strongly he might believe in the efficiency of the measures which he immediately directs, would shudder at the thought of a large population such as that of Gloucester or Leicester, for example, with organisations of such varying efficiency, being in a largely unprotected state and exposed to the virulent infection of small-pox. There can be no two opinions among such officials as to the desirability of maintaining a well-vaccinated population. And here it may be remarked that by a well-vaccinated population is meant a population with a proportion of infants 'accounted for' of at least 90 per cent. Moreover, it should be stipulated that vaccination is performed with a degree of efficiency nearly approaching that which is absolutely required of public vaccinators. The certificate of vaccination should, in fact, only be given where a defined standard has been attained. Some legislative or administrative order is necessary to secure this end. If at the same time the use of the vaccine manufactured by Government were made obligatory in all vaccinations performed by private practitioners, as well as public vaccinators, much would be done to strengthen our defences in respect of epidemic small-pox. But it is altogether another matter to advocate, as some eminent authorities on the subject have done of late, the re-vaccination of the whole population at stated periods. The inconvenience, to say the least, of the process has to be considered in relation to the risks of small-pox. In a well-vaccinated population small-pox would never be allowed to become generally epidemic if the sanitary organisation and administration were what it should be. The present writer has often directed attention to the achievements of local sanitary authorities in the way of cutting short outbreaks of small-pox and preventing epidemics. One most essential part of this system is that, concurrently with the isolation of the sick, the re-vaccination of all persons brought within the sphere and risk of small-pox is immediately attended to. The advantages of the plan, which only became possible when the system of notification came fully into operation, are obvious. The present writer desires to point out that experience has been steadily accumulating of late years which strongly confirms this view. Re-vaccination is of course an essential precaution for this purpose. It should be immediately resorted to by persons exposed to the risk of infection, and it should be part of the business of every local sanitary authority to warn them of their danger, to provide means of safety, and to enforce as far as possible the adoption of re-vaccination and other measures of precaution. With improvements in the practice of vaccination such as science has made possible, and with steadily improving local sanitary administration, we may reasonably hope that every person in any way brought within the sphere and risk of small-pox will be efficiently protected. The writer strongly

deprecates indiscriminate re-vaccination undertaken because of the fears of the individual, rather than in accordance with the judgment of the practitioner.

Such a scheme ought, in the opinion of the present writer, to be quite practicable in England. The discomfort entailed by the sore arm and a certain amount of constitutional disturbance is absolutely necessary in cases where re-vaccination is specially indicated; but the inconvenience or loss of wages would be confined to a comparatively very small number of persons—at any rate, only a fraction of those who would be incommoded by the larger and, as the present writer thinks, unnecessary scheme of general re-vaccination.

The above remarks apply to populations living under English systems of sanitary and public-health administration. In the case of persons going to the East, or to countries where small-pox is a prevailing disease, subject to no such control as that which has been established in Jenner's country, re-vaccination is recommended to all except young children with good primary vaccination-marks, or persons who have undoubtedly been already successfully re-vaccinated in adult life. EDWARD COX SEATON.

VAGINA, Diseases of.—SYNON.: Fr. *Maladies du Vagin*; Ger. *Krankheiten der Scheide*.

The vagina frequently participates in the morbid processes which affect the uterus and other neighbouring organs. The vaginal affection is then a matter of minor importance, and may not call for special recognition. But this canal is often the seat of independent diseases, the more important of which will now be considered.

1. Atresia.—Imperforate vagina is met with as a congenital or acquired malformation. *Congenital atresia* may occur alone; or it may be complicated with absence, imperfect development, or closure of the orifice of the uterus. Occasionally the vagina is bifid, and the atresia affects only one side, in which the menstrual fluid of the corresponding horn of the uterus may accumulate, producing the condition described as *hematocolpos lateralis*. The occlusion may, first, affect the organ in its entire length; or, secondly, for a half or third of its length, most frequently towards the lower extremity; or, thirdly, it may be simply membranous, as from a too complete hymen. The congenital atresia is usually complete, so that the passage to and from the uterus is perfectly occluded. *Acquired or accidental atresia*, on the other hand, may present any degree of constriction, from a slight and partial narrowing of the canal up to its total obliteration. We find it resulting, first, from sloughing after labour, when it is frequently complicated with vesical or rectal fistulæ; secondly, from sloughing after fever; thirdly, from chronic inflammations and ulcerations; fourthly, from repeated applications of caustics.

SYMPTOMS AND DIAGNOSIS.—There are three stages at which atresia of the vagina may betray itself by symptoms. First, after puberty has set in, the patient has the usual indications that ovulation is taking place, but she suffers from *amenorrhœa*. The uterine hæmorrhage is taking place; but the extravasated blood is detained above the seat of the occlusion, and does not appear externally. Secondly, in some women *dyspareunia* (impossibility of copulation) after marriage leads to the examination which discovers the obstruction. Thirdly, in the acquired variety the difficulty is

often only discovered in consequence of the *dystocia* that results from the resistance offered to the advance of the foetal head by the constriction of the canal. The physical examination may discover, first, on abdominal palpation, a swelling in the hypogastric or inguinal region, due to the accumulation of menstrual fluid in the upper parts of the sexual canals. Secondly, on vaginal exploration being attempted, either the finger is soon arrested within the labia, or a bulging, fluctuating pouch is felt. Thirdly, we then examine *per rectum* or *per vesicam* with sound or finger, or through these cavities simultaneously, combining the exploration by means of the fingers of one hand through the available openings in the pelvic floor, with pressure and palpation with the other hand above the pelvic brim. The exact seat and extent of the occlusion can thus be detected, and at the same time an estimate can be formed of the amount of accumulation that may have taken place above it.

PROGNOSIS.—(1) Where the obstructing membrane is thin, it may give way at a menstrual period, or undergo a gangrenous process. (2) The sac formed by the dilated uterine or Fallopian cavities may burst, and pour its contents into the peritoneal cavity. (3) In a considerable proportion of cases early menopause comes on, and lessens the risk. (4) Patients who suffer from atresia vaginæ, even when they have been relieved by operative measures, are said to show a tendency to die of consumption.

TREATMENT.—In view of the dangers associated with the accumulation of menstrual fluid in the genital canals, the indication usually becomes very clear for its evacuation by perforating the obstructing tissues. But this operation has often been followed by disastrous results. Not to speak of the risks of wounding the bladder or rectum, or of setting up pelvic cellulitis, in cases where there is total absence of the vagina, and a canal needs to be tunnelled to the uterus, emptying of a hæmatometra and hæmatocolpos has been followed by death, sometimes from septicæmia, sometimes from bursting of the sac above when pressure is made on it; or dangerous inflammation has been set up in the pelvic organs. Care must be taken never to empty the sac by pressure from above, but by washing it out with a warm antiseptic fluid through an opening, which must be made under antiseptic precautions. Where the atresia has been extensive, there is a strong tendency to its repeated closure. This must be averted by making the patient wear a glass or vulcanite tube filled with cotton wadding soaked in carbolised oil, through which the uterine cavity can be occasionally washed out until it has collapsed. This must be worn for some months, until the vaginal canal has fully cicatrised around it.

2. Displacements.—When the perinæum has become relaxed or lacerated, hernia of the pelvic contents is apt to occur to a greater or less extent. The descent of the uterus in such cases has commonly been regarded as their most important feature, and they have generally been described as *prolapsus uteri*. When the protrusion or herniation, however, is complete, the walls of the hernial sac are formed chiefly by the walls of the vagina, and we get the most extensive displacement, or *inversion*, of the vagina. The symptoms and treatment of this inversion belong to the history of uterine displacements. But we may have displacement of one or other of the vaginal walls as an

independent mischief, or as the most prominent disturbance, in a case where the retentive power of the pelvic floor is impaired. If the anterior wall of the vagina descend, it carries with it the back wall of the bladder—*cystocele*. If the posterior vaginal wall descend, it brings with it the anterior wall of the rectum—*rectocele*.

TREATMENT.—The treatment of these conditions is either palliative or radical. The palliative treatment is effected by the use of astringent injections, and the application of vaginal pessaries—such as the Hodge pessary with crossbars towards its lower end. The radical treatment implies a plastic operation for repair of the relaxed or ruptured perinæum, or for producing a contraction in the displaced vaginal wall.

3. Foreign Bodies.—The vaginal canal is sometimes found occupied by foreign bodies. Every gynaecologist has met with cases where pessaries have been left in for years, until by their presence and pressure they began to set up ulcerative processes in the vaginal walls. Introduced at first with a useful object, and producing for a time a beneficial effect, their presence has sometimes been forgotten, until the discharges they excited have recalled attention to them. But bodies of quite another kind are sometimes met with, usually introduced by patients with onanistic propensities. Corks, pieces of wood, pomade-pots, fir-tops, dram-glasses, &c., have been met with in such cases. Portions of glass specula and of glass syringes have sometimes broken off in the hands of practitioners or patients. Ascarides and other parasites seem occasionally to make their way from the anus into the vagina.

Some of these foreign bodies are very easy of removal. Others are a source of great difficulty—those more especially which have become embedded to some extent in the vaginal walls. Thus it may become necessary to anaesthetise the patient, to lay hold of the foreign body with a polypus-forceps or vulsellum, and to detach it with the fingers or with a knife from the tissues that have granulated round it, before it can be withdrawn.

4. Inflammation.—**SYNON.**: *Colpitis*; *Vaginitis*.—Apart from gonorrhoeal inflammation of the vagina, we may have colpitis of a non-specific character, either as a primary disease, originating in itself, or spreading to it from the neighbouring structures. The causes of inflammation commencing in the vagina are found in chills (puerperal or menstrual); injuries (as from rude use of obstetric and gynecological instruments); prolonged presence of pessaries; excessive coition; irritants (as in cases where nitrate of silver applied to the cervix has acted on the vaginal mucosa); neoplasms; and some of the fevers. In elderly women we meet with a *colpitis senilis* or *vetularum* (Ruge), the cause of which is not easily traceable, in which the mucous membrane, especially of the upper part of the vaginal tube, sheds its epithelial covering in patches, becomes studded here and there with papillary granulations, and shows a tendency to cicatricial contractions. In other women the colpitis in its acute forms is found as a catarrh of the mucosa, attended by swelling, rapid desquamation of epithelial cells, and exudation of a serous fluid, which, mixed with the granular cells, produces a milky discharge. In the chronic forms the discharge becomes more creamy or purulent as the exudation is less copious, and there is more exfoliation of

degenerated and often unripe epithelial cells; and then the surface is often thickly strewn with red papillæ, over which the epithelium is almost destroyed. The symptoms are local discomfort, and leucorrhœal discharges of various kinds; and the diagnosis is made by examination of the discharge, and exposure of the affected surface by means of the speculum.

TREATMENT.—The treatment in the acute stage is directed to keeping the part at rest, and using sedative injections. In the chronic forms the use of the injection or douche must be steadily persevered in. The canal must be washed out with a stream of hot water, in the last pint of which alum or some other astringent has been dissolved. Where an acute process is likely to become chronic, or in the chronic forms attended by granulations on the surface, it is well to apply with a mop or brush a solution of nitrate of silver containing half a drachm to the ounce. Frequently the use of pessaries of oxide of zinc, bismuth, or iodide of lead proves useful in cases where patients have difficulty in using the syringe or douche. In *colpitis senilis*, the distress which the patient feels from the irritating discharge is best relieved, and the unhealthy surface is most speedily brought to heal, by the application of bismuth-powder, through a speculum or other tube, to the mucous membrane, after washing or wiping away the secretion; or an injection of boric acid with cocaine may be used.

5. Injuries.—It is principally in connection with labour that the vaginal walls are liable to be injured. Not only in operative cases where the walls may get bruised and torn, or in tedious labours where the walls are so long compressed that they may afterwards slough and become the seat of fistulæ, but even in ordinary cases, the vaginal mucous membrane is almost always fissured, or torn more or less deeply, at its lower extremity. These lacerations are mainly important because of their liability to become the channels through which septic matters are absorbed in the puerperal patient. In every case where there is a chance of infection, the canal should be syringed with a solution of creolin, or perchloride of mercury, or carbolic acid; and it is a further safe precaution to keep the raw surface dusted with a powder of several grains of starch combined with one grain of salicylic acid, or with iodoform and bismuth.

6. Neoplasms.—*Fibromata* affect most frequently the anterior wall of the vagina. They may be either sessile or pedunculated. They do not give rise to much distress, except from their pressure on the bladder, or from their protruding through the vulva. Extirpation affords the only cure, and in carrying out the operation care has to be taken to avoid injuring the bladder or urethra when they are seated anteriorly, and the rectum when they spring from the posterior wall. *Sarcomata*, round-celled or spindle-celled, have been in some few cases found springing from the vaginal walls. They have sometimes been described as growing from the submucous tissue; and the writer has seen them in two instances develop from the submucous tissue of the back wall. In other cases the growths arose from the mucous membrane. Early extirpation is indicated. These growths tend to recur. *Carcinoma*, more markedly even than the other neoplasms, is oftener a secondary than a primary affection in the vaginal walls. It may, however, originate in the vagina itself, running a course at least as rapid

as in the uterus, and usually leading early to infiltration of the inguinal glands. It only admits of palliative treatment. *Cystomata*, like the simple solid tumours, are most frequent on the anterior wall, and arise in many cases from accumulation of fluid in portions of the duct of Gaertner that have remained unobliterated. The contents are usually pale, and, where they have attained the size of a walnut, watery. In the smaller cysts the contents are more viscid. Some seem to result from cavities into which blood has been extravasated, and then the contents are brownish. They must be freely evacuated, and iodine or nitrate of silver applied to their lining membrane; otherwise they are apt to refill.

7. Vaginismus.—Under this designation has been described an affection of the vaginal orifice which is not infrequent among recently married women, and which utterly unfits the subjects of it for enduring coition. In some of them the hymen is unruptured, and there are excoriations at its base, towards the navicular fossa. More frequently the hymen has been infringed, but the lacerations have not healed, or fissures have formed at the roots of the hymeneal flaps, and a chronic inflammation is set up in the tissues, which renders them exquisitely sensitive. In such a condition any touch, even of the finger, causes intense pain, and when an attempt is made to pass through the orifice, the constrictor vaginæ and the lower fibres of the levator ani are thrown into a tenesmic state, which prolongs the suffering. The leading symptom is the distressing dyspareunia; and the diagnosis is made by the touch of the finger, supplemented by inspection. An examination can often be effected only when the patient is placed under an anæsthetic.

TREATMENT.—Treatment of a palliative kind may be attempted, making the patient use sitz-baths and apply emollients and sedatives. But it is far more satisfactory to have recourse to radical measures. The patient being under the influence of chloroform, the fragments of the hymen should be pared or clipped off. An incision should be made on each side of the vaginal opening towards its posterior aspect, running in a direction outwards and backwards, and passing through the whole thickness of the mucous membrane and some portion of the thickness of the constrictor muscle. The points of four fingers of the hand, gathered together and well greased, should then be passed through the dilated opening, so as fully to distend it, and produce complete relaxation of the sphincter. With due care the bleeding is trifling; the wound heals kindly; and undue contraction is prevented by making the patient wear for half an hour or longer, twice a day, a thick vaginal bougie. For a time the introduction of the bougie is resented, but the pain passes off when it is kept in position; and after a few days its passage ceases to be painful. This operation can be carried out with a very confident expectation that the dyspareunia will disappear; and if no other complication be present, the probability is that conception will ensue.

ALEXANDER RUSSELL SIMPSON.

VALENCIA, on the East Coast of Spain.—A dry, variable, mild, winter climate; and important sea-bathing station. Mean winter temperature, 57° F. Prevailing winds E. (moist), W. and S.W. (rough). See CLIMATE, Treatment of Disease by.

VALS, in *Ardèche*, *France*. — Alkaline waters. See MINERAL WATERS.

VARICELLA (dim. of *Variola*).—A synonym for chicken-pox. See CHICKEN-POX.

VARICOCELE (*varix*, a dilated vein; and *κῆλη*, a tumour).—**SYNON.** : Fr. *Varicocèle*; Ger. *Krampfaderbruch*.

DEFINITION.—A dilated, elongated, and tortuous condition of the veins of the spermatic cord, due either to increased pressure within the vessels, or to diminished resistance in the walls of the vessels and the surrounding structures, or to overgrowth of Wolffian veins.

PATHOLOGY.—The testicle, probably, like other glands, receives supplies of blood varying with its activity; and the veins are numerous and tortuous, freely anastomose, are liable to intermittent compression in their passage through the inguinal canal from muscular contraction, and terminate on each side in a single vein, which, like the companion artery, is remarkable for its great length and small size. These conditions are favourable to the production of dilatation and varicosity. Diminished resistance in the walls of the vessels and surrounding structures is, however, probably the chief cause. Varicocele is so much more frequent in the left than in the right spermatic cord, and when present in both is so much larger in the left, that the inferior muscular development of the left side of the body from predominant use of the right is very possibly a predisposing cause. The termination of the left spermatic vein at right angles in the renal vein, as compared with the termination of the right vein at an acute angle in the inferior vena cava, and the relation of the left vein to the sigmoid flexure, have been suggested as possible exciting causes; but their influence, if any, must be slight. The epididymis and organ of Giralde's are developed from a highly vascular organ, the Wolffian body. It is probable that varicocele is sometimes dependent upon the non-disappearance and overgrowth of persistent Wolffian veins. Not infrequently the veins of the spermatic cord become varicose where they lie in the inguinal canal (*inguinal varicocele*). In the absence of a scrotal varicocele the tumour caused by the dilated veins is easily mistaken for an incomplete inguinal hernia.

SYMPTOMS.—Varicocele is generally painless, but is sometimes associated with neuralgia or hyperæsthesia of the testicle. The subjects of it sometimes complain of a feeling of weight or uneasiness in the part after standing or prolonged exercise; and in extreme cases, when the scrotum is very much relaxed, labouring men have found it a mechanical hindrance in their work. In the tropics a varicocele is nearly always painful, and the pain is aggravated by the fatigue of marching. The testicle is in many cases normal, but is sometimes soft and even atrophied, but these conditions of the testicle are probably the result of other causes, and not of the varicocele. In such cases the patient is generally very hypochondriacal. An *inguinal varicocele* may cause pain, appears when the patient stands up, disappears when he lies down, and has an impulse in coughing. But a scrotal varicocele is often present, and sometimes the dilated veins can be felt when the cord is pulled down.

DIAGNOSIS.—The diagnosis of scrotal varicocele is easy. The veins can be readily felt, and have

been aptly compared to a bag filled with worms. The position of the varicocele is usually above and behind the testicle; but it may vary and the tumour lie below the testis, under which circumstance it is apt to be overlooked. Varicocele may complicate inguinal hernia and hydrocele.

TREATMENT.—In slight cases of varicocele no treatment is required. The use of a light truss, with pressure sufficient to take off the weight of the superincumbent column of blood from the spermatic veins, without interfering with the current of blood through the spermatic artery, has been recommended with advantage. A well-fitting suspensory bandage, cold bathing, avoidance of constipation, and tonics, will suffice for most cases; but if operative interference be requisite, the writer (C. B. L.) has found excision of the veins most satisfactory. The bundles of veins should be transfixed, and tied so as to avoid slipping of the ligature. The latter may consist of silk or catgut. It is safest to drain the loose tissues of the scrotum for the first twenty-four hours. Subcutaneous ligature and division of the veins have been recommended; but the result is always uncertain, and sometimes unsuccessful. The treatment of inguinal varicocele consists in opening the inguinal canal, after which the veins can be isolated, transfixed, and tied, and the canal closed again.

JEREMIAH MCCARTHY.
C. B. LOCKWOOD.

'VARICOSE' GLANDS.—See FILARIASIS, p. 545.

VARICOSE VEINS.—See VEINS, Diseases of; and VARICOCELE.

VARIOLA (*varius*, spotted).—A synonym for small-pox. See SMALL-POX.

VARIOLOID (*variola*, small-pox; and *εἶδος*, form).—This term has been applied to a mild form of variola, the disease being modified by previous vaccination or inoculation. See SMALL-POX.

VEGETATIONS.—In modern pathology this term is generally applied to growths and deposits connected with the valves of the heart (see HEART, Inflammation of). The name is also given to excessive granulations on wounds, and to warty growths in any situation.

VEINS, Diseases of.—**SYNON.** : Fr. *Maladies des Veines*; Ger. *Krankheiten der Adern*.

The diseases of veins will be described in the following order: (1) Inflammation; (2) Varix; (3) Hypertrophy; (4) Atrophy; (5) Degenerations; (6) Phleboliths; (7) Wounds; (8) Air in Veins; (9) Parasites; and (10) New-Growths. Neither thrombosis nor pyæmia, except in so far as they affect the vessel-walls, will be discussed here. See PYÆMIA; THROMBOSIS; and VEINS, Entrance of Air into.

1. Inflammation.—**SYNON.** : Phlebitis; Fr. *Phlébite*; Ger. *Venenentzündung*.

DEFINITION.—Inflammation of the coats of a vein.

CLASSIFICATION.—Cases of phlebitis may be classified: (1) according to the *intensity* of the inflammation—acute, subacute, or chronic; (2) according to the *result* of the inflammation—whether the formation of new tissue (plastic) or suppuration (suppurative); (3) according to the

layer of the venous wall primarily or chiefly affected—endo-, meso-, and exo-, to which may be added as closely associated peri-phlebitis; or (4) according to the cause of the phlebitis, e.g. septic. Practically, however, we may discuss the subject under two heads, namely, *endophlebitis* and *periphlebitis*.

ÆTIOLOGY.—The following are more or less well-established causes of phlebitis:—

Mechanical injury.—This frequently, but not necessarily, leads to the formation of a thrombus—a further source of mechanical irritation. A wound of a vein is generally lost sight of in the injury of surrounding parts; but the degree of inflammation resulting in the vein-wall, like that in the wound at large, will depend upon whether the injury is or is not accompanied by infection with pyogenic organisms, or whether these subsequently gain access to the injured spot. The effect of chronic strain is shown in the thickening and sclerosis of the wall of a varicose vein.

The formation of a thrombus.—For the causes of this see THROMBOSIS. Here it will suffice to say that a certain unknown degree of alteration, by injury or disease, of the endothelial layer of a vein will induce the formation of a clot, and that this is favoured by slowness of the circulation and by some blood-states. The clot is an irritant, slight or intense. When the thrombus quickly clears up, and the vein becomes patent, we may presume that the changes in the vein-wall have been but slight; when the thrombus is the seat of infective softening, the wall of the vein may suppurate or necrose. Between these two extremes come the changes accompanying the 'organisation of a clot,' and the conversion of it and the vein-wall into a mass of fibrous tissue.

The existence of an inflammation in the tissues through which the vein passes.—When a periphlebitis exists, whether acute or chronic, it tends to spread to the wall of the vein, involving first the adventitia, and extending inwards until, perhaps, sufficient change of the intima is produced to cause thrombosis. The intensity of the phlebitis varies with that of the surrounding inflammation, but it does not follow that a venous wall exposed in an abscess will become the seat of abscess, nor even that it will be thrombosed, for the vein is not one with the tissue in which it lies. Still, it is not difficult for organisms in tissues round about a vein to invade the wall of the vein, to excite in it a process similar to that going on around it, and to penetrate to any thrombus which may form within it, causing it to undergo infective softening. In organs and parts which are the seats of sclerosis the veins undergo fibroid thickening, and contraction of their lumina. Gummatus and tubercular infiltrations of veins passing through parts which are the seats of late syphilitic or tubercular lesions occur; and softening of a tubercular lesion of a vein-wall may lead to discharge of the contents of the focus into the circulation, and to a more or less widespread outbreak of tuberculosis.

But *syphilis* and *tuberculosis* may affect the walls of veins directly, giving rise to small gummata and discrete tubercles in the walls. The former lead to changes similar to those in syphilitic arteritis. Either syphilitic or tubercular inflammation may lead to sclerosis of the wall with contraction of the lumen, and, very likely, thrombosis.

A hyperplastic phlebitis is described, correspond-

ing to endarteritis obliterans, but it is even less common, and the subendothelial thickenings are much less marked. The cause is unknown.

There is no *a priori* reason why acute inflammation excited by the presence of organisms should not start in the external and middle coats of veins, which are vascular; but there seems to be no pathological evidence of its occurrence. On the other hand, it seems probable that blood-borne germs do settle in the intima of veins in acute necrosis and in pyæmia, and induce such changes as lead to thrombosis. Again, if the changes in acute rheumatism in the non-vascular cardiac valves are not always due to organisms settling upon them, but are caused by some irritant in the blood, the vessels may be similarly affected; there is, however, nothing but theory to support this view. The recurrent phlebitis to which the gouty are liable may possibly own a pathology of this kind.

To sum up: the great causes of phlebitis are injury, the irritation of a thrombus, and infection from surrounding tissues. The settlement of germs in the endothelium from the blood seems probable.

MORBID ANATOMY AND HISTOLOGY.—When a thrombus forms, contraction of the vessel-wall and uniform reddish staining of the intima occur, exactly corresponding to the clot. The inner wall presents longitudinal folds, easily obliterated by distension. More or less injection of the vasa vasorum, more or less fluid and cellular infiltration, and consequent swelling and softening of the vein-wall, ensue, in proportion as the clot is more or less irritant. If the clot soon disappears, return to the normal is easy. If the clot organises, contraction of the vein upon the clot goes on; the clot is freely infiltrated by leucocytes from the vasa vasorum, and proportionately decolourised; new vessels form; fibrous tissue develops; and wall and clot are converted into a cord of connective tissue, in which the structure of the vein may for a long time remain more or less distinct. If the clot undergoes infective softening or putrefaction, the swelling and softening of the wall reach their greatest height; hæmorrhagic points and small collections of pus may be visible to the naked eye; under the microscope the wall is densely infiltrated with white and red corpuscles; and, finally, loss of fine structure and imperfect staining may indicate death of the part. The thrombus which has undergone infective softening may thus give rise to an abscess about the vein, and may burst through the venous wall—the most favourable termination; for it may ward off rupture of the central end of the clot into the circulation, and consequent septic embolism. These changes are well seen in cases of pyæmia, and in thrombosed veins starting from septic ulcers or wounds.

Exactly the same state of matters may be produced by a septic inflammation extending from without to the venous wall, and leading to thrombosis and infective softening of the thrombus, e.g. in thrombosis of the petrosal and lateral sinuses in middle-ear disease.

SYMPTOMS.—Thrombosis of a vein—generally of the leg—is usually spoken of as 'phlebitis.' The patient complains of pain in the part, either dull aching or sharp shooting; and on examination one feels a firm rounded cord, if the affected vein is superficial. The part is somewhat tender, there is usually slight œdema around the cord, and the

skin over it may be slightly or markedly reddened; these symptoms being due, apparently, to thrombosis causing a phlebitis and periphlebitis. Some, however, believe that these symptoms may arise from phlebitis without thrombosis, an inflammation spreading outwards from the wall of the vein. In the case of deep veins of no great size there may be no symptoms at all to indicate the occurrence of simple thrombosis, and it is evident from what we see in superficial veins that thrombi may cause very little irritation. When the vein thrombosed is a main one, more or less widespread oedema results, but this is a symptom of thrombosis, not of phlebitis.

When the thrombus undergoes infective softening, the vein-wall and the tissues for some distance round about it become the seat of an intense inflammation, the signs of which are obvious in the case of superficial veins; and in deep veins, like the jugular, fullness and tenderness along its course may be discovered. On the other hand, nothing used to be commoner than the occurrence of pyæmia, without any symptoms to guide the surgeon to the vein which was occupied by the breaking-down clot, unless a characteristic oedema was present.

No fresh symptoms accompany the spread of inflammation to the wall of a vein passing through a focus of inflammation, unless, again, thrombosis of it occurs and induces oedema.

Formerly, when almost all wounds suppurated, the thrombi which formed in veins, cut across in operations, were frequently infected with pyogenic and septic organisms, and the veins of bones which did not collapse on section were more liable than those of soft parts to infection. The result of the infection was sometimes a continued thrombosis of the vein, with infective softening of the clot, and thus arose a progressive phlebitis and a progressive inflammation of the tissues about the inflamed vein which, perhaps, ended in abscess. On the other hand, a very similar clinical result was produced by a septic inflammation of the lymphatics around the vein, leading to abscess in their course and, perhaps, to phlebitis by extension, and thrombosis with infective softening. These two conditions, forerunners of pyæmia, went by the name of *suppurative phlebitis*, and ranked high among the septic diseases of wounds.

PROGNOSIS.—In a simple phlebitis, not connected with a septic wound or an inflamed mucous membrane, the prognosis is good, as regards life. Serious embolism is the danger, but with ordinary care the probability is that the clot will completely disappear, and the vein become again pervious, or that it will become organised, and the vessels of the new connective tissue will establish a more or less complete communication between the parts above and below the obstruction. Thus oedema will be more or less completely removed. In infective phlebitis and thrombosis the prognosis is almost as serious as in former days.

TREATMENT.—In all forms of phlebitis we must ensure absolute rest by confining the patient to bed, and by fixing the whole limb between sand-bags, or by means of splints. This is essential to procure diminution of pain, and to lessen the chance of breaking off a fragment of the clot, whereby fatal embolism might be caused. Glycerine and belladonna smeared on the part, and hot fomentations or linseed poultices, are by far the most agreeable

and soothing application. When abscesses form, they should be incised and dressed with fomentations wrung out of hot perchloride-of-mercury lotion (1 : 2000). Any oedema or general thickening that may remain must be combated by Martin's elastic bandages and regular massage, after all danger of embolism is over. Medicines, except those given to support the patient's strength, such as cinchona bark, ammonia and the like, are not indicated. Opium is given only to allay pain, and the less the drug is used for that purpose the better. The treatment of infective thrombosis and phlebitis is to be conducted at first on the same principles; the further treatment is discussed under PYÆMIA. See PYÆMIA; and THROMBOSIS.

2. Varicose Veins.—SYNON.: Varix; Phlebectasis; Fr. *Varice*; Ger. *Krampfadern*.

DEFINITION.—Veins which are dilated out of proportion to the quantity of blood which should pass through them; wherefore the current in them is slowed.

ÆTIOLOGY.—It is conceivable (1) that the wall of a vein may yield under normal intravenous pressure; (2) that it may be dilated by increased intravenous pressure; or (3) that it may be dragged open. The last cause is probably at work when dilated veins are found traversing cicatricial tissue—e.g. in an organising thrombus—but it is of little importance as compared with the others mentioned.

(1) Although varicose veins are not inherited, the tendency to them appears to be hereditary. The walls of any given vein vary markedly in strength in different individuals, and at different spots in the same individual. Sabourroff has demonstrated these variations in strength and thickness of wall for the saphenous system. Weakness of venous walls may probably be acquired. Although we have no very certain knowledge about it, it is probable that from impaired circulation in a part malnutrition and degeneration of the walls of the veins in it will result, and that this degeneration will chiefly affect the contractile element, the loss of which is not compensated by increase in the inelastic, non-contractile fibrous element. Again, small varicose veins are common on mucosæ which are the seats of chronic catarrh, but their ætiology is complex. It is well known that under conditions of fatigue and exposure to heat, especially in air containing much moisture, the superficial veins dilate and form prominent objects on the surface; also, that in some persons this effect of 'relaxing' conditions is felt much sooner than in others. Thus a temporary weakness of the venous walls is produced; and if the conditions of life bring about this effect frequently, normal intravenous pressure may lead to permanent dilatation of veins—those which are weakest congenitally or owing to degeneration of muscle yielding first.

The 'normal intravenous pressure' is the sum of or difference between the driving force of the heart, the aspirating force of the thorax, and gravity. These may coincide in their action, or gravity may oppose the other two, and does so especially in the lower limbs and lower parts of the trunk, where the veins are thicker-walled in proportion to the strain which they have to bear. The effect of gravity on a vein-wall is proportionate to the height of the column of blood in the vein, and is therefore greater in the lower limbs of tall than of short people; its effect is constant as long as the height of the column of blood in the vein and the lumen of the vein are unaltered.

The element of *time* is a most important one in considering the dilating action of the normal pressure. No tissue in the body is able to bear, continuously or repeatedly, for long periods the maximum strain which it is capable of bearing without at once yielding. The more perfectly contractile, the more perfectly elastic it is, the longer will it withstand such trials; but, sooner or later, it will fail to recover perfectly—that is, permanent dilatation will begin. The veins are neither highly muscular nor elastic, yet they may recover from considerable and repeated dilatation, lasting even for months, as we see frequently in childbearing women.

The withdrawal of support which is usual may lead to dilatation of veins under normal pressure; thus a lax sphincter may encourage the development of piles.

(2) That heart- and lung-disease may cause accumulation of blood in the veins, and that these, especially the abdominal veins, dilate to accommodate it is certain; but such general obstructions to the venous return are not causes of varix. There seems some reason to doubt the correctness of the view, held by many, that cirrhosis of the liver is a direct cause of piles. When a main venous trunk becomes thrombosed or is compressed by the gravid uterus, full rectum, aneurysm, tumour, &c., dilatation of its tributaries and œdema may result; or a collateral circulation may open up through neighbouring veins, the walls of which may simply hypertrophy; or certain veins may become varicose. As Sabouroff says, the vein behaves in response to increased pressure like the bladder or heart: at first the muscle-cells hypertrophy, and then, if new vasa vasorum are developed and fuller nourishment is granted, the adventitia increases in strength; but if nourishment fails, the vein atrophies and dilates progressively. In aneurysmal varix and varicose aneurysm intravenous pressure approximates to arterial; the veins dilate and thicken greatly. It is believed by many that frequent and powerful muscular contractions, and the accompanying great afflux of blood to the muscles, lead to the throwing into the veins of quantities of blood larger than they can accommodate, and thus to their permanent dilatation. Even the effect of long standing on the leg-veins has been explained thus, the calf-muscles being specially active; this, at least, seems very doubtful. Walking, short of excessive fatigue, probably promotes the nourishment of all tissues in the leg; but when it and such exercises as cycling are carried to excess—that is, to great fatigue—they may, perhaps, lead to dilatation of veins.

ANATOMICAL CHARACTERS.—The principal seats of varix are the lower extremities; the trunk, when collateral circulation is set up as the result of blocking of the venous trunks; the rectum (*see HÆMORRHOIDS*); the spermatic plexus (*see VARICOCELE*); and the ovarian veins. But almost all veins have occasionally been found varicose—large and small, deep and superficial, a single vein or venule, or almost all in the body.

The following changes occur in the development of a varicose vein:—

(a) Simple dilatation of a vein occurs when unusual strain is thrown on its walls.

(b) If the strain endures, and increased blood-supply be sent to the vein-wall, hypertrophy of the muscle-cells in the middle coat, and hyperplasia of the connective tissue in all the coats, ensues. So far the vein is merely hypertrophied in proportion to the demands made upon it.

(c) Should the excessive strain continue, the vessel yields in all directions, becoming wider and longer, but not regularly; thus it becomes tortuous and irregular in its outline and calibre. Its wall is abnormally thick at some spots, very thin at others, especially in the neighbourhood of valves and towards the free surface.

The valves, too, become hypertrophied, and for a time perform their function; but by-and-by they fail to span the widening stream, and waste to fibrous cords. In the sac-like or spindle-shaped dilatations behind the valves the blood frequently coagulates, and the thrombus may remain small and parietal, occasionally calcifying (phlebolith), or may grow and become obstructive.

(d) A sac-like dilatation of the vein, as it approaches the surface, may by its pressure cause thinning of the superficial tissues; and these, together with its own walls, may finally rupture.

(e) The tissues round a varicose vein may appear normal; they are frequently slightly œdematous, or hard and fibroid; and perhaps there is evidence of shrinking of the new fibrous tissue. In this dense tissue the veins seem to lie in definite channels. The explanation is that the circulation is slow in proportion to the dilatation of the veins, and the tissues are under-nourished, yet are kept soaked in a transudate of poor quality, which leads to thickening of the connective tissue. Such tissues offer little resistance to the causes—especially infective—of inflammation. Thus spontaneously, or as the result of injury or of rupture of a vein, ulcers develop which show little tendency to heal. *See ULCER AND ULCERATION.*

SYMPTOMS.—It is not until veins are seen or felt to be varicose that the disease can be known to exist. When first seen a small part of a superficial vein, usually about the calf or ankle, looks slightly bluer and larger than usual. This may disappear for some time—only, however, to recur; and by-and-by the condition will become permanent, and may spread to other veins. There may be no pain with very large veins; but patches of very small varices are often very painful—probably from pressure on nerve-endings.

A limb may be somewhat swollen, and pit on pressure; or it may be hard from fibroid thickening. Here and there venous saccules may cause localised soft prominences. Signs of old or present inflammation are common; and ulcers often form in connection with varicose veins.

When a varicose vein bursts, hæmorrhage is likely to be excessive, more blood pouring from the upper than from the lower end of the vessel, the valves between the rupture and the heart being incompetent.

DIAGNOSIS.—Varicose veins, as usually found, are unmistakable in their appearance. A saccular dilatation of the internal saphenous vein, just below Poupart's ligament, has been mistaken for a femoral hernia; but there is no excuse for the error unless the sac be thrombosed, when the diagnosis from irreducible hernia may be impossible.

PROGNOSIS.—The dangers of varicose veins are: Dermatitis; ulceration; thrombosis; phlebitis, simple or infective; embolism; rupture and hæmorrhage. Much relief can be given by treatment, but cure can hardly be spoken of, except in limited cases.

TREATMENT.—The treatment in any particular instance is to remove, if possible, the cause of the

veins becoming varicose. The circulation in the dilated vessel must be encouraged—by obtaining a good cardiac and vascular tone; by support of the vein; and by particular attention to dietary and hygienic rules for some time. The vein is best supported by a well-fitting knitted stocking, by an elastic stocking, or by Martin's elastic bandage. To help the venous return, the patient when sitting should have the feet raised on another chair, and at night should sleep with the lower end of the bed slightly raised. Cold-water bathing, followed by firm rubbing with a towel upwards towards the heart, is useful. Treatment such as this no doubt promotes recovery after removal of a definite cause, e.g. gravid uterus.

It is desirable to operate in most cases in which the larger trunks and their main branches are affected, yet not so widely but that their removal can be effected by operations of reasonable extent. Operation is specially desirable (1) when the veins give rise to pain and inconvenience; (2) when hæmorrhage is imminent; (3) when ulceration threatens or is present; (4) when a patient wishes to enter the public services.

The operation usually practised is that of excising long pieces or the whole of the varicose trunks and branches, and especially all obvious points of anastomosis, through one long or a series of shorter cuts. The wounds almost always heal by first intention. The operation is easily done under eucaine, either in the leg or in the spermatic cord. Embolism is the danger, but rarely occurs. Other veins may dilate sooner or later after the operation.

3. **Hypertrophy of Walls.**—This takes place when extra work is thrown on the walls of the vessel, and is accompanied by increased blood-supply. See 2. Varicose Veins, p. 1831.

4. **Atrophy.**—When from any cause a vein, or part of a vein, falls into disuse, as after an amputation, its calibre diminishes and its coats atrophy, until it finally disappears.

5. **Degeneration.**—Degenerations in veins are probably similar to those occurring in arteries; but less is known about them. Calcareous plates are found at times in the saphenous veins, inferior vena cava, and uterine veins.

6. **Phleboliths.**—Phleboliths, or venous calculi, tend to form in veins in which circulation is slow, as in the veins of the prostate and bladder, and in varicose veins anywhere. They commence, no doubt, as precipitated fibrin, which becomes infiltrated with the less soluble salts of the blood—chiefly phosphate of calcium, and in less quantity sulphate of calcium and sulphate of potassium. Phleboliths are harmless, and require no treatment.

7. **Wounds, and Process of Healing.**—*Wounds.*—When a healthy superficial vein is wounded, dark venous blood flows in a uniform stream from the distal end; in the case of a varicose vein the blood flows from both ends, but chiefly from the cardiac. At the same time a quantity of blood escapes into the subcutaneous areolar tissue and around the sheath of the vein. When a deep vein is torn by a broken bone, without breach of surface, the part around swells, and becomes dark in colour; the part below swells and its circulation is more or less impeded, but it recovers in the great majority of cases, even when the extravasation is very extensive.

Healing process.—When gentle pressure is applied over the wound after venesection, and the limb is kept at rest, union takes place in a few days, so that no scar even can be found in the injured vein. The coats of a vein contract and retract only to a slight extent within the sheath, but still sufficiently to help in the arrest of the hæmorrhage and the formation of the clot. The extravasated blood coagulates, extends through the opening in the vein, and projects like a button into the blood-stream. The whole coagulum now contracts; the part projecting into the vein becomes 'organised,' and thus helps to close the wound. The vessel may not become obstructed, or, if it does, is soon opened up again. Instead of this favourable ending, numerous troubles may arise, such as spreading thrombosis and phlebitis; infective softening of the clot; local suppuration; embolism, simple or infective.

TREATMENT.—Hæmorrhage from a superficial vein is readily arrested by pressure over the wound and elevation of the limb. In the event of persistent hæmorrhage occurring from a deep vein after the employment of pressure and elevation, the vessel must be cut down upon and ligatured. When a large vein is pricked, and bleeds, the margins of the opening in the vein may be seized with a forceps, and tied with catgut; a larger opening, even in the inferior cava, has several times been closed by suture, or by clamp-forceps left on twelve to twenty-four hours.

8. **Air in Veins.**—See VEINS, Entrance of Air into.

9. **Parasites.**—The embryos of the *Tæniæ* and of the *Bilharzia hæmatobia* are occasionally found in the blood of the portal vein; the latter are also found in the veins of the bladder. See ENTOZOA.

10. **New-Growths.**—Tumours are not known to originate in connection with veins, but they often compress veins or grow into their lumina, and their cells are disseminated by the passing current. Often these protrusions cause thrombosis, local or spreading. For an account of venous nævi, see TUMOURS: Angiomata.

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VEINS, Entrance of Air into.—SYNON.: Fr. *Aérhémotomie*; Ger. *Luft Eintritt in die Venen*.

The result of the investigations respecting the entrance of air into veins may be summed up briefly as follows:—

1. **Amount of Air necessary to Cause Death.**—This varies with the size of the animal experimented on, and the mode of injection of the air. A small animal requires a smaller amount of air to cause death than a large one. If slowly injected, enormous quantities may be pumped into the veins with impunity; while a quantity sufficient to fill the auricle, injected suddenly, is certainly fatal. Recovery may take place after the occurrence of very serious symptoms, and on the other hand death may occur after an interval of some hours or even days.

2. **Causes of the Spontaneous Entry of Air into a Wounded Vein.**—In inspiration, the diminished pressure within the thorax causes just the same tendency for the blood in the large veins at the root of the neck and in the axilla to rush into the right auricle, as there is for the air to enter the lungs

by the trachea. This aspiratory force rarely extends beyond the veins mentioned, and the region in which they lie is often called the 'dangerous region.' If the vein be completely divided, its lax walls fall together, and thus offer a valve-like resistance to the entrance of air; but if in any way the opening be kept patent, air will rush in at each inspiration. The opening may be kept patent by a diseased condition of the walls of the vein, as when it passes through a tumour or is embedded in inflammatory products; or the surgeon may pull open a half-divided vein by traction on the parts he may be removing; or the aperture may be circular, as when a small piece is cut out of the wall of the vein, or a branch is cut off close to the main trunk. The external jugular, if divided low in the neck, remains open, on account of its connection with the cervical fascia.

The evidence that air can enter the veins of any other region and cause serious results is not altogether satisfactory, but the accident is believed to have caused sudden death during parturition, and to have been induced by intra-uterine manipulations, such as version in the treatment of placenta prævia.

After death the right side of the heart has been found distended with a frothy mixture of blood and air, and bubbles of air have been found in the pulmonary artery even as far as the smaller subdivisions.

SYMPTOMS.—As the air enters the vein, it gives rise to a noise variously described as hissing, whistling, or sucking. If the patient be not under the influence of an anæsthetic, he cries out that he is dying, or makes use of some expression indicative of great distress. He becomes immediately pale and faint. There is intense anxiety, and the most severe dyspnoea. The dyspnoea is purely cardiac in origin, the air entering the lungs freely, with violent and hurried inspirations. The sense of want of breath is due to an interruption of the flow of blood through the lungs. The pupils are usually widely dilated. Although the patient is pale and faint, the action of the heart may at first be felt through the chest-walls to be violent and irregular, and it is said that on auscultation a peculiar churning sound may be heard. The pulse rapidly becomes weak. Tetanic convulsions have occurred in some cases; in some instances violent coughing has also been noted. If a large quantity of air has entered, death may be almost instantaneous; but if a small quantity only has been sucked in, the patient may recover, occasionally after some hours of distress. In rare instances in which death has occurred some days after the accident, bronchitis was present, and possibly the entrance of air may not have been the real cause of death.

There can be no doubt that death results from arrest of the pulmonary circulation, and not from paralysis of the heart, for the cardiac action continues long after the air has entered. The want of blood in the lungs causes the sense of dyspnoea; and, the flow through the lungs having ceased, the left side of the heart will become empty, and, no blood reaching the brain, faintness, followed quickly by death, naturally occurs. Supposing the patient to recover, the air which may have got into the circulation is absorbed by the blood.

TREATMENT.—The accidental entrance of air into the veins must be prevented by careful operating. If a vein can be seen to be in danger it is advisable to compress it on the proximal side, and if it is

necessary to divide it, a double ligature must first be applied. In removing a tumour from the 'dangerous region' traction upon it should as far as possible be avoided.

If the symptoms described should appear, the further entry of air into the wounded vein is best prevented, as suggested by Treves, by at once filling the cavity of the wound with fluid, while an attempt may be made to expel the air from the right side of the heart by applying forcible compression to the front of the chest during expiration. The wounded vein should then as quickly as possible be secured and ligatured. Treatment should further be directed to the prevention of syncope by maintaining the circulation of blood to the brain. With this object the head should be lowered, pressure applied to the large arteries of the limbs, and stimulants administered hypodermically and by the rectum.

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VENEREAL DISEASES.—A common term for all forms of contagious disease usually contracted and transmitted by impure sexual intercourse. There are three principal kinds of venereal disease: namely, *gonorrhœa*, *syphilis*, and the *local venereal sore* or *chancere*. See GONORRHEA; SYPHILIS; and VENEREAL SORE.

VENEREAL SORE.—SYNON.: Local, Soft, Non-infecting Chancre or Sore; Fr. *Chancre Mou*; Ger. *Weicher Schanker*. The ambiguous term 'chancroid' has been adopted by many American authors.

DEFINITION.—A virulent, local, infectious ulcer, communicable by contact of its secretion with a breach of surface.

ÆTIOLOGY.—The ætiology of the local chancre is still a subject of dispute. A few maintain that it is connected with syphilis. Others affirm that it may be caused by the products of simple inflammation. But the view which is most in accord with the present state of knowledge on the subject is that the local chancre is due to a specific virus distinct from that of syphilis; that it never gives rise to any special constitutional symptoms; and that its effects are limited to the neighbourhood of the sore itself and the nearest lymphatic glands.

In 1889 Ducrey, of Naples, announced that he had succeeded in isolating on the human skin the pathogenic microbe of soft chancre, which he described as a short thick bacillus 1·48μ long and 0·50μ broad, with rounded ends and usually notched laterally. He had not been able to cultivate it outside the body. It seems now to be pretty generally agreed that this strepto-bacillus is peculiar to the soft chancre, but whether it is the sole causal agent or not is still disputed. Some observers claim to have isolated and cultivated it artificially, while others have failed, so that at present the question must be considered unsettled.

INFECTION.—The local chancre, like syphilis, may be communicated by direct or by mediate infection; but it is of course never transmitted by inheritance. Syphilis is not infrequently conveyed in various ways independent of sexual intercourse (see SYPHILIS). The local chancre, on the other hand, is very rarely other than a venereal disorder; chiefly because, not being part of a general disease, it does not give rise to infectious lesions on distant parts of the body.

The local chancre, like syphilis, may be conveyed from one person to another, through the medium of a third, whose cutaneous or mucous surface happens to be intact, and who thus escapes infection. One attack of the local chancre, unlike syphilis, affords no protection for the future.

DESCRIPTION.—When chancrous pus is inoculated artificially, the puncture within twenty-four hours becomes surrounded by a faint red blush; on the second day the redness extends, and the site of inoculation becomes swollen. On the third day a vesicle appears, and rapidly becomes a pustule. About the fifth day the pustule bursts, leaving a circular ulcer, with well-defined, sharply-cut edges, surrounded by a pink areola. The surface of the ulcer is uneven and spongy, the discharge is purulent, and as time goes on the borders become irregular and undermined.

When the local chancre is due to venereal infection the pustular stage is rarely, if ever, seen in practice, and it usually comes under observation as an ulcer which resembles that produced by experimental inoculation more or less closely, according to the circumstances of the particular case.

The base of the local chancre is supple, unless inflammation be present, in which case there is more or less thickening of the surrounding parts.

When the sore is in such a position that the urine frequently comes in contact with it, or when it is irritated in any other way, the thickening of its base may be sufficient to raise the sore above the surrounding parts, so that the surface, instead of being depressed, becomes more or less prominent, in which case the term *ulcus elevatum* is sometimes applied to it. The discharge is readily and repeatedly auto-inoculable; hence a plurality of sores is very common.

The *seat* of chancre is nearly always the skin covering the genital organs or their immediate neighbourhood. A primary sore on the body of the penis is much more likely to be syphilitic than local.

The *form* and *aspect* of the chancre vary in some degree according to its position. Thus in the furrow behind the glans penis the sore may be elongated; about the frænum, in the folds of mucous membrane around the anus, and about the female genitals it is often fissure-like. Again, when exposed to the air and neglected, it becomes covered by a scab. If chancrous pus gain entrance to a follicle (*follicular chancre*) the follicle may become distended, and temporarily simulate a boil or small abscess.

As regards *size*, chancres rarely exceed half an inch in diameter, unless two or more run together, in which case they form a large irregular ulcer. Such confluent chancres are more common in women.

COURSE, DURATION, AND TERMINATIONS.—

When a typical chancre is allowed to run its course, it lasts from four to eight weeks. This course is divided into three stages: (1) an increasing, (2) a stationary, and (3) a healing stage. The length of each is liable to vary from many causes. Roughly speaking, however, it may be said that the first period, during which the sore sensibly increases in size, occupies from a week to a fortnight; that the second lasts about a fortnight; and that at the end of from three to five weeks the stage of repair is reached.

By appropriate treatment the duration can be greatly shortened, but it is also influenced by the position of the sore. Thus, at the orifice of the prepuce or urethra, the irritation of the urine retards

healing, and phimosis acts in a similar manner. Chancres of the frænum, again, often continue to spread until they have perforated or destroyed it. In fact, the more mobile and exposed to irritation the part, and the greater the difficulty of keeping the dressings in place, the longer will be the duration of the sore.

If the syphilitic virus have been absorbed as well as chancrous pus, the course of events will remain uninfluenced until the incubation-period of syphilis has elapsed; when, if the sore be still unhealed, it will gradually change in appearance by the development of induration at its base, until finally it assumes the characters of the initial manifestation (*see* SYPHILIS). If, on the other hand, the local sore has healed before the end of the period of incubation proper to syphilis, the scar will become indurated, and erosion or ulceration will probably, though not necessarily, follow.

COMPLICATIONS.—The less serious complications of the local chancre include *inflammation*, *phimosis*, and *paraphimosis*. These are dealt with under their respective headings.

Phagedæna.—This graver complication of chancre may be either acute or chronic; and may attack the original sore or the consecutive bubo, or both.

In a typical case of *acute sloughing phagedæna* the patient is usually much depressed. He soon becomes feverish, with a dry, brown tongue, quick pulse, and other signs of constitutional disturbance. The discharge from the sore diminishes, and is thin, sanious and shreddy, and very offensive. The margins of the ulcer are puffy and livid at first, but soon become black and ragged, presenting a ‘gnawed’ appearance. As the process goes on, the dead tissues separate in sloughs, which vary in size according to the rapidity of the destruction. In the worst cases necrosis is very rapid, and attended by severe pain. The whole of the genital organs in either sex may thus be destroyed; while, if the groin be the seat of phagedæna, the great vessels and nerves are quickly exposed at the bottom of a deep ragged cavity. If the process be not soon checked, death may occur from hæmorrhage, from exhaustion, or from some acute intercurrent inflammation.

The above description applies to a form of phagedæna which is now fortunately rare; but less severe examples, ranging in various degrees between that described and those where a sore simply becomes inclined to spread, looks unhealthy, and has ‘gnawed’ edges, are common enough in venereal practice.

In the *chronic form* of phagedæna the morbid process is much less active, and is not usually attended by much pain or constitutional disturbance. The sore spreads gradually, but the tendency is rather to extend widely and superficially than deeply. The groin is the most usual seat of the ulceration, whence it may extend upwards along the abdominal wall or down the thigh, laying bare the deep fascia, and dissecting out the superficial vessels and nerves. It often also undermines the skin extensively before destroying it; hence the loose and irregular margins of the ulcer are detached from the deeper parts for a considerable distance. The duration of this form of ulceration is very variable. It sometimes lasts for years; at one time appearing to be stationary, while at other times it spreads in one direction and heals in another. In such cases the patient’s health suffers more or less severely, and finally he may

become exhausted by the constant and prolonged irritation and discharge. If he recover, he may be permanently crippled by the contraction of the resulting cicatrices.

Bubo.—This complication, which is less frequent in women than in men, and in private than in hospital practice, is said to occur in about 20 to 30 per cent. of the total number of cases of chancre. Bubo may be either *simple* or *virulent*, the former being most common. See BUBO, VENEREAL.

Simple or *sympathetic bubo* arises from the absorption of ordinary pyogenic material, and is similar to that which may accompany a suppurating focus of any kind.

Virulent (chancreous) bubo may be due to the inoculation of a simple abscess by chancrous pus, or to virulent matter conveyed from the sore by the lymphatics to the gland. The latter mode of production has been doubted by some observers since Ducrey failed to find the bacillus known by his name in the pus of any bubo when strict precautions had been taken to prevent inoculation from the outside.

Bubon d'emblée.—This term has been applied to

Local Chancre.

1. A local and nearly always venereal disorder, produced by the pus of a similar ulcer.
2. Irritation begins within a few hours after infection.
3. Begins as a pustule which soon bursts, leaving an excavated ulcer with sharply cut, loose, often undermined edges, and an irregular spongy floor of a dirty yellow colour.
4. Base supple unless inflamed, in which case it becomes firm like that of a boil; the firmness is diffused, resistant to the touch, ill-defined, and fades gradually into the surrounding tissues.
5. Discharge abundant, purulent, and freely and repeatedly inoculable, on the bearer as well as on others.
6. Usually multiple.
7. Very rarely seen far away from the genital organs.
8. Course acute, and attended by pain or soreness.
9. Duration greatly influenced by local treatment.
10. Inflammation and phagedæna not very uncommon.
11. Glands remain unaffected or become acutely inflamed. Suppuration common. Bubo may be simple or virulent. Only one or two glands suffer.
12. Does not cause general infection of the system.
13. Repeated attacks common.

While the characters given in the foregoing table are amply sufficient for the diagnosis of the two lesions in uncomplicated cases, it must be mentioned that the appearance of either sore may be altered in various ways, for example, by neglect of cleanliness, or by the application of irritants. Thus the local chancre, especially about the margin of the prepuce, may develop an amount of inflamma-

a variety of bubo, believed by some authors to result from the absorption of chancrous pus without the production of any lesion at the point of inoculation. The existence of such a bubo is not established.

Lymphangitis.—This, like bubo, may be simple or virulent, but is a less frequent complication of the local chancre. Bubo of either kind may exist without any perceptible change in the lymphatics leading to it; but when the vessels are affected, the glands are usually inflamed also. The thickened lymphatics can be felt as tender and often irregular cords, and their course is generally marked out by red streaks along the surface.

Simple lymphangitis may end in resolution, or one or more abscesses may form along the course of the vessel. The virulent form probably always goes on to suppuration, and the resulting sores become chancres similar to the original one.

DIAGNOSIS.—The local chancre is recognised by the characters which have been already mentioned, and which are recapitulated in the following table, where also they are contrasted with those of the initial manifestation of syphilis.

Initial Manifestation of Syphilis.

1. The first sign of a general and not necessarily venereal disease; it may be produced by the secretion of any syphilitic lesion or by the blood during the earlier stages of the disease.
2. Always a period of incubation, which averages 24 days.
3. Begins as a papule. Ulceration may be absent throughout. Edges raised, adherent, and rounded. Surface smooth and often of a ham-red colour.
4. Base more or less indurated. The hardness is sharply circumscribed, somewhat elastic to the touch, and independent of acute inflammatory action; it varies much in amount, being sometimes superficial and scanty, and feeling like a thin layer of parchment or paper; sometimes abundant, and feeling like a mass of cartilage.
5. Discharge scanty, serous, and not readily auto-inoculable.
6. Usually single.
7. Not very infrequent on the lip or finger, and on the breast in women.
8. Course indolent. Pain generally absent.
9. Duration greatly influenced by general specific treatment.
10. Inflammation and phagedæna rare.
11. Adenopathy constant, indolent, and generally multiple. Suppuration rare.
12. Is followed by constitutional symptoms.
13. A second attack rare.

tory thickening which cannot for a time be distinguished from that produced by similar causes in the syphilitic initial lesion; for the specific induration may become masked by inflammation. In such cases a positive diagnosis must be postponed until the irritation has subsided. Again, even if a patient present himself with a typical local chancre, it is of course not certain that the syphilitic poison

has not been imbibed as well ; and the incubation-period of syphilis must be allowed to elapse before the patient can be assured that his trouble is only local. Further, as regards the two most characteristic signs of primary syphilis, namely, induration of the base of the sore, and multiple enlargement of the nearest lymphatic glands, one or other may be ill-marked, or in rare instances inappreciable. Then, as regards number—the initial lesion of syphilis may be multiple if several places happen to be inoculated, as in the case of herpes for example ; and the local chancre may be single, especially in persons of careful and cleanly habits. Inflammatory action also may mask the usually separate indolent glands of syphilis ; but, apart from this, sometimes only one or two glands can be felt in the groins, in which case there may or may not be evidence that some of the glands have been spoiled previously. Absence of enlargement also appears to be sometimes due to the pressure of a truss. In very fat people, again, the glands cannot always be felt.

On the other hand, the multiple adenopathy of primary syphilis is occasionally closely simulated in some other affections — irregular herpes and gonorrhœa for example. In making a diagnosis the gradual and progressive character of syphilitic adenopathy is quite as important as its extent. Thus not one but all of the signs that may be present, together with the history of the case, must be taken into consideration ; and if the diagnosis still remains doubtful, the case must be watched until conclusive evidence is forthcoming.

Auto-inoculation is sometimes of service in diagnosis, but in the present instance would not be of much value ; for even if a typical local chancre were the result, syphilis could not be excluded until the incubation-period had elapsed. Care is always necessary in interpreting the results of inoculation ; for though it may be reasonably concluded that an active sore, the discharge from which is not auto-inoculable, is not a local chancre, yet the converse by no means always holds good. It must be remembered that matter from various lesions is inoculable on the bearer. The important point is not that inoculation produces something, but the character of the lesion which it produces.

When an immediate diagnosis is imperative, *confrontation*, or comparison of the patient with the source of his disease, may set the question at rest ; but many precautions are necessary in drawing conclusions from such evidence.

Ulcerating syphilides, especially mucous patches of the female genitals, occasionally resemble chancres, but the presence or history of other signs of syphilis would lead to a correct diagnosis.

Gummata of the genital organs and groins, after breaking down, leave ulcers which sometimes resemble local chancres or virulent buboes, but which as a rule are not auto-inoculable.

Besides syphilitic affections, there are some others which require to be mentioned in connection with the diagnosis of the local chancre. *Herpes* is usually seen as a group of vesicles or small superficial erosions on an inflamed area, but it must not be forgotten that occasionally there is only a single spot. Itching or smarting often precedes an attack of herpes, and there is frequently a history of previous attacks, which, again, are often independent of any suspicious sexual exposure, and are liable to recur at regular intervals, or in connection with diges-

tive disturbance. Herpes also usually heals in a few days under measures of simple cleanliness. See *HERPES PROGENITALIS*, p. 685.

Abrasions are irregular in form, are usually noticed by the patient at the time of their production or very soon afterwards, and heal readily in a few days if they are not irritated. Both herpes and abrasions, however, as well as the erosions due to balanoposthitis, may, under irritation of various kinds, become suppurating and inflamed ulcers, which for a time it is difficult to distinguish from chancres.

When phimosis prevents exposure of the parts, a discharge from beneath the prepuce may be due to several other causes besides the local chancre, for example, syphilis, gonorrhœa, balanitis, or warts. If the local chancre be present, its site is usually indicated by the presence of a tender spot in that situation, and pressure often causes a slight oozing of blood. Consecutive sores also quickly appear at the margin of the prepuce. In the case of *syphilis*, the presence of induration, and the multiple indolent enlargement of the inguinal glands, will usually render the diagnosis clear. In *gonorrhœa*, soreness of the deeper urethra, scalding during micturition, the presence of chordee and other signs of urethritis, and the absence of localised tenderness beneath the swollen prepuce, are points that will determine the nature of the case. In *balanitis* there will be an absence of circumscribed tenderness and of signs of urethritis. *Warts* can be felt through the prepuce, and a portion of the growth can usually be seen by putting the parts on the stretch. A small speculum is sometimes of use for diagnostic purposes in cases of phimosis.

PROGNOSIS.—The prognosis of the uncomplicated chancre is always favourable. If any complication arise, the prognosis will depend upon its nature and severity.

TREATMENT.—The treatment of chancre consists chiefly in the employment of local remedies. The general treatment at the same time is directed, on ordinary principles, to the maintenance of the patient's health, by tonics, regulation of the diet, moderation in, or abstinence from, alcohol, and as much rest as possible, in order to diminish the risk of complications.

A local chancre of only a few days' duration can generally be destroyed by one thorough application of heat or caustic. From a purely therapeutic point of view, however, destructive measures are not often required ; and as regards the prevention of syphilis, supposing the virus of that disease to have been inoculated, the results of observation so far seem to give but little hope of success.

In nearly all cases the best application is iodoform, of which the *powdered* crystals, either alone or mixed with boric acid according to circumstances, may be sprinkled on the sore by means of a quill, or applied with a moistened camel's-hair brush twice or thrice daily, according to the amount of discharge. The sore should then be covered with a piece of lint or wool, and if the dressing cannot be kept in place by the natural conformation of the parts, a narrow strip of bandage or plaster should be applied. Iodoform may also be applied as an ointment with vaseline, or an ethereal solution may be painted on the part, a thin coating of iodoform being left after the evaporation of the ether.

When for any reason the use of iodoform is

impracticable, the writer has found airol (an iodo-gallate of bismuth), which has no odour, a fairly satisfactory substitute ; or salicylate of bismuth may be used. When the healing stage is reached some preparation of boric acid or zinc usually answers best. Whatever dressing is employed, care must be taken to change it frequently, without making the sore bleed, to prevent the urine from coming in contact with it, and to arrange it so that opposed surfaces are kept apart. Chancres with phimosis must be treated by frequent injections, with a syringe having a long nozzle, of a lotion of carbolic acid (1 in 40), or of boric acid and glycerine. Any of the sores that are within reach should be dressed with iodoform, and a piece of absorbent wool placed within the preputial orifice.

In certain cases, especially in hospital practice, it may be desirable to begin treatment by thoroughly disinfecting the sores ; and for this purpose strong carbolic acid is usually effectual and gives but little pain. It is well to avoid the use of nitric acid for doubtful sores on account of the inflammatory hardness it is liable to produce. Nitrate of silver and sulphate of copper should never be used in such circumstances, not only because they are ineffectual but also because they set up an amount of hardness which may be sufficiently persistent to interfere with the diagnosis of the case if syphilis have been contracted. The actual cautery is rarely, if ever, required in uncomplicated cases. Before using either caustic or cautery the following points should be borne in mind : (1) No sore should be cauterised unless the whole of the diseased surface can be acted on. (2) The surrounding parts must be thoroughly cleansed and disinfected.

Treatment of complications.—Inflamed chancres should be treated by keeping the patient at rest, on simple diet ; by the administration of a purge ; and by the application of lead or warm boric-acid lotion. In phimosis, if the swelling is so great as to prevent the use of the syringe, or if sloughing is threatened, the prepuce must be slit up or removed altogether, and the case treated according to the directions given for the treatment of phagedæna.

If in paraphimosis the prepuce be naturally too narrow, or if strangulation occur, reduction should be effected.

On the first appearance of signs of acute *phagedæna*, the affected part should be immersed for nine or ten hours a day in water at a temperature of about 98° F. This can be accomplished by keeping the patient in a hip-bath, and alternately adding and removing small quantities of water, so that the requisite temperature is maintained. Care must of course be taken to protect the patient from cold, by placing the bath in a warm room, and by wrapping the exposed parts of the body in blankets. If this plan be adopted at an early period the sore often becomes healthy in a few days ; immersion, however, should always be continued for at least a day or two after this has occurred, to guard against relapse. The patient may generally be allowed to go to bed during the night, iodoform or some other suitable dressing being applied ; but if the diseased action continue to extend, the duration of the bath must be prolonged, or even be made continuous ; but in that case, as well as in the cases where the groin is the seat of the disease, a full-sized bath in which the patient can lie down will be necessary. In all cases the whole of the diseased surface must be fully exposed and thoroughly submerged.

If iodoform and immersion fail, or if immersion cannot be carried out, a lotion of tartarated iron (10 to 60 grains to the ounce), or a saturated solution of permanganate of potassium, should be tried.

If milder measures fail to arrest the phagedænic action, strong nitric acid or the thermo-cautery may have to be resorted to. When the surface to be cauterised is small, a solution of cocaine may be applied beforehand, but when it is extensive general anaesthesia will be advisable.

Hæmorrhage from a phagedænic sore should always be checked as soon as possible. If the bleeding is slight, the surface should be cleaned, and pellets of cotton wool, soaked in solution of persulphate of iron, pressed on each bleeding spot, and retained in position by a bandage applied as tightly as the patient can bear. In severe cases the actual cautery may be necessary ; and in extreme instances ligation of the larger arterial trunks above the seat of disease may be required.

The general treatment of phagedæna consists in the administration of good food, tonics, and opium in sufficient quantity to relieve pain ; but under the immersion-plan of treatment, pain usually quickly ceases, and little or no opium is required. Fresh air and good ventilation are also most important.

The treatment of chronic phagedæna is similar to that of the acute form, but of course more time is allowed for the trial of palliative measures before having recourse to the cautery.

For the treatment of simple bubo, see BUBO. It need only be remarked here that it is as important to avoid the application of irritants in the case of bubo as in the case of the sore.

The management of the *virulent bubo*, after evacuation of the pus, is similar to that of the local chancre, which has been described ; and the treatment already recommended for phagedæna applies equally to that morbid process, as a complication of the local chancre, of bubo, or of syphilitic ulcers, whether primary, secondary, or tertiary ; but in the case of syphilis the administration of mercury or iodides, or both, according to circumstances, forms an important element in the management. In fact, in every case of obstinate phagedæna, especially of the chronic form, most careful search should be made for signs or a history of syphilis ; for, if that taint be present, it not infrequently happens that all local applications fail until the constitutional malady is attacked.

ARTHUR COOPER.

VENESECTION (*vena*, a vein ; and *seco*, I cut). **SYNON.** : Bleeding ; Blood-letting ; Abstraction of blood by opening a vein ; *Fr.* *Vénéssection* ; *Ger.* *Aderlass*.—Venesection is performed at the level of the elbow. In practice the most prominent vein is selected ; for anatomical reasons the median cephalic is preferable, although the brachial artery can hardly be punctured except by a very bungling operator.

The skin at the level of the elbow is carefully sterilised. A bandage is tied firmly round the arm. The patient grasps some substance in the corresponding hand, as a stick or piece of bandage. A fine and very sharp sterilised knife is in readiness. The hands and nails of the operator should be cleaned with all the care he would take in a major operation (*see* ASEPTIC SURGERY). A large and prominent vein is selected and fixed by the thumb of the left hand of the operator, who grasps the limb by placing the

fingers of the same hand behind the elbow. The knife is then inserted by the side of the vein, turned across the vein, and brought out smartly so as to make an *oblique incision* across the vessel—not in its longitudinal axis. The blood flows in a continuous dark stream, and is received in a measured basin until a sufficient quantity is withdrawn. The operator checks the flow by placing the thumb on the aperture, removing the constricting bandage above, and directing the patient to drop the bandage or stick out of his hand. The puncture is then dressed with a pledget of iodoform gauze and covered with collodion. Some mercuric wool and a firmly applied 'figure of eight' gauze bandage completes the dressing, and the arm is placed in a sling. By the fourth day healing should be complete, and the puncture merely needs the protection of a little collodion and wool. The conditions in which venesection is now employed are discussed in the article on BLOOD, Abstraction of.

A. MARMADUKE SHEILD.

VENOMOUS ANIMALS.—SYNON. : Fr. *Animaux Vénéneux*; Ger. *Giftige Thiere*.

DEFINITION.—Animals which possess the power of secreting and ejecting a poison, which, when inoculated in man or other animals, produces toxic or even fatal effects.

Venomous animals are found in many classes of the animal kingdom.

1. **Reptilia.**—Reptilia furnish the most numerous and important kinds of venomous animals, and these are limited almost entirely to the order *Ophidia* or snakes.

The statement that no lizard is poisonous is not strictly correct. The heloderm (*Heloderma suspectum*) of Mexico possesses venomous properties, destructive to small animals, and injurious to man himself.

DESCRIPTION.—The poison-apparatus of a snake consists of a composite racemose gland, situated in the temporal region, secreting a clear, slightly viscid, acid fluid, which is poured through a duct into a grooved fang situated on a movable maxillary bone. The fangs are longer, more curved, more movable, and more formidable in viperine (viper; rattlesnake, &c.) than in colubrine snakes (cobra; bungarus, &c.).

Snake-poison is very deadly; more active in some snakes, quantity for quantity, than in others; and varying in activity in the same species or individual, according to season, temperature, and state of health. It acts most rapidly when injected into the blood; but it can be absorbed through mucous and serous membranes, as seen by its poisonous effects when applied to the conjunctiva, the stomach, and the peritoneum. It may neither be applied to the lips nor taken into the stomach with impunity; and sucking a snake-bite is by no means free from danger, though if the saliva be quickly ejected and the mouth washed, the danger is probably small. It may be noted here that the poison of viperine forms differs considerably from the colubrine. They both contain active principles, two of which, according to Drs. Weir Mitchell and Reichert, are proteids, one belonging to the globulins, the other to the proteins; they attribute the difference of the physiological effects produced by different species to the varying proportions of these bodies. Analysis has shown the poison to be very

nearly allied to albumen in composition: according to recent observers, one very toxic body is of the nature of an albumose. The composite nature of venom is proved by the results of heating the poison of the *pseudechis*: thus treated it retains its lethal properties, but loses the power of producing hæmorrhages and clotting of blood within the vessels. Venom is most active on warm-blooded creatures. Poisonous snakes are very insensible to the venom of other poisonous snakes, and absolutely so to that of their own species.

EFFECTS.—The action of the poison is *local* and *general*.

The *local* effects of snake-bite comprise pain; partial paralysis of the bitten part; ecchymosis; swelling; and, if death does not rapidly follow, infiltration of other and distant parts, cellulitis, and sloughing.

Associated with these local effects are many severe *general* phenomena, such as depression, fainting, nausea, hurried respiration, vomiting, exhaustion, hæmorrhagic discharges, lethargy, loss of co-ordinating power, paralysis, loss of consciousness, relaxation of sphincters, coma, and convulsions. If the quantity of poison injected be small or its nature feeble, the earlier symptoms may give way, and recovery take place. The poison is probably eliminated by the kidneys, and in the case of the venom of viperine snakes nephritis may thus be produced.

Snake-poison acts by paralysing the nerve-centres, or sometimes the peripheral distribution of the nerves; and by altering the constitution of the blood. It takes effect through the circulation; and if inserted into a large vessel, such as the jugular, humeral, axillary or other large vein, it will cause almost instant death, the heart's action stopping in systolic spasm. The respiratory centres, the spinal cord, and the peripheral nerves, may all be affected. In ordinary cases death seems to take place by arrest of the respiration, the heart's action continuing for some time after apparent death. The muscular fibre itself would appear in some cases to have its contractility impaired or destroyed. The poison also acts septicly, producing at a later period sloughing and hæmorrhage. Extravasation of blood appears to be due to a direct action of the poison upon the vessel-walls (Weir Mitchell).

There are certain points of difference in the action of viperine and colubrine venom. The poison of viperine snakes effects a direct destruction of the red corpuscles. There is greater tendency to hæmorrhage, extravasation, and fluidity of the blood. In the case of colubrine venom, the effect seems to be more directly upon the nervous system, the blood being less affected. Experiments on animals show that, generally, after death from cobra-poisoning the blood coagulates firmly, while after death from viper-poisoning the blood remains permanently fluid. In most cases of death in man the blood has been found fluid even after cobra-poisoning. It is said that the normal bactericidal power of the blood is destroyed by the action of snake-poison (Ewing).

PROGNOSIS.—In cases of moderate severity, remedies, with careful nursing and tending, may prove successful; but where the bite has been thoroughly effected by the ophiophagus, cobra, daboia, echis, rattlesnake, crotalocephalus, cerastes, and others, the prognosis is very unfavourable; in no case, however, should efforts be

relaxed until the last. The bite of the European viper is not often fatal to healthy adults.

There is often uncertainty as to the kind of snake, its condition, and the extent to which its fangs were used. The great shock or depression which follows a snake-bite may be in a measure due to fright, and will, on reassurance, pass away. The marks of two well-defined punctures attest the insertion of two fangs, and, if the snake has not been seen, may enable one to form an opinion as to its character. Many of the innocuous snakes are fierce, and bite vigorously, but their numerous teeth leave different marks from those of the poison-fangs. There are exceptions to this rule, however; a few innocent snakes have the anterior maxillary teeth developed like poison-fangs, but bites from them are not very likely to occur.

It may be well to note some of the characters that distinguish the venomous snakes, such as the form and arrangement of the teeth; an examination of the wound will often reveal the true character of the bite, and serve to indicate a correct prognosis. On opening the mouth of a venomous colubrine snake, such as *naja* or *bungarus*, two slightly movable fangs will be observed, one on either side, as well as several loose reserve fangs in various stages of development; and close behind them there may be seen one or two smaller teeth. There is no row of teeth along the outer side of the mouth, but a double row of palatine teeth will be found on either side. In the viperine and crotaline snakes, a large fang only is found on either side, and a palatine row as in the colubrine. There are no small fixed teeth behind the fangs as in colubrines, but in a fold of mucous membrane at the base of the fangs, both in vipers and colubrines, several loose reserve fangs will be found in various stages of development, one of which takes the place of the ordinary fang when it is shed, by becoming ankylosed to the maxillary bone. In *hydrophide* the fangs are arranged like those of the cobra, but are very minute, and no reliance can be placed on any mark made by them. The circumstances under which a bite is inflicted will generally help to indicate the kind of snake.

Harmless snakes have a double row of equal or nearly equal-sized teeth in the maxillary and palatine bones. But, as before stated, there are certain innocent snakes that have long anterior maxillary teeth, which might cause doubt as to the nature of the bite.

TREATMENT.—There is reason to believe that the numerous agents that have been recommended from the earliest times as antidotes of snake-poison are useless, and have no such properties as those ascribed to them.

The rational treatment of snake-poisoning is to endeavour to prevent the entry of the virus into the circulation; to neutralise it in the wound before it is absorbed; to support the failing nervous force if it have entered; and to favour its elimination.

The application of a tight ligature between the bite and the heart, and the immediate excision or destruction of the bitten spot, by cautery or caustic, are essential; and other local measures may appear necessary. The injection of a solution (5 to 10 per cent.) of permanganate of potassium into the bitten part has for its object the neutralising of the poison retained there by the ligature, and there is reason to believe that this is sometimes effected. The ligature may then be removed, thus obviating

the risk of gangrene in the part below the constriction. Calmette advises the local injection of a fresh solution (1 in 60) of calcium hypochlorite, or a solution of chloride of gold (1 in 200).

The constitutional treatment requires that the strength should be supported by stimulants, such as alcohol and ammonia. Next, if the respiration be failing, artificial respiration should be resorted to. Elimination should be promoted by stimulating diuretics. The patient should be kept warm, and must not exhaust himself by walking about. Ammonia has always held a high place among remedies in snake-poisoning. Its injection into the veins has been warmly advocated in Australia, and is said to have met with success there, which it certainly had not in India.

Dr. Mueller in Australia strongly advocates the injection of strychnine in repeated doses, and cases are cited which seem to confirm its utility; but there is not yet sufficient evidence to warrant the certainty of its efficacy. Very promising results have been obtained (Calmette; Fraser) experimentally and confirmed in a number of instances of snake-bite, by means of injection of an antitoxic serum or antivenene. This is prepared by injecting a large animal (horse) with gradually increasing doses of cobra-venom, and when immunity is established, collecting the serum for therapeutic purposes. Doses of 10 c.c. are recommended for human use, repeated if necessary. The action of the remedy is probably similar to that of antidiatheritic serum (antitoxin) (*see* ANTITOXINS). A transient immunity may be conferred by doses of antivenene. The remedy is applicable to bites from almost all poisonous snakes. Its efficacy is destroyed by heating to 70° C., but the power of venom itself is not thus affected. The serum retains its activity for at least a year (Calmette). *See also* p. 1485.

2. Amphibia.—None of the amphibia are known to possess a poison-apparatus like that of ophidia; but toads and salamanders secrete a fluid in glands along the back, connected with the integument, which yields an actively venomous principle, capable of causing local irritation, and, when injected into the blood, death, preceded by symptoms indicating action on the cerebro-spinal nerve-centres. Dogs seizing the toad *Bufo vulgaris* are known to suffer from swelling of the lips and salivation; and a case of death was related in France, in 1865, of a child in whom an abrasion of the hand came in contact with the secretion of a toad; death was preceded by vertigo, vomiting, and fainting. When this poison is injected into guinea-pigs, small birds, and other animals, violent symptoms and death soon follow. It is a viscid, milky fluid, with a slightly yellow tint and peculiar odour; it is exuded, and may be pressed, from glands behind the orbits. Zalesky has shown that the land and water salamanders, *S. maculatus* and *Triton cristatus*, and probably others, have also the power of secreting venom; and his experiments prove that it contains a very active principle—*salamandrine*—and that its action on the cerebro-spinal nerve-centres is energetic. It appears that these poisons, like those of ophidia, though effective on others, have no action on their own species.

3. Pisces.—**DESCRIPTION.**—Several fishes are provided with an apparatus consisting of a cavity at the base of, or a sac and duct leading to, a channelled spine, through which an irritating secretion is ejected. No true poison-gland, however, has as

yet been certainly made out. This secretion is apparently connected with the secreting mucous system; and in certain species it produces marked symptoms of poisoning, though never to the same extent as in venomous snakes. Fish armed with sharp or serrated opercular or fin-spines can inflict severe injuries, liable to cause great pain, and to be followed by the grave symptoms attributable to the lacerated or punctured nature of the wounds; and these may be aggravated by the irritating nature of the mucus with which they are contaminated. In several, however, in addition to the spine, there is a distinct receptacle in connection with it, either in the form of a sac or duct, as in the *thalassophryne*, or in a cavity in the spine itself, as in the *trachinus* or weever.

In the case of others, such as the sting-rays, which may produce severe wounds by their pointed and serrated spines, there is no distinct receptacle for the poisons in connection with them. While it is well known that many spiny fish are capable of inflicting wounds that are dangerous from their lacerated and punctured character, it is recognised that others increase the danger by the inoculation of an irritating fluid, as just stated.

EFFECTS.—The effect of fish-poisoning is to produce severe burning pain at and beyond the injured part, and fever. The intensity, no doubt, depends upon the quantity of the poison injected, and the state of health and constitution of the person at the time. The wound alone, even without the poison, is likely to be painful and severe from its punctured character.

TREATMENT.—Ipecacuanha, tobacco, alkalis, alum, and ammonia have all been recommended as useful local applications to allay the irritating action of such poisons. Poultices of onions, or warm applications of opium or other sedative fomentations are likely to be useful; and prompt surgical relief is necessary, if suppuration or cellulitis occurs, to relieve tension, evacuate pus, or give exit to sloughs.

The constitutional treatment is such as would be indicated by the condition and progress of any other inflamed punctured wound. In cases of depression of the heart's action, alcohol or ammonia would be indicated. Rest, quiet, and due attention to the state of the bowels, and elimination by the skin and kidneys, with careful regulation of the diet, should be observed. *See* SAPRÆMIA.

4. **Mollusca.**—*Aphysia punctata*, the sea-hare, a gasteropod, is said by some to produce an irritating secretion capable of causing urticaria and even severe inflammation, and of causing the hair to fall off.

5. **Arthropoda; Myriapoda**, family *Scolopendridæ*.—The centipedes possess mandibles, formed by a pair of dilated feet, joined at their origin, with perforated, hook-like points, and with an aperture near the apex, through which a poisonous fluid, secreted in a poison-gland, with sac and duct, is ejected when they bite, which they can severely. In the case of the larger tropical species the bite is sometimes very painful, and causes considerable local irritation, and even constitutional disturbance—fever and delirium. That of the smaller kind generally causes only local and transient irritation. Centipedes are found nearly all over the world, in Europe, Africa, America, the East and West Indies and Islands, and in the

tropics generally. Those of warm climates are the largest and most dangerous.

6. **Arachnoidea.**—*Scorpionidæ* or *Pedipalps*. **DESCRIPTION.**—Scorpions have a segmented abdomen, the last six joints of which are narrowed into a tail, terminated by a curved perforated spine or hook, with which they strike and wound. At its extremity are two small orifices, through which venom is injected from a gland-receptacle and duct at its base. Scorpions run about quickly, carrying the tail curved over the body. They live in holes in the ground, and under stones or logs of wood, in dark places. The tail is used as an offensive weapon. They seize small creatures with their palpi, and then pierce them with the sting. The venom is so active that it quickly destroys life in these animals. Those of tropical climates are most active and poisonous. They attain to the length of from two to three, four and six inches. The European genera are smaller and less active.

Scorpions exist in all tropical countries, but extend also into the warmer regions beyond the tropics. They are found in the East and West Indies, Ceylon, and other islands, Australia, Africa, Egypt, South of Europe, and America. There are several genera, and *Buthus afer*, *Androctonus*, and *Buthus Caesar*, are good examples of the active kinds. *Europæus* and *Occitanus* are also venomous, but those of Europe are less active than the tropical forms.

EFFECTS.—The effects of the sting of the scorpion and of the bite of the centipede have no doubt been exaggerated; but they may produce very painful, and in the case of the larger species, severe and serious symptoms, in their character not unlike, or even more severe than, those of the sting of the wasp, namely pain, swelling, in some cases numbness, vertigo, nausea, vomiting, temporary loss of vision, swelling of the tongue, and fever. Death may occur in delicate or sickly subjects. The local and constitutional symptoms may be severe in persons of irritable constitution, or otherwise out of health; but generally in the case of bites of ordinary scorpions or centipedes inflicted on healthy subjects, the suffering is local and soon passes away.

TREATMENT.—A variety of remedies have been recommended for scorpion-poisoning. Probably the application of a ligature above the bitten part, or a cupping-glass, or suction of the wound, as in snake-bite, might be useful. Some authorities recommend that the wound should be scarified, and emollient ointments and poultices applied. Suction of the wound, and the application of salt water, vinegar, ammonia, alum, ipecacuanha, spirit of camphor, eau de Cologne, tobacco-water, turpentine, tincture of iodine, alcohol, the leaves of cruciferous plants made into poultices, solutions of opium and lead, or other sedatives, will lessen pain and irritation. The use of diffusible stimulants, opiates, or other sedatives may be necessary, and such surgical interference as suppuration or cellulitis may require.

7. **Arachnida.**—**DESCRIPTION.**—Some spiders are venomous, and certain of the larger tropical forms are capable of inflicting painful bites. The poison-apparatus of spiders consists of falcæ or modified mandibles or jaws, the last joint of which is a hard curved fang, with a fissure near the point; there is an elongated poison-sac and duct in which the venom is elaborated and thence transmitted to the

fang, by which it is inoculated into the flesh of its prey.

EFFECTS.—The venom of spiders is a very active principle, and apparently is capable of rapidly destroying the life of the small creatures on which the spider feeds. It also causes symptoms of poisoning in man and the lower animals. Probably all the species have some venomous secretion, but it is only the larger kinds that are obnoxious to man. It may be noted that while the fangs of one section of spiders move laterally, those of the Mygalidæ move vertically. There are several species. Those reputed venomous are tropical.

Lycosa tarantula is reputed to cause extraordinary symptoms. It is poisonous, but there is no reason to believe that its effects exceed a certain amount of local irritation. See TARANTISM.

There are numerous families, genera, and species of spiders, all probably possessing an irritating fluid; but it is only the larger kinds that do so to any extent, and there is no very positive proof that even in tropical climates they inflict the grievous injuries ascribed to them, though the venom is very fatal to the creatures on which they prey.

The popular notions that the spider is very poisonous when swallowed, and that its web possesses medicinal properties, are probably exaggerated, if not altogether untrue. One species of red spider, however—perhaps a mite—called *coya*, in Popayan, is very poisonous; the juices of its body when crushed, coming into contact with the punctured skin, causes tumours, or even, it is said, death. This is no doubt an exaggeration, but it is probable that the juices are acrid and irritating, and it is therefore better not to crush these animals when detected on the person, but to brush or blow them away.

In India, a streak of almost erysipelatous redness of the skin coming on rapidly is often attributed to a spider. No one has defined the species; it is possible that it may be analogous to that just referred to.

TREATMENT.—The treatment of spider-bites is similar to that of centipedes and scorpions.

8. Acarina.—DESCRIPTION AND EFFECTS.—Some mites have the power of causing considerable irritation by a secretion ejected on the surface, or injected into the wounds they make in their burrowing operations with claws and mouth.

The *Tetranychus autumnalis*, *Leptus autumnalis*, or Harvest Bug, is brick-red in colour, and very minute. It is bred on plants, but leaves them to fasten on to animals, especially man, when it adheres firmly, and causes swelling, great irritation, and severe itching, if in numbers. The intense irritation causes fever. The symptoms are not unlike the sting of a nettle, erythema or even blistering being caused. The *leptus* is covered with hairs, and effects entrance into the skin with its claws, and thus gives rise to the great irritation, which is probably aggravated by some acrid excretion. These animals are found in Britain, France, and other parts of Europe; varieties of them in the tropics, for example, in Brazil, Honduras, on the Mosquito Coast, and in the West Indies. The *T. irritans* of the Mississippi valley causes great irritation in the same way.

TREATMENT.—The treatment is to extract the bug with a needle or the point of a knife, and then apply some soothing lotion.

Argas persicus, a gamosid, known also as the *Teigne de Miana* (venomous bug of Miana), is common in Persia. It is found in houses, and it is said that its puncture produces serious symptoms, such as convulsions, delirium, and gangrene, or even death. This is an exaggeration, though probably it is true that local irritation, and perhaps some constitutional disturbance, may be caused. It is blood-red in colour, spotted with white on the back, the feet yellow. *Argas moubata*, a native of Angola, is said to have much the same properties.

The *Argas talaje* of Guatemala produces great irritation. It bites like an ordinary bug, and the punctures are followed by great irritation, swelling, and pain. It lives in holes in the bamboo walls, or similar crevices, and issues at night to attack the sleepers.

9. Hemiptera.—Some of the *Geocorysæ* and *Hydrocorysæ*, or land- and water-bugs, have irritating properties, and also an offensive odour; they have a suctorial mouth, armed with a grooved instrument or rostrum for piercing the skin.

Cimex lectularius, the bed-bug, causes much irritation, and in some persons inflammatory action in the bitten part. The effects are transient.

Notonecta and *Nepa*, common in pools of water in our islands, are also capable of inflicting a painful puncture. *Cimex nemorum* causes nearly as much pain by its puncture as the sting of a wasp. The wheel-bug, *Reduvius serratus*, of the West Indies, gives an electric shock to the person it touches. St. Pierre mentions a species of bug in the Mauritius whose bite is as venomous as the sting of a scorpion. The *Benchucha*, or great black bug, of the pampas of South America, is more obnoxious, it is said, than the common bed-bug.

10. Aphaniptera.—*Pulicidæ* or Fleas comprise several families. *Pulex irritans*, the common flea, is universal. It varies much in size and colour; some are almost black and very large, and are found on the sandy shores of the Mediterranean. There are many species, such as *P. canis*, *P. musculus*, *P. vespertinus*, and *P. penetrans* of the West Indies and South America, known also as the jigger or chigoe (see CHIGOE). Though the irritation of the flea-bites is chiefly due to the wound, there is reason to believe that this is aggravated by the presence of some irritating secretion. No special treatment, beyond the application of ammonia, which may be useful, need be described.

11. Diptera.—DESCRIPTION AND EFFECTS.—To this order belong the gnats, mosquitoes, pipsas, sand-flies, and gad-flies, all more or less dreaded for their bites. They have a proboscis composed of a grooved and flexible sheath, through which long, slender, sharp darts are protruded, that pierce the skin and inoculate some venomous secretion, though its nature is not known. They draw blood, and raise white lumps or swellings; some, such as the pipsa of the Cossiah Hills, India, leave a livid spot of effused blood, which gives to the sufferer the appearance of a purpuric rash. They swarm in many countries, generally near water. The principal forms are the *Culex pipiens*, *C. reptans*, *C. mosquito*, *C. laniger*, and the whame-fly, *C. tabanus*. Some of these are formidable insects, and are insatiable blood-suckers. The *tsetse* or *timb* (*Glossina morsitans*) of Africa is one of the most remarkable. The bite of this poisonous insect is almost certain death to the horse, ox, or dog;

though it appears not to trouble man more than by causing slight irritation.

The female *Simulium*, or sand-fly, is irritating to man, the bite often giving rise to painful swellings. The pipsa is probably a *simulium*. It appears from the great irritation and the swelling that follows the puncture of most of these insects that some acrid secretion is injected into the wound. In young, full-blooded persons, especially recent arrivals in India or the tropics, the irritation caused by mosquito-bites is often so severe as to give rise to violent inflammatory symptoms, resulting in suppuration or ulceration, and even gangrene.

TREATMENT.—The application of common salt, solution of ammonia, soda, potash, lead, oil, ipecacuanha, or alum combined with opium, allays irritation in the first stage. The more violent inflammatory symptoms are amenable to ordinary surgical treatment. Camphor, pulegium, and lime-juice, applied to the skin, are all regarded as preventives.

12. Hymenoptera. — **DESCRIPTION AND EFFECTS.**—A number of species that secrete poison are found among the different families of hymenoptera, including bees, wasps, and ants. See **STING**.

They are distinguished by the presence of an ovipositor in the female, which is used not only for depositing the eggs, but as a weapon for injecting venom. It consists of two valves as a sheath, and three bristles which form a grooved sting. Through this groove the poison is injected into the wound, the ovipositor being connected with a poison-gland at its base.

Formicide.—Ants include *Formica smaragdina* and many others. The sting of the ant causes considerable irritation, especially if there are many. It has been suggested that formic acid is the irritating principle. There are several venomous species of ants, black and red, of various sizes. Some of the larger forms in the tropics are capable of inflicting a very painful injury. Some ants have no sting, but eject a fluid which irritates the skin.

Vespidæ.—The females and workers of the wasps and hornets are provided with a poison-sac and sting. *Vespa vulgaris* is a type of the tribe *Crabro*. It lives in communities. Its sting produces much irritation, pain, and swelling, especially when inflicted on the face, or where the cellular tissue is loose.

Apidæ.—True bees, and the *Bombidæ* or humble bees, have similar properties, their sting producing very much the same effect as that of the wasp.

Some of the parasitic *Hymenoptera* inject a poison into the wound made by their ovipositor. The best-known instance is that of the genus *Ophion*. The genus *Paripala* also injects a poison in the same way, and probably others of the *Ichneumonidæ*.

TREATMENT.—Many remedies of a simple nature have been recommended to allay the pain and irritation caused by the sting of the wasp and bee, such as vinegar, eau de Luce, ammonia, tobacco-juice, solution of soda or potash, oil, indigo, eau de Cologne, alum, and all those recommended in scorpion-stings (see **STING**). In cases of venomous stings, where constitutional disturbance is induced, stimulants or sedatives may be necessary; and as the sting is liable to be left in the wound, it ought to be picked out. In cases of wasp- or bee-stings in the mouth or throat, which may happen when children bite a peach or other fruit that conceals

a wasp, severe consequences may arise from the œdema that supervenes, and extends to the glottis. An emetic is then useful, along with the ordinary treatment of œdema; laryngotomy may become necessary. In other cases, should violent symptoms intervene, surgical aid may be required to relieve tension, or give exit to matter. Such untoward results, however, are happily rare.

Mutilla coccinea, a native of the warmer parts of North America, is said to produce loss of consciousness within five minutes of the infliction of its sting, life being in danger for some days afterwards.

13. Lepidoptera.—A stinging lepidopterous insect has been found. The species is not mentioned (F. Smith). The bee-moth of the Cape of Good Hope is said to defend itself with a sting. Though the majority of the perfect insects of this tribe are harmless, some of the caterpillars appear to be possessed of irritating properties, residing in the fine hairs with which they are cased, and which, being sharp and brittle, break off and remain in the skin, causing irritation mechanically; but also probably from the presence of some acrid substance concealed within the hairs. In Ceylon, a greenish hairy caterpillar, longitudinally striped, probably of the genus *Bombyx*, which frequents the leaves of *Hibiscus populneus*, alighting on the skin, causes as much irritation as the sting of a nettle. The larva of *Neara lepida* has similar properties. It is short and broad, of a pale green, with fleshy spines on the upper surface, each of which is charged with venom that occasions acute suffering. The larvæ of *Adolia* are also armed with venomous hairs. Another, not uncommon in certain trees in the terai of the Himalaya, is a dark-coloured hairy caterpillar, which is apt to fall on people and cause intense irritation. It is known as the *Komlah*, but the moth that produces it is not known. In schoolboys or others who handle the common 'woolly bear' caterpillar an erythematous rash may be produced.

14. Coleoptera.—Several beetles have acrid secretions capable of exciting great irritation and inflammation, raising blisters, and if absorbed causing painful strangury and great urinary irritation. Such are *Mylabris Cichorii* of India, *Cantharis* or *Lytta*, or *Meloe vesicatoria*, *Lytta gigas* of Senegal, *Lytta vittata* of America, and *Lytta ruficeps* of Chili.

15. Echinodermata.—The long sharp-pointed spines of some of the echinids are capable of inflicting painful punctured wounds, but convey no true venom. Whether, as in the case of some spiny fishes, there may be an irritating mucous secretion inoculated is uncertain.

16. Cœlenterata.—Some of the *Medusæ* or jelly-fish have the power of stinging. The poison-apparatus is placed in certain tubercles on the surface. These contain a collection of granules, among which are small vesicles. Within these corpuscles or nematocysts a spiral thread is found, which bursts out on pressure. These corpuscles are found in the mucus exuded by the creature, and to them is attributed the urticating power it possesses. There are several stinging species, some found on our own coasts, others in other seas. It is the larger forms generally that are venomous, the small ones having no effect on man. *Cyanea capillata* of our seas is a rather formidable creature, and the terror of bathers. It has a broad tawny disk, and a long train of ribbon-like streamers floating after

it; it makes its way through the waters; and whoever comes in contact with these trailing trains soon writhes in torture, the effect produced being not unlike that of the nettle.

Physalea pelagica, the 'Portuguese man-of-war,' has similar properties. It causes severe and stinging pain, extending up the limb, with feverishness, which has been known to continue for some hours, white wheals forming on the skin, as in urticaria. Several other medusæ possess these properties, and hence they have received the name of *Acalephæ*, or sea-nettles. The application of vinegar or olive oil is said to remove the unpleasant symptoms.

The *Actinia*, or sea-anemones, and the hydroid polyps, appear to possess a similar power, and are provided also with thread-cells. They cause urtication of the human skin when brought in contact with their tentacles. The *Sagartiade* furnish examples of sea-anemones with this property. The effects, however, of any of them are transient. In some parts of Europe the *Acalephæ* have been used therapeutically as counter-irritants, by being brought in contact with the patient immersed in a salt-water bath.

JOSEPH FAYRER.

VENOUS HUM.—A peculiar murmur heard on auscultation of the larger veins, especially those of the neck and chest, in anæmia, and in cases of interference with the flow of the blood through these vessels. See PHYSICAL EXAMINATION.

VENTNOR, in the Isle of Wight.—A mild, dry, tonic climate. Mean winter temperature for forty years, 42–43° F. Sheltered from N., N.E., and E.; exposed to S.S.E. and S.W. winds. See CLIMATE, Treatment of Disease by.

VENTRICLES OF THE BRAIN, Diseases of.—SYNON.: *Maladies des Ventricules du Cerveau*; Ger. *Krankheiten der Gehirnhöhlen*.—The chief morbid states of the ventricles of the brain are (1) New-growths, Degenerations, and Inflammatory changes in the *lining membrane* (ependyma) and *velum interpositum*; and (2) Accumulations of blood, pus, and serum in the *ventricular cavity*. The more important of these changes are fully described in special articles.

1. **Diseases of the Lining Membrane and Velum.**—In old age, and in some degenerative brain-diseases, such as general paralysis, the ependyma of the ventricles becomes *thickened*. The surface is smooth, or, in some cases, covered by minute warty granulations. Some of the latter may attain the size of a pea, and constitute small fibrous tumours. Similar changes are sometimes found when the brain has been subjected for a long time to passive congestion. In rare cases the thickened membrane has been found *calcified* in places.

A few *morbid growths* have been met with in the ependyma, the most common being the granulations of tuberculosis, which have been found both on the lining membrane and the choroid plexus. The latter and the velum interpositum frequently present thickening, and undue adhesion to the ependyma. In rare cases *fatty growth* has been met with in this situation. The choroid plexus may present partial *fatty degeneration*, and frequently contains *corpora amylacea*. Aggregations of *brain-sand* (see CONCRETIONS) are common in the choroid

plexus, and occasionally occur in the lining membrane.

Cystic degeneration is the most common morbid appearance in the choroid plexus, especially in that part which is within the descending cornu. The cysts are clear, delicate, colourless, transparent, from the size of a pea downwards. They consist of delicate cells pressed together; these are simply normal cellular elements of the part which have undergone a peculiar degeneration. In some of the larger cysts, these cells have been destroyed in the centre, so that a true fluid-containing cyst remains.

Adhesions sometimes take place between contiguous surfaces of the lining membrane; these may cut off one posterior cornu from the rest of the ventricle, and it may thus be obliterated by the union of its walls.

The ventricles may undergo passive *congestion* in common with other intracranial structures, or from pressure upon the veins of Galen, which carry the blood from the velum interpositum. The latter may cause effusion of fluid into the ventricles, but its influence was formerly over-estimated. The cause which obstructs the veins is generally a growth which also hinders or prevents the escape of fluid from the third into the fourth ventricle.

Inflammation involves both the ependyma and the velum interpositum. It is rarely confined to the ventricles, still more rarely to one. Commonly it is part of a general meningitis. The ependyma and the velum are thickened and pulpy, being infiltrated with cells of new-formation. The velum is always injected; the ependyma may be injected or pale. Occasionally a 'false membrane' is found upon its surface. The tissue of the brain beneath the ependyma is softened, and may be injected. The fluid in the ventricles is increased in quantity, and is turbid from pus and exudation-cells, and even (it is said) from the *débris* of nerve-fibres. The inflammation, of which this is part, is usually tubercular and fatal; but, when simple, it may pass away, the ependyma and velum remaining thickened and adherent. See MENINGES, CEREBRAL, Inflammation of, Tubercular, p. 979.

2. **Intraventricular Accumulations.**—*Hæmorrhage* rarely occurs directly into the ventricles; it has resulted from the rupture of a vein by injury; but blood often enters them from an effusion into the cerebral substance, an ordinary hæmorrhage bursting into the cavity. In meningeal hæmorrhage blood may even pass up into the ventricles from the subarachnoid space, by the passages through which it escapes. True *pus* may be found in the cavities, from the bursting into them of a cerebral abscess; and a purulent fluid may result from inflammation of the lining membrane. A slight amount of *serum* results from inflammation, but is rarely considerable, unless the escape of that secreted by the choroid plexus is prevented by the closure of the passage to the fourth ventricle or by the obliteration of the foramina in the membrane closing in the fourth ventricle, by which its cavity communicates with the subarachnoid space (Hilton) (see HYDROCEPHALUS). In atrophy of the brain, the fluid within the ventricles (as beneath the arachnoid) undergoes a considerable compensatory increase, which has been without reason regarded as a cause of symptoms.

Lastly, by violent commotions of the brain the septum lucidum may be *ruptured* (Wilks and Moxon).

Ventricular hæmorrhage and *Hydrocephalus* are described in special articles. See BRAIN, Hæmorrhage into; and HYDROCEPHALUS.

The other conditions mentioned are marked by no distinctive symptoms, and call for no special treatment. W. R. GOWERS.

VERBIGERATION.—See p. 767.

VERDIGRIS, Poisoning by.—See COPPER, Poisoning by.

VERMES (Lat. worms).—See ENTOZOA.

VERMICIDES (*vermis*, a worm; and *cædo*, I kill).—A group of anthelmintics which kill worms. See ANTHELMINTICS.

VERMIFUGES (*vermis*, a worm; and *fugo*, I expel).—A group of anthelmintics which expel worms, but do not necessarily kill them. See ANTHELMINTICS.

VERRUCA (Lat. a wart).—See WART.

VERRUCA PERUANA.—See CARRION'S DISEASE.

VERTIGO (*verto*, I turn).—SYNON.: Giddiness; Dizziness; Swimming of the head; Fr. *Vertige*; Ger. *Schwindel*.

DEFINITION.—The consciousness of disordered equilibration.

PHYSIOLOGICAL RELATIONS.—To understand vertigo normal equilibration must be briefly considered. The equipoise is maintained by a sensorimotor mechanism. The co-ordinating centre is the cerebellum; the afferent or sensory apparatus consists of visual, tactile, muscular, and labyrinthine impressions; the efferent or motor mechanisms are the muscles, chiefly those of the head, neck, spine, and lower extremities. Derangement of any part of this apparatus may lead to vertigo, by the interruption of its power of adjustment being appreciated by cortical centres. Vertigo is often associated with reeling or staggering, and is incorrectly said to cause it. Actually vertigo is the consciousness of disturbed locomotor co-ordination—a rudimentary disorder of co-ordination of locomotive movements (Hughlings Jackson)—while reeling is an adaptive effort to preserve the equilibrium. It is quite possible that cortical centres in an unstable condition may give rise to vertigo, without there being any disease or disturbance of subordinate centres, or of peripheral impressions. It is probable that migrainous vertigo is due to discharging lesions of the cortical centres concerned in the consciousness of equilibratory perceptions. Experimental researches and observations in disease have established the conclusion that the semicircular canals take an important share in normal equilibration; injury and disease of these parts occasioning locomotive inco-ordination, temporary when one side only is deranged, permanent when both sides are involved. It has been demonstrated by Flourens that injury of each canal is followed by definite locomotive disturbance, causing the body to tend to fall, or actually to fall, in a definite and precise direction, forwards, backwards, or to one or other side, according to which of them is injured. The sensory impressions originating in the semicircular canals are caused by varying tension of the endolymph, communicated to the

vestibular division of the auditory nerve spread out on the ampullæ of the membranous canal. Variations in labyrinthine tension may be produced by alterations in the position of the head, by differences in the vascular tension of the labyrinthine blood-vessels, and by the varying pressure in the middle chamber of the ear, induced by obstruction of the Eustachian tube, spasm of the tensor tympani muscle, and other causes; and it may also be due to disease of the labyrinth itself, or communicated to the labyrinth. Moreover, it is asserted that 'if the horizontal canal is laid bare, and the membranous canal opened so as to expose the endolymph, blowing gently over the opened canal with a fine glass cannula will produce a definite movement of the head, which is turned to the one side or to the other, according as the current of air drives the endolymph towards or away from the ampulla. From this it is inferred that a movement of the endolymph over, or an increased pressure of the endolymph on, the surroundings in the ampulla gives rise to afferent impulses which in some way determine the issue of efferent impulses leading to the movements of the head' (Michael Foster). In some cases of aural vertigo, the giddiness is much influenced by the position of the head, and it is only in certain positions that this symptom is produced. This is readily accounted for on the supposition that the disease causing it is in the semicircular canals, which are so arranged as to lie in the three directions of space; and change in the position must affect the pressure of the endolymph on the various ampullæ, resulting in adjustment of position when the co-ordinating centres are in proper working order, but when these are deranged leading to the consciousness of disordered equilibration or vertigo. Further, the evidence supplied in a *post-mortem* examination of a case of Ménière's disease, where hæmorrhagic inflammatory material was found in the semicircular canals, and no disease of the brain, proves that disease of the semicircular canals *per se* may give rise to vertigo. Visual and tactile impressions are liable to be deranged in many ways, for instance, by unexpected or unusual movements, as in swinging, being at sea, &c.; by local disease of the visual and tactile apparatus; and by disease in the nerve-trunks and spinal cord, interrupting conduction from the periphery to the centre. By disturbances in visual, tactile, or labyrinthine impressions the equilibrating centre is uninformed or misinformed, and inco-ordination results, outwardly shown by reeling or falling, and inwardly by the sensation we call vertigo. Loss or perversion of visual or tactile sensations may be compensated for if the two remaining sensory processes continue intact, but nothing compensates for entire loss of labyrinthine impressions (Ferrier).

PATHOLOGY.—Vertigo may be excited by variations in the local or general blood-pressure, which cause variations in the labyrinthine tension, as in anæmia, gout, and other affections. It may be excited reflexly by disease of the nose (*vertigo a naso læso*, Joul), or by disease of the larynx. The symptom is also produced by certain drugs, such as quinine, salicin, and the salicylates, which may act on the labyrinth through the vascular system, as is rendered probable by the deafness and tinnitus accompanying it, but which may be due to its action on the auditory nucleus, or the cortical centres concerned in consciousness of auditory perceptions. Vertigo may be excited by sending a galvanic

current through the head from one mastoid process to the other, but the exact mode of its action is not known.

VARIETIES.—The important forms of vertigo which occur in practice will be further considered under the following heads: (1) Ocular; (2) Auditory; (3) Gastric; (4) Nervous; (5) Epileptic; (6) Migrainous; (7) With organic brain-disease; and (8) Cardio-vascular.

1. Ocular Vertigo.—Vertigo is frequently caused by ocular disorders, and is often mistaken for serious cerebral disease. The simplest form is in paralysis of a single muscle, as the external rectus. The vertigo is not occasioned by the diplopia, but by the incorrect notion formed of external objects by the paralysed eye, due to what is known as 'erroneous projection.' The confusion thereby produced gives rise to vertigo, and often to reeling. One of the most important varieties of ocular vertigo is that occasioned by insufficiency of the internal recti muscles—*muscular asthenopia*. This is most commonly met with in myopia. During reading, these muscles, which have long been overtaxed by exertions to maintain the convergence of the eyes rendered necessary when looking at near objects, suddenly give way under the strain; they relax, the eyeballs turn out, and the letters on the page become indistinct, run into each other or overlap, and a sense of confusion and giddiness occurs. It is usually accompanied by aching at the back of the eyes, headache, and sometimes by nausea. Muscular asthenopia may occur also with hypermetropia, and as a sequel to exhausting diseases, such as fevers and diphtheria. Many instances are recorded where, owing to these causes of vertigo not being recognised, serious errors of diagnosis and treatment have resulted. For the diagnosis of the particular optical defect and treatment, the reader is referred to the article VISION, Disorders of.

2. Aural or Auditory Vertigo.—**SYNON. :** *Vertigo ab Aure Læsa*; Labyrinthine Vertigo; Apoplectiform Vertigo; Menière's Disease.

Under the term 'Menière's disease' is grouped a class of cases in which vertigo is caused by perversion or abeyance of the labyrinthine function. The labyrinthine disturbance may be caused either (1) *directly* by an affection of the labyrinth, such as (a) hæmorrhage, (b) congestion and inflammation; or (2) *indirectly*, by (a) disease of the middle ear (otitis media), (b) obstruction of the Eustachian tube, (c) spasm of the tensor tympani, or paralysis of the stapedius, or (d) irritation or obstruction of the external auditory meatus, and pressure on the membrana tympani, as by cerumen, foreign bodies, or by syringing the ears, especially when the membrana tympani is perforated. In Menière's disease, strictly speaking, there is always coincident disturbance in the functions of the semicircular canals and cochlea, as indicated by the three most important associated symptoms—vertigo, tinnitus, and deafness. Accompanying these cardinal symptoms there are accessory phenomena, due to secondary visceral disturbance, namely, pallor, faintness, and nausea or vomiting—a condition of syncope. Aural vertigo is rare in young persons, and is chiefly met with in the later half of life. It is more frequent in men than in women.

The disease makes its appearance, in a person apparently quite well, or the subject only of some chronic aural disease, with a loud noise in the ear,

compared by different persons to the whistle of a steam-engine, the firing of a gun, or the roar of the ocean. When a person, as not infrequently happens after the first attack, has an habitual noise in the ear, this at the time of the attack is sometimes, but not always, greatly exaggerated. The noise, which is wholly or principally in one ear, is soon followed by the feeling of giddiness. This is generally of a high degree, causing a sensation of movement of surrounding objects in some one direction, a feeling of translation of the patient's body in the same direction, or actual movements of the body. The movement, whether apparent or real, is usually *from* the side on which the ear is affected. In recurring attacks the movements, whether of objects or of the individual, are nearly always in the same direction. Usually the sensation of movement is from behind forward, or to one or the other side, or the patient has a feeling of rotation round a vertical axis. When in bed, the room, bed, and occupant are felt as if turning round and round, or rising or sinking. Accompanying the vertigo there is reeling, and the patient clings to surrounding objects for support. In some cases the movement is too rapid for the patient to obtain security in this way, and he is thrown to the ground, sometimes with such violence as to occasion serious injuries. One patient described the sensation as 'if his heels went up into the air.' When falling takes place, it is usually forwards or to one side. It is, however, to be especially remembered that, except in rare cases, there is no loss of consciousness; the patient being able immediately after the attack to describe the sensations he experienced, or even to answer questions in the attack itself. In some cases deafness, more or less complete, but sometimes transitory, follows the vertigo at an appreciable interval, and in some cases it is altogether wanting. Following the tinnitus and vertigo, in more or less rapid succession, there occur nausea and in most cases vomiting, accompanied by pallor of the face; the skin becomes cold and covered with a clammy sweat. In some cases oscillatory movements of the eyes are observed. It is generally asserted that objects appear to move in a direction opposite to that of the ocular movements. This is not universally true; and probably, contrary to the statements of most writers, the apparent movement of objects is in the same direction as the observed movements of the eyes. Gradually the attack passes off; the noises in the ear lessen, but deafness is left behind. The body recovers its warmth, and the pallor subsides, but vertigo and vomiting may persist for some hours or even days, both being aggravated or induced by rising from a horizontal position. Slight attacks may only last a few minutes. In cases where there is a direct lesion of the labyrinth, a certain degree of deafness—a limitation of the field of audition, that is, the loss of certain sounds in the musical scale—and tinnitus remain. The patient is in all other respects well, except for the dread of a recurrence of the attack. Occasionally, however, some amount of vertigo and reeling persist, liable to be aggravated by gastric derangement. The writer has known a patient who had extremely severe and typical attacks, who both previously and subsequently suffered from functional dysphagia between the attacks—a full-sized bougie could be easily passed. It was evidently due to disturbance in the vagus-nucleus. A patient rarely escapes with one attack. Subsequent attacks are separated by dis-

tinct intervals, but in severe cases these may become less and less, until a permanent vertiginous state, of a most distressing character, may be reached, liable to paroxysmal exacerbations. In such very grave cases spontaneous cure may occur on the establishment of complete and permanent deafness, or relief may be obtained by therapeutical measures. When the labyrinthine disturbance is secondary to disease of some other part of the auditory apparatus, removal of the primary disease, as cerumen or tympanic catarrh, will, when practicable, promptly remove the symptoms, and the attacks may not recur.

DIAGNOSIS.—Ménière's disease has to be distinguished from epilepsy, apoplexy, gastric derangement, and other forms of vertigo. From all these it is distinguished by the almost invariable co-existence of tinnitus, deafness, and vertigo; with, in addition, a tendency to syncope and nausea, or vomiting. The concurrence of the first three symptoms renders it probable that the labyrinth is involved, a point which will be further established by testing audition with a tuning-fork and watch. The vertigo is generally a sensation of movement in a certain definite and uniform direction. There is never numbness, tingling, or any sensations analogous to an aura. As to the diagnosis of the nature of the labyrinthine affection, whether primary or secondary, some rules have been laid down by authorities. If a person who has formerly heard well becomes suddenly deaf or hard of hearing, with the symptoms of an apoplectic attack, and if there is at the same time an uncertain and staggering gait, but no symptoms of paralysis in the nerve-tracts, and if the examination shows a normal membrana tympani, and perfectly permeable Eustachian tube, we may believe with great probability that there is an affection of the labyrinth (Trötsch). Deafness and tinnitus occurring without vertigo indicate an affection of the middle ear. Vertigo and tinnitus without deafness may be due to a similar affection. Vertigo, tinnitus, and deafness are certainly due to an affection of the labyrinth. When the vertigo is markedly influenced by changes in the position of the head, it is, in the writer's opinion, strong evidence that the disease is in the labyrinth. Careful otoscopic examination should be made, the permeability of the Eustachian tubes tested, and the tuning-fork and watch employed to ascertain the condition of the conducting apparatus, before an exact opinion can be formed as to the seat and nature of the disease. Vomiting, following the ingestion of some rich or indigestible food, may be so severe and lasting as to monopolise attention, and the vertigo and tinnitus may not be complained of. In such a case, a mistake may readily occur in a first attack.

PROGNOSIS.—When the labyrinthine affection is due to some remediable defect, the disease will subside on removal of the cause, such as cerumen or tympanic catarrh; hence the great importance of an exact diagnosis as to the nature of the case. When the lesion is primarily of the labyrinth, a certain degree of deafness and tinnitus is nearly always left, and recurrence of the attack is to be anticipated, though some cases recover completely under treatment. In some instances the tinnitus and vertigo may cease on the patient becoming completely deaf.

TREATMENT.—In the attack, and for a short time following it, the recumbent position should be strictly maintained. Bromide of potassium or ammo-

nium, in ten- to twenty-grain doses, should be administered, and small pieces of ice swallowed. Belladonna is also recommended, especially in combination with bromide. Next, any gastric derangement should be corrected, for in some cases such derangement excites a paroxysm in a person predisposed to it by some aural affection, insufficient alone to induce an attack. Alkalis and vegetable bitters, with or without bismuth, will generally be useful for this purpose. Any abnormal local condition must be treated. Subsequent to the attack quinine in full doses, 3 to 5 or 10 grains three times a day, perseveringly used, is sometimes attended by the best results (Charcot). The writer has seen many cases which corroborate this statement. Gelsemium and salicylate of sodium have been found useful (Gowers). Counter-irritants, including the actual cautery, applied to the mastoid region, have proved serviceable in some cases, and may be used in addition to other measures. Threatened attacks of aural vertigo may be often avoided, especially in cases in which arterial tension is high, by a dose of calomel, and it is a good plan to give a patient liable to attacks calomel-powders (gr. iij or v) to be always carried in the pocket-book, and order one to be placed on the tongue when any threatenings occur.

3. Gastric Vertigo.—**SYNON.** : *Vertigo a Stomacho Læso*.

Vertigo, occasionally of a high degree, sometimes accompanies chronic gastric derangement. It is more common with slight than with grave affections of the stomach, but has been met with in well-marked organic disease of this organ. It sometimes occurs soon after a meal, but more often when the stomach is empty (Trousseau). At the same time it is now generally agreed that cases of gastric vertigo are much less common than Trousseau's description would lead one to believe. In the majority of such cases some ear-disease is revealed by careful examination. Associated with the vertigo are usually pain and a feeling of fulness in the stomach, increased by food; heartburn; eructations; vomiting; flatulence; and pain in the left hypochondrium and chest. The bowels may be torpid, or diarrhoea may be present. The patient often suddenly experiences a swimming in the head, objects may appear to revolve, the patient's gait becomes tottering, and he may even fall. Often there is constrictive headache, faintness, and pallor with nausea, and sometimes troublesome vomiting, but there is no loss of consciousness. Visual hallucinations may be present, and buzzing in the ears experienced, but there is no deafness. The vertiginous symptoms may so predominate that the gastric symptoms may not be complained of; but treatment directed against dyspepsia cures the vertigo. When predisposing gastric disturbance is present, trivial causes, such as looking at objects which lead to confused visual impressions, may excite an attack, but this may also arise spontaneously. In many cases relief is obtained by the recumbent position, but attacks may occur when the patient is lying down.

DIAGNOSIS.—This form of vertigo is diagnosed from epilepsy by absence of loss of consciousness; and from labyrinthine vertigo by the absence of deafness, and the physical signs of aural disease. It cannot be concluded that the vertigo is essentially gastric without thorough examination of the ears, for, as already stated, vertigo may be excited by gastric disturbance when there is labyrinthine affection insufficient alone to determine an attack.

It must also be remembered that signs of gastric and intestinal derangement are induced in Menière's disease, and may be so prominent as to cause the aural affection to be overlooked.

4. **Nervous Vertigo.**—**SYNON.** : Fr. *Vertige Nerveuse*.

Not uncommonly, vertigo is one of the most troublesome symptoms of nervous exhaustion and depression. This occurs in persons unduly taxing their nervous powers by severe intellectual strain, especially when combined with anxiety, or by sexual excesses. It occurs also from the depressing effects of the immoderate use of tobacco, alcohol, and tea. The vertigo rarely reaches a high degree, manifesting itself by a sensation of confusion, or of objects revolving, only occasionally by the feeling of a tendency to fall. It may be associated with a slight reel, but more often the patient feels as if he were walking unsteadily, when there is no perceptible peculiarity of gait. As a rule giddiness is only experienced in the upright position, but in some cases it occurs when the subject is recumbent, and the patient often complains of sudden and violent startings when just in the act of falling asleep. It is often intensified by an elevated position, and in large buildings and assemblies. Hence it is often experienced in church. It is peculiarly distressing, owing to the sufferer's emotional equilibrium being easily disturbed, and is frequently associated with a dread of impending cerebral disease—epilepsy, apoplexy, insanity, &c. There often co-exist gastric derangement and flatulence, with irritability of the heart, palpitation, and sleeplessness. There may be slight and temporary buzzing in the ears, but deafness is absent, and loss of consciousness does not occur. In these respects it is readily distinguished from Menière's disease and *petit mal*.

TREATMENT.—This variety is to be treated by removal of the cause—over-work, excessive sexual indulgence, or the abuse of alcohol, tobacco, or tea; by correction of any dyspeptic symptoms; and by the administration of nervine tonics, such as iron, quinine, or strychnine. Bromides should be avoided if possible.

5. **Epileptic Vertigo.**—Vertigo may occur in a slight fit of epilepsy, or at the commencement of a severe attack. The symptom may replace an epileptic fit, or may co-exist with epilepsy. It is more common in epileptic vertigo for the patient to imagine that he himself is moving or turning round, than for external objects to appear in motion (Russell Reynolds). If the vertigo is related to change of position of the head, it is probably labyrinthine. The latter is not usually accompanied by loss of consciousness, and is more apt to be followed by vomiting (Gowers).

6. **Migrainous Vertigo.**—Vertigo commonly constitutes one of the phenomena of megrim, occurring as a rule after the disorders of sight, touch, and speech, when these form part of the seizure, and either attends or follows the development of the headache (Liveing). Vertigo sometimes replaces the attacks of megrim. It is apt to occur on change of posture, or on suddenly turning the head. As a rule, migrainous vertigo is slight in degree, but it may be quite severe, and accompanied by nausea and vomiting. It is unassociated with noises in the ear, or with deafness. See MEGRIM.

7. **Vertigo in Connection with Organic Disease of the Nervous System.**—Vertigo sometimes accompanies disease of the cerebrum, both acute, as apoplexy, and chronic, as a tumour. There are reasons for believing that vertigo may be excited by cortical lesions, thus explaining epileptic and migrainous vertigo. Diseases of the cerebellum and of its middle crura are often attended by reeling gait, and sometimes by vertigo. This symptom sometimes accompanies the ataxy of tabes dorsalis, and is a symptom of many cases of insular sclerosis.

8. **Cardio-vascular.**—Disease of the heart accounts for some cases of vertigo, while in others the symptom is due to arterio-sclerosis; but apart from cardiac or vascular disease the symptom may arise from sudden alteration of the cerebral circulation, anæmia or hyperæmia of the brain; and women at the climacteric period often suffer from giddiness.

A peculiar form of disease has been described by Gerlier, which he has called 'paralysing vertigo,' and which subsequent writers have named 'Gerlier's disease.' It consists of three groups of symptoms: (1) muscular weakness in the muscles of the neck or trunk and legs, and occasionally in the hands; (2) pain in the neck and occiput; (3) ocular symptoms, of which ptosis is the most common. Occasionally diplopia and amblyopia are present. Dizziness or mistiness before the eyes is often present, more rarely rotatory vertigo. The attacks usually occur about once or twice a day, and between the attacks the patient is well. The disease has only been observed in some Swiss villages on the French frontier, where the malady has received the name of *le tourniquet*. It appears to have had a kind of epidemic prevalence. Its nature is very doubtful, but it appears to be a functional disturbance or psychosis. No evidence of organic disease has been present in the cases recorded.

An affection described by Miura, in Japan, and known as *kubisagara*, is probably the same as 'Gerlier's disease.' See p. 1740.

STEPHEN MACKENZIE.

VESICANTS (*vesica*, I blister).—A class of counter-irritants which produce blisters. See COUNTER-IRRITANTS.

VESICLE (*vesicula*, diminutive of *vesica*, a bladder).—**SYNON.** : Fr. *Vésicule*; Ger. *Bläschen*. A vesicle is a lesion of the skin produced by a small localised collection of fluid in the epidermis. The usual contents are blood-serum with fibrin, leucocytes in varying numbers, degenerated epithelial cells, and in many cases micro-organisms. If the leucocytes are in large numbers, the contents of the vesicle are purulent, and the name 'pustule' is applied; when the vesicles become confluent, or are of large size, the name 'bulla' is made use of. Vesicles may arise in any of the layers of the epidermis. Frequently they arise immediately under the stratum corneum or in the upper layers of the stratum granulosum. Such vesicles are found in the early lesions of eczema, or in some of the varieties of pyoderma. As a rule they contain micro-organisms in considerable numbers. They may also arise in the stratum granulosum or at the junction of this layer and the stratum mucosum. In such cases the vesicle may be surrounded by the stratum granulosum or may be covered by both the stratum granulosum

and the stratum corneum. The vesicles of dysidrosis frequently arise in this situation. They may also form at various depths in the stratum mucosum. With an uninjured horny layer the contents of the vesicles may be free of organisms.

Vesicles may be divided into compartments by strands of epithelial tissue, or umbilicated when rising round one of the ducts of the skin-glands or a hair-follicle. They may be developed in rings, as in erythema multiforme; in groups, as in herpes zoster; and may be acuminate, rounded, or flattened.

Not infrequently the red blood-cells, as well as the serum, may be contained in the vesicles.

In certain diseases of the sweat-apparatus, vesicles may arise containing sweat only.

The vesicle may heal by absorption of the contents, or after their escape on account of rupture of the epidermis, followed by subsequent desquamation of the overlying epidermis. As the epidermis is reproduced no scar results from an uncomplicated lesion.

JAMES GALLOWAY.

VESICULAR EMPHYSEMA.—A form of emphysema of the lungs, in which the alveoli are distended with air. See LUNGS, Emphysema of.

VIABLE (Fr. *vie*, life).—SYNON.: Fr. *Viable*; Ger. *Lebendig*.—An epithet applied to a newly born child, to indicate its capacity for maintaining an independent existence. Viability has chiefly to be determined by the age of the fœtus, and by its condition as regards formation, health, and strength (see MONSTROSITIES; and FŒTUS, Diseases of the). It has also been supposed to depend in some measure upon the season of the year in which a child is born. The question of viability has important medico-legal bearings, for which reference should be made to works upon forensic medicine.

VIBICES (*vibex*, a wale).—SYNON.: Fr. *Vergesures*; Ger. *Striemen*.—A term applied to patches of discolouration on the surface of the body, somewhat resembling the marks of stripes or wales, and due to the presence of altered blood in the part. Vibices may arise either during life, as the result of a variety of causes (see EXTRAVASATION); or after death, as one form of cadaveric lividity or hypostasis. See DEATH, Signs of.

VIBRIO (*vibro*, I shake).—SYNON.: Fr. *Vibrion*; Ger. *Zitterthierchen*. See BACTERIA.

VICARIOUS (*vicarius*, in place of another).—This word signifies substitution, and in physiology and pathology implies that some part or organ performs certain functions, or is morbidly affected, instead of some other part or organ, thus becoming a substitute for it. The notion of vicariousness is chiefly associated with a discharge of blood, whether physiological or morbid. Thus it is very common to speak about *vicarious menstruation*, which is understood to mean that the discharge of blood that takes place normally from the uterus at the menstrual period either does not occur at all or only imperfectly, and that its place is taken by hæmorrhage from some other part, evidenced by epistaxis, hæmoptysis, hæmatemesis, or other forms of bleeding. The same idea is extended to morbid hæmorrhages, such as bleeding from piles, when this becomes habitual in an individual at frequent or regular intervals. It is supposed that bleeding may sometimes

take place from other parts as a vicarious hæmorrhage, instead of from the hæmorrhoids.

Again, discharges, whether normal or morbid, as of secretions, mucus, pus, or other materials, are believed by many to exhibit a vicarious relation to each other in some instances, coming from one part while ceasing or diminishing at another, and so on. This may be illustrated by expectoration and diarrhoea in phthisis, which appear to modify each other as to their amount in some cases of this disease. Further, secretions and excretions are regarded as acting vicariously with reference to each other. Thus some of the secretions of the alimentary canal are undoubtedly capable of acting mutually as substitutes, and this may be looked upon as an instance of vicarious action; while such a connection existing between the perspiration and urine is generally recognised.

Certain morbid conditions are also considered as having a vicarious relation. For example, congestion of, or hæmorrhage from, one part may take the place of congestion at another; or inflammation in one region may be the substitute for inflammation in another region.

Enlargement of one lung or one kidney after complete or partial destruction of the other—examples of a condition generally known as ‘compensatory hypertrophy’—may also be quoted as instances of vicarious action.

There is probably more or less truth in these notions of vicariousness, as applied in relation to physiology and pathology. In actual practice, however, no case ought to be regarded as belonging to this category, without careful and thorough investigation. It has happened that hæmorrhages supposed to be vicarious of menstruation have been important signs of grave diseases, such as gastric ulcer, or pulmonary phthisis. The principle may be of value in certain conditions as an indication for treatment.

FREDERICK T. ROBERTS.

VICHY, in Allier, France.—Thermal alkaline waters. See MINERAL WATERS.

VIDAGO, in Portugal.—Bicarbonated alkaline waters, with lithium and arsenic. See MINERAL WATERS.

VILLOUS GROWTH (*villus*, hair).—A growth composed of hypertrophied villi. See TUMOURS.

VIRGINIA SPRINGS, in Virginia, United States.—Sulphur-waters. See MINERAL WATERS.

VIRULENT (*virus*, a poison).—This word is generally employed to indicate great intensity or malignancy of a disease; for example, *virulent inflammation* and *virulent small-pox*. See also p. 721.

VIRUS (Lat.).—Literally this word signifies a poison, but in medical language it is used to designate any kind of specific organic poison. See INFECTION.

VISION, Defects of.—SYNON.: Fr. *Troubles de la Vision*; Ger. *Sehenstörungen*.

Sight may be defective as to perception of form, of colour, or of light; and the whole or only a part of the visual field may be affected. Sight is also disordered whenever *binocular single vision* becomes difficult or impossible (see STRABISMUS);

and when *visual endurance* is impaired. The terms 'vision' and 'sight,' as commonly used, indicate acuteness of vision, and refer to the perception of form at the yellow spot. In this article disorders (A) of *perception of light*, (B) of *perception of colour*, and (C) of the *visual field*, will be shortly alluded to; but attention will be chiefly given to (D) disorders of *acuteness of vision* caused by optical defects in the eyes.

A. Disorders of Perception of Light.—Perception of light is equally good in all parts of the retinal area, except the most peripheral zone, which appears to be blind. Impaired perception of light causes disproportionate defect of vision by dull light—'night-blindness.' It may affect the whole field, or only its periphery. It occurs chiefly in diseases of the outer layers of the retina, especially syphilitic retinitis and retinitis pigmentosa, and sometimes in chronic glaucoma. Lowered light-sense over the whole field occasions the symptoms in the peculiar disease known as functional or endemic nyctalopia (*torpor retina*). The opposite condition, day-blindness with true retinal photophobia, is much rarer and more obscure. It is usually congenital, and accompanied by nystagmus, amblyopia, and colour-blindness. See NYCTALOPIA; and HEMERALOPIA.

B. Colour-Blindness.—SYNON.: *Dyschromatopsia*; *Achromatopsia*; Daltonism.—This, when *congenital*, is usually not related to any other defects of vision. Congenital colour-blindness occurs with greater intensity and far greater frequency in males than in females (M. about 3·5 per cent.; F. ·2 per cent. or less). It is shown by inability to distinguish between certain colours owing to absence of one or other of the chief colour-sensations. The spectrum seen by a red-blind person may appear shorter at its red end than it does to one with normal colour-vision, and he sees in it only two chief colours. The red, orange, yellow, and green are all 'green,' the blue and violet are 'blue,' and the two are separated by a neutral or 'white' band at the part that to the normal eye is bluish-green. In green-blindness the spectrum is not shortened, but the red, orange, yellow, and green are 'yellow' of various shades, or sometimes 'red'; the neutral band is in the pure green. It is often impossible to distinguish red-blindness from green-blindness with certainty except by using the spectrum; and in ordinary examination the two forms are best grouped together as 'red-green-blindness.' In incomplete red-green-blindness, green, bluish-green, and often rose are confused with grey of corresponding shade, and red is confused with shades of brown, greenish-brown, and yellowish-brown. In a complete case full green and scarlet look identical, and dark yellows are more or less confused with them. The best test (of many) for ordinary use is Holmgren's, in which a skein of Berlin wool, of a particular colour and shade (green, rose, or red), is given to the patient, and he is required to match it with all the others which seem to him of the same or a similar colour, among a large bundle of skeins of many colours. He is not usually allowed to name the colours, because even the colour-blind often guess the colours of common objects correctly. A very pale, pure green is the first test used; and the colour-blind, even of slight degrees, will match with it not only other green skeins, but also shades of pale grey, buff, pink, or blue. Slight cases may easily be overlooked, unless

the wools are carefully selected, and the examiner practised. In deciding as to the fitness of a candidate for signal work it is often desirable to test with a lantern and coloured glasses in addition to the wools. Red and green are not well seen even by the normal eye, except at the central part of the visual field (i.e. the field for these colours is smaller than for white); but even at the periphery these colours are recognised if very brightly lighted and of large size.

Acquired colour-blindness often comes on in degenerative or inflammatory diseases which begin in the optic nerve. It is rare in diseases of the retina, and in glaucoma. Like the congenital form, it usually concerns only, or chiefly, red and green. It may affect the whole visual field of these colours, or only certain parts, a gap, or 'scotoma,' being present, on the area of which the red and green are not perceived in their true colours. When acquired colour-blindness is well marked in the whole extent of the field, in cases of disease of the optic nerve, the prognosis for sight is generally very bad; but if it be localised on a central scotoma, even though it may there reach a high degree, the prognosis is usually good. Progressive atrophy of the optic nerve, however, occasionally reaches a very high degree without any colour-defect.

C. Disorders of the Visual Field.—The visual field is the whole surface visible to one eye singly while at rest. It forms a concave surface, all the points of which are equidistant from, and perpendicular to, their corresponding points on the retina. In the outward and downward part it reaches to 95° from the centre; inwards, upwards, and downwards to about 60°. Projected on a flat surface it forms an oval. The centre ('fixation-point') corresponds to the yellow spot, and the 'blind spot' corresponding to the optic disc is about 15° outwards from this point. In order to measure the field roughly, the patient, placed with his back to the light, and covering one eye, looks steadily from a distance of eighteen inches at the nose or eye of the observer, who then moves his hand, or a small object, such as a square inch of paper, about in the different parts of the field, and notes any places where the object is invisible or badly seen. This test, carefully applied, will detect any considerable loss of the field. Or the patient may gaze at a spot on a black board about one foot off, and a piece of white chalk be moved from various places at the periphery until it comes into view; a line joining these points will form the boundary of the field. For accurate measurements a special instrument, the Perimeter, is necessary.

D. Disorders of Perception of Form.—SYNON.: Acuteness of Vision; *Visus*; *V.*; Fr. *Acuité Visuelle*; Ger. *Sehschärfe*; S.—Perception of form is normal only when the image of the object looked at falls on the retinal layer of rods and cones, at the centre of the yellow spot, is clearly defined, sufficiently bright, and of a certain minimum size.

PRINCIPLES.—The size of the image depends (1) upon the size of the 'visual angle' enclosed by the two lines drawn from the extremities of the object to the 'nodal point' just behind the crystalline lens; and (2) on the distance of the nodal point from the retina, which in the normal eye is 15 mm. The form of any letter or character is distinguished by a properly formed and healthy

eye, in moderate light, if it subtend a visual angle of five minutes, each of its separately distinguishable parts subtending an angle of one minute. If the nodal point be more than 15 mm. from the retina, the image will be larger, and the acuteness of vision therefore increased; this occurs in myopia, and also when a convex glass is held in front of the eye. The reverse is true if the distance be less than 15 mm., as in hypermetropia and when a concave glass is held before the eye. Hence convex lenses always increase, and concave lenses always diminish, the size of the retinal images. Vision or 'fixation' is called *direct* or *central* when the image of the object looked at falls on the yellow spot; *indirect* or *excentric* when, in consequence of impairment of function at the yellow spot, an image falling on some other part is better seen. The sharpness of the image depends (opacities of the media apart) upon the retina being exactly at the focus of the refracting (dioptric) media of the eye; it is also influenced somewhat by the size of the pupil, being, *ceteris paribus*, better when the pupil is small.

Normal acuteness of vision is expressed as unity (V . or S . = 1); subnormal vision being expressed as a fraction. Various *test-types* are in use, composed of letters, figures or words, of such a size that each subtends the minimum angle of five minutes at a certain distance. The test-types of Dr. Snellen are in most general use, and include letters visible under the standard angle at from 60 metres to $\frac{1}{5}$ metre. If No. 60 be read at 60 m., then $V. = \frac{60}{60}$ or 1; if No. 60 can only be seen at 6 m., $V. = \frac{6}{60}$; &c. V ., therefore, is expressed by a fraction whose numerator is the greatest distance at which a given type can be read, and the denominator the distance at which it ought to be seen; or the fraction may be reduced ($\frac{6}{60} = \frac{1}{10}$, &c.). The acuteness probably becomes somewhat lowered in some old persons without demonstrable disease.

The principal disorders of perception of form calling for description here are as follows: (1) Functional Affections of the Optic Nervous Apparatus; (2) Abnormalities of Refraction, including Hypermetropia, Myopia, and Astigmatism; (3) Anisometropia; (4) Disorders of Accommodation; and (5) Asthenopia.

I. Functional Affections of the Optic Nervous Apparatus.—Amblyopia without ophthalmoscopic changes may be permanent or temporary, and exhibit many differences in the character of the failure of sight. Many persons see much better with one eye than the other, though the defective organ shows no sign of past or present disease. Hypermetropia, or some other form of ametropia, is often present, and is usually of higher degree in the amblyopic eye. When, as is common, squint occurs in such a person, the squinting eye is almost always the defective one; and it has been assumed that the defect is a result of the squint, and due to a purposive suppression of sight in order to avoid double vision. This kind of amblyopia is, however, seen without either squint or ametropia—a fact which, with others, renders it almost certain that the defect is usually, or chiefly, congenital. Whether the seat of deficiency is in the eye or the brain remains to be proved; but it is well to realise that this form of amblyopia can seldom be improved even by long practice. In cerebral hemianæsthesia there may be blindness, or amblyopia with contraction of the field and colour-

blindness, in the eye opposite to the lesion, with a lower degree of the same condition in the other eye. Some rare cases of defective sight in one eye without changes, in which there is a history of previous paralytic symptoms, probably belong to this group. In *hemioptia* (properly *hemianopsia*) there is usually loss of the corresponding (R. or L.) halves of the visual fields, vision being lost on the side opposite to the lesion. The lesion may be in the tract, but is much more often higher up (optic thalamus or occipital lobe). Sometimes there is contraction of the remaining half field. Loss of both temporal halves indicates disease at the chiasma; neither this, nor loss of both nasal halves, is frequent. In hemioptia, even of long standing, from disease above the chiasma, the optic discs are seldom altered. In some cases of 'hemioptia' only a quarter of each field is lost. Hemioptia, usually temporary, for colours only, without loss of light- or form-sense also occurs.

Disease of the optic nerve between the eye and the chiasma causes blindness or defective sight, often at first with little or no ophthalmoscopic change; but if the loss of sight remain, ophthalmoscopic signs either of inflammation or atrophy appear in a few weeks. These changes may, however, be very slight; as in the common cases of central amblyopia caused by tobacco-smoking, in which disease of the optic nerves has been demonstrated, and in some cases of amblyopia due to disseminated sclerosis.

Temporary fogginess of sight, usually with the appearance of coloured rings around a candle, occurs in the premonitory stage of glaucoma; these last from half an hour to a day or more; they do not usually occur in both eyes at once (*see EYE, AND ITS APPENDAGES, Diseases of*). Attacks of megrim are often ushered in by a peculiar transient subjective defect of sight; a small cloud, appearing near the middle of the field, quickly spreads with a quivering movement and zigzag outline over about half the field; its borders are often brilliantly coloured; it affects *both* eyes; is equally visible whether the eyes are open or shut; lasts about a quarter of an hour; and is generally followed by the other megrim-symptoms to which the patient is subject. But some persons merely complain of a 'cloudiness' or of 'spots' before their headaches (*see MEGRIM*). Brief attacks of defect or blindness of *one* eye, coming on suddenly, and recurring in the same eye, occasionally take place; and in some of these the eye eventually remains blind with the changes characteristic of obstruction of the retinal artery.

Persons who suffer from severe neuralgic pain in the fifth nerve sometimes describe dimness of the same eye during an attack, but the opportunity of verifying the statement seldom occurs. In hysterical persons sight is sometimes much lowered, and may seem altogether lost in one or both eyes. There may be photophobia, and symptoms of accommodation-spasm, and the field is, or seems to be, highly contracted. Though it may be exceedingly difficult to say that there is conscious dissimulation, the groundless nature of the ocular symptoms is sometimes proved by the fact that acuteness of vision, even in the 'blind' eye, is at once and perfectly restored by the weakest possible lens, or by a piece of flat glass mounted to resemble a trial-lens. Intentionally feigned blindness of one eye can nearly always be detected by one device or another;

but pretended defect of both eyes is more difficult to expose.

2. Abnormalities of Refraction.—SYNON.: *Ametropia*.—These conditions are of importance by preventing the formation of clear retinal images; in addition they often make the sustained use of the eyes difficult or impossible (*asthenopia*). They include (a) *hypermetropia*, (b) *myopia*, and (c) *astigmatism*. The varieties of asthenopia will receive a short separate account at the end of this article. As ametropic conditions are remedied by optical aids, it will be convenient first to refer to the subject of spectacles.

Spectacles and Eye-glasses.—Refracting spectacles are made either of crown-glass or of rock-crystal. The latter is more expensive, but harder, less breakable, and rather lighter. Ordinary spectacles are biconvex or biconcave spherical lenses. Meniscus lenses are sometimes used, and are called 'periscopic,' because they give a larger field. In 'Franklin' or bifocal spectacles the upper part is of a different focal length from the lower; they are used by persons who need distance- and reading-glasses of different strengths in the same frame. The various non-refracting protective glasses (goggles, domed glasses, horseshoe or D protectors, &c.) are generally included under the term 'spectacles.' The most important points in the mounting of spectacles are that the hinges should be strong, the sides long enough to hold securely without uncomfortable pressure, and that the bridge should fit the nose well. The optical centres of the lenses should, unless otherwise ordered, be opposite the centres of the pupils when the glasses are in use. All concave glasses and convex distance-glasses should sit as close to the eyes as possible; convex reading-glasses may be further from the eyes, and so shaped that the eyes look over them when raised to gaze at a distant object.

For 'simple' astigmatism the correcting lens is a segment of a cylinder; for 'compound' and 'mixed' cases the effect of a cylindrical and spherical lens is required, and may be obtained either by combining two suitable cylindrical curvatures at right angles to each other, or by grinding the cylinder on the flat side of a plano-convex or concave lens; they require, of course, to be mounted with the curvature of the cylinder exactly in the right direction. When prisms are ordered they are mounted like ordinary spectacles, and a lens may be ground upon each surface of the prism if necessary; it is not practicable to wear prisms of more than about 8°. Spherical lenses can be made to act to a varying degree as prisms, by putting them with their centres nearer to or farther from each other than the pupils.

Numbering.—Spectacle-lenses are at present numbered on two different systems, namely—(1) the *inch* scale; and (2) the *metrical* scale. (1) In the old system the refractive unit is a lens of 1-inch focal length, and the inch may be English, Parisian, or other. The lenses in use being all weaker than the unit are expressed by fractions; thus the strongest in use in the trial-case being a 2-inch lens is expressed as $\frac{1}{2}$ (+ or −, according as it is convex or concave); a lens of 10 inches focus is $\frac{1}{10}$; and so on. It is desirable that the series of lenses should rise by equal refraction-intervals, and here the inch-scale is inconvenient because it introduces difficult fractions. (2) The inch-scale has largely given place to the metrical dioptric scale, in which the measure

is international, the refractive unit is a weak instead of a strong lens, and the refractive intervals are equal. The unit is a lens of 1 metre (100 cm.) focal length, and is called one *dioptre* (1 D.). Stronger lenses are written as whole numbers; thus a lens four times as strong as the unit is 4 D.; a lens equal to half the unit is .5 D. The disadvantage of the system is that the numbers do not, as on the inch-system, express the focal length of the glasses; but the latter is easily arrived at by dividing 100 by the number of the lens in dioptries; thus the focal length of 5 D. = $\frac{100}{5}$ = 20 cm.

To convert a lens made by the Paris inch into its equivalent in dioptries, multiply its inch-value by 36 (1 m. = 36 Paris inches nearly); thus, $\frac{1}{36} \times 36$ = 1 D. To convert a metrical lens into its equivalent in Paris inches, divide its value in D. by 36; thus 4 D. = $\frac{4}{36}$ = $\frac{1}{9}$.

The following are the most important equivalent numbers:—

Focal length in		Focal length in	
Dioptries (D.)	Paris inches	Dioptries (D.)	Paris inches
.5	72	4	9
(written $\frac{1}{72}$, &c.)		4.5	8
.75	50	5	7
1	36	6	6
1.25	30	7	$5\frac{1}{2}$
1.5	26	9	4
2	18	11	$3\frac{1}{2}$
2.5	14	13	3
3	12	15	$2\frac{1}{2}$
3.5	10	18	2
	(nearly)		

Several intermediate numbers found in the trial-cases have been omitted.

The several abnormalities of refraction may now be discussed in due order.

(a) **Hypermetropia.**—In hypermetropia the retina lies within, instead of at, the principal focus of the dioptric media. Parallel rays, such as come from very distant objects, therefore meet the retina before being focussed; and divergent rays, from near objects, meet it still more in advance of their focus. Hence the hypermetropic eye, in repose, sees nothing clearly. Distant objects can be seen clearly if, by exerting accommodation, the crystalline lens be made more convex; or if the rays, before they enter the eye, be made sufficiently convergent by passing through a suitable convex lens.

Hypermetropia, due to flatness of the posterior segment of the eyeball, *axial hypermetropia*, is always congenital; and extended observations by different authorities have shown that nearly all children are hypermetropic at birth. In sections the circular fibres of the ciliary muscle are, or appear to be, more abundant than in the normal eye. The cornea is not flatter, but the anterior chamber is rather shallower, and the pupil rather smaller than normal. In high degrees the eyeball is too small in all directions.

The hypermetropic eye cannot see clearly even at a long distance without using accommodation; and a proportionate increase of accommodation is required by the hypermetropic eye for near vision. The absolute quantity, amplitude, or range of accommodation appears as a rule not to be greater in hypermetropic than in normal eyes; hence in hypermetropia it becomes sooner insufficient for the needs; and the higher the degree of hypermetropia the earlier does this occur.

SYMPTOMS.—The symptoms depend on the patient's age, occupation, and health, and on the degree of hypermetropia. The lower degrees only exceptionally cause symptoms in childhood. The higher degrees in children, and the lower degrees in young adults, cause difficulty in reading, writing, or sewing, especially by artificial light, and towards the end of the day's or week's work—'accommodative asthenopia.' The difficulty is expressed in the forms of mistiness of sight, weariness or aching of the eyes, headache, sleepiness, watering, chronic congestion, and irritation of the palpebral conjunctiva. In the highest degrees the attempt to see clearly is often given up; such persons often partly compensate for the bad definition of the images by holding the book very close, and so increasing the size of the images; thus they may seem myopic. All the symptoms are worse when the health is low. As accommodation fails with age, a time arrives for every hypermetrope when, unless aided by glasses, no clear vision is possible at any distance; but spectacles are generally adopted before this occurs.

Concomitant convergent squint often arises in hypermetropia (*see* STRABISMUS). It is at first, and may remain, periodic, present only during strong accommodation; but often it becomes constant. In either case it may alternate, or may always affect the same eye. When constant and fixed, the sight of the squinting eye is usually found to be more or less defective, as already described. This defect is greatest in the nasal part of the visual field, which is shared with the good eye. It may be so great that only the largest objects are visible, yet the direct reflex activity of the pupil is never impaired.

When the crystalline lens is absent (*aphakia*) the eye is very hypermetropic. Distant vision is restored by means of a convex lens of 10 or 11 D. ($3\frac{3}{4}$ or $3\frac{1}{2}$ inches) held about half an inch in front of the cornea; objects at, say, 25 cm. (10 inches) are clearly seen through a lens of about 15 D. ($2\frac{1}{2}$ inches). Accommodation is abolished in the aphakic eye; but if the pupil be round and movable, its contraction aids a little in near vision, by cutting off the peripheral rays of light.

From the age of fifty-five and onwards the normal eye acquires a low degree of hypermetropia, owing to a change in the refraction of the crystalline lens.

Glaucoma is commoner in hypermetropic than in normal or myopic eyes. The habitual use of glasses by hypermetropic persons from early life may aid indirectly in preventing this disease.

DIAGNOSIS.—The diagnosis is made subjectively by testing with glasses, or objectively by the ophthalmoscope. The former is the more generally useful. Even distant objects are seen indistinctly by the hypermetropic eye with relaxed accommodation; but they are made clear if a suitable convex lens be held in front of the cornea. (1) This test is easy to apply when the ciliary muscle is temporarily paralysed by atropine, or abolished by natural senile changes. (2) But when it is active the matter is less simple; since the old-standing habit of exerting accommodation whenever clear vision, even at a distance, is needed, in many cases inseparably connects the effort to see with the action of the ciliary muscle. Such persons cannot relax their accommodation when looking through a convex lens at a distant object. The effect of the lens is therefore added to, instead of substituted for, that of the accommodation, and distant vision made worse;

no hypermetropia can be found by trial with glasses: it is entirely 'latent' (*H.L.*). (3) Between these extremes we find a large number who can partially relax their accommodation for distance in favour of a convex lens, but still use a part. They see well, or perfectly, in the distance without aid; they see equally well or better with convex lenses up to a certain strength. If now the accommodation be suspended by means of atropine we shall often find still more hypermetropia. The part that can be detected when accommodation is active is the 'manifest' (*H.m.*); the sum of the 'manifest' and the 'latent' is the 'total' (*H.*).

In testing hypermetropia, the patient being not less than three metres from the test-types, we begin with a very weak convex lens, and if vision is *not made worse*, try successively higher lenses until we reach the highest which allows the best attainable vision. This lens represents the manifest hypermetropia if accommodation be present, the total if it be absent. A stronger lens causes indistinctness by bringing the focus in front of the retina. In general the younger the patient the less is the manifest in proportion to the latent hypermetropia, even though troublesome asthenopia be present.

Hypermetropia is diagnosed by the ophthalmoscope if an erect image of the fundus is easily seen when the observer is at a distance of eighteen inches or more from the patient. The image is seen equally well when the observer comes as close as possible to the patient; and if he possess a 'refraction' ophthalmoscope, he can *in this position* measure the degree of hypermetropia by finding the strongest convex lens through which the details of the fundus still look perfectly clear. In this test the accommodation of both persons must be fully relaxed; the observer has to learn to do this, but the patient generally relaxes his ciliary muscle at once in the dark room, even though he could not do so when tried with glasses for the distant types.

Another method, the *shadow-test*, is based on the fact that when light is thrown by the ophthalmoscope into the eye from a distance of three or four feet, slight rotation of the mirror causes a shadow to move across the illuminated field; in hypermetropia the shadow moves in the same direction as the rotation of the *plane* mirror. This method is now largely relied upon, especially for young patients; and in skilled hands it is expeditious and very accurate.

The optic disc in hypermetropia, especially in children, often seems, and sometimes is, hazy, and is sometimes too red; and the retinal arteries are often too tortuous.

TREATMENT.—Treatment is necessary for hypermetropia whenever there is asthenopia, and when strabismus has arisen. Convex spectacles are ordered which, according to circumstances, neutralise a part or all of the hypermetropia; and are worn constantly, or only for near work. Periodic squint may always be cured by the constant use of fully correcting glasses; but in most cases where it has become constant, an operation is necessary (*see* STRABISMUS). In children with asthenopia it is usually best to order glasses for constant use, which correct almost the whole hypermetropia; but if the symptoms are in connection with weak health and the hypermetropia be slight, the temporary use of glasses for near work alone is enough. Young adults using glasses for the first time are often satisfied with those which neutralise only the

manifest hypermetropia, using them for all near work ; but after some weeks or months asthenopic symptoms often recur, we find that there is more manifest hypermetropia than before, and we are obliged to order stronger glasses. But either ophthalmoscopic estimation, or the shadow-test, will generally reveal almost the total even at the first examination ; and when these methods make it clear that the total is much greater than the manifest hypermetropia, glasses of nearly the full strength should be ordered at once. On theoretical grounds it is undoubtedly best for glasses to be worn constantly by hypermetropes, so that the accommodation may never be strained. But a good deal of latitude must be allowed to grown-up, and especially elderly, patients in regard to wearing glasses for distance, unless there be constant asthenopia.

Acuteness of sight is usually normal in corrected hypermetropia. In many cases of high degree, where vision is, both with and without glasses, subnormal, some astigmatism is also present ; but cases occur where the defect cannot be thus accounted for, and it is then assumed to be due to defective development of eye or visual centre. But probably want of early education of the retina in the perception of clear images in a great degree accounts for the phenomenon.

(b) *Myopia*.—In myopia the retina lies beyond the principal focus of the dioptric media, generally on account of lengthening of the posterior part of the eye—*axial myopia*. It is consequently at the conjugate focus of a point at some definite distance in front of the eye, which indeed is the 'far-point,' or greatest distance of distinct vision of the eye in question. The greater the elongation of the eye, the nearer is the 'far-point,' the 'shorter' the sight, or the higher the degree of myopia. By using accommodation objects can be seen at a still shorter distance.

Ætiology.—Myopia is comparatively seldom present at birth. The elongation usually comes on between about seven and fifteen years of age, progresses for a time, and stops between puberty and adult age ; but in some cases the stretching (and its attendant structural change) persistently increases through life, and leads to serious and even disastrous consequences. Myopia is often hereditary, and inheritance doubtless accounts entirely for some very severe cases where no other causes exist. But habitual use of the eyes upon close work, especially in a stooping posture and with bad light, aids strongly in its production. Any severe or lasting ill-health may determine the onset or increase of myopia in a predisposed person.

Myopia may also be caused by increased curvature of the cornea after keratitis, and is an invariable result of 'conical cornea.' Certain changes in the lens in the stages of senile cataract often produce myopia, even of considerable degree ; but this form of myopia does not, as axial myopia may, indicate degeneracy of the deeper tissues, and hence does not influence the question of operation.

ANATOMICAL CHARACTERS.—The elongation occurs chiefly in the posterior part of the eye, and especially at the yellow-spot region. The sclerotic and choroid are thinned in proportion to the distension, and the choroid often locally atrophied ; the term 'posterior staphyloma' is given to the bulging region. In high degrees the eye is enlarged, and its coats are thinned, in all directions. The term 'sclerotico-choroiditis posterior' is also used to

indicate the supposed nature of the change. In high degrees, particularly late in life, the vitreous often becomes fluid and contains opacities ; and hæmorrhagic and other effusions may occur in the choroid ; and there is a strong liability to detachment of the retina and incomplete cataract. In the ciliary muscle of myopic eyes the circular fibres are deficient or wanting. The anterior chamber is often deeper, and the pupil larger than usual. Owing to its large size the highly myopic eye is often prominent and its mobility somewhat curtailed ; and the latter condition is apt to cause fatigue of the eye-muscles.

SYMPTOMS.—A low degree of stationary myopia usually causes no inconvenience. In the higher degrees advice is sought, either because distant sight is bad or near work has to be held inconveniently close ; or on account of eye-ache, headache, watering, photophobia, or dimness ; or for inefficiency of the internal recti (muscular asthenopia), or actual divergent squint. In the highest degrees divergent squint is nearly always present at the natural distance of distinct vision, and sometimes even for distance ; and in much lower degrees there is often difficulty in keeping up convergence, and consequent pain and weariness. Aching frequently shows that the myopia is increasing ; it is made worse by use of the eyes, but is often present even when at rest in bed ; it may accompany the development of a squint, or of detachment of the retina. Myopic eyes, even of low degree, are often intolerant of bright light. Acuteness of vision is often sub-normal in the high degrees of myopia, especially in old people ; such defect, when not accounted for by visible structural changes, is assigned to congestion of the choroid.

DIAGNOSIS.—A myopic person with healthy eyes can read the smallest print fluently at his own 'far-point,' but not farther. He gains perfect distant vision by looking through a concave lens, which gives to rays of light from distant objects a divergent direction, as if they came from his natural 'far-point.' Placing him not less than three metres from the test-types we find experimentally the *weakest* concave lens that gives the best attainable vision. A stronger lens over-corrects the myopia, producing hypermetropia, which, in its turn, is corrected by the exercise of accommodation.

Myopia is diagnosed objectively as follows : (1) By the shadow-test with the *plane* mirror, the shadow moves in the opposite direction to the rotation of the mirror. (2) By direct examination close to the patient, a clear image (erect) can be obtained only by placing a concave lens behind the mirror ; the *weakest* lens which gives a clear image being the measure of the myopia. (3) By direct ophthalmoscopic examination at a long distance, an image of the fundus is seen, which, on the observer moving his head from side to side, seems to move in the opposite direction. This image disappears when the observer comes near to the eye examined. (4) By indirect examination the size of the ophthalmoscopic image increases on withdrawing the objective lens from the patient's eye.

The ophthalmoscopic changes depend chiefly on the atrophy of the choroid, which often occurs on some part of the staphylomatous area ; and the commonest form is the 'myopic crescent,' a patch of yellowish-white exposed sclerotic, due to atrophy of the choroid at the outer border of the optic disc.

But a similar 'crescent' is sometimes seen in eyes not myopic. When more advanced it extends all round the disc (annular staphyloma). There may also be myopia with signs of past or present inflammation and hæmorrhage, at the yellow spot. In high myopia with abrupt bulging of the tunics, the disc is often tilted and then looks oval, and its outer side often becomes pale.

COURSE AND PROGNOSIS.—Axial myopia cannot diminish. Though its increase as a rule ceases about the same time as the cessation of the bodily growth, it may continue, or may take a fresh start later in life, especially if the health be bad or the eyes be excessively used for fine work. But often its course seems to depend upon causes which are not under direct control; for we see myopia of high degree, leading to disastrous results or blindness, in persons who have never learnt their alphabet or strained their eyes in any way; and, on the other hand, it is common to meet with very myopic people, of studious habits and advanced age, in whom the eyes have not changed since youth. In general the prognosis is worse the higher the degree, the older the patient, and the feebler the health.

TREATMENT.—Much may doubtless be gradually done to prevent the acquisition and transmission of myopia, by improvements in the lighting of school-rooms, and construction of seats and desks, and by the choice of well-printed books. During the progress of myopia the time given to school work should, whenever possible, be shortened; and if the disorder be quickly increasing, or if there be much aching or irritation, rest of the eye should be insisted upon for several months, or longer. Myopic children should use their eyes only as much as is comfortable, and should be forbidden to read fine print, to read by bad light, or to stoop. If there be severe aching and intolerance of light, or rapid increase of the myopia, especially with diminished acuteness of sight, prolonged rest, plane smoked glasses, and the use of blisters or leeches, at intervals of a few days, with derivative treatment, are of service.

The corrective treatment consists in the use of concave glasses. Myopic children should as a rule wear glasses for distance on educational grounds. Adults may use their own judgment as to wearing distance-glasses; these glasses should be .5 D. less than the full correction in each case. If the eyes be strong the same glasses can often be used for near work, too; but if the internal recti or accommodation be weak, the full correction often cannot be continuously worn. Except in low degrees of myopia it is therefore best not to begin with fully correcting glasses for near work, for they require full action of the hitherto little exercised accommodation, and also derange the relation between accommodation and convergence, and are thus likely to tire the eyes; they also cause difficulty by diminishing the retinal images; if their use be persisted in for reading or other purposes, when they cause discomfort, they may help indirectly to increase the myopia. When the natural far-point in myopia is not nearer than 13 inches (33 cm.) (= 3 D.) reading-glasses are seldom required; but for higher degrees it is often necessary to order spectacles for reading which partly correct the myopia, that is, make the eyes less myopic, and thus remove the far-point farther off, and allow the patient to read, or use them in other ways, without

stooping. As a general rule, subject to the peculiarities and needs of each case, about half the full correction may for this purpose be safely and comfortably used. For music or painting a strength intermediate between those for distance and reading may be needed. When there is muscular asthenopia, shown by the fact that in near vision one eye, if covered, deviates outwards, relief may sometimes be given by combining with the reading glasses prisms with their bases inwards; the prisms, by allowing the convergence to be lessened, relieve the internal recti.

The curative treatment of high myopia consists in the removal of the lens by discission or absorption; the operated eye becoming emmetropic, or slightly myopic or hypermetropic, according to the degree of the original myopia. This operation gives very good results, and is much appreciated by the patients because they are able to dispense with the strong concave glasses they had been using. The durability of such eyes has, however, not yet been sufficiently tested to justify a final opinion on its place in ophthalmic surgery.

(c) **Astigmatism.**—Astigmatism may be either *regular* or *irregular*. *Regular* astigmatism depends upon the refracting surfaces of the eye, chiefly of the cornea, not being spherical, but having different curvatures, that is, focal lengths, in different meridians, the meridians of greatest and least curvatures ('chief' or 'principal' meridians) being always at right angles to each other, and the others have regularly intermediate curvatures. The meridian of greatest curvature of the cornea is generally vertical or nearly so. The astigmatism of the lens, though less regular than that of the cornea, tends to correct the latter. In 'simple' astigmatism one chief meridian is normal, the other either myopic or hypermetropic; when 'compound' both chief meridians are myopic, or both hypermetropic, but in different degrees; when 'mixed,' the eye is hypermetropic in one chief meridian, and myopic in the other. When the focal difference between the chief meridians, i.e. the degree of astigmatism, is not greater than is represented by a lens of 72 inches focus (.5 D.) it may generally be neglected; even much higher degrees often cause no trouble.

Astigmatism is to be suspected in all cases of ametropia where spherical lenses do not raise vision to the normal, no other cause of the defect being found. It is detected subjectively by numerous tests, most of which consist essentially of straight lines running in various directions, some of the lines being seen by the astigmatic eye better than others. It can also be measured by the shadow-test, the ophthalmometer, and direct ophthalmoscopic examination.

TREATMENT.—Astigmatism is corrected by cylindrical lenses, which neutralise the difference of refraction of the two chief meridians; but in the higher degrees acuteness of vision often remains even then subnormal. *Irregular* astigmatism can seldom be remedied.

3. **Anisometropia.**—This signifies different refraction in the two eyes, and is a very common condition, the difference sometimes being extreme. When one eye is normal and the other myopic, each may be, and often is, used for vision at different distances, and each remains perfect; but if one be astigmatic, or very hypermetropic, it is generally defective.

When slight, this condition may be neutralised by corresponding spectacles, but when the inequality is great, fully correcting glasses cause so much difference in the size of the images in the two eyes that equalisation is seldom possible. But it should be attempted when there is any tendency to squint, in order to encourage binocular vision.

Effect of blindness of one eye.—Acuteness of sight is always rather better with both eyes than with either alone; further, both eyes are necessary for the appreciation of solidity and distance. Patients often think that blindness or disuse of one eye throws 'double work' upon the other and 'weakens it.' Nearly always, however, in such a case some other cause can be found for the asthenopia of the sound eye.

4. Disorders of Accommodation.—(a) **Presbyopia.**—The 'amplitude' or 'range' of accommodation is expressed by the difference between the greatest distance, 'far-point,' (r), and the least distance, 'near-point,' (p), of distinct vision. Age for age, it is nearly equal in all eyes, whatever their refraction. Its natural failure with age causes presbyopia, the onset of which has been arbitrarily fixed to begin, in the emmetropic eye, at the age of about 40 to 45, when the near-point is at nine inches (22 cm.); and the failure generally progresses at a constant rate. Presbyopia is corrected by the convex lens, which enables the patient to read at nine inches; the strength of this lens varies inversely as the amplitude of accommodation; and at the age of 65, the near-point being removed to infinity, the correcting lens is one of nine inches focus.

SYMPTOMS.—Presbyopia is first shown by difficulty in reading or sewing by artificial light, or in the train or carriage; defective accommodation prevents the work being held close enough to compensate for the defective light or for the shaking, and to remedy the former the candle is often placed between the eyes and the book. When more advanced, the patient becomes 'long-sighted,' and has to put his book at arm's length unless he wear glasses. If the refraction is normal, distant sight is perfect. In hypermetropia, presbyopia begins at an earlier age, less accommodation being available for near vision; and in myopia it sets in later because less accommodation is needed for seeing at a given distance. Hence a low degree of myopia is an advantage. When the far-point in myopia is at or within nine inches (22 cm.) presbyopia does not occur.

DIAGNOSIS.—Presbyopia is to be distinguished from loss of accommodation due to paralysis of the ciliary nerves, and from failure due to feeble health or other causes, both of which may occur at any age. True presbyopia, however, sometimes occurs earlier and progresses more quickly than usual, especially in eyes which are threatened with glaucoma.

TREATMENT.—The treatment of presbyopia consists in ordering convex glasses which enable the patient to read at nine inches or a greater distance. Most people prefer glasses which enable them to read easily at twelve or fifteen inches, and with which reading at the standard nine inches is possible only for a very short time, if at all. The smaller the quantity of accommodation remaining, the less is the range of clear vision; and if accommodation is abolished, clear sight is possible only when the object is at the focus of the glasses.

Hence the increase of strength of the glasses which becomes necessary as age advances should be made gradually, that the patient may grow accustomed to the loss of range, and to the necessity for keeping his book more and more at an unvarying distance. As the book has to be placed nearer by artificial light than by daylight, it is generally best to have a rather stronger pair of glasses for evening use than for the daytime. The following table, giving the strength of the glass necessary to bring the near-point to nine inches (22 cm.), at various ages, serves as a useful basis for the selection of spectacles for presbyopia in emmetropic persons:—

Glass required to bring ' p ' to 9 in. (22 cm.)			
Age			
40	0	=	
45	$+\frac{1}{3\frac{1}{2}}$ inch	=	+1 dioptr.
50	$\frac{1}{4}$ "	=	2 "
55	$\frac{1}{3}$ "	=	3 "
60	$\frac{1}{2}$ "	=	4 "
65	$\frac{2}{3}$ "	=	4.5 "
70	1-6 $\frac{1}{2}$ "	=	5.5 "

(b) **Paralysis of accommodation.**—**SYNON.**:

Cycloplegia.—Paralysis of the ciliary muscle occurs in paralysis of the whole third nerve. But it may occur without affection of the extrinsic muscles of the eyeball. In these cases it is generally combined with paralysis, more or less complete, of the iris (*ophthalmoplegia interna*); but it may be present as an isolated symptom, the pupils being normal, and of this the commonest example is post-diphtheritic cycloplegia. The failure of accommodation in glaucoma may be accounted for in acute cases by compression of the ciliary nerves, but in old cases is doubtless due to the atrophy of the ciliary muscle which always exists. Cycloplegia, usually with some affection of the iris, is a common result of blows on the eye; sometimes recoverable, it is, however, often permanent. Lowered endurance of sight, pain, and sudden temporary failures of accommodation, are among the most important phenomena of sympathetic irritation.

(c) **Spasm of accommodation.**—Temporary spasmodic action of the ciliary muscle, often exceeding the necessary amount, frequently occurs in hypermetropia, with each effort to see clearly; it usually ceases at once on going into a dark room. In low myopia with irritative symptoms, the ciliary muscle often acts unnecessarily; and such spasm, when persistent, is probably one cause of further elongation of the eye. Spasm of accommodation also occurs in some functional and hysterical affections of the eyes, with other symptoms of ocular irritation. The function of accommodation is closely associated with that of convergence, although the two can be exerted separately to a limited extent. The accommodation of one eye cannot be exercised without, and scarcely in any different degree from, that of the other; and it is doubtful whether, as has been supposed, different parts of the ciliary muscle can act in different degrees and thus correct astigmatism.

(d) **Micropsia.**—**DEFINITION.**—Any condition of sight in which objects seem lessened in size, without diminution in the size of the retinal images. This indicates either an extreme effort of accommodation, and may be thus complained of when this function is weakened; or disease of the deep layers of the retina.

5. Asthenopia.—Asthenopia is any condition in which the eyes cannot be used for long without fatigue, pain, or other symptoms.

Muscular asthenopia is caused by difficulty in maintaining the convergence of the visual lines, and is commonest in myopia, though it is often seen with normal refraction, especially in youths and young adults. It causes, besides aching of the eyes, 'confusion' of the print, and sometimes double vision. In slight cases, accompanying myopia, partially neutralising concave glasses, which enable the book to be held at a greater distance, will often give relief. In a few cases, with or without myopia, spectacles, consisting of prisms with their bases inwards, are of service, by lessening the convergence necessary for vision at a given distance. In high degrees, tenotomy of the external rectus is called for (see STRABISMUS). Asthenopia may also be caused by slight vertical deviations (upward or downward) of one eye.

Asthenopia from deficient accommodation is also shown by inability to read for long, but there is no 'moving' or 'dancing' of the letters, nor any diplopia. The object simply becomes 'misty' or 'the sight goes' for a time, returning when the eyes are rested for a few minutes; or the eyes feel tired and hot, and ache. Headache and other discomforts may follow neglect of such symptoms. It is commonest in hypermetropia (asthenopia from excessive demand on accommodation); but is also seen in emmetropic, and even in slightly myopic eyes, if the tone of the ciliary muscle is low (asthenopia from weakness of accommodation). Asthenopic symptoms are not common in presbyopia.

Neurasthenic Asthenopia.—All causes of asthenopia are more effective in neurasthenic persons, and some of the worst cases are in those who have good range of accommodation, and no ametropia. Sensitiveness of lids, eyeball, and perhaps retina, may have been left by previous stytes, phlyctenulæ, or follicular granulations, and the onset of symptoms often dates from some piece of eye-work requiring strenuous effort; but serious breakdown of the eyes seldom occurs unless the general conditions of neurasthenia—excitability without staying power—are present.

Retinal Asthenopia.—Functional exhaustion of the retina or optic nerve is sometimes seen in optic neuritis, and other diseases of the optic nerve; sight being good, but becoming duller after a short period of use. It is not, on the whole, an important diagnostic symptom. E. NETTLESHIP.

VIS MEDICATRIX NATURÆ (Lat.), the healing power of Nature.—An expression formerly much used to indicate the tendency of wounds and diseases toward spontaneous recovery.

VITAL STATISTICS.—For the purposes of public health, vital statistics are concerned mainly with the incidence of disease and death upon communities and upon different fractions of communities, and with the renewing of population by births. Such statistics may be regarded from several points of view. It may be desired to investigate the average incidence of a certain disease, or a group of diseases, upon different populations or different sections of a population; sections marked out by race, locality, occupation, class, age, sex, or other selected characteristic. Or, again, one disease may be compared with another as to incidence upon the same population. From yet another point of view inquiry may be made into the changing incidence of a given disease upon a given population in the

course of time, the change being perhaps progressive, perhaps fluctuating at long or short intervals, regular or irregular. Thus comparison may be made between the various seasons of the year, between one year and another, or among the averages of terms of years; and the change may prove to be one of quantity, or quality, or both. By patient record and analysis of the available data, it becomes possible to learn *how*, *when*, *where*, and *whom* a given disease attacks, and hence to obtain a clue to the further problem *why* it attacks, and even to the most important matter of all, the means of prevention.

The materials necessary for such research include, on the one hand, records of births, deaths, and sickness; and, on the other hand, accurate information as to the population among which the events so recorded have happened.

Population.—A census in this country is taken at the end of the first quarter of the first year in each decade. Between these widely distant known points, estimates can be made on the assumption that the rate of increase is uniform. Thus, if P and P' be the numbers enumerated in 1881 and 1891 respectively, the rate of increase in ten years is $\frac{P'}{P}$, in one year therefore $\sqrt[10]{\frac{P'}{P}}$, in a quarter of a year $\sqrt[40]{\frac{P'}{P}}$. For the sake of brevity we may denote

the rate of annual increase, that is $\sqrt[10]{\frac{P'}{P}}$, by r , and the quarterly rate by $\sqrt[4]{r}$. Then the estimate at the end of the first quarter of 1882, one year after the census, will be $P \times r$, in two years $P \times r \times r (= Pr^2)$, in three years Pr^3 , and so on, year by year, till we come to $P \times r^{10}$, which is of course equal to P' . But the mid-year population, which is always adopted as the basis of birth-rates and death-rates, will be in advance of these estimates by one quarter's growth: $P \times \sqrt[4]{r}$ in 1881, $P \times r \times \sqrt[4]{r}$ in 1882, $Pr^2 \times \sqrt[4]{r}$ in 1883.

Further than this, it is assumed that the population P' of 1891 goes on increasing at exactly the same rate as the population P of 1881 was found to have done; the estimates for the years after 1891 are made upon the old data, until the next census again gives certainty. The estimate for the mid-year 1891 is $P' \times \sqrt[4]{r}$, for 1892 $P' \times r \times \sqrt[4]{r}$, for 1893 $P'r^2 \times \sqrt[4]{r}$, for 1900 $P'r^8 \times \sqrt[4]{r}$.

In practice, the values of P and P' are known from the published census returns, and the use of logarithms makes the calculations perfectly simple. A rougher method, sufficiently accurate for small populations, is to assume that the increment in every year is one-tenth of that recorded in the ten years, and to add $\frac{P' - P}{10}$ for each year. It is obvious that

if the rate of increase as explained above remains constant the result will only be accurate about the middle of the decade, the increment being overstated during the first half, and understated during the second; for, as the population is increasing, $P' - P$ bears a higher ratio to P than it does to P' , so that the increment in 1881 is made to be *relatively* larger than that in 1891.

Sometimes the estimate is based upon other data altogether, by adding to the last census-population the so-called 'natural increase,' that is, the excess of births over deaths, it being assumed that there is no gain or loss from other sources, or that these

balance each other. Experience has shown that while mathematical estimates are tolerably correct for very large mixed populations, such as a whole nation, they are liable to error in proportion to the smallness of the numbers concerned. In towns the growth is chiefly marginal, the central portion being capable of little increase; and the growth, even if continued uniformly, may be outside the more or less arbitrary boundaries of the area under consideration. Fluctuations of trade are of great moment as affecting local immigration and emigration. In many instances the 1891 estimates, based upon the 1871 and 1881 census, were proved by the census of 1891 to be far wide of the mark. Liverpool, instead of increasing as had been supposed, was found to have actually decreased, and the population was over-estimated by 20 per cent. Salford was over-estimated by 27 per cent., and Newcastle under-estimated by 11 per cent. For at least five years out of every decade local birth-rates and death-rates are liable to serious error, although their untrustworthiness will usually be made known either by persistent abnormality of the rates themselves, or by the discrepant results of local observations, such as influx or efflux of artisan population, records of school attendance, inhabited houses, &c.

For many statistical purposes, much further detail is required. It is necessary to know how many there are of each sex, of each group of ages, or of each occupation, for the tendency to sickness and death varies greatly, and rates may need to be calculated separately for each class. The data are supplied by the census, and the relative proportions may be assumed to remain unaltered during the intercensal decade. For the infant population, however, the recorded number of births affords a closer estimate.

Births and Deaths.—Births must be registered within six weeks, and deaths within five days, of their occurrence. These form the basis of the national statistics of births and deaths. As regards the latter, the record includes not only the time and place of death, but also the age, sex, and place of residence of the deceased, the causes (primary and secondary) of death, and their duration. The age to be given is that attained at the last birthday; if less than one year it should be stated in months, weeks, days, or hours. In compiling statistics of deaths from different causes only one cause can be accepted for each death, and when two or more are recorded it is usual to give precedence to 'zymotic' diseases over all others, to 'specific' over non-specific, and to 'primary' over 'secondary' causes. Where these criteria are indecisive, priority may be given to the cause mentioned first in the certificate. 'Uncertified' deaths, in which no certificate is given either by a registered medical practitioner or by a coroner, were in 1898 1.9 per cent. of the total number. A large proportion of deaths (in towns not often less than 10 per cent., and in London as many as 25 per cent.) take place in public institutions, such as hospitals, asylums, and workhouses. These ought to be credited to the locality of former residence, which may or may not be the same as that in which the institution is situated, and in which, therefore, the deaths are registered.

Still-births are not registered in this country; they are estimated to be equal to about 4 per cent. of the live births. Of the latter rather more than

4 per cent. are illegitimate, the proportion being greater in rural than in urban communities, and declining in England generally. Over 103 males are born for every 100 females, but owing to the higher death-rate among male infants the female population exceeds the male in the second year of life and at all later ages.

Rates.—Statistics of births and deaths have to be considered with reference, first, to the magnitude of the population concerned, and, secondly, to the length of time covered by the record. It is convenient to adopt fixed standards of population and time, and birth-rates and death-rates are usually stated 'per thousand per annum,' that is, as the number of such events among each thousand of the population in the course of a year. The true (astronomical) year has 365.24 days, while the statistics of births and deaths will usually be those of 364 days (52 weeks) or 365 days. Hence an adjustment becomes necessary if great accuracy be desired. Thus, if the record of a 'year' of 52 weeks give a death-rate of 20.0 per 1,000 per annum, the true death-rate will be $20.0 \times \frac{365.24}{364}$, or

20.07. Similarly, birth-rates and death-rates for fractions of a year are stated as *annual* rates; they are the annual rates which would be attained if the conditions observed during the period in question were continued unchanged for a whole year. In general terms, if n be the number of days during which events are recorded, the rate per 1,000 per annum will be

$$\frac{\text{No. of events recorded during } n}{\text{Population at middle of year}} \times \frac{365.24}{n} \times 1,000.$$

Thus, if there be 40 deaths in a given week in a population of 100,000, the death-rate for that week is $\frac{40}{100,000} \times \frac{365.24}{7} \times 1,000$, that is, 20.9 per thousand per annum.

The population must of course be that in which the events happen, neither more nor less. To find the death-rate at ages between 10 and 15 years, for instance, we must take as our basis, not the whole population, but merely the number of persons living at those ages; the deaths at ages 10 to 15 years must be compared with the population at ages 10 to 15 years.

Birth-rate.—The English birth-rate has declined steadily from 36.3 in 1876 to 29.4 in 1898. It is highest in districts containing the largest proportion of young married people, and high, therefore, in towns which are rapidly growing by influx of young adult persons. In prosperous mining districts, local birth-rates of 40 or 50 are not uncommon. Although birth-rates are always calculated on the whole population regardless of age or sex, it ought to be remembered that the population more immediately concerned is that of married women under 45 years of age, and that a community containing an unusually large or unusually small proportion of such persons may be expected to have a correspondingly abnormal birth-rate.

Death-rate.—Death-rates may be calculated for a community as a whole, or for a particular class or section of a community; and with regard to deaths from all causes, or deaths from one cause or group of causes only.

The English death-rate has materially declined

during the last twenty years, and, on the whole, more rapidly among females than among males.

Mean Annual Death-rates in

Years	Both Sexes	Males	Females
1841-50	22.4	23.1	21.6
1851-60	22.2	23.1	21.4
1861-70	22.4	23.6	21.3
1871-80	21.3	22.6	20.0
1881-90	19.1	20.2	18.0
1891-95	18.7	19.8	17.7
1896	17.1	18.1	16.1
1897	17.4	18.6	16.3
1898	17.6	18.7	16.6

The decline has not been free from interruptions, and, indeed, after reaching a minimum in 1894 there has since been an increase as the table shows.

In infancy and in old age the tendency to death is high, the minimum being at ages between 10 and 15 years. The following table gives the mean annual rates in England and Wales for the decades 1871-80 and 1881-90 :

Ages	Both Sexes		Males		Females	
	1871-80	1881-90	1871-80	1881-90	1871-80	1881-90
0-5	63.1	56.8	68.1	61.7	58.1	52.0
5-10	6.4	5.3	6.7	5.3	6.2	5.3
10-15	3.7	3.0	3.7	2.9	3.7	3.1
15-20	5.3	4.4	5.2	4.3	5.4	4.4
20-25	7.0	5.6	7.3	5.7	6.8	5.5
25-35	8.9	7.5	9.3	7.7	8.6	7.3
35-45	12.6	11.4	13.7	12.3	11.6	10.5
45-55	17.7	17.1	20.0	19.3	15.6	15.0
55-65	31.5	31.3	34.8	34.7	28.5	28.4
65-75	64.9	64.7	69.6	70.2	60.8	60.1
Over 75	161.6	153.7	169.1	162.2	155.8	147.3
All ages	21.4	19.1	22.7	20.2	20.1	18.0

The comparison of these two decades shows that in both the death-rate was lower among females than among males, not only at all ages taken together, but also at each group of ages separately, except in the second decade of life. It shows, too, that the general death-rate had declined in both sexes, and that the decline was shared by almost all of the age-groups, although much more marked at the earlier ages.

It is usual to state the 'infant mortality' in a different way, taking advantage of the accurate statistics of births as a basis of population, and thus avoiding the uncertainty of estimates. The infant mortality is therefore

$$\frac{\text{No. of deaths under one year}}{\text{No. of deaths during year}} \times 1,000.$$

It is not comparable with death-rates at other ages, for it is not calculated upon a mean population. It is higher in towns than in the country, and among males than among females. For the

decade 1871-80 the mean infant mortality was 149—that of males being 163 and females 134. It declined to 142 in the decade 1881-90, but has on the average exceeded 150 in the decade 1891-1900, the exact figures for which cannot yet be stated.

Notwithstanding the high mortality among infants, a high birth-rate does not usually carry with it a high general death-rate, but rather the reverse. It implies a large proportion of young adults in the population as parents, and also a large proportion of children who have passed the period of early infancy. Among these groups the death-rate is low, as is evident from the foregoing table, and as a net result the excess of infant population is more than balanced by the excess at ages of low mortality.

The age-constitution of the population ought always to be borne in mind as a possible source of fallacy in comparing crude death-rates. Extreme instances are met with in dealing with selected populations—for example, the public services. The death-rate among soldiers stationed in the United Kingdom was 6.9 during the decade 1881-90, and 4.9 in the quinquennium 1891-95. In order to form an opinion whether these rates are high or low, comparison must be made with the standard death-rate among males of military ages, and not with that of a community containing women, children, and old people. Reference has already been made to the differences in age-constitution between urban and rural communities, and between one town and another. From census-data it is possible to calculate a 'factor,' or number, by which the crude death-rate may be multiplied if it be desired to nullify the effect of a local age- and sex-distribution, differing from that of the country at large. The 'corrected death-rate' so obtained represents that which would prevail under local conditions if the local population had the same composition, as regards age and sex, as that of the kingdom as a whole. For the decade 1881-90 the crude death-rate for Lancashire was 22.4, and for Huntingdonshire 16.4; the corrected death-rates for these counties were 13.9 and 24.2, the respective 'factors' being 0.84948 and 1.07589. Rural districts usually contain an unduly large proportion of young and old persons, with comparatively few young adults, and the factor for correction is less than unity; but most towns contain an excess at those ages at which mortality ought to be low, and the factor in all such cases is greater than unity. Corrected death-rates enable us to compare the mortality of different towns upon equal terms, and the comparison is further facilitated by calling the death-rate of the whole country 1,000, and stating the 'comparative mortality figure' of each town in terms of this. The calculation is simple.

$$\text{C.M.F.} = \frac{\text{Death-rate of whole country}}{\text{Corrected local death-rate}} \times 1,000.$$

Life-Tables.—The death-toll at each age being known, it is possible to construct a life-table—that is, to estimate the gradually diminishing number who would survive at the end of each successive age-period, out of, say, a million born.

The following data are taken from life-tables constructed by Dr. Ogle (1871-80) and Dr. Tatham (1881-90). The chance of survival at each age and in both tables is greater among females than among males. The later table shows a greater number of survivors than the other at every age up to 83 in males, and 85 in females.

MALES					FEMALES				
Survivors out of a million born			Expectation of life		Survivors out of a million born		Expectation of life		
Age	1871-80	1881-90	1871-80	1881-90	1871-80	1881-90	1871-80	1881-90	
0	1,000,000	1,000,000	41'4	43'7	1,000,000	1,000,000	44'6	47'2	
10	708,990	733,477	47'6	49'0	738,382	766,151	49'8	51'1	
20	680,033	712,555	39'4	40'3	707,949	744,321	41'7	42'4	
30	630,038	669,279	32'1	32'5	658,418	700,049	34'4	34'8	
40	563,077	604,923	25'3	25'4	596,113	638,912	27'5	27'6	
50	476,980	517,639	18'9	18'8	520,901	564,299	20'7	20'6	
60	365,011	398,400	13'1	12'9	422,835	457,682	14'2	14'1	
70	222,056	238,632	8'3	8'0	277,225	299,220	9'0	8'8	
80	77,354	80,023	4'8	4'5	108,935	114,536	5'2	5'0	
90	8,015	6,786	2'7	2'4	14,225	13,418	2'9	2'8	
100	82	30	1'6	1'2	225	157	1'6	1'5	

The calculation may be carried a step farther, and an estimate made of the 'expectation of life' at each age—that is, of the average number of years which persons who have reached that age will live. It has already been stated that while the death-rate as a whole is decreasing, the decrease is mainly in the earlier age-groups. Accordingly, we find on comparing the two life-tables, that while the expectation of life is greater under the new table than under the old at all early ages, it is actually less at

general death-rate. Thus whooping-cough and measles kill few except in early childhood, while cancer kills few except in later adult life, and phthisis is most destructive in early adult life.

As regards sex, similar disproportion may be met with. The majority of diseases cause a higher mortality among males, the principal exceptions being diphtheria, whooping-cough, cancer, and, of course, the diseases connected with parturition. Cancer is nearly twice as common among females as

Mean Annual Death-Rates, 1881-90.—England and Wales.

Ages	Small-pox	Measles	Scarlet Fever	Enteric Fever	Whooping-cough	Diphtheria	Diarrhoea	Cancer	Phthisis	Heart-Disease &c.	Respiratory Diseases	Urinary Diseases
0-5	'08	3'13	1'67	'13	3'37	'69	4'35	'02	'54	'13	12'97	'19
5-10	'03	'27	'76	'18	'13	'42	'05	'01	'29	'15	'83	'09
10-15	'03	'02	'15	'21	'00	'10	'02	'01	'52	'26	'21	'06
15-20	'04	'01	'04	'29	'00	'04	'02	'02	1'55	'33	'31	'09
20-25	'06	'01	'03	'28	'00	'02	'02	'04	2'32	'36	'44	'14
25-35	'06	'01	'02	'23	'00	'02	'04	'13	2'90	'59	'78	'23
35-45	'04	'00	'01	'18	'00	'02	'06	'58	3'13	1'35	1'61	'44
45-55	'03	'00	'00	'16	'00	'01	1'55	2'74	2'57	3'17	'73	'73
55-65	'02	'00	'00	'14	'00	'02	3'0	2'87	2'17	5'71	7'10	1'36
65-75	'02	'00	'00	'11	'00	'02	'91	4'16	1'36	12'20	15'08	2'69
Over 75	'02	'00	'00	'06	'00	'01	2'58	4'30	0'52	18'86	29'09	4'17

ages over forty-five years, in each sex. The expectation at birth is also termed the 'mean duration of life,' and at later ages the 'mean after-life-time.' Owing to the high death-rate among young children, the expectation is greater at three years of age than it is at birth; thenceforward it declines uninterruptedly. The detailed 1881-90 life-table shows that out of a given number of males born, exactly half survive between 51 and 52 years of age; and this has been termed the 'probable duration of life.' For females it is between 56 and 57 years.

Causes of Death.—So far only total death-rates have been considered, without distinction of cause. If the incidence of the general death-rate upon different ages and sexes is unequal, that of single diseases or natural groups of diseases is still more so. Each disease has its characteristic curve of age-mortality; and for obvious reasons this curve is almost always more intense than that of the

among males, while in deaths from violence there is a still greater inequality in the opposite direction. These are extreme instances; but in comparing different local or specialised death-rates with each other or with a standard, it is necessary to bear in mind that the comparison is only legitimate on the assumption that there is no *material* difference with regard to age- and sex-constitution.

The following table shows the progressive changes during the last thirty years in the mortality attributed to certain causes.

The possibility of altered nomenclature of disease, as well as altered environment, must be taken into account in interpreting these changes; and as regards some of the zymotic diseases, the occurrence of epidemic variations in quantity and quality, at long or short intervals, makes it difficult to measure their average incidence against that of the more equable causes of death, or to compare the course

of the death-rate of one zymotic disease with that of another upon anything like equal terms (see PERIODICITY IN DISEASE). It is a disputed point whether the ominous increase in the mortality

Mean Annual Death-Rates.—England and Wales.

Cause of Death	1861-70	1871-80	1881-90	1891-95
Small-pox . . .	0'16	0'25	0'05	0'02
Measles . . .	0'44	0'38	0'44	0'41
Scarlet Fever . .	0'97	0'72	0'34	0'18
Typhus . . .	?	0'06	0'02	0'00
Enteric Fever . .	?	0'33	0'20	0'17
Whooping-cough .	0'53	0'51	0'45	0'40
Diphtheria . . .	0'18	0'12	0'16	0'25
Diarrhoea . . .	0'97	0'92	0'66	0'63
Cancer . . .	0'39	0'47	0'59	0'71
Phthisis . . .	2'49	2'13	1'74	1'46
Heart-Diseases, &c.	1'05	1'34	1'58	1'68
Respiratory Diseases .	3'36	3'74	3'59	3'68
Urinary Diseases .	0'27	0'35	0'43	0'45
Violence . . .	0'77	0'74	0'65	0'56
ALL CAUSES . . .	22'5	21'4	19'1	18'74

attributed to cancer is real, or only due to the better diagnosis of cases which in former years would have been assigned to other causes.

Density of Population.—Even apart from any correction for age- and sex-distribution, the death-rate is generally higher in urban than in rural districts. In both it is declining, but as the fall is more rapid in the former the difference is growing less.

England and Wales.—Urban and Rural Death-Rates, 1861-90.

Years	Persons per Square Mile	Death-rate per 1,000 living		
		England and Wales	Town Districts	Country Districts
1861-70	365	22'5	24'8	19'7
1871-80	416	21'4	23'1	19'0
1881-90	470	19'1	20'3	17'3

The average density in 1891 was 500 persons per mile, or 0'8 per acre. If below this average, the density seems to have little influence upon the death-rate; but if above it, the increasing density is in general associated with increasing death-rate. But the density per acre or per square mile is a very crude measure of the overcrowding which is hygienically most important. In a sparsely populated district there may still be overcrowding of villages, of alleys, of houses, or of single rooms. Sheffield is said to have rather less than 17 persons per acre, but this bare average goes for little, since in one registration sub-district the density is 249 per acre, and in another 0'5 only. In London the death-rate among the better class of many-storeyed 'model dwellings' was found to be lower than the average, notwithstanding a more than tenfold greater density per acre. Farr held that, *other things being equal*, the death-rate varies as the twelfth root of the density of the population; the very necessary proviso makes the formula difficult of practical application in a given case.

Each of the principal zymotic diseases causes, on the whole, higher mortality in urban than in rural districts. Observations of a narrower kind, having regard to crowding of tenements, indicate that phthisis, respiratory diseases, and infant mortality are also conspicuously increased.

Mortality as affected by Occupation.—Here we have to do with a selected population—selected sometimes (e.g. miners) on account of robust health, sometimes by unfitness for other pursuits. In all cases the age- and sex-distribution differ widely from that of a general population, and comparison has to be made after correction for this. Such a comparison upon equal terms has been made by Tatham (*Suppl. to the 55th Ann. Rep. of the Reg.-Gen.*) with regard to a large number of trades. The following may serve as examples:—

Comparative Mortality according to Occupation.

Industry	Phthisis	Respiratory Diseases	Nervous Diseases	Urinary Diseases	Liver Diseases	Heart, &c. Diseases	Comparative Mortality Figure
ALL MALES	102	224	102	44	29	112	1,000
Agriculture . . .	106	115	51	24	17	83	602
Coal-Mining . . .	97	269	67	30	17	120	925
Wool . . .	191	256	100	45	21	131	991
Plumbers . . .	217	224	131	84	22	142	1,120
Cotton . . .	202	338	114	42	25	152	1,141
Innkeepers . . .	259	287	160	90	201	193	1,642
Earthenware . . .	333	668	123	63	32	227	1,706
File-makers . . .	402	423	212	124	36	304	1,810
Dock Labourers .	325	564	114	68	26	236	1,829

The comparative mortality figure is lowest (533) among the clergy, and much higher (966) among medical men.

Sickness.—Statistics of sickness are very incomplete. Those arising out of the compulsory notification of certain infectious diseases are published annually by the Local Government Board, and in local reports. Their chief statistical value at present is the measure which they afford of the local and periodic fluctuations in diffusion and case-mortality of each disease separately. Statistics of hospitals, asylums, and poor-law practice are useful, though fragmentary; and the records of the public services and of 'Friendly Societies' have yielded valuable results. All these have reference to 'selected populations,' and are not strictly relevant to average communities. For example, it is unsafe to draw conclusions as to the comparative age-incidence of enteric fever from hospital data, for there is no assurance that the cases in hospital are a fair sample of those outside in severity or in age-distribution.

It has been estimated that in a given community there will be about two weeks of sickness per head per annum, or about two to three years of continuous illness for each death. The incidence of sickness, unlike that of births or deaths, has to be considered with regard to *degree* and *duration*, so that the data, such as they are, scarcely lend themselves to statement in the ordinary form of rates.

Statistical Tests of Sanitary Condition. It has become customary to regard certain statistical data as proof of the healthiness or unhealthiness of a locality, in the sense of prevalent impurity of air,

water, soil, or dwellings. Chief among these in popular appreciation are the general death-rate, and the zymotic death-rate. The former is, upon the whole, a fairly trustworthy index of sanitary condition, but is liable to fallacy if the figures be small, or if the population be not accurately known, or if the age- and sex-distribution be abnormal, or, lastly, if care be not taken to exclude deaths of strangers and to include deaths of residents dying outside the district. Further, purely local death-rates for limited periods of time are liable to disturbance by epidemics of a kind not dependent upon insanitary conditions.

The zymotic death-rate is of little constant significance in this respect. It is made up of the combined death-rates of the 'seven principal zymotic diseases,' of which four (small-pox, measles, whooping-cough, and scarlet fever) have little claim to rank as filth-diseases, although the others (enteric fever, typhus, diphtheria, and diarrhoea) have a much more direct significance.

An excessive infant mortality may be due to diarrhoea and other diseases attributable to filth-conditions, but may also be accounted for by maternal neglect, or by epidemics of measles or whooping-cough. With equal reason the death-rates from phthisis and from respiratory diseases deserve to be taken into account.

The *mean age at death*, obtained by adding together the ages of those who die, and dividing by the number of deaths, is often cited as evidence of longevity, but has very little significance. It depends largely upon the age-distribution of the population; if there are many young children it will be low. By far the best criterion of longevity is, of course, the expectation of life, which can only be accurately ascertained from a life-table. Approximately it may be found by Farr's formula $x = \frac{2,000}{3D} + \frac{1,000}{3B}$, x being the expectation of life, B the birth-rate, and D the death-rate.

B. A. WHITELEGGE.

VITILIGO.—See LEUCODERMIA, p. 1256.

VITILIGOIDEA (*vitulus*, a spotted calf).—A term once applied to the disease now known as *xanthoma* and *xanthelasma*. See XANTHOMA.

VITTEL, in the Vosges, France.—Earthy mineral waters. See MINERAL WATERS.

VOCAL FREMITUS.—The sensation of vibration conveyed to the hand when applied over any part of the respiratory organs during vocalisation. It is felt in health, but presents various modifications in disease. See PHYSICAL EXAMINATION.

VOCAL RESONANCE.—The sound heard on auscultation over certain parts of the respiratory organs, during vocalisation, both in health and in certain forms of disease. See PHYSICAL EXAMINATION.

VOICE, Disorders of.—SYNON.: Fr. *Troubles de la Voix*; Ger. *Störungen der Stimme*.

INTRODUCTION.—Voice is the sound produced in the larynx by air driven from the lungs through the rima glottidis, modified in accordance with acoustic laws in the upper air-passages. Vocalisation is a function needing for its perfect production a healthy condition of the respiratory muscles, of

the lungs, trachea, and larynx, of the pharyngeal, oral, and nasal cavities, and of the nerves and nervous centres on which these parts depend for their isolated or co-ordinated muscular movements and their normal sensitiveness. For the production of the simplest vocal tone the cords must be free to approximate within a line of one another, while the co-ordinated action of a large number of muscles is required, to regulate their tension and that of the walls of the air-passages, to modify the form of the latter, and to produce the current of air.

Acoustically the organ of the voice must be regarded as a combined reed and pipe; and for the production of a perfect note it is necessary that the pipe should be in perfect unison with the reed. This unison depends not only on the shape of the various cavities, but on the relative rigidity or flexibility, and the tension of their walls; every variation in the number, size, or form of vibrations of the vocal cords, effected by the intrinsic laryngeal muscles, calling for similar modifications of the shape, size, and tension of the consonating cavities.

The word 'voice,' when used alone, always implies the presence of a musical tone (periodic vibrations), but sound sufficient for every purpose of speech and articulation may be produced without a musical tone. This is the *whispering voice*, caused by the rustling of air through the half-open rima and relaxed air-passages. To this condition—absence of musical tone in the voice—the term *aphonia* is applied, and it must be distinguished from the actual inability to produce sound, which we see after tracheotomy, where, in the effort to speak, the organs of articulation and sound are seen to move, but neither noise nor musical sound is heard.

PATHOLOGICAL RELATIONS.—The morbid modifications of vocalisation are necessarily numerous, in proportion to its complexity and the number of parts concerned in its production; but it may be generally stated that, in consequence of disease or perverted action in any of these parts, the voice may be altered either (1) in *loudness or force* (size of sound-waves); (2) in *pitch*, or relative height of its note (rate of sound-waves); or (3) in *quality* (shape of sound-waves). And even before there is obvious change in any of these respects, the power of passing rapidly and easily from one pitch to another, constituting melody, may be seriously impaired. The morbid alterations of the voice in these several directions will now be discussed; and brief reference will also be made to (4) *stammering of the vocal cords*; (5) *aphonia*; and (6) *the vocal signs in the chest*.

1. Changes in Force.—The voice is weakened in every disease which lowers the general muscular tone, or depresses the nervous system. The gradual change is well seen in phthisis without laryngeal affection, where at last even the effort to approximate the vocal cords is too much for the patient, and he speaks in only a whispering voice. In the same way painful affections of any of the parts enumerated above, interfering with muscular effort, weaken the voice. Speaking generally, mere diminished loudness of the voice depends usually on general rather than on laryngeal disease.

2. Alterations in Pitch.—The note of ordinary speech may be habitually raised or lowered, and the range of the singing voice may be seriously limited. The note of the voice depends primarily on the rate of vibration of the vocal cords; and this is the mean result of the tension, the length, the

density, and the thickness of the cords at the time the tone is produced, and the force of the current of air sent through the rima. Structural changes, therefore, in any one of these respects, will alter the pitch of the voice; and change in the ordinary vocal note is usually to be referred either to an affection of the tensor muscles of the larynx, or to structural change in the mucous membrane covering the cords or in the cords themselves. Lowering of the pitch of the speaking and of the range of the singing voice occurs with any condition which relaxes the mucous membrane, weakens the nervous and muscular systems, or makes the tense condition of the vocal cords and the consonating cavities painful. Paralysis of the intrinsic muscles, which admits of the approach of the vocal cords but interferes with their tension and density, affects the pitch as well as the quality, the voice being rough and deep in the paresis of the external tensors (superior laryngeal nerve).

The imperceptible transition from the 'chest' to the 'falsetto' voice, in which, while the condition of the vocal cords is suddenly altered, the form of the larynx, trachea, and other consonating cavities is simultaneously changed, requires a perfect control of the vocal organs, attained only by accomplished vocalists. On this change of register occurring involuntarily, the cracked voice in speaking is the result; and being referable to imperfect co-ordination, it is common in males about puberty, when the form of the larynx is changing, or may even persist occasionally throughout life. A curious affection of the pitch of the voice, in speaking, when two tones of different pitch are simultaneously produced (diphonia), appears to depend on the division of the rima into an anterior and posterior opening, either by small morbid growths, strings of mucus, or irregular action of the muscles.

3. Change of Quality.—The quality of the voice is affected by every alteration, either in the cords or in the consonating cavities, the purity and character of the tone being liable to numerous modifications, until, the musical note disappearing entirely, mere noise (aphonia or whispering) remains.

Hoarseness.—Hoarseness and huskiness, a combination of whispering and a badly sustained musical note, imply imperfect and irregular approximation of the vocal cords. Over-exertion of the voice, catarrh, &c., produce it temporarily, giving rise to irregular tension of the cords, to shreds of mucus, and to swellings of the mucous lining, which interfere with their regular approximation; while all changes short of those which absolutely prevent the closure of the cords may cause it as a persistent phenomenon. To the larynx itself we look, therefore, for the source of persistent hoarseness. Among the pathological conditions of which hoarseness is a symptom are chronic swelling of the mucous membrane, general or local, interfering with the closure of the rima; exudation or ulceration, and therefore inflammation, simple, specific, or diphtheritic; neoplasms; old cicatricial contractions; paralysis of the adductors of one cord, which necessitates the crossing of the normal cord beyond the median line to meet its fellow, as well as other forms of paralysis; and fixing of one vocal cord by ankylosis of the crico-arytænoid joint. The paralysis may, of course, depend on disease of the nervous centres, on the nerves themselves, or on pressure by intrathoracic or other tumours on the nerve-trunks. Hoarse-

ness, short of aphonia, is also a symptom of general exhaustion, as seen in phthisis and cholera.

The character of the speaking voice is also altered by any change in the resonating cavities. Thus the voice is said to have a *nasal twang* when the upper pharyngeal and nasal cavities are not completely shut off in vocalisation; while, on the other hand, obstruction in the nares, preventing the passage of the air through them and the formation of the nasal consonant sounds, is popularly, though wrongly, called 'speaking through the nose.' If the obstruction be situated in front of the nares, the sounds can be produced, but not continued; if the obstruction be in the upper pharynx, or at the back of the nares, they cannot be produced at all. When with obstruction in the nares there is swelling of the pharynx and the soft palate, the voice assumes the character so familiar in cases of quinsy.

Changes in the walls of the chest and the pectoral cavities also alter the character of the voice, the hollow voice of the emaciated phthisical patient illustrating this. The phonation, on inspiration, of spasmodic croup and of child-crowing or laryngismus stridulus must be referred to as the result of spasm of the vocal cords, though this involuntary function hardly comes within the definition of 'voice,' which term should be limited to sound produced in the vocal organs to establish communication between living beings.

4. Stammering of the Vocal Cords.—Spasm and defective co-ordination are the source of this peculiar affection of the voice, in which there are sudden interruptions of the voice without affection of the articulation (Prosser James).

5. Aphonia.—The various modifications of voice hitherto considered only occur where the vocal cords are free to meet more or less perfectly. Aphonia, or complete loss of the musical tone, occurs where the cords cannot meet. Among the conditions which will prevent the approximation of the cords and cause aphonia are paralysis or paresis of the adductor muscles, on whatever cause it may depend; fixation of the cords by cicatricial contractions, or by ankylosis of the crico-arytænoid joints; their destruction by ulceration; any painful affection which makes the patient unconsciously rest them; or coating with false membrane. But by far the most frequent cause of this aphonia is the abrogation or perversion of the will, occurring in the morbid mental condition of hysteria or other nervous disease; perfect power of phonation existing, but the patient, for months or years, declining to exercise the power, or to make the necessary effort. The intimate relations of the voice to the higher functions of the brain would lead us to expect that it would be influenced by the emotions. Thus we have a person hoarse with rage, speechless with terror, &c.; and the origin of these cases of nervous aphonia is frequently some sudden emotion, causing loss of control over the voice. Ziemssen believes that, even where the vocal cords cannot meet, by a great effort the patient may produce a hoarse, monotonous tone by vicarious vibrations of the ventricular bands.

TREATMENT.—The local treatment of the various diseases of the respiratory organs which give rise to disorders of the voice is fully discussed in the several articles on these subjects (*see* LARYNX, Diseases of; STAMMERING; PHARYNX, Diseases of; TRACHEA, Diseases of). The *general* treatment

will depend upon the constitutional state. See HYSTERIA; and PHTHISIS.

6. Vocal Signs in Chest.—Some consideration must be given to the changes in the voice which are audible when the stethoscope is applied to the chest. Normally the vibrations of the vocal cords are conveyed to the ear applied to the chest by propagation along the contained air, the rigid portions of the air-chambers, and the thoracic walls, the voice being heard as a feeble, buzzing, musical tone. This sound is weakened when the original tone is weakened by laryngeal disease; when fluid is interposed between the lung and the chest-wall; and when the bronchial tubes are obstructed by secretions or other cause, preventing the conveyance of the vibrations. The sound may, on the contrary, be exaggerated, giving rise to the phenomena of *bronchophony*, *amphoric voice*, and *egophony*. Bronchophony is simple increase in the vocal resonance, and is heard under the same conditions which give rise to bronchial breathing, that is, over lung consolidated by exudation or condensed by compression, and over cavities with solid walls. The term *pectoriloquy* is commonly applied to excessive bronchophony, but Bristowe limited it to those instances in which not only the laryngeal tone, but the articulate sounds produced in the mouth, are conveyed back to the chest, and thence through the chest-wall to the ear. Amphoric voice (*amphorophony*) is the term applied where the vocal resonance is not only increased, but acquires a metallic ring, from the addition of a consonant tone acquired in large cavities of the lungs. This is sometimes heard in a marked degree in pneumothorax, though in other cases of the same disease the vocal resonance may be absent or greatly diminished.

Egophony is heard where there is a thin layer of fluid between the ear and the lung, as in small effusions or at the margin of larger effusions. It is a bleating, tremulous tone, supposed by Stone to result from the interposed fluid preventing the fundamental note-vibrations from reaching the ear, while it permits the finer and closer vibrations of harmonics to penetrate. The same sound may be heard in some cases of consolidation of the lung. See PHYSICAL EXAMINATION. THOMAS J. WALKER.

PERCY KIDD.

VOLVULUS (*volvo*, I roll).—A form of intestinal obstruction in which the bowel is bent or twisted on itself. See INTestinal OBSTRUCTION.

VOMICA (*vomo*, I vomit, I cast up).—SYNON.: *Fr. Vomique*; *Ger. Lungengeschwür*.

DEFINITION.—A term applicable to all ulcerative spaces in the lung in open communication with bronchi. See CAVITY.

VOMIT : Examination of Vomited Matters.—Vomited matters may consist either of substances present in the stomach when vomiting begins, or of substances entering it during the process. Those present in the stomach when vomiting begins include articles of food and drink, drugs, poisons, or other ingesta, more or less altered by digestion or fermentation; saliva, epithelium, mucus, pus, or blood from the nasal passages, mouth, pharynx, or œsophagus; fluid or mucus secreted by the stomach itself, epithelium-cells, casts of tubules, or even shreds of gastric mucous membrane, blood more or less altered proceeding from the walls of the stomach, cells or small pieces

from morbid growths; fungi, as *torulæ* and *sarcinæ*; parasitic worms; bile; pancreatic juice; pus from abscess of the stomach or liver; or feculent matter from the intestine. During vomiting much saliva may be swallowed; and bile, pancreatic juice, pus or feces, not originally present in the stomach, may be pressed into it by the straining. Effusion of blood into its cavity may also be caused by the efforts of retching.

Method of Examination.—In examining the vomited matters it is advisable, first, to separate the larger pieces of undigested food by filtering the vomit through canvas or muslin.

The solid residue may be investigated by washing the larger pieces and tearing them up, or making sections of them, so that their nature may be ascertained. Partially digested curd is sometimes not very easy to recognise. If a large quantity of milk have been drunk at one time, the curd which it forms in the stomach may, when vomited, have the appearance of a piece of thick, dense, grey felt.

The filtrate should be put into a conical glass and allowed to settle. The reaction of the fluid is to be ascertained by litmus paper. For the methods of identifying the various acids—hydrochloric, lactic, acetic, butyric, &c.—and other volatile substances, see STOMACH, Examination of, p. 1591.

To ascertain the presence of pepsin in the vomit we add to it its own bulk of dilute hydrochloric acid (ten minims of diluted hydrochloric acid, B.P., to an ounce of water) and a flock of fibrin or a piece of hard-boiled white of egg; let it stand for several hours in a warm place; and then see whether or not the fibrin or albumen is dissolved.

To test for trypsin, we proceed in the same manner, but use the vomit without the addition of acid; and if it be already acid, neutralise it with bicarbonate of sodium.

To test for rennet, we add some neutralised filtrate to some neutral milk, and let the mixture stand at 98° for half an hour. If rennet is present coagulation will occur.

To test the vomit for peptone, we must put some of it into a small dialyser, and let it stand for some hours. We then add to the water in which the dialyser has stood solution of corrosive sublimate, which gives a precipitate with peptones; or some liquor potassæ and a drop of very dilute solution of sulphate of copper, which gives a precipitate dissolving on shaking, and forming a rose-red solution, changing to purple when more copper is added.

Bile is tested for in the filtered liquid by Gmelin's and Pettenkofer's tests.

For blood in the vomit, see HÆMATEMESIS, p. 620.

If the vomited matter be too thick to allow the sediment to subside, a little of it should be mixed with some distilled water and allowed to settle. A drop of the sediment is then to be examined microscopically; and the examination is facilitated by adding to one specimen a drop of iodine-solution, and to another a drop of aniline red or blue solution. The substances most likely to occur are partially digested fibres of voluntary or involuntary muscle, elastic fibres, connective-tissue bundles from meat in the food, spiral fibres and green chlorophyll-granules from vegetables, starch-granules (stained blue by iodine), *torulæ* or *sarcinae*, blood-corpuscles, leucocytes, scaly epithelium from the mouth, cylindrical epithelium from the stomach, and casts of the gastric follicles—sometimes fibrinous, sometimes composed of cells and granules

which take up the aniline colour and are thus rendered more easily visible.

Poisons in vomited matters must be searched for by special chemical methods.

Clinical and Pathological Indications.—

If the vomited food be unchanged, or but little changed, it indicates either that the vomiting has occurred soon after a meal, or that the secretion of gastric juice is deficient either in quantity or in quality. The food is usually comparatively little changed in nervous vomiting, or in cancer of the cardiac extremity of the stomach. In vomiting from cancer of the pylorus, or duodenal ulceration, the food is much more digested, as it remains much longer in the stomach. If undigested food be vomited some hours after a meal, the vomit should be examined in order to ascertain whether pepsin or acid be deficient. Complete absence of hydrochloric acid has been observed in cases of cancer and amyloid degeneration of the stomach, and a deficiency of this acid has been found experimentally in acute anæmic and febrile conditions. Abnormal acidity from fermentation of saccharine or farinaceous articles of food, and the consequent production of acetic, lactic, and butyric acids, occurs in chronic catarrhal conditions. In some cases of gastric catarrh starch appears to undergo a mucous fermentation, and large quantities of glairy material are formed. When fermentation has gone on to a great extent, the vomit may have a yeasty look, and should then be examined for sarcinæ and torulæ. See SARCINA; TORULA; and STOMACH, Dilatation of, p. 1595.

Sometimes large quantities of a watery fluid are vomited. This is occasionally alkaline or neutral, contains potassium sulpho-cyanide, and digests starch. It consists of saliva, which has been secreted abundantly on account of reflex irritation arising from the stomach, and swallowed. At other times it is strongly acid, and appears to be secreted by the stomach. Sometimes the vomit appears to be a mixture of both of these fluids. Such vomiting may occur from nervous disturbance of the stomach, but may be symptomatic also of catarrh, ulcer, or cancer. Mucus in the vomit indicates catarrh of the gastric mucous membrane; and the more acute the inflammation, the more leucocytes occur in the mucus. Bile may be vomited pure, in the form of a tasteless golden-yellow substance like yolk of egg, from the action of poisons, but this rarely happens. Vomiting of bile, more or less green and diluted, or mixed with digestive secretions or food, occurs as a symptom in congestion of the liver; but it may take place in all kinds of vomiting, whatever its cause. Large quantities of bile, mixed with the secretions from the mouth and stomach, and forming a grass-green liquid (*vomitus æruginosus*), may be vomited in peritonitis and cerebral affections. The writer has also observed this character of vomit in opium-eaters. Constant absence of bile, when vomiting is persistent, points to pyloric stenosis. Pus may get into vomit from the bursting of an abscess in the mouth or tonsils; it sometimes, though rarely, may arise from an abscess in the walls of the stomach; but it is more likely to come from abscess of the liver. Blood vomited in large quantity, and of a bright red colour, usually indicates ulceration of the stomach or cirrhosis of the liver. More or less altered, and in smaller quantity, it occurs in the diseases just mentioned, and also in cancer and

yellow fever (see HÆMATEMESIS, p. 620; and YELLOW FEVER). It may also be present in hysterical persons who have swallowed blood, obtained from external sources, or by sucking hollow teeth. Cancer-cells in the vomited matters are diagnostic of the presence of malignant disease.

T. LAUDER BRUNTON.

VOMITING (Lat. *vomo*). — SYNON.: Fr. *Vomissement*; Ger. *Erbrechen*.

DEFINITION.—Forcible expulsion of the contents of the stomach through the œsophagus.

ÆTIOLOGY AND PATHOLOGY.—Two things are necessary in vomiting, namely, squeezing of the stomach, and relaxation of its cardiac orifice. Although the muscular fibres of the stomach itself may contract under the action of emetics, they seem to be unable to expel its contents unless aided by external pressure, for when the muscles of the abdomen are cut across vomiting does not occur. When the diaphragm and abdominal muscles are paralysed, vomiting is impossible, though the stomach may be in active movement. The stomach is not necessary to vomiting, which will occur when that organ is excised, and a simple bladder tied in its place; but when the stomach is present, mere pressure upon it by the diaphragm and abdominal muscles, as in coughing, does not expel its contents. In vomiting, the contents of the stomach are squeezed out of it by the mechanical pressure of the diaphragm and abdominal parietes contracting simultaneously. When these muscles contract, if the cardiac orifice of the stomach remains closed, an ineffectual effort at vomiting, or retching, occurs; but if the cardiac orifice dilate, the gastric contents are expelled. The cardiac orifice is relaxed by means of the longitudinal fibres, which run along the under end of the œsophagus below the diaphragm, and then radiate completely over the stomach. When they contract they dilate the cardiac orifice, and at the same time aid the evacuation of the stomach by drawing the whole viscus towards the diaphragm. In the act of vomiting, then, the simultaneous contraction of three sets of muscles is required: (1) of the diaphragm, (2) of the abdominal wall, and (3) of the muscular fibres just mentioned in the stomach itself. The movements of these muscles are co-ordinated by a nervous centre, situated in the floor of the fourth ventricle in the medulla oblongata. This centre is closely associated with, though of course not identical with, the respiratory centre. The motor impulses from this centre are sent to the abdominal muscles, diaphragm, stomach, and œsophagus, by the intercostal, phrenic, and pneumogastric nerves respectively. The reasons for supposing that the nervous centre for vomiting is closely associated with the respiratory centre, are that the movements of vomiting are modified respiratory movements, that emetics excite the respiratory centre, and that their action is usually preceded by increased respiratory movement, while depression of the activity of the respiratory centre stops vomiting. When the blood is rendered very arterial by excessive respiration, a condition of apnoea, in which no need of respiration is felt, and no respiratory movements are made, is produced; but if emetics are then injected into the veins, respiration not only becomes more frequent, but apnoea can no longer be induced, unless the activity of the respiratory centre be lowered by narcotics.

The vomiting centre is usually excited to action by irritation of certain *afferent nerves*. These may act either directly upon it, or through the medium of the brain. The nerves of special sense act through the brain. The sight of a disgusting object, a disagreeable stench, or an unpleasant taste, may excite vomiting, and it may also be produced by the simple thought of such subjects. A blow on the head, or inflammation of the brain or its membranes, also excites vomiting. According to Budge, the cerebral centres for the movements of the stomach are in the right corpus striatum, and especially in the right optic thalamus. When these parts are irritated the stomach moves. Irritation of the corresponding parts on the left side of the brain does not affect the stomach. Vomiting occurs in certain cerebral conditions, either affecting the brain itself or its membranes, such as cancer or tubercle of the brain, apoplexy, cerebellar hæmorrhage, softening of the cerebral substance, sometimes encephalitis, poisoning by narcotics, melan- cholia, profuse hæmorrhage, or tubercular meningitis. It is also one of the symptoms of Menière's disease of the semicircular canals. It also occurs in various diseases, in which, however, it is difficult to say whether the vomiting be due to direct affection of the brain itself, or to reflex action upon it from other organs. Such diseases are typhus, plague, yellow fever, cholera, and the cold stage of ague. Very painful impressions on sensory nerves throughout the body may excite vomiting. This is seen in cases of loose cartilages in the knee, in dislocation of a joint, or in a painful wound or operation. Here, also, it is uncertain whether the vomiting be produced through a direct connection of sensory nerves with the vomiting centre, or whether the irritation acts indirectly through the cerebrum.

Certain afferent nerves appear to have a more direct connection with the vomiting centre than others, and these require special consideration :—

1. *Branches of the glosso-pharyngeal nerve to the soft palate, the root of the tongue, and the pharynx.*—These parts have a very close connection with the vomiting centre, and tickling them with the finger or with a feather is one of the readiest means of inducing vomiting.

We find vomiting occurring in inflammation of the soft palate or tonsils, and also of the pharynx, especially in children.

2. *The nerves of the stomach.*—The sensory nerves of the stomach are chiefly branches of the vagi, but they belong partly also to the sympathetic system. When the vagi are cut, vomiting becomes difficult, but efforts at retching occur, and the writer has seen vomiting take place from the action of emetics after section of the vagi. It is therefore evident that irritation of the stomach produces vomiting reflexly through other nerves than the vagi. Vomiting may occur from irritant substances in the stomach, whether introduced into the stomach, or formed within it; from irritation within the stomach, due to an inflamed or irritated condition of its walls; or from mechanical pressure, from without or from within. Thus it may occur from the presence of undigested food, from irritating substances produced by imperfect digestion, or from irritant poisons within the stomach. It may be due to catarrh or congestion of the mucous membrane itself, to softening of the mucous membrane, or to ulcer or cancer in the gastric wall. It may be produced by extreme distension of the stomach by

gas, liquids, or solids; by compression of a part of it within the body, as in hernia of the stomach; or by the pressure of a tumour upon it. It may be caused by violent compression externally with the hands; by the pressure of a too tightly laced corset; by the pressure against the abdominal walls of hard tools or benches in certain trades. It frequently occurs in cough, especially the cough of phthisis; but here it is probable that the vomiting is due partly to the violent compression between the diaphragm and abdominal walls, and partly to the congestion of the vessels which the continued interruption of the circulation during the fit of coughing brings on.

3. *The nerves of the liver and gall-duets.*—These consist chiefly of branches of the vagus and sympathetic. From irritation of them vomiting occurs in hepatitis, or during the passage of a biliary calculus. It is from irritation of these branches, also, that vomiting may occur in pleurisy of the right side, the congestion of the pleura on the upper surface of the diaphragm having led to congestive changes in the liver.

4. *Intestinal nerves.*—Ligature of the intestine in animals produces vomiting, which is arrested by dividing the nerves passing from the ligatured parts. In man it is the almost invariable accompaniment of strangulated hernia or intussusception, and it may even occur in obstruction of the bowel by fecal matters in cases of obstinate constipation. It also takes place in peritonitis from irritation of these nerves.

5. *The renal nerves.*—Vomiting occurs from irritation of these nerves, as by calculi in the pelvis of the kidney or passing down the ureter, and also in nephritis.

6. *Vesical nerves.*—In cystitis vomiting occurs. It may possibly be due, however, not to irritation of the vesical nerves, but to extension of inflammation to neighbouring parts.

7. *Uterine nerves.*—Irritation of these nerves is one of the commonest causes of reflex vomiting. It may be produced in animals on irritation of the uterine plexus, and occurs in the human subject during pregnancy or in metritis.

8. *Ovarian nerves.*—Vomiting is a symptom of inflammation of the ovaries.

9. *The nerves of the testicle.*—A blow on this organ tends very readily to produce nausea and vomiting.

The cause of vomiting in *sea-sickness* is uncertain; but it appears to the writer to be partly due to the condition of the nerve-centres, and partly to that of the viscera. See SEA-SICKNESS.

TREATMENT.—The treatment of vomiting is to be directed to two ends—(1) to *remove the cause* if possible; and (2) to *lessen the irritability of the vomiting centre*. The chief drugs which lessen the irritability of the vomiting centre are opium, morphia, bromide of potassium, chloral hydrate, and probably, also, hydrocyanic acid and belladonna. Strychnine and small doses of ipecacuanha are also useful in vomiting, and they probably owe their power to their action on the vomiting centre. Most of these drugs have a local sedative action on the stomach, and therefore it is advantageous to give them by the mouth when possible. Even when the stomach is very irritable, they may be retained by giving them in a concentrated form. When the stomach will not retain them, they must be given by the rectum or by subcutaneous injec-

tion. In sea-sickness the effect of the position of the head is sometimes very marked, and the vomiting may occasionally be arrested completely by removing all pillows and putting the head on a level with, or rather lower than, the body.

In cases of disease of the brain or its membranes, where it is difficult or impossible to remove the cause, we must try to lessen the congestion by means of leeches and cold applications to the head; and also to soothe the vomiting centre by diluted hydrocyanic acid, or by bromide of potassium. At the same time, however, considerable benefit is obtained from the use of remedies which act locally on the stomach, these seeming to have some reflex effect upon the vomiting centre. One of the most useful is ice, which may be constantly sucked, and also swallowed in small lumps. Where the vomiting is dependent on the action of the poisons circulating in the blood, as in the later stages of contracting kidney, we must endeavour to eliminate these by increasing the action of the kidneys and of the skin. In vomiting dependent on inflammation of the mouth and fauces, we lessen the irritability by soothing or astringent gargles, confections, or glycerine. A confection or glycerine is often better than a gargle, inasmuch as it remains longer attached to the parts, and thus exercises a more prolonged effect upon them. When vomiting is due to irritant substances in the cavity of the stomach, such as indigestible food, and acrid fluids or poisons, it is best treated by evacuating them. A large draught of lukewarm water, alone or mixed with a teaspoonful of mustard, is one of the best means. Large draughts of warm water alone, even if they are not ejected, may give relief by diluting the acrid substances in the stomach so much as to prevent their irritating the mucous membrane. In this way they sometimes relieve sick-headaches. It is of great importance sometimes, not only to prevent the formation of acrid substances by slow and imperfect digestion, but to prevent the mechanical irritation of the mucous membrane by undigested food. For example, we not infrequently notice that sickness and vomiting will occur in susceptible individuals after meals containing such substances as are not only slowly digested, but are swallowed in lumps. Examples of these are uncooked apples and cheese, or even potatoes, especially when imperfectly boiled or new. These articles, instead of being crushed to a powder by the teeth, are swallowed in lumps of considerable size, and apparently, instead of passing the pylorus, are retained in the stomach, and, partly by the mechanical irritation, and partly by their giving rise to acrid products, cause sickness. Milk, when swallowed in large draughts, or when there is too much acidity in the stomach, instead of falling in fine flakes will coagulate in large lumps, which have an effect similar to that of cheese. To prevent this it is advisable to mix the milk with soda-water or lime-water, or to take it, as in the whey-cure, by sipping.

When vomiting is due to slow or imperfect digestion, which allows decomposition or fermentation of food to take place in the stomach, it may be arrested by improving the digestion. Thus five grains of calomel, by acting on the stomach through the liver, may arrest vomiting; and tincture of walnut (the active principle of which, juglandin, is an hepatic stimulant) has also been recommended. Pepsin also, by facilitating digestion, may prevent

vomiting; and bitters, such as calumba, may do so by preventing putrefaction or fermentation.

When decomposition or fermentation of food, with formation of acrid or irritating products, has once set in, it may continue a long time, as the organisms which cause it remain constantly in the stomach, and renew the process in every fresh supply of food. It may be stopped by antiseptics. Where the vomited matters are frothy and yeasty-looking, the sulphurous acid of the Pharmacopoeia, in doses of one fluid drachm, diluted with half a wine-glassful of water, often arrests such vomiting like a charm. Creosote has a similar action, but possibly has some additional action on the nervous system, as it is useful even in cases where the vomiting does not appear to be due to decomposition of food. In dilatation of the stomach these antiseptics may fail, and the best results are obtained by washing out the stomach by a syphon-apparatus. *See LAVAGE.*

For the treatment of irritant poisoning, *see POISONS.*

When the mucous membrane of the stomach itself is inflamed or irritated, we must try to lessen the irritation. The best drugs for this purpose are ice, diluted hydrocyanic acid, opium, and bismuth. The insoluble salts of bismuth, and especially the subnitrate, are to be preferred to the solutions; and it is advisable to combine them with magnesia, potash, soda, or carbonate of lime, according to the condition of the intestines, preferring the magnesia when the bowels are confined, and carbonate of lime when they are too loose. Sometimes the tendency to vomit is increased by lying on the right side. This is probably partly due to the dragging of the stomach itself upon the cardiac extremity, and partly to the difficulty with which gaseous eructations escape from the stomach in this position. When there is a tendency to vomit, therefore, the patient should lie down on the left side after a meal. In the vomiting of hepatitis, in addition to opium and diluted hydrocyanic acid, we may use ice-water, or ice swallowed, and leeches over the liver. In biliary calculus, we may give, along with opium, a full dose of ether internally, and in addition may employ ether or chloroform by inhalation; similar treatment may be adopted in cases of renal calculus.

In intussusception or hernia we must remove the cause, if possible. In peritonitis full doses of opium are best. For the vomiting in cystitis and ovarian diseases we must lessen the sensibility of the vomiting centres by the drugs already mentioned, and treat the local conditions.

In the vomiting of pregnancy we trust partly to the drugs already mentioned to act on the vomiting centre, and partly to local applications. It is sometimes arrested by the application of a 10-per-cent. solution of nitrate of silver to the os uteri, or by slight detachment of the membranes around the margin of the internal os. Where all other methods fail, the induction of premature labour must be resorted to. *See PREGNANCY, Diseases and Disorders of, p. 1307.*

T. LAUDER BRUNTON.

VON GRAEFE'S SIGN.—*See EXOPHTHALMIC GOITRE.*

VON RECKLINGHAUSEN'S DISEASE.—*SYNON.: Multiple Neuro-fibromata. — See TUMOURS.*

VOYAGES.—See SEA-VOYAGES.

VULVA, Diseases of.—**SYNON.** : Fr. *Maladies de la Vulve*; Ger. *Krankheiten der Schamritze*.—The vulva or external genitals of the female comprise all the structures external to the hymen, having the navicular fossa and perinæum behind, the urethral orifice, vestibule, clitoris, and mons Veneris in front, and at the two sides the nymphæ and labia majora. These organs may be the seat of many diseases, which will be described in the following order :—

1. **Atresia Vulvæ.**—Closure of the genital fissure is sometimes found as a *congenital* malformation. This is usually of itself of no great importance, as it is almost always associated with other defects in development, as with atresia ani, hermaphroditism, and extroversion of the bladder. *Acquired* atresia results from mismanagement of infantile vulvitis, from injuries of the pudenda in childhood, or from cicatrization following upon some ulcerative process. It may give rise to trouble in micturition if the urethral orifice be involved. In rare cases it is only after puberty that trouble arises, from retention of the menses, or after marriage, from dyspareunia; or even during labour, from narrowness of the orifice delaying the escape of the infant. In the slighter and more recent cases, where the labia have only been agglutinated, they may be torn apart by pressure with the thumbs; or by passing a probe or sound behind the line of adhesion, and tearing it up with the handle of a scalpel, or with the nail. Where the union has become organised, the edge of the knife must be employed. In any case the patient should be anæsthetised; and care must be taken to prevent the reproduction of the adhesions.

2. **Inflammation.**—**SYNON.** : Vulvitis.—Inflammation of the vulva may be (a) *general*; or (b) *localised*.

(a) *General vulvitis.*—This variety is found affecting the apposed aspects of the labia, and the whole of the mucous surface they enclose, up to the borders of the hymen. It may be due to gonorrhœal infection, in which case the catarrhal process is apt to extend to the urethra and the vagina. In infants and young children of strumous constitution, it sometimes arises from exposure to cold, want of cleanliness, or irritation from ascarides that have passed out of the anus. In the adult it may result from injury, or rude coition; or from the escape of acrid uterine or vaginal discharges. It shows itself with heat, and tenderness, or sometimes itching in the part; a discharge of viscid glairy mucus bathes the surfaces, which sometimes becomes purulent, and glues together the labia. When the labia are separated the seat of the mischief is exposed, and is seen to be red, and sometimes painful to touch. Apart from the constitutional remedies that may be indicated in individual cases, the treatment consists in keeping the parts at rest, and carefully clean; and in bathing or douching the surface with hot water, followed by an astringent lotion or dusting powder.

(b) *Localised vulvitis.*—The vulvitis may be localised—(a) in the *vestibule*; (β) in the *navicular fossa*; or (γ) in the *muciparous follicles and glands*, and especially in the *Bartholinian glands*. (a) and (β) occur under the same conditions as the more diffused inflammation, and may be a source of considerable distress in walking or when the part is

touched, without being attended by any leucorrhœal discharge. Such cases require the same treatment as cases of general vulvitis; only it is important to make the applications directly to the affected spot, and hence it is usually best to apply sulphate of copper in the form of a solid crystal. (γ) Inflammation in the Bartholinian glands is the commonest cause of *labial abscess*. It may attack females at any age, but is most frequently seen in women from twenty to thirty, whether married or unmarried. It may arise during pregnancy, and some women are liable to repeated attacks, the glands becoming swollen and cystic whenever the patient gets a chill. In some cases there is profuse secretion, which escapes freely. Far more frequently the swelling in the lining of the duct leads to occlusion. This occlusion is in some instances transitory, and when the swelling in the duct and around its orifice subsides, the secretion that had accumulated escapes. In other instances the duct becomes permanently closed. These are the cases where the patient acquires a swelling in the labium majus of the affected side, varying in size from a pigeon's to a hen's egg. The accumulated fluid may be clear and limpid; but often it is turbid and distinctly purulent. In all the suppurative cases, if the cyst be evacuated through a small opening, the fluid is apt to reaccumulate, and the cyst-walls are now more likely to be the seat of a mischievous inflammation. Hence the *treatment* consists, first, in trying to reduce the inflammation with hot fomentations or poultices, perhaps aided by sedative lotions containing belladonna or laudanum, with the view of getting the orifice relaxed; and where the contents do not escape through the duct, the cyst should be freely opened, and the cavity cauterised. Huguier's suggestion, to extirpate the gland, is not ordinarily required, especially if the evacuation be effected with a Paquelin's thermo-cautery, which first makes a large and safe cut into the cyst, and can then be applied to the interior, so as to destroy the secreting surface.

Specific inflammations.—The specific inflammations, *gonorrhœal* and *syphilitic*, are treated of in their respective articles. But it is to be noted that the vulva may be the seat of *erysipelas*; and female children have sometimes suffered from a gangrenous vulvitis or *noma*, of the same nature as the *noma* of the mouth and cheeks, which may come on after some of the eruptive fevers, such as scarlatina, and requires the same kind of treatment, with chlorate of potassium and tincture of the perchloride of iron and stimulants, from an early stage of the mischief. The labia and perinæum are also occasionally the seat of *lupus*, which may be mistaken for cancer, but is not attended by enlargement of the inguinal glands, and should be treated by curetting and the application of escharotics.

3. **Eruptions.**—Various forms of eruption may be met with in the vulva, sometimes on the nymphæ or internal aspect of the labia majora, sometimes on the external aspect, and extending to the mons Veneris, or inside of the thighs. The commonest are *eczema*, *erythema*, *herpes*, *prurigo*, and *acne*. They are diagnosed and treated in the same way as the same affections in other situations. It is to be remembered, however, that the tendency to chronicity, which is a marked feature of inflammatory processes in the sexual apparatus generally, is apt to show itself in these vulvar eruptions, and that patients, from motives of

delicacy, are apt to allow them to remain untreated for too great a length of time. Further, many of them give rise to itching, which tempts the patient to rub and scratch herself to obtain relief, and may thus perpetuate the pruritus.

4. **Pruritus.**—**ÆTIOLOGY.**—Itching of the vulva is a not infrequent symptom of some of the eruptive and inflammatory affections of the part, when these are either healing or have got into a chronic stage; and it may remain as a permanent trouble after healing has taken place, if the patient have acquired the habit of relieving herself by friction. Sometimes it is reflex, from oxyuric irritation in the rectum, or when the worms have travelled from the anus to the vulva. Or it may be associated with disease of the vagina or uterus, as in cases of chronic colpitis or carcinoma of the cervix, in which the itching may either be due to irritation from the nature of the discharge, or be a reflex phenomenon. In some instances it can be traced to circulatory disturbance in the labia, as when the veins are enlarged, and in the congestion of early pregnancy. In others the renal secretion is at fault; and it is such a common phenomenon in women who are the subjects of diabetes mellitus, that in every patient who suffers from pruritus vulvæ, the urine should be tested for sugar. Lastly, there are some cases that can only be described as idiopathic.

SYMPTOMS.—The itching may be localised on the internal aspects of the nymphæ, or of the labia majora, or around the clitoris or the perineum. In most cases of long standing it becomes diffused all around to the anus and inside of the thighs. Sometimes it is temporary, as in the pruritus of pregnancy, which passes off under careful management in a few weeks, or in rarer cases during the puerperium; in other patients it becomes chronic, and constitutes one of the most distressing troubles to which a woman is liable. The itching may come on only occasionally. For the most part it is likeliest to prove distressing when the patient is warm, as at bedtime or after exercise; but with some the feeling is never quite in abeyance, and the patient has the almost constant desire to relieve herself, as for a time she can do, by scratching or, rather, rubbing the parts with her dress or a towel, until the pain overcomes the feeling of itch, or sometimes until the collapse consequent on the onanistic orgasm which she has involuntarily produced, renders her for a time less sensitive to her trouble. The result of such friction, however, is to keep up the local irritation; so that even in cases where there may not originally have been any local pathological change, but where the itching and friction have persisted for months or years, the skin and mucous membrane become thickened and indurated, and a condition resembling that of a chronic eczema becomes induced. In several cases the writer has seen epitheliomatous nodules develop at the sides of the clitoris and in the vestibule.

TREATMENT.—When diabetes mellitus is present it must be combated; and if the patient is gouty, or have her urine too acid or alkaline, these conditions must be rectified. Morbid processes that may be present in the vulva or other neighbouring structures must be treated according to the requirements of the case. A carefully regulated non-stimulating diet should be enjoined, and a due amount of exercise. The internal remedies that have been found most helpful are bromide of potassium

and arsenic. Then, for the relief of the itching, the patient must be warned against the danger of rubbing the parts, and be taught to soothe it by bathing with very warm or sometimes with cold water, and drying the parts with a soft towel or napkin. It can be still better allayed by mopping with a lotion containing infusion of tobacco or belladonna or opium; or by applying afterwards a piece of lint soaked in black lotion, or a lotion of acetate of lead. The most effectual sedatives are ointments containing sulphur, camphor, tar, carbolic acid, thymol, iodide of lead, perchloride of mercury, bismuth, prussic acid, iodoform (deodorised with Tonquin bean), or cocaine. The free application of lunar caustic followed by cocaine has been strongly commended by Olshausen. In several otherwise intractable cases, patients in private practice and in the Buchanan ward of the Royal Infirmary of Edinburgh have been cured by ablation of the clitoris and surrounding mucous membrane, as far back as the vaginal orifice, and bringing the raw surfaces together by means of catgut sutures. See p. 1309.

5. **Tumours.**—The following enlargements may be found in the vulva:—

(a) *Hypertrophy.*—The clitoris has sometimes been found of a size sufficient to cause discomfort, and warrant its removal. Far more frequently the nymphæ are of unusual dimensions, their margins projecting beyond the labia, and then they are liable to become the seat of ulcerative processes, and require to be trimmed, which may be done with the knife or scissors, but better with the thermocautery. Enlargements of the labia majora, in the form of elephantiasis, are met with among Hindu women. The mass is sometimes of enormous size, and in consequence of the calibre of the nutrient vessels, ablation is apt to be attended by dangerous hæmorrhage, so that the application of an elastic ligature is in most cases the best means of effecting its removal.

(b) *Hernia.*—Hernial protrusions may occur into the labia, and be found among the swellings of this part. The detection and treatment are to be effected and conducted as in the case of other hernie.

(c) *Cysts.*—Cystic swellings are found in the upper part of the labia, when the canal of Nuck becomes the seat of an accumulation of fluid, which corresponds to hydrocele of the cord in the male; or lower down, when the duct of a Bartholinian gland has become occluded, and the secretion of the acini accumulates so as to distend the gland without its becoming inflamed. If a complete aspiration in either case is not followed by perfect cure, and the fluid reaccumulates, as it is apt to do, the second tapping should be accompanied by an injection of iodine; and in the case of the Bartholinian cyst, the wall of which is formed of a mucous rather than of a serous membrane, the evacuation may require to be effected through a larger opening, and followed by the application of a more powerful escharotic.

(d) *New-growths.*—Various neoplasms may have their seat in the vulva. At the orifice of the urethra not infrequently small red fleshy growths, the so-called *urethral caruncles*, make their appearance. They are sometimes unattended by any symptom; more frequently they cause intense suffering during micturition, during coitus, or when the patient takes exercise. The pain is usually referred to the urethral orifice, but it is sometimes reflected to

distant parts, as to the heel. Relief from suffering may be temporarily obtained by application of sulphate of copper or nitrate of silver; but cure is only effected by removal of the growth. It is imperatively necessary to remove not only the small red body, but the portion of the urethra from which it springs, and the raw surface should be freely cauterised if the ablation have not been effected with a thermo-cautery. *Specific* swellings, warty or gummatous, are, of course, frequently to be met with on the vulva. *Lipomata* sometimes grow under the skin of the labia pudendi. More frequently *fibromata* occur, which may attain considerable size, and demand removal. Lastly, the law that *carcinomata* have a predilection for surfaces where a transition takes place from one variety of epithelium to another is illustrated by the frequency with which different forms of cancer affect the vulva. Their development, symptoms, and treatment pre-

sent no special features. Only it is well to remember that when the mischief is met with in a stage where there is still hope of eradication, it is best to effect the removal of the neoplasm by some of the bloodless methods with which modern surgery has become familiar. The tissues in which the growth develops are very vascular; and while in some situations—as at and around the clitoris—it is comparatively easy to control hæmorrhage by pressure against the pubic bones, in the parts immediately to the side and back of the vaginal aperture the bleeding from a cut surface is apt to be uncontrollable and dangerous. Hence commencing *carcinomata* ought to be extirpated with the *écraseur*, or, better still, with Paquelin's thermo-cautery, which is the most serviceable of all instruments for the removal of the different varieties of neoplasm that infest the vulva.

ALEXANDER RUSSELL SIMPSON.

W

WANDERING OEDEMA.—See ANGIO-NEUROTIC OEDEMA, p. 70.

WARTS.—SYNON.: Lat. *Verruca* (an excrescence); Fr. *Verrue*; Ger. *Warze*.

DEFINITION.—The ordinary wart is a benign circumscribed cutaneous papilloma.

ÆTIOLOGY.—A wart is an aberration of growth of certain of the constituents of the skin; it is mostly found in children and elderly persons, and less frequently in the adult. As children become developed by growth and their tissues acquire strength, these partial exuberant growths disappear. In young persons of feeble organisation they are sometimes thrown out like an exanthem, and yield to a constitutional treatment directed towards the improved innervation and nutrition of the tissues. Their direct relation with the nervous system is often evinced by their sudden disappearance under the influence of mental emotion, a circumstance which has led to the popular use of charms for their cure. In elderly persons they are often met with on the face, where their presence must be ascribed to debility of integument; and they are frequently associated with dirt and neglect. Briefly, warts may be said to be due to aberration of nutritive function of the skin, consequent on defective organisation and vitality. Warts may be associated with Adenoma Sebaceum (p. 26).

DESCRIPTION.—Pathologically a wart is an hypertrophy or excessive growth of a small group of papillæ of the skin, forming a hard prominence of the integument. Warts vary in size, are never congenital, and are modified according to situation. They may be simple or branched, flat or acuminate, simple or multiple, or pigmented. One kind, the senile wart (*Verruca senilis*) is remarkable for the minimum of prominence, resembling a flat, dirty-looking blotch on the skin; while another, as on the hands, may have a prominence of a quarter of an inch, or on the scalp of half an inch.

Warts on the hands—the fingers, dorsum, wrist, not the palm—afford the commonest illustration of

verruca, as in this situation, from the greater nutritive energy of the skin and the abundance of epidermis, they are most frequent and most highly developed. When of recent growth they are convex and smooth on the surface, but when of longer standing the apex is flat, from the wearing away of the superficial cuticle, and the anatomy of the wart becomes disclosed. Then it is apparent that the wart is composed of a bundle of fibres, held together in a cylindrical form by a boundary of thickened cuticle. Each of these fibres is a vascular papilla of the skin, enclosed in a sheath of cuticle, and the collective mass forms the body of the growth. An old wart will frequently split up into several segments (*V. lobosa* or lobulated wart), and then its construction of fibres (*V. fibrosa*) is strikingly conspicuous. If a wart be cut through horizontally, the vascular papillæ will be cut across, and then the structure consisting of papillæ and horny sheaths is still more evident. On the fingers and especially the knuckles of children, the verruca is isolated and large; but it is not infrequently confluent, while on the back of the hands and wrists, as also on the forehead, it is often developed in crops, like an eruption; the latter, however, never attain the dimensions of the isolated warts of the fingers.

Thread-like, often pedunculated, warts (*V. filiformis*) are met with on the neck, chest, ears, and eyelids; warts may also occur on mucous surfaces, e.g. vesical (p. 145), and laryngeal (p. 853) papillomata.

Verruæ are generally sessile (*V. sessilis*); but on the scalp they are frequently pedunculate (chiefly in women), and, from a peculiarity of structure, have been denominated digitate (*V. digitata*). The digitate character of the warts of the scalp is due to the smaller quantity of epidermis occurring in that region; consequently the hypertrophied papillæ are not held together by a ring of thickened cuticle as elsewhere, but, being left to themselves, shoot out from the centre like fingers; the papillæ likewise grow to a greater length, and

their cylinder is swollen so that the bulk of the mass greatly exceeds that of the base from which they spring. Nevertheless, the digitate verruca must be distinguished from *V. acrochordon* and the cauliflower-shaped venereal warts (see ACROCHORDON). Both of these are growths of the integument, and are not restricted to the papillæ cutis alone; and thereby fall into the category of molluscum, with which, especially acrochordon, they are closely allied in pathological structure.

Telangiectatic wart-like patches are met with in angio-keratoma. See ANGIO-KERATOMA.

The normal colour of warts on the hands is a pale lemon-yellow, but from their roughness they are apt to retain dirt in their crevices, which gives them a brownish appearance. The flat warts of the trunk of the body and face are accompanied by the production of pigment, and their dirty colour is consequently more striking. See MOLLUSCUM CONTAGIOSUM; XANTHOMA; LICHEN PLANUS; MOLES; LUPUS; KERATOSIS.

Warts, especially senile flat warts, frequently give rise to pruritus, and may be the starting-point of transformation to malignancy. Black papillary formations accompany the very rare affection, Acanthosis Nigricans (see p. 12); and warty growths are not infrequently present in cases of myxœdema (p. 996).

DIAGNOSIS.—True idiopathic warts must be distinguished from other diseases which sometimes put on a warty appearance, especially carcinoma and syphilis. Epithelioma of the skin is occasionally seen as a circumscribed warty growth, but generally with adherent scabs covering superficial ulceration. These signs, together with infiltration of adjacent tissues, implication of neighbouring glands, and pain, would arouse suspicion. It must be remembered, however, that epithelioma frequently attacks a simple wart which has remained quite passive during a lifetime; rapid increase of growth with the above-mentioned symptoms would suggest the supervention of epithelioma.

Any chronic inflammatory process of the skin, especially syphilis, is liable to take on a papillary character. Without referring to the papillary growths of early syphilis (condylomata), which could scarcely be confounded with simple warts, on account of their position and moisture, mention may be made of the dry warty character assumed by old syphilitic lesions, especially such as have been preceded by ulceration. The history of the disease (previous ulceration, &c.), together with other concomitant symptoms of syphilis, would assist the diagnosis.

As of venereal origin, though never syphilitic, ordinary venereal warts must also be noted. Other names by which they have been described suggest their characters, such as 'pointed condyloma,' and 'cauliflower excrescence.' They are generally soft, bright red in colour; and the individual papillæ are pointed. The rapidity of their growth, and the situation where they usually occur (the genitals), serve to distinguish them from ordinary verruæ. Moreover, they most often accompany gonorrhœa, eczorrhœa, or other irritating discharge. Balanoposthitis may cause warts (p. 135, and p. 582). In the male they are on or under the prepuce, in the female on the inner side of the labia (see p. 1120).

Cadaveric warts, or dissecting-porter's wart or anatomical tubercle (*V. necrogenica*) is one of the rare effects of the application of tuberculous matter

to the skin. The back of the hand over the knuckles or the joints of the fingers is its usual site. The skin is warty and thickened, and may resemble epithelioma (p. 244). See SKIN, Tuberculosis of.

Peculiar wartlike granulomata characterise the remarkably infective endemic disease, Verruca Peruviana (p. 250). Melanotic warts are examples of pigmentary changes resulting from cell-action on the blood. Old-standing lesions of Lichen ruber planus occasionally take on a warty character (*Lichen verrucosus*). See *Lichen planus verrucosus*, p. 872.

PROGNOSIS.—Verruca is a blemish rather than a disease, and unimportant in its relation to the general health. By an error of diagnosis we sometimes read of malignant warts; and warts have been confounded with those fleshy growths termed 'tegumentary nævi.' In elderly persons a warty state of the skin is sometimes associated with asthenic ulceration, and occasionally with rodent ulcer, for which the nutrition of the skin, and not the wart, is responsible.

TREATMENT.—The best method of treating verruæ is to touch them with some solvent agent, such as glacial acetic acid. This acid dissolves the epidermis, and, reaching the vascular papillæ, destroys the whole structure of the wart down to its root, or a saturated solution of caustic potash may be carefully applied (p. 1759). The alkali acts more speedily than the acetic acid, and effects a more thorough cauterisation of the vascular plexus, from which the hypertrophied papillæ derive their capillary loops. This little operation may either be completed at one sitting, or it may be repeated daily until its purpose is effected. Where there are numerous verruæ to be dealt with, the process is tedious, and is generally left in the hands of the patient. The verruæ digitatæ of the scalp are speedily and easily removed by this process. In the exanthematous form the verruæ are too small and too numerous for the caustic application. These may be treated by friction with sulphur-ointment or tar-ointment; and in this latter form the verruæ are frequently entirely removed by a course of treatment with liquor arsenicalis, in three- or four-minim doses, taken immediately after meals, three times a day.

Salicylic-acid collodion, or the acid nitrate of mercury applied so that the limits of the wart are not transgressed; alum, tannin, calomel, and potassium bichromate, are also favourite remedies. For the treatment of venereal warts, see PENIS, Diseases of, p. 1179; and GLEET, p. 583.

JOHN HAROLD.

WASTING.—A synonym for atrophy. See ATROPHY, GENERAL; and ATROPHY, LOCAL.

WASTING PALSY.—A synonym for progressive muscular atrophy. See PROGRESSIVE MUSCULAR ATROPHY.

WATER, Ætiological Relations of.—See PUBLIC HEALTH.

WATER, Therapeutic use of.—See BATHS; HYDROTHERAPEUTICS; MINERAL WATERS; SEA-AIR, SEA-BATHS; and TEMPERATURE.

WATER-BRASH.—A popular synonym for pyrosis. See PYROSIS.

WATER-CANKER.—A synonym for cancrum oris. See CANCRUM ORIS.

WATER-GAS, Poisoning by.—See CARBONIC OXIDE, Poisoning by.

WATER-ON-THE-BRAIN.—A popular name for hydrocephalus. See MENINGES, CEREBRAL, Inflammation of, Tubercular; and HYDROCEPHALUS, CHRONIC.

WATERS, MINERAL.—See MINERAL WATERS.

WAXY DISEASE.—One of the synonyms for amyloid disease. See AMYLOID DISEASE.

WEAL, WALE, or WHEAL.—This is an old-English word signifying the mark of a stripe, that is, the prominent pale ridge caused by the stroke of a lash upon the skin. In Medicine the term is applied especially to the prominent risings of a lengthened figure which are met with in urticaria. See URTICARIA.

WEBER'S SYMPTOM.—See CRURA CEREBRI, Lesions of, p. 357.

WEIL'S DISEASE.—This name is sometimes applied to a febrile condition characterised by jaundice, enlargement of liver and spleen, and temporary nephritis, lasting about a fortnight, and generally ending in recovery. The disease usually occurs in epidemics, and is especially prone to attack butchers. The onset is sudden; and the jaundice, which is of the toxæmic type, occurs about the second day at the height of the fever (103° F.—104° F.). There is marked headache, prostration, and muscular pain. Relapses are infrequent. The cause of the condition is unknown, and possibly varies in different epidemics (Hunter).

H. MONTAGUE MURRAY.

WEILBACH, in Nassau, Germany.—Sulphur-waters. See MINERAL WATERS.

WEIR-MITCHELL TREATMENT.—See pp. 714 and 1073.

WEN.—A popular term for a tumour of the integument, without reference to its pathological structure. Wens are commonly fleshy or encysted; in the latter case proceeding from distension of the sac or excretory duct of a cutaneous gland, more especially a sebaceous gland.

WERLHOF'S PURPURA.—See PURPURA.

WERNICKE'S REACTION.—See p. 1065.

WESTON-SUPER-MARE, in Somerset.—A fresh, open, marine climate. Exposure W. Prevailing winds S.W. Mean temperature for the year 49° 8' F. See CLIMATE, Treatment of Disease by.

WESTPHAL'S SIGN.—The disappearance of the knee-jerk in Tabes Dorsalis. See p. 1661.

WET-PACK.—See HYDROTHERAPEUTICS.

WEYMOUTH, in Dorset.—A bright, open, marine climate. Exposure E. Mean temperature for the year 50° 1' F. See CLIMATE, Treatment of Disease by.

WHEEZING.—A peculiar sound, of a dry piping or whistling character, which may be heard in connection with the respiratory organs during the act of breathing, caused by certain forms of obstruction

to the passage of air. See ASTHMA, SPASMODIC; BRONCHI, Diseases of; and PHYSICAL EXAMINATION.

WHIP-WORM.—This term is not infrequently applied to the small human nematode that is better known to the profession as the *Trichocephalus dispar*. See ENTOZOA.

WHISPERING PECTORILOQUY.—A form of pectoriloquy in which the whispering voice is distinctly heard. See PECTORILOQUY; and PHYSICAL EXAMINATION.

WHITE GUM.—A popular name for the white form of strophulus, *S. albidus*. See STROPHULUS.

WHITE LEG.—A common name for phlegmasia dolens. See PHLEGMASIA DOLENS.

WHITE SWELLING.—A common name for tubercular disease of a joint. See JOINTS, Diseases of.

WHITLOW.—SYNON.: Paronychia; Panarium; Fr. *Panaris*; *Tourniole*; Ger. *Paronychia*.

DEFINITION.—Whitlow is a term somewhat loosely applied to any acute inflammation of the finger or thumb which tends rapidly to terminate in suppuration, and is not limited to the matrix of the nail, in which case it would be called *onychia*.

Whitlow may be divided into four chief varieties, but these often merge one into another: (1) Subepithelial Whitlow; (2) Subcutaneous Whitlow; (3) Thecal Abscess; and (4) Periosteal Whitlow.

Whitlow most commonly results from infection with the *Staphylococcus pyogenes aureus*, but less commonly in the more severe forms the *Streptococcus pyogenes* is present.

1. Subepithelial Whitlow.—SYNON.: Paronychia Ungualis.—In this variety the skin only is affected, and frequently at the side of the nail. It commences usually as the result of some slight injury, such as a bruise or puncture, or from the inoculation of septic or other irritating matter. The first signs are heat, tenderness, and itching in the inflamed part. On the third or fourth day pus forms, raising the epithelium from the cutis vera. As the pus cannot point through the dead cuticle, it remains pent up, and the tension so caused increases the pain, and if unrelieved leads to further ulceration of the true skin, the pus then finding its way into the cellular tissue beneath. The whitlow then merges into the second variety. It may also spread to the matrix of the nail, and so become complicated with onychia. If relieved early, by cutting away the cuticle which has been raised by the pus, it seldom leads to any unpleasant consequences.

TREATMENT.—The treatment consists in bathing the finger frequently in hot water, painting the part with glycerine of belladonna, and applying hot fomentations. If the presence of a minute drop of pus beneath the cuticle is detected by the appearance of a yellow point, the cuticle should be shaved off with a sharp scalpel; if the suppuration is more extensive the raised cuticle may be completely removed with scissors. A boracic-acid fomentation should then be applied. Incisions are not necessary.

A form of superficial whitlow is sometimes seen occurring without any apparent cause, and attacking one finger after another. The fluid beneath the cuticle is not always purulent—sometimes being

merely albuminous, and mixed with flakes of lymph. It never leads to any deep suppuration. It is most common in children and females, and is associated with general debility. It is described by the French under the name of *tournoie* or *panaris phlycténoïde*. It is a troublesome affection, and difficult to get rid of. Tonics should be administered, and sometimes arsenic is of use.

2. Subcutaneous Whitlow.—**SYNON.** : Paronychia Cellulosa.—The inflammation in this variety commences in the cellular tissue of the pulp of the ungual phalanx. It arises almost invariably as the result of some slight puncture or other injury, or as the consequence of neglecting the variety just described. The tip of the finger is swollen, tense, and excessively tender. There is severe burning, throbbing pain; and possibly red lines are observed spreading from the inflamed part in the course of the lymphatics. There is more or less fever, with general constitutional disturbance. Pus forms by the third or fourth day. It tends to point through the skin, but the thick cuticle usually resists its pressure for some time, and if this be not cut away early the gravest results may ensue. The pus may find its way into the sheath of the flexor tendons; or the bone may be exposed, and necrosis may follow, the disease then merging into the two following varieties. If an incision be made about the fourth or fifth day, a large slough will usually be found beneath the skin.

TREATMENT.—A free incision must be made longitudinally into the pulp of the finger as soon as the condition is recognised, and by this means all complications will be averted. If the pus have already found its way through the cutis, and be pent up beneath the epidermis, it is often sufficient merely to cut away the loosened cuticle with scissors. In other respects the treatment is the same as for the first variety.

3. Thecal Abscess.—**SYNON.** : Paronychia Tendinosa.—This is often secondary to the other varieties; it frequently begins, however, as a primary inflammation of the sheath of the flexor tendons, and may follow a penetrating wound. The whole finger swells, and becomes tense and red. The pain is most severe, usually shooting up the hand and arm. Pus forms early, and, if not evacuated by incision, exposes the bones, destroys the tendons, burrows into the joints, and rapidly destroys the finger. If affecting the thumb or little finger, the sheaths of which are continuous with the common sheath at the wrist, the suppuration rapidly extends to the palm of the hand, and to the forearm above the annular ligament. The wrist-joint may then be opened, and destruction of the whole hand result. The constitutional disturbance and fever are usually very marked. The affected finger or hand seldom recovers perfect utility; and death is not uncommon, either from exhaustion or from some secondary complication.

TREATMENT.—The early treatment should be the same as for the other varieties of whitlow. Free and early incisions are required. Stimulants, good diet, and tonics are always necessary.

4. Periosteal Whitlow.—**SYNON.** : Paronychia Osseosa.—This is a comparatively rare variety of whitlow, arising sometimes from injury, sometimes without apparent cause. It is an acute inflammation of the periosteum of the ungual phalanx. It is characterised by redness and swelling, with most intense aching and tensive pain, and

acute tenderness. If an incision be made as soon as pus is recognised, the bone will be found to be already bare and necrosed. This condition can only be averted by cutting down to the bone before suppuration has occurred, but this is rarely possible.

TREATMENT.—This is the same as in the other varieties, namely, hot applications and free incisions. The necrosed phalanx must be removed as soon as it is loose. If more than one phalanx be affected, amputation may be necessary.

One or two points common to the treatment of all varieties of whitlow require further notice. All incisions should be made as far as possible in the middle line, so as to avoid wounding the digital arteries. The sheath of the flexor tendons should on no account be opened, unless there is pus within it. In all doubtful cases an anæsthetic should be administered, the limb made bloodless by Esmarch's method, and the incision carried carefully towards the flexor tendons. In this way the exact situation of the pus can be ascertained with certainty, and an unnecessary wound of the sheath of the tendon avoided. Incisions are frequently made into the pulp of the ungual phalanx of the finger when the pus is really on the dorsum. This arises from the sense of fluctuation yielded by a swollen finger covered by somewhat thick cuticle. In all cases the cuticle should be cut away as soon as it is loose. The best application in all cases is boracic lint, three or four layers thick, wrung out in hot water, and covered by oiled silk and cotton wool. This is perfectly clean, and in every way as efficient as a poultice. Soaking the hand frequently for an hour at a time in an arm-bath usually gives great relief in the more severe forms.

MARCUS BECK.

RAYMOND JOHNSON.

WHOOPIING-COUGH.—**SYNON.** : Kin-cough; Hooping-Cough; Pertussis; Fr. *Coqueluche*; Ger. *Keuchhusten*.

DEFINITION.—An infectious disease, characterised by fever, catarrh of the respiratory passages, and a peculiar spasmodic cough, consisting of a number of quick, short, forcible expirations, followed by a long shrill inspiration, or 'whoop,' and finally ending in the expulsion of viscid mucus, or in vomiting.

ÆTIOLOGY.—The actual cause of the disease is at present unknown. That it must be some living organism is now universally admitted. Two independent observers have found a ciliated amœba, to the growth of which they attribute the disease. Other and more numerous investigators have isolated minute bacteria, and although the form and character of the organism are still in dispute, there is a very general belief that some bacterium will eventually prove to be the cause of the disease. A short bacterium first described by Czaplowski and Hensel is in most favour. This organism stains imperfectly in the centre, and has thus been described by some authors as a diplococcus. It is non-motile, does not form spores, and is easily destroyed by heat. It does not give any serum-reaction, and is pathogenic to mice.

Infection is most readily conveyed during the first week of the disease. It gradually declines as the disease progresses, but persists for nearly a month after the commencement of the whoop. It is usual to consider a patient free from infection five weeks after the first whoop occurred: after this there may

be a return of cough or spasm without fresh danger of infection. There is reason to suppose that the disease is conveyed by the expelled mucus, either directly from patient to patient, or indirectly by means of clothes or utensils; if care be taken, the spread of infection can often be prevented. The contagion clings to the clothing of convalescents for a long time.

Age.—The greatest number of cases occur in children under eight years of age. It is one of the three diseases which cause the greatest mortality among infants, and, like acute bronchitis and summer diarrhoea, is most fatal to the youngest. Whooping-cough prevails so extensively in early childhood, that it is rare to find an adult who has not suffered from it. Those who escape infection in childhood generally escape it altogether. When the disease affects adults it runs its full course with the same symptoms, and the same instinctive dread of recurrent spasm, as in infancy, but without any practical risk of complications.

Sex.—The severity of the attacks is greater in girls than in boys; and in the former the mortality is also slightly higher.

Race and climate make little or no difference in the liability to whooping-cough.

Season has an influence on its epidemic extension; this in Great Britain is always in the spring (see p. 1190). Cold weather indirectly adds to the intensity of the disease and retards the convalescence.

Other influences.—Overcrowding, bad ventilation, and the confinement of the sick in close apartments increase the severity of the disease, as well as the risk of infection. A recent attack of measles also increases the liability to infection; and the two diseases are occasionally concurrent, in which case the characteristic cough of the one generally escapes recognition until the rash of the other has disappeared. Excitement and indigestible food seem to induce and exaggerate the attacks of coughing.

The *period of incubation* is well marked in all cases, and extends from two days, as the shortest limit, to twelve days as the longest; the average duration of this period is eight days. As the disease cannot be diagnosed during the first week, it is necessary to disinfect 'contacts' (see p. 726), and isolate them for three weeks before their freedom from the disease can be certified.

MORBID ANATOMY.—In fatal cases catarrh of the upper respiratory passages is found. In some cases this is most marked in the posterior nares and naso-pharynx; in others, in the larynx, trachea or bronchi. The bronchial glands are generally enlarged. Recorded observations made on patients during life confirm the view that these changes occur during the early stage of the disease, even in mild attacks, but there is no uniformity concerning the exact locality of the commencement, which probably varies in different cases. Fatal cases generally present evidence of pulmonary collapse, pneumonia, acute emphysema or other complications.

PATHOLOGY.—The infective nature of the disease is indisputable. The observed facts concerning the spread of the disease, the early catarrhal stage, the accompanying enlargement of the bronchial glands, the subsequent spasmodic attacks, and the curious nervous condition so often observed, together suggest infection by way of the respiratory passages and the gradual introduction into the blood of a toxin or toxins which act principally on the nerve-centres; but until the cause of the disease has been definitely

determined, and its characters studied, the pathology must remain speculative.

SYMPTOMS.—The invasion of whooping-cough is often insidious and, in the absence of a prevailing epidemic, suspicion of illness may not be aroused until the child is noticed to be suffering from a troublesome cough, especially at night. Not infrequently, however, the onset is quite definite. The patient, if very young, is feverish (100° F. to 102° F.) and restless at night, and suffers from cough and sneezing; if older, is apathetic, complaining of headache and anorexia. Next morning the symptoms may be less marked, but by evening they are worse than on the previous day.

During the following ten days (*catarrhal stage*) the symptoms are very varied. In some cases the temperature remains raised, a hard, dry cough, always worse at night, persists, and a few rhonchi can be heard in the chest. In others, the temperature intermits or never rises above 100° F., and except for the harassing cough, which interferes with sleep, no illness may be suspected. In others, again, severe bronchitis sets in from the outset. As a rule, however, the general malaise and fever are not explicable by any discoverable physical signs. Marked lymphocytosis, as well as some ordinary leucocytosis, may first be found in this stage.

Towards the end of the second week the catarrhal stage passes into the *spasmodic*. The cough gradually becomes longer, louder and more exhausting, especially at night, and the quiet intervals between what may now be called the 'attacks' are more definite and of longer duration. In other words, the frequent cough of bronchial catarrh becomes grouped into less frequent spasmodic attacks, which often first suggest the correct diagnosis (see Diagnosis, p. 1875). Two or three days after this tendency is observed the cough assumes its distinctive character. Just before the attack begins, in a case of average severity, the child's face reddens, as if trying to suppress it, till the cough bursts out in a series of short, quick, forcible expiratory efforts; then the breath is drawn in with a shrill whistling sound or whoop, again followed by a similar series of rapidly recurring explosive expiratory efforts, succeeded by another whoop: after a short pause comes a less severe and shorter fit and then another, till a quantity of whitish viscid mucus is expelled through the nose or mouth, and some swallowed; or the child vomits at the same time, ejecting the contents of the stomach. In a case of average severity the patient will whoop three or four times in each attack, and the attacks will recur every two or three hours during the day and more frequently during the night.

Older children often describe a tickling in the throat before the cough, show dread of its approach, and prepare for it by steadying themselves, or by clinging to others for support; they appear to recover completely and show no sign of illness during the intervals. Not so with younger children; before the attack the pulse quickens, the breathing is short and insufficient, *râles* are heard, spasm closes the glottis, the air is forced out in sudden jerks, and then enters with the loud long-drawn whoop; this is repeated till the face becomes livid and swollen, and the child exhausted or semi-conscious. Frequent attacks of this kind keep the face puffy; they force the tongue over the lower teeth, and thus especially in infants who have only cut the

two lower central incisors, small ulcers may be produced on the frænum linguae; the eyes remain red and watery, and hæmorrhages occur beneath the conjunctivæ. In a severe paroxysm blood may start from the nose and mouth, or be seen in the matters vomited; while both urine and fæces may be passed involuntarily. In some cases diarrhœa and vomiting are serious symptoms. While the cough forces air from the lung, the percussion-note is dulled but becomes resonant again after the sibilant inspiration is heard. The heart's action is impeded, owing to the raised pressure within its cavities resulting from the cough, or from irritation of the vagus; neither endocardial nor pericardial lesion is met with in whooping-cough, and nothing is found wrong with the heart after the fit is over.

Examination of the chest in uncomplicated cases generally fails to reveal any physical signs during the intervals. In some cases, however, temporary hyper-resonance, due to the over-distension of the air-cells and bronchioles, may be made out and a few bronchitic rhonchi heard.

In the absence of complications the *third stage* of subsiding spasm and loose expectoration, with returning health and strength, may be reached in from four to six weeks. The mildest cases may seem to be over in a month, but any little want of care will intensify the symptoms, and relapses are common until six weeks are accomplished, even in cases where the second stage has not been prolonged beyond four weeks. The disease often lasts two months, and may be followed by a tedious convalescence. At this stage patients seem particularly liable to tuberculosis.

COMPLICATIONS.—In addition to the phenomena already described as occasional accompaniments of whooping-cough, patients suffering from this disease are more than usually liable to mechanical results of coughing, such as hernia, as well as to acute inflammation of many of the principal viscera, such as acute nephritis and acute lobar pneumonia. There are, moreover, several conditions which are especially associated with whooping-cough, and which gravely affect its prognosis.

(1) *Hæmorrhage.*—Hæmorrhage under the conjunctiva or from the nose or gums is exceedingly common, though of comparatively slight importance. Of rare occurrence and of more serious import is hæmorrhage from the middle ear, due to rupture of the tympanum, hæmorrhage from the lungs, and hæmorrhage within the cranium (*see p. 987*). Intracranial hæmorrhage is usually meningeal, and may give rise to coma, to aphasia, or to paralysis of one of the cranial nerves.

(2) *Convulsions.*—General convulsions may occur, especially in very young children (*see p. 343*) (*a*) when the attacks are very frequent and severe; and (*b*) when extensive pulmonary collapse occurs. They are believed to depend in both cases upon vascular engorgement of the cortex and deficient aëration of the blood.

(3) *Pulmonary.*—Whooping-cough and measles are distinguished from all other acute infectious diseases by the extreme frequency of pulmonary complications. Among these, severe bronchitis, affecting the larger tubes, capillary bronchitis, collapse of large portions of a lung, and broncho-pneumonia are common. Acute lobar pneumonia and pleurisy are far less frequently met with. The signs and symptoms of these complications differ but little from those met with in the same diseases

occurring under other circumstances, but the onset is often insidious; hence there arises a necessity for regular observations of the temperature and rate of breathing, as well as for periodical examinations of the chest. Pulmonary complications may occur during any stage of the disease, and are most likely to arise on the one hand in neglected cases, and on the other in those who are compelled to breathe stale air. The occurrence of broncho-pneumonia often modifies, in a curious way, the course of the disease. The attacks become less defined and the characteristic whoop may disappear.

SEQUELÆ.—Bronchitis may persist long after whooping-cough has disappeared, and permanent dilatation of the bronchial tubes is not unknown. Asthma, interstitial pneumonia, and a peculiar form of irritability and restlessness, are also recognised sequelæ of this disease. As has already been said, children suffering from whooping-cough are especially liable to develop tuberculosis, and in cases exhausted by vomiting and inefficient nourishment, wasting, pustular skin-eruptions and obstinate intestinal catarrh are peculiarly prone to occur.

TERMINATIONS.—The large majority of cases of whooping-cough recover. Yet so frequent is the disease, that it is one of the principal causes of infantile mortality (*see p. 1028*). Of the fatal cases, most die of some pulmonary complication. A few succumb from acute cardiac failure, from spasm of the glottis, or from some other complication or sequela.

DIAGNOSIS.—The diagnosis of whooping-cough presents difficulties both in the catarrhal and in the spasmodic stage. In the former, a correct diagnosis is, in the absence of a prevailing epidemic, an exceptional achievement. A nocturnal cough with a temperature of 101° F. and no physical signs generally indicates the onset of whooping-cough, measles, or influenza. The muscular pain and brief duration of the attack will generally suffice to distinguish influenza. The conjunctival and nasal catarrh, followed on the fifth day by the rash, will ensure the recognition of measles. When both these diseases can be excluded, special attention must be paid to the grouping of the attacks of cough. Bronchitis can generally be distinguished by the more marked physical signs. It is always well to ascertain to what diseases a sick child has been exposed, and what infectious diseases it has already had. In the spasmodic stage laryngismus stridulus (*p. 850*) is occasionally mistaken for whooping-cough, but the spasm of the glottis and the croupy inspiration persist throughout the attack. Enlarged tonsils and post-nasal adenoids, or even a long uvula, especially when occurring together, not infrequently give rise to a cough closely resembling that of whooping-cough, but the sequence of events characteristic of the latter is absent, and the physical examination will probably suffice to remove the doubt. Enlargement of the bronchial glands from other causes may give rise to attacks superficially resembling those of whooping-cough; but the disease does not run the same course, and the attacks, as in the cases of enlarged tonsils, are generally less defined. *See p. 230.*

PROGNOSIS.—The prognosis in whooping-cough depends on the age of the patient, the previous health, the condition of nutrition at the time of onset, the time of year (*Fig. 3, p. 1191*), the general severity of the attack, and the presence of complications.

Young children are least likely to make the

favourable recovery generally expected in uncomplicated whooping-cough; but with good sanitary surroundings and individual care the youngest may escape, unless debilitated by previous illness, such as rickets or measles. The risks diminish with each year of childhood; they are less in the summer than in the winter, and in boys than in girls. High fever during invasion is a frequent prelude to severe complications in the subsequent stages. Convulsions in any stage are of the worst significance; somnolence or a listless condition between the attacks, with persistent high temperature, are also bad signs. The danger in the second stage is in proportion to the severity of the disease. This may be estimated by noting the number of attacks in the twenty-four hours, their intensity and their duration; in bad attacks the expulsive efforts are more rapid, more boisterous, and more numerous, and the whoops are very shrill. In mild cases there is less spasm and the whoop occurs but once or twice, or is sometimes not recognisable at all. The duration of an attack may be from a few seconds to some minutes. Young infants may die from laryngeal spasm, but the danger of death during an attack, however bad, is very small. The number of attacks may be raised from twelve to twenty in the twenty-four hours, without danger, if the intervals are complete enough for the child to recover and take food during the day and to get sleep by night. As many as 60 to 80, or even 140, attacks have been counted. When they are so incessant as to interfere with both food and sleep the exhaustion is extreme, so that it is generally true that when the number of attacks exceeds 60 in the twenty-four hours a fatal result may be anticipated.

Capillary bronchitis and broncho-pneumonia are the complications responsible for most deaths, especially when the thorax is rickety. Œdema of the glottis or of the lung, and acute cardiac failure, are less frequent but more deadly in their effects. Chronic pneumonia may lead to bronchiectasis or culminate in tuberculosis, which generally runs a rapid course. Emphysema and pulmonary collapse tend to disappear.

TREATMENT.—General. There is no specific remedy for whooping-cough—no drug that can check its onset or stop its progress. Rest and warmth, with individual care, the utmost attention to a sufficiency of pure air, and all possible aids to the resisting powers of the patient, are requisite throughout the illness.

Two rooms, intercommunicating if possible, should be set aside—one for use by day, the other by night. The temperature of the room should be maintained at 63° to 65° F.; but thorough ventilation must be secured to ensure a plentiful supply of perfectly fresh air, free from dust and without cold draughts. No gas or gas-stove must be burnt in the room. The patient should be kept in bed until the temperature is normal, and confined to the house until the spasmodic stage is over. The cold bath in the morning should be omitted until convalescence is established. The diet should be light and nutritious, consisting largely of milk; and all food difficult of digestion should be avoided. Broth should be made with vegetables and without condiments; stewed fruit, orange-juice, and lemonade are useful. It is generally advisable to give food in small quantities at short intervals; and when vomiting is frequent this procedure is absolutely essential. Each child requires a good deal of

ready personal assistance. Some one should always be near to allay fright when the cough begins, and to steady the patient and support the head until the fit is over.

Medicinal. (1) *Inhalation.*—The value of antiseptic inhalations in whooping-cough is in dispute. To young children they are frequently a source of terror and so far inadmissible. Moreover, as has often been pointed out, medicated air generally implies in practice rebreathed air, which again is unquestionably harmful. If these difficulties could be overcome there is some reason to suppose that air impregnated with carbolic acid or other allied substances would be useful, as it has a sedative effect during the spasmodic stage. Great risks, without corresponding benefit, have been run by taking children to gas-works during the course of the complaint. Nasal insufflation of salicylic acid, iodoform, quinine, and many other drugs have been recommended, especially by those who consider that the nasal mucous membrane is the primary seat of the disorder. Others have recommended that the pharynx be painted with a solution of resorcin (1 per cent.) or of similar antiseptic, to which a small quantity of cocaine has been added. Baroux strongly recommends the use of peroxide of hydrogen as an aerial antiseptic. He recommends that two large pieces of old linen be sprinkled with two to three ounces of peroxide and hung on a cord stretched across the room; they should be renewed every four hours.

(2) *Internal remedies.*—In very many cases of whooping-cough no medicinal treatment is needed. During the *catarrhal stage* simple salines may be beneficial. To relieve the cough an occasional sip of water or some simple linctus containing a few drops of ipecacuanha or of oxymel of squill is often sufficient. In many cases bromide of potassium or ammonium must be added to an expectorant mixture in order to produce any relief. Neither opium, chloral hydrate nor belladonna is to be used until the first stage is over and secretion free. A poultice of linseed, or of mustard and linseed, is a valuable adjuvant (*see* POULTICE), especially when a few fine râles are heard at the bases. Many authorities advocate the addition of some compound tincture of camphor when the secretion is free. If the child be obviously ill with more than the usual amount of fever, quinine should be given—1 gr. *per diem* suspended in Almond-mixture for each year of age, and continued for two or three days, answers best, but sometimes larger and fewer doses seem more efficacious. If any difficulty is experienced in the administration of the quinine it should be given as an enema.

Relief of spasm is the main object of treatment in the second or *spasmodic stage* of whooping-cough, the efficacy of the means employed being measured by the diminution in the severity and number of the daily attacks. By this test belladonna proves to be the most useful remedy, but it must be given in large and continued doses. With a child of three years old one-sixth to one-fourth of a grain of the extract, or 10 to 15 minims of the tincture, may be given three to six times a day before the physiological effects indicate a diminution of the dose. Atropine ($\frac{1}{100}$ to $\frac{1}{80}$ gr.) is preferred by many; but since the official standardisation of the preparations of belladonna the selection of any particular preparation is a matter of less importance. A combination of bromides, especially that of

ammonium, with belladonna, has a well-deserved reputation. Bromoform, in an emulsion, is a favourite remedy at the present time; it may be given in doses of $\frac{1}{2}$ m to 3 m cautiously, according to the age of the child, three or more times a day. Minute doses of opium are sometimes useful in allaying vomiting, but must only be given when the secretion is free; only a few doses are permissible. Ergot, phenazone, and butyl-chloral hydrate are more or less accredited remedies, especially when the others have failed, but it must be remembered that most of the vaunted remedies for the cure of whooping-cough owe their repute to having been administered in the fifth or sixth week of the illness, when the disease is nearly over. All the more potent counter-irritants, such as croton oil and blisters, are to be avoided. In rare cases artificial respiration has been successful in resuscitating a child apparently dead of asphyxia.

In the third or *declining stage* of whooping-cough tonics and astringents are of distinct benefit. Great discretion is required in deciding when children should be allowed to go out of doors or seek change of air. Too much emphasis cannot be laid upon the rule that no child should be sent in public conveyances or to lodgings so long as there is the least risk of infection. Moreover, the end of the infectious period also corresponds with the time when the patient is most likely to receive benefit without any corresponding risk. Unquestionably change of air has a remarkable effect in restoring appetite and in removing the remaining tendency to spasm (*see* p. 1471). The treatment of the various complications is described in the articles dealing with these conditions.

H. MONTAGUE MURRAY.

WIDAL'S TEST.—*See* SERUM-REACTIONS.

WIESBADEN, in Nassau, Germany.—Thermal salt waters. *See* MINERAL WATERS.

WIGHT, Isle of.—*See* SANDOWN; SHANKLIN; UNDERCLIFF; VENTNOR; and CLIMATE, Treatment of Disease by.

WILDBAD, in Würtemberg, Germany.—Simple thermal waters. *See* MINERAL WATERS.

WILDUNGEN, in Waldeck, Germany.—Earthy waters. *See* MINERAL WATERS.

WILL, Capacity for executing.—*See* CIVIL INCAPACITY.

WINDPIPE, Diseases of.—*See* LARYNX, Diseases of; and TRACHEA, Diseases of.

WINKING, Involuntary.—*See* FACIAL SPASM.

WINTER-COUGH.—This expression is associated with those cases in which a patient is subject to more or less cough during the winter season, being free, or almost free, during the warmer portion of the year. It may come on at any period in the course of the winter, and is generally referable to some obvious cause, which produces 'a cold.' The attacks tend as a rule to become more aggravated and difficult to cure, as well as more easily excited, as time progresses.

Cases of winter-cough belong chiefly to the following classes: 1. Not uncommonly it is merely due to a slight catarrh, affecting the throat and

main air-passages. 2. Most frequently the cough depends on bronchial catarrh or bronchitis in various degrees, usually associated with more or less emphysema. 3. Winter-cough may characterise some chronic phthisical cases, this symptom subsiding during the warmer season. 4. There are certain forms of cardiac disease of which winter-cough may be a prominent phenomenon.

TREATMENT.—The treatment of winter-cough must depend on the nature of the cause which gives rise to it, and will be found discussed in the special articles descriptive of the several conditions. *See* COUGH.

FREDERICK T. ROBERTS.

WOMB, Diseases of.—*See* UTERUS, Diseases of.

WOODHALL, in Lincolnshire.—Common salt waters, containing iodine and bromine. *See* MINERAL WATERS.

WOOLSORTER'S DISEASE.—A form of anthrax. *See* PUSTULE, MALIGNANT.

WORD-BLINDNESS.—*See* APHASIA.

WORD-DEAFNESS.—*See* APHASIA.

WORMS.—*See* ENTOZOA.

WOUNDS, Healing of.—A living animal tissue when wounded has an inherent tendency to repair, but what this tendency depends upon is as yet unexplained. The bond of union need not be of the same nature as the tissue wounded. It is composed usually of white fibrous tissue, and when formed is known as a 'cicatrix.' So far as the naked-eye appearances are concerned, wounds are generally described as healing by one or other of the following five methods: (1) by 'first intention;' (2) by 'immediate union;' (3) by 'second intention' or 'granulation;' (4) by 'secondary adhesion' or union of two granulating surfaces; and (5) under a scab, or what is virtually healing under aseptic conditions. The last of these does not require special description, as the scab is simply Nature's dressing; the process of healing under such circumstances does not differ from what occurs, say, in healing by the 'first intention.' Although, on mere naked-eye examination, the above methods of repair are evident enough, yet, when the pathology of the process is inquired into it will be found that there is in reality only one method by which wounds heal, namely, by growth of the new reparative tissue from tissue of a like kind present in the wounded part. The superficial differences observed in the closing of wounds are to be regarded as accidental rather than essential, as epiphenomena, or complications, and frequently as impediments to the even onward progress of growth which constitutes the ruling feature.

1. *Healing by the 'first intention.'*—If the flaps of a wound, say, after amputation, be examined in from twelve to eighteen hours from the time of infliction of the injury, they will be found to present a glossy, moist appearance, due to the escape of lymph from the divided lymphatics—the lymph-spaces, lymph-radicles, and larger lymph-vessels, which permeate all parts of the body. If it be again examined after a lapse of about forty-eight hours, the severed tissues will be found to have lost this appearance; the quantity of liquid exuding from the wound is less than before; and the

exposed muscles and other elements of the flaps are now coated with a delicate greyish-coloured film, which can be removed by scraping the surface. Examined microscopically, this filmy deposit is found to consist of lymph-corpuscles—the corpuscles which have exuded with the lymph, and which are now entangled in the free extremities of the divided tissues. They are embedded in fibrin, which has taken the form either of a granular precipitate or a network. Supposing the two sides of the wound to have been placed in accurate contact, the two lymph-coated surfaces, together with any blood-clot which may have been upon them, adhere and establish a temporary bond of union. In from four to five days after the infliction of the wound, a layer of connective-tissue elements or fibroblasts makes its appearance beneath the lymph on each side, and this, as it grows inwards, supplants and causes absorption of the lymph and blood-clot lying in the gap, which supply, as above said, the temporary bond of union; the two layers meet in the middle line and their cells intertwine. The cells composing these two layers are at first round, many of them twice the size of a leucocyte, and are provided with either a single large nucleus or, it may be, two of these. As organisation proceeds they become spindle-shaped, and in from three to four weeks are converted into bundles of white fibrous tissue. This constitutes the permanent cicatrix. Yellow elastic tissue enters little, if at all, into the construction of the medium of repair. The source of the fibroblasts which enter into the organising layers is the white fibrous tissue near the surface of the flaps, more especially if this be of the areolar variety. The tissues at a short distance from the surface are practically unaffected, and the cicatrix, when healing is completed, is usually so delicate that its line can be followed throughout the part with difficulty, even with aided vision. As the process of repair is proceeding, loops of capillary blood-vessels make their way into the embryonic cicatrix; they are never abundant or more than what is necessary for the maintenance of the part. Hence the blanched appearance presented by a wound when healing typically by the first intention. The gap having been filled with young connective-tissue, the divided epidermis throws out a few new cells; these meet in the middle of the wound, and, coalescing, re-establish the protective covering. The resulting cicatrix is always more or less depressed.

Should the wound, however, become septic, that is to say, inoculated with the organisms of suppuration, chiefly the *Staphylococcus pyogenes aureus* and the *Streptococcus pyogenes*, the reparative ingrowth of the organising tissue is prevented; the blood-vessels are stimulated and become engorged, and excess of blood-products is poured out from them. These accumulate in the wound and fall into a state of fatty degeneration, and an abscess results. The pus which constitutes the abscess must be discharged before healing will take place. Generally the wound will gape and commence to granulate, healing being effected thereafter by the 'second intention.' Or, if actual granulation be not forthcoming, the wound may heal gradually from its depths, the quantity of cicatrix formed in either case being vastly greater than if it had closed by the first intention.

2. *Healing by immediate union.*—This is nothing more than healing by the 'first intention' under the

very best auspices. An incision is made into a part, pressure is applied immediately, all blood is squeezed out of the wound, and the amount of lymph which escapes is limited by the pressure of the opposed surfaces. Unduly great distension of blood-vessels is prevented by the same agency, and the wound remains aseptic. Under these circumstances the severed tissues unite with the least possible expenditure of material.

3. *Healing by the 'second intention.'*—In the centre of a wound healing by this method is the granulating surface. The granulations are small—mere points of a rosy red colour—and the discharge from them is slight. Each granulation consists of a tuft of capillary blood-vessels, around which there is an indefinite quantity of loose areolar fibrous tissue. So delicate and open-woven is the latter that it may present almost a myxomatous appearance. The meshes of this stroma are filled with more or less plasmatic liquid exuded from the capillary loops, and with the granulation-cells. These granulation-cells are of two kinds, namely, leucocytes and fibroblasts, the former being the more abundant. Some of the fibroblasts may approach giant-cells in size; or actual giant-cells, if the wound be tubercular, may be contained in the meshes of the stroma. The discharge from the granulating surface consists of the above liquid pressed out from the vessels, and the granulation-cells—in chief part those which are leucocytes. Accumulating as this liquid and the leucocytes do in the meshes of the stroma, they are driven out in the direction of least resistance, namely, towards the surface, and are thrown off from it. The more or less purulent character of the discharge depends upon the number of cells contained within it, and these diminish in number as the wound assumes healing characters. The amount of discharge depends upon the degree of distension of the capillary loops. The actual granulating surface is shallow, usually not more than from a sixteenth to an eighth of an inch, but beneath it a layer of tissue of primary importance in the healing of the wound is met with. It is composed of the same elements as those which form the organising layers on the sides of the wound in healing by the 'first intention,' namely, fibroblasts of round and spindle shape. Through this layer of young connective tissue the stems of the capillary loops pass to reach the granulating surface proper. When healing of the wound takes place, the granulations atrophy, any fibroblasts contained in them become fibrous, and, along with those in this deep cicatrising layer, constitute the young fibrous-tissue basis over which the epithelium spreads to complete the repair.

Round the margin is the so-called 'blue-pink pellicle'—the layer of young epidermis. Its edge is very loosely attached to the underlying cicatricial layer, so that the blade of a knife can be inserted beneath it; but later on the new epithelium becomes inseparable from the parts beneath. It is composed of young epidermic cells like those of the rete Malpighii. As the granulations atrophy at the margin of the wound, this pellicle of young epidermis spreads at first round them and finally over them. As it does so, the granulation-vessels disappear in great part; further exudation of the granulation-leucocytes is prevented; and the fibroblasts formerly entangled within the granulations, together with those lying underneath, become transformed into fibres. The part which has

healed is blanched and hard. The epithelium covering it has assumed the characters of epidermis with a rete Malpighii and horny layer, and underneath this is the coarse fibrous tissue of the cicatrix.

4. *Healing by secondary adhesion.*—If two granulating and healing surfaces are brought into accurate contact, and if equable pressure be applied, they tend to unite, especially if they are free from pyogenic organisms. The mechanism of the process is that the mutual pressure of the closely applied surfaces causes atrophy of the granulation-loops, and allows the deep cicatricial layers to meet and coalesce.

Comparison of the above methods of healing.—There is no essential difference, for although in healing by the 'second intention' the wounded surface becomes covered by granulation-loops, still these are to be regarded as an accident of the situation—as due partly to relaxation of the natural restraint exerted upon the capillaries by the skin and other elastic tissues, partly to the stimulus exerted upon the free surface by the presence of the organisms of suppuration. If an exposed wound be kept free from sepsis, and if pressure be carefully applied to its surface, it heals without granulation, and more quickly than when granulation has ensued. The granulations on a wound healing by the 'second intention' must be removed before the wound will heal. By their atrophy the true healing layer, composed of fibroblasts, and lying beneath them, comes to the surface, and the epidermis runs over it. In healing by the 'first intention' the only real difference is that this layer is double instead of single, and the same holds good of healing by secondary adhesion. The exuberant vascularity of a granulating surface is the chief means of retarding its healing; the granulations must be got rid of either by natural atrophy or by the application of pressure, &c., before the wound will close. The basis of the whole procedure, as before said, is the growth of new connective tissue from that already in the wounded part, of new epidermis from that already at each side, and of new blood-vessels from those present on the cut or otherwise wounded surfaces.

Wounds of particular tissues.—The less vascular the part wounded, the more readily it heals by first intention. An incised wound of the cornea heals quicker than any other; that of the linea alba probably comes next. Wounds of muscle unite chiefly by fibrous tissue, although newly-developed muscular fibres may be found in the cicatrix. A cicatrix usually tends to contract, but that which follows upon the sloughing of a Peyer's patch in typhoid fever is an exception to this rule. The cicatrix of the intestine resulting from dysentery or tuberculosis has, however, a notorious tendency to contract.

New-formation of blood-vessels.—The blood-vessels newly developed in a healing part are apparently derived from buds given off from those already present. From the endothelium of a capillary a nucleated protoplasmic bud or vasoblast is shot outwards. Similar buds are pushed out from the walls of neighbouring capillaries, and these anastomose by their tapering extremities. They are solid at first, but become hollowed out, and into the channels thus formed blood-corpuscles find their way.

Healing of blood-vessels.—When the end of a divided artery is ligatured with silk, and when the wound becomes septic, the channel of the artery fills

with a firm clot or thrombus, usually as high as the first large branch above the point of ligature. Blood-capillaries grow into the clot from the vasa vasorum and ramify in all directions. They convey with them the elements of organisation, namely, fibroblasts derived from the adjacent wall of the vessel. At the same time the tunica intima thickens and encroaches on the channel of the vessel. As the living tissue from these two sources makes its way into the blood-clot, it causes absorption of the latter, just as would happen in a subcutaneous effusion of blood. As the clot is absorbed, its interstices are occupied by young cicatrix derived from these two sources, which by contracting closes the aperture at the end of the vessel and for some distance above. The clot acts simply as the mould or framework in which the future cicatrix is cast. When the artery is ligatured with catgut or aseptic silk, and when the wound remains aseptic until healing is completed, there may not be found any clot in the stump of the ligatured vessel, or this may be simply filiform in character. The artery, nevertheless, closes quicker under such circumstances than under the former, and the closure is effected exclusively by a cicatricial thickening of the tunica intima. Within three weeks of the application of the ligature the catgut may have vanished entirely, its place being taken by a ring of fibrous tissue, and, at the same time, the channel of the vessel may be in great part obliterated. The thrombus, if such be deposited after the ligature is applied, hinders the closure of the channel, and must be absorbed before this can be effected.

Adhesions of serous surfaces.—Although adjacent surfaces of most of the serous membranes are in contact during life, yet they do not tend to adhere. Should the membrane become inflamed, however, there is a constant tendency, in the absence of suppuration, to coalescence taking place. From the blood-vessels of the inflamed membrane there exudes a quantity of liquid more or less plasmatic in character. It contains fibrinogen together with leucocytes and other corpuscular elements of the blood. These set free the fibrin-ferment, which, reacting upon the fibrinogen, precipitates it as fibrin. The fibrin assumes the character of a network in which are entangled the corpuscular elements. It lies upon the adjacent serous surfaces, while between them is the liquid now deprived of its fibrin-forming constituents. Comparatively little of this liquid may be present, so that the inflammation assumes a dry character, and the two fibrin-coated surfaces may thus from the first be in contact. Any excess of liquid is in course of time absorbed, or may be drawn off artificially, after which the fibrinous layers from either side lie in apposition and adhere. Such adhesion is only temporary and is easily separable; the permanent connection is effected by the following means: From the fibrous basis of the serous membrane there is thrown off a dense mass of fibroblasts which accumulate in a layer beneath the fibrin, while the individual cells composing it push themselves in rows into the interstices of the fibrin. Wherever they spread the fibrin falls to pieces and is absorbed while the leucocytes become fatty and disappear, their place being taken by the fibroblasts derived as above described. Twigs of capillaries accompany the organising elements, also derived from the serous membrane. As the fibrin becomes absorbed, the two organising layers meet an

coalesce, their cells become converted into white fibrous tissue, and a permanent bond of union is established. The process of union is almost identical with that which occurs in healing by the 'first intention.'

Skin grafts.—See SKIN-GRAFTING, p. 1491.

Grafts of other tissues.—If living bone be chopped up into fragments, and these be placed in a sac of living tissue—by preference the periosteum—the fragments of bone continue to live and grow. The entire shaft of a bone like the humerus may thus be remoulded (Macewen). Pieces of the pancreas, thyroid gland, or conjunctiva may also be transplanted into a part to which they are foreign, and continue to live and grow. In all these grafting-experiments, the tissue to be grafted should be taken from the same species of animal as that to which the graft is to be applied.

Organisation of blood-clot.—Blood-clot has of itself no more power of organisation than any other form of precipitated albumen. Its leucocytes do not appear to have any power of developing into fibrous tissue, and its network of fibrin is quite inert. Being a porous body, however, it acts readily as the mould in which organisation may proceed. Hence, if blood be effused into the gap of a granulating wound and coagulate, its substance will soon become vascular, and its surface will bleed if pricked. The blood-vessels carry in their sheath the fibroblasts derived from neighbouring tissues, and these constitute the elements of organisation. The blood-clot is gradually absorbed by the living tissue penetrating into it, just as happens in the formation of an adhesion between two serous surfaces.

Organisation of inert porous bodies.—Any porous body, if the pores freely intercommunicate, will become filled with organising tissue under similar circumstances. Pieces of sponge, elder-pith, or calcined bone are all suited for the purpose. Sponge from its free porosity is particularly adapted for the experiment. It may be applied to a granulating surface in a thin slice or be embedded bodily in a deep tissue. In the former case the edge of the sponge must be a considerable distance within the margin of the epithelial pellicle, and, in the latter, the block of sponge must not cause pressure on adjacent parts, but simply replace a portion of tissue excised. The interstices of such porous bodies become vascularised, fibroblasts penetrate into them, they become firmly united to the part, and in course of time are absorbed.

D. J. HAMILTON.

WRIST-DROP.—A result of paralysis, affecting the extensor and supinator muscles of the wrist, and commonly met with in chronic alcoholism and chronic lead-poisoning. See ALCOHOLISM; LEAD, Poisoning by; and NEURITIS, Multiple.

WRITER'S CRAMP.—SYNON.: Scrivener's Palsy; Fr. *Crampes des Ecrivains*; Ger. *Schreibekrampf*.

This disease may be taken as the most common form and most typical representative of a class of diseases which Duchenne called 'functional impotences.' In them we find the patient complaining of inability to execute some complicated act, the power to perform which had taken him perhaps years (in the case of writing) to acquire. As a rule there is no other trouble, and, as far as the patient's observation goes, all muscular acts, however deli-

cate or however complicated, are accomplished without difficulty, with the exception of one, which unfortunately is usually that with which the patient earns his living. Not only have cases of 'writer's cramp' been described, but English and foreign physicians have furnished accounts of 'piano-player's cramp,' in which an inability to strike chords with correctness is present; of violinists who have lost the power of holding the violin and fingering with the left hand; of violoncello-players who have become powerless to 'make the nut' with the phalangeal joint of the left thumb; of tailors who can no longer use the needle; of dairymen who fail in milking; of bricklayers who cannot wield the trowel; of smiths who cannot use the hammer; of composers who cannot place the type; and lately we have had accounts of telegraphists who have become unable any longer to work at their calling. 'Writer's cramp,' however, is by far the most common of these diseases, and while the others mentioned must be regarded as the rarest of medical curiosities, the one which has been chosen to illustrate the class is tolerably often met with.

SYMPTOMS AND COURSE.—The symptoms and course in a typical case of 'writer's cramp' are as follows: A clerk, who from his painstaking and energetic habits has been tempted by his employers to work overtime or possibly against time, and who perhaps (as the history of these cases often shows) is harassed by domestic troubles, discovers that he does not write quite as easily as he did, that his hand possibly aches after prolonged writing, and that he finds it convenient to adopt some new method of holding his pen. At first the trouble is hardly noticeable, and then he finds himself obliged to grasp his pen with unusual tightness to prevent its becoming unsteady. Then his handwriting begins to suffer; perhaps his forefinger refuses to remain steadily upon the penholder, and while he is making every effort to control it, the pen somehow or other eludes his grasp and falls from his hand. If he persevere—probably he is one of those who would sooner die than give in—his progress downhill is rapid and certain. Every possible method of holding the pen is adopted by turns, such as interlacing it between the fingers, grasping it firmly with the whole hand, using only the first and second fingers without the thumb, or the thumb and second finger without the first; but these expedients quickly fail to be of service. He cannot steady the pen by means of them, and he finds that the arm rolls possibly inwards or outwards, and that he can only form his letters by moving the whole arm from the shoulder, or lastly by fixing the arm to the side and swaying the whole body to and fro. The handwriting soon becomes illegible; and lastly the patient is almost unable, even by the most strenuous efforts, to make a mark upon paper. To write a word legibly in extreme cases is impossible, and there are few spectacles more distressing than to see a patient, whose handwriting was perhaps his means of livelihood, drip with perspiration while making an ineffectual effort to sign his name. His loss of writing power may be his only symptom, as was the case with an American sufferer who wrote thus to the writer: 'That fingers which could guide razor and needle, wield oar and musket, and, though numb with cold, knelt and cast off reef-points on a wet sail, should yet strike work when called on for the familiar character of their owner's name, seemed

utterly beyond comprehension.' Patients should be closely questioned as to their ability to perform acts other than writing. In the writer's experience it is usual to find that some other act or acts is affected. If the patient be asked if he can wind up his watch, carry a full teaspoon steadily to his mouth, or perform some similar act requiring a *delicate* use of the thumb and forefinger, it will very commonly be found that he has lost his deftness in performing such acts, although in using a knife or wielding a hammer, and other work requiring a coarse use of muscles other than those used in writing, he may find no difficulty whatever. Frequently there are neuralgic pains, or a sense of extreme fatigue in the hand or arm; and in some cases the effort to write has been followed by pain in the back or severe headache. In some cases the mental distress caused by the loss of power has produced a condition bordering on melancholia. Slight tremor of the hand is not uncommon, and these tremors sometimes occur independently of writing. Tenderness of the nerve-trunks (median, ulnar, or musculo-spiral) is very often present, and should always be carefully looked for, as indicative of congestion of the nerves, or neuritis. Occasionally there is objective spasm of some of the muscles of the arm when the effort to write is made; and one case has been reported by the writer, in which the symptoms, at first those of simple loss of writing power, terminated in a general clonic spasm of all the muscles of the arm, occurring at last independently of any writing effort.

Although the above is a fair statement of what occurs in a typical case of 'writer's cramp,' other cases are by no means uncommon in which impairment of writing power supervenes quite independently of any unusual writing effort, and even in persons who have done rather less than an average amount of writing. These latter cases are not usually so severe as the former, since they are not obliged, as is the professional scrivener, to goad the unwilling hand to make those efforts which are so detrimental. It has been the writer's experience, too, that in this latter class of cases it is more usual than in the former to find that the trouble is not very strictly limited to the act of writing, but that there is a certain amount of inability or clumsiness in the performance of other acts requiring minuteness and nicety.

A difficulty in writing is, in fact, a symptom of many conditions, and it is doubtful whether 'writer's cramp' can be regarded as a morbid entity. When patients come complaining of loss of writing power, it is always essential to examine them with great care, with a view to discover any changes in the nervous system, whether central or peripheral. The writer has found that difficulty in writing may be part of a scarcely noticeable hemiplegia (congenital or otherwise) depending on the usual brain-lesions, or an early symptom of general paralysis, or paralysis agitans, or disseminated sclerosis; he has found it depending upon mischief in, or connected with, the spinal cord, such as sclerosis of one or other of the columns (anterior, lateral, or posterior), meningeal thickenings, tumours, or disease of the bones; he has known it caused by an aneurysm pressing on the brachial plexus, by neuritis affecting either the large nerve-trunks or the small terminal twigs that run in the palm of the hand; he has known it caused by rheumatic thickenings of muscular attachments, by injury or disease of the

muscles themselves, or by gouty deposits in the sheaths of the tendons. It is therefore necessary to examine these cases with very great care, and to remember that the act of writing may be hampered by the failure of any muscle between the scapula and the hand.

PATHOLOGY.—The morbid anatomy of writer's cramp is unknown, and consequently the pathology of the disease is a matter of speculation. It is obvious, however, from what has been said, that the changes which bring about the difficulty in writing cannot be expected to show any uniformity of seat, but may be suspected in cerebral cortex, conducting fibre, peripheral nerve or muscle, according to the symptoms manifested. Duchenne believed that the change is in the nerve-centres first, because localised faradisation applied to the hands has no good effect on the trouble; and he is confirmed in this opinion because the malady very quickly affects the left hand if it be used to supply the place of the right. The writer has ventured to suggest that the lesion is often situated at the periphery, either in the muscles themselves or in the terminal motor nerves supplying these muscles. In real cases of 'writer's cramp' (the loss of writing power occurring in an overworked scrivener) it is rare to find any sign of central change; neither wasting or true paralysis of muscle, nor fibrillary tremor, nor general tremor of the limb, nor pain, nor spasm (except during, or immediately after, the attempt to write). It is generally observed that the disease progresses from the periphery towards the centre: that the muscles of the fingers are the first to fail, then those of the forearm, then those of the arm, and lastly, in extreme cases, those of the trunk. It is probable that the muscles of pen-prehension, as opposed to those of pen-movement (consisting of the adductor pollicis, first dorsal interosseous, &c.) drift into a condition of 'chronic fatigue' owing to the *prolonged strain* to which they are subjected when holding a pen for long periods at a time. When these normal muscles of pen-prehension are exhausted, others are used to supply their place, such as the superficial and deep flexors in the forearm, which in their turn give out, and thus the trouble steadily progresses. The writer has had no small experience of this disease, and he has rarely failed to find that a careful electrical investigation of the muscles has shown a certain diminished irritability to faradisation in the muscles of pen-prehension on the diseased side, when compared with those of the sound side. This loss of irritability is due, in many cases, he believes, to sheer over-use of the muscles, the intervals of relaxation between the long periods of contraction not being sufficient to allow of their proper nutrition. In many cases of 'writer's cramp' no over-use of the muscles has taken place, and in such cases we must suppose that, from some condition, either constitutional or situated locally in the nerves, the muscles are so deficient in 'staying power' (notwithstanding their ability to contract forcibly for a short time) that they become useless, or nearly so, when called on for prolonged steady contraction. It is obvious that the failure of one muscle (however small) which had been taught, by years of laborious education, to act in harmony with many others for the accomplishing of a complicated and delicate act, such as writing, would produce a true want of co-ordination. In other words, muscles which have been induced to *work in order together* no longer do so if one of

them fails, and the failure of one must be fatal to the accomplishment of the co-ordinated act.

DIAGNOSIS.—Diagnosis is of great importance, since failure of writing power is often an early symptom of disease the prognosis of which is very different from that of 'writer's cramp.' Evidence of chronic alcoholism is often afforded by the hand-writing. So again paralysis agitans and disseminated sclerosis of the spinal cord may very early give a want of steadiness to the pen. Reynolds recorded a case of lead-palsy which was sent to him as a case of 'writer's cramp,' and there can be no doubt that peripheral neuritis is answerable for not a few cases. Paralysis of any of the nerves supplying the hand or forearm, such as the ulna, median, or musculo-spiral, will make writing difficult; and the writer has seen cases of progressive muscular atrophy in which the shrunken and quivering interossei had made a firm grasp of the pen impossible. Among other conditions which have been sent to him as cases of 'writer's cramp,' the writer may mention cerebral embolism and hæmorrhage, cortical thrombosis, general paralysis of the insane, exophthalmic goitre, and hysteria.

TREATMENT.—The chief remedy is rest. Without it nothing can be done; and if rest be taken in the earliest stages of the disease, complete recovery not infrequently results. For such writing as is absolutely necessary, the patient should be advised to use a pencil or soft quill-pen, so that any violent grasp becomes impossible. In some cases the writer has found the rhythmical exercise of those muscles whose irritability is impaired, together with the employment of a mild galvanic current, of the greatest service, and he has recorded a few cases treated in this way which rapidly improved after having resisted every other known method of treatment for years. Injection of morphia is of no use, but in America some good has been said to result from the injection of atropine. Systematic massage is of decided service in many of these cases, but neither massage nor electrical treatment must be employed so long as the nerve-trunks are tender. The first object in the treatment of such cases is to remove the nerve-tenderness by rest and blistering. The administration of a very small dose (gr. i) of iodide of potassium combined with arsenical solution is often useful to this end; and when the tenderness has disappeared, the systematic rubbing of the muscles, especially the muscles of the hand, is most valuable. No patent penholders are of any permanent use in these cases, although they may do apparent good by shifting the burden of writing on to a new group of muscles, which are sure, if writing be persevered with, to break down in time. Typewriters are, of course, invaluable to sufferers from 'writer's cramp,' but only those forms of typewriters which are worked by a piano-like keyboard are admissible. Those which necessitate a prolonged grasp of a handle are dangerous for sufferers from this disease.

G. V. POORE.

WRY-NECK.—**SYNON.** : Torticollis; **FR.** *Torticollis*; **GER.** *Steifer Hals*.

DEFINITION.—A twisting of the neck to one side.

ÆTIOLOGY.—Wry-neck is a symptom which may be produced by very different conditions; and it is usual to distinguish between the varieties of this disease, which may either be *congenital* or *acquired*.

The *congenital wry-neck* arises either from faulty

development of the muscles on one side of the neck (in which case there are not infrequently evidences of faulty development in other regions of the body, and notably in the neighbouring parts, such as the face); or from contraction of the muscles on one side, often due to accidental injury during labour. Spencer found no fewer than sixteen cases of hæmatoma of the sterno-mastoid in 200 bodies of still-born children, and D'Arcy Power has insisted upon the connection between such congenital tumours and wry-neck. In these cases the head is fixed in its abnormal position. *Acquired wry-neck* may be a mere passing condition, due to 'muscular rheumatism,' brought on by exposure to cold, and under the name of 'stiff-neck' it is familiar to everybody (see **RHEUMATISM, MUSCULAR**). The most formidable variety is *spasmodic wry-neck*, which makes its appearance, usually in persons otherwise healthy, about the middle period of life. The muscles on one side of the neck are the seat of spasm, sometimes tonic, but usually clonic, in character. The muscle chiefly affected is generally the sterno-cleido-mastoid, and its constantly recurring contractions have the effect of turning the head away from the side which is the seat of spasm; drawing the occiput slightly downwards, and the chin slightly upwards. Other muscles, such as the scaleni, splenius, and trapezius, are not infrequently affected also; and then, in addition to the rotatory twisting of the neck, we get a lateral downward bending, and an elevation of the shoulder. At the commencement of the disease the trouble may be slight, and cause but a trifling amount of inconvenience, the impressions produced on a bystander being that the patient's movements are caused by some mal-arrangement of the dress, which he is seeking to remedy. In its advanced form the disease is a very terrible one, and may make life hardly supportable. The writer has seen one case in which the spasm was so violent that the constant forcible impinging of the chin upon the shoulder of the sound side had produced a sore place as big as a shilling. The spasm ceases during sleep; and when the patient is undisturbed, and his mind pre-occupied, it is not unusual, even in the worst cases, for considerable remissions of the symptoms to occur. In the presence of others, especially strangers, the spasm is usually intensified, and in this it presents a striking similarity to stammering and writer's cramp.

PATHOLOGY.—The pathology of spasmodic torticollis is very doubtful. It must be regarded in almost all cases as a true neurosis, and must take its stand alongside of histrionic spasm of the face, writer's cramp, and stammering. It must often be a question as to whether some of these cases are not definitely of cerebral origin. The rotation of the head to one side is effected by muscles on *both* sides of the body. Thus the right splenius capitis and the left sterno-mastoid, acting in concert, rotate the head to the right; and in many cases of clonic torticollis it is found that muscles on opposite sides of the body are both conspiring to produce the rotation. The writer has recorded one case in which extreme torticollis was associated with a history of injury to the head close to the upper end of the Rolandic area, combined with a history of syphilis. This case was practically cured by the free administration of mercury. That it is due in many cases to an irritable condition of the spinal accessory nerve there can be no doubt, but how

this condition of nerve is brought about we are unable to say. In many cases, especially in women, there is a large element of what is called a pure neurosis. Want of proper antagonisation is another factor which is always present, for without it the symptoms could not occur. In certain nervous constitutions a weakened action of one sterno-mastoid would seem of itself to be almost sufficient to excite irregular contractions in its fellow. The writer has seen one case in which it seemed tolerably clear that the sterno-mastoid and trapezius on one side had become weakened by overwork, and had drifted into a condition of chronic fatigue, while their antagonists were constantly in a state of clonic spasm, twisting the neck to the weakened side. An electrical examination of the muscles may show that, while those which are the seat of spasm have their irritability greatly increased, their antagonists are below their normal state as to irritability. Torticollis is usually, but not always, uncomplicated. The condition known as 'irritable spine' has been observed in some few female patients. Histrionic spasm, or spasm of the limbs, has occurred as a complication, and it is not a little remarkable, as showing connection between the two diseases, that Reynolds recorded three cases of torticollis in which the patient had suffered previously from writer's cramp. A difficulty of deglutition was observed in one of Reynolds's cases; and in another there was swelling of the arm, from pressure upon the subclavian vein.

PROGNOSIS.—The prognosis of these cases is bad, it being very unusual for recovery to take place after the disease has become well established.

DIAGNOSIS.—The diagnosis of wry-neck, as a rule, is not difficult, but care must be taken that simple torticollis is not confounded with cases of caries of the cervical spine, in which there is frequently a tendency for the head to twist to one side. It should be borne in mind, also, that in some few cases of organic brain-disease, in which synergic movements of the eyeballs have occurred, a spasmodic rhythmic movement of the neck has also been observed. The other symptoms of brain-disease, however, would usually prevent any such mistake.

TREATMENT.—This necessarily depends upon the cause of wry-neck. In those cases in which the head is fixed by contraction or faulty development of the muscles, tenotomy has been of service. Tenotomy has been practised also in those cases in which there has been definite clonic spasm; but notwithstanding that temporary relief has in some

cases followed, it has been found that the deep-lying muscles are almost certain to take on a spasmodic action, and that in the end the patient's discomfort is increased rather than diminished. A tenotomised muscle is liable to shorten; and it has appeared to have been the case that, although tenotomy gives temporary relief, it really aggravates the trouble in the long run.

The removal of a piece of the spinal accessory nerve in the neck, as practised by Campbell de Morgan and others, will completely paralyse the sterno-mastoid and upper part of the trapezius, and is not open to the objection urged against tenotomy. This manoeuvre is stated to have given great relief even in those cases where muscles other than those supplied by the spinal accessory have been implicated. Various mechanical contrivances for steadying the head have been devised, but few patients are able to bear the constant pressure of the apparatus, and the remedy has been found in most cases worse than the disease. The stretching of the spinal accessory, the ligature of the same nerve (Collier), and the suspension of the patient by the head, are all measures which are calculated to relieve. Some good has resulted from the long-continued use of hypodermic injections of morphine, and the exhibition of large doses (3j and over) of the succus conii. Electricity has not been particularly serviceable in the treatment of torticollis. The galvanic current has been used to control the spasm, while the weakened antagonising muscles have been stimulated by faradisation; and although improvement has been produced by these means, it has generally been of very short duration. The writer some years ago had a very severe case under his charge, which was successfully treated by the employment of the continuous galvanic current, combined with the rhythmical exercise of the affected muscles. The positive pole being placed behind the ear, the negative pole was applied over the sterno-mastoid and trapezius, and at the same time the patient was made to exercise these muscles by shrugging the shoulder, twisting the head, and other movements. The result was very rapid and permanent improvement. Another case, also, was successfully treated by the writer by faradising the antagonist muscles, which were somewhat wasted and decidedly deficient in irritability. This patient completely recovered; although tenotomy of the sterno-mastoid, which was the seat of spasm, had afforded only temporary relief.

G. V. POORE.

X

X-RAYS.—See RÖNTGEN RAYS.

XANTHELASMA (ξανθός, yellow; and ἔλασμα, a lamina).—A yellow growth in the superficial layer of the skin, assuming the form of a lamina or plate, and more frequently met with in the eyelids than elsewhere. See XANTHOMA.

XANTHOMA (ξανθός, yellow).—**SYNON.**: Xanthelasma, Vitiligoidea; Fr. *Molluscum Cholestérique* (Bazin); Ger. *Fibroma Lipomatodes* (Virchow).

DEFINITION.—A rare disease, characterised by a peculiar kind of yellow growth, in the form of plates or raised nodules, in the skin, and sometimes subsequently in areolo-fibrous tissue elsewhere.

ÆTIOLOGY.—The presence of xanthoma is frequently associated with megrim or other neuralgiae, with gouty conditions, and with disorders of menstruation. It occurs in such frequent connection with those morbid functional conditions or actual organic diseases of the liver which lead to the presence of jaundice that the influence of this as an

ætiological factor cannot be gainsaid, although the nature of the relationship is not apparent. These conditions are, in order of probable relative frequency, chronic catarrh of the bile-ducts, hypertrophic cirrhosis, biliary calculi, atrophic cirrhosis, hydatids, and carcinoma. Chronic dyspepsia, lithæmia, and other conditions attributed to hepatic disturbance are also often present. In the great majority of cases jaundice precedes xanthoma by a prolonged period—usually a year or more—and is generally present when it develops. In a certain number of instances, however, all signs of jaundice may have disappeared, while in others—noted especially by French writers—a persistent yellowish tint of skin (*xanthochromia*), not identical with jaundice, and unaccompanied by other evidences of bile-absorption, has been noted. On the other hand, xanthoma is frequently seen in persons apparently healthy in every other respect.

The female sex is much more frequently affected than the male, in the proportion, according to Hutchinson, of three to two. Xanthoma generally begins about or above the age of forty years, but a dozen cases or more are recorded in children below puberty. These have never been in association with jaundice, but many have been either congenital or hereditary, while the existence of the disease in several members of the same family, and even in successive generations, is sometimes so striking as to suggest to some recent writers the propriety of classifying it among the *nævi* (Török, Hallopeau). 'Probably slight developments of xanthoma are not so rare as is generally supposed, but give rise to no trouble, and are generally overlooked' (Crocker). Such cases are often unilateral.

ANATOMICAL CHARACTERS.—The following description of xanthoma is based upon the examination of growths in the skin alone.

The upper epidermic layers are healthy, but there is usually a small quantity of granular pigmentary deposit in the rete Malpighii. The changes in the papillary layer are slight, but in the middle and lower layers of the derma there is a good deal of yellowish-brown pigment, both free and in cells. The most prominent and characteristic feature, however, is the involvement of these latter layers by a new-growth of connective tissue, the amount of which is small in the flat, and great in the tuberos variety. In the meshes of the fibres there are numerous round, oval, or polygonal, multinuclear or granular, epithelioid cells, which vary greatly in size. Often they are arranged in masses surrounding blood-vessels or sebaceous glands. These cells, which may be considered as characteristic 'xanthoma cells,' contain many crystals of cholesterolin and tyrosine, as well as yellowish pigment and fat, the last being also abundant in the connective tissue.

There is much difference of opinion as to whether the diseased process ought to be considered a purely neoplastic or a low inflammatory one, but the presence of large endothelial and giant-cells, as well as of the changes in the arterial walls usually associated with chronic inflammation, described by Chambard and others, seems to turn the balance in favour of the latter view. The papules are not due to a collection of sebum within the follicles, as Hebra suggested.

SYMPTOMS AND COURSE.—Two types of xanthoma, (1) the *flat*, and (2) the *nodular or tuberos*, are usually recognised, but as the differences between

them are mainly of degree, and do not turn upon essential characters, they are separately described only in accordance with custom.

1. *Flat or localised xanthoma (X. planum)* is the much more frequent form, and the great majority of cases do not call for medical relief. It generally occurs in middle-aged or elderly women, especially in those of dark complexion or who suffer from neuralgia or 'any conditions capable of producing dark areolæ round the eyes' (Hutchinson). In the immense majority of cases the disease is situated upon the eyelids, where it generally begins on the left side, above and a little outside the inner canthus. Thence it extends slowly outwards along the upper lid and more slowly downwards to and along the lower lid, so that in extreme cases the eye may be surrounded by a xanthomatous ring. After a variable time the right side becomes similarly affected, and the disease pursues the same course as on the left side. The patches when first noticed vary in size from a pin's head to a pea; they are deeply embedded in the skin, which undergoes no alteration in consistence and suppleness; they vary in colour from the palest yellowish-white—to recognise which the blood of the part must be expressed by the finger—to a bright orange or dark brownish buff; and have been not inaptly described as like pieces of chamois leather 'let into' the skin. With a lens the patches can be seen to be composed of fine yellowish granules. At the spreading margin they are sometimes well defined, sometimes they are irregularly serrated, while at other times their colour gradually merges into that of the surrounding skin. Occasionally slight irritation is present, and when the eyes are encircled there may be some discomfort in the movements of the eyelids; with these exceptions no subjective symptoms are present. Single patches of flat xanthoma have in rare cases been noted on the ear, neck, chest, penis, prepuce, and tongue.

2. *Nodular or multiple xanthoma (X. tuberosum, multiplex)* is of greater rarity and more serious import than the previous type. It is also much more frequent in women than in men, especially in those who suffer, or have suffered, from jaundice. Although it may occur independently, it usually shows itself in persons suffering from flat xanthoma of the eyelids, and not infrequently the first step towards its establishment is the appearance of flat lesions of the face, other than those on the eyelids, especially about the nostrils, mouth, or external auditory meatus. The most characteristic and tuberos lesions are situated on the extremities, especially on their extensor surfaces and on points much subjected to pressure, such as the tips of the elbows and knees, buttocks, shoulders, and backs of the fingers. The colour of the nodules varies within the same wide limits as that of the lesions of the plane variety, but often they have tufts of dilated blood-vessels round or over them. In size they vary from a hemp-seed to a pea, but, by their coalescence, swellings as large as a hen's egg, or even larger, may be produced. The epidermis over these is, however, intact, and they are usually only slightly tender to touch and manipulation; occasionally they itch or tingle a little. In most cases a few flat lesions exist here and there over the chest and abdomen, and are present in greater numbers over the palms and soles, where they are ranged in rows along the naturally deep lines of the parts, which they exaggerate. Sometimes they

are similarly arranged in concentric circles round the anus. On the trunk they occasionally have the unilateral nerve-distribution of a zoster. The development of the lesions is usually very slow—extending over years; and ultimately flat xanthoma may show itself in the mucous membrane of the lips, cheeks, gums, tongue, fauces, pharynx, œsophagus, stomach, bile-ducts, intestine, peritoneum, conjunctiva, larynx, trachea, bronchi, pleura, on the endothelium of the heart or great arteries, and on tendons.

Xanthoma in children presents such well-marked peculiarities as to merit a few words of special mention. It has never been known to be associated with jaundice, but is often so with gout or rheumatism, while it is frequently hereditary, may affect several members of the same family, and is occasionally congenital. The lesions are generally asymmetrical, often unilateral, markedly tuberous; and the eyelids are comparatively seldom attacked.

DIAGNOSIS.—Flat xanthoma may be confounded with *milium* if the little, projecting, whitish masses of inspissated sebum which characterise that disease are thickly grouped together so as to form a conglomerate plaque. Almost invariably, however, a few characteristic outlying lesions are present. In any case the sebaceous contents of a milium can always be squeezed out after a shallow incision has been made, whereas in xanthoma the lesions are deeply situated in the corium, and cannot be expressed. It will suffice to mention the possibility of errors in diagnosis with *flat warts*, *colloid degeneration of the skin*, *adenomata of the sweat or sebaceous glands*, and the extremely rare condition termed *pseudo-xanthoma elasticum* (Darier, Balzer), which is a degenerative disease of the elastic tissue of the skin.

In the diagnosis of multiple xanthoma the presence or history of jaundice is an important factor. The skin-disease which most closely simulates it is *urticaria pigmentosa* (hence called *xanthelasma* by Tilbury Fox), but the history, distribution, and subjective symptoms of the two diseases easily serve to distinguish them.

PROGNOSIS.—In the great majority of cases xanthoma progresses slowly but steadily for months or years, and afterwards persists unchanged. In at least four instances, however, very gradual spontaneous recovery has been known to occur, the skin regaining its normal characters. In a very small number of cases death has been attributed to patches on the endothelium of the heart or great arteries.

TREATMENT.—Treatment is seldom resorted to except for the removal of disfiguring plaques from the eyelids. This can best be accomplished by excision, and great care is necessary not to cut too deeply, and thus give rise to subsequent ectropion or epiphora. Electrolysis has also been employed. A 10-per-cent. solution of perchloride of mercury in collodion has been successfully used by Stern for the cauterisation of painful masses on the elbows; while Kaposi states that he has removed xanthoma-nodules from the palms and soles by repeated vigorous washing with soft soap, and making the patients wear indiarubber gloves and socks. Internal treatment is of no use. J. J. PRINGLE.

XANTHOMA DIABETICORUM.—SYNON. : *X. Glycosuricum*; *Lichen Diabeticus*.—Although this lately recognised and rare condition—the

identity of which was first clearly established by Malcolm Morris in 1883—is now by universal consent considered as a member of the xanthoma-group, it presents several points of distinction which entitle it to separate description, in which its chief clinical characters may conveniently be contrasted with those of ordinary xanthoma. The following conclusions have been arrived at from a study of all the indubitable cases on record, a considerable majority of which have occurred in males, the age of the persons affected ranging from seventeen to forty-eight years.

COURSE AND SYMPTOMS.—The disease almost invariably occurs in people actually passing sugar in the urine, although frequently they have been noted as stout and apparently in good health. In a considerable proportion of the cases the presence of sugar in the urine is transitory, and unaccompanied by the other phenomena of true diabetes mellitus. In some cases, moreover, there is only a history of antecedent glycosuria. Many of the patients are alcoholic, and usually beer-drinkers; while others are remarkably obese. The presence of pentose has been discovered in the urine of one case (Colombini), and this fact has been invoked in favour of a pancreatic origin of the disease (Hallopeau).

The eruption evolves quickly, and, after a variable stationary period extending over months or years, diminishes with considerable rapidity, to disappear ultimately without leaving any trace of its existence. Sometimes its course is intermittent and successive outbreaks occur, while occasionally fresh lesions develop during the retrocession of older ones. As a rule the parts first affected are the extensor surfaces of the limbs, especially of the forearms; subsequently the lesions usually appear on the elbows and knees, where they have a great tendency to become confluent and form raised plaques. They are commonest on extensor surfaces, the buttocks, back, face, scalp, mucous membrane of the mouth, and bends of the ankle, but they do not generally affect the flexures. They have only once been observed on the eyelids, and the presence of jaundice has never been noted. The constituent nodules are at first bright red, but subsequently become yellow from fatty degeneration. They are much firmer and denser than those of ordinary xanthoma, are well defined, rounded or obtusely conical at the apex, and may present a yellow point like pus at the summit, but are in reality solid; often there is a bright zone of congestion round their base. In some cases the nodules are very pale, while in others their colour remains pink rather than yellow. Many of them are in obvious relationship to hair-follicles. Itching and tingling are often troublesome, and occasionally there is great tenderness during the evolution of the tumours, while neuralgic pains sometimes precede their eruption. Occasionally ulceration and deep scarring ensue.

ANATOMICAL CHARACTERS.—Recent researches establish the close relationship between ordinary and diabetic xanthoma; 'discrepancies in the recorded appearances being fully accounted for by variations in the rate of the inflammatory process and differences in the methods of hardening and staining employed' (Morris). Typical xanthoma-cells are present, albeit in less abundance than in flat xanthoma, while the inflammatory changes are more marked, especially in connection with sebaceous

and sweat-glands. They also extend upwards to involve the papillary layer, and the passage of fat even into the epidermis has been noted.

TREATMENT.—Mild tarry or lead-lotions, separate or combined, generally suffice to allay or moderate the itching. If sugar disappear from the urine under the influence of dietetic or other measures (codeine, salicylate of sodium, &c.) the eruption usually soon disappears.

J. J. PRINGLE.

XERODERMIA PIGMENTOSA.—SYNON.:

Xeroderma Pigmentosa.

DESCRIPTION.—This a very rare disease, which with few exceptions commences in the second year of life. Without any previous symptoms of apparent cause, but sometimes preceded by a morbilliform or patchy erythema, a copious outbreak of freckle-like pigmentation appears on the face, neck, and upper extremities, i.e. in the uncovered parts, reaching about half-way up the upper arms, the legs being sometimes slightly involved. Other lesions follow, namely telangiectases, white atrophic areas, pigmented flat warts, and ulcerations on which papillomatous growths form. These, if not removed, become epitheliomatous, and thus lead to the patient's death. Conjunctivitis and pterygium are nearly always present, and the discharge is largely responsible for the onset of the ulcers. Cicatricial contraction of the various lesions produces much disfigurement. The aetiology and pathology are unknown. The ultimate prognosis is bad; but the disease progresses very slowly, and if the eye-affections are effectually treated from time to time, the ulcers scraped with a sharp spoon, and all soft papillomatous growths promptly removed,

the cancerous stage may be averted for an indefinite period. Internal treatment is unavailing.

H. RADCLIFFE CROCKER.

XEROPHTHALMIA (ξηρός, dry; and ὀφθαλμός, the eye).—SYNON.: *Lippitudo arida*; *Xerosis Conjunctivæ*.—This term signifies a dry ophthalmia, or chronic inflammation of the conjunctiva without much (if any) watering or discharge. The conjunctiva is thick, dull, opaque, and often skin-like. In severe cases it is shrunken, and the oculo-palpebral folds may be obliterated. The epithelium of the cornea may also be involved. The morbid changes may be the result of injury, especially burns; or it may follow trachoma, diphtheria, pemphigus, and other severe forms of conjunctivitis; or it may be due to insufficient covering of the eye in the various forms of lagophthalmia. A peculiar form of xerosis conjunctivæ occurs in association with night-blindness (see NYCTALOPIA). On the ocular conjunctivæ are dry, scaly-looking patches, which by gentle friction of the lids may be worked into a fine foam which abounds in bacilli (*Xerosis-bacillus*).

J. TWEEDY.

XEROSIS-BACILLUS.—This organism is an occasional inhabitant of the normal conjunctival sac, and is found, in association with specific bacteria, in many of the pathological conditions of that structure. It derives a fictitious importance from its striking resemblance to the *B. diphtheriæ*, not only in morphology but also in several biological characters. It is, however, a pure saprophyte, and possesses no pathogenic properties whatever.

XEROSIS CONJUNCTIVÆ.—See XEROPHTHALMIA.

Y

YAWNING (A.-S. *gánian*).—SYNON.: Fr. *Bâillement*; Ger. *Gähnen*.—Yawning is one of the physiological expressions of fatigue. It consists, when fully developed, of the following phenomena: (1) A deep inspiration, with elevation of the uvula and palate. (2) A forcible spasmodic depression of the lower jaw. (3) A flow of tears. (4) A clicking sound in both ears. (5) A tendency (sometimes irresistible) to stretch the limbs, especially the arms. (6) An expiration, often accompanied by a sound, the character of which has probably given the name to the act.

ÆTIOLOGY AND PATHOLOGY.—This strange and complicated act suggests many reflections. Why, when we are tired, should we take a deep breath? It may be that the need of oxygen experienced by the fatigued tissues, laden with waste products, prompts the instinctive and forcible descent of the diaphragm; but, on the other hand, the facts that yawning is common when we are merely *ennuyé* and not genuinely fatigued, and that it is decidedly infectious, are both unaccountable on such a theory. Why should the lower jaw be spasmodically depressed when we are fatigued? The elevators of the lower jaw are the temporal, masseter, and internal pterygoid muscles, and it is probable that the external pterygoid (whose main functions it is

to move the jaw forward) has also some share in keeping the teeth in contact. These muscles are constantly in action, having to support the jaw against the force of gravity; and it is not surprising that, as in other cases of prolonged muscular action, they should be prone to evince fatigue by failure of function. Were it not for the fact that this work of supporting the jaw is divided among eight muscles (four on either side), it is probable that the jaw would fall more often than it does. When one set of muscles is fatigued, it is quite in accordance with experience that their antagonists should take on a spasmodic action, and we may fairly look upon the spasm of the depressors of the jaw, when the elevators are fatigued, as the physiological expression of an occasional pathological phenomenon. These considerations again do not apply to those instances of infectious (emotional) yawning, in which we are impelled to imitate what we see in others.

A fact of considerable interest in connection with yawning is the great extent to which its phenomena are limited to the area of the fifth nerve. The four muscles mentioned above, as concerned in the negative phase of yawning, are all supplied by the fifth; and the anterior belly of the digastric and the mylo-hyoid muscles, which are connected with

its positive phase, are also supplied by the fifth. The flow of tears and the 'click' in the ears, which is probably due to a contraction of the tensor tympani, are referable to the influence of the same nerve. The tensor palati is likewise in action; so that we may say that every muscle supplied by the fifth is concerned in yawning. Possibly yawning may be regarded as a reflex phenomenon, of which the most ordinary stimulus is the fatigued condition of the elevators of the lower jaw. The reflex effects are largely manifested in the area of the fifth nerve, but they clearly spread beyond it, and produce contraction of the diaphragm, palatal muscles, depressors of the hyoid bone and larynx, and sometimes of the muscles of the body generally, but mainly of the extensors. That the contraction of the body-muscles at least is a purely reflex phenomenon, may be gathered from the interesting fact which has been often observed in cases of hemiplegia, namely, that when a hemiplegic arm is entirely beyond the control of the will, and its flexor muscles are contracted, this contracted helpless hand will often open during yawning, the stimulus having reached the extensors of the fingers, not through the ordinary path of the will, but through the spinal cord by a reflex action travelling along some other path of nervous energy.

In these days when so much is heard of 'hypnotism' and 'suggestion' it is perhaps profitable to bear in mind the common act of yawning, which when performed in public not only compels the spectator to perform a similar act, but definitely begets in him that mental condition of fatigue and *ennui* which is almost inseparable from the physical condition.

TREATMENT.—Yawning is generally only a passing phenomenon, which is removed by rest. An undue tendency to yawn may indicate that the body is too easily fatigued; and then it may be necessary to inquire whether the demands upon it (mental, physical, or sexual) are too great. These may have to be removed or avoided. At the same time the bodily health should be carefully attended to. Iron tonics may be given; the foul atmosphere of crowded assemblies should be forbidden; the diet should be nutritious and digestible; and the amount of alcohol should be carefully restricted to that physiological minimum which may be necessary to aid digestion.

G. V. POORE.

YAWS.—See FRAMBESIA.

YELLOW FEVER.—SYNON.: Fr. *Fièvre jaune*; Ger. *Gelbes Fieber*; Span. *Fiebre amarilla*; Port. *Febre amarella* (also *Typho Americano* and *Icteroide*).

DEFINITION.—Yellow fever is an acute specific disease which has a curiously limited geographical distribution; which is generally of short duration; and one attack of which confers a considerable degree of immunity. In places where it is endemic, outbreaks of an epidemic character are liable to occur when a sufficient amount of susceptible material (as immigrant foreigners) is introduced; or, again, they may arise in places which have long been free of the disease by the importation of actual patients, if the local meteorological conditions are favourable. It is characterised by pyrexia, prostration, and often hæmorrhagic phenomena; except

in the mildest cases there is marked albuminuria, and frequently also some degree of jaundice.

DISTRIBUTION.—The home of the fever is in the warm moist climates of Central and South America, many of the West Indian Islands, and on the West Coast of Africa (Gambia, Guinea, &c.). It has frequently made ravages in the southern States of North America. At various times it has made its appearance in more northerly and remote regions, as far as Montreal and Buenos Ayres in the western continent, and Swansea, Brest, and St. Nazaire in the eastern; the most important, however, being the epidemics of Lisbon, Madrid, and Gibraltar. In none of these places, however, has the fever taken firm and lasting root. The farthest that it has reached in an easterly direction appears to have been Italy. It appears to be unknown in the Orient, and in the Australian and Polynesian regions. When epidemics have occurred in more northern climes, they have spontaneously ceased with the advent of cold weather and frost. Some elevated situations, as the city of Mexico, have constantly escaped, although the disease has long been rife in neighbouring towns at a lower level, as Vera Cruz; the same is stated of Petropolis and Rio de Janeiro. In general, it seems to have been carried about by shipping, though cases are not wanting where it has passed overland. The general kind of nidus which favours its development consists in a warm, moist climate; it is essentially a disease of towns.

Racial and Local Immunity.—It is commonly said that the inhabitants of yellow-fever areas are not susceptible to the malady; observation has, however, shown that the Cubans, for example, may suffer from typical attacks, although they certainly enjoy considerable freedom from the illness. This is almost certainly due to their having passed through it during childhood; such attacks may pass under other names (as 'Borras fever' in Cuba). Coloured races, also, are often stated to be insusceptible, but it has been seen that in places which are virgin soil (as St. Paolo) the blacks have suffered severely; occasionally cases may be seen among them in endemic areas.

Seasonal Prevalence.—In the equatorial and truly tropical regions, the fever generally exists all through the year, but it especially favours the drier period of the year (e.g. at Pará on the Amazon, from April or May onward); in such places, no time can be considered quite safe for the foreigner. On the other hand, in places where there is a distinct winter or cool season, the stranger may then live with a considerable degree of impunity (e.g. at Rio de Janeiro, the summer months, January and onward, form the dangerous period). The diminution after great prevalence is no doubt often due to the cessation of immigration of susceptible persons when notoriety of the condition has spread.

Incubation (1) of the Community.—It is an old observation that a period of about three or four weeks has elapsed after the introduction of persons suffering from the fever before it spreads further. This period has been more exactly traced by Carter; by the study of the onset in a scattered village, it was found to be about three weeks (14–21 days). Naturally this must include the period of incubation in the individual and the time required for the development, multiplication and dissemination of the causative agent. This incubation-period, as well as the necessary high external temperature, may be

considered as contributory elements in the evidence for the existence of an intermediary agent in the spread of the fever. See p. 727.

Often it has happened that the original infecting case has not been recognised, whether that may have been due to the mildness or atypical nature of the attack or to the want of astuteness of the physician; at the same time it must always be remembered that an ill-advised concealment of the presence of the disease is lamentably frequent in yellow-fever districts.

(2) *Of the Individual.*—The personal incubative stage is probably not more than a few hours shorter than three days, nor a few hours longer than six days. Former writers have given it as short as one day, and as long as several weeks. The most exact records are those of the experimentally infected cases of Reed, Carroll, and Agramonte ('American Medical and Carter's Epidemiological Enquiry,' 1901). Persons who have visited patients have themselves been taken ill on the following day; these have been regarded in the past as infections from direct contagion. However, the possibility of infection from pre-existent common sources was not duly discussed. It may be stated plainly that there is no satisfactory evidence of transmission by direct contagion; furthermore, there are many points which exclude such a mode of spread.

Mode of Dissemination.—Various factors have been accused of being the transmitting media. Water may be considered as definitely excluded; many facts are opposed to various articles of diet, of which native fruits are especially incriminated by the Brazilians; transference by means of fomites has been much debated, one direct experiment (Reed) proved negative.

Mosquito-theory.—The first to propound that the fever was transferred by gnats was Finlay of Havana in 1881; satisfactory evidence of an experimental nature was obtained by Reed, Carroll, Lazear, and Agramonte in 1900. The species of mosquito experimented with was *Stegomyia fasciata* (Theobald); it was called 'Culex mosquito' by Finlay, who selected it for his experiments as being essentially a town gnat; it has also been known as 'Culex fasciatus' and 'taeniatus.' It is a black insect with silvery markings on the thorax and at the joints of the legs; the proboscis is entirely black. It is very common about houses, and breeds freely in casual waters in tubs, tins, &c. about houses. It is the commonest mosquito between the 30th parallels. It is not yet known whether other kinds of gnats are capable of transferring the fever; since, however, *S. fasciata* is essentially a day-biting insect, it is probable that some night-biting sorts are able to do so, since there is rather credible evidence that the disease is commonly acquired at night.

Twelve successful inoculations of the fever were made upon susceptible persons at a special camp near Quemados, in Cuba; other susceptible persons in the same surroundings served as controls, and it was solely those that had been submitted to artificial infection that acquired the fever.

The mode of experiment was to feed the gnats upon patients suffering from yellow fever during the first three days of illness; the gnats were then kept for a fortnight or more, when they were allowed to feed upon the subject for experiment. Probably at least ten days' incubation in the gnat are requisite before it is capable of transferring the malady. Some of the insects were found to be

infective nearly two months after their original feed upon the yellow-fever patient. Happily all of these cases ran a comparatively mild course, though they were sufficiently well marked to leave no doubt that the attempt to infect the disease-germ by passage through the gnat had been successful. What length of time the infective agent remains in the patient in such a manner that it can infect gnats (peripheral capillaries or tissues) is not yet established; the matter, however, is one of some importance. It is perhaps only right to remark that the method of experiment is full of the risk of a fatal issue, and should not be lightly undertaken. It will be observed that when a case of yellow fever has been introduced into a virgin district, an interval of at least about a fortnight must elapse before a secondary case can occur (i.e. incubation in gnat say at least ten days, plus incubation in man say three or four days); care of course being taken that no antecedent mild case has been overlooked when testing the question. The facts relating to the occurrence of the fever on ships at Pará are highly suggestive of a transference by mosquitoes, the insects being carried in the lighters from the city, two miles distant, other modes being almost absolutely impossible (Durham).

ESSENTIAL CAUSE.—No micro-organism as yet has been satisfactorily determined as the cause of yellow fever. Various bacteria have been described by different authors—that called *Bacillus icteroides* by Sanarelli has attracted a good deal of attention recently, but there is no reason to think that it has any relation to the fever, as indeed appeared very doubtful in Sanarelli's own account. Description of this bacillus is not called for here, as it may be considered definitely excluded by the work of Reed and his colleagues, and of the writer with Myers. The writer's belief from his own observations is that the disease is due to a bacterial rather than to a protozoal parasite; moreover, several points concur in favouring this view (shortness of incubation, and, of course, rapidity of acquisition of immunity and absence of splenic enlargement). On the other hand, the part played by the gnat is suggestive of a protozoal origin; none such has as yet been discovered. By direct transference of blood from sick to healthy, Reed has proved that the organism is present in the blood during the first days of the disease.

Acquired immunity.—It is commonly stated that the immunity conferred by one attack is very complete; this means that such an individual is unlikely to suffer again from a typical or fatal attack of the fever so long as he remains in an infected neighbourhood. With an experience of rather less than a year the writer met with at least five well-determined second attacks, as well as others which could not be precisely established. It appears that an absence for a few years from an infected area leads to loss of the acquired protection; the duration of the more complete immunity has not been accurately determined; perhaps it is about four years. The resident in an infected area may be considered to be constantly having his immunity renovated by reinoculations, which have little or no effect upon his health. So long as he does not absent himself too long in uninfected places he is most unlikely to 'have the fever' again.

ANATOMICAL CHANGES.—Externally the *yellow tinge*, which almost always appears after death in cases which have died before the onset of icterus

(e.g. on fourth day), and blood about the mouth or nose may assist diagnosis.

The *liver* is slightly if at all increased in size; in colour it may be compared to that of washleather in various stages of use. Here and there it is injected or shows hæmorrhagic patches; it is sometimes rather 'nutmeggy,' and always yellow and fatty on section. Microscopically, besides the intense fatty change, the atrophic and necrotic changes in the cells are of great importance for diagnosis.

The *kidneys* are swollen and in a state of acute nephritis, the vessels being much engorged; the renal cells are full of fatty globules, and the tubules are mostly blocked with cylinders or granular débris and cast-off cells.

The *spleen* is plump and full of blood, deep red but not enlarged.

The *stomach* is injected, especially the cardiac part, and there may be ecchymoses and erosions; it is coated almost constantly with a blackish-brown layer of altered blood, and generally contains 'black vomit.'

The *duodenum* and *small intestine* likewise may contain black material or blood, and are the seat of injection or of hæmorrhage.

The *urinary bladder* is contracted and empty, except where death has occurred late (e.g. tenth day). Lymph exuding from organs is more or less intensely yellow; there may also be staining of the interior of vessels, endocardium, pelvis of kidney, &c.

The *gall-bladder* is too varied in size and contents for further remark.

The *lungs* present nothing of interest.

The *heart* shows fatty change about the papillary muscle, but the general mass appears to escape.

The *mesenteric glands* are large and pink; the writer and Myers found that the *axillary glands* were much enlarged and acutely inflamed, often hæmorrhagic; this was thought to be probably the result of absorption or dissemination of virus from specific gnat-bites about the hands and wrists.

The *central nervous system* is usually engorged with blood, and may be stained yellow, though not constantly, even when much jaundice is present.

SYMPTOMS AND COURSE.—The fever is generally described as consisting of two stages with an intervening intermission or 'period of calm'; this last is practically never seen except in books, and has been given its proper value by some of the more recent writers. The first stage is very frequently ushered in by a rigor; the temperature runs up to say 102°–104° F. Sometimes the onset is more insidious and gradual; there is most intense headache, generally frontal, and pains in the lumbar region and calves, the latter being also sometimes tender. Simple vomiting may also occur; it would seem to be especially common in the atypical form of the fever which usually affects children. The face and chest show slightly dusky congestion and the somewhat bloated 'drunken man' look of the face (yellow-fever facies) is most useful for diagnostic purposes; thirst, anorexia, and lassitude must also be added. In cases of slight or medium severity the temperature falls and regains the normal about or before the fourth day. There is no typical temperature-curve for this malady; in very severe cases the temperature may run high, as 106° or 107° F., but this does not seem to be common. Even at the early stage the pulse, which is full and

strong, is relatively slow, as 100–120, with a temperature of 104°, but this slowness is more a feature of the later periods. The typical tongue—pointed, with furred middle and bright red tip and edges—is frequently seen; the gums are generally coated with non-diagnostic white fur-like material, which soon clears off in favourable cases; later they become swollen and readily bleed. The urine is thick, high-coloured, loaded with urates, and albumin may sometimes make its appearance as early as the second day, though not abundantly until later.

The second stage is characterised by the onset of hæmorrhagic and asthenic symptoms. They may be missed in the milder cases and in children.

The fourth day may be said to be the time for the onset of these more grave symptoms. The most celebrated is the *black vomit* (*vomito negro* or *preto*). The vomit at first consists of ingesta or water becoming bile-stained; when typically 'black' it consists of clear water or fluid, in which are suspended small flakes of brownish-black material, which gravitate on standing: the flakes have been compared to flies' wings, and, though the comparison is not very apt, it suggests the appearance somewhat; the flakes are formed by small quantities of extravasated blood from the stomach, the hæmoglobin of which has been altered by the acid gastric juice. Whilst by no means necessarily a fatal sign, it is not a very reassuring one, especially to the patient, who has exaggerated notions of its import.

Of the *hæmorrhages* other than black vomit, in which the quantity of blood effused is not really great, there are bleedings from gums and nose, both of which are common in moderately severe cases. The latter is occasionally alarming, but is said never to cause death. In severe cases gastrorrhagia and enterorrhagia, and more rarely subcutaneous, subconjunctival, and intramuscular extravasations are met with. In women bleeding from the uterus is common; abortion is also mentioned as a constant feature in the pregnant.

The *urine* becomes very highly albuminous—so much so that often a sample boiled in a tube becomes almost solid; it also contains more or less abundant hyaline and granular cylinders, kidney-cells, and sometimes red blood-corpuscles. In severe cases it may take a week or more to clear up. A more grave feature is the ominous diminution in the flow of urine; complete suppression for twenty-four hours may be considered an almost infallible death-knell. Where jaundice is marked the urine becomes coloured greenish to blackish-green from the bile-pigments.

Pain and tenderness in the epigastrium are almost constant signs, and if severe and lasting they are of grave import.

In children (under ten years) albuminuria, icterus, black vomit, and hæmorrhages are said to be generally absent, vomiting and collapse being marked, death often occurring with symptoms like meningitis.

The *jaundice*, whence is derived the name of the malady, next claims attention. It appears about the fourth day as a yellowness of the conjunctivæ; more or less simultaneously a faint lemon-yellow tinge spreads over the body; this, perhaps, is the commonest extent of the colouration. In the milder cases, if present, it may be limited to a transitory tinge in the eyes; some writers lay stress upon an orange tinge of the palpebral conjunctiva. The writer has not been much impressed by this.

A more practical point is the fact (Guiteras) that a slight jaundice may be better detected at a couple of yards distance than quite close to the patient. Deeper degrees of icterus are also seen, either a generalised deep yellow, like that of an ordinary case of catarrhal jaundice, or a deep orange-brown colour. In such cases the colour may last for weeks, when the ordinary clay-coloured stools are to be observed.

The leucocytes in the blood are not increased markedly; in the last stages of fatal cases they are much diminished.

Edema of the conjunctiva is not uncommon in severe cases of a week's duration; otherwise the nephritis is not associated with cedema.

There seems to be some uncertainty about the bowels; some authors mention constipation, others diarrhoea. The constant preliminary purge and the usual subsequent administration of gentle salines and enemata make it impossible to come to a decision concerning this. Often there are small pieces of mucus in the stools, and microscopically they may show large numbers of a very fine bacillus (Sternberg, and the writer), as well as red blood-corpuscles, leucocytes, &c., in some cases. Death is almost always referable to the onset of anuria and the resulting uræmia with its convulsions. Direct intoxication with the specific poison and the absorption of intestinal products, unaltered by the action of the damaged liver, also have their share.

PROGNOSIS.—Often a very shrewd guess may be made almost from the first, from the general state of the patient. *Good signs* are the early fall of temperature (about the third day), the onset of sweating, low degree of albuminuria, good sleep, and calmness of the patient. *Bad signs* are persistent vomiting, hiccough, high degree of albuminuria, hæmorrhages, hypo-leucocytosis, great prostration, dry brown tongue, continued epigastric pain, anxiety, progressive decrease of urine passed, restlessness, delirium, continued insomnia, getting out of bed, desire for food while obviously ill, and *rising pulse with falling temperature*.

TREATMENT.—Since the writer has not had the actual care of patients, he wishes rather to lay down the general principles than to advocate any special type of treatment. Physicians in yellow-fever countries seem rather too apt to expect a fatal issue. No drug or serum is as yet known which has a specific action. Most drugs seem better avoided; in the main we are quite helpless and have to trust to time and nature; at the same time, it is possible to prevent a recoverable patient from getting well. It should always be borne in mind that the liver is soon so affected that it is incapable of dealing with drugs like alkaloids, or material like meat-extracts (cf. the effect of the latter in Pawlow's 'liverless' dogs).

The onset of suppression of urine is most to be feared, and all means calculated to avoid chilling the skin should be taken. The careful measurement of the urine passed is probably of far greater importance than the use of the thermometer or watch. If the quantity begins to decrease markedly, vapour-baths or hot packs and blankets should be used. At the same time it is necessary to supply water to the system; large enemata of normal saline (e.g. half a litre) should be commenced at the onset of the illness in all cases; later they may be increased, and the patient should be adjured to retain

them as long as possible; ingestion by the mouth is prevented by the vomiting. As a last resource saline solution may be injected beneath the skin or into a vein. The writer has seen very many drugs given (e.g. trinitrin, digitalis) without the slightest semblance of good.

It is well to remember that the worst conditions may ensue in the pyrexial stage, and to beware of a falling temperature with a rising pulse.

At the outset probably nothing is better than the old-fashioned purgation with calomel and castor-oil. Give nothing but water, mineral if preferred, for the first three or four days; the patient will not starve and will not wish for more; tell him to sip a spoonful or so at a time, or deal it out so that he cannot take a long draught, which he will only vomit up. Feed by the rectum if food must be given in the early days and a severe attack seems indicated; sucking ice and iced water perhaps tend to excite rather than allay vomiting. Alcohol, especially in the form of champagne, is often given. It seems doubtful whether it is of any value; cases treated by ships' captains with large excess of spirits seem sometimes to have recovered.

Do not allow the patient to leave his bed until the fourth or fifth day has been safely passed.

On first seeing the patient, try to ascertain whether he acquired the infection within his home or outside it; if the former, remove him to an uninfected house at once; it is not unlikely that severe and prolonged attacks are caused by multiple or continued inoculations while remaining in an infected house. Shelter him from gnats, both to prevent spreading infection and to aid him to sleep, for the insomnia is one of the most trying of the conditions which have to be passed through. Catch and crush (as in a small gauze net) all gnats, especially those in the sick-room, which are full of blood.

Hiccough, a bad sign, is associated with much gas in the stomach; possibly the careful use of a tube might be of service. The writer, from personal experience, can highly extol the effect of an ether-spray directed for a few seconds on the epigastrium.

For the headache an ice-cap is perhaps preferable to drugs, such as phenacetin, though neither effect much. Sponging with vinegar or water in the earlier stages is comforting, but is perhaps risky when the nephritis is well marked. In general, antipyretic measures are not wanted.

Reassure the patient and try to keep his spirits up. Many seem to be taken with a deadly fear if they know or suspect they have yellow fever.

In making a preliminary diagnosis do not forget to think of malarial fever, small-pox, influenza, measles and dengue; the urine will generally clinch the diagnosis in a day or two. Remember, too, that malaria and yellow fever may co-exist.

Prophylaxis of the community.—When the fever breaks out among a collection of susceptible persons it will be well to segregate the healthy in a safe detention-camp if possible.

The quarantine regulations of the United States against the importation of the fever are probably the best, and have been more tried in the face of almost daily risk than others; they entail a division into 'immunes' and 'non-immunes'; the former are not detained, the latter are kept under observation till the incubation-period has elapsed (say five or six days), temperatures being taken before dismissal if any suspicion arises.

General measures against gnats.—Clear out casual waters (roof-gutters, unconsidered tins and tubs); protect permanent waters (cisterns) with wire gauze; these are the breeding places of *S. fasciata*. For cesspools, which breed other house-mosquitoes, use kerosene. Where possible, fix permanent mosquito-bars in windows and doorways. Employ small boys with 'butterfly nets' to catch and kill adult insects, especially in infected houses or where patients are being treated. Remember that the vapour of formaldehyde, though effective against bacteria, does not affect insects; for these sulphur, pyrethrum, &c., are more suitable when burnt. It is probably well to disinfect the excreta of patients. Improved sanitary arrangements of towns have been thought to have diminished the occurrence of the fever; this may be due to an effect on the breeding of cesspool-loving gnats.

Prophylaxis of the individual.—Fever-haunted towns should be avoided during the fever-season, and residence in a healthy suburb, at any rate for the night, if possible, insisted upon. Exposure to sun, excess in food and drink, and sexual excess, are given as causes; the last only has any probable

importance in that many cases are traceable to a night spent in a brothel; similarly, drink may lead to an inadequate adjustment of the mosquito-net.

Avoidance of dishonest concealment of the existence of the disease, the early recognition and isolation of sufferers, both among new-comers and the native children, must be insisted upon. It may be laid down absolutely that every case of fever, however slight, occurring in a yellow-fever country should be looked on as a case of yellow fever, and treated accordingly, until it is proved not to be such.

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Z

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When taken in small doses, zinc produces a chronic form of poisoning, the symptoms of which are derangement of the digestive organs, and peripheral neuritis.

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PROGNOSIS.—The prognosis of zinc-poisoning is generally favourable.

TREATMENT.—Demulcent drinks, sedatives, and alcoholic stimulants should be immediately given. The administration of a solution of the common

sodium phosphate, largely diluted, is advisable. It acts as an excellent precipitant of zinc, in the form of an insoluble phosphate. Opiate enemata may prove serviceable.

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ZINC-WORKERS.—See OCCUPATION-DISEASES.

ZONA or ZOSTER (*zona*, ζώνη, a girdle).—SYNON.: Shingles; Fr. *Zona*; Ger. *Zoster*; *Gürtelrose*.

This term was originally applied only to that form of herpes which runs round one half of the trunk of the body, usually in the region of the waist. See HERPES ZOSTER.

ZYMOTIC : ZYME (ζυμάω, I ferment; ζύμη, a ferment).—SYNON.: Fr. *Zymotique*; *Zymase*; Ger. *Gährungs-fähig*; *Gährstoff*.—The terms *zyme*, *zymine*, *zymotic*, and *zymosis* were introduced by Dr. William Farr, in a Letter to the Registrar-General in 1842, and employed by him to denote in a general way the poison (and pathological processes excited by it) of 'epidemic, endemic, and contagious diseases.' In using the word ζύμη, he was careful to point out that he did not consider the morbid process to be absolutely identical with the ordinary phenomena of fermentation, and that he wished the terms *zymosis*, *zymotic*, &c., to be employed in English, 'not in the sense which they have in Greek, but as general designations of the morbid processes and their exciters.' With this qualification clearly expressed, the use of the root-form ζύμη has become general, not only in scientific literature, but also in the public press—almost invariably, however, in the adjectival form *zymotic*.

Fourteen years later (1856), in the *Sixteenth*

Annual Report of the Registrar-General, Dr. Farr described the diseases of the zymotic class as conveniently referable to four groups. These are: 1. The *Miasmatic*, diffusible through the air or water, attended by fevers of various forms; the matter by which they are communicated is derived from the human body, as in small-pox, or from the earth (as in ague). These two diseases are types of this class. 2. *Enthetic* diseases, which may properly be called *contagious*, being communicated by contact, puncture, or inoculation. Syphilis and glanders are types of this class. 3. The *Dietetic* diseases, which arise when the blood is supplied with improper or bad food. Scurvy and ergotism are the types of this class. 4. The *Parasitic* diseases, which attack especially dirty populations, and infest the skin, the intestinal canal, and all the structures of the body.

This classification, which does not now hold good, is quoted here because it continues to be spoken of. Modern pathology has necessitated its revision.

Recently, indeed, the word 'zymotic' has been restricted to the acute specific diseases, included under the first group (miasmatic) in the above classification; and at the present time it is in this limited sense that it is most commonly used.

Corresponding with the adjective *zymotic* is the substantive *zyme*. This is a useful name, by which we refer to the poisonous cause of zymotic diseases. It is simpler than the word *zymine*, originally proposed by Dr. Farr; and (what is much more important) to speak of the contagious poison as 'a zyme' does not imply the acceptance of any particular theory of disease; while, on the other hand, the use of the word 'germ' distinctly conveys the idea of some organised structure, itself the cause of the disease by subsequent growth and multiplication. See BACTERIA; and INFECTION.

The necessity for employing the word *zymosis* does not seem to be felt as yet; but the same reasons that lead us to speak of the agent as a zyme should also guide us to use *zymosis* in place of the more usual periphrases.

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